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Original Research Article

Investigating Malaria Spread and Optimal Control Measures with Prompt and Delayed Treatments

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ABSTRACT
Malaria, transmitted by Anopheles mosquitoes carrying *Plasmodium* parasites, poses a significant health burden, especially in tropical regions. This study employs a mathematical framework to explore malaria dynamics, emphasizing system stability and effective interventions that incorporate immediate (I_{E_h}) and delayed (I_{L_h}) treatment strategies. Using center manifold analysis, we investigate stability at the critical threshold R_0=1, identifying a forward bifurcation (a<0, b>0), indicating that maintaining R_0 below 1 halts disease persistence. By applying Pontryagin's principle and numerical optimization techniques, we formulate control strategies integrating rapid treatment, mosquito population management, and reduction of untreated infections, achieving a controlled reproduction number of R_0^c=0.1964, compared to an uncontrolled R_0=2.2356. These findings underscore the efficacy of combined interventions and provide actionable insights for malaria control in resource-limited settings.

INTRODUCTION

Malaria, driven by Plasmodium parasites and transmitted via Anopheles mosquitoes, remains a critical public health issue, particularly in sub-Saharan Africa (World Health Organization, 2023). Mathematical models provide valuable tools for understanding disease transmission and crafting effective interventions. This work presents an SEIIR-SEI model that distinguishes between individuals receiving prompt treatment and those with delayed care. Our objectives are to analyze the system's behavior at the threshold R_0=1 using advanced stability techniques and to develop optimized intervention strategies, including rapid treatment, mosquito population reduction, and minimizing untreated cases, to reduce transmission. Our analysis confirms a forward bifurcation at R_0=1, ensuring disease eradication when R_0<1, and offers practical intervention strategies for high-transmission, resource-constrained regions.

In regions like Nigeria, malaria's burden is exacerbated by limited healthcare access and environmental conditions conducive to mosquito proliferation (World Health Organization, 2023). Effective control demands integrated approaches targeting both human infections and mosquito vectors. This study builds on prior research by focusing on system dynamics and customized intervention strategies for high-transmission settings (Opaginni & Durojaye, 2025).

Mathematical frameworks have been widely applied to study infectious diseases, using methods like spectral analysis to estimate R_0 (van den Driessche & Watmough, 2002). Stability analyses differentiate between forward and backward bifurcations at R_0=1, with backward bifurcations complicating eradication efforts (Castillo-

Chavez & Song, 2004; Chitnis, Cushing, & Hyman, 2006). Insights from similar epidemiological modeling efforts, such as the survival analysis of TB patients using Weibull and Log-Logistic Accelerated Failure Time model (AFT) models (Exploring Accelerated Failure Time Models for Tuberculosis Survival: Log-Logistic and Weibull Survival Regression Model,) (Usman, Doguwa, Sadiq &Akor, 2025), reinforce the importance of selecting model structures that best capture disease progression variability. Vector control is critical alongside medical interventions (Isah, Ibrahim, Isah, & Magaji, 2024). Prompt treatment reduces severe outcomes, while inconsistent efforts hinder progress (Challenger et al., 2019; Mousa et al., 2020). In Nigeria, challenges such as drug resistance and overtreatment underscore the need for holistic strategies (Anjorin et al., 2023; Collins & Duffy, 2022). While previous studies have examined treatment and vaccination (Joshi, Maity, & Prajapati, 2006; Okosun & Makinde, 2013), few integrate prompt and delayed treatment with vector control. This research advances the SEIIR-SEI model to evaluate stability and optimize interventions (Opaginni & Durojaye, 2025).

Model Assumptions

The model relies on the following premises:

1. Mosquito biting frequency is constant, excluding seasonal variations.
2. Human-mosquito interactions are uniform.
3. The model omits drug resistance and spatial dynamics.
4. Recovery rates vary between prompt and delayed treatment groups.
5. Superinfection or reinfection is not considered during the study period.

