

MATHEMATICAL MODELLING OF MALARIA TRANSMISSION DYNAMICS IN TANZANIA INCORPORATING TEMPERATURE AND RAINFALL VARIABILITY WITH SUSCEPTIBILITY LEVELS

(Pemodelan Matematik Dinamik Penularan Malaria di Tanzania dengan Mengambil Kira Kebolehubahan Suhu dan Hujan serta Tahap Kerentanan)

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ABSTRACT

Malaria remains a major public health challenge in many tropical regions, where environmental conditions strongly influence disease transmission. In this study, a mathematical model is developed to investigate the impact of temperature and rainfall on malaria dynamics in Tanzania while accounting for differences in human susceptibility to mosquito bites. The model consists of interacting human and mosquito populations and incorporates climate-dependent parameters that affect mosquito survival, reproduction, and transmission potential. The analytical analysis is performed to determine the disease-free and endemic equilibrium states and to derive the basic reproduction number, which serves as a threshold indicator for disease persistence. Numerical simulations are conducted to examine the effects of varying climatic conditions on malaria transmission. The findings shows that both infected humans and infected mosquitoes increases as temperature increases from 15°C to 35°C, after which the infection decreases as the temperature continues to increase beyond 35°C. Additionally, the infection in mosquitoes increases as rainfall rises from 15 mm to 25 mm, but it decreases when rainfall increases beyond 25 mm. Thus, the findings suggest that considering temperature, rainfall and human susceptibilities can help identify effective malaria control methods.

Keywords: malaria; temperature; rainfall; mathematical modeling; low-risk; high-risk

ABSTRAK

Malaria kekal sebagai cabaran utama kesihatan awam di banyak kawasan tropika, di mana keadaan persekitaran sangat mempengaruhi penularan penyakit. Dalam kajian ini, satu model matematik dibangunkan untuk mengkaji kesan suhu dan taburan hujan terhadap dinamik malaria di Tanzania dengan mengambil kira perbezaan kerentanan manusia terhadap gigitan nyamuk. Model ini terdiri daripada interaksi antara populasi manusia dan nyamuk serta menggabungkan parameter yang bergantung kepada iklim yang mempengaruhi kelangsungan hidup, pembiakan dan potensi penularan nyamuk. Analisis secara analitik dijalankan untuk menentukan keadaan keseimbangan bebas penyakit dan keseimbangan endemik, serta untuk memperoleh bilangan pembiakan asas, yang berfungsi sebagai penunjuk ambang bagi kelangsungan penyakit. Simulasi berangka dijalankan untuk mengkaji kesan perubahan keadaan iklim terhadap penularan malaria. Dapatan kajian menunjukkan bahawa bilangan manusia yang dijangkiti dan nyamuk yang dijangkiti meningkat apabila suhu meningkat daripada 15°C kepada 35°C, sebelum jangkitan menurun apabila suhu terus meningkat melebihi 35°C. Selain itu, jangkitan dalam populasi nyamuk meningkat apabila taburan hujan meningkat daripada 15 mm kepada 25 mm, tetapi menurun apabila taburan hujan melebihi 25 mm. Oleh itu, dapatan kajian mencadangkan bahawa dengan mempertimbangkan faktor suhu, taburan hujan dan tahap kerentanan manusia, kaedah kawalan malaria yang lebih berkesan dapat dikenal pasti.

Kata kunci: malaria; suhu; taburan hujan; pemodelan matematik; risiko rendah; risiko tinggi

1. Introduction

Female *Anopheles* mosquitoes bite humans to obtain blood meals for egg development. During this process, infected mosquitoes may transmit *Plasmodium* parasites into the human bloodstream, causing malaria infection (Chacha *et al.* 2025b). The burden of malaria is particularly high in tropical regions, where the disease continues to claim numerous lives and hinder socioeconomic development. The World Health Organization reported that, in 2024, malaria accounted for over 170 million cases and approximately one million deaths worldwide (World Health Organization 2025). Despite being a global health concern, malaria disproportionately affects sub-Saharan Africa, where nearly 95% of the world's malaria-related morbidity and mortality occur (Ngonghala 2023; Orwa *et al.* 2022; Taddese *et al.* 2024).

Five species of *Plasmodium* parasites are responsible for malaria infection, namely *P. falciparum*, *P. vivax*, *P. malariae*, *P. ovale*, and *P. knowlesi*. These species vary in their pathogenicity and in the environmental conditions that favour the survival of their mosquito vectors (Rossati *et al.* 2016; Joë *et al.* 2024; Madito & Silal 2024).

Mathematical modelling has become an important tool for studying the transmission dynamics of infectious diseases and evaluating the effectiveness of intervention strategies. Early malaria models focused primarily on the transmission process between human and mosquito populations and established fundamental concepts such as threshold dynamics and the basic reproduction number (Bacaër 2011; Macdonald 1957; Anderson & May 1991). These studies laid the foundation for understanding disease persistence and assessing the potential impact of control measures.

To improve the realism of malaria models, researchers have incorporated additional biological, demographic, and environmental factors that influence disease transmission. Several studies have examined the role of human mobility and migration in spreading malaria across geographical regions (Torres-Sorando & Rodriguez 1997; Yacheur *et al.* 2019; Adegbite *et al.* 2023). The results indicate that the movement of infected individuals between communities can contribute to the introduction or persistence of malaria in previously unaffected areas. Particularly, when the local population is not in an endemic state, the introduction of infected migrants can establish endemicity, depending on the level of local population growth. Other investigations have considered age structure, demonstrating that young children often experience a higher disease burden due to their relatively low immunity (Ranjha *et al.* 2023; Forouzannia & Gumel 2014; Tchoumi *et al.* 2022). Socioeconomic factors have also been incorporated into malaria models to evaluate how access to prevention and treatment influences disease outcomes and intervention effectiveness (Olaniyi *et al.* 2022). Findings suggest that vector control outperforms other single interventions in terms of effectiveness, while antimalarial drugs provide the highest cost-effectiveness when resources are constrained. Furthermore, several mathematical models have been expanded to account for public awareness and educational efforts related to malaria transmission (Kar *et al.* 2019; Ndi & Adi 2021). Malaria control is more effective when high-intensity awareness initiatives are implemented together with vector control interventions.

Behavioural heterogeneity among individuals has received increasing attention in recent years. Differences in behaviour, dwelling location, and daily activities can lead to variations in exposure to mosquito bites (Chacha *et al.* 2025b;a; 2026). Consequently, some individuals may face a substantially greater risk of infection than others. Mathematical models that account for such heterogeneity have shown that concentrating mosquito bites among highly exposed individuals can significantly alter disease dynamics and increase transmission within the population. These findings highlight the importance of considering differences in human susceptibility when investigating malaria transmission patterns. Research efforts have also extended to modelling malaria infection at the individual host level. Such studies examine the behaviour and progression of *Plasmodium* parasites within the human host while exploring the potential of therapeutic and preventive measures, such as antimalarial medications and vaccines, to suppress parasite growth and transmission (Orwa *et al.* 2022; 2018). The findings indicate that the combined use of effective blood schizontocidal drugs and a highly efficacious blood-stage vaccine can

substantially reduce the parasite burden and facilitate the rapid elimination of malaria parasites from the human host.

Environmental conditions play a crucial role in the life cycle of mosquitoes and consequently affect malaria transmission. Temperature influences mosquito development, survival, biting frequency, and parasite maturation, while rainfall affects the availability of breeding habitats and the growth of mosquito populations (Joë *et al.* 2024; Madito & Silal 2024; Abiodun *et al.* 2016; Parham & Michael 2010). Joë *et al.* (2024) reported that, in Burundi, temperatures ranging from 20°C to 32°C, together with monthly rainfall between 10 mm and 30 mm, create favourable conditions for increased mosquito population growth. In a related study, malaria incidence in Zimbabwe was found to increase as temperatures rose from 30°C to 32°C, but to decline when temperatures exceeded 40°C (Ngarakana-Gwasira *et al.* 2014). Although numerous studies have investigated the influence of climate on malaria dynamics, relatively few have simultaneously incorporated climatic variability and heterogeneous human susceptibility into a single modelling framework. Furthermore, the availability of recent climate data provides an opportunity to reassess the relationship between environmental conditions and malaria transmission in regions where the disease remains endemic. Tanzania continues to experience a substantial malaria burden, making it an important setting for investigating the combined effects of climate and human exposure heterogeneity. Motivated by these observations, this study develops a mathematical model of malaria transmission that incorporates temperature-dependent and rainfall-dependent processes together with high-risk and low-risk susceptible human populations. The model is analysed to determine the conditions under which malaria can persist or be eliminated. In addition, numerical simulations are performed to evaluate the effects of climatic variability on disease dynamics and to provide insights that may support malaria control and prevention efforts in Tanzania.

The remainder of this paper is organised as follows: Section 2 presents the formulation of the mathematical model. Section 3 presents the analytical results, including the disease-free equilibrium, endemic equilibrium, and basic reproduction number. Model fitting and parameter estimation procedures are described in Section 4, while the sensitivity analysis is presented in Section 5. Numerical simulation results are provided in Section 6, and the main conclusions of the study are discussed in Section 7.

2. Model Formulation

This section presents a deterministic compartmental model for malaria transmission that incorporates the influence of temperature and rainfall on mosquito population dynamics. In addition, the human population is divided into groups according to their levels of exposure to mosquito bites, allowing the model to capture heterogeneity in infection risk.

The total human population at time t , denoted by $N_h(t)$, is partitioned into four epidemiological classes: low-risk susceptible individuals $S_{hL}(t)$, high-risk susceptible individuals $S_{hH}(t)$, infected individuals $I_h(t)$, and recovered individuals $R_h(t)$. The total mosquito population, represented by $N_m(t)$, is divided into susceptible mosquitoes $S_m(t)$ and infected mosquitoes $I_m(t)$. The proposed system of differential equations captures the interactions between humans and mosquitoes and examines how climatic variability influences malaria transmission.

