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ANALYSIS OF A TEN COMPARTMENTAL MATHEMATICAL MODEL OF MALARIA TRANSMISSION

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Abstract. In this paper, we analyse a ten compartmental mathematical model for malaria transmission which include non-immune and semi-immune humans. We obtain an explicit formula for the basic reproduction number R_0 which is a function of the weight of the transmission from non-immune humans to mosquito and from non-immune humans to mosquito, and the weight of the transmission from semi-immune humans to mosquito and from mosquito to semi-immune humans. The model outcome confirms that the disease free equilibrium (DFE) is globally asymptotically stable when $\mathcal{R}_0 < 1$ and and it is unstable when $\mathcal{R}_0 > 1$. We also prove that the endemic Equilibrium (EE) is unstable $\mathcal{R}_0 < 1$ and it is globally asymptotical stable when $\mathcal{R}_0 > 1$. We discuss the possibility of a control for malaria transmission throughout a definite sub- group such as non-immune or semi-immune or mosquitoes.

Keywords: Non-immune, semi-immune, disease free equilibria.
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1 Introduction

Malaria is a parasite disease caused and transmitted by the bite of an infected female anopheles mosquito; it's a result of infection with one or more Plasmodium species, pathogenic agents of 'protozoan' types. Mathematical models provide the means to generate evidence-based information on malaria disease control and play an important role in understanding the dynamics of the disease. A major extension of mathematical models is described in Macdonald's 1957 book Macdonald (1957). In a later review, Anderson and May in Anderson and May (1991) revisited many of the ideas discussed by Aron and May Aron and May (1982), they looked at different control strategies, discussing the effects of a vaccine and the reduction of transmission rates on the malaria age-prevalence profile of the human population. Other reviews on mathematical modelling in malaria include in Ducrot et al. (2009). They surveyed various data sets to statistically approximate parameters such as inoculation rates, rates of recovery and loss of immunity in humans, human-biting rates of mosquitoes and infectivity and susceptibility of humans and mosquitoes. With all these and others models at hand, it is not a trivial matter to infer the crucial features of the disease, and get a coherent understanding of the development of the models from interactions between the vector and host. An attempt has been made here to elaborate the evolution of these models by considering some representative mathematical models

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that include the increasing complexities of host-vector-parasite interactions. The major advantage of these early models was to provide a suitable control strategy through the transmission threshold criterion, which is based on the reproductive capacity of the parasite, and termed as basic reproductive number R_0.In this paper, we formulate a ten compartmental mathematical model of malaria transmission, which includes include non-immune and semi-immune humans. We investigate the possibility of a control of malaria through one of the three host type by calculating the control reproduction number specific to each host type.

The rest of paper is organised as follows: in section 1, we formulate the model and we provide. In section 3, we prove the wellposedness of the model. We determine the equilibria and calculate the reproduction number of the model in section 4 and in section 5 we analyse the stability of the disease free equilibrium and the endemic equilibrium. In section 6, some numerical analysis are done. We give concluding remarks in section 7.

2 Formulation of the Model

In order to analysis the ten compartmental mathematical modelling of malaria transmission. We divided the human population in to two groups. The first group named non-immune human, includes every humans who do not have resistance against malaria. The second group named semi-immune human is the class of humans who have at least acquired immunity in their life even if they lost it. Since the concept of natural immunisation is based on memory, the second group is supposed to be less vulnerable. We assume that, the human population, as whole, is subdivided into susceptible non-immune (S_e), exposed non-immune (E_e), infectious non-immune (I_e), susceptible semi-immune (S_a), exposed semi-immune (E_a), infectious semi-immune (I_a), recovery semi-immune (R_a). Thus, the total human population $N_h(t) = S_e + E_e + I_e + S_a + S_a + E_a + I_a + R_a$. We sub-divide the mosquito population into three subclasses: susceptible mosquitoes S_v, exposed mosquitoes E_v and infectious mosquitoes I_v. The mosquitoes stay infectious for life and do not recover. Thus, the total mosquitoes population $N_v(t) = S_v+E_v+I_v$. We assume that the infectious non-immune humans develop resistance and enter the recovered class. Disease does not kill the mosquitoes. There is no malaria transmission between humans and there is no malaria transmission between mosquitoes. The natural death rate and birth rate are considered. There are infection transmissions from I_v to S_e, from I_v to S_a, from I_e to S_v, and from I_a to S_v. We denote the non-immune human population by e, the semi-immune human population by a, and the mosquito population by v throughout this paper. When a susceptible mosquito bites an infectious non-immune humans and the susceptible mosquito could be become exposed moves to exposed mosquitoes, or when an infectious mosquito bites a susceptible humans either non-immune or semi-immune humans the parasite enters to human and the susceptible humans moves to their own exposed group. The non-immune susceptible human class is increased by non-immune human by birth through birth at per capita birth at rate of λ_e, and leave the S_e class with the rate of β_e and μ_h. The non-immune humans E_e class is increased by new infectious at a rate of β_e and is decreased ar rate of γ_e and μ_h by becoming infectious and death respectively. The population enter the I_e class at a rate γ_e, and leaves at rates α_e and μ_h. The semi-immune human enters into the susceptible semi-immune human class through birth at a rate $(\lambda_h - \lambda_e)$ and from recovery of humans at the rate of Ω_a, and leave the class S_a with the rate of β_a and μ_h. The semi-resistance humans enter the E_a class with the rate of β_a and leave the E_a class with the rate of γ_a and μ_h. Exposed semi-immune humans enter the I_a class at a rate γ_a, and leave the I_a class at rates α_a and μ_h. The infectious semi-immune humans recover at a rate α_a to enter the R_a class and they leave the class R_a at rates μ_h and Ω_a. Mosquitoes are recruited into the susceptible class by birth at a per capita birth rate of λ_v and leave the class S_v at rates β_v and μ_v. Susceptible mosquitoes enter to the class E_v with β_v and leave it with rates γ_v and μ_v. Exposed mosquitoes enter the infectious class at a rate γ_v and leave the infectious class at a rates μ_v. We assume that all the parameters are positive.

Table 1: The explanation of state variables for malaria model of ten dimensional.

Variables	The explanation of the state variables
S_e	Susceptible non-immune humans .
E_e	Exposed non- immune humans.
I_e	Infectious non-immune humans.
S_a	Susceptible semi-immune humans.
E_a	Exposed semi-immune humans.
I_a	Infectious semi-immune humans.
R_a	Recovery of humans.
S_v	The susceptible mosquitoes.
E_v	exposed mosquitoes.
I_v	Infectious mosquitoes.

