Mathematical Biosciences and Engineering

http://www.aimspress.com/journal/MBE

Mathematical Biosciences and Engineering, 16(2): 967–989.
DOI: 10.3934/mbe.2019045
Received: 18 October 2018
Accepted: 24 December 2018
Published: 30 Janurary 2019

Research article

Modeling the imported malaria to north Africa and the absorption effect of the immigrants

Souâd Yacheur[1], **Ali Moussaoui**[1] **and Abdessamad Tridane** [2,*]

[1] Department of Mathematics, University of Tlemcen, B.P.119, Tlemcen 13000 Algeria

[2] Department of Mathematical Sciences, United Arab Emirates University, P.O. Box 15551, Al Ain, UAE

* **Correspondence:** Email: a-tridane@uaeu.ac.ae; Tel: +97137136305.

Abstract: As Malaria represents one of the major health burdens in Africa, there is a risk of reappearance of this vector-borne disease in malaria-free or low risk countries such as those in North Africa. One of the factors that can lead to this situation is the flow of sub-Saharan immigrants trying to reach Europe through North Africa. In this work, we investigate such a possibility via a mathematical model. We assume that the immigrant (non-locals) population has a carrying capacity that limits their numbers in the host country, and we study how they might contribute to the disease spread. Our analysis gave conditions of the persistence of the disease and showed that the non-local population could have a positive effect by reducing the spread of Malaria.

Keywords: malaria; strains; sub-saharan immigrants; carrying capacity; basic reproduction number; stability; persistence

1. Introduction

With 216 million infection cases in 2016 and around 445000 deaths in the same year [52], Malaria is still one of the infectious diseases that has a huge burden on the world global health, particularly in the African continent with more than 90% of the cases worldwide, according to WHO [50].

Although a lot of countries in Africa have already achieved the Malaria free status [51], including the North African countries [9, 24], the road to a complete malaria free continent is still long. One of the reasons of the possible reappearance of malaria in North African countries is the fact that these countries have geographical borders with countries that are not Malaria free (see Figures 1 and 2). Moreover, there has been an increase in the flow of the Sub-Saharan immigrants along the Trans-Saharan migration route [11, 44, 27, 12]. For example, in Algeria which has been categorized as a country with the potential of eliminating local transmission of malaria by 2020 [51], there have

been cases of Malaria [23]. Also, countries such as Morocco and Tunisia are facing the same risk [7, 11, 44, 12].

On the other hand, climate change and the new Trans-Saharan Highway linking Algeria and West Africa have contributed to creating a new map of the distribution of tropical vectors. More precisely, the mosquito *A.gambie* was recently identified as a new type in the Algerian mosquito fauna [11, 23, 12, 43]. This fact will increase the risk of imported malaria to the countries [34].

Hence, there is a need to investigate the impact of the increasing number of immigrants from Sub-Saharan countries on the possible malaria infectious cases. The aim is to answer questions such as: How will the immigrant population affect the potential spread of malaria in this region?

Since the first mathematical model describing the malaria transmission by Ross [38], many researchers have extended Ross's model by considering different factors, such as the latent period of infection in mosquitoes and humans [2, 31]. Other models have been developed by introducing various features of the disease to better understand the epidemiological reality of the disease and the impacts of the external factors [6, 19, 33]. A review of different mathematical models in modelling malaria transmission can be found, for example, in [30].

On the other hand, several papers have investigated the effect of the human migration, and it's role in the spread of the disease (see for example [5, 48]). To investigate Malaria in Africa, mathematical models has been used to study the effects of climate change and migration on the spread of the diseases [32, 35, 49]. Other studies have focused on the possible control measures, like border screening, to reduce the impact of the disease [26]. Recently, these host countries adapted and implemented new policies to limit the number of immigrants [8]. The focus of this paper is to study the impact of such policies. This can be modeled by considering a logistic growth of the non-local population.

The work of Gao and Ruan [22] has focused on a multi-patch malaria model with logistic growth. Their paper investigated on the effects of population movement in the spreading of malaria between patches by analyzing the monotonicity of the basic reproduction number as a function of the travel rates. In their work, they gave the condition of the persistence of the disease in both populations.

This work investigates the effects of the logistic growth of the immigrant population in a hosting country that has a linear growth. The logistic growth is aimed to capture a limited number of immigrant population that is allowed to stay in the country. This assumption allows us to investigate the effects of the carrying capacity of this population on the dynamic of malaria. More precisely, we consider two patches. The first represents the population of the hosting country, which we refer to as the local population. This population is assumed to have a linear growth.The second is the immigrant population (Sub-Saharan immigrants), which we refer to the as non-local population. This population is assumed to have a logistic growth.

Since there is no health care preventative measure to identify the infected immigrants at the entry (borders) to the hosting country, we assume that the flow of immigrants (non-local population) enter directly as susceptible to the hosting country. On the other hand, the categorization of the non-local population to different compartments, cited below, is done inside the hosting country after arrival.

We also assume that there is no movement between the two patches. This assumption is justified by the fact that the non-local population lives with the local population in the same cities, and there are no geographical separations between the two populations.

To our knowledge, there is no study on the effect of the carrying capacity vis-à-vis the linear growth, in two populations, on the spread of Malaria.

The paper is organized as follows: First, we introduce our mathematical model in Section 2. In Section 3, we give the basic mathematical properties of the model and compute the basic reproduction number. The local and global stability of the disease-free equilibrium is treated in Section 4. In Section 5, we investigate the condition of the existence of an endemic equilibrium, and we give the disease persistence result. Finally, we study the possibility of controlling the disease by controlling the carrying capacity of non-locals in Section 6. A conclusion and discussion of the findings of this work are in Section 7.

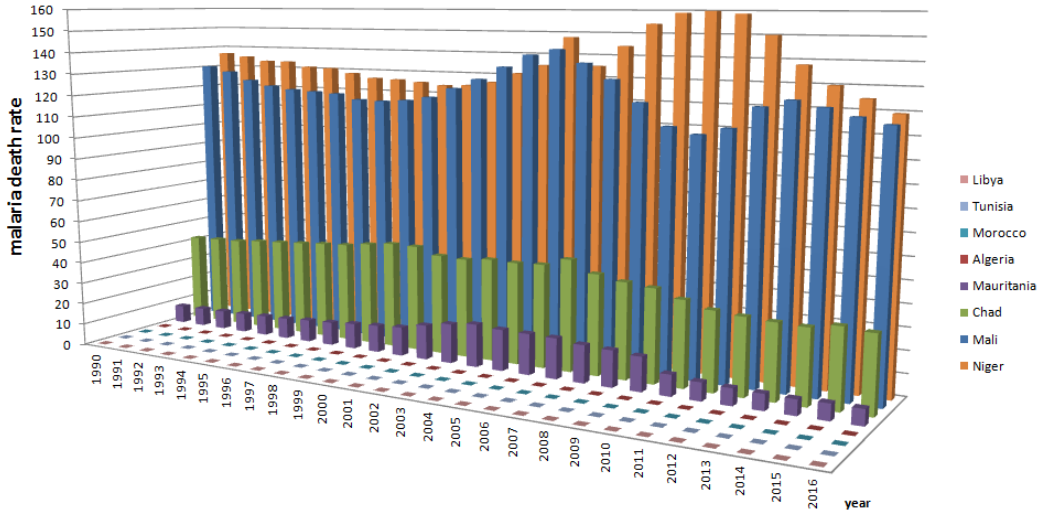

Figure 1. Malaria death rates per 100,000 in north African countries from 1990-2016 [37].

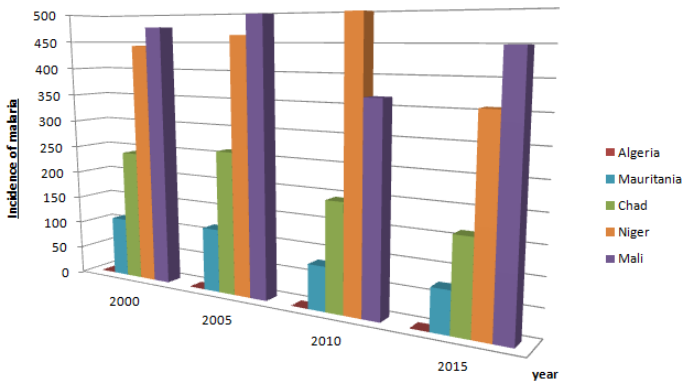

Figure 2. Incidence of malaria per 1,000 population at risk, 2000-2015, where incidence of malaria is defined as the number of new cases of malaria in a year per 1,000 population at risk [37].

2. Presentation of the model

In this work, we opted to use an SEIRS model of malaria (see for example [17, 33]). The benefit of such approach over the existing models is that it allows to us investigate the long time period dynamic of the disease for the local and non-local populations.
The human population is divided into locals, $L(t)$, and non-locals, $N(t)$. The mosquito population is denoted by $M(t)$.
The human sub-populations, L and N, are divided into four classes according to their disease status: susceptible $S(t)$, exposed $E(t)$, infectious $I(t)$ and recovered $R(t)$.

Hence, $L(t) = S_L(t) + E_L(t) + I_L(t) + R_L(t)$ and $N(t) = S_N(t) + E_N(t) + I_N(t) + R_N(t)$. The total population of human, $\sum(t)$, is time-dependent with $\sum(t) = L(t) + N(t)$.

Since mosquitoes need a period of time to develop the parasite and pass from the infected stage to the infectious stage, we divide the mosquito population into three subclasses: susceptible $S_M(t)$, infected $I_{M,1}(t)$ and infectious $I^2_M(t)$ mosquitoes remain infectious for life and have no recovered class [16, 28]. The total mosquito population, $M(t) = S_M(t) + I_{M,1}(t) + I_{M,2}(t)$ is not constant.

