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MATHEMATICAL ANALYSIS OF MALARIA TRANSMISSION MODEL

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Abstract
A mathematical analysis of a malaria transmission model was carried out. The model which is a system of five ordinary differential equations is guided by the following assumptions: mosquitoes do not recover from malaria. The rate at which infectious humans enter the recovered group is proportional to the number of infections; a human or mosquito can die naturally at any stage; The number of infected humans increases at a rate which is proportional to the rate of susceptible humans. The existence and uniqueness of the solution of the model were established. The basic reproduction number of the model was calculated and it is less than 1. The stability of the disease-free equilibrium point of the model showed that the model was asymptotically stable indicating that in time malaria disease will completely die out. It was recommended that: the use of active drugs, use of insecticide, use of mosquito bed-treated nets and regular education of the public by the government on malaria would help in controlling the transmission of malaria since there is currently no perfect vaccine against malaria in humans.

Keywords: Asymptotic stability, basic reproduction number, disease-free equilibrium.

Introduction

Over the years, malaria has remained a deadly disease of serious concern to many countries of the world. Although prevalent among tropical areas of the world such as Africa, Asia, South America, and the Eastern Mediterranean region, it is a life-threatening disease with over one hundred countries as its high endemic regions (Nadjm & Behrens, 2012). It is an aged-long vector-borne infectious disease caused by protozoan plasmodium. Malaria is transmitted between humans through a bite by an infected female anopheles mosquito (the malaria vector). Different species of protozoa are responsible for the transmission of malaria. These include plasmodium falciparum, plasmodium ovale, plasmodium malariae and plasmodium vivax. Among these species, plasmodium falciparum is recognised as the most dangerous to humans (Esteva et al., 2009). In Africa and South East Asia, it has been observed that plasmodium falciparum is responsible for 80% of all cases of malaria and 90% of death (WHO Global Malaria Programme). Sadly, in 2008, malaria was a major public health challenge with countries declared endemic to the malaria disease. Malaria is prevalent in over 100 countries with approximately 216 million cases and 655,000 deaths in 2010 (Olanyi et al., 2018; WHO,2016). World health report indicated that there were 243 million cases of malaria and nearly a million deaths –mainly of children below 5 years (WHO Global Malaria Programme; Mandal et al., 2011).

In 2015, there was a global estimate of 214 million cases of malaria resulting in about 438,000 deaths (World Health Organization, 2016; Al-Rahman et al., 2017; Koutou et al., 2018). In 2018, the WHO Africa Region recorded 93% of cases of malaria and 94% of deaths due to malaria (Oke et al., 2020). WHO (2016), World Malaria Report revealed that there were 228 million cases of malaria in December 2019 compared with 231 million cases in 2017. It was estimated that the number of deaths was 405,000 in 2018 compared with 416,000 deaths in 2017 (Oke et al., 2020). Malaria infection can result in serious health challenges that affect the brain, kidneys including other organs of the body. An individual bitten by an infected mosquito may after a few days possess symptoms like fever, pain and chills and sweats may develop (Tumwiine et al., 2005b cited in Oguntolu and Gbolarin, 2019). Malaria is worse with HIV patients as it weakens their immune system thereby making them more vulnerable to contracting the disease. For severe HIV patients, malaria increases mortality by a factor of about 25% in non-stable malaria areas and is the fifth cause of death resulting from infectious disease worldwide after tuberculosis, respiratory infections, HIV/AIDS, and diarrheal diseases (Osman et al., 2017).

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Apart from health-related challenges, malaria poses a huge economic threat to malaria-endemic nations. In Africa alone, the annual economic burden of malaria was estimated to be $8 billion (Oke et al., 2020). The impact of malaria infection is not only economic but also social. For example, it keeps children away from school and adults away from work. The cost of treatment of malaria is often very expensive for parents and drives financially disadvantaged families into extreme poverty. The national economic loss due to malaria disease is great, cementing poverty and underdevelopment, especially in low-income countries (WHO, 2016).
Currently, there is no perfect vaccine for the treatment of malaria in humans (although global efforts are underway to develop such a vaccine). However, to control the spread of malaria, preventive measures can be taken. Such measures include: the use of mosquito-reduction strategies; self-protection from mosquito bites through the use of insecticide-treated bed nets; intermittent preventive personal treatment against malaria; reduction of malaria population through the destruction of their breeding sites etc. Other intervention strategies may include the use of indoor residual spraying for killing infected indoor mosquitoes, the use of anti-malaria drugs to regulate malaria and the use of sterile insect techniques.

