

# Stability Analysis of Disease Free Equilibrium (DFE) State of a Mathematical Model of Yellow Fever Incorporating Secondary Host

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## ABSTRACT

In this paper we formulate a mathematical model of yellow fever incorporating secondary host. We obtained the Disease Free Equilibrium (DFE) Points and compute the basic reproduction number. The local and global stability of the DFE was analyzed using Jacobian Matrix stability techniques and Lyapunov function respectively. The local and global stability was asymptotically stable if  $R_0 < 1$  and  $R_0 \leq 1$ , respectively. The basic reproduction number and control parameters of the model were presented graphically.

(Keywords: basic reproduction number, disease free equilibrium, yellow fever, secondary host, stability)

## INTRODUCTION

Yellow fever, known historically as yellow jack, or yellow plague is an acute viral disease. In most cases symptoms include fever, chills, loss of appetite, nausea, muscle pains particularly in the back, and headaches. The disease is caused by the yellow fever virus and is spread by the bite of the female mosquito. It only infects humans, other primates and several species of mosquito, (Oldstone, 2009). In cities it is primarily spread by mosquitoes of the *Aedes aegypti* species. The virus is an RNA virus of the genus *Flavivirus*, (Bazin, 2011).

Yellow fever virus is mainly transmitted through the bite of the yellow fever mosquito *Aedes aegypti*, but other mosquitoes such as the tiger mosquito (*Aedes albopictus*) can also serve as a vector for this virus, (Fontenille, *et al.*, 1997).

Vector-borne diseases (e.g. malaria, dengue, fever, yellow fever, lyme disease, trypanosomiasis, and leishmania), amongst all the human infectious diseases, continue to remain a public health concern and a severe burden on economies, causing high human mortality in the world. These diseases have not only posed problems to national economies, but have also caused poverty and low living standards, especially in countries in the tropical and subtropical regions of the worlds, (Nouridine, *et al.*, 2011).

According to Monath, (1989), Van der, (1999), Figueiredo, (2000), Souza, (2010) and Auguste, (2010), Yellow fever virus (YFV) is the prototype species for the genus *Flavivirus*. Historically, YFV is one of the most important human arboviral pathogens. It continues to cause large sporadic epidemics in Africa but typically emerges as epizootics among nonhuman primates in South America with or without associated human cases. YFV emergence is cyclical; outbreaks occur  $\approx 7-10$  years apart. Several phylogenetic studies have shown that YFV is locally maintained during these interepizootic periods in Peru, Bryant, (2003), Brazil, Vasconcelos, (2004), and Auguste, (2010).

Yellow fever virus undergoes regionally independent evolution within some countries, (Bryant, 2003). The sporadic emergence of YFV in the Americas has been strongly associated with infection of red howler monkeys *Alouatta seniculus*, which are particularly susceptible to disease, (Auguste, 2015). There are three types of transmission cycle: sylvatic (or jungle) yellow fever, the intermediate cycle of yellow fever transmission and urban yellow fever. Because of this sylvatic cycle, the yellow fever cannot be eradicated, (Barrett, 2007).

Akinwande, (1996), formulated a model of yellow fever epidemics, which involves the interactions of two principal communities of hosts (humans) and Vectors (*Aedes aegypti* mosquitoes). Hui-Ming, (2008), considered an epidemic model of a vector-borne disease which has direct mode of transmission in addition to the vector-mediated transmission.

Kung'aro, (2015), described the transmission dynamics of yellow fever (YF) within two host populations, and build up a deterministic SVEIRS model with vaccination to the entire new born. They formulated a model for the spread of Yellow fever in humans, vector and primates populations. The human population was divided into five (5) classes, the vector population was divided into three classes and the primate population was divided into three classes. Fernandez, (2013), formulated a model and incorporated the biology of the urban vector of yellow fever, the mosquito *Aedes aegypti*, the stages of the disease in the host (humans). Raimundo, (2015), formulate a mathematical model to address the transmission dynamics of an infectious agent in a homogeneous population in the presence of an imperfect vaccine. The equations include the human and the vector and their eggs-population. The egg-population includes the intermediate stages, such as larvae and pupae.

