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MATHEMATICAL MODELING ON THE TRANSMISSION DYNAMICS AND CONTROL OF MALARIA WITH TREATMENT WITHIN A POPULATION

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Abstract: This paper investigates the transmission of malaria and attempts to control the disease using mathematical model. In this research, we analyse the dynamics of malaria using a system of nine differential equations. The analysis carried out reveals that if the basic reproduction number Ro<l, the disease free equilibrium point is stable, but if Ro>l, the disease free equilibrium is unstable. We conclude from the analysis that there is no endemic equilibrium and malaria – free society is feasible. Our analysis also reveals that certain control strategies are capable of reducing malaria transmission. These are: sufficient treatment of infected people with drugs; spraying of insecticides and the use of insecticide-treated bed nets; destruction of mosquito breeding sites and campaign strategy for malaria control.

Keywords: Mathematical Modelling, Disease-Free Equilibrium, Basic Reproduction Number, Endemic Equilibrium Point, Sensitivity Analysis.

1. Introduction

Malaria is an infectious disease caused by the parasite known as plasmodium that is transmitted from human to human by the female anopheles mosquito. It was about 1880 that scientists discovered that malaria was a parasitic disease which is transmitted by the anopheles mosquito. The mosquito infects the host with a single cell parasite known as plasmodium. Shortly after, they discovered that malaria is transmitted from person to person through the bite of the female mosquito, which needs blood for her eggs. In humans, malaria is caused by plasmodium falciparum, plasmodium malariae, plasmodium ovale, plasmodium vivax and plasmodium knowlesi. Of these, plasmodium falciparum is the most common cause of infection in Africa and South East Asia, and is responsible for 80% of all malaria cases and 90% of deaths (Mandal et al, 2011). Malaria therefore is a life – threatening disease.

Malaria parasites multiply in the human liver and then infect red blood cells. A malaria patient may exhibit the following symptoms: headache, aching muscles, tummy ache and weakness or lack of energy. A day after, the body temperature may rise (up to about 40 degree Celsius) and the patient may have fever, shivers, severe headache, vomiting, diarrhoea etc. The time taken for symptom to appear depends on the type of parasite one is infected with. If one is infected with plasmodium falciparum malaria can progress to more severe form and be complicated. The symptom may include: low blood sugar levels, severe anemia, jaundice, fluid on one's lungs

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(pulmonary Oedema), acute respiratory distress syndrome, kidney failure, spontaneous bleeding, and state of chock (circulatory collapse) convulsions, paralysis and coma. Severe malaria is capable of affecting the patient's brain and central nervous system and can be fatal (WHO, 2009).

According to World Health Organization (WHO) 2012 report about 3.3 billion people were at risk of malaria in 2011, with people living in sub-Sahara Africa having the highest risk of malaria infection (Silva and Torres 2013). Since the campaign to eradicate malaria in the middle of the last century failed control of malaria infection has been aimed at reduction in morbidity and mortality. However, recent initiatives are based on elimination and eventual eradication of the disease (White et al, 2009).

Mathematical models can serve as tools for studying the dynamics of the spread of infectious diseases. Several researchers have used mathematical models of epidemiology to understand the dynamics of the spread of malaria in a population. Mathematical model can be used to determine the important factors in malaria transmission (Fatimawati and Tasman, 2013). Models produce the most useful data when they are formulated with important biological, economic and practical realities in mind and when their results interpreted with care, making them another useful tool in the fight against malaria.

2. Model Formulation

Anon linear mathematical model is formulated and analysed using deterministic approach. In modeling the dynamics of malaria transmission, the total human population is denoted by N_h and total mosquito population by N_V. We use S-E-I-I_T-I_N-R to describe the various states of the model for the humans and S-E-I- for the states of mosquitoes. Human population is divided into susceptible S_h, Exposed, E_h, infected, I_h, infected and treated I_T, infected and not treated, l_N and removed, R. The mosquito population is divided into susceptible, S_v, exposed, E_v, and infected I_v.

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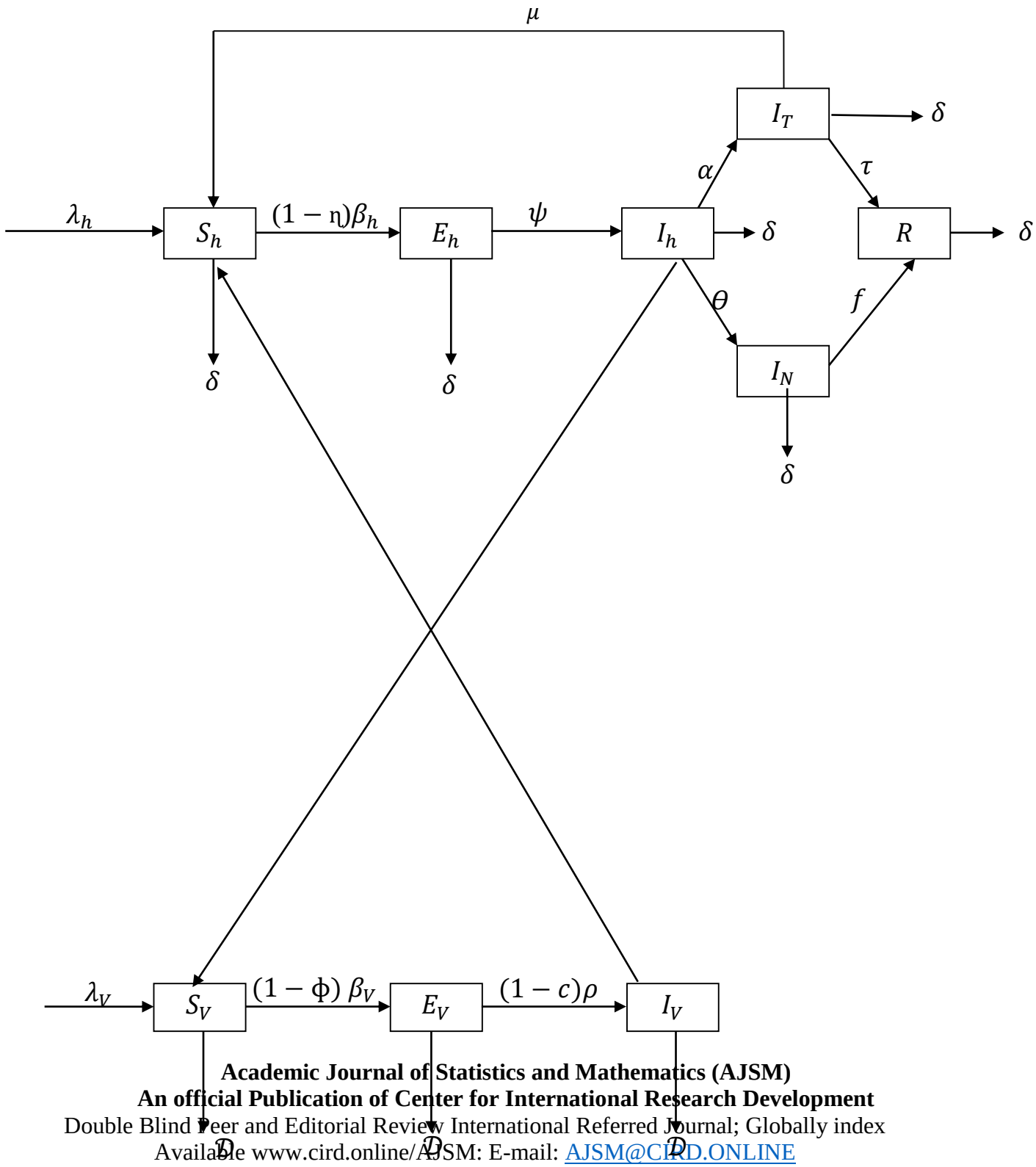
Figure 2.1. Flow diagram of the model



