

$SEIR_1R_2$ Mathematical Model for *Plasmodium falciparum* Recrudescence in Indonesia

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Abstract. Malaria remains one of the most challenging infectious diseases to eradicate, with a high prevalence in tropical regions, particularly in Indonesia, where *Plasmodium falciparum* accounts for a substantial proportion of reported cases. This study develops a compartmental mathematical model to analyze malaria transmission dynamics in human populations by incorporating both recrudescence and reinfection processes. The proposed model extends the classical SEIR framework by introducing two additional compartments: R_1 , representing individuals at risk of recurrence due to incomplete parasite clearance, and R_2 , representing individuals who have fully recovered but remain susceptible to reinfection. The model is formulated as a system of five nonlinear ordinary differential equations. The basic reproduction number, R_0 , is derived using the Next Generation Matrix method, and the stability of both the disease-free and endemic equilibrium points is examined through local stability analysis and the Routh–Hurwitz criteria. Numerical simulations are conducted to illustrate the dynamic behavior of the system under various parameter settings. The results highlight the influence of treatment, recovery, and transmission parameters on disease persistence or elimination, providing a mathematical foundation for further epidemiological analysis and the development of control strategies.

Key words and Phrases: Malaria, Mathematical model, Recrudescence, *Plasmodium falciparum*, Equilibrium analysis.

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1 INTRODUCTION

Malaria is a vector-borne infectious disease transmitted to humans through the bite of female *Anopheles* mosquitoes carrying protozoan parasites of the genus *Plasmodium*. Transmission occurs during the mosquito’s blood-feeding process, which is required for egg development. Malaria remains a major global public health concern, particularly in endemic regions of Asia, sub-Saharan Africa, and Central and South America [1]. Despite decades of control and elimination efforts, the annual number of malaria episodes continues to exceed 200 million worldwide [1]. According to the most recent World Malaria Report, approximately 263 million malaria cases and approximately 597,000 malaria-related deaths were reported in 2023, representing an increase of roughly 11 million cases compared to 2022, while mortality has remained at an unacceptably high plateau [1]. This persistent burden underscores the need for refined strategies that explicitly account for complex within-host and population-level processes sustaining transmission.

Although malaria is both preventable and treatable when appropriate diagnostic and therapeutic measures are implemented, suboptimal case management can lead to disease recurrence. Clinically, malaria recurrence arises through three primary mechanisms: recrudescence, relapse, and reinfection. Recrudescence refers to the reappearance of blood-stage parasites and clinical symptoms due to incomplete clearance of a previous infection following treatment, typically reflecting antimalarial treatment failure or an inadequate immune response [2, 3]. In contrast, reinfection corresponds to a new infectious mosquito bite in an individual who has previously cleared the parasite. Relapse results from the reactivation of dormant hypnozoites in the liver and is largely restricted to *P. vivax* and *P. ovale*, which may remain dormant for months before initiating a new blood-stage infection [4]. From an elimination perspective, recrudescence is particularly problematic because it originates in treated individuals who may be incorrectly considered cured, thereby constituting a “hidden” infectious reservoir.

Five *Plasmodium* species are known to infect humans: *P. falciparum*, *P. vivax*, *P. ovale*, *P. malariae*, and *P. knowlesi*. These species differ in both their biological characteristics and clinical manifestations. The presence of hypnozoite stages in *P. vivax* and *P. ovale* enables relapse, whereas *P. falciparum* does not form hypnozoites but may exhibit recrudescence following apparently successful treatment. Among these species, *P. falciparum* is recognized as the most virulent and is responsible for the majority of severe malaria cases and deaths, accounting for approximately 90% of malaria-related fatalities worldwide [5].

Indonesia remains malaria endemic, with 534,936 reported cases in 2024 [6]. National surveillance data indicate that *P. falciparum* accounted for approximately 55.10% of all laboratory-confirmed malaria infections in that year, underscoring its dominance within the Indonesian parasite landscape [6]. In high-burden provinces, such as Papua and West Papua, the interplay of intense transmission and persistent treatment challenges renders recrudescence *P. falciparum* infections a realistic and programmatically significant concern. Within this context, a rigorous quantitative understanding of *P. falciparum* transmission dynamics, including mechanisms that

permit treated individuals to re-enter the infectious class, is essential for the design of effective and sustainable elimination strategies.

Mathematical modelling provides a rigorous framework for investigating malaria transmission mechanisms and for systematically evaluating control interventions. A substantial body of literature has developed SEIR-type compartmental models, in which the human population is divided into susceptible, exposed, infectious, and recovered classes. Numerous extensions of the classical SEIR structure have been proposed to capture malaria-specific dynamics, including models by Ngwa and Shu [7], Khan et al. [8], and Osman et al. [9]. Huo and Qiu [10] examined the impact of recurrent malaria on system stability, while Baihaqi and Adi-Kusumo [11] explicitly incorporated recurrence by allowing recovered individuals to retain residual parasites. More recently, Ghosh et al. [4] distinguished relapse and reinfection as separate recurrence pathways, thereby providing a more nuanced representation of clinical outcomes.

In parallel, substantial methodological advances have been made in epidemic modelling more broadly, including the development of stochastic compartmental frameworks that incorporate environmental noise, media effects, and vaccination dynamics. Recent studies on hepatitis B virus (HBV) dynamics, for instance, have incorporated Lévy noise and media coverage to analyse extinction and persistence criteria [12], examined HBV transmission under Lévy-driven perturbations and vaccination strategies [13], and applied stochastic optimal control theory to design cost-effective vaccination policies [14]. Collectively, these contributions demonstrate how modern dynamical systems techniques can sharpen insights into threshold behaviour and long-term disease outcomes, thereby motivating the adoption of similarly structured modelling approaches for malaria that explicitly account for species-specific features such as recrudescence.

Motivated by the Indonesian epidemiological context and the clinical significance of *P. falciparum* recrudescence, this study focuses exclusively on *P. falciparum* malaria. We formulate a modified SEIR-type model in which the recovery class is subdivided into two distinct compartments, denoted by R_1 and R_2 , to capture heterogeneous post-treatment outcomes. The compartment R_1 represents individuals who have received treatment and exhibit clinical improvement but continue to harbour residual *Plasmodium* parasites, thereby remaining at risk of recrudescence. In contrast, R_2 represents individuals who have achieved complete parasitological clearance but remain susceptible to future infection through new mosquito bites. In the proposed framework, all individuals enter the population as susceptible at birth, the human population is assumed to be demographically closed (i.e. no migration), and loss of protection is represented implicitly through reinfection of the R_2 class rather than through an explicit return to a separate susceptible compartment.

The remainder of this manuscript is organised as follows. First, we introduce the SEIR₁R₂ model, detailing its compartmental structure, parameter assumptions, and the derivation of the basic reproduction number. We then conduct a rigorous qualitative analysis of the system, including the existence and local stability of the

disease-free and endemic equilibrium points. Next, we perform sensitivity analysis and numerical simulations, using parameter values informed by Indonesian malaria data, to investigate how recrudescence and reinfection jointly influence the long-term dynamics of *P. falciparum*. Finally, we summarise the main findings and discuss their implications for malaria control and elimination programmes in Indonesia and other endemic settings.

2 THE FORMULATION OF MODEL

2.1 The Compartmental Model for Malaria Transmission.

Earlier malaria models generally did not account explicitly for the mechanism of recrudescence. While Baihaqi et al. [15] incorporated relapse dynamics, and Tasman et al. [16] combined relapse and recrudescence within a single extended framework, the present study adopts a more targeted perspective. Specifically, we focus exclusively on the two mechanisms most relevant to *Plasmodium falciparum* transmission, namely recrudescence and reinfection, thereby aligning the model structure with the biological characteristics of the species under consideration.

