The Dynamical Model of Dengue Vertical Transmission

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Abstract

Dengue disease is usually found in many parts of the world, including Africa, Asia, South America and Australia. Dengue disease can pass from one individual to another by two distinct mechanisms such as horizontal transmission and vertical transmission. In horizontal transmission, susceptible individuals can be infected by direct or indirect contacts with infectious individuals who are stays at the same time. Vertical transmission means to direct transmission from infected parents to their offspring before or during birth. In this study, the dynamical model of dengue disease was formulated by considering the vertical transmission in *Aedes* mosquitoes. The analysis of our model was given. The results of this study should introduce the alternative ways to reduce the dengue outbreak.

Keywords: Aedes mosquitoes, dengue, dynamical model, vertical transmission

1. Introduction

Dengue virus can transmit between people by biting of infected *Aedes* mosquitoes. Dengue disease can be classified as Dengue Fever (DF), Dengue Hemorrhagic Fever (DHF) and Dengue Shock Syndrome (DSS). People who develop DHF have a 5% chance of death. They developed to be DSS where the mortality rate can rise as high as 40%. In 1981, DHF first appeared in the Americas. During this epidemic, there were 344,000 dengue cases. There were 10,300 severe cases and 158 deaths [1]. DF and DHF have increased in both incidence and distribution over the past 40 years. The first recorded outbreak of dengue disease compatible with DHF occurred in Australia in 1897. In 1928, a similar hemorrhagic disease was recorded during an epidemic in Greece and again in Taiwan in 1931. The first confirmed epidemic of DHF was recorded in the Philippines in 1953–1954. Major outbreaks of DHF with significant mortality have occurred in most countries of the South-East Asia Region, including India, Indonesia, Maldives, Myanmar, Sri Lanka, and Thailand, as well as in Singapore, Cambodia, China, Laos, Malaysia, New Caledonia, Palau, Philippines, Tahiti and Vietnam in the Western Pacific Region. *Aedesaegypti* and *Aedesalbopictus* are two species of vectors caused dengue disease. They are originally found in tropical and subtropical zones. There are 4 serotypes of dengue virus such as DEN-1, DEN-2,

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DEN-3 and DEN-4. The dengue virus is passed between human through biting of infected female vector. After an infected female Aedes mosquito acquires the virus from feeding on the blood of an infected person. Within the mosquito, the virus infects the mid-gut and spreads it to the salivary glands over a period of 8-12 days. After this period, the virus can be transmitted to humans during subsequent probing or feeding [2]. Most female Aedesaegypti vectors stay around the houses. They usually fly an average of 400 metres. Efficiency of virus transmission from a mosquito to human is depending on the magnitude and duration of viremia in the human host. A person with high viremia provides a higher infectious dose of virus to feeding mosquito. This normally leads to a greater percentage of feeding mosquitoes becoming infected. The mosquitos may be infected when there is only a very low level of virus in the blood [3, 4]. There are two types of dengue transmission such as horizontally and vertically transmissions. Horizontally transmission means human can be infected by biting of infected female Aedesmosquitoes. It can also be transmitted between the vector from an infected female to its offspring, called as vertically transmission [5]. There are many nice problems lending themselves to mathematical modeling [6]. Modelers are planners, schemers, and contrivers [7]. Mathematical model is another way of looking at biological systems and at medical phenomena. There are many advantages to this approach. Mathematics can help to understand the process of modeling makes us concentrate on separating the essential from the inessential. A model can be used for predicting situations that can not be easily done by experiment [8].

Mathematical models for dengue transmission are studied long time ago. In 1998, Esteva and Vargas formulated the mathematical model of dengue disease by assuming the constant total number of human and vector populations. Standard dynamical modeling is used to analyze their model. The controls of the vector population are discussed in terms of the threshold condition, which governs the existence and stability of the endemic equilibrium. After that, Esteva and Vargas [9] constructed the mathematical model for dengue virus infection with variable human population size. They found three threshold parameters which govern the existence of the proportion of endemic equilibrium point, the increase of human population size, and the behavior of total number of infectious human. There are many papers studied about the dynamical models of dengue disease [10-12]. In 2015, we studied the transmission model of dengue disease with two species of mosquitoes and age structure of human population [13]. Most of the research on the dynamical models of dengue disease considered the horizontal transmission of the disease. The transmission of dengue disease can be occurred by vertically transmission. The possibility of vertical transmission has known over the last 30 years [14]. In this paper, we study the dynamical model of dengue disease by considering the vertical transmission of the disease. Our model is analyzed by using standard dynamical modeling method.

