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Stability Analysis of a Mathematical Model of Endemic Malaria Transmission Dynamic

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Abstract
Malaria is a mosquito-borne disease that continues to affect people worldwide and remains a serious public health problem. It is caused by parasites from the Plasmodium genus and is transmitted through the bites of infected female Anopheles mosquitoes. In 2017, about 219 million malaria cases were recorded globally, with children accounting for a large number of related deaths. This study develops a mathematical model to understand how malaria spreads and how it can be controlled. The model examines important features such as the basic reproduction number and the conditions for disease-free and endemic states. It also investigates the occurrence of backward bifurcation using the center manifold theory to better understand how the disease behaves under certain conditions. The findings show that when specific conditions $(Z_1 - Z_2) > 0$ and $U_3 > 0$ are met, both A and B are positive, and the model shows backward bifurcation. In another case if $(Z_1 - Z_2) < 0$ and $U_3 > 0$, A is positive while B is negative, and the model still exhibits backward bifurcation. This means that malaria can remain in the population even when the basic reproduction number is less than one.

Keywords: Mathematical Model, Model Properties, Equilibrium Points, Basic Reproduction Number, Backward Bifurcation.

Introduction

Across many tropical and subtropical regions, one of the most persistent threats to human health is a vector-borne parasitic disease transmitted through the bite of infected female *Anopheles* mosquitoes. This disease, widely known as malaria, continues to impose a heavy burden on populations, particularly in developing countries where environmental and socio-economic conditions favor its transmission. The causative agents are protozoan parasites belonging to the genus *Plasmodium*. Although numerous species of this parasite exist and infect a variety of animal hosts, only a select few are responsible for infections in humans. These include *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium ovale*, and *Plasmodium malariae*. Among these species, *Plasmodium falciparum* stands out as the most virulent, often associated with severe complications and a higher risk of mortality if prompt and effective treatment is not administered. In rare instances, zoonotic transmission involving species such as *Plasmodium knowlesi* may occur, though such infections are typically isolated and do not sustain continuous transmission within human populations (Word Heath Organization, 2019).

Notwithstanding the availability of preventive and therapeutic interventions, the disease remains a leading cause of morbidity and mortality globally. The distribution of cases is heavily skewed toward sub-Saharan Africa, where a combination of climatic suitability, vector abundance, and limited access to healthcare services sustains high transmission intensity. Epidemiological records have consistently reported millions of cases annually, with children under five years of age constituting the most vulnerable group. For instance, global estimates indicated that

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approximately 219 million cases were recorded in 2017 alone, with the overwhelming majority occurring within the African region. This persistent prevalence highlights the enduring challenge posed by the disease despite ongoing control efforts (Word Heath Organization, 2019).

To better understand the mechanisms underlying disease transmission and to design effective intervention strategies, mathematical modeling has become an indispensable tool. Such models provide a systematic framework for representing the interactions between human hosts, mosquito vectors, and environmental factors. As global efforts intensify toward disease control and eventual elimination, the role of mathematical models has expanded, particularly in forecasting transmission trends and evaluating the potential impact of various intervention measures (Mandal et al., 2021). The origins of mathematical modeling in this field can be traced to the pioneering work of Sir Ronald Ross, whose early twentieth-century contributions laid the groundwork for modern epidemiological analysis (Ross, 1916; Mushtaq, 2009). Since then, a wide range of models have been developed to explore different dimensions of disease dynamics and inform public health policies (World Health Organization, 2010; McKenzie, 2000).

In contemporary research, deterministic modeling approaches have been widely employed to investigate transmission patterns and assess the effectiveness of control strategies (Chiyaka et al., 2021). For example, Mohammed and Orupke (2019) formulated a model to examine how variations in transmission parameters influence infection dynamics. Their findings emphasized that reducing the probability of mosquito infection can significantly decrease disease prevalence. However, the model did not incorporate key real-world factors such as population awareness or the presence of protected individuals, thereby limiting its applicability. In another contribution, Temesgen et al. (2022) integrated climate variability into a malaria transmission framework and applied optimal control theory to evaluate intervention strategies, including the use of insecticide-treated bed nets, treatment of infected individuals, and indoor residual spraying. Their analysis suggested that combining treatment with bed net usage offers a cost-effective approach to disease control. Nevertheless, the assumption that recovered individuals immediately return to a fully susceptible state without temporary immunity may not accurately reflect biological realities.

Similarly, Onah et al. (2019) explored the dynamics of malaria transmission with particular emphasis on environmental control measures such as reducing mosquito breeding sites and increasing insecticide application. Their results indicated that these interventions could substantially reduce transmission rates. However, the model did not account for reinfection among recovered individuals, which is a critical aspect of malaria epidemiology. Malik et al. (2019) also examined the influence of public awareness on disease transmission, concluding that informed individuals are less likely to be exposed to mosquito bites. While this underscores the importance of awareness campaigns, the assumption that awareness alone can effectively prevent infection oversimplifies the complexity of human behavior and environmental exposure.

Despite the variety of intervention strategies proposed in the literature, the disease continues to persist at high levels, particularly in regions with limited healthcare infrastructure. This persistence suggests that existing models may not fully capture the complexity of transmission dynamics. Consequently, there is a clear need for more comprehensive modeling approaches that incorporate additional real-world factors such as partial immunity, behavioral responses, and environmental variability. Against this backdrop, the present study is designed to develop and analyses an improved mathematical model that offers deeper insight into transmission dynamics while identifying more effective strategies for reducing the spread of the disease within affected populations.

Methodology

Model Development

The model divides the total population into humans and mosquitoes, where the human population is given by

$$N_h(t) = P_h + S_h + E_h + I_h + T_h + L_h + R_h + A_h$$

and the mosquito population is

$$N_m(t) = S_m + E_m + I_m.$$

The human population consists of protected (P_h), susceptible (S_h), exposed (E_h), infected (I_h), treated (T_h),

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untreated (L_h), recovered (R_h), and intervention (A_h)classes, while mosquitoes are grouped into susceptible (S_m), exposed (E_m), and infected (I_m). New humans enter the system at rate ω, with a fraction γentering the protected class and the rest joining the susceptible class. Susceptible individuals become exposed at rate $\tau_h$after contact with infectious mosquitoes, and some move to the intervention class at rate d. Exposed individuals progress to infection at rate α_h, while others move to the intervention class at rate $\alpha_1(1-y_2)$, depending on intervention effectiveness. Infected individuals receive treatment at rate ϕor remain untreated at rate κ. Treated individuals recover at rate σ, while untreated individuals recover at rate φ, and all recovered individuals enter the recovered class. Recovered individuals may adopt intervention measures at rate β. Individuals in the intervention class may return to the susceptible class at rate ηor become fully protected at rate $\delta(1-\eta)$. All human classes experience natural death at rate μ_h, while infected individuals also face disease-induced death at rate θ.

For the mosquito population, new mosquitoes enter the susceptible class at rate ω_m. Susceptible mosquitoes become exposed at rate $\tau_m$after biting infected humans, and exposed mosquitoes become infectious at rate α_m. All mosquito classes die naturally at rate μ_m. The model assumes homogeneous mixing, constant recruitment and death rates, and malaria transmission through human–mosquito interaction, while intervention strategies reduce infection risk but may weaken over time.

