



## Analysis of a Malaria Transmission Model in Children

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**ABSTRACT:** In this paper, a nine compartmental model for malaria transmission in children was developed and a threshold parameter called control reproduction number which is known to be a vital threshold quantity in controlling the spread of malaria was derived. The model has a disease free equilibrium which is locally asymptotically stable if the control reproduction number is less than one and an endemic equilibrium point which is also locally asymptotically stable if the control reproduction number is greater than one. The model undergoes a backward bifurcation which is caused by loss of acquired immunity of recovered children and the rate at which exposed children progress to the mild stage of infection.

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In most developing countries in Africa, Asia, Central America and South America, Malaria constitutes a major public health challenge for children. It is reported that from 40% to 50% of the world's population lives in malaria endemic areas (Portugal *et al.*, 2014; WHO, 2010). Malaria is a catastrophic infection with approximately 300-500 million cases yearly resulting in 1-2 million deaths, mostly among young children. Children of all ages living in areas where malaria is non endemic are equally susceptible to the malaria. Studies revealed that children under five years of age (mostly between six months and five years) in endemic areas are at the highest risk of malaria infection than other age groups. Furthermore, an estimated 660,000 malaria deaths were recorded among children around the world in 2010 and approximately 86% of these cases were less than five years of age. In high malaria transmission areas, young children with severe forms of malaria who have not acquired immunity to malaria can rapidly die of malaria. Children with malaria experience high fever which may be accompanied by chills, sweats, and headaches and other common symptoms include abdominal pain, diarrhea, vomiting, weakness, myalgia, and pallor. In children, these symptoms are frequently misdiagnosed with a viral syndrome or acute gastroenteritis. Also, in endemic areas, children with partial immunity frequently present the following symptoms: hepatosplenomegaly, anemia, and jaundice. However, the use of intravenous treatment is an appropriate plan for the medical care of children since the clinical condition of children younger than 5

years old with malaria can worsen rapidly (Metanat, 2005). In areas where malaria is not widespread, health specialists are frequently unfamiliar with the disease, and delays in detection and treatment are common. Increased awareness and knowledge of proper management strategy becomes a necessity considering the severity of this illness. The occurrence of malaria is common in patients who have a history of recent or ongoing use of a malaria chemoprophylactic agent.

This incident can be attributed to factors such as drug resistance, noncompliance with treatment, or inadequate or inappropriate administration (especially in children, because of the difficulties in administering bitter medications). Treatment must include careful supportive care, and intensive care measures should be available for treating children with complicated Plasmodium falciparum malaria. Drug regimens can include mefloquine, atovaquone-proguanil, sulfadoxine-pyrimethamine, quinine or quinidine, clindamycin, doxycycline, chloroquine, and primaquine (Stauffer and Fischer, 2003).

Mathematical models have been widely accepted as vital tools for studying the dynamics of the spread of communicable diseases. Over the years, several researchers and mathematicians have applied mathematical models to study mosquito related diseases. The present work therefore seeks to analyze the equilibrium states of a malaria model for stability and investigate the existence of backward bifurcation.

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**MATERIALS AND METHODS**

*Model Formulation:* In this work, we consider two populations consisting of children and vector population. The total population of children and vectors are divided into nine mutually exclusive classes; namely: Susceptible Children ( $S_c(t)$ ), Children with malaria at latent stage ( $E_c(t)$ ), Children with malaria at mild stage ( $I_{cm}(t)$ ), Children with malaria at severe stage ( $I_{cs}(t)$ ), Children treated of malaria ( $R_c(t)$ ), immunized children against malaria infection ( $Q(t)$ ); Susceptible vector ( $S_v(t)$ ), Vector with latent malaria infection ( $E_v(t)$ ) and Vector with malaria infection ( $I_v(t)$ ). The total number of children, ( $N_c(t)$ ) and Vector ( $N_v(t)$ ) are thus given as:

$$N_c(t) = S_c(t) + E_c(t) + I_{cm}(t) + I_{cs}(t) + R_c(t) + Q(t)$$

$$N_v(t) = S_v(t) + E_v(t) + I_v(t)$$

Susceptible children are recruited at rate  $\Lambda_c$ , while susceptible vectors are recruited at rate  $\Lambda_v$ . Also, susceptible children contact malaria with force of infection

$$\lambda_c = \frac{(1 - \epsilon q) \beta_c b_v I_v}{N_c}$$

And susceptible vectors acquire malaria infection from infected children with force of infection

$$\lambda_v = \frac{\beta_v b_v (I_{cs} + \eta_{cs} I_{cm})}{N_c}$$

The model equations are:

$$\begin{aligned} \dot{S}_c &= \Lambda_c - k_1 S_c - \lambda_c S_c + \omega_c R_c \\ \dot{E}_c &= \lambda_c S_c - k_2 E_c \\ \dot{I}_{cm} &= \gamma_c \phi_c E_c - k_3 I_{cm} \\ \dot{I}_{cs} &= (1 - \phi_c) \gamma_c E_c + \chi_c I_{cm} - k_4 I_{cs} \\ \dot{R}_c &= \sigma_c I_{cm} + \tau_c I_{cs} - k_5 R_c \\ \dot{Q} &= \psi_c S_c - \mu Q \\ \dot{S}_v &= \Lambda_v - \lambda_v S_v - k_6 S_v \end{aligned} \quad (1)$$

