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DYNAMICAL SYSTEM OF ZIKAV DISEASE SPREAD THROUGH THE ISOLATION WITH TWO GROUPS OF INFECTED POPULATION

by

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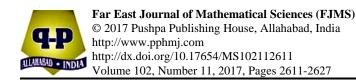
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DYNAMICAL SYSTEM OF ZIKAV DISEASE SPREAD THROUGH THE ISOLATION WITH TWO GROUPS OF INFECTED POPULATION

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Abstract

A viral disease ZIKAV (Zika virus) caused by a type of a Flavivirus closely related to dengue is primarily transmitted to humans by the bites of infected mosquitoes from the Aedes aegypti. Seeking to understand the dynamics of spread of the ZIKAV disease, we propose *SEIIJRV*₁*V*₂*V*₃ mathematical models for vector transmission of the virus, sexual contact transmission, isolation, and conducted stability analysis. Isolation is one of the ways to disease control. This isolation is done on symptomatic-infected human population to prevent the spread of the disease. We calculate the basic reproduction number \mathcal{R}_0 and show that for $\mathcal{R}_0 < 1$, the disease-free equilibrium is locally asymptotically stable. In addition, it is shown that for a special case when $\mathcal{R}_0 > 1$, the endemic equilibrium is locally asymptotically stable. Numerical simulations are shown to support the analytical results and allow us to have a clear view of the effect of isolation.

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1. Introduction

Zika virus was first isolated in Rhesus monkey in Uganda in 1947 [3]. In 1950, the virus had become epidemic in many countries of Africa, Southeast Asia, and the Pacific Islands [6]. Zika virus is very harmful to the developing fetus in pregnant women, because if the virus attacks pregnant women, it can cause brain development in the fetus which becomes abnormal, miscarriage, and microcephaly [12].

Zika virus is spread to humans through mosquito bites and sexual transmission. The other modes of the transmission are blood transfusions and perinatal transmission (transmission of the disease from mother to baby during pregnancy, birth or breastfeeding). Zika virus can be spread by a man to his sexual partners. In some case, sexual transmission may occur through people who have symptoms of the Zika virus disease [4]. The most common symptoms of the Zika virus disease are fever, rash, joint pain, conjunctivitis (red eyes), and disorders of the nervous system, including Guillain-Barre syndrome (GBS). However, there is not a vaccine or specific treatment to prevent and treat viral diseases [1].

Many researchers have developed a mathematical model and analysis regarding the transmission of Zika virus disease. For instance, Moreno et al. [8] formulated a compartmental model for Zika virus with two patch model, Kucharski et al. [7] described and analyzed transmission dynamics of Zika virus in French Polynesia, and Gao et al. [5] showed the prevention and control of Zika as a mosquito-Borne and sexually transmitted disease to a mathematical model. In this paper, we modify and analyze a disease from Zika virus spread model with two groups of infected population that was introduced by Moreno et al. [8] and sexually transmitted disease was created by Onuorah Martis et al. [10]. Modification of the model is done by adding *J* compartement [9], namely isolated population, so that this model is called *SEIIJRV*₁*V*₂*V*₃ *model*. Modifications are done by considering the assumption that symptomatic-infected individual can move into isolated population and it is assumed that individual who has been recovered cannot be re-infected by Zika virus.

The paper is organized into five sections. Section 1 describes the background and purpose of this paper. Section 2 describes the formulation of the model used. Section 3 describes the model analysis. Section 4 performs the numerical analysis. The conclusions are provided in Section 5.

2. Model Formulation

In this section, we develop a deterministic mathematical model for the dynamics of ZIKAV and introduce the modification of ZIKAV spread model through the isolation of two groups of infected population. Our model incorporate vital dynamic for both the human and vector compartements. We coupled an *SEIIJR* for the human to SEI for the vector population. Specfically, S(t), E(t), $I_a(t)$, $I_s(t)$, J(t), R(t) represent the susceptible, exposed, asymptomatic and symptomatic infected, isolated and recovered humans, respectively. While $V_1(t)$, $V_2(t)$, $V_3(t)$ represent the susceptible, exposed and infected mosquitoes, respectively.