Within the proposed compartmental framework, the susceptible class, denoted by $S(t)$, represents individuals who have never been infected with malaria and are therefore at risk of acquiring the disease through the bite of an infected mosquito carrying *Plasmodium falciparum*. Susceptible individuals move to the exposed compartment $E(t)$ following successful transmission of the parasite. This transition is driven by effective contact between susceptible and infectious individuals and is regulated by the transmission parameter α . In addition to disease-related transitions, individuals in the susceptible compartment are subject to natural mortality at rate μ .

The exposed compartment, denoted by $E(t)$, comprises individuals who have been infected through the bite of an infectious mosquito but have not yet developed clinical symptoms. This stage corresponds to the intrinsic incubation period of the malaria parasite within the human host. Upon completion of this incubation period, individuals transition to the infectious compartment, $I(t)$, at a rate β . In addition, individuals in the exposed compartment are subject to natural mortality at a constant rate μ .

The infected compartment, denoted by $I(t)$, comprises individuals who have developed clinical symptoms of malaria. These individuals arise from the exposed compartment $E(t)$ after the incubation period and may recover following appropriate medical treatment. Upon recovery, individuals transition to one of two distinct compartments: the recrudescence-risk class $R_1(t)$ at rate γ , representing individuals who remain at risk of recrudescence due to incomplete parasite clearance, or the fully recovered class $R_2(t)$ at rate δ , representing individuals who have achieved parasitological cure but remain susceptible to reinfection. Individuals in the infected compartment are subject to natural mortality at rate μ as well as malaria-induced mortality at rate ω . In addition, individuals in the recrudescence compartment

$R_1(t)$ may experience a return to the infected compartment due to parasite persistence, a process governed by the recrudescence rate θ .

The recrudescence compartment, $R_1(t)$, comprises individuals who have received antimalarial treatment and appear clinically recovered but continue to harbour residual *Plasmodium* parasites within their red blood cells. As a result, these individuals remain at risk of experiencing a renewed episode of malaria and may transition back to the infected compartment at a rate θ , or exit the population through natural mortality at a rate μ . This compartmental structure reflects the concept of recrudescence malaria, defined as a repeated malaria episode arising from the survival of blood-stage parasites, typically occurring within the first four weeks after the initial illness when treatment fails to achieve complete parasite clearance [17]. Documented drivers of recrudescence include incomplete adherence to otherwise appropriate antimalarial regimens, the inappropriate use of oral antimalarials in severe disease—particularly in cases of hyperparasitaemia, where at least 5% of red blood cells are infected—and the emergence of antimalarial drug resistance [17]. By explicitly incorporating the R_1 compartment, the model captures how incomplete parasite clearance can sustain transmission and contribute to the long-term persistence of malaria within the population.

The reinfection compartment, $R_2(t)$, comprises individuals who have achieved full parasitological recovery and no longer harbour *Plasmodium* parasites, yet remain susceptible to subsequent infection. Individuals in this class may transition back to the exposed compartment $E(t)$ following a new infectious mosquito bite, at a rate governed by the transmission parameter α . Natural mortality also acts on this compartment at a constant rate μ . This reinfection pathway reflects the persistent exposure risk in endemic settings and highlights a fundamental challenge for malaria elimination, whereby individuals may experience multiple infection episodes despite successful prior treatment.

2.2 Model Formulation.

To construct a mathematical framework that captures the time-dependent behaviour of malaria transmission dynamics, including both recrudescence (the recurrence of infection without new exposure) and reinfection (recovery followed by a new infection from a mosquito carrying *Plasmodium*), the human population is partitioned into five mutually exclusive compartments, each representing a distinct epidemiological stage in the progression of the disease.

Specifically, susceptible individuals, denoted by $S(t)$, represent those who have not yet been exposed to malaria. Exposed individuals, denoted by $E(t)$, consist of individuals who have been bitten by mosquitoes infected with *Plasmodium falciparum* but have not yet developed clinical symptoms. Infected individuals, represented by $I(t)$, correspond to those experiencing symptomatic malaria. Individuals who have recovered from infection but remain at risk of recrudescence are classified into the compartment $R_1(t)$, while those who have fully recovered yet remain susceptible to reinfection are assigned to the compartment $R_2(t)$. At any given time t , the total human population, denoted by $N(t)$, is defined as the sum of all epidemiological compartments included in the model.

$$N(t) = S(t) + E(t) + I(t) + R_1(t) + R_2(t). \quad (1)$$

The temporal evolution of the susceptible population is governed by the balance between recruitment, infection, and natural mortality. New individuals enter the susceptible class at a constant rate Λ , while natural deaths occur at a per capita rate $\mu S(t)$. Susceptible individuals become exposed to malaria through effective contact with infectious individuals via mosquito bites, which is modeled by a standard incidence term $\frac{\alpha S(t)I(t)}{N(t)}$, where α denotes the transmission rate. Consequently, the dynamics of the susceptible compartment are described by

$$\frac{dS}{dt} = \Lambda - \frac{\alpha SI}{N} - \mu S. \quad (2)$$

The dynamics of the exposed population are governed by two sources of infection: transmission from susceptible individuals through contact with infectious individuals mediated by mosquito bites, represented by $\frac{\alpha SI}{N}$, and reinfection of individuals in the recovered class R_2 , represented by $\frac{\alpha R_2 I}{N}$. The exposed population decreases due to natural mortality at rate μ and progression to the infectious class at rate β . Accordingly, the evolution of the exposed compartment is described by

$$\frac{dE}{dt} = \frac{\alpha SI}{N} + \frac{\alpha R_2 I}{N} - \mu E - \beta E. \quad (3)$$

The evolution of the infected population, represented by $\frac{dI}{dt}$, is governed by the progression of individuals from the exposed compartment into the infectious class at a rate βE , as well as by recrudescence arising from partially recovered individuals in the R_1 compartment at a rate θR_1 . These inflows are counterbalanced by several outflows, including natural mortality at a rate μI , malaria-induced mortality at a rate ωI , transition to the recrudescence class at a rate γI , and full recovery into the reinfection-susceptible class at a rate δI . The equation describing these dynamics is

$$\frac{dI}{dt} = \beta E + \theta R_1 - \gamma I - \mu I - \omega I - \delta I. \quad (4)$$

The dynamics of the recrudescence compartment are governed by the balance between inflow from the infectious class and subsequent outflows due to recrudescence and natural mortality. Specifically, individuals enter the recrudescence class $R_1(t)$ from the infected compartment at a rate γI , reflecting partial recovery with residual parasitaemia. The size of this compartment decreases through natural death at a rate μR_1 and through recrudescence, whereby individuals return to the infectious class at a rate θR_1 . Accordingly, the temporal evolution of the recrudescence population is described by

$$\frac{dR_1}{dt} = \gamma I - \mu R_1 - \theta R_1. \quad (5)$$

The rate at which the number of individuals in the recovered class changes over time, $\frac{dR_2}{dt}$, is influenced by the recovery of infected individuals at rate δI , reduced by the natural death rate μR_2 and the reinfection of recovered individuals, given by $\frac{\alpha R_2 I}{N}$. This is expressed as

$$\frac{dR_2}{dt} = \delta I - \mu R_2 - \frac{\alpha R_2 I}{N}. \quad (6)$$

Thus, the mathematical model describing malaria transmission, incorporating both recrudescence and reinfection, is formulated using the system of differential equations presented below.

$$\begin{aligned} \frac{dS}{dt} &= \Lambda - \frac{\alpha SI}{N} - \mu S, \\ \frac{dE}{dt} &= \frac{\alpha SI}{N} + \frac{\alpha R_2 I}{N} - \mu E - \beta E, \\ \frac{dI}{dt} &= \beta E + \theta R_1 - \gamma I - \mu I - \omega I - \delta I, \\ \frac{dR_1}{dt} &= \gamma I - \mu R_1 - \theta R_1, \\ \frac{dR_2}{dt} &= \delta I - \mu R_2 - \frac{\alpha R_2 I}{N}. \end{aligned} \quad (7)$$

The following diagram illustrates the transmission dynamics of malaria, incorporating the mechanisms of both recrudescence and reinfection.