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Humans enter the population at the constant recruitment rate $\lambda_h$by birth, which is the rate at which one enters the susceptible class. Infected humans that are successfully treated return to susceptible class. When a susceptible human is bitten by an infected mosquito, there is the finite probability that the person will be infected and so he proceeds to exposed class at a constant rate of β_h. The campaign strategy for malaria control such as information on sleeping under mosquito treated nets or use of insecticides denoted as ɳ when practiced completely (i.e. ɳ=1) there will be no movement to exposed class. When the exposed human becomes infected, he moves to infectious class at a constant rate ofφ. The infected human can move to infected human treated at a constant rate of α or to infected human not treated at a constant rate of θ. When treatment of infected human fails he moves to removed (dead) class at a constant rate ofτ. The infected human not treated also move to removed class at a constant rate of f. Apart from disease induced death, humans also leave the population by natural death, δ.

Female anopheles mosquitoes enter the susceptible class by birth at a constant rate of λ_v. When the mosquito bites an infected human, the vector moves from susceptible to exposed class at a constant rate of β_v. A parameter ϕ which denotes reduction of mosquito breeding sites is introduced to reduce movement of mosquitoes from susceptible to exposed class. Mosquito moves from exposed to infected class at a constant rate of ρ. The biting rate of mosquito is denoted by π. A parameter c is to reduce the movement of mosquito from exposed to infectious class if there is adherence to awareness programme on malaria control such as spraying of insecticides. Mosquito leaves the population by natural death, insecticides and other causes at the constant rate of $\mathcal{D}$.

Equations of the Model

The model in fig. (2.1) satisfies the equation system:

$$\begin{aligned}
\frac{dS_h}{dt} &= \lambda_h + \mu I_T - (1-\eta)\beta_h S_h - \delta S_h \\
\frac{dE_h}{dt} &= (1\text{-}\eta)\beta_h S_h - \psi E_h - \delta E_h \\
\frac{dI_h}{dt} &= \psi E_h - \delta I_h - \alpha I_h - \theta I_h \\
\frac{dI_T}{dt} &= \alpha I_h - \mu I_T - \delta I_T - \tau I_T \\
\frac{dI_N}{dt} &= \theta I_h - f I_N - \delta I_N \\
\frac{dR}{dt} &= \tau I_T + f I_N - \delta R \\
\frac{dS_v}{dt} &= \lambda_v - (1\text{-}\phi)\beta_v S_v - \mathcal{D} S_v \\
\frac{dE_v}{dt} &= (1\text{-}\phi)\beta_v S_v - (1-c)\rho E_v - \mathcal{D} E_v \\
\frac{dI_v}{dt} &= (1-c)\rho E_v - \mathcal{D} I_v
\end{aligned} \qquad (1)$$

3. Model Analysis

The system of nonlinear equations will be qualitatively analyzed to enable us find the condition for existence and stability of disease free equilibrium points. Analysis of the model enables us to determine the impact of treatment and campaign strategy for malaria control on the transmission of malaria infection in a population. The value of the reproduction number Ro, enables us determine if the disease is endemic in the population or can be eradicated.

3.1 Positivity of Solutions

It is necessary we prove that all solutions of system (1) are positive for all t>0**.**

This will be established as follows:

Lemma 1

Let

$\{(S_h(0), E_h(0), I_h(0), I_T(0), I_N(0), R(0), S_v(0), E_v(0), I_v(0)) > 0\}\epsilon\Omega$

Then the solution set $\{S_h, E_h, I_h, I_T, I_N, R, S_v, E_v, I_v\}(t)$ of the system (1) is positive for all t > 0.

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Proof

From the first equation in the system (1), we obtain the inequality.

$\frac{dS_h}{dt} \geq -((1-\eta)\beta_h + \delta)S_h$ (2)

Which gives

$S_{h(t)} \geq S_h(0)e^{-((1-\eta)^{-1}\beta_h+\delta)t} \geq 0$

Therefore all solutions of system (1) are positive for all t> 0, since e^{γ}>0 for all $\gamma \epsilon \mathbb{R}$.

Similar proofs can be obtained for the positivity of E_h, I_h, l_T, I_N, R, S_v, E_v, l_v

3.2 Disease Free Equilibrium (DFE)

The disease free equilibrium of the model (1) is obtained by setting

$\frac{dS_h}{dt} = \frac{dE_h}{dt} = \frac{dl_h}{dt} = \frac{dI_T}{dt} = \frac{dI_N}{dt} = \frac{dR}{dt} = \frac{dS_v}{dt} = \frac{dE_v}{dt} = \frac{dI_v}{dt} = 0$ (3)

In the absence of disease or disease-free state ($E_h = I_h = I_T = I_N = E_v = I_v = 0$) and the system (1) becomes

$\lambda_h - \delta N_h = 0$

$\lambda_v - \mathcal{D}S_v^{t0} = 0$

Therefore, the disease-free equilibrium (DFE) denoted by E_0 of the system (1) is given by:

$E_0 = \frac{\lambda_h}{\delta}, 0,0,0,0,0, \frac{\lambda v}{\mathcal{D}}, 0,0$ (4)

3.3 Local Stability of Disease-Free Equilibrium

The disease free equilibrium of the model (2.1) was given by:

$E_0 = (S_h^{t0}, E_h^{t0}, I_h^{t0}, I_T^{t0}, I_N^{t0}, R^{to}, S_v^{t0}, E_v^{t0}, I_v^{t0})$

$= \frac{\lambda_h}{\delta}, 0,0,0,0,0, \frac{\lambda v}{\mathcal{D}}, 0,0)$ (5)

The disease-free equilibrium point for system (1) is locally asymptotically stable if R_o<l and unstable if R_o>l.

