



Research article

Modeling and analysis of a novel two-strain dengue epidemics model considering secondary infections with increased mortality

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Abstract: In this study, we developed and analyzed a deterministic two-strain host–vector model for dengue transmission incorporating temporary cross-immunity, antibody-dependent enhancement (ADE), disease-induced mortality during secondary infections, and explicit DENV–DENV co-infection in mosquitoes. The model distinguishes primary and secondary human infections and allows co-infected mosquitoes to contribute to strain-specific transmission. We derived the basic reproduction number $\mathcal{R}_0$ and established the local stability of the disease-free equilibrium, while heterologous invasion of a one-strain endemic state was characterized through the corresponding invasion dynamics. Center-manifold analysis and numerical continuation revealed backward bifurcation, bistability between disease-free and endemic states, and Hopf-induced oscillations. Numerical simulations further illustrated transitions between disease-free, endemic, and periodic regimes. These results show how ADE, waning cross-immunity, and mosquito co-infection can jointly generate complex multi-strain dengue dynamics and provide a mechanistic framework for examining how the serotype composition of the vector population influences subsequent human infections.

Keywords: multi-strain modeling; stability; bifurcation analysis; vector co-infection; numerical continuation

1. Introduction

Dengue fever is one of the most significant vector-borne diseases worldwide. It causes an estimated 390 million infections and hundreds of thousands of hospitalizations per year [1]. Secondary infection with a heterologous serotype can lead to severe dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS). The dengue virus exists as four antigenically distinct serotypes (DENV-1 to DENV-4). While primary infection elicits lifelong immunity to the infecting serotype, cross-immunity to other serotypes wanes rapidly. Additionally, pre-existing antibodies can facilitate infection by a different serotype via antibody-dependent enhancement (ADE). ADE has long been considered a significant risk factor for severe dengue disease during secondary heterotypic infections. There is clinical evidence linking antibody titer ranges to disease severity in children [2].

Mathematical modeling has played a vital role in understanding the epidemiological consequences of multi-strain dynamics, ADE, and transient cross-immunity [3]. Early theoretical work demonstrated that simple two-strain models with temporary cross-immunity and immune enhancement can exhibit behaviors such as backward bifurcations, oscillations, and chaos [4]. For instance, Aguiar et al. (2008) [5] introduced a minimal two-strain susceptible-infected-recovered (SIR)-like model showing complex bifurcation structures and chaotic attractors under biologically plausible parameter regimes. Bianco et al. (2009) [6] analytically explored how weak temporary cross-immunity may stabilize otherwise chaotic ADE dynamics, and how stronger cross-immunity can induce complex epidemic cycles. More recent studies have modified these frameworks to explicitly incorporate vector-host transmission dynamics. Rashkov and Kooi (2021) [7] developed a compartmental host-vector model incorporating two dengue strains, temporary cross-immunity, and potential secondary infection enhancement. This model shows multi-strain equilibrium and bifurcation phenomena consistent with field observations. Ndi et al. (2024) [8] applied a two-serotype host-vector model to quantify the effects of ADE on epidemic persistence. They highlighted the roles of cross-immunity duration and enhancement in shaping outbreak patterns. Recently, Aguiar et al. (2024) [9] revisited a two-infection SIR-SIR model incorporating temporary immunity and disease enhancement. They uncovered codimension-2 bifurcations that serve as organizing centers for transitions between endemic and multi-strain regimes. Steindorf et al. (2022) [10] complemented this work by formulating a delay differential model that includes distributed temporary cross-immunity and ADE. They used Lyapunov functionals to explain global stability conditions and periodic outbreak dynamics.

Recent experimental evidence further indicates that multi-serotype dengue dynamics are not confined to the human host. Phanthanawiboon et al. [11] reported DENV co-infection in field-caught mosquitoes, including mosquitoes carrying multiple DENV serotypes. These observations provide direct empirical motivation for explicitly representing DENV-DENV co-infection within the vector population. Nevertheless, how the serotype distribution among single- and co-infected mosquitoes influences subsequent strain-specific infections in humans remains incompletely understood. A mathematical host-vector framework that explicitly tracks mosquito co-infection can therefore provide a useful mechanistic basis for investigating this link between vector serotype composition and human infection dynamics.

In this study, we consider a two-strain mathematical model of dengue fever. This deterministic, compartmental model describes the transmission dynamics of two interacting serotypes of the dengue virus within a host-vector framework. The model is established under the classical immunological assumption, which holds that infection with one serotype results in lifelong immunity to that serotype

but only temporary cross-immunity to others. While this long-standing assumption has formed the foundation for the majority of compartmental models, recent research has started to challenge the assumption of lifelong homotypic protection by documenting virologically confirmed homotypic reinfections [12, 13]. In this study, we adhere to the classical lifelong homotypic immunity formulation as a baseline framework.

Individuals are at increased risk of developing severe disease upon secondary infection with a different serotype due to ADE [14, 15]. Based on this concept, we present a two-strain mathematical model of dengue fever that explicitly distinguishes between primary and secondary infection classes in humans and DENV–DENV co-infection states in mosquitoes. Our framework takes into account temporary cross-immunity, immune enhancement with distinct primary and secondary infection rates α and σ , disease-induced mortality during secondary infection (δ), and vector co-infection compartments (V_{12}). In particular, the compartment V_{12} explicitly represents mosquitoes carrying both modeled serotypes and permits such co-infected vectors to contribute to subsequent strain-specific transmission to humans. The model allows us to rigorously derive the basic reproduction number $\mathcal{R}_0$, analyze the one-strain and two-strain endemic equilibria, and investigate bifurcation phenomena, including backward bifurcation, under ADE conditions ($\sigma > \alpha$).

Compared to earlier models, our formulation offers significant improvements that address biological realism and mathematical tractability. First, a distinguishing feature of the present formulation is the explicit representation of DENV–DENV co-infection in the mosquito population through the compartment V_{12} . Rather than restricting infected vectors to mutually exclusive strain-specific classes, the model permits mosquitoes infected with one serotype to acquire the heterologous serotype and subsequently contribute to transmission of either strain. This structure is motivated by recent field evidence of multi-serotype DENV infection in mosquitoes [11] and provides a mechanistic link through which the serotype composition of the vector population may influence primary and secondary infection pathways in humans. Second, the model mechanistically incorporates ADE by distinguishing between primary and secondary infection pathways with different transmission parameters, denoted by α and σ , respectively. This approach allows for a more realistic exploration of how immune history affects susceptibility and transmission risk during secondary infection events. Third, the model captures waning cross-immunity by introducing intermediate compartments, S_1 and S_2 , for partially susceptible individuals who transition from a recovered state back to susceptibility for heterologous strains [16]. This structure better reflects the immunological processes observed in empirical dengue studies. Finally, the model supports rigorous bifurcation analysis and numerical continuation methods. These methods are essential tools for identifying thresholds, bifurcation points, and regions of multi-stability in the system's parameter space. This structured modeling framework aligns with and extends previous literature demonstrating how temporary immunity and ADE interact to shape dengue dynamics. Our primary objective is to clarify the parameter conditions under which phenomena such as multi-strain coexistence, backward bifurcation, and complex epidemic oscillations occur. The explicit mosquito co-infection structure additionally establishes a framework for examining how multi-serotype circulation within the vector population interacts with these nonlinear epidemic mechanisms. These insights are critical for developing control strategies and public health interventions in dengue-endemic settings.

The remainder of the paper is organized as follows. Section 2 presents the full mathematical formulation of the two-strain host–vector system. In Section 3, we discuss model positivity, boundedness, and the disease-free equilibrium, and derive $\mathcal{R}_0$ via the next-generation matrix method. The one-

strain and two-strain endemic equilibria are analyzed in detail, followed by bifurcation and stability analysis. Section 4 presents numerical continuation results illustrating transitions between equilibria and oscillatory dynamics. Finally, Section 5 concludes with implications for dengue control strategies and future model extensions.

2. The mathematical model

In this model, we assume total human and vector populations of size N and M . The human population is divided into several compartments: susceptibles (S) individuals infected with strain 1 (I_1) or strain 2 (I_2), individuals recovered from strain 1 (R_1) or strain 2 (R_2), individuals susceptible after recovery (S_1, S_2), individuals reinfected with strain 2 after strain 1 (I_{12}), or strain 1 after strain 2 (I_{21}), and fully immune individuals (R). The mosquito population includes susceptible vectors (U), and vectors infected with strain 1 (V_1), strain 2 (V_2), or co-infected (V_{12}). The model represents primary infection with terms involving the transmission rate, α , from infectious vectors to susceptible humans. Those who recover from a primary infection I_1 or I_2 transition to the temporary immune classes R_1 and R_2 . From there, they move into the partially susceptible compartments S_1 and S_2 at rate ν . These individuals are then at risk of a secondary infection, which is represented by the compartments I_{12} and I_{21} with a transmission rate of σ , which may differ from α to reflect ADE effects. The mosquito dynamics are modeled through interactions with all infected human classes. Susceptible vectors, U , acquire infection at a rate proportional to the prevalence of human infection and transition into single or co-infected states. The co-infection compartment V_{12} represents mosquitoes that sequentially acquire the two modeled serotypes through infectious blood meals and in which heterologous superinfection is permitted. Thus, the present formulation does not explicitly model within-vector viral interference; rather, it represents the limiting case in which interference is not sufficiently strong to prevent establishment of the second serotype. This distinction is biologically relevant because experimental studies have demonstrated interference between dengue virus types in cultured mosquito cells [17]. Accordingly, V_{12} should be interpreted as a phenomenological co-infection state rather than as an explicit mechanistic representation of viral interference.

Regarding transmission from co-infected vectors, the present model assumes that a mosquito in V_{12} is independently infectious with respect to each of the two serotypes. Accordingly, V_{12} contributes to the strain-specific forces of infection for both strain 1 and strain 2, as represented by $\alpha(V_1 + V_{12})/M$ and $\alpha(V_2 + V_{12})/M$, respectively. This formulation does not imply obligatory simultaneous transmission of both serotypes during a single mosquito–human contact, nor does it assume that a co-infected mosquito randomly selects one serotype for transmission with a prescribed probability. Rather, it represents two distinct strain-specific transmission hazards generated by the same co-infected vector class. Hence, the contribution $2V_{12}$ in the total force of primary infection on fully susceptible humans arises from summing these two strain-specific hazards. The same interpretation applies to secondary infection, where V_{12} contributes to transmission of the heterologous serotype according to the infection history of the human host.

