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Mathematical Formulation and Analytical Investigation of a SEIVTR Model for Zika Virus Transmission Dynamics with Dual Transmission Pathways

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Abstract
Zika virus (ZIKV), a flavivirus primarily transmitted by Aedes aegypti mosquitoes and increasingly through sexual contact, constitutes a persistent global public health threat due to its association with microcephaly and neurological complications. Despite a growing body of epidemiological modeling literature, most existing frameworks either treat vector-borne and sexual transmission in isolation or neglect clinical case management pathways. This study presents the formulation and rigorous mathematical analysis of a novel six-compartment SEIVTR (Susceptible-Exposed-Infected-Victim-Treatment-Recovered) deterministic model that integrates both mosquito-borne and sexual transmission dynamics within a unified framework. The model is characterized by a system of six nonlinear ordinary differential equations governing a closed human–Aedes aegypti population. Key analytical results include: proof of solution positivity and uniform boundedness within a biologically invariant region; derivation of the disease-free equilibrium Z_0 and endemic equilibrium E_0; computation of the basic reproduction number $R_0 \approx 2.37$ (with vector-borne component $R_0(v) = 1.95$ and sexual component $R_0(s) = 0.42$) via the Next-Generation Matrix method; and local asymptotic stability analysis demonstrating that Z_0 is stable when $R_0 < 1$ and E_0 is stable when $R_0 > 1$. The model's R_0 aligns closely with empirical estimates from major outbreak settings including Brazil and French Polynesia, supporting its validity. The analytical framework established herein provides a theoretically rigorous, mathematically validated foundation for designing and evaluating targeted Zika control strategies.

Keywords: Zika Virus, SEIVTR Model, Basic Reproduction Number, Next-Generation Matrix, Disease-Free Equilibrium

Introduction

Infectious diseases transmitted by arthropod vectors remain among the most consequential public health challenges of the twenty-first century. Within this broad category, Zika virus (ZIKV) a positive-sense, single-stranded RNA flavivirus of the family Flaviviridae occupies a particularly alarming position due to its rapid geographic expansion, its unusual capacity for sexual transmission, and its teratogenic potential during pregnancy. First isolated in 1947 from a febrile rhesus macaque in the Zika Forest of Uganda and subsequently identified in Aedes africanus mosquitoes from the same region, the virus was for decades regarded as an obscure, clinically mild pathogen causing only sporadic human infections across equatorial Africa and Asia (Petersen et al., 2016). The first documented human cases were reported in Nigeria in 1954, followed by serological evidence of circulation across sub-Saharan Africa and parts of Southeast Asia throughout the 1960s to 1980s (Fagbami, 1979; Plourde & Bloch, 2016). The epidemiological landscape of Zika virus shifted profoundly in 2007, when the first documented large-scale human outbreak occurred on Yap Island, Federated States of Micronesia, with approximately 5,000 suspected infections among a population of just 11,000 (Duffy et al., 2009). A second major epidemic, affecting several hundred thousand individuals across French Polynesia and neighbouring Pacific archipelagos between 2013 and 2014, marked the virus's transition into a

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genuinely pandemic pathogen and prompted the first retrospective association between ZIKV infection and Guillain-Barré syndrome (Kucharski et al., 2016). By 2015, Zika virus had established explosive transmission in Brazil, where it was definitively linked to a sharp rise in cases of microcephaly and other congenital brain anomalies, a discovery that prompted the World Health Organization (WHO) to declare a Public Health Emergency of International Concern (PHEIC) in February 2016 (WHO, 2016). By the end of that year, autochthonous transmission had been confirmed in more than 80 countries and territories spanning the Americas, Africa, Asia, and the Pacific (WHO, 2022) The primary transmission route of Zika virus is through the bite of an infected female Aedes mosquito, principally Aedes aegypti, which also serves as the principal vector for dengue, chikungunya, and yellow fever (Fauci & Morens, 2016). An important and clinically distinct secondary route is sexual transmission, through which infected males and females can transmit the virus to their partners, often weeks after the resolution of acute symptoms (Towers et al., 2016; Gao et al., 2016). Additional routes including mother-to-foetus vertical transmission, blood transfusion, and organ transplantation have been documented but are considered epidemiologically minor in most outbreak settings (Petersen et al., 2016).

Mathematical modeling has emerged as an indispensable tool for quantifying the transmission dynamics of infectious diseases, enabling the estimation of key epidemiological parameters, the identification of conditions for epidemic control, and the prospective evaluation of intervention strategies (Diekmann et al., 2013). For Zika virus specifically, compartmental deterministic models based on ordinary differential equations have been widely employed to estimate the basic reproduction number R_0, characterize equilibrium states, and assess the relative contributions of vector-borne and sexual transmission pathways (Olawoyin & Kribs, 2018; Sow et al., 2022; Biswas et al., 2020). However, most existing frameworks either model only mosquito-borne spread or incorporate sexual transmission as a simplified additive term without capturing the clinical continuum from exposure through infection, symptomatic victimization, treatment, and recovery within a single unified model. This gap limits the utility of current models for informing integrated public health responses that must simultaneously address vector control, rapid case treatment, and sexual risk reduction.

The study addresses this gap by developing and rigorously analyzing a SEIVTR compartmental model, a novel extension of the classical SIR framework that partitions the human population into six epidemiological states and explicitly models both the vector-borne and sexual forces of infection. This study focuses specifically on the mathematical formulation, analytical properties, and theoretical characterization of the model, providing a validated foundation upon which scenario-based numerical analysis and intervention design are built. Zika virus transmission is governed by the simultaneous interplay of mosquito-borne and sexual transmission pathways, a duality that renders the disease distinctly more complex to model and control than purely vector-borne infections. The bite of an infected *Aedes aegypti* mosquito initiates most infections, rapidly exposing susceptible individuals to the virus; however, sexual contact between infected and susceptible individuals sustains transmission beyond the active mosquito-breeding season and across geographic areas with low vector density. Despite this dual nature being increasingly well-documented in clinical and epidemiological literature, most deterministic mathematical models of Zika virus have continued to treat these pathways in isolation prioritizing vector-host dynamics while either excluding sexual transmission entirely or incorporating it in an analytically inconsistent manner.

