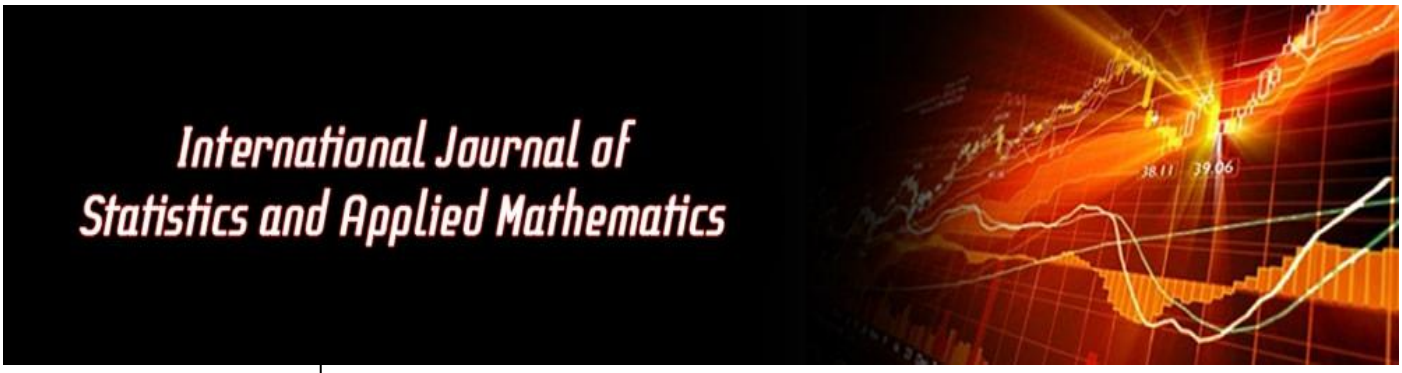

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Mathematical modeling of malaria transmission and treatment: A case of conflict zones

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Abstract
Malaria, which is spread by infected female Anopheles mosquitoes and disproportionately affects vulnerable communities in tropical and subtropical countries, has been one of the world's most urgent public health issues for decades. People with weakened immune systems and visitors to endemic regions are among the high-risk populations that are more vulnerable. Despite ongoing efforts to control the disease, including the development of mathematical models that incorporate key biological and pharmacological factors, significant gap remained in understanding mathematical modeling of malaria transmission and treatment a case in conflict zones. Developing a mathematical model approach to malaria transmission and treatment in conflict areas is the aim of this study. In addition to conducting numerical simulations of the model to confirm the analytical results and ascertain the effects of malaria transmission in war zones, the model addressed stability and sensitivity analysis to ascertain the condition of malaria transmission. This study will mitigate spread and death rates of malaria in conflict zones. Mathematica and MATLAB software was used to perform numerical simulation of model. The study employed ordinary differential equations (ODEs) to solve the progression of malaria treatment over time. The equations will help to track individuals through various compartments, highlighting the dynamics of malaria. The study will utilize numerical techniques recognized for its accuracy and efficiency with non-linear systems that pose challenge to analytical solutions. The study will offer recommendations for malaria treatment strategies a case in conflict zones, providing insights applicable to similar challenging health environments.

Keywords: Malaria, mathematical model, conflict zones, non-conflict zones, fully treatment, partially treatment, stability analysis

Introduction
[10] Research has shown that malaria is a potentially fatal illness brought on by parasites of the genus Plasmodium, which people contract through the bites of female Anopheles mosquitoes carrying the infection [10]. Research revealed that malaria can also be spread by blood transfusion or congenitally from an infected mother to her fetus [10]. Research has shown that although malaria is preventable and curable, it is nevertheless a significant public health concern, particularly in tropical and subtropical areas. Human malaria is caused by five different species of Plasmodium: *Plasmodium falciparum*, which is the most dangerous; *Plasmodium vivax*; *Plasmodium ovale*; Plasmodium *malariae*; and Plasmodium *knowles*i, which is less prevalent but is known to cause malaria in humans.
[11] developed a system of ordinary differential equations to examine the interactions between humans and mosquitoes, and he was the first to utilize mathematical models to investigate the spread and transmission of diseases. By adding important extensions to Ross's work, such as the notion of the basic reproduction number (R_O), the significance of the mosquito's biting rate, and the length of the infectious period, MacDonald (1957) created a more thorough framework for comprehending the dynamics of malaria transmission. The results of the study showed that ($R_o = \frac{m\, a^2\, \rho^n\, \beta_h\, \beta_m}{\mu_m\, \mu_h}$), where R_O>1 signals malaria spread directing control measures.
[9] Created and examined the Susceptible-Exposed-Infectious-Recovered-Susceptible (SEIRS) model to investigate the dynamics of malaria transmission between human and mosquito

populations. While the Disease-Free Equilibrium (DFE) is locally stable when R_O<1 and globally stable when R_O≤1, the disease persists if R_O>1, according to the study, which established the Basic Reproduction Number (R_O) as a threshold parameter. A foundation for researching malaria containment tactics is provided by their findings, which have been validated using numerical simulations.

[6] Highlighted the significance of randomness in disease spread and treatment outcomes, especially in low-transmission areas, by presenting a stochastic model to investigate the dynamics of malaria transmission. Eradication efforts are nevertheless hampered by issues including drug resistance and uneven treatment adherence, even with advancements in control techniques. This model offers important insights into the efficacy of therapies and the possibility of eradicating malaria. This paper's goal is to create a mathematical model of malaria treatment and transmission, specifically in conflict areas.

Model Formulation

The model is adopted from SEIR and SIR model. Where the total population is N and the model is formulated for the human population and mosquito population at time t. The human population is divided into: Susceptible human (S_H), exposed humans (E), infectious humans a case in conflict zones (I_C), infected individuals in other zones (I_F), full treatment of humans in conflict zones (T_P), under dose treatment of humans in conflict zones (T_n) and recovered humans (R), and that of mosquitoes into two divisions: Susceptible mosquitoes (S_V) and infectious mosquitoes (I_V), ($SEI_CI_FT_PT_nR - SI$). These are main features of the model; π is recruitment of susceptible humans, μ is death rates of susceptible humans, α is rate of infection of humans, λ is infectious rate to full treatment humans, θ is natural deaths rates of full treatment humans, ε is death rates infected humans in other zones, ω is treatment rate of infected of full treatment individuals, x is recovery rate of full treated individuals, e is death rate of full treatment humans, γ is recovery rate of other zones individuals, τ is death rates of infectious mosquito, Ψ is rate of infection of mosquito, ν is rate of infection of mosquito from other zones patients, φ is rate of infection of mosquitoes from infected humans in conflict zones, Λ is recruitment of susceptible mosquitoes, Z is rates of infected mosquito bites to susceptible humans and ρ is rate of recovered exposed humans.

Model Assumptions

1. The model makes the assumption that the population's overall size stays constant throughout time.
2. It is assumed that humans progress through stages of disease in a linear manner.
3. It is assumed that the primary source of infection for humans is through mosquito bites, and that human-to-human transmission is considered a secondary route.
4. For both treated and exposed individuals, recovery rates are presumed to be constant and unaffected by treatment efficacy or individual health.
5. The population is homogeneous
6. All people have equal chances of getting malaria

Table 1: Model Variables

Variables	Description
E	Exposed humans
I_C(t)	Infectious individuals in conflict zones
I_f(t)	Infectious individuals in non-conflict zones
I_V(t)	Infectious mosquitoes
R(t)	Humans who have recovered from malaria
S_H(t)	Humans that are susceptible to malaria disease
S_V(t)	Mosquitoes that consume contaminated blood are susceptible to infection.
T_n(t)	Treatment of humans being compromised
T_P(t)	Full treatment of humans

Table 2: Model parameters

Λ	Recruitment of susceptible mosquitoes
E	Death rate of full treatment humans
N	Total number of population
τ	Rate of infectious mosquito deaths
V	Mosquito infection rate in non-conflict areas
X	Recovery rate of full treatment humans
Z	Infected mosquito biting rates to susceptible humans
α	Rate of infection of humans
γ	Recovery rate of humans in non-conflict zones
ϵ	Deaths rates of compromised humans
θ	Death rate of infected human in conflict zones
λ	Human infectious rate in areas of conflict
μ_m	Rates of mosquito deaths that occur naturally
μ_h	Human natural death rates
Π	Recruitment of susceptible humans through birth rate
ρ	Recovery rate of humans to become exposed to the disease
ψ	Infection rate of mosquito
ω	Treatment rate for infected humans undergoing full treatment
Φ	Infection rate of mosquitoes from non-conflict zones

Model Equations

$$\frac{dS_H}{dt} = \pi + zI_V - \alpha S_H - \mu S_H. \quad (1)$$

$$\frac{dE}{dt} = \alpha S_H + \rho R - \lambda E - (1-\lambda)E - \mu E \quad (2)$$

$$\frac{dI_C}{dt} = \lambda E - \phi I_C - \Theta I_C - \text{ɯ} I_C - (1-\text{ɯ}) I_C - \mu I_C \quad (3)$$

$$\frac{dI_F}{dt} = (1-\lambda)E - vI_F - \epsilon I_F - \mu I_F - \gamma I_F \quad (4)$$

$$\frac{dT_P}{dt} = \text{ɯ} I_C - \epsilon T_P - \mu T_P - XT_P \quad (5)$$

$$\frac{dT_n}{dt} = (1-\text{ɯ}) I_C - eT_n - \mu T_n - (1-x)T_n \quad (6)$$

$$\frac{dR}{dt} = xT_P + \gamma I_F - \rho R - \mu R + (1-x)T_n \quad (7)$$

$$\frac{dS_V}{dt} = \wedge + \phi I_C + vI_F - \psi S_V - \mu S_V \quad (8)$$

$$\frac{dI_V}{dt} = \psi S_V - zI_V - \tau I_V \quad (9)$$

Positivity of solutions

By demonstrating the following theorem, we establish positivity.

