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ANALYZING THE EFFECTS OF TEMPERATURE AND HUMAN MOVEMENT ON MALARIA DISEASE TRANSMISSION DYNAMICS

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Abstract. Vector borne diseases like malaria are spreading worldwide. They have become a major cause of morbidity and mortality. Malaria cases are increasing due to the human movement from one place to the other. Changing temperature level has significant impact on the life cycle, biting behavior and death rates of the mosquitoes which can transmit the disease. In the present work, 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 and local stability is discussed. Numerical results show that the prevalence of the disease can be reduced by managing human movement between the patches and the temperature affects the transmission of malaria disease.

Keywords: Compartmental model, human movement, temperature, basic reproduction number, stability.

AMS Subject Classification: 97R20, 97M10, 93A30.

1. Introduction

Malaria is a vector borne disease which is caused by the protozoan parasites of genus Plasmodium. The World Health Organization 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 [40]. 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 [33, 35]. He developed a simple model, now known as Ross model [34] which explained the relationship between

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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 [16, 17] 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 infectious diseases like dengue, malaria, zika, COVID 19 etc [5, 9, 10, 11, 19, 22, 26, 25, 29, 30, 14].

Malaria disease is sensitive to the climate change. Both increase and fluctuation in temperature are affecting vectors and parasites of malaria disease. This can cause reduced prevalence of the disease in some areas, while it may increase in other [23, 29, 32, 36]. 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 [21], Zhon et al. [42] studied the relation between climate variability and malaria disease. The researchers in [1, 8, 24, 27, 28, 41] carried out the researches to oberve 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 to 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 [2]. Multi-patch models of the infectious diseases are studied by [6, 15, 18, 20, 31, 38, 39]. Arino et al. [3, 4] discussed the spreading of disease in metapopulations. Cosner et al. [12] focused their research on the effects of human movement on the persistence of vector borne diseases.

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

2. Model Formulation and Description

We consider SEIRS - SEI multi - patch model with n patches which are connected by human movement. 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_i^h, Exposed E_i^h, Infectious I_i^h and Recovered R_i^h. The total mosquito (vector) population M_i in each patch is subdivided into the three epidemiological classes: Susceptible S_i^v, Exposed E_i^v and Infectious I_i^v, $i = 1, 2, 3, \cdots n$. Due to the short life span of the mosquitoes, the recovered class in the mosquito population is not considered in the model.

The recruitment rate of host population is ζ_i^h in the patch i. The natural death rate for the host population is μ_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 $\frac{I_i^v}{M_i}$. So, the number of potentially infectious bites given by mosquito per human per day is $\left(b_i\frac{M_i}{H_i}\right)\left(\frac{I_i^v}{M_i}\right)$. Let β_i^h be the probability that a bite from infected mosquito will result in the transmission of malaria disease. Then, the force of infection from mosquitoes to humans is $\beta_i^h\left(\frac{b_iM_i}{H_i}\right)\left(\frac{I_i^v}{M_i}\right)$ *i.e.*, $\beta_i^h\frac{b_iI_i^v}{H_i}$. There are S_i^h susceptible humans, so the infectious mosquitoes can infect $\frac{b_i\beta_i^hI_i^v}{H_i}S_i^h$ humans.

Also, the exposed hosts either die due to the natural cause at the rate of μ_i^h or move the the infectious class at the rate ν_i^h after showing the clinical symptoms of malaria disease. Infectious hosts either die due to the natural cause at the rate of μ_i^h or recover at the rate γ_i^h. ρ_i^h is the rate at which recovered humans lose their immunity and join the susceptible class again.

The model parameters b_i, μ_i^v, ν_i^v depend on temperature level T. These parameters are defined as follows [24, 27]:

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 given by

$$\nu_i^v = -0.00083T^2 + 0.044T - 0.487$$

Let a_{ji} and a_{ij} respectively denote 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 [3, 15] is given by