We assume that the susceptibles are recruited into the local population by constant input rate Λ_L and have a death rate d_L. However, the non-locals are assumed to have a logistic growth with r_N growth rate and carrying capacity K_N. The death rate of non-locals is d_N. The average biting rate of mosquitoes a and c_i represent, respectively, the transmission probability from infectious mosquitoes to locals ($i = 1$), mosquitoes to non-locals ($i = 2$), locals to mosquitoes ($i = 3$) and non-locals to mosquitoes ($i = 4$). The death rate due to infection α_k, $k = L, N$, and the recovery rate is δ_k, $k = L, N$. Finally, ν_k, $k = L, N$ is the progression rate at which the exposed humans become infectious.

Similarly, we assume that susceptible mosquitoes have a constant recruitment rate Λ_M and die at the rate d_M. μ_M stands for the death rate due to the use of pesticides on the mosquito population. Finally, ν_M represents the rate in which the infected mosquitoes become infectious. All the parameters of the model are represented in Table 1, and flowchart below gives us the different path of model compartments. The equations of the spread of malaria among the local population is given by:

$$\begin{cases} \dfrac{dS_L}{dt} = \Lambda_L - ac_1\dfrac{S_L}{\sum} I_{M,2} - d_L S_L + \delta_L R_L, \\ \dfrac{dE_L}{dt} = ac_1\dfrac{S_L}{\sum} I_{M,2} - (\nu_L + d_L)E_L, \\ \dfrac{dI_L}{dt} = \nu_L E_L - (\gamma_L + \alpha_L + d_L)I_L, \\ \dfrac{dR_L}{dt} = \gamma_L I_L - (\delta_L + d_L)R_L. \end{cases} \tag{2.1}$$

For notation simplification, we call

$$\epsilon_L = \nu_L + d_L, \quad \theta_L = \gamma_L + \alpha_L + d_L, \quad \beta_L = \delta_L + d_L.$$

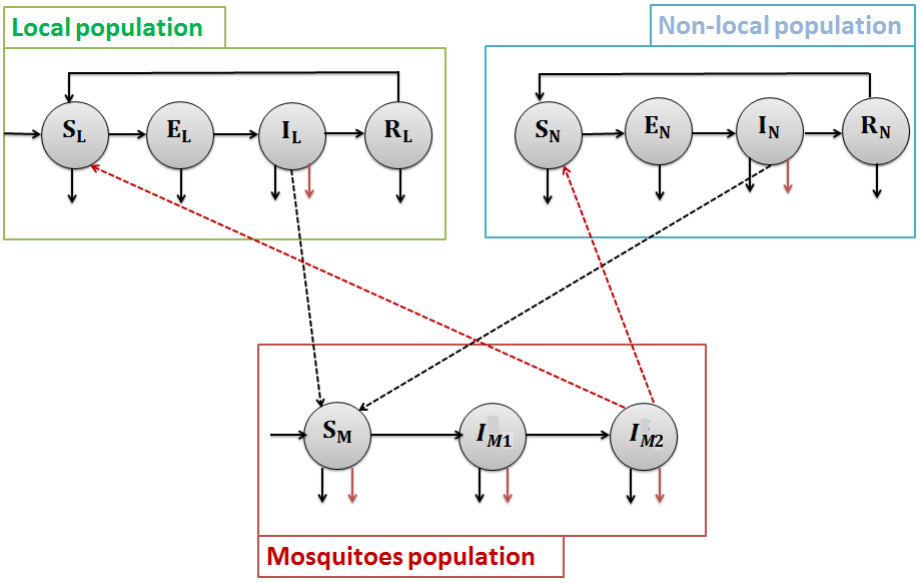

Figure 3. The flowchart of the mathematical model. *The dotted arrows* show the direction of the transmission from infectious human to susceptible mosquito or from infectious mosquitoes to susceptible humans.

The equations of the non-local population is given by:

$$\begin{cases} \dfrac{dS_N}{dt} = r_N S_N(1 - \frac{S_N}{K_N}) - ac_2 \dfrac{S_N}{\sum} I_{M,2} + \delta_N R_N, \\ \dfrac{dE_N}{dt} = ac_2 \dfrac{S_N}{\sum} I_{M,2} - (\nu_N + d_N) E_N, \\ \dfrac{dI_N}{dt} = \nu_N E_N - (\gamma_N + \alpha_N + d_N) I_N, \\ \dfrac{dR_N}{dt} = \gamma_N I_N - (\delta_N + d_N) R_N. \end{cases} \tag{2.2}$$

Here also, we denote

$$\epsilon_N = \nu_N + d_N, \quad \theta_N = \gamma_N + \alpha_N + d_N, \quad \beta_N = \delta_N + d_N.$$

The dynamic of the mosquitoes population is given by:

$$\begin{cases} \dfrac{dS_M}{dt} = \Lambda_M - ac_3 S_M \dfrac{I_L}{\sum} - ac_4 S_M \dfrac{I_N}{\sum} - (\mu_M + d_M) S_M, \\ \dfrac{dI_{M,1}}{dt} = ac_3 S_M \dfrac{I_L}{\sum} + ac_4 S_M \dfrac{I_N}{\sum} - \nu_M I_{M,1} - (\mu_M + d_M) I_{M,1}, \\ \dfrac{dI_{M,2}}{dt} = \nu_M I_{M,1} - (\mu_M + d_M) I_{M,2}. \end{cases} \tag{2.3}$$

With: $b_M = \mu_M + d_M$

Table 1. Parameters interpretation.

Parameters	Description
a	Average biting rate of mosquitoes on a human.
c_1	Transmission probability from infectious mosquitoes to locals.
c_2	Transmission probability from infectious mosquitoes to non-locals.
c_3	Transmission probability from infectious locals to mosquitoes.
c_4	Transmission probability from infectious non-locals to mosquitoes.
Λ_L	Recruitment rate of local population.
r_N	Growth rate of non-local population.
K_N	Carrying capacity of non-local population.
Λ_M	Recruitment rate of mosquitoes.
d_L	Natural death rate for locals.
d_N	Natural death rate for non-locals.
d_M	Natural death rate for mosquitoes.
ν_L	Rate of exposed locals becoming infected.
ν_N	Rate of exposed non-locals becoming infectious.
ν_M	Rate of infected mosquitoes becoming infectious.
α_L	Disease-induced death rate for locals.
α_N	Disease-induced death rate for non-locals.
γ_L	Recovery rate for infected locals.
γ_N	Recovery rate for infected non-locals.
δ_L	Rate of losing immunity of local population.
δ_N	Rate of losing immunity of non-local population.
μ_M	Pesticide-induced death rate for mosquitoes.

3. The model analysis

The first step in analyzing our model is to show that the variables of the model are positive and bounded. Let $\Omega = \mathbb{R}^3_+ \times \mathbb{R}^8_+$ and denote points in Ω by (S, E, I, R), where $S = (S_L, S_N, S_M)$, $E = (E_L, E_N, I_{M,1})$, $I = (I_L, E_N, I_{M,2})$ and $R = (R_L, R_N)$.

Using these notations, we can write all the system in a compact form as:

$$\begin{cases} \dfrac{dS}{dt} &= f_1(S, E, I, R), \\ \dfrac{dE}{dt} &= f_2(S, E, I, R), \\ \dfrac{dI}{dt} &= f_3(S, E, I, R), \\ \dfrac{dR}{dt} &= f_4(S, E, I, R). \end{cases} \tag{3.1}$$

Let,

$$\Gamma = \left\{(S,E,I,R) \in \Omega;\ \frac{\Lambda_L}{d_L+\alpha_L} \le L \le \frac{\Lambda_L}{d_L},\ 0 \le N \le \frac{(r_N+d_N)^2 K_N}{4 r_N d_N},\ 0 \le M \le \frac{\Lambda_M}{b_M}\right\} \tag{3.2}$$

Theorem 3.1. *The system* (3.1) *has a unique non-negative solution for non-negative initial conditions. Moreover,* Γ *is positively invariant and globally attracting for our system.*

Proof. The local existence and uniqueness of solutions follow from the regularity of the function $f = (f_1, f_2, f_3, f_4)$ which is of class C^1 in Γ. For the positivity of the solution, we use the standard approach [45]. Thus Ω is positively invariant.
As the system (3.1) has a unique non-negative solution, straightforward calculations show that $L, N, M \in \Gamma$, and the solution is globally defined. □

The investigated model has two disease free equilibria (DFE) in Γ;

1. E_{01} is DFE without the non-local population i.e $S_N^* = 0$.

$$E_{01} = (S_L^*, 0, 0, 0, 0, 0, 0, 0, S_M^*, 0, 0) = (\frac{\Lambda_L}{d_L}, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_M}{b_M}, 0, 0)$$

2. E_{02} is DFE with full capacity immigrant population i.e $S_N^* = K_N$.

$$E_{02} = (S_L^*, 0, 0, 0, S_N^*, 0, 0, 0, S_M^*, 0, 0) = (\frac{\Lambda_L}{d_L}, 0, 0, 0, K_N, 0, 0, 0, \frac{\Lambda_M}{b_M}, 0, 0)$$