The state variables and the parameter variables for our model are summarised in brief in Table (1) and (2) respectively. Using the standard incidence as in the model Ngwa (2004), we define and notify the infection incidences as: $\beta_e = \Upsilon\varphi_{ve}I_v$ is the infection incidences from mosquitoes to non-immune humans, $\beta_a = \Upsilon\varphi_{va}I_v$ is the infection incidences from mosquitoes to semi-immune humans and $\beta_v = (\varphi_{ev}I_e + \varphi_{av}I_a)\,\Upsilon$ is the disease occurrence from semi-immune humans or non-immune humans to mosquitoes, then β_v is given by the amount of the power of disease from I_a and I_e. From the compartmental representation in Figure 1, we derive the following system differential equations:

$$\begin{aligned}
\frac{dS_e}{dt} &= \lambda_e N_h - S_e\left(\Upsilon\varphi_{ve}I_v + \mu_h\right), && (1)\\
\frac{dE_e}{dt} &= \Upsilon I_v\varphi_{ve}S_e - E_e\left(\gamma_e + \mu_h\right), && (2)\\
\frac{dI_e}{dt} &= \gamma_e E_e - I_e\left(\alpha_e + \mu_h\right), && (3)\\
\frac{dS_a}{dt} &= \left(\lambda_h - \lambda_e\right)N_h + \Omega_a R_a - S_a\left(\Upsilon\varphi_{va}I_v + \mu_h\right), && (4)\\
\frac{dE_a}{dt} &= S_a\Upsilon\varphi_{va}I_v - E_a\left(\gamma_a + \mu_h\right), && (5)\\
\frac{dI_a}{dt} &= \gamma_a E_a - I_a\left(\mu_h + \alpha_a\right), && (6)\\
\frac{dR_a}{dt} &= \left(\alpha_a + \alpha_e\right)I_a - \left(\Omega_a + \mu_h\right)R_a, && (7)\\
\frac{dS_v}{dt} &= \lambda_v N_v - S_v\varphi_{ev}I_e\Upsilon - S_v\mu_v - \varphi_{av}S_v\Upsilon I_a, && (8)\\
\frac{dE_v}{dt} &= \left(\varphi_{ev}I_e + \varphi_{av}I_a\right)\Upsilon S_v - E_v\left(\gamma_v + \mu_v\right), && (9)\\
\frac{dI_v}{dt} &= \gamma_v E_v - I_v\mu_v. && (10)
\end{aligned}$$

with the initial positive conditions

$$\begin{aligned}
S_e\left(0\right) &= S_{e0}, E_e\left(0\right) = E_{e0}, I_e\left(0\right) = I_{e0}, S_a\left(0\right) = S_{a0}, E_a\left(0\right) = E_{a0},\\
I_a\left(0\right) &= I_{a0}, R_a\left(0\right) = R_{a0}, S_v\left(0\right) = S_{v0}, E_v\left(0\right) = E_{v0}, I_v\left(0\right) = I_{v0}
\end{aligned}$$

Table 2: The explanation of parameters for malaria model of ten dimensional.

Parameter	The explanation of the parameter
α_e	The rate at which non-immune human progress to recovery.
λ_h	Per capita birth rate of human.
γ_e	The rate at which non-immune human progress to infective.
λ_e	A per capita birth rate of non-immune human.($\lambda_h \geq \lambda_e$)
λ_v	A per capita birth rate of mosquitoes.
α_a	The rate of the infective non-immune human progress to recovery.
γ_a	The rate at which semi-immune human progress to infective.
γ_v	The rate at which mosquito progress to infective.
λ_h	A per capita delivery rate of human.
Ω_a	The rate at which recovered humans progress to susceptible.
Υ	The number of bites
μ_h	The death rate of humans.
μ_v	The death rate of of mosquitoes.
φ_{av}	The probability that the infectious will transfer from I_a to the S_v.
φ_{ev}	The probability that the infectious will transfer from I_e to the S_v.
φ_{va}	The probability that the infectious will transfer from I_v to S_a .
φ_{ve}	The probability that the infectious will transfer from I_v to S_e.

3 Existence of the solution

Theorem 1. *The malaria model* (1)-(10) *has a unique globally defined solution, which remains in the domain* $\Omega = \Omega_1 \times \Omega_2$ *for all time* $t \geq 0$, *where*

$$\Omega_1 = \left\{ \left(\frac{S_e}{N_h}, \frac{E_e}{N_h}, \frac{I_e}{N_h}, \frac{S_a}{N_h}, \frac{E_a}{N_h}, \frac{I_a}{N_h}, \frac{R_a}{N_h}, \frac{S_v}{N_v}, \frac{E_v}{N_v}, \frac{I_v}{N_v} \right) \in [0,1]^{10} : 0 \leq \frac{S_v}{N_v} + \frac{E_v}{N_v} + \frac{I_v}{N_v} \leq 1 \right.$$
$$\left. \text{and } \ 0 \leq \frac{S_a}{N_h} + \frac{E_a}{N_h} + \frac{I_a}{N_h} + \frac{R_a}{N_h} + \frac{S_e}{N_h} + \frac{E_e}{N_h} + \frac{I_e}{N_h} \leq 1 \right\}$$
$$\Omega_2 = \left\{ (N_h, N_v) \in R^2 : 0 < N_h \leq \frac{\lambda_h - \mu_h + \sqrt{(\lambda_h - \mu_h)^2 + 4\mu_h}}{\mu_h} \text{ and } 0 < N_v \leq \frac{\lambda_v - \mu_v}{\mu_v} \leq 1 \right\}$$

Proof. The local existence of the solution follows from the regularity of the function $g = (g_1, g_2, .., g_{10})$, where $\frac{dx_i}{dt} = g_i(t), i = 1, 2, ..., 10$, are continuous differentiable in the domain Ω_1. We first show that Ω_1 is forward-invariant for all $(N_h, N_v) \in \Omega_2$. It is easy to see that if $x_i = 0$ then $\frac{dx_i}{dt} = g_i(t) \geq 0, i = 1, 2, ..., 10$. It follows that if

$$\frac{S_v}{N_v} + \frac{E_v}{N_v} + \frac{I_v}{N_v} = 0 \Rightarrow \frac{d}{dt}\left(\frac{S_v}{N_v}\right) + \frac{d}{dt}\left(\frac{E_v}{N_v}\right) + \frac{d}{dt}\left(\frac{I_v}{N_v}\right) \geq 0$$

and if $\frac{S_a}{N_h} + \frac{E_a}{N_h} + \frac{I_a}{N_h} + \frac{R_a}{N_h} + \frac{S_e}{N_h} + \frac{E_e}{N_h} + \frac{I_e}{N_h} = 0$ then $\frac{S_a}{N_h} + \frac{E_a}{N_h} + \frac{I_a}{N_h} + \frac{R_a}{N_h} + \frac{S_e}{N_h} + \frac{E_e}{N_h} + \frac{I_e}{N_h} \geq 0$. Moreover, if $\frac{S_v}{N_v} + \frac{E_v}{N_v} + \frac{I_v}{N_v} = 1$ then $\frac{d}{dt}\left(\frac{S_v}{N_v}\right) + \frac{d}{dt}\left(\frac{E_v}{N_v}\right) + \frac{d}{dt}\left(\frac{I_v}{N_v}\right) = -\lambda_v < 0$ and if $\frac{S_a}{N_h} + \frac{E_a}{N_h} + \frac{I_a}{N_h} + \frac{R_a}{N_h} + \frac{S_e}{N_h} + \frac{E_e}{N_h} + \frac{I_e}{N_h} = 1$ then $\frac{d}{dt}\left(\frac{S_a}{N_h}\right) + \frac{d}{dt}\left(\frac{E_a}{N_h}\right) + \frac{d}{dt}\left(\frac{I_a}{N_h}\right) + \frac{d}{dt}\left(\frac{R_a}{N_h}\right)$ $+ \frac{d}{dt}\left(\frac{S_e}{N_h}\right) + \frac{d}{dt}\left(\frac{E_e}{N_h}\right) + \frac{d}{dt}\left(\frac{I_e}{N_h}\right) = -\frac{\beta_a R_a}{N_h} < 0$. Now, we show that Ω_2 is forward invariant for all

$$\left(\frac{S_a}{N_h}, \frac{E_a}{N_h}, \frac{I_a}{N_h}, \frac{R_a}{N_h}, \frac{S_e}{N_h}, \frac{E_e}{N_h}, \frac{I_e}{N_h}, \frac{S_v}{N_v}, \frac{E_v}{N_v}, \frac{I_v}{N_v}\right) \in \Omega_1,$$

then $\frac{dN_h}{dt} > 0$ if $\lambda_h > \mu_h$ and $\frac{dN_v}{dt} > 0$ if $\lambda_v > \mu_v$. It is easy to see that

$$\lim_{t\to\infty} \sup N_v(t) \leq \frac{\lambda_v - \mu_v}{\mu_v} \text{ and } \lim_{t\to\infty} \sup N_h(t) \leq \frac{\lambda_h - \mu_h + \sqrt{(\lambda_h - \mu_h)^2 + 4\mu_h}}{\mu_h}$$

