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Modeling the epidemiology of malaria and control with estimate of the basic reproduction number

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Abstract: Strategies for controlling the epidemiology of many infectious diseases such as malaria include a rapid reduction in both the infected and susceptible population via treatment and vaccination. In this paper, we have modified the Tumwiine *et al*. (2007) mathematical model for the transmission of malaria by including a vaccination parameter. We have shown that the model has a unique disease-free equilibrium state which is locally and globally asymptotically stable, if $R_o \leq 1$, and that the endemic equilibrium exist provided $R_o > 1$, where R_o is a parameter which depends on the given model parameters. Numerical simulations of the modified model clearly show that, with a proper combination of treatment and vaccination, offered at about 65% each on the susceptible and infected population, malaria can be eradicated from the community.

Keyword: Malaria, Disease-Free Equilibrium Point, Reproduction Number, Endemic Equilibrium Point, Global Asymptotical Stability, Lyaponuv Function

1. Introduction

Malaria is the common name for diseases caused by single-celled parasites of the genus Plasmodium. Among the parasites of the genus Plasmodium four species have been identified which can cause disease in humans. These include: Plasmodium falciparum, Plasmodium vivax, Plasmodium malaria and Plasmodium ovale. Of these, Plasmodium falciparum is of greatest risk to non-immune humans. The Plasmodium falciparum variety of parasites account for 80% of cases and 90% off deaths (Kakkilaya, 2003).Malaria remains arguably the greatest menace of our society in terms of morbidity and mortality and the occurrence of malaria in our part of the world correlates with poverty, ignorance and social deprivations in the community. An accurate knowledge of the incidence of malaria in endemic areas would be necessary towards the planning and development of effective preventive measures against the deadly scourge of malaria. Malaria is spread by the bite of an infected female mosquito, of the genus anopheles each time the infected insect takes a blood meal. The symptoms in an infected human include bouts of fever, headache, vomiting flu-like, anemia (destroying red blood cell) and malaria can kill by clogging the capillaries that carry blood to the brain (cerebral malaria) or other vital organs. On the average the incubation period of Plasmodium falciparum is about 12 days in humans. Malaria is endemic to tropical areas where the climatic and weather conditions allow continuous breeding of the mosquito. Malaria is one of the most important parasitic and infectious diseases especially in tropical and subtropical areas caused by protozoan parasites of the genus plasmodium. Malaria, affecting mainly children and pregnant women is one of the greatest menaces of our society in terms of morbidity and mortality and the occurrence of malaria in our part of the world correlates with poverty and ignorance (Perandin, 2003). Malaria is a major public health problem in the world. The WHO estimates that in tropical countries among the 500 million cases of malaria infection, one million deaths occur annually.

Malaria parasites are transmitted by female anopheles mosquitoes. Four species of plasmodium (P) causes human malaria. Among these, P. falciparum is responsible for most of the mortality P. Vivax causes considerable morbidity and P. malariae and P. ovale, are less prevalent around the world (Aslan and Seyrek, 2007). This group of human-

pathogenic Plasmodium species is usually referred to as malaria parasites The parasites multiply within red blood cells, causing symptoms that include symptoms of anaemia, as well as other general symptoms such as fever, chills, nausea, flu-like illness, and in severe cases, coma and death (Deressa et al., 2000). It is a disease that can be treated in just 48 hours, yet it can cause fatal complications if the diagnosis and treatment are delayed.

2. Role of Mathematical Model

It is important to establish the transmission dynamics of an epidemic in order to understand and predict it. Mathematical models are particularly helpful as experimental tools with which to evaluate and compare control procedures and preventive strategies, and to investigate the relative effects of various sociological, biological and environmental factors on the spread of diseases. These models have played a very important role in the history and development of vector-host epidemiology. Several authors have used mathematical models to analyse the transmission and spread of malaria. Mathematical models of malaria transmission that include both mosquito and human populations have been reviewed and discussed in detail by various authors. Nedelman (1985), did some further work on malaria model of Dietz et al. (1974), and showed that the "vaccination" rate depends on a pseudoequilibrium approximation to the differential equation describing the mosquito dynamics in the malaria model. Nedelman surveys various data sets to statistically approximate parameters such as inoculation rates, rates of recovery and loss of immunity in humans, human-biting rates of mosquitoes and infectivity and susceptibility of humans and mosquitoes. Dietz et al. (1974) proposed a model with two different classes of humans: one without immunity to malaria and one class with some immunity. As the non-immune class falls sick, some people recover with immunity. The immune class can get infected, but does not fall clinically ill and cannot be infectious. The model by Dietz et al. (1974) also included super infection, a phenomenon usually associated with macro parasites.