Model Assumptions

Model assumptions are as follows**:**

- Considers the population of those who are aware of malaria through campaign as a separate compartment.
- The entire recovered human population proceed to access malaria intervention after recovery.
- Mosquito never recover from infection, that is, the vector dies after infecting human.
- Having accessed malaria intervention; people will take all necessary precaution, such as cleaning environment, spray of insecticide, use of mosquito net and all other form of precaution against mosquito bite.
- Total human and mosquito populations are not constant.
- All parameters in the model being nonnegative.
- No recovered human move to susceptible class.

Model Equations

$$\frac{dP_h}{dt} = \gamma\omega_h + \delta(1-\eta)A_h - (\lambda(1-y)+\mu_h)P_h \quad (1)$$

$$\frac{dS_h}{dt} = (1-\gamma)\omega_h + \eta A_h + \lambda(1-y)P_h - \tau_h S_h - (\mu_h + d(1-y_1))S_h \quad (2)$$

$$\frac{dE_h}{dt} = \tau_h S_h - \alpha_h E_h - (\alpha_1(1-y_2)+\mu_h)E_h \quad (3)$$

$$\frac{dI_h}{dt} = \alpha_h E_h - (\phi + \kappa + \mu_h + \theta)I_h \quad (4)$$

$$\frac{dT_h}{dt} = \phi I_h - (\sigma + \mu_h)T_h \quad (5)$$

$$\frac{dL_h}{dt} = \kappa I_h - (\varphi + \mu_h)L_h \quad (6)$$

$$\frac{dR_h}{dt} = \sigma T_h + \varphi L - (\beta + \mu_h)R_h \quad (7)$$

$$\frac{dA_h}{dt} = \nu + \beta R_h + \alpha_1(1-y_2)E_h + d(1-y_1)S_h - (\delta(1-\eta)+\eta+\mu_h)A_h \quad (8)$$

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$$\frac{dS_m}{dt} = \omega_m - \tau_m S_m - \mu_m S_m \quad (9)$$

$$\frac{dE_m}{dt} = \tau_m S_m - \alpha_m E_m - \mu_m E_m \quad (10)$$

$$\frac{dI_m}{dt} = \alpha_m E_m - \mu_m I_m \quad (11)$$

Together with initial conditions

$$\left\{\begin{matrix} P_h(0) > P_{h0}\ S_h(0) > S_{h0}\ E_h(0) > E_{h0}\ \ I_h(0) > I_{h0}\ \ T_h(0) > T_{h0}\ \ L_h(0) > L_{h0} \\ A_h(0) > A_{h0}\ \ R_h(0) > R_{h0}\ \ S_m(0) > S_{m0}\ \ \ E_m(0) > E_{m0}\ \ \ I_m(0) > I_{m0} \end{matrix}\right\} \quad (12)$$

Where $N_h = P_H + S_h + E_h + I_h + T_h + L_h + R_h + A_h$ is the total population of host (humans) and $N_m = S_m + E_m + I_m$ is the total population of vector (mosquitoes). $\tau_h = \frac{b\rho_h I_m}{N_h}$ Is the force of infection of host and $\tau_m = \frac{b\rho_m I_h}{N_m}$ is the force infection of vector.

Table 1: Definitions of System Variables

Variable	Description
P_h	Number of individuals in the protected human class at time t.
S_h	Number of individuals in the susceptible human class at time t.
E_h	Number of individuals in the exposed human class at time t.
I_h	Number of individuals in the infected human class at time t.
T_h	Number of individuals receiving treatment at time t.
L_h	Number of infected individuals not receiving medical treatment (untreated class) at time t.
R_h	Number of recovered individuals at time t.
A_h	Number of individuals who have accessed malaria intervention programmes at time t.
S_m	Number of susceptible mosquitoes at time t.
E_m	Number of exposed (non-infectious) mosquitoes at time t.
I_m	Number of infectious mosquitoes at time t.

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Table 2: Summary of Model Parameters

Parameter	Operational Description
ω	Recruitment rate of individuals into the human population.
γ	Proportion of newly recruited individuals entering the protected class.
λ	Rate at which protected individuals lose immunity and become susceptible.
μ_h	Natural mortality rate of humans.
b	Mosquito biting rate on humans.
ρ_h	Probability of disease transmission from an infectious mosquito to a human per bite.
α_h	Rate at which exposed humans progress to the infectious stage.
α_1	Rate at which exposed individuals adopt malaria intervention measures.
ϕ	Rate at which infected individuals receive medical treatment.
κ	Rate at which infected individuals opt for non-medical (untreated) pathways.
φ	Recovery rate of untreated infected individuals.
β	Rate at which recovered individuals move into the intervention/awareness class.
η	Rate at which susceptible individuals adopt awareness or preventive measures.
d	Rate at which individuals lose awareness and return to the susceptible class.
δ	Rate at which individuals acquire protection through immunity or preventive measures.
θ	Disease-induced mortality rate due to malaria.
σ	Recovery rate of individuals under clinical treatment.
α_m	Rate at which exposed mosquitoes become infectious.
ρ_m	Probability of transmission from an infectious human to a mosquito per bite.
υ	Rate of implementation of intervention strategies.
μ_m	Natural mortality rate of mosquitoes.
ω_m	Recruitment rate of mosquitoes into the population.
y	Control parameter associated with the protected human class.
y_1	Control parameter representing awareness/intervention among susceptible individuals.
y_2	Control parameter representing awareness/intervention among exposed individuals.

Fundamental Model Properties

Invariant Region Analysis

The invariant region is used to show that all solutions of the model remain within a realistic biological range over time. To establish this, the system of equations (1–11) is divided into two groups: the human population and the mosquito population. The total human population at time t, denoted by N_h, is obtained by summing all eight human compartments, given by

$$N_h = P_h + S_h + E_h + I_h + T_h + L_h + R_h + A_h,$$

while the total mosquito population, denoted by N_m, is the sum of all mosquito compartments, expressed as

$$N_m = S_m + E_m + I_m.$$

These total population equations help define a bounded region where the model remains biologically meaningful, ensuring that population sizes do not become negative or grow without limit over time.

Theorem 1: let $\Omega_h = \left\{ P_h, S_h, E_h, I_h, T_h, L_h, R_h, A_h \in \Re_+^8 : N_h(t) \le \frac{\omega_h}{\mu_h} \right\}$ and

$\Omega_m = \left\{ S_m, E_m, I_m \in \Re_+^3 : N_m(t) \le \frac{\omega_m}{\mu_m} \right\}$ so that $\Omega = \Omega_h * \Omega_m \subset \Re_+^8 * \Re_+^3$. The biologically feasible region of the Ω of the model equation (1) to (11) is positively invariant.

Proof:

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From the total human population represented by $N_h = P_H + S_h + E_h + I_h + T_h + L_h + R_h + A_h$,
It is clear that,

$$\frac{dN_h}{dt} \leq \omega_h - \mu_h N_h \quad (13)$$

Applying separation of and integrating equation (13), we have,

$$\omega_h - \mu_h N_h \leq D_1 \ell^{-\mu_h t} \quad (14)$$

At $t = 0,\ N_h(t) = N_0$ equation (14) becomes

$$\omega_h - \mu_h N_h = D_1$$

Therefore, equation (14) becomes

$$\omega_h - \mu_h N_h \leq (\omega_h - \mu_h N_h)\ell^{-\mu_h t}$$

$$N_h \leq \frac{\omega_h}{\mu_h} - \frac{(\omega_h - \mu_h N_h)\ell^{-\mu_h t}}{\mu_h} \quad (15)$$

If $t \to \infty$ equation (15) becomes

$$N_h(t) \leq \frac{\omega_h}{\mu_h} \quad (16)$$

This implies that $0 \leq N_h(t) \leq \frac{\omega_h}{\mu_h}$.