Stability Analysis of the Malaria Model

This study refines an SEIIR-SEI model to examine malaria transmission dynamics, with a focus on equilibrium stability (Opaginni & Durojaye, 2025). The system is governed by:

$$\frac{dS_h}{dt}=\alpha_h-\sigma b\psi S_h I_m+\delta_3 R_h-\mu_h S_h, \quad (1)$$

$$\frac{dE_h}{dt}=\sigma b\psi S_h I_m-(\mu_h+\delta_1+\delta_2)E_h, \quad (2)$$

$$\frac{dI_{E_h}}{dt}=\delta_1 E_h-(\mu_h+\eta_h+\lambda_1+a)I_{E_h}, \quad (3)$$

$$\frac{dI_{L_h}}{dt}=\delta_2 E_h+aI_{E_h}-(\mu_h+\eta_h+\lambda_2)I_{L_h}, \quad (4)$$

$$\frac{dR_h}{dt}=\lambda_1 I_{E_h}+\lambda_2 I_{L_h}-(\mu_h+\eta_h+\delta_3)R_h, \quad (5)$$

$$\frac{dS_m}{dt}=\alpha_m-\sigma b\psi S_m I_{E_h}-\sigma b\psi_1 S_m I_{L_h}-\mu_m S_m, \quad (6)$$

$$\frac{dE_m}{dt}=\sigma b\psi S_m I_{E_h}+\sigma b\psi_1 S_m I_{L_h}-(\mu_m+\delta_m+\gamma_m)E_m, \quad (7)$$

$$\frac{dI_m}{dt}=\delta_m E_m-\mu_m I_m \quad (8)$$

The total populations are:

$$N_h=S_h+E_h+I_{E_h}+I_{L_h}+R_h, \quad (9)$$

$$N_m=S_m+E_m+I_m. \quad (10)$$

Parameters and Variables

Table 1: Description of Model Parameters

Parameter	Description
μ_h,μ_m	Natural mortality rates for humans and mosquitoes.
α_h	Human recruitment rate.
α_m	Mosquito recruitment rate.
b	Probability of human infection from an infectious mosquito bite.
a	Rate of transition from prompt to delayed treatment.
c	Probability of mosquito infection from biting an infectious human.
σ	Mosquito biting frequency on humans.
ψ	Interaction rate between mosquitoes and humans.
ψ_1	Interaction rate for delayed treatment cases.
δ_1	Rate of progression to infectious state with prompt treatment.
δ_2	Rate of progression to infectious state with delayed treatment.
λ_1	Recovery rate for prompt treatment cases.
λ_2	Recovery rate for delayed treatment cases.
δ_3	Rate of immunity loss, returning to susceptible state.
δ_m	Mosquito progression rate to infectious state.
γ_m	Loss rate of exposed mosquitoes due to prompt treatment.

Table 2: Model Variables

Variables	Description
N_h	Total human population.
N_m	Total mosquito population.
S_h	Susceptible human population.
E_h	Exposed human population.
I_{E_h}	Infected humans receiving prompt treatment.
I_{L_h}	Infected humans receiving delayed treatment.
R_h	Recovered human population.
S_m	Susceptible mosquito population.
E_m	Exposed mosquito population.
I_m	Infectious mosquito population.

Basic Reproduction Number

The reproduction number R_0 is determined using a spectral radius method (van den Driessche & Watmough, 2002):

$$R_0=\rho(A)=\frac{b\sigma\sqrt{\alpha_h\alpha_m\delta_m\left(a\delta_1\psi_1+a\delta_2\psi_1+\delta_1\eta_h\psi+\delta_1\lambda_2\psi+\delta_1\mu_h\psi+\delta_2\eta_h\psi_1+\delta_2\lambda_1\psi_1+\delta_2\mu_h\psi_1\right)}}{\mu_m\sqrt{\mu_h(\delta_1+\delta_2+\mu_h)(\delta_m+\gamma_m+\mu_m)(\eta_h+\lambda_2+\mu_h)(a+\eta_h+\lambda_1+\mu_h)}} \quad (11)$$

It is decomposed into contributions from prompt ($R_{0,\text{prompt}}$) and delayed ($R_{0,\text{delayed}}$) treatment:

$$R_0=\sqrt{R_{0,\text{prompt}}^2+R_{0,\text{delayed}}^2}, \quad (12)$$

where

$$R_{0,\text{prompt}}=\frac{b\sigma\sqrt{\alpha_h\alpha_m\delta_m\left(a\delta_1\psi+\delta_1(\eta_h+\lambda_2+\mu_h)\right)}}{\mu_m\sqrt{\mu_h(\delta_1+\delta_2+\mu_h)(\delta_m+\gamma_m+\mu_m)(\eta_h+\lambda_2+\mu_h)(a+\eta_h+\lambda_1+\mu_h)}} \quad (13)$$

$$R_{0,\text{delayed}}=\frac{b\sigma\sqrt{\alpha_h\alpha_m\delta_m\left(a\delta_2\psi_1+\delta_2(\eta_h+\lambda_1+\mu_h)\right)}}{\mu_m\sqrt{\mu_h(\delta_1+\delta_2+\mu_h)(\delta_m+\gamma_m+\mu_m)(\eta_h+\lambda_2+\mu_h)(a+\eta_h+\lambda_1+\mu_h)}} \quad (14)$$

