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# Transmission Dynamics of Dengue Disease Incorporating Treatment, Mass Awareness, and *Wolbachia* Intervention

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**Abstract**

Dengue Hemorrhagic Fever (DHF) remains a serious global public health threat, with its transmission dynamics strongly influenced by vector control strategies and human behavior. This study constructs and analyzes a mathematical model based on a system of differential equations to investigate the transmission dynamics of DHF by integrating three control strategies: treatment, public awareness, and the release of *Wolbachia*-infected mosquitoes. The basic reproduction number ($R_0$) is derived using the Next Generation Matrix (NGM) method and serves as a threshold parameter for disease spread. Numerical simulations show that when $R_0 < 1$, the system converges to the disease-free equilibrium, indicating that the disease will eventually die out. Conversely, by adjusting the parameter $\delta$ such that $R_0 > 1$, the system becomes stable at the endemic equilibrium, implying the persistence of the disease within the population. These findings highlight the importance of controlling key parameters through integrated intervention strategies to reduce $R_0$ below unity.

**Keywords:** Basic Reproduction Number; Dengue Hemorrhagic Fever; Mathematical Model; Stability Analysis; *Wolbachia*.



## 1. Introduction

Dengue Hemorrhagic Fever (DHF) is an infectious disease that remains a serious threat to global public health. The disease is caused by the dengue virus and is transmitted through the bites of *Aedes aegypti* and *Aedes albopictus* mosquitoes [1]. The World Health Organization (WHO) has reported a dramatic increase in global incidence, rising from 505,430 cases in 2000 to approximately 14.6 million cases in 2024, with dengue now endemic in more than 100 countries [2]. In Indonesia, the epidemiological burden of dengue continues to increase with fluctuating patterns influenced by multiple risk factors. Observational studies conducted in major cities such as Jakarta and Medan indicate that the high risk of dengue transmission is strongly associated with local environmental conditions [3–5]. Furthermore, data from the Indonesian Ministry of Health recorded a significant surge in cases in 2024, reaching 257,271 cases with 1,461 deaths, underscoring that Indonesia still faces substantial challenges in dengue outbreak management [6].

The transmission dynamics of dengue are highly complex, and control efforts involve a combination of medical and non-medical approaches. From a medical perspective, patient management through crystalloid and colloid fluid therapy has proven effective in preventing shock

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and reducing mortality, although no specific antiviral treatment is currently available [7, 8]. From a non-medical perspective, control strategies largely depend on human intervention and conventional vector control measures. These include enhancing public awareness regarding environmental sanitation to disrupt the mosquito life cycle [9, 10], as well as the application of chemical insecticides to rapidly suppress vector populations [11]. However, the effectiveness of these methods faces major challenges. Prolonged insecticide use has been shown to induce physiological resistance in mosquitoes and poses potential risks to the environment [12]. The limited long-term efficacy of conventional approaches highlights the urgent need for more sustainable biological control strategies.

In response to the limitations of conventional methods, biocontrol innovation using *Wolbachia* bacteria has emerged as a promising alternative. When introduced into *Aedes aegypti* mosquitoes, *Wolbachia* can inhibit dengue virus replication [13, 14]. Field implementation of this strategy has demonstrated encouraging results. A collaborative field study conducted by the World Mosquito Program (WMP) and Universitas Gadjah Mada (UGM) in Yogyakarta showed that the release of *Wolbachia*-infected mosquitoes reduced dengue incidence by up to 77% [15]. Numerous mathematical models have also been developed to evaluate the effectiveness of *Wolbachia*, either in combination with vaccination strategies [16, 17] or focusing solely on mosquito release interventions [18, 19]. Nevertheless, most existing models examine these strategies in isolation or primarily focus on the initial release phase.

A significant research gap remains in modeling the integration of these strategies into a comprehensive control framework. Naaly et al. (2024) developed a model incorporating treatment, public awareness, and vector control through insecticides [11]. The present study aims to modify this framework by substituting chemical control with long-term *Wolbachia*-based biological control. The novelty of this study lies not merely in variable substitution but in the alteration of the system's stability structure resulting from interaction dynamics among mosquito populations.

Unlike insecticides that directly eliminate mosquitoes, the *Wolbachia* mechanism operates through biological competition and cytoplasmic incompatibility [20]. Under this mechanism, mating between *Wolbachia*-infected males and wild females does not produce viable offspring, while matings involving *Wolbachia*-infected females result in offspring carrying *Wolbachia*, regardless of the male's infection status [13, 18]. This phenomenon leads to a decline in the wild mosquito population not through toxin-induced mortality, but through reproductive failure during interactions with *Wolbachia*-infected mosquitoes. From a modeling perspective, this process can be interpreted as biomass conversion, in which population dominance shifts from wild mosquitoes (competent vectors) to *Wolbachia*-infected mosquitoes (incompetent vectors) that become established within the ecosystem [21].

Therefore, this study constructs a novel SEITR–SEIW epidemiological model (Susceptible–Exposed–Infected–Treated–Recovered for humans and Susceptible–Exposed–Infected–*Wolbachia* for mosquitoes). The main theoretical contribution of this research lies in analyzing the dynamic system behavior in which vector reduction is driven by inter-population mosquito interactions, combined with human-centered interventions such as awareness and treatment. Through stability analysis and numerical simulations, this study is expected to provide new insights into the ecological stability thresholds required to achieve sustainable dengue elimination.

## 2. Methods

This study is a theoretical and computational investigation employing a deterministic mathematical model based on a system of ordinary differential equations (ODEs). The proposed SEITR–SEIW model is developed as a modification of the classical SEIR–SEI framework by integrating three main intervention strategies, namely human treatment, public awareness, and biological vector control using *Wolbachia* bacteria within the mosquito population. The research procedure is conducted through six systematic stages: (1) performing a comprehensive literature review to formulate fundamental assumptions, define state variables, and determine model

parameters and constraints; (2) constructing a mathematical model that accurately represents the disease transmission dynamics; (3) identifying equilibrium points and deriving the basic reproduction number ($R_0$) using the Next Generation Matrix (NGM) method; (4) conducting local stability analysis using the Routh–Hurwitz criteria to examine the solution behavior in the neighborhood of the equilibrium points; (5) performing parameter sensitivity analysis to identify key parameters that most significantly influence $R_0$; and (6) carrying out numerical simulations with selected parameter values to validate the theoretical analysis, utilizing Maple 2024 for symbolic computation and MATLAB 2025a to observe the time-series behavior of the model. The parameter values used in the simulations are adopted from the global literature and are not specifically calibrated to local incidence data. Therefore, the simulation results are intended to provide a theoretical illustration of disease transmission dynamics rather than precise quantitative predictions for a particular region.

In formulating the SEITR–SEIW model, the total population is classified according to individual health and infection status. The total human population, denoted by $N_h$, is divided into five compartments: susceptible humans ($S_h$), exposed humans who are infected but not yet infectious during the incubation period ($E_h$), infectious humans capable of transmitting the dengue virus ($I_h$), humans undergoing medical treatment due to dengue infection ($T_h$), and recovered humans ($R_h$). Meanwhile, the total mosquito population, denoted by $N_v$, consists of susceptible wild mosquitoes ($S_m$), exposed wild mosquitoes in the incubation stage ($E_m$), infectious wild mosquitoes capable of transmitting the virus ($I_m$), and mosquitoes carrying *Wolbachia* bacteria ($W$).

