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Effects of Migratory Population and Control Strategies on the Transmission Dynamics of Dengue Virus

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Abstract

In this Paper we formulate a mathematical model of dengue virus transmission in the human body to monitor the effects of migratory population and some control strategies at aquatic and adult stages of vector (mosquito). The model has a locally asymptotically stable disease-free equilibrium (DFE) whenever a certain epidemiological threshold, known as the basic reproduction number (\mathcal{R}_0) , is less than unity. It is also shown, using a Lyapunov function and Lasalle Invariance Principle that the DFE of the dengue model is globally-asymptotically stable (GAS) whenever the reproduction number (\mathcal{R}_0) is less than unity. The model has a locally-asymptotically stable endemic equilibrium point (EEP) whenever $\mathcal{R}_0 \geq 1$. With the help of Lyapunov function and Lasalle Principle (Goh-Volterra type), by considering special case, the EEP of the model is shown to be GAS whenever $\mathcal{R}_0 \geq 1$. The model simulations reveals that the migratory infected individuals increases the burden of the dengue disease and also precautionary measures at the aquatic and adult stages decrease the number of new cases of dengue

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virus. Numerical simulation indicates that if we take the precautionary measures effectively then it would be more effective then even giving the treatment to the infected individuals.

Keywords: Aedes aegyptic; Dengue Fever; Mathematical modeling; Equilibria; Local and Global Stability; Reproduction number; Migratory population; Control strategy

1 Introduction

Dengue is endemic in more than 110 countries [26, 7, 37, 39]. It infects 50 to 390 million people worldwide a year, leading to half a million hospitalizations [35, 38, 37, 36], and approximately 25,000 deaths [34, 39], For the decade of the 2000s, 12 countries in Southeast Asia were estimated to have about 3,000,000 infections and 6,000 deaths annually [34]. In the United States the rate of dengue infection among those who return from an endemic are with a fever is 3-8% [39, 37].

Dengue fever, is an infectious tropical disease caused by the dengue virus. Dengue is transmitted by several species of mosquito within the genus Aedes, principally *Aedes aegypti*. The virus has four different types [7, 16, 9, 23], but only short-term immunity to the others. Subsequent infection with a different type increases the risk of sever complications. The incidence of dengue fever has increased dramatically since the 1960s, Dengue has become a global problem since second World War.

The incubation period (time between exposure and onset of symptoms) ranges from 3-14 days, but most often it is 4-7 days [37, 21, 39]. Therefore, travellers returning from endemic area are unlikely to have dengue if fever or other symptoms start more than 14 days after arriving home [2, 39]. According to the World Tourism Organization, in 2004, 125.4 million international tourists visited countries where they might be at risk for acquiring infection 7%-45% travellers [40]. With approximately two billion people living in tropical and subtropical regions of the world, and an additional roughly 120 million people each year [40] travelling to these region, a large share of the world's

population is at risk of contracting dengue.

The burden of dengue in Africa remains poorly understood. Travellers and military personnel visiting or stationed in Africa have been identified as having laboratory-confirmed dengue infections, indicating that the virus is circulating [39, 37]. One billion people(15% of the world's populations) reside in India. India's population is twice that of south-east Asia, the region that currently reports the most dengue related deaths [39]. According to the WHO, South-East Asia Region, the majore public health problem in Bangladesh is dengue. In Indonesia dengue is hyperendemicity with all four serotypes circulating in urban areas [39, 2].

Several mathematical models have been developed in the literature to gaininsights into the transmission dynamics of dengue in a community [23, 20, 21, 22, 7, 6, 5, 25, 4, 24, 14, 41, 15]. In this paper we extended some of the earlier models by considering the effects of migrated individuals and some other control effects of the vectors. To control the dengue virus effectively and to find the effects of migratory population , we should understand the dynamics of the disease transmission and take into account all of the relevant details, such as the dynamics of the human population and vector. For a realistic model, we consider some special classes like migratory class, treatment class and vector aquatic class. We also present and analyze some control rate parameters, that will help to find the effective control strategies of the diseases. We present and analyze a non-linear ODE model that incorporates ten mutually-exclusive classes. Numerical simulations results are presented to support the analytical conclusion followed by conclusion of the present work.

2 Model Formulation

The dengue virus follows two main modes of transmission: human to mosquito and mosquito to human [8, 9, 35]. The model assumes a homogenous mixing of the human and vector (mosquito) populations, so that each mosquito bite has equal chance of transmitting the virus to susceptible human in the population (or acquiring infection from an infected human). The total number of individuals at time t, denoted by $N_H(t)$, is sub-divided into six mutually-exclusive sub-populations of susceptible humans $S_H(t)$, exposed humans $E_H(t)$, infectious humans $I_H(t)$, migrated population $M_H(t)$, treatment class $T_H(t)$ and recovered humans $R_H(t)$, so that $N_H = N_H(t) = S_H(t) + E_H(t) + I_H(t) + M_H(t) + T_H(t) + R_H(t)$.

Similarly, the total vector population at time t, denoted by $N_V(t)$, is subdivided into aquatic class $A_V(t)$, susceptible mosquitoes $S_V(t)$, exposed mosquitoes $E_V(t)$, infectious mosquitoes $I_V(t)$, so that $N_V = N_V(t) = A_V(t) + S_V(t) + E_V(t) + I_V(t)$. The susceptible human population is generated via recruitment of humans (by birth) into the community (at a constant rate, π_H). This population is decreased following infection, which can be acquired via effective contact with an exposed or infectious vector at a rate λ_H the force of infection of humans given by

$$\lambda_H = \frac{C_{HV}(\eta_V E_V + I_V)}{N_H}; 0 < \eta_V < 1$$
(1)

where the modification parameter $0 < \eta_V < 1$ accounts for the assumed reduction in transmissibility of exposed mosquitoes relative to infectious mosquitoes [7].

The functional forms of the incidence functions associated with the transmission dynamics of dengue disease will be derived. The derivation is based on the basic fact that for mosquito-borne diseases (such as dengue), the total number of bites made by mosquitoes must equal the total number of bites received by humans [1]. Since mosquitoes bite both susceptible and infected humans, it is assumed that the average number of mosquito bites received by humans depends on the total sizes of the populations of mosquitoes and humans in the community. It is assumed that each susceptible mosquito bites an infected human at an average biting rate, b_S , and the human hosts are always sufficient in abundance, so that it is reasonable to assume that the biting rate, b_S , is constant. Let,

$$C_{HV} = \rho_{HV} b_S, \tag{2}$$

be the rate at which mosquitoes acquire infection from infected humans (exposed or infectious), where ρ_{HV} is the transmission probability from an infected human to a susceptible mosquito and b_S is the biting rate per susceptible mosquito, so that C_{HV} is a constant. Similarly, let

$$C_{VH} = \rho_{VH} b_I \tag{3}$$

be the rate at which humans acquire infection from infected mosquitoes (exposed or infectious), where ρ_{HV} is the transmission probability from an infected mosquito to a susceptible human and b_I is the average biting rate per infected mosquito. Thus, for the number of bites to be conserved, the following equation must hold,

$$C_{HV}N_V = C_{HV}N_H \tag{4}$$

so that,

$$N_V = \frac{C_{VH}(N_H, N_V)N_H}{C_{HV}} \tag{5}$$

therefore,

$$\lambda_{H} = \frac{C_{HV}(\eta_{V}E_{V} + I_{V})}{N_{H}}, 0 < \eta_{V} < 1$$
(6)

Similarly, it can be shown that the force of infection of mosquitoes (denoted by λ_V) is given by,

$$\lambda_V = \frac{C_{HV}(\eta_H E_H + I_H)}{N_H}; 0 < \eta_H < 1$$
(7)

where the modification parameter $0 < \eta_H < 1$ accounts for the relative infectiousness of exposed humans in relation to infectious humans.

Let π_H is the recruitment of humans into the population (assumed susceptible), λ_H is the infection rate of susceptible humans (which results following effective contact with exposed or infectious mosquitoes) and μ_H is the natural death rate of humans. Exposed humans develop clinical symptoms of dengue disease, and move to the infectious class, at a rate σ_H . We also consider that π_1 is the migratory humans come into the population from which μ_1 is the rate at which this added to the exposed class and μ_2 is the rate at which this added to the infectious class. Infectious humans recover and move into the R_H class at a rate γ_1 and suffer disease-induced death at a rate δ_H . It is assumed that recovered individuals acquire lifelong immunity against re-infection.

The vector population is generated by birth at a rate π_V and γ_m is the mean aquatic transition rate. The aquatic state will move into the susceptible class at a rate γ_m and λ_V is the infection rate of the susceptible vector at which they move in to the exposed class. Exposed vectors develop symptoms of disease and move to the infectious class at a rate σ_V and θ_c is the extrinsic incubation rate of the vector population. Since vector (Aedes aegypt) populations can be controlled in two stages (aquatic stage and adult stage); here C_a is

considered as the controlling parameter in aquatic stage and C_m is considered as controlling parameter in adult stage. Infections vectors die due to disease at a rate δ_V .

The model for the transmission dynamics of dengue in a population is given by the following system of non-linear differential equation:

$$\frac{dS_H}{dt} = \pi_H - \lambda_H S_H - \mu_H S_H,$$

$$\frac{dE_H}{dt} = \lambda_H S_H + \mu_1 M_H - (\mu_H + \sigma_H) E_H,$$

$$\frac{dI_H}{dt} = \sigma_H E_H + \mu_2 M_H - (\tau_H + \mu_H + \delta_H) I_H,$$

$$\frac{dM_H}{dt} = \pi_1 - (\mu_1 + \mu_2 + \mu_H) M_H,$$

$$\frac{dT_H}{dt} = \tau_H I_H - (\mu_H + \gamma_1) T_H,$$

$$\frac{dR_H}{dt} = \gamma_1 T_H - \mu_H R_H,$$

$$\frac{dA_V}{dt} = \pi_V - (\gamma_m + \mu_V + C_a) A_V,$$

$$\frac{dS_V}{dt} = \gamma_m A_V - (\lambda_V + \mu_V + C_m) S_V,$$

$$\frac{dE_V}{dt} = \lambda_V S_V - (\sigma_V + \theta_c + \mu_V + C_m) E_V,$$

$$\frac{dI_V}{dt} = (\sigma_V + \theta_c) E_V - (\mu_V + \delta_V + C_m) I_V.$$
(8)

In summary, the model (8) is an extension of some earlier standard models for vector-borne diseases transmission, such as those in [3, 31, 7, 17, 14, 6, 5, 23, 20, 21, 22, 41, 25, 13, 4, 15], by

- (i) introducing the migrated class $M_H(t)$ to monitor the impact of the migratory human population in dengue transmission dynamics (where the exposed migratory population is added to the exposed class $E_H(t)$ at a rate μ_1 , and the infected migratory population is added to the infective class $I_H(t)$ at a rate μ_2 ;
- (ii) incorporating the treatment class $T_H(t)$ in dengue transmission dynamics;
- (iii) considering the vector-aquatic class $A_V(t)$ to find the effects of the control strategies at the aquatic stage;

(iv) additionally incorporating the controlling rate parameters C_a and C_m which will monitor the effects of precautionary measures at the aquatic stage (C_a) and adult stage (C_m) , respectively.

The variables of the models (8) are described in Table 1 and the parameters value of the model are given in the Table 2. Schematically the model (8) can be shown as follows:

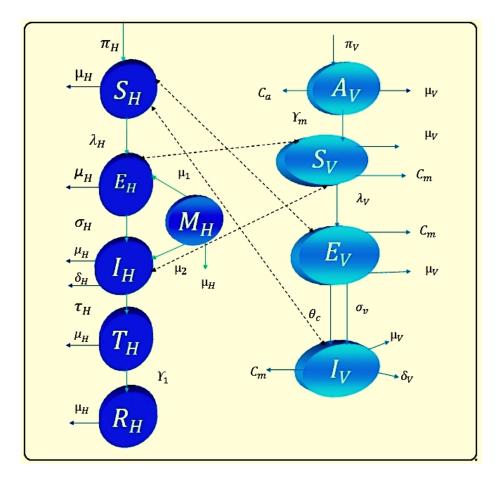


Figure 1: The diagram of the model(8)

2.1 Properties of the Model

Here the basic dynamical feature of the model (8) will be explored. We claim the following.

