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Analyzing Dengue Transmission Through a Two-Age-Class Host Population Model

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Abstract. *Age is an important risk factor for vector-borne diseases such as dengue. Children are more exposed to mosquito bites than adults due to behavioral and environmental factors. This study extends the classical host-vector modeling framework by incorporating the human population divided into two age classes, i.e, children and adults. The model also introduces additional key biological parameters, such as b, the number of mosquito bites per day; σ, the intrinsic growth rate of the mosquito population; and η, the relative probability that a mosquito bites an adult rather than a child. We derive the basic reproduction number using the next-generation matrix method and analyze the local stability of the disease-free equilibrium. Furthermore, we obtain sufficient conditions for the local asymptotic stability of the endemic equilibrium in a specific case. Sensitivity analysis is discussed to identify parameters that have the most influence. The numerical simulations are provided to support the theoretical results.*

1. Introduction

Dengue, a viral disease, is transmitted through the bites of female *Aedes aegypti* mosquitoes, which are widely distributed in tropical and subtropical regions [1]. To address this threat, mathematics serves as a critical tool for analyzing its transmission dynamics. Several basic models, such as the SIR-SI and SEIR-SEI models, have been employed to analyze the spread of dengue, focusing on the human population as the host and mosquitoes as the vector. The authors in [2–5] showed that the variables related to the host-vector, such as population density and infection level, play an important role in dengue transmission. Dengue fever models have been extended in various ways, including the introduction of multiple infectious classes to represent severe and mild symptoms, for example, the SI_1I_2RS model [6]. Additionally, the incorporation of the aquatic phase in vector dynamics has led to the SEIR–ASEI model [7–9].

However, these models use simplistic assumptions where the human population is assumed to be homogeneous, without considering the age group, which can significantly impact the dynamics of dengue transmission. On the other hand, various research studies have examined the prevalence of dengue in both children and adults, see [10–14]. In the context of dengue, children and adults exhibit varying levels of susceptibility and severity. Younger individuals face a heightened risk due to their

immature immune systems. The authors in [11] divide the population into cohorts and construct a separate SIR for each group.

Some studies have investigated the incidence of dengue among both children and adults, revealing that the most affected age groups vary across regions. For example, in Thailand, dengue cases are more frequently reported in children aged 5-9 [15], while in Vietnam, the highest rates occur in children aged between 1 and 15 years [16]. In contrast, dengue is more prevalent among the working and school-going age groups in Malaysia and Singapore [17, 18]. In Indonesia and other dengue-endemic regions, children are consistently found to be the most affected group. A national seroprevalence study showed that over 80% of children aged 10 and older had been exposed to dengue [19], with similar findings in Yogyakarta, where 88% of 10-year-olds had dengue antibodies [20]. This high childhood exposure suggests that most adults have developed immunity, making new adult infections rare, typically mild or asymptomatic, and less influential in transmission dynamics.

Therefore, in this study, we outlined four main findings as follows. The first is a new mathematical model that extends the classical host-vector modeling in [5]. The new model considers that the human population is divided into two age classes, i.e, children and adults. Second, the proposed model introduces additional key biological parameters that differ from those in [10], such as b as the average mosquito biting rate per day and σ as the intrinsic growth rate of the mosquito population. Third, sufficient conditions are obtained for the local asymptotic stability of the endemic equilibrium in a specific case. Fourth, a key feature of the model is the appearance of the mosquito biting rate b in quadratic form in the expression of the basic reproductive number. This indicates that even small changes in host-vector transmission can significantly influence the spread of infection, emphasizing the importance of vector control strategies. Additionally, the parameter η, which denotes the relative rate at which adults are bitten compared to children, is shown to contribute quadratically to the basic reproductive number.

2. Model Formulation

This study uses a host-vector model to analyze the interaction between humans and *Aedes aegypti* mosquitoes in the context of dengue fever transmission. The human population is categorized into two age groups: children and adults. Mathematically, the model consists of a system of ordinary differential equations (ODEs) that describe the transmission dynamics of dengue fever, assuming a constant population size.

The model includes six compartments for the human (host) population: susceptible children (S_c), infected children with the dengue virus (I_c), children who have recovered from dengue (R_c), adults susceptible to dengue (S_A), adults infected with the dengue virus (I_A), adults who have recovered (R_A). On the other hand, the mosquito (vector) population is divided into two compartments: susceptible mosquitoes (S_v) and mosquitoes infected with the dengue virus (I_v).

The total human population is represented by N_h and the natural mortality rate by μ_h. Under the assumption that births and natural deaths occur at the same rate, the resulting population growth rate is given by $\mu_h N_h$. We denote α to describe the rate of maturation, that is, when children become adults. Furthermore, because the duration of dengue infection is relatively short compared to the time it takes for a child to reach adulthood, we do not account for α in the maturation of infected children into infected adults.

Let b represents the average mosquito biting rate per day. Then, a total of bN_v mosquito bites occur daily. Out of these, the total number of mosquito bites per day that cause infection is given by $\frac{bN_v}{N_h}\frac{I_v}{N_v}$. If β_h is the transmission rate from mosquitoes to humans, then the infection rates for susceptible children and adults are $\beta_h\frac{bN_v}{N_h}\frac{I_v}{N_v}S_c$ and $\beta_h\frac{bN_v}{N_h}\frac{I_v}{N_v}S_A$, respectively. The parameter η represents the relative likelihood that an adult is bitten by a mosquito compared to a child. This value, known as the relative risk rate, is assumed to lie between zero and one. Lastly, we denote γ_c and γ_A as the recovery rates of children and adults, respectively.