For the human population, the feasible solutions lie within a bounded region that ensures biological realism. This region is defined as

$\Omega_h = \left\{(P_h, S_h, E_h, I_h, T_h, L_h, R_h, A_h) \in \mathbb{R}_+^8 : 0 \leq N_h(t) \leq \frac{\omega}{\mu_h}\right\}$.

Using the same approach for the mosquito population and applying the model equations, we obtain the inequality

$\frac{dN_m}{dt} \leq \omega_m - \mu_m N_m,$ (17)

which shows that mosquito growth is naturally limited. Solving this inequality gives

$N_m(t) \leq \frac{\omega_m}{\mu_m}.$ (18)

As time increases $(t \to \infty)$, the mosquito population approaches the steady level $\frac{\omega_m}{\mu_m}$. Hence, the mosquito population is confined within the region

$\Omega_m = \left\{(S_m, E_m, I_m) \in \mathbb{R}_+^3 : 0 \leq N_m(t) \leq \frac{\omega_m}{\mu_m}\right\}$.

Combining both populations, the full solution space is given by $\Omega = \Omega_h \times \Omega_m$. This region is positively invariant, meaning that any solution that starts within it will remain there for all time. This confirms that the model is both mathematically valid and biologically meaningful.

Disease-Free Equilibrium Point

The Disease-Free Equilibrium (DFE) represents a steady state where malaria does not exist in either the human or mosquito population. To determine this state, all derivatives in the system (equations 1–11) are set to zero, that is,

$\frac{dP_h}{dt} = \frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dI_h}{dt} = \frac{dT_h}{dt} = \frac{dL_h}{dt} = \frac{dR_h}{dt} = \frac{dA_h}{dt} = \frac{dS_m}{dt} = \frac{dE_m}{dt} = \frac{dI_m}{dt} = 0.$ (19)

At this point, all infection-related compartments become zero, meaning there are no exposed or infected individuals in

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both populations. The model then reduces to a simpler system that describes only the non-infected classes. For example, one of the resulting equilibrium conditions is given by

$$\gamma\omega + \delta(1-\eta)A_h - (\lambda(1-y) + \mu_h)P_h = 0, \tag{20}$$

which helps determine the steady-state value of the protected population. This equilibrium describes a situation where no new infections occur, and the system remains stable in the absence of the disease.

$$(1-\gamma)\omega_h + \eta A + \lambda(1-y)P - \tau_h S_h - (\mu_h + d(1-y_1))S_h = 0 \tag{21}$$

$$\omega_m - \tau_m S_m - \mu_m S_m = 0 \tag{22}$$

Solving the equations (20) to (22) for P_h, S_h and S_m, we have,

$$P_m = \frac{\gamma\omega_h}{\lambda(1-y)+\mu_h},\ S_h = \frac{(\lambda(1-y)+(1-\gamma)\mu_h)\omega_h}{(\mu_h+\lambda(1-y))(\mu_h+d(1-y_1))} \text{ and } S_m = \frac{\omega_m}{\mu_m}.$$

Hence, the malaria free equilibrium point E^0 is,

$$E_0 = \left(\frac{\gamma\omega_h}{\lambda(1-y)+\mu_h}, \frac{\omega_h(\lambda(1-y)+(1-\gamma)\mu_h)}{\mu_h(\mu_h+\lambda(1-y))}, 0,0,0,0,0,0, \frac{\omega_m}{\mu_m}, 0, 0\right) \tag{23}$$

Basic Reproduction Number (R_0)

A key epidemiological threshold used to describe the transmission potential of malaria within a population is the basic reproduction number, denoted by R_0. This parameter represents the expected number of secondary infections produced by a single infected individual introduced into a completely susceptible population. It provides a fundamental criterion for determining whether an infectious disease will decline over time or persist within a community.

In epidemiological interpretation, the magnitude of R_0 plays a decisive role in disease dynamics. When $R_0 < 1$, an infected individual generates less than one secondary case on average, leading to a gradual decline in infection until eventual elimination. Conversely, when $R_0 > 1$, each infected individual produces more than one new infection, resulting in sustained transmission and the potential for an outbreak. Although simple compartmental models allow for direct computation of R_0 using transmission and recovery parameters, more complex systems involving multiple interacting compartments require a more structured analytical approach. In the present study, the Next-Generation Matrix (NGM) method is employed to determine R_0, in line with the procedures established by Diekmann et al. (1990) and van den Driessche and Watmough (2002). This method provides a systematic framework for deriving the reproduction number in compartmental models with multiple infected states. Within this framework, R_0 is defined as the spectral radius of the next-generation matrix, which is obtained from the decomposition of new infection terms and transition dynamics between infected compartments In that is:

$$R_0 = \rho(FV^{-1}), \tag{24}$$

where ρ is the spectral radius, which represents the largest absolute eigenvalue of the matrix. The matrix F describes the rate of new infections entering each infected compartment, while the matrix V represents the transfer of individuals between infected compartments, including recovery and death. These matrices are defined as

$$F = \left[\frac{\partial F_i}{\partial x_j}(E_0)\right], V = \left[\frac{\partial V_i}{\partial x_j}(E_0)\right], 1 \le i, j \le m,$$

where m is the number of infected compartments and E_0 is the disease-free equilibrium point. The matrix V can also be written as

$$V_i = V_i^-(x) - V_i^+(x),$$

where $V_i^-(x)$ represents the rate at which individuals leave compartment i, and $V_i^+(x)$ represents the rate at which individuals enter it. Using this approach, the value of R_0 can be calculated for the model, which helps to determine whether the disease-free state is stable and whether malaria can invade the human and mosquito populations. Therefore, the basic reproductive number for human and mosquito populations are:

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$$R_{0h} = \sqrt{\frac{\omega_h b\rho_h\left(\lambda(1-y)+(1-\gamma)\mu_h\right)\alpha_h}{\mu_h\left(\mu_h+\lambda(1-y)\right)\left(\alpha_h+\alpha_1(1-y_2)+\mu_h\right)\left(\phi+\kappa+\mu_h+\theta_h\right)}}$$

$$R_{0m} = \sqrt{\frac{b\rho_m\alpha_m}{(\alpha_m+\mu_m)\mu_m}}$$

Thus,

$$R_0 = \sqrt{\frac{\omega_h b\rho_h\left(\lambda(1-y)+(1-\gamma)\mu_h\right)\alpha_h b\rho_m\alpha_m}{\mu_h\left(\mu_h+\lambda(1-y)\right)\left(\alpha_h+\alpha_1+\mu_h\right)\left(\phi+\kappa+\mu_h+\theta_h\right)(\alpha_m+\mu_m)\mu_m}} \tag{25}$$

Analysis of the Disease-Free Equilibrium (DFE) Point, E_0

A state of the system in which no infection is present in either the human or mosquito populations is obtained by setting all exposed and infected compartments to zero and solving the remaining equations for the non-infected classes. This condition is referred to as the disease-free equilibrium, denoted by E_0. At this point, only the susceptible and other non-infectious compartments persist, while all infection-related variables vanish. The susceptible population stabilizes at a level determined by the model parameters. This equilibrium point is essential for understanding whether malaria can invade or be eliminated in the population. If the equilibrium is stable, any small introduction of infected individuals will decay over time, returning the system to a disease-free state. However, if it is unstable, the infection can persist and spread within the population.

