

# A study of Dengue Disease Model with Vaccination Strategy

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**Abstract**— In this paper, we proposed and analyzed the effects of vaccination strategy on the transmission of the Dengue diseases. We propose SIR model with logistic recruitment rate, and analyzed the Steady state and stability of the equilibrium points. If  $R_0^* < 1$  then the non- infected steady state  $P_1^*$  will be stable. Also if  $R_0^* > 1$  then the endemic equilibrium  $P_2^*$  is stable. Numerical simulations show that the effect of newborn vaccination is significantly less effective than vaccinating susceptible population. Also the effect of vaccination is to replace multiple outbreaks with a single outbreak.

**Keywords**— Dengue disease model, Vaccination strategy, Stability analysis, Numerical analysis.

**Mathematics Subject Classification** - 93D20, 92D30, 65L07.

## 1. Introduction

Dengue is the most important human viral disease transmitted by arthropod vectors. Annually there are an estimated 50–100 million cases of dengue fever (DF), and 250 000 to 500 000 cases of dengue haemorrhagic fever (DHF) in the world. Dengue infection is classified into three categories: Dengue fever (DF), Dengue hemorrhagic fever (DHF) and Dengue shock syndrome (DSS). DF, DHF and DSS are caused by the four dengue viruses DEN 1, 2, 3, and 4. Infection in humans with one serotype provides life-long immunity to that virus but not to the others. Dengue viruses are maintained in an urban transmission cycle in tropical and subtropical areas by the mosquito *Aedes aegypti*, a species closely associated with human habitation. In some regions other *Aedes* species, such as *Ae albopictus* and *Ae polynesiensis* are also involved.

There are some epidemiological and demographical factors that contribute to the transmission of the disease. From a practical point of view, many countries organized vaccination programs to control the spread of the disease. The effects of vaccination on the transmission of infectious diseases are studied by some researchers [2, 3, 4]. These researchers studied the direct transmitted disease. In our second model we studied the effect of vaccination on an indirect transmitted disease. Recently many researchers [4, 5, 6, 7] studied the vaccination strategy for all type of dengue viruses but it is not perfect. The mathematical models for Dengue Fever found in literature [2, 7, 8] are based on compartmental dynamics. We have also used the compartmental dynamics.

## 2. Formulation of the model

In this paper we consider the effects of vaccination strategy on the transmission of the Dengue diseases. The model assumes that the host population grows logistically, and has a constant disease death rate. We also assume that the vector population has constant size with birth and death rate equal to  $\mu_V$ . The host population is subdivided into the susceptible  $S_H$ , infective  $I_H$ , and recovered  $R_H$  classes. The Vector population, due to a short life period, is subdivided into the susceptible  $S_V$ , infective  $I_V$ . The transmission model for the dengue disease is as follows:

$$\begin{aligned} \frac{dS_H}{dt} &= \mu_H N_H \left( 1 - \frac{N_H}{K_H} \right) - \lambda_H S_H \frac{I_V}{N_V} - \mu_H S_H \\ \frac{dI_H}{dt} &= \lambda_H S_H \frac{I_V}{N_V} - (\gamma + \mu_H + \alpha_H) I_H \end{aligned} \quad (2.1)$$

$$\frac{dR_H}{dt} = \gamma I_H - \mu_H R_H$$

For Vector population:

$$\begin{aligned} \frac{dS_V}{dt} &= \mu_V V - \lambda_V S_V \frac{I_H}{N_H} - \mu_V S_V \\ \frac{dI_V}{dt} &= \lambda_V S_V \frac{I_H}{N_H} - \mu_V I_V \end{aligned} \quad (2.2)$$

The initial conditions

$$N_H = S_H + I_H + R_H \text{ and } S_V + I_V = N_V$$

$$\frac{dN_H}{dt} = \mu_H N_H \left(1 - \frac{N_H}{K_H}\right) - \mu_H N_H - \alpha_H I_H \quad (2.3)$$

It is convenient to reformulate the model (6.2.4) in terms of population proportion  $x = \frac{S_H}{N_H}$ ,  $y = \frac{I_H}{N_H}$  and  $z = \frac{I_V}{N_V}$ , which are the fractions of the susceptible, infectives and removals, respectively.

