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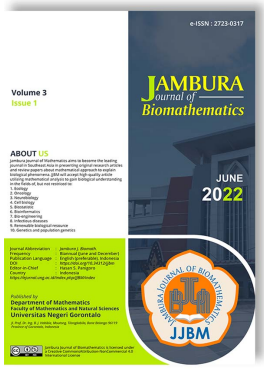
Volume 4, Issue 1, Pages 31–36, June 2023

Received 31 January 2023, Revised 27 February 2023, Accepted 1 March 2023, Published Online 30 April 2023

To Cite this Article : A. Ahkrizal, Jaharuddin, and E. H. Nugrahani,"Dynamics System in the SEIR-SI Model of the Spread of Malaria with Recurrence", *Jambura J. Biomath*, vol. 4, no. 1, pp. 31–36, 2023, *https://doi.org/10.34312/jjbm.v4i1.18754*

JOURNAL INFO • JAMBURA JOURNAL OF BIOMATHEMATICS



Homepage	:	http://ejurnal.ung.ac.id/index.php/JJBM/index
Journal Abbreviation	:	Jambura J. Biomath.
Frequency	:	Biannual (June and December)
Publication Language	:	English (preferable), Indonesia
DOI	:	https://doi.org/10.34312/jjbm
Online ISSN	:	2723-0317
Editor-in-Chief	:	Hasan S. Panigoro
Publisher	:	Department of Mathematics, Universitas Negeri Gorontalo
Country	:	Indonesia
OAI Address	:	http://ejurnal.ung.ac.id/index.php/jjbm/oai
Google Scholar ID	:	XzYgeKQAAAAJ
Email	:	editorial.jjbm@ung.ac.id

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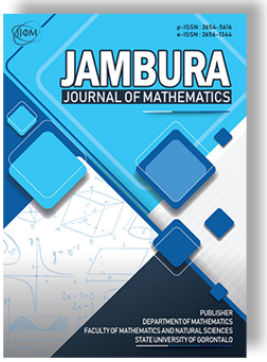

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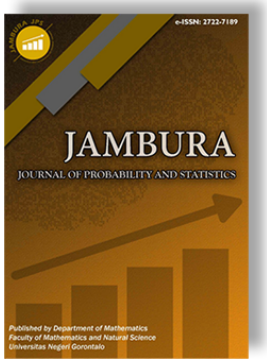

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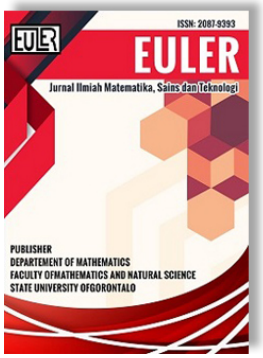

EULER : Jurnal Ilmiah Matematika, Sains, dan Teknologi

Jambura Journal of Biomathematics, Volume 4, Issue 1, Pages 31–36, June 2023 https://doi.org/10.34312/jjbm.v4i1.18754

Research Article

Dynamics System in the SEIR-SI Model of the Spread of Malaria with Recurrence

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ARTICLE HISTORY
Received 31 January 2023
Revised 27 February 2023
Accepted 1 March 2023
Published 30 April 2023

KEYWORDS
mathematical model
malaria
recurrence

ABSTRACT. *Mathematical model is used to describe the dynamics of the spread of malaria in human and mosquito populations. The model used is the SEIR-SI model. This study discusses the stability of the equilibrium point, parameter sensitivity, and numerical simulation of the spread of malaria. The analysis shows that the model has two equilibrium points, namely the disease-free and endemic equilibrium points, each of which is locally asymptotically stable. Numerical simulations show that the occurrence of disease cure in exposed humans causes the rate of malaria spread to decrease. Meanwhile, the presence of disease recurrence causes the spread of malaria to increase.*

1. Introduction

Malaria is an infectious disease caused by Plasmodium parasites. Plasmodium parasites are transmitted through the bite of a female Anopheles mosquito infected with the parasite, blood transfusions, and needle injections that have been used by malaria sufferers causing damage to red blood cells [1]. Plasmodium parasites consist of five species: Plasmodium falciparum, Plasmodium malariae, Plasmodium vivax, Plasmodium ovale, and Plasmodium knowlesi [2]. Among these species, Plasmodium falciparum is a parasite that has a high mortality rate, and Plasmodium vivax is a parasite that has the highest virulence rate [3]. In Plasmodium vivax and Plasmodium ovale, some liver trophozoites do not immediately develop into schizonts, but some become dormant forms called hypnozoites [4]. Hypnozoites are a phase of the parasite's life cycle that can later cause relapse. Plasmodium vivax can relapse, even up to 3-4 years. Plasmodium ovale can relapse for years if the treatment is not done correctly [5]. Symptoms that appear in sufferers of malaria include fever, headache, chills, and retching. In humans who do not have immunity, these symptoms will appear at least seven days after being bitten by an Anopheles mosquito, but these symptoms are very mild and difficult to distinguish from fever in general [6].

