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An Age-Structured Model for Two- Serotype Dengue Dynamics with Cross Immunity

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Abstract - Dengue fever is a mosquito-borne disease whose transmission is influenced by interactions among multiple serotypes, host immunity, and age-dependent contact patterns. In this study, we develop a deterministic age-structured model for two interacting dengue serotypes that incorporates temporary cross-immunity and vector–host dynamics. The human population is described by the McKendrick–von Foerster framework, while the mosquito population is represented by a susceptible–exposed–infectious (SEI) model. The proposed model incorporates lifelong immunity to the infecting serotype, temporary cross-immunity, waning immunity, reduced susceptibility to secondary infection, and proportionate mixing to account for age-dependent exposure. The positivity and boundedness of solutions are established, and the basic reproduction number, R_0, is derived using the next-generation operator approach. Conditions for the local stability of the disease-free equilibrium and the existence of an endemic equilibrium are also obtained. Numerical simulations, illustrated using dengue incidence data from India, reproduce recurrent epidemic patterns characterized by primary and delayed secondary outbreaks and demonstrate the influence of age structure, temporary cross-immunity, and waning immunity on disease dynamics. The proposed framework provides a useful tool for investigating multi-serotype dengue transmission and evaluating potential disease control strategies.

Keywords: Age-structured model, dengue dynamics, cross-immunity, two-wave epidemic and basic reproduction number..

1. INTRODUCTION

Dengue fever is one of the most rapidly spreading mosquito-borne viral diseases and continues to pose a major public health challenge in tropical and subtropical regions. According to the World Health Organization (WHO), the global incidence of dengue has increased substantially over the past few decades, with an estimated 390 million infections occurring annually**,** of which approximately 96–100 million are clinically apparent [4]. Nearly half of the world's population is currently at risk of dengue infection owing to rapid urbanization, climate variability, globalization, and the widespread distribution of *Aedes aegypti* mosquitoes [14,28,29]. Recent WHO reports indicate that more than 14 million dengue cases were recorded worldwide in 2024, representing one of the highest annual disease burdens reported to date [29]. These trends emphasize the need for reliable mathematical models that improve the understanding of dengue transmission dynamics and support the development of effective prevention and control strategies.

Dengue virus comprises four antigenically distinct serotypes, namely DENV-1, DENV-2, DENV-3, and DENV-4, all of which are transmitted primarily through the bite of infected *Aedes aegypti* mosquitoes. Infection with one serotype provides lifelong immunity against that specific serotype but confers only temporary cross-immunity to the remaining serotypes. As this transient protection diminishes over time, individuals become susceptible to infection with a different serotype. Secondary infections are frequently associated with an increased risk of severe clinical manifestations because of immunological mechanisms such as antibody-dependent enhancement (ADE) [16]. Consequently, interactions among multiple serotypes generate complex transmission patterns, including recurrent outbreaks, serotype replacement, and cyclical epidemic behaviour. These biological characteristics make dengue substantially more challenging to model than single-strain infectious diseases and highlight the importance of incorporating cross-immunity and multi-serotype interactions into mathematical transmission models.

India is one of the countries most severely affected by dengue, where the disease has become endemic in many regions owing to rapid urbanization, population growth, climate variability, and favourable environmental conditions for mosquito breeding. According to the National Centre for Vector Borne Diseases Control (NCVBDC), Ministry of Health and Family Welfare, Government of India, approximately 289,235 dengue cases and 485 deaths were reported during 2023 [28]. However, the actual disease burden is believed to be considerably higher, with model-based estimates suggesting nearly 5.78 million clinically diagnosed dengue cases annually in India [25]. Dengue affects individuals of all age groups, although

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epidemiological studies indicate that children, adolescents, and young adults, particularly those between 5–19 and 20–39 years of age, experience relatively higher infection rates because of increased outdoor activity, mobility, and occupational exposure [11,14]. These age-dependent differences in disease transmission highlight the importance of incorporating population age structure into mathematical models to improve the understanding of dengue dynamics and support the design of more effective control strategies.

Mathematical modelling has become an indispensable tool for understanding infectious disease transmission and supporting evidence-based public health decision-making. The mathematical foundation of age-structured population dynamics was established by McKendrick [20], introduced a framework describing the evolution of populations with respect to age and time. This pioneering work was subsequently extended by Webb [27] extended this framework by developing nonlinear age-dependent population models and further strengthened by Thieme [26], provided a rigorous mathematical treatment of structured population dynamics. The mathematical theory of ordinary differential equations developed by Hale [15] has also provided fundamental analytical tools that underpin the qualitative analysis of dynamical systems arising in epidemiological modelling. The incorporation of age heterogeneity into epidemic systems was advanced by Castillo-Chavez et al. [6], introduced epidemiological concepts such as proportionate mixing and cross-immunity, while Arino et al. [3] developed vector–host epidemic models that explicitly accounted for host age structure. A major theoretical contribution was made by Diekmann et al. [8], formalized the next-generation matrix approach for computing the basic reproduction number. This framework was later extended to multi-strain age-structured epidemic systems by Magal and Webb [19]. Comprehensive mathematical treatments of structured population dynamics and infectious disease modelling are further provided by Diekmann et al. [9] and Inaba [18], which continue to serve as fundamental references for analysing age-dependent epidemic systems.

Building upon these theoretical developments**,** numerous studies have developed models to investigate various aspects of dengue transmission. From a biological perspective, Halstead [16] established the role of antibody-dependent enhancement (ADE) in severe secondary infections, providing an immunological basis for modelling multi-serotype dengue. Ferguson et al. [11] demonstrated the importance of age-stratified seroprevalence data for understanding dengue transmission dynamics, Chowell et al. [7] investigated dengue transmission dynamics in a low-income endemic setting, demonstrating how epidemiological modelling can be used to analyse outbreak patterns and disease spread. while Dominici and Rosenfeld [10] proposed one of the earliest age-structured mathematical models for dengue epidemics. Gomes et al. [12] investigated the effects of temporary cross-immunity on the persistence and invasion of multiple serotypes, whereas Aguiar et al. [1] analysed the potential risks associated with dengue vaccination in the presence of ADE. Subsequently, Gonçalves et al. [13] incorporated vaccination and serotype interactions into a dynamic transmission model, and Billings et al. [5] further examined the epidemiological consequences of antibody-dependent enhancement. A comprehensive review of serotype interactions and mathematical modelling approaches was presented by Aguiar et al. [2]. More recently, Oliveira et al. [21] developed a multistrain dengue model incorporating cross-immunity and vector dynamics, Herdicho et al. [17] investigated the impact of public awareness and optimal control measures, and Raj et al. [22] proposed an age-structured delay differential equation model to better capture disease progression. Collectively, these studies have significantly improved the understanding of dengue transmission; however, relatively few models simultaneously incorporate continuous age structure, temporary cross-immunity, vector–host interactions, and rigorous mathematical analysis within a unified modelling framework.

