The Impact of Vaccination, Individual Protection, Treatment and Vector Controls on Dengue

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***Abstract*—In this paper, a compartmental model is presented to investigate the effects of preventive and control measures on dengue disease transmission. It is showed that the model has exactly two disease-free equilibria: a trivial equilibrium, and a biologically realistic disease-free equilibrium. The next-generation matrix technique is used to obtain the basic reproduction number, $\mathcal{R}_0$, associated with the model. This threshold parameter is used to discuss the local stability of the biologically realistic disease-free equilibrium. It is found that the biologically realistic disease-free equilibrium is locally asymptotically stable whenever $\mathcal{R}_0 < 1$, and unstable otherwise. The global asymptotic stability of the model is established using the comparison theorem. For the pre-intervention case, it is showed that the model has a unique positive endemic equilibrium when the basic reproduction number $\mathcal{R}_w$ is above unity, and no endemic equilibrium otherwise. Numerical implementation is carried out on the model and the results of simulation showed that an efficient control of dengue disease relies on the combination of human preventive and vector control measures.**

***Index Terms*—Dengue, control measures, basic reproduction number, stability.**

I. Introduction

DENGUE fever (DF), a vector-borne disease, is a serious global concern [1]–[3] and its emergence and re-emergence have become a major health issue. Dengue is predominant in urban and semi-urban areas of tropical and subtropical locales around the globe [4]. Currently, over 40% of the world's population is at risk of the disease and it is estimated that the entire world may witness about $50-100$ million dengue infections annually with over $20,000$ annual deaths due to DF and up to $500,000$ individuals develop Dengue Hemorrhagic Fever (DHF) or Dengue Shock Syndrome (DSS), a severe case of DF [5].

Dengue viruses are transmitted to human by the bites of infected *Aedes aegypti* and *Aedes albopictus* female mosquitoes, which are known as the primary and secondary vectors, respectively [1], [6]. Also, studies have shown that the virus can be transmitted vertically - a transmission from mosquito to its posterity [1]. Four serologically close but different viruses, identified as DEN 1-DEN 4, cause the disease [6]. Recovery from infection by one serotype grants life-long immunity to that strain but only a temporary cross-immunity to the others [1], [6], [7]. The recovered individual becomes more susceptible to the other three virus strains [6]. Presently, there is no effective vaccine against all the four dengue virus strains, although studies are currently under way. All the treatment are directed at relieving the disease symptoms. As a result, an effective control of dengue disease transmission is focusing on its vectors, relying on reduction of mosquito population through practices such as larviciding, adulticiding and environmental management embracing elimination of breeding sites, and personal protection [1]. These dengue control practices sometimes determine the compartmental framework to be adopted for model formulation.

Mathematical model has turned into an important tool for the comprehension of transmission dynamics of epidemic diseases and to propose control strategies for the diseases. For instance, a deterministic model was used to examine the spread of HIV infection in [8]–[11]. In the context of dengue disease, mathematical model has been used to examine distinctive aspects of the disease. There is a number of works dedicated to the use of compartmental model in gaining insights into the dynamics of dengue disease transmission [12]–[14]. In other studies, mathematical model was used to exploit the available dengue interventions for the disease prevention and control. For example, a compartmental model was constructed to predict the efficacy of hypothetical vaccine on dengue disease spread in [15], [16]. Also, mathematical investigation of the impact of vector control measures in controlling dengue disease transmission was carried out in [17], [18]. The impact of combining an imperfect vaccine together with several vector control measures on the transmission dynamics of dengue disease has also been examined [7], [19].

As there is no perfect vaccine for dengue disease currently, it is necessary to explore the available control measures towards the reduction of mosquito population and prevention of mosquito bite. Meanwhile, this study proposes a compartmental model to gain a proper insight into the impacts of vaccination of susceptible individuals, individual protection against mosquito bites, treatment of infectious humans, mechanical control (destruction of mosquitoes artificial breeding sites), the use of larvicide, and application of adulticide on dengue disease transmission, prevention and control.

The rest of this paper is organized as follows: In Section II, a compartmental dengue model with intervention is proposed. Mathematical analysis of the model, such as positive boundedness, computation of basic reproduction number and stability analysis, is also carried out. In Section III, a compartmental dengue model without intervention is presented, and the endemic equilibrium of the model is obtained. Section IV presents the numerical solution and discussion of results. Finally, conclusion is drawn in Section V.

II. Mathematical Model with Intervention

The compartmental model proposed in this study is based on the mathematical model presented in [20] and the con-

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sideration of [16]. It consists of four epidemiological compartments for humans: $S_h(t)-$ susceptible; $E_h(t)-$ exposed; $I_h(t)-$ infectious; and $R_h(t)-$ recovered (resistant). For the mosquitoes population, two stages of mosquito (immature and winged stages) are taken into consideration. There is an epidemiological state for immature (aquatic) mosquitoes, which includes eggs, larvae and pupae, and three for winged (female) mosquitoes: $A_m(t)-$ aquatic; $S_m(t)-$ susceptible; $E_m(t)-$ exposed; and $I_m(t)-$ infectious.

In addition, six parameters: u_v, u_P, u_T, u_M, u_L and u_A are incorporated into the model. u_V represents the fraction of susceptible individuals one chooses to vaccinate, u_P measures the level of individuals effort to avoid mosquito bites through the use of mosquito repellents, mosquito bed nets and other preventive practices, u_T accounts for the fraction of infectious humans that seek for timely supportive treatment, while u_M, u_L and u_A represent the levels of mechanical control, larviciding and adulticiding, respectively. Vaccination with waning immunity is considered [16].

The dynamics of dengue disease considered in this work is studied and formulated under the assumptions that:
A1: There is no consideration for vertical transmission (i.e., no infected mosquito can transmit dengue virus to its eggs).
A2: At any time t, the total human population is constant. That is,

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t). \tag{1}$$

A3: Aquatic and female mosquito populations are constant and proportional to human population. Mathematically,

$$A_m(t) = kN_h, \tag{2}$$
$$S_m(t) + E_m(t) + I_m(t) = N_m = mN_h \tag{3}$$

for some constants m and k [21].
A4: Human population is homogeneous. This means that every individual in a compartment is homogeneously mixed with other individuals.
A5: Homogeneity exists between human and mosquito populations. This means that each mosquito has the same probability to bite any host.
A6: Immigration is negligible for humans and mosquitoes.
A7: The vaccinated individuals lose their immunity over time.
A8: There is no recovery for an infected mosquito.
A9: Both humans and mosquitoes are born susceptible (i.e., no natural protection).

Suppose $S_h = S_h(t), \dot{S}_h = \frac{dS_h(t)}{dt}$ and similar notations for every other time-dependent state variables, the compartmental model which describes the dynamics between human and mosquito populations is presented by eight time-varying ordinary differential equations (ODEs) given by

$$\dot{S}_h = \mu_h N_h - (1-u_P)\frac{b\beta_{mh} I_m}{N_h} S_h - u_V S_h - \mu_h S_h + \varphi u_V R_h, \tag{4a}$$
$$\dot{E}_h = (1-u_P)\frac{b\beta_{mh} I_m}{N_h} S_h - \gamma_h E_h - \mu_h E_h, \tag{4b}$$
$$\dot{I}_h = \gamma_h E_h - \left[\alpha u_T \sigma_h + (1-u_T)\sigma_h\right] I_h - \mu_h I_h, \tag{4c}$$
$$\dot{R}_h = \left[\alpha u_T \sigma_h + (1-u_T)\sigma_h\right] I_h + u_V S_h - \varphi u_V R_h - \mu_h R_h, \tag{4d}$$

$$\dot{A}_m = \mu_b \left(1 - \frac{A_m}{u_M K}\right)(S_m + E_m + I_m) - \sigma_A A_m - (\mu_A + u_L) A_m, \tag{5a}$$
$$\dot{S}_m = \sigma_A A_m - (1-u_P)\frac{b\beta_{hm} I_h}{N_h} S_m - (\mu_m + u_A) S_m, \tag{5b}$$
$$\dot{E}_m = (1-u_P)\frac{b\beta_{hm} I_h}{N_h} S_m - \gamma_m E_m - (\mu_m + u_A) E_m, \tag{5c}$$
$$\dot{I}_m = \gamma_m E_m - (\mu_m + u_A) I_m, \tag{5d}$$

subject to the initial conditions:

$$\begin{aligned} &S_h(0) = S_{0h}, \quad I_h(0) = I_{0h}, \quad A_m(0) = A_{0m}, \\ &E_h(0) = E_{0h}, \quad R_h(0) = R_{0h}, \quad S_m(0) = S_{0m}, \\ &E_m(0) = E_{0m}, \quad I_m(0) = I_{0m}, \end{aligned} \tag{6}$$

where β_{hm} is the transmission probability of dengue virus from I_h (per bite), μ_h is the human birth and natural death rates (per day), σ_h is the viraemic period (per day), φ is the waning immunity process, α is the rate of effectiveness of anti-arboviral diseases drugs, γ_h is the intrinsic incubation period (per day), b is the average mosquito daily biting rate (per day), σ_A is the development rate from hatchling (larva) to adult mosquito (per day), β_{mh} is the transmission probability of dengue virus from I_m (per bite), μ_A is the natural mortality rate of larvae (per day), μ_m is the mosquito death rate (per day), μ_b is the number of eggs at one deposit per capita (per day), k is the number of larvae per human, γ_m is the extrinsic incubation period (per day), m is the number of female mosquito per human, and K is the maximal capacity of larvae.

