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# On Models of Malaria with Natural Recovery

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**Abstract**

This study presents a mathematical model for malaria transmission dynamics, incorporating natural recovery and public awareness/sensitization within the human population. The model evaluates the impact of sensitization alongside conventional control strategies in mitigating malaria spread. Through qualitative analysis, the basic reproduction number $R_0$ was determined to be less than unity, suggesting the feasibility of disease control. Additionally, stability analysis confirmed that the disease-free equilibrium is locally and asymptotically stable. Our findings indicate that, with a combination of natural recovery, public sensitization, and conventional interventions, malaria can be successfully eradicated from the population.

**Keywords**: Stability, Basic Reproduction Number, Ruth Hurwitz Criterion, Linearization, Next Generation Martix


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## Introduction

Malaria remains one of the most severe infectious diseases worldwide, posing a significant public health threat. It is caused by Plasmodium parasites, which are transmitted between humans through the bites of infected *Anopheles* mosquitoes, primarily from dusk to dawn. The five major Plasmodium species responsible for malaria in humans include *Plasmodium vivax*, *Plasmodium ovale*, *Plasmodium malariae*, *Plasmodium falciparum*, and *Plasmodium knowlesi*. Among these, *Plasmodium falciparum* (*P. falciparum*) is the most lethal and widespread, particularly in sub-Saharan Africa, where children under five and pregnant women face the highest risk (WHO, 2016). The incubation period for *P. falciparum* is approximately 12 days in humans and 10 days in mosquitoes.

Malaria presents with a range of symptoms, from mild to severe, including fatigue, chills, headaches, abdominal and back pain, diarrhea, vomiting, and fever. In extreme cases, complications may arise, affecting essential organs such as the brain, lungs, and kidneys. The persistence of malaria transmission, particularly in highly endemic areas, is influenced by factors such as human behavior and environmental conditions.

Despite global initiatives aimed at controlling and eliminating malaria, the disease remains prevalent, especially in sub-Saharan Africa. According to WHO (2018), 91% of global malaria deaths occurred in Africa, with Nigeria alone accounting for 24% of malaria-related fatalities, making it the most affected country on the continent.

Mathematical modeling has become an essential tool in understanding malaria transmission and evaluating intervention strategies. Researchers have examined various factors affecting malaria spread and control.

Yang (2000) analyzed the impact of environmental factors on malaria transmission. Smith and Hove-Musekwa (2008) explored how climate change influences malaria vector prevalence. Chitnis (2005) developed an SEIR model to compare malaria control strategies in areas with high and low transmission rates. Peter (2010) formulated a deterministic model to evaluate the role of protection and treatment in malaria dynamics. Adamu and Kimbir (2013) and Adamu et al. (2017) investigated the combined effects of treatment, vaccination, and protection in controlling malaria. Olaniyi and Obabiyi (2013) examined the influence of antibodies and immune response in malaria infections. Musa and Goni (2018) and Goni (2016) assessed the effectiveness of education-based interventions in reducing malaria transmission.

Natural recovery from malaria refers to the body's ability to clear the infection without medical intervention (Doolan et al. 2009). This process is influenced by immune responses, genetic factors, and environmental conditions. Understanding natural recovery can provide insights into malaria immunity, resistance, and potential eradication strategies (Marsh & Kinyanjui, 2006).

Mechanisms of natural recovery is driven by several biological and immunological factors such as Innate immune response, adaptive immunity and antibody production, genetic resistance and natural selection as well as fever and other physiological responses (Williams, 2006), (White et al. 2014), (Miller et al. 2002). Several factors affecting an individual's ability to naturally recover from malaria include Age and immunity, nutritional status, Co-infections and comorbidities as well as parasite load and strain variability (WHO, 2018)

Natural recovery from malaria is an essential aspect of disease dynamics, shaped by immune responses, genetic factors, and environmental influences (Greenwood et.al, 2008), (Olliaro, 2002). While medical treatments are vital in controlling malaria, understanding natural immunity provides valuable insights for long-term eradication strategies (Schofied & Grau, 2005), (Langhorne et al. 2008). Future research should focus on harnessing natural immunity for vaccine development and improving public health interventions.

**Mathematical Model**

The proposed model extends the framework of Musa and Goni (2018) by incorporating additional dynamics. It considers two interacting populations: humans (hosts) and mosquitoes (vectors). The human population follows a Susceptible-Protected-Exposed-Infectious-Recovered (SPEIR) framework. The mosquito population follows a Susceptible-Exposed-Infectious (SEI) structure. This study aims to assess the impact of natural recovery from malaria and sensitization efforts within the human population on the transmission dynamics of the disease.

*Assumptions of the Model*

The proposed model is based on the following assumptions:

a. Disease-Induced Mortality: In addition to natural death, infected humans may die as a result of malaria.

b. Lifelong Infectivity in Mosquitoes: Once infected, mosquitoes remain infectious for life and may die due to the disease.

c. Temporary Immunity in Recovered Humans: Individuals who recover from malaria acquire temporary immunity but eventually lose it, becoming susceptible again.

d. Antibody Response: Both susceptible humans and mosquitoes produce antibodies in response to malaria parasites.

e. Health Behavior and Protection: Susceptible humans who develop positive health behaviors can protect themselves, reducing contact with mosquitoes.

f. Loss of Protection: Protected humans may lose their protection over time, making them susceptible to infection again.

g. No Forced Migration: The model assumes there are no events causing large-scale forced migration of people.

