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Modeling the Impact of Vector Reduction and Natural Recovery on the Transmission Dynamics of Malaria

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Abstract

A mathematical modeling of the impact of vector reduction and natural recovery on the transmission dynamics of malaria was carried out. We present a deterministic model for the transmission dynamics of malaria in which natural recovery and vector reduction were both important for the disease management. We estimated the basic reproduction number using the next generation matrix method and investigated the local stability of the disease free equilibrium points of the model. Sensitivity analysis and Numerical simulations of the basic reproduction number with respect to the model parameters were carried out. Our result shows that effective vector reduction and increased natural recovery will reduce the spread of malaria.

Keywords: Linearization, Next generation matrix, Stability, Reproduction number, Equlibrium points

Introduction

Malaria, a life-threatening disease which happens to be a vector borne, is one of the major deadly infectious diseases worldwide Woldegerima *et al.* (2018). The disease is caused by the protozoan Plasmodium and it is transmitted in humans by an effective bite of an infected adult female Anopheles mosquito (the malaria vector) Okuneye *et al.* (2017). The species causing agents include Plasmodium falciparum, Plasmodium vivax, Plasmodium ovale, and Plasmodium malariae. These aforementioned species differ in microscopic appearance, geographical distribution, and clinical characteristics. By clinical characteristics, we refer to the infection potential, severity, and the ability to cause relapse. Among all the species, P. falciparum has been recognized as the most dangerous to humans. According to the World malaria report, released in December 2019, there were 228 million cases of malaria in 2018 compared to 231 million cases in 2017. The estimated number of malaria deaths stood at 405 000 in 2018, compared with 416 000 deaths in 2017. The WHO African Region continues to carry a disproportionately high share of the global malaria burden. In 2018, the region was home to 93% of malaria cases and 94% of malaria deaths. The prevalent of malaria is in over 100 countries, with approximately 216 million cases, and 655,000 deaths in 2010 Olaniyi *et al.* (2018). In various parts of the world, malaria has been widespread for many decades Forouzannia *et al.* (2014), yet it still remains a major public health burden in affected areas, predominantly the tropical and subtropical areas in Africa, Eastern Mediterranean Regions, Asia and South America. In addition, the high risk groups include pregnant women, non-immune travelers.

Mathematical Model

Mathematical models for transmission dynamics of malaria are useful in providing better insights into the behavior of the disease. The models have played great roles in influencing the decision making processes regarding intervention strategies for preventing and controlling the insurgence of malaria. The study on malaria using mathematical modeling began in 1911 with Ronald Ross. In his work title "The prevention of malaria", He introduced the first deterministic two-dimensional model with one variable representing human and the other representing mosquitoes where it was shown that reduction of mosquito population below a certain threshold was sufficient to eradicate malaria. In G. Macdonald (1957), the Ross's model was modified by considering the latency period of the parasites in mosquitoes and their survival during that period. However, in this case, it was

shown that reducing the number of mosquitoes is an inefficient control strategy that would have little effect on the epidemiology of malaria in areas of intense transmission. Further extension was described by Anderson and May R.M. Anderson and R.M. May (1991), where the latency of infection in humans was introduced by making the additional exposed class in humans. This modification further reduces the long term prevalence of both the infected humans and mosquitoes. Thus, all other models that exist for malaria dynamics are developed from the three basic models explained earlier by incorporating different factors to make them biologically more realistic in explaining disease prevalence and prediction S. Mandal *et al.* (2011). For examples, a number of epidemiological studies K. Dietz *et al.* (1974), J.L. Aron and R.M. May (1882) and Ngwa *et al.(2000)* considered the inclusion of the recovered class which incorporates a time dependent immunity developed on recovery from infection in humans. Further work on acquired immunity in malaria has been conducted by J.L. Aron (1988) and Bailey (1982). Their models take into account that acquired immunity to malaria depends on continuous exposure to reinfection. Moreover, some models have integrated other factors such as: environmental effects Welch *et al. (2002),* Yang (2000) and Ferreira *et al.* (2000), mosquito's resistance to insecticides and resistance of some parasite strains to anti-malaria drugs. Okosun *et al. (2011).*

Among others, the first symptoms of malaria include fever, headache and chills usually appear in 10–15 days after the infective mosquito bite and may be mild and difficult to recognize as malaria world health organization, (2020). While Gbenga *et et al.* (2018) observed that malaria starts with an extreme cold, followed by high fever and severe sweating. These symptoms can be accompanied by joint pain, abdominal pain, headache, vomiting, and extreme fatigue.

