

Local Stability Analysis of Mathematic Model SEIHR-VW on Dengue Haemorrhagic Fever Transmission

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Abstract—Dengue fever is caused by the dengue virus (DENV) and is mainly transmitted by mosquitoes, particularly *Aedes aegypti*. In this study, we develop a mathematical model to describe and analyze how dengue spreads within a population. The mathematical model is expressed as a nonlinear system of differential equations and consists of seven compartments (SEIHR-VW): susceptible, exposed, infected, hospitalized, and recovered humans, along with susceptible and infected mosquitoes. The model has two possible equilibrium points: a non-endemic and endemic equilibrium point. To better understand the dynamics of the model, we calculate the basic reproduction number (\mathcal{R}_0) using the Next Generation Matrix (NGM) method, and then the Routh-Hurwitz criterion method is applied to analyze the local stability of both equilibrium points. The results indicate that the non-endemic equilibrium point is asymptotically stable when $\mathcal{R}_0 < 1$, while the endemic equilibrium point becomes asymptotically stable when $\mathcal{R}_0 > 1$. In general, our analysis concludes that the proposed dengue transmission model is asymptotically stable at the endemic equilibrium point, with $\mathcal{R}_0 = 3.85011$.

Index Terms - Local Stability Analysis, Dengue Transmission, Mathematical Modeling.

I. INTRODUCTION

DENGUE Haemorrhagic Fever (DHF) is one of the fastest spreading vector-borne diseases. It is caused by the dengue virus (DENV) and transmitted to humans through the bite of female *Aedes aegypti* mosquito [1]. The dengue virus belongs to the genus *Flavivirus*, family *Flaviviridae*, and to date four serotypes have been identified: DEN-1, DEN-2, DEN-3, DEN-4, each of which triggers a different immune response in the human body [2]. DHF has become a seasonal disease and remains a major health problem in tropical and subtropical countries, including Indonesia.

In April 2024, the World Health Organization (WHO) announced that around half of the world's population is now at risk of dengue fever, with an estimated 100-400 million infections occurring each year [3]. Currently, DHF is an endemic disease in more than 100 countries within WHO regions, including Africa, the Americas, the Eastern Mediterranean, Southeast Asia, and the Western Pacific [3]. In Indonesia, according to data released by the Ministry of Health

in November 2024, through its official website, there were 114,720 cases of dengue infection in 2023, with 894 reported deaths. Until the 43rd week of 2024, a total of 210,644 cases of dengue infection have been reported, with 1,239 deaths caused by DHF occurring in 259 districts / cities in 32 provinces [4].

A possible effort to control the spread of DHF is through research and operational studies in applied technology and epidemiology, where analysis in these fields requires a significant role for mathematics, particularly mathematical modeling. A mathematical model is a representation that uses mathematical structure and expressions to explain and understand real-world problems [5], [6]. As a branch of applied mathematics, mathematical modeling provides a systematic framework for translating complex phenomena into mathematical language, thereby allowing for deeper insights and more accurate analysis [7]. In the context of infectious disease transmission, mathematical modeling is especially important, as it can be used to predict outbreaks, assess the impact of interventions, and evaluate control strategies [8], [9], [10].

Many mathematical models have been introduced to understand how dengue transmission depends on several important factors. Chen and Hsieh [11] proposed a mathematical model on dengue transmission by considering the effect of temperature. They found that a higher transmission of dengue occurred when the temperature was equal to 28°C. Recent studies on mathematical modeling have used dengue incidence data from various regions, including Kupang [12], Palu [13], Semarang [14] and China [15]. Some examples of previous studies with complex mathematical models are as follows: The SIAPR-SI model was proposed by Asamoah et al. [16], considers compartments of individuals with temporary (partial) immunity and asymptomatic infected individuals. Another study by Khan, M. A and Fatmawati [17], analyzed the spread of dengue fever using the SEIHR-SEI model, which involves a hospitalized/notified infectious human subpopulation and an exposed mosquito subpopulation. They also applied optimal control strategies to investigate the impact of prevention and insecticide use on reducing dengue transmission. In 2019, Indrajit Gosh et al. [18] analyzed a dengue transmission model using the $S_1S_2EAIPR - SEI$, which considers high-risk and low-risk susceptible individuals, asymptomatic individuals, hospitalized/notified individuals, and exposed mosquitoes as model variables.

Another study reported in 2018 by Augusto and Khan [19], proposed the SVEIR-SEI model, which includes a vaccinated human subpopulation and an exposed mosquito subpopulation.

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They also applied optimal control strategies to investigate the impact of vaccination and insecticide use in reducing dengue transmission. Next, in 2022, Abidemi and Aziz [20] proposed the SVEIR-ASEI model, which includes a vaccinated human subpopulation and a mosquito subpopulation in the aquatic phase, while also analyzing the impact of three optimal control strategies: vaccination, treatment, and insecticide application. The study by Aldila et al. [21] analyzed the SAEIHR-VW model for dengue transmission, which involves a compartment for susceptible individuals who are aware of dengue infection and considering factors such as: the impact rate of media campaigns, case detection and hospital capacity as parameters in the model.

Motivated by the literature above, this study focuses on the development of a host-vector model for dengue transmission by incorporating the hospitalization of infected cases. The human population (host) is modeled using the SEIHR model, while the mosquito population (vector) is modeled using the VW model. Based on the previous study by Aldila et al. [21], this research introduces two additional parameters into the SEIHR-VW model: the hospitalization rate of infected individuals and the recovery rate of hospitalized individuals. Despite these additions, the compartment for aware susceptible individuals (A) is not included, consequently, the media campaign parameter, which is directly associated with the (A) compartment, is also omitted. This decision is based on the observation that, in reality, increasing public awareness of the dangers of dengue infection is extremely challenging, even with media campaigns from government. Individual awareness tends not to be long-lasting, as people eventually become careless.

Furthermore, this study excludes the parameters for case detection and hospital capacity, as reported in [21], these factors were found to have minimal impact on reducing dengue transmission. Additionally, this research considers that recovered individuals may lose their temporary immunity over time, allowing for the possibility of reinfection as they return to the susceptible subpopulation (relapse cases). Based on the model, the basic reproduction number (\mathfrak{R}_0) will be determined, and both the disease-free equilibrium point (DFE) and the endemic equilibrium point (EE) will be obtained. Using the value of \mathfrak{R}_0 and these equilibrium points, a local stability analysis will be conducted using the Routh-Hurwitz criterion. Finally, a numerical simulation will be conducted to observe the dynamic behavior of dengue transmission using the modified model.

II. MATHEMATICAL MODEL FORMULATION

The spread of dengue model in this article, represent the interaction between seven subpopulations, that divided into five human population (host), S denotes susceptible population vulnerable to mosquito bites, E denotes the population exposed to dengue infection, I and H denotes for the non-hospitalized and hospitalized infected populations and R denotes the recovered population from dengue infection that has temporal immunity to dengue virus. Thus, total human population denoted by N_h is given as $N_h = S + E + I + H + R$.

