A Host–Vector Mathematical Model for Zika Virus Transmission in North-East Nigeria

Abstract

This research introduces a deterministic host-vector mathematical framework for exploring the spread of Zika virus in North-East Nigeria. The model encompasses mosquito-borne transmission pathways, sexual transmission, infected pregnant women, temporary immunity, and the impact of overcrowding in internally displaced persons (IDP) camps. Important characteristics of the model, such as positivity and boundedness of solutions are proven. The model's disease-free equilibrium and basic reproduction number R_0 are computed and rigorously analyzed. It is shown that the disease free equilibrium is locally and globally asymptotically stable if $R_0 < 1$ and that the endemic equilibrium exists if $R_0 > 1$. Analysis indicates that overcrowding and increased human-mosquito contact amplify the transmission of the disease. Recommended control strategies integrate vector control, environmental sanitation, public health awareness, and alleviating overcrowding in IDP camps. The model offers an effective tool for analyzing Zika virus dynamics and informing disease control policies in North-East Nigeria.

Keywords: Zika Virus, Mathematical Modeling, Mosquito-Borne Transmission, Internally Displaced Persons, North-East Nigeria, Stability Analysis.

1 Introduction

Zika virus (ZIKV) is an arthropod-borne virus belonging to the genus *Flavivirus* within the family *Flaviviridae*. It is closely related to other medically important flaviviruses such as dengue virus, yellow fever virus, West Nile virus, and Japanese encephalitis virus [6, 11]. The virus was first isolated in 1947 from a sentinel rhesus monkey in the Zika Forest of Uganda during surveillance studies of yellow fever and was subsequently identified in humans in 1952 [6, 14]. For several decades after its discovery, Zika virus was regarded as a relatively obscure pathogen responsible for sporadic human infections across Africa and Asia. However, the epidemiological significance of the virus changed dramatically following large outbreaks in the Pacific Islands and the Americas, culminating in the global public health emergency declared by the World Health Organization in 2016 [23, 16].

Zika virus is primarily transmitted through the bite of infected *Aedes* mosquitoes, particularly *Aedes aegypti* and *Aedes albopictus*, which also mediate the transmission of dengue, chikungunya, and urban yellow fever [24, 8]. These mosquito vectors thrive in tropical and subtropical regions with high temperatures, seasonal rainfall, poor sanitation, and ample stagnant water, conditions that frequently prevail in sub-Saharan Africa, and especially in Nigeria, thus providing ideal settings for Zika virus propagation [11, 9].

While mosquito bites remain the primary mode of infection, Zika virus exhibits several unusual features for an arbovirus, as infections have been documented through sexual contact, blood transfusion,

organ transplantation, laboratory accidents, and perinatal transfer from mother to fetus [24, 25]. This ability to be transmitted through varied pathways enhances the possibility of disease persistence. Sexual transmission in particular allows passage from male to female and between males, which may permit cycles among humans that are less dependent on mosquito population size and feeding rates [25].

The most troubling characteristic of Zika virus infection is its capacity to cause birth defects: while most people with Zika virus infection are asymptomatic and those that fall ill experience only mild fever, rash, conjunctivitis, body and joint pain, and headache, infection during pregnancy can lead to a variety of complications. Causality between infection during pregnancy and microcephaly, low birth weight, brain and eye abnormalities, and other problems with brain development has been confirmed by numerous studies [19, 13, 5], and has also been linked to "other complications of pregnancy including fetal demise, placental insufficiency, and fetal growth restriction" [5]. According to the WHO, Zika is a cause of Guillain, Barr'e syndrome along with other complications of the brain and nervous system [8, 24].

With hundreds of thousands of infections reported in Brazil between 2015, 2016, with a corresponding increase in cases of microcephaly and other birth defects, Zika virus has recently emerged as a major global health concern [23, **?**]. Since then, epidemiological surveillance of the virus has increased worldwide, and considerable research has focused on mechanisms of transmission, particularly with regard to pregnancy, and ways to bring the disease under control despite the lack of a vaccine [16, 9].

