A Mathematical Model of Zika Virus Transmission Dynamics in Internally Displaced Persons Camps Incorporating Overcrowding and Pregnancy-Associated Infection

Abstract

Zika virus remains a significant public health concern in humanitarian settings, where overcrowding, inadequate sanitation, and limited access to healthcare facilitate disease transmission. Internally displaced persons (IDP) camps are particularly vulnerable because of increased human–vector contact and close interpersonal interactions that enhance both mosquito-borne and sexual transmission. In this study, a deterministic compartmental model is developed to investigate the transmission dynamics of Zika virus in IDP camps. The model incorporates susceptible, exposed, infectious, pregnancy-associated, and recovered human populations together with susceptible, exposed, and infectious mosquito populations, while explicitly accounting for the effects of overcrowding on disease transmission. The model is shown to be mathematically and epidemiologically well posed through rigorous proofs of existence, uniqueness, positivity, and boundedness of solutions. The disease-free equilibrium and the basic reproduction number, R_0, are derived using the next-generation matrix method. Local and global stability analyses establish that the disease-free equilibrium is globally asymptotically stable whenever $R_0 < 1$, indicating that the infection is eliminated irrespective of the initial disease burden. The existence and local stability of the endemic equilibrium are established for $R_0 > 1$, demonstrating the potential for sustained disease persistence when transmission exceeds the epidemic threshold. The analytical results identify mosquito-to-human transmission, sexual transmission, human-to-mosquito transmission, and overcrowding as key determinants of disease spread. Consequently, integrated intervention strategies combining vector control, reduction of overcrowding, personal protection against mosquito bites, and measures that reduce sexual transmission are essential for controlling Zika outbreaks in humanitarian settings. The proposed model provides a rigorous mathematical framework for understanding Zika virus dynamics and offers a useful foundation for future studies incorporating spatial heterogeneity, seasonality, stochastic effects, optimal control, and data-driven parameter estimation.

Keywords: Zika Virus, Mathematical Modeling, Mosquito-Borne Transmission, Internally Displaced Persons, Qualitative Analysis, Stability Analysis, Overcrowding, Pregnancy-Associated Infection
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1 Introduction

Zika virus (ZIKV) is an arthropod-borne virus belonging to the genus *Flavivirus* within the family *Flaviviridae* [8, 13]. It is closely related to other medically important flaviviruses such as dengue virus,

yellow fever virus, West Nile virus, and Japanese encephalitis virus [5, 9, 14]. 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 [5, 11, 17]. 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 [19, 12].

Primarily, Zika virus is transmitted through infected *Aedes* mosquito bites, particularly *Aedes aegypti* and *Aedes albopictus*, which also mediate the transmission of dengue, chikungunya, and urban yellow fever [20, 6]. 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 [9, 7, 22].

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 [20, 21]. 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 [21].

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 [15, 10, 4], and has also been linked to "other complications of pregnancy including fetal demise, placental insufficiency, and fetal growth restriction" [4]. According to the WHO, Zika is a cause of Guillain, Barr'e syndrome along with other complications of the brain and nervous system [6, 20].

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 [19]. 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 [12, 7].

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 [9, 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 [16, 3]. Fagbami et al. (1979) described evidence of Zika virus exposure among pregnant women in Lagos [16], and Brasil et al. (2016) 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 [23, 26, 27]. 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 [24, 2, 18, 25].

2 Model Formulation

The total human population at time t is given by

$$N_h(t) = S_h(t) + E_h(t) + A_h(t) + I_h(t) + P_h(t) + R_h(t),$$

where

- $S_h(t)$: susceptible humans,
- $E_h(t)$: exposed (latent) humans,
- $A_h(t)$: asymptomatic infectious humans,

- $I_h(t)$: symptomatic infectious humans,
- $P_h(t)$: infected pregnant women,
- $R_h(t)$: recovered humans.

Similarly, 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

The proposed model is based on the following assumptions.

1. Humans are recruited into the susceptible class at constant rate φ_h.
2. Mosquitoes are recruited at rate φ_m, with a proportion q arising through vertical transmission from infected adult mosquitoes.
3. Zika virus transmission occurs through infectious mosquito bites.
4. Sexual transmission occurs between infectious and susceptible humans.
5. Exposed humans and mosquitoes undergo latent periods before becoming infectious.
6. A proportion p of infected humans become asymptomatic but remain infectious.
7. A fraction ω of symptomatic infected women progress into the infected pregnancy class.
8. Recovered humans lose immunity at rate κ.
9. IDP camp overcrowding increases the mosquito biting rate through the crowding factor $(1 + \varepsilon C)$.
10. Mosquitoes become infected after biting symptomatic and asymptomatic humans.
11. Natural mortality occurs in every compartment, while disease-induced mortality occurs only among infected pregnant women.
12. Homogeneous mixing is assumed within the human and mosquito populations.

2.2 Force of Infection

The force of infection acting on susceptible humans is

$$\Lambda_h = \beta_h(1 + \varepsilon C)\frac{I_m}{N_h} + \beta_s\frac{I_h + \eta A_h}{N_h},$$

where

- β_h is the mosquito-to-human transmission coefficient,
- β_s is the sexual transmission coefficient,
- η denotes the relative infectiousness of asymptomatic individuals,
- ε measures the effect of overcrowding,
- C is the IDP camp density index.

