MATHEMATICAL MODEL OF DENGUE FEVER
AND ITS SENSITIVITY ANALYSIS

Tarig Mohamed Ali[§], Mohamad Faisal Abd Karim
and **Anton Abdulbasah Kamil**
Mathematics Section, School of Distance Education,
Universiti Sains Malaysia, 11800 Minden, Penang
[§] Corresponding author

ABSTRACT

In this paper, a deterministic mathematical model describing the dynamics of dengue fever is presented. The human population is subdivided into three classes; the susceptible, infected, and recovered humans. The mosquitoes subdivided into three classes of the aquatic, susceptible, and infected mosquitoes. The basic dynamics and the reproduction number is calculated and analyzed. The sensitivity analysis of the deterministic mathematical model is carried out in order to establish the relative significance of the model parameters to the disease spread and control. Numerical analysis is carried out to show the dynamics of the disease.

KEYWORDS

1. INTRODUCTION

Dengue fever (DF) and Dengue Haemorrhagic Fever (DHF) are major public health problems in the tropic and subtropics areas [1]. Dengue viruses are transmitted to human by the bite of *Aedes aegypti* female mosquitoes causing Dengue fever (DF) [2]. Sequential infection with dengue fever increases the risk of Dengue Haemorrhagic Fever (DHF). Female *Aedes aegypti* get infection by taking a blood meal from an infected human. These infected mosquitoes transmit the pathogen to susceptible humans [3]. Four different serotypes that can cause dengue fever (DEN-1- 4) can coexist in many endemic areas [4, 5].Infection with one of dengue serotype has been shown to provide life-long immunity to that serotype but not or only short-term resistance to the other serotypes [6].

In recent decades the occurrence of the disease has grown-up significantly around the globe. Currently about 40% of the humanity are at the moment at risk from the disease. With human infectivity estimated at about 50–100 million dengue cases globally each year by WHO. Only nine nations had experienced severe dengue epidemic before 1970. The disease is currently prevalent in more than 100 nations in Africa, the Americas, the Eastern Mediterranean, South-east Asia and the Western Pacific. Generally, the critically affected nations are the Americans, South-east Asia and the Western Pacific. Cases across these nations have surpasses 1.2 million in the year 2008 and more than 2.3 million in the year 2010 [7]. An approximate 500 000 humans with severe dengue need

hospitalization every year, a huge percentage of whom are children. About 2.5% of infected humans die [8].

In the year 2013, 2.35 million cases of the disease were reported in the Americas only. Out of these cases 37687 were severe dengue. The number of cases is greater than ever as the disease stretches to new areas, but explosive epidemics are happening. The risk of a likely epidemic of the disease at the present exists in Europe and local spread of the disease was reported for the first time in France and Croatia in 2010 and imported cases were identified in three other European countries [9].

The main carriers and multipliers of the virus are the infected humans. They serve as a source of the virus for susceptible mosquitoes. The infected humans can transmit the infectivity for a period 4–5 days; utmost 12 days through *Aedes aegypti* following the appearance of the earliest symptoms [10].The vector exists in metropolitan environments and breeds predominantly in artificial containers. The *Aedes aegypti* is a daytime feeder unlike other vectors. And their height biting periods are early in the dawn and in the sunset before dusk [11].

Derouich et al., (2003) presented the dynamics of the dengue fever studied by a compartmental model. This involves ordinary differential equations for the human and the mosquito populations. The result of the simulations shows that the strategy should be based on the prevention of disease epidemic using vector control through environmental management. For instance, eliminate the larval resting places such as containers like bottles, limp of canned foods, tire or other objects susceptible to keep water. Others are the use of chemical methods (application of insecticides) which remains inadequate since it only allows delaying the outbreak of the epidemic. They concluded that, an intermediary solution would be to combine as much as possible, the environmental prevention and a partial vaccination essentially to avoid the haemorrhagic form of the disease caused by different viruses. However, in the humans and mosquitoes immigrations are not considered [12].

Nuraini et al., (2007) presented An SIR model for dengue fever spread. The authors were interested in deriving and analyzing the model taking into account the severe DHF compartment in the model. They aimed at finding a control strategy to decrease the DHF patients in the population, or to keep the number of patients at a tolerable level. The analysis of this model reveals that there are four equilibriums. One of them is the disease-free; the other three equilibriums correspond to the presence of single serotype and the coexistence of two serotypes. The authors concluded that from the numerical simulation, the dynamic of disease host will increase until it reaches a higher number in epidemic time. After several time the cases exponentially decay close to the disease-free equilibrium. However, infected and infectious human should be a class and so also the infected and infectious mosquitoes [1].