Human individuals are recruited into the human population at a constant rate Λ_h . Of these recruits, a fraction γ is assigned to the high-risk susceptible compartment, whereas the complementary fraction $(1 - \gamma)$ is assigned to the low-risk susceptible compartment. Over time, susceptible humans are assumed to move between risk groups. The movement of susceptible individuals from the low-risk class to the high-risk class occurs at a rate ϕ_1 , while the reverse movement from the high-risk class to the low-risk class occurs at a rate ϕ_2 . These movements between risk groups may arise from changes in behavioural patterns, occupational conditions, or residential settings.

Moreover, individuals who recover from malaria are assumed to develop temporary immunity.

Once this immunity wanes, they return to the susceptible population at a rate β . Of the recovered individuals who lose immunity, a fraction α is recruited into the high-risk susceptible compartment, whereas the remaining fraction $(1 - \alpha)$ is recruited into the low-risk susceptible compartment. Malaria transmission to susceptible humans occurs through bites from infectious mosquitoes. Human individuals in the low-risk and high-risk susceptible classes enter the infected compartment according to the forces of infection λ_{h1} and λ_{h2} , respectively. The mathematical expressions for these forces of infection are given by

$$\lambda_{h1} = \frac{\beta_1 r(T) I_m(t)}{N_h(t)},$$

$$\lambda_{h2} = \frac{\theta \beta_1 r(T) I_m(t)}{N_h(t)},$$

where θ measures the additional mosquito-biting exposure associated with high-risk susceptible individuals. The parameter β_1 denotes the probability that an infectious mosquito successfully transmits malaria to a human during a bite, while $r(T)$ represents the mosquito-human contact rate as a temperature-dependent function.

The recruitment rate of mosquitoes is represented by

$$\sigma_m(T, R) \left(1 - \frac{N_m(t)}{K}\right) N_m(t),$$

where $\sigma_m(T, R)$ denotes the mosquito egg-laying rate, which is influenced by both temperature T and rainfall R . The parameter K represents the environmental carrying capacity of the mosquito population, while $N_m(t)$ denotes the total mosquito population size. The number of susceptible mosquitoes increases as eggs develop into adult mosquitoes, whereas it decreases due to infection, represented by the force of infection λ_m , and natural death at rate $d_m(T)$. The mosquito force of infection is defined as

$$\lambda_m = \frac{\beta_2 r(T) I_h(t)}{N_h(t)},$$

where β_2 is the transmission probability from an infected human to a susceptible mosquito during a biting event. The number of infected mosquitoes increases through new infections among susceptible mosquitoes and decreases due to natural death.

A summary of the model variables and parameters is provided in Tables 1 and 2, respectively, while the functional relationships describing temperature- and rainfall-dependent parameters are presented in Table 3. The transmission process is further illustrated by the schematic diagram in Figure 1. The model is constructed under the following assumptions.

2.1. Model Assumptions

This subsection presents the assumptions used in the formulation of the model.

- (1) All state variables and parameters are assumed to be non-negative.
- (2) High-risk susceptible humans experience more mosquito bites than low-risk susceptible humans.
- (3) The parameter θ , which represents the relative proportion of mosquito bites experienced by high-risk susceptible humans, is greater than one.
- (4) Human susceptibility may shift between low- and high-risk classes due to changes in location, behaviour, or occupation.

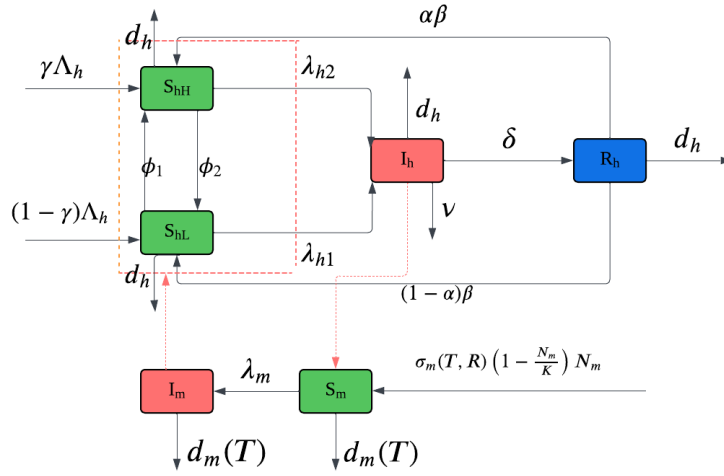


Figure 1: The flow of malaria transmission dynamics and the mosquito life cycle

$$\begin{aligned}
 \frac{dS_{hL}}{dt} &= (1 - \gamma)\Lambda_h + (1 - \alpha)\beta R_h(t) + \phi_2 S_{hH}(t) \\
 &\quad - \frac{\beta_1 r(T) I_m(t)}{N_h(t)} S_{hL}(t) - d_h S_{hL}(t) - \phi_1 S_{hL}(t), \\
 \frac{dS_{hH}}{dt} &= \gamma\Lambda_h + \alpha\beta R_h(t) + \phi_1 S_{hL}(t) - \frac{\theta\beta_1 r(T) I_m(t)}{N_h(t)} S_{hH}(t) - d_h S_{hH}(t) - \phi_2 S_{hH}(t), \\
 \frac{dI_h}{dt} &= \frac{\beta_1 r(T) I_m(t)}{N_h(t)} S_{hL}(t) + \frac{\theta\beta_1 r(T) I_m(t)}{N_h(t)} S_{hH}(t) - (\nu + d_h + \delta) I_h(t), \\
 \frac{dR_h}{dt} &= \delta I_h(t) - \beta R_h(t) - d_h R_h(t), \\
 \frac{dS_m}{dt} &= \sigma_m(T, R) \left(1 - \frac{N_m(t)}{K}\right) N_m(t) - d_m(T) S_m(t) - \frac{\beta_2 r(T) I_h(t)}{N_h(t)} S_m(t), \\
 \frac{dI_m}{dt} &= \frac{\beta_2 r(T) I_h(t)}{N_h(t)} S_m(t) - d_m(T) I_m(t).
 \end{aligned} \tag{1}$$

Table 1: Variables of the model in Eq. 1

Variable	Description
$S_{hL}(t)$	Low-risk susceptible human individuals
$S_{hH}(t)$	High-risk susceptible human individuals
$I_h(t)$	Infected human individuals
$R_h(t)$	Recovered human individuals
$S_m(t)$	Susceptible mosquitoes
$I_m(t)$	Infected mosquitoes

Table 2: Parameters of the model in Eq. 1

Parameter	Description
Λ_h	Human recruitment rate
γ	Proportion of recruited humans entering the high-risk susceptible class
ϕ_1, ϕ_2	Transition rates between the low-risk and high-risk susceptible classes, respectively
d_h	Natural death rate of humans
β	Rate of loss of temporary immunity in recovered humans
α	Proportion of recovered individuals who enter the high-risk susceptible class after losing immunity
ν	Malaria-induced human death rate
β_1	Transmission probability from mosquito to human
β_2	Transmission probability from human to mosquito
θ	Relative increase in mosquito-biting exposure among high-risk susceptible humans
$r(T)$	Temperature-dependent contact rate between mosquitoes and humans
δ	Natural recovery rate of infected humans
$\sigma_m(T, R)$	Temperature- and rainfall-dependent mosquito recruitment rate
$d_m(T)$	Natural death rate of adult mosquitoes
K	The carrying capacity

Table 3: Temperature- and rainfall-dependent parameters of the model in Eq. 1

Parameter	Functional form	Citation
$d_m(T)$	$\frac{1}{\alpha_1 T^2 + \alpha_2 T + \alpha_3}, \quad \alpha_1, \alpha_2, \alpha_3 \in \mathbb{R}$	(Joë <i>et al.</i> 2024; Madito & Silal 2024)
$r(T)$	$q_1 T(T - q_2) \sqrt{q_3 - T}, \quad q_1, q_2, q_3 \in \mathbb{R}$	(Joë <i>et al.</i> 2024; Madito & Silal 2024)
$\sigma_m(T, R)$	$\frac{N_E S_E(R) S_L(T, R) S_P(R)}{\chi_E + \chi_L(T) + \chi_P}$	(Joë <i>et al.</i> 2024)

Table 4: Parameters of the functional form in Table 3

Parameter	Description
N_E	Total number of eggs laid per adult female mosquito
$S_E(R)$	The survival probability of eggs
χ_E	Duration of egg
$S_P(R)$	The survival probability of pupa

The system in Eq. 1 can also be expressed as

$$\frac{dX}{dt} = G(X, t) = \begin{pmatrix} (1 - \gamma)\Lambda_h + (1 - \alpha)\beta R_h(t) + \phi_2 S_{hH}(t) - \frac{\beta_1 r(T) I_m(t)}{N_h(t)} S_{hL}(t) - d_h S_{hL}(t) - \phi_1 S_{hL}(t) \\ \gamma \Lambda_h + \alpha \beta R_h(t) + \phi_1 S_{hL}(t) - \frac{\theta \beta_1 r(T) I_m(t)}{N_h(t)} S_{hH}(t) - d_h S_{hH}(t) - \phi_2 S_{hH}(t) \\ \frac{\beta_1 r(T) I_m(t)}{N_h(t)} S_{hL}(t) + \frac{\theta \beta_1 r(T) I_m(t)}{N_h(t)} S_{hH}(t) - (\nu + d_h + \delta) I_h(t) \\ \delta I_h(t) - \beta R_h(t) - d_h R_h(t) \\ \sigma_m(T, R) \left(1 - \frac{N_m(t)}{K} \right) N_m(t) - d_m(T) S_m(t) - \frac{\beta_2 r(T) I_h(t)}{N_h(t)} S_m(t) \\ \frac{\beta_2 r(T) I_h(t)}{N_h(t)} S_m(t) - d_m(T) I_m(t) \end{pmatrix}. \quad (2)$$

where

$$X(t) = (S_{hL}(t), S_{hH}(t), I_h(t), R_h(t), S_m(t), I_m(t)).$$

The system in Eq. 2 can be expressed as subsystems as follows:

$$\frac{dX_1}{dt} = G_1(Z_1, t), \quad (3)$$

where

$$Z_1 = \begin{pmatrix} I_h(t) \\ I_m(t) \end{pmatrix},$$

and

$$G_1(Z_1, t) = \begin{pmatrix} \lambda_{h1}S_{hL}(t) + \lambda_{h2}S_{hH}(t) - (\nu + d_h + \delta)I_h(t) \\ \lambda_m S_m(t) - d_m(T)I_m(t) \end{pmatrix}.$$

