Analyzing the Effects of Temperature and Human Movement on Malaria Disease Transmission Dynamics

Ganga Ram Phaijoo

Department of Mathematics, School of Science
Kathmandu University, Dhulikhel, Kavre, Nepal
Email: gangaram@ku.edu.np

Abstract

Background: Malaria disease is transmitted by the bite of Anopheles mosquitoes. Plasmodium parasites are responsible for the disease. Due to human movement from one place to the other, vector borne diseases like malaria are spreading rapidly throughout the world. They have become major causes of morbidity and mortality worldwide. Changing temperature levels has significant impact on the life cycle, biting behavior and death rates of the mosquitoes which can transmit the disease.

Methods: A multi patch SEIRS - SEI deterministic compartmental model for malaria disease is developed to study the disease transmission dynamics. The impact of temperature and human movement in transmission dynamics is investigated. Both global and local basic reproduction numbers are computed for two patches in two patch setting.

Results: Disease free equilibrium is locally stable when the basic reproduction number is less than unity and unstable when the number is greater than unity. Numerical results show that the prevalence of the disease changes with the change in human movement rates between the patches; temperature affects the transmission dynamics of malaria disease.

Conclusion: The burden of malaria disease can be reduced by managing the host movement between low and high disease prevalent patches. The optimal temperature for malaria disease transmission is 25°C.

Keywords: Malaria, Human movement, Temperature, Multi-Patch Model, Basic Reproduction Number, Disease Free Equilibrium; Stability
Background

Malaria is a vector borne disease which is caused by the protozoan parasites of genus Plasmodium. WHO estimates that about 36% of the world population is exposed to the risk of malaria. There were 228 millions of cases and 405000 deaths due to malaria worldwide in 2018. Malaria has a wide distribution of endemicity that extends from South Asia and South-east Asian countries\textsuperscript{[39]}. The disease is transmitted to humans by infected Anopheles mosquitoes.

Malaria is one of the oldest vector borne infectious diseases which has been studied for a long time from different aspects. Different modeling approaches are helpful in guiding different stages of the disease through synthesizing available information and extrapolating it. Sir Ronald Ross was one among the first to publish a series of papers using mathematical functions to study transmission of malaria in early 1900\textsuperscript{[32;34]}. He developed a simple model, now known as Ross model\textsuperscript{[33]} which explained the relationship between the number of mosquitoes and incidence of malaria in humans. Mathematical models are useful tools for studying the transmission dynamics of infectious diseases. Kermack and McKendrick developed an epidemiological compartmental model\textsuperscript{[14;15]} to study transmission dynamics of infectious diseases. They divided the total populations into the subpopulations of susceptibles, infecteds and recovereds. These models have been modified to investigate transmission dynamics of vector borne infectious diseases like dengue, malaria, zika, etc\textsuperscript{[5;8;9;10;18;20;23;24;27;29;31]}.

Malaria disease is sensitive to the climate change. Both increase and fluctuation in temperature are affecting both vectors and parasites of malaria disease. This can cause reduced prevalence of the disease in some areas, while it may increase in other\textsuperscript{[21;27;28;35]}. A number of research works have been carried out to observe the impact of climatic changes on transmission dynamics of malaria disease. One of the principal determinants of mosquitoes’ survival is temperature which has been associated with seasonal changes. So, mathematical studies have been made to understand the role of temperature in transmission dynamics of malaria disease. Lou and Zhao\textsuperscript{[19]}, Zhon et al\textsuperscript{[41]} studied the relation between climate variability and malaria disease. The researchers in\textsuperscript{[1;22;25;26;40]}
carried out the researches to observe the impact of temperature on the malaria disease transmission.

Human movement between endemic and non endemic areas are causing the huge burden of morbidity and mortality worldwide. It contributes in increasing the geographic spread of the diseases. Many mathematical works have been proposed to observe the impacts of the human movement on the transmission of the human infectious diseases. Arino and Driessche (2003) proposed multicity epidemic model\cite{4}. Multipatch models of the infectious diseases are studied by\cite{6;13;16;17;30;37;38}. Arino et al\cite{2;3} discussed the spreading of disease in metapopulations. Cosner et al\cite{11} focused their research on the effects of human movement on the persistence of vector borne diseases.

