InPrime: Indonesian Journal of Pure and Applied Mathematics
Vol. 8, No. 1 (2026), pp. 1 – 16, doi: 10.15408/inprime.v8i1.50544
p-ISSN 2686-5335, e-ISSN: 2716-2478

Long-Term Dynamics of Chikungunya Transmission with Chronic Infection: Global Stability Analysis of An SICR-SI Model

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Abstract

Climate change and environmental factors have increased the risk of Chikungunya transmission in tropical regions, highlighting the need for mathematical models that capture its long-term epidemiological dynamics. This study proposes a modified SICR-SI compartmental model incorporating a chronic infection stage, where infected individuals may either recover directly or progress to a non-infectious chronic compartment before recovery. The model extends previous Chikungunya transmission frameworks by repositioning the chronic class before recovery and by establishing a new global stability analysis. Qualitative analysis is performed through positivity and invariant-region investigations, derivation of the basic reproduction number using the Next Generation Matrix approach, local stability analysis via eigenvalue and Routh–Hurwitz criteria, and global stability analysis using a Lyapunov function. The analysis shows that the disease-free equilibrium exists and is locally as well as globally asymptotically stable when the basic reproduction number satisfies $R_0 < 1$, whereas an endemic equilibrium exists and is locally asymptotically stable when $R_0 > 1$. Numerical simulations based on Chikungunya data from West Java Province, Indonesia, confirm the theoretical findings and indicate a disease-free scenario with $R_0 < 1$. Sensitivity exploration further demonstrates that increasing transmission probabilities between humans and mosquitoes significantly elevates R_0, potentially driving the system toward endemic persistence. These results emphasize the importance of reducing vector–host transmission intensity as an effective strategy for controlling Chikungunya outbreaks and provide a theoretical foundation for future epidemiological intervention studies.
Keywords: Chikungunya; SICR-SI model; Chronic infection; Basic reproduction number; Lyapunov function; Global stability.

Abstrak

Perubahan iklim dan faktor lingkungan telah meningkatkan risiko penularan Chikungunya di daerah tropis, menyoroti perlunya model matematika yang menangkap dinamika epidemiologi jangka panjangnya. Studi ini mengusulkan model kompartemen SICR-SI yang dimodifikasi yang menggabungkan tahap infeksi kronis, di mana individu yang terinfeksi dapat langsung pulih atau berkembang ke kompartemen kronis non-infeksius sebelum pulih. Model ini memperluas kerangka kerja penularan Chikungunya sebelumnya dengan memposisikan ulang kelas kronis sebelum pemulihan dan dengan menetapkan analisis stabilitas global baru. Analisis kualitatif dilakukan melalui investigasi positif dan wilayah invarian, derivasi angka reproduksi dasar menggunakan pendekatan Matriks Generasi Berikutnya, analisis stabilitas lokal melalui kriteria nilai eigen dan Routh–Hurwitz, dan analisis stabilitas global menggunakan fungsi Lyapunov. Analisis menunjukkan bahwa keseimbangan bebas penyakit ada dan stabil secara asimtotik lokal maupun global ketika angka reproduksi dasar memenuhi $R_0 < 1$, sedangkan keseimbangan endemik ada dan stabil secara asimtotik lokal ketika $R_0 > 1$. Simulasi numerik berdasarkan data Chikungunya dari Provinsi Jawa Barat, Indonesia, mengkonfirmasi temuan teoritis dan menunjukkan skenario bebas penyakit dengan $R_0 < 1$. Eksplorasi sensitivitas lebih lanjut menunjukkan bahwa peningkatan probabilitas penularan antara manusia dan nyamuk secara signifikan meningkatkan R_0, berpotensi mendorong sistem menuju persistensi endemik. Hasil ini menekankan pentingnya

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Submitted March 31st, 2026, Revised May 12th, 2026,
Accepted for publication May 16th, 2026, Published Online May 31st, 2026

mengurangi intensitas penularan vektor-inang sebagai strategi efektif untuk mengendalikan wabah Chikungunya dan memberikan landasan teoritis untuk studi intervensi epidemiologi di masa mendatang.
Kata Kunci: *Chikungunya; model SICR-SI; Infeksi kronis; Angka reproduksi dasar; Fungsi Lyapunov; Stabilitas global.*

2020MSC: 92D30, 37N25.

1. INTRODUCTION

Climate change in Indonesia may lead to an increase in infectious diseases. Chikungunya is a viral disease [1]. The first outbreak of the Chikungunya virus was recorded in 1973 in two regions: Samarinda and Jakarta [2]. According to data from [3], between January 2001 and April 2007, Indonesia recorded 15.207 cases of Chikungunya across seven provinces, with the outbreak peaking in 2003. In 2007, more than 1,200 suspected cases of Chikungunya infection were reported in 23 districts, with the majority originating from the Java region.

The Chikungunya virus is an RNA virus belonging to the genus Alphavirus, transmitted by the Aedes aegypti and Aedes albopictus mosquitoes. Transmission occurs when a mosquito bites a person carrying the virus. The disease is typically characterized by fever and joint pain [3]. The Chikungunya virus can infect people of all ages. An infected person cannot directly transmit the disease to others; transmission occurs only through mosquito bites [4].

People infected with the Chikungunya virus experience clinical symptoms, including sudden high fever, joint pain ranging from mild to severe, muscle pain in weight-bearing muscles, and so on. Joint pain is a common symptom among those infected with the Chikungunya virus and typically resolves within less than a month; however, it can persist for more than a month, leading to prolonged joint pain [2]. Therefore, this study will focus on chronic cases that require monitoring by health authorities [5].

The spread of vector-borne diseases is influenced by the dynamics of interactions between the human and vector population; therefore, in-depth research is needed to understand the patterns and mechanisms of transmission within a population [6]. One approach that can be used is mathematical modeling. Mathematical models can be used to systematically analyze the dynamics of infectious disease spread and population interactions through equilibrium analysis, system stability, and numerical simulation to understand the behavior of disease spread within a population [7, 8, 9]. Thus, mathematical models serve as an effective tool in supporting efforts to control and prevent Chikungunya.

Research by Haque et al. [10] used the SIR-SI mathematical model, taking into account saturation rates and treatment functions. Another research by Islam and Podder [11] developed the SEIR-SEI mathematical model and also considered treatment for infected individuals. Research by Arora et al. [12] used the SEIR'R-XYZ mathematical model, in which there is a recovery period before individuals fully recover. There is also research by González-Parra et al. [5] that developed the SEIRC-SEI mathematical model, in which the chronic compartment exists after the recovery compartment.