*State Variables of the Model*

Table 1: State Variables of the Model

| *State variables* | Description |
|---|---|
| $S_h(t)$ | Number of susceptible human host to malaria infection at time $t$ |
| $P_h$ | Number of protected human host at time $t$ |
| $E_h(t)$ | Number of human host exposed to malaria infection at time $t$ |
| $I_h(t)$ | Number of Infectious human host at time $t$ |
| $R_h(t)$ | Number of Recovered human host at time $t$ |
| $S_m(t)$ | Number of Susceptible mosquitoes at time $t$ |
| $E_m(t)$ | Number of exposed mosquitoes at time $t$ |
| $I_m(t)$ | Number of infectious mosquitoes at time $t$ |

*Parameters of the Model*

Table 2: Parameters of the Model

| Parameter | Description |
|---|---|
| $\Lambda_h$ | Recruitment rate of susceptible humans |
| $\Lambda_m$ | Recruitment rate of susceptible mosquitoes |
| $b$ | Biting rate of mosquito |
| $\beta_h$ | Probability that a bite by an infectious mosquito results in transmission of the disease to humans |
| $\beta_m$ | Probability that a bite results in transmission of parasite to a |

| | |
|---|---|
| | susceptible mosquito |
| $\mu_h$ | Per capita death rate of humans |
| $\mu_m$ | Per capita death rate of humans |
| $\delta_h$ | Disease-induced death rate of humans |
| $\delta_m$ | Disease-induced death rate of mosquito |
| $\alpha_h$ | Per capita rate of progression of humans from exposed state to infectious state |
| $\alpha_m$ | Per capita rate of progression of mosquito from the exposed state to infectious |
| $r$ | Per capita recovery rate for humans from the infectious state to the recovered state |
| $\omega$ | Per capita rate of loss of immunity in humans |
| $\nu_h$ | Proportion of antibody produced by humans in response to the incidence of infection caused by mosquitoes |
| $\nu_m$ | Proportion of antibody produced by mosquito in response to the incidence of infection caused be humans |
| $e$ | Sensitization rate of susceptible humans against the spread of malaria |
| $a$ | Rate of loss of protection |
| $n$ | Natural recovery rate of humans from the infectious state |

Thus, incorporating the natural recovery parameter, $n$ to Musa S. & Goni A. N (2018) malaria model equation leads to the proposed model equations below

$$\frac{dS_h}{dt} = \Lambda_h - \frac{b\beta_h S_h(t) I_m(t)}{1+\nu_h I_m(t)} - \mu_h S_h(t) - eS_h(t) + nI_h + \omega R_h(t) + aP_h \tag{1}$$

$$\frac{dP_h}{dt} = eS_h(t) - \mu_h P_h(t) - aP_h \tag{2}$$

$$\frac{dE_h}{dt} = \frac{b\beta_h S_h(t) I_m(t)}{1+\nu_h I_m(t)} - (\alpha_h + \mu_h) E_h(t) \tag{3}$$

$$\frac{dI_h}{dt} = \alpha_h E_h(t) - (r + n + \mu_h + \delta_h) I_h(t) \tag{4}$$

$$\frac{dR_h}{dt} = rI_h(t) - (\mu_h + \omega) R_h(t) \tag{5}$$

$$\frac{dS_m}{dt} = \Lambda_m - \frac{b\beta_m S_m(t) I_h(t)}{1+\nu_m I_h(t)} - \mu_m S_m(t) \tag{6}$$

$$\frac{dE_m}{dt} = \frac{b\beta_m S_m(t) I_m(t)}{1+\nu_m I_h(t)} - (\alpha_m + \mu_m) E_m(t) \tag{7}$$

$$\frac{dI_m}{dt} = \alpha_m E_m(t) - (\mu_m + \delta_m) I_m(t) \tag{8}$$

Together with the initial conditions:

$S_h(0) = S_{0h}$, $P_h(0) = P_{oh}$, $E_h(0) = E_{0h}$, $I_h(0) = I_{0h}$, $R_h(0) = R_{0h}$, $S_m(0) = S_{0m}$, $E_m(0) = E_{0m}$, $I_m(0) = I_{0m}$

**Stability Analysis**

Disease Free Equilibrium

The equilibrium state is determined by setting the right-hand side of the system (1)–(8) to zero.