Previous research works show that there is a significant role of both human movement and temperature on transmission dynamics of malaria disease. In the present work, both human movement and temperature are incorporated using multi patch SEIRS - SEIR model of malaria disease. Different temperature levels and human movement rates are considered to observe their impact on malaria disease. Basic reproduction number of individual patches and combined (global) basic reproduction number are computed and the local stability of the disease free equilibrium point of the model is studied.

**Methods:**

**Model Formulation and Description**

We consider SEIRS - SEI multi - patch model with $n$ patches. The total human (host) population in each patch is denoted by $H_i$, $i = 1, 2, 3, \cdots , n$. Human population in each patch is subdivided into the four epidemiological classes: Susceptible $S^h_i$, Exposed $E^h_i$, Infectious $I^h_i$ and Recovered $R^h_i$. The total mosquito (vector) population $M_i$ in each patch is subdivided into the three epidemiological classes: Susceptible $S^v_i$, Exposed $E^v_i$ and Infectious $I^v_i$, $i = 1, 2, 3, \cdots , n$. Due to short life span of the mosquitoes, the recovered class in the mosquito population is not considered.

The recruitment rate for host population is $\zeta^h_i$. The natural death rate for the host
population is $\mu_i^h$. $b_i$ is the biting rate of mosquitoes (average number of bites per mosquito per day). So, the number of bites by $M_i$ mosquitoes per day is $b_i M_i$ and the number of bites per day per human is

$$b_i \left( \frac{M_i}{H_i} \right).$$

The probability that mosquito is infectious is $\frac{I^v_i}{M_i}$. So, the number of potentially infectious bites given by mosquito per human per day is

$$\left( b_i \frac{M_i}{H_i} \right) \left( \frac{I^v_i}{M_i} \right).$$

Let $\beta_i^h$ be the probability that a bite from infected mosquito will result in the transmission of dengue viruses.

Then, the force of infection from mosquitoes to humans is

$$\beta_i^h \left( b_i \frac{M_i}{H_i} \right) \left( \frac{I^v_i}{M_i} \right) = \beta_i^h b_i I^v_i H_i.$$

There are $S_i^h$ susceptible humans, so the infectious mosquitoes can infect

$$\frac{b_i \beta_i^h I^v_i}{H_i} S_i^h$$

humans.

The exposed hosts either die due to the natural cause at the rate of $\mu_i^h$ or move the the infectious class at the rate $\nu_i^h$ after showing the clinical symptoms of dengue disease. Infectious hosts either die due to the natural cause at the rate of $\mu_i^h$ or recover at the rate $\gamma_i^h$. $\rho_i^h$ is the rate at which recovered humans lose their immunity and join the susceptible class again.

The model parameters $b_i$, $\mu_i^v$, $\nu_i^v$ depend on temperature. These parameters are defined as follows$^{[22;25]}$:

The mosquito biting rate is defined as:

$$b_i = -0.00014T^2 + 0.027T - 0.322$$

The mosquito death rate is given by

$$\mu_i^v = -\ln(-0.000828T^2 + 0.0367T + 0.522)$$
Further, the incubation period of the mosquitoes is defined as:

\[ \nu^v_i = -0.00083T^2 + 0.044T - 0.487 \]

\( a_{ji} \) and \( a_{ij} \) respectively are the movement rates of humans moving from patch \( i \) to patch \( j \) and from patch \( j \) to patch \( i \), \( i, j = 1, 2, 3, \cdots, n, i \neq j \).