This study addresses several gaps in the existing literature. First, a modified SICR-SI model is proposed by placing the chronic compartment before the recovery compartment, because the chronic compartment is a group of individuals who have passed through the acute phase of the disease and are therefore no longer contagious, but have not yet fully recovered [13]. Therefore, this study introduces a modified SICR model, differentiating itself from existing SIRC frameworks by redefining

the C_h compartment as a non-infectious early-chronic stage. This differs from the work of Gonzalez-Parra et al. [5]. Second, a new Lyapunov function is developed to establish global stability. Third, the model's behavior is further illustrated through numerical simulations using parameter adoption from official empirical sources and relevant literature. In this study, we use real data on chikungunya cases in West Java in 2024.

The structure of this article is organized as follows. Section 2 presents the development of the SICR-SI mathematical model, including the model assumptions, variables, parameters, and governing equations. Section 3 provides the analytical results of the model, beginning with the positivity of solutions and the invariant region, followed by the transformation into a dimensionless system. This section also derives the equilibrium points and the basic reproduction number, and investigates the local and global stability properties of the disease-free and endemic equilibria using eigenvalue analysis, the Routh–Hurwitz criterion, and Lyapunov methods. Numerical simulations are then conducted to illustrate the theoretical findings and explore the transmission dynamics of Chikungunya under different epidemiological scenarios. Finally, Section 4 discusses the biological interpretation of the results, the epidemiological implications of the model, its limitations, and potential directions for future research.

2. METHODS

The model to be used for modeling the spread of Chikungunya is the continuous SICR-SI (Susceptible, Infected, Chronic, Recovered, Susceptible, Infected) model, which is a modification of the study by González-Parra et al. [5]. In the spread of Chikungunya, the human population is divided into four compartments: susceptible humans, denoted by $S_h(t)$, which is the population of humans susceptible to Chikungunya virus infection at the time t; infected humans, denoted by $I_h(t)$, which is the population of humans infected with the Chikungunya virus at the time t; chronic humans, denoted by $C_h(t)$, which is the population of humans experiencing chronic symptoms of Chikungunya at the time t, who have become non-infectious and recovered humans, denoted by $R_h(t)$, which is the population of humans who have recovered from Chikungunya virus infection at the time t. The mosquito population is divided into two compartments: the susceptible vector population, denoted by $S_v(t)$, consists of mosquitoes susceptible to Chikungunya virus infection at the time t and the infected vector population, denoted by $I_v(t)$, consists of mosquitoes infected with the Chikungunya virus at the time t.

The assumptions used to develop the mathematical model of Chikungunya disease transmission are as follows:

1. Every individual born into the human population is a susceptible individual who has a probability of becoming infected, and all susceptible mosquitoes have the same probability of becoming infected.
2. The parameter μ_h represents the human birth rate. The birth rate (μ_h) is assumed to be equal to the human death rate.
3. The parameter μ_v represents the mosquito growth rate and is assumed to be equal to the mosquito mortality rate.
4. Susceptible individuals can become part of the infected subpopulation through transmission via the bite of an infected mosquito with parameter b and a virus transmission probability β_h. Thus, $\beta_h b$ represents the effective transmission rate of the virus.

5. Susceptible mosquitoes can become infected if effective transmission occurs when they bite an infected individual, with a probability of β_v.
6. Infected individuals transition to the chronic subpopulation at a rate of α_h. In the chronic subpopulation, individuals are no longer infected and cannot transmit the Chikungunya virus
7. Infected individuals subsequently recover from the virus at a rate of γ_h.
8. The rate at which individuals recover from chronic symptoms following a viral infection is ϕ_h.
9. The human and mosquito populations in question are located in the same area, and the absence of migration between regions.

Table 1. Definition of the parameters

Par.	Definition	Unit	Par.	Definition	Unit
μ_h	Natural birth and death rates of the human population	day^{-1}	ϕ_h	Recovery rate from chronic conditions	day^{-1}
β_h	Transmission probability from infected mosquitoes to susceptible humans	-	β_v	The possibility of virus transmission from infected individuals to susceptible mosquitoes	-
γ_h	Recovery rate among infected individuals	day^{-1}	μ_v	Birth and death rates in mosquito populations	day^{-1}
α_h	Progression rate from infected to chronic phase	day^{-1}	b	Mosquito biting rate	$\frac{\text{bite}}{\text{mosquito. day}}$

Table 1 shows the definitions of each parameter. Based on the assumptions, variables, and parameters described above, a mathematical model of Chikungunya disease transmission within a population was developed, as represented by the transfer diagram in Figure 1.

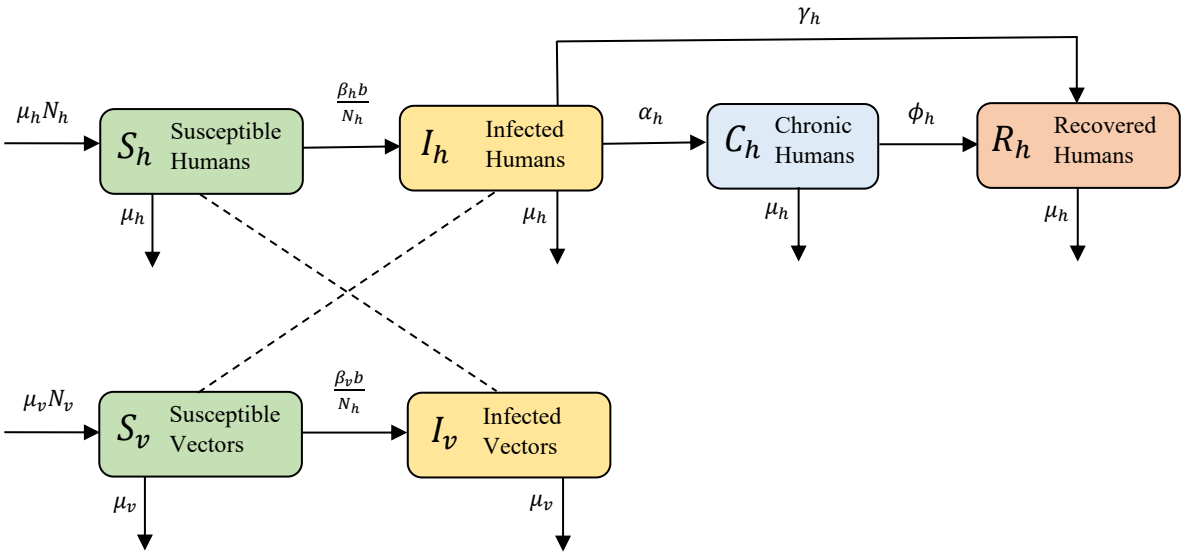

Figure 1. Transfer diagram illustrating the interaction between human (SICR) and vector (SI) populations, including infection transmission and progression pathways.

