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Mathematical Modeling of the Effect of Therapeutic Vaccine In the Control of Dengue Fever

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Abstract

An eight-compartmental deterministic model for the transmission dynamics of dengue fever with therapeutic vaccine is built and painstakingly analyzed. The model exhibits two equilibria points, namely: the disease-free and endemic. The disease-free equilibrium is locally asymptotically stable when the effective reproductive number (R_f) ***is less than unity and in such a case the endemic equilibrium does not exist. The endemic equilibrium of the model is unique and locally asymptotically stable only when*** $R_f > 1$. ***Finally, numerical simulations show that a therapeutic vaccine with negligible wanning rate which is potent enough to completely eradicate the infectiousness of infected individuals when vaccinated would be sufficient to eradicate the disease burden.***

Key word: Dengue, Epidemic model, Effective reproduction number, Endemic equilibrium, Disease-free equilibrium, Therapeutic vaccine.

1.0 Introduction

Dengue fever (DF) and Dengue Haemorrhagic Fever (DHF) are increasingly important public health problems in the tropic and subtropics areas. Dengue has been recognized in over 100 countries and 2.5 billion people live in areas where dengue is endemic. Because it is caused by one of four serotypes of the dengue virus, it is possible to get dengue fever multiple times. However, an attack of dengue produces immunity for a lifetime to that particular viral serotype to which the patient was exposed [1].The disease affects infants, children and adults and could be fatal. There are 4 distinct, but closely related, serotypes of the virus that cause dengue (DEN-1, DEN-2, DEN-3 and DEN-4).However, cross-immunity to the other serotypes after recovery is only partial and temporary. Subsequent infections by other serotypes increase the risk of developing severe dengue.

After being bitten by a mosquito carrying the virus, the incubation period ranges from three to 15 (usually five to eight) days before the signs and symptoms of dengue appear in stages. Dengue starts with chills, headache, pain upon moving the eyes, appetite loss, feeling unwell (malaise), and low backache. Painful aching in the legs and joints occurs during the first hours of illness. The temperature rises quickly as high as 104 F (40 C), with relatively low heart rate (bradycardia) and low blood pressure (hypo tension). The eyes become reddened. A flushing or pale pink rash comes over the face and then disappears.

The virus is transmitted to humans by the bite of Aedes mosquitoes. (A.aegypti and A. alb opictus are the principal transmissors). The infection in the mosquito is for life. These infected mosquitoes pass the disease to susceptible humans. Individuals who recover from the infection are to become susceptible immediately after recovery [1].

Pathetically, there is still no specific treatment for dengue. Fluid replacement therapy is used if an early diagnosis is made [2]. However, it is believed that any future dengue vaccine would not be able to offer perfect protection against all serotypes. Thus, any future dengue vaccine is expected to be imperfect. It is instructive, therefore, to assess the potential impact of such a vaccine in a community.

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Several researchers [3,4,5] have developed different mathematical models in the literature to gain insights into the transmission dynamics of dengue. The work of the aforementioned researchers showed favourable improvement in curbing dengue fever in the society. It is therefore observant that their commendable work can be exploited to further improve its efficiency that necessitated the urge to embark on this study. To further improve their work, we extended the work of [6] by studying the effect of therapeutic vaccine on the extended model and interesting results were obtained to further express the dynamics of dengue fever

2.0 Description and Analysis of the Model

The model assumes a homogeneous 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 human population at time t, denoted by $N_H(t)$, is sub-divided into five mutually exclusive sub-populations of susceptible humans $S_H(t)$, exposed humans $E_H(t)$, infectious humans $I_H(t)$ vaccinated human $V_H(t)$ and recovered humans $R_H(t)$, so that

$$N_H(t) = S_H(t) + E_H(t) + I_H(t) + V_H(t) + R_H(t).$$

Similarly, the total vector population at time t, denoted by $N_V(t)$ is split into susceptible mosquitoes $S_V(t)$, exposed mosquitoes $E_V(t)$ infectious mosquitoes $I_V(t)$, so that

$$N_V(t) = S_V(t) + E_V(t) + I_V(t).$$

The susceptible human population is generated via recruitment of humans (by birth or immigration) 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 called the force of infection of humans given by

$$\lambda_H = \frac{C_{HV}(N_H, N_V)}{N_V}(\phi_V E_V + I_V) \tag{2.1}$$

where $0 < \phi_V < 1$ which is called the modification parameter accounts for the assumed reduction in transmisibility of exposed mosquitoes relative to infectious mosquitoes. Also, the susceptible vector population is generated via recruitment of vectors usually by birth into community at a constant rate. This population is decreased following infection which can be acquired via effective contact with an exposed or infectious human at a rate called the force of infection of vectors given by

$$\lambda_V = \frac{C_{HV}(N_H, N_V)}{N_V}(\phi_H E_H + \phi_{2H} V_H + I_H) \tag{2.2}$$