2.1.1 Positivity and boundedness of solutions

Lemma 1. The closed set $\Omega = \{(S_H, E_H, I_H, M_H, T_H, R_H, A_V, S_V, E_V, I_V) \in \mathbb{R}^{10}_+ : S_H + E_H + I_H + M_H + T_H + R_H \leq \frac{\pi_H}{\mu_H}, A_V + S_V + E_V + I_V \leq \frac{\pi_V}{\mu_V}\}$ is positively-invariant and attracting with respect to the basic model (8)

Proof. Adding the first six equations and the last four equations in the model, respectively, gives:

$$\frac{dN_H}{dt} = \pi_H - \mu_H N_H - \delta_H I_H + \pi_1 \tag{9}$$

and,

$$\frac{dN_V}{dt} = \pi_V - C_a A_V - (S_V + I_V + E_V)C_m - \mu_V N_V - \delta_V I_V$$
(10)

Since $\frac{dN_H}{dt} \leq \pi_H - \mu_H N_H + \pi_1$ and $\frac{dN_V}{dt} \leq \pi_V - \mu_V N_V$, it follows that $\frac{dN_H}{dt} > 0$ and $\frac{dN_V}{dt} > 0$ if $N_H(t) < \frac{\pi_H + \pi_1}{\mu_H}$ and $N_V(t) < \frac{\pi_V}{\mu_V}$, respectively.

Thus, a standard comparison theorem [33] can be used to show that $N_H(t) \leq N_H(0)$

 $exp^{-\mu_H(t)} + \frac{\pi_H + \pi_1}{\mu_H} [1 - exp^{-\mu_H(t)}]$ and $N_V(t) \leq N_V(0) \exp^{-\mu_V(t)} + \frac{\pi_V}{\mu_V} [1 - exp^{-\mu_V(t)}]$. In particular, $N_H(t) \leq \frac{\pi_H + \pi_1}{\pi_H}$ and $N_V(t) \leq \frac{\pi_V}{\mu_V}$ if $N_H(0) \leq \frac{\pi_H + \pi_1}{\mu_H}$ and $N_V(0) \leq \frac{\pi_V}{\mu_V}$, respectively. Thus, Ω is positively invariant. Further, if $N_H(t) < \frac{\pi_H + \pi_1}{\mu_H}$ and $N_V(t) < \frac{\pi_H + \pi_1}{\mu_H}$ and $N_V(t) < \frac{\pi_V}{\mu_V}$, then either the solution enter Ω in finite time, or $N_H(t)$ approaches $\frac{\pi_H + \pi_1}{\mu_H}$ and $N_V(t)$ approaches $\frac{\pi_V}{\mu_V}$, and the infected variable $E_H, I_H, M_H, T_H, E_V, I_V$ approaches zero. Hence, Ω is attracting (i.e., all solution in \mathbb{R}^{10}_+ eventually enter Ω). Thus, in Ω , the basic model (8) is well-posed epidemiologically and mathematically [11]. Hence, it is sufficient to study the dynamics of the basic model in Ω .

2.2 Stability Analysis of the Model

2.2.1 Disease-free equilibrium (DFE)

The basic model (8) has a DFE given by

$$E_{0} = (S_{H}^{*}, E_{H}^{*}, I_{H}^{*}, M_{H}^{*}, T_{H}^{*}, R_{H}^{*}, A_{V}^{*}, S_{V}^{*}, E_{V}^{*}, I_{V}^{*}) \\ = \left[\frac{\pi_{H}}{\mu_{H}}, 0, 0, 0, 0, 0, \frac{\pi_{V}}{(\mu_{V} + C_{a} + \gamma_{m})}, \frac{\gamma_{m}\pi_{V}}{(\gamma_{m} + \mu_{V} + C_{a})(\mu_{V} + C_{m})}, 0, 0)\right]$$

The linear stability of E_0 is studied using the next generation operator technique in [32]. The associated non-negative matrix, F, for the new infection

terms, and the non-singular M - matrix, for the remaining transfer terms, are given, respectively, by

	0	0	0	0	$\frac{C_{HV}\eta_V S_H^*}{N_H^*}$	$\frac{C_{HV}S_H^*}{N_H^*} \bigg]$
F =	0	0	0	0	0	0
	0	0	0	0	0	0
	0	0	0	0	0	0
	$\frac{C_{VH}\eta_H S_V^*}{N_H^*}$	$\frac{C_{VH}S_V^*}{N_H^*}$	0	0	0	0
	0	0	0	0	0	0

and

$$V = \begin{bmatrix} k_1 & 0 & -\mu_1 & 0 & 0 & 0 \\ -\sigma_H & k_2 & -\mu_2 & 0 & 0 & 0 \\ 0 & 0 & k_3 & 0 & 0 & 0 \\ 0 & -\tau_H & 0 & k_4 & 0 & 0 \\ 0 & 0 & 0 & 0 & k_5 & 0 \\ 0 & 0 & 0 & 0 & -(\sigma_V + \theta_c) & k_6 \end{bmatrix}$$

where, $k_1 = \mu_H + \sigma_H$, $k_2 = \tau_H + \mu_H + \delta_H$, $k_3 = \mu_1 + \mu_2 + \mu_H$, $k_4 = \mu_H + \gamma_1 k_5 = \sigma_V + \theta_c + \mu_V + C_m$, $k_6 = \mu_V + \delta_V + C_m$.

The associated basic reproduction number, denoted by \mathcal{R}_0 , is then given by

 $\mathcal{R}_0 = \rho(FV^{-1})$, where ρ is the spectral radius of FV^{-1} . It follows that

$$\mathcal{R}_{0} = \frac{\left[k_{2}k_{1}k_{5}k_{6}C_{1}S_{H}^{*}C_{2}S_{V}^{*}\left\{\eta_{H}\eta_{V}k_{2}k_{6}+\eta_{H}k_{2}\sigma_{V}+\eta_{H}k_{2}\theta_{c}+\sigma_{H}\eta_{V}k_{6}+\sigma_{V}\sigma_{H}+\sigma_{H}\theta_{c}\right\}\right]^{\frac{1}{2}}}{k_{2}k_{1}k_{5}k_{6}N_{H}^{*}}$$

where, $C_1 = C_{HV}$ and $C_2 = C_{VH}$.

Lemma 2. The DFE, E_0 , of the system (8), is locally-asymptotically stable (LAS) if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$.

The threshold quantity, \mathcal{R}_0 , measures the average number of secondary cases generated by a single infected individual (or vector) in a completely susceptible human (vector) population [11, 27]. The above result implies that a small influx of infected individuals (or vector) would not generate large outbreaks if $\mathcal{R}_0 < 1$, and the disease will persist (be endemic) in the population if $\mathcal{R}_0 > 1$. However, in order for disease elimination to be independent of the initial sizes of the sub-populations of the model when $\mathcal{R}_0 < 1$, a global stability property must be established for the DFE when $\mathcal{R}_0 < 1$. This is explored below.

2.2.2 Global Stability of the DFE of Model (8)

We claim the following:

Theorem 1. The DFE, E_0 , of the model (8), is globally-asymptotically stable (GAS) in Ω if $\mathcal{R}_0 < 1$.

Proof.Consider the Lyapunov function

$$\mathcal{F} = f_1 E_H + f_2 I_H + f_3 M_H + f_4 E_V + f_5 I_V,$$

where,

$$\begin{split} f_1 &= \frac{C_2 S_V (\eta_H k_2 + \sigma_H) (\eta_V k_6 + \sigma_V + \theta_c) k_6}{\mathcal{R}_0 (k_1 k_2 k_5 k_6 N_H)}, \\ f_2 &= \frac{C_2 S_V (\eta_V k_6 + \sigma_V + \theta_c) k_1 k_6}{\mathcal{R}_0 (k_1 k_2 k_5 k_6 N_H)}, \\ f_3 &= \frac{C_2 S_V (\eta_H \mu_1 k_2 + \mu_2 k_1 + \sigma_H \mu_1) (\eta_V k_6 + \sigma_V + \theta_c) k_6}{\mathcal{R}_0 (k_1 k_2 k_5 k_6 N_H)}, \\ f_4 &= 0, \\ f_5 &= \frac{\eta_V k_6 + \sigma_V + \theta_c}{k_5}, \\ f_6 &= 1, \end{split}$$

with Lyapunov derivative given by (where a dot represents differentiation with

respect to t)

$$\begin{split} \dot{\mathcal{F}} &= f_1 \dot{E}_H + f_2 \dot{I}_H + f_3 \dot{M}_H + f_4 \dot{T}_H + f_5 \dot{E}_V + f_6 \dot{I}_V, \\ &= f_1 [\lambda_H S_H + \mu_1 M_H - k_1 E_H] + f_2 [\sigma_H E_H + \mu_2 M_H - k_2 I_H] + f_3 [\pi_1 - k_3 M_H] \\ &+ f_3 [\pi_1 - k_3 M_H] + f_5 [\lambda_V S_V - k_5 E_V] + f_6 [\sigma_V E_V + \theta_c E_V - k_6 I_V], \\ &= f_1 \left[\frac{S_H C_1 (\eta_V E_V + I_V)}{N_H} + \mu_1 M_H - k_1 E_H \right] + f_2 \left[\sigma_H E_H + \mu_2 M_H - k_2 I_H \right] \\ &+ f_3 \left[\pi_1 - k_3 M_H \right] + f_5 \left[\frac{C_2 S_V (\eta_H E_H + I_H)}{N_H} - k_5 E_V \right] + f_6 \left[\sigma_V E_V + \theta_c E_V - k_6 I_V \right], \\ &= \frac{C_2 S_V (\eta_H k_2 + \sigma_H) (\eta_V k_6 + \sigma_V + \theta_c) k_6}{\mathcal{R}_0 (k_1 k_2 k_5 k_6 N_H)} \left[C_1 S_H (\eta_V E_V + I_V) + M_H N_H - k_1 E_H N_H \right] \\ &+ \sigma_V E_V + \theta_c E_V - k_6 I_V + \frac{(\eta_V k_6 + \sigma_V + \theta_c)}{k_5 N_H} \left[k_5 E_V N_H + C_2 S_V (\eta_H E_H + I_H) \right] \\ &+ \frac{C_2 S_V (\eta_V k_6 + \sigma_V + \theta_c) k_1 k_6}{\mathcal{R}_0 (k_1 k_2 k_5 k_6 N_H)} \left[\sigma_H E_H + \mu_2 M_H - k_2 I_H \right] \\ &+ \frac{C_2 S_V (\eta_H \mu_1 k_2 + \mu_2 k_1 + \sigma_H \mu_1) (\eta_V k_6 + \sigma_V + \theta_c) k_6}{\mathcal{R}_0 (k_1 k_2 k_5 k_6 N_H)} \left[\pi_1 - k_3 M_H \right] \\ &= k_6 (\eta_V E_V + I_V) \mathcal{R}_0 + \sigma_V E_V + \theta_c E_V - k_6 I_V - E_V \eta_H k_6 - \sigma_V E_V - \theta_c E_V \\ &+ \frac{C_2 S_V (\eta_V k_6 + \sigma_V + \theta_c)}{\mathcal{R}_0 (k_1 k_2 k_3 k_5 k_6) N_H N_H} \left[k_3 k_6 (\eta_H k_2 + \sigma_H) (\mu_1 M_H N_H - k_1 E_H N_H) \\ &+ \mathcal{R}_0 k_1 k_2 k_3 k_6 N_H E_H \eta_H + \mathcal{R}_0 k_1 k_2 k_3 k_6 N_H I_H + k_1 k_3 k_6 N_H E_H \sigma_H + k_1 k_3 k_6 N_H M_H \mu_2 \\ &- k_1 k_3 k_6 k_2 N_H I_H + N_H (\eta_H \mu_1 k_2 + \mu_2 k_1 + \sigma_H \mu_1) k_6 (\pi_1 - k_3 M_H) \right] \end{aligned}$$

By considering only the exposed and infectious migrated populations, we get the following algebraic manipulation:

$$\begin{split} \dot{\mathcal{F}} &= k_6 (\eta_V E_V + I_V) (\mathcal{R}_0 - 1) \\ &+ \frac{C_2 S_V (\eta_V k_6 + \sigma_V + \theta_c)}{\mathcal{R}_0 (k_1 k_2 k_3 k_5 k_6) N_H N_H} \bigg[\mathcal{R}_0 k_1 k_2 k_3 k_6 N_H E_H \eta_H + \mathcal{R}_0 k_1 k_2 k_3 k_6 N_H I_H \\ &+ k_2 k_3 k_6 M_H N_H \eta_H \mu_1 - k_1 k_2 k_3 k_6 E_H N_H \eta_H + k_3 k_6 M_H N_H \sigma_H \mu_1 - k_1 k_3 k_6 E_H N_H \\ &+ k_1 k_3 k_6 E_H N_H \sigma_H + k_1 k_3 k_6 N_H M_H \mu_2 - k_1 k_2 k_3 k_6 N_H I_H - k_2 k_3 k_6 M_H N_H \mu_1 \eta_H \\ &- k_1 k_3 k_6 M_H N_H \mu_2 - k_3 k_6 M_H N_H \sigma_H \mu_1 \bigg] \\ &= k_6 (\eta_V E_V + I_V) [\mathcal{R}_0 - 1] + \frac{C_2 S_V (\eta_V k_6 + \sigma_V + \theta_c)}{\mathcal{R}_0 k_5 N_H} \eta_H E_H \bigg[\mathcal{R}_0 - 1 \bigg] \\ &+ \frac{C_2 S_V (\eta_V k_6 + \sigma_V + \theta_c)}{\mathcal{R}_0 k_5 N_H} I_H \bigg[\mathcal{R}_0 - 1 \bigg] \end{split}$$

Thus, $\dot{\mathcal{F}} < 0$ if $\mathcal{R}_0 < 1$ with $\dot{\mathcal{F}} = 0$ if and only if $E_H = I_H = M_H = T_H = E_V = I_V = 0$. It follows, from the Lasalle Invariance Principle [18], that $E_H \to 0, I_H \to 0, M_H \to 0, T_H \to 0, E_V \to 0$ and $I_V \to 0$ as $t \to \infty$ (i.e., the disease dies out). Thus, $(E_H, I_H, M_H, T_H, E_V, I_V) = (0, 0, 0, 0, 0, 0)$ as $t \to \infty$.