In the absence of the disease, $E_h = 0, I_h = 0, R_h = 0, E_m = 0, I_m = 0$, where each variable represents the steady-state values of the respective population classes. The system reaches a disease-free equilibrium (DFE), denoted as

$$E_0 = \left\{ \frac{2\Lambda_h ae + \Lambda_h(\mu_h - a)(\mu_h + e)}{ae(\mu_h + e) + (\mu_h - a)(\mu_h + e)^2}, \frac{e\Lambda_h}{(\mu_h + a)(\mu_h + e) - ae}, 0, 0, 0, \frac{\Lambda_m}{\mu_m}, 0, 0 \right\} \tag{9}$$

*Basic Reproduction Number*

The basic reproduction number, $R_0$ is a key parameter in analyzing the behavior of epidemiological models. It represents the expected number of secondary infections generated by a single infectious individual in a fully susceptible population. This threshold parameter determines whether an infection will persist or be eradicated within a given population: If $R_0 < 1$, the disease will die out over time. If $R_0 > 1$, the disease will spread within the population. To derive $R_0$, we apply the Next Generation Matrix (NGM) method proposed by Diekmann and Heesterbeek (2000). This approach focuses on the infected compartments of the system (1)–(8), specifically examining the following equations (3), (4), (7) and (8). By constructing the transmission and transition matrices, the

dominant eigenvalue of the next-generation matrix gives the basic reproduction number $R_0$ which serves as an important indicator of disease dynamics in the population.

Let $F_i$ be the rate of appearance of new infection in the $i$ compartment and $V_i$ be the rate of transfer of individuals out of $i$, given the disease-free equilibrium, then $R_0$ is the spectral radius (largest eigenvalue) of the next generation matrix denoted by $G = FV^{-1}$

$\frac{dx}{dt} = F_i(x) - V_i(x)$, where

$$F_i(x) = \begin{pmatrix} F_1 \\ F_2 \\ F_3 \\ F_4 \end{pmatrix} = \begin{pmatrix} \frac{b\beta_h S_h I_m}{1+\nu_h I_m} \\ 0 \\ \frac{b\beta_m S_m I_h}{1+\nu_m I_h} \\ 0 \end{pmatrix} \quad \text{and} \quad V_i(x) = \begin{pmatrix} V_1 \\ V_2 \\ V_3 \\ V_4 \end{pmatrix} = \begin{pmatrix} (\alpha_h + \mu_h)E_h \\ (r+\delta_h+n+\mu_h)I_h - \alpha_h E_h \\ (\alpha_m+\mu_m)E_m \\ (\mu_m+\delta_m)I_m - \alpha_m E_m \end{pmatrix}$$

Hence, evaluating $G = FV^{-1}$ at the disease–free equilibrium, $E_0$ and simplifying the characteristics equation, $\left|FV^{-1} - \lambda I\right| = 0$ for the largest eigenvalue of the next-generation matrix gives the basic reproduction number

$$R_0 = \sqrt{\frac{b^2\alpha_h\beta_h\Lambda_h\alpha_m\beta_m\Lambda_m[(\mu_h+a)(\mu_h+e)-2ae]}{(\alpha_h+\mu_h)(r+n+\delta_h+\mu_h)(\alpha_m+\mu_m)(\mu_m+\delta_m)\mu_m[(\mu_h+a)(\mu_h+e)^2 - ae(\mu_h+e)]}} \quad (10)$$

*Local Stability of the Disease Free Equilibrium*

*Theorem*

The disease-free equilibrium point, $E_0$, is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof:

We let

$$X_1 = \Lambda_h - \frac{b\beta_h S_h(t) I_m(t)}{1+\nu_h I_m(t)} - \mu_h S_h(t) - eS_h(t) + rI_h + \omega R_h(t) + aP_h \quad (11)$$

$$X_2 = eS_h - \mu_h P_h - aP_h \quad (12)$$

(13)

$$X_4 = \alpha_h E_h(t) - (r + n + \mu_h + \delta_h) I_h(t) \tag{14}$$

$$X_5 = r I_h(t) - (\mu_h + \omega) R_h(t) \tag{15}$$

$$X_6 = \Lambda_m - \frac{b\beta_m S_m(t) I_h(t)}{1 + \nu_m I_h(t)} - \mu_m S_m(t) \tag{16}$$

$$X_7 = \frac{b\beta_m S_m(t) I_m(t)}{1 + \nu_m I_h(t)} - (\alpha_m + \mu_m) E_m(t) \tag{17}$$

$$X_8 = \alpha_m E_m(t) - (\mu_m + \delta_m) I_m(t) \tag{18}$$

The Jacobian matrix of the system (11)-(18) evaluated at the disease-free equilibrium as illustrated below

$$J(E_0) = \begin{pmatrix} J_{11} & J_{12} & 0 & 0 & J_{15} & 0 & 0 & J_{18} \\ J_{21} & J_{22} & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & J_{33} & 0 & 0 & 0 & 0 & J_{38} \\ 0 & 0 & J_{43} & J_{44} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & J_{54} & J_{55} & 0 & 0 & 0 \\ 0 & 0 & 0 & J_{64} & 0 & J_{66} & 0 & 0 \\ 0 & 0 & 0 & J_{74} & 0 & 0 & J_{77} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & J_{87} & J_{88} \end{pmatrix} \tag{19}$$