The mathematical model corresponding to the transfer diagram is given by the following system of nonlinear differential equations:

$$\begin{aligned}
\frac{dS_h(t)}{dt} &= \mu_h N_h(t) - \frac{\beta_h b S_h(t) I_v(t)}{N_h(t)} - \mu_h S_h(t),\\
\frac{dI_h(t)}{dt} &= \frac{\beta_h b S_h(t) I_v(t)}{N_h(t)} - \alpha_h I_h(t) - \gamma_h I_h(t) - \mu_h I_h(t),\\
\frac{dC_h(t)}{dt} &= \alpha_h I_h(t) - \phi_h C_h(t) - \mu_h C_h(t),\\
\frac{dR_h(t)}{dt} &= \gamma_h I_h(t) + \phi_h C_h(t) - \mu_h R_h(t),\\
\frac{dS_v(t)}{dt} &= \mu_v N_v - \frac{\beta_v b S_v(t) I_h(t)}{N_h(t)} - \mu_v S_v(t),\\
\frac{dI_v(t)}{dt} &= \frac{\beta_v b S_v(t) I_h(t)}{N_h(t)} - \mu_v I_v(t),
\end{aligned} \tag{1}$$

where $N_h(t) = S_h(t) + I_h(t) + C_h(t) + R_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$.

3. RESULTS

3.1. Positivity of Solutions

To ensure that the model solutions represent real-world population conditions, it is necessary to show that the system solutions are non-negative, since negative population values have no biological meaning [14]. Therefore, we demonstrate that the solutions remain non-negative for all times.

Theorem 1. The solutions to the system (1) in the set of non-negative real vectors $\mathbb{R}_+^6$ are always non-negative for every time.

Proof.
Suppose that the solution to system (1) depends on $[0, \sigma)$, where $0 < \sigma < \infty$. We will prove this by reductio ad absurdum. Based on the equation $S_h(t)$, will be shown $S_h(t) > 0$ for every $t \in [0, \sigma)$. If this condition is not satisfied, then there exists $t_1 \in (0, \sigma)$ such that $S_h(t_1) \leq 0$, therefore, $\frac{dS_h(t)}{dt}|_{t=t_1} \leq 0$ and consequences $S_h(t) > 0$ for every $t \in [0, t_1)$. Next, do the same for the equation $I_h(t)$, will be shown $I_h(t) \geq 0$ for every $t \in [0, t_1)$. If this is not satisfied, then there exists $t_2 \in (0, t_1)$ such that $I_h(t_2) < 0$, therefore, $\frac{dI_h(t)}{dt}|_{t=t_2} < 0$ and we have $I_h(t) \geq 0$ for every $t \in [0, t_1)$.

We will proceed in the same manner for the equation $C_h(t)$. Will be shown $C_h(t) \geq 0$ for every $t \in [0, t_2)$. If this is not satisfied, then there exists $t_3 \in (0, t_2)$ such that $C_h(t_3) < 0$. Thus, we obtain $\frac{dC_h(t)}{dt}|_{t=t_3} = \alpha_h I_h(t_3) - \phi_h C_h(t_3) - \mu_h C_h(t_3) \geq 0$, with $C_h(t_3) < 0$, $\phi_h > 0$, and $\mu_h > 0$. This contradicts the assumption that $\frac{dC_h(t)}{dt}|_{t=t_3} < 0$. This implies that $C_h(t) \geq 0$ for every $t \in [0, t_2)$. Next, for the equation $R_h(t)$, will be shown $R_h(t) \geq 0$ for every $t \in [0, t_3)$. If this is not satisfied, then there exists $t_4 \in (0, t_3)$ such that $R_h(t_4) < 0$. Thus, we obtain $\frac{dR_h(t)}{dt}|_{t=t_4} = \gamma_h I_h(t_4) + \phi_h C_h(t_4) - \mu_h R_h(t_4) \geq 0$, with $R_h(t_4) < 0$ and $\mu_h > 0$. This contradicts the assumption that $\frac{dR_h(t)}{dt}|_{t=t_4} < 0$. This shows that $R_h(t) \geq 0$ for every $t \in [0, t_3)$.

Similarly, for the equations $S_v(t)$ and $I_v(t)$. Will be shown $S_v(t) > 0$ for every $t \in [0, \sigma)$. If this is not satisfied, then there exists $t_1 \in (0, \sigma)$ such that $S_v(t_1) \leq 0$. Thus, we obtain $\frac{dS_v(t)}{dt}|_{t=t_1} = \mu_v N_v -$

$\frac{\beta_v b S_v(t_1) I_h(t_1)}{N_h(t_1)} - \mu_v S_v(t_1) > 0$. This contradicts the assumption that $\frac{dS_v(t)}{dt}|_{t=t_1} \leq 0$. This implies that $S_v(t) > 0$ for every $t \in [0, \sigma)$. Next, will be shown $I_v(t) \geq 0$ for every $t \in [0, t_1)$. If this is not satisfied, then there exists $t_2 \in (0, t_1)$ such that $I_v(t_1) < 0$. Thus, we obtain $\frac{dI_v(t)}{dt}|_{t=t_2} = \frac{\beta_v b S_v(t_2) I_h(t_2)}{N_h(t_2)} - \mu_v I_v(t_2) \geq 0$. This contradicts the assumption that $\frac{dI_v(t)}{dt}|_{t=t_2} < 0$. This implies that $I_v(t) \geq 0$ for every $t \in [0, t_1)$. Therefore, all solutions to system (1) are non-negative for every time t in $[0, \sigma)$, where $0 < \sigma < \infty$. ■

3.2. Invariant Region

To verify that the system solutions remain within a biologically reasonable range, an invariant region will be defined [12].

