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MODELLING THE DYNAMICS OF CHIKUNGUNYA IN HUMAN AND MOSQUITO POPULATIONS

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ABSTRACT

In this paper, we developed and qualitatively analyzed a model for the transmission dynamics of chikungunya virus in a human and mosquito populations. The chikungunya virus is transmitted by Aedes mosquitoes usually Aedes albopictus mosquito and it is of big threat to global public health. Rigorous analysis of the model shows that the model has a locally asymptotically stable disease free equilibrium whenever a certain epidemiological threshold quantity, called the basic reproduction number is less than unity (hence, the diseases would not persist in the community). The model has a unique endemic equilibrium when the reproduction number exceeds unity. Furthermore, the model exhibits the phenomenon of backward bifurcation, where the stable disease free equilibrium coexists with a stable endemic equilibrium.

Keywords: Chikungunya virus; vertical transmission; stability; backward bifurcation.

INTRODUCTION

Chikungunya virus (CHK) is viral disease that is transmitted to humans through the bites of an infected female mosquitoes (vector-bone disease) (Agusto *et al.,* 2016), it is an *arboviral* disease caused by a member of the genus *alphavirus* that belongs to *togaviridae* family (Manore *et al.,* 2014; Saswat *et al.,* 2013;Naowarat et al., 2011; Yakob and Clements, 2013). CHK virus is a non-fatal disease that results in too much morbidity in an affected humans and has an effect on the country's economy as estimated by disability adjusted life years (DALY) thereby becoming a a big threat to global public health and development (Manore *et al.,* 2014; Saswat *et al.,* 2013; Jain et al., 2016). CHK virus gives permanent immunity after recovery of an individual (Manore *et al.,* 2014; Saswat et al., 2013).

CHK virus is endemic in more than 60 countries in Asia, Africa, Europe, and America resulting several clinical cases or death (Saswat *et al.,* 2013; Naowarat *et al.,* 2011; Yakob and Clements, 2013; Dumont and Tchuenche, 2011; WHO, 2016). CHK was not considered as a life threatening disease because it has a low death rate (Manore *et al.,* 2014). More than 1.3 million suspected cases of CHK and about 200 death cases have been reported in the Latin American countries, Caribbean islands, and the United States of America in April, 2015 (Yakob and Clements, 2013; WHO, 2016).

The CHK virus is a vector bone diseases (transmitted to humans via the bites of an infected mosquitoes) (Manore *et al.,* 2014). *Aedes aegypti* and *Aedes albopictus* mosquitoes are the principal vectors for the transmission of CHK virus worldwide and the viral infection has the following primary signs and symptoms in the patients which are fever, joint pain, headache, muscle pain, skin rash and incapacitating arthralgia (Manore *et al.,* 2014; Saswat *et al.,* 2013; Naowarat *et al.,* 2011).

Vertical transmission has been shown to be an important component that enables the virus to be kept up in non-conducive climatic conditions in nature (Jain et al., 2016). Recently, experimental vertical transmission of CHK virus in *Aedes aegypti* has been shown (Agarwal *et al.,* 2014). Information on vertical transmission of CHK virus in natural population is of great significance in order to comprehend the possible processes of virus survival during inter epidemic periods. In this paper we reported vertical transmission of CHK virus in aquatic and adult stages in the mosquito population.

Currently, there is no specific and effective vaccine for CHK virus at the moment, but a number of vaccines are undergoing clinical trials (Manore et al., 2014).

Although there is no specific treatment for CHK virus (Manore et al., 2014; Saswat et al., 2013). However, treatment could be done by decreasing the symptoms in order to reduce the burden of the disease (WHO, 2016; Agusto et al., 2016).

A number of mathematical models have been designed to give insights into the transmission dynamics of CHK (see, for instance, (Manore et al., 2014; Saswat et al., 2013; Naowarat et al., 2011; Yakob and Clements, 2013; Dumont and Tchuenche, 2011; Agusto et al., 2016)). To the author's knowledge, the current study give the first model for CHK virus that incorporate the aquatic stage (egg, lava and pupa stages) and vertical transmission in a mosquito population.

