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Vector Epidemic Model of Malaria with Nonconstant-Size Population

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Abstract The paper presents the dynamic characteristics of a vector-host epidemic model with direct transmission. The malaria propagation model is defined by a system of ordinary differential equations. The host population is divided into four subpopulations: susceptible, exposed, infected and recovered, and the vector population is divided into three subpopulations: susceptible, exposed and infected. Using the theory of the Lyapunov functions, certain sufficient conditions for the global stability of the disease-free equilibrium and endemic equilibrium are obtained. The basic reproduction number that characterizes the evolution of the epidemic in the population was found. Finally, numerical simulations are carried out to study the influence of the key parameters on the spread of vector-borne disease.

Keywords: malaria, mathematical modeling of epidemics, mosquito population, subpopulations, reproductive number, endemic equilibrium.

1. Introduction

Malaria is an ancient disease that has a huge social, economic and health burden. Tropical regions such as Africa, Asia and America are conducive to the rapid spread of this disease (Cai et al., 2017). Many attempts have been made to describe the complex dynamics of the host and insect population dynamics with the presence of malaria infection using mathematical models. The classical population-based models developed by Ross and Macdonald (Ross, 1916; Macdonald, 1957) are still the basis for many new approaches (Maliki et al., 2018; Baygents and Bani-Yaghoub, 2017; Mandal et al., 2011). These models are based on the SIR (Susceptible/Infected/Recovered) methodology and sometimes aim at large-scale epidemiological predictions, and in most scientific articles describing the dynamics of malaria.

The malaria life cycle describes the different phases of the development and reproduction of malaria, an infectious disease carried by mosquitoes and caused by a variety of protists known as Plasmodium. Five different varieties of Plasmodium are capable of infecting humans; Plasmodium falciparum tends to cause the most severe cases of the infection. Malaria infections in individuals are governed by several factors such as temperature, climate, environment, etc. The description of the malaria cycle omit some details. It is worth to say that malaria infection in the human population begins when sporozoites are delivered into the bloodstream by an infected female mosquito. Sporozoites migrate to the liver, and after some period (sometimes weeks, and sometimes months) they enter the bloodstream in the form of gametocytes, which the mosquito first receives when exposed by an infected person. During the development cycle in the mosquito, the injected gametocytes become gametes that first turn into zygotes, then into motile ookinetes that pierce

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the mosquito's intestine and release a large number of sporozoites. This cycle can be schematized in the form (Martens et al., 1995) given in Figure 1.

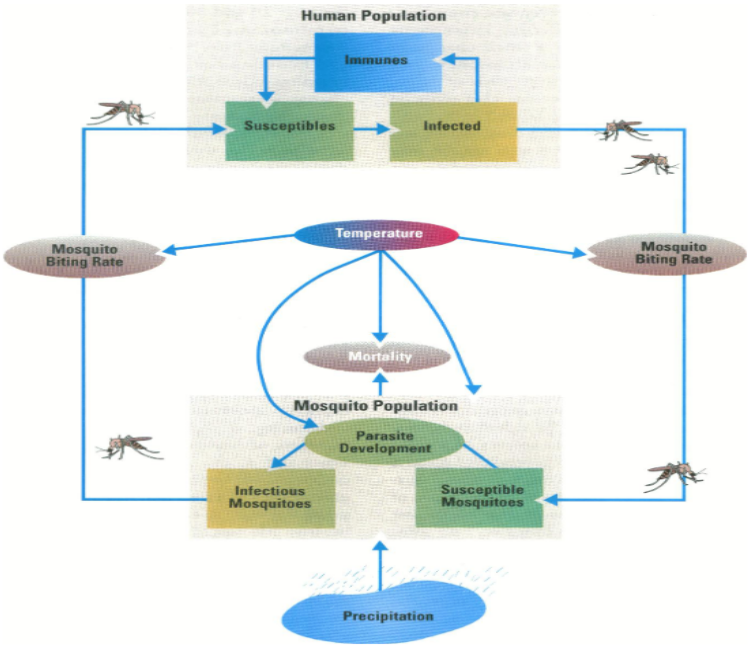

Fig. 1. Diagram of the main population and rate processes involved in the life cycle of the malaria parasite (the picture is borrowed from (Martens et al., 1995))

In recent years, a lot of scientific research has been undertaken and progress has been made in understanding host-parasite-vector interactions and their biology. However, the complexity of the parasite's life cycle, the very complex environmental and social interactions, the evolutionary pressure of drugs and control measures contributing to the parasite's resistance to drugs, the unforeseen effects of climate change and the migration of the population between endemic and non-endemic areas continued. Contribute to the enormous burden of morbidity and mortality that accompanies the disease. These have also presented new challenges to researchers and public health professionals.

In this article, a deterministic model of vector-borne disease is proposed. This model develops the transmission of the disease from mosquitoes to humans and from humans to mosquitoes as described on the malaria cycle. Mathematically, the transmission is described by differential equations (Ndiaye and Parilina, 2022; Gurarie et al., 2012; Zhang et al., 2020). We generalize the malaria model by including in the community considered the exposed, infected, cured people and a community of exposed, infected mosquitoes, taking into account the induced mortality caused by the disease in the population. We first study the stability of a system of differential equations describing a model, the analysis of which shows that there are equilibria characterizing the state of the system without disease and stable states in the presence of an epidemic. The number of the basic reproduction number R_0 is calculated. The value R_0 is the number of secondary infections that an infectious individual would create during the period of the disease, provided that

the total population, except infectious, is susceptible. Numerical simulations studying the impact of the key parameters on the spread of the disease are made in the work.

Diseases, such as yellow fever, dengue fever and malaria, transmitted by mosquitoes, are frequently observed in tropical and subtropical countries. Among these vector-borne diseases, malaria is one of the serious major diseases caused by several species of parasites transmitted by mosquitoes (Plasmodium falciparum, Plasmodium malaria, Plasmodium ovale, Plasmodium vivax, etc.) (Ghosha et al., 2013). The female anopheles mosquito is responsible for transmitting the disease to the human body through a bite (Wei et al., 2012), and after this bite it divides the individuals of the community into categories according to the parasitic density within them and the type of infection. These categories or compartments are represented using a modified model $SEIRS_kE_kI_k$ based on the pioneering work of Kermack and McKendrik (Kermack and McKendrick, 1927).

The rest of the paper is organized as follows. We describe the formulation of the model and specify the feasible region of the problem in Section 2. In Section 3, we determine the equilibrium points of the system of differential equations and the base reproductive number R_0, and then we study the stability of the system at the equilibrium points. We also provide the results of the numerical simulations in Section 4 and briefly conclude in Section 5.

2. Model $SEIRS_kE_kI_k$

We describe a modified model $SEIRS_kE_kI_k$ based on the work (Kermack and McKendrick, 192). The mosquitoes that transmit the disease pass in three phases: the susceptible (S_k), the exposed (S_k) and the infected (I_k). The bite of a female anopheles mosquito carrying the malaria virus converts a healthy (susceptible) human being (S) into a category called infected hosts (I). The human population that is not infected, but is at risk of being infected with malaria, is known as the exposed population. People recovered from the infected population through medical treatment without a threat to their lives fall into the group of recovered people (R). Figure 2 shows the interaction diagram between the human population (host) and the mosquito population (vector) for the transmission of malaria.

