USING MATHEMATICAL MODELING OF UNDERSTANDING THE CAUSE AND THE WAY TO PREVENT OUTBREAK DISEASE (ZIKA VIRUS)

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Abstract
We developed a deterministic mathematical model describing the transmission dynamics of Zika virus. The model is a system of first order ordinary differential equation (ODE), where S is susceptible human, I is infected human, R is recovered human , human and mosquitoes interact to infect human, incorporating the use of condom, water hygiene and sanitation. The equilibrium states and the analytical solution use Homotopy Perturbation Method (HPM) and generating the reproductive number \( R_0 \), using Gauss Jordan elimination method. The demographic profile of French Polynesia was used in the model to show the effect of control measure at different rate (i.e lower, medium and higher) on French Polynesia population. A numerical simulation was carried out using maple software to show the effective reproductive number to determine whether the disease is under control or out of control.

Keywords: Zika Virus, Transmission Dynamics, Model Equations, Homotopy Perturbation Method (HPM), Ordinary Differential Equations (ODE), Population and Susceptible-Infected-Recovered (SIR)

1.0 INTRODUCTION
Originally identified in Africa (Hayes, 2009) the first large reported outbreak of Zika virus (ZIKV) disease occurred in Yap in April to July 2007. Also, there was an outbreak in French Polynesia between October 2013 and April 2014 (Duffy, et.al, 2009, Cao-Lormeau, 2014) and cases in other Pacific countries (Musso 2015). In 2015, local transmission was also reported in South American countries, including Brazil (Campos, 2015, Colombia and Camacho 2016).

Zika virus is an emerging mosquito–borne virus that was also identified in Africa (Uganda) in 1947, it was subsequently discovered in humans in 1952 in Uganda and United Republic of Tanzania. The disease is caused by Zika virus which is spread to people primarily through the bite of an infected Aedes species mosquito, the most common symptoms of Zika virus are fever, rash, joint pain and conjunctivitis (red eye).

Transmission of Zika virus (ZIKV) is predominately vector-borne, but can also occur via sexual contact and blood transfusions (Musso, 2015). The virus is spread by the Aedes species of mosquito (Mallet, et al., 2015), which is also the vector for dengue virus. (DENV), Zika virus (ZIKA) is therefore likely to be capable of sustained transmission in other tropical areas as well as causing symptoms such as fever and rashes, Zika virus infection has also been linked to increased incidence of neurological sequelae, including Guillain-Barré Syndrome (GBS) and microcephaly in infants born to mothers who were infected with Zika virus during pregnancy (Schuler-Faccini et al., 2016). On 1st February 2015, the World Health Organization declared a Public Health Emergency of International Concern in
response to the clusters of microcephaly and other neurological disorders reported in Brazil, possibly linked to the recent rise in Zika virus incidence. The same phenomena were observed in French Polynesia, with 42 GBS cases (Leparc-Goffart et al., 2015) reported during the outbreak. In addition to the GBS cluster, there were 18 fetal or newborn cases with unusual and severe neurological features reported between March 2014 and May 2015 in French Polynesia, including cases with microcephaly and severe brain lesions, and 8 norm cephalic cases with severe anatomical or functional neurological abnormalities (Centre d'hygiène et de salubrité publique, 2014). Given the potential for Zika virus to spread globally, it is crucial to characterize the transmission dynamics of the infection. This includes estimates of key epidemiological parameters, such as the basic reproduction number, Ro (defined as the average number of secondary cases generated by a typical infectious individual in a fully susceptible population), and of how many individuals (including both symptomatic and asymptomatic) are typically infected during an outbreak. Such estimates could help assist with outbreak planning, assessment of potential countermeasures, and the design of studies to investigate putative associations between Zika virus infection and other conditions. Islands can be useful case studies for outbreak analysis. Small, centralized populations are less likely to sustain endemic transmission than a large, heterogeneous population (Keeling and Grenfell, 1997), which means outbreaks are typically self-limiting after introduction from external sources (Cao-Lormeau 2014). Further, if individuals are immunologically naive to a particular pathogen, it is not necessary to consider potential effect of pre-existing immunity on transmission dynamics (Ballesteros et al., 2011).

Using a mathematical model of vector-borne infection, we examined the transmission dynamics of Zika virus on six archipelagos in French Polynesia during the 2013-2014 outbreaks. We inferred the basic reproduction number, and the overall size of the outbreak, and hence how many individuals would still be susceptible to infection in coming years (Adam et al., 2016).

2.0 METHOD OF DATA COLLECTION

The information collected for the analysis of this paper work is purely secondary data that is, already made data. We used weekly reported numbers of suspected ZIKV infections from the main regions of French Polynesia between May 2015 and August 2016. The data are recorded on weekly basis so that at the end of each month the overall total will be calculated.

We used a susceptible-infected-recovered (SIR) model to simulate vector-borne transmission. Both human and mosquitoes were modeled using a susceptible-infectious-removed (SIR) framework. This model incorporated delays as a result of the intrinsic (human) and extrinsic (vector) latent periods. Since there is evidence that asymptomatic DENV-infected individuals are capable of transmitting DENV to mosquitoes, we assumed the same for ZIKV.