Model Formulation

The total human population at time t, denoted by $N_H(t)$, is divided into four mutually exclusive compartments as follows: susceptible individuals, who are at risk of infection of CHK virus ($S_H(t)$), asymptomatic CHK individuals ($E_H(t)$), CHK early infected individuals with clinical symptoms of CHK ($I_{H1}(t)$), CHK advance infected individuals with clinical symptoms of CHK ($I_{H2}(t)$), individuals who recovered from CHK ($R_H(t)$), so that:

$$N_H(t) = S_H(t) + E_H(t) + I_{H1}(t) + I_{H2}(t) + R_H(t).$$

Similarly, the total mosquito (female A*edes albopictos or Aedes aegypti* mosquito) population at time t denoted by ($N_v(t)$), is sub-divided into sub-populations of immature mosquitoes (eggs, larvae and pupae stages), denoted by $A(t)$, and adult mosquitoes (denoted by $N_I(t)$), so that: $N_v(t) = A(t) + N_I(t)$. where $N_I(t)$ is further divided into three compartments as follows: adult mosquitoes susceptible to CHK viruses ($S_v(t)$), adult mosquitoes exposed to CHK ($E_v(t)$), CHK-infected adult mosquitoes ($I_v(t)$), so that: $N_I(t) = S_v(t) + E_v(t) + I_v(t)$.

The susceptible population of individual who are at the risk of CHK virus infection ($S_H(t)$) is generated by recruitment of humans at a constant rate Π_H (all humans recruited into the population are assumed to be at risk of CHK infection). The population is diminished following infection with CHK (at a rate λ_H), and is also decreased by natural death (at a rate μ_H: this is assumed to be the same in all human compartments).

Thus, $$\frac{dS_H}{dt} = \Pi_H - \lambda_H S_H - \mu_H S_H.$$

The population of asymptomatic CHK individuals (E_H) is generated following the infection of susceptible individuals (at a rate $\lambda_1 H$). It is decreased by the development of clinical symptoms of CHK (at a rate σ_1), and natural death.

Hence, $$\frac{dE_H}{dt} = \lambda_H S_H - (\sigma_1 + \mu_H)E_H.$$

The population of CHK early infected humans with clinical symptoms of CHK (I_{H1}) is generated by progression of CHK-exposed individuals (at the rate σ_1). The population is decreased by progression to the advanced-infectious state of CHK (at a rate σ_2). The population is further decreased by natural death and CHK-induced death (at a rate δ_1). So that,

$$\frac{dI_{H1}}{dt} = \sigma_1 E_H - (\sigma_2 + \delta_1 + \mu_H)I_{H1}.$$

The population of individuals at advanced-infectious state of CHK with clinical symptoms of CHK (I_{H2}) is generated at the rate σ_2. The population is decreased by recovery of CHK (at a rate τ_H). It is further decreased by natural death and CHK-induced death (at a rate δ_2). So that,

$$\frac{dI_{H2}}{dt} = \sigma_2 E_H - (\tau_H + \delta_2 + \mu_H)I_{H2}.$$

The population of recovered CHK-infected individuals (R_H) is generated by the successful treatment of CHK-infected individuals (at the rate τ_H). It is diminished by natural death.

Therefore, $$\frac{dR_H}{dt} = \tau_H I_{H2} - \mu_H R_H.$$

Immature mosquitoes (eggs, larvae and pupae) are lumped into a single compartment (A) for computational convenience (Okuneye & Gumel, 2016). The population of immature mosquitoes is generated at the rate (Π_v), where Π_v is the egg deposition rate. The population of immature mosquitoes is decreased by maturation to susceptible adult mosquitoes (at a rate ξ), infectious adult mosquitoes (at a rate $(1-\xi)$) and natural death (at a rate μ_A). So that,

$$\frac{dA}{dt} = \Pi_v - (1 + \mu_A)A.$$

The population of susceptible adult mosquitoes (S_v) is generated at the rate (ξ). The population is diminished by infection, following effective contact with CHK-infected (at a rate λ_v) and natural death (at a rate μ_v; this rate is assumed, for mathematical convenience, to be same for all the epidemiological classes for mosquitoes).

Hence, $\frac{dS_v}{dt} = \xi A - \lambda_v S_v - \mu_v S_v$.

The population of CHK-exposed adult mosquitoes (E_v) is generated following infection with CHK (at a rate λ_v). It further decreased by the progression of CHK (at a rate σ_v), and natural death. Thus, $\frac{dE_v}{dt} = \lambda_v S_v - (\sigma_v + \mu_v)E_v$.

The population of CHK-infected adult mosquitoes (I_v) is generated at the rate (1 −ξ) and (σ_v). It is further decreased by natural death. Thus, $\frac{dI_v}{dt} = (1-\xi)A + (\sigma_v + \mu_v)E_v$.

