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Mathematical analysis of basic transmission dynamics model for Zika virus infection

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Abstract

In this article, we develop, validate and analyze an age-structured mathematical model of the Zika virus infection by incorporating vectorial, vertical and sexual transmission into the dynamics. The model is shown to be mathematically well-posed and epidemiologically feasible. Analysis of the model shows that, vectorial transmission contributes higher percent followed by sexual transmission, while vertical transmission in aquatic vectors contributes nearly insignificant percentage. Sensitivity analysis revealed that, the mosquito-human ratio constant, biting rate and the effective sexual contact rate are the most sensitive parameters in the disease reproduction numbers of the model.

Keywords: Zika virus, Age-structured model, Vertical transmission, Sexual transmission, Model fitting, Sensitivity analysis.

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1. Introduction

Zika virus (ZIKV) infection or Zika virus fever is an emerging arthropod-borne virus, shorted as arbovirus of the genus *Flavivirus* category or family transmitted to humans through the bites of day-time active *Aedes* mosquitoes such as *Aedes Aegypti*, *Aedes albopictus* and *Aedes Africanus* [8, 22, 35, 40, 42]. There is evidence of sexual transmission of the virus in humans through an extended infectious period of about 30 days, even when the blood samples of the sexually active infected adults tested negative for ZIKV [22, 26]. Moreover, a sexually active male or female may acquire the disease from having unprotected vaginal sex, anal sex, oral sex or even sharing of sex toys with an infected symptomatic or asymptomatic persons [9]. Fever illness resulting from ZIKV infection, is similar to mild form of Dengue fever or Chikungunya virus infection in terms of symptoms characterization [41], even though, most infected persons are asymptomatic, i,e., do not know or show symptoms [9]. However, Lorenzo *et al.* (2017) [24] through an investigative study on the ZIKV outbreak data from French territories reported that, the ratio of consulting to non-consulting patients (asymptomatic) is likely be as low as 1/3 to 1/4. Symptoms of ZIKV infection in humans may include all or some of the following: fever, myalgia, arthralgia, edema of extremities, maculopapular rash, retro-orbital pain, conjunctivitis and lymphadenopathies [34, 42]. Past experimental

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studies have shown that, a number of mosquito-borne *flavivirus* pathogens are vertically transmitted in their insect vectors, thereby, providing a means for persistence during harsh climatic conditions or in the absence of susceptible vertebrate host [39]. Similarly, in humans, congenital (vertical) transmission can be passed from a pregnant woman to her fetus during pregnancy or childbirth, though no information is available presently on how this happens . Apart from previously known complications during pregnancy, i.e., miscarriage and stillbirth [9], the ZIKV infection is now known to cause serious neurological disorders such as Guillain-Barre Syndrome and microcephaly in infants born to mothers who were infected with ZIKV during pregnancy [34, 39]. Presently, there is no evidence that past infection history will affect future pregnancies even when the virus has been cleared out of the human (female) body [9].

Specific laboratory investigative diagnosis of Zika virus may be conducted through virus isolation, antigen detection, viral RNA detection with molecular assays and anti-Zika virus antibodies detection with serological assays [13, 15, 56]. Among human clinical specimens, Zika virus can be cultured from blood [17], urine [21], saliva [5] and semen [32]. In spite of remarkable progress in vaccine and formal treatment development, there is no vaccine or treatment drugs available in circulation for the treatment of ZIKV infection and its associated diseases as at now. According to a report from the World Health Organization, the public health objective of the ZIKV vaccine is a vaccine candidate with minimal product characteristics for vaccines that can provide protection against congenital ZIKV syndrome especially during emergencies [54, 27]. Management of ZIKV infections in humans is usually symptomatic and the use of light pain relievers like acetaminophen (Tylenol) to reduce fever and joint pains caused by the virus [10]. Plenty of rest and intake of lots of fluids is highly recommended to avoid dehydration [10]. Aspirin and other non-steroidal anti-inflammatory drugs (NSAID) are not recommended during infection until co-infection with Dengue fever is ruled out, as this may cause bleeding in patients [10]. Human infectious period to mosquitoes is about $3-14$ days from the onset of the infectious period. Moreover, fully recovered humans are believed to be immune against the virus for life [43].

Previously, Zika virus was considered an obscure arbovirus of little public health importance. On 18 November 2016, it captured worldwide attention when WHO declared it a Public Health Emergency of International Concern (PHEIC) following the onset of a massive ZIKV outbreak in Brazil that began in May of the same year. This outbreak spread to other parts of the South American and Latin countries like Colombia and Mexico. Therefore, since then, the declaration has prompted so many researches that revisit what was already known or need to be known about the disease and how this knowledge can be harnessed into understanding the relevant parameters that govern the transmission dynamics of the infection. Notably, over the years, mechanistic and deterministic mathematical modeling approach was used to study the dynamics of the ZIKV infection. In the development of these models, basic information about the disease such as, the disease incubation period in humans and mosquitoes, transmission routes and other vital dynamics parameters were considered. Example of such models could be found in [1, 2, 6, 16, 19, 20, 26, 29, 30, 31, 33, 36, 44, 47, 48, 49, 51, 55]. However, from the existing models reviewed, vectorial, sexual and vertical transmission of ZIKV in mosquitoes was not concomitantly incorporated/considered to study the dynamics of the disease by researchers in such a way that, contributions of each of these transmission routes is associated with the right host or particular class of the host. For example, sexual transmission burdens only sexually active class of the human host. Therefore, in this paper, we developed and extensively studied a model for the basic transmission dynamics of the ZIKV infection in three completely susceptible host populations, namely; the humans, adult vectors and aquatic vectors. We will incorporate vectorial, sexual and vertical transmission in the vectors into the dynamics of the ZIKV infection. Variable vector-human contact rates to account for saturation, *gonotrophic* cycle and *anthropophilic* related constants as used by Chitnis (2008) [11] and Suparit et al. (2018) [45] will be used in estimating the forces of infection in the model.

2. Model Formulations

The model for the ZIKV transmission dynamics in this paper is an age-structured model with vital dynamics, describing the interactions between three specific host populations namely; humans, adult *Aedes* vectors and aquatic *Aedes* vectors. The model further categorized the human population $N_H(t)$ into three exclusive age groups viz; pre-sexually active $N_{H1}(t)$ ($0-14$ years, i.e., 24.5% of the total population [52]), sexually active $N_{H2}(t)$ ($15-64$ years, i.e., 64.1% of the total population [52]) and post-sexually active $N_{H3}(t)$ ($\geqslant 64$ years, i.e., 11.4% of the total population [52]) in the presence of adult *Aedes* vectors $N_V(t)$ and aquatic *Aedes* vectors $N_A(t)$ at time t. Evidence to support our age classification and definition for the sexually active humans can be found in the work of Sanchez-Franco and Gonzale-Uribe [38] where they found that, sexually active years in Colombia begins as early as $14-15$ years . Due to an observed significance in the number of asymptomatic (unreported) ZIKV infections, we divided the infectious human classes into symptomatic and asymptomatic, and we added two additional compartments in the sexually active humans to represent extended transmissibility period for the sexually active individuals through sexual contacts.