Theorem 2. The region $\Omega = \{(\mathrm{S_h}, \mathrm{I_h}, \mathrm{C_h}, \mathrm{R_h}, \mathrm{S_v}, \mathrm{I_v}) \in \mathbb{R}_+^6 : 0 < \mathrm{S_h} + \mathrm{I_h} + \mathrm{C_h} + \mathrm{R_h} \leq \mathrm{N_{h0}}, 0 < \mathrm{S_v} + \mathrm{I_v} \leq \mathrm{N_{v0}}\}$ is a positive invariant region for System (1), with non-negative initial conditions $\mathbb{R}_+^6$.

Proof.
The human population is obtained by summing the equation $S_h(t) + I_h(t) + C_h(t) + R_h(t)$. Thus, the human population is obtained from $\frac{dN_h(t)}{dt} = 0$. By integrating both sides of $\frac{dN_h(t)}{dt} = 0$, the solution is given by $N_h(t) = N_{h0}$. For $t \to \infty$, then $\lim_{t\to\infty} N_h(t) = N_{h0}$. In the same way, the vector population is obtained by summing the vector equation $S_v(t) + I_v(t)$. Thus, the vector population is obtained as $\frac{dN_v(t)}{dt} = 0$ with the solution given as $N_v(t) = N_{v0}$. Thus, when $t \to \infty$, we have $\lim_{t\to\infty} N_v(t) = N_{v0}$. ■

3.3. Transformation of Mathematical Models

Next, to simplify the notation, we will use $N_h(t) = N_h$, $N_v(t) = N_v$, $S_h(t) = S_h$, $I_h(t) = I_h$, $C_h(t) = C_h$, $R_h(t) = R_h$, $S_v(t) = S_v$, $I_v(t) = I_v$. The mathematical model of Chikungunya transmission can be scaled using the total populations N_h and N_v, so that the analysis can be simplified. The number of individuals in each compartment is then given as follows:

$$s_h = \frac{S_h}{N_h}, i_h = \frac{I_h}{N_h}, c_h = \frac{C_h}{N_h}, r_h = \frac{R_h}{N_h}, s_v = \frac{S_v}{N_v}, i_v = \frac{I_v}{N_v}.$$

Then, from the transformation results, the function r_h is a function of s_h, i_h, and c_h and function s_v merupakan fungsi dalam i_h dan i_v. Thus, the number of individuals in each compartment can be focused on the following equation:

$$\begin{aligned} \frac{ds_h}{dt} &= \mu_h - \beta_h b \theta i_v s_h - \mu_h s_h, \\ \frac{di_h}{dt} &= \beta_h b \theta i_v s_h - (\alpha_h + \gamma_h + \mu_h) i_h, \\ \frac{dc_h}{dt} &= \alpha_h i_h - (\phi_h + \mu_h) c_h, \\ \frac{di_v}{dt} &= \beta_v b (1 - i_v) i_h - \mu_v i_v, \end{aligned} \tag{2}$$

where $\theta = \frac{N_v}{N_h}$, $s_h + i_h + c_h + r_h = 1$, and $s_v + i_v = 1$.

3.4. Equilibrium Point

3.4.1. Disease-Free Equilibrium Point

By equating the right-hand side of System (2) to zero, we can find the equilibrium points of the model [15]. The first point is denoted as the disease-free equilibrium (DFE), and the other point is called the endemic equilibrium (EE). Thus, the DFE equilibrium point is obtained as DFE = $(s_h, i_h, c_h, i_v) = (1,0,0,0)$. On the other side, the EE equilibrium point is obtained as $(s_h^*, i_h^*, c_h^*, i_v^*)$. We will discuss the endemic equilibrium point and the conditions that cause.

3.4.2. Basic Reproduction Number

In infectious diseases, there is a key threshold parameter for transmission known as the basic reproduction number, usually denoted as R_0. The basic reproduction number R_0 is defined as the average number of secondary cases generated by a single infected individual in an entirely disease-free population [16]. To calculate the basic reproduction number R_0 for the mathematical model, the Next Generation Matrix technique is used [17]. The infection compartments in this chikungunya transmission model are i_h and i_v. Thus, at the DFE, we obtain,

$$F = \begin{bmatrix} 0 & \beta_h b\theta s_h \\ \beta_v b(1-i_v) & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} \alpha_h + \gamma_h + \mu_h & 0 \\ 0 & \mu_v \end{bmatrix}$$

where the F matrix is related to the rate of increase in secondary infections and the V matrix is related to the rates of disease progression, recovery, and death. Then, from the F and V matrices, the dominant eigenvalue of the R_0 matrix can be obtained as $R_0 = \rho(FV^{-1})$, with the dominant eigenvalue as follows:

$$\widehat{R_0} = \sqrt{\frac{\beta_h \beta_v b^2 \theta}{\mu_v(\alpha_h + \gamma_h + \mu_h)}}.$$

Furthermore, if $R_0 = \frac{\beta_h \beta_v b^2 \theta}{\mu_v(\alpha_h+\gamma_h+\mu_h)}$, then $\widehat{R_0} = \sqrt{R_0}$.

3.4.3. Endemic Equilibrium Point

To find the endemic equilibrium point, by setting the right-hand side of Equation (2) equal to zero, we find that at least one of the infection component values is nonzero. The values of the endemic equilibrium point $EE = (s_h^*, i_h^*, c_h^*, i_v^*)$ are obtained, where

$$s_h^* = \frac{\beta_v b\mu_v\mu_h^2(R_0-1)+\mu_h\mu_v(R_0\mu_v(\alpha_h+\gamma_h+\mu_h)+\beta_v b\mu_h)}{(\beta_h b\theta+\mu_h)(\beta_v b\mu_v\mu_h(R_0-1))+\mu_h\mu_v(R_0\mu_v(\alpha_h+\gamma_h+\mu_h)+\beta_v b\mu_h)},$$

$$i_h^* = \frac{\mu_v\mu_h}{R_0\mu_v(\alpha_h+\gamma_h+\mu_h)+\beta_v b\mu_h}(R_0-1),$$

$$c_h^* = \frac{\alpha_h\mu_v\mu_h}{(\phi_h+\mu_h)(R_0\mu_v(\alpha_h+\gamma_h+\mu_h)+\beta_v b\mu_h)}(R_0-1),$$

$$i_v^* = \frac{\beta_v b\mu_v\mu_h}{\beta_v b\mu_v\mu_h(R_0-1)+R_0\mu_v^2(\alpha_h+\gamma_h+\mu_h)+\beta_v b\mu_v\mu_h}(R_0-1).$$

Theorem 3. Let EE = $(s_h^*, i_h^*, c_h^*, i_v^*)$ be an endemic equilibrium point of System (2). If $R_0 > 1$, then the equilibrium point EE exists.