Yang (2000) describes a compartmental model where humans follow an SEIRS-type (with more than one immune class for humans) pattern and mosquitoes follow a Susceptible-Exposed-Infectious (SEI) pattern. Yang (2000) defines a reproductive number, R_O for this model and shows, through linear stability analysis, that the disease-free equilibrium is stable for $R_O < 1$. He also derived an expression for an endemic equilibrium that is biologically relevant only when $R_O > 1$. He used numerical simulations to support his proposition that for $R_O > 1$, the disease-free equilibrium is unstable and the endemic equilibrium is stable. The model for malaria transmission that we modified is an extension of the equations introduced by Tunwiine et al. (2007). In the Tunwiine model, humans follow an SIRS-like pattern and mosquitoes follow a SI pattern, similar to that described by Yang (2000) but with only one immune class for humans. Humans move from the susceptible to the infected class at some probability when they come into contact with an infectious mosquito, as in conventional SIRS models. However, infectious people can then recover with, or without, a gain in immunity; and either return to the susceptible class, or move to the recovered class. A new feature of this model is that although individuals in the recovered class are assumed to be "immune", in the sense that they do not suffer from serious illness and do not contract clinical malaria, they still have low levels of Plasmodium in their blood stream and can pass the infection to susceptible mosquitoes. After some period of time these recovered individuals return to the susceptible class. Susceptible mosquitoes get infected and move to the infected class, at some probability when they come into contact with either infectious humans or recovered humans (albeit at a much lower probability). Both humans and mosquitoes leave the population through a density dependent natural death rate. This allows the model to account for changing human and mosquito populations. Variations in mosquito populations are crucial to the dynamics of malaria, and constant population models do not account for this. The model also includes human disease-induced death as mortality for malaria in areas of high transmission can be high, especially in infants. In the modified model, we aim to capture some of the more important aspects of this epidemiology while still keeping it mathematically tractable. One of the major important factors that we include in the existing model is vaccination in order to determine its impact as a control measure for the spread of malaria.

2.1. Parameters and Terms of the Model

S_H (t) the number of susceptible human host at time t

I_H (t) the number of infected human host at time t

R_H (t) the number of partially immune human host at-time t

S_V (t) the number of susceptible mosquito vector at time t

I_V (t) the number of infected mosquito vectors at time t

$m = \frac{N_V}{N_H}$ the number of female mosquitoes per human host

a the average daily biting rate on man by a single mosquito (infection rate)

b the proportion of bites on man by a single mosquito that produce an infection

c the probability that a mosquito becomes infec- tious

γ the per capita rate of loss of immunity in human host

r the rate at which human host acquire immunity

δ the per capita death rate of infected human hosts due to the disease

v the rate of recovery of human host from the disease

λ_h the per capita natural birth rate of humans

λ_v the per capita natural birth rate of mosquitoes

μ_h the per capita natural death rate of humans

μ_v the per capita natural death rate of the mosquitoes

α the 'vaccination rate' on human

3. Equations of the Model

The model formulated by Tumwiine et al. (2007) is giving as

$$\frac{dS_H}{dt} = \lambda_h N_H - \frac{abS_H I_V}{N_H} + vI_H + \gamma R_H - \mu_h S_H \quad (3.1)$$

$$\frac{dI_H}{dt} = \frac{abS_H I_V}{N_H} - vI_H - rI_H - \delta I_H - \mu_h I_H \quad (3.2)$$

$$\frac{dR_H}{dt} = rI_H - \gamma R_H - \mu_h R_H \quad (3.3)$$

$$\frac{dS_V}{dt} = \lambda_v N_V - \frac{acS_V I_H}{N_H} - \mu_v S_V \quad (3.4)$$

$$\frac{dI_V}{dt} = \frac{acS_V I_H}{N_H} - \mu_v I_V \quad (3.5)$$

We assumed that all infected human who recovered are moved to the recovered class and vaccinated human have temporary immunity that expires over time and again become susceptible, hence by including a vaccination parameter, "α" the above model gives the modified model below

$$\frac{dS_H}{dt} = \lambda_h N_H - \frac{abS_H I_V}{N_H} + \gamma R_H - \mu_h S_H - \alpha S_H \quad (3.6)$$

$$\frac{dI_H}{dt} = \frac{abS_H I_V}{N_H} - rI_H - \delta I_H - \mu_h I_H \quad (3.7)$$

$$\frac{dR_H}{dt} = rI_H - \gamma R_H - \mu_h R_H + \alpha S_H \quad (3.8)$$

$$\frac{dS_V}{dt} = \lambda_v N_V - \frac{acS_V I_H}{N_H} - \mu_v S_V \quad (3.9)$$

$$\frac{dI_V}{dt} = \frac{acS_V I_H}{N_H} - \mu_v I_V \quad (3.10)$$

The total population sizes N_H and N_V can be determined by $S_H + I_H + R_H = N_H$ and $S_V + I_V = N_V$ or from the differential equations

$$\frac{dN_H}{dt} = (\lambda_h - \mu_h) N_H - \delta I_H \quad (3.11)$$

and

$$\frac{dN_V}{dt} = (\lambda_V - \mu_V) N_V \quad (3.12)$$

which are derived by adding equation (3.6) – (3.8) for the human population and (3.9) – (3.10) for the mosquito vector population.

In the model, the term $\frac{abS_H I_V}{N_H}$ denotes the rate at which the human hosts S_H get infected by infected mosquitoes I_V and $\frac{acS_V I_H}{N_H}$ refers to the rate at which the susceptible mosquitoes S_V are infected by infected human hosts I_H.