Thus, pre-sexually active susceptible individuals are recruited into $S_{H1}(t)$ by birth at a variable rate ΛN_H, where Λ is the birth rate. Sexually active and post-sexually active susceptible individuals are recruited into $S_{H2}(t)$ and $S_{H3}(t)$ respectively by the aging-out/maturity rates ρ_1 and ρ_2 of $S_{H1}(t)$ and $S_{H2}(t)$ respectively. A pre-sexually active and post-sexually active susceptible human in $S_{H1}(t)$ and $S_{H2}(t)$ comes into contact with ZIKV from the bite of an infectious adult *Aedes* vector in $I_V(t)$ at a variable rate $\lambda_{H1} = \lambda_{H3}$ (called the force of infection for age class $i = 1, 3$), while sexually active susceptible human in $S_{H2}(t)$ comes into contact with ZIKV either from the bite of an infectious adult *Aedes* vector in $I_V(t)$ or through having effective unprotected sexual contact with an infectious sexually active human in $I_{H,A2}(t)$, $I_{H,S2}(t)$, $J_{H,A2}(t)$, or $J_{H,S2}(t)$ at a different variable rate λ_{H2} (called the force of infection for age class $i = 2$) with,

$$\lambda_{H1} = \lambda_{H3} = \beta_{HV}\frac{I_V(t)}{N_H(t)},$$
$$\lambda_{H2} = \beta_{HV}\frac{I_V(t)}{N_H(t)} + \beta_{HH}\left(\frac{\sigma_{HH}I_{H,A2}(t) + I_{H,S2}(t) + \Phi(\sigma_{HH}J_{H,A2}(t) + J_{H,S2}(t))}{N_H(t)}\right),$$

and move to $E_{Hi}(t)$ in category i; ($i = 1, 2, 3$) accordingly, such that $\beta_{HV} = \frac{\rho_{HV}\sigma_H\sigma_V N_V(t)}{\sigma_V N_V(t)+\sigma_H N_H(t)}$ and $\beta_{HH} = s\rho_{HH}$; where β_{HV} is the per capita contact rate between infectious adult *Aedes* vector and susceptible human (per week), β_{HH} is the per capita contact rate between infectious sexually active human and susceptible sexually active human (per week), ρ_{HV} is the transmission probability from an infectious adult *Aedes* vector to a susceptible human per bite, ρ_{HH} is the transmission probability from an infectious human to susceptible human per sexual contact, s is the effective sexual contact rate (contacts per person per week), σ_H is the maximum number of adult *Aedes* vector bites a human can have per week (this is a function of the human's exposed surface area and any vector control interventions used by human to reduce exposure to adult *Aedes* vectors [11]), σ_V is the number of time one adult *Aedes* vector would want to bite humans per week if humans were freely available (this is a function of the vector's gonotrophic cycle, i.e.,the amount of time an adult *Aedes* vector requires to produce eggs, and its anthropophilic rate, i.e.,its preference for human blood [11]), Φ is the relative transmissibility from an infectious human in the J class to a susceptible human and σ_{HH} is the relative transmissibility from an infectious asymptomatic human in I or J class to a susceptible human given that an unprotected sexual contact has occurred. Note that, it is freely assumed that there is no sexual contact between all sexually active individuals and all other individuals in either pre-sexually or post sexually active classes in the model.

Thus, a proportion $q \in (0, 1)$ of the individuals in $E_{Hi}(t)$ after the disease intrinsic incubation period $1/\varepsilon$, develop symptoms of the ZIKV infection and move to $I_{H,Si}(t)$ at a rate $q\varepsilon$ while the remaining proportion $(1-q)$ of the individuals in $E_{Hi}(t)$ remain asymptomatically infectious with ZIKV infection and move $I_{H,Ai}(t)$ at a rate $(1-q)\varepsilon$. In this model, it is also assumed that vertical transmission of the ZIKV infection in humans is negligible and therefore not incorporated. Individuals in the two age groups 1 and 3 from the two infectious relative compartments $I_{H,Ai}(t)$ and $I_{H,Si}(t)$ recovers from the ZIKV infection spontaneously at different recovery rates γ and δ respectively and move to $R_{Hi}(t)$. Infectious individuals from the compartments $I_{H,A2}(t)$ and $I_{H,S2}(t)$ in age class $i = 2$, move to $J_{H,A2}(t)$ and $J_{H,S2}(t)$ respectively at different rates γ and δ due to their extended transmissibility periods through sexual route. Thus, individuals in $J_{H,A2}(t)$ and $J_{H,S2}(t)$ are only infectious to humans through sexual contacts but not to adult *Aedes* vectors, because at this period of their recovery, their blood does not contain ZIKV infection but the virus is contained in their bodily semen. All individuals in $J_{H,A2}(t)$ and $J_{H,S2}(t)$ recover fully and move to R_{H2} at a constant uniform rate r. All humans in the 3 age-group sub-populations suffer natural mortality at a uniform rate μ_H. Mortality due to the ZIKV infection is assumed negligible and hence not included into the basic dynamics model. All humans in the sub-populations in the age-structures $i = 1, 2$ progress to the next age class at a constant rate $\rho_i; i = 2, 3$ accordingly.

The adult *Aedes* vectors are recruited into the susceptible compartment $S_V(t)$ from the susceptible aquatic vectors $S_A(t)$ at a constant development/maturity rate ϕ. Therefore, an adult *Aedes* vector in $S_V(t)$ either lay a susceptible egg in $S_A(t)$ at a uniform constant laying rate θ or come into contact with ZIKV and move to the exposed class $E_V(t)$ by biting an infectious human in $I_{H,Ai}(t)$ or $I_{H,Si}(t)$; $(i = 1, 2, 3)$ at a variable rate λ_V (called the force of infection in adult *Aedes* vectors), with

$$\lambda_V = \beta_{VH} \times \sum_{i=1}^{3} \left(\frac{\sigma_{VH} I_{H,Ai}(t) + I_{H,Si}(t)}{N_H(t)} \right),$$

such that $\beta_{VH} = \frac{\rho_{VH}\sigma_H\sigma_V N_H(t)}{\sigma_V N_V(t)+\sigma_H N_H(t)}$, where β_{VH} is the per capita contact rate between infectious human to susceptible adult *Aedes* vector (per week) and ρ_{VH} is the transmission probability from an infectious human to susceptible adult *Aedes* vector per bite. An adult *Aedes* vector in $E_V(t)$ either lay a susceptible egg into $S_A(t)$ at the uniform rate θ or progress to $I_V(t)$ after the ZIKV extrinsic incubation period $1/\kappa$. Due to the fact that there is vertical transmission in the *Aedes* vectors, infectious adult *Aedes* vectors are also recruited into $I_V(t)$ from the infectious aquatic vectors $I_A(t)$ at the development/maturity rate ϕ. Thus, an infectious adult *Aedes* vector in $I_V(t)$ either lay a susceptible egg into $S_A(t)$ at the rate $(1-\pi)\theta$ or an infected aquatic egg into $I_A(t)$ at the rate $\pi\theta$, where π is the vertical transmission constant rate of ZIKV infection in the vectors. The aquatic vectors population comprises of vectors in the pre-developmental phases i.e., the egg, lava and the pupa mosquitoes of the *Aedes* category [23]. It is freely assumed here that all aquatic vectors do not bite humans and as such do not acquire/transmit the ZIKV infection from/to humans in the model. The model assumed that the infectious vectors do not recover or suffer mortality from the ZIKV infection. All the adult *Aedes* vectors suffer natural mortality at a uniform rate μ_V, and that all the aquatic vectors are assumed to suffer natural mortality throughout the aquatic phase at a uniform constant rate ξ. It is assumed that all the basic dynamics model parameters are strictly nonnegative. The model is later validated with life data from Zika outbreak of Columbia in 2016.