2. Dynamical Model

In this paper, we study the transmission of dengue disease by formulating the mathematical model. The transmission of dengue disease can occur between human and vector population. Therefore, we formulate the dynamical model by considering human and vector population. The human population is separating into Susceptible, Exposed, Infectious and Recovered classes. The vector population is divided into Susceptible, Exposed and Infectious classes. We suppose the human and vector population have constant sizes. Vertical transmission of dengue virus is considered in this model. The diagram of our model can be described in Figure 1.

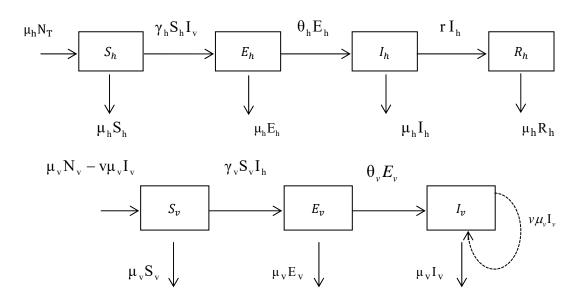


Figure 1. Diagram of our model.

The variables and parameters of our model are defined as in Table1.

Table 1. The definitions of variables and parameters:

variables/parameters	definitions
S _h (t)	Number of susceptible human at time t
$E_{h}(t)$	Number of exposed human at time t
I _h (t)	Number of infectious human at time t
R _h (t)	Number of recovered human at time t
S _v (t)	Number of susceptible vector population at time t
$E_{v}(t)$	Number of exposed vector population at time t
$I_{v}(t)$	Number of infectious vector population at time t
$\mu_{ m h}$	Birth and death rate of human population
N_{T}	Total human population
$\gamma_{\rm h}$	The transmission rate of dengue virus from vector to human population
θ_{h}	The incubation rate of dengue virus in human population
r	The recovery rate of human population
μ_{v}	The birth rate and death rate of vector population
N _v	The total vector population
$\gamma_{\rm v}$	The transmission rate of dengue virus from human to vector population
$\theta_{ m v}$	The incubation rate of dengue virus in vector population
V	Fraction of vector infected by vertical transmission

Dynamical equations of our model can be shown as follows: For human population:

$$\frac{d}{dt}S_{h}(t) = \mu_{h}N_{T} - \gamma_{h}S_{h}(t)I_{v}(t) - \mu_{h}S_{h}(t)$$
(1)

$$\frac{\mathrm{d}}{\mathrm{d}t} E_{h}(t) = \gamma_{h} S_{h}(t) I_{v}(t) - (\theta_{h} + \mu_{h}) E_{h}(t) \tag{2}$$

$$\frac{d}{dt}I_h(t) = \theta_h E_h - (\mu_h + r)I_h(t) \tag{3}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}R_{\mathrm{h}}(t) = r\,I_{\mathrm{h}} - \mu_{\mathrm{h}}R_{\mathrm{h}}(t) \tag{4}$$

For vector population:

$$\frac{d}{dt}S_{v}(t) = \mu_{v}N_{v} - v\mu_{v}I_{v}(t) - \gamma_{v}s_{v}(t)I_{h}(t) - \mu_{v}S_{v}(t)$$
(5)

$$\frac{\mathrm{d}}{\mathrm{d}t} E_{\mathrm{v}}(t) = \gamma_{\mathrm{v}} s_{\mathrm{v}}(t) I_{\mathrm{h}}(t) - (\theta_{\mathrm{v}} + \mu_{\mathrm{v}}) E_{\mathrm{v}}(t) \tag{6}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}I_{v}(t) = \theta_{v}E_{v}(t) + v\mu_{v}I_{v}(t) - \mu_{v}I_{v}(t) \tag{7}$$

with the conditions:
$$N_T = S_h + I_h + R_h$$
 and $N_v = S_v + E_v + I_v$. (8)

