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Mathematical Model of Dengue Disease Transmission with Severe DHF Compartment

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Abstract. An SIR model for dengue disease transmission is discussed here. It is assumed that two viruses namely strain 1 and strain 2 cause the disease and long lasting immunity from infection caused by one virus may not be valid with respect to a secondary infection by the other virus. Our interest here is to derive and analyse the model taking into account the severe DHF compartment in the transmission model. The aim would be to find a control measure to reduce the DHF patients in the population, or to keep the number of patients at an acceptable level. Analysis of this model reveals that there are four equilibria, one of them is the disease-free, the other three equilibria correspond to the presence of single serotype respectively, and the coexistence of two serotypes. Stability analysis of each equilibria and their relations with type reproductive numbers are shown. We also discuss the ratio between total number of severe DHF compartment with respect to the total number of first infection compartment and the total number of secondary infection compartment, respectively. This ratio is needed for practical control measure in order to predict the "real" intensity of the endemic phenomena since only data of severe DHF compartment is available in the field.

2000 Mathematics Subject Classification: 92D30

Key words and phrases: Severe DHF compartment, Type reproductive number, Equilibrium point.

1. Introduction

Dengue fever (DF) and Dengue Haemorrhagic Fever (DHF) are increasingly important public health problems in the tropic and subtropics areas. Dengue has been recognized in over 100 countries and 2.5 billion people live in areas where dengue is endemic [12]. Dengue viruses are transmitted to human by the bite of Aedes aegypti female mosquitoes, which are known as the principal vectors although some other species such as Aedes albopictus are also of importance. The infection in the

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mosquito is for life [9]. The spectrum of illness of dengue ranges from unapparent, mild disease to a severe and occasionally fatal hemorrhagic clinical picture [12]. The risk factors associated with severe and fatal dengue infections are not well understood. Epidemiological studies in Thailand and Cuba suggest that an important risk factor for DHF or dengue shock syndrome (DSS) is the presence of preexisting dengue antibodies at sub-neutralizing levels. DHF and DSS are associated with individuals with secondary infection, and with primary infections in newborn babies whose mothers were immune to dengue [9,12]. These facts led to the formulation of the secondary infection or immune enhancement hypothesis [5]. Dengue disease caused by four distinct serotypes virus known as DEN 1, DEN 2, DEN 3 and DEN 4 in which only DEN 2 and DEN 3 are mostly identified in tropical country [8]. A person infected by one of the four serotypes will never be infected again by the same serotype, but he or she could be reinfected by three other serotypes in about 12 weeks and then becomes more susceptible to developing DHF [2].

In this work we develop a mathematical modelling as an interesting tool for the understanding of these illnesses and for the proposition of strategies. Our interest here is to derive and analyse the model taking into account the severe DHF compartment in the transmission model. The model is developed from the previous work with no severe DHF compartment by Feng and Velasco-Hernandez [7] and Esteva and Vargas [5] for two-strain viruses and Esteva and Vargas [3,4,6] for one strain virus. Separating the severe DHF individuals from infected population is very important in the model. This is due to the fact that only data of hospitalized persons are known and most likely this group being isolated in the hospital, may not infect mosquitoes and viruses remain in human body for only about seven days [11]. In the next section we give formulation of the model. In section three we describe about type reproductive number of this model, the equilibrium points of this system and its stability. The last two sections give numerical results and conclusion.

2. The mathematical model

Let N_h and N_v be the human host and vector population sizes. We assume that the host and vector population have constant size. The mathematical model for this transmission is based on the transmission diagram in Figure 1.

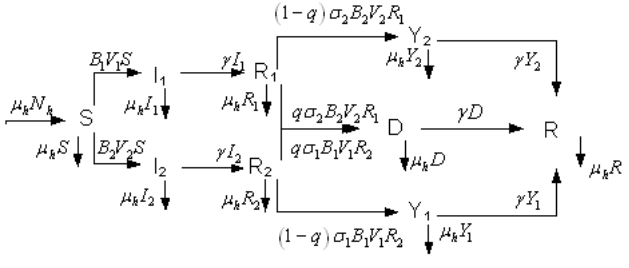


Figure 1. Transmission diagram of two-strain viruses

We have the following states: S for naive individuals (i.e. those susceptible to both strain one and two), I_i for those infected and infectious for strain i only, R_i for

those immune to strain i only, Y_i for those who are immune to strain j who have been infected with strain i and are infectious for that strain, R for those who are immune to both strains. D for those who are immune to strain 1 or strain 2 and who now become infected with the other strain and develop severe symptoms. V_0 for proportion of susceptible vectors and V_i for proportion of infected vectors strain i. The flows between compartments are: S to I_1 and I_2, I_1 to R_1, I_2 to R_2. We assume that a proportion of q individuals from R_i move to compartment D where q is the probability of severe DHF. The proportion of $1-q$ individuals move to compartment Y_i. In this model we assume that there is no transmission to the vector from the D class, but only from the I and Y classes and there is no mortality factor due to the disease. Because most likely they have been taken care in the hospital and away from mosquitoes. The aim would be to reduce the DHF patients in the population, or to keep that at an acceptable level. It implies that for the severity of DHF, it does not matter whether ones have strain 1 first and then strain 2 or the other way around then we have the value of q are the same for strain 1 or strain 2. Hence our model have the capability to predict whether control measures would make things better or worse. Suppose that the primary rate of infection from vector to host produced by either of two strains at rates $B_i = b\beta_i, i \in (1,2)$ and from host to vector at rates $A_i = b\alpha_i, i \in (1,2)$. Values of parameters used in the model are given in Table 1. The dynamical equations for host are