To find the basic reproduction number, we use the method described in [46]. Hence, we can rewrite our model as follows

$$\begin{aligned} \dot{x}_i &= \mathcal{F}_i(x,y) - \mathcal{V}_i(x,y) \qquad for\ \ i = 1, ..., 6 \\ \dot{y}_j &= g_j(x,y) \qquad\qquad\quad for\ \ j = 1, ..., 5 \end{aligned} \tag{3.3}$$

with: $x = (E_L, I_L, E_N, I_N, I_{M,1}, I_{M,2})$ and $y = (S_L, R_L, S_N, R_N, S_M)$.
$\mathcal{F}(x,y)$ is the inflow of new individuals into infected classes,

$$\mathcal{F} \ = \ \left(ac_1 \frac{S_L}{\sum} I_{M,2},\ 0,\ ac_2 \frac{S_N}{\sum} I_{M,2},\ 0,\ ac_3 S_M \frac{I_L}{\sum} + ac_4 \frac{S_M I_N}{\sum}, 0\right)^T$$

and $\mathcal{V}$ contains all other within and out of the infected class, it's given by:

$$\mathcal{V} \ = \ -(-\epsilon_L E_L,\ \nu_L E_L - \theta_L I_L,\ -\epsilon_N E_N,\ \nu_N E_N - \theta_N I_N,\ -(\nu_M + b_M) I_{M,1},\ \nu_M I_{M,1} - b_M I_{M,2})^T .$$

Let $F = D\mathcal{F}|_{(S^*,0)}$ and $V = D\mathcal{V}|_{(S^*,0)}$ be the Jacobian matrices of the maps $\mathcal{F}$ and $\mathcal{V}$, evaluated at the DFE. Following Van den Driessche and Watmough [46], the matrix FV^{-1} is well defined and is the next generation matrix that we denote $K = FV^{-1}$.

$$K \ = \ \begin{pmatrix} 0 & 0 & 0 & 0 & ac_1 \frac{S_L^*}{\sum^*} \frac{\nu_M}{(\nu_M+b_M)b_M} & ac_1 \frac{S_L^*}{\sum} \frac{1}{b_M} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & ac_2 \frac{S_N^*}{\sum^*} \frac{\nu_M}{(\nu_M+b_M)b_M} & ac_2 \frac{S_N^*}{\sum} \frac{1}{b_M} \\ 0 & 0 & 0 & 0 & 0 & 0 \\ ac_3 \frac{S_M^*}{\sum^*} \frac{\nu_L}{\epsilon_L \theta_L} & ac_3 \frac{S_M^*}{\sum^*} \frac{1}{\theta_L} & ac_4 \frac{S_M^*}{\sum^*} \frac{\nu_N}{\epsilon_N \theta_N} & ac_4 \frac{S_M^*}{\sum^*} \frac{1}{\theta_N} & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$\mathcal{R}_0$ is the spectral radius of the next generation matrix, $\mathcal{R}_0 = \rho(K)$.

For $DFE = E_{02}$, we have

$$\mathcal{R}_0 = \frac{a}{\frac{\Lambda_L}{d_L} + K_N} \sqrt{\frac{\Lambda_M}{b_M} \frac{\nu_M}{b_M(\nu_M + b_M)} \left(c_2 c_4 K_N \frac{\nu_N}{\epsilon_N \theta_N} + c_1 c_3 \frac{\Lambda_L}{d_L} \frac{\nu_L}{\epsilon_L \theta_L} \right)}. \tag{3.4}$$

It is easy to prove that we can write $\mathcal{R}_0$ as follow:

$$\mathcal{R}_0 = \frac{1}{K_N + \frac{\Lambda_L}{d_L}} \sqrt{\left(K_N \mathcal{R}_0^N\right)^2 + \left(\frac{\Lambda_L}{d_L} \mathcal{R}_0^L\right)^2}, \tag{3.5}$$

where $\mathcal{R}_0^L$ and $\mathcal{R}_0^N$ are defined by

$$\mathcal{R}_0^L = \frac{a}{\frac{\Lambda_L}{d_L}} \sqrt{\frac{\Lambda_M}{b_M} \frac{\nu_M}{b_M(\nu_M + b_M)} \left(c_1 c_3 \frac{\Lambda_L}{d_L} \frac{\nu_L}{\epsilon_L \theta_L} \right)}, \tag{3.6}$$

and

$$\mathcal{R}_0^N = \frac{a}{K_N} \sqrt{c_2 c_4 \frac{\nu_M \Lambda_M}{b_M^2(\nu_M + b_M)} K_N \frac{\nu_N}{\epsilon_N \theta_N}}, \tag{3.7}$$

where $\mathcal{R}_0^L$ represents the basic reproduction number of the local sub-population in the absence of the non-local sub-population, and $\mathcal{R}_0^N$ is the basic reproduction number of the non-local sub-population in the absence of the local sub-population.

4. Stability of the disease-free equilibria points

In this section, we investigate the conditions of the local and global stability of the disease-free equilibria points.

4.1. The local stability

By linearizing the system of differential equations (3.3), we obtain the Jacobian matrix J^* that can be written in a block structure

$$J^* = \begin{pmatrix} F - V & 0 \\ J_1 & J_2 \end{pmatrix}$$

Theorem 4.1. *1. The disease-free equilibrium point E_{01} is unstable.*

2. The disease-free equilibrium point E_{02} is locally asymptotically stable if and only if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof. The eigenvalues of the Jacobian matrix J^* are those of $F - V$ and J_2, where J_2 is given by:

$$J_2 = \begin{pmatrix} -d_L & \delta_L & 0 & 0 & 0 \\ 0 & -\beta_L & 0 & 0 & 0 \\ 0 & 0 & r_N(1 - 2\frac{S_N^*}{K_N}) & \delta_N & 0 \\ 0 & 0 & 0 & -\beta_N & 0 \\ 0 & 0 & 0 & 0 & -b_M \end{pmatrix}.$$

The spectrum of the matrix of J_2 is given by $\lambda(J_2) = \left\{-d_L, -\beta_L, r_N(1 - 2\frac{S_N^*}{K_N}), -\beta_N, -b_M\right\}$.

1 When $S_N^* = 0$, J_2 has one eigenvalue with positive real part, so it implies that the E_{01} is unstable.
2 When $S_N^* = K_N$, all the eigenvalues J_2 have negative real parts, then it remains to show in this case that all the eigenvalues of the matrix $F - V$ have all negative real parts.

We can prove easily that: F is a non-negative matrix and V is non-singular M-matrix. Using Lemma 9.2 [13], our F and V verify all conditions, so we conclude that all the eigenvalues of J^* have a negative real parts if and only if $\mathcal{R}_0 < 1$.

□

If we define the following parameters:

$$\phi_1 = \frac{K_N + \frac{\Lambda_L}{d_L}}{\sqrt{K_N^2 + \frac{\Lambda_L}{d_L}^2}}, \quad \phi_2 = \frac{\min\left(K_N, \frac{\Lambda_L}{d_L}\right)}{\max\left(K_N, \frac{\Lambda_L}{d_L}\right)},$$

then we have the following remark,

Remark 1. 1) If $\mathcal{R}_0^N < 1$ and $\mathcal{R}_0^L < 1$, then $\mathcal{R}_0 < 1$.

2) If $\max\left(\mathcal{R}_0^N, \mathcal{R}_0^L\right) \leq \phi_1$, then $\mathcal{R}_0 \leq 1$.
3) If $\max\left(\mathcal{R}_0^N, \mathcal{R}_0^L\right) \leq \sqrt{2}\phi_2$, then $\mathcal{R}_0 \leq 1$.
4) If $\min\left(\mathcal{R}_0^N, \mathcal{R}_0^L\right) > \phi_1$, then $\mathcal{R}_0 > 1$.
5) If $\min\left(\mathcal{R}_0^N, \mathcal{R}_0^L\right) > \dfrac{\sqrt{2}}{\phi_2}$, then $\mathcal{R}_0 > 1$.

Moreover, we have 2) implies 3) and 5) implies 4).

Assertions 2 and 3 show that it is possible to have $\mathcal{R}_0^i \geq 1$, $i = N, L$ yet $\mathcal{R}_0$ is less than 1. On the other hand, if the $\mathcal{R}_0^L$ and $\mathcal{R}_0^N$ are both bigger than 1, then from (3.5) we get $\mathcal{R}_0 > 1$. Therefore, the persistence of malaria in both populations does not necessarily lead to an epidemic of the disease in the total population. In fact, assertions 4 and 5 give such conditions, on the basic reproductions of the two populations, that could result in the persistence of the disease.

4.2. The global stability

To prove the global stability of E_{02}, we use the approach given in [46]. Hence, our model can be written as follows:

$$\dot{x} = (F - V)x - \psi(x, y) \tag{4.1}$$

with

$$\psi(x,y) = \begin{pmatrix} ac_1\left(\frac{S_L^*}{\sum_2^*} - \frac{S_L}{\sum}\right)I_M^2 \\ 0 \\ ac_2\left(\frac{S_N^*}{\sum_2^*} - \frac{S_N}{\sum}\right)I_M^2 \\ 0 \\ ac_3\left(\frac{S_M^*}{\sum_2^*} - \frac{S_M}{\sum}\right)I_L + ac_4\left(\frac{S_M^*}{\sum_2^*} - \frac{S_M}{\sum}\right)I_N \\ 0 \end{pmatrix}.$$

If $\mathcal{R}_0 < 1$ and the total human population $\sum \in \left[\frac{\Lambda_L}{d_L} + K_N, \frac{\Lambda_L}{d_L} + \frac{(r_N + d_N)^2}{4r_N d_N}K_N\right]$, then E_{02} is globally asymptotically stable [46].
Using remark 1, we have the following global stability result.

