Mathematical Model of Dengue Virus with Predator-Prey Interactions
(Model Matematik Virus Denggi dengan Interaksi Pemangsa-Mangsa)

SARINAH BANU MOHAMED SIDDIK*, FARAH AINI ABDULLAH & AHMAD IZANI MD. ISMAIL

ABSTRACT
In this paper, a mathematical model of dengue incorporating two sub-models that: describes the linked dynamics between predator-prey of mosquitoes at the larval stage, and describes the dengue spread between humans and adult mosquitoes, is formulated to simulate the dynamics of dengue spread. The effect of predator-prey dynamics in controlling the dengue disease at the larval stage of mosquito populations is investigated. Stability analysis of the equilibrium points are carried out. Numerical simulations results indicate that the use of predator-prey dynamics of mosquitoes at the larval stage as biological control agents for controlling the larval stage of dengue mosquito assists in combating dengue virus contagion.

Keywords: Dengue virus; endemic equilibrium; numerical simulation; predator-prey

ABSTRAK
Dalam kertas ini, satu model matematik denggi yang menggabungkan dua sub-model iaitu: menerangkan dinamik antara nyamuk pemangsa-mangsa pada peringkat jejentik dan menerangkan penyebaran denggi antara manusia dan nyamuk dewasa, diformulasikan untuk mensimulasi dinamik penyebaran denggi. Kesan dinamik pemangsa-mangsa untuk mengawal penyakit denggi pada peringkat jejentik populasi nyamuk diselidik. Analisis kestabilan titik kesimbanan dijalankan. Simulasi berangka menunjukkan bahawa penggunaan dinamik pemangsa-mangsa nyamuk pada peringkat jejentik sebagai agen kawalan biologi untuk mengawal tahap jejentik nyamuk denggi membantu dalam memerangi penularan virus denggi.

Kata kunci: Keseimbangan endemik; pemangsa-mangsa; simulasi berangka; virus denggi

INTRODUCTION
Dengue is a disease that is endemic in over a hundred countries (WHO 2016). Since the 1970s, dengue fever has spread throughout the Southeast Asia and as of 2010 about 60 million people were infected with the virus (Wen et al. 2016). Dengue virus is transmitted to humans through a type of mosquito known as *Aedes aegypti* (Zaini et al. 2019). *Aedes aegypti* is responsible for most of the global dengue diffusion although some other species such as *Aedes albopictus* is also involved (Nuraini et al. 2009; Ong 2016). Infection with dengue virus may cause either dengue fever (DF), dengue hemorrhagic fever (DHF) or dengue shock syndrome (DSS) (Derouich & Boutayeb 2006). The DHF and the DSS are the severest forms of dengue disease which may lead to fatality greater than 20% (WHO 2016). Individuals infected by one serotype of the virus will become immune to that serotype. Nevertheless, subsequent infection with any of the other three serotypes makes the individual prone to DHF and DSS. The DF is described by a sudden fever and intense headaches and persists for three to seven days. On the other hand, the DHF or DSS is characterized by a sudden fever, nausea, vomiting and fainting due to low blood pressure. It takes between two to three days to recover and may lead to death (Derouich & Boutayeb 2006). Thus investigating possible procedures for controlling the spread of dengue is important. The best way to manage dengue occurrence is to focus on prevention - keeping the mosquito population below the spread threshold (Esteva & Vargas 1998).

Many mathematical models of dengue transmission dynamics have been developed. Bailey (1975) introduced the first basic mathematical model of dengue. A mathematical model for dengue with variable humans and mosquitoes consisting of susceptible-infected-recovered model for humans and susceptible-infected for the mosquitoes was formulated. Later, Esteva and Vargas (1998) extended the Bailey’s model to establish the global stability of the endemic equilibrium to the model. They concluded that the disease-free equilibrium is stable whenever a basic reproduction number is smaller than unity. This model was used to study the effectiveness of insecticide dispersion. Derouich and Boutayeb (2006) formulated a mathematical model for dengue fever with the aim of investigating the effect of immunization. They also discussed the possibility of a partial vaccination to control a second epidemic and the evolution of dengue to DHF. Yang and Ferreira (2008)...
The model was used to establish whether the predation can check pathogen perseverance or change the stability of host-pathogen dynamics. The study showed that the absence of predation leads to proportional increment of pathogen pervasiveness in the host with vector productiveness. The predator can raise the host number indirectly by reducing or eradicating infectivity in the host population. However, only one control strategy was considered in this model.

