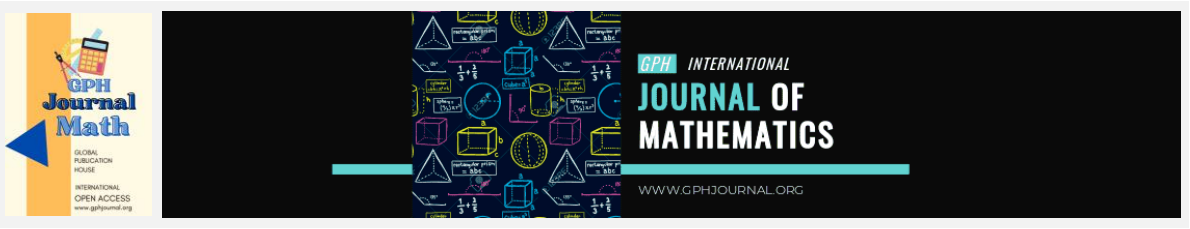
e-ISSN 3050-9645
p-ISSN 2795-3274






10.5281/zenodo.15205492 Vol. 8 Issue 03 March - 2025 Manuscript ID: #1852

# Numerical Solution of Fractional order Malaria Model via the Generalized Fractional Adams-Bashforth-Moulton Approach

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## Abstract

A fractional-order mathematical model studies the effects of contact rate and recovery rate on Malaria transmission dynamics as this paper investigates different epidemiological characteristics of malaria infection. We put forward conditions to ensure the model solution uniqueness and performs an endemic equilibrium stability assessment through Lyapunov function application. Numerical simulations running the fractional Adams–Bashforth–Moulton method reveal how fractional-order values together with model parameters affect malaria control and dynamics. Numerical surface and contour plots reveal that Malaria prevalence rises when both contact rates and recovery rate increase but the recovery rate enhances the population's resistance against the disease spread. Decreasing contact rate in the population results in lower prevalence rates of malaria in the population.

## *Keywords and phrases:*

Malaria, Fractional, Adam-Bashforth-Moulton, Transmission, Control, strategies.

***How to cite:*** Nuhu, A., Omale, D., Atokolo, W., Amos, J., Onuche Acheneje, G., Abah, E., Abah, E., Egbemhenghe, J., & Bolaji, B. (2025). Numerical Solution of Fractional order Malaria Model via the Generalized Fractional Adams-Bashforth-Moulton Approach. *GPH-International Journal of Mathematics*, 8(03), 01-25. https://doi.org/10.5281/zenodo.15205492

## 1.0 Introduction

Female anopheles' mosquitoes spread Malaria through biting humans to transmit the plasmodium parasite which lives inside humans as the parasite cause of Malaria. The protozoan plasmodium parasite has caused malaria endemics throughout large parts of the world since hundreds of thousands of years [1] yet continues to create significant public health burdens that primarily affect tropical and subtropical zones of Africa Asia and South America [2]. Malaria remains endemic in more than 100 countries across the world where it affects more than a third of the entire human population [2]. In 2010 the disease resulted in 216 million cases along with 655,000 mortalities [2,3]. Furthermore, malaria inflicts significant mortality among children under the age of five [4]. An effective and safe vaccine exists for treating human malaria but global research continues to create better versions of this vaccine [2]. The prevention of malaria relies on both preventive approaches involving mosquito population reduction and individual mosquito protection together with anti-malaria drug treatments according to [5,6,7,8,2,9].

For proper assessment of malaria disease transmission patterns and comparable diseases researchers require mathematical modeling as a fundamental tool. Research models enable scientists to uncover epidemic reasons while creating strategic defense plans. The conventional research models remain inadequate for their failure to incorporate natural biological memory effects as well as long-term dependencies within such systems. Fractional-order models solved the key drawback that existed in conventional approaches. Through non-local characteristics researchers gain access to incorporate memory effects and diffusion anomalies for disease transmission analyses [10].

Fractional differential equations (FDEs) serve as an improved modeling technique that develops integer-order models through a versatile system investigation framework. This research establishes a fractional-order mathematical model to study the spread of malaria by implementing prevention and treatment controls. Better disease spread representation occurs in the model because of its incorporation of fractional calculus memory effect properties. Different intervention strategies are simulated through the research to determine the most effective methods for malaria prevention while maintaining disease control measures.

Fractional derivatives, which capture memory and hereditary characteristics in biological systems, provide significant advantages in modeling diseases such as malaria. They allow for a more comprehensive analysis of infection progression over time and the impact of individuals' infection and treatment histories on transmission dynamics. This nuanced approach supports the design of more realistic and effective control strategies, tackling enduring issues like drug resistance, re-infection, and constraints in healthcare resources.

Recent advancements in fractional calculus, as emphasized in [11], have highlighted its effectiveness in describing the dynamic behavior of various systems. Unlike classical integer-order models that primarily address local properties, fractional-order models capture global system behavior, including memory effects. These models are not only more realistic but also

better suited for real-world applications, making them invaluable for understanding and controlling the spread of infectious diseases like malaria.

In biological applications, fractional derivatives such as the Caputo and Riemann-Liouville derivatives, which feature singular kernels, are extensively utilized. Additionally, non-singular kernel derivatives, including the Mittag-Leffler and Atangana-Baleanu operators, have gained prominence due to their improved applicability.

For instance, [11] introduced a fractional-order Sterile Insect Technology (SIT) model to control Zika virus transmission, employing the LADM technique to derive infinite series solutions converging to exact values. Likewise, [12] investigated Lassa fever dynamics using a fractional-order model with a power-law derivative to assess the effects of vaccination and treatment on disease spread.

Several other studies have explored fractional modeling in epidemiology. [13] applied a Caputo fractional-order derivative to model COVID-19 control in Nigeria, demonstrating that integer-order scenarios yielded higher recovery rates due to vaccination and treatment. Similarly, [14] developed a Caputo-based fractional-order compartmental model for soil-transmitted helminth infections, highlighting greater solution flexibility using LADM. In another study, [15] formulated a fractional model for hepatitis C transmission, utilizing the Adams-Bashforth-Moulton method to show that reducing contact rates and enhancing treatment significantly mitigated disease spread.

Further research by [16,17] employed fractional approaches to analyze HIV/AIDS and Diphtheria dynamics, respectively, underscoring the superior adaptability of fractional models compared to classical methods. [18] constructed a fractional model for chlamydia transmission, demonstrating through the Adams-Bashforth-Moulton method that lowering contact rates and improving treatment and vaccination rates effectively reduced disease spread. Additionally, [19] developed an ABC-fractional order model to examine the co-epidemic dynamics of HIV and COVID-19, while [20] reviewed hepatitis C and COVID-19 co-infection dynamics, identifying key methodologies and research gaps.

Lastly, Ullah et al. [21], as referenced by Das et al. [22], applied a hybrid Laplace transform and Adomian Decomposition Method to solve fuzzy Volterra integral equations, contributing to the theoretical advancement of fuzzy analytical dynamic equations.

Fractional-order models offer significant advantages due to their flexibility and capacity to capture non-local effects. Unlike classical derivatives, fractional derivatives provide a more precise representation of real-world phenomena by accounting for memory effects and incorporating non-local interactions—characteristics often missing in integer-order models. These features make fractional differential equations a valuable tool for tackling complex challenges in infectious disease modeling and other scientific fields.

Ali et al. [23] explored the stability and existence of solutions for a three-point boundary value problem, emphasizing different forms of Ulam stability. Their research employed classical nonlinear fractional techniques to analyze the problem, making notable contributions to the field.

The primary objectives of this paper are as follows:

- Determine the conditions that ensure the existence and uniqueness of solutions for the proposed fractional-order model.
- Analyze the stability of the endemic equilibrium point using the Lyapunov function method.
- Determine numerical solutions using the fractional Adams–Bashforth–Moulton method.
- Carry out numerical simulations to evaluate the model's behavior.

A review of existing literature on mathematical models and transmission dynamics of malaria highlights a gap in studies employing fractional calculus alongside the Adams–Bashforth–Moulton method to simulate and analyze malaria transmission and control strategies.

