Harvard Research and Publications International

June 30, 2020 Vol. 12 No. 2 ISSN: 0081 – 3998

JOURNAL OF AFRICAN SUSTAINABLE DEVELOPMENT

MATHEMATICAL MODEL FOR CONTROLLING THE SPREAD OF MALARIA FEVER USING THERAPY/VACCINATION

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Abstract

A mathematical model for transmission dynamics and qualitative analysis of malaria fever was developed incorporating preventive therapy. The therapy was assumed to be given to human population only. The Disease Free Equilibrium States of the model was evaluated and analyzed for stability and was found to be asymptotically stable. The Basic reproduction number was also

Keywords:
Mathematical model, Malaria, Therapy/vaccination, Analytical Solution and Stability Analysis.

INTRODUCTION

Malaria is caused by protozoan parasite of the genus plasmodium. The parasite is spread from person to person by a mosquito, of the genus Anopheles, each time the septic insect takes a blood meal. Symptoms in an infected human includes bout of fever and anemia. On average, the incubation period of P. falciparum is about twelve days in humans and about ten days in mosquitoes. The literature on the mathematical models for infectious disease is vast.

It is clear that the assumption of persistent population size is epidemiological models, which is relatively valid when studying disease of short period with limited effect on principles, may no

evaluated. The model equations were solved analytically using Homotopy Perturbation Method (HPM) and graphical profiles of each compartment was generated from the results using maple mathematical software. The results shows that Vaccination is likely to reduce the number of infected individuals as reduction in vaccination parameter (θ) leads to high infection and its increase leads to low infection.

longer be valid when dealing with endemic disease such as malaria. In such diseases, the effect of changes in population size and disease include morality are far from insignificant and in fact can have a crucial influence on the dynamics of the disease. Some malaria endemic population in, say, tropical Africa, have a human population growth rate of about two percent. Also the continuous contact between mosquito and human populations necessarily introduces a high variability on the mosquito population for which an assumption of constant population may not be valid. For the sake of mathematical obedience in the analysis of mathematical models for malaria transmission with variable population densities, information about the infested mosquitoes is often traded for information about all the mosquitoes and infected people through the pseudo equilibrium hypothesis

The term malaria is resulting from the Italian 'malaria', which means 'bad air', from the early connotation of the disease with swampy areas. Towards the end of the 19th century, Charles Louis Alphonse Laveran, a French army specialist, noticed parasites in the blood of a patient misery from malaria, and Dr. Ronald Ross, a British medical general in Hyderabad, India, revealed that mosquitoes conveyed malaria. The Italian professor Giovanni Battista Grassi afterward showed that human malaria could only be conveyed by *Anopheles* mosquitoes. Malaria affects a large number of countries and it has been described that the occurrence of the disease in 2004 was between 350 and 500 million cases. Over two billion people, signifying more than 40% of the world's population, are at risk of constricting malaria, and the number of malaria deaths worldwide has been estimated at 1.1–1.3 million per annum in World Health Organization (WHO) reports 1999–2004. Malaria has a broad spreading in both the subtropics and tropics, with many areas of the tropics endemic for the disease. The countries of sub-Saharan Africa account for the popular of all malaria cases, with the remainder mostly gathered in India, Brazil, Afghanistan, Sri Lanka, Thailand, Indonesia, Vietnam, Cambodia, and China . Malaria is estimated to cost Africa more than $12 billion annually and it accounts for about 25% of all deaths in children under the age of five on that continent. In many temperate areas, such as Western Europe and the USA, public health measures and economic development have been

successful in achieving near- or complete removal of the disease, other than cases introduced via worldwide travel. (World Health Organisation, 2013)

According to Chitins (2010), the mathematical modeling of malaria began in 1911with Ross, who was awarded with Nobel Prize for his work. HIS model was very simple and has been greatly prolonged during the years.

Kermack and McKendric (1997) came up with amended SIR model of epidemics. MacDonald (1957) improved the model of two dimensional model with one variable representing humans and one representing mosquitoes. An important delay of the model was proposed by Dietz, Molineux and Thomas who added the inclusion of immunity. Other extension that has been made is for example environmental dependence and drugs resistance. This phenomenal of incomplete immunity permitting transmission is known to exist for malaria and confuses disease control strategies as the reservoir of infection now include symptomatic infected individuals

In an effort to model malaria infection, Okosun and Makinde (2011) developed and formulate a malaria model. The result of their study show that the model subdivided human population in to sub population of susceptible individuals and also those exposed to malaria parasite, also individual with drug resistance symptoms(Id_h) . We noticed in their model that the individual with drugs resistance symptoms can be avoided in the model of malaria because the resistance might be as a result of another infection not necessarily malaria, and we are after malaria.

Francis *et-al* (2012) formulate a mathematical model of malaria In their work, they included exposed human (E_h) class and exposed mosquito (E_m) class which by definition and medically, exposed and susceptible means the same thing which one of them need to be removed to have standard model equations.

