Stability and Sensitivity Analysis of Yellow Fever Dynamics

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Abstract—Several West African countries have recently reported of Yellow Fever outbreaks. Ghana recently recorded an outbreak which leads to the death of three (3) people in the West Gonja District of the Northern Region. These indicate the re-emergence of the deadly disease. This research proposes a deterministic mathematical model through non-linear ordinary differential equations in order to gain an accurate insight into the dynamics of yellow fever between human beings and the vector Aedes mosquito in an unvaccinated area to control the spread of the disease. The disease threshold parameter was obtained using the next-generation matrix. The Gerschgorin theorem proved the disease-Free equilibrium and the Endemic equilibrium to be locally asymptotically stable for $R_0 < 1$ and $R_0 > 1$ respectively. The Lyapunov function proved the disease-Free Equilibrium to be globally asymptotically stable for $R_0 < 1$. In order to study the effect of the model parameters to $R_0$, the sensitivity analysis of the basic reproduction number with respect to epidemiological parameters was performed.

Index Terms—Endemic Equilibrium, Reproduction Number, Sensitivity Analysis, Transmission Rate.

I. INTRODUCTION

Yellow fever (YF) is a hemorrhagic fever caused by Flavivirus and spreads between humans, as well as between certain other primates and humans, by the bite of YF – infected Aedes mosquitoes. YF is endemic in tropic areas of Latin America and Africa with a combined population of over 900 million people [1]. YF is difficult to diagnose, especially during the early stages. It can be misdiagnosed as severe malaria, dengue hemorrhagic fever, viral hepatitis and other viral fevers. Once infected, the virus incubates in the body for 3 to 6 days, followed by an infection that occurs in three phases [2]. The first phase during which virus is present in blood, is characterized by fever, malaise, nausea, vomiting and dizziness. The second phase is characterized by improvement in symptoms, including a reduction of fever, which may last for 48 hours. Some infected individuals recover at this stage without developing jaundice. However, 15% of patients enter the last stage characterized by the return of fever, nausea, vomiting, jaundice, and bleeding diathesis [2]. According to the World Health Organization (WHO), 50% of patients who enter the toxic phase die within 10 to 14 days. Mathematical models have become important tools in analyzing the spread and control of infectious disease and as such several researchers have used mathematical models to examine the transmission and check the spread of YF. [3] performed research on Modelling and Stability Analysis of SVEIRS Yellow Fever Two Host Model. Again, [4] investigated the reproductive number for YF between primates, human beings and Aedes mosquitoes for the purpose of controlling the disease with vaccination. [5] proposed a mathematical method for the estimation of the Basic Reproduction Number, $R_0$, of YF in a dengue-infested area. Finally, [6] further studied the Equilibrium Analysis of YF Dynamics Model with Vaccination. However, most studies on the transmission dynamics of YF were performed without vaccination. In order to get adequate information about the transmission dynamics of YF, we propose in this research a six (6) compartment mathematical model for modelling the transmission dynamics of YF without vaccination. This will enable us to perform stability and sensitivity analysis of the dynamics between human and the mosquito vector on an unvaccinated geographical area.

II. MODEL FORMULATION

We consider a transmission model for the spread of YF between human and vector populations with the total population of $N_h$ and $N_v$ respectively. The populations are further compartmentalized into different epidemiological subclasses as shown in Fig. 1.

From the compartmental diagram in Fig. 1, the model divides the human population into four distinct subclasses: susceptible, $S_h$, exposed, $E_h$, Infectious, $I_h$ and Recovered, $R_h$. People only enter the susceptible class through birth and leaves the population through a natural death rate of $\mu_h$. Newly born babies do not have the disease. The model applies to completely unvaccinated geographical grounds.

Similarly, the vector population is divided into two classes.

Female mosquito enters the susceptible class through birth then leaves the population through natural death rate $\mu_v$. A susceptible human host acquires the virus through the bite from an infectious vector. The susceptible host gets expose and becomes infectious after an incubation period of 3 to 6 days. However, some get treated, recover and become susceptible to the virus again, that is to say, there is no permanent immunity. Similarly, a susceptible vector acquires the virus from an infectious human through vector
bite. The model relies on the following assumptions: the host and vector populations are confined to a particular geographic area, transmission continues in the populations, death occurs equally in all groups and migration of both vector and host is ignored.

