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Review Article

A Review of Malaria Transmission Models: Integrating Diabetes Comorbidity and Climate Change

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A B S T R A C T

Malaria and type 2 diabetes represent two grow- ing global health challenges that are increasingly intersecting in tropical and subtropical regions. Climate change further complicates this scenario by expanding mosquito habitats and worsening metabolic complications in diabetic individuals. Re- search shows that diabetic patients face 46 percent higher malaria infection risk and significantly elevated mortality rates compared to non-diabetic individuals. This review examines the evolution of mathematical models for malaria transmission over the past century, from the foundational Ross model (1911) to contemporary frameworks incorporating climate forcing and host heterogeneity. We trace how successive generations of models have added biological realism through compartmental structures, age-dependent immunity, vector population dynamics, and environmental factors. Despite substantial progress, current models largely assume uniform host susceptibility and recovery rates, overlooking the profound impact of metabolic disorders on infection dynamics. We identify critical research gaps and emphasise the urgent need for integrated modelling approaches that explicitly account for differential transmission probabilities and recovery rates in diabetic versus non-diabetic populations under climate change scenarios. Such models are essential for protecting vulnerable communities facing the converging threats of infectious and chronic diseases in a warming world.

Keywords: Malaria Transmission, Climate Change, Compart- Mental Models, Vector-Borne Diseases, Epidemiological Modeling

Introduction

Malaria remains a persistent global health challenge, with the World Health Organization reporting approximately 260 million cases and nearly 597,000 deaths annually, predomi- nantly in tropical and subtropical regions [1]. Concurrently, the prevalence of type 2 diabetes mellitus has escalated dra- matically, with projections indicating India's diabetic popula- tion alone could reach 157 million by 2050 [2]. The con- vergence of these two disease burdens, amplified by climate change, presents an unprecedented challenge to global health systems [3]. Since 1950, global temperatures have increased by approximately 0.6◦C, fundamentally altering disease trans- mission patterns by expanding mosquito habitats and extend- ing transmission seasons.[4]

Rising temperatures influence multiple components of the malaria transmission cycle: accel- erating larval development, reducing the extrinsic incubation period of parasites, increasing vector biting rates, and simulta- neously exacerbating metabolic complications in diabetic indi- viduals [5], [6], [18]. Heat stress in diabetic patients impairs glucose metabolism, increases cardiovascular strain, and dis- rupts medication efficacy, while also increasing susceptibility to infectious diseases [18], [7]. Emerging clinical evidence demonstrates bidirectional relationships between diabetes and malaria. Diabetic individuals exhibit 46 percent higher odds of malaria infection compared to non-diabetics [8], with mortality rates reaching 35.18 percent versus 13.69 percent in non- diabetic patients.[9]

This elevated risk stems from compro- mised immune function due to chronic hyperglycemia, im- paired cellular responses to parasitic infection, and prolonged recovery periods[10],[11]. As climate change expands malaria- endemic zones into regions with high diabetes prevalence, understanding the complex interactions among vector biology, parasite transmission, host immunity, and metabolic disorders becomes critical. Mathematical models have proven essential for understanding infectious disease transmission dynamics for over a century. Sir Ronald Ross pioneered malaria modeling in 1911, demonstrating that reducing mosquito numbers below a critical threshold could interrupt transmission. His work established the foundation for quantitative epidemiology and introduced the basic reproductive number (R0) as a transmis- sion threshold indicator. Over the past century, malaria models have evolved from simple two-compartment frameworks to sophisticated multi-scale systems incorporating vector pop- ulation dynamics, host immunity, age structure, spatial het- erogeneity, drug resistance, and environmental forcing.[12]

Despite substantial progress in modelling malaria transmission and diabetes epidemiology independently, critical gaps remain. Most malaria models assume homogeneous host populations with uniform susceptibility and recovery rates, despite clin- ical evidence that metabolic disorders substantially modify infection risk and disease progression[13,15]. Diabetes progression models focus on metabolic complications without incorporating increased vulnerability to infectious diseases [14], [22]. Climate-disease models incorporate temperature effects on vector biology but rarely extend to temperature impacts on host susceptibility mediated through metabolic dysfunc- tion.[13,11,16] No published models explicitly integrate differential transmission probabilities and recovery rates for diabetic versus non-diabetic individuals within malaria trans- mission frameworks under climate forcing scenarios. [5,6,20]

