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SEIHR-SEI Mathematical Model of Zika Virus Transmission with Vector Control

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Abstract

Zika virus (ZIKV) is transmitted by *Aedes Aegypti* mosquito, which is recognized as a vector for viruses causing dengue fever and chikungunya. This study uses SEIHR-SEI mathematical model to analyze the dynamics of Zika virus transmission. In this model, human population (host) is classified into five compartments: Susceptible Humans (S_h), Exposed Humans (E_h), Infected Humans (I_h), Hospitalized Humans (H_h) and Recovered Humans (R_h). Meanwhile, the mosquito population (vector) is divide into three compartments: Susceptible Vectors (S_v), Exposed Vectors (E_v), and Infected Vectors (I_v). Stability analysis is conducted using Routh-Hurwitz criteria for assessing local stability and Lyapunov function for evaluating global stability. Moreover, Basic Reproduction Number (R_0), which represents the average number of new infections produced by one infected individual in a susceptible population, is derived by using the Next Generation Matrix (NGM) method. The result shows that the equilibrium point for disease-free conditions is globally asymptotic stable when $R_0 < 1$, meanwhile the equilibrium point for endemic conditions is stable when $R_0 > 1$. The simulation result using endemic data and sensitivity analysis of three parameters, including contact rate between susceptible humans and infected humans (c), hospitalization rate of infected individuals (τ), and mosquito control rate (ω), reveals that c and ω exert a more significant effect on changes in R_0 compared to τ. Therefore, minimizing contact with infected individuals or implementing vector control is more effective than isolating or hospitalizing infected patients.

Keywords: Basic reproduction number, vector control, Lyapunov, sensitivity analysis, Zika virus
MSC2020: 92D30, 93C10, 93D05.

Abstrak

Virus Zika (ZIKV) ditularkan oleh nyamuk Aedes Aegypti yang dikenal sebagai vektor penyebab demam berdarah dan chikungunya. Penelitian ini menggunakan model matematika SEIHR-SEI untuk menganalisis dinamika penularan virus Zika. Dalam model ini, populasi manusia (host) diklasifikasikan menjadi lima kompartemen: manusia Rentan (S_h), manusia terpapar (E_h), manusia terinfeksi (I_h), Manusia dalam perawatan (H_h), dan manusia sembuh (R_h). Sementara itu, populasi nyamuk (vektor) dibagi menjadi tiga kompartemen: nymauk rentan (S_v), nyamuk terpapar (E_v), dan nyamuk terinfeksi (I_v). Analisis kestabilan lokal maupun global dilakukan dengan menggunakan kriteria Routh-Hurwitz

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dan fungsi Lyapunov. Selain itu, Angka Reproduksi Dasar (R_0), yang merupakan angka rata-rata infeksi baru yang dihasilkan oleh satu individu yang terinfeksi dalam populasi yang rentan, diturunkan dengan menggunakan metode Next Generation Matrix (NGM). Hasilnya menunjukkan bahwa titik ekuilibrium untuk kondisi bebas penyakit adalah stabil asimtotik global ketika $R_0 < 1$, adapun titik ekuilibrium untuk kondisi endemik stabil ketika $R_0 > 1$. Hasil simulasi menggunakan data endemik dan analisis sensitivitas tiga parameter yang meliputi tingkat kontak antara manusia yang rentan dan manusia yang terinfeksi (c), tingkat rawat inap individu yang terinfeksi (τ), dan tingkat pengendalian nyamuk (ω), mengungkapkan bahwa c dan ω memberikan efek yang lebih signifikan pada perubahan (R_0) dibandingkan dengan τ. Dari hasil simulasi ini dapat disimpulkan bahwa meminimalkan kontak dengan individu yang terinfeksi atau menerapkan pengendalian vektor lebih efektif daripada mengisolasi atau merawat pasien yang terinfeksi.

Kata kunci: Bilangan reproduksi dasar, kontrol vektor, Lyapunov, analisis sensitivitas, virus Zika
MSC2020: 92D30, 93C10, 93D05.

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Introduction

Zika virus (ZIKV) is capable of infecting humans and causing Zika fever. This viral infection is prevalent, particularly in tropical and subtropical regions, such as those found in Asia and Africa. The primary mode of transmission for Zika virus is through the Aedes Aegypti mosquito, a species known for transmitting various viruses, including chikungunya, dengue fever, yellow fever, West Nile, and Japanese encephalitis. Aedes Aegypti mosquito acquires the virus by feeding on the blood of infected humans. Once it is infected, the mosquito can transmit the Zika virus to susceptible humans through subsequent bites [1]. Although Aedes Aegypti is the principal vector for Zika virus transmission, it is important to note that other vectors can also contribute to spread the virus. These include Aedes Albopictus, Aedes Africanus, Aedes Luteocephalus, Aedes Vittatus, Aedes Furcifer, Aedes Hensilii, and Aedes Apicoargenteus [2][3].

Zika virus (ZIKV) was initially identified in 1947 in the Zika forests of Uganda near Lake Victoria, where Rhesus Monkeys were found to be infected during an ecological investigation of yellow fever [4]. These Rhesus monkeys, serving as sentinel animals, were intentionally placed in tree cages in the Zika forest as part of yellow fever research [5]. The first documented case of a human being infected with the Zika virus was reported in 1964. The individual, a 28-year-old worker, experienced initial symptoms such as a mild headache. Subsequently, a rash developed on the face, neck, body, arms, and legs on the following day. Other symptoms included temporary fever, malaise, and back pain. Interestingly, neither of the two patients had a fever at night, and the rash had faded. By the third day, the patient felt healthy, although a rash persisted, ultimately disappearing two days later [6]. In April 2007, a Zika virus outbreak was initially reported on the Pacific island of Yap in the Federated States of Micronesia, resulting in 108 cases exhibiting Zika virus symptoms. Subsequently, the Zika virus outbreak extended to Indonesia, Micronesia (a country situated in an archipelago near the Pacific Ocean), the Philippines, Polynesia (with the largest populations in Tahiti and Moorea), and Easter Island in the South Pacific between October 2012 and April 2014 [5]. Several previous case investigations have also indicated the presence of Zika virus in various areas in Indonesia. In Klaten, for example, serological evidence of Zika was identified in seven individuals in 1977-1978. Furthermore, in 1979, arbovirus serological research in Lombok revealed Zika antibodies in 31% of the 71 samples tested [7][8].

