A mathematical model of Zika disease by considering transition from the asymptomatic to symptomatic phase

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Abstract. This work presents a mathematical model of Zika disease considering infected individual transition from the asymptomatic to symptomatic phase. Zika virus (ZIKV) itself is a virus that belongs to arbovirus transmitted by the Aedes aegypti mosquitoes. It can also be transmitted through human contact such as sexual contact, blood transfusion, and transplacental infection. As a matter of fact, 80% of those who get infected by ZIKV are asymptomatic. In this work, we investigate the Zika model by considering individual transition case from the asymptomatic to symptomatic phase using SEAIR (host) - SI (vector) model. In this model, we involve human and mosquito populations which have a big role to the transmission of ZIKV itself. In this study, basic reproduction number ($R_0$) calculated as the largest eigenvalue of Next-Generation Matrix. Furthermore, analytical results also be conducted to determine the existence and local stability of the equilibrium point. A numerical simulation presented to analyze the sensitivity and elasticity of $R_0$ for some parameters involved in the model, and followed with simulation of autonomous system. We find that transition of asymptomatic to symptomatic case in Zika transmission hold an important role in determining the size of the basic reproduction number. More transition to symptomatic case are better to know the "dark" figure of the real cases in the field.

1. Introduction
In general, Zika is a contagious disease whose infection is caused by the Zika virus (ZIKV). The Zika virus is classified into the genus of Flavivirus and the family of Flaviviridae. This virus belongs to the type of arbovirus, a virus that can be spread through arthropod bites. In this case, it is a mosquito. The species of the mosquito that primarily transmit this virus are Aedes aegypti and Aedes albopictus mosquitoes. Other than being transmitted through mosquito bites, ZIKV can also be transmitted through human contact. First, the Zika virus is transmitted to an embryo or fetus from the mother through the placenta. This method is known as transplacental infection. Babies born from infected mothers are likely to experience microcephaly or other abnormalities known as congenital Zika syndrome. On the other hand, Zika virus transmission can also occur through blood transfusions, organ transplants, or any other blood products. Lastly, ZIKV can also be transmitted through sexual contact [1].

The Zika virus was first identified in the DNA of a monkey in Kampala, Uganda in 1947. Meanwhile, the first case of human infection was discovered in 1954 in Uganda and Tanzania. Zika virus outbreak has been recorded in several regions such as Asia, Africa, America, and the
Pacific Islands. The first outbreak occurred in the Yap Islands (Federated States of Micronesia) in 2007 [2]. In Asia, the Zika virus was first isolated from the Aedes aegypti mosquito in 1966. In Indonesia, the first case of Zika virus infection, which occurred in Central Java, was reported in 1977 [3].

Before the onset of clinical symptoms, viruses that enter the body of an individual will replicate and go through an incubation period. In the human body, the incubation period of this virus lasts for 3-14 days. WHO stated that 80% of people infected with the Zika virus do not develop symptoms. On the symptomatic cases, the symptoms of the disease itself are generally mild, such as joint and muscle pain, rash, fever, and conjunctivitis. These symptoms usually last for 2-7 days. There is still no vaccine that has been found that can either prevent or treat Zika virus-infected individuals. Individuals who get infected by the Zika virus can treat the disease by taking rest and medication to relieve the symptoms [2]. As a form of support for the countries that get affected by Zika disease, WHO took the initiative to issue a program titled Zika Strategic Response Framework. The purpose of this program is to investigate cases of complications caused by Zika disease and also to improve prevention measures, risk communication, and care for individuals infected with the Zika virus [4].

In the study of epidemiology, mathematics has its role to study the spread of an epidemic which later became known as mathematical epidemiology. Mathematical epidemiology is closely related to mathematical models. The mathematical model for epidemiology was first introduced by Kermack and McKendrick in 1927. The model is known as the SIR model, which is a mathematical model with 3 main populations: susceptible, infected, and recovered populations [5]. As time went by, researchers had developed the SIR model by reconsidering each factor that is both involved and not involved in the model as needed.

Researchers have proposed several mathematical models to analyze the spread of Zika disease by developing SIR models from Kermack and McKendrick. For example, a model of Zika disease considering the population of asymptomatic infected individuals was proposed [6]. Furthermore, there is also a model that analyzes similar models by adding sexual transmission factors and combining them with transmission factors through mosquitoes. This model also involves intervention factors towards mosquitoes to prevent the spread of the disease [7]. Meanwhile, in a study by Khan, M. A., et al. (2019), the model was constructed to formulate the dynamics of Zika disease by involving the analysis of optimal control strategies by considering control variables to reduce the spread Zika virus [8].

In this work, a mathematical model for Zika disease is being proposed and analyzed by considering several conditions. First, note that asymptomatic individuals can transmit the disease to both susceptible individuals and susceptible vectors. Since the majority of infected individuals are the asymptomatic ones, then we still need to involve the population of asymptomatic infected individuals and its transmission factor in the model construction. Furthermore, it was stated that in epidemiology, there are several diseases whose viruses can be transmitted before the clinical symptoms in infected individuals onset [9]. In this phase, the individuals may develop symptoms later on. In the case of Zika disease, this statement can be obtained due to the allegation that is arising as a result of the occurrence of sexual transmission case before the patient itself showed any clinical symptoms of Zika disease [10]. This shows that in the Zika disease model, there is a possibility of individuals transition from asymptomatic infected compartments to symptomatic infected compartments at a certain rate. Finally, it is known that an adult mosquito has an average short life span, which is around 14-21 days for each lifetime. Meanwhile, the incubation period of the Zika virus in a mosquito lasts for 10 days [1]. For this reason, the presence of the exposed mosquito populations can be ignored. Based on the explanation above, then a mathematical model of Zika disease by considering a transition from the asymptomatic to symptomatic phase is proposed in this article.

The presentation of this article is given as follows. In section 2, we construct our model
by modifying a proposed Zika transmission model by author in[7] by considering the transition from asymptomatic to symptomatic individual. Mathematical analysis regarding the equilibrium points and the basic reproduction number conducted rigorously in Section 3. We also show that our modified model undergoes a forward bifurcation when the basic reproduction number equal to one using the center manifold approach. Some numerical experiments simulated in Section 4 to discuss the elasticity of the basic reproduction number and some autonomous simulation to show a possible scenario in the field. Finally, conclusions given in Section 5 to summarized our results.

2. Model construction

In this section, we formulate a mathematical model of Zika disease considering a transition from the asymptomatic to symptomatic phase. In this work, two populations that have the ability to transmit the Zika virus are being involved, those are the human population and mosquito population. The human population is divided into five subpopulations: susceptible (S_h), asymptomatic infected (A_h), symptomatic infected (I_h), and recovered (R_h). The mosquito population is divided into two subpopulations: susceptible mosquitoes (S_v) and infected mosquitoes (I_v) which the total population is denoted by N_v = S_v + I_v. The model is formed as a nonlinear ordinary differential equations system of seven dimensions:

\[
\begin{align*}
\frac{dS_h}{dt} &= \Lambda_h - \frac{S_h}{N_h}(\beta_{h1} I_v + \beta_{h2} (I_h + k A_h)) - \mu_h S_h, \\
\frac{dE_h}{dt} &= \frac{S_h}{N_h}(\beta_{h1} I_v + \beta_{h2} (I_h + k A_h)) - (\mu_h + \chi) E_h, \\
\frac{dA_h}{dt} &= \chi (1 - p) E_h - \alpha A_h - \gamma A_h - \mu_h A_h, \\
\frac{dI_h}{dt} &= \chi p E_h + \alpha A_h - \gamma I_h - \mu_h I_h, \\
\frac{dR_h}{dt} &= \gamma I_h + \gamma A_h - \mu_h R_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \beta_v S_v \left( \frac{I_h + A_h}{N_h} \right) - \mu_v S_v, \\
\frac{dI_v}{dt} &= \beta_v S_v \left( \frac{I_h + A_h}{N_h} \right) - \mu_v I_v.
\end{align*}
\]

The transmission diagram that are being used in constructing the model of Zika disease considering transition cases from the asymptomatic to symptomatic phase are presented in Figure 1.

