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Mathematical Model for the Transmission Dynamics of Zika Virus Infection with Combined Vaccination and Treatment Interventions

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Abstract

In this paper, we studied the transmission dynamics of ZIKV in the presence of a vector under the combined effects of treatment and vaccination in a hypothetical population. The disease-free ε_0 and endemic ε_1 equilibria were established with local stability on ε_0. We established the basic reproduction number R_0 which served as a threshold for measuring the spread of the infection in the population using the next-generation matrix and computed its numerical value to be $R_0 = 0.0185903201$ using the parameter values. It was established that the disease-free equilibrium ε_0 is locally asymptotically stable since $R_0 < 1$; meaning ZIKV infection would be eradicated from the population. The computational results of the study revealed that combining the two interventions of vaccination and treatment concomitantly proffers an optimal control strategy in taming the transmission of the virus than a single intervention strategy.

Keywords

Zika Virus Infection, Flavivirus, Compartment, Asymptomatic, Basic Reproduction Number, Local Asymptotic Stability

1. Introduction

In 1948, scientists (from Yellow Fever Research Institute) caged a Rhesus macaque monkey in the Zika Forest of Uganda for epidemic study. After some time, the monkey developed fever, the scientists took a sample of the monkey's serum

and isolated a "filterable transmission agent" which was named Zika Virus [1]. ZIKV is a member of the virus family *Flaviviridae* and genus *Flavivirus*, which is related to dengue, yellow fever, and Japanese encephalitis and West Nile viruses [1]. ZIKV is transmitted by the day time active *Aedes* mosquitoes, such as *A. aegypti* and *A. albopictus* [2]. According to a report by the CDC, the infection, often causes only mild symptoms, similar to a mild form of dengue, and was treated by paracetamol (*acetaminophen*) and rest [2]. Since 1950s, ZIKV was known to occur within a narrow equatorial belt from Africa to Asia. In 2014, the virus spread eastward across the Pacific Ocean to French Polynesia, then to Easter Island, and in 2015 to Mexico, Central America, the Caribbean, and the South America, where the Zika outbreak reached pandemic levels [2] [3]. From 1951 through 1981, evidences of human infection with ZIKV was reported from African countries such as the Central African Republic, Egypt, Gabon, Sierra-Leone, Tanzania and Uganda, as well as in parts of Asia including India, Indonesia, Malaysia, the Philippines, Thailand and Vietnam [4]. From its discovery until 2007, there were only 14 confirmed human cases of ZIKV infection from Africa and Southeast Asia [4]. In April 2007, the first outbreak outside Africa and Asia occurred on the Island of Yap in the Federated States of Micronesia, the infection was characterized by rash, conjunctivitis, and arthralgia, which was initially thought to be dengue, chikungunya or Ross River Disease [5]. More recently, epidemics have occurred on Easter Island, the Cook Islands, and New Caledonia and in the French Polynesia between October 2013 and April 2014 [6]. Since April 2015, a large, on-going outbreak of Zika virus, which began in Brazil, has spread to more of South and Central America, the Caribbean and Colombia. Around 1.5 million people in Brazil have been infected in just eight months, and a total of 14 countries across South Central America, including Barbados and Mexico reported locally acquired cases [3]. Between October 2015 and January 2016, Brazilian Health authorities reported more than 3500 microcephaly cases [3]. In November 2015, El Salvador reported their first case of ZIKV infection leading to an explosive outbreak where in just two months over 6000 suspected cases were reported [6]. As at February 2016, there were evidences that Zika fever in pregnant women can cause abnormal brain development in their fetuses through mother to child transmission which may result in miscarriage or microcephaly [3].

ZIKV, a flavivirus responsible for an unprecedented epidemic in Brazil and the Americas, has been causally associated with fetal microcephaly, intrauterine growth restriction, and other birth defects in both humans and mice [7]. The rapid development of a safe and effective ZIKV vaccine is a global health priority [1], [2], but very little is currently known about ZIKV immunology and mechanisms of immune protection. [7] was able to show that a single immunization of a plasmid DNA vaccine or a purified inactivated virus vaccine provides complete protection in susceptible mice against ZIKV strain from northeast Brazil. This ZIKV strain has recently been shown to cross the placenta and induce

fetal microcephaly and other congenital malformations in mice [7]. Their findings suggest that the development of a ZIKV vaccine for humans will likely be readily achievable. From our present point of view, the history of epidemics and immunization programs can be interpreted as an extraordinary long-term empirical study of the role of spatial synchrony in ecological meta population dynamics, including extensive experimental manipulations (via vaccination) [8].