Theorem 1: If $S_H(0)$, E(0), $I_C(0)$, $I_f(0)$, $T_c(0)$, $T_f(0)$, $S_V(0)$, $I_V(0)$, R(0), have a positive outlook, and the solutions will be (s_H(t), E(t), I_C(t), I_F(t), T_C(t), T_f(t), S_V(t), I_V(t), R(t),) are positive ∀ t>0 in equations (1- 9).

Proof: Let t*= sup{t>0: S_H(t)>0, E(t)>0, I_C(t)>0, I_f(t)>0, T_C(t)>0, T_f(t)>0, S_V(t)>0, I_V(t)>0, R(t)>0}, such that t*>0. From equation (1) - (9) and considering equation (1)

$\frac{d\,S_H}{dt} = \pi + z\,I_V - \sigma\,S_H - \mu\,S_H \geq \pi + z\,I_V - (\sigma + \mu)S_H$. Applying integrating factor to equation (1);

$I(t) = e^{\int_0^{t*} \mu ds} = e^{\mu t}$ Multiplying through by It

$$e^{\mu t}\frac{dS_H}{dt} + \mu\, e^{\mu t} S_H = e^{\mu t}(\wedge - \beta\, S_H\, I_V \quad (10)$$

The Left-hand side derivative of a product. $\frac{d}{dt}(e^{\mu t} S_H) = e^{\mu t}(\wedge - \beta S_H\, I_V)$ From t=0 and t=t*, integrating both sides

$$\int_0^{t*} \frac{d}{ds}(e^{\mu t} S_H(s)ds = \int_0^{t*} e^{\mu s}\ (\wedge - \beta S_H I_V)ds \quad (11)$$

Evaluating the left-hand side $e^{\mu t} S_H(t) - S_H(0)$. *For* Right-hand side $\int_0^{t*} e^{\mu s}(\wedge - \beta S_H I_V)ds$

Therefore, $e^{\mu t} S_H(t) = S_H(0) + \int_0^{t*} e^{\mu s}(\wedge - \beta S_H I_V)ds$ (12)

We solve for $S_H(t)$; $S_H(t) = \frac{S_H(0) + \int_0^{t*} e^{\mu s}(\wedge - \beta S_H I_V}{e^{\mu t}} ds$ since$S_H(0) > 0$; from initial condition

Also $\wedge - \beta S_H I_V$ is bounded and non-negative $e^{\mu t} > 0$, ∀ t > 0. As a result, S_H (t)>0, ∀ t>0. If t>0, the solution stays positive. Thus S_H(t) remains positive. Similarly, repeating above procedure for other equation (2) - (9) shows that solutions are also positive.
We deduce that the following are positive ∀ t>0: $S_H(t), E(t), I_C(t), I_f(t), T_c(t), T_f(t), S_V(t), I_V(t)\ and\ R(t)$. These findings prove the theorem.

Boundedness of solutions

Theorem 2

Assume that the total populations of humans and mosquitoes are $N_H(t)$ and $N_M(t)$, respectively, governed by the system of equation (1) - (9) given above. If the recruitment rates π and $\wedge$ are positive and the natural death rates μ_H and $\mu_M$are strictly positive, then the total populations $N_H(t)$ and $N_M(t)$ remain bounded ∀ t≥ 0.

Proof

We prove the boundedness of solutions by taking into consideration of total human and mosquito population separately.

Step 1: The total human population (N_H)
We define total human population as:

$$N_H = S_H + E + I_C + I_f + T_P + T_n + R \tag{13}$$

$$\frac{d\,N_H}{dt} = \pi - \mu N_H$$

$$\int \frac{dN_H}{\pi - \mu N_H} = \int dt$$

$$N_H(t) = \frac{\pi}{\mu} + (N_H(0) - \frac{\pi}{\mu})e^{-\mu t} \tag{14}$$

$$N_H(t) \leq \frac{\pi}{\mu}, \forall\ t \geq 0. \tag{15}$$

Step 2: Total mosquito population (N_M)W, where total mosquito population defined as:

$$N_M = S_V + I_V \tag{16}$$

$$\frac{dN_M}{dt} = \wedge - \mu_M N_M\ ; N_M(t) = \frac{\wedge}{\mu_M} + \left(N_M(0) - \frac{\wedge}{\mu_M}\right) e^{-\mu_M t}$$

$$N_M(t) \leq \frac{\wedge}{\mu_M}, \forall\ t \geq 0. \tag{17}$$

Therefore, we conclude that $N_H(t)$ and $N_M(t)$ remain bounded $\forall\ t \geq 0$. Hence prove.

Computation of basic reproduction number
We calculate the basic reproduction number R_O using the next-generation matrix approach [12]. If $R_O > 1$, the disease is expected to spread within the population, leading to an epidemic and a stable endemic equilibrium point (EEP) exists. Conversely, if $R_O < 1$, the infection will eventually dies out, and the system will approach a disease-free equilibrium (DFE) that is stable. In this method, F represent matrix of the new infections while V represent the remaining transition terms, such as recovery or progression between compartments, excluding new infections. Now, let

$\frac{dI_C}{dt} = \lambda E - \Omega_1 I_C$, where $\Omega_1 = -(\Phi + \Theta + 1 + \mu)$

$\frac{dI_f}{dt} = (1-\lambda) - \Omega_2 I_f$, where $\Omega_2 = -(v + e + \mu + \gamma)$

$\frac{dT_P}{dt} = \omega I_C - \Omega_3 T_P$, where $\Omega_3 = -(\epsilon + \mu + x)$ (18)

$\frac{dT_n}{dt} = (1-\omega) I_C - \Omega_4 T_n$, where $\Omega_4 = -(e + \mu + 1 - x)$

$\frac{dI_V}{dt} = \psi S_v - \Omega_5 I_V$, where $\Omega_5 = -(z + \tau)$

Where the vector of new infection terms going into compartment is;

$$f = \begin{bmatrix} \lambda E \\ (1-\lambda)E \\ 0 \\ 0 \\ 0 \end{bmatrix} \tag{19}$$

The vector of transition is,

$$V = \begin{bmatrix} +\Omega_1 I_C \\ +\Omega_2 I_f \\ -\omega I_C + \Omega_3 T_P \\ (\omega - 1) I_C + \Omega_4 T_n \\ -\psi S_V + \Omega_5 I_V \end{bmatrix} \tag{20}$$

By computing the Jacobian matrices of the transition terms and the new infection terms, respectively, evaluated at the D.F.E., we are able to obtain the matrices F and V.

$$F = \begin{bmatrix} \beta E & \beta\eta_1 E & \beta\eta_2 E & \beta\eta_3 E & \beta\eta_4 E \\ (1-\beta)E & (1-\beta)E\eta_1 & (1-\beta)E\eta_2 & (1-\beta)\eta_3 E & (1-\beta)\eta_4 E \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad (21)$$

Where $I_C, I_V, I_f, T_n, and\ T_P$ are infected compartment

$$V = \begin{bmatrix} \Omega_1 & 0 & 0 & 0 & 0 \\ 0 & 0 & \Omega_2 & 0 & 0 \\ -\omega & 0 & 0 & 0 & \Omega_3 \\ (\omega - 1) & 0 & 0 & \Omega_4 & 0 \\ 0 & \Omega_5 & 0 & 0 & 0 \end{bmatrix}.$$

We calculate FV^{-1}, which is the next generation matrix; $FV^{-1} =$

$$\begin{bmatrix} \frac{\beta(-((-1+\omega)\eta_3\Omega_3+(\omega\eta_4+\Omega_3)\Omega_4)}{\Omega_1\Omega_3\Omega_4} & \frac{\beta\eta_2}{\Omega_2} & \frac{\beta\eta_4}{\Omega_3} & \frac{\beta\eta_3}{\Omega_4} & \frac{\beta\eta_1}{\Omega_5} \\ \frac{(-1+\beta)((-1+\omega)\eta_3\Omega_3-(\omega\eta_4+\Omega_3)\Omega_4}{\Omega_1\Omega_3\Omega_4} & -\frac{(-1+\beta)\eta_2}{\Omega_2} & -\frac{(-1+\omega)\eta_4}{\Omega_3} & -\frac{(-1+\beta)\eta_3}{\Omega_4} & -\frac{(-1+\beta)\eta_1}{\Omega_5} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \quad (23)$$

Then, finding the Eigen value; $X_1 = 0, X_2 = 0, X_3 = 0$, and

$$X_4 = -\frac{-\beta\eta_3\Omega_2\Omega_3+\beta\omega\eta_3\Omega_2\Omega_3-\beta\omega\eta_4\Omega_2\Omega_4-\eta_2\Omega_1\Omega_3\Omega_4+\beta\eta_2\Omega_1\Omega_3\Omega_4-\beta\Omega_2\Omega_3\Omega_4}{\Omega_1\Omega_2\Omega_3\Omega_4} \quad (24)$$

Therefore,

$$\beta = \frac{\Omega_1(\eta_2-\Omega_2)\Omega_3\Omega_4}{-\eta_3\Omega_2\Omega_3+\omega\eta_3\Omega_2\Omega_3-\omega\eta_4\Omega_2\Omega_4+\eta_4\Omega_1\Omega_3\Omega_3-\Omega_2\Omega_3\Omega_4} \quad (25)$$

Where is R_O, the dominant Eigen value and spectral radius. A value of R_O<1 often indicates that each individual can infect an average of fewer than one person, which will cause the disease to die out and result in a locally and globally asymptotically stable disease free equilibrium. The disease is predicted to persist in the population if R_O>1, which indicates that each individual may typically infect more than one person.

