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A Deterministic Malaria Mathematical Model Focusing on Immunity, Reinfection, Antimalarial Drug Resistance, Aggressive Treatment and Awareness

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Abstract: Malaria is one of the vector borne diseases which has affected peoples lives economically and has caused deaths across the globe. Therefore, we developed a malaria mathematical model which incorporated drug resistance, reinfection, immunity, aggressive treatment, and awareness on vector control. This comprehensive model has not been researched on well by the researchers, and it has therefore been discussed in this paper. This research will help us to make predictions about the effects of awareness on vector control, drug resistance, immunity, reinfections and aggressive treatment. By fitting the malaria model to the malaria data from the existing literature, important parameters associated with malaria dynamics are estimated and calculated. First, we analyzed the disease free equilibrium of the model and then we calculated the basic reproductive number. Sensitivity analysis was worked out to investigate the most influential parameters. Numerical simulations were done to explore the behavior of the malaria model which included; drug resistance, immunity, reinfection, aggressive treatment, and awareness on vector control. We found out that drug resistance, loss of immunity, reinfection and lack of sensitization increased malaria infections, and lowered the recoveries. Due to these, we did the control strategies which helped reduce the malaria infections and increase recoveries which include high immunity, awareness on vector control, aggressive treatment, and vector control. In conclusion, we found out that when all these control strategies are done at once, the malaria infections decreases, mosquitoes reduces and the recoveries increases. This study will be useful to the ministry of health and the government where they will make people aware on vector control strategies to reduce malaria infections. It will also help the health stake holders to come up with stronger and better antimalarial drugs and immune boosters to help weak immune population who become resistant to drugs.

Keywords: Aggressive Treatment, Malaria, Immunity, Reinfection, Resistance, and Awareness

1. Introduction

Malaria is one among many vector borne diseases which is still a serious problem among researchers and policy makers around the globe by Smith et al. [1]. It is caused by female anopheles mosquitoes through a bite when it feeds on human blood meal. In most cases, it is found in African countries, some parts of Indonesia, Latin America, and in South Asia by World Health Organization 2020 [2]. It has mostly affected young children below 5 years, old age above 70 years, diabetic patients, and HIV/ AIDs patients Tchoumi et al. [3], World Malaria Report 2020 [4]. There are four types of Plasmodium that cause malaria and they include; vivax, falciparum, malaria, and ovale, where P. falciparum is most deadly malaria parasite by World Health Organization 2020 [5], [6]. Several researches have been done and the mathematical modelers have come up with several malaria models. The fight against malaria infections has been done severally, but resistance and reinfections has been one of the problems and setbacks to eliminate it by Ghosh et al. [7],

and Gambhir [8]. A malaria mathematical model with partial immunity was done by Ibrahim et al. [9], where they compared effect transmission, deaths, and birth rates on the malaria dynamics. This study has focused on both partial immunity to the vulnerable human population, and strong immunity which leads to recoveries. Several models have been done on intervention strategies like use of treated bed nets, antimalarial drugs, fumigation and insecticides, and recent studies suggest blocking transmission methods by Woldegerima et al. [10]. This control has been difficult due to resistant to insecticides and anti malarial drugs Adedeji et al. [11], Hyde et al. [12]. Our study, focuses on awareness on vector control where these control strategies are a combination of mosquito nets, clearing bushes, draining stagnant water, mosquito repellents, and early treatment which has not been done before.

A malaria mathematical model on reinfection, and recrudescence by Birx et al. [13], and Price et al. [14] was developed whereby they found that these make malaria become hard to eliminate. Therefore, proper strategies should be developed to eliminate malaria and prevent recrudescence and reinfections by Lawpoolsri et al. [15]. The first models were developed by Ross et al. [16], and Macdonald et al. [17], and since then, more complex ones have been done on malaria transmission dynamics. Another malaria transmission model was developed with temporal immunity, where they found that malaria existed when the basic reproductive number was more than one by Tumwiine et al. [18]. This research did not consider strong immunity and its contribution to responding to anti malarial drugs. Several other malaria models have been done like control interventions y Handari et al. [19], human morbidity by Mugisha et al. [20], temperature dependence by Wan et al. [21], super infection by Aldila et al. [22], and malaria transmission blocking anti malarial drugs by Handari et al. [23]. A model was done on recrudescence, relapse, and reinfection by Abimbade et al. [24]. These authors did not consider intensive treatment due to antimalarial drug resistance on partial immune population, and awareness on vector control which is considered in this research.

Motivated by the above malaria models, especially research done by Al Basir et al. [25]; this research is a comprehensive model which incorporated immunity, reinfection, antimalarial drug resistance, aggressive treatment, and awareness on vector control. The main objective in this paper is to determine the effects of reinfection, strong immunity, weak immunity, antimalarial drug resistance, awareness on vector control, and aggressive treatment on humans with weak immunity who develop drug resistance. This paper is arranged as follows; Section 2 is the research methodology, Section 3 is the model analysis, Section 4 are numerical simulations, Section 5 are the Discussion, Conclusions, and Recommendations.

2. Research Methodology

2.1. Mathematical Malaria Model and Description

We developed a deterministic compartmental malaria model to determine the impact of immunity, drug resistance, recurrence, and awareness on vector control. This study has human and mosquito populations divided into eight classes. The susceptible human population increase by natural births at a rate $\mathbf{\Lambda_h}$, and decrease when some are bitten by actively infected mosquitoes. They also reduce by natural deaths at a rate $\mu_{\mathbf{h}}$. They become latently infected at a rate $\frac{b_{vh}\gamma A_v S_h}{N}$, where b_{vh} is the probability that a bite from an infected mosquito will result to an infection. γ is the biting rate, and N is the total human population. Latently infected human population (L_h), do not show any signs and symptoms and are assumed that they cannot transmit malaria. If the latently infected are aware on vector control, then they will use thw treated mosquito nets, some will go for check up, and if they have malaria they will be treated and recover at a rate $a\alpha$. Where a is the proportion of those who are aware of vector control strategies. Those who are not aware, become actively infected A_h at a rate $(1-a)\alpha$. $(1-a)$ is the proportion of the population which is not aware of the vector control strategies. Actively infected population are composed of humans with strong and weak immunity. Those with weak immunity become resistant to drugs Re_h at a rate $\phi_{\mathbf{h}}$. Those with strong immunity recover at a rate $\sigma_{\mathbf{h}}$ and move to the recovery class R_h. Those with weak immunity and are resistant to antimalarial drugs recover after being given stronger anti malarial drugs (thorough treatment) at a rate $\lambda_{\mathbf{h}}$. We assume that every human compartment has a natural birth rate, $\mu_{\mathbf{h}}$, while actively infected and resistant to drugs human population have disease induced death rates denoted by $\omega_{\mathbf{h}}$. The recovered human population become recrudescence due to ineffective treatment and reinfected due bites from the mosquitoes at a rate $\frac{b_{vh}\gamma\Upsilon R_h A_v}{N}$. We assume that once the individuals recover from malaria parasite, they can become susceptible again and lose immunity at a rate $\tau_{\mathbf{h}}$.

Mosquitoes population has three compartments namely; Susceptible vector S_v, Latently infected vector (L_v), and actively infected vector A_v. Mosquitoes are suspected to be born susceptible at a rate $\mathbf{\Lambda_v}$. They become latently infected at a rate $\frac{\gamma b_{hv} S_v A_h}{N}$, where γ is the biting rate, b_{hv} is the probability when a susceptible mosquito feeds on the infected human blood meal and becomes infected. Latently infected vector becomes actively infected at a rate ρ. We assumed that all the mosquito classes have natural death rates $\mu_{\mathbf{v}}$. Again, they die due to the spraying of insecticide, use of repellants, clearing swampy and grassy areas at a rate $\delta_{\mathbf{v}}$ in A_v, L_v, and S_v compartments. Mathematical Model Assumptions include;
1. The actively infected human and mosquito population are the ones who can transmit malaria infections to susceptible mosquito and humans because this study is focused on actively infectious population.
2. All human populations compartment have natural death rates $\mu_{\mathbf{h}}$, while the mosquito population have natural death rates denoted as $\mu_{\mathbf{v}}$.
3. All mosquito population have a constant disease induced death rates due to spraying of insecticides, clearing bushes, draining stagnant water, and use of mosquito repellants.

