



Fractional-Order Mathematical Modeling and Analysis of Malaria Incorporating Control Measures via the Adams–Bashforth–Moulton Method

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Control,
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ABSTRACT

The epidemiological features of malaria infection are taken into consideration in this paper as a fractional-order mathematical model in Caputo derivative. The activities that the model uses to manage the disease are treatment and vaccination to study the effects of the controls on the disease dynamics. The theory of Lyapunov functions determines and verifies the existence and uniqueness of solutions within the frame of the fractional order and the stability of the endemic equilibrium point. The model is numerically obtained with the help of the fractional Adams-Bashforth-Moulton algorithm that will indicate the alteration of the model parameters, and the fractional orders of the model parameters to the impact of each of the mentioned parameters on the course of the disease. It has been established through the application of simulation that the more the disease is treated and vaccinated the less the prevalence of malaria and that the fractional-order models have high level of flexibility and realism than the classical integer order equations. The paper identifies the importance of fractional modeling in the description of the interactions between the effects of memory and nonlocal interaction between the biological systems and this enhances the understanding and control of infectious diseases. The model does however assume that the population is homogeneous mixed and hypothetical values of the parameters thus preventing the empirical validation. To make the model more predictive and practical to use in the formulation of effective control schemes against malaria, then the future research must be capable of addressing the spatial heterogeneity, stochasticity.

CITATION

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INTRODUCTION

The bite of infected females Anopheles mosquitoes is the way the Plasmodium parasite enters the body of a man and leads to malaria. Since hundreds of thousands of years this protozoan parasite has been causing malaria epidemics and it is one of the key world health problems

(Global Malaria Prevention and Treatment Foundation, 2013). In spite of tremendous advances in control and treatment, malaria continues to cause a huge public health burden especially in tropical and subtropical areas of Africa, Asia, and South America (World Health Organization [WHO], 2011). The disease is endemic in

more than 100 countries and it poses a threat to the lives of more than one-third of the world population (WHO, 2011). About 216 million cases of malaria were reported to have occurred in the world in 2010, with an estimated 655,000 deaths (WHO, 2011, 2012). This is particularly true of children below five years old and the percentage of malaria mortality attributed to them is a significant percentage (Chiyaka et al., 2006). Despite the production and continuous improvement of malaria vaccines, the overall prevention strategies are still necessary. Such are the vectority control activities, the use of insecticide impregnated nets, interior residual spraying and effective antimalarial drugs treatments (Esteva et al., 2009; Fillinger et al., 2009; Rhee et al., 2005; Roberts et al., 1997; WHO, 2011; Zi et al., 2012).

Mathematical modeling is important in deciphering the dynamics of malaria transmission and guiding interventions by the population health. By using the mathematical framework, scientists have the opportunity to study the processes that cause disease transmission and assess the efficacy of different control tools. Nonetheless, the traditional integer-order models frequently do not provide biological memory effects and long-term dependencies of epidemiological systems. To overcome such limitations, fractional-order models use non-local operators to consider the memory and hereditary characteristics of the disease transmission processes (Diethelm, 2022).

FD equations Fractional different equations (FDEs) are a generalization of the classical methods of modeling to allow a more general and detailed approach to analysis. This research paper develops a fractional-order model that helps study the dynamics of malaria transmission taking into consideration prevention and treatment controls. The model is more realistic in the effects of memory by incorporating the effects of memory via the use of the fractional calculus; this enables the model to provide a realistic understanding of the infection progression and the effects of interventions. The control measures are considered in order to identify effective interventions in reducing the prevalence of malaria.

In biological modeling, the reason why fractional derivatives are particularly useful is due to the fact that they are able to reproduce memory and hereditary properties, which contribute to the progression of diseases. In contrast to classical derivatives, fractional derivatives enable the current situation of the system to be based on its historical behavior. This attribute allows analyzing more precisely the role of past infections, history of treatment and the level of immunity in the perpetuation of infection. Fractional models can therefore help shed light on chronic issues like drug resistance, re-infection and limitations on health care.

The current advances in fractional calculus have proven that it is useful in the modeling of complex dynamic

systems (Atokolo et al., 2022). Although classical integer-order models mainly model the behavior of the local system, of the global system, the fractional-order models can include the dynamics of the global system as the memory-dependent processes. This feature is what helps fractional models to be more appropriate when it comes to representing real-world epidemiological trends.

Fractional derivatives that are commonly used in biology are Caputo and RiemannLiouville derivatives, which are singular in nature (Milici et al., 2018). More recently, non-singular, e.g. the MittagLeffler and AtanganaBaleanu, derivatives have become more popular because of their better mathematical features and use in modeling actual systems.

It has been demonstrated that many studies have been able to use the fractional modeling techniques in the dynamics of infectious diseases. Atokolo et al. (2022) established a model of the fractional-order sterile insect technology that is controlled by Laplace-Adomian Decomposition Method (LADM) to reduce Zika virus outbreaks. In a similar manner, Atokolo et al. (2024) explored the dynamics of transmissions of the Lassa fever through a fractional framework to assess the effect of vaccination and treatment measures. Yunus et al. (2023) used a fractional derivatives approach to forecasting Lassa fever dynamics and Omede et al. (2024) developed a Caputo-based fractional compartmental model of soil-transmitted helminth infections and showed that LADM was more flexible in modeling the dynamics of solutions. Amos et al. (2024) developed the fractional model of hepatitis C transmission and used the AdamsBashfordMoulton numerical scheme to demonstrate that the contact rates and improvement of treatment had a massive impact on the reduction of disease transmission. The fractional approaches to HIV/AIDS and diphtheria, used by Philip et al. (2024) and Abah et al. (2024) respectively, point at the flexibility and strength of the fractional systems in relation to classical models. A model of fractional Chlamydia transmission developed by Joseph et al. (2025) based on the generalized AdamsBashforthMoulton method showed that improving treatment and vaccine coverage was an effective way to lower the prevalence of infections. Ahmed et al. (2021) developed an ABC fractional-order model to investigate the dynamics of HIV and COVID-19 co-infection, whereas Smith et al. (2023) performed a systematic review of hepatitis C and COVID-19 co-infection modeling strategies to identify the methodological issues and gaps in the literature. Das et al. (2024) also investigated the dynamics of hepatitis C and co-infection with COVID-19 in low- and middle-income states, with a particular focus on practical and structural issues in the process of disease modelling. Besides this, Ullah et al. (2020) proposed a hybrid Laplace transform and Adomian Decomposition Method to solve the fuzzy Volterra integral equations, which is an

improvement in the analytical methods of fractional systems. Ali et al. (2017) studied the solutions and stability of the fractional problems of the boundary value, particularly the Ulam stability and enhancing the theory of the fractional differential equations.

In general, the concept of fractional-order modeling is an effective and versatile tool in the analysis of the dynamics of infectious diseases. Fractional differential equations can capture memory effects and non-local interactions, which are of interest in the study of epidemiology and control policies; hence, it is specifically effective when analyzing the transmission of malaria and its control measures.

This paper aims to prepare conditions to the presence and uniqueness of solutions in a Malaria model of the fractional-order; Stability analysis of the endemic equilibrium with the method of Lyapunov function; Solve the model numerically using the fractional Adams -Bashford Moulton method; Simulate to find the dynamics of the model. Since the comprehensive literature review demonstrates that the past researchers did not combine the use of fractional calculus and Adams-Bashforth-Moulton method to model Malaria disease and control it.

Preliminary

Here, we introduce the fundamental concepts and initial findings of fractional calculus. Our analysis incorporates both the right and left Caputo fractional derivatives, building on the models established by Milici et al. (2018) and Bonyah et al. (2020). We also explore the practical applications of this mathematics, demonstrating its use in solving real-world problems across diverse fields like physics, engineering, and bio-mathematics.