Proposition 1. *If* $\mathcal{R}_0 < \dfrac{\sqrt{\min(K_N, \frac{\Lambda_L}{d_L})}}{2\max(K_N, \frac{\Lambda_L}{d_L})}$ *then* E_{02} *is globally asymptotically stable.*

Proof. To prove this result, we use the Barbashin-Krasovskii theorem [25] [Theorem 4.2, page 124]. We denoted by $x = (S, E, I, R)$ and we consider the continuous scalar function V define by,

$$V = ac_3\frac{\Lambda_M}{b_M}\frac{1}{\theta_L}(\frac{\nu_L}{\epsilon_L}E_L + I_L) + ac_4\frac{\Lambda_M}{b_M}\frac{1}{\theta_N}(\frac{\nu_N}{\epsilon_N}E_N + I_N) + I_M^1 + \frac{\nu_M + b_M}{\nu_M}I_{M,2}.$$

To show that the equilibrium E_{02} is globally asymptotically stable, we need to show that $\dot{V}(x)$ is globally negative definite i.e $\dot{V}(x) < 0, \quad \forall x \in \mathbb{R}_+^{11} \setminus \{E_{02}\}$.
We have

$$\begin{aligned}
\dot{V} &= ac_3\frac{\Lambda_M}{b_M}\frac{1}{\theta_L}\left(\frac{\nu_L}{\epsilon_L}(ac_1\frac{S_L I_{M,2}}{\sum} - \epsilon_L E_L) + \nu_L E_L - \theta_L I_L\right) \\
&\quad + ac_4\frac{\Lambda_M}{b_M}\frac{1}{\theta_N}\left(\frac{\nu_N}{\epsilon_N}(ac_2\frac{S_N I_{M,2}}{\sum} - \epsilon_N E_N) + \nu_N E_N - \theta_N I_N\right) \\
&\quad + ac_3\frac{S_M I_L}{\sum} + ac_4\frac{S_M I_N}{\sum} - (\nu_M + b_M)I_{M,1} + \frac{\nu_M + b_M}{\nu_M}(\nu_M I_{M,1} - b_M I_{M,2}) \\
&= a^2c_1c_3\frac{\Lambda_M}{b_M}\frac{\nu_L}{\epsilon_L\theta_L}\frac{S_L}{\sum}I_{M,2} + a^2c_2c_4\frac{\Lambda_M}{b_M}\frac{\nu_N}{\epsilon_N\theta_N}\frac{S_N}{\sum}I_{M,2} - b_M\frac{\nu_M + b_M}{\nu_M}I_{M,2}
\end{aligned}$$

$$-a\frac{\Lambda_M}{b_M}(c_3I_L + c_4I_N) + aS_M(c_3\frac{I_L}{\sum} + c_4\frac{I_N}{\sum})$$

Since $L(t) \geq \frac{\Lambda_L}{d_L+\alpha_L}$ and $N(t) \geq 0$, we have $\sum = L(t) + N(t) \geq \frac{\Lambda_L}{d_L+\alpha_L}$.
By the fact that $\frac{\Lambda_L}{d_L+\alpha_L} \geq 1$, we get

$$\begin{aligned}\dot{V} &\leq a^2c_1c_3\frac{\Lambda_M}{b_M}\frac{\nu_L}{\epsilon_L\theta_L}\frac{S_L}{\sum}I_{M,2} + a^2c_2c_4\frac{\Lambda_M}{b_M}\frac{\nu_N}{\epsilon_N\theta_N}\frac{S_N}{\sum}I_{M,2} - b_M\frac{\nu_M+b_M}{\nu_M}I_{M,2}\\ &\quad -a\frac{\Lambda_M}{b_M}(c_3I_L + c_4I_N) + aS_M(c_3I_L + c_4I_N)\\ &= a^2c_1c_3\frac{\Lambda_M}{b_M}\frac{\nu_L}{\epsilon_L\theta_L}\frac{S_L}{\sum}I_{M,2} + a^2c_2c_4\frac{\Lambda_M}{b_M}\frac{\nu_N}{\epsilon_N\theta_N}\frac{S_N}{\sum}I_{M,2} - b_M\frac{\nu_M+b_M}{\nu_M}I_{M,2}\\ &\quad a(c_3I_L + c_4I_N)(S_M - \frac{\Lambda_M}{b_M}).\end{aligned}$$

As $S_L, S_N \leq \sum$ and $S_M \leq M(t) \leq \frac{\Lambda_M}{b_M}$, we obtain

$$\begin{aligned}\dot{V} &\leq a^2c_1c_3\frac{\Lambda_M}{b_M}\frac{\nu_L}{\theta_L\epsilon_L}I_{M,2} + a^2c_2c_4\frac{\Lambda_M}{b_M}\frac{\nu_N}{\theta_N\epsilon_N}I_{M,2} - b_M\frac{\nu_M+b_M}{\nu_M}I_{M,2}\\ &= \frac{b_M(\nu_M+b_M)}{\nu_M}I_{M,2}\left[a^2\frac{\nu_M}{b_M(\nu_M+b_M)}c_1c_3\frac{\Lambda_M}{b_M}\frac{\nu_L}{\theta_L\epsilon_L} + a^2\frac{\nu_M}{b_M(\nu_M+b_M)}c_2c_4\frac{\Lambda_M}{b_M}\frac{\nu_N}{\theta_N\epsilon_N} - 1\right]\end{aligned}$$

$$\dot{V} \leq \frac{b_M(\nu_M+b_M)}{\nu_M}I_{M,2}\left[\frac{\Lambda_L}{d_L}(\mathcal{R}_0^L)^2 + K_N(\mathcal{R}_0^N)^2 - 1\right]. \tag{4.2}$$

Using

$$(K_N + \frac{\Lambda_L}{d_L})^2\mathcal{R}_0^2 = (\frac{\Lambda_L}{d_L}\mathcal{R}_0^L)^2 + (K_N\mathcal{R}_0^N)^2,$$

we have two cases:

Case 1, $K_N \leq \frac{\Lambda_L}{d_L}$.

We replace $K_N(\mathcal{R}_0^N)^2 = \frac{(K_N + \frac{\Lambda_L}{d_L})^2}{K_N}\mathcal{R}_0^2 - \frac{(\frac{\Lambda_L}{d_L})^2}{K_N}(\mathcal{R}_0^L)^2$, we get

$$\begin{aligned}\dot{V} &\leq \frac{b_M(\nu_M+b_M)}{\nu_M}I_{M,2}\left[\frac{\Lambda_L}{d_L}(\mathcal{R}_0^L)^2 + K_N(\mathcal{R}_0^N)^2 - 1\right]\\ &\leq \frac{b_M(\nu_M+b_M)}{\nu_M}I_{M,2}\left[\frac{(2\frac{\Lambda_L}{d_L})^2}{K_N}\mathcal{R}_0^2 - 1\right].\end{aligned}$$

In this case $\mathcal{R}_0 < \frac{\sqrt{\min(K_N, \frac{\Lambda_L}{d_L})}}{2\max(K_N, \frac{\Lambda_L}{d_L})} = \frac{\sqrt{K_N}}{2\frac{\Lambda_L}{d_L}}$ leads to $\dot{V} < 0$.

Case 2, $K_N \geq \dfrac{\Lambda_L}{d_L}$.

We replace $(\dfrac{\Lambda_L}{d_L})(\mathcal{R}_0^L)^2 = \dfrac{(K_N + \dfrac{\Lambda_L}{d_L})^2}{\dfrac{\Lambda_L}{d_L}}\mathcal{R}_0^2 - \dfrac{K_N^2}{\dfrac{\Lambda_L}{d_L}}(\mathcal{R}_0^N)^2$, we get

$$\begin{aligned}\dot{V} &\leq \frac{b_M(\nu_M + b_M)}{\nu_M} I_{M,2}\left[\frac{\Lambda_L}{d_L}(\mathcal{R}_0^L)^2 + K_N(\mathcal{R}_0^N)^2 - 1\right] \\ &\leq \frac{b_M(\nu_M + b_M)}{\nu_M} I_{M,2}\left[\frac{(2K_N)^2}{\dfrac{\Lambda_L}{d_L}}\mathcal{R}_0^2 - 1\right].\end{aligned}$$

In this case $\mathcal{R}_0 < \dfrac{\sqrt{\min(K_N, \dfrac{\Lambda_L}{d_L})}}{2\max(K_N, \dfrac{\Lambda_L}{d_L})} = \dfrac{\sqrt{\dfrac{\Lambda_L}{d_L}}}{2K_N}$ implies $\dot{V} < 0$.

□

In addition to the sharp result of the global asymptotically stability of DEF with respect to $\mathcal{R}_0$, proposition 1, gives a new global stability condition without any condition on the total population $\sum$. Moreover, from (3.5) we can show the global stability of DFE, using the same Lyapunov function, under the condition $\frac{\Lambda_L}{d_L}\left(\mathcal{R}_0^L\right)^2 + K_N\left(\mathcal{R}_0^N\right)^2 < 1$.

5. The endemic equilibrium and the uniform persistence

5.1. The endemic equilibrium

To find the possible endemic equilibria point, $EE = (S_L^*, E_L^*, I_L^*, R_L^*, S_N^*, E_N^*, I_N^* R_N^*, S_M^*, I_{M,1}^*, I_{M,2}^*)$, we define λ_1^*, λ_2^* and λ_3^* as follow:

$$\lambda_1^* = \frac{ac_1 I_{M,2}^*}{\sum^*}, \quad \lambda_2^* = \frac{ac_3 I_L^*}{\sum^*}, \quad \lambda_3^* = \frac{ac_4 I_N^*}{\sum^*}.$$

The coordinates of EE for the human population are given by

$$S_L^* = \frac{\Lambda_L}{d_L} + \frac{A_2 - A_1}{d_L} I_L^* \tag{5.1}$$

$$E_L^* = \frac{\theta_L}{\nu_L} I_L^* \tag{5.2}$$

$$R_L^* = \frac{\gamma_L}{\beta_L} I_L^* \tag{5.3}$$

$$I_L^* = \frac{\Lambda_L \lambda_1^*}{A_1(d_L + \lambda_1^*) - A_2\lambda_1^*} \tag{5.4}$$

$$S_N^* = B_1\frac{c_1}{c_2}\frac{1}{\lambda_1^*}I_N^* \tag{5.5}$$

$$E_N^* = \frac{\theta_N}{\nu_N}I_N^* \tag{5.6}$$

$$R_N^* = \frac{\gamma_N}{\beta_N}I_N^* \tag{5.7}$$

$$I_N^* = \frac{K_N c_2}{r_N(B_1c_1)^2}\lambda_1^*\left(r_N B_1 c_1 + c_2\lambda_1^*(B_2 - B_1)\right). \tag{5.8}$$

with

$$A_1 = \frac{\epsilon_L\theta_L}{\nu_L}, \quad A_2 = \frac{\delta_L\gamma_L}{\beta_L}$$

$$B_1 = \frac{\epsilon_N\theta_N}{\nu_N}, \quad B_2 = \frac{\delta_N\gamma_N}{\beta_N}.$$

Note that $A_2 - A_1 \leq 0$ and $B_2 - B_1 \leq 0$.