In our previous studies [23,24],we developed age-structured epidemic models incorporating spatial diffusion, optimal control, stochastic effects, and vector-borne disease dynamics, demonstrating the importance of age structure and spatial heterogeneity in epidemic modelling. However, these studies did not explicitly consider interactions between multiple dengue serotypes or the effects of temporary cross-immunity in recurrent outbreaks.

Despite the considerable progress achieved in dengue modelling, important challenges remain. Many existing models focus on homogeneous populations or single-serotype transmission, whereas others investigate multiple serotypes without explicitly incorporating continuous age structure or age-dependent exposure to mosquito bites. Similarly, only a limited number of studies simultaneously integrate temporary cross-immunity, heterogeneous mixing, vector–host interactions, and rigorous mathematical analysis within a unified age-structured framework. Addressing these limitations provides the primary motivation for the present study.

Motivated by these challenges, the present study develops a deterministic age-structured dengue transmission model describing the interaction between two dengue serotypes and the mosquito vector population. The human population is represented using the McKendrick–von Foerster partial differential equation framework, whereas the mosquito population is modelled through ordinary differential equations. The proposed model incorporates temporary cross-immunity, age-dependent proportionate mixing, and secondary infection

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following immunity waning. Theoretical results establish the positivity and boundedness of solutions, derive the basic reproduction number using the next-generation operator approach, analyse the stability of the disease-free equilibrium, and prove the existence of an endemic equilibrium. Numerical simulations based on epidemiological data from India illustrate the influence of age structure and cross-immunity on recurrent dengue outbreaks and provide insights that may support future dengue surveillance and control strategies.

2. MODEL FORMULATION

Dengue transmission is characterized by complex immunological interactions among multiple virus serotypes. Infection with one serotype provides lifelong immunity against the infecting serotype but only temporary cross-immunity against the remaining serotypes. After this temporary protection wanes, individuals become susceptible to secondary infection with a different serotype, which may increase the risk of severe disease because of antibody-dependent enhancement (ADE) [2,16]. These immunological processes contribute to recurrent outbreaks, alternating serotype dominance, and long-term epidemic persistence. Therefore, incorporating temporary cross-immunity into mathematical models is essential for accurately describing multi-serotype dengue transmission dynamics.

Another important feature of dengue transmission is the heterogeneity in mosquito exposure across different age groups. Variations in daily activities, mobility, and behavioural patterns result in age-dependent differences in contact with mosquito vectors. To account for this heterogeneity, we adopt the proportionate mixing assumption, whereby the force of infection is weighted by an age-dependent biting or activity function. Under this assumption, individuals with greater exposure contribute more to disease transmission, providing a more realistic representation of infection dynamics in heterogeneous populations [6].

Although dengue virus consists of four antigenically distinct serotypes, many epidemiological and mathematical studies focus on the interaction between two representative serotypes because this framework captures the essential mechanisms of temporary cross-immunity, waning immunity, secondary infection, and serotype competition while remaining mathematically tractable [1,2,21]. Moreover, outbreaks within a particular region are often dominated by one or two circulating serotypes. Accordingly, the present study considers a two-serotype framework to investigate the influence of age structure and temporary cross-immunity on dengue transmission dynamics.

Accordingly, the present study develops an age-structured mathematical model describing the transmission dynamics of two interacting dengue serotypes coupled with mosquito vector populations. The model is used to derive the basic reproduction number, investigate the stability of the disease-free and endemic equilibria, and evaluate the influence of temporary cross-immunity and age-dependent mixing on dengue transmission.

The disease transmission process is illustrated in Figure 1, which describes the interaction between an age-structured human population and the mosquito vector population through two circulating dengue serotypes. The human population is represented by a system of age-dependent compartments, whereas the mosquito population is described by a compartmental SEI model.

New-born individuals enter the susceptible class $S(a,t)$. Susceptible individuals may acquire a primary infection with either serotype 1 or serotype 2 through bites from infectious mosquitoes at age-dependent forces of infection $\lambda_1(a,\mathrm{t})$ and $\lambda_2(a,\mathrm{t})$, respectively. Following infection, individuals move to the infectious compartments $I_1(a,t)$ and $I_2(a,t)$, where they remain until recovery at rates $\gamma_1(a)$ and $\gamma_2(a)$. Recovered individuals enter the classes $R_1(a,t)$ and $R_2(a,t)$, acquiring lifelong immunity to the infecting serotype together with temporary cross-immunity against the alternate serotype.

Temporary cross-immunity wanes at rate ω, after which recovered individuals enter the partially susceptible compartments $S_1(a,t)$ and $S_2(a,t)$. Individuals in $S_1(a,t)$ are susceptible only to serotype 2, whereas individuals in $S_2(a,t)$ are susceptible only to serotype 1. Secondary infections occur at reduced transmission rates $\sigma\lambda_2(a,\mathrm{t})$ and $\sigma\lambda_1(a,\mathrm{t})$, respectively, where the parameter $\sigma \in [0,1]$ quantifies the level of susceptibility after temporary cross-immunity has waned. Natural mortality $\mu(a)$ acts on all human compartments throughout the disease progression.

The mosquito population is represented by an SEI model for each serotype. Susceptible mosquitoes $S_m(t)$become infected after feeding on infectious humans and enter the exposed compartments $E_{m,i}(t), i = 1,2$. Following the extrinsic incubation period, exposed mosquitoes progress to the infectious compartments $I_{m,i}(t)$ at rate κ_m. . Infectious mosquitoes subsequently transmit the corresponding serotype to susceptible humans during blood feeding, thereby generating the age-dependent forces of infection$\lambda_1(a,\mathrm{t})$ and $\lambda_2(a,\mathrm{t})$. Mosquitoes experience natural mortality at rate μ_m..

The interaction between the human and mosquito populations therefore forms a coupled PDE–ODE system that captures primary infection, temporary cross-immunity, secondary infection, age-dependent transmission, and vector–host interactions within a unified mathematical framework.