A. Mathematical Analysis of the Model

1) Positivity and Boundedness of Solutions: Since Model (4)-(5) is used to describe the dynamics of human and mosquito populations, it is necessary to prove that all the solutions of the state variables with nonnegative initial data remain positive at all time, t for it to be epidemiologically meaningful.

Lemma 1. *Under the dynamics of dengue disease described by Model* (4)-(5)*, the region* Ω *defined by the set*

$$\begin{aligned} \Omega = \big\{&(S_h, E_h, I_h, R_h, A_m, S_m, E_m, I_m) \in \mathbb{R}_+^8 : \\ &S_h + E_h + I_h + R_h \le N_h, A_m \le kN_h, \\ &S_m + E_m + I_m \le mN_h\big\} \end{aligned} \tag{7}$$

is positively invariant.

Proof: For the proof of this lemma, see [22]. ■

Lemma 2. *Let*

$$X(t) = (S_h(t), E_h(t), I_h(t), R_h(t), A_m(t), S_m(t), E_m(t), I_m(t))^T$$

for all t*. Suppose that* $X(0)$ *are nonnegative. Then, the solutions* $X(t)$ *remain nonnegative for all* $t > 0$*.*

Proof: To establish this lemma, let

$$\begin{aligned} \bar{t} = \sup\{t > 0 : &S_h \ge 0, E_h \ge 0, I_h \ge 0, R_h \ge 0, \\ &A_m \ge 0, S_m \ge 0, E_m \ge 0, I_m \ge 0\}. \end{aligned} \tag{8}$$

Thus, $\bar{t} > 0$. Now, from Equation (4a), we have

$$\begin{aligned}\dot{S}_h &= \mu_h N_h - (1-u_P)\frac{b\beta_{mh} I_m}{N_h} S_h - u_V S_h - \mu_h S_h \\ &\quad + \varphi u_V R_h, \\ &= \mu_h N_h - \left((1-u_P) b\beta_{mh}\frac{I_m}{N_h} + u_V + \mu_h\right) S_h \\ &\quad + \varphi u_V R_h, \\ &= \mu_h N_h - (g(t) + u_V + \mu_h) S_h + \varphi u_V R_h, \end{aligned} \tag{9}$$

where $g(t) = (1-u_P) b\beta_{mh}\frac{I_m}{N_h}$.
It follows that

$$\begin{aligned}\frac{d}{dt}\left(S_h \exp\left\{(u_V+\mu_h)x + \int_0^t g(x)\,dx\right\}\right) &= (\mu_h N_h \\ + \varphi u_V R_h)\exp\left\{(u_V+\mu_h)x + \int_0^t f(x)\,dx\right\}.\end{aligned} \tag{10}$$

Integrating both sides of Equation (10) from $t = 0$ to $t = \bar{t}$ yields

$$\begin{aligned}&S_h(\bar{t})\exp\left\{(u_V+\mu_h)\bar{t} + \int_0^{\bar{t}} g(x)\,dx\right\} - S_h(0) \\ &= \int_0^{\bar{t}} (\mu_h N_h + \varphi u_V R_h)\exp\left\{(u_V+\mu_h)z \right. \\ &\quad \left. + \int_0^y h(y)\,dy\right\} dz. \end{aligned} \tag{11}$$

Simplifying, we get

$$\begin{aligned}S_h(\bar{t}) &= S_h(0)\exp\left\{-\left[(u_V+\mu_h)\bar{t} + \int_0^{\bar{t}} g(x)\,dx\right]\right\} \\ &\quad + \exp\left\{-\left[(u_V+\mu_h)\bar{t} + \int_0^{\bar{t}} g(x)\,dx\right]\right\} \\ &\quad \times \int_0^{\bar{t}} (\mu_h N_h + \varphi u_V R_h)\exp\left\{(u_V+\mu_h)z \right. \\ &\quad \left. + \int_0^y h(y)\,dy\right\} dz, \\ &\quad \geq 0. \end{aligned} \tag{12}$$

Clearly, the right-hand side of Equation (12) is a sum of positive terms. Therefore, $S_h(\bar{t})$ is positive. In the same way, the positivity of the quantities $E_h, I_h, R_h, A_m, S_m, E_m$ and I_m for all time t can be proved. This completes the proof. ■

2) Basic Reproduction Number and Local Asymptotic Stability of the Disease-Free Equilibrium:

Theorem 1. *Let Ω be as defined by* (7)*. Also, suppose that*

$$\mathcal{M} = \frac{\mu_b \sigma_A}{(\mu_m + u_A)(\sigma_A + \mu_A + u_L)},$$

where $\mathcal{M}$ is the net reproductive number. Then, there are at most two disease-free equilibria related to the compartmental Model (4)-(5)*:*

1) *If $\mathcal{M} \leq 1$, then there exists a Disease-Free Equilibrium (DFE), a mosquito-free equilibrium which contains only humans (i.e. human without disease and no mosquito) called a Trivial Equilibrium (TE), given by*

$$\mathcal{E}_0 = \left(\frac{k_4\mu_h N_h}{k_4 k_5 - \varphi u_V^2}, 0, 0, \frac{\mu_h u_V N_h}{k_4 k_5 - \varphi u_V^2}, 0, 0, 0, 0\right);$$

2) *If $\mathcal{M} > 1$, then there is a Biologically Realistic Disease-Free Equilibrium (BRDFE), given by*

$$\mathcal{E}_1 = (S_h^*, 0, 0, R_h^*, A_m^*, S_m^*, 0, 0),$$

where

$$\begin{aligned}S_h^* &= \frac{k_4\mu_h N_h}{k_4 k_5 - \varphi u_V^2}, \qquad R_h^* = \frac{\mu_h u_V N_h}{k_4 k_5 - \varphi u_V^2} \\ A_m^* &= \left(1 - \frac{1}{\mathcal{M}}\right) u_M K, \quad S_m^* = \left(1 - \frac{1}{\mathcal{M}}\right)\frac{\sigma_A}{k_7} u_M K.\end{aligned}$$

Proof: To prove this theorem, we follow the solution technique adopted in [23]. Consider Model (4)-(5) at steady state:

$$\dot{X}(t) = 0, \tag{13}$$

where

$$\begin{aligned}\dot{X}(t) = (&\dot{S}_h(t), \dot{E}_h(t), \dot{I}_h(t), \dot{R}_h(t), \dot{A}_m(t), \dot{S}_m(t), \\ &\dot{E}_m(t), \dot{I}_m(t))^T.\end{aligned}$$

Let

$$\mathcal{E}_2 = (S_h^{**}, E_h^{**}, I_h^{**}, R_h^{**}, A_m^{**}, S_m^{**}, E_m^{**}, I_m^{**}) \tag{14}$$

be any arbitrary Endemic Equilibrium (EE) of Model (4)-(5) and

$$\lambda_h = (1-u_P) b\beta_{mh}\frac{I_m}{N_h}, \;\; \lambda_m = (1-u_P) b\beta_{hm}\frac{I_h}{N_h} \tag{15}$$

be the forces of infection of humans and mosquitoes, respectively. Then, at steady state, Equation (15) becomes

$$\lambda_h^{**} = (1-u_P) b\beta_{mh}\frac{I_m^{**}}{N_h^{**}}, \;\; \lambda_m^{**} = (1-u_P) b\beta_{hm}\frac{I_h^{**}}{N_h^{**}}. \tag{16}$$

