

Modelling the Dynamics and Transmission of Dengue Fever by the Vector, Mosquito *Aedes aegypti* in Tropical Africa Region

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Abstract: Dengue Fever (DF) is notably one of the world's common vector-borne viral disease. The current increase in the geographic distribution of its primary vector, mosquito *Aedes aegypti*, has led to a 30-fold increase in cases over the last 50 years in tropical regions of Asia, Africa, Central and South America. This paper presents an SEITR-SI model, (Susceptible, Exposed, Infected, Treatment, and Recovered) host-vector, *Aedes aegypti* mosquito to determine the dynamics of dengue fever especially in tropical regions of Africa where malaria is highly prevalent due to illegal mining activities and urbanization. We classified the human population into five epidemiological states and the mosquito population into two compartmental epidemiological states. The disease-free equilibrium point and its stability states were established and simulated. The model's threshold parameter R_0 was further estimated using the Next Generation and Jacobian matrix. Numerical simulations of the model reveal that the emergence of new cases of DF in some Africa regions and Ghana call for proper diagnosis of malaria using adequate diagnostic tools and treatment to avoid transmission as a result of misdiagnosis of febrile illnesses as malaria.

Keywords: Dengue Fever, vector, viral disease, mosquito, *Aedes aegypti*

1. Introduction

Dengue virus infection is the most common arthropod-borne disease worldwide with an increasing incidence in the tropical regions of Asia, Africa and Central and South America. There are four serotypes of the virus and these are transmitted by mosquitoes [1]. Dengue fever has rapidly become the world's most common vector-borne viral disease. An estimate of 390 million dengue infections annually occurs worldwide, with about 96 million resulting in illness. Dengue fever is transmitted by the bite of an *Aedes* mosquito infected with a dengue virus. The mosquito becomes infected when it bites a person with dengue virus in the blood. Most of the infectious cases occur in the tropical areas of the world, with greater risk occurring in the Southeast Asia, Indian subcontinent, Southern China, Taiwan, the Pacific islands, Mexico, Africa and many others [2].

The recent increase in the geographic distribution of its primary vector, the mosquito *Aedes aegypti*, has led to a 30-fold increase in cases over the last 50 years [3], and is now found throughout the tropics. Although dengue fever itself is rarely fatal, one of its more severe forms is the dengue hemorrhagic fever (DHF) which causes 22,000 deaths annually mostly among children [4].

Ghana as a warm tropical climate country in West Africa has an abundance of *Aedes aegypti* mosquitoes [5]. Cote d'Ivoire and Burkina Faso reported Dengue Virus (DENV) outbreaks in 2015 and 2016, respectively. Nonetheless, DENV infection was confirmed in Ghana in two children and four adults in two separate studies in 2018 [6]. A recent study conducted in Ghana revealed 218 (21.6%) children exposed to dengue. The study sampled a population of febrile illness patients who were screened for active dengue

infection but were properly diagnosed and confirmed as malaria patients [7].

Unfortunately, there are very limited control measures for most of the vector-borne diseases. Vaccines available for only a few diseases (yellow fever, Japanese encephalitis, tick-borne encephalitis, tularemia, plague) are not widely used, and vaccines for some widespread diseases, such as West Nile virus, malaria, and dengue fever are still not available [3]. People have to depend on vector control programs including the removal of breeding sites generated by humans in households (e.g., old toys, water containers, and tyres), larvicidal control, and Malathion spraying to target adult vector populations [4]. Other public health controls rely on shortening the mean vector span or directly reducing the vector biting rate in humans through netting, screens, and application of insecticides life to clothing or the application of insect repellents. However, these methods appear not to be sufficiently effective, as the frequency of outbreaks appears to be increasing in some areas, probably due to urbanization which readily increases the habitat of *Aedes aegypti* [5].

Recent surveys have uncovered dengue exposure throughout sub-Saharan Africa and West Africa has been identified as a potential hotspot for transmission because of the existence of the *Aedes* mosquito vector, rapid urbanization with inadequate sanitation, and low clinical knowledge of flavivirus infections [8]. Research finding of Lam *et al.* [3] estimated the annual number of cases DF worldwide from 100 million in 1997 to 500 million in 2012.

Onstad *et al.* [9] reviewed the early vector-borne disease models, and many other authors have studied vector-borne diseases, such as malaria [10] and West Nile virus [11].

Mathematical modelling of infectious diseases is an important way of studying the dynamics of epidemiological diseases such as DF. Mathematical models often serve as a framework to convey how the vector components of a host-parasite interacts by means of how the disease spreads, forecast future course of an outbreak and evaluate how an epidemic can be controlled [1].

According to Aguiar *et al.* [12] mathematical modeling is an interesting tool for understanding the epidemiology of infectious diseases and its dynamics. Moreover, due to inadequate control measures for vector-borne disease, mathematical modelling usually incorporate the ideas of how to curb the disease by focusing on the dynamics transmission of the disease using several different forms including the duration of disease, the duration of infectivity, the infection rate, the waning immunity using differential equation models as a simplified representation and making predictions about the number of infected and susceptible people over time.

A study by Chen and Hsieh [13] investigated the transmission dynamic modeling of dengue fever in Subtropical Taiwan by introducing temperature-dependent entomological parameters of *Aedes aegypti*. The vector-host transmission model was used to explore the temperature variation of pre-adult mosquito maturation, ovipositional rate, adult mosquito death rate, and virus incubation rate in the mosquito.

This paper presents an SEITR-SI model, (Susceptible, Exposed, Infected, Treatment, and Recovered) host-vector, *Aedes aegypti* mosquito to determine the dynamics of dengue fever in Africa as a result of high malaria prevalence and misdiagnosis the disease.

2. Materials and Methods

2.1 Methods

A SEITR-SI Host-Vector model for the occurrence of Dengue fever is formulated using ordinary differential equations. The host population N_H of the SEITR-SI Model is grouped into five classes namely, the Susceptible, Exposed, Infectious, Treatment and Recovery classes. The vector population N_V is also grouped into two classes namely, the Susceptible and Infectious. There is no recovered class in vector population since infection period in mosquito's ends with their death.

The study further used next generating matrix method to determine the expression for the basic reproductive number (R_0). The equilibrium stability of an Ordinary Differential Equations (ODE's) was determined by the sign of real part eigenvalues of the Jacobian matrix. Birkhoff and Rota's [14] theorem among others and functions were used to develop the mathematical modelling of Dengue Fever.

