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A MATHEMATICAL MODEL FOR THE SEXUAL TRANSMISSION OF ZIKA VIRUS BASED ON GENDER AND SYMPTOMS

Trupti Barve, S. K. Tiwari and P. Porwal

School of Studies in Mathematics, Vikram University, Ujjain - 456010, Madhya Pradesh, INDIA

E-mail : truptibarve23@gmail.com, skt_tiwari75@yahoo.co.in, pradeepratnawat@yahoo.com

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Abstract: Zika virus is a member of Flaviviridae that also causes Japanese Encephalitis, dengue, yellow fever, and West Nile fever. General symptoms of the zika virus are low-grade fever (less than 38.5°), macula-papular rash, myalgia, asthenia, headache, and transient arthritis. Zika virus can cause congenital anomalies (such as microcephaly), Guillain-Barre syndrome, and other neurological and autoimmune disorders. In the present mathematical model, we observed the effect of sexual transmission on gender and symptoms based division of the infected human population. We proposed a theorem to check the local stability of disease free equilibrium state. To verify the theorem, we performed some numerical simulations. We also analyzed the global stability of disease free equilibrium state. Furthermore, we checked the effect of different sexual transmission rates on the population dynamics by calculating normalized sensitivity indices of R_0 . Results of the present study suggest that sexual transmission rates, we can restrict the Zika virus spread.

Keywords and Phrases: Disease free equilibrium, Stability analysis, Sensitivity analysis, Basic reproduction number, Sexual transmission.

2020 Mathematics Subject Classification: 34A34, 34D20, 34D23.

1. Introduction

Zika virus was first detected in a rhesus monkey in the Zika Forest of Uganda in 1947, and the first human case was also reported in Uganda in 1952 [13]. It

is a vector born disease, and the bite of an infected female Aedes mosquito is the primary cause of its spread [5], [12]. Besides this, there are many other proven, feasible, and effective sources of secondary transmission of the Zika virus. Perinatal transmission, blood diffusion and sexual transmission are also major cause for the spread the Zika virus [11]. Among them, sexual transmission of the Zika virus from men to men, men to women, and women to men also play a significant role [10]. Banuelos S. et al. presented a mathematical model to determine the effect of sexual transmission of the Zika virus by using Wolbachia for vector control [2]. Pizza D. et al. analyzed a simulation model representing the Zika virus dynamics in the pregnant woman population to check the incidence of microcephaly [14]. Agusto F. B. et al. analyzed a ZIKV model that includes human vertical transmission, birth of babies with microcephaly and asymptotic infected individuals [1]. Kibona I. E. and Yang C. formulated an SIR model of the Zika virus to observe its spread in newborns [9]. Their study suggests that by controlling vector transmission, the Zika spread can be controlled. The present paper aims to analyze the effect of sexual transmission in the Zika virus dissemination. In analyzing the behaviour of infectious disease, the basic reproduction number is an important factor. It is defined as the average number of secondary infections produced when one infected individual is introduced into a host population where everyone is susceptible [7]. It shows whether the disease will stay or die out. The basic reproduction number depends on the various physical parameters such as different transmission or infections rates, recovery rate, birth rate, death rate, fractions of populations and many more. Normalized sensitivity index measures the relative importance of these parameters to the basic reproduction number. The normalized sensitivity index of a variable to a parameter is the ratio of the relative change in the variable to the relative change in the parameter [4]. The remaining paper organizes as follows: Section 2 represents the modified mathematical model. We obtain the disease free equilibrium point and the basic reproduction number in Section 3. In section 4, we analyze the local and global stability of disease free equilibrium. In section 5, we perform sensitivity analysis to check the robustness and validity of the results. Section 6 contains the conclusion of the study.

