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MATHEMATICAL MODEL OF MALARIA TRANSMISSION DYNAMICS: EVALUATING THE IMPACT OF ASYMPTOMATIC AND RESISTANT STRAINS IN HUMAN HOSTS

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Abstract. The paper presents a mathematical model of malaria transmission dynamics that incorporates the asymptomatic stage and resistant strains in infectious humans. We identified the disease-free (free malaria) and endemic (persistence of malaria) equilibriums of the model. Using the basic reproduction number $\mathcal{R}_0$, we constructed a suitable Lyapunov function to demonstrate the global asymptotic stability (GAS) of the disease-free equilibrium point. When $\mathcal{R}_0 \leq 1$, the disease-free equilibrium is global asymptotically stable. For $\mathcal{R}_0 > 1$, we analyzed the global asymptotic stability (GAS) of the endemic equilibrium. We conducted the local sensitivity analysis and numerical simulations for different scenarios. Our findings highlight the significant role of asymptomatic humans and resistant strains in the transmission dynamics of malaria. Therefore, the well-known strategies against malaria should be revised.

Keywords: Asymptomatic individuals, Malaria, Relapse, Ignorant infected humans, Resistant strains individuals.
AMS Subject Classification: 97M10.

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1 Introduction

Malaria, one of the mosquito-borne diseases, remains a major public health problem worldwide, particularly in African countries (CDC, 2022; WHO, 2023). Malaria is treatable and it can cause death of humans, particularly children aged under 5 years and pregnant women. It originates from the *Plasmodium* parasite, a protozoan that spreads in humans after being bitten by infected adult female *Anopheles* mosquitoes. The five species of *Plasmodium* parasites that can infect humans are *Plasmodium* falciparum (responsible of malaria severe cases), *Plasmodium* malariae, *Plasmodium* vivax, *Plasmodium* ovale, and *Plasmodium* knowlesi (Shi et al., 2024). In Africa, the African Leaders Malaria Alliance (ALMA) reveals that: the number of threats is still growing to achieving the goal of eliminating malaria in Africa by 2030 (ALMA, 2023). The same source reveals that across the continent, 1.27 billion individuals are at risk of malaria infection. Amongst this population, there were 186 cases per 1,000 persons and 47 deaths per 100,000 persons.

The use of mathematics approach for describing the complex mechanism of malaria transmission dynamics constitutes a helpful tool for better understanding and analyzing the spread of the disease in order to advice policymakers. Based on this fact, several rich models (Basir et

al., 2025; Djidjou-Demasse et al., 2020; Fatmawati et al., 2021; Gellow et al., 2023; Jaleta et al., 2025; Kaboré et al., 2024; Keno et al., 2022; Mangongo et al., 2022; Olaniyi et al., 2023, 2022, 2020; Qu et al., 2025; Rajnarayanan et al., 2025; Sualey et al., 2024; Wako et al., 2025) have been developed by researchers from the basic SEI, SEIS and SEIRS schemes. For example, Jaleta et al. (2025) discussed a mathematical model for malaria transmission dynamics. In their paper, authors analyzed the optimal control of the effect of treatment-seeking behaviors on the spread of malaria. Qu et al. (2025) analyzed a vaccination mathematical model of malaria transmission with seasonality and immune feedback. Rajnarayanan et al. (2025) analyzed a model for malaria using data-driven approach. In their paper, authors presented a new framework for modeling malaria transmission dynamics by integrating temperature and altitude-dependent transmission functions into a compartmental SIR-SI model. Wako et al. (2025) developed a mathematical model to analyze malaria transmission dynamics. In their model, they account for complications like severe anemia and organ dysfunction, which impact disease outcomes and health-care systems. Many aspects were considered in these models, such that: the environmental, climate factor in the life cycle of mosquito, partial immunity, social-hierarchical structure of humans, the relapse of ignorant infected humans, the impact of *Wolbachia* bacteria in reducing the size of mosquito population in their formulations. All of them have guided interventions such as insecticide-treated nets (ITNs), indoor residual spraying (IRS), antimalarial medications (ACT) and health education and community engagement (Akowe et al., 2025).

All the above mentioned intervention strategies focus on optimizing vector control and the treatment of symptomatic cases (Andolina et al., 2021). However, in the context of malaria, some infectious individuals are asymptomatic while others are symptomatic (Bousema et al., 2014; Galatas et al., 2016; Lindhlade et al., 2013; Prusty et al., 2021). Andolina et al. (2021) stated that symptomatic malaria cases represent only a small proportion of all *Plasmodium* infections. In addition, Tadesse et al. (2018) confirm that in the low-endemic setting aiming for malaria elimination, asymptomatic infections were highly prevalent and responsible for the majority of onward mosquito infections (Andolina et al., 2021). Therefore, the early identification and treatment of asymptomatic infections might accelerate elimination efforts. The asymptomatic cases of malaria are often dues to submicroscopic, which are often below the threshold of detection by microscopy or conventional malaria rapid test (MRT) (Andolina et al., 2021; Tadesse et al., 2018). Banegas et al. (2024) discovered the existence of asymptomatic malaria reservoirs in Honduras, which contribute to disease transmission and poses a challenge for elimination efforts. Furthermore, the biology of *Plasmodium* reveals that, *Gametocytes* typically progress through five distinct developmental stages. During the first three stages, these sexual forms are sequestered in tissues, making them potentially vulnerable to drugs targeting the asexual stages of the parasite. By stage 4, they re-enter the bloodstream, and at stage 5, mature *Gametocytes* circulate freely and are resistant to most treatments, except for the 8-aminoquinolines (Barnes & White, 2005; Pongtavornpinyo et al., 2008). To support this, the most expansive agent caused malaria, is characterized by the accrual of a reservoir of dormant parasites known as *Hypnozoites* (Mehra et al., 2014). They can remain in a dormant state for many days (one month for example) before reactivating to cause a relapse of malaria.

Considering these facts, some authors designed rich mathematical models to advice policy-makers for the control of malaria. Aguilar & Gutierrez (2020) studied a mathematical model for malaria transmission dynamics by accounting for asymptomatic carriers. Authors stated that, the correct understanding of the influence of asymptomatic individuals on transmission dynamics will provide a comprehensive description of the complex interplay between transmission agents. Beretta et al. (2018) extended the model of Aguilar & Gutierrez (2020) by structured the human population into two age groups. Recently, Shi et al. (2024) analyzed a reaction-diffusion malaria model accounting for asymptomatic carriers. In their paper, authors introduced a time periodic reaction-diffusion model for malaria spread, incorporating spatial heterogeneity, incubation periods, symptomatic and asymptomatic carriers. To incorporate resistance to anti-malarials,

Hamilton et al. (2023) introduced a mathematical model in 2023 to assess the effectiveness of vaccination and anti-malarial resistance across 42 African countries. Maithya et al. (2025) studied a mathematical malaria model by focusing on the effects of partial immunity, strong immunity, drug resistance and intensive treatment.

Due to the significance of asymptomatic carriers and the development of resistant strains in the transmission dynamics of malaria, in this paper we aim to develop a mathematical model to evaluate the impact of asymptomatic infectious and those who develop resistant strains in the dynamics transmission of malaria. We consider, in this paper, two types of individuals developing resistant strains. The first are those who, after treatment, continue to carry *Plasmodium* in their blood in a dormant form, without any symptoms and without being infectious; we call them "ignorant infected". The second are those who, after treatment, still have *Plasmodium* in their blood along with clinical signs. They are therefore infectious, and we refer to them as "infected with resistant strains".

The rest of the paper is organized as follows: In the next section, we state assumptions and formulate the mathematical model. In section 3, analytical analysis is done, starting by the well posedness, equilibrium of the proposed model and computing the basic reproduction number and analyzing the local and global stability of equilibrium points. In section 4, sensitivity analysis and numerical simulations are done to support our analytical analysis. We end the paper by some concluding remarks and discussions in section 5.

2 Formulation of the model

We consider two populations, the human and mosquito populations. The total human population at time t, $N_h(t)$ is divided into seven mutually exclusive compartments, the susceptible, exposed, asymptomatic, symptomatic, recovered, ignorant infected and infected with resistant strains compartments, denoted by $S_h, E_h, A_h, I_h, R_h, M_h$ and T_h respectively. We assume that only individuals in the M_h compartment can relapse at the rate ψ_2, as they carry the *Plasmodium* parasite without having been treated. At any time t, the total humans population follows this relation:

$$N_h(t) = S_h(t) + E_h(t) + A_h(t) + I_h(t) + R_h(t) + M_h(t) + T_h(t). \tag{1}$$

The total mosquito population at any time $t, N_m(t)$, is divided into three mutually exclusive compartments, following the common SEI scheme. We have the susceptible, exposed and infectious compartments, denoted by S_m, E_m and I_m respectively. At any time t, the total mosquito population is governed by:

$$N_m(t) = S_m(t) + E_m(t) + I_m(t). \tag{2}$$

Figure 1 gives the flow diagram of the transmission mechanism of the proposed model. The susceptible human can contract malaria through a bite of an infectious mosquito. Considering the average number of a mosquito bites, n and the probability that a bite by an infectious mosquito to a susceptible human lead to an infection of this susceptible human, c_{mh}, therefore, the force of infection of mosquito to human is given by:

$$\lambda_h = c_{mh} n \frac{I_m}{N_h}. \tag{3}$$

Additionally, susceptible mosquito can become infected when it bites an asymptomatic, symptomatic or resistant strains infected humans. Considering the average number of a mosquito bites n, and the probability that a bite by a susceptible mosquito to a symptomatic, asymptomatic or resistant strains infected lead to an infection of this susceptible mosquito, c_{hm}, therefore, we define the force of infection of human to mosquito by:

$$\lambda_m = c_{hm} n \frac{(A_h + I_h + T_h)}{N_h}. \tag{4}$$

The progression rates from asymptomatic humans to recovered, ignorant infected, and resistant strains infected are $\gamma_1\xi_1, \gamma_1\xi_2$ and $\gamma_1\xi_3$ respectively. Here, γ_1 is the recovery rate of asymptomatic humans, while ξ_1, ξ_2 and ξ_3 denote the proportions of asymptomatic humans who recover, are ignorant infected, and carry resistant strains, respectively. Parameters ξ_1, ξ_2 and ξ_3 are such that $\xi_1 + \xi_2 + \xi_3 = 1$. One can take $\xi_2 = 1 - \xi_1 - k$, where $\xi_3 = k$ and $0 \leq k \leq 1 - \xi_1$ for $\xi_1 \in [0, 1[$.

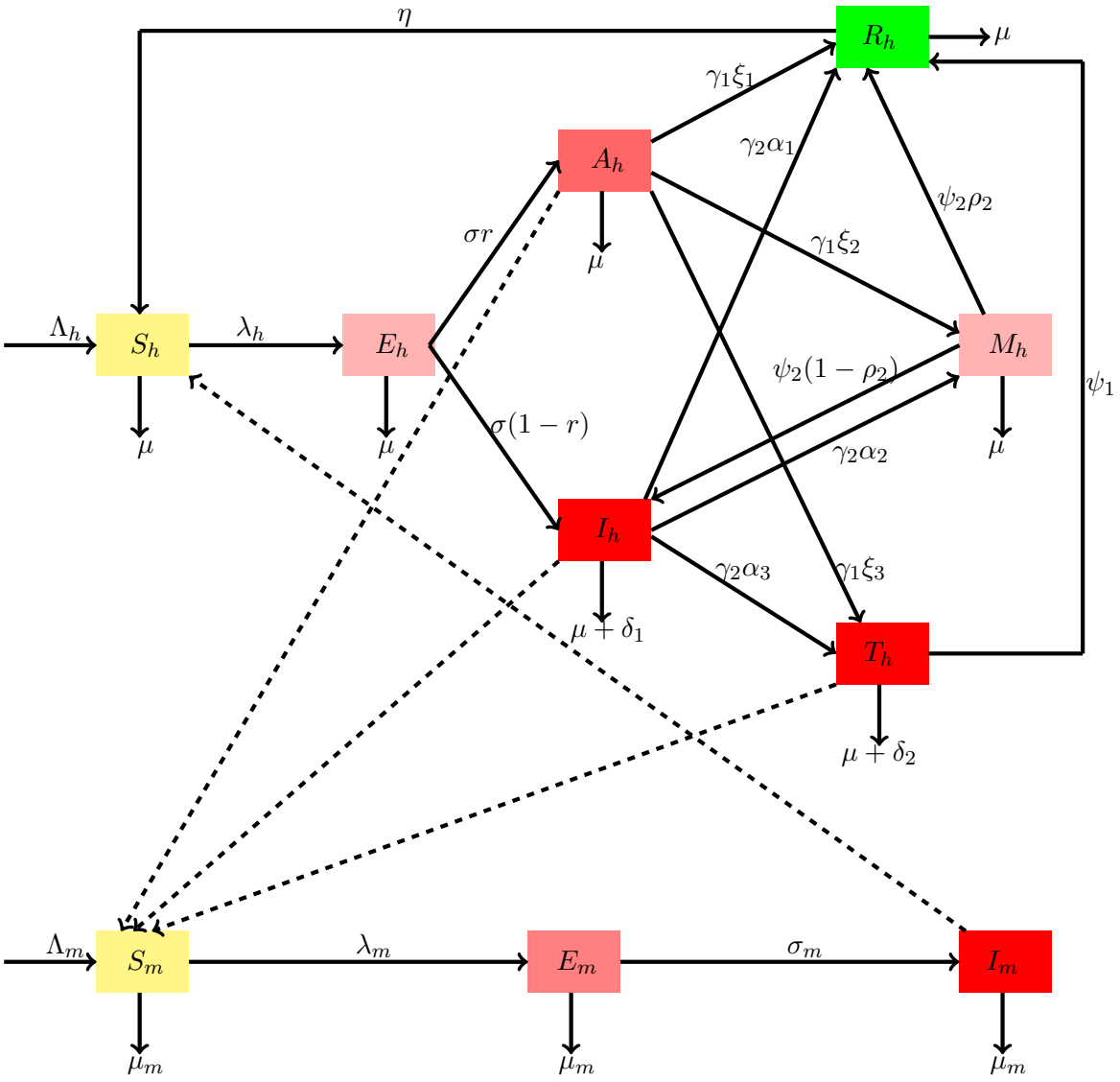


Figure 1: Flow diagram of the proposed model

The progression rates from symptomatic humans to recovered, ignorant infected, and resistant strains infected are $\gamma_2\alpha_1, \gamma_2\alpha_2$ and $\gamma_2\alpha_3$, respectively. Here, γ_2 is the recovery rate of symptomatic humans, while α_1, α_2 and α_3 denote the proportions of symptomatic humans who recover, are ignorant infected, and carry resistant strains, respectively. Parameters α_1, α_2 and α_3 are such that $\alpha_1+\alpha_2+\alpha_3 = 1$. One can take $\alpha_2 = 1-\alpha_1-p$, where $\alpha_3 = p$ and $0 \leq p \leq 1-\alpha_1$ for $\alpha_1 \in [0, 1[$.