Model Assumptions

The following are the model assumptions:

- 1) The susceptible Host has no inherited immunity. However, once an Infected Host recovers, the person receives permanent immunity. Every member of the population mixes homogeneously
- 2) Age, sex, social status, race coupled with climatic conditions does not affect the probability of an individual being infected.
- 3) The death rate for the host population is balanced by a birth rate given by μ_H .
- 4) It is assumed that once an individual is infected, he or she became exposed to the environment before becoming infectious and finally,

The disease is transmitted in a closed environment. Hence the human population, N_H of individuals remains constant.

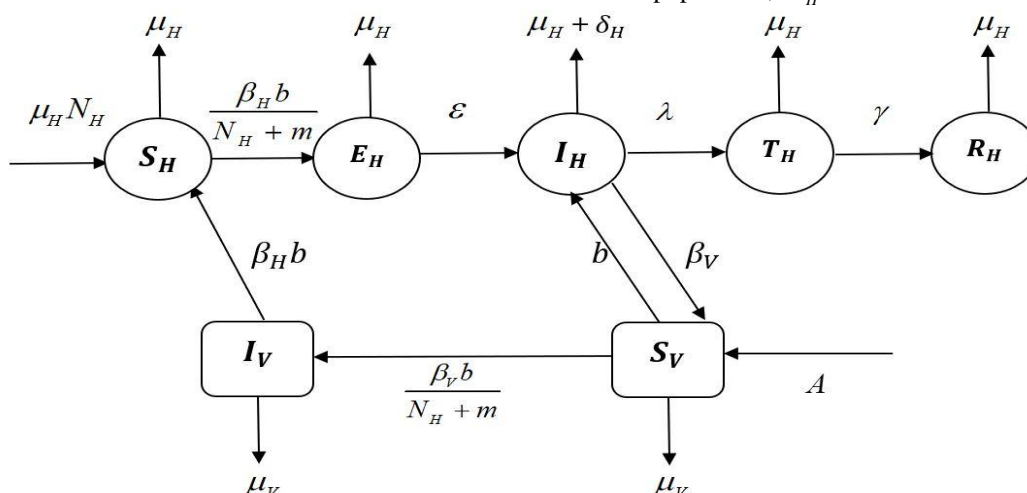


Figure 1: The Compartmental Model of Dengue Fever SEITR-SI

Model Formulation

The birth and death rate for the host population are assumed to be equal and represented as μ . Hence $\mu_H N_H$ will be the rate at which individuals are born into the susceptible class without any immunity and $\mu_H S_H$ is the rate at which they

leave the susceptible class through death. The rate at which the susceptible class changes is equal to the rate at which infections occur. This occurs when the disease is passed from an infective vector to a susceptible human. The number of susceptible-infective contacts is proportional to

the product of $S(t)$ and $I(t)$. Hence the rate of change in the susceptible individuals is given by Equation (1)

$$\frac{dS_H}{dt} = \mu_H(N_H - S_H) - \frac{\beta_H b}{N_H + m} S_H I_V \quad (1)$$

where, $\frac{\beta_H b}{N_H + m} S_H I_V$ is the rate of infection and negative

because the number of susceptible individual decreases. The rate at which individuals leave the susceptible population is equal to the rate at which they enter the exposed population. This increases the number of individuals in the exposed class.

Let εE be the rate at which an exposed individual becomes infectious. Then the rate of change of the exposed population is given by Equation (2)

$$\frac{dE_H}{dt} = \frac{\beta_H b}{N_H + m} S_H I_V - (\mu_H + \varepsilon) E_H \quad (2)$$

The rate of change of the infectious population is given by Equation (3)

$$\frac{dI_H}{dt} = \varepsilon E_H - (\mu_H + \lambda + \delta) I_H \quad (3)$$

Let γI be the rate at which an infected individual may recover. The rate of change of the Treated is given by Equation (4)

$$\frac{dT_H}{dt} = \lambda I_H - (\mu_H + \gamma) T_H \quad (4)$$

The rate of change of the recovered is given by Equation (5)

$$\frac{dR_H}{dt} = \gamma I_H - \mu_H R_H \quad (5)$$

This leads to the system equations in Equation (6)

$$\left. \begin{aligned} \frac{dS_H}{dt} &= \mu_H(N_H - S_H) - \frac{\beta_H b}{N_H + m} S_H I_V \\ \frac{dE_H}{dt} &= \frac{\beta_H b}{N_H + m} S_H I_V - (\mu_H + \varepsilon) E_H \\ \frac{dI_H}{dt} &= \varepsilon E_H - (\mu_H + \lambda + \delta) I_H \\ \frac{dT_H}{dt} &= \lambda I_H - (\mu_H + \gamma) T_H \\ \frac{dR_H}{dt} &= \gamma I_H - \mu_H R_H \\ \frac{dS_V}{dt} &= A - \frac{\beta_V b}{N_H + m} S_V I_H - \mu_V S_V \\ \frac{dI_V}{dt} &= \frac{\beta_V b}{N_H + m} S_V I_H - \mu_V I_V \end{aligned} \right\} \quad (6)$$

where μ_H and μ_V are the per capita mortality rates of humans and mosquito vector, respectively, b , the biting rate (i.e., average number of bites per mosquito per day); m , the number of alternative hosts available as the blood source; A , a constant recruitment rate for the vector; and β_H and β_V , the transmission probabilities from the vector to human and human to vector respectively. Again, the total host population is expressed as $N_H = S_H + E_H + I_H + T_H + R_H$ and the total vector population is given as $N_V = S_V + I_V$

Table I gives the description of the parameters used in the model and their symbols.

Table I: Description of Parameters

Symbols	Description
A	A constant recruitment rate for the vector
β_H	The transmission probabilities from the vector to human
β_V	The transmission probabilities from the human to vector
b	The biting rate (i.e., average number of bites per mosquito per day)
μ_H	The per capita mortality rates of humans
μ_V	The per capita mortality rates of mosquitoes
m	The number of alternative hosts available as the blood source
δ	The disease-induced death rate.
γ	The recovery rate of humans
ε	The rate at which the exposed individuals become infective or infectious.
λ	The rate at which the infectious individuals are treated.

