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# MATHEMATICAL ANALYSIS OF DENGUE CONTROL THROUGH WOLBACHIA DRIVEN PATHOGEN BLOCKING IN AEDES AEGYPTI POPULATIONS

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**ABSTRACT**
Dengue remains a major vector-borne disease in tropical and subtropical regions, where conventional vector-control approaches face limitations due to operational challenges and increasing insecticide resistance. Wolbachia-based population replacement has emerged as a promising biological intervention by reducing the vector competence of *Aedes aegypti* mosquitoes; however, its effectiveness depends on complex interactions among pathogen blocking, maternal transmission, mosquito fitness, and release strategies. This study develops a deterministic dengue–Wolbachia transmission model incorporating wild-type and Wolbachia-infected mosquito populations, imperfect maternal transmission, pathogen-blocking efficacy, and continuous augmentation of Wolbachia-infected mosquitoes. The model is analysed using positivity and boundedness theory, disease-free equilibrium analysis, basic reproduction number derivation, stability analysis, sensitivity analysis, and numerical simulations. The basic reproduction number was obtained using the next-generation matrix approach to determine the threshold conditions governing dengue invasion and elimination. Numerical simulations demonstrate that increasing Wolbachia pathogen-blocking efficacy reduces transmission potential and infectious prevalence. Under the baseline intervention scenario, the model produced a reproduction number of $R_0 = 0.9292$, indicating convergence towards the disease-free equilibrium. Increasing pathogen-blocking efficacy from 0.40 to 0.9575 reduced the reproduction number from 0.9739 to 0.9292 and decreased final infectious prevalence by approximately 55%. Sensitivity analysis identified mosquito biting rate and mosquito–human transmission probabilities as dominant drivers of dengue transmission, whereas pathogen blocking contributed to reducing epidemic potential. The findings highlight that successful Wolbachia-based dengue control depends not only on pathogen-blocking efficiency but also on maintaining effective mosquito population replacement and reducing mosquito–human contact. The proposed framework provides a mathematical basis for evaluating Wolbachia interventions and identifying key biological parameters that influence dengue transmission dynamics.

**Keywords:** Dengue; *Aedes aegypti*; Wolbachia; mathematical modelling; basic reproduction number; sensitivity analysis; pathogen blocking.

## Introduction

Dengue is one of the most important vector-borne viral diseases that is mainly spread by *Aedes aegypti* and has remained a major public health problem in tropical and subtropical countries. It is influenced by favourable climatic conditions, high urbanization, population density, and the development of mosquito breeding sites (Bhatt et al., 2013, Kolimenakis et al., 2021). Although insecticides and source reduction remain important vector-control methods, their effectiveness may be limited because of insecticide resistance in *Aedes* populations and operational challenges (Moyes et al., 2017). These limitations have increased interest in sustainable biological approaches that can reduce the ability of mosquitoes to transmit dengue.

There is evidence of dengue circulation among febrile populations in Nigeria, although the true burden remains difficult to determine because of limited surveillance coverage and the presence of other pathogens causing febrile illness. Otu et al. (2019) reported dengue infection among febrile patients in Cross River State, Nigeria, while Tizhe et al. (2022) reported serological evidence of infection and molecular confirmation of DENV-1 in Adamawa State, Nigeria. The potential for dengue and other febrile diseases, such as malaria, to coexist further complicates diagnosis and highlights the importance of improved surveillance and effective control strategies (Tizhe et al., 2022).

Mathematical modelling provides a useful framework for understanding dengue transmission dynamics, determining epidemiological thresholds, and evaluating intervention strategies. Classical mosquito-borne disease models consider interactions between humans and vectors, including mosquito biting rate, mosquito survival, and transmission efficiency between vectors and hosts (Smith et al., 2012). However, conventional homogeneous population models may not adequately represent biological interventions that alter mosquito population composition, reproductive success, and vector competence.

*Wolbachia pipientis* has emerged as a promising biological-control strategy for reducing dengue transmission in *Ae. aegypti*. The wMel strain is maternally transmitted, induces cytoplasmic incompatibility, and reduces the ability of infected mosquitoes to transmit dengue virus (Walker et al., 2011). Furthermore, mathematical and experimental studies have demonstrated that Wolbachia-mediated pathogen blocking can substantially reduce dengue transmission potential (Ferguson et al., 2015; Ndii et al., 2015). The epidemiological effectiveness of wMel has also been demonstrated through the Yogyakarta cluster-randomised trial, which reported 77.1% protection against virologically confirmed symptomatic dengue and 86.2% protection against dengue-related hospitalization (Utarini et al., 2021).

Despite these advances, the effectiveness of Wolbachia-based interventions depends on several interacting biological and epidemiological mechanisms, including vertical transmission from infected females to offspring, pathogen-blocking efficiency, mosquito fitness, and maintenance of Wolbachia-infected mosquito populations. Therefore, mathematical models that integrate human transmission dynamics with both wild-type and Wolbachia-infected mosquito populations are required to understand the conditions under which Wolbachia interventions can reduce dengue transmission.

Despite previous dengue–Wolbachia modelling studies, limited models have simultaneously integrated wild-type and Wolbachia-infected mosquito dynamics, imperfect maternal transmission, pathogen-blocking efficacy, and continuous augmentation within a unified human–vector transmission framework. Therefore, this study develops and simulates a deterministic dengue–Wolbachia model incorporating imperfect maternal transmission, pathogen blocking, and continuous augmentation of Wolbachia-infected mosquitoes.

**Aim of the Study**

This study aims to develop and model the transmission of dengue using Wolbachia-infected mosquitoes and to assess the impact of the models in reducing the transmission of dengue virus.

**Specific Objectives**

i. Develop a deterministic dengue transmission model with wild-type and Wolbachia-infested mosquito populations.
ii. Determine the basic reproduction number and explain the existence and stability of the steady state of the model.
iii. Conduct sensitivity analysis of important biological and intervention parameters of the spread of dengue.
iv. Simulate the effectiveness of Wolbachia augmentation to decrease dengue transmission.

**Review of Related Literature**

**1 Epidemiology of Dengue Transmission in Tropical Regions and Nigeria**

Many factors in both climate, entomology and human are involved in the Dengue's spread. Temperature also influences Aedes aegypti development and survival, biting activity and incubation period with the virus, but in a nonlinear, rather than linear-monotonic, manner compared to a linear increase in risk with temperature (Mordecai et al., 2017). Other factors may drive urbanisation to increase the potential for transmission, but their impact depends on local infrastructure and urbanisation trends, such as the availability of artificial larval habitats and the extent of human vector contact (Kolimenakis et al., 2021).

Therefore, the reasons for the persistence of dengue in tropical regions better be attributed to the combination of environmental and demographic factors and not to only one of these factors.

The circulation of Dengue virus (DENV) has been reported in Nigeria, although with spatial and diagnostic variations. In Cross River State, 5.7% (24/420) febrile cases have been serologically diagnosed as dengue infection by Otu et al. (2019). Tizhe et al. (2022) reported the seropositivity rate of DENV IgM to be 19.4% in 424 febrile patients in Adamawa State and identified 11 cases of DENV-1 by real-time RT-PCR and sequencing, representing 2.6% of all the febrile patients tested. The proportion of anti-DENV IgG antibody was found to be high (54.1%) in the 701 samples from Abuja, Ibadan and Jos collected between 2010 and 2018 and indicated high past exposure (Ekong et al., 2022). These estimates should not be interpreted as prevalence estimates directly comparable to any other prevalence estimates due to differences in population, geographic location, sampling time and endpoint, particularly the presence of acute IgM responses and past infection (IgG serology). The geographic overlap, however, suggests that both past and ongoing transmission of DENV is occurring in separate geographic populations within Nigeria.

