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Mathematical models of the Spread of Malaria with the vertical transmission (congenital malaria)

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Abstract

The main goal of this paper is to develop a mathematical model to study the dynamic of malaria transmission, and the direct effects of congenital malaria on the spread of malaria. In this study, we have clarified the significant impact of malaria on the human community through their impact on the newborn, and that directly increases spread of the malaria in the human community, especially in the newborns with the lower and inexperienced immunity systems. The existence and stability of the disease-free points of the system is analyzed. We established that the disease-free equilibrium point is locally asymptotically stable when the reproduction number $R_0 < 1$ and the disease always dies out. For $R_0 > 1$ the disease-free equilibrium becomes unstable and there exists a unique endemic equilibrium.

Keywords: Congenital malaria, vertical transmission, basic reproduction number, stability.

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1. Introduction

Malaria remains one of the most prevalent and lethal human infection worldwide. The World Health Organization estimates that each year 300-500 million cases of malaria occur and more than 1 million people die of malaria, especially in developing countries [41, 43]. Most deaths occur among young children. For example, in Africa, a child dies from malaria every 30 seconds [42]. Malaria is spread in three ways. The most common one is by the bite of an infected female Anopheles mosquito. However, Malaria can be transmitted by transfusion of blood from infected donors. First reported in 1911, transfusion malaria is one of the most common transfusion-transmitted infections today [9, 36]. The risk of acquiring transfusion malaria is very low (1 case per 4 million) in nonendemic countries such as the United States, whereas in the endemic countries, it is much higher (> 50 cases per million donor units) [7, 36]. Also malaria could be spread through a transfer of parasitized red cells from infected

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mother to the child either transplacentally or during labor, both can lead to malaria in the newborn, called as congenital malaria [10, 11]. Congenital malaria has been frequent in the nonimmune population in the endemic areas. In recent years, however, higher prevalence of congenital malaria ranging from 8% to 33%, has been reported from both malaria-endemic and nonendemic areas, including the United States, Europe, India, etc. [8, 13, 14, 16, 25, 26, 31, 37]. Congenital malaria was first described in 1876 [34]. It can be acquired by transmission of parasites from mother to child during pregnancy or perinatally during labour [21, 24, 30]. Moreover, congenital malaria is the most common type of human-to-human transmission, across various surveys of newborns in West Africa, between 8% to 24%, were found to be infected with malaria parasites [44].

About 300,000 foetal and infants deaths and 2,500 deaths of pregnant women are attributable to malaria [38]. Poor outcome for both mother and foetus is associated with pregnancy malaria and results in premature delivery, intrauterine growth retardation, perinatal mortality, anaemia, abortion, low birth weight and death of the mother [6, 24]. Moreover, perinatal malaria infection may cause cord parasitaemia, early neonatal malaria, as well as neonatal malaria. When the placenta is infected with malaria parasites, transplacental transmission of the parasites can occur, although the born infant may remain asymptomatic and healthy [32, 40].

Mathematical modeling of malaria began in 1911 with Rosss model [35], and major extensions are described in Macdonalds 1957 book [23]. The first models were two-dimensional with one variable representing humans and the other representing mosquitoes. An important addition to the malaria models was the inclusion of acquired immunity proposed by Dietz, Molineaux, and Thomas [12]. Anderson and May [1], Aron and May [3], Koella[18] and Nedelman [27] have written some good reviews on the mathematical modeling of malaria. Some recent papers have also included environmental effects ([22, 45] and [46]) the spread of resistance to drugs ([4] and [19]), and the evolution of immunity [20]. Recently, Ngwa and Shu [29] and Ngwa [28] proposed an ordinary differential equation (ODE) compartmental model for the spread of malaria involving variable human and mosquito populations, with a susceptible-exposed-infectious-recovered-susceptible (SEIRS) pattern for humans and a susceptible-exposed-infectious (SEI) pattern for mosquitoes, greatly developed the mathematical models of malaria, but the vertical transmission between human has not been studied. We have developed our model that includes the impact vertical transmission (congenital malaria) in this study.

In this paper, we propose a model to examine vertical transmission of malaria infection from mother to the newborn (congenital malaria), and study the direct effects of congenital malaria on the spread of malaria.

2. Mathematical Model description and analysis

2.1. Mathematical Model description

We formulate a model for the spread of malaria in the human and mosquito population with the vertical transmission from human to human. The total population size at time t given by N_H (t) and N_V (t), respectively. These are further compartmentalized into epidemiological classes shown below. The vector component of the model does not include immune class [5, 17], as mosquitoes never recover from infection, that is, their infective period ends with their death due to their relatively short life-cycle. Thus the immune class in the mosquito population is negligible and death occurs equally in all groups. The state variables (Table 1) and parameters (Table 2) for the malaria model (Figure 1) satisfy the equation (2.1). All parameters are strictly positive with the exception of the disease-induced death rate, which is nonnegative. The mosquito birth rate is greater than the density-independent, mosquito death rate, $B_V > \mu_V$, ensuring that we have a stable positive mosquito population.