On the other hand, the population of mosquitoes (vector) divided into V and W denotes for susceptible mosquitoes and infected mosquitoes. Thus, total mosquito population denoted by $N_m = V + W$. A transmission diagram to describe all the above interaction between compartments is provided in Fig. 1, and the description of the parameters are given in Table 1.

The interaction starts from individuals entering the human population, who are assumed to be newborns with a recruitment rate of θ_h , and are considered to always be in a healthy condition. Migration is considered negligible in this model. The next stage involves direct interaction between mosquitoes and humans, in which infected mosquito subpopulations (W) bite susceptible individuals (S) at an infection rate denoted by β_h . As a result, susceptible individuals who are bitten by infected mosquitoes transition into the exposed or latent human subpopulation (E). After an incubation period of the dengue virus (approximately 8 days), exposed individuals will become infectious individuals that are non-hospitalized (I) and hospitalized infectious individuals (H), with infection rate denoted by $q_1\delta$ and $q_2\delta$.

Individuals in the infected subpopulation can become part of the hospitalized subpopulation with transmission rate ω , because the dengue disease has progressed to a more severe state than before, thus requiring more intensive care in the hospitals. Then, infected individual is assumed to recover at a constant rate ρ_1 , while the hospitalized individual recover by ρ_2 . The number of recovered individual subpopulation (R) increases due to the recovery of infected and hospitalized individuals, the R decrease because of the loss of temporal immunity at a rate of η . All the human populations are assumed to have decreased due to the natural death rate of μ_h . Then for the susceptible mosquitoes (V) will increase due to the birth of newborns with a natural birth rate of θ_m and decrease due to the presence of mosquitoes infected with the dengue virus.

The susceptible mosquitoes get infected by dengue virus (DENV) after they bite infected individuals in I or H , at

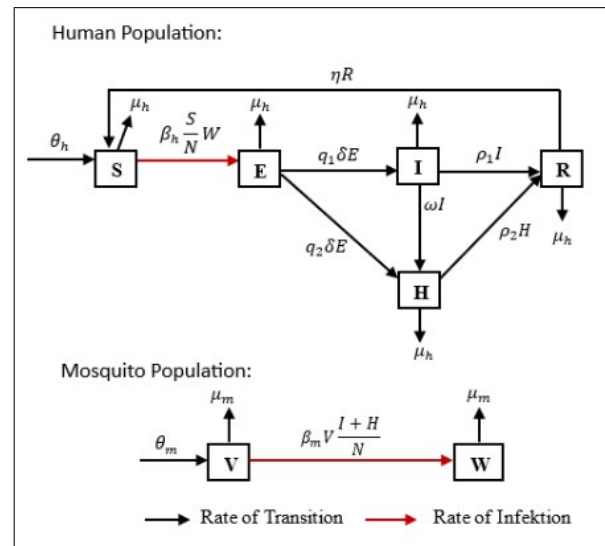


Fig. 1: Transmission Diagram of System (1).

TABLE I: Description of Parameter in System (1).

Par	Description	Units
θ_h	Recruitment rate of human population.	$\frac{\text{individual}}{\text{day}}$
θ_m	Recruitment rate of mosquito population.	$\frac{\text{mosquitoes}}{\text{day}}$
N_H	Total of human population.	individual
N_M	Total of mosquito population.	mosquitoes
μ_h	Natural death rate of human population.	$\frac{1}{\text{day}}$
μ_m	Natural death rate of mosquito population.	$\frac{1}{\text{day}}$
β_h	Infection rate for human population.	$\frac{\text{individual}}{\text{day} \times \text{mosquitoes}}$
β_m	Infection rate for mosquito population.	$\frac{1}{\text{day}}$
δ	Transition rate due to virus incubation period.	$\frac{1}{\text{day}}$
q_1	Proportion of exposed individuals who become infected but are not hospitalized.	-
q_2	Proportion of exposed individuals who become infected and are hospitalized.	-
ω	Rate of hospitalization and/or notification of infected human subpopulation.	$\frac{1}{\text{day}}$
ρ_1	Recovery rate of infected human subpopulation.	$\frac{1}{\text{day}}$
ρ_2	Recovery rate of hospitalized human subpopulation.	$\frac{1}{\text{day}}$
η	Waning rate of temporal immunity.	$\frac{1}{\text{day}}$

infection rate β_m . We assumed that the number of susceptible and infected mosquitoes decreases due to the natural death rate μ_m . We do not include the recovered stage of mosquitoes because it has short life time period, which does not give a chance to mosquitoes to recover from dengue infection. The mathematical model describe transmission of dengue infection in this article is given by the following system of ordinary

differential equations (1):

$$\begin{aligned}
\frac{dS}{dt} &= \theta_h + \eta R - \beta_h \frac{S}{N} W - \mu_h S, \\
\frac{dE}{dt} &= \beta_h W \frac{S}{N} - q_1 \delta E - q_2 \delta E - \mu_h E, \\
\frac{dI}{dt} &= q_1 \delta E - (\rho_1 + \mu_h + \omega) I, \\
\frac{dH}{dt} &= q_2 \delta E + \omega I - \rho_2 H - \mu_h H, \\
\frac{dR}{dt} &= \rho_1 I + \rho_2 H - (\eta + \mu_h) R, \\
\frac{dV}{dt} &= \theta_m - \beta_m V \frac{I+H}{N} - \mu_m V, \\
\frac{dW}{dt} &= \beta_m V \frac{I+H}{N} - \mu_m W,
\end{aligned} \tag{1}$$

Initial state: $S(0) \geq 0, E(0) \geq 0, I(0) \geq 0, H(0) \geq 0, R(0) \geq 0, V(0) \geq 0, W(0) \geq 0$.

