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Article

Mathematical Modelling and Optimal Control of Malaria Using Awareness-Based Interventions

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Abstract: Malaria is a critical fevered illness caused by Plasmodium parasites transmitted among people through the bites of infected female Anopheles mosquitoes. Public awareness about the disease is vital for control of the disease. This article proposes a mathematical model to study the dynamics of malaria disease transmission with the impact of awareness-based control measures. Some basic mathematical properties of the proposed model, such as nonnegativity and boundedness of solutions, the feasibility of the equilibrium points, and their stability properties, have been studied. The proposed model possesses two equilibria, explicitly the disease-free and endemic equilibrium. Disease-free equilibrium is stable globally if basic reproduction number ($\mathcal{R}_0$) is less than unity ($\mathcal{R}_0 < 1$). Finally, optimal control theory is applied to minimize the cost of disease control and solve the optimal control problem by formulating Hamiltonian functional. Numerical simulations have been provided for the confirmation of the analytical results. The optimal profiles of the treatment process, organizing awareness campaigns, and insecticide uses are determined for the cost-effectiveness of Malaria management. It can be concluded that media awareness with optimal control approach is best for cost-effective malaria disease management.

Keywords: Media campaign; Disease awareness; Mathematical model; Basic reproduction number ($\mathcal{R}_0$); Global stability; Optimal control

1. Introduction

Malaria is a mosquito-borne human disease. It binges through the bites of infected female Anopheles mosquitoes. Five parasite species cause Malaria in humans, and two of these species - *P. falciparum* and *P. vivax* - pose the greatest threat. Among the parasites, *P. falciparum* is the deadliest for malaria infection and *P. vivax* is the most dominant malaria parasite in most countries outside of sub-Saharan Africa(WHO). The World Health Organization (WHO), in 2020, reported approximately 241 million malaria cases were seen worldwide, whereas the number of malaria deaths was estimated as 627 000 in 2020 [1,2]. In 2020, Africa was the leading region to face 95% of malaria cases with 96% of malaria deaths. Among the total casualties, 80% were the children under the age of five years in that area [3]. Despite decades of global eradication and control efforts, the disease is re-emerging in areas where control efforts were once effective and emerging in areas thought free of the disease [4].

Media letters have been used to promote insecticide-treated net (ITN)/bed net usage to influence malaria inhibition [5]. The efforts to relate ITN messages to the public are instrumental in increasing the use of mosquito nets and having multiple ways of reaching the public and enhancing their effects [6]. The most meaningful result can be seen when a health worker or a volunteer bears malaria-related news to the people [7,8].

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Mathematical models have played important parts in the growth of the epidemiology of the disease. Mathematical models for malaria transmission dynamics offer a better knowledge of the disease, preparation for the future, and see appropriate control actions. In the past years, several numbers of mathematical models on the transmission dynamics of Malaria have been observed. Following the simple $S-I-R$ malaria model, many researchers have elaborated these models by incorporating different features associated with malaria transmission dynamics and its control [9–17]. These articles did not reflect the bearing of awareness movements for malaria disease control. Awareness movements are substantial in malaria control [18–20]. Misra et al. [21] have proposed a mathematical model projected to measure the impact of making awareness by the media on the blowout of vector-borne diseases. Moreover, the human population was separated into three groups, susceptible, infected, and aware people. A dispersed population $M(t)$, representing the number of media crusades, was measured to square the importance of media campaigns considering a constant disease transmission rate.

To the extent of our familiarity, there needs to be more model-based research on malaria disease dynamics with the influence of awareness campaigns [12,22,23]. In [23], a mathematical model for malaria disease was proposed to avoid the illness by separating the infected population into two groups, unaware and aware infected individuals. Authors further assumed that the growth rate of awareness programs impacting the population is proportional to the unaware infected individuals. Besides the effect of the awareness campaign, the aware infected individuals avoid contact with mosquitoes. Authors in [22] resulting a mathematical model for reviewing the dynamics of malaria disease and the influence of awareness-based interventions for control of the same, that depend on 'level of awareness'. They supposed that the disease spread rates, from vector to human and from human to vector, as declining functions of 'level of awareness'. Moreover, malaria disease transmission charges were implicated as a function of 'level of awareness'. The control measures were supposed to increase awareness of tempted and drenched functions, and the 'level of awareness' was expected as a model population. In [12], authors have anticipated a mathematical model by dividing the susceptible population into two sub-population: aware and unaware human populations. They assumed a constant awareness rate and assumed that a portion of unaware susceptible humans joins aware susceptible aware humans and also pragmatic optimal control theory for vector control and cost of awareness.

In this article, a deterministic mathematical model is proposed to study the dynamics of malaria disease. The impacts of intrusions, such as mosquito nets, spewing insecticides, etc., contingent on the disease's consciousness, are analyzed using the proposed model. Awareness is considered as a secluded model variable that vagaries with time. The susceptible human is divided into the aware and the unaware human classes. Aware people can become unaware, but the rate declines with awareness. Besides, recovery depends on awareness-based treatments. Lastly, three time-dependent control functions are included in the model for the cost of treatment, the cost of insecticides, and the cost of an awareness movement via social media, to reduce the cost of malaria management.

The paper is organized as follows: section 2, a mathematical model for awareness movement in governing malaria disease has been projected. Some preliminary results, namely non-negativity, boundedness, the existence of equilibria points, and characteristic equation of the model, have been provided in Section 3. Stability analysis of equilibria has been carried out, with the possible occurrence of bifurcation, using qualitative theory in sections 4. Optimal control analysis is presented in section 5. In section 6, numerical simulations confirm analytical results. In section 7, A comparison between the present work with the published articles and then the significance of the obtained results is discussed. Finally, a conclusion in section 8 finishes the paper.

2. Mathematical model derivation

In this section, the mathematical model is proposed for malaria transmission dynamics using the following assumptions.

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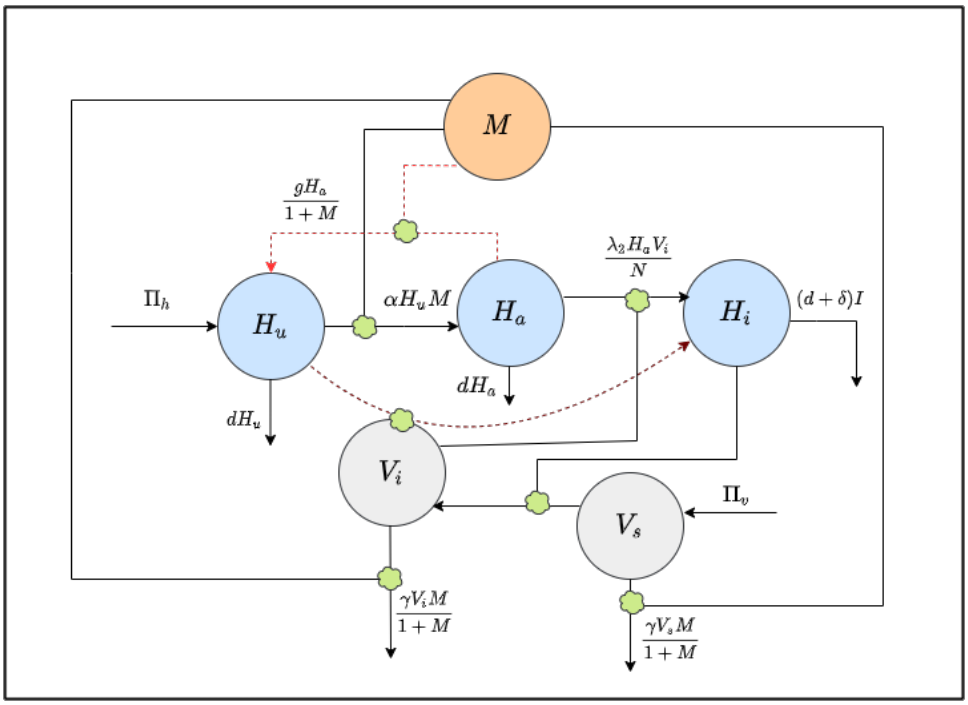

Figure 1. Schematic diagram of the model: interaction between model populations is shown.

The host population is separated into three compartments, unaware (H_u), aware H_a and infected (H_i), with a total population (N) given by $N = H_u + H_a + H_i$. Analogously, the vector population is divided into two compartments, susceptible (V_s) and infected (V_i). All newborns are supposed to be susceptible, and no infected individuals are assumed to come from outside the community. The 'level of awareness', $M(t)$, is considered as a separate population. Figure 1 shows Interactions between the model populations.

An $S - I - S$ type mathematical model is used to capture malaria transmission dynamics in a human population, as immunity to Malaria, is not fully attained and declines with time. Without new contacts, individuals may drop immune memory and become susceptible again. For a mosquito population, an $S - I$ type model is taken, assuming that the mosquito does not mend from malaria parasites and neither does the malaria parasites harm the mosquito population nor recover from infection.

Let Π_h be the constant growth of the human population either by birth or immigration. The whole human population is subject to natural mortality with a constant rate d_h; Π_v is the constant growth rate of the susceptible mosquito population. The force of infections for susceptible humans is (λ_i, $i = 1, 2$), and that of susceptible vectors is (β).

All awareness-induced intrusions affect the spread of Malaria from being aware that this disease is actually upsetting people. It is assumed that by being aware, people will take all essential protections for personal defense and fruitfully outflow the chances of getting infected. Aware people may be unaware at a rate g, but the rate is lessening with the level of awareness $M(t)$ [24]. The model captures this fact with the term $\frac{gH_a}{1+M}$.

In the modeling development, it is assumed that media movements increase the level of awareness concerning personal protection and the way of controlling mosquito population [22].