With the above assumptions, the system of ordinary differential equations which describes the transmission dynamics of malaria between \( n \) patches\(^{[2;13]} \) is given by

\[
\begin{align*}
\frac{dS^h_i}{dt} &= \zeta^h_i - \frac{b_i \beta^h_i}{H_i} S^h_i I^v_i + \sum_{j=1}^{n} a_{ij} S^h_j - \sum_{j=1}^{n} a_{ji} S^h_i - \mu^h_i S^h_i + \rho^h_i R^h_i \\
\frac{dE^h_i}{dt} &= \frac{b_i \beta^h_i}{H_i} S^h_i I^v_i + \sum_{j=1}^{n} a_{ij} E^h_j - \sum_{j=1}^{n} a_{ji} E^h_i - (\nu^h_i + \mu^h_i) E^h_i \\
\frac{dI^h_i}{dt} &= \nu^h_i E^h_i + \sum_{j=1}^{n} a_{ij} I^h_j - \sum_{j=1}^{n} a_{ji} I^h_i - (\gamma^h_i + \mu^h_i) I^h_i \\
\frac{dR^h_i}{dt} &= \gamma^h_i I^h_i + \sum_{j=1}^{n} a_{ij} R^h_j - \sum_{j=1}^{n} a_{ji} R^h_i - \rho^h_i R^h_i - \mu^h_i R^h_i
\end{align*}
\]

\( (i, j = 1, 2, 3, \cdots, n, \ i \neq j) \)

Where,

\[
\begin{align*}
S^h_i(t) + E^h_i(t) + I^h_i(t) + R^h_i(t) &= H_i(t) \quad \text{(Total host population of patch} \ i \ \text{in time} \ t) \\
S^v_i(t) + E^v_i(t) + I^v_i(t) &= M_i(t) \quad \text{(Total vector population of patch} \ i \ \text{in time} \ t)
\end{align*}
\]

**Disease Free Equilibrium Point and Basic Reproduction Number**

In this section, we compute an equilibrium point in disease free situation known as disease free equilibrium (DFE) of the system of equations (0.1) and study its stability with help of the basic reproduction number.
Proposition 1. The model (0.1) has a unique disease free equilibrium with the host and vector components $S^*_h$ and $S^*_v$ receptively.

Proof. In the absence of disease infection, $S^h_i = S^{h*}_i > 0$, $S^v_i = S^{v*}_i > 0$ and other state variables $E^h_i = 0$, $E^v_i = 0$, $I^h_i = 0$, $I^v_i = 0$ and $R^h_i = 0$ for $i = 1, 2, 3, \cdots, n$.

The model equations (0.1) for human population in disease free situation is:

$$AS^{h*} = \zeta^h$$

(0.2)

where, \[ A = \text{diag} \left( \mu^h_i + \sum_{j=1}^{n} a_{ji} \right) - \Gamma, \quad \Gamma = \begin{bmatrix} 0 & a_{12} & \cdots & a_{1n} \\ a_{21} & 0 & \cdots & a_{2n} \\ \vdots & \vdots & \ddots & \vdots \\ a_{n1} & a_{n2} & \cdots & 0 \end{bmatrix}, \]

$$\zeta^h = [\zeta^h_1, \zeta^h_2, \cdots, \zeta^h_n]^T, \quad S^h = [S^{h*}_1, S^{h*}_2, \cdots, S^{h*}_n]^T$$

The model equations (0.1) for vector population in disease free situation is:

$$BS^{v*} = \zeta^v$$

(0.3)

where, \[ B = \text{diag} (\mu^v_i), \quad S^v = [S^{v*}_1, S^{v*}_2, \cdots, S^{v*}_n]^T, \quad \zeta^v = [\zeta^v_1, \zeta^v_2, \cdots, \zeta^v_n]^T. \]

Matrix $A$ has all off-diagonal entries negative and each column sum is positive. So, $A$ is non-singular $M$ - matrix. The matrix is an irreducible as it has non-zero non-diagonal elements. So, the matrix must have positive inverse\(^\text{[7]}\). Hence, the system of equations (0.2) has a unique solution $S^{h*} = A^{-1}\zeta^h > 0$.

Further, matrix $B$ is a diagonal matrix with positive diagonal elements. So, $B^{-1}$ exists with positive diagonal elements. Hence, the system of equations (0.3) has a unique solution $S^{v*} = B^{-1}\zeta^v > 0$ and (0.1) has a unique disease free equilibrium with host and vector components $S^{h*} = A^{-1}\zeta^h > 0$ and $S^{v*} = B^{-1}\zeta^v > 0$. \(\square\)

Basic Reproduction Number: When an infective is introduced into a completely susceptible population, the average number of new infections produced by this single infective during its infectious period is called basic reproduction number.

