The Impact of Susceptible Human Immigrants on the Spread and Dynamics of Malaria Transmission

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Abstract: Malaria is one of infectious diseases and has become the most public health issue especially in developing countries. Mathematically, the spread of malaria can be modeled to predict the dynamics of the outbreak of the disease. The present research studies the impact of migration of susceptible population on the dynamics of malaria transmission. In this paper an improved mathematical model is constructed based on a set of reasonable assumptions. Validity of the model is proved by verifying positivity of the solution. Mathematical analysis is carried out including equilibrium point analysis. Basic reproduction number of the model is determined so as to study the effect of migration parameter on the malaria outbreak. It has been observed that the migration parameter is directly proportional to the malaria outbreak. Hence, it is suggested that in order to keep the malaria outbreak under control, the migration parameter is required to be minimized. That is, migration of populations is recommended to reduce so as to reduce the impact of malaria outbreak.

Keywords: Malaria Outbreak, Reproduction Number, Migration Parameter, Numerical Simulation

1. Introduction

Malaria is an infectious disease caused by the Plasmodium parasite and transmitted among humans through bites of female Anopheles mosquitoes. It remains one of the most prevalent and lethal human infections throughout the world. An estimated 40% of the world's population lives in malaria endemic areas. Most malaria cases and deaths occur in sub-Saharan Africa [1].

It causes an estimated 300 to 500 million cases and 1.5 to 2.7 million deaths each year worldwide. Africa shares 80% of the cases and 90% of deaths [2]. The environmental conditions in the tropics are the prime factor for malaria being endemic. The moderate-to-warm temperatures, high humidity and water bodies allow mosquitoes and parasites to reproduce. The epidemiological patterns of malaria usually vary with season because of its dependence on transmission from mosquitoes. The infection can lead to serious complications affecting the brain, lungs, kidneys and other organs of human body. Clinical symptoms such as fever, body pains, chills and sweats may develop a few days after

infected mosquito bites [3]. Malaria has also gained prominence in recent times since climate change or global warming is predicted. The incidence of malaria increases as the warming increases. Both the increases and fluctuations in temperature affect favorably the vector and parasite life cycles. This can cause reduced prevalence of the disease in cold areas, while it may increase in warm and hot areas. Thus, climate change can affect the malaria prevalence pattern by migration from lower latitudes to regions where the human population has not developed immunity to the disease.

Controlling of malaria is a challenging task due to many factors. The complexity of the disease control process, cost of the control program and resistance of the parasite to anti-malarial drugs, and vectors to insecticides, are some of the challenges.

There is a variation in disease patterns and transmission dynamics from place to place, by season and according to varying environmental circumstances. The approaches in the

planning and implementation of prevention and control activities also vary based on local realities.

Malaria cases are exacerbated by the high levels of HIV infection that weaken the immune system rendering people with HIV more susceptible to the disease [4]. It enhances mortality in advanced HIV patients by a factor of about 25% in non-stable malaria areas [5]. Since malaria increases morbidity and mortality, it continues to inflict major public health and socio-economic burdens in developing countries.

It is clear that poverty, while not a disease in itself, is a contributing factor not only for malaria but also for almost all diseases that face mankind. Because of poverty, communities may have poor sanitation and poor drainage, and these two factors allow the mosquitoes to breed in ever greater numbers. Poverty also means that people will not be able to afford the simple protection of a mosquito net or even screens for their windows.

A favorite hiding place for the *Anopheles* is in a dark moist room. With an increased number of vectors living with a human results in an increased chance of being bitten by an infected mosquito, which will in turn infect the human with the parasite [6].

Malaria has for many years been considered as a global issue. Many epidemiologists and other scientists have been investing their efforts in understanding the dynamics of malaria and to design the control strategies of its transmission. To complement the efforts of those scientists mathematicians have developed a significant and effective tool namely modeling technique of malaria. The models give insights into many aspects including the interaction between the human and mosquito populations, the dynamics of malaria, and also the controlling and eradication strategies of the transmission.

Mathematical modeling of malaria has been flourishing since the days of Ronald Ross who was awarded a Nobel Prize for his work in 1911. He developed a simple SIS model comprising of Susceptible, Infected, and Susceptible populations. It was based on the assumption that at any time the total population can be divided into two distinct human compartments. It was shown that malaria can be eliminated if the reproduction number is brought below a certain value. This value of reproduction number naturally depends on biological factors such as the biting rate and vectorial capacity [7].

For estimating infection and recovery rates a model is used assuming that population exposed to infection remains unchanged over time. This model has proved that reducing the number of mosquitoes is an inefficient control strategy and that had a little effect on the dynamics of malaria in areas of strong transmission [8].