The disease-free equilibrium is stable when $R_0<1$, and an endemic equilibrium arises when $R_0>1$.

Bifurcation Analysis

We apply center manifold analysis to examine system behavior at $R_0=1$ (Castillo-Chavez & Song, 2004). The system is expressed as:

$$\frac{dX}{dt}=F(X), \quad X=(S_h,E_h,I_{E_h},I_{L_h},R_h,S_m,E_m,I_m)^T. \quad (15)$$

The transmission rate is $\beta=b\sigma\psi_{\text{eff}}$, where $\psi_{\text{eff}}=\psi+\psi_1$, assuming $\psi=c\psi_1$, with $c=1$. The effective infectivity is:

$$R_0=\frac{b\sigma\psi_{\text{eff}}\sqrt{\alpha_h\alpha_m\delta_m(\delta_1+\delta_2)}}{\mu_m\sqrt{\mu_h(\delta_1+\delta_2+\mu_h)(\delta_m+\gamma_m+\mu_m)}} \quad (16)$$

$$\psi_{\text{eff}}=\sqrt{\frac{\delta_1(\eta_h+\lambda_2+\mu_h)\psi+\left[a\delta_1+\delta_2(\eta_h+\lambda_1+\mu_h)\right]\psi_1}{\delta_1+\delta_2}} \quad (17)$$

The critical transmission rate at $R_0=1$ is:

$$\beta^*=\mu_m\sqrt{\frac{\mu_h(\delta_1+\delta_2+\mu_h)(\delta_m+\gamma_m+\mu_m)(\eta_h+\lambda_2+\mu_h)(a+\eta_h+\lambda_1+\mu_h)}{\alpha_h\alpha_m\delta_m(\delta_1+\delta_2)}} \quad (18)$$

The Jacobian at the disease-free equilibrium $E_1=\left(\frac{\alpha_h}{\mu_h},0,0,0,0,\frac{\alpha_m}{\mu_m},0,0\right)$ with $\beta=\beta^*$ is:

$$J_B=\begin{bmatrix} -\mu_h & 0 & 0 & 0 & \delta_3 & 0 & 0 & -\beta^*S_h^0 \\ 0 & -k_1 & 0 & 0 & 0 & 0 & 0 & \beta^*S_h^0 \\ 0 & \delta_1 & -k_3 & 0 & 0 & 0 & 0 & 0 \\ 0 & \delta_2 & a & -k_4 & 0 & 0 & 0 & 0 \\ 0 & 0 & \lambda_1 & \lambda_2 & -k_5 & 0 & 0 & 0 \\ 0 & 0 & -\beta^*\psi S_m^0 & -\beta^*\psi_1 S_m^0 & 0 & -\mu_m & 0 & 0 \\ 0 & 0 & \beta^*\psi S_m^0 & \beta^*\psi_1 S_m^0 & 0 & 0 & -k_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \delta_m & -\mu_m \end{bmatrix} \quad (19)$$

where:

$k_1=\mu_h+\delta_1+\delta_2$,

$k_3=\mu_h+\eta_h+\lambda_1+a$,

$k_4=\mu_h+\eta_h+\lambda_2$,

$k_5=\mu_h+\delta_3$,

$k_7=\mu_m+\delta_m+\gamma_m$,

$S_h^0=\alpha_h/\mu_h$,

$S_m^0=\alpha_m/\mu_m$.