Where,

$$J_{11} = -(\mu_h + e), \quad J_{12} = a, \quad J_{15} = \omega, \quad J_{18} = -\frac{b\beta_h \Lambda_h[(\mu_h + a)(\mu_h + e) - 2ae]}{(\mu_h + a)(\mu_h + e)^2 - ae(\mu_h + e)}, \quad J_{21} = e,$$

$$J_{22} = -(\mu_h + a), \quad J_{33} = -(\alpha_h + \mu_h), \quad J_{38} = \frac{b\beta_h \Lambda_h[(\mu_h + a)(\mu_h + e) - 2ae]}{(\mu_h + a)(\mu_h + e)^2 - ae(\mu_h + e)}, \quad J_{43} = \alpha_h,$$

$$J_{44} = -(r + n + \mu_h + \delta_h), \quad J_{54} = r, \quad J_{55} = -(\mu_h + \omega), \quad J_{64} = -\frac{b\beta_m \Lambda_m}{\mu_m}, \quad J_{66} = -\mu_m,$$

$$J_{74} = \frac{b\beta_m \Lambda_m}{\mu_m}, \quad J_{77} = -(\alpha_m + \mu_m), \quad J_{87} = \alpha_m, \quad J_{88} = -(\mu_m + \delta_m)$$

yields the characteristic equation

$$(J_{55}-\lambda)[(J_{11}-\lambda)(J_{22}-\lambda)(J_{33}-\lambda)(J_{44}-\lambda)(J_{77}-\lambda)(J_{88}-\lambda)-(J_{11}-\lambda)(J_{22}-\lambda)J_{38}J_{43}J_{74}J_{87} - J_{12}J_{21}(J_{33}-\lambda)(J_{44}-\lambda)(J_{77}-\lambda)(J_{88}-\lambda)-J_{12}J_{21}J_{38}J_{43}J_{74}J_{87}]=0 \quad (20)$$

Now let

$$B_1=\mu_h+e, B_2=\mu_h+a, B_3=\alpha_h+\mu_h, B_4=r+n+\mu_h+\delta_h, B_5=\alpha_m+\mu_m, B_6=\mu_m+\delta_m$$

Thus, this becomes

$$(\lambda+B_1)(\lambda+B_2)(\lambda+B_3)(\lambda+B_4)(\lambda+B_5)(\lambda+B_6)+(\lambda+B_1)(\lambda+B_2)J_{38}J_{43}J_{74}J_{87} + (\lambda+B_3)(\lambda+B_4)(\lambda+B_5)(\lambda+B_6)J_{12}J_{21}-J_{12}J_{21}J_{38}J_{43}J_{74}J_{87}=0 \quad (21)$$

Therefore, expanding and simplifying (21) yields

$$A_6\lambda^6+A_5\lambda^5+A_4\lambda^4+A_3\lambda^3+A_2\lambda^2+A_1\lambda+A_0=0 \quad (22)$$

Routh-Hurwitz criterion is applied which states that all roots of the polynomial (22) have negative real parts if and only if the coefficients, $A_i$, are positive and the determinants of the matrices, $H_i>0$; for values of $i=0,1,2,3,4,5,6$.

Hence, we observe from equation (22) that $A_1>0,\ A_2>0,\ A_3>0,\ A_4>0,\ A_5>0,$ $A_6>0$ since $B_1,B_2,B_3,B_4,B_5,B_6$ are all positive. This implies that, $H_1=A_5>.0$

$$H_2=\begin{vmatrix}A_5 & A_6\\ A_3 & A_4\end{vmatrix}=A_4A_5-A_3A_6=A_4A_5-A_3>0$$

$$H_3=\begin{vmatrix}A_5 & A_6 & 0\\ A_3 & A_4 & A_5\\ A_1 & A_2 & A_3\end{vmatrix}=\begin{vmatrix}A_5 & 1 & 0\\ A_3 & A_4 & A_5\\ A_1 & A_2 & A_3\end{vmatrix}=A_5(A_3A_4-A_2A_5)-(A_3{}^2-A_1A_5)>0$$

$$H_4=\begin{vmatrix}A_5 & 1 & 0 & 0\\ A_3 & A_4 & A_5 & 1\\ A_1 & A_2 & A_3 & A_4\\ 0 & A_0 & A_1 & A_2\end{vmatrix}>0$$

$$H_5 = \begin{vmatrix} A_5 & 1 & 0 & 0 & 0 \\ A_3 & A_4 & A_5 & 1 & 0 \\ A_1 & A_2 & A_3 & A_4 & A_5 \\ 0 & A_0 & A_1 & A_2 & A_3 \\ 0 & 0 & 0 & A_0 & A_1 \end{vmatrix} > 0 \quad \text{and} \quad H_6 = \begin{vmatrix} A_5 & 1 & 0 & 0 & 0 & 0 \\ A_3 & A_4 & A_5 & 1 & 0 & 0 \\ A_1 & A_2 & A_3 & A_4 & A_5 & 1 \\ 0 & A_0 & A_1 & A_2 & A_3 & A_4 \\ 0 & 0 & 0 & A_0 & A_1 & A_2 \\ 0 & 0 & 0 & 0 & 0 & A_0 \end{vmatrix} > 0$$