3.0 METHODS AND MATERIALS

3.1 Formulation of the Model

We develop a model to analysis the transmission of Zika virus through ordinary differential equation. The disease free and endemic equilibrium states are addressed and the value of effective basic reproductive number $R_0$ is expressed in terms of parameters, which determine whether the disease is under control or is out of control in the population. This
model divides the total population of human into three sub-classes namely: $S_H, I_H$ and $R_H$, while the population of the vector is divide into two classes namely: Adult Vector ($C_V$) and Pupae Vector ($A_V$).

3.2 Basic Assumption
I. It is assumed that the new births of susceptible $S(t)$ are susceptible.
II. It is assumed that the virus does not kill the vector i.e. their death can be natural or accidental.
III. The infected classes of the vector are divided into two: Adult Vector and Pupae Vector

![Figure 1: Shows the Schematic Diagram of the Mathematical Model for Zika Virus Transmission.](image)

4.0 Model Equation
Applying the assumption, definition of variables and parameter and the relationship between the variables and parameters describe in the schematic diagram in the previous page, we developed a six ordinary differential equation for the transmission and control of Zika virus in a population. The differential equations are given below:

$$\frac{dS_H}{dt} = \beta_H N - \frac{\beta_1 (1-\gamma) I_H S_H}{N} + \varphi R_H - \mu_H S_H - \frac{\beta_2 B S_H}{N}$$

(1)
These equations are valid for $N_t > 0$ for all the parameters in the model are assumed positive and the total population size are;

$$N_H = \mathcal{S}_H + I_H + R_H$$  \tag{7}
$$N_V = C_V + A_V$$  \tag{8}

The differential equation of the total population are;

$$\frac{dN_H}{dt} = (\beta_H - \mu_H)N_H - \delta_H I_H$$  \tag{9}
$$\frac{dN_V}{dt} = \beta_V A_V - (\mu_V + \delta_V)N_V$$  \tag{10}

At the equilibrium states let $S_H = \mathcal{S}_H^*, I_H = I_H^*, R_H = R_H^*, C_V = C_V^*, A_V = A_V^*, B=B^*$.  \tag{11}

So we rewrite the equation and substitute for the parameters and variables.

$$\beta_H N - \beta_v \left(1-e^{-\alpha I_H}\right) S_V H + \varphi R_H - \mu_H S_H - \beta_v B S_H = 0$$  \tag{12}
$$\frac{\beta_v \left(1-e^{-\alpha I_H}\right) S_V H}{N} + \mu_H I_H - \delta_H I_H^* - \frac{\delta_v B S_H}{N} = 0$$  \tag{13}
$$\beta_v A_V - \mu_V C_V - \delta_v C_V = 0$$  \tag{15}
$$\sigma C_V = \mu_V A_V - \delta_v A_V = 0$$  \tag{16}
$$eA_V - B^*(mb - nb) = 0$$  \tag{17}

At the disease-free equilibrium

From equation (16)
\[\sigma C_V^* - A_V^* (\mu_V + \delta_V) = 0\]
\[A_V^* (\mu_V + \delta_V) - \sigma C_V^* = 0\]
\[A_V^* = \frac{\sigma C_V^*}{(\mu_V + \delta_V)}\]  \tag{18}

From equation (15) substitute for $A_V^*$
\[\beta_v \left( \frac{\sigma C_V^*}{(\mu_V + \delta_V)} \right) - \mu_V C_V^* - \sigma C_V^* - \delta_v C_V^* = 0\]
\[C_V^* \left( \frac{\sigma}{(\mu_V + \delta_V)} \right) - \mu_V - \sigma - \delta_v = 0\]

Since $(\frac{\sigma}{(\mu_V + \delta_V)} - \mu_V - \sigma - \delta_v) \neq 0$
\[C_V^* = 0\]

From equation (18) substitute for $C_V^*$
\[A_V^* = \frac{\sigma(0)}{(\mu_V + \delta_V)}\]
\[A_V^* = 0\]
Also from equation (17) substitute for $A^*_H$

$eA^*_V - B^*(mb - nb) = 0$

$B^* \frac{eA^*_V}{(mb - nb)} = e(0)$

$B^* = \frac{e(0)}{(mb - nb)}$

From equation (13) we have

$\frac{\beta_1 (1 - \tau e) S^*_H I^*_H}{N} + \mu H I^*_H - \alpha \ I^*_H + \frac{\beta_2 B^* S^*_H}{N} = 0$

Substitute for $B^*$

$\frac{\beta_1 (1 - \tau e) S^*_H I^*_H}{N} + \mu H I^*_H - \alpha \ I^*_H - \delta H I^*_H + \frac{\beta_2 (0) S^*_H}{N} = 0$

$\frac{I^*_H (\frac{\beta_1 (1 - \tau e) S^*_H I^*_H}{N} - \mu H - \alpha - \delta H)}{N} = 0$

$I^*_H = 0$

From equation (14)

$\alpha I^*_H - \mu H R^*_H - \varphi R^*_H = 0$

Substitute for $I^*_H$

$\alpha (0) - \mu H R^*_H - \varphi R^*_H = 0$

$R^*_H = \frac{\mu H + \varphi}{\alpha (0)}$

Also from equation (11)