Mathematical modeling in epidemiology provides understanding of the mechanisms that influence the spread of diseases, and suggests control strategies [9]. A work by [6], on the transmission dynamics of ZIKV in island populations, ushered in a mathematical model that was used in the analyses of the 2013-2014 French Polynesia ZIKV outbreak. In their work, they considered only treatment as a control measure in a single infectious compartment to model the trans-mission dynamics. A similar work can be traced to [10] who extended the work by [6]. In their extension, [10] treated Zika virus infection to be a vector-borne infection alone, and considered infection with Zika virus to be either symptomtic or asymptomatic and divided the infectious compartment accordingly. A similar mathematical model for the dynamics of Zika virus in the presence of a vector was developed by [11]. In developing their model, they incorporated transmission due to vector and sexual contact and used only treatment, as control measure, for the infectious class. The result of their work demonstrated that, sexual transmission of the virus is equally significant. A convalescent period was added in their model which refers to that period in time when patients with Zika virus infection recovers gradually and returns to normal, but may be a source of infection even if feeling better. For some diseases, it is found that for a period of time, a part of the infectious class does not show the symptoms. For modeling such diseases SEIR models are used [9].

A general SEIR model with vertical transmission for the dynamics of an infectious disease was studied by [12], a fraction p and a fraction q of the offspring from the exposed and the infectious classes, respectively, were assumed to be infected at birth. [12] Showed that once the disease appears, it eventually persists at the unique endemic equilibrium level, and they finally employed mathematical tools of differential analysis, persistence theory, Hopf-Andronov-Poincaré bifurcation, and linear system theory to deduce the existence of a family of periodic solutions that bifurcate from a positive interior equilibrium.

In this work, we proposed a mathematical model for the dynamics of Zika virus under the combined effects of vaccine and oral treatment. In developing the model we treated Zika virus infection as asymptomatic and symptomatic infections and partition the infected compartment into asymptomatic and symptomatic infected compartments.

2. Derivation of the Model

The model was developed on the assumptions that the birth and death per capita

mosquito rates are constant and distinct, the infectious compartment was partitioned into asymptomatic and symptomatic compartments such that asymptomatic and symptomatic infected individuals are equally infectious, the recruitment rate into the human population is by birth and is at a constant rate, the deaths in the human population are either natural or disease-induced and are constants, proportion of vaccinated susceptible per unit time are regarded removed with immunity response against Zika virus infection for a particular period of time, recovered individuals may become susceptible to infection again during a single outbreak or in the future outbreaks, recovery rates for asymptomatic and symptomatic infectious individuals are assumed the same and are constant. The vector (Mosquito) dos no recover from the infection. We targeted 50% vaccine coverage on the susceptible population, this leads us to the estimation of the proportion of susceptible vaccinated per unit time as $\alpha_h = 0.5$, we also assume that lost of temporary immunity from recovery λ_h, and that of vaccination φ_h, to become susceptible again is very small due to the strength of treatment and the vaccine, this leads to the estimation of this parameters as $\lambda_h = 0.09$ and $\varphi_h = 0.02$ respectively. For the start up population for the simulation, we used hypothetical total human population of 5120 distributed across the compartments of the model in the human host as indicated in Table 2. Any startup population for the simulation purpose could be used.

2.1. Description of the Dynamics Zika Virus under Vaccine and Treatment

The susceptible human patch S_h is recharged by birth. A susceptible human from S_h maybe vaccinated and move to the vaccinated patch V_h, or gets into contact with an infected vector from I_v and become exposed (*if not vaccinated*) to move to E_h. After the incubation period, the exposed human may become infected and move to $I_{h,a}$ or $I_{h,s}$. When treatment is administered on individuals in $I_{h,a}$ or $I_{h,s}$, they may recover and move to R_h or die naturally or as a result of the disease. A proportion of the vaccinated human in V_h may lose the temporary immunity conferred by the vaccine after some time due to the deterioration of the efficacy of the vaccine and become susceptible again, thus moving back to S_h. A vector from S_v may become exposed after coming into contact with an infected human from $I_{h,a}$ or $I_{h,s}$ and move to E_v. After the incubation period, the exposed vector becomes infected and moves to I_v and since there is no recovery for the vector, they die naturally. The cycle continues in this manner. The schematic diagram of the dynamics can be found in Figure 1.

2.2. Model Equations

Based on the transmission dynamics described above, we obtained the following model equations:

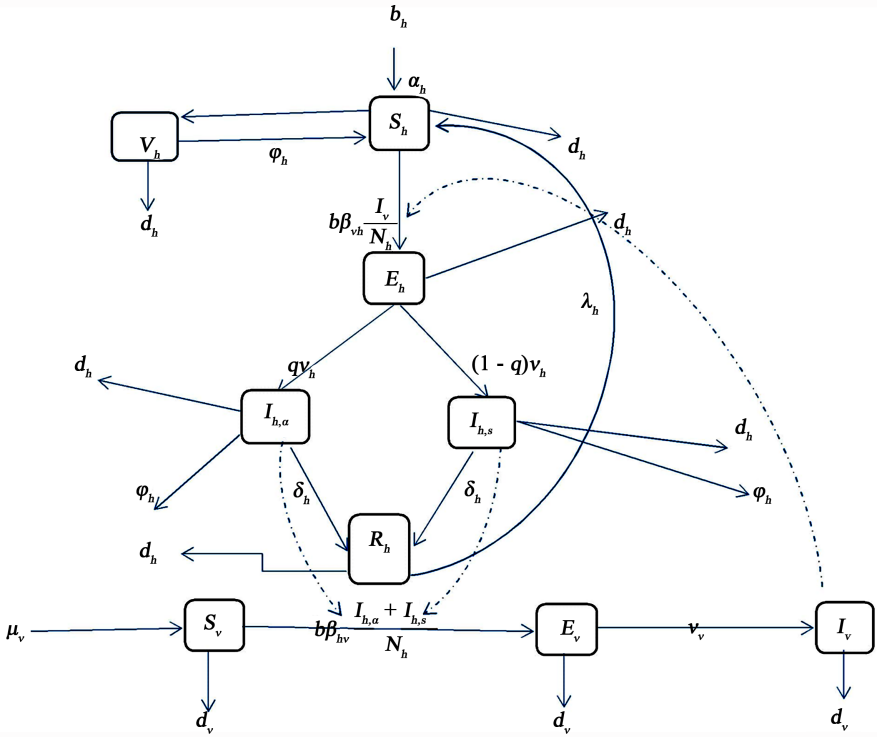

Figure 1. Schematic model diagram.

$$\left.\begin{aligned}
\dot{S}_h &= b_h - b\beta_{vh}S_h\frac{I_v}{N_h} - \alpha_h S_h + \lambda_h R_h + \varphi_h V_h - d_h S_h \\
\dot{V}_h &= \alpha_h S_h - (\varphi_h + d_h)V_h \\
\dot{E}_h &= b\beta_{vh}S_h\frac{I_v}{N_h} - v_h E_h - d_h E_h \\
\dot{I}_{h,s} &= (1-q)v_h E_h - \delta_h I_{h,s} - (d_h + \phi_h)I_{h,s} \\
\dot{I}_{h,a} &= qv_h E_h - \delta_h I_{h,a} - (d_h + \phi_h)I_{h,a} \\
\dot{R}_h &= \delta_h(I_{h,a} + I_{h,s}) - \lambda_h R_h - d_h R_h \\
\dot{S}_v &= \mu_v - b\beta_{hv}S_v\frac{I_{h,a}+I_{h,s}}{N_h} - d_v S_v \\
\dot{E}_v &= b\beta_{hv}S_v\frac{I_{h,a}+I_{h,s}}{N_h} - (d_v + v_v)E_v \\
\dot{I}_v &= v_v E_v - d_v I_v
\end{aligned}\right\} \quad (1)$$

3. Qualitative Analysis

3.1. Disease Free Equilibrium

Using standard approaches, we obtained the disease-free equilibrium $\varepsilon_\circ$ and the endemic equilibrium ε_1 and are respectively given to be:

$$\begin{aligned}
\varepsilon_\circ &= (S_h, V_h, E_h, I_{h,s}, I_{h,a}, R_h, S_v, E_v, I_v) \\
&= \left(\frac{b_h(\varphi_h + d_h)}{d_h(\alpha_h + \varphi_h + d_h)}, \frac{b_h\alpha_h}{d_h(\alpha_h + \varphi_h + d_h)}, 0, 0, 0, 0, \frac{\mu_v}{d_v}, 0, 0\right)
\end{aligned} \quad (2)$$

$$\varepsilon_1 = \left(S_h^*, V_h^*, E_h^*, I_{h,s}^*, I_{h,a}^*, R_h^*, S_v^*, E_h^*, I_v^*\right) \tag{3}$$

where:

$$S_h^* = \frac{d_v\left(d_h + v_h\right)\left(d_v + v_v\right)\left(\delta_h + d_h + \phi_h\right)\left(b_h - \phi_h I_h\right)^2\left[bd_h\beta_{hv}I_h + d_v\left(b_h - \phi_h I_h\right)\right]}{b^2 d_h^2 v_h v_v \mu_v \beta_{hv}\beta_{vh}\left(b_h - \phi_h I_h\right)}$$

$$V_h^* = \frac{\alpha_h d_v\left(d_h + v_h\right)\left(d_v + v_v\right)\left(b_h - \phi_h I_h\right)^2\left(\delta_h + d_h + \phi_h\right)\left[bd_h\beta_{hv}I_h + d_v\left(b_h - \phi_h I_h\right)\right]}{b^2 d_h^2 v_h v_v \mu_v \beta_{hv}\beta_{vh}\left(b_h - \phi_h I_h\right)\left(\varphi_h + d_h\right)}$$

$$E_h^* = \frac{\left(\delta_h + d_h + \phi_h\right)}{v_h} I_h, \quad I_{h,s}^* = \left(1 - q\right) I_h,$$

$$I_{h,a}^* = qI_h, \quad R_h^* = \frac{\delta_h}{\lambda_h + d_h} I_h$$

$$S_v^* = \frac{\mu_v\left(b_h - \phi_h I_h\right)}{bd_h\beta_{hv}I_h + \left(b_h - \phi_h I_h\right)}$$