The model is constructed based on a set of assumptions adapted from standard epidemiological modeling frameworks. The human population is assumed to be constant under the disease-free equilibrium (DFE) condition [11], where the natural birth rate is equal to the natural death rate, and migration is neglected. All human births are assumed to enter the susceptible compartment. Disease transmission occurs via a vector-borne mechanism, where susceptible humans become infected only through bites from infectious mosquitoes, and susceptible mosquitoes become infected by biting infectious humans [2].

Regarding medical intervention, infected humans are assumed to undergo hospital-based treatment to achieve recovery. Individuals in the treatment compartment are assumed to be non-infectious due to hospitalization in facilities with strict vector isolation. According to global clinical management guidelines [22], patients in the viremic phase must be protected from mosquito bites by using physical barriers, such as bed nets, or by being placed in enclosed rooms to prevent nosocomial transmission. Recovered humans are assumed to acquire temporary immunity before returning to the susceptible class [23]. In contrast, infected wild mosquitoes are assumed to remain infectious for life and do not recover [18]. The model also incorporates disease-induced mortality, assuming that the mortality rate among treated individuals is lower than that of untreated individuals due to supportive medical intervention [11]. Due to disease-induced mortality, the assumption of a constant human population is relaxed under endemic conditions, leading to a decline in the total human population.

Mosquitoes infected with *Wolbachia* ($W$) are assumed to be incompetent vectors for dengue transmission due to viral replication inhibition [24]. Competitive interactions occur between the *Wolbachia*-infected mosquito population and the wild mosquito population ($S_m$, $E_m$, $I_m$). Based on the Cytoplasmic Incompatibility (CI) mechanism, cross-mating between these two populations leads to egg hatching failure, which is represented in the model as a reduction rate of the wild mosquito population proportional to its interaction with the *Wolbachia*-infected population [20]. The *Wolbachia* population is assumed to be established in the environment through controlled release and natural recruitment, supported by field evidence of successful dissemination in Yogyakarta [25].

Based on the compartment definitions and assumptions described above, the disease transmission dynamics are as follows.

The susceptible human population ($S_h$) increases through a constant recruitment rate $\Lambda_h$ and through the return of recovered individuals ($R_h$) who become susceptible again due to waning immunity at rate $\eta R_h$. Conversely, this population decreases due to natural mortality at a rate $\mu_h S_h$ and transitions to the exposed compartment ($E_h$) following infection. Individuals enter the exposed human class through disease transmission with an incidence rate given by $\frac{(1-\alpha)B_{mh}\,p\,S_h I_m}{N_v}$, where $\alpha$ represents the efficacy of public awareness in reducing mosquito bites, $B_{mh}$ is the transmission probability per bite from mosquitoes to humans, $p$ denotes the mosquito biting rate, and $\frac{I_m}{N_v}$ is the proportion of infectious wild mosquitoes in the total vector population. Individuals in the exposed class ($E_h$) subsequently progress to the infectious class ($I_h$) at rate $k_h E_h$, or are removed due to natural mortality at rate $\mu_h E_h$.

The infectious human population ($I_h$) may enter the treatment compartment ($T_h$) at rate $\phi I_h$. This population also decreases due to natural mortality and disease-induced mortality at a combined rate of $(\sigma_1 + \mu_h)I_h$. Furthermore, individuals undergoing treatment ($T_h$) recover and move to the recovered class ($R_h$) at a rate $\gamma T_h$, or die due to natural and disease-induced mortality at a total rate of $(\sigma_2 + \mu_h)T_h$. The recovered population ($R_h$) decreases due to natural mortality and loss of immunity, causing individuals to return to the susceptible class.

The susceptible wild mosquito population ($S_m$) increases through a constant recruitment rate $\Lambda_m$ and decreases due to natural mortality at a rate $\mu_m S_m$. Disease transmission to mosquitoes occurs when susceptible mosquitoes bite infectious humans ($I_h$), leading to a transition from $S_m$ to the exposed mosquito class ($E_m$) at an infection rate given by $\frac{B_{hm}\,p\,S_m I_h}{N_h}$, where $B_{hm}$ denotes the transmission probability per bite from humans to mosquitoes, and $\frac{I_h}{N_h}$ represents the proportion of infectious humans in the total human population. Individuals in the exposed mosquito class ($E_m$) then progress to the infectious mosquito class ($I_m$) at rate $k_m E_m$, or decrease due to natural mortality at rate $\mu_m E_m$. The infectious wild mosquito population ($I_m$) decreases due to natural mortality at the rate $\mu_m I_m$.

As a form of biological control, the model incorporates a *Wolbachia*-infected mosquito compartment ($W$), which is assumed to have an input rate of $(1 - \Lambda_m)$ and a natural mortality rate of $\mu_w W$. The presence of *Wolbachia* is assumed to exert additional mortality pressure across all life stages of wild mosquitoes. Consequently, the susceptible, exposed, and infectious wild mosquito populations ($S_m$, $E_m$, and $I_m$) experience reductions proportional to their interactions with the *Wolbachia* population, represented by the terms $\delta W S_m$, $\delta W E_m$, and $\delta W I_m$, respectively.

The interactions between these compartments are visualized in the transfer diagram shown in Fig. 1.

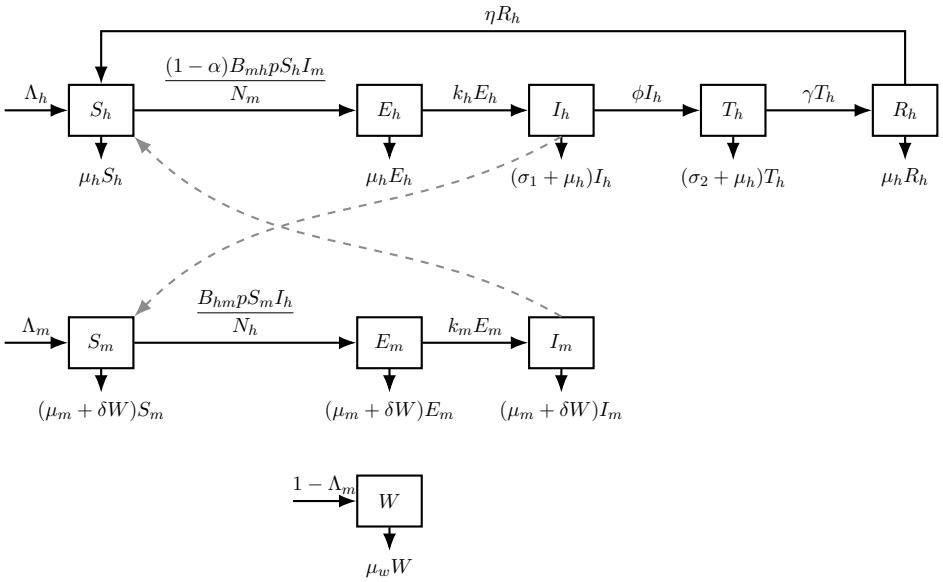



**Fig. 1:** Compartmental diagram of the SEITR–SEIW model.

Based on the transmission dynamics (Fig. 1) described above, the nonlinear system of differential equations representing the model is given as follows.

$$\begin{cases} \frac{dS_h}{dt} = \Lambda_h + \eta R_h - \frac{(1-\alpha)B_{mh}pS_hI_m}{N_v} - \mu_h S_h \\ \frac{dE_h}{dt} = \frac{(1-\alpha)B_{mh}pS_hI_m}{N_v} - (k_h + \mu_h)E_h \\ \frac{dI_h}{dt} = k_h E_h - (\phi + \sigma_1 + \mu_h)I_h \\ \frac{dT_h}{dt} = \phi I_h - (\gamma + \sigma_2 + \mu_h)T_h \\ \frac{dR_h}{dt} = \gamma T_h - (\eta + \mu_h)R_h \\ \frac{dS_m}{dt} = \Lambda_m - \frac{B_{hm}pS_mI_h}{N_h} - (\delta W + \mu_m)S_m \\ \frac{dE_m}{dt} = \frac{B_{hm}pS_mI_h}{N_h} - (k_m + \delta W + \mu_m)E_m \\ \frac{dI_m}{dt} = k_m E_m - (\delta W + \mu_m)I_m \\ \frac{dW}{dt} = (1 - \Lambda_m) - \mu_w W \end{cases} \tag{1}$$

