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Modelling Malaria Dynamics in Children under Five Years, Pregnant Women and the Influence of Temperature

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Abstract

Malaria remains a major public health problem worldwide and it affects the livelihood of people particularly children under 5 years and pregnant women. This study formulates and analyzes a mathematical model that incorporates children under five years, pregnant women and influence of temperature on the transmission dynamics of malaria. The next generation matrix method is applied to compute the basic reproduction number R_0. Analysis shows that malaria-free equilibrium point exists and it is globally asymptotically stable when $R_0 < 1$. Numerical simulations show that the rate of infections in both human and mosquito populations increases as temperature increases. Higher temperatures generally increase the rate of infection in both human and mosquito populations, with distinct impacts on children under five years and pregnant women.

Keywords: Basic reproduction number, malaria-free equilibrium, model simulation, model analysis, and temperature variations.

Introduction

Malaria is an infectious disease caused by a protozoan parasite from the genus plasmodium and spread through a bite from a female anopheles mosquito (WHO 2022). Malaria remains one of the major public health problems worldwide, with sub-Saharan Africa continuing to have the highest malaria-related mortality rates (Olaniyi and Obabiyi 2013). In 2022 the number of malaria cases worldwide were 245 million and 625,000 malaria-related deaths, with 232 million cases and 599,000 deaths from sub-Saharan Africa and 7.6 million cases and 25,464 malaria induced deaths from Tanzania (WHO 2022). In 2021, there were an estimated 247 million cases of malaria and 619,000 malaria-related deaths globally. Sub-Saharan Africa was the most affected region, with approximately 95% of the global cases, which translates to around 234 million cases, and 593,000 deaths. Within this region, Tanzania reported about 8 million malaria cases and 25,787 deaths due to malaria (WHO 2022). These figures indicate that malaria remains a significant global health issue, with sub-Saharan Africa experiencing the highest prevalence. Although there has been a slight decrease in the number of cases and deaths from 2021 to 2022, the disease continues to pose a critical challenge, highlighting the ongoing need for effective control measures.

The people that are most at risk for contracting malaria are pregnant women and children under five years (WHO 2022). Due to the loss of maternal immunity and the lack of specialized infection-specific immunity, children under five years are more susceptible to malaria than adults (Schumacher and Spinelli 2012). Additionally, pregnant women are more likely to get malaria due to the typical immunological decline that happens during

pregnancy, which is brought on by lack of cell-mediated immune response necessary to maintain the placenta (Sharma and Shukla 2017). Malaria causes severe infection in children under five years while pregnant women are at risk of getting maternal anemia (WHO 2022).

The trend of malaria transmission is affected by temperature variation. Statistics demonstrate that the burden of malaria in sub-Saharan Africa rises proportionately with temperature in the range of 16°C to 28°C (Agusto et al. 2015). When the temperature varies between 22.61°C and 28.58°C in West Africa, 16.68°C to 27.92°C in Central Africa, 19.04°C to 26.75°C in East Africa, and 16°C to 25°C in South Africa, malaria infection rates rise (Agusto et al. 2015 and Agusto, 2020). As the temperature rises, the malaria vector feeds more often because more blood is being digested, which causes the biting rate to increase (Agusto et al. 2015). Thus, it is essential to have a complete understanding of malaria transmission patterns in order to establish effective malaria management strategies, especially for children under five years and pregnant women.

Numerous mathematical models have been developed, examined, and used to study the dynamics of malaria infection. These include Forouzannia and Gumel 2014; Addawe and Pajimola 2016; Otieno et al. 2016, Azu-Tungmah et al. 2019, Kalula et al. 2021 and Agusto 2020).

Addawe and Pajimola 2016 explored how climate change affects malaria dynamics in a model that divides the human population into juveniles and adults. Their simulations showed that climate change influences malaria transmission patterns and highlighted a stable, long-term solution to the model.
Forouzannia and Gumel 2014 developed a similar age-structured model, also separating the population into juveniles and adults. Their findings revealed that increased mosquito lifespan and higher mosquito birth rates lead to more new infections and higher disease-related mortality.

Otieno et al. (2016) created a model focused on malaria transmission and control in Kenya. Their model considered vulnerable groups such as pregnant women and young children but did not include temperature effects on malaria transmission.

Azu-Tungmah et al. (2019) extended the age-structured model by incorporating four human compartments: susceptible individuals, infectious children under five, infectious individuals over five, and infectious pregnant women. They also divided mosquitoes into susceptible and infectious categories, providing a detailed view of malaria transmission dynamics.

Kalula et al. 2021 introduced a model that addressed malaria transmission among immigrants and asymptomatic carriers. This model separated the population into children under five years and adults. Building on this work, Kalula et al. 2023 added an analysis of optimal control strategies and temperature variations, although it did not include the role of children under five years and pregnant women in malaria dynamics.