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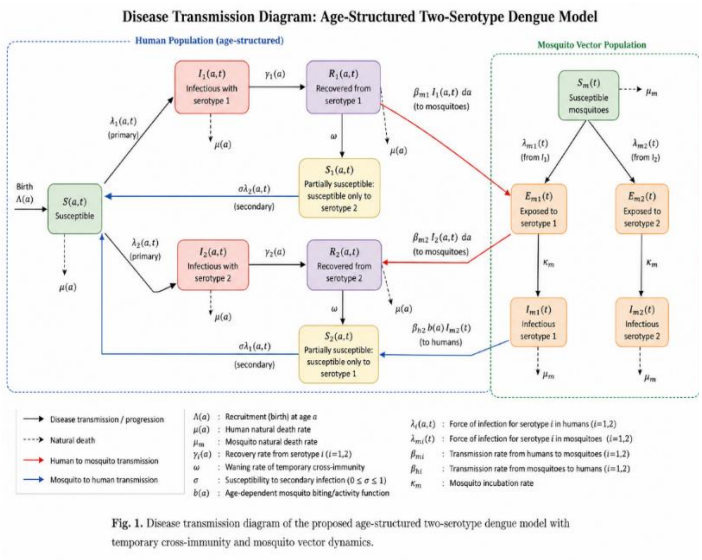

Fig. 1. Disease transmission diagram of the proposed age-structured two-serotype dengue model with temporary cross-immunity and mosquito vector dynamics.

Fig.1: Disease transmission diagram of the proposed age-structured two-serotype dengue model with temporary cross-immunity and mosquito vector dynamics.

To account for age-dependent heterogeneity in mosquito exposure, let $b(a) \geq 0$ denote the biting or activity rate of individuals of age a. Under the assumption of proportionate mixing, the probability of human infection is proportional to the level of exposure to infectious mosquito bites. Consequently, the age-specific force of infection for serotype i $(i = 1,2)$ is defined as

$$\lambda_i(a,t) = \beta_{hi} b(a) \frac{I_{m,i}(t)}{N_m(t)} \qquad (2.1)$$

where β_{hi} denotes the mosquito-to-human transmission coefficient for serotype i, $I_{m,i}(t)$ is the infectious mosquito population carrying serotype i, and $N_m(t)$ represents the total mosquito population at time t.

The proposed model is based on the following biological assumptions regarding immunity and secondary infection:

1. Infection with serotype i confers lifelong immunity against the same serotype.
2. Recovered individuals acquire temporary cross-immunity against the alternate serotype.
3. Temporary cross-immunity wanes at a constant rate $\omega > 0$.
4. Following immunity waning, individuals become partially susceptible to the alternate serotype.
5. The parameter $\sigma \in [0,1]$ represents the relative susceptibility to secondary infection after cross-immunity has waned, where

$$\sigma = \begin{cases} 0, & \text{complete cross} - \text{immunity}, \\ 1, & \text{no cross} - \text{immunity}, \\ 0 < \sigma < 1, & \text{partial cross} - \text{immunity}. \end{cases}$$

These assumptions enable the model to capture the essential immunological mechanisms governing dengue transmission, including primary infection, temporary cross-immunity, waning immunity, and secondary infection with an alternate serotype.

Let $a \geq 0$ denote the age of an individual and $t \geq 0$ denote time. The age-structured human population is divided into seven epidemiological compartments: susceptible individuals $S(a,t)$, infectious individuals with serotype 1 and serotype 2, denoted by $I_1(a,t)$ and $I_2(a,t)$, recovered individuals $R_1(a,t)$ and $R_2(a,t)$, and partially susceptible individuals $S_1(a,t)$ and $S_2(a,t)$, who become susceptible to secondary infection after the waning of temporary cross-immunity. New-born individuals enter the susceptible class at recruitment rate Λ. The movement of individuals among these compartments is governed by the coupled system of first-order hyperbolic partial differential equations given by

$$\frac{\partial S(a,t)}{\partial t} + \frac{\partial S(a,t)}{\partial a} = -(\lambda_1(a,t) + \lambda_2(a,t))S(a,t) - \mu(a)S(a,t)$$

$$\frac{\partial I_1(a,t)}{\partial t} + \frac{\partial I_1(a,t)}{\partial a} = \lambda_1(a,t)S(a,t)\sigma\lambda_1(a,t)S_2(a,t) - (\gamma_1(a) + \mu(a))I_1(a,t)$$

$$\frac{\partial I_2(a,t)}{\partial t} + \frac{\partial I_2(a,t)}{\partial a} = \lambda_2(a,t)S(a,t)\sigma\lambda_2(a,t)S_1(a,t) - (\gamma_2(a) + \mu(a))I_2(a,t)$$

$$\frac{\partial R_1(a,t)}{\partial t} + \frac{\partial R_1(a,t)}{\partial a} = \gamma_1(a)I_1(a,t) - \big(\omega + \mu(a)\big)R_1(a,t)$$

$$\frac{\partial R_2(a,t)}{\partial t} + \frac{\partial R_2(a,t)}{\partial a} = \gamma_2(a)I_2(a,t) - \big(\omega + \mu(a)\big)R_2(a,t)$$

$$\frac{\partial S_1(a,t)}{\partial t} + \frac{\partial S_1(a,t)}{\partial a} = \omega R_1(a,t) - \sigma\lambda_2(a,t)S_1(a,t) - \mu(a)S_1(a,t)$$

$$\frac{\partial S_2(a,t)}{\partial t} + \frac{\partial S_2(a,t)}{\partial a} = \omega R_2(a,t) - \sigma\lambda_1(a,t)S_2(a,t) - \mu(a)S_2(a,t) \qquad (2.2)$$

The total human population density is

$$N_h(a,t) = S(a,t) + I_1(a,t) + I_2(a,t) + R_1(a,t) + R_2(a,t) + S_1(a,t) + S_2(a,t).$$

The total human population size at time t is given by integrating the age density over all ages $N_h(t) = \int_0^\infty N_h(a,t)da$.

By summing all the model equations, the total population satisfies the demographic balance equation

$$\frac{\partial N_h(a,t)}{\partial t} + \frac{\partial N_h(a,t)}{\partial a} = -\mu(a)N_h(a,t) \qquad (2.3)$$

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which indicates that the total population changes only due to natural mortality, since infection processes merely redistribute individuals among epidemiological compartments.