Lou and Zhao (2011) presented a mathematical malaria model which describes the linked dynamics between the host-vector and the predator-prey interactions. The model modified the work of Moore et al. (2010) by focusing on the impact of the predator-larval mosquito relations on the transmission of mosquito-borne pathogens. The study evaluated the possible impacts of the biological control strategy on the spread of the disease. They concluded that introducing carnivorous fish as a biological control strategy can have important consequences for the disease dynamics. The introduction of larvivorous fish also has consequences for malaria dynamics and indicates that strong predators are required on larval mosquitoes. Nevertheless, the model did not consider the susceptible human and the recovered human and only one control strategy was considered in this model.

Ghosh et al. (2013) derived a nonlinear mathematical model for malaria which studied the introduction of predatory fish as a biological control agent by considering both human and mosquitoes populations variable. They modified the model of Lou and Zhao (2011) by incorporating all possible breeding sites of mosquitoes. They concluded that the introduced predatory fish has an effect on the spread of the disease. It should be noted the model did not consider the recovered human population since human are either recovered or get immunity and only one control strategy was used in this model.

Most of the current activities to develop biological control strategies require several challenges, thus, making a prediction of the biological predator intervention uncertain. Based on this, the model considered in this study offers some extensions to the dengue transmission model in Ali et al. (2015), and Pandey et al. (2013) which describes the linked dynamics between the predator-prey interactions, humans and adult mosquitoes interaction. The predator-prey dynamic of mosquitoes at the larval stage involve the use of larvivorous Toxorhynchites splendens mosquitoes as biological control agent for controlling the larval stage of dengue mosquito (Aedes aegypti). Moreover, the model will examine its impact in reducing the spread of dengue. The model involves the interactions between human and mosquito populations, and mathematically written as a system of ordinary differential equations.

Our paper is structured as follows: in the next section, a mathematical model is developed to simulate the dynamics of the dengue disease spread. Equilibrium
state and stability are discussed subsequently. After that, the model is investigated numerically. Finally, we present the conclusion in the last section.

METHODS

FORMULATION OF THE MODEL

The works of Ali et al. (2015) and Pandey et al. (2013) are extended by incorporating the use of predator-prey dynamics of mosquitoes at the larval stage. We develop a mathematical model that integrates two sub interactions: predator-prey (larvivorous *Tx. splendens* and larvae *Aedes aegypti* mosquitoes) and human-adult mosquitoes. We seek to examine the indirect effect of predators via a mosquito since current models of disease dynamics have yet to consider the potential role of predators in regulating mosquito populations (Moore 2010). Figure 1 shows a schematic diagram of the model.

**FIGURE 1. Schematic diagram of the model**

**MODEL STRUCTURE**

The total human population at time $t$, denoted by $H(t)$, is divided into susceptible human $H_s(t)$, infected human $H_I(t)$ and recovered human $H_R(t)$ (Pandey et al. 2013). Hence,

$$H = H_s + H_I + H_R$$  \hspace{1cm} (1)

The *Aedes aegypti* mosquito population is split into the larval stage of *Aedes aegypti* mosquitoes $L$, the larval stage of predatory mosquitoes (*Tx. splendens* mosquitoes) $P$ and the total adult *Aedes aegypti* mosquito stage at a time $t$, denoted by $V(t)$, which in turn is split into susceptible mosquitoes $V_s(t)$ and infected mosquitoes $V_I(t)$. Thus,

$$V = V_s + V_I$$  \hspace{1cm} (2)

**HUMAN POPULATIONS**

The susceptible humans are increased via recruitment of human (by birth or immigration) into the population at a constant rate $\pi_H$. They are reduced once the susceptible human acquires the disease after interacting with the infected *Aedes aegypti* mosquito at a rate $\beta_1 c$, where $\beta_1$ is the transmission probability per bite and $c$ is the biting rate of mosquitoes, $\pi_H$ is the contact rate of mosquito per human per unit time. The recovered human turn out to be susceptible again at a rate $\gamma_H$. The susceptible human experiences natural death at a rate $\mu_H$ (Ali et al. 2015; Pandey et al. 2013). This gives

$$\frac{dH_s}{dt} = \pi_H + \gamma_H H_H - \mu_H H_s.$$  \hspace{1cm} (3)

