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DETERMINISTIC MODEL OF ZIKA-VIRUS WITH CARRIER MOTHER AND RESERVOIRS: A MATHEMATICAL ANALYSIS APPROACH

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ABSTRACT

In this paper, a mathematical model for the Zika virus is suggested to investigate the transmission dynamics of infection based on humans, pregnant carrier mother, infected children and the reservoir (primates) in three connected populations. Vertical and direct transmissions from all people to primates are considered in the proposed model. The Zika virus then spreads from this reservoir of infection via the nonhuman primate population (infected mosquitoes) to other entities. This virus can be passed on to the human population through an infected mosquito. Therefore, the new model with ten compartmental models has been normalized as follows: The normalized model is analyzed in depth to explore linkages between mosquitoes, humans, and primates on the dynamics of Zika-Virus transmission. The mathematical analysis comprises positivity and boundedness of solutions, determination of the basic reproduction number R_0 via next-generation matrix approach, existence and stability of all equilibria as well as sensitivity analysis. Local and Global Stability of the Disease-free Equilibrium. Finally, numerical simulations are performed to verify the analytical results obtained and exhibit the contribution of different model parameters on disease transmission dynamics. The results prove that the interaction of forest mosquitoes with primates has a significant effect on human-Zika-Virus transmission dynamics among the susceptible population due to transitions to forested areas. Moreover, the findings suggest that the transmission probabilities and biting rates of mosquitoes on humans and primates are major parameters in transmitting the disease.

Keywords: Mathematical model, Zika-Virus, Population, Basic reproduction number, Stability analysis

INTRODUCTION

The Zika virus has recently grown to be a serious threat to human society. It is a disease carried by vectors that is contracted by mosquito-borne flavin infection. 1954 saw the first identification of this infection distinct from rhesus monkeys in the Zika forest in Uganda and Nigeria. It wasn't until 2007 that the disease began to spread throughout the human population (Olawoyin & Kribs, 2018). The greatest Zika virus outbreak happened in Polynesia, France, in 2013–2014 (Biswas et al., 2020). Saiz et al., (2016), put together a report on the largest Zika outbreak in India that was made public in 2018. Zika infection is still a threat in some of the world even though it has decreased in the Americas. The infection is dispersed by mosquito species. Furthermore, several transmission pathways have been identified (e.g. both vertically sexually). Typically, a Zika infection results in a mild illness, that is mild symptoms like fever, headache, rash, arthralgia, and conjunctivitis are what define it. Nonetheless, alarming information regarding the potential correlation between the Zika Virus (ZIKV) and neonatal mutations (microcephaly) and Guillian-Barre syndrome (GBS) is being provided by a few regions that have recently been affected by the infection. As a result, on February 1st, 2016, the WHO declared a Public Health Emergency of
International Concern, emphasizing the need to intensify efforts to reduce ZIKV infection, particularly in women who are pregnant or who are of childbearing age (Saiz et al., 2016). It will be necessary to move forward with center and speculation in order to advance Zika vaccination. Pregnant women's counteraction efforts include avoiding areas where Zika is actively spreading, avoiding mosquito bites if they live in or are visiting these areas, and getting protection against sexual transmission until a Zika antibody becomes available (Rasmussen & Jamieson, 2020). In summary, researchers from various fields are working towards apprehending the disease transmission dynamics. Mathematical models have shown to be useful in understanding the spread of infection for diseases. Though information regarding ZIKV carriers from animals is scant.

A few publications on ZIKV detection in nonhuman primates propose that they can function as reservoirs of the virus. Mathematical modeling of Zika virus transmission further suggest that there is a high probability for the establishment of sylvatic cycle in forest area of South America (Althouse et al., 2016). They add that, "On average, the simulation model suggests that there are higher chances of an increase in sylvatic cycle if a rapidly multiplying primate or other competent ZIKV host mammals were to be included." It seems, then, likely that the existence of a possible ZIKV sylvatic process 35 could be sustained by an ecosystem. Some reports on ZIKV identification in nonhuman primates raise the prospect that they could serve as reservoirs. The creation of a sylvatic Zika cycle in the forest of South America is highly likely, according to mathematical models of Zika virus transmission, as discussed in (Althouse *et al.*, 2016). This can be reinforced by a system of as rare as 6,000 primates and 10,000 mosquitoes. Also, throughout the 2011 ZIKV amplification in Kedougou, field researchers discovered that the virus was existing in all main land cover classes in the region but was discovered substantially more frequently in the forest than in other land cover types. ZIKV has been detected in two of the three monkey species present in Kedougou: African green monkeys and Patas monkeys. After that, virus was found over a large part of tropical Africa through monkey serosurveys and by virus isolation from monkeys, as well as many species of sylvatic Aedes. Scientists, therefore, expect

monkeys infected with an American-amplified sylvatic cycle of another flavivirus, Yellow Fever, to have a high enough viremia and exhibit clinical signs that can be picked up by mosquitoes. The virus endured restricted to Africa and Asia's tropical areas, contaminating monkeys, arboreal mosquitoes, and humans on infrequent occasions. Most primates that were ZIKV-positive in the wild or lookout studies are from the Old World. According to phylogenetic analysis, humans are more closely related to Old World primate class, predominantly chimps and orangutans. Hence, the absolute danger often surges in diseases that can be spread among strictly related species. They also revealed that the ZIKV genome sequence in monkeys was indistinguishable to the ZIKV circulating in humans in South America. Moreover, scientists like Buechler *et al.,* (2017) studied which ZIKV commonalities our team could find in wild African monkeys, how it has emerged and spread as well if there is any evidence for actual or when exposure to human illnesses. What these findings suggest, though, is that as many as 16% of the nonhuman primate groups they sampled may have been hit with ZIKV at one point or another.

Best et al., (2017), in their work also predicted that study of ZIKV in Non-Human Primates (NHP) models can give us understanding about the viral dynamics and will be as useful tool for testing antiviral drugs and vaccines. All the key features of human Zika infection that were identified in NHP, including a rapid control of acute viremia, early invasion of the central nervous system, and persistent viral excretion and fetal pathogenesis in gravid animals have also been delineated. Furthermore, in their study also Al-Maqrashi et al., (2021), a mathematical model of humans' movement between rural human communities and adjacent forests was established and analyzed to highlight the impact of human activity on disease transmission among human and vector populations of ZIKV. Consequently, according to the distribution of mosquito species, the vector compartment has been divided into areas situated in rural areas and areas close to forest zones. The authors continued their arguments to an infected person with only general flu-like symptoms carrying the virus from rural areas to adjacent forests in searches of food or employment. Unfavorable human interaction has also been taken into account as in the case of susceptible human mobility in the transmission of ZIKV. It is therefore clear that human penetration into forests from the rural sectors has negligible influence in raising the infected human and vector densities. Some authors argue that recovered humans are subjected to temporary immunity as it was confirmed that antibody coupled with COVID-19 may decrease in adults (Biswas et al., 2020). It is assumed that susceptible mosquitoes, Larva mosquitoes, and the susceptible humans get themselves infected through ingestion of the blood paroused by the bites of female infected mosquitoes with constant total vector populations. Mosquitoes are not biomodeled for detection of ZIKV because the common species of mosquitoes that transmits the virus has a short life span of 414 days (Biswas et al., 2020). These are presumed to continue having the disease to the end of their live span. The literature review supported by Althouse et al., (2016), revealed that various types of leaders adopted different measures towards their security team. As has been observed in monkeys, when infection develops, they show clinical symptoms plenty enough to pass the virus to the mosquito vectors (Bueno et al., 2016). Monkeys (primates) has immune response towards the virus and recovers at a fixed level (Althouse et al., 2016) and cannot be infected again when rechallenged (Dudley et al., 2016).

Therefore, the present paper expands from Al-Maqrashi et al. (2021) and offers a mathematical model that incorporates three demographic categories, specifically human (adults, carrier pregnant mother and infected children), mosquito, and monkeys. Thus, three approaches to the means of transmission are identified: direct, on the level of departments of the same hierarchical level, and on the level of different hierarchies. The mobility of susceptible humans, and pregnant women to areas prone to Aedes mosquitoes connecting the forests with monkeys to the zika-virus transmission to the rural areas that may have vulnerable residents shall be evaluated in the proposed model. This paper is organized as follows: the general description of the used model is provided in Section 2. This section provides a detailed description of the case analysis of the proposed model. The following are the features of Mathematical analysis: Solution normalization, positivity and boundedness of the solution, Sensitivity analysis, and the stability of both local and global Disease-free equilibrium states. Simulation of the model as described in the preceding section is carried out in section 4, and an analysis of the influence of variation of some model parameters on the transmission dynamics of the disease is provided. In conclusion, the discussion of all the findings will be made in Section 5.

MATERIALS AND METHODS

Model Description and Formulation

This section suggests a model regarding human (Adults, Carrier Mother and Infected Children) mosquito, and primates (Monkey) interactions for Zika virus transmission. Examples of the populations and the interactions between them are given below in Fig 1. The total human population is assumed to remain constant and is classified into five compartments: vulnerable, contagious, the pregnant ladies, affected kids, and the restored. We assume that nonhuman primate – organisms' reservoir, are monkeys and divided into three together with having the overall population of a constant value. The four species dwell specifically in the forests and can be infected only from the female Aedes mosquitoes.

