

Optimal control and cost-effectiveness analyses of a malaria model

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SUMMARY

In this work, a new mathematical model that incorporates vaccination, treatment and use of sterile-insect technology (SIT) to control the spread of malaria is formulated. The model is shown to be well-posed mathematically and epidemiologically. Optimal control analysis is performed on the model to obtain the number of susceptible humans that will be vaccinated, the number of infected humans that will be treated, and the number of SIT mosquitoes that will be interacting with females in the wild for optimal control of the disease. The results show that combining vaccination, treatment and use of SIT has a greater controlling effect than when any two of the three control methods are combined. However, cost-effectiveness analysis shows that combining treatment and vaccination is more cost-effective, and hence should be adopted.

Key words: malaria, optimal control, Pontryagin maximum principle, sterile-insect technique, vaccination

1. Introduction

Malaria is a deadly and infectious disease transmitted amongst humans through the bite of an infectious female *Anopheles* mosquito (Chitnis et al., 2006 and WHO, 2021). The disease remains highly endemic and a global threat in Asian and sub-Saharan African Countries. It is on record that in 2021, about 244 million persons were infected with malaria worldwide, with its attendant casualties over 619,000 (WHO, 2022). The most affected were the tropical regions of Africa, which accounted for 92% of cases and

93% of deaths, out of which Nigeria had the highest with 25% and Uganda the lowest with 4%. Similarly, in 2022, the number of malaria cases was recorded to be 249 million worldwide, with 608,000 malaria-related deaths (WHO, 2023). This unprecedented increase in malaria infection is traceable to an increase in the occurrence of drug-resistant malaria parasites and insecticide-resistant mosquitoes. This underscores the need for continuous research and study for new effective preventive and control mechanisms for malaria. Hence, we propose a different approach in this study for the control of malaria to overcome the aforementioned limitations. In this study, vaccination, use of a sterile insect technique, and treatment are proposed as control measures. Sterile-insect technology (SIT) is an innovative biological method for controlling insects and pests (Vreysen, 2007; Atokolo and Mbah, 2020). The use of this technology involves releasing a large number of sterile male mosquitoes into the wild to mate with their female counterparts (Dyck et al., 2021). The idea is that when mating occurs between sterile male and wild female mosquitoes, the eggs will not hatch, hence no offspring will be produced. To understand more about this technique, see Klassen (2009) and Anguelov et al. (2012).

There are many works on mathematical modelling of malaria and its control. Ayalew et al. (2024) proposed a mathematical model of malaria which shows that the use of mosquito-treated bednets, insecticides, treatment of infectious humans, and a continuous awareness campaign to protect humans optimally helps to control the spread of the disease. Mbugi et al. (2024) proposed a mathematical model for malaria with various immunity statuses. Robert et al. (2024) developed a mathematical model of malaria in children under five years as well as pregnant women under the influence of temperature. Haile et al. (2025) studied a malaria model considering relapse and humans who are unaware of being infectious. Naandam et al. (2025) worked on a malaria model with vaccination and showed how an increase in the rate of vaccination helps to reduce the endemicity of the disease. Wako et al. (2025) analyzed a malaria model with induced complications,

highlighting that increasing treatment and death rates as well as reducing the rate of transmission can reduce the burden of the disease. Al Basir et al. (2025) incorporated awareness campaigns alongside treatment as control measures to reduce the spread of malaria. Jaleta et al. (2025) analyzed a model for malaria which incorporated humans seeking treatment at health institutions and those under self-treatment. Menbiko et al. (2025) proposes a caputo fractional-order model of malaria with temperature variability. Adamu et al. (2025) developed a model for malaria considering the effect of a reduction in the mosquito population and natural recovery. All these works from the existing literature focus mainly on physical control measures for mosquitoes such as use of insecticides and spray. However, with the increasing rate of resistance by mosquitoes to spray as well as the resistance of some strains of the parasites to drugs, there is need for further study to develop alternative ways of controlling the disease. This informs the choice of the controls in this work.

This work is motivated by the works of Atokolo and Mbah (2020) and Duru and Mbah (2025). Atokolo and Mbah (2020) employed SIT to reduce the population of the *Aedes* mosquitoes responsible for zika virus disease, and discovered that the introduction of more sterile males into the wild will reduce the population of the mosquitoes with time; this forms part of our motivation in applying the same SIT in the control of *Anopheles* mosquitoes. Duru and Mbah (2025) proposed a malaria model where vaccination, treatment and use of a sterile insect technique are incorporated as control strategies. Their work mainly involved obtaining an approximate solution of the system by a homotopy analysis method. They did not perform numerical simulation of the system and optimal control analysis of the proposed control measures. In this present work, we perform optimal control analysis on the transmission model for malaria given by Duru and Mbah (2025) that incorporates treatment, vaccination and use of SIT to control the population of mosquitoes. The rest of this paper is arranged as follows. In section 2, the new model describing the disease dynamics in both

human and mosquito populations is introduced. In section 3, the model is shown to be well-posed epidemiologically and mathematically. The optimal control procedure is performed in section 4, and in section 5 we present the numerical simulations of the model. The work is concluded in section 6.

2. Model formulation

The mathematical model for malaria is made up of twelve (12) compartments comprising human and mosquito populations. The human population is divided into eight (8) classes; humans that are susceptible to malaria H , humans that are vaccinated H_v , humans that are not vaccinated H_u , humans that are exposed to malaria H_e , humans that are infectious H_i , humans that are infectious but undergoing treatment H_t , humans that are infectious but not undergoing treatment H_{un} , and infectious humans that have recovered H_r . On the other hand, in the mosquito population, we have four (4) compartments; mosquitoes that are susceptible M_s , mosquitoes that are exposed M_e , mosquitoes that are infectious M_i , and the sterile male mosquito population M_{st} . The proposed controls employed in the system are vaccination, treatment, and use of sterile-insect technology to control the vectors.

The following assumptions are made for the system:

- 1) Vaccination does not grant permanent immunity to malaria but wanes with time. Hence, the vaccinated human population becomes susceptible with time (Borginho, 2015).
- 2) The infectious human population is partitioned into two (2); those who accept treatment and those who refuse to go for treatment. Some of those who accept treatment recover from the disease, some die naturally, while others die due to the disease.
- 3) Some of those who refuse to go for treatment die from the disease, some die naturally, while others with time can suppress the symptoms and the multiplication of the parasites in their bodies due to their immune system (Med, 2022; Med. News, 2022).

Table 1. Description of state variables

Variables	Description
H	Susceptible humans
H_u	Humans who reject vaccination
H_v	Humans who accept vaccination
H_e	Exposed humans
H_i	Infectious humans
H_t	Infectious humans undergoing treatment
H_{un}	Infectious humans who reject treatment
H_r	Recovered humans
M_s	Susceptible mosquitoes
M_e	Exposed mosquitoes
M_i	Infectious mosquitoes
M_{st}	Sterile male mosquitoes

4) Those who refuse to go for treatment and have suppressed the symptoms and the multiplication of the parasites in their bodies return to the exposed compartment as the parasites are latent at this stage.

5) We assume that those who accept treatment are also infectious with the disease as they have not fully recovered.

