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APPLIED MATHEMATICS

A mathematical model of malaria transmission dynamics with multi-stage infection and dual treatment pathways

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Abstract
Malaria remains a major global health challenge due to the complex interaction between biological transmission and treatment-seeking behavior. This study aims to analyze malaria transmission dynamics by incorporating multi-stage infection and integrated dual treatment pathways consisting of herbal and formal medical treatment. A deterministic nonlinear ordinary differential equation model was developed and analyzed using the Next Generation Matrix approach to derive the basic reproduction number R_0, while local stability analysis was performed using the Routh–Hurwitz criterion. The results show that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$, indicating the threshold condition for malaria elimination. Sensitivity analysis demonstrates that increasing the treatment-seeking rate during the mild infection stage (τ_h) significantly reduces R_0 and suppresses disease transmission. Furthermore, numerical simulations reveal that the medical transition rate (ϕ), representing referral from herbal to formal medical treatment, plays an important role in reducing endemic prevalence (I_r^*), despite having negligible influence on R_0. The interaction between early treatment access and referral mechanisms further improves long-term intervention effectiveness under endemic conditions. These findings suggest that sustainable malaria elimination requires synergistic integration between rapid early-stage treatment access and efficient referral systems across treatment pathways.

Keywords: herbal treatment, dual treatment pathways, malaria dynamics, mathematical modelling, multi-stage infection

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INTRODUCTION

Malaria is an infectious disease that continues to pose a major global public health challenge, particularly in tropical and subtropical regions. The disease is caused by parasites of the genus *Plasmodium* and is transmitted through the bites of infected female *Anopheles* mosquitoes. The World Health Organization (WHO) reports that malaria remains one of the most significant global health concerns, with hundreds of millions of cases reported annually and a high mortality rate, especially in developing countries. According to the World Malaria Report, it is estimated that approximately 267 million malaria cases and more than 567,000 deaths occurred worldwide in 2023 (World Health Organization, 2023). In Indonesia, although a national decline in malaria incidence has been observed, the disease remains endemic in several regions, particularly in eastern parts of the country (Aisyah et al., 2024).

Malaria not only causes significant clinical consequences but also generates considerable social and economic burdens. High infection prevalence can reduce labor productivity, increase healthcare expenditures, and disrupt social and educational activities within communities. Over

time, these impacts may hinder economic development, particularly in malaria-endemic regions (Andrade et al., 2022; Sachs & Malaney, 2002). Therefore, a comprehensive understanding of malaria transmission mechanisms is essential for designing effective control strategies.

The transmission dynamics of malaria involve complex interactions between humans as hosts, mosquitoes as vectors, and various biological and behavioral factors. Several factors influence these dynamics, including the rate of human–mosquito contact, the incubation period of the parasite, population demographic dynamics, and individual responses to infection. Moreover, malaria infection in humans is not homogeneous. Clinically, infections may manifest at different levels of severity, each characterized by distinct symptoms and varying risks of complications (Kotepui et al., 2020). Variations in community behavior related to treatment-seeking also contribute to the dynamics of the disease. In many endemic regions, socioeconomic conditions, healthcare accessibility, cultural beliefs, and treatment costs influence whether infected individuals seek traditional herbal remedies or formal medical treatment (Adhikari et al., 2019). These differences in treatment preference may affect the duration of infection, the progression from mild to severe infection, and ultimately the overall transmission dynamics within the population. Therefore, incorporating heterogeneous treatment pathways into malaria transmission models is important for representing realistic intervention scenarios in endemic communities.

Due to the complexity of these interactions, malaria transmission dynamics cannot be fully understood through empirical observation alone. Mathematical modeling provides a quantitative framework to represent disease transmission processes systematically. By employing systems of differential equations, such models enable rigorous analysis of key epidemiological indicators, including the number of infected individuals and the basic reproduction number R_0, which are influenced by factors such as human–mosquito contact rates, transmission rates, latent periods, and treatment-seeking behavior (Jaleta et al., 2025; Kuddus & Rahman, 2021).

Mathematical modeling of malaria transmission has evolved since the classical Ross–Macdonald model, which introduced the concept of the basic reproduction number as an indicator of disease spread (Macdonald. G, 1957; Ross, 1911). This framework was subsequently extended through various compartmental models, such as the SEIR model, which separates the latent phase from active infection and incorporates vector dynamics more explicitly (Anderson & May, 1991). Subsequent studies have further integrated intervention strategies, including treatment and vector control, both of which have been shown to reduce the number of infected individuals and lower the basic reproduction number (Chitnis et al., 2008).

Recent studies on malaria transmission modeling have increasingly incorporated treatment-related interventions and behavioral responses into compartmental frameworks. Several mathematical models have investigated the effects of treatment-seeking behavior, optimal control strategies, relapse dynamics, healthcare accessibility, and behavioral heterogeneity on malaria transmission dynamics. These studies demonstrated that delayed access to treatment and variations in behavioral responses may increase disease persistence and reduce the effectiveness of malaria control strategies (Haile et al., 2025; Jaleta et al., 2025; Kuddus & Rahman, 2021). However, despite these important contributions, most existing models still represent treatment interventions in an aggregated manner and do not explicitly distinguish between multiple infection severity levels and heterogeneous treatment pathways. In particular, the interaction between disease progression and transitions between traditional herbal treatment and formal medical treatment remains insufficiently explored within a unified malaria transmission framework. To address these gaps, this study develops a deterministic mathematical model of malaria transmission that simultaneously incorporates two levels of infection severity (mild and severe) and dual treatment pathways, namely herbal and medical treatment. This integrated approach provides a more realistic representation of how clinical

progression and treatment-seeking behavior jointly influence transmission dynamics. The analysis includes equilibrium analysis, derivation of the basic reproduction number (R_0), stability analysis, sensitivity analysis, and numerical simulations to evaluate the epidemiological impact of treatment-seeking behavior. The primary objective of this study is to investigate how variations in treatment-seeking behavior across different stages of infection influence malaria transmission and endemic persistence within the population.