$$
\begin{aligned}
\frac{d\tilde{S}}{dt} &= \mu_h N_h - (B_1V_1 + B_2V_2)\tilde{S} - \mu_h\tilde{S},\\
\frac{d\tilde{I}_1}{dt} &= B_1V_1\tilde{S} - (\gamma+\mu_h)\tilde{I}_1,\\
\frac{d\tilde{I}_2}{dt} &= B_2V_2\tilde{S} - (\gamma+\mu_h)\tilde{I}_2,\\
\frac{d\tilde{R}_1}{dt} &= \gamma\tilde{I}_1 - \sigma_2B_2V_2\tilde{R}_1 - \mu_h\tilde{R}_1,\\
\frac{d\tilde{R}_2}{dt} &= \gamma\tilde{I}_2 - \sigma_1B_1V_1\tilde{R}_2 - \mu_h\tilde{R}_2,\\
\frac{d\tilde{D}}{dt} &= q(\sigma_2B_2V_2\tilde{R}_1 + \sigma_1B_1V_1\tilde{R}_2) - (\mu_h+\gamma)\tilde{D},\\
\frac{d\tilde{Y}_1}{dt} &= (1-q)\sigma_1B_1V_1\tilde{R}_2 - (\gamma+\mu_h)\tilde{Y}_1,\\
\frac{d\tilde{Y}_2}{dt} &= (1-q)\sigma_2B_2V_2\tilde{R}_1 - (\gamma+\mu_h)\tilde{Y}_2,\\
\frac{d\tilde{R}}{dt} &= \gamma(\tilde{Y}_1+\tilde{Y}_2) - \mu_h\tilde{R} + \gamma D,
\end{aligned}
\tag{2.1}
$$

and the dynamical equations for vector are as follows

$$
\begin{aligned}
\frac{dV_0(t)}{dt} &= \mu_v - [A_1(\frac{\tilde{I}_1}{N_h} + \frac{\tilde{Y}_1}{N_h}) + A_2(\frac{\tilde{I}_2}{N_h} + \frac{\tilde{Y}_2}{N_h})] - \mu_v V_0, \\
\frac{dV_1(t)}{dt} &= A_1(\frac{\tilde{I}_1}{N_h} + \frac{\tilde{Y}_1}{N_h})V_0 - \mu_v V_1, \\
\frac{dV_2(t)}{dt} &= A_2(\frac{\tilde{I}_2}{N_h} + \frac{\tilde{Y}_2}{N_h})V_0 - \mu_v V_2.
\end{aligned} \tag{2.2}
$$

The equation for $\tilde{R}$ and V_0 in (2.1)–(2.2) can be eliminated since at every time t, we have $\tilde{S} + \tilde{I}_1 + \tilde{I}_2 + \tilde{R}_1 + \tilde{R}_2 + \tilde{D} + \tilde{Y}_1 + \tilde{Y}_2 + \tilde{R} = N_h$ and $V_0 + V_1 + V_2 = 1$. To simplify the mathematical analysis of this study, we normalize the model (2.1)–(2.2) by defining new variables

$$
S = \frac{\tilde{S}}{N_h}, I_i = \frac{\tilde{I}_i}{N_h}, R_i = \frac{\tilde{R}_i}{N_h}, Y_i = \frac{\tilde{Y}_i}{N_h}, R = \frac{\tilde{R}}{N_h}, D = \frac{\tilde{D}}{N_h}, i \in (1, 2).
$$

We obtain the equations (2.1)–(2.2) as follows

$$
\begin{aligned}
\frac{dS}{dt} &= (1 - \mu_h)S - (B_1 V_1 + B_2 V_2)S, \\
\frac{dI_i}{dt} &= B_i V_i S - (\gamma + \mu_h)I_i, \\
\frac{dR_i}{dt} &= \gamma I_i - \sigma_j B_j V_j R_i - \mu_h R_i, \\
\frac{dD}{dt} &= q(\sigma_2 B_2 V_2 R_1 + \sigma_1 B_1 V_1 R_2) - (\mu_h + \delta)D, \\
\frac{dY_i}{dt} &= (1 - q)\sigma_i B_i V_i R_j - (\gamma + \mu_h)Y_i, \\
\frac{dV_i}{dt} &= A_i(I_i + Y_i)(1 - V_1 - V_2) - \mu_v V_i, i, j \in (1, 2), i \neq j.
\end{aligned} \tag{2.3}
$$

Table 1. Parameter values

Symbol	Parameter Definition	Value
μ_h^{-1}	Host life expectancy	70 years
μ_v^{-1}	Vector life expectancy	14 days
γ^{-1}	Mean length of infectious period in host	10–15 days
A_i	Biting rate x successful transmission from host to vector	Variable
B_i	Biting rate x successful transmission from vector to host	Variable
σ_i	Susceptibility index	$[0, 5]$
q	Probability of severe DHF	$[0, 1]$

3. Analysis of the model

3.1. Type-reproduction number. Now we are interested in a new threshold parameter known as a type-reproduction number introduce by Roberts and Heesterbeek [10]. This parameter is defined as the expected number of cases in individual of

type 1 caused by one infected individual of type 1 in a completely susceptible population, either directly or through chains of infection passing through any sequence of the other types. This parameter is related to R_0, but singles out the control effort needed when control is targeted at particular host type rather than at the population as a whole. We refer to the quantity as T when single type is targeted. When we have n types of epidemiologically distinct host types, we define precisely the type-reproduction number T as

$$T = e^T K(I - (I - P)K)^{-1} e \tag{3.1}$$

where $K_{n\times n}$ is the next-generation matrix (see [3] for details), $I_{n\times n}$ is the identity matrix, $e_{n\times 1}$ is the vector $(1, 0, ..., 0)$, e^T is transpose e and $P_{n\times n}$ is the projection matrix on type 1 (i.e. $p_{11} = 1$, and $p_{ij} = 0$ for all other entries). The main property of this parameter T is $T < 1 \leftrightarrow R_0 < 1$ (see details in [10]). Let $X = [I_1, I_2, Y_1, Y_2, V_1, V_2]$ be infection-related compartments vector. The next generation matrix $K = k_{ij}$ for system (2.3) is given by