Model Assumptions

i. Natural mortality rate of both human and vectors: This lapse indicates the natural death and survival rates of both the infected human, primate population and the vectors.
ii. Identical recruitment rates of both susceptible human and vector populations are recruited at the same rate.
iii. It directly affects humans in the sense that, persons who are vulnerable can become infected with ZIKV through a bite from a female mosquito. ZIKV also directly affects people through vector transmission.
iv. Direct transmission through sexual transmission, or transfer through blood transfusion of infected Blood as well as through vertical transmission in which the mother passes on the virus to her baby during birth.
v. We estimate the proportion spending vulnerable advancement to the pregnant women class, part of newborns to Infected children bearing Zika. There are studies to support that this fraction is approximately 2/3 (Bonaldo et al., 2020).
vi. A fraction K of the infected children progresses to the infected individual (adult) class.
vii. Thangamani *et al.,* (2016) claim that the zika virus transverses in the mosquito vector and is the entry way that takes the virus through the colder months.
viii. The insecticide sprays and draining the water can prevent vectors.

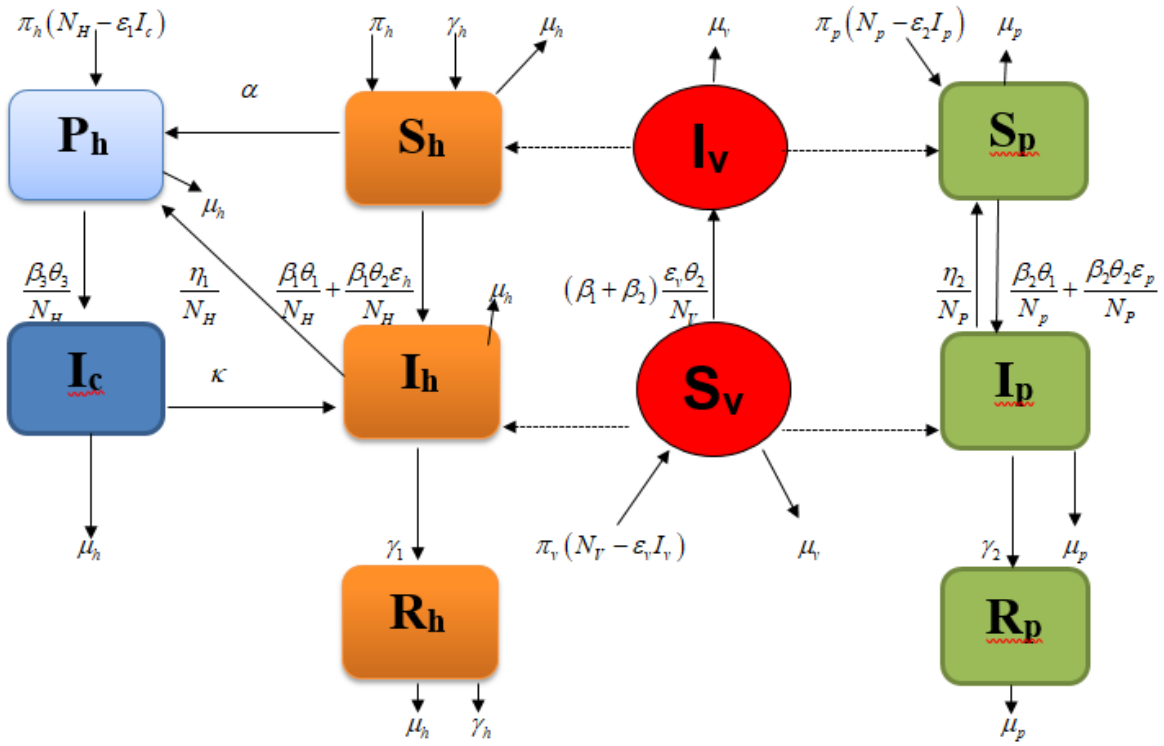

Figure 1: Schematic representation of the relationship between human, vectors and primate populations

The above in Fig 1 is the projected model given by the resulting set of equations:

$$
\left.\begin{aligned}
\frac{dS_h}{dt} &= \pi_h+\gamma_h R_h-\beta_1\theta_1 I_v\frac{S_h}{N_H}-\beta_1\theta_2\varepsilon_h I_h\frac{S_h}{N_H}-(\alpha+\mu_h)S_h\\
\frac{dI_h}{dt} &= \kappa I_c+\beta_1\theta_1 I_v\frac{S_h}{N_H}+\beta_1\theta_2\varepsilon_h I_h\frac{S_h}{N_H}-\eta_1 P_h\frac{I_h}{N_H}-(\gamma_1+\mu_h)I_h\\
\frac{dR_h}{dt} &= \gamma_1 I_h-(\gamma_h+\mu_h)R_h\\
\frac{dP_h}{dt} &= \pi_h(N_H-\varepsilon_1 I_c)+\alpha S_h-\beta_3\theta_3 I_c\frac{P_h}{N_H}+\eta_1 P_h\frac{I_h}{N_H}-\mu_h P_h\\
\frac{dI_c}{dt} &= \pi_h\varepsilon_1 I_c+\beta_3\theta_3 I_c\frac{P_h}{N_H}-(\kappa+\mu_h)I_c\\
\frac{dS_v}{dt} &= \pi_v(N_V-\varepsilon_v I_v)-(\beta_1+\beta_2)\theta_2\varepsilon_v S_v\frac{I_v}{N_V}-\mu_v S_v\\
\frac{dI_v}{dt} &= \pi_v\varepsilon_v I_v+(\beta_1+\beta_2)\theta_2\varepsilon_v S_v\frac{I_v}{N_V}-\mu_v I_v\\
\frac{dS_p}{dt} &= \pi_p(N_p-\varepsilon_2 I_p)-\beta_2\theta_1 I_v\frac{S_p}{N_P}-\beta_2\theta_2 I_p\frac{S_p}{N_P}+\eta_2 S_p\frac{I_p}{N_P}-\mu_p S_p\\
\frac{dI_p}{dt} &= \pi_p\varepsilon_2 I_p+\beta_2\theta_1 I_v\frac{S_p}{N_P}+\beta_2\theta_2 I_p\frac{S_p}{N_P}-\eta_2 S_p\frac{I_p}{N_P}-(\gamma_2+\mu_p)I_p\\
\frac{dR_p}{dt} &= \gamma_2 I_p-\mu_p R_p
\end{aligned}\right\} \qquad (1)
$$

subjected to non-negative initial conditions

$$(S_h(0)\geq 0, P_h(0)\geq 0, I_c(0)\geq 0, I_h(0)\geq 0, R_h(0)\geq 0, S_v(0)\geq 0, I_v(0)\geq 0, S_p(0)\geq 0, I_p(0)\geq 0, R_p(0)\geq 0)^T.$$

The table below provides the model (1)'s parameter values.

Table 1: Lists the parameters and variables that were employed, along with their daily values.

Variables	Descriptions	Variables	Descriptions
S_h	Susceptible Humans (adult)	S_v	Susceptible vectors (mosquitoes)
I_h	Infected Humans (adult)	I_v	Infected vectors (mosquitoes)
R_h	Recovered Human (adult)	S_p	Susceptible Primates
P_h	Pregnant Mothers	I_p	Infected Primates
I_c	Infected Children	R_p	Recovered Primates

Symbol	Description	Value	Source
π_h and π_p	The corresponding birth rates for humans and primates.	0.0461 & 0.752	Fitted
μ_h, μ_v and μ_p	The natural mortality rates of humans, vectors, and primates respectively	$\frac{1}{60*365}$, $\frac{1}{14}, \frac{1}{14}$	Fitted, (Bonyah & Okosun, 2016)
β_1, β_2 and β_3	The biting rate of mosquitoes on humans, primates, and pregnant mothers respectively	[0.33-1], [0.3-0.9], and [0.33-1]	(Olawoyin & Kribs, 2018), (Althouse *et al.*, 2016), Fitted
θ_1	Probability of infection transmission from infectious mosquitoes	[0.10-0.75]	(Maxian *et al.*, 2017)
θ_2	Probability of infection transmission to mosquito	[0.30-0.75]	(Maxian *et al.*, 2017)
θ_3	Probability of infection transmission from Mother to Infant	[0.5-0.86]	Fitted
η_1	Direct (sexual) transmission rate between humans	0.005	Fitted
η_2	Direct (sexual) transmission rate between primates	0.05	Fitted
γ_1, γ_2	Recovery rates of humans and primates respectively	0.1667, 0.2	(Suparit *et al.*, 2018), (Althouse *et al.*, 2014)
γ_h	rate of waning of immunity for humans	0.05	Fitted
ε_1 and ε_2	Probability of vertical transmission in humans and primates respectively	0.67, 0.06	(Bonaldo *et al.*, 2020), (Lai *et al.*, 2020)
ε_h	Rate of transmission from susceptible human to infected human	0.06	(Lai *et al.*, 2020)
ε_v	Rate of transmission from susceptible mosquito to infected mosquito	0.06	(Lai *et al.*, 2020)
π_v	Rate of hatching larva	0.275	Fitted
α	Progression rate of a pregnant mother	0.07143	Fitted
K	Fraction of infected children to infected human	0.3	Fitted

In this section, we normalize the proposed model (1). The normalized form of the model is (3). Next, two crucial topics, the positivity of solutions and invariant set, follow, and then the basic reproduction number and sensitivity analysis are introduced. The Equilibrium points, their local stabilities and Numerical Analysis are also discussed.