In order to control the spread of malaria, preventive measures include mosquito-reduction strategies and self-protection against mosquito bites (via the use of insecticide treated bed nets (ITNs); intermittent preventive treatment (IPT), and the reduction of vector population through the destruction of their breeding sites Gimba *et al.* (2017), Hove-Musekwa, *et al.* (2008), Dawaki *et al.* (2007), and Kamgang *et al. (2014).* Studies also revealed other intervention strategies, such as the use of indoor residual spraying (IRS) for killing infected indoors mosquitoes through the use of sterile insect technique Kamgang *et al. (2014)* and the use of anti-malaria drugs to regulate malaria Sang *et al. (2012).* Over the years, several numbers of mathematical models on the transmission dynamics of malaria have been examined. Following the simple S-I-R malaria model of Ross Sang *et al.*

(2012)and Macdonald Forouzannia *et al.* (2014), many researchers have elaborated these models by incorporating different features associated to malaria transmission dynamics and its control. This includes modeling the effect of age groups on the transmission of malaria, as shown by Addawe *et al.* (2012), the use of preventive and therapeutic strategies Kong *et al.* (2012); repeated exposure Gumel *et al.* (2008); impact of climate variables such as temperature and rainfall Dhiman *et al.* (2014)

With increased mosquito biting rate, increasing the proportions of antibodies has lower effect in reducing the burden of malaria Olaniyi *et al. (2013)*. In their research "mathematical model for malaria transmission dynamics in human and mosquito populations" also revealed that an increase in the number of disease-induced death rate of the mosquito reduces the number of infectious human. Therefore, humans need to boost their antibodies production to be able to subdue the invasion of parasites in the bloodstream. The immunity state of the individual, that is, the general health and nutritional status of the infected individual, is a factor for preventing or aiding the occurrence of malaria. Thus, leading a healthy lifestyle and eating right foods can help boost the level of antibodies in humans. It is also important to note that reducing human-mosquito contact rate plays a big role in inhibiting the prevalence of malaria. Hence, we can achieve a malaria-free state by scaling down mosquito biting rate through the regular indoor residual spraying with insecticides, the use of insecticide-treated bed nets, closing of doors and windows against mosquitoes, clearing of stagnant water and drainages, and the use of mosquito repellant lotions, which are all regarded as vector control measures. However, recent work has shown that some of the factors, as the age structure of mosquitoes population and climate effects, are very important for a better understanding of malaria transmission global dynamics, Beck-Johnson *et al.* (2013). Indeed, mosquitoes undergo complete metamorphosis going through four distinct stages of development during a lifetime: egg, larva, pupa, and adult. While it is appropriate to assume that only adult mosquitos are involved in the malaria transmission, the dynamics of the juvenile stages (larvae and pupae) has significant effects on the dynamics of the mosquito population, and then the malaria transmission global dynamics. Motivated by this work, and using the malaria model in Ducro *et al.* (2009) as our baseline model, we include the four distinct metamorphic stages of mosquito to formulate a mosquito-stage-structured autonomous model of malaria spread in a more general setting.

Model Formulation and Description

According to Segun *et al.* (2020), the model sub-divides the total human population at time (t), denoted by $N_h(t)$, into susceptible humans (S_h), infected humans (I_h), and recovered humans (R_h).

$$N_h(t) = S_h + I_h + R_h \quad (1)$$

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

$$N_h(t) = A_v + S_v + I_v \quad (2)$$

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

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

Table 1. Description of parameters & variable

Parameters	Description
R_h	Recovered Humans
S_h	Susceptible Humans
I_h	Infected Humans
A_v	Immature Mosquitoes

S_v	Susceptible Mosquitoes
I_v	Infected Mosquitoes
π_h	Recruitment rate of humans
ψ	rate of loss of immunity in humans
μ_h	Natural mortality rate of humans
τ_h	Recovery rate of infectious individuals
δ_h	Disease induced death rate of humans
β_{hv}	Probability of effective transmission from human to mosquito
β_{vh}	Probability of effective transmission from mosquito to human
$\varepsilon(b)$	Contact rate of mosquito with human
$\varepsilon_{(max)}$	Maximum mosquito biting rat
$\varepsilon_{(min)}$	Minimum mosquito biting rate
b	Proportion of treated net usage
q	Proportion of insecticide spray on the environment
η	Modification parameter
π_v	Egg deposition rate of mosquitoes
γ_v	Maturation rate of immature mosquitoes
μ_v	Natural mortality rate of mosquitoes
$\mu_{max}(b)$	Mortality rate of mosquitoes due to treated net
$\mu_{max}(q)$	Mortality rate of mosquitoes due to insecticide spray
N	Natural recovery
$\kappa_1, \kappa_2, \kappa_3$	Vector reduction parameters

Existing Model Equation by Segun *et al.* (2020)

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

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

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

$$\frac{dA_V}{dt} = \pi_v(q) - (\gamma_v + \mu(q))A_v$$

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

$$\frac{dI_V}{dt} = \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))I_v \qquad (11)$$

We assume that infected human population recovers naturally at a rate n and become susceptible for possible infection. We incorporate vector reduction parameters k_1, k_2, k_3 on the immature, susceptible and infected mosquito population and as such, we have the following modified model equation

Modified Model equation of Segun*etal.,* (2020)

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

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

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

$$\frac{dA_V}{dt} = \pi_v(q) - (\gamma_v + \mu(q))A_v - k_1 A_v$$

$$\frac{dS_V}{dt} = \gamma_v A_v - \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))S_v - k_2 S_v$$

$$\frac{dI_V}{dt} = \lambda_v(b)S_v - (\mu_v(b) + \mu_v(q))I_v - k_3 I_v \qquad (12)$$

Positivity of Solution

Here, for the malaria model (12) to be epidemiologically meaningful, it is important to establish that all its state variables are non-negative for all time $t \geq 0$. In other words, solutions of the model system (12) with non-negative initial condition will remain non-negative for all time $t > 0$.

