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MATHEMATICAL MODEL OF MALARIA WITH SEASONAL EVOLUTION OF MOSQUITOES POPULATION: CASE OF BURKINA FASO

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Abstract. This work is devoted to the formulation and study of mathematical model of malaria transmission taking into account the seasonal evolution of the vector populations.The aim is to examine the impact of the seasonal variation of mosquitoes on the fluctuation of malaria cases in Burkina Faso. The human population is divide to susceptible (S_h), exposed (E_h), infected (I_h) and recovered (R_h). The vector population is divided to susceptible (S_v) and infected (I_v). The basic mathematical properties of the model such as the boundedness and positivity of the solutions are established. The basic reproduction number R_0 of the model is determined. The global stability of the endemic and disease free equilibrium point is proven. The impact of seasonality and temperature on vector dynamics (the function f) is developed to determine the intense period of mosquitoes. The sensitivity of the parameters is studied to determine the most sensitive parameters in the evolution of the malaria disease. We use the Python software for the numerical simulation of the model. The real data on the number of malaria cases in Burkina Faso is used in the simulation section to illustrate the mathematical analysis. This study highlights that an increase in the mosquito population leads to a rise in malaria cases, while their reduction results in a decrease in cases. It also shows that the month with a high incidence of malaria in BURKINA Faso are July, August, September and October, whereas the months with fewer cases are July and February.

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1. Introduction

Malaria is an infectious disease transmitted by a mosquito called the female anophele. It was first discovered in India in the 15^{th} century ([30]). The analysis of the body of a patient died in Italy showed that the death was due to an infection called a malaria infection (Italian language). Thereafter this disease is recognized by the name malaria. It wreaked havoc in Europe during the 16^{th} century, in America through the slave trade, and nowadays hitting Africa. In year 730, the Spanish Francisco Lopez discovered the curative tree called quinine already used by the Indians to treat fever. In 1820, the french pharmacist Pelletier Caventou isolated the quinine for the first time from quinine bark. In 1880, the French doctor Alphonse Lavernat undertook observations with a microscope for the first time and discovered the malaria parasite called falciparum, hence the name malaria. In 1897 the british Roland Ross made the discovery that the female anophele mosquito spreads the disease of malaria. Until the 20^{th} century quinine remained the only medicine to treat malaria, then the other medicines were discovered later on in 1940. After spraying products (DDT) to kill anopheles in the surrounding environment in 1956, the World Health Organization (WHO) launched a global malaria eradication program. The malaria parasite fights back and became increasingly resistant to treatment. In 2001, the WHO advocated a new therapy, the action of combining the old artemisinin molecules already used by the Chinese (since the 4^{th} Century) with two other antimalarial. Resistance to this new treatment is quickly established by the parasites ([30]). Malaria, the main cause of more than 500000 deaths per year, is the first parasitic disease in the world, particularly in Africa ([30]). In 2013, more than 1980000 peoples were infected mainly in poor countries. Malaria affects more than 100 countries in the world, particularly in tropical areas. Africa who only concentrates more than 98% of malaria cases comes first before Asia and Middle Est. Nigeria and the Democratic Republic of Congo are the most infected in Africa. Over the past 10 years, the mortality rate from malaria was reduced by 50% thanks to the efforts of the WHO. Several factors explain the progress of the treatment of malaria stubs prevention (mosquito net, mosquito spraying, prevention treatment of pregnant women, children,...), the new combination of molecules with ACT. In terms of number of infections, children are the most vulnerable victims. Every minute a child under the age of 5 years dies from malaria worldwide. Access to care and screening for the parasite remains a real challenge in the fight against malaria. In Africa more than 70% of patients could be treated with ACT. Recently the cases of resistance of the parasites to the ACT is observed in some countries (Myanmar, Camboge, Thailand,...). The female anophele mosquito remains the vector of the parasite. Anopheles bites at night to feed on blood. If the individual is already infected the anopheles sucks the blood of the subject with many parasites develop in the stomach of the anophele female. To the opportunity to sting a healthy person the parasites infect the blood of the new victim. After infection the parasite enters a phase of new mutation. Sporozoites became trophozoite then merozoites and finally gametocytes. All these metamorphoses allow to escape the dam and remain in the area undetected by the immune system of new infected people. The first destination of the parasites is to reach the blood without being detected by the system. There the parasite can develop quietly and infect the person cells to be released massively into the blood causing the first symptoms(fevers, headaches, lack of appetite,...). The parasite then contaminates the red blood cells quickly and causes their burst. This new generation of the case of falciparum could be sucked up again for a anophele mosquito and the cycle is complete. In the case of falciparum malaria, it can worsen and progress to other organs such as the brain. Young children whose immune system is under construction and pregnant women whose immune system change during pregnancy are most at risk of severe malaria and death. If left untreated within 24 hours after infection, falciparum malaria can progress to sever infection (affect the brain) or even fatal. The parasite is commonly resistant to single molecule treatment. The most

active treatment of malaria is the combination of artemisinin (ACT). Since then, there is appearance of case of malaria resistant to derivative products of ACT. This resistance can be a serious threat in the case of endemic areas. Since the years 2011, the effective way of preventing seasonal malaria for children under 5 years and pregnant women, has been the use of nets, insecticide,... More than three billion people are exposed to malaria throughout the world (see [30]). In 2015 WHO declared more than 3,2 billion new cases of malaria per year ([30]). Among which 92% in Africa and 70% cases are children. The most dangerous anopheles which are very resistant in Africa can be found in western Africa. The pathogen which are parasites are protozoans type of genus plasmodium. The resistance of the parasite to the different treatment leads to a endemic situation. This deserves the attention of researches around the world especially mathematicians. The most used means are the preventive ones. A number of recent mathematical studies of malaria show the significant direct role of climate such as temperature and rainfall, play on the transmission dynamic of vectors ([1, 5, 8, 11, 13, 16, 18, 19, 20, 22, 24, 27, 31]). B. Traoré et al. studied a mathematical model of malaria taking into account mosquito larvae and transmission of malaria in a periodic environment([26]), considering a constant recruitment of both humans and larvaes. Abba B. Gumel et al. studied a malaria model taking into account the temperature and rainfall variation in the mode of malaria transmission ([16]), considering a constant recruitment of humans. CN. Ngonghala et al. study modeling the synergistic interplay between malaria dynamics and economic growth ([15]) by considering a constant recruitment of both vectors and humans population. Our contribution consists to take into account a non-constant recruitment of mosquitoes and humans in order to better describe the reality. In this paper, we develop a mathematical model of malaria that incorporates the dynamic recruitment of the human population as well as the seasonal evolution of the vectors. Our model takes into account the seasonal evolution of mosquitoes during the year to establish a link between the seasonal evolution of mosquitoes and the number of malaria cases. We use a periodical function f of 12 months period to be able to determine the period of abundance of mosquitoes in order to allow the government to support the vulnerable population (distribution of mosquito nets, preventive medicine to children under 5 years old, spraying of areas, ...). Additionally, real data on the number of malaria cases in Burkina Faso is used in the simulation section to illustrate the theoretical results.

