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Investigating the Role of Mobility between Rural Areas and Forests on the Spread of Zika

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Abstract: **A mathematical model of Zika virus transmission, incorporating human movement between rural areas and nearby forests, is presented to investigate the role of human movement in the spread of Zika virus infections in human and mosquito populations. Proportions of both susceptible and infected humans living in rural areas are assumed to move to nearby forest areas. Direct, indirect, and vertical transmission routes are incorporated for all populations. A mathematical analysis of the proposed model is presented. The analysis starts with normalizing the proposed model. The positivity and boundedness of solutions to the normalized model are then addressed. The basic reproduction number is calculated using the next-generation matrix method and its relation to the three routes of disease transmission has been presented. The sensitivity analysis of the basic reproduction number to all model parameters is investigated. The analysis also includes the existence and stability of disease-free and endemic equilibrium points. Bifurcation analysis is also carried out. Finally, numerical solutions to the normalized model are obtained to confirm the theoretical results and demonstrate human movement's role in disease transmission in human and mosquito populations.**

Keywords: **Zika, Vertical Transmission, Basic Reproduction Number, Stability Analysis, Sensitivity Analysis, Bifurcation Analysis**

I. Introduction

Zika is an arboviral disease in the genus flavivirus closely related to yellow fever, West Nile (WN), and dengue (DEN) viruses. It was first identified in 1947 in Zika Forest in Uganda during sylvatic yellow fever surveillance in a sentinel rhesus monkey [1]. In 1954, it was reported in humans for the first time in Nigeria [2]. The Zika epidemic was stated as a Public Health Emergency of International Concern (PHEIC) by the World Health Organization (WHO) on February 1st, 2016 [3]. It has attracted global attention since its worldwide spread among tropical and subtropical regions. In Yap Island, Micronesia in 2007, the first Zika outbreak occurred among humans [4]. During 2013-2014 the largest epidemic of Zika ever reported was in French Polynesia [4]. Since 2014, the Zika virus (ZIKV) has continued spreading to other pacific islands [2]. It reached southern and Central America after 2015 and Brazil and the Caribbean were highly affected by ZIKV [4]. Local transmission of ZIKV was realized in 34 countries by March 2016 [5].

ZIKV is transmitted primarily to the human popu-

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lation by bites of infected female Aedes mosquitoes. Analysts have found 19 species of Aedes mosquitoes competent of carrying Zika infection, but the foremost common is the tropical privateer, Aedes aegypti. The vector (mosquito) can pass into the human population through biting after taking a blood meal from an infected human. In addition, sexual interaction, perinatal transmission, and blood transfusion are other routes of spreading ZIKV between humans even months after infection. A pregnant woman can pass Zika to her baby, which can cause genuine birth defects. Infection with Zika increases the chances of the infant developing injury with microcephaly as reported in [6] and Guillian syndrome as reported in [2] from infected mothers [4]. In February 2016, France registered the first sexually transmitted case of ZIKV [3].

Zika disease is characterized by mild symptoms including fever, headache, maculopapular rash, joint and muscle pain, conjunctivitis, etc. The clinical symptoms duration is within two to seven days after the bites [3]. Most reports show that Zika is a self–limiting febrile disease that could be misidentified as dengue or chikungunya fever [7].

The prevention of mosquito bites and control of vectors by using insecticide, eradication of adult and larval breeding areas is the only possible treatment available till now [8].

Understanding virus transmission and disease epidemiology through mathematical modelling are of great importance for disease management. Several mathematical models have been developed to study the dynamics and propose control strategies for the transmission of ZIKV disease. In [3], the authors proposed a Zika mathematical model by assuming the standard incidence type interaction of human-to-human transmission of the illness. Also, they extended their work to include optimal control programs (insecticide-treated bed nets, mosquito-repulsive lotions, and electronic devices) to reduce the biting rate of vectors, and to decline the spread of the disease among the human population. In [8], authors proposed a Zika mathematical model including the applications of prevention, treatments, and insecticide as the best way to minimize the spread of ZIKV disease. In [9], researchers suggested a multifold Zika mathematical model. They considered the transmission of the ZIKV in the adult population and infants either directly by vector bites or through vertical transmission from mothers. The model shows that asymptomatic individuals magnify the disease weight in the community. It also indicated that postponing conception, coupled with aggressive vector control and personal protection use, decrease the cases of microcephaly and transmission of ZIKV.

Globally, the survival of around 1.6 billion rustic people depends on products obtained from local forests, in whole or in part. Those individuals live adjacent to the forest and have had simple survival conditions and livelihoods for many generations. They depend on those natural and wild resources to meet their needs [10].

In this paper, a mathematical model of ZIKV is constructed to demonstrate the specific and realistic conditions, where the nearby movement of humans may contribute to the spread of virus infections. This happens when an infected human with mild symptoms, moves from rural areas to nearby forest areas looking for work or food. Additionally, the movement of a susceptible human can affect the spread of infections via contagious mosquitoes in the forest. Hence, in this paper, we have split the vector compartment, based on mosquito location, into rural areas and nearby forest areas. Human movement between rural areas and their interaction with vector populations are illustrated in Figure 1. In addition, sexual and vertical transmissions in the human population are considered. Also, vertical transmission from a contaminated female mosquito to its offspring, is suggested as a component that guarantees the upkeep of ZIKV.

The paper is organized as follows. The model formulation is described in Section 2. The model analysis includes the positivity, boundedness of the solution, basic reproduction number, and sensitivity analysis are discussed in Section 3. Furthermore, stability analysis and bifurcation analysis are presented. A numerical analysis of the model using assumed baseline parameters is given in Section 4 to illustrate the effects of highly sensitive parameters on the human population. Finally, the conclusion is given in Section 5.

II. Model Description

In this section, we introduce a model for ZIKV transmission between humans and vectors in rural areas and nearby forests. We begin the description of the model with the human compartments. We split the human population into susceptible S_h, symptomatic I_h and recovered R_h. Susceptible humans S_h can get infected with Zika via three main routes [11]: via a mosquito bite (vector transmission), via sexual transmission or blood transfusion (direct transmission), or by being passed from a mother to a newborn child (vertical transmission).

Zika causes nearly no mortality among humans and has been a public health crisis for a relatively short pe-

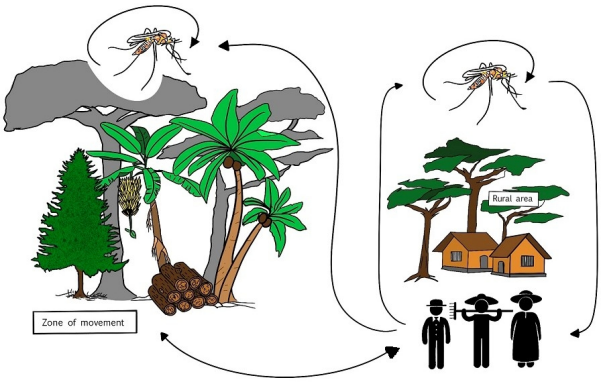

Fig. 1: Illustrated figure for human movement and their interactions with vector populations of ZIKV.

riod of time, so we assume the total human population remains constant: $S_h + I_h + R_h = N_H$.

We split the vector population into the rural population (S_v, I_v) and the nearby forest population (S_u, I_u). The overall vector populations at time t are $S_v + I_v = N_V$ and $S_u + I_u = N_U$. Rural and forest mosquitoes are assumed to be only infected by infectious humans.

The infection period of mosquitoes ends when the mosquitoes die. As mosquitoes travel distances of no more than a few kilometres, forest mosquitoes will have a direct interaction only with the human population moving from rural areas to the forest. Hence, we assume that a proportion κ_1 of the susceptible individuals may get infected by infectious mosquitoes that live in forests and nearby rural areas I_u due to their movement to forest areas, and a proportion κ_2 of the infected individuals are assumed to move from rural areas to the nearby forests such that $\kappa_1 > \kappa_2$ and hence they may infect mosquitoes that live in forests.

The proportion $(1-\kappa_1)$ of susceptible humans who stay in the rural areas can get infected by infectious mosquitoes that live in rural areas I_v, and a proportion $(1-\kappa_2)$ of infected individuals who stay in the rural areas may infect mosquitoes that live in rural areas.

Moreover, a proportion $(1-\kappa_1)$ of susceptible individuals can also get the infection by interaction with $(1-\kappa_2)$ of infectious humans (symptomatic), through sexual transmission or other direct routes.

We assume that a fraction ε_1 of newborns are affected and enter the symptomatic class. Evidence suggests that the fraction is about $2/3$ [12]. We also assume that ZIKV is transmitted vertically in the vector population [13] and this is the main pathway it survives in the colder months. We incorporate vertical transmission $\varepsilon_2, \varepsilon_3$ of the ZIKV in both vector populations, respectively.