Pongsumpun (2008) developed a mathematical model for the spread of dengue fever. The model subdivided the human population into four classes these are the susceptible, infected, infectious and recovered humans. The mosquitoes subdivided into three classes of the susceptible, infected and infectious mosquitoes. The authors analyzed the model via the dynamical study technique. The model is an extension of the earlier model developed by Esteva and Vargas [5]. The earlier model did not include the inherent and

extrinsic incubation periods of dengue virus in humans and mosquitoes. The author concluded that the humans ought to protect themselves from infection with the virus by using bed-nets to diminish the infectivity rate of the mosquitoes. This will reduce the basic reproductive number to decrease below unity. Thus, this can reduce the outbreak of the disease. However, infected and infectious human should be a class and so also the infected and infectious mosquitoes [13].

Garba et al., (2008) presented a deterministic model for the transmission dynamics of a strain of dengue fever. This permits transmission by exposed humans and mosquitoes. The model subdivided the human population into four classes which are the susceptible, exposed, infectious and recovered humans. The mosquitoes are subdivided into three classes of the susceptible, exposed and infectious mosquitoes. The models, which reasonably adopt a standard incidence formulation, allow the disease transmission by exposed humans and vectors. The model was extended to take account of an imperfect vaccine for the disease. The two models, jointly with their mass action equivalents, were severely analyzed to gain insights into their qualitative dynamics. However, susceptible and exposed humans should be a class (the susceptible) and so also the susceptible and exposed mosquitoes [3].

Surapol et al., (2011) proposed and analyzed the dynamical transmission of Dengue fever by considering the role of human population without immunity. The authors found that there are two equilibrium states, a disease-free state and endemic state. They concluded that reducing human contact with the mosquitoes will decrease the basic reproductive number to less than one. Therefore, this will reduce human vulnerability to the disease and, in turn, this can reduce the outbreak of the disease. However, the model does not put into account the recovered humans [14].

Amaku *et al.,* [15], developed a model for dengue fever, this considered the human population, the adult mosquito population and the population of immature stages. It includes eggs, larvae and pupae. The model also considered the vertical transmission of dengue in the mosquitoes and the seasonal variation in the mosquito population. The authors concluded that vector control measures, such as adulticide application is largely efficient. And followed by the reduction of the contact to mosquito bites, locating and eliminating the breeding sites and ultimately larvicides. Existing vector-control strategies are focused on mechanical eradication of mosquitoes' breeding places. Other ideas by the authors is that decreasing the contact rate between vector and hosts (biting rates) is as capable as logistically complicated but very efficient adult mosquito's control. However, in the mosquito compartment, the latent and susceptible mosquitoes can be grouped in the same compartment.

Rodrigues *et al.,* [16] developed a SIR and ASI model illustrating a dengue disease spread. A sensitivity analysis of the model is carried out in order to establish the comparative significance of the model parameters to the disease spread. The authors concluded that the analysis can provide essential information for decision makers and public health officials. The authors believed that the research path initiated will be of immense benefit to the affected citizens. It can also have impact on both the prevention and control of the disease. However, the authors did not consider the recruitment rate of humans and mosquitoes.

In this paper an epidemiology model for dengue disease is proposed based on [16]. The model considered in this study offers some extensions to the dengue transmission model in [16]. This is done by incorporating the recruitment rate of humans and mosquitoes and the maturation rate of the infected larva to adult mosquitoes. It consists of six mutually-exclusive compartments involving the interactions between humans and mosquitoes. It is mathematically written as a system of ordinary differential equations. The model is designed to describe the dynamics of the disease transmission into a population and to perform the sensitivity analysis for the model parameters.

2. METHODOLOGY

2.1 Formulation of the model

The model is based on monitoring the dynamics of the populations of susceptible humans (S_h), infected humans (I_h) recovered human's (R_h) aquatic stage or larva mosquitoes (L_m), uninfected female mosquitoes (M_s), and infected female mosquitoes (M_i), as described in the following subsections. Here, the total human population (N_h) is considered as, $N_h = S_h + I_h + R_h$ at any time t. The population is homogenous, which means that every individual of a compartment is homogenously mixed with the other individuals.