The organization of this paper is as follows: Section 2 outlines the formulation of the mathematical model, Section 3 explores its analytical properties, Section 4 presents numerical results for the fractional-order model, and Section 5 provides a summary and key findings.

Additionally, this section introduces fundamental concepts and results from fractional calculus. The study employs the right and left fractional Caputo derivatives as defined by [24,25]. Furthermore, the manuscript emphasizes the applications of fractional calculus in modeling real-world problems across various disciplines, including physics, engineering, bio-mathematics, and other scientific fields.

Definition 1: Let $f \in \Lambda^{\infty}(R)$, then the left and right Caputo fractional derivative of the function $f$ is given by

$$ {}^{c}D_{t}^{\gamma} f(t) = \left( {}^{t}0D_{t}^{-(m-\gamma)} \left( \frac{d}{dt} \right)^{m} f(t) \right) $$

$$ {}^{c}D_{t}^{\gamma} f(t) = \frac{1}{\Gamma(m-\gamma)} \int_{0}^{t} \left( (t-\lambda)^{m-\gamma-1} f^{m}(\lambda) \right) d\lambda \quad (1) $$

The same way

$$ {}_{T}^{c}D_{t}^{\gamma} f(t) = \left( {}_{t}D_{T}^{-(m-\gamma)} \left( \frac{-d}{dt} \right)^{m} f(t) \right) $$

$${}_{T}^{c}D_{T}^{\gamma} f(t) = \frac{(-1)^{m}}{\Gamma(m-\gamma)} \int_{t}^{T} \left( (\lambda - t)^{m-\gamma-1} f^{m}(\lambda) \right) d\lambda$$

Definition 2: The generalized Mittag-Leffler function $E_{\alpha,\beta}(x)$ for $x \in R$ is given by

$$E_{\alpha,\beta}(x) = \sum_{m=0}^{\infty} \frac{x^{m}}{\Gamma(\alpha m + \beta)}, \alpha, \beta > 0 \quad (2)$$

which can also be represented as

$$E_{\alpha,\beta}(x) = xE_{\alpha,\alpha+\beta(x)} + \frac{1}{\Gamma(\beta)} \qquad (3)$$

$$E_{\alpha,\beta}(x) = L\left[ t^{\beta-1} E_{\alpha,\beta(\pm\psi t^{\alpha})} \right] = \frac{S^{\alpha-\beta}}{S^{\alpha} \pm \psi} \qquad (4)$$

Proposition 1.1.

Let $f \in \Lambda^{\infty}(R) \cap C(R)$ and $\alpha \in R,\ m-1 < \alpha < m,$

Therefore, the conditions given below holds:

1. ${}_{t_0}^{c}D_{t}^{\gamma} I^{\gamma} f(t) = f(t)$

2. $I_{t_0}^{\gamma} D_{t}^{\gamma} f(t) = f(t) - \sum_{k=0}^{m-k} \frac{t^{k}}{k!} f^{k}(t_o).$

## 2.0 Material and Method

### 2.1 Model Formulation

The rate at which individuals enter the susceptible population is represented as $\pi_H$ so that $(\beta_M)$ Represents the effective contact rate between susceptible individuals and infected Anopheles mosquitoes. We denote $(\theta_M)$ Represents the recovery rate of humans infected with malaria, $\tau_{H1}$ is the progression rates from $E_{VM}$ compartment to $I_{VM}$ compartment. $\alpha_M$ is the progression rate from $E_M$ compartment to $I_M$ compartment. $\beta_{VM}$ transmission probability of malaria from infected humans to susceptible anopheles mosquitoes. The natural death rate of humans is denoted as $\delta_M$. Humans die due to the infectiousness of malaria at the rate $\delta_M$.

## 2.2 Model Flow Diagram

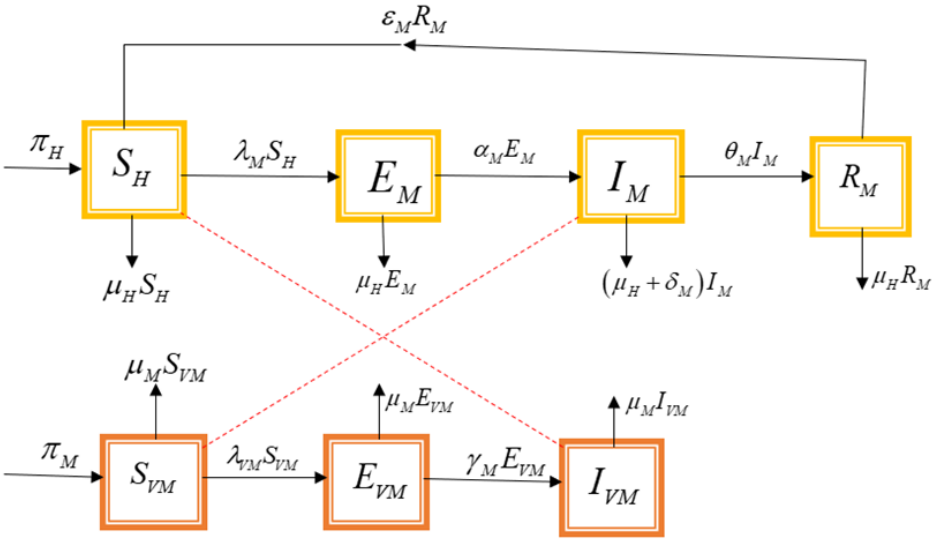


Fig.1: Malaria Model Flow Diagram

## 2.3 Model Equation

$$\frac{dS_H}{dt} = \pi_H - \frac{(1-\phi)\,\mathrm{bm}\,\beta_m I_{vm} S_H}{N} S - \mu_H S_H + \varepsilon_M \mathrm{R}_M,$$

$$\frac{dE_M}{dt} = \frac{(1-\phi)\,\mathrm{bm}\,\beta_m I_{vm} S_H}{N_H} - (\alpha_M + \mu_H)\,\mathrm{E}_M,$$

$$\frac{dI_M}{dt} = \alpha_M \mathrm{E}_M - (\theta_M + \delta_M + \mu_H) I_M$$

$$\frac{dR_M}{dt} = \theta_M I_M - (\varepsilon_M + \mu_H) R_M$$

$$\frac{dS_{VM}}{dt} = \pi_M - \frac{\mathrm{bm}\,\beta_m I_{vm} S_{VM}}{N_H} - \mu_M S_{VM}$$

$$\frac{dE_{VM}}{dt} = \frac{\mathrm{bm}\,\beta_m I_{vm} S_{VM}}{N_H} - (\gamma_M + \mu_M) E_{VM}$$

$$\frac{dI_{VM}}{dt} = \gamma_M E_{VM} - \mu_M I_{VM}$$

Where

$$\lambda_M = \frac{(1-\phi)\,\mathrm{bm}\,\beta_m I_{VM}}{N_H}$$

$\lambda_{VM} = \frac{\mathrm{bm}\,\beta_{Vm} I_M}{N_H}$ is the force of infection.