In this paper, we formulated a mathematical model of yellow fever transmission incorporating secondary host, we assumed in our model that the vaccinated susceptible humans will move to recovered class. We compute the Basic Reproduction Number,  $R_0$  using next generation matrix. We also analyzed the local and global stability of DFE using Jacobian Matrix techniques and Lyapunov function.

## MATERIAL AND METHOD

### Model Formulation

The model equations are formulated using first order ordinary differential equation.

Three populations of human, vector (mosquito) and secondary host (monkey) populations were considered. The human population is divided into three compartments of susceptible,  $S_h$ , infected,  $I_h$ , and recovery,  $R_h$ .

The vector population is divided into two compartments of Non-carrier vector,  $V_1$  and carrier vector,  $V_2$ . The monkey population was also divided into two compartments of susceptible,  $S_m$  and infectious,  $I_m$ .

We consider the total population sizes denoted by  $N_h(t)$ ,  $N_v(t)$  and  $N_m(t)$  for the humans, mosquitoes (*Aedes aegypti*) and monkeys respectively.

The human, vector and monkey populations are increased by a recruitment term;  $\Lambda_h$ ,  $\Lambda_v$  and  $\Lambda_m$  respectively.

Susceptible humans and susceptible monkeys are infected with the virus through the bite of a mosquitoes with the rates  $\alpha_1$  and  $\alpha_4$  respectively.

Non- carrier vector become infectious when it bites the infected humans and infected monkeys with the rates  $\alpha_2$  and  $\alpha_3$ , respectively. The human population is reduced through the natural death rate  $\mu_h$  and disease induced rate  $\delta_h$ . The vaccinate susceptible and recovered infected humans moved to recovered/immune class with the vaccination rate  $\nu$  and recovery rate  $\gamma_h$ .

The vector population is reduced by natural death rate  $\mu_v$  and death rate due to insecticide  $\delta_v$ . The monkey population is also reduced through the natural death rate  $\mu_m$  and disease induced rate  $\delta_m$ .