Moreover,

$$\frac{dX_2}{dt} = G_2(Z_2, t), \quad (4)$$

where

$$Z_2 = \begin{pmatrix} S_{hL}(t) \\ S_{hH}(t) \\ R_h(t) \\ S_m(t) \end{pmatrix},$$

and

$$G_2(Z_2, t) = \begin{pmatrix} (1 - \gamma)\Lambda_h + \phi_2 S_{hH}(t) + (1 - \alpha)\beta R_h(t) - \phi_1 S_{hL}(t) - \lambda_{h1} S_{hL}(t) - d_h S_{hL}(t) \\ \gamma \Lambda_h + \phi_1 S_{hL}(t) + \alpha \beta R_h(t) - \phi_2 S_{hH}(t) - \lambda_{h2} S_{hH}(t) - d_h S_{hH}(t) \\ \delta I_h(t) - \beta R_h(t) - d_h R_h(t) \\ \sigma_m(T, R) \left(1 - \frac{N_m(t)}{K}\right) N_m(t) - d_m(T) S_m(t) - \lambda_m S_m(t) \end{pmatrix}.$$

where

$$\chi_L(T) = \frac{1}{\gamma_1 T + \gamma_2}, \quad \gamma_1, \gamma_2 \in \mathbb{R},$$

denotes the temperature-dependent duration of the larval stage. Furthermore,

$$S_L(T, R) = S_{L1}(T)S_{L2}(R)$$

represents the overall larval survival probability, where $S_{L1}(T)$ and $S_{L2}(R)$ describe the survival probabilities associated with temperature and rainfall, respectively. The temperature-dependent larval survival probability is given by

$$S_{L1}(T) = e^{-a_1 T + a_2},$$

while the rainfall-dependent larval survival probability is given by

$$S_{L2}(R) = \left(\frac{4P_L \text{Max}}{R_{W0}^2} \right) R(R_{W0} - R).$$

Here, $P_L \text{Max}$ denotes the maximum larval survival probability, R_{W0} denotes the rainfall wash-out limit for larvae, R represents rainfall, and T represents temperature. In addition, $S_P(R)$ denotes the rainfall-dependent pupal survival probability, while χ_P represents the duration of the pupal stage.

2.2. Properties of the Model

Theorem 2.1. *If the initial values $S_{hL}(0)$, $S_{hH}(0)$, $I_h(0)$, $R_h(0)$, $S_m(0)$, and $I_m(0)$ of the model in Eq. 1 are non-negative, then the state variables $S_{hL}(t)$, $S_{hH}(t)$, $I_h(t)$, $R_h(t)$, $S_m(t)$, and $I_m(t)$ remain non-negative for all $t > 0$.*

In addition, if

$$N_h(0) \leq \frac{\Lambda_h}{d_h} \quad \text{and} \quad N_m(0) \leq \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)},$$

then

$$N_h(t) \leq \frac{\Lambda_h}{d_h} \quad \text{and} \quad N_m(t) \leq \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)}$$

for all $t > 0$.

In particular, the region

$$\Omega = \Omega_h \times \Omega_m$$

is positively invariant and attracting, where

$$\Omega_h = \left\{ (S_{hL}(t), S_{hH}(t), I_h(t), R_h(t)) \in \mathbb{R}^4 : N_h(t) \leq \frac{\Lambda_h}{d_h} \right\},$$

and

$$\Omega_m = \left\{ (S_m(t), I_m(t)) \in \mathbb{R}^2 : N_m(t) \leq \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)} \right\}.$$

Proof. Beginning with the first equation of the model in Eq. 1,

$$\begin{aligned} \frac{dS_{hL}(t)}{dt} &= (1 - \gamma)\Lambda_h + (1 - \alpha)\beta R_h(t) + \phi_2 S_{hH}(t) \\ &\quad - \frac{\beta_1 r(T) I_m(t)}{N_h(t)} S_{hL}(t) - d_h S_{hL}(t) - \phi_1 S_{hL}(t). \end{aligned}$$

Since all state variables and parameters are non-negative,

$$\frac{dS_{hL}(t)}{dt} \geq - \left(\frac{\beta_1 r(T) I_m(t)}{N_h(t)} + d_h + \phi_1 \right) S_{hL}(t).$$

Thus,

$$\frac{dS_{hL}(t)}{dt} + \left(\frac{\beta_1 r(T) I_m(t)}{N_h(t)} + d_h + \phi_1 \right) S_{hL}(t) \geq 0.$$

Integrating from the initial time 0 to time t gives

$$S_{hL}(t) \geq S_{hL}(0) \exp \left[- \int_0^t \left(\beta_1 r(T) \frac{I_m(b)}{N_h(b)} + d_h + \phi_1 \right) db \right] \geq 0.$$

Hence, $S_{hL}(t)$ remains non-negative for all $t > 0$. By applying a similar argument to the remaining equations, it follows that

$$S_{hL}(t), S_{hH}(t), I_h(t), R_h(t), S_m(t), I_m(t) \geq 0, \quad t > 0.$$

Moreover, summing the first four equations representing the human population gives

$$\begin{aligned} \frac{dN_h(t)}{dt} &= \Lambda_h - d_h N_h(t) - \nu I_h(t) \\ &\leq \Lambda_h - d_h N_h(t). \end{aligned}$$

By the comparison theorem,

$$N_h(t) \leq N_h(0)e^{-d_h t} + \frac{\Lambda_h}{d_h} (1 - e^{-d_h t}).$$

As $t \rightarrow \infty$, $e^{-d_h t} \rightarrow 0$, and hence

$$\limsup_{t \rightarrow \infty} N_h(t) \leq \frac{\Lambda_h}{d_h}.$$

Therefore, if

$$N_h(0) \leq \frac{\Lambda_h}{d_h},$$

then

$$N_h(t) \leq \frac{\Lambda_h}{d_h}, \quad t > 0.$$

Similarly, summing the two equations representing the mosquito population gives

$$\frac{dN_m(t)}{dt} = \sigma_m(T, R) \left(1 - \frac{N_m(t)}{K} \right) N_m(t) - d_m(T) N_m(t).$$

Assuming that

$$\sigma_m(T, R) > d_m(T),$$

the solution is given by

$$N_m(t) = \frac{N_m(0) (\sigma_m(T, R) - d_m(T))}{\frac{\sigma_m(T, R)}{K} N_m(0) + \left[(\sigma_m(T, R) - d_m(T)) - \frac{\sigma_m(T, R)}{K} N_m(0) \right] e^{-(\sigma_m(T, R) - d_m(T))t}}.$$

As $t \rightarrow \infty$,

$$e^{-(\sigma_m(T, R) - d_m(T))t} \rightarrow 0,$$

which implies that

$$N_m(t) \rightarrow \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)}.$$

Thus, if

$$N_m(0) \leq \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)},$$

then

$$N_m(t) \leq \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)}, \quad t > 0.$$

Therefore,

$$N_h(t) \leq \frac{\Lambda_h}{d_h}$$

and

$$N_m(t) \leq \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)}$$

for all $t > 0$, provided that

$$N_h(0) \leq \frac{\Lambda_h}{d_h}$$

and

$$N_m(0) \leq \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)},$$

respectively. Consequently, the region Ω is positively invariant.

Moreover, if

$$N_h(0) \geq \frac{\Lambda_h}{d_h}$$

and

$$N_m(0) \geq \frac{(\sigma_m(T, R) - d_m(T)) K}{\sigma_m(T, R)},$$

then the corresponding solutions approach these upper bounds as $t \rightarrow \infty$. Hence, the region Ω is attracting. $\square$

Since

$$S_{hL}(t), S_{hH}(t), I_h(t), R_h(t), S_m(t), I_m(t) \geq 0, \quad t > 0,$$

and all solutions of the model that originate in Ω remain in Ω , it is sufficient to study the model in Eq. 1 within Ω , where it is mathematically and biologically feasible.

3. Dynamic Stability Analysis

This section presents an analytical investigation of the dynamical stability of the model in Eq. 1. It begins by establishing the disease-free equilibrium, followed by the derivation of the basic reproduction number and an investigation of the local stability of the disease-free equilibrium. Finally, the endemic equilibrium is obtained under the condition that the basic reproduction number exceeds unity.