RESEARCH METHOD

This study employs a deterministic mathematical modeling approach to analyze the dynamics of malaria transmission by incorporating two levels of infection severity in humans and treatment-seeking behavior. The model is formulated as a system of ordinary differential equations (ODEs) representing the interactions between human and mosquito populations. The analysis includes the determination of equilibrium points, derivation of the basic reproduction number, stability analysis, sensitivity analysis of model parameters, and numerical simulations to evaluate the impact of different treatment-seeking behaviors on malaria transmission dynamics.

The compartmental structure of the malaria transmission model is illustrated in the flow diagram shown in Figure 1.

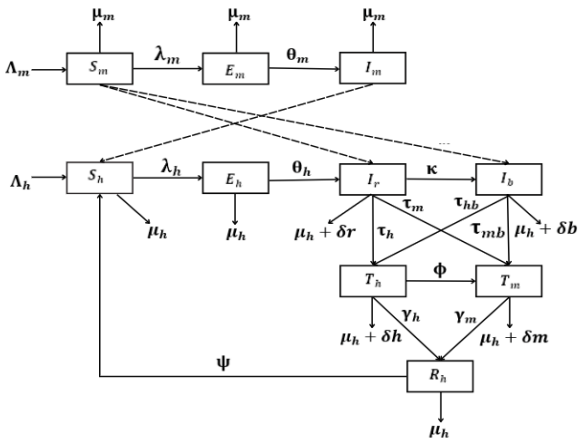


Figure 1. Compartmental diagram of the malaria transmission model.

The human population is divided into seven epidemiological compartments: S_h (susceptible humans), E_h (exposed humans), I_r (humans with mild infection), I_b (humans with severe infection), T_h (humans undergoing herbal treatment), T_m (humans undergoing medical treatment), and R_h (recovered humans). Meanwhile, the mosquito population is divided into three compartments: S_m (susceptible mosquitoes), E_m (exposed mosquitoes), and I_m (infected mosquitoes). The total human and mosquito populations at time t are respectively expressed as:

$$N_h = S_h + E_h + I_r + I_b + T_h + T_m + R_h \quad (1)$$

$$N_m = S_m + E_m + I_m \quad (2)$$

Malaria transmission in humans occurs through the bites of infectious mosquitoes, allowing susceptible individuals to become infected and enter the exposed stage. Exposed individuals subsequently progress to the mild infection stage, which may further develop into severe infection if treatment is not received. Infected individuals may undergo treatment through two pathways, namely herbal treatment and medical treatment. The recovery rate under medical treatment is assumed to be higher than that under herbal treatment $(\gamma_m > \gamma_h)$, reflecting the

generally higher clinical effectiveness of formal medical care reported in malaria treatment studies. Therefore, the positive effect associated with increased herbal treatment rates should be interpreted as the benefit of early-stage treatment-seeking behavior rather than evidence of superior efficacy of herbal treatment itself.

During the treatment period, individuals are assumed to contribute minimally to disease transmission. The treatment process occurs only within the human population. Individuals who have recovered are assumed not to acquire permanent immunity and may therefore return to the susceptible class. Within the mosquito population, susceptible mosquitoes may become infected after biting infected humans. Following an incubation period, they become infectious and remain in this state for the remainder of their lifespan, since mosquitoes do not recover from infection.

These assumptions are commonly adopted in deterministic epidemiological models to preserve biological realism while maintaining analytical tractability (Hethcote, 2000). Several parameters related to treatment-seeking behavior and treatment outcomes, including the herbal treatment rate, the transition rate from herbal to medical treatment, and treatment-associated mortality rates, were assumed due to the limited availability of epidemiological data regarding heterogeneous treatment pathways in malaria-endemic regions. The assumed parameter values were selected within biologically plausible ranges reported in previous epidemiological and behavioral studies and were further evaluated through sensitivity analysis.

The malaria transmission rate is represented through the force of infection λ_h (mosquito-to-human transmission) and λ_m (human-to-mosquito transmission), as defined in Equation (3), namely:

$$\lambda_h = \beta_h \frac{I_m}{N_m}, \quad \lambda_m = \beta_m \frac{I_r + I_b}{N_h} \tag{3}$$

Based on the compartmental structure and the assumptions described above, the malaria transmission dynamics are represented by the following system of nonlinear differential equations:

$$\begin{cases} \frac{dS_h}{dt} = \Lambda_h - \lambda_h S_h + \psi R_h - \mu_h S_h \\ \frac{dE_h}{dt} = \lambda_h S_h - \theta_h E_h - \mu_h E_h \\ \frac{dI_r}{dt} = \theta_h E_h - (\kappa + \tau_h + \tau_m + \mu_h + \delta_r) I_r \\ \frac{dI_b}{dt} = \kappa I_r - (\tau_{hb} + \tau_{mb} + \mu_h + \delta_b) I_b \\ \frac{dT_h}{dt} = \tau_h I_r + \tau_{hb} I_b - (\phi + \gamma_h + \delta_h + \mu_h) T_h \\ \frac{dT_m}{dt} = \tau_m I_r + \tau_{mb} I_b + \phi T_h - (\gamma_m + \mu_h + \delta_m) T_m \\ \frac{dR_h}{dt} = \gamma_h T_h + \gamma_m T_m - \mu_h R_h - \psi R_h \\ \frac{dS_m}{dt} = \Lambda_m - \lambda_m S_m - \mu_m S_m \\ \frac{dE_m}{dt} = \lambda_m S_m - \theta_m E_m - \mu_m E_m \\ \frac{dI_m}{dt} = \theta_m E_m - \mu_m I_m \end{cases} \tag{4}$$

The definitions and values of the parameters used in the model are presented in Table 1.