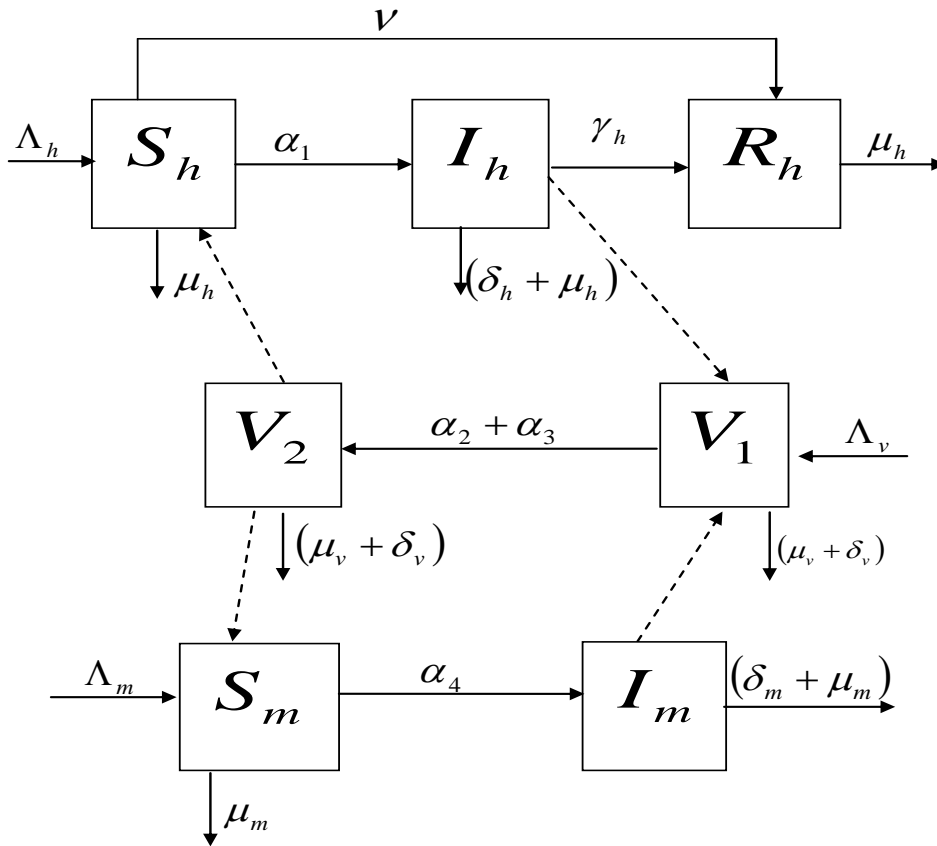


Figure 1: Model Flowchart.

$$\frac{dS_h}{dt} = \Lambda_h - \frac{\alpha_1 S_h V_2}{N_h} - (\nu + \mu_h) S_h \quad (1)$$

$$\frac{dI_h}{dt} = \frac{\alpha_1 S_h V_2}{N_h} - (\gamma_h + \mu_h + \delta_h) I_h \quad (2)$$

$$\frac{dR_h}{dt} = \nu S_h + \gamma_h I_h - \mu_h R_h \quad (3)$$

$$\frac{dV_1}{dt} = \Lambda_v - \frac{\alpha_2 V_1 I_h}{N_h} - \frac{\alpha_3 V_1 I_m}{N_m} - (\mu_v + \delta_v) V_1 \quad (4)$$

$$\frac{dV_2}{dt} = \frac{\alpha_2 V_1 I_h}{N_h} + \frac{\alpha_3 V_1 I_m}{N_m} - (\mu_v + \delta_v) V_2 \quad (5)$$

$$\frac{dS_m}{dt} = \Lambda_m - \frac{\alpha_4 S_m V_2}{N_m} - \mu_m S_m \quad (6)$$

$$\frac{dI_m}{dt} = \frac{\alpha_4 S_m V_2}{N_m} - (\mu_m + \delta_m) I_m \quad (7)$$

Where,

$$\left. \begin{aligned} N_h &= S_h + I_h + R_h \\ N_v &= V_1 + V_2 \\ N_m &= S_m + I_m \end{aligned} \right\} \quad (8)$$

**Table 1:** Variables of the Model.

Symbols	Description
$S_h(t)$	Number of susceptible humans at time $t$
$I_h(t)$	Number of infectious humans at time $t$
$R_h(t)$	Number of recovered humans at time $t$
$V_1(t)$	Number of non-carrier vectors at time $t$
$V_2(t)$	Number of carrier vectors at time $t$
$S_m(t)$	Number of susceptible secondary host at time $t$
$I_m(t)$	Number of infectious secondary host at time $t$
$N_h(t)$	Total human population at time $t$
$N_v(t)$	Total vector population at time $t$
$N_m(t)$	Total secondary vector population at time $t$

**Table 2:** Parameters of the Model.

Symbols	Description
$\alpha_1$	Effective virus Transmission rate from mosquito to humans
$\alpha_2$	Effective virus Transmission rate from humans to mosquito
$\alpha_3$	Effective virus Transmission rate from secondary host to mosquito
$\alpha_4$	Effective virus Transmission rate from mosquito to secondary host
$\Lambda_h$	Recruitment rate of human population
$\Lambda_v$	Recruitment rate of mosquito population
$\Lambda_m$	Recruitment rate of secondary vector population
$\delta_h$	Disease-induced death rate of humans
$\delta_v$	Death rate of mosquito due to application of insecticide
$\delta_m$	Disease-induced death rate of secondary host
$\mu_h$	Natural death rate of human population
$\mu_v$	Natural death rate of mosquito population
$\mu_m$	Natural death rate of secondary host population
$\gamma_h$	Recovery rate of human population due to drug administration
$\nu$	Immunization rate for the human population