The set of non-linear differential equations that represents the proposed mathematical model is given by:

$$\begin{aligned}
S_h' &= \mu_H N_H - \mu_H \varepsilon_1 I_h - (1-\kappa_1)\beta_1\theta_1 I_v \frac{S_h}{N_H} \\
&\quad - \kappa_1\beta_2\theta_1 I_u \frac{S_h}{N_H} - (1-\kappa_1)(1-\kappa_2)\lambda I_h \frac{S_h}{N_H} \\
&\quad - \mu_H S_h \\
I_h' &= \mu_H \varepsilon_1 I_h + (1-\kappa_1)\beta_1\theta_1 I_v \frac{S_h}{N_H} + \kappa_1\beta_2\theta_1 I_u \frac{S_h}{N_H} \\
&\quad + (1-\kappa_1)(1-\kappa_2)\lambda I_h \frac{S_h}{N_H} - (\gamma+\mu_H) I_h \\
R_h' &= \gamma I_h - \mu_H R_h \\
S_v' &= \mu_V N_V - \mu_V \varepsilon_2 I_v - (1-\kappa_2)\beta_1\theta_2 S_v \frac{I_h}{N_H} \\
&\quad - \mu_V S_v \\
I_v' &= \mu_V \varepsilon_2 I_v + (1-\kappa_2)\beta_1\theta_2 S_v \frac{I_h}{N_H} - \mu_V I_v \\
S_u' &= \mu_U N_U - \mu_U \varepsilon_3 I_u - \beta_2\theta_2\kappa_2 S_u \frac{I_h}{N_H} - \mu_U S_u \\
I_u' &= \mu_U \varepsilon_3 I_u + \beta_2\theta_2\kappa_2 S_u \frac{I_h}{N_H} - \mu_U I_u
\end{aligned} \tag{1}$$

with non negative initial conditions $S_h(0)$, $I_h(0)$, $R_h(0)$, $S_v(0)$, $I_v(0)$, $S_u(0)$, $I_u(0)$. In addition, the parameters and their values of the system are defined in Table I.

Let

$$\begin{aligned}
&S_H = \frac{S_h}{N_H}, \quad I_H = \frac{I_h}{N_H}, \quad R_H = \frac{R_h}{N_H}, \\
&S_V = \frac{S_v}{N_V}, \quad I_V = \frac{I_v}{N_V}, \quad S_U = \frac{S_u}{N_U}, \quad I_U = \frac{I_u}{N_U},
\end{aligned}$$

such that

$$S_H + I_H + R_H = 1, \; S_V + I_V = 1, \; S_U + I_U = 1.$$

Thus, the considered model (1) have been normalized and rewritten as follows:

$$\begin{aligned}
S_H' &= \mu_H - \mu_H \varepsilon_1 I_H - \overline{\kappa}_1\beta_1\theta_1\alpha_1 I_V S_H \\
&\quad - \kappa_1\beta_2\theta_1\alpha_2 I_U S_H - \overline{\kappa}_1\overline{\kappa}_2\lambda I_H S_H - \mu_H S_H \\
I_H' &= \mu_H \varepsilon_1 I_H + \overline{\kappa}_1\beta_1\theta_1\alpha_1 I_V S_H + \kappa_1\beta_2\theta_1\alpha_2 I_U S_H \\
&\quad + \overline{\kappa}_1\overline{\kappa}_2\lambda I_H S_H - (\gamma+\mu_H) I_H \\
R_H' &= \gamma I_H - \mu_H R_H \\
S_V' &= \mu_V - \mu_V \varepsilon_2 I_V - \overline{\kappa}_2\beta_1\theta_2 S_V I_H - \mu_V S_V \\
I_V' &= \mu_V \varepsilon_2 I_V + \overline{\kappa}_2\beta_1\theta_2 S_V I_H - \mu_V I_V \\
S_U' &= \mu_U - \mu_U \varepsilon_3 I_U - \beta_2\theta_2\kappa_2 S_U I_H - \mu_U S_U \\
I_U' &= \mu_U \varepsilon_3 I_U + \beta_2\theta_2\kappa_2 S_U I_H - \mu_U I_U
\end{aligned} \tag{2}$$

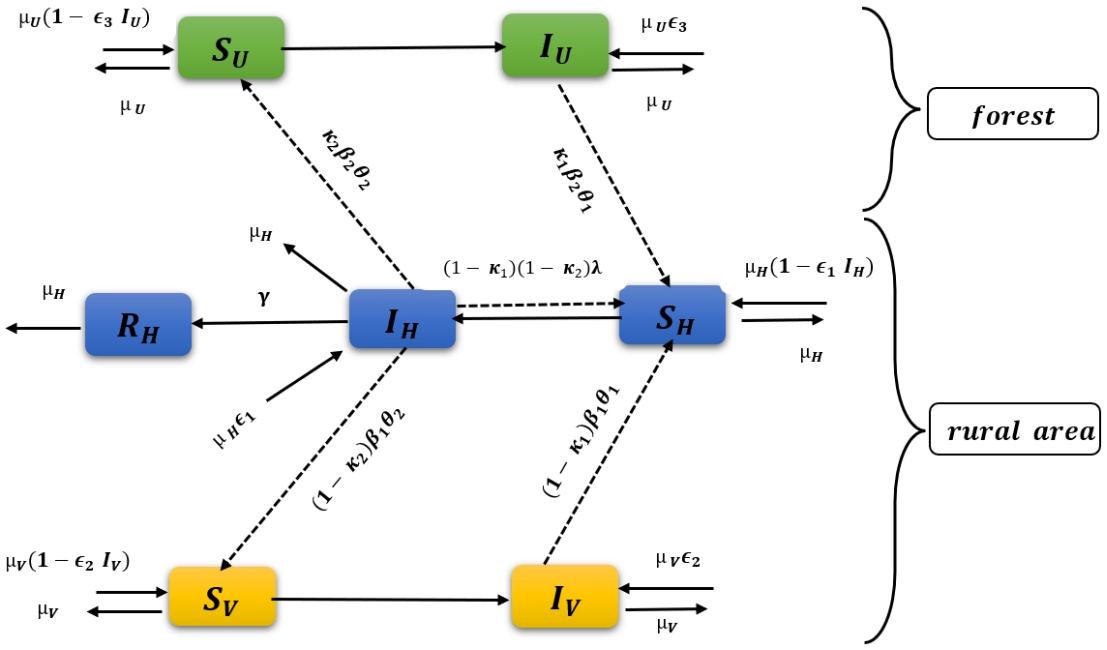

Fig. 2: Progression diagram of the proposed ZIKV model.

Table I: Parameters used in the model (1).

Parameter	Symbol	Value per day
Natural death/birth rate of humans	μ_H	1/(68.5*365) [14]
Natural death/birth rate of mosquitoes in rural areas	μ_V	[0.025-0.125] [15, 16]
Natural death/birth rate of mosquito in forest areas	μ_U	[0.025-0.125] [15, 16]
Biting rate of rural mosquitoes on humans	β_1	[0.3-1.5] [17]
Biting rate of forest mosquitoes on humans	β_2	[0.3-1.5] [17]
Transmission probability from an infectious mosquito to a susceptible human	θ_1	[0.1–0.75] [17]
Transmission probability from an infectious human to a susceptible mosquito	θ_2	[0.3–0.75] [17]
Direct (sexual) transmission rate between humans	λ	[0.01-0.47] [18]
Recovery rate of humans	γ	[0.07-0.33] [16]
Probability of vertical transmission in humans	ε_1	0.67 [12]
Probability of vertical transmission in rural mosquitoes	ε_2	0.06 [19]
Probability of vertical transmission in forest mosquitoes	ε_3	0.06 [19]
Fraction of susceptible humans moving from rural to forest areas	κ_1	[0-0.5]
Fraction of infected humans moving from rural to forest areas	κ_2	[0-0.5]

where

$$\overline{\kappa}_1 = (1-\kappa_1), \qquad \overline{\kappa}_2 = (1-\kappa_2),$$
$$\alpha_1 = \frac{N_V}{N_H}, \qquad \alpha_2 = \frac{N_U}{N_H},$$

and with non-negative initial condition

$$X(0) := \Big(S_H(0), I_H(0), R_H(0), S_V(0), I_V(0), S_U(0), I_U(0)\Big)^T.$$

III. Model Analysis

In this section, the positivity of solutions, the positive invariant set, and the basic reproduction number are discussed. Also, sensitivity analysis and results related to stability analysis and bifurcation analysis are presented.