The model is subject to the nonnegative initial conditions $S_h(0) \geq 0, E_h(0) \geq 0, I_h(0) \geq 0, T_h(0) \geq 0, R_h(0) \geq 0, S_m(0) \geq 0, E_m(0) \geq 0, I_m(0) \geq 0, W(0) \geq 0$. To simplify the analysis, new variables are introduced in the form of population proportions, defined as $s_h = \frac{S_h}{N_h}, e_h = \frac{E_h}{N_h}, i_h = \frac{I_h}{N_h}, t_h = \frac{T_h}{N_h}, r_h = \frac{R_h}{N_h}, s_m = \frac{S_m}{N_v}, e_m = \frac{E_m}{N_v}, i_m = \frac{I_m}{N_v}, w = \frac{W}{N_v}$. Accordingly, the total population proportions satisfy $s_h + e_h + i_h + t_h + r_h = 1$ and $s_m + e_m + i_m + w = 1$.

Expressed in terms of these normalized variables, the model system Eq. (1) can be rewritten in proportional form as follows:

$$\begin{cases} \frac{ds_h}{dt} = \Lambda_h + \eta r_h - (1-\alpha)B_{mh}ps_hi_m - \mu_h s_h \\ \frac{de_h}{dt} = (1-\alpha)B_{mh}ps_hi_m - (k_h + \mu_h)e_h \\ \frac{di_h}{dt} = k_h e_h - (\phi + \sigma_1 + \mu_h)i_h \\ \frac{dt_h}{dt} = \phi i_h - (\gamma + \sigma_2 + \mu_h)t_h \\ \frac{dr_h}{dt} = \gamma t_h - (\eta + \mu_h)r_h \\ \frac{ds_m}{dt} = \Lambda_m - B_{hm}ps_mi_h - (\delta w + \mu_m)s_m \\ \frac{de_m}{dt} = B_{hm}ps_mi_h - (k_m + \delta w + \mu_m)e_m \\ \frac{di_m}{dt} = k_m e_m - (\delta w + \mu_m)i_m \\ \frac{dw}{dt} = (1 - \Lambda_m) - \mu_m w \end{cases} \tag{2}$$

With initial conditions $s_h(0) \geq 0, e_h(0) \geq 0, i_h(0) \geq 0, t_h(0) \geq 0, r_h(0) \geq 0, s_m(0) \geq 0, e_m(0) \geq 0, i_m(0) \geq 0, w(0) \geq 0$.

# 3. Results and Discussion

This section presents the main analytical and numerical findings of the proposed SEITR–SEIW model. We first establish the biological feasibility of the system by proving positivity and boundedness of solutions. Next, the equilibrium points are characterized and the basic reproduction number $R_0$ is derived as a key threshold parameter governing transmission. These results are then used to study the local stability of the disease-free and endemic equilibria. Finally, parameter sensitivity analysis and numerical simulations are provided to quantify the influence of key parameters and to illustrate the model dynamics under different intervention scenarios.

## 3.1. Positivity and Boundedness of Solutions

Since system Eq. (2) represents model variables normalized into proportions, the dynamical analysis is conducted within a domain bounded between zero and one. The state variables are defined as

$$X = (s_h, e_h, i_h, t_h, r_h, s_m, e_m, i_m, w).$$

**Theorem 1.** *Let $X(t) = (s_h(t), e_h(t), i_h(t), t_h(t), r_h(t), s_m(t), e_m(t), i_m(t), w(t))$ be a solution of the differential equation system Eq. (2) with initial condition $X(0) \in \mathbb{R}^9_+$. Then, for all $t \geq 0$, the solution remains nonnegative and bounded. In other words, there exists a biologically feasible set $\Omega \subset \mathbb{R}^9_+$ such that $X(t) \in \Omega$ for all $t \geq 0$, and $\Omega$ is positively invariant.*

*Proof.* To establish the positivity of solutions, observe that each equation in system Eq. (2) is constructed such that whenever a state variable attains the value zero, its time derivative is nonnegative. For instance, when $s_h = 0$, it follows that

$$\left.\frac{ds_h}{dt}\right|_{s_h=0} \geq 0.$$

Similar arguments apply to all remaining state variables in both the human and mosquito populations. Consequently, the solution trajectories remain in $\mathbb{R}^9_+$ for all $t \geq 0$.
The boundedness of solutions follows from the system's structural properties. Human compartments are modeled as proportions, implying that each human-related variable is naturally bounded above by one. Meanwhile, the dynamics of the mosquito population, including the *Wolbachia*-infected mosquitoes, are governed by recruitment, mortality, and competitive interaction processes, which prevent unbounded growth of the solutions. Therefore, there exists a positive constant $M$ such that

$$0 \leq s_m(t),\ e_m(t),\ i_m(t),\ w(t) \leq M \quad \text{for all } t \geq 0.$$

Hence, all solutions of the system remain within the bounded region $\Omega$, and the model admits biologically meaningful solutions that are both positive and bounded. □

## 3.2. Equilibrium Points and Basic Reproduction Number ($R_0$)

Equilibrium points are obtained by setting the right-hand sides of the system Eq. (2) equal to zero. Following this approach, two types of equilibrium points are identified: the disease-free equilibrium (DFE)

$$E^0 = ({s_h}^0, {e_h}^0, {i_h}^0, {t_h}^0, {r_h}^0, {s_m}^0, {e_m}^0, {i_m}^0, w^0) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, \frac{\Lambda_m}{\frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m}, 0, 0, \frac{\delta(1-\Lambda_m)}{\mu_w}\right)$$

and the Endemic Equilibrium (EE)

$$E^* = ({s_h}^*, {e_h}^*, {i_h}^*, {t_h}^*, {r_h}^*, {s_m}^*, {e_m}^*, {i_m}^*, w^*)$$

where

$$\begin{aligned}
{s_h}^* &= \frac{\Lambda_h}{\mu_h} + \frac{{i_h}^*}{\mu_h}\left(\frac{\eta\gamma\phi}{G_2} - \frac{G_1}{k_h}\right)\\
{e_h}^* &= \frac{(\phi+\sigma_1+\mu_h){i_h}^*}{k_h}\\
{i_h}^* &= \frac{\mu_h G_1 G_2 {G_3}^2(k_m+G_3) - G_2\Lambda_h\Lambda_m k_h k_m B_{hm}B_{mh}p^2(1-\alpha)}{\Lambda_m k_m B_{hm}B_{mh}p^2(1-\alpha)(\eta\gamma\phi k_h - G_1G_2) - B_{hm}p\mu_h G_1G_2G_3(k_m+G_3)}\\
{t_h}^* &= \frac{\phi {i_h}^*}{(\gamma+\sigma_2+\mu_h)}\\
{r_h}^* &= \frac{\gamma\phi {i_h}^*}{G_2}\\
{s_m}^* &= \frac{\Lambda_m}{B_{hm}p{i_h}^* + G_3}\\
{e_m}^* &= \frac{\Lambda_m B_{hm}p{i_h}^*}{(B_{hm}p{i_h}^*+G_3)(k_m+G_3)}\\
{i_m}^* &= \frac{\Lambda_m k_m B_{hm}p{i_h}^*}{(B_{hm}p{i_h}^*+G_3)(k_m+G_3)G_3}\\
w^* &= \frac{1-\Lambda_m}{\mu_m}
\end{aligned}$$

with

$$G_1 = (k_h+\mu_h)(\phi+\sigma_1+\mu_h), \quad G_2 = (\eta+\mu_h)(\gamma+\sigma_2+\mu_h), \quad G_3 = \frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m$$