Now, by virtue of Equations (13) and (15), we have

$$\begin{aligned}\mu_h N_h - \lambda_h S_h - u_V S_h - \mu_h S_h + \varphi u_V R_h &= 0, \quad (17a)\\ \lambda_h S_h - \gamma_h E_h - \mu_h E_h &= 0, \quad (17b)\\ \gamma_h E_h - [\alpha u_T \sigma_h + (1-u_T)\sigma_h] I_h - \mu_h I_h &= 0, \quad (17c)\\ (\alpha u_T \sigma_h + (1-u_3)\sigma_h) I_h + u_V S_h - \varphi u_V R_h & \\ - \mu_h R_h &= 0, \quad (17d)\\ \mu_b\left(1 - \frac{A_m}{u_M K}\right)(S_m + E_m + I_m) - \sigma_A A_m & \\ - (\mu_A + u_L) A_m &= 0, \quad (17e)\\ \sigma_A A_m - \lambda_m S_m - (\mu_m + u_A) S_m &= 0, \quad (17f)\\ \lambda_m S_m - \gamma_m E_m - (\mu_m + u_A) E_m &= 0, \quad (17g)\\ \gamma_m E_m - (\mu_m + u_A) I_m &= 0. \quad (17h)\end{aligned}$$

Solving Equations (17a), (17b), (17c) and (17d) at steady state yields

$$\begin{aligned}S_h^{**} &= \frac{k_1 k_3 k_4 \mu_h N_h}{(k_6 + k_7\lambda_h^{**})}, \qquad E_h^{**} = \frac{k_3 k_4 \mu_h N_h \lambda_h^{**}}{(k_6 + k_7\lambda_h^{**})}, \\ I_h^{**} &= \frac{k_4 \mu_h \gamma_h N_h \lambda_h^{**}}{(k_6 + k_7\lambda_h^{**})}, \quad R_h^{**} = \frac{(k_1 k_3 u_V + k_2\gamma_h\lambda_h^{**})\mu_h N_h}{(k_6 + k_7\lambda_h^{**})},\end{aligned} \tag{18}$$

where, $k_1 = (\mu_h + \gamma_h)$, $k_2 = (\alpha u_T\sigma_h + (1-u_T)\sigma_h)$, $k_3 = (k_2 + \mu_h)$, $k_4 = (\varphi u_V + \mu_h)$, $k_5 = (u_V + \mu_h)$, $k_6 = (k_1 k_3 k_4 k_5 - \varphi k_1 k_3 u_v^2)$ and $k_7 = (k_1 k_3 k_4 - \varphi u_V \gamma_h k_2)$.

Next, the expressions for S_m, E_m and I_m, respectively, in terms of A_m using Equations (17f), (17g) and (17h) are given by

$$\begin{aligned} S_m^{**} &= \frac{\sigma_A}{(k_8+\lambda_m^{**})}A_m^{**}, \\ E_m^{**} &= \frac{\sigma_A\lambda_m^{**}}{k_9(k_8+\lambda_m^{**})}A_m^{**}, \\ I_m^{**} &= \frac{\sigma_A\gamma_m\lambda_m^{**}}{k_8k_9(k_8+\lambda_m^{**})}A_m^{**}, \end{aligned} \tag{19}$$

where, $k_8 = (\mu_m + u_A)$ and $k_9 = (\gamma_m + \mu_m + u_A)$.

Substituting Equation (19) in Equation $(17)_5$ gives

$$\begin{aligned} A_m^{**}\left\{\frac{\mu_b\sigma_A}{k_8k_9}\left(1-\frac{A_m^{**}}{u_MK}\right)\left(\frac{k_8k_9+k_{10}\lambda_m^{**}}{(k_8+\lambda_m^{**})}\right)\right. \\ \left.-k_{11}\right\} = 0, \end{aligned} \tag{20}$$

where, $k_{10} = (k_8 + \gamma_m)$ and $k_{11} = (\sigma_A + \mu_A + u_L)$.

Equation (20) has a trivial solution: $A_m^{**} = 0$. Substituting $A_m^{**} = 0$ in Equation (19) yields $S_m^{**} = 0, E_m^{**} = 0$, $I_m^{**} = 0$. Furthermore, at $I_m^{**} = 0$, $\lambda_h^{**} = 0$. Consequently, $E_h^{**} = 0$, $I_h^{**} = 0$, $R_h^{**} = \frac{\mu_h u_V N_h}{k_4k_5-\varphi u_V^2}$, $S_h^{**} = \frac{k_4\mu_h N_h}{k_4k_5-\varphi u_V^2}$. Therefore, we obtain a TE point:

$$\mathcal{E}_0 = \left(\frac{k_4\mu_h N_h}{k_4k_5-\varphi u_V^2}, 0, 0, \frac{\mu_h u_V N_h}{k_4k_5-\varphi u_V^2}, 0, 0, 0, 0\right).$$

Next, suppose that $A_m^{**} \neq 0$ in Equation (20) so that we seek the possible solution of the following Equation (21):

$$\frac{\mu_b\sigma_A}{k_8k_9}\left(1-\frac{A_m^{**}}{u_MK}\right)\left(\frac{k_8k_9+k_{10}\lambda_m^{**}}{k_8+\lambda_m^{**}}\right) - k_{11} = 0. \tag{21}$$

Resolving Equation (21) leads to

$$A_m^{**} = \left[\frac{\mu_b\sigma_A k_8k_9\left(1-\frac{1}{\mathcal{M}}\right)+\theta\lambda_m^{**}}{\mu_b\sigma_A(k_8k_9+k_{10}\lambda_m^{**})}\right]u_MK, \tag{22}$$

where, $\mathcal{M} = \frac{\mu_b\sigma_A}{k_8k_{11}}$ and $\theta = (\mu_b\sigma_A k_{10} - k_8k_9k_{11})$.

The threshold, $\mathcal{M}$, regulates the existence of mosquitoes. This threshold can be used to establish that Model (4)-(5) has exactly two equilibria with no disease in the population.

In order to compute the TE using the threshold $\mathcal{M}$, set $E_h = I_h = E_m = I_m = 0$, and consequently, $\lambda_h^{**} = \lambda_m^{**} = 0$. Hence, from Equation (22), we have

$$A_m^{**} = \left(1-\frac{1}{\mathcal{M}}\right)u_MK. \tag{23}$$

Suppose that $\mathcal{M} \leq 1$ in Equation (23). Then, we recover the TE given by

$$\mathcal{E}_0 = \left(\frac{k_4\mu_h N_h}{k_4k_5-\varphi u_V^2}, 0, 0, \frac{\mu_h u_V N_h}{k_4k_5-\varphi u_V^2}, 0, 0, 0, 0\right). \tag{24}$$

Next, suppose that $\mathcal{M} > 1$. Using Equation (23) with $\lambda_h^{**} = \lambda_m^{**} = 0$ in Equations (18) and (19) yields the BRDFE:

$$\mathcal{E}_1 = (S_h^*, 0, 0, R_h^*, A_m^*, S_m^*, 0, 0) \tag{25}$$

with

$$\begin{aligned} S_h^* &= \frac{k_4\mu_h N_h}{k_4k_5-\varphi u_V^2}, & R_h^* &= \frac{\mu_h u_V N_h}{k_4k_5-\varphi u_V^2} \\ A_m^* &= \left(1-\frac{1}{\mathcal{M}}\right)u_MK, & S_m^* &= \left(1-\frac{1}{\mathcal{M}}\right)\frac{\sigma_A}{k_7}u_MK. \end{aligned}$$

Hence, the proof. ■

Remark 1. *In the absence of prevention and control measures (i.e., $u_V = u_P = u_T = u_L = u_M = u_A = 0$), we recover the set of DFE points obtained in [20].*

It is important to obtain the basic reproduction number, $\mathcal{R}_0$, for Model (4)-(5). This epidemiological threshold accounts for the average number of dengue secondary cases produced by a typical infected individual introduced into a completely susceptible population [24], [25]. It can be used to predict whether dengue disease will be eradicated or invade a completely susceptible population. When $\mathcal{R}_0 < 1$, each infected individual, on average, produces below one new infected individual, and it is predictable that dengue disease will be cleared from the population. Otherwise, the disease will invade the susceptible population [22]. Hence, using the next generation operator method [24] on Model (4)-(5), the following theorem is established.

