Dynamical Analysis and Optimal Control in Zika Disease Transmission Considering Symptomatic and Asymptomatic Classes

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***Abstract*—Mosquito bites from the genus Aedes spread the Zika virus to humans, which can be transmitted through sexual contact and blood transfusions. This study formulated and analyzed a mathematical model for the virus in human and mosquito populations. Based on nonlinear incidence, the infected population is divided into two, namely symptomatic and asymptomatic. The existence and stability of the model equilibriums are based on the reproduction ratio. Furthermore, the stable local endemic and non-endemic equilibrium point is $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$, respectively. The significant parameter affects the number of symptomatic and asymptomatic infections. It was determined using sensitivity analysis. Also, control efforts were made to reduce transmission rates by eradicating mosquito populations using insecticides, reducing direct contact with mosquitoes, and direct routine health checks. The Pontryagin Maximum Principle showed that the three control strategies can significantly reduce the number of infected individuals.**

***Index Terms*—Zika Virus, Nonlinear Incidence, Stability Analysis, Optimal Control.**

I. Introduction

ZIKA is a flavivirus in the Flaviviridae family transmitted by Aedes genus mosquitoes to humans. This virus was discovered on a monkey in Zika Forest, Uganda 1947 and the first Nigerian population was infected in 1952 [1]. The symptoms are similar to an arboviral disease, such as dengue fever, chikungunya, West Nile virus, and others. Also, the primary symptoms include mild fever, headache, arthralgia, myalgia, conjunctivitis, and skin rashes [2]. Although the symptoms are mild, neurological complications, including GBS (Guillan-Barre Syndrome) and microcephaly´ transmitted from an infected mother to the fetus during her birth, can occur [3], [4], [5]. The most significant case of the Zika virus outbreak in around 30,000 cases was reported by French Polynesia during 2013 - 2014. Furthermore, the virus spread rapidly in South America 2015, particularly in Brazil and Colombia [6]. Several studies were conducted to maintain the survival of humans and other creatures [7], [8], [9], [10], [11], [12]. The disease transmission between humans occurs through blood transfusion and sexual intercourse. These transmissions may show no symptoms of the infection as a whole and are significant. Hence the infected individual can transmit it to healthy ones [8], [13]. Also, this virus can be transmitted vertically from infected pregnant women, through their uterus, to the fetus, which may experience microcephaly [14]. The new case showed that some patients do not experience symptoms [8], and 80% are asymptomatic [15]. Furthermore, transitioning from asymptomatic to symptomatic cases is essential in spreading Zika virus disease [16].

Several case studies were conducted to investigate Zika disease, developing mathematical models for the disease spread [17], [18], [19], [20], [21], [22], [23], [24], [25], [26], [27], [28], [29], [30], [31], [32], [33], [34], [35], [36], [37], [38]. Goswami, et.al. [10] studied the disease transmission between humans and vectors with humans in 2018, where the vectors such as carry and spread the disease. In this case, using a nonlinear incidence rate, the genus Aedes mosquito shows a psychological effect where humans limit contact when there are many infections, thereby decreasing transmission (saturation). Meanwhile, [39] examined cases of dengue fever with and without symptoms and showed that reinfection could occur in naturally cured humans due to decreased immunity.

This study discussed the transmission and stability of the Zika disease, transmitted by mosquito vectors to humans and from humans to humans. N. Anggriani, et al. [39] and D. Adila, et al. [16] made a model in line with this study, without a reduction in immunity and reinfection similar to [40]. Only asymptomatic humans are assumed to transmit the virus when the incidence rate is not linear. In addition, using optimal controls reduces the widespread spread of Zika disease. The Pontriagin Maximum Principle method is used to determine the optimal control value. This research provides new insights into the dynamics of Zika virus infection.

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TABLE I
PARAMETERS THAT AFFECT MODEL FORMATION

Parameters	Descriptions
Λ_h	Recruitment rates in human populations
Λ_v	Recruitment rate in vector populations (mosquitoes)
μ_h	Natural mortality rate in human population
μ_1	Human mortality rate due to infection
μ_v	The natural mortality rate in the human population
b	Vector-human bite rate
β_{h1}	The probability of human-to-human transmission
β_{h2}	The probability of mosquito transmission to humans
β_v	The probability of transmission of humans to vectors
γ_h	Recovery rate
p	The proportion of healthy humans who become infected without symptoms due to human-to-human transmission
q	The proportion of healthy humans who become infected without symptoms due to mosquito bites

II. FORMULATION OF THE MODEL

This section explores a mathematical model to describe Zika's dynamic behavior, consisting of six ordinary differential equations (ODE). The population of humans is divided into four compartments, including susceptible (S_H), asymptomatic $(I_{H,a})$, symptomatic $(I_{H,s})$, and recovery humans (R_H). Meanwhile, the vector population is divided into two compartments: susceptible (S_V) and infected vectors (I_V).

The mathematical model for the spread of Zika disease is based on the assumption that transmission occurs from humans to vectors, vectors to humans, and humans to humans, with a nonlinear incidence rate. Hence, a change in contact between susceptible and infected humans is required. Figure 1 illustrates this model using a diagram of the interaction between the two populations due to a large number of infections.

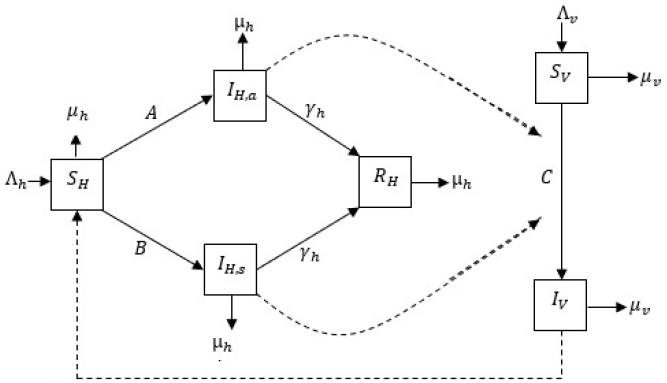

Fig. 1. Interaction Diagram of Human Populations and Mosquitoes

The model can be illustrated through an interaction diagram between two populations based on the assumptions above, as in Figure 1. where

$$A = p\frac{\beta_{h1}cI_{H,a}}{1+I_{H,a}} + q\frac{b\beta_{h2}I_V}{N_H}$$

$$B = (1-p)\frac{\beta_{h_1}cI_{H,a}}{1+I_{H,a}} + (1-q)\frac{b\beta_{h,2}I_V}{N_H}$$