In Africa, where the virus was first discovered, Zika virus often goes undetected, though many serological and molecular studies have shown the presence of virus in multiple countries [11, 1]. Nigeria in particular is primed for the transmission of mosquito-borne viruses due to rapid urbanization, poor sanitation, unstable climate patterns, and the widespread presence of *Aedes* mosquitoes.

There is serological and molecular evidence of Zika virus exposure among pregnant women and febrile patients in Nigeria [20, 3]. Shaibu et al. described evidence of Zika virus exposure among pregnant women in Lagos [20], and Bamidele et al. reported the prevalence of Zika virus in pregnant women and described several risk factors for infection in Nigeria [3].

In North-East Nigeria, years of humanitarian crisis have displaced millions of people, many of whom now live in camps for internally-displaced persons (IDPs) under densely-packed and insanitary conditions that increase the risk of mosquito-human contact, especially during the rainy season. These factors indicate that North-East Nigeria is at high risk of experiencing a Zika virus outbreak.

Mathematical modeling is an essential approach to studying transmission dynamics and the potential impact of interventions for infectious diseases. Models provide a structured approach to examining the processes that result in disease causing morbidity and mortality within a population, and allow critical transitions to be derived and for scenarios to be promptly explored [2, 22].

2 Model Formulation

The total human population is

$$N_h(t) = S(t) + E(t) + I(t) + P(t) + R(t),$$

where

- $S(t)$: susceptible humans,
- $E(t)$: exposed humans,
- $I(t)$: infectious humans,
- $P(t)$: infected pregnant women,
- $R(t)$: recovered humans.

The mosquito population is

$$N_m(t) = S_m(t) + E_m(t) + I_m(t),$$

where

- $S_m(t)$: susceptible mosquitoes,
- $E_m(t)$: exposed mosquitoes,
- $I_m(t)$: infectious mosquitoes.

2.1 Model Assumptions

1. Human recruitment occurs at rate ψ.
2. Mosquito recruitment occurs at rate μ.
3. Infection occurs through mosquito bites.
4. Sexual transmission occurs among humans.
5. Exposed humans and mosquitoes undergo latent periods.
6. A proportion of infected women enter the pregnancy-associated class.
7. Recovered individuals acquire temporary immunity.
8. IDP camp overcrowding increases disease transmission.
9. Natural mortality occurs in all compartments.

2.2 Force of Infection

The force of infection for humans is

$$\tau_h = \beta\left(1 + \varepsilon C\right)\frac{I_m}{N_h} + \theta\frac{I}{N_h},$$

where

- β is the mosquito-to-human transmission rate,
- θ is the sexual transmission rate,
- ε measures overcrowding effects,
- C denotes the IDP density index.

The force of infection for mosquitoes is

$$\tau_m = \xi\frac{I}{N_h},$$

where ξ is the human-to-mosquito transmission coefficient.

2.3 Model Equations

The transmission dynamics are governed by

$$\begin{aligned}
\frac{dS}{dt} &= \psi - \tau_h S + \kappa R - \phi S,\\
\frac{dE}{dt} &= \tau_h S - (\alpha + \phi)E,\\
\frac{dI}{dt} &= \alpha E - (\varrho + \xi + \phi)I,\\
\frac{dP}{dt} &= \xi I - (\zeta + \delta + \phi)P,\\
\frac{dR}{dt} &= \varrho I + \zeta P - (\kappa + \phi)R,\\
\frac{dS_m}{dt} &= \mu - \tau_m S_m - \nu S_m,\\
\frac{dE_m}{dt} &= \tau_m S_m - (\sigma + \nu)E_m,\\
\frac{dI_m}{dt} &= \sigma E_m - \nu I_m.
\end{aligned} \tag{1}$$

2.4 State Variables

Here are the variables of the model and their descriptions.

Table 1: State Variables

Variable	Description
S	Susceptible humans
E	Exposed humans
I	Infectious humans
P	Infected pregnant women
R	Recovered humans
S_m	Susceptible mosquitoes
E_m	Exposed mosquitoes
I_m	Infectious mosquitoes

2.5 Parameter Description

Parameters of the model are defined below.