The population of susceptible humans increases through births and the migration rate π_h and through the rate of loss of immunity to the disease after recovery, ϕ . It is reduced by the number of humans that are vaccinated and unvaccinated as well as those that die naturally at the rate μ_h . The population of humans that are exposed to malaria increases by the rate of exposure of susceptible humans to malaria. Infectious humans who refuse to go for treatment can experience a suppression of the disease in their system due to their immunity and return to the exposed class, since they may have a relapse and become infectious again. The population of humans that are infectious increases by the rate of development of infectiousness and decreases by natural death μ_h , the malaria-induced death rate μ_d , the rate at which infectious humans accept treatment ε_1 , and the rate at which infectious humans refuse treatment ε_2 . The population of mosquitoes that are susceptible increases by the level of recruitment denoted by π_m and de-

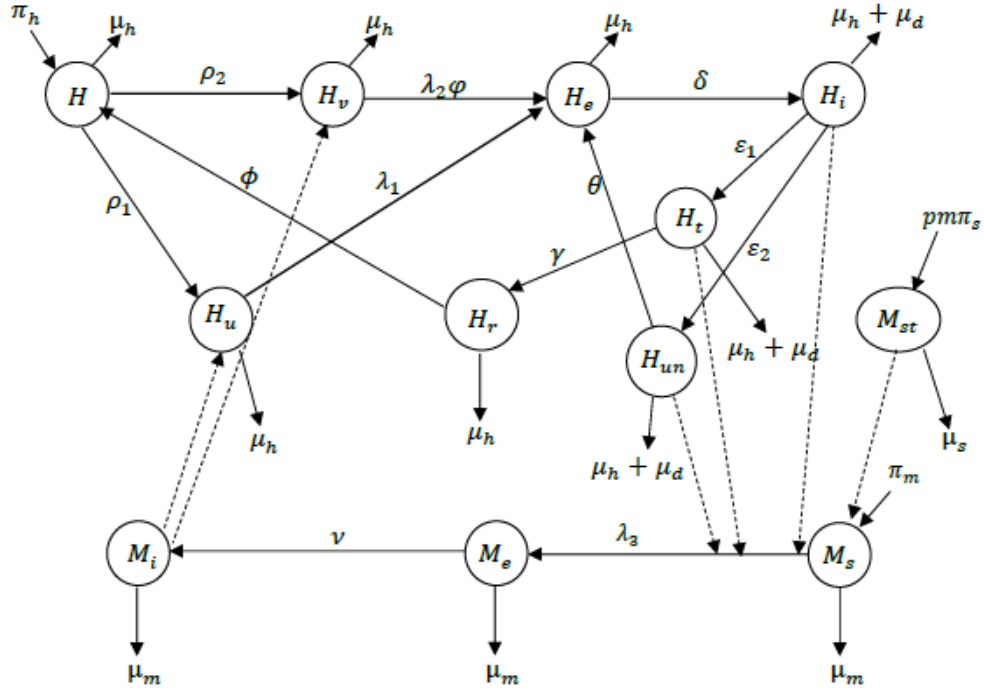


Figure 1. Flow diagram for disease transmission

creases by the natural death rate of mosquitoes μ_m , the rate of reduction λ_4 in the mosquito population due to SIT, as well as the rate of exposure of susceptible mosquitoes to infection. The population of mosquitoes that are exposed increases by the rate at which susceptible mosquitoes get infected and decreases by the natural death rate μ_m and the rate of development of infectiousness ν .

The SIT mosquitoes are recruited at a rate Λ_s with natural death rate μ_s . The parameter p represents the proportion of sterile males that will successfully join the wild mosquito population. Also, the parameter q accounts for mating competitiveness between SIT mosquitoes and the male Anopheles mosquitoes in the wild (Atokolo and Mbah, 2020). The relationships between the compartments in the system are shown in Figure 1.

Thus, the model for the system becomes

$$\begin{aligned}
\frac{dH}{dt} &= \pi_h + \phi H_r - (\rho_1 + \rho_2 + \mu_h)H, \\
\frac{dH_u}{dt} &= \rho_1 H - (\lambda_1 + \mu_h)H_u, \\
\frac{dH_v}{dt} &= \rho_2 H - (\lambda_2 \varphi + \mu_h)H_v, \\
\frac{dH_e}{dt} &= \lambda_1 H_u + \lambda_2 \varphi H_v + \theta H_{un} - (\delta + \mu_h)H_e, \\
\frac{dH_i}{dt} &= \delta H_e - (\mu_h + \mu_d + \varepsilon_1 + \varepsilon_2)H_i, \\
\frac{dH_t}{dt} &= \varepsilon_1 H_i - (\gamma + \mu_h + \mu_d)H_t, \\
\frac{dH_{un}}{dt} &= \varepsilon_2 H_i - (\theta + \mu_h + \mu_d)H_{un}, \\
\frac{dH_r}{dt} &= \gamma H_t - (\mu_h + \phi)H_r, \\
\frac{dM_s}{dt} &= \pi_m - (\lambda_3 + \lambda_4 + \mu_m)M_s, \\
\frac{dM_e}{dt} &= \lambda_3 M_s - (\nu + \mu_m)M_e, \\
\frac{dM_i}{dt} &= \nu M_e - \mu_m M_i, \\
\frac{dM_{st}}{dt} &= pq\pi_s - \mu_s M_{st},
\end{aligned} \tag{1}$$

where $\lambda_1 = \alpha_1 \beta_1 M_i$, $\lambda_2 = \alpha_2 \beta_2 M_i$, $\lambda_3 = \alpha_1 (\beta_3 H_i + \beta_4 H_t + \beta_5 H_{un})$ are the forces of infection in human and mosquito populations and $\lambda_4 = \kappa q M_{st}$. The initial conditions of the system at $t = 0$ are $H(0) = H^0$, $H_u(0) = H_u^0$, $H_v(0) = H_v^0$, $H_e(0) = H_e^0$, $H_i(0) = H_i^0$, $H_t(0) = H_t^0$, $H_{un}(0) = H_{un}^0$, $H_r(0) = H_r^0$, $M_s(0) = M_s^0$, $M_e(0) = M_e^0$, $M_i(0) = M_i^0$, $M_{st}(0) = M_{st}^0$. The solution to the system exists in the region $\Omega = \Omega_1 \cup \Omega_2 \cup \Omega_3 \subseteq R_+^8 \times R_+^3 \times R_+$ described respectively by

$$\Omega_1 = \left\{ (H, H_u, H_v, H_e, H_i, H_t, H_{un}, H_r) \in \mathcal{R}_+^8 : N_h \leq \frac{\pi_h}{\mu_h} \right\},$$

$$\Omega_2 = \left\{ (M_s, M_e, M_i) \in \mathcal{R}_+^3 : N_m \leq \frac{\pi_m}{\mu_m} \right\} \quad \text{and}$$

$$\Omega_3 = \left\{ M_{st} \in \mathcal{R}_+ : M_{st} \leq \frac{pm\pi_s}{\mu_s} \right\}.$$

3. Model analysis

3.1. Positivity of solutions and invariant region

Lemma 1: Let the initial data set for the model be $(H^0 > 0, H_u^0 > 0, H_v^0 > 0, H_e^0 > 0, H_i^0 > 0, H_t^0 > 0, H_{un}^0 > 0, H_r^0 > 0, M_s^0 > 0, M_e^0 > 0, M_i^0 > 0, M_{st}^0 > 0)$ at $t = 0$. Then the solution set $(H(t), H_u(t), H_v(t), H_e(t), H_i(t), H_t(t), H_{un}(t), H_r(t), M_s(t), M_e(t), M_i(t), M_{st}(t))$ of the model with the given initial data will remain positive for all time $t > 0$.

Proof. It can be shown from (1) that

$$\begin{aligned} \frac{dH}{dt} &\geq -(\rho_1 + \rho_2 + \mu_h)H, \\ \frac{dH_u}{dt} &\geq -(\lambda_1 + \mu_h)H_u, \\ &\vdots \\ \frac{dM_{st}}{dt} &\geq -\mu_s M_{st}. \end{aligned} \tag{2}$$

The solutions to (2) with the given initial data give

$$\begin{aligned} H &\geq H^0 e^{-\int (\rho_1 + \rho_2 + \mu_h) dt} > 0, \\ H_u &\geq H_u^0 e^{-\int (\lambda_1 + \mu_h) dt} > 0, \\ &\vdots \\ M_{st} &\geq M_{st}^0 e^{-\int \mu_s dt} > 0. \end{aligned}$$

Thus, the solution to the model system remains positive $\forall t > 0$.

The total human and mosquito populations are given by

$$N_h = H + H_u + H_v + H_e + H_i + H_t + H_{un} + H_r$$

and $N_m = M_s + M_e + M_i$, respectively.