Another difficulty to the Nigeria burden of Dengue is diagnostic uncertainty. Both Dengue and Malaria share some similar symptoms like Fever and Dengue malaria coinfection was reported at all the study sites by Tizhe et al. (2022). This also provides a clinical overlap, increasing the risk of the clinical presentation of dengue being missed in malaria endemic countries. Available prevalence estimates based on facilities are thus only indicative of transmission and not enough to determine a uniform national prevalence. This heterogeneity is advantageous for the use of locally relevant epidemiological parameters and sensitivity analysis in mathematical modelling than just taking one prevalence estimate.

**2 Mathematical Modelling of Human to Mosquito Dengue Transmission Dynamics**

The mathematical model used for transmission of mosquito-borne diseases is based on the Ross-Macdonald model, which describes the relationship between the abundance of the

mosquitoes, the biting rates and the efficiency of the mosquitoes survival and transmission (Smith et al., 2012). The models in the modern ones are primarily of the human-disease model with compartments of susceptible, exposed and infected and recovered humans, and of the mosquito-disease model with compartments of susceptible, exposed and infected mosquitoes. This SEIR–SEI model explicitly includes intrinsic incubation in humans and extrinsic incubation in mosquitoes, and is an important mechanistic formulation that is used for the analysis of transmission of dengue (Vyhmeister et al., 2022).

The benefits of these models are dependent on the balance between the biological realism and analytical tractability. However, adding environmental variables and processes in the mosquito life cycle boosted the representation of dengue dynamics as shown by Vyhmeister et al. (2022), without introducing biological detail that might further complicate the model. As long as the mechanisms that are critical for the intervention under study remain, it is useful to still have models that are parsimonious. Changes in the survival, competence, reproduction and composition of the mosquito pool may cause changes in transmission that are not detectable in human infection compartments and biological vector-control is particularly important.

**3 Basic Reproduction Number and Stability Analysis in Disease Models**

The basic reproductive number R0 is a basic value in epidemiology which indicates the infecting capacity of an infectious agent in a susceptible population. A careful formulation of R_0for a heterogeneous population was provided by Diekmann et al (1990) and is based on the dominant eigenvalue of a positive next generation operator. Van den Driessche and Watmough (2002) provided a systematic framework for calculating $R_0$ for finite dimensional compartmental systems, in which the matrix representing transitions from infected compartments to new infections has a spectrum radius that is used to calculate $R_0$. Also a good explanation of construction and interpretation of matrices in compartmental epidemic models was presented by Diekmann et al. (2010).If the next generation model has the standard assumptions, then the following interpretations are given to the $R_0$: $R_0 < 1$ means that the disease-free equilibrium is locally asymptotically stable; $R_0 > 1$ means that the disease-free equilibrium is unstable. This threshold property is, however, for the most part local and should not be deemed automatically to be adequate for global disease elimination. Under some conditions, a stable disease-free equilibrium coexist with a stable endemic equilibrium in non-linear epidemic models even for $R_0$ <1 as discussed by van den Driessche and Watmough (2002) and Safi and Gumel (2011).

The calls for the reproduction-number analysis to be complemented by an investigation on the equilibrium and stability are well suited. In general, other arguments specific to the model are required to determine the global stability of the system, but local stability can be determined by linearizing the system about an equilibrium point, and checking the corresponding local Jacobian matrix. In general, Lyapunov methods can be used to establish the global stability of endemic and disease-free solutions for large classes of epidemic systems, as shown by Shuai and van den Driessche (2013). Under some conditions, it has also been demonstrated that nonlinear epidemiological models have Goh–Volterra type Lyapunov functions and that these models converge to an equilibrium point globally under these conditions (Safi and Gumel, 2011). These methods separate the circumstances which prevent the initial invasion of the pathogen from the more stringent conditions of persistent and elimination.

**4 Wolbachia based biological control of DengueTransmission**

Wolbachia pipientis is a biological method that can be used to diminish the transmission capacity of Ae. aegypti populations, through the replacement strategy. Experimental release of the wMel strain into Ae. aegypti resulted in high levels of maternal transmission and apparent fitness

loss with very low levels of DENV-2 transmission (Walker et al., 2011). Cytoplasmic incompatibility will provide a frequency dependent reproductive advantage because mating with an infected male and normal female will produce only a small brood, while mating with an infected female and an infected or normal male will produce normal-sized broods. This reproductive asymmetry also plays a role in spreading Wolbachia when there is a threshold of invasion that is surpassed.

This population replacement technique has been field-tested. After releases in northern Australia, wMel quickly colonized natural populations of Ae. aegypti and reached fixation within a few months and modelling of the field data suggested a relatively small fitness cost and the presence of an unstable invasion threshold (Hoffmann et al., 2011). Since then, there has been robust epidemiological evidence collected in Yogyakarta, Indonesia, in a cluster-randomised controlled trial that demonstrated a 77.1% reduction in virologically confirmed symptomatic dengue cases and 86.2% reduction in hospitalisation due to dengue compared to the untreated areas (Utarini et al., 2021).

These effects have also been found to be too complex to be just a reduction in mosquitoes. Ndii et al. (2015) created a deterministic dengue model that also incorporated seasonality of mosquito mortality, Wolbachia induced impacts on dengue transmission and mosquito lifespan and demonstrated that these extra factors can result in major decrease in human infections. To emphasize population replacement, Zhang and Lui (2020) explicitly modelled cytoplasmic incompatibility, fitness costs and fitness gains of Wolbachia infected mosquitoes, demonstrating that the establishment is dependent on the balance between the Wolbachia fitness gain and fitness loss. All these studies suggest that any realistic assessment of Wolbachia-based control should explicitly take into account pathogen blocking, mosquito fitness, maternal transmission, dynamics of cytoplasmic incompatibility and release.

**5 Research Gap and Contribution of thePresent Study**

The real deficiency is not the absence of dengue Wolbachia models, but the small number of models that combine a few key biological and epidemiological processes in a single model that are necessary for a successful intervention. These processes have been studied in combination or individually in various studies, but only a few studies have modeled them together with the dynamics of the human disease, the presence of both wild-type and Wolbachia-infected mosquitoes, imperfect maternal transmission, augmentation releases, and residual transmission by infectious mosquitoes.

In the present study, this is overcome with an explicit wild-type and Wolbachia infected vector model, coupled with a deterministic model of human dengue transmission. The imperfect maternal transmission is included in the framework, along with pathogen-blocking efficacy and continual augmentation, and these are analyzed using reproduction-number analysis, equilibrium and stability analysis, sensitivity analysis and numerical simulation. It is an integrated building, where the most important parameters in the biological and intervention part affecting the control of dengue can be identified.