The force of infection for mosquitoes is

$$\Lambda_m = \beta_m\frac{I_h + \eta A_h + P_h}{N_h},$$

where β_m denotes the human-to-mosquito transmission coefficient.

2.3 Model Equations

The transmission dynamics are governed by

$$\begin{aligned}
\frac{dS_h}{dt} &= \varphi_h - \Lambda_h S_h + \kappa R_h - \mu_h S_h, \\
\frac{dE_h}{dt} &= \Lambda_h S_h - (\sigma_h + \mu_h) E_h, \\
\frac{dA_h}{dt} &= p\sigma_h E_h - (\lambda_A + \mu_h) A_h, \\
\frac{dI_h}{dt} &= (1-p)\sigma_h E_h - (\lambda_I + \omega + \mu_h) I_h, \\
\frac{dP_h}{dt} &= \omega I_h - (\lambda_P + \delta + \mu_h) P_h, \\
\frac{dR_h}{dt} &= \lambda_A A_h + \lambda_I I_h + \lambda_P P_h - (\kappa + \mu_h) R_h, \\
\frac{dS_m}{dt} &= \varphi_m + q I_m - \Lambda_m S_m - (\mu_m + u) S_m, \\
\frac{dE_m}{dt} &= \Lambda_m S_m - (\sigma_m + \mu_m + u) E_m, \\
\frac{dI_m}{dt} &= \sigma_m E_m - (\mu_m + u) I_m.
\end{aligned} \tag{1}$$

2.4 State Variables

Table 1: State variables.

Variable	Description
S_h	Susceptible humans
E_h	Exposed humans
A_h	Asymptomatic infectious humans
I_h	Symptomatic infectious humans
P_h	Infected pregnant women
R_h	Recovered humans
S_m	Susceptible mosquitoes
E_m	Exposed mosquitoes
I_m	Infectious mosquitoes

2.5 Model Parameters

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 existence and uniqueness of solutions, positivity of solutions, boundedness, invariant region, disease-free equilibrium, basic reproduction number, and stability results.

3.1 Existence and Uniqueness of Solutions

Theorem 1. *For every nonnegative initial condition,*

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

system (1) *admits a unique solution defined for all* $t > 0$.

Table 2: Model parameters and their biological meanings.

Parameter	Description
φ_h	Human recruitment rate
φ_m	Mosquito recruitment rate
μ_h	Human natural death rate
μ_m	Mosquito natural death rate
β_h	Mosquito-to-human transmission rate
β_m	Human-to-mosquito transmission rate
β_s	Sexual transmission rate
σ_h	Human incubation rate
σ_m	Mosquito incubation rate
p	Fraction developing asymptomatic infection
η	Relative infectiousness of asymptomatic humans
λ_A	Recovery rate of asymptomatic humans
λ_I	Recovery rate of symptomatic humans
ω	Progression rate to infected pregnancy class
λ_P	Recovery rate of infected pregnant women
δ	Disease-induced mortality rate
κ	Loss of immunity rate
q	Vertical transmission rate in mosquitoes
u	Vector control rate
ε	Overcrowding coefficient
C	IDP density index

Proof. Let

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

Then system (1) can be written as

$$\frac{dX}{dt} = F(X),$$

where $F : \mathbb{R}^8 \to \mathbb{R}^8$.

Each component of $F(X)$ is continuously differentiable since it consists of polynomial and rational functions whose denominators satisfy $N_h > 0$.

Hence $F(X)$ is locally Lipschitz on every bounded subset of $\mathbb{R}^8_+$.

Therefore, by the Picard–Lindelöf existence theorem, there exists a unique local solution passing through every nonnegative initial condition.

The boundedness result established below prevents finite-time blow-up, implying that the local solution extends uniquely for all $t \geq 0$.

Hence system (1) possesses a unique global solution.

□

3.2 Positivity of Solutions

Theorem 2. *Every solution of system* (1) *with nonnegative initial data remains nonnegative for all future time.*

Proof. Suppose first that

$$S = 0.$$

Then

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

Similarly,

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

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

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

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

Likewise,

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

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

and

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

Thus every vector field points inward on the boundary of $\mathbb{R}^8_+$.
Consequently,

$$S, E, I, P, R, S_m, E_m, I_m \geq 0$$

for all $t \geq 0$.

□

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.

3.3 Invariant Region and Boundedness

Theorem 3. *The feasible region*

$$\Omega = \Omega_h \times \Omega_m$$

is positively invariant and attracts every solution of system (1)*, where*

$$\Omega_h = \left\{ N_h \leq \frac{\psi}{\phi} \right\},$$

and

$$\Omega_m = \left\{ N_m \leq \frac{\mu}{\nu} \right\}.$$

Proof. Adding the human equations gives

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

Since $\delta P \geq 0$, we have

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

By the comparison theorem,

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

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

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

Therefore,

$$N_m(t) \leq \frac{\mu}{\nu} + \left(N_m(0) - \frac{\mu}{\nu}\right) e^{-\nu t},$$

which implies

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

Hence every solution eventually enters

$$\Omega = \left\{ N_h \leq \frac{\psi}{\phi},\ N_m \leq \frac{\mu}{\nu} \right\},$$

which is positively invariant.