Let a_V be the average number of bites per mosquito per unit time, then there are $\frac{a_V N_V}{N_H}$ bites per human per time. Since there are S_H susceptible humans and the proportion of the total number of bites that are potentially infectious to humans is $\frac{I_V}{N_V}$, the number of potentially infectious bites given to susceptible

humans is $\frac{a_V I_V S_H}{N_V}$ bites per time. However only a fraction of these bites, namely C_{VH} successfully infect humans. We thus have the humans infected per unit time is$(\frac{C_{VH} a_V I_V}{N_H})S_H$.

Table 1: The state variables for the malaria model

S_H	Number of susceptible humans.
I_H	Number of infectious humans.
R_H	Number of recovered (immune and asymptomatic but slightly infectious) humans.
S_V	Number of susceptible mosquitoes.
I_V	Number of infectious mosquitoes.
N_H	Total human population.
N_V	Total mosquitoes population.

Table 2: The parameters for the malaria model

μ_H	The natural death rate for humans.
μ_V	The natural death rate for mosquitoes.
θ_H	Immigration rate of humans, Humans$\times Time^{-1}$.
θ_V	Immigration rate of mosquitoes, Mosquitoes$\times Time^{-1}$.
B_H	Per capita birth rate of newborns which are susceptible , Humans$\times Time^{-1}$.
B_V	Per capita birth rate of mosquitoes, Mosquitoes$\times Time^{-1}$.
ϕ	The recovery rate that infectious humans become susceptible.
φ	Per capita disease-induced death rate for humans, Humans$\times Time^{-1}$.
ρ	Per capita rate of loss of immunity for humans. $\frac{1}{\rho}$ is the average duration of the immune period, $Time^{-1}$.
β	Rate of the newborn's birth with Infection humans.
η	Per capita recovery rate for humans from the infectious state to the recovered state. $\frac{1}{\eta}$ is the average duration of the infectious period, $Time^{-1}$.
C_{VH}	Infectivity of the mosquito, defined as the probability that a bite by an infected mosquito on a susceptible human will transfer the infection to the human.
C_{HV}	Infectivity of an infectious non-immune person, defined as the probability that a bite by a susceptible mosquito on an infected human will transfer the infection to the mosquito.
a_V	The man-biting rate of the mosquitoes, defined as the average number of bites given to humans by each mosquito per unit time.

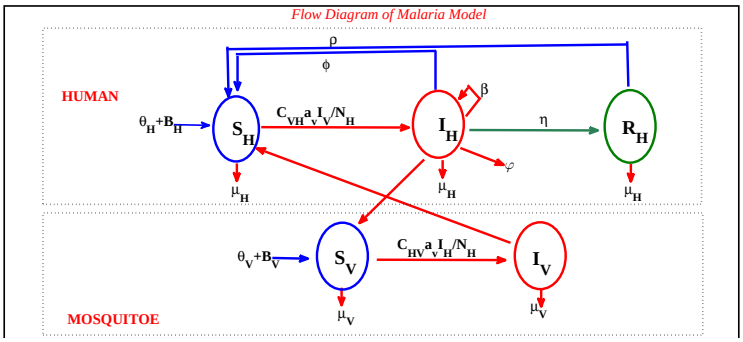

Figure 1: Schematic illustration of the model equation.

Similarly, assuming that the reservoir of possible infections from humans includes the classes I_H,then the mosquitoes infected per unit time is $(\frac{C_{HV}a_V I_H}{N_H})S_V$.

In Figure 1 we divide the human population into three classes, susceptible S_H, infectious I_H, and recovered (immune) R_H. People enter the susceptible class either through birth (or immigration) at a constant per capita rate B_H(or θ_H), or through the recovery from the infections class I_H by the rate ϕ (or from the recovery class R_H by the rate ρ). In our model, people from the susceptible class enter the infectious class at the rate $\frac{C_{VH}a_V I_V}{N_H}$, also people enter the infectious class by the infected newborns. After some time, the infectious humans recover with some immunity and move to the recovered class (at the rate η), After some period of time, they lose their immunity and return to the susceptible class (at the rate ρ). Humans leave the population through a natural death rate (at the rate μ_H), and through a per capita disease-induced death rate (at the rate φ). In addition, we divide the mosquito population into two classes, susceptible S_V and infectious I_V. Female mosquitoes (we do not include male mosquitoes in our model because only female mosquitoes bite humans), enter the susceptible class through birth (at the rate B_V) or through immigration (at the rate θ_V). The parasite (in the form of gametocytes) enters the mosquito with some probability when the mosquito bite infectious humans at the rate (C_{HV}), and once infected they progress through infectious, I_V classes. The effective contact rates between the two populations, which may be defined as the average number of contacts per day that will lead to the infection of one party if the other party was infectious, depends on a number of factors, the man biting rate of the mosquitoes, the transmission probabilities between the species and the number of individuals in both population. We briefly describe how the incident rates have been modelled.

