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Research Paper

Mathematical Model for the Dynamics and Control of Malaria in a Population

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Abstract: *Malaria, as a disease, is so rampant in the society to the extent that it is viewed as, a fast becoming endemic disease, particularly in Africa. In this study we developed a mathematical model to study the dynamics of the disease in a population, in developing the model; a population is compartmentalized into susceptible and infected classes taking into consideration the interaction between the parasites and the host (human beings), also the susceptible and the infected classes are allowed to interact freely without quarantining either class. The model ushered in a system of first order equation that describes the dynamics of the susceptible class and the infected class under the influence of the parasite. Qualitative and stability analysis were carried out, the result of the analysis showed that if preventive measures are not put in place, the susceptible and infected classes will reach a stable equilibrium point which can be disastrous to the population. The study recommended specific measures to control the disease.*

Keywords: Parasite, host, susceptible, panmictic.

1. Introduction

Malaria is a life-threatening disease widely spread in tropical and subtropical regions, including Africa, Asia, Latin America, the Middle East and some parts of Europe and that most cases and deaths occur in sub-Saharan Africa (Rollback Malaria, 2010). In 2006 there were almost 250 million cases of malaria, causing nearly one million deaths (Rollback Malaria, 2010). Malaria is caused by parasites of the species Plasmodium; the parasites are transmitted to humans through the bites of infectious female mosquitoes (vectors). The malaria parasite enters a human when an infectious mosquito bites a person. After entering a human the parasite transforms through a complicated life-cycle. The parasites multiply in the human liver and bloodstream. Finally, when it has developed into an infectious form, it spreads the disease to a new mosquito that bites the infectious human. After approximately 10 to 15

days the mosquito takes her next blood meal and can infect a new person. After a human gets bitten the symptoms appear in about 9 to 14 days (Rollback Malaria, 2010). By making appropriate models for the spread of malaria one can understand the underlying processes and develop effective prevention strategies, furthermore, one of the most basic epidemiological models is the so called SIR model (Rollback Malaria, 2010). The model describes the different states which a human can be in. The three states are susceptible, infectious and recovered. Humans enter the system in the susceptible state when born at rate μ_1. A susceptible human enters the infectious state at rate σ_1 when receiving the disease. From the infected state the human can either moves to the recovered state at rate σ_2 or the human can leave the system by death at rate d. Humans can also leave the system by immigration and natural death at rate μ_2. The total population is denoted as N, Johansson and Jacob Leander (2010). The interaction between the states of this model as given by Johansson and Jacob Leander (2010) is illustrated in Figure 1 below;

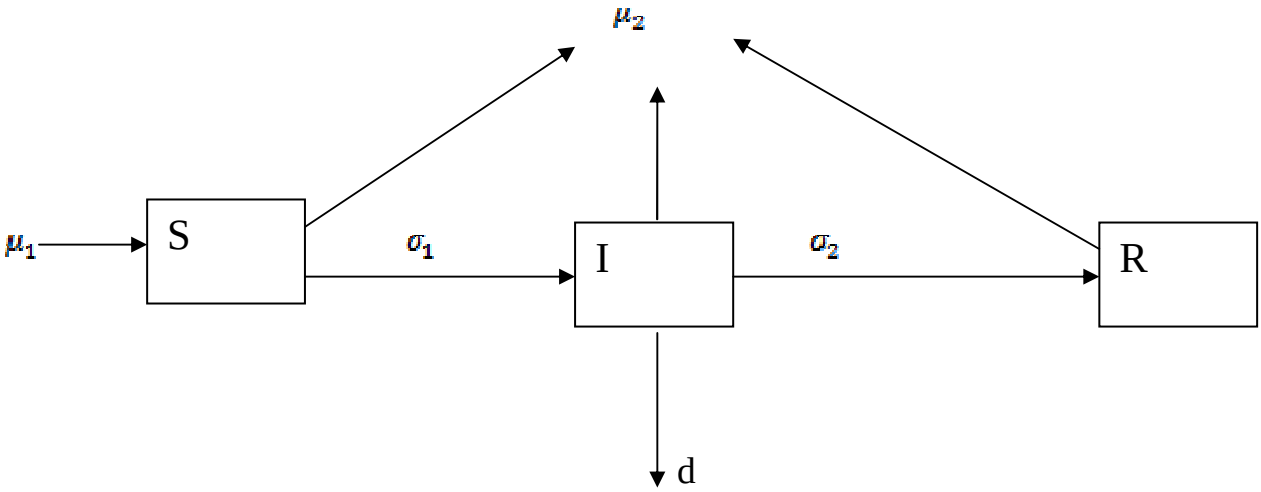


Figure 1: The basic SIR model with rates σ_1, σ_2, μ_1, μ_2 and d

Johansson and Jacob Leander (2010) developed the mathematical model, based on the above schematic model, and is given by the following system of DE