Folashade *et-al* (2012) developed a mathematical model of the impact of bed-net use on malaria prevalence. In their model, they concluded that models must include human behavior in order to provide realistic estimates of malaria dynamics. Where they also analyzed their model by considering transition dynamics of malaria infection in mosquito and human population and also investigate the impact of bed net. However, in their work, they only considered the susceptible classes for human and mosquito and also infected classes for both human and mosquito. The recovered classes were not considered which is very important because human being can recovered from an infection and become susceptible again.

Momoh *et-al* (2012) formulate a Mathematical Modeling of Malaria Transmission in North Senatorial Zone of Taraba State Nigeria . They used the SIR proposed by Kermack and McKendrick and data obtained from Essential Programme on Immunization (EPI) unit, F.M.C., Jalingo, Taraba state were used to analyze the rate of infection of malaria in the zone. From our analysis, we found out that the reproduction ratio. Based on the reproduction ratio, which is greater than 0, implies

that the force of malaria infection in Taraba North Senatorial Zone is high. The researchers also make recommendations for the reduction of malaria in the zone.
However, in their model, they only considered SIR Model of the human segment without considering the vector (mosquito) class. Also the recovered class of the human segment was not also considered.
Ndanusa *et-al* (2013) also developed and analysed a mathematical model on malaria dynamics using Ordinary differential equations. They follow the idea of Roberts and Heesterbeek and analysed the simple malaria model. Due to the nature of their model, they were not be able to put it in the form of SIR Model. In that case, they were not be able to section the models base on human and Vector Population techniques.
Furthermore, Abubakar *et al* (2013) proposes model for general infectious diseases dynamics which malaria cannot be left out and the analytical solution was obtained using Homotopy perturbation method. In their work, they only considered the human Population of a general infectious diseases without restricting to a particular infectious disease. Also the vector population was not considered
In this study, we present a mathematical model for the spread and control of malaria using therapy/vaccines

MATERIALS AND METHODS

Model Development

A mathematical model for transmission dynamics and qualitative analysis of malaria epidemiology was developed, improving on the existing models as explained in the literature review. The model is extended to include preventive therapy. The therapy is assumed to be given to human population only. The reason for this decision lies in the fact that malaria prevention is always administered to human being not mosquito.

Model Description

The model contains six (6) variables namely: susceptible human (S_h), infected human (I_h), recovered human (R_h), susceptible vector (S_v) (infected vector (I_v) and therapy class (V_h). Other parameters also includes death rate (μ), recruitment rate (Λ), transmission rate between (S_h) and (I_h) (λ_h) transmission rate between (I_h) and (R_h) (δ), transmission rate between (S_v) and (I_v) (λ_v), transmission rate from (V_h) to (S_h) (θ), transmission rate from (S_h) to (V_h) (ϕ) and natural death (μ_0).

Assumptions

The population of the Human susceptible class (S_h)increases by the recruitment of individuals into the population at the rate($\wedge_h P$), coming of Vaccinated individuals from class(V_h) at the rate (θ) and the coming in of individuals from the Malaria recovered class (R_h) at the rate r and the population decreases as susceptible human (S_h) moves in to infected human class (I_h) at the rate (λ_h) via interaction between infected vector and susceptible human, as susceptible human moves in to vaccinated class (V_h) at the rate (ϕ) and natural death (μ_h).

The population of infected Human (I_h) increases as individuals moves from (S_h) to infected human (I_h) via interaction (λ_h). It decreases as infected individuals moves to recovered class via treatment rate (δ) and also decreases as individuals dies due to infection as well as naturally ($\mu_h + \mu_0$).

The population of the recovered class increases as individuals moves from infected class to recovered class via treatment rate (δ).the population decreases as recovered individuals move back to susceptible due to lack of permanent immunity at rate (r) and also reduces as individuals can also dies naturally (μ_h)

The population of the Vaccinated class (V_h) increases by the recruitment of individuals into the population at the rate ($\wedge_h$ (1-P)) and also increases as susceptible individuals moves in to vaccinated class at the rate (ϕ). It also decreases as vaccinate individuals also moves back to the susceptible class at after receiving therapy at the rate (θ) and also decreases due to natural death (μ_h)

The pupation of the vector Susceptible class (S_v) increases with mosquitoes recruitment as (Λ_v) and reduces as population leaves to infected class via interaction with infected human at the rate (λ_v). It also reduces as mosquitoes can also dies without infection as (μ_v).

