MATHEMATICAL MODELING OF MALARIA DISEASE TRANSMISSION AND CONTROL

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ABSTRACT

Malaria is one of the diseases around the globe that have the ability to disorientate one's health, and is caused by a bite of a small vector-mosquito. In this paper, the classical model was modified by including the treated class. This new class helps to understand better how effective the control of the disease.The model is a four deterministic model expressed as a system of ordinary differential equations. The stability analysis of the disease free equilibrium point was conducted. It is also shown that the disease free equilibrium point is locally asymptotically stable if $R_0 < 1$and the system is unstable if $R_0 > 1$. Some numerical simulations are also given to explain the analytical results.

Keywords: *Reproduction Number, Red blood cell, Equilibrium Points, Malaria parasite, Insecticide treated net*

INTRODUCTION

Arum (2016), defines a model to be an image of a sector of reality created in order to satisfy a given purpose or to accomplish a given task. The goal is just to give a representation of some aspects of the real system. A model may help to explain a system and to study the effects of different components and to make predictions about behavior. The actual model is a set of functions that describes the relationship among different variables and quantities.

Malaria is a vector borne disease that has affected mankind since before recorded history. It is one of the most life threatening disease and a leading cause of mortality in the tropical regions in the world (King *et al.*, 2012), this is as a result of the rate at which the populace are ignorant of the mode in which the disease is being transmitted and it's preventive measures. In 2006, there were almost 250 million cases of malaria, causing nearly one million deaths (Roll Back Malaria, 2010). The disease is caused by parasites of the species plasmodium, the malaria parasites enters a human when an infectious mosquito bites a person.

About 40% of the world's total population live in areas where malaria is an endemic disease and as global warming occur that percentage will increase as mosquitoes range will increase due to increasing rainfall. The number of malaria victims are growing rapidly every year due to increasing resistance of the parasite to the drugs that have been used in the past to treat malaria and also due to the mosquitoes increasing resistance to the pesticides that once killed it (Chitnis, *et al.*, 2006). The World Health Organization has an estimate of 350-500 million victims infected annually, and killing an estimated 1,000,000 people per year worldwide. In Africa alone, as many as one million children die annually from malaria before they reach the age of 5. The malaria parasite kills a child every 30 seconds.

The vector that spreads the parasite is the mosquito, but not just any mosquito, as only 30-50 species of the more than 430 species of mosquito spread the parasite (National Centre for Infectious Disease (2006). The major vectors for spreading the parasite are the mosquitoes: *Anopheles gambiae*, *Anopheles arabiensis*and, *Anophelesfunestus*. The vector, like the parasite, is also growing a resistance to the insecticides that have been used in the past to treat mosquito breeding grounds. The parasite itself is a protozoan of the genus *Plasmodium* specifically: *Plasmodium falciparum*, *Plasmodium vivax*, *Plasmodium malariae*or *Plasmodium ovale* (World Health Organization, 2009). *Plasmodium falciparum* is the most deadly of the four types of malaria. Infection with *Plasmodium falciparum*is a medical emergency, about 2% of the people so infected die because of delayed treatment. Infection with malaria parasites may

result in a wide variety of symptoms, ranging from absent or very mild symptoms to severe and even death. All clinical symptoms are caused by the asexual erthrocytic or blood stage parasites. When the parasite develops in the erythrocyte, numerous known and unknown waste substances such as hemozoin pigment and other toxic factors accumulate in the infected red blood cell. These are dumped into the bloodstream when the infected cells lysis and release invasive merozoites. The hemozoin and other toxic factors such as glucose phosphate isomerase stimulates macrophages and other cells to produce cytokines and other soluble factors which act to produce fever and rigors and probably influence other severe pathophysiology associated with malaria. Following the infective bite by the Anopheles mosquito, a period of time the "incubation period" goes by before the first symptoms appear (Roll Back Malaria, 2010). The incubation period in most cases varies from 7 to 30 days. The shorter periods are observed most frequently with Plasmodium malariae. Anti-malarial drugs taken by travelers and self-medication can delay the appearance of malaria symptoms by weeks or months, long after the traveler has left the malaria endemic area. Such long delays between exposure and development of symptoms can result in misdiagnosis or delayed diagnosis. The classical (but rarely observed) malaria attack lasts 6-10 hours. It consists of:

1. A cold stage: Sensation of cold, shivering.
2. A hot stage: Fever, headache, vomiting,seizure in young children.
3. A sweating stage: sweat, return to normal temperature, tiredness.

