A SPATIOTEMPORAL MODEL ON THE TRANSMISSION DYNAMICS OF ZIKA VIRUS DISEASE

Original Research Article

Abstract

Zika virus (ZikV) is transmitted to humans via mosquito bites during blood feeding. The *Aedes species* that transmits Chikungunya and Dengue diseases, is responsible for ZikV disease transmission. Despite the preventive and control strategies in place, ZikV disease still persists especially in the Western countries and the Pacific islands. In this study, a spatiotemporal model is developed and analyzed to describe the transmission dynamics of ZikV disease and deduce potential control strategies. Positivity and boundedness of solutions of the model with zero flux boundary conditions are shown. The basic reproduction number, R_0, is computed using the next generation matrix approach. Model analysis shows that the DFE point is both locally and globally asymptotically stable provided that $R_0 < 1$, which implies that the disease would not invade the population under study. The EE is locally asymptotically stable when $R_0 > 1$, which implies that the disease would persist in the population, at manageable levels. Existence of travelling wave solutions of the spatiotemporal model is shown. These waves propagate at a speed v, connecting the DFE and EE, which is the speed at which the disease spreads if $R_0 > 1$. Sensitivity analysis with respect to key parameters of, R_0, indicates that control strategies should target reduction of the vector biting rate. From numerical simulations, control strategies such as the use of insecticides, treated mosquito bed and window nets, clearing of bushes near the homesteads, as well as treatment of symptoms and quarantine of infected humans are deduced. This study suggest that effective implementation of the proposed control strategies would greatly reduce ZikV transmission.

Keywords: Zika Virus, Diffusion, Travelling wave

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1 Introduction

ZikV disease is a mosquito-transmitted disease that is spread by *Aedes species* of mosquitoes, the same vector that spreads Dengue and Chikungunya viruses [1]. ZikV is transmitted to humans via bites of an infected female mosquito of Aedes genus (*Aedes aegypti*) [2]. There is evidence that ZikV can be sexually transmitted and also via blood transfusion, however at a reduced infectivity rate [2, 3]. The incubation period is of ZikV in humans is about 2-7 days [1]. The infection has been associated with neurological complications such as Guillain Barre Syndrome(GBS) and microcephaly [4]. Particularly, ZikV disease presents a global health burden especially in Western countries and the Pacific islands where there is tropical climatic that give an ample breeding habitat for the *Aedes species* mosquitoes. A mathematical model to examine the 2013-2014 outbreak in six main regions of French Polynesia is presented in [4]. The estimates of the basic reproduction number ranged between $2.6 - 4.8$, with an estimated value of 11.5% of the reported cases and that a total of 94% of the population were infected. The authors shown that if complete protection against ZikV infection is taken, then it would take 12-20 years before re-emergence of the disease. The study suggest that ZikV could exhibit similar dynamics to Dengue virus in the Pacific, producing large but sporadic outbreak in a small island population.

An SEIR- type model is developed in [1], to study the prevention and control of ZikV as a mosquito-borne and sexual-transmitted disease. The study suggest that the end of the viremic period coincides with the disappearance of the virus in an asymptomatically infected individual. Sensitivity analysis indicated that the basic reproduction number, R_0, was most sensitive to the biting rate and mortality rate of mosquitoes while sexual transmission increases the risk of infection, epidemic size and prolongs the disease outbreak. The study suggest that prevention and control effort against ZikV should aim at both the mosquito-borne and sexual transmission routes.

A stochastic epidemic model which is data-driven is formulated to analyse the spread of ZikV in the Americas [5]. The model uses a high spatial and temporal resolution that integrates real-world demographic, human mobility, socioeconomic, temperature and vector density data. The model provides probability distributions of the time and place of introduction of ZikV in Brazil, the estimates of the attack rate, timing of the epidemic in the affected countries, and the projected number of newborns from women infected by ZikV. The study establishes that there was a decline of the epidemic in the affected countries after July 2016, due to the fact that large outbreaks greatly depletes the pool of susceptible. The finding was supported by an epidemiological surveillance in the region.

Transmission of ZikV exhibits both spatial and temporal variations [6]. Therefore, there is need to study the spatial and temporal dynamics on the transmission of ZikV disease. In this study, a spatiotemporal model in the form of a parabolic non-linear PDEs is developed to study the dynamics of ZikV disease and deduce control strategies against ZikV infection.

2 Model Description and Formulation

ZikV transmission dynamics is influenced by human and vector mobility and human-mosquito interaction in space and time. Mathematical models of infectious diseases more often described in terms of differential equations capture the dynamics of disease transmission within a susceptible population. The spatiotemporal transmission dynamics of ZikV disease is investigated to account for human and vector mobility. The total human population at a spatial location x , in one dimension, and at a time t given by $N_h(t, x)$, is subdivided into three compartments namely, $S_h(t, x)$ the number of the susceptible (humans capable of being infected), $I_h(t, x)$ the number of infected humans and $R_h(t, x)$ the number of the recovered humans. The vector population at a spatial location x, in

one dimension, and at time t given by $N_v(t,x)$, is sub-divided into two compartments, $S_v(t,x)$ the number of the susceptible mosquitoes (mosquito population capable of being infected) and $I_v(t,x)$ the number of the infectious mosquitoes. The susceptible humans are assumed to be recruited at a constant rate Λ_h. The susceptible humans acquire infection from an infectious bite of a mosquito via a force of infection $\gamma = \frac{c\beta_{vh}I_v}{(\eta+S_h)}$, where, β_{vh} is the vector-human probability of transmission upon a bite, c is the biting rate of vector and η is the half-maximal human saturation constant. It is considered to be of Michaelis-Menten form to account for saturation of the human infection [8]. Studies have shown that the disease transmission from human to human is insignificant to cause an epidemic [9]. Therefore, this mode of transmission is ignored in this model. The effective contact rate between susceptible humans and infected vector is denoted by, α, (i.e., $\alpha = c\beta_{vh}$). The mobility of susceptible humans, infected humans, susceptible vector and infected vector is taken to be diffusive with diffusion constant D_1, D_2, D_3 and D_4 respectively.