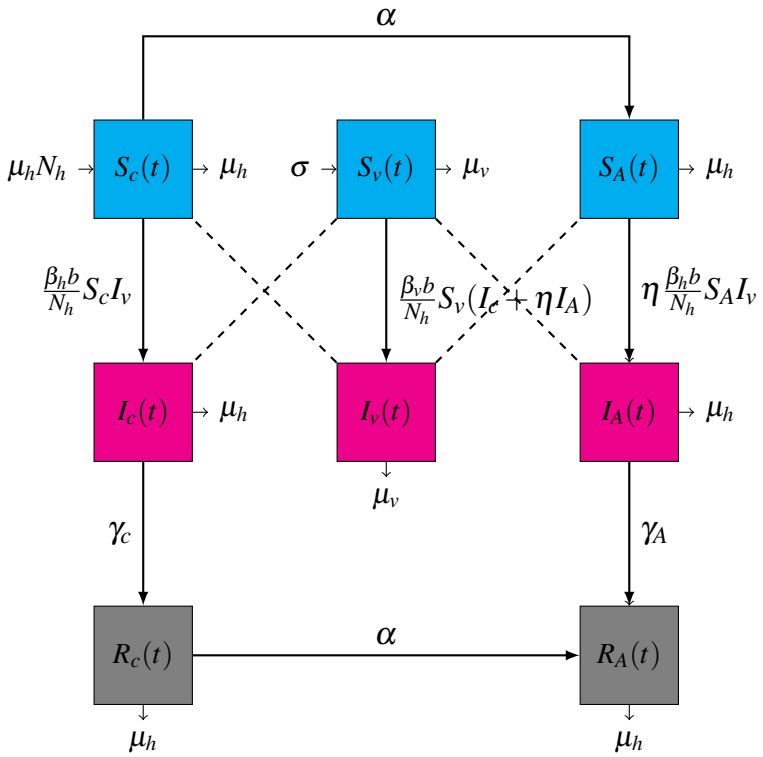

Figure 1. Compartmental diagram of the two-age-class host model

The vector population grows at a rate denoted by σ, while μ_v represents the mortality rate of the vector. Given that each mosquito bites at a rate of $\frac{b}{N_h}$ times per human per day, a mosquito encounters infected hosts at a rate of $\frac{b}{N_h} I_h$ per day. If β_v denotes the transmission rate from the infected hosts—including both children and adults—to the mosquitoes, then the rate at which susceptible vectors become infected is given by $\beta_v \frac{b}{N_h}(I_c + \eta I_A)$.

Figure 1 is provided to illustrate the structure of the mathematical model. The arrows indicate the transitions between compartments governed by infection, recovery, growth, and natural death. This model describes the transmission of the dengue virus by considering several key factors that affect how the virus spreads, as explained below:

$$
\begin{aligned}
\frac{dS_c}{dt} &= \mu_h N_h - \frac{\beta_h b}{N_h} S_c I_v - \mu_h S_c - \alpha S_c, \\
\frac{dI_c}{dt} &= \frac{\beta_h b}{N_h} S_c I_v - \mu_h I_c - \gamma_c I_c, \\
\frac{dR_c}{dt} &= \gamma_c I_c - \mu_h R_c - \alpha R_c, \\
\frac{dS_A}{dt} &= \alpha S_c - \eta \frac{\beta_h b}{N_h} S_A I_v - \mu_h S_A, \\
\frac{dI_A}{dt} &= \eta \frac{\beta_h b}{N_h} S_A I_v - \gamma_A I_A - \mu_h I_A, \\
\frac{dR_A}{dt} &= \alpha R_c + \gamma_A I_A - \mu_h R_A, \\
\frac{dS_v}{dt} &= \sigma - \frac{\beta_v b}{N_h} S_v (I_c + \eta I_A) - \mu_v S_v,
\end{aligned}
\tag{1}
$$

$$\frac{dI_v}{dt} = \frac{\beta_v b}{N_h} S_v (I_c + \eta I_A) - \mu_v I_v.$$

In this research, we define $N_h = S_c + I_c + R_c + S_A + I_A + R_A$ and $N_v = S_v + I_v$ as the host and vector population, respectively. In this case, we find that

$$\frac{dN_v}{dt} = \sigma - \mu_v N_v.$$

Therefore for time $t \to \infty$, we have N_v tends to $\frac{\sigma}{\mu_v}$. Since $S_v = N_v - I_v = \frac{\sigma}{\mu_v} - I_v$, then substituted it into the last equation of system (1). Furthermore, due to the non-appearance of R_c and R_A in the equation of S_c, I_c, S_A, I_A, then we simplify the system (1) using the following equations:

$$\begin{aligned}
\frac{dS_c}{dt} &= \mu_h N_h - \frac{\beta_h b}{N_h} S_c I_v - \mu_h S_c - \alpha S_c, \\
\frac{dI_c}{dt} &= \frac{\beta_h b}{N_h} S_c I_v - \mu_h I_c - \gamma_c I_c, \\
\frac{dS_A}{dt} &= \alpha S_c - \eta \frac{\beta_h b}{N_h} S_A I_v - \mu_h S_A, \\
\frac{dI_A}{dt} &= \eta \frac{\beta_h b}{N_h} S_A I_v - \gamma_A I_A - \mu_h I_A, \\
\frac{dI_v}{dt} &= \frac{\beta_v b}{N_h} (\frac{\sigma}{\mu_v} - I_v)(I_c + \eta I_A) - \mu_v I_v.
\end{aligned} \tag{2}$$

The initial conditions of system (2) are $S_c(0) > 0, I_c(0) \geq 0, S_A(0) > 0, I_A(0) \geq 0, I_v(0) \geq 0$. The feasible region of system (1) is given by

$$\Omega = \left\{ (S_c, I_c, S_A, I_A, I_v) \in \mathbb{R}^5_+ : S_c + I_c + S_A + I_A \leq N_h, I_v \leq N_v, S_c \leq \frac{\mu_h N_h}{\mu_h + \alpha}, S_A \leq \frac{\alpha N_h}{\mu_h + \alpha} \right\}.$$

3. Analytical Results

3.1. Positivity of the solutions

The population discussed in the system (2) is human and mosquito; therefore, it is necessary to prove that all variables are nonnegative $\forall t > 0$.