2.1. Equations of the ZIKV dynamics model

The description of the model in section 2 satisfies the following system of equations:

$$
\textbf{Human Populations}
\begin{cases}
 & = \Lambda N_H(t) - (\lambda_{H1} + \rho_1 + \mu_H) S_{H1}(t), \\
\frac{dE_{H1}(t)}{dt} & = \lambda_{H1} S_{H1}(t) - (\varepsilon + \rho_1 + \mu_H) E_{H1}(t), \\
\frac{dI_{H,A1}(t)}{dt} & = (1-q)\varepsilon E_{H1}(t) - (\gamma + \rho_1 + \mu_H) I_{H,A1}(t), \\
\frac{dI_{H,S1}(t)}{dt} & = q\varepsilon E_{H1}(t) - (\delta + \rho_1 + \mu_H) I_{H,S1}(t), \\
\frac{dR_{H1}(t)}{dt} & = \gamma I_{H,A1}(t) + \delta I_{H,S1}(t) - (\rho_1 + \mu_H) R_{H1}(t), \\
\frac{dS_{H2}(t)}{dt} & = \rho_1 S_{H1}(t) - (\lambda_{H2} + \rho_2 + \mu_H) S_{H2}(t), \\
\frac{dE_{H2}(t)}{dt} & = \rho_1 E_{H1}(t) + \lambda_{H2} S_{H2}(t) - (\varepsilon + \rho_2 + \mu_H) E_{H2}(t), \\
\frac{dI_{H,A2}(t)}{dt} & = \rho_1 I_{H,A1}(t) + (1-q)\varepsilon E_{H2}(t) - (\gamma + \rho_2 + \mu_H) I_{H,A2}(t), \\
\frac{dJ_{H,A2}(t)}{dt} & = \gamma I_{H,A2}(t) - (r + \rho_2 + \mu_H) J_{H,A2}(t), \\
\frac{dI_{H,S2}(t)}{dt} & = \rho_1 I_{H,S1}(t) + q\varepsilon E_{H2}(t) - (\delta + \rho_2 + \mu_H) I_{H,S2}(t), \\
\frac{dJ_{H,S2}(t)}{dt} & = \delta I_{H,S2}(t) - (r + \rho_2 + \mu_H) J_{H,S2}(t), \\
\frac{dR_{H2}(t)}{dt} & = \rho_1 R_{H1}(t) + r(J_{H,A2}(t) + J_{H,S2}(t)) - (\rho_2 + \mu_H) R_{H2}(t), \\
\frac{dS_{H3}(t)}{dt} & = \rho_2 S_{H2}(t) - (\lambda_{H3} + \mu_H) S_{H3}(t), \\
\frac{dE_{H3}(t)}{dt} & = \rho_2 E_{H2}(t) + \lambda_{H3} S_{H3}(t) - (\varepsilon + \mu_H) E_{H3}(t), \\
\frac{dI_{H,A3}(t)}{dt} & = \rho_2 I_{H,A2}(t) + (1-q)\varepsilon E_{H3}(t) - (\gamma + \mu_H) I_{H,A3}(t), \\
\frac{dI_{H,S3}(t)}{dt} & = \rho_2 I_{H,S2}(t) + q\varepsilon E_{H3}(t) - (\delta + \mu_H) I_{H,S3}(t), \\
\frac{dR_{H3}(t)}{dt} & = \rho_2 (J_{H,A2}(t) + J_{H,S2}(t) + R_{H2}(t)) + \gamma I_{H,A3}(t) + \delta I_{H,S3}(t) \\
 & \quad - \mu_H R_{H3}(t),
\end{cases}
\tag{2.1}
$$

$$
\textbf{Adult and Aquatic Vector Populations}
\begin{cases}
 & = \phi S_A(t) - (\lambda_V + \mu_V) S_V(t), \\
\frac{dE_V(t)}{dt} & = \lambda_V S_V(t) - (\kappa + \mu_V) E_V(t), \\
\frac{dI_V(t)}{dt} & = \kappa E_V(t) + \phi I_A(t) - \mu_V I_V(t), \\
\frac{dS_A(t)}{dt} & = \theta N_V(t) - \pi\theta I_V - (\phi + \xi) S_A(t), \\
\frac{dI_A(t)}{dt} & = \pi\theta I_V - (\phi + \xi) I_A(t).
\end{cases}
\tag{2.2}
$$

The model has the following initial data ($\forall i = 1, 2, 3$):

$$
\begin{aligned}
& S_{Hi}(0) > 0, E_{Hi}(0) \geqslant 0, I_{H,Ai}(0) \geqslant 0, I_{H,Si}(0) \geqslant 0, J_{H,A2}(0) \geqslant 0, J_{H,S2}(0) \geqslant 0, \\
& R_{Hi}(0) \geqslant 0, S_V(0) > 0, E_V(0) \geqslant 0, I_V(0) \geqslant 0, S_A(0) > 0, I_A(0) \geqslant 0,
\end{aligned}
$$

where;

$$
\begin{aligned}
N_H(t) =& S_{Hi}(t) + E_{Hi}(t) + I_{H,Ai}(t) + J_{H,A2}(t) + I_{H,Si}(t) + J_{H,S2}(t) + R_{Hi}(t), \\
N_V(t) =& S_V(t) + E_V(t) + I_V(t), N_A(t) = S_A(t) + I_A(t).
\end{aligned}
$$

2.2. Standardization of the ZIKV basic dynamics model

Define a constant parameter m, such that $m = \frac{N_V}{N_H}$, where m is the vectors-to-humans ratio (i.e., vectors per human [26]), N_V and N_H are the adult vectors and the human populations respectively. Thus, the standardized basic dynamics model equations are:

$$\begin{cases}
\frac{ds_{h1}(t)}{dt} = \Lambda - (\lambda_{h1} + \rho_1 + \mu_H)s_{h1}(t),\\
\frac{de_{h1}(t)}{dt} = \lambda_{h1}s_{h1}(t) - (\varepsilon + \rho_1 + \mu_H)e_{h1}(t),\\
\frac{di_{h,a1}(t)}{dt} = (1-q)\varepsilon e_{h1}(t) - (\gamma + \rho_1 + \mu_H)i_{h,a1}(t),\\
\frac{di_{h,s1}(t)}{dt} = q\varepsilon e_{h1}(t) - (\delta + \rho_1 + \mu_H)i_{h,s1}(t),\\
\frac{dr_{h1}(t)}{dt} = \gamma i_{h,a1}(t) + \delta i_{h,s1}(t) - (\rho_1 + \mu_H)r_{h1}(t),\\
\frac{ds_{h2}(t)}{dt} = \rho_1 s_{h1}(t) - (\lambda_{h2} + \rho_2 + \mu_H)s_{h2}(t),\\
\frac{de_{h2}(t)}{dt} = \rho_1 e_{h1}(t) + \lambda_{h2}s_{h2}(t) - (\varepsilon + \rho_2 + \mu_H)e_{h2}(t),\\
\frac{di_{h,a2}(t)}{dt} = \rho_1 i_{h,a1}(t) + (1-q)\varepsilon e_{h2}(t) - (\gamma + \rho_2 + \mu_H)i_{h,a2}(t),\\
\frac{dj_{h,a2}(t)}{dt} = \gamma i_{h,a2}(t) - (r + \rho_2 + \mu_H)j_{h,a2}(t),\\
\frac{di_{h,s2}(t)}{dt} = \rho_1 i_{h,s1}(t) + q\varepsilon e_{h2}(t) - (\delta + \rho_2 + \mu_H)i_{h,s2}(t),\\
\frac{dj_{h,s2}(t)}{dt} = \delta i_{h,s2}(t) - (r + \rho_2 + \mu_H)j_{h,s2}(t),\\
\frac{dr_{h2}(t)}{dt} = \rho_1 r_{h1}(t) + r(j_{h,a2}(t) + j_{h,s2}(t)) - (\rho_2 + \mu_H)r_{h2}(t),\\
\frac{ds_{h3}(t)}{dt} = \rho_2 s_{h2}(t) - (\lambda_{h3} + \mu_H)s_{h3}(t),\\
\frac{de_{h3}(t)}{dt} = \rho_2 e_{h2}(t) + \lambda_{h3}s_{h3}(t) - (\varepsilon + \mu_H)e_{h3}(t),\\
\frac{di_{h,a3}(t)}{dt} = \rho_2 i_{h,a2}(t) + (1-q)\varepsilon e_{h3}(t) - (\gamma + \mu_H)i_{h,a3}(t),\\
\frac{di_{h,s3}(t)}{dt} = \rho_2 i_{h,s2}(t) + q\varepsilon e_{h3}(t) - (\delta + \mu_H)i_{h,s3}(t),\\
\frac{dr_{h3}(t)}{dt} = \rho_2(j_{h,a2}(t) + j_{h,s2}(t) + r_{h2}(t)) + \gamma i_{h,a3}(t) + \delta i_{h,s3}(t) - \mu_H r_{h3}(t),\\
\frac{ds_v(t)}{dt} = \phi s_a(t) - (\lambda_v + \mu_V)s_v(t),\\
\frac{de_v(t)}{dt} = \lambda_v s_v(t) - (\kappa + \mu_V)e_v(t),\\
\frac{di_v(t)}{dt} = \kappa e_v(t) + \phi i_a(t) - \mu_V i_v(t),\\
\frac{ds_a(t)}{dt} = m\theta - \pi\theta i_v - (\phi + \xi)s_a(t),\\
\frac{di_a(t)}{dt} = \pi\theta i_v - (\phi + \xi)i_a(t).
\end{cases} \tag{2.3}$$