3.4 Basic Reproduction Number R_0

The basic reproduction number R_o is the number of secondary infections that one infectious Individual would create over the duration of the infectious period provided that everyone else is susceptible. It is a parameter used to determine how long an infection will prevail in a particular population and this is determined by the value of R_0. If the basic reproduction number R_0 < 1, it implies that an infected individual produces an average of less than one infected person, which implies that with time the disease will go into extinction in that population. But if R_0 > 1, this implies that an infected individual produces more than one infected person in the population, and so the disease will remain in the population. Therefore, for the disease to die out of the population with time, R_0 must be less than one.

R_0 is obtained by taking the largest non-negative eigen value (Spectral radius) of

$\left[\frac{\partial fi}{\partial xj}(E_0)\right]\left[\frac{\partial vi}{\partial xj}(E_0)\right]^{-1}$ (Addo, 2009) (6)

Where

F_i is the rate of appearance of new infection in compartment i

V_i is the transfer of individuals out of the compartment i by all other means.

E_o is the disease-free equilibrium.

The associated matrix at disease free equilibrium is obtained as:

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & 0 & (1-\eta)\varepsilon_{vh}\pi \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{(1-\phi)\varepsilon_{hv}\pi\delta\lambda_v}{\mathcal{D}\lambda_h} & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{bmatrix}$$

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And

$$V = \begin{pmatrix} (\psi+\delta) & 0 & 0 & 0 & 0 & 0 \\ -\psi & (\delta+\alpha+\theta) & 0 & 0 & 0 & 0 \\ 0 & -\alpha & (\mu+\delta+\tau) & 0 & 0 & 0 \\ 0 & -\theta & 0 & (f+\delta) & 0 & 0 \\ 0 & 0 & 0 & 0 & (\mathcal{D}+(1-c)\rho) & 0 \\ 0 & 0 & 0 & 0 & -(1-c)\rho & \mathcal{D} \end{pmatrix}$$

It follows that the basic reproduction number R_o for the model system (1) is given by:

$$R_0 = \sqrt{\frac{\psi(1-\phi)\epsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\epsilon_{vh}(1-c)\rho}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathcal{D}^2[\mathcal{D}+(1-c)\rho]}} \quad (7)$$

$$R_0 = \sqrt{R_{oh} x R_{ov}} \quad (8)$$

$$= \sqrt{\frac{(1-\eta)\pi\varphi\epsilon_{vh}\delta}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)}} X \frac{(1-\phi)(1-c)\rho\epsilon_{hv}\pi\lambda_v}{\mathcal{D}^2(\mathcal{D}+(1-c)\rho)}$$

3.5 Endemic Equilibrium Point

Endemic equilibrium point is a state where at least one of the infected compartments is non zero, or are steady state solutions where the disease persists in the population. It implies that malaria infection will persist in the population and the endemic Equilibrium (EEP) of the model is given as:

$$EEP = (S_h^e, E_h^e, I_h^e, I_T^e, I_N^e, R^e, S_v^e, E_v^e, I_v^e) \quad (9)$$

$$(S_h^e, E_h^e, I_h^e, I_T^e, I_N^e, R^e, S_v^e, E_v^e, I_v^e) > 0$$

To derive the EEP, we have to solve (1) by equating it to zero.

$$\lambda_h + \mu I_{hT} - \frac{(1-\eta)\varepsilon_{vh}\pi I_v S_h}{N_h} - \delta s_h = 0 \quad (10)$$

$$\frac{(1-\eta)\varepsilon_{vh}\pi I_v S_h}{N_h} - (\psi+\delta)E_h = 0 \quad (11)$$

$$\psi E_h - (\delta+\alpha+\theta)I_h = 0 \quad (12)$$

$$\alpha I_h - (\mu+\delta+\tau)I_T = 0 \quad (13)$$

$$\theta I_h - (f+\delta)I_N = 0 \quad (14)$$

$$\tau I_T + f I_N - \delta R = 0 \quad (15)$$

$$\lambda_v - \frac{(1-\phi)\varepsilon_{hv}\pi I_h S_v}{N_h} - \mathcal{D}S_v = 0 \quad (16)$$

$$\frac{(1-\phi)\varepsilon_{hv}\pi I_h S_v}{N_h} - [\mathcal{D}+(1-c)\rho]E_v = 0 \quad (17)$$

$$(1-c)\rho E_v - \mathcal{D}I_v = 0 \quad (18)$$

From the (18), we have:

$$I_v^e = \frac{(1-c)\rho E_v^e}{\mathcal{D}} \quad (19)$$

From equation (19), we have

$$E_v^e = \frac{(1-\phi)\epsilon_{hv}\pi I_h^e S_v^e}{N_h(\mathcal{D}+(1-c)\rho)} \quad (20)$$

Substituting (20) into (19), we have

$$I_v^e = \frac{[(1-c)\rho](1-\phi)\epsilon_{hv}\pi I_h^e S_v^e}{\mathcal{D}N_h[\mathcal{D}+(1-c)\rho]} \quad (21)$$

From equation (16), we have:

$$S_v^e = \frac{\lambda_v N_h}{(1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h} \quad (22)$$

Substituting (22) into (21), we have

$$I_v^e = \frac{[(1-c)\rho](1-\phi)\epsilon_{hv}\pi I_h^e \lambda_v}{\mathcal{D}[\mathcal{D}+(1-c)\rho][(1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h]}$$

$$I_v^e = \frac{\mathcal{D}[(1-c)\rho](1-\phi)\epsilon_{hv}\pi I_h^e \lambda_v}{\mathcal{D}^2[\mathcal{D}+(1-c)\rho][(1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h]}$$

From equation (8)

$$R_{ov} = \frac{(1-\phi)(1-c)\rho\epsilon_{hv}\pi\lambda_v}{\mathcal{D}^2(\mathcal{D}+(1-c)\rho)}$$

Therefore

$$I_v^e = \frac{R_{ov}\mathcal{D}I_h^e}{((1-\phi)\epsilon_{hv}\pi l_h^e + \mathcal{D}N_h)} \quad (23)$$

From equation (11), we have:

$$\frac{(1-\eta)\epsilon_{vh}\pi I_v^e S_h^e}{N_h} - (\psi+\delta)E_h^e = 0 \quad (24)$$

Substituting (23) into (24) we have

$$\frac{(1-\eta)\epsilon_{vh}\pi\mathcal{D}R_{ov}I_h^e S_h^e}{N_h((1-\phi)\epsilon_{hv}\pi l_h^e + \mathcal{D}N_h} - (\varphi+\delta)E_h^e = 0 \quad (25)$$

From equation (12) we have

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$$E_h^e = \frac{(\delta+\alpha+\theta)I_h^e}{\varphi}$$

(26)