The structure of the paper is as follows. We present the mathematical model in Section 2. We give the mathematical analysis of model in Section 3. We study the global stability of disease free equilibrium (DFE) point in Section 4. In Section 5, we study the global stability of endemic equilibrium point. In Section 6, we study the sensitivity of the parameters. We give a numerical simulation in Section 7. We conclude in Section 8.

2. The formulation of mathematical model

In this model, the human population is divided into four classes: the susceptible S_h, the exposed E_h, the infected I_h and cured R_h. The vector population (mosquitoes) is subdivided into two classes: susceptible vector S_v and infected I_v. β_{h1} is the infection probability from exposed person (E_h) to mosquito, β_{h1} is the infection probability from infected person (I_h) to mosquito, β_v represents the infection probability from mosquito to human and b_1 is the average number of bites per mosquito per day. $\beta = b_1\beta_v$ is the rate of infection from mosquitoes to humans. $\alpha_{E_h} = b_1\beta_{h1}$ represents the infection rate from exposed person to mosquitoes. $\alpha_{I_h} = b_1\beta_{h2}$ represents the infection rate from infected person to mosquitoes. $\mu_h N_h$ is the dynamic recruitment of the human population. $\gamma_1 S_h$, $\gamma_1 E_h$, $\gamma_1 I_h$ and $\gamma_1 R_h$ are the number of susceptible, exposed, infected and cured individuals respectively that naturally mortality. $\frac{\beta I_v S_h}{N_h}$ is the proportion of susceptible humans that come infected per bite infected anopheles (I_v). $\frac{\alpha_{E_h} E_h S_v}{N_v}$ and $\frac{\alpha_{I_h} I_h S_v}{N_v}$

are respectively the proportion of anopheles that bite exposed (E_h) and infected humans (I_h) than can infect them. γE_h is the total exposed population that manifests malaria disease at time t (exposed individuals who pass into the I_h class). θI_h is the set of sick humans who recover from malaria (humans who enter the R_h class). $\mu_v N_v(1 + f(t))$ is the recruitment of mosquitoes according to the periodic time of the year. μS_v and μI_v are the mosquitoes that die naturally respectively in class S_v and I_v. In our work, the recruitment is dynamic so that any individual cured of malaria disease can recontact it.

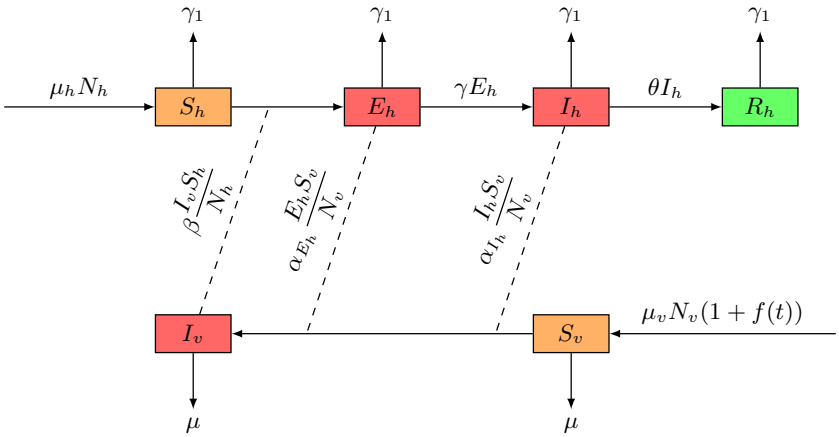

FIGURE 1. Transfer diagram: the black dashed arrows indicate the direction of the infection, the solid arrows represent the transition from one class to another.

According to diagram (1) and by inspired to the model of [32], we have the mathematical model:

$$\begin{cases} \dot{S}_h = \mu_h N_h - \beta \dfrac{I_v S_h}{N_h} - \gamma_1 S_h, \\ \dot{E}_h = \beta \dfrac{I_v S_h}{N_h} - \gamma E_h - \gamma_1 E_h, \\ \dot{I}_h = \gamma E_h - \theta I_h - (\gamma_1 + \gamma_2) I_h, \\ \dot{R}_h = \theta I_h - \gamma_1 R_h, \\ \dot{S}_v = \mu_v N_v \big(1 + f(t)\big) - \alpha_{E_h} \dfrac{E_h S_v}{N_v} - \alpha_{I_h} \dfrac{I_h S_v}{N_v} - \mu S_v, \\ \dot{I}_v = \alpha_{E_h} \dfrac{E_h S_v}{N_v} + \alpha_{I_h} \dfrac{I_h S_v}{N_v} - \mu I_v, \end{cases} \tag{1}$$

with the initial conditions:

$$S_h(0) > 0,\ S_v(0) > 0,\ E_h(0) > 0,\ I_h(0) > 0,\ I_v(0) > 0,\ R_h(0) > 0.$$

TABLE 1. The parameters description of model (1)

Symbols	**Description**
μ_h	Recruitment rate of humans.
γ_1	Natural mortality rate of humans.
μ	Natural mortality rate of mosquitoes.
μ_v	Recruitment rate of mosquitoes.
θ	Transition rate from states I_h to R_h.
α_{I_h}	Contact rate of susceptible mosquitoes with humans infected by malaria.
α_{E_h}	contact rate of susceptible mosquitoes with humans exposed to malaria.
$f(t)$	Periodic function.
β	Contact rate of infected mosquitoes with susceptible humans.
γ_2	Disease mortality rate.
γ	Transition rate from E_h to I_h.

Total human populations and number of vectors are described by the equations (2) and (3).

$$\dot{N}_h = \mu_h N_h(t) - \gamma_1 N_h(t) - \gamma_2 I_h(t) \tag{2}$$

and

$$\dot{N}_v = (1 + f(t))\, \mu_v N_v(t) - \mu N_v(t). \tag{3}$$

The $\omega-$periodic ($\omega = 12$ months) function f is continuous and bounded on $\mathbb{R}$. It describes the variation of mosquitoes during the 12 months of the year. It represents the rate of change of mosquitoes over the 12 months of the year. The rainfall and temperature are important factors in mosquitoes development. Since the increase in mosquitoes leads to an increase in contact between mosquitoes and humans, we assist to an increase of malaria cases during these periods. The variation of temperature plays an important role in the variation of rainfall, which favors mosquito breeding and proliferation. The function f determines the rate of increase of mosquitoes during these periods. After the rainy season the mosquito nits disappear which leads to a decrease of mosquitoes. The

determination of the periods of abundance of mosquitoes allows the government to take measures against malaria by spraying mosquitoes in public space, distributing mosquito nets and distributing preventive drugs to children under five years. Now we can define the function f as follow

$$f(t) = a\cos\left(k\left(\frac{\pi t}{6} + b\right)\right) + c; \quad t \geq 0, \quad a, b, k, c \in \mathbb{R}, \tag{4}$$

where a, b, c and k are constants chosen such that $1 + f(t)$ is positive.