Infected humans are generated through infection of susceptible humans following an incubation period of 4-10 days (WHO 2016). They acquire immunity at a rate $\alpha_H$, suffer a disease-induced death at a rate $\delta_H$ and natural death at a rate $\mu_H$ (Ali et al. 2015; Pandey et al. 2013). Thus,

$$\frac{dH_I}{dt} = \beta_1 c H_s V_I - (\alpha_H + \delta_H + \mu_H) H_I.$$  \hspace{1cm} (4)
Infected human evolves to the recovered class after acquiring immunity at a rate $\sigma_H$. The recovered human population losses immunity at a rate $\gamma_P$ and suffers a natural death at a rate $\mu_H$ (Ali et al. 2015; Pandey et al. 2013). Hence,

$$\frac{dH_R}{dt} = \alpha_H H_I - (\gamma_H + \mu_H) H_R . \tag{5}$$

**LARVA STAGE OF PREDATORY MOSQUITOES**

The larval stage of predatory mosquitoes is the contact rate of mosquito per unit time. Other mosquitoes larvae suffer death due to mosquito larvae at a rate $\mu_P$. The predator mosquito larval stage suffers a natural death at a rate $\mu_P$. The larva matures at a rate $\pi_P$, and leaves the larval stage to adult mosquito with the assumption that $k$ is the tropical convention efficiency. The adult *Aedes splendens* mosquitoes does not consume blood while feeding on sugar rich materials such as fruit and nectar (Benelli et al. 2016). The predation of the larvae by the predatory mosquitoes is assumed to take a linear response form with a constant rate $\gamma_L$ (Ghosh et al. 2013). This gives

$$\frac{dP}{dt} = \Lambda_P + k \gamma_L LP - (\pi_P + \mu_P) P . \tag{6}$$

**PREY MOSQUITOES POPULATIONS**

The larval stage of *Aedes aegypti* mosquitoes $L$ are generated by egg laying rate of the mosquitoes at a rate $\Lambda_L$. Other mosquitoes larvae suffer death due to larvivorous predatory *Aedes splendens* larvae feed on the mosquito larvae at a rate $\gamma_L P$ and a natural death of larvae itself at a rate $\mu_L$. Then, the larvae matures at a rate $\pi_L$, leaving the larval stage to adult mosquitoes (Ghosh et al. 2013). Thus,

$$\frac{dL}{dt} = \Lambda_L - (\pi_L + \gamma_L P + \mu_L) L . \tag{7}$$

Susceptible mosquitoes are generated via recruitment of *Aedes aegypti* mosquitoes (birth or immigration) at a constant rate $\pi_P$. They acquire the infection after the interaction with humans, at a rate $\beta_p \mathcal{E}$, where $\beta_p$ is the probability of *Aedes aegypti* mosquitoes getting infected through infected humans, $\mathcal{E}$ is the biting rate of mosquitoes and $C$ is the contact rate of mosquito per human per unit time. It experiences a natural death at a rate $\mu_P$ (Ali et al. 2015; Pandey et al. 2013). Therefore,

$$\frac{dV_s}{dt} = \pi_L L - \beta_p \mathcal{E} C V_s H_I - \mu_P V_s . \tag{8}$$