This review synthesises the evolution of mathematical models for malaria transmission, with emphasis on climate change impacts and implications for diabetic populations. We trace the development from Ross's foundational work through contemporary multi-factorial models, examining how successive generations of researchers incorporated biologi- cal realism through compartmental structures, environmental forcing, host heterogeneity, and population immunity. Sec- tion II presents the basic models establishing fundamental transmission principles.

Section III chronicles the historical evolution of modelling approaches from the 1910s through the 1990s. Section IV examines complex contemporary models incorporating immunity, age structure, climate factors, and drug resistance. Section V discusses data-based statistical modeling approaches. Section VI identifies research gaps and future directions for integrated modelling of malaria-diabetes interactions under climate change. Throughout, we emphasize how model complexity has evolved to address emerging public health challenges at the intersection of vector-borne diseases, non- communicable diseases, and environmental change.

Basic Models: Foundation of Malaria Transmission Theory

The foundational mathematical models of malaria estab- lished the core principles of vector-borne disease transmission, introducing concepts that remain central to epidemiological modelling. Three seminal models—Ross

(1911), Macdonald (1957), and Anderson-May (1991)—form the trunk from which all subsequent malaria models have branched as seen from Figure 1.

- **The Ross Model (1911)**

Sir Ronald Ross developed the first deterministic differential equation model for malaria, dividing human and mosquito populations into susceptible and infected compartments [15]. The model follows an SI structure for mosquitoes (no recov- ery due to short lifespan) and an SIS structure for humans

Fig. 1. Evolution of malaria mathematical models from 1957 to present.

The progression shows increasing biological complexity: Ross-Macdonald models established basic transmission thresholds and R0 theory, SIR/SIS frameworks introduced compartmental dynamics and equilibrium analysis, age-structured models incorporated immunity development through repeated exposure, climate-integrated approaches added environmental forcing and temperature-dependent parameters, and recent comorbidity models account for host heterogeneity including differential susceptibility in diabetic populations.

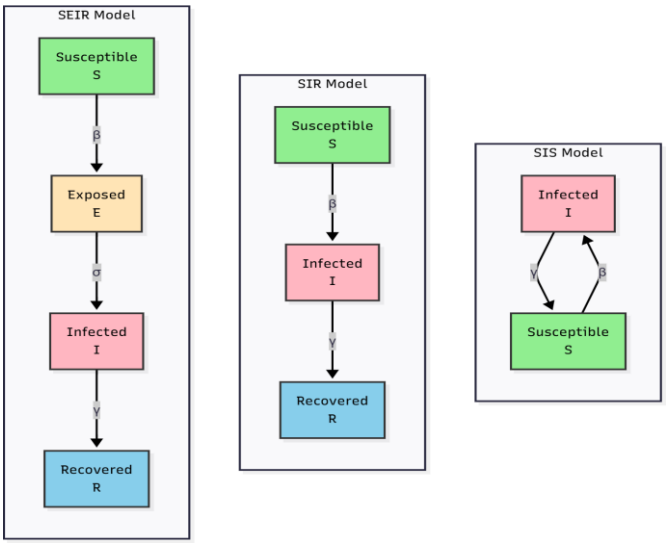

Fig. 2. Comparison of basic compartmental model structures. SIS model al- lows reinfection after recovery (malaria without lasting immunity), SIR model includes permanent recovery, and SEIR model incorporates an exposed (latent) period before infectiousness. Parameters: β (infection rate), γ (recovery rate), σ (incubation rate).