$$\dot{E}_v = \lambda_v S_v - k_7 E_v$$

$$\dot{I}_v = \gamma_v E_v - k_6 I_v$$

Where  $k_1 = (\psi_c + \mu)$ ,  $k_2 = (\gamma_c + \mu)$ ,  $k_3 = (\sigma_c + \delta_{cm} + \chi_c + \mu)$ ,  $k_4 = (\tau_c + \delta_{cs} + \mu)$ ,  $k_5 = (\omega_c + \mu)$ ,  $k_6 = (\delta_v + \mu_v)$  and  $k_7 = (\gamma_v + \delta_v + \mu_v)$

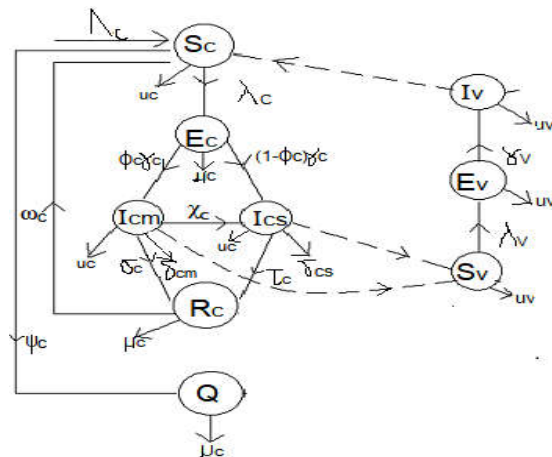


Fig 1: Flow diagram of Malaria transmission model in children

Table 1: Description of the variables of the Malaria model

Variable	Description
$S_c$	Population of susceptible children
$E_c$	Population of children with asymptomatic malaria
$I_{cm}$	Population of infected children at mild stage
$I_{cs}$	Population of infected children with severe case
$R_c$	Population of recovered children
$Q$	Immune children
$S_v, E_v, I_v$	Susceptible, Exposed and Infected Mosquitoes

Table 2: Description of the parameters of the Malaria model

Parameter	Description	Values	References
$\Lambda_c, \Lambda_v$	Recruitment rate into the children and vector population	156, 16667	Estimated
$\mu$	Natural death rate for children	1	(WHO, 2019)
$\mu_v$	Natural death rate for mosquitoes	$\frac{18.60 \times 365}{21}$	(Trpis and Hausermann, 1986)
$\beta_c, \beta_v$	Transmission probability per contact for children and susceptible mosquitoes	0.14, 0.356	(Ducrot, et al., 2009)
$\gamma_c$	Rate at which exposed children progress to the mild stage of infection	0.122	(Ducrot, et al., 2009)
$\phi_c$	Fraction of exposed children who progress to the mild stage of infection	0.5	Assumed

$\psi_c$	Vaccination rate for children	0.5	Assumed
$\delta_{cm}, \delta_{cs}$	Disease induced death rates of children at mild and severe stage	0.0003454, 0.00671	(AL-Rahman <i>et al.</i> , 2019)
$\delta_v$	Death rate of mosquitoes from insecticide	0.5	(Augusto <i>et al.</i> , 2017)
$\sigma_c, \tau_c$	Treatment rates for children at mild and severe stage	0.0082, 0.011	(Ducrot, <i>et al.</i> , 2009)
$\omega_c$	Rate at which recovered children lose their immunity	0.046	Estimated
$\gamma_v$	Rate at which exposed mosquitoes become infectious	0.18	(Ducrot, <i>et al.</i> , 2009)
$b_v$	Mosquito biting rate	0.5	(AL-Rahman <i>et al.</i> , 2019)
$\varepsilon$	Efficacy of insecticide treated nets(ITN)	0.5	(Saminu and Gimba, 2019)
$q$	Rate of ITN compliance	0.53	(Saminu and Gimba, 2019)
$\eta_c$	Infectivity modification parameters in children	0.5	Assumed
$\chi_c$	Rate at which infected children at mild stage progress to the severe stage of infection	0.11	(Ducrot, <i>et al.</i> , 2009)

*Analysis of the model: Boundedness of solutions*

Consider the region  $D_2 = \left\{ (S_c, E_c, I_{cm}, I_{cs}, R_c, Q, S_v, E_v, I_v) \in \mathfrak{R}_+^9 : N_c \leq \frac{\Lambda_c}{\mu}, N_v \leq \frac{\Lambda_v}{\mu_v} \right\}$ . It

can be shown that the set  $D_2$  is positively invariant and an attractor of all positive solutions of the model (1).