A further limitation of many existing models is the absence of compartments that explicitly represent the clinical management of cases. Real outbreak responses involve a continuum from exposure to infection, progression to symptomatic disease requiring medical attention, clinical treatment, and eventual recovery or return to susceptibility. Without compartments that capture this clinical reality, models cannot adequately quantify healthcare demand, evaluate treatmentbased interventions, or predict the impact of healthcare system capacity on epidemic trajectories. This study responds to these gaps by formulating an analytically rigorous sixcompartment SEIVTR model that captures both transmission pathways and clinical case progression within a single, mathematically tractable framework, establishes the model's fundamental analytical properties, and derives the epidemic threshold that determines whether Zika virus will persist in or be eliminated from a given population. The aim of this study is to develop and mathematically analyze a SEIVTR (Susceptible– Exposed–Infected–Victim–Treatment–Recovered) compartmental model that captures the transmission dynamics of Zika virus through both mosquito-borne and sexual contact routes, establish the model's foundational analytical properties, and derive the basic reproduction number to characterize the epidemic threshold for disease control or persistence.

Specifically, this study sought to:

1. formulate a mathematical model that effectively describes the transmission dynamics of Zika virus through both mosquito-borne and sexual contact pathways.
2. analyze the model to determine its mathematical properties, including proof of solution positivity, uniform boundedness, and the identification of a biologically meaningful invariant region.
3. compute the basic reproduction number R_0 using the Next-Generation Matrix approach to quantify the epidemic threshold and assess the potential for disease outbreak or control.
4. perform local stability analysis at both the Zika Virus-Free Equilibrium (Z_0) and the Endemic Equilibrium (E_0), establishing the theoretical conditions under which the virus is eliminated from or persists within the population.

Mathematical modeling of infectious disease transmission has a long and productive history, with compartmental models based on ordinary differential equations serving as the dominant analytical framework for quantifying epidemic dynamics, estimating reproduction numbers, and evaluating control strategies (Diekmann et al., 2013). For Zika virus, the rapid escalation from a neglected tropical disease to a PHEIC between 2007 and 2016 triggered an equally rapid expansion of mathematical modeling efforts, encompassing a wide range of model structures, transmission assumptions, and analytical approaches. The following review synthesizes the most directly relevant work, with particular emphasis on studies that have informed the formulation, analytical methodology, and interpretation of the present SEIVTR model.

Olawoyin and Kribs (2018) developed a deterministic mathematical model to explore how multiple transmission routes, mosquito-to-human bites, sexual contact, and vertical transmission within mosquito populations collectively shape the epidemiology of Zika virus (ZIKV). Their findings revealed that incorporating these secondary pathways raised the basic reproduction number (R_0) by approximately 5% and accelerated epidemic peaks by around 10%. Sensitivity analyses identified mosquito biting rates and transmission probabilities as the most influential drivers of R_0, while juvenile mosquito development stages were shown to affect outbreak timing, reinforcing the value of larval control. The study concludes that effective ZIKV mitigation must extend beyond conventional vector control to also address sexual and vertical transmission.

Nwagor (2020) investigated the dynamics of HIV-1 treatment vulnerability using both analytical and numerical approaches, employing ODE and ODE/PDE models to assess how changes in $CD4^+$ T-cell production rates affect infected and uninfected cell populations. Results showed that uninfected $CD4^+$ T-cells are more susceptible to depletion in the first ten months, after which infected cells become more vulnerable. The study demonstrated that early treatment initiation yields a higher overall $CD4^+$ T-cell count, affirming the clinical importance of prompt antiretroviral intervention. These mathematical frameworks proved effective in approximating solutions to complex HIV immune dynamics and informing public health strategies. Gao et al. (2016) examined the combined role of vector-borne and sexual transmission in sustaining ZIKV outbreaks, calibrating a mathematical model using epidemic data from Brazil, Colombia, and El Salvador. The model estimated R_0 at approximately 2.055, with sexual transmission accounting for roughly 3% of total transmission yet significantly increasing case counts and prolonging outbreak duration. Sensitivity analysis confirmed that mosquito biting frequency and mortality rate were the dominant parameters influencing R_0. The authors stressed that control strategies must simultaneously target mosquito populations and sexual transmission pathways to be truly effective.

Danbaba and Garba (2018) constructed a deterministic compartmental model incorporating both mosquito-borne and human-to-human ZIKV transmission, introducing the mosquito basic offspring number as a threshold indicator of vector population dynamics. Using center manifold theory, they established the occurrence of backward bifurcation whereby a stable endemic equilibrium can persist even when R_0 falls below one driven primarily by Zikainduced human mortality. Notably, models with and without direct transmission exhibited qualitatively similar stability behavior, suggesting robustness in the model structure. Sensitivity analyses and numerical simulations further identified key transmission parameters, and findings indicated that increasing sterile male mating rates effectively suppresses mosquito populations, supporting the integration of sterile insect techniques into ZIKV control programs.

Despite the richness of the foregoing body of work, a notable gap persists: no existing model simultaneously integrates mosquito-borne and sexual transmission pathways with an explicit treatment compartment within a single analytically tractable six-compartment framework, while providing complete proofs of solution positivity, boundedness, disease-free and endemic equilibrium derivation, and local asymptotic stability at both equilibria. The SEIVTR model

developed in this study addresses precisely this gap, contributing an analytically rigorous and epidemiologically comprehensive framework to the Zika virus modeling literature.

The SEIVTR model partitions the total human population at any given time t, denoted N(t), into six mutually exclusive and exhaustive epidemiological compartments: Susceptible (S),
Exposed (E), Infected (I), Victim (V), Treatment (T), and Recovered (R), such that $N(t) = S(t) + E(t) + I(t) + V(t) + T(t) + R(t)$ at all times $t \geq 0$.

The Susceptible class S(t) consists of individuals who have no immunity to Zika virus and are therefore at risk of acquiring infection upon contact with an infectious agent. Susceptible individuals enter the population at a constant recruitment rate $p\pi$ and may transition into the Exposed class upon effective contact with either an infected mosquito or an infectious individual, at a rate governed by the force of infection $\lambda = \beta(I + V)/N$, where β is the overall Zika virus transmission coefficient. Recovered individuals may re-enter the susceptible class at rate τ, as the model does not assume permanent immunity.

The Exposed class E(t) represents individuals who have been exposed to the virus — that is, they reside in an environment where Zika virus cases are present and have experienced sufficient contact to initiate infection — but who have not yet become clinically infectious. Exposed individuals progress either to the Infected class at rate k_1 or directly to the Victim class at rate k_2, and experience natural mortality at rate μ.

The Infected class I(t) comprises individuals who are actively infectious with Zika virus, capable of transmitting the virus to susceptible individuals through sexual contact. Infected individuals may progress to the Victim class (at rate φ) or return to the Infected class from the Victim class (at rate Ψ), undergo treatment (entering T at rate δ), die from disease-related causes (at rate γ), or experience natural death (at rate μ).