The model for the CHK is given by the following deterministic system of non-linear differential equations (a flow diagram of the model is depicted in Figure 1).

$$\frac{dS_H}{dt} = \Pi_H - \lambda_H S_H - \mu_H S_H,$$
$$\frac{dE_H}{dt} = \lambda_H S_H - (\sigma_1 + \mu_H)E_H,$$
$$\frac{dI_{H1}}{dt} = \sigma_1 E_H - (\sigma_2 + \delta_1 + \mu_H)I_{H1},$$
$$\frac{dI_{H2}}{dt} = \sigma_2 E_H - (\tau_H + \delta_2 + \mu_H)I_{H2},$$
$$\frac{dR_H}{dt} = \tau_H I_{H2} - \mu_H R_H, \quad (1)$$
$$\frac{dA}{dt} = \Pi_v - (1+\mu_A)A,$$
$$\frac{dS_v}{dt} = \xi A - \lambda_v S_v - \mu_v S_v,$$
$$\frac{dE_v}{dt} = \lambda_v S_v - (\sigma_v + \mu_v)E_v,$$
$$\frac{dI_v}{dt} = (1-\xi)A + \sigma_v E_v - \mu_v I_v,$$

where, $\lambda_H = \frac{\beta_H \sigma_m \sigma_H}{(\sigma_m N_v + \sigma_H N_H)(\eta_v E_v + I_v)}$ and $\lambda_v = \frac{\beta_H \sigma_m \sigma_H}{(\sigma_m N_v + \sigma_H N_H)(\eta_H E_H + \eta_1 I_{H1} + \eta_2 I_{H2})}$, (2)

$N_H(t) = S_H(t) + E_H(t) + I_{H_1}(t) + I_{H_2}(t) + R_H(t)$ and $N_v(t) = A + S_v(t) + E_v(t) + I_v(t)$.

In (2), β_H is the transmission probability of CHK and η_H, η_1, η_2, $\eta_v > 1$ are the modification parameters accounting for the assumption that infected humans and mosquitoes are more infectious than exposed humans and mosquitoes, respectively (Duong et al., 2015). Furthermore, $b_1(N_H, N_v)$ is the per capita biting rate of female mosquito on the human host per unit time. Similarly, $b_2(N_H, N_v)$ is the number of bites per mosquito per unit time. Following (Chitnis et al., 2006), the biting rates $b_1(N_H, N_v)$ and $b_2(N_H, N_v)$ are respectively given by,

$$b_1(N_H, N_v) = \frac{\sigma_m \sigma_H N_v}{\sigma_m N_v + \sigma_H N_H} \text{ and } b_2(N_H, N_v) = \frac{\sigma_m \sigma_H N_H}{\sigma_m N_v + \sigma_H N_H}$$

Where σ_m is the number of times one mosquito would want to bite humans per unit time (if humans were freely available) and σ_H is the maximum number of mosquito bites a human can receive per unit time.

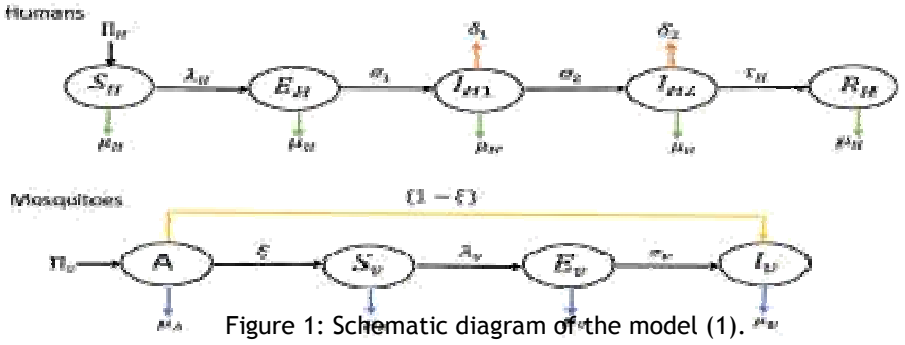

Figure 1: Schematic diagram of the model (1).

The model (1) is an extension of some of the CHK transmission models (e.g., those in (Manore et al., 2014; Yakob & Clements, 2013; Agusto et al., 2016) by (inter alia) :
i. Including the dynamics of early infectious and advanced infectious state of CHK in human population (this was not included in (Agusto et al., 2016; Yakob & Clements, 2013));
ii. Using a nonlinear biting rate (constant rate was used in (Agusto et al., 2016);
iii. Including the dynamics of an aquatic (immature) stage of the mosquito (egg, lava and pupa stages) (this was not considered in (Manore et al., 2014; Yakob & Clements, 2013; Agusto et al., 2016)).