(recovered individuals return to susceptible state) as shown in Figure 2.

$$\frac{dI_h}{dt} = abmI_m(1 - I_h) - rI_h \qquad (1)$$

$$\frac{dI_m}{dt} = acI_h(1 - I_m) - \mu_2 I_m \qquad (2)$$

where Ih and Im represent infected fractions of humans and mosquitoes respectively; a is the biting rate; b is the probability of transmission to humans per infectious bite; c is the probability of mosquito infection per bite on infected human; m is the mosquito-to-human ratio; r is the human recovery rate; and μ_2 is the mosquito mortality rate. The basic reproductive number for the Ross model is:

$$R_0 = \frac{ma^2bc}{r\mu_2} \qquad (3)$$

Ross demonstrated that malaria could be eliminated not by eradicating all mosquitoes, but by reducing their density below a critical threshold where R0 < 1. The square dependence on biting rate (a2) implied that interventions reducing mosquito-human contact (such as bed nets) would be highly effective. This insight formed the mathematical foundation for vector control strategies. [21]

- **The Macdonald Model (1957)**

George Macdonald extended Ross's framework by incor- porating the latent period (τm) during which the malaria parasite develops within the mosquito, introducing an exposed compartment (Em) for vectors.[15] This created an SEI structure for mosquitoes while maintaining the SI structure for humans.

$$\frac{dI_h}{dt} = abmI_m(1 - I_h) - rI_h \qquad (4)$$

$$\frac{dE_m}{dt} = acI_h(1 - E_m - I_m) - acI_h(t - \tau_m)e^{-\mu_2\tau_m} - \mu_2 E_m \qquad (5)$$

$$\frac{dI_m}{dt} = acI_h(t - \tau_m)e^{-\mu_2\tau_m} - \mu_2 I_m \qquad (6)$$

The term e−μ2τm represents the probability of mosquito survival through the latent period. The modified basic reproductive number becomes:

$$R_0 = \frac{ma^2bc}{r\mu_2}e^{-\mu_2\tau_m} \qquad (7)$$

- **The Anderson-May Model (1991)**

Anderson and May extended the Macdonald model by incorporating human latency (τ_h), recognizing that infected humans pass through an exposed period before becoming infectious.[16] This created parallel SEIS structures for both humans and mosquitoes.

$$\frac{dE_h}{dt} = abmI_m(1-E_h-I_h) - abmI_m(t-\tau_h)e^{-(r+\mu_1)\tau_h} - (r+\mu_1)E_h \qquad (8)$$

$$\frac{dI_h}{dt} = abmI_m(t - \tau_h)e^{-(r+\mu_1)\tau_h} - (r + \mu_1)I_h \qquad (9)$$

$$\frac{dE_m}{dt} = acI_h(1 - E_m - I_m) - acI_h(t - \tau_m)e^{-\mu_2\tau_m} - \mu_2 E_m \qquad (10)$$

$$\frac{dI_m}{dt} = acI_h(t - \tau_m)e^{-\mu_2\tau_m} - \mu_2 I_m \qquad (11)$$

where μ_1 is the human mortality rate. The basic reproductive number now incorporates human latency:

$$R_0 = \frac{ma^2bc}{r\mu_2} e^{-\mu_2\tau_m} e^{-\mu_1\tau_h} \qquad (12)$$

By accounting for both human and vector latency periods, the Anderson-May model provided more realistic predictions of epidemic progression. The model showed that inclusion of human latency (approximately 10-21 days for *Plasmod- ium falciparum*) further reduced R_0 and slowed the rate of epidemic spread. This framework became the foundation for age-structured and immunity-based models developed subse- quently.

- **Comparative Analysis and Climate Implications**

Table I summarizes the evolution and key parameters of these foundational models. Each successive model reduced predicted disease prevalence by incorporating additional bi- ological realism through latency periods.