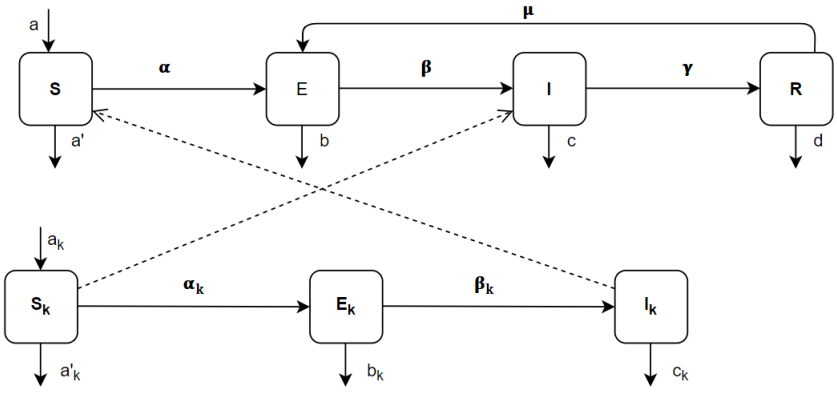

Fig. 2. The malaria model

The total host population can be represented as $N(t) = S(t)+E(t)+I(t)+R(t)$ and the total vector population is represented as $N_k(t) = S_k(t) + E_k(t) + I_k(t)$.

The mathematical model of the population dynamics (human and mosquito) can be represented analytically by the following nonlinear system of seven ordinary differential equations:

$$\begin{cases} \dfrac{dS(t)}{dt} = -\alpha S(t)I_k(t) + aN(t) - a'S(t), \\ \dfrac{dE(t)}{dt} = \alpha S(t)I_k(t) + \mu R(t) - bE(t) - \beta E(t), \\ \dfrac{dI(t)}{dt} = \beta E(t) - cI(t) - \gamma I(t), \\ \dfrac{dR(t)}{dt} = \gamma I(t) - dR(t) - \mu R(t), \\ \dfrac{dS_k(t)}{dt} = -\alpha_k S_k(t)I(t) + a_k N_k(t) - a'_k S_k(t), \\ \dfrac{dE_k(t)}{dt} = \alpha_k S_k(t)I(t) - b_k E_k(t) - \beta_k E_k(t), \\ \dfrac{dI_k(t)}{dt} = \beta_k E_k(t) - c_k I_k(t), \end{cases} \tag{1}$$

with the initial conditions

$$S(0) \geq 0,\ E(0) \geq 0,\ I(0) \geq 0,\ R(0) \geq 0,\ S_k(0) \geq 0,\ E_k(0) \geq 0,\ I_k(0) \geq 0. \tag{2}$$

The total human population dynamics are given by

$$\frac{dN}{dt} = aN_0 - a'S - bE - cI - dR.$$

The given initial conditions (2) we should satisfy the condition: $N(0) \geq 0$. Thus, the total population size $N(t)$ remains positive and bounded for all finite time $t > 0$. The dynamics of the total vector population are

$$\frac{dN_k}{dt} = aN_{0_k} - a'S_k - bE_k - cI_k.$$

The following parameters are used in the model:

$N(t)$ — the human population size;
$S(t)$ — the number of subpopulations of susceptible humans;
$E(t)$ — the number of the subpopulation of the humans exposed by the vector;
$I(t)$ — the number of subpopulations of infected humans;
$R(t)$ — the number of the subpopulation of recovered humans;
a — fertility rate in the human population;
a' — mortality rate among subpopulation S;
b — mortality rate among subpopulation E;
c — mortality rate among infected subpopulation I;
d — mortality rate among the recovered subpopulation R;
β — the intensity of the transition of people from the subpopulation E to I, i.e. with the onset of the disease symptoms;
γ — the intensity of healing of humans, i.e. the transition from the subpopulation I into R;

μ — the rate of return of humans from recovered to susceptible;
α — the probability of transmitting a bite from an infectious mosquito to a susceptible person.
$N_k(t)$ — total mosquito population;
$S_k(t)$ — the number of mosquitoes that can be infected;
$E_k(t)$ — the number of mosquitoes susceptible to the disease;
$I_k(t)$ — number of infected mosquitoes;
$a_k(t)$ — fertility rate;
$a'_k(t)$ — mortality in the population of susceptible mosquitoes;
$b_k(t)$ — mortality of the exposed mosquito population;
$c_k(t)$ — mortality of the population of infected mosquitoes;
$\beta_k(t)$ — the coefficient of mosquitoes in which the symptoms of the disease begin.

2.1. The feasible region

A mathematical model presented by the system of differential equations (1) describes the changes of the human and mosquito populations. Therefore, it is important to make sure that all solutions with non-negative initial conditions (2) will remain non-negative forever. All solutions of the proposed system, which is initiated in Ω region, remain in Ω region. Strictly speaking, this result can be generalized in the following theorem.

Theorem 1. *Let $(S, E, I, R, S_k, E_k, I_k)$ be any solution of system (1) with positive initial conditions (2). For any time $t \geq 0$ there is a domain:*

$$\Omega = \Big\{(S, E, I, R, S_k, E_k, I_k) \in \mathbb{R}^7_+, V_1 \leq \frac{aN_0}{a' + b + c + d}, V_2 \leq \frac{a_k N_{0_k}}{a'_k + b_k + c_k}\Big\}.$$

Then Ω is positively invariant and attracting under the flow described by (1).

Proof. To prove this theorem, we use Lyapunov functions. Consider the following Lyapunov function $V(t) = (V_1(t), V_2(t))$. Suppose that the functions $V_1(t)$, $V_2(t)$ are defined for $\forall\ t \geq 0$, and they are differentiable and continuously differentiable on the set Ω containing the origin.

The time derivative of function $V(t)$ is

$$\frac{dV(t)}{dt} = \begin{cases} \dfrac{dV_1(t)}{dt} = aN_0 - (a' + b + c + d)V_1 - a'S - bE - cI - dR, \\ \dfrac{dV_2(t)}{dt} = a_k N_{0_k} - (a'_k + b_k + c_k)V_2 - a'_k S_k - b_k E_k - c_k I_k. \end{cases} \tag{3}$$

For the system (3), it is obvious that

$$\begin{aligned} \frac{dV_1(t)}{dt} &\leq aN_0 - (a' + b + c + d)V_1, \\ \frac{dV_2(t)}{dt} &\leq a_k N_{0_k} - (a'_k + b_k + c_k)V_2. \end{aligned} \tag{4}$$

By the properties of the Lyapunov function, we obtain the following conditions:

$$\begin{cases} \dfrac{dV_1}{dt} \leq aN_0 - (a' + b + c + d)V_1 \leq 0 \ \text{ for } \ V_1 \geq \dfrac{aN_0}{a' + b + c + d}, \\ \dfrac{dV_2}{dt} \leq a_k N_{0_k} - (a'_k + b_k + c_k)V_2 \leq 0 \ \text{ for } \ V_2 \geq \dfrac{a_k N_{0_k}}{a'_k + b_k + c_k}. \end{cases} \tag{5}$$

From conditions (5), it follows that $\dfrac{dV(t)}{dt} \leq 0$, which means that Ω is a positively invariant and an absorbing set.