The standardized basic dynamics model have the following initial data for $i = 1, 2, 3$:

$$\begin{aligned}
&s_{hi}(0) > 0, e_{hi}(0) \geqslant 0, i_{h,ai}(0) \geqslant 0, j_{h,a2}(0) \geqslant 0, i_{h,si}(0) \geqslant 0, j_{h,s2}(0) \geqslant 0, r_{hi}(0) \geqslant 0,\\
&s_v(0) > 0, e_v(0) \geqslant 0, i_v(0) \geqslant 0, s_a(0) > 0, i_a(0) \geqslant 0,
\end{aligned} \tag{2.4}$$

with,

$$\begin{aligned} &\lambda_{h1} = \lambda_{h3} = \beta_{HV} i_v(t), \lambda_v = \beta_{VH} \sum_{i=1}^{3} \left(\sigma_{VH} i_{h,ai}(t) + i_{h,si}(t)\right); i = 1,2,3, \\ &\lambda_{h2} = \beta_{HV} i_v(t) + \beta_{HH}\left(\sigma_{HH} i_{h,a2}(t) + i_{h,s2}(t) + \Phi(\sigma_{HH} j_{h,a2}(t) + j_{h,s2}(t))\right), \end{aligned} \tag{2.5}$$

such that $\beta_{HH} = s\rho_{HH}$, $\beta_{HV} = \frac{\rho_{HV}\sigma_H\sigma_V N_v(t)}{\sigma_V N_v(t) + \sigma_H N_h(t)}$, $\beta_{VH} = \frac{\rho_{VH}\sigma_H\sigma_V N_h(t)}{\sigma_V N_v(t) + \sigma_H N_h(t)}$, and $N_v(t) = mN_h(t)$. Note that, if $N_h(t) \leqslant 1$, then $0 \leqslant \liminf_{t\to\infty} N_v(t) \leqslant \limsup_{t\to\infty} N_v(t) \leqslant m$.

3. Results

3.1. Basic Results of the Standardized Model

3.1.1. Existence, uniqueness and positivity of solutions

Theorem 3.1. *(Existence, uniqueness and positivity of solutions). Let the model in proportions given by (2.3) and (2.4) be written as an initial value problem of the form*

$$\begin{aligned} &\frac{dy}{dt} = f(t, y), \\ &y(0) = y_0, y \in \mathbb{R}^{22}_{+}. \end{aligned} \tag{3.1}$$

Suppose $J = [0, a]$ *and* $Z = \overline{B(y_0, b)}$, *where* $f : J \times Z \to \mathbb{R}^{22}_{+}$ *is Lipschitz with Lipschitz constant* L, *and* $|f(y)| \leqslant M$ *for all* $y \in Z$, *then the model system (2.3) has a unique solution* $y \in C^0(J, Z)$ *as long as the time-interval is chosen with* a *satisfying* $0 < a < \min\left(\frac{1}{L}, \frac{b}{M}\right); Lb < 1$. *Furthermore, the solution of model with nonnegative initial conditions (2.4) remain positive for all* $t > 0$.

Proof. We begin the proof by showing that f is lipschitz. Thus, we can rewrite the model given by (3.1) in the form

$$f(y) = Ay + \mathbf{g}(y) + \mathbf{h},$$

where A, $\mathbf{g}$ and $\mathbf{h}$ are matrices of orders (22×22), (22×1) and (22×1) representing the linear, nonlinear and the constant terms of the system (2.3) respectively. By the definition of a Lipschitz condition in [12], we have

$$\begin{aligned} \|f(y(t)) - f(y^*(t))\| &= \|Ay(t) - Ay^*(t) + \mathbf{g}(y(t)) - \mathbf{g}(y^*(t)) + \mathbf{h}(y) - \mathbf{h}(y^*)\|, \\ &= \|Ay(t) - Ay^*(t) + \mathbf{g}(y(t)) - \mathbf{g}(y^*(t))\|, \\ &= \|A(y(t) - y^*(t)) + \mathbf{g}(y(t)) - \mathbf{g}(y^*(t))\|, \\ &\leqslant \|A\|\|y - y^*\| + \|\mathbf{g}(y) - \mathbf{g}(y^*)\|, \\ &\leqslant (\|A\| + ^{-})\|y - y^*\|, \\ &\leqslant L\|y - y^*\|, \quad L = (\|A\| + ^{-}) < \infty \end{aligned}$$

Integrating (3.1) from 0 to t with respect to time, and applying the initial condition yielded the following integral equation:

$$y(t) = y_0 + \int_0^t f(y(s))\, ds. \tag{3.2}$$

Therefore, $y(t)$ in (3.2) is a solution of model (3.1) if and only if it is a solution of (3.2). Using equation (3.2), define an operator Γ on a bounded function $Z = \overline{B(y_0, b)}$ from a closed interval $J = [0, a]$ to $\mathbb{R}^{22}$, by

$$\Gamma(z(t)) = y_0 + \int_0^t f(z(s))\, ds. \tag{3.3}$$

satisfying $\Gamma(0) = y_0$. Therefore, to complete the proof, we need to show that, the operator Γ in (3.3) is a contraction mapping. Thus, to do that, it is enough to show that the operator Γ maps a complete metric space $C^0(J, Z)$ to itself. But firstly, for $z(t) \in Z$ we will impose a condition on a that strictly guarantees $\Gamma(z(t)) \in Z$ for all time, $t \in J$.

$$
\begin{aligned}
|\Gamma(z(t)) - y_0| &= \operatorname{Sup}_{t\in J}\left|\int_0^t f(z(s))\,ds\right|,\\
&\leqslant \int_0^a |f(z(s))|\,ds,\\
&\leqslant Ma, \quad \text{but } a < \min\left(\frac{1}{L}, \frac{b}{M}\right),\\
&\leqslant b
\end{aligned}
\tag{3.4}
$$

Thus, with this strict restriction imposed, it implied that, $\Gamma(z(t)) \in Z$ for all $t \in J$. Now, we have shown that $\Gamma : Z \to Z$. Next, we need to show that the operator Γ is a contraction mapping on $C^0(J, Z)$ using the Contraction Mapping Theorem in [37]. Therefore, given any two functions $z_1, z_2 \in C^0(J, Z)$, using the definition of the operator Γ in (3.2), we have;

$$
\begin{aligned}
|\Gamma(z_1(t)) - \Gamma(z_2(t))| &= \operatorname{Sup}_{t\in J}\left|\int_0^t [f(z_1(s)) - f(z_2(s))]\,ds\right|,\\
&\leqslant \operatorname{Sup}_{t\in J}\left(\int_0^t |f(z_1(s)) - f(z_2(s))|\,ds\right),\\
&\leqslant \int_0^b |f(z_1(s)) - f(z_2(s))|ds,\\
&\leqslant L\int_0^b |z_1(s) - z_2(s)|ds,\\
&\leqslant Lb\|z_1 - z_2\|.
\end{aligned}
\tag{3.5}
$$

Therefore, for appropriate choice of $Lb < 1$, the map Γ is contraction on $C^0(J, Z)$. Thus, by the Picard-Lindelöf Theorem of Existence and Uniqueness in [46], we conclude, there exist a unique fixed point $z \in C^0(J, Z)$ for the standardized model (2.3).