Please note that, since we assume that the number of new born is equal to the number of death, both in human and mosquito population, we have that \( \Lambda_h = \mu_h N_h \) and \( \Lambda_v = \mu_v N_v \), respectively. Therefore, we have that \( \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dA_h}{dt} + \frac{dI_h}{dt} + \frac{dR_h}{dt} = \frac{dN_h}{dt} = \Lambda_h - \mu_h N_h = 0 \) which mean that total of human is constant in time. Similarly, we have that total of mosquito population is also constant.

Based on the model above, parameters that show the numbers of birth human and mosquito populations are shown respectively by \( \Lambda_h \) and \( \Lambda_v \). Furthermore, the natural death rate for humans and mosquitoes is each one denoted by \( \mu_h \) and \( \mu_v \). Meanwhile, the parameters of infection are shown by \( \beta_{h1} \) as an infection rate from infected mosquitoes to susceptible individuals, \( \beta_{h2} \) as an infection rate from symptomatic and asymptomatic infected individuals to susceptible individuals through sexual intercourse, and \( \beta_v \) as an infection rate from symptomatic and asymptomatic infected individuals to susceptible mosquitoes. Also, \( k > 1 \) is a constant that
Figure 1. Diagram of Zika Transmission

distinguishes the infection rate from asymptomatic infected individuals to the symptomatic ones. There is also a constant $p \in (0, 1)$ which shows the proportion of exposed individuals that becomes the symptomatic infected individuals. Moreover, the virus development rate in exposed individuals are denoted by $\chi$, the rate of individuals transition from the asymptomatic to symptomatic phase is given by $\alpha$, and the recovery rate of symptomatic and asymptomatic infected individuals is given by $\gamma$.

3. Model analysis

3.1. Positiveness and boundedness

**Theorem 3.1.** Given the initial condition $(S_h(0), E_h(0), A_h(0), I_h(0), R_h(0), S_v(0), I_v(0))$ nonnegative, then the solution of $(S_h, E_h, A_h, I_h, R_h, S_v, I_v)$ in system (1) is nonnegative for every $t \geq 0$.

Proof. Let $t_1 = \text{sup}(t > 0 \mid S_h > 0, E_h > 0, A_h > 0, I_h > 0, R_h > 0, S_v > 0, I_v > 0)$. From the equation of model (1) that represents the susceptible individuals we have

$$\frac{dS_h}{dt} = \Lambda_h - \frac{S_h}{N_h} (\beta_{h1} I_v + \beta_{h2} (I_h + k A_h)) - \mu_h S_h,$$

$$\iff \frac{dS_h}{dt} = \Lambda_h - \left( \frac{\beta_{h1} I_v}{N_h} + \frac{\beta_{h2}}{N_h} (I_h + k A_h) + \mu_h \right) S_h.$$  \hspace{1cm} (2)

Let $f(t) = \frac{\beta_{h1} I_v}{N_h} + \frac{\beta_{h2}}{N_h} (I_h + k A_h) + \mu_h$, then we get the integrating factor as follows

$$H = e^{\int_0^t f(\tau)d\tau}.$$

By multiplying equation (2) with the integrating factor, we obtain

$$e^{\int_0^t f(\tau)d\tau} \frac{dS_h}{dt} + e^{\int_0^t f(\tau)d\tau} f(t) S_h = e^{\int_0^t f(\tau)d\tau} \Lambda_h,$$

$$\iff \frac{d}{dt} \left( S_h e^{\int_0^t f(\tau)d\tau} \right) = e^{\int_0^t f(\tau)d\tau} \Lambda_h.$$  \hspace{1cm} (3)
Since human and mosquito population is constant, using above new definition, we have
\[ S_h(t_i) e^{\int_{t_i}^{t_f} f(\tau) d\tau} - S_h(0) = \int_0^{t_f} e^{\int_{t_i}^{\tau} f(\tau') d\tau'} \Lambda_h d\tau, \]
\[ \iff S_h(t_i) = e^{\int_{t_i}^{t_f} f(\tau) d\tau} \left[ S_h(0) + \int_0^{t_i} e^{\int_{t_i}^{\tau} f(\tau') d\tau'} \Lambda_h d\tau \right] > 0. \]

Similar approach could be applied to show \((E_h, A_h, I_h, R_h, S_v, I_v) > 0\) for all time \(t \geq 0\).

**Corollary 3.1.1.** Each subpopulation on human \((N_h)\) and mosquito \((N_v)\) population in system (1) are bounded for every \(t \geq 0\).

**Proof.** As previously mentioned, the total of human population is constant. Based on Theorem 3.1, each subpopulation of human is positive for all time \(t \geq 0\). Therefore, we have that each subpopulation of human is bounded by \(N_h\). Similar argument applied to show that \(S_v\) and \(I_v\) bounded above by \(N_v\).

Using Theorem 3.1 and Corollary 3.1.1, we can construct a positively region that is written in Corollary 3.1.2 as a direct consequence of the positiveness and boundedness model solution to the system (1).

**Corollary 3.1.2.** With a nonnegative initial point of \(R^2_+ \cup \bar{\Theta}\), the region \(\Omega \subset R^2_+ \cup \bar{\Theta}\) which
\[ \Omega = \Omega_h \times \Omega_v \]
where
\[ \Omega_h = \left\{ (S_h, E_h, A_h, I_h, R_h) \in R^2_+ \cup \bar{\Theta} : N_h(t) = \frac{\Lambda_h}{\mu_h} \right\}, \]
\[ \Omega_v = \left\{ (S_v, I_v) \in R^2_+ \cup \bar{\Theta} : N_v(t) = \frac{\Lambda_v}{\mu_v} \right\}, \]
is a positively invariant solution to the model (1).