We normalize our equations (1)–(7) by introduce the new variables $s_h = \frac{S_h}{N_T}$, $e_h = \frac{E_h}{N_T}$

 $i_h = \frac{I_h}{N_T}, r_h = \frac{R_h}{N_T} \text{ for human class and } s_v = \frac{S_v}{N_v}, e_v = \frac{E_v}{N_v}, i_v = \frac{I_v}{N_v} \text{ for vector class. We}$

have the following reduced equations:

$$\frac{d}{dt}s_{h}(t) = \mu_{h}(1-s_{h})-\gamma_{h}s_{h}(t) i_{v}(t) N_{v}$$
(9)

$$\frac{\mathrm{d}}{\mathrm{d}t}e_{h}(t) = -e_{h}(\theta_{h} + \mu_{h}) + \gamma_{h}i_{v}(t)N_{v}s_{h}(t) \tag{10}$$

$$\frac{\mathrm{d}}{\mathrm{d}t}i_{h}(t) = \theta_{h}e_{h}(t) - i_{h}(t)(\mu_{h} + r) \tag{11}$$

$$\frac{d}{dt}s_{v}(t) = -\gamma_{v}i_{h}(t)N_{T}s_{v}(t) + \mu_{v}(1 - vi_{v}(t) - s_{v}(t))$$
(12)

$$\frac{d}{dt}i_{v}(t) = \theta_{v}e_{v}(t) - i_{v}(t)\mu_{v}(1-v)$$
(13)

were $r_h(t) = 1 - s_h(t) - e_h(t) - i_h(t)$ and $e_V = 1 - s_V(t) - i_V(t)$.

3. Results and Discussion

Follow the method of standard dynamical modeling method [15], the equilibrium points are found by setting the right hand side of equations (8) to (12). Thus, the equilibrium points are

- i) Disease free equilibrium point $D_0 = (1,0,0,1,0)$
- ii) Endemic disease equilibrium point $D_1 = (s_h^*, e_h^*, i_h^*, s_v^*, i_v^*)$ where

$$s_{h}^{*} = \frac{\mu_{h}}{\mu_{h} + \gamma_{h} i_{v}^{*} N_{v}}, \qquad (14)$$

$$e_h^* = \frac{\gamma_h i_v^* N_v s_h}{(\theta_h + \mu_h)}, \tag{15}$$

$$i_h^* = \frac{\theta_h e_h}{(r + \mu_h)}, \tag{16}$$

$$s_{v}^{*} = \frac{\mu_{v}(1 - vi_{v})}{(\mu_{v} + \gamma_{v}i_{h}^{*}N_{T})}$$
 and (17)

$$i_{v}^{*} = \frac{\mu_{h}\mu_{v}(\theta_{h} + \mu_{h})(\theta_{v} + \mu_{v})(r + \mu_{h})(R_{0} - 1)(1 - v)}{\gamma_{h}N_{v}(\theta_{h}\theta_{v}\gamma_{v}\mu_{h}N_{T} + \mu_{v}(\mu_{h}(\theta_{h} + \mu_{h})(\theta_{v} + \mu_{v}) + \theta_{h}\gamma_{v}\mu_{h}N_{T} + (\theta_{h} + \mu_{h})(\theta_{v} + \mu_{v})r)(1 v))}$$
(18)

where

$$R_0 = \frac{\theta_h \theta_v \gamma_h \gamma_v N_T N_v}{(\theta_h + \mu_h) \mu_v (\theta_v + \mu_v) (r + \mu_h) (1 - v)}$$
(19)

Next, we will analyze the stability of each equilibrium point. The local stability of each equilibrium point can be determined by looking the signs of each equilibrium state. If all eigenvalues have negative real parts, then that equilibrium point is local stable [15]. The eigenvalues can be solved from the characteristic equation:

 $det(J - \lambda I) = 0$ where J is the Jacobian matrix and I is the identity matrix.