Theorem 1. *If the initial conditions $S_c(0) > 0, I_c(0) \geq 0, S_A(0) > 0, I_A(0) \geq 0, I_v(0) \geq 0$ then the solutions $S_c(t), I_c(t), S_A(t), I_A(t), I_v(t)$ of the system (2) are nonnegative $\forall t > 0$.*

Proof. Based on the first equation of system (2), we have

$$\frac{dS_c}{dt} = \mu_h N_h - \frac{\beta_h b}{N_h} S_c I_v - \mu_h S_c - \alpha S_c \geq -\left(\frac{\beta_h b}{N_h} I_v + \mu_h + \alpha \right) S_c.$$

It leads to

$$\begin{aligned}
\frac{dS_c(t)}{S_c(t)} &\geq -\left(\frac{\beta_h b}{N_h} I_v(t) + \mu_h + \alpha \right) dt, \\
\int_0^t \frac{dS_c(w)}{S_c(w)} dw &\geq \int_0^t -\left(\frac{\beta_h b}{N_h} I_v(w) + \mu_h + \alpha \right) dw, \\
\ln\left(\frac{S_c(t)}{S_c(0)} \right) &\geq \int_0^t -\left(\frac{\beta_h b}{N_h} I_v(w) + \mu_h + \alpha \right) dw, \\
\frac{S_c(t)}{S_c(0)} &\geq \exp\left(\int_0^t -\left(\frac{\beta_h b}{N_h} I_v(w) + \mu_h + \alpha \right) dw \right),
\end{aligned}$$

$$S_c(t) \geq S_c(0)\exp\left(-\int_0^t \left(\frac{\beta_h b}{N_h} I_v(w) + \mu_h + \alpha\right) dw\right) > 0.$$

Using the same method, we obtain the following.

$$\begin{aligned}
I_c(t) &\geq I_c(0)\exp\left(-(\mu_h+\gamma_c)t\right) \geq 0\\
S_A(t) &\geq S_A(0)\exp\left(-\int_0^t \left(\eta\frac{\beta_h b}{N_h} I_v(w) + \mu_h\right) dw\right) > 0,\\
I_A(t) &\geq I_A(0)\exp\left(-(\mu_h+\gamma_A)t\right) \geq 0,\\
I_v(t) &\geq I_v(0)\exp\left(-\int_0^t \frac{\sigma}{\mu_v}\left(\frac{\beta_v b}{N_h} I_c(w) + \eta\frac{\beta_v b}{N_h} I_A(w)\right) dw\right) \geq 0.
\end{aligned}$$

Therefore, all solutions of the system (2) are nonnegative for all time $t > 0$. ■

3.2. Disease-free equilibrium point

The equilibrium condition indicates that the system maintains a constant state over time. By setting each equation in system (2) equal to zero, then we have

$$S_c = \frac{\mu_h N_h}{\frac{\beta_h b}{N_h} I_v + \mu_h + \alpha}, \tag{3}$$

$$S_A = \frac{\alpha\mu_h N_h}{\left(\frac{\beta_h b}{N_h} I_v + \mu_h + \alpha\right)\left(\eta\frac{\beta_h b}{N_h} I_v + \mu_h\right)}, \tag{4}$$

$$I_c = \frac{\beta_h b \mu_h I_v}{(\mu_h+\gamma_c)\left(\frac{\beta_h b}{N_h} I_v + \mu_h + \alpha\right)}, \tag{5}$$

$$I_A = \frac{\alpha\mu_h\eta\beta_h b I_v}{(\gamma_A+\mu_h)\left(\frac{\beta_h b}{N_h} I_v + \mu_h + \alpha\right)\left(\eta\frac{\beta_h b}{N_h} I_v + \mu_h\right)}. \tag{6}$$

The disease-free equilibrium corresponds to the situation where the population is free from infection. In this research, we denote it as E_0.

Lemma 2. *If $I_v = 0$, then system (2) has a disease-free equilibrium point $E_0 = \left(\frac{\mu_h N_h}{\mu_h+\alpha}, 0, \frac{\alpha N_h}{\alpha+\mu_h}, 0, 0\right)$.*

Proof. Assuming $I_v = 0$, then we substitute into eqs. (3) to (6), we obtain $\left(\frac{\mu_h N_h}{\mu_h+\alpha}, 0, \frac{\alpha N_h}{\alpha+\mu_h}, 0, 0\right)$ as the disease-free equilibrium. ■

3.3. Basic reproduction number

We will explore the basic reproduction number, R_0, which measures the presence of secondary infections deriving from an initial infection. This approach enables a systematic analysis of how disease dynamics are expected to evolve under specific conditions. This value is calculated using the next-generation matrix [21]. In this study, we define the F matrix as a matrix containing entries representing the first derivatives of all terms related to the rates of change of factors affecting the infectious population at the disease-free equilibrium E_0, i.e.,

$$F = \begin{bmatrix} 0 & 0 & \frac{\beta_h b\mu_h}{\mu_h+\alpha} \\ 0 & 0 & \eta\frac{\beta_h b\alpha}{\mu_h+\alpha} \\ \frac{\beta_v b\sigma}{N_h\mu_v} & \eta\frac{\beta_v b\sigma}{N_h\mu_v} & 0 \end{bmatrix}. \tag{7}$$

Meanwhile, matrix K explains the transition terms within the system (2) at the disease-free equilibrium E_0, therefore we obtain

$$K = \begin{bmatrix} \mu_h + \gamma_c & 0 & 0 \\ 0 & \mu_h + \gamma_A & 0 \\ 0 & 0 & \mu_v \end{bmatrix}. \tag{8}$$

The inverse of matrix K in eq. (8) is

$$K^{-1} = \begin{bmatrix} \frac{1}{\mu_h+\gamma_c} & 0 & 0 \\ 0 & \frac{1}{\mu_h+\gamma_A} & 0 \\ 0 & 0 & \frac{1}{\mu_v} \end{bmatrix}. \tag{9}$$

Based on eqs. (7) and (9), we have

$$FK^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_h b \mu_h}{\mu_v(\mu_h+\alpha)} \\ 0 & 0 & \eta \frac{\beta_h b \alpha}{\mu_v(\mu_h+\alpha)} \\ \frac{\beta_v b \sigma}{N_h \mu_v(\mu_h+\gamma_c)} & \eta \frac{\beta_v b \sigma}{N_h \mu_v(\mu_h+\gamma_A)} & 0 \end{bmatrix}. \tag{10}$$

The dominant eigenvalue of FK^{-1} represents the basic reproduction number, and we get

$$\bar{R}_0 = \sqrt{\frac{\beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)(\mu_h+\gamma_c)} \frac{\beta_h b \mu_h}{\mu_v} + \frac{\eta \beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)(\mu_h+\gamma_A)} \frac{\eta \beta_h b \alpha}{\mu_v}}. \tag{11}$$

If

$$R_0 = \frac{\beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)(\mu_h+\gamma_c)} \frac{\beta_h b \mu_h}{\mu_v} + \frac{\eta \beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)(\mu_h+\gamma_A)} \frac{\eta \beta_h b \alpha}{\mu_v} \tag{12}$$

then $\bar{R}_0 = \sqrt{R_0}$.