In this study, a mathematical model for malaria transmission dynamics is formulated by modifying the model by Azu-Tungmah et al. (2019). The model of this study incorporates children under five years, pregnant women, non-pregnant humans (males and non-pregnant women aged above five years) and the influence of temperature as it affects the biting behavior of mosquitoes.

Materials and Methods

Model formulation

The model divides the human population into six compartments: susceptible non-pregnant humans S_n, susceptible pregnant women S_p, susceptible children under five years S_c, infectious non-pregnant humans I_n, infectious pregnant women I_p, and infectious children under five years I_c. Thus, the total human population is given as

$$N_h(t) = S_n(t) + S_p(t) + S_c(t) + I_n(t) + I_p(t) + I_c(t). \quad (1)$$

The mosquito population is divided into two compartments, which are susceptible mosquitoes S_m and infectious mosquitoes I_m. Therefore, the total mosquito population is given as

$$N_m(t) = S_m(t) + I_m(t). \quad (2)$$

Susceptible children under five years are considered to be recruited through birth at per capita rate Λ_h. However, they decrease as they develop immune system to become susceptible non-pregnant humans at a rate σ and when they contract malaria infection at a rate

$$\lambda_c = \frac{a_{mc}b(T)I_m}{N_h}, \quad (3)$$

where a_{mc} is the transmission probability of malaria from infectious mosquito to a susceptible child per single bite and $b(T)$ is the mosquito temperature dependent biting rate (Kalula et al. 2023) defined by

$$b(T) = -0.00014T^2 + 0.027T - 0.322. \quad (4)$$

Infectious children under five years increase following malaria infection of susceptible children under five years at a rate λ_c and decrease when they suffer malaria induced mortality at the rate π_c.

Susceptible pregnant women increase when women from the susceptible non-pregnant humans get pregnancy at a rate α. The class decreases when they deliver and when they contract malaria infection at rates β and

$$\lambda_p = \frac{a_{mp}b(T)I_m}{N_h} \quad (5)$$

respectively, where a_{mp} is the transmission probability of malaria from infectious mosquito to a susceptible pregnant woman per single bite. The infectious pregnant women increase when susceptible pregnant women acquire malaria infection and when infectious non-pregnant humans get pregnancy at rates λ_p and α respectively. They suffer disease-induced mortality at a rate π_p. Susceptible non-pregnant humans replenish when children under five years develop strong immune system and when susceptible pregnant women deliver at rates σ and β respectively. They diminish when they contract malaria infections at a rate

$$\lambda_n = \frac{a_{mn}b(T)I_m}{N_h} \quad (6)$$

where a_{mn} is the the transmission probability of malaria from infectious mosquito to a susceptible non-pregnant human per single bite. The infectious non-pregnant humans grow in number following malaria infection of susceptible non-pregnant humans at a rate λ_n and suffer malaria-induced mortality at the rate π_n. All human classes suffer natural mortality at a rate μ_h.

Susceptible mosquitoes are considered to increase through birth at a rate Λ_m and diminish when they contract malaria infection after biting infectious humans at a rate

$$\lambda_m = \frac{a_{nm}b(T)I_n}{N_h} + \frac{a_{pm}b(T)I_p}{N_h} + \frac{a_{cm}b(T)I_c}{N_h}, \quad (7)$$

where a_{nm} is the transmission probability of malaria from infectious non-pregnant human to a susceptible mosquito, a_{pm} is the transmission probability of malaria from infectious pregnant women to a susceptible mosquito and a_{cm} is the transmission probability of malaria from infectious child under five years to a susceptible mosquito. Infectious mosquitoes grow in number when susceptible mosquitoes contract malaria at a rate λ_m. Both mosquito classes suffer natural-induced mortality at a rate μ_m.

Model Assumptions

In formulating the model for malaria transmission in children under five years, pregnant women and the influence of temperature, we assume the following: most infectious pregnant women receive therapeutic treatment before giving birth and thus, they cannot join the infectious non-pregnant humans (Azu-Tungmah et al. 2019); humans and mosquitoes are born susceptible to malaria infection; pregnant women, children under 5 years, and non-pregnant humans acquire malaria infection at different rates because of their immunological differences; there is no disease-induced death for mosquito due to their short life span; the recruitment rates for both human and mosquito populations are population dependent; and all parameters are assumed to be positive.

The interactions of human and mosquito populations in the presence of malaria infection are shown in Figure1.

