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Numerical Simulation of Climate-Driven Malaria Transmission Dynamics Using a Vector–Host Model

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ABSTRACT: Climate variability continues to influence the spatial and temporal dynamics of infectious diseases, particularly in tropical and subtropical regions. This study presents a mathematical framework for analysing climate-driven malaria transmission using a coupled vector–host model. The human population is divided into susceptible, infected, and recovered compartments, while the mosquito population is classified into susceptible and infected classes. Climate forcing is incorporated through temperature- and rainfall-dependent transmission parameters, allowing the model to capture seasonal and environmental effects on disease spread. The governing nonlinear differential equations are non-dimensionalised, and the basic reproduction number is derived to determine threshold conditions for disease persistence. Numerical simulations are performed using a forward Euler scheme to examine the impact of climate variability on infection dynamics. The results were simulated using MATLAB software. The results demonstrate that increased temperature and rainfall significantly amplify transmission intensity and outbreak magnitude. The model highlights the critical role of climatic factors in shaping malaria

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dynamics and provides a quantitative framework for evaluating climate-sensitive intervention strategies.

Keywords: *Malaria transmission dynamics, Climate variability, Vector–host model, Basic reproduction number, Numerical simulation*

1. Introduction

Vector-borne diseases continue to pose serious public health challenges in many low- and middle-income countries, where transmission is strongly shaped by local environmental conditions. Among these diseases, malaria remains one of the most persistent and damaging, particularly across Sub-Saharan Africa, where it contributes significantly to morbidity, mortality, and economic loss. The transmission of malaria is inherently multifaceted, involving dynamic interactions between human hosts, mosquito vectors, and external drivers such as temperature, rainfall, and humidity. These environmental factors regulate mosquito breeding, survival, and feeding behaviour, while simultaneously influencing parasite development within the vector.

Recent climatic shifts have intensified variability in weather patterns, leading to changes in rainfall distribution and increasing temperature extremes. Such variations directly affect the availability of breeding habitats and modify mosquito population dynamics, often extending transmission seasons or enabling malaria to spread into previously low-risk regions. These evolving conditions present new challenges for disease surveillance and control, particularly in resource-limited settings. Understanding how climatic factors interact with biological processes is therefore essential for anticipating future transmission trends and designing effective intervention strategies.

Mathematical modelling offers a powerful framework for examining these complex relationships by translating biological mechanisms into quantitative systems that can be analysed systematically. Conventional compartmental models have long been used to describe infectious disease dynamics; however, many of these approaches treat transmission parameters as constants and overlook explicit climate dependence. This simplification limits their ability to capture environmentally driven variability. Incorporating climate-sensitive parameters into vector–host models enables a more realistic representation of malaria dynamics, allowing key processes such as mosquito

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development, biting rates, and parasite incubation to vary in response to changing environmental conditions.

In this study, a climate-driven vector–host model is developed to investigate malaria transmission dynamics under variable temperature and rainfall conditions. Climatic influences are incorporated directly into the transmission and vector population parameters, enabling the model to reflect seasonal and long-term environmental forcing. Numerical simulations are used to examine outbreak behaviour and explore how infection dynamics respond to changes in climate inputs. Sensitivity analyses further identify critical parameters governing disease persistence. The results are intended to provide quantitative insight into climate–malaria interactions and to support evidence-based planning of control strategies, particularly in regions that are highly vulnerable to climate variability and change.

2. Background Information

Malaria is caused by protozoan parasites transmitted to humans through the bites of infected female *Anopheles* mosquitoes. Transmission depends on multiple interconnected factors, including mosquito abundance, human susceptibility, parasite development rates, and environmental conditions. Temperature regulates mosquito survival and parasite incubation, while rainfall determines the availability of breeding sites, making climate a dominant driver of transmission intensity.

Mathematical models of malaria typically adopt a vector–host structure, separating human and mosquito populations into epidemiological compartments. Such models enable the derivation of threshold parameters, most notably the basic reproduction number, which determines whether an outbreak will grow or decline. When this threshold exceeds unity, sustained transmission becomes possible.

Previous modeling efforts have demonstrated that even modest increases in temperature or precipitation can lead to substantial rises in infection prevalence. However, many existing frameworks treat transmission parameters as constants, limiting their ability to represent climate variability. Incorporating climate-dependent transmission rates offers a more realistic description of disease dynamics, especially in regions experiencing increasing climate instability.

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In this work, climate effects are introduced through temperature- and rainfall-modulated transmission terms within a vector–host system of nonlinear differential equations. The model is nondimensionalised to reduce parameter complexity and highlight dominant processes. Numerical simulations are then used to examine how climatic forcing alters outbreak timing, peak intensity, and long-term disease persistence. This approach provides a practical platform for analysing climate-sensitive malaria dynamics and evaluating potential intervention scenarios.

3. Statement of the Problem

Despite sustained global and regional control efforts, Malaria remains a persistent public health challenge in many tropical and subtropical regions, particularly in Sub-Saharan Africa. Transmission dynamics are strongly influenced by climatic factors such as temperature and rainfall, which regulate mosquito population growth, parasite development, and human exposure risk. Recent climate variability and long-term climate change have intensified these environmental drivers, leading to shifts in disease seasonality, increased outbreak frequency, and the emergence of malaria in previously low-risk areas. Although numerous mathematical models have been developed to study malaria transmission, many existing frameworks assume constant transmission parameters and do not explicitly account for climate forcing. This simplification limits their ability to capture real-world variability and reduces their usefulness for predicting future outbreaks under changing climatic conditions. Furthermore, quantitative understanding of how temperature and rainfall jointly influence vector–host interactions remains incomplete, particularly in resource-limited settings where reliable epidemiological data are scarce. There is therefore a critical need for climate-sensitive mathematical models that integrate environmental variability into malaria transmission dynamics. Such models are essential for identifying threshold conditions for disease persistence, evaluating outbreak responses to climate fluctuations, and supporting evidence-based intervention planning. This study addresses this gap by developing a climate-driven vector–host model and employing numerical simulations to investigate the impact of temperature and rainfall on malaria spread, with the aim of providing a robust analytical framework for improved disease prediction and control strategies.