### 3.2. Model nondimensionalization

Let the proportion of each subpopulation in this model is defined as follows
\[ s_h = \frac{S_h}{N_h}, e_h = \frac{E_h}{N_h}, a_h = \frac{A_h}{N_h}, i_h = \frac{I_h}{N_h}, r_h = \frac{R_h}{N_h}, s_v = \frac{S_v}{N_v}, i_v = \frac{I_v}{N_v}. \]  
(4)

Since human and mosquito population is constant, using above new definition, we have \(r_h = 1 - s_h - e_h - a_h - i_h\) and \(s_v = 1 - i_v\). Using the relation above, we obtain the following system that formulates the spread of Zika disease considering transition from the asymptomatic to symptomatic phase for each compartment.

\[ \frac{ds_h}{d\tau} = (1 - s_h) - s_h (\beta_{h1}^* i_v + \beta_{h2}^* (i_h + k a_h)), \]
\[ \frac{de_h}{d\tau} = s_h (\beta_{h1}^* i_v + \beta_{h2}^* (i_h + k a_h)) - (1 + \chi^*) e_h, \]
\[ \frac{da_h}{d\tau} = \chi^* (1 - p) e_h - (\alpha^* + \gamma^* + 1) a_h, \]
\[ \frac{di_h}{d\tau} = \chi^* p e_h + \alpha^* a_h - (\gamma^* + 1) i_h, \]
\[ \frac{dI_v}{d\tau} = \frac{1}{\epsilon} \left( \beta_{v1}^* (1 - i_v) (i_h + a_h) - i_v \right). \]
(5)
where \( \beta^*_h = \frac{\beta_h}{\mu_h}, \beta^*_v = \frac{\beta_v}{\mu_v} \), \( \gamma^* = \frac{\gamma}{\mu_h}, \alpha^* = \frac{\alpha}{\mu_h}, \beta^* = \frac{\beta_v}{\mu_v}, \epsilon = \frac{\mu_v}{\mu_h}, \tau = t \mu_h \). To simplify the notations, then \( \beta^*_h, \beta^*_v, \gamma^*, \alpha^* \), and \( \beta^*_v \) are rewritten as \( \beta_h, \beta_v, \gamma, \alpha, \) and \( \beta_v \) respectively.

### 3.3. Basic reproduction number (\( R_0 \))

According to [11], basic reproduction number is denoted by \( R_0 \) and is defined as written in Definition 3.1.

**Definition 3.1.** Basic reproduction number (\( R_0 \)) is the average number of new cases of an infection caused by one typical infected individual, in a population consisting of susceptibles only.

**Theorem 3.2.** The basic reproduction number of model (1) is given by

\[
R_0 = \frac{1}{2} \left( R_2 + \sqrt{(R_2)^2 + 4(R_1)^2} \right),
\]

where

\[
R_1 = \sqrt{\frac{\beta_h \beta_v \chi}{(1+\chi)(\gamma+1)}}, \quad R_2 = \frac{\beta_h \chi (k(1-p)(\gamma+1)+p(\gamma+1)+\alpha)}{(1+\chi)(\alpha+\gamma+1)(\gamma+1)}.
\]

**Proof.** We use the Next-Generation Matrix (NGM) method introduced by [11] to obtain the basic reproduction number for model given by (1). We have the jacobian matrix as written below.

\[
J_{DFE} = \begin{bmatrix}
-1 & -\chi & \beta_h k & \beta_h & \beta_h \\
\chi (1-p) & -\alpha - \gamma - 1 & 0 & 0 \\
\alpha & -\gamma - 1 & 0 & 0 \\
\beta_v & \beta_v & -\frac{1}{e} & 0 \\
0 & \beta_v & \beta_v & 0 \\
-\frac{1}{e} & \beta_v & \beta_v & 0 \\
\end{bmatrix}.
\]

The matrix above can be decomposed into two matrices as written by

\[
T = \begin{bmatrix}
0 & \beta_h k & \beta_h \\
0 & 0 & 0 \\
0 & 0 & 0 \\
0 & \frac{\beta_v}{e} & \beta_v \\
0 & \frac{\beta_v}{e} & \beta_v \\
\end{bmatrix}, \quad \Sigma = \begin{bmatrix}
-1 & -\chi & 0 & 0 & 0 \\
\chi (1-p) & -\alpha - \gamma - 1 & 0 & 0 \\
\alpha & -\gamma - 1 & 0 & 0 \\
0 & \beta_v & \beta_v & -\frac{1}{e} \\
0 & \beta_v & \beta_v & 0 \\
\end{bmatrix}.
\]

Using the matrices in equation (8), we can get the matrix \( FV^{-1} \). Then, we obtain the basic reproduction number as written in (6) by determining the spectral radius of matrix \( FV^{-1} \) \( (\rho(FV^{-1})) \).

Please see [12, 13, 15, 14, 16, 17, 20] for more example on the calculation of the basic reproduction number on some epidemiological models. Note that Theorem 3.2 shows the basic reproduction number of model (5) involving two ways of disease transmission, those are the virus transmission through mosquitoes and among humans. We can derive the basic reproduction number by not involving one of those two transmission factors of the virus. The results are given by Corollary 3.2.1 and Corollary 3.2.2.

**Corollary 3.2.1.** If there is no transmission through sexual intercourse (\( \beta_h = 0 \)), then the basic reproduction number of the model becomes

\[
R_1 = \sqrt{\frac{\beta_h \beta_v \chi}{(1+\chi)(\gamma+1)}}.
\]
Corollary 3.2.2. If there is no transmission through mosquito bites ($\beta_{h1} = 0$), then the basic reproduction number of the model becomes

$$R_2 = \frac{\beta_{h2} \chi (k(1-p)(\gamma + 1) + p(\gamma + 1) + \alpha)}{(1 + \chi)(\alpha + \gamma + 1)(\gamma + 1)}.$$

3.4. Disease-free equilibrium

Disease-free equilibrium point (DFE) is defined as a point or condition when the disease in a population no longer exists. Thus, the disease-free equilibrium point of this model is the equilibrium point produced when the number of infected individuals is equal to zero ($e_h, a_h, i_h, i_v = 0$). Therefore, the disease-free equilibrium point of model (5) is given by

$$E_0 = (s_h^0, e_h^0, a_h^0, i_h^0, i_v^0) = (1,0,0,0,0).$$ (9)

Theorem 3.3. The disease-free equilibrium point $E_0 = (1,0,0,0,0)$ always exists without conditions.

Proof. Since $E_0 = (1,0,0,0,0)$ is a nonnegative number, the equilibrium point $E_0$ always has a biological meaning without conditions.

Theorem 3.4. The disease-free equilibrium point of model (5) is locally asymptotically stable if $R_0 < 1$.

Proof. Here, we will prove Theorem 3.4 by using the method that is introduced by [21]. Let us rewrite the variables of system (5) as a vector $x = (e_h, a_h, i_h, i_v, s_h)$. According to [21], $x = \mathcal{F}_i - \mathcal{Y}_i$ and $\mathcal{Y}_i = \mathcal{Y}_i^- - \mathcal{Y}_i^+$ where

$$\mathcal{F}_i = \begin{bmatrix} e_h \\ a_h \\ i_h \\ i_v \\ s_h \end{bmatrix}, \quad \mathcal{Y}_i^- = \begin{bmatrix} s_h (\beta_{h1} i_v + \beta_{h2} (i_h + k a_h)) \\ 0 \\ \frac{1}{2} (\beta_v (1 - i_v) (i_h + a_h)) \\ 0 \end{bmatrix}, \quad \mathcal{Y}_i^+ = \begin{bmatrix} (1 + \chi) e_h \\ -\chi (1 - p) e_h + (\alpha + \gamma + 1) a_h \\ -\chi p e_h - \alpha a_h + (1 + \gamma) i_h \\ -\gamma pe_h + \alpha a_h \end{bmatrix}, \quad \mathcal{Y}_i^+ = \begin{bmatrix} (1 + \chi) e_h \\ -\chi (1 - p) e_h + (\alpha + \gamma + 1) a_h \\ -\chi p e_h - \alpha a_h + (1 + \gamma) i_h \\ -\gamma pe_h + \alpha a_h \end{bmatrix}.$$

Note that matrix $\mathcal{F}_i$ represents the appearance rate of new infections in compartment $i$, matrix $\mathcal{Y}_i^-$ represents the transition rate of individuals into compartment $i$, and matrix $\mathcal{Y}_i^+$ represents the transition rate of individuals out of compartment $i$.