The right and left eigenvectors are:

$$v=\begin{bmatrix} \frac{\delta_3 v_5-\beta^*S_h^0}{\mu_h} \\ \frac{\beta^*S_h^0}{k_1} \\ \frac{\delta_1\beta^*S_h^0}{k_1k_3} \\ \frac{\beta^*S_h^0(\delta_2k_3+a\delta_1)}{k_1k_3k_4} \\ \frac{\lambda_1\delta_1\beta^*S_h^0/(k_1k_3)+\lambda_2\beta^*S_h^0(\delta_2k_3+a\delta_1)/(k_1k_3k_4)}{k_5} \\ -\frac{\beta^*S_m^0\left(\psi\delta_1/(k_1k_3)+\psi_1(\delta_2k_3+a\delta_1)/(k_1k_3k_4)\right)}{\mu_m} \\ \frac{\beta^*S_m^0\left(\psi\delta_1/(k_1k_3)+\psi_1(\delta_2k_3+a\delta_1)/(k_1k_3k_4)\right)}{k_7} \\ 1 \end{bmatrix}, \quad (20)$$

$$w=\begin{bmatrix} 0 \\ \frac{\mu_m}{\beta^* S_h^0} \\ \frac{\delta_2\mu_m/(\beta^* S_h^0)+aw_4}{k_3} \\ \frac{\beta^*\psi_1 S_m^0\delta_m/k_7}{k_4} \\ 0 \\ 0 \\ \frac{\delta_m}{k_7} \\ 1 \end{bmatrix}. \quad (21)$$

Bifurcation coefficients are:

$$a=2\beta^*\left(w_2v_1v_8-\frac{w_7\beta^*(\psi v_3+\psi_1 v_4)^2 S_m^0}{\mu_m}\right), \quad (22)$$

$$b=\frac{\mu_m}{\beta^*}+\frac{\delta_m S_m^0}{\mu_m+\delta_m+\gamma_m}(\psi v_3+\psi_1 v_4). \quad (23)$$

Using parameters from (Opaginni & Durojaye, 2025), we find $\psi_{eff}\approx 0.818$, $\beta^*\approx 0.0229$, a=-4.797, b>0, confirming a forward bifurcation at R_0=1.

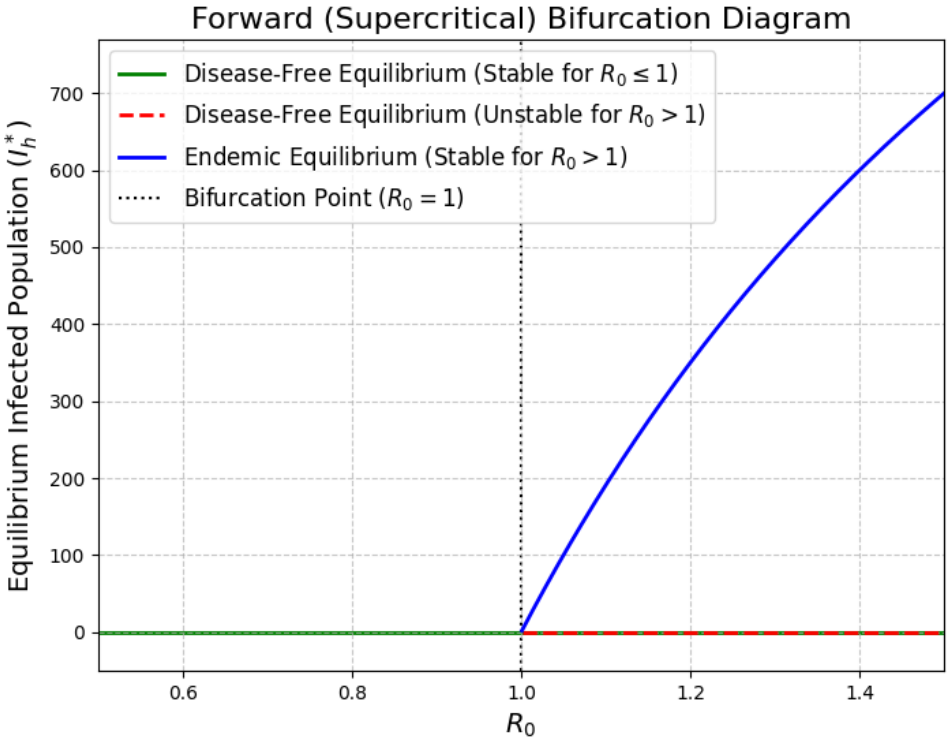


Figure 1: Forward Bifurcation at R_0=1

Figure 1 illustrates the infected human population (I_h^*) against R_0. The green curve (disease-free state) is stable when R_0<1, the red dashed curve indicates instability when R_0>1, and the blue curve depicts a stable endemic state for R_0>1.