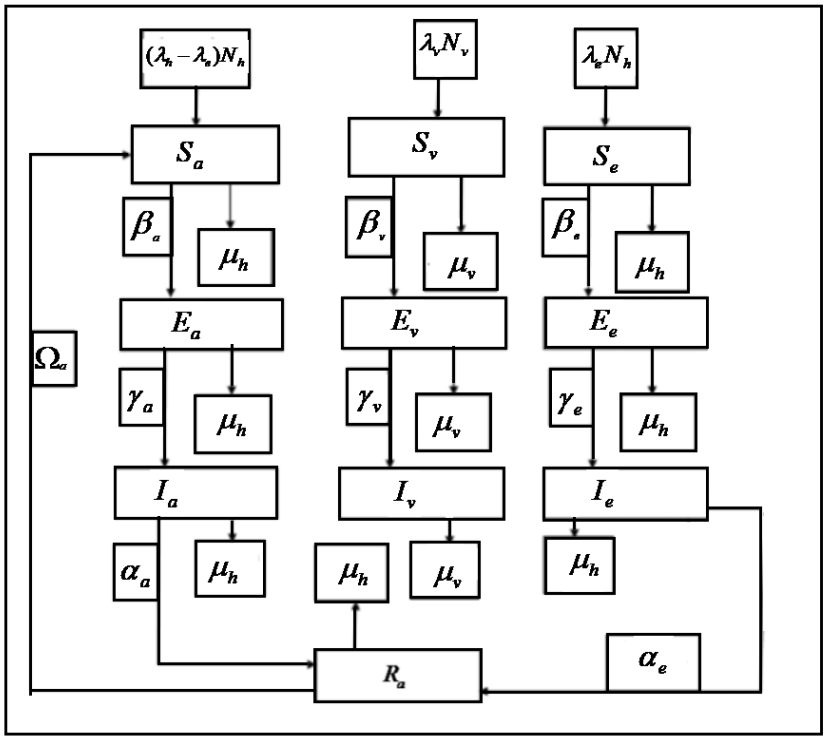

Figure 1: Flow diagram of ten compartmental model

We conclude that the solutions of (1)- (10) exist globally in a domain Ω, then it is epidemiologically and mathematically well-posed.
Let $X(t) = (S_e(t), E_e(t), I_e(t), S_a(t), E_a(t), I_a(t), R_a(t), S_v(t), E_v(t), I_v(t))$ and

$$\phi : \Gamma \to \Psi \text{ and } X :\mapsto X^{'}$$

such that $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7, \phi_8, \phi_9, \phi_{10})$, where

$$\phi_1 = \frac{dS_e}{dt} = \lambda_e N_h - S_e\left(\Upsilon\varphi_{ve} I_v + \mu_h\right), \tag{11}$$

$$\phi_2 = \frac{dE_e}{dt} = \Upsilon I_v \varphi_{ve} S_e - E_e\left(\gamma_e + \mu_h\right), \tag{12}$$

$$\phi_3 = \frac{dI_e}{dt} = \gamma_e E_e - I_e\left(\alpha_e + \mu_h\right), \tag{13}$$

$$\phi_4 = \frac{dS_a}{dt} = \left(\lambda_h - \lambda_e\right) N_h + \Omega_a R_a - S_a\left(\Upsilon\varphi_{va} I_v + \mu_h\right), \tag{14}$$

$$\phi_5 = \frac{dE_a}{dt} = S_a \Upsilon\varphi_{va} I_v - E_a\left(\gamma_a + \mu_h\right), \tag{15}$$

$$\phi_6 = \frac{dI_a}{dt} = \gamma_a E_a - I_a\left(\mu_h + \alpha_a\right), \tag{16}$$

$$\phi_7 = \frac{dR_a}{dt} = \left(\alpha_a + \alpha_e\right) I_a - R_a\left(\Omega_a + \mu_h\right), \tag{17}$$

$$\phi_8 = \frac{dS_v}{dt} = \lambda_v N_v - S_v\left(\left(\varphi_{ev} I_e + \varphi_{av} I_a\right)\Upsilon + \mu_v\right), \tag{18}$$

$$\phi_9 = \frac{dE_v}{dt} = \left(\varphi_{ev} I_e + \varphi_{av} I_a\right)\Upsilon S_v - E_v\left(\gamma_v + \mu_v\right), \tag{19}$$

$$\phi_{10} = \frac{dI_v}{dt} = \gamma_v E_v - I_v \mu_v. \tag{20}$$

Then, (1) - (10) can be written in the form of the following

$$X^{'}(t) = \phi(X(t)) : X(0) = (S_{e0}, E_{e0}, I_{e0}, S_{a0}, E_{a0}, I_{a0}, R_{a0}, S_{v0}, E_{v0}, I_{v0}) \in \Gamma$$

We follow the proof done in Chitnis et al. (2006). Suppose that there exists t_1 and t^* with $t_1 < t^*$ such that $S_e(t_1) = 0$, $\frac{dS_e(t)}{dt} < 0$ in (t_1, t^*) where all the ten compartments are positives. Then from (1),

$$\frac{dS_e(t)}{dt} = \lambda_e N_h - S_e(t)\,\Upsilon I_v(t)\,\varphi_{ev} - S_e(t)\,\mu_h \geq \lambda_e N_h \geq 0$$

which is contradiction. Hence, $S_e(t) \geq 0$. Suppose that there exist

$$t_1 = \mathrm{Sup}\left\{t > 0 : S_a, I_a, E_a, R_a, S_e, I_e, R_e, S_v, E_v, I_v > 0\right\}.$$

Then from equation (2), we get

$$\frac{d}{dt}\left(E_e(t)e^{(\gamma_e+\mu_h)t}\right) = \left(\Upsilon I_v(t)S_e(t)\right)e^{(\gamma_e+\mu_h)t}. \tag{21}$$

Integrating equation (21) from 0 to t_1, we have

$$E_e(t)e^{(\gamma_e+\mu_h)t} = E_{e0} + \int_0^{t_1}\left(\Upsilon I_v(\theta)S_e(\theta)\varphi_{ve}\right)e^{(\gamma_e+\mu_h)\theta}d\theta. \tag{22}$$

Multiply both sides of (22) by $e^{-(\gamma_e+\mu_h)t_1}$, then we have get (23)

$$\begin{aligned} E_e(t)(t_1) &= (E_{e0})\,e^{-(\gamma_e+\mu_h)t_1} + e^{-(\gamma_e+\mu_h)t_1} \\ &\quad \int_0^{t_1}\left(\Upsilon I_v(\theta)S_e(\theta)\varphi_{ve}\right)e^{(\gamma_e+\mu_h)\theta}d\theta \geq 0. \end{aligned} \tag{23}$$

Since $E_e(t) > 0$ for all $t \geq 0$, then from equation (3) we have

$$\begin{aligned} &\Rightarrow \quad \frac{dI_e(t)}{dt} \geq -(\alpha_e + \mu_e)\,I_e \\ &\Rightarrow \quad \frac{dI_e(t)}{I_e} \geq -(\alpha_e + \mu_e)\,dt \\ &\Rightarrow \quad I_e(t) \geq e^{-(\alpha_e+\mu_e)t} \geq 0. \end{aligned} \tag{24}$$