Disease-free equilibrium point

We assume there are no infected humans or mosquitoes. Therefore $I_C = I_F = T_P = T_n = I_V = 0$, hence (E)=0

Solving for S_H ,R and S_V .The equation (1) ; $\frac{dS_H}{dt} = \pi - \alpha S_H - \mu S_H = 0$ (26)

Solving for S_H^* (DFE value of S_H); $S_H^* = \frac{\pi}{\alpha+\mu}$ (27)

From equation (7), where $T_P = T_n = 0$; $\frac{dR}{dt} = -\rho R - \mu R = 0$

Thus, $R^* = 0$. From equation (8) which simplifies to $\frac{dS_V}{dt} = \wedge -\psi S_V - \mu S_V = 0$ (28)

Solving for S_V^*; $S_V^* = \frac{\wedge}{\psi+\mu}$ .Since $I_V = 0$, the infectious force λ=0.

DFE is therefore

$$(E^0 = S_H^0, E^0, I_C^0, I_F^0, T_P^0, T_n^0, R^0, S_V^0, I_V^0) = (\frac{\pi}{\alpha+\mu}, 0, 0, 0, 0, 0, 0, \frac{\wedge}{\psi+\mu}, 0) \quad (29)$$

In figure 1 below, the infection-free equilibrium point of system 1-9, or D.F.E point (E^0), is then quantitatively displayed.

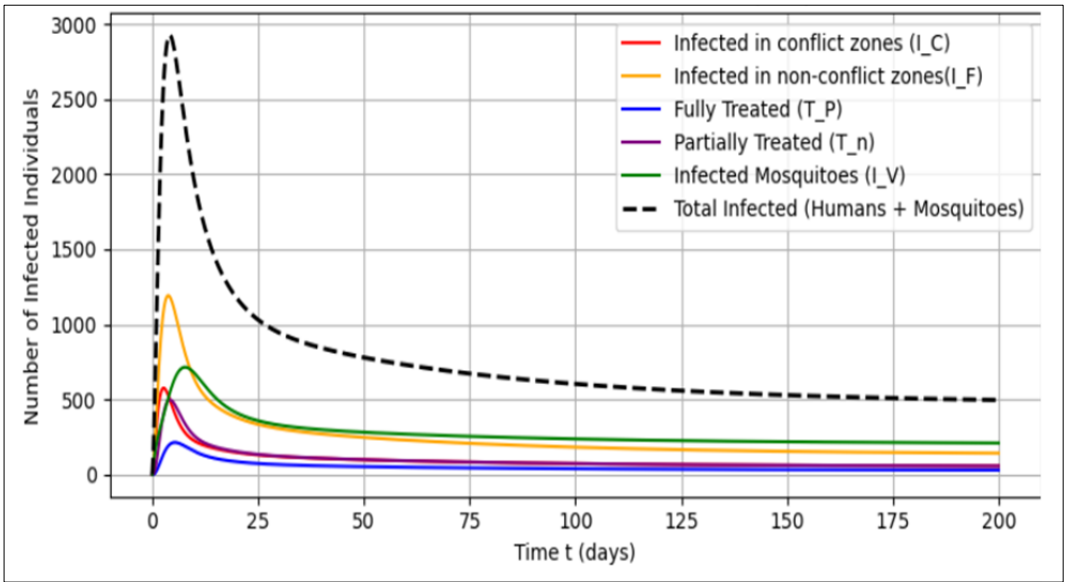


Fig 1: Malaria infection burden over tune t (DFE)

Local Stability Analysis of DFE.
Local stability is found by constructing the Jacobian Matrix J based on nine malaria compartmental model. We will simply extract and examine the portion of the Jacobian that controls the diseased compartments for the sake of conciseness, which are:

$X = E, I_C, I_F, T_P, T_n, R, I_V$

At E_0 the Jacobian J is evaluated. We then determine the eigenvalues of this reduced Jacobian and then, the system is linearized around DFE. [12] We use the Next-generation matrix approach.

Theorem 3
The malaria disease-free equilibrium E_0 is locally asymptotically stable if$R_O < 1$ and unstable if $R_O > 1$. Then, we calculate Jacobian matrix J for equation (1)-(9), such that

$$g_1 = \pi + zI_V - \alpha S_H - \mu S_H$$

$$g_2 = \alpha S_H + \rho R - \lambda E - (1-\lambda)E - \mu E$$

$$g_3 = \lambda E - \phi I_C - \Theta I_C - \omega I_C - (1-\omega) I_C - \mu I_C$$

$$g_4 = (1-\lambda)E - vI_F - \epsilon I_F - \mu I_F - \gamma I_F$$

$$g_5 = \omega I_C - \epsilon T_P - \mu T_P - XT_P$$

$$g_6 = (1 - \omega I_C - eT_n - \mu T_n - (1-x)T_n \quad (30)$$

$$g_7 = xT_P + \gamma I_F - \rho R - \mu R + (1-x)T_n$$

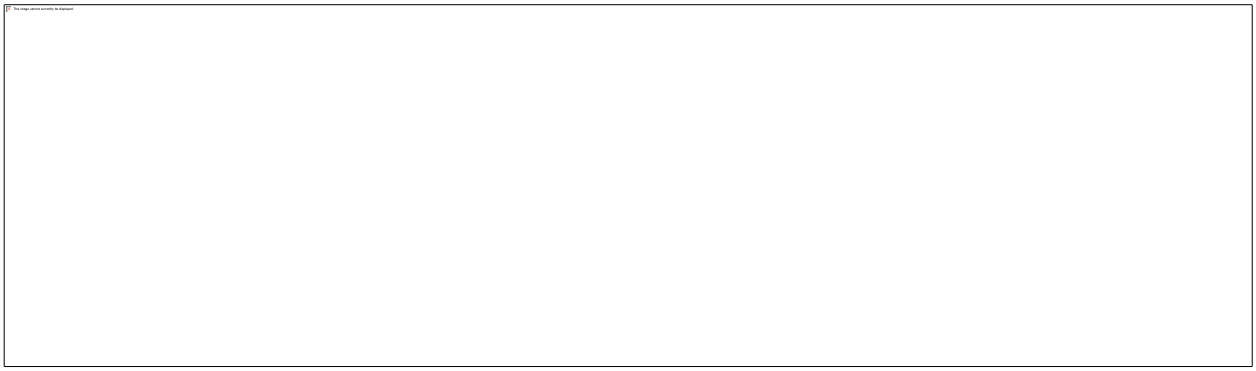
$$g_8 = \wedge + \phi I_C + vI_F - \psi S_V - \mu S_V$$

$$g_9 = \psi S_V - zI_V - \tau I_V$$

And the force of infection given by; $\lambda = \frac{\beta(I_C + \eta_1 I_V + \eta_2 I_F + \eta_3 T_n + \eta_4 T_p}{N}$

The Jacobian matrix J of malaria model is obtained and evaluated at DFE. The eigenvalues are analyzed to determine the condition for local stability of DFE.

Proof
From Jacobian matrix of model



(31)

Calculating the determinant of /J−λ_iI/=0 yields the characteristic polynomial of a Jacobian matrix, where J is a Jacobian matrix with i=1,2,3,4,5,6,7,8,9. and A Jacobian matrix's characteristic polynomial and λ is a scalar variable. According to [5], the eigenvalues that establish system stability close to an equilibrium point are the roots of this polynomial.
Then, we apply the Routh-Hurwitz criterion to determine whether those coefficients lead to all negative real parts of eigenvalues.

$$X(\lambda) = \lambda^9 + \lambda^8 a_1 + \lambda^7 a_2 + \lambda^6 a_3 + \lambda^5 a_4 + \lambda^4 a_5 + \lambda^3 a_6 + \lambda^2 a_7 + \lambda^1 a_8 + a_9 \quad (32)$$

The conditions for all coefficients are $a_1 > 0, a_2 > 0, a_3 > 0 \ldots\ldots, a_9 > 0$ The values of coefficients $a_1 > 0, a_2 > 0, a_3 > 0 \ldots\ldots, a_9 > 0$ expressed in term of R_0^* are;

$a_1 = 0$, $a_2 =$ -γ-ϵ-μ-ν-β η_2

$$a_3 = -\beta^2\eta_2 - \beta\omega\eta_2 - (-1-\mu)(-\gamma-\epsilon-\mu-\nu-\beta\eta_2) + (\alpha+Z+\mu+\tau)(-\gamma-\epsilon-\mu-\nu-\beta\eta_2) - (-1+\beta-\theta-\mu-\phi)(-\gamma-\epsilon-\mu-\nu-\beta\eta_2) - (-1-\mu-\varrho+X)(-\gamma-\epsilon-\mu-\nu-\beta\eta_2) + (-\mu-\rho)(\gamma+\epsilon+\mu+\nu+\beta\eta_2) + (-\mu-\varphi)(\gamma+\epsilon+\mu+\nu+\beta\eta_2) + (-\epsilon-\mu-\chi)(\gamma+\epsilon+\mu+\nu+\beta\eta_2) \quad (33)$$

The appendix will include detailed coefficientsa_4, a_5, a_6, a_7, a_8, and a_9 that left out of the local stability analysis for simplicity.