The stability of this equilibrium is governed by the basic reproduction number, R_0. When $R_0 < 1$, the equilibrium is locally asymptotically stable, implying that malaria cannot establish itself in the population. Conversely, when $R_0 > 1$, the equilibrium becomes unstable, indicating the potential for sustained transmission. To investigate this further, the Jacobian matrix of the system (equations 1–11) is evaluated at E_0, following the approach of Abah et al. (2015). The eigenvalues of the resulting matrix are then used to determine the local stability behaviour of the system around the equilibrium point. This matrix is used to study the eigenvalues of the system, which determine the local stability of the equilibrium point, is given as:

$$JE^0 = \begin{bmatrix} a_{10} & 0 & 0 & 0 & 0 & 0 & 0 & a_{11} & 0 & 0 & 0 \\ 0 & a_{13} & 0 & 0 & 0 & 0 & 0 & a_{27} & 0 & 0 & a_{14} \\ 0 & 0 & a_{15} & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{16} \\ 0 & 0 & 0 & a_{17} & 0 & 0 & 0 & 0 & 0 & 0 & a_{29} \\ 0 & 0 & 0 & 0 & a_{18} & 0 & 0 & 0 & 0 & 0 & a_{31} \\ 0 & 0 & 0 & 0 & 0 & a_{19} & 0 & 0 & 0 & 0 & a_{32} \\ 0 & 0 & 0 & 0 & 0 & 0 & a_{20} & 0 & 0 & 0 & a_{36} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{30} & 0 & 0 & a_{37} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & -\mu_m & 0 & a_{33} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{26} & a_{34} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & a_{38} \end{bmatrix} \tag{26}$$

Where,

$$a_{10} = -\left(\mu_h+\lambda(1-y)\right),\quad a_{11} = \delta(1-\eta),\ a_{12} = \lambda(1-y)\ a_{13} = -\left(d\left(1-y_1+\mu_h\right)\right),$$

$$a_{14} = -\frac{b\rho_h\left(\lambda(1-y)+(1-\gamma)\mu_h\right)}{\left(\mu_h+\lambda(1-y)\right)}\quad a_{15} = -\left(\alpha_h+\alpha_1(1-y_2)+\mu_h\right)\quad a_{16} = \frac{b\rho_h\left(\lambda(1-y)+(1-\gamma)\mu_h\right)}{\left(\mu_h+\lambda(1-y)\right)},$$

$$a_{17} = -\left(\phi+\kappa+\mu_h+\theta\right),\quad a_{18} = -\left(\sigma+\mu_h\right),\quad a_{19} = -\left(\varphi+\mu_h\right),\ a_{20} - \left(\beta+\mu_h\right),\quad a_{21} = d(1-y_1),$$

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$$a_{22}=\alpha_1(1-y_2), a_{23}=-(\delta(1-\eta)+\mu_h),\ a_{24}=-b\rho_m,\ a_{25}=b\rho_m,\ a_{26}=-(\alpha_m+\mu_m)$$

$$a_{27}=\eta+\frac{\lambda\delta(1-y)(1-\eta)}{\lambda(1-y)+\mu_h},\ a_{29}=\frac{\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)}$$

$$a_{30}=-(\delta(1-\eta)+\eta+\mu_h)+\frac{d(1-y_1)}{(d(1-y_1+\mu_h))}\left(\eta+\frac{\lambda\delta(1-y)(1-\eta)}{\lambda(1-y)+\mu_h}\right),$$

$$a_{31}=\frac{\phi\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{32}=\frac{\kappa\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{33}=\frac{\alpha_h b^2\rho^2{}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{34}=-\frac{\alpha_h b^2\rho^2{}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{36}=\frac{\varphi\kappa\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\varphi+\mu_h)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)},$$

$$a_{37}=\left\{\begin{array}{l}\left(\frac{d(1-y_1)}{(d(1-y_1)+\mu_h)}+\frac{\varphi\kappa\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\varphi+\mu_h)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)}\right)\\ +\left(\left(\frac{\alpha_1(1y_2)}{(\alpha_h+\alpha_1(1-y_2)+\mu_h)}\right)\left(\frac{b\rho_h(\lambda(1-y)+(1-\gamma)\mu_h)}{(\mu_h+\lambda(1-y))}\right)\right)+\left(\frac{1}{(d(1-y_1)+\mu_h)(\phi+\kappa+\mu_h+\theta)}\right)*\\ \left(\frac{\beta\varphi\kappa\alpha_h b\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\beta+\mu_h)(\varphi+\mu_h)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)}\right)\end{array}\right\}$$

$$a_{38}=-\mu-\frac{\alpha_m\alpha_h b^2\rho^2{}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_m+\mu_m)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)}$$

Thus, the eigenvalues are:

$$\left\{\begin{array}{l}\lambda_1=-(\mu_h+\lambda(1-y))<0\\ \lambda_2=-(d(1-y_1+\mu_h))<0\\ \lambda_3=-(\alpha_h+\alpha_1(1-y_2)+\mu_h)<0\\ \lambda_4=-(\phi+\kappa+\mu_h+\theta)<0\\ \lambda_5=-(\sigma+\mu_h)<0\\ \lambda_6=-(\varphi+\mu_h)<0\\ \lambda_7=-(\beta+\mu_h)<0\\ \lambda_8=-(\delta(1-\eta)+\eta+\mu_h)+\frac{d(1-y_1)}{(d(1-y_1+\mu_h))}\left(\eta+\frac{\lambda\delta(1-y)(1-\eta)}{\lambda(1-y)+\mu_h}\right)<0\\ \lambda_9=-\mu_m,<0\\ \lambda_{10}=-(\alpha_m+\mu_m)<0\\ \lambda_{11}=-\left(\mu+\frac{\alpha_m\alpha_h b^2\rho^2{}_m(\lambda(1-y)+(1-\gamma)\mu_h)}{(\alpha_m+\mu_m)(\alpha_h+\alpha_1(1-y_2)+\mu_h)(\lambda(1-y)+\mu_h)(\phi+\kappa+\mu_h+\theta)}\right)<0\end{array}\right\} \quad (27)$$

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It is clear from equation (26) that all the eigenvalues are negative and less than zero. Therefore, the solution of model equation (1) to (11) is locally asymptotically stable.

Endemic Equilibrium Point (E_e)

The endemic equilibrium point, denoted by E_e, describes a steady state where malaria remains present in the population. At this point, at least one infected compartment has a positive value, meaning the disease does not disappear but continues to circulate among humans and mosquitoes. This equilibrium is important because it shows the long-term behavior of the disease when it becomes established. The stability of this state depends on the Basic Reproduction Number, R_0. When $R_0 > 1$, the endemic equilibrium is asymptotically stable, indicating that the disease will persist in the population. However, when $R_0 < 1$, the endemic equilibrium becomes unstable, and the infection gradually dies out.