The interpretation of this value can be explained as follows: while an infected child is in the infectious period, there is a risk of transmission to susceptible children when $\frac{b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)(\mu_h+\gamma_c)}$ susceptible vectors bite the infected child. Therefore, a proportion $\frac{\beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)(\mu_h+\gamma_c)}$ of these vectors become infected. Furthermore, an infected vector transmits $\frac{b}{\mu_v}$ bites to susceptible children. These bites result in a fraction $\frac{\beta_h b \mu_h}{\mu_v}$ of new infections in the child population. The interpretation of the remaining part of the basic reproduction number is related to the adult population, which is closely aligned with the child population.

3.4. Stability analysis of disease-free equilibrium point

The stability properties will be discussed in this section. The Jacobian matrix of system (2) is as follows.

$$J = \begin{bmatrix} -\frac{\beta_h b}{N_h} I_v - \mu_h - \alpha & 0 & 0 & 0 & -\frac{\beta_h b}{N_h} S_c \\ \frac{\beta_h b}{N_h} I_v & -\mu_h - \gamma_c & 0 & 0 & \frac{\beta_h b}{N_h} S_c \\ \alpha & 0 & -\eta \frac{\beta_h b}{N_h} I_v - \mu_h & 0 & -\eta \frac{\beta_h b}{N_h} S_A \\ 0 & 0 & \eta \frac{\beta_h b}{N_h} I_v & -\gamma_A - \mu_h & \eta \frac{\beta_h b}{N_h} S_A \\ 0 & \frac{\beta_v b}{N_h} \frac{\sigma}{\mu_v} - \frac{\beta_v b}{N_h} I_v & 0 & \frac{\beta_v b}{N_h} \frac{\sigma}{\mu_v} \eta - \eta \frac{\beta_v b}{N_h} I_v & -\frac{\beta_v b}{N_h} I_c - \eta \frac{\beta_v b}{N_h} I_A - \mu_v \end{bmatrix}. \tag{13}$$

Theorem 3. *If $R_0 < 1$, then the disease-free equilibrium point E_0 is locally asymptotically stable.*

Proof. The Jacobian J at E_0 is as follows,

$$J(E_0) = \begin{bmatrix} -\mu_h - \alpha & 0 & 0 & 0 & -\frac{\beta_h b}{N_h}\frac{\mu_h N_h}{\mu_h+\alpha} \\ 0 & -\mu_h - \gamma_c & 0 & 0 & \frac{\beta_h b}{N_h}\frac{\mu_h N_h}{\mu_h+\alpha} \\ \alpha & 0 & -\mu_h & 0 & -\eta\frac{\beta_h b}{N_h}\frac{\alpha N_h}{\alpha+\mu_h} \\ 0 & 0 & 0 & -\gamma_A - \mu_h & \eta\frac{\beta_h b}{N_h}\frac{\alpha N_h}{\alpha+\mu_h} \\ 0 & \frac{\beta_v b}{N_h}\frac{\sigma}{\mu_v} & 0 & \frac{\beta_v b}{N_h}\frac{\sigma}{\mu_v}\eta & -\mu_v \end{bmatrix}. \tag{14}$$

Here, λ denotes the eigenvalue of $J(E_0)$. Therefore, the polynomial characteristic of eq. (14) is

$$(\lambda+\mu_h+\alpha)(\lambda+\mu_h)\Big[\lambda^3 + (\mu_h+\gamma_c+\gamma_A+\mu_h+\mu_v)\lambda^2 + ((\mu_h+\gamma_c)(\mu_h+\gamma_A)+\mu_v(\mu_h+\gamma_c+\gamma_A+\mu_h))\lambda$$
$$+\mu_v(\mu_h+\gamma_c)(\mu_h+\gamma_A) - \eta\frac{\beta_v b}{N_h}\frac{\sigma}{\mu_v}\eta\frac{\beta_h b}{N_h}\frac{\alpha N_h}{\mu_h+\alpha}\Big] = 0.$$

It is clear that $-\mu_h-\alpha$ and $-\mu_h$ are the first two eigenvalues. The remaining eigenvalues are solutions to the following cubic equation

$$\lambda^3 + A_1\lambda^2 + A_2\lambda + A_3 = 0,$$

where

$$\begin{aligned} A_1 &= \mu_h+\gamma_c+\gamma_A+\mu_h+\mu_v, \\ A_2 &= (\mu_h+\gamma_c)(\mu_h+\gamma_A)(1-R_0) + \frac{\beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)}\frac{\beta_h b\mu_h}{\mu_v}(\mu_h+\gamma_A) + \frac{\eta\beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)}\frac{\eta\beta_h b\alpha}{\mu_v}(\mu_h+\gamma_c) \\ &\quad + \mu_v(\mu_h+\gamma_c+\gamma_A+\mu_h), \\ A_3 &= \mu_v(\mu_h+\gamma_c)(\mu_h+\gamma_A) - \eta\frac{\beta_v b}{N_h}\frac{\sigma}{\mu_v}\eta\frac{\beta_h b}{N_h}\frac{\alpha N_h}{\mu_h+\alpha}. \end{aligned}$$

Furthermore, if

$$\begin{aligned} A_4 &= \frac{\beta_h b\mu_h}{\mu_v(\mu_h+\alpha)(\mu_h+\gamma_c)}, \\ A_5 &= \frac{\eta\beta_h b\alpha\eta}{\mu_v(\mu_h+\alpha)(\mu_h+\gamma_A)}, \\ A_6 &= \frac{1}{1+\frac{A_4}{A_5}}, \end{aligned}$$

then, the expression of A_3 can be rewritten as follows.