### Existence of Equilibrium Points of the Model

At equilibrium:

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dR_h}{dt} = \frac{dV_1}{dt} = \frac{dV_2}{dt} = \frac{dS_m}{dt} = \frac{dI_m}{dt} = 0 \quad (9)$$

Let,

$$(S_h, I_h, R_h, V_1, V_2, S_m, I_m) = (S_h^*, I_h^*, R_h^*, V_1^*, V_2^*, S_m^*, I_m^*) \quad (10)$$

be the arbitrary equilibrium point.

Substituting (10) into (1) to (7) and solving simultaneously gives:

$$\left[ \begin{aligned} &\alpha_1 \alpha_2 A_4 N_m^{*2} S_h^* V_1^* + \alpha_3 \alpha_4 A_2 N_h^{*2} S_m^* V_1^* \\ &- A_2 A_3 A_4 N_h^{*2} N_m^{*2} \end{aligned} \right] V_2^* = 0 \quad (11)$$

Where,

$$\begin{aligned} A_1 &= (\nu + \mu_h), \quad A_2 = (\gamma_h + \mu_h + \delta_h), \\ A_3 &= (\mu_v + \delta_v) \text{ and } A_4 = (\mu_m + \delta_m) \end{aligned} \quad (12)$$

From (11):

$$V_2^* = 0 \quad (13)$$

Or

$$\begin{aligned} &\alpha_1 \alpha_2 A_4 N_m^{*2} S_h^* V_1^* + \alpha_3 \alpha_4 A_2 N_h^{*2} S_m^* V_1^* \\ &- A_2 A_3 A_4 N_h^{*2} N_m^{*2} = 0 \end{aligned} \quad (14)$$

Thus, Equation (11) gives the existence of two different equilibria; one satisfying (13) and the other satisfying (14). Thus,

$$V_2^* = I_h^* = I_m^* = 0 \quad (15)$$

### Disease Free Equilibrium (DFE) Points

Let,

$$E^0 = (S_h, I_h, R_h, V_1, V_2, S_m, I_m) = (S_h^0, I_h^0, R_h^0, V_1^0, V_2^0, S_m^0, I_m^0) \quad (16)$$

be the DFE point.

Substituting (16) and (15) into (1) to (7) gives the DFE points:

$$(S_h^0, I_h^0, R_h^0, V_1^0, V_2^0, S_m^0, I_m^0) = \left( \frac{\Lambda_h}{A_1}, 0, \frac{\Lambda_h V}{\mu_h A_1}, \frac{\Lambda_v}{A_3}, 0, \frac{\Lambda_m}{\mu_m}, 0 \right) \quad (17)$$

Equation (17) is the DFE points.

### Basic Reproduction Number, $R_0$

Applying next generation matrix operator to compute the Basic Reproduction Number of the model as used by (Diekmann *et al.*,1990) and improved by Driessche, (2002). The basic reproduction number is the largest eigenvalue or spectral radius of  $FV^{-1}$ :

$$FV^{-1} = \left[ \frac{\partial F_i(E^0)}{\partial x_i} \right] \left[ \frac{\partial V_i(E^0)}{\partial x_i} \right]^{-1} \quad (18)$$

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\alpha_1 \mu_h}{A_1 A_3} & 0 \\ \frac{\alpha_2 A_5 \mu_h}{A_2 A_3} & 0 & \frac{\alpha_3 A_6 \mu_m}{A_3 A_4} \\ 0 & \frac{\alpha_4}{A_4} & 0 \end{bmatrix} \quad (19)$$

$$R_0 = \sqrt{\frac{\alpha_1 \alpha_2 A_5 \mu_h^2}{A_1 A_2 A_3^2} + \frac{\alpha_3 \alpha_4 A_6 \mu_m}{A_3^2 A_4}} \quad (20)$$

$$\text{Where, } A_5 = \frac{\Lambda_v}{\Lambda_h} \text{ and } A_6 = \frac{\Lambda_v}{\Lambda_m}$$

Equation (20) is the Basic Reproduction Number of the model.