In 2020, WHO reported that there were around 241 million malaria cases, resulting in around 627 thousand people dying. It is estimated that 77% of the fatalities are children under the age of 5 years [7]. In Indonesia, the malaria morbidity rate has increased compared to 2019, from 0.93 to 0.94 per 1000 population. Papua contributes the most malaria cases at the provincial level and has the highest malaria morbidity rate compared to other provinces at 63.12 per 1000 population [8]. This condition is exacerbated by the fact that millions of people still do not have access to proper health so that they can prevent and treat malaria. Therefore, public awareness is important to carry out prevention activities that can reduce the rate of transmission of malaria [9].

One of the studies to solve the problem of malaria spread is the study of mathematical modelling and some assumptions in the spread. Mathematical models are used to determine the dynamics of the spread of malaria in previous research, namely research conducted by Ngwa and Shu (2000), which assumed a relationship between the human population and the mosquito population in the spread of malaria [10]. Research conducted by Mojeeb et al. (2017) assumes that cured humans can return to humans susceptible to disease, mosquitoes will never recover, and births occur in infected human subpopulations [11]. Research conducted by Budhwar and Daniel (2017) assumes that the spread of malaria can also occur if there is immigration from infected humans. This occurs when a period of 10 days to 4 weeks from the time of infection to the onset of the actual disease, and humans travel or immigrate within that period [12]. Research conducted by Baihaqi and Adi-Kusumo (2020) assumes that the occurrence of recurrence of malaria in the human population raises new variables because humans who recover from malaria infections cause Plasmodium parasites to remain in the human body [13]. This study analyzes the malaria disease spread model, a modification of the model developed by Budhwar and Daniel (2017). The modification of the model carried out in this study is to assume the parameters of the recovery rate in exposed humans because parasites in the human body experience a dormant condition and the disease recurrence rate in humans who recover because they still have parasites in the body after recovering from active malaria again. Next, stability analysis, sensitivity analysis, and numerical simulation will be carried out on this modified model.

2. Model Formulation

In this section, the model developed by Budhwar and Daniel will be modified. the model modification assumes the fol-

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Homepage : http://ejurnal.ung.ac.id/index.php/JJBM/index / E-ISSN : 2723-0317

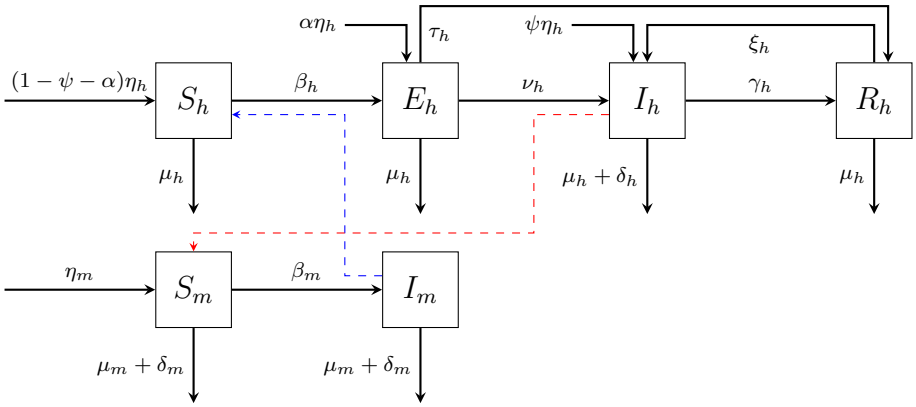

Figure 1. Compartmental Diagram of Malaria Disease Spread

Table 1. Description of Parameters Used in the Model

Parameter	Description	Dimension
η_h	Human birth and migration rates	humans $\times$ time^{-1}
ψ	Fraction of infected human migration	n/a
α	Fraction of human migration exposed	n/a
η_m	Mosquito growth rate	mosquito $\times$ time^{-1}
β_h	Contact rate of disease transmission between susceptible humans and infected mosquitoes	mosquito^{-1} $\times$ time^{-1}
β_m	Contact rate of disease transmission between susceptible mosquitoes and infected humans	humans^{-1} $\times$ time^{-1}
μ_h	The natural mortality rate of human	time^{-1}
μ_m	The natural mortality rate of mosquitoe	time^{-1}
δ_h	The human mortality rate due to disease	time^{-1}
δ_m	Mosquito mortality rate due to control	time^{-1}
ν_h	Transmission rate from exposed human to infected human	time^{-1}
γ_h	The recovery rate in infected humans	time^{-1}
τ_h	The recovery rate in exposed humans	time^{-1}
ξ_h	Disease recurrence rate	time^{-1}

lowing:

1. (E_h) to (R_h), exposed humans can recover from the disease because Plasmodium parasites are in a dormant state in the human body. this occurs as a result of the parasite in which some liver trophozoites do not develop into schizonts, but become dormant forms called hypnozoites [4]. Hypnozoite is a phase of the parasite life cycle that can cause relapse [5].
2. (R_h) to (I_h), recovered humans move to infected humans because Plasmodium parasites which are in a dormant condition reactivate after some time [5].

