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Mathematical Models of Malaria Transmission: A Hierarchical Approach to Understanding and Control

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Abstract

Mathematical models are essential for understanding malaria transmission and guiding control interventions. Models have evolved from simple deterministic systems to complex frameworks that incorporate immunity, relapse, climate effects, and intervention strategies. A hierarchical approach was used to review classical models and modern extensions, including nonlinear incidence, fractional-order, relapse-inclusive, and optimal-control models. Special emphasis was given to the ten-compartment model that distinguishes non-immune and semi-immune humans. Latency in humans and mosquitoes reduces the basic reproduction number and enhances realism. Modern models capture partial immunity, relapse, drug resistance, climatic variability, and socioeconomic factors. The ten-compartment model provides the most comprehensive structure for heterogeneous immunity and detailed vector–host dynamics. Malaria models have advanced significantly, supporting evidence-based interventions. Future research should integrate climate impacts, data-driven calibration, and immunity dynamics to improve predictive power and guide sustainable malaria elimination strategies.

Keywords: Ross–Macdonald model, Nonlinear incidence, Latency effects, Semi-immunity, Relapse dynamics, Ten-compartment model.

2010 Mathematics Subject Classification: 92B05, 92D30, 93A30.

1. Introduction

Vector-borne diseases, defined as illnesses transmitted by vectors like arthropods, are a major public health issue, especially in tropical and subtropical regions [11]. Vectors can be mechanical, simply transporting pathogens, or biological, allowing pathogens to multiply within them before transmission [12, 15]. Malaria, transmitted biologically through the female Anopheles mosquito, exemplifies the complex interactions between pathogens, vectors, and environmental factors. Environmental variables such as temperature,

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humidity, and water availability greatly influence the distribution and prevalence of vector-borne diseases [33]. Throughout history, malaria has had profound social, economic, and health consequences [3]. It remains a significant cause of illness and death in 109 endemic countries, especially in developing regions where poor drainage and stagnant water create ideal mosquito breeding grounds [15]. Accurate surveillance is often hindered by limited healthcare infrastructure, particularly in rural communities. In India, Plasmodium vivax is the most common malaria parasite, while Plasmodium ovale is rare [16]. Elsewhere, particularly in Africa, Plasmodium falciparum dominates, causing severe and often fatal disease [42].

The life cycle of the malaria parasite is intricate. When an infected female Anopheles mosquito bites a human, it injects sporozoites into the bloodstream, which migrate to the liver and develop into merozoites [17]. These then infect red blood cells, causing the clinical symptoms of malaria as the cells rupture. During this erythrocytic phase, the parasite digests hemoglobin and modifies the red blood cell's membrane to ensure survival. Some merozoites differentiate into male and female gametocytes, which are ingested by mosquitoes during subsequent blood meals, continuing the transmission cycle. Within the mosquito, gametocytes develop into gametes, fertilize, and form ookinetes that eventually mature into oocysts. These oocysts release thousands of sporozoites, which migrate to the mosquito's salivary glands, ready to infect another human host [17, 26]. As shown in Figure 1, the mosquito life cycle consists of different stages.

Historically, infectious diseases have profoundly shaped human civilization and economies. While vaccines have successfully prevented illnesses such as smallpox and tetanus, other diseases like HIV still lack effective immunization options [8, 23]. Malaria, a biological vector-borne disease that multiplies within the mosquito before being transmitted to humans, remains endemic in 109 countries, primarily in tropical and subtropical zones [5, 10]. Despite extensive research, no effective vaccine has been developed, and the emergence of drug-resistant parasite strains threatens to undermine current treatment options. Malaria affects an estimated 300–500 million people each year, resulting in 1.5–3 million deaths, predominantly among young children [22]. Urban areas with stagnant water provide ideal breeding grounds for Anopheles mosquitoes, exacerbating the spread of the disease [16].

Obtaining precise malaria statistics is challenging, especially in rural areas with limited healthcare access [11]. In India, Plasmodium vivax accounts for around 60% of infections [11], causing symptoms such as fever, chills, diarrhea, and fatigue [8, 11, 34]. Plasmodium ovale, though less common, can remain dormant in the liver for up to four years, leading to recurrent infections, but contributes to less than 1% of malaria cases in India. Malaria's burden is substantial across Africa, Southeast Asia, and South and Central America [44]. While Plasmodium vivax is widespread, the most severe malaria cases are caused by Plasmodium falciparum, responsible for 90% of malaria deaths and 80% of cases, particularly in Africa, Southeast Asia, and South America [33]. This parasite can cause severe symptoms including neurological complications, significantly increasing the disease's fatality rate [8, 15].

Mathematical models offer a powerful means to represent these complex biological processes and transmission dynamics. By simplifying and focusing on key elements, models enable researchers to analyze disease spread, evaluate control strategies, and predict outbreaks. Several global initiatives have targeted vector-borne diseases, but their resurgence highlights challenges like resistance to drugs and insecticides, public health policy shifts, and environmental changes. Mathematical modeling provides a framework to interpret empirical data, guide control measures, and optimize resource allocation in the fight against malaria [33]. Thus, this review focuses on mathematical modeling approaches for malaria transmission, aiming to provide an accessible synthesis for mathematicians, biologists, and public health professionals seeking to understand and combat this devastating disease. A model is a rough approximation of the complicated world, and the construction of a model depends on the processes that are being researched and are intended to be extrapolated. By choosing aspects that appear to be crucial to the subject being investigated in disease development and dynamics, various known biological and clinical facts are integrated in a reduced form in a mathematical model [6]. Finding the most effective ways to stop the spread of a disease or eradicate it

