Original Research Paper

Lyapunov Function for a Dengue Transmission Model where two Species of Mosquitoes are Present: Global Stability

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Abstract: In this study, we are interested in the newly observed endemic due to the ZIKA virus. In the absence of a full knowledge of the dynamics of the infection due to this virus, we consider a model of the closely related dengue virus when there are two species of mosquitoes, *A. aegypti* and *A. albopictus* mosquitoes who are both capable of transmitting the two viruses. We use a Susceptible-Infected-Recovered (SIR) model to describe the infection of the human populations. We obtain the basic reproduction ratio (R_0) and show that if R_0 is less than 1, the disease free equilibrium state is global asymptotically stable. If R_0 is greater than 1, we use the Lyapunov function approach to find the conditions for the unique dengue endemic equilibrium state to be globally stable. We then point out the insights into the global stability of the ZIKA epidemic that can be gained by looking at the global stability of a model for the dengue infection in the presence of two species of mosquitoes that can transmit the disease.

Keywords: Asymptotically Stable, Dengue Disease, Equilibrium State, Global Stability, Species, Lyapunov Function

Introduction

The big scare in the world public health community right now (Janurary 2016) is the ZIKA virus WHO (2016). This virus was discovered in the Zika forest in Uganda, Africa. In the 2007 outbreak in the Federated States of Micronesia, 5000 ZIKA infections were reported in a population of 6700 Peterson *et al*. (2016) Since the virus at the time was thought to cause only a mild febrile fever Simpson (1964), not much attention was paid to the virus. However, in the recent outbreak in Brazil, some of the babies born to pregnant female patients developed microcephaly (a disease which causes the heads of newly born babies to be undersize and the brain to be malformed Mliakar (2016). The possible connection between the ZIKA virus and microcephaly was the reason for World Health Organization (WHO) on Feb. 1, 2016 to declare the possible ZIKA pandemic to be a global health emergency (Science News, 2016). On April 13, 2016, CDC (Center for Disease Control (USA)) announced that the ZIKA virus met the conditions for causality of microcephaly by the ZIKA virus Rasmussen *et al*. (2016). This new complication

arising from this disease led the President (New York Times, 2016) of the USA to ask the US Congress for 1.8 billion dollar to combat this disease and scientists to promise that a vaccine against this disease could be obtained in eighteen months.

The virus belongs to the family *Flavivindae* genius F*lavivirus.* Another member of this family and genius is the dengue virus. The vectors of both of these viruses are mosquitoes of the *Aedes* family, the *A. aegypti* mosquitoes in urban areas Koenraadt *et al*. (2007; Chadee, 2004) and the *A. albopictus* mosquitoes in rural areas Hawley (1988; Mori and Wada, 1972). The (normal) symptoms caused by these two viruses are similar, i.e., mild fever, skin rashes, muscle and joint pains and headaches. In this paper we propose to gain some insights in the spread of the ZIKA virus in Brazil when the presence of both the *A. aegypti and A. albopictus* mosquitoes are taken into account Serpa *et al*. (2013). The reason for taking into account the presence of two species is due to difference in the effects of the different environments in the different parts of Florida, USA on the two species caused the pattern of the

infections to be different in the different parts of Florida Costanzo *et al*. (2005; Juliano *et al*., 2002).

Since a full understanding of the ZIKA epidemic (how it is spread) is not known, we propose to look at the global stability of the solutions of a transmission model of the related disease, dengue fever Keeling and Rohani (2008; Diekmann and Heesterbeek, 2000) in which there are two species of mosquitoes transmitting the virus. Returning to the point raised in the first sentence, in order to model the transmission of a disease, it is necessary to know all the important factors that affect the transmission. As pointed out by Fauci and Morens (2016), the Zika virus is the last of the four recent arthropod-borne viral disease (Zika, dengue, West Nile and Chikungunya) in the Western Hemisphere in past twenty years. The Zika, Dengue and West Nile viruses are flavivi-ruses. The first two are also transmitted by same aedes mosquitoes. Fauci and Morens suggested that one uses common platforms for quick flaviviruses development, i.e., through the expression of immunogenic antigens of the newly emerging virus. For development of the Zika vaccine, this brings up the problem of Antibody-Dependent Enhancement (ADE) since the Zika infections are occurring in areas where the dengue epidemic is also occurring Pierson and Graham (2016). ADE caused by different serotype of the flavivirus causes the viremia and severity of the disease to increase Vaughn *et al*. (2000). It has been shown Priyamvada (2016) that the human antibodies due to dengue fever are highly cross reactive to the ZIKA virus. A *in vitro* study Bardina *et al.* (2017) found that enhancement of ZIKA pathogenesis infections by the preexisting antiflavivirus The ADE in this case was mediated through the IgG engagement of the Fc_y receptors. Another study Littaua *et al*. (1990) found that the ADE leading to dengue hemorrhagic fever was the IgG to Fc receptor II. However, a newer study McCracken *et al*. (2017) using rhesus macaques did indicate that prior infection with a heterologous flaviviruses neither increase or decrease the ZIKA titers in these non human primates, Further investigations are needed to determine whether ADE should be included in a mathematic model for the transmission of the ZIKA disease. If it is, then the susceptible class must be divided into two classes, susceptible who have not been infected with the dengue virus and those who have not. This may present some difficulties since the majority of dengue and ZIKA infected patients are asymptotic cases, i.e., they do not exhibit any clinical symptoms.

Also evidence has been building that sexual transmission of the ZIKA virus is possible Musso *et al*. (2015; D'Ortenzio *et al*., 2016). Sexual transmission by women is possible since the ZIKA virus has been found in the vaginal fluids Turmel *et al*. (2016; Davidson *et al*., 2016). So far, only a few cases of sexual transmission have been reported in the West. There have been no reports of a secondary ZIKA infection resulting from STD ZIKA patient and so it is too early to include this into a disease transmission model.

The present authors have already established the conditions for local asymptotic stability of a dengue model in which two species of mosquitoes are present Sungchasit *et al*. (2013). In that paper, the authors showed that when there were seasonal variations in the rates at which of the dengue infections are transmitted from the two species to the human, limit cycle was seen n the trajectories of the two mosquitoes populations and that the endemic equilibrium state was locally asymptotically stable when the basic reproduction number is greater than one. We use the same parameter values used in that paper which lead to the fraction of *A aegypti* mosquitoes oscillating between 0.261 and 0.04587 and the fraction of *A. albopictus* oscillating between 0.010246 and 00002. In this paper, we wish to establish the global stability of this model using the Lyapunov approach. The aim is to gain insights into the stability of an illness caused by the related ZIKA virus whose nature or means of transmission are not completely known.