To prove positivity of the model solution, let

$$
\begin{aligned}
t_1 =\sup\{&s_{hi}(0) > 0, e_{hi}(0) > 0, i_{h,ai}(0) > 0, j_{h,a2}(0) > 0, i_{h,si}(0) > 0,\\
&j_{h,s2}(0) > 0, r_{hi}(0) > 0, s_v(0) > 0, e_v(0) > 0, i_v(0) > 0, s_a(0) > 0,\\
&i_a(0) > 0; i = 1, 2, 3\}.
\end{aligned}
$$

Therefore, applying change of parameters technique on the first equation in (2.3), we have;

$$
\begin{aligned}
s_{h1}(t) =&s_{h1}(0) \times \exp\left(-\int_0^{t_1} \lambda_{h1}(u)du - \rho_1(t) - \mu_H(t)\right)\\
&+\exp\left(-\int_0^{t_1} \lambda_{h1}(u)du - \rho_1(t) - \mu_H(t)\right)\\
&\times \int_0^{t_1} \Lambda\exp\left(\int_0^{z} \lambda_{h1}(u)du + \rho_1(z) + \mu_H(z)\right) dz > 0.
\end{aligned}
\tag{3.6}
$$

Similarly, in the same way, it can be shown that; $s_{h2}(t) > 0, s_{h3}(t) > 0, e_{hi}(t) > 0, i_{h,ai}(t) > 0, j_{h,a2}(t) > 0, i_{h,si}(t) > 0, j_{h,s2}(t) > 0, r_{hi}(t) > 0, s_v(t) > 0, e_v(t) > 0, i_v(t) > 0, s_a(t) > 0, i_a(t) > 0$ for all $t > 0$, such that $i = 1, 2, 3$. Hence, the solutions of the basic dynamics model (2.3) with nonnegative initial conditions in (2.4) remain positive for all $t > 0$, which concludes the proof. □

3.1.2. Boundedness of model solutions and invariant region

Lemma 3.2. *The closed set* $\mathcal{D} = \{\mathcal{D}_h \cup \mathcal{D}_v \cup \mathcal{D}_a \subset \mathbb{R}^{17}_+ \times \mathbb{R}^3_+ \times \mathbb{R}^2_+ \subseteq \mathbb{R}^{22}_+\}$, *with*

$$\mathcal{D}_h = (s_{hi}, e_{hi}, i_{h,ai}, j_{h,a2}, i_{h,si}, j_{h,s2}, r_{hi}) \in \mathbb{R}^{17}_+ : (i = 1,2,3);$$

$$e_{hi} \geqslant 0, i_{h,ai} \geqslant 0, j_{h,a2} \geqslant 0, i_{h,si} \geqslant 0, j_{h,s2} \geqslant 0, r_{hi} \geqslant 0,$$

$$s_{h1} \leqslant \frac{\Lambda}{\rho_1 + \mu_H}, s_{h2} \leqslant \frac{\rho_1}{\rho_2 + \mu_H} \times \frac{\Lambda}{\rho_1 + \mu_H}, s_{h3} \leqslant \frac{\rho_1 \rho_2}{\mu_H(\rho_2 + \mu_H)} \times \frac{\Lambda}{\rho_1 + \mu_H}; N_h(t) > 0$$

$$\mathcal{D}_v = (s_v, e_v, i_v) \in \mathbb{R}^3_+ : e_v \geqslant 0, i_v \geqslant 0, s_v \leqslant \frac{m\theta\phi}{\mu_v(\phi + \xi)}; N_v > 0$$

and

$$\mathcal{D}_a = (s_a, i_a) \in \mathbb{R}^2_+ : i_a \geqslant 0, s_a \leqslant \frac{m\theta}{\phi + \xi}; N_a > 0$$

is a positively invariant and attracting set for the basic dynamics model (2.3).

Proof. By the Comparison Theorem [18], using the appropriate methods on the system (2.3), we can see that;

$$\begin{aligned} 0 &\leqslant \liminf_{t\to\infty} s_{h1}(t) \leqslant \limsup_{t\to\infty} s_{h1}(t) \leqslant \frac{\Lambda}{\rho_1 + \mu_H}, \\ 0 &\leqslant \liminf_{t\to\infty} s_{h2}(t) \leqslant \limsup_{t\to\infty} s_{h2}(t) \leqslant \frac{\Lambda}{\rho_1 + \mu_H} \times \frac{\rho_1}{\rho_2 + \mu_H}., \\ 0 &\leqslant \liminf_{t\to\infty} s_{h3}(t) \leqslant \limsup_{t\to\infty} s_{h3}(t) \leqslant \frac{\Lambda}{\rho_1 + \mu_H} \times \frac{\rho_1}{\rho_2 + \mu_H} \times \frac{\rho_2}{\mu_H}. \end{aligned} \tag{3.7}$$

Note that, N_h is the total sum of the human sub-populations in the basic dynamics model (2.3), including s_{h1}, s_{h2} and s_{h3}. Therefore, in the absence of the disease in the humans, i.e. when $e_{hi} = i_{h,ai} = j_{h,a2} = i_{h,si} = j_{h,s2} = r_{hi} = 0$ $(\forall i = 1,2,3)$; the human population is represented by the value $s_{h1} + s_{h2} + s_{h3} \leqslant N_h(t) \leqslant \frac{\Lambda}{\mu_H}$. Obviously, $0 \leqslant \liminf_{t\to\infty} N_h(t) \leqslant \limsup_{t\to\infty} N_h(t) \leqslant \frac{\Lambda}{\mu_H}$, which implies, $N_h(t) > 0$. Hence, the feasible solution $(s_{hi}, e_{hi}, i_{h,ai}, j_{h,a2},$ $i_{h,si}, j_{h,s2}, r_{hi}); (\forall i = 1,2,3)$ of the human population in the standardized basic dynamics model system enters the positively invariant set $\mathcal{D}_h \subseteq \mathbb{R}^{17}_+$.

Similarly, in the same manner, we can see that;

$$\begin{aligned} 0 &\leqslant \liminf_{t\to\infty} s_v(t) \leqslant \limsup_{t\to\infty} s_v(t) \leqslant \frac{m\theta\phi}{\mu_v(\phi + \xi)}, \\ 0 &\leqslant \liminf_{t\to\infty} s_a(t) \leqslant \limsup_{t\to\infty} s_a(t) \leqslant \frac{m\theta}{\phi + \xi}. \end{aligned} \tag{3.8}$$

Therefore, in the absence of the ZIKV infection in the adult *Aedes* vector and vertical transmission in the aquatic vector populations, the adult and aquatic *Aedes* vector populations are represented by $s_v(t) \leqslant N_v(t) \leqslant \frac{m\theta\phi}{\mu_v(\phi+\xi)}$ and $s_a(t) \leqslant N_a(t) \leqslant \frac{m\theta}{\phi+\xi}$ respectively. Thus, obviously $0 \leqslant \liminf_{t\to\infty} N_v(t) \leqslant \limsup_{t\to\infty} < N_v(t) \leqslant \frac{m\theta\phi}{\mu_v(\phi+\xi)}$ and $0 \leqslant \liminf_{t\to\infty} N_a(t) \leqslant \limsup_{t\to\infty} N_a(t) \leqslant \frac{m\theta}{\phi+\xi}$, which implies, $N_v(t) > 0$ and $N_a(t) > 0$. Hence, the feasible solutions; $(s_v(t), e_v(t), i_v(t))$ of the adult *Aedes* vector population and $(s_a(t), i_a(t))$ of the aquatic vector population enters the positively invariant sets $\mathcal{D}_v \subseteq \mathbb{R}^5_+$ and $\mathcal{D}_a \subseteq \mathbb{R}^2_+$ respectively.