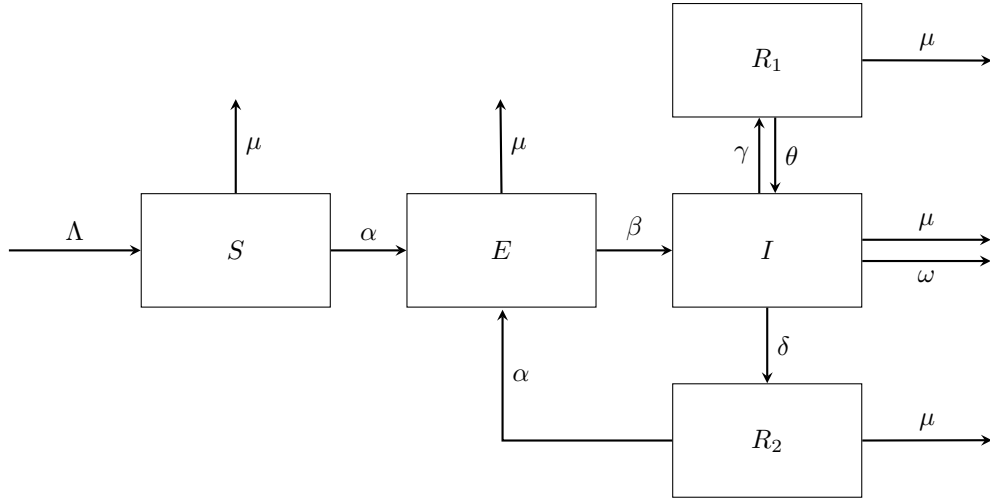


FIGURE 1. Diagram of the malaria transmission process

The descriptions of variables and parameters based on Figure 1 are presented in Table 1 and Table 2 .

TABLE 1. The variables description of the model

No	Variables	Description
1	S	Represents the number of individuals susceptible to malaria infection
2	E	Denotes the number of individuals who have been exposed to malaria through the bite of a mosquito carrying <i>Plasmodium falciparum</i> , but who have not yet developed clinical symptoms
3	I	Refers to the number of individuals actively infected with malaria caused by <i>Plasmodium falciparum</i>
4	R_1	Indicates the number of individuals who have recovered but are undergoing recrudescence
5	R_2	Represents the number of recovered individuals who have been reinfected through another bite by a mosquito infected with <i>Plasmodium falciparum</i>

TABLE 2. Description of the model parameters

No	Parameters	Description
1	Λ	Represents the constant rate at which new individuals are recruited into the human population
2	α	Transmission coefficient describing the rate at which humans become exposed to malaria following a bite from a mosquito infected with <i>Plasmodium falciparum</i>
3	β	Transition rate from the exposed compartment (E) to the infectious compartment (I)
4	γ	Recovery rate from the infectious compartment (I) to the recrudescence compartment (R_1), accounting for incomplete parasite clearance
5	θ	Rate at which individuals in the recrudescence compartment (R_1) revert to the infectious state (I) due to malaria recurrence
6	ω	Malaria-induced mortality rate resulting from infection with <i>Plasmodium falciparum</i>
7	δ	Progression rate from the infectious state (I) to the fully recovered compartment (R_2)
8	μ	Natural mortality rate within the human population

2.3 Invariant Region.

According to Equation (1), the total human population at time t , denoted by $N(t)$, can be formulated as follows.

$$N(t) = S(t) + E(t) + I(t) + R_1(t) + R_2(t).$$

The rate of change of $N(t)$ with respect to time t can be written as

$$\frac{dN}{dt} = \frac{dS}{dt} + \frac{dE}{dt} + \frac{dI}{dt} + \frac{dR_1}{dt} + \frac{dR_2}{dt}.$$

From this, we obtain the relation

$$\frac{dN}{dt} = \Lambda - \mu N - \omega I. \quad (8)$$

To derive the solution for $N(t)$, the term ωI is removed, leading to the inequality

$$\frac{dN}{dt} \leq \Lambda - \mu N. \quad (9)$$

Consequently, the analytical expression for $N(t)$ satisfies

$$N(t) \leq \frac{\Lambda}{\mu} + ce^{-\mu t}. \quad (10)$$

Taking the limit as $t \rightarrow \infty$, we arrive at

$$\lim_{t \rightarrow \infty} N(t) \leq \frac{\Lambda}{\mu}. \quad (11)$$

Hence, the feasible region of System 7 is characterized as follows.

$$D = \{(S, E, I, R_1, R_2) \in \mathbb{R}^5 : S + E + I + R_1 + R_2 \leq \frac{\Lambda}{\mu}, S, E, I, R_1, R_2 \geq 0\}. \quad (12)$$

3 ANALYTICAL ANALYSIS

3.1 The Equilibrium Point.

In this subsection, we determine the equilibrium point of the proposed model. An equilibrium point corresponds to a steady-state condition in which all state variables remain constant over time, indicating that the population dynamics have reached balance. To obtain the equilibrium point, we set the right-hand sides of the governing system of differential equations, specifically Equation (7), equal to zero. Solving the resulting algebraic system yields the steady-state values of each compartment, which are presented and analysed in the subsequent equations.

$$\Lambda - \frac{\alpha SI}{N} - \mu S = 0 \quad (13)$$

$$\frac{\alpha SI}{N} + \frac{\alpha R_2 I}{N} - (\mu + \beta)E = 0 \quad (14)$$

$$\beta E + \theta R_1 - (\gamma + \mu + \omega + \delta)I = 0 \quad (15)$$

$$\gamma I - (\mu + \theta)R_1 = 0 \quad (16)$$

$$\delta I - \mu R_2 - \frac{\alpha R_2 I}{N} = 0 \quad (17)$$

From Equations (13), (14), (15), (16), and (17) above, we obtain that

$$S^* = \frac{\Lambda N}{\alpha I^* + \mu N} \quad (18)$$

$$E^* = \frac{(\gamma + \mu + \omega + \delta)(\mu + \theta) - \theta\gamma}{\beta} I^* \quad (19)$$

$$R_1^* = \frac{\gamma I^*}{\mu + \theta} \quad (20)$$

$$R_2^* = \frac{\delta I^* N}{\mu N + \alpha I^*} \quad (21)$$

Subsequently, by substituting Equations (18), (19), and (21) into equation (14), we obtain

$$\alpha \left(\frac{\Lambda N}{\alpha I + \mu N} \right) \frac{I}{N} + \alpha \left(\frac{\delta I N}{\mu N + \alpha I} \right) \frac{I}{N} - (\mu + \beta) \left[k \frac{(\mu + \theta) - \theta\gamma}{\beta} \right] I = 0; k = \gamma + \mu + \omega + \delta.$$

Then, by simplifying it, we obtain

$$\frac{\alpha \Lambda I + \alpha \delta I^2}{\alpha I + \mu N} - (\mu + \beta) \left[k \frac{(\mu + \theta) - \theta\gamma}{\beta} \right] I = 0.$$

By letting $A = (\mu + \beta) \left[k \frac{(\mu + \theta) - \theta\gamma}{\beta} \right]$, we obtain the following equation.

$$(\alpha \delta - A \alpha) I^2 + (\alpha \Lambda - A \mu N) I = 0. \quad (22)$$

By applying the Quadratic Formula to Equation (22) to determine its solutions, we obtain

$$I_0 = 0 \quad \text{or} \quad I_1 = \frac{\alpha \Lambda - A \mu N}{A \alpha - \alpha \delta}.$$

As a result of the calculations conducted above, two equilibrium points have been identified: the disease-free equilibrium, denoted as TE_0 , and the endemic equilibrium, denoted as TE_1 .

