Temperature influences on dengue hemorrhagic fever mathematical model

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Abstract. Dengue hemorrhagic fever (DHF) has been modeled as an epidemic model by looking at the interaction between humans and mosquitoes. The Aedes Aegypti mosquito is the primary vector of DHF. Its life is influenced by climatic factors, including temperature, humidity, rainfall, and rainy days. In this study, a model for the spread of DHF will be formed by paying attention to climatic factors, namely temperature. Previous studies have shown that temperature affects mosquito populations and the frequency of mosquito bites against humans. This model will modify the mosquito population growth rate and the frequency of bites, which are usually considered constant, to be a temperature function. Using the temperature data of Semarang, we simulate to evaluate the temperature influence on the dynamic of DHF. The analysis result giving the temperature domain for the equilibrium existences, and the simulation result show that temperature affects the time it takes to converge to the equilibrium point.

1. Introduction
Dengue fever has become a major international health problem affecting tropical and sub-tropical regions worldwide - predominantly urban and suburban areas in recent years. The geographic distribution of dengue fever, the frequency of epidemic cycles, and the number of dengue cases have increased sharply over the past two decades. The frequency of potentially deadly complications of dengue, known as Dengue Hemorrhagic Fever (DHF), has begun to occur regularly in countries where previously only dengue had occurred. Dengue fever is caused by four different but closely related dengue viruses called serotypes (DEN-1, DEN-2, DEN-3, and DEN-4) and transmitted to humans through the bite of an infected mosquito, namely Aedes Aegypti as the primary vector [1].

Aedes aegypti, as the primary vector of DHF, is influenced by climatic factors, including temperature, humidity, rainfall, and rainy days. DHF is also an acute disease, endemic, but periodically it can cause extraordinary events and even epidemics. As is known so far, dengue fever will appear and spread every rainy season comes. However, during the dry season, it is also possible for someone to get dengue fever. Mosquitoes breed in stagnant water, such as in gutters that are clogged or cannot flow, and in stagnant water in used containers or water reservoirs that are rarely drained.

Many previous researchers have researched dengue fever. Some researchers modeled this disease by Susceptible-Infected-Recovery (SIR) model, while another researcher carries out Susceptible-Exposed-Infected-Recovery (SEIR) model. The SIR model of dengue disease fever was using on [2] with periodic recruitment rate, and also [3] was extending with two-age-classes transmission model. The SEIR models were used in [4] to analyze the spread of dengue fever. Control was applied on [5] and [6] with a different point of view. The SIR model with the effect of vaccination was given on [7]. The relationship between climate change and the spread of dengue fever has been carried out by [8] and [9].
On [8] conducted research to determine the likely determinants of the unprecedented in 2014, a population-based deterministic model was developed to describe the dynamics of dengue fever transmission in Guangzhou. Regional sensitivity analysis (RSA) was adopted to calibrate the model, and entomological surveillance data were used to validate the mosquito sub-model. Finally, it was found that the climate and time of the entry of cases were the main factors in the spread of dengue fever. In another way, [9] gave a fuzzy logic model to investigate the impact of climate on dengue transmission. The public health perspective on [10] also showed that weather influences dengue transmission in Malaysia. Model for effect of temperature and virus load given by [11], while the effect of rainfall given on [12]. On [13] impact of temperature and human movement included in the mathematical model.

In this study, modifications will be made to the simplest SIR model by adding the temperature that affects the transmission rate between human-mosquito, the mosquito survival rate, and the bit rate. Based on previous studies, this research will analyze the relationship between temperature and endemic conditions of dengue fever by including temperatures in some parameters and see how temperature influences the DHF mathematical model's dynamic. The simulation will be carried out using temperature data of Semarang City.

2. Mathematical Model

The mathematical model was adapted from [2] because we took the simplest model to modified with temperature factor. The transfer diagram is given in Figure 1.