Remark 1. *In our model, mosquitoes do not recover from malaria. Each person cured of malaria is brought back into the susceptible population and since recruitment is dynamic then it takes into account that each individual cured of malaria disease becomes susceptible again. To avoid extinction of different population, we assume in the following that* $\mu_h > \gamma_1$ *and* $\mu_v > \mu$.

Estimation of the total vector population at time t. Let's consider equation (3),

$$\begin{aligned} \dot{N}_v = (1 + f(t))\,\mu_v N_v(t) - \mu N_v(t) \quad &\Leftrightarrow \quad \dot{N}_v = ((1 + f(t))\,\mu_v - \mu)\,N_v(t), \\ &\Leftrightarrow \quad \ln N_v(t) = \int f(t)dt + \mu_v t - \mu t + \lambda; \quad \lambda \in \mathbb{R}. \end{aligned}$$

The vectors total population is estimated at time t by

$$N_v(t) = \lambda \exp\left(\int f(t)dt + \mu_v t - \mu t\right); \quad \lambda \in \mathbb{R}, \quad t \geq 0. \tag{5}$$

For a=1, k=1, $b = -\pi$ and c=0, the relation (5) gives :

$$N_v(t) = N_v(0) \exp\left(\frac{6}{\pi}\sin\left(\left(\frac{\pi t}{6} - \pi\right)\right) + \mu_v t - \mu t\right); \quad t \geq 0. \tag{6}$$

The function $f(t)$ can be seen in the Figure 2.

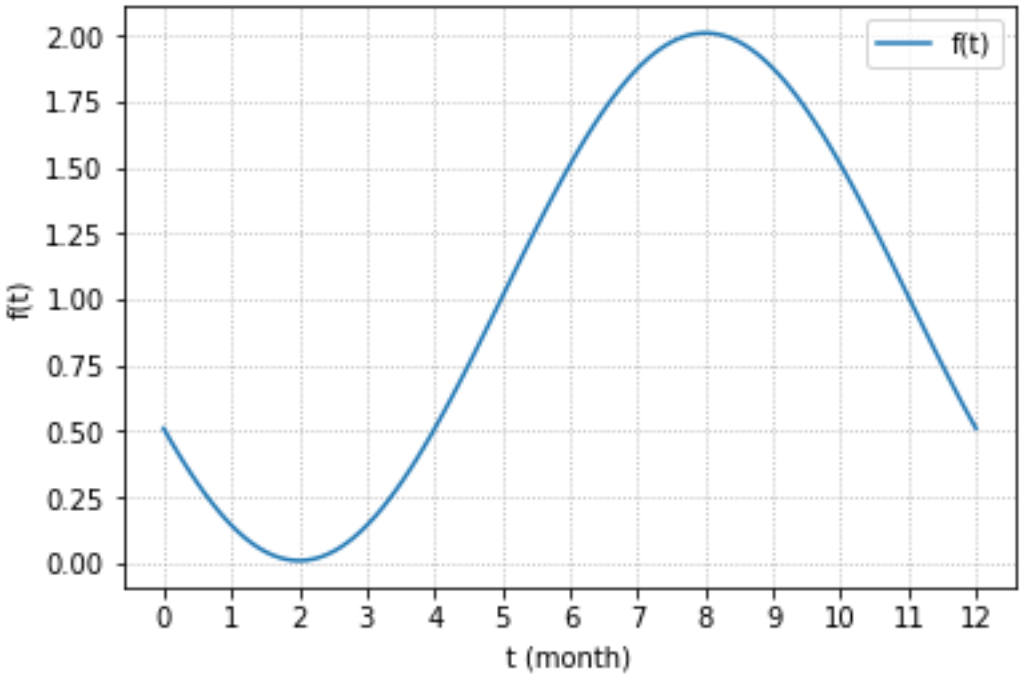

FIGURE 2. The seasonal evolution of mosquitoes during the twelve months of the year with a=1, k=1, $b = \pi/2$ and c=0.

We observe the seasonal evolution of mosquitoes during the twelve months of the year. The lowest number of mosquitoes in the year occurs in February and the period with the highest number of mosquitoes in the year is August. The decrease of mosquitoes during January and February can be explained by the absence of rainfall and the change of temperature. The most favorable period for the proliferation of mosquitoes is May and Jun, reaching their maximum numbers in August. In west Africa, rainfall is more abundant in the open season, which means that mosquito needs are much greater, favoring the proliferation of mosquitoes. After August, the mosquitoes decrease due to heavy rains that destroy their nets. Prevention efforts in the fight against malaria should be concentrated in August and the subvention of care in September and October.

Remark. When an infectious mosquito bites a susceptible person, the parasite enters in the body of the person with rate β and this person moves to the exposed class E_h. Some times after, he moves from class E_h to the infectious class I_h with constant rate γ. The infectious person heals and enters to class R_h at rate θ after treatment. In the same way, when an susceptible mosquito (S_v) becomes infected (I_v) by biting an infectious persons (I_h, E_h). This mosquito (I_v) can now infect other people (S_h). Each infected mosquito no longer recovers but remains infected until its death.

3. Mathematical analysis

We are now interested in the mathematical analysis of the model (1). Firstly we prove the positivity and the boundedness of the solutions and secondly the estimation of the basic reproduction number of system (1).

3.1. **Positivity and boundedness of solutions.** In this part, we prove the positivity and boundedness of solutions of system (1).

Proposition 1. *The unique solution of system (1) is positively invariant in*

$$\Omega = \left\{(S_h(t), E_h(t), I_h(t), S_v(t), I_v(t)) \in \mathbb{R}^6; (S_h(0), E_h(0), I_h(0), R_h(0), S_v(0), I_v(0)) \geq 0\right\}.$$

Proof. For uniqueness of solution, we consider the same techniques in [26]. Let us define the following function.

$$\dot{Y}(t) = h(t, Y(t)), \tag{7}$$

where

$$Y(t) = \begin{pmatrix} S_h(t) \\ E_h(t) \\ I_h(t) \\ R_h(t) \\ S_v(t) \\ I_v(t) \end{pmatrix}; \quad Y(0) > 0$$

and

$$h : \mathbb{R}^+ \times \mathbb{R}^6 \longrightarrow \mathbb{R}^6, \tag{8}$$

such as

$$h(t, Y(t)) = \begin{pmatrix} \mu_h N_h - \beta \dfrac{I_v S_h}{N_h} - \gamma_1 S_h \\ \beta \dfrac{I_v S_h}{N_h} - \gamma E_h - \gamma_1 E_h \\ \gamma E_h - \theta I_h - (\gamma_1 + \gamma_2) I_h \\ \theta I_h - \gamma_1 R_h \\ \mu_v N_v \big(1 + f(t)\big) - \alpha_{E_h} \dfrac{E_h S_v}{N_v} - \alpha_{I_h} \dfrac{I_h S_v}{N_v} - \mu S_v \\ \alpha_{E_h} \dfrac{E_h S_v}{N_v} + \alpha_{I_h} \dfrac{I_h S_v}{N_v} - \mu I_v \end{pmatrix}.$$

