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Mathematical Model of Dengue Virus with Predator-Prey Interactions

(Model Matematik Virus Denggi dengan Interaksi Pemangsa-Mangsa)

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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 kesimbangan 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)

extended the basic model of Bailey (1975) by testing different vector-control strategies. Mosquito maturation stages and a variable recruitment rates were taken into account. They introduced an efficiency index, defined as the reduction factor of the adult vector population after vector control, to evaluate the impact of control measures. It was found that although all the control policies were efficient to reduce vector population size with efficiency index up to 80%, this trend was not observed in the host population. For the most population, the reduction of dengue cases was less than 40%. Erikson et al. (2010a) then extended the model of Derouich and Boutayeb (2006) by adding the exposed class in human population. Other extensions of the Bailey dengue model includes the model developed by Erikson et al. (2010b) which took into account age structure in the human population. Pandey et al. (2013) developed two mathematical models of dengue transmission. In one of the models, the mosquitoes are explicitly tracked (vector-host dengue model) and the other was without explicit mosquito populations (SIR model). They studied the impact of modeling assumptions on dengue dynamics in Thailand by fitting the DHF data to a simple vector host and SIR models by using Bayesian Markov chain Monte Carlo estimation. They showed that the model selection of SIR model was superior to the vector–host model for the DHF data from Thailand. To the best of our knowledge, the aforementioned studies did not assess the impact of any control strategies to control the dengue epidemic.

Biological approaches are also being considered as an alternative method to control mosquito populations (Nyamah et al. 2011). For example, the use of *Toxorhynchites splendens (Tx. splendens)* species mosquitoes predatory in controlling *Aedes aegypti* at the larval stage of mosquito populations (Huang et al. 2017). Moreover, the use of larvivorous *Tx. splendens* mosquitoes is an environment-friendly method to control the mosquito larvae (Nyamah et al. 2011; Steffan & Evenhuis 1981). Besides, *Aedes aegypti* prey is associated with the presence of *Tx. splendens* predator because both species share the same breeding habitat. *Tx. splendens* predator larvae frequently coexist together with other species of mosquito larvae in the same habitat (Zuharah et al. 2015). Ali et al. (2015) developed a mathematical model of *Tx. splendens* mosquitoes as a biological control strategy to reduce dengue disease propagation, modified from the model of Rodrigues et al. (2012). They take account the aquatic stage of *Aedes aegypti* mosquitoes but did not observe *Tx. splendens* mosquitoes as biological control strategy at the aquatic stage. Moreover, the link between these two species of mosquitoes was not mentioned in the model. They did not consider the larval stage of both mosquitoes species.

Moore et al. (2010) derived the first mathematical model that linked the predator-prey and host-pathogen theory. The work aimed at investigating the indirect consequence of predators on vector-pathogen dynamics. 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 *Tx. 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.