Theorem 2. *If $\mathcal{M} > 1$, then the basic reproduction number associated with Model* (4)-(5) *is given by*

$$\mathcal{R}_0 = \sqrt{\frac{(1-u_P)^2b^2\beta_{hm}\beta_{mh}\gamma_m\gamma_h}{k_1k_3k_8k_9}\frac{S_m^*}{N_h^*}\frac{S_h^*}{N_h^*}} \tag{26}$$

where, $k_1 = (\gamma_h + \mu_h)$, $k_3 = (\alpha u_T\sigma_h + (1-u_T)\sigma_h + \mu_h)$, $k_8 = (\mu_m + u_A)$ and $k_9 = (\gamma_m + \mu_m + u_A)$.

Or, equivalently

$$\mathcal{R}_0 = \sqrt{\frac{(1-u_P)^2b^2\beta_{hm}\beta_{mh}\sigma_A\gamma_h\gamma_m k_4u_M k}{k_1k_3k_8^2k_9k_{12}}\left(1-\frac{1}{\mathcal{M}}\right)} \tag{27}$$

where, $k_1 = (\gamma_h + \mu_h)$, $k_3 = (\alpha u_T\sigma_h + (1-u_T)\sigma_h + \mu_h)$, $k_4 = (\varphi u_V + \mu_h)$, $k_8 = (\mu_m + u_A)$, $k_9 = (\gamma_m + \mu_m + u_A)$ and $k_{12} = (\varphi u_V + u_V + \mu_h)$.

The BRDFE, given as $\mathcal{E}_1$, is Locally Asymptotically Stable (LAS) whenever $\mathcal{R}_0 < 1$, and unstable whenever $\mathcal{R}_0 > 1$.

Proof: We prove this theorem by using the technique of next generation matrix method [24], [26], [27]. Considering only the compartments in which the disease is in progression in Model (4)-(5) leads to a subsystem given as

$$\begin{aligned} \dot{E}_h &= (1-u_P)\frac{b\beta_{mh}I_m}{N_h}S_h - \gamma_hE_h - \mu_hE_h, \\ \dot{I}_h &= \gamma_hE_h - [\alpha u_T\sigma_h + (1-u_T)\sigma_h]I_h - \mu_hI_h, \\ \dot{E}_m &= (1-u_P)\frac{b\beta_{hm}I_h}{N_h}S_m - \gamma_mE_m \\ &\quad - (\mu_m + u_A)E_m, \\ \dot{I}_m &= \gamma_mE_m - (\mu_m + u_A)I_m. \end{aligned} \tag{28}$$

Subsystem (28) can be written as $\dot{x} = \mathcal{F}(x) - \mathcal{V}(x)$, where $x = (E_h, I_h, E_m, I_m)^T$, $\mathcal{F}(x)$ accounts for the components associated to the new cases of dengue disease and $\mathcal{V}(x)$ represents the remaining components in the compartments.

It follows that

$$\mathcal{F}(x) = \begin{pmatrix} (1-u_2)b\beta_{mh}\frac{I_m}{N_h}S_h \\ 0 \\ (1-u_2)b\beta_{hm}\frac{I_h}{N_h}S_m \\ 0 \end{pmatrix},$$

$$\mathcal{V}(x) = \begin{pmatrix} k_1E_h \\ k_3I_h - \gamma_hE_h \\ k_9E_m \\ k_8I_m - \gamma_mE_m \end{pmatrix}.$$

Computing and evaluating the Jacobian matrices associated with $\mathcal{F}(x)$ and $\mathcal{V}(x)$ at x_0, respectively, gives

$$J_{\mathcal{F}(x_0)} = \begin{pmatrix} 0 & 0 & 0 & (1-u_P)b\beta_{mh}\frac{S_h^*}{N_h^*} \\ 0 & 0 & 0 & 0 \\ 0 & (1-u_P)b\beta_{hm}\frac{S_m^*}{N_h^*} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$J_{\mathcal{V}(x_0)} = \begin{pmatrix} k_1 & 0 & 0 & 0 \\ -\gamma_h & k_3 & 0 & 0 \\ 0 & 0 & k_9 & 0 \\ 0 & 0 & -\gamma_m & k_8 \end{pmatrix}$$

where, $k_1 = (\mu_h + \gamma_h)$, $k_3 = (\alpha u_T\sigma_h + (1-u_T)\sigma_h + \mu_h)$, $k_8 = (\mu_m + u_A)$, $k_9 = (\gamma_m + \mu_m + u_A)$ and x_0 is a DFE (here, we take $\mathcal{E}_1$ as x_0). Hence, $\mathcal{R}_0 = \rho\left(J_{\mathcal{F}(x_0)}J_{\mathcal{V}(x_0)}^{-1}\right)$, where $\rho(A)$ is the spectral radius (maximum eigenvalue) of a matrix A [24]. Using Maple, $\mathcal{R}_0$ is computed as

$$\mathcal{R}_0 = \sqrt{\frac{(1-u_P)^2b^2\beta_{hm}\beta_{mh}\gamma_m\gamma_h}{k_1k_3k_8k_9}\frac{S_m^*}{N_h^*}\frac{S_h^*}{N_h^*}} \tag{29}$$

where, $k_1 = (\gamma_h + \mu_h)$, $k_3 = (\alpha u_T\sigma_h + (1-u_T)\sigma_h + \mu_h)$, $k_8 = (\mu_m + u_A)$ and $k_9 = (\gamma_m + \mu_m + u_A)$.

Or, equivalently

$$\mathcal{R}_0 = \sqrt{\frac{(1-u_P)^2b^2\beta_{hm}\beta_{mh}\sigma_A\gamma_h\gamma_mk_4u_Mk}{k_1k_3k_8^2k_9k_{12}}\left(1-\frac{1}{\mathcal{M}}\right)} \tag{30}$$

where, $k_1 = (\gamma_h + \mu_h)$, $k_3 = (\alpha u_T\sigma_h + (1-u_T)\sigma_h + \mu_h)$, $k_4 = (\varphi u_V + \mu_h)$, $k_8 = (\mu_m + u_A)$, $k_9 = (\gamma_m + \mu_m + u_A)$ and $k_{12} = (\varphi u_V + u_V + \mu_h)$.

Consequently, by Theorem 2 in [24], the BRDFE, given by $\mathcal{E}_1$, associated with Model (4)-(5) is LAS if $\mathcal{R}_0 < 1$, and unstable if $\mathcal{R}_0 > 1$. This completes the proof of Theorem 2. ■

Remark 2. *Since Model* (4)-(5) *describes dengue disease transmission between human and mosquito populations, then the expression for* $\mathcal{R}_0$ *in Equation* (29) *can be written as*

$$\mathcal{R}_0 = \sqrt{\mathcal{R}_{hm} \times \mathcal{R}_{mh}}, \tag{31}$$

where, $\mathcal{R}_{hm} = \frac{(1-u_P)b\beta_{hm}\gamma_hS_m^*}{N_h^*(\gamma_h+\mu_h)(\alpha u_T\sigma_h+(1-u_T)\sigma_h+\mu_h)}$ *and* $\mathcal{R}_{mh} = \frac{(1-u_P)b\beta_{mh}\gamma_mS_h^*}{N_h^*(\gamma_m+\mu_m+u_A)(\mu_m+u_A)}$.