Model Normalization

From the proposed model (1), let

Given

$$S_H + P_H + I_C + I_H + R_H = 1, \quad S_V + I_V = 1, \quad S_P + I_P + R_P = 1 \tag{2}$$

Hence, the normalized model is prearranged by:

$$\left.\begin{aligned}
S_H' &= \pi_h + \gamma_h R_h - \beta_1\theta_1 I_v S_H - \beta_1\theta_2\varepsilon_h I_h S_H - (\alpha+\mu_h)S_H \\
I_H' &= \kappa I_c + \beta_1\theta_1 I_V S_H + \beta_1\theta_2\varepsilon_h I_H S_H - \eta_1 P_H I_H - (\gamma_1+\mu_h)I_H \\
R_H' &= \gamma_1 I_H - (\gamma_h+\mu_h)R_H \\
P_H' &= \pi_h(N_H - \varepsilon_1 I_C) + \alpha S_H + \eta_1 P_H I_H - \beta_3\theta_3 I_C P_H - \mu_h P_H \\
I_C' &= \pi_h\varepsilon_1 I_C + \beta_3\theta_3 I_C P_H - (\kappa+\mu_h)I_C \\
S_V' &= \pi_v(N_V - \varepsilon_v I_V) - (\beta_1+\beta_2)\theta_2\varepsilon_v S_V I_V - \mu_v S_V \\
I_V' &= \pi_v\varepsilon_v I_V + (\beta_1+\beta_2)\theta_2\varepsilon_v S_V I_V - \mu_v I_V \\
S_P' &= \pi_p(N_P - \varepsilon_2 I_P) - \beta_2\theta_1 I_V S_P - \beta_2\theta_2 I_P S_P + \eta_2 S_P I_P - \mu_p S_P \\
I_P' &= \pi_p\varepsilon_2 I_P + \beta_2\theta_1 I_V S_P + \beta_2\theta_2 I_P S_P - \eta_2 S_P I_P - (\gamma_2+\mu_p)I_P \\
R_P' &= \gamma_2 I_P - \mu_p R_P
\end{aligned}\right\} \quad (3)$$

where $X(0) = (S_H(0), P_H(0), I_C(0), I_H(0), R_H(0), S_V(0), I_V(0), S_P(0), I_P(0), R_P(0) \geq 0)^T$.

Positivity of Solutions and Positively Invariant Set

The non-negativity and boundedness of the state variables are established in the theorem below:

Theorem 1 *The solution*

$X(t) = (S_H(t), P_H(t), I_C(t), I_H(t), R_H(t), S_V(t), I_V(t), S_P(t), I_P(t), R_P(t))^T$ of the system (3) with non-negative initial condition $X(0)$ remains positive for all time t > 0, in a positively invariant closed set

$$\Omega = \left\{ X(t) \in R_+^{10} : 0 \leq S_H(t), P_H(t), I_C(t), I_H(t), R_H(t), S_V(t), I_V(t), S_P(t), I_P(t), R_P(t) \leq 1 \right\} \quad (4)$$

proof

Assume that system (3) has a non-negative initial condition $X(0)$. Let $\tau = sup\{t > 0; X(t) > 0\}$. Then, S_H' can be written as

$$S_H' = \pi_h + \gamma_h R_h - \beta_1\theta_1 I_v S_H - \beta_1\theta_2\varepsilon_h I_h S_H - (\alpha+\mu_h)S_H \quad (5)$$

where, $g_1 = \beta_1\theta_1 I_v + \beta_1\theta_2\varepsilon_h I_h$. It follows that

$$S_H' = \pi_h + \gamma_h R_h - (g_1 + \alpha + \mu_h)S_H \quad (6)$$

$$S_H' > -(\beta_1\theta_1 I_v + \beta_1\theta_2 I_h + (\alpha+\mu_h))S_H. \quad (7)$$

Integrating both sides over (0, *t*), we have;

$$S_H(t) > S_H(0) + e^{-(\beta_1\theta_1 I_v + \beta_1\theta_2 I_h + (\alpha+\mu_h))t}. \quad (8)$$

Also, $S_H(t)$ is positive since $S_H(0) \geq 0$. Similar calculations can be done for I_H', I_C', P_H' and R_H', we get

$$\left.\begin{aligned}
I_H(t) &\geq I_H(0) + e^{-[\eta_1 P_H + (\gamma_1+\mu_h)]t} > 0, \\
I_C(t) &\geq I_C(0) + e^{[\pi_h\varepsilon_1 - (\kappa+\mu_h)]t} > 0, \\
P_H(t) &\geq P_H(0) + e^{[\lambda_1 I_H - \mu_h]t} > 0, \\
&\textit{and} \\
R_H(t) &\geq R_H(0) + e^{[-(\gamma_h+\mu_h)]t} > 0,
\end{aligned}\right\} \quad (9)$$

subsequently initial conditions are non-negative. Likewise, one can confirm that the remaining mechanisms of $X(t)$ are positive at t. Using continuity of solution and $X(0) \geq 0$, we observed that t cannot be supremum and hence, the solution will remain positive for all $t > 0$.

Now, adopt that $\psi(t) = (\psi_1(t), \psi_2(t), \psi_3(t))^T$ where,

$$\begin{bmatrix} \psi_1(t) \\ \psi_2(t) \\ \psi_3(t) \end{bmatrix} = \begin{bmatrix} S_H(t) + I_H(t) + I_C(t) + P_H(t) + R_H(t) \\ S_V(t) + I_V(t) \\ S_P(t) + I_P(t) + R_P(t) \end{bmatrix}, \quad (10)$$

then, we have

$$\psi'(t) = \begin{bmatrix} 2\pi_h - \mu_h\psi_1(t) \\ \pi_v - \mu_v\psi_2(t) \\ \pi_p - \mu_p\psi_3(t) \end{bmatrix}. \quad (11)$$

Thus, solving for each component, we have:

$$\begin{bmatrix}\psi_1(t)\\ \psi_2(t)\\ \psi_3(t)\end{bmatrix}=\begin{bmatrix}2-\left(2-\psi_1(0)\right)e^{-\mu_h t}\\ 1-\left(1-\psi_1(0)\right)e^{-\mu_h t}\\ 1-\left(1-\psi_1(0)\right)e^{-\mu_h t}\end{bmatrix}, \tag{12}$$

where $\psi_1(0)=S_H(0)+I_H(0)+P_H(0)+I_C(0)+R_H(0)$, $\psi_2(0)=S_V(0)+I_V(0)$, and $\psi_3(0)=S_P(0)+I_P(0)+R_P(0)$. Thus,

$\psi_i(t)\le 1$ if $\psi_i(0)\le 1$, i = 1, 2, 3.

Hence, the set Ω is a positively invariant set $0\le\psi(t)\le 1$. Moreover, if $\psi(0)>1$ then $\lim_{t\to\infty}\psi(t)=1$ and then the set Ω is a globally attractive set.

Equilibria

In this model, there are two sets of equilibrium points, that is, the Zika-virus-free equilibrium point and the Zika-virus present equilibrium point. Setting the right-hand side of equation (3) equal to zero.

The Zika-virus-free equilibrium is realized in the absence of disease

$$\left(S_h^0,P_h^0,I_h^0,I_c^0,R_h^0,S_v^0,I_v^0,S_p^0,P_h^0,I_h^0\right)=\left\{\frac{\pi_h}{A_1},\frac{\pi_h N_h}{\mu_h},0,0,0,\frac{\pi_v N_v}{\mu_v},0,\frac{\pi_p N_p}{\mu_p},0,0\right\} \tag{13}$$

The Zika-virus-present equilibrium is achieved in the presence of disease

$$\left.\begin{aligned}
S_h &= \frac{Z_7\eta_1^2+\left(\beta_3\left(\theta_3A_2\beta_3\right)\left(I_v\theta_1+\varepsilon_h\theta_2\right)\left(\pi-A_2\right)\beta_1-\theta_3A_1A_1^2\beta_3+\pi\right)\beta_3\theta_3-\beta_3Z_6\eta_1}{A_1\beta_3\theta_3},\\
I_h &= \left(\left(\pi\varepsilon_1-A_4\right)\eta_1+\beta_3\theta_3\left(\pi-A_2\right)\right)\left(I_v\theta_1+\varepsilon_h\theta_2\right)\beta_1-\left(\left(\pi\varepsilon_1-A_4\right)\eta_1-A_2\beta_3\theta_3\right)A_1, P_h=\frac{\pi\varepsilon_1+A_4}{\beta_3\theta_3},\\
I_c &= \frac{Z_1\eta_1^2+\left(\pi\varepsilon_1-A_4\right)\theta_3\left(Z_2\beta_1-\left(A_1-2\alpha\right)A_1A_2\right)\beta_3\eta_1+Z_3\beta_1+\left(A_1\mu_h\varepsilon_1+\alpha\beta_3\theta_3\right)\pi+A_1Z_4}{A_1A_4\beta_3\theta_3},\\
R_h &= \frac{\left(\left(Z_5\eta_1+\beta_3\theta_3\left(\left(I_v\theta_1+\varepsilon_h\theta_2\right)\pi-A_2\left(I_v\theta_1+\varepsilon_h\theta_2\right)\right)\right)\beta_1+\left(\left(\pi\varepsilon_1-A_4\right)\eta_1-A_2\beta_3\theta_3\right)A_1\right)\gamma_1}{A_3}
\end{aligned}\right\} \tag{14}$$

$$\left.\begin{aligned}
S_v &= \frac{\pi_v\varepsilon_v+\mu_v}{\left(\beta_1+\beta_2\right)\theta_2\varepsilon_v}, \qquad I_v=\frac{\left(N_v\left(\beta_1+\beta_2\right)\theta_2+\mu_v\right)\pi_v\varepsilon_v-\mu_v^2}{\mu_v\varepsilon_v\theta_2\left(\beta_1+\beta_2\right)},
\end{aligned}\right\} \tag{15}$$