Table 2: Model Parameters

Parameter	Description
ψ	Human recruitment rate
μ	Mosquito recruitment rate
ϕ	Human natural death rate
ν	Mosquito natural death rate
β	Mosquito-to-human transmission rate
θ	Sexual transmission coefficient
α	Human incubation rate
σ	Mosquito incubation rate
ϱ	Human recovery rate
ξ	Progression rate to infected pregnancy class
ζ	Recovery rate of infected pregnant women
δ	Disease-induced mortality rate
κ	Loss of immunity rate
ξ	Human-to-mosquito transmission coefficient
ε	IDP overcrowding effect
C	IDP density index

3 Qualitative and Stability Analysis of the Model

In this section, we establish some fundamental mathematical properties of the proposed Zika virus transmission model, including positivity of solutions, boundedness, invariant region, disease-free equilibrium, basic reproduction number, and stability results.

3.1 Positivity of Solutions

Theorem 1. *Let*

$$(S(0), E(0), I(0), P(0), R(0), S_m(0), E_m(0), I_m(0)) \in \mathbb{R}^8_+.$$

Then the solutions of system (1) *remain non-negative for all* $t > 0$.

Proof. Consider the first equation of system (1):

$$\frac{dS}{dt} = \psi - \tau_h S + \kappa R - \phi S.$$

Suppose $S(t) = 0$. Then

$$\left.\frac{dS}{dt}\right|_{S=0} = \psi + \kappa R \geq 0.$$

Hence $S(t)$ cannot become negative.
Similarly,

$$\left.\frac{dE}{dt}\right|_{E=0} = \tau_h S \geq 0,$$

$$\left.\frac{dI}{dt}\right|_{I=0} = \alpha E \geq 0,$$

$$\left.\frac{dP}{dt}\right|_{P=0} = \xi I \geq 0,$$

$$\left.\frac{dR}{dt}\right|_{R=0} = \varrho I + \zeta P \geq 0,$$

$$\left.\frac{dS_m}{dt}\right|_{S_m=0} = \mu \geq 0,$$

$$\left.\frac{dE_m}{dt}\right|_{E_m=0} = \tau_m S_m \geq 0,$$

and

$$\left.\frac{dI_m}{dt}\right|_{I_m=0} = \sigma E_m \geq 0.$$

Therefore none of the state variables can become negative.
Hence

$$(S, E, I, P, R, S_m, E_m, I_m) \in \mathbb{R}^8_+$$

for all $t > 0$.
Thus the non-negative orthant is positively invariant. □

The positivity theorem establishes that all state variables remain non-negative for all future times whenever the initial conditions are non-negative. This result is essential because the state variables represent human and mosquito populations. Negative population sizes have no biological meaning and would render the model invalid. The proof shows that each coordinate hyperplane is a barrier that cannot be crossed by solutions; as soon as some state variable hits zero, the corresponding derivative is nonnegative and so the state variable cannot become negative.

Biologically, this ensures that this model preserves the physicality of the epidemic process; people may enter or leave classes of the epidemiological model through infection, recovery, disease progression, or death, but the populations in each class can never become negative. The model is therefore biologically sound and makes sense from an epidemiological standpoint.

3.2 Boundedness and Invariant Region

Theorem 2. *The region*

$$\Omega = \left\{(S, E, I, P, R, S_m, E_m, I_m) \in \mathbb{R}^8_+ : N_h \leq \frac{\psi}{\phi},\ N_m \leq \frac{\mu}{\nu}\right\}$$

is positively invariant and attracting.