2. Mathematical Model

The present model is the modification of the model discussed by S. Banuelos et al. [2]. S. Banuelos et al. divided the infected population into symptomatic and asymptomatic. We extend their model by dividing the infected population into male and female categories. Thus in the present paper, the total human population (N_H) consists of seven mutually exclusive compartments, which are as follows; Susceptible human (S_H) , ex-posed human (E_H) , infected symptomatic male

 (I_{MS}) , infected asymptomatic male (I_{MA}) , infected symptomatic female (I_{FS}) , infected asymptomatic female (I_{FA}) , recovered human (R). The vector population is divided into three classes; susceptible vectors (S_V) , exposed vectors (E_V) , and infected vectors (I_V) . Since the present model is the modification of a previously studied model, we proceed with the work by following its assumptions. Since vectors do not recover and remain infectious for the rest of their life, we do not consider the class of recovered vectors [2]. Symptomatic and asymptomatic humans recover with the same rate and do not become susceptible again [1]. We do not include disease induced human death because death due to the Zika is negligible [12]. The study does not consider birth and death rates because the model simulates only a single possible outbreak [6]. The total human population is constant. Therefore, $N_H = S_H + E_H + I_{MS} + I_{MA} + I_{FS} + I_{FA} + R$.



Figure 1: Transmission diagram

Mathematical model for the transmission dynamics of Zika is as follows

$$\frac{dS_H}{dt} = -\alpha_H b I_V \frac{S_H}{N_H} - \psi_{MS} I_{MS} \frac{S_H}{N_H} - \psi_{MA} I_{MA} \frac{S_H}{N_H} - \psi_{FS} I_{FS} \frac{S_H}{N_H} - \psi_{FA} I_{FA} \frac{S_H}{N_H}$$
$$\frac{dE_H}{dt} = \alpha_H b I_V \frac{S_H}{N_H} + \psi_{MS} I_{MS} \frac{S_H}{N_H} + \psi_{MA} I_{MA} \frac{S_H}{N_H} + \psi_{FS} I_{FS} \frac{S_H}{N_H} + \psi_{FA} I_{FA} \frac{S_H}{N_H} - \beta_H E_H$$

$$\frac{dI_{MS}}{dt} = q_1 \beta_H E_H - \gamma_1 I_{MS}$$

$$\frac{dI_{MA}}{dt} = q_2 \beta_H E_H - \gamma_1 I_{MA}$$

$$\frac{dI_{FS}}{dt} = q_3 \beta_H E_H - \gamma_2 I_{FS}$$

$$\frac{dI_{FA}}{dt} = q_4 \beta_H E_H - \gamma_2 I_{FA}$$

$$\frac{dR}{dt} = \gamma_1 (I_{MS} + I_{MA}) + \gamma_2 (I_{FS} + I_{FA})$$

$$\frac{dS_V}{dt} = \lambda_V N_V - \alpha_V (I_{MS} + I_{MA} + I_{FS} + I_{FA}) \frac{S_V}{N_V} - \mu_V S_V$$

$$\frac{dE_V}{dt} = \alpha_V b (I_{MS} + I_{MA} + I_{FS} + I_{FA}) \frac{S_V}{N_V} - \beta_V E_V - \mu_V S_V$$

$$\frac{dI_V}{dt} = \beta_V E_V - \mu_V I_V$$
(1)

Table 1. represents the description of model parameters and their associated values. **Table1: Parametric Values and Their Range**

Parameter	Description	Range	Baseline Values	
b	Mosquito biting rate	0.3-1	0.5	
λν	Birth rate of vectors per day	0.02-0.27	0.0690	
βv^{-1}	Latency period of Zika virus in vectors in days	8-12	10	
αv	Transmission probability from an infectious human to a susceptible mosquito per bite	0.3-0.75	0.4872	
μv^{-1}	Life span of a vector in days	5-20	14.5	
αн	Transmission probability from an infectious mosquito to a susceptible human per bite	0.1-0.75	0.4240	
β _H -1	Latency period of Zika virus in humans	3-14	8.5	
ΨMS	Rate of sexual transmission by symptomatic male	0.001-0.1	0.0098	
ΨMIA	Rate of sexual transmission by asymptomatic male	0.001-0.1	0.0532	
ΨFA	Rate of sexual transmission by asymptomatic female	0.001-0.1	0.0223	
ΨFS	Rate of sexual transmission by symptomatic female	0.001-0.1	0.0049	
q 1	Proportion of symptomatic male in humans		0.0924	
q ₂	Proportion of asymptomatic male in humans		0.5024	
q 3	Proportion of symptomatic female in humans		0.1031	
q 4	Proportion of asymptomatic female in humans		0.3021	
γ1	Recovery rate of male	0.2-0.3333	0.2741	
γ2	Recoveryrate of female	0.2-0.3333	0 3025	