As with any modeling endeavor, various assumptions about the underlying must be made. At this stage, we explain clearly for the assumptions of this model. The total of human population is constant. The birth rate and natural mortality rate are denoted by μ_1 . New infections result from sexual contacts between susceptible and infected individuals with an incidence rate α_2 . Then susceptible human becomes medically exposed to ZIKAV. It can also occur when they receive a bite from a mosquito that is already inducted with rate α_1 . Proportion of symptomatic and asymtomatic infections are ω and $(1 - \omega)$. The rate at which the exposed humans move to the infectious compartment I_s is σ_1 , this is done when the individual shows the symptom of Zika virus disease. Isolation is done to prevent contact with mosquitoes and avoiding sexual contact between symptomatic-infected and susceptible human beings. Furthermore, the isolated patient who infected by ZIKAV disease will be given by special treatment with the isolation rate ρ . Isolated human moves to the recovered compartment R at the rate γ . Asymptomatic and symptomatic infected human beings can move to the

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recovered compartment *R* at the rate of γ_a and γ_s , respectively. Recovered individuals did not go back to the susceptible class because the ZIKAV confers life time immunity to them. The susceptible mosquito populations V_1 is recruited via birth at the rate μ_2 . A portion of this human population becomes infected at the rate $\alpha_1\beta_2$ when they bite an individual having ZIKAV disease and thus move to the exposed compartement V_2 . When the exposed mosquitoes developed ZIKAV symptoms, they move to the infected compartment V_3 at the rate σ_2 . Every mosquito population is affected by the natural death at the rate μ_2 . Based on our assumptions and the transfer diagram, we can derive the following system of differential equations that govern our model

$$\begin{aligned} \frac{dS}{dt} &= \mu_1 N_h - \alpha_1 \beta_1 \frac{V_3}{N_h} S - \alpha_2 \frac{I_s}{N_h} S - \mu_1 S, \\ \frac{dE}{dt} &= \omega \bigg(\alpha_1 \beta_1 \frac{V_3}{N_h} S + \alpha_2 \frac{I_s}{N_h} S \bigg) - (\sigma_1 + \mu_1) E, \\ \frac{dI_a}{dt} &= (1 - \omega) \bigg(\alpha_1 \beta_1 \frac{V_3}{N_h} S + \alpha_2 \frac{I_s}{N_h} S \bigg) - (\gamma_a + \mu_1) I_a, \\ \frac{dI_s}{dt} &= \sigma_1 E - (\gamma_s + \rho + \mu_1) I_s, \\ \frac{dJ}{dt} &= \rho I_s - (\mu_1 + \gamma) J, \\ \frac{dR_h}{dt} &= \gamma J + \gamma_s I_s + \gamma_a I_a - \mu_1 R, \\ \frac{dV_1}{dt} &= \mu_2 N_v - \alpha_1 \beta_2 \frac{I_s}{N_h} V_1 - \mu_2 V_1, \\ \frac{dV_2}{dt} &= \alpha_1 \beta_2 \frac{I_s}{N_h} V_1 - (\sigma_2 + \mu_2) V_2, \\ \frac{dV_3}{dt} &= \sigma_2 V_2 - \mu_2 V_3 \end{aligned}$$
(1)

with $S(0) \ge 0$, $E(0) \ge 0$, $I_a(0) \ge 0$, $I_s(0) \ge 0$, $J(0) \ge 0$, $R(0) \ge 0$, $V_1(0) \ge 0$, $V_2(0) \ge 0$, and $V_3(0) \ge 0$.

We can normalize our equation (1) by introducing the new variables: $s = \frac{S}{N_h}, \quad e = \frac{E}{N_h}, \quad i_a = \frac{I_a}{N_h}, \quad i_s = \frac{I_s}{N_h}, \quad j = \frac{J}{N_h}, \quad r = \frac{R}{N_h}, \quad v_1 = \frac{V_1}{N_v},$ $v_2 = \frac{V_2}{N_v}, \quad v_3 = \frac{V_3}{N_v}. \text{ Then we use } r = 1 - s - e - i_a - i_s - j \text{ and } v_1 =$ $1 - v_2 - v_3. \text{ This creates a new seven-dimensional system of equation}$ $\frac{ds}{dt} = \mu_1 - \alpha_1 \beta_1 v_3 s - \alpha_2 i_s s - \mu_1 s,$ $\frac{de}{dt} = \omega(\alpha_1 \beta_1 v_3 s + \alpha_2 i_s s) - (\sigma_1 + \mu_1) e,$ $\frac{di_a}{dt} = (1 - \omega)(\alpha_1 \beta_1 v_3 s + \alpha_2 i_s s) - (\gamma_a + \mu_1) i_a,$ $\frac{di_s}{dt} = \sigma_1 e - (\gamma_s + \rho + \mu_1) i_s,$ $\frac{dj}{dt} = \rho i_s - (\mu_1 + \gamma) j,$ $\frac{dv_2}{dt} = \alpha_1 \beta_2 i_s (1 - v_2 - v_3) - (\sigma_2 + \mu_2) v_2,$ $\frac{dv_3}{dt} = \sigma_2 v_2 - \mu_2 v_3.$ (2)