Now, for any $\epsilon > 0$ sufficiently small, there exists a $t_1 > 0$ such that if $t > t_1$, then

$$E_H < \epsilon, \quad I_H < \epsilon, \quad M_H < \epsilon, \quad T_H < \epsilon, \quad E_V < \epsilon, \quad I_V < \epsilon$$
(11)

Now it follows from the equations for S_H and A_V in (8) that for $t > t_1$ (and noting (11))

$$\frac{dS_H}{dt} = \pi_H - \lambda_H S_H - \mu_H S_H \ge \pi_H - \frac{C_{HV}(\eta_V + 1)\epsilon}{N_H} - \mu_H S_H$$
$$\frac{dA_V}{dt} = \pi_V - (\gamma_m + \mu_V + C_a)A_V$$

Thus, by a standard comparison theorem [30]

$$\liminf_{t \to \infty} S_H(t) \ge \frac{\pi_H - C_{HV}(\eta_V + 1)\epsilon}{\mu_H}$$
(12)

$$\liminf_{t \to \infty} A_V(t) \ge \frac{\pi_V}{\mu_V} \tag{13}$$

Since $\epsilon > 0$ is arbitrarily small, letting $\epsilon \to 0$ in (12) gives

$$\liminf_{t \to \infty} S_H(t) \ge \frac{\pi_H}{\mu_H} \tag{14}$$

Similarly, it can be shown that

$$\limsup_{t \to \infty} S_H(t) \le \frac{\pi_H}{\mu_H} \tag{15}$$

and

$$\limsup_{t \to \infty} A_V(t) \le \frac{\pi_V}{\mu_V} \tag{16}$$

Hence, it follows from (13), (14), (15) and (16) that

$$\lim_{t \to \infty} S_H(t) = \frac{\pi_H}{\mu_H} \text{ and } \lim_{t \to \infty} A_V(t) = \frac{\pi_V}{\mu_V}$$

Thus,

$$\lim_{t \to \infty} (S_H(t), E_H(t), I_H(t), M_H(t), T_H(t), R_H(t), A_V(t), S_V(t), E_V(t), I_V(t))$$
(17)

$$= \left(\frac{\pi_H}{\mu_H}, 0, 0, 0, 0, 0, 0, \frac{\pi_V}{(\mu_V + C_a + \gamma_m)}, \frac{\gamma_m \pi_V}{(\gamma_m + \mu_V + C_a)(\mu_V + C_m)}, 0, 0\right) = E_0$$

The epidemiological implication of theorem (1) is that the classical epidemiological requirement at $\mathcal{R}_0 < 1$ is necessary and sufficient for the elimination of dengue virus in the community.

2.3 Existence of Endemic Equilibria of the Model (8)

To find the conditions for the existence of the endemic equilibria of the model (8) (that is, equilibria of the model (8) for which the disease is endemic in the population), denoted by

$$E_1 = (S_H^{**}, E_H^{**}, I_H^{**}, M_H^{**}, T_H^{**}, R_H^{**}, A_V^{**}, S_V^{**}, E_V^{**}, I_V^{**})$$

the equations in the model (8) are solved in terms of the associated forces of infection at steady-state, namely

$$\lambda_{H}^{**} = \frac{C_{HV}(\eta_{V}E_{V}^{**} + I_{V}^{**})}{N_{H}^{**}} \text{ and } \lambda_{V}^{**} = \frac{C_{HV}(\eta_{H}E_{H}^{**} + I_{H}^{**})}{N_{H}^{**}}$$
(18)

Setting the right-hand sides of the equations in (8) to zero gives (in terms of $S_H^{**} > 0$) the following expressions for the state variables of the model:

$$S_{H}^{**} = \frac{\pi_{H}}{\lambda_{H}^{**} + \mu_{H}},$$

$$M_{H}^{**} = \frac{\pi_{I}}{k_{3}},$$

$$E_{H}^{**} = \frac{\lambda_{H}^{**}S_{H}^{**}k_{3} + \mu_{I}\pi_{1}}{k_{1}k_{3}},$$

$$I_{H}^{**} = \frac{\sigma_{H}\lambda_{H}^{**}S_{H}^{**}k_{3} + \sigma_{H}\mu_{I}\pi_{1} + \mu_{2}\pi_{1}k_{1}}{k_{1}k_{2}k_{3}},$$

$$T_{H}^{**} = \frac{\sigma_{H}\lambda_{H}^{**}\tau_{H}S_{H}^{**}k_{3} + \tau_{H}\sigma_{H}\mu_{I}\pi_{1} + k_{1}\pi_{I}\mu_{2}\tau_{H}}{k_{1}k_{2}k_{3}k_{4}},$$

$$R_{H}^{**} = \frac{\gamma_{I}\sigma_{H}\lambda_{H}^{**}\tau_{H}S_{H}^{**}k_{3} + \tau_{H}\gamma_{I}\sigma_{H}\mu_{I}\pi_{1} + \mu_{2}\pi_{1}k_{1}\tau_{H}\gamma_{1}}{k_{1}k_{2}k_{3}k_{4}\mu_{H}},$$

$$A_{V}^{**} = \frac{\pi_{V}}{\gamma_{m} + \mu_{V} + C_{a}},$$

$$S_{V}^{**} = \frac{\gamma_{m}A_{V}^{**}}{\lambda_{V}^{**} + \mu_{V} + C_{m}},$$

$$E_{V}^{**} = \frac{\lambda_{V}^{**}S_{V}^{**}}{k_{5}},$$

$$I_{V}^{**} = \frac{(\sigma_{V} + \theta_{c})\lambda_{V}^{**}S_{V}^{**}}{k_{5}k_{6}},$$
(19)

Thus using (19), from λ_V^{**} in (18) we get:

$$\lambda_V^{**} = \frac{C_{HV}(\lambda_H^{**}S_H^{**}k_3 + \mu_1\pi_1)(\eta_H k_2 + \sigma_H) + C_{HV}\mu_2\pi_1k_1}{k_1k_2k_3N_H^{**}}$$
(20)

Now, substituting the value of λ_V^{**} of (20) in λ_H^{**} of (18), we get:

$$\lambda_{H}^{**} = \frac{G}{k_{5}k_{6} \left[(\lambda_{H}^{**}S_{H}^{**}k_{3} + \mu_{1}\pi_{1})B + k_{1}k_{2}k_{3}k_{4}\mu_{H}S_{H}^{**} + D \right]^{2}}$$
(21)

where,

$$B = k_2 k_4 \mu_H + k_4 \mu_H + \mu_H \sigma_H \tau_H + \sigma_H \tau_H \gamma_1,$$

$$D = \pi_1 k_1 k_4 \mu_H (k_2 k_3 + \mu_2) + \tau_H k_1 k_2 \mu_2 \pi_1 (\mu_H + \gamma_1),$$

$$G = \left[C_{HV} \lambda_H^{**} S_H^{**} k_2 k_3 \eta_H + C_{HV} \mu_1 \pi_1 \sigma_H + C_{HV} \lambda_H^{**} S_H^{**} k_3 \sigma_H + C_{HV} \mu_1 \pi_1 \eta_H k_2 + C_{HV} \mu_2 \pi_1 k_1 \right]$$

$$\left[C_{HV} S_V^{**} \eta_V k_6 + C_{HV} S_V^{**} (\sigma_V + \theta_c) \right].$$

It follows that the endemic equilibria of the model (8) satisfy the following polynomial (using (18), (20) and (21), then simplifying, we get:)

$$\begin{split} (\lambda_{H}^{**})^{3}(S_{H}^{**}k_{3})^{2}k_{5}k_{6} \bigg[((k_{2}+1)k_{4}\mu_{H})^{2} + (\sigma_{H}\tau_{H}(\mu_{H}+\gamma_{1}))^{2} \\ &+ 2k_{4}\mu_{H}\tau_{H}\sigma_{H}(k_{2}\mu_{H}+k_{2}) + \mu_{H} + \gamma_{1} \bigg] \\ &+ (\lambda_{H}^{**})^{2} \bigg[2k_{5}k_{6}k_{3}S_{H}^{**}\mu_{1}\pi_{1}bB^{2} + 2(S_{H}^{**}k_{3})^{2}Bk_{1}k_{2}k_{4}k_{5}k_{6}\mu_{H} + 2DBS_{H}^{**}k_{3}k_{5}k_{6} \bigg] \\ &+ \lambda_{H}^{**} \bigg[k_{5}k_{6}\mu_{1}\pi_{1}B^{2} + k_{5}k_{6}(k_{1}k_{2}k_{3})^{2} + (k_{4}\mu_{H}S_{H}^{**})^{2} + D^{2} \\ &+ 2k_{1}k_{2}k_{3}k_{4}k_{5}k_{6}\mu_{1}\pi_{1}B\mu_{H}S_{H}^{**} + 2DB\mu_{1}\pi_{1}k_{5}k_{6} \\ &- (C_{HV})^{2}S_{H}^{**}S_{V}^{**}k_{3}(k_{2}\eta_{H}+\sigma_{H})(\eta_{V}k_{6}+\sigma_{V}+\theta_{c}) \bigg] \\ &- (C_{HV})^{2}S_{V}^{**}(k_{2}\eta_{H}+\sigma_{H})(\eta_{V}k_{6}+\sigma_{V}+\theta_{c})(\mu_{1}\pi_{1}+\mu_{2}\pi_{1}k_{1}) = 0 \end{split}$$

i.e.,

$$X(\lambda_H^{**})^3 + Y(\lambda_H^{**})^2 + Z\lambda_H^{**} - P = 0$$
(22)

where,

$$\begin{split} X &= (S_{H}^{**}k_{3})^{2}k_{5}k_{6} \bigg[((k_{2}+1)k_{4}\mu_{H})^{2} + (\sigma_{H}\tau_{H}(\mu_{H}+\gamma_{1}))^{2} \\ &+ 2k_{4}\mu_{H}\tau_{H}\sigma_{H}(k_{2}\mu_{H}+k_{2}) + \mu_{H} + \gamma_{1} \bigg], \\ Y &= 2k_{5}k_{6}k_{3}S_{H}^{**}\mu_{1}\pi_{1}bB^{2} + 2(S_{H}^{**}k_{3})^{2}Bk_{1}k_{2}k_{4}k_{5}k_{6}\mu_{H} + 2DBS_{H}^{**}k_{3}k_{5}k_{6}, \\ Z &= k_{5}k_{6}\mu_{1}\pi_{1}B^{2} + k_{5}k_{6}(k_{1}k_{2}k_{3})^{2} + (k_{4}\mu_{H}S_{H}^{**})^{2} + D^{2} + 2k_{1}k_{2}k_{3}k_{4}k_{5}k_{6}\mu_{1}\pi_{1}B\mu_{H}S_{H}^{**} \\ &+ 2DB\mu_{1}\pi_{1}k_{5}k_{6} - (C_{HV})^{2}S_{H}^{**}S_{V}^{**}k_{3}(k_{2}\eta_{H}+\sigma_{H})(\eta_{V}k_{6}+\sigma_{V}+\theta_{c}), \\ P &= (C_{HV})^{2}S_{V}^{**}(k_{2}\eta_{H}+\sigma_{H})(\eta_{V}k_{6}+\sigma_{V}+\theta_{c})(\mu_{1}\pi_{1}+\mu_{2}\pi_{1}k_{1}), \end{split}$$

Now according to Routh-Hurwitz criterion if we look at the equation (22), then there is only one sign change, thus there is only one real root exists for the equation. Therefore the system (8) has a unique EEP, of the form

$$E_1 = (S_H^{**}, E_H^{**}, I_H^{**}, M_H^{**}, T_H^{**}, R_H^{**}, A_V^{**}, S_V^{**}, E_V^{**}, I_V^{**}),$$

We claim the following:

Lemma 3. The model (8) has one positive (endemic) equilibrium whenever $\mathcal{R}_0 > 1$, and no positive equilibrium otherwise.