More commonly, the patient presents a combination of the following symptoms: fever, chill, sweat, headache, nausea and vomiting, body ache and general malaise. In countries, where cases of malaria are infrequent, these symptoms may be attributed to influenza, a cold, or other common infections, especially if malaria is not suspected. Conversely, in countries where malaria is frequent, residents often recognize the symptoms as malaria and treat themselves without seeking diagnostic confirmation and using presumptive treatment (Global Health Division of Parasitic Disease and Malaria, 2015). Physical findings may include: elevated temperature, perspiration, weakness, enlarged spleen, mild jaundice, enlargement of the liver etc. Severe malaria occurs when infections are complicated by serious organ failures or abnormalities in the patient's blood or metabolism. The manifestations of severe malaria include:

i. celebral malaria, with abnormal behavior, impairment of consciousness, seizures coma or other neurologic abnormalities.
ii. Severe anaemia due to hemolysis
iii. Hemoglobinuria (hemoglobin in the urine)
iv. Acute kidney failure
v. Metabolic acidiosis (excessive acidity in the blood and tissue fluids).
vi. Low blood pressure caused by cardiovascular collapse. Severe malaria is a medical emergency and should be treated urgently and aggressively. Wu and Xiao (2013) further explained that malaria can be transmitted in three different ways. The most common way is through the bite of anopheles mosquitoes. It could also be by spread, through the transfusion of infected blood and sharing needle with infected person. Malaria can be controlled through the use of mosquito treated nets, use of anti-malarial drugs, maintaining high level of sanitation through tiding of our surroundings and fumigation of mosquito breeding reservoirs. Malaria control is challenging due to many factors namely; the complexity of the disease control process, the cost of the control program and resistance of the parasite to anti-malaria drugs and vectors to insecticides. The environmental condition in the tropics is a prime factor for malaria being endemic. The moderate to warm temperatures, high humidity water bodies allow mosquito 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, kidney and other organs. Clinical symptoms such as fever, pain, chill, and sweat may develop a few days after infected mosquito bites. Since malaria increases morbidity and mortality rate, 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 all the disease that faced mankind. Poverty also means that people cannot be able to afford simple protection of a mosquito net or even a screen for their windows. A favorite hiding place for the Anopheles is in a dark moist room.

Malaria has for many years been considered as a global issue, and many epidemiologists and other scientists invested their effort in learning the dynamics of malaria, how to control malaria transmission. From interactions, with those scientists, mathematicians have developed a significant and effective tool, namely mathematical models of malaria, giving an insight into the interaction between the host and vector population, the dynamics of malaria, how to control malaria transmission, and eventually how to eradicate it. This therefore, motivated the World Health Organization to declare malaria control a global priority in 1989 (Laith *et al.*, 2011).

Insecticide treated nets, particularly the long – lasting insecticidal nets, are the preferred tools for reducing malaria transmitting bites and alleviating disease burden. Mosquito nets treated with insecticides known as insecticide treated nets (ITNs) or bed nets – were developed in the 1980s for malaria prevention. ITNs are estimated to be twice as effective as untreated nets (Hall, 2006) and offer greater than 70% protection compared with no net (Bachou *et al.*, 2006). These nets are dip – treated using a synthetic parathyroid insecticide such as deltamethrin or permethrin which will double the protection over a non –treated net by killing and repelling mosquitoes. For most maximum effectiveness, ITNs should be re-impregnated every six months. The distribution of ITNs has been shown to be an extremely effective means for malaria prevention, and it is also one of the most cost–effective methods of prevention. ITNs offer protection to individuals households in two ways; firstly, it kills adult mosquitoes infected with the malaria parasite directly when in contact, which increase their mortality rate and can therefore decrease the frequency in which a person in the community is bitten by an infected mosquito. Secondly, killing mosquitoes before maturation of the malaria parasite (*Plasmodium falciparum*), ITNs can reduce the number of encounters of infected mosquitoes with humans (Killen and Smith, 2007).