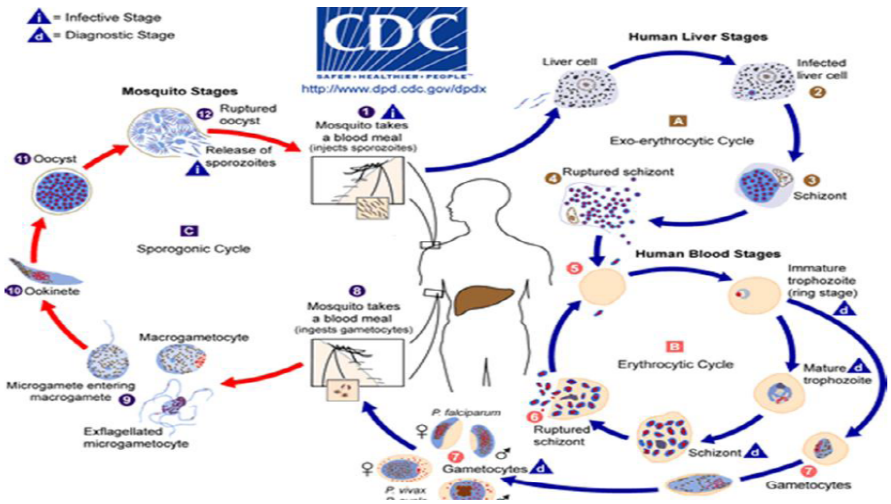

Figure 1: Architecture of the life cycle of mosquitoes

requires understanding how infections spread in terms of the number of individuals affected. In order to eradicate the diseases transmitted by vectors, numerous control initiatives were put into place worldwide [20]. Outside of Africa, the majority of these initiatives were successful. The return of vector-borne diseases is caused by a variety of circumstances. Changes in public health policies, insecticide and medication resistance, a shift away from prevention initiatives in favour of emergency preparedness, demographic and societal changes, genetic modifications of pathogens, and changes in public health policy are a few of these factors [42]. These investigations can aid in fitting empirical observations to the questions being asked and can be used to apply theory to situations that are less unknown or known enough to make predictions. The models have been rigorously analysed in order to be accessible to a broad variety of researchers studying the epidemiology, transmission, and other aspects of malaria [33]. This analysis will be helpful in identifying the various between-host models in this field and understanding how they work. This could aid mathematicians in developing more appropriate models and biologists and public health experts in improving their. Recent advances in mathematical modeling of infectious diseases have seen the application of fractional derivatives, specifically the Caputo derivative, to describe the dynamics of tuberculosis and other illnesses. [13] created a fractional model to assess tuberculosis transmission in Kenya, using real data to make more accurate predictions. Similarly, [14] used the Caputo fractional derivative for a comparative examination of TB dynamics, confirming its efficiency. [43] developed a fractional model to examine monkeypox transmission, including vaccination dynamics to evaluate its influence. These models demonstrate the expanding role of fractional calculus in infectious disease research.

This study adopts a hierarchical modeling framework because malaria epidemiology involves complexities that simpler models cannot capture. Classical Ross–Macdonald or SIR-type models assume linear incidence and homogeneous populations, overlooking relapse, immunity, heterogeneous vector–host interactions, and environmental variability. By progressively incorporating nonlinear incidence, fractional-order derivatives, and optimal control strategies, our hierarchical approach provides deeper insights into malaria dynamics. It also enables more realistic evaluation of interventions such as insecticide-treated nets, vaccination, and seasonal chemoprevention. Thus, the proposed model is both mathematically rigorous and epidemiologically relevant, bridging basic theory with practical applications in malaria control.

The primary objectives of this review are:

1. To Synthesize Existing Knowledge: The review aims to provide a comprehensive overview of the evolution of mathematical models for malaria transmission, highlighting key developments and contributions in the field.

2. To Identify Gaps in Current Research: By analyzing existing models, the review seeks to identify limitations and gaps in current research, particularly in the context of incorporating complex biological, environmental, and social factors.
3. To Inform Future Research Directions: The review aims to guide future research efforts by outlining areas that require further investigation and model refinement, particularly in light of emerging challenges such as drug resistance and climate change.

The relevance of this review lies in its potential to inform public health policy and malaria control strategies. By providing a structured analysis of mathematical modeling approaches, the review contributes to the understanding of malaria transmission dynamics and the development of effective interventions. The insights gained from this review can aid public health professionals, mathematicians, and biologists in designing targeted strategies to combat malaria, ultimately contributing to the global efforts to reduce the burden of this disease. Furthermore, as malaria continues to pose a significant public health challenge, the findings of this review are timely and critical for informing future research and intervention strategies in the fight against malaria.

2. Hierarchical Development of Malaria Models

The development of malaria transmission models has progressed through a hierarchical framework, with each successive model adding layers of biological, environmental, and social complexity to better capture disease dynamics. The foundation was laid by Ronald Ross [41], whose pioneering deterministic model emphasized the critical role of mosquito density in malaria transmission. Ross's model demonstrated that reducing mosquito populations could theoretically eliminate malaria, leading to the prioritization of vector control strategies. Later, Macdonald [35] expanded Ross's framework by incorporating the mosquito incubation period and refining the concept of the basic reproduction number (R_0). Macdonald's work established that malaria could be eradicated if $R_0 < 1$, providing a threshold-based perspective on disease control.

