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 flaviviruses. 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 (Olayewin & Kribis, 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 Guillain–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) have 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 noninfected 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.
The above in Fig 1 is the projected model given by the resulting set of equations:

\[
\begin{align*}
\frac{dS_h}{dt} & = \pi_h + \gamma_h R_h - \beta_h \theta_h I_v S_h - \beta_h \theta_h I_v S_h - (\alpha + \mu_h) S_h \\
\frac{dS_v}{dt} & = \kappa I_v + \beta_v \theta_v I_v S_h - \beta_v \theta_v I_v S_h - \eta_h I_h - (\gamma_v + \mu_v) I_v \\
\frac{dS_p}{dt} & = \gamma_i I_h - (\gamma_h + \mu_h) R_h \\
\frac{dI_c}{dt} & = \alpha S_h - \beta_h \theta_h I_v I_c + \pi_c (N_h - \varepsilon_i I_c) \\
\frac{dI_h}{dt} & = \beta_h \theta_h I_v S_h + \beta_h \theta_h I_v S_h + \gamma_v - (\alpha + \mu_h) I_v \\
\frac{dI_p}{dt} & = \gamma_i I_h - (\gamma_h + \mu_h) R_h \\
\frac{dR_h}{dt} & = \beta_h \theta_h I_v S_h + \beta_h \theta_h I_v S_h + \gamma_v - (\alpha + \mu_h) I_v \\
\frac{dR_p}{dt} & = \gamma_i I_h - (\gamma_h + \mu_h) R_h
\end{align*}
\]

subjected to non-negative initial conditions

\[
(S_h(0) \geq 0, I_v(0) \geq 0, I_v(0) \geq 0, R_h(0) \geq 0, S_v(0) \geq 0, I_v(0) \geq 0, S_p(0) \geq 0, I_p(0) \geq 0, R_p(0) \geq 0)^T.
\]

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.