Following numerous successful application of ITNs, The WHO's roll back malaria program recently set the target of 80% bed nets coverage in malaria endemic areas (World Health Organization, 2008). In addition, malaria can also be controlled by maintaining high level of sanitation by measures such as building of drainage patterns tiding of our surrounding and fumigation of mosquito breeding reservoirs. As malaria remains a major public health problem as earlier stated, understanding its history is the key to its control and eradication.

Human malaria likely originated in Africa and has co-evolved along with its hosts, mosquitoes and non human primates. The first evidence of malaria parasites was found in mosquitoes preserved in amber from palaeogene period that are approximately 30 million years old (Poinar. 2005). Malaria may have been a human pathogen for the entire history of the species.

Mathematical modeling of malaria began in 1911 with Ross's model, and major extensions are described in Macdonald book (Ngwa and Shu, 2000). The first models were two-dimensional with one variable representing human and the other representing mosquitoes. He showed that reducing the number of mosquitoes have little effect on epidemiology of malaria in areas of intense transmission. An important addition to the malaria models was the inclusion of acquired immunity proposed by (Labadin *et al.*, 2009). This addition of the model will help to further explain the spread of malaria. Ngwa and Shu (2000) on their part proposed a mathematical model for endemic malaria with variable human and mosquito population. Many researchers have similar mathematical models including the model proposed by (Tumwiine *et al.*, 2007). In the model, a Susceptible-Infective-Recovered-Susceptible class (SIRS) model for human population and a Susceptible Infected class (SI) model for the mosquito vector population were formulated.

FORMULATION OF THE MODEL

The model formulation will begin by introducing the model by Ross Ronald which is the motivation for this study. First, the building blocks of malaria transmission will be presented; along side with the assumptions, variables and parameters of the model.

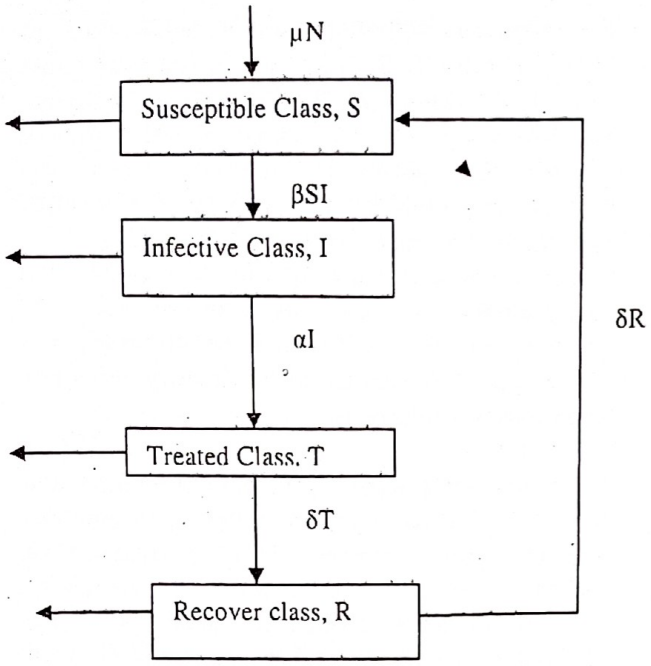

Fig 1: Building block of human population transmission of Malaria disease

Assumptions of the Modified Model

The following are the assumptions to be adopted from the formulated Model;

i. The development of malaria disease starts when the infectious female (Anopheles) mosquito bites the human host.
ii. The bite of an infected female mosquito causes infected human host.
iii. Mosquitoes bite human hosts randomly (independent of their infective status).
iv. Recovered human hosts have temporary immunity that can be lost and are again susceptible to re-infection.
v. Birth rate is equal to death rate
vi. All newborns are susceptible to infection and there is no vertical transmission.
vii. Mosquitoes never recover from infection, as it is regulated by mortality of its individuals.
viii. Infected humans after feeling sickly goes to clinic for medical treatment.
ix. Total human population is closed.
x. Total population size N = S(t) + I(t) + T(t) + R(t)