Definition 1

Let $f \in \Lambda^\infty(R)$ then the left and right Caputo fractional derivative of the function f is given by

$$\begin{aligned} CD_t^\rho f(t) &= \left(t^0 D_t^{-(n-\rho)} \left(\frac{d}{dt} \right)^n f(t) \right) \\ CD_t^\rho f(t) &= \frac{1}{\Gamma(n-\rho)} \int_0^t ((t-\lambda)^{n-\rho-1} f^n(\lambda)) d\lambda, \end{aligned} \quad (1)$$

The same way

$$\begin{aligned} CD_T^\rho f(t) &= \left(D_T^{-(n-\rho)} \left(\frac{d}{dt} \right)^n \right) f(t) \\ {}^C D_T^\rho f(t) &= \frac{(-1)^n}{\Gamma(n-\rho)} \int_t^T (\lambda - t)^{n-\rho-1} f^n(\lambda) d\lambda \end{aligned}$$

Definition 2

The generalized Mittag-Leffler function $E_{\rho,\beta}(x)$ for $x \in R$ is given by

$$E_{\rho,\beta}(x) = \sum_{n=0}^{\infty} \frac{x^n}{\Gamma(\rho n + \beta)}, \quad \gamma, \psi > 0 \quad (2)$$

which can also be represented as

$$E_{\rho,\psi}(x) = x E_{\rho,\rho+\psi}(x) + \frac{1}{\Gamma(\psi)} \quad (3)$$

$$E_{\rho,\psi}(x) = L[t^{\psi-1} E_{\rho,\psi}(\pm \omega t^\rho)] = \frac{s^{\rho-\psi}}{s^\psi \pm \omega}.. \quad (4)$$

Proposition 1

Let $f \in \Lambda^\infty(R) \cap C(R)$ and $\rho \in R, n-1 < \rho < n$, therefore, the conditions given below holds:

1. ${}^C D_t^\rho I^\rho f(t) = f(t)$
2. ${}^C D_t^\rho I^\rho f(t) = f(t) - \sum_{k=0}^{n-k} \frac{t^k}{k!} f^k(t_0)$

Model Formulation

In modeling the dynamics, the population is divided into eight groups: Susceptible human population (S_h), Exposed human population (E_h), Vaccinated human population (V_h), Infected human population (I_h), humans on malaria treatment (T_h), Recovered human population (R_h), Susceptible vector population (S_v), Infected vector population (I_v). The susceptible humans are recruited at the rate of Λ_h , while the susceptible vector are recruited at the rate of Λ_v , Contact rate between the susceptible humans and infected vector population with malaria, Contact rate between the susceptible vectors and infected human population with malaria are β_h and β_v respectively. Natural death rate of human population and vector population are μ_h and μ_v respectively. Death induced rate due to an attempt by vectors to bite humans δ_3 , Disease induced death rate of malaria infected humans, Disease induced death rate of humans on malaria treatment are δ_1 and δ_2 respectively. Mosquitoes biting rate b , Vaccination rate of susceptible human population against malaria τ_1 , Waning rate vaccine τ_2 , Progression rate from Exposed human population to malaria infected human population ϕ , Treatment rate of malaria infected human population θ , Recovery rate due to treatment of malaria η , natural recovery rate of infected human population α .

Model Assumptions

1. We assume an imperfect vaccine in the human population
2. We assume exogenous re-infection in human population
3. We assume natural death in the population
4. We assume disease induced death in the population
5. We assume natural recovery in the human population due to strong body immunity.

Malaria Model Flow Chart

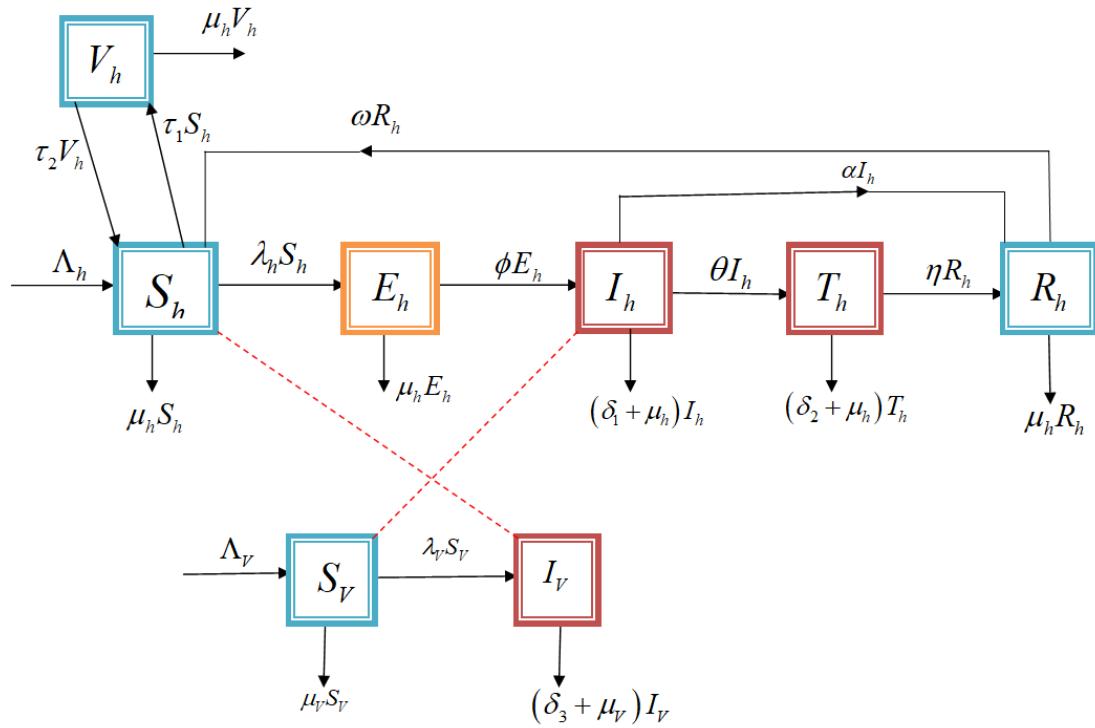


Figure 1: Malaria model flow Diagram

Figure 1 denotes shows the transmission dynamics of malaria incorporating treatment and vaccination as control measures.

Malaria Model Equations

$$\begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_h + \tau_2 V_h + \omega R_h - \lambda_h S_h - (\tau_1 + \mu_h) S_h, \\
 \frac{dE_h}{dt} &= \lambda_h S_h - (\phi + \mu_h) E_h, \\
 \frac{dV_h}{dt} &= \tau_1 S_h - (\tau_2 + \mu_h) V_h, \\
 \frac{dI_h}{dt} &= \phi E_h - (\theta + \alpha + \delta_1 + \mu_h) I_h, \\
 \frac{dT_h}{dt} &= \theta I_h - (\eta + \delta_2 + \mu_h) T_h, \\
 \frac{dR_h}{dt} &= \alpha I_h + \eta T_h - (\omega + \mu_h) R_h, \\
 \frac{dS_V}{dt} &= \Lambda_V - \lambda_V S_V - \mu_V S_V, \\
 \frac{dI_V}{dt} &= \lambda_V S_V - (\delta_3 + \mu_V) I_V.
 \end{aligned} \tag{5}$$

$$\begin{aligned}
 \frac{dS_h}{dt} &= \Lambda_h + \tau_2 V_h + \omega R_h - \lambda_h S_h - (\tau_1 + \mu_h) S_h, \\
 \frac{dE_h}{dt} &= \lambda_h S_h - (\phi + \mu_h) E_h, \\
 \frac{dV_h}{dt} &= \tau_1 S_h - (\tau_2 + \mu_h) V_h, \\
 \frac{dI_h}{dt} &= \phi E_h - (\theta + \alpha + \delta_1 + \mu_h) I_h, \\
 \frac{dT_h}{dt} &= \theta I_h - (\eta + \delta_2 + \mu_h) T_h, \\
 \frac{dR_h}{dt} &= \alpha I_h + \eta T_h - (\omega + \mu_h) R_h, \\
 \frac{dS_V}{dt} &= \Lambda_V - \lambda_V S_V - \mu_V S_V, \\
 \frac{dI_V}{dt} &= \lambda_V S_V - (\delta_3 + \mu_V) I_V.
 \end{aligned}$$

Where

$$\lambda_h = \frac{b\beta_h I_V S_h}{N_h} \quad \lambda_V = \frac{\beta_V I_h S_V}{N_h}.$$

Table 1 presents a comprehensive description of the model variables and parameters employed in this study.

Table 1: Model Variables and Parameters Description

Variables	Descriptions
S_h	Susceptible human population to malaria
E_h	Exposed human population to malaria
V_h	Vaccinated human population against malaria
I_h	Infected human population with malaria
T_h	Human population on malaria treatment
R_h	Recovered human population from malaria
S_V	Susceptible vector population to malaria
I_V	Infected vector population with malaria
Parameters	Descriptions
Λ_h	Recruitment rate of human population
Λ_V	Recruitment rate of vector population
τ_1	Vaccination rate of human population
β_h	Contact rate between susceptible humans and infected vector population
β_V	Contact rate between susceptible vectors and infected human population
τ_2	Waning rate of vaccine in the human population

μ_h	Natural death rate of human population
μ_v	Natural death rate of vector population
ϕ	Progression rate from Exposed human population to infected human population
θ	Treatment rate of infected human population
η	Recovery due to treatment rate of human population
α	Natural recovery rate of human population due to strong body immunity
ω	Rate at which recovered humans become susceptible again
δ_1	Disease induced death rate of infected humans with malaria
δ_2	Disease induced death rate of humans on malaria treatment
δ_3	Death induced due to an attempt of vectors to bite humans