The population of infected vector (I_v) increases as population moves from susceptible vector class to infected via interaction (λ_v) and decreases due to natural death (μ_v). These lead us to the following system of Ordinary Differential Equations

$$\frac{dS_h}{dt} = \wedge_h p + \theta V_h + rR_h - (\lambda_h + \phi + \mu_h)S_h \tag{3.1}$$

$$\frac{dI_h}{dt} = \lambda_h S_h - (\mu_h + \mu_0 + \delta) I_h \tag{3.2}$$

$$\frac{dR_h}{dt} = \delta I_h - (\mu_h + r) R_h \tag{3.3}$$

$$\frac{dV_h}{dt} = \wedge_h (1-p) + \phi S_h - (\theta + \mu_h) V_h \tag{3.4}$$

$$\frac{dS_v}{dt} = \wedge_v - (\lambda_v + \mu_v) S_v \tag{3.5}$$

$$\frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v \tag{3.6}$$

Equilibrium State

At equilibrium we have that

$$\frac{dS_h}{dt} = \frac{dI_h}{dt} = \frac{dR_h}{dt} = \frac{dV_h}{dt} = \frac{dS_v}{dt} = \frac{dI_v}{dt} = 0$$

Let $x_1 = S_h \;\; x_2 = I_h \; x_3 = R_h \; x_4 = V_h \; x_5 = S_v \; x_6 = I_v \; \lambda_h = \frac{\beta c \, x_6}{N}, \lambda_v = \frac{\delta \rho \, x_2}{N}$ (3.7)

Substituting equation (3.7) in to equations (3.1) to (3.6) gives

$$\wedge_h \; p + \theta x_4 + r x_3 - (\frac{\beta c \, x_6}{N} + \phi + \mu_h) x_1 = 0 \tag{3.8}$$

$$\frac{\beta c \, x_6}{N} x_1 - (\mu_h + \mu_0 + \delta) x_2 = 0 \tag{3.9}$$

$$\delta x_2 - (\mu_h + r) x_3 = 0 \tag{3.10}$$

$$\wedge_h (1-p) + \phi x_1 - (\theta + \mu_h) x_4 \; = 0 \tag{3.11}$$

$$\wedge_v - (\frac{\delta \rho \, x_2}{N} + \mu_v) x_5 \; = 0 \tag{3.12}$$

$$\frac{\delta \rho \, x_2}{N} x_5 - \mu_v x_6 = 0 \tag{3.13}$$

Solving (3.8) to (3.13) simultaneously gives as the disease free equilibrium state.

$$(x_1, x_2, x_3, x_4, x_5, x_6) = \begin{pmatrix} \frac{\theta \wedge_h p - \theta \wedge_h -(\theta+\mu_h) \wedge_h p}{\theta\phi - (\theta+\mu_h)(\theta+\mu_h)}, 0, 0, \\ \frac{(\wedge_h p - \theta \wedge_h - \wedge_h p)}{\theta\phi}, \frac{\wedge_v}{\mu_v}, 0 \end{pmatrix} \quad (3.14)$$

Basic Reproduction Number

In obtaining the basic reproduction number, we only consider the infected classes of the model equation. They are as follows

$$\frac{dI_h}{dt} = \lambda_h S_h - (\mu_h + \mu_0 + \delta) I_h \quad (3.15)$$

$$\frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v \quad (3.16)$$

From (3.15) and (3.16) we obtained the following

$$F = \begin{bmatrix} 0 & \lambda_h S_h \\ \lambda_v S_v & 0 \end{bmatrix} \quad (3.17)$$

$$V = \begin{bmatrix} (\mu_h + \mu_0 + \delta) & 0 \\ 0 & \mu \end{bmatrix} \quad (3.18)$$

The determinants of V is obtained as

$$\mu(\mu_h + \mu_0 + \delta) \quad (3.19)$$

$$V^{-1} = \frac{1}{\mu(\mu_h + \mu_0 + \delta)} \begin{bmatrix} \mu & 0 \\ 0 & (\mu_h + \mu_0 + \delta) \end{bmatrix} \quad (3.20)$$

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu_h + \mu_0 + \delta} & 0 \\ 0 & \frac{1}{\mu} \end{bmatrix} \tag{3.21}$$

$$FV^{-1} = \begin{bmatrix} 0 & \frac{\lambda_h S_h}{\mu} \\ \frac{\lambda_v S_v}{\mu_h + \mu_0 + \delta} & 0 \end{bmatrix} \tag{3.22}$$

$$\begin{bmatrix} -\lambda & \frac{\lambda_h S_h}{\mu} \\ \frac{\lambda_v S_v}{\mu_h + \mu_0 + \delta} & -\lambda \end{bmatrix} = 0 \tag{3.23}$$

Simplifying equation (3.23) we have

$$\lambda^2 = \frac{\lambda_h S_h \lambda_v S_v}{\mu(\mu_h + \mu_0 + \delta)} \tag{3.24}$$

$$\lambda = \frac{\pm\sqrt{\lambda_h S_h \lambda_v S_v}}{\mu(\mu_h + \mu_0 + \delta)} \tag{3.25}$$

Therefore, basic reproduction number is the largest of the Eigen value which is the positive.