The primary symptoms of Zika fever closely resemble to dengue fever, encompassing fever, a maculopapular rash that typically extends from the face to the body, joint and muscle discomfort, vomiting, and bilateral non-purulent conjunctivitis [9]. The incubation period for the Zika virus ranges from 2 to 7 days [10]. The Zika virus can be transmitted whether an individual is asymptomatic,

symptomatic, or even after symptoms have disappeared. Notably, a significant proportion of infected individuals either display mild symptoms or remain asymptomatic, leading to an unawareness of their Zika virus infection status [11]. Apart from the bite of Aedes Aegypti, Zika virus can also be transmitted through direct contact between susceptible and infected individuals through sexual contact [12]. This type of transmission distinguishes the Zika virus from the dengue and chikungunya viruses transmission[13]. Despite being a global threat to public health, there is presently no vaccine or cure available for Zika virus infection [12]. Addressing this challenge requires implementing measures to control and potentially suppress the spread of the Zika virus. These initiatives include enhancing public awareness through health programs, promoting the use of safety tools for couples planning to conceive during pregnancy (or abstaining from sexual relations), and implementing controls such as isolating Zika virus-infected patients in hospitals. Furthermore, vector control, a crucial aspect of Zika virus management, entails strategies to restrict or diminish the population of vectors carrying the virus. Vector control options include installing mosquito nets or fans, using aromatherapy or anti-mosquito lotions, spraying insecticides, and eliminating stagnant water that serves as a breeding ground for mosquito larvae [13].

Mathematical approach of Zika virus transmission has been undertaken through the exploration of various models and parameters. Chunxiao Dinga, Nana Tao, and Yuanguo Zhua, for example, investigated mathematical models of Zika virus transmission emphasizing optimal control parameters as an effective intervention strategy [14]. F. B. Agusto, S. Bewick, and W. F. Fagan conducted a mathematical model analysis specifically focused on the role of sexual transmission in the Zika virus spread, considering it as the only arbovirus capable of transmission through sexual contact [1]. Raúl Isea and Karl E. Lonngren analyzed mathematical models encompassing the dynamics of dengue, chikungunya, and Zika virus transmission [15]. In addition, S. K. Biswas, U. Ghosh, and S. Sarkar investigated a mathematical model addressing the spread of the Zika virus, which included the concept of vector control as a strategy for managing its transmission. They explained how the Zika virus spreads through mosquito bites and sexual interactions with infected individuals [13].

In contrast to previous studies, this paper employs a SEIHR-SEI model for Zika transmission, which encompasses the hospitality compartment within the human population. Moreover, besides mosquito transmission, the model considers horizontal transmission, where the virus spreads through human-to-human contact, including sexual contact, blood transfusion, or organ transplant. Additionally, the model integrates vector control as a strategy to manage and mitigate the spread of the Zika virus.

The remaining part of this paper discusses various aspects, including model construction, positivity and boundedness of solutions, determining equilibrium states and stability, deriving basic reproduction numbers, sensitivity analysis, numerical simulations, and interpretation.

Model Formulation

In this section, we develop a mathematical model to illustrate the population dynamics of Zika virus transmission, considering predefined assumptions. The model encompasses two distinct populations: humans and mosquitoes. The human population is divided into five compartments, namely susceptible humans (S_h), exposed humans (E_h), infected humans (I_h), hospitalized humans (H_h), and recovered humans (R_h). Meanwhile, the mosquito population is classified into three compartments: susceptible mosquitoes (S_v), exposed mosquitoes (E_v), and infected mosquitoes (I_v).

The assumptions employed in this model are outlined as follows:

1. The closure of both human and mosquito populations, implying that changes in population numbers are solely influenced by natural birth and death rates (without migration). The recruitment rates (natural births) of humans and mosquitoes are denoted by Λ_h and Λ_v, respectively, while the rates of natural deaths are represented by μ_h and μ_v for humans and mosquitoes, respectively.

2. The transmission of zika virus to the human population can transpire through the bite of

zika virus-infected mosquitoes as well as through sexual contact, blood transfusions, or organ transplants involving infected humans. b represents the rate of mosquito bites, with the virus transmission probability denoted by α_1, while c denotes the contact rate between susceptible humans and infected humans, with the virus transmission probability of α_2.

3. Susceptible mosquitoes become infected when they bite infected humans. α_3 signifies the probability of virus transmission per bite of a susceptible mosquito from an infected human.
4. Exposed humans and Exposed mosquitoes can transition to infected compartments at rates η and σ_v respectively.
5. Infected humans have the option of receiving treatment, with a hospitalization rate denoted by τ.
6. The rates at which hospitalized infected humans and those unable to recover are represented by θ_I and θ_H, respectively.
7. Infected humans, whether hospitalized or not, can recover at the corresponding rates of θ_I and θ_H.
8. Hospitalized infected humans can transmit the virus to mosquitoes, but at lower contact rates compared to untreated infected humans. In this model, η represents the relative probability of virus transmission from a hospitalized infected human.
9. Individuals who have recovered from the Zika virus either possess lifelong immunity.
10. Vector control measures are implemented to manage mosquito breeding and mitigate the spread of the Zika virus, with the mosquito control rate denoted as ω.

Compartment diagram illustrating the transmission of Zika virus in both humans and mosquitoes, incorporating the impact of hospitalization within human population and the implementation of vector control in mosquito population, is presented in Figure 1. Subsequently, referring to Figure (1), we derive the following system of non-linear differential equations:

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - \left(\frac{b\alpha_1 I_v}{N_h} + \frac{c\alpha_2 I_h}{N_h}\right) S_h - \mu_h S_h, \\
\frac{dE_h}{dt} &= \left(\frac{b\alpha_1 I_v}{N_h} + \frac{c\alpha_2 I_h}{N_h}\right) S_h - (\xi + \mu_h) E_h, \\
\frac{dI_h}{dt} &= \xi E_h - (\theta_I + \tau + \mu_h) I_h, \\
\frac{dH_h}{dt} &= \tau I_h - (\theta_H + \mu_h) H_h, \\
\frac{dR_h}{dt} &= \theta_I I_h + \theta_H H_h - \mu_h R_h, \\
\frac{dS_v}{dt} &= \Lambda_v - \left(\frac{b\alpha_3 I_h}{N_h} + \frac{b\alpha_3 \eta H_h}{N_h}\right) S_v - (\mu_v + \omega) S_v, \\
\frac{dE_v}{dt} &= \left(\frac{b\alpha_3 I_h}{N_h} + \frac{b\alpha_3 \eta H_h}{N_h}\right) S_v - (\sigma_v + \mu_v + \omega) E_v, \\
\frac{dI_v}{dt} &= \sigma_v E_v - (\mu_v + \omega) I_v,
\end{aligned} \tag{1}$$

with the initial conditions are $S_h(0) > 0$,$E_h(0) \geq 0$, $I_h(0) > 0$ $H_h(0) \geq 0$, $R_h(0) \geq 0$, $S_v(0) > 0$, $E_v(0) \geq 0$, $I_h(0) > 0$. The total of human population is denoted as N_h, with $N_h = S_h(t) + E_h(t) + I_h(t) + H_h(t) + R_h(t)$, meanwhile the total population of mosquitoes is defined as N_v, with $N_v = S_v(t) + E_v(t) + I_v(t)$.

