A Mathematical Model of the Transmission Dynamics of Chikungunya Fever

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***Abstract*- The study presented a deterministic mathematical model for investigating the dynamics of Chikungunya virus transmission into a human population. A system of nonlinear differential equations was used to formulate the mathematical model. The threshold value (R_0) for the disease was determined using the next generation matrix approach.**

As well as the numerical simulations, the stability analysis of the disease-free and resistant equilibrium solutions was also carried out. From the analysis $R_0 < 1$ and the stability also revealed that the disease-free balance is asymptotically stable locally and globally. And that of endemic equilibrium is asymptotically stable at the local level.

***Index Terms*-** Chikungunya, Disease-free equilibrium, Gershogrin Theorem,

I. INTRODUCTION

Chikungunya fever (CKIKV) is an arboviral disease which is normally caused by transmitted by the parasites genus aplhaviral. This disease was first encountered in Tanzania in 1953 (Weaver and Lecuit, 2015). The main causative agent of this disease is the Asian tiger mosquito (Aedes Albopictus). The particular type of mosquito is found in the tropics and some parts of Africa((Dubrulle et al., 2009; Tsetsarkin et al., 2011). An individual begins to show signs of the disease within two to four days after exposure. These signs include fever which may sometimes prolong between two to seven days. There are also joint pains as a result of the infection which sometimes can even last a week, months or years. The disease has a minimal mortality rate which is a little less than 0.001 per cent. There is also severe complications sometimes among the aged and infants (Anon, 2014).

Researchers have over the years tried to obtain information about the disease outbreak. For instance, Mavalankar et al, (2008) studied the mortality rate of chikungunya in India. Staple et al. (2009) modelled the epidemiological and global expansion of chikungunya fever focusing on the clinical features and the laboratory testing for the disease. Ramchum et al. (2010) also performed the excess mortality profile during the 2006 chikungunya outbreak in Mauritius. Modelling of the spread of the disease in the Reunion Island taking into consideration control measure and the latent period of the disease was considered by Dumont and Chiroleu (2010). By exploring optimal control strategies, Moulay et al., (2011) modelled and analysed a similar model on the population dynamics of mosquitoes. In order to determine the effect of the disease using reported cases, Yakob and Clements (2013) conducted a study, centred on the relationship between asymptomatic and symptomatic human cases. Also, Naowarat et al., (2011) developed a deterministic model representing the susceptible Infectious Recovered for Host and Susceptible Infectious for the transmission of chikungunya fever.

Furthermore, by incorporating the transmission dynamics of the disease using mathematical models, the effects of the disease were simulated. This enables Poletti et al. (2011) to determine how various intervention measures affect the outbreak of the disease and the possibility of future outbreaks, especially in temperate regions. Reiskind et al. (2010) compared the two causal agents Aedes albopitus and Ades aegypti which are the primary and secondary vectors respectively by modeling the chikungunya exposure and adult longevity in Aedes albopictus and Aedes aegypti. To limit the chikungunya transmission or infection in the absence of any effective treatment measures yet discovered, Alkama et al. (2015) presented an optimal control model for the disease transmission.

Most of the studies modelled the disease with control. Since the disease is emerging and gradually spreading throughout the world, there is a need to know about the transmission dynamics when there are no control measures. This would provide insight into the actual transmission pattern of the disease. Therefore, this research aims to model and analyze the transmission dynamics of chikungunya without control measures.

II. METHODOLOGY

A. Model Formulation

The Susceptible Infection and Recovered model of the transmission of chikungunya disease in human and Susceptible Exposed Infectious model of the vector population were considered in the study. This takes into consideration the transmission of chikungunya between human and vector populations since there must be an interaction between them before the disease can be spread. The human population $N_h(t)$ was partitioned into three separate epidemiological compartments which are the susceptible humans $S_h(t)$, infectious humans $I_h(t)$ and Recovered humans $R_h(t)$. The disease transmission starts when the susceptible human is bitten by an infected Asian tiger mosquito. The bitten human has a high probability of been infected. Therefore, the infected human transitions from the susceptible compartment $S_h(t)$ to the infectious compartment $I_h(t)$ and can also infect the susceptible mosquitoes when they are bitten again. Over a period, there are movement of individuals from the infectious compartment to the recovered compartment $R_h(t)$. An individual becomes immune to the pathogen for life after recovery from the disease.

Furthermore, the adult vector population $N_v(t)$ was also partitions into three compartments namely; Susceptible vector $S_v(t)$, Infectious vector $I_v(t)$, and exposed vector $E_v(t)$. When a susceptible vector bites an infectious human and becomes infected, this vector is exposed to the virus and therefore joins the exposed class $E_v(t)$ as a result of the latent period of the virus. After the latent period, the vector then becomes infectious and transitions to the infectious class $I_v(t)$.

B. Compartmental Model

The SIRSEI compartmental model is considered for this study.

Model Assumptions

The following are the model assumptions:

i. Host recover from the disease permanently and become immune for life
ii. The exposed class of the disease in humans is excluded because the incubation period (latent) of the disease is short.
iii. The omission of the removal class of the vector is because the vector does not die as a result of chikungunya.
iv. Natural death is constant in all groups.
v. It assumed that both the vector and human population are confined in a specific geographical area.

The diagrammatic representation of the various compartments under consideration in this study is indicated in figure 1.