To find the mathematical expression for the basic reproduction number, we order the
variables related to the infections by $E_1^h, E_2^h, \cdots, E_n^h, E_1^v, E_2^v, \cdots, E_n^v, I_1^h, I_2^h, \cdots, I_n^h, I_1^v, I_2^v, \cdots, I_n^v$. We find transmission matrix, $F$ and transition matrix, $V$ and compute the basic reproduction number $R_0$ using Next Generation method\cite{12;36} as,

$$R_0 = \rho \{ FV^{-1} \}$$

(0.4)

For the model equations (0.1),

$$F = \begin{bmatrix}
  0 & 0 & 0 & \text{diag} \left( \frac{b_i \beta_{1i} h_i}{H_i} S_{h1} \right) \\
  0 & 0 & \text{diag} \left( \frac{b_i \beta_{2i} h_i}{H_i} S_{v1} \right) & 0 \\
  0 & 0 & 0 & 0 \\
  0 & 0 & 0 & 0 \\
\end{bmatrix},
V = \begin{bmatrix}
  V_{11} & 0 & 0 & 0 \\
  0 & V_{22} & 0 & 0 \\
  V_{31} & 0 & V_{33} & 0 \\
  0 & V_{42} & 0 & V_{44} \\
\end{bmatrix}
$$

Here,

$$V_{11} = \begin{bmatrix}
  \sum_{j \neq 1} a_{j1} + \nu_1^h + \mu_1^h & -a_{12} & \cdots & -a_{1n} \\
  -a_{21} & \sum_{j \neq 2} a_{j2} + \nu_2^h + \mu_2^h & \cdots & -a_{2n} \\
  \vdots & \vdots & \ddots & \vdots \\
  -a_{n1} & -a_{n2} & \cdots & \sum_{j \neq n} a_{jn} + \nu_n^h + \mu_n^h \\
\end{bmatrix}$$

$$V_{22} = \text{diag} (\nu_1^v + \mu_1^v), V_{31} = \text{diag} (-\nu_1^h),$$

$$V_{33} = \begin{bmatrix}
  \sum_{j \neq 1} a_{j1} + \gamma_1^h + \mu_1^h & -a_{12} & \cdots & -a_{1n} \\
  -a_{21} & \sum_{j \neq 2} a_{j2} + \gamma_2^h + \mu_2^h & \cdots & -a_{2n} \\
  \vdots & \vdots & \ddots & \vdots \\
  -a_{n1} & -a_{n2} & \cdots & \sum_{j \neq n} a_{jn} + \gamma_n^h + \mu_n^h \\
\end{bmatrix}$$

$$V_{42} = \text{diag} (-\nu_1^v), V_{44} = \text{diag} (\mu_1^v)$$
The matrices $V_{11}$ and $V_{33}$ are irreducible non-negative $M$-Matrices. So inverses of the matrices $V_{11}$ and $V_{33}$ exist. Inverses of the matrices $V_{22}$, $V_{31}$, $V_{42}$ and $V_{44}$ exist being diagonal matrices. Hence, $V^{-1}$ exists and basic reproduction number, $R_0$ is given by

$$R_0 = \rho\{FV^{-1}\}$$

**Proposition 2** (Local Stability). *The disease free equilibrium point of the model equations (0.1) is locally asymptotically stable if $R_0 < 1$ and unstable if $R_0 > 1$.*

**Proof.** Jacobian matrix for the system of equations (0.1) at disease free equilibrium is given by

$$Z = \begin{bmatrix} X & Y \\ 0 & F - V \end{bmatrix}$$

Matrix $Z$ is triangular matrix. So, the stability of the system of equations (0.1) depends on matrices $X$ and $F - V$. Matrix $X$ can be written as

$$X = \begin{bmatrix} -[\text{diag}(\mu^h_i + \sum_{j=1}^n a_{ji} - \Gamma)] & 0 & \text{diag}(\rho^h_i) \\ 0 & -\text{diag}(\mu^v_i) & 0 \\ 0 & 0 & -[\text{diag}(\mu^h_i + \sum_{j=1}^n a_{ji} - \Gamma + \rho^h_i)] \end{bmatrix}$$