Hence, the above mathematical analysis show that the basic dengue model (8) has a globally-asymptotically stable disease-free equilibrium whenever $\mathcal{R}_0 < 1$, and a unique endemic equilibrium if $\mathcal{R}_0 > 1$.

2.3.1 Local Stability of Endemic Equilibria

The local stability of EEP, E_1 , of the model (8) is consider for the special case where we use $N_H = N_H^{**}$, disease-induced mortality is $\text{zero}(\delta_H = \delta_V = 0)$ and the definition $S_H = N_H^{**} - E_H - I_H - M_H - T_H - R_H$ and $S_V = N_V^{**} - E_V - I_V$

in (8), gives the following reduced basic model:

$$\frac{dE_{H}}{dt} = \frac{C_{1}(\eta_{V}E_{V}+I_{V})}{N_{H}^{**}} \left[N_{H}^{**} - E_{H} - I_{H} - M_{H} - T_{H} - R_{H} \right] + \mu_{1}M_{H} - (\mu_{H} + \sigma_{H})E_{H},
\frac{dI_{H}}{dt} = \sigma_{H}E_{H} + \mu_{2}M_{H} - (\tau_{H} + \mu_{H})I_{H},
\frac{dM_{H}}{dt} = \pi_{1} - (\mu_{1} + \mu_{2} + \mu_{H})M_{H},
\frac{dT_{H}}{dt} = \tau_{H}I_{H} - (\mu_{H} + \gamma_{1})T_{H},$$
(23)
$$\frac{dR_{H}}{dt} = \gamma_{1}T_{H} - \mu_{H}R_{H},
\frac{dE_{V}}{dt} = \frac{C_{2}(\eta_{H}E_{H} + I_{H})}{N_{H}^{**}} \left[N_{V}^{**} - E_{V} - I_{V} \right] - (\sigma_{V} + \theta_{c} + \mu_{V} + C_{m})E_{V},
\frac{dI_{V}}{dt} = (\sigma_{V} + \theta_{c})E_{V} - (\mu_{V} + C_{m})I_{V}.$$

Now we can rewrite the model (23) as

$$\begin{aligned} \frac{dE_{H}}{dt} &= \frac{C_{1}(\eta_{V}E_{V}+I_{V})}{N_{H}^{**}} \Big[N_{H}^{**} - E_{H} - I_{H} - M_{H} - T_{H} - R_{H} \Big] + \mu_{1}M_{H} - k_{1}E_{H}, \\ \frac{dI_{H}}{dt} &= \sigma_{H}E_{H} + \mu_{2}M_{H} - k_{2}I_{H}, \\ \frac{dM_{H}}{dt} &= \pi_{1} - k_{3}M_{H}, \\ \frac{dT_{H}}{dt} &= \tau_{H}I_{H} - k_{4}T_{H}, \\ \frac{dR_{H}}{dt} &= \gamma_{1}T_{H} - \mu_{H}R_{H}, \\ \frac{dE_{V}}{dt} &= \frac{C_{2}(\eta_{H}E_{H} + I_{H})}{N_{H}^{**}} \Big[N_{V}^{**} - E_{V} - I_{V} \Big] - k_{5}E_{V}, \\ \frac{dI_{V}}{dt} &= (\sigma_{V} + \theta_{c})E_{V} - k_{6}I_{V}. \end{aligned}$$
(24)

$$k_1 = \mu_H + \sigma_H, \quad k_2 = \tau_H + \mu_H, \quad k_3 = \mu_1 + \mu_2 + \mu_H,$$

$$k_4 = \mu_H + \gamma_H, \quad k_5 = \sigma_V + \theta_c + \mu_V + C_m, \quad k_6 = \mu_V + C_M.$$

It is easy to show that the system (23) has a unique EEP, of the form $\overline{E}_1 = (E_H^{**}, I_H^{**}, M_H^{**}, T_H^{**}, R_H^{**}, E_v^{**}, I_v^{**})$, whenever $\mathcal{R}_{01} = \mathcal{R}_0|_{\delta_H = \delta_V = 0} > 1$. We claim the following theorem:

Theorem 2. The unique endemic equilibrium, \overline{E}_1 , of the reduced basic model (23) is LAS whenever $\mathcal{R}_{01} = \mathcal{R}_0|_{\delta_H = \delta_V = 0} > 1$.

Proof. The proof of theorem is based on using a Krasnoselskii sub-linearity trick (see [10, 12, 28] and also [19, 22]). Linearizing the system (23) around the endemic equilibrium, \bar{E}_1 , gives

$$\frac{dE_{H}}{dt} = a_{3}\eta_{V}E_{V} + a_{3}I_{V} + (-a_{1} - k_{1})E_{H} - a_{1}(I_{H} + M_{H} + T_{H} + R_{H}) + \mu_{1}M_{H},$$

$$\frac{dI_{H}}{dt} = \sigma_{H}E_{H} + \mu_{2}M_{H} - k_{2}I_{H},$$

$$\frac{dM_{H}}{dt} = \pi_{1} - k_{3}M_{H},$$

$$\frac{dT_{H}}{dt} = \tau_{H}I_{H} - k_{4}T_{H},$$

$$\frac{dR_{H}}{dt} = \gamma_{1}T_{H} - \mu_{H}R_{H},$$

$$\frac{dE_{V}}{dt} = a_{4}\eta_{H}E_{H} + a_{4}I_{H} + (-a_{2} - k_{5})E_{V} - a_{2}I_{V},$$

$$\frac{dI_{V}}{dt} = (\sigma_{V} + \theta_{c})E_{V} - k_{6}I_{V}.$$
(25)

where,

$$a_{1} = \frac{C_{1}(\eta_{V}E_{V} + I_{V})}{N_{H}^{**}}, a_{2} = \frac{C_{2}(\eta_{H}E_{H} + I_{H})}{N_{H}^{**}},$$
$$a_{3} = \frac{C_{1}S_{H}}{N_{H}^{**}}, a_{4} = \frac{C_{2}S_{V}}{N_{H}^{**}}.$$

It follows that the Jacobian of the system (25), evaluated at \bar{E}_1 , is given by

$$J(\bar{E_1}) = \begin{pmatrix} -a_1 - k_1 & -a_1 & \mu_1 - a_1 & -a_1 & -a_1 & a_3\eta_V & a_3 \\ \sigma_H & -k_2 & \mu_2 & 0 & 0 & 0 & 0 \\ 0 & 0 & -k_3 & 0 & 0 & 0 & 0 \\ 0 & \tau_H & 0 & -k_4 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma_1 & -\mu_H & 0 & 0 \\ a_4\eta_H & a_4 & 0 & 0 & 0 & -a_2 - k_5 & -a_2 \\ 0 & 0 & 0 & 0 & 0 & \sigma_V + \theta_c & -k_6 \end{pmatrix}.$$

Assume that the system (25) has solution of the form

$$\bar{\mathbf{Z}}(t) = \bar{\mathbf{Z}}_0 e^{\theta t},\tag{26}$$

with $\bar{\mathbf{Z}}_0 = (Z_1, Z_2, Z_3, Z_4, Z_5, Z_6, Z_7), \quad \theta, Z_i \in \mathbb{C}(i = 1, 2, ...7).$ Substituting a

solution of the form (26) into the system (25) gives

$$\begin{aligned} \theta Z_1 &= a_3 \eta_V Z_6 + a_3 Z_7 + (-a_1 - k_1) Z_1 - a_1 (Z_2 + Z_3 + Z_4 + Z_5) + \mu_1 Z_4, \\ \theta Z_2 &= \sigma_H Z_1 + \mu_2 Z_3 - k_2 Z_2, \\ \theta Z_3 &= \pi_1 - k_3 Z_3, \\ \theta Z_4 &= \tau_H Z_2 - k_4 Z_4, \\ \theta Z_5 &= \gamma_1 Z_4 - \mu_H Z_5, \\ \theta Z_6 &= a_4 \eta_H Z_1 + a_4 Z_2 + (-a_2 - k_5) Z_6 - a_2 Z_7, \\ \theta Z_7 &= (\sigma_V + \theta_c) Z_6 - k_6 Z_7. \end{aligned}$$

$$(27)$$

System (27) is simplified as follows. Firstly, all the negative terms in the 2nd, 3rd, 4th, 5th and 7th equations of (27) are moved to the respective left-hand sides.

$$\begin{split} Z_{2} &= \frac{\sigma_{H} Z_{1}}{\theta + k_{2}} + \frac{\mu_{2} \pi_{1}}{(\theta + k_{3})(\theta + k_{2})}, \\ Z_{3} &= \frac{\pi_{1}}{\theta + k_{3}}, \\ Z_{4} &= \frac{\sigma_{H} Z_{1} \tau_{H}}{(\theta + k_{2})(\theta + k_{4})} + \frac{\mu_{2} \tau_{H} \pi_{1}}{(\theta + k_{3})(\theta + k_{2})(\theta + k_{4})}, \\ Z_{5} &= \frac{\sigma_{H} Z_{1} \tau_{H} \gamma_{1}}{(\theta + k_{2})(\theta + k_{4})(\theta + \mu_{H})} + \frac{\mu_{2} \tau_{H} \pi_{1} \gamma_{1}}{(\theta + k_{3})(\theta + k_{2})(\theta + k_{4})(\theta + \mu_{H})}, \\ Z_{7} &= \frac{\sigma_{V} + \theta_{c}}{\theta + k_{6}} Z_{6}. \end{split}$$

Secondly, the (resulting) equations are then re-written in terms of Z_1 and Z_6 and substituted into the remaining equations of (27), and all its negative terms are moved to the right-hand side. Doing all these lead to the following equations:

$$\begin{bmatrix} 1 + \frac{\theta + a_1}{k_1} + \frac{a_1 \sigma_H}{k_1 (\theta + k_2)} + \frac{\sigma_H a_1 \tau_H}{(\theta + k_2) (\theta + k_4) k_1} + \frac{\sigma_H a_1 \tau_H \gamma_1}{(\theta + k_2) (\theta + k_4) (\theta + \mu_H) k_1} \end{bmatrix} Z_1 + \Gamma = \frac{a_3 \eta_V Z_6 + a_3 Z_7 + \mu_1 Z_3}{k_1},$$

$$\begin{split} & \left[1 + \frac{\theta}{k_2}\right] Z_2 = \frac{\sigma_H}{k_2} Z_1 + \frac{\mu_1}{k_2} Z_3, \\ & \left[1 + \frac{\theta}{k_3}\right] Z_3 = \frac{\pi_1}{k_3}, \\ & \left[1 + \frac{\theta}{k_4}\right] Z_4 = \frac{\tau_H}{k_4} Z_2, \\ & \left[1 + \frac{\theta}{\mu_H}\right] Z_5 = \frac{\gamma_1}{\mu_H} Z_4, \\ & \left[1 + \frac{\theta + a_2}{k_5} + \frac{a_2(\sigma_v + \theta_c)}{k_5(\theta + k_6)}\right] Z_6 = \frac{a_4 \eta_H Z_1 + a_4 Z_2}{k_5}, \\ & \left[1 + \frac{\theta}{k_6}\right] Z_7 = \frac{(\sigma_v + \theta_c)}{k_6} Z_6. \end{split}$$