From the above equations and conditions (3)–(5), we output the inequalities for V_1 and V_2:

$$0 \leq V_1(t) \leq \frac{aN_0}{a'+b+c+d} + \mathrm{e}^{-(a'+b+c+d)t}\left(V_{0_1} - \frac{aN_0}{a'+b+c+d}\right),$$
$$0 \leq V_2(t) \leq \frac{a_kN_{0_k}}{a'_k+b_k+c_k} + \mathrm{e}^{-(a'_k+b_k+c_k)t}\left(V_{0_2} - \frac{a_kN_{0_k}}{a'_k+b_k+c_k}\right).$$

With $t \longrightarrow +\infty$, we obtain that

$$0 \leq V_1(t) \leq \frac{aN_0}{a'+b+c+d},$$
$$0 \leq V_2(t) \leq \frac{a_kN_{0_k}}{a'_k+b_k+c_k},$$

and we can conclude that Ω is an absorbing set. Indeed, for $t \longrightarrow +\infty$ we obtain the inequalities

$$\limsup_{t\to+\infty} V_1 \leq \frac{aN_0}{a'+b+c+d},$$
$$\limsup_{t\to+\infty} V_2 \leq \frac{a_kN_{0_k}}{a'_k+b_k+c_k}.$$

Thus, Ω is positively invariant, and all solutions are bounded by the interval $[0,\infty)$.

3. The Equilibria

For the model, we examine two equilibrium points for the system of differential equations (1): (i) disease-free equilibrium E_s, and (ii) endemic equilibrium E_e. Solving the following system of differential equations

$$\begin{cases} -\alpha S(t)I_k(t) + aN(t) - a'S(t) & = 0 \\ \alpha S(t)I_k(t) + \mu R(t) - bE(t) - \beta E(t) & = 0, \\ \beta E(t) - cI(t) - \gamma I(t) & = 0, \\ \gamma I(t) - dR(t) - \mu R(t) & = 0, \\ -\alpha_k S_k(t)I(t) + a_kN_k(t) - a'_kS_k(t) & = 0, \\ \alpha_k S_k(t)I(t) - b_kE_k(t) - \beta_kE_k(t) & = 0, \\ \beta_kE_k(t) - c_kI_k(t) & = 0, \end{cases} \tag{6}$$

we can find these two equilibrium points:

1. Disease-free equilibrium $E_s = (\frac{a}{a'}N_0, 0, 0, 0, \frac{a_k}{a'_k}N_{0_k}, 0, 0)$, i.e. it is a constant solution of a system, in which there is no disease;
2. Endemic equilibrium of the system $E_e = (S^*, E^*, I^*, R^*, S_k^*, E_k^*, I_k^*)$, which assumes the presence of disease.

To find the equilibria, from the first equation of the system (6) we first get $S = \dfrac{(aN_0)E}{\alpha I_k + a'}$, from the third equation we obtain $E = \dfrac{c+\gamma}{\beta}I$ or $I = \dfrac{\beta}{c+\gamma}E$, then

from the fourth equation: $R = \dfrac{\gamma}{d+\mu}I$, from the fifth equation: $S_k = \dfrac{a_k N_{0_k}}{\alpha_k I + a'_k}$, from the sixth equation: $E_k = \dfrac{\alpha_k S_k}{b_k+\beta_k}I$, from the seventh equation: $I_k = \dfrac{\beta_k E_k}{c_k}$, from the second equation: $E = \dfrac{\alpha}{b+\beta}SI_k + \dfrac{\mu}{b+\beta}R$.

Substituting the first, third, fourth, fifth, sixth, and seventh equations of the system (6) into the second equation of this system, we obtain

$$E = \frac{aa_k\alpha\alpha_k\beta_k N_0 N_{0_k}(d+\mu)(c+\gamma)I}{((b+\beta)(d+\mu)(c+\gamma)-\mu\gamma\beta)(\alpha\beta_k\alpha_k a_k N_k I + a'c_k(b_k+\beta_k)(\alpha_k I + a'_k))}.$$

Making equality with the third equation obtained in the system (6), we get:

$$I = \frac{aa_k\alpha\beta\alpha_k\beta_k N_0 N_{0_k}(d+\mu) - a'a'_k c_k(b_k+\beta_k)((b+\beta)(d+\mu)(c+\gamma)-\mu\gamma\beta)}{((b+\beta)(d+\mu)(c+\gamma)-\mu\gamma\beta)(\alpha\beta_k\alpha_k a_k N_k + a'c_k\alpha_k(b_k+\beta_k))}.$$

Hence, the endemic equilibrium of the model (1) is defined as the vector $E_e = (S^*, E^*, I^*, R^*, S_k^*, E_k^*, I_k^*)$ with the components

$$\begin{aligned}
I^* &= \frac{aa_k\alpha\beta\alpha_k\beta_k N_0 N_{0_k} - a'a'_k c_k(b_k+\beta_k)((b+\beta)(d+\mu)(c+\gamma)-\mu\gamma\beta)}{((b+\beta)(d+\mu)(c+\gamma)-\mu\gamma\beta)(\alpha\beta_k\alpha_k a_k N_k + a'c_k\alpha_k(b_k+\beta_k))},\\
S^* &= \frac{aN_0}{\alpha I_k^* + a'},\\
E^* &= \frac{c+\gamma}{\beta}I^*,\\
R^* &= \frac{\gamma}{d+\mu}I^*,\\
S_k^* &= \frac{a_k N_{0_k}}{\alpha_k I^* + a'_k},\\
E_k^* &= \frac{\alpha_k}{b_k+\beta_k}S_k^* I^*,\\
I_k^* &= \frac{a_k\alpha_k\beta_k N_{0_k}}{c_k(b_k+\beta_k)(\alpha_k I^* + a'_k)}I^*.
\end{aligned}$$

The equilibrium E_e represents the endemic point of the model, in which all population subgroups are presented. The host population tends to recover, and the recovery rate depends on the severity of the disease and the strategies adopted to eliminate the disease.

3.1. Determination of the base reproductive number R_0

The base reproductive number R_0 measures the average number of new malaria infections caused by one infected person in a fully susceptible population. To calculate R_0 for a system of equations (1), we use the *next generation matrix method* described in (Ndiaye and Parilina, 2022; Diekmann et al., 2010, Van den Driessche, 2017; Jones, 2007). The required system of equations (1) for our model can be written as

$$\begin{aligned}
\frac{dx}{dt} &= F(x) - V(x),\\
x &= (S, E, I, R, S_k, E_k, I_k)^T.
\end{aligned}$$

Using the next generation method, we will do the following. First we define the matrices F and V:

$$\mathcal{F} = \begin{pmatrix} \alpha_k S_k(t) I(t) \\ 0 \\ \alpha S(t) I_k(t) \\ 0 \end{pmatrix}, \quad \mathcal{V}^+ = \begin{pmatrix} \mu R(t) \\ \beta E(t) \\ 0 \\ \beta_k E_k(t) \end{pmatrix}, \quad \mathcal{V}^- = \begin{pmatrix} -(b+\beta)E(t) \\ -(c+\gamma)I(t) \\ -(b_k+\beta_k)E_k(t) \\ -c_k I_k(t) \end{pmatrix},$$

from which we obtain that

$$\mathcal{V} = \mathcal{V}^+ + \mathcal{V}^- = \begin{pmatrix} \mu R(t) - (b+\beta)E(t) \\ \beta E(t) - (c+\gamma)I(t) \\ -(b_k+\beta_k)E_k(t) \\ \beta_k E_k(t) - c_k I_k(t) \end{pmatrix}.$$

