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Modeling the Dynamics of Endemic Malaria Transmission with the Effects of Control Measure

Dereje Gutema Edossa, Alemu Geleta Wedajo, Purnachandra Rao Koya

Department of Mathematics, Wollega University, Nekemte, Ethiopia

Email address:
derejegutema8@gmail.com (D. G. Edossa), alemugeleta@gmail.com (A. G. Wedajo), drkpraophd@gmail.com (P. R. Koya)

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Abstract: Malaria is an infectious disease caused by Plasmodium parasite and is transmitted among humans through bites of female Anopheles mosquitoes. It is estimated 216 million people suffered from malaria in 2016, with over 400,000 deaths mainly in sub-Saharan Africa. A number of control measures have been put in place: most importantly the insecticide treated net (ITN) and indoor residual sprayings (IRS) of insecticide. Currently, the emergence and spread of resistance in mosquito populations against insecticides is the most common and widely spread .It is also poses a key obstacle to malaria control as well as jeopardizing the effects of the most efficient malaria control interventions. A mathematical model that incorporates the evolution of insecticide resistance and its impact on endemic malaria transmission i.e., effects of indoor residual sprayings (IRS) on the insecticide resistant and sensitive malaria vector strains as a control strategy is incorporated and analyzed. The object of the study is to understand qualitatively the factor that have more influence for the emergence and spread of resistance of malaria vectors against IRS and their impacts on the efficacy of IRS. Based on a Ross-Macdonald derivation of malaria model the effective reproduction number R_e isused to assess the effects of IRS in the qualitative analysis of the model. The existence and stability of the disease-free and endemic equilibria of the model are studied. It is established that the malaria can be brought under control as long as $\Re_e$ is kept below the threshold value. Numerical simulations studies are conducted so as to determine the role played by key parameters of the model. The public health implications of the results include: (i) every effort should be taken to minimize the evolution of insecticide resistance due to malaria control interventions failure and (ii) at least a combination of two types of different control measures and followed by rotation of intervention strategies could be more realistic to minimize the number of resistant malaria vector strains and essential in reducing the malaria burden in the community.

Keywords: Endemic Malaria, Infectious, Insecticide, Emergence, Resistance, Modeling, Mosquito

1. Introduction

Malaria is an infectious disease caused by the Plasmodium parasite and is transmitted among humans through bites of female Anopheles mosquitoes. Also, it is transmitted more infrequently by blood transfusion i.e., needle sharing, surgery and births [1]. Among all the diseases those can be transmit by mosquitoes, malaria has been and still remains the one having the greatest health and socioeconomic impact, from the ancient Egypt to present time [2, 3].

Nearly half of the world's population is at risk of Malaria disease. In 2016, about 216 million people suffered from malaria and among them over 0.4 million people lost their lives. The intensity is still more mainly in sub-Saharan Africa [4].

The main symptoms of malaria include fatigue, chill, headache, abdominal and back pain, diarrhea, sometimes vomiting, and fever.

In recent years global efforts have been made to control and eliminate malaria. This effort has lead to a significant reduction in malaria cases and mortality at rates of 66% and 42%in Africa respectively.

Various techniques have been followed to prevent and cure the Malaria disease. Preventive techniques of Malaria disease include: the use of insecticide-treated nets ITNs, long-lasting insecticidal nets LLINs and indoor residual sprayings IRS [5]. Curing techniques of Malaria disease include: Early diagnosis, improved drug therapies and better health infrastructure.

The 'insecticide resistance' is defined as the ability of an

insect to withstand from the effects of an IRS [7]. Repeated exposure to insecticides may make the insects more rapidly resistant to or are less sensitive to that. These individual survivors could then pass the resistance mechanism to the successive generations resulting in the production of more resistant insect populations [8].

Currently, the resistance against insecticides in mosquito populations is emerging and spreading. This face is disturbing the effectiveness of the most efficient malaria control interventions [6].

Insecticide resistant malaria vector strains limits the effectiveness of control and intervention strategies. This also resulted in higher: malaria morbidity and mortality, increased cost of malaria disease management, increased burden on the health care facilities, and increased relative malaria incidence and parasite infection prevalence within individuals.

Mathematical modeling of malaria has been flourishing since the days of Ronald Ross, in 1911, who was awarded a Nobel Prize for his contributions. A simple SI model having two compartments namely susceptible and infected is developed. This model has a basis on the assumption that at any time, the total human population can be divided into some distinct compartments. The so called mathematical model is used to show that bringing mosquito population below a certain threshold is sufficient to eliminate malaria. This threshold naturally depends on biological factors such as the biting rate and vectors capacity [9].

Macdonald G. has developed a model for estimating the infection and recovery rates. This model assumes that the amount of infective material to which a population is exposed remains unchanged. It is shown that the reduction of the number of mosquitoes is an inefficient control strategy since that had a little effect on the dynamics of malaria in areas of strong transmission [9, 10].

The Ross – Macdonald developed a reformulated model an interaction between infected human hosts and infected mosquito vectors that identifies mosquito vector longevity as the single most important variable in the force of transmission, and combined Ross's model with epidemiological and entomological file data Since then, study developed on mosquito-borne pathogen transmission and designed strategies for mosquito-borne disease prevention [11, 12, 13, 14, 15, 16, 17].

The models developed by Aronhave considered that acquired immunity to malaria depends on exposure i.e. the immunity is boosted by additional infections [18, 19].

Recently, Tumwiine, Mugisha and Luboobi have developed a compartmental model to formulate the spread of malaria, with susceptible – Infected – Recovered - Susceptible SIRS pattern for human and Susceptible – Infected SI pattern for mosquitoes [20, 21].

Yang, Wei and Li have developed a model consisting of SIR compartments for human and SI compartments for vector populations respectively. The concept of reproduction number R_0 is used. Further, the existence and stability of disease-free and an endemic equilibria are proposed [22].

Fekadu Tadege Kobe and Purnachandra Rao Koya have developed a model and shown that the spread of malaria disease can be controlled using effective intervention strategies [23].

Mathematical model is a valuable tool in the study of the dynamics of diseases. It provides the abilities of understanding and predictions of epidemiological patterns and dynamical nature of diseases.

Ross – Macdonald model focused on only one factor that mosquito vector longevity as the single most important variable. This variable is used in the force of transmission of the disease and strategies for mosquito-borne disease prevention.

Some other important issued missed to consider are:(i) complex dynamics of the host-vector interactions (ii) evolution of insecticide resistance (iii) factors that influence the resistance of malaria vectors against insecticides and (iv) the efficacy of control measures.

In the present model few of the listed factors have been incorporated and thus extended the Ross – Macdonald model. Here, the human population is compartmentalized as susceptible-infective-immune SIR and mosquito population is compartmentalized as susceptible-infective SI. Further, the infected mosquito population is divided into two classes: (i) insecticide sensitive and (ii) insecticide resistant. Also, considered that the infection with the insecticide sensitive strain will give rise the insecticide resistant strain in the event of indoor residual spray IRS fails to kill mosquito vectors.

2. Model Formulation and Analysis

2.1. Model Formulation

In this section, an improved mathematical model for the transmission and spread of malaria disease between two interacting populations of humans or the host and mosquitoes or the vector is developed. This model is an extension of that of Ross – Macdonald.

This model compartmentalizes the total human population denoted by $N_h(t)$at time t into three classes: susceptible$S_h(t)$, infected $I_h(t)$, and immune $R_h(t)$ classes. Hence, the total human population is given by$N_h(t) = S_h(t) + I_h(t) + R_h(t)$.

Similarly, the total mosquito population is divided into two classes: Susceptible $S_v(t)$ and infectious $I_v(t)$. Further, the infected mosquito population classified as: insecticide sensitive malaria vectors $I_{vs}$and insecticide resistant malaria vectors I_{vr}. Here, the subscripts s and r are added to the vector variables in order to specify the sensitive and resistant strains respectively. Thus, the total mosquito population at any time t is denoted and given by $N_v(t) = S_v(t) + I_{vs}(t) + I_{vr}(t)$.

The mosquitoes do not have any recovered class since the infected mosquitoes remain infectious for whole life. The present model ignores super-infections for mosquitocompartments and the latent periods of the disease for both human and mosquitocompartments.

The assumptions of the present model include the following: Individuals are recruited into the susceptible class with a rate of Λ_h due to births and/or immigrations. Both insecticide sensitive and infected insecticide resistant malaria

vectorsattack susceptible human with the probabilities of β_h and $(1-\beta_h)$ respectively [24].