The populations used in this model are human and mosquito populations with susceptible human (S_h), exposed human (E_h), infected human (I_h), recovered human (R_h), susceptible mosquitoe (S_m) and infected mosquitoe subpopulations (I_m). Parameters added to this model are the rate of recurrence of malaria (ξ_h) and the rate of recovery of disease in exposed humans (τ_h). Based on assumptions, the systematic spread of malaria can be depicted in Figure 1.

Based on Figure 1, a mathematical model is obtained that describes the spread of malaria in the form of a system of nonlinear differential equations as follows:

$$\begin{aligned}
\frac{dS_h}{dt} &= (1-\psi-\alpha)\eta_h - \mu_h S_h - \beta_h S_h I_m,\\
\frac{dE_h}{dt} &= \alpha\eta_h + \beta_h S_h I_m - (\nu_h + \mu_h + \tau_h)E_h,\\
\frac{dI_h}{dt} &= \psi\eta_h + \nu_h E_h + \xi_h R_h - (\mu_h + \delta_h + \gamma_h)I_h,\\
\frac{dR_h}{dt} &= \gamma_h I_h + \tau_h E_h - (\mu_h + \xi_h)R_h,\\
\frac{dS_m}{dt} &= \eta_m - \beta_m S_m I_h - (\mu_m + \delta_m)S_m,\\
\frac{dI_m}{dt} &= \beta_m S_m I_h - (\mu_m + \delta_m)I_m,
\end{aligned} \tag{1}$$

with the total human population $N_h = S_h + E_h + I_h + R_h$ and total mosquito population $N_m = S_m + I_m$. The parameters used in the system of eq. (1) are given in Table 1.

3. Results and Discussion

3.1. Equilibrium Point

The dynamic system of malaria disease spread expressed in the system of eq. (1) has two equilibrium points. They are the disease-free equilibrium point and the endemic equilibrium point. The disease-free equilibrium point is obtained when $S_h = 0$, $S_m = 0$, and the others are not equal to zero, so the obtained is $T^0(S_h, E_h, I_h, R_h, S_m, I_m) = \left(\frac{\eta_h}{\mu_h}, 0, 0, 0, \frac{\eta_m}{\mu_m+\delta_m}, 0\right)$

and the endemic equilibrium point is obtained when all subpopulations are not equal to zero, so the obtained is $T^*(S_h, E_h, I_h, R_h, S_m, I_m) = (S_h^*, E_h^*, I_h^*, R_h^*, S_m^*, I_m^*)$ with

$$S_h^* = \frac{(1-\psi-\alpha)\eta_h}{\beta_h I_m^* + \mu_h}, \qquad E_h^* = \frac{\beta_h S_h I_m^* + \alpha\eta_h}{\mu_h+\nu_h+\tau_h},$$
$$I_h^* = \frac{\nu_h E_h^* + \xi_h R_h^* + \psi\eta_h}{\gamma_h+\delta_h+\mu_h}, \qquad R_h^* = \frac{\gamma_h I_h^* + \tau_h E_h^*}{\mu_h+\xi_h},$$
$$S_m^* = \frac{\eta_m}{\beta_m I_h^* + \delta_m + \mu_m}, \qquad I_m^* = \frac{\beta_m S_m^* I_h^*}{\delta_m+\mu_m}.$$

3.2. Basic Reproduction Number

The basic reproduction number uses the next generation matrix method. The basic reproduction number is the dominant eigenvalue of the FV^{-1} matrix where F is the transmission matrix of new infection in the population and V is the transition matrix of individual movements between subpopulations (E_h), (I_h), (R_h), and (I_m), so the basic reproduction number is obtained as follows:

$$F = \begin{pmatrix} 0 & 0 & 0 & F_{14} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & F_{42} & 0 & 0 \end{pmatrix}, \; V = \begin{pmatrix} V_{11} & 0 & 0 & 0 \\ -V_{21} & V_{22} & -V_{23} & 0 \\ -V_{31} & -V_{32} & V_{33} & 0 \\ 0 & 0 & 0 & V_{44} \end{pmatrix},$$

with

$$F_{14} = \frac{\beta_h\eta_h}{\mu_h}, \quad F_{42} = \frac{\beta_m\eta_m}{\mu_m+\delta_m}, \quad V_{11} = \nu_h + \mu_h + \tau_h,$$
$$V_{21} = \nu_h, \quad V_{22} = \mu_h + \delta_h + \gamma_h, \quad V_{23} = \xi_h, \; V_{31} = \tau_h,$$
$$V_{32} = \gamma_h, \quad V_{33} = \mu_h + \xi_h, \quad V_{44} = \mu_m + \delta_m.$$