Basic qualitative properties of the model

In this section, the basic qualitative features of the model (1) will now be explored.
Let $\mu = min(\mu_A, \mu_v)$. We claim the following:

Theorem: *The system (1) preserves positivity of solutions. In other words, the solutions of the model* (1) *with positive initial data and remain positive* $\forall t > 0$. *Further,* $\lim_{t\to\infty} Sup N_H \leq \frac{\Pi_H}{\mu_H}$ and $\lim_{t\to\infty} Sup N_v \leq \frac{\Pi_v}{\mu}$.

Proof. It is clear from the first equation of the model (1) that,

$$\frac{dS_H}{dt} = \Pi_H - \lambda_H S_H - \mu_H S_H \geq -(\lambda_H + \mu_H)S_H \quad (3)$$

so that, $S_H(t) \geq S_H(0)exp^{-\int_0^t(\lambda_H+\mu_H)dt} > 0.$ (4)

Using similar approach, it can be shown that all other state variables of the model remain positive $\forall t > 0$. Furthermore, adding the first five equations in the model (1) and the last four equations of the system (1) gives, respectively,

$$\frac{dN_H(t)}{dt} = \Pi_H - \mu_H N_H - \delta_1 I_{H1} - \delta_2 I_{H2} \quad (5)$$

and $\frac{dN_v(t)}{dt} = \Pi_v - \mu_A A - \mu_v N_I$ (6)

Thus, $\Pi_H - \mu_H N_H - \delta_1 I_{H1} - \delta_2 I_{H2} \leq \frac{dN_H(t)}{dt} \leq \Pi_H - \mu_H N_H$ and $\Pi_v - \mu_A A - \mu_v N_I \leq \frac{dN_v(t)}{dt} \leq \Pi_v - \mu N_v$. So that, $\frac{\Pi_H}{\mu_H + \delta_1 + \delta_2} \leq \lim_{t\to\infty} \inf N_H(t) \leq \lim_{t\to\infty} \sup N_H(t) \leq \frac{\Pi_H}{\mu_H}$ and $\frac{\Pi_v}{\mu_A + \mu_v} \leq \lim_{t\to\infty} \inf N_v(t) \leq \lim_{t\to\infty} \sup N_v(t) \leq \frac{\Pi_v}{\mu}$. Hence, $\lim_{t\to\infty} \sup N_H(t) \leq \frac{\Pi_H}{\mu_H}$ and $\lim_{t\to\infty} \sup N_v(t) \leq \frac{\Pi_v}{\mu}$.

Lemma: *The following biologically feasible region of the model equation (1)*

$\mathbf{D} = \left\{(S_H, E_H, I_{H1}, I_{H2}, R_H, A, S_v, E_v, I_v) \in \mathbf{R}_+^9 : N_H \leq \frac{\Pi_H}{\mu_H}, N_v \leq \frac{\Pi_v}{\mu}\right\}$ *is positively invariant and attracting*. Refer to (Van-den & Watmough,2002) for the proof of the above lemma. Therefore, the model (1) is mathematically well-posed and epidemiologically reasonable since all the variables remain non-negative for all $t \geq 0$.Hence,it is sufficient to consider the dynamics of the model (1)in D (Hethcote,2000).

Asymptotic stability of disease-free equilibrium (DFE)

TDFE

Theorem: *The TDFE of the model* (1), *denoted by* Y_0, *is GAS in* Ω *whenever* $R_N \leq 1$.

Proof. Following(Okuneye&Gumel,2016),the model(1)can be re-written as $\frac{dY}{dt} = A(Y)Y + G$,

