EUROPEAN JOURNAL OF PURE AND APPLIED MATHEMATICS
Vol. 12, No. 4, 2019, 1533-1552
ISSN 1307-5543 – www.ejpam.com
Published by New York Business Global

Mathematical analysis of an immune-structured *chikungunya* transmission model

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Abstract. In this paper, we have formulated a new deterministic model to describe the dynamics of the spread of *chikunguya* between humans and mosquitoes populations. This model takes into account the variation in mortality of humans and mosquitoes due to other causes than chikungunya disease, the decay of acquired immunity and the immune sytem boosting. From the analysis, it appears that the model is well posed from the mathematical and epidemiological standpoint. The existence of a single disease free equilibrium has been proved. An explicit formula, depending on the parameters of the model, has been obtained for the basic reproduction number $\mathcal{R}_0$ which is used in epidemiology. The local asymptotic stability of the disease free equilibrium has been proved. The numerical simulation of the model has confirmed the local asymptotic stability of the disease free equilbrium and the existence of endmic equilibrium. The varying effects of the immunity parameters has been analyzed numerically in order to provide better conditions for reducing the transmission of the disease.
2010 Mathematics Subject Classifications: 81T80, 92B05, 92D30, 93D20
Key Words and Phrases: Chikungunya, Differential equations, Asymptotic stability, Basic reproduction number, Modeling and numerical simulation.

1. Introduction

Chikungunya is an infectious disease caused by a virus transmited by the bite of female mosquitoes belonging to the genus *Aedes*: *Aedes aegypti* and *Aedes albopictus*. The *chikungunya* virus is present in sub-saharan Africa and in south-east Asia since 1952 [3]. It had been isolated for the first time in Tanzania and then spread to the rest of the world. It is estimated that *chikungunya* is an emerging desease in Asia. Many vaccine trials have been conducted since the 1970s but have proved ineffective [3]. Thus *chikungunya* disease is a major public health problem. In January 2013, the mortality caused by chikungunya was estimated at 1 per 1,000: most deaths occur in newborns, immunocompromised people and the elderly [9].

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DOI: https://doi.org/10.29020/nybg.ejpam.v12i4.3532

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Many mathematical models have been formulated to describe the dynamics of *chikungunya* transmission; see for example [2, 5, 8]. According to our knowledge the existing models do not simultaneously take into account the variation of the mortality due to the other causes than the disease, the decay of immunity and immune system boosting of the host. They consider a constant mortality rate and only one compartment for recovered hosts. Our model aims at correcting these inadequacies and proposing necessary conditions to stop the spread of the disease . In [6, 7] the authors have developed a deterministic model which describes the dynamics of malaria transmission. In their model they have used death rates linearly dependent on the total population for humans and mosquitoes. In [14], the authors have formulated a deterministic model in a populaton structured by immune status and they study waning immunity and immune system boosting. They have used three compartments of immune humans but they considered constant mortality rates and their model did not take into account vector-borne diseases. By combining these two approaches, we propose a model that best describes the transmission of *chikungunya*. The model we formulate is based on the principle of compartmental analysis described in [21, 22].

This paper is organized in five sections. The introduction is given in the first section. The second section is dedicated to the formulation of the model. We present the basic asumptions, the parameters and the variables used in the model. We describe the interactions between the different classes of the model and we formulate the model. In the third section we analyze the model. The existence, the positivity and the boudedness of the solutions is demonstrasted. The disease free equilibrium and the basic reproduction number are determined. The local asymptotic stability of the disease free equilibrium is studied. In the fourth section the theoretical results of the analysis are illustrated by a numerical simulation. In the fifth section we end the paper by a conclusion.

2. The mathematical model

We devide the humans population into five copartments and the mosquitos population into two compartments. The varibles of the model are given by Table 1 and the pameters are given Table 2 .

2.1. Assumptions

- Infected mosquito bites are the only ways to transmit viruses between mosquitoes and humans.
- At birth, all newborns are supposed to be suseptible in both populations.
- Infected mosquitoes remain infected throughout the rest of their lives.
- The disease does not kill mosquitoes.
- After contact with the chikungunya virus, recovered humans no longer carry viruses. So they can no longer transmit viruses to mosquitoes.

Table 1: Variables used in the model

Variables	Description
$S(t)$	Number of susceptible humans at time t.
$I(t)$	Number of infected humans at time t.
$R_F(t)$	Number of immune humans with high level of immunity at time t.
$R_W(t)$	Number of immune humans with intermediate level of immunity at time t.
$R_C(t)$	Number of immune humans with critically low level of immunity at time t.
$N_H(t)$	Total human population at t: $N_H(t) = S(t) + I(t) + R_F(t) + R_W(t) + R_C(t)$.
$S_V(t)$	Number of susceptible mosquitoes at time t.
$I_V(t)$	Number of infective mosquitoes at time t.
$N_V(t)$	Total mosquito population at time t: $N_V(t) = S_V(t) + I_V(t)$.

- Recovered humans acquire an immunity; this immunity gradually decreases several years after recovery when a recovered human is no longer exposed to viruses.
- New exposure to viruses boosts the immune system then prolong the time in which a recovered individual is immune.
- The mortality induced by other causes than the desease depends on the total population for both human and mosquitoe populations and is given by $f_H(N_H) = d_H + d_{2H}N_H$ and $f_V(N_V) = d_V + d_{2V}N_V$ respectively for humans and mosquitoes; see [7, 15].