$0 < \phi_H < 1$ and $0 < \phi_{2H} < 1$ are called the modification parameters that account for the assumed reduction in transmissibility of exposed humans and vaccinated human relative to infectious humans. It is worth emphasizing that unlike many of the published modeling studies on dengue transmission dynamics, the current study assumes that exposed vectors can transmit dengue disease to humans (that is $\phi_H > 0, \phi_V > 0 \text{ and } \phi_{2H} > 0$).

Table 1: Parameter Description

S/N	Parameters	Meanings	Hypothetical Values	Sources
1	C_{HV}	Disease transmission coefficient	0.068	[6]
2	π_H	Recruitment rate of humans	10	[6]
3	π_V	Recruitment rate of mosquitoes	60	[8]
4	σ_H	Progression rate from	0.53	[6]
		E_H to I_H class		
5	σ_V	Progression rate from	0.2	[6]
		E_V to I_V class		
6	μ_H	Natural death rate of humans	0.0195	[6]
7	μ_V	Natural death rate of mosquitoes	0.06	[8]
8	ϕ_H	Modification Parameter associated	0.99	[6]
		with exposed individuals		
9	ϕ_V	Modification Parameter associated	0.78	[6]
		with exposed mosquitoes		
10	τ_H	Recovery rate of infected humans	0.143	[10]
11	η_H	Vaccinated rate of infected humans	(0,1]	Assumed
12	ω_H	wanning rate of therapeutic vaccine	(0,1]	Assumed
13	δ_H	disease-induced death rate of humans	0.001	[9,10]
14	δ_V	disease-induced death rate of mosquitoes	0	[9]
15	α_H	Recovery rate of vaccinated humans	0.25	[11]
16	θ	Modification parameter associated with	[0,1]	Assumed
		reduced infection of vaccinated humans		
17	$\phi_{2H} = \theta\phi_H$	Modification parameter associated	$\theta\phi_H$	Assumed
		with infection by vaccinated humans		

2.1 Model Equation

The reviewed model is modified to include a therapeutic vaccine compartment for the control of dengue epidemic. We make use of the following deterministic system of nonlinear differential equations to present the model:

$$\begin{aligned}
\dot{S}_H &= \pi_H - S_H(\mu_H + \lambda_H) \\
\dot{E}_H &= \lambda_H S_H - (\mu_H + \sigma_H)E_H \\
\dot{I}_H &= \sigma_H E_H - (\mu_H + \eta_H + \delta_H + \tau_H)I_H + \omega_H V_H \\
\dot{V}_H &= \eta_H I_H - (\mu_H + \alpha_H + \omega_H)V_H \\
\dot{R}_H &= \alpha_H V_H - \mu_H R_H + \tau_H I_H \\
\dot{S}_V &= \pi_V - S_V(\lambda_V + \mu_V) \\
\dot{E}_V &= \lambda_V S_V - (\mu_V + \sigma_V)E_V \\
\dot{I}_V &= \sigma_V E_V - (\mu_V + \delta_V)I_V
\end{aligned} \tag{2.3}$$

where $\dot{S}_H$ *represents* $\dfrac{dS_H}{dt}$

The flowchart of the model in (2.3) is given by Fig1.

