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MATHEMATICAL MODELING OF MALARIA DISEASE WITH CONTROL STRATEGY

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Abstract. This article suggested and analyzed the transmission dynamics of malaria disease in a population using a nonlinear mathematical model. The deterministic compartmental model was examined using stability theory of differential equations. The reproduction number was obtained to be asymptotically stable conditions for the disease-free, and the endemic equilibria were determined. Moreso, the qualitatively evaluated model incorporates time-dependent variable controls which was aimed at reducing the proliferation of malaria disease. The optimal control problem was formulated using Pontryagin's maximum principle, and three control strategies: disease prevention through bed nets, treatment and insecticides were incorporated. The optimality system was stimulated using an iterative technique of forward-backward Runge-Kutta fourth order scheme, so that the impacts of the control strategies on the infected individuals in the population can be determined. The possible influence of exploring a single control, the combination of two, and the three controls on the spread of the disease was also investigated. Numerical simulation was carried out and pertinent findings are displayed graphically.

Keywords: malaria; optimal control; female anopheles mosquito; stability.

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1. INTRODUCTION

Malaria, a life-threatening disease which happens to be a vector borne, and is one of the major deadly infectious diseases worldwide. [1, 2, 3, 4]. The disease is caused by the protozoan Plasmodium and it is transmitted in humans by an effective bite of an infected adult female Anopheles mosquito (the malaria vector) [1, 5, 6]. The species causing agents include; (*Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale,* and *Plasmodium malariae*).These aforementioned species differ in microscopic appearance, geographical distribution, and clinical characteristics. By clinical characteristics, we refer to the infection potential, severity, and the ability to cause relapse. Among all the species, *P. falciparum* has been recognized as the most dangerous to humans [6].

According to the latest World malaria report, released in December 2019, there were 228 million cases of malaria in 2018 compared to 231 million cases in 2017. The estimated number of malaria deaths stood at 405 000 in 2018, compared with 416 000 deaths in 2017.

The WHO African Region continues to carry a disproportionately high share of the global malaria burden. In 2018, the region was home to 93% of malaria cases and 94% of malaria deaths. The prevalent of malaria is in over 100 countries, with approximately 216 millions cases, and 655,000 deaths in 2010. [7, 8]. In various parts of the world, malaria has been widespread for many decades [5, 9], yet it still remains a major public health burden in affected areas, predominantly the tropical and subtropical areas in Africa, Eastern Mediterranean Regions, Asia and South America. In addition, the high risk groups include pregnant women, non-immune travelers.

Health related problems aside, malaria constitutes a great socioeconomic threat to malaria-endemic nations. For instance, as documented by[2, 5, 10], in Africa alone, $8 billion was estimated to be the annual economic burden of malaria. These reports have made it essential to explore the possibility of formulating numerous intervention strategies to alleviate the influence of malaria in many countries. As cited by [5], there is currently no perfect vaccine against malaria in humans (although collective global efforts are under way to develop such a vaccine [11, 12, 13, 14, 15, 16, 17]. In order to control the spread of malaria, preventive measures

include mosquito-reduction strategies and self-protection against mosquito bites (via the use of insecticide treated bed nets (ITNs); intermittent preventive treatment (IPT), and the reduction of vector population through the destruction of their breeding sites [2, 18, 19, 20]. Studies also revealed other intervention strategies, such as the use of indoor residual spraying (IRS) for killing infected indoors mosquitoes through the use of sterile insect technique [2, 21], and the use of anti-malaria drugs to regulate malaria (see, for instance, [6, 22, 23, 24, 25]).

Over the years, several numbers of mathematical models on the transmission dynamics of malaria have been examined. Following the simple S-I-R malaria model of Ross [5, 26] and Macdonald [5, 27], many researchers have elaborated these models by incorporating different features associated to malaria transmission dynamics and its control. This includes modeling the effect of age groups on the transmission of malaria, as shown by [5, 28, 29]; the use of preventive and therapeutic strategies [25]; repeated exposure [30]; impact of climate variables such as temperature and rainfall [31, 1, 32, 33].

In addition to these, a number of papers has been published on the use of optimal control theory in investigating optimal strategies for malaria control in the community. The influence of three control variables (vector-reduction, blood screening strategies and personal protection) on malaria dynamics with indirect and direct transmissions was presented by [34], while bed net and optimal vaccination control efforts in the populations with diverse ranks of naturally acquired immunity were described by [35]. [36] used optimal control theory to explore optimal approaches in controlling malaria; using insecticide treated bed nets (ITNS), spray of mosquito insecticide and treatment as the system control variables, while [37] examined the dynamics of vector-borne disease with nonlinear prevalence by employing similar control variables as in [36]. The optimal control and transmission dynamics of malaria in Kenya was investigated in [38],by incorporating four-time dependent control strategies, namely: insecticide treated bed nets (ITNs), indoor residual spray (IRS), treatment and intermittent preventive treatment of malaria in pregnancy (IPTp).

Motivated by the above studies, we design and develop a dynamical mathematical model to examine the transmission dynamics of malaria. We further explore the best control strategies to

curb the disease, through the use of optimal control theory. Specifically, we apply Pontryagin's Maximum Principle (PMP) to ascertain the required conditions for the optimal approaches to control the spread of malaria. The organization of the paper is as follows: section 2 discusses the model formulation, the basic quantitative properties of the model will be the focus in section 3, while the stability analysis of the system equilibrium points will be considered in section 4. Optimal control analysis is presented in section 5, and the discussion of results follows in sections 6 and 7.

2. PRELIMINARIES

3. MODEL FORMULATION

The model sub-divides the total human population at time t, denoted by $N_h(t)$, into susceptible humans (S_h), infected humans (I_h), and recovered humans (R_h). Thus, the total human population is given by

$$N_h(t) = S_h + I_h + R_h \tag{1}$$

Furthermore, the model sub-divides the total vector population at time t, denoted by $N_v(t)$, into immature mosquitoes (A_v) susceptible mosquitoes (S_v), and infected mosquitoes (I_v). Thus, the total vector population is given by

$$N_v(t) = A_v + S_v + I_v \tag{2}$$

As the disease progresses based on the disease status, individuals move from one class to the other. Individuals are recruited into the susceptible human population by either via birth or immigration at the rate π_h, and also by the loss of immunity of recovered humans at a rate ψ. The humans population susceptible is depopulated by infection subsequent to contact with infectious vectors at a rate $\lambda_h(b)$, defined as

$$\lambda_h(b) = \frac{\beta_{hv}\varepsilon(b)I_v}{N_h} \tag{3}$$

The parameter β_{hv} is the probability of effective transmission from human to mosquitoes, following the contact rate of mosquito to human by the rate $\varepsilon(b)$. Furthermore, the population of

susceptible humans are decreased by natural death, at a rate μ. Thus, the rate of change of the population of susceptible human is given by

$$\frac{dS_h}{dt} = \pi_h + \psi R_h - \lambda_h(b)S_h - \mu_h S_h \tag{4}$$

The population of infected humans individuals is generated by the infection of susceptible humans (at the rate $\lambda_h(b)$) and is decreased by recovery of infected individuals (at a rate τ_h), natural death (at a rate μ_h) and disease induced death (at a rate δ_h), so that