Natural mortality rate is assumed to occur in the human and vector populations at a rates μ_h and μ_v respectively. ZikV disease related deaths are rare, and therefore the disease death is not considered in this model [7]. The incubation period of ZikV disease is about 2-7 days [1]. This incubation period is relatively short and therefore it is assumed that all the exposed individuals live to become infectious and therefore, the exposed class is not considered. It has been observed that infection with ZikV confers permanent immunity upon recovery [1]. This recovery from the infection is assumed to take place at a rate λ. The recovered humans diffusivity is therefore ignored in this model since they do not affect the transmission dynamics of ZikV disease. Susceptible vectors are recruited at a constant rate Λ_v. The susceptible vectors acquire infection from an infectious human through blood feeding via a force of infection $\sigma = \frac{c\beta_{hv}I_h}{(\delta+S_v)}$, where, β_{hv} is the transmission probability from human to vector and δ is the half-maximal vector saturation constant. It is assumed to be of Michaelis-Menten form to account for saturation of the vector infection [8]. The effective contact rate between infected humans and susceptible humans is denoted by, κ, (i.e., $\kappa = c\beta_{hv}$). The associated parameters of the model are summarised in Table 1.

From the description above, the spatiotemporal model is represented by the following set of partial differential equations (pdes)

$$\begin{aligned}
\frac{\partial S_h}{\partial t} &= \Lambda_h - \frac{\alpha S_h I_v}{(\eta + S_h)} - \mu_h S_h + D_1 \nabla^2 S_h \\
\frac{\partial I_h}{\partial t} &= \frac{\alpha S_h I_v}{(\eta + S_h)} - (\mu_h + \lambda) I_h + D_2 \nabla^2 I_h \\
\frac{\partial R_h}{\partial t} &= \lambda I_h - \mu_h R_h \\
\frac{\partial S_v}{\partial t} &= \Lambda_v - \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v S_v + D_3 \nabla^2 S_v \\
\frac{\partial I_v}{\partial t} &= \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v I_v + D_4 \nabla^2 I_v
\end{aligned} \tag{2.1}$$

where $\alpha = c\beta_{vh}$, $\kappa = c\beta_{hv}$, $S_h = S_h(t,x), I_h = I_h(t,x), R_h = R_h(t,x)$ and $S_v = S_v(t,x), I_v = I_v(t,x)$ $(S_h, I_h, R_h, S_v, I_v) \in \mathbb{R}^5_+$, $\nabla^2 = \frac{\partial^2}{\partial x^2}$.

3 Analysis of the Model

The model describes human and vector populations, thus, both populations should remain non-negative and bounded. Analysis of model (2.1) is done under the zero flux boundary conditions given by equation (3.1). The zero flux boundary conditions imply that populations do not move

Table 1: A descriptive summary of the model parameters, their unit values and sources.

Parameter	**Description**	**Unit/value units**	**Source**
β_{vh}	Transmission probability from vector to human	(0.1 - 0.75) day^{-1}	[1]
β_{hv}	Transmission probability from human to vector	(0 - 1.0) day^{-1}	[1]
c	Vector biting rate	(0.3 - 1.5) day^{-1}	[1]
λ	Human recovery rate	(0 - 1.0) day^{-1}	[1]
Λ_h	Recruitment rate of humans	1.547×10^{-2} day^{-1}	[4]
μ_h	Natural death rate of humans	$3.65297\times 10^{-5}\text{day}^{-1}$	[11]
μ_v	Natural death rate of vectors	0.0714 day^{-1}	[12]
Λ_v	Recruitment rate of vectors	0.0714 day^{-1}	[20]
η	Human saturation constant	0.5	Assumed
δ	Vector saturation constant	0.5	Assumed
D_1	Susceptible humans diffusion constant	(0 - 1.0) day^{-1}	Varies
D_2	Infected humans diffusion constant	(0 - 1.0) day^{-1}	Varies
D_3	Susceptible vectors diffusion constant	(0 - 1.0) day^{-1}	Varies
D_4	Infected vectors diffusion constant	(0 - 1.0) day^{-1}	Varies

across the boundary $\partial\Psi$.

$$\frac{\partial S_h}{\partial n} = \frac{\partial I_h}{\partial n} = \frac{\partial R_h}{\partial n} = \frac{\partial S_v}{\partial n} = \frac{\partial I_v}{\partial n} = 0 \tag{3.1}$$

on $[0, +\infty) \times \partial\Psi$ where $\frac{\partial}{\partial n}$ denotes an outward normal derivative on $\partial\Psi$.
and initial conditions are given by

$$\begin{gathered} S_h(0,x) = S_h^0(x) \geq 0, I_h(0,x) = I_h^0(x) \geq 0, R_h(0,x) = R_h^0(x) \geq 0, \\ S_v(0,x) = S_v^0(x) \geq 0, I_v(0,x) = I_v^0(x) \geq 0. \end{gathered} \tag{3.2}$$

For $x \in \Psi$ and the space $(-\infty, +\infty)$, where Ψ is a bounded domain in $\Psi \subset \mathbb{R}^5_+$ with a smooth boundary $\partial\Psi$ and $t \geq 0$.

Lemma 3.1. *Suppose that the initial conditions (3.1) and (3.2) holds, then the solutions of model (2.1) are non-negative in* $[0, +\infty)$ *for all* $t \geq 0$.