Let us show that $S_a(t) \geq 0$ for all $t \geq 0$. Suppose that there exists t_1 and t^* with $t_1 < t^*$ such that $S_a(t_1) = 0$, $\frac{dS_a(t)}{dt} \leq 0$ and from equation (4),

$$\frac{dS_a(t)}{dt} = (\lambda_e - \lambda_h)\,N_h + \Omega_a R_a(t) - S_a\Upsilon\varphi_{va}I_v(t) - S_a(t)\mu_h \geq 0.$$

which is contradiction. Hence, $S_a(t) \geq 0$, $\forall t \geq 0$. From (5)

$$\frac{d}{dt}\left(E_a(t)e^{(\gamma_a+\mu_h)t}\right) = \left(S_a(t)\Upsilon I_v(t)\varphi_{va}\right)e^{(\gamma_a+\mu_h)t}. \tag{25}$$

Integrating equation (25) from 0 to t_1, we have

$$\left(E_a(t_1)e^{(\gamma_a+\mu_h)t_1}\right) = E_{a0} + \int_0^{t_1}\left(S_a(t)\Upsilon I_v(\theta)\varphi_{va}\right)e^{(\gamma_a+\mu_h)\theta}d\theta. \tag{26}$$

Multiply both sides of equation (26) by $e^{(\gamma_a+\mu_h)t_1}$, then we have get

$$E_a(t_1) = (E_{a0})\,e^{(\gamma_a+\mu_h)t_1} + e^{(\gamma_a+\mu_h)t_1}\int_0^{t_1}\left(S_a(t)\Upsilon I_v(\theta)\varphi_{va}\right)e^{(\gamma_a+\mu_h)\theta}d\theta \geq 0.$$

Hence, $E_a(t) \geq 0$ for all $t \geq 0$. Since $E_a(t) > 0$ for all $t \geq 0$ and from (6)

$$\begin{aligned} \Rightarrow \quad & \frac{dI_a(t)}{dt} \geq -I_a\left(\alpha_a + \mu_h\right) \\ \Rightarrow \quad & \frac{dI_a(t)}{I_a} \geq -\left(\alpha_a + \mu_h\right) dt \\ \Rightarrow \quad & I_a(t) \geq (I_{a0}) e^{-(\alpha_a + \mu_h)t} \geq 0 \end{aligned} \tag{27}$$

Hence, $I_a(t) > 0$ for all $t \geq 0$. Since $I_a(t) > 0,\ \forall t \geq 0$ and from (7)

$$\begin{aligned} \Rightarrow \quad & \frac{dR_a(t)}{dt} \geq -R_a\left(\Omega_a + \mu_h\right) \\ \Rightarrow \quad & \frac{dR_a(t)}{R_a} \geq -\left(\Omega_a + \mu_h\right) dt \\ \Rightarrow \quad & R_a(t) \geq (R_{a0}) e^{-(\Omega_a + \mu_h)t} \geq 0. \end{aligned} \tag{28}$$

It is simple observe that $S_v(t) > 0$ for all $t \geq 0$. Suppose that there exists t_1 and t^* with $t_1 < t^*$ such that $S_v\left(t_1\right) = 0,\ \frac{dS_v(t)}{dt} < 0$ and every the ten compartments are affirmative for $t_1 < t < t^*$. Then from equation (8),

$$\frac{dS_v(t)}{dt} = \lambda_v N_v - S_v(t)\varphi_{ev} I_e(t) - \mu_v S_v(t) - \Upsilon \varphi_{av} S_v(t) I_a(t) > 0$$

which is contradiction, hence $S_v(t) > 0,\ I_e > 0,\ I_a > 0,\ S_v > 0,\ \forall t \geq 0$

$$\begin{aligned} \Rightarrow \quad & \frac{dE_v(t)}{dt} \geq -E_v(t)\left(\gamma_v + \mu_v\right) \\ \Rightarrow \quad & \frac{dE_v(t)}{E_v} \geq -\left(\gamma_v + \mu_v\right) dt \\ \Rightarrow \quad & E_v(t) \geq (E_{v0}) e^{-(\gamma_v + \mu_v)t} > 0 \end{aligned} \tag{29}$$

Hence, $E_v(t) > 0$ for all $t \geq 0$. Since $E_v(t) > 0$ for all $t \geq 0$ (10)

$$\begin{aligned} \Rightarrow \quad & \frac{dI_v(t)}{dt} \geq -I_v(t)\mu_v \\ \Rightarrow \quad & \frac{dI_v(t)}{I_v} \geq -\mu_v dt \\ \Rightarrow \quad & I_v(t) \geq (I_{v0}) e^{-\mu_v t} > 0 \end{aligned} \tag{30}$$

Hence, $I_v(t) > 0,\ \forall t \geq 0$. Therefore, the solution of the system equation (1)- (10) is positive. Since the total number of humans population $N_v(t)$ is the sum of $S_e(t)$, $E_e(t)$, $I_e(t)$, $S_a(t)$, $E_a(t)$, $I_a(t)$ and $R_a(t)$ and the total number of mosquito population $N_v(t)$ is the sum of $S_v(t)$, $E_v(t)$ and $I_v(t)$, Since $S_e(t)+E_e(t)+I_e(t)+S_a(t)+E_a(t)+I_a(t)+R_a(t) = N_h$ and $S_v(t)+E_v(t)+I_v(t) = N_v$, then $S_e(t) \leq N_h, E_e(t) \leq N_h, I_e(t) \leq N_h, S_a(t) \leq N_h, E_a(t) \leq N_h, I_a(t) \leq N_h, R_a(t) \leq N_h$, and $S_v(t) \leq N_v, E_v(t) \leq N_v, I_v(t) \leq N_v,\ \forall t \geq 0$. Thus X is bounded. Therefore, (1)-(10) has a unique solution which is non negative and bounded. □

4 Equilibria points and Reproductive Number

4.1 Equilibria points

Theorem 2. *The model* (1)–(10) *has at least two equilibrium solutions, one disease free and one endemic equilibria.*

Proof. The equilibrium solution of the system is obtained by solving

$$\frac{dS_e}{dt} = \frac{dE_e}{dt} = \frac{dI_e}{dt} = \frac{dS_a}{dt} = \frac{dE_a}{dt} = \frac{dI_a}{dt} = \frac{dR_a}{dt} = \frac{dS_v}{dt} = \frac{dE_v}{dt} = \frac{dI_v}{dt} = 0.$$

Therefore, we set the system equation (1)- (10) equal to zero, then

$$\lambda_e N_h - S_e\left(\Upsilon\varphi_{ve} I_v + \mu_h\right) = 0. \tag{31}$$
$$\Upsilon S_e \varphi_{ve} I_v - E_e\left(\gamma_e + \mu_h\right) = 0. \tag{32}$$
$$\gamma_e E_e - I_e\left(\alpha_e + \mu_h\right) = 0. \tag{33}$$
$$\left(\lambda_h - \lambda_e\right) N_h + \Omega_a R_a - S_a\left(\Upsilon\varphi_{va} I_v + \mu_h\right) = 0. \tag{34}$$
$$S_a \Upsilon\varphi_{va} I_v - E_a\left(\gamma_a + \mu_h\right) = 0. \tag{35}$$
$$\gamma_a E_a - I_a\left(\mu_h + \alpha_a\right) = 0. \tag{36}$$
$$\left(\alpha_a + \alpha_e\right) I_a - \left(\Omega_a + \mu_h\right) R_a = 0. \tag{37}$$
$$\lambda_v N_v - S_v \Upsilon\left(\varphi_{ev} I_e + \varphi_{av} I_a\right) - S_v \mu_v = 0. \tag{38}$$
$$\left(\varphi_{ev} I_e + \varphi_{av} I_a\right) \Upsilon S_v - E_v\left(\gamma_v + \mu_v\right) = 0. \tag{39}$$
$$\gamma_v E_v - I_v \mu_v = 0. \tag{40}$$