### 2.4 Table of Model variables and parameters

| **VARIABLE** | **DESCRIPTION** |
|---|---|
| $S_H$ | Susceptible Humans |
| $E_M(t)$ | Exposed humans to Malaria |
| $I_M(t)$ | Infected humans with Malaria only |
| $R_M(t)$ | Recovered humans from Malaria only |
| $S_{VM}(t)$ | Susceptible Anopheles mosquitoes |
| $E_{VM}(t)$ | Exposed Anopheles to infected humans |
| $I_{VM}(t)$ | Infected Anopheles mosquitoes with Malaria |
| **Parameter** | **Description** |
| $\pi_H(\pi_M)$ | The rate at which humans (Anopheles mosquitoes, Aedes aegypti mosquitoes) are introduced into the population. |
| $\mu_H$ | Natural death rate for humans |
| $\beta_M$ | The likelihood of malaria transmission from infected Anopheles mosquitoes to susceptible humans. |
| $\delta_M$ | Malaria disease-induced death rate for humans |
| $b_M$ | Mosquito-biting rate for malaria only |
| $\beta_{VM}$ | The probability of malaria transmission from infected humans to susceptible Anopheles mosquitoes. |
| $\mu_H$ | Progression rate from $E_M$ compartment to $I_M$ compartment. |
| $\theta_M$ | Recovery rate of infected humans with malaria only. |
| $\varepsilon_M$ | Waning rate of infected humans with malaria only. |
| $\tau_M$ | Progression rate from $E_{VM}$ compartment to $I_{VM}$ compartment. |
| $\phi$ | Compliance rate to the usage of bed net |

### 3.1 Fractional Malaria mathematical model

In this section, we extend the integer-order malaria model from Eq. (5) by incorporating the Caputo fractional derivative operator. This modified model offers greater flexibility compared to the classical model in Eq. (5), as the fractional-order formulation allows for varying outputs and diverse response behaviors. The fractional malaria model is thus presented as follows:

$$^{C}D_t^{\gamma} S_H = \pi_H - \frac{(1-\phi) bm\beta_m I_{VM}}{N_H} S_H - A_1 S + \varepsilon_M R_M$$

$$^{c}D_t^{\gamma} E_H = \lambda_H S_H - \left(\alpha_M + \mu_H\right) \mathrm{E}_M - \mathrm{A}_2 \mathrm{E}_M,$$

$$^{c}D_t^{\gamma} I_M = \alpha_M \mathrm{E}_M - \left(\theta_{M1} + \delta_M + \mu_H\right) \mathrm{I}_M - \mathrm{A}_3 \mathrm{A},$$

$$^{c}D_t^{\gamma} R_M = \theta_{M1} I_M - \left(\varepsilon_M + \mu_H\right) R_M, \quad (6)$$

$$^{c}D_t^{\gamma} S_V = \pi_M - \frac{bm\beta_{vm} I_M S_{VM}}{N_H} - \mu_M S_{VM}$$

$$^{c}D_t^{\gamma} E_V = \lambda_V S_{VM} - \left(\tau_M + \mu_M\right) E_{VM}.$$

$$^{c}D_t^{\gamma} I_V = \tau_V E_{VM} - \mu_M I_{VM}.$$

Subject to positive initial conditions

$$S_H(0) = S_{H0},\ \mathrm{E}_M(0) = E_{M0},\ \mathrm{I}_M(0) = I_{M0}, R_M(0) = R_{M0},\ S_V(0) = S_0, \mathrm{E}_V(0) = E_{V0}, \mathrm{I}_V = \mathrm{I}_{V0}.$$

### 3.2 Positivity of model solution

We considered the non-negativity of the initial values

$$N(t) \le \frac{\pi}{\mu} \quad \text{as} \quad t \to \infty$$

Secondly, if $\limsup N_0(t) \le \frac{\pi}{\mu}$, then our model feasible domain is given by:

$$\Omega = \left\{ \left(S_H, E_M, I_M, \mathrm{R}_M, \mathrm{S}_M, \mathrm{E}_V\right) \subset R_+^7 : S + E_M + I_M + R_M + S_{VM} + E_{VM} + I_{VM} \le \frac{\pi}{\mu}, \right\}, \text{ so that}$$

$$\Omega = \Omega_H \subset R_+^7,$$

hence, $\Omega$ is positively invariant.

If $S_0, E_{M0}, I_{M0}, \mathrm{R}_{M0}, \mathrm{S}_{V0}, E_{V0}, \mathrm{I}_{V0}$. are non-negative, then the solution of model (6) will be non-negative for $t > 0$. From Eq. (6), picking the first equation, we have that

$$^{C}D_t^{\gamma} S_H = -\lambda_H S_H - A_1 S_H + \varepsilon_M R_M$$

$$^{C}D_t^{\gamma} S_H = \pi_H - (\lambda_H + A_1) S_H + \varepsilon_M R_M$$

$$^{C}D_t^{\gamma} S_H + (\lambda_H + A_1) S_H + \varepsilon_M R_M = \pi$$

But $\pi \ge 0$ then

$${}^{C}D_t^{\gamma} S_H + (\lambda_H + A_1) S_H + \varepsilon_M R_M \geq 0.$$

Applying the Laplace transform we obtained;

$$L\left[{}^{C}D_t^{\gamma} S_H\right] + L\left[(\lambda_H + A_1) S_H\right] \geq 0$$

$$S_H^{\gamma} S_H(s_H) - S_H^{\gamma-1} S_H(0) + (\sigma_H + \mu) S_H(s_H) \geq 0,$$

$$S_H(s_H) \geq \frac{S_H^{\gamma-1}}{S_H^{\gamma} + (\sigma_H + \mu)} S_H(0).$$

By taking the inverse Laplace transform, we obtained ;

$$S_H(t) \geq E_{Mr,1}\left(-(\lambda_H + \mu) t^{\gamma}\right) \mathrm{S}_{H0}. \quad \ldots(9)$$

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 $E_M \geq 0$ , $I_M \geq 0, R_M \geq 0, S_V \geq 0, \mathrm{E}_V \geq 0$ , $\mathrm{I}_V \geq 0.$ that is are positives, therefore, the solution will remain in $R_+^7$ for all $t \geq 0$ with positive initial conditions.

**3.3 Boundedness of fractional model solution.**

The total population of individuals from our model is given by;

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

$${}^{c}D_t^{\gamma} N(t) = {}^{c}D_t^{\gamma} S_H(t) + {}^{c}D_t^{\gamma} E_M(t) + {}^{c}D_t^{\gamma} I_M(t) + {}^{c}D_t^{\gamma} R_M(t) + {}^{c}D_t^{\gamma} S_V(t) + {}^{c}D_t^{\gamma} E_V(t), {}^{c}D_t^{\gamma} I_V(t)$$

$${}^{c}D_t^{\gamma} N(t) = \pi_H - \mu N(t) \qquad (10)$$

Taking the Laplace transformation of (10) we obtained;

$$L\left[{}^{c}D_t^{\gamma} N(t)\right] = L\left[\pi_H - \mu N(t)\right]$$

$$S_H^{\gamma} N(\mathrm{s}_H) - S_H^{\gamma-1} N(0) + \mu N(s) \leq \frac{\pi}{\mu},$$

$$N(\mathrm{s}_H) \leq \frac{S_H^{\gamma-1}}{\left(S^{\gamma} + \mu\right)} N(0) + \frac{\pi}{S_H\left(S_H^{\gamma} + \mu\right)} \qquad (11)$$ By taking the inverse Laplace transform of Eq. (11) we obtained;

$$N(\mathrm{t}) \leq E_{Mr,1}\left(-\mu t^{\gamma}\right) N(0) + \pi E_{Mr,\mathrm{r}+1}\left(-\mu t^{\gamma}\right) \quad (12)$$

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

$$\lim_{t \to \infty} Sup N(t) = \frac{\pi}{\mu}.$$

This means that, if $N_0 \le \frac{\pi_H}{\mu_H}$ then $N_H(t) \le \frac{\pi_H}{\mu_H}$ which implies that, $N_H(t)$ is bounded.

We now conclude that, this region $\Omega = \Omega_H$, is well posed and equally feasible epidemiologically

### 3.4 Existence and uniqueness of our model solution

Let the real non-negative be J, we consider $L = [0, \mathrm{K}]$.