The following assumptions are made in order to formulate the equations of the model:

- The development of malaria starts when the infectious female mosquito bites the human host or the newborn's birth with Infection.
- Mosquitoes bite human hosts randomly (independent of their infective status).
- Recovered human hosts have temporary immunity that can be lost and are again susceptible to reinfection.
- $B_H > \beta > \varphi$.

  This inequality means that the per capita birth rate of newborns which are susceptible is larger than the rate of the newborn's birth with infection humans, and the rate of the newborn's birth with infection humans is larger than the per capita disease-induced death rate for humans.
- Mosquitoes never recover from infection, as it is regulated by mortality of its individuals.
- Total human and mosquito populations are not constant.

Applying the assumptions, definitions of variables and parameters and description of terms above, the differential equations which describe the dynamics of malaria in the human and mosquito populations are formulated as shown below:

$$\begin{aligned}
\frac{dS_H}{dt} &= (\theta_H + B_H)N_H + \rho R_H + \phi I_H - \mu_H S_H - \frac{C_{VH}a_V I_V}{N_H}S_H,\\
\frac{dI_H}{dt} &= \frac{C_{VH}a_V I_V}{N_H}S_H - (\mu_H + \phi + \eta + \varphi)I_H + \beta I_H,\\
\frac{dR_H}{dt} &= \eta I_H - \rho R_H - \mu_H R_H,\\
\frac{dS_V}{dt} &= (\theta_V + B_V)N_V - \mu_V S_V - \frac{C_{HV}a_V I_H}{N_H}S_V,\\
\frac{dI_V}{dt} &= \frac{C_{HV}a_V I_H}{N_H}S_V - \mu_V I_V.
\end{aligned} \tag{2.1}$$

The total population sizes N_H and N_V can be determined by $S_H + I_H + R_H = N_H$, $S_V + I_V = N_V$, and

$$\begin{aligned} \frac{dN_H}{dt} &= (\theta_H + B_H - \mu_H)N_H + \beta I_H - \varphi I_H, \\ \frac{dN_V}{dt} &= (\theta_V + B_V - \mu_V)N_V, \end{aligned} \tag{2.2}$$

where all parameters in the model are assumed positive.

2.2. Mathematical model analysis

In this section we carry out qualitative analysis of the model to investigate stability of the steady states. First, we transform the system of populations into a system of proportions. The equations are obtained by differentiating each proportion with respect to time t. The proportions for the system are $s_h = \frac{S_H}{N_H}, i_h = \frac{I_H}{N_H}, r_h = \frac{R_H}{N_H}, s_v = \frac{S_V}{N_V}$ and $i_v = \frac{I_V}{N_V}$, in the classes $S_H, I_H, R_H, S_V,$ and I_V of the populations, respectively, and m is the female vector host ratio, defined as the number of female mosquitoes per human host [2, 15, 33]. This gives the following system of equations

$$\frac{ds_h}{dt} = (\theta_H + B_H)(1 - s_h) + \rho r_h + \phi i_h - C_{VH} a_v i_v m s_h + (\varphi - \beta) i_h s_h, \tag{2.3}$$

$$\frac{di_h}{dt} = C_{VH} a_v i_v m s_h - [\theta_H + \phi + \eta + \varphi + (B_H - \beta)] i_h + (\varphi - \beta) i_h^2, \tag{2.4}$$

$$\frac{dr_h}{dt} = \eta i_h - (\theta_H + B_H + \rho) r_h + (\varphi - \beta) i_h r_h, \tag{2.5}$$

$$\frac{ds_v}{dt} = (\theta_V + B_V)(1 - s_v) - C_{HV} a_V i_h s_v, \tag{2.6}$$

$$\frac{di_v}{dt} = C_{HV} a_V i_h s_v - (\theta_V + B_V) i_v. \tag{2.7}$$

Subject to the restriction $s_h + i_h + r_h = 1$ and $s_v + i_v = 1$, we note that the population sizes $N_H(t)$ and $N_V(t)$ do not appear in the system.