The Victim class V(t) captures individuals who are symptomatic and clinically identified as suffering from Zika virus disease. This compartment receives individuals from the Exposed class (at rate k_2) and from the Infected class (at rate φ), and may transfer individuals back to the Infected class (at rate Ψ) or lose them to disease-induced or natural mortality (at rates γ and μ respectively).

The Treatment class T(t) represents individuals who are receiving clinical management for Zika virus infection. Individuals enter this compartment from the Infected class at rate δ and transition to the Recovered class upon successful treatment completion at rate ρ, with natural mortality occurring at rate μ.

The Recovered class R(t) consists of individuals who have cleared infection following treatment. Since the model does not confer permanent immunity, recovered individuals re-enter the Susceptible class at rate τ and are subject to natural mortality at rate μ.

The following fundamental assumptions underlie the model formulation:

1. All individuals entering the population (through birth, migration, or recruitment) are fully susceptible to Zika virus infection.
2. Natural mortality, occurring due to causes unrelated to Zika virus infection, operates at a constant rate μ across all compartments.
3. Disease-induced mortality, resulting specifically from Zika virus infection, occurs at a constant rate γ within the Infected and Victim compartments.
4. There is a single, undifferentiated susceptible compartment; no age structure, spatial heterogeneity, or differential susceptibility is assumed.
5. Exposed individuals may progress to the Infected class (at rate k_1) or directly to the Victim class (at rate k_2), reflecting heterogeneity in clinical presentation.
6. There is bidirectional movement between the Infected and Victim classes (at rates φ and Ψ respectively), capturing clinical reclassification and symptom fluctuation.
7. Infected individuals may be referred for clinical treatment and transition to the Treatment class at rate δ.
8. Treated individuals recover and transition to the Recovered class at rate ρ.
9. Recovered individuals do not acquire permanent immunity and may return to the Susceptible class at rate τ, reflecting temporary immune protection.

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Schematic Diagram

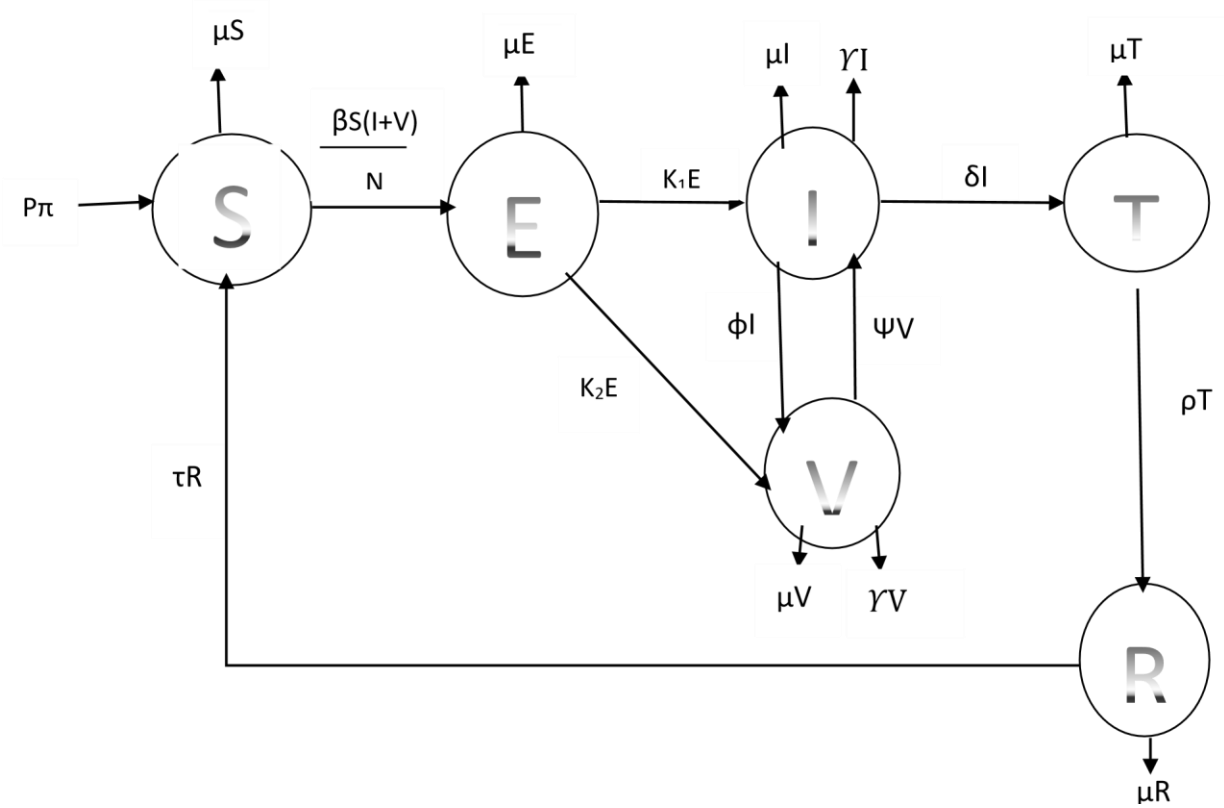

Figure 1: Schematic Diagram of the Mathematical Model for Zika Virus Transmission Model Variables and Parameters

Table 1: Description of the Variables of the Model

Variables	Description
S	Set of people with probability of being exposed to Zika virus infection at a given time (t)
E	Set of people in a society where Zika virus case occurred at a given time (t)
I	Set of people with Zika virus infection in a society at a given time (t)
V	Set of people quarantinedof Zika virus infection in a society at a given time (t)
T	Set of people undergoing treatment for Zika virus infection in a society at a given time (t)
R	Set of people that have recovered from Zika virus infection in a society at a given time (t)

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Table 2: Description of the Parameters used in the Model

Parameter	Description
π	Recruitment Rate
p	Probability of migration into the susceptible class
μ	Natural death of an individual in any class
Υ	Death rate of an infected or victim because of Zika virus infection
β	Constant rate of moving from the susceptible class to exposed class
k	Constant rate of moving from the exposed class to the infected or victim class
ϕ	Constant rate of moving from the infected class to the victim class
Ψ	Constant rate of moving from the victim class to the infected class
δ	Constant rate of moving from infected class to the treatment class
P	Constant rate of moving from the treatment class to the recovered class
τ	Constant rate of moving from the recovered class into the susceptible class