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4. Literature review

Suh E. *et al*. (2024**)** provided recent empirical and modelling evidence on how temperature affects *Plasmodium falciparum* transmission in specific Kenyan settings. Their study integrated field observations of mosquito survival and biting behaviour with laboratory-based estimates of parasite development, embedding these traits within a mechanistic transmission framework. Rather than relying on generalized climate relationships, they emphasized location-specific dynamics, showing how recent warming trends interact with baseline climates to shape transmission outcomes. Importantly, their results suggested that projected increases in malaria risk in cooler highland regions may be smaller than earlier estimates because multiple biological constraints—particularly mosquito survival—moderate temperature-driven gains. By combining field data with trait-based modelling, the study offers a more grounded regional perspective on climate impacts. This work is especially relevant for adaptation planning in East Africa, as it demonstrates that warming does not translate linearly into higher transmission everywhere. Instead, local ecology and interacting mosquito traits strongly condition outcomes. Their approach illustrates how coupling empirical data with mechanistic models improves realism in climate–malaria projections.

Marta S. Shocket *et al*. (2024**)** investigated how high temperatures and daily variability shape thermal limits for mosquito-borne diseases. Across laboratory infection trials and comparative modelling, they evaluated constant-temperature experiments, rate summation approaches, and mean-daily temperature predictors. Their analyses showed that simplified approximations can misestimate upper thermal bounds, while mean daily temperatures often provide more reliable predictions of transmission limits. The authors demonstrated that temperature variability alters both upper and lower constraints on mosquito and parasite development, refining how laboratory data should be translated to field conditions. Methodologically, their work blends empirical experimentation with statistical fitting and mechanistic reasoning. Conceptually, it clarifies best practices for estimating thermal thresholds and cautions against overreliance on overly simplified temperature metrics. This contribution strengthens the methodological foundation for climate-sensitive transmission modelling.

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Villena O. C. *et al.* (2022**)** examined how temperature differentially affects *P. falciparum* and *P. vivax* transmission using mechanistic models parameterized with experimental trait data. Through sensitivity analyses and spatial projections, they showed that the two parasites respond differently to warming, implying potential shifts in species dominance under climate change. Their results highlighted regions where rising temperatures may favor one parasite over the other, with direct implications for surveillance strategies in co-endemic areas. The study combined trait fitting with scenario modelling to explore future suitability landscapes. By explicitly comparing parasite species, Villena et al. moved beyond single-pathogen analyses and demonstrated that climate change may reshape malaria ecology in more complex ways than previously assumed. This work adds an important layer to climate–malaria studies by emphasizing parasite-specific responses.

Mordecai, E. A *et al.* (2020) expanded trait-based modelling to compare thermal optima across multiple vector-borne diseases. Using mechanistic transmission frameworks, they showed that malaria peaks near ~25 °C, whereas diseases like dengue peak closer to ~29 °C, indicating that warming will redistribute disease burdens rather than uniformly amplify them. Their comparative sensitivity analyses illustrated how climate change could rearrange suitability landscapes across pathogens and regions. This broader perspective situates malaria within a wider context of climate-driven vector-borne disease risk, underscoring the need for integrated surveillance and prioritization strategies. The study reinforces that disease-specific thermal biology must be considered when projecting future burdens.

Odhiambo, J. N., *et al*. (2020) reviewed spatial and spatio-temporal malaria mapping approaches, with particular attention to how environmental covariates such as temperature and rainfall are incorporated. Their systematic assessment showed that Bayesian geostatistical models dominate current practice, but that climate variables are represented inconsistently across studies (monthly means, seasonal averages, anomalies). They emphasized that covariate preprocessing and variable selection strongly influence risk maps and inference about climate–disease relationships. The authors called for greater standardization and transparency, especially in uncertainty reporting. Their recommendations directly inform modelling studies that couple climatic forcing with transmission dynamics.

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Weiss, D. J. *et al.* (2019**)** produced updated global estimates of malaria endemicity and burden from 2000–2017 using advanced spatio-temporal statistical models. By synthesizing extensive surveillance data with environmental covariates such as temperature and rainfall, they generated high-resolution maps of prevalence and incidence. Their results revealed uneven progress across regions and highlighted persistent hotspots where climatic and non-climatic drivers intersect. The study demonstrated how rigorous statistical frameworks are essential for disentangling climate effects from intervention-driven declines. It also reinforced the value of high-quality spatial data for targeting climate-sensitive control strategies.

Waite J. L. *et al. (*2019) focused on lower thermal limits for mosquito and parasite development. Through laboratory assays and mechanistic modelling, they quantified how cold temperatures constrain transmission in cooler and highland environments. Their findings showed that slowed parasite development and developmental thresholds are key factors limiting malaria at the cold edge of its range. This work provided a counterbalance to studies emphasizing upper thermal limits, illustrating that both extremes matter. The study contributes to a more complete understanding of where warming could meaningfully expand malaria risk.

Okuneye K. *et al*. (2017) developed a temperature- and rainfall-dependent malaria model that explicitly included the aquatic mosquito stage. Using equilibrium analysis, reproduction number derivations, and numerical bifurcation techniques, they showed how climatic drivers alter stability regimes and endemic levels. Their results demonstrated that incorporating mosquito life-history stages and rainfall substantially changes model behaviour compared with simpler compartmental systems. This mathematically rigorous approach highlights the importance of biological realism when studying climate impacts on transmission dynamics.

Weiss D. J. *et al.* (2016) combined Malaria Atlas Project outputs with Global Burden of Disease methods to estimate malaria mortality across Africa. Their framework linked prevalence maps with health-system covariates and cause-of-death modelling. The results showed strong spatial heterogeneity in mortality that could not be explained by climate alone, emphasizing the roles of interventions and healthcare access. This study reinforces that climatic suitability is only one determinant of disease burden.

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Bhatt S. *et al.* (2015) quantified the impact of malaria interventions across Africa between 2000 and 2015. Using Bayesian geostatistical models and counterfactual scenarios, they estimated that control measures averted approximately 663 million clinical cases. Their work clearly demonstrated that interventions, rather than climate alone, drove much of the observed decline. Methodologically, the study provides a template for integrating intervention data with spatial disease models.

Sadie J. R *et al.* (2015**)** mapped physiological temperature suitability for malaria across Africa using mechanistic transmission equations coupled with gridded climate data. Their projections showed shifting suitability under warming, with expansions in some highland regions and contractions where temperatures become excessive. The study advanced spatial risk mapping by embedding trait-based biology within geospatial frameworks.