The dominant eigenvalue of the matrix FV^{-1}, i.e., the basic reproduction number is:

$$\mathcal{R}_0 = \sqrt{\frac{\beta_h\beta_m\eta_h\eta_m(\xi_h\tau_h + \nu_h(\mu_h+\xi_h))}{\mu_h(\nu_h+\mu_h+\tau_h)(\mu_m+\delta_m)^2((\mu_h+\delta_h+\gamma_h)(\mu_h+\xi_h)-\xi_h\gamma_h)}}. \quad (2)$$

3.3. Disease-Free Equilibrium Point Stability

In this section, to prove the stability of the disease-free equilibrium point, we will use the following theorem:

> **Theorem 1.** *The disease-free equilibrium point T^0 in the system of eq. (1) is locally asymptotic if $R_0 < 1$.*

Proof. The stability properties of the disease-free equilibrium point T^0 can be known by linearizing the system of eq. (1) around T^0. The Jacobian matrix for the disease-free equilibrium point is obtained T^0 is as follows:

$$J_{T^0} = \begin{pmatrix} -P_{11} & 0 & 0 & 0 & 0 & -P_{16} \\ 0 & -P_{22} & 0 & 0 & 0 & P_{26} \\ 0 & P_{32} & -P_{33} & P_{34} & 0 & 0 \\ 0 & P_{42} & P_{43} & -P_{44} & 0 & 0 \\ 0 & 0 & -P_{53} & 0 & -P_{55} & 0 \\ 0 & 0 & P_{63} & 0 & 0 & -P_{66} \end{pmatrix},$$

with

$$P_{11} = \mu_h, \qquad P_{16} = P_{26} = \frac{\beta_h\eta_h}{\mu_h},$$
$$P_{22} = \nu_h + \mu_h + \tau_h, \qquad P_{32} = \nu_h,$$
$$P_{33} = \mu_h + \delta_h + \gamma_h, \qquad P_{34} = \xi_h, \quad (3)$$
$$P_{42} = \tau_h, \qquad P_{43} = \gamma_h,$$
$$P_{44} = \mu_h + \xi_h, \qquad P_{53} = P_{63} = \frac{\beta_m\eta_m}{\mu_m+\delta_m},$$
$$P_{66} = \mu_m + \delta_m.$$

The eigenvalues of the Jacobian matrix J_{T^0} can be obtained by solving $|\lambda I - J_{T^0}| = 0$ and produces negative eigenvalues, and the characteristic equation of the Jacobian matrix is as follows:

$$\lambda_1 = -P_{11}, \; \lambda_2 = -P_{55}, \text{ and} \quad (4)$$

$$\lambda^4 + a_1\lambda^3 + a_2\lambda^2 + a_3\lambda + a_4 = 0 \quad (5)$$

with

$$a_1 = P_{22} + P_{33} + P_{44} + P_{66},$$
$$a_2 = P_{22}P + P_{22}P_{44} + P_{22}P_{66} + P_{33}P_{44} + P_{33}P_{66} + P_{44}P_{66} - P_{34}P_{43},$$
$$a_3 = P_{22}P_{33}P_{44} + P_{22}P_{33}P_{66} + P_{22}P_{44}P_{66} + P_{33}P_{44}P_{66} - P_{22}P_{34}P_{43} - P_{34}P_{43}P_{66} - P_{26}P_{32}P_{63},$$
$$a_4 = P_{22}P_{33}P_{44}P_{66} - P_{26}P_{34}P_{42}P_{63} - P_{26}P_{32}P_{44}P_{63} - P_{22}P_{34}P_{43}P_{66}.$$

Then substitute eq. (3) into eq. (2) to obtain the following:

$$\mathcal{R}_0 = \sqrt{\frac{P_{26}P_{63}(P_{34}P_{42} + P_{32}P_{44})}{P_{22}P_{66}(P_{33}P_{44} - P_{34}P_{43})}}. \quad (6)$$

Because $R_0 < 1$ then

$$P_{22}P_{33}P_{44}P_{66} > P_{26}P_{34}P_{42}P_{63} + P_{26}P_{32}P_{44}P_{63} + P_{22}P_{34}P_{43}P_{66}$$
$$P_{22}P_{33}P_{44}P_{66} > P_{26}P_{34}P_{42}P_{63}$$
$$P_{22}P_{33}P_{66} > P_{26}P_{32}P_{63}$$
$$P_{33}P_{44} > P_{34}P_{43}.$$