The set of all continuous function that is defined on M is represented by $N_e^0(L)$ with norm as;

$$\|X\| = Sup\left\{\left|K(t)\right|, \mathrm{t} \in L\right\}.$$

Considering model (6) with initial conditions presented in (8) which can be denoted as an initial value problem (IVP) in (13).

$${}^{c}D_t^{\gamma}(t) = Z\left(t, X(t)\right), 0 < t < J < \infty,$$

$$X(0) = X_0.$$

Where $Y(t) = \left(S_H(t), E_M(t), I_M(t), R_M(t), S_{VM}(t), \mathrm{E}_{VM}(t), \mathrm{I}_{VM}(t)\right).$ represents the classes and Z be a continuous function defined as follows;

$$Z\left(t, X(t)\right) = \begin{pmatrix} Z_1\left(t, S_H(t)\right) \\ Z_2\left(t, \mathrm{E}_M(t)\right) \\ Z_3\left(t, \mathrm{I}_M(t)\right) \\ Z_4\left(t, \mathrm{R}_M(t)\right) \\ Z_5\left(t, \mathrm{S}_V(t)\right) \\ Z_6\left(t, \mathrm{E}_V(t)\right) \\ Z_7\left(t, \mathrm{I}_V(t)\right) \end{pmatrix} = \begin{pmatrix} \pi_H - \left(\frac{(1-\phi)\,\mathrm{bm}\,\beta_M I_{VM}}{N_H} + \mu\right) S_H + \varepsilon_M\, \mathrm{R}_M. \\ \left(\frac{(1-\phi)\,\mathrm{bm}\,\beta_M I_{VM}}{N_H} + \mu\right) S_H - (\alpha_M + \mu)\,\mathrm{E}_M. \\ \alpha_M E_M - (\theta_M + \delta_M + \mu) I_M \\ \theta_M I_M - (\varepsilon_M + \mu) R_M \\ \pi_H - \left(\frac{\mathrm{bm}\,\beta_M I_{VM} S_{VM}}{N_H} - \mu_M S_{VM}\right) \\ \frac{bm\beta_{VM} I_M S_{VM}}{N_H} - (\tau_M + \mu) E_{VM} \\ \tau_M E_{VM} + \mu_M I_{VM} \end{pmatrix} \quad (14)$$

Using proposition (2.1), we have that,

$$S_H(t) = S_{H0} + I_t^{\gamma}\left[\pi_H - \left(\frac{(1-\phi)bm\beta_m I_{VM}}{N_H} + \mu\right) S_H + \varepsilon_M\, \mathrm{R}_M.\right],$$

$$E_M(t) = E_{M0} + I_t^{\gamma}\left[\left(\frac{(1-\phi)\,\mathrm{bm}\,\beta_M I_{VM}}{N} + \mu\right)S_H - (\alpha_M + \mu)\,\mathrm{E}_M\right], \qquad (15)$$

$$I_M(t) = I_{M0} + I_t^{\gamma}\left[\alpha_M E_M - (\theta_M + \delta_M + \mu)I_M\right],$$

$$R_M(t) = R_{M0} + I_t^{\gamma}\left[\theta_M I_M - (\varepsilon_M + \mu)R_M\right],$$

$$S_V(t) = S_{V0} + I_t^{\gamma}\left[\pi_H - \frac{(1-\phi)bm\beta_{vm}I_M}{N} - \mu\right]S_{VM}.$$

$$E_V(t) = E_{V0} + I_t^{\gamma}\left[\frac{(1-\phi)bm\beta_{vm}I_M}{N} + \mu\right]S_{VM} - (\tau_M + \mu)E_{VM}$$

$$I_V(t) = I_{V0} + I_t^{\gamma}\left[(\tau_M E_{VM}) - \mu_M I_{VM}\right]$$

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

$$S_{Hn}(t) = S_{H0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_M)^{\gamma-1} Z_1\left(\lambda_M, S_{Hn-1}(\lambda_M)\right)\mathrm{d}\lambda_M,$$

$$E_{Mn}(t) = E_{M0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_M)^{\gamma-1} Z_2\left(\lambda_M, \mathrm{E}_{\mathrm{M}(n-1)}(\lambda_M)\right)\mathrm{d}\lambda_M,$$

$$I_{Mn}(t) = I_{M0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_M)^{\gamma-1} Z_3\left(\lambda_M, I_{M(n-1)}(\lambda_M)\right)\mathrm{d}\lambda_M,$$

$$R_{Mn}(t) = R_{M0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_M)^{\gamma-1} Z_4\left(\lambda_M, \mathrm{R}_{M(n-1)}(\lambda_M)\right)\mathrm{d}\lambda_M,$$

$$S_V(t) = S_{V0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_M)^{\gamma-1} Z_5\left(\lambda_M, \mathrm{S}_{\mathrm{V}(n-1)}(\lambda_M)\right)\mathrm{d}\lambda_M. \qquad (16)$$

$$E_{Vn}(t) = E_{V0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_M)^{\gamma-1} Z_6\left(\lambda_M, \mathrm{E}_{M(n-1)}(\lambda_M)\right)\mathrm{d}\lambda_M,$$

$$I_{Vn}(t) = I_{V0} + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_M)^{\gamma-1} Z_6\left(\lambda_M, \mathrm{I}_{V(n-1)}(\lambda_M)\right)\mathrm{d}\lambda_M,$$

Lemma 2. The initial value problem (6), (7) in Eq. (19) exists and will have a unique solution $\mathrm{X}(t) \in A_c^0(f)$.

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

$$\mathrm{X}(t) = S_H\left(\mathrm{X}(t)\right),$$

where S is defined as the Picard operator expressed as ;

$$S_H : A_c^0\left(f, R_+^7\right) \to A_c^0\left(f, R_+^7\right).$$

Therefore

$$S_H\left(X(t)\right) = X(0) + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_m)^{\gamma-1} Z\left(\lambda_m, \mathrm{X}(\lambda_m)\right)\mathrm{d}\lambda_m.$$

which becomes

$$\left\|S_H\left(\mathrm{X}_1(t)\right) - S_H\left(\mathrm{X}_2(t)\right)\right\|$$

$$= \left\|\frac{1}{\Gamma(\gamma)}\left[\int_0^t (t-\lambda_m)^{\gamma-1} Z\left(\lambda_m, \mathrm{X}_1(\lambda_m)\right) - Z\left(\lambda_m, \mathrm{X}_2(\lambda_m)\right)\mathrm{d}\lambda_m\right]\right\|$$

$$\leq \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_m)^{\gamma-1}\left\|Z\left(\lambda_m, \mathrm{X}_1(\lambda_m)\right) - Z\left(\lambda_m, \mathrm{X}_2(\lambda_m)\right)\mathrm{d}\lambda_m\right\|.$$

$$\leq \frac{\psi}{\Gamma(\gamma)}\int_0^t (t-\lambda_m)^{\gamma-1}\left\|\mathrm{X}_1 - \mathrm{X}_2\right\| d\lambda_m.$$

$$\left\|S_H\left(\mathrm{X}_1(t)\right) - S_H\left(\mathrm{X}_2(t)\right)\right\| \leq \frac{\psi}{\Gamma(\gamma+1)S_H}.$$

When $\frac{\psi}{\Gamma(\gamma+1)}S_H \leq 1$, then the Picard operator gives a contradiction, soEq. (6), (7) solution is unique.

We now transformed the initial value problem of Eq. (13) to obtain;

$$X(t) = X(0) + \frac{1}{\Gamma(\gamma)}\int_0^t (t-\lambda_m)^{\gamma-1} Z\left(\lambda_m, X(\lambda_m)\right)\mathrm{d}\lambda_m. \quad (17)$$

Lemma 1, The Lipchitz condition described from Eq. (14) is satisfied by vector $Z\left(\mathrm{t}, X(t)\right)$ on a set $[0, \mathrm{L}]\times R_+^7$ with the Lipchitz constant given as;

$$\psi = \max\left(\left(\beta_{M1}^* + \beta_{M2}^* + \beta_{M3}^* + \mu\right), (\alpha_M + \mu), (\theta_M + \delta_M + \mu), (\varepsilon_M + \mu), \left(\beta^*_{V1} + \beta^*_{V2} + \beta^*_{M3} + \mu\right), (\tau_M + \mu), (\tau_M + \mu)\right)$$

Proof.

$$\left\|Z_1\left(t, S_H\right) - Z_1\left(t, S_{H1}\right)\right\|$$

$$= \left\| \pi_H - \left( \frac{(1-\phi)bm\beta_m I_{VM}}{N} + \mu \right) S_H + \varepsilon_M R_M - \pi_H - \left( \frac{(1-\phi)bm\beta_m I_{VM}\beta_H}{N} + \mu \right) S_{H1} + \varepsilon_M R_M \right\|$$

$$\left\| -\pi_H - \left( \frac{(1-\phi)bm\beta_m I_{VM}}{N} + \mu \right)(S_H - S_{H1}) + \mu(S_H - S_{H1}) \right\| \leq \left( \beta_{M1}{}^* + \beta_{M2}{}^* + \beta_{M3}{}^* \right) \|S_H - S_{H1}\| + \mu \|S_H - S_{H1}\|$$

$$\therefore \|Z_1(t, S_H) - Z_1(t, S_{H1})\| \leq \left( \beta_{M1}{}^* + \beta_{M2}{}^* + \beta_{M3}{}^* + \mu \right) \|S_H - S_{H1}\|$$