From equation (31), we have obtained equation (41)

$$S_e = \frac{\lambda_e N_h}{\Upsilon\varphi_{ve} I_v + \mu_h}. \tag{41}$$

From equation (32) and (41), we have obtained the subsequent result

$$E_e = \left(\frac{\Upsilon\varphi_{ve}}{\gamma_e + \mu_h}\right)\left(\frac{\lambda_e N_h}{\Upsilon\varphi_{ve} I_v + \mu_h}\right) I_v \tag{42}$$

From equation (37), we have get the following result

$$R_a = \left(\frac{\alpha_a + \alpha_e}{\Omega_a}\right) I_a. \tag{43}$$

Substitute equation (43) into (35), we get

$$S_a = \left(\frac{\lambda_h - \lambda_e}{\Upsilon\varphi_{va} I_v + \mu_h}\right) N_h + \left(\frac{\alpha_a + \alpha_e}{\Upsilon\varphi_{va} I_v + \mu_h}\right) I_a. \tag{44}$$

From equation (33), we have

$$E_a = \frac{(\alpha_a + \mu_h) I_a}{\gamma_a}, \tag{45}$$

and from (38), we have

$$S_v = \frac{\lambda_v N_v}{\Upsilon\varphi_{ev} I_e + \mu_v + \varphi_{av} I_e \Upsilon} \tag{46}$$

From equation (40), we get

$$E_v = \frac{\mu_v I_v}{\gamma_v}. \tag{47}$$

In (47), if $I_v = 0$, we have $E_v = 0$, and from (41), we have get

$$S_e^* = \frac{\lambda_e N_h}{\mu_h}, \tag{48}$$

From equation (44), we have get

$$S_a^* = \left(\frac{\lambda_h - \lambda_e}{\mu_h}\right) N_h, \tag{49}$$

and from equation (46), we obtained

$$S_v^* = \frac{\lambda_v N_v}{\mu_v}. \tag{50}$$

From (45) if $I_a = 0$, and from (42), (45), (43) and (47), we have $E_e = 0, E_a = 0, R_a = 0, I_e = 0$ and $E_v = 0$. Therefore, we have obtained the disease free equilibrium

$$X_{dfe} = (S_e^*, 0, 0, S_a^*, 0, 0, 0, S_v^*, 0, 0)$$

where S_e^*, S_a^* and S_v^* are defined in (48), (49) and (50) respectively. To obtain the endemic equilibrium, from (38) and (39), we get

$$E_v^{**} = \frac{\lambda_v N_v}{\gamma_v + \mu_v}. \tag{51}$$

Substituting (51) into (31), we obtain

$$I_v^{**} = \frac{\gamma_v \lambda_v N_v}{\mu_v (\gamma_v + \mu_v)}. \tag{52}$$

Substitution of (52) into (41), yields

$$S_e^{**} = \left(\frac{\lambda_e N_h}{\Upsilon \varphi_{ve} \gamma_v}\right)\left(\frac{\mu_v (\gamma_v + \mu_v)}{\lambda_v N_v}\right). \tag{53}$$

Equations (53) and (32), give

$$E_e^{**} = \frac{N_h \lambda_e}{\mu_h + \gamma_e}. \tag{54}$$

Substituting (54) into (33), we get

$$I_e^{**} = \left(\frac{\gamma_e \lambda_e}{\alpha_e + \mu_h}\right)\left(\frac{N_h}{\gamma_e + \mu_h}\right). \tag{55}$$

From (37), we get

$$I_a = \left(\frac{\Omega_a + \mu_h}{\alpha_a + \alpha_e}\right) R_a. \tag{56}$$

Substitution of (56) into (34), yields

$$E_a = \left(\frac{\mu_h + \alpha_a}{\gamma_a}\right)\left(\frac{\mu_h + \alpha_a}{\gamma_a}\right)\left(\frac{\Omega_a + \mu_h}{\alpha_a + \alpha_e}\right) R_a. \tag{57}$$

Substitute (57) into (34), we have get the subsequent (58)

$$S_a = \left(\frac{\gamma_a + \mu_h}{\gamma_a}\right)\left(\frac{\Omega_a + \mu_h}{\alpha_a + \alpha_e}\right) R_a. \tag{58}$$

Substitute (58) into (35), we have

$$R_a^{**} = \frac{(\alpha_a + \alpha_e)(\lambda_e - \lambda_h) N_h}{\Omega_a (\alpha_a + \alpha_e) - (\gamma_a + \mu_h)(\mu_h + \alpha_a)(\Omega_a + \mu_h)}. \tag{59}$$

By substituting (59) into (37), we have obtained (60)

$$I_a^{**} = \frac{(\Omega_a + \mu_h)(\lambda_e - \lambda_h) N_h}{\Omega_a (\lambda_e + \lambda_a) - (\lambda_a + \mu_h)(\mu_h + \alpha_a)(\Omega_a + \mu_h)}. \tag{60}$$

By substituting (60) into (34), we have get (61)

$$E_a^{**} = \frac{(\mu_h + \alpha_a)(\Omega_a + \mu_h)(\lambda_e - \lambda_h) N_h}{\gamma_a ((\alpha_a + \alpha_e) - (\gamma_a + \mu_h)(\mu_h + \alpha_a)(\Omega_a + \mu_h))}. \tag{61}$$

By substituting (60) into (44), we have

$$S_a^{**} = \left(\frac{(\mu_h + \alpha_e)(\Omega_a + \mu_h)(\lambda_e - \lambda_h) N_h}{\Omega_a (\alpha_e + \alpha_a) - (\gamma_a + \mu_h)(\mu_h + \alpha_a)(\Omega_a + \mu_h)} \right) \left(\frac{\mu_v (\gamma_v + \mu_h)(\gamma_a + \mu_h)}{\gamma_v \lambda_v N_v \Upsilon \varphi_{va} \gamma_a} \right). \tag{62}$$

By substituting (55) (60) into (40), gives

$$S_v^{**} = \frac{\lambda_v N_v ((\Omega_a \alpha_a + \Omega_a \alpha_e - (\gamma_a + \mu_h)(\mu_h + \alpha_e)(\Omega_a + \mu_h)))((\alpha_e + \mu_h)(\gamma_e + \mu_h))}{\varphi_{ev} ((\Omega_a + \mu_h)(\lambda_e - \lambda_h)(\alpha_e + \mu_h)(\gamma_e + \mu_h) + (\gamma_e \lambda_e N_h)(\Omega_a (\alpha_e + \alpha_e) - (\gamma_a + \mu_h)(\mu_h + \alpha_a)))}$$

Consequently, the endemic equilibrium point of the system (1)–(10) is

$$(E_v^{**}, I_v^{**}, S_e^{**}, E_e^{**}, I_e^{**}, R_a^{**}, I_a^{**}, E_a^{**}, S_a^{**}, S_v^{**}),$$

where $S_e^{**}, E_e^{**}, I_e^{**}, S_a^{**}, E_a^{**}, I_a^{**}, R_a^{**}, S_v^{**}, E_v^{**}$ and I_v^{**} are defined in (51) , (52), (53), (54), (55), (59), (60), (61), (62) and (63) respectively. □