Invariant Region

It is assumed that all the model parameters are positive for all $t \geq 0$ and the initial conditions of the model are stated and analysed in a suitable feasible region as follows:

$$S_H(0) \geq 0, E_H(0) \geq 0, I_H(0) \geq 0, T_H(0) \geq 0, R_H(0) \geq 0, S_V(0) \geq 0 \text{ and } I_V(0) \geq 0$$

Positivity of Solution of the Model

Theorem 1: Given $S_H(0) \geq 0, E_H(0) \geq 0, I_H(0) \geq 0, T_H(0) \geq 0, R_H(0) \geq 0, S_V(0) \geq 0, I_V(0) \geq 0$ the solutions $[S_H(t), E_H(t), I_H(t), T_H(t), R_H(t), S_V(t)$ and $I_V(t)]$ on the model are positively invariant for all $t > 0$.

Proof

Let $t_1 = \sup\{t > 0 \mid S_H > 0, E_H > 0, I_H > 0, T_H > 0, R_H > 0, S_V > 0 \text{ and } I_V > 0\} \in [0, t]$

From Equation (6),

$$\frac{dS_H(t)}{dt} = \mu_H N_H - \mu_H S_H(t) - \frac{\beta_H b S_H(t) I_V}{N_H + m}$$

This is reformulated as Equation (7)

$$\frac{dS_H(t)}{dt} = \mu_H N_H - \left(\mu_H + \frac{\beta_H b I_V}{N_H + m} \right) S_H(t) \quad (7)$$

Let $\rho = \frac{\beta_H b}{N_H + m} > 0$

$$\frac{dS_H(t)}{dt} + (\mu_H + \rho I_V) S_H(t) = \mu_H N_H \quad (8)$$

From Equation (8) one obtains the integrating factor μ as in Equation (9)

$$\mu = e^{\int_0^t (\mu_H + \rho I_V) dt} = e^{(\mu_H + \rho I_V)t} \quad (9)$$

Multiplying through by $I.F$ and rearranging, one obtains the integral form as Equation (12)

$$\left(e^{(\mu_H + \rho I_V)t} \right) \frac{dS_H(t)}{dt} + \left(e^{(\mu_H + \rho I_V)t} \right) (\mu_H + \rho I_V) S_H(t) = \left(e^{(\mu_H + \rho I_V)t} \right) \mu_H N_H \quad (10)$$

$$\frac{d}{dt} \left(S_H(t) e^{(\mu_H + \rho I_V)t} \right) = \left(e^{(\mu_H + \rho I_V)t} \right) \mu_H N_H \quad (11)$$

$$\int_0^t \frac{d}{dt} (S_H(t) e^{(\mu_H + \rho I_V)t}) dt = \int_0^t (e^{(\mu_H + \rho I_V)t}) \mu_H N_H dt \quad (12)$$

The solution eventually comes out as Equation (13)

$$S_H(t) e^{(\mu_H + \rho I_V)t} = \mu_H N_H \int_0^t (e^{(\mu_H + \rho I_V)t}) dt \quad (13)$$

$$S_H(t) (e^{(\mu_H + \rho I_V)t}) = \frac{\mu_H N_H}{\mu_H + \rho I_V} (e^{(\mu_H + \rho I_V)t}) + S_H(0) \quad (14)$$

$$S_H(t) = \frac{\mu_H N_H}{\mu_H + \rho I_V} + S_H(0) (e^{-(\mu_H + \rho I_V)t}) \quad (15)$$

$$S_H(t) = \frac{\mu_H N_H}{\mu_H + \frac{\beta_H b}{N_H + m} I_V} + S_H(0) (e^{-(\mu_H + \rho I_V)t}) \quad (16)$$

Therefore, $S_H(t) > 0$.

Similarly, it can be shown that $S_H > 0, E_H > 0, I_H > 0, T_H > 0, R_H > 0, S_V > 0$ and $I_V > 0$ for all $t > 0$

Hence proved.

Identification of the Biological Interest of the Model

Let

$$D_- H = \{S_H, E_H, I_H, T_H, R_H\} \in \square_+^s : S_H + E_H + I_H + T_H + R_H \leq N_H$$

$$D_V = \left\{ (S_H, I_H) \in \square_+^2 : S_H + I_H \leq N_V \text{ or } \frac{A}{\mu_V} \right\}$$

$$\Omega = \{S_H, E_H, I_H, T_H, R_H, S_H, I_H\}$$

Stability Analysis of the Equilibrium Points or Steady States

In order to find the steady state, all the derivatives are set to zero. That is, the equilibrium points can be obtained by equating the rate of changes to zero;

$$\frac{dS_H}{dt} = \frac{dE_H}{dt} = \frac{dI_H}{dt} = \frac{dT_H}{dt} = \frac{dR_H}{dt} = \frac{dS_V}{dt} = \frac{dI_V}{dt} = 0 \quad (17)$$

Estimating the steady state conditions, the following two steady states were obtained.

Disease-free equilibrium

The disease-free equilibrium point is where there are no infections in the population. Thus, equating the system of equations to zero and solving to obtain the disease-free steady state as;

$$\{S_H, E_H, I_H, T_H, R_H, S_V, I_V\} = \left\{ N_H, 0, 0, 0, 0, \frac{A}{\mu_V}, 0 \right\}.$$

Disease-endemic equilibrium

The disease-endemic equilibrium of $\{S_H^*, E_H^*, I_H^*, T_H^*, R_H^*, S_V^*, I_V^*\}$ with positive components is obtained as follows:

$$S_H^* = \frac{W_1}{\varepsilon \beta_V (Ab \beta_H + m \mu_H \mu_V + N_H \mu_H \mu_V) b}$$

$$E_H^* = \frac{W_2}{\left(\varepsilon b (Ab \varepsilon \beta_H + Ab \beta_H \mu_H + m \varepsilon \mu_H \mu_V + m \mu_H^2 \mu_V) + \varepsilon N_H \mu_H \mu_V \right) \beta_V}$$

$$I_H^* = \frac{W_2}{W_3}, T_H^* = \frac{\lambda W_2}{(\gamma + \mu_H) W_3}, R_H^* = \frac{\gamma W_2 \lambda}{\mu_H (\gamma + \mu_H) W_3},$$

$$S_V^* = \frac{\mu_V (N_H + m) W_4}{b \beta_H W_1}, I_V^* = \frac{(N_H + m) W_2}{b \beta_H W_1}$$

