Mikailalsys Journal of Mathematics and Statistics

e-ISSN : 3030-816X
p-ISSN : 3030-8399

Index : Harvard, Boston, Sydney University, Dimensions, Lens, Scilit, Semantic, Google, etc

https://doi.org/10.58578/MJMS.v3i1.4985

A Mathematical Model for Malaria Disease Dynamics with Relapse Parameter

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Article Info:

Submitted:	Revised:	Accepted:	Published:
Jan 20, 2025	Feb 3, 2025	Feb 15, 2025	Feb 20, 2025

Abstract

Malaria is one of the oldest diseases that has been extensively researched from multiple perspectives. Although many infectious diseases, including malaria, are preventable, they remain widespread in numerous communities due to insufficient, delayed, or ineffective control measures. Effective disease control involves rapidly reducing the infected population when a cure is available and minimizing susceptibility through vaccination when possible. Since malaria vaccines are still under development, vaccination offers a potential strategy for reducing the number of susceptible individuals. In this paper, we have analyzed and modified the SPITR mathematical model by Adamu et al. (2017) to study the transmission and control of malaria. Our modifications include the incorporation of a relapse parameter, and we have determined the basic reproduction number for the revised model. We demonstrated that the disease-free equilibrium (DFE) is locally asymptotically stable when the reproduction number is less than one and becomes unstable when it exceeds one. This finding suggests that with a combination of effective treatment, malaria relapse rate can be reduced and malaria in general can be effectively controlled in the population if the reproduction number is kept below unity.

Keywords: Stability, Jacobian Matrix, Next Generation Matrix, Disease Free Equilibrium, Basic Reproduction Number

Introduction

Malaria, previously known as ague fever, is one of the most widespread infectious diseases and remains a significant global health concern (K. Marsh, 1998). It is caused by single-celled parasites of the *Plasmodium* genus, with five species identified as infecting humans: *Plasmodium vivax, Plasmodium malariae, Plasmodium falciparum, Plasmodium knowlesi*, and *Plasmodium ovale*. Among these, *Plasmodium falciparum* presents the greatest threat to non-immune individuals, accounting for approximately 80% of cases and 90% of malaria-related deaths (Kakkilaya, 2003). Children under five and pregnant women are particularly vulnerable, as pregnancy weakens the immune system, increasing susceptibility to infection. Currently, malaria affects over 300 million people worldwide and causes an estimated 1.5 to 3.0 million deaths annually (Ngwa and Shu, 2000; Malaria, 2010; Chitnis, 2004; Cushings and Hyman, 2006). Malaria is transmitted by the bite of an infected female Anopheles mosquito whenever the infected mosquito feeds on blood meal. The symptoms of malaria disease include fever, chills together with headache, vomiting flu-like, anemia (destroying red blood cell), diarrhea, liver and neurological damage. Malaria is endemic in tropical areas where climate and weather conditions allow continuous breeding of the mosquito.

In recent years, various control strategies and intervention programs have been implemented globally to combat malaria. These include the ongoing development of anti-malaria vaccines, insecticide-treated bed nets (ITNs), indoor residual spraying (IRS), environmental management to reduce mosquito breeding, and biological control methods. These measures are widely used in malaria-endemic regions, particularly in Sub-Saharan Africa, and have contributed to a decline in disease transmission.

Mathematical models play a vital role in malaria control. Their application dates back to Ross (1911), who demonstrated that reducing the mosquito population below a certain threshold could eliminate malaria. This threshold depends on biological factors such as biting rates and vectorial capacity. Macdonald (1957) later developed a model to estimate infection and recovery rates, assuming that exposure to infective material remained constant. Further epidemiological models by Aron and May (1982) and Anderson and May

(1991) incorporated the idea that acquired immunity to malaria is strengthened through repeated exposure to the parasite.

In this study, we modify the SPITR model of Adamu et al. (2017) by introducing a relapse parameter to determine its impact on the transmission dynamics of malaria.