$$E_v^* = \frac{bd_h\mu_v\beta_{hv}\left(b_h - \phi_h I_h\right)I_h}{\left(d_v + v_v\right)\left(b_h - \phi_h I_h\right)\left[bd_h\beta_{hv}I_h + d_v\left(b_h - \phi_h I_h\right)\right]}$$

$$I_v^* = \frac{bd_h v_v \mu_v \beta_{hv}\left(b_h - \phi_h I_h\right)I_h}{d_v\left(d_v + v_v\right)\left(b_h - \phi_h I_h\right)\left[bd_h\beta_{hv}I_h + d_v\left(b_h - \phi_h I_h\right)\right]}$$

such that $I_{h,a} + I_{h,s} = I_h$.

Since all parameters in this model are nonnegative, the solution of the model variables in the disease-free equilibrium and the endemic equilibrium is obviously positive. The total populations of the humans and the vectors in the model were defined by the following demographic equations:

$$N_h = S_h + V_h + E_h + I_{h,a} + I_{h,s} + R_h \tag{4}$$

$$N_v = S_v + E_v + I_v \tag{5}$$

Therefore, under the dynamics described by Equations (1)-(3), the region:

$$\Omega = \left\{\left(S_h, V_h, E_h, I_{h,a}, I_{h,s}, R_h\right) \in \Re_+^6 : S_h \geq 0, V_h \geq 0, E_h \geq 0, I_{h,a} \geq 0,\right.$$

$$\left. I_{h,s} \geq 0, R_h \geq 0, S_h + V_h + E_h + I_{h,a} + I_{h,s} + R_h \leq N_h \leq \frac{b_h}{d_h}\right\}$$

is positively invariant. Hence, the system is mathematically and epidemiologically well-posed. Therefore, for initial starting point, $x \in \Re_+^6$; the trajectory lies in Ω. Thus, we can restrict our analysis to the region Ω.

3.2. Local Stability Analysis of the Disease-Free Equilibrium

The basic reproduction number of the model was obtained as;

$$R_\circ = \sqrt{\frac{b^2 d_h v_h v_v \mu_v \beta_{hv}\beta_{vh}\left(\varphi_h + d_h\right)}{b_h d_v^2\left(\alpha_h + \varphi_h + d_h\right)\left(d_h + v_h\right)\left(d_v + v_v\right)\left(d_h + \delta_h + \phi_h\right)}} \tag{6}$$

The Jacobian matrix of the model was obtained as:

$$J=\begin{bmatrix} -(s+\alpha_h+d_h) & \varphi_h & 0 & 0 & 0 & \lambda_h & 0 & 0 & -w_1 \\ \alpha_h & -p & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ s & 0 & -c_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & c_2 & -x & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & qv_h & 0 & -x & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta_h & \delta_h & -c_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & -m_1 & -m_1 & 0 & -(n_1+d_v) & 0 & 0 \\ 0 & 0 & 0 & m_1 & m_1 & 0 & n_1 & -c_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & v_v & -d_v \end{bmatrix} \quad (7)$$

where: $s=b\beta_{vh}\frac{I_v^*}{N_h^*}$, $x=(\delta_h+d_h+\phi_h)$, $p=(\varphi_h+d_h)$, $w_1=b\beta_{vh}\frac{S_h^*}{N_h^*}$,

$n_1=b\beta_{hv}\frac{I_{h,a}^*+I_{h,s}^*}{N_h^*}$, $m_1=\left(b\beta_{hv}\frac{S_v^*}{N_h^*}\right)$, $c_1=(v_h+d_h)$, $c_2=(1-q)v_h$,

$c_3=(\lambda_h+d_h)$, $c_4=(v_v+d_v)$.

Evaluating J at the disease-free equilibrium $\varepsilon_\circ$, we obtained:

$$J=\begin{bmatrix} -(\alpha_h+d_h) & \varphi_h & 0 & 0 & 0 & \lambda_h & 0 & 0 & -w_2 \\ \alpha_h & -p & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -c_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & c_2 & -x & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & qv_h & 0 & -x & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \delta_h & \delta_h & -c_3 & 0 & 0 & 0 \\ 0 & 0 & 0 & -m_2 & -m_2 & 0 & -d_v & 0 & 0 \\ 0 & 0 & 0 & m_2 & m_2 & 0 & 0 & -c_4 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & v_v & -d_v \end{bmatrix} \quad (8)$$

where: $w_2=\frac{-b\beta_{vh}(\varphi_h+d_h)}{(\alpha_h+\varphi_h+d_h)}$, $m_2=\frac{bd_h\beta_{hv}\mu_v}{b_hd_v}$.