From the above assumptions and formulations together with the schematic diagram in Figure 1, the resulting system of non-linear differential equations for the dengue model is obtained:

$$\frac{dH_I}{dt} = \pi_H + \gamma_H H_R - \beta_p \mathcal{E} C H_I V_s - \mu_H H_I$$

$$\frac{dH_R}{dt} = \beta_p \mathcal{E} C H_I V_s - (\alpha_H + \delta_H + \mu_H) H_R$$

$$\frac{dH_L}{dt} = \alpha_H H_I - (\gamma_H + \mu_H) H_L$$

$$\frac{dP}{dt} = \Lambda_P + k \gamma_L LP - (\pi_P + \mu_P) P$$

$$\frac{dL}{dt} = \Lambda_L - (\pi_L + \gamma_L P + \mu_L) L$$

$$\frac{dV_s}{dt} = \pi_L L - \beta_p \mathcal{E} C V_s H_I - \mu_P V_s .$$

Thus, the feasible set of the model (9) is given by

$$\Omega = \left\{(H_I, H_R, H_L, L, P, V_s) \in \mathbb{R}^7 : H \leq \frac{\Lambda_L}{\mu_P}, P \leq \frac{\Lambda_P}{\pi_P + \mu_P}, L \leq \frac{\Lambda_L}{\pi_L}, \mu_L \right\} \tag{15}$$

The model (9) describes the interaction between susceptible, infected and recovered human population in (9) - (9) with change in time. Similarly, the susceptible and infected mosquito populations interact in (9) - (9) with change in time.

**MODEL ANALYSIS**

**INVARIANT REGION**

Let $(H_I, H_R, H_L, L, P, V_s, V_I) \in \mathbb{R}^7$ be any solution of the system with nonnegative initial conditions that is,

$$\frac{dH_I}{dt} \leq \pi_H - \mu_H H$$

Thus, from the standard comparison theorem by Cooke and Van Den Driessche (1996), it is clear that $0 \leq H \leq \frac{\Lambda_L}{\mu_P}$, such as written in a simplified form. Thus,

$$\pi_H - \mu_H H \geq K e^{-\mu_H t} \text{ where } K \text{ is a constant.} \tag{11}$$

Hence, all possible solutions of the human population of the model (9) are in the region

$$\Omega_H = \left\{(H_I, H_R, H_L) \in \mathbb{R}^3 : H \leq \frac{\mu_L}{\mu_P} \right\} \tag{12}$$

Similarly, all the possible solutions of the mosquito population of the model (9) are in the same region with human population as follow,

$$\Omega_L = \left\{(P, L) \in \mathbb{R}^2 : P \leq \frac{\Lambda_P}{\pi_P + \mu_P}, L \leq \frac{\Lambda_L}{\pi_L} \right\} \tag{13}$$

$$\Omega_V = \left\{(V_s, V_I) \in \mathbb{R}^2 : V \leq \frac{\pi_L \Lambda_L}{\mu_P (\pi_P + \mu_P)} \right\} \tag{14}$$

Therefore, the feasible set of the model (9) is given by

$$\Omega = \left\{(H_I, H_R, H_L, L, P, V_I, V_s) \in \mathbb{R}^7 : H \leq \frac{\Lambda_L}{\mu_P}, P \leq \frac{\Lambda_P}{\pi_P + \mu_P}, L \leq \frac{\Lambda_L}{\pi_L}, \mu_L, V \leq \frac{\pi_L \Lambda_L}{\mu_P (\pi_P + \mu_P)} \right\} \tag{15}$$
The positively invariant is induced by the model (9) and hence, the non-negative properties have been preserved for the total size of the population. Furthermore, in this domain, it is sufficient to consider the dynamics of the flow generated by the model (9). The investigation of invariant region includes both humans and mosquitoes population.

**Positivity of Solution**

Since the model (9) monitors human and mosquito populations, it is therefore important that all state variables involved are non-negative for all time and are bounded in $\mathbb{R}^*_+$, because the total populations of both human and mosquito are non-negative. Let the initial data be $\{H(0), H_s(0), H_k(0), P(0), L(0), V(0), V_r(0) \geq 0\} \in \Omega$.

Then, the solution set $\{H, H_s, H_k, P, L, V, V_r, V_j\}(t)$ of the model (9) is positive for all $t > 0$. Based on the model (9) the first equation yields,

$$\begin{align*}
\frac{dH}{dt} & = \mu_H H_s - (\lambda_s + \mu_H) H_s \\
\frac{dH_s}{dt} & = - (\lambda_s + \mu_H) H_s \\
\int \frac{dH_s}{H_s} & = - (\lambda_s + \mu_H) dt \\
H_s(t) & = H_s(0) e^{- (\lambda_s + \mu_H) t} \\
H_j(t) & > 0.
\end{align*}$$  \tag{16}