Effective Control Strategies

To curb malaria transmission, we propose three dynamic intervention measures:

1. $u_1(t)$: Rapid treatment to shorten infectious periods.
2. $u_2(t)$: Mosquito population management to reduce transmission.
3. $u_3(t)$: Efforts to address untreated infections to enhance recovery.

Control Model

The control model extends Equation (2.1 – 2.8):

$$\frac{dS_h}{dt}=\alpha_h-\sigma b\psi S_h I_m+\delta_3 R_h-\mu_h S_h, \quad (24)$$

$$\frac{dE_h}{dt}=\sigma b\psi S_h I_m-(\mu_h+\delta_1+\delta_2)E_h, \quad (25)$$

$$\frac{dI_{E_h}}{dt}=\delta_1 E_h-(\mu_h+\eta_h+\lambda_1+a+u_1)I_{E_h}, \quad (26)$$

$$\frac{dI_{L_h}}{dt}=\delta_2 E_h+aI_{E_h}-(\mu_h+\eta_h+\lambda_2+u_3)I_{L_h}, \quad (27)$$

$$\frac{dR_h}{dt}=\lambda_1 I_{E_h}+\lambda_2 I_{L_h}-(\mu_h+\eta_h+\delta_3)R_h+u_1 I_{E_h}+u_3 I_{L_h}, \quad (28)$$

$$\frac{dS_m}{dt}=\alpha_m-\sigma b\psi(1-u_2)S_m I_{E_h}-\sigma b\psi_1(1-u_2)S_m I_{L_h}-\mu_m S_m, \quad (29)$$

$$\frac{dE_m}{dt}=\sigma b\psi(1-u_2)S_m I_{E_h}+\sigma b\psi_1(1-u_2)S_m I_{L_h}-(\mu_m+\delta_m+\gamma_m)E_m, \quad (30)$$

$$\frac{dI_m}{dt}=\delta_m E_m-\mu_m I_m \quad (31)$$

Numerical Approach

We constructed a deterministic model for *Plasmodium falciparum* transmission, incorporating prompt and delayed treatment alongside mosquito management. Optimal interventions were derived using an optimization principle (Fleming & Rishel, 2012), solved numerically via an iterative technique. State equations were solved

forward using a high-order numerical method, adjoint equations backward, and controls updated iteratively within $0\le u_i(t)\le 1$. Simulations were performed in Python using parameters from Table 1.

Objective Functional

The cost functional is:

$$J(u_1,u_2,u_3)=\int_{t_0}^{t_f}\left[A_1 I_{E_h}(t)+A_2 I_{L_h}(t)+A_3 I_m(t)+B_1 u_1^2(t)+B_2 u_2^2(t)+B_3 u_3^2(t)\right]dt, \quad (32)$$

where A_1,A_2,A_3 weight the infectious populations, and B_1,B_2,B_3 represent intervention costs. The goal is to minimize J over $[t_0,t_f]$, with controls in:

$$U=\left\{(u_1(t),u_2(t),u_3(t))\in L^{\infty}(t_0,t_f)^3 \mid 0\le u_i(t)\le 1\right\}. \quad (33)$$

Theorem 1 (Existence of Optimal Controls) An optimal control triple (u_1^*,u_2^*,u_3^*) exists that minimizes J, provided:

1. The system has a non-empty set of solutions.
2. The control set U is convex and closed.
3. The dynamic system is continuous, bounded, and linear in the controls.
4. The cost functional's integrand is convex with respect to the controls.
5. The integrand satisfies:

$A_1 I_{E_h}+A_2 I_{L_h}+A_3 I_m+B_1 u_1^2+B_2 u_2^2+B_3 u_3^2 \ge -l_1+l_2|u_1|^l+l_3|u_2|^l+l_4|u_3|^l$,

for positive constants l_1,l_2,l_3,l_4, $l>1$.