Now we can rewrite the equations as:

$$Z_{1}[1 + F_{1}(\theta)] + \Gamma = (M\bar{Z})_{1},$$

$$Z_{2}[1 + F_{2}(\theta)] = (M\bar{Z})_{2},$$

$$Z_{3}[1 + F_{3}(\theta)] = (M\bar{Z})_{3},$$

$$Z_{4}[1 + F_{4}(\theta)] = (M\bar{Z})_{4},$$

$$Z_{5}[1 + F_{5}(\theta)] = (M\bar{Z})_{5},$$

$$Z_{6}[1 + F_{6}(\theta)] = (M\bar{Z})_{6},$$

$$Z_{7}[1 + F_{7}(\theta)] = (M\bar{Z})_{7}.$$
(28)

where,

$$\begin{split} F_{1} &= \left[\frac{\theta + a_{1}}{k_{1}} + \frac{a_{1}\sigma_{H}}{k_{1}(\theta + k_{2})} + \frac{\sigma_{H}a_{1}\tau_{H}}{(\theta + k_{2})(\theta + k_{4})k_{1}} + \frac{\sigma_{H}a_{1}\tau_{H}\gamma_{1}}{(\theta + k_{2})(\theta + k_{4})(\theta + \mu_{H})k_{1}} \right], \\ F_{2} &= \frac{\theta}{k_{2}}, \\ F_{3} &= \frac{\theta}{k_{2}}, \\ F_{3} &= \frac{\theta}{k_{3}}, \\ F_{4} &= \frac{\theta}{k_{4}}, \\ F_{5} &= \frac{\theta}{k_{4}}, \\ F_{5} &= \frac{\theta}{\mu_{H}}, \\ F_{6} &= \frac{\theta + a_{2}}{k_{5}} + \frac{a_{2}(\sigma_{v} + \theta_{c})}{k_{5}(\theta + k_{6})}, \\ F_{7} &= \frac{\theta}{k_{6}}. \end{split}$$

with,

The notation $M(\bar{Z})_i$ (with i = 1, 2, 3, 4, 5, 6, 7) denotes the *i*th coordinate of the vector $M(\bar{Z})$. It should be noted that the matrix M has non-negative entries, and the equilibrium \bar{E}_1 satisfies $\bar{E}_1 = M\bar{E}_1$.

Furthermore, since the coordinates of \bar{E}_1 are all positive, it follows then that if $\bar{\mathbf{Z}}$ is a solution of equation (28), then it is possible to find a minimal positive real number, s, such that [19, 22]

$$\bar{\mathbf{Z}} \mid \le s\bar{E}_1, \tag{29}$$

where,

$$|\bar{\mathbf{Z}}| = (|Z_1|, |Z_2|, |Z_3|, |Z_4|, |Z_5|, |Z_6|, |Z_7|)$$

with the lexicographic order and $|\cdot|$ is a norm in \mathbb{C} .

The goal is to show that $Re\theta < 0$. Assume the contrary (i.e., $Re\theta \ge 0$).

We consider two cases: $\theta = 0$ and $\theta \neq 0$.

Assume the first case $\theta = 0$. Then, equation (27) is a homogeneous linear system in the variables Z_i (i = 1, 2, 3, 4, 5, 6, 7). The determinant of this system corresponds to that of the Jacobian of system (25) evaluated at \bar{E}_1 , which is given by

$$\begin{split} & \Delta = -k_2 k_3 k_4 k_6 a_1 a_2 \mu_H - k_2 k_3 k_4 k_5 k_6 a_1 \mu_H - k_2 k_3 k_4 a_1 a_2 \sigma_V \\ & - k_2 k_3 k_4 a_1 a_2 \mu_H \theta_c - k_1 k_2 k_3 k_4 k_6 a_2 \mu_H - k_1 k_2 k_3 k_4 k_5 k_6 \mu_H \\ & - k_1 k_2 k_3 k_4 a_2 \sigma_V \mu_H - k_1 k_2 k_3 k_4 a_2 \sigma_H \sigma_V \mu_H - k_3 k_4 a_1 a_2 \sigma_H \sigma_C \mu_H \\ & - k_3 k_6 a_1 a_2 \tau_H \sigma_H \mu_H - k_3 k_5 k_6 a_1 \tau_H \sigma_H \mu_H - k_3 a_1 a_2 \tau_H \sigma_H \mu_H \sigma_V \\ & - k_3 a_1 a_2 \tau_H \sigma_H \mu_H \theta_c - k_3 k_6 a_1 a_2 \tau_H \sigma_H \gamma_1 - k_3 K_5 k_6 a_1 \tau_H \sigma_H \gamma_1 \\ & - k_3 a_1 a_2 \tau_H \sigma_H \gamma_1 \sigma_V - k_3 k_1 a_2 \tau_H \sigma_H \gamma_1 \theta_c + k_3 k_4 a_3 a_4 \sigma_U \mu_H \eta_H \\ & + k_3 k_4 a_3 a_4 \sigma_U \mu_H \eta_H + k_2 k_3 k_4 k_6 a_3 a_4 \sigma_H \mu_H \eta_V + k_2 k_3 k_4 a_3 a_4 \sigma_U \mu_H \eta_H \\ & + k_2 k_3 k_4 a_3 a_4 \theta_C \mu_H \eta_H + k_2 k_3 k_4 k_6 a_3 a_4 \mu_H \eta_H \gamma_V \\ & = - k_2 k_3 k_4 k_6 a_1 a_2 \mu_H - k_2 k_3 k_4 k_6 a_3 a_4 \mu_H \eta_H \gamma_V \\ & = - k_2 k_3 k_4 k_6 a_1 a_2 \mu_H - k_2 k_3 k_4 k_6 a_2 \mu_H - k_1 k_2 k_3 k_4 a_1 a_2 \sigma_V \\ & - k_1 k_2 k_3 k_4 a_2 \sigma_V \mu_H - k_1 k_2 k_3 k_4 a_2 \sigma_H \sigma_C \mu_H \\ & - k_1 k_2 k_3 k_4 a_2 \sigma_V \mu_H - k_1 k_2 k_3 k_4 a_2 \sigma_H \sigma_C \mu_H \\ & - k_3 k_6 a_1 a_2 \tau_H \sigma_H \mu_H - k_3 k_4 a_1 a_2 \sigma_H \sigma_V \mu_H - k_3 a_1 a_2 \tau_H \sigma_H \mu_H \sigma_V \\ & - k_3 a_1 a_2 \tau_H \sigma_H \mu_H \theta_C - k_3 k_6 a_1 a_2 \tau_H \sigma_H \gamma_1 - k_3 k_5 k_6 a_1 \tau_H \sigma_H \gamma_1 \\ & - k_3 k_4 k_3 a_4 \mu_H \left[\sigma_H \sigma_V + \sigma_H \theta_C + \sigma_H \eta_V + k_2 \sigma_v \eta_H + k_2 \theta_c \eta_H + k_2 k_6 \eta_H \eta_V \right]. \\ & = - k_2 k_3 k_4 a_1 a_2 \mu_H \theta_C - k_1 k_2 k_3 k_4 k_6 a_2 \mu_H - k_1 k_2 k_3 k_4 a_2 \sigma_V \mu_H \\ & - k_1 k_2 k_3 k_4 a_1 a_2 \mu_H \theta_C - k_3 k_4 k_6 a_2 a_1 \mu_H \sigma_V \\ & - k_3 k_4 a_1 a_2 \sigma_H \sigma_U \mu_H - k_3 a_4 a_2 \sigma_H \theta_C \mu_H - k_3 k_4 a_1 a_2 \sigma_V \mu_H \\ & - k_1 k_2 k_3 k_4 a_1 a_2 \mu_H \theta_C - k_1 k_2 k_3 k_4 k_5 k_6 a_1 \sigma_H \mu_H \\ & - k_3 k_4 a_1 a_2 \sigma_H \sigma_U \mu_H - k_3 k_4 a_1 a_2 \sigma_H \theta_C \mu_H \\ & - k_3 k_4 a_1 a_2 \sigma_H \sigma_U \mu_H - k_3 k_4 a_1 a_2 \sigma_H \theta_C \mu_H \\ & - k_3 k_6 a_1 \tau_H \sigma_H \eta_H - k_3 a_1 a_2 \tau_H \sigma_H \eta_H \sigma_C \\ & - k_3 k_6 a_1 a_2 \tau_H \sigma_H \gamma_1 - k_3 K_5 k_6 a_1 \tau_H \sigma_H \gamma_1 \\ & - k_3 k_6 a_1 a_2 \tau_H \sigma_H \gamma_1 \\ & - k_3 k_6 a_1 a_2 \tau_H \sigma_H \gamma_1 \\ & - k_3 k_6 a_1 a_2 \tau_H \sigma_H \gamma_1 \\ & - k_3 k_6 a_1$$

Since the model parameters are non-negative, and by algebraic manipulation, we finally get:

$$\Rightarrow \Delta = -k_{2}k_{3}k_{4}k_{6}a_{1}a_{2}\mu_{H} - k_{2}k_{3}k_{4}k_{5}k_{6}a_{1}\mu_{H} - k_{2}k_{3}k_{4}a_{1}a_{2}\sigma_{V} - k_{2}k_{3}k_{4}a_{1}a_{2}\mu_{H}\theta_{c} - k_{1}k_{2}k_{3}k_{4}k_{6}a_{2}\mu_{H} - k_{1}k_{2}k_{3}k_{4}a_{2}\sigma_{V}\mu_{H} - k_{1}k_{2}k_{3}k_{4}a_{2}\mu_{H}\theta_{c} - k_{3}k_{4}k_{6}a_{2}a_{1}\mu_{H}\sigma_{H} - k_{3}k_{4}k_{5}k_{6}a_{1}\sigma_{H}\mu_{H} - k_{3}k_{4}a_{1}a_{2}\sigma_{H}\sigma_{V}\mu_{H} - k_{3}k_{4}a_{1}a_{2}\sigma_{H}\theta_{c}\mu_{H} - k_{3}k_{6}a_{1}a_{2}\tau_{H}\sigma_{H}\mu_{H} - k_{3}k_{5}k_{6}a_{1}\tau_{H}\sigma_{H}\mu_{H} - k_{3}a_{1}a_{2}\tau_{H}\sigma_{H}\mu_{H}\sigma_{V} - k_{3}a_{1}a_{2}\tau_{H}\sigma_{H}\mu_{H}\theta_{c} - k_{3}k_{6}a_{1}a_{2}\tau_{H}\sigma_{H}\gamma_{1} - k_{3}K_{5}k_{6}a_{1}\tau_{H}\sigma_{H}\gamma_{1} - k_{3}a_{1}a_{2}\tau_{H}\sigma_{H}\gamma_{1}\sigma_{V} - k_{3}ka_{1}a_{2}\tau_{H}\sigma_{H}\gamma_{1}\theta_{c} - k_{1}k_{2}k_{3}k_{4}k_{5}k_{6}\left[1 - (\mathcal{R}_{0})^{2}\right] \neq 0$$

$$(30)$$

Therefore the system (25) has a trivial solution $\mathbf{Z} = 0$ (which corresponds to the DFE, E_0 .)

Now we consider the case $\theta \neq 0$. In this case, by assumption, $Re \ \theta > 0$. Thus, $|1 + F_i(\theta)| > 1$ for i = 1, 2, 3, 4, 5, 6, 7. Now, define $F(\theta) = \min |1 + F_i(\theta)|$, i = 1, 2, 3, 4, 5, 6, 7. Then, $F(\theta) > 1$. Therefore, $\frac{s}{F(\theta)} < s$. Since s is a minimal positive real number such that $|\bar{\mathbf{Z}}| \leq s\bar{E_1}$, then

$$| \bar{\mathbf{Z}} | > \frac{s}{F(\theta)} \bar{E}_1.$$
(31)

Taking norms on both sides of the third equation of (28), and using the fact that I is non-negative, gives

$$F(\theta) \mid Z_2 \mid \le I(\mid Z \mid)_2 \le s(I \mid \bar{E}_1 \mid)_2 \le sI_H^{**}.$$
(32)

Then, it follows from the above inequality that $|Z_2| \leq \frac{s}{F(\theta)} I_H^{**}$ which contradicts the equation (31). Hence, $Re \ \theta < 0$, which implies that \overline{E}_1 is LAS, if $\mathcal{R}_{01} > 1$.