Boundary and Initial Conditions

New-born individuals enter the susceptible class:

$$S(0,t) = \Lambda, I_i(0,t) = R_i(0,t) = S_i(0,t) = 0. \quad (2.4)$$

Initial age distributions are prescribed as nonnegative measurable functions:

$$S(a,0) = S_0(a), I_i(a,0) = I_{i0}(a), R_i(a,0) = R_{i0}(a), S_i(a,0) = S_{i0}(a). \quad (2.5)$$

The mosquito population is described by an SEI system for each serotype.

Force of Infection on Mosquitoes

$$\lambda_{m,i}(t) = \beta_{mh} \frac{\int_0^\infty b(a)\, I_i(a,t)da}{N_h(t)} \quad (2.6)$$

Where,β_{mh} is the human-to-mosquito transmission coefficient.

Mosquito ODE System is

$$\frac{dS_m}{dt} = \Lambda_m - \big(\lambda_{m,1}(t) + \lambda_{m,2}(t)\big)S_m(t) - \mu_m S_m(t),$$
$$\frac{dE_{m,i}}{dt} = \lambda_{m,i}(t)S_m(t) - (\kappa_m + \mu_m)E_{m,i}(t),$$
$$\frac{dI_{m,i}}{dt} = \kappa_m E_{m,i}(t) - \mu_m I_{m,i}(t), i \in \{1,2\}. \quad (2.7)$$

The total mosquito population is

$$N_m(t) = S_m(t) + E_{m,1}(t) + E_{m,2}(t) + I_{m,1}(t) + I_{m,2}(t).$$

By summing the differential equations for all mosquito compartments, we obtain the total mosquito population dynamics.

$$\frac{dN_m(t)}{dt} = \frac{dS_m}{dt} + \frac{dE_{m,1}}{dt} + \frac{dE_{m,2}}{dt} + \frac{dI_{m,1}}{dt} + \frac{dI_{m,2}}{dt}.$$

Substituting the model equations gives

$$\frac{dN_m(t)}{dt} = \Lambda_m - \mu_m S_m - \mu_m E_{m,1} - \mu_m E_{m,2} - \mu_m I_{m,1} - \mu_m I_{m,2}.$$

Factoring out μ_m yields

$$\frac{dN_m(t)}{dt} = \Lambda_m - \mu_m(S_m + E_{m,1} + E_{m,2} + I_{m,1} + I_{m,2}).$$

Using the definition of $N_m(t)$, the total mosquito population satisfies

$$\frac{dN_m(t)}{dt} = \Lambda_m - \mu_m N_m(t). \quad (2.8)$$

3. MATHEMATICAL ANALYSIS

3.1 Positivity and Positive Invariance of solutions

In epidemiological models, it is essential to ensure that all state variables representing population densities remain non-negative for all time whenever the initial data are non-negative. This property guarantees the biological feasibility of the model. In this subsection, we establish the positivity and positive invariance of solutions for the proposed age-structured PDE–ODE system.

Let the human subsystem be given by

$$\frac{\partial X}{\partial t} + \frac{\partial X}{\partial a} = F_X(a,t) - G_X(a,t)X(a,t), \quad (3.1)$$

Where,$X(a,t) \epsilon \{S, I_1, I_2, R_1, R_2, S_1, S_2\}$, and the functions $F_X(a,t) \geq 0$ and $G_X(a,t) \geq 0$ are non-negative for all admissible values of $a \geq 0$ and $t \geq 0$.

The mosquito population is governed by the system of ordinary differential equations given in Equation (2.7).

Assume that the initial conditions satisfy

$$X(a,0) \geq 0, S_m(0) \geq 0,$$
$$E_{m,i}(0) \geq 0, I_{m,i}(0) \geq 0. \quad (3.2)$$

Hence, the feasible region $\Omega = \{(S, I_1, I_2, R_1, R_2, S_1, S_2, S_m, E_{m,i}, I_{m,i}) \in \mathbb{R}_+^{10}\}$ Using $\mathbb{R}$ is the standard notation.

Theorem 3.1: Assume that the initial conditions satisfy (3.2) and that all model parameters are non-negative. Furthermore, suppose that the total human population satisfies $N_h(0) > 0$.Then the solution of the coupled PDE–ODE system (2.2)-(2.7) remains non-negative for all $t \geq 0$.Moreover, the positive cone $\chi_+ = L_+^1(0,\infty)^7 \times R_+^5$ is positively invariant under the semi flow generated by the system.

Proof: Define the total human population density as

$$N_h(a,t) = S(a,t) + I_1(a,t) + I_2(a,t) + R_1(a,t) + R_2(a,t) + S_1(a,t) + S_2(a,t)$$

Integrating over the age domain gives

$$N_h(t) = \int_0^\infty N_h(a,t)da.$$

By summing all human equations in system (2.2), we obtain

$$\frac{\partial N_h(a,t)}{\partial t} + \frac{\partial N_h(a,t)}{\partial a} = -\mu(a)N_h(a,t)$$

Integrating over $a \in (0,\infty)$ and using the boundary condition $S(0,t) = \Lambda$,we obtain

$$\frac{d}{dt}N_h(t) = \Lambda - \int_0^\infty \mu(a)N_h(a,t)da. \quad (3.3)$$

Since, $\mu(a) \geq 0$, there exists a constant $\mu_{max} > 0$ such that

$\mu(a) \leq \mu_{max}, a \geq 0.$

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Hence ,

$$\frac{dN_h(t)}{dt} \geq \Lambda - \mu_{max} N_h(t) \quad (3.4)$$

By Gronwall's inequality [15] and standard comparison arguments, it follows that $N_h(t)$ remains strictly positive for all $t > 0$. Hence the force of infection terms are well defined for all $t > 0$.

Consider the transport operator

$\frac{\partial}{\partial t} + \frac{\partial}{\partial a}$ Along characteristic curves $\frac{da}{dt} = 1$,

$$a(t) = a_0 + t, \quad (3.5)$$

Equation (3.1) reduces to the ordinary differential equation

$$\frac{d}{dt} X(a(t), t) = F_X(a(t), t) - G_X(a(t), t) X(a(t), t). \quad (3.6)$$

Integral representation, Using the variation-of-constants formula for age structure population models [18, 27], the solution along characteristics can be written as

$$X(a(t), t) = X(a_0, 0) exp\left(-\int_0^t G_X(a(s), s) ds\right) + \int_0^t F_X(a(\tau), \tau)\, exp\left(-\int_\tau^t G_X(a(s), s)\, ds\right) d\tau. \quad (3.7)$$

Non-negativity of human compartments

Since $X(a_0, 0) \geq 0, F_X(a, t) \geq 0, G_X(a, t) \geq 0$.and the exponential terms are strictly positive, both terms in (3.7) are non-negative. Hence $X(a, t) \geq 0$ This holds for all human compartments remains non-negative.