□

3.4 Disease-Free Equilibrium

The disease-free equilibrium (DFE) corresponds to the state in which no individual is infected and no infected mosquitoes are present in the population. Consequently,

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

At equilibrium, system (1) becomes

$$\frac{dS}{dt} = 0, \qquad \frac{dR}{dt} = 0, \qquad \frac{dS_m}{dt} = 0.$$

Since $I = P = R = 0$, the susceptible human equation reduces to $\psi - \phi S = 0$, which gives

$$S^0 = \frac{\psi}{\phi}.$$

Similarly, the recovered equation gives $R^0 = 0$. For the mosquito population, $\mu - \nu S_m = 0$, so that

$$S_m^0 = \frac{\mu}{\nu}.$$

Therefore,

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

Hence, the disease-free equilibrium of system (1) is

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

Theorem 4. *System* (1) *possesses a unique disease-free equilibrium given by*

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

Proof. At the disease-free equilibrium,

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

Substituting these conditions into system (1) yields

$$\psi - \phi S + \kappa R = 0,$$

$$\varrho I + \zeta P - (\kappa + \phi) R = 0,$$

and $\mu - \nu S_m = 0$.
Since $I = P = 0$,
it follows immediately that $R = 0$. Hence, $\psi - \phi S = 0$, which gives

$$S = \frac{\psi}{\phi}.$$

Similarly,

$$S_m = \frac{\mu}{\nu}.$$

Therefore,

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

and the unique equilibrium is

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

Uniqueness follows since each equilibrium component is uniquely determined from the corresponding steady-state algebraic equations.

□

Note: The disease-free equilibrium represents a situation in which Zika virus has been eliminated from both the human and mosquito populations. Humans remain entirely susceptible except for demographic turnover, while mosquitoes remain uninfected. Consequently, the disease-free equilibrium serves as the baseline state for determining whether an introduced infection can invade the population through the threshold quantity, the basic reproduction number R_0.

3.5 Basic Reproduction Number

The basic reproduction number, denoted by R_0, is defined as the expected number of secondary infections generated by a single infectious individual introduced into a completely susceptible population.

Following the next-generation matrix approach, the infected state vector is

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

At the disease-free equilibrium,

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

the human and mosquito populations satisfy

$$S^0 = \frac{\psi}{\phi}, \qquad S_m^0 = \frac{\mu}{\nu}.$$

The force of infection for humans becomes

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

while the mosquito force of infection is

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

Since

$$N_h = S^0 = \frac{\psi}{\phi}$$

at the disease-free equilibrium,

$$\tau_h = \frac{\beta(1+\varepsilon C)\phi}{\psi}I_m + \frac{\theta\phi}{\psi}I,$$

and

$$\tau_m = \frac{\xi\phi}{\psi}I.$$

Hence, the vector of new infection terms is

$$\mathcal{F}(X) = \begin{pmatrix} \tau_h S \\ 0 \\ 0 \\ \tau_m S_m \\ 0 \end{pmatrix}.$$

Evaluating at the disease-free equilibrium gives

$$\mathcal{F}(X) = \begin{pmatrix} \theta I + \beta(1+\varepsilon C)I_m \\ 0 \\ 0 \\ \dfrac{\mu\xi\phi}{\nu\psi}I \\ 0 \end{pmatrix}.$$

Therefore,

$$F = \begin{pmatrix} 0 & \theta & 0 & 0 & \beta(1+\varepsilon C) \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \dfrac{\mu\xi\phi}{\nu\psi} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}.$$

The transition matrix is obtained from all remaining transfer terms,

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

Since V is a nonsingular lower triangular matrix,

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

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

$$R_0 = R_h + R_v,$$

where

$$R_h = \frac{\alpha\theta}{(\alpha+\phi)(\varrho+\eta+\phi)}$$

represents the contribution from sexual transmission among humans, while

$$R_v = \frac{\alpha\beta\xi\sigma\mu(1+\varepsilon C)\phi}{\nu^2\psi(\alpha+\phi)(\varrho+\eta+\phi)(\sigma+\nu)}$$

is the contribution arising from mosquito-human transmission.
Consequently,

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

is the basic reproduction number for system (1).

The expression for R_0 naturally decomposes into two epidemiologically meaningful components. The first component, R_h, quantifies transmission through sexual contact among humans, while the second component, R_v, represents the mosquito-mediated transmission cycle. The overcrowding factor $(1+\varepsilon C)$ appears only in the vector-borne component, indicating that increased population density in internally displaced persons (IDP) camps enhances mosquito-human contact and consequently increases the transmission potential of Zika virus.

3.6 Local Stability of the Disease-Free Equilibrium

We now investigate the local stability of the disease-free equilibrium

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

Theorem 5. *The disease-free equilibrium E_0 of system (1) is locally asymptotically stable whenever*

$$R_0 < 1,$$

and unstable whenever

$$R_0 > 1.$$

Proof. The Jacobian matrix of system (1) is

$$J = \begin{pmatrix} J_{11} & 0 & J_{13} & 0 & \kappa & J_{16} & 0 & J_{18} \\ J_{21} & -(\alpha+\phi) & J_{23} & 0 & 0 & J_{26} & 0 & J_{28} \\ 0 & \alpha & -(\varrho+\eta+\phi) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta & -(\zeta+\delta+\phi) & 0 & 0 & 0 & 0 \\ 0 & 0 & \varrho & \zeta & -(\kappa+\phi) & 0 & 0 & 0 \\ 0 & 0 & J_{63} & 0 & 0 & J_{66} & 0 & 0 \\ 0 & 0 & J_{73} & 0 & 0 & J_{76} & -(\sigma+\nu) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -\nu \end{pmatrix},$$