Jacobian matrix of our equations are defined by

$$J = \begin{pmatrix} \frac{\partial}{\partial s_{h}} X(t) & \frac{\partial}{\partial e_{h}} X(t) & \frac{\partial}{\partial i_{h}} X(t) & \frac{\partial}{\partial s_{v}} X(t) & \frac{\partial}{\partial i_{v}} X(t) \\ \frac{\partial}{\partial s_{h}} Y(t) & \frac{\partial}{\partial e_{h}} Y(t) & \frac{\partial}{\partial i_{h}} Y(t) & \frac{\partial}{\partial s_{v}} Y(t) & \frac{\partial}{\partial i_{v}} Y(t) \\ \frac{\partial}{\partial s_{h}} Z(t) & \frac{\partial}{\partial e_{h}} Z(t) & \frac{\partial}{\partial i_{h}} Z(t) & \frac{\partial}{\partial s_{v}} Z(t) & \frac{\partial}{\partial i_{v}} Z(t) \\ \frac{\partial}{\partial s_{h}} W(t) & \frac{\partial}{\partial e_{h}} W(t) & \frac{\partial}{\partial i_{h}} W(t) & \frac{\partial}{\partial s_{v}} W(t) & \frac{\partial}{\partial i_{v}} W(t) \\ \frac{\partial}{\partial s_{h}} U(t) & \frac{\partial}{\partial e_{h}} U(t) & \frac{\partial}{\partial i_{h}} U(t) & \frac{\partial}{\partial s_{v}} U(t) & \frac{\partial}{\partial i_{v}} U(t) \end{pmatrix}$$

$$(20)$$

where $\frac{\partial}{\partial x} F(t)$ is the differential of F at x and

$$X(t) = \mu_{b} (1-s_{b}) - \gamma_{b} s_{b} (t) i_{v}(t) N_{v}$$
(21)

$$Y(t) = -e_{h}(\theta_{h} + \mu_{h}) + \gamma_{h}i_{v}(t)N_{v}s_{h}(t)$$
(22)

$$Z(t) = \theta_{h}e_{h}(t) - i_{h}(t)(\mu_{h} + r)$$
(23)

$$W(t) = -\gamma_{..}i_{b}(t)N_{T}s_{..}(t) + \mu_{..}(1 - vi_{..}(t) - s_{..}(t))$$
(24)

$$U(t) = \theta_{v} e_{v}(t) - i_{v}(t) \mu_{v}(1 - v)$$
(25)

At the disease free equilibrium point, the Jacobian matrix is defined by

$$J_{D_0} = \begin{pmatrix} -\mu_h & 0 & 0 & 0 & -\gamma_h N_v \\ 0 & -(\theta_h + \mu_h) & 0 & 0 & \gamma_h N_v \\ 0 & \theta_h & -(r + \mu_h) & 0 & 0 \\ 0 & 0 & -\gamma_v N_T & -\mu_v & -v \mu_v \\ 0 & 0 & 0 & 0 & -\mu_v (1-v) \end{pmatrix}.$$

The characteristic equation is given by $det(J_{D_0} - \lambda I) = 0$ or

$$(\lambda + \mu_h)(\lambda + (\theta_h + \mu_h))(\lambda + \mu_v)(\lambda + (r + \mu_h))(\lambda + \mu_v(1 - v)) = 0.$$

Thus the eigenvalues are

$$\lambda_1 = -\mu_h, \ \lambda_2 = -(\theta_h + \mu_h), \lambda_3 = -\mu_v, \ \lambda_4 = -(r + \mu_h), \ \lambda_5 = -\mu_v(1 - v).$$

At the endemic disease equilibrium point $D_1 = (s_h^*, e_h^*, i_h^*, s_v^*, i_v^*)$, the Jacobian matrix is defined by

$$J_{D_{l}} = \begin{pmatrix} -\mu_{h} - \gamma_{h} i_{v}^{*} N_{v} & 0 & 0 & 0 & -\gamma_{h} s_{h} N_{v} \\ \gamma_{h} i_{v}^{*} N_{v} & -(\theta_{h} + \mu_{h}) & 0 & 0 & \gamma_{h} s_{h} N_{v} \\ 0 & \theta_{h} & -(r + \mu_{h}) & 0 & 0 \\ 0 & 0 & -\gamma_{v} N_{T} s_{v} & -\mu_{v} - \gamma_{v} N_{T} i_{h} & -\mu_{v} r \\ 0 & 0 & 0 & 0 & -\mu_{v} (1 - v) \end{pmatrix}.$$