The boundary conditions, $S(0, t) = \Lambda \geq 0$, $I_i(0, t) = R_i(0, t) = S_i(0, t) = 0$ $i \in \{1,2\}$,

Also preserve non-negativity at $a = 0$.

Each mosquito equation in (2.7) has the general form $\frac{dx}{dt} = f(t) - c(t)x,$ (3.8)

Where $f(t) \geq 0$ *and* $c(t) \geq 0$.

By the comparison principle for cooperative dynamical systems [9,26], nonnegative initial conditions imply $S_m(t), E_{m,i}(t), I_{m,i}(t) \geq 0$ therefore, both the human and mosquito subsystems preserve non-negativity since $N_h(t) > 0$, for all $t \geq 0$, the force of infection remains well defined. Consequently, every solution intiated in χ_+ for all $t \geq 0$. Hence, the positive cone is positively invariant.

3.2 Boundedness of Solutions

In this subsection, we establish that the solutions of the proposed model remain uniformly bounded for all time. This result ensures that the model dynamics evolve within a biologically meaningful region of the state space.

Theorem 3.2: Under the assumptions of Theorem 3.1, Then the total human population $N_h(t)$ and total mosquito population $N_m(t)$ remain uniformly bounded for all $t \geq 0$.Furthermore, each epidemiological compartment remains bounded in $L^1(0, \infty)$ for human population and in $R_+$for the mosquito population.

Proof: The total human population size is defined as $N_h(t) = \int_0^\infty N_h(a, t) da,$

Where,$N_h(a, t) = S(a, t) + I_1(a, t) + I_2(a, t) + R_1(a, t) + R_2(a, t) + S_1(a, t) + S_2(a, t)$

Summing all human population equations and integrating over age domain yields

$$\frac{d}{dt} N_h(t) = \Lambda - \int_0^\infty \mu(a) N_h(a, t) da. \quad (3.9)$$

Since,$\mu(a) \geq 0$,

We obtain $\frac{d}{dt} N_h(t) \leq \Lambda.$ (3.10)

Integrating inequality (3.10) gives

$$N_h(t) \leq N_h(0) + \Lambda t. \quad (3.11)$$

Equation (3.11) guarantees that $N_h(t)$remains finite for every finite time interval.

To obtain a uniform bound, assume that there exists a $\mu_0 > 0$ such that $\mu(a) \geq \mu_0 > 0$

then from equation (3.9) we obtain $\frac{d}{dt} N_h(t) = \Lambda - \int_0^\infty \mu(a)\, N_h(a, t) da \leq \Lambda - \mu_0 N_h(t)$

This differential inequality implies $\frac{d}{dt} N_h(t) \leq \Lambda - \mu_0 N_h(t)$

Consider the auxiliary scalar differential equation

$$\frac{dX(t)}{dt} = \Lambda - \mu_0 X(t)$$

Its solution is given by $X(t) = \frac{\Lambda}{\mu_0} + \left(X(0) - \frac{\Lambda}{\mu_0}\right) e^{-\mu_0 t}$

By the comparison principle for differential equation [9,15], it follows that

$$N_h(t) \leq max\left\{N_h(0), \frac{\Lambda}{\mu_0}\right\} \quad (3.12)$$

Hence the human population is uniformly bounded.

For the Mosquito Population, define

$$N_m(t) = S_m(t) + E_{m,1}(t) + E_{m,2}(t) + I_{m,1}(t) + I_{m,2}(t)$$

Summing equations (2.7) gives

$$\frac{d}{dt} N_m(t) = \Lambda_m - \mu_m N_m(t)$$

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The solution of this linear differential equation is $N_m(t) = \frac{\Lambda_m}{\mu_m} + \left(N_m(0) - \frac{\Lambda_m}{\mu_m}\right) e^{-\mu_m t}$ Therefore,

$$N_m(t) \leq max\left\{N_m(0), \frac{\Lambda_m}{\mu_m}\right\} \quad (3.13)$$

Therefore, the total mosquito population is uniformly bounded. Since, all human compartments are nonnegative (Theorem 3.1), Their sum is bounded by (3.12), each human compartment is bounded in $L^1(0,\infty)$.Similarly, all mosquito compartments are non-negative and their sum is bounded by equation (3.13). Consequently, all epidemiological compartments remain non-negative and uniformly bounded. Therefore, the solution of the coupled PDE–ODE system evolves in the positively invariant bounded set

$\chi_+ = L^1_+(0,\infty)^7 \times \mathbb{R}^5_+$ for all $t \geq 0$.

3.3 Disease-Free Equilibrium

In this section, we determine the infection-free steady state of the coupled age-structured human-mosquito system. The disease-free equilibrium (DFE) corresponds to a steady-state solution in which no infection persists in either the human or mosquito populations.

Mathematically, the DFE is characterized by

$$I_1(a,t) = I_2(a,t) = 0, R_1(a,t) = R_2(a,t) = 0, \quad S_1(a,t) = S_2(a,t) = 0 \quad (3.14)$$

and $E_{m,i}(t) = I_{m,i}(t) = 0, i \in \{1,2\}.$ (3.15)

Thus, at the disease free state, only susceptible individuals remain in both populations.

At the disease-free equilibrium, the human subsystem reduces to

$$\frac{\partial S(a,t)}{\partial t} + \frac{\partial S(a,t)}{\partial a} = -\mu(a)S(a,t) \quad (3.16)$$

With boundary condition $S(0,t) = \Lambda$

At equilibrium,$\frac{\partial S}{\partial t} = 0$,and therefore (3.16) becomes $\frac{dS^0(a)}{da} = -\mu(a)S^0(a)$

Where, $S^0(a)$ denotes the equilibrium age distribution. Solving

$$S^0(a) = \Lambda \exp\left(-\int_0^a \mu(s)ds\right) \quad (3.17)$$

Equation (3.17) represents the classical stable age distribution of a population subject to age-specific mortality [18,27].Thus, at the DFE, the total human population density is $N_h^0(a) = S^0(a)$, and the total human population size at equilibrium is

Therefore,$N_h^0 = \int_0^\infty S^0(a)da$ (3.18)

The mosquito subsystem reduces to

$\frac{dS_m(t)}{dt} = \Lambda_m - \mu_m S_m(t)$, Setting $\frac{dS_m(t)}{dt} = 0$

Since no infection is present, the exposed and infectious mosquito compartments vanish therefore, the total mosquito population at equilibrium satisfies

$$N_m^0 = S_m^0 = \frac{\Lambda_m}{\mu_m}$$

The disease-free equilibrium of the coupled system is given by

$$E_0 = (S^0(a), 0,0,0,0,0,0; S_m^0, 0,0,0,0) \quad (3.19)$$

Where,$S^0(a) = \Lambda \exp\left(-\int_0^a \mu(s)\mathrm{ds}\right)$,$S_m^0 = \frac{\Lambda_m}{\mu_m}$ (3.20)

3.4. Basic Reproduction Number and Stability of the Disease-Free Equilibrium

In this section, we derive the basic reproduction number R_0 for the age-structured dengue model and establish the threshold condition governing the local stability of the disease-free equilibrium.