Theorem 1. Let the initial conditions for the malaria model (12) be $S_h(0) > 0$, $I_h(0) \geq 0$, $R_h \geq 0$, $A_v > 0$, $S_v > 0$, $I_v \geq 0$. Then the solutions (S_h, I_h, R_h, A_v, S_v, I_v) of the model with positive initial conditions will remain positive for all time $t > 0$.

Proof. Let $t_1 = \sup\{t > 0 : S_h(t) > 0, I_h(t) > 0, R_h(t) > 0, A_v(t) > 0, S_v(t) > 0, I_v(t) > 0 \in [0,t]\}$. Thus, $t_1 > 0$. It follows from the first equation of the system (12), that

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

Using the integrating factor, this can be writing as:

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

Hence,

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

so that,

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

Similarly, it can be shown that $I_h(t) \geq 0$, $R_h(t) \geq 0$, $A_v(t) > 0$, $S_v(t) > 0$, and $I_v(t) \geq 0$ for all time $t > 0$. Therefore, all the solutions to the model equation (12) remain positive for all non-negative initial conditions.

Disease Free Equilibrium of the Modified Model

At disease free Equilibrium (*DFE*), we s*et all* the rates of change of the equations in the model to zero.

It is assumed that there is no infection at this time, therefore,

$$\frac{dS_h}{dt}=\frac{dI_h}{dt}=\frac{dR_h}{dt}=\frac{dA_V}{dt}=\frac{dS_V}{dt}=\frac{dI_V}{dt}=0$$

Solving equation (12), we have;

$$E^0=\left(S_h^0,I_h^0,R_h^0,A_v^0,S_v^0,I_v^0\right)=\left(\frac{\pi_h}{\mu_h},0,0,\frac{\pi_v(q)}{(\gamma_v+\mu_v(q))+k_1)},\frac{\gamma_v\pi_v(q)}{(\gamma_v+\mu_v(q)+k_1)(\mu_v(b)+\mu_v(q)+k_2)},0\right)$$

The Basic Reproduction Number (R_o) of the Modified Model

The basic reproduction number R_o measures the average number of new infections generated by a single infected person during his or her infectious period in a population that is fully susceptible Diekmann *et al.* (1990). One can easily predict if an infection will spread in exponential progression, die off after some time or remain constant with no

further spread judging from the value of the reproduction number. When $R_o < 1$, the disease will die off because every infected person will transmit the disease to less than one person in the transmittable period. When $R_o = 1$, the disease will become endemic and will stay with each infected person transmitting to one new person. When $R_o > 1$, a disease will spread and the infected people will grow exponentially which will in the end lead to a pandemic. Using next generation method, the basic reproduction number (R_o) is given as $R_0 = \rho(FV^{-1})$ where ρ is the spectral radius.

Given the matrices F and V below,

$$F = \begin{pmatrix} 0 & \beta_{hv}\varepsilon(\mathrm{b}) \\ \dfrac{\beta_{vh}\varepsilon(\mathrm{b})S_v}{S_h} & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \tau_h + \mu_h + \delta_h + n & 0 \\ 0 & \mu_v(b) + \mu_v(q) + k_3 \end{pmatrix}$$

From V above,

$$V^{-1}\begin{pmatrix} \dfrac{1}{\alpha_1} & 0 \\ 0 & \dfrac{1}{\alpha_2} \end{pmatrix}$$

$$FV^{-1} = \begin{pmatrix} 0 & \beta_{hv}\varepsilon(\mathrm{b}) \\ \dfrac{\beta_{vh}\varepsilon(\mathrm{b})S_v}{S_h} & 0 \end{pmatrix}\begin{pmatrix} \dfrac{1}{\alpha_1} & 0 \\ 0 & \dfrac{1}{\alpha_2} \end{pmatrix} = \begin{pmatrix} 0 & \dfrac{\beta_{hv}\varepsilon(\mathrm{b})}{\alpha_2} \\ \dfrac{\beta_{vh}\varepsilon(\mathrm{b})S_v}{S_h\alpha_1} & 0 \end{pmatrix}$$