Substituting (26) into (25) we have:

$$\frac{(1-\eta)\epsilon_{vh}\pi\mathcal{D}R_{0v}I_h^eS_h^e}{N_h((1-\phi)\epsilon_{hv}\pi I_h^e+\mathcal{D}N_h)} - \frac{(\varphi+\delta)(\delta+\alpha+\theta)I_h^e}{\varphi} \quad (27)$$

$$\frac{\varphi(1-\eta)\epsilon_{vh}\pi\mathcal{D}R_{0v}I_h^eS_h^e - N_h((1-\phi)\epsilon_{hv}\pi I_h^e+\mathcal{D}N_h)(\psi+\delta)(\delta+\alpha+\theta)I_h^e}{\varphi N_h((1-\phi)\epsilon_{hv}\pi l_h^e+\mathcal{D}N_h} = 0$$

$$\varphi(1-\eta)\epsilon_{vh}\mathcal{D}R_{0v}I_h^e - N_h\big((1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h\big)(\psi+\delta)(\delta+\alpha+\theta)I_h^e = 0$$

$$I_h^e\big[\varphi(1-\eta)\epsilon_{vh}\pi\mathcal{D}R_{0v}S_h^e - N_h\big((1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h\big)(\psi+\delta)(\delta+\alpha+\theta)\big] = 0$$

$$I_h^e = 0 \ \ or$$

$$\varphi(1-\eta)\epsilon_{vh}\pi\mathcal{D}R_{0v}S_h^e - N_h\big((1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h\big)(\psi+\delta)(\delta+\alpha+\theta) = 0$$

$$\varphi(1-\eta)\epsilon_{vh}\pi\mathcal{D}R_{0v}S_h^e - N_h\big((1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h\big)(\psi+\delta)(\delta+\alpha+\theta) = 0$$

(28)

Dividing (28) by $N_h(\varphi+\delta)(\delta+\alpha+\theta)$ we have

$$\frac{\psi(1-\eta)\epsilon_{vh}\pi\mathcal{D}R_{0v}S_h^e}{N_h(\varphi+\delta)(\delta+\alpha+\theta)} - \big((1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h\big) = 0$$

$$\left(\frac{\psi(1-\eta)\epsilon_{vh}\pi}{N_h(\varphi+\delta)(\delta+\alpha+\theta)}\right)R_{0v}\mathcal{D}S_h^e - \big((1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}N_h\big) = 0$$

But $N_h \le \frac{\lambda_h}{\delta}$ *and* $\frac{1}{N_h} \le \frac{\delta}{\lambda_h}$

Therefore

$$\left(\frac{\psi(1-\eta)\epsilon_{vh}\pi\delta}{\lambda_h(\varphi+\delta)(\delta+\alpha+\theta)}\right)R_{0v}\mathcal{D}S_h^e - \left((1-\phi)\epsilon_{hv}\pi I_h^e + \frac{\mathcal{D}\lambda_h}{\delta}\right) = 0$$

Recall that

$$\frac{(1-\eta)\varphi\pi\epsilon_{vh}\delta}{\lambda_h(\varphi+\delta)(\delta+\alpha+\theta)} = R_{0h}$$

$$\Rightarrow R_{0h}\ X\ R_{0v}\mathcal{D}S_h^e - \left((1-\phi)\epsilon_{hv}\pi I_h^e + \frac{\mathcal{D}\lambda_h}{\delta}\right) = 0$$

$$R_{0h}\ X\ R_{0v}\mathcal{D}S_h^e = \frac{\delta(1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}\lambda_h}{\delta}$$

But $R_0^2 = R_{0h}\ X\ R_{0v}$

Then

$$R_0^2 DS_h^e = \frac{\delta(1-\phi)\epsilon_{hv}\pi I_h^e + \mathcal{D}\lambda_h}{\delta}$$

$$S_h^e = \frac{\delta(1-\phi)\epsilon_{hv}\pi I_h^e+\mathcal{D}\lambda_h}{R_0^2 D\delta} \quad (29)$$

Using equation (10) we have

$$\lambda_h + \mu I_T - \frac{(1-\eta)\epsilon_{vh}\pi I_v^e S_h^e}{N_h} - \delta S_h^e = 0 \quad (30)$$

Substituting (23) and (29) into (30) we have:

$$\lambda_h + \mu I_T - (1-\eta)\epsilon_{vh}\pi\left(\frac{R_{0v}\mathcal{D}I_h^e}{(1-\phi)\epsilon_{hv}\pi I_h^e+\mathcal{D}N_h}\right)\left(\frac{\delta(1-\phi)\epsilon_{hv}\pi I_h^e+\mathcal{D}\lambda_h}{R_0^2\mathcal{D}\delta}\right) - \delta\left(\frac{\delta(1-\phi)\epsilon_{hv}\pi I_h^e+\mathcal{D}\lambda_h}{R_0^2\mathcal{D}\delta}\right) = 0$$

$$\begin{aligned}
&-[(1-\eta)\epsilon_{vh}\pi R_{0v}\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi \\
&\quad + \delta^2(1-\phi)^2\epsilon_{hv}^2\pi^2]I_h^{e^2} \\
&\quad + [\lambda_h R_0^2\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi \\
&\quad + \mu I_T R_0^2\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi \\
&\quad - (1-\eta)\epsilon_{vh}\pi R_{0v}\mathcal{D}^2\lambda_h \\
&\quad - \delta^2(1-\phi)\epsilon_{hv}\pi\mathcal{D}N_h \\
&\quad - \delta\mathcal{D}\lambda_h(1-\phi)\epsilon_{hv}\pi]I_h^e \\
&\quad + [\lambda_h R_0^2\mathcal{D}^2\delta N_h - \delta\mathcal{D}^2\lambda_h N_h] = 0
\end{aligned}$$

$$\begin{aligned}
&\big[(1-\eta)\epsilon_{vh}\pi R_{0v}\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi \\
&\quad + \delta^2(1-\phi)^2\epsilon_{hv}^2\pi^2\big]I_h^{e^2} \\
&\quad - [\lambda_h R_0^2\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi \\
&\quad + \mu I_T R_0\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi - (1 \\
&\quad - \eta)\ \epsilon_{vh}\pi R_{0v}\mathcal{D}^2\lambda_h \\
&\quad - \delta^2(1-\phi)\epsilon_{hv}\pi\mathcal{D}N_h \\
&\quad - \delta\mathcal{D}\lambda_h(1-\phi)\epsilon_{vh}\pi]I_h^e \\
&\quad - \ [\lambda_h R_0^2\mathcal{D}^2\delta N_h + \mu I_T R_0^2\mathcal{D}^2\delta N_h \\
&\quad - \delta\mathcal{D}^2\lambda_h N_h] = 0
\end{aligned}$$