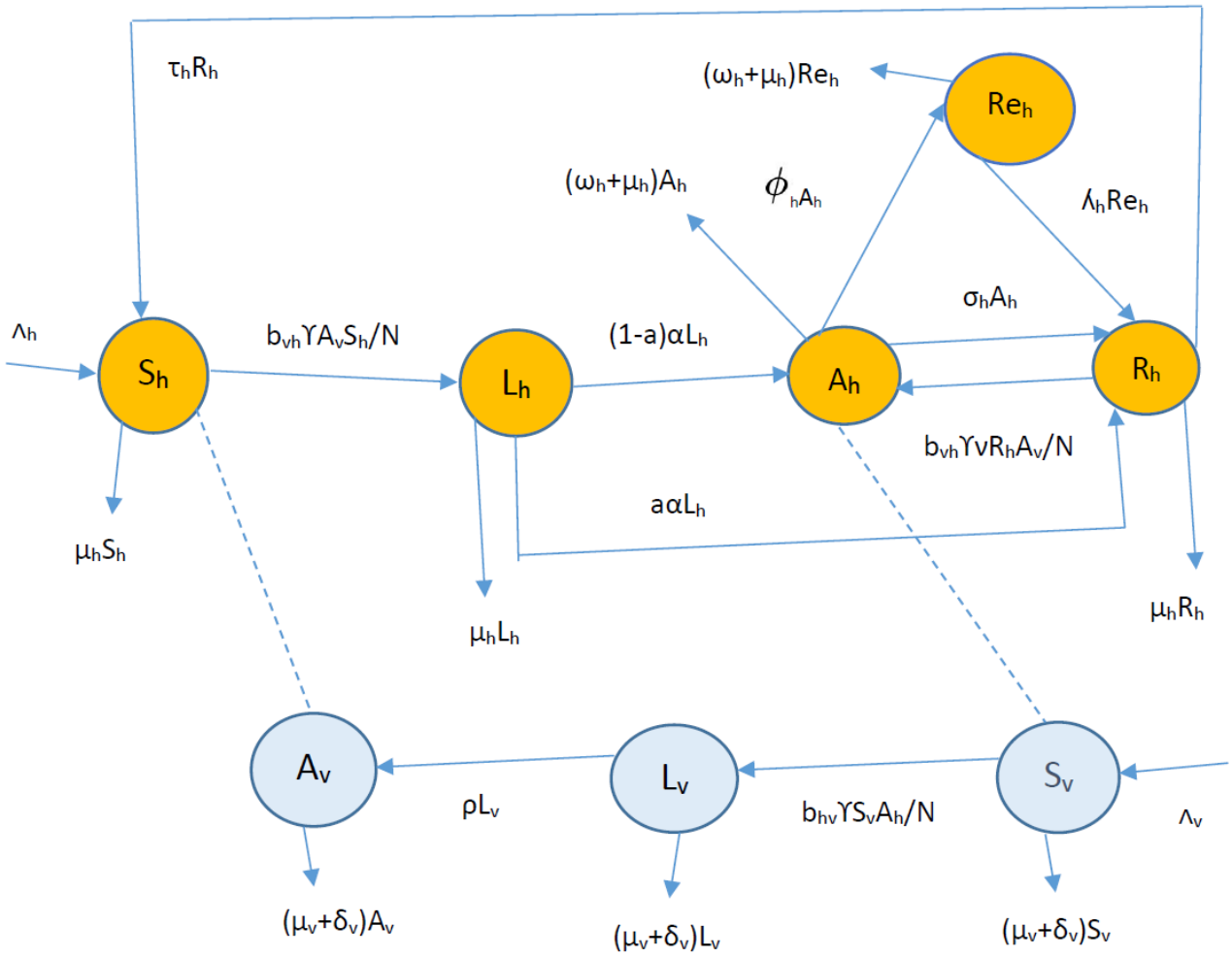


Figure 1. *Mathematical Malaria Model.*

2.2. Mathematical Malaria Model Equations

The following are Equations for the Mathematical malaria Model Figure 1;

$$\frac{dS_h}{dt} = \mathbf{\Lambda_h} + \tau_h R_h - \frac{b_{vh}\gamma A_v S_h}{N} - \mu_\mathbf{h} S_h. \tag{1}$$

$$\frac{dL_h}{dt} = \frac{b_{vh}\gamma A_v S_h}{N} - (1-a)\alpha L_h - a\alpha L_h - \mu_\mathbf{h} L_h. \tag{2}$$

$$\frac{dA_h}{dt} = (1-a)\alpha L_h + \frac{b_{vh}\gamma \Upsilon R_h A_v}{N} - \phi_h A_h - (\omega_h + \mu_\mathbf{h})A_h - \sigma_h A_h. \tag{3}$$

$$\frac{dRe_h}{dt} = \phi_\mathbf{h} A_h - \lambda_\mathbf{h} Re_h - (\omega_\mathbf{h} + \mu_\mathbf{h})Re_h. \tag{4}$$

$$\frac{dR_h}{dt} = \sigma_\mathbf{h} A_h + \lambda_\mathbf{h} Re_h + a\alpha L_h - \frac{b_{vh}\gamma \Upsilon R_h A_v}{N} - \tau_\mathbf{h} R_h - \mu_\mathbf{h} R_h. \tag{5}$$

$$\frac{dS_v}{dt} = \mathbf{\Lambda_v} - \frac{\gamma b_{hv} S_v A_h}{N} - (\mu_\mathbf{v} + \delta_v)S_v. \tag{6}$$

$$\frac{dL_v}{dt} = \frac{\gamma b_{hv} S_v A_h}{N} - \rho L_v - (\mu_\mathbf{v} + \delta_\mathbf{v})L_v. \tag{7}$$

$$\frac{dA_v}{dt} = \rho L_v - (\mu_\mathbf{v} + \delta_\mathbf{v})A_v. \tag{8}$$

2.3. Description of Mathematical Model Variables and Parameters

$\mathbf{\Lambda_h}$ is the Human birth rate.
$\mathbf{\Lambda_v}$ is the Vector birth rate.
$\mu_\mathbf{h}$ is the Natural human death rate.
$\mu_\mathbf{v}$ is the Natural vector death rate.
b_{vh} is the Probability of transmission from infected mosquito to susceptible host.
b_{hv} is the Probability of transmission from infected host to susceptible vector.
γ is the Vector biting rate on humans per day.
$(1-a)$ is the Fraction of human not aware of vector control.
a is the Fraction of the human aware of vector control.
α is the Rate at which latently infected become actively infected.
$\omega_\mathbf{h}$ is the Malaria induced death rates.
$\sigma_\mathbf{h}$ is the Rate at which strong immune recover.
$\phi_\mathbf{h}$ is the Rate at which the weak immune resist drugs.
$\lambda_\mathbf{h}$ is the Rate at which drug resistant population recover after aggressive treatment.
Υ is the Rate at which recovered become recrudescence and get active malaria.
$\tau_\mathbf{h}$ is the Rate at which recovered lose immunity and become susceptible.
$\delta_\mathbf{v}$ is the Death rates of vector due to vector control.
S_h is the Susceptible host Population.
L_h is the Latently Infected host population.
A_h is the Actively Infected host population.
Re_h is the Resistant to antimalarial drugs host population.
R_h is the Recovered host Population.
S_v is the Susceptible vector Population.
L_v is the Latently infected mosquito vector.
A_v is the Actively Infected mosquito vector population.

3. Analysis of the Mathematical Model

3.1. Mathematical Model Basic Properties

We obtained the mathematical model parameters from the existing literature except a few parameters like; α, $\phi_\mathbf{h}$, $\sigma_\mathbf{h}$, Υ, $\lambda_\mathbf{h}$, $\tau_\mathbf{h}$, and $\delta_\mathbf{v}$ whose values were assumed to obtain better results.

Theorem: The closed set $\Re = D_+^8 : N_h \leq \frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}, N_v \leq \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}}$ is invariantly positive and attracting for the malaria model figure 1.

Proof: Adding the first five equations, and the last three equations for the malaria mathematical model figure (i) gives; $\dot{N_h} = \mathbf{\Lambda_h} - \mu_\mathbf{h} N_h - \omega_\mathbf{h}(L_h + A_h + Re_h + R_h)$, and $\dot{N_m} = \mathbf{\Lambda_v} - (\mu_\mathbf{v} + \delta_\mathbf{v})N_m$.

Since $\dot{N_h} \leq \mathbf{\Lambda_h} - \mu_\mathbf{h} N_h$, and $\dot{N_m} \leq \mathbf{\Lambda_v} - (\mu_\mathbf{v} + \delta_\mathbf{v})N_m$, it follows that, $\dot{N_h} \leq 0$, and $\dot{N_m} \leq 0$, if $N_h \geq \frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}$, and N_m approaches $\frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}}$ respectively.

Therefore, comparison theorem by Lakshmikantham et al. [26], $N_h(0)e^{-\mu_\mathbf{h}(t)} \geq N_h + (1 - e^{-\mu_\mathbf{h}(t)})\frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}$, and $N_v(0)e^{-\mu_\mathbf{v}(t)} \geq 0 + (1 - e^{-\mu_\mathbf{v}(t)})\frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}}$.