Model Analysis

Fractional Malaria Mathematical Model

In this section, we provide the enhancement of the integer model of Malaria represented in Equation (5) with the Caputo fractional derivative operator. The revised model that uses the Caputo fractional derivative operator is more flexible than the classical model in Equation (5) because the response of the fractional model can be manipulated in order to obtain different responses. The fractional Malaria model is, therefore, presented with the following introduction:

$$\begin{aligned} {}^C D_t^\rho S_h &= \Lambda_h + \tau_2 V_h + \omega R_h - \lambda_h S_h - K_1 S_h, \\ {}^C D_t^\rho E_h &= \lambda_h S_h - K_2 E_h, \\ {}^C D_t^\rho V_h &= \tau_1 S_h - K_3 V_h, \\ {}^C D_t^\rho I_h &= \phi E_h - K_4 I_h, \\ {}^C D_t^\rho T_h &= \theta I_h - K_5 T_h, \\ {}^C D_t^\rho R_h &= \alpha I_h + \eta T_h - K_6 R_h, \\ {}^C D_t^\rho S_V &= \Lambda_V - \lambda_V S_V - K_7 S_V, \\ {}^C D_t^\rho I_V &= \lambda_V S_V - K_8 I_V. \end{aligned}$$

Where

$$\begin{aligned} K_1 &= (\tau_1 + \mu_h), K_2 = (\phi + \mu_h), K_3 = (\tau_2 + \mu_h), K_4 = (\theta + \alpha + \delta_1 + \mu_h), K_5 = (\eta + \delta_2 + \mu_h), \\ K_6 &= (\omega + \mu_h), K_7 = \mu_V, K_8 = (\delta_3 + \mu_V). \end{aligned}$$

Subject to positive initial conditions

$$S_h(0) = S_{h0}, E_h(0) = E_{h0}, V_h(0) = V_{h0}, I_h(0) = I_{h0}, T_h(0) = T_{h0}, R_h(0) = R_{h0}, V_h(0) = S_{V0}, I_h(0) = I_{h0} \quad (7)$$

Positivity of Model Equation

We considered the non-negativity of the initial values

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h} \quad \text{as } t \rightarrow \infty$$

Secondly, if $\limsup N_{h0}(t) \leq \frac{\Lambda_h}{\mu_h}$, then our model feasible domain is given by:

$$\Omega_h = \left\{ (S_h, E_h, V_h, I_h, T_h, R_h) \in R_+^6 : (S_h + E_h + V_h + I_h + T_h + R_h) \leq \frac{\Lambda_h}{\mu_h} \right\},$$

and $\Omega_V = \left\{ (S_V, I_V) \in R_+^2 : (S_V, I_V) \leq \frac{\Lambda_V}{\mu_V} \right\}$ so that,

$$\Omega = \Omega_h \times \Omega_V \subset R_+^8,$$

hence Ω is positively invariant.

If $(S_{h0}, E_{h0}, V_{h0}, I_{h0}, T_{h0}, R_{h0}, S_{V0}, I_{V0})$ are non-negative, then the solution of model (6) will be non-negative for $t > 0$. From Eq. (6), selecting the first equation, we obtained;

$$\begin{aligned} {}^C D_t^\rho S_h &= \Lambda_h + \tau_2 V_h + \omega R_h - \lambda_h S_h - K_1 S_h, \\ {}^C D_t^\rho S_h + (\lambda_h S_h + K_1 S_h) &= \Lambda_h + \tau_2 V_h + \omega R_h \\ \text{But } \Lambda_h + \tau_2 V_h + \omega R_h &\geq 0 \text{ then} \\ {}^C D_t^\rho S_h + (\lambda_h S_h + K_1 S_h) &\geq 0.. \\ \text{Applying the Laplace transform we obtained;} \\ L[{}^C D_t^\rho S_h] + L[(\lambda_h S_h + K_1 S_h)] &\geq 0. \\ S_h^\rho S_h(s) - S_h^{\rho-1} S_h(0) + (\lambda_h + P_1) S_h(s) &\geq 0, \\ S_h(s) \geq \frac{S_h^{\rho-1}}{S_h^{\rho} + (\lambda_h + K_1)} S_h(0) \end{aligned} \quad (8)$$

By taking the inverse Laplace transform, we obtained:

$$S_h(t) \geq E_{tp,1}(-(\lambda_h + K_1)t^\rho) S_{h0}. \quad (9)$$

Now since the term on the right-hand side of Eq. (9) is positive, we conclude that $S_h \geq 0$ for $t \geq 0$. In the same way, we also have that $(E_h \geq 0, V_h \geq 0, I_h \geq 0, T_h \geq 0, R_h \geq 0, S_V \geq 0, I_V \geq 0)$. that is positives; therefore, the solution will remain in R_+^8 for all $t \geq 0$ with positive initial conditions.

Boundedness of Fractional Model Equation

The total human population from our model is given by;

$$N_h(t) = S_h(t) + E_h(t) + V_h(t) + I_h(t) + T_h(t) + R_h(t) \\ \frac{dN_h}{dt} = S_h + I_h.$$

Likewise, the total vector population is

So from our fractional model (6), we now obtain

$$\begin{aligned} {}^C D_t^\rho N_h(t) &= {}^C D_t^\rho S_h(t) + {}^C D_t^\rho E_h(t) + {}^C D_t^\rho V_h(t) + \\ {}^C D_t^\rho I_h(t) + {}^C D_t^\rho T_h(t) + {}^C D_t^\rho R_h(t). \\ {}^C D_t^\rho N_h(t) &= \Lambda_h - \mu_h N_h(t) \end{aligned}$$

Taking the Laplace transformation of (10) we obtained:

$$\begin{aligned} L[{}^C D_t^\rho N_h(t)] &= L[\Lambda_h - \mu_h N_h(t)] \\ S_h^\rho N_h(s) - S_h^{\rho-1} N_h(0) + \mu_h N_h(s) &\leq \frac{\Lambda_h}{\mu_h}, \\ N_h(s) &\leq \frac{S_h^{\rho-1}}{(S_h^\rho + \mu_h)} N_h(0) + \frac{\Lambda_h}{S_h(S_h^\rho + \mu_h)} \end{aligned} \quad (11)$$

Taking the inverse Laplace transform of Eq. (11) we have;

$$N_h(t) \leq E_{hp,1}(\mu_h t^\rho) N_h(0) + \Lambda_h E_{hp,\rho+1}(\mu_h t^\rho) \quad (12)$$

At $t \rightarrow \infty$, the limit of Eq. (12) becomes:

$$\limsup_{t \rightarrow \infty} N_h(t) = \frac{\Lambda_h}{\mu_h} \text{ similarly, we have } \limsup_{t \rightarrow \infty} N_V(t) = \frac{\Lambda_V}{\mu_V}.$$

This means that, if $N_{h0} \leq \frac{\Lambda_h}{\mu_h}$ and $N_{V0}(t) \leq \frac{\Lambda_V}{\mu_V}$.

then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ and $N_{V0}(t) \leq \frac{\Lambda_V}{\mu_V}$. which implies that, $N_h(t)$ is bounded.

We now conclude that, this region $\Omega = \Omega_h \times \Omega_V$, is well posed and equally feasible epidemiologically.

Existence and Uniqueness of our Model Solution

Let the real non-negative be W we consider $P = [0, W]$.

All continuous function that exist on P belongs to $N_{he}^0(W)$ with norm as;

$$\|K\| = \text{Sup}\{|K(t)|, t \in W\}.$$

The modeled system (6) along with specified initial (8) enables solving for a system of differential equations presented in (13).

$${}^cD_t^\rho K(t) = Z(t, K(t)), 0 < t < W < \infty, \quad (13)$$

$$K(0) = K_0.$$

Where $K(t) = (S_h, E_h, V_h, I_h, T_h, R_h, S_V, I_V)$. represents the classes and Z be a continuous function defined as follows;

$$Z(t, K(t)) = \begin{pmatrix} Z_1(t, S_h(t)) \\ Z_2(t, E_h(t)) \\ Z_3(t, V_h(t)) \\ Z_4(t, I_h(t)) \\ Z_5(t, T_h(t)) \\ Z_6(t, R_h(t)) \\ Z_7(t, S_V(t)) \\ Z_8(t, I_V(t)) \end{pmatrix} = \begin{pmatrix} \Lambda_h + \tau_2 V_h + \omega R_h - \frac{b\beta_h I_V S_h}{N_h} - (\tau_1 + \mu_h) S_h, \\ \frac{b\beta_h I_V S_h}{N_h} S_h - (\phi + \mu_h) E_h, \\ \tau_1 S_h - (\tau_2 + \mu_h) V_h, \\ \phi E_h - (\theta + \alpha + \delta_1 + \mu_h) I_h, \\ \theta I_h - (\eta + \delta_2 + \mu_h) T_h, \\ \alpha I_h + \eta T_h - (\omega + \mu_h) R_h, \\ \Lambda_V - \frac{\beta_V I_h S_V}{N_h} - \mu_V S_V, \\ \frac{\beta_V I_h S_V}{N_h} - (\delta_3 + \mu_V) I_V. \end{pmatrix} \quad (14)$$