The absence of recovered class for the vector is because mosquitoes do not recover from the infection or die from the infection, since mosquitoes are only carriers of the virus. This implies that an infected vector becomes infectious for life.

A. Model Flow Diagram

The model for the transmission of YF in Host (human) and vector is shown Fig. 1.

![Flow Diagram](image)

Fig. 1. The model for the transmission of YF in Host (human) and vector

B. Model Equations

The differential equations which describes the dynamics of YF in the human and mosquito vector formulated as shown below:

**Human**

\[
\begin{align*}
\frac{dS_h}{dt} &= b_h N_h - \frac{k \beta S_h I_v - \mu_h S_h + c R_h}{N_h} \\
\frac{dE_h}{dt} &= \frac{k \beta S_h I_v}{N_v} - \delta_h E_h - \mu_h E_h \\
\frac{dI_h}{dt} &= \delta_h E_h - \gamma_h I_h - \mu_h I_h - \epsilon_h I_h \\
\frac{dR_h}{dt} &= \gamma_h I_h - \mu_h R_h - c R_h
\end{align*}
\]

(1)

**Vector mosquito**

\[
\begin{align*}
\frac{dS_v}{dt} &= b_i N_v - \frac{k \beta S_v I_v - \mu_i S_v}{N_h} \\
\frac{dI_v}{dt} &= \frac{k \beta S_v I_v}{N_h} - \mu_i I_v
\end{align*}
\]

(2)

In the model, the term \( \frac{k \beta S_h I_v}{N_h} \) represents the rate at which susceptible human host \( S_h \) get infected from the infected human host \( I_v \) (force of infection from vector to human) and \( \frac{k \beta S_v I_v}{N_h} \) denotes the rate at which susceptible vector \( S_v \) get infected from the infected human host \( I_h \) taking into consideration the timely biting rate of the mosquito vector. After sometime, the infectious human population recover at a rate of \( \gamma_h \) and enters the susceptible class again at the rate of \( c \). Humans leave the population by two means which are the natural death at the rate of \( \mu_h \) and the disease-induced death at a rate of \( \epsilon_h \).

The total population sizes \( N_h(t) \) and \( N_v(t) \) can be determined by;

\[
N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t)
\]

(3)

\[
N_v(t) = S_v(t) + I_v(t)
\]

(4)

III. Model Analysis

A. The Disease-Free Equilibrium

The disease-free Equilibrium is obtained by setting (1) and (2) to zero.

The system of equations has the disease-free equilibrium as:

\[
[S_h = \frac{b_h N_h}{\mu_h}, E_h = 0, I_h = 0, S_v = \frac{b_i N_v}{\mu_i}, I_v = 0]
\]

B. Determination of Basic Reproduction number \( (R_0) \)

The basic Reproduction number denoted by \( (R_0) \) is the number of secondary cases which one case would produce in a completely susceptible population. The next generating method is used in calculating the basic Reproduction number.

The disease compartments of the model are the exposed host \( (E_h) \), and the infectious human host \( (I_h) \). The system of equations can be written as

\[
\frac{dx}{dt} = f(x) - \nu(x)
\]

(5)

Where

\[
f(x) = \begin{pmatrix}
k \beta S_h I_v \\
0 \\
k \beta S_h I_v \\
N_h
\end{pmatrix}
\]

(6)

and

\[
\nu(x) = \begin{pmatrix}
-(\delta_h + \mu_v) E_h \\
-\gamma_v + \mu_v + \epsilon_v I_v + \delta_v E_v \\
-\mu_v I_v
\end{pmatrix}
\]

(7)
\[ \mathbf{v}(x) = \begin{pmatrix} (\delta_b + \mu_b)E_b \\ (\gamma_b + \mu_b + \varepsilon_0)I_b - \delta_h E_b \\ \mu_e I_e \end{pmatrix} \]

Evaluating the \( \mathbf{f}(x) \) and \( \mathbf{v}(x) \) at the disease-free equilibrium,

\[ \partial f(x)_{E_b,I_b} = \begin{pmatrix} 0 & 0 & k \beta S_b \\ 0 & 0 & 0 \\ k \beta S_b & 0 & N_b \end{pmatrix} \]

\[ F = \partial f(x)_{DFE} = \begin{pmatrix} 0 & 0 & k \beta b \mu_b \nu_b \\ 0 & 0 & 0 \\ k \beta b \mu_b \nu_b \mu_e & 0 & N_b \end{pmatrix} \]