The Ross-Macdonald mathematical model involves an interaction between infected human hosts and infected mosquito vectors. Bailey and Aron models take into account that acquired immunity to malaria depends on exposure i.e. the immunity is boosted by additional infections [9-11].

Tumwiine *et al.* used SIS and SI models in the human

hosts and mosquito vectors respectively for the study of malaria epidemics that last for a short period in which birth and immunity to the disease were ignored. The system was in equilibrium only at the point of extinction that was neither stable nor unstable. However, some important results were revealed numerically [12].

Some recent papers have also included the effects of environment and that of the resistance to drugs [13-17]. Recently, some other researchers proposed ODE compartmental models to describe the spread of malaria [18, 19]. Compartment model for the spread of malaria with susceptible-infected-recovered-susceptible (SIRS) pattern for human and susceptible-infected (SI) pattern for mosquito populations have been developed [3, 20].

Yang, Wei, and Li proposed a model dividing respectively the human and vector populations into SIR and SI compartments [21].

The reproduction number R_0 is defined and shown that the disease-free equilibrium as well as endemic equilibrium exists and is stable [3, 20, 21].

The immigration of human population is not taken in to consideration in the model [20]. Hence, this need not be a reasonable model due to the following reasons: the immigration of human population has a potential impact on the dynamics of malaria, controlling strategies and eventually eradication.

In the present study the existing model is improved by including migration of susceptible human population. The main objective of this study is to understand the impact of susceptible immigrations on the transmission dynamics and the spread of malaria disease.

2. Model Assumptions

- i. Immigration in to the human susceptible compartment alone is considered.
- ii. The number of infected people increases at a rate proportional to the number of possible contacts of infectious mosquito and susceptible human populations.
- iii. Humans move from susceptible to infectious compartment with a progression rate of β_h .
- iv. The transfer rate of humans from infectious to recovered compartment is proportional to the number of infectious humans. Humans move from infectious to recovered compartments with a progression rate of γ_h .
- v. A human can die at any stage due to natural causes. Therefore α_h is taken as natural death rate in every human compartment.
- vi. The recovered human individuals do not enter into the susceptible class again,
- vii. Mosquitoes do not recover from malaria.

3. Formulation of the Model

The model of malaria transmission considered in this study is *SIR* for human population and *SI* for mosquito

population. The model is formulated for the spread of malaria in both the human and mosquito populations with the total respective population sizes at time t are denoted by $N_h(t)$ and $N_v(t)$. Human population is compartmentalized into epidemiological classes as susceptible $S_h(t)$, infected $I_h(t)$, and recovered $R_h(t)$ compartments. Similarly, mosquito population is compartmentalized as susceptible $S_v(t)$ and infected $I_v(t)$ vector populations. The vector component of the model does not include an immune class as mosquitoes never recover from the infection, that is, their infective period ends with their death due to their relatively short lifecycle. Thus, the immune class in the mosquito population is negligible and hence omitted. Also, the natural death occurs with equal rates in all mosquito compartments.

The model can be used for diseases that persist in a population for a long period of time with vital dynamics. The basic model incorporates a set of assumptions. Both the human and vector total population sizes are assumed to be constant. The recovered individuals in human population develop permanent immunity. The populations in compartments of both humans and vectors are non-negative, and so is the case with all the parameters involved in the model as it is evident from Table 1.

Table 1. Notations and descriptions of state variables and parameters.

S_h :	Number of susceptible humans at time t .
I_h :	Number of infected humans at time t .
R_h :	Number of recovered humans at time t .
S_v :	Number of susceptible mosquitoes at time t .
I_v :	Number of infected mosquitoes at time t .
N_h :	The total human population at time t .
N_v :	The total mosquito population at time t .
μ_h :	Birth rate of human population.
α_h :	Natural death rate of humans.
ρ_h :	Disease-induced death rate of humans.
β :	Immigration rate for humans.
β_h :	The contact rate of infective vector with susceptible humans.
γ_h :	Recovery rate of infective humans.
μ_v :	Birth rate of mosquitoes.
α_v :	Natural death rate of mosquitoes.
β_v :	The contact rate of susceptible mosquitoes with infective humans.

All newborns are susceptible to infection. The development of malaria starts when the infectious female mosquito bites the susceptible human. The vectors do not die from the infection or otherwise are harmed.

Individuals move from one class to the other as their status with respect to the disease.

Humans enter the susceptible class through birth rate μ_h and immigration rate β and leave through death rate α_h , and infective rate $\beta_h S_h$.