The epidemiological implication of Theorem (8) is that the disease would persists in the community if the basic reproduction threshold $\mathcal{R}_{01} > 1$.

2.3.2 Global Stability of EEP of the Model (8)

The global stability of EEP of the model (8) is consider for the special case where the dengue induce mortality is very negligible (so that, $\delta_H = \delta_V = 0$) and at the endemic stage $S_H^{**} \ge S_H$, $E_H^{**} \ge E_H$, $S_V^{**} \ge S_V$, $E_V^{**} \ge E_V$. **Theorem 3.** The unique EEP, E_1 , of the model (8), is globally asymptotically stable in Ω whenever $\mathcal{R}_0 > 1$.

Proof. Consider the non-linear Lyapunov function

$$\mathcal{F} = \left[S_{H} - S_{H}^{**} - S^{**} ln \frac{S_{H}}{S_{H}^{**}}\right] + \left[E_{H} - E_{H}^{**} - E_{H}^{**} ln \frac{E_{H}}{E_{H}^{**}}\right] + a_{1} \left[I_{H} - I_{H}^{**} - I_{H}^{**} ln \frac{I_{H}}{I_{H}^{**}}\right] + a_{2} \left[M_{H} - M_{H}^{**} - M_{H}^{**} ln \frac{M_{H}}{M_{H}^{**}}\right] + a_{3} \left[T_{H} - T_{H}^{**} - T_{H}^{**} ln \frac{T_{H}}{T_{H}^{**}}\right] + a_{4} \left[R_{H} - R_{H}^{**} - R_{H}^{**} ln \frac{R_{H}}{R_{H}^{**}}\right] + a_{5} \left[A_{V} - A_{V}^{**} - A_{V}^{**} ln \frac{A_{V}}{A_{V}^{**}}\right] + \left[S_{V} - S_{V}^{**} - S_{V}^{**} ln \frac{S_{V}}{S_{V}^{**}}\right] + \left[E_{V} - E_{V}^{**} - E_{V}^{**} ln \frac{E_{V}}{E_{V}^{**}}\right] + a_{6} \left[I_{V} - I_{V}^{**} - I_{V}^{**} ln \frac{I_{V}}{I_{V}^{**}}\right]$$
(33)

with Lyapunov derivative of (33) given by

$$\dot{\mathcal{F}} = \left[1 - \frac{S_{H}^{**}}{S_{H}}\right] \dot{S}_{H} + \left[1 - \frac{E_{H}^{**}}{E_{H}}\right] \dot{E}_{H} + a_{1} \left[1 - \frac{I_{H}^{**}}{I_{H}}\right] \dot{I}_{H}
+ a_{2} \left[1 - \frac{M_{H}^{**}}{I_{M}}\right] \dot{M}_{H} + a_{3} \left[1 - \frac{T_{H}^{**}}{T_{H}}\right] \dot{T}_{H} + a_{4} \left[1 - \frac{R_{H}^{**}}{R_{H}}\right] \dot{R}_{H}
+ a_{5} \left[1 - \frac{A_{V}^{**}}{A_{V}}\right] \dot{A}_{V} + \left[1 - \frac{S_{V}^{**}}{S_{V}}\right] \dot{S}_{V} + \left[1 - \frac{E_{V}^{**}}{E_{V}}\right] \dot{E}_{V}
+ a_{6} \left[1 - \frac{I_{V}^{**}}{I_{V}}\right] \dot{I}_{V},$$
(34)

where,

$$a_1 = 0, \quad a_2 = \frac{\mu_1}{(\mu_1 + \mu_2 + \mu_H)}, \quad a_3 = 0,$$

$$a_4 = 0, \quad a_5 = \frac{\gamma_m}{(\gamma_m + \mu_V + C_a)}, \quad a_6 = 0,$$

Substituting these value in to the equation (34), we get

$$\begin{aligned} \dot{\mathcal{F}} &= \pi_{H} - \frac{C_{1}(\eta_{V}E_{V}+I_{V})}{N_{H}}S_{H} - \mu_{H}S_{H} - \pi_{H}\frac{S_{H}^{**}}{S_{H}} + \frac{C_{1}(\eta_{V}E_{V}^{**}+I_{V}^{**})}{N_{H}^{**}}S_{H}^{**} \\ &+ \mu_{H}S_{H}^{**} + \frac{C_{1}(\eta_{V}E_{V}+I_{V})}{N_{H}}S_{H} - (\mu_{H}+\sigma_{H})E_{H} - \frac{C_{1}(\eta_{V}E_{V}^{*}+I_{V}^{**})}{N_{H}^{**}}S_{H}^{**}\frac{E_{H}^{**}}{E_{H}} \\ &+ \mu_{1}M_{H} - \mu_{1}M_{H}^{**}\frac{E_{H}^{**}}{E_{H}} + (\mu_{H}+\sigma_{H})E_{H}^{**} + \frac{\mu_{1}}{(\mu_{1}+\mu_{2}+\mu_{H})} \left[\pi_{1} - \pi_{1}\frac{M_{H}^{**}}{M_{H}} \\ &+ (\mu_{1}+\mu_{2}+\mu_{H})M_{H}^{**} - (\mu_{1}+\mu_{2}+\mu_{H})M_{H}\right] + \frac{\gamma_{m}}{(\gamma_{m}+\mu_{V}+C_{a})} \left[\pi_{V} \\ &- \pi_{V}\frac{A_{V}^{**}}{A_{V}} - (\gamma_{m}+\mu_{V}+C_{a})A_{V} + (\gamma_{m}+\mu_{V}+C_{a})A_{V}^{**}\right] + \gamma_{m}A_{V} - (\mu_{V}+C_{m})S_{V} \\ &- \gamma_{m}A_{V}\frac{S_{V}^{**}}{S_{V}} + (\mu_{V}+C_{m})S_{V}^{**} - \frac{C_{2}(\eta_{H}E_{H}+I_{H})}{N_{H}}S_{V} + \frac{C_{2}(\eta_{H}E_{H}^{**}+I_{H}^{**})}{N_{H}^{**}}\frac{E_{V}^{**}}{E_{V}}S_{V}^{**} \\ &+ \left(\sigma_{V} + \theta_{c} + \mu_{V} + C_{m}\right)E_{V}^{**}, \end{aligned}$$

In the above equation (35), we will use the following relations:

$$\pi_{H} = \frac{C_{1}(\eta_{V}E_{V}^{**}+I_{V}^{**})}{N_{H}^{**}}S_{H}^{**} + \mu_{H}S_{H}^{**},$$

$$\mu_{1} = \frac{(\mu_{H}+\sigma_{H})E_{H}^{**} - \frac{C_{1}(\eta_{V}E_{V}^{**}+I_{V}^{**})}{N_{H}^{**}}S_{H}^{**}}{M_{H}^{**}},$$

$$M_{H}^{**} = \frac{\pi_{1}}{(\mu_{1}+\mu_{2}+\mu_{H})},$$

$$A_{V}^{**} = \frac{\gamma_{m}}{(\gamma_{m}+\mu_{V}+C_{a})},$$

$$(\mu_{V}+C_{m}) = \frac{\gamma_{m}A_{V}^{**} + \frac{C_{2}(\eta_{V}E_{V}^{**}+I_{V}^{**})}{N_{V}^{**}}S_{V}^{**}}{S_{V}^{**}},$$

$$C_{2}(\eta_{V}E_{V}^{**}+I_{V}^{**})}S_{V}^{**}$$

$$(\sigma_V + \theta_c + \mu_V + C_m) = \frac{\frac{\Theta_2(\eta_V D_V + \eta_V)}{N_V^*} S_V^{**}}{E_V^{**}}$$

Now from equation (35), we get

$$\begin{aligned} \dot{\mathcal{F}} &\leq \pi_{H} - \pi_{H} \frac{S_{H}^{**}}{S_{H}} - \frac{C_{1}(\eta_{V}E_{V}^{**} + I_{V}^{**})}{N_{H}^{**}} S_{H}^{**} \frac{E_{H}^{**}}{E_{H}} + \mu_{1}M_{H}^{**} - \mu_{1}M_{H}^{**} \frac{E_{H}^{**}}{E_{H}} - \mu_{1}M_{H} \\ &+ 2\mu_{1}M_{H}^{**} - \mu_{1}\frac{(M_{H}^{**})^{2}}{M_{H}} + \gamma_{m}A_{V}^{**} - \gamma_{m}A_{V} + \gamma_{m}A_{V}^{**} - \gamma_{m}\frac{(A_{V}^{**})^{2}}{A_{V}} \\ &+ (\gamma_{m}A_{V} - \gamma_{m}A_{V}\frac{S_{V}^{**}}{S_{V}}) - \frac{C_{2}(\eta_{H}E_{H} + I_{H})}{N_{H}}S_{V} + \frac{C_{2}(\eta_{H}E_{H}^{**} + I_{H}^{**})}{N_{H}^{**}}S_{V}^{**} \end{aligned}$$
(36)
$$&- (\mu_{V} + C_{m})S_{V} + \frac{C_{2}(\eta_{H}E_{H} + I_{H})}{N_{H}}S_{V} - (\sigma_{V} + \theta_{c} + \mu_{V} + C_{m})E_{V} \\ &+ (\mu_{V} + C_{m})S_{V}^{**} + (\sigma_{V} + \theta_{c} + \mu_{V} + C_{m})E_{V}^{**} - \frac{C_{2}(\eta_{H}E_{H}^{**} + I_{H}^{**})}{N_{H}^{**}}\frac{E_{V}^{**}}{E_{V}}S_{V}^{**}, \end{aligned}$$

Finally,

$$\dot{\mathcal{F}} \leq \pi_{H} \left(1 - \frac{S_{H}^{**}}{S_{H}}\right) - \frac{C_{1}(\eta_{V}E_{V}^{**} + I_{V}^{**})}{N_{H}^{**}} S_{H}^{**} \frac{E_{H}^{**}}{E_{H}} + \mu_{1}M_{H}^{**} \left(1 - \frac{E_{H}^{**}}{E_{H}}\right)
+ \mu_{1}M_{H}^{**} \left[2 - \frac{M_{H}^{**}}{M_{H}} - \frac{M_{H}}{M_{H}^{**}}\right] + \gamma_{m}A_{V}^{**} \left[2 - \frac{A_{V}}{A_{V}^{**}} - \frac{A_{V}^{**}}{A_{V}}\right]
+ \gamma_{m}A_{V} \left(1 - \frac{S_{V}^{**}}{S_{V}}\right) + \frac{C_{2}(\eta_{H}E_{H}^{**} + I_{H}^{**})}{N_{H}^{**}} S_{V}^{**} \left[1 - \frac{E_{V}^{**}}{E_{V}}\right],$$
(37)

Since the arithmetic mean exceeds the geometric mean, it follows then that

$$\begin{bmatrix} 2 - \frac{M_H^{**}}{M_H} - \frac{M_H}{M_H^{**}} \end{bmatrix} \le 0,$$
$$\begin{bmatrix} 2 - \frac{A_V}{A_V^{**}} - \frac{A_V^{**}}{A_V} \end{bmatrix} \le 0,$$

Also since, $S_H, E_H, M_H, A_V, S_V, E_V$, approaches $S_H^{**}, E_H^{**}, M_H^{**}, A_V^{**}, S_V^{**}, E_V^{**}$ asymptotically, or $S_H, E_H, M_H^{\dagger}, A_V, S_V, E_V$ becomes, and remains, less than $S_H^{**}, E_H^{**}, M_H^{**}, A_V^{**}, S_V^{**}, E_V^{**}$ in finite time, then from equation (37), we get

$$S_H - S_H^{**} \le 0, \quad E_H - E_H^{**} \le 0,$$

 $S_V - S_V^{**} \le 0, \quad E_V - E_V^{**} \le 0,$

i.e.,

$$S_H^{**} \ge S_H, \quad E_H^{**} \ge E_H,$$
$$S_V^{**} \ge S_V, \quad E_V^{**} \ge E_V,$$

Therefore from equation (37), we can finally say that

$$\dot{\mathcal{F}} \leq 0 \text{ for } \mathcal{R}_0 > 1$$

Thus, by the Laypunov function \mathcal{F} , and the LaSalle Invariance Principal [18], every solution to the equations in the model (8) approaches E_1 as $t \to \infty$ for $\mathcal{R}_0 > 1$.