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## Methodology

### 1 Mathematical Model Formulation

A deterministic compartmental model is formulated to investigate dengue transmission in the presence of wild-type and *Wolbachia*-infected *Aedes aegypti* mosquitoes.

**Table 1. State variables of the dengue–*Wolbachia* transmission model**

| Variable | Description | Unit |
|---|---|---|
| $S_h(t)$ | Susceptible humans | Individuals |
| $E_h(t)$ | Exposed humans | Individuals |
| $I_h(t)$ | Infectious humans | Individuals |
| $R_h(t)$ | Recovered humans | Individuals |
| $N_h(t)$ | Total human population | Individuals |
| $S_v(t)$ | Susceptible wild-type mosquitoes | Mosquitoes |
| $E_v(t)$ | Exposed wild-type mosquitoes | Mosquitoes |
| $I_v(t)$ | Infectious wild-type mosquitoes | Mosquitoes |
| $N_v(t)$ | Total wild-type mosquito population | Mosquitoes |
| $S_w(t)$ | Susceptible *Wolbachia*-infected mosquitoes | Mosquitoes |
| $E_w(t)$ | Exposed *Wolbachia*-infected mosquitoes | Mosquitoes |
| $I_w(t)$ | Infectious *Wolbachia*-infected mosquitoes | Mosquitoes |
| $N_w(t)$ | Total *Wolbachia*-infected mosquito population | Mosquitoes |

The human population is mainly divided into susceptible $S_h$, exposed $E_h$, infectious $I_h$, and recovered $R_h$classes, with

$$N_h = S_h + E_h + I_h + R_h. \qquad (1)$$

The wild-type mosquito population is also divided into susceptible $S_v$, exposed $E_v$, and infectious $I_v$classes,

$$N_v = S_v + E_v + I_v, \qquad (2)$$

while the *Wolbachia*-infected mosquito population is similarly divided as follow;

$$N_w = S_w + E_w + I_w. \qquad (3)$$

Mosquitoes remain infectious until death and therefore have no recovered compartment.

Let $a$ denote the mosquito biting rate, $\beta_{vh}$ the mosquito-to-human transmission probability, in which $\varepsilon \in [0, 1]$the pathogen-blocking efficacy of *Wolbachia*. The force of infection in humans is

$$\lambda_h = \frac{a\beta_{vh}}{N_h}[I_v + (1-\varepsilon)I_w]. \qquad (4)$$

The corresponding forces of infection in wild-type and *Wolbachia*-infected mosquitoes are

$$\lambda_v = a\beta_{hv}\frac{I_h}{N_h},\ \lambda_w = a\beta_{hw}\frac{I_h}{N_h}, \qquad (5)$$

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where $\beta_{hv}$ and $\beta_{hw}$ denote human-to-mosquito transmission probabilities.
The model Equations are

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \lambda_h S_h - \mu_h S_h, \\
\frac{dE_h}{dt} &= \lambda_h S_h - (\sigma_h + \mu_h) E_h, \\
\frac{dI_h}{dt} &= \sigma_h E_h - (\gamma_h + \mu_h + \delta_h) I_h, \\
\frac{dR_h}{dt} &= \gamma_h I_h - \mu_h R_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \lambda_v S_v - \mu_v S_v, \\
\frac{dE_v}{dt} &= \lambda_v S_v - (\sigma_v + \mu_v) E_v, \\
\frac{dI_v}{dt} &= \sigma_v E_v - \mu_v I_v, \\
\frac{dS_w}{dt} &= r_w \eta (1 - c) N_w + u - \lambda_w S_w - \mu_w S_w, \\
\frac{dE_w}{dt} &= \lambda_w S_w - (\sigma_w + \mu_w) E_w, \\
\frac{dI_w}{dt} &= \sigma_w E_w - \mu_w I_w.
\end{aligned} \tag{6}$$

Here, $\Lambda_h$and $\Lambda_v$are the human and wild-mosquito recruitment rates; $\mu_h$and $\mu_w$are natural mortality rates; $\sigma_h$and $\sigma_w$are progression rates; $\gamma_h$is the human recovery rate; and $\delta_h$is dengue-induced mortality. The parameters $r_w$, $\eta$, $c$, and $\Omega$denote the reproductive rate, maternal transmission efficiency, fitness cost, and continuous release rate of *Wolbachia*-infected mosquitoes, respectively. The recruitment term for *Wolbachia*-infected mosquitoes is formulated using the total *Wolbachia*-infected mosquito population $N_w$as an aggregated deterministic approximation, assuming that the reproductive contribution of adult infected mosquitoes is proportionally represented within the total infected mosquito population.

$$k_1 = \sigma_h + \mu_h,\ k_2 = \gamma_h + \mu_h + \delta_h,$$

$$k_3 = \sigma_v + \mu_v,\ k_4 = \sigma_w + \mu_w, \tag{7}$$

$$\kappa_w = \mu_w - r_w \eta (1 - c). \tag{8}$$

$$\kappa_w > 0, \tag{9}$$

that is,

$$\mu_w > r_w \eta (1 - c),$$

to ensure bounded growth of the *Wolbachia*-infected mosquito population.

**Table 2. Model parameters, baseline values, ranges, and sources**

| Parameter | Description | Baseline | Range | Unit | Source |
|---|---|---|---|---|---|
| $N_h$ | Total human population | 187,500 | Fixed | Individuals | Ogunlade et al. (2023) |
| $a$ | Mosquito biting rate | 0.30 | 0.20–0.63 | day$^{-1}$ | Ogunlade et al. (2023); Ndii (2020) |
| $\beta_{vh}$ | Mosquito to human transmission probability | 0.1976 | 0.10–0.30 | bite $^{-1}$ | Ogunlade et al. (2023) |
| $\beta_{hv}$ | Human to wild mosquito transmission probability | 0.1976 | 0.10–0.30 | bite $^{-1}$ | Ogunlade et al. (2023) |
| $\beta_{hw}$ | Human to *Wolbachia* mosquito transmission probability | 0.1976 | 0.10–0.30 | bite $^{-1}$ | Ogunlade et al. (2023) |
| $\varepsilon$ | *Wolbachia* blocking efficacy | 0.9575 | 0.40–0.96 | Dimensionless | Derived from Ogunlade et al. (2023) |
| $\sigma_h$ | Human progression rate | 0.1818 | 0.10–0.3333 | day$^{-1}$ | Ndii (2020); Ogunlade et al. (2023) |
| $\gamma_h$ | Human recovery rate | 0.20 | 0.0833–0.25 | day$^{-1}$ | Ndii (2020); Ogunlade et al. (2023) |
| $\mu_h$ | Human natural mortality rate | $3.4 \times 10^{-5}$ | $3.0 \times 10^{-5}$–$4.5 \times 10^{-5}$ | day$^{-1}$ | Ogunlade et al. (2023) |
| $\delta_h$ | Dengue induced mortality rate | 0 | 0–0.001 | day$^{-1}$ | Assumed |
| $\Lambda_h$ | Human recruitment rate | 6.375 | — | persons day$^{-1}$ | Derived |
| $\sigma_v$ | Wild mosquito incubation rate | 0.10 | 0.0667–0.1429 | day$^{-1}$ | Ndii (2020); Ogunlade et al. (2023) |
| $\sigma_w$ | *Wolbachia* mosquito incubation rate | 0.10 | 0.0667–0.1429 | day$^{-1}$ | Ndii (2020); Ogunlade et al. (2023) |
| $\mu_v$ | Wild mosquito mortality rate | 0.043 | 0.03–0.10 | day$^{-1}$ | Ogunlade et al. (2023) |
| $\mu_w$ | *Wolbachia* mosquito mortality rate | 0.068 | 0.05–0.10 | day$^{-1}$ | Ogunlade et al. (2023) |