So it can be concluded that $a_1, a_2, a_3, a_4, a_1a_2 - a_3$ and $a_1a_2a_3 - a_3^2 - a_1^2a_4$ according to the Routh-Hurwitz criterion [14], the disease-free equilibrium point T^0 is locally asymptotically stable. □

3.4. Endemic Equilibrium Point Stability

In this section, to prove the stability of the disease-free equilibrium point, we will use the following theorem:

> **Theorem 2.** *The endemic equilibrium point T^* in the system of eq. (1) is locally asymptotic if $\mathcal{R}_0 > 1$.*

Proof. To prove Theorem 2, the Castillo-Chaves, and Song Theorem [15] will be used. Suppose $\varphi = \beta_h$ are the bifurcation parameters and $S_h = x_1, E_h = x_2, I_h = x_3, R_h = x_4, S_m = x_5$, and $I_m = x_6$. Equation (3) is substituted to the system of eq. (1)

by supposing as follows:

$$\begin{aligned}
g_1(x_1,x_2,x_3,x_4,x_5,x_6) &= (1-\psi-\alpha)\eta_h - P_{11}x_1 - \varphi x_1 x_6,\\
g_2(x_1,x_2,x_3,x_4,x_5,x_6) &= \alpha\eta_h + \varphi x_1 x_6 - P_{22}x_2,\\
g_3(x_1,x_2,x_3,x_4,x_5,x_6) &= \psi\eta_h + P_{32}x_2 + P_{34}x_4 - P_{33}x_3,\\
g_4(x_1,x_2,x_3,x_4,x_5,x_6) &= P_{43}x_3 + P_{42}x_2 - P_{44}x_4,\\
g_5(x_1,x_2,x_3,x_4,x_5,x_6) &= \eta_m - \beta_m x_5 x_3 - P_{66}x_5,\\
g_6(x_1,x_2,x_3,x_4,x_5,x_6) &= \beta_m x_5 x_3 - P_{66}x_6,
\end{aligned} \tag{7}$$

when the conditions $\mathcal{R}_0 = 1$ and $\varphi = \beta_h$ then five eigenvalues are negative and one eigenvalue is zero. It is $a_4 = 0$. The zero eigenvalue has a right eigenvector $(u_1, u_2, u_3, u_4, u_5, u_6)$ and left eigenvector $(v_1, v_2, v_3, v_4, v_5, v_6)$ as follows:
Suppose $u_4 > 0$ and $v_4 > 0$ then

$$\begin{aligned}
u_1 &= -\frac{P_{16}}{P_{11}}\left(\frac{P_{22}P_{33}P_{44}-P_{22}P_{43}P_{34}}{P_{26}P_{42}P_{33}+P_{26}P_{43}P_{32}}\right)u_4,\\
u_2 &= \frac{P_{33}P_{44}-P_{34}P_{43}}{P_{33}P_{42}+P_{32}P_{43}}u_4,\\
u_3 &= \frac{P_{34}P_{33}P_{42}+P_{34}P_{32}P_{43}+P_{32}P_{33}P_{44}-P_{32}P_{34}P_{43}}{P_{33}^2P_{42}+P_{33}P_{32}P_{43}}u_4,\\
u_5 &= -\frac{P_{53}}{P_{55}}\frac{P_{34}P_{33}P_{42}+P_{34}P_{32}P_{43}+P_{32}P_{33}P_{44}-P_{32}P_{34}P_{43}}{P_{33}^2P_{42}+P_{33}P_{32}P_{43}}u_4,\\
u_6 &= \frac{P_{22}P_{33}P_{44}-P_{22}P_{34}P_{43}}{P_{26}P_{33}P_{42}+P_{26}P_{32}P_{43}}u_4,\\
v_1 &= v_5 = 0,\ v_2 = \frac{P_{32}P_{44}+P_{34}P_{42}}{P_{22}P_{34}}v_4,\\
v_3 &= \frac{P_{44}}{P_{34}}v_4,\ v_6 = \frac{P_{26}P_{32}P_{44}+P_{26}P_{34}P_{42}}{P_{22}P_{34}P_{66}}v_4.
\end{aligned}$$

It is obtained that $u_1 < 0, u_2 > 0, u_3 > 0, u_4 > 0, u_5 < 0, v_1 = v_5 = 0, v_2 > 0, v_3 > 0, v_4 > 0$, and $v_6 > 0$. Using the Castillo-Chavez and Song Theorem is defined as follows:

$$\sum_{k,i,j=1}^{6} v_k u_i u_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(T^0,\varphi); \sum_{k,i=1}^{6} v_k u_i \frac{\partial^2 f_k}{\partial x_i \partial \varphi}(T^0,\varphi). \tag{8}$$