Next, we used elementary row-operations to row-reduce the Jacobian matrix evaluated at disease free equilibrium as used by [13]:

$$J_{|(x_\circ)}=\begin{bmatrix} -c_7 & \varphi_h & 0 & 0 & 0 & \lambda_h & 0 & 0 & -w_2 \\ 0 & -c_6 & 0 & 0 & 0 & \alpha_h\lambda_h & 0 & 0 & -c_5 \\ 0 & 0 & c_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & -c_3x & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -c_3x & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & c_3^2x & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -d_vc_3x & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & c_4c_3x & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & d_vc_4c_3x \end{bmatrix} \quad (9)$$

where $c_5=\frac{b\alpha_h\beta_{vh}(\varphi_h+d_h)}{(\alpha_h+\varphi_h+d_h)}$, $c_6=d_h(\alpha_h+\varphi_h+d_h)$ and $c_7=(\alpha_h+d_h)$.

Therefore, since (4) is an upper triangular matrix, we obtained the following nine eigenvalues from the reduced Jacobian matrix which are the entries on the

leading diagonal:

$$\lambda_1 = -c_7, \lambda_2 = -c_6, \lambda_3 = c_3, \lambda_4 = -c_3 x, \lambda_5 = -c_3 x,$$
$$\lambda_6 = c_3^2 x, \lambda_7 = -d_v c_3 x, \lambda_8 = c_4 c_3 x, \lambda_9 = d_v c_4 c_3 x$$

After substitutions, the eigenvalues became:

$$\lambda_1 = -(\alpha_h + d_h) \tag{10}$$

$$\lambda_2 = -d_h(\alpha_h + \varphi_h + d_h) \tag{11}$$

$$\lambda_3 = -(\lambda_h + d_h) \tag{12}$$

$$\lambda_4 = -(\lambda_h + d_h)(\delta_h + d_h + \phi_h) \tag{13}$$

$$\lambda_5 = -(\lambda_h + d_h)(\delta_h + d_h + \phi_h) \tag{14}$$

$$\lambda_6 = -(\lambda_h + d_h)^2(\delta_h + d_h + \phi_h) \tag{15}$$

$$\lambda_7 = -d_v(\lambda_h + d_h)(\delta_h + d_h + \phi_h) \tag{16}$$

$$\lambda_8 = -(v_v + d_v)(\lambda_h + d_h)(\delta_h + d_h + \phi_h) \tag{17}$$

$$\lambda_9 = -d_v(v_v + d_v)(\lambda_h + d_h)(\delta_h + d_h + \phi_h) \tag{18}$$

Since $\lambda_i < 0$, for $i = 1,2,3,4,5,6,7,8,9$, therefore, the disease-free equilibrium is locally asymptotically stable from the following theorem:

Theorem 1: The disease-free equilibrium is locally asymptotically stable if $R_\circ < 1$ and unstable if $R_\circ > 1$.

The basic reproduction number was then evaluated with Scientific Work Place using parameter values in Table 1 to be $R_\circ = 0.0185903201$ which is less than 1 suggesting stability for the disease-free equilibrium.

3.3. Numerical Simulations for the Modified Model

We carried out Numerical Simulation using the values in Table 1 and Table 2. The program was implemented on MATLAB R2012b to generate graphs for our model.

4. Results

The result of the numerical simulations is given in Figures 2-9 under the following subheads:

- Graphical simulation results with combined intervention (Vaccine and treatment);
- Graphical simulation results with treatment only;
- Graphical simulation results with Vaccine only at vaccine coverage rates:

$$\alpha_h = 0.5, \alpha_h = 0.6, \alpha_h = 0.7$$

5. Discussion

5.1. Combined Treatment and Vaccination Interventions (When Vaccine Is Administered at about 50% Coverage)

Figure 2 suggested that the infected compartments in the human and vector

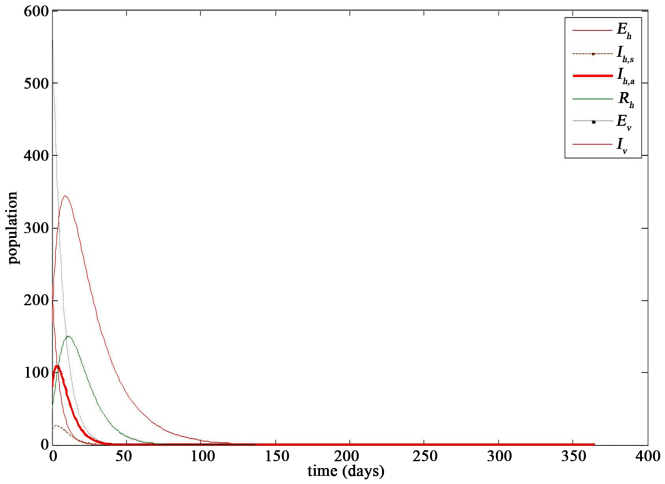

Figure 2. Graph showing the result of the model with combined vaccination and treatment interventions for E_h, $I_{h,a}$, $I_{h,s}$, R_h, E_v, I_v.