Proof.
The endemic equilibrium point means that there are always individuals infected with the disease within the population; therefore, all compartments representing infected individuals must have positive values, that is,

$$s_h^* > 0, i_h^* > 0, c_h^* > 0, \text{ and } i_v^* > 0.$$

If $R_0 > 1$, then $(R_0 - 1) > 0$. Each compartment $s_h^*, i_h^*, c_h^*, i_v^*$ contains a factor $(R_0 - 1)$, with all parameters being positive. Thus, it follows that

$$s_h^* > 0, i_h^* > 0, c_h^* > 0, \text{ and } i_v^* > 0.$$

From the results above, it has been shown that $s_h^* > 0, i_h^* > 0, c_h^* > 0, i_v^* > 0$ if $R_0 > 1$. ■

3.5. Analysis of The Model

3.5.1. Analysis of the Local Stability of Disease-Free Equilibrium Points

Theorem 4. Given the system of equations (2), where the Jacobian matrix of the DFE system is $\mathrm{J_{DFE}}$. The disease-free equilibrium point of System (2) is locally asymptotically stable if the real part of the eigenvalues of $\mathrm{J_{DFE}}$ is negative.

Proof.
The Jacobian matrix of system (2) is obtained by linearizing the mathematical model of Chikungunya disease spread around the equilibrium point $DFE = (1,0,0,0)$, is as follows:

$$J_{DFE} = \begin{bmatrix} -\mu_h & 0 & 0 & -\beta_h b\theta \\ 0 & -(\alpha_h + \gamma_h + \mu_h) & 0 & \beta_h b\theta \\ 0 & \alpha_h & -(\phi_h + \mu_h) & 0 \\ 0 & 0 & 0 & -\mu_v \end{bmatrix}.$$

Let λ be an eigenvalue of the matrix J_{DFE}, then λ is obtained from the equation $det(\lambda I - J_{DFE}) = 0$. Through algebraic calculation, the values of $\lambda_1 = -\mu_h$, $\lambda_2 = -(\phi + \mu_h)$, $\lambda_3 = -(\alpha_h + \gamma_h + \mu_h)$, and $\lambda_4 = -\mu_v$ are obtained. Recalling that the parameters are positive, it is seen that all eigenvalues of the Jacobian matrix J_{DFE} are negative. Therefore, it can be concluded that the disease-free equilibrium point DFE is locally asymptotically stable. ■

3.5.2. Analysis of the Global Stability of Disease-Free Equilibrium Points

Lyapunov functions will be used to analyze the global stability around the disease-free equilibrium point [14]. The system of equations (2) has the following invariant region:

$$\Omega = \{(s_h, i_h, c_h, i_v) \in \mathbb{R}_+^4 : s_h, i_h, c_h, i_v \geq 0\}.$$

Theorem 5. The disease-free equilibrium point $\mathrm{DFE} = (1,0,0,0)$. The system of equations (2) is globally asymptotically stable in the region Ω if $\mathrm{R_0} < 1$.

Proof.
To analyze the global stability of the disease-free equilibrium point, the Lyapunov function is defined as follows:

$$P = \frac{\beta_h b\theta}{\mu_v}\, i_v + i_h.$$

Since $i_h \geq 0$ and $i_v \geq 0$, then $P(0,0) = 0$; and since the parameters are positive, then $P(i_h, i_v) > 0, \forall (i_h, i_v) \neq (0,0)$. For every $\|(i_h, i_v)\| \to \infty$, we have $P(i_h, i_v) = \frac{\beta_h b\theta}{\mu_v} i_v + i_h \to \infty$. By taking the derivative of the Lyapunov function P with respect to time along the solutions of system (2) we obtain:

$$\dot{P} = -\big(1 - R_0(1 - i_v)\big)(\alpha_h + \gamma_h + \mu_h) i_h - (1 - s_h)\beta_h b\theta i_v.$$

If $R_0 < 1$, then we have $\dot{P} < 0, \forall (i_h, i_v) \neq (0,0)$. Therefore, the disease-free equilibrium point E_0 is globally asymptotically stable in Ω for $R_0 < 1$. ■

3.5.3. Analysis of the Local Stability of Endemic Equilibrium Points

Theorem 6. Consider System (2) and let $\mathrm{J_{EE}}$ be the Jacobian matrix evaluated at the endemic equilibrium EE. Then, EE is locally asymptotically stable if $\mathrm{R_0} > 1$.

Proof.
The Jacobian matrix of system (2) is obtained by linearizing the mathematical model of Chikungunya disease spread around the equilibrium point $EE = (s_h^*, i_h^*, c_h^*, i_v^*)$ where

$$s_h^* = \frac{\beta_v b\mu_v\mu_h^2(R_0-1) + \mu_h\mu_v(R_0\mu_v(\alpha_h+\gamma_h+\mu_h) + \beta_v b\mu_h)}{(\beta_h b\theta+\mu_h)\big(\beta_v b\mu_v\mu_h(R_0-1)\big) + \mu_h\mu_v(R_0\mu_v(\alpha_h+\gamma_h+\mu_h) + \beta_v b\mu_h)},$$

$$i_h^* = \frac{\mu_v\mu_h}{R_0\mu_v(\alpha_h+\gamma_h+\mu_h) + \beta_v b\mu_h}(R_0 - 1),$$

$$c_h^* = \frac{\alpha_h\mu_v\mu_h}{(\phi_h+\mu_h)(R_0\mu_v(\alpha_h+\gamma_h+\mu_h) + \beta_v b\mu_h)}(R_0 - 1),$$

$$i_v^* = \frac{\beta_v b\mu_v\mu_h}{\beta_v b\mu_v\mu_h(R_0-1) + R_0\mu_v^2(\alpha_h+\gamma_h+\mu_h) + \beta_v b\mu_v\mu_h}(R_0 - 1),$$

is as follows:

$$J_{EE} = \begin{bmatrix} -\beta_h b\theta i_v^* - \mu_h & 0 & 0 & -\beta_h b\theta s_h^* \\ \beta_h b\theta i_v^* & -(\alpha_h + \gamma_h + \mu_h) & 0 & \beta_h b\theta s_h^* \\ 0 & \alpha_h & -(\phi_h + \mu_h) & 0 \\ 0 & \beta_v b(1 - i_v^*) & 0 & -\beta_v b i_h^* - \mu_v \end{bmatrix}.$$