2.2. The interactions

Per time unit, $b_H N_H(t)$ humans and $b_V N_V(t)$ mosquitoes are born in the susceptible compartments S and S_V respectively. Due to bites by infcted mosquitoes, $\beta_H \frac{I_V}{N_H} S$ susceptible humans and $\beta_V \frac{I}{N_H} S_V$ susceptible mosquitoes are newly infected. Then they enter the infected compartments I and I_V respectively. γI infected humans are recovered and enter the compartment R_F with high level of immunity. When there is no new exposure after recovery, the immunity acquired by humans decays progressively and is lost. We consider three levels of immunity and we divide human population into five compartments of humans; see Table 1. μR_F humans with high level of immunity will leave R_F compartment and enter the compartment R_W of intermediate level of immunity while λR_W humans leave R_W compartment and enter the compartment R_C of critically low level of immunity and σR_C humans leave R_C compartment and return in the susceptible compartment S. When there is new exposure some humans in the R_W and R_C compartments get their immune system boosted. Thus $\beta_V \frac{I_V}{N_H}(1-\theta)R_C$ humans of R_C compartment and $\beta_H \frac{I_V}{N_H} R_W$ humans of R_W return into R_F compartment; $\beta_H \frac{I_V}{N_H}\theta R_C$ humans of R_C compartment return into R_W compartment.
Per time unit, $f_V S_V$ susceptible mosquitoes and $f_V I_V$ infected mosquitoes leave the

Table 2: Parameters used in the model

Parameters	Meaning
b_H	Humans birth rate.
b_V	Mosquitoes birth rate.
p_{vh}	Probability of virus transmission from infected mosquitoe to susceptible human.
p_{iv}	Probability of virus transmission from an infected human to a susceptible mosquito.
n_v	Number of bites done by a mosquito on the whole human population.
β_H	Average number of bites that cause a viruses transmission from infected mosquitoes to susceptible humans. $\beta_H = n_v p_{vh}$.
β_V	Average number of bites that cause a viruses transmission from infected humans. to susceptible mosquitoes. $\beta_V = n_v p_{iv}$.
d_H	Density independent part of the death rate for humans.
d_{2H}	Density dependent part of the death rate for humans.
d_V	Density independent part of the death rate for mosquitoes.
d_{2V}	Density dependent part of the death rate for mosquitoes.
d_I	*Chikungunya* induced death rate in human population
γ	Recovery rate. $\frac{1}{\gamma}$ is the average duration of the infectious period.
μ	Immunity decay rate from high level R_F to intermediate level R_W.
λ	Immunity decay rate from intermediate level R_W to critially low level R_C.
σ	Immunity loss rate.
θ	Immune system boosting probability from critially low level R_C to intermediate level R_W. $1-\theta$ is Immune system boosting probability from R_C to high level R_F.

mosquito population due to death. $f_H S$, $(f_H + d_I)I$, $f_H R_F$, $f_H R_W$ and $f_H R_C$ humans die respevtively in S, I, R_F, R_W and R_C compartments.
The quantities used in the interactions are all positive. The interactions described are illustrated by Figure 1.

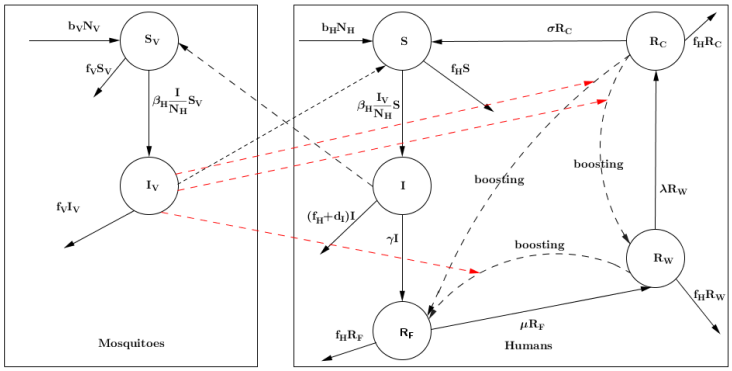

Figure 1: A compartmental model of the chikungunya virus transmission.

The arrows with full lines represent the transfers of individuals. The arrows with dotted lines indicate the direction of the infection: from humans to mosquitoes and from mosquitoes to humans. The red arrows with dotted lines indicate that immune system boost is due to bites of infected mosquitoes.

2.3. The mathematical model

Using the interactions illustrated by Figure 1, the dynamics of the transmission given by the system of non linear differential equations (1).

$$
\begin{cases}
\dfrac{dS}{dt} = b_H N_H + \sigma R_C - \beta_H \dfrac{I_V}{N_H} S - d_H S - d_{2H} N_H S \\
\dfrac{dI}{dt} = \beta_H \dfrac{I_V}{N_H} S - (\gamma + d_H + d_I) I - d_{2H} N_H I \\
\dfrac{dR_F}{dt} = \gamma I - (\mu + d_H) R_F - d_{2H} N_H R_F + \beta_H \dfrac{I_V}{N_H} ((1-\theta) R_C + R_W) \\
\dfrac{dR_W}{dt} = \mu R_F - (d_H + \lambda) R_W - d_{2H} N_H R_W + \beta_H \dfrac{I_V}{N_H} (\theta R_C - R_W) \\
\dfrac{dR_C}{dt} = \lambda R_W - (d_H + \sigma) R_C - d_{2H} N_H R_C - \beta_H \dfrac{I_V}{N_H} R_C \\
\dfrac{dS_V}{dt} = b_V N_V - d_V S_V - d_{2V} N_V S_V - \beta_V \dfrac{I}{N_H} S_V \\
\dfrac{dI_V}{dt} = \beta_V \dfrac{I}{N_H} S_V - d_V I_V - d_{2V} N_V I_V \\
\dfrac{dN_H}{dt} = (b_H - d_H) \left(1 - \dfrac{N_H}{\dfrac{b_H - d_H}{d_{2H}}} \right) N_H - d_I I \\
\dfrac{dN_V}{dt} = (b_V - d_V) \left(1 - \dfrac{N_V}{\dfrac{b_V - d_V}{d_{2V}}} \right) N_V
\end{cases}
\tag{1}
$$

where $\beta_H = n_v p_{vh}$, $\beta_V = n_v p_{iv}$.
$\frac{b_H - d_H}{d_{2H}}$ is the carrying capacity for humans population and $\frac{b_V - d_V}{d_{2V}}$ is the carrying capacity for mosquitoes population. That is to say the maximum humans and the maximum mosquitoes the ecosystem is able to sustain indefinitely.