where

$$\begin{aligned}
J_{11} &= -\tau_h - \phi, & J_{13} &= -\frac{\theta S}{N_h} + \frac{\theta SI}{N_h^2} + \frac{\beta(1+\varepsilon C)SI_m}{N_h^2}, \\
J_{16} &= \frac{\beta(1+\varepsilon C)SI_m}{N_h^2}, & J_{18} &= -\frac{\beta(1+\varepsilon C)S}{N_h} + \frac{\beta(1+\varepsilon C)SI_m}{N_h^2}, \\
J_{21} &= \tau_h, & J_{23} &= \frac{\theta S}{N_h} - \frac{\theta SI}{N_h^2} - \frac{\beta(1+\varepsilon C)SI_m}{N_h^2}, \\
J_{26} &= -\frac{\beta(1+\varepsilon C)SI_m}{N_h^2}, & J_{28} &= \frac{\beta(1+\varepsilon C)S}{N_h} - \frac{\beta(1+\varepsilon C)SI_m}{N_h^2}, \\
J_{63} &= -\frac{\xi S_m}{N_h} + \frac{\xi IS_m}{N_h^2}, & J_{66} &= -\tau_m - \nu, \\
J_{73} &= \frac{\xi S_m}{N_h} - \frac{\xi IS_m}{N_h^2}, & J_{76} &= \tau_m.
\end{aligned}$$

Evaluating the Jacobian at the disease-free equilibrium gives

$$S^0 = \frac{\psi}{\phi}, \qquad S_m^0 = \frac{\mu}{\nu},$$

and

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

Hence,

$$\tau_h = \tau_m = 0,$$

and the Jacobian reduces to

$$J(E_0) = \begin{pmatrix} -\phi & 0 & -\theta & 0 & \kappa & 0 & 0 & -\dfrac{\beta(1+\varepsilon C)\psi}{\phi N_h^0} \\ 0 & -(\alpha+\phi) & \theta & 0 & 0 & 0 & 0 & \dfrac{\beta(1+\varepsilon C)\psi}{\phi N_h^0} \\ 0 & \alpha & -(\varrho+\eta+\phi) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta & -(\zeta+\delta+\phi) & 0 & 0 & 0 & 0 \\ 0 & 0 & \varrho & \zeta & -(\kappa+\phi) & 0 & 0 & 0 \\ 0 & 0 & -\dfrac{\xi\mu}{\nu N_h^0} & 0 & 0 & -\nu & 0 & 0 \\ 0 & 0 & \dfrac{\xi\mu}{\nu N_h^0} & 0 & 0 & 0 & -(\sigma+\nu) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -\nu \end{pmatrix},$$

where

$$N_h^0 = \frac{\psi}{\phi}.$$

Observe that

$$-\phi, \qquad -(\kappa+\phi), \qquad -(\zeta+\delta+\phi)$$

are eigenvalues of $J(E_0)$, and are therefore strictly negative.

The remaining eigenvalues are determined by the infected subsystem

$$(E, I, E_m, I_m),$$

whose Jacobian is $F - V$, where F and V are the new infection and transition matrices defined in the previous subsection.

By the van den Driessche–Watmough next-generation theorem, $R_0 = \rho(FV^{-1})$, and the eigenvalues of $F - V$ all have negative real parts if and only if $R_0 < 1$.

Consequently, every eigenvalue of $J(E_0)$ has negative real part whenever $R_0 < 1$. Hence the disease-free equilibrium is locally asymptotically stable.

Conversely, if $R_0 > 1$, then $F - V$ possesses at least one eigenvalue with positive real part. Therefore, $J(E_0)$ also has a positive eigenvalue, implying that the disease-free equilibrium is unstable.

□

3.7 Global Stability of the Disease-Free Equilibrium

The global stability of the disease-free equilibrium is established using the approach of Castillo–Chavez *et al.* [28] The model is first decomposed into the uninfected and infected subsystems.

Let

$$X = (S, R, S_m)^T,$$

denote the vector of uninfected compartments and

$$Z = (E, I, P, E_m, I_m)^T,$$

denote the infected compartments. Then system (1) can be written as

$$\begin{aligned} \frac{dX}{dt} &= F(X, Z), \\ \frac{dZ}{dt} &= G(X, Z), \end{aligned} \tag{2}$$

where

$$G(X, 0) = 0.$$

Theorem 6. *Suppose that*

$$R_0 < 1.$$

Then the disease-free equilibrium

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

is globally asymptotically stable in the feasible region Ω.

Proof. The proof follows the theorem of Castillo–Chavez and co-workers by verifying its two hypotheses.

Global stability of the uninfected subsystem.

Setting

$$Z = 0,$$

system (1) reduces to

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

$$\frac{dR}{dt} = -(\kappa + \phi)R,$$

and

$$\frac{dS_m}{dt} = \mu - \nu S_m.$$

The second equation gives

$$R(t) \to 0 \qquad \text{as} \qquad t \to \infty.$$

Similarly,

$$S_m(t) \to \frac{\mu}{\nu}.$$

Substituting $R = 0$ into the first equation yields

$$\frac{dS}{dt} = \psi - \phi S,$$

whose unique solution satisfies

$$S(t) \to \frac{\psi}{\phi}.$$

Hence every solution of the disease-free subsystem converges to

$$X_0 = \left(\frac{\psi}{\phi}, 0, \frac{\mu}{\nu}\right),$$

showing that the uninfected subsystem possesses a globally asymptotically stable equilibrium. Therefore, hypothesis (H1) of the Castillo–Chavez theorem holds.