Several mathematical models have been developed to investigate the dynamics of the transmission of malaria. Foremost of these models is that by Ross who developed a simple SIS (Susceptible-Infected-Susceptible) model with the assumption that at any time, the total population can be divided into distinct compartments. Using the model, Ross showed that bringing a mosquito population below a certain threshold was necessary to eliminate malaria. According to Ross, the threshold naturally depends on biological factors such as the bit ingrate and vectorial capacity. To estimate the rates of infection and recovery of malaria disease, Macdonald developed a model in which he assumed that the amount of infective material to which a population is exposed is not changed. The model showed that reducing the number of mosquitoes effectively has some effects on the spread and control of malaria in areas with the very great transmission. The Ross-Macdonald mathematical model comprises the interaction between infected human hosts and infected mosquitoes (vector). Oguntolu and Gbolarin (2019) proposed and analysed a mathematical model which describes the transmission dynamics of the malaria epidemic. Using the Differential Transform Method (DTM) which was validated by computer in-built classical fourth-order Runge-Kutta method, it was found that the solutions obtained with both methods were efficient, accurate and convergent. It was also found that the disease-free equilibrium point of the model was locally and globally asymptotically stable having a basic reproduction number $R_o<1$. Oke et al. (2020) proposed and analysed a mathematical model of malaria disease with a control strategy. In the study, the authors used a combination of two controls at a time while setting the other to zero, to investigate and compare the effects of the control strategies on malaria progression and eradication. Using Pontryagin's Maximum Principle and numerical simulations, analysis of the optimal control problem indicated that the combination of the three control strategies may be adopted in controlling malaria disease among the human and mosquito interacting populations. Numerical results revealed that the combination of the three control strategies: medication, use of treated bed nets and use of insecticide spray, have the highest impact on the control of malaria disease. Following was the combination of medication and the use of insecticide among the human population and lastly the combination involving the use of treated bed nets and use of an insecticide. Al-Rahman et al. (2017), considered SEIR-SEI model of malaria transmission between humans and mosquitoes. With the SEIR model, the authors estimated the basic reproduction number and discussed the disease-free and endemic equilibria using the Routh-Hurwitz criterion and second additive compound matrix respectively. Global stability of the disease-free and endemic equilibrium points was established using Lasselle's invariance principle of Lyapunov functions. The analytical and numerical simulation of the model indicated that malaria disease may be controlled by reducing the rate of contact between humans and mosquitoes. Further, it was found that the use of effective malaria drugs, insecticides and mosquito-treated nets are necessary measures to reduce the mosquito population and spread of malaria disease.

Koutou et al. (2018), examined the malaria transmission model in which the immature stages of the vector (malaria disease carrier) were taken into account. In the study, two models were considered namely: a model of vector population and a model of virus transmission. Using Lyapunov function, the authors showed using mathematical proof that the endemic equilibrium of the model was globally asymptotically stable. Theoretical results supported by numerical simulation indicated that the effect of immature stages is very important in the spread of mosquito population and that management of malaria disease is concerned first by reducing the mosquito threshold parameters to a value less than one. Baihaqi and Adi-Kusumo (2020), studied a mathematical model of malaria disease in the human population that can relapse without any contact with mosquitoes. The model was a 5-dimensional system of first-order ordinary differential equations with five variables S, E, I, R, S_p. The analysis of the model showed that there was the existence of an endemic equilibrium point indicating that the disease cannot

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be removed from the population but the number of infected individuals can still be isolated into a certain value. Tumwiine, et al. (2005a), proposed and analysed a mathematical model for the dynamics of malaria within human hosts and mosquitoes in which the reservoir of the susceptible human hosts is refilled by immunity loss to the disease and newborns. The model was reformulated in terms of the proportions of the classes of the respective populations. It was found that the basic reproduction number was independent of the rate of loss of immunity and that the disease-free equilibrium point of the model was globally asymptotically stable. Also, it was found that since malaria-induced immunity wanes over time and there are no effective vaccines against malaria at moment, intervention strategies such as the use of effective drugs, treated bed nets and insecticides would reduce the mosquito population and hence reduce contact between malaria vector and the human host.