Remark 1.
The carrying capacities for human population and mostiquitoes population are strictly positve. Thus $b_H > d_H$ and $b_V > d_V$. In the rest of this paper, we consider that $b_H > d_H$ and $b_V > d_V$.

We disignate the initial condition of the model, that is to say the initial number of individuals in the different compartments, by $\left(\ S_0,\ I_0,\ R_{F_0},\ R_{W_0},\ R_{C_0},\ S_{V_0},\ I_{V_0},\ N_{H_0},\ N_{V_0} \ \right)^t$.

3. Mathematical analysis of the model

3.1. Existence, positivity and boundedness of the solutions

To prove that the model is well posed from the mathematical and epidmiological standpoint, we begin by defining the folowing sets:

$$\Omega_1 = \left\{ \left(\ S,\ I,\ R_F,\ R_W,\ R_C,\ S_V,\ I_V \ \right)^t \in \mathbb{R}_+^7 \right\}, \tag{2}$$

$$\Omega_2 = \left\{ \left(\ N_H,\ N_V \ \right)^t \ \middle| \ \begin{array}{l} 0 < N_H \leq \dfrac{b_H - d_H}{d_{2H}} \\ \\ 0 < N_V \leq \dfrac{b_V - d_V}{d_{2V}} \end{array} \right\} \tag{3}$$

and

$$\Omega = \Omega_1 \times \Omega_2.$$

Noting the elements of Ω by $x = \left(\ S,\ I,\ R_F,\ R_W,\ R_C,\ S_V,\ I_V,\ N_H,\ N_V \ \right)^t$, we rewrite the model (1) as the following autonomous differential system:

$$\frac{dx_i}{dt} = f_i(x);\ i = 1; 2; ...; 9 \tag{4}$$

We can state the following result:

Proposition 1.
For all initial condition $x_0 = \left(\ S_0,\ I_0,\ R_{F_0},\ R_{W_0},\ R_{C_0},\ S_{V_0},\ I_{V_0},\ N_{H_0},\ N_{V_0} \ \right)^t$ in Ω, the model (1) admits a unique solution defined for all time $t \geq 0$.

Proof

Each component f_i of the function f is given by the member on the right of the i-th equation of the model (1) which is a sum of polynomials and rational functions defined on Ω. It shows that $f \in \mathcal{C}^\infty(\Omega)$ and in particular $f \in \mathcal{C}^1(\Omega)$. So f is locally lipschitzian on Ω. Thus, by the Cauchy-Lipschitz theorem [4], for any initial condition $x_0 = \left(S_0,\ I_0,\ R_{F_0},\ R_{W_0},\ R_{C_0},\ S_{V_0},\ I_{V_0},\ N_{H_0},\ N_{V_0} \right)^t \in \Omega$, the model (1) admits a unique maximum solution defined for all time $t \geq 0$ ■

Theorem 1.

For all initial condition $x_0 \in \Omega$ it exits, for the model (1), a unique solution that stays in Ω for all time $t \geq 0$.

Proof

The proposition 1 guarantees the existence and the uniqueness of the solution for each initial condition. Now we must prove that Ω is positively invariant by the system (1). That is to say for $S_0 \geq 0$, $I_0 \geq 0$,
$R_{F_0} \geq 0,\ R_{W_0} \geq 0, R_{C_0} \geq 0,\ S_{V_0} \geq 0,\ I_{V_0} \geq 0,\ 0 < N_{H_0} \leq \dfrac{b_H - d_H}{d_{2H}}$ and $0 < N_{V_0} \leq \dfrac{b_V - d_V}{d_{2V}}$, the solution verifying this initial condition also satisfies the condition
$S \geq 0,\ I \geq 0,\ R_F \geq 0,\ R_W \geq 0, R_C \geq 0,\ S_V \geq 0,\ I_V \geq 0,$
$0 < N_H \leq \dfrac{b_H - d_H}{d_{2H}}$ and $0 < N_V \leq \dfrac{b_V - d_V}{d_{2V}}$.

To show the positivity of the solutions of the model we express each differential equation of the system (1) as a differential inequality and we use the technique of separation of variables. After integration of the different differential inequalities, we obtain the positivity of $S,\ I,\ R_F,\ R_W,\ R_C,\ S_V,\ I_V,\ N_H$ and N_V. This approach is also used in [1],[18].

Positivity of the number of susceptible humans S:

As $b_H N_H + \sigma R_C \geq 0$,

$$\frac{dS}{dt} \geq -\beta_H \frac{I_V}{N_H} S - d_H S - d_{2H} N_H S. \tag{5}$$

Separating the variables and integrating we obtain

$$S \geq S_0 \exp\left(-\beta_H \frac{I_V}{N_H} - d_H - d_{2H} N_H \right). \tag{6}$$

As $\exp\left(-\beta_H \dfrac{I_V}{N_H} - d_H - d_{2H} N_H \right) > 0$,
$S_0 \exp\left(-\beta_H \dfrac{I_V}{N_H} - d_H - d_{2H} N_H \right) \geq 0$ for $S_0 \geq 0$. Thus $S \geq 0$ for $S_0 \geq 0$.

Positivity of the number of infected humans I:

$\beta_H \dfrac{I_V}{N_H} S \geq 0$. That implies that

$$\frac{dI}{dt} \geq -(\gamma + d_H + d_I) I - d_{2H} N_H I \tag{7}$$

By separating the variables and integrating, we have

$$I \geq I_0 \exp\left(-(\gamma + d_H + d_I) - d_{2H} N_H\right). \tag{8}$$

We deduce that $I \geq 0$ for $I_0 \geq 0$.