3. Disease Free Equilibrium Point (DFE) And Basic Reproduction Number

3.1. Disease Free Equilibrium Point (DFE)

In the $DFE(E^0)$, $I_{MS} = 0$, $I_{MA} = 0$, $I_{FS} = 0$, $I_{FA} = 0$, $E_H = 0$, $I_V = 0$, $E_V = 0$, R = 0. Therefore, $DFE(E^0) = \left(N_H, 0, 0, 0, 0, 0, 0, 0, \frac{\lambda_V N_V}{\mu_V}, 0, 0\right)$

3.2. Basic Reproduction Number

Next generation matrix determined using [8] is

	$\boxed{ \frac{q_1\psi_{MS}}{\gamma_1} + \frac{q_2\psi_{MA}}{\gamma_1} + \frac{q_3\psi_{FS}}{\gamma_2} + \frac{q_4\psi_{FA}}{\gamma_2} }$	$\frac{\psi_{MS}}{\gamma_1}$	$\frac{\psi_{MA}}{\gamma_1}$	$\frac{\psi_{FS}}{\gamma_2}$	$\frac{\psi_{FA}}{\gamma_2}$	$\frac{\alpha_H b \beta_V}{(\beta_V + \mu_V)}$	$\frac{\alpha_H b}{\mu_V}$
$FV^{-1} =$	0	0	0	0	0	0	0
	0	0	0	0	0	0	0
	0	0	0	0	0	0	0
- /	0	0	0	0	0	0	0
	$\frac{\alpha_V b \lambda_V (q_1 \gamma_1 + q_2 \gamma_1 + q_3 \gamma_2 + q_4 \gamma_2)}{\mu_V}$	$\frac{\alpha_V b \lambda_V}{\gamma_1 \mu_V}$	$\frac{\alpha_V b \lambda_V}{\gamma_1 \mu_V}$	$\frac{\alpha_V b \lambda_V}{\gamma_2 \mu_V}$	$\frac{\alpha_V b \lambda_V}{\gamma_2 \mu_V}$	0	0
	0	0	0	0	0	0	0

Spectral radius of the next generation matrix is the basic reproduction number (R_0) and is determined by the following equation

$$\lambda^{2} - \left(\frac{q_{1}\psi_{MS}}{\gamma_{1}} + \frac{q_{2}\psi_{MA}}{\gamma_{1}} + \frac{q_{3}\psi_{FS}}{\gamma_{2}} + \frac{q_{4}\psi_{FA}}{\gamma_{2}}\right)\lambda - \frac{\alpha_{H}\alpha_{V}b^{2}\beta_{V}\lambda_{V}(q_{1}\gamma_{1} + q_{2}\gamma_{1} + q_{3}\gamma_{2} + q_{4}\gamma_{2})}{\mu_{V}(\beta_{V} + \mu_{V})} = 0$$

Therefore,

$$R_0 = \frac{R_{0S} + \sqrt{R_{0S}^2 + 4R_{0V}^2}}{2}$$

where,

$$R_{0S} = \left(\frac{q_1\psi_{MS}}{\gamma_1} + \frac{q_2\psi_{MA}}{\gamma_1} + \frac{q_3\psi_{FS}}{\gamma_2} + \frac{q_4\psi_{FA}}{\gamma_2}\right),$$
$$R_{0V} = \sqrt{\frac{\alpha_H\alpha_V b^2\beta_V\lambda_V(q_1\gamma_1 + q_2\gamma_1 + q_3\gamma_2 + q_4\gamma_2)}{\mu_V(\beta_V + \mu_V)}}$$