Here, The level of awareness among people rises at a rate ω from roughly global sources such as radio and TV campaigns, and it declines at a rate θ due to falling(fading) of memory [25].

Knowing the disease and control measures by awareness movement, people will use insecticides to execute mosquitoes at a rate γ, modeled via the term $\gamma M V_s$ and $\gamma M V_i$, where γ is the rate of insecticide usage.

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Moreover, the constants r and δ signify the human population's repossession(recovery) rate and disease-induced death rate. For the mosquito population, μ denotes the natural death rate. The recovery of infected humans will rest-on on the awareness campaign.

With the above assumptions, the following mathematical model is derived:

$$\begin{aligned}
\frac{dH_u}{dt} &= \Pi_h - \alpha H_u M - \frac{\lambda_1 H_u V_i}{N} - d_h H_u + \frac{g H_a}{1+M},\\
\frac{dH_a}{dt} &= \alpha H_u M - d_h H_a + r H_i M - \frac{\lambda_2 H_a V_i}{N} - \frac{g H_a}{1+M},\\
\frac{dH_i}{dt} &= \frac{\lambda_1 H_u V_i}{N} + \frac{\lambda_2 H_a V_i}{N} - r H_i M - (d_h + \delta) H_i,\\
\frac{dV_s}{dt} &= \Pi_v - \frac{\beta H_i V_s}{N} - \mu V_s - \gamma V_s M,\\
\frac{dV_i}{dt} &= \frac{\beta H_i V_s}{N} - \mu V_i - \gamma V_i M,\\
\frac{dM}{dt} &= \omega + \sigma H_i - \theta M.
\end{aligned} \tag{1}$$

Subjected to the initial conditions

$$\begin{aligned}
H_u(0) &= H_{u0} \geq 0, H_a(0) = H_{a0} \geq 0, H_i(0) = H_{i0} \geq 0,\\
V_s(0) &= V_{s0} \geq 0, V_i(0) = V_{i0} \geq 0, M(0) = M_0 \geq 0.
\end{aligned} \tag{2}$$

Table 1. Biological meanings of variables, parameters used in the model (1) and Values of the parameters used for numerical simulations [22,28].

Variables/ Parameters	Descriptions	Values
$H_u(t)$	Number of unaware human	—
$H_a(t)$	Number of aware human	—
$H_i(t)$	Number of infected human	—
$V_s(t)$	Number of susceptible mosquito	—
$V_i(t)$	Number of infective mosquito	—
$M(t)$	Level of awareness on due to media campaign	—
λ_1	Disease transmission from infected mosquito to unaware human	0.02
α	Rate of awareness by media campaign	0.001
λ_2	Disease transmission from infected mosquito to aware humans	0.002
β	Infection rate of vector infected human to susceptible mosquito	0.25
Π_h	Recruitment rate of susceptible human	400
Π_v	Recruitment rate of susceptible mosquito	10000
μ	Natural death rate of mosquito	0.12
r	Recovery rate of infected human due to medication	0.001
d_h	Natural death rate of human	0.002
δ	Disease-induced death rate for human population	0.01
γ	Efficacy of insecticide	0.003
θ	fading of memory	0.01

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3. Basic Properties of the model

3.1. Positivity and boundedness of the solutions

The $H_u, H_a, H_i, V_s, V_i, M$ for the effect of awareness on the transmission dynamics of malaria will be analyzed in a biologically and mathematically viable region as follows. This region should be feasible for both the human population and mosquito populations. Hereafter, the following proposition is established.

Proposition 1. *All solutions of system* (1) *with initial conditions in* (2) *are non-negative for all* $t > 0$.

Proof. Let

$$T_1 = \sup\{t > 0 : H_u(t) > 0, H_a(t) > 0, H_i(t) > 0, V_s(t) > 0, V_i(t) > 0, M(t) > 0\}.$$

Since $H_u(0) > 0, H_a(0) > 0, H_i(0) > 0, V_s(0) > 0, V_i(0) > 0$, and $M(0) > 0$, then $T_1 > 0$. If $T_1 < \infty$, then $H_u, H_a, H_i, V_s, V_i, M$ are all equal to zero at T_1.
It follows from the first equation of the system (1), that

$$\frac{dH_u}{dt} = \Pi_h - \alpha H_u M - \frac{\lambda_1 H_u V_i}{N} - d_h H_u + \frac{g H_a}{1+M}.$$

That is

$$\frac{dH_u}{dt} + \left(d_h + \alpha M + \frac{\lambda_1 V_i}{N}\right) H_u = \Pi_h + \frac{g H_a}{1+M}$$

Thus,

$$\frac{dH_u}{dt}\left\{H_u(t)\exp\left[\left(d_h + \alpha M + \frac{\lambda_1 V_i}{N}\right)t\right]\right\} = \left(\Pi_h + \frac{g H_a}{1+M}\right)\exp\left[\left(d + \alpha M + \frac{\lambda_1 V_i}{N}\right)t\right]$$

Hence,

$$\begin{aligned} &H_u(T_1)\exp\left[\left(d_h + \alpha M + \frac{\lambda_1 V_i}{N}\right)t\right] - H_u(0) \\ &= \int_0^{T_1}\left\{\left(\Pi_h + \frac{g H_a}{1+M}\right)\exp\left[\left(d_h + \alpha M + \frac{\lambda_1 V_i}{N}\right)t\right]\right\}dv \end{aligned}$$

So that,

$$\begin{aligned} H_u(T_1) = H_u(0)&\exp\left[\left(d_h + \alpha M + \frac{\lambda_1 V_i}{N}\right)t\right] \\ &+ \exp\left[\left(d_h + \alpha M + \frac{\lambda_1 V_i}{N}\right)t\right] \\ &\times \int_0^{T_1}\left\{\left(\Pi_h + \frac{g H_a}{1+M}\right)\exp\left[\left(d + \alpha M + \frac{\lambda_1 V_i}{N}\right)t\right]\right\}dv > 0 \end{aligned}$$

From the second equation of system (1), we can write

$$\frac{dH_a}{dt} = \alpha H_u M - d_h H_a + r H_i M - \frac{\lambda_2 H_a V_i}{N} - \frac{g H_a}{1+M}$$

That is

$$\frac{dH_a}{dt} + \left(d_h + \frac{\lambda_2 V_i}{N} + \frac{g}{1+M}\right) H_a = \alpha H_u M + r H_i M$$

Thus,

$$\frac{dH_a}{dt}\left\{H_a(t)\exp\left[\left(d_h + \frac{\lambda_2 V_i}{N} + \frac{g}{1+M}\right)t\right]\right\} = (\alpha H_u M + r H_i M)\exp\left[\left(d_h + \frac{\lambda_2 V_i}{N} + \frac{g}{1+M}\right)t\right]$$

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Hence,

$$H_a(T_1)\exp\left[\left(d_h+\frac{\lambda_2 V_i}{N}+\frac{g}{1+M}\right)t\right]-H_a(0)$$
$$=\int_0^{T_1}(\alpha H_u M+rH_iM)\exp\left[\left(d_h+\frac{\lambda_2 V_i}{N}+\frac{g}{1+M}\right)t\right]dv$$

So that,

$$H_a(T_1)=H_a(0)\exp\left[\left(d_h+\frac{\lambda_2 V_i}{N}+\frac{g}{1+M}\right)t\right]$$
$$+\exp\left[\left(d_h+\frac{\lambda_2 V_i}{N}+\frac{g}{1+M}\right)t\right]$$
$$\times\int_0^{T_1}(\alpha H_u M+rH_iM)\exp\left[\left(d_h+\frac{\lambda_2 V_i}{N}+\frac{g}{1+M}\right)t\right]dv>0$$

Following the same procedure, it can be shown that $H_i>0, V_i>0$ and $M>0$ for all $t>0$. □

Proposition 2. *Every solution of system (1) are uniformly bounded, in the region*

$$\Omega=\Omega_h\cup\Omega_v\cup\Omega_m\subset\mathbb{R}^3_+\times\mathbb{R}^2_+\times\mathbb{R}_+$$

$$\Omega_h=\left\{(H_u,H_a,H_i)\in\mathbb{R}^3_+:0\le H_u+H_a+H_i\le\frac{\Pi_h}{d_h}\right\},$$
$$\Omega_v=\left\{(V_s,V_i)\in\mathbb{R}^2_+:0\le V_s+V_i\le\frac{\Pi_v}{\mu}\right\}$$

and

$$\Omega_m=\left\{M\in\mathbb{R}_+:0\le M\le\frac{\omega\mu+\sigma\Pi_v}{\mu\theta}\right\}$$

Proof. At any time t, $N=H_u+H_a+H_i$, then the time derivative of N along the solution of system (1) is given by

$$\begin{aligned}\frac{dN}{dt}&=\Pi_h-\alpha H_uM-\frac{\lambda_1H_uV_i}{N}-d_hH_u+\frac{gH_a}{1+M}\\&\quad+\alpha H_uM-d_hH_a+rH_iM-\frac{\lambda_2H_aV_i}{N}-\frac{gH_a}{1+M}\\&\quad+\frac{\lambda_1H_uV_i}{N}+\frac{\lambda_2H_aV_i}{N}-rH_iM-(d_h+\delta)H_i\\&=\Pi_h-d_hH_u-d_hH_a-(d_h+\delta)H_i\\&=\Pi_h-d_h(H_u+H_a+H_i)-\delta H_i\\&=\Pi_h-d_hN-\delta H_i\\&\le\Pi_h-d_hN\end{aligned}$$

Then, from the above, we have

$$\frac{dN}{dt}\le\Pi_h-d_hN.$$

That is

$$\frac{dN}{dt}+d_hN\le\Pi_h$$
$$N(t)\le\frac{\Pi_h}{d_h}\left(1-e^{-d_ht}\right)+N_{h0}e^{-d_ht}$$