The progression rate from ignorant infected humans is $\psi_2(1 - \rho_2)$, where ψ_2 is the relapse rate and $1 - \rho_2$ is the proportion of ignorant infected humans who relapse. Consequently, ρ_2 represents the proportion of ignorant infected humans who recover.

In this paper, we made the following assumptions:

- Only individuals in the M_h compartment can relapse, and the relapse occurs exclusively

in the symptomatic compartment, I_h.

- Individuals in the T_h compartment can die due to parasite resistance in the blood at rate δ_2, or recover if the immune system enhances at rate ψ_1.
- Recovered individuals lose their acquired immunity at rate η.
- Humans can die naturally at rate μ, which is independent of the recruitment rate Λ_h.
- Mosquitoes can die naturally at rate μ_m, independent of their recruitment rate Λ_m.

All parameters of the proposed model are summarized in Table 1.

Table 1: Descriptions of parameters of the Model (5)-(14)

Parameters	**Descriptions**
Λ_h	recruitment rate of humans
c_{hm}	probability that a bite by a susceptible mosquito on a symptomatic, asymptomatic or resistant strains humans leads to infection of the mosquito
c_{mh}	probability that a bite by an infectious mosquito on a susceptible human leads to infection of the human
σ	latent rate of humans
r	proportion of asymptomatic humans
$1-r$	proportion of symptomatic humans
μ	natural death rate of humans
δ_1	malaria-induced death rate of symptomatic humans
δ_2	malaria-induced death rate of resistant strains humans
γ_1	recovery rate of asymptomatic humans
γ_2	recovery rate of symptomatic humans
ξ_1	proportion of asymptomatic humans who recover
ξ_2	proportion of ignorant infected among asymptomatic humans
ξ_3	proportion of resistant strains infected among asymptomatic humans
α_1	proportion of symptomatic humans who recover
α_2	proportion of ignorant infected among symptomatic humans
α_3	proportion of resistant strains infected among symptomatic humans
η	rate of loss of acquired immunity
ψ_1	recovery rate of resistant strains individuals
ψ_2	relapse rate
ρ_2	proportion of ignorant infected who recover
$(1-\rho_2)$	proportion of ignorant infected who relapse
Λ_m	recruitment rate of mosquitoes
μ_m	natural death rate of mosquitoes
n	the average number of mosquito bites
σ_m	latent rate of mosquitoes

Susceptible humans, $S(t)$ increase at the constant recruitment rate Λ_h and the rate of loss of acquired immunity from recovered humans η, and decrease at the rates λ_h and μ. So, the evolution in time of the susceptible humans can be modeled by the following differential equation:

$$\frac{dS_h(t)}{dt} = \Lambda_h - (\lambda_h + \mu)S_h + \mu R_h.$$

Exposed humans, $E(t)$, increase through the incidence $\lambda_h S_h$ and decrease at the latent rate, σ and the natural death rate μ of humans. Therefore, the evolution over time of the exposed humans follows this differential equation:

$$\frac{dE_h(t)}{dt} = \lambda_h S_h - (\sigma + \mu)E_h.$$

Asymptomatic humans, $A(t)$, increase through the progression rate $r\sigma$ and decrease at the recovered rate, γ_1, of asymptomatic humans and natural death rate μ of humans. Therefore,

the evolution over time of the asymptomatic humans is governed by this differential equation:

$$\frac{dA_h(t)}{dt} = r\sigma E_h - (\gamma_1 + \mu)A_h.$$

In the same manner, we can establish the evolution equations for the other human compartments in the proposed model. The same principle is applied to the mosquito compartments. These lead to the following system of ODEs, System (5)-(14).

$$\begin{aligned}
\frac{dS_h}{dt} &= \Lambda_h - (\lambda_h + \mu)S_h + \eta R_h && (5)\\
\frac{dE_h}{dt} &= \lambda_h S_h - (\sigma + \mu)E_h && (6)\\
\frac{dA_h}{dt} &= r\sigma E_h - (\gamma_1 + \mu)A_h && (7)\\
\frac{dI_h}{dt} &= (1-r)\sigma E_h + \psi_2(1-\rho_2)M_h - (\gamma_2 + \delta_1 + \mu)I_h && (8)\\
\frac{dR_h}{dt} &= \gamma_1\xi_1 A_h + \gamma_2\alpha_1 I_h + \psi_1 T_h + \psi_2\rho_2 M_h - (\eta + \mu)R_h && (9)\\
\frac{dM_h}{dt} &= \gamma_1\xi_2 A_h + \gamma_2\alpha_2 I_h - (\psi_2 + \mu)M_h && (10)\\
\frac{dT_h}{dt} &= \gamma_1\xi_3 A_h + \gamma_2\alpha_3 I_h - (\psi_1 + \mu + \delta_2)T_h && (11)\\
\frac{dS_m}{dt} &= \Lambda_m - (\lambda_m + \mu_m)S_m && (12)\\
\frac{dE_m}{dt} &= \lambda_m S_m - (\sigma_m + \mu_m)E_m && (13)\\
\frac{dI_m}{dt} &= \sigma_m E_m - \mu_m I_m && (14)
\end{aligned}$$

System (5)-(14) is appended with the following non-negative initial conditions below:

$$\begin{aligned}
&(S_h(0), E_h(0), A_h(0), I_h(0), R_h(0), M_h(0), T_h(0), S_m(0), E_m(0), I_m(0))\\
=\ &(S_{h0}, E_{h0}, A_{h0}, I_{h0}, R_{h0}, M_{h0}, T_{h0}, S_{m0}, E_{m0}, I_{m0}) \geq 0. \qquad (15)
\end{aligned}$$

3 Mathematical analysis

In this section, we conduct a qualitative analysis of the proposed model. We begin by examining the well-posedness of the model, searching for the disease free-equilibrium (DFE) point, and calculating the basic reproduction number. Subsequently, we establish the global asymptotic stability (GAS) of the DFE. In addition, we prove the local asymptotic stability (LAS) of the endemic equilibrium (EE) point of the model.

3.1 Well-posedness of the model

Let $X(t) = (S_h, E_h, A_h, I_h, R_h, M_h, T_h, S_m, E_m, I_m)$ a vector solution of system (5)-(14) and $f : \Omega \subset \mathbb{R}^{10}_+ \longrightarrow \mathbb{R}^{10}_+$, where the feasible set Ω is defined by:

$$\Omega = \left\{ (S_h, E_h, A_h, I_h, R_h, M_h, T_h, S_m, E_m, I_m) \in \mathbb{R}^{10}_+ : 0 \leq N_h(t) \leq \frac{\Lambda_h}{\mu} \; and \; 0 \leq N_m(t) = \frac{\Lambda_m}{\mu_m} \right\},$$

is a compact set of $\mathbb{R}^{10}_+$ and $f(X(t)) = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9, f_{10})$, where:

$$\begin{cases} f_1(X(t)) &= \Lambda_h - (\lambda_h + \mu)S_h + \eta R_h \\ f_2(X(t)) &= \lambda_h S_h - (\sigma + \mu)E_h \\ f_3(X(t)) &= r\sigma E_h - (\gamma_1 + \mu)A_h \\ f_4(X(t)) &= (1-r)\sigma E_h + \psi_2(1-\rho_2)M_h - (\gamma_2 + \delta_1 + \mu)I_h \\ f_5(X(t)) &= \gamma_1\xi_1 A_h + \gamma_2\alpha_1 I_h + \psi_1 T_h + \psi_2\rho_2 M_h - (\eta + \mu)R_h \\ f_6(X(t)) &= \gamma_1\xi_2 A_h + \gamma_2\alpha_2 I_h - (\psi_2 + \mu)M_h \\ f_7(X(t)) &= \gamma_1\xi_3 A_h + \gamma_2\alpha_3 I_h - (\psi_1 + \mu + \delta_2)T_h \\ f_8(X(t)) &= \Lambda_m - (\lambda_m + \mu_m)S_m \\ f_9(X(t)) &= \lambda_m S_m - (\sigma_m + \mu_m)E_m \\ f_{10}(X(t)) &= \sigma_m E_m - \mu_m I_m \end{cases}. \tag{16}$$

Theorem 1 (Well-posedness of the model)**.** *Given the non negative initial conditions in (15), the system (5)-(14) is a dynamical system on the biological feasible region Ω. Furthermore, the invariant compact set Ω is attracting in $\mathbb{R}^{10}_+$ with the given initial conditions.*

Proof. The proof consists of two steps: First, we show that, for non-negative initial conditions, system (5)-(14) admits a unique non-negative solution for all $t \geq 0$, which lies within the feasible set Ω. We also demonstrate that Ω is a positively invariant set for the system. Second, we prove that any solution of system (5)-(14) remains within Ω.

For the first step, functions f_i in (16) are $C^\infty-$functions, which implies C^1-functions. Hence, function f is differentiable. Consequently, from the standard theorem of the dynamical system (Andrew & R, 1998; Wiggins & Golubitsky, 1990), f is locally Lipschitz continuous in some open ball containing $X(0)$. Therefore, it follows by Cauchy-Lipschitz theorem that the system (5)-(14) has a unique solution, which exists locally. In addition, suppose that $X(t)$ is a solution of system (5)-(14) for $X(0) \geq 0$, and let t_0 be the smallest positive t such that $S_h(t_0) = 0$ or $E_h(t_0) = 0$ or $A_h(t_0) = 0$ or $I_h(t_0) = 0$ or $R_h(t_0) = 0$ or $M_h(t_0) = 0$ or $T_h(t_0) = 0$ or $S_m(t_0) = 0$ or $E_m(t_0) = 0$ or $I_m(t_0) = 0$. By continuity of functions $S_h, E_h, A_h, I_h, R_h, M_h, T_h, S_m, E_m$ and I_m, there exists $t^* > t_0$ such that if $S_h(t_0) = 0$, then from equation (5), we have $\frac{dS_h(t_0)}{dt} = \Lambda_h + \eta R_h(t_0) \geq 0$. Thus for all $t \in [t_0, t^*], S_h(t) \geq 0$. Consequently, S_h is non negative for all t. In the same manner, we can establish the non negativity of $E_h, A_h, I_h, R_h, M_h, T_h, S_m, E_m$ and I_m for all $t \geq 0$. Hence, the solution $X(t)$ of the model are non negatives for all $t \geq 0$. Therefore, the feasible set Ω is positively invariant, consequently for all $t \geq 0$ the solution remains positive.