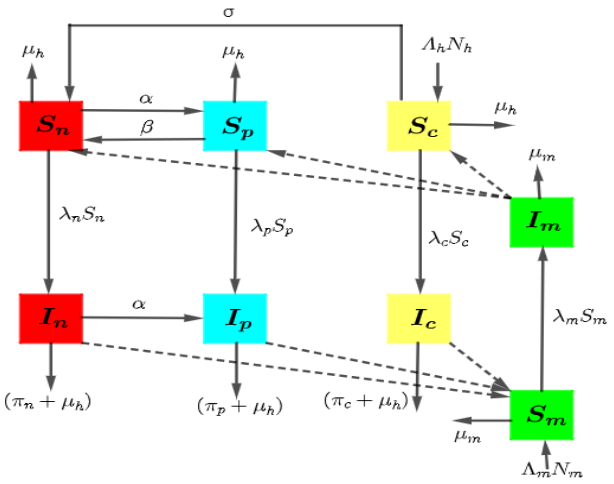

Figure 1: Compartmental diagram for the transmission dynamics of malaria

Model Equations

The dynamics of malaria transmission in children under five years and pregnant women is described by the following system of ordinary differential equations.

$$\left.\begin{aligned}
\frac{dS_n}{dt} &= \sigma S_c + \beta S_p - (\alpha + \lambda_n + \mu_h)S_n \\
\frac{dI_n}{dt} &= \lambda_n S_n - (\alpha + \mu_h + \pi_n)I_n \\
\frac{dS_p}{dt} &= \alpha S_n - (\beta + \mu_h + \lambda_p)S_p \\
\frac{dI_p}{dt} &= \lambda_p S_p + \alpha I_n - (\mu_h + \pi_p)I_p \\
\frac{dS_c}{dt} &= \Lambda_h N_h - (\sigma + \mu_h + \lambda_c)S_c \\
\frac{dI_c}{dt} &= \lambda_c S_c - (\mu_h + \pi_c)I_c \\
\frac{dS_m}{dt} &= \Lambda_m N_m - (\mu_h + \lambda_m)S_m \\
\frac{dI_m}{dt} &= \lambda_m S_m - \mu_m I_m
\end{aligned}\right\} \quad (8)$$

With initial conditions:$S_n(0) > 0$; $(0) \geq 0$; $S_p(0) > 0$; $I_p(0) \geq 0$; $S_c(0) > 0$; $I_c(0) \geq 0$; $S_m(0) > 0$; and $I_n(0) \geq 0$, where λ_n, λ_p, λ_c and λ_m are given in equations (6), (5), (3) and (7) respectively.

Model Analysis

To analyze malaria model, we normalize each class by dividing it by entire population as in Kalula et al. 2021. Let $s_n = \frac{S_n}{N_h}, s_p = \frac{S_p}{N_h}, s_c = \frac{S_c}{N_h}, s_m = \frac{S_m}{N_m}$, $i_n = \frac{I_n}{N_h}$, $i_p = \frac{I_p}{N_h}$. Taking time derivative for each variable we have

$\frac{ds_n}{dt} = \frac{1}{N_h}\left(\frac{dS_n}{dt} - s_n\frac{dN_h}{dt}\right)$, $\frac{ds_p}{dt} = \frac{1}{N_h}\left(\frac{dS_p}{dt} - s_p\frac{dN_h}{dt}\right)$ and so on for the rest of equations.

Solving for derivatives of normalized variables we have the following system of differential equations:

$$\left.\begin{aligned}
\frac{ds_n}{dt} &= \sigma s_c + \beta s_p + \left(\pi_n i_n + \pi_p i_p + \pi_c i_c\right)s_n - (\alpha + \Lambda_h + Ka_{mn}b(T)i_m)s_n \\
\frac{di_n}{dt} &= Ka_{mn}b(T)i_m s_n + \left(\pi_n i_n + \pi_p i_p + \pi_c i_c\right)i_n, -(\alpha + \Lambda_h + \pi_n)i_n \\
\frac{ds_p}{dt} &= \alpha s_n + \left(\pi_n i_n + \pi_p i_p + \pi_c i_c\right)s_p - \left(\beta + \Lambda_h + Ka_{mp}b(T)i_m\right)s_p \\
\frac{di_p}{dt} &= Ka_{mp}b(T)i_m s_p + \alpha i_n + \left(\pi_n i_n + \pi_p i_p + \pi_c i_c\right)i_p - \left(\Lambda_h + \pi_p\right)i_p \\
\frac{ds_c}{dt} &= \Lambda_h + \left(\pi_n i_n + \pi_p i_p + \pi_c i_c\right)s_c - (\sigma + \Lambda_h + Ka_{mc}b(T)i_m)s_c \\
\frac{di_c}{dt} &= Ka_{mc}b(T)i_m s_c + \left(\pi_n i_n + \pi_p i_p + \pi_c i_c\right)i_c - (\Lambda_h + \pi_c)i_c \\
\frac{ds_m}{dt} &= \Lambda_m(1 - s_m) - b(T)\left(a_{nm}i_n + a_{pm}i_p + a_{cm}i_c\right)s_m \\
\frac{di_m}{dt} &= b(T)\left(a_{nm}i_n + a_{pm}i_p + a_{cm}i_c\right)s_m - \Lambda_m i_m.
\end{aligned}\right\} \quad (9)$$

where K is the mosquito to human ratio.

