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The impact of malaria transmission from mother to the newborn on the spread of malaria

Ghoul Rafia[1*], Jing He[1], Sana Djaidja[3], Ebrahim As-Shareef[3]

[1] Department of Computer Science and Technology, College of Computer Science and Engineering, Hunan University, Changsha 410000, China

[2] Department of Medicine, Faculty of Medicine of Algiers - Mohamed Maherzi (Ex Laperrine), Algiers 16000, Algeria
[3] Department of Mathematics, School of Mathematics and Statistics, Central China Normal University, Wuhan 430000, China

Corresponding Author Email: Tigre.eco@live.fr

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ABSTRACT

The main objective of this paper is to develop a mathematical model to study the dynamic of malaria transmission for the human and mosquito populations. In this study we have clarified the significant impact of congenital malaria (vertical transmission of malaria from mother to baby before or during birth). We see the direct effects of congenital malaria on the spread of malaria and impact it on the basic reproduction number. In our model the human population is divided into three classes and the mosquito population is divided into two classes, our appropriate model of 5-dimensional nonlinear system which incorporates and includes the infection newborn shows that the disease-free equilibrium is globally asymptotically stable if 〖R〗_0<1 and if 〖R〗_0>1 the endemic equilibrium is locally asymptotically stable proved by Routh- Hurwitz criterion. Our numerical simulations and graphical results conform the analysis predictions.

1. INTRODUCTION

Malaria remains one of the most prevalent and lethal human infection worldwide. According to the World Malaria report (WMR) of 2016 published by (WHO), there were 212 million new cases of malaria worldwide, with 90% of global cases occurred in African Region followed by 7% for the South-East Asia Region and 2% for the Eastern Mediterranean Region [1] and accounted for an estimated 429 000 deaths most of them are young children. In Africa for example each 30 seconds a child dies from malaria disease [2]. In the period 2010 to 2015, malaria rates reduced by 21% globally and in the African Region and malaria mortality rates reduced by an estimation of 29% globally and by 31% in the African Region [1].

Malaria spreads in three ways; in case of infected mother it can be transmitted to the children through the parasitized red cells either transplacentally or during labor, these two cases can lead to newborn birth with malaria disease this called congenital malaria, however most of the time it is caused because of the bite of an infected female Anopheles mosquito [3]. Malaria congenital doesn't have an exact definition, some of them defined it as the illness caused by malaria parasites' placental transmission or transmitting during birth through the birth canal, others as the presence of asexual malaria parasites (P. falciparum, P. vivax, and mixed infections) in the erythrocytes of the infant aged less than seven days [4]. In the endemic areas the congenital malaria has been frequent in the non-immune population .It had been first described in 1876, till recently, it was known to be a rare condition[5].Moreover, cord parasitaemia, early neonatal malaria and neonatal malaria can be caused by perinatal malaria infection .In case that the placenta is infected with malaria parasites, transplacental transmission of the parasites can occur, although the newborn may remain asymptomatic and healthy[6]. Transplacental transmission of *P. falciparum* has been well described, and the reported frequency of this event in babies born in malaria-exposed pregnancies has ranged from 0 percent to more than 25 percent [7]. Mathematical modeling of malaria began in1911with Ross model [8]. In 1957 Macdonalds described many extensions in his book [9]. The first models were two-dimensional with one variable representing humans and the other representing mosquitoes. In [10] Dietz, Molineaux and Thomas propose a new model that include an acquired immunity. Anderson and May [11], Aron and May [12] and Koella [13], have written some good reviews on the mathematical modeling of malaria. Further, Some papers have also included environmental effets [14-16] the spread of resistance to drugs [17] and the evolution of immunity [18].Recently, Ngwa and Shu [19] and Ngwa [20] proposed an ordinary differential equation(ODE) compartmental model for the Spread of malaria that involve variable human and mosquito populations, with a susceptible-exposed-infectious-recovered-susceptible(SEIRS) pattern for human population and a susceptible-exposed-infectious (SEI) pattern for mosquitoes population, Vector control has been an important part of the global malaria control strategy Several vector control intervention programs have been implemented and have proved to be effective in providing protection to humans. Studies show that larviciding suppresses the number of malaria transmitting mosquitoes in malaria places [21-23]. However, larval control can only be effective if larval habitats are Limited and well defined. In [24] Ross-Macdonald suggests in his malaria transmission model that control methods that reduce adult mosquitoes' longevity can achieve greater malaria reduction than strategies that target larval stages. Researchers have included some intervention measures in the past in order to reduce malaria transmission such as: Insecticide-treated nets (ITNs), long-lasting insecticide treated

nets (LLINs) and Insecticide residual spray (IRS). However, increased insecticide resistance in vectors, together with outdoor transmission, has limited the efficacy of the ITN scaling-up efforts. Observations on longitudinal changes in ITN coverage and its impact on malaria transmission allow policy makers to make informed adjustments to control strategies. Authors in [25] has found that ITN ownership increased from an average of 18% to 85% in the period of 2003-2015, proved by an analyze and survey on ITN ownership, malaria parasite prevalence and malaria vector population dynamics done in seven sentinel sites in western Kenya for that period.

Since 1937, many researches use Biological control and especially larvivorous fish in order to control mosquitoes [26]. They have implemented many mathematical models to study different situations and stages of malaria transmission and the relationship between humans and mosquitoes populations. Methods and stages of control have impacted the factor of travel and movement in human and mosquitoes populations in the spread of the malaria disease. Researchers have been widely focusing and working on the methods and models of mosquito control since they discovered that it's the main factor of malaria transmission through the female Anopheles mosquitoes. Further, studies have been done on the methods of biological control for mosquitoes of the adult stages and water also hybridization methods as well. However, there is no research implementing the fact of malaria transmission from mother to children with the knowledge.

Malaria infections cases are mostly children at various ages, particularly less than 5 years. In this thesis, we have tried to highlight and discuss the impact of congenital malaria (vertical transmission of malaria from mother to baby before or during birth). We have built a mathematical model and we have insert a vertical transmission of malaria from human to human in this model, and to analyze the spread of malaria in the community. 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 and impact the vertical transmission malaria on the basic reproduction number. We present the numerical analysis of the model to illustrate the transmission of malaria disease and the impact of vertical transmission on the spread of malaria. We show that congenital malaria can help the disease to become endemic.