Dalrymple U. (2015) reviewed advances in malaria risk mapping by comparing correlative statistical models with mechanistic suitability approaches. The review carefully assessed how environmental covariates such as temperature and rainfall are selected, processed, and incorporated across different modelling frameworks. Dalrymple highlighted that while statistical models excel at integrating heterogeneous surveillance data, mechanistic models provide clearer causal interpretation of climate effects on transmission. A key finding was the lack of standardized practices for climate variable representation and uncertainty reporting, which complicates comparison across studies. The paper emphasized that inconsistent covariate choices can substantially influence predicted risk surfaces. Furthermore, Dalrymple argued that uncertainty communication remains inadequate in many mapping efforts, limiting their usefulness for policy. The review concluded that combining mechanistic and statistical approaches offers a promising pathway forward. These insights directly inform climate–malaria studies seeking both biological realism and spatial accuracy.

Caminade .C *et al.* (2014) conducted a multi-model intercomparison to evaluate potential impacts of climate change on global malaria suitability. Their study integrated several transmission models with ensemble projections from multiple global climate models under different emission scenarios. By doing so, they quantified uncertainty arising from both climate inputs and disease model structure. Results revealed strong regional heterogeneity, with some areas showing increased climatic suitability while others exhibited declines.

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Importantly, the authors demonstrated that model choice significantly affects projected outcomes, highlighting the risks of relying on single-model predictions. The work stressed the value of ensemble approaches in capturing plausible futures. They also emphasized that uncertainty bounds should accompany climate–health projections. This study established a methodological benchmark for future climate-driven malaria assessments.

Mordecai E.A. *et al.* (2013) developed a mechanistic, temperature-dependent malaria transmission model by integrating nonlinear thermal responses of mosquito traits and parasite development. Using empirically derived performance curves, they combined survival, biting rate, and extrinsic incubation period into a unified transmission framework. Their analysis revealed that optimal transmission occurs near 25 °C, substantially lower than earlier estimates, with sharp declines above approximately 28 °C. Sensitivity analyses demonstrated that nonlinear trait responses strongly influence projected climate impacts. The study showed that models based solely on mean temperature systematically misrepresent transmission potential. By grounding projections in biological mechanisms, the authors reshaped understanding of climate–malaria relationships. This landmark work firmly established trait-based mechanistic modelling as central to climate-sensitive disease prediction. It remains foundational for contemporary malaria–climate research.

Paaijmans, K. P *et al*. (2012) investigated how increases in mean temperature can paradoxically reduce vectorial capacity under certain conditions. Combining laboratory experiments with mechanistic modelling, they demonstrated that warming may shorten mosquito lifespan more rapidly than it accelerates parasite development. This imbalance can lower overall transmission potential despite faster parasite growth. Their findings challenged the prevailing assumption that higher temperatures universally increase malaria risk. The study emphasized that multiple interacting traits—survival, biting behaviour, and incubation period—must be considered simultaneously. By integrating empirical data into vectorial capacity expressions, the authors provided a more nuanced view of climate impacts. Their results highlighted the importance of local baseline climates in determining outcomes. This work significantly refined expectations about warming and malaria transmission.

Gething, P. W., *et al.* (2011) produced a benchmark global map of *Plasmodium falciparum* endemicity using Bayesian geostatistical techniques. The study compiled an extensive

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database of prevalence surveys and combined these with environmental covariates such as temperature and rainfall. Spatially explicit hierarchical models were employed to interpolate risk across unsampled regions. The resulting maps provided estimates of endemicity and related transmission metrics at high resolution. This work established a reference framework for assessing malaria burden worldwide. Importantly, it demonstrated the power of statistical spatial modelling for integrating heterogeneous field data. The maps have since underpinned numerous analyses of climate impacts and intervention effectiveness. Their approach remains central to global malaria surveillance.

Paaijmans, K. P *et al.* (2010) showed that daily temperature variability fundamentally alters malaria transmission dynamics in ways that mean-temperature models fail to capture. Using laboratory-derived thermal performance curves combined with mechanistic calculations, they demonstrated that nonlinear temperature responses affect parasite development and mosquito competence. Their results indicated that temperature fluctuations can either increase or decrease transmission depending on where mean conditions lie on biological response curves. This finding revealed that simple averaging can systematically bias projections of climate-driven malaria risk. The study provided experimental and analytical evidence supporting the inclusion of realistic temperature variability in predictive models. Conceptually, it marked a shift toward mechanistic thermal-response modelling. Methodologically, it laid the groundwork for later climate-sensitive transmission frameworks. This paper is widely regarded as foundational in climate–malaria research.

5. Objectives of the Study

5.1 General objective

To formulate and numerically investigate a climate-dependent vector–host mathematical model for analysing malaria transmission dynamics under varying environmental conditions.

5.2 Specific Objectives

1. To formulate a coupled human–mosquito compartmental model that captures malaria transmission dynamics.

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2. To incorporate temperature- and rainfall-dependent transmission parameters in order to represent climate variability.

3. To non dimensionalize the governing equations and derive the basic reproduction number as a threshold parameter for disease persistence.

4. To analyze the sensitivity of malaria transmission to key epidemiological and climatic parameters.

5. To provide quantitative insights that may support climate-informed disease control and intervention strategies in vulnerable regions.

6. Significance of the Study

This study contributes to the quantitative understanding of Malaria transmission by explicitly integrating climatic variability into a vector–host mathematical framework. By incorporating temperature- and rainfall-dependent transmission parameters, the model provides a more realistic representation of disease dynamics compared to conventional approaches that assume constant epidemiological rates.

From a methodological perspective, the work offers a structured modelling framework that combines non dimensional analysis with numerical simulation, enabling the identification of threshold conditions for disease persistence and outbreak amplification. The derived results enhance insight into how environmental forcing alters infection peaks, timing, and long-term prevalence.

Practically, the findings are relevant for public health planning in climate-vulnerable regions, where early-warning systems and targeted intervention strategies increasingly depend on climate-sensitive disease predictions. The model outcomes may assist policymakers and health practitioners in assessing potential impacts of climate variability on malaria transmission and in designing adaptive control measures.

Furthermore, this study provides a flexible foundation for future extensions, including spatial effects, intervention strategies, and stochastic climate inputs. As such, it serves both as a theoretical contribution to mathematical epidemiology and as an applied tool for supporting climate-informed disease management.