| | | | | | |
|---|---|---|---|---|---|
| $\Lambda_v$ | Wild mosquito recruitment rate | 24,187.5 | — | mosquitoes day$^{-1}$ | Derived |
| $\eta$ | Maternal transmission efficiency | 0.90 | 0.90–1.00 | Dimensionless | Dorigatti et al. (2018); Ndii (2020) |
| $c$ | *Wolbachia* fitness cost | 0.05 | 0.05–0.15 | Dimensionless | Ndii (2020); Dorigatti et al. (2018) |
| $r_w$ | Effective reproductive rate | 0.05 | 0.02–0.07 | $day-1$ | Calibrated |
| $u$ | *Wolbachia* release rate | 4,694 | 0–7,000 | mosquitoes $day-1$ | Ogunlade et al. (2023) |

**2 Positivity and Boundedness of Solutions**

The epidemiological interpretation of system (6) requires all state variables to remain non-negative and bounded.

**Lemma 1. Positivity of Solutions**

Let

$$X(0) = \left(S_h'(0), E_h'(0), I_h'(0), R_h'(0), S_v'(0)E_v(0)I_v(0)S_w(0)E_w(0)I_w(0)\right) \in \mathbb{R}_+^{10}.$$

Then every solution of system (6) remains in $\mathbb{R}_+^{10}$for all $t \geq 0$.

**Proof**

On the boundary of the non-negative orthant,

$$\left.\frac{dS_h}{dt}\right|_{S_h=0} = \Lambda_h \geq 0, \left.\frac{dE_h}{dt}\right|_{E_h=0} = \lambda_h S_h \geq 0,$$

$$\left.\frac{dI_h}{dt}\right|_{I_h=0} = \sigma_h E_h \geq 0, \left.\frac{dR_h}{dt}\right|_{R_h=0} = \gamma_h I_h \geq 0.$$

Similarly,

$$\left.\frac{dS_v}{dt}\right|_{S_v=0} = \Lambda_v \geq 0, \left.\frac{dE_v}{dt}\right|_{E_v=0} = \lambda_v S_v \geq 0,$$

$$\left.\frac{dI_v}{dt}\right|_{I_v=0} = \sigma_v E_v \geq 0,$$

and

$$\left.\frac{dS_w}{dt}\right|_{S_w=0} = r_w\eta(1-c)N_w + u \geq 0,$$

$$\left.\frac{dE_w}{dt}\right|_{E_w=0} = \lambda_w S_w \geq 0, \left.\frac{dI_w}{dt}\right|_{I_w=0} = \sigma_w E_w \geq 0.$$

Hence, the vector field points inward or is tangent to every coordinate hyperplane. Therefore, no solution initiating in $\mathbb{R}_+^{10}$can enter the negative orthant, and all state variables remain non-negative.

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**Theorem 1. Boundedness of Solutions**
Suppose Assumption 1 holds. Then all non-negative solutions of system (6) are bounded. Moreover,

$$\Omega = \left\{X, \in, \mathbb{R}_+^{10} : N_h \leq \frac{\Lambda_h}{\mu_h}\ N_v \leq \frac{\Lambda_v}{\mu_v}\ N_w \leq \frac{u}{\kappa_w}\right\} \qquad (10)$$

is positively invariant for initial conditions in Ω.

**Proof**

Adding the human equations gives

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_h I_h \leq \Lambda_h - \mu_h N_h.$$

By the comparison theorem,

$$N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t}),$$

and hence

$$\limsup_{t\to\infty} N_h(t) \leq \frac{\Lambda_h}{\mu_h}. \qquad (11)$$

For wild-type mosquitoes,

$$\frac{dN_v}{dt} = \Lambda_v - \mu_v N_v,$$

so that

$$N_v(t) = N_v(0)e^{-\mu_v t} + \frac{\Lambda_v}{\mu_v}(1 - e^{-\mu_v t}),$$

and therefore

$$\lim_{t\to\infty} N_v(t) = \frac{\Lambda_v}{\mu_v}. \qquad (12)$$

Similarly,

$$\frac{dN_w}{dt} = u - \kappa_w N_w.$$

Since $\kappa_w > 0$,

$$N_w(t) = N_w(0)e^{-\kappa_w t} + \frac{u}{\kappa_w}(1 - e^{-\kappa_w t}),$$

and

$$\lim_{t\to\infty} N_w(t) = \frac{u}{\kappa_w}. \qquad (13)$$

Thus, all solutions are bounded. If the initial state belongs to Ω, inequalities (11)–(13) ensure that the trajectory remains in Ωfor all $t \geq 0$. Hence, Ωis positively invariant. □

**3 Disease-Free Equilibrium**

The disease-free equilibrium (DFE) is obtained by setting

$$E_h = I_h = E_v = I_v = E_w = I_w = 0.$$

**Proposition 1. Disease-Free Equilibrium**

Under Assumption 1, system (6) admits the unique disease-free equilibrium

$$\mathcal{E}_0 = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, \frac{\Lambda_v}{\mu_v}, 0, 0, \frac{u}{\kappa_w}, 0, 0\right). \qquad (14)$$

**Proof**

At the disease-free state, system (6) reduces to

$$0 = \Lambda_h - \mu_h S_h,\ 0 = -\mu_h R_h,$$
$$0 = \Lambda_v - \mu_v S_v,$$

and

$$0 = u - \kappa_w S_w.$$

Therefore,

$$S_h^0 = \frac{\Lambda_h}{\mu_h},\ R_h^0 = 0,$$
$$S_v^0 = \frac{\Lambda_v}{\mu_v},\ S_w^0 = \frac{u}{\kappa_w}, \qquad (14)$$

which gives □

**4 Basic Reproduction Number $\mathcal{R}_0$**

The basic reproduction number is derived using the next-generation matrix method (Diekmann et al., 1990; van den Driessche and Watmough, 2002).