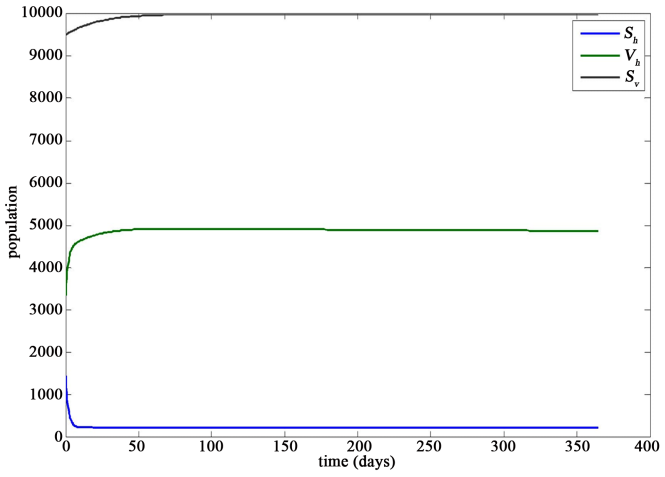

Figure 3. Graph showing the result of the model with combined vaccination and treatment intervention for S_h, V_h, S_v.

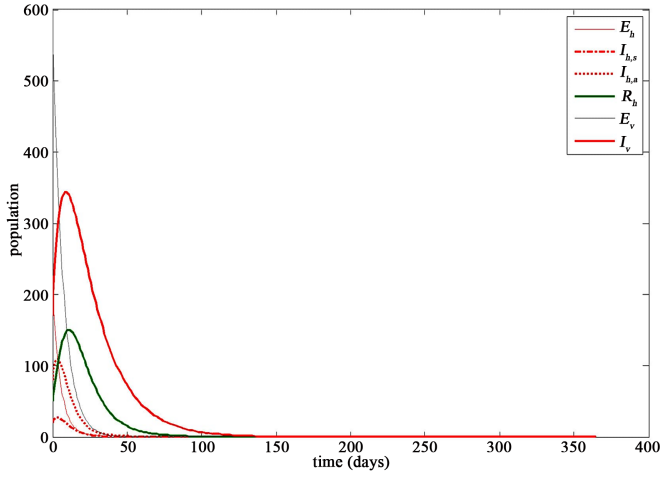

Figure 4. Graph showing the result of the model with treatment intervention only for E_h, $I_{h,a}$, $I_{h,s}$, R_h, E_v, I_v.

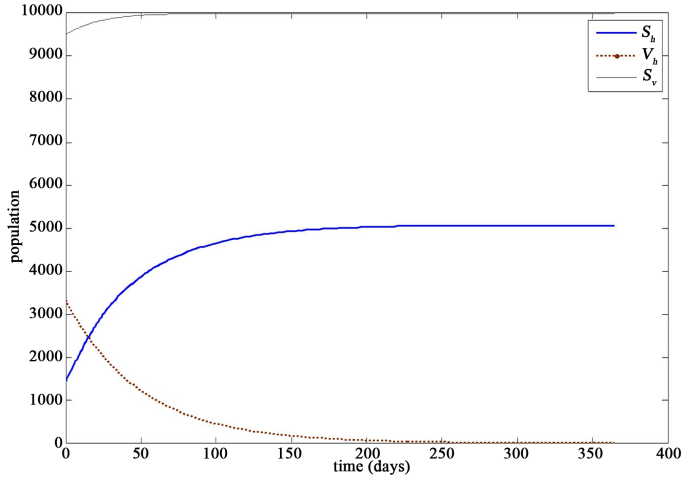

Figure 5. Graph showing the result of the model simulation with treatment only for S_h, V_h, S_v.

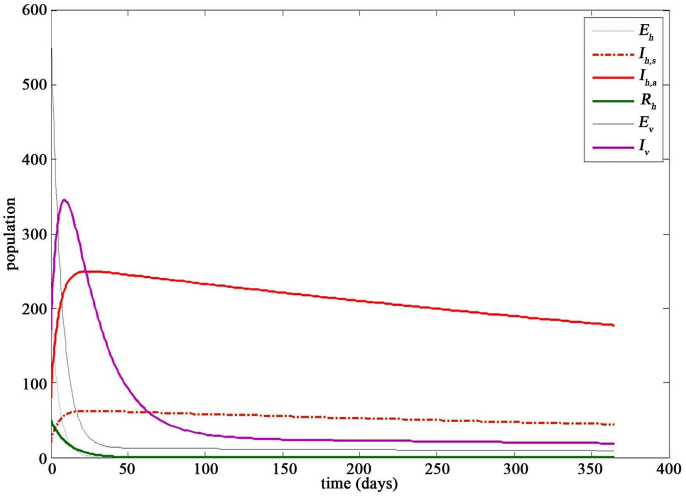

Figure 6. Graph showing the result of the model simulation with vaccination only (α_h = 0.5) for E_h, $I_{h,a}$, $I_{h,s}$, R_h, E_v, I_v.

