

SEIR-SEI DENGUE TRANSMISSION DYNAMICS IN KUPANG CITY: STABILITY AND SENSITIVITY ANALYSIS

Dhini Juniarti Putri^{1*}, Ariyanto², Mira Wadu³, Maria Lobo⁴

^{1,2,3,4} Department of Mathematics, Nusa Cendana University, Indonesia

Email: ^{1*}dhinijuniarti0906@gmail.com, ²aryanto@staf.undana.ac.id,

³mira_wadu@staf.undana.ac.id, ⁴maria_lobo@staf.undana.ac.id

*Corresponding author

Received: 13 May 2026, revised: 16 August 2026, accepted: 20 August 2026

Abstract

Dengue fever continues to pose a persistent public health challenge in Kupang City, as case counts have shown inconsistent trends from year to year. An SEIR-SEI mathematical modeling framework is applied in this study to examine the dynamics of dengue spread and to determine intervention strategies that may effectively reduce transmission. The objectives are to determine equilibrium points and the basic reproduction number (R_0), analyze the local stability of the disease-free equilibrium, and evaluate parameter sensitivity. The value of R_0 was estimated using monthly dengue case data in Kupang City from 2020 to 2024 under assumption of exponential growth in the infected population. Rather than relying on full dynamic parameter fitting or transmission rate values imported from other locations, this study adopts a simpler approach by fitting only the take-off rate from the early growth phase of a single recent outbreak, providing a locally grounded yet computationally straightforward estimate of R_0 . The analysis produced an estimated value of $R_0 = 1,64 > 1$, indicating the potential persistence and spread of dengue transmission within the population. Stability analysis shows that the disease-free equilibrium is locally unstable when $R_0 > 1$. Sensitivity analysis reveals that natural mosquito mortality (μ_v) is the most influential parameter affecting R_0 . These findings suggest that vector control measures, such as insecticide use, larviciding, and elimination of mosquito breeding sites, are likely to have the greatest impact on reducing R_0 , and therefore warrant priority consideration among dengue control strategies in Kupang City.

Keywords: SEIR-SEI model, dengue transmission, basic reproduction number, sensitivity analysis, Kupang City

1. INTRODUCTION AND PRELIMINARIES

Dengue fever is a vector-borne illness caused by the dengue virus, with its primary mode of transmission being the bite of *Aedes aegypti* and *Aedes albopictus* mosquitoes [16]. Over the two past decades, global dengue cases have increased dramatically, rising from 505.430 reported cases



in 2000 to 14.6 million cases in 2024, making 2024 the highest recorded year in dengue history [16]. In Indonesia, dengue was first reported in Surabaya in 1968, and the national incidence rate has continued to fluctuate, reaching 51,12 cases per 100.000 population in 2022 [8].

East Nusa Tenggara Province has consistently reported dengue cases each year, with incidence rates ranging from 59 to 78,1 cases per 100.000 population during the 2019-2020 period [8]. As the capital of the province, Kupang City also experiences annual fluctuations in dengue cases, decreasing from 808 cases in 2020 to 271 cases in 2024. These unstable transmission patterns indicate the need for a deeper understanding of dengue transmission dynamics in this region.

The use of mathematical models has emerged as a systematic and powerful tool for understanding how infectious diseases spread through populations [9][1]. In the SEIR-SEI framework, the human host population is partitioned into four disease states: susceptible, exposed, infected, and recovered, whereas mosquitoes as vectors are represented by three compartments: susceptible, exposed, and infected. This model is suitable for representing dengue transmission because it incorporates incubation periods in both humans and vectors, as well as indirect transmission mechanisms through mosquito bites. Several previous studies have examined the SEIR-SEI model for dengue fever in general [12][7][2][11]. Most of these adopted the transmission rate (β) from other locations or the general literature, while one applied to Kupang City itself estimated parameters through full dynamic fitting incorporating seasonal transmission patterns for optimal vaccination control. In contrast, this study adopts a substantially simpler approach: β is derived by fitting only the take-off rate (r) from the early growth phase of a single recent outbreak episode identified within the 2020-2024 case data, then substituting it into the model's equilibrium relations to obtain a locally grounded estimate of R_0 .