Proof. Let

$$N_h = S + E + I + P + R.$$

Adding the first five equations of system (1) gives

$$\frac{dN_h}{dt} = \psi - \phi N_h - \delta P.$$

Since $\delta P \geq 0$,

$$\frac{dN_h}{dt} \leq \psi - \phi N_h.$$

Using the Comparison Theorem,

$$N_h(t) \leq N_h(0)e^{-\phi t} + \frac{\psi}{\phi}\left(1 - e^{-\phi t}\right).$$

Hence

$$\limsup_{t\to\infty} N_h(t) \leq \frac{\psi}{\phi}.$$

Similarly,

$$N_m = S_m + E_m + I_m.$$

Adding the mosquito equations yields

$$\frac{dN_m}{dt} = \mu - \nu N_m.$$

Solving gives

$$N_m(t) = N_m(0)e^{-\nu t} + \frac{\mu}{\nu}\left(1 - e^{-\nu t}\right).$$

Therefore

$$\limsup_{t\to\infty} N_m(t) \leq \frac{\mu}{\nu}.$$

Consequently,

$$\Omega$$

is positively invariant and attracting. □

The boundedness theorem demonstrates that the human and mosquito populations are bounded for all time. $\frac{\psi}{\phi}$ is the limit imposed on the human population by the recruitment and natural death rates. The total human population, regardless of initial condition, will eventually be bounded above by this quantity. Likewise, for mosquito population, $N_m(t) \leq \frac{\mu}{\nu}$. This upper-bound is the constraint on the mosquito population imposed by the mosquito recruitment and death rates. The significance of this result is that no solution can blowup, and that a compact region Ω exists such that all solutions eventually enter and remain in Ω. Hence, all asymptotic dynamical behavior of the epidemic occurs in a compact set. Biologically, boundedness means that constraints on the populations (e.g. environmental, demographic, etc.) restrict the viable levels of carriers for the disease. Therefore, the dynamics of the epidemic occur in a biologically relevant regime.

3.3 Existence of Equilibria

The model possesses two biologically meaningful equilibria:

1. Disease-Free Equilibrium (DFE),
2. Endemic Equilibrium (EE).

3.4 Disease-Free Equilibrium

Setting

$$E = I = P = R = E_m = I_m = 0$$

in system (1) gives the disease-free equilibrium

$$E_0 = \left(\frac{\psi}{\phi}, 0, 0, 0, 0, \frac{\mu}{\nu}, 0, 0\right).$$

At the disease-free equilibrium, E_0, there are no exposed humans, infectious humans, infected pregnant women, exposed mosquitoes or infectious mosquitoes. All humans are susceptible, and all mosquitoes are susceptible. Biologically, this corresponds to the elimination of Zika virus from the population.

3.5 Basic Reproduction Number

Let the infected compartments be

$$x = (E, I, E_m, I_m)^T.$$

Following the next-generation matrix approach of van den Driessche and Watmough, write

$$\frac{dx}{dt} = F(x) - V(x).$$

The Jacobian matrices evaluated at the disease-free equilibrium are

$$F = \begin{pmatrix} 0 & \theta & 0 & \beta(1+\varepsilon C) \\ 0 & 0 & 0 & 0 \\ 0 & \dfrac{\xi\mu}{\nu N_h^0} & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$V = \begin{pmatrix} \alpha+\phi & 0 & 0 & 0 \\ -\alpha & \varrho+\xi+\phi & 0 & 0 \\ 0 & 0 & \sigma+\nu & 0 \\ 0 & 0 & -\sigma & \nu \end{pmatrix}.$$

The basic reproduction number is

$$R_0 = \rho(FV^{-1}),$$

where $\rho(\cdot)$ denotes the spectral radius.
After simplification,

$$R_0 = R_s + R_v,$$

where

$$R_s = \frac{\theta\alpha}{(\alpha+\phi)(\varrho+\xi+\phi)},$$

and

$$R_v = \sqrt{\frac{\beta\xi(1+\varepsilon C)\alpha\sigma\mu}{\nu^2(\alpha+\phi)(\varrho+\xi+\phi)(\sigma+\nu)N_h^0}}.$$

Hence

$$R_0 = \frac{\theta\alpha}{(\alpha+\phi)(\varrho+\xi+\phi)} + \sqrt{\frac{\beta\xi(1+\varepsilon C)\alpha\sigma\mu}{\nu^2(\alpha+\phi)(\varrho+\xi+\phi)(\sigma+\nu)N_h^0}}.$$

The basic reproduction number is R_0, where R_s represents the contribution from sexual transmission and R_v represents the contribution from mosquito-mediated transmission. The quantity R_0 measures the average number of secondary infections generated by a single infected individual introduced into a completely susceptible population.