By adding equations (5)-(11), we obtain:

$$\begin{aligned} \frac{dN_h(t)}{dt} &= \Lambda_h - \mu N_h - \delta_1 I_h - \delta_2 T_h \\ &\leq \Lambda_h - \mu N_h \end{aligned} \tag{17}$$

Applying Gronwall inequality to the relation (17), we obtain:

$$N_h(t) \leq \frac{\Lambda_h}{\mu} + \left(N_{h0} - \frac{\Lambda_h}{\mu}\right)\exp(-\mu t).$$

As $t \to +\infty$,

$$0 \leq N_h(t) \leq \frac{\Lambda_h}{\mu} \; for \; 0 \leq N_{h0} \leq \frac{\Lambda_h}{\mu}, \tag{18}$$

Likewise, adding equations (12)-(14), we get:

$$\frac{dN_m(t)}{dt} = \Lambda_m - \mu_m N_m.$$

Solving this first order differential equation, we obtain:

$$N_m(t) = \frac{\Lambda_m}{\mu_m} + \exp(-\mu_m t)\left(N_{m0} - \frac{\Lambda_m}{\mu_m}\right).$$

As $t \to +\infty$,

$$0 \leq N_m(t) = \frac{\Lambda_m}{\mu_m}. \tag{19}$$

This means that, for all $t \geq 0$, all solution of system (5)-(14) satisfies relations (18) and (19). Furthermore, when a solution of system (5)-(14) starts outside of the feasible set Ω, with $N_{h0} > \frac{\Lambda_h}{\mu}$ or $N_{m0} > \frac{\Lambda_m}{\mu_m}$, it follows from relations (18) and (19) that $\limsup_{t\to\infty} N_h(t) \leq \frac{\Lambda_h}{\mu}$ and $\limsup_{t\to\infty} N_m(t) \leq \frac{\Lambda_m}{\mu_m}$. Thus, the region Ω is attracting. Combining the above two steps and using theorem 2.1.5 in (Andrew & R, 1998), we conclude that the system (5)-(14) defines a dynamical system on Ω. Additionally, let us verify the dissipation condition to conclude about the global existence and the boundedness of the solution (Caraballo & Han, 2016).

$$\begin{aligned}
f(X).X &= (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9, f_{10}).(S_h, E_h, A_h, I_h, R_h, M_h, T_h, S_m, E_m, I_m) \\
&= [\Lambda_h - (\lambda_h + \mu)S_h + \eta R_h]S_h + [\lambda_h S_h - (\sigma + \mu)E_h]E_h + [r\sigma E_h - (\gamma_1 + \mu)A_h]A_h \\
&+ [(1-r)\sigma E_h + \psi_2(1-\rho_2)M_h - (\gamma_2 + \delta_1 + \mu)I_h]I_h \\
&+ [\gamma_1\xi_1 A_h + \gamma_2\alpha_1 I_h + \psi_1 T_h + \psi_2\rho_2 M_h - (\eta + \mu)R_h]R_h \\
&+ [\gamma_1\xi_2 A_h + \gamma_2\alpha_2 I_h - (\psi_2 + \mu)M_h]M_h + [\gamma_1\xi_3 A_h + \gamma_2\alpha_3 I_h - (\psi_1 + \mu + \delta_2)T_h]T_h \\
&+ [\Lambda_m - (\lambda_m + \mu_m)S_m]S_m + [\lambda_m S_m - (\sigma_m + \mu_m)E_m]E_m + [\sigma_m E_m - \mu_m I_m]I_m \\
&\leq (7\mu + 3\mu_m + \sigma + \gamma_1 + \gamma_2 + \delta_1 + \eta + \psi_2 + \psi_1 + \delta_2 + \sigma_m)||X||^2 \\
&+ (\Lambda_h + \eta + \sigma + \psi_2\gamma_1 + \gamma_2 + \psi_1)N_h^2 + (2c_{mh}nN_h^2 + \Lambda_m + 2c_{hm}n + \sigma_m)N_m^2 \\
&= a||X||^2 + b,
\end{aligned}$$

where $a = 7\mu + 3\mu_m + \sigma + \gamma_1 + \gamma_2 + \delta_1 + \eta + \psi_2 + \psi_1 + \delta_2 + \sigma_m$ and
$b = (\Lambda_h + \eta + \sigma + \psi_2\gamma_1 + \gamma_2 + \psi_1)N_h^2 + (2c_{mh}nN_h^2 + \Lambda_m + 2c_{hm}n + \sigma_m)N_m^2$. Hence, there exists a unique solution $X(t)$ of system (5)-(14) globally defined in time and since $S_h(t) \leq N_h(t), E_h(t) \leq N_h(t), A_h(t) \leq N_h(t), I_h(t) \leq N_h(t), R_h(t) \leq N_h(t), M_h(t) \leq N_h(t), T_h(t) \leq N_h(t), S_m(t) \leq N_m(t), E_m(t) \leq N_m(t), I_m(t) \leq N_m(t)$, for all $t \geq 0$, the solution $X(t)$ is bounded. □

3.2 Disease-free equilibrium and basic reproduction number

3.2.1 Disease-free equilibrium (DFE)

Theorem 2 (Equilibrium of the model)**.** *The system (5)-(14) admits at least one equilibrium point in the positively invariant compact set* Ω.

Proof. To determine the equilibrium of the model system (5)-(14), we set the right-hand side of system (5)-(14) equals to zero. We have then:

$$\begin{aligned}
0 &= \Lambda_h - (\lambda_h + \mu)S_h + \eta R_h && (20)\\
0 &= \lambda_h S_h - (\sigma + \mu)E_h && (21)\\
0 &= r\sigma E_h - (\gamma_1 + \mu)A_h && (22)\\
0 &= (1-r)\sigma E_h + \psi_2(1-\rho_2)M_h - (\gamma_2 + \delta_1 + \mu)I_h && (23)\\
0 &= \gamma_1\xi_1 A_h + \gamma_2\alpha_1 I_h + \psi_1 T_h + \psi_2\rho_2 M_h - (\eta + \mu)R_h && (24)\\
0 &= \gamma_1\xi_2 A_h + \gamma_2\alpha_2 I_h - (\psi_2 + \mu)M_h && (25)\\
0 &= \gamma_1\xi_3 A_h + \gamma_2\alpha_3 I_h - (\psi_1 + \mu + \delta_2)T_h && (26)\\
0 &= \Lambda_m - (\lambda_m + \mu_m)S_m && (27)\\
0 &= \lambda_m S_m - (\sigma_m + \mu_m)E_m && (28)\\
0 &= \sigma_m E_m - \mu_m I_m && (29)
\end{aligned}$$

From Equations (27), (28), (29), (26), (25), (24), (23), (22), (21) and (20) we obtain respectively:

$$S_m = \frac{\Lambda_m}{\lambda_m + \mu_m}, \tag{30}$$

$$E_m = \frac{\lambda_m \Lambda_m}{(\sigma_m + \mu_m)(\lambda_m + \mu_m)}, \tag{31}$$

$$I_m = \frac{\sigma_m \lambda_m \Lambda_m}{\mu_m (\sigma_m + \mu_m)(\lambda_m + \mu_m)}, \tag{32}$$

$$T_h = \frac{\gamma_1 \xi_3 A_h + \gamma_2 \alpha_3 I_h}{\psi_1 + \mu + \delta_2}, \tag{33}$$

$$M_h = \frac{\gamma_1 \xi_2 A_h + \gamma_2 \alpha_2 I_h}{\psi_2 + \mu}, \tag{34}$$

$$R_h = \frac{\gamma_1 \xi_1 A_h + \gamma_2 \alpha_1 I_h + \psi_2 \rho_2 M_h + \psi_1 T_h}{\eta + \mu}, \tag{35}$$

$$E_h = \frac{(\gamma_2 + \delta_1 + \mu) I_h - \psi_2 (1 - \rho_2) M_h}{(1 - r)\sigma}, \tag{36}$$

$$E_h = \frac{(\gamma_1 + \mu) A_h}{r\sigma} \text{ or } A_h = \frac{r\sigma E_h}{\gamma_1 + \mu}, \tag{37}$$

$$E_h = \frac{\lambda_h S_h}{\sigma + \mu}, \tag{38}$$

$$S_h = \frac{\Lambda_h + \eta R_h}{\lambda_h + \mu}. \tag{39}$$

Equating Relations (36) and (37), we obtain:

$$M_h = \frac{r\sigma(\gamma_2 + \delta_1 + \mu) I_h - (1 - r)\sigma(\gamma_1 + \mu) A_h}{r\sigma(1 - \rho_2)\psi_2} \tag{40}$$

Equating again Relations (34) and (40), we obtain:

$$I_h = \frac{r\sigma(1 - \rho_2)\psi_2 \gamma_1 \xi_2 + (1 - r)\sigma(\gamma_1 + \mu)(\psi_2 + \mu)}{r\sigma(\psi_2 + \mu)(\gamma_2 + \delta_1 + \mu) - r\sigma(1 - \rho_2)\psi_2 \gamma_2 \alpha_2} A_h \tag{41}$$

We notice that from Equation (41), if $A_h = 0$, then $I_h = 0$, substitute them into Equation (33), we obtain $T_h = 0$. Therefore, after some substitutions, we obtain: $E_h = M_h = R_h = 0, S_h = \frac{\Lambda_h}{\mu}$ and $S_m = \frac{\Lambda_m}{\mu_m}$. In a vector notation, we can write:

$$X^0 = \left(\frac{\Lambda_h}{\mu}, 0, 0, 0, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0 \right). \tag{42}$$

This equilibrium occurs in the absence of malaria in the population, called "disease-free equilibrium (DFE)". In the absence of malaria, the human and mosquito susceptible populations are proportional to the ratios $\frac{\Lambda_h}{\mu}$ and $\frac{\Lambda_m}{\mu_m}$ respectively. □

3.2.2 Basic reproduction number

The basic reproduction number, $\mathcal{R}_0$, (Driessche & Watmough, 2008) defined as the average number of secondary cases produced by one infectious individual during her/his entire period of infectiousness in a completely susceptible population. It is a very important threshold for the stability analysis of a given model. To compute $\mathcal{R}_0$, we use the next generation matrix as developed and explained in (Diekmann & Heesterbeek, 2000; Driessche & Watmough, 2002, 2008). The disease compartments of the model system (5)-(14) are $E_h, A_h, I_h, M_h, T_h, E_m$ and I_m. The system (5)-(14) can be reduced as $\dot{Y} = \mathcal{F} - \mathcal{V}$, where matrices $\mathcal{F}$ and $\mathcal{V}$ represent the rate of appearance of new infections and the transfer rate of individuals between the infective classes, respectively. We have then:

$$\mathcal{F}(Y) = \begin{pmatrix} \lambda_h S_h \\ 0 \\ 0 \\ 0 \\ 0 \\ \lambda_m S_m \\ 0 \end{pmatrix} \; and \; \mathcal{V}(Y) = \begin{pmatrix} (\sigma+\mu)E_h \\ -r\sigma E_h + (\gamma_1+\mu)A_h \\ -(1-r)\sigma E_h - \psi_2(1-\rho_2)M_h + (\gamma_2+\mu+\delta_1)I_h \\ -\gamma_1\xi_2 A_h - \gamma_2\alpha_2 I_h + (\psi_2+\mu)M_h \\ -\gamma_1\xi_3 A_h - \gamma_2\alpha_3 I_h + (\psi_1+\mu+\delta_2)T_h \\ (\sigma_m+\mu_m)E_m \\ -\sigma_m E_m + \mu_m I_m \end{pmatrix}$$

Setting that $\varpi_1 = \sigma+\mu, \varpi_2 = \gamma_1+\mu, \varpi_3 = \psi_2(1-\rho_2), \varpi_4 = \gamma_2+\mu+\delta_1, \varpi_5 = \psi_2+\mu, \varpi_6 = \psi_1+\mu+\delta_2$ and $\varpi_7 = \sigma_m+\mu_m$, the Jacobians of these matrices evaluated at the disease-free equilibrium, X^0 give respectively:

$$F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & c_{mh}n \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{c_{hm}n\mu\Lambda_m}{\mu_m\Lambda_h} & \frac{c_{hm}n\mu\Lambda_m}{\mu_m\Lambda_h} & 0 & \frac{c_{hm}n\mu\Lambda_m}{\mu_m\Lambda_h} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

and

$$V = \begin{pmatrix} \varpi_1 & 0 & 0 & 0 & 0 & 0 & 0 \\ -r\sigma & \varpi_2 & 0 & 0 & 0 & 0 & 0 \\ -(1-r)\sigma & 0 & \varpi_4 & -\varpi_3 & 0 & 0 & 0 \\ 0 & -\gamma_1\xi_2 & -\gamma_2\alpha_2 & \varpi_5 & 0 & 0 & 0 \\ 0 & -\gamma_1\xi_3 & -\gamma_2\alpha_3 & 0 & \varpi_6 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \varpi_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & -\sigma_m & \mu_m \end{pmatrix}.$$

And we have:

$$V^{-1} = \begin{pmatrix} \frac{1}{\varpi_1} & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{r\sigma}{\varpi_1\varpi_2} & \frac{1}{\varpi_2} & 0 & 0 & 0 & 0 & 0 \\ \frac{(1-r)\sigma}{\varpi_1\varpi_4} + \frac{k_1\varpi_3}{k_2\varpi_1\varpi_2\varpi_4} & \frac{\gamma_1\xi_2\varpi_3}{k_2\varpi_2} & \frac{1}{\varpi_4}\left(1+\frac{\alpha_2\gamma_2\varpi_3}{k_2}\right) & \frac{\varpi_3}{k_2} & 0 & 0 & 0 \\ \frac{k_1}{k_2\varpi_1\varpi_2} & \frac{\gamma_1\xi_2\varpi_4}{k_2\varpi_2} & \frac{\alpha_2\gamma_2}{k_2} & \frac{\varpi_4}{k_2} & 0 & 0 & 0 \\ k_7 & \frac{\gamma_1(\alpha_3\gamma_2\xi_2\varpi_3+\xi_3k_2)}{k_2\varpi_2\varpi_6} & \frac{\alpha_3\gamma_2(\alpha_2\gamma_2\varpi_3+k_2)}{k_2\varpi_4\varpi_6} & \frac{\alpha_3\gamma_2\varpi_3}{k_2\varpi_6} & \frac{1}{\varpi_6} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{1}{\varpi_7} & 0 \\ 0 & 0 & 0 & 0 & 0 & \frac{\sigma_m}{\mu_m\varpi_7} & \frac{1}{\mu_m} \end{pmatrix}$$

and

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \frac{\Lambda_m c_{hm}\mu n}{\Lambda_h \mu_m} k_3 & \frac{\Lambda_m c_{hm}\mu n}{\Lambda_h \mu_m \varpi_2} k_5 & \frac{\Lambda_m c_{hm}\mu n}{\Lambda_h \mu_m \varpi_4} k_6 & \frac{\Lambda_m c_{hm}\mu n \varpi_3}{k_2 \Lambda_h \mu_m}\left(1+\frac{\alpha_3\gamma_2}{\varpi_6}\right) & \frac{\Lambda_m c_{hm}\mu n}{\Lambda_h \mu_m \varpi_6} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

with $k_1 = \sigma(\gamma_2\alpha_2(1-r)\varpi_2 + \gamma_1 r\xi_2\varpi_4), k_2 = \varpi_4\varpi_5 - \alpha_2\gamma_2\varpi_3, k_3 = \frac{(1-r)\sigma}{\varpi_1\varpi_4} + \frac{k_1\varpi_3}{k_2\varpi_1\varpi_2\varpi_4}, k_4 = k_3\left(1+\frac{\alpha_3\gamma_2}{\varpi_6}\right) + \frac{r\sigma}{\varpi_1\varpi_2}\left(1+\frac{\gamma_1\xi_3}{\varpi_6}\right), k_5 = 1 + \frac{\gamma_1\varpi_3\xi_2}{k_2}\left(1+\frac{\alpha_3\gamma_2}{\varpi_6}\right) + \frac{\gamma_1\xi_3}{\varpi_6}, k_6 = \left(1+\frac{\gamma_2\alpha_3}{\varpi_6}\right)\left(1+\frac{\alpha_2\gamma_2\varpi_3}{k_2}\right)$ and $k_7 = \frac{\alpha_3\gamma_2}{\varpi_1\varpi_4\varpi_6}\left((1-r)\sigma + \frac{k_1\varpi_3}{k_2\varpi_2}\right) + \frac{\gamma_1 r\sigma\xi_3}{\varpi_1\varpi_2\varpi_6}$.