Using (5.1), we get

$$L^* = \frac{\Lambda_L}{d_L}\frac{1}{A_1d_L - (A_2 - A_1)\lambda_1^*}(A_1d_L - \lambda_1^*(A_2 - A_1 + \alpha_L))$$

$$N^* = K_N + \frac{c_2K_N}{r_NB_1c_1}\left(B_1(-1 + \frac{r_N}{\epsilon_N}) + B_2(1 + \frac{r_N}{\delta_N}) + r_N\right)\lambda_1^* + \frac{c_2^2K_N}{r_N(B_1c_1)^2}(\frac{B_1}{\epsilon_N} + \frac{B_2}{\delta_N} + 1)(B_2 - B_1)(\lambda_1^*)^2,$$

which conclude the equation of $\sum^*$ as function of λ_1^* as follows,

$$\sum^* = \frac{1}{A_1d_L + (A_1 - A_2)\lambda_1^*}\left[\alpha_0 + \alpha_1\lambda_1^* + \alpha_2(\lambda_1^*)^2 + \alpha_3(\lambda_1^*)^3\right],$$

with

$$\alpha_0 = A_1d_L(\frac{\Lambda_L}{d_L} + K_N),$$

$$\alpha_1 = -\frac{\Lambda_L}{d_L}(A_2 - A_1 + \alpha_L) - (A_2 - A_1)K_N - A_1d_L\frac{c_2K_N}{r_NB_1c_1}\left(B_1(1 - \frac{r_N}{\epsilon_N}) - B_2(1 + \frac{r_N}{\delta_N}) - r_N\right),$$

$$\alpha_2 = \frac{c_2^2K_N}{r_N(B_1c_1)^2}\left(A_1d_L(B_2 - B_1)\left(\frac{B_1}{\epsilon_N} + \frac{B_2}{\delta_N} + 1\right) + (A_1 - A_2)\frac{B_1c_1}{c_2}\left(B_1(-1 + \frac{r_N}{\epsilon_N})\right)\right)$$
$$+\frac{c_2K_N}{r_NB_1c_1}(A_1 - A_2)(B_2(1 + \frac{r_N}{\delta_N}) + r_N),$$

$$\alpha_3 = \frac{c_2^2K_N}{r_N(B_1c_1)^2}\left(\frac{B_1}{\epsilon_N} + \frac{B_2}{\delta_N} + 1\right)(A_1 - A_2)(B_2 - B_1).$$

It is easy to see that $\alpha_0 > 0$ and $\alpha_3 < 0$. Moreover, if $\frac{c_2}{c_1} \geq \frac{\epsilon_N^2\beta_N\theta_N}{d_L(\epsilon_N\beta_N\theta_N - \delta_N\gamma_N\nu_N)}$ and $r_N \in \left[\epsilon_N, \frac{d_L}{c_1}\frac{c_2}{\beta_N\epsilon_N\theta_N}(\beta_N\epsilon_N\theta_N - \delta_N\gamma_N\nu_N)\right]$, then we have $\alpha_1 > 0$ and $\alpha_2 \leq 0$.

The remaining coordinates of EE, with respect to the mosquitoes population are

$$\begin{aligned} I^*_{M,2} &= \frac{\Lambda_M \nu_M \lambda^*}{(b_M+\lambda^*)(\nu_M+b_M)(b_M)}, \\ I^*_{M,1} &= \frac{\Lambda_M \lambda^*}{(b_M+\lambda^*)(\nu_M+b_M)}, \\ S^*_M &= \frac{\Lambda_M}{b_M+\lambda^*}. \end{aligned} \tag{5.9}$$

with $\lambda^* = \lambda^*_2 + \lambda^*_3$.
Notice that we have:

$$\lambda^* = \frac{a}{\sum^*}(c_3 I^*_L + c_4 I^*_N), \tag{5.10}$$

$$\frac{1}{ac_1}\lambda^*_1 \sum^* = I^*_{M,2}. \tag{5.11}$$

From (5.9), (5.10) and (5.11), we get the two following equations of λ^* as function of λ^*_1 as follow:

$$\lambda^* = \frac{b_M \lambda^*_1 \sum^*}{(\Lambda_M a c_1 c - \lambda^*_1 \sum^*)} \tag{5.12}$$

$$\lambda^* = \frac{a}{\sum^*}\left(\frac{c_3 \Lambda_L \lambda^*_1}{A_1 d_L - (A_2 - A_1)\lambda^*_1} + \frac{c_4 c_2 K_N}{r_N (B_1 c_1)^2}(r_N B_1 c_1 \lambda^*_1 - (B_1 - B_2) c_2 (\lambda^*_1)^2)\right). \tag{5.13}$$

with $c = \dfrac{\nu_M}{(\nu_M + b_M)(b_M)}$. Using the equations (5.12) and (5.13), we have:

$$b_M \lambda^*_1 \sum^{*^2} - \frac{a(\Lambda_M a c_1 c - \lambda^*_1 \sum^*)}{\sum^*}\left(\frac{c_3 \Lambda_L \lambda^*_1}{A_1 d_L - (A_2 - A_1)\lambda^*_1} + \frac{c_2 c_4 K_N}{r_N (B_1 c_1)^2}\left(r_N B_1 c_1 \lambda^*_1 - (B_1 - B_2) c_2 (\lambda^*_1)^2\right)\right) = 0, \tag{5.14}$$

Since $\lambda^*_1 \neq 0$, by replacing $\sum^*$ by its formula, we get the following polynomial:

$$p_0 + p_1 \lambda^*_1 + p_2 (\lambda^*_1)^2 + p_3 (\lambda^*_1)^3 + p_4 (\lambda^*_1)^4 + p_5 (\lambda^*_1)^5 + p_6 (\lambda^*_1)^6 + p_7 (\lambda^*_1)^7 = 0. \tag{5.15}$$

The coefficients p_i, $i = 0, ...7$, of the polynomial (5.15) are given in the appendix 7.

Obviously, it is not easy to find the number of the exact solutions to the polynomial (5.15), and hence determine the exact number of EE. However, using the Descartes' Rule of Signs [3], we determine the possible EE depending on the sign of coefficients p_i, $i = 0, ..., 7$ of the polynomial (5.15).

By the Descartes' Rule of Signs, if $\mathcal{R}_0 < 1$, then either we have no solution or an even number of endemics equilibria (EE) and when $\mathcal{R}_0 > 1$ we have an odd number EE. We can be more specific by stating the following result.

Proposition 2. *Suppose that $p_i > 0$ for all $i = 1, \cdots, 6$ then:*

- *If $\mathcal{R}_0 < 1$ so $p_0 > 0$, there is no endemic equilibria.*
- *If $\mathcal{R}_0 > 1$ so $p_0 < 0$, there is a unique endemic equilibrium.*

This result gives us a classical scenario where for $\mathcal{R}_0 < 1$ the disease free equilibrium is globally stable and for $\mathcal{R}_0 > 1$ the disease free equilibrium is unstable. The proof of this proposition is straightforward from the paper of Levin [29].

More cases are treated in the appendix that show the possible existence of multiple endemic equilibria.

5.2. The uniform persistence

Since it is difficult to investigate the global stability of the endemic equilibria, as there are different scenarios of existence of endemic equilibria, in this section we focus on finding the conditions of the uniform persistence.

We recall that Γ, defined in (3.2), is a positively invariant subset of $\mathbb{R}^{11}_+$.

Before giving the main result, we define $\Phi_t(S, E, I, R)$ as the flow corresponding to system (3.3). In fact, $\Phi_t(S, E, I, R)$ denotes the solution of our system that starts at $S(0), E(0), I(0), R(0) \geq 0$. Moreover,

$$\Phi_t(S_0, E_0, I_0, R_0) = (S(t), E(t), I(t), R(t)),$$

where

$$S = (S_L, S_N, S_M), \;\; E = (E_L, E_N, I_{M,1}), \;\; I = (I_L, E_N, I_{M,2}), \;\; R = (R_L, R_N).$$

Using the result of the uniqueness of solution, we have the following result.

Theorem 5.1. *If $\mathcal{R}_0 > 1$ then our system is uniformly persistent.*

Proof. The system (2.1)-(2.3) is said to be uniformly persistent [40] if there exists a constant $r > 0$, independent of initial conditions, such that any solution $S(t), E(t), I(t), R(t)$ of our system satisfies the following inequalities:

$\liminf_{t\to\infty} S(t) \geq r$, $\liminf_{t\to\infty} E(t) \geq r$, $\liminf_{t\to\infty} I(t) \geq r$, $\liminf_{t\to\infty} R(t) \geq r$.

The uniform persistence of our system can be proven by applying the result in Theorem [4.3, [21]]. In fact, Φ is a continuous flow on Γ that is a closed positively invariant subset of $\mathbb{R}^{11}_+$.