Next, we derive the basic reproduction number ($R_0$). In this process, only the infected compartments of the model, namely $e_h$, $i_h$, $e_m$, and $i_m$, are considered. We then define the transition vector describing the movement of individuals among the infected compartments. Let $\mathcal{F}$ denote the rate of appearance of new infections, and let $\mathcal{V}$ represent the rate of transfer of individuals into and out of the infected compartments.

$$\mathcal{F} = \begin{pmatrix} (1-\alpha)B_{mh}ps_h i_m \\ 0 \\ B_{hm}ps_m i_h \\ 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} (k_h+\mu_h)e_h \\ -k_h e_h + (\phi+\sigma_1+\mu_h)i_h \\ \left(k_m + \frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)e_m \\ -k_m e_m + \left(\frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)i_m \end{pmatrix}$$

By defining $F$ and $V$ as the Jacobian matrices of $\mathcal{F}$ and $\mathcal{V}$ evaluated at $E^0$, respectively, we obtain the Next Generation Matrix

$$FV^{-1} = \begin{pmatrix} 0 & 0 & K_1 & K_2 \\ 0 & 0 & 0 & 0 \\ K_3 & K_4 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

where

$$K_1 = \frac{k_m(1-\alpha)B_{mh}p\Lambda_h}{\mu_h\left(k_m + \frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)\left(\frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)} \quad K_3 = \frac{k_h B_{hm}p\Lambda_m}{(k_h+\mu_h)(\phi+\sigma_1+\mu_h)\left(\frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)}$$

$$K_2 = \frac{(1-\alpha)B_{mh}p\Lambda_h}{\mu_h\left(\frac{\delta(1-\Lambda_m)}{\mu_w}+\mu_m\right)} \qquad K_4 = \frac{B_{hm}p\Lambda_m}{(\phi+\sigma_1+\mu_h)\left(\frac{\delta(1-\Lambda_m)}{\mu_w}+\mu_m\right)}$$

The basic reproduction number is defined as the largest eigenvalue of the matrix $FV^{-1}$. Thus, we obtain:

$$R_0 = \sqrt{\frac{k_h k_m(1-\alpha)B_{mh}B_{hm}p^2\Lambda_h\Lambda_m}{\mu_h(k_h+\mu_h)(\phi+\sigma_1+\mu_h)\left(k_m+\frac{\delta(1-\Lambda_m)}{\mu_w}+\mu_m\right)\left(\frac{\delta(1-\Lambda_m)}{\mu_w}+\mu_m\right)^2}}$$

### 3.3. Stability Analysis

In this section, the local stability of the model's equilibrium points is investigated. The local stability analysis is performed using a linearization about the equilibrium points. This dynamical analysis technique is a standard procedure in mathematical epidemiology for determining the asymptotic behavior of the system, as applied in recent studies by Nurkhanifah, Suryanto, and Darti. [26]. Technically, the analysis is performed by evaluating the Jacobian matrix of the system at each equilibrium point $E^k = \left(s_h^k, e_h^k, i_h^k, t_h^k, r_h^k, s_m^k, e_m^k, i_m^k, w^k\right)$, which is generally given by:

$$J(E^k) = \begin{bmatrix} -(Q_1+\mu_h) & 0 & 0 & 0 & \eta & 0 & 0 & -Q_2 & 0 \\ Q_1 & -Q_5 & 0 & 0 & 0 & 0 & 0 & Q_2 & 0 \\ 0 & k_h & -Q_6 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi & -Q_7 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -Q_8 & 0 & 0 & 0 & 0 \\ 0 & 0 & -Q_3 & 0 & 0 & -Q_4-Q_9 & 0 & 0 & -\delta s_m \\ 0 & 0 & Q_3 & 0 & 0 & Q_4 & -k_m-Q_9 & 0 & -\delta e_m \\ 0 & 0 & 0 & 0 & 0 & 0 & k_m & -Q_9 & -\delta i_m \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_w \end{bmatrix}$$

where

$$\begin{aligned} Q_1 &= (1-\alpha)B_{mh}pi_m & Q_4 &= B_{hm}pi_h & Q_7 &= \gamma+\sigma_2+\mu_h \\ Q_2 &= (1-\alpha)B_{mh}ps_h & Q_5 &= k_h+\mu_h & Q_8 &= \eta+\mu_h \\ Q_3 &= B_{hm}ps_m & Q_6 &= \phi+\sigma_1+\mu_h & Q_9 &= \frac{\delta(1-\Lambda_m)}{\mu_w}+\mu_m \end{aligned}$$

The stability analysis is conducted in the neighborhood of the equilibrium points $E^0$ and $E^*$. The analysis begins with the disease-free equilibrium $E^0$, since the stability of $E^*$ can subsequently be derived from the stability conditions of $E^0$. To examine the local stability of $E^0$, this equilibrium point is substituted into the Jacobian matrix to determine the eigenvalues that govern its stability properties. After substituting $E^0$, the Jacobian matrix $J(E^0)$ is obtained.

$$J(E^0) = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & \eta & 0 & 0 & -D_7 & 0 \\ 0 & -D_1 & 0 & 0 & 0 & 0 & 0 & D_7 & 0 \\ 0 & k_h & -D_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi & -D_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -D_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\frac{B_{hm}p\Lambda_m}{D_5} & 0 & 0 & -D_5 & 0 & 0 & -\frac{\delta\Lambda_m}{D_5} \\ 0 & 0 & \frac{B_{hm}p\Lambda_m}{D_5} & 0 & 0 & 0 & -k_m-D_5 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & k_m & -D_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_w \end{bmatrix}$$

where

$$D_1 = k_h + \mu_h \qquad D_2 = \phi + \sigma_1 + \mu_h \qquad D_3 = \gamma + \sigma_2 + \mu_h$$
$$D_4 = \eta + \mu_h \qquad D_5 = \frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m \qquad D_6 = \frac{(1-\alpha)B_{mh}p\Lambda_h}{\mu_h}$$

The eigenvalues ($\lambda$) of $J(E^0)$ are obtained by solving the characteristic equation $|J(E^0)-\lambda I| = 0$, resulting in the following equation:

$$(-\mu_h-\lambda)(-D_3-\lambda)(-D_4-\lambda)(-D_5-\lambda)(-\mu_w-\lambda)(\lambda^4+U\lambda^3+X\lambda^2+Y\lambda+Z)=0 \quad (3)$$

where the coefficients are defined as:

$$U = k_m + k_h + 2\mu_m + 2\mu_h + \phi + \sigma_1 + 2\left(\frac{\delta(1-\Lambda_m)}{\mu_w}\right)$$
$$X = \left(k_m + \frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)\left(\frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right) +$$
$$(2\mu_h + k_h + \phi + \sigma_1)\left(k_m + \frac{2\delta(1-\Lambda_m)}{\mu_w} + 2\mu_m\right) + (k_h+\mu_h)(\phi+\sigma_1+\mu_h)$$
$$Y = (k_h + 2\mu_h + \phi + \sigma_1)\left(k_m + \frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)\left(\frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right) +$$
$$(k_h+\mu_h)(\phi+\sigma_1+\mu_h)\left(k_m + \frac{2\delta(1-\Lambda_m)}{\mu_w} + 2\mu_m\right)$$
$$Z = \frac{k_h k_m (1-\alpha)B_{mh}B_{hm}p^2\Lambda_h\Lambda_m}{\mu_h(k_h+\mu_h)(\phi+\sigma_1+\mu_h)\left(k_m + \frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)\left(\frac{\delta(1-\Lambda_m)}{\mu_w} + \mu_m\right)^2} - 1$$