Where the Matrix $\Gamma$ is defined in Proposition 1. Matrix $-X$ is non-singular $M$-matrix since each column sum of the matrix is positive and each non diagonal element is non-positive. Hence, the matrix $-(X) = X$ has eigenvalues with negative real parts\cite{7} and the stability of the model (0.1) depends on the matrix $F - V$ only. Here, matrix $F$ is non-negative matrix and $V$ is a non-singular $M$-matrix. So, the matrix will have eigenvalues with negative real parts if $\rho\{FV^{-1}\} < 1$\cite{36}, i.e., $R_0 < 1$. Thus, the disease free equilibrium is locally asymptotically stable if $R_0 < 1$. If $R_0 > 1$, then $s(F - V) > 0$. Which shows that at least one eigenvalue lies in right half plane. So, the disease free equilibrium is unstable if $R_0 > 1$.

In two patch setting, the local basic reproduction numbers of patch 1 and patch 2 are
respectively computed as
\[ R_{01} = \sqrt{\frac{b_1^2 S_1^h S_1^{v_1} \beta_1 \mu_1 \nu_1^h \nu_1^v}{\mu_1^h N_1^h (\mu_1^h + a_{21} + \gamma_1^h) (\mu_1^h + a_{21} + \nu_1^h) (\mu_1^h + \nu_1^h)}} \]
\[ R_{02} = \sqrt{\frac{b_2^2 S_2^h S_2^{v_2} \beta_2 \mu_2 \nu_2^h \nu_2^v}{\mu_2^h N_2^h (\mu_2^h + a_{12} + \gamma_2^h) (\mu_2^h + a_{12} + \nu_2^h) (\mu_2^h + \nu_2^h)}} \]

The basic reproduction \( R_0 \) is obtained as:
\[ R_0 = \frac{1}{2} (a R_{01}^2 + b R_{02}^2) + \frac{1}{2} \sqrt{(a R_{01}^2 + b R_{02}^2)^2 - 4c R_{01}^2 R_{02}^2} \]

where,
\[ a = \frac{g_1 n_1 (a_{12} a_{21} \nu_2^h + \nu_1^h g_2 n_2)}{\nu_1^h (-a_{12} a_{21} + g_1 g_2) (-a_{12} a_{21} + n_1 n_2)} \]
\[ b = \frac{g_2 n_2 (a_{12} a_{21} \nu_1^h + g_1 n_1 \nu_2^h)}{\nu_2^h (-a_{12} a_{21} + g_1 g_2) (-a_{12} a_{21} + n_1 n_2)} \]
\[ c = \frac{g_1 n_1 g_2 n_2}{(a_{12} \gamma_1^h + g_3 \mu_2^h + g_3 \gamma_2^h + g_2 \mu_1^h) (a_{12} \nu_1^h + n_3 \mu_2^h + n_3 \nu_2^h + n_2 \mu_1^h)} \]
\[ g_1 = \mu_1^h + a_{21} + \gamma_1^h, \quad g_2 = \mu_2^h + a_{12} + \gamma_2^h, \quad g_3 = a_{21} + \gamma_1^h \]
\[ n_1 = \mu_1^h + a_{21} + \nu_1^h, \quad n_2 = \mu_2^h + a_{21} + \nu_2^h, \quad n_3 = a_{21} + \nu_1^h \]

**Numerical Results and Discussion**

Simulations are carried out to observe the impact of temperature and human movement in the transmission dynamics of malaria in two patch setting. The following data are used:
\[ H_1 = 50000, \quad H_2 = 25000, \quad \zeta_1^v = 25000, \quad \zeta_2^v = 12000, \quad \mu_1^h = \mu_2^h = 0.00004029, \quad \nu_1^h = \nu_1^v = 0.083, \quad \beta_1^h = \beta_2^h = 0.24, \quad \beta_1^v = \beta_2^v = 0.083, \quad \gamma_1^h = \gamma_2^h = 0.00265, \quad \rho_1^h = \rho_2^h = 0.000017. \]
The model parameters \( b_1, b_2, \mu_1^v, \mu_2^v, \nu_1^v, \nu_2^v \) are temperature dependent.\(^{22,25}\)

Figure (1) to figure (4) show that the infective human population increases initially due to interaction of humans with infectious vectors and later the infective population size starts to decrease due to natural death and recovery from malaria disease. Patch 1 is
Figure 1: Dynamics of infectious hosts of patch 1 without host movement between the patches.