Similarly, we obtained the following;

$$\|Z_2(t, \mathrm{E}_M) - Z_2(t, \mathrm{E}_{M1})\| \leq (\alpha_M + \mu) \|\mathrm{E}_M - \mathrm{E}_{M1}\|,$$

$$\|Z_3(t, \mathrm{I}_A) - Z_3(t, \mathrm{I}_{M1})\| \leq (\theta_{M1} + \delta_M + \mu) \|\mathrm{I}_M - \mathrm{I}_{M1}\|,$$

$$\|Z_4(t, \mathrm{R}_M) - Z_4(t, \mathrm{R}_{M1})\| \leq (\varepsilon_M + \mu) \|R_M - R_{M1}\|$$

$$\|Z_5(t, \mathrm{S}_V) - Z_5(t, \mathrm{S}_{M1})\| \leq \left( \beta^*{}_{V1} + \beta^*{}_{V2} + \beta^*{}_{V3} + \mu \right) \|S_M - S_{M1}\|, \tag{18}$$

$$\|Z_6(t, \mathrm{E}_V) - Z_6(t, \mathrm{E}_{V1})\| \leq (\tau_V + \mu) \|E_V - E_{V1}\|.$$

$$\|Z_7(t, \mathrm{I}_V) - Z_7(t, \mathrm{I}_{V1})\| \leq (\tau_V + \mu) \|I_V - I_{V1}\|.$$

Where we obtained

$$\|Z(t, \mathrm{X}_1(t)) - Z(t, \mathrm{X}_2(t))\| \leq \psi \|\mathrm{X}_1 - \mathrm{X}_2\|,$$

$$\psi = \max\left( \left( \beta_{M1}{}^* + \beta_{M2}{}^* + \beta_{M3}{}^* + \mu \right), (\alpha_M + \mu), (\theta_M + \delta_M + \mu), (\varepsilon_M + \mu), \left( \beta^*{}_{V1} + \beta^*{}_{V2} + \beta^*{}_{M3} + \mu \right), (\tau_M + \mu), (\tau_M + \mu) \right)$$

(19).

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

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

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

$$\mathrm{X}(t) = S_H(\mathrm{X}(t)),$$

where S is defined as the Picard operator expressed as ;

$$S_H : A_c^0(f, R_+^7) \to A_c^0(f, R_+^7).$$

Therefore

$$S_H(X(t)) = X(0) + \frac{1}{\Gamma(\gamma)} \int_0^t (t - \lambda_M)^{\gamma - 1} Z(\lambda_M, \mathrm{X}(\lambda_M)) \mathrm{d}\lambda_M.$$

which becomes

$$\left\|S_H\left(\mathrm{X}_1(t)\right)-S_H\left(\mathrm{X}_2(t)\right)\right\|$$

$$=\left\|\frac{1}{\Gamma(\gamma)}\left[\int_0^t(t-\lambda_M)^{\gamma-1}Z\left(\lambda_M,\mathrm{X}_1(\lambda_M)\right)-Z\left(\lambda_M,\mathrm{X}_2(\lambda_M)\right)\mathrm{d}\lambda_M\right]\right\|$$

$$\leq\frac{1}{\Gamma(\gamma)}\int_0^t(t-\lambda_H)^{\gamma-1}\left\|Z\left(\lambda_M,\mathrm{X}_1(\lambda_M)\right)-Z\left(\lambda_M,\mathrm{X}_2(\lambda_M)\right)\mathrm{d}\lambda_M\right\|.$$

$$\leq\frac{\psi}{\Gamma(\gamma)}\int_0^t(t-\lambda_M)^{\gamma-1}\left\|\mathrm{X}_1-\mathrm{X}_2\right\|d\lambda_M.$$

$$\left\|S_H\left(\mathrm{X}_1(t)\right)-S_H\left(\mathrm{X}_2(t)\right)\right\|\leq\frac{\psi}{\Gamma(\gamma+1)S_H}.$$

When $\frac{\psi}{\Gamma(\gamma+1)}S_H\leq 1$, then the Picard operator gives a contradiction , so Eq.(6) , (7) solution is unique.

### 3.5 The basic reproduction number ($R_0$) and model equilibrium points:

The disease-free equilibrium points of the model (5) is expressed as:

$$(\mathrm{HDFEP})=\left(\left(S_H^*,E_M^*,I_M^*,\mathrm{R}_M^*,\mathrm{S}_V^*,\mathrm{E}_V^*,\mathrm{I}_V^*\right)=\left(\frac{\pi}{\mu},0,0,0,\frac{\pi}{\mu},0,0\right)\right) \quad (20)$$

*Let* $n=\left(E_M,I_M,\mathrm{E}_V,\mathrm{I}_V\right)$

*So that* $\frac{dn}{dt}=F-V.$

$$F_{0M}=\begin{bmatrix}0&\beta_{M1}&\beta_{M2}&\beta_{M3}&0\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\\0&0&0&0&0\end{bmatrix},\quad V_{0M}=\begin{bmatrix}K_1&0&0&0&0\\-\alpha_M&K_2&0&0&0\\0&0&K_3&0&0\\0&0&0&K_4&0\\0&0&0&\tau_M&\mu_M\end{bmatrix}$$

Where $K_1=\left(\alpha_M+\mu_H\right)$, $K_2=\left(\theta_M+\delta_M+\mu_H\right), K_3=\left(\varepsilon_M+\mu_H\right), K_4=\left(\tau_M+\mu_H\right),$ $P_5=\left(\delta_H+\mu\right)$

Mathematically, the basic reproduction number is computed as $R_0=\rho\left(FV^{-1}\right)$ where $\rho$ is the dominant Eigen value of the system $\left(FV^{-1}\right)$. Where $R_0^M$ is the basic reproduction number associated with the individuals in the population.

$$R_0^M=\sqrt{\frac{(1-\phi)b^2m\beta_M\beta_{VM}\pi_M\mu_H\alpha_M\tau_M}{\pi_H\mu^2{}_MK_1K_2K_3}}$$

### 3.6 Endemic equilibrium point

We investigated the potential existence of an endemic equilibrium point, which represents a positive steady state where malaria remains present within the population. At this equilibrium, all model variables maintain nonzero values.

$\left(S_H^* \neq 0, E_M^* \neq 0, I_M^* \neq 0, \mathrm{R}_M^* \neq 0 \ , \mathrm{S}_{VM}{}^* \neq 0 \ , E_{VM}^* \neq 0 \,\text{and}\, \mathrm{I}_{VM}^* \neq 0 \ \right)$.