Li and Muldowney (1995) have studied the global stability of the solutions of the SEIR (susceptible-exposed-infected-recovered) model commonly used in epidemiological modeling. They showed that the endemic equilibrium which is locally asymptotically stable when the contact number (defined in their paper) σ≤1 and becomes globally asymptotically stable when σ>1. Beretta and Capasso (1986) found the conditions needed for the endemic equilibrium of a SIR model with constant population size to be globally stable. At the present stage of knowledge of the ZIKA epidemic, it is not known if there is an exposed stage in the humans so we will use a simpler model to describe the dynamics of this disease in humans, the SIR model. We will be looking at a similar model except that there will be two species of mosquitoes present. In this paper, we use a generalized Lyapunov function to analysize the global dynamic of the eigth – dimensional model of dengue disease of the human population and different *Aedes* mosquitoes (*Aedes aegypti* and *Aedes albopictus*), where an infected human can be one who was infected by a bite of an *A. aegypti* mosquitoes or by an *A. albopictus* mosquitoes. We will prove the global stability of the equilibrium states using Lyapunov functions LaSalle and Lefschetz (1961). Discussion and conclusion are given in the last section.

Model Formulation and Stability Analysis

We use the Susceptible–Infected–Recovered (SIR) for human and Susceptible–Infected (SI) for both species of the *Aedes* mosquitoes. In fact, there has been no report of which species of the mosquitoes is involved in the transmission of the ZIKA virus. We consider the presence of the two (the *A. aegypti* and *A. albopictus* mosquitoes*)* since both are present in Brazil. One of the reasons for mentioning only the first specie is that the alarm is centered in the urban areas of Brazil. The initial outbreak was in the city Recife in northeastern Brazil. Evidence has been accumulating that *A. albopictus* has adapted itself so that it can thrive in urban environments Benedict *et al*. (2007). The great fear is that it will spread to Rio de Janeiro, the site of the 2016 Olympics where many people from the rest of the world will come. Cancelling the Olympics is out of the question. There are studies which show that both species of the mosquitoes are present in parts of Brazil (Costanzo *et al*., 2005) and if we are to look at the transmission of the diseases in Brazil, we should take into account the presence of both species.

The dengue virus Gubler (1998) is spread by the bite of adult female *Aedes* mosquitoes only, since the female needs the blood to complete the ovigenesis cycle. Only in the viraemic period which ranges from 2 to 12 days with an average of 4-5 days, is an infected person capable of transmitting the virus. During this period, a dengue-infected person is capable of transmitting the dengue virus to the two species (*A. aegypti* and *A. albopictus)* of vectors when they are bitten by the mosquitoes Guga-Sapir and Schimmer (2005). During the blood meal, the females ingest the dengue virus from an infectious human where the virus goes into the gut of the mosquitoes. The dengue virus then undergoes an extrinsic incubation period (of approximately 8-12 days in the *Aedes* mosquitoes) before the mosquitoes become infectious and can transmit the virus to the humans. The presence of the incubation period causes a time which we ignore since it has not been established that this occurs with the ZIKA virus. If the temperature is low enough, the incubationperiod can be longer then the life time of the mosquitoes, in which case no transmission of the virus will occur.

Parameters of Model

Let N_T, N_{va} and N_{vb} represent the total number of human. In this paper, the total number of population is to be constant for each category. The human population of sizes N_T consists of susceptible humans (S), infected humans due to a bite of an *A. aegypti* (I_a) mosquito or of an *A. albopictus* (I_b) mosquito and Recovered Human (R), i.e., $N_T = S + I_a + I_b + R$. For vector population of sizes N_{va}, is total number of an adult *A. aegypti* mosquito

which consist of the susceptible (S_{va}) and infectious mosquitoes (I_{va}), i.e., $N_{vb} = S_{vb} + I_{vb}$ .. N_{vb} is the total number of *A. albopictus* mosquitoes, i.e., $N_{vb} = S_{vb} + I_{vb}$ and S_{vb} is susceptible mosquito, I_{vb} is infectious mosquito. Note that μ_h and μ_d are the average constant natural death rate of human population and death rate of human population due to the disease, μ_{va} and μ_{vb} are the average constant death rate in *A. aegypti* and *A. albopictus* mosquitoes, respectively. As we have mentioned earlier, the seasonal variation is simulated by introducing sinusoidal dependence into the rates of transmission of the virus to the humans and not into the death rates as is done by most researchers. The reason for this was given earlier.

The other parameters are defined as follows: κ is the birth rate of human population, N_h is the total human population at time *t*. ω_a and ω_b are the biting rates of *Aedes aegypti* population and *Aedes albopictus* population, λ_a and λ_b are the measure of influence on the transmission process from human population to *Aedes aegypti* and *Aedes albopictus*, α_{ha} and α_{hb} are the recovery rate of human population who be infected by an *A. aegypti* and *A. albopictus* mosquitoes. It is well known that a person infected with one serotype of the Dengue virus cannot be re infected by the same serotype but can be infected by a different serotype which may result in death causing illness. For vector population, it is assumed a constant recruitment rate Q_a and Q_b of *A. aegypti* and *A. albopictus*, λ_{va} and λ_{vb} are the measure of influence on the transmission process from *A. aegypti* and *A. albopictus*, respectively to human population, β_{va} and β_{vb} are the transmission probability of dengue disease from vector populations (*A. aegypti* and *A. albopictus*) to human population, as well as the number of infectious and susceptible of each species. In reality, the rate at which the adult mosquitoes are recruited depends on how many eggs are laid, on whether the eggs hatch and how many of the larvae survive the metamorphosis process to become adults. For our simple model, we ignore all of this and insert the sinusoidal dependence into the rates of transmission since these depend on the number of bites the mosquitoes must make to obtain the necessary amount of blood needed for the ovigenesis cycle. Our model equations are shown in the next section.

Equations of Model

In this paper, we study the transmission of dengue disease in a human population where there are two species of mosquitoes, *A. aegypti* and *A. albopictus* mosquitoes. We assume that there is only one type of dengue virus present, even though there are four serotypes; DEN1, DEN2, DEN3 and DEN4. Infection by one type confers life-long immunity to further infection by that type.

There is no immunity to infection by the others. However, infection by a different serotype opens up the possibility an allergic reaction to the antibodies of the first type, resulting in a more severe form of the disease, dengue hemorrhagic fever. Figure 1 the monthly incidence of dengue fever in Thailand in the period 2003 to 2011 MOPH (2011). The figure does not show a systematic yearly increase of the disease. The incidences can be larger or smaller year to year. They all show a sinusoidal behavior. We have therefore included sinusoidal dependence in the rates of transmission of the virus to the humans from an infectious mosquito of either species and of the transmission of the virus to either specie of mosquito from an infected human. The rates of transmissions between a human and mosquito usually contains factor giving the number of contacts between the two. If there is no contact, the rates of transmission would be zero. Other studies Fisman (2007; Greenfell *et al.*, 1995) have introduced a sinusoidal dependence in the death rates, reasoning that when the weather gets cold enough, the mosquitoes will die. Our choice of where to introduce the sinusoidal dependence is based on the fact that the *Ades* mosquitoes breeding habits are different from the other mosquito genius. The females of the *Ades* mosquitoes lay their eggs above the water line of the breeding pond. The eggs will hatch only after they are inundated with water from a new rain fall. If there is no new rain fall, there will be more mosquitoes unless humans replenish the water by artificially spraying the area.