where,

$$W_1 = \left[\begin{aligned} &(b \varepsilon N_H \beta_V \mu_H + \delta m \varepsilon \mu_V + \delta m \mu_H \mu_V + \delta \varepsilon N_H \mu_V) \\ &+ \delta N_H \mu_H \mu_V + \lambda m \varepsilon \mu_V + \lambda m \mu_H \mu_V + \lambda \varepsilon N_H \mu_V \\ &+ \lambda N_H \mu_H \mu_V + m \varepsilon \mu_H \mu_V + m \mu_H^2 \mu_V + \varepsilon N_H \mu_H \mu_V \\ &+ N_H \mu_H^2 \mu_V \end{aligned} \right] \mu_V (N_H + m)$$

$$W_2 = \left[\begin{aligned} &\mu_H (Ab^2 \varepsilon N_H \beta_H \beta_V - \delta m^2 \varepsilon \mu_V^2 - \delta m^2 \mu_H \mu_V^2) \\ &- 2 \delta m \varepsilon N_H \mu_V^2 - 2 \delta m N_H \mu_H \mu_V^2 - \delta \varepsilon N_H^2 \mu_V^2 \\ &- \delta N_H^2 \mu_H \mu_V^2 - \lambda m^2 \varepsilon \mu_V^2 - \lambda m^2 \mu_H \mu_V^2 - 2 \lambda m \varepsilon N_H \mu_V^2 \\ &- 2 \lambda m N_H \mu_H \mu_V^2 - \lambda \varepsilon N_H^2 \mu_V^2 - \lambda N_H^2 \mu_H \mu_V^2 \\ &- m^2 \varepsilon \mu_H \mu_V^2 - m^2 \mu_H^2 \mu_V^2 - 2 m \varepsilon N_H \mu_H \mu_V^2 - 2 m N_H \mu_H^2 \mu_V^2 \\ &- \varepsilon N_H^2 \mu_H \mu_V^2 - N_H^2 \mu_H^2 \mu_V^2 \end{aligned} \right]$$

$$W_3 = \left[\begin{aligned} &\beta_V (Ab \delta \varepsilon \beta_H + Ab \delta \beta_H \mu_H + Ab \lambda \varepsilon \beta_H + Ab \lambda \beta_H \mu_H) \\ &+ Ab \varepsilon \beta_H \mu_H + Ab \beta_H \mu_H^2 + \delta m \varepsilon \mu_H \mu_V + \delta m \mu_H^2 \mu_V \\ &+ \delta \varepsilon N_H \mu_H \mu_V + \delta N_H \mu_H^2 \mu_V + \lambda m \varepsilon \mu_H \mu_V + \lambda m \mu_H^2 \mu_V \\ &+ \lambda \varepsilon N_H \mu_H \mu_V + \lambda N_H \mu_H^2 \mu_V + m \varepsilon \mu_H^2 \mu_V + m \mu_H^3 \mu_V \end{aligned} \right]$$

$$W_4 = \left[\begin{aligned} &Ab \delta m \varepsilon \beta_H + Ab \delta m \beta_H \mu_H + Ab \delta \varepsilon N_H \beta_H \\ &+ Ab \delta N_H \beta_H \mu_H + Ab \lambda m \varepsilon \beta_H + Ab \lambda m \beta_H \mu_H \\ &+ Ab \lambda \varepsilon N_H \beta_H + Ab \lambda N_H \beta_H \mu_H + Ab m \varepsilon \beta_H \mu_H \\ &+ Ab m \beta_H \mu_H^2 + Ab \varepsilon N_H \beta_H \mu_H + Ab N_H \beta_H \mu_H^2 \\ &+ \delta m^2 \varepsilon \mu_H \mu_V + \delta m^2 \mu_H^2 \mu_V + 2 \delta m \varepsilon N_H \mu_H \mu_V \\ &+ 2 \delta m N_H \mu_H^2 \mu_V + \delta \varepsilon N_H^2 \mu_H \mu_V + \delta N_H^2 \mu_H^2 \mu_V \\ &+ \lambda m^2 \varepsilon \mu_H \mu_V + \lambda m^2 \mu_H^2 \mu_V + 2 \lambda m \varepsilon N_H \mu_H \mu_V \\ &+ 2 \lambda m N_H \mu_H^2 \mu_V + \lambda \varepsilon N_H^2 \mu_H \mu_V + \lambda N_H^2 \mu_H^2 \mu_V \\ &+ m^2 \varepsilon \mu_H^2 \mu_V + m^2 \mu_H^3 \mu_V + 2 m \varepsilon N_H \mu_H^2 \mu_V \\ &+ 2 m N_H \mu_H^3 \mu_V + \varepsilon N_H^2 \mu_H^2 \mu_V + N_H^2 \mu_H^3 \mu_V \end{aligned} \right]$$

To determine the stability of the model, the steady state of the system was evaluated. In solving the equations, we considered only one state at a time. The disease-free steady state, where $I_H = I_V = 0$

Stability of the Equilibrium Points

The stability of the system is determined by linearizing the systems of equations about the equilibrium points using the Jacobian matrix. The Jacobian matrix is given by Equation (18)

Local Stability of the Disease-Free Equilibrium

For the disease-free equilibrium, we evaluate the Jacobian matrix at the equilibrium points

$$w_0 \{S_H, E_H, I_H, T_H, R_H, S_V, I_V\} = \left\{ N_H, 0, 0, 0, 0, \frac{A}{\mu_V}, 0 \right\} \text{ with}$$

the theorem below.

Theorem 1: The disease-free equilibrium point $w_0 \{S_H, E_H, I_H, T_H, R_H, S_V, I_V\}$ is locally asymptotically

stable if all the eigenvalues of the Jacobian matrix $\lambda_i < 0$, or it is unstable if $\lambda_i > 0$.