4.2 Reproductive Number

Let us denote the rate of the disease spread from e to e by β_{ee}, from a to a by β_{aa}, from v to v by β_{vv}, from a to e by β_{ae}, from e to a by β_{ea}, from e to v by β_{ev}, from v to e by β_{ve}, from v to a by β_{va} and from v to a by β_{va}. We use the next generation operator approach to describe the reproductive number as the number of secondary disease that one transferable individual would make above the period of the transferable period, given that everyone else is susceptible and the next-generation matrix β can be constructed like in Fernandes Lopez et al. (2002)

$$\beta = \begin{pmatrix} \beta_{ee} & \beta_{ae} & \beta_{ve} \\ \beta_{ea} & \beta_{aa} & \beta_{va} \\ \beta_{ev} & \beta_{av} & \beta_{vv} \end{pmatrix} \tag{63}$$

where every element β_{fg} characterises the predictable number of secondary suitcases in host indexed by g formed by a characteristic primary case in the group indexed by f in a completely susceptible population, where g and f can be a, e, v. Subsequently, the non-diseases are $\beta_{ee}, \beta_{aa}, \beta_{vv}, \beta_{ea}$ and β_{ae}. Thus

$$\beta_{ee} = \beta_{aa} = \beta_{vv} = \beta_{ea} = \beta_{ae} = 0. \tag{64}$$

and the diseases are $\beta_{ev}, \beta_{ea}, \beta_{av}$ and β_{va}

$$\beta_{ev} \neq 0, \beta_{ve} \neq 0, \beta_{av} \neq 0, \beta_{va} \neq 0. \tag{65}$$

Thus using (64),(65) and (63), we have

$$\beta = \begin{pmatrix} 0 & 0 & \beta_{ve} \\ 0 & 0 & \beta_{va} \\ \beta_{ev} & \beta_{av} & 0 \end{pmatrix}.$$

The β_{fg}'s are the result of the mean duration of the transferable life span, the likelihood of transmission per get in touch with, the continued existence possibility until the transferable state and the contact number per unit time. When a disease is recently introduced in a population by one infected individual then R_0 defines as the average number of secondary cases produced

by that infected during his complete infectious period. We assume that a single newly infected mosquito in the population at the disease free equilibrium point and let β_{va} be the predictable number of susceptible semi-resistant humans that this mosquito will infect, we have

$$\beta_{va} = \left(\frac{\gamma_v}{\mu_v + \gamma_v}\right)\left(\frac{\Upsilon S_a}{N_h}\right)\left(\frac{\varphi_{va}}{\mu_v}\right) \tag{66}$$

and β_{ve} be the predictable number of vulnerable non-resistant humans for which this mosquito will contaminate, we also have

$$\beta_{ve} = \left(\frac{\gamma_v}{\mu_h + \gamma_v}\right)\left(\frac{\Upsilon S_e}{N_h}\right)\left(\frac{\varphi_{ve}}{\mu_v}\right) \tag{67}$$

$$\beta_{av} = \left(\frac{\gamma_a}{\mu_h + \gamma_a}\right)\left(\frac{\Upsilon S_v}{N_h}\right)\left(\frac{\varphi_{av}}{\mu_h + \alpha_a}\right) \tag{68}$$

If we start with a single newly infected non-resistant human, then we have

$$\beta_{ev} = \left(\frac{\gamma_e}{\mu_h + \gamma_e}\right)\left(\frac{\Upsilon S_v}{N_h}\right)\left(\frac{\varphi_{ev}}{\mu_h + \alpha_e}\right) \tag{69}$$

The reproductive number, R_0, for malaria model (1) to (10) precisely is the spectral radius of β in Diekmann et al. (1990), then we get

$$R_0^2 = \beta_{ev}\beta_{ve} + \beta_{av}\beta_{va} \tag{70}$$

where β_{va}, β_{ve}, β_{av} and β_{ev} are defined in (66), (67), (68) and (69) respectively. At the disease-free equilibrium we assume that there is one infected non-immune human and there are non semi-immune human, we have

$$S_a = 0 \text{ and } S_e = N_h. \tag{71}$$

then (67), (69) and (71) yield

$$\beta_{ve} = 0. \tag{72}$$

Substituting (72) into equation (70), we obtain

$$R_0 = \sqrt{(\beta_{va})\,(\beta_{av})}. \tag{73}$$

And if the spread from semi-resistant humans to mosquitoes is zero then as of likewise we have

$$\beta_{av} = 0 \text{ and } \beta_{va} = 0. \tag{74}$$

Subsequently equation (74) into equation (70), then we acquired (75)

$$R_0 = \sqrt{(\beta_{ve})\,(\beta_{ev})}. \tag{75}$$

Let R_1 be the reproductive number for an infection due to β_{ev} or β_{ve}, then equation (70) becomes

$$R_1 = \sqrt{(\beta_{ve})\,(\beta_{ev})}. \tag{76}$$

and let R_2 be the reproductive number for the pollution due to β_{av} or β_{va}, then equation (70) becomes

$$R_2 = \sqrt{(\beta_{va})\,(\beta_{av})}. \tag{77}$$

Definition: We define the reproductive number, R_0, by

$$R_0 = \sqrt{R_1^2 + R_2^2} \tag{78}$$

where R_1 and R_2 are defined in equation (76) and (77) respectively.

5 Stability of equilibria

5.1 Stability of disease free equilibrium

Theorem 3. *The disease free equilibrium point is unstable if $R_0 > 1$ and globally asymptotically stable when $R_0 \leq 1$.*

Proof. The Proof follows van den Driessche and Watmough (2002). We rewrite equations (1)–(10) in the form of $\frac{dS}{dt} = \psi_1(S, I)$ and $\frac{dI}{dt} = \psi_2(S, I)$ wherever $S = (S_e, S_a, S_v)$ and $I = (E_e, I_e, E_a, I_a, R_a, E_v, I_v)$. Suppose B be the Jacobean matrix of $\psi = (\psi_1, \psi_2)$ calculated at the DFE $(S, 0)$. Then we obtained

$$\mathbf{B} = \begin{pmatrix} B_1 - B_2 & 0 \\ B_1 + B_2 & B_3 \end{pmatrix}$$

where

$$\mathbf{B_1} = \begin{pmatrix} 0 & 0 & 0 & 0 & 0 & M_{ve} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & M_{va} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & M_{ev} & 0 & M_{av} & 0 & 0 \end{pmatrix}$$

$$\mathbf{B_2} = \begin{pmatrix} K_1 & 0 & 0 & 0 & 0 & 0 \\ -\nu_e & K_3 & 0 & 0 & 0 & 0 \\ 0 & 0 & K_2 & 0 & 0 & 0 \\ 0 & 0 & -\nu_v & K_4 & 0 & 0 \\ 0 & -\alpha_e & 0 & -\alpha_a & 0 & 0 \\ 0 & 0 & 0 & 0 & -\nu_v & \lambda_v \end{pmatrix}$$

$$\mathbf{B_3} = \begin{pmatrix} \widehat{B_3} & 0 \\ 0 & \lambda_v S_v - \mu_v \end{pmatrix}$$

$$\widehat{\mathbf{B_3}} = \begin{pmatrix} -\mu_h S_e - \mu_h & \lambda_h - \mu_h S_e \\ -\mu_h S_a & -\mu_h S_a - \mu_h \end{pmatrix}$$