3.1. Disease-Free Equilibrium

The disease-free equilibrium corresponds to the steady state of the system in which no infection exists in the population, such that

$$I_h = R_h = I_m = 0.$$

Thus, the disease-free equilibrium of the model in Eq. 1 is given by

$$\begin{aligned} \mathcal{E}_0 &= (S_{hL}^*, S_{hH}^*, I_h^*, R_h^*, S_m^*, I_m^*) \\ &= \left(\frac{A_7}{B_1 - \phi_1}, \frac{\Lambda_h \gamma}{B_2 - \phi_2}, 0, 0, \frac{K(\sigma_m(T, R) - d_m(T))}{\sigma_m(T, R)}, 0 \right), \end{aligned}$$

where

$$\sigma_m(T, R) > d_m(T), \quad A_7 = (1 - \gamma)\Lambda_h, \quad B_1 = d_h + \phi_2,$$

and

$$B_2 = d_h + \phi_1.$$

3.2. Basic Reproduction Number

The basic reproduction number, denoted by $\mathcal{R}_0$, is the average number of secondary infections produced by a single infected individual introduced into a fully susceptible population during the individual's infectious period. The next-generation matrix method of van den Driessche & Watmough (2002) is applied to compute the basic reproduction number.

The infection subsystem in Eq. 3 of the model in Eq. 1 yields the vector functions $\mathcal{F}$ and $\mathcal{V}$, which represent the rates of new infections and transitions between compartments, respectively. These vector functions are given by

$$\mathcal{F} = \begin{pmatrix} \lambda_{h1} S_{hL} + \lambda_{h2} S_{hH} \\ \lambda_m S_m \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} (\nu + d_h + \delta) I_h \\ d_m(T) I_m \end{pmatrix}.$$

The Jacobian matrices F and V of the vector functions $\mathcal{F}$ and $\mathcal{V}$, evaluated at $\mathcal{E}_0$, are respectively given by

$$F = \begin{pmatrix} 0 & \frac{S_{hL}^* \beta_1 d_h r(T)}{\Lambda_h} + \frac{S_{hH}^* \beta_1 d_h r(T) \theta}{\Lambda_h} \\ \frac{S_m^* \beta_2 d_h r(T)}{\Lambda_h} & 0 \end{pmatrix},$$

$$V = \begin{pmatrix} A_1 & 0 \\ 0 & d_m(T) \end{pmatrix}.$$

Hence, the next-generation matrix is

$$FV^{-1} = \begin{pmatrix} 0 & \frac{S_{hL}^* \beta_1 d_h r(T) + S_{hH}^* \beta_1 d_h r(T) \theta}{\Lambda_h d_m(T)} \\ \frac{S_m^* \beta_2 d_h r(T)}{A_1 \Lambda_h} & 0 \end{pmatrix}.$$

Therefore, the spectral radius of FV^{-1} gives the basic reproduction number

$$\mathcal{R}_0(T, R) = \frac{d_h r(T)}{\Lambda_h} \sqrt{\frac{S_{hL}^* S_m^* \beta_1 \beta_2 + S_{hH}^* S_m^* \beta_1 \beta_2 \theta}{A_1 d_m(T)}}. \quad (5)$$

Theorem 3.1. *The disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.*

Proof. We employ the approach used by Chacha *et al.* (2025b) and Tchoumi *et al.* (2022). Let $J(\mathcal{E}_0)$ denote the Jacobian matrix of the model in Eq. 1 evaluated at $\mathcal{E}_0$. Thus,

$$\mathcal{B} = J(\mathcal{E}_0).$$

$$\mathcal{B} = \begin{pmatrix} -B_1 & \phi_2 & 0 & A_8 & 0 & -\frac{S_{hL}^* \beta_1 d_h r(T)}{\Lambda_h} \\ \phi_1 & -B_2 & 0 & \alpha\beta & 0 & -\frac{S_{hH}^* \beta_1 d_h r(T) \theta}{\Lambda_h} \\ 0 & 0 & -A_1 & 0 & 0 & \frac{S_{hH}^* \beta_1 d_h r(T) \theta}{\Lambda_h} + \frac{S_{hL}^* \beta_1 d_h r(T)}{\Lambda_h} \\ 0 & 0 & \delta & -\beta - d_h & 0 & 0 \\ 0 & 0 & -\frac{S_m^* \beta_2 d_h r(T)}{\Lambda_h} & 0 & -\sigma_m(T, R) \left(\frac{S_m^*}{K} - 1 \right) - d_m(T) - \frac{S_m^* \sigma_m(T, R)}{K} & -\sigma_m(T, R) \left(\frac{S_m^*}{K} - 1 \right) - \frac{S_m^* \sigma_m(T, R)}{K} \\ 0 & 0 & \frac{S_m^* \beta_2 d_h r(T)}{\Lambda_h} & 0 & 0 & -d_m(T) \end{pmatrix}.$$

Using the block-structure approach employed by Traoré *et al.* (2017), the matrix $\mathcal{B}$ can be written in block matrix form as

$$\mathcal{B} = \begin{pmatrix} \mathcal{B}_{11} & \mathcal{B}_{12} \\ \mathcal{B}_{21} & \mathcal{B}_{22} \end{pmatrix},$$

$$\mathcal{B}_{11} = \begin{pmatrix} -B_1 & \phi_2 & A_8 & 0 \\ \phi_1 & -B_2 & \alpha\beta & 0 \\ 0 & 0 & -\beta - d_h & 0 \\ 0 & 0 & 0 & -\sigma_m(T, R) \left(\frac{S_m^*}{K} - 1 \right) - d_m(T) - \frac{S_m^* \sigma_m(T, R)}{K} \end{pmatrix}.$$

where

$$\mathcal{B}_{12} = \begin{pmatrix} 0 & -\frac{S_{hL}^* \beta_1 d_h r(T)}{\Lambda_h} \\ 0 & -\frac{S_{hH}^* \beta_1 d_h r(T) \theta}{\Lambda_h} \\ \delta & 0 \\ -\frac{S_m^* \beta_2 d_h r(T)}{\Lambda_h} & -\sigma_m(T, R) \left(\frac{S_m^*}{K} - 1 \right) - \frac{S_m^* \sigma_m(T, R)}{K} \end{pmatrix},$$

$$\mathcal{B}_{21} = \begin{pmatrix} 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

$$\mathcal{B}_{22} = F - V = \begin{pmatrix} -A_1 & \frac{S_{hH}^* \beta_1 d_h r(T) \theta}{\Lambda_h} + \frac{S_{hL}^* \beta_1 d_h r(T)}{\Lambda_h} \\ \frac{S_m^* \beta_2 d_h r(T)}{\Lambda_h} & -d_m(T) \end{pmatrix}.$$

The matrix $\mathcal{B}_{11}$ is a square matrix with eigenvalues

$$-d_h, \quad -(\phi_2 + \phi_1 + d_h), \quad -\frac{K d_m(T) - (K - 2S_m^*) \sigma_m(T, R)}{K}, \quad -(\beta + d_h).$$

All these eigenvalues have negative real parts.

Next, we consider the square matrix $\mathcal{B}_{22}$, which is exactly equal to $F - V$ and represents the infection subsystem. This matrix is associated with the next-generation matrix used to determine the basic reproduction number and is also used in the local stability analysis (van den Driessche & Watmough 2002). Consequently, the characteristic polynomial of the matrix $\mathcal{B}_{22}$ is given by

$$\mathcal{P}(x) = a_2 x^2 + a_1 x + a_0, \quad (6)$$

where

$$\begin{aligned} a_2 &= 1, \\ a_1 &= A_1 + d_m(T), \\ a_0 &= A_1 d_m(T) \left(1 - \mathcal{R}_0^2(T, R) \right). \end{aligned}$$

By the Routh–Hurwitz criterion, the roots of the characteristic polynomial have negative real parts if

$$a_1 > 0 \quad \text{and} \quad a_0 > 0.$$

Clearly,

$$a_1 = A_1 + d_m(T) > 0,$$

while

$$a_0 = A_1 d_m(T) \left(1 - \mathcal{R}_0^2(T, R) \right) > 0$$

whenever $\mathcal{R}_0(T, R) < 1$. Therefore, all eigenvalues of $\mathcal{B}_{22}$ have negative real parts when

$\mathcal{R}_0(T, R) < 1$.

Hence, the disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable when $\mathcal{R}_0(T, R) < 1$ and unstable when $\mathcal{R}_0(T, R) > 1$. $\square$

3.3. Endemic Equilibrium

This subsection determines the endemic equilibrium of the model in Eq. 1, which represents the steady-state solution when malaria persists in the population.

Theorem 3.2. *The endemic equilibrium point of the model in Eq. 1 exists whenever $\mathcal{R}_0 > 1$.*

Proof. The endemic equilibrium, denoted by

$$\mathcal{E}^{**} = (S_{hL}^{**}, S_{hH}^{**}, I_h^{**}, R_h^{**}, S_m^{**}, I_m^{**}),$$

is the steady-state solution of the model in Eq. 1 in the presence of malaria in the population. The persistence of the disease is characterised by the basic reproduction number $\mathcal{R}_0$.