$$\frac{dI_h}{dt} = \lambda_h(b)S_h - (\tau_h + \mu_h + \delta_h)I_h \tag{5}$$

Finally on human population, the population of recovered humans is generated by the recovery of infected humans (at a rate τ_h). It is decreased by loss of immunity and natural death (at a rate ψ and μ_h respectively). Thus, the recovered humans population is given by

$$\frac{dR_h}{dt} = \tau_h I_h - (\psi + \mu_h)R_h \tag{6}$$

The immature mosquitoes are populated by mosquitoes egg deposition (at a rate π_v(b)), and reduced by maturation of immature mosquitoes (at a rate γ_v) and natural mortality (at a rate $\mu_v(q)$). Thus, the immature mosquitoes population is given by

$$\frac{dA_v}{dt} = \pi_v(q) - (\gamma_v + \mu_v(q))A_v \tag{7}$$

The population of susceptible mosquitoes is generated by the maturation of immature mosquitoes (at a rate γ_v). It is further depopulated by infection following effective contact with infectious humans at a rate $\lambda_v(b)$, defined as

$$\lambda_v(b) = \frac{\beta_{vh}\varepsilon(b)I_h}{N_h} \tag{8}$$

The parameter β_{vh} is the probability of effective transmission from mosquitoes to humans, following the contact rate of mosquito to human by the rate $\varepsilon(b)$. Furthermore, the population of susceptible vectors are decreased by natural death, at a rate μ_v. Thus, the rate of change of the population of susceptible vectors is given by

$$\frac{dS_v}{dt} = \gamma_v A_v - \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))S_v \tag{9}$$

Finally, the population of infected vectors is generated by the infection of susceptible vectors (at a rate $\lambda_v(b)$) and is decreased by natural death of mosquitoes(at a rate μ_v). The rate of change of the population of infected vectors is given by

$$\frac{dI_v}{dt} = \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))I_v \tag{10}$$

Hence, following the above descriptions, the transmission dynamics of malaria in the population is given by the following nonlinear system of ordinary differential equations

$$\begin{aligned}
\frac{dS_h}{dt} &= \pi_h + \psi R_h - \lambda_h(b)S_h - \mu_h S_h \\
\frac{dI_h}{dt} &= \lambda_h(b)S_h - (\tau_h + \mu_h + \delta_h)I_h \\
\frac{dR_h}{dt} &= \tau_h I_h - (\psi + \mu_h)R_h \\
\frac{dA_v}{dt} &= \pi_v(q) - (\gamma_v + \mu_v(q))A_v \\
\frac{dS_v}{dt} &= \gamma_v A_v - \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))S_v \\
\frac{dI_v}{dt} &= \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))I_v
\end{aligned} \tag{11}$$

where,

$$\varepsilon(b) = \varepsilon_{max} - b\left(\varepsilon_{max} - \varepsilon_{min}\right), \qquad \mu_v(b) = \mu_v + \mu_{max}b, \qquad for \quad 0 \leq b \leq 1$$

$$\pi_v(q) = \pi_{vmax} - q\left(\pi_{vmax} - \pi_{vmin}\right), \qquad \mu_v(q) = \mu_v + \mu_{max}q, \qquad for \quad 0 \leq q \leq 1$$

Variable	Description
S_h	Susceptible Humans
I_h	Infected Humans
R_h	Recovered Humans
A_v	Immature Mosquitoes
S_v	Susceptible Mosquitoes
I_v	Infected Mosquitoes
Parameter	**Description**
π_h	Recruitment rate of humans
ψ	Per captia rate of loss of immunity in humans
μ_h	Natural mortality rate of humans
τ_h	Recovery rate of infectious individuals
δ_h	Disease – induced death rate of humans
β_{hv}	Probability of effective transmission from human to mosquitoe
β_{vh}	Probability of effective transmission from mosquitoe to human
$\varepsilon(b)$	Contact rate of mosquito - human
$\varepsilon(max)$	Maximum mosquito biting rate
$\varepsilon(min)$	Minimum mosquito biting rate
b	Proportion of treated net usage
q	Proportion of insecticide spray on the environment
η	Modification parameter
π_v	Egg deposition rate of mosquitoes
γ_v	Maturation rate of immature mosquitoes
μ_v	Natural mortality rate of mosqitoes
$\mu_{max}(b)$	Mortality rate of mosqitoes due to treated net
$\mu_{max}(q)$	Mortality rate of mosqitoes due to insecticide spray

Table 1: Description of the variables and parameters of the model (11).

4. Basic Quantitative Properties of the Model

4.1. Positivity and Boundedness of solutions. Here the basic properties of the malaria model (11) will be explored. Holding on to the reality of human and vector population, for the malaria model (11) to be epidemiologically meaningful, it is important to establish that all its state variables are non-negative for all time $t \geq 0$. In other words, solutions of the model system (11) with non-negative initial condition will remain non-negative for all time $t > 0$.

Theorem 3.1. *Let the initial conditions for the malaria model* (11) *be* $S_h(0) > 0, I_h(0) \geq 0, R_h \geq 0, A_v > 0, S_v > 0, I_v \geq 0$. *Then the solutions* $(S_h, I_h, R_h, A_v, S_v, I_v)$ *of the model with positive initial conditions, will remain positive for all time* $t > 0$.

Proof. Let $t_1 = \sup\{t > 0 : S_h(t) > 0, I_h(t) > 0, R_h(t) > 0, A_v(t) > 0, S_v(t) > 0, I_v(t) > 0 \in [0,t]\}$. Thus, $t_1 > 0$. It follows from the first equation of the system (11), that

$$\frac{dS_h}{dt} = \pi_h + \psi R_h - \lambda_h(b)S_h - \mu_h S_h \geq \pi_h - \lambda_h(b)S_h - \mu_h S_h \tag{12}$$

using the integrating factor, this can be written as:

$$\frac{d}{dt}\left(S_h(t)exp\left[\mu_h t + \int_0^t \lambda_h(b)(u)du\right]\right) \geq \pi_h exp\left[\mu_h t + \int_0^t \lambda_h(b)(u)du\right]$$

Hence,

$$S_h(t_1)exp\left[\mu_h t_1 + \int_0^{t_1} \lambda_h(b)(u)du\right] - S_h(0) \geq \int_0^{t_1} \pi_h \left(exp\left[\mu_h \zeta + \int_0^{\zeta} \lambda_h(b)(u)du\right]\right) d\zeta$$

so that,

$$\begin{aligned} S_h(t_1) &\geq S_h(0)exp\left[-\mu_h t_1 - \int_0^{t_1} \lambda_h(b)(u)du\right] \\ &+ exp\left[-\mu_h t_1 - \int_0^{t_1} \lambda_h(b)(u)du\right] \times \int_0^{t_1} \pi_h \left(exp\left[\mu_h \zeta + \int_0^{\zeta} \lambda_h(b)(u)du\right]\right) d\zeta > 0. \end{aligned}$$

Similarly, it can be shown that $I_h(t) \geq 0$, $R_h(t) \geq 0$, $A_v(t) > 0$, $S_v(t) > 0$, and $I_v(t) \geq 0$ for all time $t > 0$. Therefore, all the solutions of the model (11) remain positive for all non-negative

initial conditions.