$$\frac{dS}{dt} = N\mu_1 - S(\mu_2 + \sigma_1)$$
$$\frac{dI}{dt} = S\sigma_1 - I(\sigma_2 + d + \mu_2)$$
$$\frac{dR}{dt} = I\sigma_2 - R\mu_2$$

Gadeon A. Ngwa and William S. Shu (1999) derived a deterministic differential equation model for endemic malaria involving variable human and mosquito populations, they analyzed the differential equation and derived conditions for the existence of endemic and disease free equilibria. They showed that a threshold parameter $\bar{R}$ exists and the disease can persist if and only if $\bar{R}o$ exceeds 1. The disease free equilibrium always exists and is globally stable when $\bar{R}$ is below 1. They used numerical simulations to show that the endemic equilibrium, when it exists, is unique and is globally stable.

A. A. Momoh *et al* (2012) carried out a research that deals with mathematical modeling of malaria transmission in North Senatorial Zone of Taraba State, Nigeria. The SIR proposed by Kermack and McKendrick was implemented using data obtained from Essential Programme on Immunisation (EPI) unit, F.M.C., Jalingo, Taraba state, the data was used to analyse the rate of infection of malaria in the zone. From their analysis, they found out that the reproduction ratio is greater than 0, which implies

that the force of malaria infection in Taraba North Senatorial Zone is high. A.A. Momoh et al (2012) recommended specific steps for the reduction of malaria in the zone.

Jacob C. Koella (1990) carried out a review study on mathematical models of malaria with emphasis on their relevance for control. They concluded that; Ross-Macdonald model of malaria transmission has had major influence on malaria control, and that one of its main contributions is that endemicity of malaria is most sensitive to changes in mosquito imago survival rate, and hence malaria can be controlled more efficiently with imagicites than with larvicides. Jacob C. Koella (1990) further opined that Models that describe the immune response and simulate vaccination programs suggest that one of the most important determinants of the outcome of a vaccine campaign is the duration of vaccine efficacy. Apparently malaria can be controlled only if the duration of efficacy is in the order of a human life-span.

Sandip Mandal *et al* (2011) carried out a survey work, in their article, starting from the basic Ross model, the key mathematical models and their underlying features, based on their specific contributions in the understanding of spread and transmission of malaria were discussed. The first aim of their article is to develop, starting from the basic models, a hierarchical structure of a range of deterministic models of different levels of complexity. The second objective is to elaborate, using some of the representative mathematical models, the evolution of modelling strategies to describe malaria incidence by including the critical features of host-vector-parasite interactions. They laid more emphasis on the evolution of the deterministic differential equation based epidemiological compartment models with a brief discussion on data based statistical models. In their comprehensive survey, the approach has been to summarize the modelling activity in this area so that it helps reach a wider range of researchers working on epidemiology, transmission, and other aspects of malaria. This, they believed, may facilitate mathematicians to further develop suitable models in this direction relevant to the present scenario, and help the biologists and public health personnel to adopt better understanding of the modelling strategies to control the disease

Avordeh *et al* (2012) developed a mathematical model to study the transmission of malaria; they showed from the study that the model has unique disease-free and endemic equilibria in the application of treatment and preventive measures. We analyze the existence and stability of disease-free and endemic malaria equilibria. Key to their study is the definition of a reproductive number R_0 which is the number of the new infections caused by one individual in an otherwise fully susceptible population, through the duration of the infectious period. The disease-free equilibrium is locally asymptotically stable, if $R_0 \leq 1$ and that the endemic equilibrium exist provided $R_0 > 1$. In fact, their study showed clearly that effective treatment offered to about fifty percent of the infected population together with about fifty percent prevention rate is all that is required to eliminate the disease.

From the work by Johansson and Jacob Leander (2010), it can be seen that the three states which a human can be in are; susceptible, infectious and recovered. However, in reality the recovered class is not removed or quarantined from the main population, in fact they become part of the susceptible class. In this work, we present a reviewed dynamical model for the malaria parasite/host dynamics on this basis.