To obtain the endemic equilibrium, the derivatives in Eq. 1 are set equal to zero. The forces of infection λ_{h1}^{**} , λ_{h2}^{**} , and λ_m^{**} are explicitly incorporated because they determine the infection of susceptible humans and mosquitoes. Hence,

$$\begin{aligned}\lambda_{h1}^{**} &= \frac{\beta_1 r(T) I_m^{**}}{N_h^{**}}, \\ \lambda_{h2}^{**} &= \frac{\theta \beta_1 r(T) I_m^{**}}{N_h^{**}}, \\ \lambda_m^{**} &= \frac{\beta_2 r(T) I_h^{**}}{N_h^{**}},\end{aligned}$$

where

$$\begin{aligned}N_h^{**} &= S_{hL}^{**} + S_{hH}^{**} + I_h^{**} + R_h^{**}. \\ S_{hL}^{**} &= \frac{A_1 A_2 \Lambda_h \gamma \phi_2 - (A_7 \alpha \beta \delta - A_4 \Lambda_h \delta \gamma - A_1 A_2 A_7) \lambda_{h2}}{A_4 \delta \lambda_{h2} \phi_1 - (B_1 \alpha \beta \delta - A_1 A_2 B_1 - (A_1 A_2 - (\alpha \beta + A_4) \delta) \lambda_{h1}) \lambda_{h2} - (\alpha \beta \delta \lambda_{h1} - A_1 A_2 \phi_1) \phi_2}, \\ S_{hH}^{**} &= \frac{A_1 A_2 B_1 \Lambda_h \gamma - A_1 A_2 A_7 \phi_1 + (A_7 \alpha \beta \delta + (A_1 A_2 \Lambda_h - A_4 \Lambda_h \delta) \gamma) \lambda_{h1}}{A_4 \delta \lambda_{h2} \phi_1 - (B_1 \alpha \beta \delta - A_1 A_2 B_1 - (A_1 A_2 - (\alpha \beta + A_4) \delta) \lambda_{h1}) \lambda_{h2} - (\alpha \beta \delta \lambda_{h1} - A_1 A_2 \phi_1) \phi_2}, \\ I_h^{**} &= \frac{A_2 \Lambda_h \gamma \lambda_{h1} \phi_2 - A_2 A_7 \lambda_{h2} \phi_1 + (A_2 B_1 \Lambda_h \gamma + (A_2 \Lambda_h \gamma + A_2 A_7) \lambda_{h1}) \lambda_{h2}}{A_4 \delta \lambda_{h2} \phi_1 - (B_1 \alpha \beta \delta - A_1 A_2 B_1 - (A_1 A_2 - (\alpha \beta + A_4) \delta) \lambda_{h1}) \lambda_{h2} - (\alpha \beta \delta \lambda_{h1} - A_1 A_2 \phi_1) \phi_2}, \\ R_h^{**} &= \frac{\Lambda_h \delta \gamma \lambda_{h1} \phi_2 - A_7 \delta \lambda_{h2} \phi_1 + (B_1 \Lambda_h \delta \gamma + (\Lambda_h \delta \gamma + A_7 \delta) \lambda_{h1}) \lambda_{h2}}{A_4 \delta \lambda_{h2} \phi_1 - (B_1 \alpha \beta \delta - A_1 A_2 B_1 - (A_1 A_2 - (\alpha \beta + A_4) \delta) \lambda_{h1}) \lambda_{h2} - (\alpha \beta \delta \lambda_{h1} - A_1 A_2 \phi_1) \phi_2}, \\ S_m^{**} &= \frac{Q_1}{\sigma_m(T, R) (d_m(T) + \lambda_m)}, \\ I_m^{**} &= \frac{Q_2 \lambda_m}{\sigma_m(T, R) (d_m(T) + \lambda_m)}.\end{aligned}$$

where

$$Q_1 = K d_m(T) (\sigma_m(T, R) - d_m(T)),$$

$$Q_2 = K (\sigma_m(T, R) - d_m(T)),$$

and

$$A_1 = \nu + d_h + \delta, \quad A_7 = (1 - \gamma) \Lambda_h, \quad A_2 = d_h + \phi_1,$$

$$A_3 = \phi_2 + d_h, \quad A_4 = (1 - \alpha)\beta.$$

By substituting I_m^{**} into λ_{h1}^{**} and λ_{h2}^{**} , then substituting λ_{h1}^{**} and λ_{h2}^{**} into I_h^{**} , and finally substituting I_h^{**} into λ_m^{**} , we obtain

$$g(\lambda_m) = B_3\lambda_m^3 + B_2\lambda_m^2 + B_1\lambda_m. \quad (7)$$

Following the approach of Chacha *et al.* (2025b) and Tchoumi *et al.* (2022), $\lambda_m = 0$ corresponds to the trivial equilibrium. For the endemic equilibrium, $\lambda_m \neq 0$, and division by λ_m yields

$$B_3\lambda_m^2 + B_2\lambda_m + B_1 = 0, \quad (8)$$

where

$$\begin{aligned} B_3 = & (N_h^*)^2 Q_2 \beta \beta_1 d_h \delta r(T) \sigma_m(T, R) \theta (1 - \alpha) + 2(N_h^*)^2 Q_2 \beta \beta_1 \delta \phi_1 r(T) \sigma_m(T, R) \theta (1 - \alpha) \\ & + (N_h^*)^2 Q_2^2 \beta \beta_1^2 d_h r^2(T) \theta + (N_h^*)^2 Q_2^2 \beta_1^2 d_h(T)^2 r^2(T) \theta + (N_h^*)^2 Q_2^2 \beta_1^2 d_h \delta r^2(T) \theta \\ & + (N_h^*)^2 Q_2^2 \beta \beta_1^2 v r^2(T) \theta + (N_h^*)^2 Q_2^2 \beta_1^2 d_h v r^2(T) \theta \\ & + (N_h^*)^2 Q_2 \beta \beta_1 r(T) \sigma_m(T, R) \left(\theta(T)^2 - \alpha \delta \phi_2 \right) + (N_h^*)^2 Q_2 \beta_1 d_h^3 r(T) \sigma_m(T, R) \theta \\ & + (N_h^*)^2 Q_2 \beta_1 d_h^2 \delta r(T) \sigma_m(T, R) \theta \\ & + (N_h^*)^2 Q_2 \beta \beta_1 d_h v r(T) \sigma_m(T, R) \theta + (N_h^*)^2 Q_2 \beta_1 d_h^2 v r(T) \sigma_m(T, R) \theta \\ & + (N_h^*)^2 Q_2 \beta \beta_1 d_h \phi_1 r(T) \sigma_m(T, R) \theta + (N_h^*)^2 Q_2 \beta_1 d_h(T)^2 \phi_1 r(T) \sigma_m(T, R) \theta \\ & + (N_h^*)^2 Q_2 \beta_1 d_h \delta \phi_1 r(T) \sigma_m(T, R) \theta + (N_h^*)^2 Q_2 \beta \beta_1 v \phi_1 r(T) \sigma_m(T, R) \theta \\ & + (N_h^*)^2 Q_2 \beta_1 d_h v \phi_1 r(T) \sigma_m(T, R) \theta \\ & + (N_h^*)^3 \beta d_h \phi_1 \phi_2 (\sigma_m(T, R))^2 + (N_h^*)^3 d_h^2 \phi_1 \phi_2 (\sigma_m(T, R))^2 \\ & + (N_h^*)^3 \beta \delta \phi_1 \phi_2 (\sigma_m(T, R))^2 + (N_h^*)^3 d_h \delta \phi_1 \phi_2 (\sigma_m(T, R))^2 \\ & + (N_h^*)^3 \beta v \phi_1 \phi_2 (\sigma_m(T, R))^2 + (N_h^*)^3 d_h v \phi_1 \phi_2 (\sigma_m(T, R))^2 > 0, \\ B_2 = & -A_2 A_7 Q_2^2 \beta_1^2 \beta_2 r^3(T) \theta + A_1 A_2 B_1 N_h^2 Q_2 \beta_1 d_m(T) r(T) \sigma_m(T, R) \theta \\ & - A_4 B_1 N_h^2 Q_2 \beta_1 d_m(T) \delta r(T) \sigma_m(T, R) \theta + A_8 N_h^2 Q_2 \beta_1 d_m(T) \delta \phi_1 r(T) \sigma_m(T, R) \theta \\ & - A_2 A_5 B_1 N_h Q_2 \beta_1 \beta_2 r^2(T) \sigma_m(T, R) \theta + A_2 A_7 N_h Q_2 \beta_1 \beta_2 \phi_1 r^2(T) \sigma_m(T, R) \theta \\ & - A_4 N_h^2 Q_2 \beta_1 d_m(T) \delta \phi_2 r(T) \sigma_m(T, R) - A_2 A_5 N_h Q_2 \beta_1 \beta_2 \phi_2 r^2(T) \sigma_m(T, R) \\ & + 2A_1 A_2 N_h^3 d_m(T) \phi_1 \phi_2 (\sigma_m(T, R))^2 - A_2 A_5 Q_2^2 \beta_1^2 \beta_2 r^3(T) \theta, \\ B_1 = & \left(\Lambda_h^3 d_h^2 d_m^2(T) (\sigma_m(T, R))^2 \phi_1 \phi_2 + \Lambda_h^3 d_h d_m^2(T) \delta (\sigma_m(T, R))^2 \phi_1 \phi_2 \right. \\ & \left. + \Lambda_h^3 d_h d_m^2(T) v (\sigma_m(T, R))^2 \phi_1 \phi_2 \right) \left(1 - \mathcal{R}_0^2 \right) \\ & + \left(\Lambda_h^3 d_h d_m^2(T) (\sigma_m(T, R))^2 \phi_1 \phi_2 + \Lambda_h^3 d_m^2(T) \delta (\sigma_m(T, R))^2 \phi_1 \phi_2 \right. \\ & \left. + \Lambda_h^3 d_m^2(T) v (\sigma_m(T, R))^2 \phi_1 \phi_2 \right) \beta \left(1 - \mathcal{R}_0^2 \right). \end{aligned}$$

Since $B_1 < 0$ whenever $\mathcal{R}_0 > 1$, it follows that the endemic equilibrium exists for $\mathcal{R}_0 > 1$. $\square$

The coefficient B_3 in the quadratic equation 8 is always positive. The coefficient B_1 is

positive when $\mathcal{R}_0 < 1$ and negative when $\mathcal{R}_0 > 1$. The coefficient B_2 determines the possibility of backward bifurcation in the model in Eq. 1. Therefore, the quadratic equation yields the following results.

Theorem 3.3. *The model in Eq. 1 has the following properties:*

- (1) A unique endemic equilibrium exists when $\mathcal{R}_0 > 1$, corresponding to $B_1 < 0$.
- (2) A unique endemic equilibrium exists if $B_2 < 0$ and either $B_1 = 0$ or $B_2^2 - 4B_3B_1 = 0$.
- (3) Two endemic equilibria coexist if $\mathcal{R}_0 < 1$, $B_1 > 0$, $B_2 < 0$, and $B_2^2 - 4B_3B_1 > 0$.
- (4) No endemic equilibrium exists otherwise.