Consider the biologically feasible region consisting of $\mathscr{D} = \mathscr{D}_h \cup \mathscr{D}_v \subset \mathscr{R}^3_+ \times \mathscr{R}^3_+$ with

$$\mathscr{D}_h = \left\{ S_h, I_h, R_h \in \mathscr{R}^3_+ : N_h \leq \frac{\pi_h}{\mu_h} \right\}$$

and

$$\mathscr{D}_v = \left\{ A_v, S_v, I_v \in \mathscr{R}^3_+ : N_v \leq \frac{\pi_v(q)}{\mu_v} \right\}$$

where $\mu_v = \mu_v(b) + \mu_v(q)$.

Theorem 3.2 *The region $\mathscr{D} = \mathscr{D}_h \cup \mathscr{D}_v \subset \mathscr{R}^3_+ \times \mathscr{R}^3_+$ is positively-invariant for the malaria model* (11) *with non-negative initial conditions in $\mathscr{R}^6_+$.*

Proof. It follows from the model (11) using (1) and (2) it gives:

$$\begin{aligned} \frac{dN_h(t)}{dt} &= \pi_h - \mu_h N_h(t) - \delta_h I_h(t) \\ \frac{dN_v(t)}{dt} &= \pi_v(q) - \mu_v N_v(t) - \mu_v(b) A_v \end{aligned}$$

so that,

$$\frac{dN_h(t)}{dt} \leq \pi_h - \mu_h N_h(t), \quad \textit{and} \qquad \frac{dN_v(t)}{dt} \leq \pi_v(q) - \mu_v N_v(t) \tag{13}$$

Since the term $\delta_h I_h(t)$ and $\mu_v(b)A_v$ are non-negative hence, $N_h(t) \leq N_h(0)e^{-\mu_h t} + \frac{\pi_h}{\mu_h}(1 - e^{-\mu_h t})$ and $N_v(t) = N_v(0)e^{-\mu_v t} + \frac{\pi_v(q)}{\mu_v}(1 - e^{-\mu_v t})$. In particular, $N_h(t) \leq \frac{\pi_h}{\mu_h}$ and $N_v(t) \leq \frac{\pi_v(q)}{\mu_v}$, if $N_h(0) \leq \frac{\pi_h}{\mu_h}$ and $N_v(0) \leq \frac{\pi_v(q)}{\mu_v}$. Thus, the region is positively-invariant. Furthermore, if $N_h(t) > \frac{\pi_h}{\mu_h}$ and $N_v(t) > \frac{\pi_v(q)}{\mu_v}$, then either the solutions enters $\mathscr{D}$ in finite time, or $N_h(t)$ approaches $\frac{\pi_h}{\mu_h}$ and $N_v(t)$ approaches $\frac{\pi_v(q)}{\mu_v}$ asypmtotically. Hence, the region $\mathscr{D}$ attracts all solution in $\mathscr{R}^6_+$.

5. Local Stability of the Disease Free Equilibrium (DFE)

The malaria model (11) has a disease free equilibrium obtained by setting the right-hand sides of the equations in the model to zero and solving at $I_h = I_v = 0$. Thus, the disease free

equilibrium is given by

$$\begin{aligned} \phi_0 &= (S_h^*, I_h^*, R_h^*, A_v^*, S_v^*, I_v^*) \\ &= \left[\frac{\pi_h}{\mu_h}, 0, 0, \frac{\pi_v(q)}{(\gamma_v+\mu_v(q))}, \frac{\gamma_v\pi_v(q)}{(\gamma_v+\mu_v(q))(\mu_v(b)+\mu_v(q))}, 0\right] \end{aligned} \tag{14}$$

The local stability of the DFE, ϕ_0 will be explored using the next generation operator method [39, 40]. The matrices F of the new infection terms of the model (11), and V, of the transition terms of the model (11), are given respectively by

$$F = \begin{pmatrix} 0 & \beta_{hv}\varepsilon(b) \\ \frac{\beta_{vh}\varepsilon(b)S_v^*}{S_h^*} & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} k_1 & 0 \\ 0 & k_4 \end{pmatrix} \tag{15}$$

It follows that, the reproduction number is given by $\mathscr{R}_0 = \rho(FV^{-1})$, where ρ is the spectral radius of the matrix. Hence,

$$\mathscr{R}_0 = \sqrt{\frac{S_v^*\beta_{hv}\beta_{vh}\varepsilon^2(b)}{k_1k_4S_h^*}} \tag{16}$$

Substituting S_h^* and S_v^* into (16) as defined in (14) yields

$$\mathscr{R}_0 = \sqrt{\frac{\beta_{hv}\beta_{vh}\gamma_v\pi_v(q)\mu_h\varepsilon^2(b)}{k_1k_3k_4^2\pi_h}} \tag{17}$$

Hence,

$$\mathscr{R}_0 = \sqrt{\mathscr{R}_h\mathscr{R}_v}$$

where

$$\mathscr{R}_h = \frac{\beta_{hv}\mu_h\varepsilon(b)}{\pi_hk_1}, \qquad \mathscr{R}_v = \frac{\beta_{vh}\gamma_v\pi_v(q)\varepsilon(b)}{k_3k_4^2}$$

where, $k_1 = \tau_h + \mu_h + \delta_h$, $k_2 = \psi + \mu_h$, $k_3 = \gamma_v + \mu_v(q)$ and $k_4 = \mu_v(b) + \mu_v(q)$.

The result below follows from Theorem 2 of [39].

Theorem 3.1. *The malaria model* (11) *disease-free equilibrium* ϕ_0 *is locally-asymptotically stable (LAS) if* $\mathscr{R}_0 < 1$ *and unstable if* $\mathscr{R}_0 > 1$.

The basic reproduction number $\mathscr{R}_0$ measures the average number of new infections generated by a single infected person during his or her infectious period in a population that is fully susceptible [41, 42]. Thus, the epidemiological implication of theorem 3.1 above is that malaria will be controlled in the population whenever $\mathscr{R}_0 < 1$ if the initial sizes of the population of the model are in the basin of attraction of the DFE $(\mathscr{E}_0)$.

5.1. Existence of endemic equilibrium point (EEP). Here, we will explore the possible equilibra of the model and establish the conditions for the existence of an equilibrium for which malaria is endemic in the population. The infection equilibria of the malaria model (11) represented as $\phi_1 = (S_h^{**}, I_h^{**}, R_h^{**}, A_v^{**} S_v^{**}, I_v^{**})$ is obtained by equating the right hand side of the equations in (11) to be zero and solve simultaneouly. Hence, the endemic equilibria is given below as

$$
\begin{aligned}
S_h^{**} &= \frac{\pi_h k_1 k_2}{k_1 k_2 \left[\lambda_h^{**}(b) + \mu_h\right] - \tau_h \psi \lambda_h^{**}(b)}, & A_v^{**} &= \frac{\pi_v(q)}{k_3}, \\
I_h^{**} &= \frac{\lambda_h^{**}(b) \pi_h k_2}{k_1 k_2 \left[\lambda_h^{**}(b) + \mu_h\right] - \tau_h \psi \lambda_h^{**}(b)}, & S_v^{**} &= \frac{\gamma_v \pi_v(q)}{k_3 \left[\lambda_v^{**}(b) + k_4\right]}, \\
R_h^{**} &= \frac{\lambda_h^{**}(b) \tau_h \pi_h}{k_1 k_2 \left[\lambda_h^{**}(b) + \mu_h\right] - \tau_h \psi \lambda_h^{**}(b)}, & I_v^{**} &= \frac{\lambda_v^{**}(b) \gamma_v \pi_v(q)}{k_3 k_4 \left[\lambda_v^{**}(b) + k_4\right]},
\end{aligned}
\tag{18}
$$