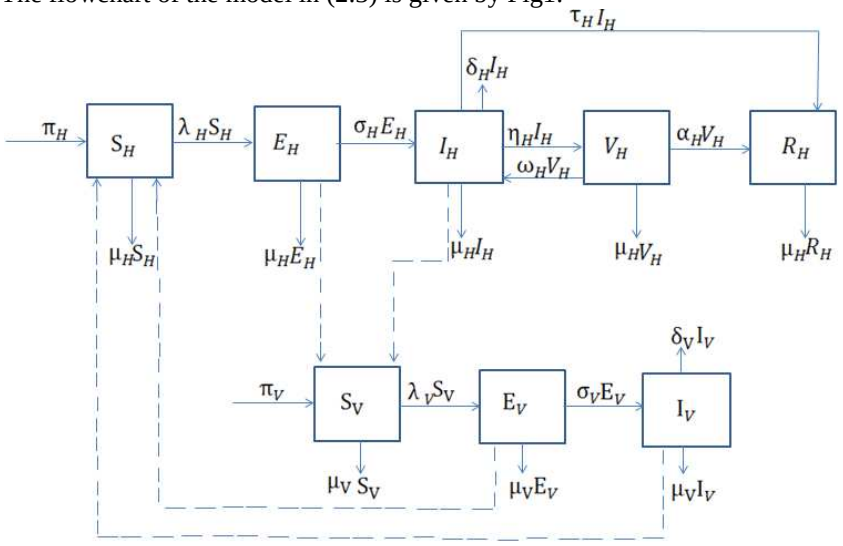

Fig. 1: Flowchart of the Model

2.2 Establishment of the Disease-free Equilibrium Point $\in_\circ$

For the disease-free equilibrium point i.e. (in the absence of disease), the following must hold

$$\dot{S}_H = \dot{E}_H = \dot{I}_H = \dot{V}_H = \dot{R}_H = \dot{S}_V = \dot{E}_V = \dot{I}_V = 0$$

and

$$\lambda_H = \lambda_V = E_H = E_V = I_V = I_H = 0.$$

So doing, we have the DFE point $(\in_\circ)$ of the model as stated below:

$$E_\circ = (\bar{S}_H, \bar{E}_H, \bar{I}_H, \bar{V}_H, \bar{R}_H, \bar{S}_V, \bar{E}_V, \bar{I}_V) = \left(\frac{\pi_H}{\mu_H}, 0,0,0,0, \frac{\pi_V}{\mu_V}, 0,0,0\right) \tag{2.4}$$

2.3 Calculation of Effective Reproduction Number (R_f)

[7] defined the effective reproduction number (R_f) as the average number of secondary cases that one can produce if introduced into a host of population where everyone is susceptible in the presence of treatment. The effective reproduction number will be used to determine the local stability of DFE of the model. It is obtained as the dominant eigenvalue (spectral radius) of the next generation matrix [7].

From the model equation (2.3), we have

$$F = \begin{bmatrix} \dfrac{C_{HV}}{N_H}(\phi_V E_V + I_V)S_H \\ 0 \\ 0 \\ \dfrac{C_{HV}}{N_H}(\phi_H E_H + \phi_{2H} V_H + I_H)S_V \\ 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} (\mu_H + \sigma_H)E_H \\ (\mu_H + \eta_H + \delta_H + \tau_H) - \omega_H V_H - \sigma_H E_H \\ (\mu_H + \alpha_H + \omega_H)V_H - \eta_H I_H \\ (\sigma_V + \mu_V)E_V \\ (\mu_V + \delta_V)I_V - \sigma_V E_V \end{bmatrix}$$

The effective reproduction number $R_f = \rho(FV)^{-1}$, is the spectral radius of the product FV^{-1} and the positive eigenvalue that emerges corresponds to

$$R_f = \frac{C_{HV}\sqrt{A_1 A_2 A_3}}{A_1}$$

where

$$N_H = S_H = \frac{\pi_H}{\mu_H}, N_V = S_V = \frac{\pi_V}{\mu_V}, Q_1 = \mu_H + \sigma_H, Q_2 = \mu_H + \eta_H + \delta_H + \tau_H,$$
$$Q_3 = \mu_H + \alpha_H + \omega_H, Q_4 = \mu_V + \sigma_v, Q_5 = \mu_V + \delta_V, A_1 = \pi_H \mu_V Q_1 Q_4 Q_5 (Q_2 Q_3 - \eta_H \omega_H),$$
$$A_2 = \pi_V \mu_H (\sigma_V + \phi_V Q_5), A_3 = \pi_H (Q_2 Q_3 - \eta_H \omega_H) + \sigma_H (\phi_{2H} \eta_H + Q_3)$$

2.4 Establishment of the Local Stability of the Model

We will establish the local stability of the DFE using the following theorem

Theorem 2.1

The disease-free equilibrium, $\in_o$, of the model (2.3), is locally asymptotically stable (LAS) if $R_f < 1$, and unstable if $R_f > 1$.