The basic reproduction number, $\mathcal{R}_0$ is defined by:

$$\mathcal{R}_0 = \rho(F.V^{-1}), \tag{43}$$

where $\rho(.)$ is the spectral radius of the next generation matrix FV^{-1}. After some computations, we obtain:

$$\begin{aligned} \mathcal{R}_0 &= \rho(F.V^{-1}) \\ &= n\sqrt{\frac{c_{mh}c_{hm}\sigma\sigma_m\mu\Lambda_m}{\mu_m^2\varpi_1\varpi_2\varpi_6\varpi_7 k_2\Lambda_h}(\mathcal{R}_{01}+\mathcal{R}_{02})}, \end{aligned} \tag{44}$$

where $\mathcal{R}_{01} = r\left[k_2(\varpi_6+\gamma_1\xi_3) + \gamma_1\xi_2\varpi_3(\gamma_2\alpha_3+\varpi_6)\right]$ and $\mathcal{R}_{02} = (1-r)\varpi_2\varpi_5(\varpi_6+\gamma_2\alpha_3)$.

Remark. The basic reproduction number, $\mathcal{R}_0$, representing the average number of secondary malaria cases produced by a single infectious individual during his/her infectious period, can be decomposed into two terms: $\mathcal{R}_{01}$, the contribution from asymptomatic humans, and $\mathcal{R}_{02}$, the contribution from symptomatic humans. Furthermore, all these contributions are weighted by the mosquito biting term, n, which represents the average number of mosquito bites per human. When the basic reproduction number $\mathcal{R}_0$ is less than one, malaria dies out. When it is greater than one, malaria continues to spread in the population. The fractions $\frac{\Lambda_m}{\Lambda_h}$ and $1/\mu_m$ represent the relative population dynamics of mosquitoes versus humans in the model and the survival period of mosquitoes respectively. The ratio μ/μ_m represents the relative measure of mortality between human and mosquito populations. When it is high, it indicates that human mortality is much greater than mosquito mortality and vice versa. This ratio is very important, as it helps in understanding population dynamics and the impact of interventions, such as mosquito control.

3.3 Global Asymptotic Stability of the DFE

Theorem 3 (GAS of the DFE)**.** *The disease free-equilibrium X^0 of model (5)-(14) is globally asymptotically stable (GAS) in the positively invariant and compact set Ω whenever $\mathcal{R}_0 \leq 1$. If $\mathcal{R}_0 > 1$, the disease-free equilibrium is unstable, the system is uniformly persistent, and there exists at least one equilibrium in int(Ω).*

Proof. To prove the GAS, we need to construct a Lyapunov function L, depending on $\mathcal{R}_0$, to determine whether its derivative is less than one. For this construction, we use the matrix-theoretical method explained in (Shuai & Driessche, 2013). Let $x = (E_h, A_h, I_h, M_h, T_h, E_m, I_m)^T$

and $y = (S_h, R_h, S_m)^T$. Using matrices $F, V, \mathcal{F}$ and $\mathcal{V}$, the computations of $f(x,y) = (F-V)x - \mathcal{F}(x,y) + \mathcal{V}(x,y)$ give:

$$\begin{aligned} f(x,y) &= (F-V)x - \mathcal{F}(x,y) + \mathcal{V}(x,y) \\ &= \begin{pmatrix} c_{mh} n I_m \left(1 - \frac{S_h}{N_h}\right) \\ 0 \\ 0 \\ 0 \\ 0 \\ \frac{c_{hm} n \mu \Lambda_m}{\mu_m \Lambda_h} \frac{(A_h + I_h + T_h)}{N_h} \left(N_h - \frac{\mu_m \Lambda_h}{\mu \Lambda_m} S_m\right) \\ 0 \end{pmatrix} \geq \begin{pmatrix} c_{mh} n I_m \left(1 - \frac{S_h}{N_h}\right) \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix} \end{aligned}$$

and

$$V^{-1}F = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & 0 & \frac{c_{mh} n}{\varpi_1} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{c_{mh} n r \sigma}{\varpi_1 \varpi_2} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{c_{mh} n}{\varpi_1 \varpi_4} \left((1-r)\sigma + \frac{k_1 \varpi_3}{k_2 \varpi_2}\right) \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{k_1 c_{mh} n}{k_2 \varpi_1 \varpi_2} \\ 0 & 0 & 0 & 0 & 0 & 0 & \frac{c_{mh} n}{\varpi_6} \left(\alpha_3 \gamma_2 k_3 + \frac{\gamma_1 r \sigma \xi_3}{\varpi_1 \varpi_2}\right) \\ 0 & \frac{\Lambda_m c_{hm} \mu n}{\Lambda_h \mu_m \varpi_7} & \frac{\Lambda_m c_{hm} \mu n}{\Lambda_h \mu_m \varpi_7} & 0 & \frac{\Lambda_m c_{hm} \mu n}{\Lambda_h \mu_m \varpi_7} & 0 & 0 \\ 0 & \frac{\Lambda_m c_{hm} \mu n \sigma_m}{\Lambda_h \mu_m^2 \varpi_7} & \frac{\Lambda_m c_{hm} \mu n \sigma_m}{\Lambda_h \mu_m^2 \varpi_7} & 0 & \frac{\Lambda_m c_{hm} \mu n \sigma_m}{\Lambda_h \mu_m^2 \varpi_7} & 0 & 0 \end{pmatrix}.$$

Since $\frac{S_h}{N_h} \leq 1$, then $f(x,y) \geq 0$. Again, we observe that $F \geq 0, V^{-1} \geq 0$ and $f\left(x, (\frac{\Lambda_h}{\mu}, 0, \frac{\Lambda_m}{\mu_m})^T\right) = 0$ in the invariant feasible set Ω. Therefore, since the matrix $V^{-1}F$ is reducible, we use theorem 2.1 of (Shuai & Driessche, 2013) to construct the Lyapunov function of the model system (5)-(14). For this construction, let suppose that $\omega^T = (\omega_1, \omega_2, \omega_3, \omega_4, \omega_5, \omega_6, \omega_7) \geq 0$ be the left eigenvector of the non-negative matrix $V^{-1}F$ corresponding to the eigenvalue $\mathcal{R}_0$. Then:

$$(\omega_1, \omega_2, \omega_3, \omega_4, \omega_5, \omega_6, \omega_7) V^{-1}F = \mathcal{R}_0 (\omega_1, \omega_2, \omega_3, \omega_4, \omega_5, \omega_6, \omega_7) \tag{45}$$

The left hand side of Equality (45) gives:

$$\begin{aligned} \omega^T V^{-1}F &= \left(0, \frac{\Lambda_m c_{hm} \mu n}{\mu_m \varpi_7 \Lambda_h} \omega_6 + \frac{\Lambda_m c_{hm} \mu n \sigma_m}{\mu_m^2 \varpi_7 \Lambda_h} \omega_7, \frac{\Lambda_m c_{hm} \mu n}{\mu_m \varpi_7 \Lambda_h} \omega_6 + \frac{\Lambda_m c_{hm} \mu n \sigma_m}{\mu_m^2 \varpi_7 \Lambda_h} \omega_7, 0, \right. \\ &\quad \frac{\Lambda_m c_{hm} \mu n}{\mu_m \varpi_7 \Lambda_h} \omega_6 + \frac{\Lambda_m c_{hm} \mu n \sigma_m}{\mu_m^2 \varpi_7 \Lambda_h} \omega_7, 0, \frac{c_{mh} n}{\varpi_1} \omega_1 + \frac{c_{mh} n r \sigma}{\varpi_1 \varpi_2} \omega_2 + A \omega_3 \\ &+ \left. \frac{k_1 c_{mh} n}{k_2 \varpi_1 \varpi_2} \omega_4 + B \omega_5 \right), \end{aligned} \tag{46}$$

where $A = \frac{c_{mh} n}{\varpi_1 \varpi_4} \left((1-r)\sigma + \frac{k_1 \varpi_3}{k_2 \varpi_2}\right)$ and $B = \frac{c_{mh} n}{\varpi_6} \left(\alpha_3 \gamma_2 k_3 + \frac{\gamma_1 r \sigma \xi_3}{\varpi_1 \varpi_2}\right)$.

Equating relations (45) and (46), we obtain: $\omega_1 = \omega_4 = \omega_6 = 0$, and $\omega_2 = \omega_3 = \omega_5 = \frac{\Lambda_m c_{hm} \mu n \sigma_m}{\mu_m^2 \varpi_7 \Lambda_h \mathcal{R}_0} \omega_7$, with $\omega_7 > 0$. Consequently,

$\omega^T = \left(0, \frac{\Lambda_m c_{hm}\mu n\sigma_m}{\mu_m^2\varpi_7\Lambda_h\mathcal{R}_0}\omega_7, \frac{\Lambda_m c_{hm}\mu n\sigma_m}{\mu_m^2\varpi_7\Lambda_h\mathcal{R}_0}\omega_7, 0, \frac{\Lambda_m c_{hm}\mu n\sigma_m}{\mu_m^2\varpi_7\Lambda_h\mathcal{R}_0}\omega_7, 0, \omega_7\right)$. By theorem 2.1 of (Shuai & Driessche, 2013), the function L defined by:

$$\begin{aligned} L &= \omega^T V^{-1} x \\ &= \frac{\Lambda_m c_{hm}\mu n\sigma_m}{\mu_m^2\varpi_7\Lambda_h\mathcal{R}_0}\left[\left(\frac{r\sigma}{\varpi_1\varpi_2} + \frac{(1-r)\sigma}{\varpi_1\varpi_4} + \frac{k_1\varpi_3}{k_2\varpi_1\varpi_2\varpi_4} + k_7\right)E_h\right. \\ &+ \left(\frac{1}{\varpi_2}\left(1 + \frac{\gamma_1\xi_2\varpi_3}{k_2} + \frac{\gamma_1\xi_3(\alpha_3\gamma_2\varpi_3 + k_2)}{k_2\varpi_6}\right)\right)A_h + \left(\frac{1}{\varpi_4}\left(1 + \frac{\alpha_2\gamma_2\varpi_3}{k_2} + \frac{\alpha_3\gamma_2(\alpha_2\gamma_2\varpi_3 + k_2)}{k_2\varpi_6}\right)\right)I_h \\ &+ \left.\frac{\varpi_3}{k_2}\left(1 + \frac{\alpha_3\gamma_2}{\varpi_6}\right)M_h + \frac{1}{\varpi_6}T_h\right]\omega_7 + \frac{\sigma_m\omega_7}{\mu_m\varpi_7}E_m + \frac{\omega_7}{\mu_m}I_m \end{aligned}$$

is a candidate Lyapunov function for system (5)-(14). The differentiation of L with respect to time t gives:

$$\begin{aligned} \dot{L} &= (\mathcal{R}_0 - 1)\omega^T x - \omega^T V^{-1} f(x,y) \\ &= (\mathcal{R}_0 - 1)\left[\frac{\Lambda_m c_{hm}\mu n\sigma_m}{\mu_m^2\varpi_7\Lambda_h\mathcal{R}_0}(A_h + I_h + T_h) + I_m\right]\omega_7 \\ &- \frac{\Lambda_m c_{hm}c_{mh}\mu n^2\sigma_m\omega_7 I_m}{\mu_m^2\varpi_7\varpi_1\Lambda_h\mathcal{R}_0}\left[\frac{r\sigma}{\varpi_2} + \frac{(1-r)\sigma}{\varpi_4} + \frac{k_1\varpi_3}{k_2\varpi_2\varpi_4} + \varpi_1 k_7\right]\left(1 - \frac{S_h}{N_h}\right). \end{aligned} \tag{47}$$

Since $\frac{S_h}{N_h} \leq 1$, if $\mathcal{R}_0 \leq 1$, then $\dot{L} < 0$, which implies that function L is a Lyapunov function for the system (5)-(14). Furthermore, $\dot{L} = 0$ implies that $A_h = I_h = T_h = I_m = 0$. Therefore, the largest invariant set of the model when $\dot{L} = 0$ in $int(\Omega)$ is the singleton $\{X^0\}$. Thus, by the LaSalle's invariance principle (LaSalle, 1976), the disease-free equilibrium X^0 is globally asymptotically stable provided that $\mathcal{R}_0 \leq 1$. In addition, if $\mathcal{R}_0 > 1$, then $\dot{L} > 0$ for $\frac{S_h}{N_h} = 1$. Therefore, by continuity, $\dot{L}$ remains positive in a small neighborhood of the disease-free equilibrium X^0, implies that X^0 is unstable when $\mathcal{R}_0 > 1$. Using theorem 2.1 of (Shuai & Driessche, 2013), the system (5)-(14) is uniformly persistent implies that there exists at least one endemic equilibrium of model system (5)-(14), noted by X^*. □