Using proposition (2.1), we have that,

$$\begin{aligned} S_h(t) &= S_{h0} + I_t^\rho \left[\Lambda_h + \tau_2 V_h + \omega R_h - \frac{b\beta_h I_V S_h}{N_h} - (\tau_1 + \mu_h) S_h \right], \\ E_h(t) &= E_{h0} + I_t^\rho \left[\frac{b\beta_h I_V S_h}{N_h} S_h - (\phi + \mu_h) E_h \right], \\ V_h(t) &= V_{h0} + I_t^\rho \left[\tau_1 S_h - (\tau_2 + \mu_h) V_h \right], \\ I_h(t) &= I_{h0} + I_t^\rho \left[\phi E_h - (\theta + \alpha + \delta_1 + \mu_h) I_h \right], \\ T_h(t) &= T_{h0} + I_t^\rho \left[\theta I_h - (\eta + \delta_2 + \mu_h) T_h \right], \\ R_h(t) &= R_{h0} + I_t^\rho \left[\alpha I_h + \eta T_h - (\omega + \mu_h) R_h \right], \\ S_V(t) &= S_{V0} + I_t^\rho \left[\Lambda_V - \frac{\beta_V I_h S_V}{N_h} - \mu_V S_V \right], \\ I_V(t) &= I_{V0} + I_t^\rho \left[\frac{\beta_V I_h S_V}{N_h} - (\delta_3 + \mu_V) I_V \right]. \end{aligned} \quad (15)$$

We obtain the Picard iteration of (15) as follows;

$$\begin{aligned} S_h(t) &= S_{h0} + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z_1(\lambda_h, S_{h(n-1)}(\lambda_h)) d\lambda_h, \\ E_h(t) &= E_{h0} + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z_2(\lambda_h, E_{h(n-1)}(\lambda_h)) d\lambda_h, \quad V_h(t) = V_{h0} + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z_3(\lambda_h, V_{h(n-1)}(\lambda_h)) d\lambda_h, \\ I_h(t) &= I_{h0} + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z_4(\lambda_h, I_{h(n-1)}(\lambda_h)) d\lambda_h, \\ T_h(t) &= T_{h0} + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z_5(\lambda_h, T_{h(n-1)}(\lambda_h)) d\lambda_h, \\ R_h(t) &= R_{h0} + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z_6(\lambda_h, R_{h(n-1)}(\lambda_h)) d\lambda_h, \\ S_V(t) &= S_{V0} + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z_7(\lambda_h, S_{V(n-1)}(\lambda_h)) d\lambda_h, \quad I_V(t) = I_{V0} + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z_8(\lambda_h, I_{V(n-1)}(\lambda_h)) d\lambda_h. \end{aligned} \quad (16)$$

Transforming equation eq. (13) to get

$$X(t) = X(0) + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z(\lambda_h, X(\lambda_h)) d\lambda_h. \quad (17)$$

Lemma 1, The equation (14) gives us the definition of the Lipchitz condition which vector satisfies; $Z(t, K(t))$ on a set $[0, W]_+^8$ with the Lipchitz constant given as;

$$\omega = \max \left((\beta_h^* + \tau_1 + \mu_h), (\phi + \mu_h), (\tau_2 + \mu_h), (\theta + \alpha + \delta_1 + \mu_h), (\eta + \delta_2 + \mu_h), (\omega + \mu_h), (\mu_V), (\delta_3 + \mu_V) \right).$$

Proof:

$$\begin{aligned} &\|Z_1(t, S_h) - Z_1(t, S_{h1})\|, \\ &= \left\| \Lambda_h + \tau_2 V_h + \omega R_h - \frac{b\beta_h I_V S_h}{N_h} - (\tau_1 + \mu_h) S_h - \Lambda_h + \tau_2 V_h + \omega R_h - \frac{b\beta_h I_V S_h}{N_h} - (\tau_1 + \mu_h) S_{h1} \right\|, \\ &= \left\| -\Lambda_h + \tau_2 V_h + \omega R_h - \frac{b\beta_h I_V}{N_h} - (\tau_1 + \mu_h)(S_h - S_{h1}) + \mu_h(S_h - S_{h1}) \right\| \leq (\beta_h^* + \tau_1 + \mu_h) \|S_h - S_{h1}\| + \mu_h \|S_h - S_{h1}\|, \quad \therefore \\ &\|Z_1(t, S_h) - Z_1(t, S_{h1})\| \leq (\beta_h^* + \tau_1 + \mu_h) \|S_h - S_{h1}\|. \end{aligned}$$

Similarly, we obtained the following:

$$\begin{aligned}
 \|Z_2(t, E_h) - Z_2(t, E_{h1})\| &\leq (\phi + \mu_h) \|E_h - E_{h1}\|, \\
 \|Z_3(t, V_h) - Z_3(t, V_{h1})\| &\leq (\tau_2 + \mu_h) \|V_h - V_{h1}\|, \\
 \|Z_4(t, I_h) - Z_4(t, I_{h1})\| &\leq (\theta + \alpha + \delta_1 + \mu_h) \|I_h - I_{h1}\|, \\
 \|Z_5(t, T_h) - Z_5(t, T_{h1})\| &\leq (\eta + \delta_2 + \mu_h) \|T_h - T_{h1}\|, \\
 \|Z_6(t, R_h) - Z_6(t, R_{h1})\| &\leq (\omega + \mu_h) \|R_h - R_{h1}\|, \\
 \|Z_7(t, S_V) - Z_7(t, S_{V1})\| &\leq (\mu_V) \|S_V - S_{V1}\|, \\
 \|Z_8(t, I_V) - Z_8(t, I_{V1})\| &\leq (\delta_3 + \mu_V) \|I_V - I_{V1}\|.
 \end{aligned} \tag{18}$$

Where we obtained:

$$\begin{aligned}
 \|Z(t, K_1(t)) - Z(t, K_2(t))\| &\leq \omega \|K_1 - K_2\|, \\
 \omega = \max & \left((\beta_h^* + \tau_1 + \mu_h), (\phi + \mu_h), (\tau_2 + \mu_h), (\theta + \alpha + \delta_1 + \mu_h), (\eta + \delta_2 + \mu_h), (\omega + \mu_h), (\mu_V), (\delta_3 + \mu_V) \right). \tag{19}
 \end{aligned}$$

Lemma 2

The initial value problem (6), (7) in Eq. (19) exists and will have a unique solution $K(t) \in D_c^0(E)$.

Applying Picard-Lindelöf fixed-point conjecture, we consider the solution of $K(t) = S_h(K(t))$

where S is defined as the Picard operator expressed as ;

$$S_h: D_c^0(E, R_+^8) \rightarrow D_c^0(E, R_+^8).$$

Therefore,

$$S_h(K(t)) = K(0) + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z(\lambda_h, K(\lambda_h)) d\lambda_h.$$

which becomes,

$$\begin{aligned}
 \|S_h(K_1(t)) - S_h(K_2(t))\| &= \left\| \frac{1}{\Gamma(\rho)} \left[\int_0^t (t - \lambda_h)^{\rho-1} Z(\lambda_h, K_1(\lambda_h)) - Z(\lambda_h, K_2(\lambda_h)) d\lambda_h \right] \right\| \\
 &\leq \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} \|Z(\lambda_h, K_1(\lambda_h)) - Z(\lambda_h, K_2(\lambda_h)) d\lambda_h\| \\
 &\leq \frac{\omega}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} \|K_1 - K_2\| d\lambda_h. \\
 \|S_h(K_1(t)) - S_h(K_2(t))\| &\leq \frac{\omega}{\Gamma(\rho+1) S_h}. \tag{20}
 \end{aligned}$$

$$\text{When } \frac{\omega}{\Gamma(\rho+1)} S_h \leq 1,$$

then the Picard operator gives a contradiction, so Eq. (6), (7) solution is unique.

Lemma 2: The initial value problem (6), (7) in Eq. (19) exists and will have a unique solution.

$$X(t) \in A_c^0(f).$$

Using Picard-Lindelöf and fixed- point theory, we consider the solution of

$$X(t) = S_h(X(t)),$$

where S is defined as the Picard operator expressed as;

$$S_h: A_c^0(f, R_+^8) \rightarrow A_c^0(f, R_+^8).$$

Therefore,

$$S_h(X(t)) = X(0) + \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} Z(\lambda_h, X(\lambda_h)) d\lambda_h.$$

This becomes,

$$\begin{aligned}
 \|S_h(X_1(t)) - S_h(X_2(t))\| &= \left\| \frac{1}{\Gamma(\rho)} \left[\int_0^t (t - \lambda_h)^{\rho-1} Z(\lambda_h, X_1(\lambda_h)) - Z(\lambda_h, X_2(\lambda_h)) d\lambda_h \right] \right\|, \leq \frac{1}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} \|Z(\lambda_h, X_1(\lambda_h)) - Z(\lambda_h, X_2(\lambda_h)) d\lambda_h\| \leq \\
 &\leq \frac{\psi}{\Gamma(\rho)} \int_0^t (t - \lambda_h)^{\rho-1} \|X_1 - X_2\| d\lambda_h. \\
 \|S_h(X_1(t)) - S_h(X_2(t))\| &\leq \frac{\psi}{\Gamma(\rho+1) S_h}. \tag{21}
 \end{aligned}$$

$$\text{When } \frac{\psi}{\Gamma(\rho+1)} S_h \leq 1, \quad \text{then the Picard operator gives a contradiction,}$$

So Eq. (6), (7) solution is unique.