All human individuals, whatever may be the status, are subject to a natural death which occurs at a rate α_h , and disease induced death rate ρ_h .

Figure 1. The flow diagram of malaria compartmental model.

With the inclusion of the model assumptions, notations of variables and parameters, the system of ordinary differential equations describing the dynamics of malaria in both the human and mosquito populations can be formulated as

$$\frac{dS_h}{dt} = (\beta + \mu_h)N_h - \beta_h S_h I_v - \alpha_h S_h \quad (1)$$

$$\frac{dI_h}{dt} = \beta_h S_h I_v - \rho_h I_h - \gamma_h I_h - \alpha_h I_h \quad (2)$$

$$\frac{dR_h}{dt} = \gamma_h I_h - \alpha_h R_h \quad (3)$$

$$\frac{dS_v}{dt} = \mu_v N_v - \beta_v S_v I_h - \alpha_v S_v \quad (4)$$

$$\frac{dI_v}{dt} = \beta_v S_v I_h - \alpha_v I_v \quad (5)$$

with initial conditions

$$S_h(0) = S_{h0}, I_h(0) = I_{h0}, R_h(0) = R_{h0}, \quad (6)$$

and

$$S_v(0) = S_{v0}, I_v(0) = I_{v0} \quad (7)$$

The total population sizes N_h and N_v can be determined by

$$S_h + I_h + R_h = N_h \quad (8)$$

$$S_v + I_v = N_v \quad (9)$$

In this model, (i) The terms $\mu_h N_h$ and $\mu_v N_v$ denote the birthrates of human and mosquito populations respectively; (ii) βN_h is the rate of immigration of susceptible humans (iii) The terms $\alpha_h S_h$, $\alpha_h I_h$ and $\alpha_h R_h$ refer to the removal rates of susceptible, infected and recovered humans due to natural deaths. (iv) the terms $\alpha_v S_v$ and $\alpha_v I_v$ are the rates of natural deaths of susceptible and infected mosquito populations (v) the term $\rho_h I_h$ is the number of removed humans because of the disease (vi) $\gamma_h I_h$ is the rate of recovered human population (vii) the term $\beta_h S_h I_v$ denotes the rate at which the susceptible human hosts S_h get infected by the infective mosquito vector I_v and (viii) $\beta_v S_v I_h$ refers to the rate at which the susceptible mosquitoes S_v are infected by the infective human hosts I_h .

However, both the terms $\beta_h S_h I_v$ and $\beta_v S_v I_h$ are important parts of the model since they describe the interactions between the human and mosquito populations.

4. Mathematical Analysis of the Model

The mathematical analysis of the model described by the system (1) to (5) is presented here.

The rate of change of total human population size $N_h(t) = S_h(t) + I_h(t) + R_h(t)$ can be obtained using equations (1) to (3) as

$$\begin{aligned} \frac{d}{dt} N_h(t) &= \frac{d}{dt} S_h(t) + \frac{d}{dt} I_h(t) + \frac{d}{dt} R_h(t) \\ &= (\beta + \mu_h - \alpha_h) N_h - \rho_h I_h \end{aligned}$$

Similarly, the rate of change of total mosquito population size $N_v(t) = S_v(t) + I_v(t)$ can be obtained using equations (4) to (5) as

$$\frac{d}{dt} N_v(t) = \frac{d}{dt} S_v(t) + \frac{d}{dt} I_v(t) = \mu_v N_v - \alpha_v (S_v + I_v)$$

4.1. Positivity of Solutions

In order that the model equations (1) to (5) are epidemiologically meaningful and well posed it is appropriate to show that all the state variables are non-

negative. This requirement is stated as a theorem and its proof is provided as follows:

Theorem 1: If $S_h(0) > 0$, $I_h(0) > 0$, $R_h(0), S_v(0) > 0$ and $I_v(0) > 0$, then the solution region $\{S_h(t), I_h(t), R_h(t), S_v(t), I_v(t)\}$ of the system of equations (1) to (5) is always non negative.

Proof: To show the positivity of the solution of the dynamical system comprising the equations (1) to (5), each differential equation is considered and shown that its solution is positive.

Positivity of susceptible human population: Consider the differential equation (1) of the dynamical system $dS_h/dt = (\beta + \mu_h)N_h - \beta_h S_h I_v - \alpha_h S_h$. Since $(\beta + \mu_h)N_h$ is a positive quantity, the equation can be expressed as an inequality as $dS_h/dt \geq -\beta_h S_h I_v - \alpha_h S_h$. Using the technique of separation of variables and up on integration gives $S_h(t) \geq S_{h0} e^{-\int_0^t (\beta_h I_v + \alpha_h) ds}$. But, for any value of the exponent, the exponential term is always a non-negative quantity, that is $e^{-\int_0^t (\beta_h I_v + \alpha_h) ds} \geq 0$. Also it is assumed that $S_{h0} > 0$. Thus, it is clear from the solution that $S_h(t)$ is positive.