where $K_1 = \nu_e + \mu_h, K_2 = \nu_a + \mu_h, K_3 = \mu_h + \alpha_e, K_4 = \alpha_a + \mu_h$, $M_{ev} = \Upsilon\varphi_{ev}\frac{S_v}{N_h}, M_{av} = \Upsilon\varphi_{av}\frac{S_v}{N_h}, M_{ve} = \Upsilon\varphi_{ve}\frac{S_e}{N_h}, M_{va} = \Upsilon\varphi_{va}\frac{S_a}{N_h}$. The DFE is unstable if B has at least one eigenvalues with positive real part and close by asymptotically stable if every eigenvalues of B have negative real parts. The eigenvalues of B are $-S_v\mu_v < 0$ and those of $\widehat{B_3}$ and $B_1 - B_2$. We know that

$$\mathrm{Tr}\left(\widehat{B_3}\right) = -\mu_h S_e - \mu_h - \mu_h S_a - \mu_h < 0 \quad \text{and} \tag{79}$$

$$\det\left(\widehat{B_3}\right) = \mu_h\left(\mu_h + \mu_h S_e - \lambda_h S_a + \mu_h S_a + 2\mu_h S_e S_a\right) > 0 \tag{80}$$

As a result all eigenvalues of $\widehat{B_3}$ has strictly negative real components. Thus, the stability of the DFE depends on the eigenvalues of $B_1 - B_2$. We observe that B_2 has negative off-diagonal components and positive column sums. And B_2 is a non-singular M-matrix Berman and Plemmons (1994). Furthermore, B_1 is a non-negative matrix, then as of van den Driessche and Watmough (2002) we obtain the following $s\left(B_1 - B_2\right) < 0 \Leftrightarrow \rho\left(B_1 B_2^{-1}\right) < 1$ or $s\left(B_1 - B_2\right) > 0 \Leftrightarrow \rho\left(B_1 B_2^{-1}\right) > 1$ where $s(Q)$ represents the maximum real component of all eigenvalues of the matrix Q. Thus

$$\mathbf{B_2^{-1}B_1} = \begin{pmatrix} 0 & 0 & 0 & 0 & \beta_{ve} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & \beta_{va} & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ \beta_{ev} & 0 & \beta_{av} & 0 & 0 & 0 \end{pmatrix}$$

where β_{ve}, β_{va}, β_{av} and β_{ev} are defined in (67), (66), (68), and (69), respectively. In this case $R_0 = \rho\left(B_2^{-1}B_1\right)$. Therefore, the disease free equilibrium point is asymptotically stable if $R_0 \leq 1$, the disease dies out and when for $R_0 > 1$ the disease free equilibrium point is unstable and consequently the disease persists. □

5.2 Effort required to control malaria

The control reproduction number R_0 is the threshold which gives an indication on how control effort can be focussed and on which host the appropriate action should be taken. In Section 4 we calculated the control reproduction number R_0 specific to each host type. It was established in Roberts and Heesterbeek (2003) the expected number of cases of individuals of kind l, caused by one infected individual of kind l in a fully susceptible population, either directly or not directly. Our model considers three host kinds: semi-resistant, non-resistant and mosquito. If we directly apply R_0 to control malaria, we must decrease R_0 to values less than one to control malaria. In every condition, we are required to target the control of each the sub-groups to decrease R_0 below one. That it is very difficult and expensive to control all sub-groups to reduce the impact of malaria: can we control malaria by targetting only one sub-group? Using the method developed in Heesterbeek and Roberts (2007), we calculate the reproductive number $\omega_e, \omega_a, \omega_v$ for each host. We denote by $\rho(Q)$ the spectral radius of a matrix Q. In Heesterbeek and Roberts (2007), $\omega_I = \nu_I^{'}\nu_I \left(I(1-\beta) - \beta\ell_I\right)^{-1}$ for all $l = e, a, v$, where

$$\begin{cases} \nu_e = (1 \quad 0 \quad 0) \\ \nu_a = (0 \quad 1 \quad 0) \\ \nu_v = (0 \quad 0 \quad 1) \end{cases} \quad , \ell_{\mathbf{e}} = \begin{pmatrix} 1 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \ell_{\mathbf{a}} = \begin{pmatrix} 0 & 1 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \ell_{\mathbf{v}} = \begin{pmatrix} 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 1 \end{pmatrix}$$

and β is the next-generation matrix given by (63). In Heesterbeek and Roberts (2007), ω_l is defined if the host kind $\beta \neq l$ cannot hold by themselves an epidemic. Logically, it is shown that ω_l is well defined if $\rho((I - \ell_I)\beta) < 1$. In reality, if ω_l is defined, decline of ω_l below one is adequate to reduce R_0 below 1, by only target a control to the specific host l. Their hypothesis is suitable when the model cannot show a backward bifurcation. But if $\rho((I - \ell_I)\beta) < 1$. Then ω_e, ω_a and ω_v are given by

$$\begin{cases} \omega_e = \frac{R_1^2}{I - R_2^2} \quad \text{and} \quad R_2 = \beta\rho(I - \ell_e) \\ \omega_a = \frac{R_2^2}{I - R_1^2} \quad \text{and} \quad R_1 = \beta\rho(I - \ell_a) \\ \omega_v = R_0^2 \quad \text{and} \quad \beta\rho(I - \ell_v) = 0 \end{cases} \tag{81}$$

Consider $\chi > 0$. The same reasoning can be applied when $\chi < 0$ by setting $\xi = 1$. It is clear that ω_e is well defined if $R_2 < \xi$, ω_a is also well defined if $R_1 < \xi$ and as $\rho\beta(I - \ell_v) = 0 < 1$, ω_v is always well defined without condition upon the semi-resistant or non-resistant. Then

1. In regions where $R_1 < \xi$ and $R_2 < \xi$ such as $1 < R_0 < \sqrt{2}\xi$ or $1 < \omega_v < 2\xi^2$, it is adequate to target a control of one of the three host types to eliminate malaria.

2. In regions where $R_1 < \xi$ and $R_2 > \xi$, it is adequate to target a control of semi-resistant or mosquito host types to control malaria.

3. In regions where $R_1 > \xi$ and $R_2 < \xi$, it is adequate to target a control of non-resistant or mosquito host to control malaria.

4. In regions where $R_1 > \xi$ and $R_2 > \xi$, we need to target a control of mosquito and simultaneously semi-resistant and non-resistant host.

Assuming that the malaria control program aims to reduce the number of infectious in a given host $l, l = a, e, v$ following one of the conditions $(1) - (4)$. Recall that the next generation matrix coefficients, denoted by β_{jl}, represent the expected number of individuals of host of type l, which would be infected by a single infectious host type j. Assuming that the above controls act linearly on β_{jl}, one can linearly reduce the number of susceptible host of type l with $l, j = a, e, v$. A proportion $s_l > 1 - \frac{\xi^2}{\omega_l}$ of susceptible host type l need to be control to eliminate over time the malaria in the three populations in (see Heesterbeek and Roberts (2007) when $\xi = 1$). For the semi-resistant or non-resistant, this control plan is feasible by using a vaccine. Concerning the mosquitoes, the vector control measures such as insecticide-treated nets in addition to indoor residual spraying with insecticides is possible. In regions where the condition (1) is satisfied, it is adequate to permanently protect a proportion of semi-resistant greater than $1 - \frac{\xi^2}{\omega_a}$ or a proportion of non-resistant greater than $1 - \frac{\xi^2}{\omega_e}$, or eliminate a fraction of mosquitoes greater than $1 - \frac{\xi^2}{\omega_v}$. In regions where the condition (2) is satisfied, it is adequate to permanently protect a proportion of semi-resistant greater than $1 - \frac{\xi^2}{\omega_a}$ or eliminate a fraction of mosquitoes greater than $1 - \frac{\xi^2}{\omega_v}$. In regions where the condition (3) is satisfied, it is adequate to permanently protect a proportion of non-resistant greater than $1 - \frac{\xi^2}{\omega_e}$ or eliminate a fraction of mosquitoes greater than $1 - \frac{\xi^2}{\omega_v}$. In regions where the condition (4) is fulfilled, it is adequate to permanently eliminate a proportion of mosquitoes greater than $1 - \frac{\xi^2}{\omega_v}$ at birth to eradicate malaria or simultaneously protect the non-resistant and the semi-resistant. Our model suggests that in a region of low malaria transmission, it is adequate to target a control to one of exact host type to eradicate malaria. In widespread region where the birth rate of semi-resistant humans is very large then the birth rate of non-resistant humans then malaria can always be controlled throughout the non-resistant. In an widespread region wherever the birth rate of non-resistant humans is very large then the birth rate of semi-resistant humans then malaria can forever be controlled throughout the semi-resistant.