Positivity of Model Solutions

In this section, we study the model properties to determine whether it is mathematically well posed and biologically meaningful. If the model's solutions are both positive and bounded, then the model is said to be mathematically well posed and biologically meaningful. To show that solutions of the model system are positive, we state and prove Lemma 1.

Lemma 1. Let the initial conditions be

$s_n(0) > 0$; $i_n(0) \geq 0$; $s_p(0) > 0$; $i_p(0) \geq 0$; $s_c(0) > 0$; $i_c(0) \geq$; $s_m(0) > 0$; and $i_m(0) \geq 0$, then the solutions $s_n(t)$, $s_p(t)$, $s_c(t)$, $s_m(t)$, $i_n(t)$, $i_p(t)$, $i_c(t)$, $i_m(t)$ of the normalized model system are positive for all $t \geq 0$.

Proof. Proving that the model solutions are positive for all $t \geq 0$, the first equation for susceptible non-pregnant humans in the model system is written as

$\frac{ds_n}{dt} = \sigma s_c + \beta s_p + (\pi_n i_n + \pi_p i_p + \pi_c i_c)s_n - (\alpha + \Lambda_h + Ka_{mn}b(T)i_m)s_n \geq -(\alpha + \Lambda_h + Ka_{mn}b(T)i_m)s_n$.

After integration by separation of variables, we have

$s_n(t) \geq s_n(0)exp\left(-\int_0^t(\alpha + \Lambda_h + Ka_{mn}b(T)i_m)dx\right) \geq 0$ for all $t \geq 0$.

Applying the same approach for the rest of equations, it can be shown that

$i_n(t) \geq 0, s_p(t) \geq 0$, $i_p(t) \geq 0, s_c(t) \geq 0$, $i_c(t) \geq 0, s_m(t) \geq 0$, $i_m(t) \geq 0$.

As a result, every solution of the model system is positive for all $t \geq 0$.

Boundedness of the model solutions

The model system is well posed if its solutions are bounded. We establish the boundedness of the model solution in this section by stating and proving Lemma 2.

Lemma 2. The solutions of the model system are bounded in the region

$\Omega = \Omega_h \times \Omega_m$ where, $\Omega_h = \left\{\left(s_n, s_p, s_c, i_n, i_p, i_c\right) \in \mathbb{R}_+^6 : N_h \leq 1\right\}$ and $\Omega_m = \{(s_m,\ i_m) \in \mathbb{R}_+^2 : N_m \leq 1\}$.

Proof. Let $s_n(0) > 0; i_n(0) \geq 0$; $s_p(0) > 0$; $i_p(0) \geq 0$; $s_c(0) > 0$; $i_c(0) \geq$; $s_m(0) > 0$; and $i_m(0) \geq 0$ be the initial conditions for the model system , and $\left\{\left(s_n(t), s_p(t), s_c(t), i_n(t), i_p(t), i_c(t)\right) \in \mathbb{R}_+^6\right\}$ and $\{(s_m(t), i_m(t)) \in \mathbb{R}_+^2\}$ be solutions.

From the total human population in equation (1), we have

$\frac{dN_h(t)}{dt} = \Lambda_h - \Lambda_h N_h - (1 - \mathrm{N}_h)\left(\pi_n i_n + \pi_p i_p + \pi_c i_c\right)$,

which implies that

$$\frac{dN_h(t)}{dt} + \Lambda_h N_h \leq \Lambda_h. \quad (10)$$

Since equation (10) is a linear differential inequality, we solve the inequality by integration factor and applying the limit as $t \to \infty$ to obtain

$$0 \leq N_h(t) \leq 1, \forall t > 0.$$

Applying the same procedures for total mosquito population in equation (2) we obtain

$$0 \leq N_m(t) = 1, \forall t > 0.$$

Thus, all solutions of the model system are positive invariant in the region:
$\Omega = \Omega_h \times \Omega_m$ where, $\Omega_h = \{(s_n, s_p, s_c, i_n, i_p, i_c) \in \mathbb{R}_+^6 : N_h \leq 1\}$ and $\Omega_m = \{(s_m,\ i_m) \in \mathbb{R}_+^2 : N_m \leq 1\}$. Therefore, the system of differential equations is mathematically well-posed in the domain Ω and we can consider the model for analysis.