It is evident that five eigenvalues from Eq. (3) are negative:

$$\lambda_1 = -\mu_h, \quad \lambda_2 = -\gamma - \sigma_2 - \mu_h, \quad \lambda_3 = -\eta - \mu_h, \quad \lambda_4 = -\frac{\delta(1-\Lambda_m)}{\mu_w} - \mu_m, \quad \lambda_5 = -\mu_w.$$

Next, the remaining eigenvalues are determined from the characteristic Eq. (4).

$$\lambda^4 + U\lambda^3 + X\lambda^2 + Y\lambda + Z = 0 \quad (4)$$

The coefficients $U$, $X$, and $Y$ are positive. Therefore, the main focus of the stability analysis lies on the constant term $Z$, which is directly related to the epidemiological threshold. Since the basic reproduction number $R_0$ in vector-borne disease models is commonly defined as the geometric mean of the transmission cycle (human–mosquito–human), the relationship between the coefficient $Z$ and the basic reproduction number is given by $Z = R_0^2 - 1$.

Based on the proposed model's structure, the *Wolbachia* intervention affects the system through a dual control mechanism: reducing the recruitment of wild mosquitoes and suppressing their reproductive potential via competitive interactions, as governed by the parameter $\delta$. This mechanism is reflected in the formulation of $R_0$, where $w = \frac{1-\Lambda_m}{\mu_w}$ represents the equilibrium population of *Wolbachia*-infected mosquitoes.

According to the Routh–Hurwitz criterion [27], since all other coefficients are positive, the condition for all roots of the characteristic equation to have negative real parts (i.e., for the system to be stable) reduces to $Z < 0$. This condition is equivalent to

$$R_0^2 - 1 < 0 \quad \Longleftrightarrow \quad R_0 < 1.$$

This result highlights the crucial role of the recruitment strategy. By setting the recruitment rate of *Wolbachia*-infected mosquitoes to $(1-\Lambda_m)$, the intervention not only increases the population $w$, which suppresses the wild mosquito population through biological interactions,

but also competitively reduces the initial input of wild mosquitoes ($\Lambda_m$) into the system. If the proportion of *Wolbachia* release is sufficiently large to drive $R_0$ below unity, then the disease-free equilibrium $E^0$ is locally asymptotically stable, implying that the disease will eventually be eliminated from the population.

Conversely, when the intervention parameters are insufficient such that $R_0 > 1$, the stability of $E^0$ is lost because the condition $Z < 0$ is no longer satisfied. This instability indicates that even a small perturbation, such as the introduction of a single infected individual, can drive the system away from the disease-free state.

Given that the population system is bounded and that the endemic equilibrium $E^*$ exists positively only when $R_0 > 1$, the asymptotic behavior of the system shifts toward the endemic equilibrium. Consequently, it can be concluded that the equilibrium point $E^*$ is locally asymptotically stable when $R_0 > 1$. This condition represents a scenario in which the control strategies fail to reduce the transmission rate below the critical threshold, leading to disease persistence within the population and convergence to a constant endemic level, as represented by the state variables at $E^*$.

### 3.4. Parameter Sensitivity Analysis

In this section, the parameters that have the most significant influence on the basic reproduction number $R_0$ are identified. To determine the dominant parameters affecting the disease transmission dynamics, the normalized sensitivity indices of $R_0$ with respect to the model parameters are computed. This sensitivity analysis approach is a standard method in mathematical epidemiology for formulating efficient control strategies, as applied by Resmawan and Yahya [28] in their analysis of a COVID-19 transmission model. The formula for computing the sensitivity index of a parameter is given as follows.

$$C_{\psi}^{R_0} = \frac{\partial R_0}{\partial \psi} \times \frac{\psi}{R_0}$$

where $\psi : \Lambda_h, \Lambda_m, B_{mh}, B_{hm}, \alpha, p, k_h, k_m, \mu_m, \mu_h, \mu_w, \phi, \sigma_1$, and $\delta$.

**Table 1:** Model parameters, descriptions, values, and references

| Parameter | Description | Value | Reference |
|---|---|---|---|
| $\Lambda_h$ | Human recruitment rate (/day) | 0.0000457 | Assumption |
| $\Lambda_m$ | Wild mosquito recruitment rate (/day) | 0.6 | [18] |
| $\mu_h$ | Natural death rate of humans (/day) | 0.0000457 | [11] |
| $\mu_m$ | Natural death rate of mosquitoes (/day) | 0.0714 | [17] |
| $\eta$ | Immunity waning rate in recovered humans (/day) | 0.011 | [11] |
| $\alpha$ | Effectiveness coefficient of public awareness (/day) | 0.1 | [11] |
| $\gamma$ | Human recovery rate (/day) | 0.14286 | [11] |
| $p$ | Mosquito biting rate (/day) | 0.5 | [11] |
| $\phi$ | Rate of infected individuals receiving treatment (/day) | 0.1 | [11] |
| $\sigma_1$ | Disease-induced death rate of infected humans (/day) | 0.01 | [11] |
| $\sigma_2$ | Disease-induced death rate of treated humans (/day) | 0.005 | Assumption |
| $\delta$ | Competitive interaction coefficient (/day) | 0.07 | Assumption |
| $B_{mh}$ | Transmission probability from infected mosquitoes to susceptible humans | 0.75 | [11] |
| $B_{hm}$ | Transmission probability from infected humans to susceptible mosquitoes | 0.375 | [11] |
| $k_h$ | Incubation rate in humans (/day) | 0.1667 | [11] |
| $k_m$ | Incubation rate in mosquitoes (/day) | 0.1428 | [11] |

The selection of the human recruitment rate parameter $\Lambda_h$ is based on the assumption that the total human population remains constant over time. By adopting the natural human mortality rate $\mu_h = 0.0000457$ from [11], population balance is maintained by setting $\Lambda_h = \mu_h = 0.0000457$. This assumption ensures that human births compensate for natural deaths, resulting in a closed and demographically stable population.

Furthermore, the choice of the disease-induced death rate for treated individuals, $\sigma_2$, is motivated by the assumption that individuals receiving medical treatment experience a lower mortality risk compared to untreated infectious individuals. By adopting the disease-induced death rate for infectious humans $\sigma_1 = 0.01$ from [11], the parameter $\sigma_2$ is set to 0.005, reflecting the protective effect of medical intervention.

Meanwhile, the competitive interaction coefficient $\delta$ is determined through a threshold analysis aimed at identifying the critical stability condition of the system, namely when the basic reproduction number satisfies $R_0 = 1$ (equivalently, $Z = 0$). Analytical calculations yield $\delta = 0.045$ as the critical threshold value. Specifically, when $\delta = 0.045$, the system satisfies $R_0 = 1$; for $\delta < 0.045$, $R_0 > 1$ indicating disease persistence; whereas for $\delta > 0.045$, $R_0 < 1$ implying disease elimination. Consequently, the parameter $\delta$ is varied to examine the model's dynamic behavior.