Malaria Treatment Relapse: Challenges and Solutions

Malaria remains one of the most significant infectious diseases worldwide, affecting millions of people annually. Despite advancements in treatment, malaria relapse continues to pose a major challenge, particularly in cases caused by *Plasmodium vivax* and *Plasmodium ovale*. Relapse occurs when dormant liver-stage parasites, known as hypnozoites, reactivate after an initial infection has been cleared, leading to recurrent episodes of the disease. Understanding the causes, risk factors, and strategies for preventing malaria relapse is crucial in achieving long-term disease control and eventual eradication.

Causes and Mechanism of Relapse

Unlike *Plasmodium falciparum*, which causes the most severe form of malaria but does not relapse, *P. vivax* and *P. ovale* have a dormant stage in the liver that can remain inactive for weeks, months, or even years before reactivating and causing new infections (White, 2011). The exact triggers for hypnozoite activation remain unclear, but factors such as immune suppression, coinfections, and environmental conditions have been implicated (Baird, 2013).

Treatment Challenges

The standard treatment for malaria involves artemisinin-based combination therapies (ACTs), which are highly effective against blood-stage parasites but do not eliminate hypnozoites. To prevent relapse, primaquine or tafenoquine, the only available drugs that target the liver-stage parasites, must be administered in combination with ACTs (Wells et al., 2010). However, the effectiveness of these drugs is hindered by challenges such as:

G6PD Deficiency: Both primaquine and tafenoquine can cause hemolysis in individuals with glucose-6-phosphate dehydrogenase (G6PD) deficiency, limiting their use in endemic populations (Howes et al., 2012).

Poor Compliance: Primaquine requires a 14-day regimen, and low adherence rates often lead to incomplete parasite clearance, increasing the risk of relapse (Bennett et al., 2013).

Drug Resistance: Emerging resistance to anti-malarial drugs, including ACTs, poses a threat to malaria control efforts (Ashley et al., 2014).

Strategies to Reduce Relapse

Efforts to improve malaria treatment outcomes include the development of shorter, more effective drug regimens, improved diagnostics for detecting hypnozoites, and integrated control strategies involving vector reduction and chemoprevention (Imwong et al., 2012). The introduction of tafenoquine, a single-dose alternative to primaquine, offers hope for improved adherence and better relapse prevention (Llanos-Cuentas et al., 2019).

Parameters and variables of the model

Table 1

Parameters and meaning	
ψ	Natural birth rate of humans
ρ	Natural birth rate of mosquito
θ_{mh}	Probability of transmission of malaria from infected mosquito to susceptible human
θ_{hm}	Probability of transmission of malaria from infected human to susceptible mosquito
$\alpha_h = \frac{\theta_{mh}\phi I_m}{N_h}$	Transfer rate of humans from susceptible to infected compartment
$\alpha_m = \frac{\theta_{hm}\phi I_h}{N_h}$	Transfer rate of mosquito from susceptible to infected compartment
μ_h	Natural death rate of humans
μ_v	Natural death rate of mosquitoes
δ_h	Death rate of humans due to malaria
r	Transfer rate of humans from infected to treated compartment
ε	Transfer rate of humans from treated to recovered compartment
τ	Transfer rate of humans from infected to recovered compartment
φ	Transfer rate of humans from recovered to susceptible compartment
γ	Fraction of natural birth rate of humans.
$(1-\gamma)\psi$	Rate at which newly born humans enter into susceptible compartment
$\gamma\psi$	Rate at which newly born humans enter into protected compartment

g	Transfer rate of humans from susceptible to protected compartment
υ	Vaccination rate on humans
k	Rate at which mosquitoes are killed
ϕ	Rate at which susceptible mosquitoes bite infected humans. (Infected mosquito also bites susceptible humans at the same rate)
ς	Relapse parameter