The expression for $\mathcal{R}_0$ *in Equation* (31) *indicates two routes of infection. These are: transmission from human to mosquito; and from mosquito to human.* $\mathcal{R}_{hm}$ *describes the number of mosquitoes that just one infectious human infects during the period of infectiousness in a susceptible mosquito population. The term* $(1-u_P)b\beta_{hm}\frac{S_m^*}{N_h^*}$ *is the product of the transmission probability of dengue from humans to mosquitoes and the number of susceptible mosquitoes per humans. Also,* $\frac{\gamma_h}{(\gamma_h+\mu_h)}$ *is the proportion of individuals that survive the exposed state (incubation period) to become infectious while* $\frac{1}{(\alpha u_T\sigma_h+(1-u_T)\sigma_h+\mu_h)}$ *is the average duration of humans infectiousness period (i.e., human's viraemic period).*

A similar interpretation holds for $\mathcal{R}_{mh}$*. It signifies the number of humans that one infectious mosquito infects over its expected infectious period in a completely susceptible population. The term* $(1-u_P)b\beta_{mh}\frac{S_h^*}{N_h^*}$ *represents the transmission probability of dengue between humans and mosquitoes in a susceptible population. Also,* $\frac{\gamma_m}{\gamma_m+\mu_m+u_A}$ *is the proportion of mosquitoes that survive the exposed state to become infectious while* $\frac{1}{\mu_m+u_A}$ *is the average duration of mosquitoes infectiousness period.*

3) Global Asymptotic Stability of the Disease-Free Equilibrium: We investigate the global asymptotic stability of Model (4)-(5) by following [28]. Let $X = (S_h, R_h, A_m, S_m)$ and $I = (E_h, I_h, E_m, I_m)$ and group Model (4)-(5) into

$$\frac{dX}{dt} = F(X, 0), \tag{32a}$$

$$\frac{dI}{dt} = G(X, I), G(X, I) = 0, \tag{32b}$$

where, $F(X, 0)$ is the right-hand side of $\dot{S}_h, \dot{R}_h, \dot{A}_m, \dot{S}_m$ with $E_h = I_h = E_m = I_m = 0$ and $G(X, I)$ is the right-hand side of $\dot{E}_h, \dot{I}_h, \dot{E}_m, \dot{I}_m$. Suppose further that $G(X, I)$ satisfies the following two conditions:

$$\begin{aligned} &\text{C1:} \quad G(X, 0) = 0, \text{ and} \\ &\text{C2:} \quad G(X, I) = D_IG(X^*, 0)I - \hat{G}(X, I), \\ &\qquad\qquad \hat{G}(X, I) \geq 0, (X, I) \in \Omega, \end{aligned} \tag{33}$$

where

$$(X^*, 0) = \mathcal{E}_1 = (S_h^*, R_h^*, A_m^*, S_m^*, 0, 0, 0, 0),$$

with $S_h^* = \frac{k_4\mu_hN_h}{(k_4k_5-\varphi u_V^2)}$, $R_h^* = \frac{\mu_hu_VN_h}{(k_4k_5-\varphi u_V^2)}$, $A_m^* = \left(1-\frac{1}{\mathcal{M}}\right)u_MK$, $S_m^* = \left(1-\frac{1}{\mathcal{M}}\right)\frac{\sigma_A}{k_8}u_MK$, $D_IG(X^*, 0)$, a M-matrix with nonnegative off-diagonals, is the Jacobian of $G(X, I)$ obtained with respect to (E_h, I_h, E_m, I_m) and evaluated at $(X^*, 0)$, and Ω is the region where Model (4)-(5) makes sense biologically. If the reduced system, Equation (32), satisfies the conditions in Equation (33), then the following theorem holds.

Theorem 3. *The DFE,* $\mathcal{E}_1$*, of Model* (4)-(5) *is Globally Asymptotically Stable (GAS) in* Ω *whenever* $\mathcal{R}_0 < 1$*, and unstable otherwise.*

Proof: From Model (4)-(5), it follows that

$$F(X, 0) = \begin{pmatrix} \mu_hN_h - (\mu_h + u_V)S_h + \varphi u_VR_h \\ u_VS_h - (\varphi u_V + \mu_h)R_h \\ \mu_b\left(1-\frac{A_m}{u_MK}\right)S_m - (\sigma_A + \mu_A + u_L)A_m \\ \sigma_AA_m - (\mu_m + u_A)S_m \end{pmatrix}$$

and

$$D_I G(X^*,0)=\begin{pmatrix} -k_1 & 0 & 0 & \Phi_h \\ \gamma_h & -k_3 & 0 & 0 \\ 0 & \Phi_m & -k_9 & 0 \\ 0 & 0 & \gamma_m & -k_8 \end{pmatrix}$$

where, $\Phi_h=(1-u_P)b\beta_{mh}\frac{S_h^*}{N_h^*}$ and $\Phi_m=(1-u_P)b\beta_{hm}\frac{S_m^*}{N_h^*}$.

Using the relation of condition C2 in Equation (33) yields

$$\hat{G}(X,I)=\begin{pmatrix} \hat{G}_1(X,I) \\ \hat{G}_2(X,I) \\ \hat{G}_3(X,I) \\ \hat{G}_4(X,I) \end{pmatrix}=\begin{pmatrix} \Psi_h \\ 0 \\ \Psi_m \\ 0 \end{pmatrix} \tag{34}$$

where, $\Psi_h=(1-u_P)b\beta_{mh}\frac{S_h^*}{N_h^*}I_m\left(1-\frac{S_h}{N_h}\frac{N_h^*}{S_h^*}\right)$ and $\Psi_m=(1-u_P)b\beta_{hm}\frac{S_m^*}{N_h^*}I_h\left(1-\frac{S_m}{N_h}\frac{N_h^*}{S_m^*}\right)$.

Since $0\leq S_h$, $0\leq S_m$, $0\leq u_P<1$, and we have that $S_h\leq N_h$, $S_m\leq mN_h$ in Ω, it is obvious that $\hat{G}(X,I)\geq 0$. Also, the DFE

$$X^*=\left(\frac{k_4\mu_h N_h}{(k_4k_5-\varphi u_V^2)},\frac{\mu_h u_V N_h}{(k_4k_5-\varphi u_V^2)},\left(1-\frac{1}{\mathcal{M}}\right)u_M K,\left(1-\frac{1}{\mathcal{M}}\right)\frac{\sigma_A}{k_8}u_M K\right)$$

is clearly a GAS equilibrium point of the reduced system, Equation (32a). Therefore, it follows from Theorem 3 that the DFE,
$\mathcal{E}_1=(X^*,0)$ is GAS. ■

Theorem 3 epidemiologically implies that it is possible to eliminate dengue disease from the population whenever $\mathcal{R}_0<1$ irrespective of the initial sizes of the state variables of Model, Equation (4)-(5).

Apart from the existence of TE and BRDFE, it is necessary to show that the system, Model (4)-(5), has an EE. This is a non-negative steady state solution where dengue disease persists in the population. In order to examine the endemic state solution, we consider the transmission dynamics of dengue disease before intervention.

III. Mathematical Model without Intervention

In the absence of preventive and control measures (i.e., $u_V=u_P=u_T=u_M=u_L=u_A=0$), the system, Model (4)-(5), becomes

$$\begin{aligned} \dot{S}_h&=\mu_h N_h-\frac{b\beta_{mh}I_m}{N_h}S_h-\mu_h S_h \\ \dot{E}_h&=\frac{b\beta_{mh}I_m}{N_h}S_h-\gamma_h E_h-\mu_h E_h, \\ \dot{I}_h&=\gamma_h E_h-\sigma_h I_h-\mu_h I_h, \\ \dot{R}_h&=\sigma_h I_h+u_V S_h-\mu_h R_h, \end{aligned} \tag{35}$$

$$\begin{aligned} \dot{A}_m&=\mu_b\left(1-\frac{A_m}{K}\right)(S_m+E_m+I_m)-\sigma_A A_m-\mu_A A_m, \\ \dot{S}_m&=\sigma_A A_m-\frac{b\beta_{hm}I_h}{N_h}S_m-\mu_m S_m, \\ \dot{E}_m&=\frac{b\beta_{hm}I_h}{N_h}S_m-\gamma_m E_m-\mu_m E_m, \\ \dot{I}_m&=\gamma_m E_m-\mu_m I_m, \end{aligned} \tag{36}$$

subject to the initial conditions:

$$\begin{aligned} &S_h(0)=S_{0h}, \quad I_h(0)=I_{0h}, \quad A_m(0)=A_{0m}, \\ &E_h(0)=E_{0h}, \quad R_h(0)=R_{0h}, \quad S_m(0)=S_{0m}, \\ &E_m(0)=E_{0m}, \quad I_m(0)=I_{0m}. \end{aligned} \tag{37}$$

As a result of biological reasons, the solution properties of Model (35)-(36) together with the initial conditions (37) is studied in the region $\mathcal{G}$ defined by the closed set

$$\mathcal{G}=\mathcal{G}_h\times\mathcal{G}_a\times\mathcal{G}_m\subset\mathbb{R}_+^4\times\mathbb{R}_+\times\mathbb{R}_+^3 \tag{38}$$

with

$$\begin{aligned} \mathcal{G}_h&=\left\{(S_h,E_h,I_h,R_h)\in\mathbb{R}_+^4\middle| S_h+E_h+I_h+R_h\leq N_h\right\}, \\ \mathcal{G}_a&=\left\{A_m\in\mathbb{R}_+\middle| A_m\leq K\right\} \text{ and} \\ \mathcal{G}_m&=\left\{(S_m,E_m,I_m)\in\mathbb{R}_+^3\middle| S_m+E_m+I_m\leq mN_h\right\}. \end{aligned}$$

The same description of parameters and state variables of Model (4)-(5) holds for Model (35)-(36).