Because the life time period of mosquitoes is shorter than that human $\mu_m^{-1} \ll \mu_h^{-1}$, that make the mosquito population has a faster dynamic, compare to the human population, to reach its equilibrium point. Hence, this study used the Quasi Steady-State Approximation (QSSA) method to approach system (1) when the mosquito population has already reached its equilibrium [21]. Therefore, solving $\frac{dV}{dt} = 0$ and $\frac{dW}{dt} = 0$ considering V and W , we obtain V^* and W^* ,

$$\begin{aligned}
V^* &= \frac{\theta_m N}{\beta_m (I+H) + \mu_m N}, \\
W^* &= \frac{\theta_m \beta_m (I+H)}{\mu_m (\beta_m (I+H) + \mu_m N)}.
\end{aligned}$$

This article used the same assumption as in [21], where $\beta_m \mu_m = \frac{1}{2}$, assume that N is constant, and $\frac{\beta_h \theta_m}{\mu_m N} = \beta$ and $2\mu_m^2 = \mu_v$. Then, substituting V^* and W^* into system (1), we obtain,

$$\begin{aligned}
\frac{dS}{dt} &= \theta_h + \eta R - \left(\frac{\beta S(I+H)}{(I+H) + \mu_v N} \right) - \mu_h S, \\
\frac{dE}{dt} &= \left(\frac{\beta S(I+H)}{(I+H) + \mu_v N} \right) - q_1 \delta E - q_2 \delta E - \mu_h E, \\
\frac{dI}{dt} &= q_1 \delta E - (\rho_1 + \mu_h + \omega) I, \\
\frac{dH}{dt} &= q_2 \delta E + \omega I - \rho_2 H - \mu_h H, \\
\frac{dR}{dt} &= \rho_1 I + \rho_2 H - (\eta + \mu_h) R.
\end{aligned} \tag{2}$$

III. RESULTS AND ANALYSIS

A. Basic Reproduction Number

The basic reproduction number (\mathfrak{R}_0) is the average number of new infection cases generated by subpopulations that can transmit the infection. If $\mathfrak{R}_0 > 1$, then each infected individual can spread the disease to more than one susceptible individual, potentially leading to an epidemic because the disease-free equilibrium (DFE) point becomes unstable [22]. However, if $\mathfrak{R}_0 < 1$, the DFE point will be locally asymptotically stable and the situation can be brought under control [23], [24].

We obtain the basic reproduction number (\mathfrak{R}_0) using the next-generation matrix (NGM) approach, based on the number of infected individuals in the dengue model described in system (2) [24]. The infected compartments are represented by E, I, H , and R . Let $\mathbf{x} = (E, I, H, R)$ and rewrite system (2) as follows:

$$\frac{dx}{dt} = F(x) - V(x), x = [E, I, H, R]^T$$

$$F(x) = \begin{bmatrix} F_1 \\ F_2 \\ F_3 \\ F_4 \end{bmatrix} = \begin{bmatrix} \frac{\beta S(I+H)}{(I+H)+\mu_v N} \\ 0 \\ 0 \\ 0 \end{bmatrix},$$

$$V(x) = \begin{bmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{bmatrix} = \begin{bmatrix} (q_1\delta + q_2\delta + \mu_h)E \\ (\rho_1 + \mu_h + \omega)I - q_1\delta E \\ \rho_2 H + \mu_h H - q_2\delta E - \omega I \\ (\eta + \mu_h)R - \rho_1 I - \rho_2 H \end{bmatrix}.$$

Matrix $\mathcal{F}(\mathcal{E}^0)$ and $\mathcal{V}(\mathcal{E}^0)$ are represented for the Jacobian matrices of $F(x)$ and $V(x)$ at a non-endemic equilibrium point $\mathcal{E}^0 = (S^0, E^0, I^0, H^0, R^0) = (\frac{\theta_h}{\mu_h}, 0, 0, 0, 0)$. Then, using NGM approach, $\text{NGM} = \mathcal{F}(\mathcal{E}^0) \times \mathcal{V}^{-1}(\mathcal{E}^0)$, we can determine the largest eigen value of the matrix NGM. The reproduction number obtained are as follows:

$$\mathfrak{R}_0 = \frac{\beta\delta((q_1+q_2)(\mu_h+\omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v(\delta(q_1+q_2) + \mu_h)(\rho_1 + \mu_h + \omega)(\rho_2 + \mu_h)}$$

B. Equilibrium Points

The mathematical model for dengue spread in system (2) has an equilibrium point if it's satisfied:

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dH}{dt} = \frac{dR}{dt} = 0. \quad (3)$$

System (2) has two equilibrium points known as endemic equilibrium (EE) denoted by $\mathcal{E}^* = (S^*, E^*, I^*, H^*, R^*)$ and disease-free/non-endemic equilibrium (DFE) denoted by $\mathcal{E}^0 = (S^0, E^0, I^0, H^0, R^0) = (\frac{\theta_h}{\mu_h}, 0, 0, 0, 0)$. Hence, we calculate the form of the endemic equilibrium point (EE) using Maple software and obtain the values $\mathcal{E}^* = (S^*, E^*, I^*, H^*, R^*)$ as follows:

$$S^* = \frac{NB(\mu_v(\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B) + A\delta(\eta + \mu_h))}{A\delta(\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B(\eta + \mu_h + \beta))},$$

$$E^* = -\frac{N(\eta + \mu_h)(\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)(\mu_v B - \delta\beta A)}{A\delta(\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B(\eta + \mu_h + \beta))},$$

$$I^* = -\frac{Nq_1(\eta + \mu_h)(\rho_2 + \mu_h)(\mu_v B - \delta\beta A)}{A(\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B(\eta + \mu_h + \beta))},$$

$$H^* = -\frac{N((q_1 + q_2)\omega + (\mu_h + \rho_1)q_2)(\eta + \mu_h)(\mu_v B - \delta\beta A)}{A(\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B(\eta + \mu_h + \beta))},$$

$$R^* = -\frac{N((q_1 + q_2)\rho_2(\rho_1 + \omega) + (q_1\rho_1 + q_2\rho_2)\mu_h)(\mu_v B - \delta\beta A)}{A(\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B(\eta + \mu_h + \beta))},$$

with values of A and B as follows:

$$A = ((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1),$$

$$B = (\delta(q_1 + q_2) + \mu_h)(\rho_1 + \mu_h + \omega)(\rho_2 + \mu_h).$$

C. Stability Analysis

Stability analysis is used to determine the behavior around the equilibrium point. Non-endemic equilibrium point (DFE) is the case in which there are no individuals infected by dengue

disease in a population. Then, the local stability analysis of the non-endemic equilibrium point for system (2) is expressed in the following theorem.

Theorem 1. If $\mathfrak{R}_0 < 1$ then non-endemic equilibrium point $\mathcal{E}^0 = (S^0, E^0, I^0, H^0, R^0)$ is locally asymptotically stable and if $\mathfrak{R}_0 > 1$, then $\mathcal{E}^0 = (S^0, E^0, I^0, H^0, R^0)$ is unstable [24].