Table 2

Variables and description	
$S_h(t)$	the number of susceptible human host at time t
$P_h(t)$	the number of protected human host at time t
$I_h(t)$	the number of infected human host at time t
$T_h(t)$	the number of treated human host at time t
$R_h(t)$	the number of recovered human host at time t
$S_m(t)$	the number of susceptible mosquito vector at time t
$I_m(t)$	the number of infected mosquito vector at time t
$N_h(t)$	the total human population at time t
$N_m(t)$	the total mosquito population at time t

The model by Adamu *et al.* (2015) is given as

$$\frac{dS_h}{dt} = (1-\gamma)\psi - \frac{\theta_{mh}\phi I_m}{N_h} S_h - \mu_h S_h - gS_h + \varphi R_h \tag{1}$$

$$\frac{dP_h}{dt} = \gamma\psi + gS_h + (1-\varphi)R_h - \mu_h P_h \tag{2}$$

$$\frac{dI_h}{dt} = \frac{\theta_{mh}\phi I_m}{N_h} - \tau I_h - rI_h - \mu_h I_h - \delta_h I_h \tag{3}$$

$$\frac{dT_h}{dt} = rI_h - \varepsilon T_h - \mu_h T_h \tag{4}$$

$$\frac{dR_h}{dt} = \varepsilon T_h + \tau I_h - (1-\varphi)R_h - \varphi R_h - \mu_h R_h \tag{5}$$

$$\frac{dS_m}{dt} = \rho - \frac{\theta_{hm}\phi I_h}{N_h} S_m - \mu_m S_m \tag{6}$$

$$\frac{dI_m}{dt} = \frac{\theta_{hm}\phi I_h}{N_h} S_m - \mu_m I_m \tag{7}$$

We assumed that some infected humans who were inadequately treated are reinfected. As a result, the inclusion of a relapse parameter ς gives the modified model below.

$$\frac{dS_h}{dt} = (1-\gamma)\psi - \frac{\theta_{mh}\phi I_m}{N_h} S_h - \mu_h S_h - gS_h + \varphi R_h - \upsilon S_h \tag{8}$$

$$\frac{dP_h}{dt} = \gamma\psi + gS_h + (1-\varphi)R_h - \mu_h P_h + \upsilon S_h \tag{9}$$

$$\frac{dI_h}{dt} = \frac{\theta_{mh}\phi I_m}{N_h} - \tau I_h - rI_h - \mu_h I_h - \delta_h I_h + \varsigma R_h \tag{10}$$

$$\frac{dT_h}{dt} = rI_h - \varepsilon T_h - \mu_h T_h \tag{11}$$

$$\frac{dR_h}{dt} = \varepsilon T_h + \tau I_h - (1-\varphi)R_h - \varphi R_h - \mu_h R_h - \varsigma R_h \tag{12}$$

$$\frac{dS_m}{dt} = \rho - \frac{\theta_{hm}\phi I_h}{N_h} S_m - \mu_m S_m - kS_m \tag{13}$$

$$\frac{dI_m}{dt} = \frac{\theta_{hm}\phi I_h}{N_h} S_m - \mu_m I_m - kI_m \tag{14}$$

The total population sizes N_h and N_m can be determined by $S_h + P_h + I_h + T_h + R_h = N_h$ and $S_m + I_m = N_m$. The initial conditions of the system of equations (8) - (14) are given by $S_h(0) = S_{h0}$, $P_h(0) = P_{h0}$, $I_h(0) = I_{h0}$, $T_h(0) = T_{h0}$, $R_h(0) = R_{h0}$, $S_m(0) = S_{m0}$, $I_m(0) = I_{m0}$.