In particular, $\dot{N_h} \leq \frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}$, and $\dot{N_v} \leq \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}}$ respectively. Thus region $\Re$ is invariantly positive for the mathematical malaria model figure (i). Again, if $N_h(0) > \frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}$, and $N_v(0) > \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}}$, then either the solutions enters $\Re$ infinite time or $N_h \to \frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}$ and $N_v \to \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}}$ as $t \to \inf$. Therefore, region $\Re$ attracts all results in D_+^8.

Since the region $\Re$ is positively invariant, the usual existence, uniqueness of the results hold the system of the mathematical malaria model figure (i). It is therefore sufficient to consider the dynamics of the flow generated by the malaria mathematical model system 1 in region $\Re$ by Hethcote et al. [27].

Theorem: Let $S_h(0) > 0, L_h(0) \geq 0, A_h(0) \geq 0, Re_h(0) \geq 0, R_h(0) \geq 0, S_v(0) > 0, L_v(0) \geq 0$ and $A_v(0) \geq 0$. Then, the solutions to the malaria mathematical model system 1; $S_h, L_h, Re_h, R_h, S_v, L_v$, and A_v are all negative at $t > 0$.

Proof: Taking equation 1 of our malaria mathematical model system figure 1, we obtain;

$$\frac{dS_h}{dt} = \mathbf{\Lambda_h} + \tau_\mathbf{h} R_h - (\frac{b_{vh}\gamma A_v}{N} + \mu_\mathbf{h})S_h. \tag{9}$$

. By setting $\xi = \frac{b_{vh}\gamma A_v}{N} + \mu_\mathbf{h}$, we reduce equation 9 to $\frac{dS_h}{dt} \geq \mathbf{\Lambda_h} - \xi S_h$. With the integrating factor $e^{(\int_0^t \xi S_h(S)dS)}$, where the inequality is solved to yield;

$$\frac{d}{dt}[S_h(t)e^{(\int_0^t \xi S_h(S)dS)}] \geq \mathbf{\Lambda_h e^{(\int_0^t \xi S_h(S) dS)}} \tag{10}$$

Integrating equation 10 both sides we get;

$$S_h(t)e^{(\int_0^t \xi S_h(S)dS)} \geq S_h(0) + \int_0^t \mathbf{\Lambda_h} e^{(\int_0^t \xi S_h(S)dS)}dx. \tag{11}$$

which yields

$$S_h(t) \geq [S_h(0) + \int_0^t \mathbf{\Lambda_h} e^{(\int_0^t \xi S_h(S)dS)dx}]e^{(-\int_0^x \xi S_h(S)dS) \geq 0}. \tag{12}$$

Therefore, $S_h(t)$ with the initial conditions $S_h(0) \geq 0$ is non negative. Again, utilizing similar approach to other variables, we can prove that all the state variables are non negative when they satisfy their respective initial conditions.

3.2. The Disease Free Equilibrium

To evaluate it, we set all the compartments to zero except S_h and S_v, that is classes L_h, A_h, Re_h, R_h, L_v, and A_v are set to zero.

That is; Disease Free Equilibrium
= $(\frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}, 0, 0, 0, 0, \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}}, 0, 0)$.

3.3. The Basic Reproductive Number

We compute the basic reproductive number by the use of a technique known as the Next Generation Matrix by Driessche et al. [28].

There are five compartments which are diseased and we will use them in this order; L_h, A_h, Re_h, L_v, and A_v.F contains only secondary infections, while V contains other terms which do not include secondary infections. Where by F is given by;

$$F = \begin{bmatrix} \frac{b_{vh}\gamma A_v S_h}{N} \\ \frac{b_{vh}\gamma Y R_h A_v}{N} \\ 0 \\ \frac{\gamma b_{hv} S_v A_h}{N} \\ 0 \end{bmatrix}^T.$$ Differentiating F matrix at the Disease Free Equilibrium, we get;

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{\mathbf{\Lambda_h} b_{vh}\gamma}{N\mu_\mathbf{h}} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\mathbf{\Lambda_v} b_{hv}\gamma}{N(\delta_\mathbf{v}+\mu_\mathbf{v})} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix}.$$ The other transmission infections in the system are given by;

$$V = \begin{bmatrix} (1-a)\alpha L_h + a\alpha L_h - \mu_\mathbf{h} L_h \\ -(1-a)\alpha L_h + \phi_\mathbf{h} A_h + (\omega_\mathbf{h} + \mu_\mathbf{h}) A_h \\ \lambda_\mathbf{h} Re_h + (\omega_\mathbf{h} + \mu_\mathbf{h}) Re_h \\ \rho L_v + (\mu_\mathbf{v} + \delta_\mathbf{v}) L_v \\ -\rho L_v + (\mu_\mathbf{v} + \delta_\mathbf{v}) A_v \end{bmatrix}^T.$$ Differentiating V matrix at the Disease Free Equilibrium, we get;

$$F = \begin{bmatrix} Q & 0 & 0 & 0 & 0 \\ \alpha(a-1) & R & 0 & 0 & 0 \\ 0 & 0 & \lambda_\mathbf{h} + \mu_\mathbf{h} + \omega_\mathbf{h} & 0 & 0 \\ 0 & 0 & 0 & \delta_\mathbf{v} + \mu_\mathbf{v} + \rho & 0 \\ 0 & 0 & 0 & -\rho & \delta_\mathbf{v} + \mu_\mathbf{v} \end{bmatrix}.$$

Let Q be $a\alpha + \alpha(1-a) - \mu_\mathbf{h}.and Rbe \mu_\mathbf{h} + \omega_\mathbf{h} + \phi_\mathbf{h}$.

With the help of Jupiter notebook in python software, we calculated the V^{-1} and FV^{-1}. Where the most dominant eigen value is given by;

$R_0 = \gamma\sqrt{\frac{-\mathbf{\Lambda_h}\mathbf{\Lambda_v}\alpha b_{vh} b_{hv}\rho(a-1)}{\mu_\mathbf{h}(\alpha-\mu_\mathbf{h})(\delta_\mathbf{v}+\mu_\mathbf{v}+\rho)(\mu_\mathbf{h}+\omega_\mathbf{h}+\phi_\mathbf{h})N^2(\delta_\mathbf{v}+\mu_\mathbf{v})^2}}$.

3.4. Local Stability of the Disease Free Equilibrium

Theorem: The disease free equilibrium is locally asymptotically stable whenever $R_0 < 1$, and unstable otherwise.

Proof: The Jacobian matrix model at the disease free equilibrium is given by,

$$J_8 = \begin{bmatrix} -\mu & 0 & 0 & 0 & \tau_\mathbf{h} & 0 & 0 & \frac{-b_{vh}\gamma\mathbf{\Lambda_h}}{\mu_\mathbf{h} N} \\ 0 & E & 0 & 0 & 0 & 0 & 0 & \frac{b_{vh}\gamma\mathbf{\Lambda_h}}{\mu_\mathbf{h} N} \\ 0 & J & A & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_\mathbf{h} & B & 0 & 0 & 0 & 0 \\ 0 & a\alpha & \sigma_\mathbf{h} & \lambda_\mathbf{h} & G & 0 & 0 & 0 \\ 0 & 0 & \frac{-\gamma b_{hv}\mathbf{\Lambda_v}}{(\mu_\mathbf{v}+\delta_\mathbf{v})N} & 0 & 0 & H & 0 & 0 \\ 0 & 0 & \frac{\gamma b_{hv}\mathbf{\Lambda_v}}{(\mu_\mathbf{v}+\delta_\mathbf{v})N} & 0 & 0 & 0 & I & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \rho & F \end{bmatrix}.$$

Let A= $-(\phi_\mathbf{h} + \omega_\mathbf{h} + \mu_\mathbf{h} + \sigma_\mathbf{h})$
E = $-(\alpha + \mu_\mathbf{h})$
F = $-(\mu_\mathbf{v} + \delta_\mathbf{v})$
G = $-(\tau_\mathbf{h} + \mu_\mathbf{h})$
H = $-(\mu_\mathbf{v} + \delta_\mathbf{v})$
I = $-(\rho + \mu_\mathbf{v} + \delta_\mathbf{v})$
J = $\alpha - a\alpha \; and \; B = -(\lambda_\mathbf{h} + \omega_\mathbf{h} + \mu_\mathbf{h})$.

The characteristic polynomial corresponding to the J_8 matrix is $P_8(\lambda) = (-\mu-\lambda)(-\alpha-\mu_\mathbf{h}-\lambda)(-\phi_\mathbf{h}-\omega_\mathbf{h}-\mu_\mathbf{h}-\sigma_\mathbf{h}-\lambda)(-\lambda_\mathbf{h}-\omega_\mathbf{h}-\mu_\mathbf{h}-\lambda)(-\tau_\mathbf{h}-\mu_\mathbf{h}-\lambda)(-\mu_\mathbf{v}-\delta_\mathbf{v}-\lambda)(-\rho-\mu_\mathbf{v}-\delta_\mathbf{v}-\lambda)(\mu_\mathbf{v}-\delta_\mathbf{v}-\lambda) = 0$, where all te eigen values of the Jacobian matrix are negative real parts, then the disease free equilibrium is locally asymptotically stable.