Positivity of the number of immune humans with high level of immunity R_F:
As $\gamma I + \beta_H \dfrac{I_V}{N_H}\left((1-\theta)R_C + R_W\right) \geq 0$,

$$\frac{dR_F}{dt} \geq -(\mu + d_H)R_F - d_{2H} N_H R_F. \tag{9}$$

By separating the variables and integrating,

$$R_F \geq R_{F_0} \exp(-(\mu + d_H) - d_{2H} N_H R). \tag{10}$$

We deduce that $R_F \geq 0$ for $R_{F_0} \geq 0$.

Positivity of the number of immune humans with intermediate level of immunity R_W:
As $\mu R_F + \beta_H \dfrac{I_V}{N_H}\left(\theta R_C - R_W\right) \geq 0$,

$$\frac{dR_W}{dt} \geq -(d_H + \lambda)R_W - d_{2H} N_H R_W. \tag{11}$$

By separating the variables and integrating,

$$R_W \geq R_{W_0} \exp(-(d_H + \lambda) - d_{2H} N_H). \tag{12}$$

Thus $R_W \geq 0$ for $R_{W_0} \geq 0$.

Positivity of the number of immune humans with critically low level of immunity R_C:
$\lambda R_W \geq 0$. That implies that

$$\frac{dR_C}{dt} \geq -(d_H + \sigma)R_C - d_{2H} N_H R_C - \beta_H \frac{I_V}{N_H} R_C. \tag{13}$$

By separating the variables and integrating, we obtain

$$R_C \geq R_{W_0} \exp\left(-(d_H + \sigma) - d_{2H} N_H - \beta_H \frac{I_V}{N_H}\right). \tag{14}$$

Thus $R_C \geq 0$ for $R_{C_0} \geq 0$.

Positivity of the number of susceptible mosquitoes S_V:
$b_V N_V \geq 0$. That implies that

$$\frac{dS_V}{dt} \geq -d_V S_V - d_{2V} N_V S_V - \beta_V \frac{I}{N_H} S_V. \tag{15}$$

By separating the variables and integrating,

$$S_V \geq S_{V_0} \exp\left(-d_V - d_{2V} N_V - \beta_V \frac{I}{N_H}\right). \tag{16}$$

Thus $S_V \geq 0$ for $S_{V_0} \geq 0$.

Positivity of the number of infected mosquitoes I_V:

$\beta_V \frac{I}{N_H} S_V \geq 0$. That implies that

$$\frac{dI_V}{dt} \geq -d_V I_V - d_{2V} N_V I_V. \tag{17}$$

By separating the variables and integrating,

$$I_V \geq I_{V_0} \exp\left(-d_V - d_{2V} N_V\right). \tag{18}$$

We deduce that $I_V \geq 0$ for $I_{V_0} \geq 0$.

We have shown that $\forall \left(S_0,\ I_0,\ R_{F_0},\ R_{W_0},\ R_{C_0},\ S_{V_0},\ I_{V_0},\ N_{H_0},\ N_{V_0} \right)^t \in \Omega$, $\left(S,\ I,\ R_F,\ R_W,\ R_C,\ S_V,\ I_V \right)^t \in \Omega_1$. Let's show now that $\forall \left(S_0,\ I_0,\ R_{F_0},\ R_{W_0},\ R_{C_0},\ S_{V_0},\ I_{V_0},\ N_{H_0},\ N_{V_0} \right)^t \in \Omega$, $\left(N_H,\ N_V \right)^t \in \Omega_2$. For that we must show that $0 < N_H \leq \frac{b_H - d_H}{d_{2H}}$ and $0 < N_V \leq \frac{b_V - d_V}{d_{2V}}$.

Boundedness of the solutions:

$N_H(t) = S(t) + I(t) + R_F(t) + R_W(t) + R_C(t)$.

$S(t),\ I(t),\ R_F(t),\ R_W(t)$ and $R_C(t)$ are positive. Thus N_H is positive.

From $\frac{dN_H}{dt} = (b_H - d_H)\left(1 - \frac{N_H}{\frac{b_H - d_H}{d_{2H}}}\right) N_H - d_I I$, we obtain the inequality

$$\frac{dN_H}{dt} \leq \left(b_H - d_H - d_{2H} N_H\right) N_H. \tag{19}$$

After separating the variables and integrating, we obtain

$$N_H(t) \leq \frac{b_H - d_H}{d_{2H}} \left(1 - \exp(-(b_H - d_H)t) + N_{H_0} \exp(-(b_H - d_H)t)\right). \tag{20}$$

Calculating the limit of $N_H(t)$ when $t \to +\infty$ we finally obtain

$$N_H(t) \leq \frac{b_H - d_H}{d_{2H}}. \tag{21}$$

As $N_V(t) = S_V(t) + I_V(t)$, $N_V(t)$ is positive.

$\frac{dN_V}{dt} = (b_V - d_V)\left(1 - \frac{N_V}{\frac{b_V - d_V}{d_{2V}}}\right) N_V$.

After integrating we obtain

$$N_V(t) = \frac{N_{V_0}(b_V - d_V)}{d_{2V} N_{V_0} + \left(b_V - d_V - d_{2V} N_{V_0}\right) e^{-(b_V - d_V)t}}. \tag{22}$$

$$\lim_{t\to+\infty} N_V(t) = \frac{b_V - d_V}{d_{2V}}. \tag{23}$$

From the relation 22 we find

$$\frac{dN_V}{dt}(t) = N_{V_0}\left(\frac{b_V - d_V}{d_{2V}N_{V_0} + (b_V - d_V - d_{2V}N_{V_0})e^{-(b_V-d_V)t}}\right)^2 (b_V - d_V - d_{2V}N_{V_0})\, e^{-(b_V-d_V)t} \tag{24}$$

For $\left(\ N_H,\ N_V\ \right)^t \in \Omega_2$, the relation 24 implies that $\dfrac{dN_V}{dt}(t) \geq 0$. It means that N_V is an increasing function. Using the relation 23, we obtain

$$\forall t \in [0\ ;\ +\infty[,\ N_{V_0} \leq N_V(t) \leq \frac{b_V - d_V}{d_{2V}}. \tag{25}$$

Thus $0 < N_V(t) \leq \dfrac{b_V - d_V}{b_{2V}}$. Therefore $\left(\ N_H,\ N_V\ \right)^t \in \Omega_2$ ■

Theorem 1 shows that the model (1) is well posed from a mathematical and epidemiological standpoint.

