

A Mathematical Model of Malaria Disease with Vertical Transmission

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Abstract

In this paper, we analyzed dynamics of malaria disease by a compartment model involving ordinary differential equations for the human and mosquito populations. An equivalent system is obtained, which has two equilibriums: a disease-free equilibrium and an endemic equilibrium. The stability of these two equilibriums is controlled by the basic reproduction number R_0 . In this model the disease-free equilibrium state is stable if $R_0 < 1$ and if $R_0 > 1$, the endemic equilibrium stable. The analytical predictions are conformed by numerical simulation and graphical results.

Keywords: Mathematical modelling, malaria disease, equilibrium, stability, basic reproduction number

1. Introduction

Mathematical models have been widely used in various areas of infectious disease epidemiology. Mathematically modelling of malaria disease transmission in human and vector populations has been done since the beginning of last century (Aron, 1988; Bailey, 1982; Ross, 1911). Epidemiology modelling can contribute the design and analysis of epidemiological survey, suggest crucial data that should be collected, identify trends, make general forecasts and estimate the uncertainty in forecasts (Anderson et al., 1991; Hale, 1969; Hethcote, 2000; Hethcote, 1976).

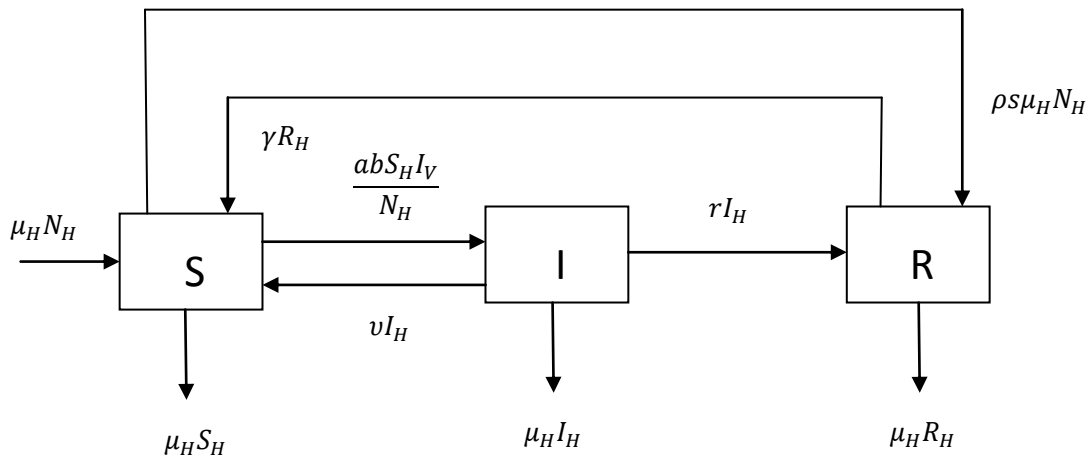
Malaria can be defined as “a mosquito-borne infectious disease of humans and many types of animals and birds”. Malaria is caused by a single-celled parasite from the genus Plasmodium. The general symptoms of Malaria typically include fever, fatigue, muscular pains and occasionally nausea, vomiting and diarrhea. In later stages it can cause yellow skin, seizures, coma or death. The disease is transmitted by the biting of mosquitoes, and the symptoms first appear one and half week to two weeks after the infectious mosquito bite. If not properly treated, malaria attacks can recur at regular time periods (in every 2 days or every 3 days). In those who have recently survived an infection, re-infection typically causes milder symptoms. This partial resistance disappears over months to years if the person has no continuing exposure to malaria. In other areas, where the infection rate is low, people do not develop immunity because they rarely infected by this disease. This makes them more susceptible to the ravages of an epidemic. During the last decades various mathematical models have been used for infectious diseases especially for malaria (Ngwa et al., 2000; Olumese, 2005; Sachs, 2002; Tumwiine et al., 2005). In case of malaria, mathematical models were used in comparing planning, implementing, evaluating and optimizing various detection, therapy and control programs.