2. Materials and Method

2.1 Assumption

- ✓ Birth rate is constant
- ✓ Natural death is constant
- ✓ Death due to infection is constant
- ✓ Population is panmictic
- ✓ There is a free intra population interaction between the populations of parasites and humans
- ✓ The disease is spread by transmission through parasite - human interaction

- ✓ At time t=0, the susceptible population S equals the birth rate, while the infected population equals to the infected persons at t=0
- ✓ The population size is large
- ✓ No selection for habitation
- ✓ No immigration allowed

2.2 Notations

S – Susceptible population
I – Infected population
B_H – Human birth rate
B_P – Parasite birth rate
β – Rate of transmission from susceptible state to infected state
λ – Rate of recovery from infected state
μ - Natural birth rate
δ – Death rate due to infection
t – Time
B_0 – Susceptible population at time t=0
I_0 – Infected population at time t=0

2.3 The Dynamics of a Population under the Influence of Malaria Parasite

A human moves through susceptible, infectious and recovered states at different rates, however when a human recovers he is neither quarantined nor conferred with immunity against the disease, as such he is still susceptible to infection by the parasites, and so he belongs to the susceptible class. Hence the recovered class is contained within the susceptible class. Now humans enter the system in the susceptible state when born at rate B_H. A susceptible human enters the infectious state at rate β when receiving the disease and can leave the susceptible class by natural death at rate μ. From the infected state the human can either move to the recovered (in this case the susceptible) state at rate λ or the human can leave the infected class by natural death at rate μ or death due to the disease at a rate δ. The interaction between the states as stated above is illustrated in Figure 1 below;

Schematic Diagram

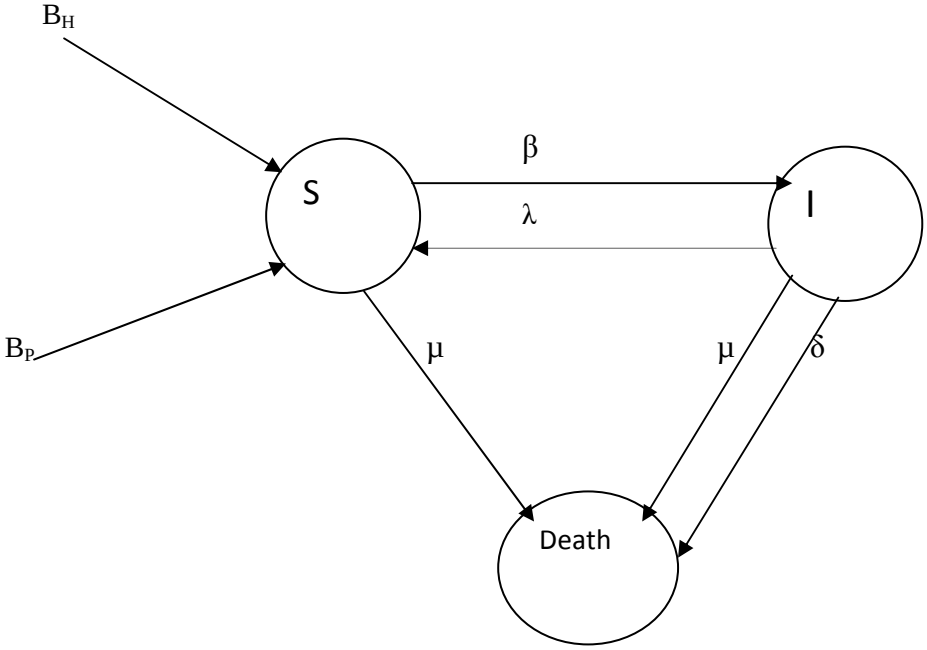


Fig 2: Schematic diagram for the dynamics of malaria

2.4 Model Development

The mathematical model governing the above schematic diagram is given as

$$\frac{dS}{dt} = B_H - \beta S + \lambda I - \mu S = B_H - (\beta + \mu)S + \lambda I\,, \qquad S(0) = B_0$$

$$\frac{dI}{dt} = \beta S - \lambda I - (\mu + \delta)I \quad = \beta S - (\lambda + \mu + \delta)I, \qquad I(0) = I_0$$

Now considering the equations

$$\frac{dS}{dt} = B_H - \beta S + \lambda I - \mu S, \qquad S(0) = B_0 \qquad \ldots \qquad (1)$$

$$\frac{dI}{dt} = \beta S - \lambda I - (\mu + \delta)I, \qquad I(0) = I_0 \qquad \ldots \qquad (2)$$