The characteristic equation is defined by $det(J_{D_1} - \lambda I) = 0$ or

$$(\lambda + (\theta_h + \mu_h))(\lambda + r + \mu_h)(\lambda + \mu_h (1 - v))(\lambda + \mu_h + (\theta_h + \mu_h)(r + \mu_h)\beta_h (R_0 - 1))$$

$$(\lambda + \frac{\mu_{v} + \theta_{h}\mu_{h}\gamma_{v}\beta_{h}N_{T}(R_{0} - 1)}{\left(\mu_{h} + \frac{(\theta_{h} + \mu_{h})(r + \mu_{h})(R_{0} - 1)}{\beta_{h}}\right)} = 0.$$

Therefore the eigenvalues are

$$\begin{split} \lambda_1 &= -(\theta_h + \mu_h) \\ \lambda_2 &= -(r + \mu_h) \\ \lambda_3 &= -\mu_v (1 - v) \\ \lambda_4 &= -\mu_h - (\theta_h + \mu_h)(r + \mu_h)\beta_h (R_0 - 1) \\ \lambda_5 &= -\mu_v - \beta_h \frac{\theta_h \mu_h \gamma_v N_T (R_0 - 1)}{\left(\mu_h + \frac{(\theta_h + \mu_h)(r + \mu_h)(R_0 - 1)}{\beta_h}\right)}. \end{split}$$

Where

$$\beta_h = \frac{\mu_h \mu_v (\theta_v + \mu_v)(1 - v)}{\theta_h \theta_v \gamma_v \mu_h N_T + \mu_v (\mu_h (\theta_h + \mu_h)(\theta_v + \mu_v) + \theta_h \gamma_v \mu_h N_T + (\theta_h + \mu_h)(\theta_v + \mu_v)r)(1 - v)}$$
 (26)
And R_0 is defined in equation (19).

It can be easily seen that all eigenvalues give negative real parts, then the disease free equilibrium point D_0 is local stability. For the endemic disease equilibrium point D_1 is local stability for $R_0 > 1$.

Next, we use Runge-Kutta Method for analyzing the numerical solutions. The parameter values are obtained from real life observations and are defined as follows: $\mu_h = 1/(365*70)$ per day corresponds to the expected life cycle of 70 years for human population in Thailand [16]. $\theta_h = 1/7$ per day corresponds to the 7 days of incubation period of dengue virus in human. $\theta_v = 1/10$ per day corresponds to the 10 days of incubation period of dengue virus in vector. $\mu_v = 1/30$ corresponds to the life cycle of 30 days for vector population. $\mathbf{r} = 1/14$ per day corresponds to the 14 days of recovery for human population [3, 4]. γ_h , γ_v

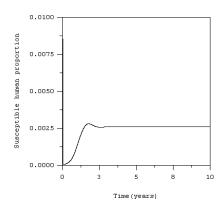


Figure 2. Time series solutions of susceptible human proportion.

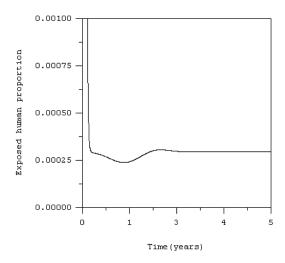


Figure 3. Time series solutions of exposed human proportion.

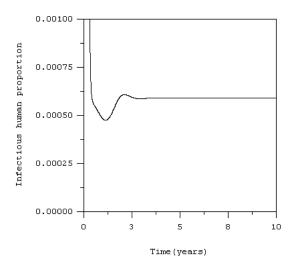


Figure 4. Time series solutions of infectious human proportion.

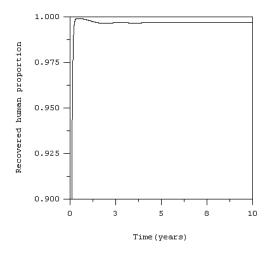


Figure 5. Time series solutions of recovered human proportion.