It has been proven that if $R_0 > 1$, then the equilibrium point EE exists. Let λ be an eigenvalue of the matrix J_{EE}, then λ is obtained from the equation $\det(\lambda I - J_{EE}) = 0$. Through algebraic calculations, we obtain

$$\lambda = -(\phi_h + \mu_h),$$

and

$$\lambda^3 + (\beta_h b\theta i_v^* + \beta_v b i_h^* + 2\mu_h + \mu_v + \alpha_h + \gamma_h)\lambda^2$$
$$+ \big((\beta_h b\theta i_v^* + \mu_h)(\alpha_h + \gamma_h + \mu_h) + (\beta_h b\theta i_v^* + \mu_h)(\beta_v b + \mu_v)$$
$$+ (\alpha_h + \gamma_h + \mu_h)(\beta_v b i_h^* + \mu_v) - \beta_h\beta_v b^2\theta s_h^*(1 - i_v^*)\big)\lambda$$
$$+(\beta_h b\theta i_v^* + \mu_h)(\alpha_h + \gamma_h + \mu_h)(\beta_v b i_h^* + \mu_v) - \beta_h\beta_v b^2\theta(1 - i_v^*)(\beta_h b\theta i_v^* + \mu_h)$$
$$+\beta_h^2\beta_v b^3\theta^2 s_h^*(1 - i_v^*) = 0.$$

For cubic polynomial equations, the Routh-Hurwitz criterion will be used to determine the sign of the real part of the eigenvalues. From the cubic polynomial equation, we obtain [18]

$a_0 = 1,$
$a_1 = \beta_h b\theta i_v^* + \beta_v b i_h^* + 2\mu_h + \mu_v + \alpha_h + \gamma_h,$
$a_2 = (\beta_h b\theta i_v^* + \mu_h)(\alpha_h + \gamma_h + \mu_h) + (\beta_h b\theta i_v^* + \mu_h)(\beta_v b + \mu_v) + (\alpha_h + \gamma_h + \mu_h)(\beta_v b i_h^* + \mu_v) - \beta_h\beta_v b^2\theta s_h^*(1 - i_v^*),$
$a_3 = (\beta_h b\theta i_v^* + \mu_h)(\alpha_h + \gamma_h + \mu_h)(\beta_v b i_h^* + \mu_v) - \beta_h\beta_v b^2\theta(1 - i_v^*)(\beta_h b\theta i_v^* + \mu_h) + \beta_h^2\beta_v b^3\theta^2 s_h^*(1 - i_v^*).$

Using algebraic manipulation, assuming all parameters are positive and given that when $R_0 > 1$ the equilibrium point EE exists, it can be proven that $a_0 > 0, a_1 > 0, a_2 > 0,$ and $a_3 > 0$. It is known that $\beta_h\beta_v b^2\theta s_h^*(1 - i_v^*) < (\beta_h b\theta i_v^* + \mu_h)(\beta_v b i_h^* + \mu_v)$, and thus it is also proven that $a_1 a_2 > a_3$. Therefore, it can be concluded that the real parts of all eigenvalues of the Jacobian matrix J_{EE} are negative. It is proven that the endemic equilibrium point EE is locally asymptotically stable. ■

3.6. Numerical Simulation

Based on the results of the theory analysis conducted, the model will be interpreted using the variable and parameter values from the numerical simulation. The initial values used for the numerical simulation of the Chikungunya transmission model are from West Java Province, covering Garut Regency, Cirebon Regency, and Cirebon City in 2024. Data from Badan Pusat Statistik (BPS) and the health profile of West Java Province indicate a total population of 5.449.760 individuals from the three regencies/cities. Of the total population, 430 individuals are infected, and 5.449.330 are susceptible individuals [19, 20]. Table 2 shows the values of the variables and parameters used, which are based on previous literature, with the following parameter values and the initial values for each variable.

Table 2. The values of the variables and parameters

Parameter	Value	References
μ_h	0.0136	[21]
γ_h	0.0073	[10]
α_h	0.33	[12]
ϕ_h	0.142	[12]
μ_v	0.016	[12]
b	2	Assumed
θ	0.0662	[19, 22]
Variable	**Initial Value**	**References**
s_h	0.99992	[19, 20]
i_h	0.00008	[19, 20]
c_h	0.0	Assumed [20]
i_v	0.019	Assumed [22]

Using the parameter values β_h and β_v, with $\beta_h = 0.361$ and $\beta_v = 0.012$ the value of $R_0 \approx 0.20431 < 1$ is obtained [5]. On the other side, using $\beta_h = 0.587$ and $\beta_v = 0.553$ we obtain $R_0 \approx 15.31 > 1$. Using the parameter values in Table 2, we obtain Figure 2, which corresponds to Theorem 4.

Figure 2 confirms Theorem 4, in which the spread of Chikungunya shows that the proportion of susceptible individuals decreases and then increases again toward one, while the proportions of infected and chronic individuals each reach an epidemic peak before eventually decreasing toward zero. Similarly, the population of infected mosquitoes continues to decrease until it stabilizes at zero. Figure 3 confirms Theorem 5, the three relationships between variables—infected individuals and infected mosquitoes, infected individuals and chronic individuals, and susceptible individuals and infected individuals—show the same pattern, where all variables eventually converge toward the disease-free equilibrium point even with different initial conditions. This also holds for all initial values far from the disease-free equilibrium point.