Proof. Using Filippov's Existence Theorem (Fleming & Rishel, 2012; Lenhart & Workman, 2007):

1. The system has a unique solution due to continuous, locally Lipschitz right-hand sides and constrained controls.
2. The control set U is convex and closed, as $0\le \lambda u_i+(1-\lambda)v_i\le 1$.
3. The dynamic equations are continuous, bounded, and linear in u_1,u_2,u_3.
4. The integrand is convex, as $(\lambda a_i+(1-\lambda)b_i)^2-\lambda a_i^2-(1-\lambda)b_i^2\le 0$.
5. The integrand satisfies the coercivity condition with $l_1=A_1 I_{E_h}+A_2 I_{L_h}+A_3 I_m$, $l_2=B_1$, $l_3=B_2$, $l_4=B_3$, $l=2$.

Thus, an optimal control exists.

Optimal Control Characterization

The Hamiltonian is:

$$\begin{aligned}H=&p_1\left(\alpha_h-\sigma b\psi S_h I_m+\delta_3 R_h-\mu_h S_h\right)\\&+p_2\left(\sigma b\psi S_h I_m-(\mu_h+\delta_1+\delta_2)E_h\right)\\&+p_3\left(\delta_1 E_h-(\mu_h+\eta_h+\lambda_1+a+u_1)I_{E_h}\right)\\&+p_4\left(\delta_2 E_h+aI_{E_h}-(\mu_h+\eta_h+\lambda_2+u_3)I_{L_h}\right)\\&+p_5\left(\lambda_1 I_{E_h}+\lambda_2 I_{L_h}-(\mu_h+\eta_h+\delta_3)R_h+u_1 I_{E_h}+u_3 I_{L_h}\right)\\&+p_6\left(\alpha_m-\sigma b\psi(1-u_2)S_m I_{E_h}-\sigma b\psi_1(1-u_2)S_m I_{L_h}-\mu_m S_m\right)\\&+p_7\left(\sigma b\psi(1-u_2)S_m I_{E_h}+\sigma b\psi_1(1-u_2)S_m I_{L_h}-(\mu_m+\delta_m+\gamma_m)E_m\right)\\&+p_8\left(\delta_m E_m-\mu_m I_m\right)\\&-\left(A_1 I_{E_h}+A_2 I_{L_h}+A_3 I_m+B_1 u_1^2+B_2 u_2^2+B_3 u_3^2\right)\end{aligned} \quad (34)$$

The adjoint equations are:

$$\dot p_1=p_1(\sigma b\psi I_m+\mu_h)-p_2\sigma b\psi I_m, \quad (35)$$

$$\dot p_2=p_2(\mu_h+\delta_1+\delta_2)-p_3\delta_1-p_4\delta_2, \quad (36)$$

$$\dot p_3=p_3(\mu_h+\eta_h+\lambda_1+a+u_1)-p_4 a-p_5(\lambda_1+u_1)+(p_6-p_7)\sigma b\psi(1-u_2)S_m+A_1, \quad (37)$$

$$\dot p_4=p_4(\mu_h+\eta_h+\lambda_2+u_3)-p_5(\lambda_2+u_3)+(p_6-p_7)\sigma b\psi_1(1-u_2)S_m+A_2, \quad (38)$$

$$\dot p_5=p_5(\mu_h+\eta_h+\delta_3)-p_1\delta_3, \quad (39)$$

$$\dot p_6=p_6\left[\sigma b\psi(1-u_2)I_{E_h}+\sigma b\psi_1(1-u_2)I_{L_h}+\mu_m\right] \quad (40)$$
$$-p_7\left[\sigma b\psi(1-u_2)I_{E_h}+\sigma b\psi_1(1-u_2)I_{L_h}\right],$$

$$\dot p_7=p_7(\mu_m+\delta_m+\gamma_m)-p_8\delta_m, \quad (41)$$

$$\dot p_8=(p_1-p_2)\sigma b\psi S_h+p_8\mu_m+A_3. \quad (42)$$

Optimal controls are:

$$u_1^*(t)=\max\left\{0,\min\left\{1,\frac{(p_5-p_3)I_{E_h}}{2B_1}\right\}\right\}, \quad (43)$$

$$u_2^*(t)=\max\left\{0,\min\left\{1,\frac{\sigma b S_m(p_6-p_7)(\psi I_{E_h}+\psi_1 I_{L_h})}{2B_2}\right\}\right\}, \quad (44)$$

$$u_3^*(t)=\max\left\{0,\min\left\{1,\frac{(p_5-p_4)I_{L_h}}{2B_3}\right\}\right\}. \quad (45)$$