So that

$$\limsup_{t\to\infty}N(t)\le\frac{\Pi_h}{d_h}$$

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That means

$$0 \leq H_u + H_a + H_i \leq \frac{\Pi_h}{d_h}$$

Similarly any time t, if we let $N_v = V_s + V_i$, then the time derivative of N_v along the solution of system (1) is given by

$$\begin{aligned}
\frac{dN_v}{dt} &= \Pi_v - \frac{\beta H_i V_s}{N} - \mu V_s - \gamma V_s M \\
&\quad + \frac{\beta H_i V_s}{N} - \mu V_i - \gamma V_i M \\
&= \Pi_v - \mu V_s - \mu V_i - \gamma(V_s + V_i)M \\
&= \Pi_v - \mu(V_s + V_i) - \gamma(V_s + V_i)M \\
&= \Pi_v - \mu N_v - \gamma(V_s + V_i)M \\
&\leq \Pi_v - \mu N_v
\end{aligned}$$

Thus the above calculation gives

$$\frac{dN_v}{dt} \leq \Pi_v - \mu N_v.$$

That is

$$\frac{dN_v}{dt} + \mu N_v \leq \Pi_v$$

$$N_v(t) \leq \frac{\Pi_v}{\mu}\left(1 - e^{-\mu t}\right) + N_{v0}e^{-\mu t}$$

So that

$$\limsup_{t\to\infty} N_v(t) \leq \frac{\Pi_v}{\mu}$$

This gives

$$0 \leq V_s + V_i \leq \frac{\Pi_v}{\mu}$$

Finally from the last equation of system (1), one can get

$$\begin{aligned}
\frac{dM}{dt} &= \omega + \sigma V_i - \theta M \\
\frac{dM}{dt} + \theta M &\leq \omega + \sigma V_i \\
\frac{dM}{dt} + \theta M &\leq \omega + \sigma\left(\frac{\Pi_h}{\mu}\right) \\
\frac{dM}{dt} + \theta M &\leq \frac{\omega\mu + \sigma\Pi_v}{\mu}
\end{aligned}$$

On solving this linear differential inequality, we obtain

$$M(t) \leq \frac{\omega\mu + \sigma\Pi_v}{\mu\theta}\left(1 - e^{-\theta t}\right) + M_0 e^{-\theta t}$$

So that

$$\limsup_{t\to\infty} M(t) \leq \frac{\omega\mu + \sigma\Pi_v}{\mu\theta}$$

Hence,

$$0 \leq M(t) \leq \frac{\omega\mu + \sigma\Pi_v}{\mu\theta}$$

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As a result, the region Ω is positively invariant. Therefore, it is adequate to contemplate the dynamics of the movement produced by (1) in Ω. In this region, the model can be well-thought-out to be biologicaly and mathematically well posed. Hence, all solutions of the model (1) with initial conditions in Ω remain in Ω for all $t > 0$.

□

4. The disease-free equilibrium point and its stability

The model system (1) has a Disease-Free Equilibrium attained by vanishing right-hand sides of the equations in the model and solving at $H_i = V_i = 0$. Thus, the disease-free equilibrium is specified by

$$E_0 = (H_u^0, H_a^0, H_i^0, V_s^0, V_i^0, M^0)$$
$$= \left(\frac{\theta\,\Pi_h(g\theta + \omega\,d_h + \theta\,d_h)}{d_h(\alpha\,\omega^2 + \theta\,(\alpha + d_h)\omega + (g + d_h)\theta^2)}, \frac{\Pi_h\alpha\,\omega\,(\theta + \omega)}{d_h(\alpha\,\omega^2 + \theta\,(\alpha + d_h)\omega + (g + d_h)\theta^2)}, 0, \frac{\Pi_v\theta}{\gamma\,\omega + \mu\,\theta}, 0, \frac{\omega}{\theta} \right)$$

4.1. The basic reproduction Number

Basic reproduction number in general denoted by R_0 and is often considered as the threshold quantity that determines the dynamic behavior of the model [26].

The method as used by Heffernan et al. in [27] has been followed for determining the basic reproduction number $\mathcal{R}_0$.

Here, the next generation matrix is denoted by **G**. It comprises two matrices, namely F and V, where

$$F = \begin{pmatrix} 0 & \frac{\lambda_1 k_1 + \lambda_2 k_2}{k_3 N_0} \\ \frac{\beta\Pi_v\theta}{(r\omega+\mu\theta)N_0} & 0 \end{pmatrix}, V = \begin{pmatrix} \frac{r\omega}{\theta} + d_h + \sigma & 0 \\ 0 & \mu - \frac{r\omega}{\theta} \end{pmatrix}$$

It follows that, the reproduction number is given by $\mathcal{R}_0 = \rho(FV^{-1})$, where ρ is the dominant eigenvalue of the matrix $\mathbf{G} = FV^{-1}$.

Hence,

$$\mathcal{R}_0 = \frac{\beta\theta^3(\lambda_1 k_1 + \lambda_2 k_2)\Pi_v}{(\mu\theta - r\omega)(r\omega + \mu\theta)(r\omega + \theta d_h + \sigma\theta)k_3 N_0^2}$$

where, $k_1 = \theta\,\Pi_h(g\theta + \omega\,d_h + \theta\,d_h), k_2 = \Pi_h\alpha\,\omega\,(\theta + \omega), k_3 = d_h\left(\alpha\,\omega^2 + \theta\,(\alpha + d_h)\omega + (g + d_h)\theta^2\right)$ and $N_0 = H_u^0 + H_i^0$.

4.2. Local Stability Analysis of the disease free equilibrium point (DFE)

The following theorem analyses the local stability of DFE.

Theorem 1. *The DFE of the model equation (1), given by E_0, is locally asymptotically stable (LAS) if $R_0 < 1$, and unstable if $R_0 > 1$.*

Proof. At the disease free equilibrium point E_0, the Jacobian matrix $J(E_0)$ is

$$\begin{bmatrix} -\frac{\alpha\,\omega}{\theta} + d_h & -\frac{\theta\,g}{\alpha\,\omega+\theta} & 0 & 0 & \frac{\lambda_1 k_1}{k_3 N_0} & \frac{\alpha\,k_1}{k_3 N_0} + \frac{\theta^2 g k_2}{k_3(\alpha\,\omega+\theta)^2} \\ -\frac{\alpha^2\omega}{\theta} & -d_h - \frac{\theta\,g}{\alpha\,\omega+\theta} & -\frac{r\alpha\,\omega}{\theta} & 0 & \frac{\lambda_2 k_2}{k_3 N_0} & -\frac{\alpha\,k_1}{k_3 N_0} - \frac{\theta^2 g k_2}{k_3(\alpha\,\omega+\theta)^2} \\ 0 & 0 & -\frac{r\alpha\,\omega}{\theta} - \delta - d_h & 0 & -\frac{\lambda_1 k_1}{k_3 N_0} - \frac{\lambda_2 k_2}{k_3 N_0} & 0 \\ 0 & 0 & \frac{\beta\,\Pi_v\theta}{(\gamma\,\omega+\mu\,\theta)N_0} & -\mu - \frac{\gamma\,\alpha\,\omega}{\theta} & 0 & \frac{\gamma\,\beta\,\Pi_v\theta}{(\gamma\,\omega+\mu\,\theta)N_0} \\ 0 & 0 & -\frac{\beta\,\Pi_v\theta}{(\gamma\,\omega+\mu\,\theta)N_0} & 0 & -\mu - \frac{\gamma\,\alpha\,\omega}{\theta} & 0 \\ 0 & 0 & 0 & 0 & -\sigma & -\theta \end{bmatrix}.$$

The characteristic polynomial to $J(E_0)$ in x is,

$$F(x) = (x + \mu + \frac{\gamma\alpha\omega}{\theta})(x + \theta)(x^2 + l_1 x + l_2)(x^2 + m_1 x + m_2) = 0, \tag{3}$$

The coefficients of (3) are given in Appendix I.

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Two eigenvalues, $-(\mu + \frac{\gamma\alpha\omega}{\theta})$ and $-\theta$, are negative. Since, l_1 and m_1 are positive. Thus, according to Routh-Hurwitz criteria, rest of the eigenvalues are negative if $l_2 > 0$ and $m_2 > 0$. This conditions are satisfied when $\mathcal{R}_0 < 1$.