Proof. Model (2.1) can be written as an abstract Banach space $X = \bar{C}(\Psi) \times C(\bar{\Psi})$ of the form:

$$\begin{aligned} u' &= Au(t) + F(u(t)), \qquad t > 0 \\ u(0) &= u^0 \in X \end{aligned} \tag{3.3}$$

where $u = (S_h, I_h, R_h, S_v, I_v)^T$, $u(0) = (S_h(0,x), I_h(0,x), R_h(0,x), S_v(0,x), I_v(0,x))^T$, $Au(t) = (D_1 S_h, D_2 I_h, 0, D_3 S_v, D_4 I_v)^T$ and

$$F(u(t)) = \begin{pmatrix} \Lambda_h - \frac{\alpha S_h I_v}{(\eta + S_h)} - \mu_h S_h \\ \frac{\alpha S_h I_v}{(\eta + S_h)} - (\mu_h + \lambda) I_h \\ \lambda I_h - \mu_h R_h \\ \Lambda_v - \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v S_v \\ \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v I_v \end{pmatrix} \tag{3.4}$$

Clearly, F is locally Lipschitz in X. Hence, model (2.1) has local solutions on the interval $[0, T_{max})$, where T_{max} is the minimal existence time for the solutions of model (2.1) [13].
Moreover, model (2.1) can also be written in the form;

$$\begin{aligned}
\frac{\partial S_h}{\partial t} - D_1\nabla^2 S_h &= F_1(S_h, I_h, R_h, S_v, I_v) \\
\frac{\partial I_h}{\partial t} - D_2\nabla^2 I_h &= F_2(S_h, I_h, R_h, S_v, I_v) \\
\frac{\partial R_h}{\partial t} &= F_3(S_h, I_h, R_h, S_v, I_v) \\
\frac{\partial S_v}{\partial t} - D_3\nabla^2 S_v &= F_4(S_h, I_h, R_h, S_v, I_v) \\
\frac{\partial I_v}{\partial t} - D_4\nabla^2 I_v &= F_5(S_h, I_h, R_h, S_v, I_v)
\end{aligned} \tag{3.5}$$

The functions $F_i(S_h, I_h, R_h, S_v, I_v), \quad i = 1, 2, 3, 4, 5$ are continuously differentiable and satisfies the following:
$F_1(0, I_h, R_h, S_v, I_v) = \Lambda_h \geq 0, F_2(S_h, 0, R_h, S_v, I_v) = 0 \geq 0, F_3(S_h, I_h, 0, S_v, I_v) = 0 \geq 0,$
$F_4(S_h, I_h, R_h, 0, I_v) = \Lambda_v \geq 0$ and $F_5(S_h, I_h, R_h, S_v, 0) = 0 \geq 0$ for all $t \geq 0$.
Since $(S_h, I_h, R_h, S_v, I_v) \geq 0$ with non-negative initial conditions of model (2.1), then the solutions are positive. This completes the proof. □

Lemma 3.2. *The solutions of the model (2.1) are bounded in the region $\Psi = \Psi_h \times \Psi_v$ for all $t \geq 0$.*

Proof. To show that all the solutions of model(2.1) are bounded in the region $\Psi = \Psi_h \times \Psi_v$ for all $t \geq 0$, model(2.1) is split into the human component (N_h) and the vector(mosquito) component (N_v).
For the human component, summing the first three equations of model (2.1) yields

$$\begin{aligned}
\frac{\partial S_h}{\partial t} + \frac{\partial I_h}{\partial t} + \frac{\partial R_h}{\partial t} = \Lambda_h - \frac{\alpha S_h I_v}{(\eta + S_h)} - \mu_h S_h + D_1\nabla^2 S_h + \frac{\alpha S_h I_v}{(\eta + S_h)} - \\
(\mu_h + \lambda) I_h + D_2\nabla^2 I_h + \lambda I_h - \mu_h R_h
\end{aligned}$$

Thus

$$\frac{\partial N_h}{\partial t} = \Lambda_h - \mu_h N_h + D_1\nabla^2 S_h + D_2\nabla^2 I_h \tag{3.6}$$

For simplicity, setting $D_1 = D_2 = D$ gives rise to the following inequality

$$\frac{\partial N_h}{\partial t} \leq \Lambda_h - \mu_h N_h + D\nabla^2 N_h \tag{3.7}$$

The inequality (3.7) has a unique solution of the form

$$\frac{\partial N_h}{\partial t} \leq \Lambda_h - \int_{-\infty}^{+\infty} \frac{\mu_h N_h}{\sqrt{4D\pi t}} e^{(\frac{(-x^2)}{4Dt})} dx \tag{3.8}$$

where the fundamental solution of the inequality (3.8) is given by

$$K(t, x) = \{ \begin{array}{lll} \frac{1}{\sqrt{4D\pi t}} e^{(\frac{-x^2}{4Dt})} & x \in \mathbb{R} & t > 0 \\ 0 & x \in \mathbb{R} & t < 0 \end{array} \tag{3.9}$$

For one-dimensional reaction-diffusion equation, equation (3.9) satisfies the following

$$K(t, x) = \int_{-\infty}^{+\infty} \frac{1}{\sqrt{4D\pi t}} e^{(\frac{-x^2}{4Dt})} dx = 1 \tag{3.10}$$

Hence

$$\frac{\partial N_h}{\partial t} \leq \Lambda_h - \mu_h N_h \tag{3.11}$$

Since $N_h(t,x) > 0$, therefore, solving this inequality (3.11) and taking the limit as $t \to \infty$ yields

$$0 < N_h \leq \frac{\Lambda_h}{\mu_h} \tag{3.12}$$

Hence $N_h(t,x)$ is bounded.
For the vector component of the model, summing the last two equations of model (2.1) yields

$$\frac{\partial S_v}{\partial t} + \frac{\partial I_v}{\partial t} = \Lambda_v - \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v S_v + D_3 \nabla^2 S_v + \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v I_v + D_4 \nabla^2 I_v$$