Thus, $\mathcal{D} = \mathcal{D}_h \cup \mathcal{D}_v \cup \mathcal{D}_a \subseteq \mathbb{R}^{22}_+$ is a positively invariant set being the union of finite invariant sets.

Clearly, under the dynamics described by the standardized basic dynamics model (2.1), the closed set $\mathcal{D} = \{\mathcal{D}_h \cup \mathcal{D}_v \cup \mathcal{D}_a \subset \mathbb{R}^{17}_+ \times \mathbb{R}^3_+ \times \mathbb{R}^2_+ \subset \mathbb{R}^{22}_+\}$ is hence positively invariant and attracting set, and all the model solutions are uniformly bounded in the set. □

Remark 3.3. From Lemma (3.2), the standardized basic dynamics model system is therefore mathematically well-posed and epidemiologically meaningful. For any starting initial point $y_0 \in \mathcal{D}$, the solution trajectories that begins in $\mathcal{D}$, remains in $\mathcal{D}$, for all time $t > 0$. Furthermore, it is sufficient to restrict the model analysis on the invariant set $\mathcal{D}$.

3.2. *Main Results of the Standardized Model*

3.2.1. *Model fitting*

To validate the model and estimate values for some of the model parameters, we fitted the basic dynamics model with real-time data in Table (1) for the 2016 Zika outbreak in Colombia as used and published in the work of Biswas *et al.* [4]. This data was initially reported by the Colombian National Institute of Health, SIVIGILA as stated by Aranda *et al.* [3]. We used the sample size population $N_H(t) = 47,630,000$; which corresponds to the estimated population size of Colombia in 2016 [25]. All efforts to obtain categorized data that fits the age classification in the model proved difficult. In view of this, we relaxed the age-structure in the basic dynamics model by neglecting maturity/aging out rates, but maintained the three transmission routes (i.e., vectorial, vertical and sexual) in the model as it is in (2.1) and (2.1).

In view of that, we went ahead with the model fitting using the Nonlinear Least Squares (NLS) method [7]. It is a statistical technique used to build regression model for data sets that contain nonlinear features. A built-in MATLAB function fminsearch was used to minimize the sum of square errors. Thus, the result of model fitting is presented in Fig. (1). The estimated parameter values by the model, and other baseline parameter values source from existing literature are presented in Table (2) and Table (3).

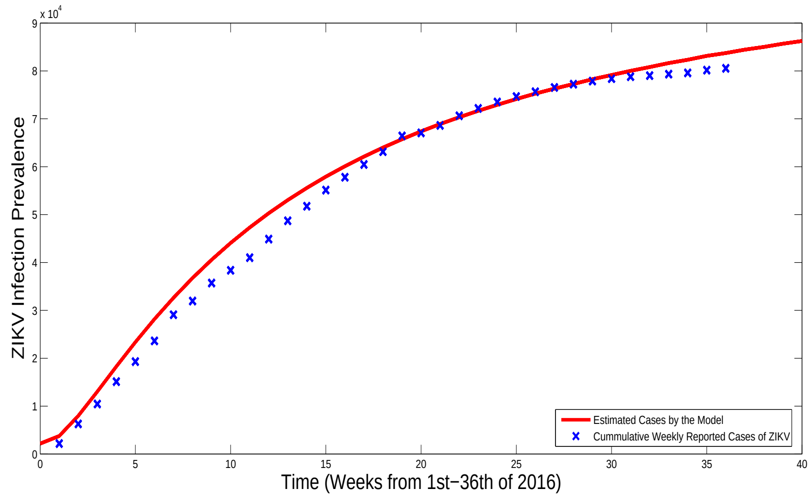

Figure 1: Model Fitting for the Basic Dynamics Model

Table 1: Weekly Reported ZIKV Cases from Week1-Week36 of 2016 [3, 4]

Week	Reported Cases	Weekly Cumulative Cases
1	2173	2173
2	4105	6278
3	4166	10444
4	4669	15113
5	4198	19311
6	4316	23627
7	5460	29087
8	2865	31952
9	3767	35719
10	2655	38374
11	2639	41013
12	3882	44895
13	3808	48703
14	3059	51762
15	3364	55126
16	2671	57797
17	2665	60462
18	2687	63149
19	3281	66430
20	638	67068
21	1567	68635
22	2014	70649
23	1539	72188
24	1344	73532
25	1128	74660
26	991	75651
27	892	76543
28	705	77248
29	648	77896
30	496	78392
31	416	78808
32	215	79023
33	301	79324
34	271	79595
35	568	80163
36	383	80546

Table 2: Baseline Human Parameters Values

Parameter	Value	Reference
Λ	$\frac{3}{200} \times \frac{1}{52} week^{-1}$	[25].
μ_H	$\frac{1}{200} \times \frac{1}{52} week^{-1}$	[53].
$\frac{1}{\varepsilon}$	$\frac{10}{7} weeks$	Estimated.
$\frac{1}{\delta}$	$\frac{4}{7} weeks$	[19].
$\frac{1}{\gamma}$	$\frac{5}{7} weeks$	[19].
$\frac{1}{r}$	$\frac{30}{7} weeks$	[26].
ρ_1	$\frac{1}{16} \times \frac{1}{52} week^{-1}$	[26].
ρ_2	$\frac{1}{49} \times \frac{1}{52} week^{-1}$	[26].
ρ_{HH}	0.99	Estimated.
σ_{HH}	0.99	Estimated.
Φ	0.081	Estimated.
s	$0.20 week^{-1}$	Estimated.
q	0.4	Estimated.

Table 3: Baseline Vector and Human-Vector Interaction Parameters Values

Parameter	Value	Reference
μ_v	$\frac{7}{13}$week^{-1}	[30].
$\frac{1}{\kappa}$	$\frac{10}{7}$weeks	[26].
σ_{VH}	0.30	Estimated.
σ_H	10.64week^{-1}	Estimated.
σ_V	0.39wekk^{-1}	Estimated.
π	$\frac{1}{290}$	[39].
θ	0.00003week^{-1}	Estimated.
ϕ	0.0808week^{-1}	Estimated.
ξ	$\frac{399}{10000}$week^{-1}	Estimated.
ρ_{HV}	0.72	Estimated.
ρ_{VH}	0.31	Estimated.
m	735.40	Estimated.

3.2.2. Disease-Free Equilibrium (DFE) and the epidemic reproduction number of the model

The DFE of the model system (2.3) is computed as;

$$\mathcal{E}_0 = \left(s_{h1}^0, 0, 0, 0, 0, s_{h2}^0, 0, 0, 0, 0, 0, 0, s_{h3}^0, 0, 0, 0, 0, s_v^0, 0, 0, s_a^0, 0\right), \tag{3.9}$$

where; $s_{h1}^0 = \frac{\Lambda}{\rho_1+\mu_H}$, $s_{h2}^0 = \frac{\rho_1\Lambda}{(\rho_1+\mu_H)(\rho_2+\mu_H)}$, $s_{h3}^0 = \frac{\rho_1\rho_2\Lambda}{\mu_H(\rho_1+\mu_H)(\rho_2+\mu_H)}$, $s_v^0 = \frac{m\theta\phi}{\mu_v(\phi+\xi)}$ and $s_a^0 = \frac{m\theta}{\phi+\xi}$.