Therefore, $\mathcal{R}_0 < 1$. Hence the disease free equilibrium of the Malaria Model (1) is locally Asymptotically stable. □

Remark 1. *From epidemological point of view, malaria can be eliminated from the community when $R_0 < 1$. If $R_0 < 1$ then, average of an infected individual produce less than one new infected individual over the period of its infectious period and the infection dies out. But if $R_0 > 1$, then each infected individual produce an average of more than one infection and the disease persist and invade the population.*

4.3. Global Stability of the Disease–Free Equilibrium Point

Theorem 2. *The disease-free equilibrium, E_0 is globally asymptotically stable if $\mathcal{R}_0 \leq 1$.*

Proof. The Lyapunov function is defined as

$$\begin{aligned} L(t) =& \frac{1}{2}(H_u - \bar{H_u})^2 + \frac{1}{2}(H_a - \bar{H_a})^2 + \frac{1}{2}(H_i - \bar{H_i})^2 \\ &+ \frac{1}{2}(V_s - \bar{V_s})^2 + \frac{1}{2}(V_i - \bar{V_i})^2 + \frac{1}{2}(M - \bar{M})^2. \end{aligned}$$

Differentiating the Lyapunov function, L with respect to t, we get

$$\begin{aligned} \frac{dL}{dt} =& \Pi_h(H_u - \bar{H_u}) + \left(\alpha(M - \bar{M}) + \frac{\lambda_1}{N_0}(V_i - \bar{V_i}) + d_h\right)\bar{H_u}(H_u - \bar{H_u}) \\ &+ \frac{g}{1 + M - \bar{M}}(H_a - \bar{H_a})(H_u - \bar{H_u}) \\ &+ (\alpha(H_u - \bar{H_u}) + r(H_i - \bar{H_i})(M - \bar{M}))(H_a - \bar{H_a}) + \left(d_h + \frac{\lambda_2(V_i - \bar{V_i})}{N_0} + \frac{g}{1 + M - \bar{M}}\right)\bar{H_a}(H_a - \bar{H_a}) \\ &+ \left(\frac{\lambda_1(H_u - \bar{H_u}) + \lambda_2(H_a - \bar{H_a})}{N_0}\right)(V_i - \bar{V_i})(H_i - \bar{H_i}) + (r(M - \bar{M}) + d_h + \delta)\bar{H_i}(H_i - \bar{H_i}) \\ &+ \Pi_v(V_s - \bar{V_s}) + \left(\frac{\beta(H_i - \bar{H_i})}{N_0} + \mu + \gamma(M - \bar{M})\right)\bar{V_s}(V_s - \bar{V_s}) \\ &+ \frac{\beta(H_i - \bar{H_i})(V_s - \bar{V_s})}{N_0}(V_i - \bar{V_i}) + (\mu + \gamma(M - \bar{M}))\bar{V_i}(V_i - \bar{V_i}) \\ &+ \omega(M - \bar{M}) + \sigma(H_i - \bar{H_i})(M - \bar{M}) + \theta\bar{M}(M - \bar{M}) \\ &- \left(\alpha(M - \bar{M}) + \frac{\lambda_1}{N_0}(V_i - \bar{V_i}) + d_h\right)H_u(H_u - \bar{H_u}) \\ &- \left(d_h + \frac{\lambda_2(V_i - \bar{V_i})}{N_0} + \frac{g}{1 + M - \bar{M}}\right)H_a(H_a - \bar{H_a}) \\ &- (r(M - \bar{M}) + d_h + \delta)H_i(H_i - \bar{H_i}) \\ &- \left(\frac{\beta(H_i - \bar{H_i})}{N_0} + \mu + \gamma(M - \bar{M})\right)V_s(V_s - \bar{V_s}) \\ &- (\mu + \gamma(M - \bar{M}))V_i(V_i - \bar{V_i}) - \theta M(M - \bar{M}) \end{aligned}$$

Then by collecting positive and negative terms together, we obtain

$$\frac{dL}{dt} = A - B$$

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where,

$$\begin{aligned}A =&\Pi_h(H_u-\bar{H_u})+\left(\alpha(M-\bar{M})+\frac{\lambda_1}{N_0}(V_i-\bar{V_i})+d_h\right)\bar{H_u}(H_u-\bar{H_u})\\&+\frac{g}{1+M-\bar{M}}(H_a-\bar{H_a})(H_u-\bar{H_u})\\&+(\alpha(H_u-\bar{H_u})+r(H_i-\bar{H_i})(M-\bar{M}))(H_a-\bar{H_a})+\left(d_h+\frac{\lambda_2(V_i-\bar{V_i})}{N_0}+\frac{g}{1+M-\bar{M}}\right)\bar{H_a}(H_a-\bar{H_a})\\&+\left(\frac{\lambda_1(H_u-\bar{H_u})+\lambda_2(H_a-\bar{H_a})}{N_0}\right)(V_i-\bar{V_i})(H_i-\bar{H_i})+(r(M-\bar{M})+d_h+\delta)\bar{H_i}(H_i-\bar{H_i})\\&+\Pi_v(V_s-\bar{V_s})+\left(\frac{\beta(H_i-\bar{H_i})}{N_0}+\mu+\gamma(M-\bar{M})\right)\bar{V_s}(V_s-\bar{V_s})\\&+\frac{\beta(H_i-\bar{H_i})(V_s-\bar{V_s})}{N_0}(V_i-\bar{V_i})+(\mu+\gamma(M-\bar{M}))\bar{V_i}(V_i-\bar{V_i})\end{aligned}$$

and

$$\begin{aligned}B=&\left(\alpha(M-\bar{M})+\frac{\lambda_1}{N_0}(V_i-\bar{V_i})+d_h\right)H_u(H_u-\bar{H_u})\\&+\left(d_h+\frac{\lambda_2(V_i-\bar{V_i})}{N_0}+\frac{g}{1+M-\bar{M}}\right)H_a(H_a-\bar{H_a})\\&+(r(M-\bar{M})+d_h+\delta)H_i(H_i-\bar{H_i})\\&+\left(\frac{\beta(H_i-\bar{H_i})}{N_0}+\mu+\gamma(M-\bar{M})\right)V_s(V_s-\bar{V_s})\\&+(\mu+\gamma(M-\bar{M}))V_i(V_i-\bar{V_i})+\theta M(M-\bar{M})\end{aligned}$$

We observe that $A-B\leq 0$ if and only if $H_u>\bar{H_u}, H_a>\bar{H_a}, H_i>\bar{H_i}, V_s>\bar{V_s}, V_i>\bar{V_i}, M>\bar{M}$ where $\bar{H_u},\bar{H_a},\bar{H_i},\bar{V_s},\bar{V_i},\bar{M}$ are the disease free equilibrium points. Also, $\frac{dL}{dt}=0$ only at the disease free equilibrium point E_0. Thus by Lasalle's principle, E_0 is globally asymptotically stable if $A<B$. □

4.4. Existence of endemic equilibrium point (EEP)

Here, we describe the conceivable equilibria of the model and found the situations for the existence of an equilibrium for which malaria is endemic in the population. The infection equilibria of the malaria model (1) represented as $E_1=(H_u^*,H_a^*,H_i^*,V_s^*,V_i^*,M^*)$ is obtained by equating the right-hand side of the equations in (1) to zero and solve it simultaneously. Therefore, the endemic equilibria are given as

$$H_u^*=\frac{\Pi_h N\left((d_h+g)N+V_i^*\lambda_2\right)\theta^2+N\left(\sigma H_i^*+\omega\right)\left(NgrH_i^*+\Pi_h Nd_h+\Pi_h V_i^*\lambda_2\right)\theta}{\left(Nd_h+V_i^*\lambda_1\right)\left((d_h+g)N+V_i^*\lambda_2\right)\theta^2+\left((d_h+\alpha)N+V_i^*\lambda_1\right)\left(Nd_h+V_i^*\lambda_2\right)\left(\sigma H_i^*+\omega\right)\theta+\alpha\left(\sigma H_i^*+\omega\right)^2N\left(Nd_h+V_i^*\lambda_2\right)}$$

$$H_a^*=\frac{N\left(\sigma H_i^*+\omega\right)\left(r\left(Nd_h+V_i^*\lambda_1\right)H_i^*+N\alpha\Pi_h\right)\theta+N\left(\sigma H_i^*+\omega\right)\left(r\left((d_h+\alpha)N+V_i^*\lambda_1\right)H_i^*+N\alpha\Pi_h\right)}{\left(Nd_h+V_i^*\lambda_1\right)\left((d_h+g)N+V_i^*\lambda_2\right)\theta^2+\left((d_h+\alpha)N+V_i^*\lambda_1\right)\left(Nd_h+V_{i*}\lambda_2\right)\left(\sigma H_i^*+\omega\right)\theta+\alpha\left(\sigma H_i^*+\omega\right)^2N\left(Nd_h+V_i^*\lambda_2\right)}$$

$$H_i^*=\frac{\lambda_1H_a^*+\lambda_2H_u^*}{(\delta+d_h+rM^*)N}V_i^*$$

$$V_s^*=\frac{\Pi_vN}{\beta H_i^*+N+rM^*},\quad V_i^*=\frac{\beta H_i^*V_s^*}{\mu+rM^*},\quad M^*=\frac{\omega+\sigma H_i^*}{\theta}.$$

The epidemic equilibrium satisfies the following third degree polynomial

$$F(H_i^*)=A(H_i^*)^3+B(H_i^*)^2+C(H_i^*) \tag{4}$$

The coefficients of (4) are given in Appendix J.

The condition $H_i^*=0$ resembles to disease free equilibrium point which we have previously established and $F(H_i^*)=0$ corresponds to a particular condition when the disease continues. In the case of backward bifurcation, several endemic equilibria must exist. This suggests that the equation $F(H_i^*)=0$ specifies that there are three cases we have to consider for $F(H_i^*)=0$ based on the signs of B and C since A is always positive. That is;

1. If $B<0$ and $C=0$ or $B^2-4AC=0$, then the equations $H_i^*=0$ and $F(H_i^*)=0$ has a sole endemic equilibrium point (only one positive root) and no backward bifurcation exists.
2. If $C>0, B>0$ and $B^2-4AC=0$, the equations $H_i^*=0$ and $F(H_i^*)=0$ have two endemic equilibria i.e, two possible roots, and therefore there is possibility of backward bifurcation to occur.

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3. If neither condition 1 nor condition 2 above holds, there does not exist any endemic equilibrium.