3.4.1. Linearized Infected Subsystem

Let $\varepsilon_0 = (S^0(a), 0,0,0,0,0,0; S_m^0, 0,0,0,0)$

Denote the disease-free equilibrium, where

$S^0(a) = \Lambda exp\left(-\int_0^a \mu(s)ds\right)$, $S_m^0 = \frac{\Lambda_m}{\mu_m}$, and

$N_h^0 = \int_0^\infty S^0(a)da.$

To determine whether infection can invade the population, we linearize the system around ε_0 and restrict attention to the infected compartments of a fixed serotype:$I(a,t)$, $E_m(t)$, $I_m(t)$.

Replacing susceptible variables by their equilibrium values and neglecting higher-order terms yields the linearized infection subsystem:

$$\frac{\partial I(a,t)}{\partial \mathrm{t}} + \frac{\partial I(a,t)}{\partial a} = \beta_h b(a)\frac{S^0(a)}{N_h^0} I_m(t) - \left(\gamma(a) + \mu(a)\right)I(a,t),$$

$$\frac{\mathrm{d}E_m(t)}{\mathrm{dt}} = \beta_m \frac{\int_0^\infty b(a)I(a,t)da}{N_h^0} S_m^0 - (\kappa_m + \mu_m)E_m(t),$$

$$\frac{\mathrm{d}I_m(t)}{\mathrm{dt}} = \kappa_m E_m - \mu_m I_m(t). \quad (3.21)$$

At the invasion stage, cross-immunity compartments vanish and the two serotypes evolve independently. Hence, it is sufficient to compute the reproduction number for a single serotype. Operator Formulation: Let $x(t) = (I(.,t), E_m(t), I_m(t))$ belong to the Banach space $X = L^1(0,\infty) \times R^2$.

The linearized system can be written abstractly as

$$\frac{dx}{dt} = \mathcal{F}\, x - \mathcal{V}x, \quad (3.22)$$

Where, $\mathcal{F}$ represents new infection terms and $\mathcal{V}$ represents transition and removal terms.

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The infection Operator $\mathcal{F}$ is defined by

$$\mathcal{F}(\mathrm{I}, E_m, I_m) = \begin{pmatrix} \beta_h b(a)\frac{S^0(a)}{N_h^0} I_m \\ \beta_m \frac{S_m^0}{N_h^0}\int_0^\infty b(a)I(a)da \\ 0 \end{pmatrix}$$

And the Transition Operator is

$$\mathcal{V}(\mathrm{I}, E_m, I_m) = \begin{pmatrix} \frac{\partial I}{\partial a} + (\gamma(a) + \mu(a))I \\ (\kappa_m + \mu_m)E_m \\ \mu_m I_m - \kappa_m E_m \end{pmatrix}$$

The operator $\mathcal{V}$ generates a positive semigroup on X.Following the next-generation operator theory for structured populations [8,18], the next-generation operator is defined as $\mathcal{K} = \mathcal{F}\mathcal{V}^{-1}$,

the basic reproduction number is$R_0 = \rho(\mathcal{K})$ Where , ρ(.) denotes the spectral radius.

To compute R_0, we solve homogeneous part of the human transport equation along characteristics: $\frac{d}{da}I(a) = -\big(\gamma(a) + \mu(a)\big)I(a)$. (3.23)

Solving this equation gives

$$I(a) = I(0)\exp\big(-\int_0^a(\gamma(s) + \mu(s))ds\big)$$

The survival probability of an infected individual up to age a is define

$$\pi(a) = \exp\big(-\int_0^a(\gamma(s) + \mu(s))ds\big) \quad (3.24)$$

An infected human generates exposed mosquitoes at rate proportional to the transmission coefficient β_m, while exposed mosquitoes survive the incubation period with probability $\frac{\kappa_m}{\kappa_m+\mu_m}$ Infectious mosquitoes then survive on average $\frac{1}{\mu_m}$ time units and transmit infection to humans at rate β_h.

Combining the human-to-mosquito and mosquito-to-human transmission steps yields the reproduction number

$$R_0 = \frac{\beta_h\beta_m S_m^0}{\mu_m N_h^0}\frac{\kappa_m}{\kappa_m+\mu_m}\int_0^\infty b(a)S^0(a)exp\big(-\int_0^a(\gamma(s) + \mu(s))ds\big)da \quad (3.25)$$

Since the two serotypes evolve independently at the disease-free equilibrium, the next-generation operator is block diagonal. Consequently, the overall basic reproduction number is given by

$$R_0 = \max\{R_{01}, R_{02}\} \quad (3.26)$$

where R_{0i} is given by (3.25) with strain-specific parameters.

3.4.2.Stability of the Disease-Free Equilibrium

We now establish the threshold behaviour of the disease-free equilibrium in terms of the basic reproduction number R_0.

Theorem 3.3: Let ε_0 denote the disease-free equilibrium defined by (3.19). Then, If $R_0 < 1$, the disease-free equilibrium ε_0 is locally asymptotically stable. If $R_0 > 1$, the disease-free equilibrium ε_0 is unstable.

Proof: From the linearization derived in Section 3.4.1, the infected subsystem around ε_0 can be written as

$$\frac{dx}{dt} = (\mathcal{F} - \mathcal{V})x,$$

Where $\mathcal{F}$ represents new infection terms and $\mathcal{V}$ represents transition and removal terms and the basic reproduction number is given by $R_0 = \rho(\mathcal{F}\mathcal{V}^{-1})$, where ρ(·) denotes the spectral radius. According to the next-generation operator theory for structured population models, [8,18], the spectral bound of the linearized operator $\mathcal{F} - \mathcal{V}$ satisfies: the spectral bound of the linearized generator changes sign precisely at R_0.