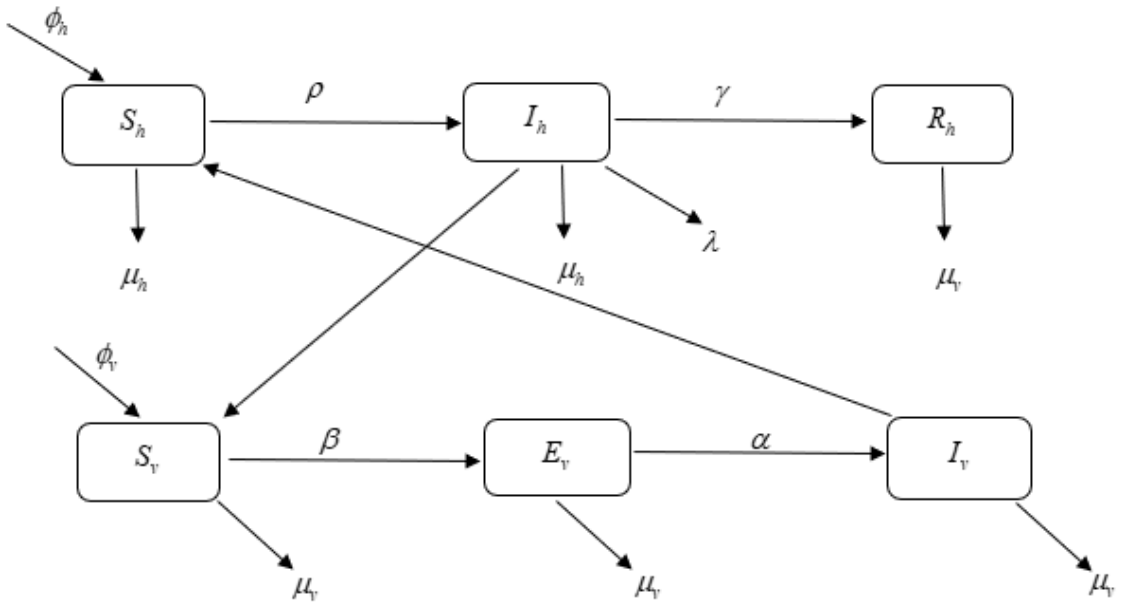

Figure 1 Compartment Model of the transmission of Chikungunya Fever

C. Compartmental Model Analysis

The host population may be easily infected over time due to the recruitment of new members. This is represented by the parameter ϕ_h. ρ is the rate of a host being infected with the chikungunya virus. The parameter is very essential in disease modelling since it shows the effect of disease transmission on the host population. It is noted that not all humans get bitten by infectious mosquitoes. Hence, the population of the mosquitoes that bite humans is I_v / N_v. The natural deaths, thus the deaths that do not occur as a result of chikungunya on the host population is represented by μ_h. The factor $\rho I_v S_h / N_v$ leaves the susceptible host class and enters the infectious host class since the moment there is an infection, members are no more susceptible; they rather become infectious. γ represents the recovery rate of an infectious host. Death due to the disease is represented by λ. The rate at which the number of infectious changes is dependent on the host infectious population. The recruitment of new members into the vector population is given by ϕ_v. A susceptible vector S_v can be easily infected by a mosquito with the chikungunya virus at a rate $\frac{\beta I_h S_v}{N_h}$ where β is the transmission rate of the virus from infected humans to susceptible vectors. α is the rate at which the exposed vector becomes infectious.
Mathematical model of the interaction between humans and vectors are defined in the form of non-linear systems of differential equations below:

$$\left.\begin{aligned}
\frac{dS_h}{dt} &= \phi_h - \mu_h S_h - \frac{\rho I_v S_h}{N_v} \\
\frac{dE_h}{dt} &= \frac{\rho I_v S_h}{N_v} - (\gamma + \mu_h + \lambda) I_h \\
\frac{dR_h}{dt} &= \gamma I_h - \mu_h R_h \\
\frac{dS_v}{dt} &= \phi_v - \mu_v S_v - \frac{\beta I_h S_v}{N_h} \\
\frac{dE_v}{dt} &= \frac{\beta I_h S_v}{N_h} - \alpha E_v - \mu_v E_v \\
\frac{dI_v}{dt} &= \alpha E_v - \mu_v I_v
\end{aligned}\right\} \quad (1)$$

D. Determination of Disease Free Equilibrium Points

To derive the disease-free equilibrium, equation (1) is equated to zero resulting in equation (2):

$$\left.\begin{aligned}
\phi_h - \mu_h S_h - \frac{\rho I_v S_h}{N_v} &= 0 \\
\frac{\rho I_v S_h}{N_v} - (\gamma + \mu_h + \lambda) I_h &= 0 \\
\gamma I_h - \mu_h R_h &= 0 \\
\phi_h - \mu_v S_v - \frac{\beta I_v S_h}{N_v} &= 0 \\
\frac{\beta I_h S_v}{N_h} - \alpha E_v - \mu_v E_v &= 0 \\
\alpha E_v - \mu_v I_v &= 0
\end{aligned}\right\} \quad (2)$$

Evaluating equation (2) gives the disease-free equilibrium points as:

$$\left[S_h^* = \frac{\phi_h}{\mu_h}, I_h^* = 0, R_h^* = 0, S_h^* = \frac{\phi_v}{\mu_v}, E_h^* = 0, I_v^* = 0 \right] \quad (3)$$