Malaria Free Equilibrium point and Basic Reproduction Number R_0

Malaria Free Equilibrium Point

When malaria does not exist in human and mosquito populations, the derivative in equation (9) are equated to zero and solving the simultaneous equations to obtain the malaria free equilibrium P^0 as

$$P^0 = \left(\frac{\sigma(\beta + \Lambda_h)}{(\sigma + \Lambda_h)(\alpha + \beta + \Lambda_h)}, 0, \frac{\alpha\sigma}{(\sigma + \Lambda_h)(\alpha + \beta + \Lambda_h)}, 0, \frac{\Lambda_h}{(\sigma + \Lambda_h)}, 0, 1, 0\right). \quad (11)$$

The Basis Reproduction Number R_0

The Basic reproduction number is the expected number R_0 of secondary infections caused by a single infectious individual in a completely susceptible population (Kalula et al. 2021). To compute the basic reproduction number R_0, we use the next generation matrix method as used by Van de Driessche and Watmough 2002. The next generation matrix method uses new infections and transfer terms from infected classes. Let D_i be the vector of new infection and B_i be the vector of transfer terms of individual from the infected compartment i_n, i_p, i_c and i_m. Then the basic reproduction number R_0 is defined as the spectral radius of the next-generation matrix, which is denoted by

$$R_0 = \rho(DB^{-1}) \quad (12)$$

where *D* and *B* are the matrices defined by

$$D = \frac{\partial D_i}{\partial \varphi_j}(P^0), \quad B = \frac{\partial B_i}{\partial \varphi_j}(P^0) \ . \quad (13)$$

From the system of differential equation (9), the vectors for new infection D_i and transfer terms B_i are $D_i = \begin{pmatrix} Ka_{mn}b(T)i_m s_n \\ Ka_{mp}b(T)i_m s_p \\ Ka_{mc}b(T)i_m s_c \\ b(T)(a_{nm}i_m + a_{pm}i_p + a_{cm}i_c) \end{pmatrix}$ and

$$B_i = \begin{pmatrix} (\alpha + \Lambda_h + \pi_n)i_n - (\pi_n i_n + \pi_p i_p + \pi_c i_c)i_n \\ (\Lambda_h + \pi_p)i_p - \alpha I_n - (\pi_n i_n + \pi_p i_p + \pi_c i_c)i_p \\ (\Lambda_h + \pi_c)i_c - (\pi_n i_n + \pi_p i_p + \pi_c i_c)i_c \\ \Lambda_m i_m \end{pmatrix}$$

Using equation (11), the basic reproduction number R_0 is given by:

$$R_0 = \sqrt{R_n + R_p + R_c} \quad (14)$$

where $R_p = \frac{\alpha\sigma((\beta+\Lambda_h)Ka_{mn}b(T)+(\alpha+\pi_n+\Lambda_h)Ka_{mp}b(T))a_{pm}b(T)}{\Lambda_m(\sigma+\Lambda_h)(\alpha+\beta+\Lambda_h)(\alpha+\pi_n+\Lambda_h)(\pi_p+\Lambda_h)}$

$R_n = \frac{\sigma(\beta+\Lambda_h)Ka_{mn}b(T)a_{nm}b(T)}{\Lambda_m(\sigma+\Lambda_h)(\alpha+\beta+\Lambda_h)(\alpha+\pi_n+\Lambda_h)}$, and $R_c = \frac{\Lambda_h Ka_{mc}b(T)a_{cm}b(T)}{\Lambda_m(\sigma+\Lambda_h)(\pi_c+\Lambda_h)}$.

R_n is the number of secondary infections as result of interaction between susceptible non-pregnant humans and infectious mosquitoes, R_p is the number of secondary infections as a result of interactions between pregnant women and infectious mosquitoes and R_c is the number

of secondary infections in children under five years caused by infected mosquitoes in the susceptible population.

Global stability of malaria free equilibrium

We employ the method as used in Castillo-Chavez et al. (2004) to analyze the global stability of malaria free equilibrium point of the model. Using this approach, the model system is written as,

$$\begin{cases} \frac{dQ_n}{dt} = Z(Q_n - Q_0) + Z_1 Q_i \\ \frac{dQ_i}{dt} = Z_2 Q_i \end{cases} \tag{15}$$

where Q_n presents the non-transmitting classes; Q_i represents the transmitting classes; Q_0 represents the value of non-transmitting variables at malaria free equilibrium and Z, $Z_1$and Z_2 are the matrices to be obtained. The malaria free equilibrium point of the model is said to be globally asymptotically stable when the eigenvalue of the matrix Z are negative and Z_2 is a Metzler matrix (Irunde et al. 2016). From equation (8) and (10) we have

$Q_n = \left(s_n, s_p, s_c, s_m\right)^T$, $Q_i = \left(i_n, i_p, i_c, i_m\right)^T$, and

$$Q_0 = \left(\frac{\sigma(\beta+\mu_h)}{(\sigma+\Lambda_h)(\alpha+\beta+\Lambda_h)}, \frac{\alpha\sigma}{(\sigma+\Lambda_h)(\alpha+\beta+\Lambda_h)}, \frac{\Lambda_h}{(\sigma+\Lambda_h)}, 1\right)^T \tag{16}$$

From equation (14) we have

$$Q_n - Q_0 = \begin{pmatrix} s_n - \frac{\sigma(\beta+\mu_h)}{(\sigma+\Lambda_h)(\alpha+\beta+\Lambda_h)} \\ s_p - \frac{\alpha\sigma}{(\sigma+\Lambda_h)(\alpha+\beta+\Lambda_h)} \\ s_c - \frac{\Lambda_h}{(\sigma+\Lambda_h)} \\ s_m - 1 \end{pmatrix} \tag{17}$$