3.4 Endemic Equilibrium (EE)

Let $X^* = (S_h^*, E_h^*, A_h^*, I_h^*, R_h^*, M_h^*, T_h^*, S_m^*, E_m^*, I_m^*)$ be the endemic equilibrium (EE) of the model system (5)-(14). Then, one can obtain the endemic equilibrium by setting that the right hand side of system (5)-(14) is equal to zero. That is:

$$\left\{\begin{array}{rcl} 0 &=& \Lambda_h - (\lambda_h^* + \mu)S_h^* + \eta R_h^* \\ 0 &=& \lambda_h^* S_h^* - (\sigma + \mu)E_h^* \\ 0 &=& r\sigma E_h^* - (\gamma_1 + \mu)A_h^* \\ 0 &=& (1-r)\sigma E_h^* + \psi_2(1-\rho_2)M_h^* - (\gamma_2 + \delta_1 + \mu)I_h^* \\ 0 &=& \gamma_1\xi_1 A_h^* + \gamma_2\alpha_1 I_h^* + \psi_1 T_h^* + \psi_2\rho_2 M_h^* - (\eta + \mu)R_h^* \\ 0 &=& \gamma_1\xi_2 A_h^* + \gamma_2\alpha_2 I_h^* - (\psi_2 + \mu)M_h^* \\ 0 &=& \gamma_1\xi_3 A_h^* + \gamma_2\alpha_3 I_h^* - (\psi_1 + \mu + \delta_2)T_h^* \\ 0 &=& \Lambda_m - (\lambda_m^* + \mu_m)S_m^* \\ 0 &=& \lambda_m^* S_m^* - (\sigma_m + \mu_m)E_m^* \\ 0 &=& \sigma_m E_m^* - \mu_m I_m^* \end{array}\right. ; \tag{48}$$

where $\lambda_m^* = c_{hm}n\frac{(A_h^* + I_h^* + T_h^*)}{N_h^*}$ and $\lambda_h^* = c_{mh}n\frac{I_m^*}{N_h^*}$. After some algebraic computations of the system (48), we obtain the endemic equilibrium $X^* = (S_h^*, E_h^*, A_h^*, I_h^*, R_h^*, M_h^*, T_h^*, S_m^*, E_m^*, I_m^*)$,

where components are given respectively by the following implicit formula : $S_h^* = \dfrac{\Lambda_h + \eta R_h^*}{\lambda_h^* + \mu}$,
$E_h^* = \dfrac{\lambda_h^* S_h^*}{\sigma+\mu}, A_h^* = \dfrac{r\sigma E_h^*}{\gamma_1+\mu}, I_h^* = \dfrac{r\sigma(1-\rho_2)\psi_2\gamma_1\xi_2 + (1-r)\sigma(\gamma_1+\mu)(\psi_2+\mu)}{r\sigma(\psi_2+\mu)(\gamma_2+\delta_1+\mu) - r\sigma(1-\rho_2)\psi_2\gamma_2\alpha_2} A_h^*$,
$R_h^* = \dfrac{\gamma_1\xi_1 A_h^* + \gamma_2\alpha_1 I_h^* + \psi_2\rho_2 M_h^* + \psi_1 T_h^*}{\eta+\mu}, M_h^* = \dfrac{\gamma_1\xi_2 A_h^* + \gamma_2\alpha_2 I_h^*}{\psi_2+\mu}, T_h^* = \dfrac{\gamma_1\xi_3 A_h^* + \gamma_2\alpha_3 I_h^*}{\psi_1+\mu+\delta_2}$,
$S_m^* = \dfrac{\Lambda_m}{\lambda_m^*+\mu_m}, E_m^* = \dfrac{\lambda_m^*\Lambda_m}{(\sigma_m+\mu_m)(\lambda_m^*+\mu_m)}$ and $I_m^* = \dfrac{\sigma_m\lambda_m^*\Lambda_m}{\mu_m(\sigma_m+\mu_m)(\lambda_m^*+\mu_m)}$.

Theorem 4. *[GAS of the EE] If $\mathcal{R}_0 > 1$, the endemic equilibrium X^* is GAS in Ω.*

Proof. We use Lyapunov function theory to prove the GAS. To construct the Lyapunov function, we use graph-theoretical method explained in (Shuai & Driessche, 2013). Let:

$L_1 = S_h - S_h^* - S_h^* \ln(S_h/S_h^*), L_2 = E_h - E_h^* - E_h^* \ln(E_h/E_h^*), L_3 = A_h - A_h^* - A_h^* \ln(A_h/A_h^*)$
$L_4 = I_h - I_h^* - I_h^* \ln(I_h/I_h^*), L_5 = R_h - R_h^* - R_h^* \ln(R_h/R_h^*), L_6 = M_h - M_h^* - M_h^* \ln(M_h/M_h^*)$
$L_7 = T_h - T_h^* - T_h^* \ln(T_h/T_h^*), L_8 = S_m - S_m^* - S_m^* \ln(S_m/S_m^*), L_9 = E_m - E_m^* - E_m^* \ln(E_m/E_m^*)$
$L_{10} = I_m - I_m^* - I_m^* \ln(I_m/I_m^*)$

Solving each equation of system (48) leads to the following quantities:

$\Lambda_h = \left(c_{mh} n \dfrac{I_m^*}{N_h^*} + \mu\right) S_h^* - \eta R_h^*, \sigma + \mu = \dfrac{c_{mh} n I_m^* S_h^*}{N_h^* E_h^*}, \gamma_1 + \mu = r\sigma E_h^*/A_h^*$
$\gamma_2 + \delta_1 + \mu = ((1-r)\sigma E_h^* + \psi_2(1-\rho_2)M_h^*)/I_h^*, \eta + \mu = (\gamma_1\xi_1 A_h^* + \gamma_2\alpha_1 I_h^* + \psi_1 T_h^* + \psi_2\rho_2 M_h^*)/R_h^*$
$\psi_2 + \mu = (\gamma_1\xi_2 A_h^* + \gamma_2\alpha_2 I_h^*)/M_h^*, \psi_1 + \delta_2 + \mu = (\gamma_1\xi_3 A_h^* + \gamma_2\alpha_3 I_h^*)/T_h^*$
$\Lambda_m = \left(\dfrac{c_{hm} n}{N_h^*}(A_h^* + I_h^* + T_h^*) + \mu_m\right) S_m^*, \sigma_m + \mu_m = \dfrac{c_{hm} n (A_h^* + I_h^* + T_h^*) S_m^*}{N_h^* E_m^*}$ and $\mu_m = \sigma_m E_m^*/I_m^*$.

Using inequalities $1 - x + \ln x \le 0$ and $2 - x - \frac{1}{x} \le 0$ for $x > 0$ in the differentiation of $L_i, i \in \{1,2,3,4,5,6,7,8,9,10\}$ with respect to t give:

$$\begin{aligned}
\dot{L}_1 &= \left(1 - \frac{S_h^*}{S_h}\right)\dot{S}_h = \left(1 - \frac{S_h^*}{S_h}\right)\left(\Lambda_h - \left(c_{mh} n \frac{I_m}{N_h} + \mu\right) S_h + \eta R_h\right) \\
&\le c_{mh} n I_m^* \left(\frac{I_m}{I_m^*} - \frac{S_h^*}{S_h} - \ln\frac{I_m}{I_m^*} - \ln\frac{S_h^*}{S_h}\right) + \eta R_h^* \left(\frac{R_h}{R_h^*} + \frac{S_h^*}{S_h} - \ln\frac{S_h^*}{S_h} - \ln\frac{R_h}{R_h^*}\right) \\
&= a_{1,10} G_{1,10} + a_{15} G_{15} \\
\dot{L}_2 &= \left(1 - \frac{E_h^*}{E_h}\right)\dot{E}_h = \left(1 - \frac{E_h^*}{E_h}\right)\left(c_{mh} n \frac{I_m}{N_h} S_h - (\sigma+\mu) E_h\right) \\
&= c_{mh} n I_m^* \left(\frac{I_m}{I_m^*} - \frac{E_h}{E_h^*} - \frac{I_m h E_h^*}{I_m^* E_h} + 1\right) \le c_{mh} n I_m^* \left(\frac{I_m}{I_m^*} - \frac{E_h}{E_h^*} - \ln\frac{E_h^*}{E_h} - \ln\frac{I_m}{I_m^*}\right) = a_{2,10} G_{2,10} \\
\dot{L}_3 &= \left(1 - \frac{A_h^*}{A_h}\right)\dot{A}_h = \left(1 - \frac{A_h^*}{A_h}\right)(r\sigma E_h - (\gamma_1+\mu) A_h) = r\sigma E_h^* \left(\frac{E_h}{E_h^*} - \frac{A_h}{A_h^*} - \frac{A_h^* E_h}{A_h E_h^*} + 1\right) \\
&\le r\sigma E_h^* \left(\frac{E_h}{E_h^*} - \frac{A_h}{A_h^*} - \ln\frac{A_h^*}{A_h} - \ln\frac{E_h}{E_h^*}\right) = a_{32} G_{32}
\end{aligned}$$

$$
\begin{aligned}
\dot{L}_4 &= \left(1-\frac{I_h^*}{I_h}\right)\dot{I}_h = \left(1-\frac{I_h^*}{I_h}\right)((1-r)\sigma E_h + \psi_2(1-\rho_2)M_h - (\gamma_2+\delta_1+\mu)I_h) \\
&\leq (1-r)\sigma E_h^*\left(\frac{E_h}{E_h^*} - \frac{I_h}{I_h^*} - \ln\frac{I_h^*}{I_h} - \ln\frac{E_h}{E_h^*}\right) + \psi_2(1-\rho_2)M_h^*\left(\frac{M_h}{M_h^*} - \frac{I_h}{I_h^*} - \ln\frac{I_h^*}{I_h} - \ln\frac{M_h}{M_h^*}\right) \\
&= a_{42}G_{42} + a_{46}G_{46} \\
\dot{L}_5 &= \left(1-\frac{R_h^*}{R_h}\right)\dot{R}_h = \left(1-\frac{R_h^*}{R_h}\right)(\gamma_1\xi_1 A_h + \gamma_2\alpha_1 I_h + \psi_1 T_h + \psi_2\rho_2 M_h - (\eta+\mu)R_h) \\
&\leq \gamma_1\xi_1 A_h^*\left(\frac{A_h}{A_h^*} - \frac{R_h}{R_h^*} - \ln\frac{R_h^*}{R_h} - \ln\frac{A_h}{A_h^*}\right) + \gamma_2\alpha_1 I_h^*\left(\frac{I_h}{I_h^*} - \frac{R_h}{R_h^*} - \ln\frac{R_h^*}{R_h} - \ln\frac{I_h}{I_h^*}\right) \\
&+ \psi_1 T_h^*\left(\frac{T_h}{T_h^*} - \frac{R_h}{R_h^*} - \ln\frac{R_h^*}{R_h} - \ln\frac{T_h}{T_h^*}\right) + \psi_2\rho_2 M_h^*\left(\frac{M_h}{M_h^*} - \frac{R_h}{R_h^*} - \ln\frac{R_h^*}{R_h} - \ln\frac{M_h}{M_h^*}\right) \\
&= a_{53}G_{53} + a_{54}G_{54} + a_{57}G_{57} + a_{56}G_{56} \\
\dot{L}_6 &= \left(1-\frac{M_h^*}{M_h}\right)\dot{M}_h = \left(1-\frac{M_h^*}{M_h}\right)(\gamma_1\xi_2 A_h + \gamma_2\alpha_2 I_h - (\psi_2+\mu)M_h) \\
&\leq \gamma_1\xi_2 A_h^*\left(\frac{A_h}{A_h^*} - \frac{M_h}{M_h^*} - \ln\frac{M_h^*}{M_h} - \ln\frac{A_h}{A_h^*}\right) + \gamma_2\alpha_2 I_h^*\left(\frac{I_h}{I_h^*} - \frac{M_h}{M_h^*} - \ln\frac{M_h^*}{M_h} - \ln\frac{I_h}{I_h^*}\right) \\
&= a_{63}G_{63} + a_{64}G_{64} \\
\dot{L}_7 &= \left(1-\frac{T_h^*}{T_h}\right)\dot{T}_h = \left(1-\frac{T_h^*}{T_h}\right)(\gamma_1\xi_3 A_h + \gamma_2\alpha_3 I_h - (\psi_1+\mu+\delta_2)T_h) \\
&\leq \gamma_1\xi_3 A_h^*\left(\frac{A_h}{A_h^*} - \frac{T_h}{T_h^*} - \ln\frac{T_h^*}{T_h} - \ln\frac{A_h}{A_h^*}\right) + \gamma_2\alpha_3 I_h^*\left(\frac{I_h}{I_h^*} - \frac{T_h}{T_h^*} - \ln\frac{T_h^*}{T_h} - \ln\frac{I_h}{I_h^*}\right) \\
&= a_{73}G_{73} + a_{74}G_{74} \\
\dot{L}_8 &= \left(1-\frac{S_m^*}{S_m}\right)\dot{S}_m = \left(1-\frac{S_m^*}{S_m}\right)\left(\Lambda_m - \left(c_{hm}n\frac{A_h+I_h+T_h}{N_h} + \mu_m\right)S_m\right) \\
&= 3c_{hm}nS_m^*\left(1 - \frac{S_m}{S_m^*} - \frac{S_m^*}{S_m} + 1\right) + \mu_m S_m^*\left(1 - \frac{S_m}{S_m^*} - \frac{S_m^*}{S_m} + 1\right) \leq 0 \\
\dot{L}_9 &= \left(1-\frac{E_m^*}{E_m}\right)\dot{E}_m = \left(1-\frac{E_m^*}{E_m}\right)\left(c_{hm}n\frac{A_h+I_h+T_h}{N_h}S_m - (\sigma_m+\mu_m)E_m\right) \\
&= 3c_{hm}nS_m^*\left(\frac{S_m}{S_m^*} - \frac{E_m}{E_m^*} - \frac{E_m^*S_m}{E_mS_m^*} + 1\right) \leq 3c_{hm}nS_m^*\left(\frac{S_m}{S_m^*} - \frac{E_m}{E_m^*} - \ln\frac{E_m^*}{E_m} - \ln\frac{S_m}{S_m^*}\right) \\
&= a_{98}G_{98}
\end{aligned}
$$