Parameters without available epidemiological estimates were assumed within biologically reasonable ranges to preserve the qualitative dynamics of malaria transmission and were further evaluated through sensitivity analysis.

The analysis of the proposed model is conducted through several stages. First, a compartmental model describing malaria transmission between humans and mosquitoes is constructed and formulated into a system of nonlinear ordinary differential equations. Subsequently, the disease-free and endemic equilibrium points are determined and the basic reproduction number R_0 is derived using the Next Generation Matrix method.

Table 1. Caption

Parameter	Description	Value	Reference
Λ_h	Human recruitment rate (individuals/day)	46	Assumed
Λ_m	Mosquito recruitment rate (individuals/day)	10^6	Assumed
μ_h	Natural mortality rate of humans (/day)	4.6×10^{-5}	(World Health Organization, 2023)
μ_m	Natural mortality rate of mosquitoes (/day)	0.1	(Smith et al., 2012)
β_h	Transmission rate from mosquitoes to humans (/day)	0.3	(Chitnis et al., 2006)
β_m	Transmission rate from humans to mosquitoes (/day)	0.2	(Ngwa & Shu, 2000)
θ_h	Incubation rate from exposed humans to mild infection (/day)	0.083	(Mandal et al., 2011)
θ_m	Incubation rate from exposed mosquitoes (/day)	0.1	(Smith et al., 2012)
κ	Progression rate from mild to severe infection (/day)	0.025	(Tangpukdee et al., 2007)
τ_h	Herbal treatment rate for mild infection in humans (/day)	0.4	Assumed
τ_{hb}	Herbal treatment rate for severe infection in humans (/day)	0.06	Assumed
τ_m	Medical treatment rate for mild infection in humans (/day)	0.05	(Jaleta et al., 2025)
τ_{mb}	Medical treatment rate for severe infection in humans (/day)	0.10	(Jaleta et al., 2025)
ϕ	Transition rate from herbal treatment to medical treatment (/day)	0.2	Assumed
γ_h	Recovery rate under herbal treatment (/day)	$\frac{1}{14}$	(Aldila, 2022)
γ_m	Recovery rate under medical treatment (/day)	$\frac{1}{7}$	(White, 2017)
δ_r	Disease-induced mortality rate for mild infection (/day)	0.0001	(White, 2017)
δ_b	Disease-induced mortality rate for severe infection (/day)	0.01	(Mandal et al., 2011)
δ_h	Mortality rate during herbal treatment (/day)	0.002	Assumed
δ_m	Mortality rate during medical treatment (/day)	0.00005	Assumed
ψ	Rate of loss of immunity (/day)	$\frac{1}{180}$	(Tumwiine et al., 2007)

The local stability of the equilibrium points is then analyzed using the Routh–Hurwitz criteria. Furthermore, parameter sensitivity analysis is performed to identify the parameters that most strongly influence malaria transmission. Finally, numerical simulations are carried out to illustrate the effects of different treatment-seeking behaviors on the dynamics of malaria transmission.

Numerical simulations were performed in Python using the odeint solver from the SciPy library, which is based on the LSODA algorithm with adaptive step-size control. The simulations were computed over the interval $t \in [0,1000]$ using 1000 discretization points.

RESULTS AND DISCUSSION

Positivity and Boundedness of Solutions

Biologically, population variables cannot take negative values. Therefore, all state variables in the system (4) must remain non-negative for all $t \geq 0$. Assume that the initial conditions satisfy

$$S_h(0), E_h(0), I_r(0), I_b(0), T_h(0), T_m(0), R_h(0), S_m(0), E_m(0), I_m(0) \geq 0. \quad (5)$$

from the structure of the differential equations in system (4), each equation consists of recruitment terms and transition terms that are proportional to the state variables. When a compartment reaches the boundary $x_i = 0$, it follows that $\dot{x}_i \geq 0$. Consequently, the trajectories of the system cannot cross the coordinate axes into the negative region. Hence, the solutions of the system remain in the non-negative orthant for all $t \geq 0$.

Furthermore, by using the definition of the total human population as given in Equation (1), we obtain

$$\frac{dN_h}{dt} = \Lambda_h - \mu_h N_h - \delta_r I_r - \delta_b I_b - \delta_h T_h - \delta_m T_m. \quad (6)$$

Since all mortality parameters are positive and all compartments are non-negative, the inequality

$$\frac{dN_h}{dt} \leq \Lambda_h - \mu_h N_h. \quad (7)$$

holds. By applying the comparison principle, the following bound is obtained:

$$0 \leq N_h(t) \leq \frac{\Lambda_h}{\mu_h}. \quad (8)$$

which indicates that the human population remains bounded (Brauer et al., 2012).
Similarly, using the definition of the mosquito population in Equation (2), we obtain

$$\frac{dN_m}{dt} = \Lambda_m - \mu_m N_m \quad (9)$$

which implies

$$0 \leq N_m(t) \leq \frac{\Lambda_m}{\mu_m}. \quad (10)$$

Therefore, system (4) possesses a positively invariant bounded region given by

$$\Omega = \left\{ R_+^{10} : N_h \leq \frac{\Lambda_h}{\mu_h}, N_m \leq \frac{\Lambda_m}{\mu_m} \right\}. \quad (11)$$

These results demonstrate that system (4) is mathematically well-posed and epidemiologically meaningful (Brauer et al., 2012).