Considering the total human population, we have $\frac{dN_h}{dt} = \psi - \mu_h N_h - \delta_h I_h$. When the term $\delta_h I_h$ vanishes in equation (8) - (14), we obtain the solution of $\frac{dN_h}{dt} = \psi - \mu_h N_h$ to be

$N_h(t) = \frac{\psi}{\mu_h} + [N_{h0} - (\psi/\mu_h)]e^{-\mu_h t}$ showing that $N_h(t) \to \frac{\psi}{\mu_h}$ as $t \to 0$. Similarly, on summing up the total mosquito population, we have $\frac{dN_m}{dt} = \rho - N_m(\mu_m + k)$ which yields the solution, $N_m(t) = \frac{\rho}{\mu_m} + [N_{m0} - [\rho/(\mu_m + k)]]e^{-\mu_m t}$ showing that $N_m(t) \to \frac{\rho}{(\mu_m + k)}$ as $t \to 0$.

Disease free equilibrium point

The disease-free equilibrium (DFE) point $E_o = (S_h^*, P_h^*, I_h^*, T_h^*, R_h^*, S_m^*, I_m^*)$ represents a steady-state solution where the disease is absent in both the human and mosquito populations. It is obtained by setting the right-hand side of equations (8) to (14) to zero and solving for equilibrium values. This process determines the state where no individuals are infected, either in the human host or the mosquito vector. Evaluating these conditions yields the equilibrium point, which serves as a critical reference for analyzing the stability of the system and understanding the potential for malaria elimination within a given population.

$$E_o = \left(\frac{\psi(1-\gamma)}{\mu_h + g + \upsilon}, \frac{\psi(\upsilon + g + \gamma\mu_h)}{\mu_h(\mu_h + g + \upsilon)}, 0, 0, 0, \frac{\rho}{(\mu_m + k)}, 0 \right).$$

Basic Reproduction Number R_o

The threshold quantity is a crucial parameter in epidemiological models, commonly known as the basic reproduction number**.** It determines whether a disease will wipes out or escalate within a population.. The threshold quantity indicates the number of secondary infections produced by a single primary infection in a completely susceptible population (Hethcote, 2000). When $R_o < 1$, each infected individual averagely produces less than one new infected individual and as such, the disease dies out completely over time. On the other hand, if $R_o > 1$, each infected individual averagely produces more than one infected individual so that the disease spread and grow in the population, thus resulting in an epidemic. In the computation of R_o, using the next generation matrix, it is relevant to distinguish new infections from all other changes in the population. We identify I_h and I_m as the relevant classes for the computation of R_o. An infectious event increases these

classes (gain terms) and loses from these classes means loss of current or future infectious individual (loss terms). Listing the gain and loss terms (Table 3) for each class and creating a matrix (F) of gain terms and matrix (V) of loss terms with each evaluated at E_o, we have

Table 3

Classes	I_h	I_m
Gains	$\theta_{mh}\phi I_m S_h + \varsigma R_h$	$\theta_{hm}\phi I_h S_m$
Losses	$\left[\tau + r + (\mu_h + \delta_h) I_h\right]$	$(\mu_m + k) I_m$

$$F = \left[\begin{array}{cc} \frac{\partial}{\partial I_h}\left(\frac{\theta_{mh}\phi I_m S_h}{N_h} + \varsigma R_h\right) & \frac{\partial}{\partial I_h}\left(\frac{\theta_{hm}\phi I_h S_m}{N_h}\right) \\ \frac{\partial}{\partial I_m}\left(\frac{\theta_{mh}\phi I_m S_h}{N_h}\right) & \frac{\partial}{\partial I_m}\left(\frac{\theta_{hm}\phi I_h S_m}{N_h}\right) \end{array}\right]_{E_o} = \left[\begin{array}{cc} 0 & \frac{\theta_{mh}\phi}{N_h}\left(\frac{\rho}{\mu_m + k}\right) \\ \frac{\theta_{mh}\phi}{N_h}\left(\frac{\psi(1-\gamma)}{\mu_h + g + \upsilon}\right) & 0 \end{array}\right]$$