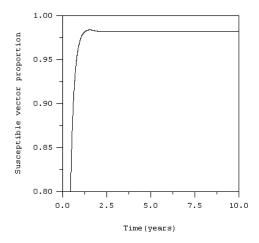


Figure 6. Time series solutions of susceptible vector proportion.

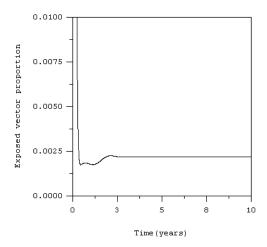


Figure 7. Time series solutions of exposed vector proportion.

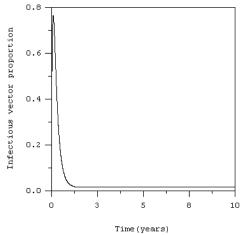


Figure 8. Time series solutions of infectious vector proportion.

From Figures 2-8, we can see that the parameters satisfy to the endemic condition. So the solutions oscillate to the endemic disease equilibrium point (0.00258958, 0.000294197, 0.000588046, 0.996528, 0.981601, 0.00216462, 0.0162345). We can see that the numerical solutions correspond to the analytical solutions.

4. Conclusions

This model considers the transmission of dengue disease between human and vector population. The vertical transmission of this disease is considered. The basic reproductive number is defined as the average number of secondary infection cases produced from primary infection. The basic reproductive number (R) is the geometric mean of R_0 and it is given by $R = \sqrt{R_0}$ where R_0 is defined in equation (19). The disease free equilibrium point exists and is local stable if the basic reproductive number is less than one and become unstable when the basic reproductive number is more than one. We used numerical simulations to show these results.

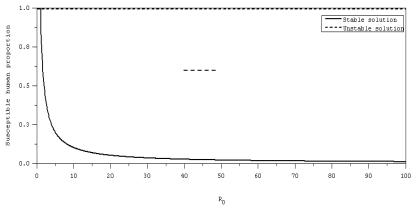
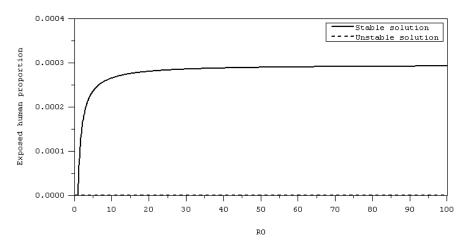


Figure 9. Bifurcation diagram of our model demonstrates the equilibrium solutions of susceptible human proportion for the different values of R_0 with $\mu_h=1/(365*70)$, $\theta_h=1/7$ $\theta_v=1/10$, $\mu_v=1/30$, r=1/14 , $\gamma_h=0.0001$, $\gamma_V=0.0005$, $N_T=1,000$, $N_v=10,000$.

represents the stable solutions and ---- represents the unstable solutions. For $R_0 < 1$, D_0 will be stable. For $R_0 > 1$, D_1 will be stable.



$$\label{eq:region_proportion} \begin{split} \text{Figure 10. Bifurcation diagram of our model demonstrates the equilibrium solutions of exposed human proportion for the different values of R_0 with $\mu_h = 1/(365*70)$, $\theta_h = 1/7$, $\theta_v = 1/10$, $\mu_v = 1/30$, $r = 1/14$, $\gamma_h = 0.0001$, $\gamma_v = 0.0005$, $N_T = 1,000$, $N_v = 10,000$. $$ $$ represents the stable solutions and ----- represents the unstable solutions. For $R_0 < 1$, $D_0 < 1$,$$

will be stable. For $R_0 > 1$, D_1 will be stable.

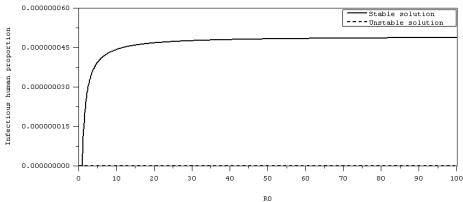
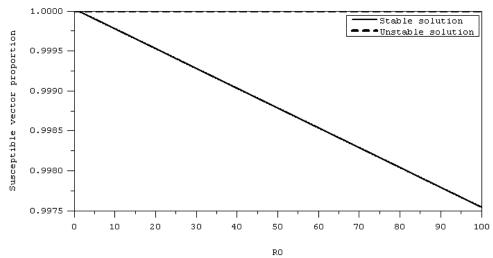


Figure 11. Bifurcation diagram of our model demonstrates the equilibrium solutions of infectious human proportion for the different values of R_0 with μ_h = 1/(365*70), θ_h = 1/7, θ_v = 1/10, μ_v = 1/30, r = 1/14, γ_h = 0.0001, γ_v =0.0005, N_T = 1,000, N_v = 10,000.

represents the stable solutions and ---- represents the unstable solutions. For $R_0 < 1$, D_0 will be stable. For $R_0 > 1$, D_1 will be stable.