Proof:

Assume $\mathcal{E}^0 = (S^0, E^0, I^0, H^0, R^0) = (\frac{\theta_h}{\mu_h}, 0, 0, 0, 0)$, we can determine the eigen values of the Jacobian matrix at \mathcal{E}^0 :

$$\det(J(\mathcal{E}^0) - YI) = 0$$

Then, the characteristic equation is obtained, as follows:

$$(Y + \mu_h)(Y + \eta + \mu_h)(a_0 Y^3 + a_1 Y^2 + a_2 Y + a_3) = 0$$

with,

$$a_0 = 1,$$

$$a_1 = 3\mu_h + q_1\delta + q_2\delta + \omega + \rho_1 + \rho_2,$$

$$a_2 = 3\mu_h^2 + 2(\omega + \rho_1 + \rho_2)\mu_h + \rho_2(\omega + \rho_1)$$

$$+ \delta(q_1 + q_2)(2\mu_h + \omega + \rho_1 + \rho_2) - \left(\frac{\beta\delta(q_1 + q_2)}{\mu_v}\right),$$

$$a_3 = (\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)$$

$$- \left(\frac{\beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v}\right).$$

According to the Routh-Hurwitz criterion, \mathcal{E}^0 is locally asymptotically stable if the coefficients of its characteristic equation have a negative root or a negative real part and its can satisfy the conditions if $a_1, a_2, a_3 > 0$ and $a_1 a_2 - a_0 a_3 > 0$.

- First, it will be shown that $a_1 > 0$, with $a_1 = 3\mu_h + q_1\delta + q_2\delta + \omega + \rho_1 + \rho_2$, because $\mu_h, \delta, q_1, q_2, \omega, \rho_1, \rho_2 > 0$, then $a_1 = 3\mu_h + q_1\delta + q_2\delta + \omega + \rho_1 + \rho_2 > 0$.
- Second, shown that $a_2 > 0$, with

$$a_2 = 3\mu_h^2 + 2(\omega + \rho_1 + \rho_2)\mu_h + \rho_2(\omega + \rho_1)$$

$$+ \delta(q_1 + q_2)(2\mu_h + \omega + \rho_1 + \rho_2) - \left(\frac{\beta\delta(q_1 + q_2)}{\mu_v}\right).$$

Denoted:

$$\mathcal{A} = (3\mu_h^2 + 2(\omega + \rho_1 + \rho_2)\mu_h + \rho_2(\omega + \rho_1)$$

$$+ \delta(q_1 + q_2)(2\mu_h + \omega + \rho_1 + \rho_2))\mu_v,$$

$$\mathcal{B} = (q_1 + q_2),$$

we can write the equation of a_2 as follows:

$$a_2 = \frac{\mu_v}{\mu_v} (3\mu_h^2 + 2(\omega + \rho_1 + \rho_2)\mu_h + \rho_2(\omega + \rho_1)$$

$$+ \delta(q_1 + q_2)(2\mu_h + \omega + \rho_1 + \rho_2)) - \frac{\beta\delta(q_1 + q_2)}{\mu_v},$$

$$a_2 = \frac{\mathcal{A}}{\mu_v} - \frac{\beta\delta\mathcal{B}}{\mu_v},$$

Since the denominator (μ_v) is positive, a_2 is positive only when the numerator is also positive. Hence, the resulting equation is as follows:

$$\mathcal{A} - \beta\delta\mathcal{B} > 0.$$

So that, the condition for $a_2 > 0$ is fulfilled if $\mathcal{A} > \beta\delta\mathcal{B}$,

- Third, shown that $a_3 > 0$, as follows:

$$a_3 = \frac{(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)}{\mu_v} - \left(\frac{\beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v} \right),$$

$$a_3 = \frac{\mu_v(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)}{\mu_v} - \left(\frac{\beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v} \right).$$

Since the denominator (μ_v) is positive, a_3 is positive, only when the numerator is also positive. Hence, the resulting equation is as follows:

$$\begin{aligned} & \mu_v(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h) \\ & - \beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1) > 0, \\ \iff & \frac{\beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v} \\ & < (\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h), \\ \iff & \frac{\beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)} < 1, \\ & \mathfrak{R}_0 < 1. \end{aligned}$$

This indicates that if $\mathfrak{R}_0 < 1$ then $a_3 > 0$. (as proven).

- Then, shown that $a_1a_2 - a_0a_3 > 0$, with $a_0 = 1$, it can be written as follows:

$$\begin{aligned} a_1a_2 - a_3 &= (3\mu_h + q_1\delta + q_2\delta + \omega + \rho_1 + \rho_2) \\ & \left(\frac{\mu_v}{\mu_v} (3\mu_h^2 + 2(\omega + \rho_1 + \rho_2)\mu_h + \rho_2(\omega + \rho_1)) \right. \\ & + \delta(q_1 + q_2)(2\mu_h + \omega + \rho_1 + \rho_2)) - \frac{\beta\delta(q_1 + q_2)}{\mu_v} \\ & - \left(\frac{\mu_v(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)}{\mu_v} \right. \\ & \left. - \frac{\beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v} \right), \end{aligned}$$

with denoted:

$$\begin{aligned} \mathcal{C} &= (3\mu_h + q_1\delta + q_2\delta + \omega + \rho_1 + \rho_2), \\ \mathcal{D} &= (3\mu_h^2 + 2(\omega + \rho_1 + \rho_2)\mu_h + \rho_2(\omega + \rho_1) \\ & + \delta(q_1 + q_2)(2\mu_h + \omega + \rho_1 + \rho_2))\mu_v, \\ \mathcal{E} &= \beta\delta(q_1 + q_2), \\ \mathcal{F} &= \beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1), \end{aligned}$$

and then we can write the equation of $(a_1a_2 - a_3)$ as follows:

$$\begin{aligned} a_1a_2 - a_3 &= \frac{\mathcal{C}(\mathcal{D} - \mathcal{E})}{\mu_v} + \left(\frac{\mathcal{F}}{\mu_v} \right. \\ & \left(\frac{(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)\mu_v}{(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)\mu_v} \right) \\ & - \frac{\mu_v}{\mu_v} (\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h), \end{aligned}$$

$$\begin{aligned} a_1a_2 - a_3 &= \frac{\mathcal{C}(\mathcal{D} - \mathcal{E})}{\mu_v} \\ & + \left(\frac{(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)\mu_v}{\mu_v} \right. \\ & \left(\frac{\beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)} - 1 \right)), \\ a_1a_2 - a_3 &= \frac{\mathcal{C}(\mathcal{D} - \mathcal{E})}{\mu_v} \\ & + \left(\frac{(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)\mu_v}{\mu_v} \right. \\ & \left. (\mathfrak{R}_0 - 1) \right). \end{aligned}$$

Since the denominator (μ_v) is positive, $(a_1a_2 - a_3)$ is positive, only when the numerator is also positive. Hence, the resulting equation is as follows:

$$\begin{aligned} & \mathcal{C}(\mathcal{D} - \mathcal{E}) + (\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)\mu_v \\ & ((q_1 + q_2)\delta + \mu_h)(\mathfrak{R}_0 - 1), \\ & (\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)\mu_v(\mathfrak{R}_0 - 1) \\ & > -\mathcal{C}(\mathcal{D} - \mathcal{E}), \\ & (\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)\mu_v(1 - \mathfrak{R}_0) \\ & < \mathcal{C}(\mathcal{D} - \mathcal{E}). \end{aligned}$$

Then, the condition for $(a_1a_2 - a_3 > 0)$ is fulfilled if $(\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)((q_1 + q_2)\delta + \mu_h)\mu_v(1 - \mathfrak{R}_0) < \mathcal{C}(\mathcal{D} - \mathcal{E})$.