Proof: The Jacobian matrix evaluated at the disease-free equilibrium is given by Equation 18

$$J = \begin{pmatrix} -\mu_H & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & -(\mu_H + \varepsilon) & 0 & 0 & 0 & 0 & 0 \\ 0 & \varepsilon & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -(\gamma + \mu_H) & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_H & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_V & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix} \quad (18)$$

The characteristic equation resulting from the Jacobian matrix is

$$J = \begin{pmatrix} -\mu_H - \frac{\beta_H b I_V}{N_H + m} & 0 & 0 & 0 & 0 & 0 & -\frac{\beta_H b S_H}{N_H + m} \\ \frac{\beta_H b I_V}{N_H + m} & -(\mu_H + \varepsilon) & 0 & 0 & 0 & 0 & \frac{\beta_H b S_H}{N_H + m} \\ 0 & \varepsilon & -(\mu_H + \lambda + \delta) & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda & -(\mu_H + \gamma) & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & -\mu_H & 0 & 0 \\ 0 & 0 & -\frac{\beta_V b S_V}{N_H + m} & 0 & 0 & -\frac{\beta_V b I_H}{N_H + m} - \mu_V & 0 \\ 0 & 0 & \frac{\beta_V b S_V}{N_H + m} & 0 & 0 & \frac{\beta_V b I_H}{N_H + m} & -\mu_V \end{pmatrix} \quad (19)$$

The eigenvalues from the characteristic equation given by Equation (20) are $\lambda_1 = \lambda_2 = 0$, $\lambda_3 = \lambda_4 = -\mu_H$, $\lambda_5 = -\mu_H - \varepsilon$, $\lambda_6 = -\mu_V$ and $\lambda_7 = -\mu_H - \gamma$.

The eigenvalues are all negatives except $\lambda_1 = 0$ and $\lambda_2 = 0$

Theorem 2: The disease-free equilibrium is locally asymptotically stable for $R_0 < 1$ and unstable for $R_0 > 1$.

The Basic Reproduction Number (R_0)

The basic reproductive number (R_0) is the average number of new cases of an infection caused by one typical infected individual in a population consisting of susceptible only [15]. The model Equation (6), always has a disease-free equilibrium

$$\{S_H, E_H, I_H, T_H, R_H, S_V, I_V\} = \left\{ N_H, 0, 0, 0, 0, \frac{A}{\mu_V}, 0 \right\}.$$

Let $X = (S_H, E_H, I_H, T_H, R_H, S_V, I_V)^T$. Then the model (6)

$$\text{can be written as } \frac{dX}{dt} = F(X) - v(X)$$

Now applying the Next Generation Operator approach gives the transmission (\mathcal{F}) and transition (\mathcal{V}) states of the model as Equation (21) and (22):

$$\lambda^2 (\lambda^5 + a_4 \lambda^4 + a_3 \lambda^3 + a_2 \lambda^2 + a_1 \lambda + a_0) = 0 \quad (20)$$

where, $a_0 = \mu_H^2 (\mu_H + \varepsilon) (\gamma + \mu_H) \mu_V$

$$a_1 = \left(\gamma \varepsilon \mu_H^2 + 2 \gamma \varepsilon \mu_H \mu_V + \gamma \mu_H^3 + 3 \gamma \mu_H^2 \mu_V + \varepsilon \mu_H^3 + 3 \varepsilon \mu_H^2 \mu_V + \mu_H^4 + 4 \mu_H^3 \mu_V \right)$$

$$a_2 = \left(2 \gamma \varepsilon \mu_H + \gamma \varepsilon \mu_V + 3 \gamma \mu_H^2 + 3 \gamma \mu_H \mu_V + 3 \varepsilon \mu_H^2 + 3 \varepsilon \mu_H \mu_V + 4 \mu_H^3 + 6 \mu_H^2 \mu_V \right)$$

$$a_3 = (\gamma \varepsilon + 3 \gamma \mu_H + \gamma \mu_V + 3 \varepsilon \gamma \mu_H + \varepsilon \mu_V + 6 \mu_H^2 + 4 \mu_H \mu_V)$$

$$a_4 = (4 \mu_H + \varepsilon + \gamma + \mu_V)$$

$$F(x) = \begin{pmatrix} \frac{\beta_H b}{N_H + m} S_H \\ 0 \\ \frac{\beta_V b}{N_H + m} S_V I_H \end{pmatrix} \quad (21)$$

$$v(x) = \begin{pmatrix} (\mu_H + \varepsilon) E_H \\ -\varepsilon E_H + (\mu_H + \lambda + \delta) I_H \\ \mu_V I_V \end{pmatrix} \quad (22)$$

The Jacobian matrix of the transmission (\mathcal{F}) and transition (\mathcal{V}) states of the model evaluated at the disease-free equilibrium are as follows:

$$F = \begin{pmatrix} 0 & 0 & \frac{\beta_H b}{N_H + m} S_H \\ 0 & 0 & 0 \\ 0 & \frac{\beta_H b}{N_H + m} S_H & 0 \end{pmatrix} \quad (23)$$

$$V = \begin{pmatrix} (\mu_H + \varepsilon) & 0 & 0 \\ -\varepsilon & (\mu_H + \lambda + \delta) & 0 \\ 0 & 0 & \mu_V \end{pmatrix} \quad (24)$$

This inverse of the Equation (24) is obtained as Equation (25)

$$V^{-1} = \begin{pmatrix} \frac{1}{(\mu_H + \varepsilon)} & 0 & 0 \\ \frac{\varepsilon}{(\mu_H + \varepsilon)(\mu_H + \lambda + \delta)} & \frac{1}{(\mu_H + \lambda + \delta)} & 0 \\ 0 & 0 & \frac{1}{\mu_V} \end{pmatrix} \quad (25)$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_H b}{N_H + m} S_H \\ 0 & 0 & 0 \\ 0 & \frac{\beta_H b}{N_H + m} S_H & 0 \end{pmatrix} \begin{pmatrix} \frac{1}{(\mu_H + \varepsilon)} & 0 & 0 \\ \frac{\varepsilon}{(\mu_H + \varepsilon)(\mu_H + \lambda + \delta)} & \frac{1}{(\mu_H + \lambda + \delta)} & 0 \\ 0 & 0 & \frac{1}{\mu_V} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \frac{\beta_H b N_H}{(N_H + m)\mu_V} \\ 0 & 0 & 0 \\ \frac{\beta_H b N_H \varepsilon}{(N_H + m)\mu_V (\mu_H + \varepsilon)(\mu_H + \lambda + \delta)} & \frac{\beta_H b N_H}{(N_H + m)\mu_V (\mu_H + \lambda + \delta)} & 0 \end{pmatrix} \quad (26)$$