The main contribution of this study is twofold: (i) it derives the equilibrium points, basic reproduction number, and local stability conditions of the SEIR-SEI model analytically, and (ii) rather than relying on full dynamic parameter fitting or transmission rate values imported from other locations, it grounds the estimate of R_0 in a take-off rate fitted directly from a single recent outbreak episode in Kupang City's own case data, offering a locally grounded yet computationally straightforward basis for dengue control prioritization.

Through sensitivity analysis of model parameters, it becomes possible to identify which factors exert the greatest influence on basic reproduction number (R_0), which in turn guides decision-makers in prioritizing the most impactful control measures. This study aims to develop a SEIR-SEI model for dengue transmission in Kupang City, determine equilibrium points and the basic reproduction number, analyze local stability, and perform local sensitivity analysis.

2. MATERIAL AND METHODS

2.1 Model Assumptions

The model was constructed under the following assumptions:

1. Both the human host and mosquito vector populations are assumed to maintain a constant total size over the entire modeling period.
2. Transmission occurs only through mosquito-human contact,
3. Exposed humans and vectors experiences incubation periods,
4. Mortality occurs only due to natural causes,
5. Recovered humans acquire permanent immunity.

2.2 SEIR-SEI Model Formulation

Based on these assumptions, the schematic representation of dengue transmission is presented in Figure 2.1. The human population is categorized into four compartments: individuals who are vulnerable to infection (*susceptible*/ S_h), those who have been exposed to the virus but have not

yet shown symptoms (*exposed*/ E_h), those who are actively experiencing the disease (*infected*/ I_h), and those who have fully recovered (*recovered*/ R_h). Meanwhile, the mosquito population as the disease vector is structured into three compartments, namely mosquitoes that remain vulnerable (*susceptible*/ S_v), those that have been exposed the pathogen (*exposed*/ E_v), and those that are actively carrying and transmitting the infection (*infected*/ I_v).

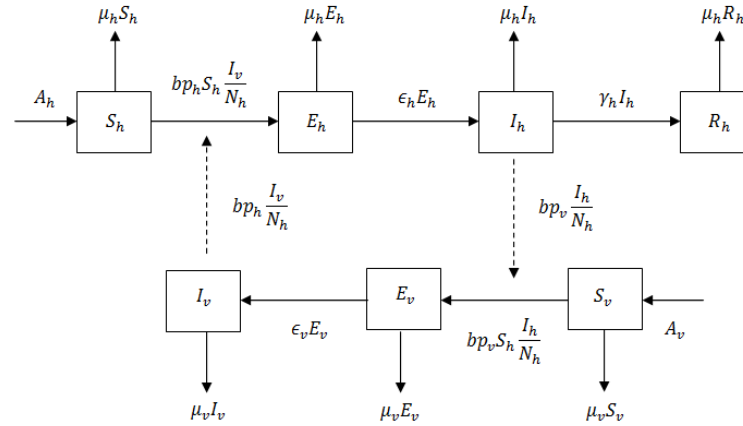


Figure 2.1. Schematic Diagram of Dengue Transmission

The system is represented by the following differential equations:

$$\frac{dS_h}{dt} = A_h - bp_h S_h \frac{I_v}{N_h} - \mu_h S_h \quad (2.1)$$

$$\frac{dE_h}{dt} = bp_h S_h \frac{I_v}{N_h} - \epsilon_h E_h - \mu_h E_h \quad (2.2)$$

$$\frac{dI_h}{dt} = \epsilon_h E_h - \gamma_h I_h - \mu_h I_h \quad (2.3)$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h \quad (2.4)$$

$$\frac{dS_v}{dt} = A_v - bp_v S_v \frac{I_h}{N_h} - \mu_v S_v \quad (2.5)$$

$$\frac{dE_v}{dt} = bp_v S_v \frac{I_h}{N_h} - \epsilon_v E_v - \mu_v E_v \quad (2.6)$$

$$\frac{dI_v}{dt} = \epsilon_v E_v - \mu_v I_v \quad (2.7)$$

The model parameters are described in Table 2.1.