There are two host populations and one vector in the model, and it was shown from the model flow diagram (Figure 1) that the vector transmits the infection to human host and secondary host (monkey). Hence, the Basic Reproduction Number can be represented as:

$$R_0 = \sqrt{R_{hv} + R_{mv}} \text{ or } R_0^2 = R_{hv} + R_{mv} \quad (21)$$

Such that:

$$R_{hv} = \frac{\alpha_1 \alpha_2 A_5 \mu_h^2}{A_1 A_2 A_3^2} \quad (22)$$

Which is the basic reproduction number of human-vector compartments and represents the infection from vector to human and human to vector, and:

$$R_{mv} = \frac{\alpha_3 \alpha_4 A_6 \mu_m}{A_3^2 A_4} \quad (23)$$

Which is the basic reproduction number of monkey-vector compartments and represents the infection from vector to monkey and monkey to vector.

## RESULT AND DISCUSSION

### Local Stability of Disease Free Equilibrium (DFE), $E_0$

**Theorem 1:** The Disease Free Equilibrium of the model system (1)-(7) is locally asymptotically stable if  $R_0 < 1$  and unstable if  $R_0 > 1$

**Proof:**

$$J(E_0) = \begin{bmatrix} -A_1 & 0 & 0 & 0 & -B_1 & 0 & 0 \\ 0 & -A_2 & 0 & 0 & B_1 & 0 & 0 \\ v & \gamma_h & -\mu_h & 0 & 0 & 0 & 0 \\ 0 & -B_2 & 0 & -A_3 & 0 & 0 & -B_3 \\ 0 & B_2 & 0 & 0 & -A_3 & 0 & B_3 \\ 0 & 0 & 0 & 0 & -B_4 & -\mu_m & 0 \\ 0 & 0 & 0 & 0 & B_4 & 0 & -A_4 \end{bmatrix} \quad (24)$$

Where,

$$B_1 = \frac{\alpha_1 \Lambda_h}{A_1 N_h}, B_2 = \frac{\alpha_2 \Lambda_v}{A_3 N_h}, B_3 = \frac{\alpha_3 \Lambda_v}{A_3 N_m}, B_4 = \frac{\alpha_4 \Lambda_m}{\mu_m N_m} \quad (25)$$

Using Gaussian elimination row operation on (24) gives the characteristics Equation:

$$(-A_1 - \lambda)(-A_2 - \lambda)(-A_1 A_2 \mu_h - \lambda)(-A_2 A_3 - \lambda)(K_1 - \lambda)(-K_1 \mu_m - \lambda)(-(K_2 + K_1 A_4) - \lambda) = 0 \quad (26)$$

Where

$$\begin{aligned} K_1 &= B_1 B_2 - A_2 A_3, & K_2 &= A_2 B_3 B_4 \\ \lambda_1 &= -A_1, & \lambda_2 &= -A_2, & \lambda_3 &= -A_1 A_2 \mu_h, & \lambda_4 &= -A_2 A_3, & \lambda_5 &= K_1, & \lambda_6 &= -K_1 \mu_m \\ \lambda_7 &= -(K_2 + K_1 A_4) \end{aligned} \quad (27)$$