Definitions of Variables and Parameters of the Formulated Model

μ = birth rate
μ = death rate
N = total human population
β = contact rate (infectivity)
δ = recovery rate
α = rate at which infected are treated
δ = rate at which the recovered class become susceptible
S = su ... 'e class at time, t
I = inf ass at time, t
T = tre: is of persons at time, t
R = rec class at time, t

Equations of the New Model

$$S^1 = \mu N - \beta SI - \mu s + \delta R$$
$$I^1 = \beta SI - \alpha I - \mu I$$
$$T^1 = \alpha I - \delta T - \mu T$$
$$R^1 = \delta T - \delta R - \mu R$$

Since the system is closed, this implies that;

$$N^1 = \mu N - \mu(S + I + T + R) = \mu N - \mu N = 0$$

Therefore, the reduced model is now (SIT) and system becomes:

$$S^1 = \mu N + \delta R - \mu S - \beta SI \quad 2.1$$

$$I^1 = \beta SI - \alpha I - \mu I \quad 2.2$$

$$T^1 = \alpha I - \delta T - \mu T \quad 2.3$$

The non-negative initial conditions for the modified model are $S(0) = S_0, I(0) = I_0, T(0) = T_0, R(0) = R_0$ and all the parameters of the modified model are assumed to be non-negative.

MODEL ANALYSIS

In this section, the new model which is a closed systemat an equilibrium state the system is written as

$\frac{dS}{dt} = \frac{dI}{dt} = \frac{dT}{dt} = 0.$

Equating the equation (2.1) – (2.3) to zero, we have the following equations:

$$\frac{dS}{dt} = \mu N + \delta R - \mu S - \beta SI = 0 \quad 3.1$$

$$\frac{dI}{dt} = \beta SI - \alpha I - \mu I = 0 \quad 3.2$$

$$\frac{dT}{dt} = \alpha I - \delta T - \mu T = 0$$

3.3

Determination of the Basic Reproduction Number

An important notion in epidemiological models is the basic reproduction number, usually denoted by R_0. This number can be understood as the average number of secondary infectious infected by an infective individual during whose whole cause of disease in the case that all members of the population are susceptible. It is

an important parameter that predicts whether an infection will spread through the population or not. The basic reproduction number, R_0, could be seen as a point where there is no change in the infective class.

That is,

$I^1 = 0$

$$\beta SI - \alpha I - \mu I = 0$$
$$\beta SI - I(\alpha + \mu) = 0$$
$$\beta SI = I(\alpha + \mu)$$

$\frac{\beta S}{\alpha+\mu} = 1$, but N = S

Hence, $R_0 = \frac{\beta N}{\alpha+\mu}$ 3.4

Theorem 3.1: Routh Hurwitz Criteria

Given a system of equation of n^{th} order, the characteristics polynomial can be written in the general form as:

$\lambda^n + a_1\lambda^{n-1} + \cdots + a_n = 0$ 3.5

where the coefficients a_i, $i = 0,1,\ldots,n$ are all real and $a_n \neq 0$. Then a necessary and sufficient condition for all the characteristics roots of (3.13) to have negative real parts is that $D_k > 0$ for $k = 0,1,\ldots,n$.

where,

$$D_1 = a_1,\ D_2 = \begin{vmatrix} a_1 & a_3 \\ 1 & a_2 \end{vmatrix}, \cdots, D_k = \begin{vmatrix} a_1 & a_3 & \cdots & a_{2k-1} \\ 1 & a_2 & \vdots & a_{2k-2} \\ \vdots & \vdots & \vdots & \vdots \\ 0 & 0 & \cdots & a_k \end{vmatrix} \text{ with } a_j = 0 \text{ for } j > k.$$