The basic reproduction number $\mathcal{R}_0$ is defined to be the largest eigenvalue or spectral radius of the matrix $(\mathcal{F}\mathcal{V}^{-1})$ [14], where $\mathcal{F}$ is the Jacobian matrix of new infection terms, $(\mathcal{F}_i)$, and $\mathcal{V}$ is the Jacobian matrix of transition terms, $(\mathcal{V}_i)$ in an infectious class i of the standardized basic dynamics model (2.3). Therefore, using the notation definitions in Van den Driessche and James (2002) [50], we defined

$$\mathcal{F}_i = \begin{pmatrix} \lambda_{h1}s_{h1} \\ 0 \\ 0 \\ \lambda_{h2}s_{h2} \\ 0 \\ 0 \\ 0 \\ 0 \\ \lambda_{h3}s_{h3} \\ 0 \\ 0 \\ \lambda_v s_v \\ 0 \\ 0 \end{pmatrix}, \mathcal{V}_i = \begin{pmatrix} (\varepsilon+\rho_1+\mu_H)e_{h1} \\ -(1-q)\varepsilon e_{h1} + (\gamma+\rho_1+\mu_H)i_{h,a1} \\ -q\varepsilon e_{h1} + (\gamma+\rho_1+\mu_H)i_{h,a1} \\ -\rho_1 e_{h1} + (\varepsilon+\rho_2+\mu_H)e_{h2} \\ -\rho_1 i_{h,a1} - (1-q)\varepsilon e_{h2} + (\gamma+\rho_2+\mu_H)i_{h,a2} \\ -\gamma i_{h,a2} + (r+\rho_2+\mu_H)j_{h,a2} \\ -\rho_1 i_{h,s1} - q\varepsilon e_{h2} + (\delta+\rho_2+\mu_H)i_{h,s2} \\ -\delta i_{h,s2} + (r+\rho_2+\mu_H)j_{h,s2} \\ -\rho_2 e_{h2} + (\varepsilon+\mu_H)e_{h3} \\ -\rho_2 i_{h,a2} - (1-q)\varepsilon e_{h3} + (\gamma+\mu_H)i_{h,a3} \\ -\rho_2 i_{h,s2} - q\varepsilon e_{h3} + (\delta+\mu_H)i_{h,s3} \\ (\kappa+\mu_V)e_v \\ -\kappa e_v - \phi i_a + \mu_V i_v \\ -\pi\theta i_v + (\phi+\xi)i_a \end{pmatrix}.$$

Using the next-generation matrix method to find the analytical representation for the epidemic reproduction number $\mathcal{R}_0$, we obtained an implicit and inexpressible solution when we incorporate vectorial, vertical and sexual transmission in the age-structured model. Hence, we resolve to computing the numerical equivalent of the epidemic reproduction number by finding the numerical equivalent of the Jacobian matrices $\mathcal{F}$ and $\mathcal{V}$ using the baseline parameter values in Table (2) and Table (3). Thus, evaluating the characteristic equation $|\mathcal{F}\mathcal{V}^{-1} - \lambda I| = 0$, we obtained the ZIKV epidemic reproduction number for the model $\mathcal{R}_0 = 0.58459$, which is the dominant/largest eigenvalue of the numerical version of the matrix $(\mathcal{F}\mathcal{V}^{-1})$.

3.2.3. Assessing contributions of the transmission routes on dynamics of ZIKV infection

To asses the contribution or relevance of each of the transmission route incorporated into the dynamics of the ZIKV infection described by the model (2.3), we computed a straight-forward solutions for the vectorial and the vertical transmission only ($\mathcal{R}_V^0$), and the sexual transmission only ($\mathcal{R}_S^0$) epidemic reproduction numbers. In the computation of $\mathcal{R}_V^0$, we ignored human demographies in the model (2.3) to obtain

$$\begin{aligned}\mathcal{R}_V^0 &= \sqrt{\mathcal{R}_{A\times V}^0 + \mathcal{R}_{S\times V}^0},\\ \mathcal{R}_V^0 &= \sqrt{\frac{\rho_{HV}\rho_{VH}\kappa m^2\sigma_H^2\sigma_V^2\theta\phi}{\mu_V^2(m\sigma_V+\sigma_H)^2(\kappa+\mu_V)(\phi+\xi)(1-\mathcal{R}_a^0)}\left(\frac{\sigma_{VH}(1-q)}{\gamma}+\frac{q}{\delta}\right)},\end{aligned} \tag{3.10}$$

where

$\mathcal{R}_a^0 = \frac{\pi\theta\phi}{\mu_V(\phi+\xi)}$ is the vertical reproductive number in the aquatic vectors,
$\mathcal{R}_{A\times V}^0 = \frac{\rho_{HV}\rho_{VH}\kappa m^2\sigma_H^2\sigma_V^2\theta\phi\sigma_{VH}(1-q)}{\mu_V^2(m\sigma_V+\sigma_H)^2(\kappa+\mu_V)(\phi+\xi)(1-R_a^0)\gamma}$ is the vectorial reproduction number through the asymptomatic infectious humans, and $\mathcal{R}_{S\times V}^0 = \frac{\rho_{HV}\rho_{VH}\kappa m^2\sigma_H^2\sigma_V^2\theta\phi q}{\mu_V^2(m\sigma_V+\sigma_H)^2(\kappa+\mu_V)(\phi+\xi)(1-R_a^0)\delta}$ is the vectorial reproduction number through the symptomatic infectious humans. Note that, $\mathcal{R}_V^0, \mathcal{R}_{A\times V}^0$ and $\mathcal{R}_{S\times V}^0$ exists if and only if, $0 \leqslant \mathcal{R}_a^0 < 1$.

Similarly, the sexual transmission only reproduction number is computed in the same manner and is found to be:

$$\begin{aligned}\mathcal{R}_S^0 &= \mathcal{R}_{AS}^0 + \mathcal{R}_{SS}^0,\\ \mathcal{R}_S^0 &= \frac{s\rho_{HH}\rho_1\Lambda}{(\rho_1+\mu_H)(\rho_2+\mu_H)}\left(\frac{\sigma_{HH}(1-q)}{\gamma}\times\frac{(r+\Phi\gamma)}{r}+\frac{q}{\delta}\times\frac{(r+\Phi\delta)}{r}\right),\end{aligned} \tag{3.11}$$

where $\mathcal{R}_{AS}^0 = \frac{s\rho_{HH}\rho_1\Lambda\sigma_{HH}(1-q)(r+\Phi\gamma)}{(\rho_1+\mu_H)(\rho_2+\mu_H)\gamma r}$ is the asymptomatic sexually active human's infectious period through their extended transmissibility period in the $j_{h,a2}$ class, and $\mathcal{R}_{SS}^0 = \frac{s\rho_{HH}\rho_1\Lambda q(r+\Phi\delta)}{(\rho_1+\mu_H)(\rho_2+\mu_H)\delta r}$ is the symptomatic sexually active human's infectious period through their extended transmissibility period in the $j_{h,s2}$ class.

Using the baseline parameter values in Table (3.1) and Table (3.2), we compute the vectorial plus vertical transmission only $\mathcal{R}_V^0 = 0.35757$, the vertical transmission only $\mathcal{R}_a^0 = 1.7594 \times 10^{-6}$ and the sexual transmission only $\mathcal{R}_S^0 = 0.21899$ reproduction numbers numerically.