Hence the system can be written as

$$\begin{aligned} \frac{dx}{dt} &= \mu_H \left(1 - \frac{N_H}{K_H}\right) - \lambda_H xz - \mu_H x \\ \frac{dy}{dt} &= \lambda_H xz - My \\ \frac{dz}{dt} &= \lambda_V (1-z)y - \mu_V z \end{aligned} \quad (2.4)$$

where  $M = \gamma + \mu_H + \alpha_H$

We consider two types of vaccination, one that is being administered to a portion of new born host and another one is being administered to a portion of susceptible host. The main question here is whether it is enough to vaccinate only new born host in order to control the spread of the disease or it is necessary to vaccinate the larger susceptible host [4].

Let a portion  $\rho$ ,  $0 \leq \rho \leq 1$ , of newborn host be vaccinated. Assume that the vaccine is not perfect and assume that the effectiveness of the vaccine is  $s$ , and then  $(1 - \rho s)\mu_H \left(1 - \frac{N_H}{K_H}\right)$  newborns remain susceptible, and  $\rho s \mu_H \left(1 - \frac{N_H}{K_H}\right)$  directly being removed to  $R_H$ . The corresponding dynamic equation for  $x$  is given by

$$\frac{dx}{dt} = \mu_H(1 - \rho s) \left( 1 - \frac{N_H}{K_H} \right) - \lambda_H xz - \mu_H x \quad (2.5)$$

and the other two equations in (2.4) remain the same. On the other hand, let a portion  $\sigma$ ,  $0 \leq \sigma \leq 1$  of susceptible host be vaccinated. Then the dynamical equation of  $x$  and  $y$  is as follows:

$$\begin{aligned} \frac{dx}{dt} &= \mu_H \left( 1 - \frac{N_H}{K_H} \right) - \lambda_H xz(1 - \sigma s) - \mu_H x \\ \frac{dy}{dt} &= \lambda_H xz(1 - \sigma s) - My \end{aligned} \quad (2.6)$$

These two cases (2.5) and (2.6) are written on one system as follows:

$$\begin{aligned} \frac{dx}{dt} &= \mu_H(1 - \rho s) \left( 1 - \frac{N_H}{K_H} \right) - \lambda_H(1 - \sigma s)xz - \mu_H x \\ \frac{dy}{dt} &= \lambda_H xz(1 - \sigma s) - My \\ \frac{dz}{dt} &= \lambda_v(1 - z)y - \mu_v z \end{aligned} \quad (2.7)$$

In succession by putting  $\sigma = 0$  or  $\rho = 0$ , these two equations (2.6) and (2.7) can be generated. Rescale  $t$  by  $\lambda_v$ , the system of equations (2.7) simplified to

$$\begin{aligned} \frac{dx}{dt} &= b(1 - r)\theta - \alpha xz - bx \\ \frac{dy}{dt} &= \alpha xz - \beta y \\ \frac{dz}{dt} &= (1 - z)y - \delta z \end{aligned} \quad (2.8)$$

where  $\delta = \frac{\mu_v}{\lambda_v}$ ,  $\alpha = \frac{\lambda_H(1 - \sigma s)}{\lambda_v}$ ,  $\beta = \frac{M}{\lambda_v}$ ,  $\theta = \left( 1 - \frac{N_H}{K_H} \right)$ ,  $r = \rho s$ ,  $b = \frac{\mu_H}{\lambda_v}$