$$\begin{aligned}
\frac{dS_i^h}{dt} &= \zeta_i^h - \frac{b_i\beta_i^h}{H_i}S_i^hI_i^v + \sum_{j=1}^{n}a_{ij}S_j^h - \sum_{j=1}^{n}a_{ji}S_i^h - \mu_i^hS_i^h + \rho_i^hR_i^h \\
\frac{dE_i^h}{dt} &= \frac{b_i\beta_i^h}{H_i}S_i^hI_i^v + \sum_{j=1}^{n}a_{ij}E_j^h - \sum_{j=1}^{n}a_{ji}E_i^h - (\nu_i^h + \mu_i^h)E_i^h \\
\frac{dI_i^h}{dt} &= \nu_i^hE_i^h + \sum_{j=1}^{n}a_{ij}I_j^h - \sum_{j=1}^{n}a_{ji}I_i^h - (\gamma_i^h + \mu_i^h)I_i^h \\
\frac{dR_i^h}{dt} &= \gamma_i^hI_i^h + \sum_{j=1}^{n}a_{ij}R_j^h - \sum_{j=1}^{n}a_{ji}R_i^h - \rho_i^hR_i^h - \mu_i^hR_i^h \\
\frac{dS_i^v}{dt} &= \zeta_i^v - \frac{b_i\beta_i^v}{H_i}S_i^vI_i^h - \mu_i^vS_i^v \\
\frac{dE_i^v}{dt} &= \frac{b_i\beta_i^v}{H_i}S_i^vI_i^h - (\nu_i^v + \mu_i^v)E_i^v \\
\frac{dI_i^v}{dt} &= \nu_i^vE_i^v - \mu_i^vI_i^v \\
&\quad (i, j = 1, 2, 3, \cdots, n, \quad i \neq j)
\end{aligned} \tag{1}$$

Where,

$$\begin{aligned} S_i^h(t) + E_i^h + I_i^h(t) + R_i^h(t) &= H_i(t) \qquad \text{(Total host population of patch } i \text{ in time } t) \\ S_i^v(t) + E_i^v + I_i^v(t) &= M_i(t) \qquad \text{(Total vector population of patch } i \text{ in time } t) \end{aligned}$$

3. Disease Free Equilibrium Point and Basic Reproduction Number

In this section, we compute an equilibrium point in disease free situation which is known as disease free equilibrium (DFE) of the system of equations (1) and study its stability.

Theorem 3.1. *The model* (1) *has a unique disease free equilibrium with the host and vector components* S_h^* *and* S_v^* *respectively.*

Proof. In the absence of disease infection, $S_i^h = S_i^{h*} > 0$, $S_i^v = S_i^{v*} > 0$ and other state variables $E_i^h = 0$, $E_i^v = 0$, $I_i^h = 0$, $I_i^v = 0$ and $R_i^h = 0$ for $i = 1, 2, 3, \cdots, n$.
The model equations (1) for human population in disease free situation is:

$$AS^{h*} = \zeta^h \tag{2}$$

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

$$\zeta^h = [\zeta_1^h, \zeta_2^h, \cdots, \zeta_n^h]^T, \quad S^h = [S_1^{h*}, S_2^{h*}, \cdots, S_n^{h*}]^T$$

The model equations (1) for vector population in disease free situation is:

$$BS^{v*} = \zeta^v \tag{3}$$

where,

$$B = \text{diag}(\mu_i^v), \quad S^v = [S_1^{v*}, S_2^{v*}, \cdots, S_n^{v*}]^T, \quad \zeta^v = [\zeta_1^v, \zeta_2^v, \cdots, \zeta_n^v]^T.$$

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 [7]. Hence, the system of equations (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 (3) has a unique solution $S^{v*} = B^{-1}\zeta^v > 0$. So, the model equation (1) has a unique disease free equilibrium with host and vector components $S^{h*} = A^{-1}\zeta^h > 0$ and $S^{v*} = B^{-1}\zeta^v > 0$. □

3.1. Basic Reproduction Number.

The basic reproduction number measures the transmission potential of a disease. It is an average number of new infections produced by a single infective during its infectious period when introduced into a completely susceptible population.

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 [13, 37] as,

$$R_0 = \rho\{FV^{-1}\} \tag{4}$$

For the model equations (1),

$$F = \begin{bmatrix} 0 & 0 & 0 & \operatorname{diag}\left(\frac{b_i\beta_i^h}{H_i}S_i^{h*}\right) \\ 0 & 0 & \operatorname{diag}\left(\frac{b_i\beta_i^h}{H_i}S_i^{v*}\right) & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix}, V = \begin{bmatrix} V_{11} & 0 & 0 & 0 \\ 0 & V_{22} & 0 & 0 \\ V_{31} & 0 & V_{33} & 0 \\ 0 & V_{42} & 0 & V_{44} \end{bmatrix}$$

Here,

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

$$V_{22} = \operatorname{diag}\left(\nu_i^v + \mu_i^v\right), V_{31} = \operatorname{diag}(-\nu_i^h),$$

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

$$V_{42} = \operatorname{diag}(-\nu_i^v), V_{44} = \operatorname{diag}(\mu_i^v)$$

The matrices V_{11} and V_{33} are irreducible non-negative M-Matrices. So inverses of the matrices V_{11} and V_{33} exist. Further, the matrices V_{22}, V_{31}, V_{42} and V_{44} are diagonal matrices, so their inverses exist. Hence, V^{-1} exists and basic reproduction number, R_0 is given by

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

Theorem 3.2 (Local Stability)**.** *The disease free equilibrium point of the model equations* (1) *is locally asymptotically stable if* $R_0 < 1$ *and unstable if* $R_0 > 1$.