Thus

$$\frac{\partial N_v}{\partial t} = \Lambda_v - \mu_v N_v + D_3 \nabla^2 S_v + D_4 \nabla^2 I_v \tag{3.13}$$

where for simplicity, setting $D_3 = D_4 = D^\star$ gives rise to the following inequality

$$\frac{\partial N_v}{\partial t} \leq \Lambda_v - \mu_v N_v + D^\star \nabla^2 N_v \tag{3.14}$$

The inequality (3.14) has a unique solution of the form

$$\frac{\partial N_v}{\partial t} \leq \Lambda_v - \int_{-\infty}^{+\infty} \frac{\mu_v N_v}{\sqrt{4D^\star \pi t}} e^{(\frac{(-x^2)}{4D^\star t})} dx \tag{3.15}$$

where the fundamental solution of inequality (3.15) is given by

$$K^\star(t,x) = \begin{cases} \frac{1}{\sqrt{4D^\star \pi t}} e^{(\frac{-x^2}{4D^\star t})} & x \in \mathbb{R} \quad t > 0 \\ 0 & x \in \mathbb{R} \quad t < 0 \end{cases} \tag{3.16}$$

For one-dimensional reaction-diffusion equation, equation (3.16) satisfies the following

$$K^\star(t,x) = \int_{-\infty}^{+\infty} \frac{1}{\sqrt{4D^\star \pi t}} e^{(\frac{-x^2}{4D^\star t})} dx = 1 \tag{3.17}$$

Therefore

$$\frac{\partial N_v}{\partial t} \leq \Lambda_v - \mu_v N_v \tag{3.18}$$

Since $N_v(t,x) > 0$, therefore, solving inequality (3.18) and taking the limit as $t \to \infty$ yields

$$0 < N_v \leq \frac{\Lambda_v}{\mu_v} \tag{3.19}$$

Hence $N_v(t,x)$ is bounded.
Having shown that $N_h(t,x)$ and $N_v(t,x)$ are bounded on $[0,\infty) \times \Psi \subset \mathbb{R}^5_+$, it follows that the solutions of the model (2.1) are bounded in the considered region Ψ. □

From Lemma 3.1 and Lemma 3.2 above, it is clear that solutions of model (2.1) are positive and bounded for $t \geq 0$. Therefore, model (2.1) is mathematically and epidemiologically meaningful and it is now sufficient to consider its solutions in Ψ.

3.1 Local Stability of the Disease-Free Equilibrium

The stability analysis of the equilibria points is carried out to predict the long term behaviour of the solutions of the model. The disease-free equilibrium (DFE) is the state at which there is no infection in a certain population (absence of infection).

Theorem 3.3. *The disease-free equilibrium E_0 is locally asymptotically stable whenever $R_0 < 1$ and unstable when $R_0 > 1$.*

Proof. The Jacobian matrix of the model (2.1) without the diffusion terms is given by

$$J = \begin{bmatrix} \frac{-\alpha\eta I_v}{(\eta+S_h)^2} - \mu_h & 0 & 0 & 0 & \frac{-\alpha S_h}{(\eta+S_h)} \\ \frac{\alpha\eta I_v}{(\eta+S_h)^2} & -(\mu_h+\lambda) & 0 & 0 & \frac{\alpha S_h}{(\eta+S_h)} \\ 0 & \lambda & -\mu_h & 0 & 0 \\ 0 & \frac{-\kappa S_v}{(\delta+S_v)} & 0 & \frac{-\kappa\delta I_h}{(\delta+S_v)^2} - \mu_v & 0 \\ 0 & \frac{\kappa S_v}{(\delta+S_v)} & 0 & \frac{\kappa\delta I_h}{(\delta+S_v)^2} & -\mu_v \end{bmatrix} \tag{3.20}$$

Evaluating (3.20) at DFE $E^0 = (\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0)$ denoted as J_{E^0}, we obtain

$$J_{E^0} = \begin{bmatrix} -\mu_h & 0 & 0 & 0 & \frac{-\alpha\Lambda_h}{(\mu_h\eta+\Lambda_h)} \\ 0 & -(\mu_h+\lambda) & 0 & 0 & \frac{\alpha\Lambda_h}{(\mu_h\eta+\Lambda_h)} \\ 0 & \lambda & -\mu_h & 0 & 0 \\ 0 & \frac{-\kappa\Lambda_v}{(\mu_v\delta+\Lambda_v)} & 0 & -\mu_v & 0 \\ 0 & \frac{\kappa\Lambda_v}{(\mu_v\delta+\Lambda_v)} & 0 & 0 & -\mu_v \end{bmatrix} \tag{3.21}$$

Clearly, three of the eigenvalues of (3.21) are $-\mu_h, -\mu_h$ and $-\mu_v$ which are negative and the remaining two eigenvalues can be determined from:

$$A = \begin{bmatrix} -(\mu_h+\lambda) & \frac{\alpha\Lambda_h}{(\mu_h\eta+\Lambda_h)} \\ \frac{\kappa\Lambda_v}{(\mu_v\delta+\Lambda_v)} & -\mu_v \end{bmatrix}$$

where A is a 2×2 reduced matrix of J_{E^0}. We apply the Routh-Hurwitz Criterion to study the eigenvalues of matrix A. A negative trace and positive determinant of matrix A, which will ensure that the eigenvalues of matrix A have negative real parts. The trace (A)=$\tau(A)$=-$(\mu_h+\lambda)-\mu_v < 0$. Furthermore, the determinant of matrix A is given by: $det(A) = \mu_v(\mu_h+\lambda)(1-R_0^2)$
where R_0 is the basic reproduction number, which is the average number of secondary infections produced by a single individual introduced into a fully susceptible population during an individual entire infectious period [10, 13], calculated using the next generation matrix approach [14] and is given by

$$R_0 = \sqrt{\frac{\beta_{vh}\beta_{hv}c^2\Lambda_h\Lambda_v}{\mu_v(\mu_h+\lambda)(\mu_h\eta+\Lambda_h)(\mu_v\delta+\Lambda_v)}} \tag{3.22}$$

The $det(A)$ is positive when $R_0 < 1$. Therefore, the DFE is locally asymptotically stable whenever $R_0 < 1$ and unstable when $R_0 > 1$. □

Theorem 3.3 implies that for a small perturbation of the DFE, the solutions of model (2.1) will eventually converge to the DFE whenever $R_0 < 1$. Epidemiologically, it implies that if a few infectious individuals are introduced into a fully susceptible population, the disease would die out whenever $R_0 < 1$, otherwise the disease would spread.