$\beta H N - \frac{\beta_1 (1 - \tau e) S^*_H I^*_H}{N} + \varphi R^*_H - \mu H S^*_H - \frac{\beta_2 B^* S^*_H}{N} = 0$

Substitutes for $I^*_H, R^*_H$, and $B^*$

$\beta H N - \frac{\beta_1 (1 - \tau e) S^*_H (0)}{N} + \varphi (0) - \mu H S^*_H - \frac{\beta_2 (0) S^*_H}{N} = 0$

$\beta H N - \mu H S^*_H = 0$

$\mu H S^*_H = \beta H N$

$S^*_H = \frac{\beta H N}{\mu H}$

Hence, the disease-free equilibrium is
For endemic equilibrium

\[ S_H > 0, I_H > 0, R_H > 0, C_H > 0, A_H > 0, B^* > 0 \]

For human population

Recall from equation (14)

\[ \alpha I_H - \mu_H R_H^* - \varphi R_H^* = 0 \]

\[ R_H^* (\mu_H + \varphi) = \alpha I_H \]

\[ R_H^* = \frac{\alpha I_H}{\mu_H + \varphi} \]

Also from equation (12)

\[ \beta_H N - \frac{\beta_1 (1 - \tau \varepsilon) S_H^* I_H}{N} + \varphi R_H^* - \mu_H S_H^* - \frac{\beta_2 B^* S_H^*}{N} = 0 \]

\[ -S_H^* \left( \frac{\beta_1 (1 - \tau \varepsilon) I_H}{N} + \mu_H + \frac{\beta_2 B^*}{N} \right) + \beta_H N + \varphi R_H^* = 0 \]

\[ S_H^* \left( \frac{\beta_1 (1 - \tau \varepsilon) I_H}{N} + \mu_H + \frac{\beta_2 B^*}{N} \right) = \beta_H N + \varphi R_H^* \]

\[ S_H^* = \frac{\beta_H N + \varphi R_H^*}{\left( \frac{\beta_1 (1 - \tau \varepsilon) I_H}{N} + \mu_H + \frac{\beta_2 B^*}{N} \right)} \]

\[ S_H^* = \frac{\beta_1 (1 - \tau \varepsilon) I_H + N \mu_H + \beta_2 B^*}{N (\mu_H + \varphi) + \varphi (\alpha I_H)} \]

From equation (33)

\[ \frac{\beta_1 (1 - \tau \varepsilon) S_H^* I_H}{N} + \mu_H I_H^* - \alpha I_H^* + \frac{\beta_2 B^* S_H^*}{N} = 0 \]

\[ -I_H^* \left( \frac{\beta_1 (1 - \tau \varepsilon) S_H^*}{N} + \mu_H + \alpha + \delta_H \right) + \frac{\beta_2 B^* S_H^*}{N} = 0 \]

\[ I_H^* \left( \frac{\beta_1 (1 - \tau \varepsilon) S_H^*}{N} + \mu_H + \alpha + \delta_H \right) = \frac{\beta_2 B^* S_H^*}{N} \]

\[ I_H^* = \frac{\beta_2 B^* S_H^*}{N (\mu_H + \alpha + \delta_H) - \beta_1 (1 - \tau \varepsilon) S_H^*} \]

Similarly, for the vector population
From equation (15)
\[ \beta_v A'\psi - \mu_v C'\psi - \sigma C'\psi - \delta_v C'\psi = 0 \]
\[-C'\psi (\mu_v + \sigma + \delta_v) + \beta_v A'\psi = 0 \]
\[C'\psi (\mu_v + \sigma + \delta_v) = \beta_v A'\psi \]
\[C'\psi = \frac{\beta_v A'\psi }{ (\mu_v + \sigma + \delta_v) } \quad (21) \]

Also from equation (3.16) we have
\[ \sigma C'\psi - A'\psi (\mu_v + \delta_v) = 0 \]
\[A'\psi (\mu_v + \delta_v) = \sigma C'\psi \]
\[A'\psi = \frac{\sigma C'\psi }{ (\mu_v + \delta_v) } \quad (22) \]

From equation (17)
\[ eA'\psi - B^*(mb - nb) = 0 \]
\[ B^*(mb - nb) = eA'\psi \]
\[B^* = \frac{eA'\psi }{ (mb - nb) } \quad (23) \]

Hence, the endemic equilibrium is given by
\[ E_E = (S_H^*, I_H^*, R_H^*, C_H^*, A_H^*, B^*) \]
\[= \left\{ \left[ \frac{N(\beta_v N(\mu_H + \varphi) + \varphi(\alpha I_H^*))}{(\mu_H + \varphi)[\beta_1(1 - \tau \epsilon)I_H^* + \mu_H + \beta_2 B^* \alpha I_H^*]} \right] \left[ \frac{\beta_2 B^* S_H^*}{N(\mu_H + \varphi + \delta_H) - \beta_1(1 - \tau \epsilon) S_H^* (\mu_H + \varphi)} \right] \right\} \]
\[= \left\{ \left[ \frac{\beta_v A'\psi}{(\mu_v + \sigma + \delta_v) } \right] \left[ \frac{\sigma C'\psi}{ (\mu_v + \delta_v) } \right] \right\} \]