Model Description

At any time t under consideration, the population of the malaria transmission model is divided into five compartments, namely: susceptible humans (S), infected humans (I), recovered humans (R), susceptible mosquitoes (V_1) and infected mosquitoes (V_2).

Model Assumptions

a) Mosquitoes do not recover from malaria, and the recovered humans do not enter the susceptible group again.
b) The rate at which infectious humans enter the recovered compartment is proportional to the number of infections.
c) The number of infected humans increases at a rate proportional to both the number of infectious humans and the number of susceptible humans.
d) A human or mosquito can die naturally at any stage.

Model Formulation

Following Oguntola and Gbolarin (2019), the description of malaria transmission dynamics is given by the following differential equations:

$$\frac{dS}{dt} = \wedge_h - \beta_h(1-\theta)SV_2 - \mu_h S \quad (1)$$

$$\frac{dI}{dt} = \beta_h(1-\theta)SV_2 - \delta_h I - \gamma_h I - \mu_h I \quad (2)$$

$$\frac{dR}{dt} = \gamma_h I - \mu_h R \quad (3)$$

$$\frac{dV1}{dt} = \wedge_v - \beta_v V_1 I - -\mu_v V_1 \quad (4)$$

$$\frac{dV2}{dt} = \beta_v V_1 I - -\mu_v V_2 \quad (5)$$

with $S(0) = 60>0$, $I(0)=40>0$, $R(0)=20>0$, $V_1=100>0$ and $V_2(0)=60>0$ (6)

and $N(t) = S(t) + I(t) + R(t) + V_1(t) + V_2(t)$ (7)

where: $\wedge_h$ is per capita birth rate of the human population per time, θ is the control parameter, μ_h is the natural death rate of the human population per time, β_h is the human contact rate per time, γ_h is the recovered rate of humans per time, δ_h is induced death rate for humans, β_v is mosquitoes contact rate per time, $\wedge_v$ is per capita birth rate of mosquito population per time and μ_v is the natural death rate of mosquito population per time.

Table1: Model parameter values

Parameter	Value	Source
$\wedge_h$	0.0013	Oguntola and Gbolarin (2018)
θ	0.25	”
μ_v	0.00135	”
β_h	0.0067	”
Y_h	0.5	”
δ_h	0.068	”
β_v	0.0019	”
$\wedge_v$	0.05	”
μ_h	0.0029	”

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3. Method of Solution

3.1 Positivity of Solution

In this section, we prove that the solution of the system of equations (1) – (5) with positive initial value will remain positive for all time (t).

Theorem: Given the closed set

$$\left\{ \begin{pmatrix} S \\ I \\ R \\ V_1 \\ V_2 \end{pmatrix} \in R^5_+ \;\middle|\; \begin{array}{l} S(0)>0 \\ I(0)>0 \\ R(0)>0 \\ V_1(0)>0 \\ V_2(0)>0 \\ S+I+R \leq \frac{\wedge_h}{\mu_h} \\ V_1 + V_2 \leq \frac{\wedge_v}{\mu_v} \end{array} \right\} \qquad (8)$$

then the solution [S(t), I(t), R(t), V1(t), V_2(t)] of the system of differential equations (1) –(5) is positive for all time t $\geq$0. In equation (1) above,

$\frac{dS}{dt} = \wedge_h - \beta_v(1-\theta)SV_2\mu_h - \mu_h S$

In the absence of infected mosquitoes, $\frac{dS}{dt} = \wedge_h - \mu_h S$.

By separation of variables, we have $\frac{dS}{S} + \mu_h S = \wedge_h$ (9)

Solving the first-order differential equation (9) by the method of integrating factor I.F, we have

I.F = $e^{\int \mu ht} = e^{\mu ht}$

$\frac{d}{dt}[S(e^{\mu ht})] = \wedge_h e^{\mu ht}$

$S(e^{\mu ht}) = \int \wedge h e^{\mu ht} dt$

$Se^{\mu ht} = \frac{\wedge h e^{\mu ht}}{\mu h} + c$

$S(t) = \frac{\wedge h}{\mu h} + ce^{-\mu ht}$ (10)

At t = 0, then $S(0) = \frac{\wedge h}{\mu h} + c$

$S_0 = \frac{\wedge h}{\mu h} + c$

$S_0 - \frac{\wedge h}{\mu h} = c$ (11)

Substituting (11) into (10), we have

$S(t) = \frac{\wedge h}{\mu h} + (S_0 - \frac{\wedge h}{\mu h}) e^{-\mu ht}$ (12)

Similarly, $I(t) > 0$, $R(t) > 0$, $V_1 > 0$, and $V_2 > 0$ (13)

Equations (12) and (13) show that the solution [S(t), I(t), R(t), V_1(t), V_2(t)] of the system of differential equations (1) – (5) is positive for all t $\geq$ 0.