Existence and stability of Disease free Equilibrium

The Disease Free Equilibrium Point, E_0 is the steady state of the model in the absence of infection, where E_0 = (N,0,0). This is obtained from the system (3.1)-(3.3) by setting the right hand side equal to zero. The local stability of E_0 is then determined from the signs of the eigen values of the Jacobian matrix. The Jacobian matrix at the disease free equilibrium is given by:

$$J_{E_0} = \begin{bmatrix} \mu N + \delta R - \mu & \mu N + \delta R & 0 \\ 0 & -\alpha - \mu & 0 \\ 0 & \alpha & -\delta - \mu \end{bmatrix}$$

The characteristics equation of this matrix is given by

where I is the square identity matrix of order 3. The equation thus becomes

$$-\lambda^3 + \lambda^2(\mu N + \delta R - 3\mu - \alpha - \delta - \mu^2) - \lambda(2\mu^2 N + \mu N\delta + \delta R\alpha + 2\delta R\mu + \delta^2 R - 2\alpha\mu - 3 - \mu^2 - 2\mu\delta) - (\mu N\alpha\delta + \mu^2 N\alpha + \mu N\alpha + \mu^2 N\delta + \mu^3 N + \delta^2\alpha R + \alpha\delta R\mu + \delta^2 R\mu + \delta R\mu^2 - \mu\alpha\delta - \alpha\mu^2 - \mu^2\delta - \mu^3) = 0$$ 3.6

Let

$$a_1 = \mu N + \delta R - 3\mu - \alpha - \delta - \mu^2$$
$$a_2 = 2\mu^2 N + \mu N\delta + \delta R\alpha + 2\delta R\mu + \delta^2 R - 2\alpha\mu - 3\mu^2 - 2\mu\delta - \alpha\delta$$
$$a_3 = \mu N\alpha\delta + \mu^2 N\alpha + \mu N\alpha + \mu^2 N\delta + \mu^3 N + \delta^2\alpha R + \delta R\alpha\mu + \delta^2 R\mu + \delta R\mu^2 - \mu\alpha\delta - \alpha\mu^2 - \mu^2\delta - \mu^3$$ 3.7

We employ the Routh Hurwitz criterion which states that all roots of the polynomial (characteristics polynomial) have negative real parts if and only if the coefficients a_i are positive and matrices $D_i > 0$ for i = 0, 1,2,3. It is seen from above that $a_1 < 0$, $a_2 > 0$, $a_3 > 0$.

Moreover, if $R_0 < 1$, it follows from (3.6), $a_0 = -1$. Also, the Hurwitz matrices for the characteristics polynomial are found to be positive. That is,

$H_1 = A_3 > 0$, $H_2 = \begin{vmatrix} a_1 a_3 \\ 1\ a_2 \end{vmatrix} > 0$, $H_3 = \begin{vmatrix} a_1 a_3 a_1 \\ 1\ a_2 a_3 \\ 0\ 0\ a_3 \end{vmatrix} > 0$

Therefore, all the eigenvalues of the Jacobian matrix $J(E_0)$ have negative real parts when $R_0 < 1$ thus, the disease-free equilibrium point is locally asymptotically stable. However, when $R_0 > 1$, we see that $a_0 < 0$ and by Descartes rule of signs (Polyanin and Manzhirov, 2007). There is exactly one sign change in the sequence a_3, a_2,a_1, a_0 of coefficients of the polynomial (3.6). So, there is one eigen value with positive real part and the disease- free equilibrium point is unstable.