5.0 Analytical Solution of the Model

5.1 Analytical solution of the model using Homotopy Perturbation Method (HPM)

Fundamentals of Homotopy Perturbation Method (HPM) were first proposed by[12]. To illustrate the basic ideas of this Method, the following nonlinear differential equation was considered:
\[ A(u) - f(r) = 0, \quad r \in \Omega \quad (24) \]

Subject to the boundary condition:
\[ B \left( \frac{\partial u}{\partial n} \right) = 0, \quad r \in \Gamma \quad (25) \]

Where A is a general differential operator, B a boundary operator, f(r) is a known analytical function and \( \Gamma \) is the boundary of the domain \( \Omega \). The operator A can be divided into two parts L and N, where L is the linear part, and N is the nonlinear component. Equation (16) may therefore be rewritten as:
\[ L(u) + N(U) - f(r) = 0, \quad r \in \Omega \quad (26) \]

The Homotopy perturbation structure is shown as follows
\[ H(v, p) = (1 - p)[L(v) - L(u_0)] + p[A(v) - f(r)] = 0 \quad (27) \]

Where:
\[ v(r, p): \Omega \in [0,1] \to R \quad (28) \]
In equation (28) p \( \in [0,1] \) is an embedding parameter and \( u_0 \) is the first approximation that satisfies the boundary condition. It can be assumed that the solution of equation (28) can be written as a power series as follows:
And the best approximation for the solution is:

$$u = p^{-1} V = V_0 + PV_1 + p^2 V_2 + \cdots$$  \hspace{1cm} (30)

The series (29) is convergent for the most cases. However, the convergence rate depends on the nonlinear operator $A(v)$.

### 6.0 Solution of the Model Equation

\begin{align*}
\frac{dS_H}{dt} - \beta_H N + \frac{\beta_1 (1-\tau \epsilon) I_H S_H}{N} - \varphi R_H + \mu_H S_H + \frac{\beta_2 S_H}{N} = 0 \tag{31} \\
\frac{dI_H}{dt} - \beta_1 \frac{(1-\tau \epsilon) I_H S_H}{N} - \beta_2 S_H + \mu_H I_H + \alpha I_H + \delta_H I_H = 0 \tag{32} \\
\frac{dR_H}{dt} - \alpha I_H + \mu_H R_H + \varphi R_H = 0 \tag{33} \\
\frac{dC_V}{dt} - \beta_V A_V + \mu_V C_V + \sigma C_V + \delta V C_V = 0 \tag{34} \\
\frac{dA_V}{dt} - \sigma C_V + \mu_V A_V + \delta V A_V = 0 \tag{35} \\
\frac{dB}{dt} - e A_V + B(mb - nb) = 0 \tag{36}
\end{align*}

With the initial condition $S(0) = S_0, I(0) = I_0, R(0) = R_0, C_V(0) = C_0, A_V(0) = A_0$ and $B(0) = B_0$

Applying Homotopy Perturbation Method to equation (31)

$$(1 - P) \frac{dS_H}{dt} + P \left( \frac{dS_H}{dt} - \beta_H N + \frac{\beta_1 (1-\tau \epsilon) I_H S_H}{N} - \varphi R_H + \mu_H S_H + \frac{\beta_2 S_H}{N} \right) = 0 \tag{37}$$

Let

\begin{align*}
S_H &= U_0 + P U_1 + P^2 U_2 + \cdots \tag{38} \\
I_H &= V_0 + P V_1 + P^2 V_2 + \cdots \tag{39} \\
R_H &= W_0 + P W_1 + P^2 W_2 + \cdots \tag{40} \\
C_V &= X_0 + P X_1 + P^2 X_2 + \cdots \tag{41} \\
A_V &= Y_0 + P Y_1 + P^2 Y_2 + \cdots \tag{42} \\
B &= Z_0 + P Z_1 + P^2 Z_2 + \cdots \tag{43}
\end{align*}

After Substitute for $S_H, I_H, R_H$ and collecting the coefficient of power of $P$ we have

\begin{align*}
P^0 &= U_0' = 0 \tag{44} \\
P^1 &= U_1' + \frac{\beta_1 (1-\tau \epsilon) V_0 U_0}{N} + \mu_U U_0 + \frac{\beta_2 U_0}{N} - \varphi W_0 - \beta_H N = 0 \tag{45} \\
P^2 &= U_2' + \frac{\beta_1 (1-\tau \epsilon) V_1 U_1}{N} + \mu_U U_1 + \frac{\beta_2 U_1}{N} - \varphi W_1 = 0 \tag{46}
\end{align*}
Applying HPM to the equation 3.32

\[(1 - P) \frac{dI_H}{dt} + P \left( \frac{dH}{dt} - \beta_1 \frac{(1-\tau)\Delta_H S_H}{N} - \frac{\beta_2 B S_H}{N} + \mu_H I_H + \alpha I_H + \delta_H I_H \right) = 0 \quad (47)\]