Similarly, the total adult mosquito population is considered as, $N_m = M_s + M_i$. Transition from each state is governed by a set of probabilities or transition rates for each individual. It is assumed homogeneity between hosts and vector populations means that each vector has an equal probability to bite any host. Humans and mosquitoes are assumed to be born susceptible. Schematic illustration of the model is depicted in figure 1
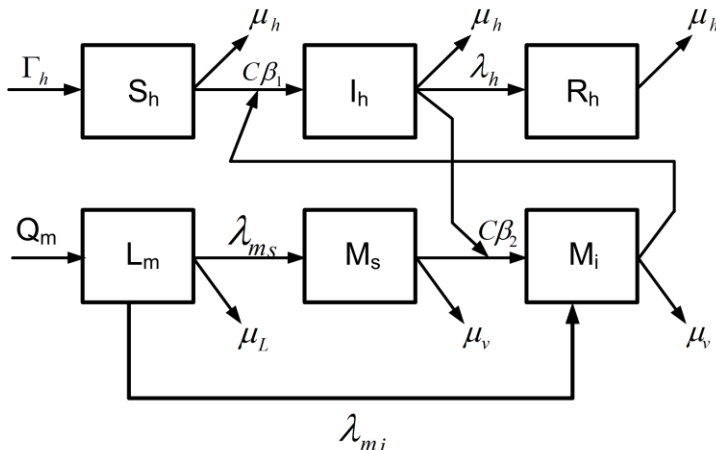
Fig. 1: Schematic Diagram of the Dengue Model

2.1.1 Human Populations

The population of uninfected (susceptible) humans is increased via recruitment of humans (by birth or immigration) into the community at a constant rate (Γ_h). It is decreased either by the contact rate $(C\beta_1)$ between infectious mosquitoes and susceptible hosts, or due to natural death at a rate μ_h. The flow can be represented using the differential equation:

$$\frac{dS_h}{dt} = \Gamma_h - \left(C\beta_1 \frac{M_i}{N_h} + \mu_h \right) S_h \tag{1}$$

Infected humans are increased by the contact rate $(C\beta_1)$ between infectious mosquitoes and susceptible hosts and diminishes either by natural death at a rate (μ_h) or viremic period rate (λ_h).

$$\frac{dI_h}{dt} = C\beta_1 \frac{M_I}{N_h} S_h - (\lambda_h + \mu_h) I_h \tag{2}$$

The infected humans who recover move into the recovered population at rate (λ_h). And the recovered populations are decreased by the natural death at a rate (μ_h). The above assumption gives:

$$\frac{dR_h}{dt} = \lambda_h I_h - \mu_h R_h \tag{3}$$

2.1.2 Mosquitoes Populations

The population of aquatic state (larva compartment) mosquitoes is generated by the birth rate (Q_m) of (susceptible mosquitoes M_s and infected mosquitoes M_i). It decreases either by the maturation rate from (susceptible and infected) larva to adult at a rate $(\lambda_{ms} + \lambda_{mi})$ or natural mortality of larva at a rate (μ_L). Thus

$$\frac{dL_m}{dt} = Q_m \left(1 - \frac{L_m}{fN} \right)(M_s + M_i) - (\lambda_{ms} + \lambda_{mi} + \mu_L) L_m \tag{4}$$

The population of uninfected (susceptible) female mosquitoes is generated by the maturation rate from larva to adult at a rate (λ_{ms}). It diminishes by contact rate $(C\beta_2)$ between infectious humans and susceptible mosquitoes, and natural death at a rate (μ_v). So that,

$$\frac{dM_s}{dt} = \lambda_{ms} L_m - \left(C\beta_2 \frac{I_h}{N_h} + \mu_v \right) M_s \tag{5}$$

The infected female mosquito population is generated by maturation rate of the infected larva to adult at rate λ_{mi} and through the infection of susceptible mosquitoes contact with infected humans $(C\beta_2)$ and it diminishes by natural death at a rate (μ_v). This yield

$$\frac{dM_i}{dt} = \lambda_{mi}L_m + C\beta_2 \frac{I_h}{N_h}M_s - \mu_v M_i \tag{6}$$

The state variable and the parameters of the model are described in Table 1 and 2 respectively.