Global stability of disease-free equilibrium (D.F.E)
It is also possible to demonstrate that the system of equations (1-9) lies in the positive region. The Marzler matrix stability approach, which was proposed by [1], is used to examine the global stability of disease-free equilibrium.

$$\frac{d\,X}{dt} = F(X,Z)$$

$$\frac{dZ}{dt} = G(X,Z), G(X,0)$$

$Z = (I_C, T_P, T_n, I_F, I_V) \in R_+^5$ Indicates the infectious malaria compartment, while $X = (S_H, S_V, E, R) \in R_+^4$indicates the non-infected malaria compartments. If this point meets the following criteria, $E_0 = (X^*, 0)$ denotes the system's disease-free equilibrium:

i) $\frac{dX}{dt} = F(X,0)$, in which X^* is asymptotically stable globally.
ii) $\frac{dZ}{dt} = D_z G(X,0)Z - G(X,Z) \geq 0$ If the following theorem is true, we can argue that E_0 is locally asymptotically stable for any (X,Z)∈Ω:

Theorem 4
The equilibrium point $E_0(X^*, 0)$ of the system is globally asymptotically stable if $R_0^* \leq 1$ and the conditions (i) and (ii) are satisfied, otherwise unstable.

Proof:
In the system model, let $X = (S_H, S_V, E, R)$ and $Z = (I_C, T_P, T_n, I_F, I_V)$ be the new variables and sub-systems. After obtaining the vector function G(X,Z), we examine reduced systems to

$$F(X,0) = \begin{pmatrix} \pi - \mu S_H \\ \wedge - \mu S_V \\ 0 \\ 0 \end{pmatrix} \quad (34)$$

The convergence of the solutions of the reduced system (34) is global in Ω since it is observed that this is an asymptotic dynamics system independent of the initial conditions in Ω. This may be calculated by computing:

$G^{\wedge}(X,Z) = D_z G(X^*,0)z - G(X,Z)$

$G^{\wedge}(X,Z) \geq 0$. Now let $A = D_z G(X^*,0)$, which is the Jacobian of $G^{\wedge}(X,Z)$ taken in $(I_C, T_P, T_n, I_F, I_V)$ and evaluated at $(x^*,0)$, such that the matrix A is given by;

$$A = \begin{bmatrix} \Omega_1 & \beta\eta_2 & \beta\eta_4 & \beta\eta_3 & \beta\eta_1 \\ -\beta & -\beta\eta_2 - \Omega_2 & -\beta\eta_4 & -\beta\eta_3 & -\beta\eta_1 \\ \omega & 0 & \Omega_3 & 0 & 0 \\ 1-\omega & 0 & 0 & \Omega_4 & 0 \\ \Phi & 0 & \vee & 0 & 0 \end{bmatrix} \quad (34)$$

Where $\beta - \Phi - \Theta - 1 - \mu = \Omega_1$, $-\vee -\epsilon - \mu - \gamma = \Omega_2$, $-\epsilon - \mu - x = \Omega_3$ and $-e - \mu - 1 + x = \Omega_4$
The matrix provides the values for G^∧ (X, Z).

$$AZ = \begin{bmatrix} (1-\frac{S}{N})\beta(I_C + \eta_1 I_V + \eta_2 I_F + \eta_3 T_n + \eta_4 T_p) \\ 0 \\ 0 \\ 0 \\ 0 \end{bmatrix} \quad (35)$$

Therefore, if $G(X,Z) \geq 0$, then the disease-free equilibrium $(E_0)$is globally asymptotically stable; otherwise, it is unstable. Since $G(X,Z)$
$S_H \leq N, \frac{S_H}{N} \leq 1$, Then $G(X,Z) \geq 0 \; \forall \; X, Z \in R_+^5$, therefore, the disease-free equilibrium will be asymptotically stable globally. The non-negative off-diagonal elements of matrix A make it an M-Matrix. The global disease-free equilibrium (G.D.F.E.) is therefore shown to be globally asymptotically stable. This is the proof. Thus, regardless of the original conditions, malaria dies off whenever$R_O^* < 1$.

2.9 Existence of Endemic Equilibrium Point (E.E.P)
Theorem 5: Endemic Equilibrium Point exists whenever $R_O > 1$
Proof: All infectious classes must be bigger than zero in order for E.E.P. to exist.

, $I_C > 0, I_F > 0, T_P > 0, T_n > 0$ and $I_V > 0$.

Where,

$I_C = \lambda E - (\Phi + \Theta + \omega - (1-\omega) + \mu)I_C$. This implies that

$A = \Phi + \Theta + 1 + \mu$, Therefore

$$I_C = \frac{\lambda E}{A} \quad (36)$$

Also $I_F = (1-\lambda)E - (v + e + \mu + \gamma)I_F$, Let $B = v + e + \mu + \gamma$, Therefore

$$I_F = \frac{(1-\lambda)E}{B} \quad (37)$$

Also $T_P = \omega I_C - (\epsilon + \mu + X)T_P$, Let $C = \epsilon + \mu + X$, Hence$T_P = \frac{\omega I_C}{C}$. (38) $T_n = (1-\omega)I_C - (\epsilon + \mu + (1-X))T_n$. Let$D = \epsilon + \mu + (1-X)$.

$$,T_n = \frac{(1-\omega)I_C}{D}, \quad (39)$$

$$I_V = \psi S_V - (z + \tau)I_V, \text{ let} = z + \tau \; ; I_V = \frac{\psi S_V}{G} \quad (40)$$

Substituting equation (36-40) into the force of infection

$$\lambda = \frac{\beta}{N\beta}\left[\frac{\lambda E}{A} + \eta_1 \frac{\psi S_V}{G} + \eta_2 \frac{(1-\lambda)E}{B} + \eta_3 \frac{(1-\omega).\lambda\frac{\lambda E}{A}}{D} + \eta_4 \frac{\frac{\lambda E}{A}}{C}\right]. \quad (41)$$

Group terms

$$\lambda = \frac{\beta}{N}\left[\frac{\lambda E}{A} + \eta_1 \frac{\psi S_V}{G} + \eta_2 \frac{(1-\lambda)E}{B} + \frac{\lambda E}{A}\left(\frac{\eta_3(1-\omega)}{D} + \frac{\eta_4 \omega}{C}\right)\right], \quad (42)$$

Group λEterms

$$\lambda = \frac{\beta}{N}\left[\eta_1 \frac{\psi S_V}{G} + \frac{(1-\lambda)E\eta_2}{B} + \frac{\lambda E}{A}\left(1 + \frac{\eta_3(1-\omega)}{D} + \frac{\eta_4 \omega}{C}\right)\right]. \tag{43}$$

Let $= \frac{1}{A}(1 + \frac{\eta_3(1-\omega)}{D} + \frac{\eta_4 \omega}{C}$; $F = \eta_1 \frac{\psi S_V}{G}$ and $Q = \frac{\eta_2 E}{B}$

Solving for λ becomes

$$\lambda = \frac{\beta F+Q)}{N+\beta Q-\beta EM} > 1 \tag{44}$$

Hence, E.E.P proved.
The endemic equilibrium point is thus shown numerically in the figure below.

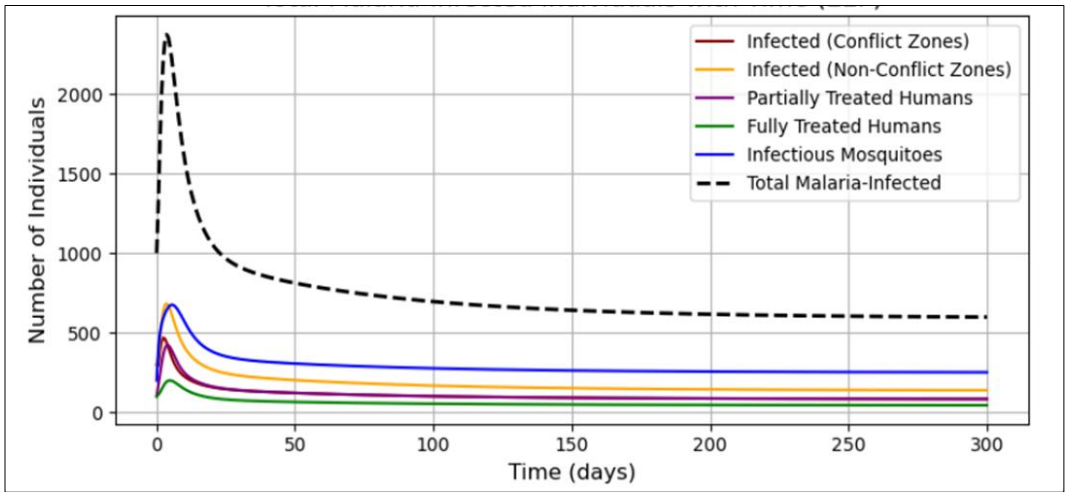


Fig 2: Total Malaria-infected individuals with time (EEP)