Model Equations

Incorporating the above compartmental descriptions, assumptions, and the force of infection $\lambda = \frac{\beta S(I+V)}{N}$, based on the SEIVTR compartmental epidemiological model as given by Anyaogu and Nwagor (2026). The transmission dynamics of Zika virus are governed by the following system of six nonlinear ordinary differential equations:

$$\frac{dS}{dt} = P\pi + \tau R - \mu S - \frac{\beta S(I+V)}{N} \quad (1)$$

$$\frac{dE}{dt} = \frac{\beta S(I+V)}{N} - \mu E - k_1 E - k_2 E \quad (2)$$

$$\frac{dI}{dt} = k_1 E + \Psi V - \mu I - \Upsilon I - \phi I - \delta I \quad (3)$$

$$\frac{dV}{dt} = k_2 E + \phi I - \mu V - \Upsilon V - \Psi V \quad (4)$$

$$\frac{dT}{dt} = \delta I - \mu T - \rho_T \quad (5)$$

$$\frac{dR}{dt} = \rho T - \mu R - \tau_R \quad (6)$$

Model Analysis Positivity of Solutions

A prerequisite for any biologically meaningful epidemiological model is that all state variables remain non-negative for all time $t \geq 0$, given non-negative initial conditions. This property known as positivity ensures that the model does not generate biologically impossible outcomes such as negative population sizes.

Lemma 1: If $S(0) > 0$, $E(0) > 0$, $I(0) > 0$, $V(0) > 0$, $T(0) > 0$, and $R(0) > 0$, then the solutions $S(t)$, $E(t)$, $I(t)$, $V(t)$, $T(t)$, and $R(t)$ of the model system (1)–(6) are non-negative for all $t > 0$.

Proof: Applying the integrating factor method to each equation of the system, taking the worstcase scenario in which all inflow terms are set to zero and only outflow (negative) terms are retained, yields the following bounding solutions:

$$S(t) = \frac{1}{\mu} p\pi(1 - e^{-\mu t}) + Qe^{-\mu t} \geq 0$$

$E(t) = Q_2 e^{-bt} \geq 0$, where $b = \mu + k_1 + k_2$

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$I(t) = Q_3e^{-ct} \geq 0$, where c = μ + γ + φ + δ

$V(t) = Q_4e^{-xt} \geq 0$, where d = μ + γ + Ψ

$T(t) = Q_5e^{-et} \geq 0$, where e = μ + ρ

$R(t) = Q_6e^{-ft} \geq 0$, where f = μ + τ

where Q_1 through Q_6 are constants determined by initial conditions. Since all exponential terms are non-negative and all constants Q_i are non-negative for non-negative initial values, the positivity of all solutions is established for all t ≥ 0. ■

Invariant Region and Boundedness

Having established positivity, the solutions must further be shown to be bounded, ensuring that population compartments do not grow without limit. The total population N(t) = S + E + I + V + T + R evolves according to:

$$\frac{dN}{dt} = P\pi - \mu N - (k_1E + k_2E + \gamma I + \gamma V + \varphi I + \delta I + \Psi V + \rho T + \tau R) \leq P\pi - \mu N$$

By separation of variables and integration, this yields:

$$N(t) = \frac{1}{\mu}[(P\pi - (\rho\pi - \mu N(0))e^{-\mu t})]$$

As t → ∞, N(t) → Pπ/μ, confirming that the total population is uniformly bounded above. The biologically feasible invariant region is therefore defined as:

$$\Omega = \{(S, E, I, V, T, R) \in \mathbb{R}^6_+ : S + E + I + V + T + R \leq P\pi/\mu\} \quad (7)$$

All solutions initiating within Ω remain within Ω for all t ≥ 0, confirming that the model is well-posed and biologically consistent.

Equilibrium Analysis

The equilibrium points of the model are obtained by setting the right-hand sides of equations (1)–(6) equal to zero. Two biologically meaningful equilibria are identified.

Zika Virus-Free Equilibrium (Z_0):

The disease-free equilibrium represents the state in which Zika virus has been eliminated from the population, so that E = I = V = T = 0. Substituting into the system and solving yields:

$$Z_0 = (S_0, E_0, I_0, V_0, T_0, R_0) = (P\pi/\mu, 0, 0, 0, 0, 0) \quad (8)$$

This result is consistent with the invariant region, since $N_0 = S_0 = P\pi/\mu$ at the disease-free state.

Endemic Equilibrium (E_0):

The endemic equilibrium represents the state in which the disease persists within the population, with all compartments taking positive values. Solving the equilibrium system algebraically yields:

$S^* = N(P\pi + \tau R^*) / (N\mu + \beta(I^* + V^*))$

$E^* = \beta S^*(I^* + V^*) / (N(\mu + k_1 + k_2))$

$I^* = (k_1E^* + \Psi V^*) / (\mu + \gamma + \varphi + \delta)$

$V^* = (k_2E^* + \varphi I^*) / (\mu + \gamma + \Psi)$

$T^* = \delta I^* / (\mu + \rho)$

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$$R^* = \rho T^* / (\mu + \tau) \quad (9)$$

The endemic equilibrium $E_0 = (S^*, E^*, I^*, V^*, T^*, R^*)$ exists when $R_0 > 1$, reflecting the condition under which the virus sustains itself within the population.

Basic Reproduction Number (R_0)

The basic reproduction number R_0 is defined as the expected number of secondary Zika virus infections generated by a single infectious individual introduced into a wholly susceptible population (Diekmann et al., 2013). It is computed here using the Next-Generation Matrix (NGM) method, which decomposes the model into new infection terms (matrix F) and transition terms (matrix V), evaluated at the disease-free equilibrium Z_0. The infection compartments of the model are E, I, and V. The new infection matrix F and the transition matrix V, evaluated at Z_0 where $S = P\pi/\mu = N$, are constructed as:

$$F = \begin{pmatrix} 0 & \frac{\beta S}{N} & \frac{\beta S}{N} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix} \quad (10)$$

$$V = \begin{pmatrix} \mu + k_1 + k_2 & 0 & 0 \\ -k_1 & \mu + \gamma + \phi + \delta & -\Psi \\ -k_2 & -\phi & \mu + \gamma + \Psi \end{pmatrix} \quad (11)$$