Equilibrium Point

The equilibrium points of system (4) are obtained by imposing the condition $\frac{dX}{dt} = 0$, which corresponds to the state where all variables in the system remain constant over time (Perko, 2013). From this condition, two types of equilibrium solutions are obtained, namely the disease-free equilibrium and the endemic equilibrium.

The disease-free equilibrium (DFE) is obtained by setting all infection-related compartments equal to zero, $E_h^0 = I_r^0 = I_b^0 = T_h^0 = T_m^0 = R_h^0 = E_m^0 = I_m^0 = 0$ so that the system only retains the susceptible compartments $S_h^0 = \frac{\Lambda_h}{\mu_h}, S_m^0 = \frac{\Lambda_m}{\mu_m}$. Hence, the disease-free equilibrium point is

$$E_0 = \left(\frac{\Lambda_h}{\mu_h}, 0,0,0,0,0,0, \frac{\Lambda_m}{\mu_m}, 0,0\right) \tag{12}$$

Biologically, this state represents a situation where the entire human and mosquito populations are susceptible and no infection is present.

The endemic equilibrium occurs when malaria persists within the population and is denoted by

$$E^* = (S_h^*, E_h^*, I_r^*, I_b^*, T_h^*, T_m^*, R_h^*, S_m^*, E_m^*, I_m^*) \tag{13}$$

The components satisfy the following nonlinear algebraic system:

$$S_h^* = \frac{\Lambda_h + \psi R_h}{\mu_h + \lambda_h}, \qquad T_m^* = \frac{\tau_m I_r + \tau_{mb} I_b + \phi T_h}{a_4},$$
$$E_h^* = \frac{\lambda_h S_h}{\theta_h + \mu_h}, \qquad R_h^* = \frac{\gamma_h T_h + \gamma_m T_m}{\mu_h + \psi},$$
$$I_r^* = \frac{\theta_h E_h}{a_1}, \qquad S_m^* = \frac{\Lambda_m}{\mu_m + \lambda_m},$$
$$I_b^* = \frac{\kappa I_r}{a_2}, \qquad E_m^* = \frac{\lambda_m S_m}{\theta_m + \mu_m},$$
$$T_h^* = \frac{\tau_h I_r + \tau_{hb} I_b}{a_3}, \qquad I_m^* = \frac{\theta_m E_m}{\mu_m}.$$

The components satisfy the following nonlinear algebraic system:

$$a_1 = \kappa + \tau_h + \tau_m + \mu_h + \delta_r, \qquad a_3 = \phi + \gamma_h + \delta_h + \mu_h,$$
$$a_2 = \tau_{hb} + \tau_{mb} + \mu_h + \delta_b, \qquad a_4 = \gamma_m + \mu_h + \delta_m.$$

The equilibrium values satisfy a nonlinear algebraic system determined by the forces of infection λ_h and λ_h. The existence of this equilibrium depends on the value of the basic reproduction number R_0, which acts as a threshold parameter determining whether the infection dies out or persists in the population.

Basic Reproduction Number R_0

The basic reproduction number R_0 is derived using the Next Generation Matrix method (Van den Driessche & Watmough, 2002). The infected compartments involved in the linearization around the disease-free equilibrium are E_h, I_r, I_b, E_m, I_m. Let F denote the rate of new infections

and V represent the transition terms. By evaluating the Jacobian matrices of F and V at the disease-free equilibriumE_0, the transmission matrices are obtained as:

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & \beta_h \frac{S_h^0}{N_m} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \beta_m \frac{S_m^0}{N_h} & \beta_m \frac{S_m^0}{N_h} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{14}$$

$$V = \begin{pmatrix} \theta_h + \mu_h & 0 & 0 & 0 & 0 \\ -\theta_h & a_1 & 0 & 0 & 0 \\ 0 & -\kappa & a_2 & 0 & 0 \\ 0 & 0 & 0 & \theta_m + \mu_m & 0 \\ 0 & 0 & 0 & -\theta_m & \mu_m \end{pmatrix} \tag{15}$$

The next-generation matrix is obtained as:

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{\beta_h \frac{S_h^0}{N_m} \theta_m}{(\theta_m + \mu_m)\mu_m} & \frac{\beta_h \frac{S_h^0}{N_m}}{\mu_m} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ \frac{\beta_m \frac{S_m^0}{N_h} \theta_h (a_2 + \kappa)}{(\theta_h + \mu_h) a_1 a_2} & \frac{\beta_m \frac{S_m^0}{N_h} (a_2 + \kappa)}{a_1 a_2} & \frac{\beta_m \frac{S_m^0}{N_h}}{a_2} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \tag{16}$$

The basic reproduction number is defined as the spectral radius of the next-generation matrix, namely $R_0 = \rho(FV^{-1})$. After algebraic simplification, the expression for the basic reproduction number becomes

$$R_0 = \sqrt{\frac{\beta_h \beta_m S_h^0 S_m^0 \theta_h \theta_m (a_2 + \kappa)}{N_h N_m (\theta_h + \mu_h) a_1 a_2 (\theta_m + \mu_m) \mu_m}}. \tag{17}$$

The square-root structure reflects the two-step transmission cycle characteristic of vector-borne diseases, namely the human–mosquito–human transmission pathway. Epidemiologically, the value of R_0 determines the potential for disease invasion. If $R_0 < 1$, each infected individual generates less than one secondary case on average and the infection eventually disappears. Conversely, if $R_0 > 1$, the disease can spread within the population and an endemic state may occur (Brauer et al., 2012).