The flow chart for the transmission of the dengue virus when there are two species of mosquitoes present is shown in Fig. 2.

The Mathematical Representation of the Flow Chart

The mathematical representation of the model is given by the following system of ordinary differential equations:

$$\frac{dS}{dt} = \kappa N_h - \omega_a \left(1 + \lambda_a \sin \eta t\right) I_{va} S - \mu_d S - \mu_h S - \omega_b \left(1 + \lambda_b \sin \eta t\right) I_{vb} S \tag{1}$$

$$\frac{dI_a}{dt} = \omega_a \left(1 + \lambda_a \sin \eta t\right) I_{va} S - \mu_d I_a - \mu_h I_a - \alpha_{ha} I_a \tag{2}$$

$$\frac{dI_b}{dt} = \omega_b \left(1 + \lambda_b \sin \eta t\right) I_{vb} S - \mu_d I_b - \mu_h I_b - \alpha_{hb} I_b \tag{3}$$

$$\frac{dR}{dt} = -\mu_d R - \mu_h R + \alpha_{ha} I_a + \alpha_{hb} I_b \tag{4}$$

$$\frac{dS_{va}}{dt} = Q_a - \beta_{va} \left(1 + \lambda_{va} \sin \eta t\right) S_{va} I_a - \mu_{va} S_{va} \tag{5}$$

$$\frac{dI_{va}}{dt} = \beta_{va} \left(1 + \lambda_{va} \sin \eta t\right) S_{va} I_a - \mu_{va} I_{va} \tag{6}$$

$$\frac{dS_{vb}}{dt} = Q_b - \beta_{vb} \left(1 + \lambda_{vb} \sin \eta t\right) S_{vb} I_b - \mu_{vb} S_{vb} \tag{7}$$

$$\frac{dI_{vb}}{dt} = \beta_{vb} \left(1 + \lambda_{vb} \sin \eta t\right) S_{vb} I_b - \mu_{vb} I_{vb} \tag{8}$$

All parameters in our model are non-negative. We will now show that the solutions to the equations given by (1) – (8), in the non–negative octant R_+^8 are positive invariant (where R_+^8 denotes the non–negative region). With respect to system (1) – (8), we have the following results.
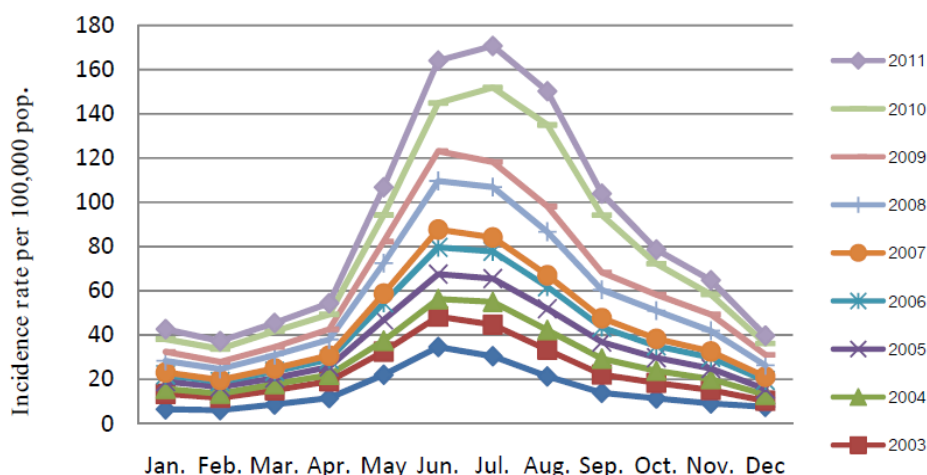

Fig. 1. Reported cases of Dengue disease per 100,000 population in Thailand during 2003 and 2011 (month - by - month) [Division of Epidemiology, 2002-2011]
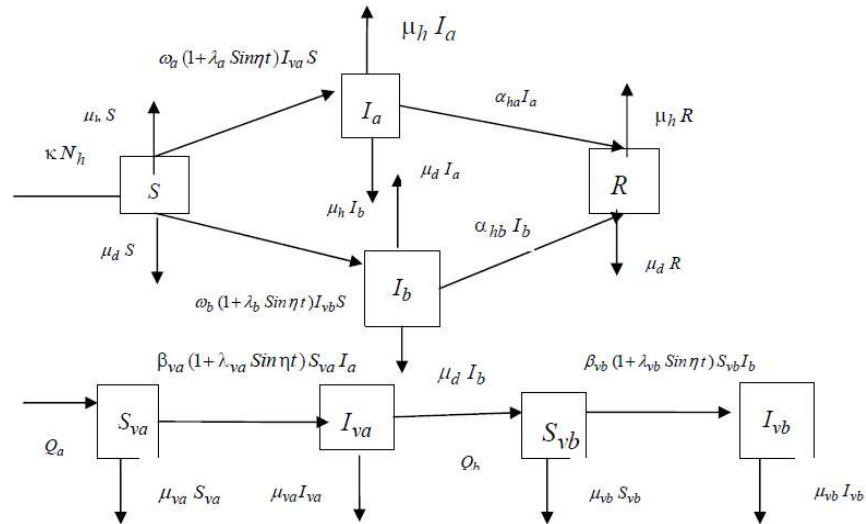
Fig. 2. SIR model

Proposition 1

Let $(S(t)), I_a(t), I_b(t), R(t), S_{va}(t), I_{va}(t), S_{vb}(t), I_{vb}(t)$ be the solution of (1) – (8) with the initial condition $(S(0), I_a(0), I_b(0), R(0), S_{va}(0), I_{vb}(0), S_{vb}(0), I_{vb}(0))$ and the compact set:

$$\Omega_a = \left\{ \begin{array}{l} \left(S, I_a, I_b, R, S_{va}, I_{va}, S_{vb}, I_{vb}\right) \in R_+^8, W_1 \leq N_T \\ = \dfrac{\kappa N_h}{\mu_d + \mu_h}, W_2 \leq N_{va} = \dfrac{Q_a}{\mu_{va}}, W_3 \leq N_{vb} = \dfrac{Q_b}{\mu_{vb}} \end{array} \right\}$$

Then, under the flow described by (1) – (8), Ω_a is a positively invariant set that attracts all solutions in R_+^8.