Building upon this foundation, Anderson and May [7] introduced human exposure compartments and applied stability analysis using R_0 within SEIR-type models. Their refinements provided deeper insights into the persistence and eradication conditions for malaria, emphasizing how biological realities could alter transmission outcomes. Figure 2 illustrates how malaria models have evolved, with each new layer enhancing the biological realism necessary to model the complex host-vector-pathogen interactions. Incorporation of Biological, Environmental, and Social Factors : Modern malaria models increasingly integrate biological, environmental, and socioeconomic parameters to provide more accurate and predictive frameworks.

Immunity and Age Structure: Immunity development plays a critical role in malaria dynamics, particularly in endemic regions where repeated exposures lead to partial and temporary immunity. Gender and age significantly influence malaria burden: in sub-Saharan Africa, malaria mortality is highest among children under five, while immunity in adults reduces disease severity. Outside Africa, limited exposure results in susceptibility persisting into adulthood. The inclusion of immunity and age structure into models has been emphasized by researchers such as Koella an Antia [33], who highlighted the necessity of modeling these factors to improve accuracy and predict vaccination outcomes. Early work by Dietz et al. [24] introduced immunity compartments into compartmental models, demonstrating a good fit with empirical data from Northern Nigeria. Later, Anderson and May [7] incorporated age structure into the classical Ross model, showing how infection dynamics vary across age groups and time. However, empirical observations suggested that simple age-immunity models did not fully capture real-world prevalence trends, prompting the development of more sophisticated immunity functions [25, 31].

Climatic Variables: Environmental factors such as temperature, rainfall, and humidity critically impact malaria transmission by influencing mosquito life cycles [37]. Recent models explicitly link mosquito density to climatic variables, where temperature-dependent factors such as biting rate, mortality, and the parasite's

extrinsic incubation period play vital roles [30, 40]. Rainfall creates breeding habitats, and temperature fluctuations significantly alter transmission dynamics, especially under changing climate conditions. These insights are crucial for understanding and predicting malaria endemicity, invasion, and extinction across regions.

Economic and Social Factors: Socioeconomic conditions profoundly affect malaria transmission. Regions with persistent malaria often experience higher poverty rates, with economic factors sometimes outweighing environmental influences. Mathematical models incorporating economic stratification, such as Yang's model [46], demonstrate how variations in health care access, infrastructure, and living standards alter disease dynamics. Simulation studies have also evaluated the efficacy of interventions like insecticide-treated nets (ITNs) and indoor residual spraying (IRS) in different socioeconomic contexts [20].

Human Mobility: Migration and Visitation: Human mobility has emerged as a critical factor in the reintroduction and spread of malaria [11, 35]. Migration involves permanent relocation, while visitation refers to temporary travel followed by return. Models that account for movement patterns illustrate how human travel facilitates disease transmission across geographic boundaries, emphasizing the need for surveillance systems and mobile health interventions.

Semi- and Non-Permanent Immunity: Models increasingly account for the non-permanence of immunity. Aron and May [10] proposed an $SIRS$ framework, where individuals could recover, develop temporary immunity, and eventually return to the susceptible state. Ngwa and Shu [38] further extended this by introducing dynamic population models that accommodate disease-induced mortality and migration. In their model, human compartments are divided into Susceptible (S_h), Exposed (E_h), Infected (I_h), and Immune (R_h) groups, while mosquito compartments include Susceptible (S_m), Exposed (E_m), and Infected (I_m) stages. Their analysis showed that if $R_0 > 1$, the disease persists, while $R_0 < 1$ leads to disease elimination.

Demographic factors must be carefully considered when estimating disease-related deaths. Chitins et al. [18, 19] introduced the concept of constant migration of susceptible individuals, showing that after accounting for immigration and removing direct recovery from the transmissible to the susceptible class, humanity tends toward either an asymptotically stable endemic equilibrium or a disease-free equilibrium, depending on the initial number of susceptibles. Anti-malarial immunity develops gradually with prolonged exposure to malaria and is better represented as a continuum of varying protection levels rather than a single category. Yang [46] modeled human immunity using three classes—immune, partially immunized, and non-immune (but retaining immunological memory)—each exhibiting different immune responses. His mathematical analysis demonstrated that these immune states delay the return of individuals to full susceptibility after infection. As immunity accumulates, communities with intense malaria exposure tend to have a lower percentage of individuals showing clinical disease or carrying contagious gametocytes, while also experiencing reduced severe disease incidence. Despite significant progress, the mechanisms underlying the development of immunity and its role in disease onset remain poorly understood due to the lack of validated immunological protection measures. Prior models treated immunologically protected individuals as a distinct class without investigating the mechanisms of immunity acquisition. Filipe et al. [25] enhanced the SEI model by including three age-specific immunity functions and classified infected individuals into asymptomatic, severely diseased, and undetectable parasite density groups. Their model incorporated the force of infection, adjusted by mosquito density, and highlighted how early-life clinical exposure decreases with age, while anti-parasitic immunity emerges later, accelerating parasite clearance.