E. Determination of Basic Reproductive Number (R_0)

The basic reproductive number (R_0) measures the average number of secondary infections generated by a primary case in a state of most susceptible individuals (Chowell et al., 2004). It is the main determinant of whether a possible outbreak of a disease can generate into an epidermic (Heersterbeek, 2002). According to Otoo et al, (2019) when $R_0 < 1$, the infection dies out and there is no epidemic hence the disease-free equilibrium (DFE) is said to be locally asymptotically stable. When $R_0 > 1$ the infection will spread rapidly throughout a given population and the DFE is said to be unstable. This happens because there would be an increasing number of infective individuals within the population. In certain situations, the basic reproductive number may not be the main determinant in measuring whether an outbreak will be epidemic or not. Since at times certain conditions aiding the spread of the disease may change as the disease begins to spread. However, the basic reproductive number is still important in measuring the spread of disease since it can be expressed as an increasing function of the peak prevalence of infected hosts and the final size.

To compute R_0, the following are calculated $^{\chi}$, $V = \left(p_i^- - p_i^+ \right)$, V^{-1}, and $A = KV^{-1}$ where A is the Next Generation matrix, R_0 is the maximum eigenvalue of A. The disease compartments of the model are in the determination of the basic reproductive number considered which includes the infectious host (I_h), the Exposed vector (E_v) and the infectious vector (I_v) as indicated in equation (4)

$$\left.\begin{aligned} \frac{dI_h}{dt} &= \frac{\rho I_v S_h}{N_v} - (\gamma + \mu_h + \lambda) I_h \\ \frac{dE_v}{dt} &= \frac{\beta I_h S_v}{N_h} - \alpha E_v - \mu_v E_v \\ \frac{dI_v}{dt} &= \alpha E_v - \mu_v I_v \end{aligned}\right\} \quad (4)$$

The rate of occurrence of new infections (Y) in the compartment is given in equation (5) as:

$$Y = \begin{bmatrix} \frac{\rho I_v S_h}{N_v} \\ \frac{\beta I_v S_h}{N_h} \\ 0 \end{bmatrix} \quad (5)$$

Therefore, evaluating *Y* at the disease-free equilibrium (DFE*)* results in equation (6)

$$X = \partial Y \mid_{I_h, E_v, I_v} = \begin{pmatrix} 0 & 0 & \frac{\rho S_h}{N_v} \\ \frac{\beta S_v}{N_h} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \Rightarrow X = \begin{pmatrix} 0 & 0 & \frac{\rho \phi_h}{\mu_h N_v} \\ \frac{\beta \phi_v}{\mu_v N_h} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (6)$$

The rate of transfer of individuals into the i^{th} compartment is p_i^+, while that of individuals out of compartment the i^{th} compartment is p_i^-. The transfer state is $p = \left(p_i^- - p_i^+ \right)$. Next, the transfer is computed as indicated in equation (7):

$$p_i^- = \begin{bmatrix} (\gamma+\mu_h+\lambda)I_h \\ (\alpha+\mu_v)E_v \\ \mu_v E_v \end{bmatrix},\ p_i^+ = \begin{bmatrix} 0 \\ 0 \\ \alpha E_v \end{bmatrix} \Rightarrow p = \begin{bmatrix} (\gamma+\mu_h+\lambda)I_h \\ (\alpha+\mu_v)E_v \\ \mu_v E_v - \alpha E_v \end{bmatrix} \quad (7)$$

Evaluating the transfer state at the disease-free equilibrium points results in a non-singular matrix V as indicated in equation (9)

$$V = \partial P\Big|_{I_h,E_v,I_v} = \begin{pmatrix} (\gamma+\mu_h+\lambda) & 0 & 0 \\ 0 & (\alpha+\mu_v) & 0 \\ 0 & -\alpha & \mu_v \end{pmatrix} \quad (8)$$

$$V = \begin{pmatrix} (\gamma+\mu_h+\lambda) & 0 & 0 \\ 0 & (\alpha+\mu_v) & 0 \\ 0 & -\alpha & \mu_v \end{pmatrix} \quad (9)$$

$$V^{-1} = \begin{pmatrix} \frac{1}{(\gamma+\mu_h+\lambda)} & 0 & 0 \\ 0 & \frac{1}{(\alpha+\mu_v)} & 0 \\ 0 & \frac{-\alpha}{(\alpha+\mu_v)} & \frac{1}{\mu_v} \end{pmatrix} \quad (10)$$

Therefore, the Next Generation Matrix (A) is given by equation (11) as:

$$A = XV^{-1} = \begin{pmatrix} 0 & \frac{\rho\phi_h\alpha}{N_v\mu_h\mu_v(\alpha+\mu_v)} & \frac{\rho\phi_h}{N_v\mu_h\mu_v} \\ \frac{\beta\phi_v}{N_h\mu_v(\gamma+\mu_h+\lambda)} & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (11)$$

The eigenvalues of the next generation matrix A in equation (11) are indicated in equation (12)

$$\left(0, \frac{\sqrt{N_h N_v(\alpha+\mu_v)(\gamma+\mu_h+\lambda)\alpha\beta\rho\phi_h\phi_v}}{N_h N_v\mu_h(\gamma+\mu_h+\lambda)(\alpha+\mu_v)\mu_v}, -\frac{\sqrt{N_h N_v\mu_h(\alpha+\mu_v)(\gamma+\mu_h+\lambda)\beta\rho\phi_h\phi_v}}{N_h N_v\mu_h(\gamma+\mu_h+\lambda)(\alpha+\mu_h)\mu_v}\right) \quad (12)$$