The total population N_h^{**} is obtained by summing up the sub-population as given below

$$\begin{aligned} N_h^{**} &= S_h^{**} + I_h^{**} + R_h^{**} \\ &= \frac{\pi_h k_1 k_2 + \lambda_h^{**}(b)\pi_h k_2 + \lambda_h^{**}(b)\tau_h \pi_h}{k_1 k_2 \left[\lambda_h^{**}(b) + \mu_h\right] - \tau_h \psi \lambda_h^{**}(b)} \end{aligned} \tag{19}$$

Hence, perturbing the two force of infection below

$$\lambda_h^{**}(b) = \frac{\beta_{hv}\varepsilon(b) I_v^{**}}{N_h^{**}}, \qquad \lambda_v^{**}(b) = \frac{\beta_{vh}\varepsilon(b) I_h^{**}}{N_h^{**}} \tag{20}$$

The endemic equilibria of the model (11), satisfies the following polynomial

$$a_0 \lambda_h^{**2} + b_0 \lambda_h^{**} + c_0 = 0 \tag{21}$$

where

$$\begin{aligned} a_0 &= P_2 P_3 \\ b_0 &= P_1 P_3 + P_2 k_1 k_2 k_4 - P_4 \beta_{hv}\beta_{vh}\varepsilon^2(b)\gamma_v \pi_v k_2 \\ c_0 &= k_1 k_3 k_4^2 \pi_h \left(1 - \mathscr{R}_0^2\right) \end{aligned} \tag{22}$$

for

$$P_1 = k_1 k_2 k_3 k_4 \pi_h, \quad P_2 = \pi_h k_3 k_4 (k_2 + \tau_h), \quad P_3 = \beta_{vh}\varepsilon(b) k_2 + k_2 k_4 + k_4 \tau_h, \quad P_4 = k_1 k_2 - \psi \tau_h$$

The quadratic equation (21) above can be analyzed for the possibility of multiple endemic equilibria whenever $\mathscr{R}_0 < 1$. It should be noted that the coefficient a_0, of the quadratic equation (21) is always positive; and the constant term c_0, is negative (positive) whenever $\mathscr{R}_0$ is greater (less) than unity. Hence, the following result is established.

Theorem 3.2: *The malaria model* (11) *has*:

(i) *a unique endemic equilibrium if* $c_0 < 0 \Longleftrightarrow \mathscr{R}_0 > 1$;

(ii) *a unique endemic equilibrium if* ($b_0 < 0$ *and* $c_0 = 0$) *or* $b_0^2 - 4a_0c_0 = 0$;

(iii) *two endemic equilibria if* $c_0 > 0$, $b_0 < 0$ *and* $b_0^2 - 4a_0c_0 > 0$;

(iv) *no endemic equlibrium otherwise*.

Thus, it is clear from case (i) of Theorem 3.2 that the malaria model (11) has a unique EEP (of the form ϕ_1) whenever $\mathscr{R}_0 > 1$. Furthermore, case (iii) of Theorem 3.2 indicates the possibility of backward bifurcation, where a LAS DFE co-exists with a LAS endemic equilibrium when the associated reproduction number R_0 is less than unity ($R_0 < 1$). The epidemiological importance of the phenomenon of backward bifurcation is that the requirement of having $R_0 < 1$ is although necessary, but not sufficient for disease elimination. In this case, disease elimination will depend upon the initial sizes of the sub-populations of the model.

To check for the possibility of backward bifurcation in the malaria model (11), the discriminant $b_0^2 - 4a_0c_0$ of quadratic (21), is set to zero and the result solved for the critical value of R_0 (denoted by $R_C < 1$). This result to

$$\mathscr{R}_C = \sqrt{1 - \frac{b_0^2}{4a_0k_1k_2k_4^2\pi_h}} \tag{23}$$

for which the following theorem holds.

Theorem 3.3. *The malaria model* (11) *undergoes a backward bifurcation when Case (iii) of Theorem 3.2 holds such that* $R_C < R_0 < 1$.

6. Numerical Simulations

Parameter	Value Baseline	Range	Reference
π_h	Variable	$[10-800]$ days	Assumed
ψ	0.0005275	$[5.5\times10^{-5}-1.1\times10^{-2}]$/day	[43, 44]
μ_h	$\frac{1}{65\times356}$	$\left[\frac{1}{80\times356}-\frac{1}{58\times356}\right]$/day	[43, 44]
τ_h	0.0092	$[0.0014-0.017]$/day	[1]
δ_h	0.0003454	$[1\times10^{-15}-4.1\times10^{-4}]$/day	[45, 44, 43]
β_{hv}	0.24	$[0.072-0.64]/day$	[45, 44, 43]
β_{vh}	0.321	$[0.027-0.64]/day$	[45, 44, 43]
$\varepsilon(max)$	0.5	$[0.1-1]$	[46, 47]
$\varepsilon(min)$	0.0696	$[0-0.1]$	[46, 47]
b	0.53	$[0-1]$	[46]
η	1	$[0.1-1]$	Assumed
π_v	1.84	$[1-500]$	[1, 45]
γ_v	0.343	$[0.333-1]$	[1, 45]
μ_v	$\frac{1}{18}$	$[\frac{1}{21}-\frac{1}{3}]$/day	[48, 46]
$\mu_{max}(b)$	$\frac{1}{14}$	$[\frac{1}{21}-\frac{1}{14}]$/day	[48, 46]
$\mu_{max}(q)$			Assumed

Table 2: Values and ranges of the parameters of the malarial model (11).

7. Optimal Control Analysis

This section seeks to explore the application of Pontryagin's Maximum Principle (PMP) to determine the necessary conditions for the optimal control of the Malaria disease. We endeavor to incorporate time dependent controls into the proposed system (14), in order to determine the best optimal strategy which can be used to control the disease. Thus, we have

$$\frac{dS_h}{dt} = \pi_h - \frac{\beta_{hv}\left(\beta_{\max} - u_1\left(\beta_{\max} - \beta_{\min}\right)\right) I_v S_h}{S_h + I_h + R_h} - \mu_h S_h + \psi R_h$$

$$\frac{dI_h}{dt} = \frac{\beta_{hv}\left(\beta_{\max} - u_1\left(\beta_{\max} - \beta_{\min}\right)\right) I_v S_h}{S_h + I_h + R_h} - \left(\tau_h + \delta_h + \mu_h\right) I_h - \frac{\varepsilon\, u_2 I_h}{1 + \alpha I_h}$$

$$\frac{dR_h}{dt} = \tau_h I_h - \left(\psi + \mu_h\right) R_h$$

(24)

$$\frac{dA_v}{dt} = \pi_{vmax} - u_3\left(\pi_{vmax} - \pi_{vmin}\right) - \left(u_3 \mu_{vmax} + \gamma_v + \mu_v\right) A_v$$

$$\frac{dS_v}{dt} = \gamma_v A_v - \frac{\beta_{vh}\left(\beta_{\max} - u_1\left(\beta_{\max} - \beta_{\min}\right)\right) I_h S_v}{S_h + I_h + R_h} - \left(2\,\mu_v + \left(u_1 + u_3\right)\mu_{vmax}\right) S_v$$

$$\frac{dI_v}{dt} = \frac{\beta_{vh}\left(\beta_{\max} - u_1\left(\beta_{\max} - \beta_{\min}\right)\right) I_h S_v}{S_h + I_h + R_h} - \left(2\,\mu_v + \left(u_1 + u_3\right)\mu_{vmax}\right) I_v$$