$$C = \frac{b\beta_V(I_{H,a}+I_{H,s})}{N_H}$$

According to the interaction diagram, the spread of the Zika virus among the humans and mosquito populations is as follows:

$$\frac{dS_H}{dt} = \Lambda_h - \frac{\beta_{h1}cI_{H,a}}{1+I_{H,a}}S_H - \frac{b\beta_{h2}I_V}{N_H}S_H - \mu_h S_H \quad (1)$$

$$\frac{dI_{H,a}}{dt} = p\frac{\beta_{h1}cI_{H,a}}{1+I_{H,a}}S_H + q\frac{b\beta_{h2}I_V}{N_H}S_H - (\mu_h+\mu_1+\gamma_h)I_{H,a} \quad (2)$$

$$\frac{dI_{H,s}}{dt} = (1-p)\frac{\beta_{h1}cI_{H,a}}{1+I_{H,a}}S_H + (1-q)\frac{b\beta_{h2}I_V}{N_H}S_H - (\mu_h+\mu_1+\gamma_h)I_{H,s} \quad (3)$$

$$\frac{dR_H}{dt} = \gamma_h(I_{H,a}+I_{H,s}) - \mu_h R_H \quad (4)$$

$$\frac{dS_V}{dt} = \Lambda_V - \frac{b\beta_V(I_{H,a}+I_{H,s})}{N_H}S_V - \mu_v S_V \quad (5)$$

$$\frac{dI_V}{dt} = \frac{b\beta_V(I_{H,a}+I_{H,s})}{N_H}S_V - \mu_v I_V \quad (6)$$

The model showed that only asymptomatic infected humans transmit the virus, through blood transfusions or sexual intercourse.

III. BASIC ANALYTICAL RESULT

The basic analysis results from models (1) - (6) showed the positively and boundedness of the model solution, equilibrium point, basic reproduction number, stability of the disease-free case, global sensitivity, and optimal control problem.

1) Non-endemic equilibrium point: This model is obtained by solving (1)-(6), setting $I_{Ha} = 0, I_{Hs} = 0, R_H = 0, I_V = 0$, and substituting into (1)-(6) to obtain:

$$E_0 = (S_H, I_{Ha}I_{Hs}, R_H, S_V, I_V) = \left(\frac{\lambda}{\mu_h}, 0, 0, 0, \frac{\lambda}{\mu_v}, 0\right)$$

2) Basic reproduction ratio: The number of secondary infections caused by primary infections in the population is relevant in epidemiology. Furthermore, the Basic Reproduction Ratio $(\mathcal{R}_0))$ is obtained by using the next generation method to determine the most dominant eigenvalues FV^{-1} symbolized by $\xi(FV^{-1})$.

$$F = \begin{bmatrix} \frac{cp\beta_{h1}\Lambda_h}{\mu_h} & 0 & \frac{bp\beta_{h2}\Lambda_h}{N_H\mu_h} \\ \frac{c(1-p)\beta_{h1}\Lambda_H}{\mu_h} & 0 & \frac{b(1-p)\beta_{h2}\Lambda_h}{N_H\mu_h} \\ \frac{b\beta_V\Lambda_V}{N_H\mu_V} & \frac{b\beta_V\Lambda_V}{N_H\mu_V} & 0 \end{bmatrix}$$

$$V = \begin{bmatrix} (\gamma_h+\mu_1+\mu_h) & 0 & 0 \\ 0 & (\gamma_h+\mu_1+\mu_h) & 0 \\ 0 & 0 & \mu_V \end{bmatrix}$$

$$F = \begin{bmatrix} \frac{cp\beta_{h1}\Lambda_h}{\mu_h} & 0 & \frac{bp\beta_{h2}\Lambda_h}{N_H\mu_h} \\ \frac{c(1-p)\beta_{h1}\Lambda_H}{\mu_h} & 0 & \frac{b(1-p)\beta_{h2}\Lambda_h}{N_H\mu_h\mu_V} \\ \frac{b\beta_V\Lambda_V}{N_H(\gamma_h+\mu_1+\mu_h)\mu_V} & \frac{b\beta_V\Lambda_V}{N_H(\gamma_h+\mu_1+\mu_h)\mu_V} & 0 \end{bmatrix}$$

Where F and V are the Jacobian matrix of f (newly infected matrix) and v (exit matrix), respectively, evaluated at

the disease-free equilibrium point. The $(\mathcal{R}_0))$ for the model of the disease spread in humans and mosquito populations is obtained using Castillo-Chavez et al. [41] method, as follows:

$$\mathcal{R}_0 = \frac{1}{2}\left(\mathcal{R}_{0hh} + \sqrt{4\mathcal{R}_{0hv} + (\mathcal{R}_{0hh})^2}\right)$$

with

$$\mathcal{R}_{0hh} = \frac{cp\beta_h 1\lambda_h}{\mu_h(\gamma_h + \mu_1 + \mu_h)}$$

$$\mathcal{R}_{0hv} = \frac{b^2\beta_{hv}\beta_v\lambda_v\lambda_h}{N_H^2\mu_H^2\mu_h(\gamma_h + \mu_1 + \mu_h)}$$

where $\mathcal{R}_{0hh}$ are R_0 for transmission of the disease between humans and humans and mosquitoes.

3) Endemic Equilibrium Points: The endemic point for a compilation of this disease is endemic in certain areas for a certain period, which releases the Zika virus in humans and mosquitoes. For example $S_H^*, I_{H,a}^*, I_{H,s}^*, R_H^*, S_V^*$ are solutions for $S_H, I_{H,a}, I_{H,s}, R_H, S_V$ from models (1) - (5), using the Wolfram Mathematica 11.3 software, $S_H, I_{H,a}, I_{H,s}, R_H, S_V = S_H^*, I_{H,a}^*, I_{H,s}^*, R_H^*, S_V^*$ is accepted. Furthermore, the point above is substituted into equation (6) so that it is obtained

$$AI_v^2 + BI_v + C = 0 \tag{7}$$

with

$$\begin{aligned} A =& 2b\beta_{h_2}\mu_v(b\beta_v - p\mu_v N_H) \\ & (b\beta_v\Lambda_h + N_H(\gamma_h + \mu_1 + \mu_h)\mu_v) \end{aligned} \tag{8}$$