Existence of the Endemic Equilibrium point

The endemic equilibrium point is a positive steady state solution where the disease persists in the population. The following are the conditions to be satisfied:

$$I \neq 0, \quad S^1 = 0, \quad T^1 = 0$$

From equation (3.2)

$$I^1 = \beta SI - \alpha I - \mu I = 0$$
$$I(\beta s - \alpha - \mu) = 0$$

$S = \frac{\alpha+\mu}{\beta}$ 3.8

Substituting the values of S in equation (3.1)

$$S^1 = \mu N + \delta R - \mu\left(\frac{\alpha+\mu}{\beta}\right) - \beta\left(\frac{\alpha+\mu}{\beta}\right)I = 0$$

$$\mu N + \delta R - \frac{\mu\alpha + \mu^2}{\beta} - (\alpha + \mu)I = 0$$

$$\beta\mu N + \beta\delta R - (\mu\alpha + \mu^2) - \beta(\alpha + \mu)I = 0$$

$$\beta\mu N + \beta\delta R - \mu\alpha - \mu^2 = \beta(\alpha +)I$$

$$I = \frac{\beta\mu N + \beta\delta R - \mu\alpha - \mu^2}{\beta(\alpha+\mu)} \quad 3.9$$

Substituting the value of I in equation (3.3)

$$T^1 = \alpha I - \delta T - \mu T = 0$$

$$\alpha\left(\frac{\beta\mu N + \beta\delta R - \mu\alpha - \mu^2}{\beta(\alpha + \mu)}\right) - T(\delta + \mu)$$

$$\alpha(\beta\mu N + \beta\delta R - \mu\alpha - \mu^2) - T(\alpha + \mu)\beta(\delta + \mu) = 0$$

$$T = \frac{\alpha\beta\mu N + \alpha\beta\delta R - \mu\alpha^2 - \mu^2\alpha}{\beta(\alpha+\mu)(\delta+\mu)} \quad 3.10$$

The Endemic Equilibrium point, $E_2 = (\frac{\alpha+\mu}{\beta}, \frac{\beta\mu N+\beta\delta R-\mu\alpha-\mu^2}{\beta(\alpha+\mu)}, \frac{\alpha\beta\mu N+\alpha\beta\delta R-\mu\alpha^2-\mu^2\alpha}{\beta(\alpha+\mu)(\delta+\mu)})$

NUMERICAL RESULTS AND DISCUSSION

In this section, the numerical simulations examined the effect of different combinations of treatment and preventions on the transmission of the disease using Matlab. The main strategy considered for controlling malaria is the reduction in the number of infected humans through a program preventive measure.

TABLE 1: Table of Parameter values for the Numerical Experiments

Parameters \ Experiment	1	2	3	4	5
μ	0.000096274	0.000096274	0.000096274	0.000096274	0.000096274
δ	0	0	0.2	0.5	0.8
α	0	0.045	0.225	0.49	0.85
I	0	0.09	0.09	0.09	0.09
S	0.91	0.91	0.91	0.91	0.91

As shown on Table 1, the numerical experiments are meant to study the following cases.

1. The situation whereby there is no disease (i.e. no infection)
2. The effect of absence of treated mosquito nets (no control) on the infected human population.
3. The effect of less use of treated mosquito nets (weak control) on the infected human population
4. The effect of an effective use of treated mosquito nets (strong control) on the infected human population
5. The effect of very effective use of treated mosquito nets (very strong control) on the infected human population

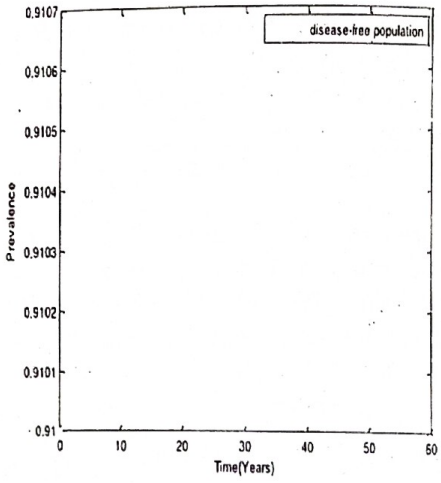

Figure 2: Graph of susceptible humans against time in a disease- free population. Parameter values are as defined on Table 1.