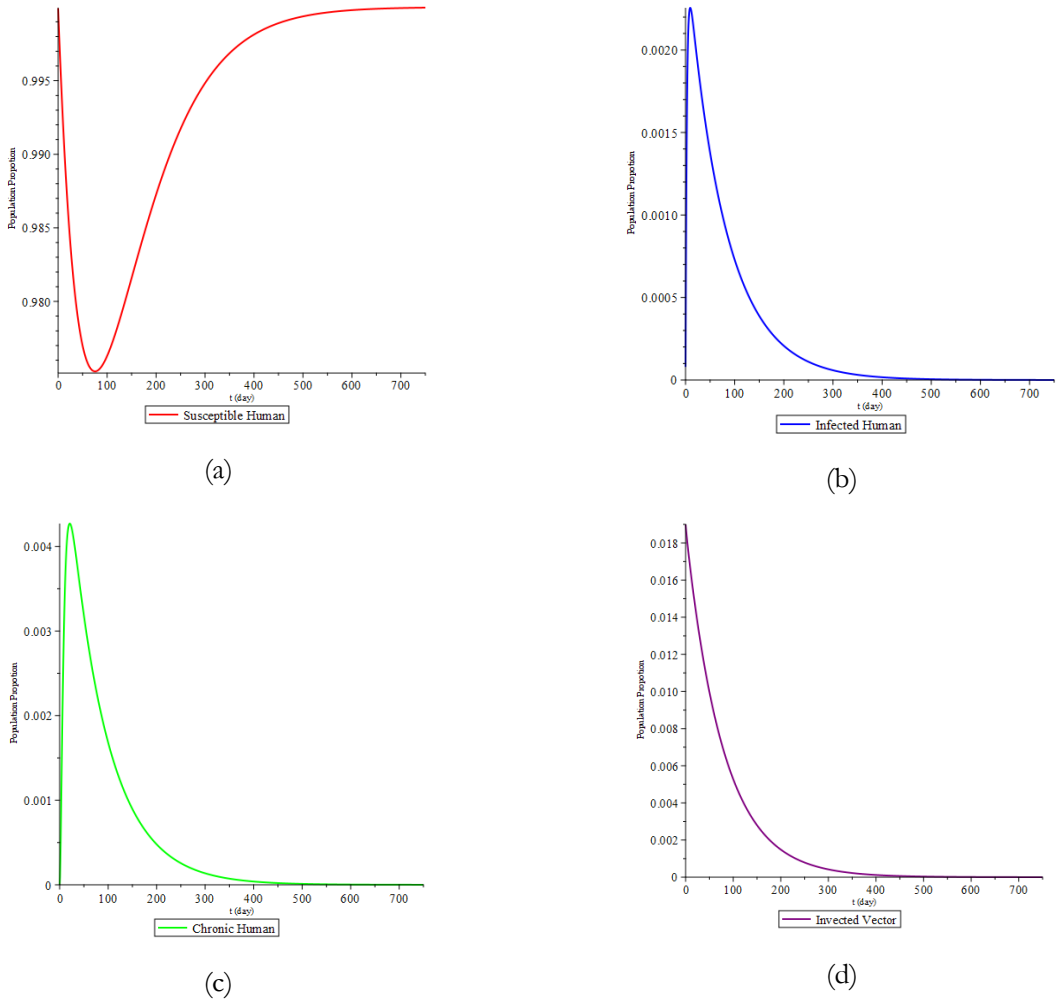

Figure 2. Numerical Simulation of Disease-Free Equilibrium Points $R_0 < 1$.

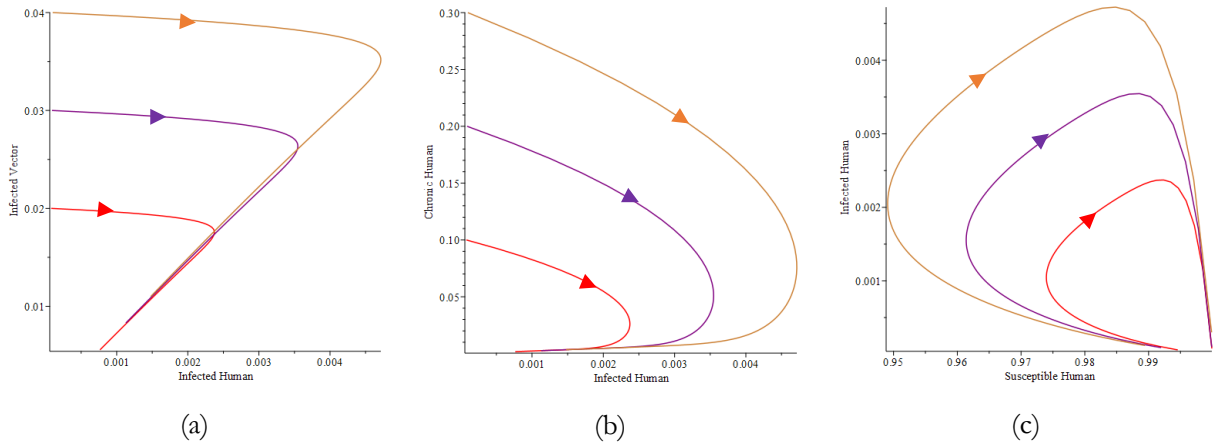

Figure 3. Time Evolution of Model Variables at the Disease-Free Equilibrium

Figure 4 confirms Theorem 6, in endemic conditions, it is shown that all variables fluctuate before eventually stabilizing around the endemic equilibrium point, namely susceptible individuals stabilize around 0.204, infected individuals stabilize around 0.031, chronic individuals stabilize around 0.065, and infected mosquitoes stabilize around 0.681. This occurs due to an increase in the contact rate parameter between infected mosquitoes and susceptible individuals, as well as between infected individuals and susceptible mosquitoes, which results in an $R_0 > 1$. Therefore, it can be said that the disease is sustained in the population and has the potential to become endemic.

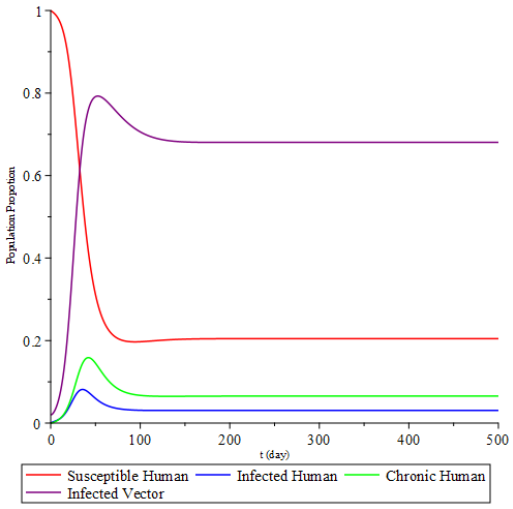

Figure 4. Numerical Simulation of Endemic Equilibrium Points $R_0 > 1$.

Figure 5 shows the relationship between the transmission probability parameters β_h and β_v and the basic reproduction number R_0. The surface plot in Figure 5a shows that an increase in both β_h and β_v leads to an increase in R_0, indicating that the intensity of contact between individuals and mosquitoes plays a significant role in determining the potential spread of Chikungunya. Figure 5b shows contour curves for constant R_0 values of 0.5, 1.2, and 2.2, where combinations of β_h and β_v values in the $R_0 > 1$ region drive the system toward endemic conditions, while combinations in the $R_0 < 1$ region indicate that the spread of Chikungunya tends to decrease toward a disease-free state.