Disease Free Equilibrium Point of Malaria Model

Disease free Equilibrium point is the steady state where there is no disease in the population. At DFE

$$S_h \neq 0, E_h = 0, V_h \neq 0, I_h = 0, T_h = 0, R_h = 0, S_V \neq 0, I_V = 0.$$

$$E_0 = (S_h^0, E_h^0, V_h^0, I_h^0, T_h^0, R_h^0, S_V^0, I_V^0) = \left(\frac{\Lambda_h(\tau_2 + \mu_h)}{\mu_h(\tau_2 + \tau_1 + \mu_h)}, 0, \frac{\tau_1 \Lambda_h}{\mu_h(\tau_2 + \tau_1 + \mu_h)}, 0, 0, 0, \frac{\Lambda_V}{\mu_V}, 0 \right), \quad (22)$$

Basic Reproduction Number of Malaria

Basic Reproduction number is the secondary cases of infection when an infected vector is introduced into a susceptible human population.

It is represented by $R_0^M = \rho FV^{-1}$ where ρ is the dominant Eigen value, F is the non-negative matrix and V is the other transition term.

$$F = \begin{pmatrix} 0 & 0 & 0 & \frac{b\beta_h(\tau_2 + \mu_h)}{(\tau_2 + \tau_1 + \mu_h)} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_V \Lambda_V \mu_h}{\Lambda_h \mu_V} & 0 & 0 \end{pmatrix}, \text{ and } V = \begin{pmatrix} A_1 & 0 & 0 & 0 \\ -\phi & A_2 & 0 & 0 \\ 0 & -\theta & A_3 & 0 \\ 0 & 0 & 0 & A_4 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{A_1} & 0 & 0 & 0 \\ \frac{\phi}{A_2 A_1} & \frac{1}{A_2} & 0 & 0 \\ \frac{\theta \phi}{A_2 A_1 A_3} & \frac{\theta}{A_2 A_3} & \frac{1}{A_3} & 0 \\ 0 & 0 & 0 & \frac{1}{A_4} \end{pmatrix} \quad (23)$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & 0 & \frac{b\beta_h(\tau_2 + \mu_h)}{(\tau_2 + \tau_1 + \mu_h) A_4} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ \frac{\beta_V \Lambda_V \mu_h \phi}{\Lambda_h \mu_V A_1 A_2} & \frac{\beta_V \Lambda_V \mu_h}{\Lambda_h \mu_V A_2} & 0 & 0 \end{pmatrix} \quad (24)$$

$$R_0^M = \frac{\sqrt{\Lambda_h \mu_V A_1 A_2 (\tau_2 + \tau_1 + \mu_h) A_4 \beta_V \Lambda_V \mu_h \phi b \beta_h (\tau_2 + \mu_h)}}{\Lambda_h \mu_V A_1 A_2 (\tau_2 + \tau_1 + \mu_h) A_4}. \quad (25)$$

which is the dominant Eigen value

Endemic Equilibrium Point of Malaria Model

Endemic equilibrium point is a point where malaria persists in the population.

At endemic equilibrium point

$$S_h \neq 0, E_h \neq 0, V_h \neq 0, I_h \neq 0, T_h \neq 0, R_h \neq 0, S_V \neq 0, I_V \neq 0.$$

$$S_h^* = -\frac{\Lambda_h K_3 K_2 K_4 K_5 K_6}{((-\Lambda_h K_4 (\lambda_h + K_1) K_2 + \lambda_h \phi \alpha \omega) K_5 + \eta \omega \phi \theta \lambda_h) K_3 + K_2 K_4 K_5 K_6 \tau_1 \tau_2},$$

$$E_h^* = -\frac{\Lambda_h K_3 K_4 K_5 K_6 \lambda_h}{((\alpha \omega \phi - K_2 K_4 K_6) \lambda_h - K_6 K_4 K_2 K_1) K_5 + \eta \omega \phi \theta \lambda_h) K_3 + K_2 K_4 K_5 K_6 \tau_1 \tau_2},$$

$$V_h^* = -\frac{\Lambda_h K_2 K_4 K_5 K_6 \tau_1}{(((-\lambda_h - K_1) K_3 + \tau_1 \tau_2) K_4 K_6 K_2 + \lambda_h \phi \alpha \omega K_3) K_5 + \eta \omega \phi \theta K_3 \lambda_h},$$

$$= -\frac{\lambda_h \Lambda_h K_5 K_6 K_3 \phi}{((\alpha \omega \phi - K_2 K_4 K_6) \lambda_h - K_6 K_4 K_2 K_1) K_5 + \eta \omega \phi \theta \lambda_h) K_3 + K_2 K_4 K_5 K_6 \tau_1 \tau_2},$$

$$T_h^* = -\frac{\Lambda_h \Lambda_h K_6 K_3 \phi \theta}{((\alpha \omega \phi - K_2 K_4 K_6) K_5 + \eta \phi \omega \theta) \lambda_h - K_5 K_6 K_1 K_2 K_4) K_3 + K_2 K_4 K_5 K_6 \tau_1 \tau_2},$$

$$R_h^* = -\frac{\lambda_h \Lambda_h K_3 \phi (\alpha K_5 + \eta \theta)}{\alpha \omega \phi K_3 K_5 \lambda_h + \eta \omega \phi \theta K_3 \lambda_h - K_1 K_2 K_3 K_4 K_5 K_6 - K_2 K_3 K_4 K_5 K_6 \lambda_h + K_2 K_4 K_5 K_6 \tau_1 \tau_2},$$

$$S_V^* = \frac{\Lambda_p}{\lambda_v + K_7},$$

$$I_V^* = \frac{\Lambda_v \lambda_p}{(\lambda_v + K_7) K_8}.$$

Substituting these into the force of infection $\lambda_h = \frac{b\beta_h I_V S_h}{N_h}$ and $\lambda_V = \frac{\beta_V I_h S_V}{N_h}$.

We obtain;