At disease free equilibrium, we obtain the characteristics polynomial given below,

$$\lambda^2 - \frac{\beta_{vh}\varepsilon^2(\mathrm{b})\beta_{hv}\gamma_v\pi_v(\mathrm{q})\mu_h}{\gamma_v\left((\mu_v(b)+\mu_v(q)+k_2)+\mu_v(q)(\mu_v(b)+\mu_v(q)+k_2)+k_1\pi_h\alpha_1\alpha_2(\mu_v(b)+\mu_v(q)+k_2)\right)}$$

$$\lambda^2 = \frac{\beta_{vh}\varepsilon^2(\mathrm{b})\beta_{hv}\pi_v(\mathrm{q})\mu_h}{\pi_h\alpha_1\alpha_2\alpha_3\alpha_4}$$

Solving the polynomial for eigen values we obtain

$$\lambda = \pm\sqrt{\frac{\beta_{vh}\varepsilon^2(\mathrm{b})\beta_{hv}\gamma_v\pi_v(\mathrm{q})\mu_h}{\pi_h\alpha_1\alpha_2\alpha_3\alpha_4}}$$

We now have the reproduction number below;

$$R_0 = \sqrt{\frac{\beta_{vh}\varepsilon^2(\mathrm{b})\beta_{hv}\pi_v(\mathrm{q})\mu_h}{\pi_h\alpha_1\alpha_2\alpha_3\alpha_4}}$$

Where $\alpha_1 = \tau_h + \mu_h + \delta_h + n$

$$\alpha_2 = \mu_v(b) + \mu_v(q) + k_3$$

$$\alpha_3 = \gamma_v + \mu_v(q) + k_1$$

$$\alpha_4 = \alpha_4 = \mu_v(b) + \mu_v(q) + k_2$$

Local Stability of Disease Free Equilibrium

Theorem 2: The disease free equilibrium of the model equation is locally stable if $R_o < 1$ and unstable if $R_o > 1$.

Proof: To determine the local stability, the Jacobian matrix is computed and evaluated at the disease free equilibrium point.

$$E^0 = \left(\frac{\pi_h}{\mu_h}, 0, 0, \frac{\pi_v(q)}{(\gamma_v + \mu_v(q)) + k_1)}, \frac{\gamma_v\pi_v(q)}{(\gamma_v + \mu_v(q) + k_1)(\mu_v(b) + \mu_v(q) + k_2)}, 0\right)$$

$$J = \begin{pmatrix} -\mu_h & n & \psi & 0 & 0 & \frac{\beta_{hv}\varepsilon(\mathrm{b})\pi_h}{N_h\mu_h} \\ 0 & -\alpha_1 & 0 & 0 & 0 & 0 \\ & \tau_h & -C_1 & 0 & 0 & 0 \\ 0 & 0 & 0 & -C_2 & 0 & 0 \\ 0 & \frac{\beta_{vh}\varepsilon(\mathrm{b})\pi_v(\mathrm{q})}{N_h\alpha_3} & 0 & \gamma_v & -C_3 & 0 \\ 0 & \frac{\beta_{vh}\varepsilon(\mathrm{b})\pi_v(\mathrm{q})}{N_h\alpha_3} & 0 & 0 & 0 & -C_4 \end{pmatrix}$$

At disease free equilibrium, we have the matrix

$$J=\begin{pmatrix} -\mu_h & n & \psi & 0 & 0 & \dfrac{\beta_{hv}\varepsilon(\mathrm{b})\pi_h}{\mu_h(q)} \\ 0 & -\alpha_1 & 0 & 0 & 0 & 0 \\ & \tau_h & -C_1 & 0 & 0 & 0 \\ 0 & 0 & 0 & -C_2 & 0 & 0 \\ 0 & \dfrac{\beta_{vh}\varepsilon(\mathrm{b})\pi_v(\mathrm{q})\mu_h}{\pi_h\alpha_3} & 0 & \gamma_v & -C_3 & 0 \\ 0 & \dfrac{\beta_{vh}\varepsilon(\mathrm{b})\pi_v(\mathrm{q})\mu_h}{\pi_h\alpha_3} & 0 & 0 & 0 & -C_4 \end{pmatrix}$$

$$\left|J(\mathrm{E}_0)-\lambda I\right|=\begin{pmatrix} -\mu_h-\lambda & n & \psi & 0 & 0 & \dfrac{\beta_{hv}\varepsilon(\mathrm{b})\pi_h}{\mu_h(q)} \\ 0 & -\alpha_1-\lambda & 0 & 0 & 0 & 0 \\ & \tau_h & -C_1-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & -C_2-\lambda & 0 & 0 \\ 0 & \dfrac{\beta_{vh}\varepsilon(\mathrm{b})\pi_v(\mathrm{q})\mu_h}{\pi_h\alpha_3} & 0 & \gamma_v & -C_3-\lambda & 0 \\ 0 & \dfrac{\beta_{vh}\varepsilon(\mathrm{b})\pi_v(\mathrm{q})\mu_h}{\pi_h\alpha_3} & 0 & 0 & 0 & -C_4-\lambda \end{pmatrix}$$