Based on the system of eq. (7), the following is obtained:

$$\begin{aligned}
\frac{\partial^2 f_1}{\partial x_1 \partial x_6}(T^0,\varphi) &= \frac{\partial^2 f_1}{\partial x_6 \partial x_1}(T^0,\varphi^*) = -\varphi,\\
\frac{\partial^2 f_2}{\partial x_1 \partial x_6}(T^0,\varphi) &= \frac{\partial^2 f_2}{\partial x_6 \partial x_1}(T^0,\varphi^*) = \varphi,\\
\frac{\partial^2 f_5}{\partial x_3 \partial x_5}(T^0,\varphi) &= \frac{\partial^2 f_5}{\partial x_5 \partial x_3}(T^0,\varphi) = -\beta_m,\\
\frac{\partial^2 f_6}{\partial x_3 \partial x_5}(T^0,\varphi) &= \frac{\partial^2 f_6}{\partial x_5 \partial x_3}(T^0,\varphi) = \beta_m,\\
\frac{\partial^2 f_2}{\partial x_6 \partial \varphi}(T^0,\varphi) &= \frac{\eta_h}{P_{11}}.
\end{aligned}$$

Based on eq. (8), the following is obtained:

$$a = 2v_2u_1u_6\varphi + 2v_6u_3u_5\beta_m; b = v_2u_6\frac{\eta_h}{P_{11}} \tag{9}$$

because $u_1, u_5 < 0, P_{33}P_{44} - P_{34}P_{43} > 0$, all parameters are positive, then the values of $a < 0$ and $b > 0$. This result is consistent with the criteria of case 4 in the Castillo-Chaves and Song Theorems. Consequently, when φ changes from $\varphi < \varphi(\mathcal{R}_0 < 1)$ to $\varphi > \varphi(\mathcal{R}_0 > 1)$ then the endemic equilibrium point T^* changes from negative to positive and is locally asymptotically stable. So, it is proven that if $\mathcal{R}_0 > 1$ then the endemic equilibrium point T^* is locally asymptotically stable. □

3.5. Sensitivity Analysis

Sensitivity analysis is carried out on the endemic equilibrium point, which aims to determine the parameters that affect the change in value. $\mathcal{R}_0$. The dynamics of the spread of malaria are influenced by the parameter values in Table 2 below.

Table 2. Parameter Values Used in the Model

Parameter	Value	Source
η_h	154	[8]
ψ	0.00032	Assumed
α	0.00065	Assumed
η_m	3500	[11]
β_h	0.000002024	[11]
β_m	0.00003214	[11]
μ_h	0.0416	[8]
μ_m	0.05	[11]
δ_h	0.000032	[8]
δ_m	0.01	[16]
ν_h	0.05	[11]
γ_h	0.035	[11]
τ_h	0.055	[17]
ξ_h	0.01	[18]

Sensitivity analysis uses the sensitivity index value with the following formula:

$$\Upsilon_p^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0} \tag{10}$$

with p is parameter in the model. Sensitivity analysis is carried out by inputting the values of the parameters to obtain the sensitivity index value of each parameter using eq. (10). The parameters influence the dynamics of the spread of malaria in Table 2, and then the sensitivity index value is obtained in Table 3 below.

Table 3. Sensitivity Index Values of the Model

Parameter	Sensitivity Index
η_h	0.5
ψ	0
α	0
η_m	0.5
β_h	0.5
β_m	0.5
μ_h	-1.19
μ_m	-0.83
δ_h	-0.00036
δ_m	-0.17
ν_h	0.24
γ_h	-0.032
τ_h	-0.099
ξ_h	0.077

Table 3 shows that the parameter of recovery rate in exposed humans (τ_h) has a negative sensitivity index, meaning that the parameter is inversely proportional to changes in the value of $\mathcal{R}_0$that is, if the recovery rate in exposed humans is (τ_h) increases, there will be a decrease in the value of $\mathcal{R}_0$ and vice versa. While the recurrence rate of (ξ_h) has a positive sensitivity index, meaning that the parameter is directly proportional to the change in the value of $\mathcal{R}_0$ if the recurrence rate of (ξ_h) is increased, there will be an increase in the value of $\mathcal{R}_0$ and vice versa. Then a numerical simulation will be carried out to show the dynamics of the human population on the spread of malaria due to recurrence which is affected by changes in the values of the parameters (τ_h) and (ξ_h).