At the end of the discussion, we can say that, the model (8) has a globallyasymptotically stable DFE whenever $\mathcal{R}_0 \leq 1$ and a unique EEP for $\mathcal{R}_0 > 1$. It is shown that the unique EEP of the model (8) is globally-asymptotically stable, for the special case whenever disease-induce mortality is very negligible and the threshold quantity that is the basic reproduction number greater than the unity ($\mathcal{R}_0 > 1$).

3 Numerical Simulations and Discussions

The model (8) is simulated, using the parameter values given in Table 2 and Table 3 (unless otherwise stated).

Figure 2 presents the simulations of the dengue transmission model (8), showing a contour plot of the reproduction threshold \mathcal{R}_0 which depicts that if the rate $C_2 = C_{VH}$ at which human acquire infection from infected mosquitoes (exposed or infectious) and the rate $C_1 = C_{HV}$ at which mosquitoes acquires infection from infected humans (exposed or infectious) decreases then the burden of the dengue disease decreases (in line with theorem 1). However if the rates C_1 and C_2 increases then the burden of the disease increases (in line with Theorem 3).

Figure 3 presents the simulations of the dengue transmission model (8), showing a contour plot of the reproduction threshold \mathcal{R}_0 which indicates that if the rate σ_V at which the vector individuals transfer from exposed class to infected class increases and at the same time if we have the effective precautionary measures the we would be able to control the disease spread and no endemic will occur (in line with Theorem 1), otherwise the disease burden will increases.

Figure 4 depicts that if the rate σ_H at which the exposed human population developed clinical symptoms of dengue disease move to infectious class decreases and the rate σ_V , at which the exposed vectors developed clinical symptoms of dengue disease move to infectious class decreases, then the total number of infected human population also decreases, otherwise burden of the disease increases.

Figure 5, 6, 7, 8 and 9 monitor the effect of the effective vector control rate C_m and C_a . If we do not have any necessary precautionary measures, then the total number of vector population increases rapidly (Figure 5) and persist in the community ultimately. If we take the precautionary measures in the aquatic stage (i.e., if the control rate C_a increases), the number of total infected vector I_V decreases rapidly as like figure (7). However if we take the necessary precautionary measures in the adult stage (i.e., if the control rate C_m increases), the total infected vector I_V also decreases, (Figure 6). Additionally if we take the precautionary step in the aquatic and adult both stage, then the total number of infected vector decreases drastically (Figure 8). To see the total changes in the vector population after some necessary precautionary measures, we have, from Figure 8, that the total vector population N_V decreases rapidly. Figure 5 and 6, present the comparative situation before and after the precautionary measures have taken.

Figure 10 and 11 present the effect of the migratory infected individuals. From Figure 11 we see that if the rate μ_1 at which the migratory population added to the exposed class E_H and the rate μ_2 at which the migratory individuals added to the infected class I_H varies, then the number of new infectious cases varies as well.

From Figure 10, we see that, small rate of increase in μ_1 and in μ_2 can increases the total number of infection and can create an endemic. From Figure 10, we can also see a comparative presentation of the model (8) simulation where, if $\mu_1 = \mu_2 = 0$, then the number of new infected population decreases. If $\mu_1 = \mu_2 \neq 0$, then the number of new infected individuals increases rapidly and converges to the endemic situation.

4 Conclusion

A deterministic model for dengue transmission dynamics presented and rigorously analysed. The disease-free equilibrium, E_0 , is shown to be locally asymptotically stable when the associated epidemic threshold known as the basic reproduction number, \mathcal{R}_0 , for the model is less than unity. This equilibrium (DFE) is shown to be globally-asymptotically stable whenever \mathcal{R}_0 is less than unity (Theorem 7). The model has a unique endemic equilibrium (EEP), E_1 , is shown to be locally asymptotically stable whenever \mathcal{R}_0 is greater than unity (Theorem 8). By considering special cases EEP is shown to be globally-asymptotically stable whenever $\mathcal{R}_0 > 1$ (Theorem 9). Numerical simulation reveals that if the rate at which human acquire infection from infected mosquitoes (C_{VH}) and the rate at which mosquitoes acquires infection from infected humans (C_{HV}) increases then the burden of the dengue disease increases. Numerical simulations indicates that if the rate of migratory exposed (μ_1) or migratory infected (μ_2) individuals increases then the rate of cumulative number of new cases increases. Numerical simulations suggest that proper treatment decreases the rate of infectiousness. Numerical simulation depicts that if we take the precautionary measures more seriously then it would be more effective then even giving the treatment to the infected individuals. Numerical simulations reveals that the spread of dengue virus can be controlled more effectively, if we take the precautionary measures at the aquatic and adult stages.

References

- C. Bowman, A.B. Gumel, P. van den Drissche, J. Wu and H. Zhu, Mathematical model for assessing control strategies against West Nile virus, *Bulletin of 682 Mathematical Biology*, 67, (2005), 1107-1133.
- [2] CDC. Locally acquired dengue-key West, Florida, 2009-2010, MMWR 2010, 59, (2010), 577–581.
- [3] Chen LH, Wilson ME Dengue and chikungunya infections in travelers, Curr Opin Infect Dis, 23, (2010),438444

- [4] C.J. Struchiner, P.M. Luz, C.T. Codeco, F.C. Coelho and E. Massad, Current research issues in mosquito-borne diseases modelling, *Contemporary Mathematics*, 410, (2006), 349-352.
- [5] F.A.B. Coutinho, M.N. Burattini, L.F. Lopez and E. Massad, Threshold conditions for a non-autonomous epidemic system describing the population dynamics of dengue, *Bulletin of Mathematical Biology*, 68, (2006), 2263-2282.
- [6] G. Chowell, P. Diaz-Duenas, J.C. Miller, A. Alcazar-Velazco, J.M. Hyman, P.W. Fenimore and C. Castillo Chavez Estimation of the reproduction number of dengue fever from spatial epidemic data, *Mathematical Biosciences*, 208(2), (2007), 571-589.
- [7] Garba, S. M., Gumel, A. B. and Abu Bakar, M.R., Backward bifurcations in dengue transmission dynamics, *Mathematical Biosciencees*. 201(1), (2008), 11-25.
- [8] Halstead SB, Dengue (Tropical Medicine: Science and Practice). River Edge, N.J: Imperial College Press. pp. 110. ISBN 1-84816-228-6, 2008.
- Holmes, P. and Guckenheimer, J., Nonlinear Oscillations, Dynamical Systems, and Bifurcations of Vector Fields, Springer-Verlag, New York Inc, 1990.
- [10] H. R. Thieme, Local Stability in Epidemic Models for Heterogenous Populations, *Lecture Notes in Biomathematics*, (V. Capasso, E. Grosso, S. L. Paveri-FontanaEds.,) Springer, **57**, (1985), 185-189.
- [11] H. W. Hethcote, The mathematics of infectious diseases, SIAM Rev., 42, (2000), 599-653.
- [12] H. W. Hethcote and H. R. Thieme, Stability of the endemic equilibrium in epidemic models with sub populations, *Math. Biosci.*,**75**, (1985), 205-227.
- [13] I. Kawaguchi, A. Sasaki and M. Boots, Why are dengue virus serotypes so distantly related? Enhancement and limiting serotype similarity between dengue virus srains, *Proceedings of the Royal Society of London B*, 270, (2003), 2241-2247.

- [14] Jelinek T., Dengue Fever in International Travelers, Clin Infect Dis, 31, (2000), 144147.
- [15] J.J. Tewa, J.L. Dimi and S. Bowang, Lyapunov functions for a dengue disease transmission model , *Chaos, Solitons and Fractal*, (2007), doi:10.1016/j.chaos.2007.01.069
- [16] Kautner I, Robinson M.J., Kuhnle U., Dengue virus infection: Epidemiology, pathogenesis, clinical presentation, diagonosis, and prevention, *The Journal of Pediatrics*, **131**, (1997), 516-524.
- [17] Koopman JS, Prevots DR, Mann MAV, Dantes HG, Aquino MLZ, et al., Determinants and Predictors of Dengue Infection in Mexico, Am. J. Epidemiol., 133, (1991), 11681178.
- [18] LaSalle, J.P., The stability of dynamical system, Regional Conference Series in Applied Mathematics, SIAM. Philadelphia, (1976).
- [19] L. Esteva, A. Gumel, and C. Vargas, Qualitative study of transmission dynamics of drug-resistant malaria, *Math. Comput. Modelling*, **50**, (2009), 611-630.
- [20] L. Esteva and C. Vargas, Analysis of a dengue disease transmission model, Mathematical Biosciences, 150, (1998), 131-151.
- [21] L. Esteva and C. Vargas, A model for dengue disease with variable human population, *Journal of Mathematical Biology*, 38, (1999), 220-240.
- [22] L. Esteva and C. Vargas, Influence of vertical and mechanical transmission on the dynamics of dengue disease, *Math. Biosci.*, 167, (2000), 51-64.
- [23] L. Esteva and C. Vargas, Coexistence of dierent serotypes of dengue virus, Journal of Mathematical Biology, 46, (2003), 31-47.
- [24] M. Derouich and A. Boutayeb, Dengue fever: mathematical modelling and computer simulation, Applied Mathematics and Computation, 177(2), (2006), 528-544.
- [25] N.M. Ferguson, C.A. Donnelly and R.M. Anderson, Transmission dynamics and epidemiology of dengue: Insights from age-stratied sero-prevalence

surveys, *Philosophical Transactions of the Royal Society of London B.*, **354**, (1999), 757-768.

- [26] Ranjit S, Kissoon N., Dengue hemorrhagic fever and shock syndromes, *Pediatr. Crit. Care Med.*, **12**(1), (January, 2011), 90100. doi:10.1097/PCC.0b013e3181e911a7, PMID 20639791.
- [27] R.M. Anderson and R.M. May, Infectious Diseases of Humans: Dynamics and Contro, Oxford University, London/New York, 1991.
- [28] Safi, A.M., A Thesis Paper on Mathematical Analysis of The Role of Quarantine and Isolation in Epidemiology.
- [29] Sharomi, O., Podder, C. N., Gumel, A.B., Elbasha, E.H. and Watmough, J., Role of incidence function in vaccine-induced backward bifurcation in some HIV models, *Bull. Math. Biol*, **210**, (2007), 436-463.
- [30] Smith H.L. and Waltman P., *The Theory of the Chemostat*, Cambridge University Press, 1995.
- [31] Takahashi LT, Maidana NA, Ferreira WC Jr, Pulino P, Yang HM, Mathematical models for the Aedes aegypti dispersal dynamics: travelling waves by wing and wind, *Bull Math Biol.*, 67, (2005), 509528.
- [32] Vanden Driessche, P. and Watmough, J., Reproduction numbers and subthreshold endemic equilibria for compartmental models of disease transmission, *Math. Biosci*, 180, (2002), 29-48.
- [33] V. Lakshmikantham, S. Leela and A. A. Martynyuk, Stability Analysis of Nonlinear Systems. Marcel Dekker, Inc., New York and Basel, 1989.
- [34] Varatharaj A., Encephalitis in the clinical spectrum of dengue infection, *Neurol. India*, 58(4), (2010), 585591. doi:10.4103/0028-3886.68655. PMID 20739797.
- [35] Whitehorn J, Farrar J., Dengue, Br. Med. Bull., 95, (2010), 161173. doi:10.1093/bmb/ldq019. PMID 20616106.
- [36] Wilder-Smith A., Schwartz E., Dengue in travelers, N Engl. J Med., 353, (2005), 924-932.

- [37] WWW.CDC.Gov/Dengue.
- [38] WWW.google.org/dengue.
- [39] WWW.WHO.int/denguecontrol/faq/en/index6.html.
- [40] WWW.World-Tourism.org/fact(menu.html)
- [41] Z. Feng and X. Jorge Velasco-Hernandez (1997). Competitive exclusion in a vector-host model for the dengue fever. Journal of Mathematical Biology. 35: 523-544.