Where $Y = (S_H, E_H, I_{H1}, I_{H2}, R_H, A, S_v, E_v, I_v)^T$, $A(Y)$ is a 9x9 M-matrix (Metzler-Matrix) given by,

$$A(Y) = \begin{bmatrix} \frac{-\beta_H\sigma_m\sigma_H(\eta_v E_v + I_v)}{\sigma_m N_v + \sigma_H N_H} - \mu_H & 0 & 0 & 0 & 0 & 0 & 0 & \frac{-\beta_H\sigma_m\sigma_H\eta_v S_H}{\sigma_m N_v + \sigma_H N_H} & \frac{-\beta_H\sigma_m\sigma_H S_H}{\sigma_m N_v + \sigma_H N_H} \\ \frac{\beta_H\sigma_m\sigma_H(\eta_v E_v + I_v)}{\sigma_m N_v + \sigma_H N_H} & -\sigma_H - \mu_H & 0 & 0 & 0 & 0 & 0 & \frac{\beta_H\sigma_m\sigma_H\eta_v S_H}{\sigma_m N_v + \sigma_H N_H} & \frac{\beta_H\sigma_m\sigma_H S_H}{\sigma_m N_v + \sigma_H N_H} \\ 0 & \sigma_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -\mu_H & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -1-\mu_A & 0 & 0 & 0 \\ 0 & \frac{-\beta_v\sigma_m\sigma_H\eta_H S_v}{\sigma_m N_v + \sigma_H N_H} & \frac{-\beta_v\sigma_m\sigma_1\eta_H S_v}{\sigma_m N_v + \sigma_H N_H} & \frac{-\beta_v\sigma_m\sigma_1\eta_2 S_v}{\sigma_m N_v + \sigma_H N_H} & 0 & \xi & \frac{-\beta_v\sigma_m\sigma_H(\eta_H E_H + \eta_1 I_{H1} + \eta_2 I_{H2})}{\sigma_m N_v + \sigma_H N_H} - \mu_H & 0 & 0 \\ 0 & \frac{\beta_v\sigma_m\sigma_H\eta_H S_v}{\sigma_m N_v + \sigma_H N_H} & \frac{\beta_v\sigma_m\sigma_1\eta_H S_v}{\sigma_m N_v + \sigma_H N_H} & \frac{\beta_v\sigma_m\sigma_1\eta_2 S_v}{\sigma_m N_v + \sigma_H N_H} & 0 & 0 & \frac{\beta_v\sigma_m\sigma_H(\eta_H E_H + \eta_1 I_{H1} + \eta_2 I_{H2})}{\sigma_m N_v + \sigma_H N_H} & -\sigma_v - \mu_v & 0 \\ 0 & 0 & 0 & 0 & 0 & 1-\xi & 0 & \sigma_v & -\sigma_v \end{bmatrix}$$

And $G=(\Pi_H, 0,0,0,0,0,0,0,0)^T$. let $R_N < 1$ (so the model (1) has only the TDFE, τ_0). Furthermore, let $Z = Y - TDFE$. Thus, the equation $\frac{dY}{dt} = A(Y)Y + G$ can be re-written as $\frac{dZ}{dt} = B(Z)Z$, where $B(Z)$ is the 9 x 9 matrix of coefficients of the model (1) with variables $Z_i (i = 1,2,3,4,5,6,7,8,9)$. It is clear that $TDFE_Z = (0,0,0,0,0,0,0,0,0)$ is the only equilibrium of the system $\frac{dZ}{dt} = B(Z)Z$. Consider the Lyapunov function $V(Z) = \langle W, Z \rangle$ with $W = \langle \frac{1,1,1,1,1,1}{\Pi_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v}, \frac{1}{\mu_v} \rangle \gg 0$ (Okuneye & Gumel, 2016). Thus $V(Z) > 0$ except at $Z = TDFE_Z$. Furthermore (where a prime now denotes differentiation with respect to Z),

$$V'(Z) = \langle W, B(Z)Z \rangle = -(Z_7 + Z_8 + Z_9) - \frac{1+\mu_A}{\Pi_v} Z_6 + \frac{1}{\mu_v} Z_6 = -(Z_7 + Z_8 + Z_9) - \frac{1+\mu_A}{\Pi_v(1-R_N)Z_6}$$

.

Since $R_N \leq 1$ in $C([0], \mathbb{R}_+^9)$, it follows that $V'(Z) \leq 0$. Furthermore, it follows from La Salles's Invariance Principle (Theorem 6.4 of (LaSalle, 1976)) that the maximal invariant set contained in $\frac{V}{V'(Z)} \leq 0$ is the $TDFE_Z$. Thus, the transformed equilibrium, $TDFE_Z$, is GAS in $C([0], \mathbb{R}_+^9)$ if $R_N \leq 1$.