In the process, the parasite injects sporozoites into the blood and move to the infectious compartment $I_h(t)$. Infectious humans recover due to immunity resistance with a rate of γ. Since immunity against malaria infection is temporary, it is lost at a constant rate θ and the immune humans become susceptible again. All humans are subjected to a non-disease related per capita natural death rate μ_h. Additionally, disease related deaths occur with a constantrate of$\delta_h$in the infective class.

In mosquito population the birth rate is considered to be a constant Λ_v per capita. Thesusceptible mosquito becomes infected with a probability β_v from biting infectious human $I_h(t)$. Let a fraction of ρ susceptible mosquitoes moveto insecticide sensitive malaria vector strains and the remaining fraction $(1-\rho)$ moveto insecticide resistant malaria vector strains, where $0 \leq \rho \leq 1$.

The effect of IRS is incorporated solely for the death of the mosquito populations. That is, both the susceptible female mosquitoes S_v and insecticide sensitive malaria vector strains I_{vs} are expected to be killed by IRS. The parameter α is the rate of removal of mosquitoes from different classes associated with IRS. The values of IRSapplied per day ranges from 0 to 1. The amount of IRS used is incremented by a constant so as to account for a wide range of efficacies and compliancesapplicable.

The evolution of insecticide resistancedue to improper use of IRSleading to the control intervention failure is also incorporated in the model. Thus, the rate at whichinsecticide sensitive malaria vector strains progress to theinsecticide resistant malaria vector strains class is considered to be φ. All mosquitoes are subjected to a non-diseasednatural death rate $\mu_v$per capita. Additionally, disease related death rate is considered to be a constant δ_v in both the sensitive and resistant infective classes.

2.2. Model Assumptions

The formulation of the present model is guided by the following assumptions:

(i) The total sizes of both humans and mosquito populations are not constant.
(ii) The mosquito populations cannot be completely eliminated and thus there will be ongoing transitions of the disease.
(iii) Insecticideresistant malaria vector strains will affect both mosquito populations and the effectiveness of control intervention and hence maximizes the potential of disease transmission.
(iv) The emergency of insecticide resistant malaria vectorstrainisbased on quality (improper usage) and quantity (types) of control interventions.
(v) Spraying of IRS on the places where mosquitoes reproduce and on the home walls leads to the death of mosquito populations.
(vi) On recovery, humans will havetemporary immunity.
(vii) The number of insecticide resistant vectors will increase with time due to the failure of effectiveness of IRS on sensitive vectors.
(viii) The populations in compartmentsof both humans and vectors are non-negative and so areall the parameters involved in the model.
(ix) Malaria isactivein a population for a long period of time.

Table 1. *Description of state variables.*

State Variable	Description
S_h (t)	Number of Susceptible Humans
I_h (t)	Number of Infected Humans
R_h(t)	Number of Recovered Humans
S_v(t)	Number of Susceptible Mosquitoes
I_{vs}(t)	Number of Infected insecticide sensitive Mosquitoes
I_{vr}(t)	Number of Infected insecticide resistant Mosquitoes

Table 2. *Description ofmodelparameters.*

Parameter	Description
Λ_h	Recruitment rate of Susceptible Humans
Λ_v	Recruitment rate of Susceptible Mosquitoes
μ_h	Natural death rate of Humans
δ_h	Disease-induced death rate of Humans
β_h	Contact rate of Infective Vector and Susceptible Humans
γ	Recovery rate of Infective Humans
μ_v	Natural death rate of Mosquitoes
δ_v	Disease-induced death rate of Humans
θ	Rate of loss of immunity in Humans
β_v	Contact rate of Susceptible Mosquitoes and Infective Humans
α	Rate of prevention of Malaria vector using IRS

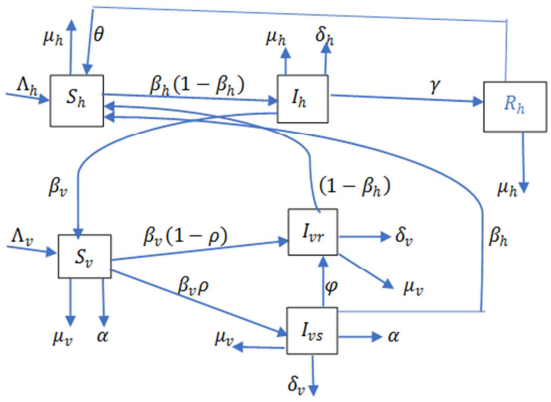


Figure 1. *Flow of Malaria parasite between Humans and Mosquitoes.*

Based on the model assumptions, description of model parameters and the state variables and the flow chart given in the Figure 1, the system of model equations can be developed as follows:

$$dS_h/dt = \Lambda_h + \theta R_h - [\beta_h(1-\beta_h)(I_{vs}+I_{vs})S_h]/N_h - \mu_h S_h \quad (1)$$

$$dI_h/dt = [\beta_h(1-\beta_h)(I_{vs}+I_{vs})S_h]/N_h - (\gamma + \mu_h + \delta_h)I_h \quad (2)$$

$$dR_h/dt = \gamma I_h - (\mu_h + \theta)R_h \quad (3)$$

$$dS_v/dt = \Lambda_v - [\beta_v S_v I_h]/N_v - (\alpha + \mu_v)S_v \quad (4)$$

$$dI_{vs}/dt = [[\rho(1-\varphi)]\,\beta_v S_v I_h]/N_v - (\alpha + \mu_v + \delta_v)I_{vs} \quad (5)$$

$$dI_{vr}/dt = [(1-(1-\varphi)\rho)\beta_v S_v I_h]/N_v - (\mu_v + \delta_v)I_{vr} \quad (6)$$

$$dN_h/dt = \Lambda_h - \mu_h N_h - \delta_h I_h \quad (7)$$

$$dN_v/dt = \Lambda_v - \mu_v N_v - \alpha(S_v + I_{vs}) - \delta_v(I_v s - I_{vr}) \quad (8)$$

With initial conditions

$$S_h(0) = S_{0h}, I(0) = I_{0h}, R_h(0) = R_{0h}, S_v(0) = S_{0v}, N_v(0) = N_0 v I_{vs}(0) = I_{0vs}, I_{vr}(0) = I_{0vr}, N_h(0) = N_{0h}, \quad (9)$$

2.3. Model Analysis

2.3.1. Existence and Positivity of Solutions

In this section, it is shown that the malaria model governed by the system of equations (1) to (6) is epidemiologically and mathematically well posed. Specifically, the feasible region is identified as $\Omega = \{\Omega_h \times \Omega_v\} \subset \{\mathbb{R}_+^3 \times \mathbb{R}_+^3\}$ where $\Omega_h = \{(S_h, I_h, R_h) \in \mathbb{R}_+^3 : N_h \leq (\Lambda_h/\mu_h)\}$ and $\Omega_v = \{(S_v, I_{vs}, I_{vr}) \in \mathbb{R}_+^3 : N_v \leq [\Lambda_v/\mu_v]\}$.

Theorem 1 The solution $\{S_h, I_h, R_h, S_v, I_{vs} I_{vr}\}$ of the system of equations (1) to (6) is bounded and contained in the domain Ω.

Proof: Let the solution of the system of equations (1) to (6) together with positive the initial conditions given in (9) are $\Omega = \{S_h, I_h, R_h, S_v, I_{vs} I_{vr}\}$. Also, let $N_h(t) = S_h(t) + I_h(t) + R_h(t)$ and $N_v(t) = S_v(t) + I_{vs}(t) + I_{vr}$. Now, in order to show that both the human and mosquito populations are bounded it is enough to show that the respective two total populations i.e., $N_h(t)$ and $N_v(t)$ are bounded.

Boundedness of $N_h(t)$: Addition of human compartments of the system of equations (1) to (3) leads to $dN_h/dt = \Lambda_h - \mu_h N_h(t) - \delta_h I_h(t)$. After dropping the negative term $-\delta_h I_h(t)$ appearing on the right-hand side the fore going equation can be expressed without loss of generality as an inequality as $dN_h/dt \leq \Lambda_h - \mu_h N_h(t)$ or equivalentl yas $dN_h/dt + \mu_h N_h(t) \leq \Lambda_h$. It is a first order linearordinary differential equation and has the generals olution $N_h(t) \leq (\Lambda_h/\mu_h) + A exp(-\mu_h t)$. Here, the integral constant A takes theform, on applying the initial conditions as $A = [N_{0h} - (\Lambda_h/\mu_h)]$. Hence, the complete solution is given by $N_h(t) \leq (\Lambda_h/\mu_h) + [N_{0h} - (\Lambda_h/\mu_h)] exp(-\mu_h t)$. Now, clearly it can be observed that $N_h(t) \leq (\Lambda_h/\mu_h)$ as $t \to \infty$ and also, according to the initial conditions $N_h(t) = N_h$ at the initial time $t = 0$. Hence, the total human population is bounded i.e. $N_{0h} \leq N_h(t) \leq (\Lambda_h/\mu_h)$.