$$
\begin{aligned}
\dot{L}_{10} &= \left(1-\frac{I_m^*}{I_m}\right)\dot{I}_m = \left(1-\frac{I_m^*}{I_m}\right)(\sigma_m E_m - \mu_m I_m) = \sigma_m E_m^*\left(\frac{E_m}{E_m^*} - \frac{I_m}{I_m^*} - \frac{I_m^*E_m}{I_mE_m^*} + 1\right) \\
&\leq \sigma_m E_m^*\left(\frac{E_m}{E_m^*} - \frac{I_m}{I_m^*} - \ln\frac{I_m^*}{I_m} - \ln\frac{E_m}{E_m^*}\right) = a_{10,9}G_{10,9}
\end{aligned}
$$

From these derivatives, the weighted associated digraph (G, A) is shown on Figure (2).

The weighted matrix A of the digraph is $A = [a_{ij}]_{10\times 10}$, where $a_{1,10} = a_{2,10} = c_{mh}nI_m^*, a_{15} = \eta R_h^*, a_{32} = r\sigma E_h^*, a_{46} = \psi_2(1-\rho_2)M_h^*, a_{42} = (1-r)\sigma E_h^*, a_{53} = \gamma_1\xi_1 A_h^*, a_{54} = \gamma_2\alpha_1 I_h^*, a_{57} = \psi_1 T_h^*, a_{56} = \psi_2\rho_2 M_h^*, a_{73} = \gamma_1\xi_3 A_h^*, a_{74} = \gamma_2\alpha_3 I_h^*, a_{63} = \gamma_1\xi_2 A_h^*, a_{64} = \gamma_2\alpha_2 I_h^*, a_{10,9} = \sigma_m E_m^*, a_{98} = 3c_{hm}nS_m^*$ and all other $a_{ij} = 0$. The value a_{ij} represents the weight of arc (j, i). From all these calculations, the first condition of Theorem 3.5 of (Shuai & Driessche, 2013) is satisfied. Let check now the second condition of the cited theorem. In the only one directed cycle, we have $G_{64} + G_{46} = 0$. Then, the second condition of Theorem 3.5 of (Shuai & Driessche, 2013) is also

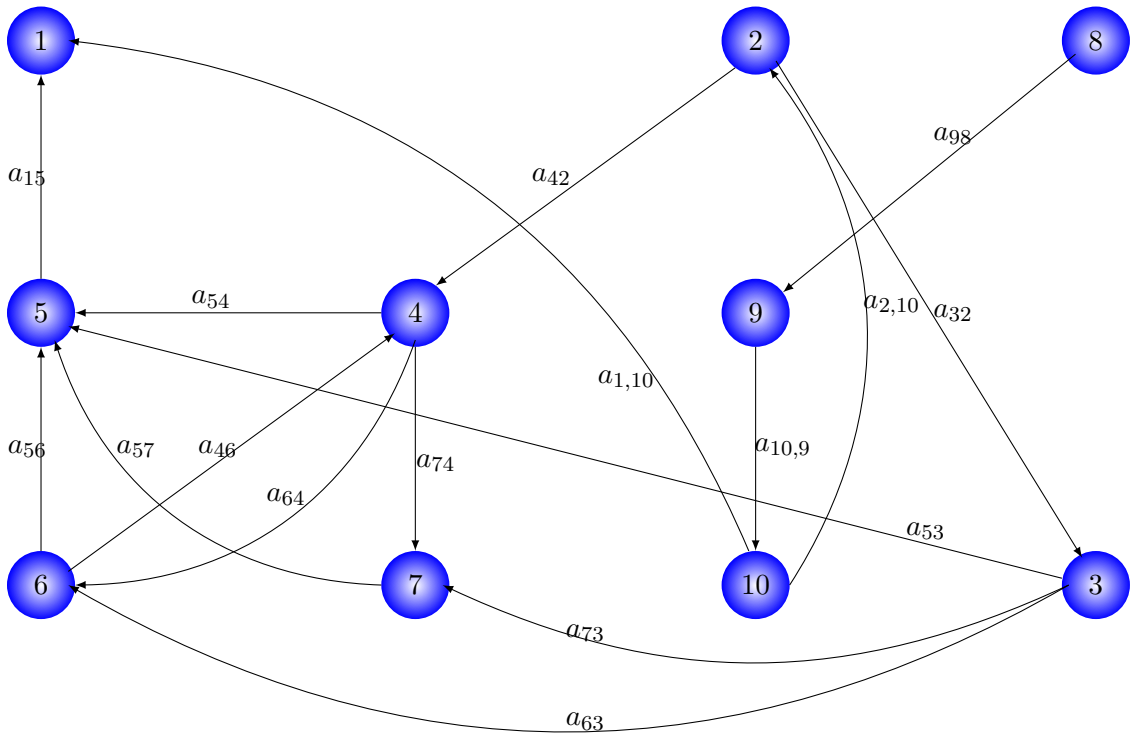


Figure 2: The weighted digraph (G, A) constructed from derivatives $\dot{L}_i$.

satisfied. Therefore, by the cited theorem, there exists $c_i, i \in \{1, 2, 3, 4, 5, 6, 7, 8, 9, 10\}$ such that

$$\mathcal{L} = \sum_{i=1}^{10} c_i L_i, \tag{49}$$

is a Lyapunov function for the model system (5)-(14). The relations between c_i can be derived from Theorems (3.3) and (3.4) of (Shuai & Driessche, 2013) as follow: $c_1 a_{15} = c_5(a_{53} + a_{54} + a_{56} + a_{57}); c_5 a_{57} = c_7(a_{73} + a_{74}); c_{10} a_{10,9} = c_9 a_{98}; c_{10} a_{10,9} = c_1 a_{1,10} + c_2 a_{2,10}; c_2 a_{2,10} = c_3 a_{32} + c_4 a_{42}; c_3 a_{32} = c_7 a_{73} + c_6 a_{63}$. Therefore, $c_1 = c_5(a_{53} + a_{54} + a_{56} + a_{57})/a_{15}; c_5 = c_7(a_{73} + a_{74})/a_{57}; c_2 = (c_3 a_{32} + c_4 a_{42})/a_{2,10}; c_{10} = (c_1 a_{1,10} + c_2 a_{2,10})/a_{10,9}; c_9 = c_{10} a_{10,9}/a_{98}$ and $c_3 = (c_6 a_{63} + c_7 a_{73})/a_{32}$. The fact that $\dot{\mathcal{L}} = \sum_{i=1}^{10} c_i \dot{L}_i \leq 0$, implies that $X = X^*$. Consequently, the largest invariant set for system (5)-(14) where $\dot{L} = 0$ is the singleton set $\{X^*\}$. This proves the uniqueness and global asymptotic stability of $\{X^*\}$ in the interior of Ω provided that $\mathcal{R}_0 > 1$. □

4 Numerical analysis

In this section, we present a quantitative analysis. After introducing the parameters, we conduct the local sensitivity analysis and conclude with numerical simulations to support our qualitative findings.

4.1 Parameters presentation

We present here the baseline values of parameters which will be used for numerical simulations. Most of them, have been taken from the literature (Fatmawati et al., 2018; Mangongo et al., 2022; Ndoen et al., 2012; Olaniyi et al., 2020) and others are computed and reasonable assumed. The Table 2 gives description, baseline value, range for each parameter and their related sources. The parameters $\sigma, \delta_1, \delta_2, \gamma_1, \gamma_2, \xi_1, \xi_2, \xi_3, \alpha_1, \alpha_2, \alpha_3, \eta, \psi_1, \psi_2, \rho_2$ and σ_m have for dimension day^{-1}.

The parameters c_{hm} and c_{mh} are dimensionless. The parameters μ and μ_m have for dimensions $humans^{-1} \times day^{-1}$ and $mosquitoes^{-1} \times day^{-1}$ respectively. The parameters Λ_h and Λ_m have for dimensions $humans \times day^{-1}$ and $mosquitoes \times day^{-1}$ respectively.

Table 2: Baseline values for parameters of the model (5)-(14)

Param	**Descriptions**	Values	Range of values	Ref.
Λ_h	recruitment rate of humans	11,883.26		computed
c_{hm}	probability that a bite by a susceptible mosquito on a symptomatic, asymptomatic or resistant strains humans leads to infection of the mosquito	0.64	$[0, 0.8]$	(Agusto & Tchuenche, 2013)
c_{mh}	probability that a bite by an infectious mosquito on a susceptible human leads to infection of the human	0.64	$[0, 0.8]$	(Agusto & Tchuenche, 2013)
σ	latent rate of humans	0.054	-	(Ndoen et al., 2012)
r	proportion of asymptomatic humans	0.67	-	(Andolina et al., 2021)
μ	natural death rate of humans	0.00421	$[0, 0.05]$	(Agusto & Tchuenche, 2013)
δ_1	malaria-induced death rate of symptomatic humans	0.0001285	-	(Mangongo et al., 2022)
δ_2	malaria-induced death rate of resistant strains humans	0.0001285	-	(Mangongo et al., 2022)
γ_1	recovery rate of asymptomatic humans	0.071	$[1/1500, 1]$	(Kamaldeen et al., 2019)
γ_2	recovery rate of symptomatic humans	0.71	$[1/1500, 1]$	(Kamaldeen et al., 2019)
ξ_1	proportion of asymptomatic humans who recover	0.2	-	(Mangongo et al., 2022)
ξ_3	proportion of resistant strains infected among asymptomatic humans	0.6	-	assumed
α_1	proportion of symptomatic humans who recover	0.2	-	(Mangongo et al., 2022)
α_3	proportion of resistant strains infected among symptomatic humans	0.6	-	assumed
η	rate of loss of acquired immunity	0.02	-	(Mangongo et al., 2022)
ψ_1	recovery rate of resistant strains individuals	0.0471	$[1/1500, 1]$	(Kamaldeen et al., 2019)
ψ_2	relapse rate	0.5	-	assumed
ρ_2	proportion of ignorant infected who recover	0.2	-	(Mangongo et al., 2022)
Λ_m	recruitment rate of mosquitoes	1,000	-	(Cai et al., 2017)
μ_m	natural death rate of mosquitoes	0.1435	$[0.02, 0.2]$	(Anguelov et al., 2012) (Strugarek et al., 2018) (Zhang et al., 2020)
n	the average number of mosquito bites	25	$[0.1, 50]$	(Tchoumi et al., 2023)
σ_m	latent rate of mosquitoes	0.0769		(Mangongo et al., 2022)

To compute Λ_h, we use the following formula $\Lambda_h = \frac{\tau \times N_h}{365 \times 1000}$, where τ is the birth rate per 1,000 per year. Taking the birth rate per 1000 per year of the Democratic Republic of Congo (DRC) for 2023, which is estimated to $\tau = 41$ per 1,000 habitants (WBG, 2025) and the population of DRC was about 105.79 million in 2023. Using these informations, we found $\Lambda_h = 11,883.26$. The latent period of malaria is ranged between 7 and 30 days Ndoen et al. (2012). Taking inverse of the mean of this range we obtain the latent rate equals to $\sigma = 0.054$. Because the asymptomatic malaria cases represent a high proportion of all *Plasmodium*

infections, we take $r = 2/3$ as the proportion of asymptomatic humans. We take equal the proportions $\alpha_1 = \xi_1 = 0.2$ of asymptomatic and symptomatic humans who recover (Mangongo et al., 2022). The numerical simulations will be done using the following initial conditions $S_{h0} = 50000, E_{h0} = 1000, A_{h0} = 1000, I_{h0} = 1000, R_{h0} = 5000, M_{h0} = 1000, T_{h0} = 1000, S_{m0} = 500, E_{m0} = 250, I_{m0} = 250$. That is $N_{h0} = 60000$ and $N_{m0} = 1000$.

4.2 Local sensitivity analysis

In this subsection, we provide the local sensitivity analysis of the basic reproduction number $\mathcal{R}_0$. In analysis of the spread of a disease, the role of threshold $\mathcal{R}_0$ should be highlighted, in sense that, it determines the extinction of a disease if it is less than unity ($\mathcal{R}_0 < 1$). The sensitivity analysis gives an idea of the most important parameters to reduce very significantly $\mathcal{R}_0$ in order to control malaria. Therefore, it's very important to analyze its sensitivity according to each control parameter composed it. For this purpose, we compute $\frac{\partial \mathcal{R}_0}{\partial \varrho}\frac{\varrho}{\mathcal{R}_0}$ for each parameter. This leads to the sensitivity indices, displayed in Table 3, which measure the ratio of the relative change in $\mathcal{R}_0$ to the relative change in parameter ϱ (Zhou & Liu, 2008).