![Figure 1. Transfer diagram infection of DFH](image)

Then we get a system of the differential equation below:
- Human population:

\[
\frac{dS_h}{dt} = \mu_h N_h - \frac{b\beta_h}{N_h} I_v S_h - \mu_h S_h
\]

\[
\frac{dI_h}{dt} = \frac{b\beta_h}{N_h} I_v S_h - (\mu_h + \gamma_h) I_h
\]

\[
\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h
\]

\[N_h = S_h + I_h + R_h\]  \(\ldots\) (1)

- Vector (mosquito) population:

\[
\frac{dS_v}{dt} = \Lambda - \frac{b\beta_v}{N_h} I_h S_v - \mu_v S_v
\]

\[
\frac{dI_v}{dt} = \frac{b\beta_v}{N_h} I_h S_v - \mu_v I_v
\]

\[N_v = S_v + I_v\]

The model can be rescaling into:

\[
\frac{ds_h}{dt} = (1 - s_h) - \frac{b\beta_h \Lambda}{\mu_h \mu_v N_h} i_v S_h
\]

\[
\frac{di_h}{dt} = \frac{b\beta_h \Lambda}{\mu_h \mu_v N_h} i_v S_h - \frac{(\mu_h + \gamma_h)}{\mu_h} i_h
\]

\[
\frac{dr_h}{dt} = \frac{\gamma_h}{\mu_h} i_h - r_h
\]

\[
\frac{ds_v}{dt} = \mu_v - \frac{b\beta_v}{\mu_h} i_h S_v - \frac{\mu_v}{\mu_h} S_v
\]

\[
\frac{di_v}{dt} = \frac{b\beta_v}{\mu_h} i_h (1 - i_v) - \frac{\mu_v}{\mu_h} i_v
\]

One of the factors of climate change is temperature, which significantly affects the spread of dengue fever. Several parameters for the mosquito population model are influenced or controlled by climate change factors. To enter a temperature-dependent feature \((T)\) into a parameter, it is defined temperature \((T)\) as a function of time. In this thesis, the effect of temperature \((T)\) is added to the parameters \((\beta_h, \beta_v, \mu_v, \text{dan } b)\) based on several previous studies and obtained as follows:

1. Rate of transmission of the virus from infected humans to susceptible mosquitoes \((\beta_v)\)

   Based on empirical data for various flaviviruses (West Nile virus, Murray Valley encephalitis virus, and St. Louis encephalitis virus), [14] obtained an association between temperature and infection rates.

   \[
   \beta_v(T) = \begin{cases} 
   0.0729T - 0.9037, & (12.4^\circ C \leq T \leq 26.1^\circ C) \\
   1, & (26.1^\circ C < T < 32.5^\circ C)
   \end{cases}
   \]

   This relationship is partially linear, with an increasing probability starting at 12 \(^\circ C\) and a constant probability of 1 over a temperature of 26.1\(^\circ C\).
2. Rate of transmission of the virus from infected mosquitoes to susceptible humans (β_h)

On [14] also described an equation for the rate of transmission of the virus from infected mosquitoes to susceptible humans using the following thermodynamic functions:

\[ \beta_h(T) = 0.001044T(T - 12,286)\sqrt{32,461 - T} \quad \text{for} \quad (12,286 ^\circ C \leq T \leq 32,461 ^\circ C) \]

β_h increasing linearly as T for 12.3°C ≤ T < 26°C, decreasing sharply while T > 28°C, and decreasing to zero while T ≥ 32.5°C.

3. Mosquito survival rate (μ_v)

From research by[15] on female Aedes Aegypti temperature around 10.54°C ≤ T ≤ 33.41°C got dead rate 0.027 (0.27%) per day to 0.092 (0.92%) per day with the highest survival rate at 27.6°C and lowest survival rate at T < 14°C dan T > 32°C. They got polynomial of degree 4th

\[ \mu_v(T) = 0.8692 - 0.1590T + 0.01116T^2 - 3,408 \times 10^{-4}T^3 + 3,809 \times 10^{-6}T^4 \]

4. Bite rate(b)

By [16] in their study explained that the bite rate of Aedes aegypti mosquitoes collected weekly increases linearly with weekly T in Thailand after converting weeks to days as follows:

\[ b(T) = 0.0043T + 0.0943 \quad (day^{-1})(21^\circ C \leq T \leq 32^\circ C) \]

This relationship was statistically significant (p = 0.05 and \( R^2 = 0.08 \)).