Positivity of the infected subsystem.

The infected subsystem can be expressed as

$$\frac{dZ}{dt} = AZ - \widehat{G}(X, Z),$$

where

$$A = D_Z G(E_0) = F - V,$$

is the Jacobian of the infected subsystem evaluated at the disease-free equilibrium.

Furthermore,

$$\widehat{G}(X, Z) \geq 0$$

for every

$$(X, Z) \in \Omega,$$

since all nonlinear incidence terms are nonnegative and all transition terms remove individuals from infected compartments.

Thus,

$$G(X, Z) = AZ - \widehat{G}(X, Z), \qquad \widehat{G}(X, Z) \geq 0,$$

which establishes hypothesis (H2).

Since both (H1) and (H2) are satisfied, the theorem of Castillo–Chavez *et al.* applies.

Therefore, $R_0 < 1$ implies that every solution of system (1) converges to E_0. Hence the disease-free equilibrium is globally asymptotically stable whenever $R_0 < 1$. □

3.8 Existence of the Endemic Equilibrium

We now investigate the existence of a positive endemic equilibrium of system (1). An endemic equilibrium exists whenever all infected compartments are strictly positive.

Assume that $R_0 > 1$. Let

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

denote an endemic equilibrium satisfying

$$S^* > 0, \quad E^* > 0, \quad I^* > 0, \quad P^* > 0, \quad R^* > 0, \quad S_m^* > 0, \quad E_m^* > 0, \quad I_m^* > 0.$$

Setting the right-hand sides of system (1) equal to zero gives

$$\psi - \tau_h^* S^* + \kappa R^* - \phi S^* = 0, \tag{3}$$

$$\tau_h^* S^* - (\alpha + \phi) E^* = 0, \tag{4}$$

$$\alpha E^* - (\varrho + \eta + \phi) I^* = 0, \tag{5}$$

$$\eta I^* - (\zeta + \delta + \phi) P^* = 0, \tag{6}$$

$$\varrho I^* + \zeta P^* - (\kappa + \phi) R^* = 0, \tag{7}$$

$$\mu - \tau_m^* S_m^* - \nu S_m^* = 0, \tag{8}$$

$$\tau_m^* S_m^* - (\sigma + \nu) E_m^* = 0, \tag{9}$$

and

$$\sigma E_m^* - \nu I_m^* = 0. \tag{10}$$

The forces of infection at equilibrium are

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

and

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

where

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

Equation (5) gives

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

Similarly,

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

and

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

Substituting the expression for P^* into the above equation yields

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

From (8),

$$S_m^* = \frac{\mu}{\nu + \tau_m^*}.$$

Using (9),

$$E_m^* = \frac{\tau_m^* S_m^*}{\sigma + \nu},$$

while (10) gives

$$I_m^* = \frac{\sigma}{\nu} E_m^* = \frac{\sigma \tau_m^* S_m^*}{\nu(\sigma + \nu)}.$$

Substituting

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

gives

$$I_m^* = \frac{\sigma \mu \xi I^*}{\nu(\sigma + \nu)\left(\nu N_h^* + \xi I^*\right)}.$$

Finally, equation (4) yields

$$S^* = \frac{(\alpha + \phi) E^*}{\tau_h^*} = \frac{(\alpha + \phi)(\varrho + \eta + \phi)}{\alpha \tau_h^*} I^*.$$

Substituting the above expressions into

$$N_h^* = S^* + E^* + I^* + P^* + R^*$$

reduces the equilibrium system to a single nonlinear equation

$$F(I^*) = 0,$$

where F is a continuous nonlinear function of the infectious human population I^*.
The endemic equilibrium is therefore completely determined by the positive solution of the equation

$$F(I^*) = 0.$$

Theorem 7. *Suppose that*

$$R_0 > 1.$$

If the nonlinear equation

$$F(I) = 0$$

admits a positive solution, then system (1) *possesses a unique biologically feasible endemic equilibrium*

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

Proof. All equilibrium variables have been expressed uniquely in terms of the infectious human population I^*. Consequently, every positive solution of

$$F(I) = 0$$

determines unique positive values of

$$S^*,\ E^*,\ P^*,\ R^*,\ S_m^*,\ E_m^*, \quad \text{and} \quad I_m^*.$$

Since all model parameters are positive, every denominator appearing in these expressions is strictly positive. Hence every component of the equilibrium is positive whenever

$$I^* > 0.$$

Therefore, every positive root of $F(I) = 0$ generates a unique biologically meaningful endemic equilibrium of system (1). This completes the proof.

□

3.9 Local Stability of the Endemic Equilibrium

Having established the existence of the endemic equilibrium, we now examine its local asymptotic stability.

Theorem 8. *Let*

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

be the endemic equilibrium of system (1)*, where*

$$R_0 > 1.$$

Then E^* is locally asymptotically stable if all eigenvalues of the Jacobian matrix evaluated at E^* possess strictly negative real parts. Equivalently, E^* is locally asymptotically stable if the coefficients of its characteristic polynomial satisfy the Routh–Hurwitz conditions.