$$\begin{aligned} B =& -2b^3\beta_{h_2}\beta_v^2\Lambda_h\Lambda_v - b^2N_H\beta_{h_2}\beta_v\Lambda_v(\gamma_h - p\Lambda_h \\ & + \mu_1 + \mu_h)\mu_v + 2bN_H^2\beta_v(-cp\beta_{h_1}\Lambda_h \\ & + \mu_h(\gamma_h + \mu_1 + \mu_h))\mu_v^2 - 2N_H^3p(c\beta_{h_1} + \mu_h) \\ & (\gamma_h + \mu_1 + \mu_h)\mu_v^3 \end{aligned} \tag{9}$$

$$\begin{aligned} C =& bN_H\beta_v\Lambda_v(cN_Hp\beta_{h_1}\Lambda_h - N_H\mu_h(\gamma_h + \mu_1 + \mu_h) \\ & + (b^2I_V^2\beta_{h_2}^2(\gamma_h + p\Lambda_h + \mu_1 + \mu_h)^2 \\ & + N_H^2(cp\beta_{h_1}\Lambda_h - \mu_h(\gamma_h + \mu_1 + \mu_h))^2 \\ & + 2bI_VN_H\beta_{h_2}(\gamma_h + p\Lambda_h + \mu_1 + \mu_h) \\ & (cp\beta_{h_1}\Lambda_h + \mu_h(\gamma_h + \mu_1 + \mu_h)))^{\frac{1}{2}})\mu_v \end{aligned} \tag{10}$$

Then a positive I_V^* root is ensured if $b\beta_{h_2}p\mu_vN_H$ and $cp\beta_{h1}\lambda_H < \mu_h(y_h + \mu_1 + \mu_h)$. Based on the conditions, equation (8) has a single positive root, I_V^*, hence an endemic equilibrium point is obtained, E_1.

4) Stability Analysis: Theorem 1. the non-equilibrium point (E_0) is asymptotically stable locally when $R_0 < 1$, and unstable when $R_0 > 1$. Proof of Theorem 1. Diekmann and Heesterbeek's [42] method and from (1)-(6), the characteristic equation of the Jacobian matrix at the disease-free equilibrium point is:

$$\begin{aligned} P(\lambda) = \frac{1}{N_H\mu_v}(\lambda+\mu_h)^2(\lambda+(\gamma_h+\mu_1+\mu_h))(-b^2\beta_{h2}\beta_v\Lambda_v + \\ N_H(\lambda - cN_Hp\beta_{h1} + \gamma_h + \mu_1 + \mu_h)\mu_v(\lambda + \mu_v)) \end{aligned} \tag{11}$$

From the above equation obtained: $\lambda = \{-\mu_h, -\mu_v, -(\gamma_h + \mu_1, \mu_h), -\mu_v\}$ and $A\lambda^2 + B\lambda + C = 0$, where

$$\begin{aligned} A &= N_H^2\mu_h\mu_v \\ B &= N_H^2\mu_v(\mu_h(\gamma_h + \mu_1 + \mu_h) - cp\beta_{h1}\Lambda_h) \\ C &= N_H^2(-cp\beta_{h1}\Lambda_h + \mu_h((\gamma_h + \mu_1 + \mu_h)))\mu_v \\ &\quad - b^2\beta_{h2}\beta_v\Lambda_h\Lambda_v \end{aligned}$$

According to the Routh-Hurwitz stability criteria, $A, B, C > 0$ and $BC > 0$ if $\mathcal{R}_0 < 1$ where $\mathcal{R}_0 = \mathcal{R}_{0hh} + \mathcal{R}_{0hv}$, the non-equilibrium $E_0 = S_H^*, I_{H,a}^*, I_{H,s}^*, R_H^*, S_V^*$ point is asymptotically stable locally.

IV. Sensitivity Analysis

Latin Hypercube Sampling (LHS) method and Partial Rank Correlation Coefficient (PRCC) method were used to show model sensitivity [43]. Furthermore, 3,000 samples were used to measure the increase in symptomatic and asymptomatic infections, and each parameter was assumed to be between 0 and 1. The results were shown in Figures 2 and 3, respectively.

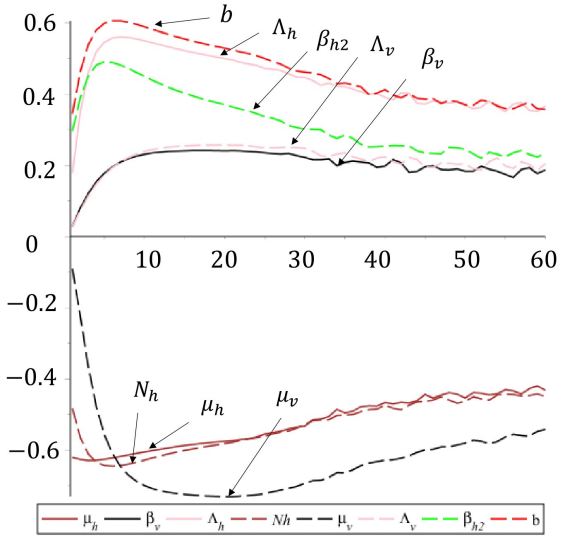

Fig. 2. PRCC analysis plots for the model. The PRCC analysis is calculated by increasing number of symptomatic infection

Figure 2 showed the influential parameters for all time, including $b, \lambda_h, \beta_{h1}, \lambda_v, \beta_v, N_h, \mu_h$, and μ_v. These parameters are sensitive to an increase or decrease in symptomatic infections. The first five parameters are positively related, indicating that the number of symptomatic infections rises as the value of $b, \lambda_h, \beta_{h1}, \lambda_v or \beta_v$ increases. Meanwhile, the other three parameters have a negative relationship. As the $N_h, \mu_h, and \mu_v$ values increase, the number of symptomatic infections decreases.

Figure 3 showed that the influence parameters for all times are $b, \lambda_h, \beta_{h1}, \lambda_v, \beta_v, q, N_h, \mu_h$, and μ_v. The first six parameters have a positive relationship, indicating that an increase in values, increases the number of asymptomatic infections. The other three parameters have an inverse relationship, where an increase in values decreases asymptomatic infections.