### 3. Steady state and Stability Analysis

Equilibrium points are obtained by setting time derivatives of  $x$ ,  $y$  and  $z$  equal to zero then the system(2.8) has two possible equilibria, i.e., the non-endemic equilibrium  $P_1^* \left( (1 - r)\theta, 0, 0 \right)$  and the endemic equilibrium  $P_2^* \left( x^{**}, y^{**}, z^{**} \right)$ , where

$$x^{**} = \frac{b\theta(1-r) + \beta\delta}{\alpha + b} = \frac{\mu_H \lambda_V (1-\rho s) \left(1 - \frac{N_H}{K_H}\right) + M \mu_V}{\lambda_V [\mu_H + \lambda_H (1-\sigma s)]}$$

$$y^{**} = \frac{b[\alpha\theta(1-r) - \beta\delta]}{\beta(\alpha + b)} = \frac{\mu_H \left[ \lambda_H \lambda_V (1-\sigma s)(1-\rho s) \left(1 - \frac{N_H}{K_H}\right) - M \mu_V \right]}{M \lambda_V [\mu_H + \lambda_H (1-\sigma s)]}$$

$$z^{**} = \frac{b[\alpha\theta(1-r) - \beta\delta]}{\alpha[b\theta(1-r) + \beta\delta]} = \frac{\mu_H \left[ \lambda_H \lambda_V (1-\sigma s)(1-\rho s) \left(1 - \frac{N_H}{K_H}\right) - M \mu_V \right]}{\lambda_H (1-\sigma s) \left[ \mu_H \lambda_V (1-\rho s) \left(1 - \frac{N_H}{K_H}\right) + M \mu_V \right]}$$

It is clear that the non-endemic equilibrium point  $P_1^* ((1-r)\theta, 0, 0)$  will be stable if  $R_0^* < 1$ .

At the endemic equilibrium point  $P_2^* (x^{**}, y^{**}, z^{**})$  the variation matrix becomes

$$Z_2 = \begin{bmatrix} -\alpha z^{**} - b & 0 & -\alpha x^{**} \\ \alpha z^{**} & -\beta & \alpha x^{**} \\ 0 & (1-z^{**}) & -y^{**} - \delta \end{bmatrix}$$

its characteristics equation

$$\lambda^3 + A\lambda^2 + B\lambda + C = 0$$

$$\text{where } A = \beta + \frac{b\theta(1-r)(\alpha + b)}{[b\theta(1-r) + \beta\delta]} + \frac{\alpha[b\theta(1-r) + \beta\delta]}{\beta(\alpha + b)}$$

$$B = \frac{b\beta\theta(1-r)(\alpha + b)}{[b\theta(1-r) + \beta\delta]} + \frac{\alpha b\theta(1-r)}{\beta} + \frac{b[\alpha\theta(1-r) - \delta\beta]}{(\alpha + b)}$$

$$C = b[\alpha\theta(1-r) - \delta\beta]$$

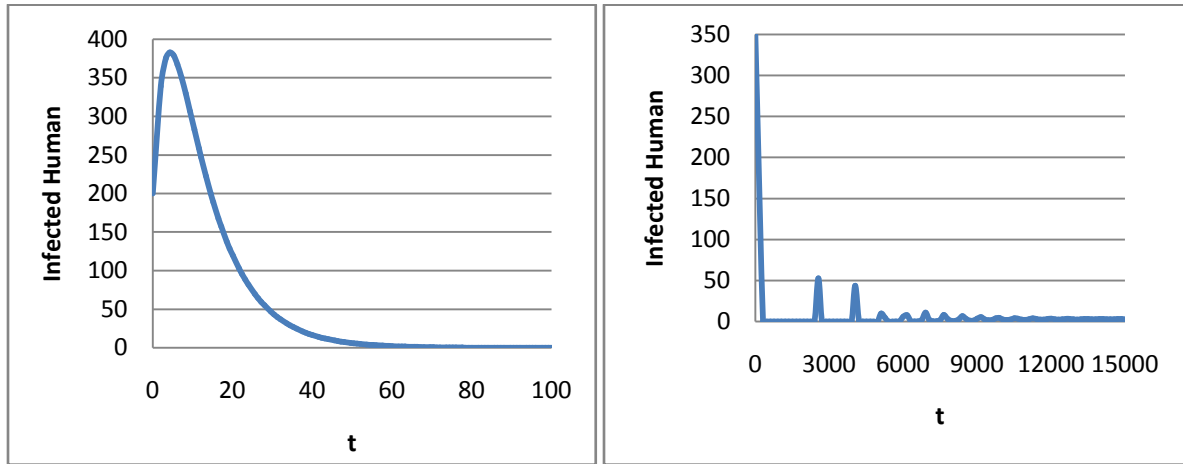
It is clear that all the coefficient of the characteristics polynomial are positive. Direct calculation shows that  $A > 0$ ,  $C > 0$  and  $AB > C$