As $t \to \infty$, $H_j(t) > 0$, from the model (9) the seventh equation gives

$$\begin{align*}
\frac{dV_j}{dt} & = -\lambda_V V_j - \mu_V V_j \\
\frac{dV_s}{dt} & = - \mu_V V_j \\
\int \frac{dV_j}{V_j} & = - \mu_V V_j dt \\
V_j(t) & = V_j(0) e^{- \mu_V t} \\
V_j(t) & > 0.
\end{align*}$$  \tag{17}

As $t \to \infty$, $V_j(t) > 0$. Similarly, it can be shown that $H_j, H_k, L, P, V_s$ are greater than zero for all time, $t > 0$.

**Equilibrium Points and Stability**

In this section, the model (9) is analyzed to identify the equilibrium points of the system and their stability properties. To find the equilibrium points of the model (9), the model is set to zero on the right hand side to obtain the following system of equations;

$$\begin{align*}
\pi_H + a_H H_s - \beta V_s H_j - \mu_H H_s = 0 \\
\beta V_s H_j - (\delta_H + a_H + \mu_H) H_j = 0 \\
\mu_H H_j - (\gamma_H + \mu_H) H_k = 0 \\
\Lambda_r + k \gamma_r L - (\pi_r + \mu_r) L = 0 \\
\Lambda_L - (\gamma_r L + \pi_r + \mu_L) L = 0 \\
\pi_r L - \beta V_s H_j H_k - \mu_s V_s = 0 \\
\beta V_s H_j H_k - \mu_s V_s = 0
\end{align*}$$  \tag{18}

The model (9) exhibits two types of equilibrium which are disease-free equilibrium (DFE) and disease-free equilibrium state $E_0$ in the absence of infection by the disease (dengue). The DFE are equilibrium-point solutions when there is no disease (dengue). Diseased compartments are classes of both human and mosquito populations that are infected with dengue in the model (9). Therefore, in the absence of infection, that is when $H_j, V_s = 0$, the model (9) has a point known to be infection free equilibrium or disease-free equilibrium state $E_0$ as a steady state. If $H_j, V_s = 0$ is substituted in the model (9), the system reduces to

$$\pi_H - \mu_H H_s = 0$$

implying that $H_s^* = \frac{\pi_H}{\mu_H}$ for human population and for mosquito population, the system reduces to

$$\pi_r L - \mu_s V_s = 0$$

implying that $V_s^* = \frac{\pi_r L}{\mu_s}$. Consequently, the DFE point of the model (9) is attained by putting,

$$\begin{align*}
\frac{dH}{dt} & = \frac{dH_s}{dt} = \frac{dP}{dt} = \frac{dL}{dt} = \frac{dV}{dt} = \frac{dV_s}{dt} = 0.
\end{align*}$$  \tag{19}

Thus, from (19) the infection free equilibrium point is given by,

$$E_0 = (H_s^*, H_j^*, H_k^*, L, P, V, V_s^*, V_r^*),
= \left( \frac{\pi_H}{\mu_H}, 0, L, \frac{\Lambda_r}{\pi_r + \mu_r}, \frac{\Lambda_L}{\pi_L}, \frac{\pi_r \Lambda_r}{\mu_r + \mu_L} \right).$$  \tag{20}

This represents the state where there is no infectivity in a community and it is known as the disease free equilibrium point.

**Basic Reproduction Number, $R_0$**

In a fully susceptible population, the basic reproduction number is the number of new cases that one infected individual will produce for the duration of its period of infectiousness. As $R_0 < 1$, each infected individual typically leads to less than one diseased individual and
hence the infection dies out. If \( R_0 < 1 \), each infected individual will produce more than one new infected individual. Thus, the infection overruns the population. Therefore, it is a measure of the severity of the epidemic. Next Generation Operator is then used to establish the linear stability of the disease (Diekmann et al. 1990). Since the concern is with the populations that spread the infection (Heffernan et al. 2005), we thus consider only the infected compartments \( H_I, V_I \) of the model (9). In order to compute the basic reproduction number \( R_0 \), we have

\[
\begin{align*}
\frac{dH_I}{dt} &= \lambda_I H_S - (\delta_H + \mu_H + \alpha_H) H_I \\
\frac{dV_I}{dt} &= \lambda_V V_S - \mu_V V_I
\end{align*}
\]