Figures 2 and 3 demonstrated that parameters β_{h2} and β_v significantly affect the number of asymptomatic and

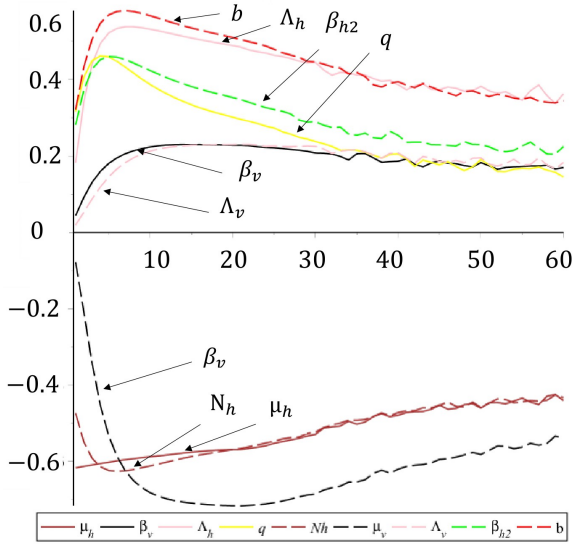

Fig. 3. PRCC analysis plots for the model. The PRCC analysis is calculated by increasing number of symptomatic infection

symptomatic infections and parameter β_{h1} does not. This shows that the spread of the Zika disease, based on the (1)-(6) model, is influenced by transmission from vectors than from humans. Also, the rate of humans bitten by the vector, parameter b, showed a positive association between the epidemic of the disease and the increase in asymptomatic and symptomatic infections.

V. Optimal Control Problem

This section expanded the mathematical model of the disease spread by adding optimal control problems using three control variables, namely u_1, u_2, and u_3. The control variable u_1 represents efforts to reduce mosquito bites, such as anti-mosquito lotions and sprays, electronic devices to repel mosquitoes, and mosquito nets. Then, the control variable $u_2$represents an attempt to increase the rate of mosquito mortality. The control variable u_3 maximizes the effect of holding regular health check counseling, such as an appeal to check the health of individuals returning from endemic areas or check health before participating in blood donation. Checking health every six months minimizes the occurrence of Zika disease transmission from infected humans without symptoms to other healthy humans. Based on these assumptions, the optimal control model for this problem is:

$$\frac{dS_H}{dt} = \Lambda_h - (1-u_3)\frac{\beta_{h1}cI_{H,a}}{1+I_{H,a}}S_H - (1-u_1)\frac{b\beta_{h2}I_V}{N_H}S_H - \mu_h S_H \tag{12}$$

$$\frac{dI_{H,a}}{dt} = p(1-u_3)\frac{\beta_{h1}cI_{H,a}}{1+I_{H,a}}S_H + q(1-u_1)\frac{b\beta_{h2}I_V}{N_H}S_H - (\mu_h+\mu_1+\gamma_h)I_{H,a} \tag{13}$$

$$\frac{dI_{H,s}}{dt} = (1-p)(1-u_3)\frac{\beta_{h1}cI_{H,a}}{1+I_{H,a}}S_H - (\mu_h+\mu_1+\gamma_h)I_{H,s} + (1-q)(1-u_1)\frac{b\beta_{h2}I_V}{N_H}S_H \tag{14}$$

$$\frac{dR_H}{dt} = \gamma_h(I_{H,a}+I_{H,s}) - \mu_h R_H \tag{15}$$

$$\frac{dS_V}{dt} = \Lambda_V - \frac{b\beta_V(I_{H,a}+I_{H,s})}{N_H}S_V - (\mu_v+u_2)S_V \tag{16}$$

$$\frac{dI_V}{dt} = \frac{b\beta_V(I_{H,a}+I_{H,s})}{N_H}S_V - (\mu_v+u_2)I_V \tag{17}$$

with boundary conditions

$$\begin{aligned} &0<t<t_f, 0<u_i<1, \forall i.\\ &S_H(0)=S_{H0}\geq 0, I_{H,a}(0)=I_{H,a0}\geq 0,\\ &I_{H,s}(0)=I_{H,s0}\geq 0, R_H(0)=R_{H0}\geq 0,\\ &S_V(0)=S_{V0}\geq 0, I_V(0)=I_{V0}\geq 0 \end{aligned} \tag{18}$$

$$J=\int_0^{tf}(A_1(I_{H,a}+I_{H,s})+A_2(S_V+I_V)+A_3u_1^2 + A_4u_2^2+A_5u_3^2)dt \tag{19}$$

With $A_i \geq 0$ for $i = 1,2,3,\ldots 6$,which represent the weight constant. Then the optimal control u_1^*,u_2^*, and u_3^* is determined such that

$$J(u_1^*,u_2^*,u_3^*) = \min_{u_1,u_2,u_3\in\Omega} q_k(s)J(u_1,u_2,u_3) \tag{20}$$

Where, $\Omega = \{u_1,u_2,u_3 : [0,t_f] \to [0,1], u_1,u_2,u_3$ can be measured $\}$. The Pontryagin's Maximum Principles [44], [45] are used for the objective functions, as follows:

$L(I_{H,a},I_{H,s},S_V,I_V,u_1,u_2,u_3) = A_1(I_{H,a}+I_{H,s}) + A_2(S_V+I_V)+A_3u_1^2+A_4u_2^2+A_5u_3^2$

and the Hamiltonian for this problem can be defined as:

$$H = L(I_{H,a},I_{H,s},S_V,I_V,u_1,u_2,u_3) + \lambda_1\frac{dS_H}{dt} + \lambda_2\frac{dI_{H,a}}{dt} + \lambda_3\frac{dI_{H,s}}{dt} + \lambda_4\frac{dR_H}{dt} + \lambda_5\frac{dS_V}{dt} + \lambda_6\frac{dI_V}{dt} \tag{21}$$

- State condition

$$\dot{S_H} = \Lambda_h - (1-u_3)\frac{\beta_{h1} c I_{H,a}}{1+I_{H,a}} S_H - \mu_h S_H - (1-u_1)\frac{b\beta_{h2} I_V}{N_H} S_H \quad (22)$$

$$\dot{I_{H,a}} = p(1-u_3)\frac{\beta_{h1} c I_{H,a}}{1+I_{H,a}} S_H + q(1-u_1)\frac{b\beta_{h2} I_V}{N_H} S_H - (\mu_h + \mu_1 + \gamma_h) I_{H,a} \quad (23)$$

$$\dot{I_{H,s}} = (1-p)(1-u_3)\frac{\beta_{h1} c I_{H,a}}{1+I_{H,a}} S_H + (1-q)(1-u_1)\frac{b\beta_{h2} I_V}{N_H} S_H - (\mu_h + \mu_1 + \gamma_h) I_{H,s} \quad (24)$$

$$\dot{R_H} = \gamma_h (I_{H,a} + I_{H,s}) - \mu_h R_H \quad (25)$$

$$\dot{S_V} = \Lambda_V - \frac{b\beta_V (I_{H,a} + I_{H,s})}{N_H} S_V - (\mu_v + u_2) S_V \quad (26)$$

$$\dot{I_V} = \frac{b\beta_V (I_{H,a} + I_{H,s})}{N_H} S_V - (\mu_v + u_2) I_V \quad (27)$$