Table I: COMPARISON OF BASIC MALARIA TRANSMISSION MODELS

Model	Human Structure	Vector Structure	Key Addition
Ross (1911)	SI	SI	Transmission threshold concept
Macdonald (1957)	SI	SEI	Vector latency (τm), exponential survival
Anderson-May (1991)	SEIS	SEI	Human latency (τ_h), mortality

These basic models have direct implications for under- standing climate change impacts on malaria transmission. Temperature affects multiple parameters in the R_0 expressions:

- **Biting rate (a)**: Increases with temperature within the range 16-32°C [11]
- **Mosquito mortality (μ_2)**: U-shaped relationship with temperature, with optimal survival at 25-28°C [9]
- **Latency periods (τ_m, τ_h)**: Decrease dramatically with warming—parasite development in mosquitoes reduces from 55 days at 16°C to 7 days at 28°C [16]

The exponential terms $e^{-\mu 2\tau m}$ and $e^{-\mu 1\tau h}$ create strong nonlinear temperature dependencies. Small increases in tem- perature can simultaneously reduce latency periods and im- prove mosquito survival, leading to rapid increases in R_0 within the thermal optima window. However, these basic models do not account for host heterogeneity—particularly differential susceptibility and recovery rates in populations with metabolic disorders such as type 2 diabetes, which may exhibit altered responses to both infection and temperature stress.

HISTORICAL EVOLUTION OF MALARIA MODELS (1960S-1990S)

The decades following Macdonald's work witnessed sys- tematic efforts to incorporate biological complexities observed in field studies, particularly from large-scale epidemiological projects in Africa. These models progressively addressed lim- itations of the basic frameworks by introducing age structure, acquired immunity, spatial heterogeneity, and environmental forcing.

Age Structure and Acquired Immunity

Field observations in Africa revealed distinct age-dependent patterns in malaria prevalence that basic models could not explain [2]. Infection rates rose sharply in early childhood, peaked around age 5-10 years, then declined progressively in adolescents and adults. The basic Ross-Macdonald frame- works treated all humans as epidemiologically identical re- gardless of age, assuming uniform susceptibility and recovery rates—an assumption contradicted by field data showing dra- matically different infection prevalence across age groups.

Anderson and May addressed this by reformulating infec- tion prevalence as a function of both chronological age and calendar time, allowing infection dynamics to vary across age cohorts.[17]

However, when validated against parasitological surveys from the Garki project in northern Nigeria, these age-structured models overpredicted infection prevalence in adults and failed to capture the characteristic rapid decline in parasitemia after the childhood peak. The discrepancy revealed that age alone was insufficient—the critical missing element Dietz, Molineaux and Thomas (1974) developed the first immunity-incorporating model by dividing the human popula- tion into seven compartments based on infection and immune status. Their model introduced the concept that immunity is not permanent but wanes in the absence of new infections, and can be boosted by re-infection. The average per capita rate of immunity loss γ(h, τ) was formulated as:

$$\gamma(h,\tau) = \frac{he^{-h\tau}}{1-e^{-h\tau}} \qquad (13)$$

where h is the force of infection and τ is the duration of immunity in the absence of new infections. This for- mulation captured the observation that in high-transmission areas, immunity loss occurs very slowly because continu- ous re-exposure maintains immune memory, while in low- transmission settings, immunity wanes rapidly [7].
Building on this foundation, Aron and May (1982) pro- posed a simplified age-immunity model using three com- partments—Susceptible (S_h), Infected (I_h), and Immune (R_h)—following a SIRS structure [2]:

$$\frac{dS_h}{d\alpha} = -hS_h + rI_h + \gamma(h,\tau)R_h \qquad (14)$$

$$\frac{dI_h}{d\alpha} = hS_h - rI_h - qI_h \qquad (15)$$

$$\frac{dR_h}{d\alpha} = qI_h - \gamma(h,\tau)R_h \qquad (16)$$

was acquired immunity. The Garki project (1969-1976) provided detailed longitudi- nal data showing that individuals in endemic areas gradually acquired immunity through repeated exposure, leading to reduced disease severity and faster parasite clearance despite continued infection.[18]

where α represents age, r is the recovery rate, and q is the rate of acquiring immunity. This model successfully reproduced the characteristic age-prevalence curve observed in tropical Africa, with peak prevalence of approximately 70 percent in children aged 5-7 years, declining to 10-15 percent in adults at high transmission intensities.[2]