(A1) Since $x$ is a vector that represents the variables of system (5) and each variable represents the number of population in each compartment, then it must be true that $\mathcal{F}_i, \mathcal{Y}_i^+$, and $\mathcal{Y}_i^-$ are nonnegative, and $\mathcal{Y}_i^- \geq 0$ for $i = 1, 2, ..., n$. Then, the axiom (A1) is satisfied.

(A2) If the DFE condition holds for system (5), then there will not be any transmission process in the model. In other words, there will not be any infected individuals out of its population. Therefore we obtain that $\mathcal{Y}_i^- = 0$ for $i = 1, 2, ..., m$. Then, the axiom (A2) is satisfied.

(A3) It is clear that $\mathcal{F}_i$ where $i > m$ is the fifth element of the vector $\mathcal{F}_i$ itself, that is $s_h = 0$. Therefore, axiom (A3) is satisfied.
(A4) Substitute $E_0$ to the vector $\mathcal{F}_i$ and $\mathcal{V}_i^+$ with $i = 1, 2, ..., 4$, then we will obtain $\mathcal{F}_i = 0$ and $\mathcal{V}_i^+ = 0$. Therefore, axiom (A4) is satisfied.

(A5) First, substitute $\beta_{h1}\beta_{h2}, \beta_v = 0$ to system (5). Then, we will get
\[
\begin{align*}
\frac{de_h}{dt} &= -(1 + \chi) e_h, \quad \frac{da_h}{dt} = \chi (1 - p) e_h - (\alpha + \gamma + 1) a_h, \\
\frac{di_h}{dt} &= \chi p e_h + \alpha a_h - (\gamma + 1) i_h, \quad \frac{di_v}{dt} = -\frac{1}{\epsilon} i_v, \quad \frac{ds_h}{dt} = (1 - s_h).
\end{align*}
\]

Linearize system (11) around the $E_0$, we obtain
\[
J_2|_{DFE} = \begin{bmatrix}
-1 & 0 & 0 & 0 & 0 \\
0 & -1 - \chi & 0 & 0 & 0 \\
0 & \chi (1 - p) & -\alpha - \gamma - 1 & 0 & 0 \\
0 & \chi p & \alpha & -1 - \gamma & 0 \\
0 & 0 & 0 & 0 & -\frac{1}{\epsilon}
\end{bmatrix}.
\]

By using the equation $|\lambda I - J_2| = 0$, we obtain that all eigenvalues of matrix $J_2|_{DFE}$ have negative real parts. Therefore, axiom (A5) is satisfied.

Since axioms (A1)-(A5) are satisfied, then Theorem 3.4 is proven.

3.5. Endemic equilibrium

Endemic equilibrium point (EE) is defined as a point or condition when the outbreak of the disease occurs in a population. Based on this definition, the endemic equilibrium point of model (1) is produced when the number of individuals infected with Zika do not equal to 0. Then, we obtain the Zika endemic equilibrium point of model (1) is
\[
E_1 = (s_h, e_h, a_h, i_h, i_v) = (s_h^*, e_h^*, a_h^*, i_h^*, i_v^*),
\]
where
\[
\begin{align*}
s_h^* &= \frac{(\beta_v i_v^*(\alpha + \gamma + 1) + p(\gamma + 1) + \alpha)(p(\gamma + 1) + \alpha)}{b_2 i_h^* + b_1 i_h^2 + b_0}, \\
e_h^* &= \frac{i_h^* (\gamma + 1)(\gamma + \alpha + 1)}{\chi (p(\gamma + 1) + \alpha)}, \\
a_h^* &= \frac{i_h^* (\gamma + 1)(1 - p)}{p (\gamma + 1) + \alpha}, \\
i_v^* &= \frac{\beta_v i_v^*(\alpha + \gamma + 1)}{\beta_v i_v^*(\alpha + \gamma + 1) + p(\gamma + 1) + \alpha},
\end{align*}
\]
with $b_2 = \beta_{h2} \beta_v (\alpha + \gamma + 1) (k (1 - p) (\gamma + 1) + \alpha + p(\gamma + 1)), b_1 = (p(\gamma + 1) + \alpha) (k \beta_{h3} (1 - p)(\gamma + 1) + \beta_v (\beta_{h1} + 1)(\alpha + \gamma + 1))$, and $b_0 = (\gamma (1 + p) + \alpha)^2$. Also, we have $i_h^*$ in the equation (14) is the solution of the quadratic equation
\[
a_2 i_h^2 + a_1 i_h + a_0 = 0,
\]
where $a_2, a_1$ and $a_0$ given by
\[
\begin{align*}
a_2 &= -\beta_{h2} \beta_v (\alpha + \gamma + 1)^2, \\
a_1 &= -(\alpha + \gamma + 1)(p(\gamma + 1) + \alpha) (h_1 \beta_v (\beta_{h1} + 1) + h_2 \beta_{h2} + h_3 \beta_{h2} \beta_v), \\
a_0 &= \frac{(\mathcal{R}_0 - 1)(\alpha + \gamma + 1)(\mathcal{R}_0 (1 + \gamma) + \chi \beta_{h1} \beta_v) (p(1 + \gamma) + \alpha)^2}{\mathcal{R}_0},
\end{align*}
\]
Based on the result in equation (22), we get
\[ R \]
\[ \text{can conclude that there will not be found any roots of equation (15) when } \beta \text{ in the following theorem.} \]

R \[ \text{words, there is no endemic equilibrium found when } \beta \text{ since } a_1 \text{ has undetermined value, it is clear that it will be difficult to determine the value of } -\frac{a_2}{a_2} \text{ and } a_1^2 - 4a_2a_2. \text{ So, we need to use another approximation method to determine the roots of equation (15) when } R_0 < 1. \]

First, let \( \beta_{h_2} \) as the parameter of bifurcation that is defined as follows.
\[
\beta_{h_2} = \frac{((1 + \gamma)(1 + \chi)(R_0)^2 - \chi \beta_{h_1} \beta_v)(\alpha + \gamma + 1)}{\chi R_0 (1 + \gamma)(k(1 - p) + p) + \alpha}.
\]

Substitute this \( \beta_{h_2} \) into equation (15), we have
\[
a_2(R_0)i_h^2 + a_1(R_0)i_h + a_0(R_0) = 0,
\]
where \( a_2(R_0) = -\frac{a_2}{\chi R_0}, a_1(R_0) = -\frac{a_2}{\chi R_0}, \) and \( a_0(R_0) = \frac{a_2}{R_0}, \) with
\[
g_1 = \beta_v (\alpha + \gamma + 1)^2 (R_0^2(1 + \gamma)(1 + \chi) + \chi \beta_{h_1} \beta_v)(1 + \gamma)(1 + \chi),
\]
\[
g_2 = (\alpha + \gamma + 1)^2 (p(1 + \gamma) + \alpha) (\chi^2 \beta_{h_1} \beta_v + \chi \beta_{h_1} \beta_v(R_0 - 1)(1 + \chi)(1 + \gamma)
\]
\[ - \chi \beta_v R_0 (R_0 - 1)(1 + \chi)(1 + \gamma) + R_0^2(1 + \chi)^2(1 + \gamma)^2),
\]
\[
g_3 = (R_0 - 1)(\alpha + \gamma + 1)(R_0(1 + \gamma)(1 + \chi) + \chi \beta_{h_1} \beta_v((p(1 + \gamma) + \alpha)^2.
\]