$$K = \begin{pmatrix} 0 & 0 & 0 & 0 & \frac{A_1}{\mu_h+\gamma} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{A_2}{\mu_h+\gamma} \\ 0 & 0 & 0 & 0 & \frac{A_1}{\mu_h+\gamma} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{A_2}{\mu_h+\gamma} \\ \frac{\beta_1}{\mu_v} & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_2}{\mu_v} & 0 & 0 & 0 & 0 \end{pmatrix} \tag{3.2}$$

where k_{ij} is the expected number of secondary cases in type i that would arise from typical primary case in type j in a susceptible population. In this matrix K the humans cannot infect humans and mosquitoes cannot infect mosquitoes, hence $k_{ij} = 0$, for $i, j \in 1, 2, 3, 4$ and $k_{mn} = 0$, for $m, n \in 5, 6$. The entry k_{15}, k_{26}, k_{35}, k_{46} are defined as the expected number of humans that are infected by a single mosquito, and k_{51}, k_{62} are defined as the expected number of mosquitoes infected by a single human. The other elements of K are zero, which means that there is no secondary infection in mosquitoes. Using K we determine the expected number of infected hosts resulting from an infectious host with serotype $i, i \in 1, 2$, $T_i = \frac{A_i B_i}{\mu_v(\mu_h+\gamma)}$. The value of T_i for primary and secondary infections with serotype i are given in Table 2 below. Now, we define the type reproductive number for model (2.3) as $T_i = \frac{A_i B_i}{\mu_v(\mu_h+\gamma)}, i \in 1, 2$, here i is the serotype of virus. This parameter will be used to analyse the stability of equation (2.3) through equilibrium points.

Table 2. The value of type-reproduction number for model (2.3)

Related infection	Value of T
First infection of host with serotype 1 (I_1)	$\frac{A_1 B_1}{\mu_v(\mu_h+\gamma)}$
First infection of host with serotype 2 (I_2)	$\frac{A_2 B_2}{\mu_v(\mu_h+\gamma)}$
Secondary infection of host with serotype 1 (Y_1)	$\frac{A_1 B_1}{\mu_v(\mu_h+\gamma)}$
Secondary infection of host with serotype 2 (Y_2)	$\frac{A_2 B_2}{\mu_v(\mu_h+\gamma)}$

3.2. Equilibrium points. In this section we will find the equilibrium points of system (2.3) in the region of Ω, with

$$\Omega = \{(S, I_i, R_i, Y_i, D, V_i) \in \mathcal{R}_+^{10} | V_1 + V_2 \leq 1, S + I_i + R_i + Y_i + D \leq 1\}$$

where $i = 1, 2$. We present some result concerning the existence of equilibrium points of system (2.3).

3.2.1. *Non-endemic equilibrium.* We can immediately see that the disease free equilibrium $E_0 = (1, 0, 0, 0, 0, 0, 0, 0, 0, 0)$ is a solution of system (2.3). The stability of E_0 is given by the following theorem.

Theorem 3.1. *The model formulated in (2.3) has $E_0 = (1, 0, 0, 0, 0, 0, 0, 0, 0, 0)$ as a locally stable disease free equilibrium point if and only if $T_i < 1, i = 1, 2$. Otherwise E_0 is an unstable disease free equilibrium.*

Proof. The local stability of this equilibrium solutions can be examined by linearizing system (2.3) around E_0. This gives the Jacobian matrix D_{E_0} as follow

$$D_{E_0} = \begin{bmatrix} -\mu_h & 0 & 0 & -B_1 & 0 & 0 & -B_2 & 0 & 0 & 0 \\ 0 & -\mu_h - \gamma & 0 & -B_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma & -\mu h & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & A_1 & 0 & -\mu v & 0 & 0 & 0 & A_1 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu h - \gamma & 0 & B_2 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & A_2 & 0 & -\mu v & 0 & A_2 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_h - \gamma & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_h - \gamma & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_h - \gamma \end{bmatrix}.$$

The eigenvalues of D_{E_0} are $-\mu_h$ with multiplicity 3, $-\mu h - \gamma$ with multiplicity 3, and the roots of polynomial $p_i(x) = x^2 + ax + b_i, i = 1, 2$, where

$$\begin{aligned} a &= \mu h + \mu_v + \gamma > 0, \\ b &= (\mu_h + \gamma)\mu_v(1 - T_i), i = 1, 2. \end{aligned}$$

Using the Routh-Hurwitz criteria, the roots of polynomial p_i have negative real part when $T_i < 1$. We deduce that E_0 is a locally asymptotically stable when $T_i < 1$, and a saddle point where $T_i > 1$. This proves Theorem 3.1. ∎

3.2.2. *Endemic equilibria.* We determine now other equilibrium of system (2.3). Suppose that only serotype i is present, $i = 1, 2$. Then we have the following equilibria

$$E_1 = (S_1^*, I_1^*, 0, R_1^*, 0, 0, 0, 0, V_1^*, 0),$$

$$E_1 = (S_2^*, 0, I_2^*, 0, R_2^*, 0, 0, 0, 0, V_2^*),$$

where

$$S_i^* = \frac{\mu_h T_i + B_i}{T_i(\mu_h + B_i)}, I_i^* = \frac{\mu_h B_i(T_i - 1)}{(\mu_h + \gamma)(\mu_h + B_i)T_i},$$

$$R_i^* = \frac{\gamma I_i^*}{\mu_h(\mu_h + \gamma)}, V_i^* = \frac{\mu_h(T_i - 1)}{\mu_h T_i + B_i}, i = 1, 2.$$

The endemic points, E_i exist if and only if $T_i > 1, i = 1, 2$.

Theorem 3.2. *The equilibria $E_i = i = 1, 2$ is a locally asymptotically stable endemic point if and only if $T_i > 1$ and*

$$T_j < \frac{T_i}{1 + \dfrac{\gamma\sigma_j B_i(1-q)(T_i-1)}{(\mu_h T_i + B_i)(\mu_h+\gamma)^2}}, i, j = 1, 2, i \neq j. \tag{3.3}$$

Otherwise E_i is a non stable endemic equilibrium.