For any $Y \in \Omega$, $h(t, Y(t))$ is continuous in $(t, Y(t))$ and Lipschitzian in Y. By application of theorem 2.2.1 and theorem 2.2.3 of Hale and Verduyn Lunel ([7]), the system (1) has a unique solution in Ω.
By considering the first equation of system (1), we have

$$\begin{aligned} \dot{S}_h(t) &= \mu_h N_h - \beta_{I_v} \frac{I_v S_h}{N_h} - \gamma_1 S_h(t), \\ \dot{S}_h &\geq -S_h(t) \left(\beta I_v(t) + \gamma_1\right), \\ S_h(t) &\geq S_h(0) \exp\left(-\left(\beta ||I_v||_\infty + \gamma_1\right) t\right) > 0, \\ S_h(t) &\geq 0, \quad \forall t \in \mathbb{R}^+. \end{aligned}$$

We use the second equation of system (1) to obtain

$$\begin{aligned} \dot{E}_h(t) &= \beta \frac{I_v S_h}{N_h} - \gamma E_h - \gamma_1 E_h, \\ \dot{E}_h(t) &\geq -\left(\gamma + \gamma_1\right) E_h(t), \\ E_h(t) &\geq E_h(0) \exp\left(-\left(\gamma + \gamma_1\right) t\right) \geq 0, \\ E_h(t) &\geq 0, \quad \forall t \in \mathbb{R}^+. \end{aligned}$$

By considering the third equation of system (1), we have

$$\begin{aligned} \dot{I}_h &= \gamma E_h(t) - \theta I_h(t) - (\gamma_1 + \gamma_2) I_h(t), \\ \dot{I}_h &\geq (\theta + \gamma_1 + \gamma_2)\, I_h(t), \\ I_h(t) &\geq I_h(0) \exp\left(-(\theta + \gamma_1 + \gamma_2)\, t\right), \\ I_h(t) &\geq 0, \quad \forall t \in \mathbb{R}^+. \end{aligned}$$

Using the fourth equation of system (1), we get

$$\begin{aligned} \dot{R}_h(t) &= \theta I_h - \gamma_1 R_h, \\ \dot{R}_h(t) &\geq -\gamma_1 R_h(t), \\ R_h(t) &\geq R_h(0) \exp\left(-\gamma_1 t\right), \\ R_h(t) &\geq 0, \quad \forall\, t \in \mathbb{R}^+. \end{aligned}$$

After that, the fifth equation of system (1) gives

$$\begin{aligned} \dot{S}_v &= \mu_v N_v\big(1 + f(t)\big) - \alpha_{E_h} \frac{E_h S_v}{N_v} - \alpha_{I_h} \frac{I_h S_v}{N_v} - \mu S_v, \text{ this gives ,} \\ & \dot{S}_v \geq -\left(\alpha_{E_h} \frac{E_h}{N_h} + \alpha_{I_h} \frac{I_h}{N_h} + \mu\right) S_v(t), \\ & \dot{S}_v \geq -\left(\alpha_{E_h} \frac{||E_h||_\infty}{N_h} + \alpha_{I_h} \frac{||I_h||_\infty}{N_h} + \mu\right) S_v(t), \\ & S_v \geq S_v(0) \exp\left(-\left(\alpha_{E_h} \frac{||E_h||_\infty}{N_h} + \alpha_{I_h} \frac{||I_h||_\infty}{N_h} + \mu\right) t\right) \geq 0, \\ & S_v(t) \geq 0, \quad \forall\, t \in \mathbb{R}^+. \end{aligned}$$

Then, using the sixth equation of system (1), we have

$$\begin{aligned} \dot{I}_v &= \alpha_{E_h} \frac{E_h S_v}{N_v} + \alpha_{I_h} \frac{I_h S_v}{N_v} - \mu I_v, \\ &\geq -\mu I_v, \\ I_v(t) &\geq I_v(0) \exp(-\mu t) \geq 0, \\ I_v(t) &\geq 0, \quad \forall\, t \in \mathbb{R}^+. \end{aligned}$$

The system (1) is mathematically well defined, now we are interested by the biological definition. In this part, we prove the boundedness of solution of system (1). The world human population is finite let's note the $N_h(0)$ as well as that of mosquitoes and let's note $N_v(0)$. Let,

$$\Gamma = \left\{(S_h, E_h, I_h, R_h, S_v, I_v)\,;\; S_h + E_h + I_h + R_h \leq N_h(0);\; S_v + I_v \leq N_v(0)\right\}.$$

Proposition 2. *The compact set Γ is a positively invariant set, which attracts all positive orbits in $\mathbb{R}^6_+$. Moreover we have, all solutions of system (1) are bounded in Γ.*

Proof. Consider the total population of humans at time t

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t) \tag{9}$$

and vectors population

$$N_v(t) = S_v(t) + I_v(t). \tag{10}$$

The equations (9) and (10) give

$$\begin{aligned} \dot{N_h}(t) &= \dot{S_h}(t) + \dot{E_h}(t) + \dot{I_h}(t) + \dot{R_h}(t), \\ \dot{N_h}(t) &= \mu_h N_h(t) - \gamma_1 N_h(t) - \gamma_2 I_h(t) \end{aligned}$$

and

$$\begin{aligned}\dot{N}_v(t) &= \dot{S}_v(t) + \dot{I}_v(t),\\ \dot{N}_v(t) &= \mu_v(1+f(t))N_v(t) - \mu N_v(t).\end{aligned}$$

Considering the system (1), we obtain

$$\begin{aligned}\dot{N}_h(t) &= \dot{S}_h(t) + \dot{E}_h(t) + \dot{I}_h(t) + \dot{R}_h(t),\\ \dot{N}_h(t) &= \mu_h N_h(t) - \gamma_1 N_h(t),\\ N_h(t) &= N_h(0)\exp((\mu-\gamma_1)t) \leq N_h(0).\end{aligned}$$

and

$$\begin{aligned}\dot{N}_v(t) &= \dot{S}_v(t) + \dot{I}_v(t),\\ \dot{N}_v(t) &= \mu_v(1+f(t))N_v(t) - \mu N_v(t) \leq N_v(0).\end{aligned}$$

Therefore, any solution of (1) is bounded by zero and N_{max} where

$N_{max} = max\{N_v(0), N_h(0)\}$.

3.2. **Determination of basic reproduction number R_0.** R_0 is the number of humans infected by an infectious individual initially introduced into a totally susceptible population through female anopheles. The R_0 of our malaria model is given by the following proposition. The function f is continuous on [0;12], so we can define f^0 as follows, $f^0 = \min_{t\in[0;12]} f(t)$.