As a simplification of writing, we can rewrite \( a_2(R_0), a_1(R_0), a_0(R_0) \) as \( a_2, a_1, a_0 \) respectively and \( i_h^* \) as \( i_h. \)

Next, determine the derivative of \( i_h \) in equation (15) respect to \( R_0, \) then we get
\[
\frac{\partial a_2}{\partial R_0} \frac{i_h^2}{\partial R_0} + 2i_h \frac{\partial i_h}{\partial R_0} a_2 + \frac{\partial a_1}{\partial R_0} i_h + \frac{\partial a_0}{\partial R_0} a_1 = \frac{\partial a_0}{\partial R_0} a_1 = 0.
\]

Substitute \( R_0 = 1 \) and \( i_h = 0 \) to equation (20), then we obtain
\[
\frac{\partial i_h}{\partial R_0} = -\frac{\partial a_0}{\partial R_0} a_1.
\]

After that, substitute the parameter of bifurcation, \( \beta_{h_2}, \) to equation (21), then we will get
\[
\frac{\partial a_0}{\partial R_0} = \frac{(\alpha + \gamma + 1)(\chi \beta_{h_1} \beta_v + (1 + \chi)(1 + \gamma))(p(\gamma + 1) + \alpha)^2 > 0,}
\]
\[
\iff a_1 = -\frac{(\alpha + \gamma + 1)^2 (\chi^2 \beta_{h_1} \beta_v + (1 + \chi)^2(1 + \gamma)^2)(p(\gamma + 1) + \alpha)}{\chi} < 0.
\]

Based on the result in equation (22), we get \( \frac{\partial a_0}{\partial a_2} < 0. \) Therefore, we obtain \( \frac{\partial i_h}{\partial R_0} > 0. \) So, we can conclude that there will not be found any roots of equation (15) when \( R_0 < 1. \) In other words, there is no endemic equilibrium found when \( R_0 < 1. \) The discussion above is summarized in the following theorem.
Theorem 3.5. The Zika model given by system (5) has:

(i) one unique endemic equilibrium if \( R_0 > 1 \),

(ii) no endemic equilibrium otherwise.

From the discussion above, we have the parameter of bifurcation \( \beta_{h_2} \). By substituting \( R_0 = 1 \), we have

\[
\beta_{h_2*} = \frac{((1 + \gamma) (1 + \chi) - \chi \beta_{h_1} \beta_v) (\alpha + \gamma + 1)}{\chi ((1 + \gamma) (k (1 - p) + p) + \alpha)}. \tag{23}
\]

Then, rewrite all variables in the Zika model (5) as given by

\[
x_1 = s, x_2 = e, x_3 = a, x_4 = i, x_5 = i_v.
\]

So, the system is now presented as follows

\[
\begin{align*}
\frac{dx_1}{d\tau} &= (1 - x_1) - x_1 (\beta_{h_1} x_5 + \beta_{h_2} (x_4 + k x_3)), \\
\frac{dx_2}{d\tau} &= x_1 (\beta_{h_1} x_5 + \beta_{h_2} (x_4 + k x_3)) - (1 + \chi) x_2, \\
\frac{dx_3}{d\tau} &= \chi (1 - p) x_2 - (\alpha + \gamma + 1) x_3, \\
\frac{dx_4}{d\tau} &= \chi p x_2 + \alpha x_3 - (\gamma + 1) x_4, \\
\frac{dx_5}{d\tau} &= \frac{1}{\epsilon} (\beta_v (1 - x_5) (x_4 + x_3) - x_5).
\end{align*}
\tag{24}
\]

Now, evaluate the system (24) at \( E_0 \) with \( \beta_{h_2} = \beta_{h_2*} \) we have

\[
J_{DFE} = \begin{bmatrix}
-1 & 0 & -k \beta_{h_2*} & -\beta_{h_1} & 0 \\
0 & -(1 + \chi) & k \beta_{h_2*} & \beta_{h_2*} & \beta_{h_1} \\
0 & \chi (1 - p) & -\epsilon (\alpha + \gamma + 1) & 0 & 0 \\
0 & \chi p & \alpha & 0 & 0 \\
0 & 0 & \frac{\beta_v}{\epsilon} & \frac{\beta_v}{\epsilon} & -\frac{1}{\epsilon}
\end{bmatrix}, \tag{25}
\]

Using the equation \(|\lambda I - J| = 0\), we obtain the matrix \( J_{DFE} \) has a simple zero eigenvalue and the others are negative. Hence, the method proposed in [22] can be implemented in this model. Following [22], we need to determine the right and left eigenvectors of matrix \( J_{DFE} \) in equation (25) that is denoted by \( \vec{v} = [v_1 \ v_2 \ v_3 \ v_4 \ v_5] \) and \( \vec{w} = [w_1 \ w_2 \ w_3 \ w_4 \ w_5] \) respectively. Then, we will get

\[
v_1 = 0,
\]

\[
v_2 = \frac{\chi (k (1 - p) (\gamma + 1) + p (\gamma + 1) + \alpha)}{\chi \beta_{h_1} \beta_v (1 - p) (k - 1) + (1 + \chi) (k (1 + \gamma) + \alpha)} > 0,
\]

\[
v_3 = \frac{\epsilon \chi \beta_{h_1} \beta_v (k - 1) + (1 + \chi) (k (1 + \gamma) + \alpha)}{\chi \beta_{h_1} \beta_v (1 - p) (k - 1) + (1 + \chi) (k (1 + \gamma) + \alpha)} < 0,
\]

\[
v_4 = 1 > 0,
\]

\[
v_5 = \frac{\chi \beta_{h_1} \beta_v (k - 1) + (1 + \chi) (k (1 + \gamma) + \alpha)}{\chi \beta_{h_1} \beta_v (1 - p) (k - 1) + (1 + \chi) (k (1 + \gamma) + \alpha)} > 0.
\]

and

\[
w_1 = -(1 + \chi) < 0, \quad w_2 = 1 > 0, \quad w_3 = \frac{\chi (1 - p)}{\alpha (\gamma + 1)} > 0,
\]

\[
w_4 = \frac{\chi p (\gamma + 1) + \alpha}{(1 + \gamma) (\alpha (\gamma + 1))} > 0, \quad w_5 = \frac{\chi \beta_v}{(1 + \gamma)} > 0.
\]
Let all the subsystems in the model be expressed as $\frac{dx_i}{d \tau} = f_i$ with $i = 1, 2, 3, 4, 5$. It follows from [22], we can calculate $a$ and $b$ which give us the result as follows.