$$\begin{aligned} A_3 &= \mu_v(\mu_h+\gamma_A)\left[(\mu_h+\gamma_c) - \frac{A_5}{A_4+A_5}R_0\right] \\ &= \mu_v(\mu_h+\gamma_A)(\mu_h+\gamma_c)\left[1-\frac{A_6}{\mu_h+\gamma_c}R_0\right]. \end{aligned}$$

Since all the parameters are positive, we have $A_1 > 0$. If $R_0 < 1$, it implies $A_2 > 0$. Since $0 < A_6 < 1$, $R_0 < 1$ and $\mu_h+\gamma_c > A_6$, then $A_3 > 0$. Note that $A_1A_2 > \mu_v(\mu_h+\gamma_c)(\mu_h+\gamma_A) + \frac{\beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)}\beta_h b\mu_h(\mu_h+\gamma_A) + \frac{\eta\beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)}\eta\beta_h b\alpha > A_3$. By the Routh-Hurwitz criteria, then the eigenvalues have negative real parts, and consequently the E_0 will be asymptotically stable. ■

3.5. Existence of the endemic equilibrium point

Besides the dengue-free E_0, system (2) also has a nontrivial equilibrium, namely a dengue-endemic equilibrium point denoted by E_1. We discuss this in Theorem 4.

Theorem 4. *If $R_0 > 1$, then there exists an endemic equilibrium point E_1 of the system (2).*

Proof. We note that based on eqs. (5) and (6) , I_c and I_A are substituted in the last equation of the system (2), which is equal to zero; then we have the following quadratic equation

$$p(I_v) = B_1 I_v^2 + B_2 I_v + B_3, \tag{15}$$

where

$$\begin{aligned} B_1 =& -\eta \left(\frac{\beta_h b}{N_h}\right)^2 [\mu_h(\gamma_A+\mu_h)\beta_v b + \mu_v(\mu_h+\gamma_c)(\mu_h+\gamma_A)], \\ B_2 =& \beta_h b \mu_h(\gamma_A+\mu_h)\eta \frac{\beta_h b}{N_h}\frac{\beta_v b \sigma}{N_h \mu_v} - \beta_h b \mu_h(\gamma_A+\mu_h)\mu_h \frac{\beta_v b}{N_h} - \eta^2 \alpha \mu_h \beta_h b(\gamma_c+\mu_h)\frac{\beta_v b}{N_h}, \\ & - \mu_v(\mu_h+\gamma_c)(\mu_h+\gamma_A)\frac{\beta_h b}{N_h}\mu_h - \mu_v(\mu_h+\gamma_c)(\mu_h+\gamma_A)(\alpha+\mu_h)\eta\frac{\beta_h b}{N_h}, \\ B_3 =& \mu_h \mu_v(\gamma_A+\mu_h)(\gamma_c+\mu_h)(\alpha+\mu_h)(R_0-1). \end{aligned} \tag{16}$$

We can see that the value of $p(0)$ in eq. (15) is equal to B_3 as shown in eq. (16). Since $R_0 > 1$, then $B_3 > 0$. Furthermore, it is important to note that all the parameters are positive, which implies that B_1 is negative. Consequently, $p(I_v)$ exhibits a concave downward behavior. Moreover, we have $B_2^2 - 4B_1B_3 > 0$, it implies that $p(I_v)$ has only one positive solution, namely I_v^*, and we conclude that our endemic equilibrium point is $E_1 = (S_c^*, I_c^*, S_A^*, I_A^*, I_v^*)$, where

$$\begin{aligned} S_c^* &= \frac{\mu_h N_h}{\frac{\beta_h b}{N_h} I_v^* + \mu_h + \alpha}, \\ S_A^* &= \frac{\alpha \mu_h N_h}{(\frac{\beta_h b}{N_h} I_v^* + \mu_h + \alpha)(\eta \frac{\beta_h b}{N_h} I_v^* + \mu_h)}, \\ I_c^* &= \frac{\beta_h b \mu_h I_v^*}{(\mu_h+\gamma_c)(\frac{\beta_h b}{N_h} I_v^* + \mu_h + \alpha)}, \\ I_A^* &= \frac{\alpha \mu_h \eta \beta_h b I_v^*}{(\gamma_A+\mu_h)(\frac{\beta_h b}{N_h} I_v^* + \mu_h + \alpha)(\eta \frac{\beta_h b}{N_h} I_v^* + \mu_h)}. \end{aligned}$$

■

In the next subsection, we explain the stability analysis for the endemic equilibrium point.

3.6. Stability analysis of endemic equilibrium point: special case

The stability of the endemic equilibrium is determined by looking at the eigenvalues of the Jacobian evaluated at E_1. Based on eq. (13) at E_1, we have

$$J(E_1) = \begin{bmatrix} -\frac{\beta_h b}{N_h} I_v^* - \mu_h - \alpha & 0 & 0 & 0 & -\frac{\beta_h b}{N_h} S_c^* \\ \frac{\beta_h b}{N_h} I_v^* & -\mu_h - \gamma_c & 0 & 0 & \frac{\beta_h b}{N_h} S_c^* \\ \alpha & 0 & -\eta \frac{\beta_h b}{N_h} I_v^* - \mu_h & 0 & -\eta \frac{\beta_h b}{N_h} S_A^* \\ 0 & 0 & \eta \frac{\beta_h b}{N_h} I_v^* & -\gamma_A - \mu_h & \eta \frac{\beta_h b}{N_h} S_A^* \\ 0 & \frac{\beta_v b}{N_h}\frac{\sigma}{\mu_v} - \frac{\beta_v b}{N_h} I_v^* & 0 & \frac{\beta_v b}{N_h}\frac{\sigma}{\mu_v}\eta - \eta \frac{\beta_v b}{N_h} I_v^* & -\frac{\beta_v b}{N_h} I_c^* - \eta \frac{\beta_v b}{N_h} I_A^* - \mu_v \end{bmatrix}, \tag{17}$$

where S_c^*, S_A^*, I_v^* are given in Theorem 4. The characteristic equation will be a fifth-order polynomial in λ, with complex eigenvalue expressions, making analysis challenging. However, the analysis can be simplified by making an additional assumption that no adults are infected with dengue. It can be explained as follows.