Proposition 3. *Considering model (1) we have the basic reproduction number*

$$R_0 = \sqrt{\frac{\mu_h\mu_v(1+f^0)\beta}{\gamma_1\mu^2(\gamma+\gamma_1)}\left(\alpha_{E_h} + \frac{\alpha_{I_h}\gamma}{\theta+\gamma_1+\gamma_2}\right)}. \tag{11}$$

Proof. The transition vector ([28]) $\mathcal{V}$ is

$$\mathcal{V} = \begin{pmatrix} (\gamma+\gamma_1)E_h \\ -\gamma E_h + (\theta+\gamma_1+\gamma_2)I_h \\ \mu I_v \end{pmatrix}$$

and the contact vector is defined by

$$\mathcal{F} = \begin{pmatrix} \frac{\beta I_v S_h}{N_h} \\ 0 \\ \alpha_{E_h}\frac{E_h S_v}{N_v} + \alpha_{I_h} I_h \frac{S_v}{N_v} \end{pmatrix}.$$

Then we get the matrix V by

$$V = \begin{pmatrix} \gamma+\gamma_1 & 0 & 0 \\ -\gamma & \gamma_1+\gamma_2+\theta & 0 \\ 0 & 0 & \mu \end{pmatrix} \quad \Leftrightarrow \quad V^{-1} = \begin{pmatrix} \frac{1}{\gamma+\gamma_1} & 0 & 0 \\ \frac{\gamma}{(\beta+\gamma_1)(\gamma+\gamma_1)} & \frac{1}{\theta+\gamma_1} & 0 \\ 0 & 0 & \frac{1}{\mu} \end{pmatrix} \tag{12}$$

and the matrix F by

$$F = \begin{pmatrix} 0 & 0 & \beta\dfrac{S_h^0}{N_h^0} \\ 0 & 0 & 0 \\ \alpha_{E_h}\dfrac{S_v^0}{N_v^0} & \alpha_{I_h}\dfrac{S_v^0}{N_v^0} & 0 \end{pmatrix}. \tag{13}$$

Which gives

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \beta\dfrac{S_h^0}{\mu N_h^0} \\ 0 & 0 & 0 \\ \alpha_{E_h}\dfrac{S_v^0}{(\gamma+\gamma_1)N_v^0} + \alpha_{I_h}\dfrac{\gamma S_v^0}{(\theta+\gamma_1+\gamma_2)(\gamma+\gamma_1)N_v^0} & 0 & 0 \end{pmatrix},$$

where $S_v^0 = \dfrac{\mu_v(1+f^0)N_v^0}{\mu}$ represents the susceptible vector population in DFE and $S_h^0 = \dfrac{\mu_h N_h^0}{\gamma_1}$ represents the susceptible human population in DFE. The terms N_h^0 and N_v^0 represent the total population of humans and mosquitoes respectively at DFE. Therefore, the basic reproduction number R_0 of system (1) is given by the spectral radius of the matrix $-FV^{-1}$ ([28]),

$$R_0 = \rho(-FV^{-1}) \quad \Leftrightarrow \quad R_0 = \sqrt{\frac{\mu_h\mu_v(1+f^0)\beta}{\gamma_1\mu^2(\gamma+\gamma_1)}\left(\alpha_{E_h} + \frac{\alpha_{I_h}\gamma}{\theta+\gamma_1+\gamma_2}\right)}. \tag{14}$$

We define R_e called the effective infection number as the average number of secondary infections caused by an infectious individual initially introduced into a totally susceptible population via female anopheles at time t. This secondary infection number R_e is given by ([17])

$$R_e(t) = \sqrt{\frac{\beta\mu_h\mu_v(1+f(t))}{\mu(\gamma+\gamma_1)\gamma_1\mu}\left(\alpha_{E_h} + \frac{\alpha_{I_h}\gamma}{\theta+\gamma_1}\right)}.$$

The evolution of the effective basic reproduction number (R_e) is given by the graph of Figure 3.

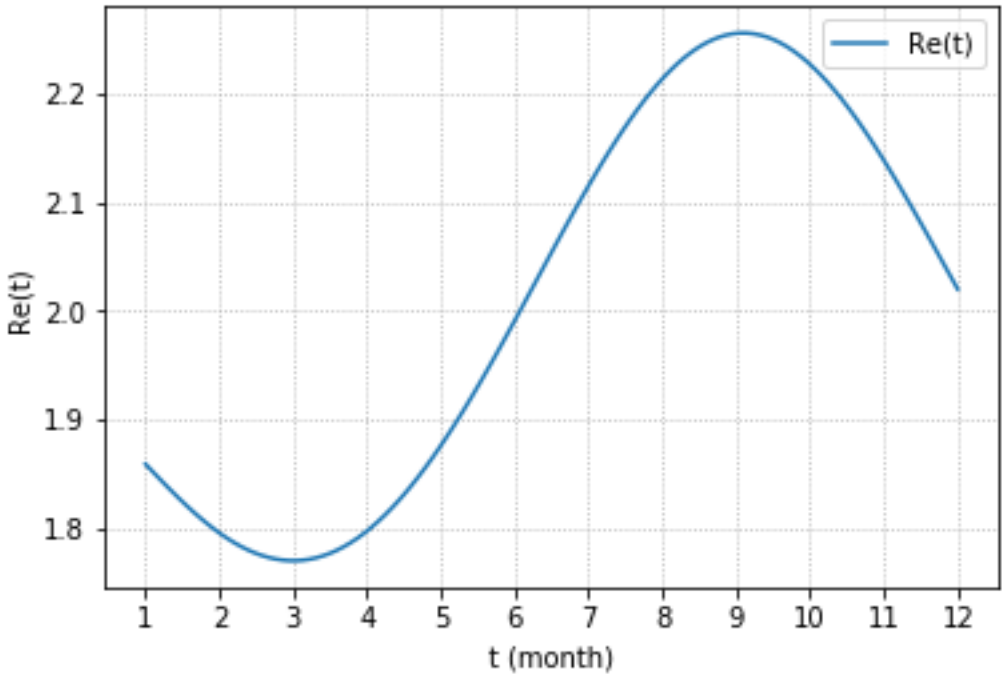

FIGURE 3. Evolution of the effective basic reproduction number R_e.

We notice that the infection rate progressively decreases from February to March due to the reduction in the number of infected individuals during this period. After the month of March, the infection rate ($R_e(t)$) increases and reaches its maximum in September due to the intensive presence of mosquitoes in that month, then decreases until December. That aligns with the data from Burkina Faso.