2. MATHEMATICAL MODEL DESCRIPTION AND ANALYSIS

2.1 Mathematical model description

Table 1. Description of malaria model state variables

S_H	Number of susceptible humans (The host) to malaria infection.
I_H	Number of infected humans with malaria disease.
R_H	Number of recovered humans to malaria infection.
S_V	Number of susceptible mosquitoes.
I_V	Number of infectious mosquitoes.
N_H	Total human population.
N_V	Total mosquito population.

Table 2. Description of malaria model parameters

μ_H	The natural death rate for humans.
μ_V	The natural death rate for mosquitoes.
B_1	Per capital susceptible newborns birth rate, Humans $\times Time^{-1}$.
B_V	Per capital susceptible Mosquitoes birth rate, Mosquitoes $\times Time^{-1}$.
φ	Per capital recovery rate for humans from the infectious state to the recovered state
ϕ	Per capital disease-induced death rate for humans, Humans $\times Time^{-1}$.
ρ	Per capital rate of loss of immunity in human population, such that $\frac{1}{\rho}$ is the average duration of the immune period, $Time^{-1}$.
B_2	Rate of the newborn's birth with Infection humans.
η	Per capital recovery rate for humans from the infectious state to the recovered state. $\frac{1}{\eta}$ is the average duration of the infectious period, $Time^{-1}$.
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.
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.
m	Female vector host ratio, defined as the number of female mosquitoes per human host

In order to study the spread of malaria in humans(The host) population and mosquito (the vector) population with the impact of malaria transmission from mother to children,we formulate a mathematical model that divide the total Human population at time t denoted by $N_H(t)$ into three epidemiological classes: Susceptible class S_h, Infectious class I_h and Recovered class R_h and divide the total mosquito population at time t denoted by $N_V(t)$ into two epidemiological classes: Susceptible class S_v and Infectious class I_v.Mosquito population does not include recovered class as mosquitoes never recover from infection,that is,their infective period ends with their death due to their relatively

short life-cycle.The state variables and parameters used for malaria transmission model are shown in (Table (1)) and (Table (2)) respectively. In the model the parameters are strictly positive except ϕ nonnegative. In order to maintain a stable positive mosquito population, we suppose that the mosquito birth rate is greater than the density-independent, $B_V > \mu_V$.

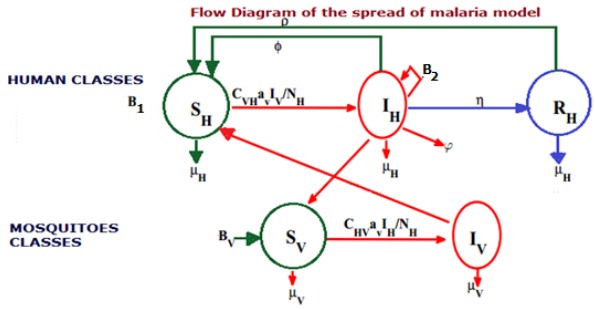

Figure 1. Schematic of the model

In our model we assume the following assumptions in order to formulate the equations of the model:

1) The two population $N_H(t)$ and $N_V(t)$ are variable.

2) The malaria disease starts the development when the infected female mosquito bites the human host or when the newborn's birth with Infection.

3) Mosquitoes bite human randomly.

4) $\eta > \rho$ and $B_1 + \rho > |B_2 - \phi|$.

The following system of nonlinear ordinary differential equations describe the dynamics of malaria in the human and mosquito populations

$$\begin{cases} \dfrac{dS_H}{dt} = B_1 N_H + \rho R_H + \varphi I_H - \mu_H S_H - \dfrac{C_{VH} a_V I_V}{N_H} S_H, \\ \dfrac{dI_H}{dt} = \dfrac{C_{VH} a_V I_V}{N_H} S_H - (\mu_H + \varphi + \eta + \phi) I_H + B_2 I_H, \\ \dfrac{dR_H}{dt} = \eta I_H - \rho R_H - \mu_H R_H, \\ \dfrac{dS_V}{dt} = B_V N_V - \mu_V S_V - \dfrac{C_{HV} a_V I_H}{N_H} S_V, \\ \dfrac{dI_V}{dt} = \dfrac{C_{HV} a_V I_H}{N_H} S_V - \mu_V I_V, \end{cases} \quad (1)$$

We determine the total population sizes N_H and N_V by $N_H = S_H + I_H + R_H$, $N_V = S_V + I_V$,

$$\begin{cases} \dfrac{dN_H}{dt} = (B_1 - \mu_H) N_H + B_2 I_H - \phi I_H, \\ \dfrac{dN_V}{dt} = (B_V - \mu_V) N_V, \end{cases} \quad (2)$$

In the model we assume that all parameters are positive.

2.2 Mathematical model analysis

To examine the behavior of this model we transform the system of populations into a system of proportions to investigate stability of the steady states. We obtain the equations by differentiating each proportion at time t .

$$s_h = \frac{S_H}{N_H}, i_h = \frac{I_H}{N_H}, r_h = \frac{R_H}{N_H}, s_v = \frac{S_V}{N_V} \text{ and } i_v = \frac{I_V}{N_V}.$$

These fractions give the following system :

$$\begin{cases} \dfrac{ds_h}{dt} = B_1(1-s_h) + \rho r_h + \varphi i_h - C_{VH} a_v i_v m s_h + (\phi - \beta_2) i_h s_h, \\ \dfrac{di_h}{dt} = C_{VH} a_v i_v m s_h - [B_1 + \varphi + \eta + (\phi - B_2)] i_h + (\phi - B_2) i_h^2, \\ \dfrac{dr_h}{dt} = \eta i_h - (B_1 + \rho) r_h + (\phi - B_2) i_h r_h, \\ \dfrac{ds_v}{dt} = (B_V)(1-s_v) - C_{HV} a_V i_h s_v, \\ \dfrac{di_v}{dt} = C_{HV} a_V i_h s_v - (\theta_V + B_V) i_v. \end{cases} \quad (3)$$

The all parameters are assumed non-negative in order to analyze and investigate the existence and stability of the associated equilibrium points. We set the right-hand sides of the equation in system (3) to zero.

$$\begin{cases} B_1(1-s_h) + \rho r_h + \varphi i_h - C_{VH} a_v i_v m s_h + (\phi - B_2) i_h s_h = 0, \\ C_{VH} a_v i_v m s_h - [B_1 + \varphi + \eta + (\phi - B_2)] i_h + (\phi - B_2) i_h^2 = 0, \\ \eta i_h - (B_1 + \rho) r_h + (\phi - B_2 b) i_h r_h = 0, \\ B_1(1-s_v) - C_{HV} a_V i_h s_v = 0, \\ C_{HV} a_V i_h s_v - B_V i_v = 0 \end{cases} \quad (4)$$