$$Q_1 \lambda_h^2 + Q_2 \lambda + Q_3 = 0 \quad (27)$$

Where

$$\begin{aligned}
Q_1 &= \alpha^2 \phi^2 K_3^2 K_5^2 K_7^2 K_8 + 2\alpha\eta\phi^2\theta K_3^2 K_5 K_7^2 K_8 + 2\alpha\phi^2\theta K_3^2 K_5 K_6 K_7^2 K_8 \\
&+ 2\alpha\phi^2 K_3^2 K_5^2 K_6 K_7^2 K_8 + \alpha\phi^2 K_3^2 K_5^2 K_6 K_8 \Lambda_V \beta_V + 2\alpha\phi K_3^2 K_4 K_5^2 K_6 K_7^2 K_8 \\
&+ \eta^2 \phi^2 \theta^2 K_3^2 K_7^2 K_8 + 2\eta\phi^2 \theta^2 K_3^2 K_6 K_7^2 K_8 + 2\eta\phi^2 \theta K_3^2 K_5 K_6 K_7^2 K_8 \\
&+ \eta\phi^2 \theta K_3^2 K_5 K_6 K_8 \Lambda_V \beta_V + 2\eta\phi \theta K_3^2 K_4 K_5 K_6 K_7^2 K_8 + \phi^2 \theta^2 K_3^2 K_6^2 K_7^2 K_8 \\
&+ 2\phi^2 \theta K_3^2 K_5 K_6^2 K_7^2 K_8 + \phi^2 \theta K_3^2 K_5 K_6^2 K_8 \Lambda_V \beta_V + \phi^2 K_3^2 K_5^2 K_6^2 K_7^2 K_8 \\
&+ \phi^2 K_3^2 K_5^2 K_6^2 K_8 \Lambda_V \beta_V + 2\phi\theta K_3^2 K_4 K_5 K_6^2 K_7^2 K_8 + 2\phi K_3^2 K_4 K_5^2 K_6^2 K_7^2 K_8 \\
&+ \phi K_3^2 K_4 K_5^2 K_6^2 K_8 \Lambda_V \beta_V + K_3^2 K_4^2 K_5^2 K_6^2 K_7^2 K_8 + \alpha^2 \phi^2 K_3^2 K_5^2 K_7^2 K_8 \\
&+ 2\alpha\eta\phi^2 \theta K_3^2 K_5 K_7 K_8 + 2\alpha\phi^2 \theta K_3^2 K_5 K_6 K_7 K_8 + 2\alpha\phi^2 K_3^2 K_5^2 K_6 K_7 K_8 \\
&+ 2\alpha\phi K_3^2 K_4 K_5^2 K_6 K_7 K_8 + 2\eta\phi^2 \theta^2 K_3^2 K_6 K_7 K_8 + 2\eta\phi^2 \theta K_3^2 K_5 K_6 K_7 K_8 \\
&+ 2\eta\phi \theta K_3^2 K_4 K_5 K_6 K_7 K_8 + \phi^2 \theta^2 K_3^2 K_6^2 K_7 K_8 + 2\phi^2 \theta K_3^2 K_5 K_6^2 K_7 K_8 \\
&+ \phi^2 K_3^2 K_5^2 K_6^2 K_7 K_8 + 2\phi\theta K_3^2 K_4 K_5 K_6^2 K_7 K_8 + 2\phi K_3^2 K_4 K_5^2 K_6^2 K_7 K_8 \\
&+ K_3^2 K_4^2 K_5^2 K_6^2 K_7 K_8, \\
Q_2 &= 2\alpha\phi K_2 K_3^2 K_4 K_5^2 K_6 K_7^2 K_8 + 2\alpha\phi K_2 K_3 K_4 K_5^2 K_6 K_7^2 K_8 \tau_1 + 2\eta\phi \theta K_2 K_3^2 K_4 K_5 K_6 K_7^2 K_8 \\
&+ 2\eta\phi \theta K_2 K_3 K_4 K_5 K_6 K_7^2 K_8 \tau_1 + 2\phi\theta K_2 K_3^2 K_4 K_5 K_6^2 K_7^2 K_8 + 2\phi\theta K_2 K_3 K_4 K_5 K_6^2 K_7^2 K_8 \tau_1 \\
&+ 2\phi K_2 K_3^2 K_4 K_5^2 K_6^2 K_7^2 K_8 + \phi K_2 K_3^2 K_4 K_5^2 K_6^2 K_8 \Lambda_V \beta_V + 2\phi K_2 K_3 K_4 K_5^2 K_6^2 K_7^2 K_8 \tau_1 \\
&+ \phi K_2 K_3 K_4 K_5^2 K_6^2 K_8 \Lambda_V \beta_V \tau_1 + 2K_2 K_3^2 K_4^2 K_5^2 K_6^2 K_7^2 K_8 + 2K_2 K_3 K_4^2 K_5^2 K_6^2 K_7^2 K_8 \tau_1 \\
&+ 2\alpha\phi K_2 K_3^2 K_4 K_5^2 K_6 K_7 K_8 + 2\alpha\phi K_2 K_3 K_4 K_5^2 K_6 K_7 K_8 \tau_1 + 2\eta\phi \theta K_2 K_3^2 K_4 K_5 K_6 K_7 K_8 \\
&+ 2\eta\phi \theta K_2 K_3 K_4 K_5 K_6 K_7 K_8 \tau_1 + 2\phi\theta K_2 K_3^2 K_4 K_5 K_6^2 K_7 K_8 + 2\phi\theta K_2 K_3 K_4 K_5 K_6^2 K_7 K_8 \tau_1 \\
&+ 2\phi K_2 K_3^2 K_4 K_5^2 K_6^2 K_7 K_8 + 2\phi K_2 K_3 K_4 K_5^2 K_6^2 K_7 K_8 \tau_1 + 2K_2 K_3^2 K_4^2 K_5^2 K_6^2 K_7 K_8 \\
&+ 2K_2 K_3 K_4^2 K_5^2 K_6^2 K_7 K_8 \tau_1, \\
Q_3 &= K_2^2 K_3^2 K_4^2 K_5^2 K_6^2 K_7^2 K_8 + (1 - (R_0^M)^2)
\end{aligned} \tag{28}$$

This implies that the model has an unstable endemic equilibrium point.

Sensitivity Analysis

The parameters for infection spread control determination are investigated through sensitivity analysis methods.

The Malaria model reproduces the sensitivity index of its reproduction number as a function of specific parameter p given by:

$$\begin{aligned}
\mathfrak{S}_p^{R_0^M} &= \frac{\partial R_0^M}{\partial p} \times \frac{p}{R_0^M}, \\
S_\theta^{R_0^M} &= -\frac{\theta}{2\theta+2\alpha+2\delta_1+2\mu_h} = -0.2054, S_\alpha^{R_0^M} = -\frac{\alpha}{2\theta+2\alpha+2\delta_1+2\mu_h} = -0.00924, \\
S_\phi^{R_0^M} &= \frac{\phi+2\mu_h}{2\phi+2\mu_h} = 0.50015, S_{\tau_1}^{R_0^M} = -\frac{\tau_1}{2\tau_2+2\tau_1+2\mu_h} = -0.14994, \\
S_{\tau_2}^{R_0^M} &= 1/2 \frac{(2\tau_1+\tau_2+\mu_h)\tau_2}{(\tau_2+\tau_1+\mu_h)(\tau_2+\mu_h)} = 0.64956, \\
S_{\beta_h}^{R_0^M} &= 1, S_{\beta_V}^{R_0^M} = \frac{1}{2}, S_{\Lambda_V}^{R_0^M} = \frac{1}{2}, \\
S_{\delta_h}^{R_0^M} &= -\frac{1}{2}, S_b^{R_0^M} = 1, S_{\delta_1}^{R_0^M} = -\frac{\delta_1}{2\theta+2\alpha+2\delta_1+2\mu_h} = -0.28523, \\
S_{\delta_3}^{R_0^M} &= -\frac{\delta_3}{2\delta_3+2\mu_V} = -0.2222, S_{\mu_V}^{R_0^M} = \frac{-\delta_3-2\mu_V}{2\delta_3+2\mu_V} = -0.7778.
\end{aligned} \tag{29}$$

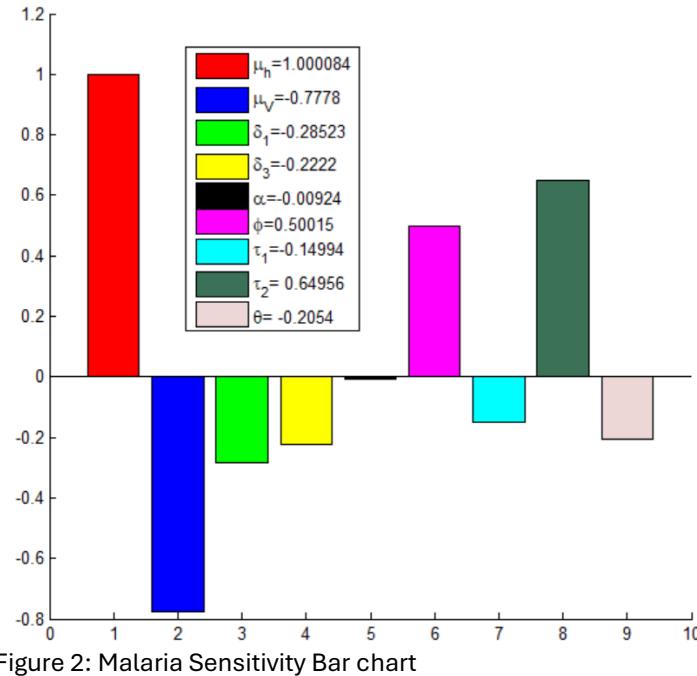


Figure 2: Malaria Sensitivity Bar chart

Interpretation of Malaria Sensitivity Bar Chart

The figure 2 shows the sensitivity indices of basic reproduction number of malaria disease. The value of the malaria model parameters is positive and specific values are used to establish the ability of the malaria model index to increase the spread of the disease when the index increases. When any parameter changes in a certain direction, the basic reproduction number will likewise change in that direction. Disease-burden reducing parameters increase in value when they act as disease protective factors which cause the reduced basic reproduction numbers.

Numerical Results of the Fractional-Order Model

To replicate the dynamics of our malaria model, we employed a numerical approach known as the generalized fractional Adams-Bashforth-Moulton method, based on the methodology outlined by Amos et al. (2024). The simulations were carried out using the parameter values presented in Table 1, with various fractional orders examined to assess their impact on the results (ρ)

Implementation of the Fractional Adams-Bashforth-Moulton Method

In this paper we use a fractional Adams-Bashforth-Moulton algorithm, as in the study of Diethelm (2012), and Baskonus et al. (2015), to estimate the solution of our fractional malaria model (6). The presentation of this model is modified after Amos et al. (2024) and it is provided as follows:

$${}^cD_t^\rho M(t) = N(t, m(t)), 0 < t < \psi,$$

$$M^{(n)}(0) = M_0^{(n)}, n = 1, 0, \dots, m, m = [\rho].$$

Where $M = (S_h^*, E_h^*, V_h^*, I_h^*, T_h^*, R_h^*, S_V^*, I_V^*) \in R_+^8$ and $Q(t, m(t))$ is a real valued function that is continuous.