Proof

At equilibrium, the model (2.3) is written as

$$\begin{aligned} \pi_H - S_H(\mu_H + \lambda_H) &= 0 \\ \lambda_H S_H - (\mu_H + \sigma_H)E_H &= 0 \\ \sigma_H E_H - (\mu_H + \eta_H + \delta_H + \tau_H)I_H + \omega_H V_H &= 0 \\ \eta_H I_H - (\mu_H + \alpha_H + \omega_H)V_H &= 0 \\ \alpha_H V_H - \mu_H R_H + \tau_H I_H &= 0 \\ \pi_V - S_V(\lambda_V + \mu_V) &= 0 \\ \lambda_V S_V - (\mu_V + \sigma_V)E_V &= 0 \\ \sigma_V E_V - (\mu_V + \delta_V)I_V &= 0 \end{aligned} \qquad (2.5)$$

We form the jacobian matrix of this system as follows

$$J(\in_\circ) = \begin{bmatrix} -\mu_H & 0 & 0 & 0 & 0 & -C_{HV}\phi_V & -C_{HV} \\ 0 & -Q_1 & 0 & 0 & 0 & C_{HV}\phi_V & C_{HV} \\ 0 & \sigma_H & -Q_2 & \omega_H & 0 & 0 & 0 \\ 0 & 0 & \eta_H & -Q_3 & 0 & 0 & 0 \\ 0 & -\frac{C_{VH}\pi_V \mu_H \phi_H}{\pi_H \mu_V} & -\frac{C_{VH}\pi_V \mu_H}{\pi_H \mu_H} & -\frac{C_{VH}\pi_V \mu_H \phi_{2H}}{\pi_H \mu_V} & -\mu_V & 0 & 0 \\ 0 & \frac{C_{VH}\pi_V \mu_H \phi_H}{\pi_H \mu_V} & \frac{C_{VH}\pi_V \mu_H}{\pi_H \mu_V} & \frac{C_{VH}\pi_V \mu_H \phi_{2H}}{\pi_H \mu_V} & 0 & -Q_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_V & -Q_5 \end{bmatrix}$$

By elementary row transformation, it becomes

$$\overline{J}(\in_o) = \begin{bmatrix} -\mu_H & 0 & 0 & 0 & 0 & -C_{HV}\phi_V & -C_{HV} \\ 0 & -Q_1 & 0 & 0 & 0 & C_{HV}\phi_V & C_{HV} \\ 0 & 0 & -Q_2 & \omega_H & 0 & \frac{C_{VH}\pi_V\sigma_H}{Q_1} & \frac{C_{VH}\sigma_H}{Q_1} \\ 0 & 0 & 0 & -\frac{J_1}{Q_1} & 0 & \frac{C_{VH}\pi_V\sigma_H\eta_V}{Q_1} & \frac{C_{VH}\sigma_H\eta_V}{Q_1} \\ 0 & 0 & 0 & 0 & -\mu_V & -\frac{C_{VH}^2\phi_V\pi_V\mu_H A_3}{\pi_H\mu_V Q_1 J_1} & -\frac{C_{VH}^2\pi_V\mu_H A_3}{\pi_H\mu_V Q_1 J_1} \\ 0 & 0 & 0 & 0 & 0 & \frac{J_2}{\pi_H\mu_V Q_1 J_1} & \frac{C_{VH}^2\pi_V\mu_H A_3}{\pi_H\mu_V Q_1 J_1} \\ 0 & 0 & 0 & 0 & 0 & 0 & -\frac{J_3}{J_2} \end{bmatrix}$$

Thus, the diagonal elements are the eigenvalues of system (2.3) at $\in_o$. $\therefore$

$$\lambda_1 = -\mu_H < 0, \lambda_2 = -Q_1 < 0, \lambda_3 = -Q_2 < 0, \lambda_4 = -\frac{J_1}{Q_1} < 0, \lambda_5 = -\mu_V < 0$$

For $R_f \leq 1$, $\lambda_6 = \frac{J_2}{\pi_H\mu_H Q_1 J_1} < 0, \lambda_7 = -\frac{J_3}{J_2} < 0$ where

$$J_1 = Q_2Q_3 - \eta_H\omega_H > 0, J_2 = -\frac{A_1\{\sigma_V + \mu_H\pi_H\phi_V Q_5[1-R_f^2]\}}{Q_5A_2} \text{ and } J_3 = -A_1[1-R_f^2].$$

Hence, whenever $R_f \leq 1$, all the eigenvalues are non positives, thus concluding the proof that the model is locally asymptotically stable at $\in_o$.