Existence and Uniqueness of Solution of Differential Equations 1-5

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Theorem: Given the differential equation $\frac{dy}{dt}$ + P(t)y = g(t), if the functions p and g are continuous within an open interval I: α<t<β containing the point t=t_0, then there is a unique function y = ϕ(t) that satisfies the differential equation

$\frac{dy}{dt}$ + p(t)y = g(t) (14)

for every t in I, and that also satisfies the condition y(t_0) = y_0 (15)

where y_0 is an arbitrary initial value.

Proof: Let $\phi(t) = S(t) + I(t) + R(t)$ and then $\varphi(t) = V_1(t) + V_2(t)$ (16)

$\phi(t) = \wedge_h - \beta_h(1-\theta)SV_2 - \mu_h S + \beta_h(1-\theta)SV_2 - \delta_h I - \gamma_h I - \mu_h I + \gamma_h I - \mu_h R$

$\phi(t) = \wedge_h - \mu_h S - \delta_h I - \mu_h I - \mu_h R$

If $\delta_h = 0$, then

$\phi(t) = \wedge_h - \mu_h(S+I+R)$

$\frac{d}{dt}\phi(t) = \wedge_h - \mu_h \phi(t)$

$\frac{d}{dt}\phi(t) + \mu_h\phi(t) = \wedge_h$ (17)

Observe that equations (14) and (17) are similar and are first-order linear differential equations. Solving equation (17) by the method of integrating factor (I.F), we have

I.F = $e^{\int \mu h dt} = e^{\mu h t}$

$\frac{d}{dt}[\phi(t)e^{\mu h t}] = \wedge_h e^{\mu h t}$

Integrating both sides of the equation, we have

$\phi(t)\, e^{\mu h t} = \int \wedge h\, e^{\mu h t}$

$\phi(t)\, e^{\mu h t} = \frac{\wedge h}{\mu h} e^{\mu h t} + c$

$\phi(t) = \frac{\wedge h}{\mu h} + c e^{-\mu h t}$ (18)

Equation (18) is the general solution of equation (17). At the initial point when t = 0, then

$\phi(0) = S(0) + I(0) + R(0) = S_0 + I_0 + R_0 = \phi_0$ (19)

Substituting equation (19), into equation (18), we have

$\phi(0) - \frac{\wedge_h}{\mu_h} = c$ (20)

Rewriting equation (18) by substituting the value of c as in equation (20), we have

$\phi(t) = \frac{\wedge h}{\mu h} + [\phi_0 - \frac{\wedge h}{\mu h}]e^{-\mu h t}$ (21)

Similarly,

$\varphi(t) = \frac{\wedge v}{\mu v} + [\varphi_o - \frac{\wedge v}{\mu v}]e^{-\mu h t}$ (22)

Substituting equations (18) and (19) into equation (16), we have

$S(t = \left[\frac{\wedge h}{\mu h} + [\phi 0 - \frac{\wedge h}{\mu h}]e^{-\mu h t}\right] - [I(t) + R(t)]$ (23)

$I(t) = \left[\frac{\wedge h}{\mu h} + [\phi 0 - \frac{\wedge h}{\mu h}]e^{-\mu h t}\right] - [S(t) + R(t)]$ (24)

$R(t) = \left[\frac{\wedge h}{\mu h} + [\phi 0 - \frac{\wedge h}{\mu h}]e^{-\mu h t}\right] - [S(t) + I(t)]$ (25)

$V_1(t) = \left[\frac{\wedge v}{\mu v} + (\varphi o - \frac{\wedge v}{\mu v})e^{-\mu h t}\right] - V_2(t)$ (26)

$V_2(t) = \left[\frac{\wedge v}{\mu v} + [\varphi o - \frac{\wedge v}{\mu v}]e^{-\mu h t}\right] - V_1(t)$ (27)

Equations (21) – (27) show that there is a unique solution of the system of differential equations (1) – (5). Hence, the theorem has been proven.