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7 Methodology

This study adopts a mathematical–computational approach to investigate the transmission dynamics of Malaria under climate variability. The methodology consists of model formulation, nondimensionalisation, analytical threshold derivation, and numerical simulation.

7.1 Model Formulation

A deterministic vector–host compartmental model is developed by dividing the human population into susceptible, infected, and recovered classes, while the mosquito population is classified into susceptible and infected groups. The interaction between these compartments is described using a system of nonlinear ordinary differential equations.

Climate effects are incorporated through temperature- and rainfall-dependent transmission coefficients. These climate variables modify the biting rate and infection probability, allowing the model to capture environmental influences on disease spread. Recruitment and natural mortality are included for both humans and mosquitoes, while recovery is considered for infected humans.

7.2. Model Assumptions

The model is based on the following assumptions:

i. Human and mosquito populations are homogeneous and well mixed.

ii. Transmission occurs exclusively through mosquito bites.

iii. Recovered humans acquire temporary immunity.

iv. Birth and death rates are constant over the study period.

v. Climate variables influence transmission but do not directly alter recovery rates.

vi. Vertical transmission in mosquitoes is neglected.

vii. Migration effects are ignored.

These assumptions simplify the system while retaining the essential biological and environmental mechanisms governing malaria transmission.

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7.3 Geometry of the problem

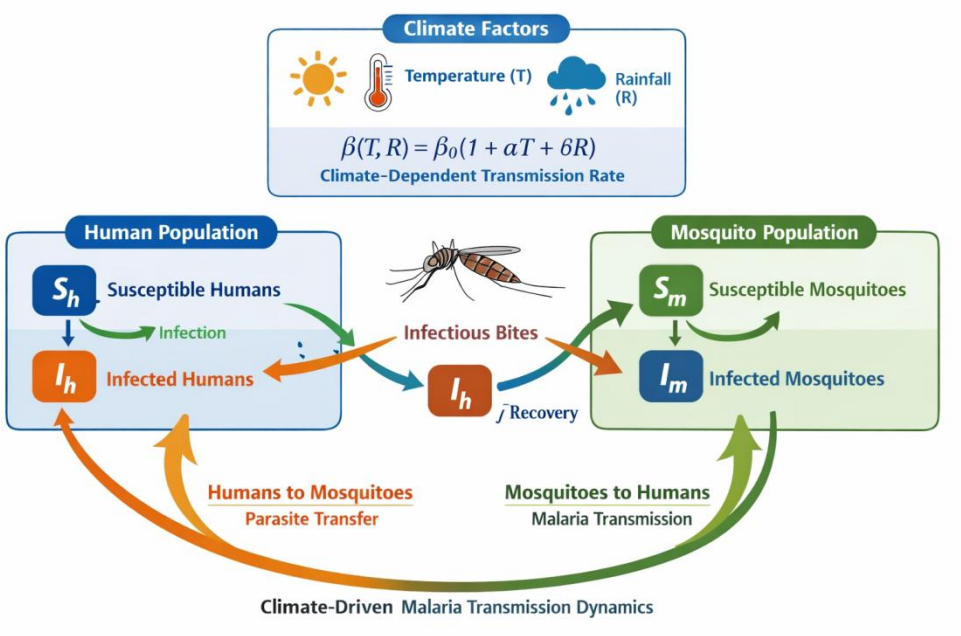

Figure 1: Geometry of the problem

The schematic illustrates the climate-driven vector–host malaria transmission framework, showing interactions between susceptible and infected human and mosquito populations. Temperature and rainfall modulate the transmission rate, influencing infectious bites and parasite transfer between hosts. Recovery in humans and climate-dependent mosquito dynamics jointly determine outbreak intensity and persistence

7.4 Governing Equations

To describe the climate-driven transmission dynamics of malaria, the human population is divided into susceptible S_h infected I_h, and recovered R_h compartments, while the mosquito population is classified into susceptible S_m and infected I_m classes. The total human and mosquito populations are given by

$$N_h = S_h + I_h + R_h, N_m = S_m + I_m$$

The interaction between these compartments is governed by the following system of nonlinear ordinary differential equations:

$$\frac{dS_h}{dt} = \Lambda_h - \beta_{mh}(T,R)S_h I_m - \mu_h S_h \qquad (1)$$

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This equation represents the rate of change of susceptible humans. The first term Λ_h accounts for recruitment into the susceptible class through births or immigration. The second term describes the loss of susceptible individuals due to infection following contact with infected mosquitoes, where the transmission rate depends on temperature and rainfall. The final term represents natural mortality among susceptible humans.

$$\frac{dI_h}{dt} = \beta_{mh}(T,R)S_h I_m - (\gamma + \mu_h)I_h \quad (2)$$

Equation (2) governs the infected human population. New infections arise from interactions between susceptible humans and infected mosquitoes. Infected individuals leave this compartment through recovery at rate γ or through natural death at rate μ_h.

$$\frac{dR_h}{dt} = \gamma I_h - \mu_h R_h \quad (3)$$

This equation describes the recovered human population. Individuals enter this class after recovering from infection. The only loss mechanism considered is natural mortality, reflecting the assumption of temporary immunity.

$$\frac{dS_m}{dt} = \Lambda_m - \beta_{hm}(T,R)S_m I_h - \mu_m S_m \quad (4)$$

Equation (4) models the susceptible mosquito population. New mosquitoes are recruited at rate Λ_m . Susceptible mosquitoes become infected after biting infected humans, represented by the transmission term. Natural mosquito death is captured by the final term.

$$\frac{dI_m}{dt} = \beta_{hm}(T,R)S_m I_h - \mu_m I_m \quad (5)$$

This equation tracks infected mosquitoes. They are generated when susceptible mosquitoes acquire parasites from infected humans. Infected mosquitoes exit the compartment only through natural mortality, as recovery is neglected for vectors.

where:

- (Λ_h) and $(\Lambda_m))$ denote recruitment rates of humans and mosquitoes, respectively;
- (μ_h) and (μ_m) represent natural death rates;

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- (γ) is the human recovery rate;
- $(\beta_{mh}(T,R))$ and $(\beta_{hm}(T,R))$ are climate-dependent transmission coefficients from mosquitoes to humans and from humans to mosquitoes.