The first equilibrium point, TE_0 , represents a scenario where the disease is completely eradicated from the population. It is given by

$$TE_0 = (S^*, E^*, I^*, R_1^*, R_2^*) = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \right).$$

The second equilibrium point, TE_1 , represents a state where the disease persists in the population at a stable level. It is defined as

$$TE_1 = (S^*, E^*, I^*, R_1^*, R_2^*)$$

under the following conditions.

$$\begin{aligned}
S^* &= \frac{\Lambda N}{\alpha I^* + \mu N}, \\
E^* &= \frac{(\gamma + \mu + \omega + \delta)(\mu + \theta) - \theta\gamma}{\beta} I^*, \\
I^* &= \frac{\alpha\Lambda - \frac{\mu N}{\beta}(\mu + \beta)((\gamma + \mu + \omega + \delta)(\mu + \theta) - \theta\gamma)}{\frac{\alpha}{\beta}(\mu + \beta)((\gamma + \mu + \omega + \delta)(\mu + \theta) - \theta\gamma) - \alpha\delta}, \\
R_1^* &= \frac{\gamma I^*}{\mu + \theta}, \\
R_2^* &= \frac{\delta I^* N}{\mu N + \alpha I^*}.
\end{aligned}$$

3.2 Disease-Free Equilibrium.

3.2.1 Basic Reproduction Number (R_0).

The disease-free equilibrium of the system is denoted by TE_0 and is expressed as

$$TE_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0 \right).$$

The basic reproduction number R_0 associated with System 7 is computed using the Next Generation Matrix (NGM) approach, following the methodology introduced by Driessche and Watmough [18].

Letting $X = (E, I, R_1, R_2, S)^T$, System 7 can be reformulated in the compact form

$$x' = \mathcal{F}(x) - \mathcal{V}(x)$$

with the matrices defined as

$$\mathcal{F}(x) = \begin{bmatrix} \frac{\alpha SI}{N} + \frac{\alpha R_2 I}{N} \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix}, \quad \mathcal{V}(x) = \begin{bmatrix} (\mu + \beta)E \\ -\beta E - \theta R_1 + (\gamma + \mu + \omega + \delta)I \\ -\gamma I + (\mu + \theta)R_1 \\ -\delta I + \mu R_2 + \frac{\alpha R_2 I}{N} \\ -\Lambda + \frac{\alpha SI}{N} + \mu S \end{bmatrix}.$$

The Jacobian matrices of $\mathcal{F}(x)$ and $\mathcal{V}(x)$ evaluated at the disease-free equilibrium TE_0 are given by

$$D\mathcal{F}(TE_0) = \begin{bmatrix} 0 & \alpha & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}, \quad D\mathcal{V}(TE_0) = \begin{bmatrix} \mu + \beta & 0 & 0 & 0 & 0 \\ -\beta & \gamma + \mu + \omega + \delta & -\theta & 0 & 0 \\ 0 & -\gamma & \mu + \theta & 0 & 0 \\ 0 & -\delta & 0 & \mu & 0 \\ 0 & \alpha & 0 & 0 & \mu \end{bmatrix}.$$

To facilitate the computation of R_0 , these Jacobian matrices can be partitioned as

$$D\mathcal{F}(x) = \begin{bmatrix} F & 0 \\ 0 & 0 \end{bmatrix}, \quad D\mathcal{V}(x) = \begin{bmatrix} V & 0 \\ J_1 & J_2 \end{bmatrix},$$

where the submatrices are defined as

$$F = \begin{bmatrix} 0 & \alpha & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, \quad V = \begin{bmatrix} \mu + \beta & 0 & 0 & 0 \\ -\beta & \gamma + \mu + \omega + \delta & -\theta & 0 \\ 0 & -\gamma & \mu + \theta & 0 \\ 0 & -\delta & 0 & 0 \end{bmatrix},$$

$$J_1 = \begin{bmatrix} 0 & \alpha & 0 & 0 \end{bmatrix}, \quad J_2 = \begin{bmatrix} \mu \end{bmatrix}.$$

The basic reproduction number R_0 is determined as the spectral radius (ρ) of the matrix FV^{-1} , which is computed as

$$FV^{-1} = \begin{bmatrix} \frac{\alpha\beta(\mu+\theta)}{\beta k + \delta l + \mu(\mu m + \omega\theta)} & \frac{\alpha(\mu+\theta)}{\mu(n+\delta) + \omega\theta} & \frac{\alpha\theta}{\mu(n+\delta) + \omega\theta} & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix},$$

with the parameters

$$k = \delta\mu + \delta\theta + \gamma\mu + \mu^2 + \mu\omega + \mu\theta + \omega\theta, \quad l = \mu^2 + \mu\delta,$$

$$m = \gamma + \mu + \omega + \theta, \quad n = \delta + \gamma + \mu + \omega + \theta.$$

Hence, the basic reproduction number R_0 is derived as

$$R_0 = \frac{\alpha\beta(\mu+\theta)}{(\beta + \mu)(\delta\mu + \delta\theta + \gamma\mu + \mu^2 + \mu\omega + \mu\theta + \omega\theta)}. \quad (23)$$

The disease-free equilibrium $TE_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right)$ is locally asymptotically stable if $R_0 < 1$, and becomes unstable when $R_0 > 1$.

3.2.2 Local Stability Analysis.

This section is devoted to the analysis of the local stability of the disease-free equilibrium (DFE), which provides a key criterion for determining whether malaria can be eliminated from the population.

Theorem 3.1. *Consider System (7) and its disease-free equilibrium*

$$TE_0 = \left(\frac{\Lambda}{\mu}, 0, 0, 0, 0\right).$$

Then the malaria-free equilibrium TE_0 is locally asymptotically stable if $R_0 < 1$, and unstable if $R_0 > 1$.