**Lemma 1:** *The region  $D_2$  is positively invariant for the model (1).*

Proof: The rate of change of the total children population is given thus

$$\dot{N}_c = \dot{S}_c + \dot{E}_c + \dot{I}_{cm} + \dot{I}_{cs} + \dot{R}_c + \dot{Q} = \Lambda_c - \mu N_c - \delta_{cm} I_{cm} - \delta_{cs} I_{cs} \tag{2}$$

By standard comparison theorem

$$N_c \leq \frac{\Lambda_c}{\mu} \tag{3}$$

By integrating factor method,

$$N_c(t) = N_c(0)e^{-\mu t} + \frac{\Lambda_c}{\mu}(1 - e^{-\mu t}) \tag{4}$$

The rate of change of total vector population gives

$$\dot{N}_v = \dot{S}_v + \dot{E}_v + \dot{I}_v = \Lambda_v - \mu_v N_v - \delta_v N_v \tag{5}$$

By standard comparison theorem;

$$N_v \leq \frac{\Lambda_v}{\mu_v}$$

By integrating factor method, we obtained

$$N_v(t) = N_v(0)e^{-\mu_v t} + \frac{\Lambda_v}{\mu_v}(1 - e^{-\mu_v t}) \tag{6}$$

In particular,  $N_c(t) \leq \frac{\Lambda_c}{\mu}$  if  $N_c(0) \leq \frac{\Lambda_c}{\mu}$  and  $N_v(t) \leq \frac{\Lambda_v}{\mu_v}$  if  $N_v(0) \leq \frac{\Lambda_v}{\mu_v}$  respectively.

Hence  $D_2$  is a positively invariant set and the solution enter  $D_2$  in finite time or  $N_c(t) \rightarrow \frac{\Lambda_c}{\mu}$  and

$N_v(t) \rightarrow \frac{\Lambda_v}{\mu_v}$  as  $t \rightarrow \infty$ . Hence it is sufficient to consider the dynamics of the model (1) in  $D_2$ . In this region,

the model (1) is seen as being mathematically and epidemiologically well posed.

Positivity of solution

**Lemma 2:** Let the initial data for the model (1) be

$$S_c(t) > 0, E_c(t) > 0, I_{cm}(t) > 0, I_{cs}(t) > 0, R_c(t) > 0, Q(t) > 0, S_v(t) > 0, E_v(t) > 0, I_v(t) > 0$$

Then the solution  $\{S_c(t), E_c(t), I_{cm}(t), I_{cs}(t), R_c(t), Q(t), S_v(t), E_v(t), I_v(t)\}$  with positive initial data will remain positive for all time  $t > 0$ .

**Proof:**

$$\text{Let } t_1 = \sup \left\{ \begin{array}{l} t > 0; S_c(t) > 0, E_c(t) > 0, I_{cm}(t) > 0, I_{cs}(t) > 0, R_c(t) > 0, Q(t) > 0, S_v(t) > 0 \\ E_v(t) > 0, I_v(t) > 0 \end{array} \right\} > 0$$

From the model (1),

$$\dot{S}_c \geq \Lambda_c - (k_1 + \lambda_c)S_c$$

This implied that:

$$\frac{d}{dt} \left\{ S_c(t) \left( \exp(k_1)t + \int_0^t \lambda_c(\tau) d\tau \right) \right\} \geq \Lambda_c \exp \left( (k_1)t + \int_0^t \lambda_c(\tau) d\tau \right) \tag{7}$$

$$S_c(t_1) \exp \left( (k_1)t_1 + \int_0^{t_1} \lambda_c(\tau) d\tau \right) - S_c(0) \geq \int_0^{t_1} \Lambda_c \exp \left( (k_1)\tau + \int_0^\tau \lambda_c(\tau) d\tau \right) d\tau \tag{8}$$

$$\begin{aligned} S_c(t_1) &\geq S_c(0) \exp \left( -(k_1)t_1 - \int_0^{t_1} \lambda_c(\tau) d\tau \right) \\ &+ \left[ \exp \left( -(k_1)t_1 - \int_0^{t_1} \lambda_c(\tau) d\tau \right) \right] \times \int_0^{t_1} \left[ \Lambda_c \exp \left( (k_1)y + \int_0^y \lambda_c(\tau) d\tau \right) \right] dy > 0 \end{aligned}$$

Similarly, it can be shown that all state variables of the model remain positive for all time,  $t > 0$  so that

$$E_c(t) > 0, I_{cm}(t) > 0, I_{cs}(t) > 0, R_c(t) > 0, Q(t) > 0, S_v(t) > 0, E_v(t) > 0, I_v(t) > 0$$

for all time  $t > 0$ .

**Local Stability of Disease Free Equilibrium (DFE)**

The model (1) has a disease -free equilibrium obtained by setting the right hand sides to zero and all the disease classes to zero to give

$$\xi^* = (S_c^*, E_c^*, I_{cm}^*, I_{cs}^*, R_c^*, Q^*, S_v^*, E_v^*, I_v^*) = \left( \frac{\Lambda_c}{(\psi_c + \mu)}, 0, 0, 0, 0, \frac{\psi_c \Lambda_c}{\mu(\psi_c + \mu)}, \frac{\Lambda_v}{(\delta_v + \mu_v)}, 0, 0 \right) \tag{9}$$

The stability of  $\xi^*$  is established using the next generation operator method by using the notation in (Van Den Driessche and Watmough, 2002), so that the matrices F and V are computed as;