The total human and mosquito populations satisfy the differential equations

$$\begin{aligned} \frac{dN_h}{dt} &= \pi_h - \mu_h N_h - \mu_d(H_i + H_t + H_{un}), \\ \frac{dN_m}{dt} &= \pi_m - \mu_m N_m, \end{aligned}$$

respectively. As $t \rightarrow \infty$, both differential equations give the following solutions on integration: $0 \leq N_h \leq \frac{\pi_h}{\mu_h}$ and $0 \leq N_m \leq \frac{\pi_m}{\mu_m}$. Similarly, the sterile male population given by $\frac{dM_{st}}{dt} = pm\pi_s - \mu_s M_{st}$ gives $0 \leq M_{st} \leq \frac{pm\pi_s}{\mu_s}$ on integration. This shows that all the solutions of the system are positive and bounded. Hence, the region Ω is positively invariant and is an attractor of all positive solutions to the system. Thus, (1) is epidemiologically well-posed since all the state variables remain non-negative for all $t > 0$. This is a sufficient condition to study and analyze the system (1).

3.2. Existence and uniqueness of solutions

To show the existence and uniqueness of solutions to the malaria model, the Banach fixed point theorem, which relates to a special kind of Lipschitz continuity, is employed. Hence, we first state both concepts before applying them.

Lemma 2. (*Lipschitz continuity*): A function $f(t, z)$ is said to be Lipschitz continuous in z if there exists a constant $K \geq 0$ such that

$$|f(t, z_1) - f(t, z_2)| \leq K|z_1 - z_2|.$$

Here, K is called a Lipschitz constant and f is said to be K -Lipschitz $\forall z_1, z_2 \in \Omega$.

Theorem 1: (*Banach fixed point theorem*): Let Ω be a complete metric space and let $f : \Omega \rightarrow \Omega$ be a contraction, that is, $\exists c \in (0, 1)$ such that for all $z_1, z_2 \in \Omega$; then

$$d(f(z_1), f(z_2)) \leq cd(z_1, z_2).$$

Then, f has a unique fixed point in Ω . That is, there exists a unique $z^* \in \Omega$ such that

$$f(z^*) = z^*.$$

Proof. The system (1) can be expressed as

$$X'(t) = f(X(t)), \quad X(t_0) = X_0,$$

where $X(t) = (H, H_u, H_v, H_e, H_i, H_t, H_{un}, H_r, M_s, M_e, M_i, M_{st})$

$$\text{and } f(X(t)) = \begin{bmatrix} \pi_h + \phi H_r - (\rho_1 + \rho_2 + \mu_h)H \\ \rho_1 H - (\lambda_1 + \mu_h)H_u \\ \rho_2 H - (\lambda_2 \varphi + \mu_h)H_v \\ \lambda_1 H_u + \lambda_2 \varphi H_v + \theta H_{un} - (\delta + \mu_h)H_e \\ \delta H_e - (\mu_h + \mu_d + \varepsilon_1 + \varepsilon_2)H_i \\ \varepsilon_1 H_i - (\gamma + \mu_h + \mu_d)H_t \\ \varepsilon_2 H_i - (\theta + \mu_h + \mu_d)H_{un} \\ \gamma H_t - (\mu_h + \phi)H_r \\ \pi_m - (\lambda_3 + \lambda_4 + \mu_m)M_s \\ \lambda_3 M_s - (\nu + \mu_m)M_e \\ \nu M_e - \mu_m M_i \\ pm\pi_s - \mu_s M_{st} \end{bmatrix}.$$

Let $\|\cdot\|$ be the maximum norm in Ω , taken to be the Banach domain for

continuous functions where

$$\|X_i(t)\| = \sum_{i=1}^{12} \|X_i\|_{\infty}.$$

Let $\|H\| \leq k_1, \|H_u\| \leq k_2, \|H_v\| \leq k_3, \|H_e\| \leq k_4, \|H_i\| \leq k_5, \|H_t\| \leq k_6, \|H_{un}\| \leq k_7, \|H_r\| \leq k_8, \|M_s\| \leq k_9, \|M_e\| \leq k_{10}, \|M_i\| \leq k_{11}, \|M_{st}\| \leq k_{12}$ and $0 \leq m_i < 1$. From equation (1), we will have that for any H_1 and $H_2 \in \Omega$,

$$\begin{aligned} \|f(t, H_1) - f(t, H_2)\| &= \|(\pi_h + \phi H_r - (\rho_1 + \rho_2 + \mu_h)H_1) \\ &\quad - (\pi_h + \phi H_r - (\rho_1 + \rho_2 + \mu_h)H_2)\| = \|(\rho_1 + \rho_2 + \mu_h)(H_1 - H_2)\| \\ &\leq (\rho_1 + \rho_2 + \tau_1)\|H_1 - H_2\| \leq m_1\|H_1 - H_2\|. \end{aligned}$$

Lipschitz continuity in H is established with m_1 as the Lipschitz constant. Similarly, we can establish Lipschitz continuity in other state variables as follows;

$$\begin{aligned} \|f(t, H_{u1}) - f(t, H_{u2})\| &\leq m_2\|H_{u1} - H_{u2}\|, \\ \|f(t, H_{v1}) - f(t, H_{v2})\| &\leq m_3\|H_{v1} - H_{v2}\|, \\ \|f(t, H_{e1}) - f(t, H_{e2})\| &\leq m_4\|H_{e1} - H_{e2}\|, \\ \|f(t, H_{i1}) - f(t, H_{i2})\| &\leq m_5\|H_{i1} - H_{i2}\|, \\ \|f(t, H_{t1}) - f(t, H_{t2})\| &\leq m_6\|H_{t1} - H_{t2}\|, \\ \|f(t, H_{un1}) - f(t, H_{un2})\| &\leq m_7\|H_{un1} - H_{un2}\|, \\ \|f(t, H_{r1}) - f(t, H_{r2})\| &\leq m_8\|H_{r1} - H_{r2}\|, \\ \|f(t, M_{s1}) - f(t, M_{s2})\| &\leq m_9\|M_{s1} - M_{s2}\|, \\ \|f(t, M_{e1}) - f(t, M_{e2})\| &\leq m_{10}\|M_{e1} - M_{e2}\|, \\ \|f(t, M_{i1}) - f(t, M_{i2})\| &\leq m_{11}\|M_{i1} - M_{i2}\|, \\ \|f(t, M_{st1}) - f(t, M_{st2})\| &\leq m_{12}\|M_{st1} - M_{st2}\| \end{aligned}$$

where $m_1 = \rho_1 + \rho_2 + \mu_h$, $m_2 = \alpha_1\beta_1k_{11} + \mu_h$, $m_3 = \alpha_1\beta_1\varphi k_{11} + \mu_h$,

$m_4 = \delta + \mu_h$, $m_5 = \mu_h + \mu_d + \varepsilon_1 + \varepsilon_2$, $m_6 = \gamma + \mu_h + \mu_d$, $m_7 = \theta + \mu_h + \mu_d$, $m_8 = \mu_h + \phi$, $m_9 = \alpha_1(\beta_3 k_5 + \beta_4 k_6 + \beta_5 k_7 + \kappa q k_{12} + \mu_m)$, $m_{10} = \nu + \mu_m$, $m_{11} = \mu_m$, $m_{12} = \mu_s$. Thus, Lipschitz continuity in the state variables has been established and by the Banach fixed point theorem, the solution to the system (1) exists and is unique.