Now we intend to analyze and investigate the existence and stability of the associated equilibrium points. Assuming that all the parameters are non-negative, and solving for the equilibrium points by setting the right-hand sides of equations (2.3)-(2.7) to zero, the system takes the form as shown,

$$(\theta_H + B_H)(1 - s_h) + \rho r_h + \phi i_h - C_{VH} a_v i_v m s_h + (\varphi - \beta) i_h s_h = 0, \tag{2.8}$$

$$C_{VH} a_v i_v m s_h - [\theta_H + \phi + \eta + \varphi + (B_H - \beta)] i_h + (\varphi - \beta) i_h^2 = 0, \tag{2.9}$$

$$\eta i_h - (\theta_H + B_H + \rho) r_h + (\varphi - \beta) i_h r_h = 0, \tag{2.10}$$

$$(\theta_V + B_V)(1 - s_v) - C_{HV} a_V i_h s_v = 0, \tag{2.11}$$

$$C_{HV} a_V i_h s_v - (\theta_V + B_V) i_v = 0. \tag{2.12}$$

We express the solutions in terms of i_h for easy analysis of the steady states to obtain

$$s_h = \frac{[B_H + \theta_H + \rho + (\phi - \rho) i_h][B_V + \theta_V + C_{HV} a_V i_h]}{(B_H + \theta_H + \rho - (\varphi - \beta) i_h)(B_V + \theta_V + C_{HV} a_V i_h) + C_{HV} C_{VH} a_V^2 m i_h}, \tag{2.13}$$

$$r_h = \frac{\eta i_h}{B_H + \theta_H + \rho - (\varphi - \beta) i_h}, \tag{2.14}$$

$$s_v = \frac{\theta_V + B_V}{\theta_V + B_V + C_{HV} a_V i_h}, \tag{2.15}$$

$$i_v = \frac{C_{HV} a_V i_h}{\theta_V + B_V + C_{HV} a_V i_h}. \tag{2.16}$$

When we substitute (2.13)-(2.16) into (2.8), i_h can be determined.

3. Disease-free equilibrium point and reproductive number.

3.1. *stability of disease-free equilibrium* E_0

A disease-free equilibrium point is a steady-state solution of system (2.3)-(2.7) where there is no disease, namely $s_h^* \text{ and } s_v^* > 0$, and all other variables $i_h^*, i_v^*, r_h^* = 0$, $E_0 = (s_h^*, i_h^*, r_h^*, s_v^*, i_v^*) = (1, 0, 0, 1, 0)$.

The local stability of this point is established from the Jacobian of the system (2.3)-(2.7) evaluated at E_0. The Jacobian matrix of the system is given by

$$J_E = \begin{pmatrix} J_1 & J_2 \end{pmatrix}, \tag{3.1}$$

where

$$J_1 = \begin{pmatrix} -[B_H + \theta_H + C_{VH}a_V i_v m - (\varphi - \beta)i_h] & \phi + (\varphi - \beta)s_h \\ C_{VH}a_V i_v m & -[\theta_H + \phi + \eta + \varphi + (B_H - \beta) - 2(\varphi - \beta)i_h] \\ 0 & \eta + (\varphi - \beta)r_h \\ 0 & -C_{HV}a_V s_v \\ 0 & C_{HV}a_V s_v \end{pmatrix}$$

$$J_2 = \begin{pmatrix} \rho & 0 & -C_{VH}a_V m s_h \\ 0 & 0 & C_{VH}a_V m s_h \\ -[\theta_H + B_H + \rho - (\varphi - \beta)i_h] & 0 & 0 \\ 0 & -(\theta_V + B_V + C_{HV}a_V i_h) & 0 \\ 0 & C_{HV}a_V i_h & -(\theta_V + B_V) \end{pmatrix}.$$

The Jacobian matrix (3.1) evaluated at $E_0 = (s_h^*, i_h^*, r_h^*, s_v^*, i_v^*) = (1, 0, 0, 1, 0)$ gives

$$J_{E_0} = \begin{pmatrix} -(B_H + \theta_H) & \phi + (\varphi - \beta) & \rho & 0 & -C_{VH}a_V m \\ 0 & -(\theta_H + \phi + \eta + \varphi + (B_H - \beta)) & 0 & 0 & C_{VH}a_V m \\ 0 & \eta & -(\theta_H + B_H + \rho) & 0 & 0 \\ 0 & -C_{HV}a_V & 0 & -(\theta_V + B_V) & 0 \\ 0 & C_{HV}a_V & 0 & 0 & -(\theta_V + B_V) \end{pmatrix}. \tag{3.2}$$