4. Stability of disease free equilibrium (DFE)

In this section, we prove the global stability of the DFE (Disease-Free Equilibrium point). In the DFE, we have

$$E_h = 0, \quad I_h = 0, \quad I_v = 0, \quad S_h^0 = \frac{\mu_h N_h^0}{\gamma_1} \text{ and } S_v^0 = \frac{\mu_v(1+f^0)N_v^0}{\mu}.$$

Theorem 4.1. *The DFE $E_0 = (S_h^0, 0, 0, 0, S_v^0, 0)$ of system (1) is globally asymptotically stable when $R_0 < 1$.*

Proof. Let consider the infected class I_h, I_v, E_h. The equations corresponding to these states, we have:

$$\begin{cases} \dot{E}_h = \beta \dfrac{I_v S_h}{N_h} - \gamma E_h - \gamma_1 E_h, \\ \dot{I}_h = \gamma E_h - \theta I_h - (\gamma_1 + \gamma_2) I_h, \\ \dot{I}_v = \alpha_{E_h} \dfrac{E_h S_v}{N_v} + \alpha_{I_h} \dfrac{I_h S_v}{N_v} - \mu I_v. \end{cases} \tag{15}$$

The matrix linearised M associated to the system (15) at X_0 is given by :

$$M = \begin{pmatrix} \gamma + \gamma_1 & 0 & \beta \\ -\gamma & \gamma_1 + \gamma_2 + \theta & 0 \\ \alpha_{E_h} & \alpha_{I_h} & \mu \end{pmatrix}.$$

$M = F + V$ where F is given in (13) and V is given in (12). Moreover $F > 0$ and V^{-1} given in (12) is defined positive. Thus $R_0 = \rho(FV^{-1}) < 1$ and from the Varga theorem [29], the matrix M is asymptotically stable when $R_0 < 1$. The eigenvalues of M has negative real part, by standard comparison theorem [9, 23] when $t \longrightarrow +\infty$ we have $(E_h, I_h, I_v) \longrightarrow (0, 0, 0)$. For system (1), substituting $E_h = 0$, $I_h = 0$, $I_v = 0$, we get $S_v \longrightarrow \dfrac{\mu_v(1+f^0)N_v^0}{\mu}$ and $S_h \longrightarrow \dfrac{\mu_h N_h^0}{\gamma_1}$ as $t \longrightarrow +\infty$. Thus $(S_h, E_h, I_h, S_v, I_v) \longrightarrow \left(\dfrac{\mu_h N_h^0}{\gamma_1}, 0, 0, 0, \dfrac{\mu_v(1+f^0)N_v^0}{\mu}, 0\right)$ as $t \longrightarrow +\infty$ for system (1), $R_0 < 1$. Therefore the DFE E_0 is globally asymptotically stable when $R_0 < 1$.

5. The endemic equilibrium point

In this section we determine and study the global stability of the endemic equilibrium point. In this part, we assume that f is constant (f^*) representing the maximum mosquito production in the endemic case, $f^* = \max_{t\in[0;12]} f(t)$. The terms $N_h^* = S_h^* + E_h^* + I_h^* + R_h^*$ and $N_v^* = S_v^* + I_v^*$ represent the total population of humans and mosquitoes respectively at endemic equilibrium.

$$\begin{cases} \mu_h N_h^* - \beta \dfrac{I_v^* S_h^*}{N_h^*} - \gamma_1 S_h^* = 0, \\ \beta \dfrac{I_v^* S_h^*}{N_h^*} - \gamma E_h^* - \gamma_1 E_h^* = 0, \\ \gamma E_h^* - \theta I_h^* - (\gamma_1 + \gamma_2) I_h^* = 0, \\ \theta I_h^* - \gamma_1 R_h^* = 0, \\ \mu_v N_v \big(1 + f^*\big) - \alpha_{E_h} \dfrac{E_h^* S_v^*}{N_v^*} - \alpha_{I_h} \dfrac{I_h^* S_v^*}{N_v^*} - \mu S_v^* = 0, \\ \alpha_{E_h} \dfrac{E_h^* S_v^*}{N_v^*} + \alpha_{I_h} \dfrac{I_h^* S_v^*}{N_v^*} - \mu I_v^* = 0. \end{cases}$$

This gives

$$S_h^* = \frac{\mu N_v^*}{\dfrac{\beta I_v^*}{N_v^*} + \gamma_1}, \qquad E_h^* = \frac{\mu\beta I_v^* N_h^*}{(\gamma+\gamma_1)(\beta I_v^* + \gamma_1 N_h^*)},$$

$$I_h^* = \frac{\gamma\mu\beta I_v^* N_h^*}{(\theta+\gamma_1+\gamma_2)(\gamma+\gamma_1)(\beta I_v^* + \gamma_1 N_h^*)}, \qquad S_v^* = \frac{\mu N_h^{*2}(1+f^*)}{\alpha_{E_h} E_h^* + \alpha_{I_h} I_h^* + \mu N_h^*},$$

$$R_h^* = \frac{\theta}{\gamma_1} \frac{\gamma\mu\beta I_v^* N_h^*}{(\theta+\gamma_1)(\gamma+\gamma_1)(\beta I_v^* + \gamma_1 N_h^*)},$$

where
$N_h^* = S_h^* + E_h^* + I_h^* + R_h^*$ and $N_v^* = S_v^* + I_v^*$.

Theorem 5.2. *If $R_0 > 1$, then the endemic equilibrium $X^* = (S_h^*, E_h^*, I_h^*, R_h^*, S_v^*, I_v^*)$ is globally asymptotically stable in*

$$\Gamma^* = \{X \in \Gamma; \quad X^* - X \geq 0\}.$$

Proof. Consider the following Lyapunov candidate function

$$V = N_h^* - N_h + N_v^* - N_v.$$

The function V is derivable. We have $V = 0$ at $X = X^*$ and positive in Γ^*.

$$\begin{aligned} \dot{V} &= -\dot{N}_h - \dot{N}_v, \\ &= -(\dot{S}_h + \dot{E}_h + \dot{I}_h + \dot{R}_h) - (\dot{S}_v + \dot{I}_v), \\ &= -(\mu_h N_h - \gamma_1 N_h) - \gamma_2 I_h - [\mu_v(1 + f(t))N_v - \mu N_v], \\ &= (\gamma_1 - \mu_h)N_h - \gamma_2 I_h - (\mu_v + \mu_v f(t) - \mu)N_v, \\ &\leq 0. \end{aligned}$$

Therefore $\dot{V} \leq 0$, then V is a Lyapunov function in Γ^*. Hence, LaSalle's invariant principle ([10]) implies that X^* is globally asymptotically stable.

6. Global sensitivity analysis for R_0

Sensitivity analysis helps us to identify the parameters that have a big impact on the disease transmission. Such an information is important not only for experimental design but also for data assimilation and reduction to complex nonlinear models ([21]). Normally, in the epidemiological model, the analysis is used to discover parameters that have greatest influence on the basic reproduction number R_0 and should be targeted by the control strategies.

Definition 6.1. *([3]) The normalized forward sensitivity index of reproduction number R_0, which depends differentiably on a parameter p, this is defined by*

$$\partial_p R_0 = \frac{\partial R_0}{\partial p} \times \frac{p}{R_0}. \tag{16}$$

Recall that the value of R_0 for the system (1) is given by (14).
This gives the table 2.

Table 2. Parameter description and elasticity value

Symbols	**Elasticity index**
γ	-0.47
γ_1	-0.46
γ_2	-0.014
μ	0.55
θ	-0.0086
μ_h	-0.55
μ_v	-0.45
α_{I_h}	0.3
β	0.5

This gives the Figure 4.