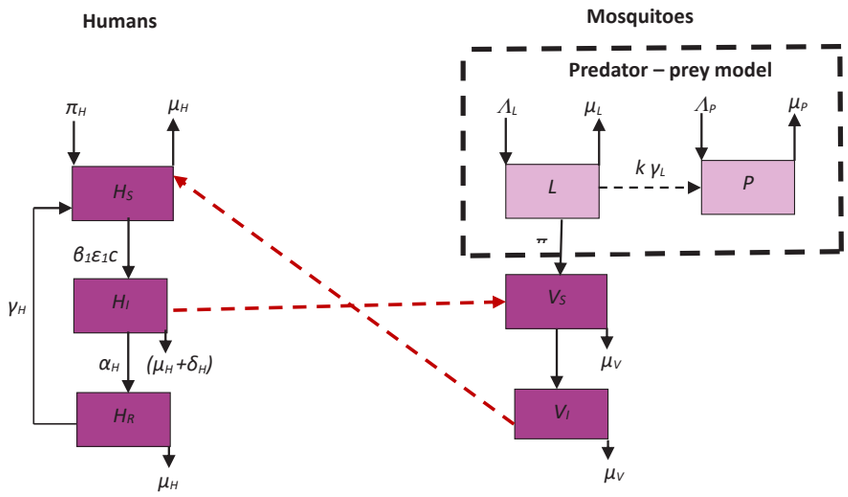

FIGURE 1. Schematic diagram of the model

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,

$$H = H_S + H_I + H_R \tag{1}$$

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,

$$V = V_S + V_I. \tag{2}$$

HUMAN POPULATIONS

The susceptible humans are increased via recruitment of human (by birth or immigration) into the population at a constant rate π_H. They are reduced once the susceptible human acquires the disease after interacting with the infected *Aedes aegypti* mosquito at a rate $\beta_1\varepsilon_1 c$, where β_1 is the transmission probability per bite and ε_1 is the biting rate of mosquitoes, c is the contact rate of mosquito per human per unit time. The recovered human turn out to be susceptible again at a rate γ_H. The susceptible human experiences natural death at a rate μ_H (Ali et al. 2015; Pandey et al. 2013). This gives

$$\frac{dH_S}{dt} = \pi_H + \gamma_H H_R - \beta_1\varepsilon_1 c H_S V_I - \mu_H H_S\,. \tag{3}$$

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 α_H, suffer a disease-induced death at a rate δ_H and natural death at a rate μ_H (Ali et al. 2015; Pandey et al. 2013). Thus,

$$\frac{dH_I}{dt} = \beta_1\varepsilon_1 c H_S V_I - (\alpha_H + \delta_H + \mu_H)H_I\,. \tag{4}$$

Infected human evolves to the recovered class after acquiring immunity at a rate α_H. The recovered human population losses immunity at a rate γ_H and suffers a natural death at a rate μ_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 main contribution of this paper is where the dengue model is extended to the larval stage of predatory mosquitoes. The predatory mosquito here is *Tx. splendens* mosquitoes. It is assumed that the larval stage of predatory mosquitoes P is generated by egg laying rate of the mosquitoes at a rate Λ_P. Predatory mosquito larval stage suffers a natural death at a rate μ_P. The larva matures at a rate π_P and leaves the larval stage to adult mosquito with the assumption that k is the tropical convention efficiency. The adult *Tx. 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 γ_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 Λ_L. Other mosquitoes larvae suffer death due to larvivorous predatory *Tx. splendens* larvae feed on the mosquito larvae at a rate $\gamma_L P$ and a natural death of larvae itself at a rate μ_L . Then, the larvae matures at a rate π_V, leaving the larval stage to adult mosquitoes (Ghosh et al. 2013). Thus,

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

Susceptible mosquitoes are generated via recruitment of *Aedes aegypti* mosquitoes (birth or immigration) at a constant rate π_V. They acquire the infection after the interaction with humans, at a rate $\beta_2 \varepsilon_2 c$, where β_2 is the probability of *Aedes aegypti* mosquitoes getting infected through infected humans, ε_2 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 μ_V (Ali et al. 2015; Pandey et al. 2013). Therefore,

$$\frac{dV_S}{dt} = \pi_V L - \beta_2 \varepsilon_2 c V_S H_I - \mu_V V_S . \tag{8}$$

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:

$$\left.\begin{aligned}
\frac{dH_S}{dt} &= \pi_H + \gamma_H H_R - \beta_1 \varepsilon_1 c H_S V_I - \mu_H H_S \\
\frac{dH_I}{dt} &= \beta_1 \varepsilon_1 c H_S V_I - (\alpha_H + \delta_H + \mu_H) H_I \\
\frac{dH_R}{dt} &= \alpha_H H_I - (\gamma_H + \mu_H) H_R \\
\frac{dP}{dt} &= \Lambda_P + k\gamma_L LP - (\pi_P + \mu_P) P \\
\frac{dL}{dt} &= \Lambda_L - (\gamma_L P + \pi_V + \mu_L) L \\
\frac{dV_S}{dt} &= \pi_V L - \beta_2 \varepsilon_2 c V_S H_I - \mu_V V_S \\
\frac{dV_I}{dt} &= \beta_2 \varepsilon_2 c V_S H_I - \mu_V V_I
\end{aligned}\right\} \tag{9}$$

The model (9) describes the interaction between susceptible, infected and recovered human population in (9_1) - (9_3) with change in time. Similarly, the susceptible and infected mosquito populations interact in (9_6) - (9_7) with change in time.