To analyze the endemic equilibrium point, the model equations are solved based on the force of infection affecting the human population. From the fractional malaria model (6), the endemic equilibrium state is expressed as follows:

$E_{**} = \left(S_H^{**}, E_M^{**}, I_M^{**}, R_M^{**} \ , S_{VM}{}^{**} E_{VM}^{**}, \mathrm{I}_{VM}{}^{**}\right),$

defined as;

$$S_H{}^{**} = \frac{\pi_H K_1 K_2 K_3}{(\lambda_M^{**} + \mu_H) K_1 K_2 K_3 - \lambda_M^{**} \alpha_M \theta_M \varepsilon_M},$$

$$E_M{}^{**} = \frac{\lambda_M^{**} \pi_H K_1 K_2 K_3}{(\lambda_M^{**} + \mu_H) K_1 K_2 K_3 - \lambda_M^{**} \alpha_M \theta_M \varepsilon_M},$$

$$I_M{}^{**} = \frac{\lambda_M^{**} \pi_H \alpha_M K_3}{(\lambda_M^{**} + \mu_H) K_1 K_2 K_3 - \lambda_M^{**} \alpha_M \theta_M \varepsilon_M},$$

$$R_M{}^{**} = \frac{\lambda_M^{**} \pi_H \alpha_M \theta_M}{(\lambda_M^{**} + \mu_H) K_1 K_2 K_3 - \lambda_M^{**} \alpha_M \theta_M \varepsilon_M},$$

$$S_{VM}{}^{**} = \frac{\pi_M}{(\lambda_{VM}^{**} + \mu_M)},$$

$$E_{VM}{}^{**} = \frac{\lambda_{VM}^{**} \pi_M}{(\lambda_{VM}^{**} + \mu_M) \mathrm{K}_4},$$

$$I_{VM}{}^{**} = \frac{\lambda_{VM}^{**} \pi_M \tau_M}{(\lambda_{VM}^{**} + \mu_M) \mu_M \mathrm{K}_4},$$

Where $K_1 = \left(\alpha_M + \mu_H\right),\ K_2 = \left(\theta_M + \delta_M + \mu_H\right), K_3 = \left(\varepsilon_M + \mu_H\right), K_4 = \left(\tau_M + \mu_H\right),$

Substituting into the force of infection $\lambda_M{}^{**} = \dfrac{(1-\phi) bm \beta_M I_{VM}{}^{**}}{N_H{}^{**}}$ and

$$\lambda_{VM}{}^{**} = \frac{bm \beta_{vm} I_M^{**}}{N_H{}^{**}},$$

$$f(\lambda_M{}^{**}) = A_1 \lambda_M{}^{**2} + A_2 \lambda_M{}^{**} + A_3$$

Where

$$A_1 = \pi^2{}_H \mu_M K_4 (bm \beta_{VM} \alpha_M K_3 + \mu_M (K_2 K_3 + \alpha_M (K_3 + \theta_M)))(K_2 K_3 + \alpha_M (K_3 + \theta_M))$$

$$A_2 = \pi^2{}_H \mu_M K_1 K_2 K_3 K_4 (bm\beta_{VM}\alpha_M K_3 + \mu_M (K_2 K_3 + \alpha_M (K_3 + \theta_M)))(K_2 K_3 + \alpha_M (K_3 + \theta_M))) +$$
$$\pi^2{}_H \mu_M K_1 K_2 K_3 K_4 (K_2 K_3 + \alpha_M (K_3 + \theta_M)) + (1-\phi)\pi_H \pi_M b^2 m \beta_M \beta_{VM} \alpha^2{}_M \tau_M \theta_M \varepsilon_M K_3 - (1-\phi)\pi_H \pi_M b^2 m \beta_M \beta_{VM} \alpha_M \tau_M K_1 K_2 K_3{}^2,$$

$$A_3 = \pi^2{}_H \mu^2{}_M K_1{}^2 K_2{}^2 K_3{}^2 K_4{}^2 (1 - \mathrm{R}^2{}_{0M})$$

$R_0^M - 1 > 0$

Which implies that , the endemic equilibrium point of model (6) is stable.

### 3.7 Global stability analysis at endemic equilibrium state

The global stability of the equilibrium point is analyzed using the direct Lyapunov method. The endemic equilibrium point is considered globally stable when $R_0 > 1$. Epidemiologically, this implies that the disease will spread within the population regardless of the initial population size. Based on the fractional model (6),

$$\lambda_M = \frac{\beta_{M1} I_M}{N} + \frac{\beta_{M2} I_M}{N} + \frac{\beta_{M3} I_V}{N}$$

Where $P = \left(S_H^*, E_M^*, I_M^*, \mathrm{R}_M^*, \mathrm{S}_{VM}^*, \mathrm{E}_{VM}^*, \mathrm{I}_{VM}^*\right) \in R_+^7$,

then $\lambda_M = \beta_{M1} I_M + \beta_{M2} I_M + \beta_{M3} I_{VM}$

our fractional model now becomes

$${}^C D_t^{\gamma} S_H = \pi_H - \frac{(1-\phi) bm \beta_m I_{VM}}{N_H} S_H - A_1 S + \varepsilon_M R_M$$

$${}^c D_t^{\gamma} E_H = \lambda_H S_H - \left(\alpha_M + \mu_H\right) \mathrm{E}_M - \mathrm{A}_2 \mathrm{E}_M,$$

$${}^c D_t^{\gamma} I_M = \alpha_M \mathrm{E}_M - \left(\theta_{M1} + \delta_M + \mu_H\right) \mathrm{I}_M - \mathrm{A}_3 \mathrm{A},$$

$${}^c D_t^{\gamma} R_M = \theta_{M1} I_M - \left(\varepsilon_M + \mu_H\right) R_M,$$

$${}^c D_t^{\gamma} S_V = \pi_M - \frac{bm \beta_{vm} I_M S_{VM}}{N_H} - \mu_M S_{VM}$$

$${}^c D_t^{\gamma} E_V = \lambda_V S_{VM} - \left(\tau_M + \mu_M\right) E_{VM}.,$$
$${}^c D_t^{\gamma} I_V = \tau_V E_{VM} - \mu_M I_{VM}. \quad (24)$$

.

At equilibrium point Eq. (24) has the following results

$$\pi = \lambda_{M1}^* S_H^* + \mu S_H^*,\ A_2 E_M^* = \lambda_{M1}^* S^*,\ A_3 I_M^* = \theta_M E_M^*,\ A_4 R_M^* = \varepsilon_{M1} R_M^*,\ A_5 S_{VM}{}^* = \lambda_{VM2} S_V^*,$$
$$A_6 E_{VM}{}^* = \lambda_{VM} S_{VM}^* + \tau_M E^*{}_{VM}. = A_7 I_{VM}{}^* = \tau_M E^*{}_{VM} + \mu_M I^*{}_{VM}.$$

Theorem 1.

Prove that the system Model (24) is globally asymptotically stable at disease free equilibrium, moreover, at Ro<1.

Proof

We construct the lyapunor function to prove the results,

$$L^1 = \pi_H u_1 + \alpha_M S_H E_M (u_2 - u_1) + (1-\alpha_M)(u_3 - u_1) +$$
$$+\theta_H (u_3 - u_2) + \alpha_M (u_4 - u_2) + \varepsilon_M (u_4 - u_3)$$
$$\alpha_M (u_5 - u_4) + \theta_H (u_6 - u_4) + \varepsilon_M (u_7 - u_4)$$

where $u_{1,} u_{2,} u_{3,} u_{4,} u_{5,} u_6$ *and* $u_7$ *are positive constant*

Taking the derivative of the Lyapunov, we have

$R > 1.$

Choosing the positive constants

$$u_{1,} = u_{2,} = u_{3,} = u_{4,} = u_{5,} = u_6 = u_7 = 1$$

*and* $N > \frac{\pi}{\mu}$, then we have

$$L^1 = \pi_H - U_H N_h$$

$$L^1 = \left(U_H N_h - \pi_H\right) > 0,$$

Hence, the system (5) is globally asymptotically

Unstable at the disease free equilibrium and at $R > 1.$ (26)

## 4. 0 Fractional order model numerical results

The fractional-order malaria model was numerically solved using the generalized fractional Adams–Bashforth–Moulton method as described in [24,25]. The parameter values utilized in the model are provided in Table 1, with varying fractional-order values. ($\gamma$) are considered and simulated.

### 4.1. Implementation of fractional Adams–Bashforth–Moulton method

The approach outlined in [26,27] is applied in this study. An approximate solution for the fractional malaria model in (6) is obtained using the fractional Adams–Bashforth–Moulton method. The fractional model (6) is now expressed as follows:

$$^c D_t^{\gamma} P(t) = Q\left(t, q(t)\right),\ 0 < t < \beta,\ \ldots(27)$$

$$P^{(n)}(0) = P_0^{(n)},\ n = 1, 0, \ldots, q, q = [\gamma].$$

Where $P = \left(S_H^*, E_M^*, I_M{}^*, R_M^*, S_{VM}^*, E_{VM}^*, I_{VM}^*\right) \in R_+^7$ and $M\left(t, q(t)\right)$ is a real valued function that is continous.