3.2. Disease free equilibrium and basic reproduction number $\mathcal{R}_0$

3.2.1. Disease free equilibrium

Theorem 2.
The model (1) has a unique disease free equilibrium given by

$$E_{dfe} = \left(\ \frac{b_H - d_H}{d_{2H}},\ 0,\ 0,\ 0,\ 0,\ \frac{b_V - d_V}{d_{2V}},\ 0,\ \frac{b_H - d_H}{d_{2H}},\ \frac{b_V - d_V}{d_{2V}}\ \right)^t. \tag{26}$$

Proof
When there is no disease, $I = I_V = 0$. We note the disease free equilibrium by $E_{dfe} = \left(\ S^s,\ 0,\ R_F^s,\ R_W^s,\ R_C^s,\ S_V^s,\ 0,\ N_H^s,\ N_V^s\ \right)^t$ where N_H^s and N_V^s are the total populations respectively of humans and mosquitoes at the equilibrium. So $N_H \neq 0$ and $N_V \neq 0$.

By replacing I and I_V by 0 in the model (1), we find E_{dfe} after solving the sytem below:

$$\begin{cases} b_H N_H + \sigma R_C - d_H S^s - d_{2H} N_H S^s = 0 \\ -(\mu + d_H) R_F^s - d_{2H} N_H^s R_F^s = 0 \\ -(\mu + d_H) R_F^s - d_{2H} N_H^s R_F^s = 0 \\ \mu R_F^s - (d_H + \lambda) R_W^s - d_{2H} N_H^s R_W^s = 0 \\ \lambda R_W^s - (d_H + \sigma) R_C^s - d_{2H} N_H^s R_C^s = 0 \\ b_V N_V^s - d_V S_V^s - d_{2V} N_V^s S_V^s = 0 \\ (b_H - d_H)\left(1 - \dfrac{N_H}{\dfrac{b_H - d_H}{d_{2H}}}\right) N_H = 0 \\ (b_V - d_V)\left(1 - \dfrac{N_V}{\dfrac{b_V - d_V}{d_{2V}}}\right) N_V = 0 \end{cases} \tag{27}$$

After solving, we find $N_H^s = \dfrac{b_H - d_H}{d_{2H}}$, $N_V^s = \dfrac{b_V - d_V}{d_{2V}}$, $S^s = N_H^s$, $S_V^s = N_V^s$ and $R_F = R_C = R_W = 0$. Thus

$$E_{dfe} = \left(\frac{b_H - d_H}{d_{2H}},\ 0,\ 0,\ 0,\ 0,\ \frac{b_V - d_V}{d_{2V}},\ 0,\ \frac{b_H - d_H}{d_{2H}},\ \frac{b_V - d_V}{d_{2V}} \right)^t \blacksquare$$

3.2.2. Basic reproduction number

The basic reproduction number is the average number of secondary infections produced by a single infected individual in a population completely susceptible. We determine the basic reproduction number by using the next generation method described in [11, 17]. After *chikungunya* disease, recovered humans no longer have virus in their body. So the infected compartments are only I and I_V. We define the new infections rates in the infected comparments by the relation (28) and the exchange rates of each infected compartment

with the other compartments is given by the relation (29).

$$\mathcal{F}(X) = \begin{pmatrix} \mathcal{F}_I(X) \\ \\ \mathcal{F}_{I_V}(X) \end{pmatrix} = \begin{pmatrix} \beta_H \dfrac{I_V}{N_H} S \\ \\ \beta_V \dfrac{I}{N_H} S_V \end{pmatrix}, \tag{28}$$

$$\mathcal{V}(X) = \begin{pmatrix} \mathcal{V}_I(X) \\ \\ \mathcal{V}_{I_V}(X) \end{pmatrix} = \begin{pmatrix} (\gamma + d_H + d_I)I + d_{2H} N_H I \\ \\ d_V I_V + d_{2V} N_V I_V \end{pmatrix} \tag{29}$$

With these functions, we calculate the next generation matrix FV^{-1} where F and V are the following matrices:

$$F = \begin{pmatrix} \dfrac{\partial \mathcal{F}_I}{\partial I}(E_{dfe}) & \dfrac{\partial \mathcal{F}_I}{\partial I_V}(E_{dfe}) \\ \\ \dfrac{\partial \mathcal{F}_{I_V}}{\partial I}(E_{dfe}) & \dfrac{\partial \mathcal{F}_{I_V}}{\partial I_V}(E_{dfe}) \end{pmatrix} = \begin{pmatrix} 0 & \beta_H \\ \\ \beta_V \dfrac{N_V^s}{N_H^s} & 0 \end{pmatrix} \tag{30}$$

$$V = \begin{pmatrix} \dfrac{\partial \mathcal{V}_I}{\partial I}(E_{dfe}) & \dfrac{\partial \mathcal{V}_I}{\partial I_V}(E_{dfe}) \\ \\ \dfrac{\partial \mathcal{V}_{I_V}}{\partial I}(E_{dfe}) & \dfrac{\partial \mathcal{V}_{I_V}}{\partial I_V}(E_{dfe}) \end{pmatrix} = \begin{pmatrix} b_H + \gamma + d_I & 0 \\ \\ 0 & b_V \end{pmatrix} \tag{31}$$

Thus the next generation matrix is given by

$$FV^{-1} = \begin{pmatrix} 0 & \dfrac{\beta_H}{b_V} \\ \\ \beta_V \dfrac{N_V^s}{N_H^s (b_H + \gamma + d_I)} & 0 \end{pmatrix} \tag{32}$$

$\mathcal{R}_0$ is the spectral radius of the next generation matrix; $\mathcal{R}_0 = \rho\left(FV^{-1}\right)$. After detrmining the eigenvalues of FV^{-1}, we obtain $\mathcal{R}_0 = \sqrt{\dfrac{\beta_H \beta_V N_V^s}{N_H^s \left(b_H + \gamma + d_I\right) b_V}}$.

