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# Stability Analysis of Disease-Free Equilibrium of Dengue Fever Transmission Dynamics with Mild and Severe Compartments Incorporating Two-Dose Primary Vaccine

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## *Abstract*

In this paper, sensitivity analysis of disease-free equilibrium of dengue fever transmission dynamics with mild and severe compartments incorporating two-dose primary vaccine is carried out. The model considered the human population $N_h$ and the vector population $N_v$, which were further subdivided into eight classes. The model Disease Free Equilibrium (DFE) was obtained and analysed for stability. The effective reproduction number $R_C$ for the model was also computed and the result shows Locally Asymptotically Stable (LAS) when $R_C < 1$ and Globally Asymptotically Stable (GAS) when $R_C \leq 1$ at dengue free equilibrium, otherwise unstable whenever $R_c > 1$.

**Keywords**: dengue; basic reproduction number; vaccine; equilibria; stability.

## *Introduction*

Dengue fever is a mosquito-borne viral infection caused by Dengue Virus (DENV), it mostly occurs in tropical and sub-tropical areas of the world (World Health Organisation [WHO], 2019b). It is one of the most significant and widely spread mosquito viral diseases vectored primarily by *Aedes aegypti* and the secondary vectors are *Aedes*

*albopictus* and *Aedes galliosi* (Isa *et al.,* 2021). There are four types of dengue virus which are genetically related but distinct serotypes, these are; dengue virus 1, dengue virus 2, dengue virus 3, and dengue virus 4 (Gubler *et al.* 2014). A person who recovered from an infection caused by one of the virus serotypes remains immune for life to that type but susceptible to other types, and the victim may as well have Dengue Hemorrhagic Fever (DHF) or Dengue Shock Syndrome (DSS), a situation known as severe dengue (WHO 2023).

The Symptoms of dengue fever may include high fever, headache, joint and muscle pains, skin irritation, nausea, sore throat, vomiting, and skin rashes. These appear between three to fourteen (3-14) days after coming in contact with the virus. Dengue virus is transmitted from human to mosquito and vice versa, these happen when a female vector feeds on the blood of human. Human to human transmission is through organ transplant, direct blood transfusion and other blood related substances (WHO 2023).

Approximately over 3.9 billion people in more than 129 countries are at risk of dengue virus infection with more than 390 million dengue fever infections occurring per year in which 96 million manifests clinically (WHO, 2023). The mortality rate of dengue fever increases from 0.8% - 2.5% and up to 26% in some critical cases (*Kularatne, 2015; Ranjit and Kissoon, 2011; WHO 2009).*

There is no cure for dengue fever although, health personal recommended maintaining body fluid to avoid dehydration, which can cause the fever to be high. Patients with low level of hydration are recommended to take oral dehydration therapy and some bed rest. While those with severe dengue fever signs, intravenous fluid administration is recommended in addition to bed rest and close monitoring (Taghikhani *et al.,* 2020 and WHO 2017). Dengue fever is more life threatening when the patient is having a chronic disease like, hepatitis, high blood pressure or diabetes (WHO 2019a).

Currently, there are two types of dengue fever vaccine recommended so far. Dangvaxia (CYD – TDV), which is approved and licensed in December 2015 by WHO and Qdenga, which has been approved by the European Medicines Agency (EMA) on 5th December, 2022 (Robert Koch Institute, 2023).

**Statement of the Problem**

Almost half of the population of the world are liable to have dengue with almost four hundred million cases every year despite the fact that numerous studies on mathematical model of dengue fever have been carried out by many different authors and researchers over years, using different styles of modelling and control strategies. Some authors like, Yang and Ferreira (2008), Wei *et al.* (2008), Andrand *et al.* (2012), Anggriani *et al*. (2013), Phaijoo and Gurung (2017), and Kim *et al.* (2020), consider only human and mosquito populations, while Eegunjobi *et al.* (2023), added two-strain. Others included vaccine, like Garba *et al*. (2008) and Carvalho *et al.* (2015). None of the aforementioned formulated a model considering mild and severe compartments in the human population. This paper will propose a new

mathematical model of dengue fever transmission dynamics with mild and severe compartments incorporating double dose primary vaccine.