After the substitute for \(S_H, I_H, R_H\) and collecting the coefficient of the power of \(P\) we have

\[P^0 = V_0' = 0 \quad (48)\]

\[P^1 = V_1' - \beta_1 \frac{(1-\tau)\Delta_H S_H}{N} - \frac{\beta_2 B S_H}{N} + \mu_H V_0 + \alpha V_0 + \delta_H V_0 = 0 \quad (49)\]

\[P^1 = V_2' - \beta_1 \frac{(1-\tau)\Delta_H S_H}{N} - \frac{\beta_2 B S_H}{N} + \mu_H V_1 + \alpha V_1 + \delta_H V_1 = 0 \quad (50)\]

Applying HPM to the equation (33)

\[(1 - P) \frac{dR_H}{dt} + P \left( \frac{dR_H}{dt} - \alpha I_H + \mu_H R_H + \phi R_H \right) = 0 \quad (3.51)\]

After the substitute for \(I_H, R_H\) and collecting the coefficient of the power of \(P\) we have

\[P^0 = W_0' = 0 \quad (52)\]

\[P^1 = W_1' - \alpha V_0 + \mu_H W_0 + \phi W_0 = 0 \quad (53)\]

\[P^2 = W_2' - \alpha V_1 + \mu_H W_1 + \phi W_1 = 0 \quad (54)\]

Applying HPM to the equation 3.34

\[(1 - P) \frac{dC_V}{dt} + P \left( \frac{dC_V}{dt} - \beta_V A_V + \mu_V C_V + \sigma_V C_V + \delta_V C_V \right) = 0 \quad (55)\]

After the substitute for \(C_V\) and collecting the coefficient of the power of \(P\) we have

\[P^0 = X_0' = 0 \quad (56)\]

\[P^1 = X_1' - \beta_V Y_0 + \mu_V X_0 + \sigma_V X_0 + \delta_V X_0 = 0 \quad (57)\]

\[P^2 = X_2' - \beta_V Y_1 + \mu_V X_1 + \sigma_V X_1 + \delta_V X_1 = 0 \quad (58)\]

Applying HPM to the equation 3.35

\[(1 - P) \frac{dA_V}{dt} + P \left( \frac{dA_V}{dt} - \sigma_V C_V + \mu_V A_V + \delta_V A_V \right) = 0 \quad (59)\]

After the substitute for \(A_V\) and collecting the coefficient of the power of \(P\) we have

\[P^0 = Y_0' = 0 \quad (60)\]

\[P^1 = Y_1' - \sigma_V X_0 + \mu_V Y_0 + \delta_V Y_0 = 0 \quad (61)\]

\[P^2 = Y_2' - \sigma_V X_1 + \mu_V Y_1 + \delta_V Y_1 = 0 \quad (62)\]

Applying HPM to the equation 36
After the substitute for B and collecting the coefficient of the power of P we have

\[ P^0 = Z_0^0 = 0 \]  
\[ P^1 = Z_1^0 - eY_0 + Z_0^0(mb - nb) = 0 \]  
\[ P^1 = Z_1^1 - eY_1 + Z_1^1(mb - nb) = 0 \]

Applying the initial condition

\[ U_0 = S_0 = S(0) \]  
\[ P^0 = U_0^0 = 0 \Rightarrow U_0^0 = 0 \]

Integrate it

\[ U_0 = A \]  
\[ S_0 = A \]

Apply the same technique to other variables

\[ I_0 = C = V_0 \]  
\[ R_0 = D = W_0 \]  
\[ C_0 = E = X_0 \]  
\[ A_0 = F = Y_0 \]  
\[ B_0 = G = Z_0 \]

Where A, C, D, E, F, G are the constant of the integral

\[ P^1 = U_1^0 + \frac{\beta_1(1 - \tau e)V_0 U_0}{N} + \mu_h U_0 + \frac{\beta_2 Z_0 U_0}{N} - \varphi W_0 - \beta_h N = 0 \]

\[ U_1^0 = -\frac{\beta_1(1 - \tau e)V_0 U_0}{N} - \mu_h U_0 - \frac{\beta_2 Z_0 U_0}{N} + \varphi W_0 + \beta_h N \]

Integrating with respect to t we have

\[ U_1 = \left( -\frac{\beta_1(1 - \tau e)V_0 U_0}{N} - \mu_h U_0 - \frac{\beta_2 Z_0 U_0}{N} + \varphi W_0 + \beta_h N \right) t + J \]

Where J is constant of integration. Apply the initial condition \( U_1(0) = 0 \)

gives J = 0 and now we have

\[ U_1(t) = \left( -\frac{\beta_1(1 - \tau e)V_0 U_0}{N} - \mu_h U_0 - \frac{\beta_2 Z_0 U_0}{N} + \varphi W_0 + \beta_h N \right) t \]  
\[ (69) \]