Boundedness of $N_v(t)$: Addition of mosquito compartments of the system of equations (1) to (6) leads to $d N_v/dt = \Lambda_v - \mu_v N_v - \alpha(S_v + I_{vs}) - \delta_v(I_v s + I_{vr})$. After dropping the negative term $-\alpha(S_v + I_{vs}) - \delta_v(I_{vs} + I_{vr})$ appearing on the right-hand side, the fore going equation can be expressed without loss of generality as an inequality as. $dN_v/dt \leq \Lambda_v - \mu_v N_v(t)$, or equivalently it is $dN_v/dt + \mu_v N_v(t) \leq \Lambda_v$. It is a first order linear ordinary differential equation and has the general solution $N_v(t) \leq [\Lambda_v/\mu_v] + B exp[(-\mu_v)t]$. Here, the integral constant Btakes the form, on applying the initial condition, as $B = [N_{0v} - (\Lambda_v/\mu_v)]$. Hence, the complete solution is given by $N_h(t) \leq (\Lambda_v/\mu_v) + [N_{0v} - (\Lambda_v/\mu_v)] exp(-\mu_v t)$. Now, clearly it can be observed that $N_v(t) \leq [\Lambda_v/\mu_v]$ as $t \to \infty$ and also according to the initial condition$N_0(t) = N_{0v}$at the initial time$t = 0$. Hence, the total mosquito population is bounded i.e. $N_{0v} \leq N_v(t) \leq [\Lambda_v/\mu_v]$.

Thus, the solutions of the model variables representing human populations$\{S_h(t), I_h(t), R_h(t)\}$ are confined in the feasible region $\Omega_h = \{(S_h, I_h, R_h) \in \mathbb{R}_+^3 : N_h \leq (\Lambda_h/\mu_h)\}$. Similarly, the solutions of the model variables representing mosquito populations$\{(S_v, I_{vs} I_{vr})\}$ are confined in the feasible region $\Omega_v = \{(S_v, I_{vs} I_{vr}) \epsilon \mathbb{R}_+^3 : N_v \leq [\Lambda_v/\mu_v]\}$. This shows that the feasible region of the model equations (1) to (6) is bounded and is given by $\Omega = \{S_h(t), I_h(t), R_h(t), S_v(t), I_{vs}(t) I_{vr}(t)\} \in \mathbb{R}_+^6$ or equivalently $\Omega = \{\Omega_h \times \Omega_v\} \subset \{\mathbb{R}_+^3 \times \mathbb{R}_+^3\}$.

Positivity of the model equations is verified. The results are stated and proved in the form of a theorem as follows:

Theorem 2: The solutions $\{S_h(t), I_h(t), R_h(t), S_v(t), I_{vs}(t) I_{vr}(t)\}$ of the malaria model given in equations (1) to (6) together with the non-negative initial conditions given in (1)are all non-negative for all $t > 0$.

Proof:

Positivity of S_h : Consider the equation for susceptible humans from the system of equations (1) to (6) i.e. $dS_h/dt = \Lambda_h + \theta R_h - [\beta_h(1-\beta_h)(I_{vs} + I_{vs})S_h]/N_h - \mu_h S_h$. After dropping the positive terms Λ_h and θR_h appearing on the right-hand side, the fore going equation can be expressed without loss of generality as an inequality $dS_h/dt \geq -S_h[\beta_h(1-\beta_h)(I_{vs} + I_{vr})]/N_h - \mu_h S_h$. Also$N_h$satisfies the boundary condition

$N_h \leq (\Lambda_h/\mu_h)$ as showninTheorem1Thus, it can be equivalently expressed as:

$dS_h/dt \geq -S_h[[[\beta_h(1-\beta_h)(I_{vs} + I_{vr})]\mu_h/\Lambda_h] + \mu_h]$. It is a first order linear ordinary differential equation and has the general solution:

$S_h(t) \geq exp[S_{0h} - [[\beta_h(1-\beta_h)(I_{0vs} + I_{0vr})\mu_h/\Lambda_h] + \mu_h]t] \geq 0$. Therefore$S_h(t) \geq 0$ for all$t > 0$.

Positivity of I_h: Consider the equation for infected humans from the system of equations (1) to (6) i.e. $dI_h/dt = [[\beta_h(1-\beta_h)(I_{vs} + I_{vr})]\mu_h/N_h] - (\gamma + \mu_h + \delta_h)I_h$. After dropping the positive term $[\beta_h(1-\beta_h)(I_{vs} + I_{vr})]\mu_h/N_h$ appearing on the right-hand side the fore going equation it can be expressed without loss of generality as an inequality $dI_h/dt \geq -[\gamma + \mu_h + \delta_h]I_h$. It is a first order linear ordinary differential equation and has the general solution. $I_h(t) = exp[I_{0h} - (\gamma + \mu_h + \delta_h)t] \geq 0$. Therefore $I_h(t) \geq 0$ for all $t > 0$.

Positivity of R_h : Consider the equation for recovered humans from the system of equations (1) to (6) i.e. $dR_h/dt = \gamma I_h - (\mu_h + \theta)R_h$. After dropping the positive term γI_h appearing on the right-hand side the fore going equation can be expressed without loss of generality as an inequality $dR_h/dt \geq -(\mu_h + \theta)R_h$. It is a first order linear ordinary differential equation and has the general solution $R_h(t) = exp[R_{0h} - (\mu_h + \theta)t] > 0$. Therefore $R_h(t) \geq 0$ for all $t > 0$.

Positivity of S_v : Consider the equation for susceptible vector from the system of equations (1) to (6) i.e. $dS_v/dt =$

$\Lambda_v - [\beta_v S_v I_h]/N_v - (\alpha + \mu_v)S_v$. After dropping the positive term Λ_v appearing on the right-hand side, the fore going equation can be expressed without loss of generality as $dS_v/dt \geq -S_v[[\beta_v I_h/\mathrm{N_v}] + (\alpha + \mu_v]$. Since $\mathrm{N_v} \leq \Lambda_v/\mu_v$ and hence, it can be equivalently expressed as $dS_v/dt \geq -S_v[(\mu_v\beta_v I_h/\Lambda_v) + (\alpha + \mu_v)]$. It is a first order linear ordinary differential equation and has the general solution $S_v(t) = exp[S_{0v} - [[\mu_v\beta_v I_{0h}/\Lambda_v] + (\alpha + \mu_v)]t] > 0$. Therefore $S_v(t) \geq 0$ for all $t > 0$.

Positivity of I_{vs} : Consider the equation for insecticide sensitive malariavectors from the system of equations (1) to (6) i.e. $dI_{vs}/dt = [\rho(1-\varphi)\beta_v S_v I_h/N_v] - (\alpha + \mu_v + \delta_v)I_{vs}$. After dropping the positive term$[\rho(1-\varphi)\beta_v S_v I_h/N_v]$ which is appearing on the right-hand side, the fore going equation can be expressed without of loss of generality as$dI_{vs}/dt \geq -(\alpha + \mu_v + \delta_v)I_{vs}$. It is a first order linear ordinary differential equation and has solution$I_{vs}(t) = exp[I_{0vs} - (\alpha + \mu_v + \delta_v)t] > 0$. Therefore, $I_{vs}(t) \geq 0$ for all $t > 0$.

Positivity of I_{vr} : Consider the equation for insecticideresistant malaria vectors from the system of equations (1) to (6) i.e. $dI_{vr}/dt = [[1-(1-\varphi)\rho]\beta_v S_v I_h]/N_v - (\mu_v + \delta_v)I_{vr}$. After dropping the positive term $\{[1-(1-\varphi)\rho]\beta_v S_v I_h/N_v\}$which is appearing on the right-hand side, the fore going equation can be expressed without loss of generality as$dI_{vs}/dt \geq -(\mu_v + \delta_v)I_{vr}$. It is a first order linearordinary differential equation and has the general solution $I_{vr}(t) = \exp[I_{0vr} - (\mu_v + \delta_v)t] > 0$. Therefore, $I_{vr}(t) \geq 0$for all $t > 0$.