4. Stability Analysis of DFE

4.1. Local Stability of DFE

We propose following theorem to check the local stability of DEF condition

Theorem 4.1. Disease free equilibrium point E^0 is locally asymptotically stable when

i. $[q_1\psi_{MS} + q_2\psi_{MA} + q_3\psi_{FS} + q_4\psi_{FA} - (\gamma_1 + \gamma_2)]\beta_H - \gamma_1\gamma_2 < 0$ ii. $\beta_H < \gamma_1 + \gamma_2$ iii. $(q_1\psi_{MS} + q_2\psi_{MA})\beta_H < \gamma_1 + \gamma_2$ iv. $(q_3\psi_{FS} + q_4\psi_{FA})\beta_H < \gamma_1 + \gamma_2$ Provided $R_{0S} < 1$. **Proof.** Jacobian matrix of the system of equations (1) at disease free equilibrium

is

Seven roots of Jacobian matrix are $\lambda = -\mu_V, -\mu_V, -\mu_V, -\beta_V, 0, 0, -\gamma_i, -\gamma_2$ and the rest of the three roots are given by

$$\lambda^{3} + (\gamma_{1} + \gamma_{2} + \beta_{H})\lambda^{2} - \{ [q_{1}\psi_{MS} + q_{2}\psi_{MA} + q_{3}\psi_{FS} + q_{4}\psi_{FA} - (\gamma_{1} + \gamma_{2})]\beta_{H} - \gamma_{1}\gamma_{2}\}\lambda - (q_{1}\psi_{MS}\gamma_{2} + q_{2}\psi_{MA}\gamma_{2} + q_{3}\psi_{FS}\gamma_{1} + q_{4}\psi_{FA}\gamma_{1} - \gamma_{1}\gamma_{2})\beta_{H} = 0$$

Necessary conditions to apply Routh-Hurwitz criteria are as follows i. $[q_1\psi_{MS} + q_2\psi_{MA} + q_3\psi_{FS} + q_4\psi_{FA} - (\gamma_1 + \gamma_2)]\beta_H - \gamma_1\gamma_2 < 0$ ii. $R_{0S} < 1$.

According to Routh-Hurwitz criteria, sufficient conditions for the system (1) to be stable are

i. $[q_1\psi_{MS} + q_2\psi_{MA} + q_3\psi_{FS} + q_4\psi_{FA} - (\gamma_1 + \gamma_2)]\beta_H - \gamma_1\gamma_2 < 0$ ii. $\beta_H < \gamma_1 + \gamma_2$ iii. $(q_1\psi_{MS} + q_2\psi_{MA})\beta_H < \gamma_1 + \gamma_2$ iv. $(q_3\psi_{FS} + q_4\psi_{FA})\beta_H < \gamma_1 + \gamma_2$ Hence, the theorem is proved.

4.2. Numerical Simulation. To verify the theorem, we found the numerical solution of the system of equations (1) and represented the solution graphically.

For this, we selected the parameter values from the range given in Table 1 such that they satisfy the necessary conditions of the theorem. Table 1 takes the parameter ranges taken from [2]. FIGURE 2 represents the numerical solution of the system of equations (1) in the DEF state of the populations and verifies the proposed theorem. It shows that all infected, exposed, and recovered populations remain zero for a long duration, if the required conditions of the proposed theorem are satisfied.



Figure 2: Time series for all populations

4.3. Global Stability OF DFE

Theorem 4.2. The fixed point $U_0(x^*, 0)$ is a globally asymptotic stable equilibrium of system provided $R_0 < 1$ (local asymptotically stable) and those assumptions H_1 and H_2 are satisfied [3].

The given system is expressed in the form:

$$\frac{dX}{dt} = F(X, Z)$$
$$\frac{dZ}{dt} = G(X, Z), \ G(X, 0) = 0$$

Where $X \in \mathbb{R}^m$ denotes (its components), the number of uninfected individuals and $Z \in \mathbb{R}^n$ denotes (its components) the number of infected individuals including latent, infectious etc. $U_0 = (x^*, 0)$ denotes the disease free equilibrium of this system.