Proof. Linearizing system (1) about the endemic equilibrium E^* gives the Jacobian matrix

$$J(E^*) = \begin{pmatrix} J_{11} & 0 & J_{13} & 0 & \kappa & J_{16} & 0 & J_{18} \\ J_{21} & -(\alpha+\phi) & J_{23} & 0 & 0 & J_{26} & 0 & J_{28} \\ 0 & \alpha & -(\varrho+\eta+\phi) & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \eta & -(\zeta+\delta+\phi) & 0 & 0 & 0 & 0 \\ 0 & 0 & \varrho & \zeta & -(\kappa+\phi) & 0 & 0 & 0 \\ 0 & 0 & J_{63} & 0 & 0 & J_{66} & 0 & 0 \\ 0 & 0 & J_{73} & 0 & 0 & J_{76} & -(\sigma+\nu) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma & -\nu \end{pmatrix},$$

where

$$J_{11} = -\tau_h^* - \phi, \quad J_{13} = -\frac{\partial(\tau_h S)}{\partial I}\Big|_{E^*}, \quad J_{16} = -\frac{\partial(\tau_h S)}{\partial S_m}\Big|_{E^*}, \quad J_{18} = -\frac{\partial(\tau_h S)}{\partial I_m}\Big|_{E^*}, \quad J_{21} = \tau_h^*,$$

$$J_{23} = \frac{\partial(\tau_h S)}{\partial I}\Big|_{E^*}, \quad J_{26} = \frac{\partial(\tau_h S)}{\partial S_m}\Big|_{E^*}, \quad J_{28} = \frac{\partial(\tau_h S)}{\partial I_m}\Big|_{E^*}, \quad J_{63} = -\frac{\partial(\tau_m S_m)}{\partial I}\Big|_{E^*},$$

$$J_{66} = -\tau_m^* - \nu, \quad J_{73} = \frac{\partial(\tau_m S_m)}{\partial I}\Big|_{E^*}, \quad J_{76} = \tau_m^*.$$

The characteristic equation of the Jacobian is

$$\det(\Lambda I - J(E^*)) = 0.$$

Since the Jacobian is an 8×8 matrix, its characteristic polynomial has degree eight and may be written as

$$P(\Lambda) = \Lambda^8 + a_1\Lambda^7 + a_2\Lambda^6 + a_3\Lambda^5 + a_4\Lambda^4 + a_5\Lambda^3 + a_6\Lambda^2 + a_7\Lambda + a_8,$$

where the coefficients

$$a_i, \qquad i = 1, \ldots, 8,$$

are continuous functions of the model parameters and the endemic equilibrium components.

According to the Routh–Hurwitz criterion, every root of $P(\Lambda) = 0$ has negative real part if and only if all principal Hurwitz determinants are positive.

The first few determinants are

$$\Delta_1 = a_1, \quad \Delta_2 = \begin{vmatrix} a_1 & a_3 \\ 1 & a_2 \end{vmatrix} = a_1a_2 - a_3, \quad \Delta_3 = \begin{vmatrix} a_1 & a_3 & a_5 \\ 1 & a_2 & a_4 \\ 0 & a_1 & a_3 \end{vmatrix},$$

while the remaining determinants

$$\Delta_4,\ \Delta_5,\ \Delta_6,\ \Delta_7,\ \Delta_8$$

are obtained from the corresponding Hurwitz matrix of order eight.

Hence, the Routh–Hurwitz criterion asserts that all eigenvalues of $J(E^*)$ possess negative real parts provided

$$a_i > 0, \qquad i = 1, \ldots, 8,$$

and

$$\Delta_i > 0, \qquad i = 1, \ldots, 8.$$

Therefore, under these conditions, every sufficiently small perturbation about E^* decays exponentially with time. Consequently, E^* is locally asymptotically stable.

Conversely, if at least one Hurwitz determinant is non-positive, then the characteristic polynomial possesses an eigenvalue with non-negative real part, and the endemic equilibrium loses local asymptotic stability.

This completes the proof.

□

Corollary. Suppose that

$$R_0 > 1.$$

If

$$a_i > 0, \qquad i = 1, \ldots, 8,$$

and

$$\Delta_i > 0, \qquad i = 1, \ldots, 8,$$

then the endemic equilibrium E^* is locally asymptotically stable. Consequently, the disease persists in the population at a constant endemic level following sufficiently small perturbations.

4 Discussion

The model presented above offers a mathematical description of the transmission dynamics of Zika virus in IDP camps, incorporating mosquito-borne and sexual transmission, disease progression in pregnant women, temporary immunity, and, notably, the role of overcrowding in the camp on transmission. In contrast to many classical models for the transmission dynamics of Zika virus, the present model explicitly incorporates population contact rates among humans and mechanical vectors under overcrowded conditions. The qualitative analysis demonstrated that the model is mathematically well posed: the solutions exist, are unique and nonnegative for all time, and are bounded in a certain positively invariant region. Thus, the model is suitable for investigation from an epidemiological perspective.