We find the matrices

$$D\mathcal{F}(E_s) = \begin{pmatrix} 0 & \alpha_k S_k^0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & \alpha S^0 \\ 0 & 0 & 0 & 0 \end{pmatrix},$$

$$D\mathcal{V}(E_s) = \begin{pmatrix} -(b+\beta) & 0 & 0 & 0 \\ \beta & -(c+\gamma) & 0 & 0 \\ 0 & 0 & -(b_k+\beta_k) & 0 \\ 0 & 0 & \beta_k & -c_k \end{pmatrix}.$$

Therefore,

$$F = \begin{bmatrix} 0 & \alpha_k S_k^0 \\ 0 & 0 \end{bmatrix}, \quad F' = \begin{bmatrix} 0 & \alpha S^0 \\ 0 & 0 \end{bmatrix},$$
$$V = \begin{bmatrix} -(b+\beta) & 0 \\ \beta & -(c+\gamma) \end{bmatrix}, \quad V' = \begin{bmatrix} -(b_k+\beta_k) & 0 \\ \beta_k & -c_k \end{bmatrix}.$$

Calculate R_0 by the formula $R_0 = \rho(-FV^{-1})$, where

$$V^{-1} = \frac{1}{det(V)} t_{(com(V))},$$

and

$$det(V) = (b+\beta)(c+\gamma), \quad t_{(com(V))} = \begin{bmatrix} -(c+\gamma) & 0 \\ -\beta & -(b+\beta) \end{bmatrix}.$$

Substituting $det(V)$ and $t_{(com(V))}$ into expression of V^{-1}, we get

$$V^{-1} = \frac{1}{(b+\beta)(c+\gamma)} \begin{bmatrix} -(c+\gamma) & 0 \\ -\beta & -(b+\beta) \end{bmatrix},$$
$$(V')^{-1} = \frac{1}{(b_k+\beta_k)c_k} \begin{bmatrix} -c_k & 0 \\ -\beta_k & -(b_k+\beta_k) \end{bmatrix}.$$

Finally, we obtain the following expressions:

$$FV^{-1} = \begin{bmatrix} -\frac{\alpha_k \beta S_k^0}{(b+\beta)(c+\gamma)} & -\frac{\alpha_k S_k^0}{c+\gamma} \\ 0 & 0 \end{bmatrix},$$
$$FV'^{-1} = \begin{bmatrix} -\frac{\alpha \beta_k S^0)}{c_k(b_k+\beta_k)} & -\frac{\alpha S^0}{c_k} \\ 0 & 0 \end{bmatrix},$$

and calculate R_h and R_k:

$$R_h = \rho(-FV^{-1}) = \frac{\alpha_k \beta S_k^0}{(b+\beta)(c+\gamma)}, \quad R_k = \rho(-FV'^{-1}) = \frac{\alpha\beta_k S^0)}{c_k(b_k+\beta_k)},$$

from which we obtain the base reproductive number R_0 in the form

$$R_0 = R_h \times R_k = \frac{\alpha_k \beta S_k^0 \alpha\beta_k S^0}{c_k(b_k+\beta_k)(b+\beta)(c+\gamma)},$$

where $(S^0, S_k^0) = (\frac{a}{a'}N_0, \frac{a_k}{a'_k}N_{0_k})$, and finally we write down the formula for R_0:

$$R_0 = \frac{\alpha\beta\alpha_k\beta_k a a_k N_0 N_{0_k}}{a' a'_k c_k (b+\beta)(c+\gamma)(b_k+\beta_k)}.$$

If $R_0 \leq 1$ and at least one person is infected, but the infection cannot develop, and the system (1) is stable. If $R_0 \geq 1$, i.e., at least one infected person can infect several people, the number of infected humans is growing, and the disease can cover the entire population. A numerical study will give us a more representative picture of the disease spread among the population depending on the number R_0.

Note that an endemic equilibrium point E_e exists if $R_0 > 1$.

3.2. Study of stability at equilibrium points

First we analyze the stability of the disease-free equilibrium of the system of equations (1) using the base reproductive number R_0 in the following theorem.

Theorem 2. *The disease-free equilibrium E_s is locally asymptotically stable if $R_0 \leq 1$ and $c_k > \frac{\alpha\beta\alpha_k\beta_k N_0 N_{0_k}(d+\mu)}{(b+\beta)(c+\gamma)(d+\mu)(b_k+\beta_k)+\beta\gamma\mu(b_k+\beta_k)}$, and unstable if $R_0 > 1$.*

Proof. The Jacobian matrix of the system (1) is written in the form:

$$J(S,E,I,R,S_k,E_k,I_k) = \begin{pmatrix} -\alpha I_k - a' & 0 & 0 & 0 & 0 & 0 & -\alpha S \\ \alpha I_k & -b-\beta & 0 & \mu & 0 & 0 & \alpha S \\ 0 & \beta & -c-\gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -d-\mu & 0 & 0 & 0 \\ 0 & 0 & -\alpha_k S_k & 0 & \alpha_k I - a'_k & 0 & 0 \\ 0 & 0 & \alpha_k S_k & 0 & \alpha_k I & -(b_k+\beta_k) & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_k & -c_k \end{pmatrix}.$$

The Jacobian matrix at the disease-free equilibrium point E_s reads as

$$J(E_s) = \begin{pmatrix} -a' & 0 & 0 & 0 & 0 & 0 & -\alpha\frac{N_0}{a} \\ 0 & -b-\beta & 0 & \mu & 0 & 0 & \alpha\frac{N_0}{a} \\ 0 & \beta & -c-\gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -d-\mu & 0 & 0 & 0 \\ 0 & 0 & -\alpha_k\frac{N_{0_k}}{a_k} & 0 & -a'_k & 0 & 0 \\ 0 & 0 & \alpha_k\frac{N_{0_k}}{a_k} & 0 & 0 & -(b_k+\beta_k) & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_k & -c_k \end{pmatrix}.$$

We define the eigenvalues of this matrix by equating its next determinant, that is,

$$\begin{vmatrix} -a'-\lambda & 0 & 0 & 0 & 0 & 0 & -\alpha\frac{N_0}{a} \\ 0 & -b-\beta-\lambda & 0 & \mu & 0 & 0 & \alpha\frac{N_0}{a} \\ 0 & \beta & -c-\gamma-\lambda & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -d-\mu-\lambda & 0 & 0 & 0 \\ 0 & 0 & -\alpha_k\frac{N_{0_k}}{a_k} & 0 & -a'_k-\lambda & 0 & 0 \\ 0 & 0 & \alpha_k\frac{N_{0_k}}{a_k} & 0 & 0 & -(b_k+\beta_k)-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_k & -c_k-\lambda \end{vmatrix}$$

to zero. We obtain the characteristic equation

$$(a'+\lambda)(a'_k+\lambda)[\lambda^5+C_1\lambda^4+C_2\lambda^3+C_3\lambda^2+C_4\lambda+C_5]=0,$$

where

$$\begin{aligned}
C_1 &= c_k+d+\mu+b_k+\beta_k+b+\beta+c+\gamma,\\
C_2 &= (d+\mu)(b_k+\beta_k)+(b+\beta+c+\gamma)(d+\mu+b_k+\beta_k)\\
&\quad +(b+\beta)(c+\gamma)+c_k(d+\mu+b_k+\beta_k),\\
C_3 &= (b+\beta+c+\gamma)((d+\mu)(b_k+\beta_k)+c_k(d+\mu+b_k+\beta_k))+\\
&\quad +(b+\beta)(c+\gamma)(d+\mu+b_k+\beta_k+c_k)+c_k(d+\mu)(b_k+\beta_k),\\
C_4 &= (b+\beta)(c+\gamma)((d+\mu)(b_k+\beta_k)+c_k(d+\mu+b_k+\beta_k)\\
&\quad +\beta\gamma\mu(c_k+b_k+\beta_k)-\frac{\alpha\beta\alpha_k\beta_kN_0N_{0_k}}{aa_k},\\
C_5 &= c_k(b+\beta)(c+\gamma)(d+\mu)(b_k+\beta_k)+\beta\gamma\mu c_k(b_k+\beta_k)-\alpha\beta\alpha_k\beta_kN_0N_{0_k}(d+\mu).
\end{aligned}$$