4. Optimal control of malaria

In this section, we seek to obtain the proportion of susceptible humans that will be vaccinated, infectious humans that will be treated and SIT mosquitoes that will be interacting with wild mosquitoes for optimal control of malaria given the initial data under consideration. In this case, it is then assumed that the rate of vaccination ρ_2 , the rate of treatment ε_1 and the number of SIT mosquitoes interacting with females in the wild M_{st} are not constant but a function of time; $\rho_2 = \rho_2(t)$, $\varepsilon_1 = \varepsilon_1(t)$ and $M_{st} = M_{st}(t)$ respectively. Therefore, an optimal control problem with objective function given by

$$J(x; \rho_2, \varepsilon_1, M_{st}) = \int_0^T (A_1 H(t) + A_2 H_e(t) + A_3 H_i(t) + A_4 M_s(t) + \frac{A_5}{2} \rho_2^2(t) + \frac{A_6}{2} \varepsilon_1^2(t) + \frac{A_7}{2} M_{st}^2(t)) dt. \quad (3)$$

where the constants $A_i, i = 1, 2, 3, 4, 5, 6, 7$ are positive weights which help to balance each term in the integrand, is considered. The quadratic terms, $\frac{A_5}{2} \rho_2^2(t)$, $\frac{A_6}{2} \varepsilon_1^2(t)$ and $\frac{A_7}{2} M_{st}^2(t)$ represent the costs involved in vaccination, treatment and interaction of SIT mosquitoes with those in the wild at any time t . The control parameters in (3), $\rho_2^*(t)$, $\varepsilon_1^*(t)$ and $M_{st}^*(t)$ are assumed to be Lebesgue measurable with $a < \rho_2^*(t) \leq b, \forall t \in [0, t_f]$, $a < \varepsilon_1^*(t) \leq b, \forall t \in [0, t_f]$ and $a < M_{st}^*(t) \leq b, \forall t \in [0, t_f]$. Therefore, we seek an optimal control, $\rho_2^*(t)$, $\varepsilon_1^*(t)$ and $M_{st}^*(t)$ such that

$$J(\rho_2^*(t)) = \min\{J(\rho_2(t)) : \rho_2(t) \in \Omega\},$$

$$\begin{aligned}
J(\varepsilon_1^*(t)) &= \min\{J(\varepsilon_1(t)) : \varepsilon_1(t) \in \Omega\}, \\
J(M_{st}^*(t)) &= \min\{J(M_{st}(t)) : M_{st}(t) \in \Omega\},
\end{aligned}$$

where the control set Ω is given by

$$\begin{aligned}
\Omega_1 &= \{\rho_2(t) : \rho_2(t) \text{ is piecewise continuous on } [0, T], a \leq \rho_2(t) \leq b\}, \\
\Omega_2 &= \{\varepsilon_1(t) : \varepsilon_1(t) \text{ is piecewise continuous on } [0, T], a \leq \varepsilon_1(t) \leq b\}, \quad (4) \\
\Omega_3 &= \{M_{st}(t) : M_{st}(t) \text{ is piecewise continuous on } [0, T], a \leq M_{st}(t) \leq b\},
\end{aligned}$$

where $\Omega = \Omega_1 \cup \Omega_2 \cup \Omega_3$, a and b being nonnegative constants, with $0 < a \leq 1$ and $0 < b \leq 1$ for the first two equations in (4) and with $0 < b \leq 5000$ for the last equation.

4.1. Existence of optimal controls

Theorem 2: Let the objective functional J in (3) be defined on the control set Ω , with the system (1) as the constrained control, then there exist optimal controls $(\rho_2^*(t), \varepsilon_1^*(t), M_{st}^*(t))$ such that $J(\rho_2^*, \varepsilon_1^*, M_{st}^*) = \min\{J(\rho_2, \varepsilon_1, M_{st}) : \rho_2, \varepsilon_1, M_{st} \in \Omega\}$ provided that the following properties hold:

- (a) The admissible control set Ω is closed and convex.
- (b) The state variables of the system are bounded by a linear function in the state and control variables.
- (c) The integrand of the objective functional is convex with respect to the control.
- (d) There exist constants $m_1, m_2 > 0$ and $m_3 > 1$ such that the integrand is bounded below by $m_1 (\|u_i\|^2)^{\frac{m_3}{2}} - m_2$.

Proof.

- (a) By definition, the control set (4) is closed. Also, for every $\bar{x}, \bar{y} \in \Omega$ and $\lambda \in (0, 1)$, $\lambda\bar{x} + (1 - \lambda)\bar{y} \in \Omega$ where $\bar{x} = (\rho_{12}^*, \varepsilon_{11}^*, M_{st1}^*)$ and $\bar{y} = (\rho_{22}^*, \varepsilon_{12}^*, M_{st2}^*)$, showing that the control set is convex.
- (b) The associated optimal solutions, $(H_h^*, H_u^*, H_v^*, H_e^*, H_i^*, H_t^*, H_{un}^*, H_r^*, M_s^*, M_e^*, M_i^*, M_{st}^*)$ are bounded as proved earlier in section 3.2, where

the state system satisfies the Lipschitz property with respect to the state variables.

(c) It remains to show the convexity of the integrand,

$L(t, H, H_e, H_i, M_s, \rho_2, \varepsilon_1, M_{st}) = A_1 H(t) + A_2 H_e(t) + A_3 H_i(t) + A_4 M_s(t) + \frac{A_5}{2} \rho_2^2(t) + \frac{A_6}{2} \varepsilon_1^2(t) + \frac{A_7}{2} M_{st}^2(t)$, with respect to the control parameters, $\rho_2, \varepsilon_1, M_{st}$ in the set Ω . Therefore, we need to show that

$$\begin{aligned} &L(t, H, H_e, H_i, M_s, \lambda \rho_2 + (1 - \lambda) \rho'_2, \lambda \varepsilon_1 + (1 - \lambda) \varepsilon'_1, \lambda M_{st} + (1 - \lambda) M'_{st}) \\ &\leq \lambda L(t, H, H_e, H_i, M_s, \rho_2, \varepsilon_1, M_{st}) + (1 - \lambda) L(t, H, H_e, H_i, M_s, \rho'_2, \varepsilon'_1, M'_{st}), \end{aligned}$$

for $\lambda \in (0, 1)$. Note that

$$\begin{aligned} &L(t, H, H_e, H_i, M_s, \lambda \rho_2 + (1 - \lambda) \rho'_2, \lambda \varepsilon_1 + (1 - \lambda) \varepsilon'_1, \lambda M_{st} + (1 - \lambda) M'_{st}) \\ &= A_1 H(t) + A_2 H_e(t) + A_3 H_i(t) + A_4 M_s(t) + \frac{A_5}{2} (\lambda \rho_2 + (1 - \lambda) \rho'_2)^2 \\ &\quad + \frac{A_6}{2} (\lambda \varepsilon_1 + (1 - \lambda) \varepsilon'_1)^2 + \frac{A_7}{2} (\lambda M_{st} + (1 - \lambda) M'_{st})^2 \end{aligned} \tag{5}$$

and

$$\begin{aligned} &\lambda L(t, H, H_e, H_i, M_s, \rho_2, \varepsilon_1, M_{st}) + (1 - \lambda) L(t, H, H_e, H_i, M_s, \rho'_2, \varepsilon'_1, M'_{st}) \\ &= A_1 H(t) + A_2 H_e(t) + A_3 H_i(t) + A_4 M_s(t) + \frac{A_5}{2} \lambda \rho_2^2 + \frac{A_5}{2} (1 - \lambda) \rho'^2_2 \\ &\quad + \frac{A_6}{2} \lambda \varepsilon_1^2 + \frac{A_6}{2} (1 - \lambda) \varepsilon'^2_1 + \frac{A_7}{2} \lambda M_{st}^2 + \frac{A_7}{2} (1 - \lambda) M'^2_{st}. \end{aligned} \tag{6}$$

This means that from (5) and (6), we must prove that $(\lambda \rho_2 + (1 - \lambda) \rho'_2)^2 \leq \lambda \rho_2^2 + (1 - \lambda) \rho'^2_2$, $(\lambda \varepsilon_1 + (1 - \lambda) \varepsilon'_1)^2 \leq \lambda \varepsilon_1^2 + (1 - \lambda) \varepsilon'^2_1$, and $(\lambda M_{st} + (1 - \lambda) M'_{st})^2 \leq \lambda M_{st}^2 + (1 - \lambda) M'^2_{st}$.