It is imperative to note that C is always positive if $\mathcal{R}_0 < 1$ and negative if $\mathcal{R}_0 > 1$. Hence our argument fallouts to the following outcome

Theorem 3. *For the model system (1) we have*

(a) *A unique endemic equilibrium if* $C < 0 \Leftrightarrow \mathcal{R}_0 > 1$
(b) *Exactly one unique endemic equilibrium if* $B < 0$ *and* $C = 0$ *or* $B^2 - 4AC = 0$
(c) *Only two endemic equilibria if* $C > 0, B < 0$ *and* $B^2 - 4AC > 0$
(d) *There does not exist any endemic equilibrium then.*

5. The optimal control problem

In this section, the model system (1) is reformulated by incorporating three time dependent control functions, $C_1(t)$, $C_2(t)$ and $C_3(t)$, with the first control $C_1(t)$ as controlling cost of treatment, the second control $C_2(t)$ as controlling cost of insecticides, and the third control $C_3(t)$ as a control cost of consciousness movement. At this point the goal is to reduce the price of control. That means the goal here is to discover the optimal strictures $C_1^*(t)$, $C_2^*(t)$ and $C_3^*(t)$ using the Pontryagin minimum principle methods assumed in [29].

Therefore, our system (1) is improved to the tempted state nonlinear dynamics given by:

$$\begin{cases} \frac{dH_u}{dt} = \Pi_h - \alpha H_u M - \frac{\lambda_1 H_u V_i}{N} - d_h H_u + \frac{g H_a}{1+M}, \\ \frac{dH_a}{dt} = \alpha H_u M - d_h H_a + C_1 r H_i M - \frac{\lambda_2 H_a V_i}{N} - \frac{g H_a}{1+M}, \\ \frac{dH_i}{dt} = \frac{\lambda_1 H_u V_i}{N} - (d_h + \delta) H_i - C_1 r H_i M + \frac{\lambda_2 H_a V_i}{N}, \\ \frac{dV_s}{dt} = \Pi_v - \frac{\beta H_i V_s}{N} - \mu V_s - C_2 \gamma V_s M, \\ \frac{dV_i}{dt} = \frac{\beta H_i V_s}{N} - \mu V_i - C_2 \gamma V_i M, \\ \frac{dM}{dt} = C_3 \omega + \sigma H_i - \theta M. \end{cases} \tag{5}$$

with the initial conditions

$$H_u(0) = H_{u0}, H_a(0) = H_{a0}, H_i(0) = H_{i0}, V_s(0) = V_{s0}, V_i(0) = V_{i0} \quad \text{and} \quad M(0) = M_0$$

The cost function for the minimization problem is proposed as

$$J(C_1(t), C_2(t), C_3(t)) = \int_0^{t_f} [A_1 C_1(t)^2 + A_2 C_2(t)^2 + A_3 C_3(t)^2 + P_1 H_i - P_2 H_a^2] dt$$

Where the quantities A_1, A_2 and A_3 are the positive weight constants on the advantage of the cost, while the terms P_1 and P_2 are the penalty multipliers.

A quadratic cost functional on the controls is assumed as an approximation for nonlinear function depending on the assumption that the cost take nonlinear form and also to prevent the bang bang or singular optimal control cases[30]. The control set is defined on $[t_0, t_f]$ subject to the conditions $0 < C_i(t) < 1, i = 1, 2, 3$, where t_0 and t_f are initial and final time of giving control, respectively. The intention here is to find the optimal profile of $C_1(t)$, $C_2(t)$ and $C_3(t)$, denoted respectively as $C_i{}^*(t), i = 1, 2, 3$, so that $J(C_1, C_2, C_3)$ is smallest, that means,

$$J(C_1^*(t), C_2^*(t), C_3^*(t)) = \min(J(C_1(t), C_2(t), C_3(t)) : (C_1, C_2, C_3) \in \mathcal{U}), \tag{6}$$

subject to the state system (5), where,

$$\mathcal{U} = \Big\{ u = (C_1, C_2, C_3) / 0 \le C_{1\min} \le C_1(t) \le C_{1\max} \le 1, 0 \le C_{2\min} \le C_2(t) \le C_{2\max} \le 1, \\ 0 \le C_{3\min} \le C_3(t) \le C_{3\max} \le 1, t \in [0, t_f] \Big\} \tag{7}$$

is an admissible control set.

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5.1. Existence of the optimal control triple

Theorem 4. *Given the objective functional*

$$J(C_1(t), C_2(t), C_3(t)) = \int_0^{t_f} \left[A_1 {C_1}^2 + A_2 {C_2}^2 + A_3 {C_3}^2 + P_1 H_i - P_2 {H_a}^2 \right] dt$$

where

$$\mathcal{U} = \{(C_1(t), C_2(t), C_3)(t) : C_i(t) \text{ is Lebesgue measurable and } \quad 0 \leq C_i(t) \leq 1, \quad t \in [0, t_f]\}$$

subject to the system (5) with the initial their conditions. Then there exists an optimal control triple $({C_1}^*, C_2^*, C_3^*)$ *and corresponding state solution* ${H_u}^*, {H_a}^*, {H_i}^*, {V_s}^*, {V_i}^*, M^*$ *such that* $J(C_1^*(t), C_2^*(t), C_3^*(t)) = \min_{\mathcal{U}} J(C_1(t), C_2(t), C_3(t))$ *if the following conditions are met*

(i) *The set of solutions to the system (5) with control variables in (7) are non-empty.*
(ii) *The control set* $\mathcal{U}$ *is convex and closed*
(iii) *Each right hand side of the state system(5) is continuous, is bounded above by a sum of the bounded control and the state, and can be written as a linear function of u with coefficients depending on time and the state.*
(iv) *The integrand function of the objective functional is convex on* $\mathcal{U}$
(v) *There exist positive numbers* $\ell_1, \ell_2, \ell_3, \ell_4$ *and a constant* $\ell > 1$ *such that*

$$J(C_1(t), C_2(t), C_3(t)) \geq -\ell_1 + \ell_2 |C_1|^{\ell} + \ell_3 |C_2|^{\ell} + \ell_4 |C_3|^{\ell}$$

Proof. A detail proof of this Theorem can be obtained in [31] or [30]. □

5.2. Characterization of the optimal control

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

$$\begin{aligned}
\mathcal{H} =& A_1 {C_1}^2 + A_2 {C_2}^2 + A_3 {C_3}^2 + P_1 H_i - P_2 {H_a}^2 \\
&+ \xi_1 \left(\Pi_h - \alpha H_u M - \frac{\lambda_1 H_u V_i}{N} - d_h H_u + \frac{g H_a}{1+M} \right) \\
&+ \xi_2 \left(\alpha H_u M - d_h H_a + C_1 r H_i M - \frac{\lambda_2 H_a V_i}{N} - \frac{g H_a}{1+M} \right) \\
&+ \xi_3 \left(\frac{\lambda_1 H_u V_i}{N} - (d_h + \delta) H_i - C_1 r H_i M + \frac{\lambda_2 H_a V_i}{N} \right) \\
&+ \xi_4 \left(\Pi_v - \frac{\beta H_i V_s}{N} - \mu V_s - C_2 \gamma V_s M \right) \\
&+ \xi_5 \left(\frac{\beta H_i V_s}{N} - \mu V_i - C_2 \gamma V_i M \right) \\
&+ \xi_6 (C_3 \omega + \sigma H_i - \theta M)
\end{aligned}$$

where $\xi_1, \xi_2, \xi_3, \xi_4, \xi_5, \xi_6$ are adjoints variable or co–state variables. The system of equations is obtained by considering the partial derivatives of the Hamiltonian $\mathcal{H}$ with respect to the linked state variable by using Pontryagin's Maximum Principle.

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Theorem 5. *Given the optimal controls* $(C_1(t)^*, C_2(t)^*, C_3(t)^*)$ *and the solutions* $H_u{}^*, H_a{}^*, H_i{}^*, V_s{}^*, V_i{}^*, M^*$ *of the corresponding state system* (5)*, then there exist adjoint variables* $\xi_1, \xi_2, \xi_3, \xi_4, \xi_5, \xi_6$ *satisfying the following system of equations*

$$\begin{cases} \frac{d\xi_1}{dt} = \left(\alpha M + \frac{\lambda_1 V_i}{N} + d_h\right)\xi_1 - \alpha M\xi_2 - \frac{\lambda_1 V_i}{N}\xi_3, \\ \frac{d\xi_2}{dt} = -2\,P_2\,H_a - \frac{g}{1+M}\xi_1 + \left(d_h + \frac{\lambda_2 V_i}{N} + \frac{g}{1+M}\right)\xi_2 - \frac{\lambda_2 V_i}{N}\xi_3, \\ \frac{d\xi_3}{dt} = -P_1 - C_1 rM\xi_2 + (C_1 rM + d_h + \delta)\xi_3 + \frac{\beta V_s}{N}\xi_4 - \frac{\beta V_s}{N}\xi_5 - \sigma\xi_6, \\ \frac{d\xi_4}{dt} = \left(\frac{\beta H_i}{N} + \mu + C_2\gamma M\right)\xi_4 - \frac{\beta H_i}{N}\xi_5, \\ \frac{d\xi_5}{dt} = \frac{\lambda_1}{N}H_u\xi_1 + \frac{\lambda_2}{N}H_a\xi_2 - \frac{\lambda_1 H_u + \lambda_2 H_a}{N}\xi_3 + (\mu + C_2\gamma M)\xi_5, \\ \frac{d\xi_6}{dt} = \left(\alpha H_u + \frac{gH_a}{(1+M)^2}\right)\xi_1 - \left(\alpha H_u + C_1 rH_i + \frac{gH_a}{(1+M)^2}\right)\xi_2 + C_1 rH_i\xi_3 + C_2\gamma V_s\xi_4 + C_2\gamma V_i\xi_5 + \theta\xi_6, \end{cases} \tag{8}$$

with transversality conditions

$$\xi_1(t_f) = \xi_2(t_f) = \xi_3(t_f) = \xi_4(t_f) = \xi_5(t_f) = \xi_6(t_f) = 0 \tag{9}$$