Then we determine the existence of equilibrium points; computing the effective basic reproduction number, and establishing the conditions for stability of the equilibria points.

Lemma 1. Let the initial data set be $s(0) \ge 0$, $e(0) \ge 0$, $i_a(0) \ge 0$, $i_s(0) \ge 0$, $j(0) \ge 0$, $v_2(0) \ge 0$ and $v_3(0) \ge 0$. Then the solution set $\{s, e, i_a, i_s, j, v_2, v_3\}(t)$ is positive for all time t > 0.

3. Model Analysis

The disease-free equilibrium of the system (1) is given by $T_0(s, e, i_a, i_s, j, v_2, v_3) = (1, 0, 0, 0, 0, 0, 0)$ and the endemic equilibrium of the system (1) is given by

$$T^*(s, e, i_a, i_s, j, v_2, v_3) = (s^*, e^*, i_a^*, i_s^*, j^*, v_2^*, v_3^*),$$

where

$$s^{*} = \frac{\mu_{1}}{i_{s}^{*}\alpha_{2} + v_{3}^{*}\alpha_{1}\beta_{1} + \mu_{1}}, \quad e^{*} = \frac{s(\alpha_{2}i_{s}^{*} + v_{3}^{*}\alpha_{1}\beta_{1})\omega}{\mu_{1} + \sigma_{1}},$$
$$i_{a}^{*} = \frac{s(\alpha_{2}i_{s}^{*} + v_{3}^{*}\alpha_{1}\beta_{1})\omega}{\gamma_{a} + \mu_{1}}, \quad i_{s}^{*} = \frac{\sigma_{1}e^{*}}{\gamma_{s} + \mu_{1} + \rho}, \quad j^{*} = \frac{\rho i_{s}^{*}}{\gamma + \mu_{1}},$$
$$v_{2}^{*} = \frac{\alpha_{1}\beta_{2}i_{s}^{*}(1 - v_{3}^{*})}{\alpha_{1}\beta_{2}i_{s}^{*} + \sigma_{2} + \mu_{2}}, \quad v_{3}^{*} = \frac{\sigma_{2}v_{2}^{*}}{\mu_{2}}.$$
(3)

We calculated the basic reproduction number using by the next generation operator approach by van den Driessche and Watmough [11]. The next generation matrix at the disease-free equilibrium T_0 is given by:

	(0	$\omega \alpha_2$	0	0	$\omega \alpha_1 \beta$	1)		
	0	0	0 0	0	0			
F =	0	0	0	0	0	,		
	0	$\alpha_1\beta_2$	0	0	0			
	(0	0	0	0	0)		
	(σ1	+ µ1		0		0	0	0
	-	$-\sigma_1$	$\gamma_s +$	ρ+μ	ι1	0	0	0
V =		0	-	-ρ	γ	+ µ1	0	0.
		0		0		0	$\sigma_2 + \mu_2$	0
		0		0		0	$-\sigma_2$	μ_2

The basic reproduction number \mathcal{R}_0 is dominant eigenvalue of FV^{-1} , thus we get

$$\mathcal{R}_0 = \frac{1}{2} \mathcal{R}_0^1 + \frac{1}{2} \sqrt{4 \mathcal{R}_0^2 + (\mathcal{R}_0^1)^2}$$
(4)

with

$$\mathcal{R}_0^1 = \frac{\alpha_2 \sigma_1 \omega}{(\gamma_s + \rho + \mu_1)(\sigma_1 + \mu_1)}, \quad \mathcal{R}_0^2 = \frac{\alpha_1^2 \beta_1 \beta_2 \sigma_1 \sigma_2 \omega}{\mu_2 (\gamma_s + \rho + \mu_1)(\sigma_1 + \mu_1)(\sigma_2 + \mu_2)}.$$

The stability of system (1) is dependent on the basic reproduction number \mathcal{R}_0 . The stability analysis of both the equilibrium T_0 and T^* will be provided through the following theorems.