2.6 Establishment of the Endemic Equilibrium Point and It's Stability

In order to find the endemic equilibrium point of the model, (i.e. point where at least one of the infected components of the model is non-zero), the following steps are taken, Let $E_1 = (S_H^*, E_H^*, I_H^*, V_H^*, R_H^*, S_V^*, E_V^*, I_V^*, N_H^*)^T$ represents any arbitrary endemic equilibrium of the model (2.3). Solving the equations in (2.3), at steady states gives

$$E_1 = \left\{ \begin{array}{llll} S_H^* = \frac{\pi_H}{\mu_H + \lambda_H^*}, & E_H^* = \frac{\lambda_H^*\pi_H}{(\mu_H + \lambda_H^*)Q_1}, \\ I_H^* = \frac{\sigma_H\lambda_H^*\pi_H Q_3}{Q_1(\mu_H + \lambda_H^*)(Q_2Q_3 - \omega_H\eta_H)}, & V_H^* = \frac{\sigma_H\lambda_H^*\pi_H\eta_H}{Q_1(\mu_H + \lambda_H^*)(Q_2Q_3 - \omega_H\eta_H)}, \\ R_H^* = \frac{\sigma_H\lambda_H^*\pi_H(\alpha_H\eta_H + \tau_H Q_3)}{\mu_H Q_1(\mu_H + \lambda_H^*)(Q_2Q_3 - \omega_H\eta_H)} & S_V^* = \frac{\pi_H}{\mu_V + \lambda_V^*}, \\ E_V^* = \frac{\lambda_V^*\pi_V}{(\mu_V + \lambda_V^*)Q_4} & I_V^* = \frac{\sigma_V\lambda_V^*\pi_V}{(\mu_V + \lambda_V^*)Q_4Q_5}, \\ N_H^* = \frac{(a_1\lambda_H^* + a_2)\pi_H}{Q_1\mu_H(\mu_H + \lambda_H^*)(Q_2Q_3 - \omega_H\eta_H)} & \lambda_H^* = \frac{C_{HV}(\phi_V E_V^* + I_V^*)}{N_H^*}, \\ \lambda_V^* = \frac{C_H V}{N_H^*}(\phi_H E_H^* + \phi_{2H} V_H^* + I_H^*) \end{array} \right\} \quad (2.6)$$

where

$$a_1 = \mu_H(Q_2Q_3 - \omega_H\eta_H) + \sigma_H[Q_3(\mu_H + \tau_H) + \eta_H(\mu_H + \alpha_H)] > a_2 = \mu_H Q_1(Q_2Q_3 - \omega_H\eta_H)$$

It is obvious to note that $(Q_2Q_3 - \omega_H\eta_H) > 0$.

Theorem 2.2

The system (2.3) has a unique positive endemic equilibrium if and only if $R_f > 1$.

Proof

By algebraic manipulation of (2.3), we obtain a quadric equation (in terms of λ_H^*)

$$B_1(\lambda_H^*)^2 + B_2\lambda_H^* + B_3 = 0$$

where

$$B_1 = a_1\pi_H Q_4 Q_5(C_H V A_3\mu_H + a_1\mu_V), B_2 = a_2\pi_H Q_4 Q_5[C_{HV}A_3\mu_H + \mu_V[2a_1 - (Q_2Q_3 - \eta_H\omega_H)R_f^2]]$$

$$B_3 = a_2^2\pi_H\mu_V Q_4 Q_5[1 - R_f^2]$$

It is clear that $B_1 > 0$ since all model parameters are assumed positive and $B_3 < 0$ for $R_f > 1$. Hence, validating that (2.3) has a unique positive endemic equilibrium.

Theorem 2.3

The endemic equilibrium $\in_1$ of the system (2.3) is locally asymptotically stable for $R_f > 1$ and unstable for $R_f < 1$.