<table>
<thead>
<tr>
<th>Variables</th>
<th>Descriptions</th>
<th>Variables</th>
<th>Descriptions</th>
</tr>
</thead>
<tbody>
<tr>
<td>$S_h$</td>
<td>Susceptible Humans (adult)</td>
<td>$S_v$</td>
<td>Susceptible vectors (mosquitoes)</td>
</tr>
<tr>
<td>$I_h$</td>
<td>Infected Humans (adult)</td>
<td>$I_v$</td>
<td>Infected vectors (mosquitoes)</td>
</tr>
<tr>
<td>$R_h$</td>
<td>Recovered Human (adult)</td>
<td>$S_p$</td>
<td>Susceptible Primates</td>
</tr>
<tr>
<td>$P_h$</td>
<td>Pregnant Mothers</td>
<td>$I_p$</td>
<td>Infected Primates</td>
</tr>
<tr>
<td>$I_c$</td>
<td>Infected Children</td>
<td>$R_p$</td>
<td>Recovered Primates</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Symbol</th>
<th>Description</th>
<th>Value</th>
<th>Source</th>
</tr>
</thead>
<tbody>
<tr>
<td>$\pi_h$</td>
<td>The corresponding birth rates for humans and primates.</td>
<td>0.0461 &amp; 0.752</td>
<td>Fitted</td>
</tr>
<tr>
<td>$\pi_p$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\mu_h$, $\mu_v$</td>
<td>The natural mortality rates of humans, vectors, and primates respectively</td>
<td>$\frac{1}{60+365}$</td>
<td>Fitted, (Bonyah &amp; Okosun, 2016)</td>
</tr>
<tr>
<td>$\beta_1$, $\beta_2$</td>
<td>The biting rate of mosquitoes on humans, primates, and pregnant mothers respectively</td>
<td>[0.33-1], [0.3-0.9], and [0.33-1]</td>
<td>(Olawoyin &amp; Kribs, 2018), (Althouse et al., 2016), Fitted</td>
</tr>
<tr>
<td>$\beta_3$</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>$\theta_1$</td>
<td>Probability of infection transmission from infectious mosquitoes</td>
<td>[0.10-0.75]</td>
<td>(Maxian et al., 2017)</td>
</tr>
<tr>
<td>$\theta_2$</td>
<td>Probability of infection transmission to mosquito</td>
<td>[0.30-0.75]</td>
<td>(Maxian et al., 2017)</td>
</tr>
<tr>
<td>$\theta_3$</td>
<td>Probability of infection transmission from Mother to Infant</td>
<td>[0.5-0.86]</td>
<td>Fitted</td>
</tr>
<tr>
<td>$\eta_1$</td>
<td>Direct (sexual) transmission rate between humans</td>
<td>0.005</td>
<td>Fitted</td>
</tr>
<tr>
<td>$\eta_2$</td>
<td>Direct (sexual) transmission rate between primates</td>
<td>0.05</td>
<td>Fitted</td>
</tr>
<tr>
<td>$\gamma_1$, $\gamma_2$</td>
<td>Recovery rates of humans and primates respectively</td>
<td>0.1667, 0.2</td>
<td>(Suparit et al., 2018), (Althouse et al., 2014)</td>
</tr>
<tr>
<td>$\gamma_h$</td>
<td>Rate of waning of immunity for humans</td>
<td>0.05</td>
<td>Fitted</td>
</tr>
<tr>
<td>$\epsilon_1$, $\epsilon_2$</td>
<td>Probability of vertical transmission in humans and primates respectively</td>
<td>0.67, 0.06</td>
<td>(Bonaldo et al., 2020), (Lai et al., 2020)</td>
</tr>
<tr>
<td>$\epsilon_h$</td>
<td>Rate of transmission from susceptible human to infected human</td>
<td>0.06</td>
<td>(Lai et al., 2020)</td>
</tr>
<tr>
<td>$\epsilon_v$</td>
<td>Rate of transmission from susceptible mosquito to infected mosquito</td>
<td>0.06</td>
<td>(Lai et al., 2020)</td>
</tr>
<tr>
<td>$\pi_v$</td>
<td>Rate of hatching larva</td>
<td>0.275</td>
<td>Fitted</td>
</tr>
<tr>
<td>$\alpha$</td>
<td>Progression rate of a pregnant mother</td>
<td>0.07143</td>
<td>Fitted</td>
</tr>
<tr>
<td>$K$</td>
<td>Fraction of infected children to infected human</td>
<td>0.3</td>
<td>Fitted</td>
</tr>
</tbody>
</table>

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

\[
S_h + P_h + I_h + R_h = 1, \quad S_v + I_v = 1, \quad S_p + I_p + R_p = 1
\]

Hence, the normalized model is prearranged by:
\[ S_H' = \pi_s + \gamma_r R_H - \beta I H S_H - \beta I c e I c S_H - (\alpha + \mu_h) S_H \]
\[ I_H' = \kappa I + \beta I H S_H + \beta I c e I c S_H - \eta P_H I_H - (\gamma_1 + \mu_h) I_H \]
\[ R_H' = \gamma I H - (\gamma_1 + \mu_h) R_H \]
\[ P_H' = \pi_s (N H - e I c) + \alpha S_H + \eta P_H I_H - \beta \theta I c P_H - \mu_P P_H \]
\[ I_c' = \pi_s e I c + \beta \theta I c P_H - (\kappa + \mu_h) I_c \]
\[ S_c' = \pi_s (N e - e I c) - (\beta_1 + \beta_2) \theta e S_I V - \mu_s V \]
\[ I_v' = \pi_s e I v + (\beta_1 + \beta_2) \theta e S_I V - \mu_v V \]
\[ S_p' = \pi_s (N_p - e I p) - \beta \theta I p S_p - \beta \theta I p S_p + \eta S_p I_p - \mu_s S_p \]
\[ I_p' = \pi_s e I p + \beta \theta I p S_p - \beta_2 \theta I p S_p - \eta S_p I_p - (\gamma_2 + \mu_p) I_p \]
\[ R_p' = \gamma I p - \mu_R R_p \]

where \( X(0) = (S_H(0), P_H(0), I_H(0), I_c(0), S_c(0), I_v(0), S_v(0), I_p(0), S_p(0), I_p(0), R_p(0)) \geq 0 \)