Table 3: Sensitivity indices

param	formula: $\frac{\partial \mathcal{R}_0}{\partial \varrho}\frac{\varrho}{\mathcal{R}_0}$	values	sens. index
n	1	25	1
Λ_m	$\frac{1}{2}$	1,000	$\frac{1}{2}$
γ_2	$\frac{\gamma_2\left[\frac{\partial \mathcal{R}_{01}}{\partial \gamma_2}+\frac{\partial \mathcal{R}_{02}}{\partial \gamma_2}\right]}{2(\mathcal{R}_{01}+\mathcal{R}_{02})}-\frac{\gamma_2\frac{\partial k_2}{\partial \gamma_2}}{2k_2}$	0.71	-0.0453
ψ_2	$\frac{\psi_2\left[\frac{\partial \mathcal{R}_{01}}{\partial \psi_2}+\frac{\partial \mathcal{R}_{02}}{\partial \psi_2}\right]}{2(\mathcal{R}_{01}+\mathcal{R}_{02})}-\frac{\psi_2(\varpi_4-\alpha_2\gamma_2(1-\rho_2))}{2k_2}$	0.5	-0.0452
γ_1	$\frac{\gamma_1\varpi_2\left[\frac{\partial \mathcal{R}_{01}}{\partial \gamma_1}+\frac{\partial \mathcal{R}_{02}}{\partial \gamma_1}\right]}{2\varpi_2(\mathcal{R}_{01}+\mathcal{R}_{02})}-\frac{\gamma_1}{2\varpi_2}$	0.071	-0.00244
r	$\frac{\mathcal{R}_{01}}{2(\mathcal{R}_{01}+\mathcal{R}_{02})}$	0.67	0.000282
ξ_3	$\frac{r\gamma_1\xi_3k_2}{2(\mathcal{R}_{01}+\mathcal{R}_{02})}$	0.6	0.000116
ξ_2	$\frac{\gamma_1\xi_2\varpi_3(\gamma_2\alpha_3+\varpi_6)}{2(\mathcal{R}_{01}+\mathcal{R}_{02})}$	0.2	0.0000365
α_3	$\frac{\gamma_2\alpha_3(r\gamma_1\xi_2\varpi_3+(1-r)\varpi_2\varpi_5)}{2(\mathcal{R}_{01}+\mathcal{R}_{02})}$	0.6	0.00000935
α_2	$\frac{-\gamma_2\alpha_2\varpi_3(r(\varpi_6+\gamma_1\xi_3)-\mathcal{R}_{01}-\mathcal{R}_{02})}{2k_2(\mathcal{R}_{01}+\mathcal{R}_{02})}$	0.2	-0.00000837

The derivatives of $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ with respect to γ_1 are given by $\frac{\partial \mathcal{R}_{01}}{\partial \gamma_1} = rk_2\xi_3+\xi_2\varpi_3(\gamma_2\alpha_3+\varpi_6)$ and $\frac{\partial \mathcal{R}_{02}}{\partial \gamma_1} = (1-r)\varpi_5(\varpi_6+\gamma_2\alpha_3)$ respectively. The derivatives of $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ with respect to γ_2 are given by $\frac{\partial \mathcal{R}_{01}}{\partial \gamma_2} = r\gamma_1\xi_2\varpi_3\alpha_3 + r(\varpi_6+\gamma_1\xi_3)(\varpi_5-\alpha_2\varpi_3)$ and $\frac{\partial \mathcal{R}_{02}}{\partial \gamma_2} = (1-r)\varpi_2\varpi_5\alpha_3$ respectively. The derivatives of $\mathcal{R}_{01}$ and $\mathcal{R}_{02}$ with respect to ψ_2 are given by $\frac{\partial \mathcal{R}_{01}}{\partial \psi_2} = r[(\varpi_6+\gamma_1\xi_3)(\varpi_4-\alpha_2\gamma_2(1-\rho_2))+\gamma_1\xi_2(1-\rho_2)(\gamma_2\alpha_3+\varpi_6)]$ and $\frac{\partial \mathcal{R}_{02}}{\partial \psi_2} = (1-r)\varpi_2(\varpi_6+\gamma_2\alpha_3)$ respectively. Finally, the derivative of k_2 with respect to γ_2 is given by $\frac{\partial k_2}{\partial \gamma_2} = \varpi_5 - \alpha_2\varpi_3$.

We consider ten controllable parameters. Arranging in the descending order of the absolute value of their sensitivity indices, the average number of mosquito bites n takes the top of list with sensitivity index 1 as shown on Figure 3. This means, reducing mosquito bites ensure significantly the reduction of threshold $\mathcal{R}_0$. The recruitment rate of mosquitoes Λ_m comes in the second position with sensitivity index +0.5. So, we have to reduce the recruitment of

mosquitoes in order to control malaria. This can be implemented by maintaining a health environment by destroyed mosquito eggs in puddles. The recovery rate of symptomatic humans γ_2 comes in the third position with sensitivity index -0.0453. The recovery rate of asymptomatic humans γ_1 takes the fourth position with sensitivity index -0.00244.

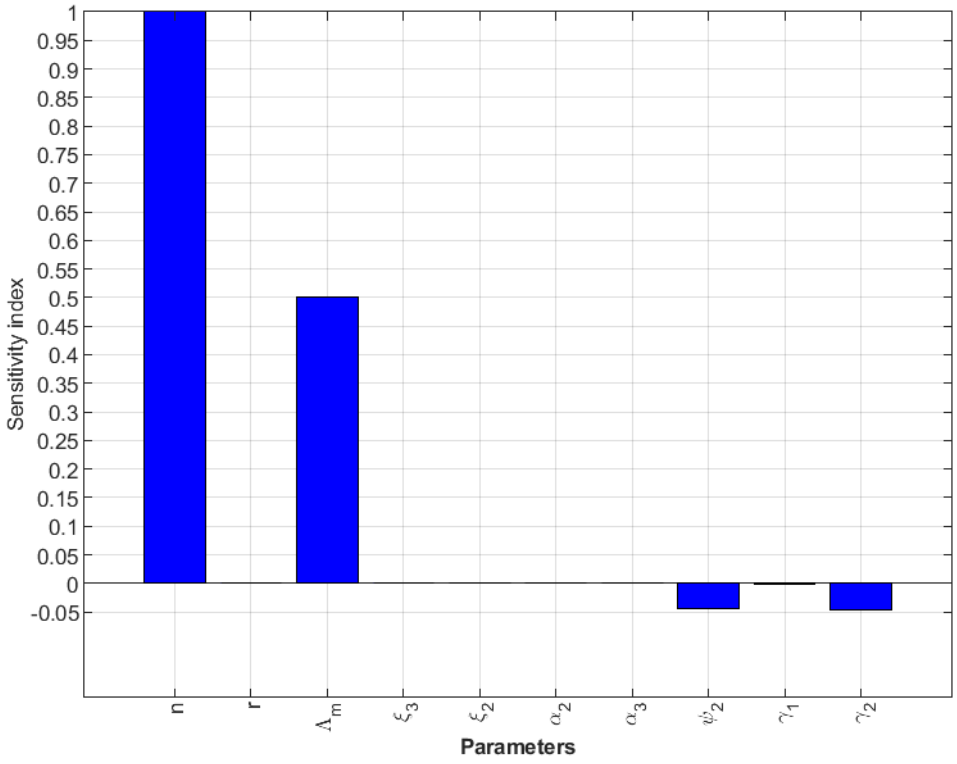


Figure 3: Sensitivity indices.

This shows the impact of asymptomatic humans in the spread of malaria. We have to increase the recovery rate of asymptomatic humans in order to reduce the basic reproduction number $\mathcal{R}_0$, which implies to control malaria. This can be implemented by a mass screening of humans population and their treatment until complete recovered. Parameters $r, \xi_3, \xi_2, \psi_2, \alpha_3$ and α_2 do not influence significantly the basic reproduction number because of their very low sensitivity indices. In the next subsection, we provide some numerical simulations.

4.3 Numerical simulations

In this subsection, we provide some numerical simulations for the model system (5)-(14) by giving first the endemic trends of both human and mosquito populations. We give the time evolution of the recovered individuals R_h for different values of the recovery rate of asymptomatic humans γ_1. In addition, we provide scenarios for resistant strains humans for different configurations of α_3 and ξ_3. Finally, we present a disease-free trend of the model.

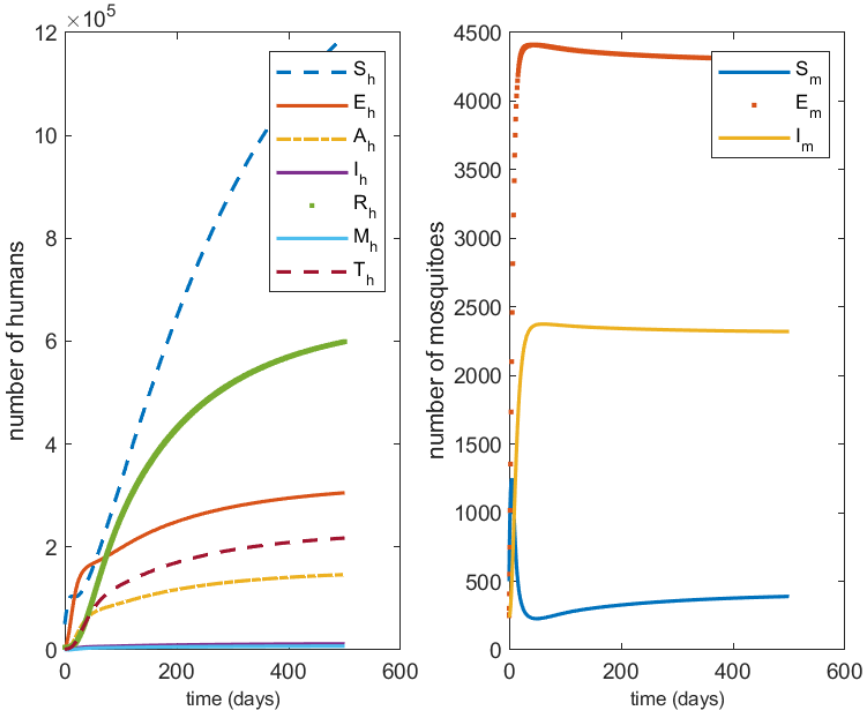


Figure 4: Endemic trends of humans and mosquitoes for parameter values on Table 2. We obtain $\mathcal{R}_0 = 1.03 > 1$ and the estimated endemic point is (1189340,305063,146230,11776,598736,7426,217163,390,4303,2321).

Figure 4 shows the endemic trends of both human and mosquito populations for parameter values on Table 2. The related basic reproduction number $\mathcal{R}_0 = 1.03$, is in the same range with the one obtained in the literature (Mangongo et al., 2022, 2021). This confirms the global endemicity situation which occurs when the basic reproduction number is greater than unity as proved in Theorem 4. That is, malaria continues to spread in the population.

Figures 5 and 6 show the important role of asymptomatic and resistant strains infected humans in the spread of malaria. We can state that all the previous known strategies (optimizing vector control and treatment of symptomatic cases) against malaria should be revised by taking into account the presence of asymptomatic humans and those with resistant strains. Parameters γ_1, α_3 and ξ_3 are highlighted the role of asymptomatic and resistant strains infected humans in the dynamics transmission of malaria.

Figure 5 shows the bad role of asymptomatic humans in the transmission dynamics of malaria. The number of recovered humans increases with the recovery rate of asymptomatic humans. We observe that with a high recovery rate of asymptomatic humans, many individuals (asymptomatic, symptomatic and ignorant infected humans) recover. When reducing the recovery rate of asymptomatic humans, a small number of individuals (asymptomatic, symptomatic and ignorant infected humans) recover. The role of recovery rate γ_1 of asymptomatic humans should be capitalized in order to control malaria transmission dynamics.

Figure 6 shows many comparison of resistant strains infected humans under different configurations of the proportion of resistant strains infected among symptomatic humans α_3 and the proportion of resistant strains infected among asymptomatic humans ξ_3. Subfigure (a) sets the value of ξ_3 at 0.1 and shows six configurations of T_h for six values of α_3. In subfigure (b), the value of ξ_3 is fixed at 0.2, and six configurations of T_h are given for six values of α_3. Subfigure (c) sets ξ_3 to 0.3 and presents six configurations of T_h corresponding to six values of α_3. Subfigure (d) fixes ξ_3 at 0.4 and illustrates six configurations of T_h for different values of α_3. In subfigure (e), ξ_3 is set to 0.5, and six configurations of T_h are displayed for six values of α_3.