Furthermore, the corresponding expression to $\mathcal{R}_0$ given by Equation (30) in the absence of any intervention (i.e., $u_i=0$, $i\in\{V,P,T,M,L,A\}$) is the basic reproduction number related to Model (35)-(36). Therefore, the basic reproduction number of Model (35)-(36) is given by

$$\mathcal{R}_w^2=\frac{b^2\beta_{hm}\beta_{mh}\sigma_A\gamma_m\gamma_h k}{(\gamma_h+\mu_h)(\sigma_h+\mu_h)\mu_m^2(\gamma_m+\mu_m)}\left(1-\frac{1}{\mathcal{N}}\right) \tag{39}$$

where, $\mathcal{N}=\frac{\mu_b\sigma_A}{\mu_m(\sigma_A+\mu_A)}$.

In the next subsection, the threshold $\mathcal{R}_w$ is used to establish the existence of a unique EE.

A. Existence of the Endemic Equilibrium

Here, we claim that Model (35)-(36) has a unique positive EE in the following result.

Theorem 4. *Let $\mathcal{N}>1$. Then, the compartmental dengue Model* (35)-(36) *has a unique positive EE when $\mathcal{R}_w>1$, and no EE otherwise.*

Proof: Let the EE of Model (35)-(36) be represented by

$$\mathcal{E}_3=(S_h^{**},E_h^{**},I_h^{**},R_h^{**},A_m^{**},S_m^{**},E_m^{**},I_m^{**}). \tag{40}$$

Solving Model (35)-(36) at steady state yields the following components of $\mathcal{E}_3$:

$$\begin{aligned} I_h^{**}&=\frac{(\gamma_m+\mu_m)\mu_m^2\mu_h N_h\left(\mathcal{R}_w^2-1\right)}{[\mu_m\mu_h(\gamma_m+\mu_m)N_h b\beta_{hm}+b^2\beta_{mh}\beta_{hm}\sigma_A\gamma_m A_m^{**}]}N_h, \\ S_h^{**}&=N_h-\frac{(\gamma_h+\mu_h)(\sigma_h+\mu_h)}{\mu_h\gamma_h}I_h^{**}, \\ E_h^{**}&=\frac{\sigma_h+\mu_h}{\gamma_h}I_h^{**}, \\ R_h^{**}&=\frac{\sigma_h}{\mu_h}I_h^{**}, \\ A_m^{**}&=\left(1-\frac{1}{\mathcal{N}}\right)K, \\ S_m^{**}&=\frac{\sigma_A}{\mu_m+b\beta_{hm}\frac{I_h^{**}}{N_h}}A_m^{**}, \\ E_m^{**}&=\frac{b\beta_{hm}}{\gamma_m+\mu_m}\frac{I_h^{**}}{N_h}\frac{\sigma_A}{\mu_m+b\beta_{hm}\frac{I_h^{**}}{N_h}}A_m^{**}, \\ I_m^{**}&=\frac{\gamma_m}{\mu_m}\frac{b\beta_{hm}}{\gamma_m+\mu_m}\frac{I_h^{**}}{N_h}\frac{\sigma_A}{\mu_m+b\beta_{hm}\frac{I_h^{**}}{N_h}}A_m^{**}, \end{aligned} \tag{41}$$

where $\mathcal{N} = \frac{\mu_b \sigma_A}{\mu_m(\sigma_A+\mu_A)}$ and $\mathcal{R}_w$ is as defined in Equation (39).

It is clear from Equation (41) that a positive EE exists only at $\mathcal{R}_w > 1$. Hence the proof. ■

IV. Numerical Implementation and Discussion

In this section, we take up the numerical experiments on Model (4)-(5) in order to gain insight into the efficacy of preventive and control measures for dengue under investigation. Model (4)-(5) with the associated state initial conditions (6) is simulated using `ode45` routine in MATLAB. The initial values for the system of ODEs: $S_{0h} = 479350, E_{0h} = 216$, $I_{0h} = 434$, $R_{0h} = 0$, $A_{0m} = kN_h$, $S_{0m} = mN_h$, $E_{0m} = 0$, $I_{0m} = 0$ are taken from [22] while the parameter values and their sources are as presented in Table I. The simulation is carried out in two folds: when the preventive and control measures are applied separately; and when they are combined.

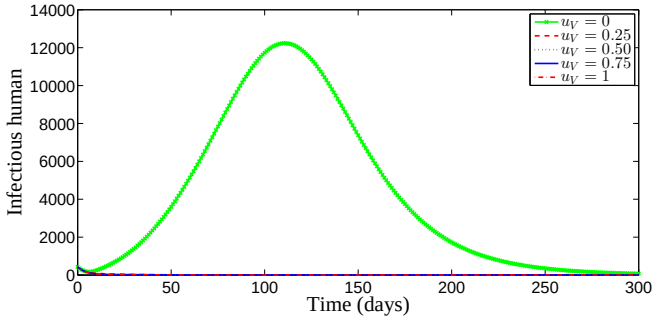
TABLE I: THE VALUES AND SOURCES OF MODEL PARAMETERS

Parameter	Value	Source
β_{hm}	0.25	[18], [29]
μ_h	$\frac{1}{71\times365}$	[16], [22]
σ_h	$\frac{1}{3}$	[16], [22]
φ	0.05	[16]
α	0.3	[30]
$\frac{1}{\gamma_h}$	4 days	[22]
b	1	[22]
σ_A	$\frac{1}{9}$	[18], [29]
β_{mh}	0.25	[18], [29]
μ_A	0.25	[22]
μ_m	$\frac{1}{15}$	[18], [29]
μ_b	6	[16], [18], [22], [29]
k	0.9	[18], [29]
γ_m	$\frac{1}{11}$	[22]
m	6	[22]
K	kN_h	[22]

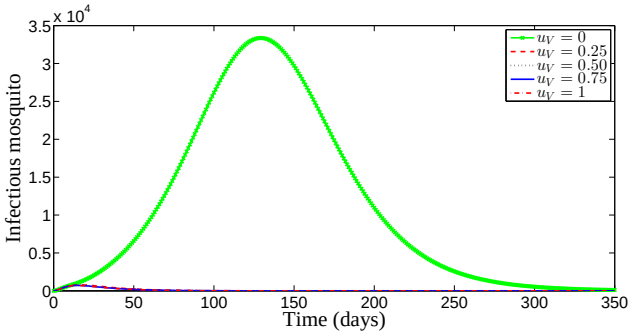
In the absence of the control interventions (that is, $u_i = 0$ for $i = \{V, P, T, M, L, A\}$), the basic reproduction number, $\mathcal{R}_0$, is computed to be 1.5315 for the disease outbreak. The epidemiological indication of this is that dengue disease will persist in the population. Hence, the choice of control parameters, u_V, u_P, u_T, u_M, u_L, and u_A, can put $\mathcal{R}_0$ below unity.

The results of the numerical simulations are presented in Fig. 1- Fig. 9. In the case of no intervention, it can be observed from Fig. 1-Fig. 9 that the peak of the number of human infection occurred between the period of 60 to 150 days. There is a delay in the mosquito infection with the number of infection having a peak between the 80th and the 190th days. The use of certain proportion of the preventive and control measures reduces these peak values of the number of human and mosquito infections almost to zero after the 50th day of dengue outbreak (see Fig. 1, Fig. 2, Fig. 6, Fig. 7, Fig. 8 and Fig. 9).