Figure 2: Dynamics of infectious hosts of patch 2 without host movement between the patches.

considered to have temperature within the range of 20°C to 30°C and patch 2 is considered to have temperature within the range to 15°C to 25°C. Here, patch 1 is high disease prevalent patch in comparison to patch 2. Figure (1) and (2) are simulated to observe impact of temperature on the disease prevalence without human movement. According to [22], the optimal malaria transmission occurs at 25°C. In figure (1), the infective population increases when temperature increases from 20°C to 25°C and decreases at 30°C. In figure (2), the disease prevalence increases with the increasing temperature as the highest temperature in the second patch is 25°C.

Figure (3) and figure (4) describe the impact of human movement when humans move in one direction only. Figure (3) shows that when humans are allowed to move to patch 1 from patch 2 only, more humans of patch 1 is observed infected while only few in patch 2. Meanwhile, when only the humans from patch 1 are allowed to moved to patch 2, more infections in patch 2 can be seen and few cases of infection in patch 1 (Figure (4)). Thus, proper human movement can help in decreasing the burden of malaria disease.

In epidemiology, basic reproduction number is considered a metric which determines whether the disease persists or dies out. Greater the value of the number, higher the disease prevalence. Figures (5) and (6) demonstrate role of human movement on local
Figure 3: Dynamics of infectious hosts of patch 1 and patch 2 with $a_{21} = 0$.

Figure 4: Dynamics of infectious hosts of patch 1 and patch 2 with $a_{12} = 0$.

Figure 5: Basic reproduction number of patch 1 against movement rates.

Figure 6: Basic reproduction number of patch 2 against movement rates.

basic reproduction numbers $R_{01}$ and $R_{02}$. It is observed that due to human movement from high disease prevalent patch to the low disease prevalent patch ($a_{21}$), $R_{01}$ decreases and $R_{02}$ increases. Similarly, increase in human movement from low patch 2 to the patch 1 ($a_{12}$) causes increase in $R_{01}$ and decrease in $R_{02}$. Thus, human movement from high prevalent patch to the low prevalent patch contributes in increasing the disease prevalence in low disease prevalent patch and decreasing the disease prevalence in high prevalent patch. Also, human movement from low prevalent patch to the high prevalent patch...
patch contributes in increasing the disease prevalence in high disease prevalent patch and decreasing the disease prevalence in low prevalent patch.

Figure 7: Basic reproduction number against temperature.

Figure 8: Basic reproduction number against temperature.

Figure (7) and (8) are drawn for local basic reproduction numbers against temperature. The figures show that the malaria disease prevalence increases along with temperature upto the $25^\circ C$ temperature\textsuperscript{[22]} when temperature level is more than $25^\circ C$, disease prevalence starts decreasing.

Figure 9: Global basic reproduction number against local basic reproduction numbers.

The local basic reproduction numbers determine the value of global basic reproduc-
tion number. With the increase/decrease in local basic reproduction numbers, there is increase/decrease in global basic reproduction number. It shows that the disease dominances in the local patches determine the global disease dominance (Figure (9)).

Conclusions

Malaria disease is one of the leading infectious diseases which is causing millions of cases worldwide. The disease is increasing its dominance due to human movement and changing climatic situations. A muti-patch SEIRS-SEI epidemic compartmental model is developed to study impact of temperature and human movement in malaria disease transmission dynamics in the present work.