Proof. The Jacobian matrix of System (7) evaluated at the disease-free equilibrium TE_0 is given by

$$J(TE_0) = \begin{bmatrix} -\mu & 0 & -\alpha & 0 & 0 \\ 0 & -\mu - \beta & \alpha & 0 & 0 \\ 0 & \beta & -(\gamma + \mu + \omega + \delta) & \theta & 0 \\ 0 & 0 & \gamma & -\mu - \theta & 0 \\ 0 & 0 & \delta & 0 & -\mu \end{bmatrix}.$$

The characteristic equation is obtained from

$$\det(J(TE_0) - \lambda I) = 0$$

where

$$J(TE_0) - \lambda I = \begin{bmatrix} -\mu - \lambda & 0 & -\alpha & 0 & 0 \\ 0 & -\mu - \beta - \lambda & \alpha & 0 & 0 \\ 0 & \beta & -(\gamma + \mu + \omega + \delta) - \lambda & \theta & 0 \\ 0 & 0 & \gamma & -\mu - \theta - \lambda & 0 \\ 0 & 0 & \delta & 0 & -\mu - \lambda \end{bmatrix}.$$

A direct computation shows that the characteristic polynomial factorises as

$$(-\mu - \lambda)^2 (a\lambda^3 + b\lambda^2 + c\lambda + d) = 0, \quad (24)$$

where

$$a = -1,$$

$$b = -\beta - \delta - \gamma - 3\mu - \omega - \theta,$$

$$c = \alpha\beta - \beta\delta - \beta\gamma - 2\beta\mu - \beta\omega - \beta\theta - 2\delta\mu - \delta\theta - 2\gamma\mu - 3\mu^2 - 2\mu\omega - 2\mu\theta - \omega\theta,$$

$$d = \alpha\beta\mu + \alpha\beta\theta - \beta\delta\mu - \beta\delta\theta - \beta\gamma\mu - \beta\mu^2 - \beta\mu\omega - \beta\mu\theta - \beta\omega\theta - \delta\mu^2 - \delta\mu\theta - \gamma\mu^2 - \mu^3 - \mu^2\omega - \mu^2\theta - \mu\omega\theta.$$

Moreover, d can be rewritten in terms of the basic reproduction number R_0 as

$$d = K(R_0 - 1)$$

for some positive constant

$$K = \frac{1}{(\beta + \mu)(\delta\mu + \delta\theta + \gamma\mu + \mu^2 + \mu\omega + \mu\theta + \omega\theta)} > 0$$

so that the sign of d is entirely determined by the sign of $R_0 - 1$. From (24), two eigenvalues are immediately given by

$$\lambda_1 = \lambda_2 = -\mu$$

which are strictly negative since $\mu > 0$. The remaining three eigenvalues, denoted by $\lambda_3, \lambda_4, \lambda_5$, are the roots of the cubic equation

$$a\lambda^3 + b\lambda^2 + c\lambda + d = 0.$$

To determine the location of these roots in the complex plane, we apply the Routh–Hurwitz criterion for cubic polynomials as presented in [19] and further discussed in [20]. From the expressions above we have

$$a < 0, \quad b < 0,$$

and, as noted, $\text{sign}(d) = \text{sign}(R_0 - 1)$. In particular, when $R_0 < 1$ we have $d < 0$, and one can verify that the inequality

$$c < \frac{ad}{b}$$

is satisfied. Under these conditions, the Routh–Hurwitz criterion guarantees that all three roots $\lambda_3, \lambda_4, \lambda_5$ have negative real parts. Combined with $\lambda_1 = \lambda_2 = -\mu < 0$, this implies that all eigenvalues of $J(TE_0)$ lie in the left half-plane, so that TE_0 is locally asymptotically stable when $R_0 < 1$. On the other hand, when $R_0 > 1$ we have $d > 0$, and the Routh–Hurwitz conditions are violated in such a way that the cubic polynomial admits exactly one positive real root. Hence, in this case the Jacobian matrix $J(TE_0)$ has an eigenvalue with positive real part, and the disease-free equilibrium TE_0 is unstable. $\square$

As an illustration, consider how the parameters α and θ influence the basic reproduction threshold $\mathcal{R}_0$, with the other parameters fixed as $\Lambda = 55078.53$, $\delta = 0.07$, $\beta = \frac{1}{10}$, $\mu = 0.00692$, $\gamma = 0.1$, and $\omega = 0.00016$. The threshold $\mathcal{R}_0 = 1$ separates two distinct regions: one where $\mathcal{R}_0 < 1$, indicating disease elimination, and another where $\mathcal{R}_0 > 1$, indicating potential endemic persistence. To demonstrate the behavior in each region, two representative parameter points are considered. For $\alpha = 0.1$ and $\theta = 0.03$, the resulting value $\mathcal{R}_0 = 0.97604 < 1$ shows that the disease dies out. In this case, the susceptible population approaches $\frac{\Lambda}{\mu} \approx 7,960,198.84$, while the other compartments converge to zero, reflecting long-term malaria elimination. In contrast, for $\alpha = 0.12$ and $\theta = 0.1$, we obtain $\mathcal{R}_0 = 1.3433 > 1$, implying that malaria persists within the population. The compartments approach $S = 3,065,297.75$, $E = 611,208.26$, $I = 731,337.71$, $R_1 = 683,927.24$, and $R_2 = 2,846,170.18$, indicating a stable endemic state.

3.3 Stability Analysis of the Endemic Equilibrium Point.

We now investigate the local behaviour of System (7) in a neighbourhood of the endemic equilibrium

$$TE_1 = (S^* E^* I^* R_1^* R_2^*)$$

and derive sufficient and necessary conditions for its local asymptotic stability in terms of the coefficients of the characteristic polynomial.

Theorem 3.2. *Consider System (7) and assume that it admits a biologically meaningful endemic equilibrium*

$$TE_1 = (S^* E^* I^* R_1^* R_2^*).$$

Let $J(TE_1)$ denote the Jacobian matrix of System (7) evaluated at TE_1 , and let the associated characteristic equation be written as

$$-\lambda^5 + a\lambda^4 + b\lambda^3 + c\lambda^2 + d\lambda + e = 0 \quad (25)$$

where the real coefficients a, b, c, d, e are given by the expressions above. Then the endemic equilibrium TE_1 is locally asymptotically stable if and only if the following Routh–Hurwitz inequalities hold:

$$a < 0, \quad c < 0, \quad e < 0, \quad b < \frac{c}{a}, \quad d < \frac{e}{a}. \quad (26)$$

Proof. To analyse the local dynamics near TE_1 , we linearise System (7) around this equilibrium. The Jacobian matrix at TE_1 is

$$J(TE_1) = \begin{bmatrix} -\frac{\alpha I^*}{N} - \mu & 0 & -\frac{\alpha S^*}{N} & 0 & 0 \\ \frac{\alpha I^*}{N} & -\mu - \beta & \frac{\alpha S^*}{N} + \frac{\alpha R_2^*}{N} & 0 & \frac{\alpha I^*}{N} \\ 0 & \beta & -(\gamma + \mu + \omega + \delta) & \theta & 0 \\ 0 & 0 & \gamma & -\mu - \theta & 0 \\ 0 & 0 & \delta - \frac{\alpha R_2^*}{N} & 0 & -\mu - \frac{\alpha I^*}{N} \end{bmatrix}$$

where, for convenience, we have denoted

$$k = \gamma + \mu + \omega + \delta.$$

The characteristic polynomial of $J(TE_1)$ is obtained from

$$\det(J(TE_1) - \lambda I) = 0.$$

A direct (though lengthy) computation shows that this determinant can be written in the form

$$-\lambda^5 + a\lambda^4 + b\lambda^3 + c\lambda^2 + d\lambda + e = 0$$

that is, precisely Equation (25), where the coefficients a, b, c, d, e are the functions of the parameters and equilibrium coordinates $(S^*, E^*, I^*, R_1^*, R_2^*)$ displayed above.