Table 1
State Variables of the Model

State Variable	Description
S_h	Susceptible humans
I_h	Infected humans
R_h	Recovered humans
L_m	Aquatic stage
M_s	Susceptible mosquitoes
M_i	Infected mosquitoes

Table 2
Describes the Model Parameters with their Values

Parameter	Interpretation	Parameter Value	Reference
N_h	Total human population	480×10^3	[17]
C	Average daily biting (per day)	0.8	[18]
β_1	Transmission probability from infected mosquito to human (per bite)	0.375	[17]
β_2	Transmission probability from infected human to mosquito (per bite)	0.375	[17]
Γ_h	Recruitment rate of humans	30	[19]
$1/\mu_L$	Natural mortality of larva (per day)	4	[17]
λ_{ms}	Maturation rate from susceptible larva to adult mosquitoes	0.08	[17]
λ_{mi}	Maturation rate from infected larva to adult mosquitoes	0.01	Assumed
$1/\lambda_h$	Mean viremic period (in days)	3	[17]
Q_m	Recruitment rate of mosquitoes	400	[19]
$1/\mu_h$	Average lifespan of humans	71*365	[17]
$1/\mu_v$	Average lifespan of adult mosquitoes (in days)	10	[17]

2.1.3 Model Analysis

In this section, model (1)-(6) will be analyzed in order to understand the dynamics of the dengue disease spread. The thresholds parameters which determine persistence or elimination of dengue disease will be identified and studied. Thus the invariant region of the model will be represented, and the solutions of the model (1)-(6) will be shown to be positive for all t >0.

For obtaining the invariant region, system (1)-(6) can be written as follows:

$$\frac{dX}{dt} = M(X)X + F \quad \text{where} \quad \left(X = S_h, I_h, R_h, L_m, M_s, M_i\right), \quad F = \left(\Gamma_h, 0,0,0,0,0\right)^T$$

and

$$M(X) =$$

$$
\begin{bmatrix}
-C\beta_1 \dfrac{M_i}{N_h} - \mu_h & 0 & 0 & 0 & 0 & 0 \\[2ex]
C\beta_1 \dfrac{M_i}{N_h} & -\left(\lambda_h + \mu_h\right) & 0 & 0 & 0 & 0 \\[2ex]
0 & \lambda_h & -\mu_h & 0 & 0 & 0 \\[2ex]
0 & 0 & 0 & \begin{array}{c} -Q_m\left(M_s + M_i\right) \\ -\left(\lambda_{ms} + \lambda_{mi} + \mu_L\right) \end{array} & Q_m & Q_m \\[3ex]
0 & 0 & 0 & \lambda_{ms} & -\left(C\beta_2 \dfrac{I_h}{N_h} + \mu_v\right) & 0 \\[2ex]
0 & 0 & 0 & 0 & C\beta_2 \dfrac{I_h}{N_h} & -\mu_v
\end{bmatrix}
$$

Since $M(X)$ has all off-diagonal entries nonnegative (X) is a Metzeler matrix. By applying the fact that $F \geq 0$, system (1)-(6) is positively invariant in R_+^6 [20] which implies that any trajectory of the system starting from an initial state in the positive or than t R_+^6 remains in R_+^6. In order for the model (1)-(6) to be epidemiology meaningful, we have to prove that all the state variables are positive for all t > 0.

Theorem 1

Let $\left(S_h(0), I_h(0), R_h(0), L_m(0), M_s(0), M_i(0)\right) \geq 0$, then the solution set $\left[(S_h(t), I_h(t), R_h(t), L_m(t), M_s(t), M_i(t)\right]$, of the model (1)-(6) is positive for all $t \geq 0$

Proof:

From the first equation of the system (1)-(6), we have;

$$\frac{dS_h}{dt} = \Gamma_h - \left(C\beta_1 \frac{M_I}{N_h} + \mu_h\right) S_h$$

By putting $\delta = C\beta_1 \dfrac{M_I}{N_h}$, in equation (1) and (2), then,

$$\frac{dS_h}{dt} = \Gamma_h - \left(\delta + \mu_h\right)S_h$$

Thus;

$$\frac{dS_h}{dt} \geq -\left(\delta + \mu_h\right)S_h \text{ It then follows that;}$$

$$\frac{dS_h}{S_h} \geq -(\delta + \mu_h)dt$$

By separating the variables and integrate, it gives

$$S_h \geq S_h(0)e^{-(\delta + \mu_h)t} > 0 \text{ if and only if } \left(\delta + \mu_h\right) > 0$$

Similarly, we can generalize for the other compartments of the model (1)-(6), and get the same result. Then it can be shown that the state variables of the model (1)-(6), are all positive for all $t > 0$.

2.2 Equilibrium Points and Stability

In this section, the equilibrium points of the system (1)-(6), and their stabilities are identified. The equilibrium point is said to be a disease free equilibrium (DFE); if there is no disease for both humans and mosquitoes that is $\left(I_h = M_i = 0\right)$ otherwise if, $\left(I_h \neq 0, orM_i \neq 0\right)$ then the equilibrium points is said to be endemic (EE) [17].

Let $E\left(S_h, I_h, R_h, L_m, M_s, M_i\right)$ be the equilibrium points of the system (1) - (6).