Expanding $(\lambda \rho_2 + (1 - \lambda) \rho'_2)^2$ gives

$$(\lambda \rho_2 + (1 - \lambda) \rho'_2)^2 = \lambda^2 \rho_2^2 + 2\lambda(1 - \lambda) \rho_2 \rho'_2 + (1 - \lambda)^2 \rho'^2_2,$$

$$\begin{aligned}
&= \lambda(\lambda - 1 + 1)\rho_2^2 + 2\lambda(1 - \lambda)\rho_2\rho_2' + (1 - \lambda)(1 - \lambda)\rho_2'^2, \\
&= \lambda\rho_2^2 + (1 - \lambda)\rho_2'^2 - \lambda(1 - \lambda) [\rho_2^2 - 2\rho_2\rho_2' + \rho_2'^2], \\
&= \lambda\rho_2^2 + (1 - \lambda)\rho_2'^2 - \lambda(1 - \lambda) [\rho_2 - \rho_2']^2, \\
&\leq \lambda\rho_2^2 + (1 - \lambda)\rho_2'^2.
\end{aligned}$$

Similarly, $(\lambda\varepsilon_1 + (1 - \lambda)\varepsilon_1')^2$ and $(\lambda M_{st} + (1 - \lambda)M_{st}')^2$ can be respectively obtained. This proves the convexity of the integrand on the singleton, Ω .

- (d) Given the Lagrangian $L(t, H, H_e, H_i, M_s, \rho_2, \varepsilon_1, M_{st}) = A_1 H(t) + A_2 H_e(t) + A_3 H_i(t) + A_4 M_s(t) + \frac{A_5}{2} \rho_2^2(t) + \frac{A_6}{2} \varepsilon_1^2(t) + \frac{A_7}{2} M_{st}^2(t)$, we can see that

$$\begin{aligned}
L(t, H, H_e, H_i, M_s, \rho_2, \varepsilon_1, M_{st}) &\geq \frac{A_5}{2} \rho_2^2(t) + \frac{A_6}{2} \varepsilon_1^2(t) + \frac{A_7}{2} M_{st}^2(t), \\
&\geq m_1 \left(|\rho_2|^2 + |\varepsilon_1|^2 + |M_{st}|^2 \right)^{\frac{m_3}{2}} + m_2,
\end{aligned}$$

where $m_1 = \frac{1}{2} \min\{A_5, A_6, A_7\}$, $m_2 > 0$ and $m_3 = 2$.

By Fleming and Rishel [10], the existence of the optimal controls is established.

4.2. Characterization of the optimal controls

Theorem 3: (*Pontryagin Maximum Principle*): Suppose u^* and x^* are optimal for the control problem

$$\min \int_{t_0}^{t_f} f(x(t), u(t)) dt \quad \text{subject to} \quad \frac{dx(t)}{dt} = g(x(t), u(t)), \quad x(t_0) = x_0.$$

then there exists a piecewise differentiable function $\vartheta(t)$ called the adjoint variable or costate such that the Hamiltonian defined by

$$H(t, x, u, \vartheta) = f(t, x(t), u(t)) + \vartheta g(t, x(t), u(t)), \tag{7}$$

satisfies

$$\begin{aligned}
H(t, x, u, \vartheta) &\leq H(t, x^*, u^*, \vartheta), \\
\frac{dH(t, x, u, \vartheta)}{du} &= 0, \\
\frac{d\vartheta(t)}{dt} &= -\frac{dH(t, x^*, u^*, \vartheta)}{dx}, \\
\frac{dx(t)}{dt} &= \frac{dH(t, x^*, u^*, \vartheta)}{d\vartheta},
\end{aligned} \tag{8}$$

for all controls u at each time t . The adjoint variable ϑ satisfies the transversality condition $\vartheta(t_f) = 0$.

Proof. In Theorem 3, $x \in \{H_h, H_u, H_v, H_e, H_i, H_t, H_{un}, H_r, M_s, M_e, M_i, M_{st}\}$, $u \in \{\rho_2, \varepsilon_1, M_{st}\}$, $f(t, x(t), u(t))$ is the objective functional and $g(t, x(t), u(t))$ is the right-hand side of the system (1).

From (7), the Hamiltonian will be given by

$$\begin{aligned}
H = & A_1 H(t) + A_2 H_e(t) + A_3 H_i(t) + A_4 M_s(t) + \frac{A_5}{2} \rho_2^2(t) + \frac{A_6}{2} \varepsilon_1^2(t) \\
& + \frac{A_7}{2} M_{st}^2(t) + \vartheta_1(t)(\pi_h + \phi H_r(t) - (\rho_1 + \rho_2 + \mu_h)H(t)) + \vartheta_2(t)(\rho_1 H(t) \\
& - (\lambda_1 + \mu_h)H_u(t)) + \vartheta_3(t)(\rho_2 H(t) - (\lambda_2 \varphi + \mu_h)H_v(t)) + \vartheta_4(t)(\lambda_1 H_u(t) \\
& + \lambda_2 \varphi H_v(t) + \theta H_{un}(t) - (\delta + \mu_h)H_e(t)) + \vartheta_5(t)(\delta H_e(t) \\
& - (\mu_h + \mu_d + \varepsilon_1 + \varepsilon_2)H_i(t)) + \vartheta_6(t)(\varepsilon_1 H_i(t) - (\gamma + \mu_h + \mu_d)H_t(t)) \\
& + \vartheta_7(t)(\varepsilon_2 H_i(t) - (\theta + \mu_h + \mu_d)H_{un}(t)) + \vartheta_8(t)(\gamma H_t(t) - (\mu_h + \phi)H_r(t)) \\
& + \vartheta_9(t)(\pi_m - (\lambda_3 + \lambda_4 + \mu_m)M_s(t)) + \vartheta_{10}(t)(\pi_3 M_s(t) - (\nu + \mu_m)M_e(t)) \\
& + \vartheta_{11}(t)(\nu M_e(t) - \mu_m M_i(t)) + \vartheta_{12}(t)(pm\pi_s - \mu_s M_{st}(t)),
\end{aligned}$$

and using (8), the adjoint variables $\vartheta_i(t)$, $i = 1, 2, \dots, 12$, satisfy the system of ordinary differential equations

$$\begin{aligned}
\frac{d\vartheta_1}{dt} &= (\rho_1 + \rho_2 + \mu_h)\vartheta_1 - \rho_1\vartheta_2 - \rho_2\vartheta_3 - A_1, \\
\frac{d\vartheta_2}{dt} &= \lambda_1(\vartheta_2 - \vartheta_4) + \mu_h\vartheta_2,
\end{aligned}$$

$$\begin{aligned}
\frac{d\vartheta_3}{dt} &= \lambda_2\varphi(\vartheta_3 - \vartheta_4) + \mu_h\vartheta_3, \\
\frac{d\vartheta_4}{dt} &= (\delta + \mu_h)\vartheta_4 - \delta\vartheta_5 - A_2, \\
\frac{d\vartheta_5}{dt} &= (\mu_h + \mu_d + \varepsilon_1 + \varepsilon_2)\vartheta_5 + \alpha_1\beta_3M_s(\vartheta_9 - \vartheta_{10}) - \varepsilon_1\vartheta_6 - \varepsilon_2\vartheta_7 - A_3, \\
\frac{d\vartheta_6}{dt} &= (\gamma + \mu_h + \mu_d)\vartheta_6 + \alpha_1\beta_4M_s(\vartheta_9 - \vartheta_{10}) - \gamma\vartheta_8, \\
\frac{d\vartheta_7}{dt} &= (\theta + \mu_h + \mu_d)\vartheta_7 + \alpha_1\beta_5M_s(\vartheta_9 - \vartheta_{10}) - \theta\vartheta_4, \\
\frac{d\vartheta_8}{dt} &= (\mu_h + \phi)\vartheta_8 - \phi\vartheta_1, \\
\frac{d\vartheta_9}{dt} &= \lambda_3(\vartheta_9 - \vartheta_{10}) + (\lambda_4 + \mu_m)\vartheta_9 - A_4, \\
\frac{d\vartheta_{10}}{dt} &= (\nu + \mu_m)\vartheta_{10} - \nu\vartheta_{11}, \\
\frac{d\vartheta_{11}}{dt} &= \mu_m\vartheta_{11} + \alpha_1\beta_1H_u(\vartheta_2 - \vartheta_4) + \alpha_1\beta_2\varphi H_v(\vartheta_3 - \vartheta_4), \\
\frac{d\vartheta_{12}}{dt} &= \vartheta_{10}kqM_s + \vartheta_{12}\mu_s
\end{aligned}$$

with the transversality condition $\vartheta_i(T) = 0, i = 1, 2, \dots, 11$. Using the optimality condition, we obtain that $\rho_2^*(t) = \frac{(\vartheta_1(t) - \vartheta_3(t))H(t)}{A_5}$, $M_{st}^*(t) = \frac{\vartheta_9(t)\kappa_1M_s(t)}{A_7}$ and $\varepsilon_1^*(t) = \frac{(\vartheta_5(t) - \vartheta_6(t))H_i(t)}{A_6}$, which can be characterized as

$$\begin{aligned}
\rho_2^*(t) &= \max \left(a, \min \left(b, \frac{(\vartheta_1(t) - \vartheta_3(t))H(t)}{A_5} \right) \right), \\
\varepsilon_1^*(t) &= \max \left(a, \min \left(b, \frac{(\vartheta_5(t) - \vartheta_6(t))H_i(t)}{A_6} \right) \right), \\
M_{st}^*(t) &= \max \left(a, \min \left(b, \frac{\vartheta_9(t)\kappa_1M_s(t)}{A_7} \right) \right).
\end{aligned} \tag{9}$$