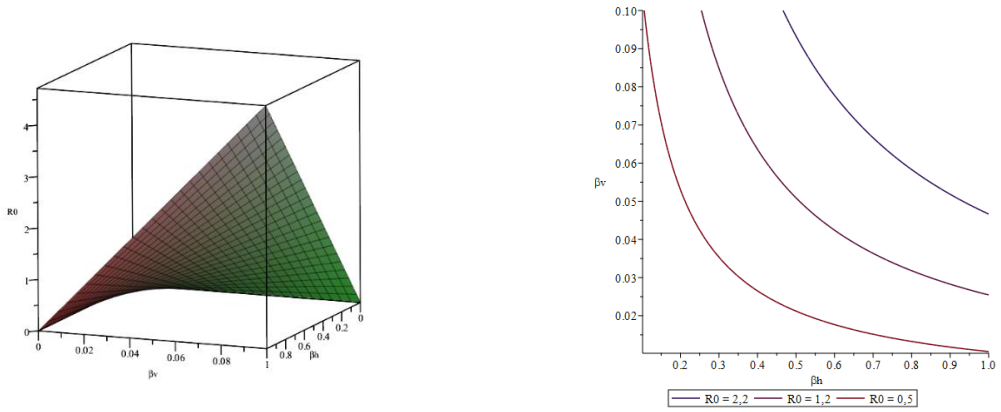

Figure 5. Relationship between β_h, β_v and R_0.

4. DISCUSSION

This study developed a modified SICR-SI model to describe the transmission dynamics of Chikungunya involving interactions between human and mosquito populations. Similar to previous compartmental models proposed by Haque et al. [10], Islam and Podder [11], and Arora et al. [12], the present model incorporates vector–host transmission mechanisms and uses the basic reproduction number as the main threshold parameter governing disease persistence. However, unlike the model developed by González-Parra et al. [5], the chronic compartment is positioned before the recovery compartment, reflecting clinical evidence that chronic Chikungunya patients experience prolonged symptoms but are no longer infectious [13].

The analytical results show that the disease-free equilibrium is locally and globally asymptotically stable when $R_0 < 1$. This finding is consistent with the general theory of infectious disease dynamics, which states that a disease cannot invade a population when the basic reproduction number remains below unity [16,17]. Similar stability behavior has also been reported in mathematical models of Chikungunya transmission [10–12] and in other vector-borne disease models [14].

The existence and local stability of the endemic equilibrium for $R_0 > 1$indicate that Chikungunya can persist within the population once transmission exceeds the epidemic threshold. This result agrees with the findings of González-Parra et al. [5] and Arora et al. [12], who demonstrated that increased transmission intensity between humans and mosquitoes can sustain long-term disease circulation. The numerical simulations further confirm that larger transmission probabilities lead to higher values of R_0, emphasizing the critical role of vector–host contact rates in disease spread.

From an epidemiological perspective, the chronic compartment acts as a non-infectious class that reduces the number of actively infectious individuals. This interpretation is supported by clinical studies showing that chronic Chikungunya manifestations are characterized mainly by persistent arthralgia rather than ongoing viral transmission [13]. Consequently, intervention strategies aimed at reducing mosquito biting rates and shortening the infectious period are expected to decrease $R_0$and drive the system toward the disease-free equilibrium. Similar conclusions have been reported in previous studies on Chikungunya and other mosquito-borne diseases [10,11,14].

The global stability result obtained through the Lyapunov approach extends previous analyses that focused primarily on local stability properties [5,10,12]. By establishing global asymptotic stability of the disease-free equilibrium, this study provides stronger theoretical guarantees that the disease will eventually be eliminated whenever $R_0 < 1$, regardless of the initial population distribution. Such global stability analyses have also proven useful in other epidemiological models for understanding long-term disease behavior and evaluating control strategies [14,23].

Although the proposed model captures the essential transmission mechanisms of Chikungunya, several simplifying assumptions remain. The model assumes homogeneous mixing between hosts and vectors and does not account for demographic heterogeneity. Previous studies have shown that incorporating age structure can provide a more detailed understanding of disease transmission dynamics and improve the effectiveness of control policies [23]. Future work may therefore extend the model by including age-dependent transmission, waning immunity, or optimal control strategies, as commonly considered in modern epidemiological modeling studies [6,14].

5. CONCLUSION

This study proposed and analyzed a modified SICR-SI mathematical model for Chikungunya transmission that incorporates a chronic compartment positioned before the recovery stage. The model was developed to better represent the clinical progression of Chikungunya, where infected individuals may either recover directly or experience a chronic phase before full recovery. The positivity of solutions and the existence of an invariant region were established, ensuring that the model remains biologically meaningful.

Using the Next Generation Matrix approach, the basic reproduction number (R_0) was derived as the threshold parameter governing disease transmission. The analytical results showed that the disease-free equilibrium exists and is locally as well as globally asymptotically stable when $R_0 < 1$. Furthermore, the endemic equilibrium exists and is locally asymptotically stable when $R_0 > 1$. The global stability of the disease-free equilibrium was established through the construction of a Lyapunov function, providing a rigorous characterization of the long-term behavior of the system.

Numerical simulations based on Chikungunya data from West Java Province confirmed the theoretical findings. The simulations demonstrated that when $R_0 < 1$, the infected human, chronic human, and infected mosquito populations gradually approach zero, indicating disease elimination. Conversely, increasing the transmission probabilities between humans and mosquitoes raises the value of R_0 and may drive the system toward endemic persistence.

These findings highlight the importance of reducing vector–host transmission intensity through effective mosquito control measures and public health interventions. The proposed model contributes to the mathematical epidemiology literature by introducing a modified SICR-SI framework and providing a global stability analysis of the disease-free equilibrium. Future research may extend the model by incorporating age structure, spatial heterogeneity, waning immunity, optimal control strategies, or global stability analysis of the endemic equilibrium to obtain a more comprehensive understanding of Chikungunya transmission dynamics.

Author Contributions: Author 1: Conceptualization, methodology, formal analysis, writing—original draft preparation, Author 2: Supervision, funding acquisition, writing—review and editing.

Funding: The authors express their gratitude to Universitas Negeri Yogyakarta for the financial support provided through the Modeling and Optimization Control Research Group (Grant Number: 111/DST/UN34.13/T/PT.01.03/2026).

Conflicts of Interest: All authors declare that they have no conflicts of interest.

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