Disease-induced mortality during secondary infection is included via the parameter δ , which can be set to zero for analytic tractability. The vector infection rate β governs transmission from infected humans to mosquitoes. The schematic representation of the host–vector transmission dynamics of a two-strain pathogen is shown in Figure 1. Consistent with the independent strain-specific

transmission assumption described above, the total force of primary infection acting on susceptible humans is the sum of the two strain-specific forces, $\lambda_1 = \frac{\alpha}{M}(V_1 + V_{12})$ and $\lambda_2 = \frac{\alpha}{M}(V_2 + V_{12})$. Thus, $S(\lambda_1 + \lambda_2) = \frac{\alpha}{M}S(V_1 + V_2 + 2V_{12})$, which yields the infection-loss term appearing in Eq (2.1a). Transmission terms are formulated using standard-incidence (frequency-dependent) contact rates: infection of mosquitoes occurs at rate $\frac{\beta}{N}U(\cdot)$ and infection of humans at rate $\frac{\alpha}{M}S(\cdot)$. In the schematic diagram, Figure 1, arrows are labeled by the strain-specific infectious classes, while the common multiplicative factors S/M and U/N are omitted for readability.

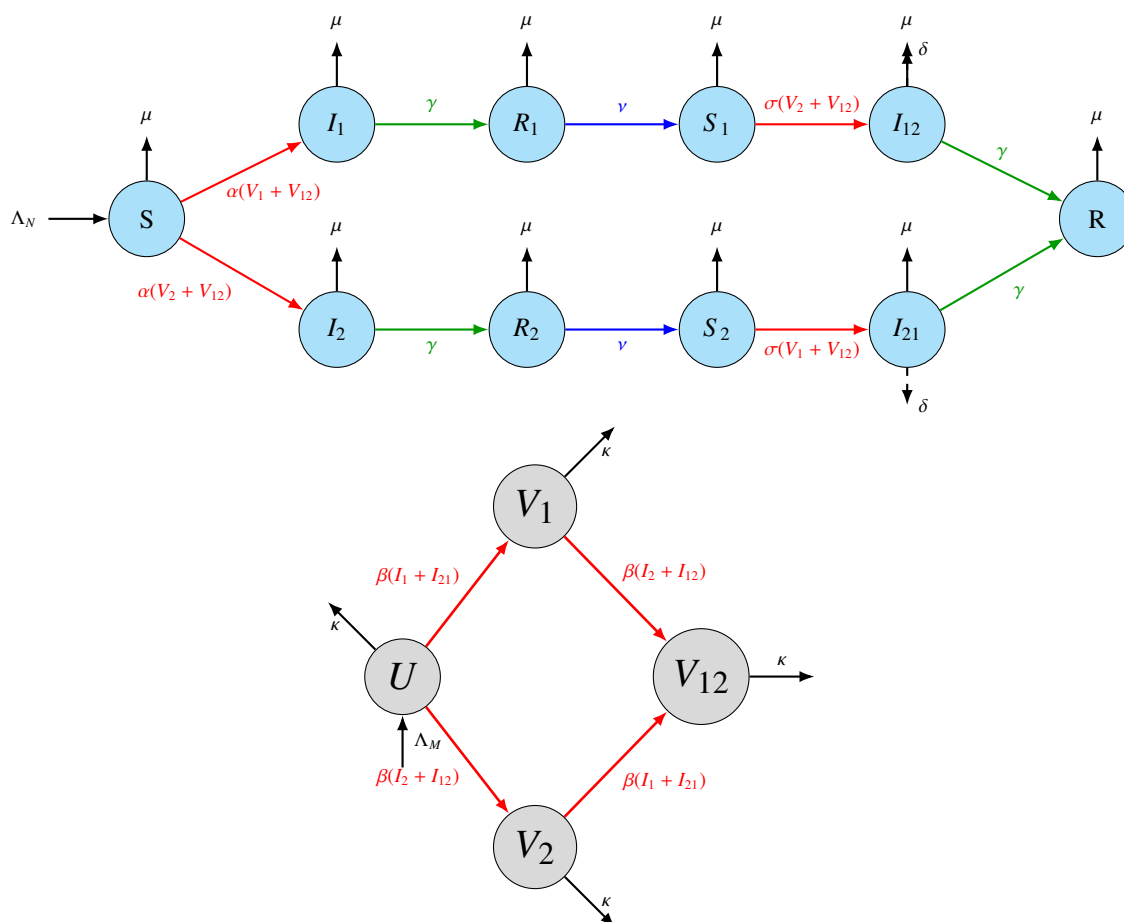


Figure 1. Schematic representation of the host–vector transmission dynamics of a two-strain pathogen. The human population N is divided into compartments based on infection status: susceptible (S), infected with strain i (I_i), recovered from strain i (R_i), partially susceptible after infection with strain i (S_i), reinfected with strain j after recovering from strain i (I_{ij}), and fully recovered (R), where $i, j \in 1, 2$. The vector population includes susceptible vectors (U) and vectors infected with strain 1, strain 2, or both (V_1, V_2, V_{12}). Arrows indicate transitions due to infection, recovery, waning immunity, and disease-induced mortality.

The model is given by

$$\dot{S} = \Lambda_N - \frac{\alpha}{M}S(V_1 + V_2 + 2V_{12}) - \mu S, \quad (2.1a)$$

$$\dot{I}_1 = \frac{\alpha}{M}S(V_1 + V_{12}) - (\gamma + \mu)I_1, \quad (2.1b)$$

$$\dot{I}_2 = \frac{\alpha}{M}S(V_2 + V_{12}) - (\gamma + \mu)I_2, \quad (2.1c)$$

$$\dot{R}_1 = \gamma I_1 - (\nu + \mu)R_1, \quad (2.1d)$$

$$\dot{R}_2 = \gamma I_2 - (\nu + \mu)R_2, \quad (2.1e)$$

$$\dot{S}_1 = \nu R_1 - \frac{\sigma}{M}S_1(V_2 + V_{12}) - \mu S_1, \quad (2.1f)$$

$$\dot{S}_2 = \nu R_2 - \frac{\sigma}{M}S_2(V_1 + V_{12}) - \mu S_2, \quad (2.1g)$$

$$\dot{I}_{12} = \frac{\sigma}{M}S_1(V_2 + V_{12}) - (\gamma + \mu)I_{12} - \delta I_{12}, \quad (2.1h)$$

$$\dot{I}_{21} = \frac{\sigma}{M}S_2(V_1 + V_{12}) - (\gamma + \mu)I_{21} - \delta I_{21}, \quad (2.1i)$$

$$\dot{R} = \gamma(I_{12} + I_{21}) - \mu R, \quad (2.1j)$$

and

$$\dot{U} = \Lambda_M - \frac{\beta}{N}U(I_1 + I_2 + I_{12} + I_{21}) - \kappa U, \quad (2.1k)$$

$$\dot{V}_1 = \frac{\beta}{N}U(I_1 + I_{21}) - \frac{\beta}{N}V_1(I_2 + I_{12}) - \kappa V_1, \quad (2.1l)$$

$$\dot{V}_2 = \frac{\beta}{N}U(I_2 + I_{12}) - \frac{\beta}{N}V_2(I_1 + I_{21}) - \kappa V_2, \quad (2.1m)$$

$$\dot{V}_{12} = \frac{\beta}{N}V_1(I_2 + I_{12}) + \frac{\beta}{N}V_2(I_1 + I_{21}) - \kappa V_{12}. \quad (2.1n)$$

The model parameters are informed by epidemiological data. The average human lifetime is taken as $\mu^{-1} = 12 \times 65$ per month, the recovery period is $\gamma^{-1} = 0.5$ per month, and the cross-immunity period is $\nu^{-1} = 9$ per month [15]. The average vector lifespan is $\kappa^{-1} = 1\text{--}1.5$ per month. Disease-induced mortality during secondary infection is represented by the rate δ . For the analytical results presented below, we set $\delta = 0$, in which case the total human population remains constant. The human infection rates α, σ for the primary and secondary infection as well as the vector infection rate β are less clear. In particular, the ADE effect is modeled by varying σ relative to α . Three biological scenarios are typically examined:

- $\sigma < \alpha$: secondary infections are less likely than primary infections, implying partial protection;
- $\sigma = \alpha$: equal risk of primary and secondary infections;
- $\sigma > \alpha$: ADE enhances susceptibility to secondary infection.

The model variables and parameters are shown in Table 1.

This model builds on earlier multi-strain and host–vector frameworks [7, 18, 19], while incorporating enhanced biological realism through explicit co-infection classes and multiple infection stages. These models are essential for understanding the nonlinear dynamics of dengue outbreaks, determining the role of ADE in epidemic amplification, and developing public health strategies, including vector control and vaccination.

Table 1. Descriptions of the variables and parameters of model (2.1).

Variable	Description
N	Total human population
M	Total vector population
S	Fully susceptible (never infected)
I_1, I_2	Primary infected with strain 1 or 2, respectively
R_1, R_2	Recovered from strain 1 or 2, with temporary immunity
S_1, S_2	Partially susceptible after immunity wanes from infection with strain 1 or 2
I_{12}, I_{21}	Secondary infected, previously recovered from strain 1 (or 2), now infected by strain 2 (or 1)
R	Fully recovered after two infections
U	Uninfected mosquitoes
V_1	Infected with strain 1
V_2	Infected with strain 2
V_{12}	Co-infected with both strains
Parameter	Description
Λ_N, Λ_M	Recruitment of humans/vectors
μ, κ	Natural death rates of humans and vectors
α	Primary infection rate from infected vectors
σ	Secondary infection rate (may differ from α)
γ	Human recovery rate
ν	Rate of loss of temporary immunity
δ	Disease-induced mortality in secondary infections
β	Transmission from infected humans to vectors

In particular, the present formulation explicitly distinguishes between primary and secondary human infections and allows mosquitoes to exist in single- or double-infected states. The inclusion of the co-infected vector class V_{12} enables the description of sequential acquisition of different serotypes by the same vector, which is an essential feature for understanding viral competition and coexistence within the mosquito population. In the present formulation, however, the transition to V_{12} does not include an explicit interference factor that reduces or prevents acquisition of the heterologous serotype. The model therefore permits coexistence of the two serotypes within a mosquito once sequential infection occurs. Experimental evidence of within-vector interference between dengue virus types [17] suggests that such interference may modify the probability of establishing a secondary vector infection. Incorporating an interference-dependent reduction in the transitions from V_1 to V_{12} and from V_2 to V_{12} would constitute a biologically meaningful extension of the present model.

