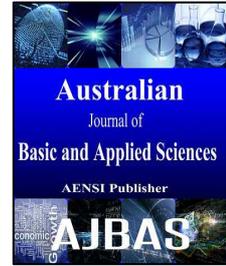




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Mathematical Model on the Transmission of Dengue Fever

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ABSTRACT

The objectives of this study were to propose the mathematical model on the transmission of Dengue fever and to analyze the mathematical model of Dengue fever transmission. The human population is divided into 3 subclass, that is, the susceptible human, infected human and recovered human and the mosquito population which divided into 2 subclass, that is, the susceptible mosquito and infected mosquito. We investigate the effect of parameter β_{vh} (the transmission rate from mosquito – to – human) and β_{hv} (the transmission rate from human – to – mosquito) and the calculation of the level of infection (Basic Reproductive Number : R_0) and to study disease – free equilibrium and disease endemic equilibrium states. After that, to determine the solutions by using the numerical simulation at each equilibrium points which affected to the local asymptotically stabilities, to determine of parameter β_{vh} (the transmission rate from mosquito – to – human) and β_{vh} (the transmission rate from mosquito – to – human), by using $\beta_{vh} = 0.05$ and $\beta_{hv} = 0.05$ which showed $R_0 = 0.965215 < 1$. The results showed that the all subclass of human were uninfected, it also showed that there were no the transmission of the disease. In addition, as regards to the numerical simulation and the parameter at disease – free equilibrium which affected to the local asymptotically stabilities, the results of the parameter showed; β_{hv} and β_{vh} , by using $\beta_{hv} = 0.9$ and $\beta_{vh} = 0.9$ the results showed $R_0 = 1.47083 > 1$ that mean there are the transmission of the disease.

INTRODUCTION

Dengue is a mosquito-borne viral disease that has rapidly spread in all regions of WHO in recent years. Dengue virus is transmitted by female mosquitoes mainly of the species *Aedes aegypti* and, to a lesser extent, *Ae. albopictus*. This mosquito also transmits chikungunya, yellow fever and Zika infection. Dengue is widespread throughout the tropics, with local variations in risk influenced by rainfall, temperature and unplanned rapid urbanization.

Severe dengue (also known as Dengue Haemorrhagic Fever) was first recognized in the 1950s during dengue epidemics in the Philippines and Thailand. Today, severe dengue affects most Asian and Latin American countries and has become a leading cause of hospitalization and death among children in these regions. There

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are 4 distinct, but closely related, serotypes of the virus that cause dengue (DEN-1, DEN-2, DEN-3 and DEN-4). Recovery from infection by one provides lifelong immunity against that particular serotype. However, cross-immunity to the other serotypes after recovery is only partial and temporary. Subsequent infections by other serotypes increase the risk of developing severe dengue. The incidence of dengue has grown dramatically around the world in recent decades. The actual numbers of dengue cases are underreported and many cases are misclassified. One recent estimate indicates 390 million dengue infections per year (95% credible interval 284–528 million), of which 96 million (67–136 million) manifest clinically (with any severity of disease).¹ Another study, of the prevalence of dengue, estimates that 3.9 billion people, in 128 countries, are at risk of infection with dengue viruses. (WHO, 2016). The *Aedes aegypti* mosquito is the primary vector of dengue. The virus is transmitted to humans through the bites of infected female mosquitoes. After virus incubation for 4–10 days, an infected mosquito is capable of transmitting the virus for the rest of its life. Infected symptomatic or asymptomatic humans are the main carriers and multipliers of the virus, serving as a source of the virus for uninfected mosquitoes. Patients who are already infected with the dengue virus can transmit the infection (for 4–5 days; maximum 12) via *Aedes* mosquitoes after their first symptoms appear. (CDC, 2015). In this paper, we proposed a mathematical model to describe the transmission of Dengue fever.

2. Model Formulation:

In our model, we assume that the human is constant. We formulate the model of Dengue fever transmission by using basic ideas taken from epidemiology that is mosquito-borne is high which effect to Dengue fever epidemic. The model is obtained by assuming:

The total human population N is divided into four compartments: Susceptible human denote by S_h (the members of the human population who may become infected). Infected mosquitoes denoted by I_v (the infected) and Recover human denoted by R_h (the numbers of recovered individuals). The dynamics of the disease is depicted in the flow chart shown in **Fig. 1**.