Table 2.1. Model Parameters

Parameter	Description
b	Biting rate
p_h	Probability of dengue transmission to humans
p_v	Probability of dengue transmission to mosquitoes
$\beta_h = bp_h$	Human transmission rate
$\beta_v = bp_v$	Mosquito transmission rate
A_h	Human recruitment rate
A_v	Mosquito recruitment rate
N_h	Total human population
μ_h	Human natural mortality
μ_v	Mosquito natural mortality

ϵ_h	Human incubation rate
ϵ_v	Mosquito incubation rate
γ_h	Human recovery rate
$m = \frac{N_v}{N_h}$	Mosquito-human population ratio

Several parameters values were obtained from demographic data and relevant literature

Table 2.2. Parameter Values

Parameter	Value (per month)	Source
N_h	474.801	[3]
μ_h	0,00116	Calculations
A_h	550,75	Calculations
N_v	949.602	Assumptions
μ_v	1,5	[15]
A_v	118.700	Calculations
ϵ_h	6	[4]
ϵ_v	4,5	[4]
γ_h	4,29	[16]

2.3 Equilibrium Points

The model has two equilibrium points obtained by setting the right-hand side of Equations (2.1) – (2.7) to zero under specific conditions. The disease-free equilibrium point is determined by the conditions $E_h = 0, I_h = 0, E_v = 0$ and $I_v = 0$, yielding:

$$E_0 = (S_h^0, E_h^0, I_h^0, R_h^0, S_v^0, E_v^0, I_v^0) = \left(\frac{A_h}{\mu_h}, 0, 0, 0, \frac{A_v}{\mu_v}, 0, 0 \right) = (N_h, 0, 0, 0, N_v, 0, 0) \quad (2.8)$$

The endemic equilibrium point is obtained under the conditions $E_h \neq 0, I_h \neq 0, E_v \neq 0$, and $I_v \neq 0$, given by:

$$E^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, E_v^*, I_v^*)$$

With:

$$\begin{aligned} S_h^* &= \frac{N_h}{bp_h\mu_v(C-m) + N_h(bp_vK_h\mu_h + \mu_vbp_h)} \\ E_h^* &= \frac{bp_h\mu_h\mu_v(C-m)}{(\epsilon_h + \mu_h)(bp_h\mu_v(C-m)(bp_vK_h\mu_h + \mu_vbp_h) + N_h(bp_vK_h\mu_h + \mu_vbp_h)^2)} \\ I_h^* &= \frac{\mu_h\mu_vN_h(C-1)}{bp_hK_v\mu_v m + \mu_hbp_v} \\ R_h^* &= \frac{\gamma_h\mu_vN_h(C-1)}{bp_hK_v\mu_v m + \mu_hbp_v} \\ S_v^* &= \frac{bp_v\mu_h(C-1) + (bp_hK_v\mu_v m + \mu_hbp_v)}{bp_v m\mu_h\mu_v(C-1)} \\ E_v^* &= \frac{(\epsilon_v + \mu_v)(bp_v\mu_h(C-1)(bp_hK_v\mu_v m + \mu_hbp_v) + (bp_hK_v\mu_v m + \mu_hbp_v)^2)}{\mu_h\mu_v(C-m)} \\ I_v^* &= \frac{bp_vK_h\mu_h + \mu_vbp_h}{bp_vK_h\mu_h + \mu_vbp_h} \end{aligned}$$

$$\text{where } m = \frac{N_v}{N_h}, C = \frac{b^2 p_h p_v \epsilon_h \epsilon_v N_v}{(\epsilon_v + \mu_v)(\epsilon_h + \mu_h)(\gamma_h + \mu_h)\mu_v N_h}, K_h = \frac{bp_h \epsilon_h}{(\epsilon_h + \mu_h)(\gamma_h + \mu_h)} \text{ and } K_v = \frac{bp_v \epsilon_v}{(\epsilon_v + \mu_v)\mu_v}.$$

The endemic equilibrium exists only when $C > 1$, indicating that dengue transmission can persist within the population under this condition.