Controlled Reproduction Number

The controlled reproduction number R_0^c is evaluated using a spectral radius approach (van den Driessche & Watmough, 2002):

$$FV^{-1}=\begin{pmatrix}0 & 0 & 0 & \frac{\sigma b\psi S_h^0\delta_m}{k_4\mu_m} & \frac{\sigma b\psi S_h^0}{\mu_m}\\ 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0\\ \frac{\sigma b\psi(1-u_2)S_m^0}{k_1} & \frac{\sigma b\psi(1-u_2)S_m^0\delta_1}{k_1k_2} & \frac{\sigma b\psi(1-u_2)S_m^0(a\delta_1+\delta_2k_2)}{k_1k_2k_3} & 0 & 0\\ 0 & 0 & 0 & 0 & 0\end{pmatrix} \quad (46)$$

where:

$$k_1=\mu_h+\delta_1+\delta_2, \quad (47)$$

$$k_2=\mu_h+\eta_h+\lambda_1+a+u_1, \quad (48)$$

$$k_3=\mu_h+\eta_h+\lambda_2+u_3, \quad (49)$$

$$k_4=\mu_m+\delta_m+\gamma_m. \quad (50)$$

The spectral radius is:

$$R_0^c=\sqrt{\frac{\sigma^2 b^2(1-u_2)^2 a_h a_m\delta_m\left(\psi^2\delta_1^2k_3+\psi\psi_1\delta_1(a\delta_1+\delta_2k_2)+\psi_1^2\delta_2^2k_2\right)}{\mu_h\mu_m^3k_1^2k_2k_3k_4}+\frac{\sigma^2b^2\psi^2a_h\delta_m}{\mu_h\mu_m^2k_4}} \quad (51)$$

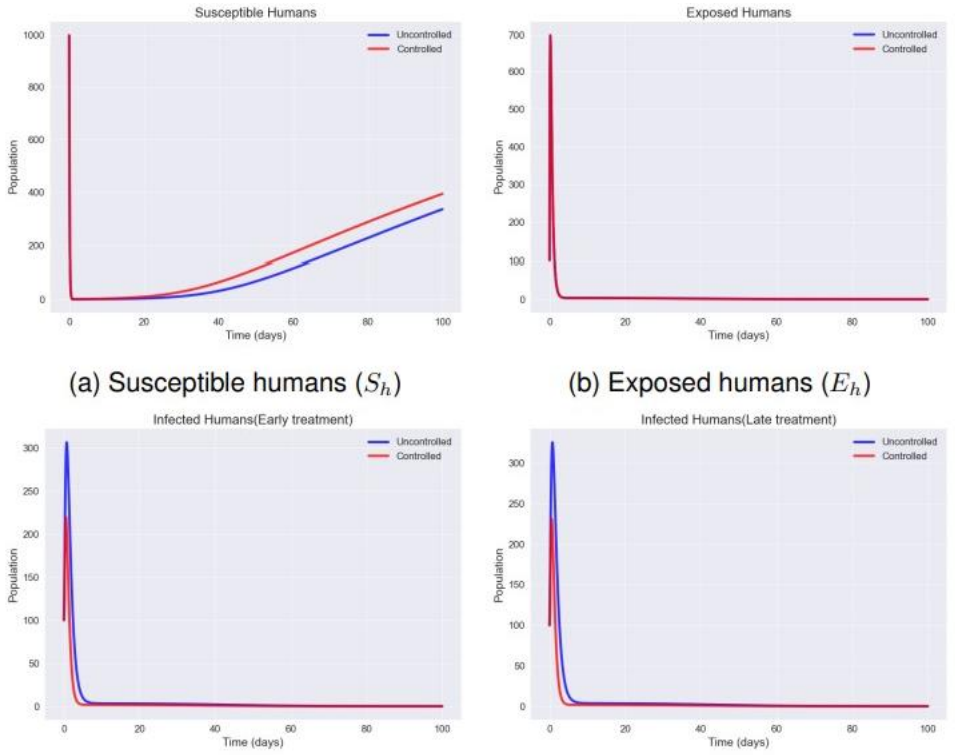
RESULTS AND DISCUSSION

The optimal intervention strategy reduced R_0^c to 0.1964 from an uncontrolled R_0=2.2356, as derived in previous work (Opaginni & Durojaye, 2025),, demonstrating effective transmission suppression. Average intervention intensities are:

u_1^*=0.4107: Prompt treatment.

u_2^*=0.7204: Mosquito control.