Let the infected-state vector be

$$x = (E_h, I_h, E_v, I_v, E_w, I_w)^T. \qquad (15)$$

The infected subsystem is written as

$$\dot{x} = \mathcal{F}(x) - \mathcal{V}(x).$$

At $\mathcal{E}_0$, the new-infection matrix is

$$F = \begin{pmatrix} 0 & 0 & 0 & a\beta_{vh} & 0 & a\beta_{vh}(1-\varepsilon) \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a\beta_{hv}\frac{S_v^0}{N_h^0} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & a\beta_{hw}\frac{S_w^0}{N_h^0} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \qquad (16)$$

while the transition matrix is

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$$V = \begin{pmatrix} k_1 & 0 & 0 & 0 & 0 & 0 \\ -\sigma_h & k_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & k_3 & 0 & 0 & 0 \\ 0 & 0 & -\sigma_v & \mu_v & 0 & 0 \\ 0 & 0 & 0 & 0 & k_4 & 0 \\ 0 & 0 & 0 & 0 & -\sigma_w & \mu_w \end{pmatrix}. \qquad (17)$$

The basic reproduction number is

$$R_0 = \rho(FV^{-1}) \qquad (18)$$

where $\rho$denotes the spectral radius.
The next-generation matrix $FV^{-1}$is obtained from the new infection matrix $F$and the transition matrix $V$. The dominant eigenvalue of $FV^{-1}$is determined from the characteristic equation

$$\lambda^2 - (R_v^2 + R_w^2) = 0$$

which gives

$$\rho(FV^{-1}) = \sqrt{R_v^2 + R_w^2}.$$

Therefore, the basic reproduction number becomes

$$\mathcal{R}_0 = \sqrt{\mathcal{R}_v^2 + \mathcal{R}_w^2}, \qquad (19)$$

where

$$\mathcal{R}_v^2 = \frac{a^2\beta_{vh}\beta_{hv}\sigma_h\sigma_v}{k_1k_2k_3\mu_v}\frac{S_v^0}{N_h^0}, \qquad (20)$$

and

$$\mathcal{R}_w^2 = \frac{a^2\beta_{vh}\beta_{hw}(1-\varepsilon)\sigma_h\sigma_w}{k_1k_2k_4\mu_w}\frac{S_w^0}{N_h^0}. \qquad (21)$$

Using

$$N_h^0 = \frac{\Lambda_h}{\mu_h},\ S_v^0 = \frac{\Lambda_v}{\mu_v},\ S_w^0 = \frac{u}{\kappa_w},$$

equation (19) becomes

$$\mathcal{R}_0 = \left[\frac{a^2\beta_{vh}\beta_{hv}\sigma_h\sigma_v\Lambda_v\mu_h}{k_1k_2k_3\mu_v^2\Lambda_h} + \frac{a^2\beta_{vh}\beta_{hw}(1-\varepsilon)\sigma_h\sigma_w u\mu_h}{k_1k_2k_4\mu_w\kappa_w\Lambda_h}\right]^{1/2}. \qquad (22)$$

Using equation (19), the reproduction number is expressed as

$$R_0 = \sqrt{R_v^2 + R_w^2}.$$

The terms $R_v$and $R_w$represent the contributions of wild-type and *Wolbachia*-infected mosquitoes, respectively, to dengue transmission.

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The terms $\mathcal{R}_v$and $\mathcal{R}_w$represent the contributions of wild-type and *Wolbachia*-infected mosquitoes, respectively, to dengue transmission.

**5 Numerical Stability of the Endemic Equilibrium**

Because system (6) is ten-dimensional, direct expansion of the characteristic polynomial at $\mathcal{E}^*$produces a high-order expression with limited analytical usefulness. Local stability of the endemic equilibrium is therefore assessed numerically.

Let

$$J^* = J(\mathcal{E}^*) = \left.\frac{\partial F}{\partial X}\right|_{\mathcal{E}^*} \qquad (40)$$

denote the Jacobian matrix evaluated at the numerically determined endemic equilibrium.

**Proposition 2. Numerical Stability Criterion**

For a specified parameter set with $\mathcal{R}_0 > 1$, the endemic equilibrium $\mathcal{E}^*$is locally asymptotically stable if all eigenvalues of $J^*$satisfy

$$\mathrm{Re}\,(\lambda_i) < 0,\ i = 1, \dots, 10. \qquad (41)$$

Equivalently,

$$\max_{1 \le i \le 10} \mathrm{Re}\,(\lambda_i) < 0. \qquad (42)$$

The linearized perturbation equation about $\mathcal{E}^*$is

$$\dot{\xi} = J^* \xi. \qquad (43)$$

By the linearization theorem for autonomous systems, perturbations decay asymptotically whenever all eigenvalues of $J^*$lie strictly in the left half of the complex plane. Conversely, an eigenvalue with positive real part implies local instability.

For each numerical parameter set, the endemic equilibrium is obtained by solving

$$F(\mathcal{E}^*) = 0,$$

after which the eigenvalues of $J^*$are computed numerically. The resulting maximum real eigenvalue should be reported alongside the corresponding value of $\mathcal{R}_0$.

**4. Sensitivity Analysis**

Sensitivity analysis is used to determine the relative influence of model parameters on the dengue invasion threshold.

For a parameter $p$, the normalized forward sensitivity index of $\mathcal{R}_0$is

$$\Upsilon_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p}\frac{p}{\mathcal{R}_0}. \qquad (44)$$

A positive index indicates that increasing $p$increases $\mathcal{R}_0$, whereas a negative index indicates an inverse relationship.

Define

$$\theta_v = \frac{\mathcal{R}_v^2}{\mathcal{R}_0^2},\ \theta_w = \frac{\mathcal{R}_w^2}{\mathcal{R}_0^2},\ \theta_v + \theta_w = 1. \qquad (45)$$

The principal analytical sensitivity indices are

$$\Upsilon_a^{\mathcal{R}_0} = 1, \qquad (46)$$

$$\Upsilon_{\beta_{vh}}^{\mathcal{R}_0} = \frac{1}{2}, \qquad (47)$$

$$\Upsilon_{\beta_{hv}}^{\mathcal{R}_0} = \frac{\theta_v}{2}, \Upsilon_{\beta_{hw}}^{\mathcal{R}_0} = \frac{\theta_w}{2}, \quad (48)$$

$$\Upsilon_{\varepsilon}^{\mathcal{R}_0} = -\frac{\varepsilon\theta_w}{2(1-\varepsilon)}, \quad (49)$$

$$\Upsilon_{u}^{\mathcal{R}_0} = \frac{\theta_w}{2}, \quad (50)$$

$$\Upsilon_{\gamma_h}^{\mathcal{R}_0} = -\frac{\gamma_h}{2k_2}, \quad (51)$$

$$\Upsilon_{\sigma_h}^{\mathcal{R}_0} = \frac{\mu_h}{2k_1}, \quad (52)$$

$$\Upsilon_{\sigma_v}^{\mathcal{R}_0} = \frac{\theta_v\mu_v}{2k_3}, \quad (53)$$

and

$$\Upsilon_{\sigma_w}^{\mathcal{R}_0} = \frac{\theta_w\mu_w}{2k_4}. \quad (54)$$

For parameters governing *Wolbachia* persistence,

$$\Upsilon_{\eta}^{\mathcal{R}_0} = \frac{\theta_w}{2}\frac{r_w\eta(1-c)}{\kappa_w}, \quad (55)$$

$$\Upsilon_{r_w}^{\mathcal{R}_0} = \frac{\theta_w}{2}\frac{r_w\eta(1-c)}{\kappa_w}, \quad (56)$$

and

$$\Upsilon_{c}^{\mathcal{R}_0} = -\frac{\theta_w}{2}\frac{cr_w\eta}{\kappa_w}. \quad (57)$$