So, based on the proof analysis above, it can be concluded that if $a_1, a_2, a_3 > 0$ and $a_1a_2 - a_0a_3 > 0$ are satisfied, then the coefficients of the characteristic equation will have a negative root or a negative real part. Therefore, it can be stated that the non-endemic equilibrium point $\mathcal{E}^0 = (S^0, E^0, I^0, H^0, R^0) = \left(\frac{\theta_h}{\mu_h}, 0, 0, 0, 0 \right)$ is locally asymptotically stable. ■

This can be interpreted that each actively infected individuals only able to infect/transmit the disease to less than one susceptible individual, which implies that, over time, the disease will naturally disappear (disease-free).

Theorem 2. If $\mathfrak{R}_0 > 1$ then endemic equilibrium point $\mathcal{E}^* = (S^*, E^*, I^*, H^*, R^*)$ is locally asymptotically stable and if $\mathfrak{R}_0 < 1$, then $\mathcal{E}^* = (S^*, E^*, I^*, H^*, R^*)$ is unstable [24].

Proof:

Assume $\mathcal{E}^* = (S^*, E^*, I^*, H^*, R^*)$, we can determine the eigen values of the Jacobian matrix at \mathcal{E}^* :

$$\det(J(\mathcal{E}^*) - YI) = 0$$

Then, the characteristic equation is obtained, as follows:

$$(Y + \mu_h)(a_0Y^4 + a_1Y^3 + a_2Y^2 + a_3Y + a_4) = 0$$

with,

$$a_0 = 1,$$

$$a_1 = \frac{A^*\beta}{\mu_v N + A^*} + ((q_1 + q_2)\delta + \omega + \rho_1 + \rho_2 + \eta + 4\mu_h),$$

$$\begin{aligned}
a_2 &= \frac{\beta}{(\mu_v N + A^*)^2} (\delta(q_1 + q_2)(N\mu_v(A^* - S^*) + A^{*2}) \\
&\quad + A^*(\omega + \rho_1 + \rho_2 + \eta + 3\mu_h)(\mu_v N + A^*) + 6\mu_h^2 \\
&\quad + ((q_1 + q_2)(\omega + \rho_1 + \rho_2 + \eta + 3\mu_h)\delta \\
&\quad + 3\mu_h(\omega + \rho_1 + \rho_2 + \eta) + \rho_2(\omega + \rho_1 + \eta) \\
&\quad + \eta(\omega + \rho_1)), \\
a_3 &= \frac{1}{(\mu_v N + A^*)^2} (\beta(A^*(\mu_v N + A^*)(((q_1 + q_2)\delta + 2\mu_h) \\
&\quad (\omega + \rho_1 + \rho_2 + \eta) + 2\mu_h\delta(q_1 + q_2) + 3\mu_h^2 \\
&\quad + (\rho_2 + \eta)(\omega + \rho_1) + \eta\rho_2) - ((q_1 + q_2) \\
&\quad (\omega + \eta + 2\mu_h) + q_1\rho_2 + q_2\rho_1)S^*\mu_v N\delta)) \\
&\quad + (3\mu_h^2 + 2\mu_h(\omega + \rho_1 + \eta) + \eta(\omega + \rho_1) \\
&\quad + (\omega + \rho_1 + \eta + 2\mu_h)\rho_2)(q_1 + q_2)\delta + 4\mu_h^3 \\
&\quad + 3\mu_h^2(\omega + \rho_1 + \rho_2 + \eta) + 2\mu_h(\rho_2 + \eta)(\omega + \rho_1) \\
&\quad + \eta\rho_2(\omega + \rho_1 + 2\mu_h), \\
a_4 &= \frac{1}{(\mu_v N + A^*)^2} (\beta(A^*(\mu_v N + A^*)(((q_1 + q_2)(\mu_h + \omega) \\
&\quad + \rho_1 q_2 + \rho_2 q_1)\delta\eta + (\mu_h + \rho_2)(\omega + \rho_1 + \mu_h) \\
&\quad (\eta + \mu_h + (q_1 + q_2)\delta)) - \mu_v N S^*(\eta + \mu_h) \\
&\quad (((q_1 + q_2)(\mu_h + \omega) + \rho_1 q_2 + \rho_2 q_1)\delta)) + (\mu_h + \rho_2) \\
&\quad (\omega + \rho_1 + \mu_h)(\mu_h + (q_1 + q_2)\delta)(\eta + \mu_h)).
\end{aligned}$$

with,

$$\begin{aligned}
A^* &= I^* + H^* \\
&= \frac{N\mu_v B(\eta + \mu_h)(\mathfrak{R}_0 - 1)}{\beta\eta(A\delta + (\mu_h + \rho_2)(\omega + \rho_1 + \mu_h)) + B(\eta + \mu_h + \beta)}.
\end{aligned}$$

According to the Routh-Hurwitz criterion, \mathcal{E}^* is locally asymptotically stable if all the roots of the characteristic polynomial are negative as shown below:

First, shown that $Y_1 + Y_2 + Y_3 + Y_4 = -\frac{b}{a} < 0$, with value of b and a as follow:

$$\begin{aligned}
a &= a_0 = 1 \\
b &= a_1 = \frac{A^*\beta}{\mu_v N + A^*} + ((q_1 + q_2)\delta + \omega + \rho_1 + \rho_2 \\
&\quad + \eta + 4\mu_h)
\end{aligned}$$

We found,

$$\begin{aligned}
Y_1 + Y_2 + Y_3 + Y_4 &= -\frac{b}{a} = \\
&= \frac{\beta B(\eta + \mu_h)(\mathfrak{R}_0 - 1)}{\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B((\eta + \mu_h)\mathfrak{R}_0 + \beta)} \\
&= \frac{\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B((\eta + \mu_h)\mathfrak{R}_0 + \beta)}{\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B((\eta + \mu_h)\mathfrak{R}_0 + \beta)} \\
&\quad - \frac{((q_1 + q_2)\delta + \omega + \rho_1 + \rho_2 + \eta + 4\mu_h)}{\beta\eta(A\delta + (\rho_2 + \mu_h)(\rho_1 + \mu_h + \omega)) + B((\eta + \mu_h)\mathfrak{R}_0 + \beta)}
\end{aligned}$$

Since the denominator is positive, $(Y_1 + Y_2 + Y_3 + Y_4)$ is negative, only when the numerator is negative. Hence, the resulting equation is as follows:

$$\begin{aligned}
&-(\beta B(\eta + \mu_h)(\mathfrak{R}_0 - 1) + (\beta\eta(A\delta + (\rho_2 + \mu_h) \\
&\quad (\rho_1 + \mu_h + \omega)) + B((\eta + \mu_h)\mathfrak{R}_0 + \beta)) \\
&\quad ((q_1 + q_2)\delta + \omega + \rho_1 + \rho_2 + \eta + 4\mu_h)) < 0.
\end{aligned}$$

Then, the condition above is fulfilled, if the value of $(\mathfrak{R}_0 - 1)$ must be positive, $(\mathfrak{R}_0 - 1) > 0$, $\mathfrak{R}_0 > 1$

Using the same method, we can obtain for,

Second, $Y_1 Y_2 + Y_1 Y_3 + Y_1 Y_4 + Y_2 Y_3 + Y_2 Y_4 + Y_3 Y_4 = \frac{c}{a} > 0$, with value of $c = a_2$ and $a = a_0$.