As the first column contains only the diagonal term, then this diagonal term forms one eigenvalue of the Jacobian $\lambda_1 = -(B_H + \theta_H)$. Similarly, the other eigenvalues are $\lambda_2 = -(\theta_H + B_H + \rho)$ and $\lambda_3 = -(\theta_V + B_V)$. Note that the eigenvalues $\lambda_1, \lambda_2 \text{ and } \lambda_3$ are all negative. Remaining two eigenvalues can be obtained from the eigenvalues of the 2×2 block matrix given by

$$\Lambda = \begin{pmatrix} -[\theta_H + \phi + \eta + \varphi + (B_H - \beta)] & C_{VH}a_V m \\ C_{HV}a_V & -(\theta_V + B_V) \end{pmatrix},$$

whose trace and determinant are given by

$$\begin{aligned} Tr\Lambda &= -[\theta_H + \theta_V + B_V + \phi + \eta + \varphi + (B_H - \beta)] < 0, \\ Det\Lambda &= [\theta_H + \phi + \eta + \varphi + (B_H - \beta)](\theta_V + B_V) - C_{VH}C_{HV}a_V^2 m \\ &= [(\theta_H + \phi + \eta + \varphi + (B_H - \beta))(\theta_V + B_V)][1 - \tfrac{C_{VH}C_{HV}a_V^2 m}{[(\theta_H + \phi + \eta + \varphi + (B_H - \beta))(\theta_V + B_V)]}] \\ &= [(\theta_H + \phi + \eta + \varphi + (B_H - \beta))(\theta_V + B_V)][1 - R_0], \end{aligned}$$

where

$$R_0 = \frac{C_{VH}C_{HV}a_V^2 m}{[\theta_H + \phi + \eta + \varphi + (B_H - \beta)](\theta_V + B_V)}.$$

Thus, E_0 is locally asymptotically stable if and only if $R_0 < 1$, and we have thus established the following theorem.

Theorem 3.1. *The disease-free equilibrium* E_0 *is locally asymptotically stable if* $R_0 < 1$, *and unstable if* $R_0 > 1$.

The quantity R_0 is the basic reproduction number of the disease. It represents the average number of new infections produced by one infected individual. It is a useful quantity in the study of a disease as it sets the threshold for its establishment. If $R_0 < 1$, then the disease-free equilibrium is locally stable. The reproduction number depends on the product of the transmission coefficients, $a_V C_{VH} m$ and $a_V C_{HV}$, the average residence time $\frac{1}{(\theta_H+\phi+\eta+\varphi+(B_H-\beta))}$ in the infective class and the average life span $\frac{1}{(\theta_V+B_V)}$ of the mosquito. It is also dependent on the rate of acquisition of immunity, rate of recovery from infection ϕ, disease induced mortality rate φ, and the rate of the newborn's birth with infection β. We can quantify that higher values of coefficients of transmission between humans and mosquito vectors m, a_V, C_{VH} and C_{HV} can allow the establishment of the disease. From the expression of R_0 and the numerical simulations in Section 4, we can see that R_0 is sensitive to the parameter β. So the control of congenital malaria will play a important role in the transmission of the disease. There is need to pay attention to processes that can limit the spread of the disease such as protection of the vulnerable groups special for the pregnant woman from human mosquito interaction by use of treated mosquito nets, providing prompt and effective treatment to those who are sick.

Pregnancy is a time when complex physiological phenomena occur in order to adapt the organism to a new environment. This adaptation includes a certain degree of immune suppression, which leads to an increased susceptibility of the pregnant woman to a number of pathologies in which immune regulation plays an important role. Parasitic infections are among those infections whose frequency and severity is increased during pregnancy.

3.2. *Existence and stability of endemic equilibrium* E_1

We first discuss the existence and stability of endemic equilibrium E_1 when $R_0 > 1$.

Let $r_h = 1 - s_h - i_h$ and $s_v = 1 - i_v$, then we can reduce system (2.3)-(2.7) to a 3-dimensional system. We solve for the equilibrium E_1 by setting the right-hand sides of (2.3), (2.4) and (2.7) to zero

$$(\theta_H + B_H + \rho)(1 - s_h^*) + (\phi - \rho)i_h^* - C_{VH}a_v i_v^* m s_h^* + (\varphi - \beta)i_h^* s_h^* = 0, \tag{3.3}$$

$$C_{VH}a_v i_v^* m s_h^* - [\theta_H + \phi + \eta + \varphi + (B_H - \beta)]i_h^* + (\varphi - \beta)i_h^{*2} = 0, \tag{3.4}$$

$$C_{HV}a_V i_h^*(1 - i_v^*) - (\theta_V + B_V)i_v^* = 0, \tag{3.5}$$

From (3.4) and (3.5) we have

$$s_h^* = \frac{[-\beta + B_H + \theta_H + \phi + \eta + \varphi + (\beta - \varphi)i_h^*](\theta_V + B_V + C_{HV}a_V i_h^*)}{C_{VH}C_{HV}a_V^2 m}, \tag{3.6}$$

$$i_v^* = \frac{C_{HV}a_V i_h^*}{\theta_V + B_V + C_{HV}a_V i_h^*}. \tag{3.7}$$

Substituting (3.6) and (3.7) into (3.3), we have $i_h^{*3} + A_1 i_h^{*2} + A_2 i_h^* + A_3 = 0$ where