Mathematical models play a critical role in forecasting infectious disease epidemics and guiding malaria eradication efforts [7]. Rather than relying on a single modeling style, combining multiple approaches has proven more effective [33]. The surge in eradication efforts has fueled a boom in research, leading to the development of within-host models—focusing on parasite-immune system interactions—and population ge-

netic models that study parasite evolution under variable immunity, mortality, and mosquito abundance. Ross, the Nobel Prize winner, laid the foundation for malaria modeling [41]. His simple model explained the relationship between malaria prevalence and mosquito density but neglected the parasite's latency period within mosquitoes, resulting in overestimation of the epidemic speed and infectious mosquito prevalence. Building on Ross's model, later transmission models regulated host and vector transitions through epidemiological compartments influenced by biological and environmental factors. Age and gender were also found critical, with most malaria deaths occurring before age five in Africa, while older Africans, through repeated exposure, developed partial immunity. Outside Africa, where exposure is less constant, malaria burden persists into adulthood. Including immunity in malaria models is crucial. As shown by Koella [33], omitting immunity results in unrealistic predictions, while including it not only improves model realism but also helps forecast vaccination campaign outcomes. Several studies [7, 10, 9] modeled immunity alongside human age structure, recognizing that immunity and age interact to shape disease transmission.

Some models [34] introduced a distinct immune class, while others like Filipe et al. [25] embedded complex immunity functions. Dietz et al. [24] introduced a model with seven compartments, allowing individuals to either recover directly to the susceptible class or pass through a temporary immune stage before possible reinfection. Environmental variability, such as stochastic mosquito abundance and periodic force of infection, was later incorporated into models [7, 41]. For example, Parham and Michael [40] developed a model including temperature and rainfall effects on mosquito dynamics. Environmental factors like mosquito mortality, biting rate, and the sporogonic cycle were treated as temperature-dependent, while adult mosquito birth rates were influenced by both rainfall and temperature. This model concluded that rainfall patterns greatly influence malaria endemicity and vector abundance, while temperature primarily affects transmission rates once vector populations are sustained. Socioeconomic factors were found to be even more influential than environmental ones, linking malaria closely to poverty. By modeling different economic and climatic scenarios, it was shown that disease transmission depends heavily on environmental management and healthcare systems. Beyond deterministic differential equation (DE) models, researchers explored more nuanced transmission dynamics using SEIR-type compartmental structures. Ross's foundational model, further expanded by Macdonald [35] and Anderson and May [7], introduced exposed (E) compartments to capture latency periods in both humans and mosquitoes, leading to models like SEI, SEIS, SEIR, and SEIRS [1, 36, 45].

In these models, latency periods lower the basic reproduction number (R_0) and reduce the prevalence of infection. The Macdonald model [35] added mosquito latency, splitting the mosquito population into SEI compartments, while Anderson and May [7] added human latency, leading to a SEIS structure for humans. As a result, these extensions predicted lower infection rates compared to Ross's original model. Comparative analyses among Ross [41], Macdonald [35], and Anderson and May [7] revealed that including latency better captured real-world malaria transmission, with Ross's model overestimating epidemic intensity. Importantly, these models demonstrated how vector control strategies—such as reducing mosquito density, biting rate, or lifespan—can dramatically lower malaria transmission. Specifically, increasing mosquito mortality was shown to be a particularly effective intervention. The basic reproduction number (R_0) emerges as the cornerstone of malaria control strategies. Even early models stressed its importance: a pathogen's potential to invade or be eradicated hinges on whether R_0 exceeds unity. Adding latency to mosquitoes or humans lowered R_0, highlighting interventions that shorten mosquito lifespans (e.g., insecticide-treated nets) as powerful tools for malaria control. More recently, temperature-dependent models [46] advanced the field further. These models accounted for temperature effects on mosquito development rates and Plasmodium incubation, offering more realistic dynamics of malaria transmission under changing climate conditions.

The model developed in [7] investigates the effects of global warming on malaria transmission. Using an estimated temperature increase of 1.0 to 3.5°C by the year 2100, the study shows that in certain regions, the basic reproduction number (R_0) may rise above one, causing shifts from disease-free equilibrium (DFE)

to endemic equilibrium (EE), and from low endemic levels to higher endemicity. However, the authors emphasize that economic and social factors outweigh the effects of temperature alone and that an effective healthcare system with strong malaria control programs can mitigate these negative impacts. The model considers individuals progressing through various stages of a standard SEIR framework, while retaining a history of previous infections, following the formulation derived in [7]. The mosquito population is subdivided into juvenile and adult stages through a sub-model, whose steady-state adult mosquito population is integrated into the main malaria transmission model. The mosquito sub-model introduces environmental dependence into its parameters and explores the impact of environmental changes on R_0. The spread of drug-resistant Plasmodium strains and the development of immunity have been addressed in more recent models [33]. Specifically, a model starting from the classical Ross-Macdonald framework has been extended to incorporate treatment-resistant strains. Even in basic versions of these models, there exists a threshold below which drug resistance cannot be sustained and above which it persists. In a host-parasite coevolution model, [33] demonstrate that parasites evolve mechanisms to evade host immunity while hosts gradually enhance their immune responses.

In the study by [31], the mosquito population follows an SEI structure similar to [32], while humans follow a $SEIRS$-like pattern with one immune class. Upon contact with infected mosquitoes, susceptible humans become exposed and then infectious, following traditional $SEIRS$ dynamics. Infected individuals may recover with or without gaining immunity, either returning to the susceptible class or moving to a recovered class. Uniquely, recovered individuals, although protected from severe disease, retain low parasite levels in their blood and can still infect mosquitoes. Recovered individuals eventually lose immunity and rejoin the susceptible pool. Disease-induced deaths and fluctuating mosquito populations are accounted for, unlike constant-population models. In [31], a linear per capita mortality rate is assumed, and dimensionless variables are introduced, leading to the definition of a new R_0. They prove that an endemic equilibrium (EE) exists when $R_0 > 0$ and is unique in the absence of disease-related deaths. Linear stability analysis reveals that the DFE is locally asymptotically stable (LAS) when $R_0 < 1$, while the unique EE is LAS for $R_0 > 1$. Numerical simulations support these results.