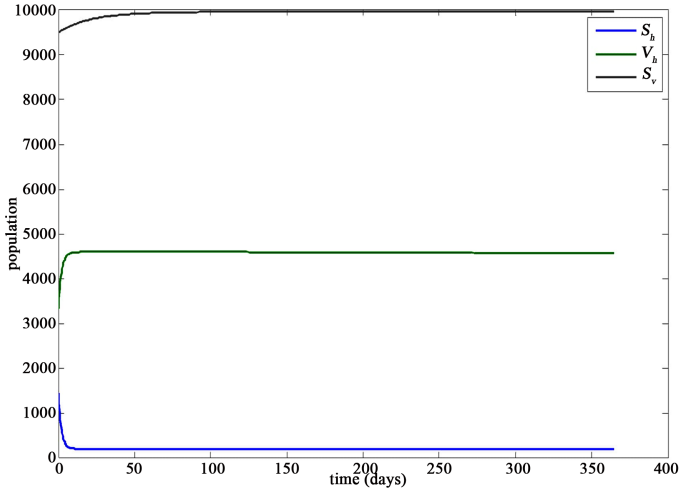

Figure 7. Numerical result for the model with vaccination only (α_h = 0.5) for S_h, V_h, S_v.

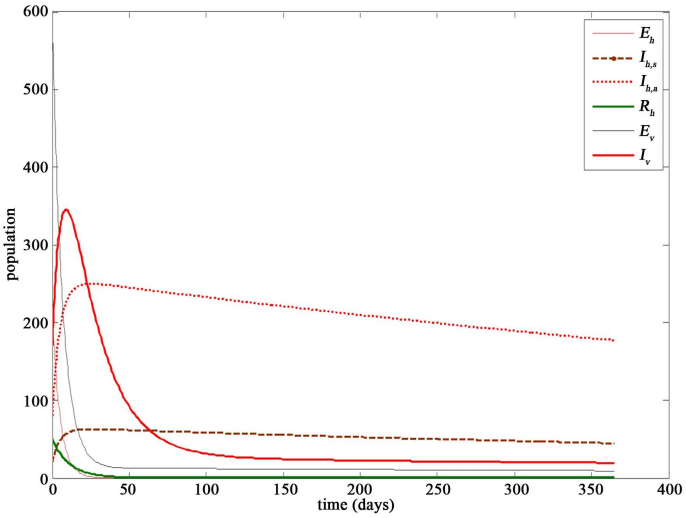

Figure 8. Numerical result for the model with vaccination only ($\alpha_h = 0.6$) for E_h, $I_{h,a}$, $I_{h,s}$, R_h.

Table 1. Baseline values for parameters in the model.

Parameter	Description	Value	Source
β_{vh}	Rate of infectiousness of human to mosquitoes	0.01	[2]
B_{hv}	Rate of infectiousness of mosquitoes to humans	0.01	[2]
b	Biting rate	0.3	[14]
b_h	Recruitment rate into the human population	0.0391389432	[14]
d_h	Natural death rate in the human population	0.00003913894325	[14]
d_v	Natural death rate in the mosquito's population	0.0476190476	[14]
ϕ_h	Disease-induced death rate in human population	0.001	[14]
ν_h	Human's incubation rate	0.2	[2]
ν_v	Vector incubation rate	0.1	[2]
q	Proportion of latent that become asymptomatic and infectious	0.8	[8]
δ_h	Human recovery rate	0.1666666667	[2]
μ_v	Recruitment rate into the vector population	476.19047619	[14]
α_h	Proportion of susceptible vaccinated per unit time	0.5	Assumed
λ_h	Proportion of the recovered that loses temporary immunity and become susceptible again	0.09	Assumed
φ_h	Proportion of vaccinated individuals that lose immunity and become susceptible again	0.02	Assumed

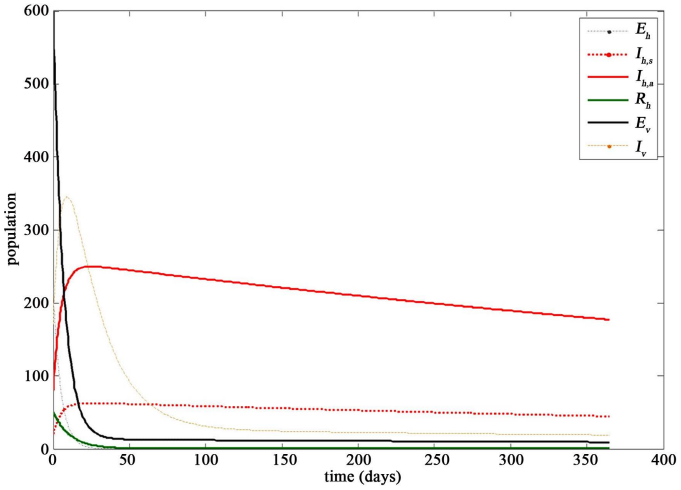

Figure 9. Numerical result for the model with vaccination only ($\alpha_h = 0.7$) for S_h, V_h, S_v.