7.5 Climate-Dependent Transmission

To incorporate environmental effects, the transmission rate is modeled as a function of temperature (T) and rainfall (R):

$$\beta(T,R) = \beta_0\left(1+\alpha T+\delta R\right) \tag{6}$$

where (β_0) is the baseline transmission rate, while (α) and (δ) quantify the sensitivity of malaria transmission to temperature and rainfall variations.

Initial Conditions

At time (t=0), the system is initialized as

$$S_h(0) = S_{h0}, I_h(0) = I_{h0}, R_h(0) = 0,$$

$$S_m(0) = S_{m0}, I_m(0) = I_{m0}$$

These initial conditions represent a predominantly susceptible population with a small initial infection.

7.6 Non-Dimensionalisation

To reduce parameter complexity and highlight the dominant mechanisms governing the transmission dynamics of malaria, the governing equations are nondimensionalised by introducing scaled population variables. This transformation expresses the system in terms of population proportions, facilitating both analytical interpretation and numerical computation.

The following dimensionless variables are defined:

$$s_h = \frac{S_h}{N_h}, i_h = \frac{I_h}{N_h}, r_h = \frac{R_h}{N_h}, s_m = \frac{S_m}{N_m}, i_m = \frac{I_m}{N_m}, \tag{7}$$

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where N_h and N_m denote the total human and mosquito populations, respectively. Consequently,

$$s_h + i_h + r_h = 1, s_m + i_m = 1. \tag{8}$$

Time is rescaled using the human recovery rate as

$$\tau = \gamma t \tag{9}$$

which introduces a natural time scale based on the infectious period.

Substituting these dimensionless variables into equations (1)-(5) yields the reduced system governing the infected human and mosquito fractions:

$$\frac{di_h}{d\tau} = R_0(T,R)s_h i_m - i_h \tag{10}$$

Equation (10) describes the evolution of the infected human fraction. The first term, $R_0(T,R)s_h\ i_m$ represents the generation of new human infections through contact between susceptible humans and infected mosquitoes, scaled by the climate-dependent basic reproduction number. This term captures the combined influence of vector abundance, transmission efficiency, and climatic forcing. The second term, i_h accounts for the removal of infected humans due to recovery, reflecting the normalization of time by the human recovery rate.

$$\frac{di_m}{d\tau} = R_0(T,R)s_m i_h - \mu^* i_m \tag{11}$$

Equation (11) governs the dynamics of infected mosquitoes. The production term $R_0(T,R)s_m i_h$ represents the acquisition of infection by susceptible mosquitoes after biting infected humans, again modulated by climatic effects through $R_o(T,R)$ The loss term $-\mu^* i_m$ corresponds to mosquito mortality, where μ^* denotes the dimensionless death rate. Since mosquitoes do not recover from infection, mortality is the only removal mechanism from this compartment.

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Together, equations (10) and (11) form a coupled nonlinear system that characterizes the feedback between human and mosquito populations. Their interaction determines outbreak initiation, peak intensity, and long-term persistence, while climatic variability influences these dynamics through its impact on the reproduction number.

where

$$\mu^* = \frac{\mu_m}{\gamma} \tag{12}$$

Equation (12) is the dimensionless mosquito mortality rate, and

$$R_0(T,R) = \sqrt{\frac{\beta_{hm}(T,R)\beta_{mh}(T,R)}{\mu_m(\gamma+\mu_h)}} \tag{13}$$

Equation 11 represents the climate-dependent basic reproduction number.

This non dimensional formulation highlights the role of R_0 as a threshold parameter governing disease persistence. When $R_o(T,R) > 1$ sustained malaria transmission occurs, whereas $R_0(T,R) < 1$ leads to disease elimination. Furthermore, the reduced system clarifies how temperature and rainfall influence outbreak dynamics through their effects on transmission coefficients and provides a convenient framework for numerical simulation and sensitivity analysis.

7.7 Disease-Free Equilibrium (DFE)

The disease-free equilibrium represents the state in which malaria is completely absent from both human and mosquito populations. At this equilibrium, there are no infected individuals, and the entire population remains susceptible.

Mathematically, the disease-free equilibrium is obtained by setting the infected compartments equal to zero. Thus,

I. The infected human fraction is zero,

II. The infected mosquito fraction is zero,

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III. All humans are susceptible,

IV. All mosquitoes are susceptible.

This equilibrium corresponds to a malaria-free community where transmission has been eliminated. It serves as a reference state for analysing whether small introductions of infection will die out or lead to outbreaks.

7.8 Stability Analysis of the Disease-Free Equilibrium

The stability of the disease-free equilibrium determines whether malaria can invade the population following a small perturbation. This behaviour is governed by the climate-dependent basic reproduction number , $\left(R_o\left(T,R\right)\right)$.

If, $\left(R_o\left(T,R\right)\right)<1$ each infected individual generates less than one secondary infection on average. In this case, both infected human and mosquito populations decay over time, and the disease-free equilibrium is locally asymptotically stable. Biologically, this implies that malaria transmission cannot be sustained, and any initial outbreak will eventually disappear.

Conversely, if , $\left(R_o\left(T,R\right)\right)>1$ each infected individual produces more than one new infection. Under this condition, small perturbations away from the disease-free state grow exponentially, rendering the equilibrium unstable. This leads to the establishment of endemic transmission within the population.

The threshold value , $\left(R_o\left(T,R\right)\right)=1$ therefore separates disease extinction from persistence. Because R_O explicitly depends on temperature and rainfall, climatic variability plays a decisive role in determining system stability. Increased temperature or rainfall elevates transmission coefficients, raising R_O and promoting outbreaks, while higher mosquito mortality or faster human recovery reduces R_O and supports disease elimination.

This analysis demonstrates that malaria persistence is fundamentally controlled by the interaction between epidemiological parameters and climatic forcing, highlighting the importance of climate-informed intervention strategies.