Proof. We study now the stability of $E_i, i = 1, 2$. The corresponding Jacobian matrix is

$$D_{E_i} = \begin{bmatrix} G_1 & G_2 \\ 0 & G_4 \end{bmatrix}$$

where

$$G_1 = \begin{bmatrix} -\mu_h - B_i V_i^* & 0 & 0 & -B_i S^* \\ 0 & -\mu_h - \gamma & 0 & -B_i S^* \\ 0 & \gamma & -\mu h & 0 \\ 0 & A_i(1 - V_i^*) & 0 & -\mu v - A_i I_i^* \end{bmatrix}$$

and

$$G_4 = \begin{bmatrix} -\mu h - \gamma & 0 & B_j S^* & 0 & 0 & 0 \\ \gamma & -\mu_h - \sigma_i B_i V_i^* & 0 & 0 & 0 & 0 \\ A_j(1 - V_i^*) & 0 & -\mu v & 0 & A_j(1 - V_i^*) & 0 \\ 0 & (1-q)\sigma_i B_i V_i^* & 0 & -\mu_h - \gamma & 0 & 0 \\ 0 & 0 & (1-q)\sigma_j B_j R_i^* & 0 & -\mu_h - \gamma & 0 \\ 0 & q\sigma_i B_i V_i^* & q\sigma_j B_j R_i^* & 0 & 0 & -\mu_h - \gamma \end{bmatrix}.$$

The eigenvalues of D_{E_i} are given by the eigenvalues of G_1 and G_4. The eigenvalues of G_1 are $-\mu_h$, and the roots of polynomial $p(x) = x^3 + a_i x^2 + b_i x + c_i, i = 1, 2$, where

$$\begin{aligned} a_i &= \mu h + \mu_v + \kappa + \phi_i + \varphi_i, \\ b_i &= (\mu_v + \gamma)\mu_h + \mu_h^2 + \phi_i\varphi i + (\kappa + \mu_v)\varphi_i + (\kappa + \mu_h)\phi_i, \\ c_i &= \mu_h\kappa\phi_i + \kappa\mu_v\varphi_i + \kappa\varphi_i\phi i, \\ \phi_i &= \frac{A_i(T_i - 1)}{\lambda_i + T_i M}, \varphi_i = \frac{B_i\lambda_i(T_i - 1)}{T_i(\lambda_i + M)}, \\ \lambda_i &= \frac{A_i}{\mu_v}, \kappa = \mu_h + \gamma, M = \frac{\kappa}{\mu_h}, i = 1, 2. \end{aligned}$$

Observe that $a_i, b_i, c_i > 0$ when $T_i > 1$. Also it can be seen that

$$\begin{aligned} c_i &< 2\mu_h^2 + \mu_h(\mu_v + \gamma)\phi_i + (\mu_h + \mu_v + \kappa)(\mu_h + \mu_v + 2\kappa)(\phi_i + \varphi_i), \\ &\quad +(2\mu_h + 2\mu_v + 2\kappa)\varphi_i\phi i, \\ &< a_i b_i. \end{aligned}$$

Therefore by Routh-Hurwitz criteria we deduce that the roots of the polynomial $p(x)$ have negative real part when $T_i > 1$. The eigenvalues of G_4 are $-\mu_h - \gamma$ with multiplicity 3, $-\mu_h - \sigma_i B_i V_i^*, i = 1, 2$, and the roots of polynomial $g_i(x) = x^2 + p_i x + q_i, i = 1, 2$ where

$$\begin{aligned} p_i &= \mu h + \mu_v + \gamma > 0, \\ q_i &= (\mu_h + \gamma)\mu_v - A_j B_j(1 - V_i^*)[S^* + (1-q)\sigma_j R_i^*], i = 1, 2, i \neq j. \end{aligned}$$

Applying Routh-Hurwitz criteria to polynomial $g(x)$, the roots of this polynomial have negative real part when $b_i > 0, i = 1, 2$ and we have the following inequality in term of type reproductive number,

$$T_j < \frac{T_i}{1 + \dfrac{\gamma\sigma_j B_i(1-q)(T_i-1)}{(\mu_h T_i + B_i)(\mu_h+\gamma)^2}}, i, j = 1, 2, i \neq j.$$

We can deduce that the equilibria E_i is locally asymptotically stable when

$$T_i > 1 \text{ and } T_j < \frac{T_i}{1 + \dfrac{\gamma\sigma_j B_i(1-q)(T_i-1)}{(\mu_h T_i + B_i)(\mu_h+\gamma)^2}}, i, j = 1, 2, i \neq j.$$

This proves Theorem 3.2. ∎

Observe that for $T_1 > 1$ and $T_2 > 1$, the inequalities given by (3.3) for $i = 1, 2$ cannot be fulfilled simultaneously, therefore E_1 and E_2 can not be locally stable at the same time. Figure 2 illustrates the stability diagram of E_0, E_1 and E_2 depending on the type reproductive numbers for different values of σ_1 and σ_2 (susceptibility index of serotype i). We notice that the stability region of E_1 and E_2 become smaller as the σ_1 and σ_2 increases. We obtain Figure 2 from the inequality (3.3) under a set of fixed parameters. In Figure 2(a), $\sigma_1 = \sigma_2 = 0$, here we have three regions for E_0, E_1 and E_2. In Figure 2(b) the values of susceptibility index are $\sigma_1 = 0.01$ and $\sigma_2 = 0.08$, respectively. In Figure 2(c) we increase the values of susceptibility index into $\sigma_1 = 0.5$ and $\sigma_2 = 1.4$. Analysing Figure 2, we notice that the results are similar to those of [5]. But in our model we observe that for $\sigma_1 > 1$ and $\sigma_2 > 1$ we have Figure 2(d) which is not found in [5].