Evaluating \( \mathbf{v}(x) \) at the disease-free equilibrium:

\[ \partial v(x)_{E_b,I_b} = \begin{pmatrix} \delta_b + \mu_b & 0 & 0 \\ -\delta_b & \gamma_b + \mu_b + \varepsilon_0 & 0 \\ 0 & 0 & \mu_e \end{pmatrix} \]

\[ \partial v(x)_{DFE} = \begin{pmatrix} \delta_b + \mu_b & 0 & 0 \\ -\delta_b & \gamma_b + \mu_b + \varepsilon_0 & 0 \\ 0 & 0 & \mu_e \end{pmatrix} \]

The next generation matrix for the model is

\[ FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{k \beta b \mu_b N_b}{\mu_e N_e} \\ 0 & 0 & 0 \\ \frac{k \beta b \mu_b N_b}{\mu_e N_e} & \frac{k \beta b \mu_b N_b}{\mu_e N_e} & 0 \end{pmatrix} \]

\[ \Rightarrow \mathbf{R}_e = \sqrt{\mu_e \mu_b (\delta_b (\epsilon_b + \gamma_b + \mu_b) + \mu_e (\epsilon_b + \gamma_b + \mu_b))} \]

C. The Endemic Equilibrium

Simplification of the Endemic Equilibrium gives:

\[ \mathbf{S}_e = \frac{N_e (\epsilon_b + \gamma_b + \mu_b) \delta_b + \mu_e (\epsilon_b + \gamma_b + \mu_b) (\mathbf{R}_e^2 \beta - 1)}{k \beta \delta_b \mu_e (\mathbf{R}_e^2 \beta + 1 + \eta_e)} \]

\[ \mathbf{E}_e = \frac{N_e (\epsilon_b + \gamma_b + \mu_b) \mu_e \mathbf{R}_e (\mathbf{R}_e^2 \beta - 1)}{k \beta \delta_b \mu_e (\mathbf{R}_e^2 \beta + 1 + \eta_e)} \]

\[ \mathbf{I}_e = \frac{N_e (\epsilon_b + \gamma_b + \mu_b) \mu_e \mathbf{R}_e (\mathbf{R}_e^2 \beta - 1)}{k \beta \delta_b \mu_e (\mathbf{R}_e^2 \beta + 1 + \eta_e)} \]

Where \( \eta_1, \eta_2, \eta_3, \eta_4 \) and \( \eta_5 \) are defined as

\[ \eta_1 = \mu_e \mu_b \delta_b \mu_e \mathbf{R}_e (\mathbf{R}_e^2 \beta + 1 + \eta_e) \]

D. Local Stability of Disease-Free Equilibrium

The local stability analysis of the disease-Free Equilibrium is performed using Geschgorin’s theorem of stability.

Theorem

The Disease-Free Equilibrium (DFE) is locally asymptotically stable if \( \mathbf{R}_e < 1 \) whereas unstable if \( \mathbf{R}_e > 1 \) [7].

Proof

The Jacobian matrix of the system of (1) and (2) is given by

\[ J = \begin{pmatrix} \frac{k \beta b I}{N_e} - \mu_e & 0 & 0 & c & 0 & \frac{k \beta b S}{N_e} \\ \frac{k \beta b I}{N_e} & -\delta_e & 0 & 0 & 0 & 0 \\ 0 & \delta_e & -\epsilon_e - \gamma_e - \mu_e & 0 & 0 & 0 \\ 0 & 0 & \gamma_e & -c - \mu_e & 0 & 0 \\ 0 & 0 & 0 & \frac{k \beta b S}{N_e} & 0 & 0 \\ 0 & 0 & 0 & 0 & -\frac{k \beta b I}{N_e} & -\mu_e \end{pmatrix} \]
From Geschgorin’s theorem,

\[
\begin{align*}
\mu_h &> c + \frac{k \beta_h b_h N_h}{\mu_s N_v} \\
\delta_h + \mu_h &> \frac{k \beta_h b_h N_h}{\mu_s N_v} \\
\varepsilon_h + \gamma_h + \mu_h &> \delta_h \\
\nu_e > \frac{k \beta_h b_h N_e}{\mu_s N_v} \\
\mu_e &> \frac{k \beta_h b_h N_e}{\mu_s N_v}
\end{align*}
\]