Positivity of infective human population: Considering the differential equation (2) of the dynamical system $dI_h/dt = \beta_h S_h I_v - (\rho_h + \gamma_h + \alpha_h)I_h \geq -(\rho_h + \gamma_h + \alpha_h)I_h$. The equation can be expressed as an inequality as $dI_h/dt \geq -(\rho_h + \gamma_h + \alpha_h)I_h$. Using the technique of separation of variables and up on integration gives $I_h(t) \geq I_{h0} e^{-\int_0^t (\rho_h + \gamma_h + \alpha_h) ds} > 0$. But, for any value of the exponent, the exponential term is always a non-negative quantity, that is $e^{-\int_0^t (\rho_h + \gamma_h + \alpha_h) ds} \geq 0$. Also it is assumed that $I_{h0} > 0$. Thus, it is clear from the solution that $I_h(t)$ is positive.

Positivity of infective human population: From the differential equation (3) it can be expressed as $dR_h/dt = \gamma_h I_h - \alpha_h R_h \geq -\alpha_h R_h$. The equation can be expressed as an inequality as $dR_h/dt \geq -\alpha_h R_h$. Using the technique of separation of variables and up on integration gives $R_h(t) \geq R_{h0} e^{-\int_0^t \alpha_h ds} > 0$. But, for any value of the exponent, the exponential term is always a non-negative quantity, that is $e^{-\int_0^t \alpha_h ds} \geq 0$. Also it is assumed that $R_{h0} > 0$. Thus, it is clear from the solution that $R_h(t)$ is positive.

Positivity of susceptible mosquito population: Consider the differential equation (4) of the dynamical system $dS_v/dt = \mu_v N_v - \beta_v S_v I_h - \alpha_v S_v$. Since $\mu_v N_v$ is a positive quantity, the equation can be expressed as an inequality as $dS_v/dt \geq -\beta_v S_v I_h - \alpha_v S_v$. Using the technique of separation of variables and up on integration gives $S_v(t) \geq S_{v0} e^{-\int_0^t (\beta_v I_h + \alpha_v) ds}$. But, for any value of the exponent, the exponential term is always a non-negative quantity, that is $e^{-\int_0^t (\beta_v I_h + \alpha_v) ds} \geq 0$. Also it is assumed that $S_{v0} > 0$. Thus, it is clear from the solution that $S_v(t)$ is positive.

Positivity of infective mosquito population: Consider the differential equation (5) of the dynamical system $dI_v/dt = \beta_v S_v I_h - \alpha_v I_v$. The equation can be expressed as an inequality as $dI_v/dt \geq -\alpha_v I_v$. Using the technique of

separation of variables and up on integration gives $I_v(t) \geq I_{v0} e^{-\int_0^t \alpha_v ds}$. But, for any value of the exponent, the exponential term is always a non-negative quantity, that is $e^{-\int_0^t \alpha_v ds} \geq 0$. Also it is assumed that $I_{v0} > 0$. Thus, it is clear from the solution that $I_v(t)$ is positive.

The five dimensional solution space shows that all the solutions are positive. Hence, the feasible region containing all the solutions of the system of equations (1)-(5) is given by the set $\Omega = \{(S_h, I_h, R_h, S_v, I_v) \in \mathbb{R}^{+5}\}$. Here the quantities S_h, I_h, R_h, S_v, I_v are all non – negatives.

4.2. Scaling of the Model

In order to simplify analysis of the malaria model given by the equations (1)-(5), it is recommended to work with fractional quantities instead of actual quantities. The fractional quantities can be achieved by scaling the model variables and parameters.