For easy analysis of the steady states we express the solutions in terms of i_h and we obtain:

$$\begin{cases} s_h = \dfrac{[B_1 + \rho + (\varphi - \rho) i_h](B_V + C_{HV} a_v i_v)}{[B_1 + \rho - (\varphi - B_2) i_h](B_V + C_{HV} a_v i_v) + (C_{HV} C_{VH} a_v^2 m i_h)}, \\ r_h = \dfrac{\eta i_h}{B_1 + \rho - (\varphi - B_2) i_h}, \\ s_v = \dfrac{B_V}{B_V + C_{HV} a_v i_h}, \\ i_v = \dfrac{C_{HV} a_v i_h}{B_V + C_{HV} a_v i_h} \end{cases} \quad (5)$$

3. DISEASE-FREE EQUILIBRIUM POINT AND REPRODUCTION NUMBER

3.1 Steady stability of disease-free equilibrium E_0

We define a disease-free equilibrium point in our study as a steady-state solution of the system (3) where there is no disease, by assuming s_{h0} and $s_{v0} \geq 0$, and all other variables, $i_{h0}, i_{v0}, r_{h0} = 0$, now we have the disease-free equilibrium point

$$E_0 = (s_{h0}, i_{h0}, r_{h0}, s_{v0}, i_{v0}) = (1,0,0,1,0).$$

Theorem 1 If $R_0 \leq 1$ then the disease-free equilibrium E_0 is locally asymptotically stable, if $R_0 > 1$ then this point is

unstable .

Proof: From the Jacobian matrix of the system (5) at E_0 given as above the local stability of this point is established .

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

$J_1 =$

$$\begin{pmatrix} -[B_1+\rho+C_{VH}a_V i_v m-(\phi-B_2)i_h] & \varphi+(\phi-B_2)s_h \\ C_{VH}a_V i_v m & -[B_1+\varphi+\eta+(\phi-B_2)-2(\phi-B_2)i_h] \\ 0 & \eta+(\phi-B_2)r_h \\ 0 & -C_{HV}a_V s_v \\ 0 & C_{HV}a_V s_v \end{pmatrix}$$

$J_2 =$

$$\begin{pmatrix} \rho & 0 & -C_{VH}a_V m s_h \\ 0 & 0 & C_{VH}a_V m s_h \\ -[B_1+\rho-(\phi-B_2)i_h] & 0 & 0 \\ 0 & -(B_V+-C_{HV}a_V i_h) & 0 \\ 0 & C_{HV}a_V i_h & -B_V \end{pmatrix}$$

Let's evaluate the Jacobian matrix J_E at

$$E_0 = (s_{h0}, i_{h0}, r_{h0}, s_{v0}, i_{v0}) = (1,0,0,1,0)$$

$J_{E_0} =$

$$\begin{pmatrix} -B_1 & \varphi+(\phi-B_2) & \rho & 0 & -C_{VH}a_V m \\ 0 & -(B_1+\varphi+\eta+\phi-B_2) & 0 & 0 & C_{VH}a_V m \\ 0 & \eta & -(B_V+\rho) & 0 & 0 \\ 0 & C_{HV}a_V & 0 & -B_V & 0 \\ 0 & -C_{HV}a_V & 0 & 0 & -B_V \end{pmatrix}$$

We can see that first column contains only the diagonal term, then this diagonal term forms one eigenvalue of the Jacobian $\lambda_1 = -B_H$ Similarly, the other eigenvalues are $\lambda_2 = -(B_1+\rho)$ and $\lambda_3 = -B_V$. Note that the eigenvalues λ_1, λ_2 *and* λ_3 are all negative. Remaining two eigenvalues can be obtained from the eigenvalues of the 2×2 block matrix given by

$$\Lambda = \begin{pmatrix} -[B_1+\varphi+\eta+(\phi-B_2)] & C_{VH}a_V m \\ C_{HV}a_V & -B_V \end{pmatrix},$$

whose trace and determinant are given by

$$Tr\Lambda = -[B_1+\theta_V+B_V+\varphi+\eta+(\phi-B_2)] < 0,$$

$$\begin{aligned} Det\Lambda &= [B_1+\varphi+\eta+(\phi-B_2)]B_V - C_{VH}C_{HV}a_V^2 m \\ &= [(B_1+\varphi+\eta+(\phi-B_1))B_V]* \\ &[1-\frac{C_{VH}C_{HV}a_V^2 m}{[(B_1+\varphi+\eta+(\phi-B_2))B_V]}] \\ &= [(B_1+\varphi+\eta+(\phi-B_2))B_V][1-R_0], \end{aligned}$$

where

$$R_0 = \frac{C_{VH}C_{HV}a_V^2 m}{[B_1+\varphi+\eta+(\phi-B_2)]B_V}.$$

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

3.2 The reproduction number R_0

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 $\frac{1}{(B_1+\varphi+\eta+(\phi-B_2))}$ in the infective class and the average life span $\frac{1}{B_V}$ of the mosquito. It is also dependent on the rate of acquisition of immunity η, rate of recovery from infection φ, disease induced mortality rate ϕ, and the rate of the newborn's birth with infection B_2

So hence by the Routh-Hurwitz, the disease-free equilibrium E_0 is locally asymptotically stable.

The disease free equilibrium point explains the final behavior of the disease when the community is free of the disease, it shows the final reachable situations for this disease, even though there could be infinitely many different initial distributions of malaria in a community these equilibrium points are the final reachable situations.

3.3 Existence and stability of endemic equilibrium E^*

Let $r_h^* = 1 - s_h^* - i_h^*$, *and* $s_v^* = 1 - i_v^*$, That we will get 3-dimensional system instead of 5. We will discuss the equilibrium point at the endemic level E^* . The endemic equilibrium point is the final reachable situation, even though there could be infinitely many different initial distributions of malaria in a community.