The DFE will be asymptotically stable if,

$$\lambda_5 < 0 \quad (28)$$

This implies that,

$$B_1 B_2 < A_2 A_3 \quad (29)$$

$$\alpha_1 \alpha_2 A_5 \mu_h^2 < A_1 A_2 A_3^2 \quad (30)$$

$$\frac{\alpha_1 \alpha_2 A_5 \mu_h^2}{A_1 A_2 A_3^2} < 1 \quad (31)$$

$$R_{hv} < 1 \quad (32)$$

So, also

$$\lambda_7 < 0, \quad (33)$$

implies that,

$$A_2 B_3 B_4 + A_4 B_1 B_2 < A_2 A_3 A_4 \quad (34)$$

$$\frac{A_5 \alpha_1 \alpha_2 \mu_h^2}{A_1 A_2 A_3^2} + \frac{A_6 \alpha_3 \alpha_4 \mu_m}{A_3^2 A_4} < 1 \quad (35)$$

$$R_0 < 1 \quad (36)$$

### Global Stability of Disease Free Equilibrium (DFE), $E_0$

**Theorem 2:** The DFE,  $E_0$  of the model system is globally asymptotically stable if  $R_0 \leq 1$ .

**Proof:**

Consider the Lyapunov-Lasalle function:

$$V(S_h, I_h, R_h, V_1, V_2, S_m, I_m) = \frac{A_4 \alpha_2 \Lambda_V \mu_h I_h}{A_3 \Lambda_h} + \frac{A_2 \alpha_3 \Lambda_V \mu_m I_m}{A_3 \Lambda_m} + A_2 A_4 V_2 \quad (37)$$

Differentiating (37) gives:

$$\frac{dV}{dt} = \frac{A_4 \alpha_2 \mu_h \Lambda_V}{A_3 \Lambda_h} \left[ \frac{\alpha_1 S_h V_2}{N_h} - A_2 I_h \right] + \frac{A_2 \alpha_3 \mu_m \Lambda_V}{A_3 \Lambda_m} \left[ \frac{\alpha_4 S_m V_2}{N_m} - A_4 I_m \right] + A_2 A_4 \left[ \frac{\alpha_2 V_1 I_h}{N_h} + \frac{\alpha_3 V_1 I_m}{N_m} - A_3 V_2 \right] \quad (38)$$

Since

$$S_h \leq S_h^0, V_1 \leq V_1^0, S_m \leq S_m^0, N_h \leq N_h^0 \text{ and } N_m \leq N_m^0 \quad (39)$$

Equation (38) becomes:

$$\frac{dV}{dt} \leq A_2 A_3 A_4 \left[ \frac{A_5 \alpha_1 \alpha_2 \mu_h^2}{A_1 A_2 A_3^2} + \frac{A_6 \alpha_3 \alpha_4 \mu_m}{A_3^2 A_4} - 1 \right] V_2 \quad (40)$$

$$\frac{dV}{dt} \leq A_2 A_3 A_4 [R_0^2 - 1] V_2 \quad (41)$$

$$\frac{dV}{dt} \leq 0 \quad (42)$$

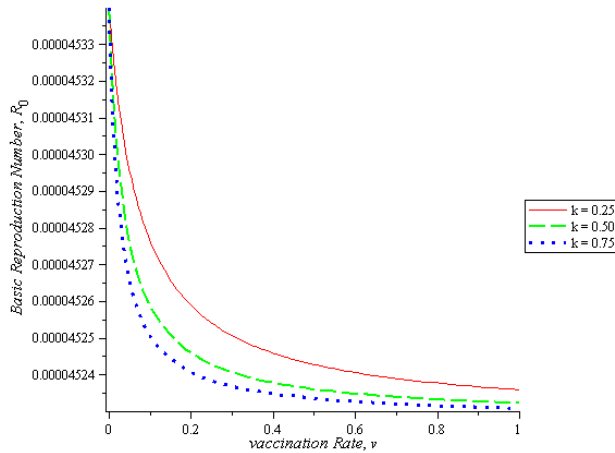
if  $R_0^2 \leq 1$ , then

$$R_0 \leq 1 \quad (43)$$

Hence, the DFE is globally asymptotically stable.