3.2 Global stability of Disease-Free Equilibrium

In this subsection, the Castillo Chavez theorem [15] is applied to study the global stability of the disease-free equilibrium. We rewrite model (2.1) with $D_1 = D_2 = D_3 = D_4 = 0$ in the form;

$$\frac{dX}{dt} = H(X;Z) \quad \text{and} \quad \frac{dZ}{dt} = G(X;Z); \qquad G(X;0) = 0$$

where $X \in \mathbb{R}^3$ denotes the uninfected compartments i.e. $X = (S_h, R_h, S_v)$ and $Z = (I_h, I_v), Z \in \mathbb{R}^2$ denotes the infected compartments. At DFE

$$E^0 = (X^*, Z^*) = (X^*, 0), \qquad X^* = (\frac{\Lambda_h}{\mu_h}, 0, \frac{\Lambda_v}{\mu_v}) \tag{3.23}$$

The conditions in (3.24) must be satisfied to guarantee global asymptotic stability.

$\frac{dX}{dt} = H(X, 0)$, X^* is globally asymptotically stable.

$$G(X, Z) = BZ - \hat{G}(X, Z), \qquad \hat{G}(X, Z) \geq 0 \tag{3.24}$$

where $B = D_Z G(X^*, 0)$ is a M-matrix (the off-diagonal elements of B are non-negative). Conditions (3.24) must be satisfied to guarantee global asymptotic stability.

Theorem 3.4. *The fixed point $E^0 = (X^*, 0)$ is a globally asymptotically stable equilibrium point of model (2.1) whenever $R_0 < 1$ and the conditions in (3.24) are satisfied, otherwise it is unstable.*

Proof. From model (2.1), we have

$$H(X, 0) = \begin{bmatrix} \Lambda_h - \mu_h S_h \\ 0 \\ \Lambda_v - \mu_v S_v \end{bmatrix} \quad \text{and} \quad G(X, Z) = BZ - \hat{G}(X, Z)$$

where

$$B = \begin{bmatrix} -(\mu_h + \lambda) & \frac{\alpha\Lambda_h}{(\mu_h\eta + \Lambda_h)} \\ \frac{\kappa\Lambda_v}{(\mu_v\delta + \Lambda_v)} & -\mu_v \end{bmatrix} \qquad \hat{G}(X, 0) = \begin{pmatrix} \hat{G}_1(X, Z) \\ \hat{G}_2(X, Z) \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \end{pmatrix}$$

Since $\hat{G}(X, Z) \geq 0$ and the conditions in (3.24) are satisfied, therefore, E^0 is globally asymptotically stable whenever $R_0 < 1$. □

Theorem 3.4 implies that given a large perturbation of the DFE, the solutions of model (2.1) will eventually converge to the DFE whenever $R_0 < 1$. Epidemiologically, it implies that if a large number of infectious individuals are introduced in to a fully susceptible population the disease would die out whenever $R_0 < 1$, otherwise, the disease would spread.

3.3 Local stability of the Endemic Equilibrium

The endemic equilibrium (EE) point of the model (2.1) is defined as the state at which ZikV disease persists in population under study.

Theorem 3.5. *The endemic equilibrium point EE, denoted (E^*) is locally asymptotically stable whenever $R_0 > 1$ and unstable whenever $R_0 < 1$.*

Proof. Consider the Jacobian matrix (3.20) evaluated at endemic equilibria point $E^* = (S_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$ given by

$$J_{E^*} = \begin{bmatrix} \frac{-\alpha\eta I_v^*}{(\eta+S_h^*)^2} - \mu_h & 0 & 0 & 0 & \frac{-\alpha S_h^*}{(\eta+S_h^*)} \\ \frac{\alpha\eta I_v^*}{(\eta+S_h^*)^2} & -(\mu_h+\lambda) & 0 & 0 & \frac{\alpha S_h^*}{(\eta+S_h^*)} \\ 0 & \lambda & -\mu_h & 0 & 0 \\ 0 & \frac{-\kappa S_v^*}{(\delta+S_v^*)} & 0 & \frac{-\kappa\delta I_h^*}{(\delta+S_v^*)^2} - \mu_v & 0 \\ 0 & \frac{\kappa S_v^*}{(\delta+S_v^*)} & 0 & \frac{\kappa\delta I_h^*}{(\delta+S_v^*)^2} & -\mu_v \end{bmatrix} \tag{3.25}$$

Clearly, one of the eigenvalue of J_{E^*} is $-\mu_h$ along the main diagonal and the other four remaining eigenvalues are obtained from the reduced 2×2 matrices, C and D given by

$$C = \begin{bmatrix} -(\frac{\alpha\eta I_v^*}{(\eta+S_h^*)^2} + \mu_h) & 0 \\ \frac{\alpha\eta I_v^*}{(\eta+S_h^*)^2} & -(\mu_h+\lambda) \end{bmatrix} \qquad D = \begin{bmatrix} -(\frac{\kappa\delta I_h^*}{(\delta+S_v^*)^2} + \mu_v) & 0 \\ \frac{\kappa\delta I_h^*}{(\delta+S_v^*)^2} & -\mu_v \end{bmatrix}$$