Multiplying second inequality in (20) by also the third inequality in (20) results in

\[
\mu_h (\delta_h + \mu_h) (\varepsilon_h + \gamma_h + \mu_h) > \delta_h \frac{k \beta_h b_h N_h}{N_v}
\]

(21)

Multiplying (21) by the fifth inequality in (20) and dividing through by the left-hand side of the obtained equation yields

\[
1 > \frac{\mu^2 \mu_h (\delta_h + \mu_h) (\varepsilon_h + \gamma_h + \mu_h)}{k^2 \beta_h b_h b_h \delta_h}
\]

(22)

\[
\Rightarrow 1 > \mathfrak{R}_h^2
\]

(23)

Hence

\[
\mathfrak{R}_h^2 < 1
\]

(24)

This affirms that the DFE is asymptotically stable.

**E. Local Stability of Endemic Equilibrium (EE)**

The local stability analysis of the Endemic Equilibrium is performed using Geschorin’s theorem of stability.

**Theorem**

The Endemic Equilibrium (EE) is locally asymptotically stable if \( \mathfrak{R}_h > 1 \) [7].

In likewise manner, the local stability of the Endemic Equilibrium is found by differentiating the model equations (1) and (2) with respect to \( S_h, E_h, I_h, R_h, S_v \) and \( I_v \) which gives the Jacobian matrix and evaluating the Endemic Equilibrium solution in the Jacobian.

Evaluating the Endemic Equilibrium solution in the Jacobian matrix (19) gives:

\[
J_{EE} = \begin{pmatrix}
-\mu_e & 0 & 0 & c & -k \beta_h b_h N_h \\
0 & -\delta_h - \mu_h & 0 & 0 & 0 \\
0 & \delta_h & -\gamma_h - \mu_h & 0 & 0 \\
0 & 0 & \gamma_h & -c - \mu_h & 0 \\
0 & 0 & -k \beta_h b_h N_e & 0 & -\mu_e \\
0 & 0 & k \beta_h b_h N_e & 0 & -\mu_e
\end{pmatrix}
\]

(19)

\[
\omega = \beta_h N_h (\varepsilon_h + \gamma_h + \mu_h) (\delta_h + \mu_h) (\mathfrak{R}_e \eta_e + 1 + \eta_e) \\
\beta_i N_i (\mathfrak{R}_e \eta_e + 1 + \eta_e)
\]

(20)

\[
\delta_h + \mu_h > \frac{(c + \mu_h) \eta_e (\mathfrak{R}_e - 1) + (c + \mu_h) \eta_e (\mathfrak{R}_e - 1) + \eta_e}{(c + \mu_h) \eta_e (\mathfrak{R}_e - 1) + \eta_e (\mathfrak{R}_e - 1) + \eta_e}
\]

(21)

\[
\Rightarrow 1 > \mathfrak{R}_e^2
\]

(22)

Hence

\[
\mathfrak{R}_e^2 < 1
\]

(23)

This affirms that the DFE is asymptotically stable.

**F. Global Stability Analysis of the Disease-Free Equilibrium (DFE)**

The Lyapunov function is applied to determine the global stability of the Disease-Free Equilibrium (DFE).

**Theorem**

If \( \mathfrak{R}_h \leq 1 \), then the free disease equilibrium of the system of (1) and (2) is globally asymptotically stable [7].

**Proof**

The Lyapunov function is constructed as follows:

\[
V = (S_h - S_h^* \ln S_h) + E_h + I_h + R_h + (S_v - S_v^* \ln S_v) + I_v
\]

(33)

With the assumption that

\[
\mu_e = \frac{k \beta S_e^*}{N_e} \quad \varepsilon_e = \frac{k \beta S_e^*}{N_e}
\]

(34)

\[
\omega = \frac{(c + \mu_h) \eta_e (\mathfrak{R}_e - 1) + \eta_e (\mathfrak{R}_e - 1) + \eta_e}{(c + \mu_h) \eta_e (\mathfrak{R}_e - 1) + \eta_e (\mathfrak{R}_e - 1) + \eta_e}
\]

(25)}
Differentiating (33) with respect to time gives
\[ \dot{V} = S_h(1 - \frac{S^*}{S_h}) + \dot{E}_h + \dot{I}_h + \dot{R}_h + \dot{S}_\iota (1 - \frac{S^*}{S_\iota}) + \dot{I}_\iota \]  
(35)