3.1. Existence and Stability of Equilibrium Solutions

In this section, we establish that the disease free equilibrium E_o exists if R_O <1. We also establish that the endemic equilibrium E_E exist for R_O >1.

Setting equation R.H.S of equation (3.6) - (3.10) to zero gives the disease equilibrium points:

$$E_O = \left(S_H^o, I_H^o, R_H^o, S_V^o, I_V^o\right) =$$

$$\left\{\frac{\lambda_h N_H(\mu_h+\gamma)}{\mu_h(\mu_h+\gamma+\alpha)}, 0, \frac{\lambda_h N_H \alpha}{\mu_h(\mu_h+\gamma+\alpha)}, \frac{\lambda_v N_V}{\mu_v}, 0\right\}$$

The disease-free equilibrium E_o exists for all nonnegative values of its parameters.

At the steady states of the model, the Jacobian matrix at E is given by

$$J_E = \begin{vmatrix} \frac{-abI_V}{N_H} - \mu_h - \alpha & 0 & \gamma & 0 & \frac{-abS_H}{N_H} \\ \frac{abI_V}{N_H} & -r-\delta-\mu_h & 0 & 0 & \frac{abS_H}{N_H} \\ \alpha & r & -\gamma-\mu_h & 0 & 0 \\ 0 & \frac{-acS_V}{N_H} & 0 & \frac{-acI_H}{N_H} - \mu_v & 0 \\ 0 & \frac{acS_V}{N_H} & 0 & \frac{acI_H}{N_H} & -\mu_v \end{vmatrix} \quad (3.13)$$

Evaluating the Jacobian matrix at E_O gives

$$J_{E_O} = \begin{vmatrix} -\mu_h - \alpha & 0 & \gamma & 0 & \left(\frac{-ab}{N_H}\right)\frac{\lambda_h N_H(\gamma+\mu_h)}{\mu_h(\mu_h+\alpha+\gamma)} \\ 0 & -r-\delta-\mu_h & 0 & 0 & \left(\frac{ab}{N_H}\right)\frac{\lambda_h N_H(\gamma+\mu_h)}{\mu_h(\mu_h+\alpha+\gamma)} \\ \alpha & r & -\gamma-\mu_h & 0 & 0 \\ 0 & \frac{-ac\lambda_v N_V}{N_H\mu_v} & 0 & -\mu_v & 0 \\ 0 & \frac{ac\lambda_v N_V}{N_H\mu_v} & 0 & 0 & -\mu_v \end{vmatrix} \quad (3.14)$$

The stability of the disease free equilibrium state can be obtained from studying the eigenvalues of J_{E_O}. If all the eigenvalues have negative real parts, then the equilibrium

points are locally asymptotically stable.

The five eigenvalues of J_{E_O} are

$$\lambda_1 = \lambda_4 = -\mu_h < 0$$

$\lambda_2 = K\left(-B + \sqrt{C}\right)$. This may be either nega-tive or positive

$$\lambda_3 = -K\left(B + \sqrt{C}\right) < 0$$

$$\lambda_5 = -\mu_h - \alpha - \gamma < 0$$

where

$$K = \frac{1}{2}\left\{\frac{1}{\mu_v \mu_h (\mu_h + \gamma + \alpha)}\right\} \quad (3.15)$$

$$\begin{aligned} B = & -\alpha\mu_v\mu_h\delta - \mu_h\mu_v r\gamma \\ & -\alpha\mu_v\mu_h r - \mu_h\mu_v\delta\gamma - \alpha\mu_v\mu_h^2 \\ & -\mu_h^2\mu_v r - \mu_h^2\mu_v\delta - \mu_h^2\mu_v\gamma \\ & -\alpha\mu_v^2\mu_h - \mu_v^2\mu_h^2 - \mu_h\mu_v^2\gamma - \mu_h^3\mu_v \end{aligned}$$