A. Positivity of Solutions and Positively Invariant Set

It is clear that model (1) together with the given non-negative initial condition has a unique solution. Next, we show that all solutions remain non-negative for all $t \in [0, \infty)$ for arbitrary choice of initial con-

ditions to have an epidemiological convincing result. The following theorem demonstrates the positivity and boundedness of state variables:

Theorem 1. *The solutions $S_H(t)$, $I_H(t)$, $R_H(t)$, $S_V(t)$, $I_V(t)$, $S_U(t)$ and $I_U(t)$ of system (2) with non-negative initial conditions $S_H(0)$, $I_H(0)$, $R_H(0)$, $S_V(0)$, $I_V(0)$, $S_U(0)$, $I_U(0)$ remain non-negative for all time $t \geqslant 0$ in a positively invariant closed set*

$$\Omega := \Big\{(S_H, I_H, R_H, S_V, I_V, S_U, I_U)^T \in R_+^7 : 0 \leqslant S_H(t), I_H(t), R_H(t), S_V(t), I_V(t), S_U(t), I_U(t) \leqslant 1\Big\}.$$

Proof: Assume that the initial conditions of the system (2) are non-negative. Let $t_1 > 0$ be the first time at which there exists at least one component which is equal to zero and other components are non-negative on $[0, t_1)$. In the following, we will show that none of the components can be zero at t_1. Let's first assume that $S_H(t_1) = 0$ and other components are non-negative on $[0, t_1)$. Now, S'_H can be written as

$$S'_H = \mu_H(1-\varepsilon_1)+\mu_H\varepsilon_1 R_H - m_1 S_H - \mu_H(1-\varepsilon_1)S_H,$$

where

$$m_1 = \overline{\kappa}_1\beta_1\theta_1\alpha_1 I_V + \kappa_1\beta_2\theta_1\alpha_2 I_U + \overline{\kappa}_1\overline{\kappa}_2\lambda I_H > 0.$$

Then, at t_1, we have

$$\left.\frac{S_H(t)}{dt}\right|_{t=t_1} = \mu_H(1-\varepsilon_1) + \mu_H\varepsilon_1 R_H(t_1) > 0,$$

which means that $S_H(t)$ is strictly monotonically increasing at t_1, that is $S_H(t) < S_H(t_1)$ for all $t \in (t_1 - \varepsilon, t_1)$, where $\varepsilon > 0$. Since $S_H(t_1) = 0$, then, $S_H(t) < 0$ on $(t_1 - \varepsilon, t_1)$. This leads to a contradiction. Therefore, $S_H(t)$ cannot be zero at t_1.

Now, we assume that $I_H(t_1) = 0$ and other components are non-negative. Then

$$\left.\frac{I_H(t)}{dt}\right|_{t=t_1} = S_H(t_1)\big(\overline{\kappa}_1\beta_1\theta_1\alpha_1 I_V(t_1) + \kappa_1\beta_2\theta_1\alpha_2 I_U(t_1)\big) > 0,$$

which means that $I_H(t)$ is strictly monotonically increasing at t_1. Hence, we also get a contradiction.

Next, assume that $R_H(t_1) = 0$ and other components are non-negative. Then

$$\left.\frac{R_H(t)}{dt}\right|_{t=t_1} = \gamma I_H(t_1) > 0,$$

which again leads to a contradiction.

Similarly, one can prove that the remaining components of vector populations $S_V(t)$, $I_V(t)$, $S_U(t)$, $I_U(t)$ cannot be zero at t_1. Hence, from the above, we conclude that such a point t_1 at which at least one component is zero does not exist. Hence, all components remain non-negative for all time $t \geqslant 0$.

For the positively invariant closed set Ω, we first note that the set Ω is said to be positively invariant if the initial conditions are in Ω implies that

$$\Big(S_H(t), I_H(t), R_H(t), S_V(t), I_V(t), S_U(t), I_U(t)\Big)^T \in \Omega.$$

Let

$$\Phi(t) = (\Phi_1(t), \Phi_2(t), \Phi_3(t))^T,$$

where

$$\begin{aligned}\Phi_1(t) &= S_H(t) + I_H(t) + R_H(t),\\ \Phi_2(t) &= S_V(t) + I_V(t),\\ \Phi_3(t) &= S_U(t) + I_U(t).\end{aligned}$$

Then

$$\Phi'(t) = \begin{bmatrix} \mu_H - \mu_H\Phi_1(t) \\ \mu_V - \mu_V\Phi_2(t) \\ \mu_U - \mu_U\Phi_3(t) \end{bmatrix}.$$

Now, solving for Φ_1, Φ_2 and Φ_3, we get

$$\begin{aligned}\Phi_1(t) &= 1 - (1 - \Phi_1(0))\, e^{-\mu_H t},\\ \Phi_2(t) &= 1 - (1 - \Phi_2(0))\, e^{-\mu_V t},\\ \Phi_3(t) &= 1 - (1 - \Phi_3(0))\, e^{-\mu_U t},\end{aligned}$$

where

$$\begin{aligned}\Phi_1(0) &= S_H(0) + I_H(0) + R_H(0),\\ \Phi_2(0) &= S_V(0) + I_V(0),\\ \Phi_3(0) &= S_U(0) + I_U(0).\end{aligned}$$

It is straightforward to conclude that

$$\begin{aligned}\Phi_1(t) \leqslant 1 \quad &\text{if} \quad \Phi_1(0) \leqslant 1,\\ \Phi_2(t) \leqslant 1 \quad &\text{if} \quad \Phi_2(0) \leqslant 1,\\ \Phi_3(t) \leqslant 1 \quad &\text{if} \quad \Phi_3(0) \leqslant 1.\end{aligned}$$

Thus, we have

$$0 \leqslant S_H(t), I_H(t), R_H(t), S_V(t), I_V(t), S_U(t), I_U(t) \leqslant 1$$

and hence the set Ω is positively invariant set. Moreover, the set Ω is a globally attractive set since if $\Phi_i(0) > 1$ then $\lim_{t\to\infty} \Phi_i(t) = 1$ for $i = 1, 2, 3$. ■

B. The Basic Reproduction Number

The model (2) has a disease-free equilibrium (DFE):

$$Z^0 := (S_H^0, 0, 0, S_V^0, 0, S_U^0, 0) \in \Omega,$$

where $S_H^0 = S_V^0 = S_U^0 = 1$.

The number of new infections produced by a typical infected individual in a population at DFE is called the basic reproduction number R_0 which can be obtained by applying the Next Generation Method [20]. The next-generation matrix is:

$$PQ^{-1} = \begin{bmatrix} \frac{\lambda\overline{\kappa}_1\overline{\kappa}_2}{\gamma+\mu_H(1-\varepsilon_1)} & \frac{\overline{\kappa}_1\alpha_1\beta_1\theta_1}{\mu_V(1-\varepsilon_2)} & \frac{\kappa_1\alpha_2\beta_2\theta_1}{\mu_U(1-\varepsilon_3)} \\ \frac{\overline{\kappa}_2\beta_1\theta_2}{\gamma+\mu_H(1-\varepsilon_1)} & 0 & 0 \\ \frac{\kappa_2\beta_2\theta_2}{\gamma+\mu_H(1-\varepsilon_1)} & 0 & 0 \end{bmatrix},$$

where P is the Jacobian of the transmission matrix which describes the production of new infections, whereas Q is the Jacobian of the transition matrix which describes changes in state and they are given by

$$P = \begin{bmatrix} \lambda\overline{\kappa}_1\overline{\kappa}_2 & \overline{\kappa}_1\alpha_1\beta_1\theta_1 & \kappa_1\alpha_2\beta_2\theta_1 \\ \overline{\kappa}_2\beta_1\theta_2 & 0 & 0 \\ \kappa_2\beta_2\theta_2 & 0 & 0 \end{bmatrix}$$

and

$$Q = \begin{bmatrix} \gamma+\mu_H(1-\varepsilon_1) & 0 & 0 \\ 0 & \mu_V(1-\varepsilon_2) & 0 \\ 0 & 0 & \mu_U(1-\varepsilon_3) \end{bmatrix}.$$

The basic reproduction number R_0 is the dominant eigenvalue of PQ^{-1}, which can be expressed as:

$$R_0 = \frac{1}{2}\left(R_{HH} + \sqrt{R_{HH}^2 + 4(R_{HV} + R_{HU})}\right),$$

where

$$R_{HH} = \frac{\overline{\kappa}_1\overline{\kappa}_2\lambda}{\gamma+\mu_H(1-\varepsilon_1)},$$

$$R_{HV} = \frac{\alpha_1\overline{\kappa}_1\overline{\kappa}_2\beta_1^2\theta_1\theta_2}{\mu_V(1-\varepsilon_2)\big(\gamma+\mu_H(1-\varepsilon_1)\big)},$$

$$R_{HU} = \frac{\kappa_1\kappa_2\alpha_2\beta_2^2\theta_1\theta_2}{\mu_U(1-\varepsilon_3)\big(\gamma+\mu_H(1-\varepsilon_1)\big)}.$$

Note that R_{HH} represents the contribution to the reproduction number due to human-to-human transmission, R_{HV} represents the contribution to the reproduction number due to interaction between human and vector in a rural area, and R_{HU} represents the contribution to the reproduction number due to interaction between human and vector in the forest area. The square root arises from the two "generations" required for an infected vector or host to "reproduce" itself [20]. Moreover, the threshold of the disease occurs at $R_0 = 1 \iff R_{HH} + R_{HV} + R_{HU} = 1$. Also, it can be easily proven that $R_0 < 1$ implies $R_{HH} + R_{HV} + R_{HU} < 1$, which means the disease to die out, all the transmission routes represented by R_{HH}, R_{HV} and R_{HU} need to be reduced. Clearly, this will also imply that $R_0 > 1$ whenever R_{HH}, R_{HV} or R_{HU} is greater than one.