$$R_0 = \frac{+\sqrt{\lambda_h S_h \lambda_v S_v}}{\mu(\mu_h + \mu_0 + \delta)} \tag{3.26}$$

Analytical solution of the Model using Homotopy Perturbation Method (HPM)

Consider the system of the equations

$$\frac{dS_h}{dt} = \wedge_h p + \theta V_h + rR_h - (\lambda_h + \phi + \mu_h)S_h \tag{3.27}$$

$$\frac{dI_h}{dt} = \lambda_h S_h - (\mu_h + \mu_0 + \delta) I_h \tag{3.28}$$

$$\frac{dR_h}{dt} = \delta I_h - (\mu_h + r) R_h \tag{3.29}$$

$$\frac{dV_h}{dt} = \wedge_h (1-p) + \phi S_h - (\theta + \mu_h) V_h \tag{3.30}$$

$$\frac{dS_v}{dt} = \wedge_v - (\lambda_v + \mu_v) S_v \tag{3.31}$$

$$\frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v \tag{3.32}$$

Using Homotopy Perturbation Method (HPM) With initial conditions
$S_h(0) = S_{h0},\ I_h(0) = I_{h0},\ R_h(0) = R_{h0},\ S_V(0) = S_{v0},\ I_V(0) = I_{v0}$
we obtained the general analytical solutions to the model as

$$s_h(t) = s_h^0 + (\wedge_h prR_h^0 - \theta v_h^0 + \mu_h + \lambda_h s_h^0)t + \begin{bmatrix} \wedge_h p(\delta I_h^0 R_h^0 + \mu_h R_h^0 + r s_h^0 R_h^0) - \theta(\wedge_h (1-p) + \theta s_h^0 v_h^0 + \\ \lambda_h(\wedge_h prR_h^0) - \theta v_h^0 + \mu_h + \lambda_h s_h^0) + \lambda_h(-\lambda_v s_v^0 I_h^0 - \mu_V I_v^0 s_h^0) \end{bmatrix} \frac{t^2}{2} \tag{3.33}$$

$$I_h(t) = (I_h^0) + (\lambda_h(s_h^0)(I_h^0) - (\mu_h + \mu_0 + \delta) I_h 0)t + \begin{bmatrix} \lambda_h(s_h^0) - \lambda_v(s_v^0)(I_v^0) - \mu_v(I_v^0) + \lambda_h(\wedge_h pr(R_h^0) - (\theta - \varphi) v_h^0 + \\ \mu_h + \lambda_h(s_v^0)(I_v^0) - (\mu_h + \mu_0 + \delta)(\lambda_h(s_h^0)(I_v^0) - (\mu_h + \mu_0 + \delta) I_h 0 \end{bmatrix} \frac{t^2}{2} \tag{3.34}$$

$$R_h(t) = (R_h^0) + (\delta(I_h^0)(R_h^0) + \mu_h(R_h^0) + r(s_h^0)(R_h^0))\, t + \begin{bmatrix} \delta(I_h^0)(\delta(I_h^0)(R_h^0) + \mu_h(R_h^0) + r(s_h^0)(R_h^0) + \delta(\lambda_h(s_h^0)(-\mu_h + \mu_0 + \delta)(R_h^0) \\ + \mu_h(\delta(I_h^0)(R_h^0) + \mu_h(R_h^0) + r(R_h^0)) + r(\delta(I_h^0)(R_h^0) + \mu_h(R_h^0) + r(R_h^0) \end{bmatrix} \frac{t^2}{2} \tag{3.35}$$

$$v_h(t) = v_h^0 + (\wedge_h (1-p) + \varphi(s_h^0)(v_h^0))t + \begin{bmatrix} \theta(s_h^0)(\wedge_h (1-p) - \varphi(v_h^0)) + \theta(\wedge_h pr(R_h^0) \\ -\varphi v_h^0 + \mu_h + \lambda_h((I_v^0)(s_h^0))(v_h^0) \end{bmatrix} \frac{t^2}{2} \tag{3.36}$$

$$S_V(t) = (s_v^0) + = (\wedge_v \mu_v(s_v^0) + \lambda_v(s_h^0)(s_v^0))t + \begin{bmatrix} \wedge_v \mu_v(\wedge_v + \mu_v(s_v^0) + \lambda_v(s_h^0)(s_v^0)) + \lambda_v(s_h^0)(\wedge_v + \mu_v(\wedge_v + \mu_v(s_v^0) + \\ \lambda_v(s_h^0)(s_v^0) + \lambda_v(s_v^0)(\lambda_h(s_h^0)(I_h^0) - \mu_h + \mu_0 + \delta) \end{bmatrix} \frac{t^2}{2} \tag{3.37}$$

$$I_v(t) = I_v^0 + (-\lambda_v(s_v^0)(I_h^0) - \mu_v(I_v^0))t + \begin{bmatrix} -(s_v^0)(\lambda_v(s_h^0)(I_h^0) - \mu_h + \mu_0 + \delta) - (\wedge_v \mu_v(s_v^0) + \\ \lambda_v(s_h^0)(s_v^0)(I_h^0) - \mu_v(-\lambda_v(s_v^0)(I_h^0) - \mu_v(I_v^0) \end{bmatrix} \frac{t^2}{2} \tag{3.38}$$