In the proposed mathematical model for dengue virus transmission, the human population is divided into two main groups: children and adults. In this context, children are defined as individuals under 15, while adults refer to those aged 15 years and above [22].

In Indonesia, where dengue fever is endemic, children are consistently identified as the most affected demographic. A national seroprevalence study indicated that over 80% of children have been exposed to the dengue virus [19]. Furthermore, a study conducted in Yogyakarta revealed that 88% of children possessed dengue antibodies [20]. The high exposure to dengue during childhood suggests that most adults in endemic areas have acquired at least partial immunity to the same dengue virus serotype [23]. In light of these considerations, the model is simplified by removing the infectious class for the adult population, allowing for a more focused analysis on the age group most affected by dengue. Therefore, in the specific case of System (1), we put $\eta = 0$. In this subsection, we assume that adults are immune to dengue infections, which is why they do not become infected. Consequently, the compartments I_A and R_A will not be present. The dynamics of our host-vector model are

$$\begin{aligned}
\frac{dS_c}{dt} &= \mu_h N_h - \frac{\beta_h b}{N_h} S_c I_v - \mu_h S_c - \alpha S_c, \\
\frac{dI_c}{dt} &= \frac{\beta_h b}{N_h} S_c I_v - \mu_h I_c - \gamma_c I_c, \\
\frac{dR_c}{dt} &= \gamma_c I_c - \mu_h R_c - \alpha R_c, \\
\frac{dS_A}{dt} &= \alpha S_c - \mu_h S_A, \\
\frac{dS_v}{dt} &= \sigma - \frac{\beta_v b}{N_h} S_v I_c - \mu_v S_v, \\
\frac{dI_v}{dt} &= \frac{\beta_v b}{N_h} S_v I_c - \mu_v I_v..
\end{aligned} \tag{18}$$

We have a more straightforward analysis using system (18). Introducing the new total population for human, i.e., $N_h = S_c + I_c + R_c + S_A$, and for the mosquito, i.e., $N_v = S_v + I_v$. We obtain that

$$\frac{dN_v}{dt} = \sigma - \mu_v N_v.$$

For time $t \to \infty$, we have N_v tends to $\frac{\sigma}{\mu_v}$. Since R_c and S_A only appear in their own equations, the system (18) can be simplified.

$$\begin{aligned}
\frac{dS_c}{dt} &= \mu_h N_h - \frac{\beta_h b}{N_h} S_c I_v - \mu_h S_c - \alpha S_c, \\
\frac{dI_c}{dt} &= \frac{\beta_h b}{N_h} S_c I_v - \mu_h I_c - \gamma_c I_c, \\
\frac{dS_v}{dt} &= \sigma - \frac{\beta_v b}{N_h} S_v I_c - \mu_v S_v, \\
\frac{dI_v}{dt} &= \frac{\beta_v b}{N_h} S_v I_c - \mu_v I_v..
\end{aligned} \tag{19}$$

We focus on determining the endemic equilibrium point for system (19). Basically, the values of (19) are the same as in system (2) by taking $\eta = 0$; therefore, the basic reproduction number for

system (19) is as follows.

$$R_0^{**} = \frac{\beta_v b(\sigma/\mu_v)}{N_h(\mu_h+\alpha)(\mu_h+\gamma_c)}\frac{\beta_h b\mu_h}{\mu_v}. \tag{20}$$

Moreover, the endemic equilibrium point in the system (19) is the same as eq. (2) when $\eta = 0$. In this case, the endemic equilibrium point is $E_1^* = (S_c^{**}, I_c^{**}, S_v^{**}, I_v^{**})$, where

$$\begin{aligned}
S_c^{**} &= \frac{\mu_v(\mu_h+\gamma_c)(\frac{\beta_v b}{\mu_v N_h}I_c^{**}+1)}{\frac{\beta_h b}{N_h}\frac{\beta_v b}{\mu_v N_h}\sigma},\\
I_c^{**} &= \frac{(\mu_h+\gamma_c)\mu_v(\mu_h+\alpha)(R_0^{**}-1)}{(\mu_h+\gamma_c)(\frac{\beta_h b}{N_h}\frac{\beta_v b}{\mu_v N_h}\sigma+\mu_v(\mu_h+\alpha)\frac{\beta_v b}{\mu_v N_h})},\\
S_v^{**} &= \frac{\sigma}{\frac{\beta_v b}{N_h}I_c^{**}+\mu_v},\\
I_v^{**} &= \frac{\frac{\beta_v b}{\mu_v N_h}\sigma I_c^{**}}{\mu_v(\frac{\beta_v b}{\mu_v N_h}I_c^{**}+1)}.
\end{aligned}$$

Consequently, we have a positive endemic equilibrium E_1^* if $R_0^{**} > 1$. Furthermore, the Jacobian based on system (19) is

$$J(E_1^*) = \begin{bmatrix}
-\frac{\beta_h b}{N_h}I_v^{**}-\mu_h-\alpha & 0 & 0 & -\frac{\beta_h b}{N_h}S_c^{**}\\
\frac{\beta_h b}{N_h}I_v^{**} & -\mu_h-\gamma_c & 0 & \frac{\beta_h b}{N_h}S_c^{**}\\
0 & -\frac{\beta_v b}{N_h}S_v^{**} & -\frac{\beta_v b}{N_h}I_c^{**}-\mu_v & 0\\
0 & \frac{\beta_v b}{N_h}S_v^{**} & \frac{\beta_v b}{N_h}I_c^{**} & -\mu_v
\end{bmatrix}. \tag{21}$$