$$C = \left\{ \begin{aligned} & 4\mu_h^2\mu_v\alpha\lambda_v\lambda_v N_V\left(\frac{ab}{N_H}\right)\lambda_h N_H\left(\frac{ac}{N_H}\right) + 4\mu_h\mu_v\gamma^2\lambda_v N_V\left(\frac{ab}{N_H}\right)\lambda_h N_H\left(\frac{ac}{N_H}\right) + \\ & 8\mu_h^2\mu_v\lambda_v N_V\left(\frac{ab}{N_H}\right)\lambda_h N_H\left(\frac{ac}{N_H}\right)\gamma + 2\mu_h^5\mu_v^2 r - 4\mu_h^4\mu_v^3\gamma + \mu_h^4\mu_v^2\gamma^2 + 2\mu_h^5\mu_v^2\delta \\ & + \alpha^2\mu_v^4\mu_h^2 - 2\mu_h^4\mu_v^3\delta + 4\mu_h^3\mu_v\lambda_v N_V\left(\frac{ab}{N_H}\right)\lambda_h N_H\left(\frac{ac}{N_H}\right) + 2\alpha\mu_v^4\mu_h^3 + 2\mu_h^5\mu_v^2\gamma \\ & + 2\alpha^2\mu_v^2\mu_h^3\delta - 2\alpha^2\mu_v^3\mu_h^3 - 4\alpha\mu_v^3\mu_h^4 + \mu_h^4\mu_v^2 r^2 + 2\alpha\mu_v^2\mu_h^5 - 2\mu_h^4\mu_v^3 r + \alpha^2\mu_v^2\mu_h^2\delta^2 \\ & + 2\mu_h^2\mu_v^2 r\gamma^2\delta + 4\mu_h^3\mu_v^2 r\gamma\alpha + 4\mu_h^3\mu_v^2 r\gamma\delta - 4\mu_h^2\mu_v^3 r\gamma\alpha + 4\alpha\mu_v^2\mu_h^2\delta r\gamma + \mu_h^2\mu_v^4\gamma^2 \\ & - 2\alpha^2\mu_v^3\mu_h^2\delta - 4\alpha\mu_v^3\mu_h^3\delta + 4\alpha\mu_v^2\mu_h^4\delta + \mu_h^2\mu_v^2 r^2\gamma^2 + 2\mu_h^3\mu_v^2 r^2\gamma + \\ & 4\mu_h\mu_v\alpha\lambda_v N_V\left(\frac{ab}{N_H}\right)\lambda_h N_H\left(\frac{ac}{N_H}\right)\gamma + 2\mu_h^3\mu_v^2 r\gamma^2 + 2\alpha\mu_v^4\mu_h^2\gamma + \alpha^2\mu_v^2\mu_h^4 \\ & + 2\mu_v^4\mu_h^3\gamma + 2\alpha^2\mu_v^2\mu_h^2\delta r + 2\alpha\mu_v^2\mu_h^2\delta^2\gamma + 4\alpha\mu_v^2\mu_h^3\delta r + 4\alpha\mu_v^2\mu_h^3\delta\gamma - 4\alpha\mu_v^3\mu_h^2\delta\gamma \\ & + 2\alpha\mu_v^2\mu_h^3\delta^2 + 2\mu_h^4\mu_v^2 r\delta - 4\alpha\mu_v^3\mu_h^3 r + 4\alpha\mu_v^2\mu_h^4 r - \mu_h^2\mu_v^2\delta^2\gamma^2 + 2\mu_h^3\mu_v^2\delta^2\gamma + \\ & 2\mu_h^3\mu_v^2\delta\gamma^2 - 4\mu_h^3\mu_v^3\delta\gamma - 2\mu_h^2\mu_v^3\delta\gamma^2 + 4\mu_h^4\mu_v^2\delta\gamma + 2\alpha\mu_v^2\mu_h^4\gamma - 4\alpha\mu_v^3\mu_h^3\gamma + 4\mu_h^4\mu_v^2 r\gamma \\ & + \alpha^2\mu_v^2\mu_h^2 r^2 + 2\alpha^2\mu_v^2\mu_h^3 r + 2\alpha\mu_v^2\mu_h^3 r^2 - 2\alpha^2\mu_v^3\mu_h^2 r - 2\mu_h^3\mu_v^3\gamma^2 + 2\mu_h^2\mu_v^2 r^2\gamma\alpha \\ & - 4\mu_h^3\mu_v^3 r\gamma - 2\mu_h^2\mu_v^3 r\gamma^2 + \mu_h^4\mu_v^2\delta^2 + \mu_v^4\mu_h^4 - 2\mu_v^3\mu_h^5 + \mu_h^6\mu_v^2 \end{aligned} \right\}^{\frac{1}{2}} \quad (3.16)$$

The condition for λ_2 to be negative is that $-B + \sqrt{C}$ <0,

$$\text{i.e } B^2 - C > 0 \quad (3.17)$$

Equation 3.17 simplifies to give further simplification leads to

$$4\begin{pmatrix}\mu_h+\gamma\\+\alpha\end{pmatrix}\mu_h\mu_v\left\{\begin{matrix}\mu_h\mu_v^2\begin{pmatrix}\mu_h+r\\+\delta\end{pmatrix}\\-\lambda_v N_V\left(\frac{ab}{N_H}\right)\lambda_h N_H\left(\frac{ac}{N_H}\right)(\mu_h+\gamma+\alpha)\end{matrix}\right\} > 0 \quad (3.18)$$

$$\frac{\lambda_v N_V\left(\frac{ab}{N_H}\right)\lambda_h N_H\left(\frac{ac}{N_H}\right)(\mu_h+\gamma)}{\mu_h\mu_v^2(\mu_h+\delta+r)(\alpha+\gamma+\mu_h)} < 1 \quad (3.19)$$

The expression on the L.H.S is R_O, the basis reproduction number, therefore

$$R_O = \frac{\lambda_v N_V\left(\frac{ab}{N_H}\right)\lambda_h N_H\left(\frac{ac}{N_H}\right)(\mu_h+\gamma)}{\mu_h\mu_v^2(\mu_h+\delta+r)(\alpha+\gamma+\mu_h)} < 1 \quad (3.20)$$

R_O is an important threshold quantity. It is the expected number of secondary infection that one infectious individual would create over the duration of the infectious period. It is a determining factor as to whether a disease dies out or assumes endemicity.