2.4 Basic Reproduction Number

The basic reproduction number R_0 quantifies how many new infection cases, on average, a single infected individual will generate when introduced into a population where all individuals remain susceptible [5]. In this study, R_0 was determined using the *Next Generation Matrix* method by focusing on the exposed and infected compartments represented in Equations (2.2), (2.3), (2.6), and (2.7). Both the transmission matrix F and transition matrix V were assessed at the disease-free equilibrium point E_0 in Equation (2.8).

$$F = \begin{bmatrix} 0 & 0 & 0 & bp_h \\ 0 & 0 & 0 & 0 \\ 0 & \frac{bp_v N_v}{N_h} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad V = \begin{bmatrix} \epsilon_h + \mu_h & 0 & 0 & 0 \\ -\epsilon_h & \gamma_h + \mu_h & 0 & 0 \\ 0 & 0 & \epsilon_v + \mu_v & 0 \\ 0 & 0 & -\epsilon_v & \mu_v \end{bmatrix}$$

The Next Generation Matrix was obtained as:

$$NGM = FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{bp_h \epsilon_v}{(\epsilon_v + \mu_v) \mu_v} & \frac{bp_h}{\mu_v} \\ 0 & 0 & 0 & 0 \\ \frac{bp_v N_v \epsilon_h}{N_h (\epsilon_h + \mu_h) (\gamma_h + \mu_h)} & \frac{bp_v N_v}{N_h (\gamma_h + \mu_h)} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad (2.9)$$

So the eigenvalues obtained from NGM in Equation (2.9) are $\lambda_1 = 0, \lambda_2 = \sqrt{\frac{bp_h \epsilon_v}{(\epsilon_v + \mu_v) \mu_v} \frac{bp_v N_v \epsilon_h}{N_h (\epsilon_h + \mu_h) (\gamma_h + \mu_h)}}$, and $\lambda_3 = -\sqrt{\frac{bp_h \epsilon_v}{(\epsilon_v + \mu_v) \mu_v} \frac{bp_v N_v \epsilon_h}{N_h (\epsilon_h + \mu_h) (\gamma_h + \mu_h)}}$. Then the R_0 value is determined from the spectral radius of eigenvalues, yielding:

$$R_0 = \sqrt{\frac{bp_h \epsilon_v}{(\epsilon_v + \mu_v) \mu_v} \frac{bp_v N_v \epsilon_h}{N_h (\epsilon_h + \mu_h) (\gamma_h + \mu_h)}}, \quad \text{where } m = \frac{N_v}{N_h} \quad (2.10)$$

To estimate R_0 based on data, a reconstruction was performed using the concept of initial growth of the infected populations. It was assumed that the number of humans and mosquitoes infected with dengue virus grew exponentially at the same growth rate, namely:

$$\begin{aligned} E_h(t) &= E_{h0} e^{rt} \Rightarrow \frac{dE_h}{dt} = r E_{h0} e^{rt} \\ I_h(t) &= I_{h0} e^{rt} \Rightarrow \frac{dI_h}{dt} = r I_{h0} e^{rt} \\ E_v(t) &= E_{v0} e^{rt} \Rightarrow \frac{dE_v}{dt} = r E_{v0} e^{rt} \\ I_v(t) &= I_{v0} e^{rt} \Rightarrow \frac{dI_v}{dt} = r I_{v0} e^{rt} \end{aligned} \quad (2.11)$$

Substituting Equation (2.11) into Equations (2.2), (2.3), (2.6), and (2.7) produced:

$$\begin{aligned} \frac{I_{v0}}{E_{h0}} &= \frac{r + \epsilon_h + \mu_h}{bp_h} \\ \frac{E_{h0}}{I_{h0}} &= \frac{r + \gamma_h + \mu_h}{\epsilon_h} \end{aligned} \quad (2.12)$$

$$\frac{I_{h0}}{E_{v0}} = \frac{r + \epsilon_v + \mu_v}{bp_v m}$$

$$\frac{E_{v0}}{I_{v0}} = \frac{r + \mu_v}{\epsilon_v}$$

Thus, the following relationship was obtained:

$$\frac{I_{v0}}{E_{h0}} \frac{E_{h0}}{I_{h0}} \frac{I_{h0}}{E_{v0}} \frac{E_{v0}}{I_{v0}} = 1 \quad (2.13)$$