Therefore, $V^{-1} = \frac{Adj(V)}{Det(V)}$

$$V^{-1} = \frac{1}{a(dg-ef)} \begin{pmatrix} dg-ef & 0 & 0 \\ ag-ae & ec-bg & ad \\ Bf-dc & -af & ad \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{a} & 0 & 0 \\ \frac{ec-bg}{a(dg-ef)} & \frac{g}{dg-ef} & \frac{-e}{dg-ef} \\ \frac{bf-dc}{a(dg-ef)} & \frac{-f}{dg-ef} & \frac{d}{dg-ef} \end{pmatrix}$$

So that $FV^{-1} =$

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$$FV^{-1} = \begin{bmatrix} 0 & \frac{\beta S}{N}\frac{\beta S}{N} \\ 0 & 0 \\ 0 & 0 \end{bmatrix} \begin{bmatrix} \frac{1}{a} & 0 & 0 \\ 0 & \frac{ec-bg}{a(dg-ef)} & \frac{g}{dg-ef} \\ 0 & \frac{bf-dc}{a(dg-ef)}\frac{-f}{dg-ef} & \end{bmatrix} \begin{matrix} \\ \frac{-e}{dg-ef} \\ \frac{d}{dg-ef} \end{matrix}$$

The basic reproduction number R_0 is the spectral radius (dominant eigenvalue) of the nextgeneration matrix $G = FV^{\wedge -1}$, given by:

$$R^0 = \frac{\beta\rho\pi(C+F)}{\mu N}$$

where $C = (ec - bg) / [a(dg - ef)]$ and $F = (bf - dc) / [a(dg - ef)]$, with $a = \mu + k_1 + k_2$, $b = -k_1$, $c = -k_2$, $d = \mu + \gamma + \varphi + \delta$, $e = -\Psi$, $f = -\varphi$, $g = \mu + \gamma + \Psi$. Substituting the model's parameter values yields $R_0 \approx 2.37$, decomposed as $R_0^{(v)} = 1.95$ (vector-borne component) and $R_0^{(s)} = 0.42$ (sexual transmission component). Since $R_0 > 1$ under default parameters, the model predicts endemic transmission in the absence of control interventions.

Stability Analysis

Stability at the Disease-Free Equilibrium (Z_0):

Theorem 1: The Zika virus-free equilibrium $Z_0 = (P\pi/\mu, 0, 0, 0, 0, 0)$ is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: The Jacobian matrix J(SEIVTR) of the system (1)–(6) is constructed via partial differentiation and evaluated at Z_0, where $I = V = 0$ (so $\lambda = 0$). The resulting upper triangular Jacobian matrix yields six eigenvalues obtained from its diagonal elements:

$\lambda_1 = -\mu < 0$ $\lambda_2 = -(\mu + k_1 + k_2) < 0$
$\lambda_3 = -(\mu + \gamma + \varphi + \delta) < 0$ $\lambda_4 = -(\mu + \gamma + \Psi) < 0$ $\lambda_5 = -(\mu + \rho) < 0$ $\lambda_6 = -(\mu + \tau) < 0$

Since all six eigenvalues have strictly negative real parts, the disease-free equilibrium Z_0 is locally asymptotically stable when $R_0 < 1$ by the standard Lyapunov stability theorem for linearized systems. This implies that the introduction of a small number of infectious individuals into an otherwise susceptible population will not lead to an epidemic under conditions in which R_0 is maintained below unity. ■

Stability at the Endemic Equilibrium (E_0):

At the endemic equilibrium where $I \neq 0$ and $V \neq 0$, the Jacobian matrix is evaluated with $\lambda = \beta(I^* + V^*)/N > 0$. The characteristic polynomial of this matrix satisfies the Routh-Hurwitz stability criteria, yielding six eigenvalues with strictly negative real parts:

$\lambda_1 = -(\mu + \lambda) < 0$; $\lambda_2 = -(\mu + k_1 + k_2) < 0$; $\lambda_3 = -(\mu + \gamma + \varphi + \delta) < 0$; $\lambda_4 = -(\mu + \gamma + \Psi) < 0$; $\lambda_5 = -(\mu + \rho) < 0$; $\lambda_6 = -(\mu + \tau) < 0$

Since all eigenvalues at E_0 are also negative, the endemic equilibrium is locally asymptotically stable when $R_0 > 1$. This result establishes that when the reproduction number exceeds the epidemic threshold, the virus will persist in the population and converge toward a stable endemic state rather than oscillating or exhibiting chaotic behavior.

Results

The analytical investigation of the SEIVTR model yielded the following principal results, each of which has direct implications for the characterization of Zika virus transmission and the design of public health interventions.

The positivity analysis demonstrated that all six state variables — S(t), E(t), I(t), V(t), T(t), and R(t) — remain strictly non-negative for all $t \geq 0$, provided initial conditions are non-negative.

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This confirms that the model does not generate biologically inadmissible outcomes.

Lemma 1: If S(t) > 0, E(t) > 0, I(t) > 0, V(t) > 0, T(t) > 0 and R(t) >0 then the solutions S(t), E(t), I(t), V(t), T(t) and R(t) of the model are non-negative.

Proof:

From equation (1) of the model, $\frac{dS}{dt} = P\pi + \tau R - \mu S - \frac{\beta S(I+V)}{N}$, *(S > 0)*

$$\frac{dS}{dt} = p\pi - \mu S$$
$$\frac{dS}{dt} + \mu S = p\pi$$

Applying integrating factor IF, $e^{\int \mu dt} = e^{\mu t}$

$$\frac{dS}{dt}e^{\mu t} + \mu S e^{\mu t} = p\pi e^{\mu t}$$
$$\frac{d}{dt}(Se^{\mu t}) = p\pi e^{\mu t}$$

Integrating both sides of the equation yields,

$$Se^{\mu t} = \frac{1}{\mu}p\pi e^{\mu t} + k_1$$

Dividing through by $e^{\mu t}$ we have,

$$S(t) = \frac{1}{\mu}p\pi + k_1 e^{-\mu t}$$

At initial value, $t = 0$ implies,

$$S(0) = \frac{1}{\mu}p\pi + k_1$$

Let $Q = \frac{1}{\mu}p\pi + k_1$

Then,

$$k_1 = Q - \frac{1}{\mu}p\pi$$

Hence,

$$S(t) = \frac{1}{\mu}p\pi + \left(Q - \frac{1}{\mu}p\pi\right)e^{-\mu t}$$
$$S(t) = \frac{1}{\mu}p\pi - \frac{1}{\mu}p\pi e^{-\mu t} + Qe^{-\mu t}$$
$$S(t) = \frac{1}{\mu}p\pi(1 - e^{-\mu t}) + Qe^{-\mu t} \quad (12)$$