From model (1)

$$F = \begin{bmatrix} 0 & 0 & 0 & 0 & \frac{(1 - \varepsilon q)\beta_c b_v S_c}{N_c} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & \frac{\beta_v b_v S_v \eta_c}{N_c} & \frac{\beta_v b_v S_v}{N_c} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{bmatrix} \tag{10}$$

$$V = \begin{bmatrix} k_2 & 0 & 0 & 0 & 0 \\ -\gamma_c \phi_c & k_3 & 0 & 0 & 0 \\ -(1 - \phi_c)\gamma_c & -\chi_c & k_4 & 0 & 0 \\ 0 & 0 & 0 & k_7 & 0 \\ 0 & 0 & 0 & -\gamma_v & k_6 \end{bmatrix} \tag{11}$$

Where,  $k_2 = (\gamma_c + \mu)$ ,  $k_3 = (\sigma_c + \delta_{cm} + \chi_c + \mu)$ ,  $k_4 = (\tau_c + \delta_{cs} + \mu)$ ,  $k_7 = (\gamma_v + \delta_v + \mu_v)$

$$k_6 = (\delta_v + \mu_v)$$

Therefore, the spectral radius is given by

$$\rho(FV^{-1}) = \sqrt{\frac{\beta_v b_v S_v \gamma_v \cdot \beta_c b_c S_c \gamma_c ((1 - \phi_c)k_3 + \chi_c \phi_c + \eta_c \phi_c k_4)(1 - \varepsilon q)}{N_c^* k_6 k_7 N_c k_2 k_3 k_4}} = R_E \tag{12}$$

$$R_E = \sqrt{R_v \cdot R_c} \tag{13}$$

$$R_v = \frac{\beta_v b_v S_v \gamma_v}{N_c^* k_6 k_7} \tag{14}$$

$$R_c = \frac{\beta_c b_c S_c \gamma_c ((1 - \phi_c)k_3 + \chi_c \phi_c + \eta_c \phi_c k_4)(1 - \varepsilon q)}{N_c^* k_2 k_3 k_4} \tag{15}$$

The value  $R_E$  is the effective reproduction number.

**Lemma3:** The Disease free equilibrium of the model (1) is locally asymptotically stable if  $R_E < 1$  and unstable if  $R_E > 1$ .

By Lemma 3, biologically speaking, malaria is eliminated from the population of children when  $R_E < 1$  if the initial sizes of the subpopulations of the model are in the region of attraction of  $\zeta^*$

Existence of Endemic Equilibrium Point (EEP)

Denoting the endemic equilibrium point (EEP) of model (1) by  $\zeta^{**} = (S_c^{**}, E_c^{**}, I_{cm}^{**}, I_{cs}^{**}, R_c^{**}, Q^{**}, S_v^{**}, E_v^{**}, I_v^{**})$  and solving model (1) in terms of force of infection at steady state gives

$$\begin{aligned} S_c^{**} &= \frac{\Lambda_c}{k_1 + \lambda_c^{**}}, E_c^{**} = \frac{\Lambda_c \lambda_c^{**}}{k_2(k_1 + \lambda_c^{**})}, I_{cm}^{**} = \frac{\gamma_c \phi_c \Lambda_c \lambda_c^{**}}{k_3 k_2 (k_1 + \lambda_c^{**})} \\ I_{cs}^{**} &= \frac{\gamma_c \Lambda_c ((1 - \phi_c)k_3 + \chi_c \phi_c) \lambda_c^{**}}{k_4 k_3 k_2 (k_1 + \lambda_c^{**})}, \\ R_c^{**} &= \frac{\sigma_c \phi_c \gamma_c \Lambda_c k_4 \lambda_c^{**} + \tau_c \gamma_c \Lambda_c ((1 - \phi_c)k_3 + \chi_c \phi_c) \lambda_c^{**}}{k_5 k_4 k_3 k_2 (k_1 + \lambda_c^{**})}, Q = \frac{\psi_c \Lambda_c}{\mu(k_1 + \lambda_c^{**})} \\ S_v^{**} &= \frac{\Lambda_v}{(k_6 + \lambda_v^{**})}, E_v^{**} = \frac{\Lambda_v \lambda_v^{**}}{k_7(k_6 + \lambda_v^{**})}, I_v^{**} = \frac{\Lambda_v \gamma_v \lambda_v^{**}}{k_7 k_6 (k_6 + \lambda_v^{**})} \end{aligned} \tag{16}$$

Recall that

$$\lambda_c^{**} = \frac{(1 - \varepsilon q) \beta_c b_v I_v}{N_c^{**}} \text{ and } \lambda_v^{**} = \frac{\beta_v b_v (I_{cs} + \eta_c I_{cm})}{N_c^{**}} \tag{17}$$

Substituting the value of  $I_v$  into (18) gives

$$\lambda_c^{**} = \frac{[(1 - \varepsilon q) \beta_c b_v \gamma_v \Lambda_v] \lambda_v^{**}}{N_c^{**} k_7 k_6 (k_6 + \lambda_v^{**})} \tag{18}$$

Substituting the value of  $I_{cm}$  and  $I_{cs}$  into (18) gives

$$\lambda_v^{**} = \frac{\beta_v b_v \gamma_c \Lambda_c [(1 - \phi_c)k_3 + \phi_c \chi_c + \eta_c \phi_c k_4] \lambda_c^{**}}{N_c^{**} k_4 k_3 k_2 (k_1 + \lambda_c^{**})} \tag{19}$$