Substituting Equation (2.12) into Equation (2.13) produced:

$$b^2 p_h p_v m = \frac{(r + \epsilon_h + \mu_h)(r + \gamma_h + \mu_h)(r + \epsilon_v + \mu_v)(r + \mu_v)}{\epsilon_h \epsilon_v} \quad (2.14)$$

Or equivalent to:

$$\mathcal{R}_0 = \sqrt{\left(1 + \frac{r}{\epsilon_h + \mu_h}\right) \left(1 + \frac{r}{\gamma_h + \mu_h}\right) \left(1 + \frac{r}{\epsilon_v + \mu_v}\right) \left(1 + \frac{r}{\mu_v}\right)} \quad (2.15)$$

The R_0 value in Equation (2.15) is used to estimate R_0 in Equation (2.10).

The value of R_0 determines the epidemiological outcome: when $R_0 < 1$, each generation of infections produces fewer cases than the previous one, so the disease eventually dies out; conversely, when $R_0 > 1$, the infection continues to expand across successive generations, suggesting that endemic transmission is possible [5].

Next, to find the parameter values of the human transmission rate (β_h) and mosquito transmission rate (β_v), Equation (2.14) will be used, yielding:

$$\beta_h \beta_v = \frac{(r + \epsilon_h + \mu_h)(r + \gamma_h + \mu_h)(r + \epsilon_v + \mu_v)(r + \mu_v)}{\epsilon_h \epsilon_v m} \quad (2.16)$$

Since Equation (2.16) contains two unknown transmission parameters, the simplifying assumption $\beta_h = \beta_v = \beta$ was introduced to enable unique parameter estimation. Thus, the transmission parameter is obtained as:

$$\beta = \sqrt{\frac{(r + \epsilon_h + \mu_h)(r + \gamma_h + \mu_h)(r + \epsilon_v + \mu_v)(r + \mu_v)}{\epsilon_h \epsilon_v m}} \quad (2.17)$$

2.5 Local Stability Analysis

The local stability of the disease-free equilibrium was analyzed using the Jacobian matrix [9][6] of Equations (2.1) – (2.7), given by:

$$J = \begin{bmatrix} -bp_h \frac{I_v}{N_h} - \mu_h & 0 & 0 & 0 & 0 & 0 & -bp_h \frac{S_h}{N_h} \\ bp_h \frac{I_v}{N_h} & -(\epsilon_h + \mu_h) & 0 & 0 & 0 & 0 & bp_h \frac{S_h}{N_h} \\ 0 & \epsilon_h & -(\gamma_h + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & -bp_v \frac{S_v}{N_h} & 0 & -bp_v \frac{I_h}{N_h} - \mu_v & 0 & 0 \\ 0 & 0 & bp_v \frac{S_v}{N_h} & 0 & bp_v \frac{I_h}{N_h} & -(\epsilon_v + \mu_v) & 0 \\ 0 & 0 & 0 & 0 & 0 & \epsilon_v & -\mu_v \end{bmatrix} \quad (2.18)$$

Equation (2.18) is evaluated at the disease-free equilibrium point (E_0) Equation (2.8):

$$J(E_0) = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & -bp_h \\ 0 & -(\epsilon_h + \mu_h) & 0 & 0 & 0 & 0 & bp_h \\ 0 & \epsilon_h & -(\gamma_h + \mu_h) & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma_h & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & -\frac{bp_v N_v}{N_h} & 0 & -\mu_v & 0 & 0 \\ 0 & 0 & \frac{bp_v N_v}{N_h} & 0 & 0 & -(\epsilon_v + \mu_v) & 0 \\ 0 & 0 & 0 & 0 & 0 & \epsilon_v & -\mu_v \end{bmatrix} \quad (2.19)$$

Cofactor expansion of Equation (2.18) yields three immediate eigenvalues: $\lambda_1 = \lambda_2 = -\mu_h$ and $\lambda_3 = -\mu_v$. The remaining four eigenvalues are determined by the fourth-order characteristic equation:

$$(\lambda + A)(\lambda + B)(\lambda + C)(\lambda + D) - E = 0 \quad (2.20)$$

Where $A = \epsilon_h + \mu_h$, $B = \gamma_h + \mu_h$, $C = \epsilon_v + \mu_v$, $D = \mu_v$ and $E = b^2 p_h p_v \epsilon_h \epsilon_v m$. Expanding Equation (2.20) gives : $\lambda^4 + a_1 \lambda^3 + a_2 \lambda^2 + a_3 \lambda + a_4$.