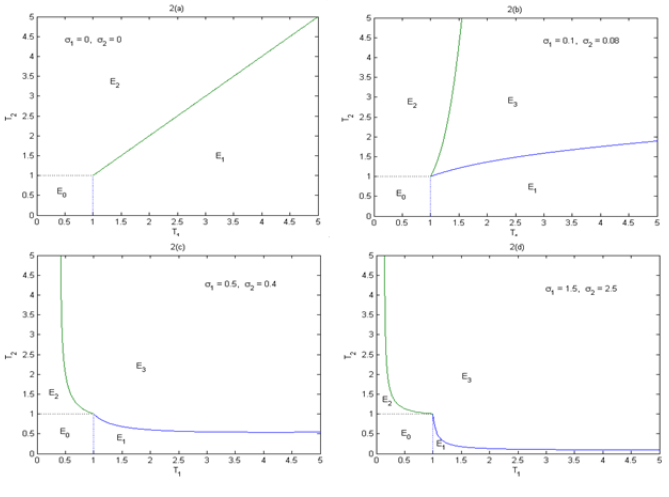


Figure 2. Diagram of existence and stability of equilibria E_i for different value of σ_1 and σ_2. Parameter values are $\gamma = 0.1428, A_1 = 1.5, A_2 = 3, B_1 = 2.5, B_2 = 1$, and $q = 0.02$.

3.2.3. *Coexistence of endemic equilibrium.* We obtain the coexistence of endemic equilibrium points when we make the left hand side of system (2.3) equal to zero, that is $E_3 = \{S^{**}, I_i^{**}, R_i^{**}, Y_i^{**}, D^{**}, V_i^{**}\}$ where

$$\begin{aligned}
S^{**} &= \frac{\mu_h}{\mu_h + B_1 V_1^{**}}, I_i^{**} = \frac{B_i V_i^{**} S^{**}}{\mu_h + \gamma}, \\
R_i^{**} &= \frac{\gamma \mu_h I_i^{**}}{\sigma_j B_j V_j^{**} + \mu_h}, Y_i^{**} = \frac{(1-q)\gamma B_j V_j^{**} \sigma_i I_i^{**}}{(\mu_h + \gamma)(\sigma_i B_i V_i^{**} + \mu_h)}, \\
D^{**} &= \frac{q\mu_h M[\mu_h(\sigma_1 + \sigma_2) + \sigma_1\sigma_2(B_1 V_1^{**} + B_2 V_2^{**})] R_1^{**} R_2^{**}}{\gamma(\mu_h + \gamma) S^{**}}, i, j = 1, 2, i \neq j.
\end{aligned}$$

Substituting the above expressions in system (2.3), we obtain the following equations for the variables V_1 and V_2.

$$\begin{aligned}
F_1 &= \frac{dV_1}{dt} = a_1 {V_1^{**}}^2 + b_1 {V_2^{**}}^2 + c_1 V_1^{**} V_2^{**} + d_1 V_1^{**} + e_1 V_2^{**} + f_1 = 0, \\
F_2 &= \frac{dV_2}{dt} = a_2 {V_2^{**}}^2 + b_2 {V_1^{**}}^2 + c_2 V_2^{**} V_1^{**} + d_2 V_2^{**} + e_2 V_1^{**} + f_2 = 0,
\end{aligned} \tag{3.4}$$

where

$$\begin{aligned}
a_i &= B_i^2 \sigma_i \gamma M(A_i \mu_h + \mu_v \gamma M), \\
b_i &= A_i B_i B_j \sigma_i \mu_h \gamma (1-q), \\
c_i &= B_i \sigma_i \mu_h [A_i B_i \mu_h M + A_i B_j \gamma (1-q) + B_j \mu_v \mu_h M^2], \\
d_i &= \mu_h^3 B_i M \mu_v [\lambda_i + M + \sigma_i M (1 - T_i)], \\
e_i &= \mu_h^2 \mu_v M [T_i(\mu_h^2 M - B_j \sigma_i \gamma (1-q)) + B_j \mu_h M], \\
f_i &= \mu_h^4 M^2 \mu_v (1 - T_i), M = \frac{\mu_h + \gamma}{\mu_h}, \lambda_i = \frac{A_i}{\mu_v}, i, j = 1, 2, i \neq j.
\end{aligned}$$

Suppose that $0 < V_1^{**}, V_2^{**} \leq 1$, the existence of E_3 is fulfilled if

$$F_1(V_1^{**}, 0) < F_2(V_1^{**}, 0), F_2(0, V_2^{**}) < F_1(0, V_2^{**}) \tag{3.5}$$

or

$$F_1(V_1^{**}, 0) > F_2(V_1^{**}, 0), F_2(0, V_2^{**}) > F_1(0, V_2^{**}) \tag{3.6}$$

where F_1 and F_2 are monotone decrease function of equation (3.4) and

$$\begin{aligned}
F_1(V_1^{**}, 0) &= \frac{-d_1 + \sqrt{d_1^2 - 4a_1 f_1}}{2a_1}, F_2(V_1^{**}, 0) = \frac{-d_2 + \sqrt{d_2^2 - 4a_2 f_2}}{2a_2}, \\
F_1(0, V_2^{**}) &= \frac{-e_1 + \sqrt{e_1^2 - 4b_1 f_1}}{2b_1}, F_2(0, V_2^{**}) = \frac{-e_2 + \sqrt{e_2^2 - 4b_2 f_2}}{2b_2}.
\end{aligned}$$

We illustrate the condition (3.5) in Figure 3 (left). Let

$$G_1 = F_1(V_1^{**}, 0) - F_2(V_1^{**}, 0) = G_1(T_1, T_2)$$

and

$$G_2 = F_2(0, V_2^{**}) - F_1(0, V_2^{**}) = G_2(T_1, T_2).$$

We transform the condition (3.5) into the region B in Figure 3 (right). Hence we have the condition (3.6) for the parameter values of T_1 and T_2 in region A of Figure 3 (right).