NDFE

Using the next generation operator method on the system(Van-den&Watmough,2002), the associated basic reproduction number of the model (9), denoted by R_0, is given by,

$$R_0 = \sqrt{\frac{\xi\Pi_v\Pi_H\beta_v\beta_C\mu_H(\eta_v\mu_v + \sigma_v)(\eta_H Q_2 + \eta_1\sigma_1)Q_3 + \eta_2\sigma_1\sigma_2)\sigma_H^2\sigma_m^2}{Q_1Q_2Q_3Q_4(1+\mu_A)\mu_v^2(\mu_H\sigma_m\Pi_v + \mu\sigma_H\Pi_H)^2}}$$,where,

$C_1 = \frac{\Pi_H\beta_C\sigma_m\sigma_H\mu}{\mu_H\sigma_m\Pi_v + \mu\sigma_H\Pi_H}$, $C_2 = \frac{\Pi_v\xi\beta_v\sigma_m\sigma_H\mu\mu_H}{\mu_v(1+\mu_A)(\mu_H\sigma_m\Pi_v + \mu\sigma_H\Pi_H)}$, $Q_1 = \sigma_1 + \mu_H$, $Q_2 = \sigma_2 + \delta_1 + \mu_H$, $Q_3 = \tau_H + \delta_2 + \mu_H$, and $Q_4 = \sigma_v + \mu_v$.

Lemma: The DFE (E_0), of the CHK model(1) is locally asymptotically stable (LAS) if $R_0 < 1$, and unstable if $R_0 > 1$.

Existence of backward bifurcation

Here, the center manifold theory will be use to investigate the conditions on the parameter values in the model that cause forward or backward bifurcation to occur (Carr,1981;Castillo-Chavez&Song,2004;Van-den & Watmough,2002).

Consider the system, $\frac{dx}{dt} = f(x, \psi)$, where ψ is the bifurcation parameter, f is continuously differentiable at least twice in both x and ψ. The disease-free equilibrium is the line (x_0, ψ) and the local stability of the disease-free equilibrium at the point (x_0, ψ) (Van-den & Watmough, 2002). Now it shall show that there are non-trivial equilibrium near the bifurcation parameter.

Solving for $R_0 = 1$ gives

$$\beta_C = \beta_C^* = \frac{Q_1Q_2Q_3Q_4(1+\mu_A)\mu_v^2(\mu_H\sigma_m\Pi_v + \mu\sigma_H\Pi_H)^2}{\xi\Pi_v\Pi_H\beta_v\beta_C\mu_H(\eta_v\mu_v + \sigma_v)(\eta_H Q_2 + \eta_1\sigma_1)Q_3 + \eta_2\sigma_1\sigma_2)\sigma_H^2\sigma_m^2}.$$

By Lemma 3.3, the disease-free equilibrium $\mathcal{E}_0$ is locally stable when $\beta_C < \beta_C^*$ and unstable when $\beta_C > \beta_C^*$. Here $\beta_C - \beta_C^*$ is the bifurcation value. For convenience, let $S_H = x_1$, $E_H = x_2$, $I_{H1} = x_3$, $I_{H2} = x_4$, $R_H = x_5$, $A = x_6$, $S_v = x_7$, $E_v = x_8$, $I_v = x_9$, so that $N_H(t) = x_1 + x_2 + x_3 + x_4 + x_5$ and $N_v(t) = x_6 + x_7 + x_8 + x_9$. Further, by adopting the same vector notations with $x = (x_1, x_2, \ldots x_9)^T$, the model (1) can be written in the form $\frac{dx}{dt} = F(X)$ where $f = (f_1, f_2, \ldots f_9)^T$.

The Jacobian of the transformed system, evaluated at the DFE (E_0) with $\beta_C - \beta_C^*$ (denoted by (E_0)), is given by

$$J(E_0) = \begin{bmatrix} -\mu_H & 0 & 0 & 0 & 0 & 0 & 0 & -s_1 & -s_2 \\ 0 & -Q_1 & 0 & 0 & 0 & 0 & 0 & s_1 & s_1 \\ 0 & \sigma_1 & -Q_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_2 & -Q_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \tau_H & -\mu_H & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -Q_5 & 0 & 0 & 0 \\ 0 & -s_3 & -s_4 & -s_5 & 0 & \xi & -\mu_v & 0 & 0 \\ 0 & s_3 & s_4 & s_5 & 0 & 0 & 0 & -Q_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & Q_6 & 0 & \sigma_v & -\mu_v \end{bmatrix} \quad (7)$$

where $S_1 = \frac{\beta_H \sigma_m \sigma_H \eta_v \Pi_H \mu_v (1+\mu_A)}{(\Pi_H (1+\mu_A)\sigma_H + \sigma_m \Pi_v \mu_H)\mu_v + \sigma_m \Pi_v \mu_H \xi}$,