From an immuno-epidemiological perspective, the compartments S_1 and S_2 play a critical role in describing individuals who have lost temporary cross-immunity and have returned to a partially susceptible state [16]. The transition from R_i to S_i at rate ν reflects the finite duration of heterologous protection following recovery from a primary infection. This process introduces a biologically relevant delay between infections, enabling the model to capture realistic inter-epidemic intervals observed in dengue-endemic regions. The secondary infection process, parameterized by the transmission rate σ , accounts for ADE effects by modifying susceptibility to reinfection. The ratio σ/α thus quantifies the degree of enhancement or protection during heterologous exposure, where $\sigma > \alpha$ corresponds to enhanced transmission due to ADE and $\sigma < \alpha$ corresponds to partial cross-protection.

The inclusion of disease-induced mortality, represented by δ , allows the model to account for severe outcomes of secondary infections such as DHF and DSS. This term introduces an additional loss pathway for infected individuals and provides a means to assess how mortality alters disease persistence and endemic equilibria. Although δ is typically small in magnitude, its impact on model dynamics can be nontrivial, especially when combined with ADE-driven secondary infection amplification.

Overall, the system (2.1) provides a unified mechanistic description of multi-strain dengue dynamics that links immunological processes at the individual level (temporary cross-immunity, ADE, secondary mortality) with transmission mechanisms at the population level (vector co-infection, human reinfection). This structure not only facilitates rigorous mathematical analysis of equilibria and bifurcation behavior but also supports biologically meaningful interpretation of parameters. The model thus serves as a comprehensive foundation for exploring threshold phenomena, the coexistence of dengue strains, and oscillatory epidemic patterns under varying epidemiological and environmental conditions.

3. Analytical results

For any initial values at a given time, the right-hand side of system (2.1a)–(2.1n) has a non-negative solution for $t > 0$. Let $N(t) = S(t) + I_1(t) + I_2(t) + R_1(t) + R_2(t) + S_1(t) + S_2(t) + I_{12}(t) + I_{21}(t) + R(t)$ and $M(t) = U(t) + V_1(t) + V_2(t) + V_{12}(t)$. Then

$$\frac{dN}{dt} = \Lambda_N - \mu N - \delta(I_{12} + I_{21}) \leq \Lambda_N - \mu N, \quad (3.1)$$

$$\frac{dM}{dt} = \Lambda_M - \kappa M. \quad (3.2)$$

This implies that N and M remain bounded on any finite time interval. Using differential inequalities for N and M leads to

$$\limsup_{t \rightarrow \infty} N(t) \leq \frac{\Lambda_N}{\mu} \quad \text{and} \quad \limsup_{t \rightarrow \infty} M(t) \leq \frac{\Lambda_M}{\kappa}.$$

3.1. Disease-free equilibrium (DFE)

The trivial equilibrium of model (2.1a)–(2.1n) is $E^{\text{DF}} = (S^{\text{DF}}, 0, 0, 0, 0, 0, 0, 0, 0, 0, U^{\text{DF}}, 0, 0, 0)$ with $S^{\text{DF}} = \Lambda_N/\mu$ and $U^{\text{DF}} = \Lambda_M/\kappa$. The superscript *DF* denotes the disease-free equilibrium. Linearization around the disease-free steady state leads to the Jacobian matrix, where the Jacobian can be re-expressed as

$$\mathcal{J} = [\mathcal{M}_{14 \times 7} \quad \mathcal{N}_{14 \times 7}]$$

with

$$\mathcal{M} = \begin{bmatrix} -\mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\bar{\gamma} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\bar{\gamma} & 0 & 0 & 0 & 0 \\ 0 & \gamma & 0 & -\bar{\nu} & 0 & 0 & 0 \\ 0 & 0 & \gamma & 0 & -\bar{\nu} & 0 & 0 \\ 0 & 0 & 0 & \nu & 0 & -\mu & 0 \\ 0 & 0 & 0 & 0 & \nu & 0 & -\mu \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\frac{\beta U^{\text{DF}}}{S^{\text{DF}}} & -\frac{\beta U^{\text{DF}}}{S^{\text{DF}}} & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta U^{\text{DF}}}{S^{\text{DF}}} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{\beta U^{\text{DF}}}{S^{\text{DF}}} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

and

$$\mathcal{N} = \begin{bmatrix} 0 & 0 & 0 & 0 & -\frac{\alpha S^{\text{DF}}}{U^{\text{DF}}} & -\frac{\alpha S^{\text{DF}}}{U^{\text{DF}}} & -\frac{2\alpha S^{\text{DF}}}{U^{\text{DF}}} \\ 0 & 0 & 0 & 0 & \frac{\alpha S^{\text{DF}}}{U^{\text{DF}}} & 0 & \frac{\alpha S^{\text{DF}}}{U^{\text{DF}}} \\ 0 & 0 & 0 & 0 & 0 & \frac{\alpha S^{\text{DF}}}{U^{\text{DF}}} & \frac{\alpha S^{\text{DF}}}{U^{\text{DF}}} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ -\bar{\delta} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -\bar{\delta} & 0 & 0 & 0 & 0 & 0 \\ \gamma & \gamma & -\mu & 0 & 0 & 0 & 0 \\ -\frac{\beta U^{\text{DF}}}{S^{\text{DF}}} & -\frac{\beta U^{\text{DF}}}{S^{\text{DF}}} & 0 & -\kappa & 0 & 0 & 0 \\ 0 & \frac{\beta U^{\text{DF}}}{S^{\text{DF}}} & 0 & 0 & -\kappa & 0 & 0 \\ \frac{\beta U^{\text{DF}}}{S^{\text{DF}}} & 0 & 0 & 0 & 0 & -\kappa & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\kappa \end{bmatrix},$$

where $\bar{\nu} = \nu + \mu$, $\bar{\delta} = \gamma + \mu + \delta$. Here the characteristic polynomial $\det(J|_{E^{\text{DF}}} - \Sigma I_{14}) = 0$, where I_{14} denotes the 14×14 identity matrix. This polynomial results in eigenvalues corresponding to the disease-free steady state: $-\mu$, $-\kappa$, $-(\mu + \nu)$, $-(\mu + \gamma + \delta)$ (counted with multiplicity), and

$$\Sigma = -\frac{1}{2} \left((\gamma + \kappa + \mu) \pm \sqrt{(\gamma + \kappa + \mu)^2 - 4(\gamma\kappa + \mu\kappa - \alpha\beta)} \right),$$

which are the roots of the polynomial

$$\Sigma^2 + (\gamma + \kappa + \mu)\Sigma + \gamma\kappa + \mu\kappa - \alpha\beta = 0. \quad (3.3)$$

The linearization around a generic steady state is given in Section 5. The polynomial given in Eq (3.3) has a positive real root if the free term is negative, namely,

$$\frac{\alpha\beta}{\kappa(\gamma + \mu)} > 1.$$

This relationship is associated with the basic reproduction number, which will be formally derived in the following section.

3.2. Basic reproduction number

In this section, following the ideas given in [20], the basic reproduction number for (2.1a)–(2.1n) is derived using the next generation matrix method. For the disease free state, the matrix F associated with new infections and the matrix V representing the remaining expressions are given by

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\alpha S^{\text{DF}}}{M^{\text{DF}}} & 0 & \frac{\alpha S^{\text{DF}}}{M^{\text{DF}}} \\ 0 & 0 & 0 & 0 & 0 & \frac{\alpha S^{\text{DF}}}{M^{\text{DF}}} & \frac{\alpha S^{\text{DF}}}{M^{\text{DF}}} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta U^{\text{DF}}}{N^{\text{DF}}} & 0 & 0 & \frac{\beta U^{\text{DF}}}{N^{\text{DF}}} & 0 & 0 & 0 \\ 0 & \frac{\beta U^{\text{DF}}}{N^{\text{DF}}} & \frac{\beta U^{\text{DF}}}{N^{\text{DF}}} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix},$$

$$V = -\mathcal{V} = \begin{bmatrix} \gamma + \mu & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma + \mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma + \mu + \delta & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma + \mu + \delta & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \kappa & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \kappa & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \kappa \end{bmatrix},$$

where $M^{\text{DF}} = U^{\text{DF}}$, $N^{\text{DF}} = S^{\text{DF}}$, $N^{\text{DF}} = \Lambda_N/\mu$, and $M^{\text{DF}} = \Lambda_M/\kappa$. Since the inverse of a diagonal matrix is given by replacing the main diagonal components of the matrix with their reciprocals, we have

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\alpha S^{\text{DF}}}{\kappa M^{\text{DF}}} & 0 & \frac{\alpha S^{\text{DF}}}{\kappa M^{\text{DF}}} \\ 0 & 0 & 0 & 0 & 0 & \frac{\alpha S^{\text{DF}}}{\kappa M^{\text{DF}}} & \frac{\alpha S^{\text{DF}}}{\kappa M^{\text{DF}}} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\beta U^{\text{DF}}}{(\gamma + \mu)N^{\text{DF}}} & 0 & 0 & \frac{\beta U^{\text{DF}}}{(\gamma + \mu + \delta)N^{\text{DF}}} & 0 & 0 & 0 \\ 0 & \frac{\beta U^{\text{DF}}}{(\gamma + \mu)N^{\text{DF}}} & \frac{\beta U^{\text{DF}}}{(\gamma + \mu + \delta)N^{\text{DF}}} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

The reproduction number $\mathcal{R}_0$ is defined to be the spectral radius of the matrix FV^{-1} . The characteristic equation of this matrix is given by $\det(FV^{-1} - PI_7) = 0$, where I_7 denotes the 7×7 identity matrix and P denotes the corresponding eigenvalues, leading to

$$(P^4 - 2A_1A_2P^2 + A_1^2A_2^2)P^3 = 0, \quad (3.4)$$

where $A_1 = \frac{\alpha S^{\text{DF}}}{\kappa M^{\text{DF}}}$ and $A_2 = \frac{\beta U^{\text{DF}}}{(\gamma + \mu)N^{\text{DF}}}$. The nonzero roots of Eq (3.4) are $P_{1,2} = \pm \sqrt{A_1A_2}$. Hence the basic reproduction number is associated with the dominant eigenvalue P_1 . Replacing $N^{\text{DF}} = S^{\text{DF}} = \frac{\Lambda_N}{\mu}$ and $M^{\text{DF}} = U^{\text{DF}} = \frac{\Lambda_M}{\kappa}$ leads to

$$\mathcal{R}_0 = \sqrt{A_1A_2} = \left(\frac{\alpha\beta}{\kappa(\gamma + \mu)} \right)^{1/2}. \quad (3.5)$$

Theorem 1. *The basic reproduction number $\mathcal{R}_0$ of system (2.1) is given by*

$$\mathcal{R}_0 = \left(\frac{\alpha\beta}{\kappa(\gamma + \mu)} \right)^{1/2}.$$

The disease-free equilibrium is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$ [20].