Thus, $R_0 = \frac{\sqrt{N_h N_v(\alpha+\mu_v)(\gamma+\mu_h+\lambda)\alpha\beta\rho\phi_h\phi_v}}{N_h N_v\mu_h(\gamma+\mu_h+\lambda)(\alpha+\mu_h)\mu_v}$ (13)

This can be simplified to:

$$R_0 = \sqrt{\frac{\alpha\beta\rho\phi_h\phi_v}{N_h N_v\mu_h(\gamma+\mu_h+\lambda)(\alpha+\lambda+\mu_v)}} \quad (14)$$

R_0 represents the basic reproductive number. The presence of the square is because two generations are required for an infected vector or host to reproduce itself.

F. Local Stability of Disease-Free Equilibrium

Theorem 1 (Gershgorin Theorem)

Every eigenvalue of a square matrix satisfies the inequality $\sum_{i\neq j}|Q_{ij}| \geq |\lambda - Q_{ij}|$, $i = (1,2,3,...,n)$ where λ are the eigenvalues of the matrix Q (Dodd and Gupta, 1969).

Definition 1 (Lyapunov Function)
Suppose that x_0 is an equilibrium point for a system of equation and let V (Lyapunov Function) be a positive definite continuous differentiable function, then;

i. If $\dot{V}$ is negative semi-definite, then x_0 is stable

ii. If $\dot{V}$ is negative definite, then x_0 is asymptotically stable.

The Lyapunov functions are scalar functions which are used to prove the stability of an ordinary differential equation.
The stability analysis of the disease-free equilibrium is performed next by evaluating the Jacobian matrix as a result of equation (1) at the disease-free equilibrium point and the application of the Gershgorin theorem. Thus,

$$J(DFE) = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & 0 & \frac{-\rho\phi_h}{\mu_v\mu_h} \\ 0 & -\gamma-\mu_h-\lambda & 0 & 0 & 0 & \frac{\rho\phi_h}{N_v\mu_h} \\ 0 & \gamma & -\mu_h & 0 & 0 & 0 \\ 0 & \frac{-\beta\phi_h}{N_h\mu_v} & 0 & -\mu_v & 0 & 0 \\ 0 & \frac{\beta\phi_h}{N_h\mu_v} & 0 & 0 & -\alpha-\mu_v & 0 \\ 0 & 0 & 0 & 0 & \alpha & -\mu_h \end{bmatrix} \quad (15)$$

If $R_0 = \delta\left(KV^{-1}\right) < 1$, then the DFE is asymptotically stable and when $R_0 > 1$, the DFE is unstable.
To prove the local stability of the disease free equilibrium, Gershgorin's theorem is employed. By the Gershgorin theorem,

$$\mu_h > \frac{\rho\phi_h}{N_v\mu_h} \quad (15a)$$

$$(\gamma+\mu_h+\lambda) > \frac{\rho\phi_h}{N_v\mu_h} \quad (15b)$$

$$(\mu_h > \gamma) \quad (15c)$$

$$\mu_v > \frac{\beta\phi_h}{N_h\mu_v} \quad (15d)$$

$$(\alpha+\mu_v) > \frac{\beta\phi_v}{N_h\mu_v} \quad (15e)$$

$$\mu_v > \alpha \quad (15f)$$

Equation (15b) multiplied by equation (15e) gives:

$$(\gamma+\mu_h+\lambda)(\alpha+\mu_v) > \frac{\rho\phi_h}{N_v\mu_h}.\frac{\beta\phi_v}{N_h\mu_v} \quad (15g)$$

$$(\alpha+\mu_v)(\gamma+\mu_h+\lambda) > \frac{\beta\rho\phi_h\phi_v}{N_v\mu_h N_h\mu_v} \quad (15h)$$

Also, equation (15h) multiplied equation (15f) gives:

$$\mu_v(\alpha+\mu_v)(\gamma+\mu_h+\lambda) > \frac{\beta\rho\phi_h\phi_v}{N_v\mu_h N_h\mu_v}.\alpha \quad (15i)$$

$$\frac{N_v\mu_h\mu_v^2\mu_h(\alpha+\mu_v)(\gamma+\mu_h+\lambda)}{N_v\mu_h\mu_v^2\mu_h(\alpha+\mu_v)(\gamma+\mu_h+\lambda)} > \frac{\alpha\beta\rho\phi_h\phi_v}{N_v\mu_h\mu_v^2\mu_h(\alpha+\mu_v)(\gamma+\mu_h+\lambda)} \quad (16)$$

(16)

Therefore,

$$1 > \frac{\alpha\beta\rho\phi_h\phi_v}{N_v\mu_h\mu_v^2\mu_h(\alpha+\mu_v)(\gamma+\mu_h+\lambda)} \quad (17)$$

$$1 > R_0^2$$

$$\Rightarrow R_0 < 1 \quad (18)$$

Therefore, since $R_0 < 1$, then the disease-free equilibrium is locally asymptotically stable.