**Objectives**

The aim of the research is achieved through the following objectives; to formulate a mathematical model of dengue fever transmission dynamics with mild and severe compartments incorporating double-dose primary vaccine, to determine the Disease-Free Equilibrium (DFE) of the model, to compute the effective reproduction number $R_C$ for the model and to analysed the DFE for stability.

**Research Question**

Is the disease-free equilibrium state of the model Locally Asymptotically Stable (LAS) when $R_C < 1$ and Globally Asymptotically Stable (GAS) when $R_C \leq 1$? Is it otherwise unstable whenever $R_c > 1$?

**Development of the Model**

The total human population $N_h(t)$ is subdivided into six (6) subpopulation classes, namely: Susceptible human $S(t)$, Vaccinated human $V(t)$, Mildly Infected human $M(t)$, Severely Infected human $W(t)$, Hospitalised human $H(t)$ and Removed human $R(t)$, such that:

$$N_h(t) = S(t) + V(t) + M(t) + W(t) + H(t) + R(t). \tag{1}$$

On the other hand, the vector (*Aedes aegypti* mosquito) population $N_v(t)$ is subdivided into two (2) classes. Adult Female Susceptible mosquitos $F_n(t)$ and Adult Female mosquitos infected with dengue virus $F_d(t)$, such that:

$$N_v(t) = F_n(t) + F_d(t). \tag{2}$$

The susceptible human population class $S(t)$, is generated through the recruitment rate $\Lambda_h$. The class is decreased by natural death at the rate of $\mu_h$ and the rate of first dose of vaccine $\rho_1$. The force of infection $\lambda_1 = \frac{\beta_1 F_d}{N_h}$, also reduces the population of $S(t)$. The vaccinated

class $V(t)$ increases with taking the first dose of vaccine at the rate of $\rho_1$ and deceases by the number of population who took the second dose of vaccine at the rate of $\rho_2$, loss of efficacy of the first dose of vaccine at the rate of $(1-\nu)$ as well as natural death at the rate of $\mu_h$.

Mildly infected class $M(t)$ increases through the infection of $S(t)$ at the rate of $\lambda_1 = \frac{\beta_1 F_d}{N_h}$, as well as the loss of efficacy of the first dose at the rate of $(1-\nu)$. The class decreases through the progression of the mild population class $M(t)$ to the severe class at the rate of $\sigma$. It also decreases by natural recovery at the rate of $\gamma$ as well as natural death at the rate of $\mu_h$.

The severely infected compartment $W(t)$, increases by the progression of mildly infected population at the rate of $\sigma$. This is as a result of not taking proper vaccination. The class decreases as a result of natural death $\mu_h$, death induced by dengue fever at the rate of $\delta_1$, as well as hospitalisation (quarantine) at the rate of $\psi$. However, hospitalisation of members of this population will increase the population of hospitalisation class $H(t)$ at the rate of $\psi$, which eventually decreases by the natural death rate $\mu_h$, death induced by dengue fever at the rate of $\delta_2$, as well as treatment at the rate of $\tau$.

The population of the Removed class $R(t)$ increases through the second dose of vaccination by the Vaccinated class at the rate of $\rho_2$, the natural recovery of the Mildly infected population at the rate of $\gamma$ and treatment of the hospitalised class at the rate of $\tau$. The compartment decreases through natural death at the rate of $\mu_h$.

In the mosquito population, the susceptible mosquito class $F_n(t)$, is generated through recruitment at the rate of $\Lambda_v$. The class decreases through natural death rate $\mu_v$ and death induced by human actions $\delta_v$. It also decreases as a result of interaction with $M(t)$ and $W(t)$ at the rate of infection $\frac{\beta_2 (M + \eta W) F_n}{N_v}$, thereby increasing the population of infected mosquito class $F_d(t)$, which eventually decreases by natural death rate $\mu_v$ and death induced by human actions at the rate of $\delta_v$.