The disease free equilibrium points of the model (1)-(6) is obtained by considering;

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dL_m}{dt} = \frac{dM_s}{dt} = \frac{dM_i}{dt} = 0$$

By using maple software it was found that the disease frees equilibrium points:

$$E_0\left(S_h, I_h, R_h, L_m, M_s, M_i\right) = \left(\frac{\Gamma_h}{\mu_h}, 0, 0, \frac{Q_m}{Q_m + \mu_L + \lambda_{ms}}, \frac{\lambda_{ms}Q_m}{\mu_v\left(Q_m + \lambda_{ms} + \mu_L\right)}, 0\right)$$

The endemic equilibrium (EE) of the model (1)-(6) is given by:

$$E_i = \left(S_h^*, I_h^*, R_h^*, L_m^*, M_s^*, M_i^*\right) \neq 0$$

2.2.1 Basic Reproduction Number R_0

The basic reproduction number denoted by R_0 is the one of the most useful threshold parameters in epidemiology models. This defines the expected number of secondary

cases produced, in a completely susceptible population, by a typical infective individual [21]. In order to evaluate the stability of disease free equilibrium (DFE), and the endemic equilibrium (EE), the computation of R_0 is required.

The local stability of the disease free equilibrium point E_0 can be found by applying the next generation operator approach [21], on the system (1)-(6). The matrices F and V of new infection and of transition terms associated with the system (1)-(6), respectively are given by:

$$F = \begin{bmatrix} C\beta_1 \dfrac{M_i}{N_h} S_h \\[2ex] C\beta_2 \dfrac{I_h}{N_h} M_s \end{bmatrix} \quad V = \begin{bmatrix} (\lambda_h + \mu_h) I_h - \lambda_{mi} \\[1ex] \mu_v M_i \end{bmatrix}$$

It follows that the basic reproduction number $R_0 = P(FV^-)$ where P stands for the spectral radius is given by

$$R_0 = \sqrt{\dfrac{C^2 \beta_1 \beta_2 \Gamma_h \lambda_{ms} Q_m}{N_h^2 \mu_h \mu_v^2 (Q_m + \lambda_{ms} + \mu_L)(\lambda_h + \mu_h)}} \tag{7}$$

By applying theorem (2) of [17], we establish the following result:

Theorem 2:
The disease free equilibrium (DFE) of the system (1)-(6), is asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:
Let $F(x)$ and $V(x)$ represents the new infection and the transition terms associated with the model (1)-(6), respectively are given by:

$$F = \begin{bmatrix} C\beta_1 \dfrac{M_i}{N_h} S_h \\[2ex] C\beta_2 \dfrac{I_h}{N_h} M_s \end{bmatrix} \quad V = \begin{bmatrix} (\lambda_h + \mu_h) I_h - \lambda_{mi} \\[1ex] \mu_v M_i \end{bmatrix}$$

Let us consider the Jacobian matrices associated with F and V

$$J_{F(X)} = \begin{bmatrix} 0 & \dfrac{C\beta_1 S_h}{N_h} \\[2ex] \dfrac{C\beta_2 M_s}{N_h} & 0 \end{bmatrix} \quad J_{V(X)} = \begin{bmatrix} \lambda_h + \mu_h & 0 \\ 0 & \mu_v \end{bmatrix}$$

Based on [22] the basic reproduction number $R_0 = P\left(J_{F(X_0)}J_{V^{-1}(X_0)}\right)$ where X_0 is a disease free equilibrium (DFE) and P represents the eigenvalue of the matrix.

Local stability of E_0 is governed by the eigenvalues of the matrix at (DFE)

$$J(E_0) = \begin{pmatrix} -P & \dfrac{C\beta_1\Gamma_h}{N_h\mu_h\mu_v} \\ \dfrac{C\beta_2\lambda_{ms}Q_m}{N_h\mu_v\left(Q_m+\lambda_{ms}+\mu_{L)}\right)\left(\lambda_h+\mu_h\right)} & -P \end{pmatrix}$$

The characteristics equation of the matrix is given by;

$$P^2 - \frac{C\beta_1\Gamma_h}{N_h\mu_h\mu_v} \times \frac{C\beta_2\lambda_{ms}Q_m}{N_h\mu_v\left(Q_m+\lambda_{ms}+\mu_{L)}\right)\left(\lambda_h+\mu_h\right)} = 0$$

$$P_{1,2} = \pm\sqrt{\frac{C^2\beta_1\beta_2\Gamma_h\lambda_{ms}Q_m}{N_h^2\mu_h\mu_v^2\left(Q_m+\lambda_{ms}\mu_L\right)\left(\lambda_h+\mu_h\right)}}$$

Therefore the roots (eigenvalue) of the characteristic equation P_1 and P_2 have negative real parts. According to the principal of linearised stability [23]; the disease free equilibrium is asymptomatically stable for $R_0 < 1$. The above theorem indicates that dengue disease can be eliminated from human and mosquitoes' populations.