Using equation (15) and (16) we have

$$\begin{pmatrix} \sigma s_c + \beta s_p + \left(\pi_n i_n + \pi_p i_p + \pi_c i_c\right)s_n - (\alpha + \Lambda_h + K a_{mn} b(T) i_m)s_n \\ \alpha s_n + (\pi_n i_n + \pi_p i_p + \pi_c i_c)s_p - (\beta + \Lambda_h + K a_{mp} b(T) i_m)s_p \\ \Lambda_h + (\pi_n i_n + \pi_p i_p + \pi_c i_c)s_c - (\sigma + \Lambda_h + K a_{mc} b(T) i_m)s_c \\ \Lambda_m(1 - s_m) - b(T)\left(a_{nm} i_n + a_{pm} i_p + a_{cm} i_c\right)s_{m,} \end{pmatrix} =$$

$$Z \begin{pmatrix} s_n - \frac{\sigma(\beta+\mu_h)}{(\sigma+\Lambda_h)(\alpha+\beta+\Lambda_h)} \\ s_p - \frac{\alpha\sigma}{(\sigma+\Lambda_h)(\alpha+\beta+\Lambda_h)} \\ s_c - \frac{\Lambda_h}{(\sigma+\Lambda_h)} \\ s_m - 1 \end{pmatrix} + Z_1 \begin{pmatrix} i_n \\ i_p \\ i_c \\ i_m \end{pmatrix}$$

and

$$\begin{pmatrix} K a_{mn} b(T) i_m s_n + (\pi_n i_n + \pi_p i_p + \pi_c i_c) i_n, -(\alpha + \Lambda_h + \pi_n) i_n \\ K a_{mp} b(T) i_m s_p + \alpha i_n + (\pi_n i_n + \pi_p i_p + \pi_c i_c) i_p - (\Lambda_h + \pi_p) i_p \\ K a_{mc} b(T) i_m s_c + (\pi_n i_n + \pi_p i_p + \pi_c i_c) i_c - (\Lambda_h + \pi_c) i_{c,} \\ b(T)\left(a_{nm} i_n + a_{pm} i_p + a_{cm} i_c\right)s_m - \Lambda_m i_{m.} \end{pmatrix} = Z_1 \begin{pmatrix} i_n \\ i_p \\ i_c \\ i_m \end{pmatrix}.$$

The matrices Z and Z_2 are defined by: $Z = \frac{dQ_n}{dX_n}$ and $Z_2 = \frac{dQ_i}{dX_i}$

where X_n and X_i are non-infectious and infectious compartments respectively.

From the definitions of Z and Z_2 we have

$$Z = \begin{pmatrix} -(\alpha + \Lambda_h) & 0 & 0 & 0 \\ \alpha & -(\Lambda_h + \beta) & 0 & 0 \\ 0 & 0 & -(\Lambda_h + \sigma) & 0 \\ 0 & 0 & 0 & -\Lambda_m \end{pmatrix} \text{ and}$$

$$Z_2 = \begin{pmatrix} -(\alpha+\Lambda_h+\pi_n) & 0 & 0 & Ka_{mn}b(T)s_n \\ 0 & -(\Lambda_h+\pi_p) & 0 & Ka_{mp}b(T)s_p \\ 0 & 0 & -(\Lambda_h+\pi_c) & Ka_{mc}b(T)s_c \\ a_{nm}b(T)s_m & a_{pm}b(T)s_m & a_{cm}b(T)s_m & -\Lambda_m \end{pmatrix}.$$

The eigenvalues of matrix Z are $-(\alpha+\beta+\Lambda_h)$,$-(\Lambda_h+\sigma)$, $-\Lambda_m$ and $-\Lambda_h$. Since all eigenvalues of matrix Z are negative and all off diagonal elements of matrix Z_2 are non-negative, then the disease free equilibrium point is globally asymptotically stable when the basic reproduction number is less than one.

Existence of malaria Equilibrium point

Let $E^* = (s_n{}^*, i_n{}^*, s_p{}^*, i_p{}^*, s_c{}^*, i_c{}^*, s_m{}^*, i_m{}^*)$ be the endemic equilibrium point, then E^* is a steady state solution where the disease continues to exist. It is obtained by setting each equation in normalized system equals to zero. To show the existence of malaria equilibrium point we use the approach by Tumwiine et al. 2007 and Stephano et al. 2022. Using this approach the sum of human compartments at malaria equilibrium point gives

$\Lambda_h + (\pi_n i_n{}^* + \pi_n i_p{}^* + \pi_n i_c{}^*)n_h = \Lambda_h + (\pi_n i_n{}^* + \pi_n i_p{}^* + \pi_n i_c{}^*)$.