Global stability analysis of the Endemic Equilibrium point

To determine the prerequisites for the stability of the endemic equilibrium point, a Lyapunov criterion was used. This involved identifying the necessary conditions for the derivative of the derivative of the Lyapunov fuction to be negative definite, which confirms global asymptotic stability for the reduced equations of the system (1-9);then simplifying the system by introducing Ω_i;

$$\frac{dS_H}{dt} = \pi + zI_V - \alpha S_H - \mu S_H = \pi + zI_V - \Omega_1 S_H$$

$$\frac{dE}{dt} = \alpha S_H + \rho R - \lambda E - (1-\lambda)E - \mu E = \alpha S_H + \rho R - \Omega_2 E$$

$$\frac{dI_C}{dt} = \lambda E - \phi I_C - \Theta I_C - \omega I_C - (1-\omega) I_C - \mu I_C = \lambda E - \Omega_3 I_C$$

$$\frac{dI_F}{dt} = (1-\lambda)E - vI_F - \epsilon I_F - \mu I_F - \gamma I_F = (1-\lambda)E - \Omega_4 I_F \tag{45}$$

$$\frac{dT_P}{dt} = \omega I_C - \epsilon T_P - \mu T_P - X T_P = \omega I_C - \Omega_5 T_P$$

$$\frac{dT_n}{dt} = (1-\omega) I_C - eT_n - \mu T_n - (1-x)T_n = (1-\omega) I_C - \Omega_6 T_n$$

$$\frac{dR}{dt} = xT_P + \gamma I_F - \rho R - \mu R + (1-x)T_n = xT_P + (1-x)T_n + \gamma I_F - \Omega_7 R$$

$$\frac{dS_V}{dt} = \wedge + \phi I_C + vI_F - \psi S_V - \mu S_V = \wedge + \phi I_C + vI_F - \Omega_8 S_V$$

$$\frac{dI_V}{dt} = \psi S_V - zI_V - \tau I_V = \psi S_V - \Omega_9 I_V$$

The control reproduction number (R_O^*), the force of infection (λ^*), D.F.E
$E^0 = (S_H^0, E^0, I_C^0, I_F^0, T_P^0, T_n^0, R^0, S_V^0, I_V^0) = \left(\frac{\pi}{\mu}, 0,0,0,0,0,0,0,0,0\right)$ and E.E.P $E^* = (S^*, E^*, I_C^*, I_F^* T_P^*, T_n^*, R^*, S_V^*, I_V^*$of the system is given by;

$$R_O^* = \frac{\beta}{N}\left(\frac{S^0}{\Omega_1} + \frac{S^0\eta_1}{\Omega_1\Omega_6} + \frac{S^0\eta_2}{\Omega_1\Omega_2} + \frac{S^0\eta_3}{\Omega_1\Omega_4} + \frac{S^0\eta_4}{\Omega_1\Omega_3}\right. \quad (46)$$

The system of equation in (4.1.8.1) we propose Lyaponuv function

$$K(S_H, E, I_C, I_F, T_P, T_n, R, S_V, I_V) = S_H - S_H^* - S_H^* ln\frac{S_H}{S_H^*} + y_1\left(E - E^* - E^* ln\frac{E}{E^*}\right) + y_2\left(I_C - I_C^* - I_C^* ln\frac{I_C}{I_C^*}\right) + y_3\left(I_F - I_F^* - I_F^* ln\frac{I_F}{I_F^*}\right) + y_4\left(T_P - T_P^* - T_P^* ln\frac{T_P}{T_P^*}\right) + y_5\left(T_n - T_n^* - T_n^* ln\frac{T_n}{T_n^*}\right) + y_6\left(R - R^* - R^* ln\frac{R}{R^*}\right) + y_7\left(S_V - S_V^* - S_V^* ln\frac{S_V}{S_V^*}\right) + y_8\left(I_V - I_V^* - I_V^* ln\frac{I_V}{I_V^*}\right) \quad (47)$$

$y_1, y_2, \ldots, y_8$ are all positive to be determined.

The Lyapunov function $K(S_H, E, I_C, I_F, T_P, T_n, R, S_V, I_V)$ satisfies the condition $K(S^*, E^*, I_C^*, I_F^* T_P^*, T_n^*, R^*, S_V^*, I_V^*) = 0$ and $K(S_H, E, I_C, I_F, T_P, T_n, R, S_V, I_V) > 0$, hence it's definite for;

$\frac{dk(S_H,E,I_C,I_F,T_P,T_n,R,S_V,I_V)}{dt}$ (48) For it to be negative definite, it must satisfy,

$\frac{dk(S^*,E^*,I_C^*,I_F^*T_P^*,T_n^*,R^*,S_V^*,I_V^*}{dt} = 0$ And $\frac{dk(S^*,E^*,I_C^*,I_F^*T_P^*,T_n^*,R^*,S_V^*,I_V^*}{dt} < 0$

Therefore, the E.E.P $E^* = (S^*, E^*, I_C^*, I_F^* T_P^*, T_n^*, R^*, S_V^*, I_V^*$ for the system satisfies,

$\pi = \alpha S_H^{**} + \mu S_H^{**} - \mathrm{z} I_V^{**}$, $\Omega_2^{**} E^{**} = \alpha^{**} S_H^{**} + \rho^{**} R^{**}$, $\Omega_3^{**} I_C^{**} = \lambda^{**} E^{**}$, $(1-\lambda)^{**} E^{**} = \Omega_4 I_F^{**}$,
$\mathrm{ɯ} I_C^{**} = \Omega_5 T_P^{**}$, $(1-\mathrm{ɯ}) I_C^{**} = \Omega_6 T_n^{**}$, $x T_P^{**} + (1-x) T_n^{**} + \gamma I_F^{**} = \Omega_7 R^{**}$,
$\wedge + \phi I_C^{**} + v I_F^{**} = \Omega_8 S_V^{**}$ and $\psi S_V^{**} = \Omega_9 I_V^{**}$

$$dk((S_H, E, I_C, I_F, T_P, T_n, R, S_V, I_V) = \left(1 - \frac{S_H^{**}}{S_H}\right)\left(\frac{dS_H}{dt}\right) + y_1\left(1 - \frac{E^{**}}{E}\right)\frac{dE}{dt} + y_2\left(1 - \frac{I_C^{**}}{I_C}\right)\frac{dI_C}{dt} + y_3\left(1 - \frac{I_F^{**}}{I_F}\right)\frac{dI_F}{dt} + y_4\left(1 - \frac{T_P^{**}}{T_P}\right)\frac{dT_P}{dt} + y_5\left(1 - \frac{T_n^{**}}{T_n}\right)\frac{dT_n}{dt} + y_6\left(1 - \frac{R^{**}}{R}\right)\frac{dR}{dt} + y_7\left(1 - \frac{S_V^{**}}{S_V}\right)\frac{dS_V}{dt} + y_8\left(1 - \frac{I_V^{**}}{I_v}\right)\frac{dI_V}{dt} \quad (48)$$

Substituting $\frac{dS_H}{dt}, \frac{dE}{dt}, \frac{dI_C}{dt}, \frac{dI_F}{dt}, \frac{dT_P}{dt}, \frac{dT_n}{dt}, \frac{dR}{dt}, \frac{dS_V}{dt}, \frac{dI_V}{dt}$ in (48);

$$dk((S_H, E, I_C, I_F, T_P, T_n, R, S_V, I_V) = \left(1 - \frac{S_H^{**}}{S_H}\right)(\alpha S_H^{**} + \mu S_H^{**} - \mathrm{z} I_V^{**} + \mathrm{z} I_V - \Omega_1 S_H) + y_1\left(1 - \frac{E^{**}}{E}\right)(\alpha S_H + \rho R - \Omega_2 E) + y_2\left(1 - \frac{I_C^{**}}{I_C}\right)(\lambda E - \Omega_3 I_C) + \left(y_3\left(1 - \frac{I_F^{**}}{I_F}\right)(1-\lambda)E - \Omega_4 I_F\right) + y_4\left(1 - \frac{T_P^{**}}{T_P}\right)(\mathrm{ɯ} I_C - \Omega_5 T_P) + y_5\left(1 - \frac{T_n^{**}}{T_n}\right)((1-\mathrm{ɯ}) I_C - \Omega_6 T_n) + y_6\left(1 - \frac{R^{**}}{R}\right)(x T_P + (1-x) T_n + \gamma I_F - \Omega_7 R) + y_7\left(1 - \frac{S_V^{**}}{S_V}\right)(\wedge + \phi I_C + v I_F - \Omega_8 S_V) + y_8\left(1 - \frac{I_V^{**}}{I_v}\right)(\psi S_V - \Omega_9 I_V) \quad (49)$$

$$P = +\left(\frac{\beta(\eta_1 I_V(t) + \eta_2 I_F(t) + \eta_3 T_n(t) + \eta_4 T_P(t) + I_C}{E(t) + I_C(t) + I_F(t) + R(t) + S_H(t) + T_P(t) + T_n(t)} - 1\right) E(t)) + \left(\frac{I_C^{**}}{I_C(t)} - 1\right)\left(-\frac{\beta(\eta_1 I_V(t) + \eta_2 I_F(t) + \eta_3 T_n(t) + \eta_4 T_P(t) + I_C}{E(t) + I_C(t) + I_F(t) + R(t) + S_H(t) + T_P(t) + T_n(t)} + \mu \mathrm{IC}(t) + \omega \mathrm{IC}(t) + \phi \mathrm{IC}(t) + \theta \mathrm{IC}(t) - (\omega - 1)\mathrm{IC}(t)\right) + \left(\frac{I_F^{**}}{I_F(t)} - 1\right)(\epsilon \mathrm{IF}(t) + \gamma \mathrm{IF}(t) + \mu \mathrm{IF}(t) + v \mathrm{IF}(t) + \left(\frac{\beta(\eta_1 I_V(t) + \eta_2 I_F(t) + \eta_3 T_n(t) + \eta_4 T_P(t) + I_C}{E(t) + I_C(t) + I_F(t) + R(t) + S_H(t) + T_P(t) + T_n(t)} - 1\right) E(t)) + \left(\frac{I_V^{**}}{I_V(t)} - 1\right)(-\psi \mathrm{SV}(t) + \tau \mathrm{IV}(t) + z \mathrm{IV}(t)) + \left(\frac{R^{**}}{R(t)} - 1\right)(-\gamma \mathrm{IF}(t) + \mu R(t) + \rho R(t) - x \mathrm{TP}(t) + (x-1)\mathrm{Tn}(t)) + \left(\frac{S_H^{**}}{S_H(t)} - 1\right)(\alpha \mathrm{SH}(t) + \mu \mathrm{SH}(t) - \pi - z \mathrm{IV}(t)) + \left(\frac{T_P^{**}}{T_P(t)} - 1\right)(X \mathrm{TP}(t) + \epsilon \mathrm{TP}(t) + \mu \mathrm{TP}(t) - \omega \mathrm{IC}(t)) + \left(\frac{T_n^{**}}{T_n(t)} - 1\right)(e \mathrm{Tn}(t) + \mu \mathrm{Tn}(t) + (\omega - 1)\mathrm{IC}(t) - (x-1)\mathrm{Tn}(t)) \quad (50)$$