STABILITY ANALYSIS OF DISEASE FREE EQUILIBRIUM (DFE)

We recall that the system of equation of the model at equilibrium is:

$$\frac{dS_h}{dt} = \wedge_h p + \theta V_h + rR_h - (\lambda_h + \phi + \mu_h)S_h = 0 \quad (4.1)$$

$$\frac{dI_h}{dt} = \lambda_h S_h - (\mu_h + \mu_0 + \delta)I_h = 0 \quad (4.2)$$

$$\frac{dR_h}{dt} = \delta I_h - (\mu_h + r)R_h = 0 \quad (4.3)$$

$$\frac{dV_h}{dt} = \wedge_h (1-p) + \phi S_h - (\theta + \mu_h)V_h = 0 \quad (4.4)$$

$$\frac{dS_v}{dt} = \wedge_v - (\mu_v + \lambda_v)s_v = 0 \quad (4.5)$$

$$\frac{dI_v}{dt} = \lambda_v S_v - \mu_v I_v \quad (4.6)$$

We obtained the characteristics equation from jacobian matrix as

$$\begin{vmatrix} rR_h + \theta v_h - \mu_h - \lambda_h) - \lambda & 0 & rs_h & \varphi s_h & 0 & -\beta s_h \\ \lambda_h I_v & -(\mu_h + \mu_0 + \delta) - \lambda & 0 & 0 & 0 & \lambda_h s_h \\ -rR_h & \delta R_h & \delta I_h - (\mu_h + rS_h) - \lambda & 0 & 0 & 0 \\ (\theta - \varphi)v_h & 0 & 0 & (\varphi s_h) - \lambda & 0 & 0 \\ 0 & -\lambda_v s_v & 0 & 0 & (\mu_v + \lambda_v) - \lambda & 0 \\ 0 & \lambda_v s_v & 0 & 0 & \lambda_v I_h & (-\mu_v) - \lambda \end{vmatrix} = 0 \quad (4.7)$$

Evaluating this gives

$$(\theta v_h - \mu_h - \beta I_v) - \lambda)((\mu_h + \mu_0 + \delta) - \lambda)(\delta I_h - (\mu_h + rS_h) - \lambda)(\phi s_h - \lambda)$$
$$(\mu_v + eI_h) - \lambda)(-\mu_v) - \lambda) + rs_h(\beta I_v rR_h)(-rR_h)(\theta v_h)(-(\mu_v + eI_h) - \lambda) -$$
$$(\theta s_h)(\beta I_v)(-(\mu_h + \mu_0 + \delta) - \lambda)(\delta I_h - \mu_h - rS_h) - \lambda)(-(\mu_v + eI_h) - \lambda)(-\mu_v) - \lambda) = 0 \quad (4.8)$$

But we recall from (3.46) that the DFE is given as

$$(x_1, x_2, x_3, x_4, x_5, x_6) = \begin{pmatrix} \frac{\theta \wedge_h p - \theta \wedge_h -(\theta+\mu_h)\wedge_h p}{\theta\phi-(\theta+\mu_h)(\theta+\mu_h)}, 0, 0, \\ \frac{(\wedge_h p - \theta \wedge_h - \wedge_h p)}{\theta\phi}, \frac{\wedge_v}{\mu_v}, 0 \end{pmatrix} \quad (4.9)$$

Substitute (4.9) in to (4.8) and simplify

$$\theta\frac{-\wedge_h(1-p)\mu_h}{\theta\wedge_h p+\wedge_h(1-p)}-\mu_h)-\lambda)(-(\mu_h+\mu_0+\delta)-\lambda)((-\mu_h-r\left[\frac{\wedge_h p^2+\wedge_h(1-p)}{\mu_h}\right])-\lambda)(\theta\left[\frac{\wedge_h p^2+\wedge_h(1-p)}{\mu_h}\right])-\lambda)$$
$$(-\mu_v)-\lambda)(-\mu_v)-\lambda) \ =0 \quad (4.10)$$

Simplifying (4.10) and find the different values of λ, we have

$$\lambda_1 = \frac{-\wedge_h(1-p)\mu_h}{\wedge_h p+\wedge_h(1-p)}-\mu_h) \quad (4.11)$$

$$\lambda_2 = -(\mu_h+\mu_0+\delta) \quad (4.12)$$

$$\lambda_3 = -\mu_h - r\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right] \quad (4.13)$$

$$\lambda_4 = \theta\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right]-\phi\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right] \quad (4.14)$$

$$\lambda_5 = -\wedge_v \quad (4.15)$$

$$\lambda_6 = -\wedge_v \quad (4.16)$$

Therefore, $\lambda_1, \lambda_2, \lambda_3, \lambda_5 \ and \ \lambda_6$ are all less than zero (0), but λ_4 Is also less than zero iff $\theta\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right] < \phi\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right]$

Hence Disease Free Equilibrium State will be stable if $\theta\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right] < \phi\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right]$, and unstable if $\theta\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right] > \phi\left[\frac{\wedge_h p+\wedge_h(1-p)}{\mu_h}\right]$ i.e. if $\theta > \phi$

NUMERICAL SIMULATIONS OF THE MODEL.