Theorem 3.21

The disease-free equilibrium E_o in 3.21 is locally asymptotically stable if and only if $R_o \le 1$. Li et al (1999)

From the above theorem, it has been proven that the disease-free equilibrium is asymptotically stable if R_O <1

Existence of Endemic Equilibrium E_E exist if R_O > 1

The system of equation (3.5) – (3.10) has endemic equilibrium $E_E = \left(S_H^{'}, I_H^{'}, R_H^{'}, S_V^{'}, I_V^{'}\right)$ given by

$$S_H^{'} = \frac{A}{B}$$

$$I_H^{'} = \frac{C}{B}(Z-1)$$

$$R_H^{'} = \frac{C}{B}\left\{G + r\left(\frac{Z}{\mu_h+\gamma} - \frac{1}{\mu_h+\gamma+\alpha}\right)\right\}$$

$$S_V^{'} = \frac{D}{E}$$

$$I_V^{'} = \frac{C(Z-1)}{E}$$

where $Z = \dfrac{\left(\dfrac{ab}{N_H}\right)\left(\dfrac{ac}{N_H}\right)\lambda_h N_H \lambda_v N_V(\mu_h+\gamma)}{\mu_h\mu_v^2(\mu_h+\gamma+\alpha)(\mu_h+r+\delta)}$

and

$$A = \mu_v\left(\begin{matrix}\mu_v\\ +r+\delta\end{matrix}\right)\left\{\begin{matrix}\mu_h\mu_v\delta+\mu_h\mu_v\gamma\\ +\mu_v\delta\gamma+\lambda_h N_H\left(\dfrac{ac}{N_H}\right)\mu_h\\ +\lambda_h N_H\left(\dfrac{ac}{N_H}\right)\gamma+\mu_h^2\mu_v\\ +\mu_h\mu_v r\end{matrix}\right\}$$

$$B = \left\{\begin{matrix}\mu_h^3\mu_v+\mu_h^2\mu_v r\\ +\mu_h^2\lambda_v N_V\left(\dfrac{ab}{N_H}\right)+\alpha\mu_v\mu_h^2\\ +\mu_h\lambda_v N_V r\left(\dfrac{ab}{N_H}\right)+\alpha\mu_v\mu_h r+\mu_h^2\mu_v\gamma+\\ \mu_h\mu_v r\gamma+\mu_h^2\mu_v\delta\\ +\alpha\mu_v\mu_h\delta+\mu_h\delta\lambda_v N_V\left(\dfrac{ab}{N_H}\right)\\ +\mu_h\gamma\lambda_v N_V\left(\dfrac{ab}{N_H}\right)+\gamma\delta\lambda_v N_V\left(\dfrac{ab}{N_H}\right)\\ +\mu_h\mu_v\delta\gamma\end{matrix}\right\}\frac{ac}{N_H}$$

$$C = \mu_h\mu_v^2(\mu_h+\delta+r)(\alpha+\gamma+\mu_h)$$

$$D = \mu_h^3\mu_v+\mu_h^2\mu_v r$$
$$+\mu_h^2\lambda_v N_V\left(\frac{ab}{N_H}\right)+\alpha\mu_v\mu_h^2$$
$$+\mu_h\lambda_v N_V r\left(\frac{ab}{N_H}\right)+\alpha\mu_v\mu_h r$$
$$+\mu_h^2\mu_v\gamma+\mu_h\mu_v r\gamma+$$
$$\mu_h^2\mu_v\delta+\alpha\mu_v\mu_h\delta+\mu_h\delta\lambda_v N_V\left(\frac{ab}{N_H}\right)$$
$$+\mu_h\gamma\lambda_v N_V\left(\frac{ab}{N_H}\right)+\gamma\delta\lambda_v N_V\left(\frac{ab}{N_H}\right)+\mu_h\mu_v\delta\gamma$$

$$E = \frac{ab}{N_H}\left\{\begin{matrix}\mu_h\mu_v\delta+\mu_h\mu_v\gamma\\ +\mu_v\delta\gamma+\lambda_h N_H\left(\dfrac{ac}{N_H}\right)\mu_h\\ +\lambda_h N_H\left(\dfrac{ac}{N_H}\right)\gamma+\mu_h^2\mu_v+\mu_h\mu_v r\end{matrix}\right\}$$

$$G = \frac{\alpha\left\{\mu_v\delta+\mu_h\mu_v+\left(\dfrac{ac}{N_H}\right)\lambda_h N_H\right\}}{\mu_h\mu_v(\alpha+\gamma+\mu_h)}$$

Theorem 3.22

The endemic equilibrium, E_E exists if $R_O > 1$.

Proof: The endemic equilibrium point given in equation (3.6) – (3.10) from which theorem follows. We examine the following cases to establish the existence of the endemic equilibrium

Case 1

Invoking the positivity condition in the case of $I_H^{'}$ and $I_V^{'}$, it can be clearly verified that $R > 1$.