Substituting the value  $\lambda_v^{**}$  into (19)

$$\lambda_c^{**} = \frac{[(1 - \varepsilon q)\beta_c b_c \gamma_v \Lambda_v] \left\{ \frac{\beta_v b_v \gamma_c \Lambda_c [(1 - \phi_c)k_3 + \phi_c \chi_c + \eta_c \phi_c k_4] \lambda_c^{**}}{N_c^{**} k_4 k_3 k_2 (k_1 + \lambda_c^{**})} \right\}}{N_c^{**} k_7 k_6 \left[ k_6 + \left\{ \frac{\beta_v b_v \gamma_c \Lambda_c [(1 - \phi_c)k_3 + \phi_c \chi_c + \eta_c \phi_c k_4] \lambda_c^{**}}{N_c^{**} k_4 k_3 k_2 (k_1 + \lambda_c^{**})} \right\} \right]} \quad (20)$$

Simplifying (20) gives

$$B_1 \lambda_c^{**2} + B_2 \lambda_c^{**} = 0 \quad (21)$$

$$\lambda_c^{**} (B_1 \lambda_c^{**} + B_2) = 0 \quad (22)$$

$$\text{Either } \lambda_c^{**} = 0 \text{ or } B_1 \lambda_c^{**} + B_2 = 0 \quad (23)$$

$$B_1 = N_c^{**} k_7 k_6 N_c^{**} k_4 k_3 k_2 k_6 + [\beta_v b_v \gamma_c \Lambda_c ((1 - \phi_c)k_3 + \chi_c \phi_c + \eta_c \phi_c k_4)] N_c^{**} k_7 k_6$$

$$B_2 = N_c^{**} k_4 k_3 k_2 N_c^{**} k_6 k_7 k_6 k_1 - \beta_v b_v \gamma_c \Lambda_c [(1 - \phi_c)k_3 + \phi_c \chi_c + \eta_c \phi_c k_4] (1 - \varepsilon q) \beta_c b_c \gamma_v \Lambda_v$$

$$= N_c^{**2} k_4 k_3 k_2 k_7 k_6^2 k_1 \left[ 1 - \frac{\beta_v b_v \gamma_v \Lambda_v \Lambda_c \beta_c b_c \gamma_c [(1 - \phi_c)k_3 + \phi_c \chi_c + \eta_c \phi_c k_4] (1 - \varepsilon q)}{N_c^{**} k_6 k_4 k_3 k_2 N_c^{**} k_7 k_6 k_1} \right]$$

$$B_2 = N_c^{**2} k_4 k_3 k_2 k_7 k_6^2 k_1 [1 - R_E^2] \quad (24)$$

$$B_2 < 0 \text{ if } R_E > 1$$

So the system of the model (1) has a unique (stable) endemic equilibrium if  $R_E > 1$

since  $\lambda_c > 0$  for  $R_E > 1$

Local Stability of Endemic Equilibrium Point

The Jacobian expressed in terms of force of infection result thus:

$$J(\xi^{**}) = \begin{bmatrix} -(k_1 + \lambda_c^{**}) & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ \lambda_c^{**} & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & \gamma_c \phi_c & -k_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (1 - \phi_c) \gamma_c & \chi_c & -k_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_c & \tau_c & -k_5 & 0 & 0 & 0 & 0 \\ \psi_c & 0 & 0 & 0 & 0 & -\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & -(k_6 + \lambda_v^{**}) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \lambda_v^{**} & -k_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_v & -k_6 \end{bmatrix}$$

The eigenvalues are:

$$\lambda_1 = -(\lambda_c^{**} + k_1), \lambda_2 = -k_2 < 0, \lambda_3 = -k_3 < 0, \lambda_4 = -k_4 < 0, \lambda_5 = -k_5 < 0,$$

$$\lambda_6 = -\mu < 0, \lambda_7 = -(k_6 + \lambda_v^{**}), \lambda_8 = -k_7 < 0, \lambda_9 = -k_7 < 0$$

From (23)

$$\lambda_c^{**} = -\frac{\beta_2}{\beta_1} = \frac{N_c^{**2} k_4 k_3 k_2 k_7 k_6^2 k_1 [R_E^2 - 1]}{N_c^{**} k_7 k_6 N_c^{**} k_4 k_3 k_2 k_6 + [\beta_v b_v \gamma_c \Lambda_c ((1 - \phi_c)k_3 + \chi_c \phi_c + \eta_c \phi_c k_4)] N_c^{**} k_7 k_6} > 0 \quad (25)$$

if  $R_E > 1$

Therefore,

$$\lambda_1 = -(\lambda_c^{**} + k_1) < 0 \text{ if } R_E > 1$$

Also

$$\lambda_7^{**} = -(k_6 + \lambda_v^{**}) \text{ but } \lambda_v^{**} = \frac{\beta_v b_v \gamma_c \Lambda_c [(1 - \phi_c)k_3 + \chi_c \phi_c + \eta_c k_4 \phi_c] \lambda_c^{**}}{N_c^{**} k_2 k_3 k_4 (k_1 + \lambda_c^{**})} > 0 \text{ if } R_E > 1$$

$$\lambda_7^{**} = -(k_6 + \lambda_v^{**}) < 0 \text{ if } R_E > 1$$

We conclude that the endemic equilibrium point (EEP) for Malaria model in Children is locally asymptotically stable (LAS) if  $R_E > 1$ .