We therefore define the control problem as per the above discussion for control policies and costs incurred, as follows:

(25)

$$J_1(u_1(t), u_2(t), u_3(t)) = \int_0^{T_f} \left(w_1 I_h(t) + w_2 A_v(t) + w_3 I_v(t) + w_4 u_1^2(t) + w_5 u_2^2(t) + w_6 u_3^2(t)\right) dt$$

$$\min_{J_1(u_1,u_2,u_3)} (u_1, u_2, u_3 \in U)\; U = \{u_1(t),\; u_2(t),\; \&\; u_3(t) : 0 \le u_1(t) \le 1,\; 0 \le u_2(t) \le 1, 0 \le u_3(t) \le 1\}$$

$t \in [0, T_f]$ and u_1, u_2 and u_3 are Lebesgue measurable subject to the model system (1):

Following the initial conditions

$S_h(0) \ge 0,\; I_h(0) \ge 0,\; R_h(0) \ge 0,\; A_v(0) \ge 0\; S_v(0) \ge 0\; \&\; I_v(0) \ge 0$

The objective function J represents the total cost incurred as a result of the application of control plans and the burden of the disease.

$$
\begin{aligned}
H &= w_1 I_h + w_2 A_v + w_3 I_v + w_4 {u_1}^2 + w_5 {u_2}^2 + w_6 {u_3}^2 \\
&+ \lambda_1 \left(\pi_h - \frac{\beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min} \right) \right) I_v S_h}{S_h + I_h + R_h} - \mu_h S_h + \psi R_h \right) \\
&+ \lambda_2 \left(\frac{\beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min} \right) \right) I_v S_h}{S_h + I_h + R_h} - \left(\tau_h + \delta_h + \mu_h \right) I_h - \frac{\varepsilon u_2 I_h}{1 + \alpha I_h} \right) \\
&+ \lambda_3 \left(\tau_h I_h - \left(\psi + \mu_h \right) R_h \right) + \lambda_4 \left(\pi_{vmax} - u_3 \left(\pi_{vmax} - \pi_{vmin} \right) - \left(u_{3;n-1} \mu_{vmax} + \gamma_v + \mu_v \right) A_v \right) \\
&+ \lambda_5 \left(\gamma_v A_v - \frac{\beta_{vh} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min} \right) \right) I_h S_v}{S_h + I_h + R_h} - \left(2 \mu_v + \left(u_1 + u_3 \right) \mu_{vmax} \right) S_v \right) \\
&+ \lambda_6 \left(\frac{\beta_{vh} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min} \right) \right) I_h S_v}{S_h + I_h + R_h} - \left(2 \mu_v + \left(u_1 + u_3 \right) \mu_{vmax} \right) I_v \right)
\end{aligned}
$$

where $\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6$ are adjoints variable or co-state variables.The system of equations is found by taking the appropriate partial derivatives of the Hailtonian H with respect to the associated state variable by using Pontryagin's Maximum Principle.

Theorem 3.3.*Given that optimal control variables $u_1^*,\ u_2^*, \& u_3^*$ and $S_h^*, I_h^*, R_h^*, A_v^*, S_v^* \& I_v^*$ are corresponding optimal state variables of the control system (14) and (15), there exists an adjoint variable $\lambda = (\lambda_1, \lambda_2, \lambda_3, \lambda_4, \lambda_5, \lambda_6) \in \Re_+^6$ that satisfies the following equations.*

$$
-\frac{d\lambda_i}{dt} = \frac{\partial H}{\partial u_i}, \tag{26}
$$

where $i = S_h, I_h, R_h, A_v, S_v, I_v$ and with transversality conditions

$\lambda_1(T_f) = \lambda_2(T_f) = \lambda_3(T_f) = \lambda_4(T_f) = \lambda_5(T_f) = \lambda_6(T_f) = 0$

The corresponding optimal controls $u_1^*,\ u_2^*$ & u_3^* are given as,

$$
u_1^* = min\left\{ max\left\{ 0, G_1 \right\}, 1 \right\}, \tag{27}
$$

$$
u_2^* = min\left\{ max\left\{ 0, G_2 \right\}, 1 \right\} \tag{28}
$$

and

$$
u_3^* = min\left\{ max\left\{ 0, G_3 \right\}, 1 \right\} \tag{29}
$$

where;

$$G_1 = \frac{1}{2(S_h+I_h+R_h)w_4}\Big(- S_h I_v \lambda_1 \beta_{hv}\beta_{\max} + S_h I_v \lambda_1 \beta_{hv}\beta_{\min} + S_h I_v \lambda_2 \beta_{hv}\beta_{\max} - S_h I_v \lambda_2 \beta_{hv}\beta_{\min}$$

$$- S_v I_h \lambda_5 \beta_{\max}\beta_{vh} + S_v I_h \lambda_5 \beta_{\min}\beta_{vh} + S_v ih \lambda_6 \beta_{\max}\beta_{vh} - S_v I_h \lambda_6 \beta_{\min}\beta_{vh} + R_h S_v \lambda_5 \mu_{vmax} + \tag{30}$$

$$R_h I_v \lambda_6 \mu_{vmax} + S_h S_v \lambda_5 \mu_{vmax} + S_h I_v \lambda_6 \mu_{vmax} + S_v I_h \lambda_5 \mu_{vmax} + I_h I_v \lambda_6 \mu_{vmax}\Big)$$

$$G_2 = \frac{\lambda_2 \varepsilon I_h}{2\left(1+\alpha I_h\right) w_5}$$

$$G_3 = \frac{I_v \lambda_6 \mu_{vmax} + \lambda_4 \pi_{vmax} - \lambda_4 \pi_{vmin}}{2w_6}$$

Proof. Corrollary 4.1 of Fleming and Rishel [49] gives the existence of an optimal control due to the convexity of the integrand of J with respect to u_1, u_2 *and* u_3, a priori boundedness of the state solutions, and the *Lipschitz* property of the state system with respect to the state variables. The differential equations governing the adjoint variables are obtained by differentiation of the Hamiltonian function, evaluated at the optimal control. Then the adjoint equation can be written as

$$\frac{d\lambda_1}{dt} = -\lambda_1 \left(-\frac{\beta_{hv}\left(\beta_{\max} - u_1\left(\beta_{\max}-\beta_{\min}\right)\right) I_v}{S_h+I_h+R_h} + \frac{\beta_{hv}\left(\beta_{\max} - u_1\left(\beta_{\max}-\beta_{\min}\right)\right) I_v S_h}{\left(S_h+I_h+R_h\right)^2} - \mu_h \right)$$

$$-\lambda_2 \left(\frac{\beta_{hv}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_v}{S_h+I_h+R_h} - \frac{\beta_{hv}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_v S_h}{\left(S_h+I_h+R_h\right)^2} \right) - \frac{\lambda 5\, \beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_h S_v}{\left(S_h+I_h+R_h\right)^2}$$

$$+\frac{\lambda_6 \beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_h S_v}{\left(S_h+I_h+R_h\right)^2}$$