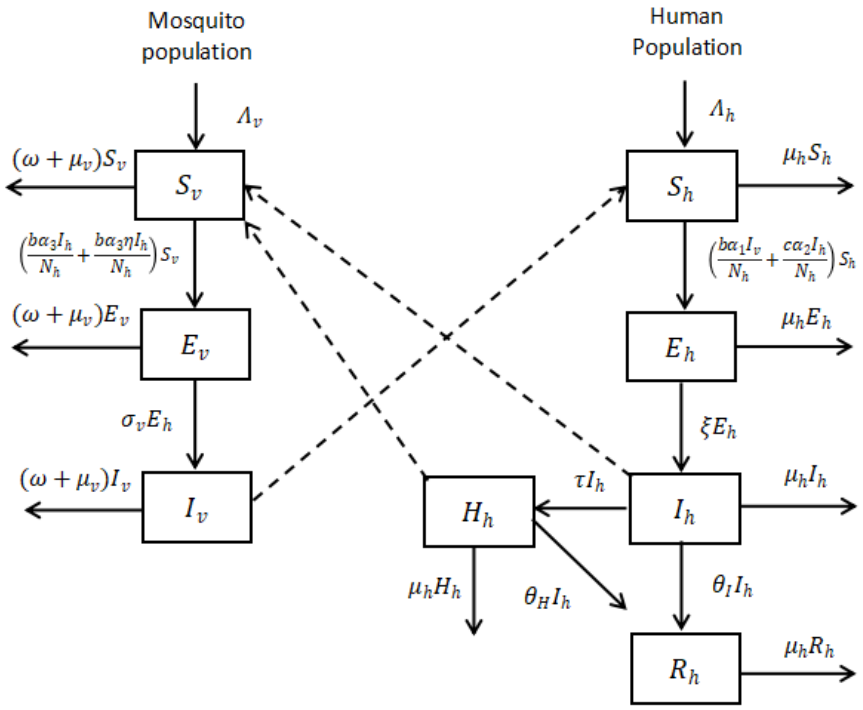

Figure 1. Compartment diagram of SEIHR-SEI model for Zika virus transmission.

The non-negativity and boundedness of solution

To analyze that the solutions for each compartment are non-negative, observe that:

$$\frac{dS_h}{dt} = \Lambda_h - \left(\frac{b\alpha_1 I_v}{N_h} + \frac{c\alpha_2 I_h}{N_h}\right) S_h - \mu_h S_h, \geq -\left[\left(\frac{b\alpha_1 I_v}{N_h} + \frac{c\alpha_2 I_h}{N_h}\right) + \mu_h\right] S_h,$$
$$\frac{dS_h}{S_h} \geq -\left[\left(\frac{b\alpha_1 I_v}{N_h} + \frac{c\alpha_2 I_h}{N_h}\right) + \mu_h\right] dt, \quad (2)$$

By integrating both sides of equation, we obtain:

$$S_h(t) \geq S_h(0) \cdot \exp \int -\left[\left(\frac{b\alpha_1 I_v}{N_h} + \frac{c\alpha_2 I_h}{N_h}\right) + \mu_h\right] dt \geq 0 \quad (3)$$

Similar techniques can be applied to the remaining compartments to demonstrate that the solutions of each equation (1) are non-negative. Next, to determine that the solution is bounded, add up all the differential equations from each human and mosquito compartment as follow:

$$\frac{dN_h}{dt} = \frac{dS_h}{dt} + \frac{dE_h}{dt} + \frac{dI_h}{dt} + \frac{dH_h}{dt} + \frac{dR_h}{dt} = \Lambda_h - \mu N_h,$$
$$\frac{dN_h}{dt} + \mu_h N_h = \Lambda_h.$$

by using integrating factors [16], we get

$$N_h(t) = \frac{\Lambda_h}{\mu_h}(1 - e^{-\mu_h t}) + N_h(0)e^{-\mu_h t}.$$

When $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$, then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$, and for $t \to \infty$, we get $0 < N_h(t) \leq \frac{\Lambda_h}{\mu_h}$.

Meanwhile, by summing up the differential equations for the mosquito population, we obtain:

$$\frac{dN_v}{dt} = \frac{dS_v}{dt} + \frac{dE_v}{dt} + \frac{dI_v}{dt} = \Lambda_v - (\omega + \mu_v)N_h, \quad \longleftrightarrow \quad \frac{dN_v}{dt} + (\omega + \mu_v)N_v = \Lambda_v.$$

by integrating both sides of the equation, we have

$$N_v(t) = \frac{\Lambda_v}{(\omega + \mu_v)}(1 - e^{-(\omega+\mu_v)t}) + N_v(0)e^{-(\omega+\mu_v)t}.$$

When $N_v(0) \leq \frac{\Lambda_v}{(\omega+\mu_v)}$, then $N_v(t) \leq \frac{\Lambda_v}{(\omega+\mu_v)}$, and for $t \to \infty$, we get $0 < N_v(t) \leq \frac{\Lambda_v}{(\omega+\mu_v)}$. Consequently we have

$$\Omega = \left\{ (S_h, E_h, I_h, H_h, R_h, S_v, E_v, I_v) \in \mathbf{R}^8_+ \,|\, 0 < N_h(t) \leq \frac{\Lambda_h}{\mu_h}, \text{ and } 0 < N_v(t) \leq \frac{\Lambda_v}{(\omega + \mu_v)} \right\}, \quad (4)$$

which is a positively invariant and attracting set of the model (1).

Equilibrium States

The equilibrium state is reached when the population size remains constant, indicating that S_h, E_h, I_h, H_h, R_h, S_v, E_v, and I_v remain unchanged. In the SEIHR-SEI mathematical model, there are two equilibriums: Disease-free equilibrium and Endemic Equilibrium. The equilibrium states are determined by equating the right-hand side of the differential equations system (1) to zero,

$$\dot{X} = \bar{f}(X) = 0 \quad (5)$$

with $X = (S_h, E_h, I_h, H_h, R_h, S_v, E_v, I_v)$. The disease-free equilibrium is characterized by the absence of Zika virus spread, resulting in no new infections and no individuals infected with Zika virus within the population. By substituting $I_h = 0$ and $I_v = 0$ to equation (5), then the disease-free equilibrium state is expressed as follows:

$$E^0 = \left(S_h^0, E_h^0, I_h^0, H_h^0, R_h^0, S_v^0, E_v^0, I_v^0\right) = \left(\frac{\Lambda_h}{\mu_h}, 0, 0, 0, 0, \frac{\Lambda_v}{\mu_v + \omega}, 0, 0\right). \quad (6)$$