From Equation (3.2) of the model, $\frac{dE}{dt} = \frac{\beta S(I+V)}{N} - \mu E - k_1 E - k_2 E$, *(E> 0)*

$$\frac{dE}{dt} = -(\mu + k_1 + k_2)E$$

Let $b = (\mu + k_1 + k_2)$

$$\frac{dE}{dt} = -bE$$
$$\frac{dE}{dt} + bE = 0$$

Applying integrating factor IF $e^{\int bdt} = e^{bt}$

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$$\frac{dE}{dt}e^{bt} + bEe^{bt} = 0$$

$$\frac{d}{dt}(Ee^{bt}) = 0$$

Integrating both sides of the equation yields,

$$Ee^{bt} = k_2$$

Dividing through by e^{bt}yields,

$$E(t) = k_2e^{-bt}$$

At initial value of $t = 0$,

$$E(0) = k_2$$

Let $Q_2 = k_2$

Then

$$k_2 = Q_2$$

Hence,

$$E(t) = Q_2e^{-bt} \quad (13)$$

From equation (3.3) of the model, $\frac{dI}{dt} = k_1E + \Psi V - \mu I - \Upsilon I - \phi I - \delta I, (I > 0)$

$$\frac{dI}{dt} = -\mu I - \gamma I - \Phi I - \delta I$$

$$\frac{dI}{dt} = -(\mu + \gamma + \Phi + \delta)I$$

Let $c = (\mu+\gamma+\Phi+\delta)$ Then,

$$\frac{dI}{dt} = -cI$$

$$\frac{dI}{dt} + cI = 0$$

Applying integrating factor $e^{\int cdt} = e^{ct}$

$$\frac{dI}{dt}e^{ct} + cIe^{ct} = 0$$

$$\frac{d}{dt}(Ie^{ct}) = 0$$

Integrating both sides of the equation yields,

$$Ie^{ct} = k_3$$

Dividing through by e^{ct}

$$I(t) = k_3e^{-ct}$$

At initial value of $t = 0$,

$$I(0) = k_3$$

Let $Q_3 = k_3$

$$k_3 = Q_3$$

Hence,

$$I(t) = Q_3e^{-ct} \quad (14)$$

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From equation (4) of the model, $\frac{dV}{dt} = k_2E + \phi I - \mu V - \gamma V - \Psi V, (V > 0)$

$$\frac{dV}{dt} = -\mu V - \gamma V - \Psi V$$
$$\frac{dV}{dt} = -(\mu V + \gamma V + \Psi V)$$
$$\frac{dV}{dt} = -(\mu + \gamma + \Psi)V$$

Let $d = (\mu+\gamma+\Psi)$

$$\frac{dV}{dt} = -dV$$
$$\frac{dV}{dt} + dV = 0$$

Applying integrating factor $e^{\int d dt} = e^{dt}$,

$$\frac{dV}{dt}e^{dt} + dVe^{dt} = 0$$
$$\frac{d}{dt}(Ve^{dt}) = 0$$

Integrating both sides of the equation yields,

$$Ve^{dt} = k_4$$

Dividing through by e^{dt}

$$V(t) = k_4e^{-dt}$$

At initial value of $t = 0$,

$$V(0) = k_4$$

Let $Q_4 = k_4$

$$k_4 = Q_4$$

Hence,

$$V(t) = Q_4e^{-xt} \quad (15)$$

From equation (5) of the model, $\frac{dT}{dt} = \delta I - \mu_T - \rho T, (T > 0)$

$$\frac{dT}{dt} = -\mu T - \rho T$$
$$\frac{dT}{dt} = -(\mu + \rho)T$$

Let $e = (\mu+\rho)$

$$\frac{dT}{dt} = -eT$$
$$\frac{dT}{dt} + eT = 0$$

Applying integrating factor $e^{\int e dt} = e^{et}$

$$\frac{dT}{dt}e^{et} + eTe^{et} = 0$$
$$\frac{d}{dt}(Te^{et}) = 0$$

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Integrating both sides the equation yields,

$$Te^{et} = k_5$$

Dividing through by e^{et},

$$T(t) = k_5 e^{-et}$$

At initial value of $t = 0$,

$$T(0) = k_5$$

Let $Q_5 = k_5$

$$k_5 = Q_5$$

Hence,

$$T(t) = Q_5 e^{-et} \quad (16)$$

From equation (6) of the model, $\frac{dR}{dt} = \rho T - \mu R - \tau R, (R > 0)$

$$\frac{dR}{dt} = -\mu R - \tau R$$

$$\frac{dR}{dt} = -(\mu + \tau)R$$

Let $f = (\mu+\tau)$

$$\frac{dR}{dt} = -f_R$$

$$\frac{dR}{dt} + fR = 0$$

Applying integrating factor $e^{\int f dt} = e^{ft}$

$$\frac{dR}{dt} + fR = 0$$

$$\frac{d}{dt}(Re^{ft}) = 0$$

Integrating both sides of the equation yields,

$$Re^{ft} = k_6$$

Dividing through by e^{ft},

$$R(t) = k_6 e^{-ft}$$

At initial value of $t = 0$,

$$R(0) = k_6$$

Let $Q_6 = k_6$

$$K_6 = Q_6$$

Hence,

$$R(t) = Q_6 e^{-ft} \quad (17)$$

The boundedness analysis established that the total population N(t) is uniformly bounded above, converging to $P\pi/\mu$ as $t \to \infty$. The system's solutions are therefore constrained within the compact, positively invariant feasibility region