Case 2

In the case of $R_H^{'}$, we have a different situation. Here

$$\frac{R_O}{\mu_h+\gamma} - \frac{1}{\mu_h+\gamma+\alpha} > 0$$

If $R = 1$, $\dfrac{1}{\mu_h+\gamma} > \dfrac{1}{\mu_h+\gamma+\alpha}$

If $R > 1$, the inequality still holds. Therefore, the endemic equilibrium exists if $R > 1$. Clearly, the components of R are the same as R_O. Then the endemic equilibrium exists if $R_O > 1$

Global Asymptotic stability of the disease free equilibrium point

Theorem: The disease free-equilibrium E_o is globally asymptotically stable if $R_O \leq 1$.

Given that $R_O \leq 1$, then there exist only disease free equilibrium points.

Proof

At the disease free equilibrium, E_o, the following conditions hold

$$\lambda_h N_H = \left(\frac{ab}{N_H}\right) S_H^o I_H^o - \mu_h S_H^o + \gamma R_H^o - \alpha S_H^o$$

$$\alpha S_H^o + r I_H^o = (\mu_h + \gamma) R_H^o$$

$$\lambda_h N_H - \delta I_H^o = \mu_h N_H^o$$

$$\left(\frac{ab}{N_H}\right) S_H^o I_V^o = (\mu_h + r + \delta) I_H$$

$$\lambda_v N_V = \left(\frac{ac}{N_H}\right) S_V^o I_H^o + \mu_v S_V^o$$

$$\left(\frac{ac}{N_H}\right) S_V^o I_H^o = \mu_v I_V^o$$

Considering the Lyapunov function candidate $V(S_H, I_H, R_H, S_V, I_V): R^5 \rightarrow R^+$ defined as

$$\frac{1}{2}\left(S_H - S_H^o\right)^2 + \frac{1}{2}\left(I_H - I_H^o\right)^2 + \frac{1}{2}\left(R_H - R_H^o\right)^2 + \frac{1}{2}\left(S_V - S_V^o\right)^2 + \frac{1}{2}\left(I_V - I_V^o\right)^2$$

Differentiating V gives

$$\dot{V} = \left(S_H - S_H^o\right)\dot{S_H} + I_H \dot{I_H} + \left(R_H - R_H^o\right)\dot{R_H} + \left(S_V - S_V^o\right)\dot{S_V} + I_V \dot{I_V} + \left(N_H - N_H^o\right)\dot{N_H}$$

Imposing the condition on $\dot{V}$, gives the following

$$\dot{V} = \left(S_H - S_H^o\right)\begin{Bmatrix} \left(\frac{ab}{N_H}\right) S_H^o I_V^o \\ -\mu_h S_H^o + \gamma R_H^o - \alpha S_H^o \\ -\left[\left(\frac{ab}{N_H}\right) S_H I_V - \mu_h S_H + \gamma R_H^o - \alpha S_H\right] \end{Bmatrix}$$

$$+ I_H \begin{bmatrix} (\mu_h + r + \delta) I_H^o \\ -(\mu_h + r + \delta) I_H \end{bmatrix} + \left(R_H - R_H^o\right)\begin{bmatrix} (\mu_h + \gamma) R_H \\ -(\mu_h + \gamma) R_H^o \end{bmatrix} +$$

$$\left(N_H - N_H^o\right)\left[\mu_h N_H^o - \mu_h N_H\right]$$

$$+ \left(S_V - S_V^o\right)\begin{Bmatrix} \left(\frac{ac}{N_H}\right) S_V^o I_H^o \\ -\mu_v S_V^o - \left[\left(\frac{ac}{N_H}\right) S_V I_H - \mu_v S_V\right] \end{Bmatrix} +$$

$$I_V\left(\mu_v I_V^o + \mu_v I_V\right)$$

Finally

$$\dot{V} = S_H^o\left[\left(\frac{ab}{N_H}\right) I_V^o + \mu_h + \alpha\right]$$

$$-S_H\left[\left(\frac{ab}{N_H}\right) I_V + \mu_h + \alpha\right]$$

$$-I_H\left(I_H - I_H^o\right)(\mu_h + r + \delta)$$

$$-\left(R_H - R_H^o\right)^2(\mu_h + \gamma) - \left(N_H - N_H^o\right)^2 \mu_h$$

$$-\left(S_V - S_V^o\right)\left\{S_V\left[\left(\frac{ac}{N_H}\right) I_H + \mu_v\right] - S_V^o\left[\left(\frac{ac}{N_H}\right) I_H^o + \mu_v\right]\right\}$$

$$-I_V\left(\mu_v I_V - \mu_v I_V^o\right)$$

The following assumptions are made for the Lyapunov function $\dot{V}$ above

$$S_H^o\left[\left(\frac{ab}{N_H}\right) I_V^o + \mu_h + \alpha\right] - S_H\left[\left(\frac{ab}{N_H}\right) + \mu_h + \alpha\right]$$

is negative provided $I_V > I_V^o$

This condition warrants $-I_V\left(\mu_v I_V - \mu_v I_V^o\right)$ being negative.

$$-I_H\left(I_H - I_H^o\right)(\mu_h + r + \delta)$$

is negative if $I_H > \overset{o}{I}_H$ which when applied to

$$-\left(S_V - \overset{o}{S}_V\right)\left\{S_V\left[\left(\frac{ac}{N_H}\right)I_H + \mu_v\right] - \overset{o}{S}_V\left[\left(\frac{ac}{N_H}\right)\overset{o}{I}_H + \mu_v\right]\right\}$$

makes it negative.