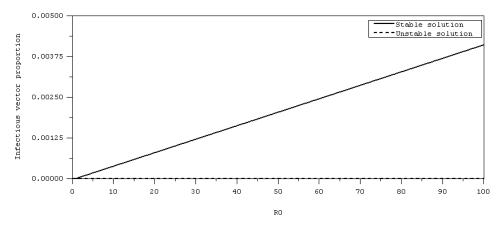


Figure 13. Bifurcation diagram of our model demonstrates the equilibrium solutions of infectious vector proportion for the different values of R_0 with $\mu_h=1/(365*70)$, $\theta_h=1/7$, $\theta_v=1/10$, $\mu_v=1/30$, r=1/14, $\gamma_h=0.0001$, $\gamma_v=0.0005$, $N_T=1,000$, $N_v=10,000$.
—represents the stable solutions and ----- represents the unstable solutions. For $R_0<1$, D_0 will be stable. For $R_0>1$, D_1 will be stable.

From Figures 9-13, we can see that if the basic reproductive number is greater than one, the proportions of susceptible human and susceptible vector decrease. The proportions of exposed human, infectious human and infectious vector increase. These subsequent behaviours occur because there are enough susceptible population to be infected from infectious population.

Furthermore, we vary the fraction of vector infected by vertical transmission(v) by consider the time series of susceptible vector proportion, we can see that when the higher fraction of vector infected by vertical transmission, the susceptible vector proportion decrease and the infectious vector proportion increase as shown in Figures 14-15. This behaviour occurs because there are enough susceptible vector to be infected from infectious vector and some infectious vector get virus from their parents by vertical transmission.

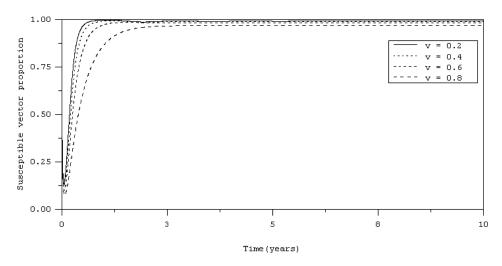


Figure 14. Time series solutions of susceptible vector proportion for the different fractions of vertical transmission with $\mu_h = 1/(365*70)$, $\theta_h = 1/7$, $\theta_v = 1/10$, $\mu_v = 1/30$, r = 1/14, $\gamma_h = 0.0001$, $\gamma_v = 0.0005$, $N_T = 1,000$, $N_v = 10,000$.

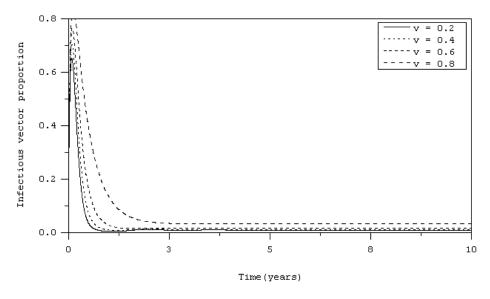


Figure 15. Time series solutions of infectious vector proportion for the different fractions of vertical transmission with $\mu_h=1/(365*70)$, $\theta_h=1/7$, $\theta_v=1/10$, $\mu_v=1/30$, r=1/14, $\gamma_h=0.0001$, $\gamma_V=0.0005$, $N_T=1,000$, $N_v=10,000$.

The vertical transmission of dengue virus may support the fact that it is originally the virus of mosquitoes which has developed inside the mosquito to be adult [5]. Therefore we should consider the vertical transmission of dengue virus because it effects to the outbreak of dengue disease.

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