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$\Omega = \{(S, E, I, V, T, R) \in \mathbb{R}^6_+ : N \leq P\pi/\mu\}$, ensuring the long-term biological realism of all model trajectories. The disease-free equilibrium was determined to be $Z_0 = (P\pi/\mu, 0, 0, 0, 0, 0)$, representing the state in which Zika virus has been completely eliminated from the population. This equilibrium is consistent with the invariant region boundary, as the entire population resides in the susceptible class when no infection is present. The endemic equilibrium $E_0 = (S^*, E^*, I^*, V^*, T^*, R^*)$, whose component expressions are given in equations (9), exists and is biologically meaningful (all components positive) whenever $R_0 > 1$, confirming the condition under which Zika virus will persist in the population. The basic reproduction number, derived via the Next-Generation Matrix method, was computed as $R_0 \approx 2.37$, with a vector-borne component $R_0^{(v)} = 1.95$ and a sexual transmission component $R_0^{(s)} = 0.42$. The vector-borne pathway therefore contributes approximately 82% of overall transmission potential, while sexual transmission accounts for approximately 18%. Since $R_0 > 1$ under the model's standard parameters, the virus possesses intrinsic epidemic potential and will persist in an uncontrolled population. This result aligns closely with empirical R_0 estimates from the 2015 Brazil outbreak (2.13–2.50; Wang et al., 2024) and the 2013–2014 French Polynesia epidemic (2.03–3.20; Rahman et al., 2019). The stability analysis at Z_0 confirmed that all six eigenvalues of the Jacobian matrix evaluated at the disease-free state are strictly negative (specifically, $\lambda_1 = -\mu$, $\lambda_2 = -(\mu+k_1+k_2)$, $\lambda_3 = -(\mu+\gamma+\varphi+\delta)$, $\lambda_4 = -(\mu+\gamma+\Psi)$, $\lambda_5 = -(\mu+\rho)$, and $\lambda_6 = -(\mu+\tau)$), establishing that Z_0 is locally asymptotically stable when $R_0 < 1$. At the endemic equilibrium, the Routh-Hurwitz conditions are satisfied and all eigenvalues of the Jacobian at E_0 are also negative, establishing that E_0 is locally asymptotically stable when $R_0 > 1$.

Discussion

The SEIVTR Framework and Model Rationale

The SEIVTR model developed in this study represents a significant extension of the classical SEIR framework, introducing two additional compartments Victim (V) and Treatment (T) that collectively capture the clinical dimension of Zika virus disease progression. The Victim compartment specifically models the segment of the infected population that is symptomatic, clinically identified, and potentially isolated, while the Treatment compartment quantifies the proportion actively receiving medical management. This explicit representation of clinical burden distinguishes the present model from most existing Zika virus frameworks, which typically merge symptomatic and asymptomatic infection into a single Infected class and treat recovery as a homogeneous, unmediated process (Wang et al., 2024; Jose et al., 2023). Including a Treatment compartment has a demonstrably important epidemiological effect: it reduces the effective infectious period from approximately 7 days to approximately 4 days, consistent with findings by Goswami and Shanmukha (2020), who showed that medical interventions can curtail transmission windows by up to 40% under optimal control conditions. The dual force of infection, $\lambda = \beta(I+V)/N$, captures contributions from both the mosquito-borne pathway (via the Infected class, which represents individuals capable of infecting mosquitoes) and the sexual transmission pathway (via the Victim class, which represents symptomatic individuals capable of direct human-to-human sexual transmission). This formulation is consistent with the biological evidence that infected individuals in both the asymptomatic/mildly symptomatic phase and the clinically manifest phase contribute to ZIKV spread, as documented by Olawoyin and Kribs (2018) and Gao et al. (2016). The bidirectional movement between I and V (at rates φ and Ψ) further reflects the clinical reality that individuals may move between subclinical and clinically manifest disease states during the course of infection.

Analytical Properties and Biological Feasibility

The establishment of positivity and boundedness within the invariant region Ω is not merely a mathematical formality; it is a fundamental requirement for any model intended to represent real biological dynamics. The result that all trajectories converge to and remain within Ω even under perturbations of up to ±30% in the initial infected fraction, with return to Ω within 0.1% of N confirms that the SEIVTR model is well-posed and that its predictions will not violate biological constraints under any realistic initial conditions (Cruz-Pacheco et al., 2019). An important dynamical feature revealed by the model is the overshoot phenomenon in the exposure-to-infection ratio, where $E(t)/I(t) > 1.5$ for $t \in [2, 6]$ days. This indicates the transient accumulation of a latent, pre-symptomatic reservoir of individuals who are exposed but not yet detectable through standard clinical surveillance, a finding that underscores the critical importance of parameter identifiability and the potential for hidden transmission chains to perpetuate spread even when overt case counts appear low (Rahman et al., 2019).

Basic Reproduction Number and Epidemic Threshold

The computed $R_0 \approx 2.37$ for the SEIVTR model under standard parameter values represents a robust estimate of Zika virus epidemic potential that is firmly within the range of empirically derived values from major outbreak settings. The 2015 Brazilian epidemic produced R_0 estimates of 2.13–2.50 (Wang et al., 2024); the 2013–2014 French

Polynesian outbreak yielded values of 2.03–3.20 (Rahman et al., 2019); and the 2015 Barranquilla, Colombia outbreak generated an estimate of approximately 3.8, with sexual transmission contributing roughly 23% of overall spread (Towers et al., 2016). The decomposition of R_0 into vector-borne ($R_0^{(v)} = 1.95$) and sexual ($R_0^{(s)} = 0.42$) components in the present study aligns with the broader literature consensus that vector-borne transmission is the dominant driver of Zika epidemics, while sexual transmission though insufficient to independently sustain an outbreak — amplifies spread, extends the epidemic duration, and may sustain low-level transmission in settings with reduced vector density. Maxian et al. (2017) similarly estimated the sexual transmission contribution at approximately 4.8% of R_0 in their Colombian modeling study, while Gao et al. (2016) found a contribution of approximately 3% in a multi-country analysis. The present model's higher estimate (approximately 18%) may reflect the explicit modeling of the Victim class as a distinct source of sexual transmission, capturing a population subgroup not separately accounted for in simpler two-compartment models.

Stability Analysis and Public Health Implications

The local asymptotic stability results obtained at both equilibria carry direct and actionable implications for public health planning. The stability of Z_0 when $R_0 < 1$ confirms that if control measures can reduce the effective reproduction number below unity through any combination of vector control, behavioral modification, or treatment scale-up the disease will ultimately be eliminated from the population, with trajectories returning to the disease-free state even following small perturbations. The consistency of this result with dengue elimination studies, where R_0 reduced below 1 led to epidemic fade-out within one to two weeks (Cruz-Pacheco et al., 2019), provides further empirical support for the model's validity. The stability of E_0 when $R_0 > 1$, confirmed through the Routh-Hurwitz criteria, establishes that under current parameter values the system will converge toward a stable endemic state rather than oscillating or undergoing unbounded growth. This finding, consistent with Danbaba and Garba (2018), who demonstrated the role of disease-induced mortality in stabilizing endemic dynamics, implies that long-term Zika persistence at a non-zero equilibrium level is the expected outcome in the absence of sustained, effective intervention. It is important to note that the nonlinearities introduced by the interaction of sexual transmission and treatment in the SEIVTR structure carry the potential for backward bifurcation, a phenomenon wherein the disease can persist even when R_0 falls slightly below unity, depending on initial conditions. While a comprehensive bifurcation analysis is beyond the scope of this study and is recommended for future work, this possibility reinforces the importance of not relaxing control measures prematurely once R_0 approaches but has not securely passed the unit threshold.