This stability condition was then examined using the Routh-Hurwitz criterion, which requires all coefficients to be positive and the Hurwitz determinants to satisfy the positivity conditions for all eigenvalues to have negative real parts [9][14].

Since $a_1, a_2, a_3 > 0$, the stability condition reduces to: $a_4 > 0$ which equivalent to:

$$ABCD(1 - R_0^2) > 0 \Leftrightarrow R_0 < 1.$$

Thus, disease free equilibrium is locally asymptotically stable if $R_0 < 1$.

2.6 Local Sensitivity Analysis

To assess how individual parameter changes affect the value of R_0 , a local sensitivity was conducted. Following [7], the normalized forward sensitivity index, which measures the proportional change in R_0 relative to a proportional change in parameter p , is expressed as:

$$C_p^{R_0} = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0} \quad (2.21)$$

3. RESULTS AND DISCUSSION

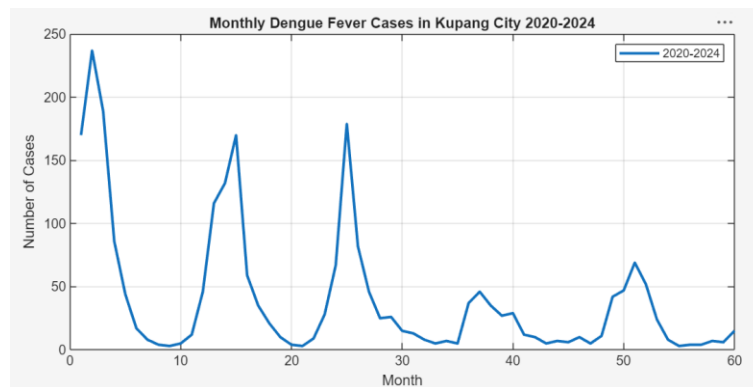


Figure 3.1. Monthly Dengue Fever Cases in Kupang City 2020-2024

Data on individuals infected with dengue fever in Kupang City in 2020-2024 is presented in Figure 3.1. From Figure 3.1, the period was selected as the epidemic take-off phase through an exhaustive comparison across all possible windows. Several windows exhibited excellent exponential fit ($R^2 > 0.95$), but with negative growth coefficient ($b < 0$), reflecting the declining tail of the previous outbreak rather than a genuine take-off. Among the remaining candidates with a positive growth trend, several others were also statistically significant ($p < 0.05$). However, only the selected period satisfied all pre-defined criteria simultaneously, achieving the highest goodness-of-fit among all pre-peak candidate windows ($R^2 = 0.9978$; $RMSE = 1.68$ cases) and a statistically significant fit ($p = 0.0011$), while also achieving the lowest prediction error ($MAPE = 14.26\%$). Assuming the number of infected individuals grows exponentially, the final model was obtained by nonlinear regression of the selected period using MATLAB software, yielding:

$$I_h(t) = 1.5975e^{0.9353t} \quad (3.1)$$

The corresponding exponential curve is presented in Figure 3.2. The analysis results show that R^2 value is 99.78% and the take-off rate is 0.9353. Substituting take-off rate value into equation (2.15) yields $R_0 = 1.64$. This indicates that one infected person can produce 1 to 2 new cases. Since $R_0 = 1.64 > 1$, E_0 is unstable, meaning the disease will continue to spread in the population.