Then, the matrices \( F_i \) and \( V_i \) are the rates of appearance of new infection in the compartment \( i \) and the removal of persons/mosquitoes into and out of compartment \( i \) by all other means respectively. Hence, they are obtained as follows,

\[
F_i = \begin{bmatrix} \lambda_I H_S \\ \lambda_V V_S \end{bmatrix},
\]

and

\[
V_i = \begin{bmatrix} (\delta_H + \mu_H + \alpha_H) H_I \\ \mu_V V_I \end{bmatrix}.
\]

Thus, to obtain the appearance of new infection \( (F) \) and the removal of individuals in and out of the compartment \( i \) and \( V \) from model (9), two infected classes need to be considered; one class of humans and the other one from \( Aedes aegypti \) mosquito populations. Partial derivatives of \( F_i \) and \( V_i \) with respect to the infected classes, \( H_I, V_I \) are given respectively, as

\[
F = \begin{bmatrix} 0 & \beta_i \epsilon_i c_i H_S \\ \beta_i \epsilon_i c_i V_S & 0 \end{bmatrix},
\]

\[
V = \begin{bmatrix} \delta_H + \mu_H + \alpha_H & 0 \\ 0 & \mu_V \end{bmatrix}.
\]

Evaluating \( F \) at \( DFE \), \( E_0 \) yield

\[
F = \begin{bmatrix} 0 & \beta_i \epsilon_i c_i \frac{\pi_H}{\mu_H} \\ \beta_i \epsilon_i c_i \frac{\pi_V A_i}{\mu_i (\pi_V + \mu_i)} & 0 \end{bmatrix},
\]

and

\[
V^{-1} = \begin{bmatrix} 1 & 0 \\ 0 & \frac{1}{\mu_V} \end{bmatrix}.
\]

The two matrices \( F \) and \( V^{-1} \) are conducted using Maple-17 to obtain the basic reproduction numbers and it is given by,

\[
R_0 = \rho(FV^{-1}) = \frac{\beta_i \epsilon_i c_i \pi_H \beta_i \epsilon_i c_i \pi_V A_i}{\mu_H (\pi_V + \mu_V) (\delta_H + \mu_H + \alpha_H) \mu_i} \quad (22)
\]

Hence, The DFE of the model (9) is given by \( E_0 \) which is locally asymptotically stable. This means the disease will die out in the community (stable) if \( R_0 < 1 \) and unstable if \( R_0 > 1 \).

### Numerical Simulations

In order to illustrate the behavior of both mosquito and human population interact with and without control of predatory mosquitoes, numerous simulations were conducted by applying the set of parameters values depicted in Table 1. All the simulations and graph were obtained using MATLAB software.

| Parameter | Description | Est. value | References |
|-----------|-------------|------------|------------|
| \( \pi_H \) | Recruitment rate of human | 60 | Andraud et al. (2012) |
| \( \pi_V \) | Recruitment rate of \( Aedes aegypti \) mosquitoes | 3000 | Andraud et al.(2012) |
| \( \mu_H \) | Natural death of human | 0.0000457 | Al-Sulami et al. (2014) |
| \( \delta_H \) | Disease-induced death of human | 0.05 | Hove-Musekwa et al. (2008) |
| Parameter | Description | Value | Source |
|-----------|-------------|-------|--------|
| $\gamma_H$ | Rate of loss of human immunity | 0.02877 | Menach et al. (2005) |
| $\beta_1$ | Probability of human getting infected | 0.75 | Al-Sulami et al. (2014) |
| $\alpha_1$ | Biting rate of human | 1 | Derouich and Boutayeb (2006) |
| $c$ | Contact rate of *Aedes aegypti* mosquito per human per unit | 0.75 | Derouich and Boutayeb (2006) |
| $\beta_2$ | Probability of *Aedes aegypti* mosquitoes getting infected | 1 | Al-Sulami et al. (2014) |
| $\epsilon_2$ | Biting rate of *Aedes aegypti* mosquitoes | 0.5 | Al-Sulami et al. (2014) |
| $\alpha_H$ | Recovery rate of human | 0.1428 | Al-Sulami et al. (2014) |
| $\mu_f$ | Natural death of *Aedes aegypti* mosquitoes | 0.25 | Al-Sulami et al. (2014) |
| $\Lambda_p$ | Egg laying rate of predatory mosquitoes | 50 | Ghosh et al. (2013) |
| $\Lambda_L$ | Egg laying rate of *Aedes aegypti* mosquitoes | 10 | Ghosh et al. (2013) |
| $k$ | Tropical convention efficiency | 0.1 | Ghosh et al. (2013) |
| $\gamma_L$ | Predation of larva stage by predatory mosquitoes | 0.1 | Ghosh et al. (2013) |
| $\pi_p$ | Larva stage mature of predatory mosquitoes | 0.0625 | Ghosh et al. (2013) |
| $\mu_p$ | Natural death of predatory mosquitoes | 0.36 | Ghosh et al. (2013) |
| $\mu_L$ | Natural death of *Aedes aegypti* larva | 0.05 | Ghosh et al. (2013) |