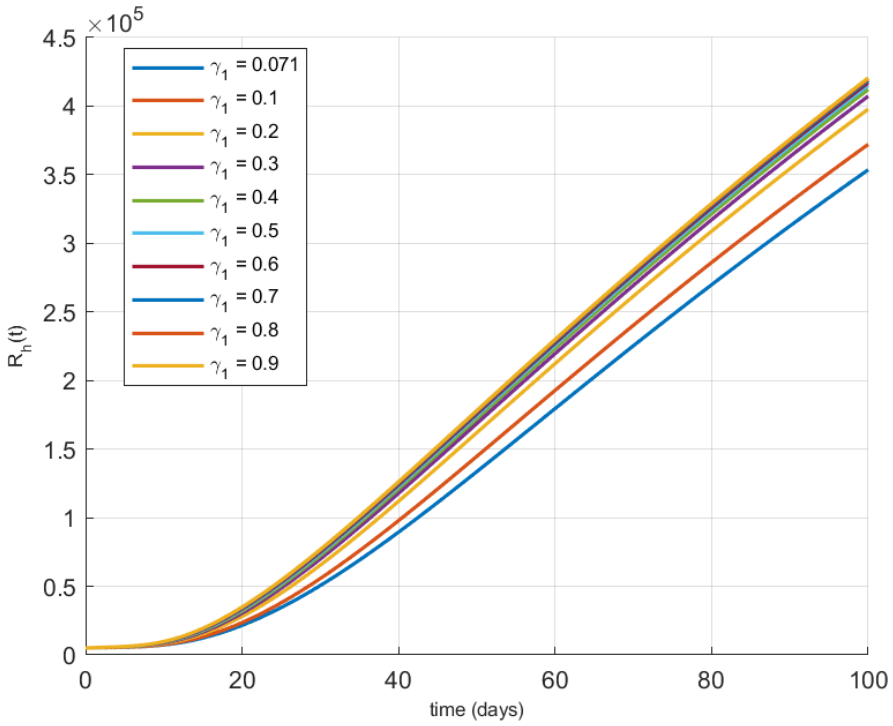


Figure 5: Evolution of recovered individuals for different values of γ_1.

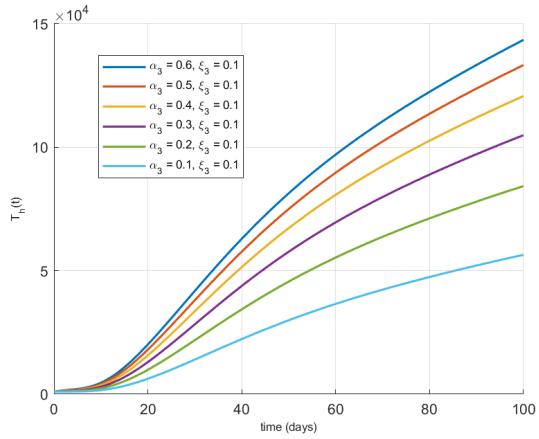
Finally, in subfigure (f), the value of ξ_3 is fixed at 0.6, and six configurations of T_h are shown for six values of α_3. The population of resistant strains infected humans is more influenced by the proportions α_3 and ξ_3. Reducing these proportions in the population help to reduce the number of individuals with resistant strains in the population. People should be treated until to be completely recovered.

Figure 7 shows the malaria free trends for both humans and mosquitoes for parameter values on Table 2 except $\gamma_1 = 0.5, n = 5$ and $\Lambda_m = 500$. We obtain the basic reproduction number $\mathcal{R}_0 = 0.81 < 1$ and the estimated disease-free point (2500000,0,0,0,0,0,0,3500,0,0). This shows the global stability of the disease-free when the basic reproduction number is less than unity. This confirms Theorem 3. We reach the disease-free equilibrium by reducing parameters Λ_m and N, and by increasing parameter γ_1. This shows again the important role of asymptomatic humans in the spread of malaria. That is, we have to increase the recovery rate of asymptomatic humans which will reduce their number on the human populations.

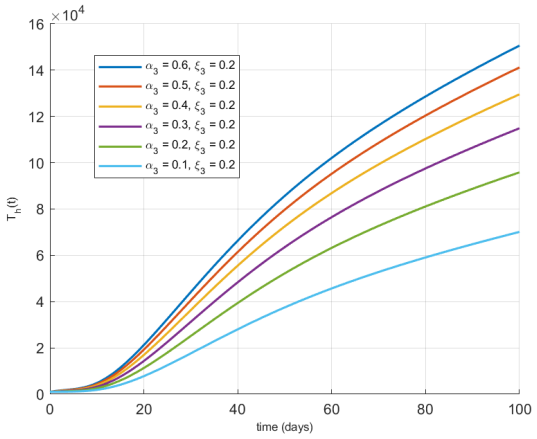
5 Discussion and concluding remarks

In this paper, we analyzed a model of malaria transmission dynamics by incorporating the asymptomatic and resistant strains individuals in the human populations. To describe the dynamics of mosquito populations, we used the common SEI scheme. After proving the well posedness of the model, we compute the basic reproduction number $\mathcal{R}_0$, which is composed of two different terms ($\mathcal{R}_{01}$ and $\mathcal{R}_{02}$, contributions of asymptomatic and symptomatic humans). All these contributions are weighted by the mosquito biting term. We proved that the disease-free equilibrium (DFE) of the model is global asymptotically stable (GAS) whenever the basic reproduction number is less than or equal to unity. For the case where the basic reproduction number is greater than unity, we analyzed the global asymptotic stability of the endemic equilibrium (EE).

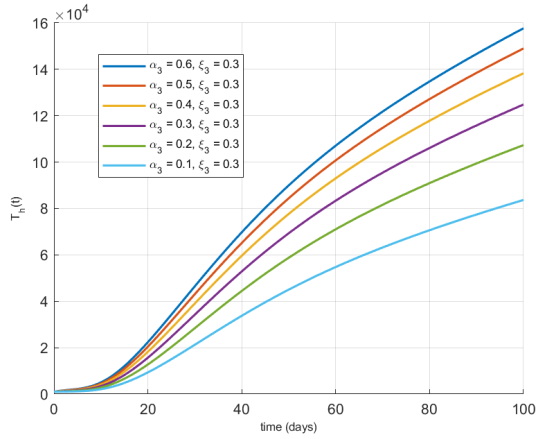
Sensitivity analysis, Table 3, reveals that the basic reproduction number $\mathcal{R}_0$, consequently



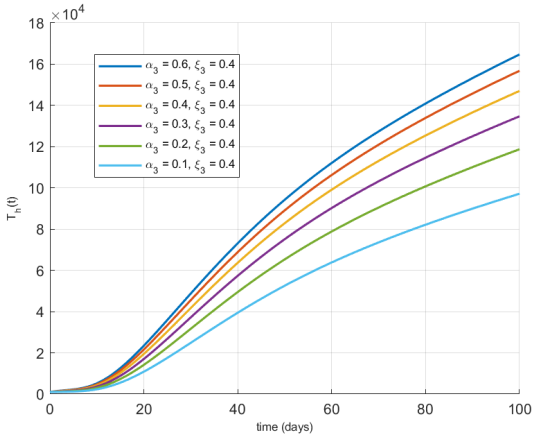
(a) $\xi_3 = 0.1$ and $\alpha_3 = 0.6, 0.5, 0.4, 0.3, 0.2, 0.1$



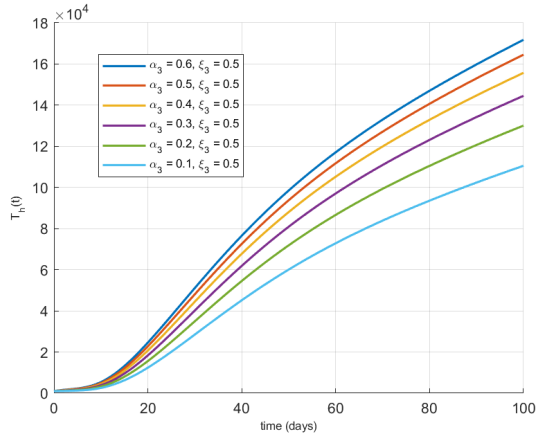
(b) $\xi_3 = 0.2$ and $\alpha_3 = 0.6, 0.5, 0.4, 0.3, 0.2, 0.1$



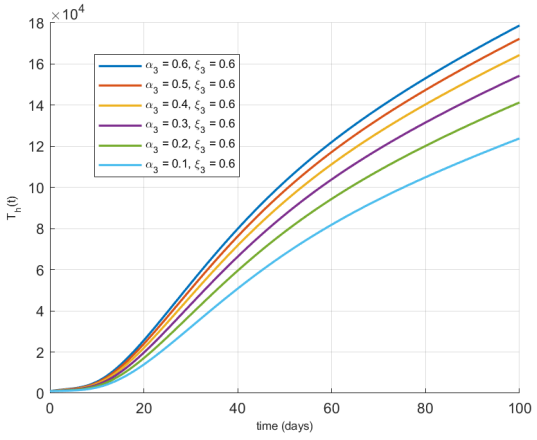
(c) $\xi_3 = 0.3$ and $\alpha_3 = 0.6, 0.5, 0.4, 0.3, 0.2, 0.1$



(d) $\xi_3 = 0.4$ and $\alpha_3 = 0.6, 0.5, 0.4, 0.3, 0.2, 0.1$



(e) $\xi_3 = 0.5$ and $\alpha_3 = 0.6, 0.5, 0.4, 0.3, 0.2, 0.1$



(f) $\xi_3 = 0.6$ and $\alpha_3 = 0.6, 0.5, 0.4, 0.3, 0.2, 0.1$

Figure 6: Comparison of $T_h(t)$ under different α_3 and ξ_3 configurations.

the spread of malaria, is more influenced by the average number n of mosquito bites with sensitivity index + 1, the recruitment rate of mosquitoes Λ_m with sensitivity index + 1/2, the recovery rate of symptomatic human γ_2 with sensitivity index -0.0453, the relapse rate ψ_2 with

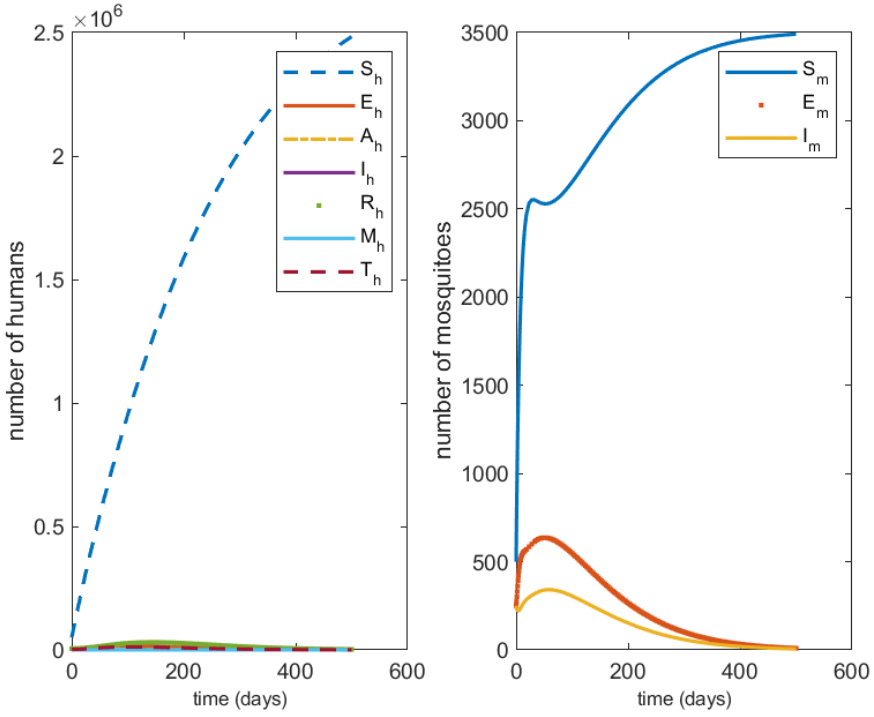


Figure 7: Disease-free trends of humans and mosquitoes for parameter values on Table 2, except $\gamma_1 = 0.5, n = 5$ and $\Lambda_m = 500$. We obtain $\mathcal{R}_0 = 0.81 < 1$ and the estimated disease-free point is (2500000,0,0,0,0,0,0,3500,0,0).

sensitivity index -0.0452 and the recovery rate of asymptomatic humans γ_1 with sensitivity index - 0.00244. Others parameters do not influence very significantly the basic reproduction number $\mathcal{R}_0$, see Figure 3. Using Table 2, we found that the basic reproduction number is greater than one ($\mathcal{R}_0 = 1.03 > 1$). This ensure the endemic situation of malaria as shown on Figure 4. Reducing the recruitment rate of mosquitoes at $\Lambda_m = 500$, the average number of mosquito bites at $n = 5$ and, increasing the recovery rate of asymptomatic humans at $\gamma_1 = 0.5$, we reach the disease-free equilibrium point of the model, as shown on Figure 7. So, malaria die out! The corresponding basic reproduction number is $\mathcal{R}_0 = 0.81 < 1$.

Figures 5 and 6 highlight the significant role of asymptomatic and resistant strains infected humans in the spread of malaria. When the recovery rate increases, the number of recovered individuals also increases, and if it decreases, the number of recovered individuals likewise decreases. In the past, this class of infected individuals was completely ignored. However, we have now contributed by highlighting the important role it plays in the transmission dynamics of malaria. Increasing the recovery rate γ_1 of asymptomatic humans helps to decrease the proportions α_3 and ξ_3 of resistant strains humans among symptomatic and asymptomatic respectively as shown on Figure 6. Therefore reduces the resistant strains infected humans in the population. All well-known malaria control strategies must be revised to take into account asymptomatic cases and those carrying resistant strains. The epidemiological implication and the resulting public health policy is that mass screening programs should be implemented to identify asymptomatic individuals and ensure their full recovery. Additionally, to avoid drug resistance and relapse of ignorant infected individuals, a control test should be organized after each malaria treatment.

Data availability

All data used to support our findings are included within the article.

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Conflicts of interests

The authors declare there is no conflicts of interests regarding the publication of this paper.

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