Therefore, the other variables

\[ V_1(t) = \left( \frac{\beta_1(1 - \tau e)V_0 U_0}{N} + \frac{\beta_2 Z_0 U_0}{N} - \mu_h V_0 - \delta_h V_0 \right) t \]  
\[ (70) \]

\[ W_1(t) = (\alpha V_0 - \mu_h W_0 - \varphi W_0 ) t \]  
\[ (71) \]
Letting $p = 1$
\[ S_h(t) = \lim_{p \to 1} S_h(t) = U_0 + U_1 + U_2 + \ldots \]
Since $U_0 = S_0, V_0 = I_0, W_0 = R_0, X_0 = C_0, Y_0 = A_0, Z_0 = B_0$
Therefore
\[ S_h(t) = S_0 + \left( -\frac{\beta_1(1-\tau \epsilon)}{N} S_0 - \mu_h S_0 - \frac{\beta_2 A_0 S_0}{N} + \phi R_0 + \beta_h N \right) t \]

6.1 Effective basic reproduction number, $R_0$
One of the most important concerns about any infectious disease is its ability to invade a population. The basic reproduction number, $R_0$, is a measure of the potential for disease spread in a population, and is inarguably “one of the foremost and most valuable ideas that mathematical thinking has brought to epidemic theory” (Heesrbeek and Dietz, 1996). It represents the average number of secondary cases generated by an infected individual if introduced into a susceptible population with no immunity to the disease in the absence of interventions to control the infection. If $R_0 < 1$, then on average, an infected individual produces less than one newly infected individual over the course of his infection period. In this case, the infection may die out in the long run. Conversely, if $R_0 > 1$, each infected individual produces, on average more than one new infection, the infection will be able to spread in a population. A large value of $R_0$ may indicate the possibility of a major epidemic. Using the next generation operator technique described by (Diekmann and Heesterbeek, 2000) subsequently analyzed by (Vanden and Watmough, 2005), we obtained the basic reproduction number of the model equations (1) - (6) with is the spectral radius ($\rho$) of the next generation Matrix, $K$.

\[ R_0 = \rho K \] where $K = FV^{-1}$

Now, to find the value of the basic Reproductive Number $R_0$, we must first find the matrix $FV^{-1}$,
and \( V^{-1} = \frac{\text{adj}(V)}{|V|} \). To find \( V^{-1} \), we have to find \( V \) and \( V \) is defined as \( V^- = V^+ \) where

\[
V = \begin{bmatrix}
\mu_H + \infty + \delta_v & 0 & 0 & 0 \\
0 & \mu_v + \sigma + \delta_v & -\beta_v & 0 \\
0 & -\sigma & \mu_v + \delta_v & 0 \\
0 & 0 & -e & nb - mb
\end{bmatrix}
\]

In order to get determinant of the matrix \( V^{-1} \) we use the Gauss Jordan elimination method

\[
V = \begin{bmatrix}
\mu_H + \infty + \delta_v & 0 & 0 & 0 \\
0 & \mu_v + \sigma + \delta_v & -\beta_v & 0 \\
0 & -\sigma & \mu_v + \delta_v & 0 \\
0 & 0 & -e & nb - mb
\end{bmatrix}
= \begin{bmatrix}
1 & 0 & 0 & 0 \\
0 & 1 & 0 & 0 \\
0 & 0 & 1 & 0 \\
0 & 0 & 0 & 1
\end{bmatrix}
\]

To get \( R_1 = \frac{\text{adj}(V)}{|V|} \)

\[
R_1 = \frac{\mu_H + \infty + \delta_v}{\mu_H + \infty + \delta_v}
\]

\[
R_2 = \frac{\mu_v + \sigma + \delta_v}{\mu_v + \sigma + \delta_v}
\]

\[
R_3 = \frac{\mu_v + \delta_v}{\mu_v + \delta_v}
\]

\[
R_4 = \frac{\mu_v + \delta_v}{nb - mb}
\]

We have
Let \( U \) be the new \( R_2 = U \times R_2 \)

We have

\[
\begin{pmatrix}
1 & 0 & 0 & 0 \\
0 & 1 & 0 & 0 \\
0 & -\sigma & 1 & 0 \\
0 & -e & 1 & 1
\end{pmatrix}
\begin{pmatrix}
1 \\
\frac{1}{\mu + \alpha + \delta_v} \\
\frac{1}{\mu_v + \sigma + \delta_v} \\
\frac{1}{nb - mb}
\end{pmatrix}
\begin{pmatrix}
1 & 0 & 0 & 0 \\
0 & 1 & 0 & 0 \\
0 & -\sigma & 1 & 0 \\
0 & -e & 1 & 1
\end{pmatrix}
\]
To get new $R_3$ we use $R_3 = \frac{\sigma}{\mu_V + \delta_V} \times R_2$

We have

\[
\begin{pmatrix}
1 & 0 & 0 & 0 \\
0 & 1 & 0 & 0 \\
0 & 0 & 1 & 0 \\
0 & 0 & -\frac{e}{nb - mb} & 1
\end{pmatrix}
\begin{pmatrix}
\frac{1}{\mu_V + x + \delta_V} \\
0 \\
0 \\
0
\end{pmatrix}
\begin{pmatrix}
0 \\
0 \\
0 \\
\frac{1}{nb - mb}
\end{pmatrix}
\]