C. Sensitivity Analysis of the Basic Reproduction Number

A fundamental and valuable numeric value for the study of infectious disease dynamics is the basic reproduction number R_0, since it predicts whether an outbreak will be expected to continue (when $R_0 > 1$) or die out (when $R_0 < 1$). Sensitivity analysis of the basic reproduction number allows us to determine which model parameters have the most impact on R_0. A highly sensitive parameter leads to a high quantitative variation in R_0. Moreover, sensitivity analysis highlights the parameters that must be attacked by intervention and treatment strategies. Here, we adopt the elasticity index (normalized forward sensitivity index) [21], $E_P^{R_0}$, which computes the relative change of R_0 with respect to any parameter P as follows:

$$E_P^{R_0} = \frac{P}{R_0}\lim_{\Delta p\to 0}\frac{\Delta R_0}{\Delta P} = \frac{P}{R_0}\frac{\partial R_0}{\partial P}. \tag{3}$$

Evidently, the corresponding model parameters will affect the basic reproduction number either positively or negatively. The positive sign of the sensitivity indices of the parameter denotes the increase of the basic reproduction number R_0 as that parameter changes, whereas the negative sign of the sensitivity indices of the parameter denotes the decrease of the basic reproduction number R_0 as that parameter changes. Moreover, the magnitude denotes the relative importance of the spotlight parameter.

The biting rate of mosquitoes is a significant parameter of the epidemiology of the parasite or pathogen since it is directly related to the basic reproduction rate and affects the dynamics of disease transmission in both areas. It varies depending on the local abundance of vectors, vector host preferences, and host attractiveness [22]. Mosquitoes adjust their preferences depending on the availability of a specific host species to enhance their reproductive success [22].

It is also affected by environmental factors such as temperature, humidity, and larval food sources. The terms $(1-\kappa_1)(1-\kappa_2)\beta_1^2$ and $\kappa_1\kappa_2\beta_2^2$ in R_0 suggest that

the signs of the elasticity indices of the proportion of susceptible humans, moving from rural to forest areas κ_1 and the proportion of infected humans moving from rural to forest areas κ_2, depend on the biting rates of mosquitoes β_1 and β_2. To investigate this dependence, we use relation (3) to obtain the following relations which describe the elasticity indices of κ_1 and κ_2, respectively, in terms of both β_1 and β_2, taking all other parameters to be fixed, namely, $\kappa_1 = 0.3$, $\kappa_2 = 0.2$, $\alpha_1 = 2$, $\alpha_2 = 3$, $\varepsilon_1 = 0.67$, $\varepsilon_2 = 0.06$, $\varepsilon_3 = 0.06$, $\mu_H = 0.00004$, $\mu_V = 0.07$, $\mu_U = 0.07$, $\lambda = 0.235$, $\gamma = 0.16$, $\theta_1 = 0.33$, $\theta_2 = 0.3$:

$$E_{\kappa_1}^{R_0} = \frac{0.3\left(-0.5874 + \frac{1}{4}\frac{-1.9325 - 60.1774\beta_2^2 + 22.5665\beta_1^2}{\sqrt{0.6763 + 42.1241\beta_2^2 + 6.7699\beta_1^2}}\right)}{0.4112 + \frac{1}{2}\sqrt{0.6763 + 42.1241\beta_2^2 + 6.7699\beta_1^2}}$$

$$E_{\kappa_2}^{R_0} = \frac{0.2\left(-0.5140 + \frac{1}{4}\frac{-1.6909 - 52.6552\beta_2^2 - 33.8497\beta_1^2}{\sqrt{0.6763 + 42.1241\beta_2^2 + 6.7699\beta_1^2}}\right)}{0.4112 + \frac{1}{2}\sqrt{0.6763 + 42.1241\beta_2^2 + 6.7699\beta_1^2}}$$

Clearly, the above two equations show that the signs of the sensitivity indices of κ_1 and κ_2 depend on the values of β_1 and β_2. This dependence is illustrated in Figure 3 based on the range of values of β_1 and β_2 given in Table I. Figure 3 shows three different regions for the signs of the indices depending on the values of β_1 and β_2, namely, a region of negative indices, a region of positive indices, and a region of negative index for κ_1 and a positive index for κ_2. These three regions are all feasible for low values of the rural mosquito's biting rate β_1 up to a certain limit, taken here to be around $\beta_1 = 0.75$. In this case, the region of positive indices is only feasible for higher values of the forest mosquito's biting rate β_2, taken here to be higher than 0.8. This means that the movement of susceptible and infected humans from rural to forest areas will have the effect of increasing the basic reproduction number if the disease transmission in the forest areas is relatively higher than in the rural areas.

However, for higher values of β_1, the region of positive indices is not feasible even for high values of β_2. The only feasible positive index is for κ_2, whereas the index of κ_1 remains negative for all values of β_2 since the disease transmission in the rural area is high. Using $\beta_1 = 0.35$ and $\beta_2 = 0.9$, which correspond to the region of positive indices, we calculate the elasticity

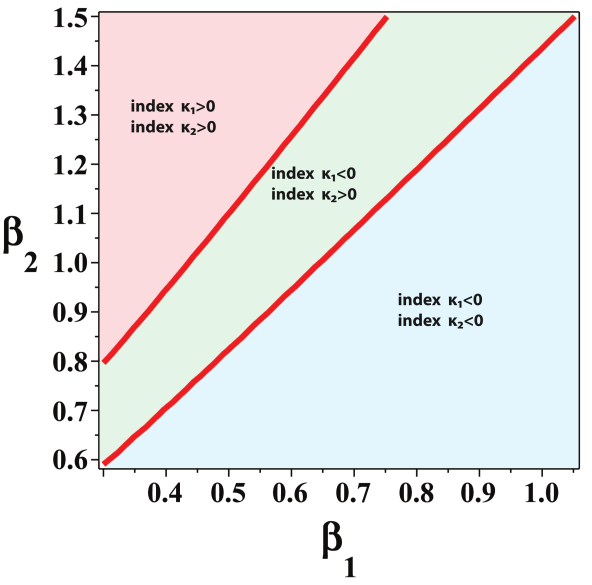

Fig. 3: Signs of the sensitivity indices of the movement rates κ_1 and κ_2 in terms of the biting rates β_1 and β_2.

Table II: Sensitivity indices and their interpretation.

Para.	Value	Sens. ind.	Interpretation	
β_1	0.35	0.36629	β_1 by 10%	R_0 by 37%
β_2	0.9	0.38926	β_2 by 10%	R_0 by 39%
λ	0.235	0.24444	λ by 10%	R_0 by 24%
κ_1	0.3	0.01138	κ_1 by 10%	R_0 by 1%
κ_2	0.2	0.08773	κ_2 by 10%	R_0 by 9%
θ_1	0.33	0.37778	θ_1 by 10%	R_0 by 38%
θ_2	0.3	0.37778	θ_2 by 10%	R_0 by 38%
γ	0.16	-0.62217	γ by 10%	R_0 by 62%
α_1	2	0.18315	α_1 by 10%	R_0 by 18%
α_2	3	0.19463	α_2 by 10%	R_0 by 19%
μ_H	0.00004	-0.00005	μ_H by 10%	R_0 by 0.005%
μ_V	0.07	-0.18315	μ_V by 10%	R_0 by 18%
μ_U	0.07	-0.19463	μ_U by 10%	R_0 by 19%
ε_1	0.67	0.000104	ε_1 by 10%	R_0 by 0.01%
ε_2	0.06	0.01169	ε_2 by 10%	R_0 by 0.11%
ε_3	0.06	0.01242	ε_3 by 10%	R_0 by 12%

indices for all parameters. The obtained values and their interpretations are listed in Table II.

Clearly, the most efficacious parameter is the biting rate of forest mosquitoes on humans β_2, i.e., it has a strong positive impact on the value of R_0. Also, the biting rate of forest mosquitoes on humans β_1 has a positive impact on R_0. The transmission probabilities per bite – per human θ_1 and per mosquito θ_2 – have a positive influence on the value of R_0. Similarly, one can note that the proportions of movement for susceptible humans κ_1 and infected κ_2 have a small positive effect on R_0, with κ_2 having a higher index than κ_1. On the

other hand, the recovery rate of humans γ has the most negative sensitivity index, as it will decrease R_0 by 62% when it increases by 10%. Clearly, there is a very small positive effect of the vertical transmission of humans and both vectors $\varepsilon_1, \varepsilon_2, \varepsilon_3$ with ε_3 having the highest index among them.

Using the parameter values listed in Table II, the basic reproduction number is estimated to be $R_0 = 2.093$. This value is close to the one obtained in [23], in which the authors have estimated from notification data that the basic reproduction number for ZIKV in Rio de Janeiro is $R_0 = 2.33$.

D. Local Stability of the DFE

Here we discuss the local stability of the DFE by finding the eigenvalues of the linearized system. The following theorem is devoted to the local stability of the DFE, i.e., the disease would be eliminated under certain conditions.