Earlier foundational work by [10] is revisited and expanded in [7]. [10] compiles critical datasets, including latent periods for humans and mosquitoes, human recovery rates, mosquito lifespans, and malaria prevalence across age groups. Both [10] and [7] examine the inclusion of age structure into malaria models, building from the Ross-Macdonald framework [41, 35]. They also discuss the impact of vaccines and declining transmission on malaria age-prevalence distributions. Nedelman conducts statistical analyses of various datasets to estimate key parameters such as inoculation rates, recovery and immunity loss rates in humans, and mosquito biting rates. [33] extends the Ross-Macdonald model by adding a latent stage for mosquitoes, examining parameter variability and introducing infection-dependent immunity duration.

In [33], the efficacy of vaccines targeting asexual blood stages is compared to those aimed at blocking transmission, concluding that blood-stage vaccines are generally more effective. The model incorporates immunity loss linked to infection rates, recognizing that continuous reinfection is crucial for sustained immunity. Without ongoing exposure, immunity wanes rapidly, while high infection rates sustain long-term protection. Unlike earlier models, [19, 21] account for continuous human immigration and structure the human population into four classes: susceptible, exposed, infectious, and immune. The mosquito population is divided into three classes: susceptible, exposed, and infectious. Recovered humans, although immune to clinical disease, can transmit infection due to persistent low parasitemia and eventually lose immunity to become susceptible again. [19, 21] also provide the first formal description of a mathematically and epidemiologically well-posed malaria model. They define the R_0, demonstrate the existence and stability of a DFE for $R_0 < 1$, and the existence of at least one EE for $R_0 > 1$. Numerical simulations show a supercritical transcritical bifurcation at $R_0 = 1$ in the absence of disease-induced deaths and a potential subcritical bifurcation at higher disease-induced death rates.

The incorporation of acquired immunity in malaria modeling, pioneered by [24], marked a major advancement. In this model, individuals either have no immunity or some level of immunity; infected individuals can recover into the immune class. Immune individuals cannot develop clinical disease or transmit malaria. The model also includes the phenomenon of superinfection, typically associated with macroparasites, as previously discussed in [7]. [11] further describes Dietz's superinfection model, emphasizing the temporary nature of acquired immunity in malaria. [10] provides comprehensive reviews of transient immunity models. In this review, we aim to highlight essential epidemiological aspects while maintaining mathematical tractability.

The architectural structure of malaria modeling is illustrated in Figure 2. The human compartments—susceptible (S_h), exposed (E_h), infectious (I_h), and recovered (R_h)—are presented on the left, while mosquito compartments—susceptible (S_m), exposed (E_m), and infectious (I_m)—are on the right. Generally, human classes cycle back to susceptibility, while infected mosquitoes ultimately die from infection. Complex factors such as age, immunity, environmental changes, and socioeconomic conditions are indicated by dotted arrows and red highlights, marking innovations in modeling. Subscripts $j = 1, 2, 3$ denote further subdivisions of compartments. With numerous models developed, extracting essential features and fully understanding the interaction between host, vector, and parasite remains a challenging but critical task.

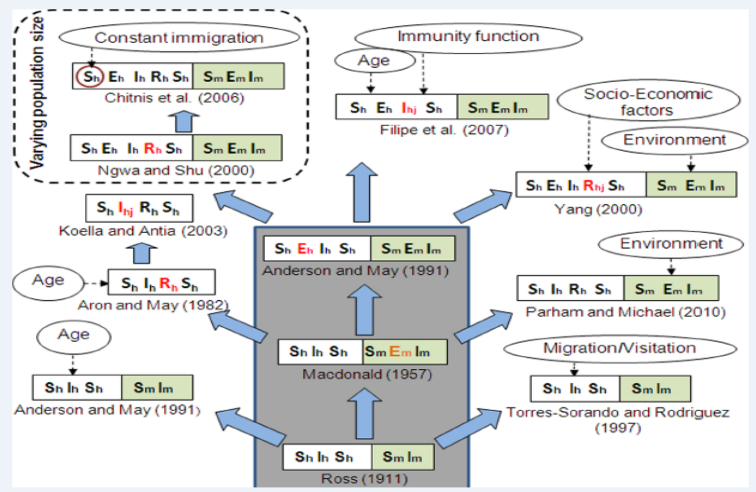

Figure 2: Architectural structure of malaria modeling development, from foundational to advanced frameworks.

The summary of malaria model evolution is depicted in Figure 2, outlining the hierarchical enhancement from simple to complex structures.

3. Modern Ten-Compartmental Model

The proposed ten-compartment malaria model captures the transmission cycle between humans and mosquitoes while distinguishing non-immune and semi-immune individuals. Non-immune humans (S_e) are recruited through birth at rate λ_e, become exposed (E_e) when bitten by infectious mosquitoes at incidence $\beta_e = \Upsilon\varphi_{ve}I_v$, progress to infectious (I_e) at rate γ_e, and recover at rate α_e, entering the semi-immune recovered class (R_a). Semi-immune susceptibles (S_a) arise either from birth at rate $\lambda_h - \lambda_e$ or from recovered individuals losing immunity at rate Ω_a. They become exposed (E_a) when bitten by infectious mosquitoes at incidence $\beta_a = \Upsilon\varphi_{va}I_v$, progress to infectious (I_a) at rate γ_a, and recover at rate α_a, joining R_a. Recovered semi-immune individuals may lose immunity and return to S_a at rate Ω_a. All human compartments are subject to natural mortality μ_h.