Lemma 2. For system (1), the disease-free equilibrium T_0 exists. Moreover, endemic equilibrium T^* is unique and positive if and only if $\Re_0 > 1$.

Theorem 1. The disease-free equilibrium T_0 is locally asymtotically stable if $\Re_0 < 1$.

Proof. The Jacobian matrix at T_0 for system (1) is given by

	$\left(-\mu_{1}\right)$	0	0	$-\alpha_2$	0	0	$-\alpha_1\beta_1$
	0	$-\mu_1 - \sigma_1$	0	$\alpha_2 \omega$	0	0	$\alpha_1\beta_1\omega$
	0	0	$-\gamma_a - \mu_1$	$\alpha_2(1-\omega)$	0	0	$\alpha_1\beta_1(1-\omega)$
$J_{T_0} =$	0	σ_1	0	$-\gamma_s - \mu_1 - \rho$	0	0	0
-	0	0	0	ρ	$-\gamma-\mu_1$	0	0
	0	0	0	$\alpha_1\beta_2$	0	$-\mu_2 - \sigma_2$	0
	0	0	0	0	0	σ_2	$-\mu_2$)

The characteristic polynomial of the matrix J_{T_0} is

$$(-J_{11} + \lambda)(-J_{33} + \lambda)(-J_{55} + \lambda)(\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4) = 0$$

with

$$\begin{aligned} a_1 &= \gamma_s + 2\mu_1 + 2\mu_2 + \rho + \sigma_1 + \sigma_2 > 0, \\ a_2 &= (\mu_2 + (\sigma_2 + \mu_2))((\gamma_s + \rho + \mu_1) + (\sigma_1 + \mu_1)) + \mu_2(\sigma_2 + \mu_2) \\ &+ (\gamma_s + \rho + \mu_1)(\sigma_1 + \mu_1)(1 - \mathcal{R}_0^1), \\ a_3 &= (\gamma_s + \rho + \mu_1) \Big(\mu_2(\sigma_2 + \mu_2) + \frac{\mu_2(\sigma_1 + \mu_1)(\sigma_2 + \mu_2)}{(\gamma_s + \rho + \mu_1)} \Big) \\ &+ (\gamma_s + \rho + \mu_1)((\sigma_2 + \mu_2) + \mu_2(\sigma_1 + \mu_1))(1 - \mathcal{R}_0^1), \\ a_4 &= \mu_2(\gamma_s + \rho + \mu_1)(\sigma_1 + \mu_1)(\sigma_2 + \mu_2)(1 - \mathcal{R}_0^1 + \mathcal{R}_0^2). \end{aligned}$$

We have three negative eigenvalues: $\lambda_1 = J_{11} = -\mu_1 < 0$, $\lambda_2 = J_{33} = -\gamma_a - \mu_1 < 0$ and $\lambda_3 = J_{55} = -\gamma - \mu_1 < 0$. While λ_4 , λ_5 , λ_6 and λ_7 can be obtained by solving the equation below:

$$\lambda^{4} + a_{1}\lambda^{3} + a_{2}\lambda^{2} + a_{3}\lambda + a_{4} = 0.$$
 (5)

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Based on Routh-Hurwitz criteria, characteristic equation (5) for fixed point T_0 is stable, if it is eligible for the following conditions:

$$a_1 > 0, a_2 > 0, a_3 > 0, a_4 > 0, a_1a_2 > a_3, a_1a_2a_3 > (a_3^2 + a_1^2a_4).$$
 (6)

Because all parameters are positive, $a_1 > 0$. Thereafter, while $\mathcal{R}_0 < 1$, a_2 and a_3 are positive. Afterward, a_4 will be positive or negative, it depends with \mathcal{R}_0 . If $\mathcal{R}_0 < 1$, then there are equations as follow:

Let $\mathcal{R}_0^1 < 1$. Because $\mathcal{R}_0^2 > 0$, $\mathcal{R}_0^1 - \mathcal{R}_0^2 < 1$, and hence $a_4 > 0$. Based on $\mathcal{R}_0 < 1$, $a_4 > 0$, and the value of parameters at numerical simulation is obtained from $a_1a_2 > a_3$, and $a_1a_2a_3 > (a_3^2 + a_1^2a_4)$. Thus, for $\mathcal{R}_0 < 1$, (6) holds.