To determine the endemic equilibrium, we consider the system at steady state by setting all time derivatives equal to zero, that is, $\frac{d}{dt} = 0$ for all model equations. This implies that the population sizes in all compartments remain constant over time. Solving the resulting system gives the endemic equilibrium point

$$E_e = (P_h^*, S_h^*, E_h^*, I_h^*, T_h^*, L_h^*, R_h^*, A_h^*, S_m^*, I_m^*, E_m^*),$$

where each variable with an asterisk represents its steady-state value. These values describe the level at which the disease persists in both the human and mosquito population

That is,

$$\gamma\omega_h + \delta(1-\eta)A - (\lambda(1-y) + \mu_h)P = 0 \tag{27}$$

$$(1-\gamma)\omega_h + \eta A_h + \lambda(1-y)P_h - \tau_h S_h - (\mu_h + d(1-y_1))S_h = 0 \tag{28}$$

$$\tau_h S_h - \alpha_h E_h - (\alpha_1(1-y_2) + \mu_h)E_h = 0 \tag{29}$$

$$\alpha_h E_h - (\phi + \kappa + \mu_h + \theta)I_h = 0 \tag{30}$$

$$\phi I_h - (\sigma + \mu_h)T_h = o \tag{31}$$

$$\kappa I_h - (\varphi + \mu_h)L_h = 0 \tag{32}$$

$$\sigma T_h + \varphi L_h - (\beta + \mu_h)R_h = 0 \tag{33}$$

$$\beta R_h + \alpha_1(1-y_2)E_h + d(1-y_1)S_h - (\delta(1-\eta) + \eta + \mu_h)A_h = 0 \tag{34}$$

$$\omega_m - \tau_m S_m - \mu_m S_m = 0 \tag{35}$$

$$\tau_m S_m - \alpha_m E_m - \mu_m E_m = 0 \tag{36}$$

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$$\alpha_m E_m - \mu_m I_m = 0 \tag{37}$$

Solving equations (27) to (37), we obtain the endemic equilibrium points as:

$$I_h^* = \frac{\mu_h^2(\mu_h + \lambda(1-y))(\delta(1-\eta)+\eta+\mu_h)R_0^2\omega_m M}{d(1-y_1)(\lambda(1-y)+(1-\gamma)\mu_h)\omega_h^2 b\rho_m} - \frac{(\lambda(1-y)+(1-\gamma)\mu_h)\omega_h^2\omega_m}{d(1-y_1)(\lambda(1-y)+(1-\gamma)\mu_h)\omega_h^2 b\rho_m}$$

$$P_h^* = \frac{\gamma\omega_h}{\mu_h+\lambda(1-y)} + \frac{\delta(1-\eta)M}{\mu_h+\lambda(1-y)} \qquad S_h^* = \frac{\omega_h^2(\lambda(1-y)+(1-\gamma)\mu_h)}{\mu_h^2(\mu_h+\lambda(1-y))R_0^2\omega_m}(b\rho_m F+\omega_m)$$

$$E_h^* = \frac{(\phi+\kappa+\mu_h+\theta)F}{\alpha_h}\ ',\ T_h^* = \frac{\phi F}{\sigma+\mu_h},\ R_h^* = \left(\frac{\sigma\phi}{\sigma+\mu_h}+\frac{\varphi\kappa}{\varphi+\mu_h}\right)\frac{F}{(\beta+\mu_h)}$$

$$S_m^* = \frac{\omega_m^2}{\mu_m(b\rho_m F+\omega_m)},\ E_m^* = \frac{R_{0m}^2 b\rho_m\omega_m^2 F}{(\alpha_m+\mu_m)(b\rho_m F+\omega_m)^2},\ I_m^* = \frac{R_{0m}^2\omega_m F}{b\rho_m F+\omega_m}$$

Where, $F = \frac{\mu_h^2(\mu_h+\lambda(1-y))(\delta(1-\eta)+\eta+\mu_h)R_0^2\omega_m M}{d(1-y_1)(\lambda(1-y)+(1-\gamma)\mu_h)\omega_h^2 b\rho_m} - \frac{(\lambda(1-y)+(1-\gamma)\mu_h)\omega_h^2\omega_m}{d(1-y_1)(\lambda(1-y)+(1-\gamma)\mu_h)\omega_h^2 b\rho_m}$

Backward Bifurcation Analysis

Backward bifurcation is an important concept in disease modeling because it shows that reducing the basic reproduction number, R_0, below one may not be enough to eliminate the disease. In this situation, two steady states can exist at the same time: a disease-free equilibrium and an endemic equilibrium where the infection persists. This means the disease can remain in the population even when $R_0 < 1$, making control strategies more difficult. In this study, the model equations (1–11) are examined to check whether this phenomenon occurs. The analysis focuses on parameter values, especially those related to transmission, around the critical point where $R_0 = 1$, to observe how the system changes. To confirm the presence of backward bifurcation, Centre Manifold Theory is applied. This method simplifies the system near the critical point and helps determine the stability and direction of the bifurcation. The process involves shifting the disease-free equilibrium to the origin, evaluating the Jacobian matrix at that point, and computing bifurcation coefficients following the approach of Castillo-Chavez and Song (2004). The signs of these coefficients indicate whether the bifurcation is forward or backward. A backward bifurcation is confirmed when a stable endemic state exists even when $R_0 < 1$.

To simplify the analysis, a change of variables is introduced. The model variables are redefined as

$$P_h = v_1,\ S_h = v_2,\ E_h = v_3,\ I_h = v_4,\ T_h = v_5,\ L_h = v_6,$$
$$R_h = v_7,\ A_h = v_8,\ S_m = v_9,\ E_m = v_{10},\ I_m = v_{11}.$$

Letting $V = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9, v_{10}, v_{11})^T$, the system of equations can be written in compact form as

$$\frac{dV}{dt} = f(v),$$

where $f = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9, f_{10}, f_{11})^T$. This transformation makes it easier to carry out the mathematical analysis needed to determine the presence of backward bifurcation.

Details are as follows:

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$$\left.\begin{aligned}
\frac{dV_{1h}}{dt} &= \gamma\omega_h + \delta(1-\eta)V_{8h} - (\lambda(1-y)+\mu_h)V_{1h}\\
\frac{dV_{21h}}{dt} &= (1-\gamma)\omega_h + \eta V_{8h} + \lambda(1-y)V_{1h} - \tau_h V_{1h} - (\mu_h + d(1-y_1))V_{2h}\\
\frac{dV_{4h}}{dt} &= \alpha_h V_{3h} - (\phi+\kappa+\mu_h+\theta)V_{4h}\\
\frac{dV_5}{dt} &= \phi V_{4h} - (\sigma+\mu_h)V_{5h}\\
\frac{dV_{6h}}{dt} &= \kappa V_{4h} - (\varphi+\mu_h)V_{6h}\\
\frac{dV_{7h}}{dt} &= \sigma V_{5h} + \varphi V_{6h} - (\beta+\mu_h)V_{7h}\\
\frac{dV_{8h}}{dt} &= \upsilon + \beta V_{7h} + \alpha_1(1-y_2)V_{3h} + d(1-y_1)V_{2h} - (\delta(1-\eta)+\eta+\mu_h)V_{8h}\\
\frac{dV_{9m}}{dt} &= \omega_m - \tau_m V_{9m} - \mu_m V_{9m}\\
\frac{dV_{10m}}{dt} &= \tau_m V_{9m} - \alpha_m V_{10m} - \mu_m V_{10m}\\
\frac{dV_{11m}}{dt} &= \alpha_m V_{10m} - \mu_m V_{11m}
\end{aligned}\right\} \quad (38)$$

We let the probability of biting by an infected mosquito which results in transmission of malaria parasite toa susceptible human $\rho_h = \rho_h^*$ be the bifurcation parameter and $R_0 = 1$ be the bifurcation point. Then, from equation (25), the bifurcation parameter

$$\rho_h = \rho_h^* = \frac{\mu_h(\mu_h+\lambda(1-y))(\alpha_h+\alpha_1+\mu_h)(\phi+\kappa+\mu_h+\theta)(\alpha_m+\mu_m)\mu_m}{b^2\omega_h\alpha_m\alpha_h\rho_m(\lambda(1-y)+(1-\gamma)\mu_h)}$$