2.3.2. Existence of Equilibriumpoints

In this section, the model is analyzed quantitatively by investigating the existence and stability of both Disease-free equilibrium E_0 and at endemic equilibriumE^*.

The disease-free equilibrium points of the model are its steady state solutions in the absence of infection or disease. Consider the disease free-equilibrium points denoted by $E_0 = \{S_h^0,\ I_h^0,\ R_h^0,\ S_v^0,\ I_{vs}^0,\ I_{vr}^0\}$. All the components of E_0 are obtained by setting$R_h^0 = 0$, $I_{vs}^0 = 0$, $I_{vr}^0 = 0, I_h^0 = 0$ in the malaria model equations (1) to (6)and solving the resultant equations: $dS_h/dt = 0$ gives $S_h^0 = \Lambda_h/\mu_h$ and similarly $dS_v/dt = 0$ gives$S_v^0 = \Lambda_v/\mu_v$. Thus,

$$E_0 = \{\Lambda_h/\mu_h,\ 0,\ 0,\ \Lambda_v/\mu_v,\ 0,\ 0\} \quad (10)$$

Let the endemic equilibrium point be denoted by $E^* = \{S_h^*,\ I_h^*,\ R_h^*,\ S_v^*,\ I_{vs}^*,\ I_{vr}^*\}$. It is the non-trivial positive equilibrium of the malaria model equations (1) to (6). Eachcomponent of $E^*$is obtained by setting the right hand sides of all equations (1) to (6) equal to zero i.e.

$$\Lambda_h + \theta R_h - [\beta_h(1-\beta_h)(I_{vs} + I_{vs})S_h]/N_h - \mu_h S_h = 0$$

$$[\beta_h(1-\beta_h)(I_{vs} + I_{vs})S_h]/N_h - (\gamma + \mu_h + \delta_h)I_h = 0$$

$$\gamma I_h - (\mu_h + \theta)R_h = 0$$

$$\Lambda_v - [\beta_v S_v I_h]/N_v - (\alpha + \mu_v)S_v = 0$$

$$[(\rho(1-\varphi)\beta_v)S_v I_h]/N_v - (\alpha + \mu_v + \delta_v)I_{vs} = 0$$

$$[(1-(1-\varphi)\rho)\beta_v S_v I_h]/N_v - (\mu_v + \delta_v)I_{vr} = 0$$

Up on computing the resultant equations as listed above, thecomponents of $E^*$are obtained as follows:

$$S_h^* = \frac{\Lambda_h(\mu_h+\theta)+[\theta\gamma-(\mu_h+\theta)(\gamma+\mu_h+\delta_h)]I_h^*}{\mu_h(\mu_h+\theta)} \quad (11)$$

$$I_h^* = \frac{\Lambda_v(\mu_h+\theta)\left[[\Lambda_h\Lambda_v(\gamma+\mu_h+\delta_h)R_e^2]-(\alpha+\mu_v)\right]}{R_e^2\Lambda_v^2[(\mu_h+\theta)(\gamma+\mu_h+\delta_h)-\theta\gamma]+(\mu_h\mu_v\beta_v)(\mu_h+\theta)} \quad (12)$$

$$R_h^* = \frac{\gamma I_h^*}{(\mu_h+\theta)} \quad (13)$$

$$S_v^* = \frac{\Lambda_v}{\beta_v I_h^* + (\alpha+\mu_v)} \quad (14)$$

$$I_{vs}^* = \frac{\Lambda_v\beta_v[(1-\varphi)\rho]}{(\alpha+\mu_v+\delta_v)[\beta_v I_h^* + (\alpha+\mu_v)]} \quad (15)$$

$$I_{vr}^* = \frac{\Lambda_v\beta_v[(1-(1-\varphi)\rho]}{(\mu_v+\delta_v)[\beta_v I_h^* + (\alpha+\mu_v)]} \quad (16)$$

2.3.3. Reproduction Number

The basic reproduction number denoted by R_0 is the average number of secondary infectious infected by an infective individual during his or her whole course of disease in case that all of the population are susceptible [25]. This helps to check whether an infection will spread through the population or die out from the population.

2.3.4. The Effective Reproduction Number

The effective reproduction numberR_e is a key parameter that determines the behavior of the model in the presence of indoor residual spray IRS. The term 'effective reproduction number' is used to distinguish it from the basic reproduction number R_0. The latter is used when there is no indoor residual spray IRS.

In order to analyze the stability of system 2a-fthe threshold condition for the establishment of the disease is required tobe obtained.

Here the effective reproduction number is computed using the next generation operator method that is developed by van den Driessche and Watmough [26]. A reproduction number obtained this way determines the local stability of the disease-free equilibrium point with local asymptotic stability for $R_e < 1$ and instability for $R_e > 1$. Now let the system be rearranged by beginning with the infected classes as follows: Let $X = (\mathrm{I_h},\ \mathrm{I_{vs}},\ \mathrm{I_{vr}},\ \mathrm{S_h},\ R_h,\ S_v)^T$. Then the new infections be distinguished from all other class transitions in the population.

The infected classes are $\mathrm{I_h}$, $\mathrm{I_{vs}}$and $\mathrm{I_{vr}}$among all the classes of both human host and mosquito vector. The vector of rates of the appearance of new infections in each compartment is denoted by F. Further, $V = V^+ + V^-$ where $V^+$is the vector rate of transfer into the particular compartment and $V^-$is the vector rateoftransfer out of the particular compartment. In the model equations it is clear that there are three compartments for the infected. Thus,

$$F(X_i)=\begin{bmatrix}\beta_h(1-\beta_h)S_h(I_{vr}+I_{vs})/N_h\\ \rho\beta_v S_v I_h/N_v\\ [1-(1-\varphi)\rho]\beta_v S_v I_h/N_v\\ 0\\ 0\\ 0\end{bmatrix} \text{ and } V(X_i)=\begin{bmatrix}(\gamma+\mu_h+\delta_h)I_h\\ (\mu_v+\delta_v+\eta)I_{vs}\\ (\mu_v+\delta_v)I_{vr}\\ 0\\ 0\\ 0\end{bmatrix}$$

Now, the matrices F and V at the disease–free equilibrium point E_0 are defined as $F=\frac{\partial F(X_i)}{\partial X_i}(E_0)$ and $V=\frac{\partial V(X_i)}{\partial X_i}(E_0)$. After computation they are obtained as

$$F=\begin{bmatrix}0 & \beta_h(1-\beta_h) & \beta_h(1-\beta_h)\\ [(1-\varphi)\rho]\beta_v & 0 & 0\\ [1-(1-\varphi)\rho]\beta_v & 0 & 0\end{bmatrix}$$

and

$$V=\begin{bmatrix}(\gamma+\mu_h+\delta_h) & 0 & 0\\ 0 & (\mu_v+\delta_v+\alpha) & 0\\ 0 & 0 & (\mu_v+\delta_v)\end{bmatrix}$$

Note that F is nonnegative, V is a nonsingular matrix whose inverse V^{-1}, is nonnegative, and the next generation matrix and FV^{-1} [25, 26] is nonnegative. The dominant eigenvalue corresponding to the spectral radius (FV^{-1}) of the matrix

$$FV^{-1}=\begin{bmatrix}0 & \frac{\beta_h(1-\beta_h)}{(\mu_v+\delta_v+\alpha)} & \frac{\beta_h(1-\beta_h)}{(\mu_v+\delta_v)}\\ \frac{[(1-\varphi)\rho]\beta_v}{(\gamma+\mu_h+\delta_h)} & 0 & 0\\ \frac{[1-(1-\varphi)\rho]\beta_v}{(\gamma+\mu_h+\delta_h)} & 0 & 0\end{bmatrix}$$

FV^{-1} is given by computing the corresponding characteristic equation$\det(FV^{-1}-\lambda I_6)=0$ which gives the effective reproduction numberdenoted andgivenby R_e, where

$$R_e=\sqrt{\frac{\beta_h(1-\beta_h)[(\mu_v+\delta_v)((1-\varphi)\rho)+(\mu_v+\delta_v+\alpha)(1-(1-\varphi)\rho)]}{(\gamma+\mu_h+\delta_h)(\mu_v+\delta_v+\alpha)(\mu_v+\delta_v)}} \quad (17)$$