Furthermore for $t \in [0, t_f]$*, the optimal controls* C_1^*, C_2^* *and* C_3^* *are characterized by*

$$\begin{aligned} C_1{}^* &= \max\left\{0, \min\left\{1, \frac{(\xi_2 - \xi_3)rH_iM}{2A_1}\right\}\right\}, \\ C_2{}^* &= \max\left\{0, \min\left\{1, \frac{(V_s\xi_4 - V_i\xi_5)\gamma M}{2A_2}\right\}\right\}, \\ C_3{}^* &= \max\left\{0, \min\left\{1, -\frac{\omega\xi_6}{2A_3}\right\}\right\}. \end{aligned} \tag{10}$$

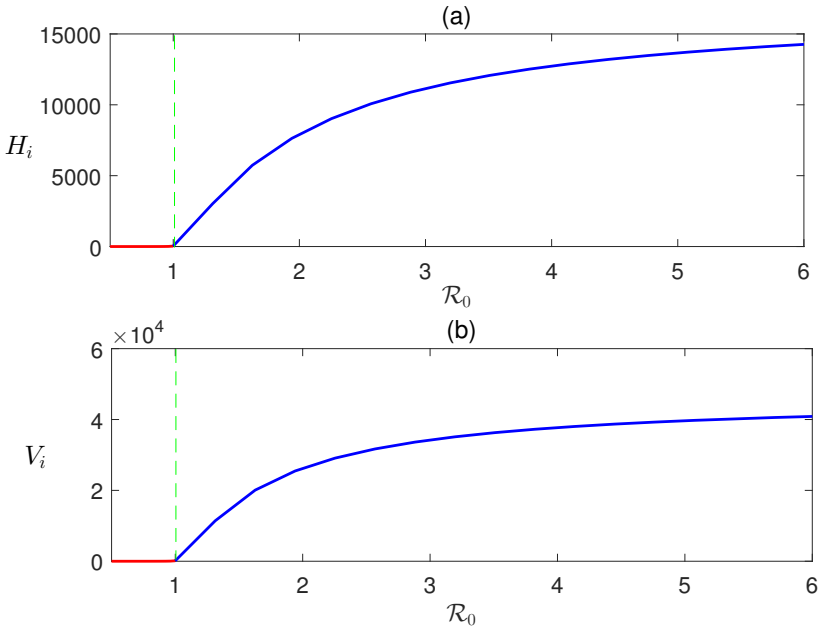

Figure 2. Forward transcritical bifurcation: equilibrium values of infected human and infective vectors are plotted with respect to the basic reproduction number $\mathcal{R}_0$. The parameter β is varied and rest of the parameters' values are taken from Table 1.

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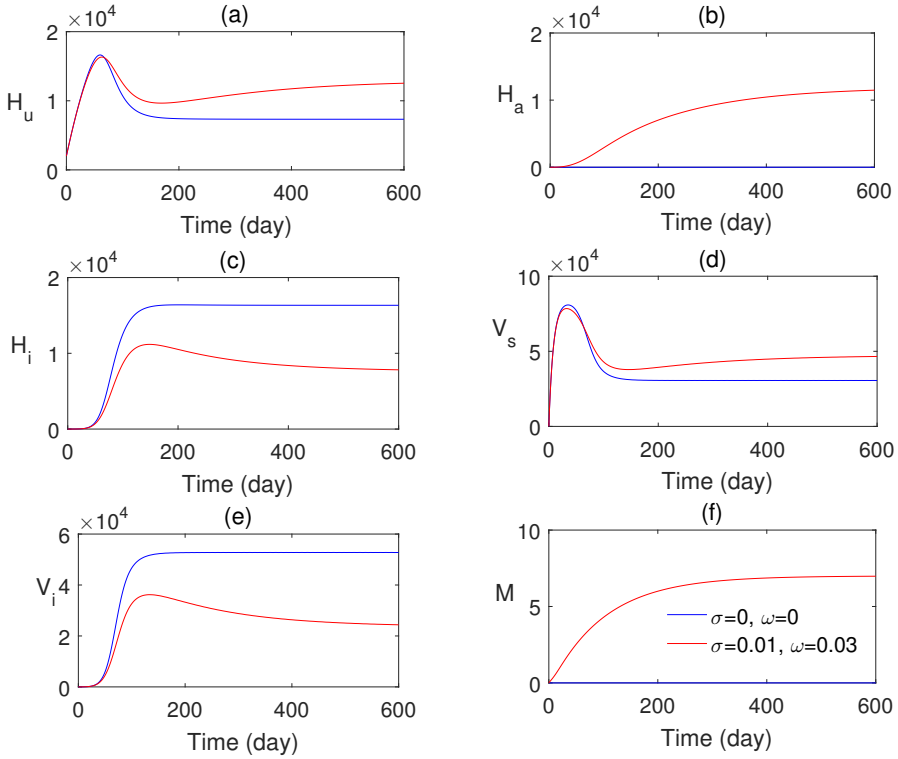

Figure 3. Numerical solution of the system 1 with and without the impact of awareness.

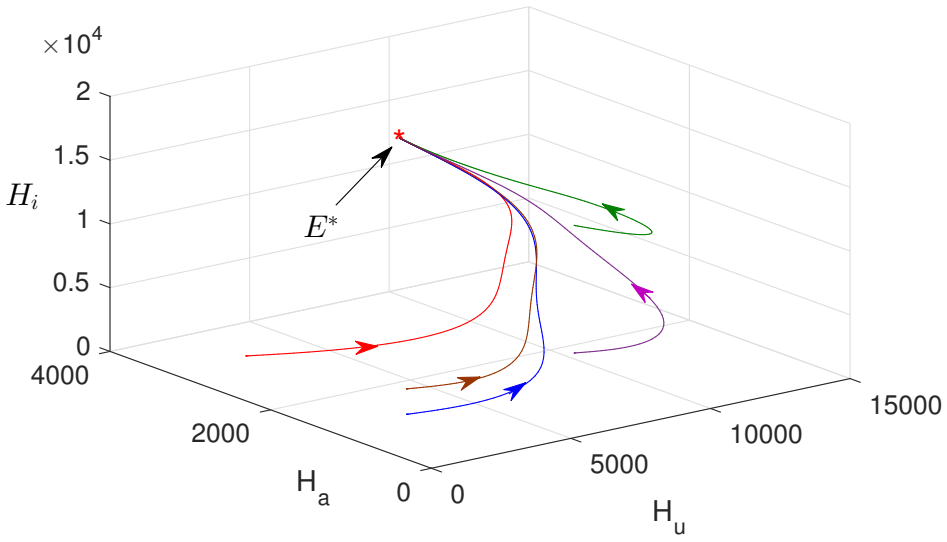

Figure 4. Phase portrait is plotted in $H_u - H_a - H_i$ phase space. Parameters values are same as in Figure 3.

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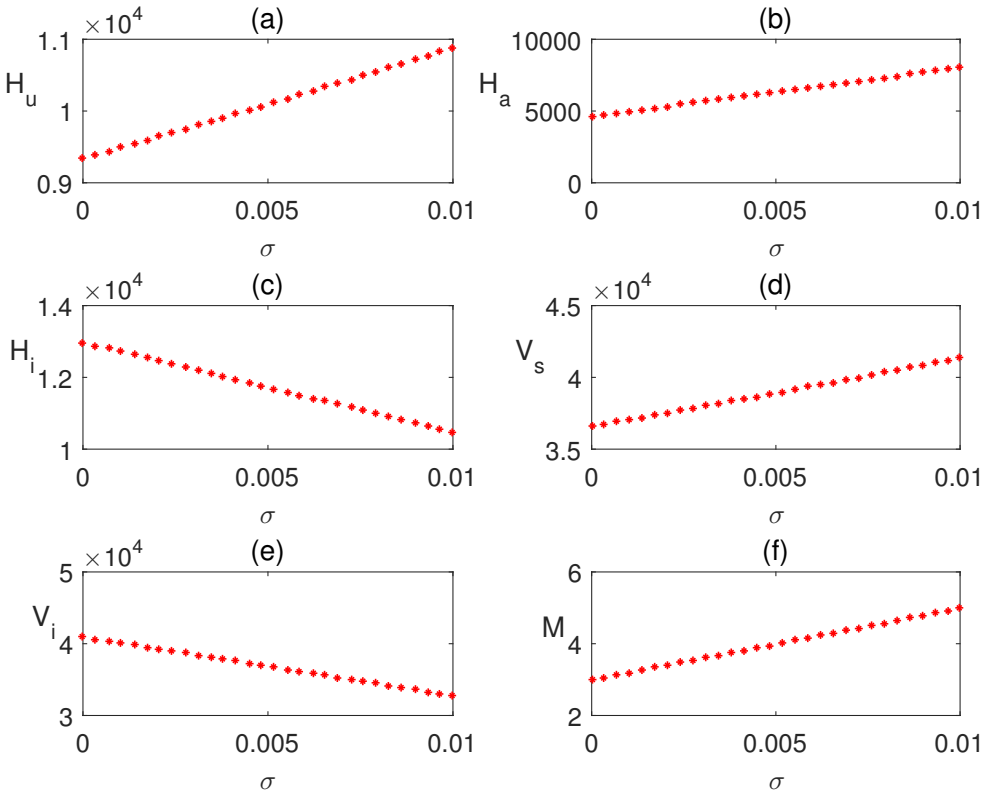

Figure 5. Effect of local awareness is shown varying the parameter σ.