Theorem 2. *If $R_0 \leq 1$, the DFE of the model (2) is locally asymptotically stable. If $R_0 > 1$, it is unstable.*

Proof: The linearized matrix of the system (2) at the disease-free equilibrium Z^0 is:

$$J_{Z^0} = \begin{bmatrix} -\mu_H & -\mu_H\varepsilon_1 - \lambda\overline{\kappa}_1\overline{\kappa}_2 & 0 & 0 \\ 0 & -\gamma - \mu_H(1-\varepsilon_1) + \lambda\overline{\kappa}_1\overline{\kappa}_2 & 0 & 0 \\ 0 & \gamma & -\mu_H & 0 \\ 0 & -\beta_1\overline{\kappa}_2\theta_2 & 0 & -\mu_V \\ 0 & \beta_1\overline{\kappa}_2\theta_2 & 0 & 0 \\ 0 & -\beta_2\kappa_2\theta_2 & 0 & 0 \\ 0 & \beta_2\kappa_2\theta_2 & 0 & 0 \end{bmatrix}$$

$$\begin{bmatrix} -\overline{\kappa}_1\alpha_1\beta_1\theta_1 & 0 & -\kappa_1\alpha_2\beta_2\theta_1 \\ \overline{\kappa}_1\alpha_1\beta_1\theta_1 & 0 & \kappa_1\alpha_2\beta_2\theta_1 \\ 0 & 0 & 0 \\ -\mu_V\varepsilon_2 & 0 & 0 \\ -\mu_V(1-\varepsilon_2) & 0 & 0 \\ 0 & -\mu_U & -\mu_U\varepsilon_3 \\ 0 & 0 & -\mu_U(1-\varepsilon_3) \end{bmatrix}.$$

It is clear that the system has three negative eigenvalues which are $\ell_1 = -\mu_V$, $\ell_2 = -\mu_U$ and $\ell_3 = -\mu_H$ with multiplicity two. The remaining eigenvalues can be found from the characteristic equation $k(\ell) = 0$, where $k(\ell)$ is given by:

$$k(\ell) = \ell^3 + k_1\ell^2 + k_2\ell + k_3,$$

with

$$\begin{aligned} k_1 &= \xi(1 - R_{HH}) + \mu_V(1-\varepsilon_2) + \mu_U(1-\varepsilon_3), \\ k_2 &= \xi\mu_V(1-\varepsilon_2)(1 - R_{HH} - R_{HV}) \\ &\quad + \xi\mu_U(1-\varepsilon_3)(1 - R_{HH} - R_{HU}) \\ &\quad + \mu_V\mu_U(1-\varepsilon_2)(1-\varepsilon_3), \\ k_3 &= \\ &\quad \xi\mu_V\mu_U(1-\varepsilon_2)(1-\varepsilon_3)(1 - R_{HH} - R_{HV} - R_{HU}), \end{aligned}$$

where $\xi = \big(\gamma + \mu_H(1-\varepsilon_1)\big)$.

It is clear that $k_3 > 0$ if $R_{HH} + R_{HV} + R_{HU} < 1$ which also implies that $k_1 > 0$ and $k_2 > 0$. Hence, in order to use Routh's stability criterion [24] to show that the roots of the above characteristic equation have negative real parts, it remains to show that $k_1k_2 - k_3$ is positive, that is:

$$\begin{aligned} k_1k_2 - k_3 &= 2\xi\mu_V\mu_U(1-\varepsilon_2)(1-\varepsilon_3)(1-R_{HH}) \\ &\quad + \xi^2\mu_V(1-\varepsilon_2)(1-R_{HH})(1-R_{HH}-R_{HV}) \\ &\quad + \xi^2\mu_U(1-\varepsilon_3)(1-R_{HH})(1-R_{HU}-R_{HV}) \\ &\quad + \xi\mu_V^2(1-\varepsilon_2)^2(1-R_{HH}-R_{HV}) \\ &\quad + \xi\mu_U^2(1-\varepsilon_3)^2(1-R_{HH}-R_{HU}) \\ &\quad + \mu_V^2\mu_U(1-\varepsilon_2)^2(1-\varepsilon_3) \\ &\quad + \mu_V\mu_U^2(1-\varepsilon_2)(1-\varepsilon_3)^2. \end{aligned}$$

Clearly, $k_1k_2 - k_3 > 0$ if and only if $R_{HH} + R_{HV} < 1$ and $R_{HH} + R_{HU} < 1$.

Therefore, by Routh's stability criterion, the roots of the characteristic equation $k(\ell) = 0$ have negative real parts, and hence we conclude that the DFE is locally asymptotically stable whenever $R_0 \leq 1$. Otherwise, it is unstable. ■

E. Global Stability of the DFE

When the solution of the dynamical system (2) approaches a unique equilibrium point regardless of initial conditions then the equilibrium point is globally asymptotically stable. The global stability of the DFE will ensure that the disease is eliminated under all initial conditions. In this regard, we state and prove the following theorem:

Theorem 3. *If $R_0 \leq 1$, the disease-free equilibrium Z^0 is globally asymptotically stable on the compact set Ω.*

Proof: Applying Castillo-Chavez theorem [25], consider the following two compartments:

$$X(t) = \begin{bmatrix} S_H(t) \\ R_H(t) \\ S_V(t) \\ S_U(t) \end{bmatrix}, \qquad Y(t) = \begin{bmatrix} I_H(t) \\ I_V(t) \\ I_U(t) \end{bmatrix},$$

which describe the uninfected and infected individuals of the system (2), respectively. So that system (2) can be written as:

$$\frac{dX}{dt} = F(X,Y), \quad \frac{dY}{dt} = G(X,Y), \quad G(X,0) = 0,$$

where $F(X,Y)$ and $G(X,Y)$ are the corresponding right hand side of system (2). To guarantee the global asymptotic stability of the DFE, according to the Castillo-Chavez theorem, the following two conditions must be satisfied:

$(H1)$ For $\dfrac{dX}{dt} = F(X,0)$,
$X^0 = (1,0,1,1)^T$ is globally asymptotically stable.
$(H2)$ $\hat{G} \geqslant 0$, where $\hat{G}(X,Y) = AY - G(X,Y)$ and $A = D_Y G(X^0, 0)$ is an Metzler matrix $\forall (X,Y) \in \Omega$.

To check the first condition, we find:

$$F(X,0) = \begin{bmatrix} -\mu_H S_H + \mu_H \\ -\mu_H R_H \\ -\mu_V S_V + \mu_V \\ -\mu_U S_U + \mu_U \end{bmatrix}.$$

Solving the system of ODEs in $(H1)$, we obtain the following behavior of each component:

$$\begin{aligned} S_H(t) &= 1 + S_H(0)e^{-\mu_H t} &&\Longrightarrow \lim_{t\to\infty} S_H(t) = 1, \\ R_H(t) &= R_H(0)e^{-\mu_H t} &&\Longrightarrow \lim_{t\to\infty} R_H(t) = 0, \\ S_V(t) &= 1 + S_V(0)e^{-\mu_V t} &&\Longrightarrow \lim_{t\to\infty} S_V(t) = 1, \\ S_U(t) &= 1 + S_U(0)e^{-\mu_U t} &&\Longrightarrow \lim_{t\to\infty} S_U(t) = 1. \end{aligned}$$

Hence, the first condition is satisfied. Now, to check the second condition, we first find:

$$A = \begin{bmatrix} -\xi + \lambda\overline{\kappa}_1\overline{\kappa}_2 & \overline{\kappa}_1\alpha_1\beta_1\theta_1 & \kappa_1\alpha_2\beta_2\theta_1 \\ \overline{\kappa}_2\beta_1\theta_2 & -\mu_V(1-\varepsilon_2) & 0 \\ \kappa_2\beta_2\theta_2 & 0 & -\mu_U(1-\varepsilon_3) \end{bmatrix},$$

where $\xi = \big(\gamma + \mu_H(1-\varepsilon_1)\big)$. Then,

$$\hat{G}(X,Y) = AY - G(X,Y) = \begin{bmatrix} (\overline{\kappa}_1\alpha_1\beta_1\theta_1 I_V + \kappa_1\alpha_2\beta_2\theta_1 I_U + \overline{\kappa}_1\overline{\kappa}_2\lambda I_H)(1-S_H) \\ \beta_1\overline{\kappa}_2\theta_2 I_H(1-S_V) \\ \beta_2\kappa_2\theta_2 I_H(1-S_U) \end{bmatrix}$$

Since $0 \leqslant S_H \leqslant 1$, $0 \leqslant S_V \leqslant 1$ and $0 \leqslant S_U \leqslant 1$ then $\hat{G} \geqslant 0$ for all $(X,Y) \in \Omega$. Thus, Z^0 is globally asymptotically stable provided that $R_0 \leq 1$. ■

F. Existence of Endemic Equilibrium

The endemic equilibrium is the state where the infection cannot be totally eradicated and the disease progression persists in a population at all times but in relatively low frequency. Here, we discuss the existence of endemic equilibrium.

Theorem 4. *For model* (2) *there exists an endemic equilibrium* $Z^* \in \Omega$ *whenever* $R_0 > 1$.