Assuming a steady state for human birth rate, i.e. $B_H(t) = B_H$, and differentiating (1) with respect to t we have;

$$\frac{d^2S}{dt^2} = -(\beta + \mu)S' + \lambda I' \qquad \ldots \qquad (3)$$

Using (1) and (2) in (3), we have

$$\frac{d^2S}{dt^2} = -(\beta + \mu)(B_H - (\beta + \mu)S + \lambda I) + \lambda(\beta S - (\lambda + \mu + \delta)I)$$
$$= ((\beta + \mu)^2 + \lambda\beta)S - \big((\beta + \mu)\lambda + (\lambda + \mu + \delta)\lambda\big)I - (\beta + \mu)B_H \ldots (4)$$

From (1), we have;

$$I = \frac{1}{\lambda}\left(\frac{dS}{dt} - B_H + (\beta + \mu)S\right) \qquad \ldots \qquad (5)$$

Therefore

$$\frac{d^2S}{dt^2} = ((\beta + \mu)^2 + \lambda\beta)S - \big((\beta + \mu)\lambda + (\lambda + \mu + \delta)\lambda\big)\frac{1}{\lambda}\left(\frac{dS}{dt} - B_H + (\beta + \mu)S\right)$$
$$- (\beta + \mu)B_H \qquad \ldots \qquad (6)$$

$$= -\frac{1}{\lambda}\big((\beta + \mu)\lambda + (\lambda + \mu + \delta)\lambda\big)\frac{dS}{dt} + \Big[(\beta + \mu)^2 + \lambda\beta - \frac{1}{\lambda}\big((\beta + \mu)\lambda + (\lambda + \mu + \delta)\lambda\big)(\beta + \mu)\Big]S + \Big[\frac{1}{\lambda}\big((\beta + \mu)\lambda + (\lambda + \mu + \delta)\lambda\big) - (\beta + \mu)\Big]B_H$$

=

$$-\big((\beta + \mu) + (\lambda + \mu + \delta)\big)\frac{dS}{dt} + \big[(\beta + \mu)^2 + \lambda\beta - \big((\beta + \mu) + (\lambda + \mu + \delta)\big)(\beta + \mu)\big]S + \big[\big((\beta + \mu) + (\lambda + \mu + \delta)\big) - (\beta + \mu)\big]B_H$$

$$= -(\beta + 2\mu + \lambda + \delta)\frac{dS}{dt} + [\,(\beta+\mu)^2 + \lambda\beta - (\beta + 2\mu + \lambda + \delta)(\beta+\mu)]S + (\lambda + \mu + \delta)\,B_H$$

Let K = $\beta + 2\mu + \lambda + \delta$, then;

$$\frac{d^2S}{dt^2} = -K\frac{dS}{dt} + [\,(\beta+\mu)^2 + \lambda\beta - K(\beta+\mu)]S + (\lambda + \mu + \delta)\,B_H$$

$$\frac{d^2S}{dt^2} = -K\frac{dS}{dt} + [(\beta+\mu)[(\beta+\mu) - K] + \lambda\beta]S + (\lambda + \mu + \delta)\,B_H$$

Let $\sigma = (\beta+\mu)[(\beta+\mu) - K] + \lambda\beta$

$\gamma = \lambda + \mu + \delta$

Therefore

$$\frac{d^2S}{dt^2} = -K\frac{dS}{dt} + \sigma S + \gamma B_H$$

$$\equiv \frac{d^2S}{dt^2} + K\frac{dS}{dt} - \sigma S = \gamma B_H \quad \ldots \quad (7)$$

We now solve the above non homogeneous O.D.E. The complementary solution is given as

$$S_c(t) = C_1 exp\frac{1}{2}\left[-K + \sqrt{K^2 + 4\sigma}\right]t + C_2 exp\frac{1}{2}[-K - \sqrt{K^2 + 4\sigma}]t$$

For the particular solution $S_p(t)$, we have, using the method of undetermined coefficient, that;

$$S_p(t) = \frac{\gamma}{\sigma}B_H \quad \ldots \quad (8)$$

Therefore the complete solution for the O.D.E is given by

$$S = S_c(t) + S_p(t) = C_1 exp\frac{1}{2}\left[-K + \sqrt{K^2 + 4\sigma}\right]t + C_2 exp\frac{1}{2}\left[-K - \sqrt{K^2 + 4\sigma}\right]t + \frac{\gamma}{\sigma}B_H \ldots (9)$$