A unique endemic equilibrium of the model in Eq. 1 exists in case (i) when $\mathcal{R}_0 > 1$. In case (ii), a unique endemic equilibrium also exists at $\mathcal{R}_0 = 1$. In case (iii), a backward bifurcation may occur when $\mathcal{R}_0 < 1$. The critical value of $\mathcal{R}_0$, denoted by $\mathcal{R}_0^c$, is given by Eq. 9. This critical value determines the threshold at which backward bifurcation may occur in the model.

$$\mathcal{R}_0^c = \sqrt{1 - \frac{B_2^2}{4B_3\Theta}}. \quad (9)$$

where

$$\begin{aligned} \Theta = & \left(\Lambda_h^3 d_h^2 d_m^2(T) (\sigma_m(T, R))^2 \phi_1 \phi_2 + \Lambda_h^3 d_h d_m^2(T) \delta (\sigma_m(T, R))^2 \phi_1 \phi_2 \right. \\ & \left. + \Lambda_h^3 d_h d_m^2(T) \nu (\sigma_m(T, R))^2 \phi_1 \phi_2 \right) \\ & + \left(\Lambda_h^3 d_h d_m^2(T) (\sigma_m(T, R))^2 \phi_1 \phi_2 + \Lambda_h^3 d_m^2(T) \delta (\sigma_m(T, R))^2 \phi_1 \phi_2 \right. \\ & \left. + \Lambda_h^3 d_m^2(T) \nu (\sigma_m(T, R))^2 \phi_1 \phi_2 \right) \beta. \end{aligned}$$

4. Model Fitting and Parameter Estimation

This section presents the validation of the model. The model is fitted to monthly reported malaria cases in Tanzania from September 2022 to December 2023 (Health Ministry Tanzania 2023). In addition to the malaria case data, monthly temperature and rainfall records for the same period were obtained from the Tanzania Meteorological Authority (TMA). These climatic variables facilitate the estimation of parameters that depend on temperature, rainfall, or both and influence malaria transmission dynamics. Incorporating these factors enables an assessment of the model's ability to reproduce realistic disease dynamics (Asamoah *et al.* 2020).

The least-squares method is employed following the approaches of Asamoah *et al.* (2020) and Joë *et al.* (2024). The method estimates the model parameters by minimising the discrepancy between the observed data and the corresponding model solutions (Sene 2019). Mathematically, the objective function is given by

$$\min \sum_{i=1}^{16} (x_i - f(y_i))^2, \quad (10)$$

where x_i represents the observed number of confirmed malaria cases and $f(y_i)$ denotes the corresponding model solution.

The estimated parameters obtained using Python's `scipy.optimize.curve_fit` function are presented in Table 5, while Figure 2 illustrates the agreement between the model solution and the observed data. However, not all parameters were estimated through model fitting; some

were calculated using formula-based approaches described by Chacha *et al.* (2025b) and Kifle & Obsu (2023).

For instance, based on the reported number of malaria-related deaths in 2022, which was 430,058 (Health Ministry Tanzania 2023), and a total human population of 65,497,748 (World Bank 2023), the disease-induced death rate is calculated as

$$\nu = \frac{430058}{365 \times 65497748} \times 30 \\ \approx 0.00053967 \text{ per month.}$$

Similarly, using Tanzania's life expectancy of 66.77 years in 2022 (World Bank 2023), the natural death rate is calculated as

$$d_h = \frac{1}{66.77 \times 365} \times 30 \\ \approx 0.00123097 \text{ per month.}$$

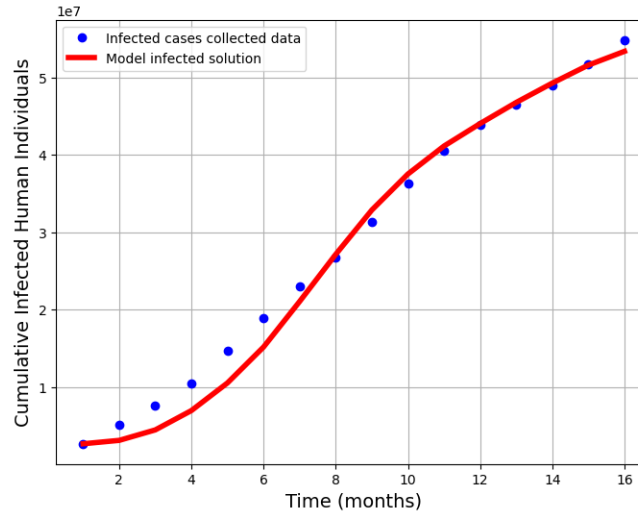


Figure 2: Model fitting to the reported human malaria cases, temperature, and rainfall data collected in Tanzania from September 2022 to December 2023

5. Sensitivity Analysis

This section investigates the parameters that play a dominant role in shaping malaria transmission dynamics by performing a sensitivity analysis of the basic reproduction number. This approach allows us to identify the parameters that most strongly influence increases or decreases in the basic reproduction number as their values vary. Consequently, changes in highly sensitive parameters may have a substantial effect on the overall transmission dynamics of malaria.

The sensitivity analysis of the model in Eq. 1 is performed using the normalised forward sensitivity index of $\mathcal{R}_0$, following the approaches of Chacha *et al.* (2025b) and Joë *et al.* (2024). Mathematically, the sensitivity index of $\mathcal{R}_0$ with respect to the parameter κ is given by

$$\Gamma_{\kappa}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \kappa} \frac{\kappa}{\mathcal{R}_0}.$$

Table 5: Parameter values used in the model

Parameter	Value	Source
Λ_h	83310	Estimated
δ	0.09509	Fitted
β_1	0.005	Fitted
β_2	0.03976	Fitted
d_h	0.08300	Fitted
β	1.56958	Fitted
θ	1.5	Assumed
ν	0.00236721	Estimated
α	0.7	Assumed
γ	0.7	Assumed
K	1.8×10^{14}	Assumed
α_1	4	Assumed
α_2	5	Assumed
α_3	6	Assumed
q_1	0.00203	Assumed
q_2	11.7	Assumed
q_3	42.3	Assumed
ϕ_1	0.6	Assumed
ϕ_2	0.7	Assumed

Table 6 presents the sensitivity indices of $\mathcal{R}_0$ with respect to the model parameters, while Figure 3 displays the corresponding partial rank correlation coefficients (PRCCs). The results indicate that Λ_h , β , K , and β_1 have relatively strong influences on malaria transmission dynamics in the model in Eq. 1.

Table 6: Sensitivity indices of $\mathcal{R}_0$ for the parameters of the model in Eq. 1

Parameter	Sensitivity index of $\mathcal{R}_0$
Λ_h	-0.50
γ	0.1296
β	0.5
θ	0.3888
ν	-0.4602
K	0.5
β_1	0.4999
β_2	0.0624
δ	0.0004199
d_h	0.9384
ϕ_1	-0.2205
ϕ_2	-0.2573

Figure 4 illustrates the relationship between the climatic variables and the basic reproduction number. For temperatures ranging from 25°C to 35°C and rainfall ranging from 10 mm to 45 mm, $\mathcal{R}_0$ generally increases as temperature rises.

Figures 5 and 6 show the sensitivity indices of $\mathcal{R}_0$ with respect to temperature and rainfall, respectively. Temperatures between 15°C and 33°C correspond to positive sensitivity indices, whereas temperatures of approximately 33°C and above correspond to negative sensitivity indices. Similarly, mean monthly rainfall below 30 mm corresponds to positive sensitivity

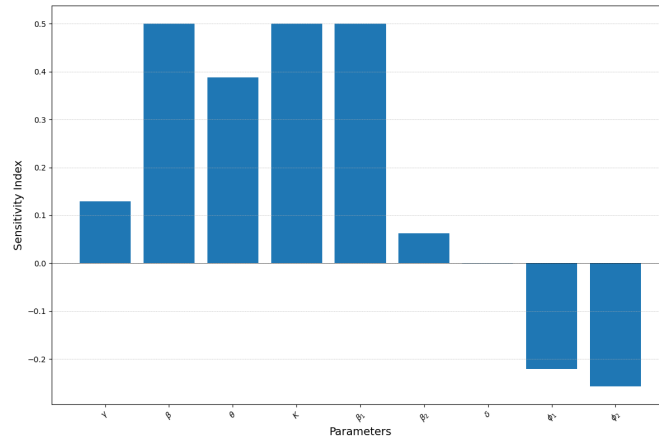


Figure 3: Sensitivity analysis of $\mathcal{R}_0$ with respect to parameters of the model (1).

indices, while rainfall above 30 mm corresponds to negative sensitivity indices.

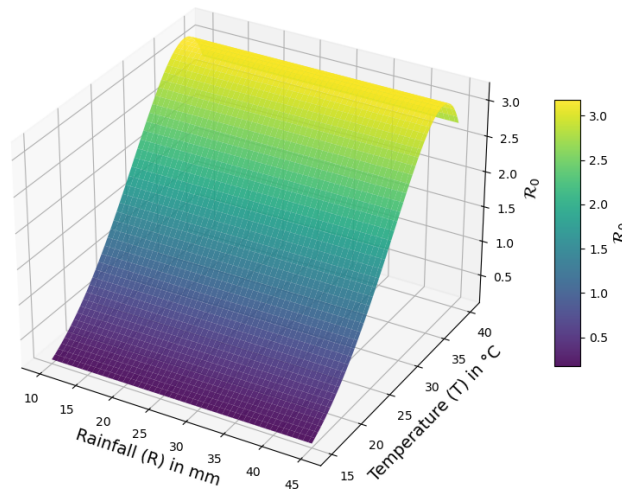


Figure 4: Basic reproduction number plotted against mean monthly rainfall and mean monthly temperature.