3. RESULTS AND DISCUSSION

3.1 Sensitivity Analysis

Sensitivity analysis is very important for mathematical models. It studies the variations of the outputs of a model caused by variations in the inputs. It is used to determine parameters that have a high impact on R_0 and should be targeted by prevention strategies. It also determines which parameters and initial conditions inputs will affect the quantities of interest outputs of the model [24]. Moreover, sensitivity analysis characterizes the response of model outputs to parameter variation [25]. In order to find out the relative importance of the model parameters to dengue disease propagation, analytical sensitivity analysis on all the parameters with respect to basic reproductive number R_0 is performed [26].

3.1.1 Numerical Sensitivity Analysis

By using the approach [27], sensitivity analysis is carried out by computing sensitivity indices of basic reproductive number R_0, which measure initial disease prevalence. Sensitivity indices measures the relative change in state variable when the parameter changes. The normalized forward sensitivity index of a variable to a parameter is a ratio of the relative change in the variable to the relative change in the parameter. When a variable is a differentiable function of the parameter, the sensitivity index may be alternatively defined using partial derivatives [26].

The normalized forward sensitivity index of a variable R_0 that depends differentiable on a parameter P can be defined as [27]:

$$\Psi_P^{R_0} = \frac{\partial R_0}{\partial P} \times \frac{P}{R_0} \tag{8}$$

As given by the formula for R_0 in equation (8), we derive an analytical expression for sensitivity of R_0 as $\Psi_P^{R_0} = \frac{\partial R_0}{\partial P} \times \frac{P}{R_0}$, to each of its parameter. So, the sensitivity index of R_0 with respect to C is given as

$$\Psi_C^{R_0} = \frac{\partial R_0}{\partial C} \times \frac{C}{R_0} = +1.00$$

Other numerical values of sensitivity indices of R_0 obtained using maple software are shown in Table (3).

Table 3
Numerical Values of Sensitivity Indices of R_0

Parameter	Parameter Value	Sensitivity Index
C	0.8	+1.00
μ_v	0.1	-1.00
μ_h	0.00003	- 0.50005
β_1	0.375	+0.5
β_2	0.375	+0.5
Γ_h	30	+0.5
λ_h	0.333	-0.49995
λ_m	0.08	0.49990
Q_m	400	0.00041
μ_L	0.25	-0.00031

3.1.2 Interpretation of Sensitivity Analysis

The most sensitive parameter is the average daily biting C. It then follows by the natural death rate of mosquitoes μ_v, the human natural death rate μ_h, the transmission probability from infected mosquitoes to susceptible host (per bite), β_1 and the transmission probability from infected human to susceptible mosquitoes (per bite), β_2. For these three parameters, the most sensitivity indices to R_0 agree with the result obtained by [16]. Other important parameters include; the recruitment rate of humans Γ_h, followed by the viremic period rate, λ_h and the maturation rate from larva to adult

mosquitoes λ_m. The recruitment rate of mosquitoes Q_m, natural death rate of larva mosquitoes μ_L, is having small impact in R_0.

This result can be interpreted that the increase in daily biting rate C, and the transmission rate from infected mosquitoes to susceptible host β_1, as well as the transmission rate from infected host with susceptible mosquitoes β_2 increases the disease spread through the community. Meanwhile, the decrease in these parameters values through applying intervention strategies, and controlling the rate of incoming infected immigrants into the community will lead to decrease of the reproduction number R_0.

3.2 Numerical Simulations

In order to simulate the dengue disease spread, numerous numerical simulations of the model were carried out using the set of parameters values given in table 1. To introduce a better analysis, all the simulations and graphs are obtained using *Mathematica* software.