Now from equation (5), we have;

$$I = \frac{1}{\lambda}\left(\frac{dS}{dt} - B_H + (\beta+\mu)S\right)$$

=

$$\frac{1}{\lambda}\{\tfrac{1}{2}\left[-K + \sqrt{K^2 + 4\sigma}\right]C_1 exp\tfrac{1}{2}\left[-K + \sqrt{K^2 + 4\sigma}\right]t + \tfrac{1}{2}\left[-K - \sqrt{K^2 + 4\sigma}\right]C_2 exp\tfrac{1}{2}\left[-K - \sqrt{K^2 + 4\sigma}\right]t$$

$$-B_H + (\beta+\mu)[C_1 exp\tfrac{1}{2}\left[-K + \sqrt{K^2 + 4\sigma}\right]t + C_2 exp\tfrac{1}{2}\left[-K - \sqrt{K^2 + 4\sigma}\right]t + \frac{\gamma}{\sigma}B_H]\}$$

$$= \frac{1}{\lambda}\{\tfrac{1}{2}[-K + \sqrt{K^2 + 4\sigma} + (\beta + \mu)]C_1 exp\tfrac{1}{2}[-K + \sqrt{K^2 + 4\sigma}]t + \tfrac{1}{2}[-K - \sqrt{K^2 + 4\sigma} + (\beta + \mu)]C_2 exp\tfrac{1}{2}[-K - \sqrt{K^2 + 4\sigma}]t$$
$$-B_H + \frac{\gamma}{\sigma}B_H]\}$$

Let $\eta_1 = \frac{1}{2}[-K + \sqrt{K^2 + 4\sigma}]$

$\eta_2 = \frac{1}{2}[-K - \sqrt{K^2 + 4\sigma}]$

We then have

$$I = \frac{1}{\lambda}\{[\eta_1 + (\beta + \mu)]C_1 exp(-\eta_1)t + [\eta_2 + (\beta + \mu)]C_2 exp(-\eta_2)t - B_H + \frac{\gamma}{\sigma}B_H]\}$$

$$I = [\eta_1 + (\beta + \mu)]\frac{C_1}{\lambda} exp(-\eta_1)t + [\eta_2 + (\beta + \mu)]\frac{C_2}{\lambda} exp(-\eta_2)t$$

$$-\frac{B_H}{\lambda} + \frac{\gamma}{\sigma\lambda}B_H] \quad \ldots \quad (10)$$

Equation (9) and (10) constitute the solution to the system (equation (1) & (2))

Next we determine the constants C_1 and C_2 using the initial conditions. Now;

$S(0) = B_0 = C_1 + C_2 + \frac{\gamma}{\sigma}B_H$ … (11)

$I(0) = I_0 = [\eta_1 + (\beta + \mu)]\frac{C_1}{\lambda} + [\eta_2 + (\beta + \mu)]\frac{C_2}{\lambda} - \frac{B_H}{\lambda} + \frac{\gamma}{\sigma\lambda}B_H]$ … (12)

From equation (11), we have;

$$C_1 = B_0 - C_2 - \frac{\gamma}{\sigma}B_H \quad \ldots \quad (13)$$

Using (13) in (12), we have;

$$I_0 = [\eta_1 + (\beta + \mu)]\frac{(B_0 - C2 - \frac{\gamma}{\sigma}B_H)}{\lambda} + [\eta_2 + (\beta + \mu)]\frac{C_2}{\lambda} - \frac{B_H}{\lambda} + \frac{\gamma}{\sigma\lambda}B_H]$$

$$= [\eta_1 + (\beta + \mu)]\frac{B_0}{\lambda} - \frac{\gamma}{\sigma\lambda}B_H[\eta_1 + (\beta + \mu)] + [(\eta_2 + (\beta + \mu)) - (\eta_1 + (\beta + \mu))]\frac{C_2}{\lambda}$$
$$-\frac{B_H}{\lambda} + \frac{\gamma}{\sigma\lambda}B_H]$$

$$\Rightarrow C_2 = \frac{\left[I_0 + \frac{\gamma}{\sigma\lambda}B_H[\eta_1 + (\beta + \mu)] - [\eta_1 + (\beta + \mu)]\frac{B_0}{\lambda} + \frac{B_H}{\lambda} - \frac{\gamma}{\sigma\lambda}B_H\right]\lambda}{(\eta_2 + (\beta + \mu)) - (\eta_1 + (\beta + \mu))}$$

$$C_2 = \frac{\left[I_0 + \frac{\gamma}{\sigma\lambda}B_H[\eta_1 + (\beta + \mu)] - [\eta_1 + (\beta + \mu)]\frac{B_0}{\lambda} + \frac{B_H}{\lambda} - \frac{\gamma}{\sigma\lambda}B_H\right]\lambda}{(\eta_2 - \eta_1)} \quad \ldots \quad (14)$$