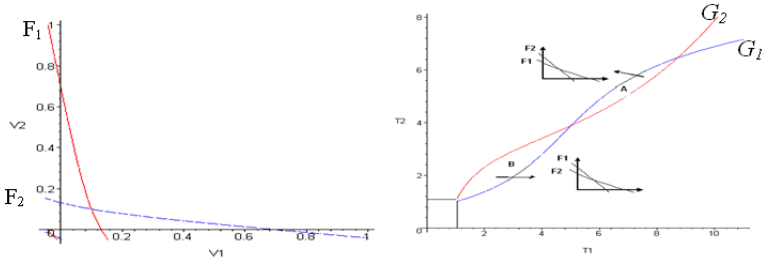


Figure 3. The sketch of equations (3.4) 3 (left) and region of coexistence of two serotype viruses 3 (right) under a fix parameter values $\mu_v = \frac{1}{14}, \gamma = 0.071, \beta_1 = 0.5, \beta 2 = 0.36, \mu_h = \frac{1}{70}, \alpha_1 = 0.61, \alpha_2 = 0.34, q = 0.02, b = 1, \sigma_1 = 0.6, \sigma_2 = 0.8$.

The stability of E_3 derives from the location of the jacobian eigenvalues of matrix of system (2.3) evaluated at E_3. From Gerschgorin disk Theorem [1] we obtain the following conditions for stability of this equilibrium.

$$
\begin{aligned}
2\lambda_i(1 - V_1^{**} - V_2^{**}) - 1 \leq 0,\\
V_i^{**} + S^{**} - (1-q)\sigma_i(V_i^{**} + R_j^{**}) \leq 0,\\
\sigma_i B_i(R_j^{**} - V_i^{**}) + \gamma - \mu_h \leq 0,\\
(B_1 + B_2)S^{**} - (\mu_h + B_1 V_1^{**} + B_2 V_2^{**}) \leq 0, \qquad i, j = 1, 2.
\end{aligned} \tag{3.7}
$$

Since in general it is not possible to find the exact solution of equations (3.4) in explicit form, we analyse the special case when the characteristic transmission and the susceptibility index for both serotype are identical. It means that $A_1 = A_2 = A, B_1 = B_2 = B, \sigma_1 = \sigma_2 = \sigma, T_1 = T_2 = T$. Equation (3.4) becomes

$$
aV^{**^2} + bV^{**} + c = 0 \tag{3.8}
$$

where

$$
\begin{aligned}
a &= 2B^2\sigma[A\mu_h(\mu_h + \gamma + \gamma(1-q)) + \mu_v(\mu_h + \gamma)],\\
b &= B\mu_h[(\mu_h + \gamma)(2A\mu_h + \mu_v(\mu_h + \gamma)(2 + \sigma)) - AB\sigma(\mu_h + \gamma + \gamma(1-q))],\\
c &= \mu_h^2\mu_v(\mu_h + \gamma)^2(1 - T).
\end{aligned}
$$

The equation (3.8) will have a unique positive solution V^{**} if and only if $T > 1$ and $T = \frac{AB}{\mu_v(\mu_h + \gamma)}$. In this case, the endemic equilibrium E_3 is given by

$$
E_{3_a} = (S^{**}, I_i^{**} = I^{**}, R_i^{**} = R^{**}, Y_i^{**} = Y^{**}, D^{**})
$$

where

$$
\begin{aligned}
S^{**} &= \frac{\mu_h}{\mu_h + 2BV^{**}}, \\
I_i^{**} &= I^{**} = \frac{BV^{**}S^{**}}{\mu_h + \gamma}, \\
R_i^{**} &= R^{**} = \frac{\gamma I^{**}}{\sigma BV^{**} + \mu_h}, \\
Y_i^{**} &= Y^{**} = \frac{(1-q)\sigma BV^{**}R^{**}}{\mu_h + \gamma}, \\
D^{**} &= \frac{2q(\mu_h + \gamma)Y^{**}}{(1-q)(\mu_h + \gamma)}, i = 1, 2,
\end{aligned} \tag{3.9}
$$

where V^{**} is the positive solution of equation (3.8). The solution of equation (3.8) depends on the value of type reproductive number, as a consequence the equilibrium in (3.9) also depends on this parameter. In order to find the stability of the endemic equilibrium E_{3_a} in the equations (3.9) we have the following theorem.

Theorem 3.3. *The equilibrium E_{3_a} in equations (3.9) is a locally asymptotically stable endemic point if and only if*

$$
1 < T < \frac{B(B\sigma\mu_v + 2A\mu_h^2 + \Lambda(2+\sigma))}{2\mu_h\Lambda} + 1, \Lambda = \mu_h\mu_v(\mu_h + \gamma). \tag{3.10}
$$

Proof. The jacobian matrix of system (2.3) at the equilibrium E_{3_a} is given by

$$
D_{E_{3_a}} = \begin{bmatrix}
-\mu_h - 2\check{V} & 0 & 0 & -\check{S} & 0 & 0 & -\check{S} & 0 & 0 & 0 \\
-\check{V} & \chi & 0 & -\check{S} & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \gamma & -\mu h - \sigma\check{V} & 0 & 0 & 0 & -\check{R} & 0 & 0 & 0 \\
0 & \Delta & 0 & -\mu v - \Gamma & 0 & 0 & -\Gamma & \Delta & 0 & 0 \\
\Pi & 0 & 0 & 0 & \chi & 0 & \check{S} & 0 & 0 & 0 \\
0 & 0 & 0 & \check{R} & \gamma & -\mu_h - \sigma\check{V} & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & -\Gamma & \Delta & 0 & -\mu v - \Gamma & 0 & \Delta & 0 \\
0 & 0 & 0 & (1-q)\check{R} & 0 & (1-q)\sigma\check{V} & 0 & \chi & 0 & 0 \\
0 & 0 & (1-q)\sigma\check{V} & 0 & 0 & 0 & (1-q)\check{R} & 0 & \chi & 0 \\
0 & 0 & q\sigma\check{V} & q\check{R} & 0 & q\sigma\check{V} & q\check{R} & 0 & 0 & \chi
\end{bmatrix}
$$

where $\Gamma = A(I^{**} + Y^{**})$, $\Delta = A(1 - 2V^{**})$, $\check{V} = BV^{**}$, $\check{R} = \sigma BR^{**}$, $\check{S} = BS^{**}$, $\chi = -\mu_h - \gamma$.