Similarly

$$V = \left[\begin{array}{cc} \frac{\partial}{\partial I_h}\left[\tau + r + (\mu_h + \delta_h)\right] I_h & \frac{\partial}{\partial I_h}(\mu_m + k) I_m \\ \frac{\partial}{\partial I_m}\left[\tau + r + (\mu_h + \delta_h)\right] I_h & \frac{\partial}{\partial I_m}(\mu_m + k) I_m \end{array}\right]_{E_o} = \left[\begin{array}{cc} \tau + r + (\mu_h + \delta_h) & 0 \\ 0 & \mu_m + k \end{array}\right]$$

Taking the inverse of V yields

$$V^{-1} = \left[\begin{array}{cc} \frac{1}{\tau + r + (\mu_h + \delta_h)} & 0 \\ 0 & \frac{1}{(\mu_m + k)} \end{array}\right] \text{ and evaluating } G = FV^{-1} \text{ gives}$$

$$G = \left[\begin{array}{cc} 0 & \frac{\rho\theta_{hm}\phi}{N_h(\mu_m + k)^2} \\ \frac{\psi(1-\gamma)\theta_{mh}\phi}{N_h(\mu_h + g + \upsilon)\left[\tau + r + (\mu_h + \delta_h)\right]} & 0 \end{array}\right].$$

R_o is the dormant eigenvalue, $\lambda_{\max}$ of G, with $\lambda_{\max} = \frac{T}{2} + \sqrt{\left(\frac{T^2}{2}\right) - D}$ where T and D are the trace and determinant of matrix G. Since $T = 0$, therefore

$$R_o = \lambda_{\max} = \sqrt{-D} = \sqrt{\frac{(1-\gamma)\theta_{mh}\theta_{hm}\phi^2\rho\mu_h^2}{\psi(\mu_m + k)^2(\mu_h + g + \upsilon)[\tau + r + (\mu_h + \delta_h)]}}. \quad (15)$$

Stability of the disease free equilibrium point

To establish the disease free equilibrium, the Jacobian matrix of equation (8) - (14) is computed and evaluated at E_o, thus yielding

$$J_{E_o} = \begin{pmatrix} -Q & 0 & 0 & 0 & \varphi & 0 & -\left(\frac{\theta_{mh}\phi\psi(1-\gamma)}{N_h Q}\right) \\ g+\upsilon & -\mu_h & 0 & 0 & 1-\varphi & 0 & 0 \\ 0 & 0 & -M & 0 & \varsigma & 0 & \left(\frac{\theta_{mh}\phi\psi(1-\gamma)}{N_h Q}\right) \\ 0 & 0 & r & -(\varepsilon+\mu_h) & 0 & 0 & 0 \\ 0 & 0 & \tau & \varepsilon & -(1+\mu_h+\varsigma) & 0 & 0 \\ 0 & 0 & -\left(\frac{\theta_{hm}\phi\rho}{N_h L}\right) & 0 & 0 & -L & 0 \\ 0 & 0 & \left(\frac{\theta_{hm}\phi\rho}{N_h L}\right) & 0 & 0 & 0 & -L \end{pmatrix},$$

where $Q = (\mu_h + g + \upsilon)$, $M = (\tau + r + \mu_h + \delta_h)$ and $L = (\mu_m + k)$.

To get the eigenvalues, we obtain the characteristic equation.