The schematic diagram of the model is given in figure 1

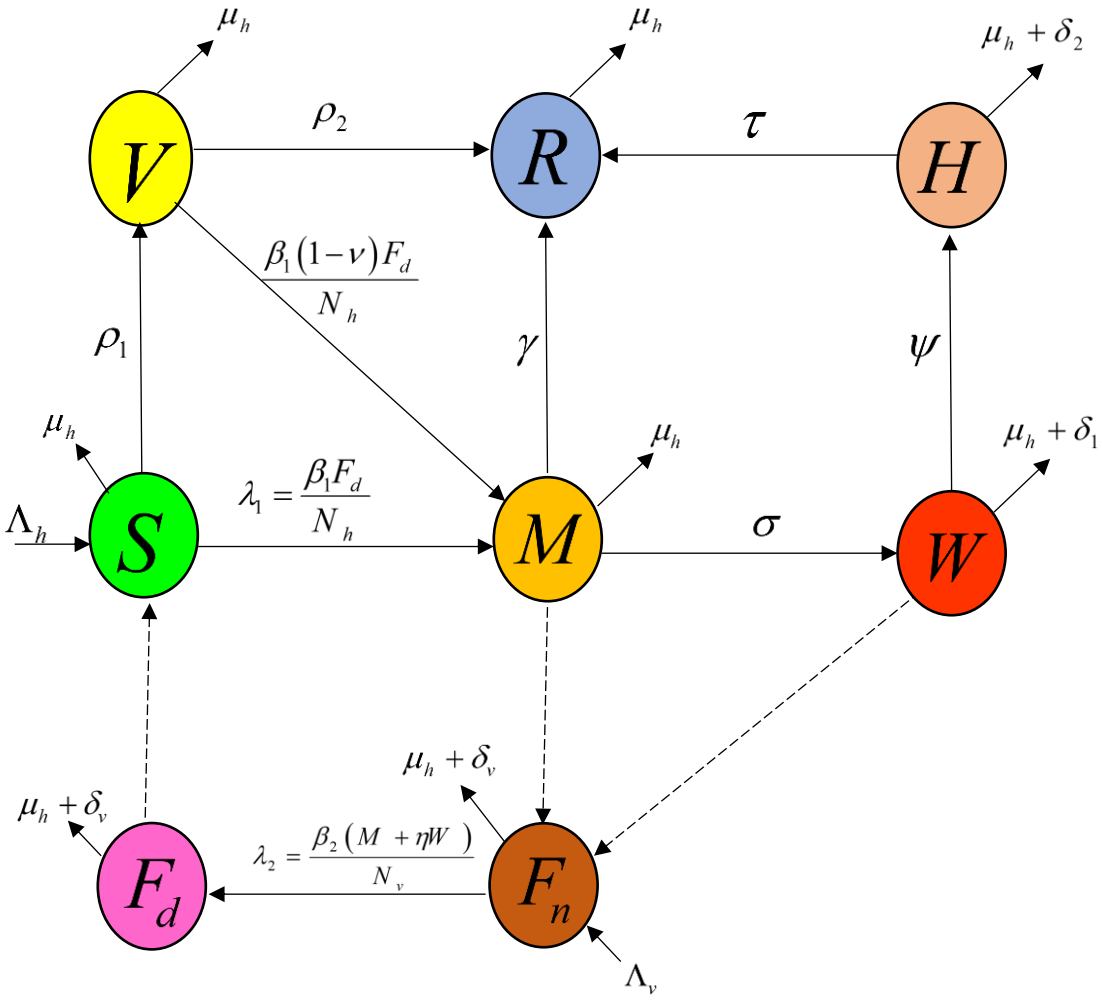



**Figure 1: Schematic diagram with vaccine**

The mathematical equations representing the model is given in equations (3) - (12),

$$\frac{dS}{dt} = \Lambda_h - \lambda_1 S - k_1 S \quad (3)$$

$$\frac{dV}{dt} = \rho_1 S - \lambda_1(1-\nu)V - k_2 V \quad (4)$$

$$\frac{dM}{dt} = \lambda_1 S + \lambda_1(1-\nu)V - k_3 M \quad (5)$$

$$\frac{dW}{dt} = \sigma M - k_4 W \quad (6)$$

$$\frac{dH}{dt} = \psi W - k_5 H \quad (7)$$

$$\frac{dR}{dt} = \rho_2 V + \gamma M + \tau H - \mu_h R \quad (8)$$

$$\frac{dF_n}{dt} = \Lambda_v - \lambda_2 F_n - k_6 F_n \quad (9)$$

$$\frac{dF_d}{dt} = \lambda_2 F_n - k_6 F_d \quad (10)$$

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_1 W - \delta_2 H \quad (11)$$