Then, we evaluated the Jacobian matrix of equation (48) at the disease free equilibrium points E_0 and ρ_h^*. That is,

$$J(E_0.\rho_h^*) = \begin{bmatrix}
-C_1 & 0 & 0 & 0 & 0 & 0 & 0 & \delta(1-\eta) & 0 & 0 & 0\\
\lambda(1-Y) & -C_2 & 0 & 0 & 0 & 0 & 0 & \eta & 0 & 0 & -C_3\\
0 & 0 & -C_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & C_5\\
0 & 0 & \alpha_h & -C_6 & 0 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \phi & -C_7 & 0 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & \kappa & 0 & -C_8 & 0 & 0 & 0 & 0 & 0\\
0 & 0 & 0 & 0 & \sigma & \varphi & -C_9 & 0 & 0 & 0 & 0\\
0 & C_{10} & C_{11} & 0 & 0 & 0 & \beta & -C_{12} & 0 & 0 & 0\\
0 & 0 & 0 & -C_{13} & 0 & 0 & 0 & 0 & -C_{14} & 0 & 0\\
0 & 0 & 0 & C_{15} & 0 & 0 & 0 & 0 & 0 & -C_{16} & 0\\
0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & -\mu_m
\end{bmatrix} \quad (39)$$

where,

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$$C_1 = \left(\mu_h + \lambda(1-y)\right) \quad , \quad C_2 = \left(d(1-y_1+\mu_h)\right) \quad ,$$

$$C_3 = \frac{\mu_h\left(\mu_h+\lambda(1-y)\right)\left(\alpha_h+\alpha_1+\mu_h\right)\left(\phi+\kappa+\mu_h+\theta\right)\left(\alpha_m+\mu_m\right)\mu_m}{b^2\omega_h\alpha_m\alpha_h\rho_m\left(\lambda(1-y)+(1-\gamma)\mu_h\right)}, \; C_4 = \left(\alpha_h+\alpha_1(1-y_2)+\mu_h\right)$$

$$C_5 = \frac{\mu_h\left(\mu_h+\lambda(1-y)\right)\left(\alpha_h+\alpha_1+\mu_h\right)\left(\phi+\kappa+\mu_h+\theta\right)\left(\alpha_m+\mu_m\right)\mu_m}{b^2\omega_h\alpha_m\alpha_h\rho_m\left(\lambda(1-y)+(1-\gamma)\mu_h\right)}$$

$$C_6 = \left(\phi+\kappa+\mu_h+\theta\right), \; C_7 = \left(\sigma+\mu_h\right), C_8 = \left(\varphi+\mu_h\right) \; , \; C_9 = \left(\beta+\mu_h\right) \; , \; C_{10} = d(1-y_1)$$

$$C_{11} = \alpha_1(1-y_2) \; , C_{12} = \left(\delta(1-\eta)+\mu_h\right) \; , \; C_{13} = b\rho_m \; , \; C_{14} = \mu_m \, , \; C_{15} = b\rho_m \, , \; C_{16} = \left(\alpha_m+\mu_m\right)$$

From Equation (49), we observe at least one simple zero eigenvalue. This allows us to compute the corresponding right and left eigenvectors for this specific eigenvalue.

We consider the condition $\mathrm{U}\cdot\mathrm{J}\,|_{E_0} = 0$,

which implies that the row vector $\mathbf{U} = [U_1, U_2, \ldots, U_{11}]$, lies in the left null space of the Jacobian matrix evaluated at the disease-free equilibrium E_0. Thus,

Thus,

$$[U_1 \; U_2 \; \cdots \; U_{11}]\begin{pmatrix} -C_1 & 0 & \ldots & 0 \\ \lambda(1-\gamma) & -C_2 & \ldots & -C_3 \\ \vdots & \vdots & \ddots & \vdots \\ 0 & 0 & \ldots & -\mu_m \end{pmatrix} = \mathbf{0}. \tag{40}$$

Then, from matrix equation (40), we have

$$-U_1C_1 + U_2\lambda(1-Y) = 0 \tag{41}$$

$$-U_2C_2 + U_8C_{10} = 0 \tag{42}$$

$$-U_3C_4 + U_4\alpha_h + U_8C_{11} = 0 \tag{43}$$

$$-U_4C_6 + U_5\phi + U_6\kappa - U_9C_{13} + U_{10}C_{15} = 0 \tag{44}$$

$$-U_5C_7 + U_7\sigma = 0 \tag{45}$$

$$-U_6C_8 + U_7\varphi = 0 \tag{46}$$

$$-U_7C_9 + U_8\beta = 0 \tag{47}$$

$$U_1\delta(1-\eta) + U_2\eta - U_8C_{12} = 0 \tag{48}$$

$$-U_9C_{14} = 0 \tag{49}$$

$$-U_{10}C_{16} + U_{11}\alpha_m = 0 \tag{50}$$

$$-U_2C_3 + U_3C_5 - U_{11}\mu_m = 0 \tag{51}$$

Solving equations (41) to (51) and make U_3 free we obtained

$$U_1; U_2, \frac{C_4}{\alpha_h}U_3 \, , \, U_5, \, U_6, \, U_7, \, U_8, \, U_9 = 0, \; U_{10} = \frac{C_5}{\mu_m}U_3, \; U_{11} = \frac{C_5\alpha_m}{C_{16}\mu_m}U_3$$

Therefore,

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$$U = \left(0,\ 0, U_3, \frac{C_4}{\alpha_h}U_3, 0, 0, 0, 0, 0, \frac{C_5}{\mu_m}U_3, \frac{C_5\alpha_m}{C_{16}\mu_m}U_3 \right)$$

In the same way, we compute $J\left(E_0\rho_h^*\right)M^T = 0$

To determine the right eigenvector, a vector **M**consisting of eleven components is introduced. This vector represents the non-trivial solution of the homogeneous system obtained when the Jacobian matrix is evaluated at the critical point where the associated eigenvalue is zero. By enforcing the condition $J \cdot \mathbf{M} = \mathbf{0}$,

the following matrix system is obtained:

$$\begin{bmatrix} -C_1 & 0 & 0 & 0 & 0 & 0 & 0 & \delta(1-\eta) & 0 & 0 & 0 \\ \lambda(1-\gamma) & -C_2 & 0 & 0 & 0 & 0 & 0 & \eta & 0 & 0 & -C_3 \\ 0 & 0 & -C_4 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & C_5 \\ 0 & 0 & \alpha_h & -C_6 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \phi & -C_7 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \kappa & 0 & -C_8 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \sigma & \varphi & -C_9 & 0 & 0 & 0 & 0 \\ 0 & C_{10} & C_{11} & 0 & 0 & 0 & \beta & -C_{12} & 0 & 0 & 0 \\ 0 & 0 & 0 & -C_{13} & 0 & 0 & 0 & 0 & -C_{14} & 0 & 0 \\ 0 & 0 & 0 & C_{15} & 0 & 0 & 0 & 0 & C_{16} & -C_{17} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \alpha_m & -\mu_m \end{bmatrix} \begin{pmatrix} M_1 \\ M_2 \\ M_3 \\ M_4 \\ M_5 \\ M_6 \\ M_7 \\ M_8 \\ M_9 \\ M_{10} \\ M_{11} \end{pmatrix} = \begin{pmatrix} 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (52)$$

This formulation produces a system of eleven linear homogeneous equations. The components of **M** are then obtained by solving this system, typically by expressing each variable in terms of a chosen free parameter to ensure a non-trivial solution.