In this section, we use maple software to plot the graph of the analytical solutions of our model.

Table 4.1: parameters and state variables showing their descriptions, values and sources

Parameters/state variables	Description	Values	Sources
S_h	Susceptible human	12000	Assumed
I_h	Infected Human	7000	Assumed
R_h	Recovered Human	2000	Assumed
V_h	Vaccinated Human	2000	Assumed
S_v	Susceptible Vector	1500	Assumed
I_v	Infected Vector	1500	Assumed
$\Lambda_h p$	Human recruitment rate	100	Kbenesh et al.(2009)
Λ_h	Human recruitment in vaccinated class	100	Kbenesh et al.(2009)
λ_h	Force of Infection in human	0.8333	Kbenesh et al.(2009)
μ_h	Death by infection in human	0.00004	Kbenesh et al.(2009)
μ_0	Natural death in human	0.00004	Kbenesh et al.(2009)
δ	Treatment rate in human	0.20	Kbenesh et al.(2009)
r	Probability of human being recovered	1/730	Kbenesh et al.(2009)
θ	Vaccinated parameter	0.2	Assumed
ϕ	Probability of human being vaccinated	0.1	Assumed

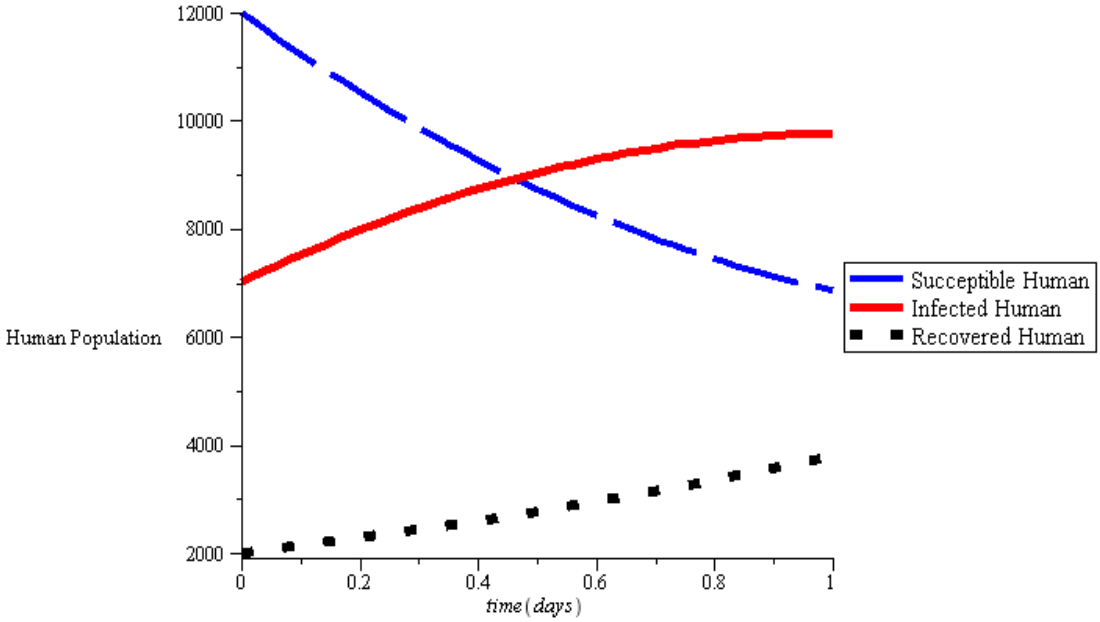

Figure 4.1 is a graph of Human population (SIR) against time at treatment rate δ = 0.20