Mosquitoes are recruited into the susceptible class (S_v) at rate λ_v. When they bite infectious humans (I_e or I_a), they become exposed (E_v) at incidence $\beta_v = \Upsilon(\varphi_{ev} I_e + \varphi_{av} I_a)$, progress to infectious (I_v) at rate γ_v, and remain infectious until death at rate μ_v. There is no mosquito recovery, and no direct transmission between humans or between mosquitoes.

Each parameter has a clear biological interpretation: $\lambda_h, \lambda_e, \lambda_v$ represent birth or recruitment rates; μ_h, μ_v are natural death rates; Υ is the biting rate; $\varphi_{ve}, \varphi_{va}, \varphi_{ev}, \varphi_{av}$ are transmission probabilities per bite; $\beta_e, \beta_a, \beta_v$ are forces of infection; $\gamma_e, \gamma_a, \gamma_v$ are progression rates from exposed to infectious; α_e, α_a are recovery rates; and Ω_a is the rate of immunity loss. Together these flows capture the biological cycle of malaria transmission between humans and mosquitoes, while highlighting the role of immunity in shaping susceptibility and recovery. Figure 3 presents a detailed schematic of the compartmental transitions and transmission pathways between human and mosquito populations, as proposed by [27, 28, 29].

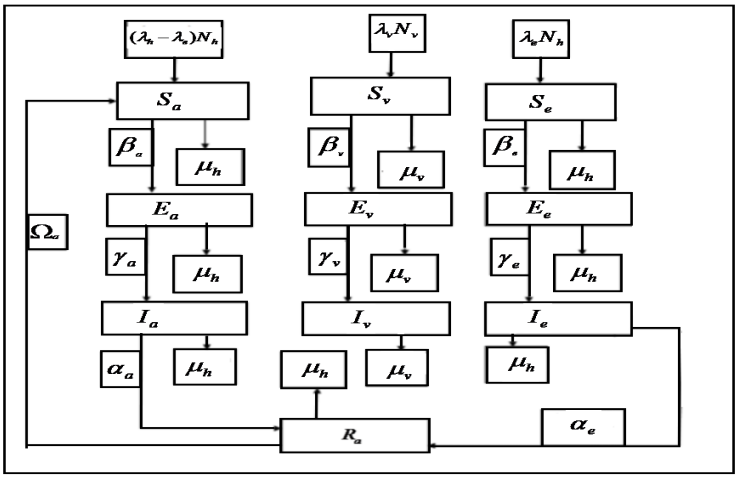

Figure 3: Schematic diagram of the ten-compartment malaria model

4. Comparative Analysis of Malaria Models

The transmission of infectious agents within host populations is the fundamental mechanism described in epidemiological theories of infectious disease. Classical compartmental notation $S-E-I-R$ originates from the pioneering work of Kariuki et al. [32], where populations are divided into susceptible (S), exposed (E), infectious (I), and recovered (R). Depending on the disease, compartments may vary, giving rise to models such as $SI, SIS, SEI, SEIS, IR, SIRS, SEIR$, and $SEIRS$. These frameworks have been progressively adapted to malaria, reflecting its unique host–vector–parasite dynamics.

Ross Model

Ross [41] introduced the first mathematical model of malaria transmission, dividing humans into susceptible (S_h) and infected (I_h), and mosquitoes into susceptible (S_m) and infected (I_m):

$$\frac{dI_h}{dt} = \beta_{mh} S_h I_m - \gamma I_h, \quad \frac{dI_m}{dt} = \beta_{hm} S_m I_h - \mu I_m, \tag{4.1}$$

where β_{mh} and β_{hm} are transmission rates, γ is the human recovery rate, and μ is mosquito mortality. This model emphasized mosquito survival as the weakest link in malaria cycling.

Macdonald Model

Macdonald [35] extended Ross's framework by incorporating mosquito latency. The mosquito population is divided into susceptible (S_m), exposed (E_m), and infectious (I_m):

$$\frac{dE_m}{dt} = \beta_{hm} S_m I_h - (\mu + \sigma) E_m, \quad \frac{dI_m}{dt} = \sigma E_m - \mu I_m, \tag{4.2}$$

where σ is the rate at which exposed mosquitoes become infectious. This addition reduced R_0 as latency increased, making the model more realistic.

Anderson–May Model

Anderson and May [7] refined Macdonald's model by adding an exposed class for humans, capturing the latent period before infectiousness. The compartments are S_h, E_h, I_h, R_h for humans and S_m, E_m, I_m for mosquitoes:

$$\frac{dE_h}{dt} = \beta_{mh} S_h I_m - (\gamma + \delta) E_h, \quad \frac{dI_h}{dt} = \delta E_h - \gamma I_h, \tag{4.3}$$

$$\frac{dE_m}{dt} = \beta_{hm} S_m I_h - (\mu + \sigma) E_m, \quad \frac{dI_m}{dt} = \sigma E_m - \mu I_m, \tag{4.4}$$

where δ is the progression rate from exposed to infectious humans. This model showed that latency in both hosts and vectors significantly lowers R_0.

Modern Extensions

Beyond these classical models, malaria epidemiology has been enriched by several extensions:

- **Relapse Models [2]:** Incorporate relapse in *P. vivax*, allowing recovered individuals to return to the infectious class:
$$\frac{dI_h}{dt} = \beta_{mh} S_h I_m - \gamma I_h + \rho R_h,$$
where ρ is the relapse rate.