- Co-state condition

$$\begin{aligned}
\dot{\lambda_1}(t) =& -\frac{\beta_{h1} c I_{H,a}(-1+u_3)(\lambda_1 - p\lambda_2 + (-1+p)\lambda_3)}{1+I_{H,a}} \\
& - \frac{b I_V (-1+u_1)\beta_{h2}(\lambda_1 - q\lambda_2 + (-1+q)\lambda_3)}{N_H} \\
& + \lambda_1 \mu_h \\
\dot{\lambda_2}(t) =& -\frac{\beta_{h1} c S_H (-1+u_3)(\lambda_1 - p\lambda_2 + (-1+p)\lambda_3)}{(1+I_{H,a})^2} \\
& \gamma_h(\lambda_2 - \lambda_4 + \frac{b S_V \beta_V (\lambda_5 - \lambda_6)}{N_H} + \lambda_2(\mu_1 + \mu_H) \\
& - A_1 \\
\dot{\lambda_3}(t) =& - A_1 + \gamma_h(\lambda_3 - \lambda_4) + \frac{b S_V \beta_V (\lambda_5 - \lambda_6)}{N_H} \\
& + \lambda_3(\mu_1 + \mu_h) \\
\dot{\lambda_4}(t) =& \lambda_4 \mu_h \\
\dot{\lambda_5}(t) =& - A_2 - \frac{b(I_{H,a} + I_{H,s}\beta_v(\lambda_5 - \lambda_6)}{N_H} + \lambda(u_2 + \mu_v) \\
\dot{\lambda_6}(t) =& -\frac{b I_V (-1+u_1)\beta_{h2}(\lambda_1 - q\lambda_2 + (-1+q)\lambda_3)}{N_H} \\
& + \lambda_6(u_2 + \mu_v) - A_2
\end{aligned}$$

- Optimality conditions

$$\begin{aligned}
\frac{\partial H}{\partial u_1} =& -\frac{b I_V (-1+u_1)\beta_{h2}(\lambda_1 - q\lambda_2 + (-1+q)\lambda_3)}{N_H} \\
& + 2u_1 A_3 \\
\frac{\partial H}{\partial u_2} =& 2u_2 A_4 - \lambda_5 S_V - \lambda_6 I_V \\
\frac{\partial H}{\partial u_3} =& 2A_5 u_3 + \frac{\beta_1 c I_{H,a} S_H (\lambda_1 - p\lambda_2 + (-1+p)\lambda_3)}{1+I_{H,a}}
\end{aligned}$$

Thus, the stationary conditions u_1, u_2, and u_3 are obtained, as follows:

$$\begin{aligned}
u_1 =& -\frac{b I_V (-1+u_1)\beta_{h2}(\lambda_1 - q\lambda_2 + (-1+q)\lambda_3)}{2A_3 N_H} \\
u_2 =& \frac{\lambda_5 S_V + \lambda_6 I_V}{2A_4} \\
u_3 =& -\frac{\beta_{h1} c I_{H,a} S_H (\lambda_1 - p\lambda_2 + (-1+p)\lambda_3)}{2A_5(1+I_{H,a})}
\end{aligned}$$

Because $\leq u_1, u_2, u_3 \leq 1$, then

$$\begin{aligned}
u_1^* =& min\Bigg\{1, max\Big\{0, -\frac{b I_V (-1+u_1)\beta_{h2}}{2A_3 N_H} \times \\
& \frac{(\lambda_1 - q\lambda_2 + (-1+q)\lambda_3)}{2A_3 N_H}\Big\}\Bigg\} \\
u_2^* =& min\Bigg\{\Big\{\frac{\lambda_5 S_V + \lambda_6 I_V}{2A_4}\Big\}\Bigg\} \\
u_3^* =& min\Bigg\{\Big\{-\frac{\beta_{h1} c I_{H,a} S_H (\lambda_1 - p\lambda_2 + (-1+p)\lambda_3)}{2A_5(1+I_{H,a})}\Big\}\Bigg\}
\end{aligned}$$

VI. Numerical Simulation

Numerical simulation is conducted to support the analytical results in the previous section. The population dynamics are compared when parameter values change, referring to those values used in Goswami et al. [10], Moreno et al. [43], and some others are assumed.

TABLE II
Parameter values and initial values

Parameter/ Initial Value	Value	
	$\mathcal{R}_0 < 1$	$\mathcal{R}_0 > 1$
b	0.2	0.5
c	1	1
p	6.25×10^{-6}	1.23×10^{-6}
q	0.00001	0.9
Λ_h	20	15
Λ_v	20	15
N_H	2400	2400
β_v	0.08	0.4
μ_h	0.00007	1/55(365)
μ_1	0.04227	0.0097
γ_h	0.0005	0.00017
μ_v	0.00007	1/14
β_{h1}	0.002	0.05
β_{h2}	0.054414	0.4
S_H	1800	1700
$I_{H,a}$	350	440
$I_{H,s}$	250	260
R_H	0	0
S_V	1900	1600
I_V	200	490

A. Numerical simulation models without control

Using parameter values and initial values as given in Table 2, we get:

Figure 4 showed that the infection decreased in the human and mosquito populations at the specified time interval due to natural mortality factors. Meanwhile, Figure 5 illustrated that the number of infected humans increase due to transmission and the most dominant transmission occurred between humans and mosquitoes. This is observed from the rate magnitude of β_{h2} and β_V greater than β_{h1}, indicating that the probability of human transmission from mosquitoes and vice

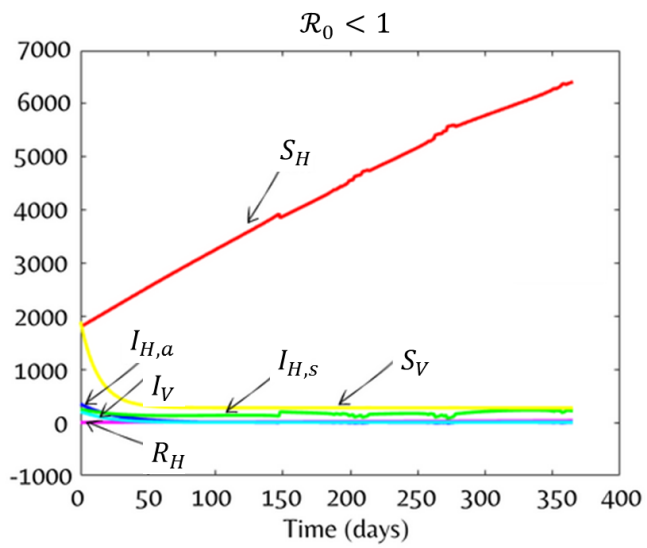

Fig. 4. Population Dynamics at the Non- Endemic Equilibrium Point.