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Basic Reproduction Number (R_0) of the Malaria Transmission Model.

The basic reproduction number, (R_0) is the expected number of secondary cases of infection brought about by a single (typical) infection in a completely susceptible population. To calculate R_0 of the system of differential equations (1) – (5), we use the next generation matrix denoted G. The matrix G consists of two matrices F and V defined as:

$$F = \begin{pmatrix} 0 & \beta h(1-\theta)S \\ {}^{\beta}\nu V1 & 0 \end{pmatrix} \text{ and } V = \begin{pmatrix} \delta h + \gamma h + \mu h & 0 \\ 0 & \mu v \end{pmatrix} \quad (28)$$

$$\text{with } V^{-1} = \begin{pmatrix} \frac{1}{\delta h+\gamma h+\mu h} & 0 \\ 0 & \frac{1}{\mu v} \end{pmatrix} \quad (29)$$

where F represents the matrix of new infections while matrix V transfers infections from one compartment to another and $G = FV^{-1}$.

$$G = FV^{-1} = \begin{pmatrix} 0 & \beta h(1-\theta)S \\ \frac{\beta v V1}{\delta h+\gamma h+\mu h} & 0 \end{pmatrix} \quad (30)$$

But at disease-free equilibrium, I(t) = 0, $S = \frac{\wedge h}{\mu h}$ and $V_1 = \frac{\wedge v}{\mu v}$ (31)

Substituting equation (31) into equation (30) we have

$$G = FV^{-1} = \begin{pmatrix} 0 & \frac{\Lambda h\beta h(1-\theta)}{\mu h\mu v} \\ \frac{\beta v\Lambda v}{(\delta h+\gamma h+\mu h)\mu v} & 0 \end{pmatrix} \quad (32)$$

The basic reproduction number of the model equations (1) – (5) is the dominant eigenvalue or the spectral radius of the matrix G. Therefore,

$$R_0 = \sqrt{\frac{\Lambda h\Lambda v\beta h\beta v(1-\theta)}{(\delta h+\gamma h+\mu h)\mu h\ \mu v2}} \quad (33)$$

Stability Analysis of Disease–Free Equilibrium (DFE) Point E^0.

In this section, we qualitatively analyse the model equations (1) – (5) to investigate the existence and stability of its associated equilibria. Point E^0 is the steady state solution of the model equations (1) – (5) in the absence of infection. To establish the stability of E^0, the Jacobian matrix of the differential equations (1) – (5) is computed and evaluated based on the signs of the eigenvalues of the Jacobian matrix. Point E^0 is locally stable if the real parts of the eigenvalues are all negative and unstable if all the eigenvalues are not negative.

Local Stability of Disease-Free Equilibrium Point (E^0)

Lemma: The point E^0 of the model equations (1) – (5) is locally asymptotically stable if R_o <1 and unstable if R_o>1.

Proof: Let $G_1 = \frac{dS}{dt}$, $G_2 = \frac{dI}{dt}$, $G_3 = \frac{dR}{dt}$, $G_4 = \frac{dv}{dt}$ and $G_5 = \frac{dv2}{dt}$.

At the steady states of the model equations (1) – (5), the Jacobian matrix is

$$J(E^0) = \begin{pmatrix} \frac{\partial G1}{\partial S} & \frac{\partial G1}{\partial I} & \frac{\partial G1}{\partial R} & \frac{\partial G1}{\partial V1} & \frac{\partial G1}{\partial V2} \\ \frac{\partial G2}{\partial S} & \frac{\partial G2}{\partial I} & \frac{\partial G2}{\partial R} & \frac{\partial G2}{\partial V1} & \frac{\partial G2}{\partial V2} \\ \frac{\partial G3}{\partial S} & \frac{\partial G3}{\partial I} & \frac{\partial G3}{\partial R} & \frac{\partial G3}{\partial V1} & \frac{\partial G3}{\partial V2} \\ \frac{\partial G4}{\partial S} & \frac{\partial G4}{\partial I} & \frac{\partial G4}{\partial R} & \frac{\partial G4}{\partial V1} & \frac{\partial G4}{\partial V2} \\ \frac{\partial G5}{\partial S} & \frac{\partial G5}{\partial I} & \frac{\partial G5}{\partial R} & \frac{\partial G5}{\partial V1} & \frac{\partial G5}{\partial V2} \end{pmatrix} \quad (34)$$