**Bifurcation analysis:** To investigate the existence of a backward bifurcation at  $R_E < 1$ , we use the Center Manifold theorem as presented by (Agusto, 2017; Castillo-Chavez and Song, 2004).

**Theorem:** The Model (1) undergoes backward bifurcation at  $R_E = 1$  under certain condition.

**Proof:** The proof is based on the Centre Manifold Theorem

From Model (1) Let:

$$x_1 = S_c, x_2 = E_c, x_3 = I_{cm}, x_4 = I_{cs}, x_5 = R_c, x_6 = Q, x_7 = S_v, x_8 = E_v \text{ and } x_9 = I_v$$

The following are the transformed equation for model (1)

$$\begin{aligned} \dot{x}_1 &= \Lambda_c - \frac{(1 - \varepsilon q)}{N_c} \beta_c b_c x_9 x_1 + \omega_c x_5 - k_1 x_1 \\ \dot{x}_2 &= \frac{(1 - \varepsilon q)}{N_c} \beta_c b_c x_9 x_1 - k_2 x_2 \\ \dot{x}_3 &= \gamma_c \phi_c x_2 - k_3 x_3 \\ \dot{x}_4 &= (1 - \phi_c) \gamma_c x_2 + \chi_c x_3 - k_4 x_4 \\ \dot{x}_5 &= \sigma_c x_3 + \tau_c x_4 - k_5 x_5 \\ \dot{x}_6 &= \psi_c x_1 - \mu x_6 \\ \dot{x}_7 &= \Lambda_v - \beta_v b_v \frac{(x_4 + \eta_c x_3) x_7}{N_c} - k_6 x_7 \\ \dot{x}_8 &= \beta_v b_v \frac{(x_4 + \eta_c x_3) x_7}{N_c} - k_7 x_8 \\ \dot{x}_9 &= \gamma_v x_8 - k_6 x_9 \end{aligned} \tag{26}$$

The Jacobian of the transformed equation (26), evaluated at the DFE, is given as:

$$J(\xi^*) = \begin{bmatrix} -k_1 & 0 & 0 & 0 & \omega_c & 0 & 0 & 0 & -\frac{(1 - \varepsilon q)}{N_c} \beta_c b_v x_1 \\ 0 & -k_2 & 0 & 0 & 0 & 0 & 0 & 0 & \frac{(1 - \varepsilon q)}{N_c} \beta_c b_v x_1 \\ 0 & \gamma_c \phi_c & -k_3 & 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & (1 - \phi_c) \gamma_c & \chi_c & -k_4 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & \sigma_c & \tau_c & -k_5 & 0 & 0 & 0 & 0 \\ \psi_c & 0 & 0 & 0 & 0 & -\mu & 0 & 0 & 0 \\ 0 & 0 & -\frac{\beta_v b_v \eta_c}{N_c} x_7 & -\frac{\beta_v b_v x_7}{N_c} & 0 & 0 & -k_6 & 0 & 0 \\ 0 & 0 & \frac{\beta_v b_v \eta_c}{N_c} x_7 & \frac{\beta_v b_v x_7}{N_c} & 0 & 0 & 0 & -k_7 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & \gamma_v & -k_6 \end{bmatrix}$$

Let consider the case when  $\beta_c = \beta_c^*$  is chosen as the bifurcation parameter at  $R_E = 1$ , we have that:

$$\beta_c^* = \frac{N_c^* k_2 k_3 k_4 N_c^* k_6 k_7}{\beta_v b_v S_v^* \gamma_v \gamma_c S_c^* [(1 - \varphi_c) k_3 + \chi_c \varphi_c + \eta_c \varphi_c k_4] (1 - \varepsilon q)}$$

The right eigenvector of  $J(\xi^*)_{\beta_c = \beta_c^*}$  is given as:

$w = (w_1, w_2, w_3, w_4, w_5, w_6, w_7, w_8, w_9)^T$  where;

$$w_1 = \frac{(1 - \varepsilon q) b_v x_1 [\omega_c \beta_c \gamma_c (\sigma_c \varphi_c k_4 + \tau_c (1 - \varphi_c) k_3 + \tau_c \chi_c \varphi_c) - \beta_v k_2 k_3 k_4 k_5] w_9}{N_c^* k_1 k_2 k_3 k_4 k_5}$$

$$w_2 = \frac{(1 - \varepsilon q) \beta_c b_v x_1 w_9}{N_c^* k_2}, \quad w_3 = \frac{(1 - \varepsilon q) \beta_c b_v \varphi_c \gamma_c x_1 w_9}{N_c^* k_2 k_3}, \quad w_4 = \frac{(1 - \varphi_c)(1 - \varepsilon q) \beta_c b_v x_1 [k_3 w_9 + \gamma_c \varphi_c]}{N_c^* k_2 k_3 k_4}$$