From a theoretical perspective, the parameter $\delta$ lies within the interval $0 \leq \delta \leq 1$. However, to preserve biological realism, the analysis is restricted to relatively small values, namely $\delta < 0.1$. Larger values of $\delta$ would represent near-perfect suppression of wild mosquito reproduction, which is biologically implausible. In natural settings, cytoplasmic incompatibility induced by *Wolbachia* does not completely eliminate reproductive success in wild mosquito populations, as it is influenced by environmental factors, population heterogeneity, and mating dynamics. Therefore, restricting $\delta < 0.1$ allows the model to capture strong yet realistic competitive effects while maintaining stability and interpretability of the system dynamics.

Based on the derived expression of $R_0$ and the parameter values listed in Table 1, normalized sensitivity indices are computed for each parameter. The resulting numerical values are presented in Table 2.

**Table 2:** Sensitivity indices of model parameters with respect to $R_0$

| **Parameter** | **Value** | **Sensitivity Index** | **Impact on $R_0$** |
|---|---|---|---|
| $\Lambda_m$ | 0.6 | 2.254017 | Positive (Increasing $R_0$) |
| $\delta$ | 0.15 | -1.169345 | Negative (Reducing $R_0$) |
| $\mu_w$ | 0.0714 | 1.169345 | Positive (Increasing $R_0$) |
| $p$ | 0.5 | 1.000000 | Positive (Increasing $R_0$) |
| $\mu_h$ | 0.0000457 | -0.500345 | Negative (Reducing $R_0$) |
| $\Lambda_h$ | 0.0000457 | 0.500000 | Positive (Increasing $R_0$) |
| $B_{mh}$ | 0.75 | 0.500000 | Positive (Increasing $R_0$) |
| $B_{hm}$ | 0.375 | 0.500000 | Positive (Increasing $R_0$) |
| $\phi$ | 0.1 | -0.454357 | Negative (Reducing $R_0$) |
| $k_m$ | 0.1428 | 0.382248 | Positive (Increasing $R_0$) |
| $\mu_m$ | 0.0714 | -0.212903 | Negative (Reducing $R_0$) |
| $\alpha$ | 0.1 | -0.055556 | Negative (Reducing $R_0$) |
| $\sigma_1$ | 0.01 | -0.045436 | Negative (Reducing $R_0$) |
| $k_h$ | 0.1667 | 0.000137 | Positive (Increasing $R_0$) |

The sensitivity analysis results indicate that the sensitivity indices of the parameters $\delta$, $\mu_h$, $\phi$, $\mu_m$, $\alpha$, and $\sigma_1$ have a negative impact on $R_0$. This implies that increasing the values of these parameters decreases the basic reproduction number. Conversely, the sensitivity indices of the parameters $\Lambda_m$, $\mu_w$, $p$, $\Lambda_h$, $B_{mh}$, $B_{hm}$, $k_m$, and $k_h$ exhibit a positive impact on $R_0$, meaning that an increase in these parameters results in a higher value of the basic reproduction number.

Furthermore, it can be observed that the parameters $\Lambda_m$, $\delta$, and $\mu_w$ exert the most significant influence on variations in $R_0$. This finding suggests that the most effective strategy to mitigate dengue transmission should focus on controlling mosquito populations through a *Wolbachia*-based intervention. Such efforts can be implemented by reducing the recruitment rate of wild mosquitoes ($\Lambda_m$), regulating the natural death rate of *Wolbachia*-infected mosquitoes ($\mu_w$), and increasing the intensity of interactions between *Wolbachia*-infected and wild mosquito populations ($\delta$).

Enhancing this interaction is crucial for suppressing the wild mosquito population, which is the primary vector for dengue transmission.

### 3.5. Numerical Simulation

In this section, numerical simulations are presented in MATLAB R2025a to illustrate the dynamics of dengue transmission under the proposed SEITR–SEIW model. The initial population proportions used in the simulations are given by

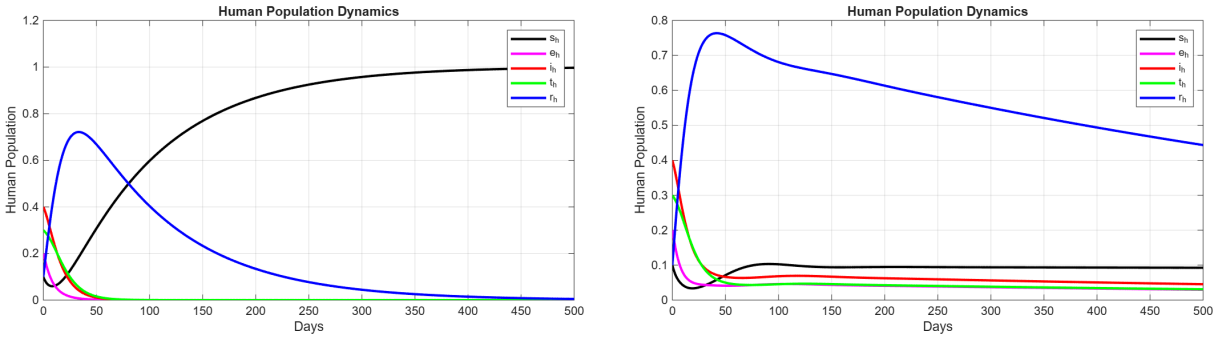
$$(s_h, e_h, i_h, t_h, r_h, s_m, e_m, i_m, w) = (0.1, 0.2, 0.4, 0.3, 0.1, 0.1, 0.2, 0.4, 0.3).$$

The first numerical simulation is conducted using the parameter values listed in Table 1. Based on the computation, the basic reproduction number is obtained as $R_0 = 0.615$. Since $R_0 < 1$, this result indicates that dengue transmission cannot be sustained in the population, and the system evolves toward the disease-free equilibrium. According to the local stability analysis, when $R_0 < 1$, the disease-free equilibrium is locally asymptotically stable. Consequently, the solution trajectories converge to the disease-free equilibrium $E^0$, given by

$$E^0 = (s_h^0, e_h^0, i_h^0, t_h^0, r_h^0, s_m^0, e_m^0, i_m^0, w^0) = (1, 0, 0, 0, 0, 1.3, 0, 0, 5.6).$$

The second numerical simulation is performed by reducing the interaction intensity between wild and *Wolbachia*-infected mosquitoes, achieved by setting $\delta$ to 0.01. Under this scenario, the computed basic reproduction number increases to $R_0 = 3.35$. Since $R_0 > 1$, this result indicates the occurrence of a dengue outbreak. From the stability analysis, when $R_0 > 1$, the endemic equilibrium is locally asymptotically stable. The system trajectories converge to the endemic equilibrium $E^*$, which is given by

$$E^* = (s_h^*, e_h^*, i_h^*, t_h^*, r_h^*, s_m^*, e_m^*, i_m^*, w^*) = (0.09, 0.03, 0.046, 0.032, 0.4, 4.2, 0.2, 0.22, 5.6).$$