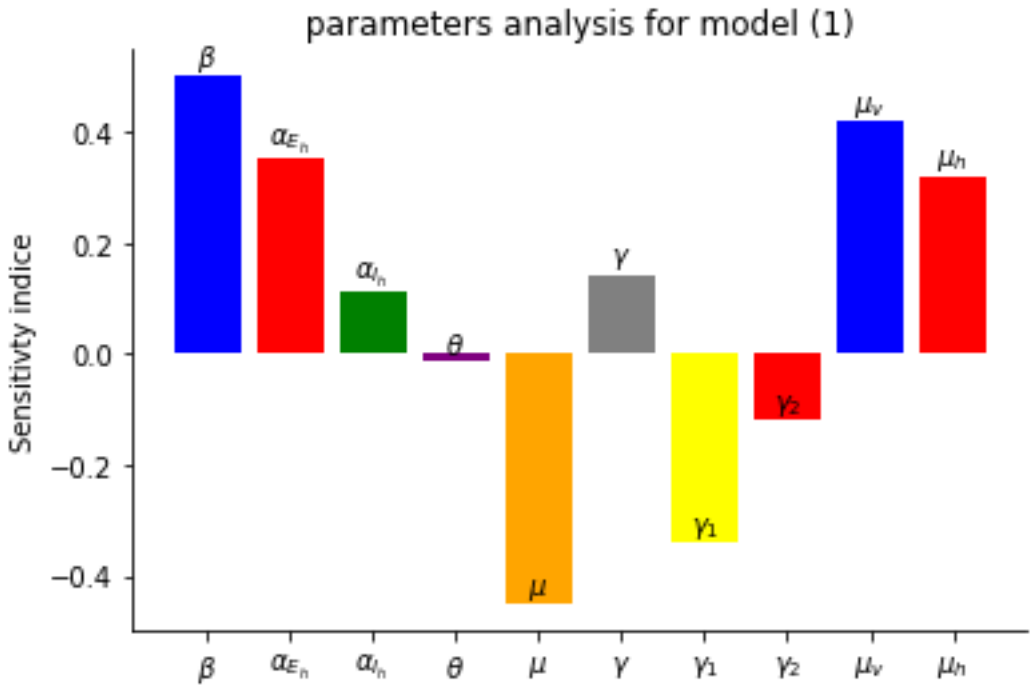

FIGURE 4. Parameters sensitivity of model (1)

The index values for the parameters in the model (1) are represented in a Table 2. Parameters with a positive sensitivity index indicate an increase in the transmission of malaria in the population for an increase in these values. On the other hand, parameters with a negative sensitivity index mean that an increase in these values leads to a decrease in the transmission of malaria in the population. The sensitivity index of μ in R_0 is -0.5. This implies that an increase 1% in the value of μ leads to a decrease 0.5% in the value of R_0. The sensitivity index of β in R_0 is 0.5. This implies that an increase 1% in the value of β leads to an increase 0.5% in the value of R_0. In the same way, the elasticity of γ_1 in R_0 is -0.34 means that the increase of 1% in the value of γ_1 implies the decrease of 0.34% in R_0. The sensitivity index of α_{E_h} and β are the same, that shows that these parameters have the same impact on the secondary infection rate R_0. The fact that $\partial_\gamma R_0 = -0.143$ means that 1% increase in value of γ will produce 0.143% decrease in R_0. Also The fact that $\partial_\theta R_0 = -0.00112$ means that 1% increase in value of θ will produce 0.00112% decrease in R_0. The fact that $\partial_{\alpha_{I_h}} R_0 = 0.00111$ means that 1% increase in α_{I_h} will produce 0.00111% increase in R_0.

7. Numerical simulation

In this section, we simulate the model (1) to illustrate our mathematical results. we use the same technique for [2]. To better understand the seasonal periodicity of mosquito evolution, we conducted numerical simulation in this section. Specifically, we applied system (1) to Burkina Faso. The literature [14] emphasizes that all mosquito traits relevant to transmission-biting, egg-to-adult survival and development, faculty-strongly respond to temperature and peak between 23^oC and 34^oC. This literature also shows that transmission of malaria by anopheles peaked at 26.4^oC and declined to zero below 16.2^oC and

above 31.6^oC. Based on these information, we can assume that the proliferation rate ($f(t)$) and transmission probability ($\alpha_{E_h}, \alpha_{I_h}$, β) are significantly affected by temperature and rainfall. Additionally, we can also note that the mosquito-biting depends on their evolution ($f(t)$). The more mosquitoes we have, the higher the biting rate and the probability of malaria transmission. In view of this we have decided to track the periodic evolution of mosquitoes through function f. Other authors steadied the tracking the periodic bites (see [26, 33]). The Table 3 shows the evolution of the malaria cases based on rainfall and temperature according to data from Burkina Faso. In this model, we are using data from Burkina Faso for the year 2020. This function f models how the mosquito population varies based on environmental and seasonal conditions that either facilitate or hinder their reproduction. Based on this information, we can define the 12-periodic function f as follows :

$$f(t) = 5 - 3\cos(\pi t/6). \tag{17}$$

The INSD (Institute of National Statistics and Development) data on temperatures and precipitation for Burkina Faso ([4]) are given in Table 3.

TABLE 3. Evolution of the number of malaria cases in the Burkina Faso, based on monthly average temperature (^oC) and rainfall (mm)

Months	Temperatures(^oC)	Rainfall(mm)	Malaria cases
January	25.8	0	5980
February	28.77	7	57910
March	31.11	32	47400
April	30.75	80	43489
May	29.54	140	38096
June	27.5	147	38441
July	25.95	210	56587
August	25.04	264	85014
September	25.57	224	100628
October	27.31	100	132447
November	28.33	25	108336
December	25.36	5	73839

The parameters value for model (1) are recorded in Table 4.

TABLE 4. The values of the parameters for the sismulation of model

Symbol	values	source
α_{E_h}	0.48	[3]
γ_1	0.008	[6]
γ_2	0.01	[12]
γ	0.304	[12]
μ	0.03	[25]
μ_h	0.4	[6]
μ_v	0.34	[25]
θ	0.083	[12]
β	0.48	[3]
α_{I_h}	0.22	[3]

In this part, the function f is not constant. The function f is 12 months periodic. The periodic function f allows us to track the annual seasonal evolution of mosquitoes. This seasonal tracking of mosquitoes enables us to identify the period of maximum mosquito production during the year, which in turn helps determine the period of maximum malaria infection in the human population. If this period is known, the government could implement preventive measures such as raising public awareness, destroying mosquito breeding sites, distributing mosquito nets to vulnerable populations, and administering preventive medications. Consider model (1) with the periodic function f and the Table (3).