Proof: Let $Z^* := (S_H^*, I_H^*, R_H^*, S_V^*, I_V^*, S_U^*, I_U^*)$ be the endemic equilibrium of the model (2) such that:

$$\begin{aligned} S_H^* &= \frac{\mu_H - (\gamma+\mu_H)I_H^*}{\mu_H}, \\ R_H^* &= \frac{\gamma I_H^*}{\mu_H}, \\ S_V^* &= \frac{\mu_V(1-\varepsilon_2)}{\mu_V(1-\varepsilon_2) + \overline{\kappa}_2\beta_1\theta_2 I_H^*}, \\ I_V^* &= \frac{\overline{\kappa}_2\beta_1\theta_2 I_H^*}{\mu_V(1-\varepsilon_2) + \overline{\kappa}_2\beta_1\theta_2 I_H^*}, \\ S_U^* &= \frac{\mu_U(1-\varepsilon_3)}{\mu_U(1-\varepsilon_3) + \kappa_2\beta_2\theta_2 I_H^*}, \\ I_U^* &= \frac{\kappa_2\beta_2\theta_2 I_H^*}{\mu_U(1-\varepsilon_3) + \kappa_2\beta_2\theta_2 I_H^*}, \end{aligned}$$

and I_H^* satisfies the following equation:

$$q_1 I_H^{*4} + q_2 I_H^{*3} + q_3 I_H^{*2} + q_4 I_H^* = 0,$$

where

$$\begin{aligned} q_1 &= \beta_1\beta_2\lambda\theta_2^2\kappa_2\overline{\kappa}_2(\gamma+\mu_H), \\ q_2 &= \xi\beta_1\beta_2\kappa_2\overline{\kappa}_2\theta_2^2\mu_H(1-R_{HH}) \\ &\quad + \xi\beta_2\kappa_2\theta_2\mu_V(1-\varepsilon_2)(\gamma+\mu_H)(R_{HH}+R_{HV}) \\ &\quad + \xi\overline{\kappa}_2\beta_1\theta_2\mu_U(1-\varepsilon_3)(\gamma+\mu_H)(R_{HH}+R_{HU}), \\ q_3 &= \xi\beta_2\kappa_2\theta_2\mu_H\mu_V(1-\varepsilon_2)(1-R_{HH}-R_{HV}) \\ &\quad + \xi\beta_1\theta_2\overline{\kappa}_2\mu_H\mu_U(1-\varepsilon_3)(1-R_{HH}-R_{HU}) \\ &\quad + \xi\mu_V\mu_U(1-\varepsilon_2)(1-\varepsilon_3)(\gamma+\mu_H) \\ &\qquad (R_{HH}+R_{HV}+R_{HU}), \\ q_4 &= \xi\mu_H\mu_V\mu_U(1-\varepsilon_2)(1-\varepsilon_3) \\ &\qquad (1-R_{HH}-R_{HV}-R_{HU}), \end{aligned}$$

where $\xi = \big(\gamma + \mu_H(1-\varepsilon_1)\big)$.

Solving the above equation we get $I_H^* = 0$, which corresponds to the DFE (Z^0) and the remaining roots satisfy the cubic equation:

$$q_1 I_H^{*3} + q_2 I_H^{*2} + q_3 I_H^* + q_4 = 0.$$

Clearly, if $R_{HH} + R_{HV} + R_{HU} > 1$, then the above equation has a positive root, since $q_1 > 0$ and $q_4 < 0$.

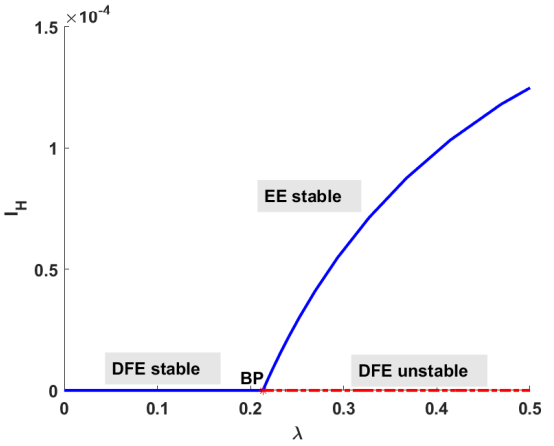

Fig. 4: Bifurcation figure when λ is taken as a bifurcation parameter of system (2) with a bifurcation value $\lambda^* = 0.2132$ at $R_0 = 1$ and by fixing parameter $\alpha_1 = 2$, $\alpha_2 = 3$, $\kappa_1 = 0.3$, $\kappa_2 = 0.2$, $\varepsilon_1 = 0.67$, $\varepsilon_2 = 0.06$, $\varepsilon_3 = 0.06$, $\mu_H = 0.00004$, $\mu_V = 1/14$, $\mu_U = 1/14$, $\beta_1 = 0.15$, $\beta_2 = 0.1$, $\gamma = 0.16$, $\theta_1 = 0.33$, $\theta_2 = 0.3$.

Now, note that q_3 can be written in terms of q_2 as follows:

$$\begin{aligned} q_3 = \xi\Big(&\mu_H\theta_2\big(\beta_2\kappa_2\mu_V(1-\varepsilon_2) + \beta_1\overline{\kappa}_2\mu_U(1-\varepsilon_3)\big) \\ &+ \mu_V\mu_U(1-\varepsilon_2)(1-\varepsilon_3)(\gamma+\mu_H) \\ &\quad (R_{HH}+R_{HV}+R_{HU})\Big) \\ &- \frac{\mu_H}{\gamma+\mu_H}\big(q_2 - \xi\beta_1\beta_2\kappa_2\overline{\kappa}_2\theta_2^2\mu_H(1-R_{HH})\big). \end{aligned}$$

To ensure the uniqueness of the positive roots, we apply Descartes's Sign Rule [26]. There exists a unique positive root when $q_2 > 0$ regardless of the sign of q_3 and this happens if $R_{HH} < 1$ and $R_{HH} + R_{HU} + R_{HV} > 1$. However, when $q_2 < 0$ and $R_{HH} + R_{HU} + R_{HV} > 1$ there exist at least one positive root. Note that the existence of three positive roots is only possible when $q_2 < 0$ and $q_3 > 0$. ■

G. Bifurcation Analysis

When the stability of a system is changed as a parameter changes causing the emergence or disappearance of new stable points, then the system is said to undergo bifurcation. In this section, we prove that system (2) has transcritical bifurcation. The proof is based on the Sotomayor theorem described in [27]. Let F be defined as the right-hand side of the system (2) and

$$Z = (S_H, I_H, R_H, S_V, I_V, S_U, I_U)^T.$$

At $R_0 = 1$, we can check that the constant term of the characteristic equation of J_{Z^0} is zero which implies that J_{Z^0} has a simple zero eigenvalue. Here, we choose λ as a bifurcation parameter such that the bifurcation value corresponding to $R_0 = 1$ is given by:

$$\lambda^* = \frac{(\gamma + \mu_H(1-\varepsilon_1))(1 - R_{HV} - R_{HU})}{\overline{\kappa}_1\overline{\kappa}_2}.$$

Solving $J_{(Z^0,\lambda^*)}\mathrm{v} = 0$, where

$$\mathrm{v} = (v_1, v_2, v_3, v_4, v_5, v_6, v_7)^T$$

is a nonzero right eigenvector of $J_{(Z^0,\lambda^*)}$ corresponding to the zero eigenvalue, we obtain:

$$\mathrm{v} = \begin{bmatrix} -\dfrac{\gamma+\mu_H}{\gamma} \\ \dfrac{\mu_H}{\gamma} \\ 1 \\ -\dfrac{\beta_1\overline{\kappa}_2\theta_2\mu_H}{\mu_V\gamma(1-\varepsilon_2)} \\ \dfrac{\beta_1\overline{\kappa}_2\theta_2\mu_H}{\mu_V\gamma(1-\varepsilon_2)} \\ -\dfrac{\beta_2\kappa_2\theta_2\mu_H}{\mu_U\gamma(1-\varepsilon_3)} \\ \dfrac{\beta_2\kappa_2\theta_2\mu_H}{\mu_U\gamma(1-\varepsilon_3)} \end{bmatrix} v_3, \quad v_3 \neq 0.$$

Next we find the corresponding nonzero left eigenvector $\mathrm{w} = (w_1, w_2, w_3, w_4, w_5, w_6, w_7)^T$, which satisfies $J^T_{(Z^0,\lambda^*)}\mathrm{w} = 0$. We get:

$$\mathrm{w} = \begin{bmatrix} 0 \\ 1 \\ 0 \\ 0 \\ \dfrac{\alpha_1\beta_1\overline{\kappa}_1\theta_1}{\mu_V(1-\varepsilon_2)} \\ 0 \\ \dfrac{\alpha_2\beta_2\kappa_1\theta_1}{\mu_U(1-\varepsilon_3)} \end{bmatrix} w_2, \quad w_2 \neq 0.$$

Model (2) can be written as $dZ/dt = F(Z)$, where $F(Z)$ is the right hand side of the model. Now, we check the conditions of the Sotomayor theorem and begin with finding $F_\lambda(\lambda^*, Z^0)$:

$$F_\lambda(\lambda^*, Z^0) = (0,0,0,0,0,0,0)^T.$$

So, the first condition is satisfied:

$$\mathrm{w}^T F_\lambda(\lambda^*, Z^0) = 0.$$

Next, we find the Jacobian of $F_\lambda(\lambda^*, Z)$ as follows:

$$DF_\lambda(\lambda^*, Z) = \begin{bmatrix} -\overline{\kappa}_1\overline{\kappa}_2 I_H & -\overline{\kappa}_1\overline{\kappa}_2 S_H & 0 & 0 & 0 & 0 & 0 \\ \overline{\kappa}_1\overline{\kappa}_2 I_H & \overline{\kappa}_1\overline{\kappa}_2 S_H & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$