Proof. Jacobian matrix for the system of equations (1) at disease free equilibrium is given by

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

Matrix Z is trianglular matrix. So, the stability of the system of equations (1) depends on matrices X and $F - V$. Here,

$$X = \begin{bmatrix} -[\text{diag}(\mu_i^h + \sum_{j=1}^{n} a_{ji} - \Gamma] & 0 & \text{diag}(\rho_i^h) \\ 0 & -\text{diag}(\mu_i^v) & 0 \\ 0 & 0 & -[\text{diag}(\mu_i^h + \sum_{j=1}^{n} a_{ji} - \Gamma + \rho_i^h] \end{bmatrix}$$

Where the Matrix Γ is defined in Theorem 3.1. 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 [7] and the stability of the model (1) depends on the matrix $F - V$ only. F is non-negative matrix and V is a non-singular M-matrix. So, the matrix will have eigenvalues with negative real parts if $\rho\{FV^{-1}\} < 1$ [37], 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

$$R_{01} = \sqrt{\frac{b_1^2 S_1^{h*} S_1^{v*} \beta_1^h \beta_1^v \nu_1^h \nu_1^v}{\mu_1^v N_1^{h^2} (\mu_1^h + a_{21} + \gamma_1^h)(\mu_1^h + a_{21} + \nu_1^h)(\mu_1^v + \nu_1^v)}}$$

$$R_{02} = \sqrt{\frac{b_2^2 S_2^{h*} S_2^{v*} \beta_2^h \beta_2^v \nu_2^h \nu_2^v}{\mu_2^v N_2^{h^2} (\mu_2^h + a_{12} + \gamma_2^h)(\mu_2^h + a_{12} + \nu_2^h)(\mu_2^v + \nu_2^v)}}$$

The combined (global) basic reproduction R_0 is obtained as:

$$R_0 = \sqrt{\frac{1}{2}\left(aR_{01}^2 + bR_{02}^2\right) + \frac{1}{2}\sqrt{\left(aR_{01}^2 + bR_{02}^2\right)^2 - 4cR_{01}^2 R_{02}^2}}$$

where,

$$a = \frac{g_1 n_1 (a_{12} a_{21} \nu_2^h + \nu_1^h g_2 n_2)}{\nu_1^h (-a_{12} a_{21} + g_1 g_2)(-a_{12} a_{21} + n_1 n_2)}$$

$$b = \frac{g_2 n_2 (a_{12} a_{21} \nu_1^h + g_1 n_1 \nu_2^h)}{\nu_2^h (-a_{12} a_{21} + g_1 g_2)(-a_{12} a_{21} + n_1 n_2)}$$

$$c = \frac{g_1 n_1 g_2 n_2}{(a_{12}\gamma_1^h + g_3 \mu_2^h + g_3 \gamma_2^h + g_2 \mu_1^h)(a_{12}\nu_1^h + n_3 \mu_2^h + n_3 \nu_2^h + n_2 \mu_1^h)}$$

$$g_1 = \mu_1^h + a_{21} + \gamma_1^h, \quad g_2 = \mu_2^h + a_{12} + \gamma_2^h, \quad g_3 = a_{21} + \gamma_1^h$$

$$n_1 = \mu_1^h + a_{21} + \nu_1^h, \quad n_2 = \mu_2^h + a_{21} + \nu_2^h, \quad n_3 = a_{21} + \nu_1^h$$

4. Simulations 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: $H_1 = 50000$, $H_2 = 25000$, $\zeta_1^v = 25000$, $\zeta_2^v = 12000$, $\mu_1^h = \mu_2^h = 0.00004029$, $\nu_1^h = \nu_1^h =$

0.083, $\beta_1^h = \beta_2^h = 0.24$, $\beta_1^v = \beta_2^v = 0.083$ $\gamma_1^h = \gamma_2^h = 0.00265$, $\rho_1^h = \rho_2^h = 0.000017$. The model parameters b_1, b_2, μ_1^v, μ_2^v, ν_1^v, ν_2^v are temperature dependent [24, 27].