Clearly, the traces of both matrices C and D are negative and their determinants are positive. i.e.,trace(C)=$\tau(C) = -(\frac{\alpha\eta I_v^*}{(\eta+S_h^*)^2}+2\mu_h+\lambda) < 0$, det(C)=$(\frac{\alpha\eta I_v^*}{(\eta+S_h^*)^2}+\mu_h)(\mu_h+\lambda) > 0$, trace(D)=$\tau(D) = -(\frac{\kappa\delta I_h^*}{(\delta+S_v^*)^2}+2\mu_v) < 0$ and det(D)=$\mu_v(\frac{\kappa\delta I_h^*}{(\delta+S_v^*)^2}+\mu_v) > 0$ provided that $I_h^* > 0$ and $I_v^* > 0$. Therefore, Endemic equilibrium point (EE) is locally asymptotically stable provided that $R_0 > 1$ otherwise, it is unstable. □

Theorem 3.5 implies that for a small perturbation of the EE, the solutions of model (2.1) will always converge to the EE whenever $R_0 > 1$. Epidemiologically, it implies that if a few infectious individuals are introduced in a fully susceptible population with $R_0 > 1$, then the disease would persist in the population.

4 Sensitivity Analysis

Sensitivity is the degree to which an input parameter to a model affects its output. Sensitive parameters are those which have a significant impact on the dynamics of infection/transmission [16, 17]. Using the normalized forward sensitivity index of R_0, the sensitivity index with respect to the model parameter z is given by

$$S_z^{R_0} = \frac{dR_0}{dz} \times \frac{z}{R_0} \tag{4.1}$$

where z is the parameter whose sensitive index is to be determined [16]. Table 2 gives a summary of the sensitivity indices of R_0 evaluated at the baseline parameters values given in Table 1.
From Table 2, an increase of the transmission probabilities β_{vh} and β_{hv} where, β_{vh} is the transmission probability from the vector to human and the transmission probability from human to vector respectively by 1% leads to an increase of the value of the basic reproduction number R_0 by 0.5%. An increase of the biting rate c by 1% increases the value of R_0 by 1%, whereas an increase of the rate of recovery from infection, λ, by 1% leads to a decrease of the value of R_0 by 0.49963%. Sensitivity analysis of the model (2.1) parameters indicated that R_0 is most sensitive to the biting rate, the transmission probability from human to the vector, the transmission probability from vector to humans, the natural mortality rate of the vector and the recovery rate from infection. The sensitivity analysis results proposes that control strategies should target reduction of the vector population, as well as the invention of vaccines that would help improve human immune system.

Table 2: **Sensitivity indices of R_0 with respect to the model parameters.**

Parameter	Sensitivity index
β_{vh}	0.5
β_{hv}	0.5
c	1
λ	-0.49963
Λ_h	0.00058964
Λ_v	0.16667
μ_h	-0.00095467
μ_v	-0.66667
η	-0.00058964
δ	-0.16667

5 Existence of Travelling Wave Solutions

In this section, we analyze model (2.1) to investigate the existence of travelling wave solutions using the approach done in [18, 19].

Theorem 5.1. *There exists traveling wave solutions of the model (2.1).*

Proof. Define a new variable of the form $z = x - vt$, $v > 0$, where v is the propagating wave speed of model (2.1) in one-dimensional space. Let $S_h(t,x) = S_h(z)$, $I_h(t,x) = I_h(z)$, $R_h(t,x) = R_h(z)$, $S_v(t,x) = S_v(z)$, and $I_v(t,x) = I_v(z)$. Then model (2.1) is transformed into the following set:

$$\begin{aligned} D_1 S_h'' + vS_h' + \Lambda_h - \frac{\alpha S_h I_v}{(\eta + S_h)} - \mu_h S_h &= 0 \\ D_2 I_h'' + vI_h' + \frac{\alpha S_h I_v}{(\eta + S_h)} - (\mu_h + \lambda) I_h &= 0 \\ D_3 S_v'' + vS_v' + \Lambda_v - \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v S_v &= 0 \\ D_4 I_v'' + vI_v' + \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v I_v &= 0 \end{aligned} \tag{5.1}$$

here $'$ denotes the derivative with respect to the variable z. Model (5.1) may be expressed in the form

$$\begin{aligned} S_h'' + G_1 S_h' + F_1(S_h, I_h, S_v, I_v) &= 0 \\ I_h'' + G_2 I_h' + F_2(S_h, I_h, S_v, I_v) &= 0 \\ S_v'' + G_3 S_v' + F_3(S_h, I_h, S_v, I_v) &= 0 \\ I_v'' + G_4 I_v' + F_4(S_h, I_h, S_v, I_v) &= 0 \end{aligned} \tag{5.2}$$

where $F_1 = d_1(\Lambda_h - \frac{\alpha S_h I_v}{(\eta + S_h)} - \mu_h S_h)$, $F_2 = d_2(\frac{\alpha S_h I_v}{(\eta + S_h)} - (\mu_h + \lambda) I_h)$, $F_3 = d_3(\Lambda_v - \frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v S_v)$, $F_4 = d_4(\frac{\kappa S_v I_h}{(\delta + S_v)} - \mu_v I_v)$ and $d_i = \frac{1}{D_i}$, $\quad G_i = \frac{v}{D_i}$, $\quad i = 1, 2, 3, 4$. Let $x_1 = S_h'$, $x_2 = I_h'$, $x_3 = S_v'$

and $x_4 = I_v'$. Then model (5.1) is transformed into a system of first order differential equations, where $X = [x_1, S_h, x_2, I_h, x_3, S_v, x_4, I_v]^T \in \mathbb{R}^8$ and

$$\frac{dX}{dz} = f(X) = \begin{bmatrix} -G_1x_1 - F_1 \\ x_1 \\ -G_2x_2 - F_2 \\ x_2 \\ -G_3x_3 - F_3 \\ x_3 \\ -G_4x_4 - F_4 \\ x_4 \end{bmatrix} \tag{5.3}$$