The effective reproduction number, R_e is defined as the number of secondary infections derived from a single primary infection in a population of susceptible [26, 27]. Its biological meaning is readily interpreted from sum of the terms denoted by R_{es} and R_{er} in which

$$R_{vs}=\sqrt{\frac{\beta_h(1-\beta_h)\beta_v((1-\varphi)\rho)}{(\gamma+\mu_h+\delta_h)(\mu_v+\delta_v+\alpha)}} \text{ and } R_{vr}=\sqrt{\frac{\beta_h(1-\beta_h)\beta_v[(1-(1-\varphi)\rho)]}{(\gamma+\mu_h+\delta_h)(\mu_v+\delta_v)}} \quad (18)$$

If there is no any control strategy i.e., $\alpha=0$, then the effective reproduction number R_e for the model equations 2a-freduces to the basic reproduction number denoted and is given by

$$R_o=\sqrt{\frac{\beta_h(1-\beta_h)\beta_v[((1-\varphi)\rho)+(1-(1-\varphi)\rho)]}{(\gamma+\mu_h+\delta_h)(\mu_v+\delta_v)}} \quad (19)$$

2.3.5. Local Stability of the Disease-Free Equilibrium Point

Here, the stability analysis of the disease-free Equilibrium point $E_0=\{S_h^0,\ I_h^0,\ R_h^0,\ S_v^0\ I_{vs}^0\ I_{vr}^0\}$ of model equations (1) to (6) computing its Jacobian matrix. The Jacobian matrix is computed by differentiating the left-hand side function of each equation in the system with respect to the state variables $S_h,\ I_h,\ R_h,\ S_v\ I_{vs}\ I_{vr}$. That is, the followingsystem of modelequations will be considered to construct Jacobian matrix and to conduct further analysis.

The stability analysis and the results are stated and proved in Theorem 3.

$$dS_h/dt=\Lambda_h+\theta R_h-[\beta_h(1-\beta_h)(I_{vs}+I_{vs})S_h]/N_h-\mu_h S_h$$

$$dI_h/dt=[\beta_h(1-\beta_h)(I_{vs}+I_{vs})S_h]/N_h-(\gamma+\mu_h+\delta_h)I_h$$

$$dR_h/dt=\gamma I_h-(\mu_h+\theta)R_h$$

$$dS_v/dt=\Lambda_v-[\beta_v S_v I_h]/N_v-(\alpha+\mu_v)S_v$$

$$dI_{vs}/dt=[[\rho(1-\varphi)]\beta_v S_v I_h]/N_v-(\alpha+\mu_v+\delta_v)I_{vs}$$

$$dI_{vr}/dt=\left[[1-(1-\varphi)\rho]\beta_v S_v I_h\right]/N_v-(\mu_v+\delta_v)I_{vr}$$

Theorem 3: The Disease-free Equilibrium point E_0 is locally asymptotically stableif $R_e<1$ but unstable if $R_e>1$.

Proof:

The Jacobian matrix of the system of equations (1) to (6) evaluated at the disease-free equilibrium point $E_0$is given by:

$$J(E_0)=\begin{bmatrix}-\mu_h & 0 & \theta & 0 & 0 & 0\\ 0 & -(\gamma+\mu_h+\delta_h) & 0 & 0 & 0 & 0\\ 0 & \gamma & -(\theta+\mu_h) & 0 & 0 & 0\\ 0 & \beta_v & 0 & -(\mu_v+\alpha) & 0 & 0\\ 0 & [(1-\varphi)\rho]\beta_v & 0 & 0 & -(\mu_v+\delta_v+\alpha) & 0\\ 0 & [1-(1-\varphi)\rho]\beta_v & 0 & 0 & 0 & -(\mu_v+\delta_v)\end{bmatrix}$$

In order to prove the statement, it is required to show that all the eigenvalues of $J(E_0)$are negative. Since the

first and fourth, fifth, and sixth columns contain only diagonal terms they givefour negative eigenvalues $\lambda_1 = -\mu_h$, $\lambda_2 = -(\mu_v + \alpha)$, $\lambda_3 = -(\mu_v + \delta_v + \alpha), \lambda_4 = -(\mu_v + \delta_v)$. The other two eigenvalues can be computed from the sub-matrix$J_1(E_0)$ formed by excluding the first and the third rows and columns of $J(E_0)$. Hence $J_1(E_0)$ is given by

$$J_1(E_0) = \begin{bmatrix} -(\gamma + \mu_h + \delta_h) & 0 \\ \gamma & -(\theta + \mu_h) \end{bmatrix}$$

Now, the characteristic equation $\det[J_1(E_0) - \lambda \mathrm{I}] = 0$ takes the form as:

$$\begin{vmatrix} -(\mu_h + \delta_h + \gamma) - \lambda & 0 \\ \gamma & -(\theta + \mu_h) - \lambda \end{vmatrix} = 0$$

Also, the characteristic equation can be expressed in a quadratic form as

$$\lambda^2 + (2\mu_h + \delta_h + \gamma + \theta)\lambda + (\mu_h + \delta_h + \gamma)(\theta + \mu_h) = 0$$

Upon solving the quadratic equation, the 5rdand 6th eigenvalues λ_5 and $\lambda_6$are obtained as

$$\lambda_5 = -\left\{(2\mu_h + \delta_h + \gamma + \theta) + \sqrt{[(2\mu_h + \delta_h + \gamma + \theta)^2 - 4(\mu_h + \delta_h + \gamma)(\theta + \mu_h)]}\right\}/2$$

$$\lambda_6 = -\left\{(2\mu_h + \delta_h + \gamma + \theta) - \sqrt{[(2\mu_h + \delta_h + \gamma + \theta)^2 - 4(\mu_h + \delta_h + \gamma)(\theta + \mu_h)]}\right\}/2$$

Here, it can be observed that the eigenvalue λ_5 is absolutely a negative. However, the eigenvalue λ_6 is a negative if the condition
$\sqrt{[(2\mu_h + \delta_h + \gamma + \theta)^2 - 4(\mu_h + \delta_h + \gamma)(\theta + \mu_h)]}/2 < 0$ is valid.

Thus, all the eigenvalues of the Jacobian matrix at the disease-free equilibrium $J(E_0)$ arenegative provided that $[(2\mu_h + \delta_h + \gamma + \theta)^2 - 4(\mu_h + \delta_h + \gamma)(\theta + \mu_h)] < 0$ if and only if$\frac{T^2}{R_e^2}\left(R_e^2 - \frac{MK}{NT^2}\right) < 0$ for which

$MK > NT^2$ or equivalently $R_e < 1$ is valid, Where $K = (\theta + \mu_h), N = (\mu_v + \delta_v + \alpha)(\mu_v + \delta_v)$

$$R_e = \sqrt{\frac{\beta_h(1-\beta_h)\beta_v[(\mu_v+\delta_v)((1-\varphi)\rho)+(\mu_v+\delta_v+\alpha)(1-(1-\varphi)\rho)]}{(\gamma+\mu_h+\delta_h)(\mu_v+\delta_v+\alpha)(\mu_v+\delta_v)}}, T = (2\mu_h + \delta_h + \gamma + \theta)$$

$$M = 4\beta_h(1-\beta_h)\left[(\mu_v + \delta_v)\big((1-\varphi)\rho\big) + (\mu_v + \delta_v + \alpha)(1-(1-\varphi)\rho)\right]$$

$$(\gamma + \mu_h + \delta_h) = \frac{\beta_h(1-\beta_h)\beta_v\left[(\mu_v + \delta_v)\big((1-\varphi)\rho\big) + (\mu_v + \delta_v + \alpha)(1-(1-\varphi)\rho)\right]}{R_e^2(\mu_v + \delta_v + \alpha)(\mu_v + \delta_v)}$$

Therefore, the disease-free equilibrium point is locally asymptotically stable if $R_e < 1$ and unstable if $R_e > 1$.

Theorem 4. If $R_e < 1$, then the disease free equilibrium point E_0 is globally asymptotically stable and unstable. if $R_e > 1$.

Proof: Consider the following Lyapunov function to show the global stability of E_0.

$$L[I_h, (I_{vs} + I_{vr})] = [(\mu_v + \delta_v)I_h/\beta_h(1-\beta_h)] - (I_{vs} + I_{vr})$$

$$dL/dt = -[(\mu_v + \delta_v)I_h/\beta_h(1-\beta_h)](\gamma + \mu_h + \delta_h)I_h - \beta_V I_h + 2(\mu_v + \delta_v)(I_{vs} + I_{vr}) + \alpha I_{vs}$$

$$dL/dt = -\left[[(\mu_v + \delta_v)I_h/\beta_h(1-\beta_h)](\gamma + \mu_h + \delta_h) - \beta_V\right]I_h + 2(\mu_v + \delta_v)(I_{vs} + I_{vr}) + \alpha I_{vs}$$

$$dL/dt \le -\left[[(\mu_v + \delta_v)I_h/\beta_h(1-\beta_h)](\gamma + \mu_h + \delta_h) - \beta_V\right]I_h \le 0$$. Thus, $dL/dt \le 0$. This is equivalent to $R_e < 1$ from LaSalle's invariant principle [28]. Therefore, the disease-free equilibrium $E_0$is globally asymptotically stable in Ω if $R_e < 1$.