The characteristic equation admits seven proper roots, the first two values of which λ_1 and λ_2 are written as: $\lambda_1=-a'$ and $\lambda_2=-a'_k$. The remaining five eigenvalues are obtained from solving the following equation:

$$\lambda^5+C_1\lambda^4+C_2\lambda^3+C_3\lambda^2+C_4\lambda+C_5=0.$$

This equation does not admit trivial solutions, and it is difficult to find the roots of this polynomial. To determine the nature of the stability of the equilibrium point E_s. We use the Routh-Hurwitz criterion to study stability. To do this we write an auxiliary matrix

$$\begin{vmatrix} 1 & C_2 & C_4 & 0 & 0 & 0 \\ C_1 & C_3 & C_5 & 0 & 0 & 0 \\ \frac{C_1C_2-C_3}{C_1} & \frac{C_1C_4-C_5}{C_1} & 0 & 0 & 0 & 0 \\ C_3-\frac{C_1C_4-C_5}{C_1C_2-C_3} & C_5 & 0 & 0 & 0 & 0 \\ \frac{C_1C_4-C_5}{C_1}-\frac{C_5(C_1C_2-C_3)^2}{C_3(C_1C_2-C_3)-C_1C_4+C_5} & 0 & 0 & 0 & 0 & 0 \\ C_5 & 0 & 0 & 0 & 0 & 0 \end{vmatrix}.$$

Applying the Hurwitz criterion, we obtain that the system (1) is asymptotically stable at the equilibrium point E_s if the inequalities are met,

$$\begin{aligned} C_1 &> 0, \\ C_5 &> 0, \\ \frac{C_1C_2 - C_3}{C_1} &> 0, \\ C_3 - \frac{C_1C_4 - C_5}{C_1C_2 - C_3} &> 0, \\ \frac{C_1C_4 - C_5}{C_1} - \frac{C_5(C_1C_2 - C_3)^2}{C_3(C_1C_2 - C_3) - C_1C_4 + C_5} &> 0. \end{aligned}$$

Then from $\frac{C_1C_2-C_3}{C_1} > 0$ and $C_1 > 0$, it follows that $C_1C_2 - C_3 > 0$. The fourth inequality is equivalent to $C_3(C_1C_2 - C_3) - C_1C_4 + C_5 > 0$, or $C_1C_2C_3 - C_1C_4 - C_3^2 + C_5 > 0$. Then the last inequality can be simplified as $(C_1C_4 - C_5)(C_1C_2C_3 - C_1C_4 - C_3^2 + C_5) - C_1C_5(C_1C_2 - C_3)^2 > 0$.

Therefore, we obtain the system:

$$\begin{aligned} C_1 &> 0, \\ C_5 &> 0, \\ C_1C_2 - C_3 &> 0, \\ C_1C_2C_3 - C_1C_4 - C_3^2 + C_5 &> 0, \\ (C_1C_4 - C_5)(C_1C_2C_3 - C_1C_4 - C_3^2 + C_5) - C_1C_5(C_1C_2 - C_3)^2 &> 0. \end{aligned}$$

The first two eigenvalues λ_1 and λ_2 have negative real parts, and the remaining five eigenvalues have negative real parts if they satisfy the Routh-Hurwitz criteria. Thus, all eigenvalues of the characteristic equation have negative real parts if and only if $R_0 < 1$ and $C_1C_2C_3 + C_5 > C_1C_4 + C_3^2$, that is to say $c_k > \frac{\alpha\beta\alpha_k\beta_k N_0 N_{0_k}(d+\mu)}{(b+\beta)(c+\gamma)(d+\mu)(b_k+\beta_k)+\beta\gamma\mu(b_k+\beta_k)}$, which shows that the disease-free equilibrium E_s is locally asymptotically stable. This finishes the proof.

Theorem 3. *The endemic equilibrium point E_e is locally asymptotically stable if $R_0 > 1$ and $c_k > \frac{\alpha\beta\beta_k\alpha_k S_k^*}{(\alpha_k I^* + a'_k)(a' + \alpha I_k^*)(b_k+\beta_k)}$.*

Proof. The Jacobian matrix of the system (1) is written in the form:

$$J(S, E, I, R, S_k, E_k, I_k) = \begin{pmatrix} -\alpha I_k - a' & 0 & 0 & 0 & 0 & 0 & -\alpha S \\ \alpha I_k & -b-\beta & 0 & \mu & 0 & 0 & \alpha S \\ 0 & \beta & -c-\gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -d-\mu & 0 & 0 & 0 \\ 0 & 0 & -\alpha_k S_k & 0 & \alpha_k I - a'_k & 0 & 0 \\ 0 & 0 & \alpha_k S_k & 0 & \alpha_k I & -(b_k+\beta_k) & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_k & -c_k \end{pmatrix}.$$

The Jacobian matrix at the endemic equilibrium point $E_e = (S^*, E^*, I^*, R^*, S_k^*, E_k^*, I_k^*)$ can be written as

$$J(E_e) = \begin{pmatrix} -\alpha I_k^* - a' & 0 & 0 & 0 & 0 & 0 & -\alpha S^* \\ \alpha I_k^* & -b-\beta & 0 & \mu & 0 & 0 & \alpha S^* \\ 0 & \beta & -c-\gamma & 0 & 0 & 0 & 0 \\ 0 & 0 & \gamma & -d-\mu & 0 & 0 & 0 \\ 0 & 0 & -\alpha_k S_k^* & 0 & \alpha_k I^* - a_k' & 0 & 0 \\ 0 & 0 & \alpha_k S_k^* & 0 & \alpha_k I^* & -(b_k+\beta_k) & 0 \\ 0 & 0 & 0 & 0 & 0 & \beta_k & -c_k \end{pmatrix}.$$