In this paper, we are developing mathematical models to better understand the transmission and spread of malaria disease. We modified the model of Tumwiine, Mugisha and Luboobi (2007) by considering a fraction of transmitted part is shifted to infectious and remaining part gets recovered without becoming infectious. The aim of this study is to investigate the effects of vaccination in human population and vector population.

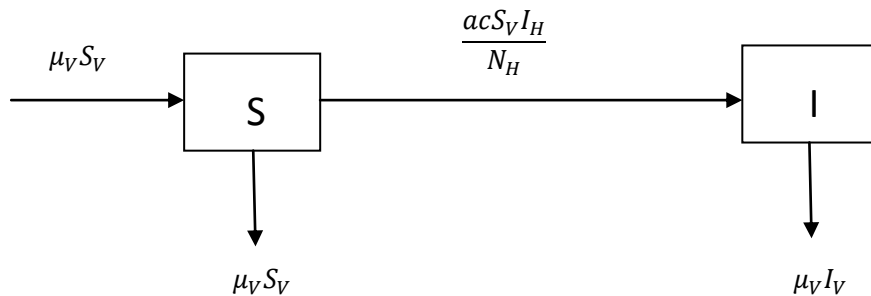
2. Model Formulation

Let N_H and N_V denote the human and mosquito population with the total population size at time t . We assume that the host and vector population has constant size with birth and death rate equal to μ_H and μ_V . The human population of size N_H is formed of Susceptible S_H , Infective I_H and Recovered R_H and vector population divided into S_V and I_V .

Human Population:



Vector population:



Let a portion ρ , $0 \leq \rho \leq 1$, of newborn host be vaccinated. Assume that the vaccine is not perfect and let effectiveness of the vaccine is s , then $\mu_H(1 - \rho s)N_H$ newborn remains susceptible, and $\mu_H \rho s N_H$ directly being removed to R_H . The governing equations are:

Human population:

$$\begin{aligned} \frac{dS_H}{dt} &= \mu_H(1 - \rho s)N_H - \frac{abS_H I_V}{N_H} + vI_H + \gamma R_H - \mu_H S_H \\ \frac{dI_H}{dt} &= \frac{abS_H I_V}{N_H} - vI_H - rI_H - \mu_H I_H \end{aligned} \tag{2.1}$$

$$\frac{dR_H}{dt} = \mu_H \rho s N_H + rI_H - \gamma R_H - \mu_H R_H$$

Vector population:

$$\frac{dS_V}{dt} = \mu_V N_V - \frac{acS_V I_H}{N_H} - \mu_V S_V \tag{2.2}$$

$$\frac{dI_V}{dt} = \frac{acS_V I_H}{N_H} - \mu_V I_V$$

Here the parameters in the model stand for

Table 2.1 Description of variable of the model

Variables	Interpretation
a	The average infection rate on man by a single mosquito.
b	The proportion of bites on man that produce an infection.
c	The probability that a mosquito becomes infectious.
γ	The per capita rate of loss of immunity in human hosts.
r	The rate at which human hosts acquire immunity.
v	The rate of recovery of human hosts from the disease.

Using $S_H + I_H + R_H = N_H$ and $S_V + I_V = N_V$, the system (2.1) and (2.2) become

$$\begin{aligned} \frac{dS_H}{dt} &= \mu_H(1 - \rho s)N_H - \frac{abS_H I_V}{N_H} + vI_H + \gamma(N_H - S_H - I_H) - \mu_H S_H \\ \frac{dI_H}{dt} &= \frac{abS_H I_V}{N_H} - vI_H - rI_H - \mu_H I_H \\ \frac{acI_H(N_V - I_V)}{N_H} &- \mu_V I_V \end{aligned} \tag{2.3}$$

Writing the system (2.3) in population proportion

$S_h = \frac{S_H}{N_H}$, $I_h = \frac{I_H}{N_H}$ and $I_v = \frac{I_V}{N_V}$, We write

$$\begin{aligned} \frac{dS_h}{dt} &= \mu_H(1 - \rho s) - ab\phi S_h I_v + vI_h + \gamma - \gamma(S_h + I_h) - \mu_H S_h \\ \frac{dI_h}{dt} &= ab\phi S_h I_v - vI_h - rI_h - \mu_H I_h \\ \frac{dI_v}{dt} &= acI_h(1 - I_v) - \mu_V I_v \end{aligned} \tag{2.4}$$

Where $\phi = \frac{N_V}{N_H}$ is the ratio of host and vector population.