**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

\[ X(t) = (S_H(t), P_H(t), I_H(t), I_c(t), S_c(t), I_v(t), S_v(t), I_p(t), S_p(t), I_p(t), R_p(t)) \]

of the system (3) with non-negative initial condition \( X(0) \) remains positive for all time \( t > 0 \), in a positively invariant closed set

\[ \Omega = \{ X(t) \in \mathbb{R}^{10} : 0 \leq S_H(t), P_H(t), I_H(t), I_c(t), S_c(t), I_v(t), S_v(t), I_p(t), S_p(t), I_p(t), R_p(t) \leq 0 \} \]

**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

\[ S_H'' = \pi_s + \gamma_r R_H - \beta \theta I H S_H - \beta \theta S H S_H - (\alpha + \mu_h) S_H \]

where, \( g_1 = \beta \theta I H + \beta \theta S H + (\alpha + \mu_h) \). It follows that

\[ S_H'' > (\beta \theta I H + \beta \theta S H + (\alpha + \mu_h) S_H \]

Integrating both sides over \( (0, \tau) \), we have;

\[ S_H(t) > S_H(0) + e^{\int_{0}^{\tau} [\beta \theta I H + \beta \theta S H + (\alpha + \mu_h)] dt} \]

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

\[ I_H(t) \geq I_H(0) + e^{\int_{0}^{t} \beta \theta I H dt} > 0 \]
\[ I_c(t) \geq I_c(0) + e^{\int_{0}^{t} \beta \theta S H dt} > 0 \]
\[ P_H(t) \geq P_H(0) + e^{\int_{0}^{t} (\alpha + \mu_h) dt} > 0 \]
\[ R_H(t) \geq R_H(0) + e^{\int_{0}^{t} (\gamma_1 + \mu_h) dt} > 0 \]

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 \( \tau \) cannot be supremum and hence, the solution will remain positive for all \( t > 0 \).

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

\[
\begin{bmatrix}
\psi_1(t) \\
\psi_2(t) \\
\psi_3(t)
\end{bmatrix} =
\begin{bmatrix}
S_H(t) + I_H(t) + I_c(t) + P_H(t) + R_H(t) \\
S_v(t) + I_v(t) \\
S_p(t) + I_p(t) + R_p(t)
\end{bmatrix}
\]

then, we have

\[ \psi'(t) = \begin{bmatrix}
\pi_s - \mu_s \psi_1(t) \\
\pi_s - \mu_s \psi_2(t) \\
\pi_s - \mu_s \psi_3(t)
\end{bmatrix} \]

Thus, solving for each component, we have:
\[
\begin{align*}
\psi_1(t) &= 2 - (2 - \psi_1(0)) e^{-t}, \\
\psi_2(t) &= 1 - (1 - \psi_1(0)) e^{-t}, \\
\psi_3(t) &= 1 - (1 - \psi_1(0)) e^{-t},
\end{align*}
\]

where \( \psi_1(0) = S_H(0) + I_H(0) + I_C(0) + R_H(0) \), \( \psi_2(0) = S_V(0) + I_V(0) \), and \( \psi_3(0) = S_I(0) + I_I(0) + R_I(0) \). Thus,
\[
\psi_i(t) \leq 1 \quad \text{if} \quad \psi_i(0) \leq 1, \ i = 1, 2, 3.
\]

Hence, the set \( \Omega \) is a positively invariant set \( 0 \leq \psi(t) \leq 1 \). Moreover, if \( \psi(0) > 1 \)
then \( \lim_{t \to + \infty} \psi(t) = 1 \) and then the set \( \Omega \) 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

\[
(S_v^0, I_v^0, R_v^0, S_h^0, I_h^0, R_h^0, S_p^0, I_p^0, R_p^0, I_c^0, R_c^0) = \left( \pi_v, \pi_v N_h, 0, 0, 0, \pi_v N_p, 0, 0, 0, 0, 0 \right).
\]