$$w_5 = \frac{(1 - \varepsilon q) \beta_c b_v \gamma_c x_1 [(k_4 \varphi_c w_9 + \tau_c (1 - \varphi_c)(k_3 w_9 + \gamma_c \varphi_c)]}{N_c^* k_2 k_3 k_4 k_5},$$

$$w_6 = \frac{(1 - \varepsilon q) b_v x_1 \psi_c [\omega_c \beta_c \gamma_c (\sigma_c \varphi_c k_4 + \tau_c (1 - \varphi_c) k_3 + \tau_c \chi_c \varphi_c) - \beta_v k_2 k_3 k_4 k_5] w_9}{\mu N_c^* k_1 k_2 k_3 k_4 k_5},$$

$$w_7 = \frac{-\beta_v b_v^2 x_7 \beta_c (1 - \varepsilon q) \gamma_c x_1 [(\eta_c \varphi_c k_4 + (1 - \varphi_c) k_3 + \chi_c \varphi_c)] w_9}{N_c^{*2} k_2 k_3 k_4 k_6},$$

$$w_8 = \frac{\beta_v b_v^2 x_7 \beta_c (1 - \varepsilon q) \gamma_c x_1 [(\eta_c \varphi_c k_4 + (1 - \varphi_c) k_3 + \chi_c \varphi_c)] w_9}{N_c^{*2} k_2 k_3 k_4 k_7}, \quad w_9 = w_9 > 0$$

Likewise,  $J(\xi^*)$  has a left eigenvector

$$v = (v_1, v_2, v_3, v_4, v_5, v_6, v_7, v_8, v_9) \text{ Where; } \quad v_1 = v_5 = v_6 = v_7 = 0, \quad v_2 = \frac{N_c^* k_6}{(1 - \varepsilon q) \beta_c b_v x_1} v_9,$$

$$v_3 = \frac{\beta_v b_v \gamma_v x_7 (\chi_c + \eta_c k_4)}{N_c^* k_3 k_4 k_7} v_9, \quad v_4 = \frac{\beta_v b_v x_7 \gamma_v}{N_c^* k_4 k_7} v_9, \quad v_8 = \frac{\gamma_v v_9}{k_7}, \quad v_9 = v_9 > 0$$

It follows from the Castillo-chavez and Song theorem in (Castillo-Chavez and Song, 2004) that the associated non-zero partial derivatives of system (26) required to calculate the bifurcation coefficients  $a$  and  $b$  at (DFE) are defined as follow:

$$a = \sum_{k,i,j=1}^n v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j} (0,0) \tag{27}$$

which gives

$$a = 2v_2 w_1 w_9 \frac{\partial^2 f_2}{\partial x_1 \partial x_9} (0,0) + 2v_8 w_3 w_7 \frac{\partial^2 f_8}{\partial x_3 \partial x_7} (0,0) + 2v_8 w_4 w_7 \frac{\partial^2 f_8}{\partial x_4 \partial x_7} (0,0)$$

which simplifies to

$$a = G_1 - (G_2 + G_3 + G_4), \text{ where} \tag{28}$$

$$G_1 = \frac{2k_6 v_9 (1 - \varepsilon q) \beta_c b_v \omega_c \beta_c \gamma_c (\sigma_c \varphi_c k_4 + \tau_c (1 - \varphi_c) k_3 + \tau_c \chi_c \varphi_c) w_9}{N_c^* k_1 k_2 k_3 k_4 k_5}$$

$$G_2 = \frac{2v_9 k_6 (1 - \varepsilon q) \beta_c b_v \beta_v k_2 k_3 k_4 k_5 w_9}{N_c^* k_1 k_2 k_3 k_4 k_5} \tag{29}$$

$$G_3 = \frac{2\gamma_v v_9 (1 - \varepsilon q) \beta_c^2 \varphi_c \gamma_c^2 x_1^{*2} (1 - \varepsilon q) \beta_v^2 b_v^4 x_7 [(\eta_c \varphi_c k_4 + (1 - \varphi_c) k_3 + \chi_c \varphi_c)] w_9 \eta_c}{N_c^{*4} k_2^2 k_3^2 k_4 k_6 k_7} \tag{30}$$



$$G_4 = \frac{2\gamma_v v_9 (1 - \varepsilon q)^2 \beta_c^2 b_v^4 \gamma_c^2 x_1^{*2} [(1 - \varphi_c)k_3 + \chi_c \varphi_c] \beta_v^2 x_7^* [(\eta_c \varphi_c k_4 + (1 - \varphi_c)k_3 + \chi_c \varphi_c]}{N_c^{*4} k_2^2 k_3^2 k_4^2 k_6 k_7}$$

and

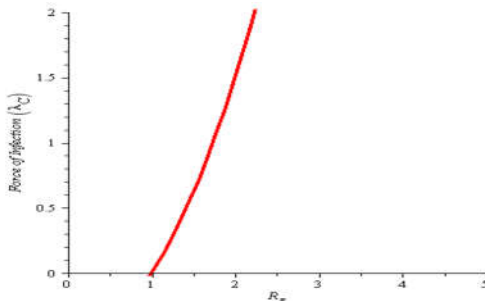
$$b = \sum_{k,i=1}^n v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial \beta_c^*}(0,0) \tag{31}$$

This gives:

$$b = \sum_{k,i=1}^n v_2 w_9 \frac{\partial^2 f_2}{\partial x_9 \partial \beta_c^*}(0,0)$$

$$b = \frac{k_6 v_9 w_9}{\beta_c} > 0 \tag{32}$$

Since the bifurcation coefficient  $b$  is positive. It follows from theorem 2 of (Castillo-Chavez and Song, 2004) that the transformed model (27) will undergo a backward bifurcation if the bifurcation coefficient  $a$  is positive.