To quantify the local influence of the principal intervention-related parameters on the epidemic threshold, we consider the normalized forward sensitivity index [21–23],

$$\Upsilon_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \frac{p}{\mathcal{R}_0}, \quad p \in \{\alpha, \beta, \kappa, \gamma\},$$

with the remaining parameters held fixed. Since $\mathcal{R}_0 = \left(\frac{\alpha\beta}{\kappa(\gamma + \mu)} \right)^{1/2}$, direct differentiation yields

$$\Upsilon_{\alpha}^{\mathcal{R}_0} = \Upsilon_{\beta}^{\mathcal{R}_0} = \frac{1}{2}, \quad \Upsilon_{\kappa}^{\mathcal{R}_0} = -\frac{1}{2}, \quad \Upsilon_{\gamma}^{\mathcal{R}_0} = -\frac{\gamma}{2(\gamma + \mu)}.$$

Hence, locally, a relative increase in either transmission parameter α or β produces one-half of that relative increase in $\mathcal{R}_0$, whereas a relative increase in the vector mortality rate κ produces, to first order, a relative decrease in $\mathcal{R}_0$ of one-half the same magnitude. Increasing the human recovery rate γ , equivalently shortening the infectious period, also decreases $\mathcal{R}_0$. Since $\gamma, \mu > 0$, it follows that

$$-\frac{1}{2} < \Upsilon_{\gamma}^{\mathcal{R}_0} < 0, \quad \Upsilon_{\gamma}^{\mathcal{R}_0} \longrightarrow -\frac{1}{2} \quad \text{as} \quad \frac{\gamma}{\mu} \longrightarrow \infty.$$

Thus, within this local sensitivity framework, reducing human–vector transmission, increasing vector mortality, and shortening the human infectious period act directly to decrease the epidemic threshold.

3.3. One-strain equilibrium

Let E^{S1} denote the equilibrium where the second dengue strain is absent, namely,

$$E^{S1} = (S^{S1}, I_1^{S1}, 0, R_1^{S1}, 0, S_1^{S1}, 0, 0, 0, 0, U^{S1}, V_1^{S1}, 0, 0).$$

In this context, model (2.1a)–(2.1n) leads to

$$I_1^{S1} = \frac{\alpha}{(\gamma + \mu)M^{S1}} S^{S1} V_1^{S1}, \quad (3.6)$$

$$R_1^{S1} = \frac{\gamma}{(\gamma + \mu)} I_1^{S1} = \frac{\gamma\alpha}{(\gamma + \mu)(\gamma + \mu)M^{S1}} S^{S1} V_1^{S1}, \quad (3.7)$$

$$S_1^{S1} = \frac{\nu}{\mu} R_1^{S1} = \frac{\nu\gamma\alpha}{\mu(\gamma + \mu)(\gamma + \mu)M^{S1}} S^{S1} V_1^{S1}, \quad (3.8)$$

with $N^{S1} = S^{S1} + I_1^{S1} + R_1^{S1} + S_1^{S1}$ and $M^{S1} = U^{S1} + V_1^{S1}$. Letting $X_1 = S^{S1}$ and $X_2 = S^{S1} V_1^{S1}$, we obtain

$$\begin{aligned} N^{S1} &= X_1 + \frac{\alpha}{(\gamma + \mu)M^{S1}} \left(1 + \frac{\gamma}{(\gamma + \mu)} + \frac{\nu\gamma}{\mu(\gamma + \mu)} \right) X_2, \\ &= X_1 + \frac{\alpha}{\mu M^{S1}} X_2. \end{aligned} \quad (3.9)$$

Using Eqs (2.11) and (3.6), one obtains

$$\kappa = \frac{\beta}{N^{S1}} \left(\frac{\alpha}{(\gamma + \mu)} X_1 - \frac{\alpha}{(\gamma + \mu)M^{S1}} X_2 \right). \quad (3.10)$$

Solving (3.9) and (3.10) leads to

$$X_1 = \frac{N^{S1}}{\alpha + \mu} \left(\mu + \frac{\kappa(\gamma + \mu)}{\beta} \right), \quad (3.11)$$

$$X_2 = \frac{\mu M^{S1} N^{S1}}{\alpha + \mu} \left(1 - \frac{\kappa(\gamma + \mu)}{\alpha\beta} \right). \quad (3.12)$$

Therefore considering $\mathcal{R}_0 = \left(\frac{\alpha\beta}{\kappa(\gamma + \mu)} \right)^{1/2}$ and using Eqs (3.6)–(3.8), the non-zero components for the human population are

$$S^{S1} = \frac{N^{S1}}{\alpha + \mu} \left(\mu + \frac{\kappa(\gamma + \mu)}{\beta} \right) = \frac{\alpha N^{S1}}{\alpha + \mu} \left(\frac{\mu}{\alpha} + \frac{1}{\mathcal{R}_0^2} \right), \quad (3.13)$$

$$I_1^{S1} = \frac{\alpha\mu N^{S1}}{(\gamma + \mu)(\alpha + \mu)} \left(1 - \frac{1}{\mathcal{R}_0^2}\right), \quad (3.14)$$

$$R_1^{S1} = \frac{\alpha\mu\gamma N^{S1}}{(\nu + \mu)(\gamma + \mu)(\alpha + \mu)} \left(1 - \frac{1}{\mathcal{R}_0^2}\right), \quad (3.15)$$

$$S_1^{S1} = \frac{\alpha\gamma\nu N^{S1}}{(\nu + \mu)(\gamma + \mu)(\alpha + \mu)} \left(1 - \frac{1}{\mathcal{R}_0^2}\right). \quad (3.16)$$

On the other hand, using Eqs (2.1k) and (3.14), one obtains

$$U^{S1} = \frac{\Lambda_M}{\kappa + \varphi}, \quad \varphi = \frac{\alpha\beta\mu}{\bar{\gamma}\bar{\alpha}} \left(1 - \frac{1}{\mathcal{R}_0^2}\right), \quad (3.17)$$

where $\bar{\gamma} = \gamma + \mu$ and $\bar{\alpha} = \alpha + \mu$. Besides, using Eq (3.14) in (2.1l) leads to

$$V_1^{S1} = \frac{\Lambda_M\varphi}{\kappa(\kappa + \varphi)} \quad (3.18)$$

and thus

$$S^{S1} = \frac{\Lambda_N(\kappa + \varphi)}{(\mu(\kappa + \varphi) + \alpha\varphi)}, \quad I_1^{S1} = \frac{\Lambda_N\alpha\varphi}{\bar{\gamma}(\mu(\kappa + \varphi) + \alpha\varphi)}, \quad R_1^{S1} = \frac{\gamma}{\bar{\nu}} I_1^{S1}, \quad S_1^{S1} = \frac{\nu\gamma}{\bar{\nu}\mu} I_1^{S1}, \quad (3.19)$$

with $\bar{\nu} = \nu + \mu$. Similar calculations can be performed for the equilibria where the second dengue strain is absent (E^{S2}). Note that the components of one-strain equilibrium E^{S1} and E^{S2} are positive if and only if $\mathcal{R}_0 > 1$. The stability of a one-strain equilibrium with respect to the introduction of the heterologous strain is determined by the linearized dynamics in the corresponding invasion subspace. In particular, for E^{S1} , the relevant perturbations are those associated with strain 2 and with secondary infection following strain 1. Thus, heterologous invasion is possible precisely when the dominant eigenvalue of this invasion subsystem has a positive real part. The parameter σ enters this subsystem through the secondary-infection pathway from S_1 to I_{12} and therefore directly modifies the invasion threshold. Consequently, the condition $\sigma > \alpha$ should be interpreted as the antibody-dependent enhancement regime, in which heterologous secondary transmission is enhanced relative to primary transmission, rather than, by itself, as a universal stability threshold for the one-strain equilibrium. The same conclusion holds, by symmetry, for the invasion of strain 1 about E^{S2} .

3.4. Two-strain equilibrium

Now we consider that both virus strains are present in the population. Denoting any arbitrary two-strain equilibrium point (E^F) of model (2.1a)–(2.1n) as

$$E^F = (S^F, I_1^F, I_2^F, R_1^F, R_2^F, S_1^F, S_2^F, I_{12}^F, I_{21}^F, R^F, U^F, V_1^F, V_2^F, V_{12}^F)$$

and considering

$$y_k = \frac{\alpha}{M^F} V_k^F, \quad k = 1, 2, 12,$$

we obtain

$$S^F = \frac{\Lambda_N}{\mu + y_1 + y_2 + 2y_{12}}.$$

In the two-strain scenario, the total human and vector populations are represented by N^F and M^F . Assuming that a two-strain equilibrium emerges with the assumptions $y_1 = y_2 = y$ and $x = y + y_{12}$, this leads to

$$\begin{aligned} I_1^F &= I_2^F = \frac{\Lambda_N x}{\bar{\gamma}(\mu + 2x)}, \\ R_1^F &= R_2^F = \frac{\gamma \Lambda_N x}{\bar{\nu} \bar{\gamma}(\mu + 2x)}, \\ S_1^F &= S_2^F = \frac{\nu \gamma \Lambda_N x}{\bar{\nu} \bar{\gamma}(\mu + 2x)(\mu + px)}, \\ I_{12}^F &= I_{21}^F = \frac{p \nu \gamma \Lambda_N x^2}{\bar{\nu} \bar{\gamma} \bar{\delta}(\mu + 2x)(\mu + px)}, \\ R^F &= \frac{2 p \nu \gamma^2 \Lambda_N x^2}{\mu \bar{\nu} \bar{\gamma} \bar{\delta}(\mu + 2x)(\mu + px)}, \quad U^F = \frac{\Lambda_M}{\kappa + 2 \frac{\beta}{N^F} f(x)}, \end{aligned}$$

where $\bar{\nu} = \nu + \mu$, $\bar{\gamma} = \gamma + \mu$, $\bar{\delta} = \delta + \gamma + \mu$, and

$$f(x) = \frac{\Lambda_N x}{\bar{\gamma}(\mu + 2x)} \left[1 + \frac{p q x}{\mu + p x} \right],$$

with $p = \frac{\sigma}{\alpha}$ and $q = \frac{\nu \gamma}{\bar{\nu} \bar{\delta}}$.