If $R_0 < 1$, the spectral bound is negative, implying exponential decay of all small perturbations in the infected components. Hence, the disease-free equilibrium is locally asymptotically stable.

If $R_0 > 1$, the spectral bound becomes positive, and perturbations grow exponentially. Hence the disease-free equilibrium loses stability whenever $R_0 > 1$.

3.4.3. Existence of the Endemic Equilibrium

In this section, we investigate the existence of a non-trivial steady state of the system when the basic reproduction number exceeds unity.

Definition of the Endemic Equilibrium: An endemic equilibrium $\varepsilon^*$is a steady-state solution of the full system satisfying $S^*(a) > 0, I_i^*(a) > 0, E_{m,i}^* > 0, I_{m,i}^* > 0$ for at least one serotype $i \in \{1,2\}$.

At equilibrium, all time derivatives vanish and the age-structured equations reduce to ordinary differential equations in the age variable.

Theorem 3.4: Let R_0 denote the basic reproduction number of the system. If $R_0 > 1$, then the model admits at least one endemic equilibrium with strictly positive infected components.

If $R_0 < 1$, the disease-free equilibrium is the only nonnegative equilibrium, and no endemic equilibrium exists.

Proof: We analyse the steady-state system obtained by setting all time derivatives equal to zero.

At equilibrium, the susceptible human population satisfies

$$\frac{dS^*(a)}{da} = -(\lambda_1^*(a)+\lambda_2^*(a) + \mu(a))S^*(a),$$

with boundary condition $S^*(0) = \Lambda$

Solving this equation yields

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$$S^*(a) = \Lambda \exp(-\int_0^a (\lambda_1^*(s)+\lambda_2^*(s) + \mu(s))ds\ )$$

At equilibrium, the infected human equation becomes

$$\frac{dI_i^*(a)}{da} = -(\gamma_i(a) + \mu(a))I_i^*(a) + \lambda_i^*(a)S^*(a),$$

Where, $\lambda_i^*(a)$ denotes the equilibrium force of infection. Solving this first-order linear equation along characteristics yields

$$I_i^*(a) = \int_0^a \lambda_i^*(s)S^*(s)exp\big(-\int_s^a(\gamma_i(r) + \mu(r))dr\big)ds.$$

Thus, the equilibrium infection profile depends on the cumulative force of infection and the survival probability during the infectious period.

At equilibrium, the mosquito subsystem satisfies

$$0 = \beta_{mi}\frac{S_m^*}{N_h^*}\int_0^\infty b(a)I_i^*(a)da - (\kappa_m + \mu_m)E_{m,i}^*,$$

$$0 = \kappa_m E_{m,i}^* - \mu_m I_{m,i}^*.$$

From the second equation, $I_{m,i}^* = \frac{\kappa_m}{\mu_m}E_{m,i}^*$.

Substituting into the first equation gives a consistency condition linking human and mosquito infection levels.

$$E_{m,i}^* = \frac{\beta_{mi}S_m^*}{(\kappa_m+\mu_m)N_h^*}\int_0^\infty b(a)\, I_i^*(a)da.$$

Therefore, $I_{m,i}^* = \frac{\beta_{mi}S_m^*}{\mu_m N_h^*}\frac{\kappa_m}{\kappa_m+\mu_m}\int_0^\infty b(a)\, I_i^*(a)da.$

Fixed-Point Formulation:

The equilibrium force of infection satisfies $\lambda_i^*(a) = \mathcal{T}(\lambda_i^*)(a)$ Where, $\mathcal{T}$ is a positive integral operator acting on the force of infection. The trivial solution $\lambda_i^*(a) = 0$ corresponds to the disease-free equilibrium.

Role of R_0:

The Fréchet derivative of $\mathcal{T}$ at zero coincides with the next-generation operator κ Hence, $\rho(\kappa) = R_0$

If $R_0 < 1$, then the spectral radius of the linearized operator does not exceed unity. In this case, the trivial fixed point is locally attracting in a neighbourhood of zero, and no positive fixed point exists.

If $R_0 > 1$, then the spectral radius exceeds unity. he above equations define a nonlinear positive integral operator on the feasible state space. Under the assumptions of positivity, boundedness (Theorems 3.1–3.2), and compactness of the associated solution operator, the existence of a positive fixed point follows from Schauder's fixed-point theorem. Since the linearization of this operator at the disease-free equilibrium has spectral radius R_0, a non-trivial positive solution exists whenever $R_0 > 1$.

4. NUMERICAL ILLUSTRATION

The numerical simulations are performed using parameter values obtained from epidemiological literature, official dengue surveillance reports, and biologically plausible assumptions. The recovery rate is assumed to be constant, with $\gamma = \frac{1}{7}\text{day}^{-1}$, corresponding to an average infectious period of approximately seven days, consistent with reports of the World Health Organization (WHO). The waning immunity rate is taken as $\omega = \frac{1}{40}, \frac{1}{90}, \frac{1}{200}\ \text{day}^{-1}$, representing temporary cross-immunity lasting approximately one to six months, in agreement with Ferguson et al. [11]. The cross-immunity parameter satisfies $0 < \sigma < 1$, where σ denotes the reduced susceptibility to secondary infection following the waning of temporary immunity. Representative values $\sigma = 0.2,0.4,0.7$ are considered to illustrate different levels of partial protection. The transmission coefficients are chosen as $\beta_1 = 0.5$ and $\beta_2 = 0.6$, which lie within biologically plausible ranges and produce realistic epidemic dynamics. Although the mathematical model is formulated using a continuous age variable, the numerical simulations classify the population into three representative age categories (children, adults, and elderly) to facilitate comparison with the available epidemiological data. The simulations are informed by state-wise dengue surveillance data reported by the National Centre for Vector Borne Disease Control (NCVBDC), Ministry of Health and Family Welfare, Government of India.

The state-wise distribution of reported dengue cases and deaths in India during 2021–2025 is presented in the following table based on official surveillance data reported by the National Centre for Vector Borne Disease Control (NCVBDC), Ministry of Health and Family Welfare.