Since $\Lambda_h > 0$ and $\pi_j > 0, j = n, p, c$ then $s_n > 0$, $i_n \geq 0$, $s_p > 0$, $i_p \geq 0$, $s_c > 0$ and , $i_c \geq 0$. Using the same procedures, the mosquito population gives, $s_m > 0$ and $i_m \geq 0$.

Since $s_n > 0$,$i_n \geq 0$, $s_p > 0$, $i_p \geq 0$, $s_c > 0$, $i_c \geq 0$, $s_m > 0$ and $i_m \geq 0$, thus the malaria equilibrium exists.

Results and Discussion

To understand well the transmission dynamics of malaria in children under five years, pregnant women and non-pregnant women, we carry out numerical simulations of the normalized system using proportions of initial conditions $s_n(0) = 0.46$, $i_n(0) = 0.10$, $s_p = 0.16$, $i_p(0) = 0.01$, $s_c(0) = 0.25$, $i_c(0) = 0.02$, $s_m(0) = 0.90$ and $i_m(0) = 0.1$ and the parameter values in Table 1.

Table 1: Parameter Values (Unit: month^{-1})

Parameter	Value	Source	Parameter	Value	Source
σ	0.59999	Assumed	a_{mc}	0.02798	Azu-Tungmah et al. 2019
α	0.001395	Azu-Tungmah *et al.* 2019	a_{mn}	0.07249	Azu-Tungmah et al. 2019
β	0.0014	Assumed	a_{mp}	0.10587	Assumed
Λ_h	0.002716	Addawe and Lope 2012	a_{cm}	0.09994	Assumed
Λ_m	0.56	Addawe and Lope 2012	a_{nm}	0.09864	Assumed
π_c	0.001717	Azu-Tungmah et al. 2019	a_{pm}	0.00454	Azu-Tungmah et al. 2019
π_n	0.015928	Azu-Tungmah et al. 2019	$b(T)$	28.0	Agusto et al. 2015
π_p	0.020507	Assumed	K	1.2	Addawe and Pajimola 2016

Figure 2(a) shows the dynamics of malaria in children under five years. The graph indicates that the proportion of susceptible children under five years get infections and decrease exponentially while the proportion of infected children under five years increase exponentially. This shows that malaria persists in the population. In Figure 2(b), the dynamics of malaria in pregnant women is shown. The proportion of infected pregnant

women grows with time until when it reaches its equilibrium condition. This results into decreasing of the proportion of susceptible pregnant women for 2.5 months when it reaches its equilibrium state. From Figure 2(c) it is observed that the proportion of infected non-pregnant humans initially increases to its highest point in the first three months until when it attains the stable state. Figure 2(d) shows the disease prevalence in mosquito population. It can be seen that as time increases, the fraction of infected mosquitoes' increases, which means that malaria becomes endemic in the population as the proportion of susceptible mosquitoes decreases to the steady state after the third month.