$$\left.\begin{aligned}
S_p &= \pi_p\left(N_p\left(\eta_2+c_1-\beta_2\theta_2\right)+\varepsilon_2\mu_p\right)+\mu\left(-\eta_2-c_1+\beta_2\theta_2\right)-I_v\beta_2c_1\theta_1-N_p\pi_p^2\varepsilon_2\\
I_p &= \frac{I_v^*\theta_1\left(c_1I_v\theta_1+N_p\pi_p\theta_2-\mu\theta_2\right)\beta_2^2+\left(N_p\varepsilon_p\pi^2I_v\theta_1+Z_9\right)\beta_2+Z_{10}}{N_p\pi^2\eta_2\varepsilon_2+Z_{11}\beta_2+Z_{12}+\left(-c_1I_v\theta_1-N_p\pi_p\theta_2+\mu\theta_2\right)\beta_2^2}\\
R_p &= \frac{\gamma_2\left(\beta_2\mu\theta_2-\eta_2\mu\right)\left(\beta_2\theta_1Z_8I_v^*-\pi_pN_p+\mu Z_8\right)}{\left(\beta_2\theta_2\left(Z_8-N_p\pi^2\varepsilon_2+c_1N_p\pi_p\right)+\pi\varepsilon_2-\eta_2\left(\left(\beta_2\mu\theta_2-\eta_2\mu\right)+Z_8\right)\right)\mu_p}
\end{aligned}\right\} \tag{16}$$

where, $Z_1=\left(\pi\varepsilon_1-A_4\right)^2\left(A_1-\alpha\right)\left(\left(I_v\theta_1+\varepsilon_h\theta_2\right)\beta_1+A_1\right)$, $Z_2=\left(\left(A_1-\alpha\right)\pi-A_2\left(A_1-2\alpha\right)\right)\left(I_v\theta_1+\varepsilon_h\theta_2\right)$,

$Z_3=\beta_3^2\theta_3^2A_2\alpha\left(\pi-A_2\right)\left(I_v\theta_1+\varepsilon_h\theta_2\right)$, $Z_4=\pi N\beta_3\theta_3-\beta_3^2\theta_3^2A_2^2\alpha-A_4\mu_h$, $A_1=\alpha+\mu_h$, $A_2=\gamma_1+\mu_h$

$Z_5=\varepsilon_h\left(I_v\theta_1+\varepsilon_h\theta_2\right)\pi-A_4\left(I_v\theta_1+\varepsilon_h\theta_2\right)$, $Z_6=\left(\left(I_v\theta_1+\varepsilon_h\theta_2\right)\left(\pi-2A_2\right)\beta_1-2A_1A_2\right)\theta_3\left(\pi\varepsilon_1-A_4\right)$,

$Z_7=\left(\left(I_v\theta_1+\varepsilon_h\theta_2\right)\beta_1+A_1\right)\left(\pi\varepsilon_1-A_4\right)^2$, $A_3=\gamma_h+\mu_h$, $A_4=\kappa+\mu_h$, $A_5=\gamma_2+\mu_p$

$Z_8=\left(-I_v^*\beta_2c_1\theta_1-N_p\pi_p\beta_2\theta_2+N_p\pi_p\eta_2+\pi_p\mu_p\varepsilon_2-c_1\mu_p\right)-N_p\pi_p^2\varepsilon_2+N_p\pi_pc_1$

$Z_9=\left(\left(-I_v^*\varepsilon_2\theta_1+N_p\theta_2\right)\mu_p-N_pI_v^*\theta_1\left(c_1+\eta_2\right)\right)\pi_p-\mu_p\left(\mu_p\theta_2-2\left(c_1+\frac{\eta_2}{2}\right)I_v^*\theta_1\right)$

$Z_{10}=N_p\pi_p^2\mu_p\varepsilon_2+\left(-\mu_p^2\varepsilon_2-N_p\left(c_1+\eta_2\right)\mu_p+N_p\right)\pi_p+\mu_p^2\left(c_1+\eta_2\right)$

$Z_{11}=-N_p\pi_p^2\theta_2\varepsilon_2+\left(\mu_p\varepsilon_2+N_p\left(c_1+2\eta_2\right)\theta_2\pi_p-\theta_2\left(c_1+2\eta_2\right)\mu_p+I_v^*c_1\eta_2\theta_1\right)$

$Z_{12}=\left(-\eta_2\mu_p\varepsilon_2-\eta_2\left(c_1+\eta_2\right)N_p+\varepsilon_2\right)\pi_p+\mu_p\eta_2\left(c_1+\eta_2\right)$

The Basic Reproduction Number

The model has two equilibrium positions: disease-free equilibrium and endemic equilibrium. The disease-free equilibrium exists and is given by $E^0 = (S_H^0,\ 0,\ P_H^0,\ 0,\ 0,\ S_V^0,\ 0,\ S_P^0,\ 0,\ 0)$, where, $S_H^0 = P_H^0 = S_V^0 = S_P^0 = 1$. The threshold quantity R_0 is well-defined as the average number of secondary infections produced by one event in an entirely susceptible population. It is calculated using the Next Generation Method by Van den Driessche & Watmough, (2002).

The appearance of new infections in the compartments is:

$$F = \begin{bmatrix} \beta_1\theta_2 - \eta_1 & \beta_1\theta_1 & 0 \\ 0 & (\beta_1+\beta_2)\theta_2\varepsilon_v & 0 \\ 0 & \beta_2\theta_1 & \beta_2\theta_2 - \eta_2 \end{bmatrix} \tag{17}$$

and Movement in and out of the compartment

$$V = \begin{bmatrix} (\gamma_1 + \mu_h) & 0 & 0 \\ 0 & \mu_v - \pi_v\varepsilon_v & 0 \\ 0 & 0 & \mu_p - \pi_p\varepsilon_2 \end{bmatrix} \tag{18}$$

then, the next generation matrix FV^{-1} is

$$FV^{-1} = \begin{pmatrix} \frac{\beta_1\theta_2 - \eta_1}{\gamma_1 + \mu_h} & \frac{\beta_1\theta_1}{-\pi_v\varepsilon_v + \mu_v} & 0 \\ 0 & \frac{(\beta_1+\beta_2)\theta_2\varepsilon_v}{-\pi_v\varepsilon_v + \mu_v} & 0 \\ 0 & \frac{\beta_2\theta_1}{-\pi_v\varepsilon_v + \mu_v} & \frac{\beta_2\theta_2 - \eta_2}{-\pi_p\varepsilon_2 + \mu_p + \gamma_2} \end{pmatrix}. \tag{190}$$

The basic reproduction number R_0 is the spectral radius FV^{-1} given by the followings;

$$R_{0H} = \frac{\beta_1\theta_2 - \eta_1}{\gamma_1 + \mu_h},\quad R_{0V} = \frac{(\beta_1+\beta_2)\theta_2\varepsilon_v}{-\pi_v\varepsilon_v + \mu_v},\quad R_{0P} = \frac{\beta_2\theta_2 - \eta_2}{-\pi_p\varepsilon_2 + \mu_p + \gamma_2} \tag{20}$$

where R_{0H}, R_{0V}, R_{0P} are the basic reproduction numbers for humans, mosquitos and primates respectively.

Sensitivity Analysis of the Basic Reproduction Number

In this section, we have illustrated a brief of the sensitivity analysis of the basic reproduction number R_0. This enables the determination of the parameters that will give a rigorous shift in the threshold ratio R_0 of the model, this means that a change in a sensitive parameter will yield a great quantitative difference in R_0 and may bring about qualitative changes. These parameters should be considered worthy of attention by the management and the development of control activities. Here, the index of forward sensitivity analysis called the elasticity index (Rodrigues *et al.*, 2013) is applied and it is defined as the proportion between the absolute change of R_0 and the absolute change of the parameter corresponding to the relative change as the follow:

$$\Upsilon_{\ell}^{R_0} = \frac{\ell}{R_0} \times \frac{\partial R_0}{\partial \ell} \tag{21}$$

Since R_{0H}, R_{0V}, R_{0P} are the basic reproduction number for humans, mosquitos and primates respectively. The sensitivity analysis of R_0 to each of its parameters will be evaluated via the sensitivities of each R_{0H}, R_{0V}, R_{0P} such that

$$\Upsilon_{\ell_1}^{R_{0H}} = \frac{\ell_1}{R_{0H}} \times \frac{\partial R_{0H}}{\partial \ell_1},\quad \Upsilon_{\ell_2}^{R_{0V}} = \frac{\ell_2}{R_{0V}} \times \frac{\partial R_{0V}}{\partial \ell_2},\quad \Upsilon_{\ell_3}^{R_{0P}} = \frac{\ell_3}{R_{0P}} \times \frac{\partial R_{0P}}{\partial \ell_3} \tag{22}$$

where ℓ_1, ℓ_2, ℓ_3 denotes the parameter related to R_{0H}, R_{0V}, and R_{0P}, respectively. Via the obvious expression of the basic reproduction number R_0 per the baseline values of parameters enumerated in Table 1, the values of the sensitivity's indices are shown in Table 2.

Table 2. Sensitivity indices of R_{0H}, R_{0V}, and R_{0P}.