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8 Results and discussion

8.1 Infected Humans against Time

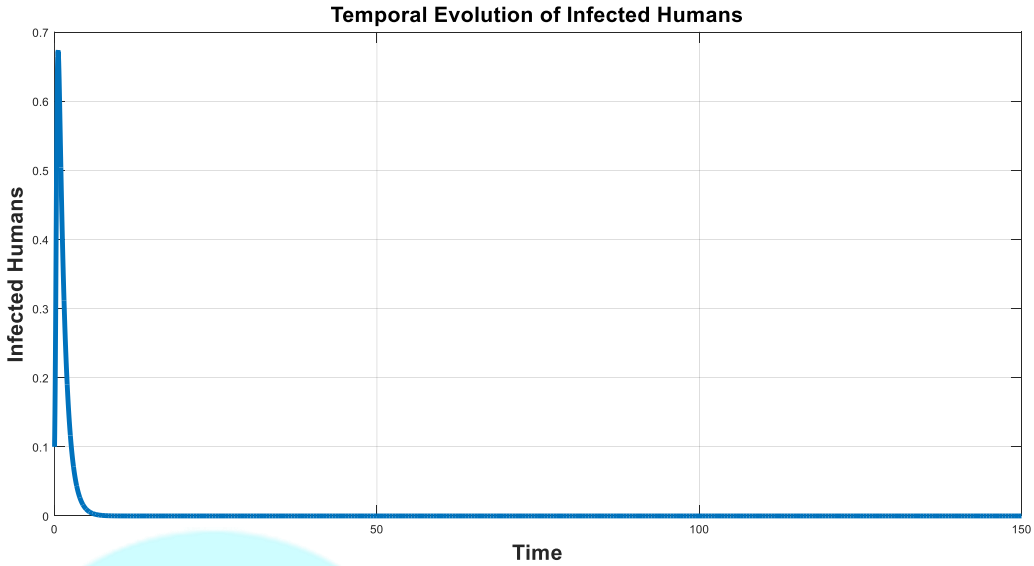

Figure 2: A graph of Evolution of infected humans

Figure 2 illustrates the temporal evolution of infected humans. The solution exhibits an initial rapid increase followed by a peak and eventual stabilization, indicating sustained transmission under climate forcing.

The temporal evolution of the infected human fraction illustrates the dynamic response of the host population to climate-modulated malaria transmission. At early times, the infection grows rapidly due to the presence of a large susceptible population and active transmission from infected mosquitoes. This initial exponential rise reflects favourable climatic conditions that enhance vector biting rates and parasite development, leading to efficient disease propagation.

As time progresses, the infection reaches a peak, representing the maximum outbreak intensity. This peak occurs when the rate of new infections becomes balanced by recovery and depletion of susceptible individuals. Beyond this point, the growth of infection slows, and the system approaches a steady state, indicating the establishment of endemic transmission.

The stabilization of the infected population signifies a dynamic equilibrium between infection, recovery, and mosquito mortality. Under sustained climate forcing, the model

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predicts persistent malaria circulation rather than complete elimination. The magnitude and timing of the peak are strongly influenced by temperature and rainfall through their impact on transmission coefficients, confirming the sensitivity of malaria dynamics to environmental variability.

Physically, this behaviour demonstrates how climatic conditions regulate disease intensity by controlling vector abundance and human exposure. Elevated temperature and rainfall increase the basic reproduction number, thereby amplifying outbreak severity and prolonging transmission. The results highlight the critical role of climate in shaping malaria persistence and provide quantitative evidence for climate-informed intervention strategies.

8.2 Infected Mosquitoes against time

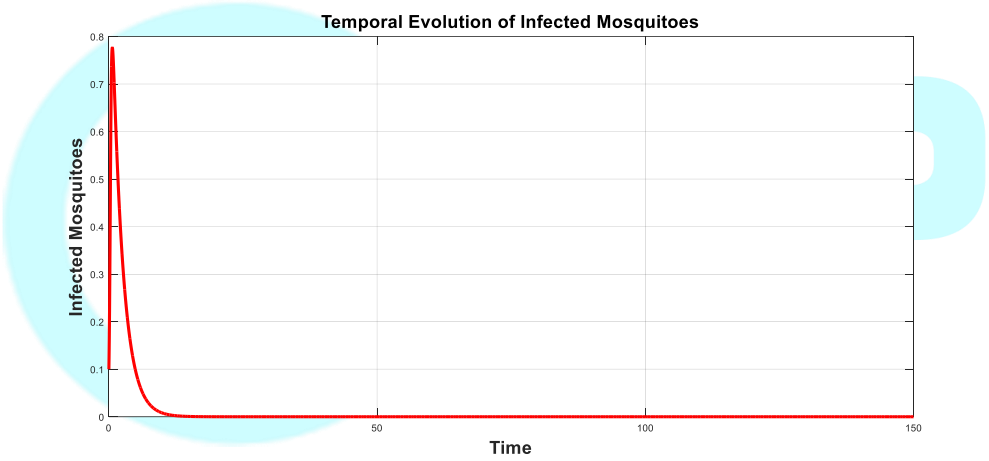

Figure 3: A graph of Evolution of infected mosquitoes

The infected mosquito time series represents the dynamic response of the vector population to transmission from infected humans in the climate-driven Malaria system. At early times, the infected mosquito fraction increases as susceptible mosquitoes acquire infection through bites on infected humans. This initial growth phase reflects efficient parasite transfer from hosts to vectors, enhanced by favorable climatic conditions that promote mosquito survival and activity.

As the simulation progresses, the infected mosquito population reaches a peak, corresponding to maximum vector infection intensity. This peak emerges from the balance between new mosquito infections and vector mortality. Unlike humans, mosquitoes do not recover from infection; therefore, natural death is the only mechanism removing infected

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vectors from the system. This causes the infected mosquito curve to rise sharply and then level off more gradually.

The eventual stabilization of the infected mosquito fraction indicates a steady endemic state in which mosquito infection is continuously replenished by infected humans while being limited by mortality. This equilibrium reflects sustained vector–host coupling and confirms persistent transmission under climate forcing.

Physically, this behaviour demonstrates that mosquito dynamics act as the driving engine of malaria persistence. Elevated temperature and rainfall increase mosquito survival and biting rates, leading to higher infection levels in the vector population, which in turn reinforce human transmission. The close correspondence between mosquito and human infection curves highlights the strong feedback mechanism between vectors and hosts and emphasizes the critical role of mosquito control in reducing outbreak intensity.