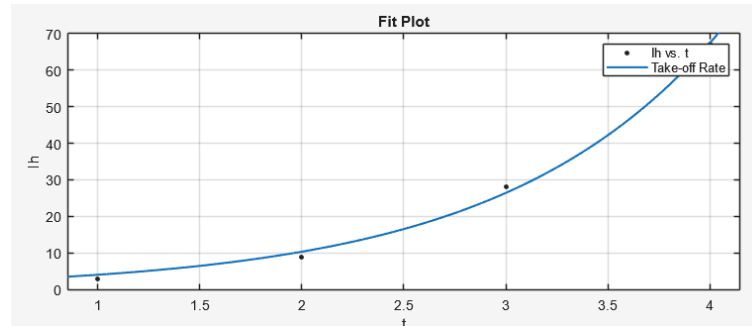


Figure 3.2. Exponential Regression Results

The take-off rate value is also substituted in Equation (2.17). Using the parameter values in Table 2.2 together with estimated take-off rate $r = 0.9353$, this yields $\beta_h \beta_v \approx 11,609$. As no entomological data are available to separately distinguish the transmission probability from mosquito to human and vice versa, a symmetric treatment $\beta_h = \beta_v = \beta$ is adopted, giving $\beta = \sqrt{\beta_h \beta_v} = \sqrt{11,609} \approx 3,407$.

Thus, all parameter values required for parameter sensitivity analysis have been obtained. The normalized sensitivity index R_0 for each parameter is calculated analytically using Equation (2.21) to obtain the following results:

Table 3.1. Sensitivity Analysis With 5% Variation

Parameter	Equation	Value	R_0 if $p + 5\%$	R_0 if $p - 5\%$
β_h	1	0,5	1,681	1,599
	2			
β_v	1	0,5	1,681	1,599
	2			
m	1	0,5	1,681	1,599
	2			

ϵ_h	$\frac{1}{2}\left(1 - \frac{\epsilon_h}{\epsilon_h + \mu_h}\right)$	0,000113	1,640009	1,639990
ϵ_v	$\frac{1}{2}\left(1 - \frac{\epsilon_v}{\epsilon_v + \mu_v}\right)$	0,125	1,65025	1,62975
γ_h	$-\frac{1}{2}\left(\frac{\gamma_h}{\gamma_h + \mu_h}\right)$	-0,49986	1,5990	1,6809
μ_v	$-\frac{1}{2}\left(1 + \frac{\mu_v}{\epsilon_v + \mu_v}\right)$	-0,625	1,58875	1,69125

The calculation results in Table 3.1 show that natural mosquito mortality (μ_v) has the largest absolute negative sensitivity index ($-0,625$), followed by the transmission rate (β_h and β_v) and the mosquito-to-human population ratio (m) with an index of $+0,5$, the human recovery rate (γ_h) with an index of $-0,5$, the mosquito incubation rate (ϵ_v) with an index of $+0,125$, and the human incubation rate (ϵ_h) with an index of $0,000113$.

Natural mosquito mortality (μ_v) is the most influential parameter. This reflects that the faster mosquitoes die, the lower the chance of virus transmission to humans. The transmission rate (β) and the mosquito-to-human ratio (m) also have a significant effect. The human incubation rate (ϵ_h) has almost no effect due to its very small value. Since mosquito-related parameters collectively exert the strongest influence on R_0 , interventions targeting the vector population including insecticide application, larvicidal treatments, elimination of mosquito breeding habitats [13], and the deployment of Wolbachia-based biocontrol [10] constitute the most promising approaches to reducing dengue burden.

4. CONCLUSIONS

In this work, a SEIR-SEI compartmental model was constructed to characterize dengue fever transmission in Kupang City by capturing the coupled dynamics between human hosts and mosquito vectors. This model has two equilibrium points: a disease-free equilibrium point (E_0) and an equilibrium point (E^*). The R_0 value of $1,64 > 1$ obtained from the Next Generation Matrix method and estimates based on monthly case data for the 2020-2024 period indicates that the disease has the potential to spread in the population. Local stability analysis using the Routh-Hurwitz criterion proves that E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$. Local sensitivity analysis showed that mosquito-related parameters dominated the influence on R_0 , so control strategies focused on mosquito control, including insecticides, larvicides, breeding-site elimination, and Wolbachia technology, were the most effective strategies for suppressing the spread of dengue fever in Kupang City. Further research is recommended to integrate seasonal factors so that the model can represent long-term endemic patterns.