Thus, all eigenvalues of the polynomial (22) have negative real parts. That is, $\lambda_3 < 0$, $\lambda_4 < 0$, $\lambda_5 < 0$, $\lambda_6 < 0$. We clearly observe that $\lambda_i < 0$, for $i = 1,2,3,4,5,6,7,8$ when $R_0 < 1$ and therefore, conclude that the disease-free equilibrium point is locally asymptotically stable. On the other hand, if $R_0 > 1$, we notice that $A_0 < 0$ , if $B_1B_2B_3B_4B_5B_6 < B_3B_4B_5B_6ae$. By Descartes' rule of signs, there is exactly one sign change in the sequence, $A_6, A_5, A_4, A_3, A_2, A_1, A_0$. of the coefficients of the polynomial (22). This is an indication that, one eigenvalue exists with a positive real part. Hence, the disease-free equilibrium point becomes unstable.

**Numerical Experiment**

We carry out some numerical experiments using Maple to study the effects of the added parameter on the model when varied

Table 3. Parameter values and initial variables (in fraction) and sources

| | | |
|---|---|---|
| $\Lambda_h$ | 0.000215 | Olaniyi & Obabiyi (2013) |
| $\Lambda_m$ | 0.007 | Olaniyi & Obabiyi (2013) |
| $b$ | 0.12 | Olaniyi & Obabiyi (2013) |
| $\beta_h$ | 0.1 | Olaniyi & Obabiyi (2013) |
| $\beta_m$ | 0.09 | Olaniyi & Obabiyi (2013) |
| $\mu_h$ | 0.0000548 | Olaniyi & Obabiyi (2013) |
| $\mu_m$ | 1/15 | Olaniyi & Obabiyi (2013) |
| $\delta_h$ | 0.001 | Olaniyi & Obabiyi (2013) |
| $\delta_m$ | 0.01 | Olaniyi & Obabiyi (2013) |
| $\alpha_h$ | 1/17 | Olaniyi & Obabiyi (2013) |
| $\alpha_m$ | 1/18 | Olaniyi & Obabiyi (2013) |
| $r$ | 0.05 | Olaniyi & Obabiyi (2013) |
| $\omega$ | 1/730 | Olaniyi & Obabiyi (2013) |

| | | |
|---|---|---|
| $v_h$ | 0 | Olaniyi & Obabiyi (2013) |
| $v_m$ | 0 | Olaniyi & Obabiyi (2013) |
| $e$ | 0.2, 0.4,…., 1.0 | Assumed |
| $a$ | 0.04 | Olaniyi & Obabiyi (2013) |
| $n$ | 0.2, 0.4,…, 1.0 | Assumed |
| $S_h(0)$ | 1.00 | Olaniyi & Obabiyi (2013) |
| $E_h(0)$ | 0,20 | Olaniyi & Obabiyi (2013) |
| $I_h(0)$ | 0.10 | Olaniyi & Obabiyi (2013) |
| $R_h(0)$ | 0.0 | Olaniyi & Obabiyi (2013) |
| $S_m(0)$ | 0.1000 | Olaniyi & Obabiyi (2013) |
| $E_m(0)$ | 0,20 | Olaniyi & Obabiyi (2013) |
| $I_m(0)$ | 0.30 | Olaniyi & Obabiyi (2013) |
| $P_h(0)$ | 0.5 | Olaniyi & Obabiyi (2013) |