The condition (H1) and (H2) below must be met to guarantee local asymptotic stability.

H1: for $\frac{dX}{dt} = F(X,0), X^*$ is globally asymptotically stable (g.a.s.), H2: $G(X,Z) = AZ - \widehat{G}(X,Z), \widehat{G}(X,Z) \ge 0$ or $(X,Z) \in \Omega$, where $A = D_Z G(X^*,0)$ is an M matrix (the off diagonal elements of A are non -negative) and Ω is the region where the model makes biological sense. To apply the above theorem to present model, the model (1) can be expressed in the following form

$$\frac{dX}{dt} = F(X, Z)
= \begin{bmatrix}
-\alpha_H b I_V \frac{S_H}{N_H} - \psi_{MS} I_{MS} \frac{S_H}{N_H} - \psi_{MA} I_{MA} \frac{S_H}{N_H} - \psi_{FS} I_{FS} \frac{S_H}{N_H} - \psi_{FA} I_{FA} \frac{S_H}{N_H} \\
\gamma_1 (I_{MS} + I_{MA}) + \gamma_2 (I_{FS} + I_{FA}) \\
\lambda_V N_V - \alpha_V (I_{MS} + I_{MA} + I_{FS} + I_{FA}) \frac{S_V}{N_V} - \mu_V S_V
\end{aligned}$$

$$\begin{aligned} \frac{dZ}{dt} &= G(X, Z) \\ &= \begin{bmatrix} \alpha_H b I_V \frac{S_H}{N_H} + \psi_{MS} I_{MS} \frac{S_H}{N_H} + \psi_{MA} I_{MA} \frac{S_H}{N_H} + \psi_{FS} I_{FS} \frac{S_H}{N_H} + \psi_{FA} I_{FA} \frac{S_H}{N_H} - \beta_H E_H \\ q_1 \beta_H E_H - \gamma_1 I_{MS} \\ q_2 \beta_H E_H - \gamma_1 I_{MA} \\ q_3 \beta_H E_H - \gamma_2 I_{FS} \\ q_4 \beta_H E_H - \gamma_2 I_{FA} \\ \alpha_V b (I_{MS} + I_{MA} + I_{FS} + I_{FA}) \frac{S_V}{N_V} - \beta_V E_V - \mu_V S_V \\ \beta_V E_V - \mu_V I_V \end{aligned}$$

where $X = (S_H, R, S_V)$ and $Z = (E_H, I_{MS}, I_{MA}, I_{FS}, I_{FA}, E_V, I_V)$ $U_0(x^*, 0) = E^0 = (X^*, 0)$ where $X^* = \left(N_H, 0, \frac{\lambda_V N_V}{\mu_V}\right)$ is DFE of $\frac{dX}{dt}$. Therefore, $G(X, 0) = (0 \ 0 \ 0 \ 0 \ 0 \ 0)^T$ and $F(X^*, 0) = \begin{pmatrix} 0 \\ 0 \\ 0 \end{pmatrix}$

$$\hat{G}(X,Z) = \begin{pmatrix} -\beta_H & \frac{\psi_{MS}S_H}{N_H} & \frac{\psi_{MA}S_H}{N_H} & \frac{\psi_{FS}S_H}{N_H} & \frac{\psi_{FA}S_H}{N_H} & 0 & \frac{\alpha_H bS_H}{N_H} \\ q_1\beta_H & 0 & 0 & 0 & 0 & 0 & 0 \\ q_2\beta_H & 0 & 0 & 0 & 0 & 0 & 0 \\ q_3\beta_H & 0 & 0 & 0 & 0 & 0 & 0 \\ q_4\beta_H & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\alpha_V bS_V}{N_V} & \frac{\alpha_V bS_V}{N_V} & \frac{\alpha_V bS_V}{N_V} & \frac{\alpha_V bS_V}{N_V} & \beta_V - \mu_V & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \beta_V & -\mu_V \end{pmatrix}$$