3.5. Global Stability at the Disease Free Equilibrium

Theorem: The disease free equilibrium point for the malaria model system figure 1 is globally asymptotically stable if $R_0 < 1$.

Proof: To proof the global stability at the disease free equilibrium, the approach in by Castillo-Chavez et al. [42] is used. The total population is divided in to uninfected and diseased population. The malaria mathematical model is then written as;

$\frac{dX}{dt} = F(X, X_1)$, and $\frac{dX_1}{dt} = G(X, X_1), G(X, 0) = 0$.

Where $X = (S_h, R_h, S_v) \epsilon \top_+^3$, represents the number of uninfected human and mosquito population and $X_1 =$

$[L_h, A_h, Re_h, L_v, A_v]\epsilon\top_+^5$ represents the diseased human and mosquito populations. The disease free equilibrium is then denoted as $E_0 = (X_0, 0)$. It is important to prove these two criteria;
a). For $\frac{dX}{dt} = F(X,0)$, X_0 is globally asymptotically stable and,
b). $\hat{G} = JX_1 - \hat{G}(X, X_1), G(X, X_1) \geq 0$, for all $(X, X_1)\epsilon\psi$. Where $J = G(X_0, 0)$ is a Metzler Matrix meaning the off diagonal elements of J are not negative and ψ is biologically meaningful region of the model.

Case 1: Consider the uninfected class;
$X^1(t) = \frac{d}{dt}\begin{bmatrix} S_h \\ R_h \\ S_v \end{bmatrix} = \begin{bmatrix} \mathbf{\Lambda_h} + \tau_\mathbf{h} R_h - \frac{b_{vh}\gamma A_v S_h}{N} - \mu_\mathbf{h} S_h \\ \sigma_\mathbf{h} A_h + \lambda_\mathbf{h} Re_h + a\alpha L_h - \frac{b_{vh}\gamma \mathbf{\Upsilon} R_h A_v}{N} - \tau_\mathbf{h} R_h - \mu_\mathbf{h} R_h \\ \mathbf{\Lambda_v} - \frac{\gamma b_{hv} S_v A_h}{N} - (\mu_\mathbf{v} + \delta_\mathbf{v}) S_v \end{bmatrix}$. At the disease free equilibrium when $X_1 = 0$, that is $L_h = I_h = Re_h = L_v = A_v = R_h = 0$, the uninfected population becomes;
$\frac{d}{dt}\begin{bmatrix} S_h \\ R_h \\ S_v \end{bmatrix} = \begin{bmatrix} \mathbf{\Lambda_h} - \mu_\mathbf{h} S_h \\ 0 \\ \mathbf{\Lambda_v} - (\mu_\mathbf{v} + \delta_\mathbf{v}) S_v \end{bmatrix}$. Integrating the system by use of the separation of variables gives;
$S_h(t) = \frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}} + (S_h(0) - \frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}})e^{-\mu_\mathbf{h} t}$,
$S_v(t) = \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}} + (S_v(0) - \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}})e^{-(\mu_\mathbf{v}+\delta_\mathbf{v})t}$. So that $t \to \infty$, $S_h(t) \to \frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}$,
$R_h(t) \to 0$, $S_v(t) \to \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}}$. Regardless of the values $S_h(0), R_h(0), S_v(0)$, then $X_0 = (\frac{\mathbf{\Lambda_h}}{\mu_\mathbf{h}}, 0, \frac{\mathbf{\Lambda_v}}{\mu_\mathbf{v}+\delta_\mathbf{v}})$ is globally asymptotically stable for the subsystem $F(X, 0)$.

Case 2: It is also necessary to prove $\hat{G}(X, X_1) = JX_1 - G(X, X_1), \hat{G}(X, X_1) \geq 0$. Therefore the Jacobian matrix of the diseased subsystem is computed as follows;

$$X_1^1(t) = G(X, X_1) = \frac{d}{dt}\begin{bmatrix} \frac{b_{vh}\gamma A_v S_h}{N} - (1-a)\alpha L_h - a\alpha L_h - \mu_\mathbf{h} L_h \\ (1-a)\alpha L_h + \frac{b_{vh}\gamma \mathbf{\Upsilon} R_h A_v}{N} - \phi_\mathbf{h} A_h - (\omega_\mathbf{h} + \mu_\mathbf{h}) A_h - \sigma_\mathbf{h} A_h \\ \phi_\mathbf{h} A_h - \lambda_\mathbf{h} Re_h - (\omega_\mathbf{h} + \mu_\mathbf{h}) Re_h \\ \frac{\gamma b_{hv} S_v A_h}{N} - \rho L_v - (\mu_\mathbf{v} + \delta_\mathbf{v}) L_v \\ \rho L_v - (\mu_\mathbf{v} + \delta_\mathbf{v}) A_v \end{bmatrix}.$$

The Jacobian matrix at the Disease Free Equilibrium becomes;

$$J_5 = \begin{bmatrix} -(\alpha + \mu_\mathbf{h}) & 0 & 0 & 0 & \frac{b_{vh}\gamma \mathbf{\Lambda_h}}{\mu_\mathbf{h} N} \\ (1-a)\alpha & L & 0 & 0 & 0 \\ 0 & \phi_\mathbf{h} & M & 0 & 0 \\ 0 & \frac{\gamma b_{hv} \mathbf{\Lambda_v}}{(\mu_\mathbf{v}+\delta_\mathbf{v})N} & 0 & N & 0 \\ 0 & 0 & 0 & \rho & -(\mu_\mathbf{v} + \delta_\mathbf{v}) \end{bmatrix}.$$

Where L = $-(\phi_\mathbf{h} + \omega_\mathbf{h} + \mu_\mathbf{h} + \sigma_\mathbf{h})$
M = $-(\lambda_\mathbf{h} + \omega_\mathbf{h} + \mu_\mathbf{h})$ *and* $N = -(\rho + \mu_\mathbf{v} + \delta_\mathbf{v})$.
But $\hat{G}(X, X_1) = JX_1 - G(X, X_1)$, now subtract the matrix which gives;

$$\hat{G}(X, X_1) = \begin{bmatrix} \hat{G}_1(X, X_1) \\ \hat{G}_2(X, X_1) \\ \hat{G}_3(X, X_1) \\ \hat{G}_4(X, X_1) \\ \hat{G}_5(X, X_1) \end{bmatrix} = \begin{bmatrix} \frac{b_{vh}\gamma A_v S_h^0}{N} - \frac{b_{vh}\gamma A_v S_h}{N} \\ 0 \\ 0 \\ \frac{\gamma b_{hv} S_v^0 A_h}{N} - \frac{\gamma b_{hv} S_v A_h}{N} \\ 0 \end{bmatrix} = \begin{bmatrix} \frac{b_{vh}\mathbf{\Upsilon} A_v}{N}(1 - \frac{S_h}{S_h^0}) \\ 0 \\ 0 \\ \frac{b_{hv}\mathbf{\Upsilon} A_h}{N}(1 - \frac{S_v}{S_v^0}) \\ 0 \end{bmatrix}.$$

It is clearly seen that, $S_h^0 > S_h$ and $S_v^0 > S_v$, then $\hat{G}(X, X_1) \geq 0$ for all $(X, X_1)\epsilon\psi$. Thus case 2 satisfied. Since case 1 and case 2 are satisfied, then the disease free equilibrium point is globally asymptotically stable. Biologically, the above means that no matter how huge the disease spread is globally, the malaria infections will perish out in the population given $R_0 < 1$. Otherwise, if $R_0 > 1$, the malaria disease will continue to spread in the population.