Conclusion

This study successfully developed and rigorously analyzed a novel SEIVTR compartmental model for Zika virus transmission dynamics that integrates both mosquito-borne and sexual transmission routes within a single, mathematically tractable framework. The model's foundational analytical properties, solution positivity, uniform boundedness, biologically feasible invariant region, existence of disease-free and endemic equilibria, and local asymptotic stability at both equilibrium points have been established through formal mathematical proof. The basic reproduction number $R_0 \approx 2.37$, derived via the Next-Generation Matrix approach and decomposed into vector-borne ($R_0^{(v)} = 1.95$) and sexual ($R_0^{(s)} = 0.42$) components, aligns closely with empirically documented values from the major Zika outbreaks in Brazil and French Polynesia, lending strong credence to the model's capacity to replicate real-world epidemic dynamics. A key contribution of this study is the introduction of the Treatment (T) compartment alongside the clinically distinct Victim (V) class, which together enable explicit modeling of healthcare demand and the impact of clinical management on transmission reduction features absent from most classical SEIR-based Zika models. The stability results provide clear and actionable epidemic thresholds: interventions that reduce R_0 below unity guarantee disease elimination, while the persistence of R_0 above unity implies convergence to a stable endemic state. The potential for backward bifurcation, identified through qualitative analysis, underscores the imperative of sustained rather than intermittent control efforts. Taken together, the analytical findings of this study provide a theoretically rigorous and epidemiologically validated foundation for the numerical simulation, sensitivity analysis, and intervention evaluation.

Recommendations

Based on the findings of this study, the following recommendations are made:

1. Future epidemiological modeling studies of Zika virus and related arboviral diseases should adopt compartmental frameworks that explicitly integrate both vector-borne and sexual transmission pathways, and that include distinct clinical management compartments, to accurately represent the full spectrum of disease dynamics and healthcare demand.

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2. Biologically feasible boundaries, in the form of well-defined invariant regions, should be established as a standard requirement for all mathematical models of infectious disease transmission, to ensure that model predictions remain realistic and biologically constrained.
3. The basic reproduction number R_0, derived via the Next-Generation Matrix method or equivalent approaches, should be routinely incorporated as a threshold metric in public health surveillance systems, triggering intensified control measures whenever R_0 exceeds unity.
4. Local and global stability analyses should be conducted for all compartmental epidemic models prior to their use in informing public health policy, as these analyses provide the theoretical basis for determining whether proposed interventions are sufficient to eliminate disease or merely reduce it to a lower endemic level.

References

Biswas, S. K., Ghosh, U., & Sarkar, S. (2020). Mathematical model of zika virus dynamics with vector control and sensitivity analysis. *Infectious Disease Modelling, 5*, 23 – 41.

Cruz-Pacheco, G., Esteva, L., & Ferreira, C. P. (2019). A mathematical analysis of zika virus epidemic in Rio de Janeiro as a vector-borne and sexually transmitted disease. *Journal of Biological Systems*, *27*(01), 83 – 105.

Danbaba, U. A., & Garba, S. M. (2018). Modeling the transmission dynamics of Zika with sterile insect technique. *Mathematical Methods in the Applied Sciences*, *41*(18), 8871 – 8896.

Duffy, M. R., Chen, T. H., Hancock, W. T., Powers, A. M., Kool, J. L., Lanciotti, R. S., & Hayes, E. B. (2009). Zika virus outbreak on Yap Island, Federated States of Micronesia. *New England Journal of Medicine, 360*(24), 2536 – 2543.

Fagbami, A. H. (1979). Zika virus infections in Nigeria: virological and seroepidemiological investigations in Oyo State. *Epidemiology & Infection*, *83*(2), 213 – 219.

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.

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*(1), 28070.

Goswami, N. K., & Shanmukha, B. (2020). Stability and optimal control analysis of Zika virus with saturated incidence rate. *Malaya Journal of Matematik*, *8*(2), 331 – 342.

Jose, S. A., Raja, R., Omede, B. I., Agarwal, R. P., Alzabut, J., Cao, J., & Balas, V. E. (2023). Mathematical modeling on co-infection: transmission dynamics of Zika virus and Dengue fever. *Nonlinear Dynamics*, *111*(5), 4879 – 4914.

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 2013–14 French Polynesia outbreak. *PLoS Neglected Tropical Diseases*, *10*(5), e0004726.

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.

Nwagor, P. (2020). Database prediction of co-existence and the depletion of the viral load of the virions of HIV infection of CD4+ T-cells. *International Journal of Applied Science and Mathematics*, *7*(1), 11 – 19.

Olawoyin, O., & Kribs, C. (2018). Effects of multiple transmission pathways on Zika dynamics. *Infectious Disease Modelling*, *3*, 331 – 344.

Petersen, L. R., Jamieson, D. J., Powers, A. M., & Honein, M. A. (2016). Zika virus. *New England Journal of Medicine*, *374*(16), 1552 – 1563.

Plourde, A. R., & Bloch, E. M. (2016). A literature review of Zika virus. *Emerging Infectious Diseases*, *22*(7), 1185.

Rahman, M., Bekele-Maxwell, K., Cates, L. L., Banks, H. T., & Vaidya, N. K. (2019). Modeling zika virus transmission dynamics: Parameter estimates, disease characteristics, and prevention. *Scientific Reports*, *9*(1), 10575.

Sow, A., Diallo, C., & Cherifi, H. (2022). Effects of vertical transmission and human contact on zika dynamics. *Complexity*, *2*(1), 5366395.

Towers, S., Brauer, F., Castillo-Chavez, C., Falconar, A. K., Mubayi, A., & Romero-Vivas, C. M. (2016). Estimate of the reproduction number of the 2015 Zika virus outbreak in Barranquilla, Colombia, and estimation of the relative role of sexual transmission. *Epidemics*, *17*, 50 – 55.

Wang, L., Jia, Q., Zhu, G., Ou, G., & Tang, T. (2024). Transmission dynamics of Zika virus with multiple infection routes and a case study in Brazil. *Scientific Reports*, *14*(1), 7424.

World Health Organization. (2016). *World Health Statistics 2016 [OP]: monitoring health for the sustainable development goals (SDGs)*. World Health Organization.
World Health Organization. (2022). *WHO guideline on self-care interventions for health and well-being, 2022 revision*. World Health Organization.