Parameters are ranked according to

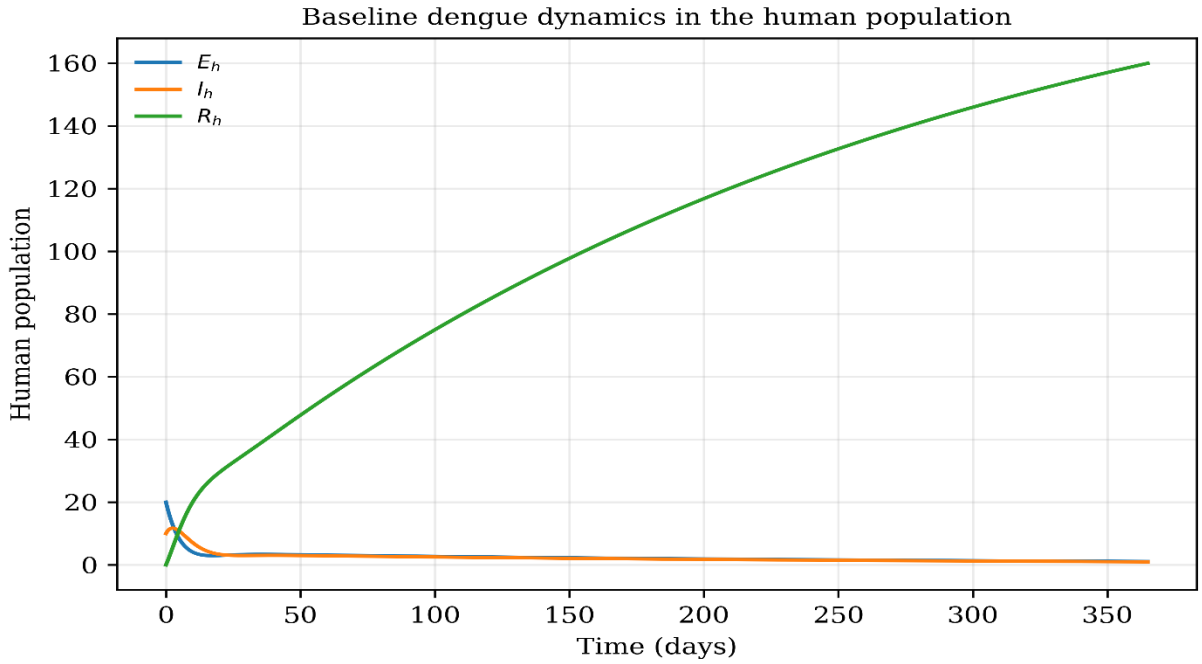
$$\left|\Upsilon_{p}^{\mathcal{R}_0}\right|.$$

Large absolute sensitivity indices identify parameters to which the dengue transmission threshold is most responsive.

## 5. Numerical Simulations

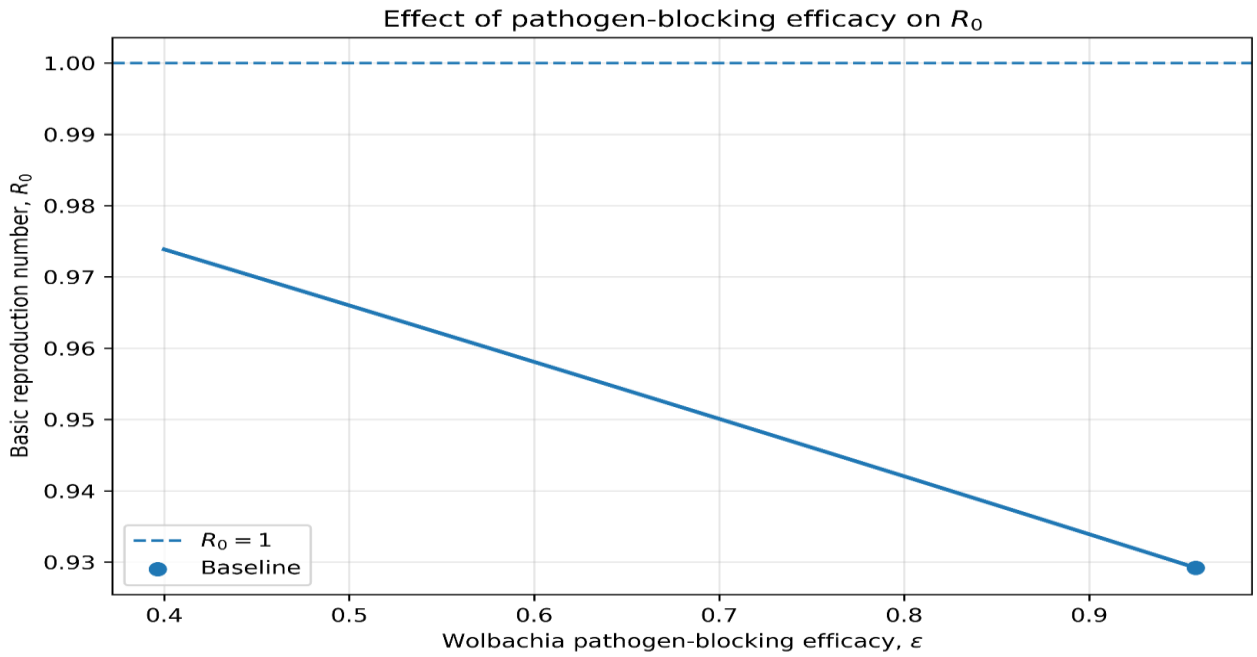
Numerical simulations are performed using the baseline parameter values and initial conditions reported in Tables 2 and 3. System (6) is integrated using an adaptive Runge Kutta method implemented in MATLAB. The simulations are used to illustrate the theoretical results and examine how key *Wolbachia*-related parameters affect dengue dynamics.