The eigenvalues of $D_{E_{3_a}}$ are $-\mu_h - \gamma$ and the roots of polynomial

$$
q_1 = s^4 + c_1 s^3 + c_2 s^2 + c_3 s + c_4
$$

and

$$
q_2 = s^5 + k_1 s^4 + k_2 s^3 + k_3 s^2 + k_4 s + k_5,
$$

where c_i, $i = (1, 2, 3, 4)$ and k_j, $j = 1, 2, 3, 4, 5$ are functions of the parameters shown in Table 1 (we omit the details). Using Descartes rule of sign [1] for the coefficient of polynomials q_1 and q_2 we have that all the eigenvalues have negative real part if and only if $2V^{**} - 1 < 0 \Longleftrightarrow V^{**} < \frac{1}{2}$, where V^{**} is a positive solution of equation (3.8). This condition is satisfied if

$$
\begin{aligned}
V^{**} = \frac{-b + \sqrt{b^2 - 4ac}}{2a} < \frac{1}{2} &\iff -a - b - 4c < 0, \\
&\iff -4c < a + 2b,
\end{aligned}
$$

$$\begin{aligned}
&\iff -4\mu_h\Lambda(1-T) < a+2b,\\
&\iff 0 < T-1 < \frac{a+2b}{4\mu_h\Lambda},\\
&\iff 1 < T < \frac{B(B\sigma\mu_v + 2A\mu_h^2 + \Lambda(2+\sigma))}{2\mu_h\Lambda} + 1,\\
&\qquad \Lambda = \mu_h\mu_v(\mu_h+\gamma).
\end{aligned}$$

where a, b, and c are coefficients of equation (3.8). This proves Theorem 3.3. ∎

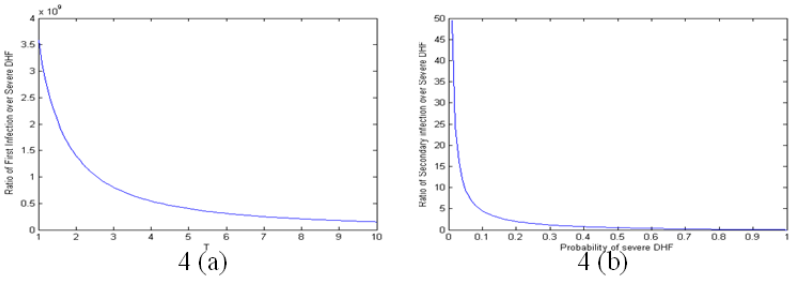
Now, we are interested in ratio between severe DHF compartment over first infection compartment and secondary infection compartment. Those ratios explain the evidence for "ice-berg" phenomena of dengue fever cases [8]. Moreover, they can be used for practical control measure in order to predict the "real" intensity of the endemic phenomena using data of severe DHF given in the hospital. From equation (3.9) we have

$$\frac{I^{**}}{D^{**}} = \frac{\lambda(\sigma B V^{**} + \mu_h)}{2\sigma\gamma q T V^{**}},$$

where V^{**} is a positive solution of equation (3.8) and

$$\frac{Y^{**}}{D^{**}} = \frac{(1-q)}{2q}.$$

In Figure 4(a), we show that the ratio of severe DHF compartment will decrease as the type reproductive number increase, and in Figure 4(b) we have that if the probability of severe DHF is greater than $\frac{1}{3}$ then the ratio of secondary infection compartment over severe DHF compartment will be less than one. Analytically that the ratios tend to infinity as q tends to 0, it means that there is no infection host move to severe DHF compartment.



4 (a) 4 (b)

Figure 4. The diagram of ratio between first infection over severe DHF compartment as the parameter T decreases in Figure 5(a) and the ratio between secondary infection over severe DHF compartment for under fix parameter $\gamma = 0.071, \beta_1 = 0.35, \beta_2 = 0.37, \alpha_1 = 0.17, \alpha_2 = 0.15, b = 1, \sigma_1 = 1.5, \sigma_2 = 2.5$.

4. Numerical simulation

In order to illustrate the dynamics of each epidemic, numerical simulation are carried out using MATLAB routines with different values of the parameters implied in this model. We have generated simulations of system (2.3) for different values of parameters. The typical behaviour or solutions is illustrated in Figures 5 and 6.

Figures 5 and 6 show some sensitivity analysis of the dynamics by varying the susceptibility index and type reproductive number, respectively. The results in Table 3 indicate that the susceptibility index (σ) increases as well as the dynamics of I, Y, and D increase with respect to time. In Table 4, we observe that the type reproductive number increase have an impact to the dynamics of I, Y, and D. In all of the simulations the total population $N_h = 1000$, and the initial number of infected host for each serotype equal to one.