Third, $Y_1 Y_2 Y_3 + Y_1 Y_2 Y_4 + Y_2 Y_3 Y_4 = -\frac{d}{a} < 0$, with value of $d = a_3$ and $a = a_0$.

Fourth, $Y_1 Y_2 Y_3 Y_4 = \frac{e}{a} > 0$, with value of $e = a_4$ and $a = a_0$.

According to the analysis, a fourth-degree polynomial satisfies the requirements and $\mathfrak{R}_0 > 1$, with result showing that the roots of this polynomial have negative real parts. So, the endemic equilibrium points $\mathcal{E}^* = (S^*, E^*, I^*, H^*, R^*)$ is locally asymptotically stable, if $\mathfrak{R}_0 > 1$. ■

This can be interpreted that each actively infected individual is able to transmit the disease to multiple susceptible individuals, which implies that the disease will spread throughout the population.

D. Numerical Simulation

The numerical simulation of the dengue transmission model is performed using Maple software. The initial values of $S(0), E(0), I(0), H(0), R(0), V(0), W(0)$ and the parameter values used in the simulation are shown in Table 2. Several parameter values in Table 2 were adopted from Aldila et al. [21], which analyzed dengue case data from Jakarta Province in 2020. These values are considered relevant for this study because Jakarta shares comparable epidemiological characteristics with other Indonesian regions, particularly its tropical climate and the common vector *Aedes aegypti*. Moreover, these parameters are relatively stable over time, as they primarily represent biological and healthcare system factors that do not change substantially within a short annual period. Due to the unavailability of complete and updated dengue case data for several required variables in the target city of this study, these literature-based parameters were therefore employed to conduct numerical simulations.

The parameters labeled as "Estimated" in Table 2 were derived using demographic and clinical assumptions commonly applied in dengue epidemiological modeling. Specifically, the human recruitment rate (θ_h) and natural death rate (μ_h) were calculated based on the total human population (1670379) and an average human life expectancy of 65 years. The hospitalization rate of infected individuals (ω) was obtained as the inverse of the hospitalization period of 6 days, while the recovery rate of hospitalized individuals (ρ_2) was estimated as the inverse of an average recovery duration for hospitalized individuals of approximately 7 days.

Based on the substitution of the parameter values from Table 2 into the basic reproduction number formula, the number of susceptible individuals that can be infected by three infected individual was determined.

$$\begin{aligned}
\mathfrak{R}_0 &= \frac{\beta\delta((q_1 + q_2)(\mu_h + \omega) + q_1\rho_2 + q_2\rho_1)}{\mu_v(\delta(q_1 + q_2) + \mu_h)(\rho_1 + \mu_h + \omega)(\rho_2 + \mu_h)}, \\
\mathfrak{R}_0 &= 3.85011.
\end{aligned}$$

TABLE II: Parameter Values for The Dengue Model Numeric Simulation.

Par	Values	References
θ_h	$\frac{1670379}{65 \times 365} = 70.40586$	Estimated
μ_h	$\frac{1}{65 \times 365} = 0.00004$	Estimated
μ_v	0.00454	[21]
β	0.00200	[21]
δ	$\frac{1}{8} = 0.12500$	[21]
q_1	0.83100	[21]
q_2	0.16900	[21]
ω	$\frac{1}{6} = 0.16667$	Estimated
ρ_1	0.07143	[21]
ρ_2	$\frac{1}{7} = 0.14285$	Estimated
η	0.02778	[21]

Since the basic reproduction number is greater than one ($\mathcal{R}_0 = 3.85011 > 1$), this means that on average, one infected individual can infect more than one susceptible individual, which specifically means that, on average, one individual can infect about three susceptible individuals. This indicates that the dengue transmission model is locally asymptotically stable at the endemic equilibrium point $\mathcal{E}^* = (S^*, E^*, I^*, H^*, R^*)$, thus allowing the disease to persist and spread within the population over time without the implementation of effective control strategies. According to equation (4), the endemic equilibrium point was calculated as $\mathcal{E}^* = (S^*, E^*, I^*, H^*, R^*) = (1552560, 17893, 7805, 11749, 80370)$.

Next, by substituting the parameter values from Table 2. into equation (2), the dynamic model of dengue transmission is obtained as follows:

$$\begin{aligned}
 \frac{dS}{dt} &= (70.40586) + (0.02778)R \\
 &\quad - \frac{(0.002)S(I+H)}{(I+H) + (0.00454)N} - (0.00004)S, \\
 \frac{dE}{dt} &= \frac{(0.002)S(I+H)}{(I+H) + (0.00454)N} - (0.10388)E, \\
 \frac{dI}{dt} &= (0.10388)E - (0.23814)I, \\
 \frac{dH}{dt} &= (0.02113)E + (0.16667)I - (0.14289)H \\
 \frac{dR}{dt} &= (0.07143)I + (0.14285)H - (0.02782)R,
 \end{aligned} \tag{4}$$

Based on equation (4) and assuming the following initial condition values: $S(0) = 1665506, E(0) = 3581, I(0) = 500, H(0) = 404, R(0) = 388$, a numerical simulation of the SEIHR-VW model for dengue transmission was conducted to determine the dynamic behavior of each subpopulation over time using the Maple 2019 software package. These initial condition values were derived from dengue incidence data reported in Semarang City, Indonesia, in 2023 (obtained from the Semarang City Health Office).