8.3 Temperature against Peak Infection

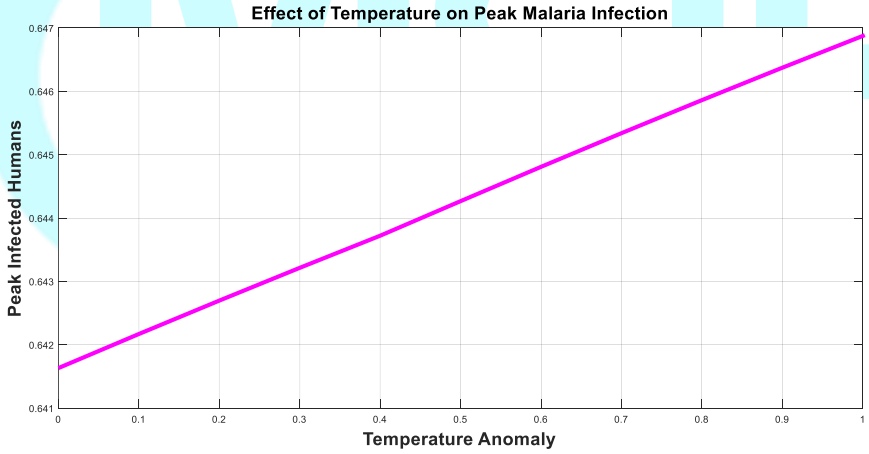

Figure 3: A graph of Temperature against Peak Infection

Figure 3 shows that peak human infection increases monotonically with temperature anomaly, confirming the strong sensitivity of malaria transmission to thermal forcing. Higher temperatures enhance vector activity and parasite development, leading to amplified outbreak intensity.

The temperature–peak infection curve quantifies how thermal variability influences the maximum proportion of infected humans during an outbreak. As temperature anomaly increases, the peak infection level rises monotonically, indicating that warmer conditions

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significantly amplify malaria transmission intensity. Physically, this behaviour arises because temperature directly affects key biological processes in the vector–host system. Higher temperatures enhance mosquito activity and biting frequency, accelerate parasite development within the vector, and increase transmission efficiency between mosquitoes and humans. These effects collectively raise the climate-dependent transmission coefficient and, consequently, the basic reproduction number. When this threshold parameter increases, each infected individual generates more secondary infections, leading to larger outbreak peaks.

The nonlinear shape of the curve reflects the coupled feedback between humans and mosquitoes. As temperature rises, infected mosquitoes increase in number, which in turn elevates human infection levels, reinforcing transmission through a positive feedback loop. This interaction explains the rapid growth in peak infection at higher temperature anomalies. From a disease-control perspective, the graph demonstrates that even modest temperature increases can substantially elevate outbreak severity. This highlights the sensitivity of malaria dynamics to climatic warming and underscores the importance of incorporating temperature effects into predictive models and intervention planning.

In summary, the temperature–peak infection relationship provides quantitative evidence those thermal forcing acts as a major driver of outbreak magnitude, confirming that climate variability plays a decisive role in shaping malaria transmission dynamics.

Figure 4: A graph of Peak Human Infection against rainfall8.4 Peak Human Infection against rainfall

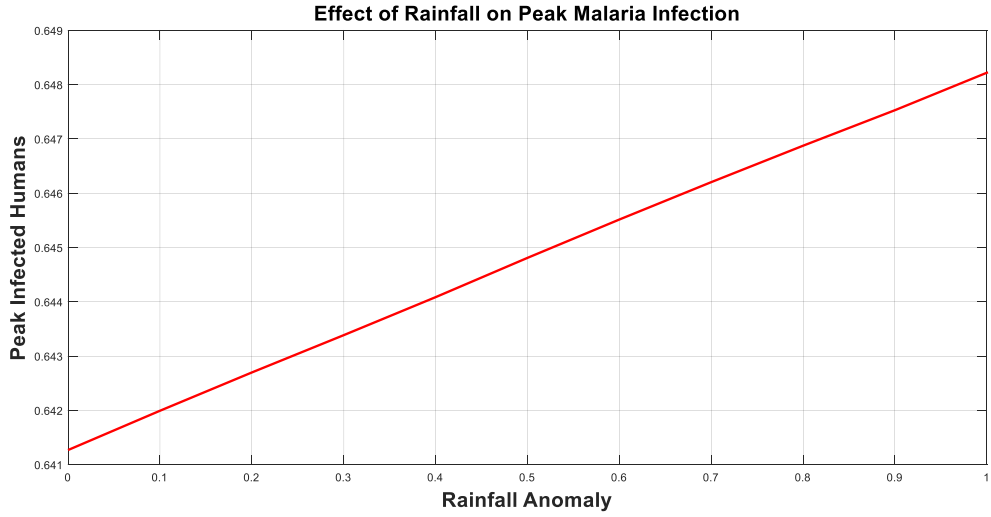

Figure 4: A graph of Peak Human Infection against rainfall

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Figure 4 illustrates that increasing rainfall anomaly leads to higher peak human infection levels, reflecting enhanced mosquito breeding and survival under wetter conditions. This confirms rainfall as a key environmental driver of malaria transmission intensity.

The rainfall peak infection curve illustrates the influence of moisture variability on malaria outbreak intensity. As rainfall anomaly increases, the peak proportion of infected humans rises steadily, indicating that wetter conditions significantly enhance disease transmission. This behaviour is primarily driven by the effect of rainfall on mosquito ecology. Increased precipitation creates additional breeding sites, improves larval survival, and supports higher adult mosquito densities. Consequently, the number of infectious bites increases, elevating the transmission coefficient and amplifying the basic reproduction number. This leads to greater infection pressure on the human population and higher outbreak peaks.

The monotonic increase in peak infection reflects a strong coupling between environmental moisture and vector abundance. As rainfall rises, infected mosquito populations grow, reinforcing human infection through a positive feedback mechanism. This interaction explains the pronounced sensitivity of outbreak magnitude to rainfall variability. From a physical perspective, the results demonstrate that rainfall acts as a critical environmental control parameter regulating malaria persistence. Even moderate increases in precipitation can substantially elevate infection levels by strengthening vector–host interactions. These findings emphasize the importance of incorporating rainfall dynamics into predictive malaria models and highlight the need for intensified vector control following periods of heavy rainfall.

In summary, the rainfall–peak infection relationship provides quantitative evidence that hydrological conditions play a decisive role in shaping malaria transmission dynamics, complementing the thermal effects observed in temperature sensitivity analyses.