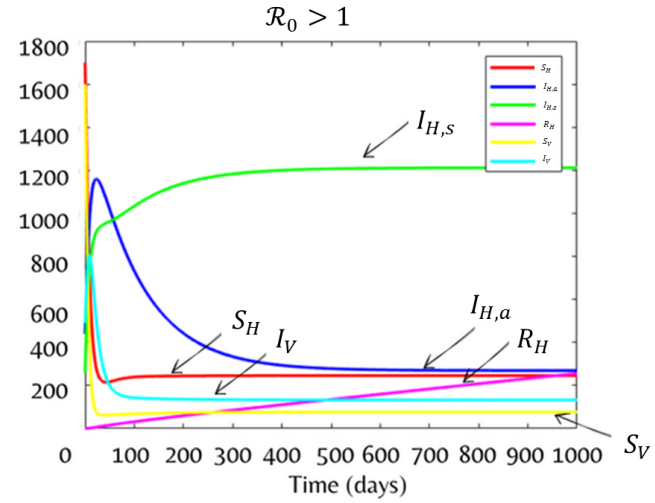

Fig. 5. Population Dynamics at the Endemic Equilibrium Point.

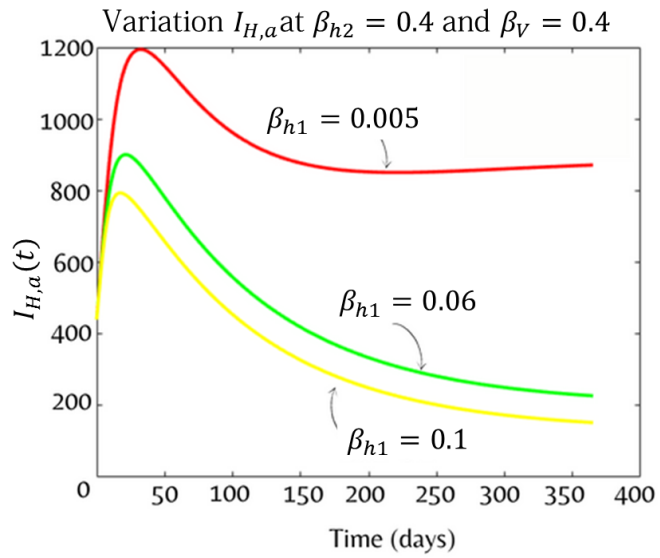

Fig. 6. Human Populations Infected with asymptomatic when β_{h1} Changed.

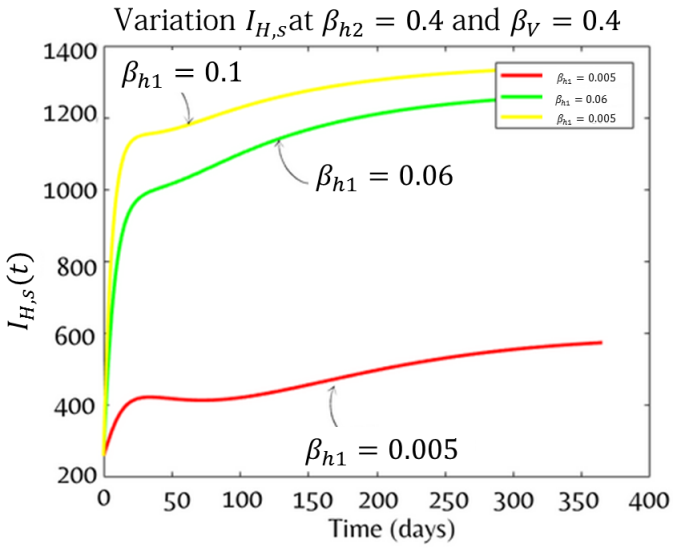

Fig. 7. Human Population Infected with Symptoms When β_{h1} Changed.

versa is significant. Furthermore, this causes healthy human and vector populations to decrease.

Figures 6, 7, and 8 demonstrated the differences in the population growth of humans and infected vectors. An increase in the transmission speed, decreases the population of humans infected without symptoms, hence the infected with symptoms increase. Meanwhile, the infective vectors experience a less significant change in growth. Figure 8 showed an initial increase. After 50 days, the infected vector decreased in the three levels of human-to-human transmission (β_{h1}). This shows that human-to-human transmission does not significantly affect the number of infected vectors.

Figures 9, 10, and 11 show the differences in the population growth of humans and infective vectors. The changes in infected asymptomatic and symptomatic humans were reversed from the previous cases, Figure 6 and Figure 7, due to the proportion of q and the proportion of infected humans without symptoms as a result of vector transmission. Similarly, the vector population affects the rate of transmission (β_{h2}).

Figures 12, 13, and 14 showed the differences in the growth of human populations and infected vectors. The dynamics of the infected human population are influenced by the infected vector, though they are not directly related to the rate of human-to-vector transmission. Therefore, an increase in infected vectors leads to a rise in infected humans. The infective vector population affects the transmission rate (β_{h2}), similar to the previous situation. The dynamics of the infected vector's population were unaffected by the number of infected individuals with or without symptoms.