Figure 2 shows that the number of susceptible individuals

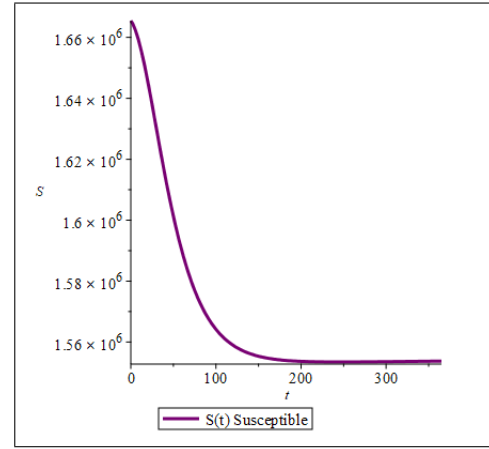


Fig. 2: Numerical Simulation of The Dengue Transmission Model for system (2): Subpopulation of Susceptible Human

was approximately 1665505 at $t=0$, but it decreased sharply during the first 100 days, reflecting the rapid spread of infection in the early phase of the disease. After this period, the rate of decline slowed considerably, and the curve gradually approached a steady state. From around day 200 onward, $S(t)$ stabilizes near $S^* = 1552560$. This indicates that although a large portion of the subpopulation remains susceptible, the system reaches a dynamic balance in which the inflow of individuals entering the susceptible subpopulation is balanced with the outflow of those leaving the subpopulation.

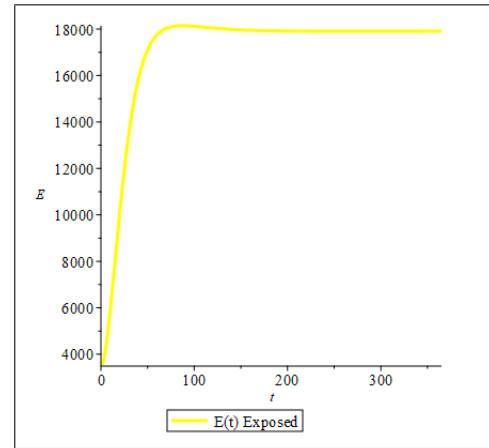


Fig. 3: Numerical Simulation of The Dengue Transmission Model for system (2): Subpopulation of Exposed Human

Figure 3 illustrates that, initially, at $t=0$, the exposed population consists of 3581 individuals. During the early phase, there is a rapid increase in the number of exposed individuals, reflecting the swift transmission of the disease within the susceptible subpopulation. Around day 100, the growth rate slows significantly as the number of exposed individuals approaches the endemic equilibrium point of 17893, indicating stabilization in disease spread. After this period, the curve levels off, showing minor fluctuations around the equilibrium, which suggests that the disease has reached a steady state in which the number of people entering and leaving the exposed

subpopulation is balanced.

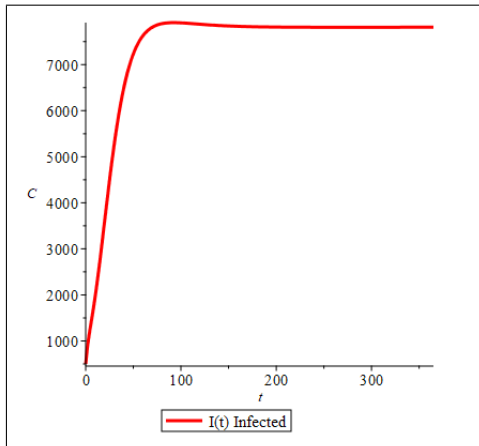


Fig. 4: Numerical Simulation of The Dengue Transmission Model for system (2): Subpopulation of Infected Human

Figure 4 shows a rapid increase in the number of infected individuals, rising from 500 to nearly 7000 by day 50 due to a high transmission rate and a large susceptible population. Between days 50 and 100, the growth slows as new infections decline and more individuals move to the hospitalized or recovered subpopulations. After day 150, the curve flattens, indicating the system approaches an endemic equilibrium, with the number of infected individuals stabilizing at around 7805.

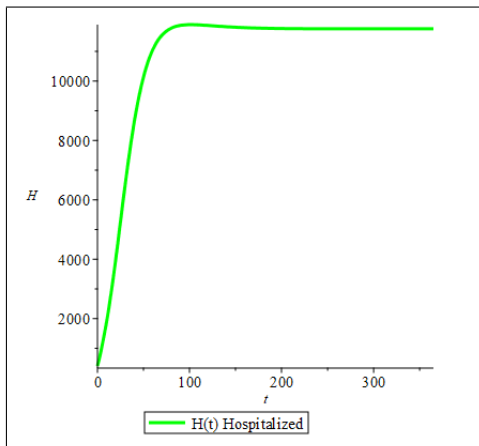


Fig. 5: Numerical Simulation of The Dengue Transmission Model for system (2): Subpopulation of Hospitalized Human

Figure 5 shows that at the beginning, when there are 404 hospitalized individuals, the number increases rapidly as more infections progress to severe symptoms. After approximately day 50, the growth slows down, and the curve begins to flatten. This indicates that the spread of the disease is stabilizing, either because fewer people remain at risk or due to natural limitations or interventions. Eventually, the curve flattens near 11749 hospitalized individuals, indicating that the system has reached a steady state. At this point, the number of people entering and leaving the hospital is close to equal. This

steady show that the disease persists within the population and continues to place sustained pressure on healthcare systems.

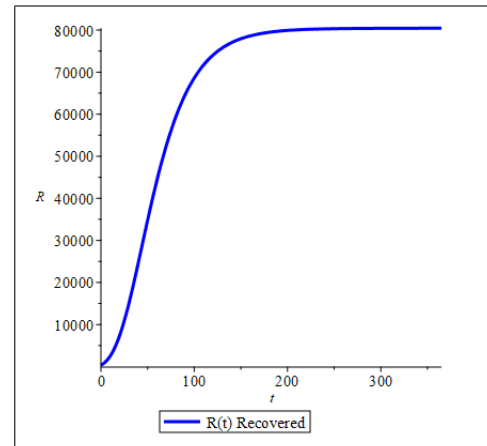


Fig. 6: Numerical Simulation of The Dengue Transmission Model for system (2): Subpopulation of Recovered Human

Figure 6 shows that initially, the number of recovered individuals is very low at 388. In the early stage, $R(t)$ increases rapidly due to the rising number of individuals who recover from infection. This growth continues rapidly until around $t=100$ days, after which the curve gradually slows as it approaches the endemic equilibrium point at 80370, representing the long-term number of recovered individuals in the population. From the graph, it can be observed that the system reaches this equilibrium approximately at $t=200$, after which $R(t)$ stabilizes without significant further increase. This indicates that recovery has saturated and the disease dynamics have settled into a stable endemic state.