Checking the second condition, we have:

$$\mathrm{w}^T DF_\lambda(\lambda^*, Z^0)\mathrm{v} = \overline{\kappa}_1\overline{\kappa}_2 w_2 v_2 \neq 0.$$

Finally, we check the third condition by finding $D^2F(\lambda^*, Z^0)$, where D^2 denotes the matrix of the partial derivatives of each component of $DF(Z)$ and we get:

$$D^2F(\lambda^*, Z^0)(\mathrm{v},\mathrm{v}) = \begin{bmatrix} -2\lambda\overline{\kappa}_1\overline{\kappa}_2 v_1 v_2 - 2\alpha_1\overline{\kappa}_1\beta_1\theta_1 v_1 v_5 - 2\kappa_1\alpha_2\beta_2\theta_1 v_1 v_7 \\ 2\lambda\overline{\kappa}_1\overline{\kappa}_2 v_1 v_2 + 2\alpha_1\overline{\kappa}_1\beta_1\theta_1 v_1 v_5 + 2\kappa_1\alpha_2\beta_2\theta_1 v_1 v_7 \\ 0 \\ -2\overline{\kappa}_2\beta_1\theta_2 v_4 v_2 \\ 2\overline{\kappa}_2\beta_1\theta_2 v_4 v_2 \\ -2\kappa_2\beta_2\theta_2 v_6 v_2 \\ 2\kappa_2\beta_2\theta_2 v_6 v_2 \end{bmatrix}$$

Thus,

$$\begin{aligned} &\mathrm{w}^T\big(D^2F(\lambda^*, Z^0)(\mathrm{v},\mathrm{v})\big) \\ &\quad = 2v_1(\alpha_2\beta_2\theta_1\kappa_1 v_7 + \alpha_1\beta_1\theta_1\overline{\kappa}_1 v_5 + \lambda\overline{\kappa}_1\overline{\kappa}_2) \\ &\quad + 2\beta_1\theta_2\overline{\kappa}_2 v_2 v_4 w_5 + 2\beta_2\theta_2\kappa_2 v_2 v_6 w_7. \end{aligned}$$

By substituting the values of v's and w's, we get:

$$\begin{aligned} &\mathrm{w}^T\big(D^2F(\lambda^*, Z^0)(\mathrm{v},\mathrm{v})\big) \\ &\quad = -\bigg(\frac{2\mu_H(\gamma+\mu_H)(\gamma+\mu_H(1-\varepsilon_1))}{\gamma^2} \\ &\qquad + \frac{2\beta_2^3\kappa_1\kappa_2^2\theta_2^2\theta_1\alpha_2}{\gamma^2\mu_U^2(1-\varepsilon_3)^2}\bigg) w_2 v_3^2, \end{aligned}$$

which is nonzero since w_2 and v_3 are nonzero.

Hence, the system (2) experiences a transcritical bifurcation at Z^0 as the parameter λ passes through the bifurcation value $\lambda = \lambda^*$. The bifurcation diagram is created by using the MATCONT package [28] and is illustrated in Figure 4. This leads us to establish the following theorem:

Theorem 5. *Model* (2) *undergoes transcritical bifurcation at the DFE* (Z^0) *when the parameter* λ *passes through the bifurcation value* $\lambda = \lambda^*$.

Remark. We can establish the local stability of endemic equilibrium using the above calculations. We note that based on Theorem 4 in [20], a and b are given by:

$$\begin{aligned} a &= \frac{1}{2}\mathrm{w}^T\big(D^2F_\lambda(\lambda^*, Z^0)(\mathrm{v},\mathrm{v})\big) \\ &= \frac{1}{2}\sum_{i,j,k=1}^{n} v_i v_j w_k \frac{\partial^2 F_i}{\partial x_j \partial x_k}(\lambda^*, Z^0), \\ b &= \mathrm{w}^T DF_\lambda(\lambda^*, Z^0)\mathrm{v} = \sum_{i,j=1}^{n} v_i w_j \frac{\partial^2 F_i}{\partial x_j \partial \lambda}(\lambda^*, Z^0). \end{aligned}$$

According to the calculations in this section, it is clear that $b \neq 0$ and $a < 0$ if w_2 is positive. Thus, there exists $\delta > 0$ such that the endemic equilibrium Z^* is locally asymptotically stable near Z^0 for $0 < \lambda < \delta$. Moreover, according to Castillo-Chavez and Song [29] the direction of the bifurcation of the system (2) at $R_0 = 1$ is forward (supercritical bifurcation).

IV. Numerical Analysis

In this section, the forgoing theoretical results are confirmed by presenting the numerical results of the Zika SIR-SI model (2). The asymptotic behavior of the model is characterized by solving the system numerically using the numerical simulations of MATLAB with baseline parameters listed in Table I with appropriate initial conditions. We assume that the human population size is 200000, the rural mosquito population size is 400000, and the forest mosquito population size is 600000. These values are obtained by taking into consideration desired conditions or from literature.

Phase diagram for the case $R_0 < 1$ is illustrated in Figure 5. Here, the biting rates of rural and forest mosquitoes on humans are taken to be $\beta_1 = \beta_2 = 0.3$. Figure 5 shows that all populations reach the disease-free equilibrium with the disease disappearing from vector populations faster than the human population.

For the case $R_0 > 1$, we consider $q_2 > 0$ and $q_3 > 0$ and the biting rate of rural mosquitoes and of forest mosquitoes on humans to be $\beta_1 = 0.35$, $\beta_2 = 0.8$, respectively. The unique endemic equilibrium for this case is given by:

$$\begin{aligned} Z^* := (&0.05867, 0.00054, 0.940789, \\ &0.99808, 0.00192, 0.99890, 0.00109) \end{aligned}$$

and the disease dynamics are illustrated in Figure 6. It shows that the solution exhibits oscillations before reaching its steady state.

Now, we present numerical simulations for the effects of variations of κ_1 and κ_2. In Figure 7, we change the

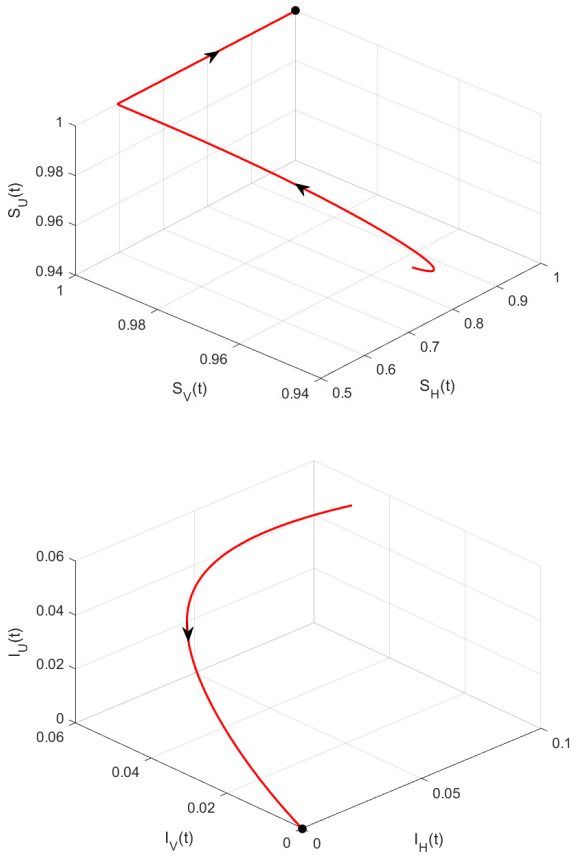

Fig. 5: Phase diagram when $R_0 < 1$ with parameter values taken to be $\alpha_1 = 2$, $\alpha_2 = 3$, $\kappa_1 = 0.3$, $\kappa_2 = 0.2$, $\varepsilon_1 = 0.67$, $\varepsilon_2 = 0.06$, $\varepsilon_3 = 0.06$, $\mu_H = 0.00004$, $\mu_V = 0.07$, $\mu_U = 0.07$, $\beta_1 = 0.3$, $\beta_2 = 0.3$, $\lambda = 0.01$, $\gamma = 0.16$, $\theta_1 = 0.2$, $\theta_2 = 0.3$.