It is also assumed that $R_H = \overset{o}{R}_H$

We have shown that $\dot{V} \leq 0$ provided $S_H > \overset{o}{S}_H$, $I_H > \overset{o}{I}_H$, $I_V > \overset{o}{I}_V$ and $S_V > \overset{o}{S}_v$

It is also important to note that $\dot{V} = 0$ only at disease-free equilibrium point, E^o

4. Numerical Experiment

Some numerical experiments are performed on our model with two main strategies considered for controlling the infectious disease, malaria:

a reduction in the number of infected humans through treatment and

a reduction in the number of susceptible humans through vaccination.

3.1. Graphical Representation of Results

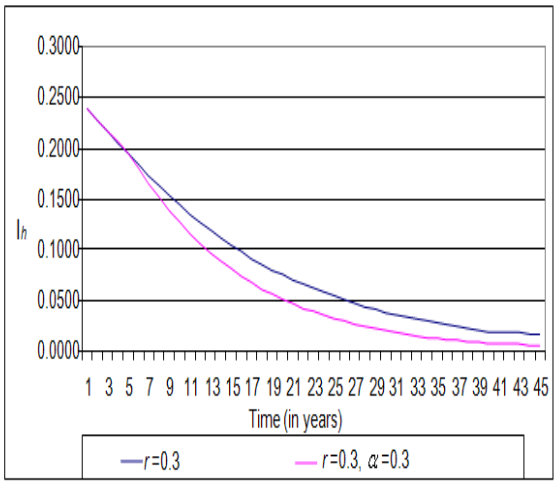

Figure 4.1.1*. Graph showing the comparison between the dynamics of the disease between the original Tumwiine et al. (2007) model with a treatment rate of 0.3 and the modified model with same treatment rate along with a vaccination rate of 0.3, on the infected human population, corresponding to table 1 in the appendix.*

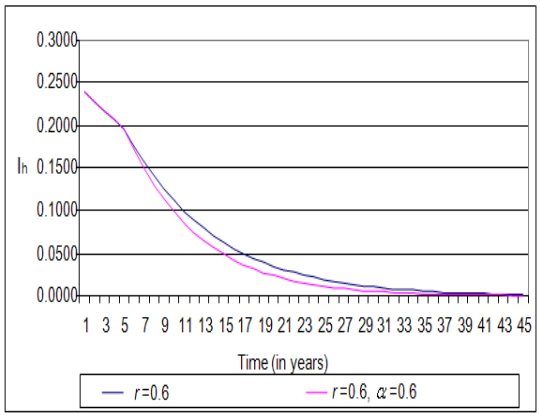

Figure 4.1.2. *Graph showing the comparison between the dynamics of the disease between the original Tumwiine et al. (2007) model with a treatment rate of 0.6 and the modified model with same treatment rate along with a vaccination rate of 0.6, on the infected human population, corresponding to table 2 in the appendix.*

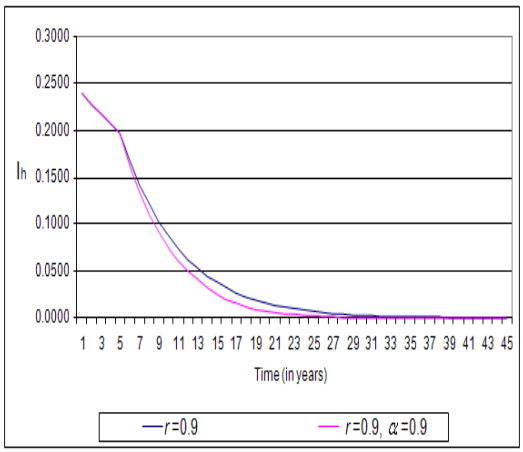

Figure 4.1.3. *Graph showing the comparison between the dynamics of the disease between the original Tumwiine et al. (2007) model with a treatment rate of 0.9 and the modified model with same treatment rate along with a vaccination rate of 0.9, on the infected human population, corresponding to table 3 in the appendix.*

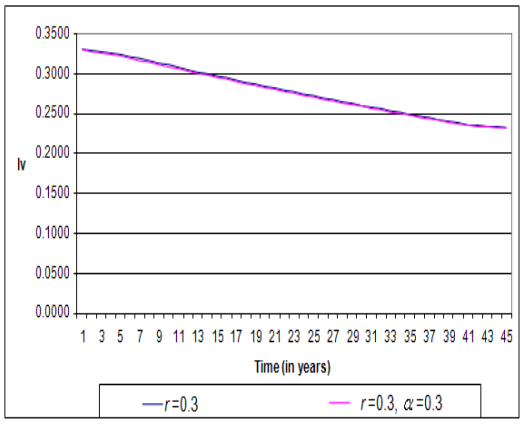

Figure4.1.4. *Graph showing the comparison between the dynamics of the disease between the original Tumwiine et al. (2007) model with a treatment rate of 0.3 and the modified model with same treatment rate along with a vaccination rate of 0.3, on the infected vector population, corresponding to table 4 in the appendix.*