Lets now solve the system (3) for the equilibrium E^* ,we set the right-hand of the system to zero

$$\begin{cases} (B_1+\rho)(1-s_h^*)+(\varphi-\rho)i_h^* - C_{VH}a_v i_v^* m s_h^* + (\phi-B_2)i_h^* s_h^* = 0, \\ C_{VH}a_v i_v^* m s_h^* - [B_1+\varphi+\eta+(\phi-\beta)]i_h^* + (\phi-B_2)i_h^{*2} = 0, \\ C_{HV}a_v i_h^*(1-i_v^*) - B_V i_v^* = 0, \end{cases} \quad (7)$$

From (7) we have

$$\begin{cases} s_h^* = \dfrac{[B_1 - B_2 + +\varphi + \eta + \phi + (B_2 - \phi)i_h^*](C_{HV}a_v i_h^* + B_V)}{C_{VH}C_{VH}a_v^2 m}, \\ i_v^* = \dfrac{C_{HV}a_v i_h^*}{C_{HV}a_v i_h^* + B_V}, \end{cases} \tag{8}$$

Substituting it into (7) we have $i_h^{*3} + A_1 i_h^{*2} + A_2 i_h^* + A_3 = 0$ where

$$\begin{aligned} A_1 &= \frac{1}{(B_2-\phi)a_V C_{HV}}[(B_2-\phi)B_V + ma_V^2 C_{HV}C_{VH} \\ &+ a_V C_{HV}(\varphi+\eta+\rho+\phi-B_2+2B_1)] \\ &= \frac{B_V}{a_V C_{HV}} + \frac{1}{(B_2-\phi)}[ma_V C_{HV}(1+\frac{a_V C_{HV}}{R_0 B_V}) + \rho + B_1], \end{aligned}$$

$$\begin{aligned} A_2 &= \frac{1}{(B_2-\phi)^2 a_V C_{HV}}[(B_2-\phi)B_V(\varphi+\eta+\phi-B_2+2(B_1+\rho) \\ &+ a_V C_{HV}B_V(\varphi+\eta+\phi-B_2+B_1)(B_1+\rho) \\ &+ ma_V^2 C_{HV}C_{VH}(\varphi+\eta+\phi-B_2+B_1+\rho-\varphi)] \\ &= \frac{1}{(B_2-\phi)^2 a_V C_{HV}}[\frac{ma_V^2 C_{HV}C_{VH}}{R_0 B_V}((B_2-\phi)B_V \\ &+ a_V C_{HV}(B_1+\rho) + ma_V^2 C_{HV}C_{VH}) \\ &+ (B_2-\phi)B_V(B_1+\rho) + ma_V^2 C_{HV}C_{VH}(\rho-\varphi)], \end{aligned}$$

$$\begin{aligned} A_3 &= \frac{1}{(B_2-\phi)^2 a_V C_{HV}}[(B_1+\rho)(-ma_V^2 C_{HV}C_{VH}) \\ &+ (-B_2+\eta+\varphi+\phi+B_1)B_V] \\ &= \frac{a_V m C_{VH}}{(B_2-\phi)^2}(\frac{1}{R_0}-1)(B_1+\rho). \end{aligned}$$

For the existence of endemic equilibrium $E^* = (s_h^*, i_h^*, i_v^*)$ 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

$$F(0) = A_3 < 0,$$

$$\begin{aligned} F(1) &= 1 + A_1 + A_2 + A_3 \\ &= \frac{1}{(B_2-\phi)^2 a_V C_{HV}}[ma_V^2 C_{HV}C_{VH} + (B_V + a_V C_{HV})B_1 \\ &+ \eta + \varphi)(B_1 + \rho + \phi - B_2)] > 0. \end{aligned}$$

So $F(i_h^*) = 0$ has at least one root $i_h^* \in (0,1)$ when $R_0 > 1$. Denote

$$A = A_1^2 - 3A_2,\ B = A_1 A_2 - 9A_3,\ C = A_2^2 - 3A_1 A_3,$$
$$\text{and } \Delta_1 = B^2 - 4AC,$$

then from the Cardan formulas, $F(i_h^*) = 0$ has an unique real root if and only if $\Delta_1 > 0$. From (8) and the assumption $B_1 + \rho > |B_2 - \phi|$ we can see $1 > s_h^* > 0$, and $1 > i_v^* > 0$. The assumption that $B_1 + \rho > |B_2 - \phi|$ is of significant importance and plays a great role when malaria persists. It shows that mortality rate due to malaria should be less than that at which the susceptible human population is refilled due to birth and loss of immunity to malaria .

Now we come to discuss the stability of the endemic equilibrium point E^*. Assume $F(i_h) = 0$ has an unique real root $i_h \in (0,1)$. For the system (7), the Jacobian matrix is

$J_E =$

$$\begin{pmatrix} -[B_1+\rho-(\phi-B_2)i_h^* + C_{VH}a_V m i_v^*] & \varphi+(\phi-B_2)s_h^*-\rho & -C_{VH}a_V m s_h^* \\ C_{VH}a_V m i_v^* & -[B_1+\varphi+\eta+(\phi-B_2)-2(\phi-B_2)i_h^*] & C_{VH}a_V m s_h^* \\ 0 & C_{HV}a_V(1-i_v^*) & -(B_V + C_{HV}a_V i_h^*) \end{pmatrix}.$$

For

$$\phi - B_2 = l_1,\ B_1 = l_h,\ B_V = l_v,\ C_{VH}a_V m = b,\ C_{HV}a_V = c,$$
$$w = l_h + \rho - l_1 i_h^* \ \text{and}\ \ w^* = l_h + \eta - l_1 i_h^*.$$

by the assumptions $\eta > \rho$ and $B_1 + \rho > |B_2 - \phi|$, we can see $w, w^* > 0$ *and* $w^* > w$. Then we have

$$J_E = \begin{pmatrix} -(l_h+\rho-l_1 i_h^* + b i_v^*) & \varphi + l_1 s_h^* - \rho & -b s_h^* \\ b i_v^* & -(l_h+\varphi+\eta+l_1-2l_1 i_h^*) & b s_h^* \\ 0 & c(1-i_v^*) & -(l_v^* + c i_h^*) \end{pmatrix}.$$

To prove the stability of the equilibrium point we have to calculate the roots.

$$det(J_E - I\lambda) = \lambda^3 + K_1\lambda^2 + K_2\lambda + K_3 = 0,$$

where K_1, K_2 *and* K_3 are given by the expressions,

$$K_1 = b i_v^* + w + w^* + \varphi + l_1(1-i_h) + l_v + c i_h,$$

$$\begin{aligned} K_2 &= (b i_v^* + w)[w^* + \varphi + l_1(1-i_h^*) + l_v + c i_h^*] \\ &+ [w^* + \varphi + l_1(1-i_h^*)](l_v + c i_h^*) \\ &- bcs_h^*(1-i_v^*) - b i_v^*(\varphi - \rho + l_1 s_h^*), \end{aligned}$$

$$\begin{aligned} K_3 &= (b i_v^* + w)[(w^* + \varphi + l_1(1-i_h^*))(l_v + c i_h^*) - bcs_h^*(1-i_v^*)] \\ &+ b i_v^*[bcs_h^*(1-i_v^*) - (\varphi - \rho + l_1 s_h^*)(l_v + c i_h^*)]. \end{aligned}$$