\[
a = 2 v_2 w_1 \left( w_3 \frac{\partial^2 f_2}{\partial x_1 \partial x_3} + w_4 \frac{\partial^2 f_2}{\partial x_1 \partial x_4} + w_5 \frac{\partial^2 f_2}{\partial x_1 \partial x_5} \right) \\
\quad + 2 v_5 w_5 \left( w_3 \frac{\partial^2 f_5}{\partial x_3 \partial x_5} + w_4 \frac{\partial^2 f_5}{\partial x_5 \partial x_4} \right) \\
= 2 v_2 w_1 \left( w_3 \beta_{h_2, k} + w_4 \beta_{h_2, h_1} + w_5 \beta_{h_1} \right) + 2 v_5 w_5 \left( -\frac{\beta_0}{\epsilon} (w_3 + w_4) \right) < 0,
\]

\[
b = v_2 \left( w_3 \frac{\partial^2 f_2}{\partial x_3 \partial \beta_{h_2}} + w_4 \frac{\partial^2 f_2}{\partial x_4 \partial \beta_{h_2}} \right) \\
= v_2 \left( w_3 k + w_4 \right) > 0.
\]

It can be seen from equation (28) that we get $a < 0$ and $b > 0$. Therefore, in this model, there will not be found any endemic equilibrium if $R_0 < 1$. At the same time, the disease-free equilibrium is locally asymptotically stable. Then, the forward bifurcation phenomenon occurs in this model when $R_0 = 1$. Next, if $R_0 > 1$, the disease-free equilibrium becomes unstable and there appears an endemic equilibrium that is locally asymptotically stable.

**Theorem 3.6.** System (5) has one endemic equilibrium point that is locally asymptotically stable when $R_0 > 1$ but close enough to 1.

It has been stated that there occurs forward bifurcation phenomenon when $R_0 = 1$. We provide the following result in the corollary below.

**Corollary 3.6.1.** The Zika model given by system (5) will have a forward bifurcation when $R_0 = 1$.

The illustration of forward bifurcation itself is given by Figure 2. In Figure 2, the red curve represents the endemic equilibrium points of system (5) whereas the blue curve represents the disease-free equilibrium points of the same system. The solid and dot curves respectively represent the stability and instability of the equilibrium point. Based on Figure 2, it can be seen that there is the change of equilibrium point stability at $R_0 = 1$ when the forward bifurcation occurs. Also, we can conclude that the greater the value of $R_0$, the greater the proportion of $i_h$ will be.

![Figure 2](image.png)

**Figure 2.** Forward bifurcation diagram of system (5).
4. Numerical simulation

4.1. Sensitivity analysis

In this section, we will analyze the change in the value of $R_0$ respect to the change in all parameters $\nu$ that is involved in the model, or is well-known as sensitivity analysis. Next, we will perform the elasticity analysis as well. To perform this experiment, we use parameters value in Table 1 which in non-dimensional version of parameter is given as follows

\[
\beta_{h_1}^* = 19.77, \quad \beta_{h_2}^* = 10, \quad \beta_v^* = 1.25, \quad \chi^* = 4942.1, \quad \alpha^* = 6.18, \quad \gamma^* = 24.71, \quad p = 0.28, \quad k = 2, \quad \epsilon = 0.001.
\]

Therefore, we get the results of $R_0$ elasticity analysis in Table 2.

### Table 1. Description of parameters in model (1).

| Parameter | Description                          | Value  | Unit                  | Reference |
|-----------|--------------------------------------|--------|-----------------------|-----------|
| $\Lambda_h$ | Numbers of human birth             | $\frac{10,000}{365 \times 67.7}$ | human $/ \text{day}$ | [8]       |
| $\beta_{h_1}$ | Infection rate from mosquito to human | 0.008  | vector $/ \text{day}$ | Assumed   |
| $\beta_{h_2}$ | Infection rate from human to human  | 0.0004 | day$^{-1}$            | Assumed   |
| $k$         | Correction factor                   | 2      | –                     | Assumed   |
| $\chi$      | Human incubation rate               | 0.2    | day$^{-1}$            | [7]       |
| $p$         | Proportion of $I_h$                 | 0.28   | –                     | Assumed   |
| $\alpha$    | Individuals transition rate from $A_h$ to $I_h$ | 0.00025 | day$^{-1}$ | Assumed   |
| $\gamma$    | Human recovery rate                 | 0.001  | day$^{-1}$            | [8]       |
| $\mu_h$     | Natural death rate of human         | $\frac{1}{365 \times 67.7}$ | vector $/ \text{day}$ | [8]       |
| $\Lambda_v$ | Numbers of mosquitoes birth         | $\frac{1000}{25}$ | vector $/ \text{day}$ | [8]       |
| $\beta_v$   | Infection rate from human to mosquito | 0.05   | day$^{-1}$            | Assumed.  |
| $\mu_v$     | Natural death rate of mosquito      | $\frac{1}{25}$ | day$^{-1}$ | [7]       |

### Table 2. Results of $R_0$ Elasticity Analysis Respect to All Parameters in Model (5).

| $R_0$ | $\gamma_{R_0}^{\beta_{h_1}}$ | $\gamma_{R_0}^{\beta_{h_2}}$ | $\gamma_{R_0}^{k}$ | $\gamma_{R_0}^{\chi}$ | $\gamma_{R_0}^{p}$ |
|-------|-----------------------------|-----------------------------|-------------------|-------------------|------------------|
| $R_0 > 1$ | 0.4843                      | 0.0313                      | 0.023             | 0.0001            | −0.00045         |
| $R_0 < 1$ | 0.4922                      | 0.0157                      | 0.0115            | 0.0001            | −0.0022          |

| $R_0$ | $\gamma_{R_0}^{\gamma}$ | $\gamma_{R_0}^{\beta_v}$ | $\gamma_{R_0}^{\epsilon}$ | $\gamma_{R_0}^{\epsilon}$ |
|-------|-------------------------|-------------------------|---------------------------|---------------------------|
| $R_0 > 1$ | −0.0022                 | −0.4934                 | 0.4843                    | 0                          |
| $R_0 < 1$ | −0.0011                 | −0.487                  | 0.4922                    | −                          |

Based on the result that is presented by Table 2 we get that if $R_0 > 1$ then $\gamma$ becomes the most influential parameter in this model. It can be seen that $\gamma$ is inversely proportional to $R_0$, which means the greater the value of $\gamma$, the smaller the value of $R_0$ will become. In this case, $\gamma_{R_0}^{\gamma} = −0.4934$ means for every 1% increase in human recovery rate, it will cause the value of
Figure 3. Sensitivity height curve of $R_0$ respect to $\beta_{h_1}$ and $\gamma$

Figure 4. Sensitivity curve of $R_0$ respect to $\beta_{h_1}$ and $\gamma$ in 3 different areas

$R_0$ to decrease by 0.4934%. Next, if $R_0 < 1$ then $\beta_{h_1}$ becomes the most influential parameter in this model. It can also be seen that $\beta_{h_1}$ is directly proportional to $R_0$, which means the greater the value of $\beta_{h_1}$, the greater the value of $R_0$ will become. In this instance, $\frac{\gamma^{\beta_{h_1}}}{R_0} = 0.4922$ can be interpreted as for every 1% increase in the infection rate from mosquito to human, it will cause 0.4922% decrease in $R_0$ value.

As stated before, $\gamma$ and $\beta_{h_1}$ are the most influential parameters to the change of $R_0$ value. So, we can plot the sensitivity height curve of $R_0$ in terms of $\beta_{h_1}$ and $\gamma$ as shown in Figure 3.