$$\begin{aligned}
A_1 =& \frac{B_V + \theta_V}{a_V C_{HV}} + \frac{1}{(\beta - \varphi)}[m a_V C_{HV}(1 + \frac{a_V C_{HV}}{R_0(B_V + \theta_V)}) + \rho + B_H + \theta_H],\\
A_2 =& \frac{1}{(\beta - \varphi)^2 a_V C_{HV}}\{\frac{m a_V^2 C_{HV} C_{VH}}{R_0(B_V + \theta_V)}[(\beta - \varphi)(B_V + \theta_V) + a_V C_{HV}(B_H + \theta_H + \rho) +\\
& m a_V^2 C_{HV} C_{VH}] + (\beta - \varphi)(B_V + \theta_V)(B_H + \theta_H + \rho) + m a_V^2 C_{HV} C_{VH}(\rho - \phi)\},\\
A_3 =& \frac{a_V m C_{VH}}{(\beta - \varphi)^2}(\frac{1}{R_0} - 1)(B_H + \theta_H + \rho).
\end{aligned}$$

With the assumptions $B_H > \beta > \varphi$ there are $A_1 > 0$ and $A_2 > 0$. For the existence of endemic equilibrium $E_1 = (s_h^*, i_h^*, i_v^*)$ its coordinates should satisfy the conditions $1 > s_h^*, i_h^*, i_v^* > 0$.

Denote $F(i_h) = i_h^3 + A_1 i_h^2 + A_2 i_h + A_3$, then

$$\begin{aligned} F(0) =& A_3 < 0, \\ F(1) =& 1 + A_1 + A_2 + A_3 \\ =& \frac{1}{(\beta-\varphi)^2 a_V C_{HV}} [m a_V^2 C_{HV} C_{VH} + (B_V + \theta_V + a_V C_{HV})(B_H \\ & + \theta_H + \eta + \phi)(\theta_H + \rho + \varphi + B_H - \beta)] > 0, \\ F'(i_h) =& 3i_h^2 + 2A_1 i_h + A_2 > 0. \end{aligned}$$

So $F(i_h^*) = 0$ has an unique root $i_h^* \in (0,1)$ when $R_0 > 1$.

Now we turn to discuss the situation when $R_0 < 1$. In this situation, we can see that $F(0) = A_3 > 0$ and $F(1) = 1 + A_1 + A_2 + A_3 > 0$. Note that $F'(i_h) = 3i_h^2 + 2A_1 i_h + A_2 > 0$, hence $F(i_h) = 0$ has no real root in $(0,1)$. Then we get the following theorem.

Theorem 3.2. *If $R_0 > 1$, there exists an unique endemic equilibrium E_1. If $R_0 < 1$, there exists no endemic equilibrium.*

4. Numerical simulations

In this section, we present the numerical analysis of the model to illustrate the transmission of malaria disease. The ode45 and fsolve functions in Matlab were used in this study, the parameter values in Table 3 are used in the simulations to illustrate the behavior of the model.

Table 3: The model parameters for the estimation

μ_H	0.00004/day
μ_V	0.05/day
θ_H	0.0005
θ_V	0.00002
B_H	0.0015875/day
B_V	0.071/day
ϕ	0.0022/day
φ	0.00333/day
ρ	0.000017/day
β	0 0.001 0.002 0.003
η	0.00019/day
a_V	0.029
C_{VH}	0.75
C_{HV}	0.75

At time $t = 0$, we have the following initial conditions in the proportions:

$$(s_h, i_h, r_h, s_v, i_v) = (0.990, 0.010, 0, 0.9, 0.1),$$

we study the dynamical behavior of the model for variation of the $\beta = 0, 0.001, 0.002, 0.003$, the general behavior of the model is shown in Figures 2-5, we simulate approximately 2000 d, from observation of the figures we conclude that as when β is increased from 0 to 0.03 there is a corresponding increase in the number of infectious humans and infectious mosquitoes, for any very small changes in β we have a big increases infectious classes.

In Figure 6, we can see the relationship between the infection human and variation of the β. Finally, for showing the effect of newborn's birth with infection rate to the basic reproduction number, we give

the relation between R_0 and beta (Figure 7). From Figure 7, we know that R_0 is increasing with respect to the β rate. These numerical results support the results earlier obtained analytically that the endemic equilibrium is stable.