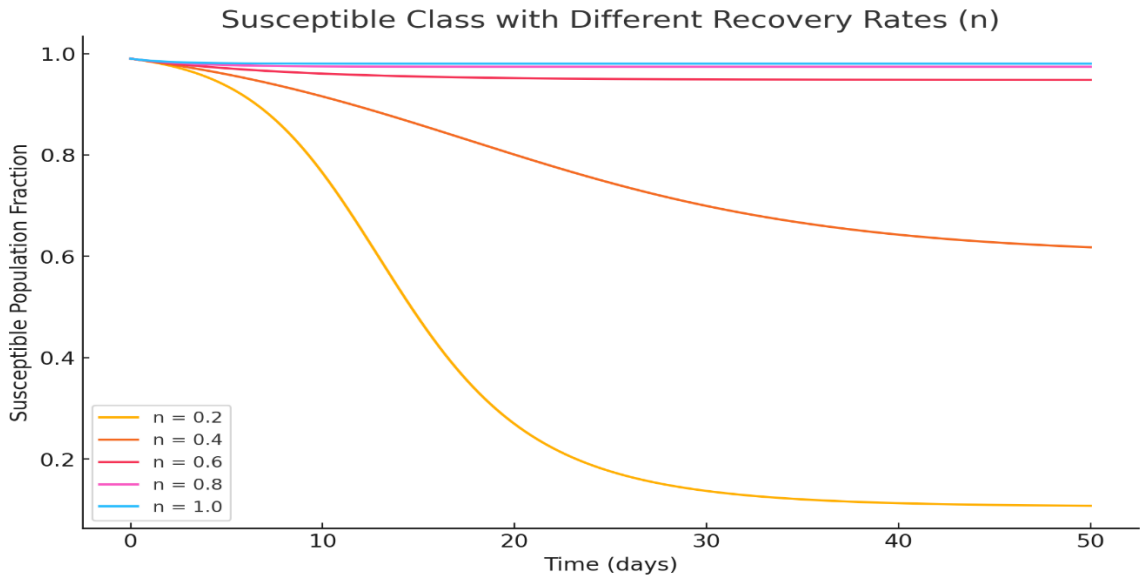


Fig 1

The graph shows the susceptible class (*S*) over time for different natural recovery rates, *n* where *n*=0.2, 0.4, 0.6, 0.8, &1. The number of susceptibles (*S*) decreases as more people get infected. Higher natural recovery rates, *n* slow down the depletion of susceptibles, as infections clear up faster, reducing further transmission. Lower natural recovery rates (*n=0.2*) cause a faster decline in (*S*), since infected individuals remain contagious longer.

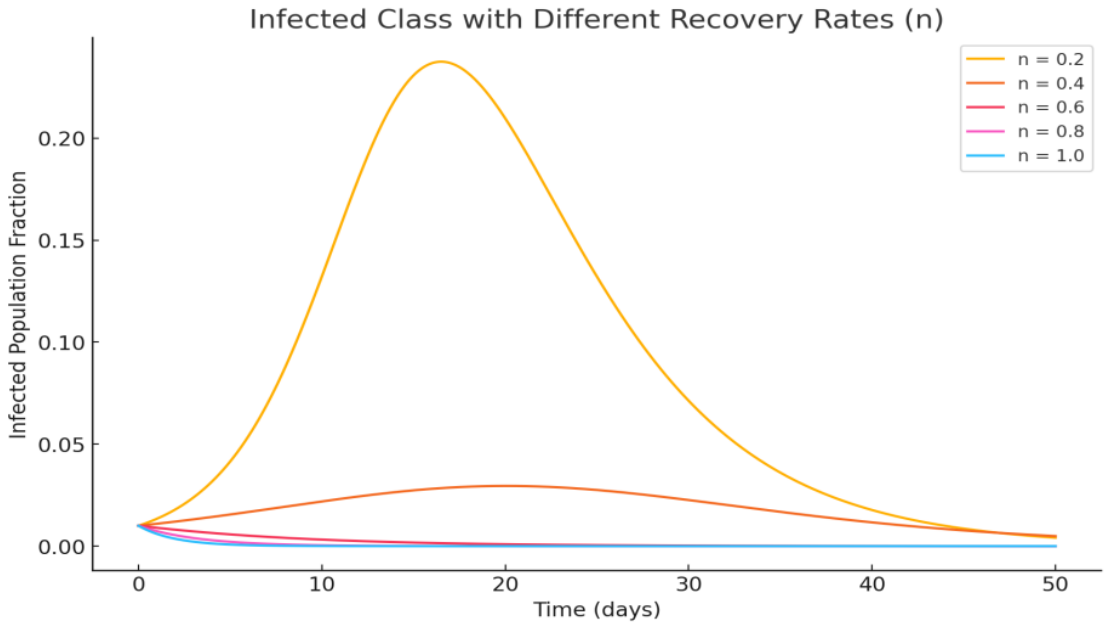


Fig 2

The graph shows the infected class ($I$) over time for different natural recovery rates, $n$ where $n$ = 0.2, 0.4, 0.6, 0.8 & 1.0. Higher natural recovery rates, $n$ lead to a faster decline in infections. Lower natural recovery rates ($n$ = 0.2) result in a higher peak and longer duration of infection since infected individuals remain contagious for a longer time. When $n$ = 1, infections clear up the fastest, leading to the lowest infection peak. This demonstrates how increasing the recovery rate (e.g., through treatment) can significantly reduce malaria infections