We define the eigenvalues of this matrix by equating its determinant to zero, and we obtain the following characteristic equation:

$$\lambda^7 + A_1\lambda^6 + A_2\lambda^5 + A_3\lambda^4 + A_4\lambda^3 + A_5\lambda^2 + A_6\lambda + A_7 = 0,$$

where

$$\begin{aligned}
A_1 &= b+\beta+c+\gamma+d+\mu+a'+\alpha I_k^* + b_k+\beta_k+\alpha_k I^* + a_k' + c_k,\\
A_2 &= (d+\mu)(b+\beta+c+\gamma) + (b+\beta)(c+\gamma) + (b+\beta+c+\gamma+d+\mu+a'\\
&\quad + \alpha I_k^* + b_k+\beta_k+\alpha_k I^* + a_k' + c_k) + c_k(\alpha_k I^* + a_k') + (\alpha I_k^* + a')(b_k+\beta_k)\\
&\quad + (a' + \alpha I_k^* + b_k+\beta_k)(\alpha_k I^* + a_k' + c_k),\\
A_3 &= (d+\mu)(b+\beta)(c+\gamma) + (d+\mu)(b+\beta+c+\gamma) + (b+\beta)(c+\gamma) + a'\\
&\quad + \alpha I_k^* + b_k+\beta_k+\alpha_k I^* + a_k' + c_k + c_k(\alpha_k I^* + a_k') + (\alpha I_k^* + a')(b_k+\beta_k)\\
&\quad + (a' + \alpha I_k^* + b_k+\beta_k)(\alpha_k I^* + a_k' + c_k)(b+\beta+c+\gamma+d+\mu)\\
&\quad + c_k(\alpha_k I^* + a_k')(a' + \alpha I_k^* + b_k+\beta_k) + (\alpha I_k^* + a')(b_k+\beta_k)(\alpha_k I^* + a_k' + c_k),\\
A_4 &= (a' + \alpha I_k^* + b_k+\beta_k+\alpha_k I^* + a_k' + c_k)(d+\mu)(b+\beta)(c+\gamma)\\
&\quad + [c_k(\alpha_k I^* + a_k') + (\alpha I_k^* + a')(b_k+\beta_k) + (a' + \alpha I_k^* + b_k+\beta_k)(\alpha_k I^* + a_k' + c_k)]\\
&\quad \times [(d+\mu)(b+\beta+c+\gamma) + (b+\beta)(c+\gamma)] + (b+\beta+c+\gamma+d+\mu)\\
&\quad \times [c_k(\alpha_k I^* + a_k')(a' + \alpha I_k^* + b_k+\beta_k) + (\alpha I_k^* + a')(b_k+\beta_k)(\alpha_k I^* + a_k' + c_k)]\\
&\quad + c_k(\alpha_k I^* + a_k')(a' + \alpha I_k^*)(b_k+\beta_k) - \alpha\beta\beta_k\alpha_k S_k^*,\\
A_5 &= [c_k(\alpha_k I^* + a_k')(a' + \alpha I_k^*)(b_k+\beta_k) + (a' + \alpha I_k^* + b_k+\beta_k)(\alpha_k I^* + a_k' + c_k)]\\
&\quad \times (b+\beta)(c+\gamma)(d+\mu) + [c_k(\alpha_k I^* + a_k')(a' + \alpha I_k^* + b_k+\beta_k) + c_k(\alpha_k I^* + a_k')\\
&\quad \times (a' + \alpha I_k^*)(b_k+\beta_k)(\alpha_k I^* + a_k' + c_k)][(d+\mu)(b+\beta+c+\gamma) + (b+\beta)(c+\gamma)]\\
&\quad + c_k(b+\beta+c+\gamma+d+\mu)(\alpha_k I^* + a_k')(a' + \alpha I_k^*)(b_k+\beta_k)\\
&\quad - \alpha\beta\beta_k\alpha_k S_k^*(d+\mu+a_k' + \alpha S^* I^* + \alpha I_k^* + a'),\\
A_6 &= (d+\mu)(b+\beta)(c+\gamma)[c_k(\alpha_k I^* + a_k')(a' + \alpha I_k^* + b_k+\beta_k)\\
&\quad + (\alpha I_k^* + a')(b_k+\beta_k)(\alpha_k I^* + a_k' + c_k)] + c_k(\alpha_k I^* + a_k')(a' + \alpha I_k^*)(b_k+\beta_k)\\
&\quad \times [(d+\mu)(b+\beta+c+\gamma) + (b+\beta)(c+\gamma)]\\
&\quad - \alpha\beta\beta_k\alpha_k S_k^*(a_k'(d+\mu) + (d+\mu+a_k')(\alpha S^* I^* + \alpha I_k^* + a')),\\
A_7 &= c_k(\alpha_k I^* + a_k')(a' + \alpha I_k^*)(b_k+\beta_k)(d+\mu)(b+\beta)(c+\gamma)\\
&\quad - \alpha\beta\beta_k\alpha_k S_k^* a_k'(d+\mu)(\alpha S^* I^* + \alpha I_k^* + a').
\end{aligned}$$

The eigenvalues of this matrix are the solutions of the characteristic equation. The equation admits seven eigenvalues. We use the Routh-Hurwitz criterion, which states

that all roots of a characteristic equation have negative real parts if and only if the conditions of the Routh-Hurwitz criteria are satisfied.

We use the Hurwitz criterion to study stability, and write an auxiliary matrix

$$\begin{vmatrix} 1 & A_2 & A_4 & A_6 & 0 & 0 \\ A_1 & A_3 & A_5 & A_7 & 0 & 0 \\ \dfrac{A_1A_2-A_3}{A_1} & \dfrac{A_1A_4-A_5}{A_1} & \dfrac{A_1A_6-A_7}{A_1} & 0 & 0 & 0 \\ A_3-\dfrac{A_1(A_1A_4-A_5)}{A_1A_2-A_3} & A_5-\dfrac{A_1(A_1A_6-A_7)}{A_1A_2-A_3} & A_7 & 0 & 0 & 0 \\ A_1' & A_2' & 0 & 0 & 0 & 0 \\ A_3' & A_7 & 0 & 0 & 0 & 0 \\ \dfrac{A_3'A_2'-A_1'A_7}{A_3'} & 0 & 0 & 0 & 0 & 0 \\ A_7 & 0 & 0 & 0 & 0 & 0, \end{vmatrix},$$

where

$$\begin{aligned} A_1' &= \frac{A_1A_4-A_5}{A_1} - \frac{(A_1A_2-A_3)(A_5(A_1A_2-A_3)-A_1(A-1A_6-A_7))}{A_1(A_3(A_1A_2-A_3)-A_1(A_1A_4-A_5))}, \\ A_2' &= \frac{A_1A_6-A_7}{A_1} - \frac{A_7(A_1A_2-A_3)^2}{A_1(A_3(A_1A_2-A_3)-A_1(A_1A_4-A_5))}, \\ A_3' &= A_5 - \frac{A_1(A_1A_6-A_7)}{A_1A_2-A_3} - \frac{A_3(A_1A_2-A_3)-A_1(A_1A_4-A_5)}{A_1A_2-A_3}\frac{A_2'}{A_1'}. \end{aligned}$$

Applying the Hurwitz criterion, we obtain that the system (1) is asymptotically stable at the equilibrium point E_e if the inequalities are met

$$\begin{aligned} A_1 &> 0, \\ A_7 &> 0, \\ \frac{A_1A_2-A_3}{A_1} &> 0, \\ A_3 - \frac{A_1(A_1A_4-A_5)}{A_1A_2-A_3} &> 0, \\ \frac{A_1A_4-A_5}{A_1} - \frac{(A_1A_2-A_3)(A_5(A_1A_2-A_3)-A_1(A-1A_6-A_7))}{A_1(A_3(A_1A_2-A_3)-A_1(A_1A_4-A_5))} &> 0, \\ \frac{A_1A_6-A_7}{A_1} - \frac{A_7(A_1A_2-A_3)^2}{A_1(A_3(A_1A_2-A_3)-A_1(A_1A_4-A_5))} &> 0, \\ A_5 - \frac{A_1(A_1A_6-A_7)}{A_1A_2-A_3} - \frac{A_3(A_1A_2-A_3)-A_1(A_1A_4-A_5)}{A_1A_2-A_3}\frac{A_2'}{A_1'} &> 0 \\ \frac{A_3'A_2'-A_1'A_7}{A_3'} &> 0, \end{aligned}$$