**Fig2:** Backward bifurcation diagram for the model (1) showing the force of infection  $\lambda_c$  as a function of the control reproduction number  $R_E$  with all the parameters used as stated in Table 2

### RESULTS AND DISCUSSION

Epidemiologically, malaria can be eliminated from the children population if the initial size of the population is small enough such that the control reproduction number can be brought below unity. The model (1) will undergo a backward bifurcation whenever a stable disease free equilibrium point coexists with a stable endemic equilibrium point when the associated reproduction number is less than unity. The epidemiological implication of the backward bifurcation of the model (1) is that the classical requirement of the reproduction number being less than unity becomes only a necessity, but not sufficient condition for malaria control. Thus, it follows from the Castillo Chavez theorem (Castillo-Chavez and Song, 2004) that model (1) does not undergo the phenomenon of backward bifurcation if  $\omega_c = \gamma_c = 0$ . Hence, this study shows that the loss of acquired

immunity of recovered children ( $\omega_c$ ) and the rate at which exposed children progress to the mild stage of infection ( $\gamma_c$ ) are the causes of backward bifurcation in the malaria transmission model.

**Conclusion:** A vector-borne compartmental model was formulated to control the spread of malaria among children. The model was seen to exhibit the disease free equilibrium state which is locally asymptotically stable whenever the control reproduction number is less than unity and unstable otherwise. The endemic equilibrium state was proved to be locally asymptotically stable if the control reproduction number was greater than one. The model undergoes the phenomenon of backward bifurcation whenever the stable DFE coexists with a stable endemic equilibrium.

### REFERENCES

Augusto, FB; Bewick, S; Fagan, WF (2017). Mathematical Model of Zika Virus with Vertical Transmission. *Infectious Disease Modelling.* 2, 244-267.

Samiru, B ; Gimba,B (2019). Global Sensitivity Analysis to Study the Impacts of Bed-Nets, Drug Treatment, and Their Efficacies on a Two-Strain Malaria Model. *Math. Comput. Applicat.* 24(32), 1-28.

Bonyah, E; Okosun, O (2016) Mathematical Modeling of Zika Virus. *Asian Pacific J. Tropical Diseases.* 6 (9), 673-579.

Castillo-Chavez, C; Song, B (2004). Dynamical model of tuberculosis and their applications". *Math. Biosci. Eng.* 1, 361-404.

Ducrot, ASB; Sirima, BS; Zongo, P(2009). A Mathematical Model for Malaria Involving Differential Susceptibility, Exposedness and Infectivity of Human Host. *J. Biol. Dynamics.* 3(6), 574-598.

Metanat, M (2005). Malaria in Children, *International J. Infectious Dis.* (1), 1-2.

AL-Rahman,M; Osman, E; Yang, C; Adu,I.K. (2019). Mathematical Model of Malaria Transmission with Three Optimal Controls Applied to Democratic Republic of the Congo. *Global J. Sci.*

- Frontier Res.* 2(1), 1-14.
- Portugal, S; Moebius, J; Skinner, J; Doumbo, S ; Doumtabe, D ;Kone, Y (2014).Exposure-dependent control of malaria-induced inflammation in children. *PLoSPathog.*10 (4), 1-16.
- Stauffer, W;Fischer, PR (2003). Diagnosis and Treatment of Malaria in Children *Clinic. Infectious Dis.*37, 1340-1348.
- Trpis, MW; Hausermann,W (1986). Dispersal and Other Population Parameters of *Aedes Aegypti* in an African Village and Their Possible Significance in Epidemiology of Vector-Borne Diseases.” *Am. J. Trop. Med.Hygiene.*6(1), 1263-1279.
- Trpis, MW ; Hausermann, W ; Craig, GB(1995). Estimates of Population Size, Dispersal, and Longevity of Domestic *Aedes Aegypti Aegypti* (Diptera: Culicidae) by Mark-Release-Recapture in the Village of Shauri Moyo in Eastern Kenya. *J. Medic.Etomol.*32(1), 27-33.
- Van Den Driessche, P; Watmough, J (2002). Reproduction Numbers and Sub-Threshold Endemic Equilibria for Compartmental Models of Disease Transmission. *Math. Biosc.*180, 29-48.
- WHO (2019).*World Malaria Report.* <https://www.who.int/publications-detail/world-malaria-report-2019>.
- World Health Organization. (2010) Malaria in children under five. Available from: [http://www.who.int/malaria/areas/high\\_risk\\_groups/children/en/.Last](http://www.who.int/malaria/areas/high_risk_groups/children/en/.Last).