So that the eigen values of the jacobian matrix is given as;

$$\begin{vmatrix} -\mu_h-\lambda \\ -\alpha_1-\lambda \\ -C_1-\lambda \\ -C_2-\lambda \\ -C_3-\lambda \\ -C_4-\lambda \end{vmatrix}$$

Where $C_1=\psi+\mu_h$

$$C_2=\gamma_v+\mu_v(q)+k_1$$

$$C_3=\mu_v(b)+\mu_v(q)+k_2$$

$$C_4=\mu_v(b)+\mu_v(q)+k_3$$

Since all eigenvalues of the characteristic equation are negative, it then shows that the disease free equilibrium is locally asymptotically stable.

Sensitivity Analysis

The sensitivity analysis is used to investigate how sensitive the threshold quantity i.e. the basic reproduction number R_o behaves with respect to its parameters. Thus, it enables us to determine the basic reproduction number R_o and parameters that have high impact on R_o. These should be targeted by intervention strategies so as to find the most effective control of the disease. The analysis also tells us how crucial each parameter affects disease transmission. The normalized forward sensitivity index of the reproduction number with respect to natural recovery and the incorporated vector reduction will be computed below.

Definition: supposing a variable 'p' which is differentiable depends on a parameter 'w', then, normalized forward sensitivity index of 'p' with respect to 'w' is denoted by Xp, which is defined as

$$Xp = \frac{p}{w}\frac{\partial w}{\partial p}$$

As we have explicit for R_o, we derive an analytical expression for the sensitivity of R_o as

$$X_w^{R_0} = \frac{dR_0}{dw} \times \frac{w}{R_0}$$

For each parameter involved in R_o, the results of the sensitivity indicesof R_o are as shown in the table below;

Table 2: Numerical values of sensitivity Analysis of the parameters

Parameter	Descriptions	Sensitivity Values
R_0	Reproduction number	0.0002040541639
$\varepsilon(b)$	Contact rate of mosquito with human	1
k_1	Vector reduction parameter from immature mosquitos	0.965000
k_2	Vector reduction parameter from susceptible mosquitos	0.4625800
k_3	Vector reduction parameter from infected mosquitos	0.2355769231
N	Natural recovery	−0.4909740423

β_{vh}	Probability of effective transmission from mosquito to humans	0.50000000
β_{hv}	Probability of effective transmission from human to mosquitoes	0.500000000
π_h	Recruitment rate of humans	-0.50000000
γ_v	Maturation rate of immature mosquitoes	0.50000000
τ_h	Recovery rate of infectious individuals	-0.005018845765
μ_h	Natural mortality rate of humans	0.4961813128
δ_h	Disease induced death rate of humans	-0.0001884249269
$\mu_v(q)$	Death rate of mosquitoes due to insecticidal spray	-0.1201923077
$\mu_v(b)$	Death rate of mosquitoes due to treated net usage spray	-0.1442307692
$\pi_v(q)$	Egg deposition rate of mosquitoes	0.500000000

Numerical Simulation

The numerical behavior of equation (12) is studied using *Maple 18* software with parameters values presented in the table below;

Table 3. Description of parameters with values

Parameter	Descriptions	Sensitivity Values
$\varepsilon(b)$	Contact rate of mosquito with human	0.5
k_1	Vector reduction parameter from immature mosquitos	0.0-1.0
k_2	Vector reduction parameter from susceptible mosquitos	0.0-1.0
k_3	Vector reduction parameter from infected mosquitos	0.0-1.0
N	Natural recovery	0.0-1.0
β_{vh}	Probability of effective transmission from mosquito to humans	0.321

β_{hv}	Probability of effective transmission from human to mosquitoes	0.240
π_h	Recruitment rate of humans	10-100
γ_v	Maturation rate of immature mosquitoes	0.343
τ_h	Recovery rate of infectious individuals	0.0092
μ_h	Natural mortality rate of humans	0.0000421
δ_h	Disease induced death rate of humans	0.0003454
$\mu_v(q)$	Death rate of mosquitoes due to insecticidal spray	0.0555556
$\mu_v(b)$	Death rate of mosquitoes due to treated net usage spray	0.00555556
$\pi_v(q)$	Egg deposition rate of mosquitoes	1.84