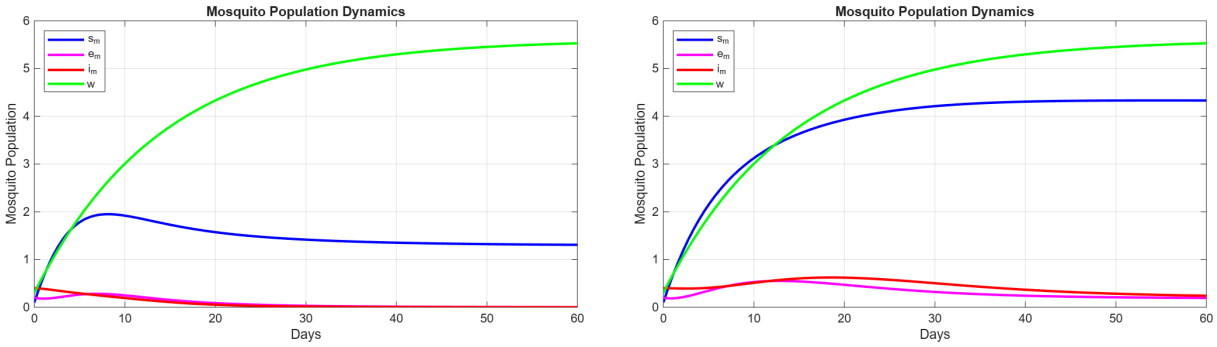



**(a)** **(b)**

**Fig. 2:** Simulation of the system dynamics in the human population

Under the DFE condition (Fig. 2a), the exposed, infected, and treated human populations appear only in the early period and subsequently decline to zero. The recovered human population also dominates at the initial stage; however, this dominance is not sustained, and the population eventually decreases to zero. In contrast, the susceptible human population increases gradually and ultimately dominates the system, indicating that disease transmission cannot be maintained in the long term. This behavior confirms that the system converges to the disease-free equilibrium. Under the EE condition (Fig. 2b), all human population compartments converge to positive equilibrium values. The recovered human population dominates at the initial stage, but decreases over time without approaching zero. The susceptible human population does not dominate the system at the early stage due to ongoing transmission; however, as time progresses, it becomes dominant again as the recovered population declines. Overall, the human population decreases

due to disease-induced mortality. The persistence of all human population compartments indicates that dengue infection remains widespread, albeit at a relatively low endemic level.




**(a)** **(b)**

**Fig. 3:** Simulation of the system dynamics in the vector population

Under the DFE condition (Fig. 3a), the exposed and infectious wild mosquito populations decline toward zero, while the *Wolbachia*-infected mosquito population increases and becomes dominant. This dominance effectively suppresses virus transmission from mosquitoes to humans, thereby interrupting the transmission cycle. Under the EE condition (Fig. 3b), all mosquito population compartments converge to positive equilibrium values. Although the exposed and infectious wild mosquito populations remain at relatively low levels due to the dominance of *Wolbachia*-infected mosquitoes, transmission is not completely eliminated. This persistence is reflected in the continued presence of human infections, as shown in Fig. 2b.

Overall, the comparison between Fig. 2 and Fig. 3 highlights that human population dynamics are strongly driven by mosquito population dynamics as the disease vector. The dominance of *Wolbachia*-infected mosquitoes effectively suppresses the infectious mosquito population and reduces the intensity of dengue transmission.

### 3.6. Discussion

In this section, a comparison is presented between the present study and previous related research. Based on the results obtained, the proposed SEITR–SEIW model can be regarded as an extension of earlier studies. Structurally, this model shares similarities with the work in [11], which investigated the effects of mass awareness, treatment, and insecticide use on the transmission dynamics of Dengue Fever. The main distinction between the present study and the model in [11] lies in the explicit incorporation of *Wolbachia*-infected mosquitoes. In [11], the reduction in mosquito populations within the compartments $S_m$, $E_m$, and $I_m$ was modeled as direct mortality resulting from insecticide application. In contrast, the model developed in this study introduces an additional compartment representing *Wolbachia*-infected mosquitoes. The presence of this compartment alters overall mosquito population dynamics, leading to a reduction in the wild mosquito population ($S_m$, $E_m$, and $I_m$) through interactions with *Wolbachia*-infected mosquitoes. The inclusion of the *Wolbachia* mosquito compartment is intended to explicitly capture its influence on the dynamics of the wild mosquito population. This aspect is crucial, since in the proposed model the decline in wild mosquito populations is governed by interaction terms of the form $\delta S_m W$, $\delta E_m W$, and $\delta I_m W$, which directly depend on the size of the *Wolbachia*-infected mosquito population ($W$). Consequently, the explicit representation of the $W$ compartment is essential to consistently and quantitatively describe the competitive effects between wild mosquitoes and *Wolbachia*-infected mosquitoes.

The analytical results obtained from the extended SEITR–SEIW model indicate that the combined implementation of treatment, enhanced mass awareness, and the release of *Wolbachia*-infected mosquitoes is effective in reducing the transmission rate of Dengue Fever. Therefore, this comparative analysis demonstrates that the present study successfully extends existing

models into a new modeling framework that incorporates alternative and complementary control strategies.

## 4. Conclusion

In this article, we developed a SEITR–SEIW model consisting of nine compartments, namely susceptible humans, exposed humans, infected humans, treated humans, recovered humans, susceptible mosquitoes, exposed mosquitoes, infected mosquitoes, and *Wolbachia*-infected mosquitoes. The proposed SEITR–SEIW model admits two equilibrium points: the disease-free equilibrium and the endemic equilibrium. The disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, whereas the endemic equilibrium is locally asymptotically stable when $R_0 > 1$. The results of the sensitivity analysis indicate that the parameters $\Lambda_m$, $\delta$, and $\mu_w$ have a significant influence on the basic reproduction number $R_0$. These findings provide theoretical support for the idea that mosquito population control, particularly through mechanisms associated with *Wolbachia*-infected mosquitoes, may play an important role in suppressing the transmission of Dengue Fever. Nevertheless, this interpretation remains conceptual, as the developed model does not incorporate economic costs, operational feasibility, or practical implementation constraints in real-world settings.

The model proposed in this study still relies on certain simplifying assumptions in representing the dynamics of *Wolbachia*-infected mosquito populations. n particular, the impact of interactions between wild and *Wolbachia*-infected mosquitoes on wild mosquito recruitment is not explicitly modeled; instead, it is represented indirectly through variations in recruitment parameters. Therefore, future research may extend the current framework by explicitly linking the interaction intensity between these two mosquito populations to the recruitment process, thereby providing a more comprehensive and biologically realistic representation of the system.

## CRediT Authorship Contribution Statement

**Rafika Nanda Agustina:** Conceptualization, Methodology, Software, Formal Analysis, Investigation, Writing – Original Draft, Visualization. **Budi Priyo Prawoto:** Conceptualization, Methodology, Validation, Writing – Review & Editing, Supervision.

## Declaration of Generative AI and AI-assisted technologies

During the preparation of this work, the authors utilized Gemini 3 Pro was employed to assist with conceptual organization, structural refinement, and preliminary drafting, while Deepl and Grammarly was used to enhance grammatical accuracy and sentence fluency. The authors reviewed and edited the content as needed and take full responsibility for the content of the publication.

## Declaration of Competing Interest

The authors declare no competing interests.

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## Data and Code Availability

All data and source code used in this study are openly available and can be accessed via public repositories.

## References


[1] H. Harapan, A. Michie, R. T. Sasmono, and A. Imrie. "Dengue: A minireview". In: *Viruses* 12.8 (Aug. 2020), p. 829. DOI: 10.3390/v12080829.

[2] World Health Organization. *Dengue and severe dengue*. Accessed: 2024. 2024. https://www.who.int/news-room/fact-sheets/detail/dengue-and-severe-dengue.

[3] A. Sofyan Anas et al. "Faktor Risiko Penyakit Demam Berdarah Dengue (Risk Factors for Dengue Fever): Artikel Review". In: *Jurnal Kolaboratif Sains* 8.6 (2025), pp. 3169–3176. DOI: 10.56338/jks.v8i6.7913.

[4] M. Sobari, I. G. N. M. Jaya, and B. N. Ruchjana. "Spatial Analysis of Dengue Disease in Jakarta Province". In: *CAUCHY: Jurnal Matematika Murni dan Aplikasi* 7.4 (May 2023), pp. 535–547. DOI: 10.18860/ca.v7i4.17423.