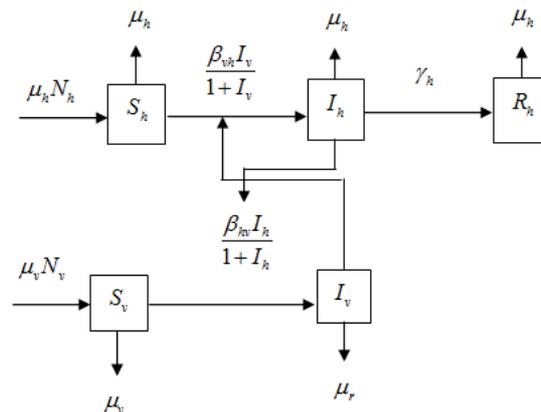


Fig. 1: Flow chart of the dynamics of dengue fever .

The dynamics of dengue is described by the following ordinary differential equations:

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_{vh}}{1+I_v} I_v S_h - \mu_h S_h \quad (1)$$

$$\frac{dI_h}{dt} = \frac{\beta_{vh}}{1+I_v} I_v S_h - (\mu_h + \gamma_h) I_h \quad (2)$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \mu_h R_h \quad (3)$$

$$\frac{dS_v}{dt} = \Lambda_v - \frac{\beta_{hv}}{1+I_h} I_h S_v - \mu_v S_v \quad (4)$$

$$\frac{dI_v}{dt} = \frac{\beta_{hv}}{1+I_h} I_h S_v - \mu_v I_v \quad (5)$$

with $N = S_h + R_h + I_h$

where

- S_h is the susceptible humans,
 I_h is the infective humans ,
 R_h is the recovered humans ,
 Λ_h is the birth rate of humans,
 μ_h is the natural death rate of humans,
 γ_h is the recovery rate of disease,
 β_{hv} is the infection rate from humans to mosquitoes ,
 N_h is the total human population ,
 S_v is the susceptible mosquitoes,
 I_v is the infected mosquitoes,
 μ_v is the natural death rate of mosquitoes,
 β_{vh} is the infection rate from mosquitoes to humans,
 N_v is the total number mosquitoes.

3. Analysis of the Model:

Equilibrium Points:

The system has two equilibrium points; a disease free equilibrium point and an endemic equilibrium point. We obtained these by setting the right hand sides of equations. (1) - (5) to zero. Doing this, we obtained

Disease Free Equilibrium Point: (E_0):

In the absence of the disease, i.e., $I_h = 0$, $I_v = 0$ equation (1), (2) becomes

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\beta_h}{1 + I_v} I_v S_h - \mu_h S_h \text{ and}$$

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

The solution to this equation is

$$S_h = \frac{\Lambda_h \mu_v^2 + (\beta_{nv} \mu_v + \mu_v^2 + \beta_{nv} \Lambda_v) \Lambda_h I_h}{\mu_h \mu_v^2 + (\beta_{nv} \mu_v \mu_h + \mu_v^2 \mu_h + \beta_{vh} \beta_{nv} \Lambda_v + \mu_h \beta_{nv} \Lambda_v)} \text{ and } R_h = \frac{\gamma_h I_h}{\mu_h} . \text{ The disease free state is}$$

$$E_0 = (1, 0, 0)$$

Endemic Equilibrium Point: (E_1):

In the case where the disease is presented, by setting

$I_h \neq 0, I_v \neq 0$. This gives

$$S_h^* = \frac{\Lambda_h \mu_v^2 + (\beta_{nv} \mu_v + \mu_v^2 + \beta_{nv} \Lambda_v) \Lambda_h I_h}{\mu_h \mu_v^2 + (\beta_{nv} \mu_v \mu_h + \mu_v^2 \mu_h + \beta_{vh} \beta_{nv} \Lambda_v + \mu_h \beta_{nv} \Lambda_v) I_h}$$

$$I_h^* = \frac{B_{vh} \Lambda_h \beta_{nv} \Lambda_v - \mu_h \mu_v^2 (\mu_h + \gamma_h)}{(\beta_{nv} \mu_v \mu_h + \mu_v^2 \mu_h + B_{vh} \beta_{nv} \Lambda_v + \mu_h \beta_{nv} \Lambda_v) (\mu_h + \gamma_h)} > 0$$

$$I_v^* = \frac{\beta_{nv} I_h \Lambda_v}{\mu_v^2 + (\beta_{nv} + \mu_v) \mu_v I_h}$$