To get new $R_4$, we use $R_4 = R_4 + \frac{nb - mb}{\sigma} \times R_3$ and let

\[
M = \frac{e\sigma}{(nb - mb)(\mu_V + \delta_V)[\mu_V + \sigma + \delta_V] - \beta_V}\sigma
\]

Also

\[
p = \frac{e}{(nb - mb)(\mu_V + \delta_V)[\mu_V + \sigma + \delta_V] - \beta_V}\sigma
\]

\[
D = (\mu_V + \delta_V)[\mu_V + \sigma + \delta_V] - \beta_V\sigma
\]

We have

\[
\begin{pmatrix}
1 & 0 & 0 & 0 \\
0 & 1 & 0 & 0 \\
0 & 0 & 1 & 0 \\
0 & 0 & 0 & 1
\end{pmatrix}
\begin{pmatrix}
\frac{1}{\mu_V + x + \delta_V} \\
0 \\
0 \\
0
\end{pmatrix}
\begin{pmatrix}
0 \\
0 \\
0 \\
\frac{1}{nb - mb}
\end{pmatrix}
\]
\[
\begin{bmatrix}
1 & 0 & 0 & 0 \\
0 & 1 & 0 & 0 \\
0 & 0 & 1 & 0 \\
0 & 0 & 0 & 1
\end{bmatrix}
\]

\[
\begin{bmatrix}
\frac{1}{\mu_\nu + \alpha + \delta_\nu} & 0 & 0 & 0 \\
0 & \frac{1}{(\mu_\nu + \sigma + \delta_\nu) - \beta_\nu \sigma} & \beta_\nu / \rho & 0 \\
0 & \sigma & \frac{(\mu_\nu + \sigma + \delta_\nu)}{D} & 0 \\
0 & D & \frac{M}{P} & 1
\end{bmatrix}
\]

Therefore \( V^{-1} = \) for

\[
\begin{bmatrix}
1 & 0 & 0 & 0 \\
0 & \frac{1}{(\mu_\nu + \sigma + \delta_\nu) - \beta_\nu \sigma} & \beta_\nu / \rho & 0 \\
0 & \sigma & \frac{(\mu_\nu + \sigma + \delta_\nu)}{D} & 0 \\
0 & (nb - mb) & \frac{e(\mu_\nu + \sigma + \delta_\nu)}{(nb - mb)(\mu_\nu + \sigma + \delta_\nu) - \beta_\nu \sigma} & 1
\end{bmatrix}
\]

So \( FV^{-1} \) we have

\[
\begin{bmatrix}
\beta_1 (1 - \tau \varepsilon) I_\mu S_H / N & 0 & 0 & \beta_2 S_H / N \\
0 & 1 & 0 & 0 \\
0 & 0 & \frac{1}{(\mu_\nu + \sigma + \delta_\nu) - \beta_\nu \sigma} & 0 \\
0 & 0 & \frac{\sigma}{D} & \frac{(\mu_\nu + \sigma + \delta_\nu)}{D} \\
0 & 0 & \frac{M}{P} & 1
\end{bmatrix}
\]

\[
\begin{bmatrix}
1 & 0 & 0 & 0 \\
0 & \frac{1}{(\mu_\nu + \sigma + \delta_\nu) - \beta_\nu \sigma} & 0 & 0 \\
0 & \frac{\sigma}{D} & 1 & 0 \\
0 & \frac{M}{P} & 0 & 1
\end{bmatrix}
\]

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Then, for the Basic Reproductive Number $R_0$, we have

\[
R_0 = \frac{\beta_i (1 - \tau \varepsilon) S^*_H}{N (\mu_H + \alpha + \delta_v)}
\]

Since

\[
S^*_H = \frac{\beta_H N}{\mu_H}
\]

So we have

\[
R_0 = \frac{\beta_i (1 - \tau \varepsilon) \beta_H}{\mu_H (\mu_H + \alpha + \delta_v)}
\]

(81)

### 7.0 RESULT

**Population data for main regions of French Polynesia**

The total population value of region of French Polynesia is taking to be 162,470,000 and the life expected at birth is given as 52.05 for the year 2015 (UNICEF, 2015). The birth rate is given as 39.23 births for 1000 peoples and the natural death rate as $\frac{1}{52} = 0.0192$ for the year 2015 (WHO, 2015)

\[
\beta_H = \frac{39.32}{1000} = 0.03923 \quad \mu_H = 0.0192
\]

(82)

For $\beta_1$, the probability of transmission of infection for an infectious human to be susceptible human given that a contact between the two occurs is $\rho = 0.43$ (CDC, 2000) and assuming the average number of contacts is equal to $2 = c$

Then,

\[
\beta_1 = \rho \times c = 2 \times 0.43 = 0.86
\]

(83)

For $\beta_2$, the probability of transmission of infection from Zika to a susceptible human given that they come in contact with each other is $\rho = 0.05$ (CDC, 2000) and assuming the average number of contacts is $c = 1$

Then,

\[
\beta_2 = 0.05
\]