Then from $\frac{A_1A_2-A_3}{A_1} > 0$ and $A_1 > 0$, it follows that $A_1A_2 - A_3 > 0$. The fourth inequality is equivalent to $A_3(A_1A_2 - A_3) - A_1(A_1A_4 - A_5) > 0$, or $A_1A_2 - A_3 > 0$. The fifth inequality is equivalent to $A_1(A_1A_4 - A_5)(A_3(A_1A_2 - A_3) - A_1(A_1A_4 - A_5)) - A_1(A_1A_2 - A_3)(A_5(A_1A_2 - A_3) - A_1(A - 1A_6 - A_7)) > 0$, or $A_1^2(A_3(A_1A_2 - A_3) - A_1(A_1A_4 - A_5) > 0$. The sixth inequality is equivalent to $A_1(A_1A_6 - A_7)(A_3(A_1A_2 - A_3) - A_1(A_1A_4 - A_5)) - A_1A_7(A_1A_2 - A_3)^2 > 0$, or

$A_1' > 0$. The seventh inequality is equivalent to $A_5A_1'(A_1A_2 - A_3) - A_1A_1'(A_1A_6 - A_7) - A_2'(A_3(A_1A_2 - A_3) - A_1(A_1A_4 - A_5)) > 0$. Then the last inequality can be simplified as $A_3'A_2' - A_1'A_7 > 0$, or $A_3' > 0$.

Therefore, we obtain the system:

$$\begin{aligned}
A_1 &> 0,\\
A_7 &> 0,\\
A_1A_2 - A_3 &> 0,\\
A_3(A_1A_2 - A_3) - A_1(A_1A_4 - A_5) &> 0,\\
A_1(A_1A_4 - A_5)(A_3(A_1A_2 - A_3) - A_1(A_1A_4 - A_5))-&\\
-A_1(A_1A_2 - A_3)(A_5(A_1A_2 - A_3) - A_1(A - 1A_6 - A_7)) &> 0,\\
A_1(A_1A_6 - A_7)(A_3(A_1A_2 - A_3) - A_1(A_1A_4 - A_5)) - A_1A_7(A_1A_2 - A_3)^2 &> 0,\\
A_5A_1'(A_1A_2 - A_3) - A_1A_1'(A_1A_6 - A_7) - A_2'(A_3(A_1A_2 - A_3)-&\\
-A_1(A_1A_4 - A_5)) &> 0\\
A_3'A_2' - A_1'A_7 &> 0,
\end{aligned}$$

The seven eigenvalues have negative real parts if they meet the Routh-Hurwitz criteria. Thus, all eigenvalues of the characteristic equation have negative real parts if and only if $R_0 > 1$ and $A_3A_1A_2 + A_1A_5 > A_3^2 + A_1^2A_4$, that is satisfied when $c_k > \frac{\alpha\beta\beta_k\alpha_kS_k^*}{(\alpha_kI^* + a_k')(a' + \alpha I_k^*)(b_k + \beta_k)}$, which shows that the endemic equilibrium E_e is locally asymptotically stable. This finishes the proof.

4. Numerical Simulations

Numerical modeling is one of the best methods to understand the dynamics of malaria. It gives us the evolution of each subgroup of the population depending on the severity of the disease. In our model, we made several representations of the dynamics of malaria disease in the time interval $[0, 60]$ with different values of parameters, i.e., different R_0. The resulting curves are performed using Matlab software. The parameters, for which the numerical simulations are performed, are presented in the tables.

Table 1. Parameters for modeling represented in Fig. 3

α	α_k	β	β_k	γ	γ_k	μ	μ_k	a	a'	a_k	a_k'	b	b_k	c	c_k	d	d_k	R_0
In Fig. 3 (first run)																		
0.9	0.7	1.2	0.5	0.4	0.5	0.15	0.2	0.3	0.2	0.4	0.3	0.3	0.7	0.03	0.4	0.15	0.25	2.44
In Fig. 3 (second run)																		
1.8	0.5	1.5	0.6	0.3	0.5	0.1	0.15	0.6	0.3	0.4	0.2	0.5	0.65	0.01	0.4	0.12	0.17	10.45

In Figure 3 we represent the two runs of simulations, for which $R_0 = 2.44$ (the first row of graphs), and $R_0 = 10.45$ (the second row of graphs). We can notice that the disease exists in the populations (the host and vector ones), and that the representative curves of susceptible subpopulations decrease, and the existing population in it changes into a different behavior. At the same time, the representative curves of the subpopulations (exposed, infected and recovered) converges to the equlibrium values, and we notice a significant presence of the disease in the population. If no

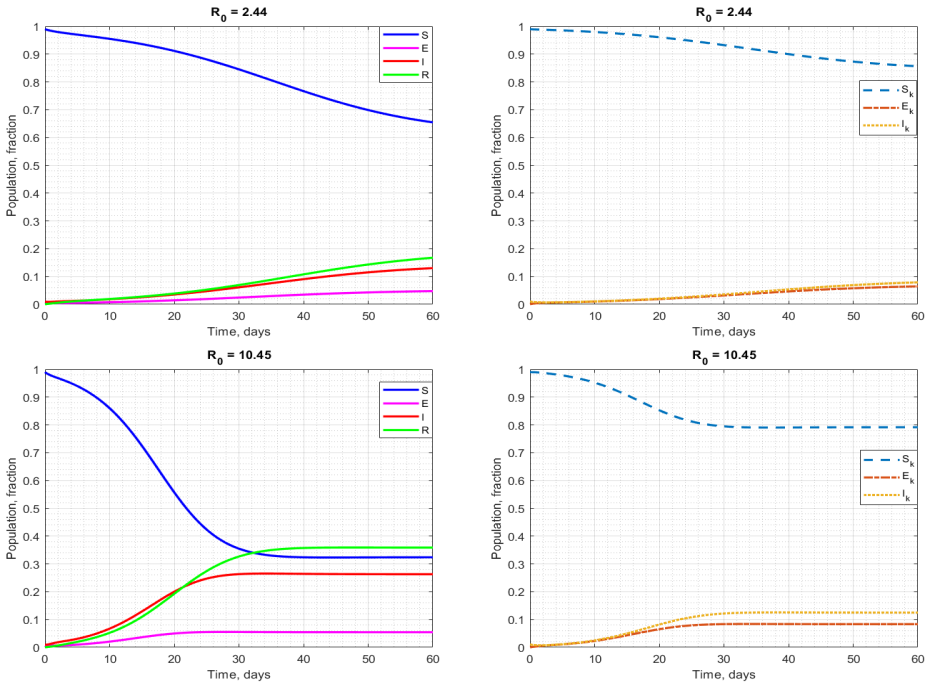

Fig. 3. The epidemic process for various values $R_0 > 1$ ($R_0 = 2.44$ and $R_0 = 10.44$)

intervention is planned, there is a risk that the disease will persist in the population, since the obtained baseline reproduction rate shows that at least one infected person can infect several people (it is true for both runs).