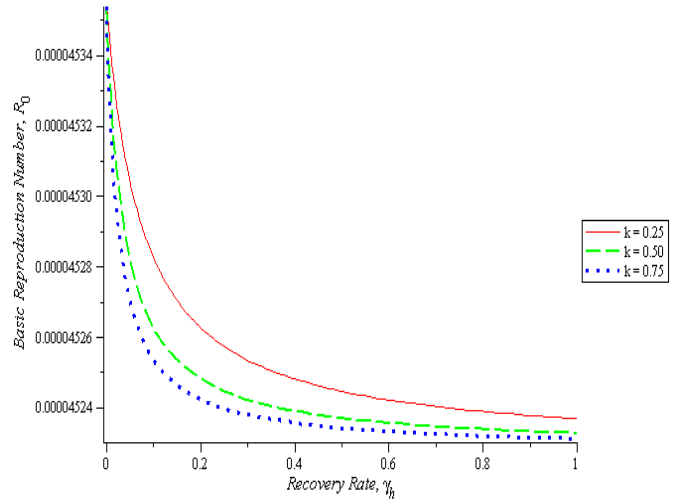
### Graphical Representation of Basic Reproduction Number with Control Parameters

We vary the control parameters in the model with basic reproduction number,  $R_0$ . The control parameters are vaccination rate, recovery rate and death of mosquito due to application insecticide. Where  $k$  is the different proportion of each control parameters.



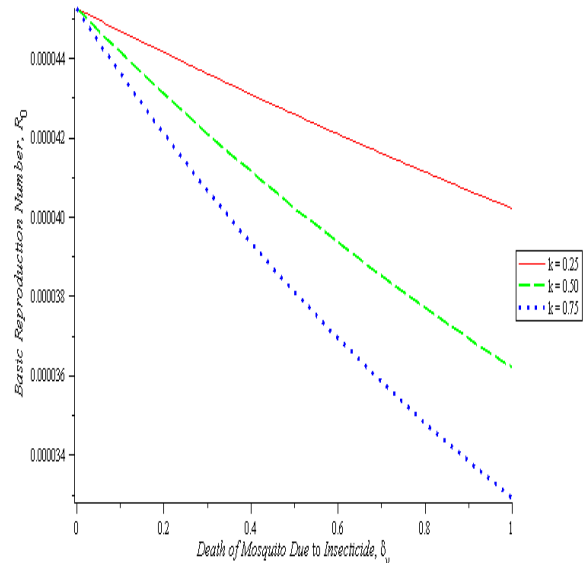
**Figure 2:** Effect of Vaccination Rate of Humans on Basic Reproduction Number.

Figure 2, shows that, as vaccination rate increases the basic reproduction number decreases. This shows that immunization of susceptible individuals will reduce the outbreak of yellow fever. Vaccination rate is the key parameter in controlling the outbreak of yellow fever.



**Figure 3:** Effect of Recovery Rate of Humans on Basic Reproduction Number.

Figure 3, shows that, as recovery rate of humans increases the basic reproduction decreases. This shows that more people are treated of the disease the less the future outbreak. As people recovered from yellow fever, they become permanently immune.



**Figure 4:** Effect of Death of Mosquitoes due to Insecticide of Mosquito on Basic Reproduction Number.

Figure 4 shows that, as application of insecticide increases the basic reproduction number decreases. Eradication of mosquitoes from the community will drastically reduce the outbreak of yellow fever.



It is observed from Figure 4 that the 75% application of insecticide reduced the basic reproduction number to almost zero.

## CONCLUSION

It was observed from above that the local and global stability of Disease Free Equilibrium (DFE) is asymptotically stable if  $R_0 < 1$  and if  $R_0 \leq 1$ , respectively. This implies that once the disease enters the population it can die out with time. The graphical presentation of the basic reproduction number against the control parameters shows that, with high; vaccination rate, recovery rate and absence of the mosquitoes in the community will bring yellow fever under control. It was also observed from all the graphs that the basic reproduction number is less than one (i.e.  $R_0 < 1$ ) which implies that the disease will not persist in the population.

Government should put more effort in immunization of yellow fever and people should be sensitized to avoid the bite of mosquito.

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