Next, the endemic equilibrium point is a state in which individuals are continuously infected with Zika virus, allowing the virus to persist and spread within the population, resulting in more than one new infection. By solving equation (5), with $I_h \neq 0$ and $I_v \neq 0$, the endemic equilibrium state is given by

$$E^* = \left(S_h^*, E_h^*, I_h^*, H_h^*, R_h^*, S_v^*, E_v^*, I_v^*\right), \quad (7)$$

where

$$S_h^* = \frac{\Lambda_h}{\lambda_h + \mu_h}, \qquad E_h^* = \frac{\lambda_h \Lambda_h}{K_1\left(\lambda_h + \mu_h\right)}, \qquad I_h^* = \frac{\lambda_h \xi \Lambda_h}{K_1 K_2\left(\lambda_h + \mu_h\right)}$$
$$H_h^* = \frac{\lambda_h \xi \tau \Lambda_h}{K_1 K_2 K_3\left(\lambda_h + \mu_h\right)}, \quad R_h^* = \frac{\left(\theta_I K_3 + \theta_H \tau\right)\lambda_h \xi \Lambda_h}{K_1 K_2 K_3\left(\lambda_h + \mu_h\right)\mu_h},$$
$$S_v^* = \frac{\Lambda_v}{\lambda_v + K_4}, \qquad E_v^* = \frac{\lambda_v \Lambda_v}{K_5\left(\lambda_v + K_4\right)}, \qquad I_v^* = \frac{\sigma_v \lambda_v \Lambda_v}{K_4 K_5\left(\lambda_v + K_4\right)}.$$

and

$$K_1 = \eta + \mu_h, \quad K_2 = \theta_I + \tau + \mu_h, \quad K_3 = \theta_H + \mu_h, \quad K_4 = \mu_v + \omega \quad K_5 = \sigma_v + \mu_v + \omega,$$
$$\lambda_h = \frac{b\alpha_1 I_v^*}{N_h} + \frac{c\alpha_2 I_h^*}{N_h}, \quad \lambda_v = \frac{b\alpha_3 I_h^*}{N_h} + \frac{b\alpha_3 \eta H_h^*}{N_h}.$$

Basic Reproduction Number (R_0)

Basic reproduction number (R_0) represent the average number of newly infected susceptible individuals (secondary infections) generated by a single infected individual (primary infection) within a susceptible population [17]. In this study, the Next Generation Matrix (NGM) method will be employed to calculate this number. The population involved in determining the basic reproduction number is the infected compartment, encompassing E_h, I_h, H_h, E_v, and I_v.

Let J_0 represents the Jacobi matrix of the infected compartment variable, which is substituted under disease-free conditions. Subsequently, decompose the matrix J_0 into $F - V$, where F is the interaction matrix (non-linear), and V is the transition matrix (linear).

$$F = \begin{bmatrix} 0 & \frac{c\alpha_2\Lambda_h}{N_h\mu_h} & 0 & 0 & \frac{b\alpha_1\Lambda_h}{N_h\mu_h} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{b\alpha_3\Lambda_v}{N_hK_4} & \frac{b\alpha_3\eta\Lambda_v}{N_hK_4} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} K_1 & 0 & 0 & 0 & 0 \\ -\xi & K_2 & 0 & 0 & 0 \\ 0 & -\tau & K_3 & 0 & 0 \\ 0 & 0 & 0 & K_5 & 0 \\ 0 & 0 & 0 & -\sigma_v & K_4 \end{bmatrix}.$$

The basic reproduction number (R_0) is the spectral radius or the largest positive eigenvalue of the Next Generation Matrix or a product of FV^{-1}, then we get

$$R_0 = \frac{1}{2}\frac{c\alpha_2\xi}{K_1K_2} + \frac{1}{2}\sqrt{\frac{c^2{\alpha_2}^2\xi^2}{{K_1}^2{K_2}^2} + \frac{4b^2\alpha_3\mu_h\Lambda_v\xi\left(K_3+\eta\tau\right)\sigma_v\alpha_1}{\Lambda_h{K_4}^2K_1K_2K_3K_5}}. \tag{8}$$

We will let $R_0 = R_1 + R_2$, where

$$R_1 = \frac{1}{2}\frac{c\alpha_2\xi}{K_1K_2}, \quad R_2 = \frac{1}{2}\sqrt{\frac{c^2{\alpha_2}^2\xi^2}{{K_1}^2{K_2}^2} + \frac{4b^2\alpha_3\mu_h\Lambda_v\xi\left(K_3+\eta\tau\right)\sigma_v\alpha_1}{\Lambda_h{K_4}^2K_1K_2K_3K_5}}.$$

Local Stability Analysis

Local stability analysis is used to evaluate the stability properties of equilibrium points in a mathematical system. An equilibrium point is considered locally stable if all eigenvalues of the Jacobian matrix have negative real parts. Conversely, if any eigenvalue has a positive real part, the equilibrium point is considered unstable [18]. The Jacobian matrix of the SEIHR-SEI model is shown in Equation (9).

$$J = \begin{bmatrix} -\lambda_h-\mu_h & 0 & -\frac{c\alpha_2S_h}{N_h} & 0 & 0 & 0 & 0 & -\frac{b\alpha_1S_h}{N_h} \\ \lambda_h & -K_1 & \frac{c\alpha_2S_h}{N_h} & 0 & 0 & 0 & 0 & \frac{b\alpha_1S_h}{N_h} \\ 0 & \xi & -K_2 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau & -K_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & \theta_I & \theta_H & -\mu_h & 0 & 0 & 0 \\ 0 & 0 & -\frac{b\alpha_3S_v}{N_h} & -\frac{b\alpha_3\eta S_v}{N_h} & 0 & -\lambda_v-K_4 & 0 & 0 \\ 0 & 0 & \frac{b\alpha_3S_v}{N_h} & \frac{b\alpha_3\eta S_v}{N_h} & 0 & \lambda_v & -K_5 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \sigma_v & -K_4 \end{bmatrix}, \tag{9}$$