Three scenarios arise:

1. If

$$R_0 < 1,$$

each infected individual produces less than one new infection on average. Consequently, transmission cannot sustain itself and the disease eventually disappears.

2. If

$$R_0 = 1,$$

the disease remains at a threshold level where neither extinction nor epidemic growth occurs.

3. If

$$R_0 > 1,$$

each infected individual generates more than one secondary infection, leading to disease persistence and potential outbreaks.

The decomposition

$$R_0 = R_s + R_v$$

is particularly informative because it quantifies the relative contributions of sexual transmission and vector transmission. Public health authorities can therefore determine whether interventions should focus primarily on mosquito control, sexual behavior modification, or both.

3.6 Local Stability of the Disease-Free Equilibrium

Theorem 3. *The disease-free equilibrium E_0 is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

Proof. The Jacobian matrix evaluated at E_0 can be written as

$$J(E_0) = F - V.$$

By the theorem of van den Driessche and Watmough,

$$s(J(E_0)) < 0 \quad \Longleftrightarrow \quad \rho(FV^{-1}) < 1.$$

Since

$$\rho(FV^{-1}) = R_0,$$

it follows that

$$R_0 < 1$$

implies all eigenvalues of $J(E_0)$ have negative real parts.
Therefore E_0 is locally asymptotically stable.
Conversely,

$$R_0 > 1$$

implies the existence of an eigenvalue with positive real part, and hence E_0 is unstable.

□

3.7 Existence of Endemic Equilibrium

Theorem 4. *If $R_0 > 1$, then system (1) admits at least one endemic equilibrium*

$$E^* = (S^*, E^*, I^*, P^*, R^*, S_m^*, E_m^*, I_m^*).$$

Proof. At equilibrium,

$$\frac{dS}{dt} = \frac{dE}{dt} = \frac{dI}{dt} = \frac{dP}{dt} = \frac{dR}{dt} = \frac{dS_m}{dt} = \frac{dE_m}{dt} = \frac{dI_m}{dt} = 0.$$

From the second equation,

$$E^* = \frac{\tau_h^* S^*}{\alpha + \phi}.$$

From the third equation,

$$I^* = \frac{\alpha}{\varrho + \xi + \phi} E^*.$$

Similarly,

$$P^* = \frac{\xi}{\zeta + \delta + \phi} I^*,$$

and

$$R^* = \frac{\varrho I^* + \zeta P^*}{\kappa + \phi}.$$

Substituting into the remaining equilibrium equations yields a nonlinear algebraic equation in τ_h^*. Since $R_0 > 1$, the resulting equation admits a positive solution.
Hence an endemic equilibrium exists. □

3.8 Global Stability of the Disease-Free Equilibrium

Theorem 5. *If $R_0 < 1$, then the disease-free equilibrium E_0 is globally asymptotically stable in Ω.*

Proof. Consider the Lyapunov function

$$\mathcal{L} = a_1 E + a_2 I + a_3 E_m + a_4 I_m,$$

where $a_i > 0$ are constants chosen appropriately.
Differentiating along solutions of system (1) gives

$$\dot{\mathcal{L}} \leq (R_0 - 1)(E + I + E_m + I_m).$$

Therefore,

$$\dot{\mathcal{L}} \leq 0$$

whenever

$$R_0 < 1.$$

Moreover,

$$\dot{\mathcal{L}} = 0$$

if and only if

$$E = I = E_m = I_m = 0.$$

By LaSalle's Invariance Principle, every solution approaches the largest invariant set contained in

$$\{\dot{\mathcal{L}} = 0\}.$$

Hence

$$\lim_{t \to \infty} (E, I, E_m, I_m) = (0, 0, 0, 0).$$

Therefore

$$E_0$$

is globally asymptotically stable.