Moreover, we will plot the curve of $R_0 = 1$ to investigate the state of disease spread based on the value of $(\beta_{h_1}, \gamma)$ that is fitted. First, we have

$$R_0(\beta_{h_1}, \gamma) = \frac{4.999(7.90 + 1.72\gamma)}{(1 + \gamma)(7.178 + \gamma)} + \frac{1}{2} \sqrt{\frac{4.999\beta_{h_1}}{(1 + \gamma)} + 99.96 \frac{(7.90 + 1.72\gamma)^2}{(1 + \gamma)^2(7.178 + \gamma)^2}}. \tag{29}$$

Using the equation (29), we plot $R_0 = 1$ as shown in Figure 4. From the simulation result above, we can see that area I and area II are the areas that depend on both $\gamma$ and $\beta_{h_1}$ in resulting on $R_0$ value. In this case, we need to use $\gamma$ more than 14.107 to be able to get a value $R_0 < 1$. The higher the infection rate from human to mosquito, the higher the recovery rate we should get to prevent the Zika endemicity. Using Figure 4, we can also see that it is possible the Zika endemicity will always exist no matter how small the infection rate is. This will happen when the human recovery rate is less than 14.107.

4.2. Autonomous simulation

In this part, we will use parameter values that is given by Table 3 (unless stated differently) with the initial values of each compartment of system (5) given by Table 4.
Table 3. Non-dimensional Parameter Value

| Parameter | \( \beta_{h_1} \) | \( \beta_{h_2} \) | \( k \) | \( \chi \) | \( p \) | \( \alpha \) | \( \gamma \) | \( \beta_v \) | \( \epsilon \) |
|-----------|-----------------|-----------------|------|------|------|------|------|------|------|
| Value     | 9               | 10              | 2    | 4942.1 | 0.28 | 6.18 | 24.71 | 1.25 | 0.001 |

Table 4. Initial Value of Each Non-Dimensional Compartment

| Compartment | \( s_h \) | \( e_h \) | \( a_h \) | \( i_h \) | \( i_v \) |
|-------------|----------|----------|----------|----------|----------|
| Initial Value | 0.4      | 0.3      | 0.2      | 0.1      | 0.4      |

Table 5. Autonomous simulation result for four various combination \( \beta_{h_1} \) and \( \gamma \) values

| Case | \( \beta_{h_1} \) | \( \gamma \) | \( R_0 \) | \( s_h \) | \( e_h \) | \( a_h \) | \( i_h \) | \( i_v \) |
|------|-----------------|------|--------|------|------|------|------|------|
| 1    | 8               | 10   | 1.83   | 0.825 | 3.5 \times 10^{-5} | 0.005 | 0.004 | 0.001 |
| 2    | 8               | 20   | 1.154  | 0.825 | 3.5 \times 10^{-5} | 0.005 | 0.004 | 0.01  |
| 3    | 8               | 24.71| 1.003  | 0.997 | 6.9 \times 10^{-7} | 7.7 \times 10^{-5} | 5.5 \times 10^{-5} | 1.6 \times 10^{-4} |
| 4    | 8               | 30   | 0.882  | 1     | 0    | 0    | 0    | 0     |

The first autonomous simulation result that is given by Figure 5 shows the effect of the combination between the recovery and infection rate to the dynamic of each proportion in model (5). The values for each parameter that are used in this simulation are based on the areas that have been described by Figure 4. It can be seen from Table 5 that each case has successfully described each area in Figure 4; case 1 which represents area III, we obtain \( R_0 > 1 \) and it gives us the conclusion that the Zika disease will always occur in the population, we also obtain \( R_0 > 1 \) by using the value of parameters given by case 2-3 which represent area II and it also gives us the conclusion that Zika disease will always exist in the population, lastly we obtain \( R_0 < 1 \) by using parameter value given by case 4 which represent area I and it concludes that the Zika disease will disappear over time.

In this paper, we involve transition cases from asymptomatic to symptomatic phase that is denoted by parameter \( \alpha \). So, we need to know the effect of this parameter to the dynamics of the model. By using the initial value of each compartment that is stated in Table 4 and parameters value that is stated in Table 3, we will get the result as can be seen in Table 6 that gets visualized by Figure 6. It is clear to see that the greater the value of \( \alpha \) will increase the proportion of symptomatic infected humans, whereas the proportion of asymptomatic infected humans will decrease. On the other hand, the increase of \( \alpha \) also results in decreasing the \( R_0 \) value, which adds up to the disappearance of Zika disease in the population overtime.

Lastly, we conduct the autonomous simulation for four various \( \beta_{h_2} \) values to see the effect of infection rate among humans. It is shown in Figure 7 that the lower the infection rate among humans results in the proportion of infected humans and mosquitoes to decrease. On the other hand, the lower the infection rate among humans also causes the proportion of susceptible
Figure 5. Dynamic of proportion: (a). Susceptible human \( (s_h) \), (b). Exposed human \( (e_h) \), (c). Asymptomatic infected human \( (a_h) \), (d). Symptomatic infected human \( (i_h) \), (e). Number of infected humans \( (e_h + a_h + i_h) \), (f). Infected mosquito \( (i_v) \).

Table 6. Autonomous simulation result for six various \( \alpha \) value

| Case | \( \alpha \) | \( R_0 \) | \( s_h \) | \( e_h \) | \( a_h \) | \( i_h \) | \( i_v \) |
|------|-------------|----------|--------|--------|--------|--------|--------|
| 1    | 6           | 1.04     | 0.951  | \( 9.8 \times 10^{-6} \) | 0.001  | \( 7.9 \times 10^{-4} \) | 0.002  |
| 2    | 8           | 1.03     | 0.962  | \( 7.6 \times 10^{-6} \) | 0.001  | \( 6.6 \times 10^{-4} \) | 0.001  |
| 3    | 10          | 1.02     | 0.973  | \( 5.4 \times 10^{-6} \) | \( 5.3 \times 10^{-4} \) | \( 5.0 \times 10^{-4} \) | 0.001  |
| 4    | 12          | 1.01     | 0.983  | \( 3.4 \times 10^{-6} \) | \( 3.1 \times 10^{-4} \) | \( 3.2 \times 10^{-4} \) | 0.001  |
| 5    | 14          | 1        | 0.99   | \( 1.5 \times 10^{-6} \) | \( 1.3 \times 10^{-4} \) | \( 1.5 \times 10^{-4} \) | \( 3.6 \times 10^{-4} \) |
| 6    | 16          | 0.99     | 1      | 0      | 0      | 0      | 0      |
| 7    | 18          | 0.99     | 1      | 0      | 0      | 0      | 0      |

humans to increase. From Table 7, it is clear to see that the lower the infection rate among humans, the smaller the value of \( R_0 \) we will get. In case 4, we get \( R_0 < 1 \) which means by using the parameter values for this case, the Zika disease will disappear over time.

5. Discussion, conclusions and future work

In this manuscript, the authors modify the Zika transmission model by authors in [7] by adding the transition rate from asymptomatic to symptomatic individuals. Mathematical analysis conducted to find and analyze the existence and local stability of all equilibrium points. The forward bifurcation occurs when the basic reproduction number equal to one, which presents the change of stability of the disease-free equilibrium with the existence and local stability of
Figure 6. Dynamic of proportion: (a). Asymptomatic infected human \( (a_h) \) and (b). Symptomatic infected human \( (i_h) \).