Thus,

$$\left|J_{E_O}-\lambda I\right|=\begin{vmatrix} -Q-\lambda & 0 & 0 & 0 & \varphi & 0 & -\left(\frac{\theta_{mh}\phi\psi(1-\gamma)}{N_hQ}\right) \\ g+\upsilon & -\mu_h-\lambda & 0 & 0 & 1-\varphi & 0 & 0 \\ 0 & 0 & -M-\lambda & 0 & \varsigma & 0 & \left(\frac{\theta_{mh}\phi\psi(1-\gamma)}{N_hQ}\right) \\ 0 & 0 & r & -(\varepsilon+\mu_h)-\lambda & 0 & 0 & 0 \\ 0 & 0 & \tau & \varepsilon & -(1+\mu_h+\varsigma)-\lambda & 0 & 0 \\ 0 & 0 & -\left(\frac{\theta_{hm}\phi\rho}{N_hL}\right) & 0 & 0 & -L-\lambda & 0 \\ 0 & 0 & \left(\frac{\theta_{hm}\phi\rho}{N_hL}\right) & 0 & 0 & 0 & -L-\lambda \end{vmatrix}$$

$$=\left[-\mu_h-\lambda\right]\left[-Q-\lambda\right]\left[-(\varepsilon+\mu_h)-\lambda\right]\left[-(1+\mu_h)-\lambda\right]\left[-L-\lambda\right]\begin{vmatrix} -M-\lambda & \left(\frac{\theta_{mh}\phi\psi(1-\gamma)}{N_hQ}\right) \\ \left(\frac{\theta_{hm}\phi\rho}{N_hL}\right) & -L-\lambda \end{vmatrix}=0$$

$$=\left[-\mu_h-\lambda\right]\left[-Q-\lambda\right]\left[-(\varepsilon+\mu_h)-\lambda\right]\left[-(1+\mu_h)-\lambda\right]\left[-L-\lambda\right]\left[\begin{matrix}\lambda^2+\left[M+L\right]\lambda+ML- \\ \frac{\theta_{mh}\theta_{hm}\phi^2\rho\psi(1-\gamma)}{N_h^2LQ}\end{matrix}\right]=0$$

so the eigenvalues of the characteristic equation are then given by

$$\left[-\mu_h\right],\left[-Q\right],\left[-(\varepsilon+\mu_h)\right],\left[-(1+\mu_h)\right],\left[-L\right],\frac{-\left[M+L\right]\pm\sqrt{\left[M+L\right]^2-4\left[ML-\frac{\theta_{mh}\theta_{hm}\phi^2\rho\psi(1-\gamma)}{N_h^2LQ}\right]}}{2}.$$

And from equation (15), we have the eigenvalues in terms of R_o to be

$$\left[-\mu_h\right],\left[-Q\right],\left[-(\varepsilon+\mu_h)\right],\left[-(1+\mu_h)\right],\left[-L\right],\frac{-\left[M+L\right]\pm\sqrt{\left[M+L\right]^2-4ML\left[1-R_o\right]}.}{2}$$

Thus we obtain

$$R_o=\frac{\left[\frac{\theta_{mh}\theta_{hm}\phi^2\rho\psi(1-\gamma)}{N_h^2LQ}\right]}{ML}=\frac{1}{ML}\left[\frac{\theta_{mh}\theta_{hm}\phi^2\rho\psi(1-\gamma)}{N_h^2LQ}\right]$$ and by substituting the expressions for M, N_h, L and Q yields $R_o=\frac{(1-\gamma)\theta_{mh}\theta_{hm}\phi^2\rho\mu_h^2}{\psi(\mu_m+k)^2(\mu_h+g+\upsilon)\left[\tau+r+(\mu_h+\delta_h)\right]}$

which conforms to the solution in equation (15) obtained based on the next generation matrix method.

The eigenvalues are hereby analyzed below

$\lambda_1 = -\mu_h < 0$, $\lambda_2 = -Q = -(\mu_h + g + \upsilon) < 0$, $\lambda_3 = -(\varepsilon + \mu_h) < 0$, $\lambda_4 = -(1 + \mu_h) < 0$, $\lambda_5 = -L = -(\mu_m + k) < 0$.