Using (14) into (13) to solve for C_2, we have

$$C_1 = B_0 - \frac{\left[I_0 + \frac{\gamma}{\sigma\lambda}B_H[\eta_1 + (\beta+\mu)] - [\eta_1 + (\beta+\mu)]\frac{B_0}{\lambda} + \frac{B_H}{\lambda} - \frac{\gamma}{\sigma\lambda}B_H\right]\lambda}{(\eta_2 - \eta_1)} - \frac{\gamma}{\sigma}B_H$$

$$C_1 = \frac{\left[\left(B_0 - \frac{\gamma}{\sigma}B_0\right)(\eta_2 - \eta_1) - I_0\lambda + \left(\frac{\gamma B_H}{\sigma} - B_0\right)(\eta_1 + (\beta+\mu))\left(1 - \frac{\gamma}{\sigma}\right)B_H\right]}{(\eta_2 - \eta_1)}$$

3. Results and Discussion

3.1 Results

The mathematical model describing the interaction between malaria parasites and the host as developed above is given as;

$$\frac{dS}{dt} = B_H - \beta S + \lambda I - \mu S, \qquad S(0) = B_0$$

$$\frac{dI}{d2t} = \beta S - \lambda I - (\mu + \delta)I, \qquad I(0) = I_0$$

The solution to the system of equations above is given as

$$S = C_1 exp\frac{1}{2}\left[-K + \sqrt{K^2 + 4\sigma}\right]t + C_2 exp\frac{1}{2}\left[-K - \sqrt{K^2 + 4\sigma}\right]t + \frac{\gamma}{\sigma}B_H$$

$$I = [\eta_1 + (\beta + \mu)]\frac{C_1}{\lambda}exp(-\eta_1)t + [\eta_2 + (\beta + \mu)]\frac{C_2}{\lambda}exp(-\eta_2)t - \frac{B_H}{\lambda} + \frac{\gamma}{\sigma\lambda}B_H]$$

Where

$$C_1 = \frac{\left[\left(B_0 - \frac{\gamma}{\sigma}B_0\right)(\eta_2 - \eta_1) - I_0\lambda + \left(\frac{\gamma B_H}{\sigma} - B_0\right)(\eta_1 + (\beta+\mu))\left(1 - \frac{\gamma}{\sigma}\right)B_H\right]}{(\eta_2 - \eta_1)}$$

$$C_2 = \frac{\left[I_0 + \frac{\gamma}{\sigma\lambda}B_H[\eta_1 + (\beta + \mu)] - [\eta_1 + (\beta + \mu)]\frac{B_0}{\lambda} + \frac{B_H}{\lambda} - \frac{\gamma}{\sigma\lambda}B_H\right]\lambda}{(\eta_2 - \eta_1)}$$

3.2 Discussion

3.2.1 Equilibrium Analysis

At equilibrium point, we have

$$\frac{dS}{dt} = 0 \Rightarrow B_H - (\beta + \mu)S + \lambda I = 0$$

$$\Rightarrow S = \frac{B_H + \lambda I}{\beta + \mu} \qquad \ldots \qquad 15$$

$$\frac{dI}{dt} = 0 \Rightarrow \beta S - (\lambda + \mu + \delta)I = 0$$

$$\Rightarrow I = \frac{\beta}{\lambda + \mu + \delta} \quad S \qquad \ldots \qquad 16$$

15 and 16 gives the S and I null clines,

For the equilibrium points, from the null clines 15 and 16, we have

$$\left.\begin{array}{l}(\beta + \mu)S + \lambda I = B_H \\ \beta S - (\lambda + \mu + \delta)I = 0\end{array}\right\} \qquad \ldots \qquad (17)$$