Hence, (9) gives the number of susceptible humans that will be vaccinated, the number of infectious humans that will be treated and the number of SIT mosquitoes that will be interacting with wild female mosquitoes for optimal control of malaria.

5. Numerical simulations

The forward-backward sweep technique was used to obtain a numerical solution to the optimal control problem. The procedure involves solving the optimal state equation using the forward Runge–Kutta order 4 method with an initial value for the optimal control parameter and then solving the adjoint system of equations with the backward Runge–Kutta order 4 method, starting with the transversality conditions $\vartheta_i(T) = 0$. The optimal control is updated with the current state and adjoint solutions at the end of each iteration. The iteration is repeated until the criterion for convergence is achieved. The initial values used for the state variables are $H = 500, H_u = 362, H_v = 9, H_e = 48, H_i = 35, H_t = 21, H_{un} = 8, H_r = 10, M_s = 500, M_e = 320, M_i = 170$. We also used $A_1 = 1000, A_2 = 100, A_3 = 10, A_4 = 10, A_5 = 1000, A_6 = 10000, A_7 = 1$. For recruitment of SIT mosquitoes, $b = 5000$. The parameter values used in the numerical experiment were assumed from the disease dynamics as referenced from other works and shown in Table 1. Also, in the Figures, $u_1 \equiv \rho_2$, $u_2 \equiv \varepsilon_1$ and $u_3 \equiv M_{st}$.

5.1. Strategy one: $\rho_2 \neq 0, \varepsilon_1 \neq 0$ and $M_{st} = 0$

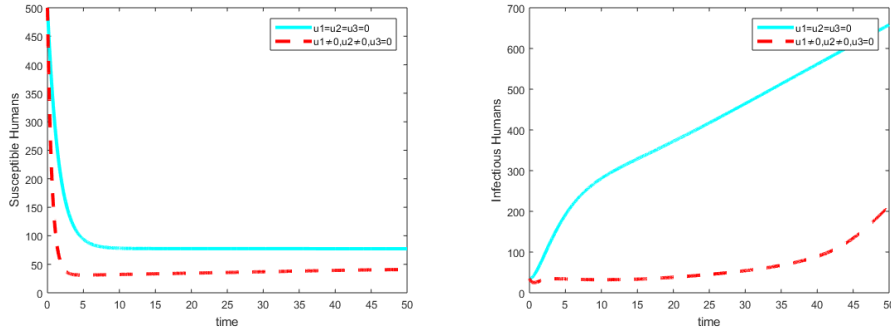
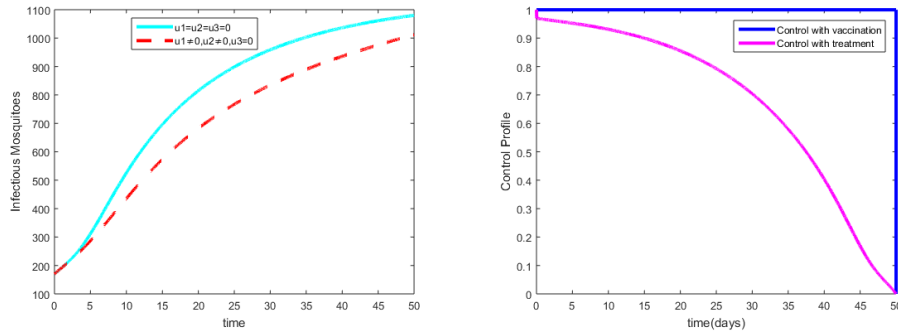
In this case, we consider the situation where only vaccination, ρ_2 and treatment, ε_1 are employed as control measures, in the absence of SIT mosquitoes, M_{st} , in the optimal control analysis. The results of this simulation are shown in Figures (2–3). Figure (2) describes the effects of vaccination and treatment on susceptible and infectious human compartments. Both controls successfully reduce the population of humans in these classes. Vaccination of humans against malaria and treatment of infectious humans were seen to reduce the rate of susceptible humans getting infected as more humans are protected from malaria. The reduction of the susceptible human population causes a corresponding decrease in the population of infectious humans as shown in Figure (2i).

Table 2. Description of parameters

Parameter	Description	Value	Source
π_h	Rate of recruitment of humans	50	Assumed
π_m	Rate of recruitment of <i>Anopheles</i> mosquitoes	100	Assumed
π_s	Rate of recruitment of sterile mosquitoes	100	Assumed
φ	Rate at which vaccinated humans lose immunity	0.0125	Assumed
α_1	Contact rate of mosquitoes with humans	0.4	Agusto et al. (2015)
β_1	Probability of transmission from M_i to H_u	0.034	Day et al. (2011)
β_2	Probability of transmission from M_i to H_v	0.013	Assumed
β_3	Probability of transmission from H_i to M_s	0.0044	Agusto et al. (2015)
β_4	Probability of transmission from H_t to M_s	0.0022	Assumed
β_5	Probability of transmission from H_{un} to M_s	0.0044	Agusto et al. (2015)
δ	Rate of development of infectiousness in humans	0.0833	CDC (2021)
μ_h	Natural death rate of humans	0.00004	Okosun and Makinde (2014)
μ_d	Disease-induced death rate in humans	0.00032338	Agusto et al. (2015)
μ_m/μ_s	Natural death rate of mosquitoes	0.0556	Duru et al. (2024)
ρ_1	Rate at which susceptible humans reject vaccination	0.65	Duru et al. (2024)
ρ_2	Rate at which susceptible humans accept vaccination	0.28	Duru et al. (2024)
ϕ	Rate of loss of immunity to malaria from recovered class	0.0146	Duru and Mbah (2025)
ε_1	Rate at which infectious humans accept treatment	0.62	Duru et al. (2025)
ε_2	Rate at which infectious humans refuse to be treated	0.21	Duru et al. (2025)
θ	Level of movement from H_{un} to H_e	0.13	Duru and Mbah (2025)

Table 2. Description of parameters (continued)

Parameter	Description	Value	Source
γ	Rate of recovery of infectious humans undergoing treatment	0.25	Okosun and Makinde (2014)
ν	Rate of development of infectiousness in M_s	0.1	CDC (2021)
κ	Mating rate of SIT mosquitoes with <i>Anopheles</i> population	0.5	Assumed
q	Mating competitiveness of males in the wild and sterile mosquitoes	0.5	Assumed
p	Proportion of M_{st} that successfully join the wild mosquitoes	300	Assumed

**Figure 2.** (i) Susceptible humans and (ii) infectious humans.**Figure 3.** (i) Infectious mosquitoes and (ii) $\rho_2 \neq 0$, $\varepsilon_1 \neq 0$ and $M_{st} = 0$.

In the mosquito compartments shown in Figure (3i), vaccination and treatment also reduced the populations of the infectious mosquitoes slightly. As more humans are vaccinated and treated for malaria, it reduces the rate at which humans will infect mosquitoes, hence the reduction in the infected mosquito populations though the reduction is not as significant as in cases two, three and four. In Figure 3ii, the proportion of humans that should be treated or vaccinated for optimal control of the disease is shown.