Graphical Representation of Sensitivity Analysis and Numerical Simulation

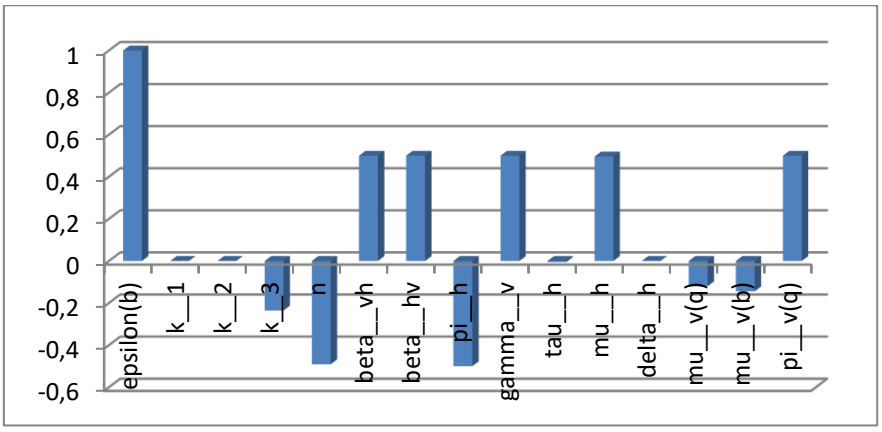

Fig 1 Graph of sensitivity analysis

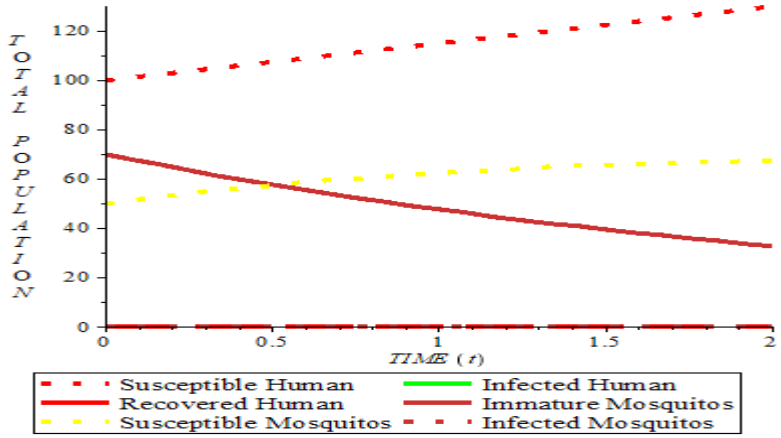

Fig 2 Graph for the total population at disease free equilibrium state (DFE)

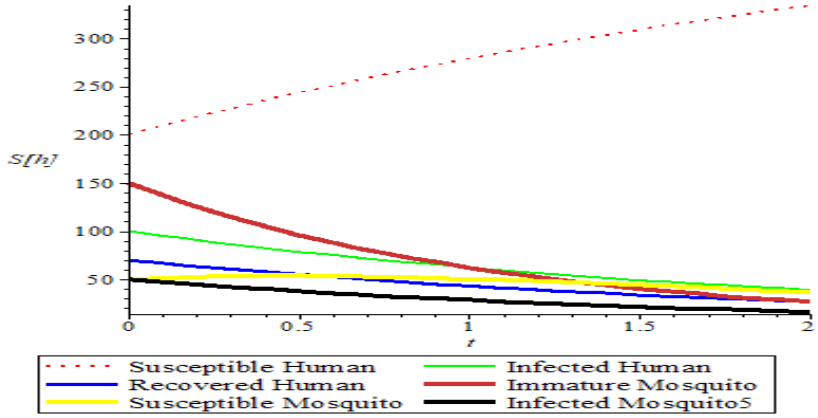

Fig 3 Graph showing the impact of vector reduction and natural recovery on all six compartment of of the model

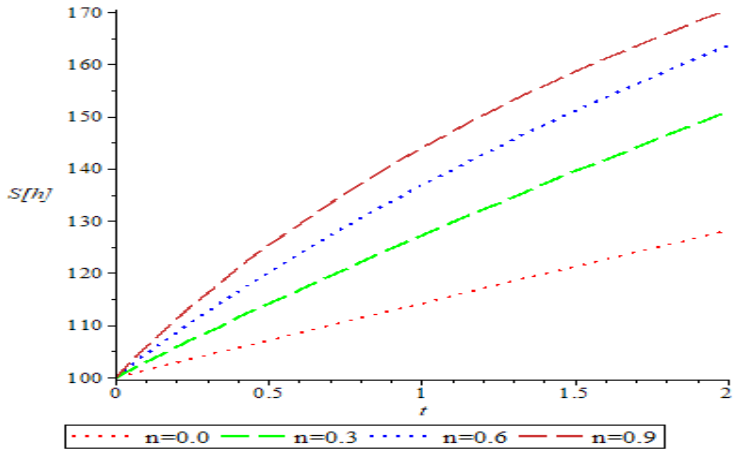

Fig 4 Graph showing the impact of natural recovery at different (0.0, 0.3, 0.6, and 0.9) on the susceptible human population