$$\hat{G}(X,Z) = \begin{pmatrix} (I_{MS}\psi_{MS} + I_{MA}\psi_{MA} + I_{FS}\psi_{FS} + I_{FA}\psi_{FA}) \left(1 - \frac{S_H}{N_H}\right) \\ 0 \\ 0 \\ \alpha_V \left(I_{MS} + I_{MA} + I_{FS} + I_{FA}\right) \left(\frac{\lambda_V}{\mu_V} - \frac{S_V}{N_V}\right) \\ 0 \end{pmatrix}$$

 $\widehat{G}(X,Z)$ is greater than or equal to zero because $N_H \ge S_H$ and $\frac{\lambda_V N_V}{\mu_V} \ge S_V$. It is clear by observation that A is an M matrix. Also X^* is globally asymptotically stable equilibrium of $\frac{dX}{dt} = F(X,0)$. Hence, by the above theorem $DFE \ E^0$ is globally asymptotically stable.

5. Sensitivity Analysis

The normalized forward sensitivity index of a variable u that depends differentiable on a parameter p is defined as $\gamma_P^u = \frac{\partial u}{\partial P} \times \frac{P}{u}$ [4]. Baseline values for parameters $b, \lambda_V, \beta_H, \alpha_H, \beta_V, \alpha_V, \mu_V$ are stated in Table 1, [2]. On the other hand, we chose the baseline values for $\gamma_1, \gamma_2, \psi_{MS}, \psi_{MA}, \psi_{FS}, \psi_{FA}, q_1, q_2, q_3, q_4$ based on the following assumption. Symptomatic individuals and asymptomatic individuals are equally infectious. But since symptomatic individuals know about their illness, they are less sexually active than asymptomatic individuals. If symptomatic individuals are sexually active, they use protection [2]. The possibility of virus transmission from men to women is more than from women to men [5]. The Zika virus remains present in the male body for a longer time than in the female body [5]. Among all infected individuals, about 80% remain asymptomatic [11]. In view of the above points, we made the following assumptions.

I.
$$\gamma_1 < \gamma_2$$
.
II. $\psi_{FS} < \psi_{MS} < \psi_{FA} < \psi_{MA}$
III. $q_1 + q_3 < 0.20$
IV. $q_1 < q_2, q_1 < q_4, q_3 < q_2, q_3 < q_4$
Calculated normalized sensitivity indices of R0 for some parameters are as follows
 $\gamma_{\psi_{MA}}^{R_0} = 0.8329, \gamma_{\psi_{FA}}^{R_0} = 0.2889, \gamma_{\psi_{MS}}^{R_0} = 0.0485, \gamma_{\psi_{FS}}^{R_0} = 0.0.0152, \gamma_{\gamma_1}^{R_0} = -0.4243, \gamma_{\gamma_0}^{R_0} = -0.109.$

Obtained sensitivity indices show that among all considered modes of sexual transmission, R_0 is most sensitive to transmission rate of asymptomatic male and least to transmission rate of symptomatic female. Negative indices of γ_1 and γ_2 , show that as the recovery rates of the male and the female increase basic reproduction number decreases and system becomes free from disease. FIGURE 3 represents the basic reproduction number with respect to some parameters and supports the observations done by sensitivity indices.



Figure 3: Basic reproduction number verses model parameters

6. Conclusion

In the present paper, we analyzed a deterministic model for the Zika virus transmission. The current model included transmission through vectors and sexual transmission through humans. We studied transmission through the symptomatic male, symptomatic female, asymptomatic male, and asymptomatic female. To analyze the disease spread, we obtained the conditions for local stability and global stability of disease free equilibrium. Further, we proposed a theorem to examine the local stability of DFE and verified it using numerical simulation. The present study proved that the local stability of disease free equilibrium rates, recovery rates, and proportion of several infected classes. Values of sensitivity indices show that sexual transmission can be a significant cause of the Zika virus spread. Zika virus spread can be restricted by improving recovery rates.

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