As a result, the disease-free equilibrium T_0 for system (1) is locally asymptotically stable if $\mathcal{R}_0 < 1$.

Theorem 2. If $\mathcal{R}_0 > 1$, then the endemic equilibrium T^* is locally asymptotically stable.

Proof. The proof is based on Castillo-Chaves and Song [2]. Let $\varphi = \beta_1$ be the bifurcation parameter and $x_1 = s$, $x_2 = e$, $x_3 = i_a$, $x_4 = i_s$, $x_5 = j$, $x_6 = v_2$, $x_7 = v_3$. System (1) becomes

$$f_{1}(x_{1}, x_{2}, x_{3}, x_{4}, x_{5}, x_{6}, x_{7}) = \mu_{1} - \alpha_{1}\varphi x_{1}x_{7} - \alpha_{2}x_{1}x_{4} - \mu_{1}x_{1},$$

$$f_{2}(x_{1}, x_{2}, x_{3}, x_{4}, x_{5}, x_{6}, x_{7}) = \omega(\alpha_{1}\varphi x_{1}x_{7} + \alpha_{2}x_{1}x_{4}) - (\alpha_{1} + \mu_{1})x_{2},$$

$$f_{3}(x_{1}, x_{2}, x_{3}, x_{4}, x_{5}, x_{6}, x_{7}) = (1 - \omega)(\alpha_{1}\varphi x_{1}x_{7} + \alpha_{2}x_{1}x_{4}) - (\gamma_{a} + \mu_{1})i_{a},$$

$$f_{4}(x_{1}, x_{2}, x_{3}, x_{4}, x_{5}, x_{6}, x_{7}) = \sigma_{1}x_{2} - (\gamma_{s} + \rho + \mu_{1})x_{4},$$

$$f_{5}(x_{1}, x_{2}, x_{3}, x_{4}, x_{5}, x_{6}, x_{7}) = \rho x_{4} - (\gamma_{j} + \mu_{1})x_{5},$$

$$f_{6}(x_{1}, x_{2}, x_{3}, x_{4}, x_{5}, x_{6}, x_{7}) = \alpha_{1}\beta_{2}x_{4}(1 - (x_{6} + x_{7})) - (\sigma_{2} + \mu_{2})x_{6},$$

$$f_{7}(x_{1}, x_{2}, x_{3}, x_{4}, x_{5}, x_{6}, x_{7}) = \sigma_{2}x_{6} - \mu_{2}x_{7}.$$

Based on condition $\mathcal{R}_0 = 1$,

$$\varphi = \varphi^* = \frac{2\mu_2(\mu_1 + \sigma_1)(\mu_2 + \sigma_2)(\gamma_s + \mu_1 + \rho) - \alpha_2\omega\mu_2(\mu_2 + \sigma_2)}{2\alpha_1^2\beta_2\alpha_1\sigma_2\omega}$$

and disease-free equilibrium T_0 has one zero eigenvalue and seven negative eigenvalues. The zero eigenvalue has a right eigenvector $(u_1, u_2, u_3, u_4, u_5, u_6, u_7)$ and a left eigenvector $(v_1, v_2, v_3, v_4, v_5, v_6, v_7)$ as follows:

Let $u_4 > 0$. Then

$$u_{1} = -\frac{\alpha_{2}u_{4} + \alpha_{1}\beta_{1}u_{7}}{\mu_{1}} < 0, \quad u_{2} = \frac{(\gamma_{s} + \mu_{1} + \rho)u_{4}}{\sigma_{1}} > 0,$$
$$u_{3} = \frac{\alpha_{2}(1 - \omega)u_{4} + \alpha_{1}\beta_{1}(1 - \omega)u_{7}}{\gamma_{a} + \mu_{1}} > 0, \quad u_{5} = \frac{\rho u_{4}}{\gamma + \mu_{1}} > 0,$$

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$$u_6 = \frac{\alpha_1 \beta_2 u_4}{\mu_2 + \sigma_2} > 0, \quad u_7 = \frac{\sigma_2 u_6}{\mu_2} > 0,$$

 $v_1 = 0, v_3 = 0$ and $v_5 = 0$.