2.3.6. Local Stability of the Endemic Equilibrium Solution

A disease is endemic in a given population if it continues to persistinthat population. The stability of endemic equilibrium of the model is studied in the following theorem.

Theorem5: The endemic equilibrium solution E^* of the model equations (1) to (6) is locally asymptotically stable if $R_e > 1$ and unstable if $R_e < 1$.

Proof:

let $E^* = \{S_h^*, I_h^*, R_{h,}^* S_v^*, I_{vs}^*, I_{vr}^*\}$ and $det(J(E^*) - \lambda I_6) = \begin{vmatrix} J_0 & J_1 \\ J_2 & J_3 \end{vmatrix} = 0$ where

$$J_0 = \begin{vmatrix} -\frac{\beta_h(1-\beta_h)\mu_h(I_{vs}^* + I_{vr}^*)}{\Lambda_h} - \mu_h - \lambda & 0 & \theta \\ \frac{\beta_h(1-\beta_h)\mu_h(I_{vs}^* + I_{vr}^*)}{\Lambda_h} & -(\gamma + \mu_h + \delta_h) - \lambda & 0 \\ 0 & \gamma & -(\theta + \mu_h) - \lambda \end{vmatrix} = 0$$

$$J_1 = \begin{vmatrix} 0 & -\dfrac{\beta_h(1-\beta_h)\mu_h S_h^* I_{vr}^*}{\Lambda_h} & -\dfrac{\beta_h(1-\beta_h)\mu_h S_h^* I_{vs}^*}{\Lambda_h} \\ 0 & \dfrac{\beta_h(1-\beta_h)\mu_h S_h^* I_{vr}^*}{\Lambda_h} & \dfrac{\beta_h(1-\beta_h)\mu_h S_h^* I_{vs}^*}{\Lambda_h} \\ 0 & 0 & 0 \end{vmatrix} = 0$$

$$J_2 = \begin{vmatrix} 0 & -\dfrac{\mu_v\beta_v S_v^*}{\Lambda_v} & 0 \\ 0 & \dfrac{[(1-\varphi)\rho]\mu_v\beta_v S_v^*}{\Lambda_v} & 0 \\ 0 & \dfrac{[1-(1-\varphi)\rho]\mu_v\beta_v S_v^*}{\Lambda_v} & 0 \end{vmatrix} = 0$$

$$J_3 = \begin{vmatrix} -\dfrac{[\beta_v I_h^* + (\mu_v+\alpha)\mu_v]}{\Lambda_v} - \lambda & 0 & 0 \\ \dfrac{[(1-\varphi)\rho]\mu_v\beta_v I_h^*}{\Lambda_v} & -(\mu_v+\delta_v+\alpha) - \lambda & 0 \\ \dfrac{[1-(1-\varphi)\rho]\mu_v\beta_v I_h^*}{\Lambda_v} & 0 & -(\mu_v+\delta_v) - \lambda \end{vmatrix} = 0$$

Here,

$\lambda_1 = -\frac{\beta_h(1-\beta_h)\mu_h(I_{vs}^*+I_{vr}^*)}{\Lambda_h} - \mu_h = -\frac{(\gamma+\mu_h+\delta_h)\Lambda_v I_h^* R_e^2}{\beta_v I_h^*+(\mu_v+\alpha)} - \mu_h$

$\lambda_2 = -(\gamma + \mu_h + \delta_h), \lambda_3 = -(\theta + \mu_h)$ and $\lambda_4, \lambda_5,$ and λ_6 Can be estimatedorcomputed from the characteristicequation; that is,

$$\lambda^3 + d_1\lambda^2 + d_2\lambda + d_3 \qquad (20)$$

By Using the Routh-Hurwitz criterion, it can be seen that all the eigenvalues of the characteristic equation (20) have negative real part if and only if$d_1 > 0$, $d_3 > 0$ and $d_1 d_2 > d_3$[29].

Where $d_1 = \left(\frac{(\gamma+\mu_h+\delta_h)\Lambda_v I_h^* R_e^2}{\beta_v I_h^*+(\mu_v+\alpha)} + \mu_h\right) + (\gamma + \mu_h + \delta_h) + (\theta + \mu_h)$

$$d_2 = \left(\frac{(\gamma + \mu_h + \delta_h)\Lambda_v I_h^* R_e^2}{\beta_v I_h^* + (\mu_v+\alpha)} + \mu_h\right)(2\mu_h + \gamma + \delta_h + \theta) + (\theta + \mu_h)(\gamma + \mu_h + \delta_h)$$

$$d_3 = \left(\frac{(\gamma+\mu_h+\delta_h)\Lambda_v I_h^* R_e^2}{\beta_v I_h^*+(\mu_v+\alpha)} + \mu_h\right)(\theta + \mu_h)(\gamma + \mu_h + \delta_h) + \frac{(\gamma+\mu_h+\delta_h)\Lambda_v I_h^* R_e^2}{\beta_v I_h^*+(\mu_v+\alpha)}\theta\gamma$$

Clearly d_1 and d_3 are positive because both of them are a sum of positive variables but $d_1 d_2 > d_3$ if $\frac{(\gamma+\mu_h+\delta_h)\Lambda_v I_h^* R_e^2}{\beta_v I_h^*+(\mu_v+\alpha)}\theta\gamma < 0$ equivalently $R_e - 1$ must be positive which leads to $R_e > 1$. Thus, the endemic equilibrium will be locally asymptotically stable if and only if $R_e > 1$and unstable if $R_e < 1$.

3. Simulations and Discussions

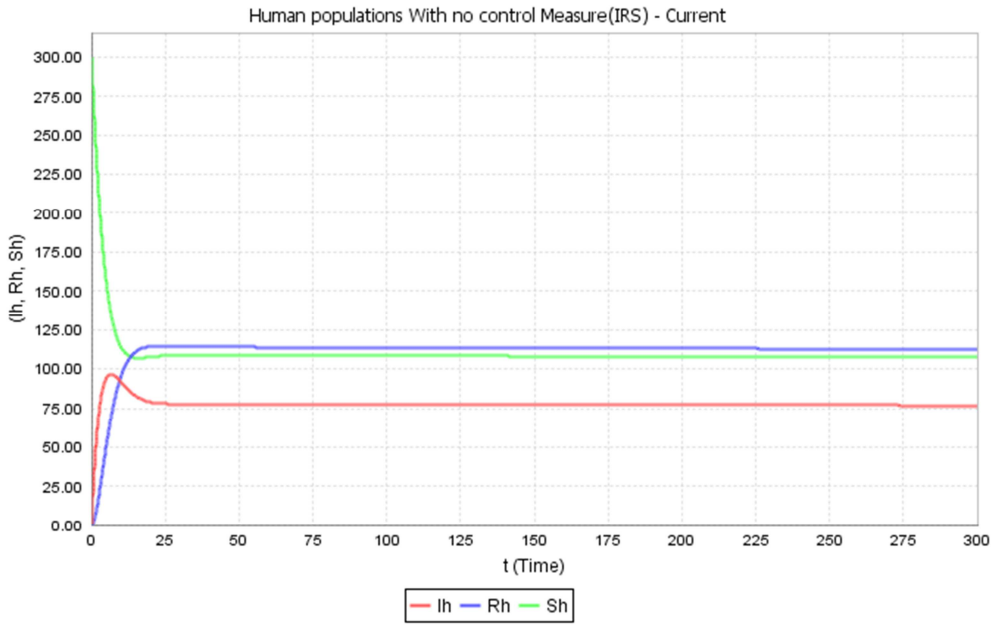
In the present study an SIR-SImodel has been formulated and mathematically analyzed. The main objective of this study is to understand qualitatively the factors that has more impact on the efficacy of the incorporated control strategy for the transmission and spread of endemic malaria disease and its effectiveness on insecticide resistant and sensitive malaria vector strains. Here, simulation study is conducted in support of mathematical analysis. Numerical simulation of model system equations (1) to (6) is carried out using DE Discover Solver. The initial population sizes and a set of parameter values are chosen based on similar studies of [30, 31, 32]. Graphical representations showing the human and mosquito populations with and without control measure (IRS)are provided in Figures (2) – (3). And variations in reproduction numbers with respect to contact rate between the infected humans and the infected mosquitoes are provided in Figures (4) –(6). Since values of someparametersare not available in the real world, data from literature is used for some parameters and for others estimated values are assigned. Table 3 and 4show the values assigned tostate variables and parametersrespectively and these values have been used in conducting simulations study.