MODEL ANALYSIS

INVARIANT REGION

Let $(H_S, H_I, H_R, P, L, V_S, V_I) \in \mathbf{R}_+^7$ be any solution of the system with nonnegative initial conditions that is,

$$\frac{dH}{dt} \le \pi_H - \mu_H H \tag{10}$$

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

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

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

$$\Omega_H = \left\{ (H_S, H_I, H_R) \in \mathbf{R}_+^3 : H \le \frac{\pi_H}{\mu_H} \right\} \tag{12}$$

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

$$\Omega_L = \left\{ (P, L) \in \mathbf{R}_+^2 : P \le \frac{\Lambda_P}{\pi_P + \mu_P}, L \le \frac{\Lambda_L}{\pi_V + \mu_L} \right\}, \tag{13}$$

$$\Omega_V = \left\{ (V_S, V_I) \in \mathbf{R}_+^2 : V \le \frac{\pi_V \Lambda_L}{\mu_V (\pi_V + \mu_L)} \right\}. \tag{14}$$

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

$$\Omega = \left\{ \begin{array}{c} (H_S, H_I, H_R, L, P, V_S, V_I) \in \mathbf{R}_+^7 : H_S, H_I, H_R, L, P, V_S, V_I \ge 0; \\ H \le \frac{\pi_H}{\mu_H}, P \le \frac{\Lambda_P}{\pi_P + \mu_P}, L \le \frac{\Lambda_L}{\pi_V + \mu_L}, V \le \frac{\pi_V \Lambda_L}{\mu_V (\pi_V + \mu_L)} \end{array} \right\} \tag{15}$$

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 $\mathbf{R}_+^7$, because the total populations of both human and mosquito are non-negative. Let the initial data be $\{H_S(0), H_I(0), H_R(0), P(0), L(0), V_S(0), V_I(0) \geq 0\} \in \Omega$,

Then, the solution set $\{H_S, H_I, H_R, P, L, V_S, V_I\}(t)$ of the model (9) is positive for all $t > 0$. Based on the model (9) the first equation yields,

$$\left.\begin{aligned} &\frac{dH_S}{dt} = \pi_H + \gamma_H H_R - (\lambda_1 + \mu_H)H_S \geq -(\lambda_1 + \mu_H)H_S \\ &\frac{dH_S}{dt} \geq -(\lambda_1 + \mu_H)H_S \\ &\int \frac{dH_S}{H_S} \geq -\int (\lambda_1 + \mu_H)\,dt \\ &H_S(t) \geq H_S(0)e^{-(\lambda_1+\mu_H)t} \\ &H_S(t) \geq 0. \end{aligned}\right\}. \tag{16}$$

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

$$\left.\begin{aligned} &\frac{dV_I}{dt} = -\lambda_2 V_S - \mu_V V_I \geq -\mu_V V_I \\ &\frac{dV_I}{dt} \geq -\mu_V V_I \\ &\int \frac{dV_I}{V_I} \geq -\int \mu_V V_I dt \\ &V_I(t) \geq V_I(0)e^{-\mu_V V_I t} \\ &V_I(t) \geq 0. \end{aligned}\right\}. \tag{17}$$

As $t \to \infty$, $V_I(t) > 0$. Similarly, it can be shown that H_I, H_R, 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;

$$\left.\begin{aligned} &\pi_H + \gamma_H H_R - \beta_1\varepsilon_1 c_1 H_S V_I - \mu_H H_S = 0 \\ &\beta_1\varepsilon_1 c_1 H_S V_I - (\delta_H + \alpha_H + \mu_H)H_I = 0 \\ &\alpha_H H_I - (\gamma_H + \mu_H)H_R = 0 \\ &\Lambda_P + k\gamma_L LP - (\pi_P + \mu_P)P = 0 \\ &\Lambda_L - (\gamma_L P + \pi_V + \mu_L)L = 0 \\ &\pi_V L - \beta_2\varepsilon_2 c_1 V_S H_I - \mu_V V_S = 0 \\ &\beta_2\varepsilon_2 c_1 V_S H_I - \mu_V V_I = 0 \end{aligned}\right\} \tag{18}$$