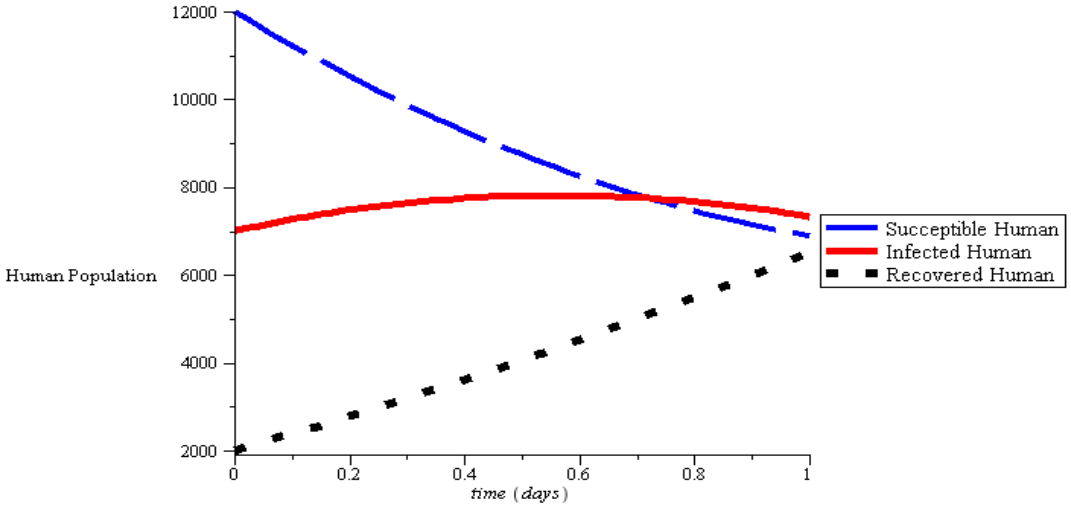

Figure 4.2 is a graph of Human Population (SIR) against time at treatment rate δ =0.55

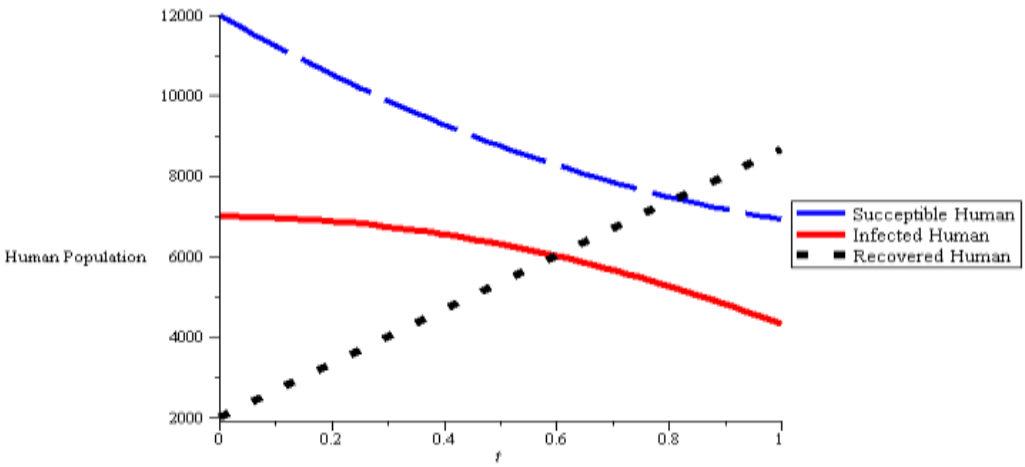

Figure 4.3 is a graph of Human Population (SIR) against time at treatment rate δ= 0.98

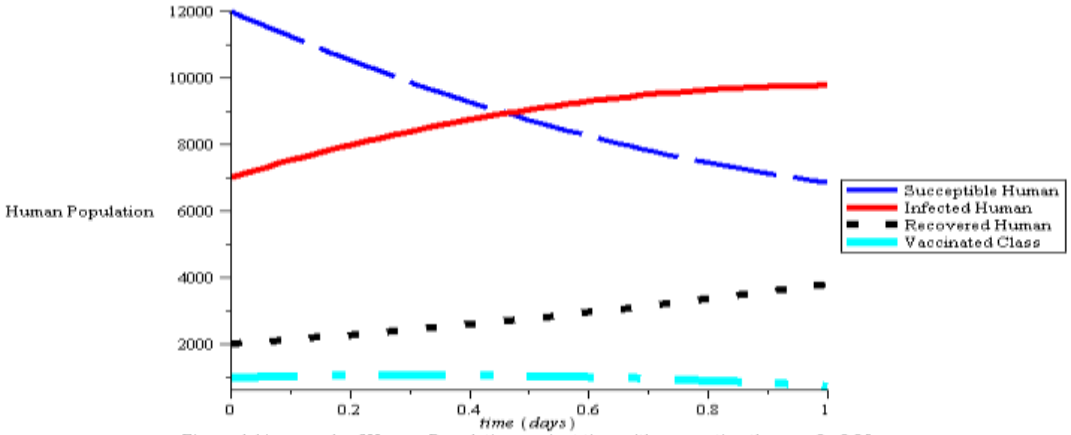

Figure 4.4 is a graph of Human Population against time with preventive therapy θ =0.05