$S_2 = \frac{\beta_H \sigma_m \sigma_H \Pi_H \mu_v (1+\mu_A)}{(\Pi_H (1+\mu_A)\sigma_H + \sigma_m \Pi_v \mu_H)\mu_v + \sigma_m \Pi_v \mu_H \xi}$,

$S_3 = \frac{\beta_v \sigma_m \sigma_H \eta_H \xi \Pi_v \mu_H}{\sigma_m \Pi_v (\mu_v + \xi)\mu_H + \sigma_H \Pi_H \mu_v (1+\mu_A)}$, $S_4 = \frac{\beta_v \sigma_m \sigma_H \eta_1 \xi \Pi_v \mu_H}{\sigma_m \Pi_v (\mu_v + \xi)\mu_H + \sigma_H \Pi_H \mu_v (1+\mu_A)}$,

$S_5 = \frac{\beta_v \sigma_m \sigma_H \eta_2 \xi \Pi_v \mu_H}{\sigma_m \Pi_v (\mu_v + \xi)\mu_H + \sigma_H \Pi_H \mu_v (1+\mu_4)}$

The Jacobian $J(E_0)$ of the linearized system has a simple zero eigen value (with all other eigen values having negative real part). Using the notation in (Castito-Chavez & Song, 2004), the following computations are carried out.

Eigen vectors of $J(E_0)_{\beta_C = \beta_C^*}$: For the case when $R_0 = 1$ it can be shown that the $J(E_0)$ has a right eigen vector (Corresponding to the zero eigen value), given by $w = [w_1, w_2, \ldots, w_9]^T$ where,

$w_1 = \frac{-1}{\mu_H}\left[\frac{S_1 \mu_v (S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5) + S_2 \sigma_v (S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5)}{\sigma_1 \sigma_2 Q_4 \mu_v}\right] w_4$,

$w_2 = \frac{Q_2 Q_3}{\sigma_1 \sigma_2} w_4$, $w_4 = \frac{Q_3}{\sigma_2} w_4$, $w_4 > 0$, $w_5 = \frac{\tau_H}{\mu_H} w_4$, $w_6 = 0$,

$w_7 = -\frac{S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5}{\sigma_1 \sigma_2 \mu_v} w_4$, $w_8 = \frac{S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5}{\sigma_1 \sigma_2 Q_4} w_4$,

$w_9 = \frac{S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5}{\sigma_1 \sigma_2 \mu_v Q_4} \sigma_v w_4$. Similarly, the components of the left eigenvector of $J(E_0)$ (corresponding to the zero eigen value), denoted by $v = [v_1, v_2, \ldots, v_9]$, are given by,

$v_1 = 0$, $v_2 = \frac{\sigma_1\sigma_2 S_5 + \sigma_1 S_4 Q_3 + S_3 Q_2 Q_3}{Q_1 Q_2 S_5} v_4$, $v_3 = \frac{\sigma_2 S_5 + \sigma_1 S_4 Q_3}{Q_2 S_5} v_4$, $v_4 > 0$, $v_5 = 0$,

$v_6 = \frac{\sigma_1\sigma_2 S_2 S_5 + \sigma_1 S_2 S_4 Q_3 + S_2 Q_2 Q_3}{Q_1 Q_2 S_5} Q_6 v_4$, $v_7 = 0$, $v_8 = \frac{Q_3}{S_5} v_4$,

$v_9 = \frac{\sigma_1\sigma_2 S_2 S_5 + \sigma_1 S_2 S_4 Q_3 + S_2 Q_2 Q_3}{Q_1 Q_2 S_5} v_4$.

It is worth mentioning that the free right eigenvectors, v_4 and left eigenvector, v_4, are chosen to be $v_4 = 1$ and

$w_4 = \frac{1}{A_1 + A_2}$, where,

$A_1 = \frac{(\sigma_1\sigma_2 S_5 + \sigma_1 S_4 Q_3 + S_3 Q_2 Q_3) Q_2 Q_3 + (\sigma_2 S_5 + S_4 Q_3) Q_1 \sigma_1 Q_3 + Q_1 Q_2 \sigma_1 \sigma_2 S_5}{Q_1 Q_2 S_5 \sigma_1 \sigma_2}$ and