Eq. (27) can be consequently be denoted using the notion of fractional integral as follows:

$$M(t) = \sum_{n=0}^{m-1} M_0^{(n)} \frac{t^n}{n!} + \frac{1}{\Gamma(\rho)} \int_0^t (t-y)^{\rho-1} R(y, m(y)) dy.$$

We apply the method described by Amos et al.(2024), let consider the step size $g = \frac{\psi}{N}, N \in N$ with a grid that is uniform on $[0, \psi]$. Where $t_c = cr, c = 0, 1, 1, \dots, N$. This implies that, the fractional order model of malaria model presented in (6) can approximately be expressed as:

$$\begin{aligned}
 S_{h(k+1)}(t) &= S_{h0} + \frac{g^\rho}{\Gamma(\rho+2)} \{ \Lambda_h + \tau_2 V_h^n + \omega R_h^n - \lambda_h S_h^n - (\tau_1 + \mu_h) S_h^n \} + \\
 &\quad \frac{g^\rho}{\Gamma(\rho+2)} \sum_{y=0}^k dy, k + 1 \{ \Lambda_h + \tau_2 V_{hy} + \omega R_{hy} - \lambda_h S_{hy} - (\tau_1 + \mu_h) S_{hy} \}, \\
 E_{h(k+1)}(t) &= E_{h0} + \frac{g^\rho}{\Gamma(\rho+2)} \{ \lambda_h S_h^n - (\phi + \mu_h) E_h^n \} + \\
 &\quad \frac{g^\rho}{\Gamma(\rho+2)} \sum_{y=0}^k dy, k + 1 \{ \lambda_h S_{hy} - (\phi + \mu_h) E_{hy} \}, \tag{30}
 \end{aligned}$$

$$\begin{aligned}
V_{h(k+1)}(t) &= V_{h0} + \frac{g^\rho}{\Gamma(\rho+2)} \{ \tau_1 S_h^n - (\tau_2 + \mu_h) V_h^n \} + \\
&\quad \frac{g^\rho}{\Gamma(\rho+2)} \sum_{y=0}^k dy, k+1 \{ \tau_1 S_{hy} - (\tau_2 + \mu_h) V_{hy} \}, \\
I_{h(k+1)}(t) &= I_{h0} + \frac{g^\rho}{\Gamma(\rho+2)} \{ \phi E_h^n - (\theta + \alpha + \delta_1 + \mu_h) I_h^n \} + \\
&\quad \frac{g^\rho}{\Gamma(\rho+2)} \sum_{y=0}^k dy, k+1 \{ \phi E_{hy} - (\theta + \alpha + \delta_1 + \mu_h) I_{hy} \}, \\
T_{h(k+1)}(t) &= T_{h0} + \frac{g^\rho}{\Gamma(\rho+2)} \{ \theta I_h^n - (\eta + \delta_2 + \mu_h) T_h^n \} + \\
&\quad \frac{g^\rho}{\Gamma(\rho+2)} \sum_{y=0}^k dy, k+1 \{ \theta I_{hy} - (\eta + \delta_2 + \mu_h) T_{hy} \}, \\
R_{h(k+1)}(t) &= R_{h0} + \frac{g^\rho}{\Gamma(\rho+2)} \{ \alpha I_h^n + \eta T_h^n - (\omega + \mu_h) R_h^n \} + \\
&\quad \frac{g^\rho}{\Gamma(\rho+2)} \sum_{y=0}^k dy, k+1 \{ \alpha I_{hy} + \eta T_{hy} - (\omega + \mu_h) R_{hy} \}, \\
S_{V(k+1)}(t) &= S_{V0} + \frac{g^\rho}{\Gamma(\rho+2)} \{ \Lambda_V - \lambda_V S_V^n - \mu_V S_V^n \} + \\
&\quad \frac{g^\rho}{\Gamma(\rho+2)} \sum_{y=0}^k dy, k+1 \{ \Lambda_V - \lambda_V S_{VY} - \mu_V S_{VY} \} \\
I_{V(k+1)}(t) &= I_{V0} + \frac{g^\rho}{\Gamma(\rho+2)} \{ \lambda_V S_V^n - (\delta_3 + \mu_V) I_V^n \} + \\
&\quad \frac{g^\rho}{\Gamma(\rho+2)} \sum_{y=0}^k dy, k+1 \{ \lambda_V S_{VY} - (\delta_3 + \mu_V) I_{VY} \}.
\end{aligned}$$

Where

$$\begin{aligned}
S_{h(k+1)}(t) &= S_{h0} + \frac{1}{\Gamma(\rho)} \sum_{y=0}^k f_{y,k+1} \{ \Lambda_h + \tau_2 V_{hy} + \omega R_{hy} - \lambda_h S_{hy} - (\tau_1 + \mu_h) S_{hy} \}, \\
E_{h(k+1)}(t) &= E_{h0} + \frac{1}{\Gamma(\rho)} \sum_{y=0}^k f_{y,k+1} \{ \lambda_h S_{hy} - (\phi + \mu_h) E_{hy} \}, \\
V_{h(k+1)}(t) &= V_{h0} + \frac{1}{\Gamma(\rho)} \sum_{y=0}^k f_{y,k+1} \{ \tau_1 S_{hy} - (\tau_2 + \mu_h) V_{hy} \}, \\
I_{h(k+1)}(t) &= I_{h0} + \frac{1}{\Gamma(\rho)} \sum_{y=0}^k f_{y,k+1} \{ \phi E_{hy} - (\theta + \alpha + \delta_1 + \mu_h) I_{hy} \}, \\
T_{h(k+1)}(t) &= T_{h0} + \frac{1}{\Gamma(\rho)} \sum_{y=0}^k f_{y,k+1} \{ \theta I_{hy} - (\eta + \delta_2 + \mu_h) T_{hy} \}, \\
R_{h(k+1)}(t) &= R_{h0} + \frac{1}{\Gamma(\rho)} \sum_{y=0}^k f_{y,k+1} \{ \alpha I_{hy} + \eta T_{hy} - (\omega + \mu_h) R_{hy} \}, \\
S_{V(k+1)}(t) &= S_{V0} + \frac{1}{\Gamma(\rho)} \sum_{y=0}^k f_{y,k+1} \{ \Lambda_V - \lambda_V S_{VY} - \mu_V S_{VY} \}, \\
I_{V(k+1)}(t) &= I_{V0} + \frac{1}{\Gamma(\rho)} \sum_{y=0}^k f_{y,k+1} \{ \lambda_V S_{VY} - (\delta_3 + \mu_V) I_{VY} \}.
\end{aligned} \tag{31}$$

We obtained the result below from (30) and (31).

$$\begin{aligned}
dy_{,k+1} &= K^{\rho+1} - (k - \rho)(k + \rho)^\rho, y = 0 \\
(k - y + 2)^{\rho+1} + (k - \rho)^{\rho+1} - 2(k - y + 1)^{\rho+1}, 1 \leq y \leq k \\
1, y = k + 1
\end{aligned}$$

and

$$f_{y,k+1} = \frac{g^\rho}{\rho} [(k - y + 1)^\rho (k - y)^\rho], 0 \leq y \leq k.$$