Using Eqs (2.11) and (2.1n) leads to

$$\frac{\beta}{N^F} f(x) \left[\frac{\Lambda_M}{\kappa + \frac{2\beta}{N^F} f(x)} - \frac{M^F}{\alpha} y \right] - \frac{\kappa M^F}{\alpha} y = 0, \quad (3.20)$$

$$\frac{2\beta}{N^F} y f(x) - \kappa(x - y) = 0. \quad (3.21)$$

Solving Eqs (3.20) and (3.21) for the x and y variables, it is possible to obtain y and $y_{12} = x - y$. Therefore, a two-strain equilibrium (with the assumption of $y_1 = y_2$) can be obtained as a function of M^F and N^F . Note that there may exist various equilibria where $y_1 \neq y_2$, yet the complexity of the model does not allow further analytical investigation.

3.5. Backward bifurcation analysis

Using center manifold theory, the backward bifurcation analysis can be carried out [24,25]. Following the ideas given in Theorem 4.1 in [24], the following change of variables is considered:

$$(S, I_1, I_2, R_1, R_2, S_1, S_2, I_{12}, I_{21}, R, U, V_1, V_2, V_{12}) = (x_1, x_2, \dots, x_{14}).$$

Considering that $\mathcal{X} = (x_1, x_2, \dots, x_{14})^T$, the model can be rewritten as

$$\frac{d\mathcal{X}}{dt} = F(\mathcal{X}, p),$$

where $F = (F_1, F_2, \dots, F_{14})$ and p denotes the parameter set. Now, consider the case for which $\mathcal{R}_0 = 1$ and choose α as the bifurcation parameter. Solving the parameter α from $\mathcal{R}_0 = 1$ leads to

$$\alpha^* = \frac{\kappa(\gamma + \mu)}{\beta}.$$

Using center manifold theory, the dynamics of the system can be analyzed in the vicinity of $\alpha = \alpha^*$. In this context, it is necessary to compute the right and left eigenvectors of the Jacobian evaluated at the disease-free equilibrium. We denote the right eigenvector of $J|_{E^{DF}}$ as $\mathbf{w} = (\omega_1, \omega_2, \dots, \omega_{14})^T$, where

$$\begin{aligned}\omega_1 &= -\frac{\bar{\gamma}}{\mu}(\omega_2 + \omega_3), & \omega_4 &= \frac{\gamma}{\bar{\nu}}\omega_2, & \omega_5 &= \frac{\gamma}{\bar{\nu}}\omega_3, & \omega_6 &= \frac{\nu}{\mu}\omega_4 = \frac{\nu\gamma}{\mu\bar{\nu}}\omega_2, \\ \omega_7 &= \frac{\nu}{\mu}\omega_5 = \frac{\nu\gamma}{\mu\bar{\nu}}\omega_3, & \omega_8 &= 0, & \omega_9 &= 0, & \omega_{10} &= 0, & \omega_{11} &= -\frac{\bar{\gamma}U^{DF}}{\alpha^*S^{DF}}(\omega_2 + \omega_3), \\ \omega_{12} &= \frac{\bar{\gamma}U^{DF}}{\alpha^*S^{DF}}\omega_2, & \omega_{13} &= \frac{\bar{\gamma}U^{DF}}{\alpha^*S^{DF}}\omega_3, & \omega_{14} &= 0, & \text{with } \omega_2 &> 0, & \omega_3 &> 0.\end{aligned}\quad (3.22)$$

In a similar way, the left eigenvector of $J|_{E^{DF}}$ can be denoted as $\mathbf{v} = (v_1, v_2, \dots, v_{14})$:

$$\begin{aligned}v_1 &= 0, & v_4 &= 0, & v_5 &= 0, & v_6 &= 0, & v_7 &= 0, & v_8 &= \frac{\bar{\gamma}}{\bar{\delta}}v_3, & v_9 &= \frac{\bar{\gamma}}{\bar{\delta}}v_2, & v_{10} &= 0, & v_{11} &= 0, \\ v_{12} &= \frac{\alpha^*S^{DF}}{\kappa U^{DF}}v_2, & v_{13} &= \frac{\alpha^*S^{DF}}{\kappa U^{DF}}v_3, & v_{14} &= \frac{\alpha^*S^{DF}}{\kappa U^{DF}}(v_2 + v_3), & \text{with } v_2 &> 0, & v_3 &> 0,\end{aligned}\quad (3.23)$$

where $\bar{\gamma} = \gamma + \mu$, $\bar{\delta} = \delta + \gamma + \mu$. Thus one obtains

$$\mathbf{w} \cdot \mathbf{v} = \left(1 + \frac{\bar{\gamma}}{\kappa}\right)(v_2\omega_2 + v_3\omega_3) > 0.$$

Consider a to be the bifurcation coefficient presented in [20]. Rewriting model (2.1a)–(2.1n) in the form of $\dot{x}_i = F_i(x_i)$, $i = \{1, 2, \dots, 14\}$, this coefficient is given by

$$a = \sum_{k,i,j=1}^{14} v_k \omega_i \omega_j \frac{\partial^2 F_k}{\partial x_i \partial x_j}(0, 0). \quad (3.24)$$

Using Eqs (3.23) and (3.24), it becomes

$$\begin{aligned}a &= v_2 \sum_{i,j=1}^{14} \omega_i \omega_j \frac{\partial^2 F_2}{\partial x_i \partial x_j} + v_3 \sum_{i,j=1}^{14} \omega_i \omega_j \frac{\partial^2 F_3}{\partial x_i \partial x_j} + \frac{\bar{\gamma}}{\bar{\delta}} v_3 \sum_{i,j=1}^{14} \omega_i \omega_j \frac{\partial^2 F_8}{\partial x_i \partial x_j} \\ &+ \frac{\bar{\gamma}}{\bar{\delta}} v_2 \sum_{i,j=1}^{14} \omega_i \omega_j \frac{\partial^2 F_9}{\partial x_i \partial x_j} + \frac{\alpha^*S^{DF}}{\kappa U^{DF}} v_2 \sum_{i,j=1}^{14} \omega_i \omega_j \frac{\partial^2 F_{12}}{\partial x_i \partial x_j} + \frac{\alpha^*S^{DF}}{\kappa U^{DF}} v_3 \sum_{i,j=1}^{14} \omega_i \omega_j \frac{\partial^2 F_{13}}{\partial x_i \partial x_j} \\ &+ \frac{\alpha^*S^{DF}}{\kappa U^{DF}}(v_2 + v_3) \sum_{i,j=1}^{14} \omega_i \omega_j \frac{\partial^2 F_{14}}{\partial x_i \partial x_j},\end{aligned}\quad (3.25)$$

where

$$\begin{aligned}F_2 &= \frac{\alpha^* x_1 (x_{12} + x_{14})}{x_{11} + x_{12} + x_{13} + x_{14}} - \bar{\gamma} x_2, \\ F_3 &= \frac{\alpha^* x_1 (x_{13} + x_{14})}{x_{11} + x_{12} + x_{13} + x_{14}} - \bar{\gamma} x_3, \\ F_8 &= \frac{\sigma x_6 (x_{13} + x_{14})}{x_{11} + x_{12} + x_{13} + x_{14}} - (\bar{\gamma} + \delta) x_8,\end{aligned}$$

$$\begin{aligned}
F_9 &= \frac{\sigma x_7(x_{12} + x_{14})}{x_{11} + x_{12} + x_{13} + x_{14}} - (\bar{\gamma} + \delta)x_9, \\
F_{12} &= \frac{\beta [x_{11}(x_2 + x_9) - x_{12}(x_3 + x_8)]}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8 + x_9 + x_{10}} - \kappa x_{12}, \\
F_{13} &= \frac{\beta [x_{11}(x_3 + x_8) - x_{13}(x_2 + x_9)]}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8 + x_9 + x_{10}} - \kappa x_{13}, \\
F_{14} &= \frac{\beta [x_{12}(x_3 + x_8) + x_{13}(x_2 + x_9)]}{x_1 + x_2 + x_3 + x_4 + x_5 + x_6 + x_7 + x_8 + x_9 + x_{10}} - \kappa x_{14}.
\end{aligned}$$

Using Eq (3.22), we eventually obtain the bifurcation coefficient evaluated at the disease-free equilibrium as

$$a|_{E^{DF}} = \frac{2\bar{\gamma}}{S^{DF}} \left(-A_1(v_2\omega_2 + v_3\omega_3)(\omega_2 + \omega_3) + \frac{A_2}{\alpha^*}(v_2 + v_3)\omega_2\omega_3 \right), \quad (3.26)$$

with $A_1 = \frac{\beta}{\kappa} + \frac{\bar{\gamma}}{\mu}$, $A_2 = \frac{\sigma\gamma v\bar{\gamma}}{v\delta\mu}$. Here we want to determine the sign of a . Since we have assume that $v_2 > 0$, $v_3 > 0$, $\omega_2 > 0$, and $\omega_3 > 0$, the bifurcation coefficient a is found to be positive if

$$\alpha^* < \frac{A_2(v_2 + v_3)\omega_2\omega_3}{A_1(v_2\omega_2 + v_3\omega_3)(\omega_2 + \omega_3)} = \alpha_c. \quad (3.27)$$

In the notation given in [20], one also obtains the coefficient

$$b = \sum_{k,i=1}^{14} \omega_k \omega_i \frac{\partial^2 f_k}{\partial x_i \partial \alpha}(0, 0), \quad (3.28)$$

which evaluated, at the disease-free equilibrium leads to

$$b|_{E^{DF}} = \frac{\bar{\gamma}^2}{\alpha^*\mu}(\omega_2 + \omega_3)^2 + \frac{\bar{\gamma}}{\alpha^*}(\omega_2^2 + \omega_3^2) > 0. \quad (3.29)$$

The model exhibits backward bifurcation whenever $\alpha^* < \alpha_c$ in Eq (3.27).