G. The Endemic Equilibrium (EE) Solution

From the system of equations in equation (1), the endemic equilibrium solution is given by the set of equation(19):

$$\left.\begin{aligned} S_h &= \frac{N_v\varepsilon_3\left(R_0^2\varepsilon_2+1\right)}{\mu_v\beta\varepsilon_5\left(R_0^2\varepsilon_1+1\right)}, I_h = \frac{N_v\mu_h\varepsilon_3\left(R_0^2-1\right)}{\beta\varepsilon_4\varepsilon_5\left(R_0^2\varepsilon_1+1\right)}, R_h = \frac{\gamma N_v\mu_h\varepsilon_3\left(R_0^2-1\right)}{\mu\beta\varepsilon_4\varepsilon_5\left(R_0^2\varepsilon_1+1\right)} \\ S_v &= \frac{N_h\varepsilon_4\varepsilon_5\left(R_0^2\varepsilon_1+1\right)}{\alpha\rho N_h\varepsilon_4\left(R_0^2\varepsilon_2\varepsilon_6+1\right)}, E_v = \frac{N_v\mu_h\varepsilon_3\left(R_0^2-1\right)}{\alpha\rho N_h\varepsilon_4\varepsilon_6\left(R_0^2\varepsilon_1+1\right)}, I_v = \frac{N_v\mu_h\varepsilon_3\left(R_0^2-1\right)}{\rho\varepsilon_3\left(R_0^2\varepsilon_2\varepsilon_6+1\right)} \end{aligned}\right\} \quad (19)$$

Where ε_1, ε_2, ε_3, ε_4, ε_5, ε_6 are given by:

$$\left.\begin{aligned} \varepsilon_1 &= \frac{N_h\mu_v\left(\gamma+\lambda+\mu_h\right)}{\beta\phi_h}, \varepsilon_2 = \frac{N_h\mu_v\mu_h}{\alpha\rho\phi_v}, \varepsilon_3 = N_h\mu_v^2\left(\alpha+\mu_v\right)\left(\gamma+\lambda+\mu_h\right) \\ \varepsilon_4 &= \mu_v\left(\gamma+\lambda+\mu_h\right), \varepsilon_5 = N_h\mu_v\left(\gamma+\lambda+\mu_h\right), \varepsilon_6 = (\alpha+\mu_v) \end{aligned}\right\} \quad (20)$$

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H. Local Stability of Endemic Equilibrium Solution

Due to the complex nature in computing the eigenvalues from the characteristics polynomial, Feng et al, (2001) described an approach with the introduction of the basic reproduction number, R_0 in the analysis of the endemic equilibrium states. According to Feng et al., (2001), when $R_0 > 1$, the system has a unique endemic equilibrium that is locally asymptotically stable. Applying this technique to analyze the local stability of the endemic equilibrium, a Jacobian matrix, J was formulated by differentiating each of the system of equations in equation (1) with respect to $S_h, I_h, R_h, S_v, E_v, I_v$, and evaluated at the endemic equilibrium points.
The Jacobian matrix evaluated at the endemic equilibrium is represented in equation (21):

$$J(EE)=\begin{pmatrix} -\mu_h-\frac{\mu_h\left(R_0^2-1\right)}{R_0^2\varepsilon_2\varepsilon_3+1} & 0 & 0 & 0 & 0 & -\frac{\rho\varepsilon_3\left(R_0^2\varepsilon_2+1\right)}{\mu_v\beta\varepsilon_5\left(R_0^2\varepsilon_1+1\right)} \\ \frac{\mu_h\left(R_0^2-1\right)}{R_0^2\varepsilon_2\varepsilon_3+1} & -\gamma-\mu_h-\lambda & 0 & 0 & 0 & \frac{\rho\varepsilon_3\left(R_0^2\varepsilon_2+1\right)}{\mu_v\beta\varepsilon_5\left(R_0^2\varepsilon_1+1\right)} \\ 0 & \gamma & -\mu_h & 0 & 0 & 0 \\ 0 & -\frac{\beta\varepsilon_5\left(R_0^2\varepsilon_2+1\right)}{\alpha\rho\left(R_0^2\varepsilon_2\varepsilon_6+1\right)N_h} & 0 & -\mu_v-\frac{N_v\mu_h\varepsilon_3\left(R_0^2-1\right)}{\varepsilon_4\varepsilon_5\left(R_0^2\varepsilon_1+1\right)N_h} & 0 & 0 \\ 0 & \frac{\beta\varepsilon_5\left(R_0^2\varepsilon_2+1\right)}{\alpha\rho\left(R_0^2\varepsilon_2\varepsilon_6+1\right)N_h} & 0 & \frac{N_v\mu_h\varepsilon_3\left(R_0^2-1\right)}{\varepsilon_4\varepsilon_5\left(R_0^2\varepsilon_1+1\right)N_h} & -\alpha-\mu_v & 0 \\ 0 & 0 & 0 & 0 & -\alpha & -\mu_v \end{pmatrix} \quad (21)$$