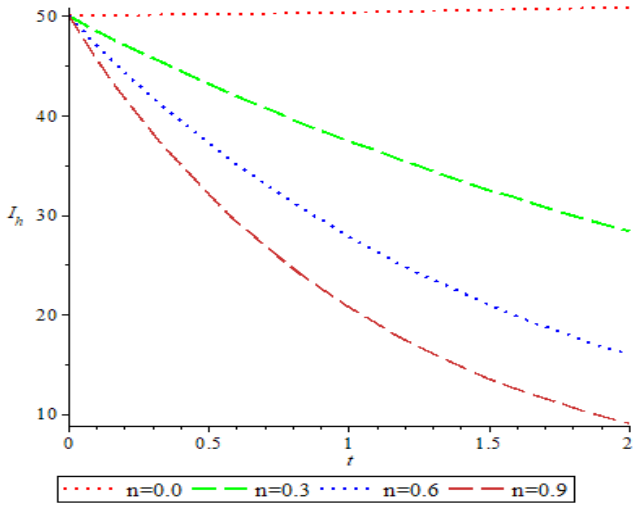

Fig 5 Graph showing the impact of natural recovery at different rate (0.0, 0.3, 0.6, and 0.9) on the infected human population

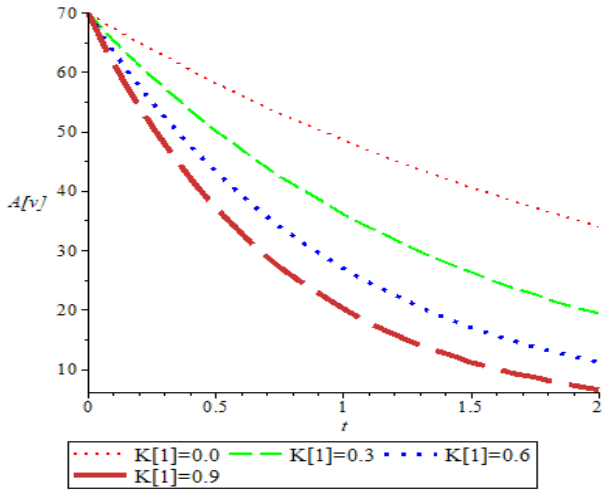

Fig 6 Graph showing the impact of vector reduction on the immature vector population

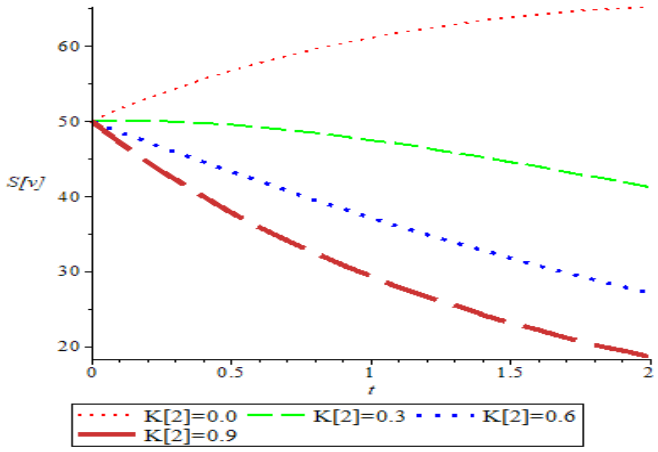

Fig 7 Graph showing the impact of vector reduction on the susceptible vector population

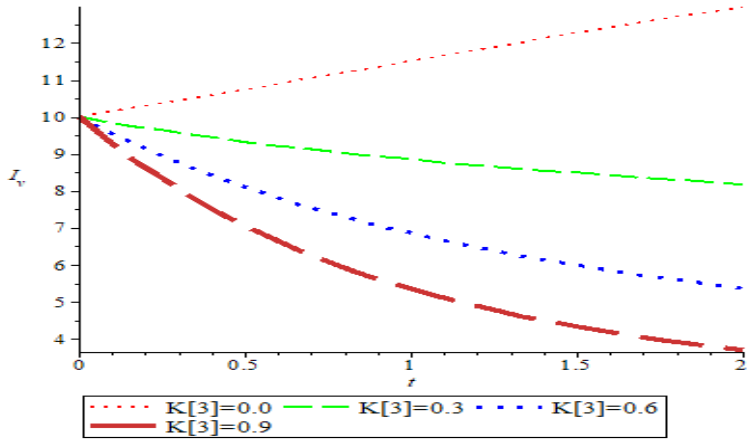

Fig 8 Graph showing the impact of vector reduction on the infected vector populati