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

\[
S_h = \left( (\pi_h - A_h) \eta + (\beta, (\theta, A_h, \beta)(I, \theta, e, \theta)(\pi - A_h)) \beta - \theta_A, A_h, \beta, A_h, \beta) \beta + \beta, A_h, \beta - \beta Z H \right),
\]

\[
I_h = \left( (\pi_h - A_h) \eta + (\beta, (\theta, A_h, \beta)(I, \theta, e, \theta)(\pi - A_h)) \beta - \theta_A, A_h, \beta, A_h, \beta) \beta + \beta, A_h, \beta - \beta Z H \right),
\]

\[
R_h = \left( (\pi_h - A_h) \eta + (\beta, (\theta, A_h, \beta)(I, \theta, e, \theta)(\pi - A_h)) \beta - \theta_A, A_h, \beta, A_h, \beta) \beta + \beta, A_h, \beta - \beta Z H \right),
\]

\[
S_v = \left( \pi_v, \pi_v N_v, 0, 0, 0, \pi_v N_v, 0, 0, 0, 0, 0 \right),
\]

\[
I_v = \left( (\pi_v - A_v) \eta + (\beta, (\theta, A_v, \beta)(I, \theta, e, \theta)(\pi - A_v)) \beta - \theta_A, A_v, \beta, A_v, \beta) \beta + \beta, A_v, \beta - \beta Z V \right),
\]

\[
R_v = \left( (\pi_v - A_v) \eta + (\beta, (\theta, A_v, \beta)(I, \theta, e, \theta)(\pi - A_v)) \beta - \theta_A, A_v, \beta, A_v, \beta) \beta + \beta, A_v, \beta - \beta Z V \right),
\]

where \( Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \( Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]

\[
Z_h = (\pi_h - A_h) \gamma (A_h - \alpha)(I_h + e, \theta)(\beta_A - A_h) \), \ Z_v = (\pi_v - A_v) \gamma (A_v - \alpha)(I_v + e, \theta)(\beta_A - A_v) \).
\]
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 \( E^0 = (S^0_W, 0, P^0_H, 0, S^0_V, 0, S^0_P, 0, 0) \), where, \( S^0_W = P^0_H = S^0_V = S^0_P = 1 \). 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:

\[
F = \begin{bmatrix}
\beta_1 \theta_1 - \eta_1 & \beta_2 \theta_1 & 0 \\
0 & (\beta_1 + \beta_2) \theta_1 \varepsilon_v & 0 \\
0 & \beta_2 \theta_1 & \beta_3 \theta_1 - \eta_2
\end{bmatrix}
\]

and Movement in and out of the compartment

\[
V = \begin{bmatrix}
(\gamma_1 + \mu_h) & 0 & 0 \\
0 & \mu_v - \pi_v \varepsilon_v & 0 \\
0 & 0 & \mu_p - \pi_p \varepsilon_p
\end{bmatrix}
\]

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

\[
FV^{-1} = \begin{bmatrix}
\frac{\beta_2 \theta_1 - \eta_1}{\gamma_1 + \mu_h} & \frac{\beta_1 \theta_1}{\gamma_1 + \mu_h} & 0 \\
0 & \frac{(\beta_1 + \beta_2) \theta_1 \varepsilon_v}{-\pi_v \varepsilon_v + \mu_v} & 0 \\
0 & \frac{\beta_2 \theta_1}{-\pi_v \varepsilon_v + \mu_v} & \frac{\beta_3 \theta_1 - \eta_2}{-\pi_v \varepsilon_v + \mu_v + \gamma_1}
\end{bmatrix}
\]

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

\[
R_{0H} = \frac{\beta_1 \theta_1 - \eta_1}{\gamma_1 + \mu_h}, \quad R_{0V} = \frac{(\beta_1 + \beta_2) \theta_1 \varepsilon_v}{-\pi_v \varepsilon_v + \mu_v}, \quad R_{0P} = \frac{\beta_3 \theta_1 - \eta_2}{-\pi_v \varepsilon_v + \mu_p + \gamma_1}
\]

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:

\[
Y_{\ell_i}^{R_0} = \frac{\ell_i}{R_0} \frac{\partial R_0}{\partial \ell_i}
\]