$$\lambda_6 = \frac{-[M+L] + \sqrt{[M+L]^2 - 4ML[1-R_o]}}{2}$$

$$\lambda_7 = \frac{-[M+L] - \sqrt{[M+L]^2 - 4ML[1-R_o]}}{2}$$

If $1 - R_o > 0$, then $R_o < 1$ and

$$\lambda_6 < \frac{-[M+L]}{2} + \frac{\sqrt{[M+L]^2}}{2} = 0, \text{ and}$$

$$\lambda_7 < \frac{-[M+L]}{2} - \frac{\sqrt{[M+L]^2}}{2} = -[M+L] = -[(\tau + r + \mu_h + \delta_h) + (\mu_m + k)]$$

Therefore, $\lambda_6 < 0$ and $\lambda_7 < 0$ thus establishing $\lambda_1 < 0$, $\lambda_2 < 0$, $\lambda_3 < 0$, $\lambda_4 < 0$, $\lambda_5 < 0$, $\lambda_6 < 0$, $\lambda_7 < 0$

Theorem

Given the system of equations in (8) - (14) and that $\gamma, \psi, \varphi, \tau, \upsilon, r, g, \mu_h, \mu_m, k, \varepsilon, \delta > 0$, the disease-free equilibrium E_o is locally asymptotically stable if and only if, $R_o \leq 1$, Li et al. (1999).

Graphical Representaation

Since the relapse parameter affects the transition between the recovered and infected classes. We plot the graph of the Infected class vs. Recovered class for the revised malaria model with relapse parameters of 0.25, 0.5, and 0.75 as shown in fig 1 below

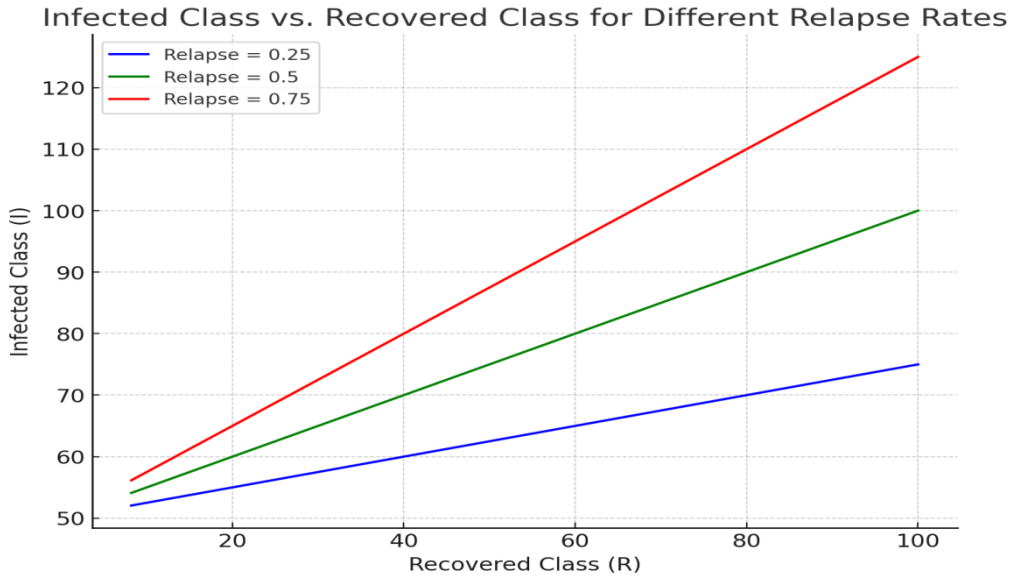

Fig 1

In fig 1, the graph clearly shows that as the number of recovered individuals increases, the infected class also increases proportionally based on the relapse rate.