Further we rescale t by ac and let $x = S_h, y = I_h$ and $z = I_v$,

$$\begin{aligned} \frac{dx}{dt} &= \mu(1 - \pi) - \sigma x - \eta x z + ky + \varphi - \varphi y \\ \frac{dy}{dt} &= \eta x z - ky - my \\ \frac{dz}{dt} &= y(1 - z) - \omega z \end{aligned} \tag{2.5}$$

Where

$$\mu = \frac{\mu_H}{ac}, \pi = \rho s, \eta = \frac{b\phi}{c}, k = \frac{v}{ac}, \varphi = \frac{\gamma}{ac}, \sigma = \frac{\mu_H + \gamma}{ac}, m = \frac{\mu_H + r}{ac}, \omega = \frac{\mu_V}{ac}$$

3. Steady State and Equilibrium Points

The system (2.5) has a disease-free equilibrium point $E_0 \left(\frac{\mu(1-\pi)+\varphi}{\sigma}, 0, 0 \right)$ and an endemic equilibrium Point

$E_1(x_e, y_e, z_e)$, where

$$x_e = \frac{(k + m)[\mu(1 - \pi) + \varphi + (m + \varphi)\omega]}{\eta(m + \varphi) + \sigma(k + m)} = \frac{(v + \mu_H + r)[ac\{\mu_H(1 - \rho s) + r\} + (\mu_H + r + \gamma)\mu_V]}{ac[ab\phi(\mu_H + r + \gamma) + (\mu_H + \gamma)(v + \mu_H + r)]}$$

$$y_e = \frac{\eta[\mu(1-\pi) + \varphi] - \sigma(k+m)\omega}{\eta(m+\varphi) + \sigma(k+m)} = \frac{a^2bc\phi[\mu_H(1-\rho s) + \gamma] - (\mu_H + \gamma)(v + \mu_H + r)\mu_v}{ac[ab\phi(\mu_H + r + \gamma) + (\mu_H + \gamma)(v + \mu_H + r)]}$$

$$z_e = \frac{\eta[\mu(1-\pi) + \varphi] - \sigma(k+m)\omega}{\eta[\mu(1-\pi) + \varphi + (m+\varphi)\omega]} = \frac{a^2bc\phi[\mu_H(1-\rho s) + \gamma] - (\mu_H + \gamma)(v + \mu_H + r)\mu_v}{ab\phi[ac(\mu_H(1-\rho s) + \gamma) + (v + \mu_H + r)\mu_v]}$$

This has been obtained by setting the time derivatives of system (2.5) equal to zero. Here the basic reproduction number R_0 is defined by

$$R_0 = \frac{a^2bc\phi[\mu_H(1-\rho s) + \gamma]}{(\mu_H + \gamma)(v + \mu_H + r)\mu_v}$$

And the endemic equilibrium $E_1(x_e, y_e, z_e)$ is stable when

$$\eta[\mu(1-\pi) + \varphi] - \sigma(k+m)\omega > 0$$

4. Asymptotic Behavior of the Model

Theorem 4.1: If $R_0 < 1$, then the disease-free equilibrium E_0 is locally stable and if $R_0 = 1$, E_0 is stable and if $R_0 > 1$, the stable endemic equilibrium E_1 will appear.