Parameter	Sign	$\Upsilon_{\ell_1}^{R_{0H}}$	Parameter	Sign	$\Upsilon_{\ell_2}^{R_{0V}}$	Parameter	Sign	$\Upsilon_{\ell_3}^{R_{0P}}$
β_1	+ve	1.0256	β_1	+ve	0.3448	β_2	+ve	1.1515
θ_2	+ve	1.0256	β_2	+ve	0.6552	θ_2	+ve	1.1515
η_1	-tve	0.0256	θ_2	+tve	1.0000	η_2	-tve	0.1515
γ_1	-tve	0.9997	ε_v	+tve	1.3004	π_p	-tve	2.5200
μ_h	-tve	0.0003	π_v	+tve	0.3004	ε_2	-tve	2.5200
			μ_v	-tve	1.3004	μ_p	+tve	0.4000
						γ_2	+tve	1.1200

The values of the sensitivity indices are discussed based on the magnitudes as well as its signs. As shown in the table, the sensitivity indices indicate that increasing in those parameters will lead to an increase in the epidemic's basic reproduction number R_0 or to its decrease in case of their decrease. On the other hand, it rises (drops) as the parameters with negative sensitivity indices decrease (increase). However, sensitivity indices indicate the overall level of significance concerning such factors that define the spread of the disease. In the human population β_1, θ_2 biting rate of humans by the mosquito and probability of transmission of infection to a mosquito are those parameters which carries the maximum positive effect on efficacy value R_{0H}. The most sensitive parameter for the given form of the basic reproduction number of the primates and vectors are β_2, θ_2. In addition, the results assert that θ_1, and θ_2 the probabilities of transmission of the virus from human to mosquito and vice versa are equally crucial as biting rates of the mosquito to both human and primate populace. Also, the short life span of mosquitoes will result in less numbers of people getting infected and therefore, decrease the reproduction rate R_0. Therefore, to mitigate the spread of the Zika virus, it is recommended to maintain the mosquito population while addressing the mosquito breeding areas near house in rural regions.

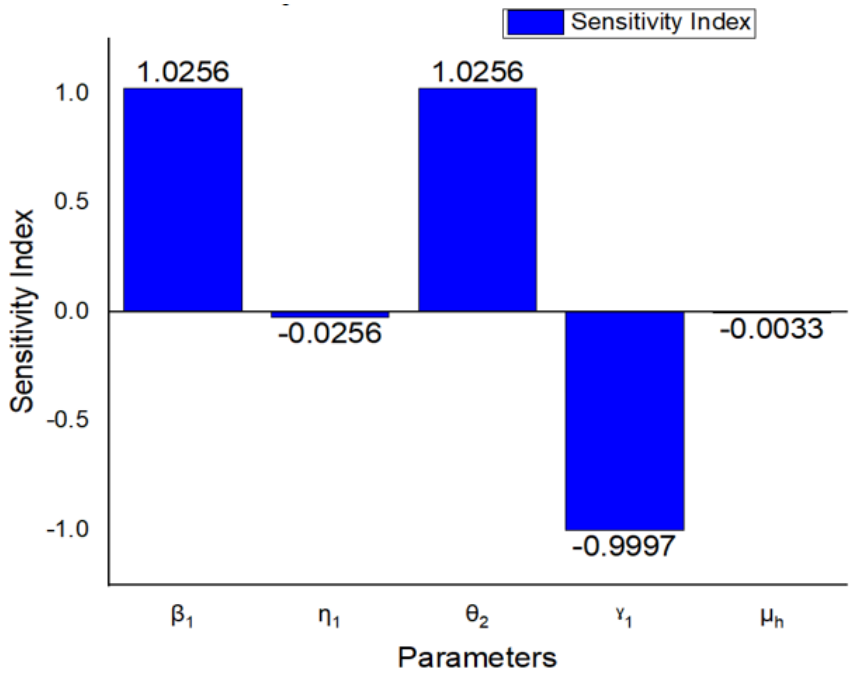

Figure 2: Sensitivity indices of R_0 (Human) concerning the model parameters.

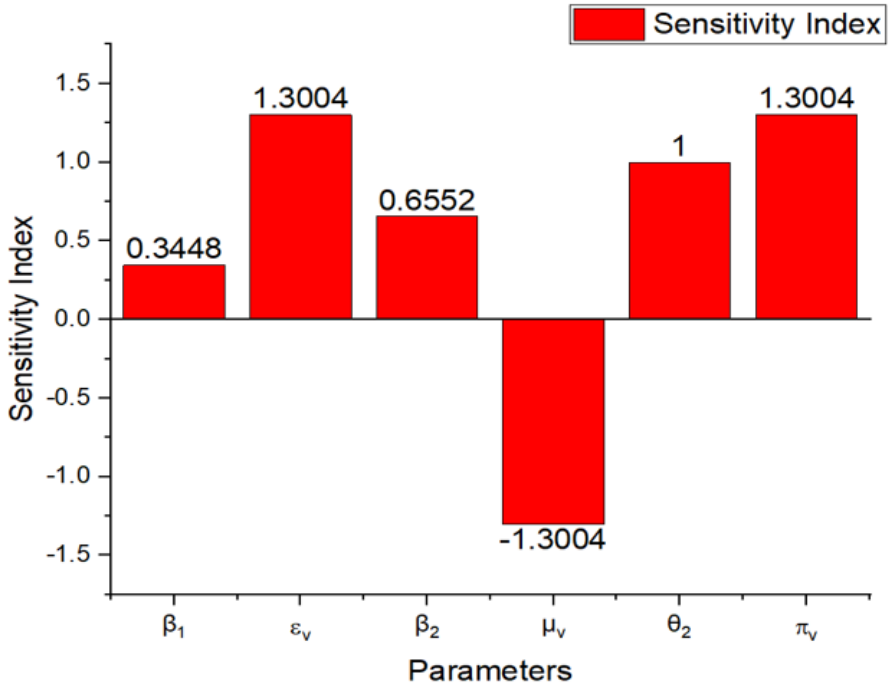

Figure 3: Sensitivity indices of R_0 (Vector) concerning the model parameters

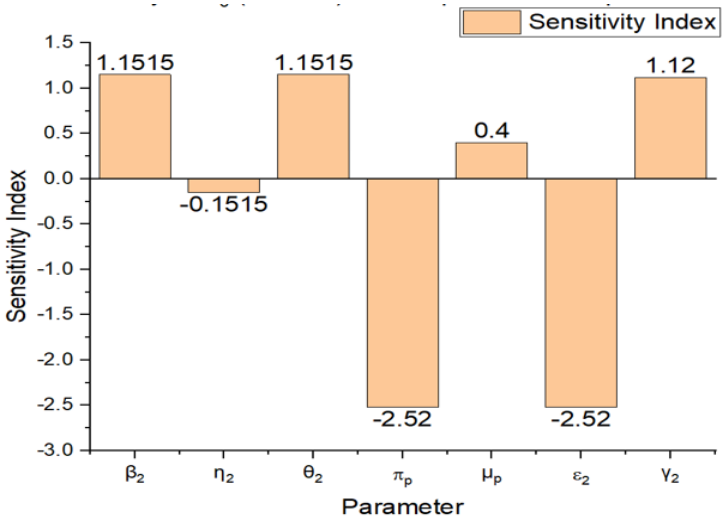

Figure 4: Sensitivity indices of R_0 (Primate) concerning the model parameters

Local Stability of the Disease-Free Equilibrium

Now, we discuss the local stability of the disease-free equilibrium E^0 by obtaining the eigenvalues of the linearized system of ODE (3). The product is given in the following theorem:

Theorem 2 *The Disease-Free Equilibrium (DFE, given by E^0, the model (3) is locally asymptotically stable if $R_0 < 1$. Otherwise, it is unstable.*

proof

Then, the linearized matrix of the system of ODE (3) at the disease-free equilibrium E^0 is given as

$$J=\begin{pmatrix} -Z_{11} & -\beta_1\theta_2\varepsilon_h S_H & \gamma_h & 0 & 0 & 0 & -\beta_1\theta_1 S_H & 0 & 0 & 0 \\ Z_{21} & -Z_{22} & 0 & -\eta_1 I_H & \kappa & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_1 & -Z_{33} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha & \eta_1 P_H & 0 & -Z_{44} & -Z_{45} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \beta_3\theta_3 I_C & -Z_{55} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -Z_{66} & -Z_{67} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & Z_{76} & -Z_{77} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\beta_2\theta_1 S_P & -Z_{88} & -Z_{89} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \beta_2\theta_1 S_P & Z_{98} & -Z_{99} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_2 & -Z_{100} \end{pmatrix}, \tag{23}$$

where,

$$Z_{11}=-\left(\beta_1\theta_1 I_v+\beta_1\theta_2\varepsilon_h I_h+\alpha+\mu_h\right),\quad Z_{22}=-\left(-\beta_1\theta_2\varepsilon_h S_H+\eta_1 P_H+\gamma_1+\mu_h\right),\quad Z_{21}=\beta_1\theta_1 I_V+\beta_1\theta_2\varepsilon_h I_H$$

$$Z_{33}=-\left(\gamma_h+\mu_h\right),\quad Z_{44}=-\left(-\eta_1 I_H+\beta_3\theta_3 I_C+\mu_h\right),\ Z_{45}=-\left(\pi_h\varepsilon_1+\beta_3\theta_3 P_H\right),$$

$$Z_{55}=-\left(-\pi_h\varepsilon_1-\beta_3\theta_3 P_H+\kappa+\mu_h\right),\ Z_{66}=-\left(\left(\beta_1+\beta_2\right)\theta_2\varepsilon_v I_V+\mu_v\right),\ Z_{67}=-\left(\pi_v\varepsilon_v+\left(\beta_1+\beta_2\right)\theta_2\varepsilon_v S_V\right),$$