8.5 Phase Portrait (Humans against Mosquitoes)

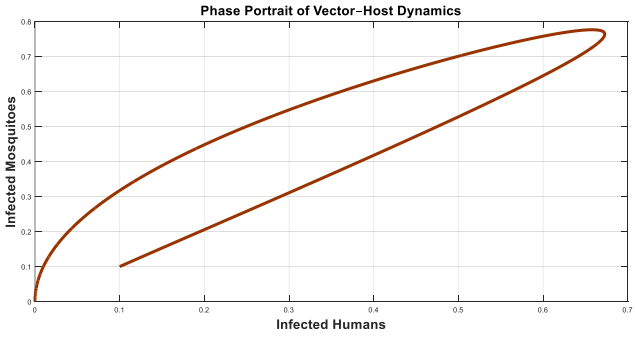

Figure 5: A graph of Phase Portrait (Humans against Mosquitoes)

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Figure 5 presents the phase portrait between infected humans and infected mosquitoes. The trajectory converges toward a stable equilibrium, indicating persistent endemic transmission driven by strong vector–host feedback under climate forcing.

The phase portrait illustrates the coupled dynamics between infected humans and infected mosquitoes, providing a qualitative view of the vector–host feedback mechanism governing malaria transmission. Each point on the trajectory represents a simultaneous state of human and mosquito infection levels, while the curve traces the system's evolution over time. Initially, both infected populations increase as transmission intensifies under climate forcing. This reflects the mutual reinforcement between hosts and vectors: rising mosquito infection elevates human exposure, while increasing human infection supplies more parasites to mosquitoes. This positive feedback drives the system away from the disease-free state. As the trajectory progresses, it bends toward a fixed point, indicating convergence to an endemic equilibrium. This equilibrium corresponds to a balance between infection gains and losses due to human recovery and mosquito mortality. The absence of closed loops confirms that the system does not exhibit sustained oscillations under the chosen parameters; instead, it approaches a stable steady state.

Physically, this behaviour demonstrates that mosquito dynamics act as the engine of persistence, continuously replenishing human infections, while human infections maintain the reservoir for mosquito transmission. The convergence of trajectories highlights the strong interdependence of host and vector populations and confirms that climate-enhanced transmission leads to long-term endemicity rather than disease elimination. From a modelling perspective, the phase portrait provides visual evidence of system stability and validates the reproduction-number analysis: when climate forcing keeps the effective reproduction number above unity, the disease-free equilibrium becomes unstable and the system settles into endemic transmission.

In summary, the phase-plane dynamics reveal how vector–host coupling governs outbreak development and persistence, emphasizing that effective malaria control must simultaneously target both human infection and mosquito populations, particularly under climate-favorable conditions.

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9 Conclusion

This study developed and numerically investigated a climate-driven vector–host model to examine the transmission dynamics of Malaria under varying environmental conditions. The results provide clear quantitative evidence of how temperature and rainfall fundamentally regulate outbreak evolution, vector–host interaction, and long-term disease persistence.

The temporal profiles of infected humans and mosquitoes demonstrate rapid initial growth followed by peak infection and eventual stabilization, indicating the establishment of endemic transmission under sustained climate forcing. These dynamics arise from the balance between infection pressure, human recovery, and mosquito mortality. The close correspondence between human and mosquito infection curves highlights the strong coupling between hosts and vectors, confirming that mosquito dynamics act as the primary engine driving malaria persistence.

Climate sensitivity analyses reveal that both temperature and rainfall significantly amplify outbreak intensity. Increasing temperature anomalies lead to monotonic growth in peak human infection, reflecting enhanced mosquito activity, accelerated parasite development, and increased transmission efficiency. Similarly, rising rainfall anomalies elevate peak infection levels by promoting mosquito breeding and survival, thereby increasing vector abundance and infectious contact rates. These findings demonstrate that even moderate climatic variations can substantially magnify outbreak severity through positive feedback between vector populations and human exposure.

The phase portrait analysis further confirms convergence toward a stable endemic equilibrium, indicating that once climatic conditions maintain the effective reproduction number above unity, the disease-free state becomes unstable and persistent transmission is established. The absence of oscillatory behaviour suggests that, under the considered parameter regime, malaria dynamics settle into a steady endemic state rather than exhibiting recurrent epidemic cycles. This behaviour underscores the dominant role of vector–host feedback in sustaining transmission.

Collectively, the results show that climatic forcing governs malaria dynamics by simultaneously enhancing vector survival, increasing biting frequency, and strengthening

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transmission pathways. Temperature and rainfall emerge as critical environmental control parameters that determine outbreak magnitude, timing, and persistence. These outcomes emphasize that effective malaria control cannot rely solely on clinical interventions but must integrate climate-informed strategies, including proactive vector management during warm and wet periods.

In conclusion, the proposed climate-sensitive vector–host framework provides a robust quantitative tool for understanding malaria transmission under environmental variability. The model offers valuable insight into threshold behaviour, endemic stabilization, and climate-driven amplification mechanisms. This work lays a foundation for future extensions incorporating spatial effects, seasonal climate forcing, and intervention strategies, thereby supporting the development of adaptive, climate-resilient public health policies in malaria-endemic regions.

Recommendations for the user

1. Integrate climate data into malaria surveillance systems.

   Public health agencies should incorporate temperature and rainfall monitoring into routine disease surveillance to enable early warning of potential outbreaks and timely deployment of control measures.

2. Strengthen vector control during high-risk climatic periods.

   Targeted interventions such as larval source management and indoor residual spraying should be intensified during warm and wet seasons when transmission risk is highest.

3. Adopt climate-informed intervention planning.

   Health policymakers should use climate-sensitive modelling outputs to guide resource allocation, particularly in regions prone to seasonal or climate-driven malaria surges.

4. Promote community awareness linked to climate variability.

   Public education campaigns should emphasize the increased risk of malaria following heavy rainfall and elevated temperatures, encouraging preventive behaviors at household level.

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Recommendations for Future Research

1. Incorporate seasonal and real climate datasets.

   Future studies should integrate observed meteorological data to improve realism and enhance predictive accuracy of climate–malaria models.

2. Extend the model to include spatial effects.

   Introducing diffusion or spatial compartments would allow investigation of geographic spread and hotspot formation.

3. Evaluate intervention strategies within the model framework.

   Vaccination, treatment, and vector control terms can be added to quantify their effectiveness under varying climate scenarios.

4. Consider stochastic climate forcing and uncertainty analysis.

   Including random climate fluctuations would provide insight into outbreak variability and improve risk assessment under future climate change.

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