Examining the impact of the six control measures on the dynamics of infectious humans and mosquitoes separately, it is observed from Fig. 1, Fig. 2 and Fig. 6 that continuous vaccination of 25% of susceptible individuals, the use of individual protection by 75% of susceptible individuals, and application of adulticide with 25% coverage are all enough to decrease the number of infectious humans and mosquitoes to zero. However, the use of either anti-arboviral disease drugs, elimination of artificial water collects or administration of larvicide at mosquitoes breeding sites is not sufficient at any level ($u_i = 0, 0.25, 0.50, 0.75, 1$, $i = \{T, M, L\}$) to decrease the infectious humans and mosquitoes to zero, although the impact of the control measures can be seen as the peak value of the number of both the infectious humans and mosquitoes continued to decrease at each control level as shown in Fig. 3, Fig. 4 and Fig. 5.



(a) Infectious humans

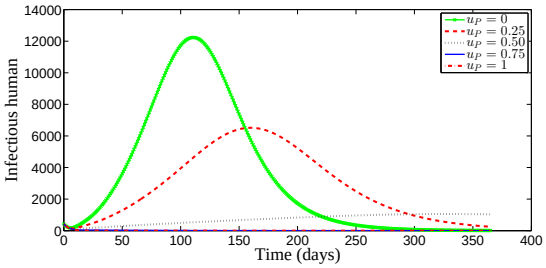


(b) Infectious mosquitoes

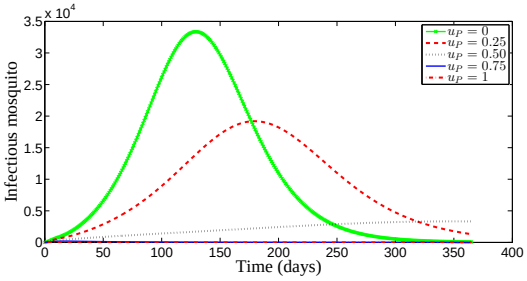
Fig. 1: Infected humans and mosquitoes with various levels of vaccination ($u_V = 0, 0.25, 0.50, 0.75, 1$).

On the other hand, combination of several control measures depicts significant results on dengue disease spread. This strategy of integration of separate use of dengue control measures drastically reduces the large proportion of each control measure needed to eliminate the disease spread. Fig. 7 shows the dynamics of infectious human and mosquito populations when vaccination, individual protection and treatment are simultaneously used at different levels ($u_i = 0, 0.01, 0.05, 0.10, 0.15$, $i = \{V, P, T\}$). It is observed that combining 5% each of the controls is enough to make the number of infectious individuals and mosquitoes to remain near zero. Similarly, Fig. 8 shows the significant impact of mechanical control, larvicide and adulticide at different levels of their combination ($u_i = 0, 0.01, 0.05, 0.10, 0.15$, $i = \{M, L, A\}$) on the dynamics of infectious human and mosquito populations. It is found that only 5% of each control is sufficient for the infectious individuals and mosquitoes to stay close to zero. Furthermore, Fig. 9 presents the dynamics of infectious humans and mosquitoes using the six control measures simultane-

ously at different levels ($u_i = 0, 0.01, 0.05, 0.10, 0.15$, $i = \{V, P, T, L, M, A\}$). It is observed that only 5% of each control is sufficient for the number of infectious individuals and mosquitoes to stay close to zero.

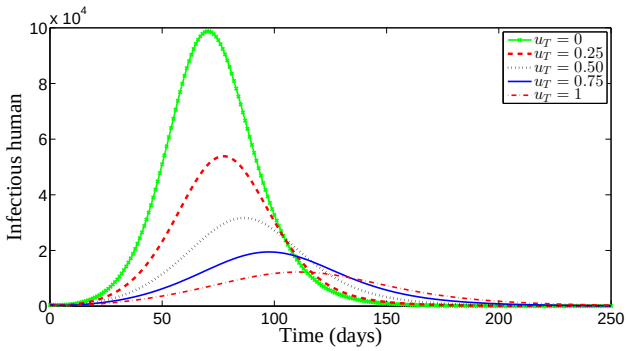


(a) Infectious humans

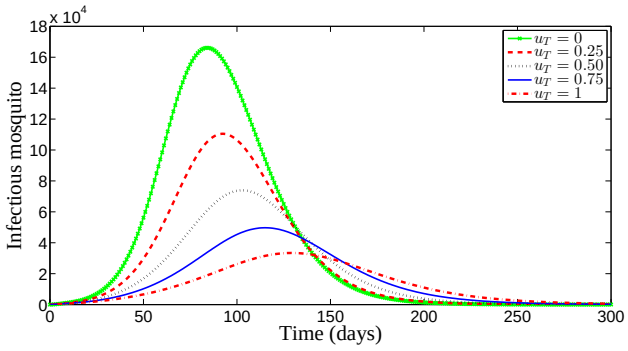


(b) Infectious mosquitoes

Fig. 2: Infectious humans and mosquitoes with various levels of individual protection ($u_P = 0, 0.25, 0.50, 0.75, 1$).

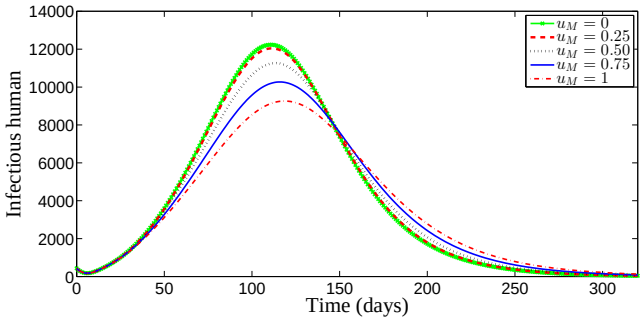


(a) Infectious humans

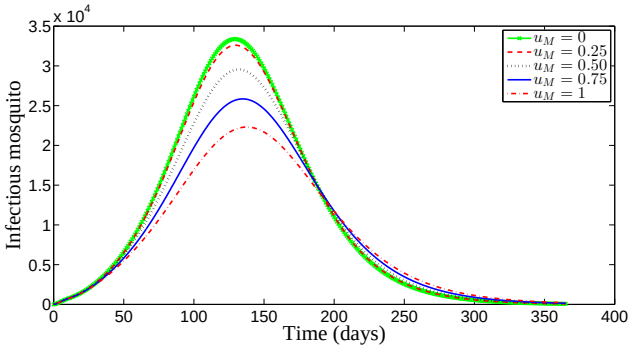


(b) Infectious mosquitoes

Fig. 3: Infectious humans and mosquitoes with various levels of treatment ($u_T = 0, 0.25, 0.50, 0.75, 1$).

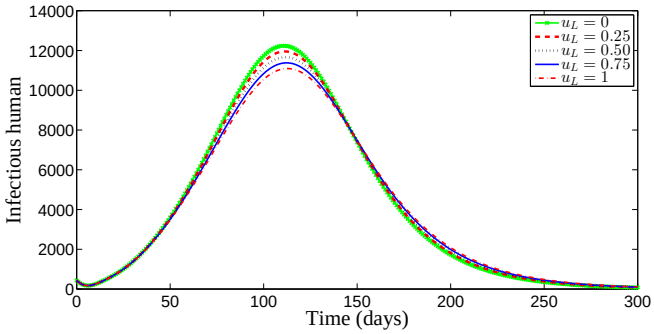


(a) Infectious humans

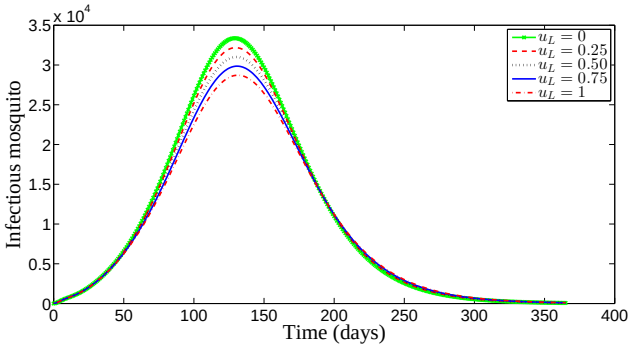


(b) Infectious mosquitoes

Fig. 4: Infectious humans and mosquitoes with various levels of mechanical control ($u_M = 0, 0.25, 0.50, 0.75, 1$).