Climatic factors like temperature has a significant impact on malaria disease transmission. Temperature affects mainly the biting behaviour of mosquitoes, incubation period and death rate of mosquitoes. These are incorporated in the present work. It is shown that optimal transmission of the disease occurs at $25^\circ C$ as in \cite{22} and temperature below $16^\circ C$ is not favourable for malaria.

Human movement helps in spreading the disease from endemic regions to the non-endemic regions. It contributes in further expansion of the disease. In two patch setting, it is noticed that the disease prevalence can be reduced by managing human movement between high and low disease prevalent patches. We have discussed the local stability of disease free equilibrium point of the model equations. The basic reproduction numbers which can indicate whether disease is going the invade the population or dying out is computed. With help of this number, it is observed that the disease free equilibrium point is locally asymptotically stable when basic reproduction number $R_0 < 1$ and unstable when $R_0 > 1$. Simulated results show that, basic reproduction number depends on temperature and human movement. The prevalence of disease can increase or decrease with temperature and host movement from one patch to the other. Present work suggests that the burden of the disease can be reduced by managing the host movement between low and high disease prevalent patches. The optimal temperature for malaria disease transmission is $25^\circ C$. 

13
List of Abbreviations
SEIRS: Susceptible-Exposed-Infectious-Recovered-Susceptible; SEI: Susceptible-Exposed-Infectious; DFE: Disease Free Equilibrium; WHO: World Health Organization

Ethics approval and consent to participate
Not applicable

Consent for publication
The author read and approved the final revised manuscript for the publication.

Funding
Not applicable

Competing interests
The author declares that no competing interests exist.

References

[1] FB Agusto, AB Gumel, and PE Parham, Qualitative assessment of the role of temperature variations on malaria transmission dynamics, Journal of Biological Systems 23 (2015), 1 – 34.

[2] J Arino and P Van den Driessche, Disease spread in metapopulations, Field institute communications 48 (2006), 1–12.

[3] J Arino, A Durcot, and P Zongo, A meta population model for malaria with transmission blocking partial immunity in hosts, J Math Biol 64 (2012), 423 – 448.

[4] J Arino and P van den Driessche, A multicity epidemic model, Math. Popul. Studies 10 (2003), 175–193.

[5] JL Aron, Mathematical modeling of immunity to malaria, Math BioSci 90 (1988), 385 – 396.
[6] P Auger, E Kouokam, G Sallet, M Tchuente, and B Tsanou, *The Ross-Macdonald model in patchy environment*, Mathematical Biosciences 216 (2008), 123 – 131.

[7] A Berman and RJ Plemmons, *Non-negative matrices in mathematical sciences*, Academic press, New York, 1979.

[8] G Bhuju, GR Phaijoo, and DB Gurung, *Mathematical study on impact of temperature in malaria disease transmission dynamics*, Advances in Computer Sciences 1 (2018), 1 – 8.

[9] N Chitnis, JM Cushing, and JM Hyman, *Bifurcation analysis of mathematical model for malaria transmission*, Siam J Appl Math 67 (2006), 24 – 45.

[10] C Chiyaka, W Garira, and S Dube, *Transmission of endemic malaria in partially immune population*, Math Comput Model 46 (2007), 806 – 822.

[11] C Cosner, JC Beier, RS Cantrell, D Impoinvil, L Kapitanski, MD potts, A Troyo, and S Ruan, *The effects of human movement on the persistence of vector borne diseases*, Journal of Theoretical Biology 258 (2009), 550 – 560.

[12] O Diekmann, JAP Heesterbeek, and JAJ Metz, *On the definition and computation of the basic reproduction ratio $R_0$ in models for infectious diseases in heterogeneous populations*, Journal of Mathematical Biology 28 (1990), 365–382.

[13] YH Hsieh, P van den Driessche, and L Wang, *Impact of travel between patches for spatial spread of disease*, Bulletin of mathematical biology 69 (2007), 1355–1375.

[14] WO Kermack and AG McKendrick, *A contribution to the mathematical theory of epidemics*, Proceedings of the Royal Society of London 115 (1927), 700–721.

[15] _______; *Contribution to the mathematical theory of epidemics - I*, Bulletin of Mathematical Biology 53 (1991), 33–55.