Figure 7 shows that the mosquito recruitment rate increases as rainfall rises from 10 mm to 30 mm and decreases when rainfall exceeds 30 mm, for mean monthly temperatures ranging from 20°C to 40°C. This pattern is consistent with the findings reported by Joë *et al.* (2024).

Figure 8 shows that the basic reproduction number increases as ϕ_1 decreases, with the effect being more pronounced than that associated with ϕ_2 . The basic reproduction number also increases as ϕ_2 decreases, although the magnitude of this effect is smaller. Overall, both parameters have relatively limited effects on $\mathcal{R}_0$ compared with the more influential parameters identified in the sensitivity analysis.

In addition, Figure 9 demonstrates that, across the considered range of mean monthly rainfall, an increase in θ leads to an increase in the basic reproduction number, indicating greater malaria transmission potential in the population. Figure 10 further shows that $\mathcal{R}_0$ increases with temperature up to approximately 37°C. Higher values of θ are associated with larger values of $\mathcal{R}_0$, while beyond approximately 37°C the basic reproduction number remains relatively high over the range considered.

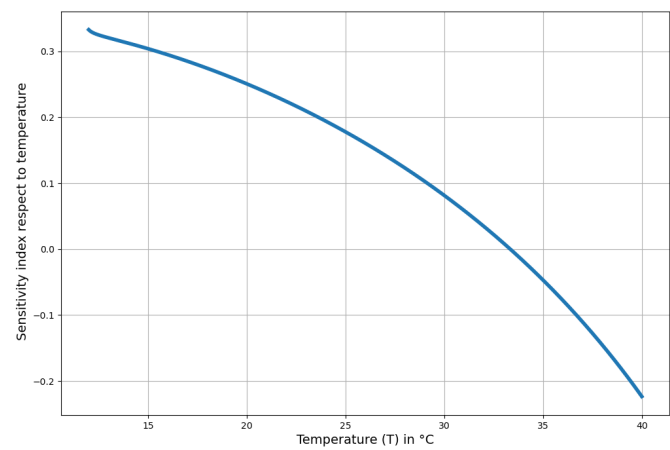


Figure 5: Sensitivity indices of $\mathcal{R}_0$ with respect to temperature.

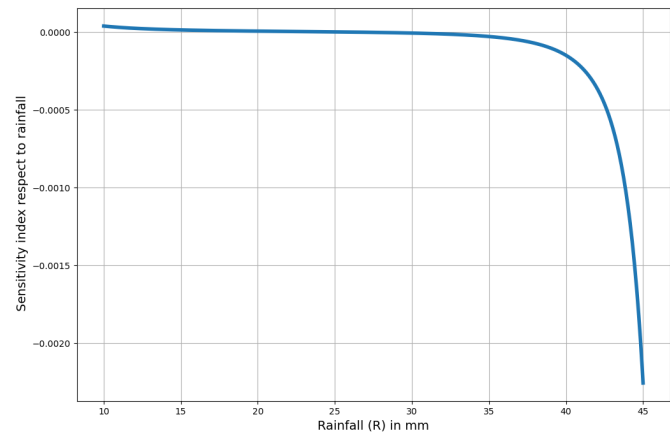


Figure 6: Sensitivity indices of $\mathcal{R}_0$ with respect to rainfall.

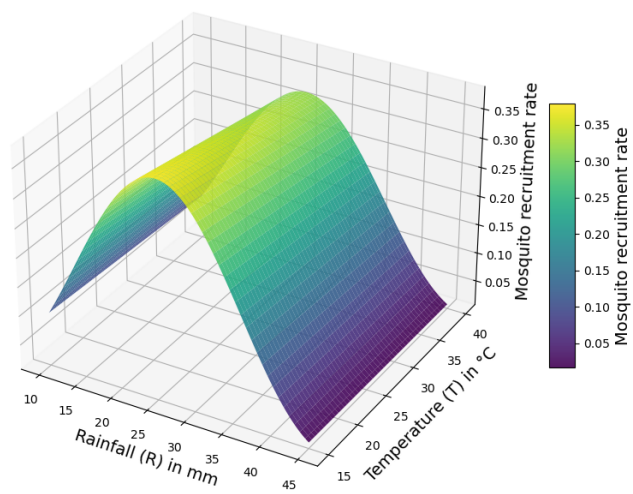


Figure 7: Mosquito recruitment rate with varying temperature and rainfall.

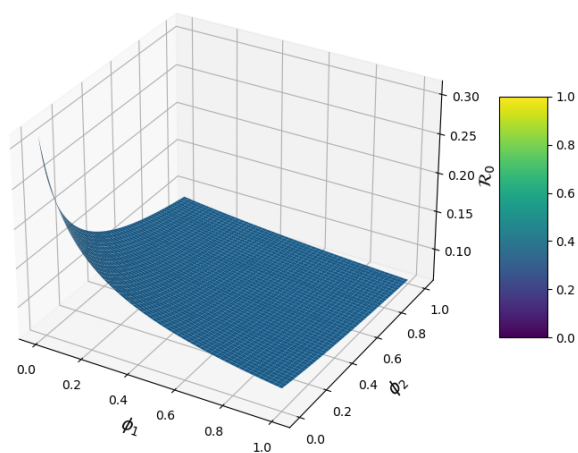


Figure 8: 3D contour plot of R_0 with respect to ϕ_1 and ϕ_2

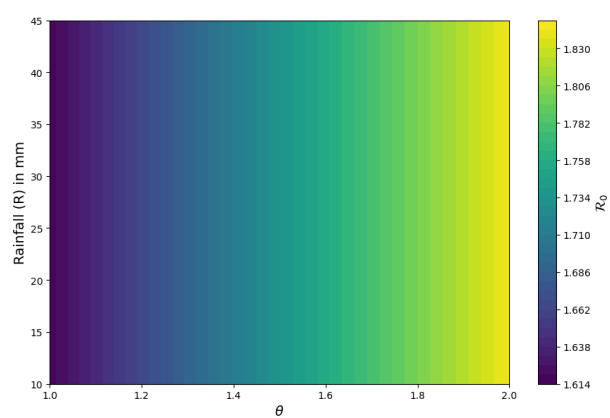


Figure 9: 2D contour plot of R_0 with respect to θ and rainfall

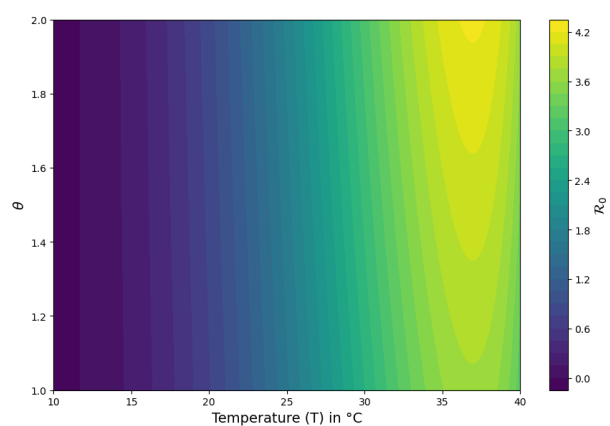


Figure 10: 2D contour plot of R_0 with respect to θ and temperature

6. Numerical Simulation

This section investigates the transmission dynamics of malaria using the model in Eq. 1, which is solved numerically using the fourth-order Runge–Kutta method in Python 3. Monthly malaria case data from Tanzania, together with monthly temperature and rainfall data, are used to investigate the model dynamics. The main objective is to examine the effects of temperature and rainfall on malaria transmission in Tanzania. Different values of temperature and rainfall are considered in the numerical simulations to determine their effects on the numbers of infected humans, susceptible mosquitoes, and infected mosquitoes.

The parameter values used in the simulations are presented in Table 5. The initial conditions for the human population are $S_{hL}(0) = 300$, $I_h(0) = 300$, $R_h(0) = 1$, and $N_h(0) = 1000$, where $S_{hH}(0) = N_h(0) - (S_{hL}(0) + I_h(0) + R_h(0)) = 399$. For the mosquito population, the initial conditions are $N_m(0) = 1800$ and $I_m(0) = 500$, with $S_m(0) = N_m(0) - I_m(0) = 1300$.

First, the effect of temperature is investigated by fixing rainfall at 25 mm and varying the temperature at 15°C, 20°C, 25°C, 30°C, 35°C, 40°C, and 50°C. Figure 11 shows that, at a relatively low temperature of approximately 15°C, the number of infected humans is also relatively low. As the temperature increases to 20°C and 25°C, the number of infected humans increases and reaches its highest level at approximately 35°C. Beyond this temperature, the number of infected humans decreases as the temperature continues to rise.

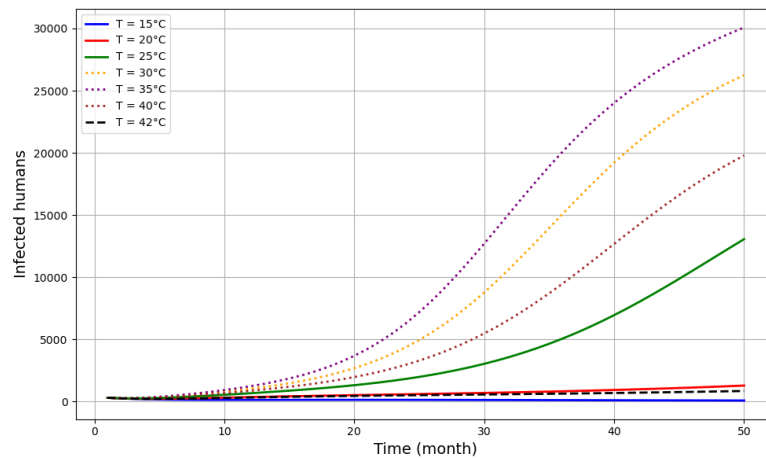


Figure 11: Infected humans with varying of temperature

Similarly, Figure 12 shows that the number of infected mosquitoes increases with temperature and reaches its highest level at approximately 35°C. Beyond this point, the number of infected mosquitoes decreases as the temperature increases further. Figure 13 illustrates the corresponding effect of temperature on the number of susceptible mosquitoes.