$$Q = -\left(\frac{E^{**}}{E(t)} - 1\right)(\alpha \mathrm{SH}(t) - \frac{\beta(\eta_1 I_V(t) + \eta_2 I_F(t) + \eta_3 T_n(t) + \eta_4 T_P(t) + I_C(t) E(t)}{E(t) + I_C(t) + I_F(t) + R(t) + S_H(t) + T_P(t) + T_n(t)} - \mu E(t) + \rho R(t) - \left(\frac{S_V^{**}}{S_V(t)} - 1\right)(\Lambda - \mu \mathrm{SV}(t) + \phi \mathrm{IC}(t) - \psi \mathrm{SV}(t) + v \mathrm{IF}(t)) \quad (51)$$

Where$\frac{dK}{dt} = 0$, holds only when $(S_H = S_H^{**}. E = E^{**}, I_C = I_C^{**}, I_F = I_F^{**}, T_P = T_P^{**}, T_n = T_n^{**}, R = R^{**}, S_V = S_V^{**},$ and$I_V = I_V^{**})$, then the maximal compact invariant set in $(S; E, I) \in \cap : \frac{dv}{dt} = 0$ is singleton E_*^{**} Lasalle's invariance principal, $\frac{dL(S,I,A,R)}{dt} < 0$ if and only if $P > Q$ [2]. This outcome indicates that malaria will continue to exist whenever$P > Q$, regardless of the initial conditions

Bifurcation analysis
We will employ this method to determine the existence of backward and forward bifurcation.

Theorem 5
The model exhibits a forward bifurcation at $R_O = 1$. Hence, the endemic equilibrium point $E^*$is locally asymptotically stable for $R_0 > 1$ but close to 1.

Proof

We perform a bifurcation analysis using the Centre Manifold Theorem (Liu and Zhang, 2011). We rewrite the human component of the system using notations for simplicity. Let$y_1 = S_H$, $y_2 = E$, $y_3 = I_C$, $y_4 = I_f$, $y_5 = T_n$, $y_6 = T_P$, $y_7 = R$, $y_8 = S_V$, and $y_9 = I_V$, such that $N = y_1 + y_2 + y_3 + y_4 + y_5 + y_6 + y_7 + y_8 + y_9$ (52)

Introducing vector notation, $(y = y_1, y_2, y_3, y_4, y_5, y_6, y_7, y_8, y_9)^T$, model system (3.1-3.9) can be written in the form , $\frac{dy}{dt} = F(y)$ with

$F = (f_1, f_2, f_3, f_4, f_5, f_6, f_7, f_8, f_9)^T$ as follows:

$\frac{dS_H}{dt} = f_1 = \pi + \mathrm{z}I_V - y_1 S_H$, $\frac{\mathrm{dE}}{\mathrm{dt}} = f_2 = \alpha S_H + \rho R - y_2 E$, $\frac{\mathrm{d}I_C}{\mathrm{dt}} = f_3 = \lambda E - y_3 I_C$, $\frac{\mathrm{d}I_F}{\mathrm{dt}} = f_4 = (1-\lambda)E - y_4 I_F$, $\frac{\mathrm{d}T_P}{\mathrm{dt}} = f_5 = \mathrm{ɯ} I_C - y_5 T_P$, $\frac{\mathrm{d}T_n}{\mathrm{dt}} = f_6 = (1-\mathrm{ɯ})I_C - y_6 T_n$, $\frac{\mathrm{dR}}{\mathrm{dt}} = f_7 = xT_P + (1-x)T_n + \gamma I_F - y_7 R$, $\frac{\mathrm{dS}_V}{\mathrm{dt}} = f_8 = \wedge + \phi I_C + vI_F - y_8 S_V$ and $\frac{\mathrm{d}I_V}{\mathrm{dt}} = f_9 = \psi S_V - y_9 I_V$ (53)

Where $\lambda = \beta^*(\frac{I_C + \beta_1 I_V + \beta_2 I_f + \beta_3 T_n + \beta_4 T_P}{N}$.

The Jacobian system (4.1.8.1) at disease-free equilibrium point is obtained as, where $\beta = \beta^*$

$$J(E^0, \beta^*) = \begin{pmatrix} -\Omega_1 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & \zeta \\ \alpha & \Omega_2 & 0 & 0 & 0 & 0 & \rho & 0 & 0 \\ 0 & 0 & -\Omega_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 1 & 0 & -\Omega_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \mathrm{ɯ} & 0 & -\Omega_5 & 0 & 0 & 0 & 0 \\ 0 & 0 & 1-\mathrm{ɯ} & 0 & 0 & -\Omega_6 & 0 & 0 & 0 \\ 0 & 0 & 0 & \gamma & x & 1-x & -\Omega_7 & 0 & 0 \\ 0 & 0 & \Phi & v & 0 & 0 & 0 & -\Omega_8 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \psi & -\Omega_9 \end{pmatrix} \quad (54)$$

Considering a case where $R_O = 1$and let $\beta = \beta^*$ is a bifurcation parameter. Therefore, solving β from $R_O = 1$, We get

$$\beta = \beta^* = \frac{\mu N}{\pi} \cdot [\frac{\mathrm{ɯ}}{\Omega_3 \Omega_5} + \frac{1-\mathrm{ɯ}}{\Omega_3 \Omega_6} + \frac{\gamma}{\Omega_4} + \frac{\eta_2}{\Omega_4} + \frac{\eta_3}{\Omega_6} + \frac{\eta_4}{\Omega_5} + \frac{\wedge \psi \eta_1}{\mu \Omega_9}]^{-1} \quad (55)$$

Where $J(E^*)$ with $\beta = \beta^*$has simple zero eigenvalue. Thus, applying Center Manifold theory to analyze dynamics of model around

$\beta = \beta^*$. Then $J(E^*)$ near $\beta = \beta^*$have both right eigenvector and a left eigenvector that match with zero eigenvalue given by $k = (k_1, k_2, k_3, k_4, k_5, k_6, k_7, k_8, k_9)^T$ and $l = (l_1, l_2, l_3, l_4, l_5, l_6, l_7, l_8, l_9)^T$,respectively. Multiplying right eigenvector k with $J(E^0, \beta^*)$ and equating to zero. We solve to obtain right eigenvector k and left eigenvector l.

Let $k_1 = 1$, then $k_2 = \frac{1}{\Omega_4}$, $k_3 = \frac{1}{\Omega_2 \Omega_4}$, $k_4 = \frac{1-\mathrm{ɯ}}{\Omega_3 \Omega_6}$, $k_5 = \frac{\mathrm{ɯ}}{\Omega_3 \Omega_5}$,$k_6 = \frac{\gamma}{\Omega_1 \Omega_7} + \frac{1-x}{\Omega_6 \Omega_7} + \frac{x}{\Omega_5 \Omega_7}$ and $k_7 = \frac{\Phi}{\Omega_3 \Omega_9} + \frac{v}{\Omega_4 \Omega_9}$

Also let $l_1 = 1$, then $l_2 = \frac{1}{\Omega_2}$, $l_3 = \frac{1}{\Omega_2 \Omega_4}$, $l_4 = \frac{1-x}{\Omega_6 \Omega_7}$, $l_5 = \frac{x}{\Omega_5 \Omega_7}$, $l_6 = \frac{\rho}{\Omega_2 \Omega_7}$, $l_7 = \frac{\Phi}{\Omega_3 \Omega_9} + \frac{v}{\Omega_4 \Omega_9}$ respectively.

Finding transpose of $J(E^0, \beta^*)$, we obtain

$$J(E^0, \beta^*)^T = \begin{pmatrix} -\Omega_2 & 0 & 1 & 0 & 0 & 0 & 0 \\ 0 & -\Omega_3 & 0 & 1-\mathrm{ɯ} & \mathrm{ɯ} & 0 & \Phi \\ 0 & 0 & -\Omega_4 & 0 & 0 & \gamma & v \\ 0 & 0 & 0 & -\Omega_6 & 0 & 1-x & 0 \\ 0 & 0 & 0 & 0 & -\Omega_5 & x & 0 \\ \rho & 0 & 0 & 0 & 0 & -\Omega_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -\Omega_9 \end{pmatrix} \quad (56)$$

Multiply the left eigenvector l with matrix $J(E^0, \beta^*)^T$ and equating to zero. Solving to obtain $l_1 = 1$, then $l_2 = \frac{1}{\Omega_2}$, $l_3 = \frac{1}{\Omega_2 \Omega_4}$, $l_4 = \frac{1-x}{\Omega_6 \Omega_7}$, $l_5 = \frac{x}{\Omega_5 \Omega_7}$, $l_6 = \frac{\rho}{\Omega_2 \Omega_7}$, $l_7 = \frac{\Phi}{\Omega_3 \Omega_9} + \frac{v}{\Omega_4 \Omega_9}$, therefore l lies in the left null space of the Jacobian — satisfying the condition for applying Center Manifold Theory near $R_O = 1$ with $\beta = \beta^*$