$A_2 = \frac{Q_1 Q_2 S_4 \mu_v (S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5) + (\sigma_1 \sigma_2 S_2 S_5 + \sigma_1 S_2 S_4 Q_3 + S_2 Q_2 Q_3)(S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5) \sigma_v S_5}{Q_1 Q_2 Q_3 S_4 S_5 \sigma_1 \sigma_2 \mu_v}$

, so that $v.w = 1$ (in line with (Castillo-Chavez & Song,2004)).It can be shown, by computing the non-zero partial derivatives of the right-hand side functions, f_i (i=1,...,9), that the associated backward bifurcation coefficients, a and b ,are given, respectively, by(seeTheorem4.1in(Castillo-Chavez & Song,2004)):

$$a = \sum_{k,i,j=1}^{n} v_k w_i w_j \frac{\partial^2 f_k(0,0)}{\partial x_i \partial x_j} = \frac{-2}{(\Pi_H(1+\mu_A)\sigma_H + \sigma_m \Pi_v \mu_H)\mu_v + \sigma_m \Pi_v \mu_H \xi^2} \mu_H (1+\mu_A)((-\Pi_H(1+\mu_A)(v_2(w_9 + w_8\eta_v)(w_2 + w_3 + w_4 + w_5)\beta_H - v_3 w_7 \beta_v(\eta_1 w_3 + \eta_H w_2 + \eta_2 w_4))\sigma_H + (-v_2\beta_H(1+\mu_A)(w_9 + w_8\eta_V)(w_7 + w_8 + w_9)\Pi_H + \Pi_V \mu_H(v_2 w_1(w_9 + w_8\eta_V)\beta_H + v_8 w_7 \beta_v(\eta_1 w_3 + w_2\eta_H + \eta_2 w_4)))\sigma_m)\mu_V + \Pi_V \mu_H(-v_8\beta_v(\eta_1 w_3 + w_2\eta_H + \eta_2 w_4)(w_1 + w_2 + w_3 + w_4 + w_5)\sigma_H + \sigma_m(v_2 w_1(w_9 + w_8\eta_V)\beta_H - v_8\beta_v(w_8 + w_9)(\eta_1 w_3 + \eta_H w_2 + \eta_2 w_4)))\xi)\mu_V \sigma_m \sigma_H$$

and $b = \sum_{k,i=1}^{n} v_k w_i \frac{\partial^2 f_k(0,0)}{\partial x_i \partial \beta_v^*} = \frac{(\eta_1 w_8 + \eta_H w_2 + \eta_2 w_4)\Pi_v \xi \mu_H \sigma_H \sigma_m v_8}{(\sigma_m \Pi_v(\mu_v + \xi)\mu_H + \sigma_H \Pi_H \mu_v(1+\mu_A))}$

$R_0 = 1$

$w_4 = \frac{1}{A_1 + A_2}$, where, $A_1 = \frac{(\sigma_1\sigma_2 S_5 + \sigma_1 S_4 Q_3 + S_3 Q_2 Q_3) Q_2 Q_3 + (\sigma_2 S_5 + S_4 Q_3) Q_1 \sigma_1 Q_3 + Q_1 Q_2 \sigma_1 \sigma_2 S_5}{Q_1 Q_2 S_5 \sigma_1 \sigma_2}$ and

$A_2 = \frac{Q_1 Q_2 S_4 \mu_v (S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5) + (\sigma_1 \sigma_2 S_2 S_5 + \sigma_1 S_2 S_4 Q_3 + S_2 Q_2 Q_3)(S_3 Q_2 Q_3 + \sigma_1 S_4 Q_3 + \sigma_1 \sigma_2 S_5) \sigma_v S_5}{Q_1 Q_2 Q_3 S_4 S_5 \sigma_1 \sigma_2 \mu_v}$

Since the coefficient *b* is automatically positive, it follows that the model (1) will undergo backward bifurcation if the coefficient *a*, is positive.

CONCLUSION

This paper presents a deterministic model for the transmission dynamics of chikungunya. The model, which realistically adopts a standard incidence formulation, allows chikungunya transmission by exposed humans and mosquitoes. The model is rigorously analyzed to gain insights into the qualitative dynamics of chikungunya. The main theoretical and epidemiological findings of the study show that:

- The TDFE of the model is GAS whenever the threshold quantity is less than or equal to one.
- The model exhibits the phenomenon of backward bifurcation, where the stable NDFE co-exists with a stable endemic equilibrium, even when the reproduction number is less than unity.

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