The Basic reproduction number (R_0) is calculated as the Spectral radius (ρ) which is also known as the dominant eigenvalue of FV^{-1} , thus

$$R_0 = \rho(FV^{-1}) \quad (27)$$

$$R_0 = \frac{\sqrt{(\delta\varepsilon + \delta\mu_H + \varepsilon\lambda + \varepsilon\mu_H + \lambda\mu_H + \mu_H^2)\beta_V A\varepsilon\beta_H N_H b}}{(\delta\varepsilon + \delta\mu_H + \varepsilon\lambda + \varepsilon\mu_H + \lambda\mu_H + \mu_H^2)(N_H + m)\mu_V} \quad (28)$$

This can further be simplified as,

$$R_0 = \frac{\sqrt{(\mu_H + \lambda + \delta)(\mu_H + \varepsilon)\beta_V A\varepsilon\beta_H N_H b}}{(\mu_H + \lambda + \delta)(\mu_H + \varepsilon)(N_H + m)\mu_V} \quad (29)$$

Proof of the Stability of the Disease-Free Equilibrium

We now investigate the linear stability of the Disease-free equilibrium point, by substituting the parameter values in Table 4 above into Equation (6). When $R_0 = 1.854008341 > 1$, the eigenvalues corresponding to the infectious free equilibrium are

$$\lambda = \begin{pmatrix} -0.00500 \\ -0.00500 \\ -0.58221 \\ -0.100 \\ -0.6168 + 0.35699i \\ -0.61681 - 0.35699i \\ 0.122625 \end{pmatrix} \quad (32)$$

Because one of eigenvalues in Equation (32) is positive, the disease-free equilibrium is a saddle point therefore unstable. This implies that the presence of a person infected will eventually result in an outbreak of the disease.

When the parameters are varied with $R_0 < 1$, all the eigenvalues corresponding to the infectious free equilibrium become negative.

$$\lambda = \begin{pmatrix} -0.00550 \\ -0.00550 \\ -0.58271566490 \\ -0.25000 \\ -0.4222262091 + 0.20650392i \\ -0.4222262091 - 0.20650392i \\ -0.042547581 \end{pmatrix}$$

Numerical simulations of the model

Numerical simulations of the model are done to determine the effect the effect of the model parameters on the spread of the disease. The assumed parameters used are shown in TableII.

Table II: Parameter Values used in the Model

Parameters	Values/ Range	Reference	Units
A	5000	[16]	
β_H	0.1 - 0.9	[17]	
β_V	0.4 - 0.9	[17]	
b	0 - 1	[17]	day ⁻¹
μ_H	0 - 0.005	[17]	day ⁻¹
μ_V	0.025 - 0.3	[17]	day ⁻¹
m	0 - 100	[16]	
λ	0.3 - 1	[16]	day ⁻¹
γ	0.167 - 0.333	[16]	day ⁻¹
ε	0 - 1	[18]	day ⁻¹
δ	0.001	[16]	day ⁻¹
N_V	0-10000	[17]	

The basic reproductive number R_0 is calculated by substituting the parameter values in Table II into Equation (29), we have,

$$\sqrt{\frac{(0.005 + 0.5 + 0.001)(0.005 + 0.5)(500)(0.8)(10000)}{*(0.7)(0.4)(0.5)}} \quad (30)$$

$$R_0 = 2.621963740 \quad (31)$$

Since the reproductive number $R_0 > 1$, it signifies that an outbreak of dengue will result in an epidemic.

When basic reproduction number is less than one, the disease-free equilibrium point is asymptotically stable, therefore an outbreak wouldn't result in an epidemic. On the other hand, when the basic reproduction number is greater than one, the equilibrium point is unstable, therefore an outbreak would result in an epidemic. Looking at the basic Reproduction number,

$$R_0 = \frac{\sqrt{(\mu_H + \lambda + \delta)(\mu_H + \varepsilon)\beta_V A \varepsilon \beta_H N_H b}}{(\mu_H + \lambda + \delta)(\mu_H + \varepsilon)(N_H + m)\mu_V}$$

one key parameter that influences the R_0 is the death rate of the vector population (μ_V).

Table III: Parameter Values For Stability

Parameters	Values	Units
A	1000	
β_H	0.7	
β_V	0.4	
b	0.8	day ⁻¹
μ_H	0.005	day ⁻¹
μ_V	0.1	day ⁻¹
m	100	
λ	0.5	day ⁻¹
γ	0.167	day ⁻¹
ε	0.5	day ⁻¹
δ	0.001	day ⁻¹
N_V	10000	

3. Results and Discussion

In these simulations we use the parameter values given in Table II, for the model equations to depict the dynamics of the compartments during an outbreak. The initial proportions for $S_H, E_H, I_H, T_H, R_H, S_V, I_V$ is assumed to be 8000, 1000, 1000, 0, 0, 6000, and 4000 respectively.

It was observed that, most of the parameter values were not readily available, so some were estimated. However, with initial conditions taken at time zero (0) and the final time was considered as 500, 500, 500, 500, 520, 2000, 2000, 2000, 7000, 8000 and 10000 [19]. The results of the simulation study are presented in Fig. 2.

The simulated result in Fig. 2 shows that the susceptible human population decreases steadily at first but begins to rise and fall but eventually maintains equilibrium with time. The exposed human population in Fig.3 increases sharply and decreases steadily with time while the Infectious human population increases at certain point in time decreases until it becomes asymptotically stable. From Fig. 5, the simulation study shows that the Treated human population increases from 0 to approximately 3500 sharply, then decreases steadily until it attains equilibrium. Moreover, the simulated result in Fig. 6 shows that the recovered human population increases to approximately

9000, then begins to decrease to approximately 8000 until it attains equilibrium with time.

Again Fig. 8, exhibits a decline in the susceptible vector population and then increases to approximately 9500 and then eventually maintains some equilibrium with time and finally, the simulated result in Fig. 10 shows the relationship between both hosts (human) and vector (mosquito) population.

The positivity of solution to the model was proved as shown in Equation (4) to (17). The Basic Reproduction Number R_0 was computed from the deterministic model that was developed in Equation (4). The model's equilibria were determined and conditions for the equilibria were also established with their stabilities investigated in terms of the classic threshold R_0 . The disease-free equilibrium (DFE) was found to be locally asymptotically stable for $R_0 < 1$. as shown in Equation (37). The endemic equilibrium E_{ee} of the model was also found to be locally asymptotically stable for $R_0 > 1$. However, it was very difficult to deal with the stability of the endemic equilibrium analytically due to the nature of transmission model developed. Applying the different initial conditions for the simulation, the orbits show the same points as time increases.