**Figure 1. Baseline temporal dynamics of human dengue compartments.**



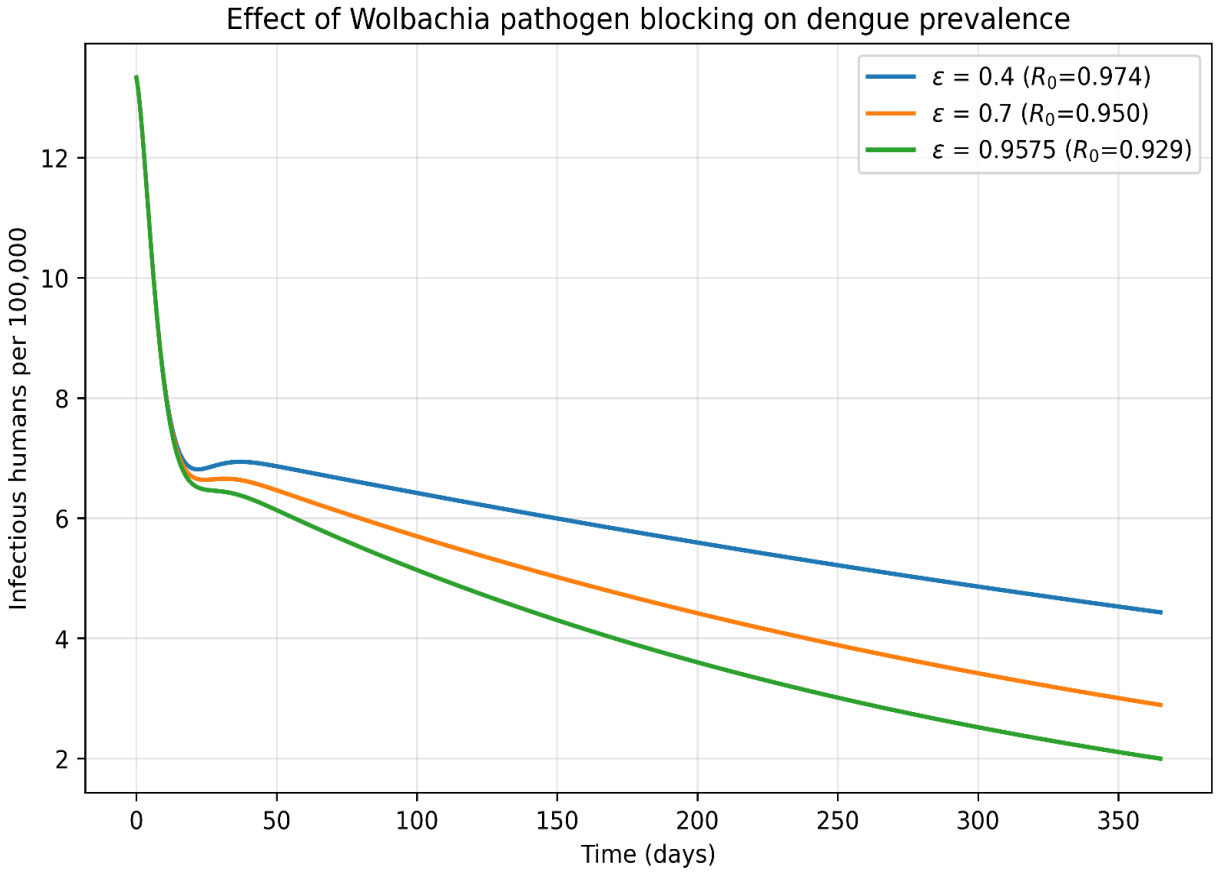

The baseline simulation depicts a decreasing trend in dengue infection. The infectious prevalence dropped from about 13.33 individuals per 100,000 down to 2.00 over 365 days, which is a drop of about 85%. There was a similar pattern of decline for the exposed population and the recovered population continued to rise. This behaviour is consistent with the theoretical condition R_0<1 (convergence towards the disease-free state)

**Figure 2. Effect of Wolbachia pathogen-blocking efficacy on infectious human prevalence.**



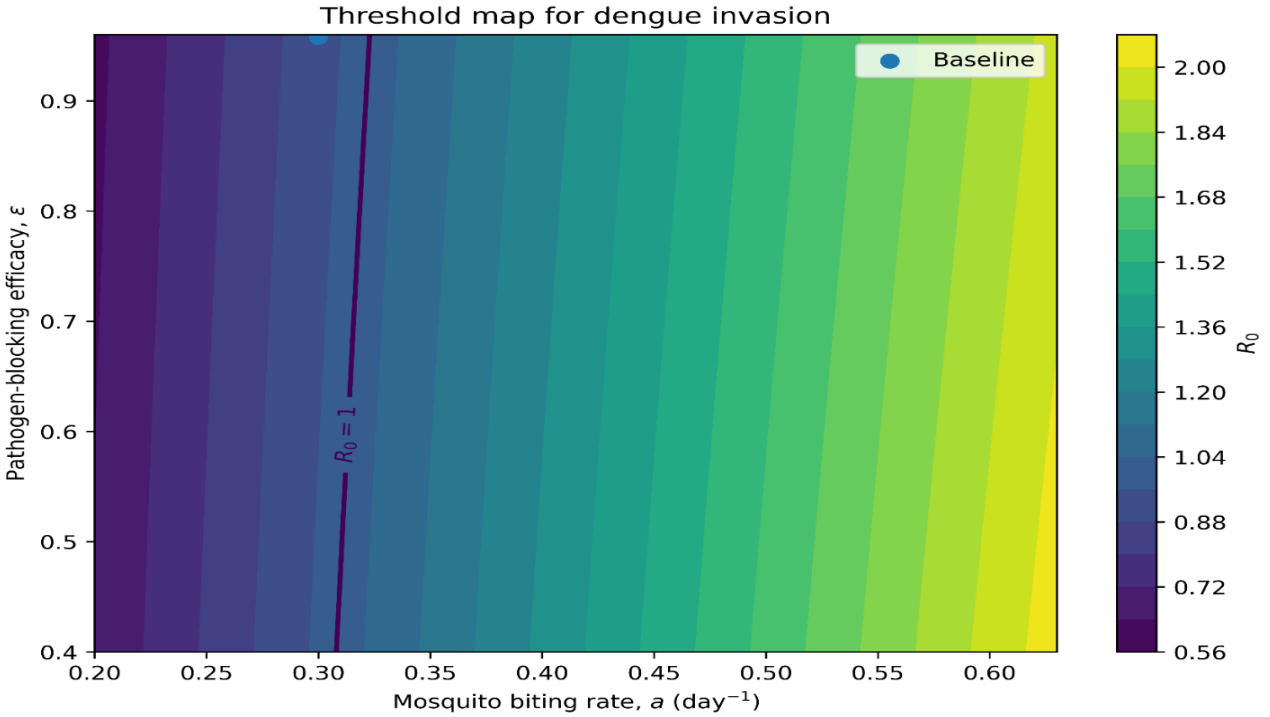

The more effective pathogen blocking, the less reproduction number and infectious prevalence. For ϵ=0.40, 0.70, and 0.9575, the corresponding reproduction numbers were 0.9739, 0.9501, and 0.9292, respectively. In the scenarios, the infectious prevalence reduced to around 4.43, 2.89 and 2.00 in days 365, 510 and 650 respectively. Therefore, there was an effect of approximately 55% reduction in final infectious prevalence with an increase in ϵ from 0.40 to 0.9575.