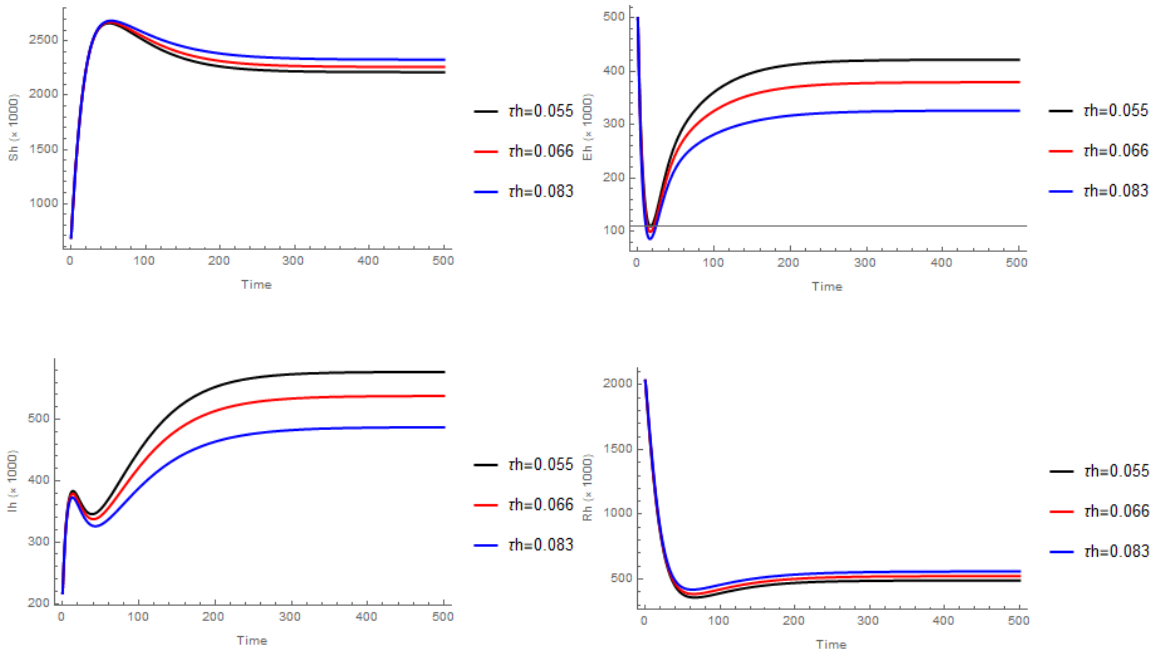

Figure 2. Dynamics of Human Population to Changes in τ_h

3.6. *Numerical Simulation*

Simulations were conducted on changes in the parameter values of the recovery rate in exposed humans (τ_h) and recurrence rate (ξ_h) with the initial value in each subpopulation is $S_h(0) = 686, E_h(0) = 500, I_h(0) = 217, R_h(0) = 2033, S_m(0) = 3000, I_m(0) = 2000$.

3.6. *Human Population Dynamics with Parameter Changes τ_h*

Sensitivity index for the parameter of the recovery rate in exposed humans (τ_h) is negative, meaning that there is a decrease in the value of the basic reproduction number $\mathcal{R}_0$ if this parameter is increased, and vice versa. Changes in the value of $\mathcal{R}_0$ and the human population can be seen in the simulation results in Table 4 below.

Table 4. Changes in parameter values τ_h against $\mathcal{R}_0$

τ_h	$\mathcal{R}_0$	Fixed Point $(S_h^*, E_h^*, I_h^*, R_h^*)$
0.055	1.47622	(2214,422,578,489)
0.066	1.44857	(2262,380,539,523)
0.0833	1.41189	(2329,327,488,559)

Based on Table 4, there is a change $\mathcal{R}_0$ as the value of τ_h. This will also be shown in Figure 2 for each simulation subpopulation. Figure 2 shows that changes in the recovery rate of exposed humans (τ_h) can affect the size of the human population. If this parameter increases, the number of susceptible and recovered human subpopulations also increases. Meanwhile, the number of exposed and infected human subpopulations will decrease. This suggests that recovery from disease caused by Plasmodium parasites dormant in the human body can reduce the infected subpopulation. However, caution is still important if the parasite reactivates and attacks the human body.

3.6. *Population Dynamics with Parameter Changes ξ_h*

Sensitivity index for the parameter of the recurrence rate (ξ_h) is positive, meaning that there is an increase in the value of the basic reproduction number $\mathcal{R}_0$ when increased, and vice versa. This can be seen in the simulation results in Table 5 below. Based on Table 5, there is a change $\mathcal{R}_0$ as the value of ξ_h. This

Table 5. Changes in Parameter Values ξ_h against $\mathcal{R}_0$

ξ_h	$\mathcal{R}_0$	Fixed Point $(S_h^*, E_h^*, I_h^*, R_h^*)$
0.0	1.33015	(2500,341,379,483)
0.0055	1.41944	(2315,394,498,496)
0.01	1.47622	(2214,422,578,489)

will also be shown in Figure 3 for each simulation subpopulation. Figure 3 shows that changes in the recurrence rate can affect the size of the human population. If this parameter increases, the number of exposed and infected human subpopulations will also increase. Meanwhile, the number of susceptible human subpopulations will decrease. However, in recovered humans, there are different population changes. It can be seen in the endemic fixed point in Table 5. This shows that the spread of malaria will increase if there is a recurrence of the disease at a certain time, so one of the efforts made is to immediately treat recovered human but still have parasites in the body to emphasize the recurrence of malaria.