$$\frac{dN_v}{dt} = \Lambda_v - k_6 N_v \quad (12)$$

where,

$$\lambda_1 = \frac{\beta_1 F_d}{N_h} \quad (13)$$

$$\lambda_2 = \frac{\beta_2 (M + \eta W)}{N_v} \quad (14)$$

$$k_1 = (\rho_1 + \mu_h) \quad (15)$$

$$k_2 = (\rho_2 + \mu_h) \quad (16)$$

$$k_3 = (\gamma + \sigma + \mu_h) \quad (17)$$

$$k_4 = (\psi + \mu_h + \delta_1) \quad (18)$$

$$k_5 = (\tau + \mu_h + \delta_2) \quad (19)$$

$$k_6 = (\mu_v + \delta_v) \quad (20)$$

**Variables and Parameters Used**

$S(t)$ Population of susceptible human at time t.

$V(t)$ Population of vaccinated human at time t.

$M(t)$ Population of mildly infected human at time t.

$W(t)$ Population of severely (worse case) infected humans at time t.

$H(t)$ Population of hospitilised human at time t.

$R(t)$ Population of removed human (permanent recovery) at time t.

$F_n(t)$ Population of non-*Wolbachia* (wild) adult female mosquitos at time t.

$F_d(t)$ Population of carrier mosquitos with dengue virus at time t.

$N_h(t)$ Total population of human at time t.

$N_v(t)$ Total population of vector at time t.

$\Lambda_h$ Recruitment rate for susceptible human population

$\Lambda_v$ Recruitment rate of female adult non-*Wolbachia* (wild) mosquito

$\mu_h$ Natural death rate of human

$\mu_h$ Natural death rate of mosquito

$\delta_1$ Death induced by dengue virus infection at severe stage

$\delta_2$ Death induced by dengue virus infection at hospitalisation stage

$\delta_v$ Death caused by human activities against mosquito.

$\beta_1$ Effective contact rate as susceptible human is infected with dengue virus

$\beta_2$ Effective contact rate as susceptible mosquito is infected with dengue virus

$\rho_1$ Vaccination rate for first dose of Qdangua vaccine

$\rho_1$ Vaccination rate for second dose of Qdangua vaccine

$\gamma$ Rate of recovery from mild infection

$\nu$ Vaccine efficacy rate

$\sigma$ Rate of progression to severe case

$\eta$ Parameter associated with decrease infectious rate

$\psi$ Rate of hospitalisation

$\tau$ Treatment rate

## MODEL ANALYSIS

### Dengue Free Equilibrium State $\left(E^0\right)$

At the disease-free equilibrium state there is absence of infections $\left(\lambda_1^* = \lambda_2^* = 0\right)$. Setting the left-hand side of equation (3) to (12) to zero and solving them simultaneously, we have the Disease Free Equilibrium (DFE) state of model which exists at the point,

$$E^0 = \left(\frac{\Lambda_h}{k_1}, \frac{\rho_1\Lambda_h}{k_1k_2}, 0, 0, 0, \frac{\rho_1\rho_2\Lambda_h}{\mu_h k_1 k_2}, \frac{\Lambda_v}{k_6}, 0, \frac{\Lambda_h}{\mu_h}, \frac{\Lambda_v}{k_6}\right) \tag{21}$$

where,

$$E^0 = \left(S^0, V^0, M^0, W^0, H^0, R^0, F_n{}^0, F_d{}^0, N_h{}^0, N_v{}^0\right) \tag{22}$$

### Effective Reproduction Number, $R_C$

The basic reproduction number, denoted by $R_0$, was defined by Diekmann *et al.* (1990) and Anderson and May (1991), as the expected number of secondary cases produced in a

completely susceptible population, by a typical infective individual. However, the effective reproduction number, $R_C$ represents the number of secondary cases generated by an infected individual in a completely susceptible population where control strategies are used. The next generation matrix, as explained by Driessche and Watmough (2002), was used to compute the effective reproduction number.