Table 2. The values of the parameters (monthly units) in our model 2.1 are based on information about dengue epidemiology.

Parameter	Value (month ⁻¹)	Notes	Reference
Λ_N	12.8	Recruitment rate of humans	[26]
Λ_M	10^5	Recruitment rate of vectors, and 28,000 per week	[27]
μ	0.00128	Avg. human lifespan ≈ 65 –70 yrs	[15]
γ	2.0	Avg. infectious period ≈ 15 days, $\gamma^{-1} = 0.5$ month	[15, 28]
ν	0.111	Cross-immunity period	[5, 15, 29, 30]
δ	0.01	Disease-induced mortality (rare, severe DHF/DSS)	[14, 31]
κ	1	Vector lifespan, $\kappa \in [0.67, 1.0]$	[28, 32]
α	0.39	Primary infection rate from infected vectors	[28, 33, 34]
σ	0.1–0.8	Secondary infection rate (ADE scenarios)	[15, 28, 34]
β	6	Vector infection rate from humans	[28, 35]

Note that in the parameter list shown in Table 2, if we take $\delta = 0$, we would have a quasi-constant human population $N = \frac{\Lambda_N}{\mu}$, as well as a quasi-constant mosquito population $M = \frac{\Lambda_M}{\kappa}$, see (3.1) and (3.2). Our numerical investigation, however, considers the positive disease-induced mortality displayed in the table.

Importantly, our analytical results are derived under the condition that $a > 0$ in Eq (3.26), which allows for the possibility of backward bifurcation and bistability, yet evaluating these coefficients with our baseline parameter set given in Table 2 yields $a < 0$, see Section 4. Therefore, the numerical continuation results in a standard forward bifurcation at $\alpha \approx 0.33355$.

4. Numerical investigation of the two-strain dengue epidemics model

In this section, we perform a numerical investigation of the dynamical behavior of the two-strain dengue model (2.1), with emphasis on how qualitative outcomes change under variations of key parameters. The computations combine direct time integration of the ODE system with path-following (continuation) methods implemented in the software package COCO [36]. COCO is a MATLAB-based platform for numerical continuation and bifurcation analysis of parametrized smooth ODEs; in spirit and functionality, it is comparable to established tools such as AUTO [37] and MATCONT [38]. Throughout, we keep the model structure and parameter meanings as defined previously (see Sections 2 and 3), and use the baseline parameter values summarized in Table 2, unless explicitly stated otherwise.

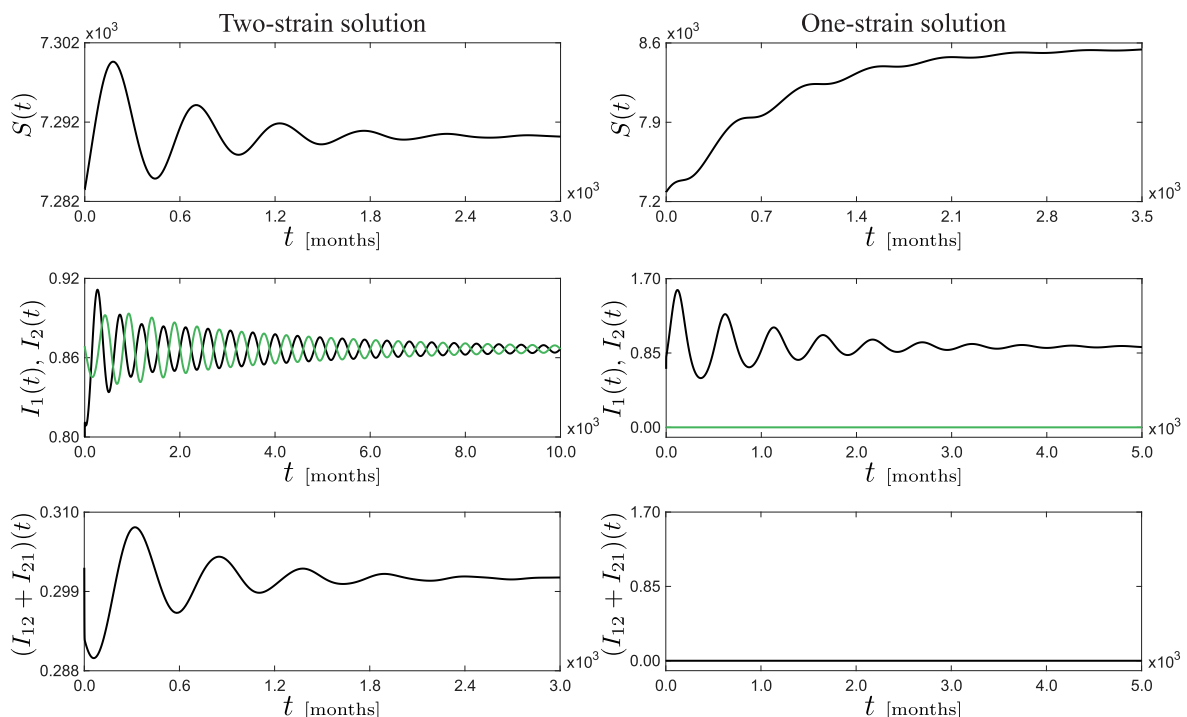


Figure 2. One- and two-strain transient responses of the dengue epidemics model (2.1), computed for the parameter values given in Table 2, with $\sigma = 0.45$. In this picture, the time histories for $I_1(t)$ and $I_2(t)$ are plotted in black and green, respectively.

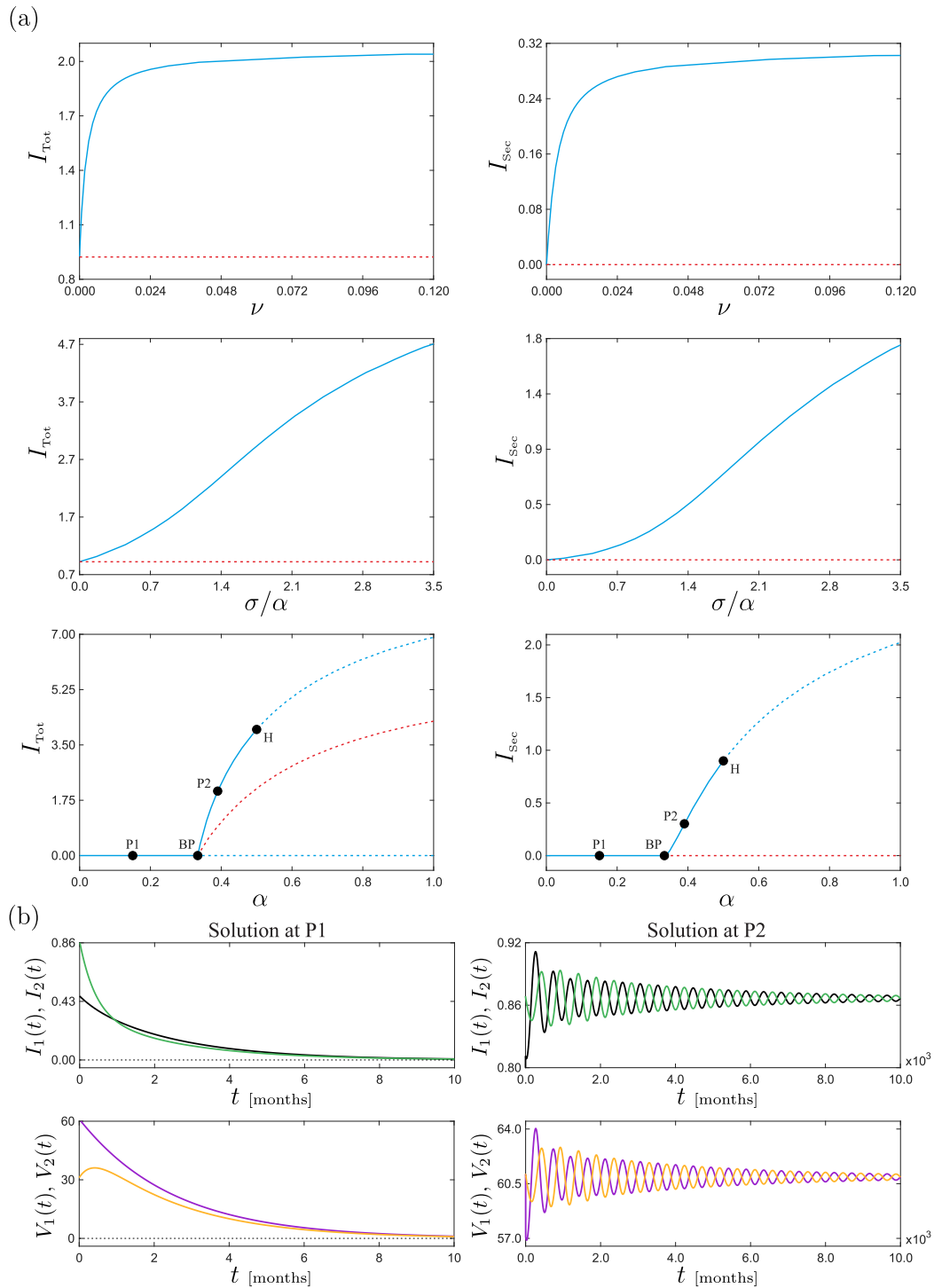


Figure 3. (a) One-parameter continuation of the two-strain (blue curve) and one-strain (red curve) equilibria calculated from Figure 2. In these diagrams, the vertical axes are given by $I_{\text{Tot}} = I_1 + I_2 + I_{12} + I_{21}$ and $I_{\text{Sec}} = I_{12} + I_{21}$. The BP and H points stand for branching ($\alpha \approx 0.33355$) and Hopf ($\alpha \approx 0.50012$) bifurcations, respectively. Solution branches of stable equilibria are plotted with solid lines, while dashed lines denote instability. (b) System responses are calculated at the test points P1 ($\alpha = 0.15$) and P2 ($\alpha = 0.39$). Here, the color code is as follows: $I_1(t)$ (black), $I_2(t)$ (green), $V_1(t)$ (purple), and $V_2(t)$ (yellow).