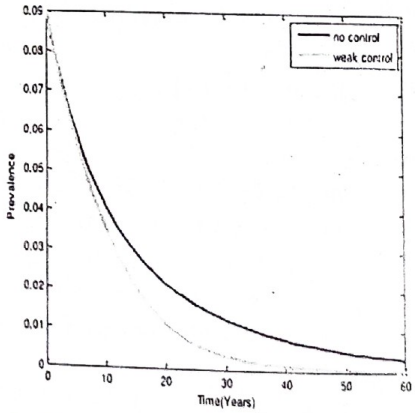

Figure 3: Graphs of infected humans against time in the case of no control ($\delta = 0$) and weak control ($\delta = 0.2$). Parameter values are as defined on Table 1 (Experiment 2 and 3)

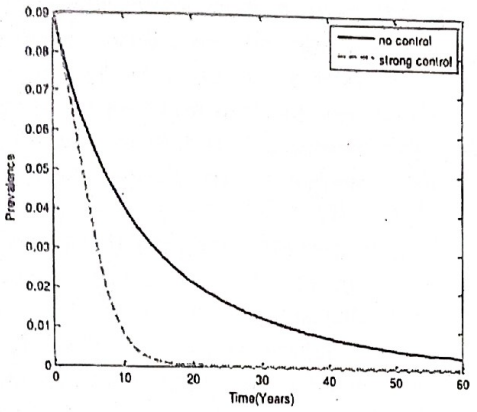

Figure 4: Graphs of infected humans against time in the case of no control ($\delta = 0$) and strong control ($\delta = 0.5$). Parameter values are as defined on Table 1 (Experiments 2 and 4)

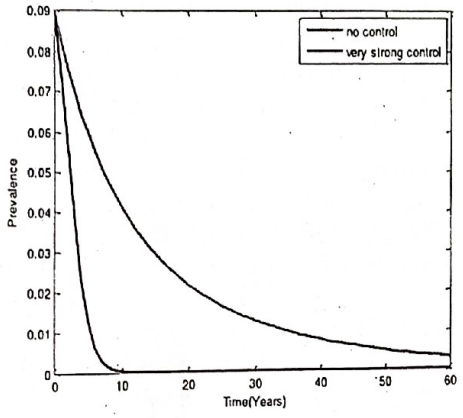

Figure 5: Graphs of infected humans against time in the case of no control ($\delta = 0$)and very strong control ($\delta = 0.8$). Parameter values are as defined on Table 1 (Experiments 2 and 5)

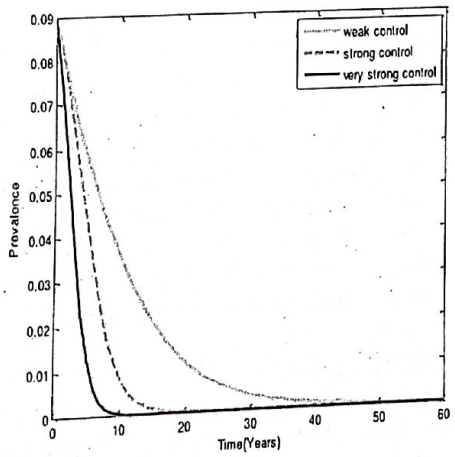

Figure 6: Graphs of infected humans against time for weak control ($\delta = 0.2$), strong control ($\delta = 0.5$), and very strong control ($\delta = 0.8$).

Discussion of the Numerical Results of the new Model Equations.

In our model, the following results were obtained.

Experiment One

Here, we investigated the situation where there was no infection (i.e. there is no transmission of the disease), Figure (2) shows that the susceptible human population grows rapidly in accordance with the population of a linear model of population growth (Rubinow, 1975).

Experiment Two

Here, we studied the effect of the disease on the infected human population compartments when control was not introduced. The graph with red colour in Figures 3-5 illustrates a steady decline in the number of infected humans as a result of the temporary immunity as proposed by (Tumwiine *et al.*, 2007) model. However, this does not eradicate the disease. This graph is being compared with the rest of the graphs as seen in Figures 3-5 (see experiments 2-5)

Experiment Three

In this experiment we investigated the effect of weak control (i.e. less use of ITNs and control strategies) on the dynamics of the infection. The graph with green colour shows weak control with infected human's prevalence decreasing more with high tendency of disease eradication (see Figure 3).