$$F = \begin{pmatrix} 0 & 0 & 0 & \dfrac{\beta_1\left(S^0 + (1-\nu)V^0\right)}{N_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \dfrac{\beta_2 F_n{}^0}{N_v} & \dfrac{\beta_2 \eta F_n{}^0}{N_v} & 0 & 0 \end{pmatrix} \tag{23}$$

and

$$V = \begin{pmatrix} k_3 & 0 & 0 & 0 \\ -\sigma & k_4 & 0 & 0 \\ 0 & -\psi & k_5 & 0 \\ 0 & 0 & 0 & k_6 \end{pmatrix} \tag{32}$$

Applying elementary row operations on (33) we obtained $V^{-1}$ as,

$$V^{-1} = \begin{pmatrix} \dfrac{1}{k_3} & 0 & 0 & 0 \\ \dfrac{\sigma}{k_3 k_4} & \dfrac{1}{k_4} & 0 & 0 \\ \dfrac{\sigma\psi}{k_3 k_4 k_5} & \dfrac{\psi}{k_4 k_5} & \dfrac{1}{k_5} & 0 \\ 0 & 0 & 0 & \dfrac{1}{k_6} \end{pmatrix} \tag{34}$$

from (31) and (34) we have,

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \dfrac{\beta_1\left(S^0 + (1-\nu)V^0\right)}{k_6 N_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \dfrac{\beta_2 F_n{}^0}{k_3 N_v} + \dfrac{\beta_2 \eta \sigma F_n{}^0}{k_3 k_4 N_v} & \dfrac{\beta_2 \eta \sigma F_n{}^0}{k_4 N_v} & 0 & 0 \end{pmatrix} \tag{35}$$

To get the spectral radius $\rho\left(FV^{-1}\right)$, we evaluate $\left|FV^{-1} - \lambda I\right| = 0$,

$$R_c = \rho\left(FV^{-1}\right) = \left|FV^{-1} - \lambda I\right| = \begin{vmatrix} -\lambda & 0 & 0 & \dfrac{\beta_1\left(S^0 + (1-\nu)V^0\right)}{k_6 N_h} \\ 0 & -\lambda & 0 & 0 \\ 0 & 0 & -\lambda & 0 \\ \dfrac{\beta_2 F_n^{\,0}}{k_3 N_v} + \dfrac{\beta_2 \eta\sigma F_n^{\,0}}{k_3 k_4 N_v} & \dfrac{\beta_2 \eta\sigma F_n^{\,0}}{k_4 N_v} & 0 & -\lambda \end{vmatrix} = 0 \qquad (36)$$

Which can be written as,

$$\left|FV^{-1} - \lambda I\right| = (-\lambda)(-\lambda)\left(\lambda^2 - \left[\frac{\beta_1\left(S^0 + (1-\nu)V^0\right)}{k_6 N_h}\right]\left[\frac{\beta_2 F_n^{\,0}}{k_3 N_v} + \frac{\beta_2 \eta\sigma F_n^{\,0}}{k_3 k_4 N_v}\right]\right) = 0 \qquad (37)$$

$\lambda = \mathbf{O}$, twice (38)

or

$$\lambda^2 = \left[\frac{\beta_1\left(S^0 + (1-\nu)V^0\right)}{k_6 N_h}\right]\left[\frac{\beta_2 F_n^{\,0}}{k_3 N_v} + \frac{\beta_2 \eta\sigma F_n^{\,0}}{k_3 k_4 N_v}\right] \qquad (39)$$

Therefore,

$$R_C = \rho\left(FV^{-1}\right) = \sqrt{\frac{\beta_1\left(S^0 + (1-\nu)V^0\right)}{k_6 N_h}\left(\frac{\beta_2 F_n^{\,0}}{k_3 N_v} + \frac{\beta_2 \eta\sigma F_n^{\,0}}{k_3 k_4 N_v}\right)} \qquad (40)$$

Substituting equation (30) in (40) and simplifying we have,

$$R_C = \rho\left(FV^{-1}\right) = \sqrt{\frac{\beta_1\beta_2\mu_h\left(k_4 + \eta\sigma\right)\left[k_2 + \rho_1(1-\nu)\right]}{k_1 k_2 k_3 k_4 k_6}} \qquad (41)$$

**Local Stability of Dengue Free Equilibrium State $\left(E^0\right)$**

**Theorem 1:** The disease-free equilibrium state of the model is Locally Asymptotically Stable (LAS) if $R_C < 1$, otherwise unstable.