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The above equation is quadratic. i.e

$$A[I_h^e]^2 + BI_h^e + c = 0 \qquad (31)$$

Where

$$A = (1-\eta)\epsilon_{vh}\pi R_{ov}\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi + \delta^2(1-\phi)^2\epsilon_{hv}^2\pi^2$$

$$B = -[\lambda_h R_0^2\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi + \mu I_T R_0\mathcal{D}\delta(1-\phi)\epsilon_{hv}\pi - (1-\eta)\epsilon_{vh}\pi R_{0v}\mathcal{D}^2\lambda_h - \delta^2(1-\phi)\epsilon_{hv}\pi\mathcal{D}N_h - \delta\mathcal{D}\lambda_h(1-\phi)\epsilon_{vh}\pi]$$

$$C = -[\lambda_h R_0^2\mathcal{D}^2\delta N_h + \mu I_{hT}R_0^2\mathcal{D}^2\delta N_h - \delta\mathcal{D}^2\lambda_h N_h]$$

Applying the quadratic formula

$$x = \frac{-B \pm \sqrt{B^2-4AC}}{2A}$$

We have

$$I_h^e = \frac{-B \pm \sqrt{B^2-4AC}}{2A}$$

$$= \frac{-B+\sqrt{B^2-4AC}}{2A} \; or \quad \frac{-B-\sqrt{B^2-4AC}}{2A}$$

$$I_h^e = \frac{-B+\sqrt{B^2-4AC}}{2A} = \frac{\sqrt{B^2-4AC}-B}{2A} = \boldsymbol{G}, \qquad I_h^e \geq 0 \qquad (32)$$

From equation (29), we have:

$$S_h^e = \frac{\delta(1-\phi)\epsilon_{hv}\pi\boldsymbol{G}+\mathcal{D}\lambda_h}{R_0^2\mathcal{D}\delta} \qquad (33)$$

From equation (26), we have:

$$E_h^e = \frac{(\delta+\alpha+\theta)G}{\varphi} \qquad (34)$$

From equation (22) we have

$$S_v^e = \frac{\lambda_v N_v}{(1-\phi)\epsilon_{hv}\pi\boldsymbol{G}+\mathcal{D}N_h} \qquad (35)$$

From equation (20), we have:

$$E_v^e = \frac{(1-\phi)\epsilon_{hv}\pi I_h^e S_v^e}{N_h[\mathcal{D}+(1-c)\rho]}$$

Substituting for S_v^e, we have:

$$E_v^e = \frac{(1-\phi)\epsilon_{hv}\pi I_h^e\lambda_v}{[\mathcal{D}+(1-c)\rho][(1-\phi)\epsilon_{hv}\pi I_h^e+\mathcal{D}N_h]}$$

$$E_v^e = \frac{(1-\phi)\epsilon_{hv}\pi\boldsymbol{G}\lambda_v}{[\mathcal{D}+(1-c)\rho][(1-\phi)\epsilon_{hv}\pi\boldsymbol{G}+\mathcal{D}N_h]} \qquad (36)$$

From equation (23) we have:

$$I_v^e = \frac{R_{ov}\mathcal{D}\boldsymbol{G}}{\left((1-\phi)\epsilon_{hv}\pi\boldsymbol{G}+\mathcal{D}N_h\right)} \qquad (37)$$

This situation leads to the following theorem:

Theorem 4.4

The malaria model (4.1) has

i. Precisely one unique endemic equilibrium if $C < 0 \Leftrightarrow R_0 < 1$
ii. Precisely one unique endemic equilibrium if $B < 0 \; and \; C = 0 \; or \; B^2 - 4AC = 0$
iii. Precisely two unique endemic equilibria if $C > 0, B < 0 \; and \; B^2 - 4AC > 0$.
iv. No endemic equilibrium if otherwise.

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4. Estimation of Parameters

Table 1: parameters estimation

Parameter	Value	Source
λ_h	50000	ESTIMATED
δ	0.00004566	Fatmawati (2013)
Ψ	0.08333	Chitnis (2008)
μ	0.25	Silva (2013)
η	0.75	Onah (2015)
θ	0.0723	ESTIMATED
α	0.533	ESTIMATED
λ_v	23350	Niger (2008)
β_v	0.09091	Chitnis (2006)
ρ	0.75	Lakshmikantham (2004)
Ɗ	0.0476	Silva (2013)
T	0.05	ESTIMATED
Ƒ	0.4	ESTIMATED
ϕ	0.59	Onah (2015)
ε_{hv}	0.86	Niger (2008)
ε_{vh}	0.083	Miranda *et al* (2009)
π	0.50	Johansson (2010)
c	0.65	ESTIMATED

From (7), the basic reproduction number R_O is given as

$$R_O = \sqrt{\frac{\psi(1-\phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)Ɗ^2[Ɗ+(1-c)\rho]}}$$

$R_O = 0.004904929309$

5. Sensitivity Analysis

Sensitivity analysis is used to determine the sensitivity of a model to changes in the values of the parameters of the model and to changes in the structure of the model (Atokolo and Omale, 2018). Based on model (1) the effective reproduction number.

R_e

$$= \frac{\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)Ɗ^2[Ɗ+(1-c)\rho]}$$

The sensitivity index of π with respect to R_e

$$= \frac{\partial R_e}{\partial \pi} \times \frac{\pi}{R_e}$$

$$= \frac{2\pi^2\psi(1-\Phi)\varepsilon_{hv}\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho\lambda_h(\psi+\delta)(\delta+\alpha+\theta)Ɗ^2[Ɗ+(1-c)\rho]}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)Ɗ^2[Ɗ+(1-c)\rho]\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

$= 2.$

The sensitivity index of ε_{vh} with respect to R_e is

$$= \frac{\partial R_e}{\partial \varepsilon_{vh}} \times \frac{\varepsilon_{vh}}{R_e}$$

$$= \frac{\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)(1-c)\rho\varepsilon_{vh}\lambda_h(\psi+\delta)(\delta+\alpha+\theta)Ɗ^2[Ɗ+(1-c)\rho]}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)Ɗ^2[Ɗ+(1-c)\rho]\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

$= 1.$

The sensitivity index of Ɗ with respect to R_e is

$$= \frac{\partial R_e}{\partial Ɗ} \times \frac{Ɗ}{R_e},$$

$$= \frac{-\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho[3D^2+2D\rho-2cD\rho]D\lambda_h(\psi+\delta)(\delta+\alpha+\theta)Ɗ^2[Ɗ+(1-C)\rho]}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\{Ɗ^2[Ɗ+(1-C)\rho]\}^2\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-C)\rho}$$

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$$= \quad -2.153$$

The sensitivity index of δ with respect to R_e is

$$=\frac{\partial R_e}{\partial \delta} \times \frac{\delta}{R_e}$$

$$= \quad \frac{\varphi(1-\Phi)\varepsilon_{hv}\pi^2\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho\{\delta(\alpha+\theta+\delta)+\psi(\alpha+\theta)\}\delta\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\,D^2[\mathrm{D}+(1-c)\rho]}{\lambda_h D^2[\mathrm{D}+(1-c)\rho][\delta(\psi+\alpha+\theta+\delta)+\psi(\alpha+\theta)]^2\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