By assuming $\eta > \rho$ and $B_1 + \rho > |B_2 - \phi|$, we have

$$\begin{aligned} K_1 &= b i_v^* + w + w^* + \varphi + l_1(1-i_h^*) + l_v + c i_h^* \\ &= (l_h + \rho - l_1 i_h^*) + (b i_v^* - l_1 i_h^*) + [l_h + \eta + l_1(1-i_h^*)] + \varphi + l_v + c i_h^*. \end{aligned}$$

From $\eta > \rho, 0 < i_h^* < 1$ *and* $l_h + \rho > |l_1|$ we get $l_h + \eta > |l_1|(1-i_h^*)$, it is clear that $b i_v^* > l_1 i_h^*$ so $K_1 > 0$.

$$\begin{aligned} K_2 &= (b i_v^* + w)[w^* + \varphi + l_1(1-i_h^*) + l_v + c i_h^*] \\ &+ [w^* + \varphi + l_1(1-i_h^*)](l_v + c i_h^*) \\ &- bcs_h^*(1-i_v^*) - b i_v^*(\varphi - \rho + l_1 s_h^*) \\ &= w[(w^* + \varphi + l_1(1-i_h^*) + l_v + c i_h^*)] + b i_v^*[w^* + l_1(1-i_h^* - s_h^*) + \rho] \end{aligned}$$

$+[w^* + \varphi + l_1(1-i_h^*) + bi_v^*][l_v + ci_h^*] - bcs_h^*(1-i_v^*),$

Its clear that $l_h + \rho > |l_1|(1-i_h^* - s_h^*)$ by similarly $1 > s_h^* > 0$ and from (7)and (8) its easy to see that

$$[w^* + \varphi + l_1(1-i_h^*) + bi_v^*](l_v + ci_h^*) - bcs_h^*(1-i_v^*)$$
$$= [l_h + \eta - l_1 i_h^* + \varphi + l_1(1-i_h^*) + bi_v^*](l_v + ci_h^*) - bcs_h^*(1-i_v^*)$$
$$= ci_h^*\left(\frac{bi_v^* - l_1 i_h^*}{i_v^*}\right) + bcs_h^* i_v^*$$
$$\Rightarrow K_2 > 0.$$

and

$$K_3 = (bi_v^* + w)[(w^* + \varphi + l_1(1-i_h^*))(l_v + ci_h^*) - bcs_h^*(1-i_v^*)]$$
$$+bi_v^*[bcs_h^*(1-i_v^*) - (\varphi - \rho + l_1 s_h^*)(l_v + ci_h^*)].$$

1) In case that $l_1 < 0$

$$= (bi_v^* + w)\{[w^* + \varphi + l_1(1-i_h^*)](l_v + ci_h^*) - bcs_h^*(1-i_v^*)\}$$
$$+bi_v^*[bcs_h^*(1-i_v^*) - (\varphi - \rho + l_1 s_h^*)(l_v + ci_h^*)]$$
$$= w[(w^* + \varphi + l_1(1-i_h^*))(l_v + ci_h^*) - bcs_h^*(1-i_v^*)]$$
$$+bi_v^*\{[w^* + \varphi + l_1(1-i_h^*)](l_v + ci_h^*) - bcs_h^*(1-i_v^*) + bcs_h^*(1-i_v^*)$$
$$-(\varphi - \rho + l_1 s_h^*)(l_v + ci_h^*)\}$$
$$= w[(l_h + \eta - l_1 i_h^* + \varphi + l_1(1-i_h^*))(l_v + ci_h^*) - bcs_h^*(1-i_v^*)]$$
$$+bi_v^*\{[l_h + \eta - l_1 i_h^* + l_1(1-i_h^*)](l_v + ci_h^*) + (\rho - l_1 s_h^*)(l_v + ci_h^*)\}$$
$$= bi_v^*\{[l_h + \eta + \rho + l_1(1-2i_h^* - s_h^*)](l_v + ci_h^*)\}$$
$$+w[(l_h + \eta - l_1 i_h^* + \varphi + l_1(1-i_h^*))(l_v + ci_h^*) - bcs_h^*(1-i_v^*)]$$

we can see

$$bcs_h^* = [l_h + \varphi + \eta + l_1(1-i_h^*)](l_v + ci_h^*) \quad and \quad l_h + \rho > l_1(1-2i_h^* - s_h^*)$$

so we have

$$> bi_v^*\eta(l_v + ci_h^*)$$
$$+w[(l_h + \eta - l_1 i_h^* + \varphi + l_1(1-i_h^*))(l_v + ci_h^*) - bcs_h^*(1-i_v^*)]$$
$$> w[(l_h + \eta - l_1 i_h^* + \varphi + l_1(1-i_h^*))(l_v + ci_h^*)$$
$$-[l_h + \varphi + \eta + l_1(1-i_h^*)](l_v + ci_h^*)(1-i_v^*)]$$
$$= w(l_v + ci_h^*)\{-l_1 i_h^* + [l_h + \varphi + \eta + l_1(1-i_h^*)]i_v^*\} > 0 \Rightarrow K_3 > 0$$

then

2) In case that $l_1 > 0$

$$= bi_v^*[w^* l_v + \varphi l_v + l_1(1-i_h^*)l_v + w^* ci_h^* + \varphi ci_h^*$$
$$+l_1(1-i_h^*)ci_h^* - bcs_h^*(1-i_v^*)$$
$$+bcs_h^*(1-i_v^*) - \varphi l_v + \rho l_v - l_1 s_h^* l_v - \varphi ci_h^* + \rho ci_h^* - l_1 s_h^* ci_h^*]$$
$$+w[w^* l_v + \varphi l_v + l_1(1-i_h^*)l_v + w^* ci_h^* + \varphi ci_h^*$$
$$+l_1(1-i_h^*)ci_h^* - bcs_h^*(1-i_v^*)]$$
$$= bi_v^*[l_1(1-i_h^* - s_h^*)l_v + l_1(1-i_h^* - s_h^*)ci_h^* + \rho l_v$$
$$+\rho ci_h^*] + bi_v^* w^*(l_v + ci_h^*)$$
$$+w[w^* l_v + \varphi l_v + l_1(1-i_h^*)l_v + w^* ci_h^* + \varphi ci_h^*$$
$$+l_1(1-i_h^*)ci_h^* - bcs_h^*(1-i_v^*)]$$