Let the scaled proportions for the quantities S_h, I_h, R_h, S_v and I_v be chosen as $S_h = N_h s_h, I_h = N_h i_h, R_h = N_h r_h, S_v = N_v s_v, I_v = N_v i_v$. The original time coordinate $(\mu_h t)$ is transferred to the scaled time coordinate ast . Thus, the scaled version of the equations (1)-(5) then takes the form as

$$\frac{ds_h}{dt} = 1 + \xi - Bs_h i_v - \alpha s_h \quad (10)$$

$$\frac{di_h}{dt} = Bs_h i_v - (\alpha + \gamma) i_h \quad (11)$$

$$\varepsilon \frac{di_v}{dt} = \vartheta(1 - i_v) i_h - \delta i_v \quad (12)$$

Similarly, the scaled initial conditions are $s_h(0) = s_{h0}, i_h(0) = i_{h0}, i_v(0) = i_{v0}$. Here in (10) to (12) the dimensionless parameters represent the following expressions:

$$\varepsilon = (\mu_h / \mu_v), \xi = \frac{\beta}{\mu_h}, B = \frac{\beta_h N_v}{\mu_h}, \alpha = \frac{\alpha_h}{\mu_h}, \gamma = \frac{\rho_h + \gamma_h}{\mu_h}, \vartheta = \frac{\beta_v N_h}{\mu_v}, \delta = \frac{\alpha_v}{\mu_v}$$

Clearly it is true that the scaling of the model reduces the population sizes of both human and mosquito to one unit. That is, $s_h + i_h + r_h = 1$ and $s_v + i_v = 1$. Equivalently, these can be expressed as $r_h = 1 - s_h - i_h$ and $s_v = 1 - i_v$.

4.3. Disease Free Equilibrium

Disease – free equilibrium points are steady state solutions where there is no malaria in the human population or plasmodium parasite in the mosquito population.

The model compartments can be classified in to two categories: infectious and non-infectious categories. The human and mosquito infectious compartments are i_h and i_v . The absence of the disease implies that $i_h = 0$ and $i_v = 0$.

The disease free equilibrium point requires the conditions $\frac{ds_h}{dt} = \frac{di_h}{dt} = \frac{di_v}{dt} = 0$ and $i_h = 0$ and $i_v = 0$. The substitution of these requirements in the non – linear system (10) to (12) leads to the result $1 + \xi - \alpha s_h = 0$ giving $s_h = [(1 + \xi) / \alpha]$.

Thus the disease – free equilibrium point of malaria model (2.10) to (2.12) is given by

$$E_0 = (s_h^0, i_h^0, i_v^0) = \left\{ \frac{1+\xi}{\alpha}, 0, 0 \right\} \quad (13)$$

In (13), E_0 represents the state in which there is no infection in the society i.e., either in human or mosquito population. That is, the malaria disease is absent.

4.4. Basic Reproduction Number

The reproduction number R_0 is defined 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. Here the next generation operator approach as described by Diekmann et al., (1990) is used to find an expression for the basic reproduction number. Reproduction number R_0 is the threshold for many epidemiology models. It determines whether a disease can invade in a population or not. If $R_0 < 1$ each infected individual produces on average less than one new infected individual so the disease is expected to die out. On the other hand if $R_0 > 1$, each individual produces more than one new infected individual so that the disease is expected to spread in the population. This means that the value of the threshold quantity R_0 for eradicating the disease is to be reduced to less than one.

The expression for the reproduction number R_0 is determined here using the next generation approach. The following steps are followed to compute R_0 :

The basic reproduction number cannot be determined from the structure of the mathematical model alone but from the definition of infected and uninfected compartments. Assuming that there are n compartments in the model and of which the first m compartments are of infected individuals [22].

Let the column vector $V_i(x)$ be defined as $V_i(x) = V_i^-(x) - V_i^+(x)$. Here $V_i^+(x)$ is the rate of transfer of individuals into compartment i by all means. Similarly, $V_i^-(x)$ is the rate of transfer of individuals out of compartment i . It is assumed that each function is continuously differentiable at least twice with respect to every dependent variable.

In general, a disease transmission model consists of nonnegative initial conditions together with a system of equations as follows

$$\dot{x}_i = h_i(x) = F_i(x) - V_i(x), i = 1, 2, 3, \dots, n$$

Here $\dot{x}$ denotes the time derivative representing the rate of change of x .

The next step is the computation of the square matrices F and V of order $m \times m$. Here m denotes the number of infected classes. Now, the matrices F and V are defined as $F = \left[\frac{\partial F_i}{\partial x_j}(x_0) \right]$ and $V = \left[\frac{\partial V_i}{\partial x_j}(x_0) \right]$ with $1 \leq i, j \leq m$. Here, F is non-negative; V is non-singular; and x_0 is the disease – free equilibrium point or DFE point.

Since F is non-negative and V non-singular, then V^{-1} is non-negative and also FV^{-1} is nonnegative. Hence the matrix FV^{-1} is called the next generation matrix for the

model.

Finally the basic reproduction number R_0 is given by

$$R_0 = \rho(FV^{-1}) \quad (14)$$

Here in (14), $\rho(A)$ denotes the spectral radius of matrix A and the spectral radius is defined as the biggest non-negative eigenvalue of the matrix.