Eq. (27) can be therefore be represented using the concept of fractional integral as follows;

$$P(t)=\sum_{n=0}^{m-1}P_0^{(n)}\frac{t^n}{n!}+\frac{1}{\Gamma(\gamma)}\int_0^t(t-y)^{\gamma-1}R\big(y,m(y)\big)dy \quad ...(28)$$

Using the method described in [24], we let the step size $g=\frac{\beta}{N}$, $N\in \mathrm{N}$ with a grid that is uniform on $[0,\beta]$. Where $t_c=cr,\ c=0,1,1,...N.$ Therefore, the fractional order model of malaria model presented in (6) can be approximated as :

$$S_{Hk+1}(t)=S_{H0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\left\{\lambda_M-\left(\beta_{M1}I_M^n+\beta_{M2}I_M^n+\beta_{M3}I_{VM}^n\right)\frac{S_H^n}{N_H^n}-\mu S_H^n\right\}+$$
$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\left\{\lambda_M-\left(\beta_{M1}I_M^n+\beta_{H2}I_M^n+\beta_{H3}I_{VM}^n\right)\frac{S_y}{N_{Hy}}-\mu S_y\right\}$$

$$E_{Mk+1}(t)=E_{M0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\left\{\left(\beta_{M1}I_A^n+\beta_{M2}I_S^n+\beta_{M3}I_{VM}^n\right)\frac{S^n}{N^n}-A_2E_M^n\right\}+$$
$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\left\{\left(\beta_{M1}I_M^n+\beta_{M2}I_M^n+\beta_{M3}I_{VM}^n\right)\frac{S_y}{N_{Hy}}-A_2E_{My}\right\}, \quad \dots (29)$$

$$I_{Mk+1}(t)=I_{M0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\left\{\alpha_M\,\mathrm{E}_M^n-\left(\theta_{M1}+\delta_M+\mu\right)I_M^n-\mathrm{A}_3\mathrm{A}^n\right\}+$$
$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\left\{\alpha_M E_{My}-\left(\theta_{M1}+\delta_{My}+\mu\right)\mathrm{I}_{My}-\mathrm{A}_3\mathrm{A}_{MHy}\right\},$$

$$S_{VMk+1}(t)=S_{VM0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\left\{\pi_{H1}-\lambda_{VM}S_{VM}-\mu_M S_{VM}\right\}+$$
$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\left\{\varepsilon_{M1}R_{My}-\left(\lambda_{VM}S_{VM}-\mu_M S_{VM}\right)S_{VMy}\right\},$$

$$E_{k+1}(t)=E_0+\frac{g^\gamma}{\Gamma(\gamma+2)}\left\{\lambda_{V2}S_{VM}^n-\left(\tau_M+\mu\right){E_{VM}}^n\right\}+$$
$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\left\{\lambda_{V2}S_{VMy}-\left(\tau_M+\mu\right)E_{VMy}\right\},$$

$$I_{VMk+1}(t)=I_{VM0}+\frac{g^\gamma}{\Gamma(\gamma+2)}\left\{\tau_M E_{VM}^n-\mu_M I_{VM}\right\}+$$
$$\frac{g^\gamma}{\Gamma(\gamma+2)}\sum_{y=0}^{k}dy,k+1\left\{\tau_M E_{VMy}-\mu_M I_{VM}\right\},$$

Where

$$S_{k+1}^{n}(t)=S_0+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\lambda-\left(\beta_{M1}I_{My}+\beta_{M1}I_{My}+I_{VMy}\right)\frac{S_y}{N_y}-\mu S_y\right\},$$

$$E_{Mk+1}^{n}(t)=E_{M0}+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\left(\beta_{M1}I_{My}+\beta_{M1}I_{My}+I_y\right)\frac{S_y}{N_{Hy}}-\mu E_{My}\right\},$$

$$I_{Mk+1}^{n}(t)=I_{M0}+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\rho E_{My}-A_2I_{My}\right\},\ \ldots(30)$$

$$R_{Mk+1}^{n}(t)=R_{M0}+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\varepsilon_M I_{My}-A_3I_{My}\right\},$$

$$S_{VMk+1}^{n}(t)=S_{M0}+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\pi_M-\lambda_{VM}S_{VM}-\mu_M S_{VM}\right\}.$$

$$E_{VMk+1}^{n}(t)=E_0+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\lambda_{VM2}S_{VM}^{n}-\left(\tau_M+\mu_M\right)E_{VM}^{n}\right\},$$

$$I_{VMk+1}^{n}(t)=I_0+\frac{1}{\Gamma(\gamma)}\sum_{y=0}^{k}f_{y,k+1}\left\{\tau_{VM}E_{VM}^{n}-\mu_M I_{VM}^{n}\right\},$$

From (29) and (30) obtained;

$$dy,_{K+1}=K^{\gamma+1}-(k-\gamma)(k+\gamma)^{\gamma},\ y=0$$

$$(k-y+2)^{\gamma+1}+(k-\gamma)^{\gamma+1}-2(k-y+1)^{\gamma+1},\ 1\le y\le k$$

$$1,\ y=k+1$$

and

$$f_{y,k+1}=\frac{g^{\gamma}}{\gamma}\left[(k-y+1)^{\gamma}(k-y)^{\gamma}\right],\ 0\le y\le k.$$

**Numerical Simulation**

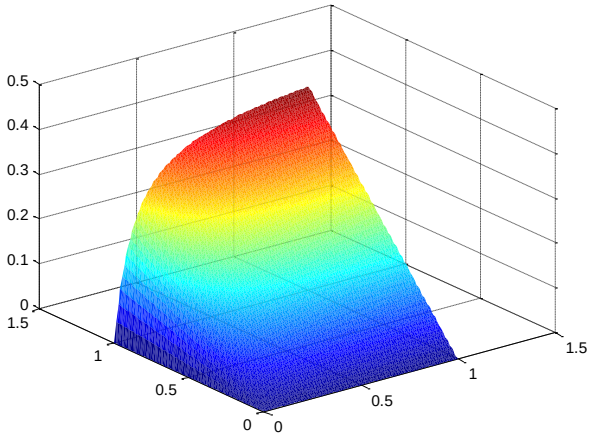


Fig. (1a): Surface plot showing effect of $R_0$ on $\theta_M$ and $\beta_H$

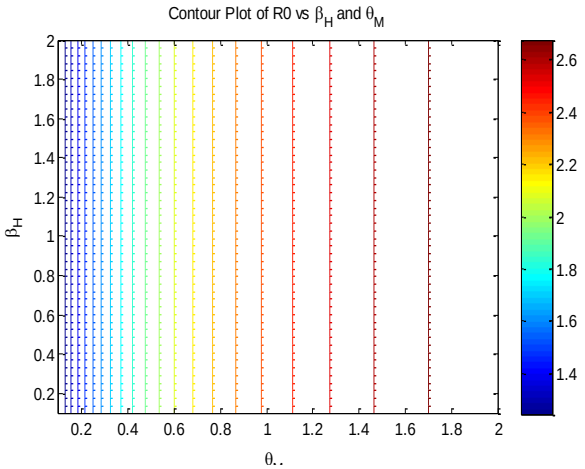


Fig. (1b): Contour plot showing impact of $R_0$ on $\theta_M$ and $\beta_H$

(1a), it can be observed that the basic reproduction number $R_0$ reaches a peak below one (1) as the values of $\beta_H$ reduces and the value of $\theta_M$ increases. This indicates that reducing $\beta_H$ and increasing $\theta_M$ will ultimately alleviate the impact of malaria in the population. Conversely, if appropriate measures are not implemented, $\beta_H$ can exacerbate the prevalence of malaria. This is evident from their effect on $R_0$. (1b) illustrates the contour plot of concerning $R_0$. Upon examination of the numerical streams within the graph, it is evident that the maximum value of $R_0$ attained by varying these parameters is 0.7, indicating a value below unity (1). This observation suggests that augmenting these parameters would not trigger a significant outbreak of malaria in the population.