by $w^* > w$

$$> bi_v^*[l_1(1-i_h^* - s_h^*)l_v + l_1(1-i_h^* - s_h^*)ci_h^* + \rho l_v$$
$$+\rho ci_h^*] + bi_v^* w(l_v + ci_h^*)$$
$$+w[w^* l_v + \varphi l_v + l_1(1-i_h^*)l_v + w^* ci_h^* + \varphi ci_h^*$$
$$+l_1(1-i_h^*)ci_h^* - bcs_h^*(1-i_v^*)]$$
$$= bi_v^*[l_1(1-i_h^* - s_h^*)l_v + l_1(1-i_h^* - s_h^*)ci_h^* + \rho l_v + \rho ci_h^*]$$
$$+w[(w^* + \varphi + l_1(1-i_h^*) + bi_v^*)(l_v + ci_h^*) - bcs_h^*(1-i_v^*)]$$

Similarly with above we can see $K_3 > 0$

$$K_1 K_2 - K_3 = [bi_v^* + w + w^* + \varphi + l_1(1-i_h^*) + l_v$$
$$+ci_h^*]w[w^* + \varphi + l_1(1-i_h^*) + l_v + ci_h^*]$$
$$+[bi_v^* + w + w^* + \varphi + l_1(1-i_h^*) + l_v + ci_h^*]bi_v^*[w^*$$
$$+l_1(1-i_h^* - s_h^*) + l_v + ci_h^* + \rho]$$
$$+[bi_v^* + w + w^* + \varphi + l_1(1-i_h^*) + l_v + ci_h^*]$$
$$[w^* l_v + \varphi l_v + l_1(1-i_h^*)l_v + w^* ci_h^* + \varphi ci_h^*$$
$$+l_1(1-i_h^*)ci_h^* - bcs_h^*(1-i_v^*)]$$
$$-bi_v^*[w^* l_v + l_1(1-i_h^* - s_h^*)l_v + w^* ci_h^*$$
$$+l_1(1-i_h^* - s_h^*)ci_h^* + \rho l_v + \rho ci_h^*]$$
$$-w[w^* l_v + \varphi l_v + l_1(1-i_h^*)l_v + w^* ci_h^* + \varphi ci_h^*$$
$$+l_1(1-i_h^*)ci_h^* - bcs_h^*(1-i_v^*)]$$
$$= w[bi_v^* + w + w^* + \varphi + l_1(1-i_h^*)$$
$$+l_v + ci_h^*][(w^* + \varphi + l_1(1-i_h^*) + l_v + ci_h^*)]$$
$$+bi_v^* w(l_v + ci_h^*)$$
$$+bi_v^*[bi_v^* + w + w^* + \varphi + l_1(1-i_h^*)][w^* + l_1(1-i_h^* - s_h^*) + \rho]$$
$$+[bi_v^* + w^* + \varphi$$
$$+l_1(1-i_h^*) + l_v + ci_h^*]\{[w^* + \varphi + l_1(1-i_h^*) + bi_v^*](l_v + ci_h^*)$$
$$-bcs_h^*(1-i_v^*)\}$$

As shown above $K_1 K_2 - K_3 > 0$, and we have: $K_1 > 0, K_2 > 0, K_3 > 0,$ *and* $K_1 K_2 - K_3 > 0$. Hence by the Routh-Hurwitz, the endemic equilibrium is locally asymptotically stable.

Lets discuss now the situation when $R_0 < 1$. It is clear that $F(0) = A_3 > 0$ *and* $F(1) = 1 + A_1 + A_2 + A_3 > 0$. Note that $F'(i_h) = 3i_h^2 + 2A_1 i_h + A_2$, if $\Delta_2 = A_1^2 - 3A_2 < 0$, then $F'(i_h) > 0$, hence $F(i_h) = 0$ has no real root in $(0,1)$. Then we get the following theorem.

Theorem 2 If $R_0 > 1$ and $\Delta_1 > 0$, there exists an unique endemic equilibrium E^*, which is locally asymptotically stable under the assumptions $\eta > \rho$, and $B_1 + \rho > |B_2 - \phi|$. If $R_0 < 1$ and $\Delta_2 < 0$, there exists no endemic equilibrium. Where Δ_1 and Δ_2 are defined as above.

4. NUMERICAL SIMULATION

In this section, we present the numerical analysis of the model to illustrate the transmission of malaria disease and the

impact of vertical transmission on spread of malaria and show the relations betwen R_0 and B_2 and a_v. We show baseline values in Table 3 for the parameters described in Table 2. The ode 45 and solve functions in Matlab was used in this numerical simulation, the parameter values in Table 3 are used in the simulations to illustrate the behaviour of the model.

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

$X^* = (s_h^*, i_h^*, r_h^*, s_v^*, i_v^*)$= (0.99,0.01,0,0.9,0.1), we study the dynamical behavior of the model for variation of the $B_2 = 0, 0, 001, 0, 002, 0, 003$, Figures 2-5 show the general behavior of the model ,we observe that when is increased from 0 to 0. 003 the number of infectious humans and infectious mosquitoes increase as well,we conclude that for any very small changes in B_2 we have a big increases infectious classes.

In the Figure 6, we can see The relationship between the infection human and variation of the B_2 , Finally, for showing the effect of newborn's birth with Infection rate to the basic reproduction number, we gives the relation between R_0 and B_2 (Fig 7), from (Fig 7), we know that R_0 is increasing with respect to the B_2 rate. These numerical results support the results earlier obtained analytically that the endemic equilibrium is stable.