Denote the restriction of Φ_t to $\partial\Gamma$ by $\partial\Phi_t$. The maximal invariant set of $\partial\Phi_t$ on $\partial\Gamma$ is the singleton $\mathcal{S} = \{E_{02}\}$ that is a closed invariant set and also is isolated.

Let $\{\mathcal{S}_\alpha\}_{\alpha\in A}$ denote the cover of $\mathcal{S}$ where A is a non empty index set , $\mathcal{S}_\alpha \subset \partial\Gamma$, $\mathcal{S} \subset \bigcup_{\alpha\in A} \mathcal{S}_\alpha$ and $\mathcal{S}_\alpha$ are pairwise disjoint closed invariant sets.

No subset of the $\{\mathcal{S}\alpha\}$ forms a cycle *i.e* there exist no $\alpha \in A$ such that $\mathcal{S}_\alpha = \mathcal{S}_{\alpha_0}$.

The corresponding sets are denoted by $\gamma(E_{02})$, $\gamma^-(E_{02})$, $\gamma^+(E_{02})$ and are, respectively, called the *trajectory, positive trajectory and negative trajectory*.

All hypothesis (**H**) of [21] holds for system (1). Therefore, if $\mathcal{R}_0 > 1$ then E_{02} is unstable, which gives the necessary and sufficient condition of Theorem 4.3 [21], and we conclude that our system is uniformly persistent. □

Whether there is one or more endemic equilibrium, the proven result shows that if $\mathcal{R}_0 > 1$, the disease-free equilibrium is unstable and the disease persists.

6. The effect of the carrying capacity K_N

As our main results depend on $\mathcal{R}_0$, which is a function of K_N, the carrying capacity of the non-local population, our aim is to investigate the positive effect of the carrying capacity K_N in reducing the spread of the disease infection in the total population.

Therefore, we study the sign of the function $1-\mathcal{R}_0(K_N)$. By rearranging this function and from (3.4), our problem reduces to studying the sign of the function $\mathbf{P}(K_N)$, where

$$\mathbf{P}(K_N) = (K_N)^2 + \left(2\frac{\Lambda_L}{d_L} - a^2c_2c_4\frac{\Lambda_M\nu_M}{b_M^2(\nu_M+b_M)}\frac{\nu_N}{\epsilon_N\theta_N}\right)K_N + \left(\frac{\Lambda_L}{d_L}\right)^2\left(1-\left(\mathcal{R}_0^L\right)^2\right). \tag{6.1}$$

The roots of $P(K_N)$ are given by

$$K_{1,2} = \frac{1}{2}\left[-\left(2\frac{\Lambda_L}{d_L} - a^2c_2c_4\frac{\Lambda_M\nu_M}{b_M^2(\nu_M+b_M)}\frac{\nu_N}{\epsilon_N\theta_N}\right) \mp \sqrt{\Delta}\right],$$

with

$$\Delta = \left(2\frac{\Lambda_L}{d_L} - a^2c_2c_4\frac{\Lambda_M\nu_M}{b_M^2(\nu_M+b_M)}\frac{\nu_N}{\epsilon_N\theta_N}\right)^2 + 4\left(\frac{\Lambda_L}{d_L}\right)^2\left(\left(\mathcal{R}_0^L\right)^2-1\right).$$

Let

$$\xi_{NM} = a^2c_2c_4\frac{\Lambda_M\nu_M}{b_M^2(\nu_M+b_M)}\frac{\nu_N}{\epsilon_N\theta_N}.$$

Hence, we have the following cases:

Case 1. If $\mathcal{R}_0^L > 1$ then $\Delta > 0$, the quadratic equation (6.1) has only one unique positive root K_2.

Case 2. If $\mathcal{R}_0^L < 1$ then it depends on the sign of $2\frac{\Lambda_L}{d_L} - \xi_{NM}$ and we can observe the following possibilities:

- If $2\frac{\Lambda_L}{d_L} < \xi_{NM}$ our quadratic polynomial (6.1) has two positives roots $K_1 < K_2$.
- If $2\frac{\Lambda_L}{d_L} > \xi_{NM}$ the quadratic polynomial (6.1) has no positive root.

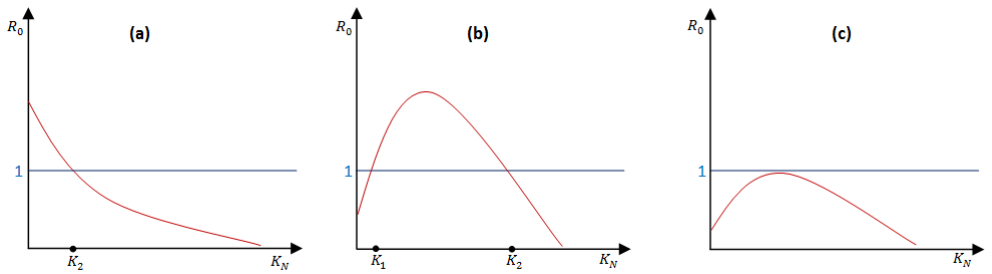

Figure 4. The plot of $\mathcal{R}_0$ as function of K_N i.e., $\mathcal{R}_0(K_N)$. the three graphs show all the possible cases of the level of $\mathcal{R}_0$ as K_N increases.

Figure 4 represents the plots of all the different possible cases.
Figure 4 (a) represents case 1, where the basic reproduction number of the disease in the local population is above 1. As the carrying capacity of the non local K_N increases, $\mathcal{R}_0$ decreases, leading to the eradication of the disease as K_N exceeds the critical carrying capacity value K_2. On the other hand, if the basic reproduction number of the local population is below 1, there will be two scenarios.

(i) The first scenario is represented by Figure 4 (b). In this case, we have two critical carrying capacity values K_1 and K_2. When K_N is below K_1 or above K_2, $\mathcal{R}_0$ is less than one, and the disease will die out. The second situation is when K_N belongs to the interval $]K_1, K_2[$, $\mathcal{R}_0$ is above 1, and the disease will persist in the total population.

(ii) Second scenario, Figure 4 (c), $\mathcal{R}_0$ remains always below one and the increase of the carrying capacity of the non-local population has no effect on the transmission of the disease among the total population.

7. Discussion and conclusion

As Malaria is still a global health threat, there are ongoing scientific efforts to eradicate the disease that burdens several regions in the world including Sub-Saharan countries. This work is aimed at studying the effect of the sub-Saharan immigrants on the possible importation of malaria to North African countries. The goal is to investigate the impact of immigrants (non-local population) on the possible reappearance of Malaria in the North African countries.

Therefore, we introduced a mathematical model that included two human populations (locals and non-locals) and the mosquito population. Unlike the existing models, our study considered two types of growth for each human population. The local population with a linear growth and the immigrant population with a logistic growth. The choice of having a logistic growth for the non-local population was justified by the fact that the hosting country might impose a carrying capacity on the number of the immigrants. Plus there are no mechanisms to screen the health status of the immigrants coming to the host country.

Using the basic reproduction number of the disease $\mathcal{R}_0$, which was calculated by the standard next-generation matrix method, we gave, in the remark 1, the characterization of all possible conditions, on $\mathcal{R}_0^L$ and $\mathcal{R}_0^N$, which led to $\mathcal{R}_0 < 1$ and $\mathcal{R}_0 > 1$. This result showed that the disease could persist, slightly, in both human populations (locals and non-locals) although $\mathcal{R}_0 < 1$. On the other hand, the transmission of the disease in both populations should reach a specific level, i.e., the minimum of the basic reproduction number of locals and non-locals have to exceed ϕ_1 or $\frac{\sqrt{2}}{\phi_2}$ (remark 1), before it could become highly infectious in all the population.

The global stability analysis showed that the threshold condition $\mathcal{R}_0 < 1$ alone could not guarantee the global stability of the disease-free equilibrium. In fact, the total population $\sum$ must have upper and lower bounds to have the global stability. However, via a Lyapunov function, we showed that if $\mathcal{R}_0$ is less than a constant, which is below one, then the disease died out from the total population (Theorem 1).

Using Descartes Rule of Signs, we were able to find the conditions of the existence of endemic equilibrium. Depending on the signs of the coefficients of the polynomial (5.15), propositions 3, 4 and 5 gave all the possible cases of the number of endemic equilibria. More precisely the number of endemic equilibriums are even (0 or 2) if $\mathcal{R}_0 < 1$ and odd (1 or 3) if $\mathcal{R}_0 > 1$. As we could not give a general result for the global stability of an endemic equilibrium, we proved, in Theorem 5.1, that if $\mathcal{R}_0 > 1$, then we had the uniform persistence of the solution.

Finally, we investigated the impact of the carrying capacity of the non-local population on the transmission of the disease in both populations. Our findings showed that if $\mathcal{R}_0^L > 1$, then as the carrying capacity increased, the disease changed from being persistent ($\mathcal{R}_0 > 1$) to a possible eradication; in this case, it had *an absorption effect* of the malaria infection in the total population. However, if $\mathcal{R}_0^L < 1$, there were two scenarios: If the local population growth was not high (i.e., $\frac{\Lambda_L}{d_L} < \frac{\xi_{NM}}{2}$), then the increase of carrying capacity of the non-locals would not affect the transmission of the disease until it reached a specific threshold K_1, after that the disease became persistent. The increase of the carrying capacity, to reach another threshold K_2, led to a decline of the disease infection among the total population, and again we observed the *absorption effect*. The second scenario was when the local population growth was high enough (i.e. $\frac{\Lambda_L}{d_L} > \frac{\xi_{NM}}{2}$). In this case, an increase of the carrying capacity of non-local would not affect the malaria transmission in the population.

Our finding suggests that the imported malaria infections in the North African countries cannot be blamed on the increasing number of the immigrant from the Sub-Saharan countries. If the disease is already endemic in the local population, then the increase of the carrying capacity of the immigrant has an absorption effect on the infection. However, if the disease is not endemic among the local population, the transmission of the imported malaria depends on the level of the growth of the local population.