Table IV: Parameter Values for Simulation

Parameters	Values/ Range	Units
A	500	
β_H	0.7	
β_V	0.4	
b	0.8	day ⁻¹
μ_H	0.005	day ⁻¹
μ_V	0.05	day ⁻¹
m	100	
λ	0.5	day ⁻¹
γ	0.167	day ⁻¹
ε	0.5	day ⁻¹
δ	0.001	day ⁻¹
N_V	10000	day ⁻¹

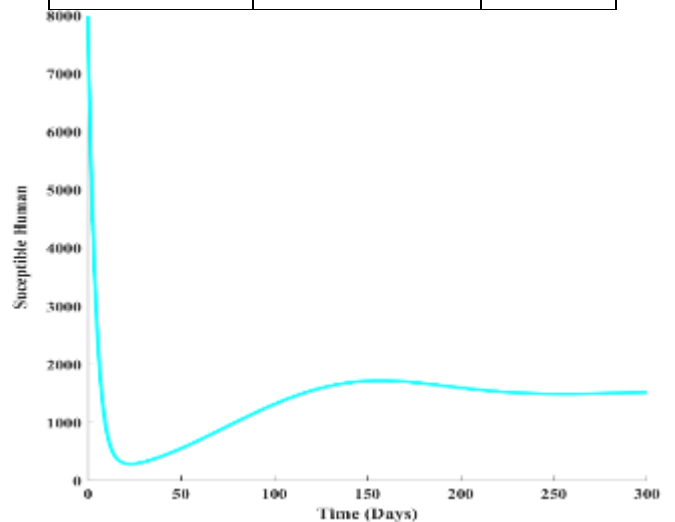


Figure 2: Dynamics of Susceptible Host

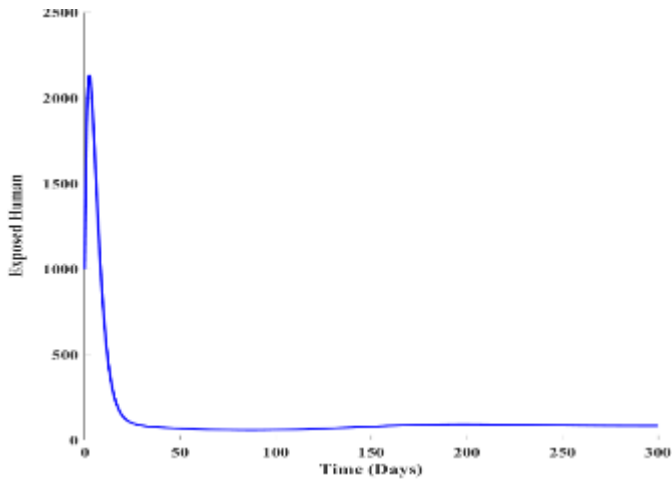


Figure 3: Dynamics of Exposed Host

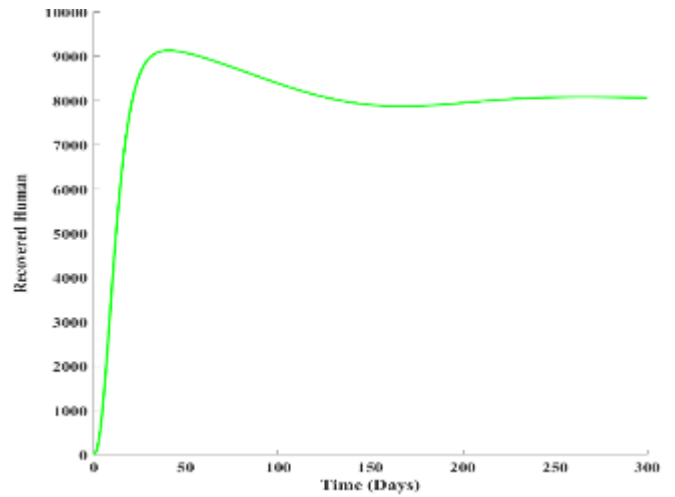


Figure 6: Dynamics of Recovered Hosts

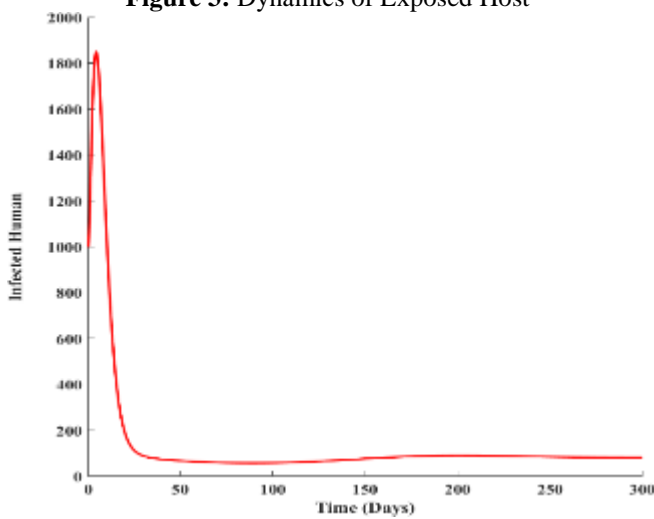


Figure 4: Dynamics of Infected Hosts

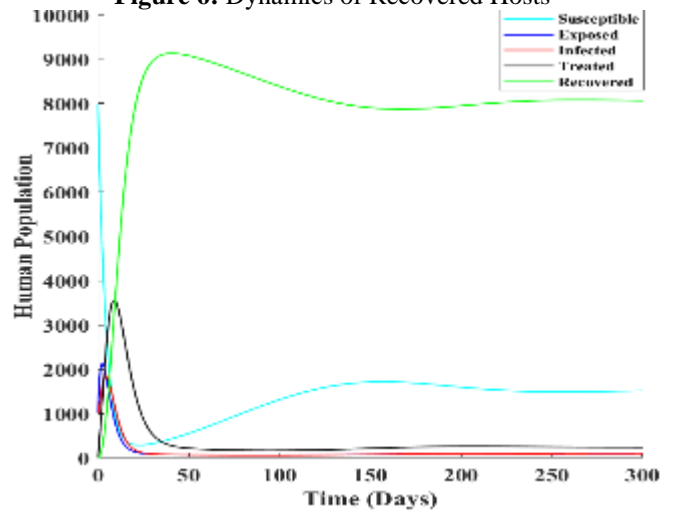


Figure 7: Dynamics of Hosts Populations

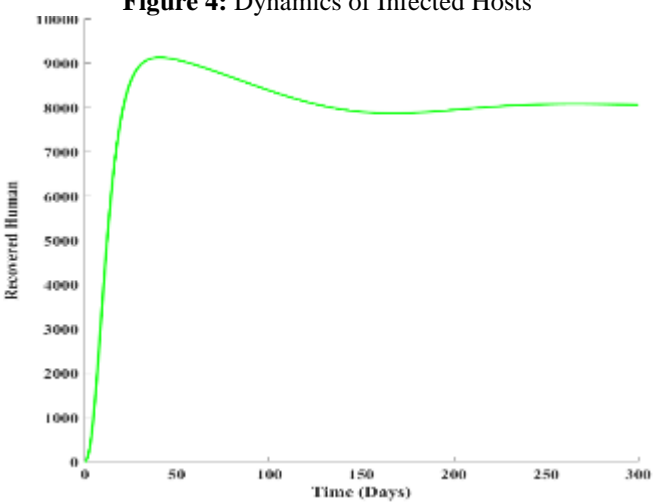


Figure 5: Dynamics of Treated Hosts

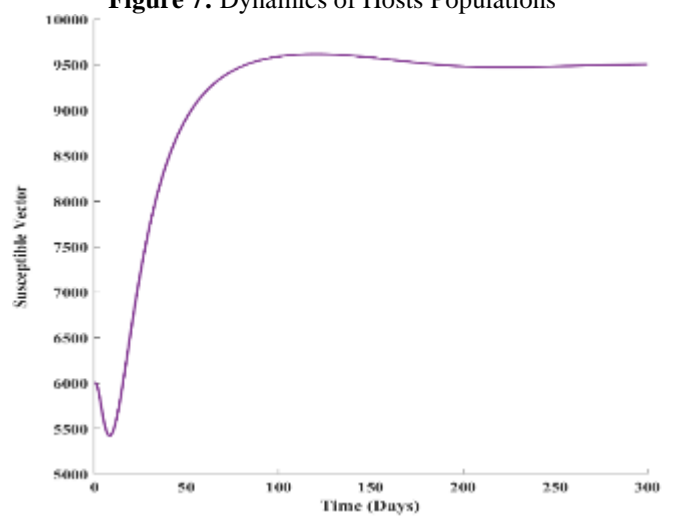


Figure 8: Dynamics of Susceptible Vectors

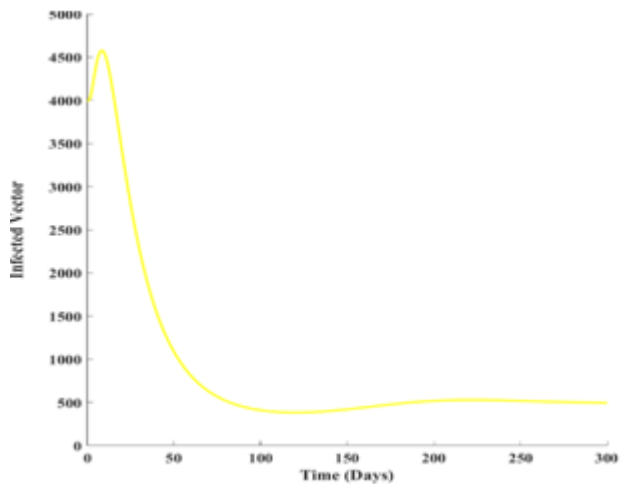


Figure 9: Dynamics of Infected Vectors

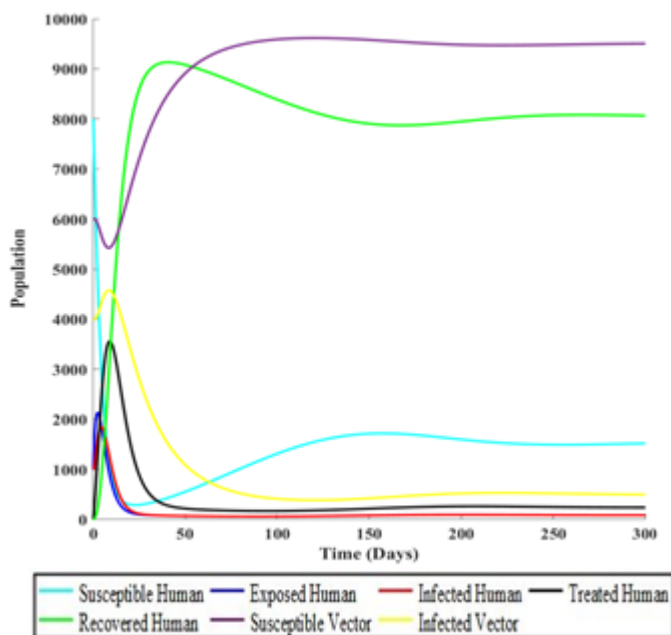


Figure 10: Dynamics of Population

4. Conclusions

A Mathematical model of dengue Fever has been formulated using nonlinear differential equations and Next Generation method. The criteria for stability states of the various equilibrium points have been established while the simulations of the model were carried out using different parameter values from literature. The simulated dynamics of dengue fever was discussed and illustration as shown in Figs. 2 to 10. The basic reproduction number for modelling DF was derived and estimated. The reproduction number estimated was greater than one, $R_0 = 2.62$ and can easily be brought to a number less than one by incorporating control and preventive measures that will minimize the spread of the disease. The study concludes that the emergence of new cases of DF in some Africa regions and Ghana call for proper diagnosis of malaria using adequate diagnosis and treatment to avoid transmission as a result of misdiagnosis of febrile illnesses as malaria. The paper suggests an intensive education for Healthcare officials on how to curb the disease through adequate malaria treatment and proper laboratory diagnosis to avoid misdiagnosis. The paper

further suggests that, to prevent the spread of the disease, educational campaign geared towards sensitizing the masses on the danger of dengue fever to help keep to the precautionary measures, such as; keeping the environments clean, avoiding wetlands or stagnant water, mass insecticide spraying to control mosquito breeding habitats.

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