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$$J(E^0) = \begin{pmatrix} -[\beta h(1-\theta)V2 + \mu h] & 0 & 0 & 0 & -\beta h(1-\theta)S \\ \beta h(1-\theta)V2 & -(\delta h + \gamma h + \mu h) & 0 & 0 & \beta h(1-\theta)S \\ 0 & \gamma h & -\mu h & 0 & 0 \\ 0 & -{}^{\beta}vV1 & 0 & -({}^{\beta}vI + \mu v) & 0 \\ 0 & {}^{\beta}vV1 & 0 & {}^{\beta}vI & -\mu v \end{pmatrix} \quad (35)$$

By evaluating the Jacobian matrix (35) at the point E^0, we have

$$J(E^0) = \begin{pmatrix} -\mu h & 0 & 0 & 0 & -\beta h(1-\theta)S \\ 0 & -(\delta h + \gamma h + \mu h) & 0 & 0 & \beta h(1-\theta)S \\ 0 & \gamma h & -\mu h & 0 & 0 \\ 0 & 0 & 0 & -\mu v & 0 \\ 0 & 0 & 0 & 0 & -\mu v \end{pmatrix} \quad (36)$$

By applying matrix row operation, the matrix (36) becomes

$$J(E^0) = \begin{pmatrix} -\boldsymbol{\mu}_h & 0 & 0 & 0 & -\beta_h(1-\boldsymbol{\theta})S \\ 0 & -(\delta_h + \gamma_h + \mu_h) & 0 & 0 & -\beta_h(1-\boldsymbol{\theta})S \\ 0 & 0 & -\mu_h & 0 & \frac{\beta h \gamma h(1-\theta)S}{Sh+\gamma h+\mu h} \\ 0 & 0 & 0 & -\mu_v & \frac{\beta h \gamma h(1-\theta)S}{Sh+\gamma h+\mu h} \\ 0 & 0 & 0 & 0 & \frac{[\beta h \beta h(1-\theta)sV1 - \mu h \mu v - \mu v Sh - \mu v \gamma h}{Sh+\gamma h+\mu h} \end{pmatrix} \quad (37)$$

The characteristic equation of the Jacobian matrix (37) is

$$[J-\lambda I] = \begin{pmatrix} -\boldsymbol{\mu}_h - \lambda_1 & 0 & 0 & 0 & -\beta_h(1-\boldsymbol{\theta})S \\ 0 & -(_h+\gamma_h+\mu_h) - \lambda_2 & 0 & 0 & \beta_h(1-\boldsymbol{\theta})S \\ 0 & 0 & -\mu_h - \lambda_3 & 0 & \frac{-\beta h \gamma h(1-\theta)s}{Sh+\gamma h+\mu h} \\ 0 & 0 & 0 & -\mu v - \lambda_4 & \frac{\beta h \gamma h(1-\theta)s}{Sh+\gamma h+\mu h} \\ 0 & 0 & 0 & 0 & \frac{[\beta h \beta h(1-\theta)sV1 - \mu h \mu v - \mu v Sh - \mu v \gamma h] - \lambda 5}{Sh+\gamma h+\mu h} \end{pmatrix} \quad (38)$$

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The eigenvalues of the characteristic equation are:

$\lambda_1 = -\mu_h < 0$ (39)

$\lambda_2 = -(S_h+\gamma_h + \mu_h) < 0$ (40)

$\lambda_3 = -\mu_h < 0$ (41)

$\lambda_4 = -\mu_h < 0$ (42)

$\lambda_5 = -\frac{[\beta h\beta v(1-\Theta)SV1-\mu h\mu v-\mu vSh-\mu v\gamma h]}{Sh+\gamma h+\mu h} < 0$ (43)

From equation (40), we see that

$\beta_h\beta_v(1-\theta)SV - (\mu h + Sh + \gamma h)\mu_v < O$

$\frac{\beta h\beta v(1-\theta)SV1}{(\mu h+Sh+\gamma h)\mu v} - 1 < 0$

$\frac{\beta h\beta v(1-\theta)SV1}{(\delta h+\gamma h+\mu h)\mu v} < 1$ (44)