by Routh-Hurwitz criteria. Hence the endemic equilibrium will be stable if  $R_0^* > 1$  where  $R_0^* = \frac{\lambda_H \lambda_V (1-\sigma s)(1-\rho s) \left(1 - \frac{N_H}{K_H}\right)}{M \mu_V}$

#### 4. Results and Discussions.

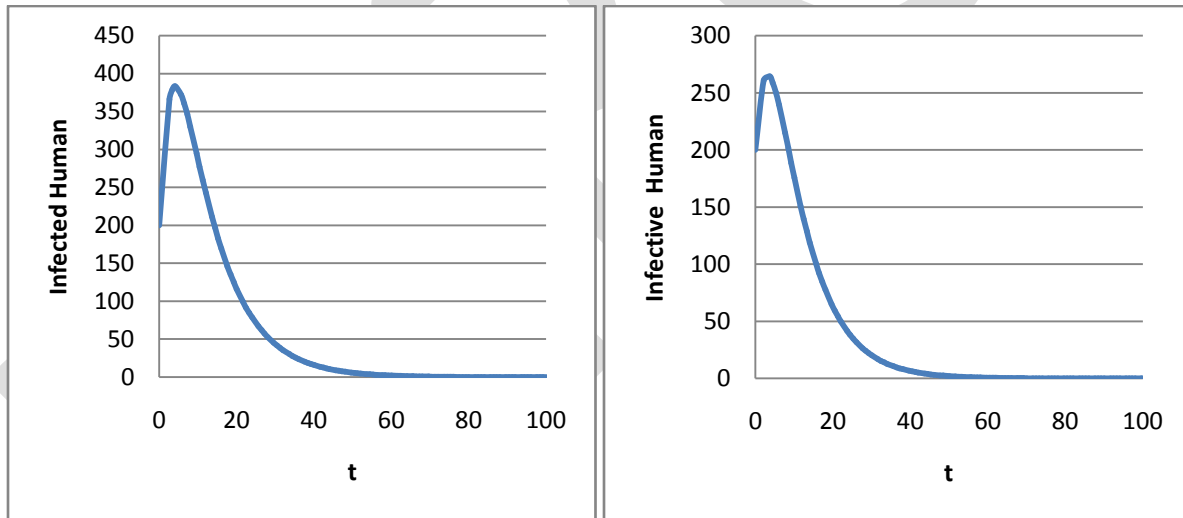
The system of equation (2.8) was solved numerically using mathematical software. The values of the parameters are taken from [10]. After simulation, we observed that the dynamic of infected human and infected vector are not affected by newborn vaccination. In Figure 1, (a) and (b) show the proportion of the infective human population without vaccination. We see that first outbreak occurs at approximately  $t=6$ . Second outbreak begin from approximately  $t=2400$ . If the newborn vaccination is applied, Figure 1(c) shows that still one outbreak occurs followed by exponential decay. However, if the susceptible vaccination is applied, Figure 1(d) shows that there is almost no outbreak and the numbers of subsequent cases exponentially decay.

Fig. 2 presents the dynamics of  $S_H$ ,  $I_H$  and  $I_V$  in 100 days after one infected entered the population. We found that  $S_H$  drops significantly in a relatively small period of time. Infected human  $I_H$  and infected vector  $I_V$  increases significantly during the period of 12 days and then oscillate around the endemic equilibrium state.



(a)

(b)



(c)

(d)

Fig. 1: Dynamics of  $I_H$  population without vaccination and the  $I_H$  population with newborn vaccination and susceptible vaccination.