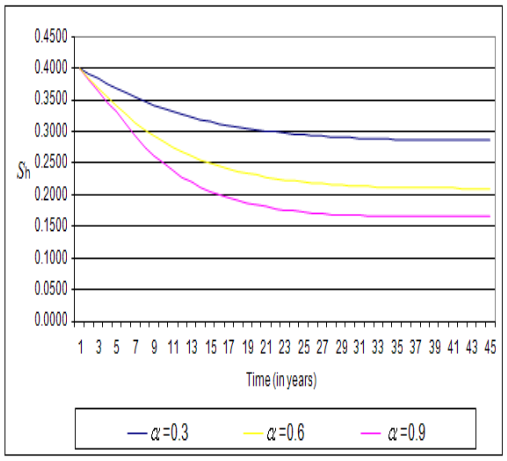

Figure 4.1.5. *Graph showing the dynamics of the disease on the modified model with a vaccination rate of 0.3, 0.6 and 0.9 on the susceptible human population, corresponding to table 5 in the appendix.*

4.2. Experiment One

Here the dynamics of the disease is compared between the old model and the modified. The experiment is carried out to establish the fact that a combination of both treatment and vaccination reduces the infectious population much more than applying treatment alone, as it was, in the result obtained from the old model.

4.3. Experiment Two

In this experiment, the dynamics of the disease of the modified model under the treatment and vaccination rate of 0.6 is carried out and compared with the old model of treatment rate of 0.6. We observed that a combination of the control measures causes a further decline in the infectious population from 0.24 to 0.008 through 0.0984 and 0.0101

4.4. Experiment Three

In this experiment, the dynamics of the disease of the modified model under the treatment and vaccination rate of 0.9 is carried out and compared with the old model with just treatment rate of 0.9. The result in this experiment shows that eradication is possible provided that both control measure rates are maintained.

4.5. Experiment Four

In this experiment, the dynamics of the disease on infected vector of the modified model under the treatment and vaccination rate of 0.3 is carried out and compared with the old model of treatment rate of 0.3. The infected vector population drops from 0.330 to 0.2330 and 0.2320. This will mean that less infectious vector population will be available for susceptible human to become infectious. Furthermore, the existence of mosquitoes will not necessarily increase the rate of malaria infection. There are many places in the world where mosquitoes abound but have not yet recorded malaria cases. Such places include Cape Town in South Africa, Maryland in USA, Kyoto in Japan, etc.

4.6. Experiment Five

In this experiment, we examine the effect of increasing vaccination rate from 30% through to 90%, we observed that the susceptible human population drop from 0.4000 to 0.1654 through 0.2102. Since the susceptible human population will not much be available, it makes it difficult for infectious mosquitoes to cause infections on human population. This in the long run should result into a malaria-free society.

4.7. Discussion of Results

The result from experiment one shows that in the absence of vaccination, eradication of the disease cannot be achieved so fast compared with combining vaccination along with treatment, as in the case in experiment two and three. The result for the infectious human population in experiment three carried out under a combined treatment and vaccination rate of 0.9 declines faster, thus resulting in a malaria-free society.

5. Summary, Conclusion, and Recommendation

5.1. Summary

The Tumwiine *et al*. (2007) mathematical model for the dynamics of malaria within human host and mosquito vectors was modified by adding a vaccination parameter. The model was analyzed in terms of actual population. The stability of the equilibrium point obtained were analyzed and found to be locally asymptotically stable. The effect of vaccination on the susceptible human class of the modified SIR host and SI vector model was considered. It was observed that, gradually increasing the vaccination rate alone reduces the number of susceptible human population against possible re-infection, thus in the long run decrease the number of infectious human population gradually to a barest minimal level. Numerical experiments carried out on the modified model clearly shows that, with a proper combination of treatment and a concerted effort aimed at prevention, malaria can be eliminated.

5.2. Conclusion

This study modified a model of malaria formulated by Tumwiine *et al*. (2007) by including a vaccination parameter, α . Analytical study was carried out on both models using the method of linearized stability and the results showed that the disease-free equilibrium points are locally asymptotically stable for both models. The results of numerical experiments carried out on both models also revealed that eradication is possible if a combination of both treatment and vaccination rate are maintained at least 0.65 level.

Recommendations

In consideration of the findings of this study as well as the incidental observations, we recommend that a combination of treatment and vaccination rates should be maintained at 0.65 level in order to eradicate malaria in the population.

Finally, it should be possible to validate this model by applying it to a smaller population, and then to a larger portion of any country. This will allow us to make informed decisions about the level of control strategies, "vaccination", that provide the most effective way of eradicating malaria.

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