with boundary conditions

$$\lim_{z \longrightarrow -\infty} (x_1, S_h, x_2, I_h, x_3, S_v, x_4, I_v) = E^0$$

$$\lim_{z \longrightarrow +\infty} (x_1, S_h, x_2, I_h, x_3, S_v, x_4, I_v) = E^*$$

where E^0 is the disease-free equilibrium point and E^* is the endemic equilibrium point. Then a travelling wave solution is a trajectory that joins E^0 and E^*. Computing the Jacobian matrix of the model (5.2) and evaluating at the disease-free equilibrium $E^0 = (\frac{\Lambda_h}{\mu_h}, 0, 0, \frac{\Lambda_v}{\mu_v}, 0)$. This yields

$$J_{E^0} = \begin{bmatrix} \frac{-v}{D_1} & \frac{\mu_h}{D_1} & 0 & 0 & 0 & 0 & 0 & \frac{\alpha\Lambda_h}{D_1(\mu_h\eta+\Lambda_h)} \\ 1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \frac{-v}{D_2} & \frac{(\mu_h+\lambda)}{D_2} & 0 & 0 & 0 & \frac{-\alpha\Lambda_h}{D_2(\mu_h\eta+\Lambda_h)} \\ 0 & 0 & 1 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{\kappa\Lambda_v}{D_3(\mu_v\delta+\Lambda_v)} & \frac{-v}{D_3} & \frac{\mu_v}{D_3} & 0 & 0 \\ 0 & 0 & 0 & 0 & 1 & 0 & 0 & 0 \\ 0 & 0 & 0 & \frac{-\kappa\Lambda_v}{D_4(\mu_v\delta+\Lambda_v)} & 0 & 0 & \frac{-v}{D_4} & \frac{\mu_v}{D_4} \\ 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \end{bmatrix} \tag{5.4}$$

It is clear that the Jacobian matrix (5.4) evaluated at disease-free equilibrium has 8 real-valued eigenvalues given by; $\lambda_{1,2} = \frac{-v \pm \sqrt{v^2+4\mu_h D_1}}{2D_1}$, $\lambda_{3,4} = \frac{-v \pm \sqrt{v^2+4(\mu_h+\lambda)D_2}}{2D_2}$, $\lambda_{5,6} = \frac{-v \pm \sqrt{v^2+4\mu_v D_3}}{2D_3}$, $\lambda_{7,8} = \frac{-v \pm \sqrt{v^2+4\mu_v D_4}}{2D_4}$ All the eigenvalues are real valued. Therefore, there exists a travelling wave solution of the model (2.1) with a propagation speed $v > 0$. □

Therefore, there exists a travelling wave profile that connects the disease-free equilibrium to the endemic equilibrium. Biologically, this means that if infectious individuals are introduced into a fully susceptible population, then there would be the formation of a transition zone of infectious individuals with a spread speed, v, whenever $R_0 > 1$.

6 Numerical Simulation and Discussion

Simulation analysis of the model (2.1) are presented to graphically illustrate the behaviour of the solutions of the model. This helps to deduct control strategies against ZikV infection. Currently, ZikV disease is controlled via effective use of treated mosquito bed-nets, spraying of insecticide, removal of mosquito breeding sites near the homesteads [12]. In all our simulations, the initial populations are taken to be $S_h^0 = 200$, $I_h^0 = 70$, $R_h^0 = 50$, $S_v^0 = 500$ and $I_v^0 = 100$, with a high mosquito population compared with human population for the infection to spread [7].

Figure 1 shows the temporal dynamics of the infected humans with varied values of the transmission

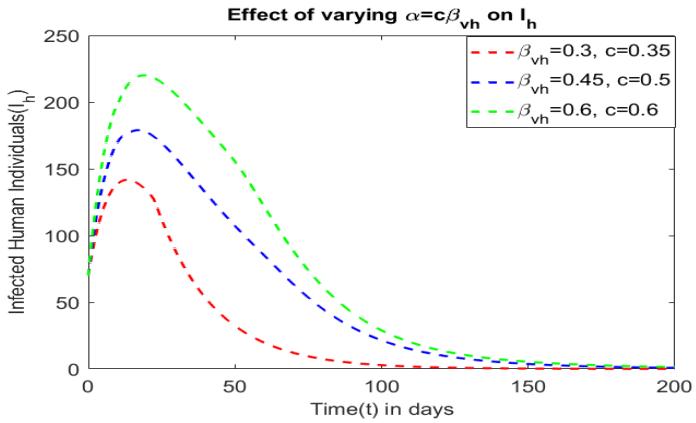


Figure 1: Temporal simulations of the infected humans. The plot shows a reduction of the infected human population as both the transmission probability from vector to human, β_{vh}, and biting rate of vector, c, increases.

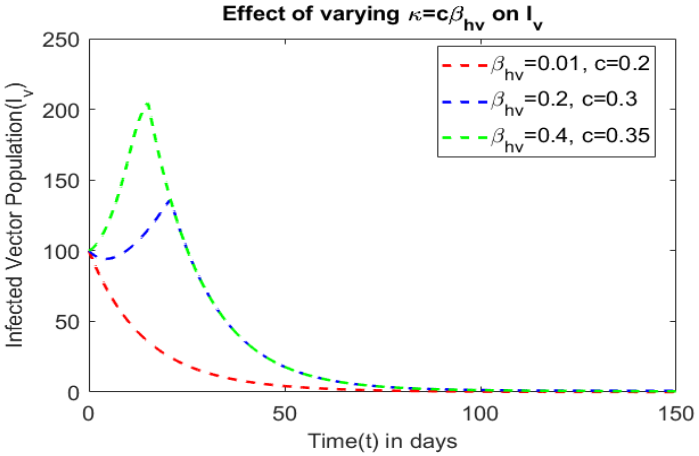
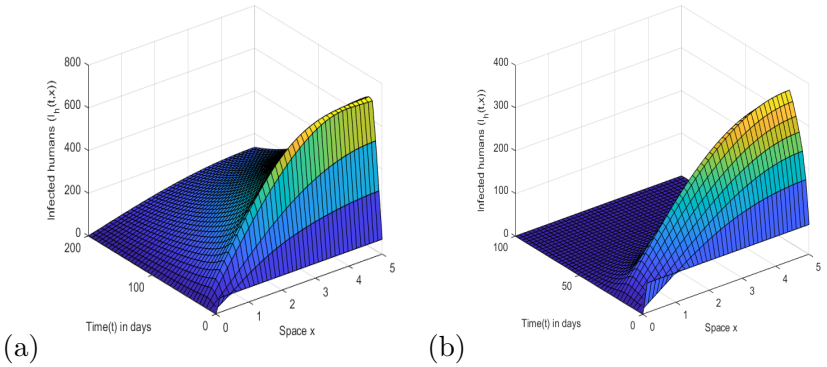