Table 2. Parameters for modeling represented in Fig. 4

α	α_k	β	β_k	γ	γ_k	μ	μ_k	a	a'	a_k	a'_k	b	b_k	c	c_k	d	d_k	R_0
In Fig. 4 (first run)																		
0.7	0.8	0.9	0.7	0.5	0.4	0.3	0.4	0.4	0.316	0.6	0.3	0.5	0.8	0.65	0.8	0.2	0.3	0.46
In Fig. 4 (second run)																		
0.8	0.7	0.9	0.6	0.5	0.65	0.2	0.3	0.4	0.35	0.5	0.45	0.4	0.75	0.02	0.5	0.05	0.3	0.84

In Figure 4 we represent the two runs of simulations, for which $R_0 = 0.46$ (the first row of graphs) and $R_0 = 0.84$ (the second row of graphs), we can notice that the disease is practically absent and that all representative curves of subpopulations (the host and vector ones) are almost linear. It explains that the sizes of the subpopulations are almost constants. The disease does not pose a danger to the population for these sets of parameters since each infected person infects less than one person, which explains why the disease can disappear from the population after some time.

In Figure 5 we represent the two runs of simulations, for which $R_0 = 23.23$ (the first row of graphs) and $R_0 = 0.04$ (the second row of graphs). In the first run of simulations, we should notice that the size of infected subpopulations (both host and vector) increases significantly, as shown by the corresponding curve (see the first row of the graphs), which grows exponentially. The entire population is at risk

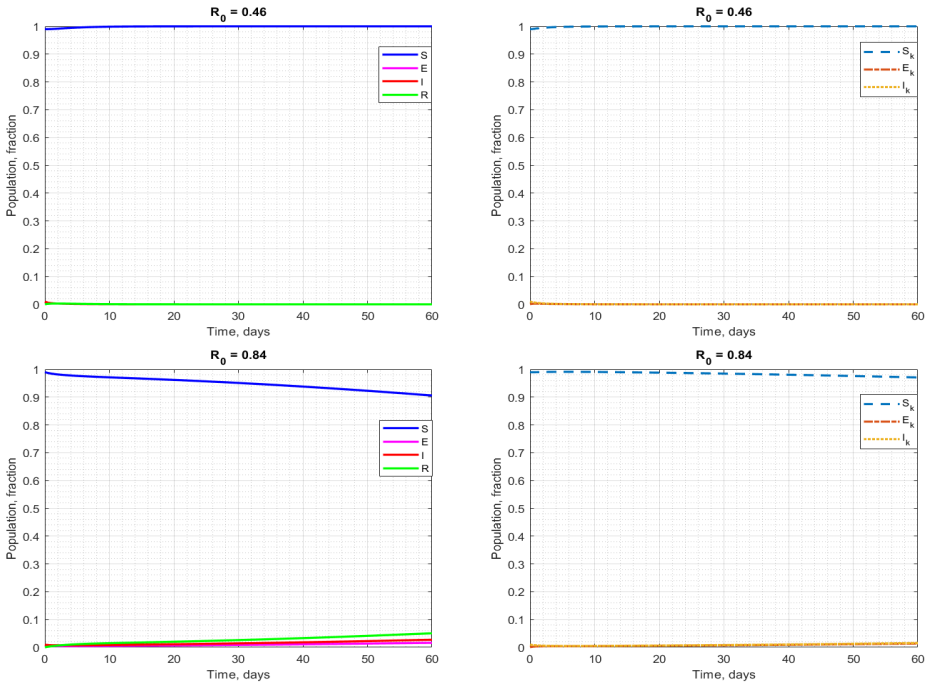

Fig. 4. The epidemic process for various values $R_0 < 1$ ($R_0 = 0.46$ and $R_0 = 0.84$)

Table 3. Parameters for modeling represented in Fig. 5

α	α_k	β	β_k	γ	γ_k	μ	μ_k	a	a'	a_k	a'_k	b	b_k	c	c_k	d	d_k	R_0
In Fig. 5 (first run)																		
0.7	0.8	0.9	0.7	0.5	0.6	0.3	0.4	0.4	0.4	0.6	0.3	0.45	0.55	0.05	0.45	0.2	0.3	23.23
In Fig. 5 (second run)																		
0.7	0.8	0.9	0.7	0.5	0.4	0.3	0.4	0.4	0.4	0.6	0.3	1.5	1.8	1.65	1.8	0.2	0.3	0.04

of infection if measures are not taken to combat the disease. On the second run, we represent the case when $R_0 = 0.04$, i.e., the basic reproduction number is almost zero, which explains the absence of the disease. The population remains stable.

We can see that the base reproductive number R_0 plays an important role in the study of mathematical modeling of diseases. This allows us to examine the speed of the spread of the disease among the population.

5. Conclusions

Malaria is a tropical infectious disease. By these days, scientists have failed to develop an effective vaccine to combat this disease which can be very dangerous with many deaths in the human population. Mathematical modeling of this disease plays a crucial role in understanding of transmission dynamics and appropriate prevention strategies. In this paper, we study the $SEIRS_kE_kI_k$ model to give predictions of the spread of malaria. For the model, we examine two stable equilibrium points: a disease-free equilibrium, in which the disease is not present in the populations; and an endemic equilibrium point, when there is a non-zero infected subpopula-

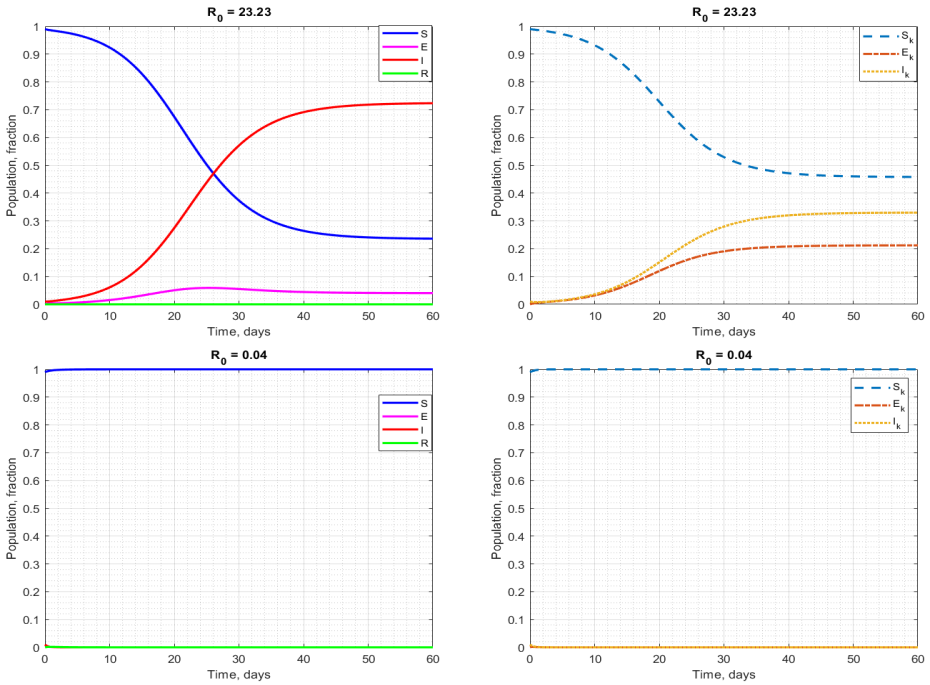

Fig. 5. The epidemic process for various values R_0 ($R_0 = 23.23$ and $R_0 = 0.04$)

tion. We establish the stability results for these two equilibria. We use the theory of the Lyapunov functions to examine stability of both equilibrium points. It is proved that the dynamical process is completely determined by the base reproduction number R_0. If $R_0 \leq 1$, the disease-free equilibrium is locally asymptotically stable. If $R_0 > 1$, there is an endemic equilibrium that is globally asymptotically stable. The simulation results significantly showed how the disease spreads among the population. The spread of this disease can be prevented through effective awareness-raising strategies in regions where it has spread rapidly.

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