Stability Analysis

The local stability of the disease-free equilibrium point is analyzed by linearizing system (4) around this equilibrium using the Jacobian matrix (Perko, 2013). The Jacobian matrix is obtained by taking the partial derivatives of each equation with respect to the state variables. In general, the Jacobian matrix of the system can be written as

$$J=\begin{pmatrix} -(\mu_h+\lambda_h) & 0 & 0 & 0 & 0 & 0 & \psi & 0 & 0 & -b_1 \\ 0 & -(\theta_h+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & b_1 \\ 0 & \theta_h & -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \kappa & -a_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_h & \tau_{hb} & -a_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_m & \tau_{mb} & \phi & -a_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma_h & \gamma_m & -\mu_h-\psi & 0 & 0 & 0 \\ 0 & 0 & -b_2 & -b_2 & 0 & 0 & 0 & -(\mu_m+\lambda_m) & 0 & 0 \\ 0 & 0 & b_3 & b_3 & 0 & 0 & 0 & \lambda_m & -(\theta_m+\mu_m) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta_m & -\mu_m \end{pmatrix} \quad (18)$$

where:

$$b_1=\beta_h\frac{S_h^0}{N_m}+\beta_h\frac{I_mS_h^0}{N_m^2}, \qquad b_2=\beta_m\frac{S_m^0}{N_h}+\beta_m\frac{(I_r+I_b)I_mS_m^0}{N_h^2},$$
$$b_3=\beta_m\frac{S_m^0}{N_h}-\beta_m\frac{(I_r+I_b)I_mS_m^0}{N_h^2}.$$

To analyze the local stability of the disease-free equilibrium E_0 , the Jacobian matrix is evaluated at this point. Since all infected compartments are zero at the disease-free equilibrium, the transmission terms simplify accordingly. Substituting these values yields

$$J(E_0)=\begin{pmatrix} -\mu_h & 0 & 0 & 0 & 0 & 0 & \psi & 0 & 0 & -\beta_h\frac{S_h^0}{N_m} \\ 0 & -(\theta_h+\mu_h) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \beta_h\frac{S_h^0}{N_m} \\ 0 & \theta_h & -a_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \kappa & -a_2 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_h & \tau_{hb} & -a_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \tau_m & \tau_{mb} & \phi & -a_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \gamma_h & \gamma_m & -(\mu_h+\psi) & 0 & 0 & 0 \\ 0 & 0 & -\beta_m\frac{S_m^0}{N_h} & -\beta_m\frac{S_m^0}{N_h} & 0 & 0 & 0 & -\mu_m & 0 & 0 \\ 0 & 0 & \beta_m\frac{S_m^0}{N_h} & \beta_m\frac{S_m^0}{N_h} & 0 & 0 & 0 & \lambda_m & -(\theta_m+\mu_m) & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \theta_m & -\mu_m \end{pmatrix} \quad (19)$$

The eigenvalues of the system are obtained from the characteristic equation $det(J(E_0)-rI)=0$. After simplification, the characteristic equation can be expressed in the factorized form

$$|J(E_0)-rI|=(-\mu_h-r)(-a_3-r)(-a_4-r)(-(\mu_h+\psi)-r)(-\mu_m-r)(r^5+A_1r^4+A_2r^3+A_3r^2+A_4r+A_5).$$

According to the Routh–Hurwitz criterion, the equilibrium point E_0 is locally asymptotically stable if all coefficients $A_i>0$ and the associated Hurwitz determinants are satisfied. By applying the Routh–Hurwitz criterion, it follows that these conditions hold whenever $R_0<1$. Therefore, the disease-free equilibrium is locally asymptotically stable when the transmission potential of malaria remains below the epidemic threshold.

For the endemic equilibrium E^*, deriving explicit analytical stability conditions is mathematically challenging due to the high dimensionality and strong nonlinear coupling of the proposed system. Consequently, the local stability behavior of the endemic equilibrium is investigated numerically rather than analytically. Numerical simulations under the condition $R_0>1$ consistently show that the trajectories of all state variables converge toward positive constant values, indicating convergence toward a stable endemic state. These numerical findings suggest the local asymptotic stability of the endemic equilibrium and confirm the persistence of malaria transmission within the population under endemic conditions.

Sensitivity Analysis of R_0

Sensitivity analysis was conducted to identify the parameters that exert the greatest influence on malaria transmission dynamics. Following the reviewer's suggestion, the analysis was

extended to evaluate not only the basic reproduction number (R_0), but also the endemic prevalence represented by the equilibrium value of the mildly infected compartment (I_r^*). This extension is important because, once malaria becomes endemic, the objective of intervention shifts from reducing the outbreak threshold to suppressing the long-term number of infectious individuals within the population. The normalized forward sensitivity index of R_0 with respect to a parameter p is defined as $\Upsilon_p^{R_0} = \frac{\partial u}{\partial p} \times \frac{p}{u}$. The calculated sensitivity indices for both R_0 and I_r^* are presented in Table 2.

The results show that the transmission parameters (β_h, β_m) have the largest positive sensitivity indices for both R_0 and endemic prevalence, indicating that increased human–mosquito transmission substantially increases malaria burden. In contrast, the mosquito mortality rate (μ_m) exhibits the strongest negative sensitivity index, confirming that vector control remains one of the most effective intervention strategies. Among the treatment-related parameters, the herbal treatment rate (τ_h) produces a strong negative sensitivity index for both R_0 and I_r^*, demonstrating the importance of early-stage treatment accessibility in suppressing malaria transmission. Meanwhile, the progression parameter (κ) shows a positive sensitivity effect on endemic prevalence, indicating that faster progression toward severe infection increases long-term disease burden. Interestingly, the transition parameter (ϕ), representing the movement from herbal treatment to formal medical treatment, has no direct influence on R_0 and only a negligible direct sensitivity effect on I_r^*.