(31)

$$\frac{d\lambda_2}{dt} = -w_1 - \frac{\lambda_1 \beta_{hv}\left(\beta_{\max} - u_1\left(\beta_{\max}-\beta_{\min}\right)\right) I_v S_h}{\left(S_h+I_h+R_h\right)^2}$$

$$-\lambda_2 \left(-\frac{\beta_{hv}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_v S_h}{\left(S_h+I_h+R_h\right)^2} - \tau_h - \delta_h - \mu_h - \frac{\varepsilon u_2}{1+\alpha I_h} + \frac{\varepsilon u_2 I_h}{\left(1+\alpha I_h\right)^2} \right)$$

$$-\lambda_3 \tau_h - \lambda_5 \left(-\frac{\beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))S_v}{S_h+I_h+R_h} + \frac{\beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_h S_v}{\left(S_h+I_h+R_h\right)^2} \right)$$

$$-\lambda_6 \left(\frac{\beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))S_v}{S_h+I_h+R_h} - \frac{\beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_h S_v}{\left(S_h+I_h+R_h\right)^2} \right)$$

$$\frac{d\lambda_3}{dt} = -\lambda_1 \left(\frac{\beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min}\right)\right) I_v Sh}{\left(S_h + I_h + R_h\right)^2} + \psi \right) + \frac{\lambda_2 \beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min}\right)\right) I_v S_h}{\left(S_h + I_h + R_h\right)^2}$$
$$-\lambda_3 \left(-\psi - \mu_h\right) - \frac{\lambda_5 \beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_h S_v}{\left(S_h + I_h + R_h\right)^2} + \frac{\lambda_6 \beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_h S_v}{\left(S_h + I_h + R_h\right)^2}$$

$$\frac{d\lambda_4}{dt} = -w_2 - \lambda_4 \left(-\mu_{vmax} u_3 - \gamma_v - \mu_v\right) - \lambda_5 \gamma_v$$

$$\frac{d\lambda_5}{dt} = -\lambda_5 \left(-\frac{\beta_{vh} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min}\right)\right) I_h}{S_h + I_h + R_h} - 2\,\mu_v - \left(u_1 + u_3\right) \mu_{vmax} \right)$$
$$-\frac{\lambda_6 \beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_h}{S_h + I_h + R_h}$$

$$\frac{d\lambda_6}{dt} = -w_3 + \frac{\lambda_1 \beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min}\right)\right) S_h}{S_h + I_h + R_h} - \frac{\lambda_2 \beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min}\right)\right) S_h}{S_h + I_h + R_h}$$
$$-\lambda_6 \left(-2\,\mu_v - \left(u_1 + u_3\right) \mu_{vmax}\right)$$

Solving for u_1^*, u_2^* *and* u_3^* subject to the constraints, the characterization (16 - 19) can be derived and we have

$$-\frac{d\lambda_1}{dt} = \frac{\partial H}{\partial S_h} = -\lambda_1 \left(-\frac{\beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min}\right)\right) I_v}{S_h + I_h + R_h} + \frac{\beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min}\right)\right) I_v S_h}{\left(S_h + I_h + R_h\right)^2} - \mu_h \right)$$

$$-\lambda_2 \left(\frac{\beta_{hv} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_v}{S_h + I_h + R_h} - \frac{\beta_{hv} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_v S_h}{\left(S_h + I_h + R_h\right)^2} \right) - \frac{\lambda 5\, \beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_h S_v}{\left(S_h + I_h + R_h\right)^2}$$

$$+\frac{\lambda_6 \beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_h S_v}{\left(S_h + I_h + R_h\right)^2}$$

$$-\frac{d\lambda_2}{dt} = \frac{\partial H}{\partial I_h} = -w_1 - \frac{\lambda_1 \beta_{hv} \left(\beta_{\max} - u_1 \left(\beta_{\max} - \beta_{\min}\right)\right) I_v S_h}{\left(S_h + I_h + R_h\right)^2}$$
$$-\lambda_2 \left(-\frac{\beta_{hv} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_v S_h}{\left(S_h + I_h + R_h\right)^2} - \tau_h - \delta_h - \mu_h - \frac{\varepsilon\, u_2}{1 + \alpha I_h} + \frac{\varepsilon\, u_2 I_h}{\left(1 + \alpha I_h\right)^2} \right)$$

$$-\lambda_3\, \tau_h - \lambda_5 \left(-\frac{\beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) S_v}{S_h + I_h + R_h} + \frac{\beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_h S_v}{\left(S_h + I_h + R_h\right)^2} \right)$$

$$-\lambda_6 \left(\frac{\beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) S_v}{S_h + I_h + R_h} - \frac{\beta_{vh} (\beta_{\max} - u_1 (\beta_{\max} - \beta_{\min})) I_h S_v}{\left(S_h + I_h + R_h\right)^2} \right)$$

$$-\frac{d\lambda_3}{dt}=\frac{\partial H}{\partial R_h}=-\lambda_1\left(\frac{\beta_{hv}\left(\beta_{\max}-u_1\left(\beta_{\max}-\beta_{\min}\right)\right)I_v Sh}{\left(S_h+I_h+R_h\right)^2}+\psi\right)-\lambda_3\left(-\psi-\mu_h\right)$$
$$+\frac{\lambda_2\beta_{hv}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_v S_h}{(S_h+I_h+R_h)^2}-\frac{\lambda_5\beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_h S_v}{(S_h+I_h+R_h)^2}+\frac{\lambda_6\beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_h S_v}{(S_h+I_h+R_h)^2}$$

$$-\frac{d\lambda_4}{dt}=\frac{\partial H}{\partial A_v}=-w_2-\lambda_4\left(-\mu_{vmax}u_3-\gamma_v-\mu_v\right)-\lambda_5\gamma_v$$

$$-\frac{d\lambda_5}{dt}=\frac{\partial H}{\partial S_v}=-\lambda_5\left(-\frac{\beta_{vh}\left(\beta_{\max}-u_1\left(\beta_{\max}-\beta_{\min}\right)\right)I_h}{S_h+I_h+R_h}-2\mu_v-\left(u_1+u_3\right)\mu_{vmax}\right)$$
$$-\frac{\lambda_6\beta_{vh}(\beta_{\max}-u_1(\beta_{\max}-\beta_{\min}))I_h}{S_h+I_h+R_h}$$

$$-\frac{d\lambda_6}{dt}=\frac{\partial H}{\partial I_v}=-w_3+\frac{\lambda_1\beta_{hv}\left(\beta_{\max}-u_1\left(\beta_{\max}-\beta_{\min}\right)\right)S_h}{S_h+I_h+R_h}-\frac{\lambda_2\beta_{hv}\left(\beta_{\max}-u_1\left(\beta_{\max}-\beta_{\min}\right)\right)S_h}{S_h+I_h+R_h}$$
$$-\lambda_6\left(-2\mu_v-\left(u_1+u_3\right)\mu_{vmax}\right)$$

with transversality conditions

$$\lambda_1(T_f)=\lambda_2(T_f)=\lambda_3(T_f)=\lambda_4(T_f)=\lambda_5(T_f)=\lambda_6(T_f)=0$$