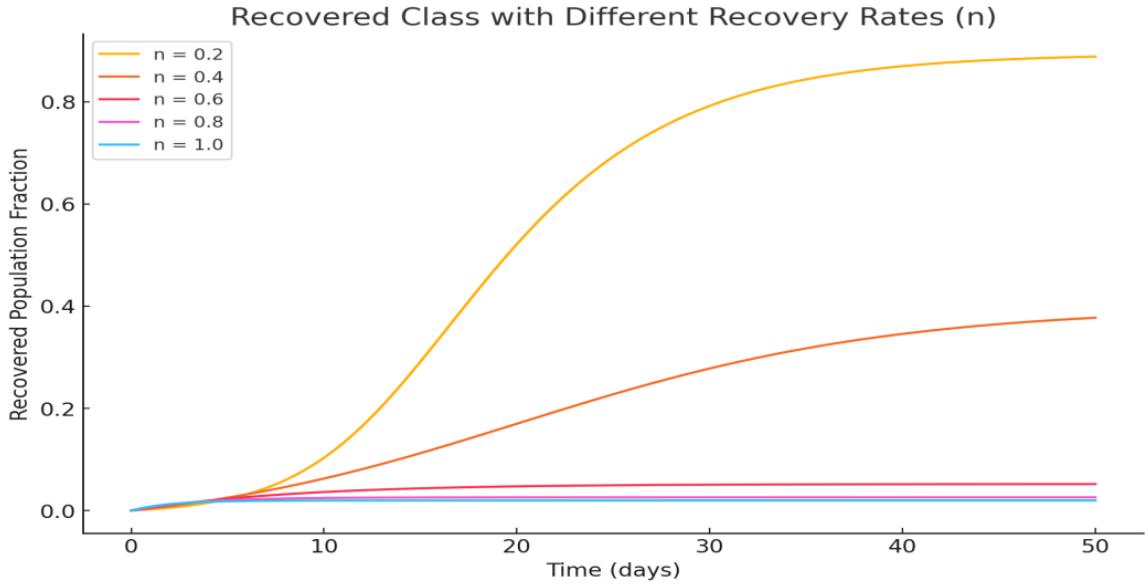


Fig 3

The graph shows the recovered class ($R$) over time for different natural recovery rates, $n$ where $n$ = 0.2, 0.4, 0.6, 0.8 & 1.0. Higher recovery rates, $n$ result in a faster accumulation of recovered individuals. Lower natural recovery rates ($n$ = 0.2) cause a slower rise in the recovered population since infected individuals take longer to recover. When $n$ = 1, the recovered class grows the fastest, meaning more people clear the infection quickly.

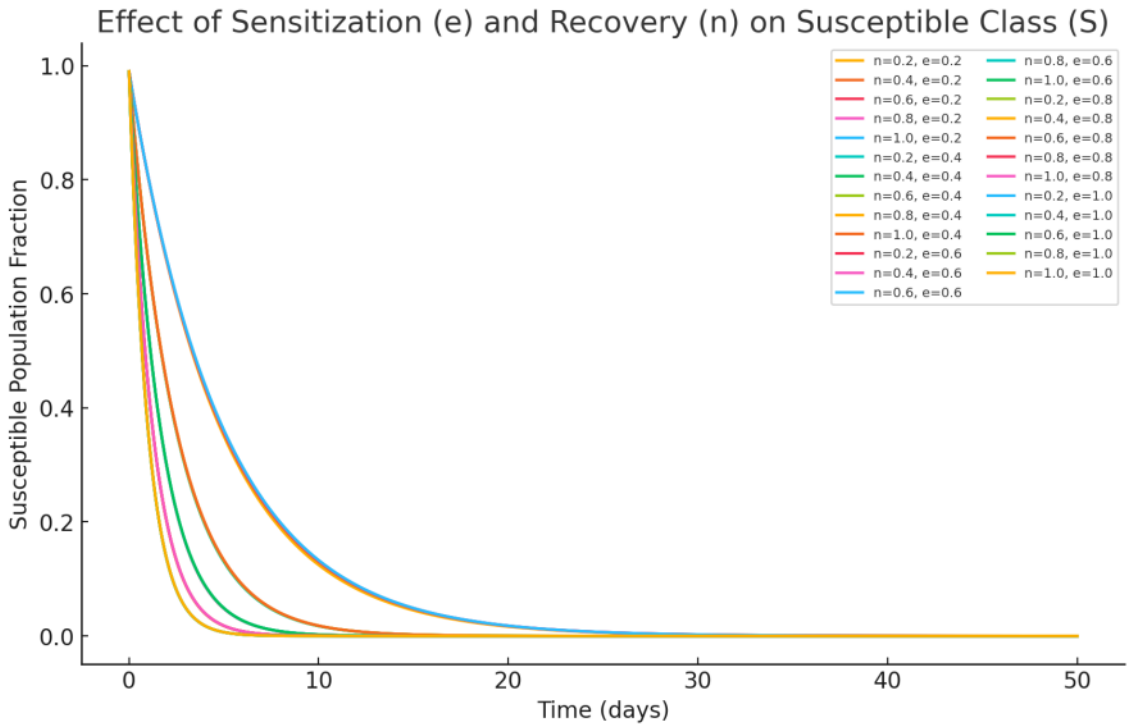


Fig 4

The graph shows how the susceptible class ($S$) changes over time for different combinations of:

a. Sensitization rates, $e$ which reduce the number of susceptibles by moving them directly to the recovered class.

b. Natural recovery rates, $n$ which indirectly influence ($S$) by affecting the number of infected individuals.

Also in fig 4 above, higher sensitization rates, $e$ cause a faster decline in susceptibles since more people take preventive measures. Higher natural recovery rates, n slow down the depletion of susceptibles by reducing the infection period. When both $e$ and $n$ are high, the susceptible class drops sharply and quickly, leading to better malaria control.

## Conclusion

This study analyzed a mathematical model comprising an 8-dimensional system of ordinary differential equations to describe the transmission dynamics of malaria. The Disease-Free Equilibrium (DFE) of the system (1)–(8) was established and is represented by equation (9). Additionally, the basic reproduction number $R_0$ was derived using the Next Generation Matrix method and is given by equation (10).

To assess the stability of the Disease-Free Equilibrium, the Routh-Hurwitz criterion was applied. The analysis showed that all eigenvalues are negative, i.e $\lambda_i < 0$ for $i = 1,2,3,4,5,6,7,8$ implying that the disease free equilibrium is locally asymptotically stable when $R_0 < 1$. This clearly shows that malaria can be eradicated from the population over time if control measures are effectively implemented.