B. Numerical Simulation Models Using Controls

Numerical simulations applied to the control model compare population dynamics before and after being given control and the model used in this simulation refers to a system (10). The parameter values in Table 2 are appropriate when $\mathcal{R}_0 > 1$, as control is required when a disease or virus in an area is epidemic. This treatment reduces infection and the

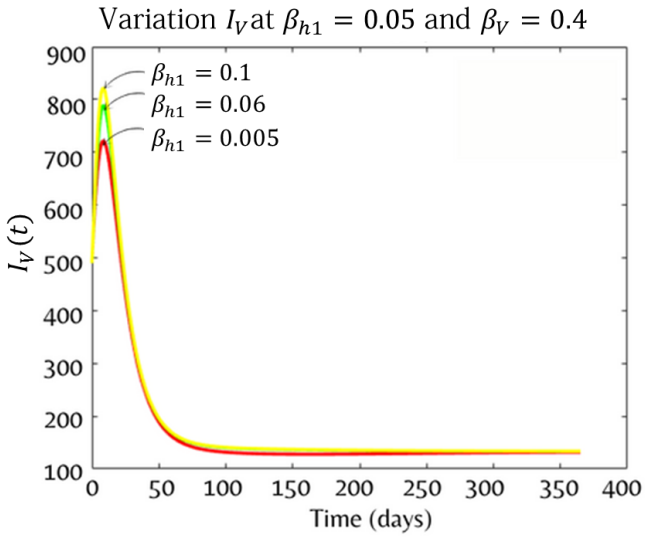

Fig. 8. Mosquito Population Infected when β_{h1} Changed.

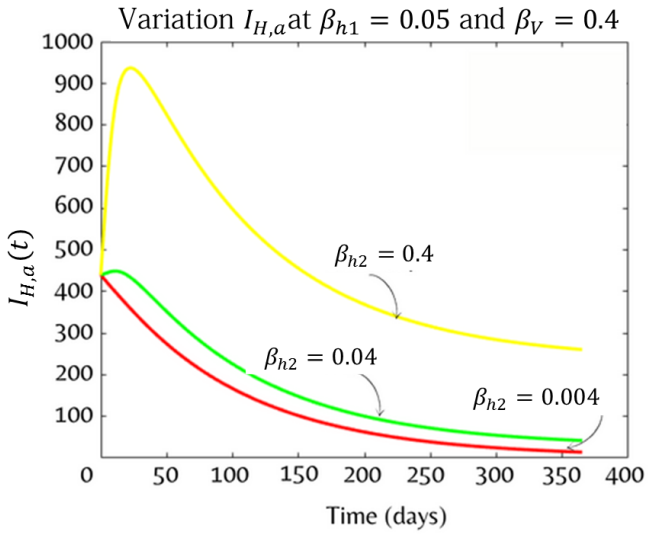

Fig. 9. Mosquito Population Infected when β_{h2} Changed.

spread of disease in a population. The weighting constant is then given $A_i, i = 1, 2, ..., 5$. $A_1 = 75, A_2 = 75, A_3 = 60, A_4 = 50, A_5 = 80$

Figures 15, 16, and 17, showed that mosquito eradication efforts, limiting direct contact with mosquitoes, and conducting counseling at regular health checks, reduces the spread of the disease to prevent transmission from asymptomatic humans to healthy ones.

VII. Conclusion

The model in endemic and non-endemic conditions is stable if the basic reproductive ratio is $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$, respectively. Furthermore, the sensitivity analysis showed that the spread of the disease is influenced by parameters related to vector-borne transmission than through humans. When Zika spreads in a population, efforts to control mosquito populations, lessen mosquito bites, educate the public about periodic checks, and eradicate mosquitoes hasten the decline of humans and infected vector's population. The three control

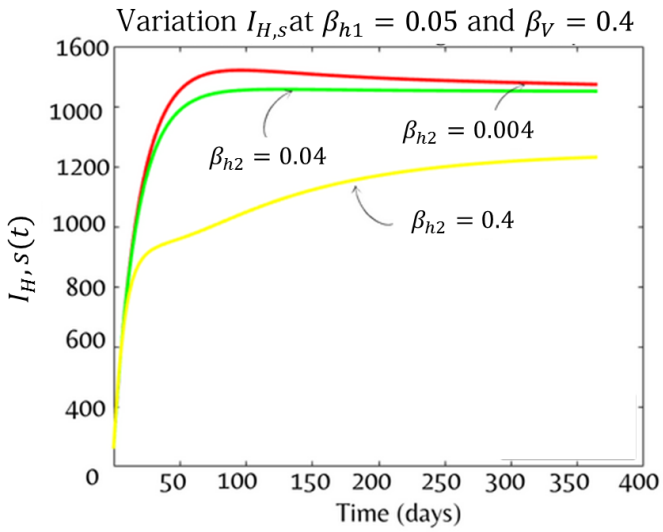

Fig. 10. Human Populations Infected with asymptomatic when β_{h2} Changed.

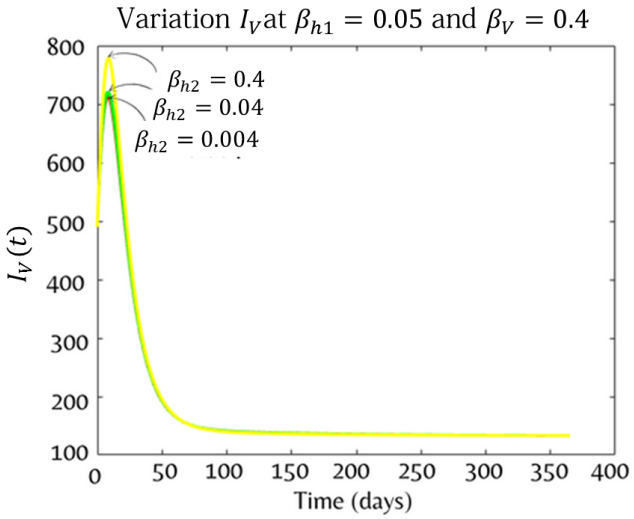

Fig. 11. Mosquito Population Infected When β_{h2} Changed.

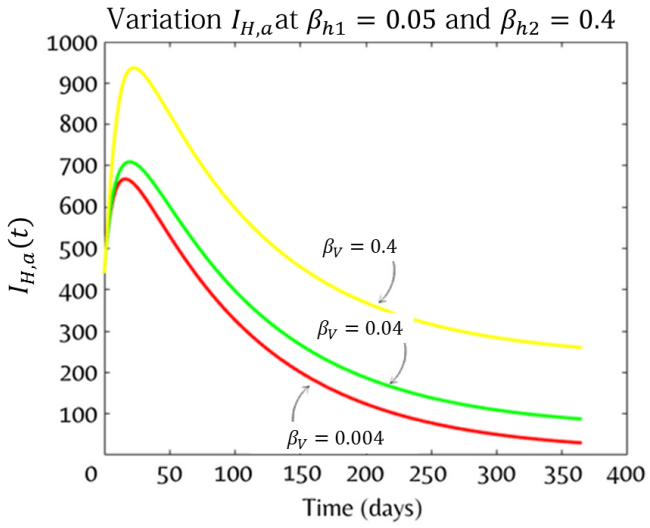

Fig. 12. Human Populations Infected with asymptomatic when β_v Changed.