Let $v_6 > 0$. Then

$$v_2 = \frac{\mu_2 v_7}{\sigma_2} > 0, \quad v_4 = \frac{(\mu_1 + \sigma_1)v_2}{\alpha_2 \omega} > 0, \quad v_7 = \frac{(\mu_2 + \sigma_2)v_6}{\sigma_2} > 0.$$

Define

$$a = \sum_{k,i,j=1}^{7} v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (T^0, \phi^*),$$

$$b = \sum_{k,i,j=1}^{7} v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \phi} (T^0, \phi^*).$$
 (7)

We have

$$\frac{\partial^2 f_2}{\partial x_1 \partial x_4} (T^0, \, \varphi^*) = \omega \alpha_2, \quad \frac{\partial^2 f_6}{\partial x_4 \partial x_6} (T^0, \, \varphi^*) = -\alpha_1 \beta_2,$$
$$\frac{\partial^2 f_2}{\partial x_1 \partial x_7} (T^0, \, \varphi^*) = \omega \alpha_1 \varphi^*, \quad \frac{\partial^2 f_2}{\partial x_7 \partial \varphi} (T^0, \, \varphi^*) = \omega \alpha_1.$$

From (7), we drive

$$\begin{split} a &= -\frac{\mu_2(\mu_2 + \sigma_2)v_6}{\mu_1 \sigma_2^2} \\ & \cdot \left(\alpha_2 u_4 + \alpha_1 \varphi^* \frac{\sigma_2}{\mu_2} \left(\frac{\alpha_1 \beta_2 u_4}{\mu_2 + \sigma_2}\right)\right) \left(u_4 \omega \alpha_2 + \frac{\sigma_2}{\mu_2} \left(\frac{\alpha_1 \beta_2 u_4}{\mu_2 + \sigma_2}\right) \omega \alpha_1 \varphi^*\right) \\ & - \alpha_1 \beta_2 v_6 \frac{\alpha_1 \beta_2 u_4}{\mu_2 + \sigma_2} u_6 < 0, \\ b &= \frac{\alpha_1^2 \omega \beta_2 \mu_2 \sigma_2 (\mu_2 + \sigma_2) u_4 v_6}{\sigma_2^2 \mu_2 (\mu_2 + \sigma_2)} > 0. \end{split}$$

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The values of *a* and *b* satisfies condition (iv) in [2]. When φ changes from negative $\varphi < \varphi^*$ ($\Re_0 < 1$) to positive $\varphi > \varphi^*$ ($\Re_0 > 1$), the disease-free equilibrium T_0 changes its stability from stable to unstable. Correspondingly, a negative unstable equilibrium T^* becomes positive and locally asymptomatically stable. So this achieves the proof that the endemic equilirium T^* is locally asymptotically stable if $\Re_0 > 1$.

4. Numerical Simulation

Numerical solutions for the system are discussed in this section. We make several interesting observations by numerically simulating in the range of parameter values. The parameter values used in this simulation are $\beta_1 = 0.4$, $\beta_2 = 0.5$, $\alpha_1 = 0.5$, $\alpha_2 = 0.00035$, $\mu_1 = 0.00493$, $\mu_2 = 0.35$, $\sigma_1 = 0.53$, $\sigma_2 = 0.2$, $\gamma_a = 0.14286$, $\gamma_s = 0.071428$, $\gamma = 0.196429$ and $\omega = 0.18$.

In the next discussion, our objectives were justify the stability properties of the equilibrium points based on the theorm in Section 3 and to see the influence of parameter variations. The dynamics of human populations and the the mosquitoes is observed when $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$. In this case, \mathcal{R}_0 is the basic reproduction number define in equation (4). The initial values used are s(0) = 0.6, e(0) = 0.4, $i_a(0) = 0$, $i_s(0) = 0$, j(0) = 0, $v_2(0) = 0.4$ and $v_3(0) = 0.2$.

Suppose $\Re_0 < 1$. We set the parameter value for this simulation, so that the condition $\Re_0 = 0.183207$ is satisfied. It is found that there is a diseasefree equilibrium $T_0(s, e, i_a, i_s, j, v_2, v_3) = (1, 0, 0, 0, 0, 0, 0)$. Figure 1 shows that the curves *s*, *e*, *i_a*, *i_s*, *j*, *r*, *v₂* and *v₃* asymptotically approaching the disease-free equilibrium point T_0 . The simulation result are consistent with Theorem 1. These results indicate that if the parameters of the model are setting to get $\Re_0 < 1$, then the ZIKAV disease could be extinct because the population system will stable at a disease-free equilibrium point.