[5] R. Hutapea and I. Husein. "Model Koefisien Bervariasi Spasial Bayesian untuk Memperkirakan Risiko Relatif Penyakit Demam Berdarah Dengue di Kota Medan". In: *Jurnal Matematika Juli* 5 (2025). https://ejurnal.unisap.ac.id/leibniz/index.

[6] Kementerian Kesehatan RI. *Profil Kesehatan Indonesia 2024*. Kementerian Kesehatan Republik Indonesia. 2024. https://kemkes.go.id/id/profil-kesehatan-indonesia-2024.

[7] A. A. Hershan. "Dengue Virus: Molecular Biology and Recent Developments in Control Strategies, Prevention, Management, and Therapeutics". In: *Journal of Pharmacy and Bioallied Sciences* (June 2023). DOI: 10.1177/0976500X231204401.

[8] M. Palanichamy Kala, A. L. St. John, and A. P. S. Rathore. "Dengue: Update on Clinically Relevant Therapeutic Strategies and Vaccines". In: *Current Treatment Options in Infectious Diseases* 15.2 (Apr. 2023), pp. 27–52. DOI: 10.1007/s40506-023-00263-w.

[9] M. Narendran, S. Chate, and R. Patil. "Community-based intervention to dengue prevention: Insights from urban residents in Pune, using the health belief model". In: *Clinical Epidemiology and Global Health* 30 (Nov. 2024). DOI: 10.1016/j.cegh.2024.101779.

[10] C. A. Djuma, N. Achmad, A. R. Nuha, I. K. Hasan, and A. Arsal. "Model Matematika Penyebaran Penyakit Demam Berdarah Dengue dengan Faktor Kesadaran Sosial: Analisis dan Simulasi". In: *Jambura Journal of Mathematics* 7.2 (Aug. 2025). DOI: 10.37905/jjom.v7i2.33921.

[11] B. Z. Naaly, T. Marijani, A. Isdory, and J. Z. Ndendya. "Mathematical modeling of the effects of vector control, treatment and mass awareness on the transmission dynamics of dengue fever". In: *Computer Methods and Programs in Biomedicine Update* 6 (Jan. 2024). DOI: 10.1016/j.cmpbup.2024.100159.

[12] W. H. Cahyati, N. Siyam, and E. Nugroho. "Distribution of Voltage-gated Sodium Channel Mutations in Aedes Aegypti Populations from Rural Areas of Indonesia". In: *The Open Public Health Journal* 16.1 (Jan. 2024). DOI: 10.2174/0118749445255879231003110635.

[13] A. Ahamed, S. Ali, and M. Hoque. "Wolbachia-Based biocontrol of Aedes aegypti: Current Progress, Challenges, and future prospects". In: *Journal of Invertebrate Pathology* (Feb. 2025), p. 108468. DOI: 10.1016/j.jip.2025.108468.

[14] K. L. Anders et al. "Reduced dengue incidence following deployments of Wolbachia-infected Aedes aegypti in Yogyakarta, Indonesia: A quasi-experimental trial using controlled interrupted time series analysis". In: *Gates Open Research* 4 (2020). DOI: 10.12688/gatesopenres.13122.1.

[15] A. Utarini, C. Indriani, R. A. Ahmad, et al. "Efficacy of Wolbachia-Infected Mosquito Deployments for the Control of Dengue". In: *New England Journal of Medicine* 384.23 (June 2021), pp. 2177–2186. DOI: 10.1056/nejmoa2030243.

[16] A. Sa'adah and D. K. Sari. "Mathematical Models of Dengue Transmission Dynamics with Vaccination and Wolbachia Parameters and Seasonal Aspects". In: *Barekeng: Jurnal Ilmu Matematika dan Terapan* 17.4 (Dec. 2023), pp. 2305–2316. DOI: 10.30598/barekengvol17iss4pp2305-2316.

[17] M. Z. Ndii, N. Anggriani, B. S. Djahi, S. T. Tresna, and F. Inayaturohmat. "Numerical simulations of a two-strain dengue model to investigate the efficacy of the deployment of Wolbachia-carrying mosquitoes and vaccination for reducing the incidence of dengue infections". In: *Journal of Biosafety and Biosecurity* 6.4 (Dec. 2024), pp. 244–251. DOI: 10.1016/j.jobb.2024.08.003.

[18] H. Zhang and R. Lui. "Releasing Wolbachia-infected Aedes aegypti to prevent the spread of dengue virus: A mathematical study". In: *Infectious Disease Modelling* 5 (Jan. 2020), pp. 142–160. DOI: 10.1016/j.idm.2019.12.004.

[19] S. Safaei, M. Derakhshan-sefidi, and A. Karimi. "Wolbachia: A bacterial weapon against dengue fever- a narrative review of risk factors for dengue fever outbreaks". In: *New Microbes and New Infections* (June 2025). DOI: 10.1016/j.nmni.2025.101578.

[20] A. Minwuyelet et al. "Symbiotic Wolbachia in mosquitoes and its role in reducing the transmission of mosquito-borne diseases: updates and prospects". In: *Frontiers in Microbiology* (Oct. 2023). DOI: 10.3389/fmicb.2023.1267832.

[21] Abu Hanifah Al Faruqy and Budi Priyo Prawoto. "Bilangan Reproduksi Dasar Model Penyebaran Penyakit Demam Berdarah Dengue dengan Adanya Penyebaran Bakteri Wolbachia". In: *MATHunesa: Jurnal Ilmiah Matematika* 12.02 (2024), pp. 284–291. https://doi.org/10.26740/mathunesa.v12n2.p284-291.

[22] World Health Organization. *Dengue: Guidelines for Diagnosis, Treatment, Prevention and Control*. World Health Organization. Geneva, 2009. https://www.who.int/tdr.

[23] A. Khanam, H. Gutiérrez-Barbosa, K. E. Lyke, and J. V. Chua. "Immune-Mediated Pathogenesis in Dengue Virus Infection". In: *Viruses* (Nov. 2022). DOI: 10.3390/v14112575.

[24] F. D. Frentiu et al. "Limited Dengue Virus Replication in Field-Collected Aedes aegypti Mosquitoes Infected with Wolbachia". In: *PLoS Neglected Tropical Diseases* 8.2 (2014). DOI: 10.1371/journal.pntd.0002688.

[25] W. Tantowijoyo et al. "Stable establishment of wMel Wolbachia in Aedes aegypti populations in Yogyakarta, Indonesia". In: *PLoS Neglected Tropical Diseases* 14.4 (Apr. 2020), pp. 1–13. DOI: 10.1371/journal.pntd.0008157.

[26] N. Nurkhanifah, A. Suryanto, and I. Darti. "Dynamics of Lumpy Skin Disease Model with Vaccination and Environmental Transmission". In: *CAUCHY: Jurnal Matematika Murni dan Aplikasi* 10.1 (2025), pp. 133–146. DOI: 10.18860/ca.v10i1.29969.

[27] D. Medhi, G. Sarma, and A. Shyam. "Stability Analysis of Linear Systems Using the Routh-Hurwitz Criterion: Theory and Applications". In: *Journal of Computational Analysis and Aplication* 33.8 (2024), pp. 903–907. https://www.eudoxuspress.com/index.php/pub/article/view/1496.

[28] R. Resmawan and L. Yahya. "Sensitivity Analysis of Mathematical Model of Coronavirus Disease (COVID-19) Transmission". In: *CAUCHY: Jurnal Matematika Murni dan Aplikasi* 6.2 (May 2020), pp. 91–99. DOI: 10.18860/ca.v6i2.9165.