Thus

$$E_1(S_h^*, I_h^*, I_v^*)$$

Basic Reproductive Number:

The basic reproductive number is obtained by the next generation matrix. In the notation of Van den Driessche and Watmough (2002), we start with

$$\frac{dx}{dt} = F(x) - V(x) \quad (6)$$

where $F(x)$ is the matrix of new infectious and $V(x)$ is the matrix of the transfers between the compartments in the infective equations. We obtained

$$F(x) = \begin{bmatrix} 0 \\ \frac{\beta_{vh} I_v S_h}{1 + I_v} \\ 0 \end{bmatrix}$$

and

$$V(x) = \begin{bmatrix} \frac{\beta_{vh} I_v S_h + \mu_h S_h - \mu_h N_h}{1 + I_v} \\ (\mu_h + \gamma_h) I_h \\ -\frac{\beta_{hv} I_h (\Lambda_v - \mu_v I_v)}{(1 + I_h) \mu_v} + \mu_v I_v \end{bmatrix}$$

$$\text{where } F = \begin{bmatrix} \frac{\partial F_i(E_0)}{\partial X_i} \end{bmatrix} \text{ and } V = \begin{bmatrix} \frac{\partial V_i(E_0)}{\partial X_i} \end{bmatrix}$$

for all $i, j = 1, 2, 3$. This are the Jacobian matrix of $F(x)$ and $V(x)$ at E_0 . The basic reproductive number, R_0 , is the threshold for indicating the degree of epidemiology of the disease. It can be determined by noting that

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

For our model, the Jacobian matrices are

$$F = \begin{bmatrix} 0 & 0 & 0 \\ 0 & 0 & \beta_{vh} \\ 0 & 0 & 0 \end{bmatrix} \text{ and}$$

$$V = \begin{bmatrix} \mu_h & 0 & \beta_{vh} \\ 0 & \mu_h + \gamma_h & 0 \\ 0 & -\beta_{hv} & \mu_v \end{bmatrix}$$

The inverse of is V

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu_h} & \frac{-\beta_{hv} \beta_{vh}}{\mu_h \mu_v (\mu_h + \gamma_h)} & \frac{-\beta_{vh}}{\mu_h \mu_v} \\ 0 & \frac{1}{\mu_h + \gamma_h} & 0 \\ 0 & \frac{\beta_{hv}}{\mu_v (\mu_h + \gamma_h)} & \frac{1}{\mu_v} \end{bmatrix}$$

This leads to

$$FV^{-1} = \begin{bmatrix} 0 & 0 & 0 \\ 0 & \frac{\beta_{vh} \beta_{hv}}{\mu_v (\mu_h + \gamma_h)} & \frac{\beta_{vh}}{\mu_v} \\ 0 & 0 & 0 \end{bmatrix}$$

Thus,

$$R_0 = \frac{\beta_{vh}\beta_{hv}}{\mu_v(\mu_h + \gamma_h)} \quad (7)$$

Local Asymptotically Stability:

The local stability of an equilibrium point is determined from the Jacobian matrix of the ordinary differential equation (1), (2) and (5) evaluated at E_0 . The Jacobian matrix at E_0 is

$$J_0 = \begin{bmatrix} -\mu_h & 0 & -\beta_{vh} \\ 0 & -\mu_h\gamma_h & \beta_{vh} \\ 0 & \beta_{hv} & -\mu_v \end{bmatrix}$$

The eigenvalues of the J_0 are obtained by solving $\det(J_0 - \lambda I) = 0$. From this, we obtain the characteristic equation,

$$(\lambda + \mu_h)[(-\mu_h - \gamma_h - \lambda)(-\mu_v - \lambda) - \beta_{hv}\beta_{vh}] = 0 \quad (\lambda + \mu_h)[\lambda^2 + (\gamma_h + \mu_h + \mu_v)\lambda + (\mu_h + \gamma_h)\mu_v - \beta_{hv}\beta_{vh}] = 0$$

$$(\lambda + \mu_h)[\lambda^2 + B\lambda + C] = 0$$

where

$$B = \mu_v + \mu_h + \gamma_h$$

$$C = (\mu_h + \gamma_h)\mu_v - \beta_{hv}\beta_{vh}$$

From the characteristic equation, we see that two eigenvalues are $\lambda_1 = -\mu_h < 0$. The other two are the solutions of the characteristic equation. The roots of this equation will be negative if two coefficients satisfied with the Routh-Hurwitz criteria (Allen, 2006).