Substituting model equations (1) and (2) into (35) and using the earlier stated assumptions (34) results in:
\[ \dot{V} = b_h N_h (1 - \frac{S^*}{S_h}) + \mu_h S^* (1 - \frac{S}{S_h}) - c_R h (\frac{S^*}{S_h}) \\
- (E_h + I_h + R_h) \mu_h + b_i N_i (1 - \frac{S^*}{S_i}) + \mu S^* (1 - \frac{S}{S_i}) \]
(36)

At the DFE;
\[ S_h^* = \frac{b_h N_h}{\mu_h} \quad \text{and} \quad S_i^* = \frac{b_i N_i}{\mu_i} \]
\[ \dot{V} = -b_h N_h \left[ \frac{(S_h - S^*)^2}{S_h S^*} \right] - b_i N_i \left[ \frac{(S_i - S^*)^2}{S_i S^*} \right] \\
- c_R h (\frac{S^*}{S_h}) - (E_h + I_h + R_h) \mu_h \]
(37)

\[ \Rightarrow \dot{V} < 0 \]  
(38)

This affirms that the DFE is globally asymptotically stable.

IV. SENSITIVITY ANALYSIS OF $R_0$

In order to determine how best to reduce mortality and morbidity due to YF infection, it is necessary to know the relative importance of the different factors responsible for its transmission and prevalence [8]. Initial disease transmission is directly related to $R_0$, and disease prevalence is directly related to the endemic equilibrium points, specifically to the magnitudes of $S_h$, $E_h$, $I_h$, $R_h$, $S_i$, and $I_i$.

Thus, sensitivity analysis of the basic reproduction number with respect to model parameters is performed. The sensitivity analysis will assist in curtailing the transmission of the disease by using the appropriate intervention strategies [4].

The sensitivity index of $R_0$ with respect to the biting rate of mosquito $k$, for example is
\[ \frac{\partial R_0}{\partial k} \times \frac{k}{R_0} = 1 \]  
(39)

The biting rate of the mosquito, $k$ and the death rate of the vector are seen to be the most sensitive parameter by analysis their sensitivity indices. The birth rate on vector, $b_v$, birth rate of human, $b_h$, the probability of disease transmission from infected vector to susceptible human, $\beta_i$, the probability of disease transmission from infected human to susceptible vector, $\beta_h$, the progression rate of exposed human $\delta_h$ are considered relevant parameters in the spread of YF.

The reproduction number $R_0$ is directly related to the biting rate of mosquito, transmission probability of vector to human as well as the progression rate of exposed vector and human which conforms to the research of [4] and inversely related to death rate of vector and human.

Since $Y^{R_0}_k = 1$, increasing (or decreasing) $k$ by 20% increases (or decreases) $R_0$ by 20%. Also $Y^{R_0}_b = -1$ implies increasing the rate at which mosquitoes die by 10% (shorting the lifespan of the vector) will decrease the reproduction number $R_0$ by 10%. Similarly increasing (or reducing) $\delta_h$, $\beta_i$, and $\beta_h$ by 10% increases (decreases) $R_0$ by 5%. Reducing the biting rate and contact rate (the probability of transmitting YF) between human and vector will have the largest effect on disease transmission as seen from the analysis. The use of treated bednets, insect repellents, insecticides and larvacides are therefore suggested the strategies that can be applied in controlling the disease targeting the mosquito biting rate and death rate.

A. Model Simulations

This section presents some numerical results for the model. The values of the parameters used in the simulation of the model were obtained from various sources. The human birth and mortality rates were taken as that of Ghana as estimated in 2014 by index Mundi. The values of parameters and sources used for the simulation are given in Table II. All simulations were performed using the Maple Software.
TABLE II: DESCRIPTION OF PARAMETERS VALUES OF MODEL SYSTEM