The corresponding optimal controls $u_1^*,\ u_2^*$ & u_3^* are given as

$$\begin{aligned}0=\frac{\partial H}{\partial u_1}=\frac{1}{2(S_h+I_h+R_h)w_4}\Big(&-S_h I_v\lambda_1\beta_{hv}\beta_{\max}+S_h I_v\lambda_1\beta_{hv}\beta_{\min}+S_h I_v\lambda_2\beta_{hv}\beta_{\max}\\&-S_h I_v\lambda_2\beta_{hv}\beta_{\min}-S_v I_h\lambda_5\beta_{\max}\beta_{vh}+S_v I_h\lambda_5\beta_{\min}\beta_{vh}+\\&S_v ih\lambda_6\beta_{\max}\beta_{vh}-S_v I_h\lambda_6\beta_{\min}\beta_{vh}+R_h S_v\lambda_5\mu_{vmax}+R_h I_v\lambda_6\mu_{vmax}\\&+S_h S_v\lambda_5\mu_{vmax}+S_h I_v\lambda_6\mu_{vmax}+S_v I_h\lambda_5\mu_{vmax}+I_h I_v\lambda_6\mu_{vmax}\Big)\end{aligned}$$

(32)

$$0=\frac{\partial H}{\partial u_2}=\frac{\lambda_2\varepsilon I_h}{2\left(1+\alpha I_h\right)w_5}$$

$$0=\frac{\partial H}{\partial u_3}=\frac{I_v\lambda_6\mu_{vmax}+\lambda_4\pi_{vmax}-\lambda_4\pi_{vmin}}{2w_6}$$

Hence, we obtain (21, 22 & 23) by using Lenhart and Workman [50, 51]

$$u_1^*=min\left\{max\left\{0,G_1\right\},1\right\},\tag{33}$$

$$u_2^*=min\left\{max\left\{0,G_2\right\},1\right\}\tag{34}$$

and

$$u_3^* = min\{max\{0, G_3\}, 1\} \tag{35}$$

By standard control arguments involving the bounds on the controls, we conclude that

$$u_1^* = \begin{cases} 0 & if\ G_1 \leq 0, \\ G_1 & if\ 0 < G_1 < 1, \\ 1 & if\ G_1 \geq 1, \end{cases}$$

,

$$u_2^* = \begin{cases} 0 & if\ G_2 \leq 0, \\ G_2 & if\ 0 < G_2 < 1, \\ 1 & if\ G_2 \geq 1, \end{cases}$$

and

$$u_3^* = \begin{cases} 0 & if\ G_3 \leq 0, \\ G_3 & if\ 0 < G_3 < 1, \\ 1 & if\ G_3 \geq 1, \end{cases}$$

where;

$$G_1 = \frac{1}{2(S_h + I_h + R_h)w_4}\Big(- S_h I_v \lambda_1 \beta_{hv}\beta_{\max} + S_h I_v \lambda_1 \beta_{hv}\beta_{\min} + S_h I_v \lambda_2 \beta_{hv}\beta_{\max} -$$
$$S_h I_v \lambda_2 \beta_{hv}\beta_{\min}$$
$$- S_v I_h \lambda_5 \beta_{\max}\beta_{vh} + S_v I_h \lambda_5 \beta_{\min}\beta_{vh} + S_v ih \lambda_6 \beta_{\max}\beta_{vh} - S_v I_h \lambda_6 \beta_{\min}\beta_{vh} + R_h S_v \lambda_5 \mu_{vmax} +$$
$$R_h I_v \lambda_6 \mu_{vmax} + S_h S_v \lambda_5 \mu_{vmax} + S_h I_v \lambda_6 \mu_{vmax} + S_v I_h \lambda_5 \mu_{vmax} + I_h I_v \lambda_6 \mu_{vmax}\Big)$$

$$G_2 = \frac{\lambda_2 \varepsilon I_h}{2(1+\alpha I_h) w_5}$$

$$G_3 = \frac{I_v \lambda_6 \mu_{vmax} + \lambda_4 \pi_{vmax} - \lambda_4 \pi_{vmin}}{2w_6}$$

Hence, we discuss the numerical solutions of the optimality system and the corresponding results of varying the optimal controls u_1, u_2 & u_3 the parameter choices, and the interpretations from various cases.

Hence, we discuss the numerical solutions of the optimality system and the corresponding results of varying the optimal controls u_1, u_2 & u_3 the parameter choices, and the interpretations from various cases.

7.1. Descriptions of Control. The impact of these control on malaria disease progression is investigated. We also optimize the cost incurred, in order to implement the controls. The description of the three controls is done in this section and the corresponding cost incurred is determined.

i Provision of treated pesticide Bed nets to susceptible population. Preventive measure such as provision of pesticide treated bed nets will reduce the susceptibility of susceptible individuals to malaria infection. u_1 represent time preventive control through treated bed net use such that $0 \leq u_1(t) \leq 1$. $u_1 = 0$ represents no use of treated bed nets while $u_1 = 1$ terms the full use of treated bed nets. In order to eliminate the uses of treated bed-net, we include cost of providing and distribution of the nets to individuals. We shall find the optimal response of susceptible individuals via treated bed nets

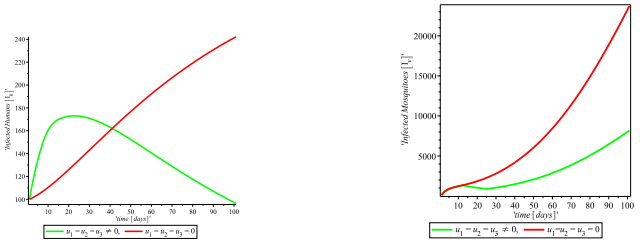
ii Providing treatment to infected population. Treatment of infected individual does not only reduce the disease prevalence but also affects its progression. We assume that the treatment is available to infected individual. We also include treatment as a saturation rate function $\frac{\varepsilon u_2 I_h}{1+\alpha I_h}$, since the availability of medical related resources such as diagnosis, treatment e.t.c can not be unlimited . ε is the treatment rate with intensity u_2 and α is the saturation constant. The treatment intensity u_2 is considered as a control variable such

that $0 \leq u_2(t) \leq 1$. The cost of providing treatment will include diagnosis, medicines, health-care, hospitalized and other related cost.

iii Providing pesticides/insecticides to destroy larva population. As we know, spraying insecticide/ pesticides on larva site which net as breeding ground for mosquitoes will not only reduce the susceptible mosquito populations but also increase the fatality on both adult susceptible mosquitoes and infected mosquitoes. Suppose the pesticides are available for spraying on the larva breeding site, then the intensity of spraying u_3 pesticides on the larva is taken as control such that $0 \leq u_3(t) \leq 1$. The cost of actualizing this control includes purchase of spraying equipments, cost of chemicals, clearing of sites, e.t.c