Acknowledgements

The authors would like to thank the anonymous reviewers for their valuable comments and suggestions which helped us improve the quality of our work.

Conflict of interest

All authors declare no conflicts of interest in this paper.

References

1. F.B. Agusto and J. M. Tchuenche, Control strategies for the spread of malaria in humans with variable attractiveness, *Math. Popul. Stud.* **20** (2013), 82–100.
2. R. M. Anderson and R. M. May, *Infectious diseases of humans: dynamics and control*, Oxford University Press, London, 1991.

3. B. Anderson, J. Jackson and M. Sitharam, The American Mathematical Monthly, Descartes Rule of Signs Revisited, **105** (1998), 447–151.
4. R. Anguelov, Y. Dumont, J. Lubuma and E. Mureithi, Stability analysis and dynamics preserving nonstandard finite difference schemes for a malaria model, *Math. Popul. Stud.*, (2013), 101–122.
5. J. Arino, Diseases in metapopulations, In: Z. Ma, Y. Zhou, and J. Wu (Eds.), *Modeling and dynamics of infectious diseases*, Higher Education Press, Beijing, (2009), 64–122.
6. J.L. Aron and R.M. May, The population dynamics of malaria, The Population Dynamics of Infectious Diseases: Theory and Applications. Population and Community Biology, Springer US, (1982),139–179.
7. S. Belhadj, O. Menif, E. Kaouech, S. Anane, H. Jeguirim, T. Ben Chaabane, K. Kallel and E. Chaker, Le paludisme d'importation en Tunisie: bilan de 291 cas diagnostiqués à l'hôpital La Rabta de Tunis (1991-2006), *Revue Francophone des Laboratoires*, **399** (2008), 95–98.
8. J. Ben Yahia, Algeria's migration policy conundrum, *Institute For Security Studies, ISS Today*, Available from: https://issafrica.org/iss-today/algerias-migration-policy-conundrum, 2018.
9. H. Benzerroug, Paludisme importé de Tanzanie en Algérie. á propos d'un cas résistant a la chloroquine, *Ann. Soc. Belg. Med.*, **65** (1985), 9–85.
10. A. Berman and R. J. Plemmons, *Non-negative Matrices in the Mathematical Sciences*, Academic Press, New York, 1999.
11. S. C. Boubidi, I. Gassen, Y. Khechache, K. Lamali, B. Tchicha, C. Brengues, M. Menegon, C. Severini, D. Fontenille and Z. Harrat , Plasmodium falciparum Malaria, Southern Algeria, 2007, *Emerg. Infect. Dis.*, **16** (2010), 301–303.
12. N. Bouzouaia, Guide National de prise en charge du Paludisme in Tunisei, *Organisation Mondial de la Sante, Bureau regional de la Mediterranee Orientale*, Ministere de la Sante Publique, 2016.
13. F. Brauer and C. Castillo-Chavez, *Mathematical Models in Population Biology and Epidemiology*, Springer, 2012.
14. N. Chitnis, J. M. Cushing and J. M. Hyman, Bifurcation analysis of a mathematical model for malaria transmission, *SIAM J. Appl. Math.*, **67** (2006), 24–45.
15. N. Chitnis, J. M. Hyman and J.M. Cushing, Important parameters in the spread of malaria through the sensitivity analysis of a mathematical model, *B. Math. Biol.*, **70** (2008), 1272–1296.
16. N. Chitnis and J. M. Hyman and C.A. Manore, Modelling vertical transmission in vector-borne diseases with applications to Rift Valley fever, *J. Biol. Dynam.*, **7** (2013),11–40.
17. C. Chiyakaa, W. Gariraa and S. Dubeb, Transmission model of endemic human malaria in a partially immune population, *Math. Comput. Model.*, **46** (2007), 806–822.
18. O. Diekmann and J. A. P. Heesterbeek, Mathematical epidemiology of infectious diseases: Model building, analysis and interpretation, WILEY, USA, 2000.
19. K. Dietz, L. Molineaux and A. Thomas, A malaria model tested in the African savannah, *B. World Health Organ.*, **50** (1974), 347–357.
20. J.A.N. Filipe, E. M. Riley, C. J. Drakeley, C.J. Sutherl and A.C. Ghani, Determination of the processes driving the acquisition of immunity to malaria using a mathematical transmission model, *PLoS Comput. Biol.*, **3** (2007), 806–822.

21. H.I Freedman, S. Ruan and M.Tang, Uniform Persistence and Flows near a Closed Positively Invariant Set, *J. Dyn. Differ. Equ.*, **6** (1994), 583–600.
22. D. Gao and S. Ruan, A multipatch malaria model with logistic growth populations, *SIAM J. Appl. Math.*, **72** (2012), 819–841.
23. D. Hammadi, S.C. Boubidi, S.E. Chaib, A. Saber, Y. Khechache, M. Gasmi and Z. Harrat, Malaria in Algerian Sahara, *B. Soc. Pathol. Exot.*, **102** (2009), 185–192.
24. Kabrane, Principales caractéristiques épidémiologiques du paludisme d'importation en Algérie (1988–1993), Relevé épidémiologique mensuel (INSP Alger), **9** (1994), 73–80.
25. H. K. Khalil, *Nonlinear Systems* , 3^rd Edition, Prentice Hall, USA, 2002.
26. S. Kim and A. Tridane and D. E. Chang, Human migrations and mosquito-borne diseases in Africa, *Math. Popul. Stud.*, **23** (2016), 123–146.
27. R. A. Korba, S. Boukraa, M. S. Alayat, M. L. Bendjeddou, F. Francis, S.C. Boubidi and Z. Bouslama, Preliminary report of mosquitoes survey at Tonga Lake (North-East Algeria), *Adv. Environ. Biol.*, **9**, (2016), 288–294.
28. A.A. Lashari, S. Aly, K. Hattaf, G. Zaman, I.H. Jung and X. Z. Li, Presentation of Malaria Epidemics Using Multiple Optimal Controls, *J. Appl. Math.*, **2012** (2012).
29. S. A. Levin, Descartes' Rule of Signs - How hard can it be? (2002).
30. S. Mandal, R. R. Sarkar and S. Sinha, Mathematical models of malaria - a review, *Malaria J.*, **10** (2011), 202.
31. G. Macdonald, *The epidemiology and control of malaria*, Oxford University Press, London, 1957.
32. E.M. Kakmeni Moukam, R. Y. A. Guimapi, F. T. Ndjomatchoua, S. A. Pedro, J. Mutunga and H.E. Z. Tonnang, Spatial panorama of malaria prevalence in Africa under climate change and interventions scenarios, *Int. J. Health Geogr.*, **17** (2018), 2.
33. G. A. Ngwa and W. S. Shu, A mathematical model for endemic malaria with variable human and mosquito populations, *Math. Comput. Model.*, **32** (2000), 747–763.
34. S. Odolini, P. Gautret and P. Parola, Epidemiology of imported malaria in the Mediterranean region, *Mediterr. J. Hematol. Infect. Dis.*, **4** (2012), e2012031.
35. K. Okuneye and A. B. Gumel, Analysis of a temperature and rainfall dependent model for malaria transmission dynamics, *Math. Biosci.*, **287** (2017), 72–92.
36. M. G. Roberts and J. A. P. Heesterbeek, A new method for estimating the effort required to control an infectious disease, *Roy. Soc.*, **270** (2003), 1359–1364.
37. M. Roser and H. Ritchie, Malaria, *Published online at OurWorldInData.org*, Available from: https://ourworldindata.org/malaria, (2018).
38. R. Ross, The prevention of malaria, *John Murray*, (1911).
39. M. Samsuzzoha, M. Singh and D. Lucy, Uncertainty and sensitivity analysis of the basic reproduction number of a vaccinated epidemic model of influenza, *Appl. Math. Model.*, **37** (2013), 903–915.
40. H. L. Smith and H. R.Thieme, *Dynamical Systems and Population Persistence*, American Mathematical Society Providence, Rhode Island, 2011.

41. R. J. Smith and S. D. Hove-Musekwa, Determining Effective Spraying Periods to Control Malaria via Indoor Residual Spraying in Sub-Saharan Africa, *Hindawi Publishing Corporation Journal of Applied Mathematics and Decision Sciences*, 2008.
42. J. Smoller, *Shock Waves and Reaction-Diffusion Equations*, 2^{nd} edition, Springer-Verlag, New York, 1994.
43. N. Sogoba, P. Vounatsou, M. M. Bagayoko, S. Doumbia, G. Dolo, L. Gosoniu, S. F. Traoré, T. A. Smith and Y. T. Touré, Spatial distribution of the chromosomal forms of anopheles gambiae in Mali, *Malaria J.*, **7** (2008), 205.
44. B.Trari and P. Carnevale, Malaria in Morocco: from pre-elimimation to elimination, what risks for the future? *Société de pathologie exotique*, **104**, (2011).
45. H. R.Thieme, *Mathematics in population biology*, Princeton Series In Theoretical and Computational Biology, 2003.
46. P. Van den Driessche and J. Watmough, Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission, *Math. Bio.*, **180** (2002), 29–48.
47. M. Vidyasagar, Decomposition techniques for large-scale systems with non-additive interactions: stability and stabilizability, IEEE Trans, *Automat*, **25** (1980), 773–779.
48. W. Wang, Y. Takeuchi, Y. Iwasa and K. Sato, Epidemic models with population dispersal, Mathematics for Life Sciences and Medicine,Springer Berlin, (2007), 67–95.
49. O. Watson, H. C. Slater, R. Verity, J. B. Parr, M. K. Mwandagalirwa, A. Tshefu, S. R. Meshnick, A. C Ghani Modelling the drivers of the spread of *Plasmodium falciparum hrp2* gene deletions in sub-Saharan Africa, *eLife*. eLife Sciences Publications, Ltd, **6** (2017), e25008.
50. WHO, *Factsheet on the World Malaria Report 2012*, Available from: `https://www.who.int/malaria/media/world_malaria_report_2012_facts/en/`.
51. WHO, *Eliminating Malaria*, WHO, Geneva, Available from: `http://apps.who.int/iris/bitstream/10665/205565/1/WHO_HTM_GMP_2016.3_eng.pdf`, 2016.
52. *World Health Organization*, Available from: `http://www.who.int/malaria/en/`.
53. Y. Xiao and X. Zou, Can Multiple Malaria Species Co-Peprsist? *SIAM J. Appl. Math.*, **73** (2013), 351–373.