| Parameter | Value          | Source                      |
|-----------|----------------|-----------------------------|
| $N_0$     | 1 000 000      | Estimated                   |
| $N_b$     | 20 0000        | Estimated                   |
| $S_b(0)$  | 198 000        | Estimated                   |
| $E_b(0)$  | 2 000          | Estimated                   |
| $I_b(0)$  | 0              | Estimated                   |
| $R_b(0)$  | 0              | Estimated                   |
| $S_c(0)$  | 950 000        | Estimated                   |
| $I_c(0)$  | 50 000         | Estimated                   |
| $\beta_1$| 0.75           | (Kung’aro et al., 2014)     |
| $\beta_2$| 0.375          | (Kung’aro et al., 2014)     |
| $k$       | 0.75           | (Kung’aro et al., 2014)     |
| $\delta_b$| 0.1            | (Kung’aro et al., 2014)     |
| $\gamma_b$| 0.083          | (Kung’aro et al., 2014)     |
| $b_v$     | 0.5            | Side and Noorani (2013)     |
| $b_b$     | 8.603 x 10^{-5} | Index Mundi(www.indexmundi.com) |
| $\mu_b$  | 2.019 x 10^{-5} | Index Mundi(www.indexmundi.com) |
| $\epsilon_b$ | 2.5 x 10^{-4} | (Kung’aro et al., 2014)     |
| $\mu_r$  | 0.35           | (Kung’aro et al., 2014)     |

Fig. 2. Dynamics of YF

Fig. 2 illustrates the dynamics of YF in a period of 200 days in an unvaccinated geographical area of 200 000 susceptible human population when a population of 1 000 000 mosquito vectors of which 50 000 are YF infectious are introduced into the human population. The simulation indicates the population of the various compartment varies with time as seen in Fig. 2.

The population of susceptible as indicated in Fig. 3 is equal to the total host population when there is no disease infection for the human population. At the onset of infection, the susceptible human begins to decreases. As time increases it begins to rise slightly. This is because recovered individual tends to join the susceptible class since there is no permanent immunity to YF. A major factor that accounts for this is the rate of loss of immunity for humans to YF.

Fig. 4 clearly illustrates the variation of the exposed human population as time changes. It could be observed that, the exposed human population begins to rise from 0 (no expose individual at time 0) and increases with time as the susceptible human population tends to decrease and become asymptotic to the horizontal axes with time. The contact rate between host and vector and the biting rate of the vector affect greatly the observed variation in the exposed population over time.
The infectious population Fig. 5 behaves similar to the exposed population. Few individuals were initially infected but over time, the infection spreads throughout the population. However, with time the infected population tends to decrease. This being the reason that about 50% of the infected individual without treatment would die of YF [12]. This accounts for the smooth decline in the infected population as a result of the disease induce death. However, the curve tends to be asymptotic to the horizontal line since not all infected individuals will die. Meanwhile the recovered population as seen in Fig. 6 continuously increases steadily over time. The dynamics however shows that the curve remains at a constant from a point in time. This depicts the point in time when the people entering the recovery compartment balance those leaving to the susceptible compartment.

The population of the susceptible mosquito vector increases slightly within a very short time. This illustrates how fast a female mosquito can breed within its short life span. On average a mosquito’s life span is typically 2-3 weeks, however for the lucky mosquitoes who can find a hideout from unfavourable weather, have the chance of living for up to 6 months. Furthermore, female mosquitoes can lay 100 to 300 eggs at a time. A female mosquito can lay between 1000 and 3000 eggs in their lifespan [11]. Meanwhile, a very sharp decrease in the susceptible vector population within a very short time as shown in Fig.7 indicates that susceptible mosquitoes have a very high probability of getting infected. However, the susceptible population tends to increase again due to the high birth rate of female mosquitoes until it balances with the death rate over time.

Fig. 8 reveals an increase in the infectious vector as susceptible vectors get infected and become infectious. However, the population tends to decrease within time since mosquito has a very short average life span [11].

V. CONCLUSIONS AND RECOMMENDATION

A. Conclusions

The study formulated a compartmental model for YF using nonlinear differential equations and analyzed the model to determine the stability and sensitivity analysis of the YF dynamics. From the analysis, the basic reproduction number was obtained to 5.25 which is greater than one (1). This indicates an endemic situation. Also, from the sensitivity analysis conducted, the biting rate of the mosquito and the death rate of the mosquito were seen to be more sensitive.

The stability analysis showed that the basic reproduction number is less than unity at the DFE and greater than unity at the EE. Furthermore, it was observed that an increase in the proportion of infected vector and biting rate of vector to human contributes greatly to an increase in YF transmission dynamics.

B. Recommendations

Results from the model calls for attention to parameters regarding daily biting rate of mosquitoes, recruitment rates of vectors and probability of contact between infected mosquito vector and Human. Therefore, much attention and strategies should be given to these parameters in the quest to control and eradicate YF. These can be achieved by vector control strategies to minimize breeding of mosquitoes, ensuring personal preventive measures which includes the use of mosquito repellents and treated bed-nets.

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