For the purpose of applying the Routh–Hurwitz criterion, it is convenient to normalise the leading coefficient. Multiplying (25) by -1 , we obtain an equivalent polynomial

$$\lambda^5 - a\lambda^4 - b\lambda^3 - c\lambda^2 - d\lambda - e = 0$$

which we may rewrite in the standard form

$$\lambda^5 + A_1\lambda^4 + A_2\lambda^3 + A_3\lambda^2 + A_4\lambda + A_5 = 0$$

with

$$A_1 = -a, \quad A_2 = -b, \quad A_3 = -c, \quad A_4 = -d, \quad A_5 = -e.$$

The classical Routh–Hurwitz theorem for a fifth-degree polynomial with real coefficients

$$\lambda^5 + A_1\lambda^4 + A_2\lambda^3 + A_3\lambda^2 + A_4\lambda + A_5 = 0$$

states that all its roots have negative real parts if and only if a suitable sequence of Hurwitz determinants (or equivalently, the entries of the Routh array) are strictly positive. In the present setting, and after substituting $A_1, \dots, A_5$ in terms of $a, \dots, e$, these Routh–Hurwitz conditions reduce to the set of inequalities

$$a < 0, \quad c < 0, \quad e < 0, \quad b < \frac{c}{a}, \quad d < \frac{e}{a},$$

see, for instance, [19, 20] for the general form of the criterion and its application to systems of similar structure. Consequently, all eigenvalues of $J(TE_1)$ have negative real parts if and only if the inequalities in (26) are satisfied. Since the eigenvalues of the Jacobian at TE_1 determine the local behaviour of the nonlinear system, we conclude that the endemic equilibrium TE_1 is locally asymptotically stable precisely under conditions (26). $\square$

4 NUMERICAL ANALYSIS

This section presents numerical simulations to illustrate the dynamical behaviour predicted by the theoretical analysis. All simulations and numerical experiments were implemented in Python 3 and executed on a personal computer equipped with a 12th Gen Intel(R) Core(TM) i3-1215U processor (1.20 GHz), a 64-bit operating system with x64-based architecture, and 8 GB of RAM. The parameter values used in the simulations are summarized in Table 3 and are also indicated in the corresponding figures.

TABLE 3. The parameters value of the model

No	Parameters	Calculation and References	Values
1	Λ	[21]	400
2	α	0.551 [6] $\times (0.3)(0.05)$ [22]	0.008265
3	β	[22]	1/17
4	γ	0.4×0.01 [22]	0.004
5	θ	[22]	0.2
6	ω	[6]	0.0004
7	δ	0.6×0.01 [22]	0.006
8	μ	[7]	0.03

In this study, the parameter values are specified based on epidemiological data from Indonesia. In particular, the value 0.551 represents the estimated proportion of malaria cases reported in Indonesia in 2024 that are attributable to *Plasmodium falciparum*. This implies that approximately 55.1% of all documented malaria infections are assumed to be falciparum malaria. Consequently, the proposed model is primarily calibrated to capture the transmission dynamics of *P. falciparum*, which is recognized as the most clinically severe and epidemiologically significant malaria species in the Indonesian context.

The value 0.3 represents the probability that a mosquito becomes infected after biting an infected individual. This implies that, on average, there is a 30% chance that a mosquito acquires the parasite and becomes infectious during a blood meal on a human host carrying *P. falciparum*. The value 0.05 denotes the average number of mosquito bites per person per unit time, corresponding to the mean biting rate experienced by an individual. This parameter characterizes the intensity of human, mosquito contact and therefore directly determines the force of infection within the model.

In the absence of detailed population-level data from Indonesia distinguishing between *Plasmodium falciparum* infections with incomplete parasite clearance and those achieving full parasitological cure, we decompose the total recovery rate, set to 0.01, into two mutually exclusive pathways. Specifically, we assume that 40% of recovering individuals transition into the recrudescence-risk compartment R_1 , while the remaining 60% enter the fully recovered yet reinfection-susceptible compartment R_2 . By construction, this assumption satisfies $\gamma + \delta = 0.01$ and is motivated by two qualitative considerations. First, complete recovery is expected to be more common than persistent sub-microscopic parasitaemia, consistent with the high clinical efficacy of artemisinin-based combination therapies, implying $\delta > \gamma$. Second, the proportion assigned to R_1 is chosen sufficiently large to allow recrudescence to exert a non-negligible influence on the system dynamics. We emphasise that the values 0.4 and 0.6 serve as baseline assumptions; additional numerical experiments indicate that varying δ over a broad range does not affect the qualitative stability properties of either the disease-free or endemic equilibria.

4.1 General Graphical Representation.

Based on the numerical results illustrated in Figure 2, the temporal dynamics of the system are examined under two distinct scenarios determined by the basic reproduction number R_0 . The upper panel corresponds to the case $R_0 < 1$, whereas the lower panel depicts the system behavior when $R_0 > 1$. In the scenario $R_0 < 1$, the susceptible population $S(t)$ increases rapidly and converges to its steady-state value, while the exposed $E(t)$, infected $I(t)$, recrudescence $R_1(t)$, and reinfective $R_2(t)$ compartments asymptotically approach zero. This behavior indicates that malaria transmission cannot be sustained and that the system converges to the disease-free equilibrium $TE_0 = (S, 0, 0, 0, 0)$, corresponding to the eventual elimination of the disease from the population.

Conversely, when $R_0 > 1$, the exposed E and infectious I populations increase rapidly during the initial phase, indicating the onset of an outbreak. This growth is followed by a substantial rise in the recovered compartments R_1 and R_2 , reflecting the accumulation of individuals who have undergone treatment, either with a risk of recrudescence or full recovery with susceptibility to reinfection. At the same time, the susceptible population S declines markedly due to sustained transmission. After the transient dynamics subside, all compartments converge to positive steady-state values, indicating the establishment of an endemic equilibrium. These numerical results are fully consistent with the analytical findings, confirming that the system converges to the disease-free equilibrium when $R_0 < 1$ and to an endemic equilibrium when $R_0 > 1$.

4.2 Graph of R_0 Region (Parameter Interdependencies).

The graphs illustrate the relationship between the transmission parameter α and the recrudescence parameter θ under varying values of δ and β . As shown, when treatment is introduced with an initial recovery rate of $\delta = 0.001$ and subsequently increased, the potential for malaria transmission decreases markedly. This effect is

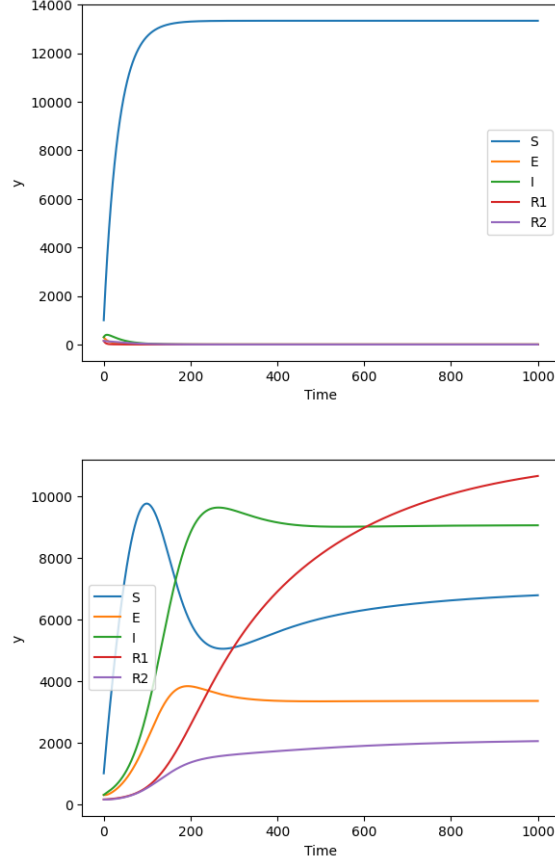


FIGURE 2. Trajectory of System 7 with different values of R_0 .

reflected in the expansion of the region where $R_0 < 1$, corresponding to the disease-free regime. An increase in δ , which represents the effectiveness of treatment and medical intervention, therefore enlarges the parameter space associated with disease elimination. These results indicate that enhanced treatment efficacy plays a crucial role in suppressing malaria transmission and reducing the likelihood of sustained endemicity.