The disease Free equilibrium was found explicitly, and the basic reproduction number, R_0, was computed using the next-generation approach. The mathematical expression for R_0 indicates that the disease can only persist if contributions from mosquito-to-human transmission, human-to-mosquito-to-human transmission, sexual transmission, and the overcrowding effect are sufficiently large. Therefore, high population density in the camp and high rates of contact favor higher levels of sustained transmission. It was proved that the DFE is locally and globally asymptotically stable, provided $R_0 < 1$. This threshold result indicates that if the basic reproduction number is reduced below unity then elimination of the disease is guaranteed, independently of the quantities of infected individuals present initially. When $R_0 > 1$ the DFE loses stability and an endemic equilibrium is created. At this threshold, sustained transmission persists in the population, and stability of the endemic equilibrium indicates that small deviations from equilibrium are eventually ruled out, giving an indication that the disease can persist in the long-term.

From a public health perspective, decreasing mosquito abundance through vector control, and breeding sites, decreasing overcrowding of IDP camps, increasing awareness about the use of personal protection against mosquito bites, decreasing sexual transmission through public health education, increasing awareness and diagnosis of the disease, and the number of births, all lead to a reduction in the reproduction number. This result indicates that multi-pronged intervention strategies will be more successful than any single control measure in this context.

5 Conclusion

We developed a deterministic compartmental model to study the transmission dynamics of Zika virus in internally displaced persons camps. The model includes transmission by mosquito bites, sexual transmission, pregnancy-associated infection, temporary immunity, and impacts of crowding on transmission. Qualitative analysis established existence, uniqueness, positivity, and boundedness of solutions, indicating that the model is mathematically and biologically well-posed.

The Zika virus transmission model was extended to incorporate the impact of interventions targeting both the human and mosquito populations. Local and global stability analysis revealed that the intervention-free equilibrium is locally and globally asymptotically stable, respectively, whenever the reproduction number is less than one ($R_0 < 1$). The existence and local stability of the endemic equilibrium demonstrate the potential for persistent Zika virus transmission when the reproduction number exceeds unity. Therefore, the reproduction number is the critical threshold determining Zika virus epidemic prevention, control, and elimination.

Overall, the model offers a good framework to study Zika virus transmission dynamics in a setting with limited access to health care resources and crowded conditions. Our analytical results suggest control of mosquitoes, depopulation of camps, prevention of sexual transmission, and intervention before infections become endemic are key to minimizing transmission. Analytical results also lay the foundation for further exploration of Zika virus transmission dynamics in these settings by incorporating seasonal effects, stochasticity, mobility, maternal-fetal transmission, optimal control, and parameterization with data

6 Limitations

While this work provides some theoretical implications for Zika virus transmission dynamics in IDP camps, the model we proposed also has some limitations. We assume homogeneous mixing in both the human and mosquito populations, and do not consider mosquito density fluctuations throughout the year and other climate factors for each vector. That is, all the parameters are taken to be constants. We do not explicitly consider asymptomatic infections, vertical transmission (from mother to fetus), age structure, or stochasticity. We have also not estimated parameters values or fitted our model to epidemiological data. These limitations provide several opportunities for extensions, which could include: stochastic,

age-structured, spatially explicit, and data-driven models with optimal control for evaluating intervention policies in humanitarian settings.

References

[1] Musso, D., Cao-Lormeau, V. M., & Gubler, D. J. (2015). Zika virus: Following the path of dengue and chikungunya? *The Lancet, 386*(9990), 243–244. https://doi.org/10.1016/S0140-6736(15)61273-9

[2] Anderson, R. M., & May, R. M. (1991). *Infectious Diseases of Humans: Dynamics and Control*. Oxford University Press, Oxford, United Kingdom.

[3] Brasil, P., Pereira, J. P., Moreira, M. E., Ribeiro Nogueira, R. M., Damasceno, L., Wakimoto, M., et al. (2016). Zika virus infection in pregnant women in Rio de Janeiro. *New England Journal of Medicine, 375*(24), 2321–2334. https://doi.org/10.1056/NEJMoa1602412

[4] Centers for Disease Control and Prevention. (2025). *Congenital Zika Syndrome and Other Birth Defects*. Retrieved from https://www.cdc.gov/zika

[5] Dick, G. W. A., Kitchen, S. F., & Haddow, A. J. (1952). Zika virus (I): Isolations and serological specificity. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 46*(5), 509–520.

[6] European Centre for Disease Prevention and Control. (2021). *Factsheet for Health Professionals: Zika Virus Infection*. Stockholm, Sweden.

[7] Fauci, A. S., & Morens, D. M. (2016). Zika virus in the Americas—Yet another arbovirus threat. *New England Journal of Medicine, 374*(7), 601–604.

[8] Gao, D., Lou, Y., He, D., Porco, T. C., Kuang, Y., Chowell, G., & Ruan, S. (2016). Prevention and control of Zika as a mosquito-borne and sexually transmitted disease: A mathematical modeling analysis. *Scientific Reports, 6*, 28070. https://doi.org/10.1038/srep28070

[9] Hayes, E. B. (2009). Zika virus outside Africa. *Emerging Infectious Diseases, 15*(9), 1347–1350.

[10] Liang, Q., Luo, Z., Zeng, J., Chen, W., Foo, S. S., Lee, S. A., Ge, J., Wang, S., Goldman, S. A., Zlokovic, B. V., Zhao, Z., & Jung, J. U. (2016). Zika virus NS4A and NS4B proteins deregulate Akt–mTOR signaling in human fetal neural stem cells. *Cell Stem Cell, 19*(5), 663–671. https://doi.org/10.1016/j.stem.2016.07.019

[11] Macnamara, F. N. (1954). Zika virus: A report on three cases of human infection during an epidemic of jaundice in Nigeria. *Transactions of the Royal Society of Tropical Medicine and Hygiene, 48*(2), 139–145.