[16] S Lee and C Castillo-Chavez, *The role of residence times in two-patch dengue transmission dynamics and optimal strategies*, Journal of Theoretical Biology 374 (2015), 152 – 164.
[17] MY Li and Z Shuai, *Global stability of an epidemic model in a patchy environment*, Canadian Applied Mathematics Quarterly 17 (2009), 175–187.

[18] Li Jia, *Malaria model with stage-structured mosquitoes*, Mathematical Biosciences and Engineering 8 (2011), 1272 – 1296.

[19] Y Lou and XQ Zhao, *A climate based malaria transmission model with structured vector population*, SIAM J. Appl. Math 70 (2010), 2023 – 2044.

[20] G MacDonald, *The analysis of equilibrium in malaria*, Trop Dis Bull 49 (1952), 813 – 829.

[21] WJM Martens, LW Niessen, J Rotmans, TH Jetten, and AJ McMichael, *Potential impact of global climate change on malaria risk*, Environ Health Perspect 103 (1995), 458 – 464.

[22] EA Mordecai, *Optimal temperature for malaria transmission is dramatically lower than previously predicted*, Ecol Lett 16 (2013), 22 – 30.

[23] GA Ngwa, *Modeling the dynamics of endemic malaria in growing populations*, Discrete Contin Dyn Syst - Ser B 4 (2004), 1173 – 1202.

[24] GA Ngwa and WS Shu, *A mathematical model for endemic malaria with variable human and mosquito populations*, Math Comput Model 32 (2000), 747– 763.

[25] A Nwankwo and D Okuonghae, *A mathematical model for the population dynamics of malaria with a temperature dependent control*, Differential Equations and Dynamical Systems (2019), https://doi.org/10.1007/s12591-019-00466-y.

[26] K Okuneye and AB Gumel, *Analysis of temperature and rainfall dependent model for malaria transmission dynamics*, Math Bios 287 (2017), 72 – 92.

[27] PE Parham and E Michael, *Modeling the effects of weather and climate change on malaria transmission*, Environ Helath Perspect 118 (2010), 620 – 626.

[28] AT Peterson, *Shifting suitability for malaria vectors across Africa with warming climates*, BMC Infect Dis 9 (2009), 59.
[29] GR Phaijoo and DB Gurung, *Mathematical model on analysis of awareness in controlling dengue disease*, International Journal of Advanced Research 4 (2016), 999–1006.

[30] ______, *Mathematical study of dengue disease transmission in multi-patch environment*, Applied Mathematics 7 (2016), 1521–1533.

[31] M AL - Rahman, El - Nor Osman, A Ebenezer, and IK Adu, *A SEIR - SEI malaria transmission model with optimal control*, Journal of Advances in Mathematics and Computer Science 28 (2018), 1 – 7.

[32] R Ross, *The prevention of malaria*, London: John Murray (1911).

[33] ______, *Some a priori pathometric equations*, Br Med J 1 (1915), 546 – 547.

[34] ______, *An application of the theory of probabilities to the study of a priori pathometry- I*, Proc R Soc A92 (1916), 204 – 230.

[35] FC Tanser, B Sharp, and D le Sueur, *Potential effect of climate change of malaria transmission in Africa*, Kancet 362 (2003), 1792 – 1798.

[36] P van den Driessche and J Watmough, *Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission*, Math. Biosci. 180 (2002), 29–48.

[37] W Wang and G Mulone, *Threshold of disease transmission in a patch environment*, J. Math. Anal. Appl. 285 (2003), 321–335.

[38] W Wang and XQ Zhao, *An epidemic model in a patchy environment*, Math. Biosci. 112 (2004), 97–112.

[39] WHO, *World malaria report - 2019*, https://www.who.int/malaria/en/.

[40] HM Yang, *A mathematical model for malaria transmission relating global warming and local socioeconomic conditions*, Rev Savde Publica 35 (2001), 224 – 231.
[41] G Zhon, M Noboru, A Githeko, and G Yan, Association between climate variability and malaria epidemic in the east African highlands, Proc Natl Acad Sci USA 101 (2003), 2375 – 2380.