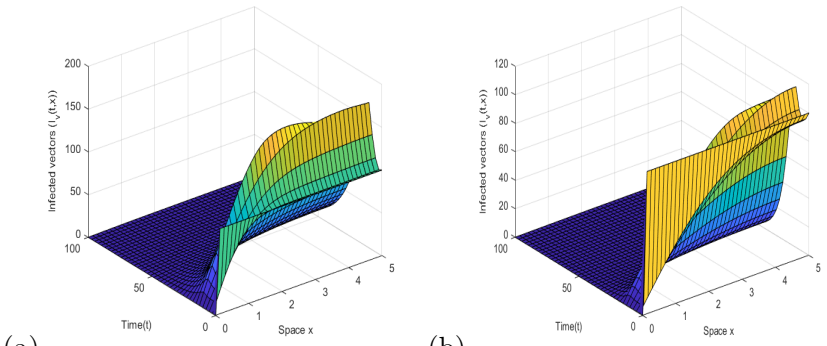
Figure 2: Temporal simulations of the infected vector population. The plot shows a reduction of the number of the infected vectors decrease as the transmission probability from human to vector, β_{hv}, and biting rate of vector, c, decreases.

probability from the vector to human, β_{vh}, and the biting rate of vector, c on the infected humans. It is observed that the infected humans reduce with a decrease in both β_{vh} and c. This implies that reducing the transmission probability from the vector to human and the biting rate of vector would gradually reduce the number of infected humans in the population. This may be achieved by use of treated mosquito bed-nets, spraying of mosquitoes breeding sites and clearing of bushes near the homesteads. Figure 2 shows the effect of transmission probability from human to the vector, β_{hv} and the biting rate, c, on the infected vectors. It is shown that the infected vector population rises for the first few days and then reduces as the transmission probability from human to the vector decreases. This implies that reduction of the transmission probability from human to vector, control strategies such use of treated mosquito window and bed-nets and treatment of symptoms



(a) (b)

Figure 3: Spatiotemporal simulations of the infected humans. Plots (a) & (b) show that the number of the infected humans reduce as the rate of diffusion, D_2 increases in both space and time, corresponding to (a) $D_2 = 0.1$, (b)$D_2 = 0.9$ day^{-1}.



(a) (b)

Figure 4: Spatiotemporal simulations of the infected vectors. Plots (a) & (b) show that the number of the infected vectors decrease with an increase of the rate of diffusion, D_4 in both space and time, corresponding to (a) $D_4 = 0.1$, (b)$D_4 = 0.5$ day^{-1}.

like fever, headache, joint pain, on the infected humans may be applied.

Figure 3 (a) & (b) shows the spatiotemporal simulations of the infected humans for varying values of diffusivity rate D_2. It shows that for the first 20 days, the number of the infected humans is high with a lower diffusion ($D_2 = 0.1$), then starts reducing. This means that, restricting infected human mobility through quarantine, would reduce the spread of the infection. Therefore, use of control strategy such as quarantine of the infected humans would reduce the transmission of the disease. Figure 4 (a) & (b) shows the spatiotemporal simulations of the infected vectors for varying values of diffusivity rate D_4. It is shown that the infected vectors increase for the first 20 days then reduce at a faster rate with an increase of the diffusivity rate. This implies that effective control measures should target reduction of the mobility of the susceptible and infected vectors. This would reduced the spread of the infection.

7 Conclusion

In this study, a spatiotemporal model on the transmission dynamics of ZikV disease is presented and analysed. First, the model well-posedness is proved. The basic reproduction number, R_0, is computed using the next generation matrix approach. The stability results showed that the model solutions would always converge to the DFE whenever $R_0 < 1$ which epidemiologically implies that if a few infectious individuals are introduced into a fully susceptible population, the disease would die out if there are no secondary infections produced whenever $R_0 < 1$, otherwise the disease would spread. Further, the stability analysis revealed that the model solutions would converge to the EE, for small perturbation whenever $R_0 > 1$. This epidemiologically implies that if a few infectious individuals are introduced in a fully susceptible population and there are new secondary infections produced whenever $R_0 > 1$, then the disease would persist in the population. The model is shown to exhibit travelling wave solutions. These waves propagate at a speed v that joins the two equilibria points. Sensitivity analysis is carried out on the parameters of, R_0, to ascertain which parameters are most influential for the virus to invade a population. The results here suggest that the vector biting rate, c, is the most influential parameter of R_0. Therefore, control measures should target reducing the biting rate of the vector. From numerical simulations, the effect of the transmission probabilities and the vector biting rate, control strategies such as the use of insecticide, treated mosquito bed and window nets, clearing of bushes near the homesteads are deduced to be more significant in reducing the spread of ZikV disease. Furthermore, from the simulations of the diffusion model, control strategies such as treatment of symptoms and quarantine of infected humans are also deduced. These proposed control strategies have also been identified by studies such as [1, 7].

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