In contrast, the graph below indicates that increasing the value of β leads to a contraction of the region where $R_0 < 1$. This contraction implies a higher likelihood of sustained transmission, as a larger proportion of the parameter space corresponds to $R_0 > 1$. Consequently, the number of infected individuals tends to increase, while the disease-free state becomes more difficult to achieve. Since β represents the rate at which exposed individuals progress to the infectious class, higher values of this parameter accelerate the onset of infectiousness and amplify malaria transmission within the population.

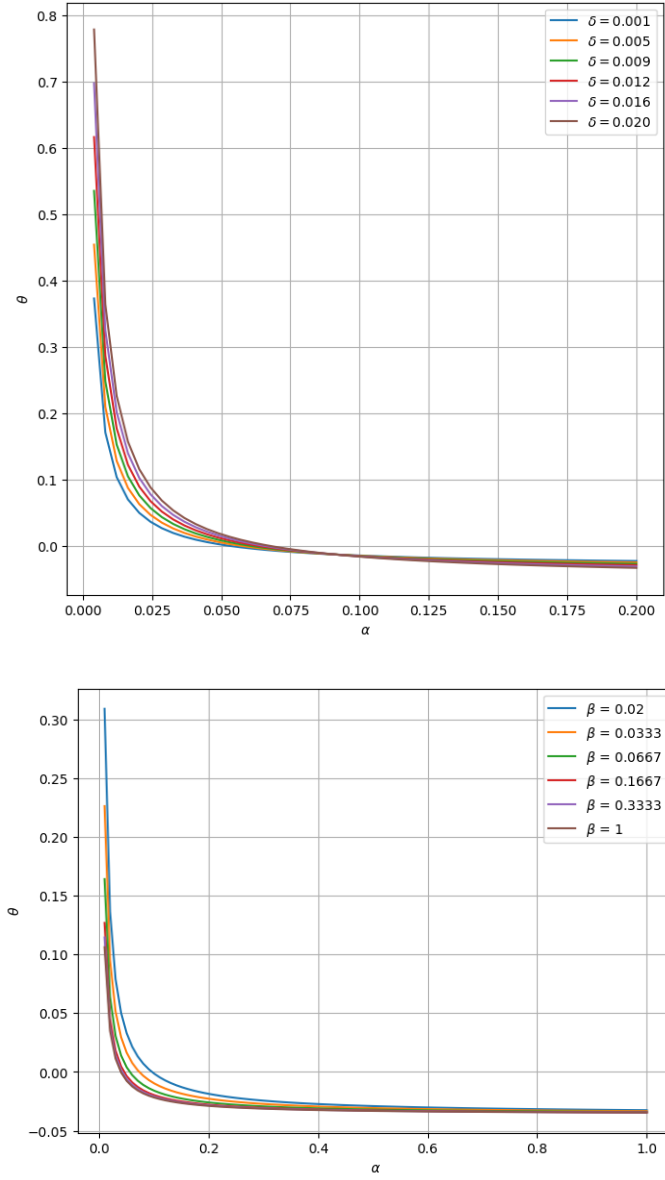


FIGURE 3. Parameter effects on *Plasmodium falciparum* Dynamics

4.3 Graphs of Inter-Variable Relationships.

This section presents various phase portraits that illustrate the interaction dynamics among different population groups in the malaria transmission model. Each

graph depicts the relationship between two key variables in the system, demonstrating how the number of individuals in a particular category evolves over time

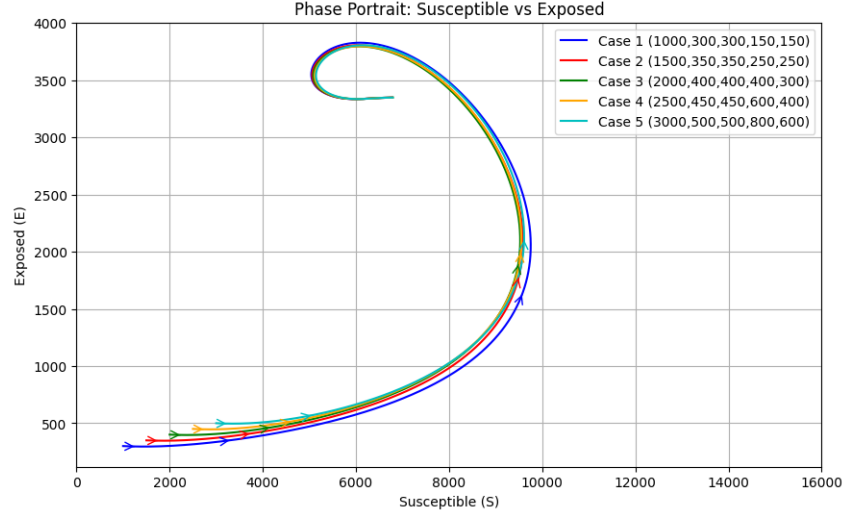


FIGURE 4. S vs E

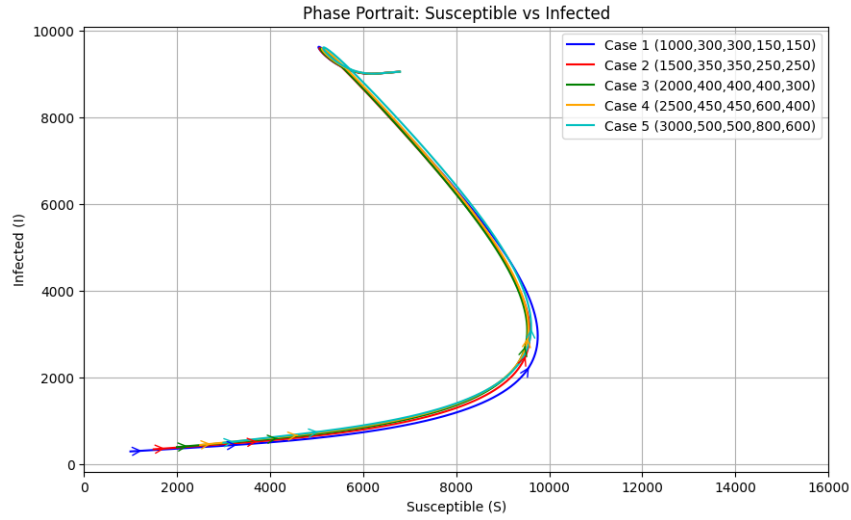


FIGURE 5. S vs I

In Figure 4 and Figure 5, as the number of susceptible individuals (S) increases, the number of exposed (E) and infected (I) individuals also rises. However,

after reaching an inflection point, which marks the transition from the exponential phase to the stable phase, the trend stabilizes. This suggests that malaria may persist in the population with a stable infection rate.