**FIGURE 2.** The effect of interaction of human populations without predatory mosquitoes with $H_s = 8000$, $H_i = 0$ and $H_L = 0$.
The graph in Figure 2 indicates that the susceptible population is decreasing where every susceptible person is being infected by day 31. The line for a less infectious disease would slope more gently to the right while the infected population is increasing rapidly up to a maximum of 3400 peoples by the day of 34, and then falls more slowly until the day of 60. The recovered population increased continuously to the day of 60. This is because there is no control strategy (predatory mosquitoes) to control the spread of the virus. The graphs in Figure 3 indicate the typical behavior of human populations with the control of predatory mosquitoes. It has been shown that in the presence of control of predatory mosquitoes, the number of human decreases between the 50th and 60th day. The graph in Figure 4 shows that predatory mosquitoes can reduce the Aedes aegypti mosquitoes at the larval stage.
since the egg laying rate is 50 for predatory mosquitoes and 10 for prey.

**CONCLUSION**

Biocontrol strategies for mosquito populations are needed to help reducing the use of insecticides that are currently used for mosquito control. Despite significant improvement to the existing methods so far, larger scale trials are needed to determine the effective method of mosquito biocontrol. In view of this finding has shown that the use of predator-prey dynamic of mosquitoes at the larval stage (larvivorous *Tx. splendens* larvae) can be a potential biological control agent for controlling the larval stage of dengue mosquito (*Aedes aegypti*) and thus in controlling the dengue virus.

**ACKNOWLEDGEMENTS**

We acknowledge financial support from Fundamental Research Grant Scheme, 203/PMATHS/6711570.

**REFERENCES**

Ali, T.M., Kamil, A.A. & Karim, M.F.A. 2015. Deterministic mathematical model of dengue disease spread. *Far East Journal of Mathematical Sciences* 96(4): 419-436.

Al-Sulami, H., El-Shahed, M., Nieto, J.J. & Shammas, W. 2014. On fractional order dengue epidemic model. *Hindawi Publishing Corporation* 2014: 456537.

Andraud, M., Hens, N., Marais, C. & Beutels, P. 2012. Dynamic epidemiological models for dengue transmission: A systematic review of structural. *PLoS Comput. Biol.* 7(11): 332-346.

Bailey, N.T.J. 1975. *The Mathematical Theory of Infectious Diseases and its Applications*. London: Griffin.

Benelli, G., Jeffries, C.L. & Walker, T. 2016. Biological control of mosquito vectors: Past, present and future. *Insects* 7(4): e52.

Cooke, K.L. & Van Den Driessche, P. 1996. Analysis of an SEIRS epidemic model with two delays. *Journal of Mathematical Biology* 35(2): 240-260.

Derouich, M. & Boutayeb, A. 2006. Dengue fever: Mathematical modelling and computer simulation. *Applied Mathematics and Computation* 177(2): 528-544.

Diekmann, O., Heesterbeek, J. & Metz, J.A. 1990. On the definition and the computation of the basic reproduction ratio *R*0 in models for infectious diseases in heterogeneous populations. *Journal of Mathematical Biology* 28(4): 365-382.