Table 3.1: Description of variables of the dengue model (8):

Variables	Description			
$S_H(t)$	Susceptible humans			
$E_H(t)$	Exposed humans			
$I_H(t)$	Infected humans			
$M_H(t)$	Migrated class of individuals comes from different parts of the			
	world to the host country and contains the virus of dengue			
$T_H(t)$	Treated humans			
$R_H(t)$	Recovered individuals			
$A_V(t)$	Aquatic class			
$S_V(t)$	Susceptible mosquitoes			
$E_V(t)$	Exposed mosquitoes			
$I_V(t)$	Infected mosquitoes			

Parameter	Description	Baseline values
π_H	Recruitment rate of humans	$20 day^{-1}$ [7]
π_V	Recruitment rate of vectors	$5000 day^{-1}[20]$
$\frac{1}{\mu_H}$	Natural death rate of humans	67 years [20]
$\frac{\frac{1}{\mu_H}}{\frac{1}{\mu_V}}$	Natural death rate of vectors	[4, 14] days[20, 41]
C_{HV}	Contact rate from host to vector	$0.75 day^{-1}[24]$
C_{VH}	Contact rate from vector to host	$0.375 day^{-1}[24]$
σ_H	Exposed individuals with develop clinical symptoms	
	of dengue disease move to infectious class at that rate	$(0,1) day^{-1}$ [29]
σ_V	Exposed vectors develop symptom of disease and	
	move to infections class at this rate	(0,1) assumed
$ au_H$	Rate of treatment	Variable
δ_H	Disease induced death	$10^{-3} day^{-1} [5]$
π_2	Migrated population	Variable
μ_1,μ_2	Transition rates between E_H and I_H classes	Variable
γ_1	Transfer rate from treatment class to recovery class	$0.1428 day^{-1}[5, 24]$
δ_V	Disease induced death rate for infectious	negligible
γ_m	The mean aquatic transition rate	Variable
C_a	Control effect rate	Variable
η_H, η_V	Modification parameters	(0,1][7]
C_m	Control effect rate	Variable
$ heta_c$	Extrinsic incubation rate of vector	Variable

Table 3.2: The value of the parameters of the dengue model (8):

Table 3.3: The values for variables for the figure (3.2–3.11)

S_H	E_H	I_H	M_H	T_H	R_H	A_V	S_V	E_V	I_V
6000	500	300	50	290	280	1000000	10000	5000	3000

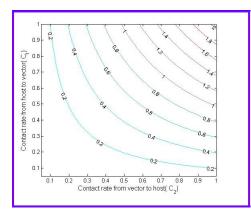


Figure 2: Simulations of the model (8) showing a contour plot of \mathcal{R}_0 as a function of contact rate from vector to host $(C_2 = C_{VH})$ and contact rate from host to vector $(C_1 = C_{HV})$. Parameter values used are as given in table (3.2), (3.3), with $\Pi_H = 20$, $\sigma_V = 0.0130$, $\delta_H = 0.0001$, $\delta_V = 0.01$, $\sigma_H = 0.0230$, $\gamma_1 = 0.0428$, $\gamma_m = 0.00575$, $C_a = 0.850$, $C_m = 0.650$, $\pi_1 = 7$, $\tau_H = 0.190$, $\eta_H = .03902$, $\eta_V = 0.0129$, $\theta_c = .075$, $\mu_1 = 0.0$, $\mu_2 = 0.0$, $\pi_V = 5000$, $\mu_H = 0.01492537$, $\mu_V = 0.363$

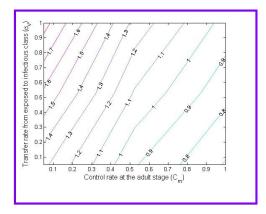


Figure 3: Simulations of the model (8) showing a contour plot of \mathcal{R}_0 as a function of control rate at the adult stage (C_m) and transfer rate from exposed to infected class (σ_V) . Parameter values used are as given in table (3.2), (3.3), with $\Pi_H = 2$, $C_1 = 0.75$, $C_2 = 0.375$, $\delta_H = 0.0001$, $\delta_V = 0.01$, $\gamma_1 = 0.001428$, $\gamma_m = 0.003575$, $C_a = 0.450$, $\pi_1 = 7$, $\tau_H = 0.190$, $\eta_H = .02902$, $\eta_V = 0.0129$, $\theta_c = .075$, $\mu_1 = 0.0$, $\mu_2 = 0.0$, $\pi_V = 5000$, $\mu_H = 0.01492537$, $\mu_V = 0.363$

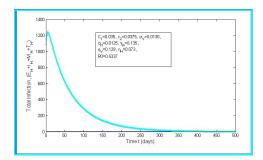


Figure 4: Simulations of the model (8) showing the total number of infected human population $(E_H + I_H + M_H + T_H)$ as a function of time (for reducing values of σ_H and σ_V), using the parameter values in table (3.2) and (3.3) with $\Pi_H = 20, C_1 = 0.035, C_2 = 0.0375, \sigma_V = 0.0130, \sigma_H = 0.01250, \delta_H = 0.0001,$ $\delta_V = 0.01, \gamma_1 = 0.01428, \gamma_m = 0.013575, C_a = 0.0, C_m = 0.0, \pi_1 = 7,$ $\tau_H = 0.0, \eta_H = .12902, \eta_V = 0.073, \theta_c = .075, \mu_1 = 0.0, \mu_2 = 0.0, \pi_V = 5000,$ $\mu_H = 0.01492537, \mu_V = 0.363333, \mathcal{R}_0 = 0.5337.$

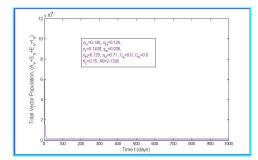


Figure 5: Simulations of the model (8) (without precautionary measures $C_a = C_m = 0$) showing the total number of vector population $(A_V + S_V + E_V + I_V)$ as a function of time, using the parameter values in table (3.2) and (3.3) with $\Pi_H = 20, C_1 = 0.75, C_2 = 0.375, \sigma_V = 0.135, \sigma_H = 0.125, \delta_H = 0.0001, \delta_V = 0.01, \gamma_1 = 0.1428, \gamma_m = 0.035, C_a = 0.0, C_m = 0.0, \pi_1 = 7, \tau_H = 0.0, \eta_H = .02902, \eta_V = 0.037103, \theta_c = 0.75, \mu_1 = 0.0, \mu_2 = 0.0, \pi_V = 5000, \mu_H = 0.01492537, \mu_V = 0.363333, \mathcal{R}_0 = 2.1326.$

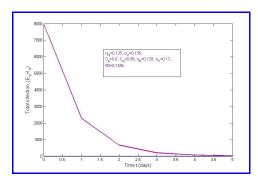


Figure 6: Simulations of the model (8) (with precautionary measures at the adult stage $C_m \neq 0$ and aquatic stage $C_a = 0$) showing the total number of infected vector individuals ($E_V + I_V$) as a function of time, using the parameter values in table (3.2) and (3.3) with $\Pi_H = 20$, $C_1 = 0.75$, $C_2 = 0.375$, $\sigma_V = 0.135$, $\sigma_H = 0.125$, $\delta_H = 0.0001$, $\delta_V = 0.01$, $\gamma_1 = 0.01428$, $\gamma_m = 0.03575$, $C_a = 0.0$, $C_m = 0.89$, $\pi_1 = 7$, $\tau_H = 0.0$, $\eta_H = .129$, $\eta_V = 0.171$, $\theta_c = .0075$, $\mu_1 = 0.0$, $\mu_2 = 0.0$, $\pi_V = 5000$, $\mu_H = 0.01492537$, $\mu_V = 0.363333$, $\mathcal{R}_0 = 0.7455$.

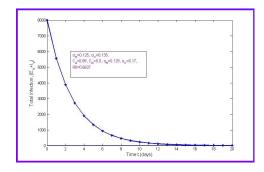


Figure 7: Simulations of the model (8) (with precautionary measures at the aquatic stage $C_a \neq 0$ and $C_m = 0$) showing the total number of infected vector individuals ($E_V + I_V$) as a function of time, using the parameter values in table (3.2) and (3.3) with $\Pi_H = 20$, $C_1 = 0.75$, $C_2 = 0.375$, $\sigma_V = 0.135$, $\sigma_H = 0.125$, $\delta_H = 0.0001$, $\delta_V = 0.01$, $\gamma_1 = 0.01428$, $\gamma_m = 0.03575$, $C_a = 0.89$, $C_m = 0.0$, $\pi_1 = 7$, $\tau_H = 0.0$, $\eta_H = .12902$, $\eta_V = 0.17$, $\theta_c = .01175$, $\mu_1 = 0.0$, $\mu_2 = 0.0$, $\pi_V = 5000$, $\mu_H = 0.01492537$, $\mu_V = 0.363333$, $\mathcal{R}_0 = 0.6637$.

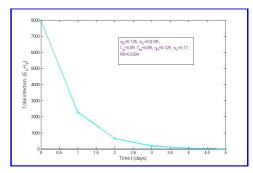


Figure 8: Simulations of the model (8) (with precautionary measures at the aquatic stage $C_a \neq 0$ and both adult stage $C_m \neq 0$) showing the total number of infected vector individuals ($E_V + I_V$) as a function of time, using the parameter values in table (3.2) and (3.3) with $\Pi_H = 20$, $C_1 = 0.75$, $C_2 = 0.375$, $\sigma_V = 0.0135$, $\sigma_H = 0.125$, $\delta_H = 0.0001$, $\delta_V = 0.01$, $\gamma_1 = 0.01428$, $\gamma_m = 0.013575$, $C_a = 0.89$, $C_m = 0.89$, $\pi_1 = 7$, $\tau_H = 0.42$, $\eta_H = 0.129$, $\eta_V = 0.173$, $\theta_c = .01175$, $\mu_1 = 0.0$, $\mu_2 = 0.0$, $\pi_V = 5000$, $\mu_H = 0.01492537$, $\mu_V = 0.363333$, $\mathcal{R}_0 = 0.6304$.

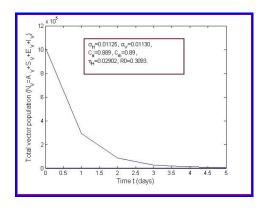


Figure 9: Simulations of the model (8) (with precautionary measures at the aquatic stage $C_a \neq 0$ and both adult stage $C_m \neq 0$) showing the total number of vector individuals $(A_V + S_V + E_V + I_V)$ as a function of time, using the parameter values in table (3.2) and (3.3) with $\Pi_H = 20$, $C_1 = 0.75$, $C_2 = 0.375$, $\sigma_V = 0.01130$, $\sigma_H = 0.01125$, $\delta_H = 0.0001$, $\delta_V = 0.01$, $\gamma_1 = 0.01428$, $\gamma_m = 0.013575$, $C_a = 0.889$, $C_m = 0.89$, $\pi_1 = 7$, $\tau_H = 0.42$, $\eta_H = .02902$, $\eta_V = 0.01137103$, $\theta_c = .01175$, $\mu_1 = 0.0$, $\mu_2 = 0.0$, $\pi_V = 5000$, $\mu_H = 0.01492537$, $\mu_V = 0.363333$, $\mathcal{R}_0 = 0.3093$.

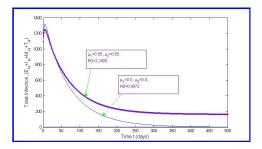


Figure 10: Simulations of the model (8) (with $\mu_1 = \mu_2 \neq 0$) showing the total number of infected human population ($E_H + I_H + M_H + T_H$) as a function of time, using the parameter values in table (3.2) and (3.3) $\Pi_H = 20, C_1 = 0.75,$ $C_2 = 0.375, \sigma_V = .130, \sigma_H = 0.1250, \delta_H = 0.0001, \delta_V = 0.01, \gamma_1 = 0.01428,$ $\gamma_m = 0.03575, C_a = .450, C_m = .650, \pi_1 = 20, \tau_H = 0.19000, \eta_H = .012902,$ $\eta_V = 0.0137103, \theta_c = .075, \mu_1 = 0.050000, \mu_2 = 0.69000, \pi_V = 5000, \mu_H = 0.01492537, \mu_V = 0.363333.$

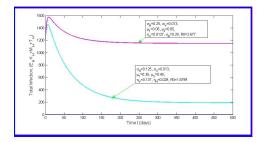


Figure 11: Simulations of the model (8) by considering different values of μ_1 and μ_2 , where $\Pi_H = 20$, $C_1 = 0.75$, $C_2 = 0.375$, $\delta_H = 0.0001$, $\delta_V = 0.01$, $\gamma_1 = 0.01428$, $\gamma_m = 0.03575$, $\pi_1 = 20$, $\theta_c = .075$, $\pi_V = 5000$, $\mu_H = 0.01492537$, $\mu_V = 0.363333$, and other parameters are as in table (3.2) and (3.3).