4. Conclusion

In this study, the SEIR-SI model is used to show the dynamics of the spread of malaria in human and mosquito populations. In this model, only the dynamics of the human population are studied because the human population considers the existence of disease recovery from exposed humans and disease recurrence due to dormant parasites that become active again at a certain time. The analysis shows that this model has two equilibrium points, namely the disease-free equilibrium point and the endemic equilibrium point. The disease-free equilibrium point is locally asymptotically stable when the basic reproduction number is less than one, meaning the disease will run out within a certain time. The endemic equilibrium point is locally asymptot-

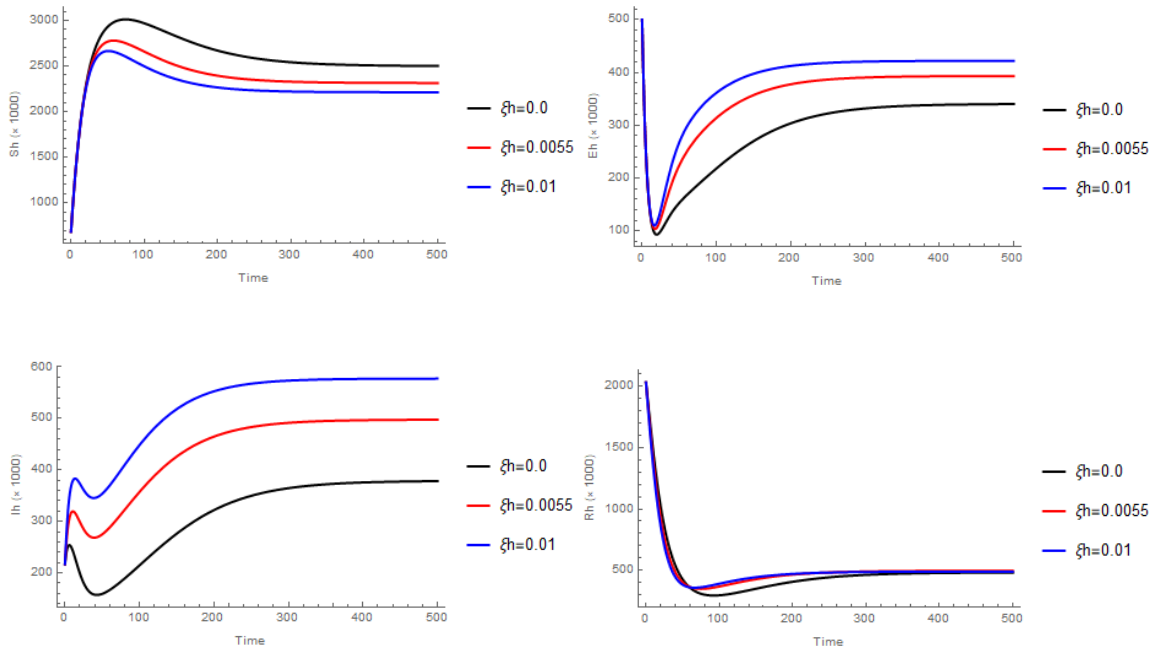

Figure 3. Dynamics of Human Population to Changes in ξ_h

ically stable when the basic reproduction number is more than one, meaning that malaria will persist as the number of infected humans increases. The results of further analysis show that increasing disease recovery from exposed humans can reduce the spread of malaria and increasing malaria recurrence can increase the spread of malaria. Therefore, efforts that can be made to reduce the spread of malaria are to reduce the rate of malaria recurrence.

Author Contributions. **Afdhal Ahkrizal:** Conceptualization, methodology, software, validation, formal analysis, investigation, resources, data curation, writing—original draft preparation, writing—review and editing, visualization, project administration, and funding acquisition. **Jaharuddin:** validation, formal analysis, and supervision. **Endar H. Nugrahani:** validation, formal analysis, supervision. All authors have read and agreed to the published version of the manuscript.

Acknowledgement. The authors are grateful to the handling editor and reviewers for their helpful comments and suggestions that have improved the quality of the manuscript.

Funding. This research received no external funding.

Conflict of interest. The authors declare no conflict of interest.

Data availability. Not applicable.

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