$$1) B > 0$$

$$2) C > 0$$

Disease Endemic Equilibrium Point:

To determine the stability of the endemic equilibrium point. We examine the eigenvalues of Jacobian matrix at E_1 , which is

$$J_1 = \begin{bmatrix} \frac{\beta_{vh}I_v^*}{1+I_v^*} - \mu_h & 0 & -\frac{\beta_{vh}S_h^*}{(1+I_v^*)^2} \\ \frac{\beta_{vh}I_v^*}{1+I_v^*} & -\mu_h - \gamma_h & \frac{\beta_{vh}S_h^*}{(1+I_v^*)^2} \\ 0 & \frac{\beta_{hv}(\Lambda_v - \mu_v I_v^*)}{(1+I_h^*)^2 \mu_v} & -\frac{\beta_{hv}I_h^*}{1+I_h^*} - \mu_v \end{bmatrix}$$

Where are given by equations (6). The characteristic equation of Jacobian matrix at E_1 given by equations (1), (2) and (5) becomes,

$$\lambda^3 + P\lambda^2 + Q\lambda + R = 0$$

where

$$u = -\frac{\beta_{vh}I_v^*}{1+I_v^*} - \mu_h, \quad v = \frac{\beta_{vh}I_v^*}{1+I_v^*}, \quad w = -\mu_h - \gamma_h,$$

$$x = \frac{\beta_{hv}(\Lambda_v - \mu_v I_v^*)}{(1+I_h^*)^2 \mu_v}, \quad y = \frac{\beta_{vh}S_h^*}{(1+I_v^*)^2},$$

$$z = -\frac{\beta_{hv}I_h^*}{1+I_h^*} - \mu_v$$

$$P = -u - w - z$$

$$Q = uz + wz + uw - xy$$

$$R = vxy + uxy - uwz$$

The three eigenvalues of $\lambda^3 + P\lambda^2 + Q\lambda + R = 0$ will have negative real part if they satisfy the Routh - Hurwitz criteria (Allen,2006), that is $PQ > R$.

4 Numerical Results:

The value of parameters used in the numerical simulation are given in Table 1.

Table 1: Parameter values used in numerical simulations at disease free state.

Parameter	Values	Unit
Λ_h	0.000046	day ⁻¹
μ_h	0.000046	day ⁻¹
γ_h	0.328833	day ⁻¹
β_{hv}	0.05	-
N_h	1,000	-
Λ_v	0.0323	day ⁻¹
μ_v	0.0323	day ⁻¹
β_{vh}	0.05	-
N_v	50,000	-

Stability of the disease free state: Using the values of parameters listed in Table 1. We find the eigenvalues and basic reproductive number to be:

$$\lambda_1 = -0.000046, \lambda_2 = -0.027011, \lambda_3 = -0.000350228, \text{ and } R_0 = 0.965215 < 1$$

Since all of the eigenvalues are negative and the basic reproductive number is less than one, the equilibrium state will be the disease free state E_0 , as seen in **Fig. 2**

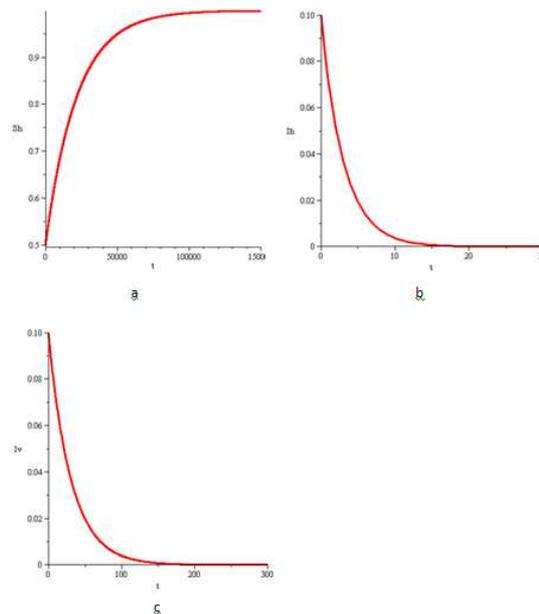


Fig. 2: The time series of (a) the susceptible humans , (b) the infected humans and (c) infected mosquitoes. As shown, all the state variables approach to the disease free state as seen, all the state variables approach their disease free state values $E_0 = (1, 0, 0)$.