In case of the present model, F_i and V_i are constructed as

$$F_i = \begin{bmatrix} Bs_h i_v \\ \vartheta i_h - \vartheta i_h i_v \end{bmatrix} \quad (15)$$

$$V_i = \begin{bmatrix} (\alpha + \gamma) i_h \\ \delta i_v \end{bmatrix} \quad (16)$$

The partial derivatives of (15) with respect to (i_h, i_v) and the Jacobian matrix of F_i at the disease – free equilibrium point (13) takes the form as

$$F = \begin{bmatrix} 0 & Bs_h \\ \vartheta - \vartheta i_v & -\vartheta i_h \end{bmatrix} \quad (17)$$

Similarly, the partial derivatives of (16) with respect to (i_h, i_v) and the Jacobian matrix of V_i at the disease – free equilibrium point (13) takes the form as

$$V = \begin{bmatrix} \alpha + \gamma & 0 \\ 0 & \delta \end{bmatrix} \quad (18)$$

The inverse of the matrix V is given by

$$V^{-1} = \begin{bmatrix} \frac{1}{\alpha + \gamma} & 0 \\ 0 & \frac{1}{\delta} \end{bmatrix} \quad (19)$$

Now the next generation matrix FV^{-1} can be constructed as

$$FV^{-1} = \begin{bmatrix} 0 & \frac{Bs_h}{\delta} \\ \frac{\vartheta - \vartheta i_v}{\alpha + \gamma} & \frac{-\vartheta i_h}{\delta} \end{bmatrix} \quad (20)$$

Hence, at the DFE point

$$FV^{-1}(E_0) = \begin{bmatrix} 0 & \frac{B(1+\xi)}{\delta\alpha} \\ \frac{\vartheta}{\alpha + \gamma} & 0 \end{bmatrix} \quad (21)$$

For (21), eigenvalues are to be determined to calculate the basic reproduction number R_0 as the spectral radius of the matrix FV^{-1} . Thus, the eigenvalues are the solutions of the characteristic equation $|FV^{-1}(E_0) - \lambda I| = 0$. That is,

$$\begin{vmatrix} -\lambda & \frac{B(1+\xi)}{\delta\alpha} \\ \frac{\vartheta}{\alpha + \gamma} & -\lambda \end{vmatrix} = 0$$

$$\Rightarrow \lambda^2 = \left(\frac{B\vartheta}{\alpha + \gamma}\right) \left(\frac{1 + \xi}{\delta\alpha}\right)$$

$$\Rightarrow \lambda = \pm \sqrt{\left(\frac{B\vartheta}{\alpha + \gamma}\right) \left(\frac{1 + \xi}{\delta\alpha}\right)}$$

The dominant eigenvalue of the matrix FV^{-1} is

$$\lambda = \sqrt{\left(\frac{B\vartheta}{\alpha + \gamma}\right) \left(\frac{1 + \xi}{\delta\alpha}\right)}$$

Therefore the basic reproduction number is found to have the expression

$$R_0 = \sqrt{\left(\frac{B\vartheta}{\alpha + \gamma}\right) \left(\frac{1 + \xi}{\delta\alpha}\right)} \quad (22)$$

From (22), it can be quantified that higher values of B, ϑ and ξ (the immigration rate) can allow the outbreak of the disease. Conversely, for smaller values the disease dies out.

The reproduction number is a powerful parameter which measures the existence and stability of the disease in the human and mosquito populations. If $B\vartheta(1 + \xi) < \delta\alpha(\alpha + \gamma)$, i.e., $R_0 < 1$, the disease-free equilibrium is the only equilibrium and then the disease dies out. If $B\vartheta(1 + \xi) > \delta\alpha(\alpha + \gamma)$ i.e., $R_0 > 1$ the unique endemic equilibrium exists and the disease persists with the population.

4.5. Stability of the Disease Free Equilibrium Point Analysis

The equilibria are obtained by equating the right hand side of the system (10)-(12) to zero. Disease-free equilibrium (DFE) of the model is the steady-state solution of the model in the absence of the malaria disease. Hence, the DFE of the scaled malaria model (10)-(12) is given by

$$E_0 = (s_h^0, i_h^0, i_v^0) = \left\{ \frac{1 + \xi}{\alpha}, 0, 0 \right\}$$

Theorem 1: The DFE, E_0 of the system (10)-(12) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof: To find the Jacobian matrix let

$$\begin{aligned} f_1 &= 1 + \xi - Bs_h i_v - \alpha s_h, f_2 \\ &= Bs_h i_v - (\alpha + \gamma) i_h, f_3 = [\vartheta(1 - i_v) i_h] / \epsilon \\ &\quad - \delta i_v / \epsilon \end{aligned}$$

Hence, $ds_h/dt = f_1, di_h/dt = f_2, di_v/dt = f_3$.