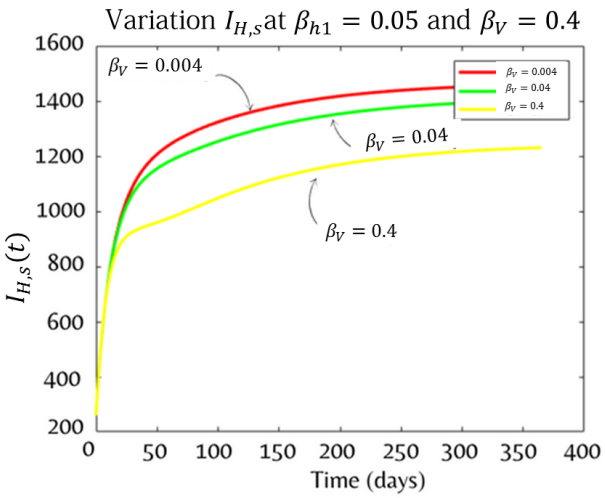

Fig. 13. Human Population Infected with Symptoms When β_v Changed.

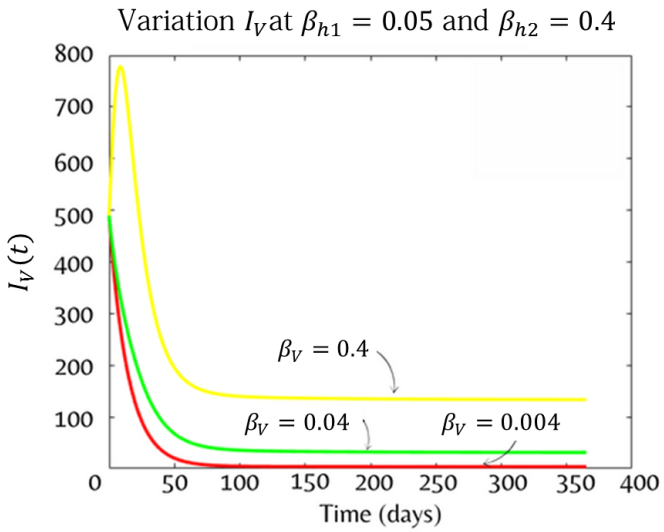

Fig. 14. Mosquito Population Infected When β_v Changed.

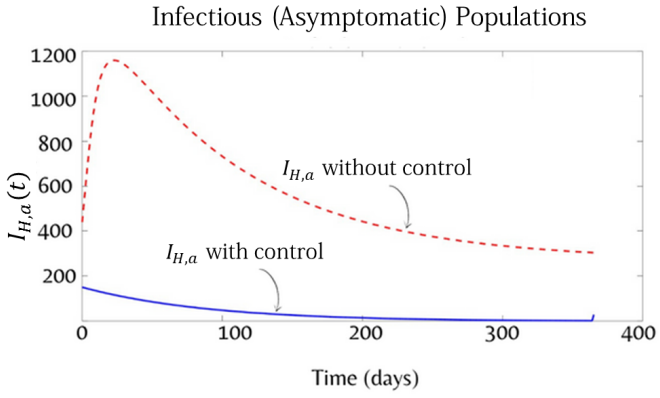

Fig. 15. Dynamics of an Asymptomatic Infected Human Population.

strategies combined in the model significantly reduce the number of infected individuals.

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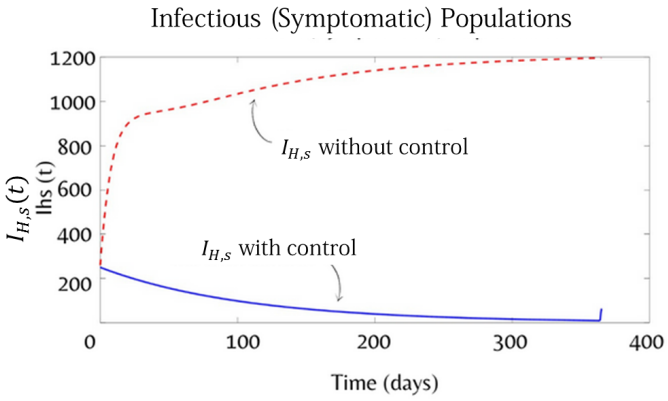

Fig. 16. Dynamics of Symptomatic Infected Human Populations.

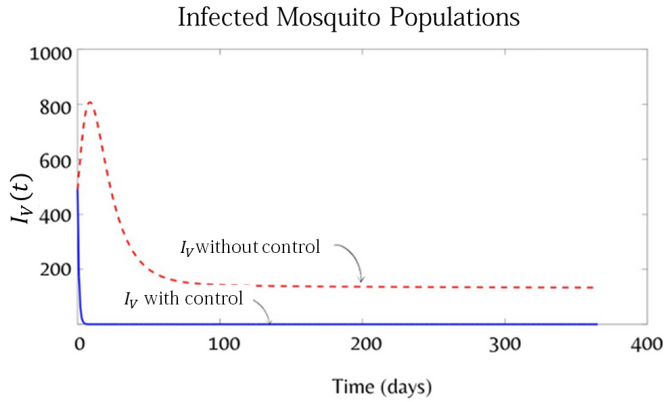

Fig. 17. Population Dynamics of Infected Mosquitoes.

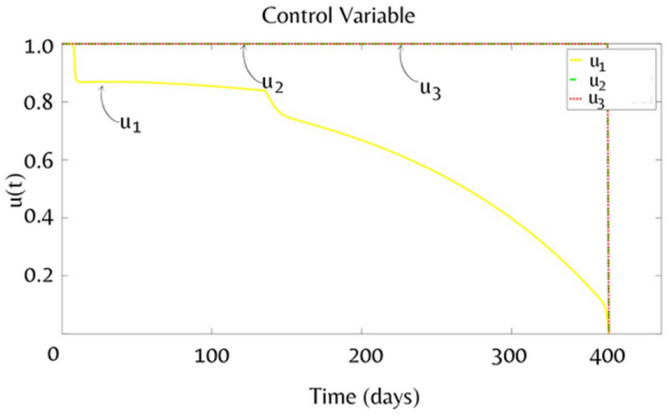

Fig. 18. The comparison of the control profiles u_i.

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