Fig 2 and Fig 3 describe the typical behavior of both human and mosquito populations without using any control strategy. It has shown that the number of both infected human and mosquitoes increased and reach maximum value between the 0th and 20th per day. Moreover Fig 4 represents the human and mosquitoes populations with nonexistence of any control measures.
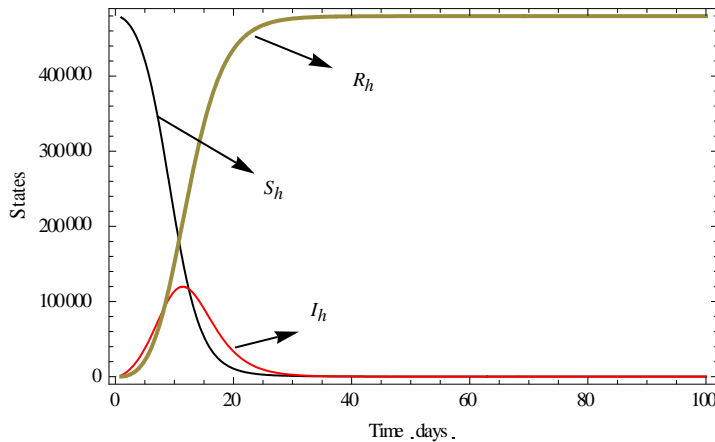
Fig. 2: Human Population without Control Strategy
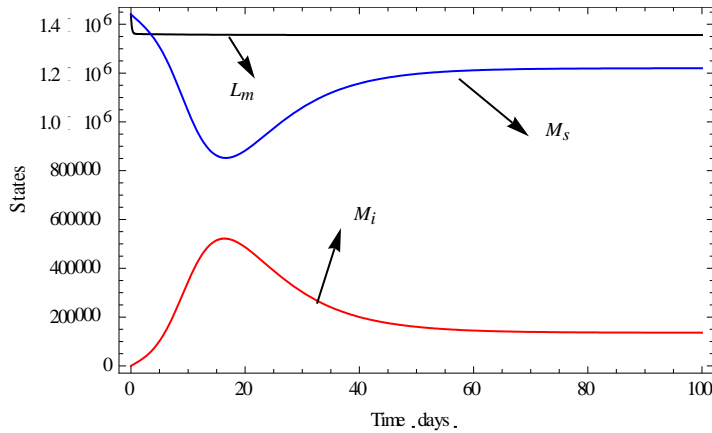
Fig. 3: Mosquito Population without Control Measure
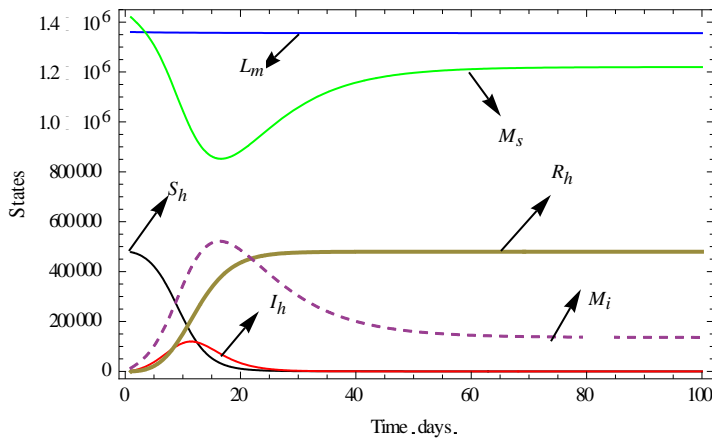
Fig. 4: Human and Mosquito Population without Control Strategy

4. CONCLUSION

A deterministic mathematical model describing the dynamics of dengue fever is studied. The sensitivity index of the basic reproduction number is carried out in order to establish the relative significance of the model parameters in the disease spread. The result of the analysis indicated that the average daily biting C, the natural death rate of mosquitoes μ_v, are the most sensitive. These are followed by human natural death rate μ_h, the transmission probability from infected mosquitoes to susceptible host β_1 and the transmission probability from infected human to susceptible mosquitoes β_2. And the less sensitive is the natural death rate of larva mosquitoes μ_L. This information permits us to recognize the strength of the model forecast with respect to parameter values. It also allows us to acknowledge where to channel the control strategies. The analysis can be

vital for public health workers and policy makers who possibly will have to deal with the certainty of the diseases.

COMPETING INTERESTS

Authors declare that no competing interest exists.