3.6. Endemic Equilibrium

After some algebraic calculations, the endemic equilibrium is showed below;
$E_2^* = (S_h^*, L_h^*, A_h^*, Re_h^*, S_v^*, L_v^*, A_v^*.)$ for the malaria disease infections to persist in human and mosquito populations the state variables take the following form; Where;

$$S_h^* = \frac{-\mathbf{\Lambda_h} - \tau_\mathbf{h} R_h}{\frac{b_{vh}\gamma A_v}{N} + \mu_\mathbf{h}}. \tag{13}$$

$$L_h^* = \frac{b_{vh}\gamma A_v S_h}{N([1-a]\alpha + a\alpha + \mu_\mathbf{h})}. \tag{14}$$

$$A_h^* = \frac{(1-a)\alpha L_h + b_{vh}\gamma \mathbf{\Upsilon} R_h A_v}{N(\phi_\mathbf{h} + \omega_\mathbf{h} + \mu_\mathbf{h} + \sigma_\mathbf{h})}. \tag{15}$$

$$Re_h^* = \frac{\phi_\mathbf{h} A_h}{\lambda_\mathbf{h} + \omega_\mathbf{h} + \mu_\mathbf{h}}. \tag{16}$$

$$S_v^* = \frac{\mathbf{\Lambda_v}}{\frac{\gamma b_{hv} A_h}{N} + \mu_\mathbf{v} + \delta_\mathbf{v}}. \tag{17}$$

$$L_v^* = \frac{\gamma b_{hv} S_v A_h}{N(\rho + \mu_{\mathbf{v}} + \delta_{\mathbf{v}})}. \quad (18) \qquad A_v^* = \frac{\rho L_v}{\mu_{\mathbf{v}} + \delta_{\mathbf{v}}}. \quad (19)$$

3.7. Local Stability at the Endemic Equilibrium Point

Theorem: The endemic equilibrium is said to be locally asymptotically stable if $R_0 > 1$, otherwise it is unstable.

Proof: The jacobian matrix of the malaria mathematical model system in figure 1 is given by;

$$J_8 = \begin{bmatrix} -\mu & 0 & 0 & 0 & \tau_{\mathbf{h}} & 0 & 0 & \frac{-b_{vh}\gamma\Lambda_{\mathbf{h}}}{\mu_{\mathbf{h}}N} \\ 0 & -(\alpha+\mu_{\mathbf{h}}) & 0 & 0 & 0 & 0 & 0 & \frac{b_{vh}\gamma\Lambda_{\mathbf{h}}}{\mu_{\mathbf{h}}N} \\ 0 & \alpha - a\alpha & M & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \phi_{\mathbf{h}} & P & 0 & 0 & 0 & 0 \\ 0 & a\alpha & \sigma_{\mathbf{h}} & \lambda_{\mathbf{h}} & X & 0 & 0 & 0 \\ 0 & 0 & \frac{-\gamma b_{hv}\Lambda_{\mathbf{v}}}{(\mu_{\mathbf{v}}+\delta_{\mathbf{v}})N} & 0 & 0 & Y & 0 & 0 \\ 0 & 0 & \frac{\gamma b_{hv}\Lambda_{\mathbf{v}}}{(\mu_{\mathbf{v}}+\delta_{\mathbf{v}})N} & 0 & 0 & 0 & Z & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \rho & -(\mu_{\mathbf{v}}+\delta_{\mathbf{v}}) \end{bmatrix}$$. Where $M = -(\phi_{\mathbf{h}} + \omega_{\mathbf{h}} + \mu_{\mathbf{h}} + \sigma_{\mathbf{h}})$

X = $-(\tau_{\mathbf{h}} + \mu_{\mathbf{h}})$, Y = $-(\mu_{\mathbf{v}} + \delta_{\mathbf{v}})$

Z = $-(\rho + \mu_{\mathbf{v}} + \delta_{\mathbf{v}})$ and $P = -(\lambda_{\mathbf{h}} + \omega_{\mathbf{h}} + \mu_{\mathbf{h}})$. And by expanding the determinant of the characteristic equation, determinant of $(J(E_1) - \lambda I) = 0$, the result shows; $\lambda_{\mathbf{1}} = -\mu, \lambda_{\mathbf{2}} = -(\alpha + \mu_{\mathbf{h}}), \lambda_{\mathbf{3}} = -(\phi_{\mathbf{h}} + \omega_{\mathbf{h}} + \mu_{\mathbf{h}} + \sigma_{\mathbf{h}}), \lambda_{\mathbf{4}} = B = -(\lambda_{\mathbf{h}} + \omega_{\mathbf{h}} + \mu_{\mathbf{h}}), \lambda_{\mathbf{5}} = -(\tau_{\mathbf{h}} + \mu_{\mathbf{h}}), \lambda_{\mathbf{6}} = -(\mu_{\mathbf{v}} + \delta_{\mathbf{v}}), \lambda_{\mathbf{7}} = -(\rho + \mu_{\mathbf{v}} + \delta_{\mathbf{v}})$, and $\lambda_{\mathbf{8}} = -(\mu_{\mathbf{v}} + \delta_{\mathbf{v}})$. The remaining eigenvalues can be obtained from the matrix;

$$J_6 = \begin{bmatrix} Z_1 & 0 & 0 & \frac{b_{vh}\gamma\Upsilon A_v}{N} & 0 & \frac{b_{vh}\gamma S_h}{N} \\ (1-a)\alpha & Y_1 & 0 & 0 & 0 & \frac{b_{vh}\gamma\Upsilon}{N} \\ 0 & \phi_{\mathbf{h}} & L_1 & 0 & 0 & 0 \\ a\alpha L_h & \sigma_{\mathbf{h}} & \lambda_{\mathbf{h}} & X_1 & 0 & -(\frac{b_{vh}\gamma\Upsilon R_h}{N}) \\ 0 & \frac{\gamma b_{hv} S_v}{N} & 0 & 0 & -\rho & 0 \\ 0 & 0 & 0 & 0 & \rho & -(\mu_{\mathbf{v}} + \delta_{\mathbf{v}}) \end{bmatrix}$$. Let $Z_1 = -(\alpha + 2a\alpha + \mu_{\mathbf{h}})$

$Y_1 = -(\phi_{\mathbf{h}} + \omega_{\mathbf{h}} + \mu_{\mathbf{h}})$ $X_1 = -(\frac{b_{vh}\gamma\Upsilon A_v}{N} + \tau_{\mathbf{h}} + \mu_{\mathbf{h}})$

and $L_1 = -(\lambda_{\mathbf{h}} + \omega_{\mathbf{h}} + \mu_{\mathbf{h}})$.

The characteristic polynomial for the matrix $J_6 = \lambda^{\mathbf{6}} + B_1\lambda^{\mathbf{5}} + B_2\lambda^{\mathbf{4}} + B_3\lambda^{\mathbf{3}} + B_4\lambda^{\mathbf{2}} + B^5\lambda^{\mathbf{1}} + B_6$. Its corresponding Routh-Horwitz matrix is given by; $J_6 = \begin{bmatrix} B_1 & B_3 & B_5 & 0 & 0 & 0 \\ 1 & B_2 & B_4 & B_6 & 0 & 0 \\ 0 & B_1 & B_3 & B_5 & 0 & 0 \\ 0 & 1 & B_2 & B_4 & B_6 & 0 \\ 0 & 0 & B_1 & B_3 & B_5 & 0 \\ 0 & 0 & 1 & B_2 & B_4 & B_6 \end{bmatrix}$.

The endemic equilibrium point is locally asymptotically stable if and only if the principle leading minors of M_n are all positive for $n = 1, 2, 3, 4, 5, 6$. Then, the six eigen values of the jacobian matrix J_6 have negative real parts if they satisfy the Routh- Hurwitz criteria, that is $\Delta M_1, \Delta M_2, \Delta M_3, \Delta M_4, \Delta M_5, \Delta M_6 > 0$. Since all conditions of Routh Hurwitz meaning the leading minors of ΔM_n are satisfied, the following theorem is established.

Theorem: If the leading minors of ΔM_n are all positive where $n = 1, 2, 3, 4, 5, 6$, then the endemic equilibrium point of the malaria model system figure 1 is locally asymptotically stable, otherwise unstable.

3.8. Global Stability at the Endemic Equilibrium Point

Theorem: The E_0 of the malaria mathematical model system Figure (i) is globally asymptotically stable if $R_0 < 1$.

Proof: The Lyapunov function is defined as ; $V = L_h + v_1A_h + v_2Re_h + v_3L_v + v_4A_v$. where v_1, v_2, v_3 and v_4 are the non-negative real numbers to be determined. The derivative of V is; $\dot{V} = \dot{L_h} + v_1\dot{A_h} + v_2\dot{Re_h} + v_3\dot{L_v} + v_4\dot{A_v}$. $\dot{V} = [\frac{b_{vh}\gamma A_v S_h}{N} - (1-a)\alpha L_h - a\alpha L_h - \mu_{\mathbf{h}} L_h] + v_1[(1-a)\alpha L_h + \frac{b_{vh}\gamma\Upsilon R_h A_h}{N} - \phi_{\mathbf{h}} A_h - (\omega_{\mathbf{h}} + \mu_{\mathbf{h}})A_h - \sigma_{\mathbf{h}} A_h] + v_2[\phi_{\mathbf{h}} A_h - \lambda_{\mathbf{h}} Re_h - (\omega_{\mathbf{h}} + \mu_{\mathbf{h}})Re_h] + v_3[\frac{\gamma b_{hv} S_v A_h}{N} - \rho L_v - (\mu_{\mathbf{v}} + \delta_{\mathbf{v}})L_v] + v_4[\rho L_v - (\mu_{\mathbf{v}} + \delta_{\mathbf{v}})A_v]$.