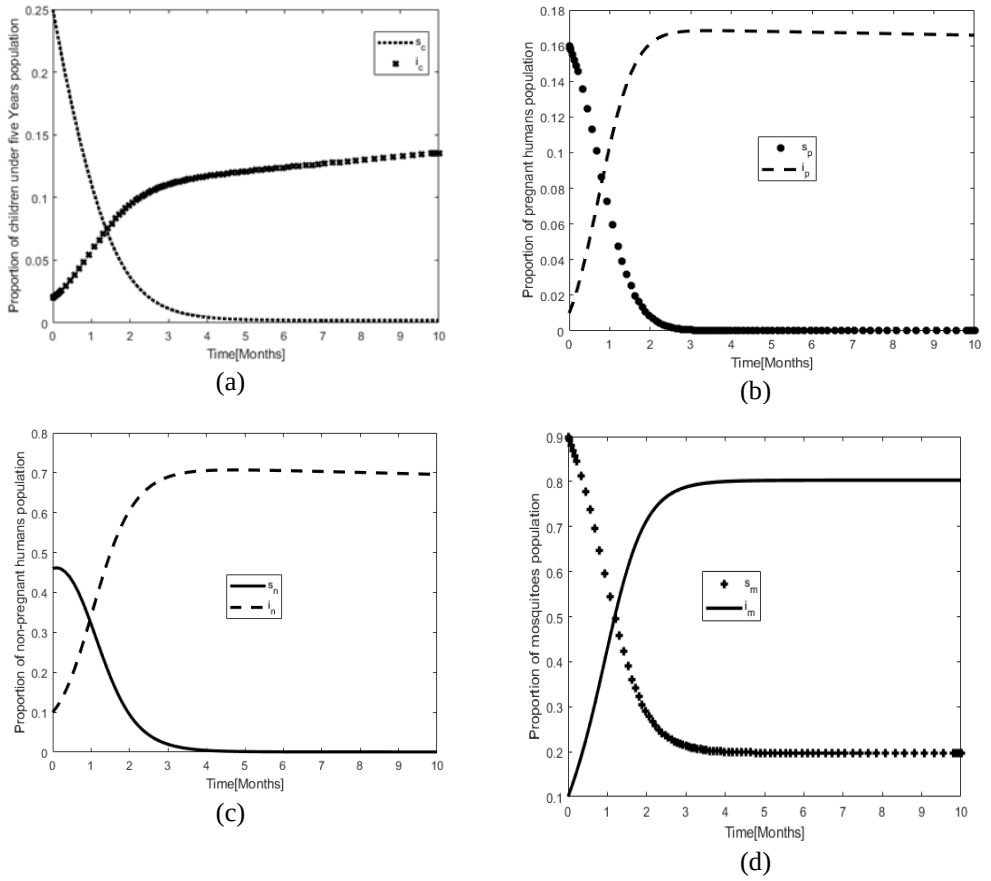

Figure 2: Dynamics of malaria transmission in the human and mosquito population.

Figures 3 (a), (b), (c) and (d) demonstrate how the number of infectious non-pregnant humans, pregnant women, children under five years and mosquitoes vary with respect to temperature dependent biting rate $b(T)$. The figures show that increasing temperature dependent biting rate $b(T)$ increases the number of infectious non-pregnant humans, pregnant women, children under five years and mosquitoes. As the temperature dependent mosquito's biting rate increases from 28 to 43 per month the rate of infections increase proportionally.

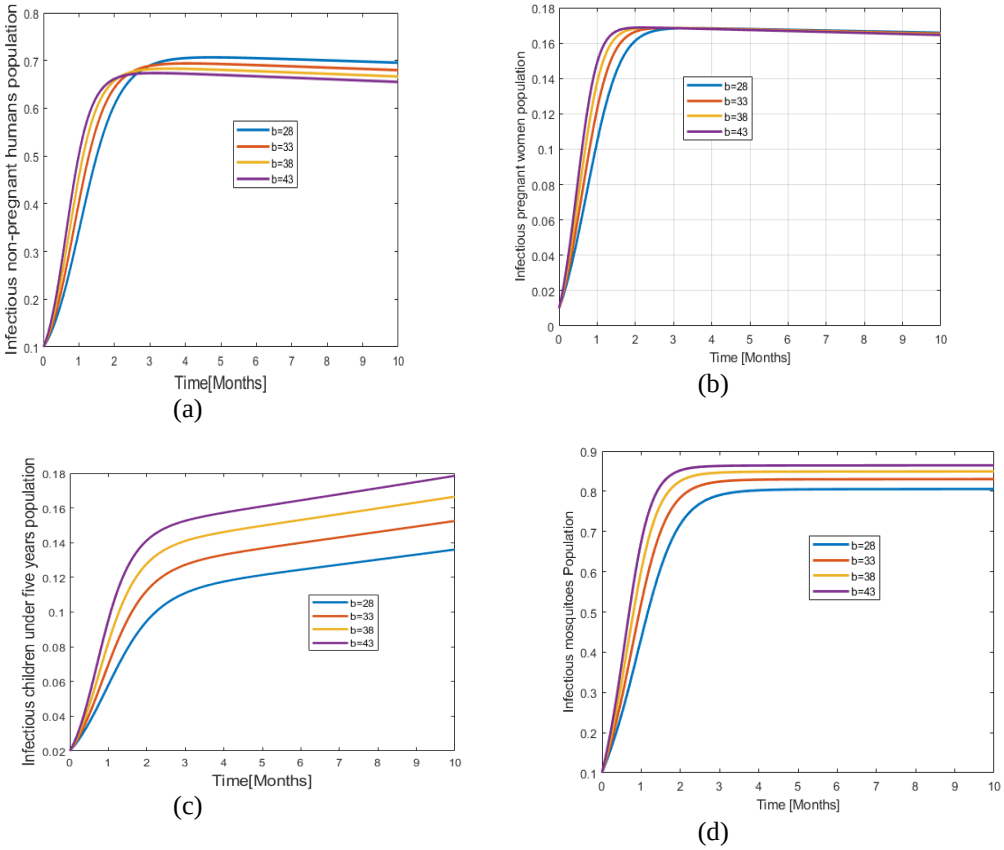

Figure 3: Variation of infectious classes with temperature dependent mosquito biting rate $b(T)$

Conclusion

The basic model of malaria transmission dynamics with humans and mosquito population has been formulated and analyzed. The main objective of this study was to understand malaria transmission dynamics in children under five years and pregnant women. The basic reproduction number R_0 has been computed using the next generation matrix method. Analysis of the model shows that malaria-free equilibrium point exists and it is globally asymptotically stable when $R_0 < 1$. Numerical simulations show that the malaria prevalence increases with time due to the absence of control interventions in the community. Similarly, increasing temperature dependent biting rate $b(T)$ increases the number of infectious non-pregnant humans, pregnant women, children under five years and mosquitoes. Therefore, to eradicate the malaria different control measures should be used.

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