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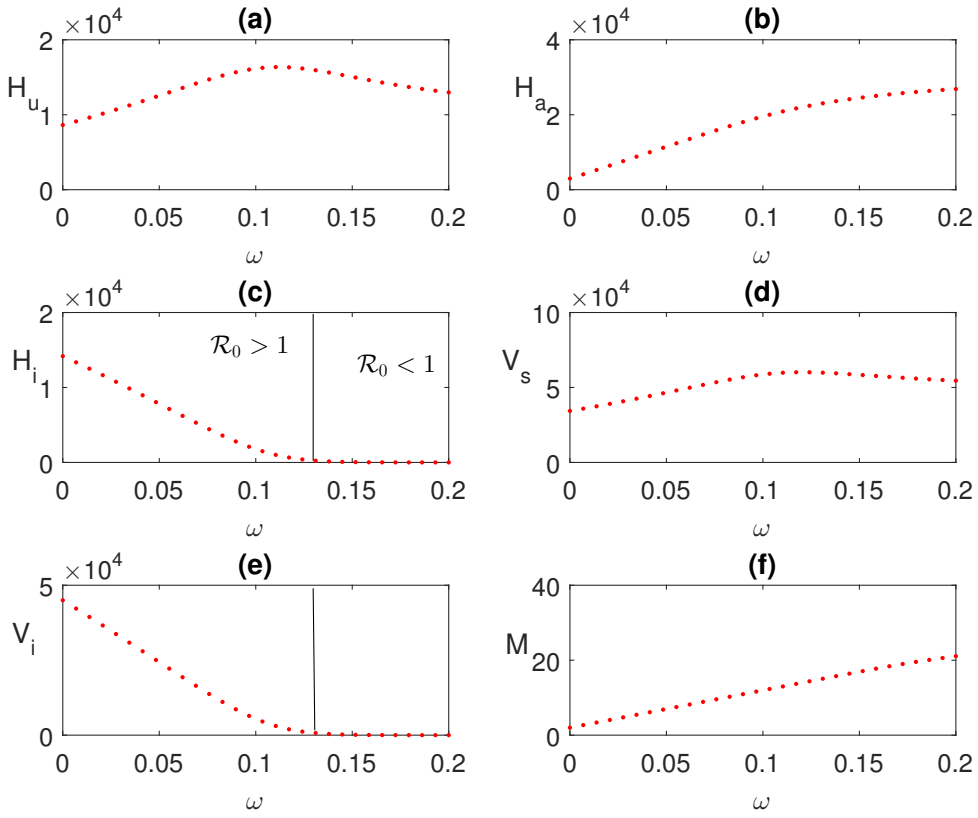

Figure 6. Effect of global awareness is shown varying the parameter ω. Other parameters values are as taken in figure 3.

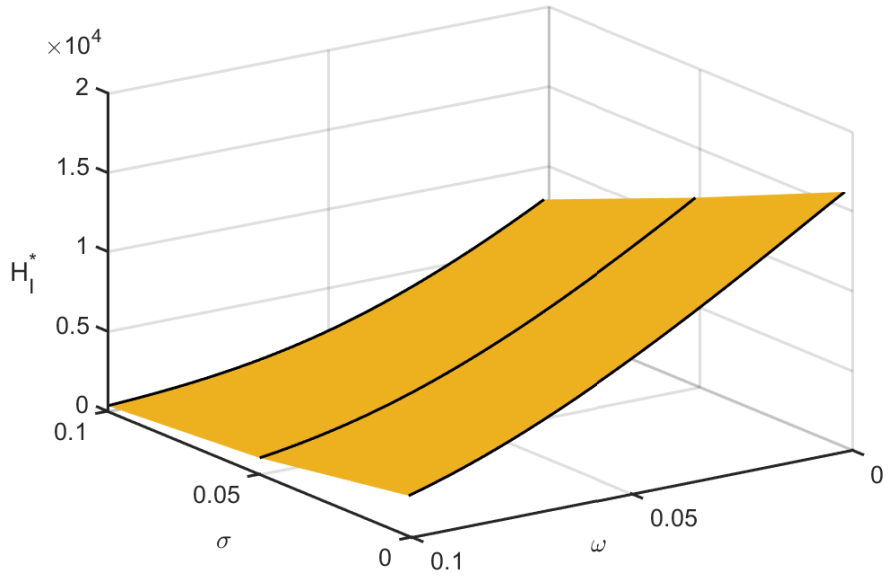

Figure 7. Combined effects of local and global awareness on infected population is shown.

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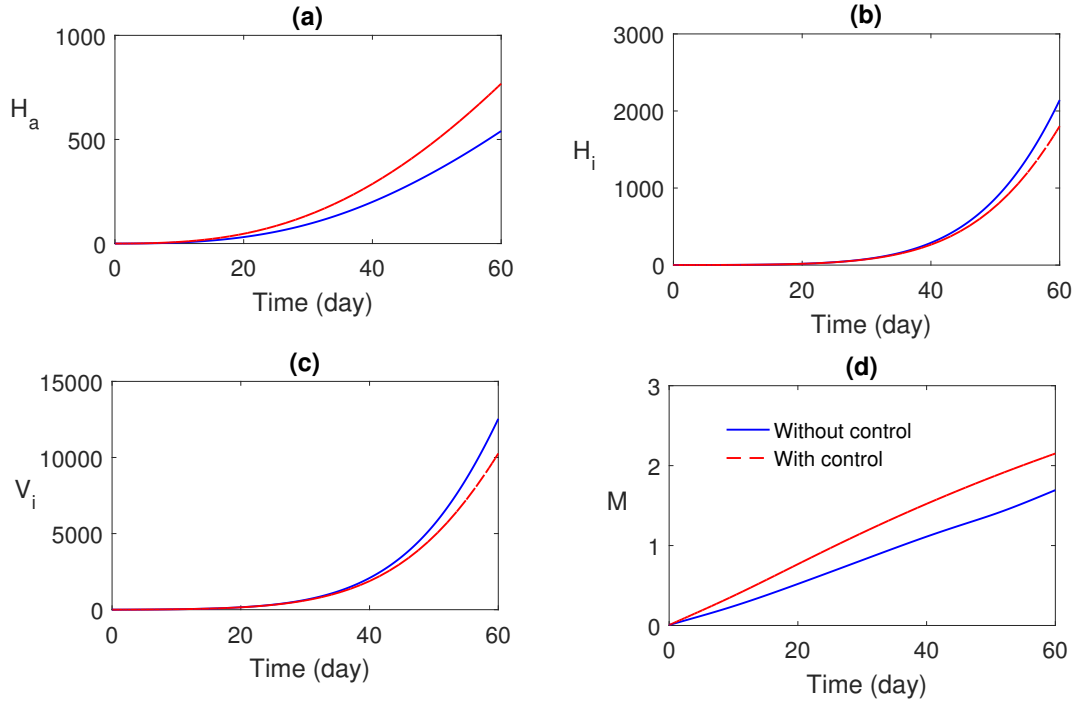

Figure 8. Comparison between the system with and without optimal control.

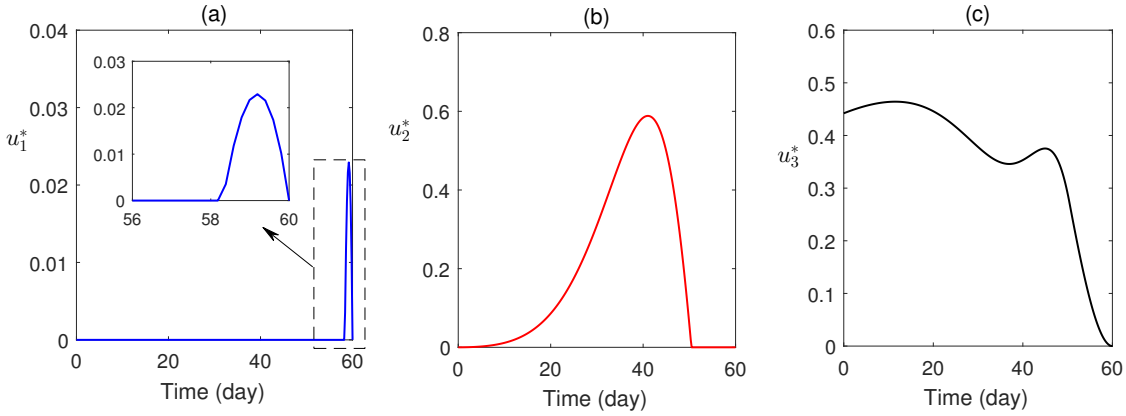

Figure 9. The profiles of optimal controls are plotted as function of time.

6. Numerical simulations

In this section, numerical results are achieved on the basis of analytical calculations. The values of the parameters used in numerical simulations are listed in Table 1.

Figure 2, forward bifurcation of $\mathcal{R}_0$ is sketched. For $\mathcal{R}_0 < 1$, the disease-free equilibrium E_0 is stable and unstable otherwise. Consequently, transcritical bifurcation has occurred at $\mathcal{R}_0 = 1$. This shows the existence of a unique endemic equilibrium E^*.

In Figure 3 ((a)-(f)), the numerical solution of the proposed model system is plotted with two different values of awareness rates. This figure confirms that the influence of consciousness over media has an important role in monitoring malaria disease transmission. Figure 4 shows that the endemic equilibrium point E^*, when it exists, is nonlinearly stable i.e., all the phase portraits converge to the same endemic equilibrium for different initial values.

In Figure 5 ((a)-(f)), the equilibrium values of the infected human population are plotted concerning local awareness rate σ. Infection condensed significantly due to the effect of the local awareness campaign. So, local health centers should organize consciousness movements about the disease. We also plotted the steady state values of the infected human population with respect to

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global awareness ω. A rapid decrease in the infected population is observed in Figure 6. Hence through global media (radio, TV, etc.), awareness about the disease is suggested.

In Figure 7, the instantaneous effect of local and global responsiveness movement is revealed on the infected human population in $\omega - \sigma - H_I^*$ space. Infection condensed due to the impact of both consciousness movements.

6.1. Numerical solution of the Optimal control problem

Here, the results from the numerical simulations are presented to the optimality system (1) with the help of MATLAB.