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_I, $V_I = 0$, 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_I, $V_I = 0$ 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 $\pi_V L - \mu_V V_S = 0$, implying that $V_S^* = \frac{\pi_V \Lambda_L}{\mu_V(\pi_V + \mu_L)}$. Consequently, the DFE point of the model (9) is attained by putting,

$$\frac{dH_S}{dt} = \frac{dH_I}{dt} = \frac{dH_R}{dt} = \frac{dP}{dt} = \frac{dL}{dt} = \frac{dV_S}{dt} = \frac{dV_I}{dt} = 0. \tag{19}$$

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

$$\begin{aligned} E_0 &= (H_S^*, H_I^*, H_R^*, P^*, L^*, V_S^*, V_I^*), \\ &= \left(\frac{\pi_H}{\mu_H}, 0, 0, \frac{\Lambda_P}{\pi_P + \mu_P}, \frac{\Lambda_L}{\pi_V + \mu_L}, \frac{\pi_V \Lambda_L}{\mu_V(\pi_V + \mu_L)}, 0\right). \end{aligned} \tag{20}$$

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, $\mathfrak{R}_0$

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 $\mathfrak{R}_0 < 1$, each infected individual typically leads to less than one diseased individual and

hence the infection dies out. If $\Re_0 < 1$, 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, considers only the infected compartments H_I, V_I of the model (9). In order to compute the basic reproduction number $\Re_0$, we have

$$\left.\begin{aligned} \frac{dH_I}{dt} &= \lambda_1 H_S - (\delta_H + \mu_H + \alpha_H) H_I \\ \frac{dV_I}{dt} &= \lambda_2 V_S - \mu_V V_I \end{aligned}\right\} \tag{21}$$

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,

$$F_i = \begin{bmatrix} \lambda_1 H_S \\ \lambda_2 V_S \end{bmatrix},$$

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_i) and the removal of individuals in and out of the compartment i and V_i 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

$$F = \begin{bmatrix} 0 & \beta_1 \varepsilon_1 c_1 H_S \\ \beta_2 \varepsilon_2 c_1 V_S & 0 \end{bmatrix},$$

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

Evaluating F at DFE , E_0 yield

$$F = \begin{bmatrix} 0 & \beta_1 \varepsilon_1 c_1 \dfrac{\pi_H}{\mu_H} \\ \beta_2 \varepsilon_2 c_1 \dfrac{\pi_V \Lambda_L}{\mu_V (\pi_V + \mu_L)} & 0 \end{bmatrix},$$

and

$$V^{-1} = \begin{bmatrix} \dfrac{1}{\delta_H + \mu_H + \alpha_H} & 0 \\ 0 & \dfrac{1}{\mu_V} \end{bmatrix}.$$

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

$$\Re_0 = \rho(FV^{-1}) = \frac{\beta_1 \varepsilon_1 c_1^2 \pi_H \beta_2 \varepsilon_2 \pi_V \Lambda_L}{\mu_H (\pi_H + \mu_L)(\delta_H + \mu_H + \alpha_H) \mu_V^2} \tag{22}$$

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 $\Re_0 < 1$ and unstable if $\Re_0 > 1$.

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.

TABLE 1. Description of parameters for model (9)

Parameter	Description	Est. value	References
π_H	Recruitment rate of human	60	Andraud et al. (2012)
π_V	Recruitment rate of *Aedes aegypti* mosquitoes	3000	Andraud et al.(2012)
μ_H	Natural death of human	0.0000457	Al-Sulami et al. (2014)
δ_H	Disease-induced death of human	0.05	Hove-Musekwa et al. (2008)