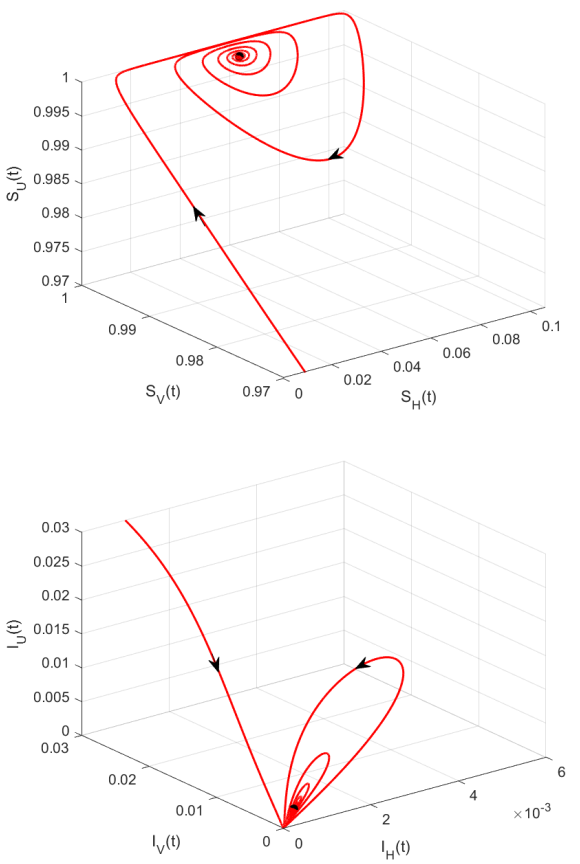

Fig. 6: Phase diagram when $R_0 > 1$ with parameter values taken to be $\alpha_1 = 2$, $\alpha_2 = 3$, $\kappa_1 = 0.3$, $\kappa_2 = 0.2$, $\varepsilon_1 = 0.67$, $\varepsilon_2 = 0.06$, $\varepsilon_3 = 0.06$, $\mu_H = 0.00004$, $\mu_V = 0.025$, $\mu_U = 0.025$, $\beta_1 = 0.35$, $\beta_2 = 0.8$, $\lambda = 0.235$, $\gamma = 0.07$, $\theta_1 = 0.33$, $\theta_2 = 0.3$.

fraction of susceptible humans moving to forest area κ_1 and fix the other parameters as listed in Table II. We note that increasing the values of κ_1 leads to a slight increase in the maximum of both infected humans and infected vectors in forest areas. The infections reach their maximum and their endemic steady states slightly earlier as the movement of susceptible humans increases.

Figure 8 illustrates the effect of varying the proportion of infected humans moving to forest area κ_2 and fixing all other parameters. It shows that increasing the proportion of infected humans moving to forest areas has the effect of increasing the number of infected vectors in forest areas and the number of infected humans. The time it takes to reach the maximum number of infections remains the same for the vector population and it becomes slightly earlier for the human population as κ_2 increases.

Note that when $\kappa_2 = 0$ the number of infected mosquitoes in the forest area reaches zero, which means that the disease will disappear from the forest since the model assumes that infected humans are the only source of infection for the vector population in the forest. However, there have been some reports of ZIKV being found in non-human primates, raising the possibility that they could act as reservoirs [30,31]. These sources of infection will be considered in future works.

V. Conclusion

A mathematical model of ZIKV disease including human movement and three transmission routes, namely,

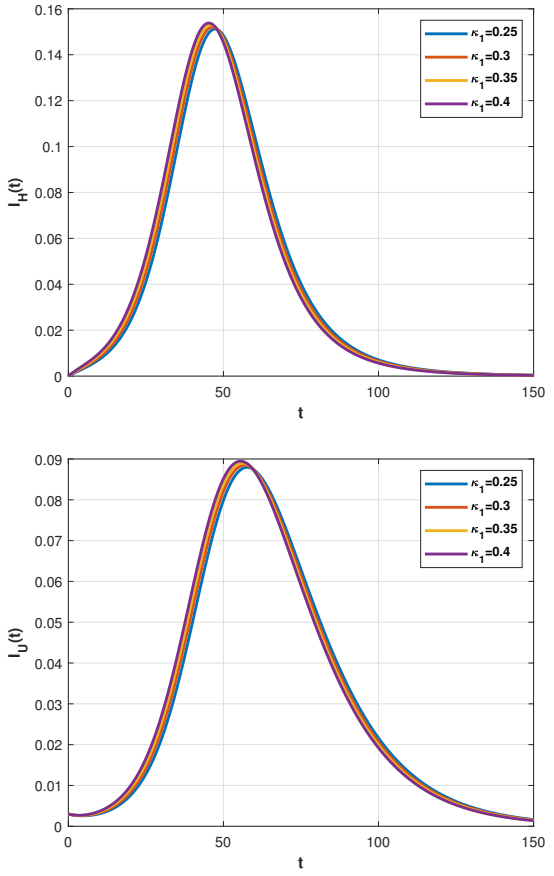

Fig. 7: Number of infected populations for different values of κ_1, where other parameters are fixed $\kappa_2 = 0.2$, $\alpha_1 = 2$, $\alpha_2 = 3$, $\varepsilon_1 = 0.67$, $\varepsilon_2 = 0.06$, $\varepsilon_3 = 0.06$, $\mu_H = 0.00004$, $\mu_V = 0.07$, $\mu_U = 0.07$, $\beta_1 = 0.35$, $\beta_2 = 0.9$, $\lambda = 0.235$, $\gamma = 0.16$, $\theta_1 = 0.33$, $\theta_2 = 0.3$.

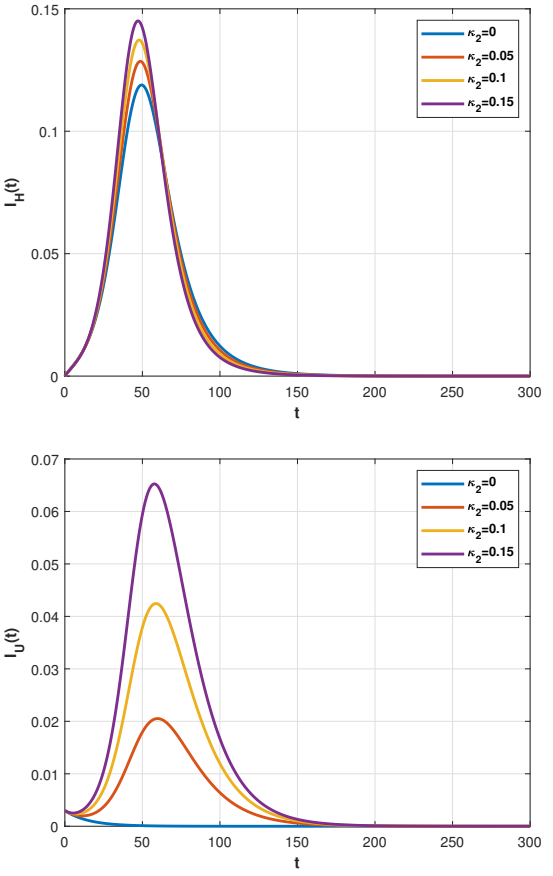

Fig. 8: Number of infected populations for different values of κ_2, where other parameters are fixed $\kappa_1 = 0.3$, $\alpha_1 = 2$, $\alpha_2 = 3$, $\varepsilon_1 = 0.67$, $\varepsilon_2 = 0.06$, $\varepsilon_3 = 0.06$, $\mu_H = 0.00004$, $\mu_V = 0.07$, $\mu_U = 0.07$, $\beta_1 = 0.35$, $\beta_2 = 0.9$, $\lambda = 0.235$, $\gamma = 0.16$, $\theta_1 = 0.33$, $\theta_2 = 0.3$.

human-to-human transmission, vector transmission, and vertical transmission has been proposed. The model has been analyzed and studied to investigate the role of human movement from rural areas to forest areas on the spread of ZIKV. The positivity of the solution and the boundedness of the invariant region were discussed.

The basic reproduction number R_0 was computed and expressed in terms of reproduction numbers related to the interactions between humans R_{HH}, between human and vector in rural area R_{HV} and between human and vector in forest area R_{HU}. It was found that the threshold of the disease which occurs at $R_0 = 1$ is equivalent to $R_{HH} + R_{HV} + R_{HU} = 1$ and hence all transmission routes need to be controlled to reduce the spread of the disease.

Sensitivity analysis of R_0 was carried out and it showed that R_0 is sensitive to almost all model parameters either positively or negatively, except the parameters κ_1 and κ_2, representing the proportions of susceptible and infected humans moving to forest areas, respectively, where their signs of sensitivity indices were found to depend on the biting rates when fixing all other parameters. This dependence has been calculated and illustrated graphically.

It has been found that the indices are both positive when the forest mosquito's biting rate is high and the rural mosquito's biting rate is small up to a certain limit. However, this positive effect on R_0 was found to be very small, with κ_2 having a higher effect than κ_1. The most positive influential parameters are the biting rate

of rural and forest mosquitoes on humans, while the recovery rate of humans has the most negative impact.

Then, the local and global stability of the disease-free equilibrium was derived whenever R_0 is less than unity. Furthermore, the system was shown to possess a unique endemic equilibrium under certain conditions, and it is locally asymptotically stable when R_0 is greater than unity since the direction of the bifurcation was found to be forward. The bifurcation analysis was presented both analytically and graphically. Finally, numerical simulations were presented to demonstrate the obtained theoretical results. They confirmed that the human movement from rural areas to forests has a small effect on increasing the infected human and vector populations, with the movement of infected humans having a higher effect than the movement of susceptible humans.

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