Proof

We choose the Lyapunov function:

$$W(t) = \left(W_1(t), W_2(t), W_3(t)\right)$$
$$= \left(S + I_a + I_b + R, S_{va} + I_{va}, S_{vb} + I_{vb}\right)$$

to be positive definite on R_+^8 and we have:

$$\frac{dW}{dt} = \left[\frac{dW_1}{dt}, \frac{dW_2}{dt}, \frac{dW_3}{dt} \right]$$
$$= \left[\frac{dS}{dt} + \frac{dI_a}{dt} + \frac{dI_b}{dt} + \frac{dR}{dt}, \frac{dS_{va}}{dt} + \frac{dI_{va}}{dt}, \frac{dS_{vb}}{dt} + \frac{dI_{vb}}{dt} \right]$$
$$= \left(\begin{array}{l} \kappa N_h - \left(\mu_d + \mu_h\right)\left(S + I_a + I_b + R\right), Q_a - \mu_{va}\left(S_{va} + I_{va}\right), \\ Q_b - \mu_{vb}\left(S_{vb} + I_{vb}\right) \end{array} \right)$$
$$= \left(\kappa N_h - \left(\mu_d + \mu_h\right)N_T, Q_a - \mu_{va}N_{va}, Q_b - \mu_{vb}N_{vb} \right)$$

We used the fact that $N_T = \dfrac{\kappa N_h}{\mu_d + \mu_h}, N_{va} = \dfrac{Q_a}{\mu_{va}}$ and

$N_{vb} \dfrac{Q_b}{\mu v_b}$.

With this in mind, it is not difficult to show that:

$$\frac{dW_1}{dt} = \kappa N_h - \left(\mu_d + \mu_h\right)W_1 \leq 0, \quad for\, W_1 \geq \frac{\kappa N_h}{\mu_d + \mu_h} \tag{9}$$

$$\frac{dW_2}{dt} = Q_a - \mu_{va}W_2 \leq 0, \quad for \quad W_2 \geq \frac{Q_a}{\mu_{va}} \tag{10}$$

$$\frac{dW_3}{dt} = Q_b - \mu_{vb}W_3 \leq 0, \quad for \quad W_3 \leq \frac{Q_b}{\mu_{vb}} \tag{11}$$

From the above equations (9) – (11) one has that $\dfrac{Wd}{dt} \leq 0$ which implies that Ω_a is a positively invariant set. In other words, by solving (9) – (11), we obtain:

$$0 \leq \left(W_1(t), W_2(t), W_3(t)\right)$$
$$\leq \left(\begin{array}{l} \left(\kappa N_h / (\mu_d + \mu_h)\right) \\ +W_1(0)e^{-(\mu_d + \mu_h)t}, (Q_a / \mu_{va}) + \\ W_2(0)e^{-\mu_{va}t}, (Q_b / \mu_{vb}) + W(0)e^{-\mu_{vb}t} \end{array} \right),$$

where, $W_1(0), W_2(0)$ and $W_3(0)$ are respectively, the initial conditions of $W_1(t)$, $W_2(t)$ and $W_3(t)$. Thus, as $t \to \infty$, $0 \leq (W_1(t), W_2(t), W_3(t)) \leq (\kappa N_h / \mu_d + \mu_h, Q_a / \mu_{va}, Q_b / \mu_{vb}) = (N_T, N_{va}, N_{vb})$ and one can conclude that Ω_a is an attractive set.

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Equilibrium Points

From equations (1) – (8), we set the right hand side of all equations to zero. We obtain two equilibrium points:

- (I) If $R_0 \le 1$, the only equilibrium is the disease free equilibrium point:

$$J_1\left(S, I_a, I_b, R, S_{va}, I_{va}, S_{vb} I_{vb}\right)$$
$$= J_1 = \left(\frac{\kappa N_h}{\mu_d + \mu_h}, 0, 0, 0, \frac{Q_a}{\mu_{va}}, I_{va}, \frac{Q_b}{\mu_{vb}}, 0\right) \in \Omega_a.$$

- (II) If $R_0 > 1$, there is the endemic equilibrium point:

$$J_2\left(S^*, I_a^*, I_b^*, R^*, S_{va}^*, I_{va}^*, S_{vb}^*, I_{vb}^*\right) \in \Omega_a$$

where, $S^*, I_a^*, I_b^*, R^*, S_{va}^*, I_{va}^*, S_{vb}^*, I_{vb}^* > 0$ satisfy:

$$S^* = \frac{\left(\mu_{va}\left(I_{a\gamma AA}^* + \mu_{va}\right)\mu_{vb}\left(N_{h\gamma BB}\kappa + \left(\alpha_{hb} + \mu_d + \mu_h\right)\mu_{vb}\right)\right)}{\begin{pmatrix}\gamma_{BB}\mu_{va}^2\left(\gamma_{HB}Q_b + \left(\mu_d + \mu_h\right)\mu_{vb}\right) + I_{a\gamma AA\gamma BB}^* \\ \left(\gamma_{HB}Q_b\mu_{vb} + \gamma_{HA}Q_b\mu_{vb} + \left(\mu_d + \mu_h\right)\mu_{va}\mu_{vb}\right)\end{pmatrix}} \quad (12)$$

$$I_b^* = \frac{\begin{pmatrix} N_h\gamma_{BB}\gamma_{HB}Q_b\kappa\mu_{va}(I_a^*\gamma_{AA} + \mu_{va}) \\ -(\alpha_{hb} + \mu_d + \mu_h) \\ \left((\mu_d + \mu_h)\mu_{va}^2 + I_a^*\gamma_{AA}\left(\gamma_{HA}Q_a + (\mu_d + \mu_h)\mu_{va}\right)\mu_{vb}^2\right)/ \end{pmatrix}}{\begin{pmatrix} \gamma_{BB}(\alpha_{hb} + \mu_d + \mu_h) \\ \begin{pmatrix} \mu_{va}^2(\gamma_{BH}Q_b + (\mu_d + \mu_h)\mu_{vb}) \\ + I_a^*\gamma_{AA}(\gamma_{BH}Q_b\mu_{va} + \gamma_{BA}Q_a\mu_{vb} + (\mu_d + \mu_h)\mu_{va}\mu_{vb}) \end{pmatrix} \end{pmatrix}} \quad (13)$$