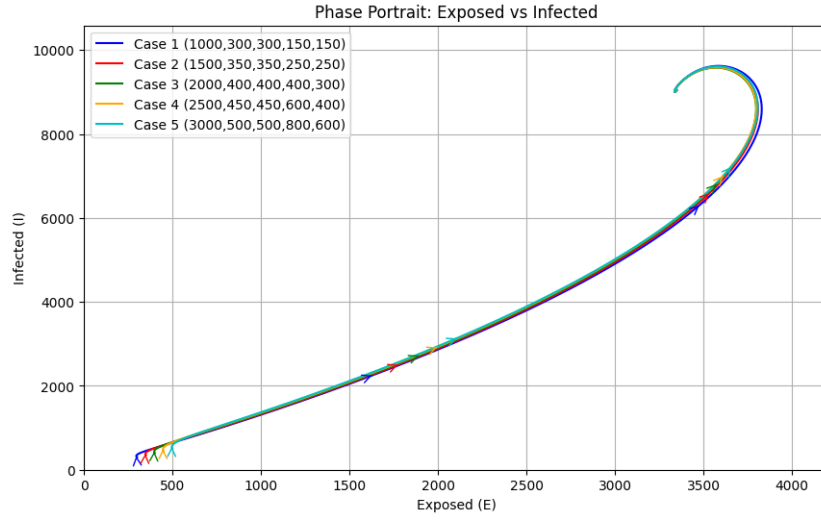


FIGURE 6. E vs I

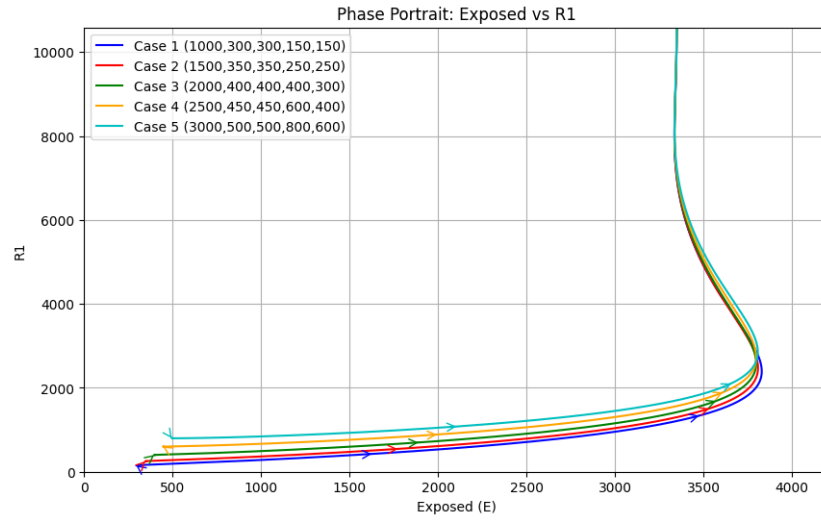
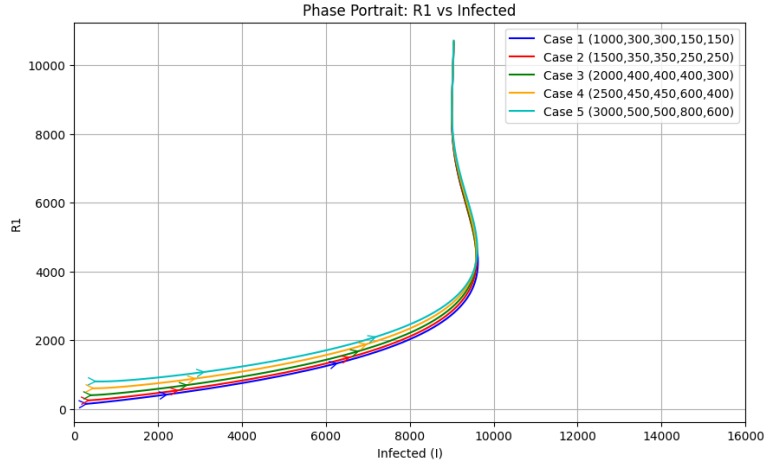
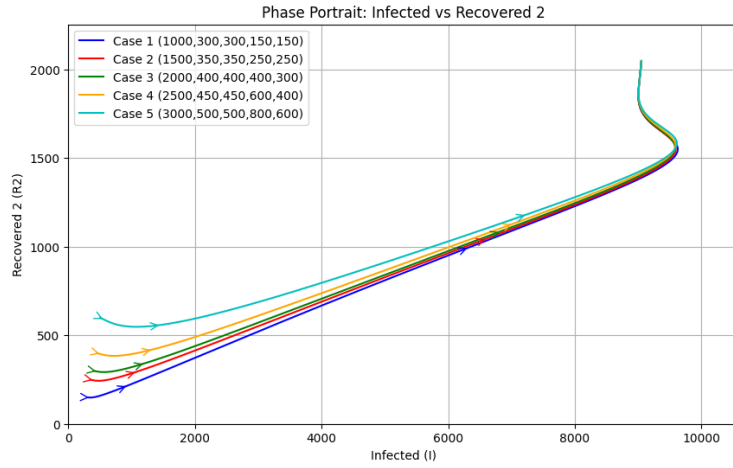


FIGURE 7. E vs R_1

In Figure 6 and Figure 7, as the exposed population increases, the number of infected individuals (I) and recrudescence cases (R_1) also rises. After a certain

period, the number of exposed individuals begins to decline, while the number of infected individuals continues to increase and shows a stabilizing trend (equilibrium), which may indicate factors of recrudescence and the effects of interventions (such as treatment or prevention). In Figure 7, an increase in E does not immediately cause a significant spike in R_1 , but once a certain threshold is surpassed, recrudescence increases drastically. This suggests that resistance to malaria drugs may be developing.

FIGURE 8. I vs R_1 FIGURE 9. I vs R_2

In Figure 8, the graph illustrates the progression of disease dynamics, revealing that an increase in the number of infected individuals is associated with a greater probability of recrudescence cases. The pronounced peak observed in the graph may reflect a critical threshold in the transmission process, wherein infections escalate rapidly before subsequently declining. Meanwhile, Figure 9 displays an approximately linear correlation between the infected population and the recovered individuals in the R_2 class, suggesting that the immune response or medical intervention is sufficiently effective in managing the spread of the disease without inducing severe complications.

5 CONCLUDING REMARKS

This study proposes an extended SEIR-type framework for malaria transmission that explicitly incorporates both *recrudescence* and *reinfection* through two additional compartments, R_1 and R_2 . In contrast to many existing malaria models, which typically assume complete recovery or treat recurrent infection in an aggregated manner, the present formulation distinctly separates recurrence arising from incomplete parasite clearance from renewed susceptibility following full recovery. By doing so, the model provides a more nuanced representation of post-treatment outcomes and extends earlier SEIR-based approaches to recurrent malaria reported in the literature. This formulation is particularly relevant for endemic settings, where repeated *Plasmodium* infections and treatment-related recurrences play a critical role in sustaining transmission.

Using the Next Generation Matrix approach, we derived the basic reproduction number R_0 and demonstrated how transmission, recovery, and recrudescence parameters jointly govern the long-term behavior of malaria transmission. In agreement with classical infectious disease theory and previous malaria modelling studies, both the analytical results and numerical simulations indicate that $R_0 < 1$ leads to disease elimination, whereas $R_0 > 1$ gives rise to a stable endemic equilibrium. The numerical findings further reveal that enhancing treatment effectiveness, reducing the likelihood of recrudescence, and limiting reinfection can substantially decrease R_0 and, consequently, lower the long-term prevalence of malaria infection.

From a practical standpoint, these findings reinforce conclusions from earlier modelling and epidemiological studies that emphasize the importance of strengthening treatment protocols, post-treatment follow-up, vector control, and community awareness. In particular, calibrating and validating the proposed model using district-level surveillance data from Indonesia would enable the estimation of context-specific recrudescence and reinfection rates, thereby situating the present work within the growing body of data-informed malaria models and enhancing its relevance for policy design in endemic regions. Future extensions of the model may incorporate an explicit mosquito subsystem, age or immunity structure, seasonal forcing, and stochastic effects to further improve its realism and alignment with observed malaria transmission dynamics.

Acknowledgement. Acknowledgements may be presented here, if any.

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