Let J_0 is jacobian matriks of J evaluated in the disease free equilbrium, then the caracteristic equation of matrix $(J_0 - \lambda I)$ is

$$P(\lambda) = (\lambda+\mu_h)^2(\lambda+K_4)(a_5\lambda^5 + a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0) = 0, \tag{10}$$

where

$$
\begin{aligned}
a_5 =& 1,\\
a_4 =& K_1 + K_2 + K_3 + K_4 + K_5,\\
a_3 =& \left(K_4 + K_5 + K_3\right)\left(K_1 + K_2\right) + \left(K_3 + K_5\right)K_4 + K_3K_5 + K_1K_2\left(1 - 2R_1\right),
\end{aligned}
$$

$$
\begin{aligned}
a_2 =& \left(\left(K_3 + K_5\right)K_4 + K_3K_5\right)K_1 + \left(\left(K_3 + K_5\right)K_4 + K_3K_5\right)K_2 + K_4K_5K_3 +\\
& K_1K_2\left(K_4 + K_5 + K_3\right)\left(1 - 2R_1\right),\\
a_1 =& K_1K_2\left(K_3K_4 + K_5K_4 + K_3K_5\right)\left(1 - 2R_1\right) + \frac{K_1K_2K_3K_4K_5}{K_3 + \eta\tau},\\
& \left(\frac{K_3 + \eta\tau}{K_1} + \frac{K_3 + \eta\tau}{K_2} + {R_1}^2 - {R_2}^2\right),\\
a_0 =& K_5K_4K_3K_2K_1\left(1 - {R_0}^*\right),
\end{aligned}
$$

where $R_0^* = R_0^2 + 2R_1(1 - R_0)$ [19]. The first three eigenvalues are negative, namely $-\mu_h, -\mu_h, -K_4$. As for fifth-order polynomials, based on the Routh-Hurwitz criterion, the equation (10) will have all negative roots when $R_0 < 1$, $R_1 < 1/2, R_1 > R_2$, and $a_4a_3 > a_2a_5$, $a_2b_1 > a_4b_2$, with $b_1 = (a_4a_3 - a_2a_5)/a_4$ and $b_2 = (a_4a_1 - a_0a_5)/a_4$. Noted that, if $R_0^* < 1$ result in $R_0 < 1$.

Moreover, for local stability under the endemic conditions (END), let J_1 represents the jacobian matrix evaluated at the endemic equilibrium, the characteristic equation of matrix $(J_1 - \lambda I)$ is presented in Equation 11.

$$
P(\lambda) = (\lambda + \mu_h)(\lambda + K_4)(\lambda^6 + a_5\lambda^5 + a_4\lambda^4 + a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + b_0) = 0, \tag{11}
$$

with

$$
\begin{aligned}
z^* =& \lambda_h^* + \lambda_v^* + \mu_h,\\
a_5 =& (K_1 + K_2 + K_3 + K_4 + K_5 + z^*),\\
a_4 =& ((K_2 + K_3 + K_4 + K_5 + z^*)K_1 + (K_3 + K_4 + K_5 + z^*)K_2\\
& + (K_4 + K_5 + z^*)K_3 + (K_4 + z^*)K_5 + (\lambda_h^* + \mu_h)(\lambda_v^* + K_4) - \xi c\alpha_2 S_h^*)/N_h,\\
a_3 =& ((((K_1 + K_2 + K_4 + z^*)K_5 + (K_2 + K_4 + z^*)K_1 + (K_4 + z^*)K_2 + (\lambda_h^* + \mu_h)(\lambda_v^* + K_4))K_3\\
& + ((K_2 + K_4 + z^*)K_1 + (K_4 + z^*)K_2 + (\lambda_h^* + \mu_h)(\lambda_v^* + K_4))K_5 + ((K_4 + z^*)K_2\\
& + (\lambda_h^* + \mu_h)(\lambda_v^* + K_4))K_1 + K_2(\lambda_h^* + \mu_h)(\lambda_v^* + K_4))N_h - \xi c\alpha_2 S_h^*(K_3 + K_4 + K_5 + \lambda_v^* + \mu_h))/N_h,\\
a_2 =& ((((K_2 + K_4 + z^*)K_1 + (K_4 + z^*)K_2 + (\lambda_h + \mu_h)(\lambda_v + K_4))K_5 + ((K_4 + z^*)K_2\\
& + (\lambda_h + \mu_h)(\lambda_v + K_4))K_1 + K_2(\lambda_h + \mu_h)(\lambda_v + K_4))K_3 + (((K_4 + z^*)K_2\\
& + (\lambda_h + \mu_h)(\lambda_v + K_4))K_1 + K_2(\lambda_h + \mu_h)(\lambda_v + K_4))K_5 + K_1K_2(\lambda_h + \mu_h)(\lambda_v + K_4))\\
& - ((K_4 + K_5 + \lambda_v + \mu_h)K_3 + (K_4 + \lambda_v + \mu_h)K_5 + \mu_h(K_4 + \lambda_v))\xi c\alpha_2 S_h^*/N_h\\
& - \xi b^2\sigma_v\alpha_1\alpha_3 S_h^* S_v^*/N_h^2,\\
a_1 =& (((((K_4 + z^*)K_2 + (\lambda_h + \mu_h)(\lambda_v + K_4))K_1 + K_2(\lambda_h + \mu_h)(\lambda_v + K_4))K_5\\
& + K_1K_2(\lambda_h + \mu_h)(\lambda_v + K_4))K_3 + K_1K_2K_5(\lambda_h + \mu_h)(\lambda_v + K_4))\\
& - (((K_4 + \lambda_v + \mu_h)K_5 + \mu_h(K_4 + \lambda_v))K_3 + \mu_hK_5(K_4 + \lambda_v))\xi c\alpha_2 S_h^*/N_h\\
& - \xi b^2\sigma_v\alpha_1\alpha_3 S_h^* S_v^*(\eta\tau + \mu_h + K_3)/N_h^2,\\
a_0 =& K_1K_2K_3K_5(\lambda_h + \mu_h)(\lambda_v + K_4) - K_3K_5\xi c\alpha_2\mu_h S_h^*(\lambda_v + K_4)/N_h\\
& - \xi b^2\sigma_v\alpha_1\alpha_3\mu_h S_h^* S_v^*(\eta\tau + K_3)/N_h^2.
\end{aligned}
$$

By using Routh-Hurwitz criteria of stability, considering that

$$b_1 = \frac{a_4a_3 - a_2a_5}{a_4}, \quad b_2 = \frac{a_4a_1 - a_0a_5}{a_4}, \quad c_1 = \frac{b_1a_2 - b_2a_4}{b_1}, \quad c_2 = a_0, \quad d_1 = \frac{c_1b_2 - a_0b_1}{c_1},$$

then endemic equilibrium is locally asimptotically stabil when $b_i > 0$, $i = 0, 1, ..., 6$ and satisfy the contitions $a_5a_4 > a_6a_3$, $b_1a_3 > a_5b_2$, $c_1b_2 > b_1c_2$, and $d_1c_2 > c_1a_0$.