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

\[
Y_{\ell_i}^{R_{0H}} = \frac{\ell_i}{R_{0H}} \frac{\partial R_{0H}}{\partial \ell_i}, \quad Y_{\ell_i}^{R_{0V}} = \frac{\ell_i}{R_{0V}} \frac{\partial R_{0V}}{\partial \ell_i}, \quad Y_{\ell_i}^{R_{0P}} = \frac{\ell_i}{R_{0P}} \frac{\partial R_{0P}}{\partial \ell_i}
\]

where \( \ell_1, \ell_2, \ell_3 \) 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>
<thead>
<tr>
<th>Parameter</th>
<th>Sign</th>
<th>( Y_{\ell_1}^{R_{0H}} )</th>
<th>Parameter</th>
<th>Sign</th>
<th>( Y_{\ell_1}^{R_{0V}} )</th>
<th>Parameter</th>
<th>Sign</th>
<th>( Y_{\ell_1}^{R_{0P}} )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \beta_1 )</td>
<td>+ve</td>
<td>1.0256</td>
<td>( \beta_1 )</td>
<td>+ve</td>
<td>0.3448</td>
<td>( \beta_2 )</td>
<td>+ve</td>
<td>1.1515</td>
</tr>
<tr>
<td>( \theta_2 )</td>
<td>+ve</td>
<td>0.6552</td>
<td>( \theta_2 )</td>
<td>+ve</td>
<td>1.0000</td>
<td>( \eta_2 )</td>
<td>-ve</td>
<td>0.1515</td>
</tr>
<tr>
<td>( \eta_1 )</td>
<td>-ve</td>
<td>0.0526</td>
<td>( \eta_2 )</td>
<td>+ve</td>
<td>1.0000</td>
<td>( \eta_2 )</td>
<td>-ve</td>
<td>0.1515</td>
</tr>
<tr>
<td>( \gamma_1 )</td>
<td>-ve</td>
<td>0.9997</td>
<td>( \varepsilon_v )</td>
<td>+ve</td>
<td>1.3004</td>
<td>( \pi_p )</td>
<td>-ve</td>
<td>2.5200</td>
</tr>
<tr>
<td>( \mu_h )</td>
<td>-ve</td>
<td>0.0003</td>
<td>( \pi_v )</td>
<td>+ve</td>
<td>0.3004</td>
<td>( \varepsilon_p )</td>
<td>-ve</td>
<td>2.5200</td>
</tr>
<tr>
<td>( \mu_v )</td>
<td>-ve</td>
<td>1.3004</td>
<td>( \pi_v )</td>
<td>+ve</td>
<td>0.3004</td>
<td>( \varepsilon_p )</td>
<td>-ve</td>
<td>2.5200</td>
</tr>
<tr>
<td>( \gamma_2 )</td>
<td>+ve</td>
<td>1.1200</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
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, $\beta_1$, $\theta_2$ biting rate of humans by the mosquito and probability of transmission of the virus 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 $\beta_2$, $\theta_2$. In addition, the results assert that $\theta_1$, and $\theta_2$; 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.](image1)

![Figure 3: Sensitivity indices of $R_0$ (Vector) concerning the model parameters](image2)
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

$$
J = \begin{pmatrix}
-Z_{11} & -\beta_1 \theta_1 e_s S_H & \gamma_1 & 0 & 0 & 0 & -\beta_1 \theta_1 S_D & 0 & 0 & 0 \\
Z_{21} & -Z_{22} & \eta_1 I_H & \kappa & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & \gamma_1 & -Z_{33} & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
\alpha & \eta_2 P_H & 0 & -Z_{44} & -Z_{45} & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & \beta_2 \theta_2 I_C & -Z_{55} & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & -Z_{66} & -Z_{67} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & Z_{76} & -Z_{77} & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_2 & -Z_{100} \\
\end{pmatrix}
$$

(23)

where,

$Z_{11} = -\left(\beta_1 \theta_1 I + \beta_1 \theta_1 e_s I_a + \alpha + \mu_a\right)$, $Z_{22} = -\left(-\beta_1 \theta_1 e_s S_H + \eta_1 P_H + \gamma_1 + \mu_a\right)$, $Z_{21} = \beta_2 \theta_2 I_C + \beta_2 \theta_2 e_s I_D$,