Then, from matrix equation (52), we have

$$-M_1C_1 + M_8\delta(1-\eta) = 0 \quad (53)$$

$$M_1\lambda(1-y) - M_2C_2 + M_8\eta - M_{11}C_3 = 0 \quad (54)$$

$$-M_3C_4 + M_{11}C_5 = 0 \quad (55)$$

$$M_3\alpha_h - M_4C_6 = 0 \quad (56)$$

$$M_4\phi - M_5C_7 = 0 \quad (57)$$

$$M_4\kappa - M_6C_8 = 0 \quad (58)$$

$$M_5\sigma + M_6\varphi - M_7C_9 = 0 \quad (59)$$

$$M_2C_{10} + M_3C_{11} + M_7\beta - M_8C_{12} = 0 \quad (60)$$

$$-M_4C_{13} - M_9C_{14} = 0 \quad (61)$$

$$M_4C_{15} - M_{10}C_{16} = 0 \quad (62)$$

$$M_{10}\alpha_m - M_{11}\mu_m = 0 \quad (63)$$

Solving equations (53) to (63) and make M_3 free we obtained

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$$M_1 = \frac{\delta(1-\eta)(C_{19}+C_{10}C_{17})}{C_{12}+C_{10}C_{18}}M_3 \quad M_2 = \left(C_{17} - \frac{C_{18}(C_{19}+C_{10}C_{17})}{C_{12}+C_{10}C_{18}}\right)M_3$$

$$M_3 = M_3, \quad M_4 = \frac{\alpha_h}{C_6}M_3, \; M_5 = \frac{\phi\alpha_h}{C_6C_7}M_3, \quad M_6 = \frac{\kappa\alpha_h}{C_8C_6}M_3, \; M_7 = \left(\frac{\sigma\phi}{C_7}+\frac{\varphi}{C_8}\right)\frac{\alpha_h}{C_6}M_3,$$

$$M_8 = \frac{(C_{19}+C_{10}C_{17})}{C_{12}+C_{10}C_{18}}M_3, \; M_9 = -\frac{\alpha_h C_{13}}{C_6C_{14}}M_3, \; M_{10} = \frac{\alpha_h C_{15}}{C_6C_{16}}M_3, \; M_{11} = \frac{\alpha_h\alpha_m C_{15}}{C_6C_{16}\mu_m}M_3,$$

Where,

$$C_{17} = \frac{\alpha_h\alpha_m(C_3C_{15})}{C_2C_6C_{16}\mu_m}, \; C_{18} = \frac{\delta(1-\eta)}{C_1C_2+} + \frac{\eta}{C_2}, \; C_{19} = C_{11} + \left(\frac{\sigma\phi}{C_{17}}+\frac{\varphi}{C_8}\right)\frac{\alpha_h\beta}{C_6}$$

Thus,

$$M = \begin{pmatrix} \frac{\delta(1-\eta)(C_{19}+C_{10}C_{17})}{C_{12}+C_{10}C_{18}}M_3, \left(C_{17} - \frac{C_{18}(C_{19}+C_{10}C_{17})}{C_{12}+C_{10}C_{18}}\right)M_3, M_3, \frac{\alpha_h}{C_6}M_3, \\ \frac{\phi\alpha_h}{C_6C_7}M_3, \frac{\kappa\alpha_h}{C_8C_6}M_3, \left(\frac{\sigma\phi}{C_7}+\frac{\varphi}{C_8}\right)\frac{\alpha_h}{C_6}M_3, \frac{(C_{19}+C_{10}C_{17})}{C_{12}+C_{10}C_{18}}M_3, -\frac{\alpha_h C_{13}}{C_6C_{14}}M_3, \\ \frac{\alpha_h C_{15}}{C_6C_{16}}M_3, \frac{\alpha_h\alpha_m C_{15}}{C_6C_{16}\mu_m}M_3, \end{pmatrix}$$

Now, we make U_3 and M_3 such, that $U.M^T = 1$ that is,

$$\left(0+0+U_3M_3+\frac{C_4}{C_6}U_3M+0+0+0+0+0+\frac{C_5C_5\alpha_h}{C_6C_{16}\mu_m}U_3M_3+\frac{C_5\alpha_m^2\alpha_h}{C_6C_{16}\mu_m^2}U_3M_3\right)=1$$

These implies that,

$$\left(1+\frac{C_4}{C_6}+\frac{C_5C_5\alpha_h}{C_6C_{16}\mu_m}+\frac{C_5\alpha_m^2\alpha_h}{C_6C_{16}\mu_m^2}\right)U_3M_3 = 1$$

That is,

$$U_3M_3 = \frac{1}{\left(1+\frac{C_4}{C_6}+\frac{C_5C_5\alpha_h}{C_6C_{16}\mu_m}+\frac{C_5\alpha_m^2\alpha_h}{C_6C_{16}\mu_m^2}\right)} > 0. \tag{64}$$

From (64), is not enough to say that the model equations (38) exhibit backward bifurcation.

Now, we need to select U_3 and M_3 which have $U_3 > 0$ and $M_3 > 0$ or $U_3 < 0$ and $M_3 < 0$.

Estimation of the coefficient of A and B.

Since $[U_1\ U_2\ \cdots\ U_{11}] = 0$, we shall only compute the part derivative of f_3, f_{45}, f_{10}, f_{11} at $J(E_0\rho_h^*)$.

For $A = \sum_{k,i,j=1}^{n} U_k M_i M_j \frac{\partial^2 f_k}{\partial V_i \partial V_j}(E_0\rho_h^{\bullet})$, we therefore computation the non-zero second-order partial derivatives for A as follows:

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$$U_3M_{11}M_3\frac{\partial^2 f_3}{\partial V_{11}\partial V_3}\left(E_0\rho_h^{\bullet}\right)=\frac{C_5\alpha_{h\alpha_m}b\rho_m}{\left(\phi+\kappa+\mu_h+\theta\right)\left(\alpha_m+\mu_m\right)}U_3M_3^2$$

$$U_3M_3^2\frac{\partial^2 f_3}{\partial V_3^2}\left(E_0\rho_h^{\bullet}\right)=\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)U_3M_3^2$$

$$U_4M_3M_4\frac{\partial^2 f_4}{\partial V_3\partial V_4}\left(E_0\rho_h^{\bullet}\right)=\frac{\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)}{\left(\phi+\kappa+\mu_h+\theta\right)}U_3M_3^2$$

$$U_4M_4^2\frac{\partial^2 f_4}{\partial V_4^2}\left(E_0\rho_h^{\bullet}\right)=\frac{\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)\alpha_h}{\left(\phi+\kappa+\mu_h+\theta\right)}U_3M_3^2$$

$$U_{10}M_4M_{10}\frac{\partial^2 f_{10}}{\partial V_4\partial V_{10}}\left(E_0\rho_h^{\bullet}\right)=\frac{C_5b^3\rho_m^3\alpha_m\alpha_h}{\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}U_3M_3^2$$

$$U_{10}M_{10}^2\frac{\partial^2 f_{10}}{\partial V_{10}^2}\left(E^0\rho_h^{\bullet}\right)=\frac{C_5b^2\rho_m^2\alpha_m\alpha_h^2}{\mu_m\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}U_3M_3^2$$

$$U_{11}M_{10}M_{11}\frac{\partial^2 f_{11}}{\partial V_{10}\partial V_{11}}\left(E^0\rho_h^{\bullet}\right)=\frac{C_5b^2\rho_m^2\alpha_m^2\alpha_h^2}{\mu_m\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}U_3M_3^2$$

$$U_{11}M_{11}^2\frac{\partial^2 f_{11}}{\partial V_{11}^2}\left(E_0\rho_h^{\bullet}\right)=\frac{b^2\rho_m^2\alpha_m^2\alpha_h^2}{\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}U_3M_3^2$$