The Jacobian matrix or variational matrix is given by:

$$J = \begin{pmatrix} \partial f_1 / \partial s_h & \partial f_1 / \partial i_h & \partial f_1 / \partial i_v \\ \partial f_2 / \partial s_h & \partial f_2 / \partial i_h & \partial f_2 / \partial i_v \\ \partial f_3 / \partial s_h & \partial f_3 / \partial i_h & \partial f_3 / \partial i_v \end{pmatrix}$$

Hence,

$$J = \begin{pmatrix} -Bi_v - \alpha & 0 & -Bs_h \\ Bi_v & -\alpha - \gamma & Bs_h \\ 0 & \frac{\vartheta(1 - i_v)}{\epsilon} & \frac{-\vartheta i_h - \delta}{\epsilon} \end{pmatrix}$$

Therefore the Jacobian matrix of (10)-(12) at the disease-free equilibrium E_0 is

$$J(E_0) = \begin{pmatrix} -\alpha & 0 & -B\left(\frac{1+\xi}{\alpha}\right) \\ 0 & -\alpha - \gamma & B\left(\frac{1+\xi}{\alpha}\right) \\ 0 & \frac{\vartheta}{\epsilon} & \frac{-\delta}{\epsilon} \end{pmatrix} \quad (23)$$

The characteristic equation of (23) is given by

$$|J(E_0) - \lambda I| = 0$$

$$\begin{vmatrix} -\alpha - \lambda & 0 & -B\left(\frac{1+\xi}{\alpha}\right) \\ 0 & -\alpha - \gamma - \lambda & B\left(\frac{1+\xi}{\alpha}\right) \\ 0 & \frac{\vartheta}{\epsilon} & \frac{-\delta}{\epsilon} - \lambda \end{vmatrix} = 0$$

$$(-\alpha - \lambda) \begin{vmatrix} -\alpha - \gamma - \lambda & B\left(\frac{1+\xi}{\alpha}\right) \\ \frac{\vartheta}{\epsilon} & \frac{-\delta}{\epsilon} - \lambda \end{vmatrix} = 0$$

$$(-\alpha - \lambda) \left[(-\alpha - \gamma - \lambda) \left(\frac{-\delta}{\epsilon} - \lambda \right) - \left(\frac{\vartheta}{\epsilon} \right) B\left(\frac{1+\xi}{\alpha}\right) \right] = 0$$

$$(-\alpha - \lambda) \left[(\alpha + \gamma + \lambda) \left(\frac{\delta}{\epsilon} + \lambda \right) - \left(\frac{\vartheta}{\epsilon} \right) B\left(\frac{1+\xi}{\alpha}\right) \right] = 0$$

$$(-\alpha - \lambda) \left[\lambda^2 + \left(k + \frac{\delta}{\epsilon} \right) \lambda + k \frac{\delta}{\epsilon} - k \frac{\delta}{\epsilon} R_0^2 \right] = 0$$

where $k = \alpha + \gamma > 0$

$$(-\alpha - \lambda) \left[\lambda^2 + \left(k + \frac{\delta}{\epsilon} \right) \lambda + k \frac{\delta}{\epsilon} (1 - R_0^2) \right] = 0$$

$$-\alpha - \lambda = 0, \lambda^2 + \left(k + \frac{\delta}{\epsilon} \right) \lambda + k \frac{\delta}{\epsilon} (1 - R_0^2) = 0$$

Thus the eigenvalues of (2.19) are

$$\lambda_1 = -\alpha; \lambda_2 = \frac{-\left(k + \frac{\delta}{\epsilon}\right) - \sqrt{\left(k - \frac{\delta}{\epsilon}\right)^2 + 4k \frac{\delta}{\epsilon} R_0^2}}{2};$$

$$\lambda_3 = \frac{-\left(k + \frac{\delta}{\epsilon}\right) + \sqrt{\left(k - \frac{\delta}{\epsilon}\right)^2 + 4k \frac{\delta}{\epsilon} R_0^2}}{2}$$

It can be observed that the eigenvalues λ_1 and λ_2 are always negative quantities. However, λ_3 is negative if $R_0 < 1$ and it is positive if $R_0 > 1$.