$$Z_{77}=-\left(-\pi_v\varepsilon_v-\left(\beta_1+\beta_2\right)\theta_2\varepsilon_v S_V+\mu_v\right),\ Z_{76}=\left(\beta_1+\beta_2\right)\theta_2\varepsilon_v I_V,\ Z_{88}=-\left(\beta_2\theta_1 I_V+\beta_2\theta_2 I_P-\eta_2 I_P+\mu_p\right),$$

$$Z_{89}=-\left(\pi_p\varepsilon_2+\beta_2\theta_2 S_P-\eta_2 S_P\right),\ Z_{99}=-\left(-\pi_p\varepsilon_2-\beta_2\theta_2 S_P+\eta_2 S_P+\gamma_2+\mu_p\right),$$

$$Z_{98}=\beta_2\theta_1 I_V+\beta_2\theta_2 I_P-\eta_2 I_P,\quad Z_{100}=-\mu_p$$

$$J\left(E^0\right)=\begin{pmatrix} -(\alpha+\mu_h) & -\beta_1\theta_2\varepsilon_h S_H & \gamma_h & 0 & 0 & 0 & -\beta_1\theta_1 S_H & 0 & 0 & 0 \\ 0 & -Z_{22} & 0 & 0 & \kappa & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_1 & -Z_{33} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \alpha & \eta_1 P_H & 0 & -\mu_h & -Z_{45} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & -Z_{55} & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\mu_v & -Z_{67} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -Z_{77} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\beta_2\theta_1 S_P & -\mu_p & -Z_{89} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \beta_2\theta_1 S_P & 0 & -Z_{99} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_2 & -\mu_p \end{pmatrix}. \tag{24}$$

The above Jacobian has seven, negative eigenvalues, which are:

$$\lambda_1 = -\mu_v,\ \lambda_2 = -\mu_h,\ \lambda_3 = -A_3,\ \lambda_4 = -A_1,\ \lambda_5 = -\mu_p,\ \lambda_6 = -\mu_p,\ \lambda_7 = -\left(\frac{\beta_1\pi_h\theta_2\varepsilon_h + N_h\eta_1\pi_h + A_2\mu_h}{\mu_h}\right) \tag{25}$$

The remaining eigenvalues is given in the characteristic equation $P(\lambda) = 0$, which is given by

$$P(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3 \tag{26}$$

where

$$a_1 = \left(\left(\mu_v^2 + \left(-\pi_h\varepsilon_1 - \pi_v\varepsilon_v + A_4 + A_5\right)\mu_v - \pi_v\theta_2 N_v\left(\beta_1 + \beta_2\right)\right)\mu_p + \pi_p^2\varepsilon_2 N_p\mu_v\left(-\beta_2\theta_2 + \eta_2\right)\right)\mu_h - N_h\beta_3\mu_p\mu_v\pi_h\theta_3$$

$$a_2 = \left(\begin{array}{l}\left(\left(-\pi_h\varepsilon_1 + A_4 + A_5\right)\mu_p + \mu_v\varepsilon_2 N_p\left(-\beta_2\theta_2 + \eta_2\right)\right)\mu_v^2 + \left(\left(-\varepsilon_v\left(-\pi_h\varepsilon_1 + A_4 + A_5\right)\mu_v + A_5\left(-\pi_h\varepsilon_1 + A_4\right)\mu_p\right)\right)\mu_p \\ +\pi_p^2\varepsilon_2 N_p\left(-\beta_2\theta_2 + \eta_2\right)\left(-\pi_h\varepsilon_1 - \pi_v\varepsilon_v + A_4\right)\mu_v - \left(\beta_1 + \beta_2\right)\mu_v\theta_2\left(\begin{array}{l}\left(-\pi_h\varepsilon_1 + A_4 + A_5\right)\mu_p \\ +\pi_p^2\varepsilon_2 N_p\left(-\beta_2\theta_2 + \eta_2\right)\end{array}\right)N_v\end{array}\right)\mu_h$$

$$-\pi_h N_h\left(\mu_p\mu_v^2 + \left(\left(-\pi_v\varepsilon_v + A_5\right)\mu_p + \pi_p^2\varepsilon_2 N_p\left(-\beta_2\theta_2 + \eta_2\right)\right)\mu_v - \pi_v\theta_2 N_v\mu_p\left(\beta_1 + \beta_2\right)\right)\beta_3\theta_3$$

$$a_3 = \frac{\left(-N_p\beta_2\pi_p^2\theta_2\varepsilon_2 + N_p\eta_2\pi_p^2\varepsilon_2 + A_5\mu_p\right)\left(\theta_2\pi_v N_v\beta_1 + \theta_2\pi_v N_v\beta_2 + \pi_v\varepsilon_v\mu_v - \mu_v^2\right)\left(\theta_3\pi_h N_h\beta_3 + \pi_h\varepsilon_1\mu_h - A_4\mu_h\right)}{\mu_p\mu_v\mu_h}$$

Since all parameters are positive, we say that $a_1, a_2, a_3 > 0$ for $R_0 < 1$.

Thus, the necessary conditions for stability via the Routh Hurwitz stability criterion by Sambariya & Prasad, (2012) are satisfied. The sufficient conditions, namely, $H_1 = a_1 > 0$, and

$H_2 = \frac{a_1a_2 - a_3}{a_1} > 0$ for $a_1a_2 - a_3 > 0$, we have,

$$a_1a_2 - a_3 = \frac{1}{\mu_p\mu_v\mu_h}\left(-\pi_h\varepsilon_1 + A_4 + 1\right)\left(-N_p\beta_2\pi_p^2\theta_2\varepsilon_2 + \pi_p^2\varepsilon_2 N_p\eta_2 + \mu_p\left(A_5 + 1\right)\right)\mu_v^2$$

$$+\left(\begin{array}{l}\varepsilon_v\left(-\pi_h\varepsilon_1 + A_4 + 1\right)\left(-N_p\beta_2\pi_p^2\beta_2\varepsilon_2 + \pi_p^2\varepsilon_2 N_p\eta_2 + \mu_p\left(A_5 + 1\right)\right)\pi_v - \pi_p^2\beta_2\varepsilon_2 N_p\left(-\pi_h\varepsilon_1 + A_4 + 1\right)\theta_2 \\ +\pi_p^2 N_p\eta_2\left(-\pi_h\varepsilon_1 + A_4 + 1\right)\varepsilon_2 + \left(-\varepsilon_1\left(A_5 + 1\right)\pi_h + \left(A_5 + 1\right)A_4 + A_5\right)\mu_p\end{array}\right)\mu_v$$

$$-\left(\beta_1 + \beta_2\right)\mu_v\left(-\pi_h\varepsilon_1 + A_4 + 1\right)\theta_2 N_v\left(-N_p\beta_2\pi_p^2\theta_2\varepsilon_2 + \pi_p^2\varepsilon_2 N_p\eta_2 + \mu_p\left(A_5 + 1\right)\right)$$

$$+\left(-\mu_v^2 + \left(\pi_v\varepsilon_v - 1\right)\mu_v + \pi_v\theta_2 N_v\left(\beta_1 + \beta_2\right)\right)\pi_h N_h\left(-N_p\beta_2\pi_p^2\theta_2\varepsilon_2 + \pi_p^2\varepsilon_2 N_p\eta_2 + \mu_p\left(A_5 + 1\right)\right)\theta_3\beta_3 > 0$$

(after simplification) and $H_3 = a_3 > 0$.

Hence, we conclude that the eigenvalues of p(λ) = 0 have negative real parts, whenever $R_0 < 1$, which suggests that the Disease-Free Equilibrium (E^0) is locally asymptotically stable.

Global Stability of the Disease-Free Equilibrium

The global stability of the disease-free equilibrium (E^0) will guarantee that the disease is removed under all initial conditions. Hence, the following theorem:

Theorem 3 *The disease-free equilibrium, given by E^0 the model (3) is globally asymptotically stable if $R_0 < 1$.*

proof

Using the Castillo-Chavez theorem (Chavex *et al.*, 2002). Let $X(t)$ and $Y(t)$represent the compartments define the uninfected and infected classes of the system (3), respectively:

$$X(t) = \begin{pmatrix} S_h(t) \\ P_h(t) \\ R_h(t) \\ S_v(t) \\ S_p(t) \\ R_p(t) \end{pmatrix}, \qquad Y(t) = \begin{pmatrix} I_h(t) \\ I_c(t) \\ I_v(t) \\ I_p(t) \end{pmatrix} \tag{27}$$

Thus, system (3) can be written as

$$\frac{dX}{dt} = F(X,Y),\ \frac{dY}{dt} = G(X,Y),\ G(X,0) = 0 \tag{28}$$

where F and G are the conforming right-hand sides in the system (3). According to Castillo-Chavez theorem, to pledge the global asymptotic stability of the DFE (E^0), the following two conditions (H_1) and (H_2) must be satisfied:

$\hat{}\ (H_1)$ $E^0 = (1,1,0,1,1,0)^T$ is globally asymptotically stable $\frac{dX}{dt} = F(X,0)$.