Now compute for bifurcation coefficients a and b . Since$l_1 = 0$. We will compute partial derivatives of $f_1, f_2, \ldots, f_9$at disease free equilibrium point. For the model, the associated nonzero partial derivatives of $f_1, f_2, \ldots, f_9$ is given by ; nonzero second order partial derivatives as follows; $f_3 = \lambda E - \Omega_3 I_C$:

$$\frac{\partial^2 f_3}{\partial I_C \partial E} = \frac{\beta}{N}, \frac{\partial^2 f_3}{\partial I_V \partial E} = \frac{\beta \eta_1}{N}, \frac{\partial^2 f_3}{\partial I_F \partial E} = \frac{\beta \eta_2}{N}, \frac{\partial^2 f_3}{\partial T_n \partial E} = \frac{\beta \eta_3}{N} \text{ and } \frac{\partial^2 f_3}{\partial T_P \partial E} = \frac{\beta \eta_4}{N} \quad (57)$$

$f_4 = (1-\lambda)E - \Omega_4 I_F$:

$$\frac{\partial^2 f_4}{\partial I_C \partial E} = -\frac{\beta}{N}, \frac{\partial^2 f_4}{\partial I_V \partial E} = -\frac{\beta \eta_1}{N}, \frac{\partial^2 f_4}{\partial I_F \partial E} = -\frac{\beta \eta_2}{N}, \frac{\partial^2 f_4}{\partial T_n \partial E} = -\frac{\beta \eta_3}{N} \text{ and } \frac{\partial^2 f_4}{\partial T_P \partial E} = -\frac{\beta \eta_4}{N} \quad (58)$$

Computing bifurcation coefficients a and b as follows:

$$a = \frac{\beta k_2}{N}(l_3 - l_4)(k_3 + \eta_1 k_9 + \eta_2 k_4 + \eta_3 k_5 + \eta_4 k_6)$$

$$\text{And } b = \frac{k_2}{N}(l_3 - l_4)(k_3 + \eta_1 k_9 + \eta_2 k_4 + \eta_3 k_5 + \eta_4 k_6) \quad (59)$$

If a<-0 and b>0b > 0b>0, the model exhibits a forward bifurcation, where the disease-free equilibrium is locally asymptotically stable when R_O<1, and a unique endemic equilibrium exists and is stable when R_O>1.
If a>0and b>0 the model exhibits a backward bifurcation, indicating the possibility of multiple endemic equilibria coexisting with the disease-free state when R_O<1, and thus, control strategies must lower $R_O$significantly below 1 to eliminate the disease. Hence prove.

Simulations parameters of the model
Utilizing the fourth-order Runge-Kutta method in Matlab, model simulation is carried out and numerical simulations are performed, which is used to study dynamic behavior of model state variables using model parameters. Numerical simulations is performed taking care of initial conditions and parameters provided above and results of are presented graphically as shown below.

Normalizing sensitivity analysis of basic reproduction number
A crucial method in mathematical epidemiology is sensitivity analysis, which evaluates the impact of model parameter changes on significant outcomes such as the fundamental reproduction number R_O. Relative changes in outcomes brought on by parameter changes are measured by the normalized forward sensitivity index. This analysis helps prioritize intervention strategies by highlighting the parameters that significantly impact disease transmission. A positive index suggests that increasing the parameter boosts disease spread, while a negative index indicates that increasing the parameter aids in controlling the disease.

Table 1: For Normalized Sensitivity Indices of Parameters.

Parameters	Sensitivity index, R0R_0R0
β	+1.0000
η₁	+0.1964
η₂	+0.2455
η₃	+0.1498
η₄	+0.0783
Φ	-0.2541
Θ	-0.1906
Ω	0.0000
ν	-0.2156
E	-0.1672
Γ	-0.1275
X	-0.0513
μ_H	-0.3408
z	-0.2957
τ	-0.2957

The sensitivity analysis shows that the transmission rate (β) has the largest positive influence on R_O (β= +1.0000), meaning that any proportional change in results in an equivalent proportional change inβ. This confirms that reducing transmission through interventions. The infectivity parameters (η_1, η_2, η_3 *and* η_4) also have positive indices, indicating that increased infectiousness from specific compartments particularly non-conflict infected humans and infectious mosquitoes will significantly increase. Negative sensitivity indices identify parameters that help reduce transmission when increased. The human death rate (μ_H) and mosquito mortality parameters (Z) and (τ) are among the most influential, suggesting that measures that shorten the life span of mosquitoes or infected individuals substantially reduce . Recovery and disease progression rates (Φ,Θ , ν, e,ϵ ,γ , x, ψ) also contribute negatively, with (Φ) and ν being particularly important as they shorten the infectious period. Interestingly, the treatment initiation rate (w) has no direct effect on R_O in this formulation, indicating that its influence may operate indirectly through other model processes. In summary, the most effective strategies for controlling malaria in this setting should focus on reducing, increasing mosquito mortality rates (z ,τ), and accelerating recovery or removal from infectious states (Φ, ν,), particularly in conflict-affected areas where treatment access is constrained.

Malaria's effects on the entire population in the absence of treatment

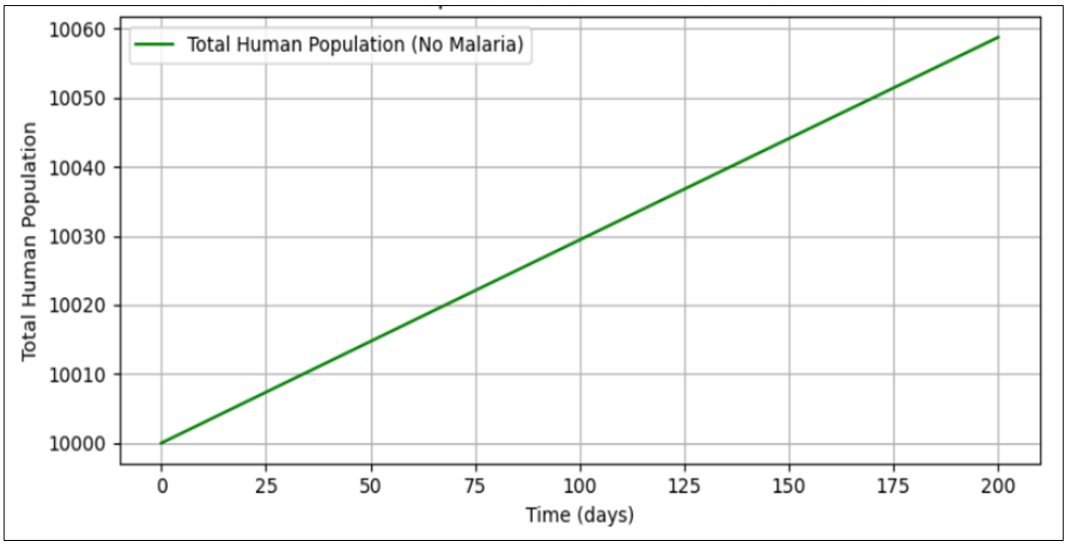


Fig 3: Total human population over time in absence of malaria

The dynamics of the entire human population over time, without malaria infection, are depicted in the graph. When malaria transmission parameters are set to zero, natural birth and death rates alone control population change; neither disease-induced mortality nor infection-related transitions occur. The curve shows a gradual and steady increase in total population due to the consistent recruitment rate exceeding the natural mortality rate. Since no individuals enter exposed or infectious compartments, the entire population remains in the susceptible and recovered states, with minimal losses due to natural death. This trend reflects the ideal scenario where, in the absence of malaria, the human population would grow predictably without disruption, underscoring the significant demographic burden malaria imposes in endemic settings.

The impact of malaria on treated population over time

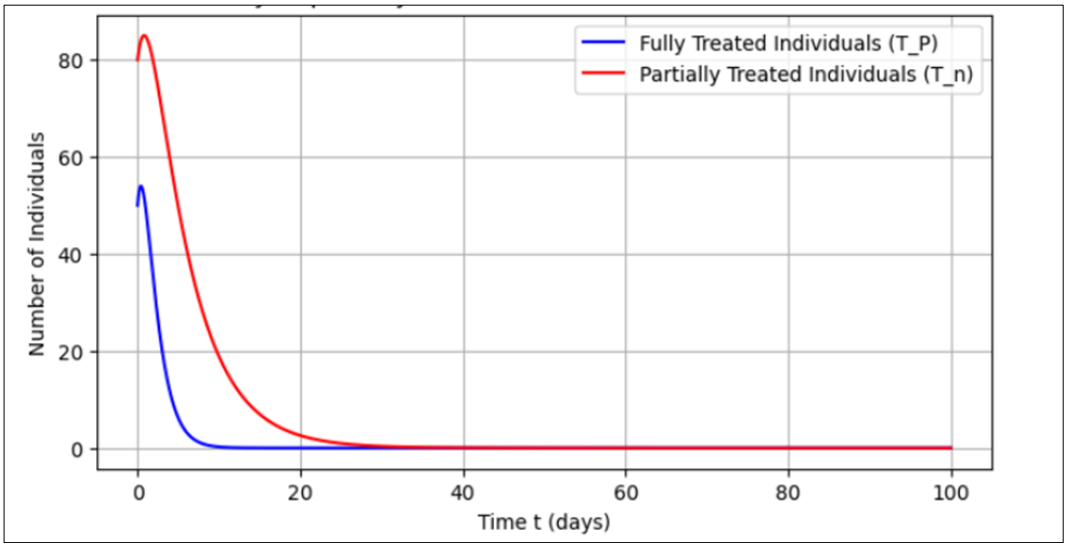


Fig 4: Fully vs partially treated malaria individuals over time

The graph illustrates the progression of malaria-infected individuals undergoing full treatment (T_P) and partial treatment (T_n) over time. Initially, the number of partially treated individuals is higher, but both compartments exhibit a declining trend as treatment progresses. Fully treated individuals decline more rapidly, reflecting the higher recovery and transition rates associated with effective treatment. In contrast, the slower decline in the partially treated group suggests lower recovery efficacy or incomplete adherence, resulting in prolonged infection duration. Over time, both populations approach zero, indicating the eventual clearance of infections with sustained treatment efforts. This highlights the critical importance of full treatment coverage to effectively reduce the malaria burden.