Substituting equation (31) into equation (44), we have

$\frac{\Lambda 1\Lambda v\beta h\beta v(1-\theta)}{(\delta_h+\gamma_h+\mu_h)\mu v\mu v^2} < 1$

$\sqrt{\frac{\Lambda_h \Lambda_v \beta_h\beta_v(1-\theta)}{(\delta_h+\gamma_h+\mu_h)\mu_h\mu v^2}} < 1$

$R_0 < 1$ (45)

Since $\lambda_1 < 0$, $\lambda_2 < 0$, $\lambda_3 < 0$, $\lambda_4 < 0$, and $\lambda_5 < 0$ and $R_0 < 1$, then the disease – free equilibrium point E^0 is locally asymptotically stable.

Global Stability of Disease – Free Equilibrium Point (E^0)

Lemma: The point E^0 of the model system of differential equations (1) – (5) is globally asymptotically stable if $R_0 \leq 1$.

Proof: Consider the Lyapunov-Lasalle function

$L(S,I,R,V_1,V_2) = \wedge_v\beta_v\boldsymbol{\mu}_h I + (\boldsymbol{\delta}_h+\gamma_h+\mu_h)\,\mu_h\,\mu_v V_2$ (46)

Differentiating equation (43), we have

$\frac{dL}{dt} = \wedge_v\beta_v\boldsymbol{\mu}\frac{dI}{dt} + (\boldsymbol{\delta}_h+\gamma_h+\mu_h)\,\mu_h\,\mu_v\frac{dV2}{dt}$

$= \wedge_v\beta_v\boldsymbol{\mu}_h[\beta_v(1-\Theta)\,\boldsymbol{\delta}V_2 - (\boldsymbol{\delta}_h+\gamma_h+\mu_h)\,I] + (\boldsymbol{\delta}_h+\gamma_h+\mu_h)\,\mu_h\,\mu_v\,(\beta_v V_1 I - \mu_v V_2)$ (47)

Since $S \leq S^0$, $I \leq I^0$, $R \leq R^0$ and $V_1 \leq V_1{}^0$, then equation (44) becomes

$\frac{dL}{dt} \leq [\frac{\Lambda_h \Lambda_v\beta_h\beta_v(1-0)}{(\delta_h+\gamma_h+\mu_h)\mu_h\mu_v 2} - 1\,]V_2$

$\frac{dL}{dt} \leq (R_0{}^2 - 1)V_2$

If $\frac{dL}{dt} \leq O$, then $R_0{}^2 - 1 \leq 0$

$R_o{}^2 \leq 1$ (48)

Equation (48) shows that the disease-free equilibrium point E^0 is globally asymptotically stable.

Result

In equation (33) above, $R_0 = \sqrt{\frac{\Lambda h\Lambda v\beta h\beta v(1-\theta)}{(\delta h+\gamma h+\mu h)\mu h\,\mu v2}}$. By substituting the model parameter values: $\wedge_h = 0.0013$, $\wedge_v = 0.00135$, $\beta_h = 0.0067$, $\beta_v = 0.0019$, $\boldsymbol{\theta} = 0.25$, $\boldsymbol{\delta}_h = 0.068$, $\gamma_h = 0.5$, $\mu_h = 0.0029$ and $\mu_v = 0.00135$ as given in table 1, we have

$R_0 = 0.4535 < 1$

Discussion of Findings

The result $R_0 = 0.4535 < 1$ verifies the mathematical proof of equation (33). It shows that the disease–free equilibrium point E^0 of the system of differential equations (1) – (5) is stable that is, malaria will in time die out.

Conclusion

This paper examined an SIR mathematical model which describes the dynamics of the transmission of malaria in a given population. The model which is a deterministic system of non-linear ordinary differential equations was analysed to determine the possibility of the eradication of malaria. The basic reproduction number of the model was calculated and the value is less than 1. The eigenvalues of the Jacobian matrix of the disease-free equilibrium

point were all negative indicating that the disease-free equilibrium point is asymptotically stable. This shows that malaria will in time die out.

Recommendations

To help control the transmission of malaria, the following measures would prove helpful.

i Use active malaria drugs
ii Use of insecticide
iii Use of malaria bed-treated nets
iv Regular education of the public by the government on malaria.

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