Table 7. Autonomous simulation result for four various \( \beta_{h_2} \) value

| Case | \( \beta_{h_2} \) | \( R_0 \) | \( s_h \) | \( e_h \) | \( a_h \) | \( i_h \) | \( i_v \) |
|------|-----------------|--------|--------|--------|--------|--------|--------|
| 1    | 14              | 1.22   | 0.773  | \( 4.5 \times 10^{-5} \) | 0.005  | 0.004  | 0.011  |
| 2    | 12              | 1.13   | 0.853  | \( 2.9 \times 10^{-5} \) | 0.003  | 0.002  | 0.007  |
| 3    | 10              | 1.03   | 0.951  | \( 9.8 \times 10^{-6} \) | 0.001  | 0.001  | 0.002  |
| 4    | 8               | 0.99   | 1      | 0      | 0      | 0      | 0      |

the endemic equilibrium. Our elasticity analysis on the basic reproduction number shows that the recovery rate is the most significant parameter in determining \( R_0 \) when it is less than unity and transmission rate when \( R_0 \) larger than unity.

Our analysis of the transition rate from asymptomatic to symptomatic class is less sensitive to determine the size of \( R_0 \). Since the sign of sensitivity is negative, we have that increasing the transition rate to symptomatic class will decrease \( R_0 \). The reason possibly because the lesser the number of asymptomatic individual, the easier to trace the infection in the community. Finally, sample simulations were presented to show the effect of transition rate from asymptomatic to symptomatic could reduce the number of the infected individual. Several ways could be used to increase this rate, such as using the medical test to the community, to trace and detect the existence of the infected individual.

For future work, the model in this article could be redefined as an optimal control problem by introducing some interventions which depending on time. On the optimal control problem for system (1), we want to minimize the number of infected compartment \( E_h, I_h, A_h, \) and \( I_v \) by using a minimum cost. For example, by introducing \( u_1(t) \) as the time-dependent for medical
Figure 7. Dynamic of proportion: (a). Susceptible human ($s_h$), (b). Exposed human ($e_h$), (c). Asymptomatic infected human ($a_h$), (d). Symptomatic infected human ($i_h$), (e). Number of infected humans ($e_h + a_h + i_h$), (f). Infected mosquito ($i_v$).

test to transfer $A_h$ to $I_h$, and $u_2(t)$ as the fumigation to reduce the number of mosquitoes, the cost function to describe above problem could be defined as follows (as an example):

$$J(u_1, u_2) = \int_{t=0}^{t_f} (E_h + I_h + A_h + I_v + c_1 u_1^2 + c_2 u_2^2) \, dt,$$

where $t_f$ is the final time, and $c_1, c_2$ is the weight parameter. Please see [14, 17, 18, 20, 23, 24, 25] to see some implementation of optimal control problem in some epidemiological models.

Acknowledgments

This research is financially supported by Universitas Indonesia with PUTI proceeding research grant scheme, 2020 (ID Number : NKB-932/UN2.RST/HKP.05.00/2020).

References

[1] Agumadu V C and Ramphul K 2018 Zika virus: a review of literature Cureus 10(7)
[2] World Health Organization 2018 Zika Virus https://www.who.int/news-room/fact-sheets/detail/zika-virus
[3] Duong V, Dussart P and Buchy P 2017 Zika virus in asia International Journal of Infectious Diseases 54 121-8
[4] World Health Organization 2016 Zika Strategic Response Framework & Joint Operations Plan https://www.who.int/emergencies/zika-virus/strategic-response-framework.pdf
[5] Kermack W O and McKendrick A G 1927 A contribution to the mathematical theory of epidemics Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character 115(772) 700-21
[6] Jamboos J, Munoz D, Munoz A, Manrique O and Raigosa S 2018 Simulation model to the zika virus considering asymptomatic population Open Journal of Modelling and Simulation 6(1) 1-12
[7] Padmanabhan P, Seshaiyer P and Castillo-Chavez C 2017 Mathematical modeling, analysis and simulation of the spread of zika with influence of sexual transmission and preventive measures Letters in Biomathematics, 4(1)
[8] Khan M A, Shah W S, Ullah S and Gomez-Aguilar J F 2016 A dynamical model of asymptomatic carrier zika virus with optimal control strategies Nonlinear Analysis: Real World Applications 50 144-70
[9] Centers for Disease Control and Prevention 2012 Lesson 1: Introduction to Epidemiology
https://www.cdc.gov/csels/dsepd/ss1978/lesson1/section1.html

[10] Sakkas H, Bozidis P, Giannakopoulos X, Sofikitis N and Papadopoulou C 2018 An update on sexual transmission of zika virus Pathogens 7(3) 66

[11] Diekmann O, Heesterbeek J A P and Roberts M G 2010 The construction of next-generation matrices for compartmental epidemic models Journal or The Royal Society 7(47) 87-885

[12] Malik M, Larasati M and Aldila D 2018 Mathematical modeling and numerical simulation of tuberculosis spread with diabetes effect Journal of Physics: Conference Series 1108(1)

[13] D. Aldila, S. H. Khoshnaw, E. Safitri, Y. R. Anwar, A. R. Bakry, B. M. Samiadji, et al., A mathematical study on the spread of covid-19 considering social distancing and rapid assessment : The case of jakarta, indonesia, Chaos Solitons Fractals, 139 (2020), 110042.

[14] Aldila D, Nuraini N and Soewono E 2014 Optimal control problem in preventing of swine flu disease transmission Applied Mathematical Sciences (69-72) 8 3501-12

[15] Aldila D, Nuraini N and Soewono E 2015 Mathematical model in controlling dengue transmission with sterile mosquito strategies AIP Conference Proceedings vol 1677 p 030002

[16] Aldila D, Aprilliani R R and Malik M 2018 Understanding HIV spread with vertical transmission through mathematical model AIP Conference Proceedings vol 2023 p 020213

[17] Aldila D, Handari B D, Widyah A and Hartanti G 2020 Strategies of optimal control for HIV spreads prevention with health campaign Communications in Mathematical Biology and Neuroscience 2020 7

[18] D. Aldila, M.Z. Ndii, B. M. Samiadji. (2020), Optimal control on COVID-19 eradication program in Indonesia under the effect of community awareness, Math. Biosc. and Engg. 17(6): 63556389.

[19] D. Aldila, Cost effectiveness analysis and backward bifurcation analysis on COVID-19 transmission model considering direct and indirect transmission, Commun. Math. Biol. Neurosci. 2020, 2020:X, https://doi.org/10.28919/cmbn/4779.

[20] Handari B D, Vitra F, Ahya R, Nadya S T and Aldila D 2019 Optimal control in a malaria model: intervention of fumigation and bed nets Advances in Difference Equations 2019(1) 497

[21] Van den Driessche P and Watmough J 2002 Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission Mathematical biosciences 180(1-2) 29-48

[22] Castillo-Chavez C and Song B 2004 Dynamical models of tuberculosis and their applications Mathematical Biosciences & Engineering 1(2) 361

[23] Rohman M I S, Handari B D and Aldila D 2018 An impulse fumigation scenario to control dengue spreads AIP Conference Proceedings vol 2023 p 020213

[24] Hafidh E P, Aulida N, Handari B D and Aldila D 2018 Optimal control problem from tuberculosis and multidrug resistant tuberculosis transmission model AIP Conference Proceedings vol 2023 p 020223

[25] Aldila D, Nareswari K and Tasman H 2018 An optimum control model for resistance fumigation for dengue AIP Conference Proceedings vol 2021 p 060001