It is observed that $\dot{V} < 0$ if $R_0 < 1$, and $\dot{V} = 0$ if $L_h = A_h = Re_h = L_h = A_h = 0$. Thus, the maximal compact set $[(S_h, L_h, I_h, Re_h, S_v, L_v, A_v) \epsilon \mathbf{\Omega} : \dot{V} = 0]$, when $R_0 \leq 1$ is the singleton E_0. Following Lasalle's Invariance Principle by LaSsalle et al. [29], Pontryagin et al. [31], E_0 is globally asymptotically stable if $R_0 \leq 1$.

Theorem: If $R_0 > 1$, E_0, is globally asymptotically stable in $\mathbf{\Omega}$.

Proof: The malaria endemic equilibrium exists if $R_0 > 1$. Let us now look at a Lyapunov function described; $L = (S_h - S_h^* - S_h^* \ln S_h) + A(L_h - L_h^* - L_h^* \ln L_h) + B(A_h - A_h^* - A_h^* \ln A_h) + C(Re_h - Re_h^* - Re_h^* \ln Re_h) + D(R_h - R_h^* - R_h^* \ln R_h) + E(S_v - S_v^* - S_v^* \ln S_v) + F(L_v - L_v^* - L_v^* \ln L_v) + G(A_v - A_v^* - A_v^* \ln A_v)$. Where A, B, and C are real numbers to be evaluated. The derivative L is, $\dot{L} = (1 - \frac{S_h^*}{S_h})\dot{S_h} + A(1 - \frac{L_h^*}{L_h})\dot{L_h} + B(1 - \frac{A_h^*}{A_h})\dot{A_h} + C(1 - \frac{Re_h^*}{Re_h})\dot{Re_h} + D(1 - \frac{R_h^*}{R_h})\dot{R_h} +$

$E(1-\frac{S_v^*}{S_v})\dot{S}_v + F(1-\frac{L_v^*}{L_v})\dot{L}_v + G(1-\frac{A_v^*}{A_v})\dot{A}_v$. The derivative L is, $\dot{L} = (1-\frac{S_h^*}{S_h})[\mathbf{\Lambda_h} + \tau_\mathbf{h} R_h - \frac{b_{vh}\gamma A_v S_h}{N} - \mu_\mathbf{h} S_h] + A(1-\frac{L_h^*}{L_h})[\frac{b_{vh}\gamma A_v S_h}{N} - (1-a)\alpha L_h - a\alpha L_h - \mu_\mathbf{h} L_h] + B(1-\frac{A_h^*}{A_h})[(1-a)\alpha L_h + \frac{b_{vh}\gamma \Upsilon R_h A_v}{N} - \phi_\mathbf{h} A_h - (\omega_\mathbf{h}+\mu_\mathbf{h})A_h - \sigma_\mathbf{h} A_h] + C(1-\frac{Re_h^*}{Re_h})[\phi_\mathbf{h} A_h - \lambda_\mathbf{h} Re_h - (\omega_\mathbf{h}+\mu_\mathbf{h})Re_h] + D(1-\frac{R_h^*}{R_h})[\sigma_\mathbf{h} A_h + \lambda_\mathbf{h} Re_h + a\alpha L_h - \frac{b_{vh}\gamma\Upsilon R_h A_v}{N} - \tau_\mathbf{h} R_h - \mu_\mathbf{h} R_h] + E(1-\frac{S_v^*}{S_v})[\mathbf{\Lambda_v} - \frac{\gamma b_{hv} S_v A_h}{N} - (\mu_\mathbf{v}+\delta_\mathbf{v})S_v] + F(1-\frac{L_v^*}{L_v}[\frac{\gamma b_{hv} S_v A_h}{N} - \rho L_v - (\mu_\mathbf{v}+\delta_\mathbf{v})L_v] + G(1-\frac{A_v^*}{A_v})[\rho L_v - (\mu_\mathbf{v}+\delta_\mathbf{v})A_v]$. The remaining terms are negative and $-\mu_\mathbf{h}\omega[\frac{S_h - S_h^*}{S_h}]^2 \leq 0$ and $[\frac{S_h - S_h^*}{S_h}]^2 \leq 0$. The largest invariant set where $\dot{L} = 0$ is the singleton of Endemic Equilibrium. Endemic Equilibrium is therefore globally asymptotically stable with respect to the invariant set $\mathbf{\Omega}$ by the Lasalle's Invariance Principle by Alverson et al. [29], and Pontryagin et al. [30].

3.9. Sensitivity Analysis

Sensitivity of the malaria model parameters with respect to the basic reproductive number is investigated. This is done to identify the most sensitive parameters in the model. Normalized forward sensitivity index approach is used by Powell et al. [31]. It is differentiable with respect to the given model parameters, ξ defined as; $Y_\xi^{R_0} = \frac{\partial R_0}{\partial \xi} \times \frac{\xi}{R_0}$. where ξ is an arbitrary parameter in the expression of R_0. From our calculation, we found out that, the most sensitive parameters were $\delta_\mathbf{v}$, α, Υ, $\sigma_\mathbf{h}$, ϕ, $\tau_\mathbf{h}$, and $\lambda_\mathbf{h}$.

Table 1. *Values of Variables and their references.*

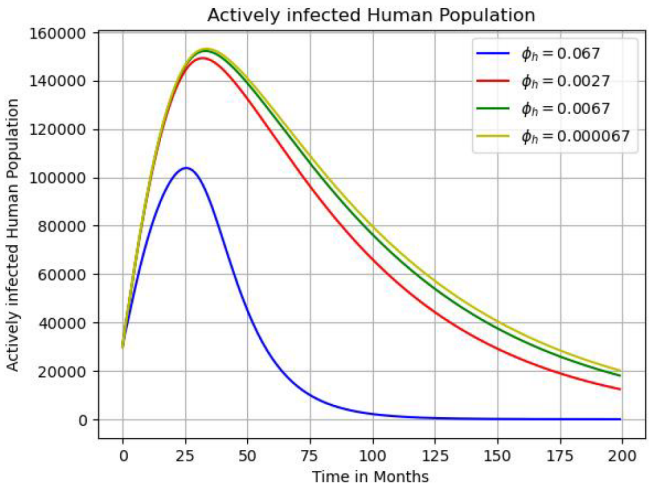
Variables	Value	Reference
$\mathbf{\Lambda_h}$	0.0421	[33, 34]
$\mathbf{\Lambda_v}$	0.05	[35]
$\mu_\mathbf{h}$	0.0356	[36]
$\mu_\mathbf{v}$	0.05	[37]
b_{vh}	2.5	[38]
b_{hv}	0.993	[38]
γ	0.25	[39]
$(1-a)$	0.005	Assumed
a	0.005	Assumed
α	0.05	Assumed
$\omega_\mathbf{h}$	7.8×10^{-6}	[40]
$\sigma_\mathbf{h}$	0.0095	Assumed
$\phi_\mathbf{h}$	0.067	Assumed
$\lambda_\mathbf{h}$	0.075	Assumed
Υ	100	Assumed
$\tau_\mathbf{h}$	0.03	[41]
$\delta_\mathbf{v}$	0.5	Assumed
N	120000	Assumed

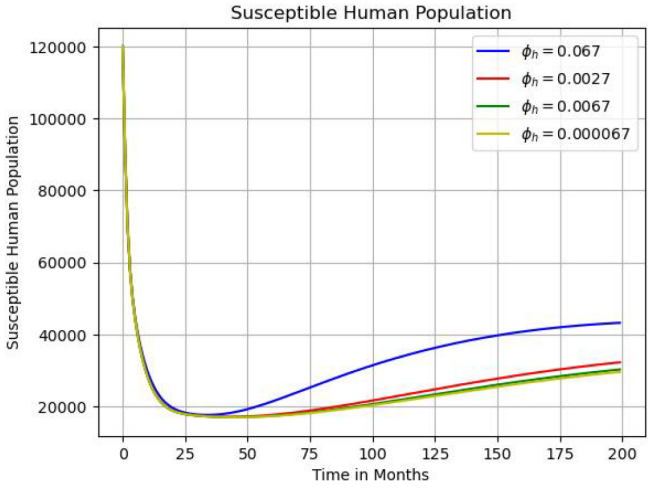
4. Numerical Simulations

Model parameter values in Table 1 are used for the numerical illustrations. The parameter values in this study is from the existing literature, and the initial conditions are assumed to be; $S_h = 120000, L_h = 85000, A_h = 30000, Re_h = 5000, R_h = 2000, S_v = 100000, L_v = 50000$, and $I_v = 24000$.

4.1. Impact of Immunity on Human Population

In this sub-section, we are going to do simulations of impact of Immunity on the human population dynamics.