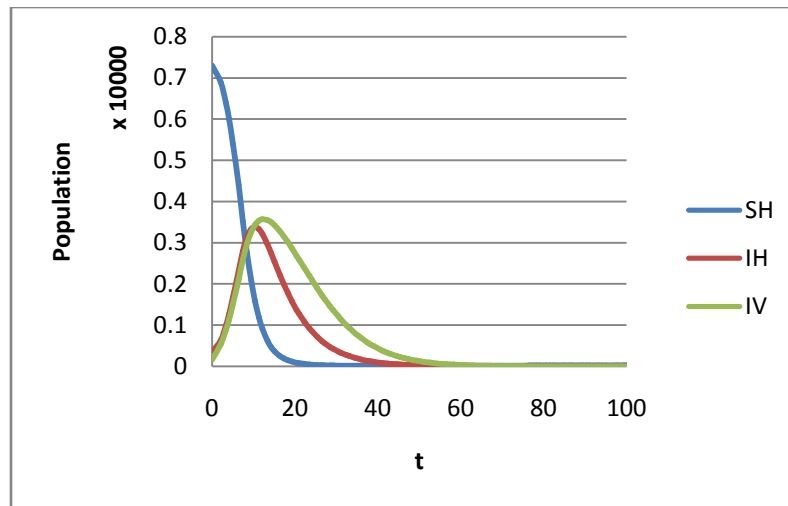


Fig. 2: Dynamics of  $S_H$ ,  $I_H$  and  $I_V$  in 100 days with the initial condition  $(0.73, 0.035, 0.025)$  for

$$\mu_V=0.25, \mu_H=1/(60 \times 365), \gamma=0.1428, \lambda_H=0.75, \lambda_V=1, \alpha_H=0, K_H=4 \times 10^5,$$

$$N_H=10000, N_V=6400. \text{ With these parameters, } R_0=11.51 > 1.$$

Fig. 3 shows simulations with different proportions of the susceptible vaccination in endemic equilibrium state for  $\sigma=0, 0.25, 0.50, 0.75, 1$ . We observed that vaccination decreases the number of infectives. That is, the dose of susceptible vaccination is increases then the number of infectives with respect to time is exponential decreases.

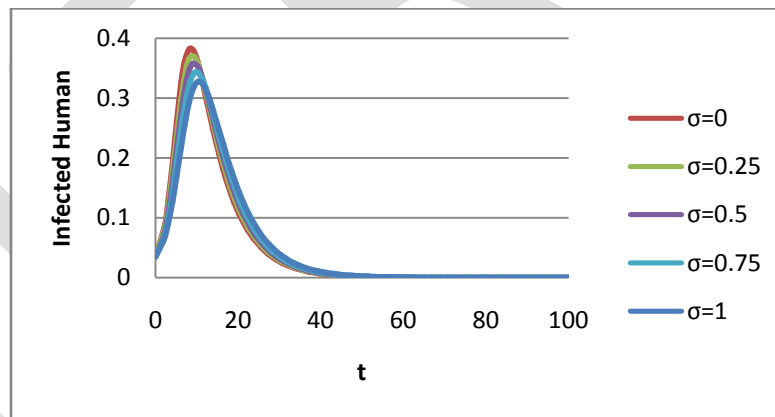


Fig. 3: Dynamics of infected Human with different proportions of the susceptible

Vaccination in endemic equilibrium state for  $\sigma=0, 0.25, 0.50, 0.75, 1$ .

## 5. Conclusion.

In this paper we consider two types of vaccination, one that is being administered to a portion of new born host and another one is being administered to a portion of susceptible host. If  $R_0^* < 1$  then the non- infected steady state  $P_1^*$  will be stable and if  $R_0^* > 1$  then the endemic equilibrium  $P_2^*$  is stable. Numerical simulations show that the effect of newborn vaccination is significantly less effective than vaccinating susceptible population. Also the effect of vaccination is to replace multiple outbreaks with a single

outbreak. However, if we apply the susceptible vaccination there is almost no outbreak occurs and the cases exponentially decay approaching the disease-free equilibrium (Figure 1(d)).

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