- **Nonlinear Incidence Models [39]:** Replace linear incidence with saturation functions:
$$\frac{dI_h}{dt} = \frac{\beta_{mh} S_h I_m}{1 + \alpha I_m} - \gamma I_h,$$
where α captures transmission saturation in high-burden settings.

- **Fractional-Order Models [4]:** Introduced fractional derivatives to capture memory effects:
$${}^{CF}D_t^{\nu} I_h(t) = \beta_{mh} S_h I_m - \gamma I_h,$$
where ${}^{CF}D_t^{\nu}$ is the Caputo–Fabrizio fractional derivative of order ν.

- **Optimal Control Models [20]:** Incorporate intervention strategies explicitly:
$$\frac{dS_h}{dt} = -\beta_{mh}(1 - u_1) S_h I_m + u_2 R_h, \quad \frac{dI_h}{dt} = \beta_{mh}(1 - u_1) S_h I_m - (\gamma + u_3) I_h,$$
where u_1, u_2, u_3 represent controls for mosquito biting reduction, immunity boosting, and treatment.

Comparative Insights

The Ross, Macdonald, and Anderson–May models highlight the foundational role of mosquito survival and latency in malaria transmission. Modern extensions add relapse, nonlinear incidence, memory effects, and explicit control strategies, making models more realistic and policy-relevant. Together, these models form a hierarchy that progresses from simple theoretical constructs to advanced frameworks capable of guiding malaria elimination strategies.

Model	Ross (RR)
Compartments	$S_h, I_h; S_m, I_m$
Key Features	Linear incidence; no latency
Insights	Mosquito survival critical; R_0 depends on biting and mortality.
Model	Macdonald (MC)
Compartments	$S_h, I_h; S_m, E_m, I_m$
Key Features	Adds mosquito latency
Insights	More realistic vector dynamics; R_0 decreases with incubation.
Model	Anderson–May (AM)
Compartments	$S_h, E_h, I_h, R_h; S_m, E_m, I_m$
Key Features	Adds human latency
Insights	Latency in both host and vector reduces R_0; lower prevalence.
Model	Relapse Models
Compartments	S_h, E_h, I_h, R_h with relapse term
Key Features	Recovered individuals can relapse
Insights	Captures *P. vivax* relapse; sustains transmission.
Model	Nonlinear Incidence
Compartments	Same as AM but nonlinear force of infection
Key Features	Saturation effects in transmission
Insights	More realistic for high-burden settings.
Model	Fractional-Order
Compartments	Classical compartments with fractional derivatives
Key Features	Memory effects in transmission
Insights	Captures history dependence; better fit to real data.
Model	Optimal Control
Compartments	Compartments and control variables u_1, u_2, u_3
Key Features	Intervention strategies modeled explicitly
Insights	Evaluates nets, vaccines, treatment effectiveness.
Model	Gizachew model
Compartments	$S_e E_e I_e S_a E_a I_a R_a S_v E_v I_v$
Key Features	Separates non-immune and semi-immune humans; full mosquito staging
Insights	Captures heterogeneous immunity; highly detailed vector–host dynamics.

Table 1: Hierarchical comparison of malaria models from classical to modern formulations

Summary Comparison Table

This comparative analysis demonstrates the hierarchical evolution of malaria models. Starting from Ross's foundational framework, successive refinements by Macdonald and Anderson–May introduced latency effects, while modern extensions incorporated relapse, nonlinear incidence, fractional calculus, and optimal control. This hierarchy illustrates how mathematical models have advanced from simple abstractions to sophisticated tools for understanding malaria epidemiology and guiding control strategies.

Key Findings of the comparission

1. Historical Development: The foundational work of Ronald Ross established the importance of mosquito density in malaria transmission, leading to the prioritization of vector control strategies. Subsequent models, particularly those developed by Macdonald and Anderson-May, introduced essential concepts such as the basic reproduction number (R_0) and the incorporation of latency periods in both human and mosquito populations. These early models laid the groundwork for more sophisticated approaches that account for the complexities of malaria transmission.
2. Integration of Complex Factors: Modern malaria models increasingly integrate various factors that influence transmission dynamics, particularly focusing on immunity:
3. Immunity and Age Structure: The development of immunity through repeated exposure to malaria is crucial for understanding disease dynamics. Models that incorporate age structure and immunity provide a more accurate representation of malaria transmission, particularly in endemic regions. The distinction between semi-immune and non-immune individuals is vital, as semi-immune individuals often experience milder symptoms and lower mortality rates, which can significantly alter transmission dynamics.
4. Semi-Immunity: Semi-immune individuals, who have either gained or lost immunity at some point in their lives, represent a critical component of malaria transmission models. These individuals are less susceptible to severe disease but can still transmit the infection. Understanding the dynamics of semi-immunity is essential for predicting disease spread and evaluating the effectiveness of vaccination strategies.
5. Non-Immunity: Non-immune individuals, particularly children and those in regions with limited exposure, are at a higher risk of severe malaria. Models that account for the proportion of non-immune individuals in a population can better predict outbreaks and inform targeted interventions. Environmental Influences: Climatic variables such as temperature, rainfall, and humidity significantly impact mosquito life cycles and, consequently, malaria transmission. Recent models explicitly link these environmental factors to mosquito density and disease dynamics, emphasizing the need for adaptive strategies in response to climate change.
6. Socioeconomic Conditions: The socioeconomic status of populations plays a vital role in malaria transmission. Regions with higher poverty rates often experience greater malaria burden, highlighting the need for models that incorporate economic stratification and healthcare access. The interplay between socioeconomic factors and immunity can influence the overall disease dynamics within a community.