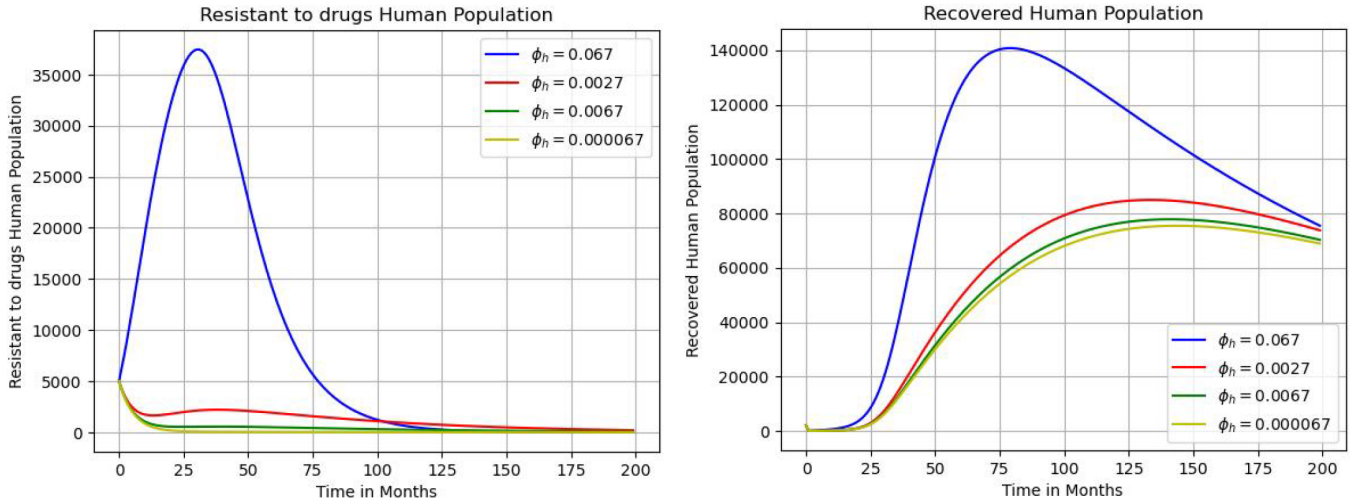


Figure 2. *Shows graphs of Uncomplicated malaria Infections, Drug resistance, and recovered Human Populations on varying values of ι against time in months that is the higher the rate of immunity, the lower the rate of infections.*

4.2. Effect of Reinfections on Human Population

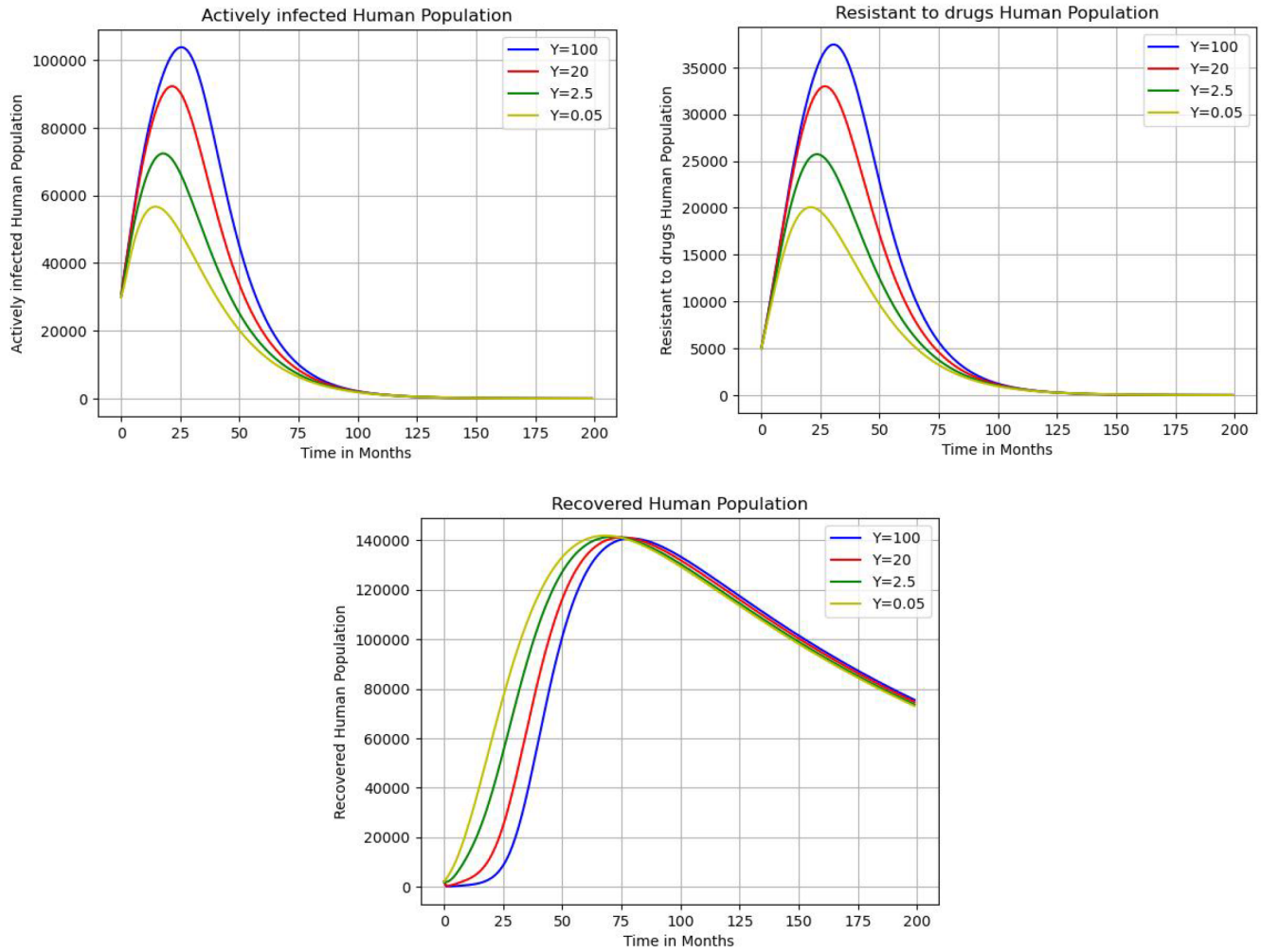


Figure 3. *Graphs of human population on varying values of γ against time in months that is the higher the rate of reinfections, the higher the number of those who are infected with malaria, and the lower the recoveries. This implies that there is need to stop the reinfections by reducing the mosquito bites.*

4.3. Effect of Loss of Immunity on Human Population

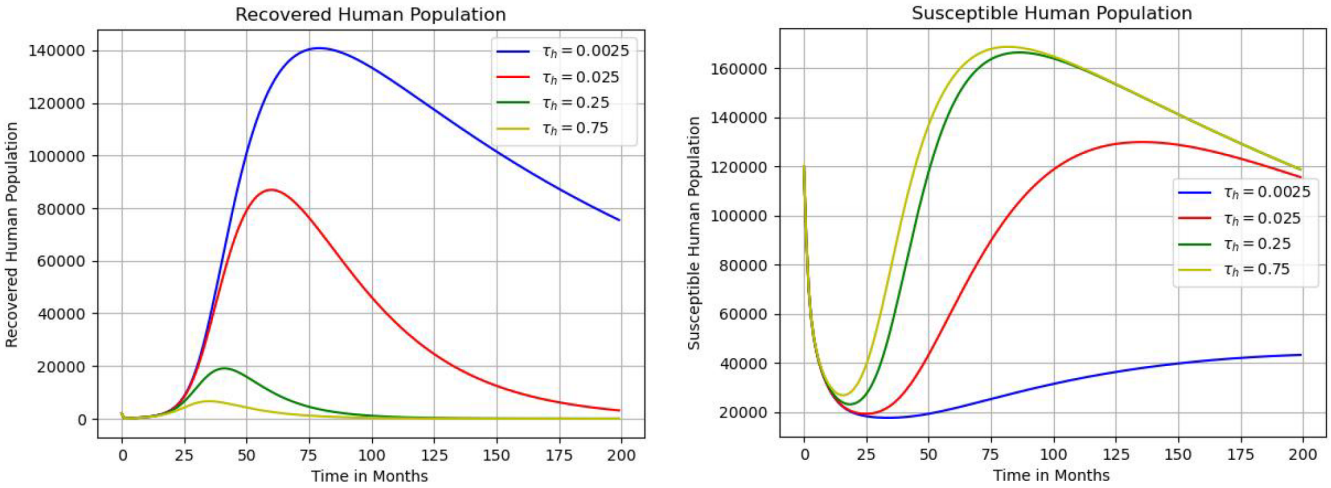


Figure 4. *Graphs of human population on varying values of τ_h against time in months. They show that the higher the rate of loss of immunity, the higher the number of those who are susceptible to malaria, and the lower the recoveries. This implies that there is need to improve immunity so that the susceptible humans can reduce, and the recoveries to increase.*