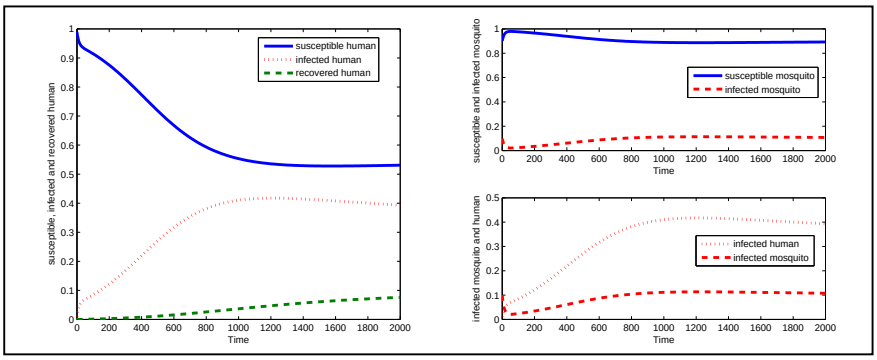

Figure 2: $\beta = 0$ and $R_0 = 1.7284$, Endemic patterns of the susceptible, infected and recovered human populations , and the susceptible and infected mosquito populations. Starting at the initial conditions $(s_h, i_h, r_h, s_v, i_v) = (0.990, 0.010, 0, 0.9, 0.1)$ having i_h and i_v values and particular baseline parameters, the system (2.3)-(2.7) approaches the endemic point (0.5308 , 0.3934 , 0.0758 , 0.8924 , 0.1076) , and the relationship between i_h and i_v.

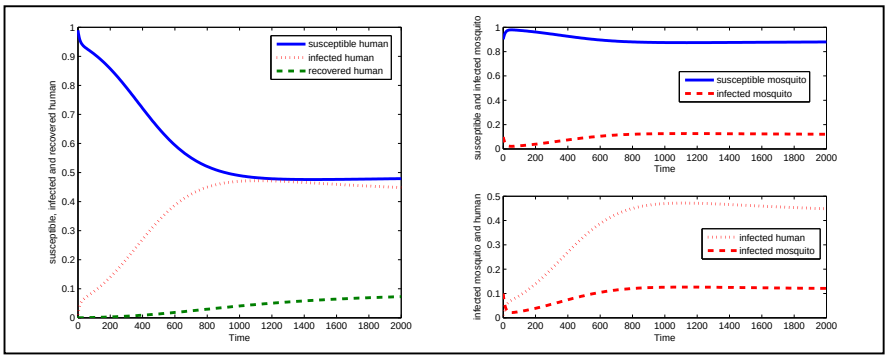

Figure 3: $\beta = 0.001$ and $R_0 = 1.9861$, the system (2.3)-(2.7) approaches the endemic point (0.4790, 0.4484, 0.0727, 0.8793, 0.1207) , and see the relationship between i_h and i_v.

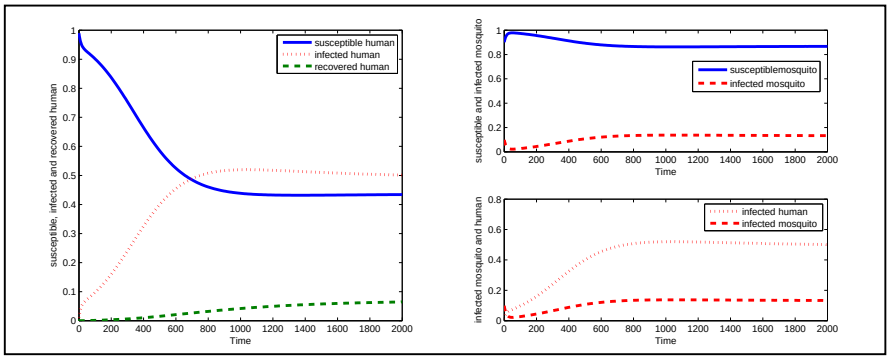

Figure 4: $\beta = 0.002$ and $R_0 = 2.3341$, the system (2.3)-(2.7) approaches the endemic point (0.4342, 0.5013, 0.0645, 0.8669, 0.1331), and see the relationship between i_h and i_v.

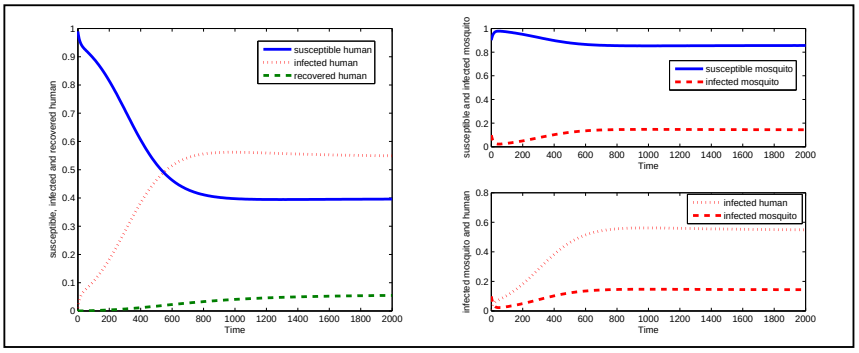

Figure 5: $\beta = 0.003$ and $R_0 = 2.8299$, the system (2.3)-(2.7) approaches the endemic point (0.3959, 0.5492, 0.0549, 0.8560, 0.1440), and see the relationship between i_h and i_v.