The eigenvalues of eq. (21) are solutions to the following equations

$$\lambda^4 + D_1\lambda^3 + D_2\lambda^2 + D_3\lambda + D_4 = 0 \tag{22}$$

where

$$\begin{aligned}
D_1 &= \frac{\beta_h b}{N_h}I_v^{**}+\alpha+2\mu_h+\gamma_c+\frac{\beta_v b}{N_h}I_c^{**}+2\mu_v,\\
D_2 &= (\frac{\beta_h b}{N_h}I_v^{**}+\alpha+\mu_h)(\mu_h+\gamma_c)+(\frac{\beta_h b}{N_h}I_v^{**}+\alpha+\mu_h)(\frac{\beta_v b}{N_h}I_c^{**}+\mu_v)+(\frac{\beta_h b}{N_h}I_v^{**}+\alpha+\mu_h)\mu_v\\
&\quad+(\mu_h+\gamma_c)(\frac{\beta_v b}{N_h}I_c^{**}+\mu_v)+(\mu_h+\gamma_c)\mu_v+(\frac{\beta_v b}{N_h}I_c^{**}+\mu_v)\mu_v,\\
D_3 &= (\frac{\beta_h b}{N_h}I_v^{**}+\alpha+\mu_h)(\mu_h+\gamma_c)(\frac{\beta_v b}{N_h}I_c^{**}+\mu_v)+(\frac{\beta_h b}{N_h}I_v^{**}+\alpha+\mu_h)(\mu_h+\gamma_c)\mu_v,\\
&\quad+(\frac{\beta_h b}{N_h}I_v^{**}+\alpha+\mu_h)(\frac{\beta_v b}{N_h}I_c^{**}+\mu_v)\mu_v+(\mu_h+\gamma_c)(\frac{\beta_v b}{N_h}I_c^{**}+\mu_v)\mu_v,\\
D_4 &= (\frac{\beta_h b}{N_h}I_v^{**}+\alpha+\mu_h)(\mu_h+\gamma_c)(\frac{\beta_v b}{N_h}I_c^{**}+\mu_v)\mu_v.
\end{aligned}$$

By the Routh-Hurwitz criterion, then all the eigenvalues are negative. Therefore, the endemic equilibrium E_1^* is asymptotically stable.

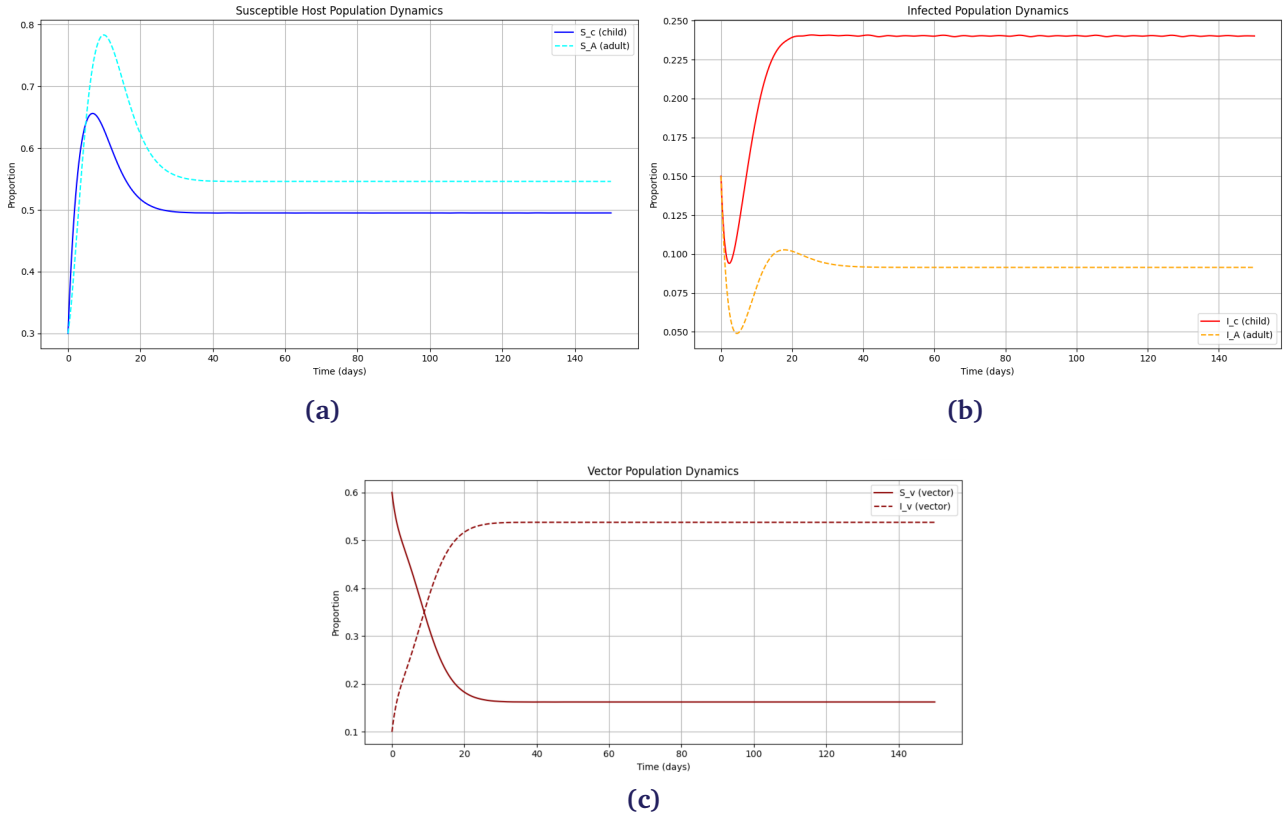

Figure 2. (a) The dynamics of susceptible children and susceptible adult when $R_0 > 1$. (b) The dynamics of infected children and infected adult when $R_0 > 1$ (c) The dynamics of vector population when $R_0 > 1$.

4. Numerical Results

In this section, we visualize the analytical result to show the population dynamics. We use Python 3.12.11 to perform numerical simulations, solve the differential equation system, and illustrate the dynamics of the model. The values of each parameter are shown in Table 1. The initial conditions used are: $S_c(0) = 0.3, I_c(0) = 0.15, S_A(0) = 0.3, I_A(0) = 0.15, I_v(0) = 0.10$. For numerical simulation needs, the population variables are normalized. Consequently, an initial condition of 0.3 for the susceptible class represents 300 susceptible individuals among 1000 people. Furthermore, using $\eta = 0.48, \beta_v = 0.6, \beta_h = 0.5$, we obtain $R_0 > 1$.