Table 3. Numerical result for dynamic of host and outbreaks time in Figure 6

Fig. no	I^{**}	Y^{**}	D^{**}	t_I	t_Y	t_D	σ
6(a)	0.0623	0.0045	0.002	51.85	66.81	66.81	0.5
6(b)	0.065	0.0157	0.0071	54.18	68.84	68.84	1.5
6(c)	0.0679	0.0304	0.0139	52.63	68.76	68.76	2.5
6(d)	0.0708	0.0496	0.0226	53.72	69.01	71.18	3.5

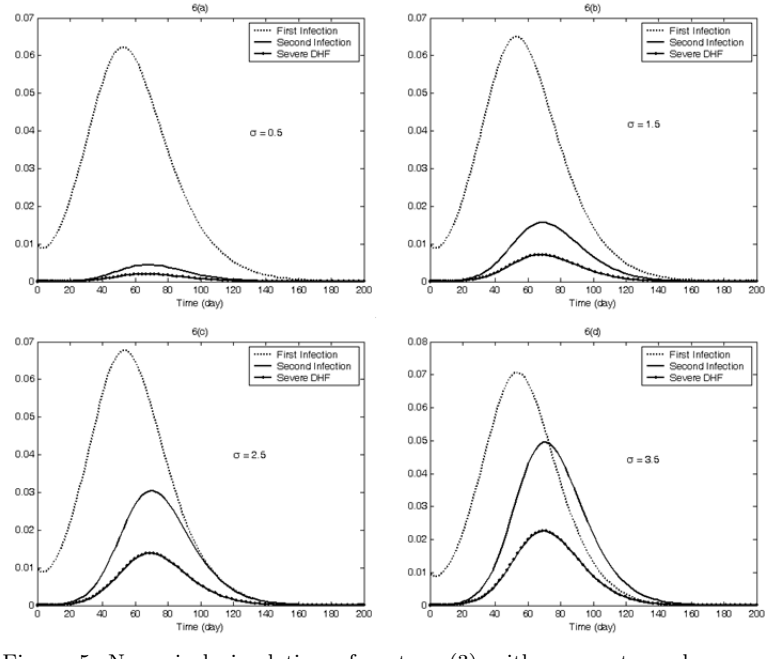


Figure 5. Numerical simulation of system (3) with parameter values $\gamma = 0.071, \beta_1 = 0.1, \beta_2 = 0.1, \alpha_1 = 0.2, \alpha_2 = 0.2, b = 3$, and different values of σ.

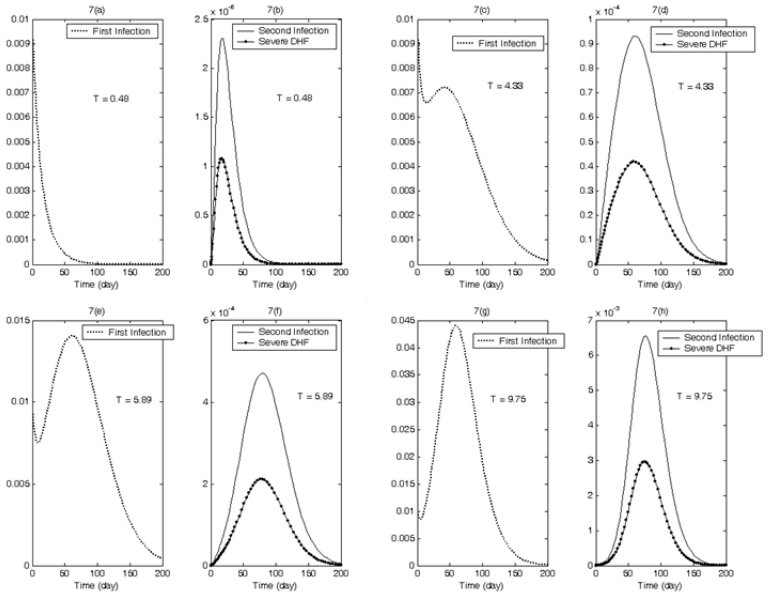


Figure 6. Numerical simulation of system (3) with parameter values $\gamma = 0.071, \beta_1 = 0.1, \beta_2 = 0.1, \alpha_1 = 0.2, \alpha_2 = 0.2, b = 3$, and different values of σ.

Table 4. Numerical result for dynamic of host and outbreaks time in Figure 7

Fig. no	I^{**}	Y^{**}	D^{**}	t_I	t_Y	t_D	T
7(a) and 7(b)	0.01	2.3×10^{-6}	1.1×10^{-6}	-	16.85	16.85	0.48
7(c) and 7(d)	0.01	9.3×10^{-5}	4.2×10^{-5}	-	62.02	57.77	4.33
7(e) and 7(f)	0.01	4.7×10^{-4}	2.1×10^{-4}	61.7	78.92	78.92	5.89
7(g) and 7(h)	0.044	0.0066	0.003	60.5	77.01	74.28	9.75

5. Conclusion

We obtain the value of type reproductive number for the model (2.3) as

$$T_i = \frac{A_i B_i}{\mu_v(\mu_h + \gamma)}, i \in 1, 2,$$

where T_1 for serotype one, and T_2 for serotype two. Analysis of this model reveals the existence of four equilibrium points. One is the disease-free equilibrium and it is locally asymptotically stable if and only if $T_i < 1$. The other two equilibria for one serotype only, are locally asymptotically stable when

$$T_i > 1 \text{ and } T_j < \frac{T_i}{1 + \dfrac{\gamma\sigma_j B_i(1-q)(T_i-1)}{(\mu_h T_i + B_i)(\mu_h + \gamma)^2}}, i, j = 1, 2, i \neq j.$$

The fourth equilibrium is the coexistence of two serotype viruses, we propose the same characteristic of transmission virus for serotype one and serotype two and this

equilibrium will be a locally asymptotically stable endemic point if and only if

$$1 < T < \frac{B(B\sigma\mu_v + 2A\mu_h^2 + \Lambda(2+\sigma))}{2\mu_h\Lambda} + 1, \Lambda = \mu_h\mu_v(\mu_h + \gamma).$$

We obtain the ratio between the total number of severe DHF compartment and the total number of first infection compartment given as

$$\frac{I^{**}}{D^{**}} = \frac{\lambda(\sigma BV^{**} + \mu_h)}{2\sigma\gamma qTV^{**}},$$

where V^{**} is a positive solution of equation (3.8). The ratio between the total number of severe DHF compartment and the total number of secondary infection compartment is

$$\frac{Y^{**}}{D^{**}} = \frac{(1-q)}{2q}.$$

The numerical simulation indicates that the dynamic of infection host will increase until it reaches a maximum number in outbreaks time, after some time the cases exponentially decay approaching the disease-free equilibrium.

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