Global Stability Analysis

The global stability analysis at the disease-free equilibrium point (DFE) will be conducted by using Lyapunov function. Let X=(S_h, E_h, I_h, H_h, R_h, S_v, E_v, I_v), then define the Lyapunov fuction below

$$\begin{aligned} V(X) =& W_1\left(S_h - S_h^0 - S_h^0 \ln\left(\frac{S_h}{S_h^0}\right)\right) + W_2E_h + W_3I_h + W_4H_h + W_5\left(S_v - S_v^0 - S_v^0 \ln\left(\frac{S_v}{S_v^0}\right)\right) \\ &+ W_6E_v + W_7I_v, \end{aligned} \tag{12}$$

with coefficients W_i, for $i = 1, .., 7$ are all positif.

Base on the definition [20] [21] [22], we will examine whether the Lyapunov function (12) satisfy the following conditions: (i). $V(X) = 0$ for $X = E^0$; (ii). $V(X) > 0$ For all $X \in \Omega$ and (iii). $V'(X) < 0$ for all $X \in \Omega$.

(i) For $X = E^0$, then

$$\begin{aligned} V(E^0) =& W_1\left(S_h^0 - S_h^0 - S_h^0 \ln\left(\frac{S_h^0}{S_h^0}\right)\right) + W_2E_h^0 + W_3I_h^0 + W_4H_h^0 \\ &+ W_5\left(S_v^0 - S_v^0 - S_v^0 \ln\left(\frac{S_v^0}{S_v^0}\right)\right) + W_6E_v^0 + W_7I_v^0, \\ V(E^0) =& W_1(0) + W_2(0) + W_3(0) + W_4(0) + W_5(0) + W_6(0) + W_7(0) = 0. \end{aligned} \tag{13}$$

(ii) For all $X \in \Omega$, then

$$\begin{aligned} V(X) =& W_1\left(S_h - S_h^0 - S_h^0 \ln\left(\frac{S_h}{S_h^0}\right)\right) + W_2E_h + W_3I_h + W_4H_h \\ &+ W_5\left(S_v - S_v^0 - S_v^0 \ln\left(\frac{S_v}{S_v^0}\right)\right) + W_6E_v + W_7I_v, \\ V(X) =& W_1S_h^0\left(\frac{S_h}{S_h^0} - 1 - \ln\left(\frac{S_h}{S_h^0}\right)\right) + W_2E_h^0 + W_3I_h^0 + W_4H_h^0 \\ &+ W_5S_v^0\left(\frac{S_v}{S_v^0} - 1 - \ln\left(\frac{S_v}{S_v^0}\right)\right) + W_6E_v^0 + W_7I_v^0. \end{aligned} \tag{14}$$

$V(X) \geq 0$ only if $\left(\frac{S_h}{S_h^0} - 1 - \ln\left(\frac{S_h}{S_h^0}\right)\right) \geq 0$ and $\left(\frac{S_v}{S_v^0} - 1 - \ln\left(\frac{S_v}{S_v^o}\right)\right) \geq 0$. Let $\frac{S_h}{S_h^0} = z_1$ and $\frac{S_v}{S_v^0} = z_2$, then suppose a function $f(z_i) = z_i - 1 - \ln z_i$. This function $f(z_i)$ will attains a global minimum at $z_i = 1$ and $f(1) = 0$, thus $f(z_i) \geq 0$ for all z_i. Consequently, $V(X)$ will be positive for all $X \in \Omega$,

(iii) The first derivative of the Lyapunov function is given by

$$\begin{aligned}V'(X) =& W_1\left(S_h' + 0 - S_h^o\left(\frac{S_h'}{S_h}\right)\right) + W_2E_h' + W_3I_h' + W_4H_h' + W_5\left(S_v' + 0 - S_v^o\left(\frac{S_v'}{S_v}\right)\right)\\ &+ W_6E_v' + W_7I_v',\\ =& W_1S_h'\left(1 - \frac{S_h^o}{S_h}\right) + W_2E_h' + W_3I_h' + W_4H_h' + W_5S_v'\left(1 - \frac{S_v^o}{S_v}\right) + W_6E_v' + W_7I_v'.\end{aligned}$$

Substitute the system (1) into $V'(X)$, then we have

$$\begin{aligned}V'(x) =& W_1\left(\Lambda_h - (b\alpha_1I_v + c\alpha_2I_h)\frac{S_h}{N_h} - \mu_hS_h\right)\left(1 - \frac{S_h^o}{Sh}\right)\\ &+ W_2\left(-(b\alpha_1I_v + c\alpha_2I_h)\frac{S_h}{N_h} - K_1E_h\right) + W_3(\xi E_h - K_2I_h)\\ &+ W_4(\tau I_h - K_3H_h)\\ &+ W_5\left(\Lambda_v - b\alpha_3(I_h + \eta H_h)\frac{S_v}{N_h} - K_4S_v\right)\left(1 - \frac{Sv^o}{Sv}\right)\\ &+ W_6\left(b\alpha_3(I_h + \eta H_h)\frac{S_v}{N_h} - K_5E_v\right) + W_7(\sigma_vE_v - K_4I_v).\end{aligned}$$

By substituting $S_h^0 = \frac{\Lambda_h}{\mu_h}$, $S_v^0 = \frac{\Lambda_v}{K_4}$ and simplifying the last equation of $V'(t)$, we obtain

$$\begin{aligned}V'(x) =& -\mu_hW_1\frac{(S_h - S_h^o)^2}{S_h} - K_4W_5\frac{(S_v - S_v^o)^2}{S_v}\\ &+ (W_2 - W_1)(b\alpha_1I_v + c\alpha_2I_h)\frac{S_h}{N_h} + (W_6 - W_5)b\alpha_3(I_h + \eta H_h)\frac{S_v}{N_h}\\ &+ E_h(W_3\xi - W_2K_1) + I_h\left(W_1c\alpha_2 + W_4\tau + W_5\frac{b\alpha_3\Lambda_v\mu_h}{(K_4\Lambda_h)} - W_3K_2\right)\\ &+ H_h\left(W_5\frac{b\alpha_3\eta\Lambda_v\mu_h}{(K_4\Lambda_h)} - W_4K_3\right) + E_v(W_7\sigma_v - W_6K_5)\\ &+ I_v(W_1b\alpha_1 - W_7K_4).\end{aligned}$$

Next, select the coefficient of W_1, W_2, W_3, W_4, W_5, W_6, W_7 as follows:
$w_1 = w_2 = \frac{\xi}{K_1K_2}$, $w_3 = \frac{1}{K_2}$, $w_4 = \frac{b^2\alpha_3\mu_h\Lambda_v\xi\eta\sigma_v\alpha_1}{\Lambda_hK_4^2K_1K_2K_3K_5}$, $w_5 = w_6 = \frac{b\xi\sigma_v\alpha_1}{K_1K_2K_4K_5}$, and $w_7 = \frac{b\xi\alpha_1}{K_1K_2K_4}$. Subsequently, the derivative of the Lyapunov function becomes

$$V'(x) = -\frac{\mu_h\xi(S_h - S_h^o)^2}{(K_2K_1S_h)} - \frac{b\xi\sigma_v\alpha_1(S_v - S_v^o)^2}{(K_1K_2K_5S_v)} - I_h(1 - R_0^*). \tag{15}$$

Then, $V'(x) < 0$ holds when $R_0 < 1$.