Solving for I and S from the simultaneous equation (17) we have

$$I = \frac{\beta B_H}{\beta\lambda + (\beta + \lambda)(\lambda + \mu + \delta)} \qquad \ldots \qquad (18)$$

$$S = \frac{B_H(\lambda + \mu + \delta)}{\beta\lambda + (\beta + \lambda)(\lambda + \mu + \delta)} \qquad \ldots \qquad (19)$$

Equation (18) and (19) gives the equilibrium point solution for the model system of equations

The Jacobian of the system (consisting of ((1) & (2)) given as

$$\frac{dS}{dt} = B_H - (\beta + \mu)S + \lambda I = f(S,I)$$

$$\frac{dI}{dt} = \beta S - (\lambda + \mu + \delta)I = g(S,I)$$

is given by;

$$\begin{bmatrix} \frac{\partial f(S,I)}{\partial S} & \frac{\partial f(S,I)}{\partial I} \\ \frac{\partial g(S,I)}{\partial S} & \frac{\partial g(S,I)}{\partial I} \end{bmatrix} = \begin{bmatrix} -(\beta + \mu) & \lambda \\ \beta & -(\lambda + \mu + \delta) \end{bmatrix}$$

Now the determinant $\det(J-\lambda_1 I)=0$ gives the Eigen values, hence

$$\det(J - \lambda_1 I) = \begin{vmatrix} -(\beta + \mu) & \lambda \\ \beta & -(\lambda + \mu + \delta) \end{vmatrix} = 0$$

From which we obtained

$$\lambda_1 = \frac{-(\beta+2\mu+\lambda+\delta) \pm \sqrt{(\beta+2\mu+\lambda+\delta)^2 - 4(\beta+\mu)(\mu+\delta)+\mu\lambda}}{2}$$

$$\equiv \lambda_1 = \frac{-(\beta+2\mu+\lambda+\delta) \pm \sqrt{\beta^2+\lambda(2\beta+\lambda+2\delta)+\delta^2-2\beta\delta}}{2}$$

This gives the two roots $\lambda_{11}, \lambda_{12}$ as

$$\lambda_{11}=\frac{-(\beta+2\mu+\lambda+\delta)-\sqrt{(\beta^2+\lambda(2\beta+\lambda+2\delta)+\delta^2-2\beta\delta}}{2}$$

$$\lambda_{12}=\frac{-(\beta+2\mu+\lambda+\delta)+\sqrt{(\beta^2+\lambda(2\beta+\lambda+2\delta)+\delta^2-2\beta\delta}}{2}$$

Consider $(\beta^2+\lambda(2\beta+\lambda+2\delta)+\delta^2-2\beta\delta$. Rearranging the terms, we can re - write it as follows

$$\beta^2+\delta^2+\lambda^2+2\beta\lambda+2\delta(\lambda-\beta) \quad \ldots \quad (20)$$

Clearly, 20 is positive for $\forall\ \lambda \geq \beta$ and since all parameters are positive, therefore $\lambda_{11}<0$, while λ_{12} will be greater than zero ($\lambda_{12}>0$) if $\sqrt{(\beta^2+\lambda(2\beta+\lambda+2\delta)+\delta^2-2\beta\delta} > (\beta+2\mu+\lambda+\delta)$ and λ_{12} will be less than zero ($\lambda_{12}<0$) if

$$\sqrt{(\beta^2+\lambda(2\beta+\lambda+2\delta)+\delta^2-2\beta\delta} < (\beta+2\mu+\lambda+\delta).$$

Therefore, for $\lambda_{11}<0$, and $\lambda_{12}<0$, the equilibrium point is a sink, i.e. the solutions dies at the equilibrium point, and for $\lambda_{11}<0$, and $\lambda_{12}>0$, the equilibrium is a saddle point, and solutions explode as $t\rightarrow +\infty\ or\ t\rightarrow -\infty$ except at the separatix.

Now for λ_{12} to be greater than zero (0), we must have

$$(\beta^2+\lambda(2\beta+\lambda+2\delta)+\delta^2-2\beta\delta) > (\beta+2\mu+\lambda+\delta)^2$$

This simplifies to

$$4\mu(\beta+\lambda)+4\mu(\mu+\delta)+2\beta\delta < 0 \quad \ldots \quad (21)$$

Since all the parameters $\mu, \beta, \lambda\ \&\ \delta$ are positive, it means (21) is not possible, i.e. $\lambda_{11}<0$, and $\lambda_{12}>0$ is not possible, hence it is not possible for the solution S(t) & I(t) to explode away from the equilibrium points.