REFERENCES

- [1] Aguiar, M., Anam, V., Blyuss, K.B., Estadilla, C.D.S., & Guerrero, B.V., 2020. Mathematical Models for Dengue Fever Epidemiology: A 10-year Systematic Review. *Psychiatry Research*, Vol. 14(4), 293.
- [2] Bhuju, G., Phaijoo, G. R., & Gurung, D. B., 2022. Sensitivity and Bifurcation Analysis of Fuzzy SEIR-SEI Dengue Disease Model. *Journal of Mathematics*, Vol. 2022. <https://doi.org/10.1155/2022/1927434>
- [3] BPS, 2025. *Statistik Daerah Kota Kupang 2025 - Badan Pusat Statistik Kota Kupang*.
- [4] Chan, M., & Johansson, M. A., 2012. The Incubation Periods of Dengue Viruses. *PLoS ONE*, Vol. 7, No. 11, 1–7. <https://doi.org/10.1371/journal.pone.0050972>

- [5] Diekmann, O., Heesterbeek, J. A. P., & Roberts, M. G., 2010. The construction of next-generation matrices for compartmental epidemic models. *Journal of the Royal Society Interface*, Vol 7, No. 47, 873–885. <https://doi.org/10.1098/rsif.2009.0386>
- [6] Harianto, J., Angelika, V., & Seru, F., 2023. Kestabilan Lokal Titik Ekuilibrium Model. *Jurnal Matematika UNAND*, Vol. 12, No. 2, 153-167.
- [7] Hasan, M. R., Hobiny, A., & Alshehri, A., 2022. Analysis of Vector-host SEIR-SEI Dengue Epidemiological Model. *International Journal of Analysis and Applications*, Vol. 20, 1–19. <https://doi.org/10.28924/2291-8639-20-2022-57>
- [8] Kementerian Kesehatan Republik Indonesia., 2023. Deteksi Dini Demam Berdarah Dengue (DBD) dan Pengendaliannya di Indonesia Tahun 2023.
- [9] Ndi, M. Z., 2020a. *Pemodelan Matematika Dinamika Populasi dan Penyebaran Penyakit: Teori, Aplikasi, dan Numerik*.
- [10] Ndi, M. Z., 2020b. Modelling the use of vaccine and Wolbachia on dengue transmission dynamics. *Tropical Medicine and Infectious Disease*, Vol. 5, No. 2. <https://doi.org/10.3390/tropicalmed5020078>
- [11] Ndi, M. Z., Mage, A. R., Messakh, J. J., & Djahi, B. S., 2020c. Optimal vaccination strategy for dengue transmission in Kupang city, Indonesia. *Heliyon*, Vol. 6, No. 11. <https://doi.org/10.1016/j.heliyon.2020.e05345>
- [12] Phaijoo, G. R., & Gurung, D. B., 2018. Sensitivity Analysis of SEIR-SEI Model of Dengue Disease. *Journal of Gwalior Academy of Mathematical Sciences*, Vol. 6(a).
- [13] Purnamasari, S., Istichomah, B. A. Y., & Hamida, M., 2025. Faktor yang Mempengaruhi Pemberantasan Sarang Nyamuk (PSN) Melalui 3M Plus dalam Upaya Pencegahan Demam Berdarah Dengue (DBD). *Jurnal Ilmu Kesehatan*, Vol. 14, No. 01, 93–112. <https://journal.umg.ac.id/index.php/manajerial/article/view/10630>
- [14] Putra, R. T., Sukatik, & Nita, S., 2016. Kestabilan Model Epidemi SEIR dengan Matriks Hurwitz. *Jurnal Ilmiah Poli Rekayasa*, Vol. 11, No. 2, 74. <https://doi.org/10.30630/jipr.11.2.76>
- [15] Rahman, M. M., Hye, M. A., Miah, M. S., Islam, R., & Hossain, M. S., 2026. Insights and implications of a dynamical systems approach to dengue transmission and epidemic behaviour. *Scientific Reports*, Vol.16, No. 1. <https://doi.org/10.1038/s41598-026-38445-3>
- [16] World Health Organization., 2025. *Dengue*.