Erikson, R.A., Presley, S.M., Allen, L.J.S., Long, K.R. & Cox, S.B. 2011a. A stage-structured, *Aedes albopictus* population model. *Ecological Modelling* 221(9): 1273-1282.

Erikson, R.A., Presley, S.M., Allen, L.J.S., Long, K.R. & Cox, S.B. 2011b. A dengue model with a dynamic *Aedes albopictus* vector population. *Ecological Modelling* 221(24): 2899-2908.

Esteva, L. & Vargas, C. 1998. Analysis of a dengue disease transmission model. *Mathematical Biosciences* 150(2): 131-151.

Ghosh, M., Lashari, A.A. & Li, X.Z. 2013. Biological control of malaria: A mathematical model. *Applied Mathematics and Computation* 219(15): 7923-7939.

Heffernan, J.M., Smith, R.J. & Wahl, L.M. 2005. Perspectives on the basic reproductive ratio. *Journal of the Royal Society Interface* 2(4): 281-293.

Huang, Y.J.S., Stephens, H. & Vanlandingham, D.L. 2017. Biological control strategies for mosquito vectors of arboviruses. *Insect* 8(1): 21-28.

Hove-Musekwa, S.D. 2008. Determining effective spraying periods to control malaria via indoor residual spraying in Sub-Saharan Africa. *Journal of Applied Mathematics and Decision Sciences* 2008: 745463.

Lou, Y. & Zhao, X.Q. 2011. Modelling malaria control by introduction of larvivorous fish. *Bulletin of Mathematical Biology* 73(10): 2384-2407.

Menach, A.L., McKenzie, F.E., Flahault, A. & Smith, D.L. 2005. The unexpected importance of mosquitoes oviposition behavior for malaria: Non-productive larval habitats can be sources for malaria transmission. *Malaria Journal* 4(1): e23.

Moore, S.M., Borer, E.T. & Hosseini, P.R. 2010. Predators indirectly control vector borne disease: Linking predator-prey and host-pathogen models. *Journal of the Royal Society Interface* 7(42): 161-176.

Nuraini, N., Tasman, H., Soewono, E. & Sidarto, K.A. 2009. A with-in host dengue infection model with immune response. *Mathematical and Computer Modelling* 49(5-6): 1148-1155.

Nyamah, M.A., Sulaiman, S. & Omar, B. 2011. Field observation on the efficacy of *Toxorhynchites splendens* (wiedemann) as a biocontrol agent against *Aedes albopictus* (sluse) larvae in a cemetery. *Trop. Biomed.* 28(2): 312-319.

Ong, S.Q. 2016. Dengue vector control in Malaysia: A review for current and alternative strategies. *Sains Malaysia* 45(5): 777-785.

Pandey, A., Mubayi, A. & Medlock, J. 2013. Comparing vector-host and SIR models for dengue transmission. *Mathematical Biosciences* 246(2): 252-259.

Rodrigues, H.S., Monteiro, M.T.T., Torres, D.F.M. & Zinober, A. 2012. Dengue disease, basic reproduction number and control. *International Journal of Computer Mathematics* 89(3): 334-346.

Steffan, W.A. & Evenhuis, N.L. 1981. Biology of *Toxorhynchites*. London: Griffin.

Tsai, C.T. & Chin, W.C.B. 2016. Evaluating the role of disease importation in the spatiotemporal transmission of indigenous dengue outbreak. *Applied Geography* 76: 137-146.

Wen, T.H., Tsai, C.T. & Chin, W.C.B. 2016. Evaluating the role of disease importation in the spatiotemporal transmission of indigenous dengue outbreak. *Applied Geography* 76: 137-146.

Yang, H.M. & Ferreira, C.P. 2008. Assessing the effects of vector control on dengue transmission. *Applied Mathematics and Computation* 198: 401-413.

Zaini, Z.I.I., Othman, H., Karim, N., Rashid, N.A.A., Abas, M.B.H., Sahani, M., Hod, R. & Nordin, S.A. 2019. Knowledge and practices regarding *Aedes* control amongst residents of dengue hotspot areas in Selangor: A cross-sectional study. *Sains Malaysia* 48(4): 841-849.