Table 3. The parameters values for the the baseline scenario

μ_H	0.000038 / *day*
μ_V	0.05 / *day*
B_1	0.0015900 / *day*
B_V	0.072 / *day*
φ	0.0022 / *day*
ϕ	0.00332 / *day*
ρ	0.000017 / *day*
B_2	0 0.001 0.002 0.003
η	0.00019 / *day*
a_V	0.03
C_{VH}	0.75
C_{HV}	0.75

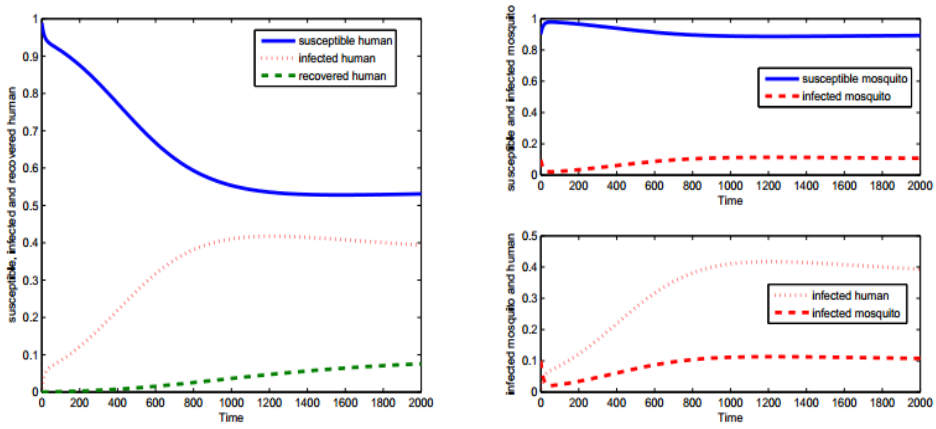

Figure 2. $B_2 = 0$ and $R_0 = 1.7$, Endemic patterns of the susceptible, infected and recovered human populations, and the susceptible and infected mosquito populations. Starting at the initial conditions $X^* = (s_h^*, i_h^*, r_h^*, s_v^*, i_v^*)$= (0.99,0.01,0,0.9,0.1), the system (3) approaches the endemic point (0.5308, 0.3934, 0.0758, 0.8924, 0.1076), and we see the relationship between i_h *and* i_v

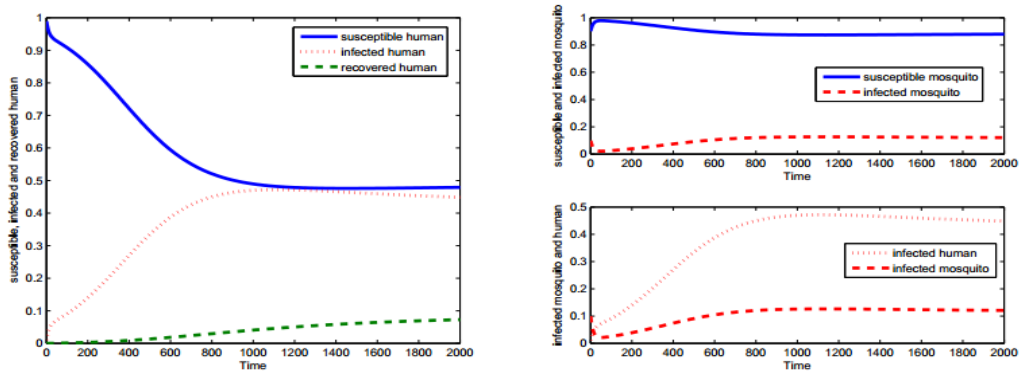

Figure 3. $B_2 = 0,001, and$ $R_0 = 1.99$, Endemic patterns of the susceptible, infected and recovered human populations, and the susceptible and infected mosquito populations. Starting at the initial conditions $X^* = (s_h^*, i_h^*, r_h^*, s_v^*, i_v^*)$= (0.99,0.01,0,0.9,0.1), the system (3) approaches the endemic point (0.48, 0.449 , 0.07 , 0.88 , 0.12), and we see see the relationship between i_h *and* i_v

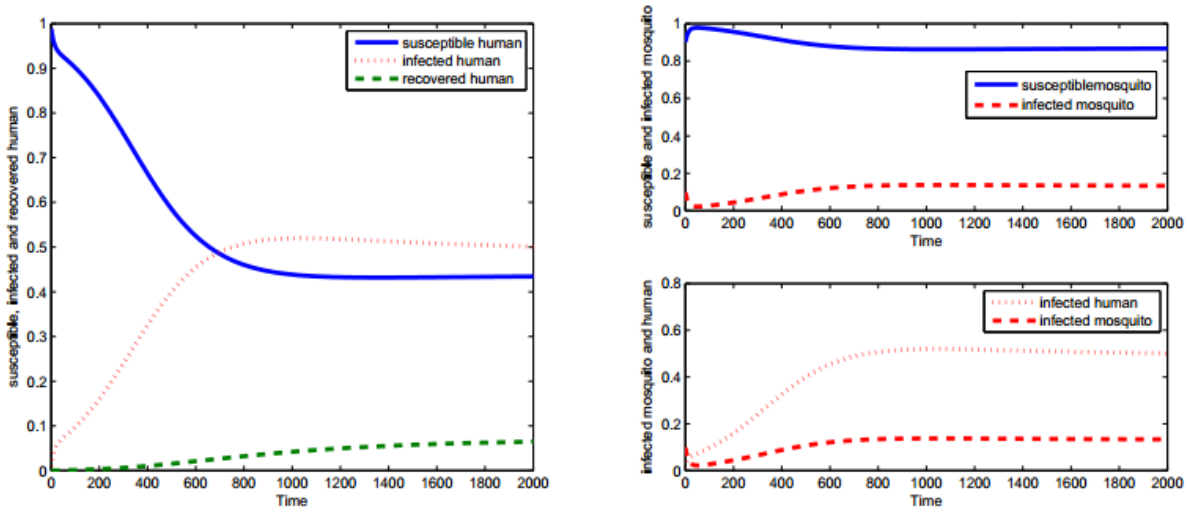

Figure 4. $B_2 = 0,002$ *and* $R_0 = 2.33$, Endemic patterns of the susceptible, infected and recovered human populations, and the susceptible and infected mosquito populations. Starting at the initial conditions $X^* = (s_h^*, i_h^*, r_h^*, s_v^*, i_v^*)$= (0.99,0.01,0,0.9,0.1), the system (3) approaches the endemic point (0.4300 , 0.501 , 0.0651 , 0.8901 , 0.1331), and we see see the relationship between i_h *and* i_v