Vector Dynamics and Environmental Forcing

Earlier models treated mosquito population size as fixed despite clear seasonal fluctuations driven by rainfall and tem- perature. Researchers in the 1980s-1990s began incorporating explicit mosquito population dynamics with density- dependent larval mortality, allowing mosquito density to respond to environmental carrying capacity and providing a mechanistic link between habitat availability and adult mosquito abundance.[2]

By the mid-1990s, concerns about climate change motivated efforts to incorporate temperature-dependent parameters into transmission models. Martens et al. (1995) developed one of the first global malaria models driven by climate projections, parameterizing sporogonic cycle duration, mosquito survival probability, and biting rate as explicit functions of temper- ature [19]. Laboratory data showed that sporogonic duration decreased from 56 days at 16°C to 9 days at 28°C, while daily survival probability peaked around 25°C. The model projected that 2-3°C warming could expand malaria risk zones to higher latitudes and altitudes, potentially exposing an additional 260- 320 million people by 2050 [19], though these projections did not account for socioeconomic development or adaptive interventions.[20]

By the late 1990s, malaria modeling had evolved from Ross's simple two-equation system to sophisticated frame- works incorporating age structure, acquired immunity, spatial structure, vector ecology, and environmental forcing. How- ever, these models shared a common assumption of host homogeneity beyond age differences. They did not distin- guish individuals with underlying health conditions that could alter susceptibility to infection, disease severity, or recovery rates—a critical gap as type 2 diabetes prevalence increased in tropical regions where malaria remained endemic.

Contemporary Models (2010S-2025)

The past decade has witnessed fundamental shifts in malaria modeling, driven by climate change urgency, computational advances, and growing recognition of host heterogeneity. Con- temporary frameworks integrate real-time environmental data, nonlinear thermal responses, and multi-pathogen interactions that earlier models could not capture.

Climate-Integrated Transmission Models

Recent models explicitly couple temperature and humidity effects with vector and parasite biology through empirically-derived nonlinear relationships. Parham and Michael (2010) developed process-based climate-driven transmission models demonstrating that rainfall and temperature simultaneously affect mosquito population dynamics, malaria invasion, and local seasonal extinction[23]. Their analysis identified a critical temperature window around 32-33°C where endemic transmission and disease spread rates are optimized, providing mechanistic insights into how temperature shifts affect geo-graphic distribution of at-risk regions.

Mordecai et al. (2013) revolutionized temperature- transmission modeling by incorporating empirically-derived nonlinear thermal responses of mosquito and parasite traits [14]. Their model predicted optimal malaria transmission at 25°C—dramatically lower than the 31°C predicted by earlier linear models—with transmission decreasing sharply above 28°C. Validation against African entomological inoculation rate data spanning 40 years confirmed both the 25°C optimum and the high-temperature decline, fundamentally altering projections about climate change impacts on malaria distribution.

Liu-Helmersson et al. (2014) extended this framework to *Aedes aegypti*, demonstrating nonlinear relationships be- tween temperature and vectorial capacity with implications for dengue and other arboviral diseases [11]. Ghosh et al. (2024) applied similar approaches to *Anopheles stephensi*, showing that temperature and nutritional stress interact synergistically to alter vector competence, with laboratory validation that warming beyond 32°C dramatically reduces mosquito survival and biting rates.[9]

Arquam et al. (2020) integrated environmental tempera- ture directly into SIR frameworks for vector-borne diseases, demonstrating that seasonal forcing creates complex dynamics including multi-year cycles and threshold behaviors [3]. Nur et al. (2018) applied Lyapunov stability analysis to climate- driven dengue transmission, establishing mathematical condi- tions under which climate variability destabilizes disease-free equilibria [16]. Ryan et al. (2020) projected shifting trans- mission risk across Africa under multiple climate scenarios, identifying regions where warming may paradoxically reduce transmission suitability while expanding seasonal transmission windows at higher elevations.[21]