As a result, it can be concluded that the disease-free equilibrium point (DFE) is globally asymptotically stable when $R_0 < 1$.

Numerical Simulation

Through graphical representations, numerical simulations are performed to depict the dynamics of each compartment of the SEIHR-SEI model for Zika virus transmission in the presence of vector control, using Maple software. The data used in these numerical simulations are arbitrary but comply with the conditions required for the existence and stability of the disease-free equilibrium (DFE) and

endemic equilibrium (END). Data for numerical simulations under disease-free conditions are shown in Table 1.

Table 1. Data of parameter values for desease-free simulation.

Λ_h	Λ_v	μ_h	μ_v	θ_H	θ_I	b	c
7	100	0.27	0.55	0.329	0.269	0.575	0.373
ω	ξ	τ	σ_v	α_1	α_2	α_3	η
0.594	0.581	0.987	0.693	0.772	0.375	0.765	0.153

By substituting the data from Table 1 into the equation for R_0, we get $R_0 \approx 0.38$. This value indicates that, on average, one hundred infected individuals can infect 38 susceptible people, suggesting that the disease would diminish within the population. As illustrated in Figure 2, starting with an initial population of 30 individuals in each compartment, over time, the number of susceptible humans stabilizes at 25, while the other compartments reach a steady state on zero.

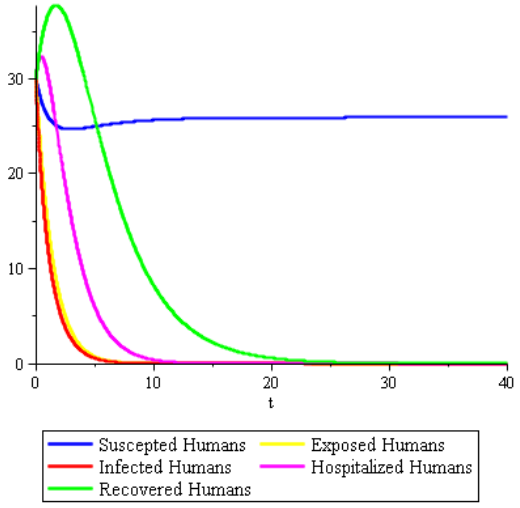

Figure 2. The dynamic of human population under disease-free condition.

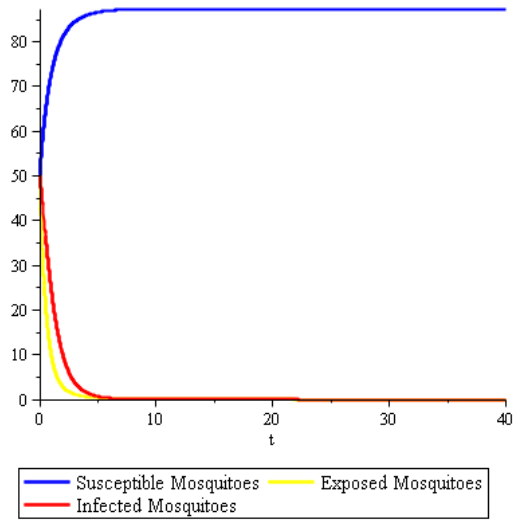

Figure 3. The dynamic of mosquitoes population under disease-free condition.

Similarly, the dynamics of the mosquito population reached stability with 87 susceptible mosquitoes. At the same time, the populations of exposed and infected mosquitoes stabilized to 0, as shown in Figure 3.

Furthermore, the parameter values used in endemic conditions are shown inTable 2

Table 2. Data of parameter values for endemic state simulation.

Λ_h	Λ_v	μ_h	μ_v	θ_H	θ_I	b	c
15	120	0.23	0.53	0.235	0.227	3.785	3.585
ω	ξ	τ	σ_v	α_1	α_2	α_3	η
0.375	3.375	0.335	3.425	0.784	0.745	0.753	0.523

Using data from Table 2, the value of the basic reproduction number is $R_0 \approx 6.77$. This indicates that, on average, one infected person can infect 6 susceptible individuals, suggesting the virus's sustained spread within the population. With this dataset, dynamic simulations for human and mosquito populations are depicted in Figures 4 and 5. Starting with an initial condition of 30 individuals in each human compartment and 50 in each mosquito compartment, the populations stabilize over time to an endemic equilibrium, where $(S_h^*, E_h^*, I_h^*, H_h^*, R_h^*, S_v^*, E_v^*, I_v^*) = (14, 3, 14, 10, 24, 95, 8, 30)$.

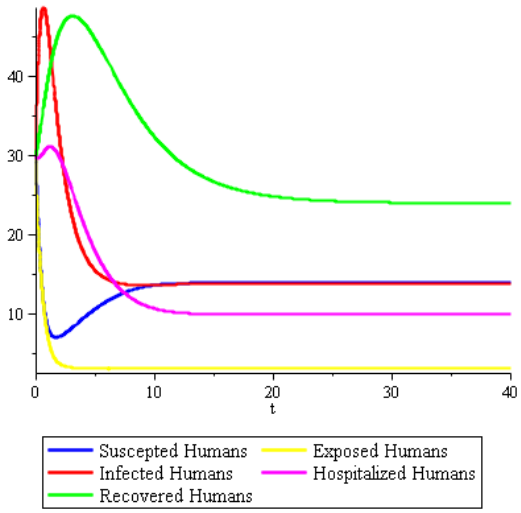

Figure 4. The dynamic of human population under the endemic condition.

Next, sensitivity analysis will be conducted to investigate how the change of parameters impact the basic reproduction number (R_0). Three parameters will be analyzed: the contact rate between susceptible humans and infected humans c, hospitalization rate of infected individuals τ, and mosquitos control rate ω. The normalized sensitivity index is calculated by normalizing the alteration in parameter values on the basic reproduction number R_0, as expressed in the following equation [17][23][24].

$$SI_p^{R_0} = \frac{\partial R_0}{\partial p}\frac{p}{R_0}, \tag{16}$$

where p is the parameter of system (1). Using endemic equilibrium (END) data in Table 2, the parameter sensitivity index to the basic reproduction number (R_0) is presented in Table 3, Meanwhile, graphical simulations for each parameter are illustrated in Figures 6, 7, and 8.