Table 2. Baseline initial values for variables in the modified model.

Variable	Description	Value	Source
S_h	Susceptible humans	1430	Assumed
S_v	Susceptible vectors	9500	Assumed
E_h	Exposed/Latent humans	220	Assumed
E_v	Exposed/Latent vectors	600	Assumed
$I_{h,a}$	Asymptomatic infectious humans	80	Assumed
$I_{h,s}$	Symptomatic infectious humans	20	Assumed
I_v	Infected vector	170	Assumed
R_h	Recovered humans	50	Assumed
V_h	Vaccinated humans	3320	Assumed
N_h	Total human population	5120	Assumed
N_v	Total vector population	10270	Assumed

populations decreases gradually within the first hundred 100 days of the intervention and remained asymptotic to zero, while Figure 3 shows the susceptible humans decreases exponentially within the first few days and stabilizing at 200 head count thereafter, while both the susceptible vectors and the vaccinated humans grow slowly and achieved stability at about 10,000 and 5000 head count respectively.

5.2. Treatment Intervention Only

Figure 4 shows that; with treatment intervention only: infected humans, infected vectors and recovered compartments increase within less than 100 days, and dies out to remain asymptotic to zero (0) headcount in the population in the same vein, the exposed humans, and the exposed vectors dies out gradually within less than 50 days. Figure 5 indicates exponential growth in the suscepti-

ble humans at initial stage and then stability at about 5000 head count in the population, while the vaccinated compartment dies out with treatment over time.

5.3. Vaccine Intervention Only

Figure 6 shows that; with vaccine intervention only at $\alpha_h = 0.5$, the population of symptomatic and the asymptomatic infected humans grow rapidly in a logarithmic fashion within 30 days, and drops slowly over time, while the infected vector population shot up initially at an alarming rate, but later fell to as low as about 40 head count in the population (from the initial assumed population of 170), which is a significant decrease. Finally Figure 6 revealed that, the infected human and infected vector compartments dies out very slowly over time during the experimental study.

Figure 7 revealed that; with vaccine intervention only at $\alpha_h = 0.5$, the susceptible human population which started around 800 head count, decreases and stabilized below 200 due to continuous vaccination on the susceptible compartment. The vaccinated human population increased from 4000 head count and stabilized around 5000 head count, while the susceptible vectors population grew from around 9600 to 10,000 head count.

Figure 8 and Figure 9 revealed that; with various experiments conducted with vaccination only at coverage rates of $\alpha_h = 0.6$ and $\alpha_h = 0.7$, the rate α_h has great effect only on the vaccinated and susceptible human subpopulations, while it has very little impact on the infected compartments in the human and vector populations especially for a low-value vaccination rate, α_h.

6. Conclusion

From the results of the numerical simulations above, we see that: 1) Treatment intervention alone or vaccine intervention alone have impact on reducing the spread of the current ongoing Zika virus outbreak; 2) Vaccine intervention alone reduces the susceptible human population that may become exposed and later infected; 3) Combined vaccination and treatment interventions therapy have greater impact in reducing the spread of the virus in fewer days of the joint interventions with vaccine administered at about 50% coverage or greater on the susceptible human population, hence combined therapy gives a better result in a shorter period than treatment or vaccination alone. Therefore, we recommend combining vaccine and treatment in a population cohort intervention program to control the spread of Zika virus infection.

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Abbreviations

1) Description of Model State Variables

Variable Description

S_h : Susceptible human population

S_v : Susceptible vector population

E_h : Expose/Latent human population

E_v : Expose/Latent vectorpopulation

$I_{h,a}$: Asymptomatic infectious humanpopulation

$I_{h,s}$: Symptomatic infectious humanpopulation

I_v : Infectious vectorpopulation

R_h : Recovered humanpopulation

V_h : Vaccinated human population

N_h : Total human population

N_v Total vector population

2) Description of the Model Parameters

Parameter Description

β_{vh} : Rate of infectiousness of human to mosquito

β_{hv} : Rate of infectiousness of mosquito to human

b : Biting rate

b_h : Recruitment rate into the human population

d_h : Natural death rate in the human population

d_v : Natural death rate in the vector population

ϕ_h : Disease-induced death rate in the human population

v_h : Disease incubation rate in human

q : Proportion of latent that becomes asymptomatic & infectious

δ_h : Recovery rate for human

μ_v : Recruitment rate into the vector population

v_v : Disease incubation rate in mosquito (vector)

α_h : Proportion of susceptible vaccinated per unit time

λ_h : Proportion of treated individuals that becomes susceptible again

φ_h : Proportion of vaccinated individuals that lose immunity and become susceptible again

3) Noun

ZIKV: Zika Virus

CDC: Center for Disease Prevention and Control

DNA: Deoxyribonucleic acid

SEIR: Susceptible, Exposed, Infected and Recovered