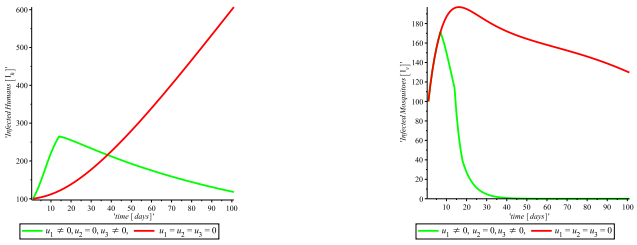
8. Main Results

8.1. Numerical Simulation. In this section, we study the impact of these control on malaria disease progression and also optimize the cost incurred in their implementation numerically. The optimal is obtained by solving the optimality system, consisting of six Ordinary Differential Equation's (ODEs) from the state and adjoint equations. An iterative scheme is used for solving the state equations with a guess for the controls over the simulated time using fourth order Runge-Kutta Scheme. Due to the transversality condition (26), the adjoint equations are solved backward in time using the current iteration solutions of the state equations. Then the controls are updated by using a convex combination of the previous controls and the value from the characterization (27-29). This process continues until the difference between the values of unknowns at the previous iteration and that of the present iteration is negligibly [Lenhart & Workman].



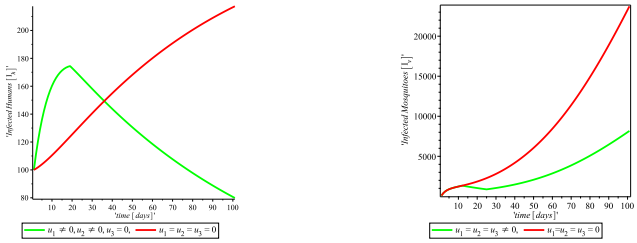
Simulations of the malaria model showing the effect of the optimal strategies of medication and insecticide

8.2. Optimal medication treatment & Insecticide spray. With this strategy, the control (u_2) on medication and the control on (u_3) insecticide spraying are both used to optimize the objective function J, while the control on treated bed-nets (u_1) are set to zero. The results in Figure 1 shows a significant difference in the Infected human (I_h) and Infected mosquitoes (I_v) with optimal control strategy compared to (I_h) and (I_v) without control. We noticed in Figure 1(a) that the control strategies resulted in decrease in the number of symptomatic humans (I_h) as against increases in the uncontrolled case. Similarly, in Figure 1(b), the uncontrolled case resulted increased number of infected mosquitoes (I_v), while the control strategy lead to drastically decrease in the number of infected mosquitoes starting from 20 days and mosquitoes grow resistant after 50 days. The strategy is not effective in controlling the infectious mosquitoes (I_v).



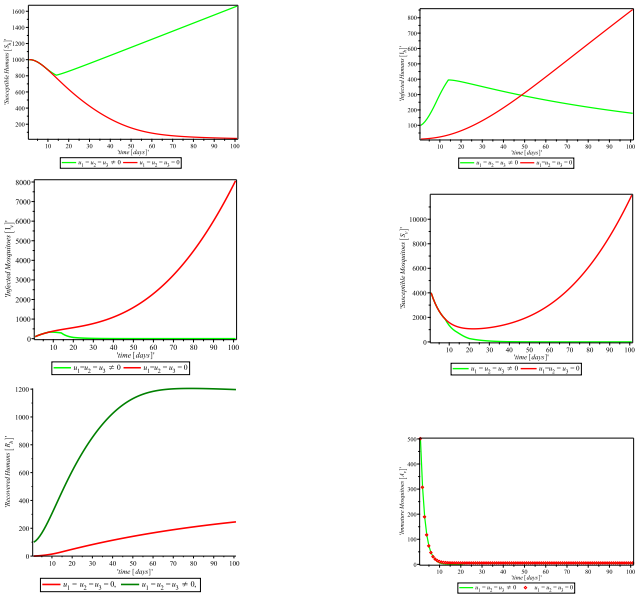
Simulations of the malaria model showing the effect of the optimal strategies of treated bednets and insecticide

8.3. Optimal strategy of treated bed-nets and insecticide spray. Here, the control on treated bed-nets (u_1) and the spray insecticide are used to optimize the objective function J while setting the control on treatment $(u_2) = 0$. For this strategy, shown in Figure 2, we observed that the number of symptomatic human (I_h) and mosquitoes (I_v) differs considerably from the uncontrolled case. Figure 2(a), shows that symptomatic humans (I_h) is reduced in comparison with the case without control. While Figure 2(b), reveals a similar result of decreased number of infected mosquitoes (I_v) for the controlled strategy as compared with the strategy without control.



Simulations of the malaria model showing the effect of the optimal strategies of treated bed-nets and medications

8.4. Optimal treated bed-nets and medication treatment. For this strategy, the control on treated bed-nets (u_1) and the medication (u_2) are used to optimize the objective function J while putting the control on spray of insecticide $(u_3) = 0$. With this strategy, shown in Figure 3, there is a significant difference in the infected human (I_h) and infected mosquitoes (I_v).



Simulations of the malaria model showing the effect of all the control strategies on each of the compartment

8.5. Optimal treated bed-net, treatment medication and spray insecticide. In this strategy, all three controls $(u_1(t), u_2(t)$ & $u_3(t))$ are used to optimize the objective function J. It is obvious in Figure 4(a) that there is a significant difference between the number of infected mosquitoes (I_v) under control the number of infected mosquitoes were initially controlled and after 20 days it was more significant that the control strategy employed is effective in controlling the infected mosquitoes. In Figure 4(b), there is significant difference between the presence of

control and without control cases. In Figure 4(c), there is significant difference between the number of infected humans (I_h) were initially rising but after 15 days it starts reducing. It is shows that the strategy is very effective in controlling (I_h). It was observed that that the number of susceptible mosquitoes (S_v) which was initially increase has been reduced. There is significant difference between Figure 4(d) and Figure 4(e). In Figure 4(f), there is no significant difference between the number of immature mosquitoes (A_v) under the control and without control. The strategy is not effective in controlling the immature mosquitoes (A_v).

8.6. Conclusion. In this paper, we presented a malaria model using system of ordinary differential equations and established that the model is locally asymptotically stable when the associated reproduction number is less than unity. In the optimal control problem considered, we use the combination of two controls at a time, while setting the other to zero, to investigate and compare the effects of the control strategies on malaria progression eradication. The analysis made possible by Pontryagin's Maximum Principle coupled with numerical simulations reveal that the combination of the three control strategies may be adopted in controlling malaria progression disease among the human and mosquito interacting populations . Our numerical results shows that the combination of the three (3) controls, treated bed-nets, medication and insecticides spray, has the highest impact on the control of the disease (malaria). This is followed by the combination of medication and insecticide among the human population; and lastly by the combination involving the use of treated bed-nets and insecticide use. In communities where resources are scarce, we suggest that the combination of treatment and treated bed-nets should be adopted, having observed from the comparison of all three control strategies in Figure 4, that there is significant difference between this strategy and the combination of the all three (3) controls.

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CONFLICT OF INTERESTS

The author(s) declare that there is no conflict of interests.

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