$$R^* = \left\{ \begin{matrix} \alpha_{ha}\begin{pmatrix} -\gamma_{BB}\gamma_{HB}Q_b(\alpha_{ha} + \mu_d + \mu_h)\mu_{va} - \gamma_{BB} \\ \left(N_h\gamma_{AA}\gamma_{HA}Q_a\kappa + (\mu_d + \mu_h)(\alpha_{hb} + \mu_d + \mu_h)\mu_{vb}^2\right) \\ \mu_{vb} + \gamma_{AA}\gamma_{HA}Q_a(\alpha_{hb} + \mu_d + \mu_h)\mu_{vb}^2\end{pmatrix} \\ / \begin{pmatrix} \gamma_{AA}(\alpha_{ha} + \mu_d + \mu_h)\begin{pmatrix}\gamma_{HB}Q_b\mu_{va} + \gamma_{HA}Q_a\mu_{vb} + \\ (\mu_d + \mu_h)\mu_{va}\mu_{vb}\end{pmatrix} + \\ \begin{pmatrix}\alpha_{hb}\begin{pmatrix}N_h\gamma_{BB}\gamma_{HB}Q_b\kappa\mu_{va}(I_a^*\gamma_{AA} + \mu_{va}) - (\alpha_{hb} + \mu_d + \mu_h) \\ \left((\mu_d + \mu_h)\mu_{va}^2 + I_a^*\gamma_{AA}(\gamma_{HA}Q_a + (\mu_d + \mu_h)\mu_{va})\right)\end{pmatrix}\mu_{vb}^2\end{pmatrix} \\ /\begin{pmatrix}(\alpha_{hb} + \mu_d + \mu_h)(\mu_{va}^2(\gamma_{HB}Q_b + (\mu_d + \mu_h)\mu_{vb}) + \\ I_a^*\gamma_{AA}(\gamma_{HB}Q_b\mu_{va} + \gamma_{HA}Q_a\mu_{vb}) + (\mu_d + \mu_h)\mu_{va}\mu_{vb}\end{pmatrix}\end{pmatrix} \\ /\{\gamma_{BB}(\mu_d + \mu_h)\} \end{matrix} \right\} \quad (14)$$

$$S_{va}^* = \frac{Q_a}{I_a^*\gamma_{AA} + \mu_{va}} \quad (15)$$

$$I_{va}^* = \frac{I_a^*\gamma_{AA}Q_a}{I_a^*\gamma_{AA}\mu_{va} + \mu_{va}^2} \quad (16)$$

$$S_{vb}^* = \frac{\begin{pmatrix}(\alpha_{hb} + \mu_d + \mu_h) \\ \begin{pmatrix}\mu_{va}^2\left(\gamma_{HB}Q_b + (\mu_d + \mu_h)\mu_{vb}\right) + I_a^*\gamma_{AA} \\ \left(\gamma_{HB}Q_b\mu_{va} + \gamma_{HA}Q_a\mu_{vb}(\mu_d + \mu_h)\mu_{va}\mu_{vb}\right)\end{pmatrix}\end{pmatrix}}{\left(\gamma_{HB}\mu_{va}(I_a^*\gamma_{AA} + \mu_{va})\left(N_h\gamma_{BB}\kappa + (\alpha_{hb} + \mu_d + \mu_h)\mu_{vb}\right)\right)} \quad (17)$$

$$I_{vb}^* = \frac{\begin{pmatrix}N_h\gamma_{BB}\gamma_{HB}Q_b\kappa\mu_{va}(I_a^*\gamma_{AA} + \mu_{va}) \\ -(\alpha_{hb} + \mu_d + \mu_h)\begin{pmatrix}(\mu_d + \mu_h)\mu_{va}^2 \\ + I_a^*\gamma_{AA}\left(\gamma_{HA}Q_a + (\mu_d + \mu_h)\mu_{va}\right)\end{pmatrix}\mu_{vb}^2\end{pmatrix}}{\left(\gamma_{HB}\mu_{va}(I_a^*\gamma_{AA} + \mu_{va})\mu_{vb}\left(N_h\gamma_{BB}\kappa + (\alpha_{hb} + \mu_d + \mu_h)\mu_{vb}\right)\right)} \quad (18)$$

$$I_a^* = \frac{\begin{pmatrix}-\gamma_{BB}\gamma_{HB}Q_b(\alpha_{ha} + \mu_d + \mu_h)\mu_{va}^2 - \gamma_{BB} \\ \begin{pmatrix}-N_h\gamma_{AA}\gamma_{HA}Q_a\kappa + (\mu_d + \mu_h)(\alpha_{ha} + \mu_d + \mu_h)\mu_{va}^2\end{pmatrix} \\ \mu_{vb} + \gamma_{AA}\gamma_{HA}Q_a(\alpha_{hb} + \mu_d + \mu_h)\mu_{vb}^2\end{pmatrix}}{\left(\gamma_{AA}\gamma_{BB}(\alpha_{ha} + \mu_d + \mu_h)\begin{pmatrix}\gamma_{HB}Q_b\mu_{va} + \gamma_{HA}Q_a\mu_{vb} \\ + (\mu_d + \mu_h)\mu_{va})\mu_{vb}\end{pmatrix}\right)} \quad (19)$$

And:

$$\gamma_{HA} = \omega_a\left(1 + \lambda_a(\sin \eta t)\right), \quad \gamma_{HB} = \omega_b\left(1 + \lambda_b(\sin \eta t)\right)$$
$$\gamma_{AA} = \beta_{va}\left(1 + \lambda_{va}(\sin \eta t)\right), \quad \gamma_{BB} = \beta_{vb}\left(1 + \lambda_{vb}(\sin \eta t)\right)$$

The basic reproduction number for system (1) – (8), is given by:

$$R_0 = \frac{\gamma_{AA}\gamma_{HA}Q_a\mu_{va}\left(N_h\gamma_{BB}\kappa + (\alpha_{hb} + \mu_d + \mu_h)\mu_{vb}\right)}{\gamma_{BB}(\alpha_{hb} + \mu_d + \mu_h)\mu_{va}^2\left(\gamma_{HB}Q_b + (\mu_d + \mu_h)\mu_{vb}\right)}$$

Global Stability of the Disease Free Equilibrium

We study the global behavior of the disease free equilibrium state for the system defined by equations (1) – (8). We define new time dependent components of the death rate of the mosquitoes infected with the virus as:

$$\begin{cases} \left(\mu_{va}\right)(t) &= \omega_a\left(1 + \lambda_a\sin\eta t\right)S^*, \\ \left(\mu_{vb}\right)(t) &= \omega_b\left(1 + \lambda_b\sin\eta t\right)S^*, \\ \left(\mu_d + \mu_h\right)(t) &= \begin{matrix}\beta_{va}\left(1 + \lambda_{va}\sin\eta t\right)S_{va}^*, \\ \beta_{vb}\left(1 + \lambda_{vb}\sin\eta t\right)S_{vb}^*\end{matrix}\end{cases} \quad (20)$$

Then Equations (1), (5) and (7) reduce to:

$$\frac{dS}{dt} = \kappa N_h - \mu_h{}'(t)S \text{ where } \mu_h{}'(t)$$
$$= \mu_h + \mu_a + \omega_{v_a}(1 + \lambda_a\sin\eta t)I_{va} + \omega_{v_b}(1 + \lambda_b\sin\eta t)I_{vb} \quad (1')$$

$$\frac{dS_{va}}{dt} = Q_a - \mu'_{va}(t)s_{va} \quad where \ \mu'_{va}(t)$$
$$= \mu_{va} + \beta_{va}\left(1 + \lambda_{va}\sin\eta t\right)I_a \tag{5'}$$

And:

$$\frac{dS_{vb}}{dt} = Q_b - \mu'_{vb}(t)S_{va} \quad where \ \mu'_{va}(t)$$
$$= \mu_{va} + \beta_{va}(1 + \lambda_{va}\sin\eta t)I_a \tag{7'}$$

with the rest of the equations remaining the same.