5.2. Strategy two: $\rho_2 \neq 0, \varepsilon_1 = 0$ and $M_{st} \neq 0$

Here, we looked at the effects of vaccination and SIT only in the absence of treatment as shown in Figures (4–5). The control measures adopted here were also effective in reducing the infected populations in humans and mosquitoes. In Figure (4i), we can see that the control measures in case one had more effect in reducing the infectious human population than the control measures used in case two as shown in Figure (4i). This shows

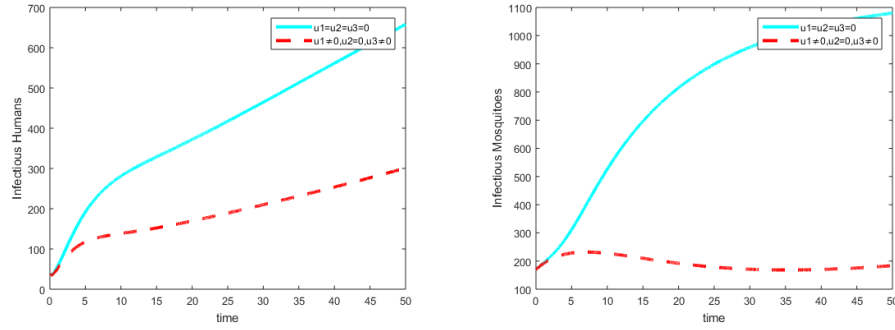


Figure 4. (i) Infectious humans and (ii) infectious mosquitoes

that while vaccination and use of SIT mosquitoes had more effect in protecting more humans against malaria initially, with time they fail to reduce the population of infected humans due to the absence of treatment. This underlines the importance of treatment as a control measure which must not be overlooked in controlling infectious diseases. In Figure 4ii, vaccination and use of SIT produced a better result in the mosquito population than vaccination and treatment as used in case one. This is because as SIT reduces the population of susceptible mosquitoes, the infected classes in the human population also decrease with time correspondingly, since there are fewer mosquitoes in circulation to infect them. Also, vaccination helps to protect humans from contracting malaria and infecting more mosquitoes. The proportions of humans that will be vaccinated and sterile mosquitoes which will be interacting with the female mosquitoes in the target population for optimal control of malaria are shown in Figure 5.

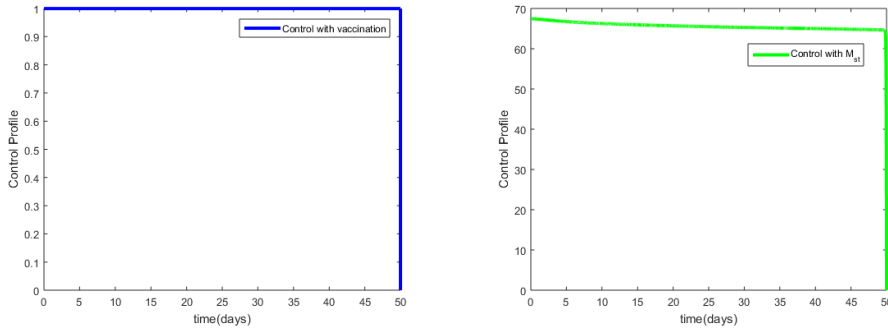


Figure 5. $\rho_2 \neq 0$, $\varepsilon_1 = 0$ and $M_{st} \neq 0$

5.3. Strategy three: $\rho_2 = 0, \varepsilon_1 \neq 0$ and $M_{st} \neq 0$

The effects of treatment and SIT in the absence of vaccination against malaria are considered here. The results are shown in Figures (6–7). In Figure (6i), we can see that the number of infectious humans decreased under the application of treatment and SIT as in case one, showing that the spread of malaria can be reduced by proper treatment and use of SIT procedures. Also, the infectious mosquito populations were reduced much more than in the first two cases, showing that using treatment and SIT procedures produces a greater effect in reducing the spread of malaria in the mosquito population, as shown in Figure 6ii.

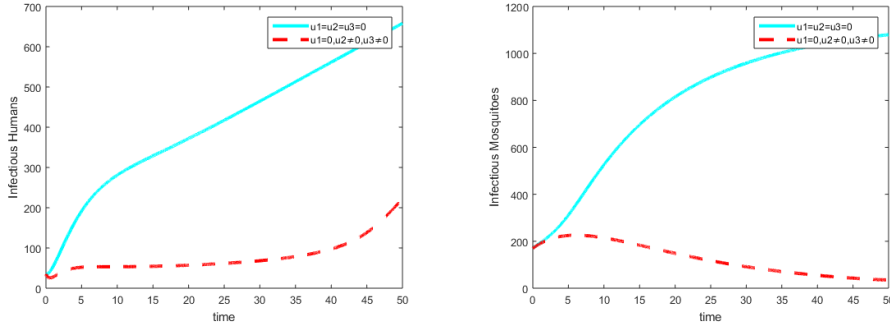


Figure 6. (i) Infectious humans and (ii) infectious mosquitoes

Also, comparing the three cases, we see that combining vaccination and treatment had more effect in reducing the spread of malaria amongst humans, while combining treatment and the SIT procedure produced more effect in the mosquito population. Hence, we suggest that if the three controls are employed simultaneously, malaria will be controlled more effectively, as demonstrated in case four. Figure 7 shows the number of infectious

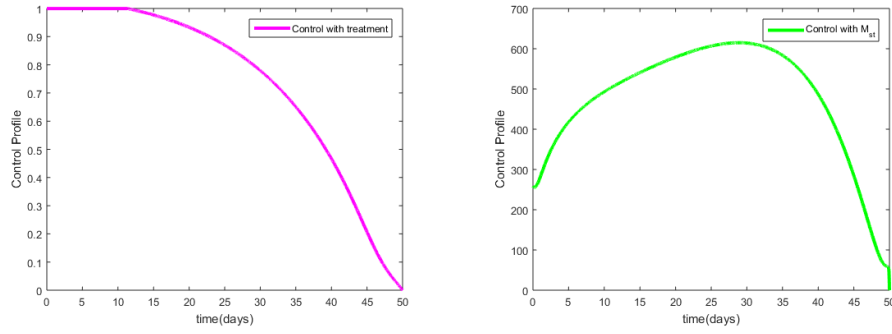


Figure 7. $\rho_2 = 0$, $\varepsilon_1 \neq 0$ and $M_{st} \neq 0$

humans that should be treated and sterile mosquitoes that should be interacting with females in the target population for optimal control of the disease.

5.4. Strategy four: $\rho_2 \neq 0, \varepsilon_1 \neq 0$ and $M_{st} \neq 0$

The optimal control results for employing the three controls are shown in Figures (8–9). Figure (8i) shows how effective the three controls were in causing a corresponding decrease in the number of infectious humans with malaria. Consequently, the infectious mosquito population shown in Figure (8ii) is reduced very considerably, showing that malaria can be eradicated if the population of the mosquitoes can be reduced or eliminated. It is clear to see that case four combines the optimal effects of cases one to three. Figures (9) shows the proportion of humans that should be vaccinated, the proportion of infectious humans that should be treated and the amount of SIT mosquitoes that should mate with females in the wild for optimal control of malaria.