Proof

For the sake of convenient multiplication, we again ignore the fifth equation of system (2.3), and obtain its Jacobian matrix evaluated as $\in_1$ as

$$J(\in_1) = \begin{bmatrix} -(\lambda_H^* + \mu_H) & 0 & 0 & 0 & 0 & -\frac{C_{HV}\phi_V S_H^*}{N_H^*} & \frac{C_{HV}S_H^*}{N_H^*} \\ \lambda_H^* & -Q_1 & 0 & 0 & 0 & \frac{C_{HV}\phi_V S_H^*}{N_H^*} & \frac{C_{HV}S_H^*}{N_H^*} \\ 0 & \sigma_H & -Q_2 & \omega_H & 0 & 0 & 0 \\ 0 & 0 & \eta_H & -Q_3 & 0 & 0 & 0 \\ 0 & -\frac{C_{VH}\phi_H S_V^*}{N_H^*} & \frac{C_{VH}S_V^*}{N_H^*} & \frac{C_{VH}\phi_{2H}S_V^*}{N_H^*} & -(\lambda_V^* + \mu_V) & 0 & 0 \\ 0 & \frac{C_{VH}\phi_V S_V^*}{N_H^*} & \frac{C_{VH}S_V^*}{N_H^*} & \frac{C_{VH}\phi_{2H}S_V^*}{N_H^*} & \lambda_V^* & -Q_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & \sigma_V & -Q_5 \end{bmatrix}$$

By elementary row transformation, we get

$$\bar{J}(\in_1) = \begin{bmatrix} -(\lambda_H^* + \mu_H) & 0 & 0 & 0 & 0 & -G_1\phi_V & -G_1 \\ 0 & -Q_1 & 0 & 0 & 0 & \frac{G_1\phi_V\mu_H}{\lambda_H^* + \mu_H} & \frac{G_1\mu_H}{\lambda_H^* + \mu_H} \\ 0 & 0 & -Q_2 & \omega_H & 0 & \frac{G_1\phi_V\mu_H\sigma_H}{Q_1(\lambda_H^* + \mu_H)} & \frac{G_1\mu_H\sigma_H}{Q_1(\lambda_H^* + \mu_H)} \\ 0 & 0 & 0 & -\frac{J_1}{Q_2} & 0 & \frac{G_1\mu_H\phi_V\sigma_H\eta_H}{Q_1Q_2(\lambda_H^* + \mu_H)} & \frac{G_1\mu_H\sigma_H\eta_H}{Q_1Q_2(\lambda_H^* + \mu_H)} \\ 0 & 0 & 0 & 0 & -(\lambda_V^* + \mu_V) & \frac{G_1G_2\mu_H\phi_V A_3}{J_1(\lambda_H^* + \mu_H)} & \frac{G_1G_2\mu_H A_3}{J_1(\lambda_H^* + \mu_H)} \\ 0 & 0 & 0 & 0 & 0 & -\frac{G_3}{G_4} & \frac{G_1G_2\mu_H\mu_V A_3}{G_4} \\ 0 & 0 & 0 & 0 & 0 & 0 & -\frac{G_5}{G_3} \end{bmatrix}$$

Hence, the eigenvalues of the system at $\in_1$ are

$$\lambda_1 = -(\lambda_H^* + \mu_H) < 0, \lambda_2 = -Q_1 < 0, \lambda_3 = -Q_2 < 0, \lambda_4 = -\frac{J_1}{Q_2} < 0$$

$$\lambda_5 = -(\lambda_V^* + \mu_V) < 0, \lambda_6 = -\frac{G_3}{G_4} < 0(\text{if } R_f > 1), \lambda_7 = -\frac{G_5}{G_3} < 0(\text{if } R_f > 1)$$

where

$$G_3 = \frac{a_2\pi_V^2}{[a_3\lambda_H^* + a_2\mu_V]^2 S_H^* S_V^* \pi_V \mu_H Q_5}\pi_H Q_4 Q_5 a_3\lambda_H^*(a_3\lambda_H^* + 2a_2\mu_V) + a_2\mu_H\mu_V[A_1(1-R_f^2) + C_{HV}^2\mu_H\sigma_V A_3]$$

$$G_4 = Q_1 J_1(\lambda_H^* + \mu_H)(\lambda_V^* + \mu_V)$$

$$G_5 = \frac{Q_1 Q_4 Q_5 \pi_H \pi_V J_1}{[a_3\lambda_H^* + a_2\mu_V]^2 S_H^* S_V} a_3\lambda_H^*(a_3\lambda_H^* + 2a_2\mu_V) + a_2^2\mu_V^2(1-R_f^2)$$