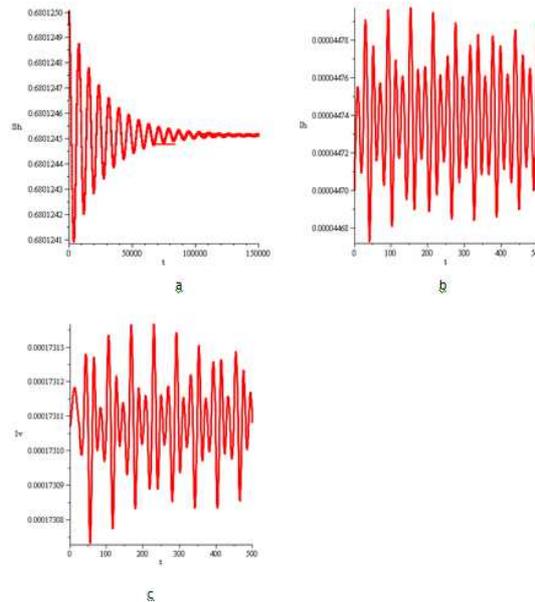


Fig. 3: The time series of (a) the susceptible humans, (b) the infected humans and (c) infected mosquitoes. Two the value of β_{vh} and β_{hv} has been changed to $\beta_{vh} = 0.9$ and $\beta_{hv} = 0.9$. All the state variables approach to endemic state.

Stability of the endemic state: Using the values of parameter listed in Table 1. except the value of β_{vh} and β_{hv} . We set to be equal 0.9. This values represented the value of infection from human to mosquitoes and the value of infection from mosquitoes to humans.

$$E_1 = (0.863379, 0.00600819, 0.0808781)$$

The eigenvalues and basic reproductive number become greater than one and the outcome is quite different:

$$\lambda_1 = -0.0270463, \quad \lambda_2 = -0.0000265039 - 0.0000813441i$$

$$\lambda_3 = -0.0000265039 + 0.0000813441i \text{ and } R_0 = 1.47083 > 1$$

Since all of the eigenvalues are to be negative and the basic reproductive number is greater than one, the equilibrium state will be the endemic state, E_1 as demonstrated in **Fig. 3**.

Discussion and Conclusion:

In this study, we proposed and analyzed the transmission model of Dengue fever with effect of the infection rate between human and mosquitoes. Model analysis by using standard modeling method. The basic reproductive number is obtained through the use of spectral radius of the next generation matrix. The basic

reproductive number is $R_0 = \frac{\beta_{vh}\beta_{hv}}{\mu_v(\mu_h + \gamma_h)}$. From Fig. 3, we see that if the infection rate both human-to-

mosquito and mosquito- to- human increase, the number of infected human increase. So that the disease will occur. But when the infection rate both human-to-mosquito and mosquito- to- human decrease, the number of infected human decrease. In this case the disease will died out.

It can summarize as follows:

1) The spreading of dengue fever has two states: the disease-free state and the endemic state. The happening of a state depends on β_{vh} and β_{hv} . If $\beta_{vh} = 0.05$ and $\beta_{hv} = 0.05$ provided $R_0 < 1$, then the disease-free state will occur, but if $\beta_{vh} = 0.9$ and $\beta_{hv} = 0.9$ provided $R_0 > 1$ then the endemic state will occur.

2) The stability of the model is determined. Routh-Hurwitz criteria is used to prove that each equilibrium point is locally asymptotically stable. In addition, for any initial population, over a long time, the population will converge to the equilibrium points as shown in Fig 2-3.

3) The higher of the infection from mosquitoes to humans and the infection from people to mosquitoes (β_{hv}) and (β_{vh}) increase the infected individuals as shown in Fig 3 . It concluded that if the infected mosquitoes is high, the number of dengue infection will be increase.

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