Recall that an equilibrium point is stable if all the eigenvalues of the next generation matrix are negatives; and unstable if at least one of the eigenvalues is positive.

Thus it can be concluded that the disease free equilibrium E_0 of the present model is stable if $R_0 < 1$ and unstable if $R_0 > 1$. Hence the statement of theorem 1 is proved.

5. Numerical Simulations

In this section the numerical simulations of the model are carried out using a DED is cover 2.6.4 and the results are presented. The initial conditions used are: $s_{h0} = 0.5$, $i_{h0} = 0.5$, and $i_{v0} = 0.1$. The parametric values chosen for the purpose of simulation are given in Table 2.

Figure 2. Numerical simulation of fraction of population with respect to time with $R_0 = 0.433013 < 1$.

Figure 3. Numerical simulation of fraction of population with respect to time with $R_0 = 0.433229 < 1$.

Figure 4. Numerical simulation of fraction of population with respect to time with $R_0 = 3.107277 > 1$.

Figure 5. Numerical simulation of fraction of population with respect to time with $R_0 = 10.8562 > 1$.

Figure 6. Numerical simulation of fraction of population with respect to time with $R_0 = 0.433662 < 1$.

Table 2. Parametric values used in the simulation study.

Figure	B	α	γ	ϑ	δ	ϵ	ξ	R_0
2	0.01	1	0.6	0.3	1	0.1	0	0.433013
3	0.01	1	0.6	0.3	1	0.1	0.001	0.433229
4	12	0.9	2	10	0.5	0.7	0.4	3.107277
5	15	1	0.4	110	1	0.001	0.5	10.8562
6	0.01	1	0.6	0.3	1	0.001	0.003	0.433662

Figure 7. Reproduction number versus Immigration rate of susceptible human population.

6. Results and Discussion

In this paper, a SIR model is applied to malaria transmission between human and mosquito populations and studied the dynamics of the disease. The model scaled so as to reduce the number of parameters involved and to highlight the important parameters. The basic reproduction number is derived to visualize the dynamics of the disease. The existence of disease free equilibrium of model is identified and shown that it is stable.

The mathematical analysis and the simulation study shows that if the reproduction number is less than one then the DFE is locally asymptotically stable. Also in this case thought the initial sizes of human and mosquito populations are positive quantities, as the time progresses only susceptible human population remain present in the system while the other populations reduce to zero. That is, the disease dies out over a period of time. This fact is very evident in the simulation results presented in figures 2, 3, and 6.

Further, the mathematical analysis and the simulation study also show that if the reproduction number is greater than one then the DFE is unstable. Also in this case the human and mosquito population sizes start with some positive values and as the time progresses they reach their respective carrying capacities and remain present in the system. That is, the disease does not die. This fact is very evident in the simulation results presented in figures 4 and 5.

Clearly, from the numerical simulations, the DFE is asymptotically stable whenever the reproduction number is less than one. Further, it is noticed that in order to reduce the basic reproduction number below one, it is needed to focus on reduction of the contact rates between mosquitoes and human populations, and also reduce the immigration rate.

Lastly, it is revealed that as the immigration rate increases the reproduction number increases and after certain time progress leading the reproduction number to exceed one

which is shown in graph 7. Therefore the malaria disease persists in the population.

7. Conclusion

In this paper, a mathematical model for describing the dynamics of malaria disease is formulated taking into account the compartmentalization of both the human and mosquito populations. An SIR model with the inclusion of immigration in to susceptible compartment is formulated for human population. An SI model is formulated for mosquito population. The dynamics of mosquito population is studied along with that of the human population since the mosquito population determines whether a malaria disease will occur or not.

The transmission coefficient between susceptible humans and infected mosquitoes β_h ; infected humans and susceptible mosquitoes β_v are the important factors in the spread of malaria.

The reproduction number being an indicator that indicates the spread of malaria is derived. By definition it is true that disease persists if the reproduction number exceeds one. The stability condition of the equilibrium points are derived and it is seen that if the reproduction number is less than one, the non-trivial equilibrium point is always locally asymptotically stable.

Simulation studies are performed by varying the values of some parameters of the model. If the immigration constant β increases, then it will lead to an increase in the number of infected humans, giving an endemic shape to the disease. So one way to control the outbreak of malaria is to put a check on the immigration coefficient β , i.e., to curb the immigration of susceptible population.

In conclusion, there is need for effective control program against susceptible human Immigrants as they contribute to the outbreak of malaria. Spraying insecticides and proper drainage system should be a major target to eliminate mosquitoes. Early diagnosis and proper treatment with anti-malarial drugs can help to control the spread of malaria.

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