**Proof:**

We calculate the eigenvalues $\lambda_i$, where $i = 1,2,3,4,5,6,7,8.$ of the Jacobian matrix of the model to determine the nature of the eigenvalues using the characteristic equation $\left|J\left(E^0\right) - \lambda I\right| = 0.$ To analyze the local stability for the disease free equilibrium for the model, we first obtain the Jacobian matrix of the model equations (1) – (8) at DFE.

$$J\left(E^{0}\right)=\begin{bmatrix} -k_1 & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_1\mu_h}{k_1} \\ \rho_1 & -k_2 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_1(1-\nu)\rho_1\mu_h}{k_1k_2} \\ 0 & 0 & -k_3 & 0 & 0 & 0 & 0 & \frac{\mu_h\beta_1\left(k_2+\rho_1(1-\nu)\right)}{k_1k_2} \\ 0 & 0 & \sigma & -k_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \psi & -k_5 & 0 & 0 & 0 \\ 0 & \rho_2 & \gamma & 0 & \tau & -\mu_h & 0 & 0 \\ 0 & 0 & -\beta_2 & -\beta_2\eta & 0 & 0 & -k_6 & 0 \\ 0 & 0 & \beta_2 & \beta_2\eta & 0 & 0 & 0 & -k_6 \end{bmatrix} \tag{43}$$

Computing the characteristic equation for the upper triangular Jacobian matrix (44) we have,

$$\left|J\left(E^{0}\right)-\lambda I\right|=\begin{vmatrix} -k_1-\lambda & 0 & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_1\mu_h}{k_1} \\ 0 & -k_2-\lambda & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_1\mu_h\rho_1\left[k_1(1-\nu)+k_2\right]}{k_1^{2}k_2} \\ 0 & 0 & -k_3-\lambda & 0 & 0 & 0 & 0 & \frac{\mu_h\beta_1\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2} \\ 0 & 0 & 0 & -k_4-\lambda & 0 & 0 & 0 & \frac{\beta_1\mu_h\sigma\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2k_3} \\ 0 & 0 & 0 & 0 & -k_5-\lambda & 0 & 0 & \frac{\beta_1\mu_h\sigma\psi\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2k_3k_4} \\ 0 & 0 & 0 & 0 & 0 & -\mu_h-\lambda & 0 & -A_1+\frac{\gamma}{k_3}A_2+\frac{\tau}{k_5}A_3 \\ 0 & 0 & 0 & 0 & 0 & 0 & -k_6-\lambda & -\left(\frac{\beta_2}{k_3}\right)A_2-\left(\frac{\beta_2\eta}{k_4}\right)A_4 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & -k_6+\frac{\beta_2}{k_3}A_2+\frac{\beta_2\eta}{k_4}A_4-\lambda \end{vmatrix}=0 \tag{46}$$

this gives,

$$\left.\begin{aligned} &\lambda_1=-k_1,\quad \lambda_2=-k_2,\quad \lambda_3=-k_3,\quad \lambda_4=-k_4, \\ &\lambda_5=-k_5,\quad \lambda_6=-k_6,\quad \lambda_7=-\mu_h,\quad \lambda_8=-k_6+\frac{\beta_2}{k_3}A_2+\frac{\beta_2\eta}{k_4}A_4 \end{aligned}\right\} \tag{47}$$

where,

$$\left.\begin{aligned} A_1 &= \frac{\beta_1\mu_h\rho_1\rho_2\left[k_1(1-\nu)+k_2\right]}{{k_1}^2{k_2}^2}, \quad A_2 = \frac{\mu_h\beta_1\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2} \\ A_3 &= \frac{\beta_1\mu_h\sigma\psi\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2k_3k_4}, \quad A_4 = \frac{\beta_1\mu_h\sigma\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2k_3} \end{aligned}\right\} \tag{45}$$

The DFE will be locally asymptotically stable if $\lambda_8 < 0$.

$$-k_6 + \frac{\beta_2}{k_3}A_2 + \frac{\beta_2\eta}{k_4}A_4 < 0 \tag{48}$$

Substituting equations (45) in (48) we have,

$$\frac{\beta_2}{k_3}\left(\frac{\mu_h\beta_1\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2}\right)+\frac{\beta_2\eta}{k_4}\left(\frac{\beta_1\mu_h\sigma\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2k_3}\right) < k_6 \tag{49}$$

Simplifying () gives,

$$\frac{\beta_1\beta_2\mu_h\left(k_4+\eta\sigma\right)\left[k_2+\rho_1(1-\nu)\right]}{k_1k_2k_3k_4k_6} < 1 \tag{51}$$

that is,

$$R_C < 1 \tag{52}$$

Thus, the disease-free equilibrium state of the model is Locally Asymptotically Stable (LAS).