**Figure 3. Variation of the basic reproduction number with Wolbachia pathogen-blocking efficacy.**



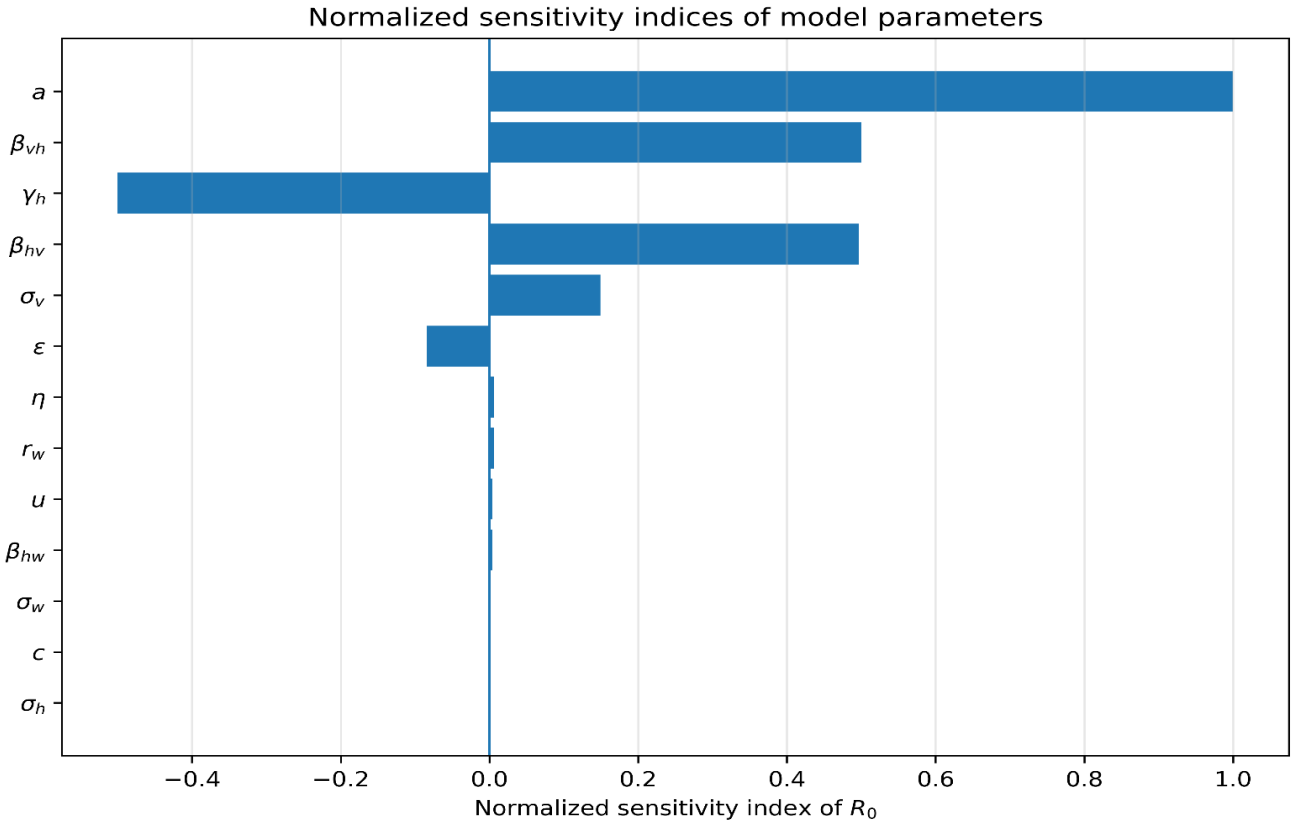

The reproduction number was monotonically decreasing with increasing pathogen blocking efficacy from about 0.974 for ϵ=0.40 to 0.929 for ϵ=0.9575. However, as the pathogen blocking increased the system approached the epidemic threshold more and R_0retained its value below unity, which created better conditions for dengue control.

**Figure 4; shows the combined effects of mosquito biting rate and *Wolbachia*-induced pathogen-blocking efficacy on the basic reproduction number, $R_0$.**




The contour $R_0 = 1$represents the epidemic threshold, separating parameter combinations that lead to disease elimination from those that permit dengue invasion. At the baseline pathogen-blocking efficacy of $\epsilon = 0.9575$, the critical biting rate was approximately $a_c = 0.3229$ day$^{-1}$, compared with the baseline value of $a = 0.30$ day$^{-1}$. Therefore, an increase of about 7.6% in the mosquito biting rate would move the system towards the epidemic threshold. This indicates that reducing mosquito–human contact can enhance the effectiveness of *Wolbachia*-mediated pathogen blocking in controlling dengue transmission.

**Figure 5. Normalized sensitivity indices of the model parameters to $\mathcal{R}_0$**




The parameter that most strongly influenced the mosquito biting rate had a normalized sensitivity index of +1.000, which was the mosquito biting rate itself. Similarly, the mosquito to human and human to wild mosquito transmission probabilities had strong positive impacts, with indices of +0.500and +0.496, respectively. In contrast, the human recovery rate comes in at around -0.500, and the Wolbachia pathogen-blocking efficacy is negative, around -0.085. The results indicate that mosquito biting, the probability of mosquito transmission, human recovery, and pathogen blocking are the most important factors in the model when determining the spread of dengue.

**Discussion**

The present study developed a deterministic dengue–*Wolbachia* transmission model incorporating wild-type mosquitoes, *Wolbachia*-infected mosquitoes, imperfect maternal transmission, pathogen blocking, and continuous augmentation. The analytical results demonstrate that dengue transmission dynamics are influenced by interactions between mosquito transmission processes and *Wolbachia*-associated biological mechanisms. The findings indicate that reducing mosquito competence through pathogen blocking can lower transmission potential, although the overall impact remains dependent on the underlying transmission intensity of the vector–host system (Dennison et al., 2014; Okomo et al., 2026). The sensitivity analysis identified mosquito biting rate and mosquito–human transmission probability as dominant drivers of dengue transmission. This result is biologically consistent because these parameters directly determine the frequency and efficiency of infectious contacts between mosquitoes and humans. Similar dengue modelling studies have shown that transmission-related parameters, including biting rate and mosquito–human transmission probability, strongly influence dengue invasion potential (Luz et

al., 2011; Olmo et al., 2023). Therefore, reducing mosquito–human contact remains an important complementary strategy for enhancing the effectiveness of *Wolbachia*-based interventions.

The findings suggest that *Wolbachia* deployment should be integrated with other dengue control measures because intervention success depends not only on pathogen blocking but also on maintaining effective interruption of mosquito-mediated transmission (Barlow & Adams, 2025; Utarini et al., 2021).The model does not incorporate spatial heterogeneity, seasonal environmental variation, or human mobility patterns and assumes homogeneous mixing within the population. These limitations may influence dengue transmission dynamics because mosquito populations and human exposure patterns vary across ecological and demographic settings. Future studies incorporating location-specific entomological data, seasonal forcing, and spatial structure could provide more realistic predictions of *Wolbachia*-based dengue control outcomes.

**Conclusion**

This study developed and analyzed a deterministic dengue–*Wolbachia* transmission model incorporating wild-type mosquitoes, *Wolbachia*-infected mosquitoes, imperfect maternal transmission, pathogen blocking, and continuous augmentation. The analytical results demonstrate that *Wolbachia*-based interventions can reduce dengue transmission potential by decreasing mosquito vector competence and disrupting transmission pathways. The sensitivity analysis further highlights the importance of mosquito biting rate and mosquito–human transmission probability in determining dengue transmission dynamics, emphasizing the role of vector-related processes in shaping intervention outcomes. The model provides insights into the influence of biological and intervention-related parameters on dengue control; however, it assumes homogeneous population mixing and does not explicitly consider spatial variation, seasonal environmental changes, or human mobility patterns. These limitations may influence the representation of dengue transmission dynamics because ecological and demographic factors affect mosquito populations and disease spread. Therefore, while the present framework provides a theoretical basis for understanding *Wolbachia*-mediated dengue control, further model developments incorporating these factors are required to improve its applicability under realistic transmission settings.

**Recommendations**

Based on the findings and limitations of this study, the following recommendations are proposed:

I. Future extensions of the model should incorporate spatial heterogeneity, seasonal forcing, and human mobility patterns to provide a more realistic representation of dengue transmission dynamics and *Wolbachia*-based intervention outcomes.

II. The deterministic framework may be extended using fractional-order formulations, particularly incorporating the Caputo memory effect, to account for historical dependence in transmission processes and better represent complex biological interactions in dengue–*Wolbachia* dynamics.

III. Future studies should integrate location-specific epidemiological and entomological data with uncertainty and sensitivity analyses to improve parameter estimation and enhance the applicability of the model for dengue control planning.

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