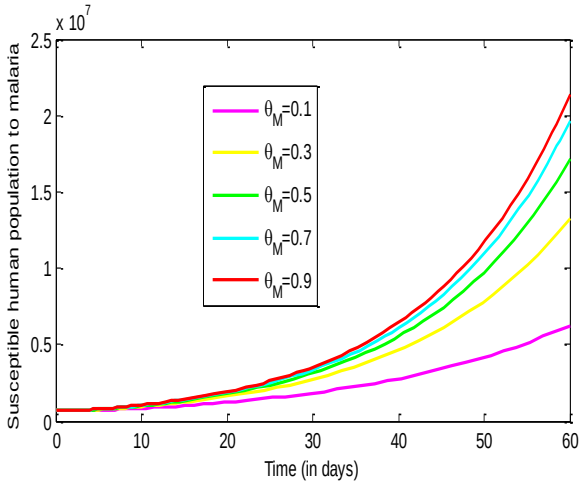


Fig. (2a): Simulation of Susceptible human population to malaria

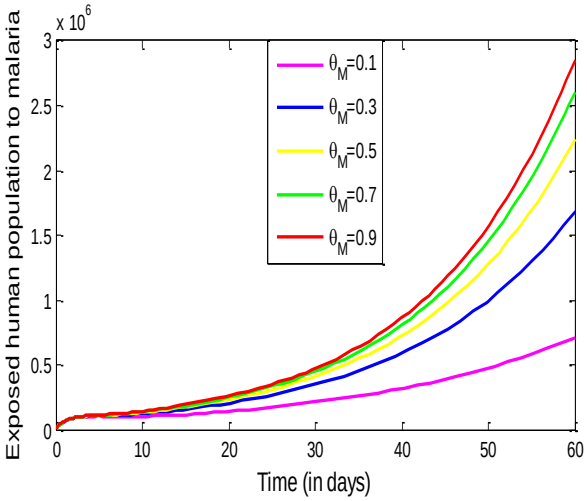


Fig. (2b): Simulation of Exposed human population to malaria

(2a) shows a method to display the effect of recovery rate changes on the system $(\theta_M)$ our research focuses on how malaria spreads in people who are susceptible to malaria. It is observed that, as the recovery rate $(\theta_M)$ when recovery rates increases the number of human susceptible increases. (2b) shows a method to display the effect of recovery rate changes on the system $(\theta_M)$ our research focuses on how malaria spreads in people who are exposed to malaria. It is observed that, as the recovery rate $(\theta_M)$ when recovery rates increases the number of Exposed humans increases.

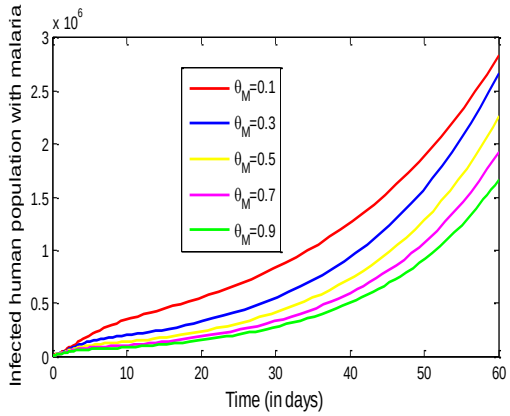


Fig. (2a): Simulation of Susceptible human population to malaria

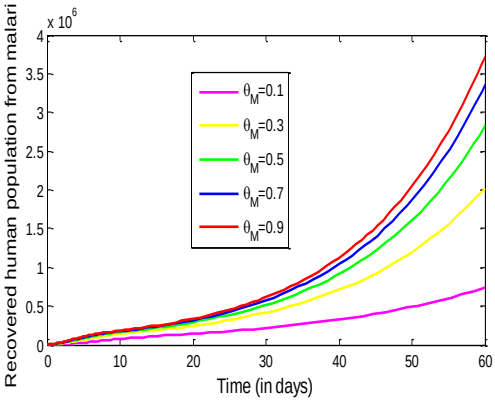


Fig. (2b): Simulation of Exposed human population to malaria

(2c) shows a method to display the effect of recovery rate changes on the system $(\theta_M)$ our research focuses on how malaria spreads in people who are infected with malaria. It is observed that, as the recovery rate $(\theta_M)$ when recovery rates increases the number of human infected decreases. (2d) shows a method to display the effect of recovery rate changes on the system $(\theta_M)$ our research focuses on how malaria spreads in people recovered humans from malaria. It is observed that, as the recovery rate $(\theta_M)$ when recovery rates increases the number of recovered humans from malaria increases.

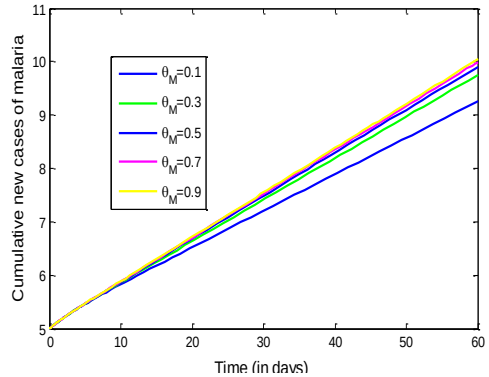


Fig. (2a): Simulation of Susceptible human population to malaria

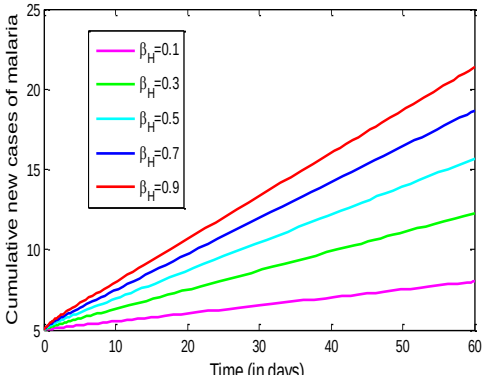


Fig. (2b): Simulation of Exposed human population to malaria

(2e) shows a method to display the effect of recovery rate changes on the system $(\theta_M)$ our research focuses on how the cumulative new cases of malaria spread. It is observed that, when the recovery rates $(\theta_M)$ increases the cumulative new case of malaria decreases. (2f) shows a method to display the effect of contact rate $(\beta_H)$ changes on the system. Our

research focuses on how the cumulative new cases of malaria spread. It is observed that, when the contact rate $(\beta_H)$ increases the cumulative new case of malaria increases.

## 5.0 Conclusions

This study presents a mathematical model to examine the transmission dynamics and control strategies for malaria, incorporating the Caputo fractional derivative. Given the significance of fractional modeling, we conducted a comprehensive theoretical analysis of the fractional malaria model, focusing on the existence and uniqueness of solutions as well as the stability of equilibrium points. For numerical solutions, the fractional Adams–Bashforth–Moulton method was utilized. Simulations demonstrated how model parameters and different fractional orders of the Caputo operator influence disease incidence. Additionally, we investigated the effects of adjusting key parameters, such as the contact rate between infected and susceptible individuals and the recovery rate. The findings indicate that lowering the contact rate while increasing the recovery rate can effectively reduce malaria prevalence in the population. Future research could explore the application of symbolic computing techniques, such as those proposed in [27], to solve nonlinear partial differential equations and obtain analytical solutions.

## Data Availability

All data used in the course of this work are well cited in the work and referenced accordingly.

## Conflict of interest

The authors declared no conflict of interest

## 4.23 Parameter Values and Sources

| Parameter | Value | Source |
|---|---|---|
| $\pi_H$ | 1520 | Estimated |
| $\pi_V$ | 500 | Estimated |
| $\mu_H$ | 0.00004 | [9] |
| $\mu_M$ | 0.05 | [9] |
| $\beta_H$ | 0.18 | [5] |
| $\beta_M$ | 0.8333 | [9] |
| $\delta_H$ | 0.0003454 | [9] |
| $b_M$ | 0.1 | Estimated |
| $\theta_M$ | 0.43 | Estimated |
| $\gamma_M$ | 0.0014 | [9] |
| $\varepsilon_M$ | 0.3 | Estimated |

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