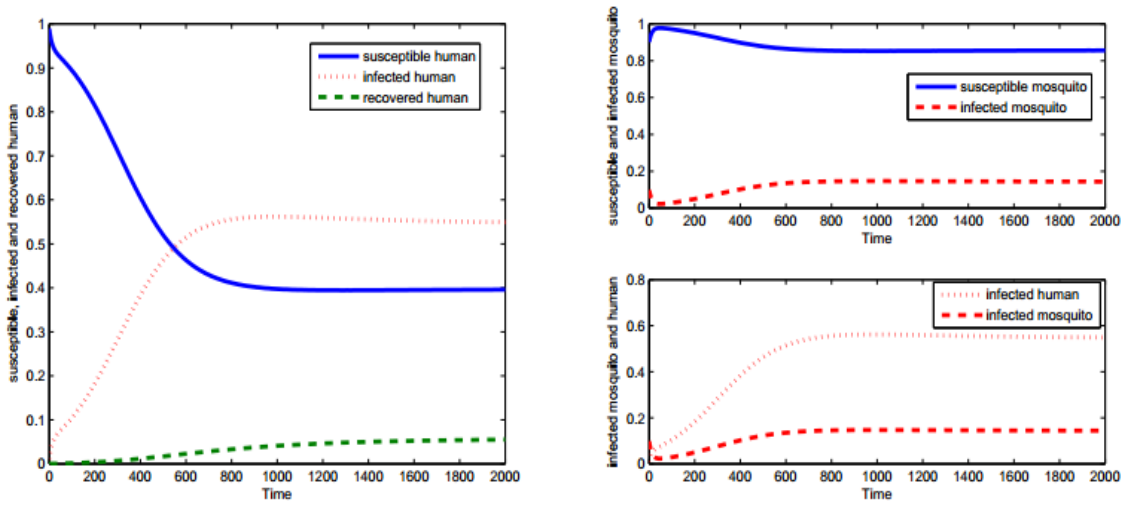

Figure 5. $B_2 = 0,003$ *and* $R_0 = 2.83$, Endemic patterns of the susceptible, infected and recovered human populations, and the susceptible and infected mosquito populations. Starting at the initial conditions $X^* = (s_h^*, i_h^*, r_h^*, s_v^*, i_v^*)$= (0.99,0.01,0,0.9,0.1), the system (3) approaches the endemic point (0.3959 , 0.5492 , 0.0549 , 0.8560 , 0.1439), and we see see the relationship between i_h *and* i_v

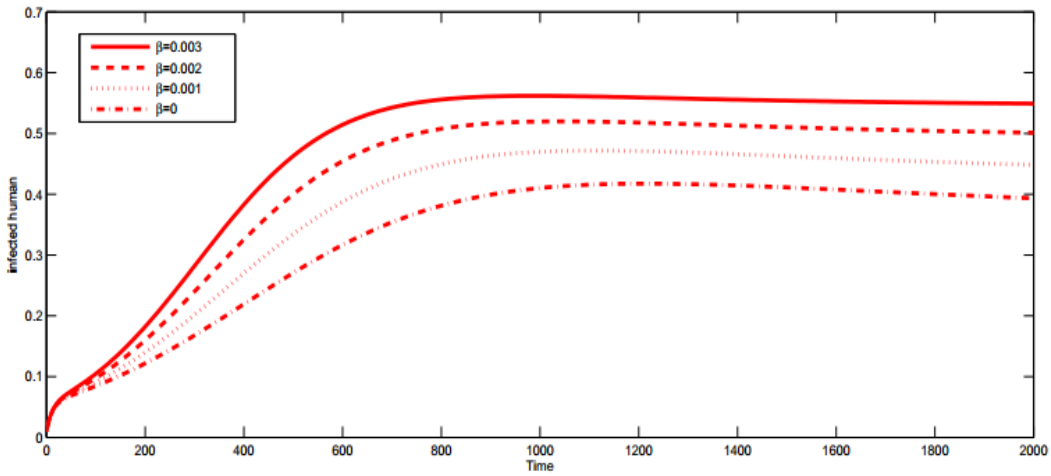

Figure 6. The variation of infected human population with time for different values of B_2

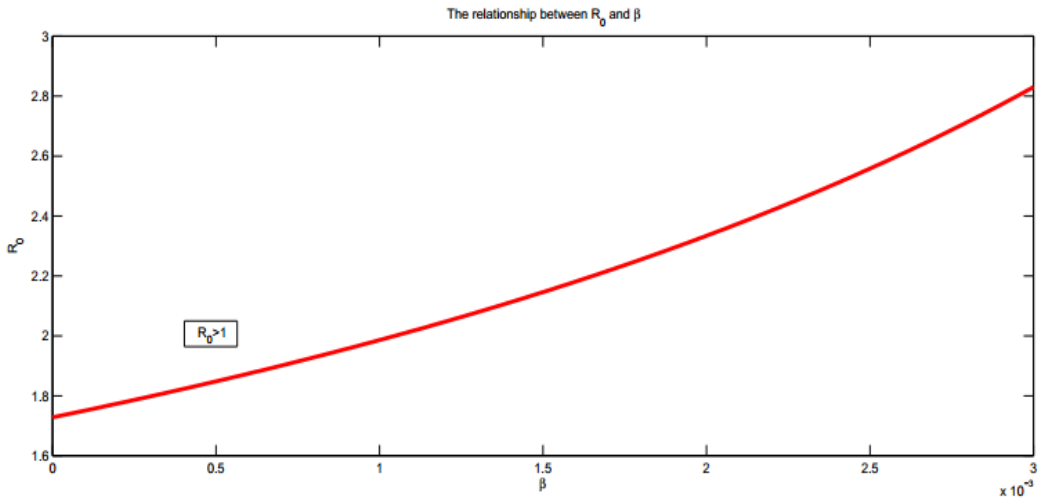

Figure 7. When rate of the newborn's birth with infection number gets larger the value of the reproduction number gets larger

5. DISCUSSION

In our study we have developed our model that includes the impact vertical transmission (congenital malaria) in the spread of malaria, we have derived and analyzed a mathematical model of 5-dimensional system of a nonlinear mathematical model which incorporates and include the infection newborn to better understand the transmission and spread of malaria. We change the parameter B_2 and keep all other parameters as in Figures 2-5 in order to see the effect of vertical transmission (congenital malaria) . In the Figures 2-5, from observation of the Figures we conclude that as when B_2 is increased from 0 to 0.003 there is a corresponding increase in the number of infectious humans and infectious mosquitoes, for any very small changes in B_2 we have a big increases infectious classes,

The variation of infected human population i_h with time for different B_2 is shown in Figure 6, It is observed that for each big value of B_2 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 reproduction 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 $R_0 > 1$.

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