Proof:

To discuss the stability of the model the governing dynamical system is

$$F_1 = \mu(1-\pi) - \sigma x - \eta x z + k y + \varphi - \varphi y \quad (4.1)$$

$$F_2 = \eta x z - k y - m y \quad (4.2)$$

$$F_3 = y(1-z) - \omega z \quad (4.3)$$

The Variation matrix of the system (4.1) to (4.2) is given by

$$J = \begin{pmatrix} -\sigma - \eta z & k - \varphi & -\eta x \\ \eta z & -k - m & \eta x \\ 0 & 1 - z & -y - \omega \end{pmatrix}$$

For disease-free equilibrium point $E_0\left(\frac{\mu(1-\pi)+\varphi}{\sigma}, 0, 0\right)$ the variation matrix will be

$$J(E_0) = \begin{pmatrix} -\sigma & k - \varphi & -\frac{\eta[\mu(1-\pi) + \varphi]}{\sigma} \\ 0 & -k - m & \frac{\eta[\mu(1-\pi) + \varphi]}{\sigma} \\ 0 & 1 & -\omega \end{pmatrix}$$

The characteristic equation of it will be

$$(\sigma + \lambda) \left[\lambda^2 + (k+m+\omega)\lambda + (k+m)\omega - \frac{\eta[\mu(1-\pi) + \varphi]}{\sigma} \right] = 0$$

By above equation at Eigen values, one can easily seen that disease-free equilibrium E_0 is locally stable if

$$(k+m)\omega - \frac{\eta[\mu(1-\pi) + \varphi]}{\sigma} > 0$$

i.e. $R_0 < 1$

Now we shall discuss about an endemic equilibrium and study its stability. For the endemic equilibrium $E_1 = (x_e, y_e, z_e)$, the variation matrix will be

$$J(E_1) = \begin{pmatrix} -\sigma - \eta z_e & k - \varphi & -\eta x_e \\ \eta z_e & -k - m & \eta x_e \\ 0 & 1 - z_e & -y_e - \omega \end{pmatrix}$$

$$J(E_1) = \begin{bmatrix} -\frac{(\sigma + \eta)[\mu(1 - \pi) + \varphi] + \sigma\varphi\omega - \sigma\omega k}{[\mu(1 - \pi) + \varphi + (m + \varphi)\omega]} & k - \varphi & -\frac{\eta(k + m)[\mu(1 - \pi) + \varphi + (m + \varphi)\omega]}{\eta(m + \varphi) + \sigma(k + m)} \\ \frac{\eta[\mu(1 - \pi) + \varphi] - \sigma(k + m)\omega}{[\mu(1 - \pi) + \varphi + (m + \varphi)\omega]} & -k - m & \frac{\eta(k + m)[\mu(1 - \pi) + \varphi + (m + \varphi)\omega]}{\eta(m + \varphi) + \sigma(k + m)} \\ 0 & \frac{\eta(m + \varphi)\omega + \sigma(k + m)\omega}{\eta[\mu(1 - \pi) + \varphi + (m + \varphi)\omega]} & -\frac{\eta[\mu(1 - \pi) + \varphi + (m + \varphi)\omega]}{\eta(m + \varphi) + \sigma(k + m)} \end{bmatrix}$$

Then the characteristic equation will be

$$a_3\lambda^3 + a_2\lambda^2 + a_1\lambda + a_0 = 0$$

Where

$$a_3 = [\eta(m + \varphi) + \sigma(k + m)][\mu(1 - \pi) + \varphi + (m + \varphi)\omega] > 0$$

$$a_2 = [\eta(m + \varphi) + \sigma(k + m)][(\sigma + \eta)\{\mu(1 - \pi) + \varphi\} + \sigma\varphi\omega - \sigma\omega k] + (k + m)[\mu(1 - \pi) + \varphi + (m + \varphi)\omega][\eta(m + \varphi) + \sigma(k + m)] + \eta[\mu(1 - \pi) + \varphi + (m + \varphi)\omega][\mu(1 - \pi) + \varphi + (m + \varphi)\omega] > 0$$

$$a_1 = [\eta\{\mu(1 - \pi) + \varphi\} - \sigma(k + m)\omega][(k + m)\{\mu(1 - \pi) + \varphi + (m + \varphi)\omega\} + (\varphi - k)\{\eta(m + \varphi) + \sigma(k + m)\}] + [(k + m)\eta(m + \varphi) + (k + m)^2\sigma + \eta\{\mu(1 - \pi) + \varphi\} + \eta\omega(m + \varphi)][(\sigma + \eta)\{\mu(1 - \pi) + \varphi\} + \sigma\varphi\omega - \sigma\omega k]$$