To initiate our analysis, we adopt the parameter values of the two-strain dengue epidemics model (2.1) as listed in Table 2, with $\sigma = 0.45$. With these values, the system response, obtained via direct numerical integration, is presented in Figure 2. The figure shows time series for the following state variables: $S(t)$ (susceptible individuals that can be infected by all dengue strains), I_1 (individuals infected with strain 1), I_2 (individuals infected with strain 2), and $I_{12} + I_{21}$ (individuals suffering secondary infections, i.e., those that have been infected by both strains, one after another). Interestingly, for the same set of parameter values, two types of behavior can be detected: one showing the existence of both strains in the population, and one response showing one strain only (I_1 in this case). The first solution is obtained using nonzero initial conditions for all compartments, while the second scenario is produced by setting $I_2 = R_2 = S_2 = I_{12} = I_{21} = R = V_2 = V_{12} = 0$.

Due to symmetry, another solution may be computed, corresponding to the existence of strain 2 only, but for the sake of brevity, only the former case will be considered. The existence of only one strain can be clearly verified from the second column of Figure 2, where the solutions corresponding to I_2 (in green) and secondary infections $I_{12} + I_{21}$ are zero. As can be seen in the time histories, the system in both cases eventually settles down to an equilibrium, one with both strains present, and one with strain 1 only. These equilibria will serve as the baseline for our numerical exploration, where we will focus on how changes in critical parameters affect the steady state, with special attention put on variations of dengue infections with respect to parameter perturbations.

We now apply numerical continuation (path-following) methods to investigate how the equilibria identified earlier evolve under gradual variation of selected parameters. The outcome of this procedure is illustrated in Figure 3(a). Here, two solution measures are displayed: $I_{\text{Tot}} = I_1 + I_2 + I_{12} + I_{21}$, showing the total number of dengue infections (primary and secondary), and $I_{\text{Sec}} = I_{12} + I_{21}$, measuring secondary infections only. The continuation of two-strain and one-strain equilibria are plotted using blue and red colors, respectively. The first parameter considered for the numerical continuation is the cross-immunity factor ν , with $1/\nu$ being the average cross-immunity duration. As explained earlier, this parameter defines how long an individual remains immune to a secondary dengue infection. In the absence of strain 2 (see the second column in Figure 2), this parameter has no effect in the system, due to which a horizontal straight line (red) is obtained in Figure 3(a). When both strains are present, on the contrary, ν does affect the level of dengue infections, as can be seen from the behavior of the blue line. As can be expected, larger values of ν (meaning shorter cross-immunity duration) increase the level of both total and secondary dengue infections.

Another parameter considered in our study is the secondary infection rate σ . For a better understanding, in Figure 3(a), we have displayed the ratio σ/α (with α fixed) to compare the levels of secondary and primary infection rates. Analogously to the case studied before, this ratio has no effect whatsoever when only one strain is present in the system. However, the situation clearly changes when both strains are circulating in the model, as can be seen in Figure 3(a). As before, when σ increases, higher levels of total and secondary dengue infections are produced in the model. Next, we will consider variations in the primary infection rate α . As can be seen in Figure 3(a), for low values of this parameter, a stable disease-free equilibrium is present, with basic reproduction number $\mathcal{R}_0 < 1$. As α grows, $\mathcal{R}_0$ grows too (see Section 3.2), until the critical value 1 is reached, for $\alpha \approx 0.33355$. Here, a classical forward bifurcation occurs, giving rise to three branches: unstable disease-free, stable two-strain, and unstable one-strain equilibria, see Figure 3(a). The instability of the one-strain branch displayed here corresponds to instability with respect to heterologous-strain perturbations. Hence, a small introduction of the absent

strain can grow and drive the system toward the two-strain state. This numerical invasion property is consistent with the enhanced secondary-transmission mechanism when $\sigma > \alpha$; however, $\sigma > \alpha$ identifies the ADE regime and should not be interpreted as an independent necessary and sufficient condition for loss of stability of every one-strain equilibrium. Notice that the red curve corresponding to one-strain solutions shows increasing behavior of the total number of dengue infections (influenced by the primary infection I_1), while secondary infections remain zero, as expected. On the other hand, the two-strain solution shows growing behavior in both dengue indicators. To confirm the numerical observations, two sample solutions are calculated via direct numerical integration in Figure 3(b), corresponding to the test points P1 (disease-free) and P2 (two-strain solution).

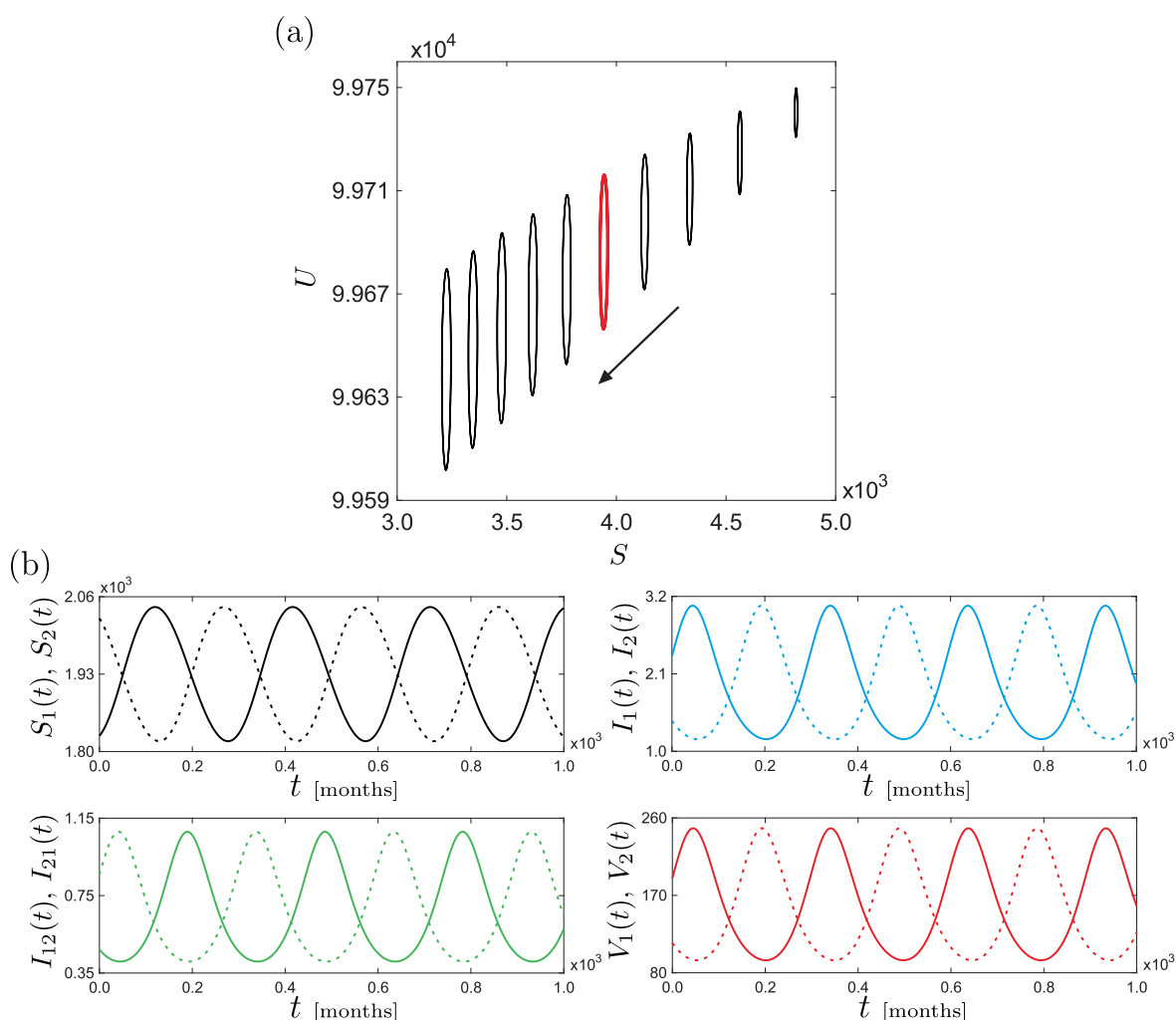


Figure 4. (a) Family of periodic solutions produced by the Hopf bifurcation detected in Figure 3(a), calculated from $\alpha = 0.529$ to $\alpha = 0.75$. The arrow indicates the direction of increasing α . (b) Time histories corresponding to the solution highlighted in red ($\alpha = 0.627$) in panel (a). In these plots, solid and dashed lines correspond to state variables with the leading subscript 1 and 2, respectively.

Our numerical investigation also reveals the presence of a supercritical Hopf bifurcation, detected when the primary infection rate reaches the critical value $\alpha \approx 0.50012$, with a negative first Lyapunov coefficient equal to -1.036432 (calculated using MATCONT [38]). Here, the two-strain equilibrium becomes unstable (represented by a dashed line), and a family of stable periodic solutions is born, see Figure 4. In this figure, panel (a) displays a sequence of periodic orbits with growing amplitude as α increases beyond the Hopf point (from $\alpha = 0.529$ to $\alpha = 0.75$). A test solution is depicted in panel (b), showing the oscillatory behavior of both human and vector compartments.

In summary, this section presented the numerical investigation of the dynamics of the two-strain dengue model (2.1), using continuation methods (COCO) complemented by direct time integrations, adopting the baseline parameters in Table 2 (with $\sigma = 0.45$). Varying the primary infection rate α uncovers a classical forward (branching) bifurcation at $\alpha \approx 0.33355$, where the disease-free equilibrium loses stability and a stable endemic branch emerges, hence marking an outbreak of the disease. If the primary infection rate further increases, a supercritical Hopf bifurcation is detected at $\alpha \approx 0.50012$, after which the two-strain equilibrium becomes unstable and a family of stable periodic orbits is born. In this way, the forward bifurcation $\alpha \approx 0.33355$ and Hopf bifurcation $\alpha \approx 0.50012$ are the main dynamical phenomena producing critical changes in the qualitative behavior of the two-strain dengue model (2.1).