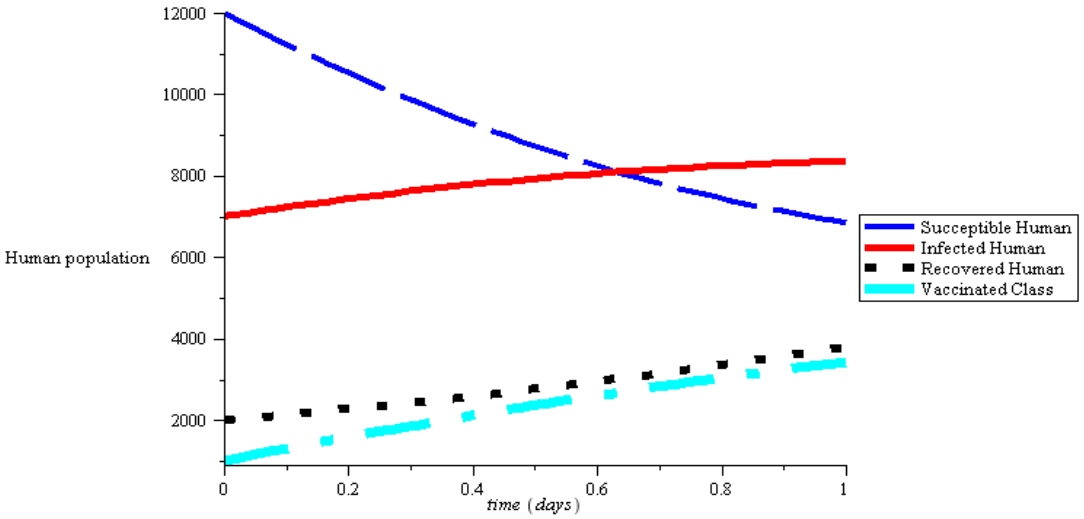

Figure 4.5 is a graph of Human Population (SIR) against time with preventive therapy θ =0.15

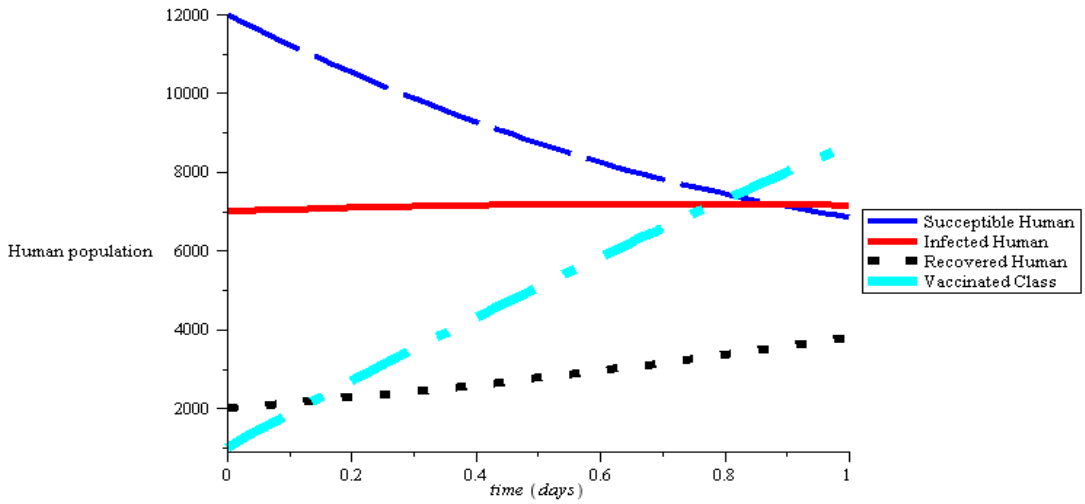

Figure 4.6 is a graph of Human Population (SIR) against time with preventive Therapy θ =0.55

DISCUSSION OF RESULTS

Figure 4.1 shows the graphs of Human population against time. When the treatment rate is low (δ=0.20) it was observed that the infected individuals are high and recovered individuals decreases

Figure 4.2 shows the graph of human population against time. When the treatment rate is moderate (δ=0.55), we discovered that the infected individual decreases and recovered individuals increases with time.

Figure 4.3 is a graph of Human population against time. When the treatment rate is high (δ= 0.98), the infected individuals decreases towards zero as recovered individuals increases

In figure 4.4, when the vaccination was low (θ = 0.05), the vaccinated class remain stable while infected class increases rapidly.

In figure 4.5, when the vaccination was moderate (θ= 0.15), the vaccinated class increases and infected classes decreases.

In figure 4.6, when the vaccination was high (θ=0.55), the vaccinated class increases and infected individuals decreases more than that of figure 4.5.

CONCLUSION

In this study, we investigated how malaria as a disease spreads in the population and possibility of its prevention and controls. We presented a mathematical model for transmission dynamics and qualitative analysis of malaria incorporating Therapy/Vaccination. The Disease Free Equilibrium States of the model was

obtained, analyzed for stability and was asymptotically stable. The Basic reproduction number was also evaluated and graphical profiles of each compartment was generated using maple mathematical software. The result shows that Vaccination plays crucial role in the eradication of the disease.

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