By applying the Gershgorin's theorem to equation (21) and multiplying through by (-1) results in the following:

$$\mu_h+\frac{\mu_h\left(R_0^2-1\right)}{R_0^2\varepsilon_2\varepsilon_3+1}>\frac{\rho\varepsilon_3\left(R_0^2\varepsilon_2+1\right)}{\mu_v\beta\varepsilon_5\left(R_0^2\varepsilon_1+1\right)} \quad (22)$$

$$(\gamma+\mu_h+\lambda)>-\left(\frac{\mu_h\left(R_0^2-1\right)}{R_0^2\varepsilon_2\varepsilon_3+1}+\frac{\rho\varepsilon_3\left(R_0^2\varepsilon_2+1\right)}{\mu_v\beta\varepsilon_5\left(R_0^2\varepsilon_1+1\right)}\right) \quad (23)$$

$$\mu_h>-\gamma$$

$$\left(\mu_v+\frac{N_v\mu_h\varepsilon_3\left(R_0^2-1\right)}{\varepsilon_4\varepsilon_5\left(R_0^2\varepsilon_1+1\right)N_h}\right)>\frac{\beta\varepsilon_5\left(R_0^2\varepsilon_2+1\right)}{\alpha\rho\left(R_0^2\varepsilon_2\varepsilon_6+1\right)N_h} \quad (24)$$

$$\alpha+\mu_v>-\left(\frac{\beta\varepsilon_5\left(R_0^2\varepsilon_2+1\right)}{\alpha\rho\left(R_0^2\varepsilon_2\varepsilon_6+1\right)N_h}+\frac{N_v\mu_h\varepsilon_3\left(R_0^2-1\right)}{\varepsilon_4\varepsilon_5\left(R_0^2\varepsilon_1+1\right)N_h}\right) \quad (25)$$

$$\mu_v>-\alpha \quad (26)$$

By analyzing the above inequalities, R_0 is positive and for this accession to hold, $R_0^2-1>0$. Hence $R_0^2>1$. This shows that the endemic equilibrium is locally asymptotically stable.

I. Global Stability Of Disease-Free Equilibrium

With reference to Tewa et al. (2009), our Lyapunov function is defined to be

$$V=\left(S_h-S_h^*\ln S_h\right)+I_h+R_h+\left(S_v-S_v^*\ln S_v\right)+E_v+I_v \quad (27)$$

Next,

$$\dot{V}=\left(\phi_h-\mu_hS_h-\frac{\rho I_vS_h}{N_v}\right)\left(1-\frac{S_h^*}{S_h}\right)+\frac{\rho I_vS_h}{N_v}-\gamma I_h-\mu_hI_h-\lambda I_h+\gamma I_h-R_h\mu_h+\left(\phi_v-\mu_vS_v-\frac{\beta I_hS_v}{N_h}\right)+\frac{\beta I_hS_v}{N_h}-\alpha E_v-\mu_vE_v+\alpha E_v-\mu_vI_v \quad (28)$$

Cancelling the terms; $-\gamma I_h,\gamma I_h,-\alpha E_v,\alpha E_v$

$$=\phi_h\left(1-\frac{S_h^*}{S_h}\right)-\mu_hS_h\left(1-\frac{S_h^*}{S_h}\right)+\frac{\rho I_vS_h^*}{N_v}+\phi_v\left(1-\frac{S_v^*}{S_v}\right)-\mu_vS_v\left(1-\frac{S_v^*}{S_v}\right)-\mu_hI_h-\lambda I_h+\frac{\beta I_hS_v}{N_h}-R_h\mu_h-\mu_vE_v-\mu_vI_v \quad (29)$$

Assume that: $\lambda=\frac{\beta S_v^*}{N_h},\mu_v=\frac{\rho S_h^*}{N_v}$ and at the DFE: $S_h^*=\frac{\phi_h}{\mu_h},S_v^*=\frac{\phi_v}{\mu_v}$

$$\overset{*}{V} = \phi_h\left(1-\frac{S_h^*}{S_h}\right)+\mu_h\frac{\phi_h}{\mu_h}\left(1-\frac{S_h}{S_h^*}\right)+\phi_v\left(1-\frac{S_v^*}{S_v}\right)+\mu_v\frac{\phi_v}{\mu_v}\left(1-\frac{S_v}{S_v^*}\right)-\mu_h I_h - R_h\mu_h - \mu_v E_v \quad (30)$$

$$\overset{*}{V} = -\phi_h\left(\frac{S_h^*-S_h}{S_h}\right)-\phi_h\left(\frac{S_h-S_h^*}{S_h^*}\right)-\phi_v\left(\frac{S_v^*-S_v}{S_v}\right)-\phi_v\left(\frac{S_h-S_h^*}{S_h^*}\right)-\mu_h I_h - R_h\mu_h - \mu_v E_v \quad (31)$$

$$\overset{*}{V} = -\phi_h\left[\frac{S_h^*\left(S_h^*-S_h\right)+S_h\left(S_h-S_h^*\right)}{S_hS_h^*}\right]-\phi_v\left[\frac{S_v^*\left(S_v^*-S_v\right)+S_v\left(S_v-S_v^*\right)}{S_vS_v^*}\right]-\mu_h I_h - R_h\mu_h - \mu_v E_v \quad (32)$$

$$\overset{*}{V} = -\phi_h\left[\frac{S_h\left(S_h-S_h^*\right)^2}{S_hS_h^*}\right]-\phi_v\left[\frac{S_v\left(S_v-S_v^*\right)^2}{S_vS_v^*}\right]-\mu_h I_h - R_h\mu_h - \mu_v E_v < 0 \quad (33)$$

$$\therefore \overset{*}{V} < 0$$

Hence since the solution for $\overset{*}{V}$ will always produce strictly non-zero results, it therefore proves that the disease free equilibrium is globally asymptotically stable.