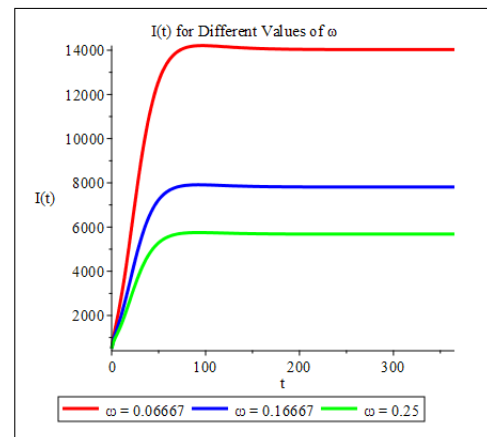


Fig. 7: The effect of variations in parameter ω on the infected individuals

Furthermore, a simulation was carried out to examine the effect of varying the parameter value ω on the number of infected individuals (I) and hospitalized individuals (H), as illustrated in Figures 7 and 8. In addition, a numerical simulation was performed to observe the impact of changes in the parameter ρ_2 on the number of recovered individuals (R), as shown in Figure 9. A more detailed explanation is

provided in the following section. First, the simulation was run for $\omega = 0.06667$, 0.16667 , and 0.25 . The results in Figure 7 show that as ω increases, the number of infected individuals decreases. By the 14th day, the number of infected individuals reaches 3818 for $\omega = 0.06667$, decreases to 2589 for $\omega = 0.16667$, and further drops to 2012 for $\omega = 0.25$.

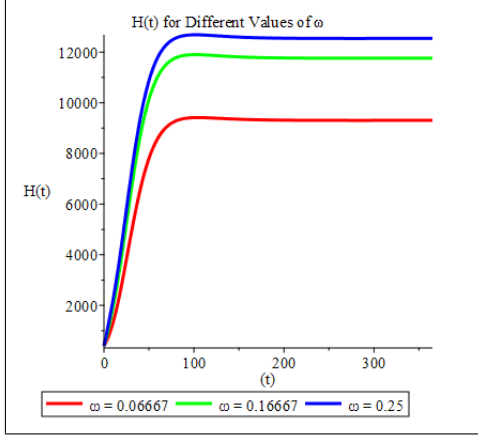


Fig. 8: The effect of variations in parameter ω on the hospitalized individuals

Second, Figure 8 presents the numerical simulation of hospitalized individuals by varying the parameter value ω , while keeping other parameters constant. The simulation was run for the same three values of ω . It can be observed that as ω increases, the number of hospitalized individuals also increases. By the 14th day, when $\omega = 0.06667$, the number of hospitalized individuals reaches 1864. For $\omega = 0.16667$, the number rises to 2684, and for $\omega = 0.25$, it further increases to 3047. Finally, Figure 9 shows the numerical simulation

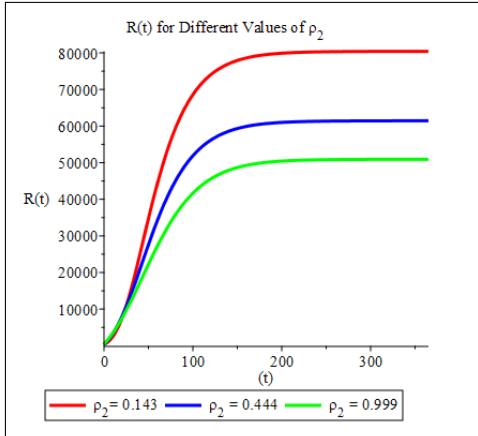


Fig. 9: The effect of variations in parameter ρ_2 on the recovered individuals

of recovered individuals by varying the parameter value ρ_2 , while keeping other parameters constant. The simulation was conducted for three values of $\rho_2 = 0.143$, 0.444 , and 0.999 . It can be observed that as ρ_2 increases, the number of recovered individuals decreases. By the 14th day, when $\rho_2 = 0.143$, the number of recovered individuals reaches 2682. For $\rho_2 = 0.444$,

the number decreases to 963, and for $\rho_2 = 0.999$, it drops further to 393.

E. Sensitivity Analysis

Sensitivity analysis was conducted to identify the parameters that significantly influence the value of \mathfrak{R}_0 in the spread of dengue disease. The first step is to determine the sensitivity index of each parameter with respect to \mathfrak{R}_0 . The sensitivity index is obtained by finding the partial derivative of \mathfrak{R}_0 to the parameter P .

$$C_P^{\mathfrak{R}_0} = \frac{\partial \mathfrak{R}_0}{\partial P} \times \frac{P}{\mathfrak{R}_0}$$

Next, substitute the parameter values of μ_h , μ_v , β , δ , q_1 , q_2 , ω , ρ_1 , ρ_2 to their respective sensitivity index analysis equation, an index of sensitivity values to \mathfrak{R}_0 is presented in the following table:

TABLE III: Index of Parameter Sensitivity.

Par	Sensitivity Index
μ_h	-0.00063
μ_v	-1
β	1
δ	0.00031
q_1	0.03398
q_2	-0.03366
ω	-0.13962
ρ_1	-0.25937
ρ_2	-0.60068

The sensitivity analysis in Table III shows that the mosquito mortality rate (μ_v) and the transmission rate (β) are the most influential parameters affecting the basic reproduction number (\mathfrak{R}_0). A 10% increase in μ_v reduces \mathfrak{R}_0 by 10%, while a 10% increase in β raises \mathfrak{R}_0 by 10%. The recovery rates of hospitalized individuals (ρ_2) and non-hospitalized individuals (ρ_1) also have a strong negative influence, highlighting their role in reducing transmission. The hospitalization rate (ω) has a moderate effect, whereas the progression parameters (q_1 , q_2) have only a minor impact. In contrast, the human mortality rate (μ_h) and the incubation parameter (δ) exert minimal influence on \mathfrak{R}_0 .

IV. CONCLUSIONS

The host-vector SAEIHR-VW model was modified into the SEIHR-VW model for dengue transmission by removing the subpopulation of susceptible humans aware of dengue infection (A) and excluding the parameters for case detection and hospital capacity. The resulting model includes seven subpopulations: susceptible humans, exposed humans, infected humans, hospitalized humans, recovered humans, susceptible mosquitoes, and infected mosquitoes. Both the disease-free equilibrium point (DFE) and the endemic equilibrium point (EE) were obtained in this study. Based on local stability analysis using the basic reproduction number (\mathfrak{R}_0) and the Routh–Hurwitz criterion, it was found that $\mathfrak{R}_0 > 1$, indicating that the model is locally asymptotically stable at the endemic

equilibrium point. Furthermore, numerical simulations showed that $\mathcal{R}_0 = 3.85011 > 1$, confirming that dengue transmission in the model remains locally asymptotically stable at the endemic equilibrium point. Sensitivity analysis further revealed that the mosquito mortality rate (μ_v) and the infection rate (β) are the most influential parameters.

For future research, this study can be extended by exploring longer time horizons to capture long-term transmission dynamics, integrating richer and more recent datasets to improve parameter accuracy, and incorporating additional epidemiological or environmental factors such as seasonal variation, climate influence, healthcare interventions, as well as the application of optimal control strategies. These extensions would enable the model to better reflect real-world conditions and provide stronger insights for dengue control strategies.

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