The impact of malaria in conflict zones and other zones

Conflict and non-conflict areas' total numbers of malaria-infected people throughout time are contrasted in the figure below. It makes it abundantly evident that the burden of infection is far larger and more persistent in the conflict zone. This is due to the increased transmission rate and reduced treatment access modeled for conflict zones, resulting in a rapid rise and slower decline of infections. In contrast, the non-conflict zone shows a lower peak and quicker stabilization, reflecting better healthcare access and

treatment efficacy. This trend highlights the critical impact of conflict on malaria dynamics, emphasizing the need for targeted intervention and healthcare support in vulnerable regions.

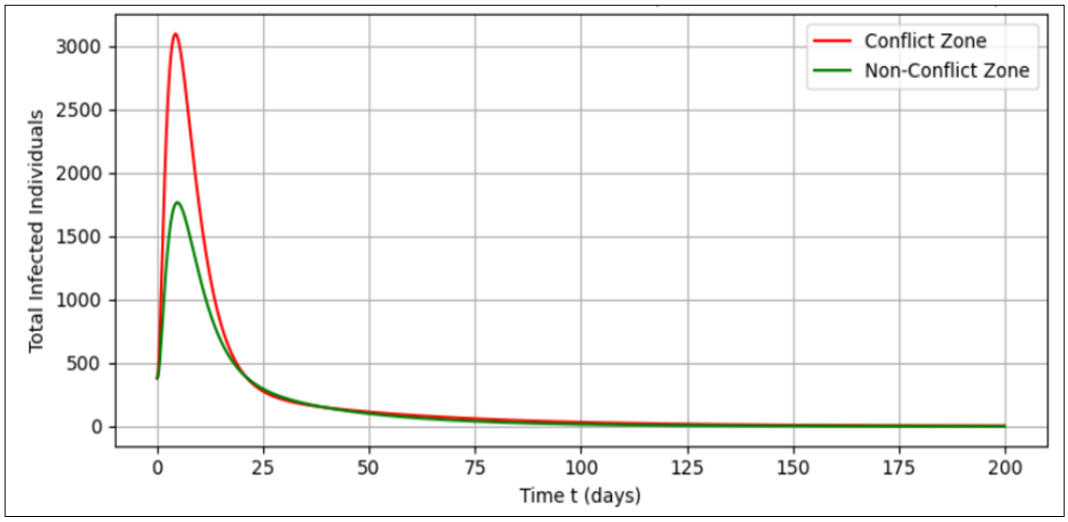


Fig 5: Total malaria infected individuals over time (Conflict vs Non-conflict Zone)

Implication of malaria treatment on total population over time

The graph compares the total human population over time under two scenarios: with malaria treatment and without malaria treatment. In the untreated case (red dashed line), the total population declines more rapidly due to increased morbidity and mortality caused by uncontrolled malaria infections. Conversely, the treated case (green solid line) shows a more stable population size, with only a mild decline followed by gradual stabilization. This demonstrates that effective treatment not only reduces the disease burden but also helps maintain a healthier and more stable population size. The divergence between the two curves becomes more pronounced over time, emphasizing the long-term benefits of implementing widespread malaria treatment programs.

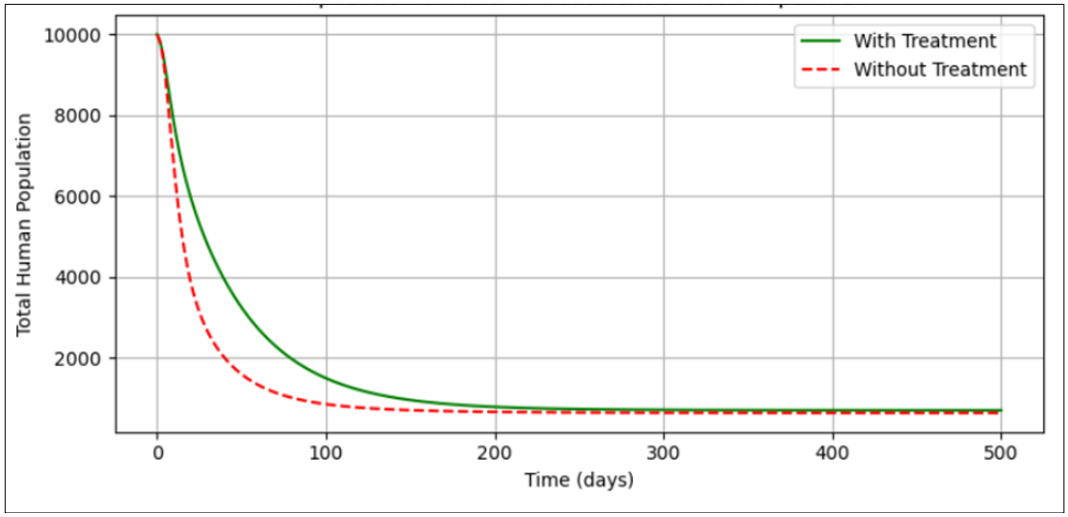


Fig 6: Infection of malaria treatment on total population

Effects of Malaria Recovery rate on total population over time

The graph below illustrates the implications of varying recovery rates (γ) on the total human population over time in a malaria-endemic setting. In contrast to situations with lower recovery rates (γ=0.05), which result in more substantial population reduction or stagnation, scenarios with higher recovery rates (γ=0.2) preserve a more stable and larger overall population. This is because a slower recovery rate causes people to take longer to recover from an infection, which lengthens the period of illness and in turn increases the disease burden and death from malaria. Conversely, higher recovery rates help reduce the infectious population more rapidly, mitigating disease spread and associated deaths, thereby preserving the total population size. The graph underscores the critical role of improving recovery (e.g., through effective treatment) in sustaining population health and controlling malaria impact.

Relationship between total treated and total infected individuals

The graph below illustrates a positive but nonlinear relationship between total treated individuals and total infected individuals. Initially, an increase in infections leads to a steady rise in treated individuals, indicating a responsive healthcare system. However, as infections escalate, particularly in conflict zones, the growth of treated individuals levels off, suggesting a saturation or delay in treatment coverage. This could be attributed to healthcare infrastructure limitations and access challenges. Furthermore, the fact

that partial treatment exceeds full treatment points to a significant treatment gap, emphasizing the need for improved healthcare systems and targeted interventions in conflict-affected areas.

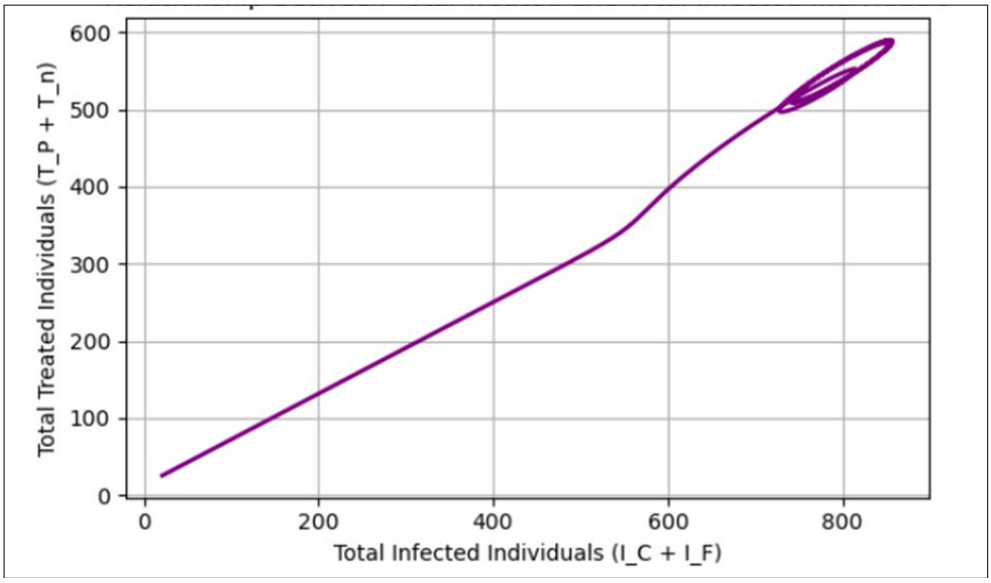


Fig 7: Relationship between total related and total infected individuals

Conclusion

We developed a mathematical model for the transmission and treatment of malaria in conflict areas in this research. It looks at the stability of equilibrium points that are endemic and disease-free. The results show that the disease-free equilibrium is both locally and globally stable when the basic reproduction number R_O is less than 1, indicating that lowering R_O below one can stop the spread of illness. On the other hand, when R_O >1, the endemic equilibrium is asymptotically stable. Malaria spreads more quickly in conflict areas than in other areas, according to numerical data, yet the disease can be completely eradicated with the right intervention.

Conflict of Interest

None of the authors' personal or financial ties might have an inappropriate influence on their work. They don't have any personal or professional ties to any businesses, goods, or services that could affect the evaluation or content of their papers review.

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