Theorem 1

When $R_0 \leq 1$, the disease free equilibrium J_1 is globally asymptotically stable on Ω_a.

Proof

Working now with the new set of equations to construct the Lyapunov function on Ω_a., we get:

$$\psi(t) = (S - S^*\ln S) + I_a + I_b + R$$
$$+ (S_{va} - S^*_{va}\ln S_{va}) + I_{va} + (S_{vb} - S^*_{vb}\ln S_{vb})I_{vb}$$

The derivative with respect to time yields:

$$\frac{d\psi(t)}{dt} = \frac{dS}{dt}\left(1 - \frac{S^*}{S}\right) + \frac{dI_a}{dt} + \frac{dI_b}{dt} + \frac{dR}{dt} + \frac{dS_{va}}{dt}\left(1 - \frac{S^*_{va}}{S_{va}}\right)$$
$$+ \frac{dI_{va}}{dt} + \frac{dS_{vb}}{dt}\left(1 - \frac{S^*_{vb}}{S_{vb}}\right) + \frac{dI_{vb}}{dt}$$

$$\frac{d\psi(t)}{dt} = \left(\begin{array}{c} \kappa N_h - \omega_a(1 + \lambda_a \sin\eta t)I_{va}S - \mu_d S \\ - \mu_h S - \omega_b(1 + \lambda_b \sin\eta t)I_{vb}S \end{array}\right)\left(1 - \frac{S^*}{S}\right)$$
$$+ \left(\omega_a(1 + \lambda_a \sin\eta t)I_{va}S - \mu_d I_a - \mu_h I_a - \alpha_{ha}I_a\right)$$
$$+ \left(\omega_b(1 + \lambda_b \sin\eta t)I_{vb}S - \mu_d I_b - \mu_h I_b - \alpha_{hb}I_b\right)$$
$$+ \left(-\mu_d R - \mu_h R - \alpha_{ha}I_a - \alpha_{hb}I_b\right)$$
$$+ \left(Q_a - \beta_{va}\left(1 + \lambda_{va}\sin\eta t\right)S_{va}I_a - \mu_{va}S_{va}\right)\left(1 - \frac{S^*_{va}}{S_{va}}\right)$$
$$+ \left(\beta_{va}(1 + \lambda_{va}\sin\eta t)S_{va}I_a - \mu_{va}I_{va}\right)$$
$$+ \left(Q_b - \beta_{vb}(1 + \lambda_{vb}\sin\eta t)S_{vb}I_b - \mu_{vb}S_{vb}\right)\left(1 - \frac{S^*_{vb}}{S_{vb}}\right)$$
$$+ \left(\beta_{vb}(1 + \lambda_{vb}\sin\eta t)S_{vb}I_b - \mu_{vb}I_{vb}\right)$$

Rearranging the terms in the above expression, we get:

$$\frac{d\psi(t)}{dt} = \kappa N_h\left(1 - \frac{S^*}{S}\right) + Q_a\left(1 - \frac{S^*_{va}}{S_{va}}\right) + Q_b\left(1 - \frac{S^*_{vb}}{S_{vb}}\right)$$
$$+ I_{va}\left(\omega_a(1 + \lambda_a \sin\eta t)S^* - \mu_{va}\right)$$
$$+ I_{vb}\left(\omega_b(1 + \lambda_b \sin\eta t)S^* - \mu_{vb}\right)$$

$$+ I_a\left(\beta_{va}(1 + \lambda_{va}\sin\eta t)S^*_{va} - (\mu_d + \mu_h)\right)$$
$$+ \left(\beta_{vb}(1 + \lambda_{vb}\sin\eta t)S^*_{vb} - (\mu_d + \mu_h)\right)$$
$$+ \mu_h S^*\left(1 - \frac{S}{S^*}\right) + \mu_d S^*\left(1 - \frac{S}{S^*}\right) \tag{21}$$
$$+ \mu_{va}S^*_{va}\left(1 - \frac{S_{va}}{S^*_{va}}\right) + \mu_{vb}S^*_{vb}\left(1 - \frac{S_{vb}}{S^*_{vb}}\right) - \mu_d R - \mu_h R$$

Using the new equilibrium states obtained from Equation (1'), (5") and (7') $S^* = \dfrac{\kappa N_h}{\mu_d + \mu_h}, S^*_{va} = \dfrac{Q_a}{\mu_{va}}$ and $S^*_{vb} = \dfrac{Q_b}{\mu_{vb}}$, expression becomes:

$$\frac{d\psi(t)}{dt} = \kappa N_h\left(1 - \frac{S^*}{S}\right) + Q_a\left(1 - \frac{S^*_{va}}{S_{va}}\right) + Q_b\left(1 - \frac{S^*_{vb}}{S_{vb}}\right)$$
$$+ \mu_h S^*\left(1 - \frac{S}{S^*}\right) + \mu_d S^*\left(1 - \frac{S}{S^*}\right) \tag{22}$$
$$+ \mu_{va}(t)S^*_{va}\left(1 - \frac{S_{va}}{S^*_{va}}\right) + \mu_{vb}(t)S^*_{vb}\left(1 - \frac{S_{vb}}{S^*_{vb}}\right)$$
$$- \mu_d R - \mu_h R$$

$$\frac{d\psi(t)}{dt} = \kappa N_h\left(2 - \frac{S^*}{S} - \frac{S}{S^*}\right) + Q_a\left(2 - \frac{S^*_{va}}{S_{va}} - \frac{S_{va}}{S^*_{va}}\right)$$
$$+ Q_b\left(2 - \frac{S^*_{vb}}{S_{vb}} - \frac{S_{vb}}{S^*_{vb}}\right) - \mu_d R - \mu_h R$$

$$\frac{d\psi(t)}{dt} = -\kappa N_h\frac{(S^* - S)^2}{S^*S} - Q_a\frac{(S^*_{va} - S_{va})^2}{S^*_{va}S_{va}} \tag{23}$$
$$- Q_b\frac{(S^*_{vb} - S_{vb})^2}{S^*_{vb}S_{vb}} - \mu_d R - \mu_h R$$

We can see that all terms in (23) are always non-positive. Using the LaSalle's extension to Lyapunov's theorem, we have $\dfrac{d\psi(t)}{dt} \leq 0$ and so the function $\dfrac{d\psi(t)}{dt}$ is seen to be negative definite. The limit set of each solution is contained in the largest invariant set for which $S = S^*$, $S_{va} = S^*_{va}$, $S_{vb} = S^*_{vb}$ and $R^n = 0$ which is the singleton $\{J_1\}$. Then, the LaSalle's invariant principle implies that the disease free equilibrium J_1 is globally asymptotically stable on Ω_a.

Next, we consider the global property of the endemic equilibrium of (1)-(8).