However, controlling malaria remains a challenge due to the possibility of protected individuals becoming susceptible after losing immunity and being exposed to infected mosquito bites. Analytical results indicate that the DFE remains locally asymptotically stable when $R_0 < 1$, highlighting the crucial role of sensitization in malaria elimination.

To achieve a malaria-free society, governments, public health organizations, and other stakeholders should prioritize massive and continuous sensitization to raise awareness about malaria prevention. Public enlightenment campaigns should incorporate incentives to encourage positive changes, ultimately contributing to the successful eradication of malaria in affected communities.

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## References

Adamu, A. K. and Kimbir, A. R. (2013). Modeling the Epidemiology of Malaria and Control with Estimate of the Basic Reproduction Number, Pure and Applied Mathematics Journal. Vol.2, pp. 42-50.

Adamu, A. K., Ochigbo, J., Williams, B and Okorie, C. (2017), Local Stability Analysis of a Susceptible Protected Infected Treated Recovered (SPITR) Mathematical Model for Malaria Disase Dynamics. FUW Trends in Science & Technology Journal, www.ftstjournal.com e-ISSN: 24085162; p-ISSN: 20485170: Vol. 2 No. 1A, pp. 169-174.

Chitnis N., (2005). Using Mathematical Models in Controlling the Spread of Malaria, Ph.D. thesis, Program in Applied Mathematics, University of Arizona, Tucson, AZ

Diekmann, O. and Heesterbeek J. A. P. (2000). Mathematical Epidemiology of Infectious Diseases: "Application of Matrix Theory to Model Building, Analysis and Interpretation" JAMA 271(9): 698-702

Doolan, D. L., Dobano, C., & Baird, J. K. (2009). Acquired immunity to malaria. *Clinical Microbiology Reviews, 22*(1), 13-36.

Goni, A. N., (2016), Mathematical Model for the Control of Malaria using Education-Based Intervention, M.Sc. Research Project, Modibbo Adama University of Technology Yola

Langhorne, J., Ndungu, F. M., Sponaas, A. M., & Marsh, K. (2008). Immunity to malaria: More questions than answers. *Nature Immunology, 9*(7), 725-732.

Marsh, K., & Kinyanjui, S. (2006). Immune effector mechanisms in malaria. *Parasite Immunology, 28*(1-2), 51-60.

Miller, L. H., Baruch, D. I., Marsh, K., & Doumbo, O. K. (2002). The pathogenic basis of malaria. *Nature, 415*(6872), 673-679.

Brian M. Greenwood, David A. Fidock, Dennis E. Kyle, Stefan H. I. Kappe, Pedro L. Alonso, Frank H. Collins, Patrick E. Duffy. (2008). Malaria: Progress, perils, and prospects for eradication. *Journal of Clinical Investigation, 118*(4), 1266-1276.

Musa S. and Goni A. N., (2018). Modelling the Effect of Education-Based Intervention in the Control of Malaria, Science World Journal Vol. 13 (No. 4), pp. 1-7; www.scienceworldjournal.org

Olaniyi S., and Obabiyi O. S. (2013). Mathematical Model for Malaria Transmission Dynamics In Human and Mosquito Populations with Nonlinear Forces of Infection, International Journal of Pure and Applied Mathematics, Volume 88 No. 1, 125-156. ISSN: 1314-3395 (on-line version), url: http://www.ijpam.eu

Olliaro, P. (2001). Mode of action and mechanisms of resistance for antimalarial drugs. *Pharmacology & Therapeutics, 89*(2), 207-219.

Peter, M. M. M., (2010), Modelling the Effects of Multi-Intervention Campaigns for the Malaria Epidemic in Malawi, M.Sc. Thesis, University of Da res Salaam

Schofield, L., & Grau, G. E. (2005). Immunological processes in malaria pathogenesis. *Nature Reviews Immunology, 5*(9), 722-735.

Smith R. J. and Hove-Musekwa D. S. (2008), Determining effective spraying periods to control malaria via indoor residual spraying in Sub-Saharan Africa. Apllied Maths and Deci Sc. doi:10, Volume 1155. No.74563, Pp 1-19

WHO (2018). World Malaria Report 2018. *World Health Organization.*

Greenwood, B. M., Fidock, D. A., Kyle, D. E., Kappe, S. H., Alonso, P. L., & Collins, F. H. (2014). Malaria. *The Lancet, 383*(9918), 723-735.

Williams, T. N. (2006). Human red blood cell polymorphisms and malaria. *Current Opinion in Microbiology, 9*(4), 388-394.

World Health Organization (2016). Fact Sheet on Malaria. Electronic, www.who.int/mediacentre/factsheets/fs094/en/

World Health Organization (2018), World malaria report, WHO Press, Switzerland. http//www.who.int/malaria

Yang H. M., (2000). Malaria transmission model for different levels of acquired immunity and temperature-dependent parameters (vector), Revista de SatudePublica, 34, pp. 223{231.