=0.9999

The sensitivity index of ε_{hv} with respect to R_e is

$$= \quad \frac{\partial R_e}{\partial \varepsilon_{hv}} \times \frac{\varepsilon_{hv}}{R_e}$$

$$= \frac{\varphi(1-\Phi)\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho\varepsilon_{hv}\lambda_{h\,(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2\,[\mathrm{D}+(1-c)\rho]}}{\lambda_{h\,(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[\mathrm{D}+(1-c)\rho]\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda v(1`-\eta)\varepsilon_{vh}(1-c)\rho}}$$

= 1

The sensitivity index of ρ in respect to R_e is

$$= \quad \frac{\partial R_e}{\partial \rho} \times \frac{\rho}{R_e}$$

$$= \frac{\varphi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho\lambda_{h\,(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^3\,[\mathrm{D}+(1-c)\rho]}}{\lambda_{h\,(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[\mathrm{D}+(1-c)\rho]^2\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda v(1`-\eta)\varepsilon_{vh}(1-c)\rho}}$$

= 0.1535

The sensitivity index of ψ with respect to R_e is

$$= \frac{\partial R_e}{\partial \psi} \times \frac{\psi}{R_e}$$

$$= \frac{\delta^2(1-\Phi)\varepsilon_{hv}\pi^2\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho\psi\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[D+(1-c)\rho]}{\lambda_h(\psi+\delta)^2(\delta+\alpha+\theta)\mathrm{D}^2[\mathrm{D}+(1-c)\rho]\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

= 0.0005476

The sensitivity index of α with respect to R_e is

$$= \frac{\partial R_e}{\partial \alpha} \times \frac{\alpha}{R_e}$$

$$= \frac{-\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho\alpha\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[D+(1-c)\rho]}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)^2\mathrm{D}^2[\mathrm{D}+(1-c)\rho]\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

= −0.8805

The sensitivity index of θ with respect to R_e *is*

$$= \frac{\partial R_e}{\partial \theta} \times \frac{\theta}{R_e}$$

$$= \frac{-\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho\theta\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[D+(1-c)\rho]}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)^2\mathrm{D}^2[\mathrm{D}+(1-c)\rho]\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

=−0.1194

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The sensitivity index of Φ with respect to R_e *is*

$$= \frac{\partial R_e}{\partial \Phi} \times \frac{\Phi}{R_e}$$

$$= \frac{-\psi\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho\Phi\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[D+(1-c)\rho]}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[\mathrm{D}+(1-c)\rho]\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

$= -1.439$

The sensitivity index of η with respect to R_e *is*

$$= \frac{\partial R_e}{\partial \eta} \times \frac{\eta}{R_e}$$

$$= \frac{-\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v\varepsilon_{vh}(1-c)\rho\eta\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[D+(1-c)\rho]}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[\mathrm{D}+(1-c)\rho]\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

$= -3$

The sensitivity index of c with respect to R_e *is*

$$= \frac{\partial R_e}{\partial \mathrm{c}} \times \frac{\mathrm{c}}{R_e}$$

$$= \frac{-\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}\rho\{[\mathrm{D}+(1-\mathrm{c})\rho+(1-\mathrm{c})\rho]\}\mathrm{c}\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[D+(1-c)\rho]}{\lambda_h(\psi+\delta)(\delta+\alpha+\theta)\mathrm{D}^2[\mathrm{D}+(1-c)\rho]^2\psi(1-\Phi)\varepsilon_{hv}\pi^2\delta\lambda_v(1-\eta)\varepsilon_{vh}(1-c)\rho}$$

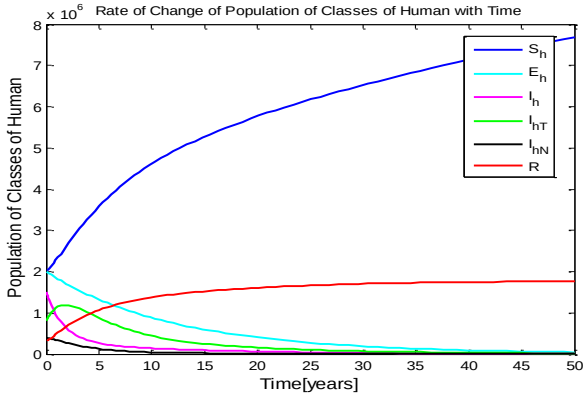
$= -3.469$

6. Numerical Simulations

The numerical simulations of the model (1) are conducted to find out the dynamics of the disease in the human population. The simulations are conducted using MATLAB. The initial conditions were

$S_h = 2{,}000{,}000.$ $E_h = 2{,}000{,}000$
$I_h = 1500{,}000$ $I_T = 800{,}000$
$I_N = 400{,}000$ $R = 300{,}000$
$S_v = 300{,}000$ $E_v = 150{,}000$
$I_v = 50{,}000$

The simulation was conducted in years.



I_T
I_N

Fig 4.1: This illustrates the changes in the six classes of human. The susceptible humans, exposed humans, infected humans, infected humans treated, infected humans not treated and removed humans with time. it shows the transmission through the model affecting all the

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parameters, including treatment rate, campaign strategy for malarial control, use of insecticides and insecticides – treated bed nets and reduction of mosquito breeding sites.

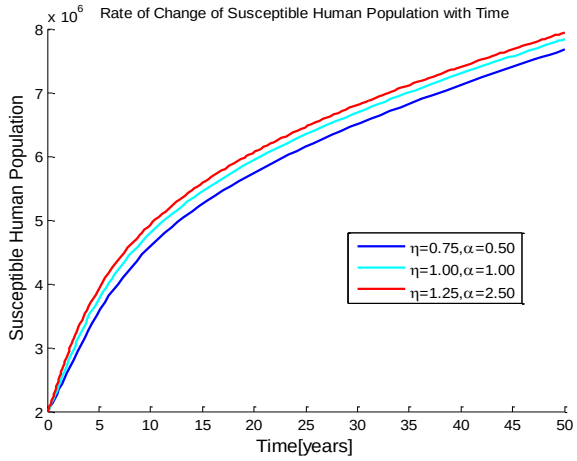

Fig 4.2(a): This shows the response of susceptible human population to increase in treatment rate and campaign strategy for malaria control. The susceptible human population increases with increase in treatment rate and campaign strategy for malaria control. The increase in susceptible human population is as a result of rising number of treated people returning to susceptible class.