We can also write $\mathcal{R}_0 = \sqrt{\mathcal{R}_{0H} \mathcal{R}_{0V}}$ where $\mathcal{R}_{0H} = \dfrac{\beta_H}{b_V}$ and $\mathcal{R}_{0V} = \dfrac{\beta_V N_V^s}{N_H^s \left(b_H + d_I + \gamma\right)}$.

$\mathcal{R}_{0H}$ is the average number of secondary infections produced by a single infected mosquito in a population of humans all susceptibles. $\mathcal{R}_{0V}$ is the average number of secondary infections produced by a single infected human in a population of mosquitoes all susceptibles. Replacing β_H and β_V respectively by $n_v p_{vh}$ and $n_v p_{iv}$, we obtain $\mathcal{R}_{0H} = \dfrac{n_v p_{vh}}{b_V}$,

$\mathcal{R}_{0V} = \dfrac{n_v p_{iv} N_V^s}{N_H^s\,(b_H + d_I + \gamma)}$ and $\mathcal{R}_0 = n_v \sqrt{\dfrac{p_{v_h} p_{iv} N_V^s}{N_H^s\,(b_H + \gamma + d_I)\, b_V}}$.

Substituting N_H^s by $\dfrac{b_H - d_H}{d_{2H}}$ and N_V^s by $\dfrac{b_V - d_V}{d_{2V}}$ we finally obtain

$$\mathcal{R}_0 = n_v \sqrt{\frac{d_{2H} p_{vh} p_{iv} (b_V - d_V)}{b_V d_{2V} (b_H - d_H)(b_H + \gamma + d_I)}}. \tag{33}$$

3.2.3. Stability of disease free equilibrium

Theorem 3.
The disease free equilibrium E_{dfe} is locally asymptoptically stable for $\mathcal{R}_0 < 1$ and unstable for $\mathcal{R}_0 > 1$.

Proof
The jacobian matrix of the system (1) at the disease free equilibrium is given below:

$$\mathcal{J} = \begin{pmatrix} -b_H & 0 & 0 & 0 & \sigma & 0 & -\beta_H & d_H & 0 \\ 0 & \mathcal{J}_{22} & 0 & 0 & 0 & 0 & \beta_H & 0 & 0 \\ 0 & \gamma & \mathcal{J}_{33} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mu & \mathcal{J}_{44} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \lambda & \mathcal{J}_{55} & 0 & 0 & 0 & 0 \\ 0 & -\beta_V & 0 & 0 & 0 & -b_V & 0 & 0 & d_V \\ 0 & \beta_V \dfrac{N_V^*}{N_H^*} & 0 & 0 & 0 & 0 & -b_V & 0 & 0 \\ 0 & -d_I & 0 & 0 & 0 & 0 & 0 & \mathcal{J}_{88} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -d_{I_V} & 0 & \mathcal{J}_{99} \end{pmatrix}$$

where $\mathcal{J}_{22} = -(b_H + \gamma + d_I)$; $\mathcal{J}_{33} = -(b_H + \mu)$; $\mathcal{J}_{44} = -(b_H + \lambda)$;
$\mathcal{J}_{55} = -(b_H + \sigma)$; $\mathcal{J}_{88} = -(b_H - d_H)$; $\mathcal{J}_{99} = -(b_V - d_V)$.
The characteristic polynomial of $\mathcal{J}$ is given by

$$P(x) = (x + b_H)(x + b_V)(x - \mathcal{J}_{33})(x - \mathcal{J}_{44})(x - \mathcal{J}_{55})(x - \mathcal{J}_{88})(x - \mathcal{J}_{99})T(x) \tag{34}$$

where

$$T(x) = x^2 + (b_H + \gamma + d_I + b_V)x + b_V(b_H + \gamma + d_I)\left(1 - \mathcal{R}_0^2\right). \tag{35}$$

$-b_H,\ -b_V,\ -(b_H+\mu),\ -(b_H+\lambda),\ -(b_H+\sigma), -(b_H-d_H),\ -(b_V-d_V)$ are eigenvalues of $\mathcal{J}$ which are all negative reals. The other eigenvalues of $\mathcal{J}$ are the roots of the polynomial T.
For $\mathcal{R}_0 < 1$, the coefficients of T namely 1, $(b_H + \gamma + d_I + b_V)$ and
$b_V(b_H + \gamma + d_I)\left(1 - \mathcal{R}_0^2\right)$ are all strictly positive. For $\mathcal{R}_0 > 1$ the coefficients 1 and $(b_H + \gamma + d_I + b_V)$ are strictly positive while the coefficients

$b_V(b_H+\gamma+d_I)\left(1-\mathcal{R}_0^2\right)$ is strictly negative. As T is a polynomial of second degree, the criterion of Routh-Hurwitz [13] allows us to affirm that the roots of T have all their real parts strictly negative for $\mathcal{R}_0<1$ and T has at least an eigenvalue whose real part is strictly positive for $\mathcal{R}_0>1$. Thus the eigenvalues of $\mathcal{J}$ have all their real parts strictly negative for $\mathcal{R}_0<1$ and at least an eigenvalue of $\mathcal{J}$ has a strictly positive real part for $\mathcal{R}_0>1$. Therefore E_{dfe} is locally asymptotically stable for $\mathcal{R}_0<1$ and unstable for $\mathcal{R}_0>1$ ■

The proof of Theorem 3 is based on the analysis of the characteristic polynomial of Jacobian matrix, see [16] for a similar approach.