Table 2. Sensitivity Index

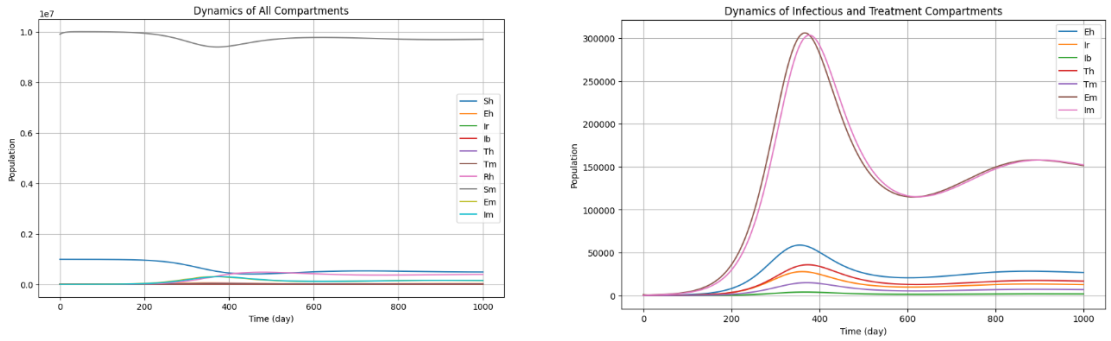
Parameter	Parameter Value	$(\Upsilon_p^{R_0})$	$\Upsilon_p^{I_r^*}$	Impact on R_0
β_h	0.3	$+0.500$	$+35.377$	Strong increase
β_m	0.2	$+0.500$	$+34.634$	Strong increase
θ_h	0.083	$+0.000$	-3.739	Moderate decrease
θ_m	0.1	$+0.250$	$+15.404$	Moderate increase
μ_h	4.6×10^{-5}	-0.000	-0.046	Very low decrease
μ_m	0.1	-0.750	-38.550	Strong decrease
κ	0.025	-0.038	$+2.382$	Increase in severe infection burden
τ_h	0.4	-0.421	-23.128	Strong decrease
τ_{hb}	0.06	-0.023	-1.501	Mild decrease
τ_m	0.05	-0.053	-3.248	Moderate decrease
τ_{mb}	0.10	-0.038	-2.484	Moderate decrease
ϕ	0.2	0.000	-0.0002	Negligible effect

Nevertheless, numerical simulations show that this parameter remains epidemiologically important because it governs the redistribution of infected individuals between treatment pathways. Overall, the sensitivity analysis demonstrates that malaria control strategies should focus not only on reducing the transmission threshold $(R_0 < 1)$, but also on suppressing endemic prevalence through vector control, early treatment accessibility, and efficient integration between herbal and medical treatment pathways.

Numerical Simulation

Numerical simulations were performed in Python using the odeint solver from the SciPy library, which is based on the LSODA algorithm with adaptive step-size control. The simulations were conducted to validate the analytical findings and to investigate the combined effects of the herbal treatment rate (τ_h) and the transition rate from herbal treatment to medical treatment (ϕ) on malaria transmission dynamics. The simulations used the initial population vector $y_0 = (9.9 \times 10^5,\ 100,\ 50,\ 10,\ 0,\ 0,\ 0,\ 9.9 \times 10^6,\ 1000,\ 100)$ with parameter values listed in Table 1. Simulation outputs were evaluated over the interval $t \in [0,1000]$ using 1000 evaluation points. In contrast to the previous simulation framework that only varied τ_h, the revised simulations additionally incorporate variations in ϕ to explicitly capture the epidemiological role

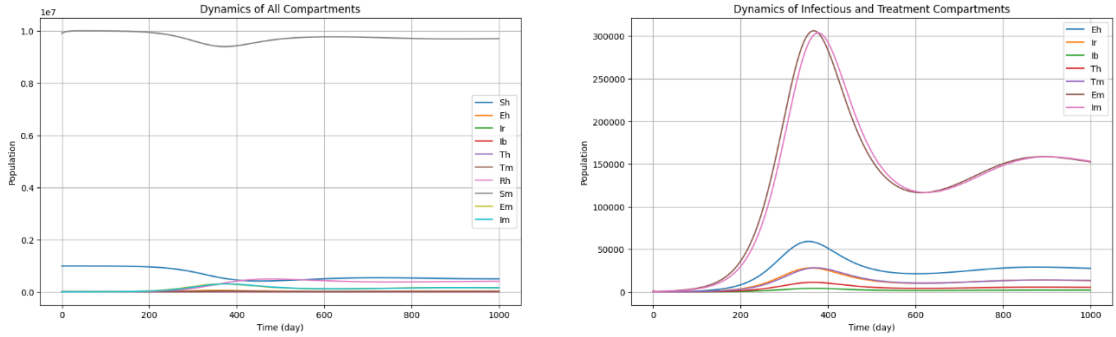
of treatment transition pathways. In this context, ϕ represents the transition mechanism from herbal treatment to formal medical treatment, reflecting referral or escalation processes within malaria healthcare systems.



(a) (b)

Figure 2. Numerical simulation for Case 1 ($\tau_h = 0.1,\ \phi = 0.01$). (a) Dynamics of All Compartments, (b) Dynamics of Infectious and Treatment Compartments.

In Case 1, the parameters were set as $\tau_h = 0.1$ and $\phi = 0.01$, representing limited early treatment access combined with a weak transition from herbal treatment to formal medical treatment. The simulation trajectories converge toward a stable endemic equilibrium characterized by persistent infection in both human and mosquito populations. The equilibrium values show a relatively high level of mild infection ($I_r \approx 12{,}814$), accompanied by a large herbal treatment population ($T_h \approx 16{,}834$). As illustrated in Figure 2(b), the infectious compartments initially increase during the transient outbreak phase before stabilizing at positive equilibrium values, confirming the persistence of endemic malaria transmission. These results indicate that low treatment accessibility together with inefficient referral mechanisms is insufficient to reduce long-term endemic transmission.