Table 1. Parameter value

Parameter	Value	References
μ_h	0.000039	assumed
β_h	[0,1]	vary
b	1	assumed
α	0.000283	[12]
γ_c	0.071	[24]
γ_A	0.071	[1]
η	[0,1]	vary
β_v	[0,1]	vary
μ_v	0.036	[25]

Although an explicit analysis at the endemic equilibrium point of system (2) was not conducted,

the visualization was based on parameter values provided in Table 1. The following graphical representations show the interaction among variables and the disease dynamics. In Figure 2a, the susceptible children (S_c) are lower than those of adults (S_A), which is due to the relatively high infection rate that leads individuals from S_c to I_c. As shown in Figure 2b, the infected children (I_c) exhibit a sharper increase compared to infected adults (I_A). This observation supports the relevance of the analysis conducted in Subsection 3.6. Consequently, the susceptible vector population S_v will gradually decline as more vectors become infected through contact with infectious hosts, and the infected vector population I_v will increase as shown in Figure 2c.

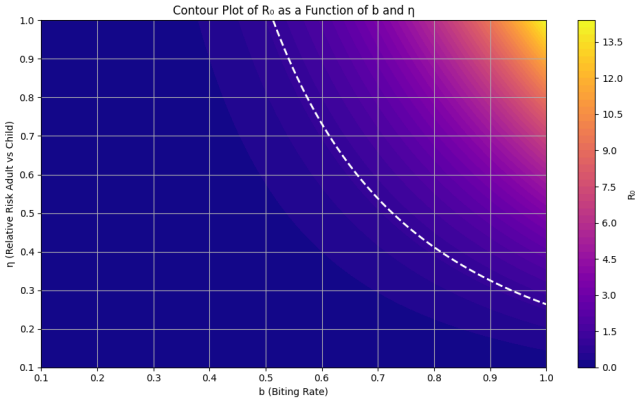

Figure 3. The contour plot of R_0 as a function of b and η. The dashed line denotes the threshold $R_0 = 1$ separating the disease-free and endemic regions

The basic reproduction value shows that the parameters b and η give a quadratic form. Intuitively, this makes it possible that both parameters significantly affect the R_0 value. Figure 3 presents a contour map of the basic reproduction number as a function of mosquito biting rate (b) and the susceptibility ratio of adults to children (η).

The color scheme in the graph reflects the magnitude of R_0, where brighter colors indicate higher values, and darker shades represent lower values. The white dashed line represents the isocline $R_0 = 1$, which separates two key regions in disease dynamics. The area above this line $(R_0 > 1)$ indicates parameter combinations that may lead to an epidemic, while the region below $(R_0 < 1)$ corresponds to conditions under which disease transmission is unsustainable and tends to decline. Therefore, combinations of b and η that result in $R_0 > 1$ should receive particular attention in disease control strategies, as they can lead to outbreak scenarios. In contrast, reducing b or lowering adult susceptibility η has been shown to effectively shift the system toward a non-epidemic region.

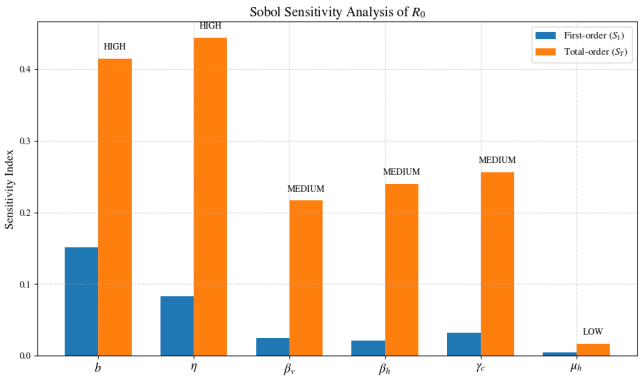

Figure 4. Sobol sensitivity analysis of R_0, showing the first order (S_1) and total-order (S_T) sensitivity indices for each parameter

The sensitivity analysis using the Sobol method (Figure 4) reveals that the mosquito biting rate (b) and the susceptibility ratio between adults and children (η) exhibit the highest total-order sensitivity indices, indicating their dominant influence on the basic reproduction number (R_0). This suggests that variations in these two parameters significantly affect the potential for dengue transmission within the model. In contrast, the transmission probabilities from humans to vectors (β_v) and from vectors to humans (β_h) show moderate influence, highlighting their supporting but less dominant role in determining the disease dynamics. Interestingly, the sensitivity index of β_v is lower than that of β_h, which may reflect the structure of the model in which vector-to-human transmission plays a more sustained role in maintaining the infection cycle, especially under conditions where host infectivity and mosquito population dynamics are tightly coupled.

5. Conclusion

In this study, we proposed an age-structured host-vector model that extends the classical dengue transmission framework by incorporating key biological and behavioral parameters, such as the mosquito biting rate (b) and the relative risk of being bitten (η). The model differentiates between children and adults, assuming that adults in endemic areas are largely immune and contribute less to disease dynamics. We derived the basic reproduction number R_0 using the next-generation matrix and established conditions for the local stability of the disease-free equilibrium, while analyzing the stability of endemic equilibria through a specific case. Our findings reveal that both b and η enter R_0 in a quadratic form, underscoring the disproportionate impact of children's exposure on dengue transmission.

Although the endemic equilibrium was analyzed under the assumption $\eta = 0$, this simplification was made to facilitate analytical tractability. However, the sensitivity analysis confirms that η has a significant impact on the R_0. This indicates that while it was set to zero for the equilibrium analysis, its influential role in determining disease transmission potential supports and reinforces the insights obtained from the endemic analysis. Thus, both approaches complement each other in highlighting the importance of age-related susceptibility in the model dynamics.

This result suggests that intervention strategies focusing on reducing mosquito contact with children through vector control, vaccination, or behavior-based prevention could significantly lower transmission rates. The model's assumptions are particularly relevant in endemic regions where repeated exposure leads to early-life immunity. Future work may consider reintroducing adult infection classes to study secondary infections or include spatial heterogeneity and seasonality for a more comprehensive analysis.

Supplementary Information

Author Contributions. **Eminugroho Ratna Sari:** Conceptualization, methodology, formal analysis, writing–original draft preparation. **Dwi Lestari:** Software, validation, data curation, visualization. **Nikenasih Binatari:** Investigation, resources, writing–review and editing. **Retno Subekti:** Supervision, writing–review and editing. **Fitriana Yuli Saptaningtyas:** Project administration, funding acquisition, supervision.

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Data availability. Not applicable.

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