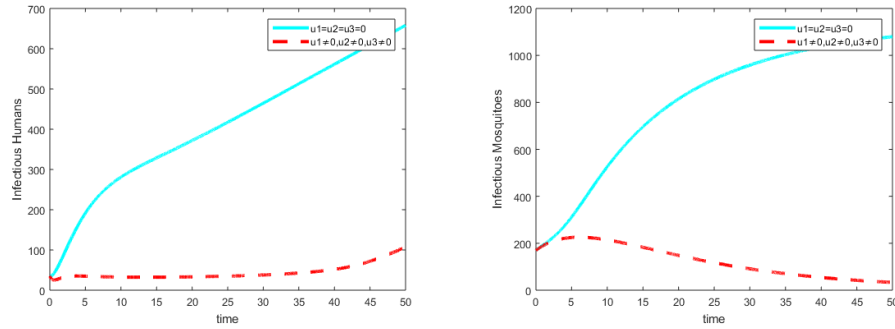


Figure 8. (i) Infectious humans and (ii) infectious mosquitoes

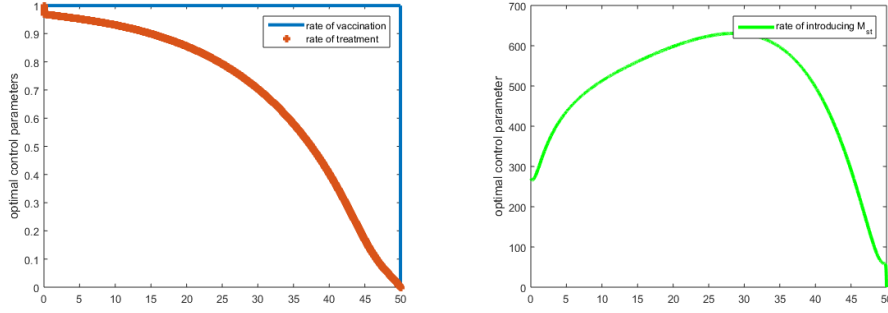


Figure 9. Optimal vaccination, treatment, and interaction by SIT mosquitoes

5.5. Cost-effectiveness analysis

Cost-effectiveness analysis is carried out in this section to determine the profitability of the control strategies by comparing them against each other. This is necessary since these control strategies are expensive and labour-intensive. Hence, the analysis is done to determine the most cost-effective strategy to apply. The cost-effectiveness analysis is carried out using the following cost analysis approaches: average cost-effectiveness ratio (ACER) and incremental cost-effectiveness ratio (ICER).

5.5.1. Average cost-effectiveness ratio

The average cost-effectiveness ratio (ACER) is defined as the ratio of the total cost incurred in implementing a particular strategy to the benefit derived from such strategy. This method is used to weigh a single intervention strategy against its baseline option. Mathematically, it is given as

$$\text{ACER} = \frac{\text{Total cost of an intervention}}{\text{Number of infections averted by the intervention}}.$$

The total cost (TC) is obtained from the objective functional (??), and the total number of infections averted (TA) is given by

$$TA = \int_0^{t_f} ((H + H_e + H_i + M_s) - (H^* + H_e^* + H_i^* + M_s^*)) dt,$$

where H^*, H_e^*, H_i^*, M_s^* are the optimal solutions associated with the optimal control parameters $\rho_2^*, \varepsilon_1^*$, and M_{st}^* . The ACER values are presented in Table 3 in increasing order of infection averted. The strategy with the lowest ACER value is the most cost-effective.

Table 3. TA and ACER for the system

Strategies	Intervention	TA	Cost (\$)	ACER
Strategy 2	ρ_2, M_{st}	3.3×10^2	1.643501×10^6	498.0308
Strategy 3	ε_1, M_{st}	6.35×10^4	1.4699013×10^7	231.4805
Strategy 1	ρ_2, ε_1	6.39×10^4	7.660546×10^6	119.8834
Strategy 4	$\rho_2, \varepsilon_1, M_{st}$	9.67×10^4	1.482635×10^7	153.3232

5.5.2. Incremental cost-effectiveness ratio

The incremental cost-effectiveness ratio is defined as the ratio of the difference between the costs of two competing strategies to the difference in the effect of the control strategies. Given any two control strategies i and j, ICER is mathematically defined as

$$ICER = \frac{\text{Difference in total cost between interventions i and j}}{\text{Difference in number of infections averted by interventions i and j}}.$$

To incrementally compare competing control strategies, one control strategy is compared with the next less effective alternative strategy. Using the information in Table 3, the ICER values comparing strategies 2 and 3 become

$$\begin{aligned}
 ICER(2) &= \frac{TC(2)}{TA(2)} = \frac{1.643501 \times 10^6}{3.3 \times 10^2} = 498.0306. \\
 ICER(3) &= \frac{TC(3) - TC(2)}{TA(3) - TA(2)} = \frac{1.4699013 \times 10^7 - 1.643501 \times 10^6}{6.35 \times 10^4 - 3.3 \times 10^2} = 216.8690. \\
 ICER(1) &= \frac{TC(1) - TC(3)}{TA(1) - TA(3)} = \frac{7.660546 \times 10^6 - 1.4699013 \times 10^7}{6.39 \times 10^4 - 6.35 \times 10^4} = -17,596.1675. \\
 ICER(4) &= \frac{TC(4) - TC(1)}{TA(4) - TA(1)} = \frac{1.482635 \times 10^7 - 7.660546 \times 10^6}{9.67 \times 10^4 - 6.39 \times 10^4} = 218.4696.
 \end{aligned}$$

Since ICER(2) is more dominant than ICER(3), strategy 3 is found to be more cost-effective than strategy 2, thus strategy 2 is eliminated from the list of options recommended for the control of the disease.

Table 4. Comparison of strategies 2 and 3

Strategies	Intervention	TA	Cost (\$)	ICER
Strategy 2	ρ_2, M_{st}	3.3×10^2	1.643501×10^6	498.0308
Strategy 3	ε_1, M_{st}	6.35×10^4	1.4699013×10^7	216.8690
Strategy 1	ρ_2, ε_1	6.39×10^4	7.660546×10^6	-17,596.1675
Strategy 4	$\rho_2, \varepsilon_1, M_{st}$	9.67×10^4	1.482635×10^7	218.4696

Next, strategy 3 is compared with strategy 1; the ICER values are presented in Table 5. The ICER value for strategy 1 is less than the ICER value for strategy 3. This shows

that strategy 1 is more cost-effective than strategy 3, hence strategy 3 is eliminated from the list of recommended cost-effective controls.

Table 5. Comparison of strategies 1 and 3

Strategies	Intervention	TA	Cost (\$)	ICER
Strategy 3	ε_1, M_{st}	6.35×10^4	1.4699013×10^7	231.4805
Strategy 1	ρ_2, ε_1	6.39×10^4	7.660546×10^6	-17,596.1675
Strategy 4	$\rho_2, \varepsilon_1, M_{st}$	9.67×10^4	1.482635×10^7	218.4696

Furthermore, strategy 1 is compared with strategy 4; the result is presented in Table 6. Since $ICER(4) < ICER(1)$, strategy 1 is more cost-effective than strategy 4, hence it is suggested that implementing strategy 1 will avert more infections with less cost. This confirms the result based on ACER values as shown in Table 3. The cost-effectiveness

Table 6. Comparison of strategies 1 and 4

Strategies	Intervention	TA	Cost (\$)	ICER
Strategy 1	ρ_2, ε_1	6.39×10^4	7.660546×10^6	119.8834
Strategy 4	$\rho_2, \varepsilon_1, M_{st}$	9.67×10^4	1.482635×10^7	218.4696

analysis showed that strategy 1, which involves employing vaccination and treatment, is more cost-effective in controlling the disease. Though strategy 4 averted more infections human than strategy 1, it is more cost-intensive. This shows that employing vaccination and treatment is cheaper than using the three controls: vaccination, treatment and the sterile insect technique. Employing the three controls averts more possible infections than strategy 1, but is costlier. Thus, strategy 1 is recommended for use since it is both affordable and advantageous.

6. Conclusion

In this work, we studied a mathematical model for the control of malaria. The model incorporates vaccination of humans against malaria, treatment of infectious humans, and recruitment of SIT mosquitoes to reduce the mosquito population. The optimal control analysis shows that the control measures used were highly effective in reducing the spread of malaria. It was shown that combining the three strategies produced a better result than when any two of the methods were employed. Hence, we conclude that malaria can be eliminated in any area where malaria is endemic if health practitioners can adopt mass vaccination of humans and deploy an SIT technique to reduce the population of mosquitoes, while ensuring that infected humans are properly treated. We also advocate that based on this research, malaria will be effectively controlled if intervention measures are focused on both human and mosquito populations simultaneously. Cost-effectiveness

analysis was also performed using the ACER and ICER techniques to identify the strategies which are more cost-effective. It was shown that combining treatment of infectious humans and vaccination is more cost-effective. The performance of SIT against other mosquito control measures, as well as the effect of combining biological and physical measures for control of mosquitoes, should be investigated in future research.

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