(a) (b)

Figure 3. Numerical simulation for Case 2 ($\tau_h = 0.1,\ \phi = 0.2$). (a) Dynamics of All Compartments, (b) Dynamics of Infectious and Treatment Compartments.

In Case 2, the herbal treatment rate remained fixed at $\tau_h = 0.1$, while the transition rate was increased to $\phi = 0.2$. Although the system still converges toward an endemic equilibrium ($R_0 > 1$), a substantial redistribution between treatment compartments is observed. The herbal treatment population decreases from $T_h \approx 16{,}834$ in Case 1 to $T_h \approx 5{,}248$, whereas the medical treatment population increases from $T_m \approx 7{,}013$ to $T_m \approx 13{,}356$. This result indicates that increasing ϕ accelerates the transition of infected individuals from herbal treatment to formal medical treatment. Although endemic transmission persists, the higher transition rate improves treatment allocation efficiency and reduces prolonged dependence on herbal

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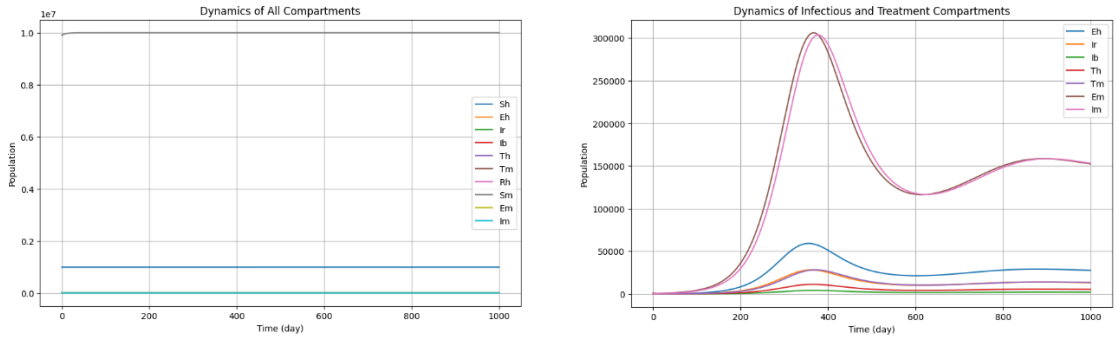
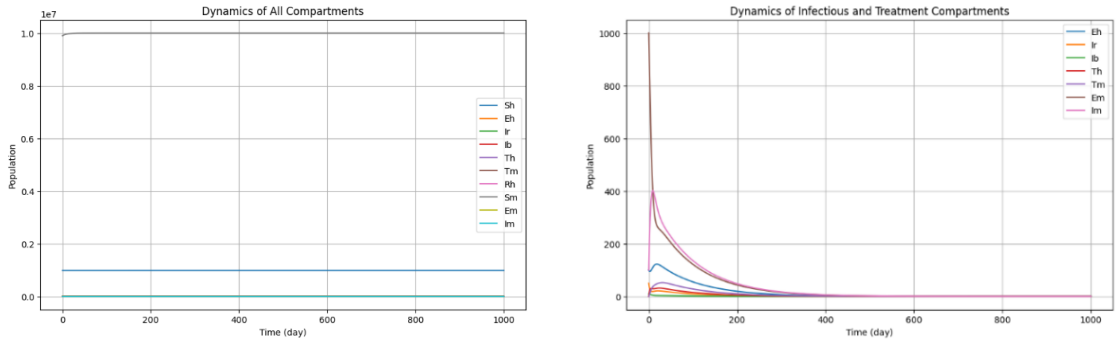
treatment. The reduction of approximately 68% in the herbal treatment compartment compared with Case 1 highlights the importance of referral mechanisms in improving malaria management under endemic conditions.



(a) (b)

Figure 4. Numerical simulation for Case 3 $(\tau_h = 0.4,\ \phi = 0.01)$. (a) Dynamics of All Compartments, (b) Dynamics of Infectious and Treatment Compartments.

In Case 3, the herbal treatment rate was increased to $\tau_h = 0.4$ while maintaining a low transition rate $\phi = 0.01$. The simulation results show that all infectious compartments decline asymptotically toward zero, indicating convergence to the disease-free equilibrium. At the end of the simulation period, the infected compartments satisfy $I_r \approx 10^{-3}$, $I_b \approx 10^{-4}$, $I_m \approx 10^{-2}$, confirming the numerical stability of the disease-free equilibrium. As illustrated in Figure 4(b), the infected human and mosquito populations rapidly decrease after a short transient phase before approaching zero. These numerical findings are consistent with the analytical results, where $R_0 < 1$ guarantees the local asymptotic stability of the disease-free equilibrium. Epidemiologically, the results indicate that increasing early-stage treatment access through herbal treatment substantially reduces malaria transmission and drives the system toward disease elimination.



(a) (b)

Figure 5. Numerical simulation for Case 4 $(\tau_h = 0.4,\ \phi = 0.2)$. (a) Dynamics of All Compartments, (b) Dynamics of Infectious and Treatment Compartments.

Case 4 represents the most effective intervention scenario, where both herbal treatment accessibility and the transition toward formal medical treatment are simultaneously strengthened $(\tau_h = 0.4,\ \phi = 0.2)$. Under this configuration, the system converges toward the disease-free equilibrium, with all infectious compartments approaching values close to zero. Compared with Case 3, the increased transition rate (ϕ) improves the redistribution of infected individuals from herbal treatment into formal medical treatment pathways, resulting in a more efficient elimination process. The herbal treatment compartment becomes substantially

smaller, indicating that individuals requiring further care are transferred more rapidly into medical treatment. These results suggest a synergistic interaction between early treatment accessibility and effective referral mechanisms. While the herbal treatment rate (τ_h) primarily determines the transition from endemic persistence to disease elimination, the transition parameter (ϕ) enhances the efficiency and robustness of the elimination dynamics.