The effect of rainfall is then investigated by fixing the temperature at 30°C and varying rainfall at 15 mm, 20 mm, 25 mm, 30 mm, 35 mm, and 42 mm. Figure 14 shows that the number of susceptible mosquitoes increases as rainfall rises and reaches its highest level at approximately 25 mm. Beyond this point, the number of susceptible mosquitoes decreases as rainfall continues to increase.

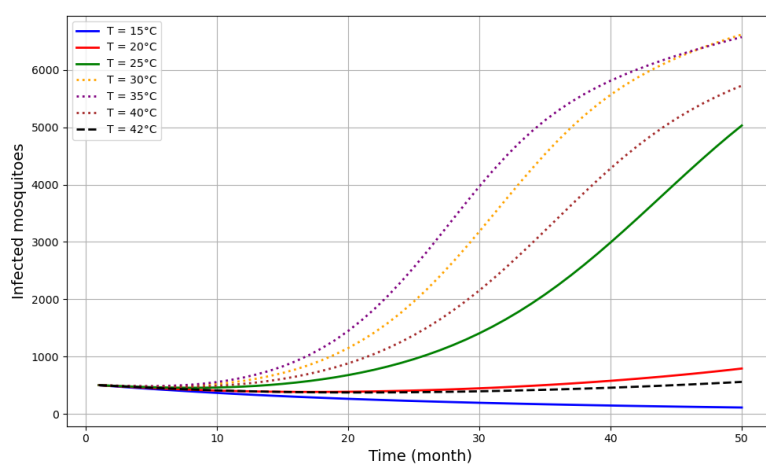


Figure 12: Infected mosquitoes with varying of temperature

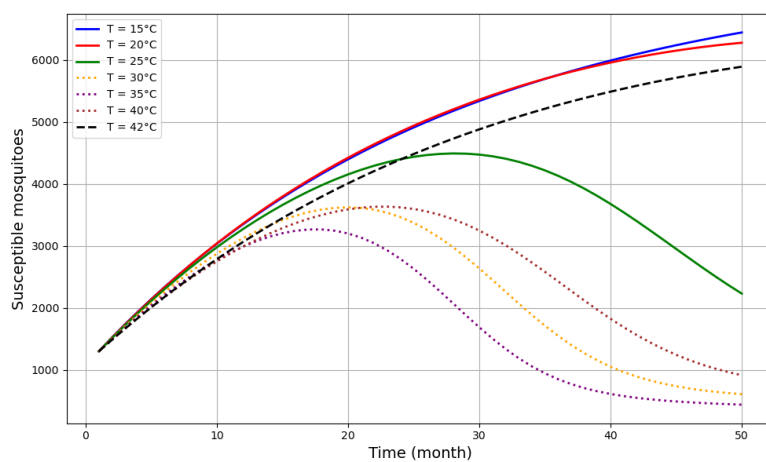


Figure 13: Susceptible mosquitoes with varying of temperature

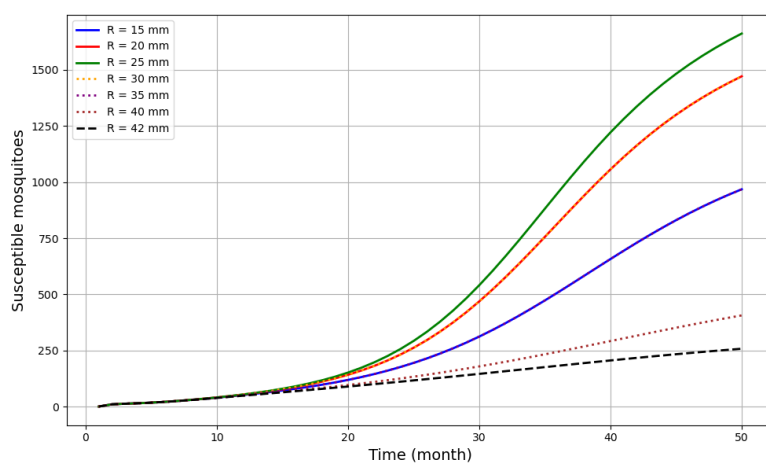


Figure 14: Susceptible mosquitoes with varying of rainfall

Similarly, Figure 15 shows that the number of infected humans increases as rainfall rises from 15 mm to 25 mm and subsequently decreases when rainfall exceeds 25 mm. Figure 16 demonstrates a similar pattern for infected mosquitoes: their number increases as rainfall rises from 15 mm to 25 mm and decreases as rainfall increases beyond 25 mm.

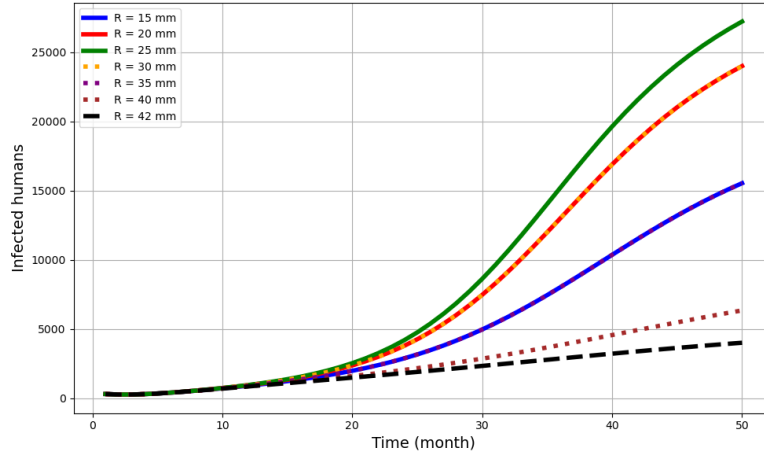


Figure 15: Infected humans with varying rainfall

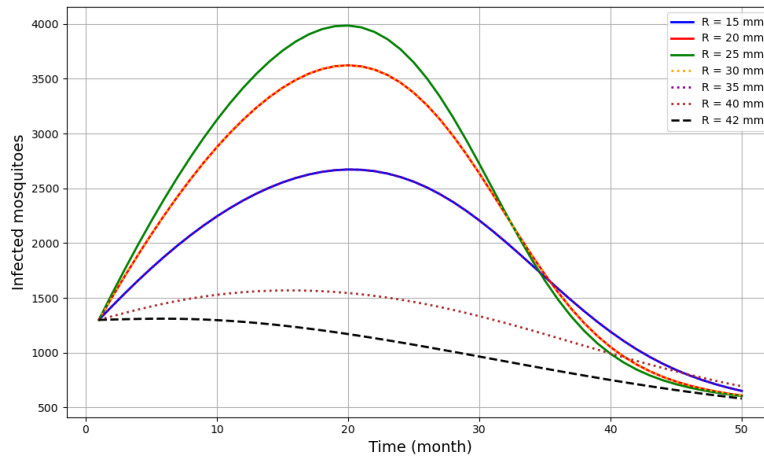


Figure 16: Infected mosquitoes with varying of rainfall

7. Conclusion

A mathematical framework is proposed in which the human population is divided into high-risk and low-risk susceptible groups, while temperature and rainfall are incorporated as key environmental drivers of malaria transmission. The model is investigated analytically and numerically. Particular attention is given to the disease-free equilibrium and its local stability in terms of the basic reproduction number, $\mathcal{R}_0$. The results show that the disease-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. The basic reproduction number is derived using the next-generation matrix approach, while the endemic equilibrium is shown to exist when $\mathcal{R}_0 > 1$. The analysis also indicates the possibility of backward bifurcation under certain parameter conditions.

To validate the proposed model, it is fitted to reported malaria case data from Tanzania together with the corresponding temperature and rainfall data using the least-squares estimation method. A sensitivity analysis based on the normalised forward sensitivity index is subsequently performed to identify the parameters that most strongly influence malaria transmission and to assess the effects of temperature and rainfall. The results indicate that temperatures ranging from approximately 15°C to 33°C are associated with positive sensitivity indices, whereas temperatures above approximately 33°C are associated with negative sensitivity indices. A positive sensitivity index indicates that an increase in temperature increases $\mathcal{R}_0$, whereas a negative sensitivity index indicates that further increases in temperature reduce $\mathcal{R}_0$. Similarly, mean monthly rainfall below approximately 30 mm is associated with positive sensitivity indices, while rainfall above 30 mm is associated with negative sensitivity indices.

The analysis further shows that an increase in the relative mosquito-biting exposure of high-risk susceptible humans, represented by θ , increases the basic reproduction number. The numerical solution of the model is obtained using the fourth-order Runge–Kutta method in Python 3 to investigate the effects of temperature and rainfall on malaria dynamics. The simulations show that the numbers of infected humans and infected mosquitoes increase as temperature rises from 15°C to approximately 35°C and subsequently decrease as temperature increases beyond 35°C. Similarly, the numbers of infected humans and infected mosquitoes increase as rainfall rises from 15 mm to approximately 25 mm and decrease when rainfall exceeds 25 mm. Overall, the findings highlight the important roles of temperature, rainfall, and heterogeneity in human susceptibility in shaping malaria transmission dynamics and suggest that these factors should be taken into consideration when developing malaria control strategies in Tanzania.

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