γ_H	Rate of loss of human immunity	0.02877	Menach et al. (2005)
β_1	Probability of human getting infected	0.75	Al-Sulami et al. (2014)
ε_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)
β_2	Probability of *Aedes aegypti* mosquitoes getting infected	1	Al-Sulami et al. (2014)
ε_2	Biting rate of *Aedes aegypti* mosquitoes	0.5	Al-Sulami et al. (2014)
α_H	Recovery rate of human	0.1428	Al-Sulami et al. (2014)
μ_V	Natural death of *Aedes aegypti* mosquitoes	0.25	Al-Sulami et al. (2014)
Λ_P	Egg laying rate of predatory mosquitoes	50	Ghosh et al. (2013)
Λ_L	Egg laying rate of *Aedes aegypti* mosquitoes	10	Ghosh et al. (2013)
k	Tropical convention efficiency	0.1	Ghosh et al. (2013)
γ_L	Predation of larva stage by predatory mosquitoes	0.1	Ghosh et al. (2013)
π_P	Larva stage mature of predatory mosquitoes	0.0625	Ghosh et al. (2013)
μ_P	Natural death of predatory mosquitoes	0.36	Ghosh et al. (2013)
μ_L	Natural death of *Aedes aegypti* larva	0.05	Ghosh et al. (2013)

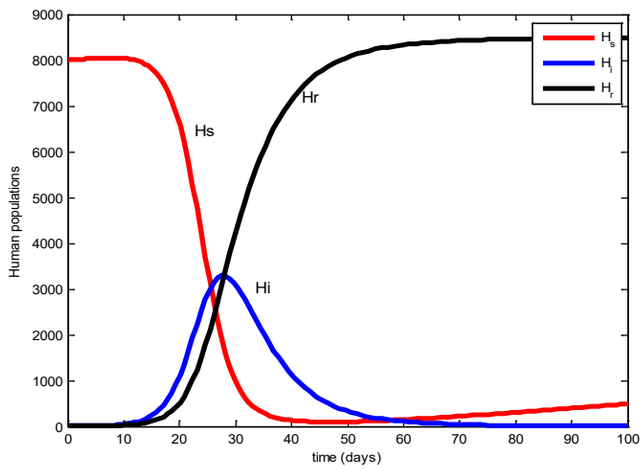

FIGURE 2. The effect of interaction of human populations without predatory mosquitoes with $H_S = 8000$, $H_I = 0$ and $H_R = 0$

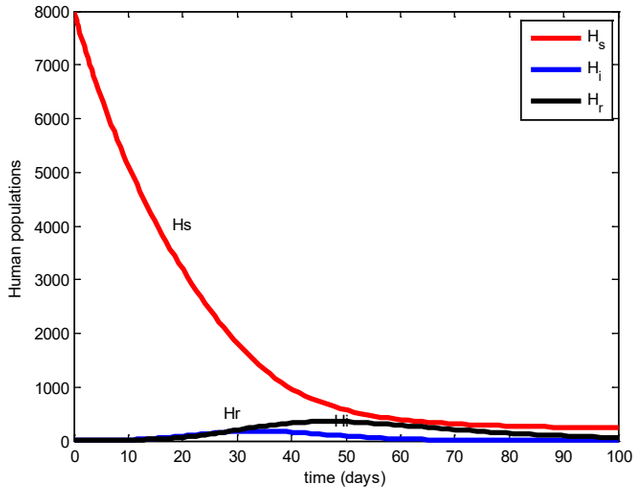

FIGURE 3. The effect of interaction of human populations with predatory mosquitoes with $H_S = 8000$, $H_I = 0$ and $H_R = 0$

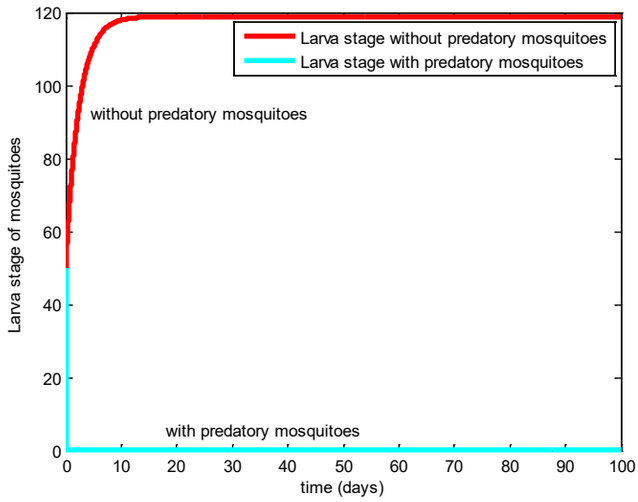

FIGURE 4. . The effect of interaction on larva mosquitoes with and without predatory mosquitoes with $L = 50$ and $P = 50$

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.

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