Overall, the simulations demonstrate that the proposed dual treatment pathway model captures not only the threshold behavior associated with R_0, but also the influence of treatment-transition mechanisms on intervention effectiveness. The findings indicate that sustainable malaria elimination strategies should integrate both rapid early-stage treatment access and efficient referral systems toward formal medical care.

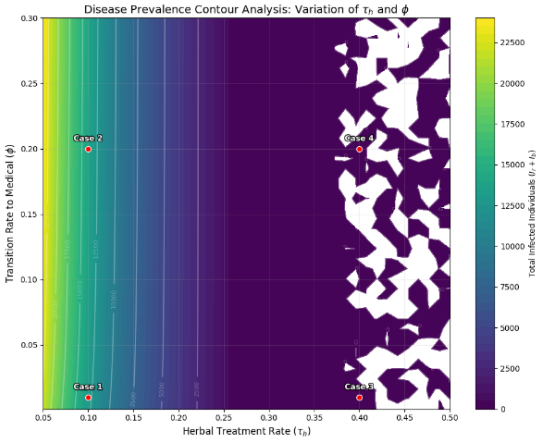


Figure 6. Contour plot of the total infected population $(I_r + I_b)$ under variations of (τ_h) and (ϕ). Darker regions indicate lower endemic prevalence.

The contour analysis reveals that the transition parameter (ϕ) plays an important role in improving treatment pathway efficiency under endemic conditions. In particular, the transition from Case 1 to Case 2 shows that increasing ϕ while maintaining a low herbal treatment rate $(\tau_h = 0.1)$ substantially reduces the number of individuals remaining in the herbal treatment compartment (τ_h) and increases the population receiving medical treatment (T_m). Epidemiologically, this finding suggests that stronger referral mechanisms can improve treatment allocation and reduce prolonged dependence on herbal treatment, even when early treatment access remains limited. In contrast, the transition from Case 1 to Case 3 demonstrates that the herbal treatment rate (τ_h) is the dominant factor controlling disease elimination. As τ_h increases from 0.1 to 0.4, the endemic prevalence decreases significantly and approaches the disease-free region $(R_0 < 1)$. This result confirms that early-stage treatment accessibility is a critical determinant in suppressing malaria transmission.

Furthermore, the region surrounding Case 4 represents the optimal intervention zone, where high treatment accessibility and efficient referral mechanisms act synergistically to produce the lowest endemic prevalence. Overall, the contour analysis confirms that the proposed model captures both the threshold behavior associated with R_0 and the redistribution dynamics between alternative treatment pathways. These findings highlight the importance of integrating treatment accessibility with effective referral systems in malaria control strategies.

CONCLUSION

The proposed malaria transmission model successfully captures the interaction between infection severity levels and dual treatment pathways through the incorporation of herbal treatment, formal medical treatment, and treatment-transition mechanisms. The analytical

results show that the disease-free equilibrium is locally asymptotically stable when $R_0 < 1$ whereas endemic persistence occurs when $R_0 > 1$. Numerical simulations further confirm the long-term stability behavior of the system under various intervention scenarios.

The sensitivity and numerical analyses indicate that malaria intervention effectiveness should be evaluated from two complementary epidemiological perspectives, namely reducing the epidemic threshold and suppressing endemic prevalence. The herbal treatment rate (τ_h) strongly reduces both R_0 and endemic infection prevalence, highlighting its important role in limiting transmission and driving the system toward disease elimination. In contrast, the treatment-transition parameter (ϕ) and medical recovery parameter (γ_m) have negligible effects on R_0, but significantly reduce endemic prevalence, particularly I_r^*. These findings suggest that treatment-transition mechanisms may not determine the initial outbreak occurrence, but they play a critical role in long-term disease suppression once malaria becomes endemic.

Furthermore, the simulation scenarios and contour analysis reveal a synergistic interaction between early herbal treatment access and referral efficiency toward formal medical care. Increasing (τ_h) primarily reduces transmission intensity, whereas increasing (ϕ) improves the transfer of infected individuals into more effective medical treatment pathways, thereby lowering endemic prevalence. This confirms that effective malaria control depends not only on expanding treatment coverage, but also on strengthening the integration between traditional and formal healthcare systems.

Therefore, the proposed model provides new epidemiological insights into how treatment-seeking behavior influences both outbreak dynamics and endemic persistence. From a public health perspective, the results suggest that sustainable malaria control strategies should integrate community-based herbal treatment access with efficient referral systems toward formal medical facilities, particularly in endemic regions where traditional treatment practices remain prevalent.

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Author could express a gratitude to their colleagues here. For reference list, we encourage author to prioritize looking for primary sources first. We strongly suggest author to use referencing tool to make sure the citations and references are one-to-one corresponding.

DECLARATION

Author contribution

All authors contribute in the research and/or writing the paper, and approved the final manuscript.

Panca Dewi Fitriyana	Conceptualizing the research idea, leading the investigation, and setting up the methodology.
Budi Priyo Prawoto	Assisting the investigation, reviewing the validity of the methodology, analyzing the data, and writing the original draft.

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Conflict of interest

All authors declare that they have no competing interests.

Ethics declaration

We as authors acknowledge that this work has been written based on ethical research that conforms with the regulations of our institutions and that we have obtained the permission from the relevant institutes when collecting data. We support the Bulletin of Applied Mathematics and Mathematics Education (BAMME) in maintaining the high standards of personal conduct, practicing honesty in all our professional practices and endeavors.

The use of artificial intelligence
We do not use any generative AI tools to write any part of this paper.

Additional information
Not available.

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