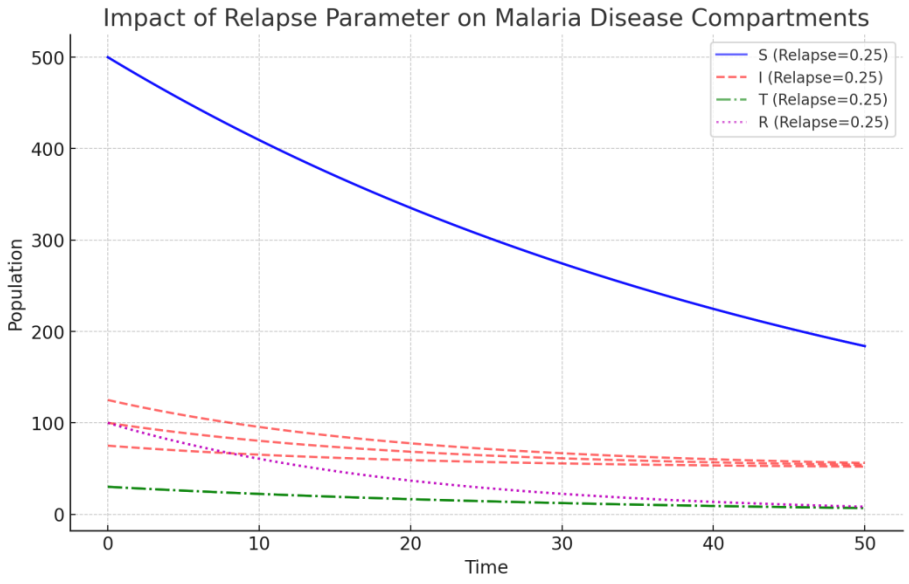

Fig 2

We present in fig 2 the graph showing the impact of the relapse parameter on different compartments in malaria disease dynamics.

The Susceptible (S) gradually decreases over time as people move into infected or recovered classes. The Infected (I) increases with higher relapse rates as more recovered

individuals become reinfected. The Treated (T) decreases over time, possibly due to recovery or lack of new infections The Recovered (R) declines faster when relapse rates are high, as more individuals move back to the infected class This demonstrates how relapse significantly affects malaria control efforts.

Conclusion

The SPITR mathematical model by Adamu et al. (2017) was modified to include a parameter for failed or ineffective treatment to assess its impact on malaria dynamics within human hosts and mosquito vectors. The model was analyzed using actual population data, and the stability of the equilibrium points was examined through linearization. The results showed that the equilibrium point is locally asymptotically stable.

The relapse parameter incorporated plays a crucial role in the dynamics of malaria transmission by influencing the transition from the recovered class back to the infected class. Higher relapse rates lead to an increase in the number of infected individuals, prolonging the persistence of malaria within a population. This makes disease control more challenging, as individuals who recover may become reinfected instead of remaining immune.

As relapse increases, the recovered population (R) declines faster, shifting more individuals into the infected class (I), which sustains disease transmission. This reduces the effectiveness of treatment and control strategies, as a larger infected population increases the burden on healthcare systems. Additionally, the susceptible class (S) decreases more gradually, as infections remain prevalent for a longer time. The treated class (T) also declines over time, but higher relapse rates may require more treatment interventions.

A combination of vaccination, effective treatment, and vector control measures is necessary to counteract the effects of high relapse rates. Reducing relapse can significantly lower malaria prevalence and improve eradication efforts. Without proper interventions, a high relapse rate can lead to recurrent outbreaks, making malaria a persistent public health threat.

Based on the findings of this study, we found that reducing malaria relapse requires a combination of effective treatment, early diagnosis, and vector control. Primaquine and Tafenoquine are essential drugs that target dormant liver-stage parasites, preventing

recurrence. Ensuring full adherence to prescribed treatment is crucial. Rapid diagnostic tests (RDTs) and follow-up monitoring help detect and treat relapses early. Vector control measures, such as insecticide-treated nets (ITNs), indoor residual spraying (IRS), and eliminating mosquito breeding sites, reduce reinfection risks. Strengthening public health policies, increasing awareness, and improving healthcare accessibility can further minimize relapse rates. Ongoing research on malaria vaccines may also contribute to long-term relapse reduction.

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