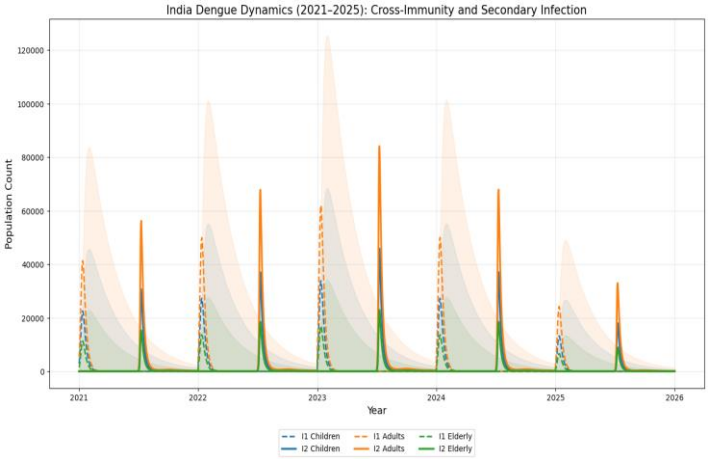

Fig.2: Age-structured dengue model with cross immunity and secondary infection.

Figure 2 illustrates the simulated age-structured dengue dynamics in India during 2021–2025, showing recurrent annual waves of primary (I_1) and secondary(I_2) infections. For

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visualization, the continuous age distribution is grouped into three representative categories: children, adults, and the elderly. The results indicate clear age-dependent transmission, with adults consistently exhibiting the highest infection burden, followed by children and the elderly. The primary infection peaks increase from 2021 to 2023, consistent with the reported rise in dengue incidence, while the reduced epidemic intensity in 2025 agrees with the decline in reported cases.

The recovered compartment (R_1) increases following each primary outbreak, reflecting the accumulation of temporarily immune individuals. As immunity wanes through the parameter ω\omegaω, individuals gradually become susceptible to secondary infection, resulting in delayed secondary epidemic waves across all age groups. The simulations indicate that age structure and temporary cross-immunity influence both the timing and magnitude of dengue outbreaks. Adults experience the largest epidemic burden, whereas children and the elderly exhibit comparatively smaller but synchronized transmission patterns, highlighting the importance of incorporating age-dependent transmission and immune dynamics in dengue modelling.

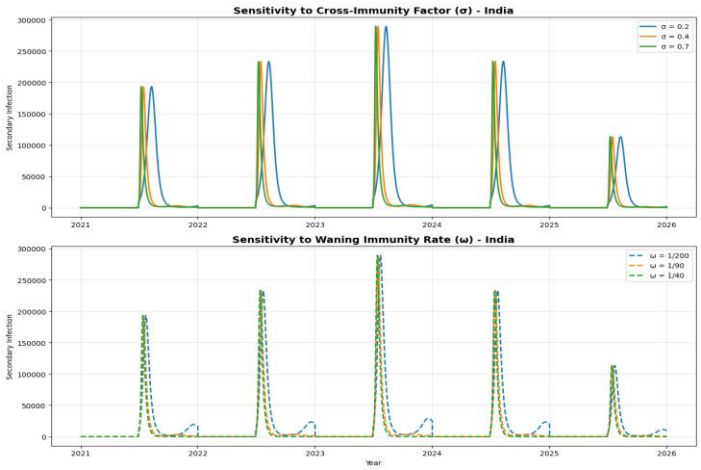

Fig.3: sensitivity to cross immunity factor σ and wanning immunity ω.

Figure 3 presents the sensitivity of the nationwide dengue model to the cross-immunity parameter (σ) and the waning immunity rate (ω). The upper panel shows that changes in σ substantially affect the magnitude and duration of secondary infections (I_2). Lower values of σ (e.g., σ = 0.2 produce broader epidemic waves with higher secondary infection peaks, particularly during 2023–2024, whereas higher values (e.g., $\sigma = 0.7$) result in earlier and narrower outbreaks. These results suggest that the level of residual protection following primary infection strongly influences the progression of secondary dengue transmission.

The lower panel illustrates the effect of the waning immunity rate (ω). Slower waning (e.g., $\omega = 1/200$ days) delays the onset of secondary outbreaks, producing wider epidemic curves with prolonged transmission. In contrast, faster waning (e.g., $\omega = 1/40$ days) accelerates the return of recovered individuals to the partially susceptible class, leading to earlier and sharper epidemic peaks. Although the peak magnitudes remain broadly comparable, the timing and persistence of outbreaks vary noticeably with different values of ω.

Overall, the sensitivity analysis indicates that both cross-immunity and waning immunity influence the recurrence and temporal pattern of dengue epidemics. These findings highlight the importance of incorporating immune dynamics into age-structured dengue models to improve the assessment of long-term transmission and support the evaluation of intervention strategies.

5. CONCLUSION

In this study, an age-structured dengue transmission model incorporating two interacting serotypes and vector–host dynamics was developed to investigate the effects of temporary cross-immunity on dengue transmission. By integrating age-dependent exposure, proportionate mixing, and waning immunity, the proposed model provides a biologically meaningful framework for describing multi-serotype dengue dynamics. The mathematical analysis establishes the positivity and boundedness of solutions and derives the basic reproduction number (R_0) as the threshold governing disease invasion and persistence. Furthermore, the stability analysis shows that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ and unstable when $R_0 > 1$, while the existence of an endemic equilibrium for $R_0 >$ 1supports the persistence of dengue transmission.

The numerical simulations reproduce recurrent epidemic patterns characterized by primary and delayed secondary outbreaks and reveal clear age-dependent differences in disease burden. The sensitivity analysis further demonstrates that both the cross-immunity parameter (σ) and the waning immunity rate (ω) significantly influence the timing and magnitude of secondary outbreaks. These findings indicate that immune dynamics play an important role in shaping the long-term behaviour of dengue epidemics and should be incorporated into mathematical models of multi-serotype transmission.

Overall, the proposed model provides a useful framework for understanding the interaction between age structure, temporary cross-immunity, and dengue transmission dynamics. It may also support the assessment of public health interventions, including vaccination strategies, vector control programmes, and age-specific surveillance. Future research may extend the present model by incorporating all four dengue serotypes, seasonal mosquito dynamics, spatial heterogeneity, and parameter estimation using epidemiological data to improve its predictive capability.

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Author Contributions

The manuscript was jointly conceptualized, developed, analyzed, and written by all authors, each of whom has read and approved the final submission.

Conflicts of interest:

The authors declare that have no conflicts of interest.

Data Availability

The data used in this study were obtained from publicly available epidemiological reports cited in the manuscript. All numerical simulations were generated using the proposed mathematical model and the parameter values reported in this study.

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