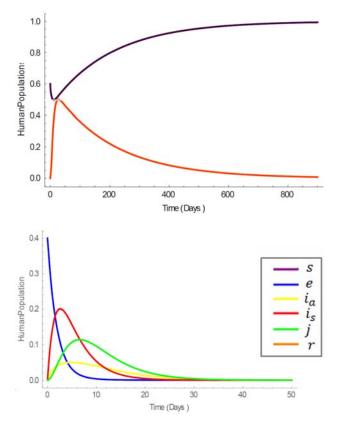


Figure 1. Dynamics human population with condition $\mathcal{R}_0 < 1$.

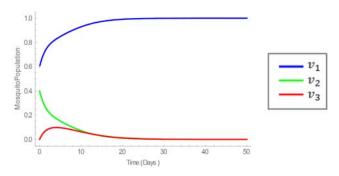


Figure 2. Dynamics mosquito population with condition $\Re_0 < 1$.

Suppose $\Re_0 > 1$. We set the parameter values as $\alpha_2 = 0.25$, $\mu_2 = 0.028571$, $\sigma_2 = 0.095238$, and $\omega = 0.8$, so that the condition $R_0 = 2.355895$ is satisfied. It is found that there is a endemic equilibrium point $T^*(s, e, i_a, i_s, j, v_2, v_3) = (s^*, e^*, i_a^*, i_s^*, j^*, v_2^*, v_3^*)$ with $s^* = 0.235603$, $e^* = 0.005635$, $i_a^* = 0.0051$, $i_s^* = 0.010808$, $j^* = 0.010735$, $v_2^* = 0.019939$, and $v_3^* = 0.066465$. From Figures 3 and 4, simulation result are found to be consistent with Theorem 2. These results indicate that if the parameters of the model are setting to get $\Re_0 > 1$, then the ZIKAV disease could be exist because the population system will stable at a endemic equilibrium point.

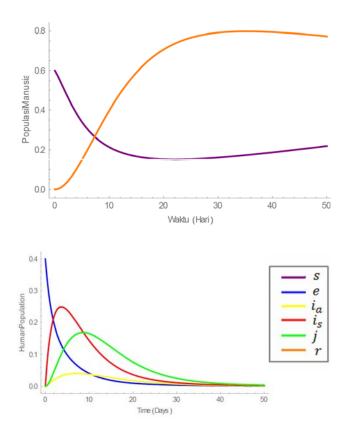


Figure 3. Dynamics human population with condition $\Re_0 > 1$.

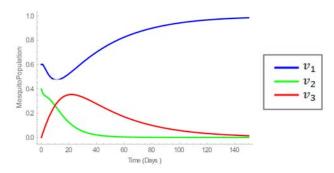


Figure 4. Dynamics mosquito population with condition $\Re_0 > 1$.

Then, under $\mathcal{R}_0 > 1$, the sensitivity analysis is carried out to show which parameter gives more effect to \mathcal{R}_0 when the disease is spreading. The result of this analysis is shown at Table 1.

	Substituting for $50 > 1$
Parameter	Index sensitivity $(\Upsilon_p^{\mathcal{R}_0})$
α_1	0.820508
α_2	0.179492
β_1	0.410254
β_2	0.410254
μ_1	-0.015956
μ_2	-0.504927
γ _a	0
γ_s	-0.152427
ω	0.589746
ρ	-0.426799
σ_1	0.005435
σ_2	0.094673
γ	0

Tabel 1. Index sensitivity for $\mathcal{R}_0 > 1$

Table 1 explains that if α_1 increases or decreases by one unit, then increase or decrease in \mathcal{R}_0 is by 0.820508 unit, and if ρ decreases or

increases by one unit, then increase or decrease in \mathcal{R}_0 is by 0.426799 unit. From sensitivity analysis, we got some influential parameters which are α_1 , β_1 , β_2 , μ_2 , ω and ρ .