J. Numerical Simulations

In this section, the numerical simulations of the Chikungunya model are presented using parameter values which were taken from various sources and others estimated. These parameter values are indicated in table 1. All simulations in this section were performed using Maple Software.

TABLE 1: PARAMETERS, VALUES, AND THEIR SOURCES

Parameter	Value	Source
$S_h(0)$	80,000	Estimated
$I_h(0)$	20,000	Estimated
$R_h(0)$	0	Estimated
$S_v(0)$	20000	Estimated
E_v	6000	Estimated
$I_v(0)$	4000	Estimated
N_h	100,000	Estimated
ϕ_h	(0.0201-0.0223) day^{-1}	Naowarat et al,. 2012
μ_h	(0.0285-0.03021) day^{-1}	Nishiura(2006)
ρ	0.245-0.323	Moulay and Piagne (2012)
λ	$\leq 0.001\%$	Anon (2016)
γ	0.0472-0.1428	Tang et al,. (201l)
N_v	30, 000	Estimated
ϕ_v	0.022-0.026	Dumont and Chiroleu (2010)
μ_v	(0.0153-0.0156) day^{-1}	Naowarat et al., 2011
α	(0.246-0.254) day^{-1}	Tsetsarkin et al., 2011
β	(0.0561-0.0589)$^{-1}$	Manore et al.,

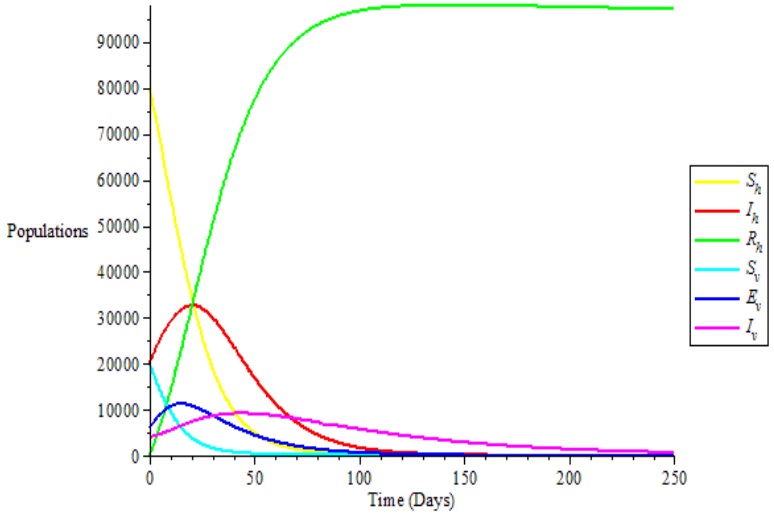

Figure 2 Simulations of the Human and Mosquito Population

Figure 2 shows a steady decline in the number of susceptible human population which corresponded to an increase in the infective and the recovery populations of humans during the initial stage of the disease. As time increases, the susceptible human, the infective human and the recovery human populations become asymptotic to the horizontal axes. Similarly, the susceptible mosquito population showed a sharp decline at the onset of the disease and after some time moves in a characteristic curve. The exposed vector population also started to increase considerably in the onset of the disease while the infectious mosquito population behaved similarly to that of the infective human population.
Simulations for the individual compartments are displayed in the following graphs.