**Global Stability of Disease-Free Equilibrium** $\left(E^0\right)$

Solutions approaches the equilibrium for all initial conditions in global asymptotic stability. This removes the restrictions on the initial conditions of the model variables. In this research work, the Lyapunov theorem is used to establish the global stability of the disease-free equilibrium of the model.

**Theorem 2:** The dengue free equilibrium, $E^0$ of (3) – (12) is globally asymptotically stable (GAS) in $\Omega$ if $R_C \le 1$.

**Proof:** Consider the Lyapunov function:

$$f\left(M,W,F_d\right) = \left(\eta\sigma + k_4\right){N_v}^0M + \eta k_3{N_v}^0W + {N_h}^0F_d \tag{53}$$

its derivatives along the solutions of the model equations is,

$$f'\left(M,W,F_d\right) = \left(\eta\sigma + k_4\right){N_v}^0M' + \eta k_3{N_v}^0W' + {N_h}^0F_d' \tag{54}$$

Substituting (6) and (7) into (54) and simplifying we have,

$$f' = \left\{ \begin{array}{l} \left[ \dfrac{\beta_1 \left[ S + (1-\nu) V \right] F_d \left( \eta\sigma + k_4 \right) N_v^{\,0}}{N_h} + \dfrac{\beta_2 \left( M + \eta W \right) F_n N_h^{\,0}}{N_v} \right] \\ \quad - \left[ k_6 F_d N_h^{\,0} + k_3 k_4 \left( M + \eta W \right) N_v^{\,0} \right] \end{array} \right\} \tag{55}$$

Since

$$\left( a + b + c \right) \le \left( abc \right) \qquad \forall\, a, b, c \ge 1 \tag{56}$$

We have,

$$f' \le \left\{ \begin{array}{l} \left[ \dfrac{\beta_1 \left[ S + (1-\nu) V \right] F_d \left( \eta\sigma + k_4 \right) N_v^{\,0}}{N_h} \times \dfrac{\beta_2 \left( M + \eta W \right) F_n N_h^{\,0}}{N_v} \right] \\ \quad - \left[ k_6 F_d N_h^{\,0} \times k_3 k_4 \left( M + \eta W \right) N_v^{\,0} \right] \end{array} \right\} \tag{57}$$

that is,

$$f' \le k_3 k_4 k_6 N_h^{\,0} N_v^{\,0} \left( M + \eta W \right) F_d \left\{ \frac{\beta_1 \beta_2 \left( \eta\sigma + k_4 \right) \left[ S + (1-\nu) V \right] F_n}{k_3 k_4 k_6 N_h N_v} - 1 \right\} \tag{58}$$

Also, since $\left( S, V, F_n, N_h, N_v \right) \le \left( S^0, V^0, F_n^{\,0}, N_h^{\,0}, N_v^{\,0} \right)$, we have

$$f' \le k_3 k_4 k_6 N_h^{\,0} N_v^{\,0} \left( M + \eta W \right) F_d \left\{ \frac{\beta_1 \beta_2 \left( \eta\sigma + k_4 \right) \left[ S^0 + (1-\nu) V^0 \right] F_n^{\,0}}{k_3 k_4 k_6 N_h^{\,0} N_v^{\,0}} - 1 \right\} \tag{59}$$

Thus, substituting the DFE points and simplifying we have,

$$f' \le k_3 k_4 k_6 N_h^{\,0} N_v^{\,0} \left( M + \eta W \right) F_d \left\{ \frac{\beta_1 \beta_2 \mu_h \left( k_4 + \eta\sigma \right) \left[ k_2 + \rho_1 (1-\nu) \right]}{k_1 k_2 k_3 k_4 k_6} - 1 \right\} \tag{60}$$

Hence,

$$f' \le k_3 k_4 k_6 N_h^{\,0} N_v^{\,0} \left( M + \eta W \right) F_d \left\{ R_C - 1 \right\} \tag{61}$$

Since all model parameters are non-negative, it follows that when $R_C \le 1$, $f' \le 0$; the equality $f' = 0$ holds when $R_C = 1$ or $\left( M + \eta W \right) F_d = 0$. Therefore, the largest compact invariant set in $\left\{ \left( S, V, M, W, H, R \right) \in \Omega : f' = 0 \right\}$ is the singleton $\left\{ E^0 \right\}$. Hence, by the LaSalle invariance principle (Lasalle, 1976), $E^0$ is overall globally asymptotically stable in $\Omega$ and hence, the proof.