5. Conclusions

In this work, we proposed a novel, deterministic, two-strain host–vector dengue transmission model. The model advances previous two-strain frameworks by distinguishing between primary and secondary human infections, integrating ADE effects through distinct transmission parameters (α, σ), and allowing vectors to host both single and double infections, thereby providing a more realistic representation of dengue ecology.

Analytically, we derived the basic reproduction number using the next-generation matrix method, obtaining $\mathcal{R}_0 = \sqrt{\frac{\alpha\beta}{\kappa(\gamma+\mu)}}$, and established that the disease-free equilibrium is locally asymptotically stable whenever $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$. The analysis of the one-strain endemic states shows that their stability against introduction of the heterologous strain is governed by the corresponding invasion dynamics. In particular, the secondary-infection parameter σ enters the heterologous invasion subsystem directly, so that enhanced secondary transmission in the ADE regime ($\sigma > \alpha$) promotes invasion of the absent strain. Consistently, the numerical continuation considered here identifies parameter regimes in which the one-strain equilibrium is unstable to heterologous perturbations and the two-strain state is stable. Through center-manifold theory, we demonstrated that the system admits backward bifurcation under suitable parameter regimes, implying that dengue persistence is possible even when $\mathcal{R}_0 < 1$, due to the coexistence of stable disease-free and endemic equilibria. This mathematical property highlights the challenge of dengue eradication: simple threshold control strategies based solely on $\mathcal{R}_0$ may fail when enhancement mechanisms are strong.

Our numerical continuation and bifurcation analysis (performed with COCO) revealed a rich dynamical landscape consistent with the analytical findings. For baseline parameters, the model exhibits bistability between single- and two-strain endemic equilibria. As the primary infection rate α increases, a forward (branching) bifurcation occurs near $\alpha \approx 0.33355$, marking the transition from the disease-free to the endemic regime. A subsequent Hopf bifurcation at $\alpha \approx 0.50012$ gives rise to a family of stable periodic orbits, characterized by oscillations in both human and vector infection classes. The amplitude

of these oscillations grows with α , indicating how ADE and high transmission intensity can produce recurrent epidemic outbreaks. Variations in cross-immunity loss ν and the secondary-to-primary infection ratio σ/α further demonstrated how shorter immunity durations and stronger enhancement intensify total and secondary infection levels, respectively. Together, these results confirm that ADE, temporary immunity, and vector co-infection jointly generate multistability and oscillatory behavior observed in multi-strain dengue epidemics in endemic regions. These dynamics may give insights into the epidemiological patterns of dengue disease. In this context, forward bifurcation demonstrates the critical threshold required for an endemic state, while Hopf bifurcation may represent recurrent outbreaks triggered by secondary infections. These results could provide useful guidance for public health authorities to establish monitoring strategies to identify and anticipate outbreak peaks before they occur, instead of relying mainly on control measures employed after the initial emergence of an outbreak.

From an epidemiological perspective, these findings suggest that reducing $\mathcal{R}_0$ below unity may be insufficient for disease elimination when backward bifurcation or bistability occur. The local sensitivity indices of $\mathcal{R}_0$ further show that reductions in human–vector transmission, increased vector mortality, and a shorter human infectious period all act directly to lower the invasion threshold. Control measures that reduce secondary transmission (σ), prolong temporary immunity ($1/\nu$), or mitigate ADE effects could play a more effective role than focusing solely on primary infection reduction. Moreover, understanding how parameter interactions drive oscillatory outbreaks can inform vaccination design, especially for vaccines that modulate immune enhancement or cross-protection.

Future work could extend the present framework by incorporating age or spatial structure, stochastic effects, or seasonally forced mosquito dynamics to better capture the temporal variability observed in real-world dengue outbreaks. The model can also be coupled with optimal control formulations to assess the efficiency of combined interventions such as vaccination, vector control, and public health campaigns. Ultimately, the proposed two-strain host–vector model provides a rigorous and biologically grounded platform for exploring complex dengue transmission dynamics and for guiding the development of evidence-based control strategies in dengue-endemic regions.

Use of AI tools declaration

The authors declare that there are no Artificial Intelligence (AI) tools used in the creation of this article.

Data availability statement

Data sharing is not applicable to this article as no datasets were generated or analyzed during the current study.

Conflict of interest

Burcu Gürbüz is a guest editor for *Electronic Research Archive* and was not involved in the editorial review or the decision to publish this article. The authors state that there is no conflict of interest.

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Appendix

Linearization around a generic steady state

$$E^F = (S^F, I_1^F, I_2^F, R_1^F, R_2^F, S_1^F, S_2^F, I_{12}^F, I_{21}^F, R^F, U^F, V_1^F, V_2^F, V_{12}^F),$$

leads to the Jacobian matrix

$$\mathcal{J} = [J_{ij}]_{14 \times 14}, \quad \text{for } i, j = 1, 2, \dots, 14, \quad (\text{A1})$$

where the Jacobian can be re-expressed as

$$\mathcal{J} = [\mathcal{M}_{14 \times 7} \quad \mathcal{N}_{14 \times 7}],$$

with

$$M = \begin{bmatrix} -\alpha M^F A_V - \mu & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha M^F A_1 & -\bar{\gamma} & 0 & 0 & 0 & 0 & 0 \\ \alpha M^F A_2 & 0 & -\bar{\gamma} & 0 & 0 & 0 & 0 \\ 0 & \gamma & 0 & -\bar{\nu} & 0 & 0 & 0 \\ 0 & 0 & \gamma & 0 & -\bar{\nu} & 0 & 0 \\ 0 & 0 & 0 & \nu & 0 & -\sigma M^F A_2 - \mu & 0 \\ 0 & 0 & 0 & 0 & \nu & 0 & -\sigma M^F A_1 - \mu \\ 0 & 0 & 0 & 0 & 0 & \sigma M^F A_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma M^F A_1 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \beta U^F A_I & -\beta U^F A_N & -\beta U^F A_N & \beta U^F A_I & \beta U^F A_I & \beta U^F A_I & \beta U^F A_I \\ A_{UV_1} & \beta U^F / N^F + A_{UV_1} & -\beta V_1^F / N^F + A_{UV_1} & A_{UV_1} & A_{UV_1} & A_{UV_1} & A_{UV_1} \\ A_{UV_2} & -\beta V_1^F / N^F + A_{UV_2} & \beta U^F / N^F + A_{UV_2} & A_{UV_2} & A_{UV_2} & A_{UV_2} & A_{UV_2} \\ A_{VV} & \beta V_2^F / N^F + A_{VV} & \beta V_1^F / N^F + A_{VV} & A_{VV} & A_{VV} & A_{VV} & A_{VV} \end{bmatrix}$$

and

$$N = \begin{bmatrix} 0 & 0 & 0 & \alpha S^F A_V & -\alpha S^F A_U & -\alpha S^F A_U & -\alpha S^F A_{UV} \\ 0 & 0 & 0 & -\alpha S^F A_1 & \alpha S^F A_{U2} & -\alpha S^F A_1 & \alpha S^F A_{U2} \\ 0 & 0 & 0 & -\alpha S^F A_2 & -\alpha S^F A_2 & \alpha S^F A_{U1} & \alpha S^F A_{U1} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \sigma S_1^F A_2 & \sigma S_1^F A_2 & -\sigma S_1^F A_{U1} & -\sigma S_1^F A_{U1} \\ 0 & 0 & 0 & \sigma S_2^F A_1 & -\sigma S_2^F A_{U2} & \sigma S_2^F A_1 & -\sigma S_2^F A_{U2} \\ -\bar{\delta} & 0 & 0 & -\sigma S_1^F A_2 & -\sigma S_1^F A_2 & \sigma S_1^F A_{U1} & \sigma S_1^F A_{U1} \\ 0 & -\bar{\delta} & 0 & -\sigma S_2^F A_1 & \sigma S_2^F A_{U2} & -\sigma S_2^F A_1 & \sigma S_2^F A_{U2} \\ \gamma & \gamma & -\mu & 0 & 0 & 0 & 0 \\ -\beta U^F A_N & -\beta U^F A_N & \beta U^F A_I & -\beta N^F A_I - \kappa & 0 & 0 & 0 \\ -\beta V_1^F / N^F + A_{UV_1} & \beta U^F / N^F + A_{UV_1} & A_{UV_1} & N^F A_{I1} & -N^F A_{I2} - \kappa & 0 & 0 \\ \beta U^F / N^F + A_{UV_2} & -\beta V_2^F / N^F + A_{UV_2} & 0 & N^F A_{I2} & 0 & -N^F A_{I1} - \kappa & 0 \\ \beta V_1^F / N^F + A_{UV_2} & \beta V_2^F / N^F + A_{UV_2} & A_{UV_2} & 0 & N^F A_{I2} & N^F A_{I1} & -\kappa \end{bmatrix},$$

where $\bar{\gamma} = \gamma + \mu$, $\bar{\nu} = \nu + \mu$, $\bar{\delta} = \gamma + \mu + \delta$, and

$$\begin{aligned} A_V &= \frac{V_1^F + V_2^F + 2V_{12}^F}{(M^F)^2}, \quad A_1 = \frac{V_1^F + V_{12}^F}{(M^F)^2}, \quad A_2 = \frac{V_2^F + V_{12}^F}{(M^F)^2}, \quad A_U = \frac{(U^F - V_{12}^F)}{(M^F)^2}, \\ A_{UV} &= \frac{V_1^F + V_2^F + 2U}{(M^F)^2}, \quad A_{U1} = \frac{V_1^F + U^F}{(M^F)^2}, \quad A_{U2} = \frac{V_2^F + U^F}{(M^F)^2}, \quad A_I = \frac{I_1^F + I_2^F + I_{12}^F + I_{21}^F}{(N^F)^2}, \\ A_N &= \frac{S^F + R_1^F + R_2^F + S_1^F + S_2^F + R^F}{(N^F)^2}, \quad A_{I1} = \beta \frac{I_1^F + I_{21}^F}{(N^F)^2}, \quad A_{I2} = \beta \frac{I_2^F + I_{12}^F}{(N^F)^2}, \\ A_{UV_1} &= -U^F A_{I1} + V_1^F A_{I2}, \quad A_{UV_2} = -U^F A_{I2} + V_2^F A_{I1}, \quad A_{VV} = -V_1^F A_{I2} - V_2^F A_{I1}. \end{aligned} \quad (A2)$$



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