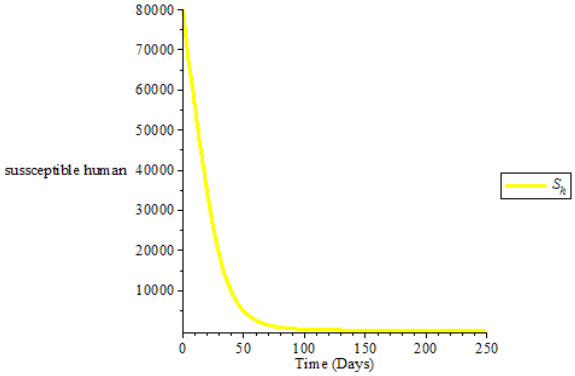

Figure 3 Simulation of Susceptible Human

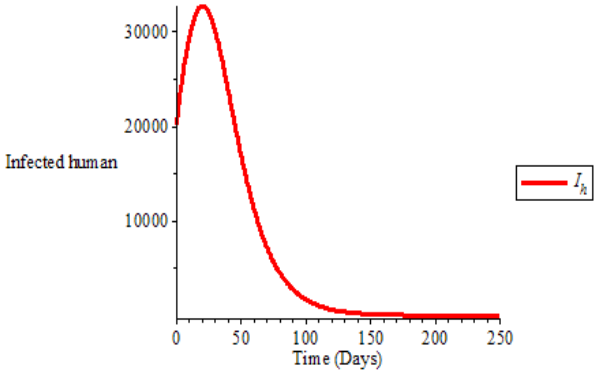

Figure 4 Simulations of Infectious Human

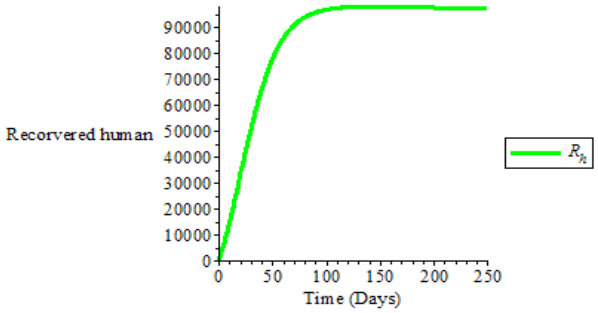

Figure 5 Simulations of Recovered Human

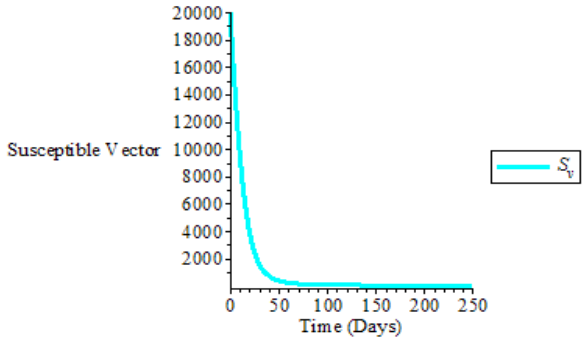

Figure 6 Simulation of Susceptible Vector

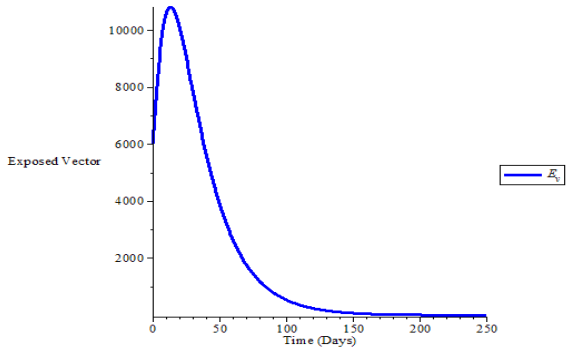

Figure 7 Simulations of Exposed Vector

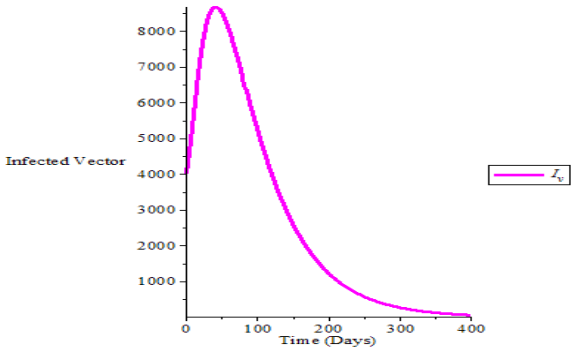

Figure 8 Simulation of Infectious Vector

K. Analysis Of The Numerical Simulations Of The Model Compartments

From Figure 3 at the onset of an epidemic, no individual has immunity to Chikungunya fever and hence, the human susceptible population equals the total host population. When ineffective are introduced into the population, the population begins to decline gradually and becomes asymptotic over time, which results in a corresponding rise in the infectives of the population. The reason is being that, when the disease enters the population, there is a progression from the susceptible class and hence the infectious individuals begin to increase causing the susceptible host population to decrease. Eventually, since the disease induces permanent immunity on the individual, those that recover do not come back to the susceptible class again.

Also from Figure 4, it was observed that initially, few individuals get infected with the disease but as time increases, the infection multiples and the population become at risks. Hence, the infectious rises to its peak and begins to decline as time elapses and consequently becomes asymptotic to the horizontal axis. The graph also demonstrates that the transmission rate has a significant impact on the spread of the disease through the population. If the transmission rate is observed to be high then the rate of infection of the disease will also be high.

Furthermore, Figure 5 shows a gradual rise from an initial value of 0 to around 100,000 in about eighty days after the onset of the disease. It then continues to remain constant due to the moderate host recovery rate and a very low disease-induced death rate. Recovered individuals gain permanent immunity to the disease and hence individuals do not become susceptible again.

Figure 7 represents the numerical simulation of the exposed vector at a given time. As infective are introduced in the susceptible class, they become exposed to the disease. The graph shows a continuous increase in the number of exposed vector until it reaches its maximum and then begins to fall and eventually become asymptotically stable. After some days, members of this compartment become symptomatic of the disease and are then transferred into the infected class. Also, as the exposed vector population increases that of the susceptible population decreases as indicated in Figure 6. There is an inverse relationship between the exposed vector population the susceptible vector population.

For the infected vector class as indicated in Figure 8, the number of infectious will increase since the mosquitoes that are exposed to the disease keeps increasing. It continues to rise to a maximum point after which it begins to decline as some of the mosquitoes infected die naturally. The number of infected mosquitoes will keep on decreasing until they all become extinct leaving only susceptible mosquitoes in the system. In that case, the disease dies out.

CONCLUSIONS

The study modelled and analyzed the transmission dynamics of Chikungunya without any control measure. The basic reproductive number determined was less the maximum threshold which implies the disease will not be epidemic. From the stability analysis performed, it was realized that both the disease-free equilibrium and the local endemic equilibrium were stable which confirms the basic reproductive number achieved. Also, from the numerical simulations, it was observed that the disease can spread rapidly in a geographical area when the numbers of infected individuals are higher.

RECOMMENDATION

The effect of seasonality on the transmission of the disease was not taken into consideration in the model. Therefore, the effect of seasonality in disease transmission is recommended as further studies.

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