The optimal control problem deals with the control's effect on the development of malaria disease and also improves the cost sustained in their implementation numerically. The optimal solution is obtained by explaining the optimality system, consisting of six Ordinary Differential Equations (ODEs) from the state and adjoint equations. An iterative structure is used for solving the state equations with an initial guess for the control functions over the pretended time using the fourth-order Runge–Kutta Scheme. Due to the transversality condition (9), the adjoint equations are solved backward in time using the current iteration solutions of the state equations. Then the control functions will be updated by using a convex combination of the preceding control functions and the values from the characterization. This process endures until the change between the values of unknowns at the earlier iteration and that of the current iteration is negligible [32].

Numerical simulations of the optimal control problem are plotted in Figure 8 and 9. Figure 8 compares the system with and without optimal control. It is found that optimal control has a substantial protagonist in monitoring the system. The corresponding optimal profiles of the control variables are plotted in Figure 9. The optimal profiles of the controlling agents indicate that an extra quantity of insecticide spraying is essential.

7. Discussion

This article uses a mathematical model to analyze media campaigns' influence on Malaria's dynamics. To our knowledge, a few articles are available on the impact of malaria disease dynamics and media campaigns. Before discussing the main results, a comparison between the proposed model with the existing mathematical models is made.

Al Basir et al., 2020 [22] have proposed a mathematical model (using delay differential equations) taking human (susceptible and infected) and mosquito populations (susceptible and infected). They have assumed the 'level of awareness' as a separate population for the impact of campaigns through social media. They have yet to apply optimal control theory but focus on the effect of delay in organizing the campaign. In this research, media awareness is assumed as a separate model population that changes with time. Also, susceptible humans are divided into aware and unaware human classes. Moreover, optimal control theory has been applied to maximize the awareness level and cost-effectiveness.

In [23], authors have divided infected humans into aware and unaware infected human populations. Also assumed media as separate model variables whose growth is assumed as proportional to the unaware infected human population. They have not divided the susceptible human into an aware and unaware human. In this research, we divide the susceptible human into aware and unaware susceptible humans, which is more realistic [21]. Moreover, the infected human recovers through awareness-induced treatment, and after recovery, they will join the aware human population. This hypothesis is more realistic.

In [12], authors have formulated a mathematical model taking human (susceptible, infected, recovered) and mosquito (susceptible and infected) populations. The susceptible population is divided into aware and unaware susceptible humans. Finally, they applied optimal control for cost minimization and optimal control of the disease. They have not assumed that recovered people again become susceptible and aware of the disease. Also, the effect of awareness is modeled using constant terms.

In this research, it is assumed additionally that the infected human recovered by awareness-induced treatment, and after recovery, they will join the aware human population, which is more realistic. In the proposed model, 'level of awareness' is taken as a model variable, increasing due to awareness campaigns (as adopted by Al Basir et al. [22]). The local awareness (due to the information from local people and relatives) and global awareness (due to radio and TV campaigns) are also included in the model. Further assumed that the aware people become unaware but decrease with the level of awareness, $M(t)$. Aware people may become infected at a much lower rate than unaware

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humans. Applied optimal control theory has been applied to maximize awareness and minimize the disease control cost.

Thus the awareness-based model proposed here is more functional that can capture the dynamics of Malaria with awareness-based interventions. Also, the control-induced model can minimize the cost of malaria management.

The dynamics of malaria propagation have been studied using the proposed mathematical models analytically and numerically. Using the next-generation matrix, the basic reproduction number $\mathcal{R}_0$ is derived. Equilibria assessment shows two equilibria of the proposed model: the disease-free and endemic. The disease-free equilibrium is stable for $\mathcal{R}_0 < 1$ and endemic equilibrium exists for $\mathcal{R}_0 > 1$ that is when the disease-free equilibrium becomes unstable. The endemic equilibrium, when it exists, is globally asymptotically stable.

Optimal control theory has been applied to awareness-induced intrusions for the cost-effective administration of Malaria. The proposed optimal system is analytically solved using the Pontryagin minimum principle (section 5) and numerically solved (using the scheme stated in subsection 6.1) and plotted the optimal profiles of the control variables (Figure 9). It has been established that the optimally controlled system is essential and effective in malaria disease control (Figure 8).

8. Conclusion

Malaria, the world's most significant dominant disease, is a mosquito-borne human disease caused by a parasite transmitted by a female Anopheles mosquito. Mathematical modeling and control theory helps in predicting the dynamics of the disease and are also helpful for practical policy-making. Awareness campaign about the disease is also equally important in controlling the disease.

In this article, a mathematical model is proposed for a malaria disease dynamic, considering the impact of awareness-based control approaches. The dynamics of the system are analyzed using qualitative stability theory. The optimal control concept is applied for cost minimization to disease control. The maximum principle is implemented for the optimization of the system.

The control-induced model helps optimal disease control with a minimum advertisement, insecticide, and treatment costs using the maximum principle. The obtained results are helpful for policymakers in proposing suitable control strategies against Malaria. In a nutshell, the awareness movement is vital for controlling Malaria, and applying optimal control theory sideways with media consciousness is required.

Funding

Not Applicable.

Competing interests

The authors declare that they have no competing interests.

Authors' contributions

All authors contributed equally to each part of this work.

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Appendix I

The coefficients of (3) is given below:

$$l_1 = -a_{11} - a_{22}, \;\; l_2 = -a_{12}a_{21} + a_{11}a_{22},$$

and

$$m_1 = -a_{33} - a_{44}, \;\; m_2 = -a_{34}a_{43} + a_{33}a_{44},$$

where,

$$\begin{aligned}
a_{11} &= -\frac{\alpha\,\omega}{\theta} - d_h,\\
a_{12} &= -\frac{\theta\, g}{\alpha\,\omega + \theta},\\
a_{14} &= \frac{\lambda_1 k_1}{k_3 N_0},\\
a_{21} &= -\frac{\alpha^2\omega}{\theta},\\
a_{22} &= -d_h - \frac{\theta\, g}{\alpha\,\omega + \theta},\\
a_{23} &= -\frac{r\alpha\,\omega}{\theta},\\
a_{24} &= \frac{\lambda_2 k_2}{k_3 N_0},\\
a_{33} &= -\frac{r\alpha\,\omega}{\theta} - \delta - d_h,\\
a_{34} &= -\frac{\lambda_1 k_1}{k_3 N_0} - \frac{\lambda_2 k_2}{k_3 N_0},\\
a_{43} &= -\frac{\beta\,\Pi_v\theta}{(\gamma\,\omega + \mu\,\theta)N_0},\\
a_{44} &= -\mu - \frac{\gamma\,\alpha\,\omega}{\theta}.
\end{aligned}$$

Appendix J

where $H_i^* = 0$ or $A(H_i^*)^2 + B(H_i^*) + C = 0$ and

$$\begin{aligned}
A =&(\gamma\,\omega + \mu\,\theta)^2 N^4 \alpha\,\delta\,\mu^2\omega\,\theta^4 d_h + \left(N\left(2\,\alpha\,\delta\,\gamma\,\mu\,\omega^2 d_h + \alpha\,\delta\,\mu^2\omega^2 d_h + 2\,\alpha\,\gamma\,\mu\,\omega^2 {d_h}^2\right)\right)^2\theta^3\\
&+ N^2\gamma\, d_h\alpha\,\omega^3(\delta\,\gamma + 2\,\delta\,\mu + \gamma\, d_h + 2\,\mu\, r + 2\,\mu\, d_h)\theta^2\\
B =&N^4\mu^2 d_h(\delta\, g + \delta\, d_h + g d_h)\theta^5(\gamma\,\omega + \mu\,\theta)^2 N^2\\
&+ N^2\mu\,\omega\, d_h(\alpha\,\mu\, d_h + 2\,\delta\, g\gamma + 2\,\delta\,\gamma\, d_h + \delta\,\mu\, d_h + 2\, g\gamma\, d_h + g\mu\, r)\theta^4\\
&- N^2\omega^2 d_h\left(\alpha\,\mu^2 r + \alpha\,\mu^2 d_h + \delta\, g\gamma^2 + \delta\,\gamma^2 d_h + 2\,\delta\,\gamma\,\mu\, d_h + g\gamma^2 d_h + 2\, g\gamma\,\mu\, r + \gamma^2 {d_h}^2\right)\theta^3\\
&- N^2\omega^3 d_h\left(\alpha\,\mu^2 r + \delta\,\gamma^2 d_h + g\gamma^2 r + \gamma^2 r d_h + \gamma^2 {d_h}^2\right)\theta^2 + N^2\gamma^2\omega^4 r\theta\, {d_h}^2\\
C =&(\gamma\,\omega + \mu\,\theta)^2 N^2\left(N^2\mu^2 {d_h}^3 - \beta\, g\Pi_v\pi_h\lambda_1 - \beta\,\Pi_v\pi_h d_h\lambda_1\right)\theta^5\\
&+ \omega\left(2\, N^2\gamma\,\mu\, {d_h}^3 + N^2\mu^2 r {d_h}^2 + N^2\mu^2 {d_h}^3 - \alpha\,\beta\,\Pi_v\pi_h\lambda_2 - \beta\,\Pi_v\pi_h d_h\lambda_1\right)\theta^4\\
&+ \omega^2\left(2\, N^2\gamma\,\mu\, r {d_h}^2 + 2\, N^2\gamma\,\mu\, {d_h}^3 + N^2\mu^2 r {d_h}^2 - \alpha\,\beta\,\Pi_v\pi_h\lambda_2\right)\theta^3 + 2\, N^2\gamma\,\mu\,\omega^3 r\theta^2 {d_h}^2
\end{aligned}$$