Also for $\lambda_{12}<0$, we must have

$$(\beta^2+\lambda(2\beta+\lambda+2\delta)+\delta^2-2\beta\delta) < (\beta+2\mu+\lambda+\delta)^2$$

$$\equiv 4\mu(\beta+\lambda)+4\mu(\mu+\delta)+2\beta\delta > 0 \quad \ldots \quad (22)$$

Since all the parameters $\mu, \beta, \lambda\ \&\ \delta$ are positive, it means (22) is possible, i.e. $\lambda_{11}<0$, and $\lambda_{12}<0$ is possible,

In this case the equilibrium point is a sink, which means solutions dies at the equilibrium point.

From our qualitative analysis above, we have the following results;

1) The S and I null clines are given as $S=\frac{B_H+\lambda I}{\beta+\mu}$ and $I=\frac{\beta}{\lambda+\mu+\delta}S$ respectively

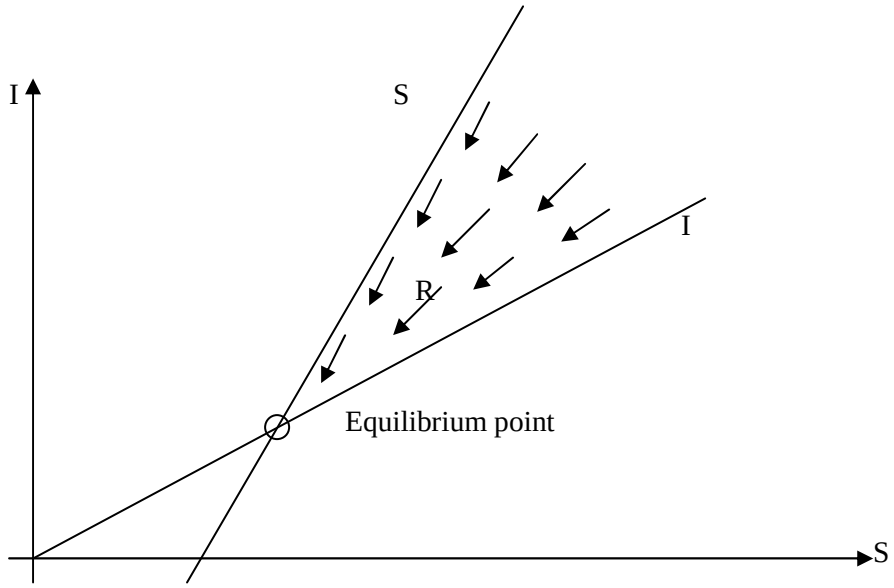
2) The equilibrium points were given as;

$$I=\frac{B_H\beta}{\beta\lambda+(\beta+\lambda)(\lambda+\mu+\delta)}$$

$$S = \frac{B_H(\lambda + \mu + \delta)}{\beta\lambda + (\beta + \lambda)(\lambda + \mu + \delta)}$$

The Eigen values were all real –ve (and distinct), this implies that the equilibrium point is a sink, i.e. all solutions dies at the equilibrium point.

Sketching the graph of the null clines and using the Eigen values of the system of equations gives;



Since the Eigen values are both real negative and distinct, the direction of motion (as long as the trajectories lies within the region R) is the same (left down).

From the above results, the direction movement (of S (t) & I(t)) is left down towards the equilibrium point suggest that malaria as a disease (if not controlled by any therapy) will gravitate towards a point where the infected and the susceptible population remain static. This may be partly due to the following reason;

> Malaria is caused by plasmodium parasites that resides comfortably in the body of mosquitoes (host), and without deliberate control policy, mosquitoes (host) will thrive well and controlled only by ecological factors like natural selection and thus interact freely with human population and consequently transmit the plasmodium parasite.

4. Conclusion

We conclude with the following recommendations.

a) Effort should be geared towards reducing the population of mosquitoes to bearest minimum, this will reduce the rate of human- mosquito contact and thus reducing the rate of spread of the plasmodium parasite that causes malaria. This can be achieved by providing good drainage system so that stagnant water and gutters which serves as breading sites for mosquitoes will be avoided.

b) Environment should cleared of grasses, shrubs, waste dump sites which also serves as breading sites for mosquitoes.

c) Fumigating public drainages and flower beds to eliminate mosquitoes living in those areas.

d) Providing periodic routine vaccinations for any reported case of infected person(s).

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