According to the sensitivity index in Table 3, the mosquito control rate (ω) and the contact rate between susceptible humans and infected humans (c) exert a significant impact on variations in the basic reproduction number (R_0) in comparison to the hospitalization rate of infected humans (τ). Specifically, c is directly proportional to R_0, indicating that an increase in the c value leads to an elevation in R_0. In contrast to c, the parameters τ and ω are inversely proportional to R_0, indicating

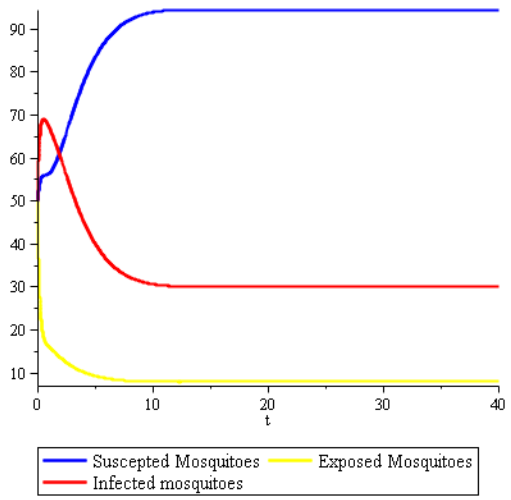

Figure 5. The dynamic of mosquitoes population under the endemic condition.

Table 3. Parameter sensitivity index and the change of R0

Parameter (P)	Indeks Sensitivitas	R_0			
		$P+10\%$	$P-10\%$	$P+20\%$	$P-20\%$
c	0.3040672221	6.828	6.713	6.885	6.656
τ	-0.1805690304	6.447	7.189	6.190	7.754
ω	-0.3185055932	6.256	7.421	5.842	8.268

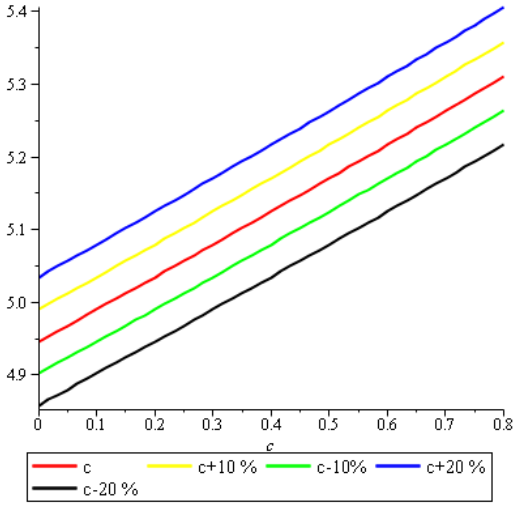

Figure 6. The effect of contact rate c on R_0

that an increase in either parameters results in a decrease in the value of R_0. The graphics illustrating the effect of changing parameters c, τ, and ω on the basic reproduction number (R_0) are presented in Figures 6, 7, 8.

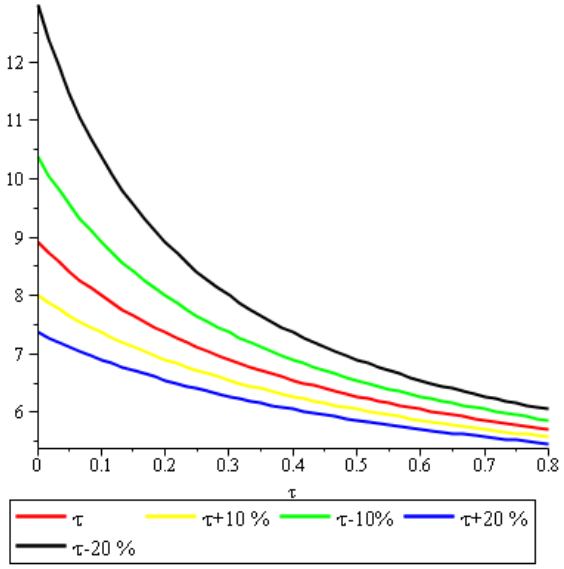

Figure 7. The effect of hospitalized rate τ on R_0

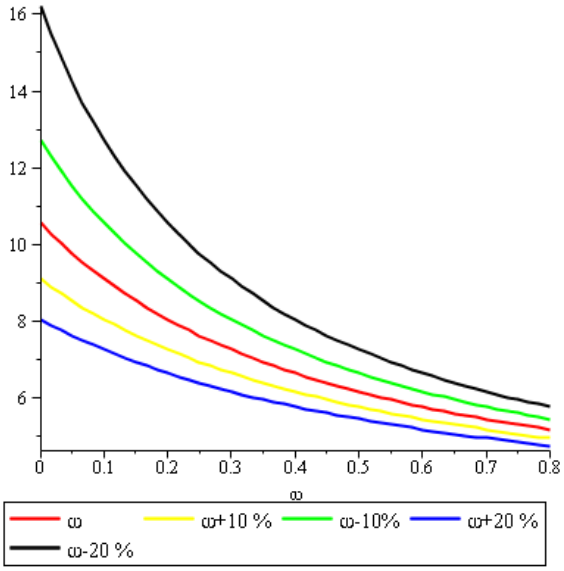

Figure 8. The effect of vector control rate ω on R_0

Conclusion

In this study, SEIRH-SEI mathematical model is constructed to describe the transmission of Zika virus. Human population is divided into five compartments: Susceptible Humans (S_h), Exposed Humans (E_h), Infected Humans (I_h), Hospitalized Humans (H_h) and Recovered Humans (R_h). Meanwhile the mosquito population (vector) is divided into three compartments, namely Susceptible Vectors (S_v), Exposed Vectors (E_v), and Infected Vectors (I_v). In human populations, the model assumes that the virus spreads through contact with infected humans or bites from virus-infected mosquitoes. As for mosquitoes, transmission occurs when susceptible mosquitoes bite infected humans. Additionally, a control vector is introduced to manage mosquito growth, potentially reducing virus transmission between humans and mosquitoes.

The analysis result reveals two equilibrium states: disease-free (DFE) and endemic (END). DFE stability is achieved when the basic reproduction number, representing the average number of new infections produced by one infected individual in a susceptible population, is $R_0 < 1$. Conversely,

END stability occurs when $R_0 > 1$. Sensitivity analysis based on endemic simulation data indicates that among the considered parameters (contact rate between susceptible and infected humans c, treatment rate for infected humans τ, and mosquito control rate ω), c and ω have a more pronounced impact on changing R_0 than τ. Essentially, reducing contact with infected individuals and implementing vector control are more effective strategies than isolating or treating infected patients.

Policy makers can prioritize minimizing human-to-human contact by implementing outreach programs to raise awareness about the spread of the Zika virus and promoting social distancing measures among affected individuals. Additionally, it is crucial to continue advocating for mosquito control measures, including fumigation and the use of anti-mosquito, particularly during the rainy season when mosquitoes are most active in breeding.

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