Theorem 2

If $R_0 > 1$, the endemic equilibrium state $J_2\left(S^*, I_a^*, I_b^*, R^*, S^*_{va}, I^*_{va}, S^*_{vb}, I^*_{vb}\right) \in \Omega_a$ exists and is globally asymptotically stable on Ω_a. To see this, we now

redefine the time dependent components of death rates, Equation (20), as:

$$
\begin{cases}
\mu_{va}(t) = \left(\omega_a(1+\lambda_a\sin\eta t)N_h\right) \\
\mu_{vb}(t) = \left(\omega_b(1+\lambda_a\sin\eta t)N_h\right) \\
(\mu_d+\mu_h+\alpha_{ha})(t) = Q\beta_{va}(1+\lambda_{va}\sin\eta t)S_{va}^* \\
(\mu_d+\mu_h+\alpha_{hb})(t) = Q\beta_{vb}(1+\lambda_{vb}\sin\eta t)S_{vb}^*
\end{cases}
\tag{24}
$$

Proof

The Lyapunov function is in the form:

$$
\begin{aligned}
\rho(t) =\;& (S - S^* InS) + I_a + I_b \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\left(S_{va}-S_{va}^* InS_{va}\right) \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right) I_{va} \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\left(S_{vb}-S_{vb}^* InS_{vb}\right) \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right) I_{vb}
\end{aligned}
\tag{25}
$$

With:

$$
\begin{aligned}
A_1(t) &= \omega_a(1+\lambda_a\sin\eta t) \\
A_2(t) &= \omega_b(1+\lambda_b\sin\eta t)
\end{aligned}
$$

Its derivative along the trajectories of (1)-(8):

$$
\begin{aligned}
\frac{d\rho(t)}{dt} =\;& \frac{dS}{dt}\left(1-\frac{S^*}{S}\right) + \frac{dI_a}{dt} + \frac{dI_b}{dt} \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right) \\
&\frac{dS_{va}}{dt}\left(1-\frac{S_{va}^*}{S_{va}}\right) + \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\frac{dI_{va}}{dt} \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\frac{dS_{vb}}{dt}\left(1-\frac{S_{vb}^*}{S_{vb}}\right) \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\frac{dI_{vb}}{dt}
\end{aligned}
\tag{26}
$$

$$
\begin{aligned}
\frac{d\rho(t)}{dt} =\;& \left(\begin{array}{c}\kappa N_h - \omega_a(1+\lambda_a\sin\eta t)I_{va}S \\ -\mu_d S - \mu_h S - \omega_b(1+\lambda_b\sin\eta t)I_{vb}S\end{array}\right)\left(1-\frac{S^*}{S}\right) \\
&+ \left(\omega_a(1+\lambda_a\sin\eta t)I_{va}S - \mu_d I_a - \mu_h I_a - \alpha_{ha}I_a\right) \\
&+ \left(\omega_b(1+\lambda_b\sin\eta t)I_{vb}S - \mu_d I_b - \mu_h I_b - \alpha_{hb}I_b\right) \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\left(\begin{array}{c}Q_a - \beta_{va}(1+\lambda_{va}\sin\eta t) \\ S_{va}I_a - \mu_{va}S_{va}\end{array}\right)\left(1-\frac{S_{va}^*}{S_{va}}\right)
\end{aligned}
$$

$$
\begin{aligned}
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\left(\beta_{va}(1+\lambda_{va}\sin\eta t)S_{va}I_a - \mu_{va}I_{va}\right) \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\left(\begin{array}{c}Q_b - \beta_{vb}(1+\lambda_{vb}\sin\eta t) \\ S_{vb}I_b - \mu_{vb}S_{vb}\end{array}\right)\left(1-\frac{S_{vb}^*}{S_{vb}}\right) \\
&+ \left(\frac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)\left(\begin{array}{c}\beta_{vb}(1+\lambda_{vb}\sin\eta t) \\ S_{vb}I_b - \mu_{vb}I_{vb}\end{array}\right)
\end{aligned}
\tag{27}
$$

Since we assume that total number of populations are constants, so we have $\kappa N_h = N_T(\mu_d + \mu_h)$, $Q_a = N_{va}\mu_{va} = \mu_{va}(S_{va} + I_{va})$ and $Q_b = N_{vb}\mu_{vb} = \mu_{vb}(S_{vb} + I_{vb})$. Then above equation become:

$$
\begin{aligned}
\frac{d\rho(t)}{dt} =\;& (\mu_d+\mu_h)N_T\left(1-\frac{S^*}{S}\right) \\
&+ Q\mu_{va}N_{va}\left(1-\frac{S_{va}^*}{S_{va}}\right) + Q\mu_{vb}N_{vb}\left(1-\frac{S_{vb}^*}{S_{vb}}\right) \\
&+ I_{va}\left(\omega_a(1+\lambda_a\sin\eta t)N_h\right) - Q\mu_{va}\frac{S_{va}^*}{S_{va}} \\
&+ I_{vb}\left(\omega_b(1+\lambda_b\sin\eta t)N_h\right) - Q\mu_{vb}\frac{S_{vb}^*}{S_{vb}} \\
&+ I_a\left(Q\beta_{va}(1+\lambda_{va}\sin\eta t)S_{va}^* - (\mu_d+\mu_h+\alpha_{ha})\right) \\
&+ I_b\left(Q\beta_{vb}(1+\lambda_{vb}\sin\eta t)S_{vb}^* - (\mu_d+\mu_h+\alpha_{hb})\right)
\end{aligned}
\tag{28}
$$

when $Q = \left(\dfrac{\mu_d+\mu_h+\alpha_{ha}+\alpha_{hb}}{A_1 S_{va}^* + A_2 S_{vb}^*}\right)$.

Substituting conditions (24) into (28), we have:

$$
\begin{aligned}
\frac{d\rho(t)}{dt} =\;& (\mu_d+\mu_h)N_T\left(1-\frac{S^*}{S}\right) \\
&+ Q\mu_{va}S_{va}\left(1-\frac{S_{va}^*}{S_{va}}\right) + Q_1\left(1-\frac{S_{va}^*}{S_{va}}-\frac{S_{va}}{S_{va}^*}\right) \\
&+ QS_{vb}\mu_{vb}\left(1-\frac{S_{vb}^*}{S_{vb}}\right) + Q_2\left(1-\frac{S_{vb}^*}{S_{vb}}-\frac{S_{vb}}{S_{vb}^*}\right) \\
&+ I_{va}\left((\omega_a(1+\lambda_a\sin\eta t)N_h) - \mu_{va}\right) \\
&+ I_{vb}\left((\omega_b(1+\lambda_b\sin\eta t)N_h) - \mu_{vb}\right) \\
&+ I_a Q\beta_{va}(1+\lambda_{va}\sin\eta t)S_{va}^* - (\mu_d+\mu_h+\alpha_{ha})I_a \\
&+ I_b Q\beta_{vb}(1+\lambda_{vb}\sin\eta t)S_{vb}^* - (\mu_d+\mu_h+\alpha_{hb})I_b
\end{aligned}
\tag{29}
$$