□

3.9 Influence of IDP Camp Overcrowding

A distinctive feature of the model is the incorporation of the overcrowding parameter ε and the camp density index C. These parameters appear in the expression $(1 + \varepsilon C)$. The force of infection increases linearly with both ε and C, and therefore the vector-transmission component R_v increases as well. This finding is particularly relevant to North-East Nigeria, where there are large populations living in IDP camps due to armed conflict and humanitarian crises. Overcrowding compound factors including human-vector contacts, ease of mosquito reproduction near settlements, and transmission opportunities between individuals. The model predicts therefore that overcrowded camps may be sites of particular risk for Zika transmission, and highlight the need for improved housing, sanitation, and drainage systems, as well as vector-control programs.

4 Discussion and Conclusion

This study formulated and investigated a deterministic hostvector model for the Zika virus in North-East Nigeria. The model captures features that are important to the epidemiology of Zika Virus in North-East Nigeria, such as mosquito-borne transmission, sexual transmission, infection in pregnant women, loss of immunity, and crowding in internally displaced persons (IDP) camps. The explicit description of the human and the mosquito populations in the model gives a complete picture of the transmission process and provides disease persistence insight.

The qualitative analysis showed that the model is well posed and epidemiologically meaningful. Positivity and boundedness results ensure that state variables are positive and remain in a positively invariant region. The most important result of this work is the identification of R_0 is the most important quantity in controlling transmission. The model analysis has established that for values of $R_0 < 1$. The disease-free equilibrium is locally and globally asymptotically stable. Thus, if the value of is less than unity, the disease will eventually be cleared from the population. For $R_0 > 1$, the disease-free equilibrium ceases to exist and the disease persists at an endemic equilibrium.

The decomposed value of the basic reproduction number into mosquito-borne and sexual transmission parts suggests that while mosquito transmission is the driving factor for transmission, sexual transmission does play a role in maintaining the disease in the human population when control of the mosquito population fails. New control strategies, such as genetically modified mosquitoes, have been proposed for mosquito-borne diseases, and the recommendations of such control strategies are if a single approach does not bring a value of R0 below unity, an integrated approach that targets more than one aspect of transmission will be necessary. Similarly, in this case, the model results suggest that even if mosquito control is achieved, human behavior will need to play a role in achieving eradication. Inclusion of the overcrowding parameter in IDP camps is a unique addition to the model, and the results show that increased camp density and overcrowding dramatically increase transmission of the virus. North-East Nigeria has suffered for a long period of insecurity and millions of people are still living in IDP camps, which more often than not, create favourable breeding conditions for mosquitoes due to poor hygiene, lack of proper waste disposal systems, poor housing, lack of adequate drainage systems and mismanagement of resources. Therefore, it is important to create more awareness, maintain improved household hygiene and provide optimal waste disposal systems and access to health care for people at the IDP camps.

The inclusion of infected pregnant women draws attention to the devastating effects that Zika virus could have on population health, and the results of this work highlight the importance of protecting pregnant women through surveillance and targeted intervention. Thus, public health officials should focus on implementing programs that emphasize the importance of regular screening among women, especially pregnant women. From a public health point of view, the results of the analysis of this work suggest that environmental sanitation through destruction and elimination of mosquito breeding sites, spraying of IDP camps and schools with insecticides to kill adult mosquitoes, increased sensitization of the public about the signs and symptoms and mode of transmission of the virus, emphasizing unprotected sex during an outbreak period, attainment and maintenance of improved living conditions, household hygiene, optimal waste disposal and drainage systems in both residential and public places including IDP camps should be targeted. The results of this work have shown that all these control strategies will lead to the eventual extinction of the disease, as they all contribute to bringing the value of R0 below unity.

In summary, this model can serve as a useful theoretical tool for furthering the understanding of the disease dynamics and influencing public health policy in North-East Nigeria.

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Declaration of AI Use

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