Numerical Simulation

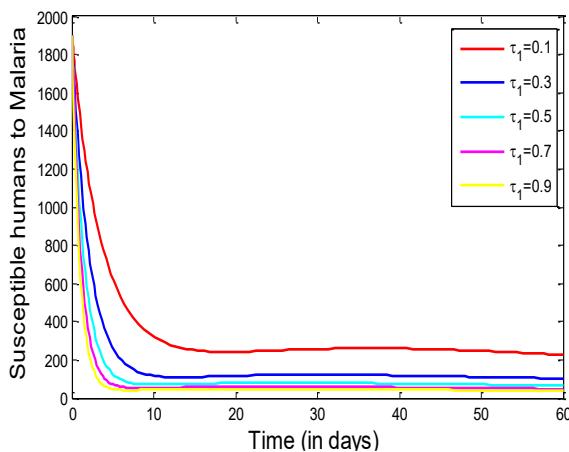


Figure 3a: Simulation of susceptible humans to malaria

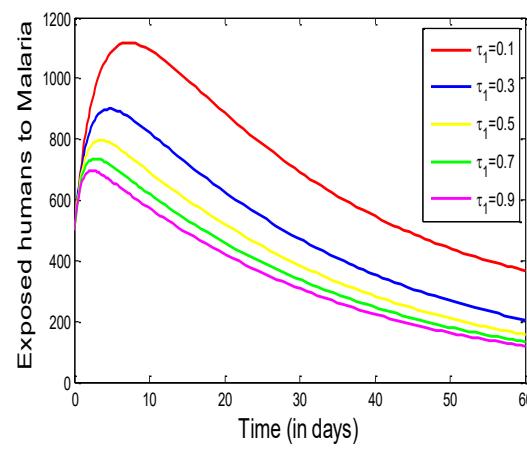


Figure 3b: Simulation of Exposed humans to malaria

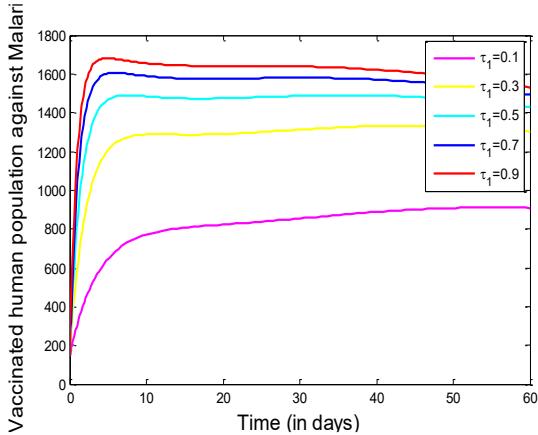


Figure 3c: Simulation of vaccinated humans against malaria

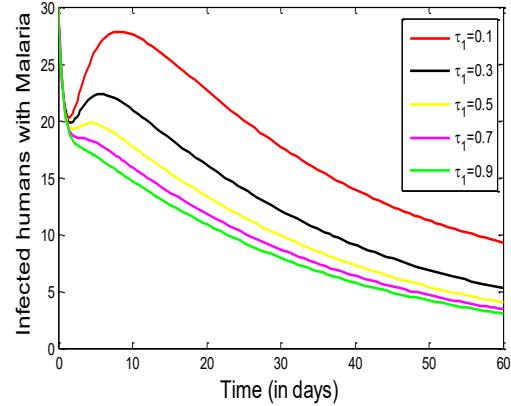


Figure 3d: Simulation of infected humans with malaria

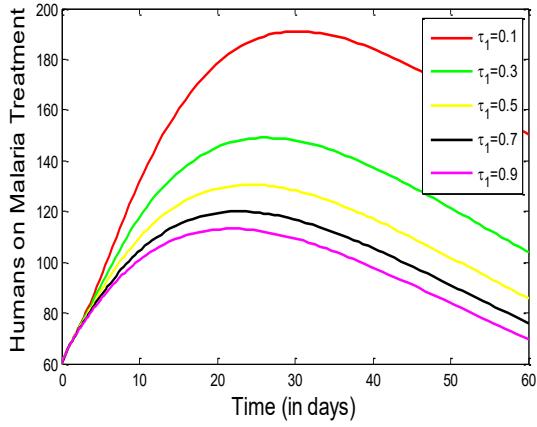


Figure 3e: Simulation of humans on treatment of malaria

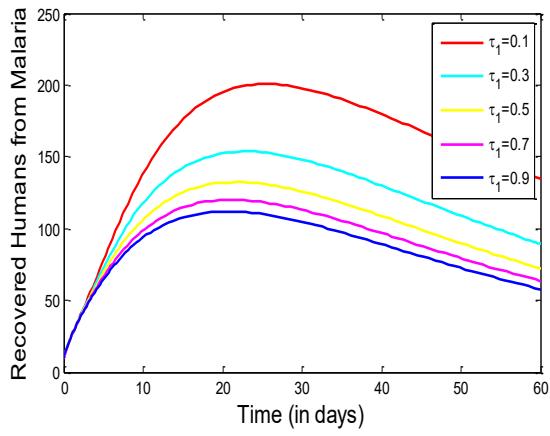


Figure 3f: Simulation of Recovered humans from malaria

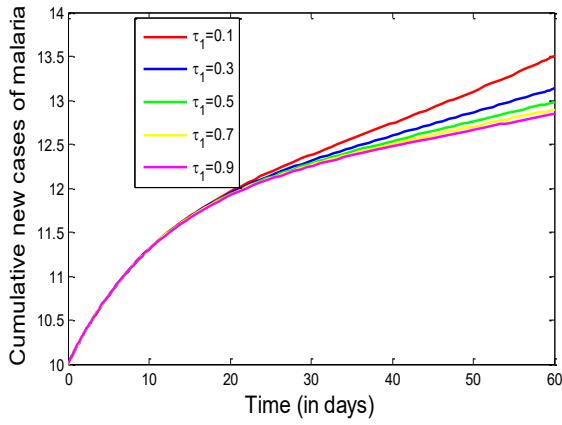


Figure 3g: Simulation of cumulative new cases of malaria

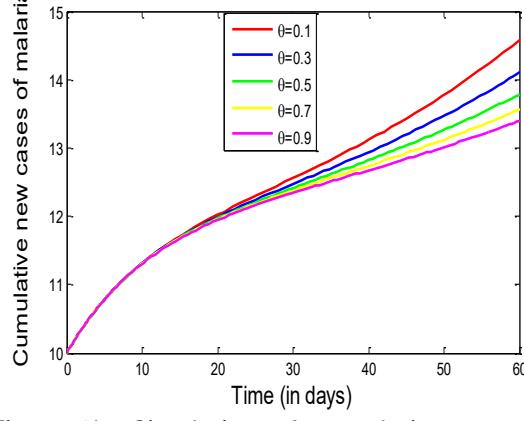


Figure 3h: Simulation of cumulative new cases of malaria

The Figure (3a) illustrates how the malaria rate among the susceptible population is simulated under the influence of the rate of vaccination(τ_1). It can be noted that, when the rate of vaccinated people (τ_1) is high, the number of susceptible people is less. Figure (3b) illustrates the simulation of the effect of the rate of vaccinated people (τ_1) on the malaria in the Exposed population. It is seen that, with the increase in the vaccinated rate (τ_1), the

number of Exposed individuals reduces. Figure (3c) illustrates the simulation of the influence of the vaccinated rate (τ_1) in the human population of malaria on the vaccinated individuals. It is seen that the higher the vaccination rate (τ_1) the higher the vaccinated human population.

Figure (3d) Demonstrate the modeling of the impact of the vaccinated rate (τ_1) of malaria in the infected human

population. One may also note that, with increase in the vaccinated rate (τ_1) on the one hand, there is a decreasing trend in the number of people infected with malaria in the treatment human population which is simulated in Figure (3e). It is noted that, the higher the rate of vaccination (τ_1), the more the human population is treated. Figure (3f) illustrates how the vaccinated rate (τ_1) of the vaccination affects the malaria in the recovered human population. As is seen, with the increasing vaccinated rate (τ_1), the recovered human population declines. Figure (3g)

illustrates the simulation of the vaccinated rate (τ_1) on malaria cumulative new cases of malaria. It is seen that, with increase in the vaccinated rate (τ_1) the cumulative new cases of malaria reduces. Figure (3h) shows the simulation of the rate of treatment (θ), that is, rate of vaccination (τ_1) on the cumulative new cases of malaria. It is noted that the more the treatment rate (θ) is, the lower the cumulative number of new cases of malaria.

Table 2 presents a comprehensive model parameters values and their sources employed in this study.

Table 2: Parameter Values and Sources

Parameter	Value	Source
Λ_h	0.564	Esteva et al.(2009)
Λ_V	0.245	Esteva et al.(2009)
μ_h	0.00004	Zi et al.(2012)
μ_V	0.05	Zi et al.(2012)
β_h	0.18	Esteva et al.(2009)
β_V	0.8333	Zi et al.(2012)
δ_1	0.0003454	Zi et al.(2012)
δ_2	0.03454	Assumed
δ_3	0.00003454	Assumed
ϕ	0.54	Zi et al.(2012)
b	0.1	Estimated
θ	0.43	Estimated
ω	0.0014	Zi et al.(2012)
α	0.3	Zi et al.(2012)
τ_1	0.67	Assumed
τ_2	0.43	Assumed

CONCLUSION

In this paper, we provide a mathematical model to analyze the malaria transmission and control measures that use the Caputo fractional derivative. Due to the importance of the fractional modeling, a detailed theoretical study of the fractional malaria model, in terms of the presence and uniqueness of solutions and the stability of the equilibrium points, was carried out. Fractional AdamsMoultonBashforth method, which is used in numerical solutions was employed. The impact of model parameters and various fractional orders of Caputo operator on the incidence of disease were studied by means of simulations. We also explored how the important parameters can be manipulated, including the rate of vaccination and treatment. The results show that both the rates of vaccination and treatment can be used to successfully decrease the incidence of malaria within the population. Further studies would be interested in the use of symbolic computing methods like those suggested by Zang et al (2022) to address nonlinear partial differential equations and find an analytical solution.

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