$$a_0 = [\eta\{\mu(1 - \pi) + \varphi\} - \sigma(k + m)\omega][(k + m)(\sigma + \eta)\{\mu(1 - \pi) + \varphi\} + \sigma\varphi\omega - \sigma\omega k] + (\varphi - k)\{\mu(1 - \pi) + \varphi + (m + \varphi)\omega\} + \omega(k + m)\{\eta(m + \varphi) + \sigma(k + m)\}$$

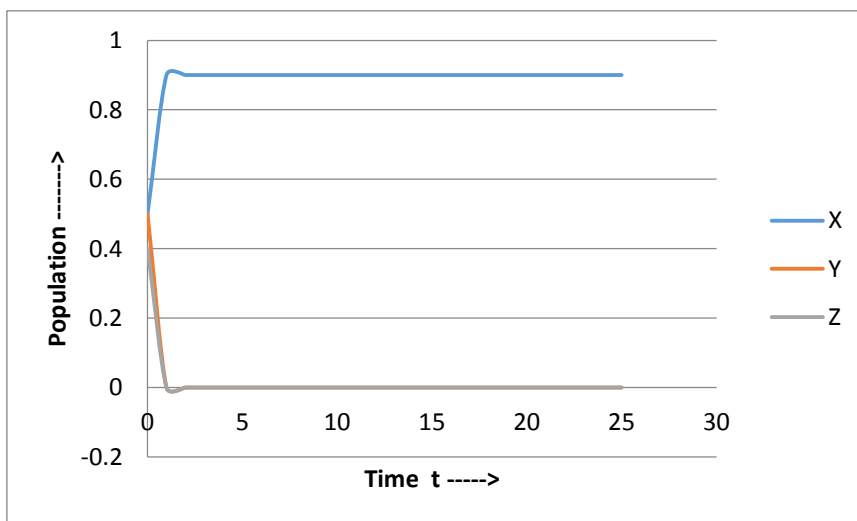
5. Numerical Result

To demonstrate the theoretical results obtained in this paper, we will give some numerical simulations. From practical point of view, numerical solutions are very important beside analytical study. In system (2.5),

Stability of disease-free state

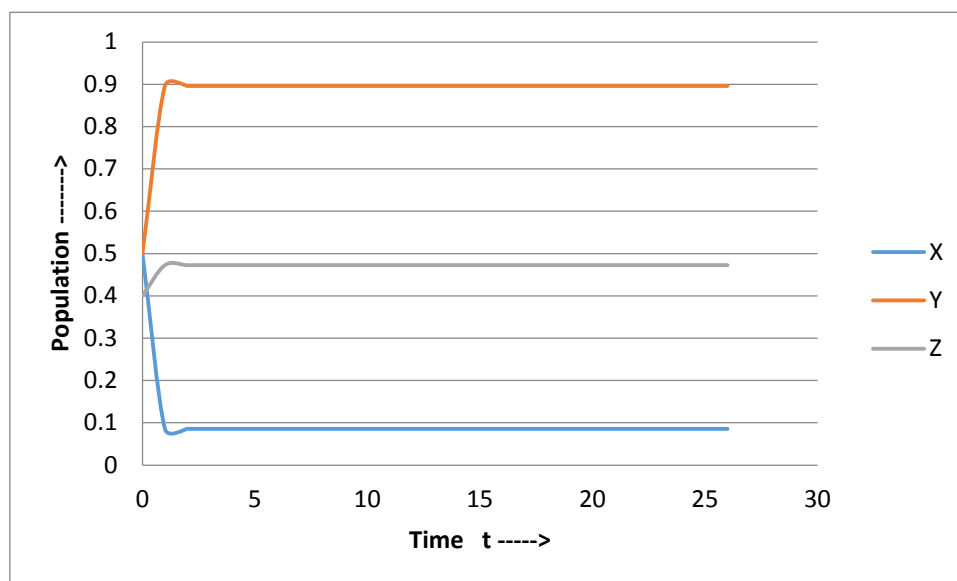
Let $\mu = 1.16, \pi = 0.009, \sigma = 2.1724, \eta = 0.00492, \psi = 0.9, k = 0.0026, m = 0.011,$