In Ω_a, we have $Q_1 = I_{va}Q\mu_{va}$ and $Q_2 = I_{vb}Q\mu_{vb}$. The above equation (29) becomes:

$$
\begin{aligned}
\frac{d\rho(t)}{dt} =\;& (\mu_d+\mu_h)N_T\left(1-\frac{S^*}{S}\right) \\
&+ Q\mu_{va}(t)S_{va}\left(1-\frac{S_{va}^*}{S_{va}}\right) + Q_1\left(1-\frac{S_{va}^*}{S_{va}}-\frac{S_{va}}{S_{va}^*}\right) \\
&+ QS_{vb}\mu_{vb}(t)\left(1-\frac{S_{vb}^*}{S_{vb}}\right) + Q_2\left(1-\frac{S_{vb}^*}{S_{vb}}-\frac{S_{vb}}{S_{vb}^*}\right)
\end{aligned}
$$

$$+I_{va}\left(\left(\omega_a(1+\lambda_a\sin\eta t)N_h\right)-\mu_{va}(t)\right)$$
$$+I_{vb}\left(\left(\omega_b(1+\lambda_b\sin\eta t)N_h\right)-\mu_{vb}(t)\right) \qquad (30)$$
$$+I_a Q\beta_{va}(1+\lambda_{va}\sin\eta t)S_{va}^* - (\mu_d+\mu_h+\alpha_{ha})(t)I_a$$
$$+I_b Q\beta_{vb}(1+\lambda_{vb}\sin\eta t)S_{vb}^* - (\mu_d+\mu_h+\alpha_{hb})(t)I_b$$

$$\frac{d\rho(t)}{dt} = -(\mu_d+\mu_h)N_T\frac{(S-S^*)^2}{SS^*}$$
$$-Q\mu_{va}(t)S_{va}\frac{(S_{va}-S_{va}^*)^2}{S_{va}S_{va}^*} - Q_1\frac{(S_{va}-S_{va}^*)^2}{S_{va}S_{va}^*} \qquad (31)$$
$$-QS_{vb}\mu_{vb}(t)\frac{(S_{vb}-S_{vb}^*)^2}{S_{vb}S_{vb}^*} - Q_2\frac{(S_{vb}-S_{vb}^*)^2}{S_{vb}S_{vb}^*}$$

$$\frac{d\rho(t)}{dt} = (\mu_d+\mu_h)N_T\left(2-\frac{S^*}{S}-\frac{S}{S^*}\right)$$
$$+Q\mu_{va}(t)S_{va}\left(2-\frac{S_{va}^*}{S_{va}}-\frac{S_{va}}{S_{va}^*}\right)+Q_1\left(2-\frac{S_{va}^*}{S_{va}}-\frac{S_{va}}{S_{va}^*}\right) \qquad (32)$$
$$+QS_{vb}\mu_{vb}(t)\left(2-\frac{S_{vb}^*}{S_{vb}}-\frac{S_{vb}}{S_{vb}^*}\right)+Q_2\left(2-\frac{S_{vb}^*}{S_{vb}}-\frac{S_{vb}}{S_{vb}^*}\right)$$

We use the LaSalle's invariant principle to show that $\frac{d\rho(t)}{dt}\leq0$ for all $(S^*,I_a^*,I_b^*,R^*,S_{va}^*,I_{va}^*,S_{vb}^*,I_{vb}^*)\in\Omega_a$ and the strict equality $\frac{d\rho(t)}{dt}=0$ holds only for $S=S^*,I_a=I_a^*,I_b=I_b^*,R=R^*,S_{va}=S_{va}^*,I_{va}=I_{va}^*,S_{vb}=S_{vb}^*$ and $I_{vb}=I_{vb}^*$. Then, the equilibrium state J_2 is the only invariant set of solutions of the equations (1)–(8) contained entirely in $\left\{\begin{array}{l}(S^*,I_a^*,I_b^*,R^*,S_{va}^*,I_{va}^*,S_{vb}^*,I_{vb}^*),S=S^*,I_a=I_a^*,I_b=I_b^*,R=R^*,\\ S_{va}=S_{va}^*,I_{va}=I_{va}^*,S_{vb}=S_{vb}^* \text{ and } I_{vb}=I_{vb}^*\end{array}\right\}$ and hence the asymptotic stability theorem, the positive endemic equilibrium state J_2 is global asymptotic stability in Ω_a.

Discussion and Conclusion

Let:

$$R_0 = \frac{\gamma_{AA}\gamma_{HA}Q_a\mu_{va}\left(N_h\gamma_{BB}\kappa+(\alpha_{hb}+\mu_d+\mu_h)\mu_{vb}\right)}{\gamma_{BB}(\alpha_{ha}+\mu_d+\mu_h)\mu_{vb}^2\left(\gamma_{HB}Q_b+(\mu_d+\mu_h)\mu_{vb}\right)}$$

be the threshold parameters. Then, we define $R_0=\sqrt{R_0}$ as the basic reproductive number of disease. Also, it represents the average number of secondary cases produced from susceptible population. We consider human and vector (*A. aegypti* and *Aedes albopictus*) populations. It depends on the transmission rate of dengue virus.

In this paper, we have studied a mathematical model of dengue disease in which the virus is being transmitted by two different species of the *Aedes* mosquitoes by looking at the global stability of our model. The global stability of transmission of dengue disease in human and vector (*Aedes aegypti* and *A. albopictus*) was determined been by using Lyapanov functions. If $R_0\leq1$, then to the disease–free equilibrium state is globally asymptotically stable. In the feasible region and the disease dies out of population. If $R_0>1$, then there is the unique endemic equilibrium state which is globally asymptotically stable in the interior of feasible region and the disease is present. If the disease is present in the population, then it will persist. Nothing in the model depends directly on the dengue virus directly except for the use of a sinusoidal variation of the transmission rates which were inferred by the behavior seen in Figure 1. Since the ZIKA epidemic has only been around for less than a year, there has not been enough time to observe what the seasonal dependence is. When that information becomes available, the present work can be modified so that it can be applied to possible ZIKA pandemic which might arise in the near future.

Author's Contributions

Assoc. Prof. Dr. Puntani Pongsumpun: was the advisor of Rattiya Sugchasit who did the work for her Ph.D. degree. She guided the student on a day to day basis and wrote the first draft of the paper with Ms. Rattiya Sungchasit.

Dr. Rattiya Sungchasit: did the work for her Ph.D. which has received. She did the analysis the model and carried out the computer calculations. She is the one who suggested using the Lyapanov function method to establish the global stability of the model.

Prof. Dr. I-Ming Tang: was the person who suggested the problem, the spread of the ZIKA virus and the mathematical model to describe the transmission of this disease and its connection the spread of the dengue disease. He is responsible for the incorporation of the two species of the *Aedes* mosquitoes. The final version of the paper was written by him.

Conflict of Interests

The authors declare that there is no conflict of interest regarding this research.

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