$\hat{}\ (H_2)$ $\hat{G} \geq 0$, where $\hat{G}(X,Y) = AY - G(X,Y)$ and $A = D_Y - G\left(X^0,0\right)$ is an Metzler matrix $\forall (X,Y) \in \Omega$

For the first condition (H_1), we have,

$$\frac{dX}{dt} = F(X,0) = \begin{pmatrix} \pi_h - \mu_h S_h \\ \pi_h - \mu_h P_h \\ -\mu_h R_h \\ \pi_v - \mu_v S_v \\ \pi_p - \mu_p S_p \\ -\mu_p R_p \end{pmatrix}. \tag{29}$$

Assuming recruitment and death rates are equal, the behaviour of each compartment can be determined by solving the above system (27), hence, we have.

$$\begin{pmatrix} S_h(t) \\ P_h(t) \\ R_h(t) \\ S_v(t) \\ S_p(t) \\ R_p(t) \end{pmatrix} = \begin{pmatrix} 1 + S_h(0)e^{-\mu_h t} \\ 1 + P_h(0)e^{-\mu_h t} \\ R_h(0)e^{-\mu_h t} \\ 1 + S_v(0)e^{-\mu_v t} \\ 1 + S_p(0)e^{-\mu_p t} \\ R_p(0)e^{-\mu_p t} \end{pmatrix} \tag{30}$$

Now since $\lim_{t\to\infty} X^0$ as $t \to \infty$ That is, the first condition is satisfied. For the second condition, (H_2):

$$A = \begin{pmatrix} -\beta_1\theta_2\varepsilon_h - A_2 - \eta_1 & \kappa & \beta_1\theta_1 & 0 \\ 0 & \beta_3\theta_3 + \pi_h\varepsilon_1 - A_4 & 0 & 0 \\ 0 & 0 & \pi_v\varepsilon_v + (\beta_1 + \beta_2)\theta_2\varepsilon_v - \mu_v & 0 \\ 0 & 0 & \beta_2\theta_1 & \beta_2\theta_2 + \pi_p\varepsilon_2 - A_5 - \eta_2 \end{pmatrix}. \tag{31}$$

We have,

$$G(X,Y) = AY - G(X,Y) \tag{32}$$

$$G(X,Y) = \begin{pmatrix} (1-S_h)(I_v\theta_1 - I_h\theta_2\varepsilon_h)\beta_1 - I_h\eta_1(1-P_h) \\ \beta_3\theta_3 I_c(1-P_h) \\ I_v\theta_2\varepsilon_v(\beta_1+\beta_2)(1-S_v) \\ \left((I_p\theta_2 + I_v\theta_1)\beta_2 - I_p\eta_2\right)(1-S_p) \end{pmatrix} \tag{33}$$

It is obvious that since $G \geq 0 \ \forall (X,Y) \in \Omega$ where $0 \leq (S_h, P_h, S_v, S_p) \leq 1$ thus the proof is complete, hence E^0 is the globally asymptotically stable only if $R_0 < 1$.

Numerical Analysis

In this section, numerical simulations are performed in order to illustrate the theoretical investigation results obtained for the Zika-Virus model (3) as well as to investigate the impact of some model parameters which are involved in transmitting the disease to the human population. Since the system is a nonlinear model, we shall assume a baseline value of the parameters as summarized below in Table 1 and some suitable initial conditions.

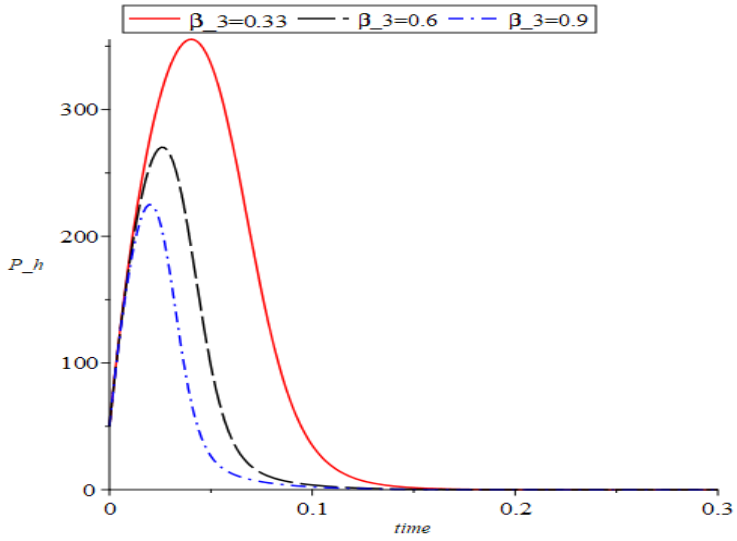

Figure 5: Graph showing dynamics β_3 with respect to Carrier Mother class

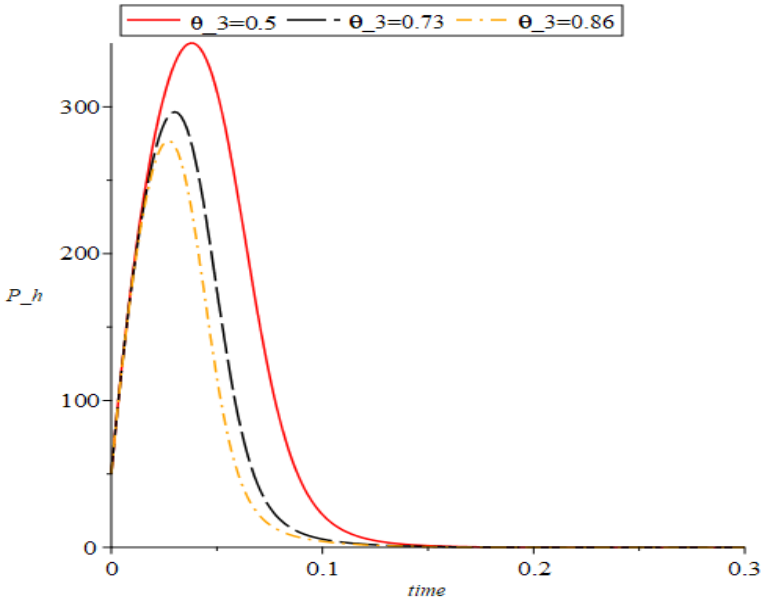

Figure 6: Graph showing dynamics θ_3 with respect to Carrier Mother class

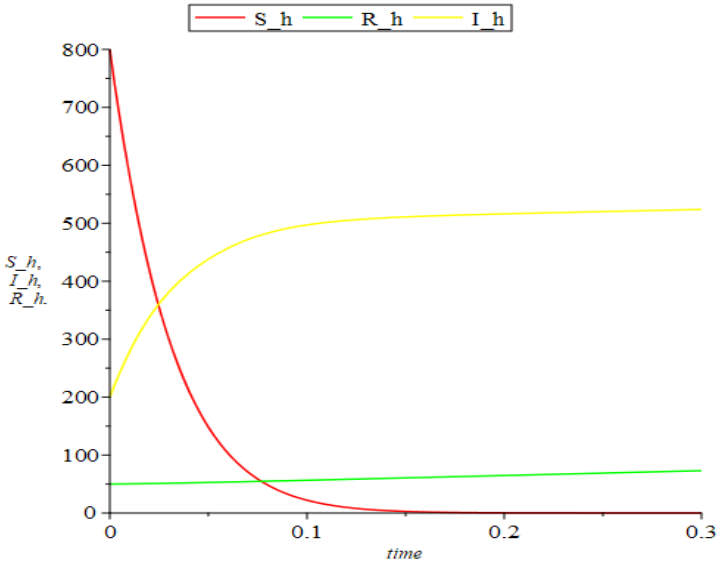

Figure 7: The dynamics of Susceptible Infected and Recovered human class.

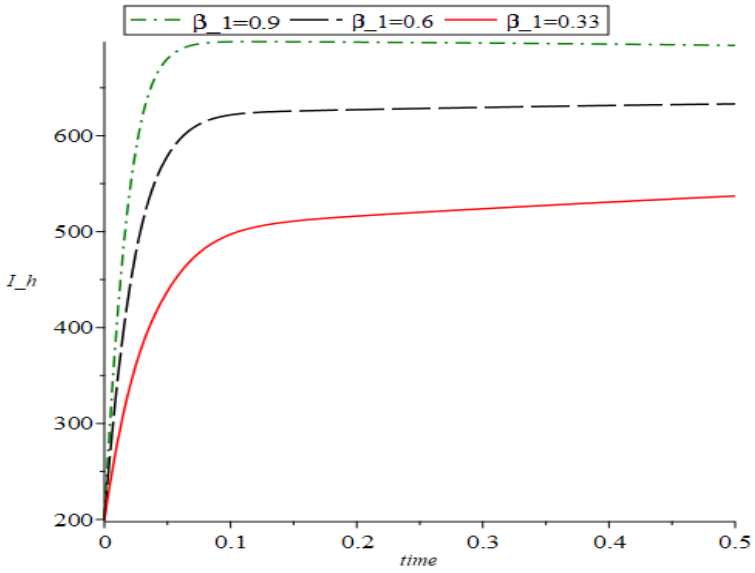

Figure 8: Graph showing the dynamics β_1 with respect to the Infected human class