6 Numerical Simulation

Figure 2 is plot the constraints in Table 3 in region two, $R_1 = 2.62339 > 1$, $R_2 = 1.4225 > 1$ and $R_0 = 2.97458 > 1$. In Figure 3, the constraints in Table 3 in region one $R_1 = 0.445567 < 1$, $R_2 = 0.36789 < 1$, $R_0 = 0.73795 < 1$, $\omega_e = 1.6876$, $\omega_a = 17.29$ and $\omega_v = 1.3937$ with $E_e = 0$, $E_a = 0$, $I_e = 2$, $I_a = 1$, $R_a = 29$, $E_v = 18$, $I_v = 13.00$, $N_h = 395$ and $N_v = 13,000$. A mathematical imitation $R_0 = 2.9838, R_1 = 2.6449$ and $R_2 = 1.4245$ with primary situation: $E_e = 0, E_a = 0, I_e = 2, I_a = 1, R_a = 29, E_v = 190, I_v = 11, N_v = 396$ and $N_v = 13,000$ is plotted with constraint worths defined in Table 3 region one is specified in Figure 2. Figure 4 viewing the widespread stability values for the quantity of transferable non-resistant humans when we used the constraints in Table 3 on region two.

Table 3: The Base Line principles and variety for ten dimensional malaria model

No	parameter (constraint)	region one	region two	low	high
1	λ_v	0.23	0.23	0.4	0.39
2	φ_{ve}	0.0430	0.092	0.033	0.45
3	φ_{va}	0.044	0.044	0.03	0.49
4	φ_{ev}	0.33	0.66	0.078	0.96
5	φ_{av}	0.09	0.6600	0.083	0.93
6	γ_e	0.3	0.3	0.087	0.45
7	γ_a	0.09	0.09	0.088	0.04
8	γ_v	0.091	0.094	0.094	0.33
9	α_a	0.03	0.03	0.0025	0.035
10	α_e	0.007	0.006	0.0066	0.087
11	μ_v	0.055	0.066	0.008	0.9
12	Υ	0.48	0.49	0.55	0.77
14	Ω_a	0.88×10^{-5}	0.45×10^{-4}	0.33×10^{-3}	0.77×10^{-6}

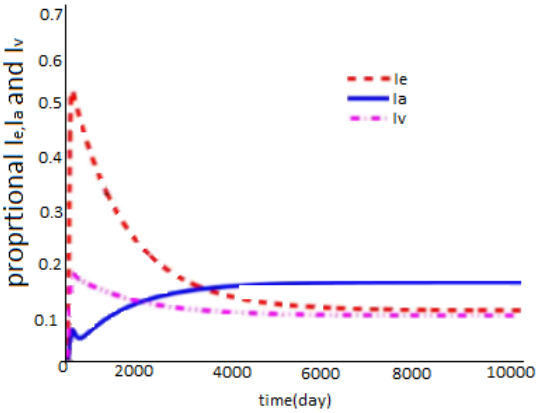

Figure 2: Infectious and semi-infectious class: $R_1 > 1$ and $R_2 > 1$

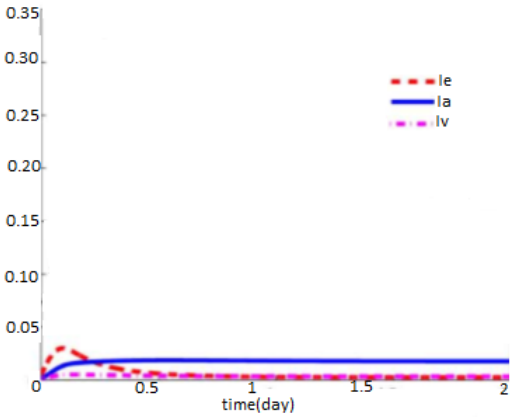

Figure 3: Infectious and semi-infectious class: $R_1 < 1$ and $R_2 < 1$

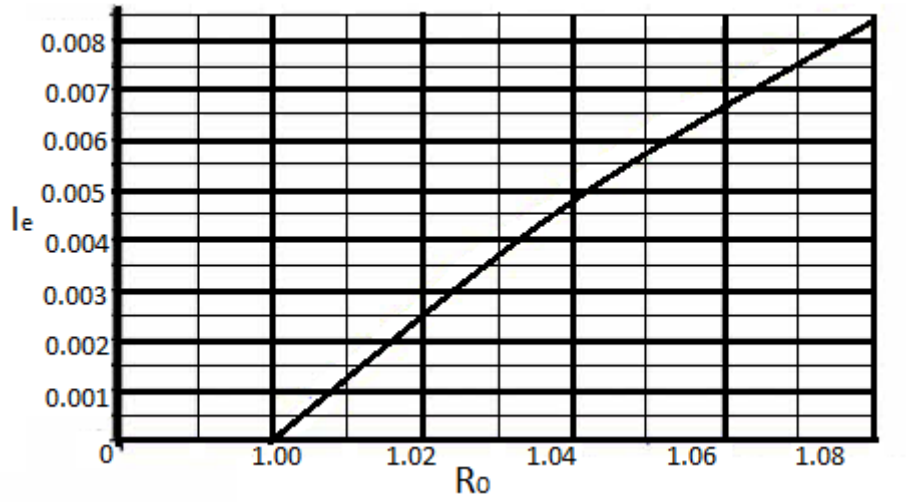

Figure 4: Forward bifurcation

7 Conclusion

In this paper, we studied a ten dimensional model for malaria transmission. We divided the human host into two major kinds: we named the first host kind, non-resistant, which included every human who do not have resistance against malaria; we named the second host kind semi-resistant. On the other hand, we divided partly-resistant humans into vulnerable (susceptible), uncovered (exposed), transferable (infectious) and resistant (recovered). We divided non-resistant humans being into vulnerable (susceptible), uncovered (exposed), transferable (infectious). We divided the mosquito population into three classes: (susceptible), uncovered (exposed), transferable (infectious). We obtained an explicit formula for the reproductive number, R_0, derived the local stability of the DFE point. We described R_1, the influence of the transmission non-resistant-mosquito-non-resistant and R_2, the weight of the transmission semi-resistant-mosquito-semi-resistant. Consequently, the reproductive number for the whole population is a square root of the summation of the square of these weights for the two contact types. For $R_0 < 1$, the disease free equilibrium point is globally asymptotically stable, implying that malaria dies out. And for $R_0 > 1$, malaria persists in the population. The simulations are carried out to illustrate the results and explore the possible behaviour of the formulated model.

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