Furthermore, the effect of parameters variation to human beings infected is shown in the following Figure 5. We vary the isolation rate. The human populations illustrated in Figure 5 show that if isolation rate (ρ) is increased and the other values of parameters remain constant, then it causes reduction in number of the asymptomatic infected population and the symptomatic infected population. Similarly, if isolation rate (ρ) decreases until zero, then the asymptomatic infected population, and the symptomatic infected population increase. The following Figure 5 is about the effect of isolation rate.

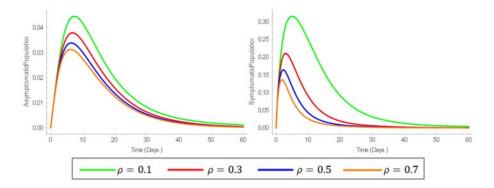


Figure 5. Dynamics human population due to the influence of isolation rate.

5. Conclusions

The model discussed in this study is a modification of the existing model where there is an additional assumption concerning isolation with two infected populations and sexual transmission. Analysis of this dynamic system shows that there are two equilibria, namely, disease-free equilibrium and endemic equilibrium. Moreover, the disease-free equilibrium of system is locally asymptotically stable if and only if $\mathcal{R}_0 < 1$. The endemic

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equilibrium is positive and locally asymptotically stable if $\mathcal{R}_0 > 1$. The simulation results show that if the isolation rate increases, then R_0 will decrease, but decreasing \mathcal{R}_0 value can only bring down the spread of ZIKAV disease.

References

- V. M. Cao-Lormeau, M. Didier and E. J. Nilles, Rapid spread of emerging Zika virus in the Pacific area, Clinical Microbiology and Infection 20 (2014), 0595-0596. DOI: 10.1111/1469-0691.1270.
- [2] C. Castillo-Chaves and B. Song, Dynamical models of tuberculosis and their applications, Math. Biosci. Eng. 1 (2004), 361-404. DOI: 10.1038/nm089-815.
- G. W. Dick, S. F. Kitchen and A. J. Haddow, Zika virus: isolations and serological specificity, Trans *R* Soc Trop Med Hy. 46 (1952), 509-520.
 DOI: 10.1016/0035-9203(52)90042-4.
- [4] M. Didier, R. Claudine, R. Emilie, N. Tuxuan, A. Teissier and V. M. Cao-Lormeau, Potential sexual transmission of Zika virus, Emerging Infectious Diseases 21 (2015), 359-361. DOI: 10.3201/eid2012.141363.
- [5] D. Gao, Y. Lou, D. He, T. C. Porco, Y. Kuang, G. Chowell and S. Ruan, Prevention and control of Zika as a mosquito-borne and sexually transmitted disease: a mathematical modeling analysis, Scientific Reports 6 (2016), 28070. DOI: 10.1038/srep28070.
- [6] M. K. Kindhauser, T. Allen, V. Frank, R. S. Santhana and C. Dye, Zika, the origin and spread of a mosquito-borne virus, Bull World Health Organ 94 (2016), 675-686.
- [7] A. J. Kucharski, S. Funk, R. M. Eggo, H. P. Mallet, W. J. Edmunds and E. J. Nilles, Transmission dynamics of Zika virus in island populations: a modelling analysis of the 2013-14 French Polynesia Outbreak, PloS NegI Trop Dis. 10 (2016), e0004726. DOI: 10.371/journal.pntd.0004726.
- [8] V. Moreno, B. Espinoza, D. Bichara, S. A. Holechek and C. Castillo-Chavez, Role of short-term dispersal on the dynamics of Zika virus in an extreme idealized environment, Infectious Disease Modelling 2 (2017), 21-34. DOI: 10.1016/j.idm.2016.12.002.
- [9] T. O. Oluyo and M. O. Adeyemi, Mathematical analysis of Zika epidemic model, IOSR Journal of Mathematics 12 (2016), 21-33. DOI: 10.9790/5728-1206042133.

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- [10] O. Onuorah Martins, A. Ademu, E. I. Obi and A. M. Hasheem, Deterministic mathematical model of Zika virus, Researchjournali's Journal of Math. 3 (2016). DOI: 10.14445/22315373/IJMTT-V30P502.
- [11] P. van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, Math. Biosci. 180 (2002), 29-48. DOI: 10.1016/s0025-5564(02)00108-6.
- [12] WHO Health Organization, Western Pacific Region, Zika Virus 4 January 2016, http://www.wpro.who.int/mediacentre/factsheets/fs_05182015_zika/en/.