REFERENCES

1. Nuraini, N., Soewono, E. and Sidarto, K.A. (2007). Mathematical model of dengue disease transmission with severe DHF compartment. *Bull. Malays. Math. Sci. Soc.*, 30(2), 143-157.
2. Isidoro, C., Fachada, N., Barata, F. and Rosa, A. (2011). Agent-based model of dengue disease transmission by Aedes aegypti populations. *Lecture Notes in Computer Science*, 5777, 345-352.
3. Garba, S.M., Gumel, A.B. and Abu-Bakar, M.R. (2008). Backward bifurcations in dengue transmission dynamics. *Math. Biosci.*, 215(1), 11-25.
4. Gibbons, R.V., Kalanarooj, S., Jarman, R.G., Nisalak, A., Vaughn, D.W., Endy, T.P., Mammen, M.P. Jr. and Srikiatkhachorn, A. (2007). Analysis of repeat hospital admissions for dengue to estimate the frequency of third or fourth dengue infections resulting in admissions and dengue hemorrhagic fever, and serotype sequences. *Am. J. Trop. Med. Hyg*., 77(5), 910-3.
5. Gubler, D.J. (1998). Dengue and dengue hemorrhagic fever. *Clin. Microbiol. Rev*., 11(3), 480-96.
6. Kumar, K., Singh, P.K., Tomar, K. and Baijal, S. (2010). Dengue: epidemiology, prevention and pressing need for vaccine development. *Asian Pac. J. Trop Med*., 3(12), 997-1000.
7. Coller, B.A. and Clements, D.E. (2011). Dengue vaccines: progress and challenges. *Curr. Opin. Immunol.*, 23(3), 391-8.
8. Helmersson, J. (2012). Mathematical Modeling of Dengue-Temperature Effect on Vectorial Capacity. Master thesis, Umeå University.
9. Schaffner, F., Medlock, J.M. and Van Bortel, W. (2013). Public health significance of invasive mosquitoes in Europe. *Clin. Microbiol. Infect*., 19(8), 685-92.
10. Brower, J. and Chalk, P. (2003). *The global threat of new and reemerging infectious diseases: reconciling US national security and public health policy*. Rand Corporation.
11. Organization, W.H. (2012). *Dengue and Severe Dengue*. Fact sheet. 117.
12. Derouich, M., Boutayeb, A. and Twizell, E.H. (2003). A model of dengue fever. *Biomed. Eng. Online*., 2(1), 4.
13. Pongsumpun, P. (2008). Mathematical model of dengue disease with the incubation period of virus. *World Acad Sci Eng Technol*. 44, 328-32.
14. Naowarat, S., Korkiatsakul, T. and Tang, I.M. (2011). Dynamical Model for Determining Human Susceptibility to Dengue Fever. *Am. J. Appl. Sci*., 8(11), 1101.
15. Amaku, M., Coutinho, F.A., Raimundo, S.M., Lopez, L.F., Nascimento Burattini M. and Massad, E. (2014). A comparative analysis of the relative efficacy of vector-control strategies against dengue fever. 76(3), 697-717.

16. Rodrigues, H.S., Monteiro, M.T.T. and Torres, D.F. (2013). Sensitivity Analysis in a Dengue Epidemiological Model, in *Conference Papers in Mathematics*, Hindawi Publishing Corporation.

17. Rodrigues, H.S., Monteiro, M.T.T., Torres, D.F.M. and Zinober, A. (2012). Dengue disease, basic reproduction number and control. *Int. J. Comput. Math.*, 89(3), 334-346.

18. Bowman, C., Gumel, A.B., van den Driessche, P., Wu, J. and Zhu, H. (2005). A mathematical model for assessing control strategies against West Nile virus. *Bull. Math. Biol.*, 67(5), 1107-33.

19. Esteva, L. and Vargas, C. (1988). Analysis of a dengue disease transmission model. *Math. Biosci.*, 150(2), 131-51.

20. Abate, A., Tiwari, A. and Sastry, S. (2009). Box invariance in biologically-inspired dynamical systems. *Automatica*. 45(7), 1601-1610.

21. Hethcote, H.W., (2000). The mathematics of infectious diseases. *Siam Rev.*, 42(4), 599-653.

22. van den Driessche, P. and Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Math. Biosci.*, 180(1), 29-48.

23. Lenka, B. (2007). *The mathematics of infectious disease*. M.Sc. thesis published, Comenius.

24. Mikucki, M.A. (2012). *Sensitivity analysis of the basic reproduction number and other quantities for infectious disease models*. Colorado State University.

25. Wu, J.Y., Dhingra, R., Gambhir, M. and Remais, J.V. (2013). Sensitivity analysis of infectious disease models: methods, advances and their application. *J.R. Soc. Interface*, 10(86), 20121018.

26. Shabani, I., Massawe, E. and Makinde, O. (2010). *Modelling the effect of screening on the spread of HIV infection in a Homogeneous population with infective immigrants*. University of Dar es Salaam.

27. Iddi, A.J. (2011). *Modelling the impact of infected immigrants on vector-borne diseases with direct transmission*. University of Dar es Salaam.