Due to the complexity of the system (1), the existence of endemic equilibrium is not shown analytically. However, we can confirm the existence of endemic equilibrium numerically in the next section.

4. Numerical simulation

In this section, we illustrate by the numerical simulation the theoretical results obtained from the analysis; see Theorem 3 . Our objective is to show the
conformity between the theoretical results of the analysis and the numerical results. We use MATLAB ode45; see [10, 12, 19, 20].
We set the initial condition to $S_0=4950,\ I_0=50,\ R_{F_0}=0,\ R_{W_0}=0,\ R_{C_0}=0,\ S_{V_0}=9900,\ I_{V_0}=100,\ N_{H_0}=5000$ and $N_{V_0}=10000$.
We take the same values for b_H, d_H, d_{2H}, b_V, d_V and d_{2V} as in [6] and we assume that an infectious human will stay infectious during 100 days.
$\frac{1}{\gamma}$ is the average time that an infectious human will stay in the infectious compartment.
By analogy $\frac{1}{\mu}$ is the average time that a human of R_F compartment will stay in this compartment before moving to R_W compartment; $\frac{1}{\lambda}$ is the average time that a human of R_W compartment will stay in this compartment before moving to R_C compartment and $\frac{1}{\sigma}$ is the average time that a human of R_C compartment will stay in this compartment before loosing his immunity.
A recovered human acquire lasting immunity. Several years are necessary so that immunity is lost. We estimate that one year lasts 365 days. We assume that, when there is no new exposure, immunity will decay to the intermediate level after 25 years, to the critically low level after 35 years and it is lost after 45 years.
When there is new exposure, we assume that the probability of immune system boosting from critically low level to intermediate level is $\theta=0.6$. It means that $1-\theta=0.4$. The values used for the numerical simulation are given by Table 3. With these values, we obtain $E_{dfe}=\left(\ 133280,\ 0,\ 0,\ 0,\ 0,\ 12125,\ 0,\ 133280,\ 12125\ \right)^t$.

Table 3: Values used for the numerical simualtion.

Parameters	Values	References
b_H	0.04	[6]
b_V	0.13	[6]
p_{vh}	0.07	[6]
p_{iv}	0.45	[6]
d_H	1.6×10^{-5}	[6]
d_{2H}	3×10^{-7}	[6]
d_V	0.033	[6]
d_{2V}	8×10^{-6}	[6]
d_I	0.001	[9]
γ	0.01	assumed
μ	1.096×10^{-4}	assumed
λ	7.828×10^{-5}	assumed
σ	6.088×10^{-5}	assumed
θ	0.6	assumed

4.1. The varying effects of $\mathcal{R}_0$ on the behaviour of the model

We fix the values of Table 3 and we choose n_v appropriately so that $\mathcal{R}_0 < 1$ on the one hand and $\mathcal{R}_0 > 1$ on the other hand.
For $n_v = 1.1408$, $\mathcal{R}_0 = 0.75$. Then the dynamics of the transmission is given by Figure 2 for humans and Figure 3 for the mosquitoes.

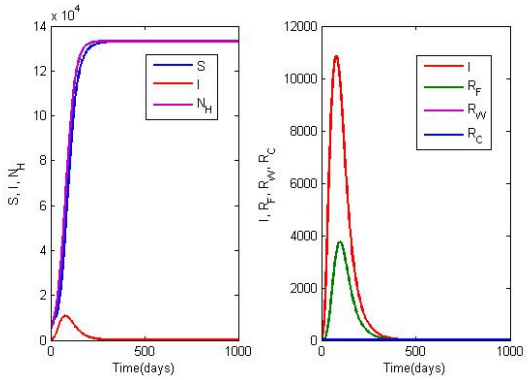

Figure 2: Dynamics of the transmission in the human population for $\mathcal{R}_0 = 0.75 < 1$.

The disease will die out and all humans will be susceptible.

With $n_v = 11.4079$, we obtain $\mathcal{R}_0 = 7.5 > 1$. Then the dynamics of the transmission is given by the Figure 4 for humans and Figure 5 for the mosquitoes.
Figure 2 and Figure 3 show that the solution of the model approaches the disease free eqilibrium when $\mathcal{R}_0 < 1$. This confirms the stability of the desease free equilibrium. Figure 4 and Figure 5 show that the solution of the model does not approache the disease free

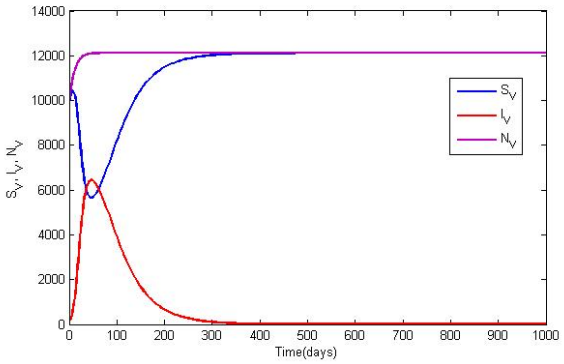

Figure 3: Dynamics of the transmission in the mosquioes population for $\mathcal{R}_0 = 0.75 < 1$.

The disease will die out and all mosquitoes will be susceptible.

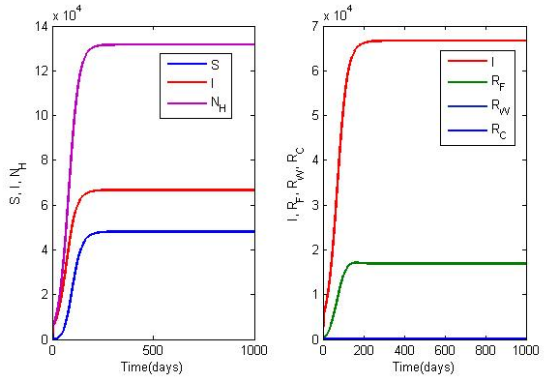

Figure 4: Dynamics of the transmission in the human population for $\mathcal{R}_0 = 7.5 > 1$.