$\omega = 1$ and $(x(0), y(0), z(0)) = (0.5, 0.5, 0.4)$. Then the calculated disease free equilibrium point and basic reproduction number are: $E_0(x, 0, 0) = E_0(0.9002, 0, 0)$ and $R_0 = 0.325661507 < 1$. Fig.1 shows that $x(t)$ goes to its steady state, while $y(t)$ and $z(t)$ goes to zero with respect to time. Hence disease dies out.



Stability of endemic state

We change the value $\mu = 0.0024, \sigma = 1.0024, \eta = 0.301$ and all other parameter are as above. Then $E_1(x_e, y_e, z_e) = E_1(0.08566, 0.8960, 0.4725)$ and $R_0 = 19.91964755 > 1$. Therefore the endemic equilibrium E_1 is locally asymptotically stable. Fig.2 shows that (x_e, y_e, z_e) goes to their steady state values. Hence the disease becomes endemic.



5. Conclusion

In this paper, a mathematical model of malaria disease with vertical transmission analyzed. An equivalent system is obtained, which has two equilibriums: a disease-free equilibrium and an endemic equilibrium. The stability of these two equilibriums is controlled by the basic reproduction number R_0 . In this model the disease-free equilibrium state is stable if $R_0 < 1$ and if $R_0 > 1$, the endemic equilibrium stable.

References

- Anderson, R. M., & May, R.M. (1991). *Infectious Diseases of Humans: Dynamics and Control*, Oxford University Press, Oxford.
- Aron, J. L. (1988). Mathematical modelling of immunity to malaria. *Mathematical Biosciences*, 90, 385–396. [http://dx.doi.org/10.1016/0025-5564\(88\)90076-4](http://dx.doi.org/10.1016/0025-5564(88)90076-4)
- Bailey, N. T. J. (1982). *The Biomathematics of Malaria*, Charles Griff, London.
- Hale, J. K. (1969). *Ordinary Differential Equations*, John Wiley, New York.
- Hethcote, H. W. (2000). *The Mathematics of Infectious diseases*, SIAM.
- Hethcote, H. W. (1976). Qualitative analysis of communicable disease models. *Mathematical Biosciences*, 28, 335–356. [http://dx.doi.org/10.1016/0025-5564\(76\)90132-2](http://dx.doi.org/10.1016/0025-5564(76)90132-2)
- Ngwa, G. A., & Shu, W. S. (2000). A mathematical model for endemic malaria with variable human and mosquito populations. *Mathematics and Computer Modelling*, 32, 747–763. [http://dx.doi.org/10.1016/S0895-7177\(00\)00169-2](http://dx.doi.org/10.1016/S0895-7177(00)00169-2)
- Olumese, P. (2005). Epidemiology and surveillance: changing the global picture of malaria-myth or reality? *Acta Tropica*, 95, 265–269. <http://dx.doi.org/10.1016/j.actatropica.2005.06.006>
- Ross, R. (1911). *The Prevention of Malaria*, Murry, London.
- Sachs, J. D. (2002). A new global effort to control malaria. *Science*, 298, 122–124. <http://dx.doi.org/10.1126/science.1077900>
- Tumwiine, J., Luboobi, L. S., Mugisha, J. Y. T. (2007). A mathematical model for the dynamics of malaria in a human host and mosquito vector with temporary immunity. *Applied Mathematics and Computation*, 189, 1953–1965. <http://dx.doi.org/10.1016/j.amc.2006.12.084>
- Tumwiine, J., Luboobi, L. S., & Mugisha, J. Y. T. (2005). Modelling the effect of treatment and mosquitoes control on malaria transmission. *International Journal of Management and Systems*, 21, 107–124.

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