Experiment Four

In this case, we studied the effect of the effective use of treated mosquito nets on the transmission dynamics of the disease. The graph coloured purple with broken points in figure 4demonstrates a sharper decline in the number of infected humans eliminating the disease within a period of twenty years.

Experiment Five

Here, we investigated the effect of very effective use of treated mosquito nets and control measures on the dynamics of infection. The graph coloured blue shows that the number of infected human's prevalence decreases more faster bringing the infection to total control/eradication in a period of 10 years (see Figure 5). This clearly shows a more desiring and effective method than those in experiments 3 and 4 (see Figure 6).

Control Strategies

Measures to prevent or decrease the prevalence of malaria, are being used with a degree of success in some part of the globe. Some of these methods used are;

1. Larval Control: This strategy includes methods such as the destruction of breeding sites which aim to reduce the number of mosquitoes.
2. Indoor Residual Spraying (IRS): Spraying reduces mosquito longevity (perhaps also fertility). This strategy is also likely to kill mosquitoes that rest within the house after feeding and thus increases the death rate of infective mosquitoes.
3. Insecticide-Treated bed Nets (ITN): Roll Back Malaria has been promoting the use of ITN in many countries of Africa where the disease is endemic. This is done in order to reduce the transmission of malaria and has succeeded in doing so in many regions. Preventing mosquito-human contacts should decrease the number of bites per mosquito. This would translate into the mosquito biting other animals or not biting at all. Reducing the number of blood meal that each female mosquito receives would also lower the mosquito birth rate and perhaps reduce the number of mosquitoes. This seems to be the most effective control strategy in reducing disease transmission.
4. Prompt and Effective Case Management (PECM): This strategy involves the quick identification and treatment of malaria cases. Although it may seem obvious, PECM is not always possible in many places because of poor health infrastructure and a lack of resources. This strategy is more commonly practiced in areas of low transmission because these areas usually have more resources and malaria infection is easier. Quick treatment is doubly effective because it directly reduces the suffering and lack of productivity due to malaria and it reduces the transmission of infection to mosquitoes.
5. Gametocytocidal Drugs: These drugs kill gametocytes in humans, reducing human-to-mosquito disease transmission. This is useful in areas like South East Asia where there is low transmission and most sick people can be reached. This would not be useful in many parts of Africa where there is high level of transmission and there are not sufficient resources to allow the drugs to be dispensed to all people with parasite loads.
6. Intermittent Prophylactic Treatment for Infants (IPTI): As our model shows no distinction among infant, adults and pregnant women, we can only model this strategy as a general reduction in the probability of transmission of infectious mosquito to a susceptible human, β. The treatment also probably causes a slight increase in the human recovery rate, δ, as it may result in some infected people beginning treatment.

Therefore, all these control strategies are an effective way of controlling most of the parameters which are involved in our model. In determining how best malaria, and reduce malaria mortality, it is necessary to know the relative importance of the different factors responsible for its transmission and prevalence. The fraction of infected humans is especially important because it represents the people who suffer the most and are directly related to the total number of malarial deaths. The value of the basic reproduction number R_0 with an increasing mosquito to human disease transmission rate, β, leads to an increase in malaria deaths.

We would like to classify parameters of our model into different categories depending on whether they are important in disease transmission and malaria outbreaks, and whether we have control of the parameter through the intervention strategies. In the first category, we include parameters that are important for disease transmission and spread, that we have control of the human to mosquito to contact rate, β, is controlled by gametocytocidal drugs, ITNs, IPTI control strategy. The second category is an important human demographic parameter, the natural birth rate of the human population, μ, which one cannot be easily control.

CONCLUSION

In this research, the model by Ross (1911) was modified by incorporating the treated class and studied the new version to investigate the effects and preventive measures of the various parameter used. The model was derived with the aid of the model flow diagram, parameters and assumptions. The disease free equilibrium point

and the endemic equilibrium point was obtained, the stability analysis of the first was also carried out.

According to the results of the model, if the population who are infected are being properly treated and preventive measures which was previously stated are taken effectively, it will reduce R_0, below one leading to the disease eradication.

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