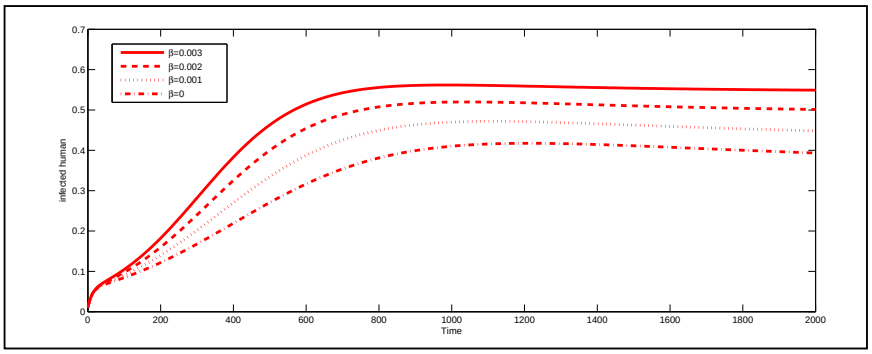

Figure 6: the variation of infected human population with time for different values of β.

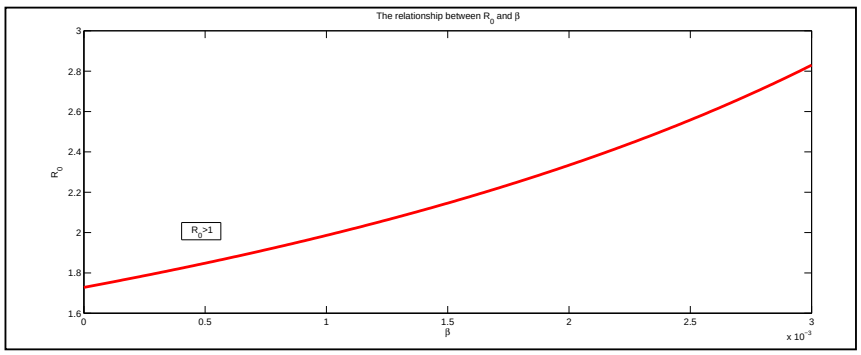

Figure 7: When rate of the newborn's birth with infection number gets larger the value of the reproductive number gets larger.

5. Discussion

Congenital malaria is the least known manifestation of malaria and a very neglected area of research. Most of the existing information is limited to case reports in children born to non-immune women. With the use of molecular techniques, congenital infection is being increasingly detected among infants born to semi-immune women in endemic countries. However, many gaps in knowledge remain. The mechanisms and timing of infection are unclear. Furthermore, there is a lack of information on the impact of congenital malaria infection on the subsequent risk of malaria and general morbidity in the infant. With great development mathematical models of malaria, but it has not been studied the vertical transmission between human, more research is needed in order to establish adequate preventive and management recommendations to avoid this consequence of malaria in pregnancy. We have developed our model that includes the impact vertical transmission (congenital malaria) in this study. In this paper we have

derived and analyzed a mathematical model of 5-dimensional system of a nonlinear mathematical model for spread of malaria which incorporates and include the infection newborn to better understand the transmission and spread of malaria.

To see the effect of vertical transmission (congenital malaria), we change the parameter β and keep all other parameters as in Figures 2-5. The variation of infected human population i_h with time for different β is shown in Figure 6. It is observed that for large value of β the equilibrium level of i_h is high, shown in Figure 7 when rate of the newborn's birth with infection number gets larger the value of the reproductive number gets larger becomes very difficult to control the spread of the disease, it becomes more difficult to control the infection of the population. Equilibria of the model are found and stability of these equilibria are discussed the disease free equilibrium is locally asymptotically stable whenever $R_0 < 1$ and is unstable for $R_0 > 1$.

Pregnant women in particular are more likely to have increased susceptibility to malaria infection during this stage, and would suffer from severely disease with the risk of death. Because pregnancy increases a woman's susceptibility to malaria infections, and their protective acquired immunity is lost in low prevalence situations. So it is necessary for pregnant women to be protected from exposure of the infection entirely, therefore, targeted communication will be required among adult populations to explain the increased risk of severe illness and deaths, as well as stronger epidemics management, with surveillance systems to promptly detect risks of epidemics and to rapidly deploy adequate response systems. We see that there is a direct increases the spread of the malaria in the human community, especially with the newborns in the lower and inexperienced immunity systems, elucidating this problem will help to rationalize and target the malaria control measures best suited to protect both mother and infant during pregnancy. Also note Strategies that reduce mosquito-human contacts, such as the use of insecticide-treated bed nets and indoor residual spraying (IRS). These strategies would be the most effective in reducing initial transmission.

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