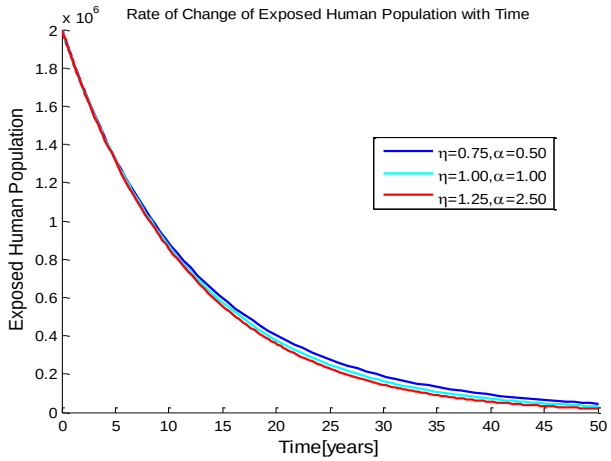

Fig 4.2(b): The graph shows the response of exposed human population to increase in treatment rate and campaign strategy for malaria control. It is observed from the graph that the increase in treatment rate and campaign strategy causes decrease in the exposed human population.

From the graph, there is a sharp decrease in the exposed human population up to a period of about 10years after which the decrease becomes gradual.

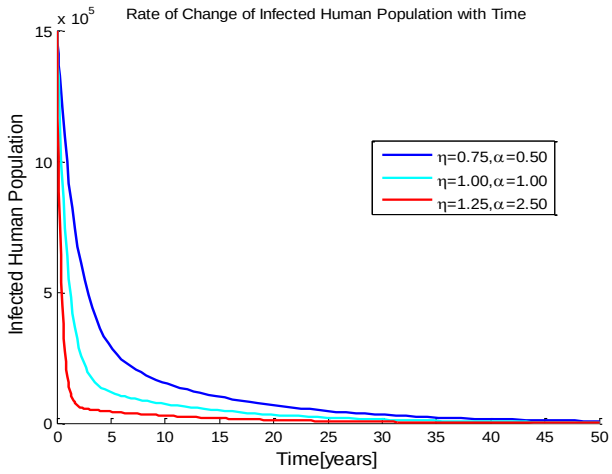

Fig 4.2(c): The graph illustrates the response of infected human population to increase in treatment rate and campaign strategy for malaria control. It is observed from the graph that as more people are treated of malaria and the campaign strategy increases, the number of infected people reduces. It can also be observed that there is a sharp decline in the number of infected people up to about 2years after which the number decreases gradually. This indicates high recovery rate from the disease.

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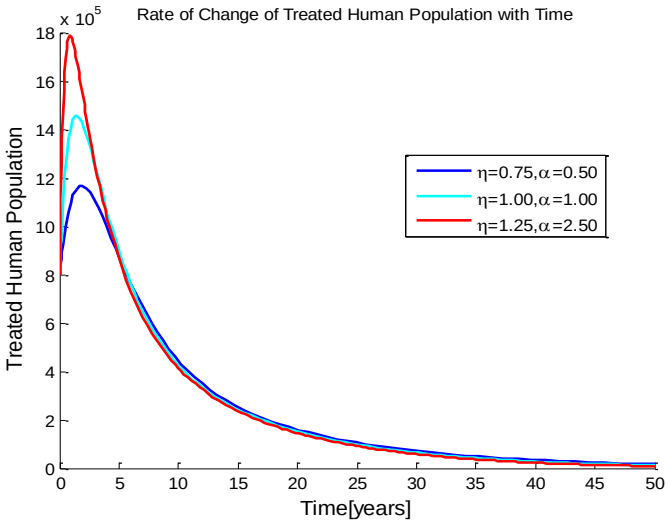

Fig 4.2(d): this shows the response of the population of treated people to increase in treatment rate and campaign strategy. It is observed from the graph that as more people are treated and control strategy increased, the population of treated people increased in the first 2years, and after that begins to reduce drastically. This may be as a result of the diminishing rate of the disease

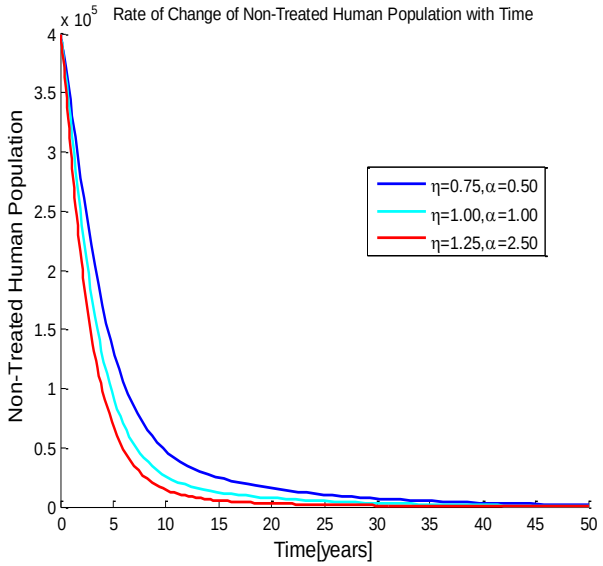

Fig 4.2(e): The graph indicates the response of the population of untreated people to increase in treatment rate and campaign strategy. It is observed from the graph that as the treatment rate and campaign strategy are increased, the untreated human population reduces. The number of untreated people decreases sharply in the first 5years and after that reduces gradually. This may be due to the fact that the untreated people die of the disease

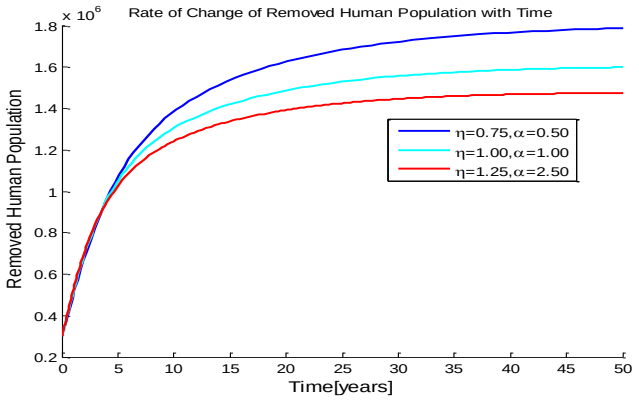

Fig. 4.2(f): This shows the response of the removed human population to increase in treatment rate and campaign strategy for malaria control. It is observed from the graph that as more people are treated of malaria, and the campaign strategy increases, the number of people dying of malaria decreases. The decline in the number of people dying of malaria begins to manifest after 4 years.