Comorbidity and Host Heterogeneity Models

The intersection of climate change and non-communicable diseases has emerged as a critical modeling frontier. Ratter- Rieck et al. (2023) synthesized evidence showing that diabetic individuals face compounded risks under climate change: impaired thermoregulation during heat stress, reduced medica- tion efficacy at elevated temperatures, and increased infection susceptibility.[22] Zheng et al. (2024) quantified these effects through spatiotemporal analysis in Shandong, China, revealing that temperature and humidity jointly predict type 2 diabetes mortality with location-specific thresholds.[25]

Epidemiological studies have established that diabetes sub- stantially modifies malaria risk and outcomes. Danquah et al. (2010) demonstrated that type 2 diabetes patients exhibit 46 percent higher odds of malaria infection compared to non-diabetics [6]. Wyss et al. (2017) extended these findings through Swedish nationwide data, confirming that obesity and diabetes constitute risk factors for severe *Plasmodium falciparum* malaria [24]. Thatoi (2018) documented that mor- tality rates reach 35.18 percent in diabetic malaria patients versus 13.69 percent in non-diabetics, with prolonged recovery periods and increased complications [20]. Carrillo-Larco et al. (2019) systematically reviewed observational studies, confirm- ing consistent associations between diabetes and both malaria incidence and severity across multiple geographic contexts.[5] Recent modeling efforts have begun incorporating these co- morbidity effects. McClure et al. (2025) developed household- level entomological measures linked to individual infection risk, demonstrating that host factors including metabolic sta- tus substantially modify transmission probability even within shared environments.[24] Nana-Kyere et al. (2024) provided asymptotic stability analysis and cost-effectiveness evaluation for stratified populations, though diabetes-specific parameters remain to be fully integrated.[15]

These advances represent initial steps toward comprehensive models capturing interac- tions among climate forcing, vector dynamics, and host het- erogeneity—a critical frontier as diabetic populations expand in malaria-endemic regions.

Future Directions

Advancing malaria modeling requires several priority shifts. First, empirical studies quantifying how specific comorbidi- ties alter infection kinetics must inform model parameteri- zation. Clinical trials measuring parasitemia clearance rates in diabetic versus non-diabetic patients would provide es- sential calibration data.[6,20] Such work should extend to other prevalent conditions including HIV, malnutrition, and cardiovascular disease [5], [24]. Methodologically, hy- brid modeling approaches combining mechanistic transmis- sion models with machine learning techniques could capture complex climate-disease interactions without oversimplifica- tion .[25,17] Agent-based models incorporating household- level transmission, human mobility patterns, and healthcare- seeking behavior offer complementary insights to determin- istic compartmental frameworks.[13] Crucially, validation against longitudinal surveillance data—not just cross-sectional snapshots—should become standard practice.[13,11] From a policy perspective, models must generate actionable out- puts. Rather than producing abstract reproduction numbers, frameworks should identify high risk subpopulations, optimal intervention timing windows, and resource allocation strategies.[15,19] As climate change accelerates and chronic disease burdens grow, integrated disease surveillance systems tracking both communicable and non-communicable conditions will become essential infrastructure.[18,25]

This review synthesized mathematical modeling approaches for malaria transmission, highlighting the evolution from sim- ple Ross-Macdonald frameworks to increasingly sophisticated representations incorporating seasonality, age structure, and immunity. While these advances improved our understanding of disease dynamics, significant gaps remain—particularly regarding comorbidity interactions and climate change im- pacts on vulnerable populations. The convergence of rising temperatures, expanding vector habitats, and growing dia- betic populations creates unprecedented challenges for malaria control in endemic regions. Future modeling efforts must move beyond treating human hosts as interchangeable units and recognize that vulnerability varies systematically across populations based on underlying metabolic conditions. As global health systems face compounding pressures from both infectious and chronic diseases, mathematical models pro- viding integrated risk assessments will prove invaluable for identifying high-risk subpopulations and informing targeted interventions. Developing frameworks that explicitly account for host heterogeneity—particularly differential susceptibility and recovery rates in diabetic versus non-diabetic individu- als under climate forcing—represents a critical frontier for epidemiological modeling. Such integrated approaches will be essential for protecting the most vulnerable communities as climate change continues to reshape disease transmission landscapes.

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