(84)
Table 1 The parameter value for main region of French Polynesia

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Value</th>
<th>Detail</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\mu_H$</td>
<td>0.0192</td>
<td>UNICEF, 2015</td>
</tr>
<tr>
<td>$\beta_H$</td>
<td>0.0392</td>
<td>UNICEF, 2015</td>
</tr>
<tr>
<td>$\delta_H$</td>
<td>0.000011</td>
<td>WHO</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>0.03836</td>
<td>CDC</td>
</tr>
<tr>
<td>$\varepsilon$</td>
<td>0.8</td>
<td>Estimate/Assumed</td>
</tr>
<tr>
<td>$\beta_1$</td>
<td>0.86</td>
<td>CDC, 2000</td>
</tr>
<tr>
<td>$\beta_2$</td>
<td>0.05</td>
<td>CDC, 2000</td>
</tr>
<tr>
<td>Mb</td>
<td>0.0175</td>
<td>CDC</td>
</tr>
<tr>
<td>Nb</td>
<td>0.0233</td>
<td>CDC</td>
</tr>
<tr>
<td>$\beta_V$</td>
<td>0.0167</td>
<td>WHO</td>
</tr>
<tr>
<td>E</td>
<td>0.015</td>
<td>Garba and Gumel (2010)</td>
</tr>
<tr>
<td>$\sigma$</td>
<td>0.06667</td>
<td>Whitney</td>
</tr>
<tr>
<td>$\mu_V$</td>
<td>0.0082</td>
<td>Spyghana</td>
</tr>
<tr>
<td>$\tau$</td>
<td>0-1</td>
<td>Abdulraham (2014)</td>
</tr>
</tbody>
</table>

Effective Basic Reproduction Number $R_0$

From equation

When the control measure (compliance to the use of condom, insecticide, water hygiene and sanitation) is 0.25

$$R_0 = \frac{0.86(0.8)(0.03923)}{(0.0192)[0.0192 + 0.03836 + 0.25]}$$

$R_0 = 4.57$

When the control measure (compliance to the condom usage, insecticide, water hygiene and sanitation) is 0.50

$$R_0 = \frac{(0.86)(0.60)(0.03923)}{(0.0192)[0.0192 + 0.03836 + 0.50]}$$

$R_0 = 0.010705$

$R_0 = 1.89$

When the control measure (compliance to condom usage, insecticide, water hygiene and sanitation) is 0.75

$$R_0 = \frac{(0.86)(0.40)(0.03923)}{(0.0192)[0.0192 + 0.03836 + 0.75]}$$

$R_0 = 0.015505$
When the control measure (compliance to condom usage, insecticide, water hygiene and sanitation) is 1.00
\[
R_0 = \frac{(0.86)(0.20)(0.03923)}{(0.0192)[0.0192 + 0.03836 + 1]}
\]
\[
R_0 = \frac{0.20305152}{0.00674756}
R_0 = 0.332
\]

Table 2 Description of effective basic reproductive number and the rate of compliance to the use of vaccination

<table>
<thead>
<tr>
<th>Control measure (compliance to the use of vaccination) ( \tau ) and ( \delta_v )</th>
<th>Effective basic reproductive number ( R_0 )</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.1</td>
<td>16.04</td>
</tr>
<tr>
<td>0.2</td>
<td>5.73</td>
</tr>
<tr>
<td>0.3</td>
<td>3.73</td>
</tr>
<tr>
<td>0.4</td>
<td>2.61</td>
</tr>
<tr>
<td>0.5</td>
<td>1.89</td>
</tr>
<tr>
<td>0.6</td>
<td>1.39</td>
</tr>
<tr>
<td>0.7</td>
<td>1.02</td>
</tr>
<tr>
<td>0.8</td>
<td>0.74</td>
</tr>
<tr>
<td>0.9</td>
<td>0.51</td>
</tr>
</tbody>
</table>

8 Discussion of Results
The effective basic reproductive number tells us how important each parameter is to disease transmission. Such information is crucial not only to experimental design, but also to data assimilation and reduction of complex nonlinear model. When we have low control measure we see that the reproductive number is greater than one and again when we use 75% of control measure and above we see that the reproductive number is lesser than one and when this occur it means the disease is under control. These indicate that we can use effective basic reproductive number to determine whether disease is of control or out of control.

9 Conclusion
In this paper, a Mathematical Model with standard incidence is developed and analyzed to study the transmission and control of Zika virus. Mathematically we modeled Zika virus as a 6–dimensional system of non-linear ordinary differential equation. We first show that there exist a domain \( D \) where our model is Mathematically and Epidemiologically well posed. The Model incorporates two control parameters, condom usage and personal hygiene efficacy \( (\tau) \) and compliance \( (\delta_v) \) and which is the rate at which both the dormant and active vector are killed due to the use of insecticide. The Disease Free Equilibrium points of the model were obtained, and analyzed for stability. We obtained an important threshold parameter Effective Reproductive Number \( R_0 \), it is known that when \( R_0 < 1 \) the disease dies out, and when \( R_0 > 1 \) the disease persists in the population. However, there are many human activities that will militate against the achievement of a Zika virus free society. The factors are:
I. Refusing of uses of condom doing intercourse
II. Lack of health education and low awareness in most rural areas.
III. Keeping of bushes and stagnant water around human homes.

10 References

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