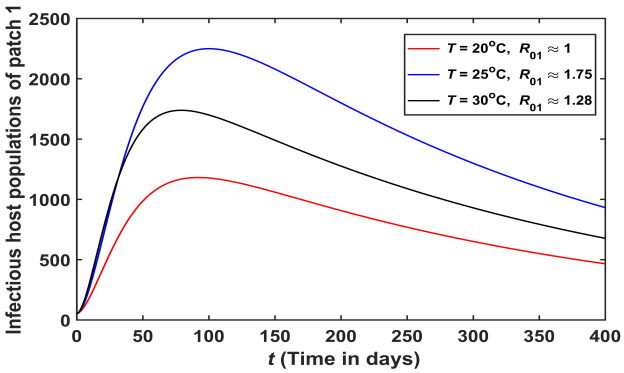

FIGURE 1. Dynamics of infectious hosts of patch 1 without host movement between the patches.

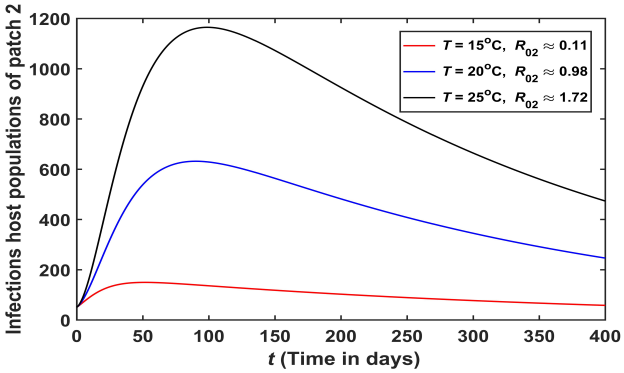

FIGURE 2. Dynamics of infectious hosts of patch 2 without host movement between the patches.

Figure (1) to figure (4) show that the infective human population increases initially and then decreases after attaining its peak. In fact, the population size increases at first 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.

To observe the mathematical results graphically, patch 1 is considered to have temperature within the range of 20^oC to 30^oC and patch 2 is considered to have temperature within the range to 15^oC to 25^oC. 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 [24], the optimal malaria transmission occurs at 25^oC. In figure (1), the infective population increases when temperature increases from 20^oC to 25^oC and decreases at 30^oC. In figure (2), the disease prevalence increases with the increasing temperature as the highest temperature in the second patch is 25^oC.

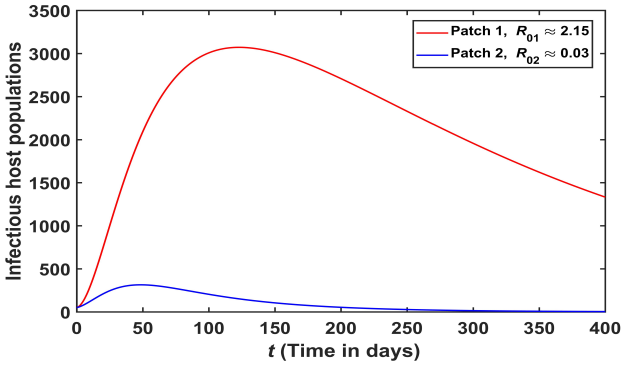

FIGURE 3. Dynamics of infectious hosts of patch 1 and patch 2 with $a_{21} = 0$.

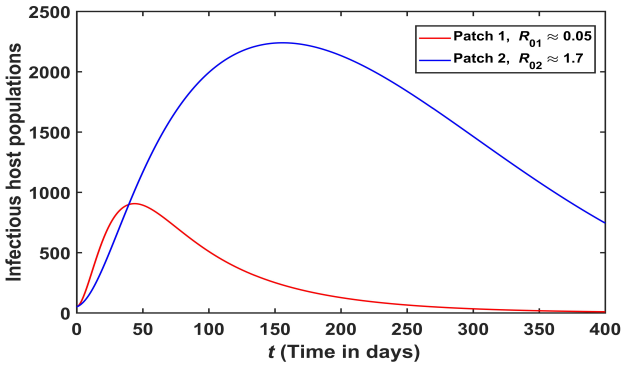

FIGURE 4. Dynamics of infectious hosts of patch 1 and patch 2 with $a_{12} = 0$.

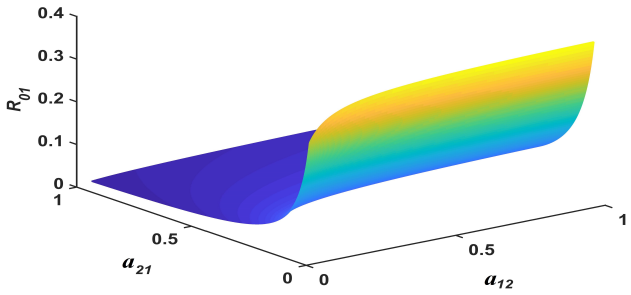

FIGURE 5. Basic reproduction number of patch 1 against movement rates.