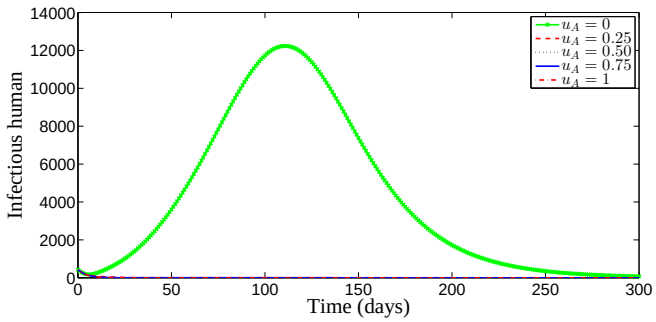


(a) Infectious humans

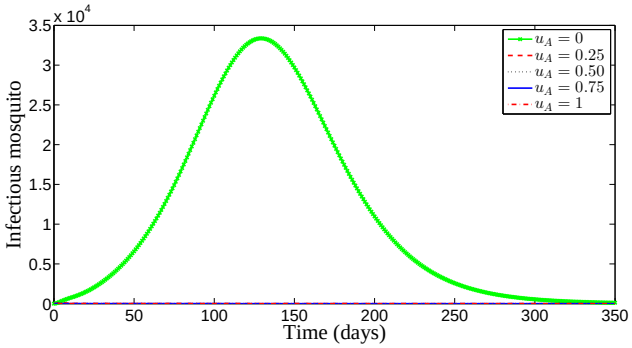


(b) Infectious mosquitoes

Fig. 5: Infectious humans and mosquitoes with various levels of larvicide ($u_L = 0, 0.25, 0.50, 0.75, 1$).

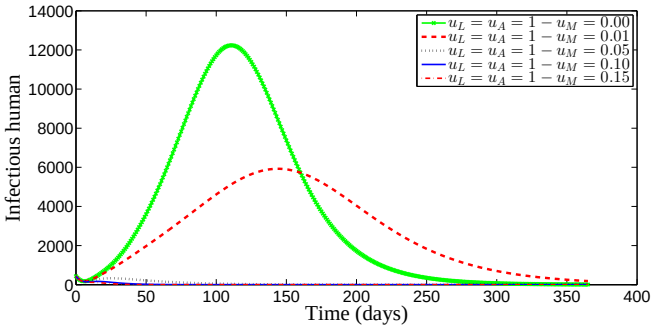


(a) Infectious humans

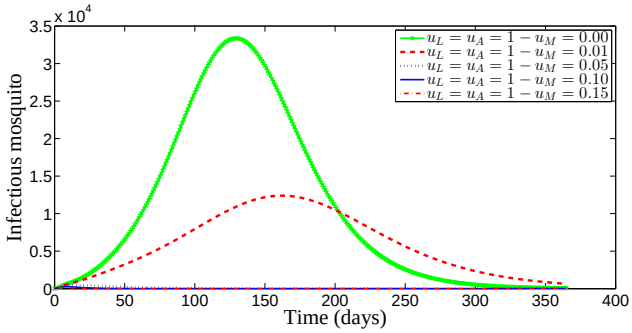


(b) Infectious mosquitoes

Fig. 6: Infectious humans and mosquitoes with various levels of adulticide $(u_A = 0, 0.25, 0.50, 0.75, 1)$.

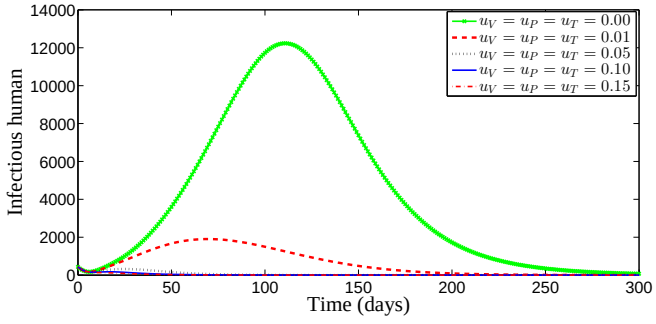


(a) Infectious humans

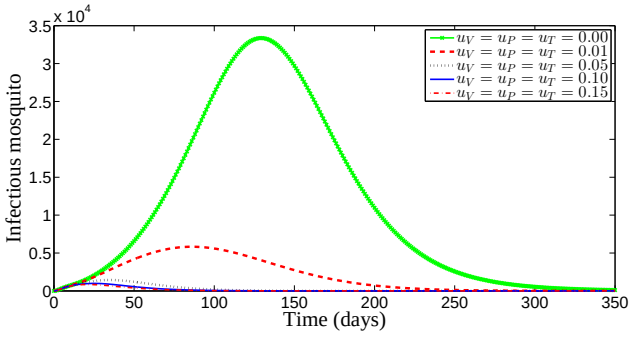


(b) Infectious mosquitoes

Fig. 8: Infectious humans and mosquitoes with various levels of control $(u_L = u_A = 1 - u_M = 0, 0.01, 0.05, 0.10, 0.15)$.

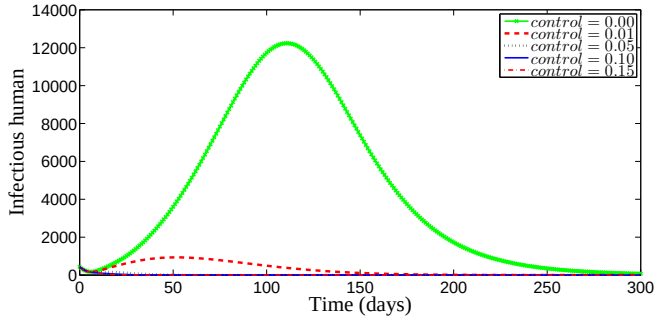


(a) Infectious humans

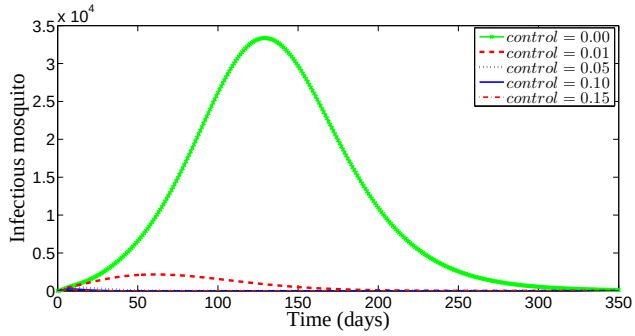


(b) Infectious mosquitoes

Fig. 7: Infectious humans and mosquitoes with various levels of control $(u_V = u_P = u_T = 0, 0.01, 0.05, 0.10, 0.15)$.



(a) Infectious humans



(b) Infectious mosquitoes

Fig. 9: Infected humans and mosquitoes with various levels of control implementation $(u_V = u_P = u_T = 1 - u_M = u_L = u_A = 0, 0.01, 0.05, 0.10, 0.15)$.

V. Conclusion

In this study, a mathematical model was proposed and analysed for dengue disease transmission and control. The model considered both the aquatic and adult stages of mosquito for proper investigation of the impacts of larvicide administered at mosquito breeding sites and open space spray of insecticide on mosquito population control. In addition, other control measures (vaccination, individual protection and treatment) that target human population were

incorporated into the model. We showed that the model has exactly two disease-free equilibria: the TE; and the BRDFE. Computation of $\mathcal{R}_0$ associated with the model was carried out, and it was used to discuss the stability of the BRDFE. It was found that the BRDFE is LAS whenever $\mathcal{R}_0 < 1$, and unstable whenever $\mathcal{R}_0 > 1$. We used the comparison theorem to prove that the BRDFE is GAS. It was showed that, for pre-intervention case, the model has a unique positive EE only at $\mathcal{R}_w > 1$.

From the results of our numerical solution, it was found that focusing on a single control measure for dengue disease requires huge proportion of such measure to eliminate the disease spread. Of all the six control measures considered, only three of them (vaccination, adoption of individual protection practice and application of adulticide) are enough to diminish the number of infected humans and mosquitoes to zero. While the others (treatment, mechanical control and use of larvicide) only reduces the disease burden, but insufficient for the disease elimination. Also, it was found that less equal proportion of each of the control measures is needed to eliminate the disease spread by the following combinations:

1) Vaccination, individual protection and treatment;
2) Mechanical control, larvicide and adulticide; and
3) Vaccination, individual protection, treatment, mechanical control, larvicide and adulticide.

We therefore concluded that combining several control measures guarantees an effective control and minimization of the proportion of control measures require for preventing and curtailing dengue disease transmission.

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