There are infected humans every time.

eqilibrium when $\mathcal{R}_0 > 1$. There will always be infected humans and infected mosquitoes when $\mathcal{R}_0 > 1$. This confirms the existence of endemic equilibrium of the system (1) when $\mathcal{R}_0 > 1$.

4.2. The varying effects of the immunity parameters on the behaviour of the model

In this subsection we provide further simulations showing the varying effects of the immunity parameters (μ, λ, σ and θ) on the behaviour of the system (1). We analyze these effects for $\mathcal{R}_0 > 1$. For each parameter, we fixe the other values of table 3 and we simulate the varying effects of the parameter concerned. Figures 6, 7 and 8 show the varying effects of μ, λ and σ respectively on the dynamics of the infected humans. As for θ, its varying effects are difficult to be observed by the curve of I but the curve of R_W shows clearly its varying effects; see Figure 9.

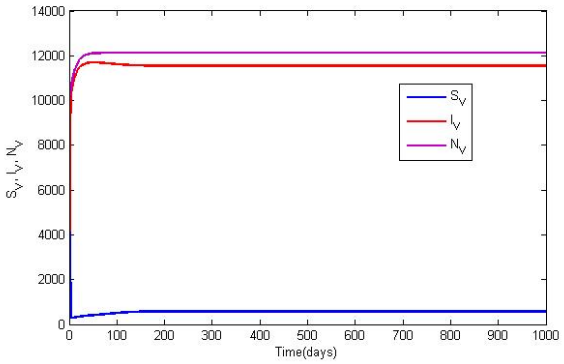

Figure 5: Dynamics of the transmission in the population of mosquitoes for $\mathcal{R}_0 = 7.5 > 1$.

There are infected mosquitoes every time.

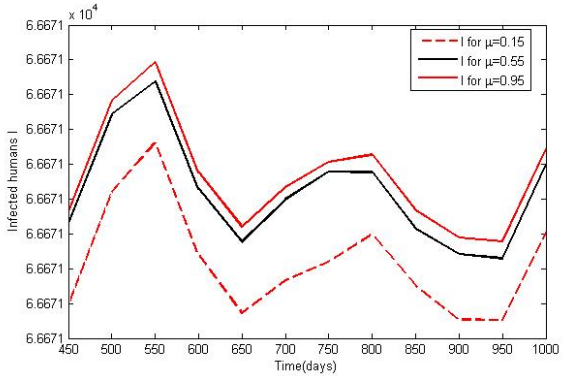

Figure 6: Varying effects of μ.

The increase of μ towards 1 favors the transmission of the disease in the human population.

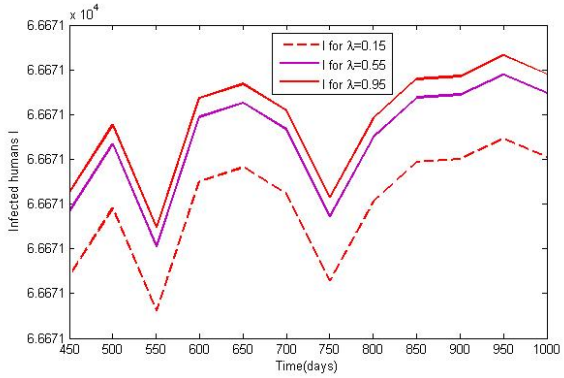

Figure 7: Varying effects of λ.

The increase of λ towards 1 favors the transmission of the disease in the human population.

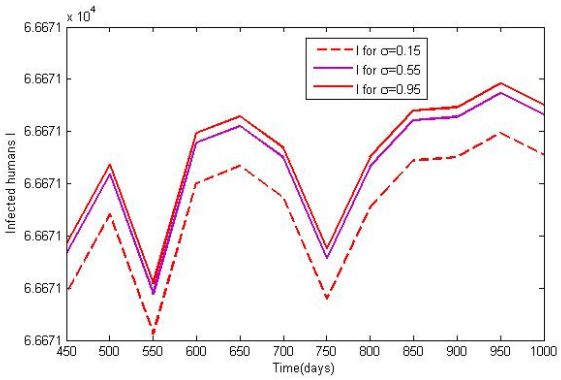

Figure 8: Varying effects of σ.

The increase of σ *towards* 1 *favors the transmission of the disease in the human population.*

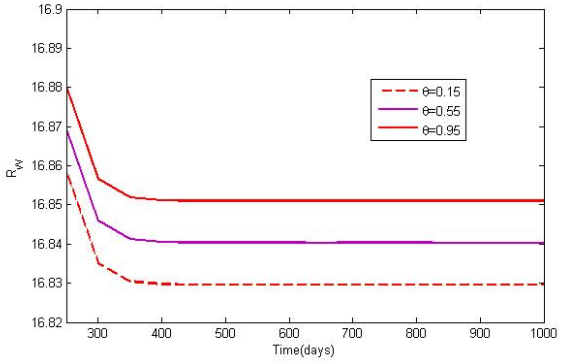

Figure 9: Varying effects of θ.

The increase of θ *towards* 1 *has a positive effect on the dynamics of the intermediate level of immunity.*

5. Conclusion

In this paper a deterministic model have been formulated to describe *chikungunya* transmission between a population of mosquitoes and a human population structured by immune status. The analysis of the model and the basic reproduction number expressed in terms of the parameters made possible to predict whether the disease will disappear ($\mathcal{R}_0 < 1$) or persist ($\mathcal{R}_0 > 1$). The numerical simulation have permitted to confirm the theoretical results obtained from the analysis. The numerical results have also shown that the decay of the immunity is a factor which favors the transmission of the disease and boosting the immune system helps to get more immune humans. Some medical researches must aim at preventing the decay of the immunity and boosting the immune system .

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