Figure (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 burdern of malaria disease.

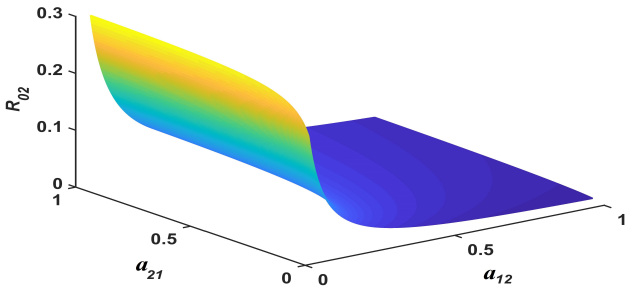

FIGURE 6. Basic reproduction number of patch 2 against movement rates.

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 the role of human movement on local 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 contributes in increasing the disease prevalence in high disease prevalent patch and decreasing the disease prevalence in low prevalent patch.

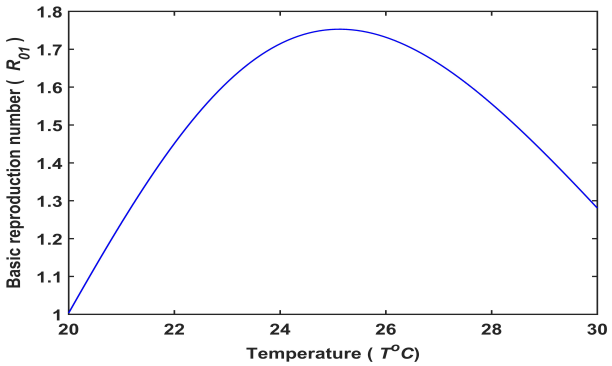

FIGURE 7. Basic reproduction number against temperature.

Figure (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^oC temperature [24] when temperature level is more than 25^oC, disease prevalence starts decreasing. Also, the local basic reproduction number determines the value of global basic reproduction number, R_0. 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)).

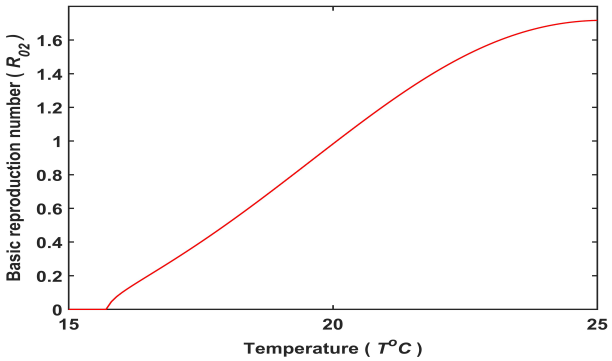

FIGURE 8. Basic reproduction number against temperature.

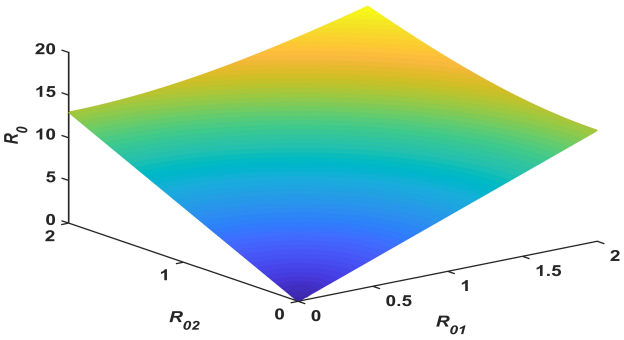

FIGURE 9. Global basic reproduction number against local basic reproduction numbers.

5. Conclusion

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 multi-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 have a significant impact on malaria disease transmission. Temperature affects mainly the biting behavior of mosquitoes, incubation period and death rate of mosquitoes. The dynamics of infected population is observed taking different temperature levels via simulation. The simulated results show that the maximum number of infected humans are observed at 25^oC [24]. Also, the temperature below 16^oC is not favorable for malaria disease transmission.

Human movement is an important driver of spatial spread of infectious diseases. The geographic spread of infectious pathogens is caused by the travelling of infected individuals between areas of active transmission and disease-free areas. Human movement contributes in further expansion of the disease. Simulations of the model are made in the two patch setting to investigate the impact of human movement on spatial spread of malaria disease. It is observed 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 number of the model is computed. 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^oC.

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