$Z_{33} = -\left(\gamma_1 + \mu_a\right)$, $Z_{44} = -\left(-\gamma_1 I_H + \beta_2 \theta_2 e_s I_a + \mu_a\right)$, $Z_{45} = -\left(\gamma_1 e_s + \beta_2 \theta_2 P_H\right)$,

$Z_{55} = -\left(-\gamma_1 e_s - \beta_2 \theta_2 P_H + \kappa + \mu_a\right)$, $Z_{66} = -\left((\beta_1 + \beta_2) e_s I_a + \mu_a\right)$, $Z_{67} = -\left(\pi_s e_s + (\beta_1 + \beta_2) e_s S_H\right)$,

$Z_{77} = -\left(-\pi_s e_s - (\beta_1 + \beta_2) e_s S_H + \mu_a\right)$, $Z_{76} = -\left(\beta_1 \theta_1 I_C + \beta_2 \theta_2 e_s I_a + \mu_a\right)$, $Z_{77} = -\left(\beta_1 \theta_1 I_C + \beta_2 \theta_2 e_s I_a - \gamma_1 I_H + \mu_a\right)$,

$Z_{98} = -\left(\pi_s e_s + \beta_1 \theta_1 e_s L - \eta_1 S_p\right)$, $Z_{99} = -\left(-\pi_s e_s - \beta_1 \theta_1 e_s L + \eta_1 S_p + \gamma_2 + \mu_p\right)$,

$Z_{99} = \beta_2 \theta_2 I_C + \beta_2 \theta_2 e_s L - \eta_1 S_p$, $Z_{100} = -\mu_p$.

**Theorem 2**

The above Jacobian has seven, negative eigenvalues, which are:
\[ \lambda_i = -\mu, \lambda_2 = -\mu, \lambda_3 = -\lambda_2, \lambda_4 = -\lambda_3, \lambda_5 = -\mu, \lambda_6 = -\mu, \lambda_7 = \left(\frac{\beta_i \tau_i \theta_i \varepsilon_i + N_\eta_\pi_\varsigma_i + A_\mu_\nu}{\mu_\nu}\right) \]  

The remaining eigenvalues are given in the characteristic equation \( P(\lambda) = 0 \), which is given by  
\[ P(\lambda) = \lambda^3 + a_1 \lambda^2 + a_2 \lambda + a_3 \]  
where  
\[ a_1 = \left(\mu_1^2 + (-\pi_1 \varepsilon_1 - \pi_2 \varepsilon_1 + A_1 + A_2) \mu + \pi_2 \varepsilon_2 N_\mu \right) \mu_2 + \pi_2 \varepsilon_2 N_\mu \mu_\pi \theta_i \]  
\[ a_2 = \left( -\pi_1 \varepsilon_1 + A_1 + A_2 \right) \mu_2 + \pi_2 \varepsilon_2 N_\mu \left(-\beta_2 \theta_2 + \eta_2\right) \mu_2 + \left(-\varepsilon_1 \left(-\pi_1 \varepsilon_1 + A_1 + A_2\right) \mu_1 + A_1 \left(-\pi_1 \varepsilon_1 + A_1\right) \mu_1 \right) \mu_\pi \theta_i \]  
\[ a_3 = \left(-\mu_1^2 + \left(-\pi_1 \varepsilon_1 + A_1\right) \mu_2 + \pi_2 \varepsilon_2 N_\mu \left(-\beta_2 \theta_2 + \eta_2\right) \mu_2 \right) - \pi_1 \varepsilon_1 \mu_\pi \theta_i \left(-\pi_1 \varepsilon_1 + A_1 + A_2\right) \mu_1 + \left(-\beta_2 \theta_2 + \eta_2\right) \mu_\pi \theta_i \]  
\[ a_4 = \left(-N_\beta_2 \pi_2 \varepsilon_2 \theta_2 \varepsilon_2 + A_1 \mu_2 \right) \left(\theta_1 \pi_1 \varepsilon_1 \beta_1 + \theta_2 \pi_1 \varepsilon_1 \beta_2 + \pi_1 \varepsilon_1 \mu_\pi \theta_i - \mu_\pi \theta_i \right) \left(\varepsilon_1 \left(-\pi_1 \varepsilon_1 + A_1 + A_2\right) \mu_1 + A_2 \left(-\pi_1 \varepsilon_1 + A_1\right) \mu_1 \right) \mu_\pi \theta_i \]  
\[ (after \ simplification) \]  
\[ H_3 = \frac{a_4}{a_2} - a_3 > 0 \]  
Thus, we conclude that the eigenvalues of \( p(\lambda) = 0 \) 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 (Chavez et al., 2002). Let \( X(t) \) and \( Y(t) \) represent the compartments define the uninfected and infected classes of the system (3), respectively:

\[ X(t) = \begin{bmatrix} S_0(t) \\ P_0(t) \\ R_0(t) \\ S_1(t) \\ R_1(t) \end{bmatrix}, \quad Y(t) = \begin{bmatrix} I_0(t) \\ I_1(t) \end{bmatrix} \]  

(27)

Thus, system (3) can be written as

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

(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:

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

* (\( H_2 \)) \( \tilde{G} \geq 0 \), where \( \tilde{G} (X, Y) = AY - G(X, Y) \) and \( A = D_Y - G(X^0, 0) \) is an Metzler matrix \( \forall (X, Y) \in \Omega \).
For the first condition \((H_1)\), we have,

\[
\frac{dX}{dt} = F(X,0) = \begin{pmatrix}
\pi_h - \mu S_h \\
\pi_h - \mu P_h \\
-\mu R_h \\
\pi_v - \mu S_v \\
-\mu P_v
\end{pmatrix}.
\]

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

\[
\begin{align*}
S_h(t) &= (1 + S_h(0))e^{-\mu t} \\
P_h(t) &= (1 + P_h(0))e^{-\mu t} \\
R_h(t) &= R_h(0)e^{-\mu t} \\
S_v(t) &= (1 + S_v(0))e^{-\mu t} \\
P_v(t) &= P_v(0)e^{-\mu t}
\end{align*}
\]

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

\[
\begin{pmatrix}
-\beta \theta \varepsilon_i - A_i - \eta_i \\
0 \\
\beta \theta_i + \pi _e e_i - A_i \\
0 \\
0 \\
0 \\
\pi _v e_i + (\beta _1 + \beta _2) \theta e_i - \mu_v \\
0 \\
0 \\
\beta _2 \theta_i \\
\beta _2 \theta_i + \pi _e e_i - A_i - \eta _i
\end{pmatrix} = 0.
\]

We have,

\[
G(X,Y) = AY - G(X,Y)
\]

\[
G(X,Y) = \begin{pmatrix}
(1 - S_h)(I, \theta _h - I_1 \theta _h \varepsilon_i) \beta _1 - I_1 \eta _h (1 - P_i) \\
\beta _1 \theta _h (1 - P_i) \\
I_1 \theta _h e_i (\beta _1 + \beta _2)(1 - S_v) \\
(1 - S_h)(I_1 \theta _h e_i (\beta _1 + \beta _2)(1 - S_v)
\end{pmatrix}
\]

It is obvious that since \(G \geq 0\) \(X,Y) \in \Omega\) where \(0 \leq (S_h, P_h, S_v, P_v) \leq 1\) 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 $\beta_3$ with respect to Carrier Mother class.

Figure 6: Graph showing dynamics $\theta_3$ with respect to Carrier Mother class.

Figure 7: The dynamics of Susceptible Infected and Recovered human class.
Figure 8: Graph showing the dynamics $\beta_1$ with respect to the Infected human class.

Figure 9: Graph showing the dynamics $\theta_1$ with respect to the Infected human class.

Figure 10: The dynamics $\theta_2$ with respect to the Infected human class.
Finally, through graphical methods, we examine the behaviors of the model by portraying the solution of Pregnant women against $\beta_3$ 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 $\beta_1$, $\theta_1$ and $\theta_2$. 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 $\varepsilon_v$ 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 \( R_{0_H} \), mosquitoes \( R_{0_V} \) (Vector) and Monkey \( R_{0_P} \), (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 \( \beta_1 \) and \( \beta_2 \) while the most negative influential parameter is \( \mu_H \). 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.

REFERENCES