Table 3. *Estimated values of state variables.*

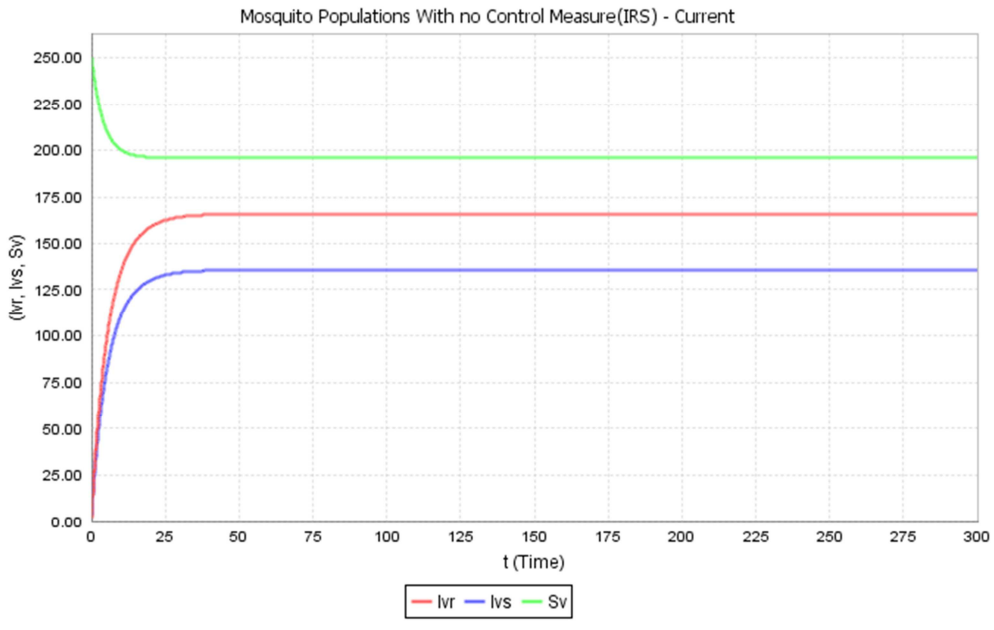
State variable	Initial value	Source
S_h	300	[30, 31, 32]
I_h	1	[30, 31,32]
R_h	0	[30, 31, 32]
S_v	250	[30, 31, 32]
I_{vs}	4	Estimated
I_{vr}	2	Estimated

Table 4. *Estimated values of parameters.*

Parameter	Value	Source
Λ_h	0.0280	[30]
μ_h	0.0000391	[30]
δ_h	0.00040	[30]
β_h	0.4500	Estimated
Λ_v	50.000	Estimated
μ_v	0.04000	[30]
δ_v	0.1000	[30]
β_v	0.2150	Estimated
θ	0.0140	Estimated
γ	0.2050	[30]
α	0.5000	Estimated
φ	0.2000	Estimated
ρ	0.3000	Estimated



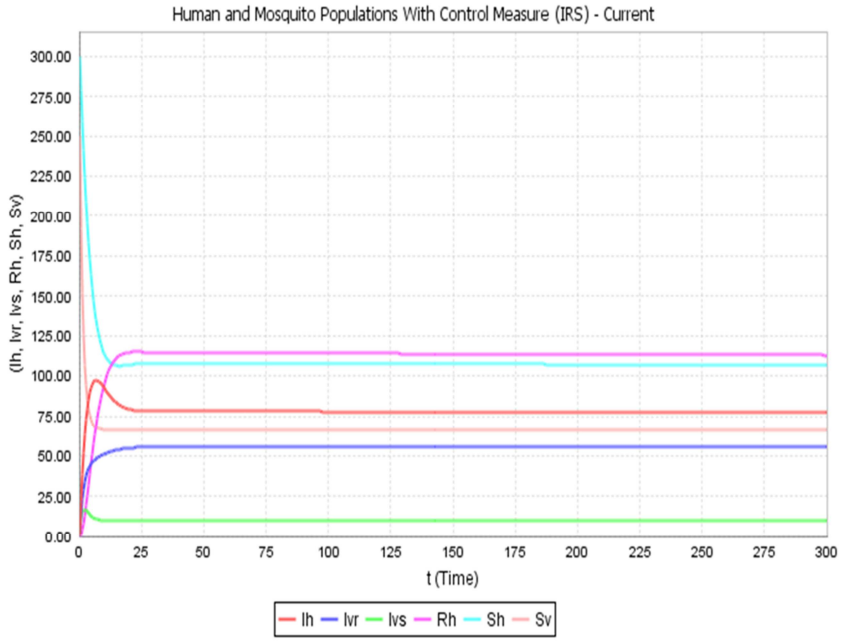
(a)



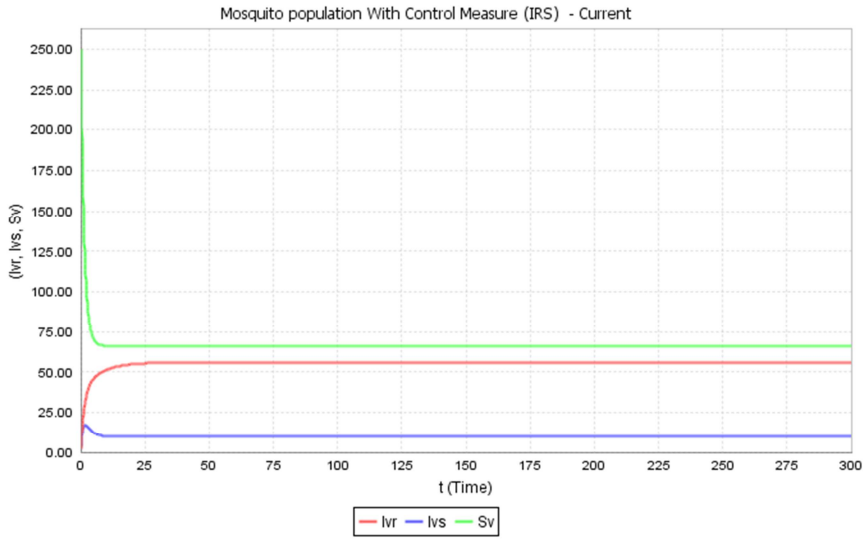
(b)

Figure 2. *Human and Mosquito populations with no control measure (IRS).*

Figure 2 presents typical solution plots for the human populations and the mosquito populations. As the individuals leave one class for another, the curves change accordingly. Once the curves level off, we stop the simulation as the populations reached equilibrium. With no intervention strategy that is, when IRS =0, the final infected human populations is non-zero; however, infected human populations smaller than the susceptible and recovered classes of human populations. Since the populations are non-zero, malaria has not been eradicated at the equilibrium state. For the mosquito classes, notice that the equilibrium state contains nearly almost an equal number of infected insecticide (IRS) resistant mosquito strains as susceptible mosquitoes while the infected insecticide sensitive mosquito strains are less in number than the insecticide(IRS) resistant mosquito strains. It is important to note that, here, the built in DE Discover solver will find a stable solution, which may have a dependence on initial conditions, if the system has more than one steady state.



(a)



(b)

Figure 3. *Human and Mosquito, and mosquito populations with control measure (IRS).*

Figure 3 showthat the human and mosquito populations (top) and the mosquito populations separately (bottom) as a function of time. Thesefiguresare created using initial conditions from the Table 3, using the parameter values in Table 4. When IRS=0.5. The equations were solved in DE Discover solvers. Unlike Figure 2, In each mosquito

populations classes therehas been seen a reduction of their numbers relatively. This is due to the fact that removal of mosquitoes from the different classes associated with IRS. Since the human populations are non-zero, malaria has not been eradicated at the equilibrium state. This phenomenon is due to the fact that the mosquito population becomes so small that the birth rate of humans greater than the death rate.