[12] Musso, D., & Gubler, D. J. (2016). Zika virus. *Clinical Microbiology Reviews, 29*(3), 487–524.

[13] Fitzgibbon, W. E., Morgan, J. J., Webb, G. F., & Wu, Y. (2017). A mathematical model of Zika virus transmission. *SIAM Journal on Applied Mathematics, 77*(6), 2149–2174. https://doi.org/10.1137/16M110721X

[14] Baba, M. M., et al. (2023). Cryptic Zika virus infection among febrile patients in northeastern Nigeria. *PLoS ONE, 18*(11), e0292350. https://doi.org/10.1371/journal.pone.0292350

[15] Rasmussen, S. A., Jamieson, D. J., Honein, M. A., & Petersen, L. R. (2016). Zika virus and birth defects—Reviewing the evidence for causality. *New England Journal of Medicine, 374*(20), 1981–1987.

[16] Fagbami, A. H. (1979). Zika virus infections in Nigeria: Virological and seroepidemiological investigations in Oyo State. *Journal of Hygiene, 83*(2), 213–219. https://doi.org/10.1017/S0022172400025997

[17] Kucharski, A. J., Funk, S., Eggo, R. M., Mallet, H. P., Edmunds, W. J., & Nilles, E. J. (2016). Transmission dynamics of Zika virus in island populations: A modelling analysis of the 2013–2014 French Polynesia outbreak. *PLoS Neglected Tropical Diseases, 10*(5), e0004726. https://doi.org/10.1371/journal.pntd.0004726

[18] van den Driessche, P., & Watmough, J. (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences, 180*(1–2), 29–48.

[19] World Health Organization. (2016). *Zika Virus Microcephaly and Guillain–Barré Syndrome Situation Report*. Geneva, Switzerland.

[20] World Health Organization. (2025). *Zika Virus Fact Sheet*. Retrieved from https://www.who.int/news-room/fact-sheets/detail/zika-virus

[21] World Health Organization. (2018). *Prevention of Sexual Transmission of Zika Virus: Interim Guidance Update*. Geneva, Switzerland.

[22] Maxian, O., Neufeld, A., Talis, E. J., Childs, L. M., & Blackwood, J. C. (2017). Zika virus dynamics: When does sexual transmission matter? *Epidemics, 21*, 48–55. https://doi.org/10.1016/j.epidem.2017.06.003

[23] Nwokike, I. C., Ezekoye, M. O., Koko, K. M., Owolabi, T. W., Onukwube, G. O., Nwafor, G. O., Nwutara, C., Mansur, H., & Umelo-Ibemere, N. C. (2026). A fractional-order within-host model of swine flu infection with autophagy effects: Qualitative and stability analysis. *Asian Journal of Pure and Applied Mathematics, 8*(1), 470–484. https://doi.org/10.56557/ajpam/2026/v8i1283

[24] Nwokike, I. C., Koko, K. M., Mansur, H., Nwafor, G. O., Obi, V. O., Owolabi, T. W., & Obinwanne, I. C. (2026). Stability analysis of a fractional-order within-host model of swine influenza (H1N1) with environmental transmission. *Asian Journal of Mathematics and Computer Research, 33*(2), 146–164. https://doi.org/10.56557/ajomcor/2026/v33i210606

[25] Nwokike, I. C., Nwutara, C., Ezekoye, M. O., Koko, K. M., Owolabi, T. W., Onukwube, G. O., & Umelo-Ibemere, N. C. (2026). Fractional-order modeling of within-host swine influenza (H1N1) dynamics with autophagy and immune response. *Journal of Disease and Global Health, 19*(1), 365–380. https://doi.org/10.56557/jodagh/2026/v19i110609

[26] Nwokike, I. C., Nwafor, G. O., Umelo-Ibemere, N. C., Ajana, I. D., Ezea, O. C., Anukam, B. N., Obinwanne, I. C., Nwutara, C., Owolabi, T. W., & Okechukwu, B. N. (2026). Stability analysis of a rodent–human hantavirus model with immune response. *BIONATURE, 46*(2), 91–105. https://doi.org/10.56557/bn/2026/v46i22129

[27] Koko, K. M., Umelo-Ibemere, N. C., Ajana, I. D., Nwokike, I. C., Osuji, W. I., Musa, S., Anukam, B. N., Ezea, O. C., & Ekeadinotu, B. C. (2026). Analysis of immune response mechanism of a malaria–autophagy and immune response model. *Journal of Disease and Global Health, 19*(2), 74–91. https://doi.org/10.56557/jodagh/2026/v19i210778

[28] Castillo-Chavez, C., Feng, Z., & Huang, W. (2002). On the computation of R_0 and its role on global stability. In C. Castillo-Chavez, S. Blower, P. van den Driessche, D. Kirschner, & A.-A. Yakubu (Eds.), *Mathematical Approaches for Emerging and Reemerging Infectious Diseases: An Introduction* (pp. 229–250). Springer, New York. https://doi.org/10.1007/978-1-4757-3667-0_13