$$a_2 = C_{HV}A_3\mu_H + a_1\mu_V$$

It is instructive to note that the denominators of G_3 and G_5 are positive if $R_f > 1$, but the numerators are not expressively non negative when $R_f > 1$. Thus, to establish that G_3 and G_4 are positive, we further simplify their numerator to have,

$$G_5 = \frac{Q_1 Q_4 Q_5 \pi_H \pi_V J_1[C_{HV}A_3\mu_H a_3(\lambda_H^*)^2 + \mu_V a_2[C_{HV}A_3\mu_H + \mu_V J_1 R_f^2]\lambda_H^*]}{[a_3\lambda_H^* + a_2\mu_V]^2 S_H^* S_V^*}$$

$$G_3 = \frac{a_2\pi_V}{[a_3\lambda_H^* + a_2\mu_V]^2 S_H^* S_V^* \mu_H Q_5}\times$$

$$[\pi_H Q_4 Q_5[a_3(\lambda_H^*)^2 + a_2\pi_H^2\mu_V\lambda_H^*(C_{HV}A_3\mu_H + J_1\mu_V R_f^2)] + C_{HV}^2\mu_H^2\sigma_V\mu_V A_3 a_2]$$

Since the eigenvalues are all negative when $R_f > 1$, we conclude that the system is locally asymptotically stable at $\in_1$.

3.0 Numerical Simulation and Discussion of Results

In this section, we perform numerical simulation of model (2.3) to study the dynamical behavior of the model and show that both the quantitative and qualitative results are in agreement.

Table 2: Effect of R_f on number of Dengue fever cases at steady state.

S/N	θ	η_H	ω_H	R_f	$E_H^* + I_H^* + V_H^*$	$E_V^* + I_V^*$	Remarks
1	1	0.1	1	1.0295	3.4549	7.5567	$\in_1$ stable (no eradication)
2	1	0.2	1	1.0145	0.9782	2.1496	$\in_1$ stable (no eradication)
3	1	0.2	0.9	1.0122	0.7720	1.6972	$\in_1$ stable (no eradication)
4	0.8	0.2	0.9	1.0011	0.0586	0.1263	$\in_1$ stable (no eradication)
5	0.8	0.4	0.6	0.9539	0	0	$\in_o$ stable(disease eradication)
6	0.8	0.4	0.4	0.9389	0	0	$\in_o$ stable(disease eradication)
7	0.6	0.4	0.4	0.9118	0	0	$\in_o$ stable(disease eradication)
8	0.4	0.6	0.2	0.8155	0	0	$\in_o$ stable(disease eradication)
9	0.2	0.8	0.2	0.7433	0	0	$\in_o$ stable(disease eradication)
10	0	0.8	0.2	0.6913	0	0	$\in_o$ stable(disease eradication)
11	0	1	0	0.6126	0	0	$\in_o$ stable(disease eradication)

Note: Table 2 is generated by using parameter value in Table 1 while varying the values of θ, η and ω.

The results displayed in Table 2, show that dengue fever infections increases as R_f increases. Furthermore, the qualitative results are validated, since the table shows dengue fever can be eradicated when $R_f < 1$ and persists for values of $R_f > 1$. It is paramount to note that decrease in either ϕ_{2H}, ω_H or both reduces total number of dengue infection cases and increase in η_H will also reduces disease burden.

4.0 Conclusion

In this paper, the epidemiological dynamics of dengue fever in the presence of therapeutic vaccine was qualitatively and quantitatively explored by deriving and analyzing an eight-dimensional deterministic model. The effective reproduction number R_f, is computed and used to establish the local stability of the two equilibria (i.e. the disease-free and endemic equilibrium). The equilibrium corresponding to disappearance of disease $\in_o$ is locally asymptotically stable if $R_f < 1$ while the unique endemic equilibrium $\in_1$, is locally asymptotically stable whenever $R_f > 1$. Numerical simulations are in agreement with the qualitative results and reveal that a therapeutic vaccine with negligible wanning rate, which is potent enough to stop the infectiousness of infected individuals when vaccinated would be beneficial in eradicating the disease burden.

5.0 References

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