7. Human Mobility: The review emphasizes the importance of human mobility in the spread of malaria. Migration and visitation patterns can facilitate the reintroduction of malaria into previously controlled areas, underscoring the need for surveillance systems that account for human movement.

8. Drug Resistance: The emergence of drug-resistant strains of Plasmodium poses a significant challenge to malaria control efforts. Models that incorporate the dynamics of drug resistance are essential for developing effective treatment strategies and understanding the long-term implications of resistance on malaria transmission.

5. Limitations and Gaps in Malaria Research

Despite significant progress in the mathematical modeling of malaria transmission, several limitations remain across existing frameworks. Classical models such as Ross and Macdonald provided foundational insights but oversimplified host immunity and neglected relapse dynamics. Anderson–May introduced latency, yet did not fully capture heterogeneous immunity or long-term waning effects. Even modern extensions, including fractional-order and optimal control models, face challenges related to parameter estimation, computational complexity, and empirical validation.

Limitations of Existing Models

- Oversimplification of immunity: Many models assume homogeneous populations, overlooking differences between non-immune, semi-immune, and immune individuals.
- Neglect of relapse and waning immunity: Relapse in *P. vivax* and gradual loss of immunity are often underrepresented.
- Limited integration of socioeconomic and behavioral factors: Human mobility, healthcare access, and socioeconomic influences are rarely incorporated.
- Challenges in parameter estimation: Complex models, especially fractional-order systems, require parameters that are difficult to measure in field settings.

Gaps in Current Strategies

- Vector control limitations: Resistance to insecticides reduces long-term effectiveness.
- Case management gaps: Current treatment strategies inadequately address relapse and drug resistance.
- Vaccination challenges: Existing vaccines provide partial protection and do not fully account for heterogeneous immunity.
- Climate change impacts: Shifts in vector ecology due to climate variability are insufficiently modeled.

Novel Approaches Proposed

- Integration of within-host and between-host processes: Linking immune response dynamics with transmission models to capture realistic host heterogeneity.
- Stochastic modeling: Incorporating randomness to reflect environmental variability and human behavior.
- Climate-adaptive modeling: Developing frameworks that anticipate long-term changes in vector populations and transmission patterns.
- Data-driven approaches:Leveraging surveillance and empirical datasets to validate and refine models for policy relevance.

By extending the comparative analysis and hierarchical development of malaria models to include these limitations and gaps, the review provides a more comprehensive perspective. This ensures that mathematical theory not only reflects past advancements but also guides novel, evidence-based strategies for malaria prevention and control.

6. Conclusion

Mathematical modeling remains a powerful tool for understanding malaria transmission and informing public health policy for disease control and eradication. This review highlights the hierarchical evolution of malaria models, from Ross's foundational two-compartment system to the modern ten-compartment framework of Gellow–Munganga–Jafari. The major insights are clear:

- Mosquito survival and latency are critical determinants of transmission, as emphasized by Ross and Macdonald.
- Human latency and immunity heterogeneity significantly reduce R_0, as shown in Anderson–May and GMJ models.
- Relapse dynamics, nonlinear incidence, and fractional-order formulations capture realistic complexities such as *P. vivax* relapse, saturation effects, and memory dependence.
- Optimal control models bridge theory and practice by explicitly incorporating interventions.

Building on these insights, several actionable public health strategies emerge:

Vector Control (Mosquito Survival Reduction)

Mechanistic basis: Reduces mosquito lifespan (μ), directly lowering R_0 in Ross's model.
Comparison: Aligns with Ross's emphasis on mosquito mortality and Macdonald's latency; modern approaches include insecticide-treated nets and indoor residual spraying.

Case Management and Relapse Prevention

Mechanistic basis: Shortens infectious period (γ) and reduces relapse rate (ρ), limiting sustained transmission in relapse models.
Comparison: Extends beyond classical models by addressing *P. vivax* relapse, complementing WHO's current focus on radical cure with primaquine or tafenoquine.

Vaccination and Immunity Boosting

Mechanistic basis: Expands recovered/semi-immune compartments (R_h, R_a), reducing susceptibility and progression rates.
Comparison: Builds on Anderson–May's inclusion of human latency and GMJ's separation of immune groups; complements ongoing RTS,S/AS01 malaria vaccine programs.

Behavioral Interventions (Bed Nets, Repellents)

Mechanistic basis: Reduce biting rate (β_{mh}, β_{hm}), lowering force of infection.
Comparison: Explicitly modeled in optimal control frameworks (u_1); widely implemented in endemic regions with proven effectiveness.

Integrated Control Strategies

Mechanistic basis: Combine vector control, treatment, and vaccination, modeled through multiple control variables (u_1, u_2, u_3).
Comparison: Reflects modern optimal control models; consistent with WHO's integrated vector management and elimination strategies.

Final Insight: From Ross's early abstraction to GMJ's detailed ten-compartment realism, malaria models have progressively captured biological complexity and intervention dynamics. Translating these insights into public health strategies—vector control, relapse management, vaccination, behavioral interventions, and integrated approaches—ensures that mathematical theory directly informs practical elimination efforts. Ultimately, these models contribute to global efforts to reduce the burden of malaria, and their ongoing evolution will be essential in adapting to the changing landscape of malaria transmission and control.

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