3. Result and Discussion

3.1. Analysis

The equilibria of system (2) solved with software Wolfram Mathematica, got disease-free equilibrium point \( F_1 = (1, 0, 0) \) and the endemic equilibrium point \( F_2 = (s_h^*, i_h^*, i_v^*) \) with

\[ s_h^* = \frac{\gamma + \beta \delta}{\gamma(1 + \alpha)} \]
\[ i_h^* = \frac{\alpha \gamma - \beta \delta}{\beta \gamma(1 + \alpha)} \]
\[ i_v^* = \frac{\alpha \gamma - \beta \delta}{\alpha(\gamma + \beta \delta)} \]

as \( \alpha = \frac{b(T)\beta_h(T)\alpha}{\mu_h\mu_v(T)N_h} \), \( \beta = \frac{\mu_h + \gamma_h}{\mu_h} \), \( \gamma = \frac{b(T)\beta_v(T)}{\mu_h} \), \( \delta = \frac{\mu_v(T)}{\mu_h} \).

The primary reproduction ratio of the system is: \( R_0 = \frac{\alpha \gamma}{\beta \delta} \).

Because \( \mu_v(T) \) is a polynomial of degree 4, for the analysis we took \( \mu_v \) is constant. Then we substitute \( \beta_v(T), \beta_h(T)b(T) \) into equilibria and primary reproduction number. By analyzing the positivity of equilibria and primary reproduction number, we got the temperature domain is 12,286°C < T < 32,461°C.

Equilibria existence and local stability analysis giving that if \( R_0 < 1 \) then only one equilibrium exist, which is \( F_1 \) and it is locally asymptotically stable. If \( R_0 > 1 \) then \( F_1 \) is unstable and \( F_2 \) is locally asymptotically stable. The analysis found that \( R_0 > 1 \) related with interval temperature 21°C < T < 32°C.
3.2. Simulation
To do the simulation, we took some parameters as follows in Table 1.

| Parameter | Value | Unit   | Citation |
|-----------|-------|--------|----------|
| $\Lambda$ | 400   | /day   | Assumption |
| $\mu_h$   | 0.000046 | /day   | [17]     |
| $\gamma_h$| 0.328833 | /day   | [17]     |

Based on the meteorological and geophysical agency's (BMKG) online data, the average daily temperature in Semarang during 2019 is 28.501 °C. For some parameters that influence by temperature, we were taking three different temperatures to see the difference. Then we were calculating $\beta_v(T), \beta_h(T), \mu_v(T)b(T)$, as can be seen in Table 2.

| Temperature | $\beta_v(T)$ | $\beta_h(T)$ | $\mu_v(T)$ | $b(T)$ |
|-------------|--------------|--------------|-------------|--------|
| 21 °C       | 0.6467681206 | 0.6272       | 0.036389329 | 0.1846 |
| 32 °C       | 0.4471731222 | 1.0          | 0.035731584 | 0.2319 |
| 28.501 °C   | 0.9601191738 | 1.0          | 0.026175563 | 0.2168543 |

Then from those parameter values, we get the equilibrium point and primary reproduction number for each temperature as in Table 3.

| Temperature $(T)$ | Equilibrium point | $R_0(T)$ |
|-------------------|-------------------|----------|
| 21 °C             | (0.7876753106, 0.000029697656, 0.000094481081) | 1.269678606 |
| 28.501 °C         | (0.1248956070, 0.000122400379, 0.0010130092) | 8.014805807 |
| 32 °C             | (0.4367425443, 0.000078782297, 0.00051104026) | 2.290849185 |

By taking the initial value $(0.9, 0, 0.1)$, we get the infected human's dynamic for each temperature as in Figure 2.

![Figure 2. Dynamic of infected human on different temperature](image-url)
Even it starts from the same initial condition, different temperatures giving different results in their dynamics. While the temperature is 21 ℃, the infected human increases sharply, then decline slowly and start to converge to the equilibrium condition at \( t = 20 \). At temperatures 32 ℃ and 28.501 ℃ giving almost the same behavior, but it is starting to converge at around \( t = 10 \). From the three different temperatures, it can be seen that the number of infected human is highest than two different temperatures in the average temperature.

4. Conclusion
The analysis result giving an interval of temperature for the existence of the equilibrium is \( 12,286°C < T < 32,461°C \) and the interval of the existence of the endemic equilibrium is \( 21°C < T < 32°C \). Simulation results showed that the temperature was giving an influence to the dynamic of DFH. The time needed to converge to the endemic equilibrium is shorter while temperature increasing. The temperature also influences the proportion of infected humans, but we can’t conclude how it works from the simulation. We should do more analysis to see further and probably do a continuation of the parameter. It could become the future work for this research.

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