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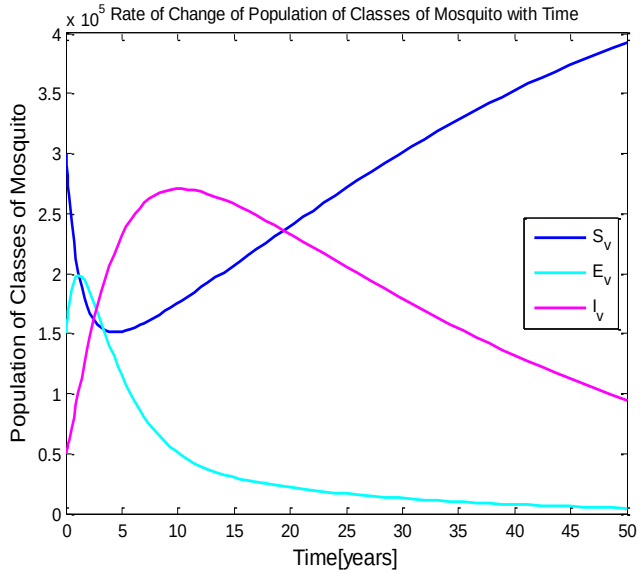

Fig. 4.3: This graph illustrates malaria transmission through the various classes of the vector. These classes are the susceptible vectors, exposed vectors and infected vectors.

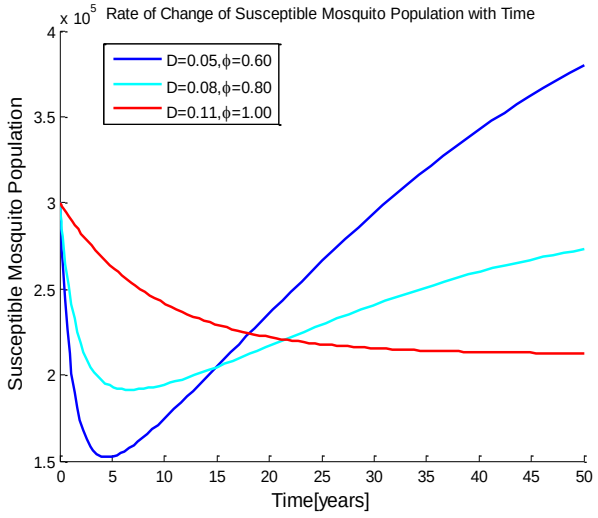

Fig. 4.4(a): This shows the response of susceptible mosquito population to increase in the use of insecticides and destruction of mosquito breeding sites. From the graph, increase of the use of insecticides and destruction of mosquito breeding areas causes decrease of the susceptible mosquito population. This is as a result of mosquitoes dying of insecticides and natural death.

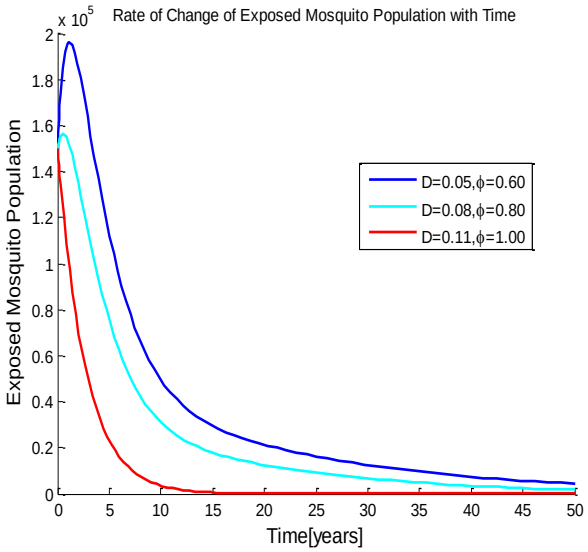

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Fig. 4.4(b): The graph shows the response of the exposed mosquito population to increase in the use of insecticides and destruction of mosquito breeding sites. It is observed from the graph that as more insecticides are used and more mosquito breeding areas are destroyed, the number of exposed mosquitoes is reduced. It can be observed that there is a sharp dectine in the number of mosquitoes exposed to malaria in the first 5 years, followed by gradual decline in the subsequence years.

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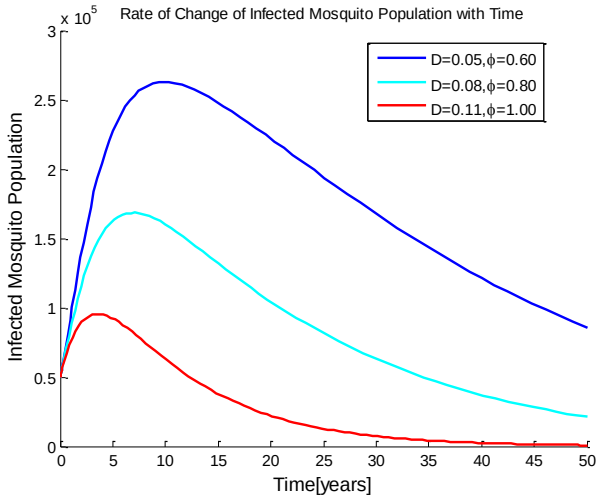

Fig. 4.4(c): The graph indicates the response of infected mosquito population to increase in the use of insecticides and destruction of mosquito breeding sites. It is observed from the graph that as the use of insecticides and destruction of mosquito breeding areas are increased, the number of infected mosquitoes is decreased. It can also be observed that after 45 years the number of infected mosquitoes is tending to O, indicating that the eradication of malaria is imminent

7. **Conclusion**

We formulated and analysed a mathematical model that enables us understand the transmission dynamics of malaria in the population. The model considered human population that incorporated recruitment of individuals into the susceptible class through birth. Our model incorporated effective measures that can control the transmission of malaria such as the use of insecticides, insecticides – treated bed nets, reduction of mosquito breeding sites, treatment with drugs etc.

In our mathematical approach, we modeled malaria as a 9-dimensional system of ordinary differential equations. We showed that there exists a domain where the model is epidemiologically and mathematically well – posed. We defined the basic reproduction number, Ro, as the number of secondary infections that one infectious individual would create over the duration of the infectious period provided that everyone else is susceptible. We proved that if Ro < 1, the disease cannot persist in the community and when Ro > 1, the disease can persist. We performed stability analysis of the model and we proved that the disease – free equilibrium is locally asymptotically stable if Ro < 1 and unstable when Ro >1. A theorem was proved to show that the model has no endemic equilibrium.

The numerical analysis of the model revealed that the most effective strategies for controlling and eradicating malaria are: embarking on awareness programme on malaria control practices such as the use of insecticides and insecticide – treated bed nets; Prompt diagnosis and treatment of infected individuals with drugs; and destruction of mosquito breeding sites by environmental sanitation and fumigation. This study concurs with the intention of the Millennium Development Goal (MDG), stated in Fatimawati and Tasman (2013) that, the MDG has a target of halting the spread of malaria, and to achieve this, it embarked on several interventions such as the use of larvicides, insecticide, mass treatment with drugs and insecticide – treated bed nets to reduce the malaria transmission.

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