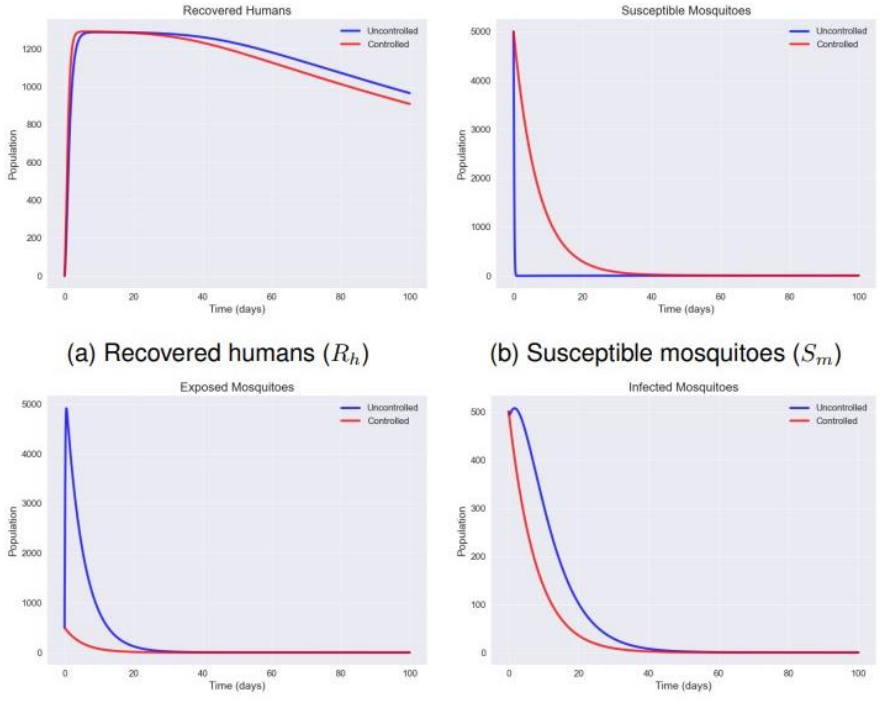
u_3^*=0.4297: Reduction of untreated cases.



(a) Susceptible humans (S_h) (b) Exposed humans (E_h)

(c) Infected with prompt treatment (I_{E_h}) (d) Infected with delayed treatment (I_{L_h})

Figure 2: Dynamics under uncontrolled (blue) and controlled (red) scenarios over 100 days



(a) Recovered humans (R_h) (b) Susceptible mosquitoes (S_m)

(c) Exposed mosquitoes (E_m) (d) Infected mosquitoes (I_m)

Figure 3: Dynamics under uncontrolled (blue) and controlled (red) scenarios over 100 days

The controlled scenario reveals:

1. Increased S_h, indicating fewer infections.
2. Lower E_h, showing effective prevention.
3. Reduced I_{E_h} and I_{L_h}, due to timely treatment and case management.
4. Higher R_h, reflecting improved recovery rates.
5. Increased S_m, with lower E_m and I_m, indicating disrupted mosquito transmission.

Discussion

The forward bifurcation, with a=-4.797 and b>0, indicates that keeping R_0 below 1 ensures disease eradication. The integrated strategy of rapid treatment, mosquito management, and addressing untreated cases reduced R_0^c to 0.1964, demonstrating robust control over malaria spread. Prompt treatment is most effective early, mosquito control sustains long-term impact, and managing untreated cases significantly reduces infected populations within 100 days, offering a practical approach for resource-scarce regions.

CONCLUSION

This study explores malaria transmission dynamics using an SEIIR-SEI model that differentiates between immediate and delayed treatment. The forward bifurcation at R_0=1 (a<0, b>0) confirms that maintaining R_0<1 eliminates the disease. Optimized interventions (u_1^*=0.4107, u_2^*=0.7204, u_3^*=0.4297) achieved a controlled R_0^c=0.1964, significantly reducing transmission. These results highlight the effectiveness of integrated strategies for malaria control in high-prevalence regions like Nigeria. Future research could investigate the effects of drug resistance and spatial dynamics on transmission.

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