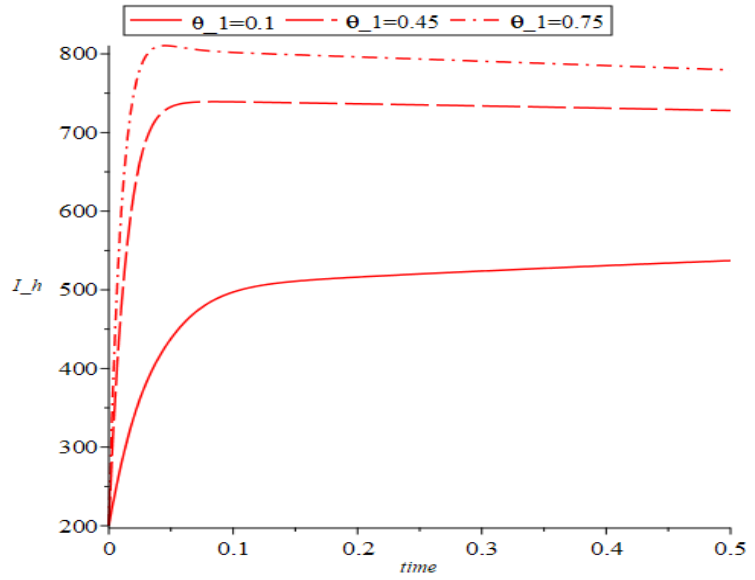

Figure 9: Graph showing the dynamics θ_1 with respect to the Infected human class

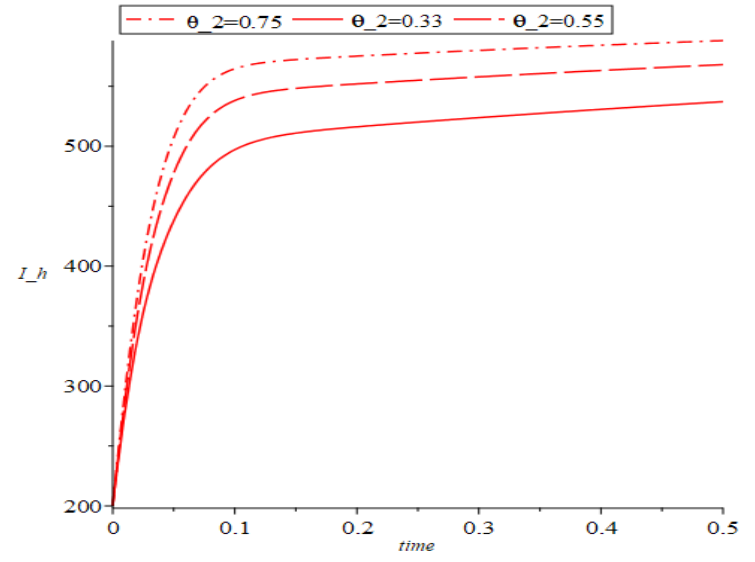

Figure 10: The dynamics θ_2 with respect to the Infected human class

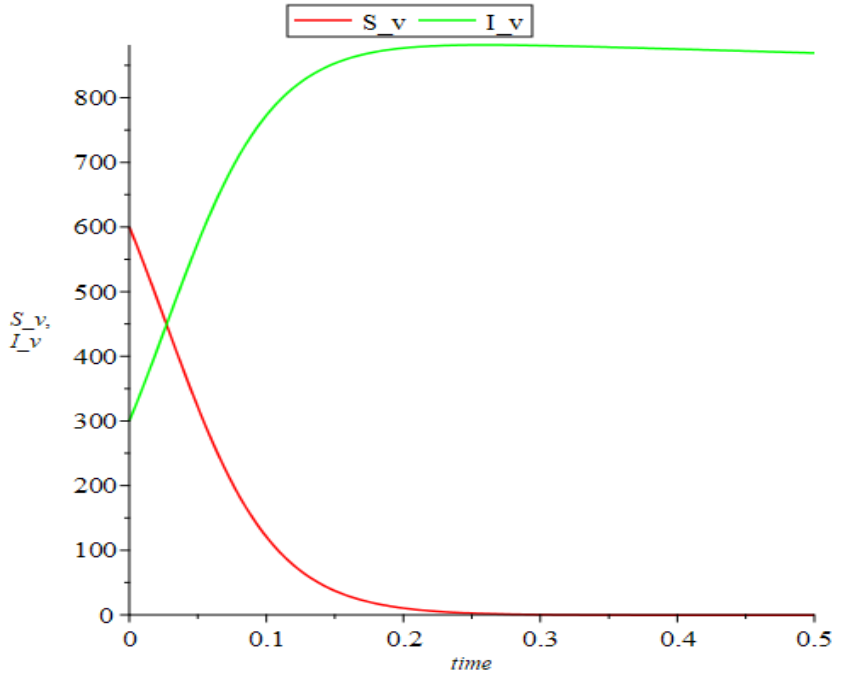

Figure 11: Dynamics of Susceptible and Infected vector class

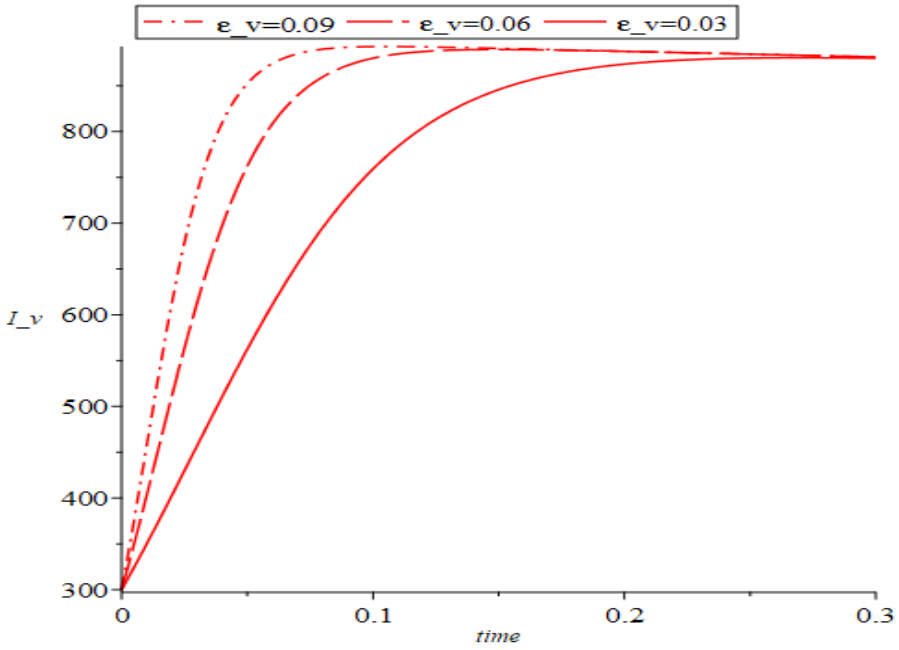

Figure 12: Graph showing the dynamics ε_v with respect to the Infected vector class

Finally, through graphical methods, we examine the behaviors of the model by portraying the solution of Pregnant women against β_3 and $\theta_3$respectively. Figures 5 gives the relationship between the number of mosquito bites and pregnant mothers depicting that as the former increases, the latter decreases. Similarly, figure 6 gives the relationship between mother to infant transmission rate and pregnant mothers indicating that as the former increases, the latter decreases. From the figure 7 depicting the SIR model of the population in the adult human class, it is evident that all populations are affected by disease when the basic reproduction number is greater than unity. Figures 8, 9 and 10 analyze how the measure Dynamics of infected humans depends on at some specified values of β_1, θ_1 and θ_2. Figure 8 depicts the impact of changing the values of the biting rate of rural mosquitoes on the level of human infection with this disease as the figure shows the correspondence between the increase in biting rates and the number of infected humans, Figures 8 & 9 also depicts that as the probabilities of transmission by the infectious mosquitoes and the probability of getting infected by the mosquitoes rises, more humans are infected. The last two figures include figure 11 where the relation between susceptible and infected vectors is depicted, and finally figure 12 that indicates if ε_v the transmission from susceptible mosquitoes to infected ones is increased, the number of infected humans would also rise. This poses a big risk as it increases chances of contact between the humans and the Zika Virus if people move closer to the forest areas. Thus,

in terms of controlling the spread of Zika-Virus within forests, the control strategies as discussed earlier should put measures like limiting human mobility from healthy areas to infected forest regions.

CONCLUSION

Human-to-mosquito transmission with carrier Mother and Infected Children and vertical transmission with monkey primate and mosquito has also been addressed mathematically with suitable parameters. There has also been consideration given to the transfer of the human being back to the natural setting of the primates, that is the forest area. This model was examined and comprehensively reviewed to identify the effects of Extracting and Moving Primates and Humans in the Forest on the Zika-virus. The properties of non-negativity and stability of the solution and the boundedness of the region containing the solution were examined. The basic reproduction number R_0 was estimated/quantified in three perspectives including human transmission R_{0H}, mosquitoes R_{0V} (Vector) and Monkey R_{0P}, (Primate). The proposed model was found to have two equilibria: basic reproduction number R_0; an outbreak phase in which the disease is produced by an individual not infected with the initial group; a disease-free equilibrium (DFE); and an endemic equilibrium (EE) characterizing an endemic area which occurs when $R_0 > 1$. Next, the study undertook an examination of the robustness of R_0 through a sensitivity analysis showing that some parameters are highly sensitive than all other model parameters either positively or negatively. Nonetheless, the most positive influential parameters are; β_1 and β_2 while the most negative influential parameter is μ_h. The sensitivity analysis also revealed that the parameters affecting the stability of the dynamic system include; the recovery rate of primates, the rate of transmission of the infection to the mosquitoes, and the rate of transmission of the disease among the primates. A stability of disease-free equilibrium, both local and global was postulated and proved. As illustrated before, the effect of the transmission probabilities has been simulated numerically in order to determine the degree of influence it has. At last, some numerical studies have been overview to explain the results which have been calculated theoretically and to analyze the impact of some parameters involved in the disease transmission model.

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