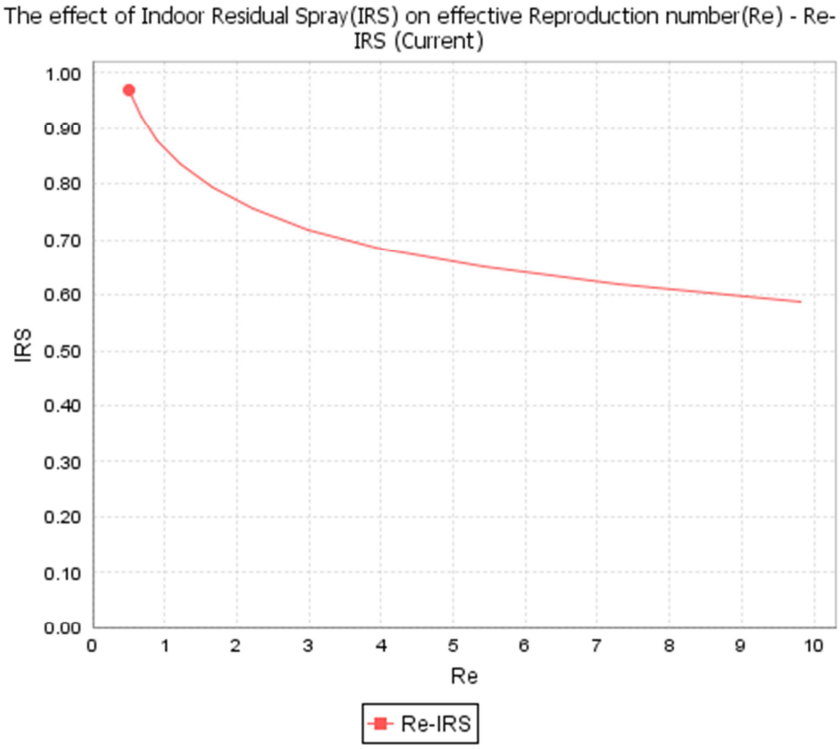


Figure 4. *Indoor Residual SprayIRS VS Effective Reproduction number $R_e$graph.*

These plots show the effect of IRS on effective reproduction number, $R_e$as a function of IRSand where IRS= 0.97. Notice that the reproductive number drops below one for IRSbetween 0.85 and 1.

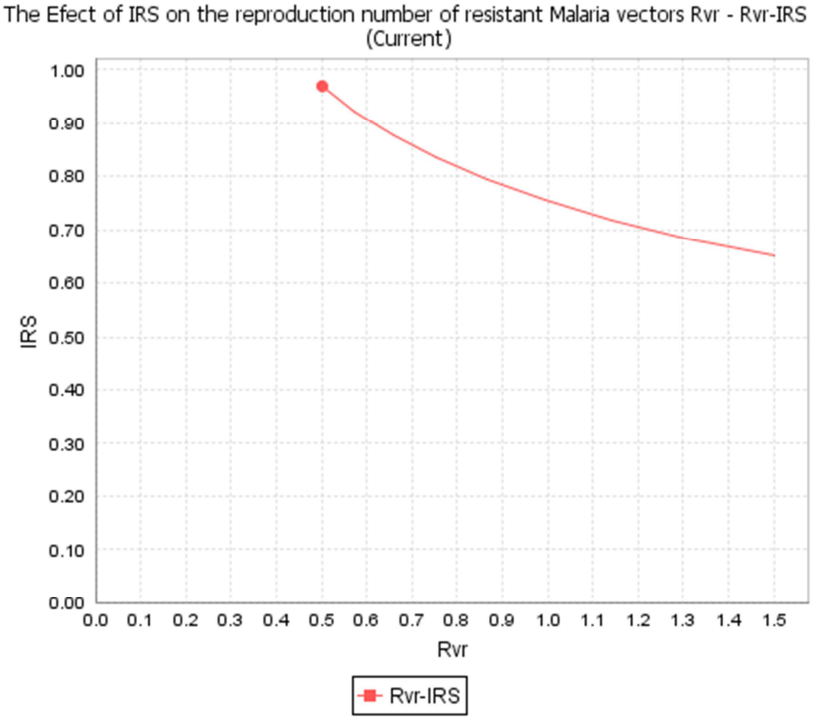


Figure 5. *Indoor Residual SprayIRS VS Reproduction number for the Resistant malaria vector strainsR_{vr}graph.*

Figure 5 shows that the effect of IRSonthe reproduction numberofinsecticidesresistant malaria vector strains, R_{vr} as a function of IRSandwhere IRS= 0.97. Notice that the reproductive number drops below one for IRSbetween 0.75 and 1.

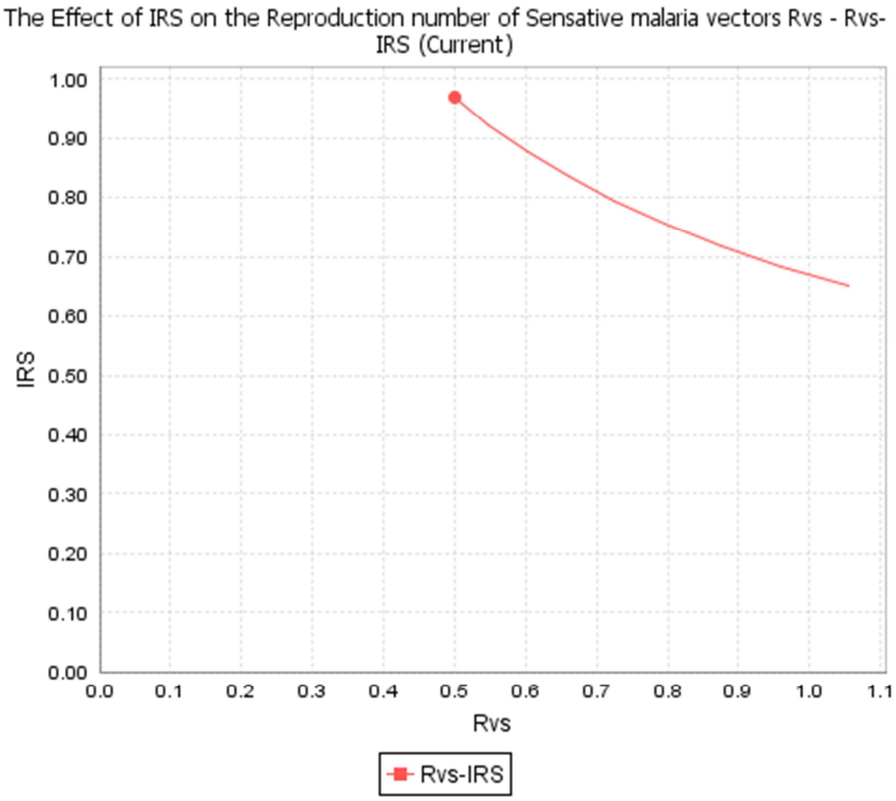


Figure 6. *Indoor Residual SprayIRS VS Reproduction number for the sensitive malaria vector*R_{vs}*graph.*

Figure 6 shows that the effect of IRS on the reproductive number of insecticide sensitive malaria vector strains, R_{vs}as a function of IRSand where IRS= 0.97. Notice that the reproductive number drops below one for IRS between 0.65 and 1.

4. Conclusion and Recommendation

In our analysis of the modeling the dynamics of endemic malaria transmission with the effects of control measure (IRS), we ran a number of simulations using the initial population sizes and a set of parameter values in Table 3 and Table 4 respectively in this model. Where IRS, is ranging from 0 to 1. We have seen that as the use IRS increases in amount, the reproductive number gets closer to 1, eventually falling below that critical value. As the evolution of insecticide resistance that allows for small proportion of mosquitoes possessing resistance genes allowing them to resist and survive the effects of the insecticide (IRS) increases, the change in reproduction number of resistant malaria vector strains R_{vr} against insecticide is clearly seen. This consequently resulted in increasing the overall effective reproduction number R_e . Because of only single insecticide (IRS) is used during intervention and (IRS) is not properly used at a time i.e., the same control measure is used for long periods without followed by rotation of different types of control measures, insecticide sensitive malaria vector strains progress to the insecticide resistant malaria vector strains. This also leads to an increase in reproduction number of insecticide resistant malaria vector strains R_{vr}, consequently increasing the overall effective reproduction number R_e which is resulted in control intervention failure which also resulted in malaria disease burden in the community.

The public health implications of the results include: (i) every effort should be taken to minimize the evolution of insecticide resistance due to malaria control interventions failure and (ii)) at least the combination of two different types of control measures and followed by rotation of intervention strategies could be more realistic to minimize the number of resistant malaria vector strains and essential in reducing the malaria disease burden in the community.

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