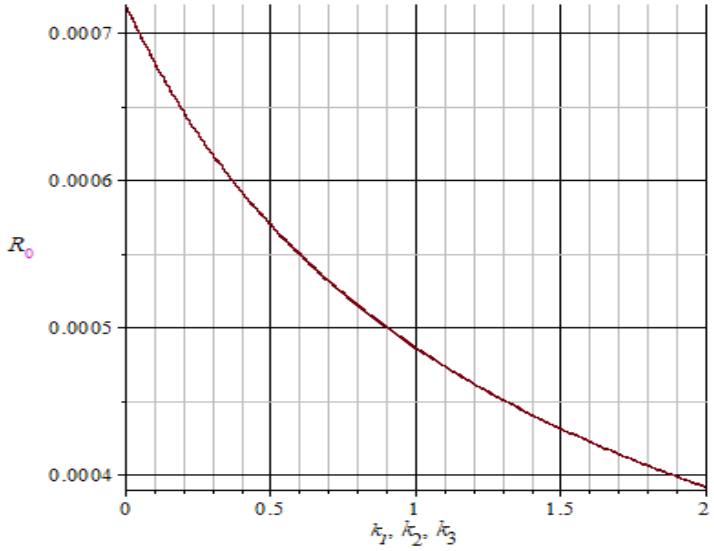

Fig 9 Graph showing the impact of vector reduction (K_1 ,K_2 K_3) on the transmission dynamics of malaria disease

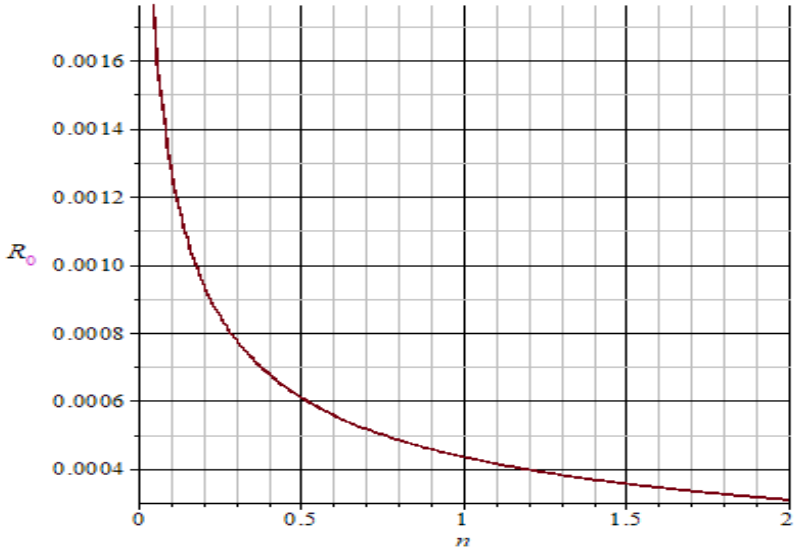

Fig 10 Graph showing the impact of natural recovery (n) on the transmission dynamics of malaria disease

Discussion of Results

In this research, we studied the dynamics of an *SIR-ASI* and modified the existing model of Segun *et al.* (2020) by incorporating vector reduction and natural recovery and applied it to malaria transmission between human and mosquito. We derived the basic reproduction number and discussed the existence and stability of Disease-Free Equilibrium (*DFE*) of the modified model. Our analysis shows that if the reproduction number is less than one then the (*DFE*) is locally asymptotically stable, this implies that only susceptible human immature mosquito, and susceptible mosquito class is present and the other populations reduces to zero, and the disease dies out. And if the reproduction number is greater than one, then (*DFE*) of the model is unstable. This has been verified numerically by simulations in Fig 2. And if the reproduction number is greater than one then the (*DFE*) is unstable, this implies that all the populations exist for the model as shown in Fig 3.

As people recovers naturally from malaria disease and become susceptible again, the infection reduces thereby reducing the population of the infected human class as shown in Fig 4 and 5 respectively.

The analysis also shows that as there is an increase in vector reduction, the number of immature,susceptible and infected mosquitos reduce as shown In Fig 6, 7 and 8 which will surely reduce the spread of malaria disease.

Conclusion

In order to limit the spread and possible elimination of the deadly malaria disease, a six compartmental model with two population categories which include susceptible human, infected human, recovered human, immature mosquitos, susceptible mosquitos and the infected mosquitos was formulated. The result of the basic reproduction number R_o which determines whether malaria disease will die off or become endemic, was carefully calculated using the method of Next Generation Matrix and it was revealed that the disease will become endemic whenever $R_o > 1$and will die off if $R_o < 1$ We notice that in order to reduce the basic reproduction number below one, we need to focus on reduction of vector population and the contact rate of mosquito with human. Sensitivity analysis was performed on basic reproduction number. Numerical simulation was analyzed by *Maple 18* software to analyze the transmission/spread of the disease.

In conclusion, consistent spray of environment and use of insecticidal treated net will reduce the vector population thereby reducing the spread of malaria to the nearest minimum. When the value of vector reduction is high, it reduced the basic reproduction number, since the transmission of disease is dependent on the value of R_o. To this extent, effort should be made toward vector reduction as a control strategy in order to reduce the spread of malaria in our society. In order to have a community that is free of malaria, people should be encouraged to sanitize and spray their surroundings Efforts should be put in place by individuals who are "well-to-do" in the aspect of organizing campaigns in order to inform both the rural and urban dwellers about the importance of insecticidal treated net usage.

The government should make provision for the production of more insecticidal treated net as well as insecticide for her citizens.

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