**RESULTS AND DISCUSSION**

Equation (21) shows the disease-free equilibrium point, equation (41) shows the effective reproduction number which is the basis for the stability analysis. Equation (51) have shown

the proof of the local stability for the DFE of the model while equation (61) shows the proof of global stability for the DFE. A graphical representation of the model is given in figure 2 proving the analytical solution for the global stability.

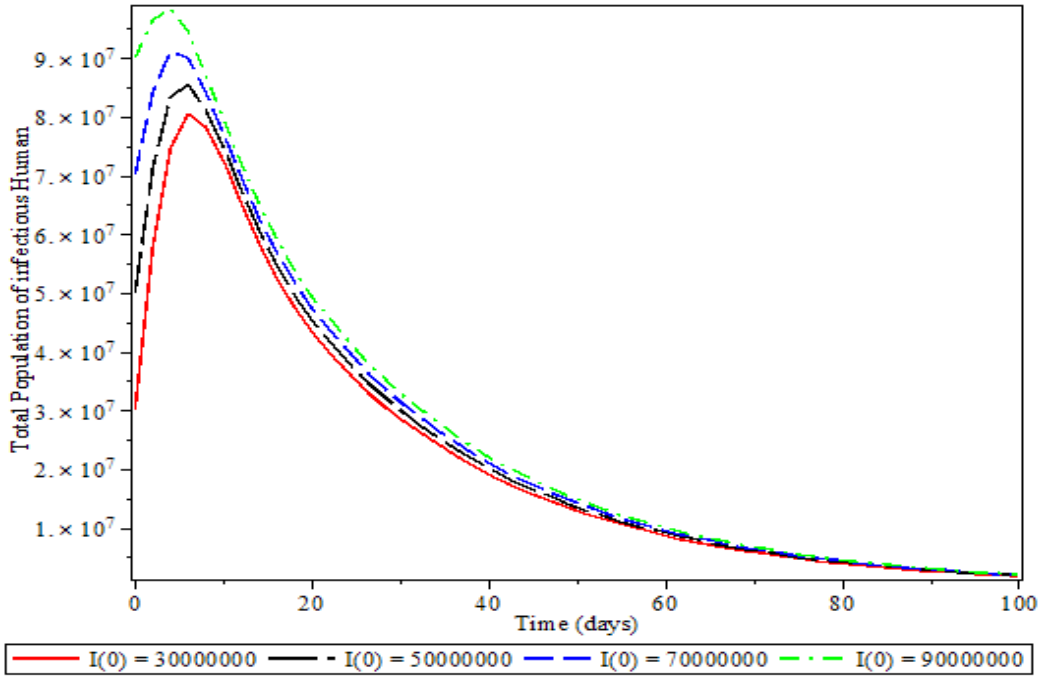



**Figure 2: Different Initial Conditions for Total Number of Infectious Human**

Figure 2 above shows the global asymptotic stability of the disease-free equilibrium condition with $\psi = \rho_1 = \rho_2 = 0.03$, $\delta_v = 0.5$ and $\tau = 0.05$. It can be observed that, irrespective of the initial condition of the infectious population, they converge at disease free equilibrium. This confirms the analytical result of the global asymptotic stability of the model with $R_C = 0.1865$.

## CONCLUSION

It can be observed that the both local and global stability of the DFE are asymptotically stable if $R_C < 1$ and $R_C \leq 1$ respectively. Based on the results established, governments and health agencies are advised to intensify efforts on providing adequate control measure so they can bring the effective reproduction number down.

## RECOMMENDATIONS

Further research may be conducted on the local and global stability of endemic equilibrium, sensitivity analysis and optimal control of the model.

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