From the relation (using the numerically computed $\mathcal{R}_0$)

$$\frac{\mathcal{R}_0 - \mathcal{R}_0^\star}{\mathcal{R}_0} \times 100\% \tag{3.12}$$

where $\mathcal{R}_0^\star$ is any of the numerically computed $\mathcal{R}_V^0, \mathcal{R}_a^0$ or $\mathcal{R}_S^0$, we discovered, the vectorial and the vertical transmission route contributed about 62% of the reported cases, while the sexual transmission route

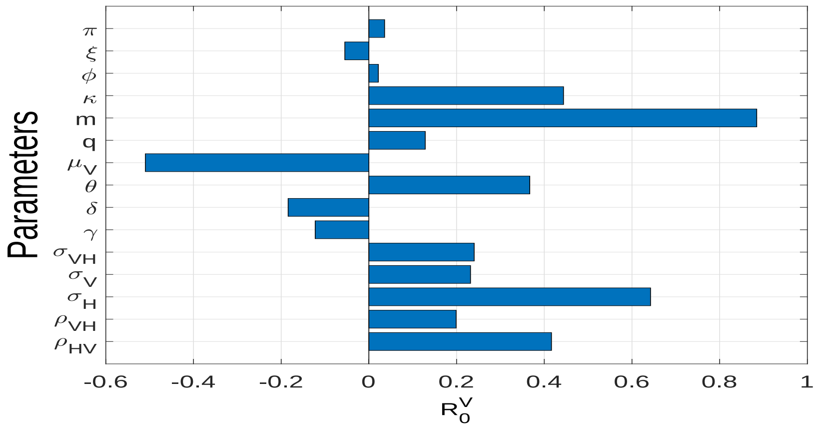

Figure 2: PRCC Results for $\mathcal{R}_0^V$

contributed about 38% of the reported cases in the 2016 Colombia outbreak. It is therefore noticed that, sexual transmission contributed significant percentage of the cases, while the vertical transmission in the aquatic vectors contributed nearly insignificant percentage (i.e., below 1%) of the ZIKV cases.

3.2.4. *Global sensitivity analysis*

In this section, we conducted global sensitivity analysis to assess the sensitivity indices of each model parameter in the vectorial transmission only $(\mathcal{R}_0^V)$ and sexual transmission only $(\mathcal{R}_0^S)$ reproduction numbers using Partial Rank Correlation Coefficient (PRCC) method based on Latin hypercube sampling as applied by Ali (2021) [28]. The results are summarized in Fig. (2) and Fig. (3) respectively. In each experiment, we computed the minimum, mean and the maximum values of the two variables $\mathcal{R}_0^V$ $(0.0004, 0.032, 0.2799)$ and $\mathcal{R}_0^S$ $(0.0004, 0.0319, 0.2758)$.

From the results of the experiment in Fig. (2), we observed that, the mosquito-to-human ratio parameter (m), biting rate (σ_H), egg laying rate (θ), aquatic vectors' maturity/development rate (ϕ), aquatic and adult vectors' mortality rates $(\xi$ and $\mu_V)$ and the recovery rates for symptomatic and asymptomatic $(\delta$ and $\gamma)$ infectious humans are the most sensitive parameters in the vectorial and vertical transmission only reproduction number, $\mathcal{R}_0^V$.

Moreover, the results of the experiment presented in Fig. (3) indicates that, the sexual contact rate (s), the probability of transmission from an infectious sexually active human to a susceptible sexually active human (ρ_{HH}), relative transmissibility constants of asymptomatic infectious humans (σ_{HH}) and the recovery rates $(\delta, \gamma,$ and $r)$ are the most sensitive parameters in the sexual transmission only reproduction number, $\mathcal{R}_0^S$.

4. Summary

In this paper, a deterministic mathematical model for the dynamics of ZIKV infection was formulated and analyzed. The model was shown to be mathematically well-posed and epidemiologically meaningful by validation and proving the existence of unique, positive and bounded model solution in a certain invariant set. In formulating the model, agent-based vectorial, sexual and vertical transmission of the virus in the *Aedes* vectors were considered due to the fact that, ZIKV like other *flavivirus* pathogens of the same clan (e.g., Dengue virus) is vertically transmitted in the insect vectors. Variable vector contact rates are used in the representation of the disease forces of infection due to an observed correlation between the density of the vector population and the human population. Due to the size and stiff nonlinearity of the system, we computed the disease reproduction number $\mathcal{R}_0$ numerically using the parameter values

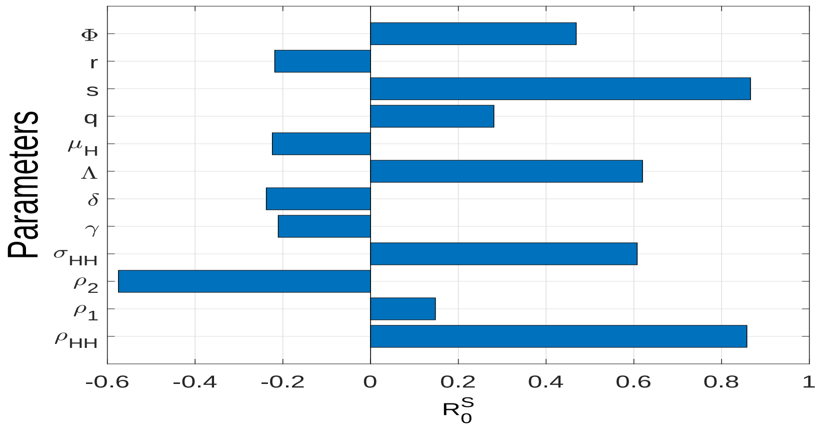

Figure 3: PRCC Results for $\mathcal{R}_0^S$

estimated by the model. To assess the contribution of the transmission routes included in the dynamics, we computed the numerical values of the vectorial and vertical transmission only reproduction number $\mathcal{R}_V^0$ and the sexual transmission only reproduction number $\mathcal{R}_S^0$. It is important to note that, the numerical value of the ZIKV epidemic reproduction number $\mathcal{R}_0$ is approximately the sum of the numerical values of the vectorial and vertical transmission only reproduction number $\mathcal{R}_V^0$ and the sexual transmission only reproduction number $\mathcal{R}_S^0$, i.e., $\mathcal{R}_0 \approx \mathcal{R}_V^0 + \mathcal{R}_S^0 = 0.60$, correct to one significant figure. This value suggests that, the prevalence of the ZIKV cases will die out in the populations naturally over time as a result of low transmission probabilities estimated by the model using the reported data, and the use of variable contact rates between humans and the *Aedes* vectors that takes humans exposure, availability, vector's gonotrophic cycle and its anthropophilic constants (i.e., its preference for human blood) into account. The computational analysis reveal that, sexual transmission in humans is more significant when the value of the epidemic reproduction number $\mathcal{R}_0$ is above 2.632, and that, vertical transmission in the aquatic vectors is not of importance on the general dynamics of the ZIKV infection as it contributed less than 1% of the reported ZIKV cases. The results also indicated that, vertical transmission alone cannot trigger an outbreak of the virus over time. This is as a result that, the aquatic vectors through the vertical transmission generates only about 3.68% of new recruits into the infectious mature vectors population. Hence, a mathematical situation that guarantees $R_a^0 \geqslant 1$ is of no epidemiological or biological meaning as seen in the value of analytic $\mathcal{R}_V^0$ in equation (3.10). Global sensitivity analysis results suggest that, any control intervention that combine efforts to minimize; human activities that enhances breeding and development ground for the adult and aquatic vectors such as stagnant water, refuse and empty damp containers/cans, human exposure to adult vector bites by wearing full-cover clothing and use of mosquito bednets, recovery periods of infectious humans through early testing and use of formal therapeutics can help in reducing the ZIKV cases caused by the vectorial transmission. Additionally, implementing control strategy that prevents unprotected sexual contact between infectious and susceptible sexually active humans through the use of condoms will help in reducing the ZIKV cases caused by sexual transmission.

5. Conclusion

A new deterministic model that incorporates vectorial, sexual and vertical transmission in *Aedes* vectors into the basic dynamics of Zika virus infection is formulated and validated. The use of variable contact rates to represent vector-human interactions gives low estimate values of the new infections generated by the incidence functions in the model. Results of the model analysis indicated that sexual transmission in the humans is significantly important especially when the value of the disease reproduction number is above 2.632. The vertical transmission in the *Aedes* vectors is not important on the dynamics of the Zika virus since its contribution in the reported cases is below 1%, and the route alone cannot trigger an outbreak over time. Thus. the computed numerical values of the disease reproduction numbers suggests that, Zika virus infection will be wiped out from the populations over time.

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