Appendix

The endemic equilibrium

If we denote by, $\sum_2^* = \dfrac{\Lambda_L}{d_L} + K_N$ and recall that, $c = \dfrac{\nu_M}{b_M(\nu_M + b_M)}$ then the coefficients p_i of the polynomial (5.15) are given as follow:

$$p_0 = b_M A_1^2 d_L^2 (\sum_2^*)^2 (1 - (\mathcal{R}_0)^2),$$

$$p_1 = 2b_M\alpha_0\alpha_1 + a^2\Lambda_M cc_2c_4\frac{K_N}{B_1}A_1A_2 + a^2\Lambda_M cc_2c_4\frac{K_N}{r_NB_1}A_1^2d_L\left((B_1 - B_2)\frac{c_2}{B_1c_1}d_L - r_N\right)$$

$$+A_1 d_L \frac{b_M}{ac_1 c\Lambda_M}(\mathcal{R}_0)^2(\sum_2^*)^2((\alpha_0 d_L - a\Lambda_M c_1 c)A_1 + a\Lambda_M c_1 c A_2),$$

$$\begin{aligned}
p_2 &= b_M\alpha_1^2 + \alpha_0 b_M\left(2\alpha_2 - A_1(A_2-A_1)d_L\frac{1}{ac_1c\Lambda_M}(\mathcal{R}_0)^2(\sum_2^*)^2\right)\\
&\quad + ac_4\frac{c_2K_N}{r_NB_1c_1}\left(r_NA_2 + A_1((B_1-B_2)\frac{c_2}{B_1c_1}d_L - r_N)\right)((a\Lambda_Mc_1c - \alpha_0d_L)A_1 - a\Lambda_Mc_1cA_2),\\
p_3 &= b_M\alpha_0\left[2\alpha_3 + \alpha_2A_1d_L\frac{\sum_2^*}{ac_1c\Lambda_M}(\mathcal{R}_0)^2\right] + \alpha_1b_M\left[2\alpha_2 - (A_2-A_1)A_1A_2d_L\frac{(\sum_2^*)^2}{ac_1c\Lambda_M}(\mathcal{R}_0)^2\right]\\
&\quad + ac_2c_4\frac{K_N}{r_NB_1c_1}\left(r_NA_2 + A_1((B_1-B_2)\frac{c_2}{B_1c_1}d_L - r_N)\right)(\alpha_0A_2 - a\alpha_1A_1d_L)\\
&\quad + ac_2^2c_4(A_2-A_1)\frac{K_N}{r_N(B_1c_1)^2}(B_1-B_2)\,(a\Lambda_Mc_1c(A_2-A_1) + \alpha_0A_1d_L),\\
p_4 &= -a\alpha_0c_2^2c_4\frac{K_N}{r_N(B_1c_1)^2}(B_1-B_2)(A_2-A_1)^2 + a\alpha_1c_2^2c_4A_1d_L\frac{K_N}{r_N(B_1c_1)^2}(A_2-A_1)(B_1-B_2)\\
&\quad + ac_2c_4\frac{K_N}{r_NB_1c_1}\left(r_NA_2 + A_1((B_1-B_2)\frac{c_2}{B_1c_1}d_L - r_N)\right)(\alpha_1A_2 - (\alpha_1+\alpha_2d_L)A_1)\\
&\quad + \alpha_3b_M\left[2\alpha_1 + \frac{(A_1d_L)^2(\sum_2^*)^2}{acc_1\Lambda_M}(\mathcal{R}_0)^2\right] + \alpha_2b_M\left[\alpha_2 - (A_2-A_1)\frac{A_1d_L(\sum_2^*)^2}{acc_1\Lambda_M}(\mathcal{R}_0)^2\right],\\
p_5 &= ac_2c_4\frac{K_N}{r_NB_1c_1}\left(r_NA_2A_1(d_L(B_1-B_2)\frac{c_2}{c_1B_1} - r_N)\right)(\alpha_2(A_2-A_1) - \alpha_3A_1d_L)\\
&\quad + \alpha_3b_M\left[2\alpha_2 - (A_2-A_1)\frac{A_1d_L(\sum_2^*)^2}{acc_1\Lambda_M}(\mathcal{R}_0)^2\right]\\
&\quad - ac_2^2c_4\frac{K_N}{r_N(B_1c_1)^2}(A_2-A_1)(B_2-B_1)\,(\alpha_1A_2 - A_1(\alpha_1+\alpha_2d_L)),\\
p_6 &= b_M\alpha_3^2 - a\alpha_2c_2^2c_4\frac{K_N}{r_N(B_1c_1)^2}(B_1-B_2)(A_2-A_1)^2 + a\alpha_2c_2^2c_4\frac{K_N}{r_N(B_1c_1)^2}A_1d_L(A_2-A_1)(B_1-B_2)\\
&\quad + ac_2c_4\alpha_2(A_2-A_1)\frac{K_N}{r_NB_1c_1}\left(r_NA_2 + A_1((B_1-B_2)\frac{c_2}{B_1c_1}d_L - r_N)\right),\\
p_7 &= -c_2^4c_4\frac{K_N^2}{r_N^2(B_1c_1)^4}\left(\frac{B_1}{\epsilon_N} + \frac{B_2}{\delta_N} + 1\right)(A_2-A_1)^3(B_1-B_2).
\end{aligned}$$

Since $A_2 - A_1 \le 0$ and $B_2 - B_1 \le 0$, we have $p_7 > 0$. On the other hand, if $\mathcal{R}_0 < 1$ then $p_0 > 0$, and respectively if $\mathcal{R}_0 > 1$ then $p_0 < 0$.

Multiple endemic equilibria

The proposition 2 deals with one case of existence of endemic equilibria among several others. Depending on the sign of p_i, there are more cases of the number of possible endemic equilibria.

If $\frac{c_2}{c_1} \geq \frac{\epsilon_N^2 \beta_N \theta_N}{d_L(\epsilon_N \beta_N \theta_N - \delta_N \gamma_N \nu_N)}$ and $r_N \in \left[\epsilon_N, \frac{d_L}{c_1}\frac{c_2}{\beta_N \epsilon_N \theta_N}(\beta_N \epsilon_N \theta_N - \delta_N \gamma_N \nu_N)\right]$ then we can have the following results:

1. $p_6 > 0$.
2. If $(\mathcal{R}_0)^2 \leq 2\alpha_2 \frac{\beta_L \nu_L}{\delta_L \gamma_L \nu_L - \epsilon_L \theta_L \beta_L} \frac{d_L^2}{\Lambda_L + d_L K_N}$ then,
   - when $\frac{\epsilon_L \theta_L}{\nu_L} \geq \frac{a\Lambda_M c_1 c}{(\Lambda_L + d_L K_N) d_L}$ we obtain that $p_1 > 0$ and $p_3 < 0$.
   - when $\alpha_1 \frac{\delta_L \gamma_L \nu_L - \epsilon_L \theta_L \beta_L}{\beta_L \nu_L} - \frac{\epsilon_L \theta_L d_L}{\nu_L} d_L \alpha_2 \leq 0$ we have $p_5 > 0$.
3. If $(\mathcal{R}_0)^2 \geq 2\alpha_2 \frac{\beta_L \nu_L}{\delta_L \gamma_L \nu_L - \epsilon_L \theta_L \beta_L} \frac{d_L^2}{\Lambda_L + d_L K_N}$ then,
   - when $\frac{\epsilon_L \theta_L d_L}{\nu_L}(\Lambda_L + d_L K_N) \leq a\Lambda_M c_1 c(1 - \frac{\nu_L \delta_L \gamma_L}{\epsilon_L \theta_L \beta_L})$ we have $p_2 > 0$.
   - when $\alpha_1 \frac{\delta_L \gamma_L \nu_L - \epsilon_L \theta_L \beta_L}{\beta_L \nu_L} - \frac{\epsilon_L \theta_L d_L}{\nu_L} d_L \alpha_2 \leq 0$ we get $p_4 < 0$

Proposition 3. *If $p_1, p_5, p_6 \geq 0$ and $p_3 \leq 0$ then;*

- *an even number of positive real roots of $P(x)$ (0 or 2) when $\mathcal{R}_0 < 1$,*
- *an odd number of positive real roots of $P(x)$ (1 or 3) when $\mathcal{R}_0 > 1$.*

Proposition 4. *If $p_2, p_6 \geq 0$ and $p_4 \leq 0$ lead to the following result.*

- *When $\mathcal{R}_0 < 1$ we have an even number of possible positive real roots of $P(x)$ (0 or 2).*
- *When $\mathcal{R}_0 > 1$ we have an odd number of possible positive real roots of $P(x)$ (1 or 3).*

Proposition 5. *If $p_6 \geq 0$ we can have up to 6 endemic equilibria when $\mathcal{R}_0 < 1$ and no more then 5 when $\mathcal{R}_0 > 1$.*

AIMS Press