4.4. Effect of Use of All the Control Strategies; Sensitization on Vector Control; α, Vector Control; δ_v, High Immunity; σ_h, and Intensive Treatment; Λ_h on Human and Mosquito Populations

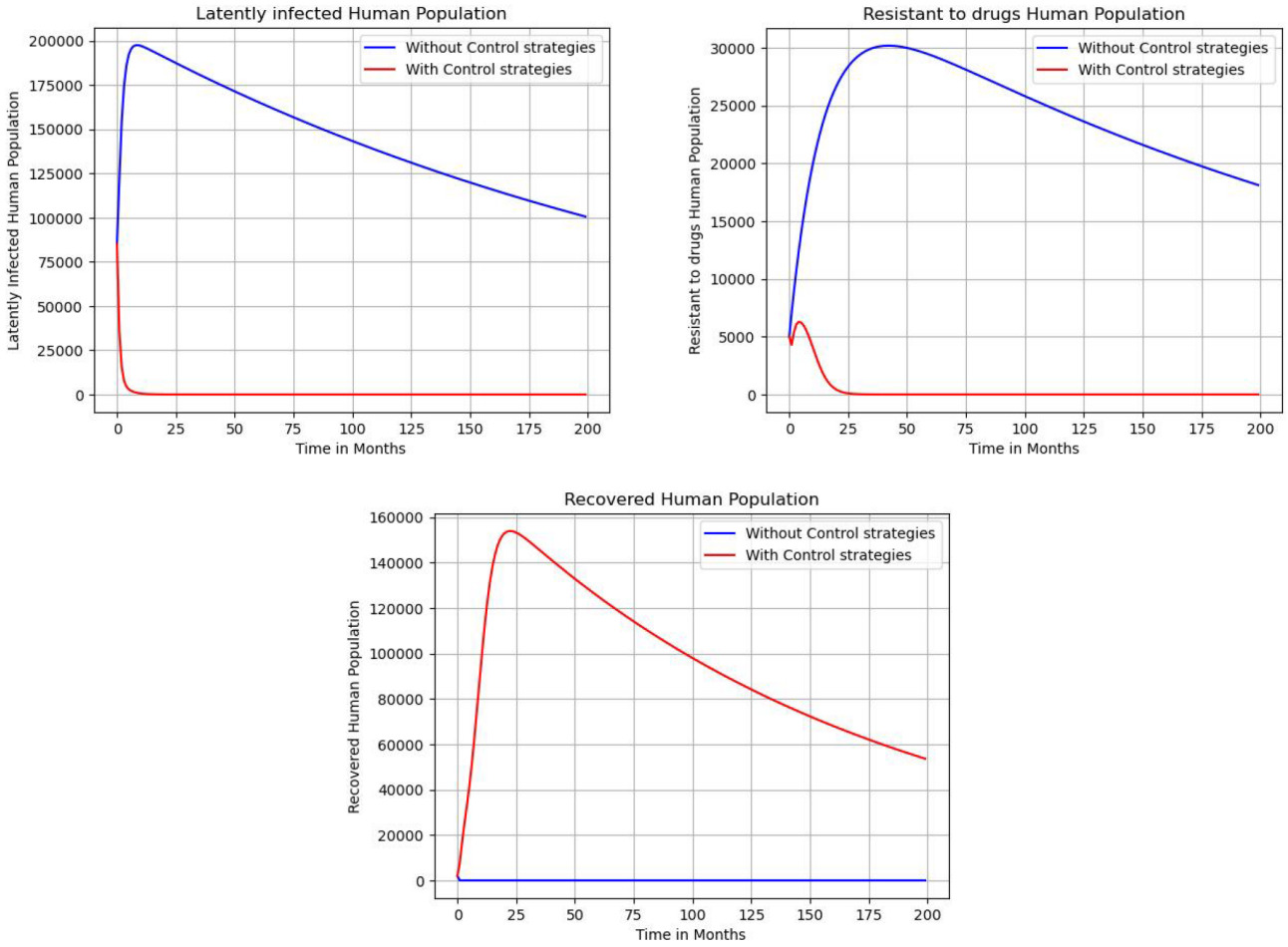


Figure 5. *Graphs of human population with control strategies; $\sigma_h = \lambda_h = a\alpha = \delta_v = 1.25$ and without control strategies when; $\sigma_h = \lambda_h = a\alpha = \delta_v = 0$.*

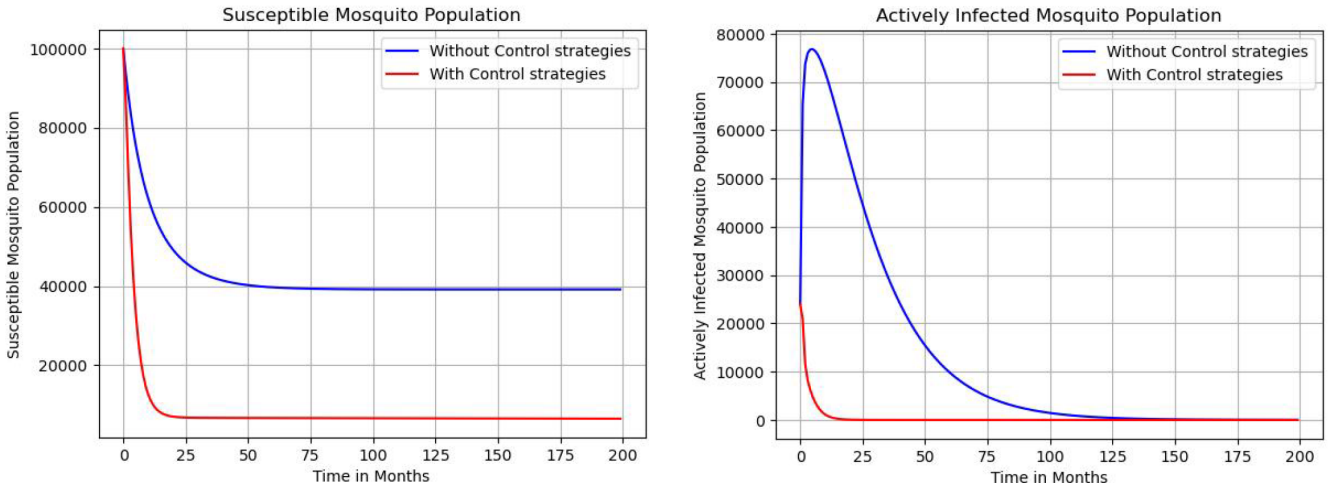


Figure 6. *Graphs of Mosquito Population with control strategies;* $\sigma_h = \lambda_h = a\alpha = \delta_v = 1.25$ *and without control strategies when;* $\sigma_h = \lambda_h = a\alpha = \delta_v = 0$.

5. Discussion

Malaria mathematical model dynamics is an important tool in understanding the malaria disease transmissions, and the process of making the right decisions for the control strategies over many years by Castillo-Chavez et al. [42]. In this study, a deterministic malaria mathematical model was developed that incorporated immunity, reinfection, antimalarial drug resistance, aggressive treatment, and awareness on vector control. Most of the analysis was done numerically because the malaria mathematical model was complex. Some parameter values were estimated and others were from the existing literature. Positivity and boundedness was conducted to prove that all state variables were non negative and satisfy the initial conditions. Disease free equilibrium and the basic reproductive number were carried out. The next generation matrix method was used to calculate the basic reproductive number. Local stability and global stability at the disease free equilibrium and the malaria endemic equilibrium points was also calculated. Sensitivity analysis was carried out to find out the most influential parameters on the basic reproductive number. Global stability at the endemic equilibrium point was carried out and found out that it was globally asymptotically stable if $R_0 < 0$.

6. Conclusion

According to this study, low immunity, reinfections, loss of immunity, and lack of awareness on vector control led to increased malaria infections, and decreased recoveries. Several intervention strategies helped reduce the infections and increase recoveries. These intervention strategies include; awareness on vector control, aggressive treatment on the human population resistant to drugs and weak immunity, vector control and high immunity. This study concluded that when all these intervention strategies are done at once, the malaria disease may die out and the recoveries may increase. This study did not consider immunity, reinfection, and vector control as a parameter variable with time, in future studies, they should be considered as compartments to obtain better results. Additionally, this study did not consider cost effectiveness and optimal control. In future, a research should be carried out that will consider optimal control that will minimize cost in order to eliminate malaria. This study will be useful to the government and non governmental stakeholders to ensure awareness on vector control is done to eliminate malaria infections.

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Author Contributions

Grace Maithya: Writing original draft, Conceptualization, Methodology, Analysis and Simulation
Virginia Kitetu: Review, Editing, and Supervision
Isaac Okwany: Review, Editing, and Supervision

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Conflicts of Interest

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