This implies that,

$$A=\left(\begin{array}{l}\frac{C_5\alpha_{h\alpha_m}b\rho_m}{\left(\phi+\kappa+\mu_h+\theta\right)\left(\alpha_m+\mu_m\right)}+\frac{\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)}{\left(\phi+\kappa+\mu_h+\theta\right)}+\frac{\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)\alpha_h}{\left(\phi+\kappa+\mu_h+\theta\right)}+\\ \frac{C_5b^3\rho_m^3\alpha_m\alpha_h}{\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}+\frac{C_5b^2\rho_m^2\alpha_m\alpha_h^2}{\mu_m\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}+\frac{C_5b^2\rho_m^2\alpha_m^2\alpha_h^2}{\mu_m\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}\\ +\frac{b^2\rho_m^2\alpha_m^2\alpha_h^2}{\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}-\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)\end{array}\right)U_3M_3^2$$

Then,

$$A=\left(Z_1-Z_2\right)U_3M_3^2 \tag{65}$$

Where,

$$C_5=\frac{\mu_h\left(\mu_h+\lambda\left(1-y\right)\right)\left(\alpha_h+\alpha_1+\mu_h\right)\left(\phi+\kappa+\mu_h+\theta\right)\left(\alpha_m+\mu_m\right)\mu_m}{b^2\omega_h\alpha_m\alpha_h\rho_m\left(\lambda\left(1-y\right)+\left(1-\gamma\right)\mu_h\right)}$$

$$Z_1=\left(\begin{array}{l}\frac{C_5\alpha_{h\alpha_m}b\rho_m}{\left(\phi+\kappa+\mu_h+\theta\right)\left(\alpha_m+\mu_m\right)}+\frac{\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)}{\left(\phi+\kappa+\mu_h+\theta\right)}+\frac{\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)\alpha_h}{\left(\phi+\kappa+\mu_h+\theta\right)}+\\ \frac{C_5b^3\rho_m^3\alpha_m\alpha_h}{\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}+\frac{C_5b^2\rho_m^2\alpha_m\alpha_h^2}{\mu_m\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}+\frac{C_5b^2\rho_m^2\alpha_m^2\alpha_h^2}{\mu_m\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}\\ +\frac{b^2\rho_m^2\alpha_m^2\alpha_h^2}{\left(\phi+\kappa+\mu_h+\theta\right)^2\left(\alpha_m+\mu_m\right)^2}\end{array}\right)$$

$$Z_2=-\left(\alpha_h+\alpha_1\left(1-y_2\right)+\mu_h\right)$$

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For $B = \sum_{k,i,j=1}^{n} U_k M_i M_j \frac{\partial^2 f_k}{\partial V_i \partial \rho_{h_j}^*}\left(E^0 \rho_h^{\bullet}\right)$, we also computation the non-zero second-order partial derivatives for B as follows:

$$U_3 M_{11} M_3 \frac{\partial^2 f_3}{\partial V_{11} \partial \rho_h^*}\left(E^0 \rho_h^{\bullet}\right) = \frac{b\left(\lambda(1-y)+(1-\gamma)\mu_h\right)}{\left(\mu_h + \lambda(1-y)\right)} \frac{\alpha_{h\alpha_m} b\rho_m}{\left(\phi+\kappa+\mu_h+\theta\right)\left(\alpha_m+\mu_m\right)} U_3 M_3^2 > 0$$

Therefore,

$$B = \frac{b\left(\lambda(1-y)+(1-\gamma)\mu_h\right)}{\left(\mu_h + \lambda(1-y)\right)} \frac{\alpha_{h\alpha_m} b\rho_m}{\left(\phi+\kappa+\mu_h+\theta\right)\left(\alpha_m+\mu_m\right)} U_3 M_3^2 \tag{66}$$

Discussion

The analytical investigation of the proposed model reveals important characteristics of the system's dynamical behavior. From the analytical expressions derived in equation (65), it is observed that the signs of the bifurcation coefficients critically determine the type of bifurcation exhibited by the system. Specifically, when $(z_1 - z_2) > 0$and $U_3 > 0$, both bifurcation coefficients $A$and $B$are positive. Under this parameter regime, the system demonstrates a backward bifurcation phenomenon.

This backward bifurcation implies that the model supports the coexistence of a stable disease-free equilibrium (DFE) and a stable endemic equilibrium even when the basic reproduction number satisfies $R_0 < 1$. In such a situation, the classical epidemiological assumption that reducing R_0below unity guarantees disease elimination no longer holds. Instead, the system exhibits a more complex dynamic in which the disease may persist depending on the initial conditions and parameter configuration.

On the other hand, when $(z_1 - z_2) < 0$while $U_3 > 0$, the structure of the bifurcation changes. In this case, coefficient Abecomes negative whereas coefficient Bremains positive. This parameter setting leads to a forward bifurcation behaviour. Under forward bifurcation, the disease-free equilibrium is locally asymptotically stable whenever $R_0 < 1$, and the system transitions to endemic persistence only when $R_0 > 1$, which aligns with the classical threshold dynamics observed in standard infectious disease models.

These results indicate that the transmission dynamics of the system are highly sensitive to variations in key parameters. The presence of both backward and forward bifurcation regimes highlights the nonlinear complexity of the model and emphasizes that disease control cannot rely solely on reducing the reproduction number. Instead, control strategies must also account for system parameters that influence bifurcation behavior in order to ensure reliable disease elimination.

Conclusion

This study formulated and analyzed a deterministic mathematical model to investigate the transmission dynamics of malaria within a population framework. The analysis focused on key epidemiological properties, including the existence and stability of equilibrium points and the role of the basic reproduction number as a threshold parameter governing disease invasion. The model confirmed the existence of both disease-free and endemic equilibria under specific parameter conditions. The derived reproduction number served as a critical threshold for determining whether malaria would die out or persist in the population. Furthermore, bifurcation analysis revealed that the system may exhibit either backward or forward bifurcation depending on the relationship between parameters.

In particular, when $(z_1 - z_2) > 0$and $U_3 > 0$, both bifurcation coefficients remain positive, resulting in backward bifurcation. This indicates the coexistence of stable equilibria and suggests that simply reducing R_0below unity is not sufficient for disease eradication, as the system may still sustain endemic persistence. Conversely, when $(z_1 - z_2) < 0$and $U_3 > 0$, the system undergoes forward bifurcation, implying that once $R_0$is reduced below one, the disease-free state becomes globally attractive and elimination becomes achievable. In conclusion, the results demonstrate that malaria transmission dynamics in the model are strongly influenced by nonlinear parameter interactions. Therefore,

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effective control strategies must go beyond basic threshold reduction and consider deeper system structures that govern bifurcation behavior and long-term disease persistence.

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