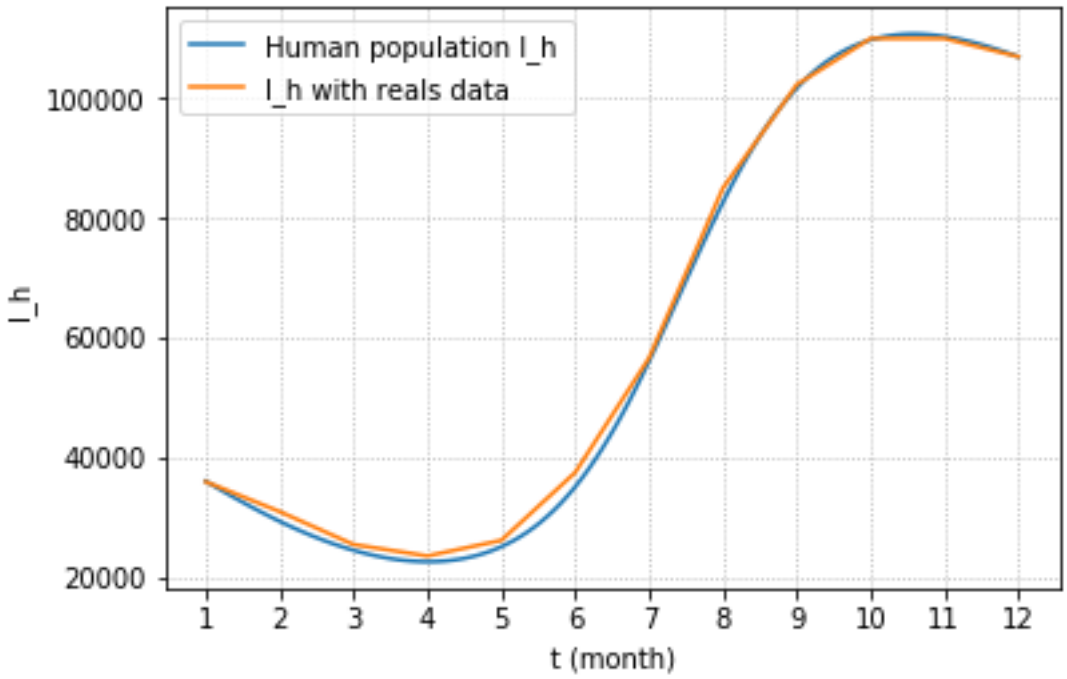

FIGURE 5. Evolution of infected individuals (I_h) from the model and the infected individuals (I_h) from data of Burkina Faso.

In Figure 5, the red curve represents the evolution of infections (I_h) with the actual data of Burkina Faso and the blue curve indicates the evolution of infections (I_h) according to the model. Allows for the observation of the malaria trend in Burkina Faso over a period of 12 months.

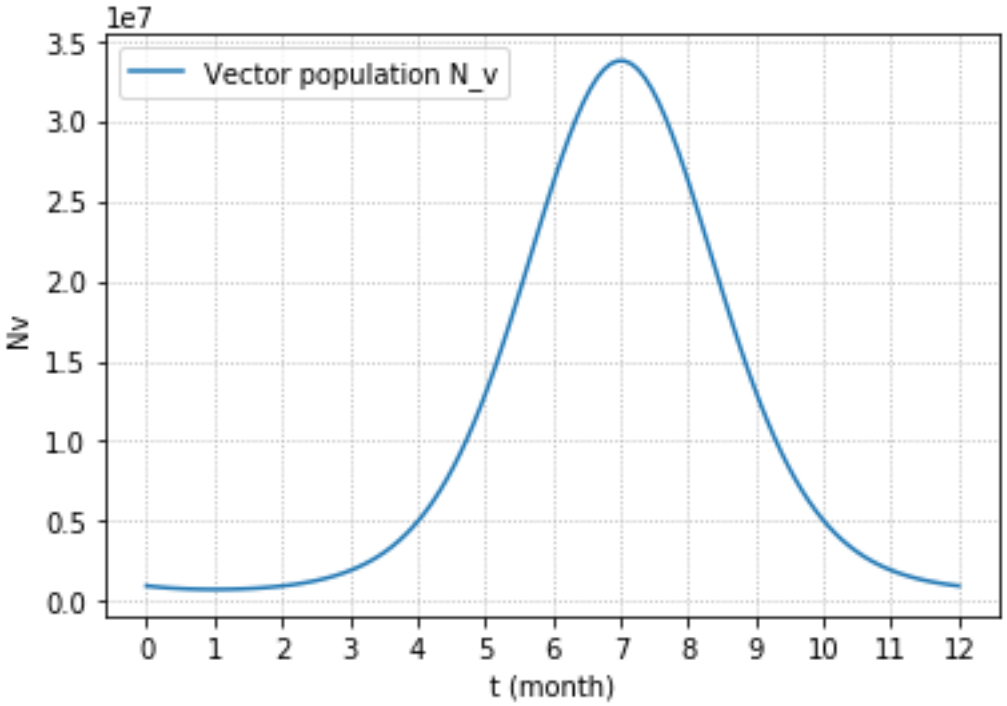

FIGURE 6. The seasonal evolution of the total number of mosquitoes (N_v).

Depending on the temperature and rainfall, the total number of mosquitoes (N_v) follows a seasonal pattern over 12 months and exhibits a peak in the month of July. The mosquito population decreases after the months of July due to a lack of rain and unexpected temperature fluctuations.

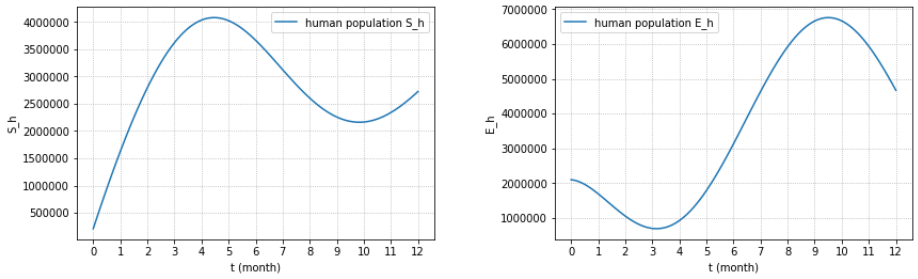

FIGURE 7. Seasonal evolution of susceptible (S_h) and exposed (E_h) persons with $R_0 > 1$.

In Figure 7, with these parameter values, we observe the seasonal evolution of the susceptible human population (S_h) and the exposed human population (E_h) in twelve months. The susceptible individuals curve shows growth and reaches its peak in March. The decrease in susceptible after the month of Mars is due to the increase in infected (I_h) and exposed (E_h) individuals. The minimum of the exposed population occurs in

February. This can be explained by the decrease in temperature and lack of rainfall. The peak of exposed individuals occurs in June and July, which can be attributed to the presence of rainfall and the stabilization of temperatures. The detection of periods of intense malaria exposed may depend on the data from the concerned regions. In this paper, we used data from Burkina Faso, but the model developed here can be applied in any other region if need be.

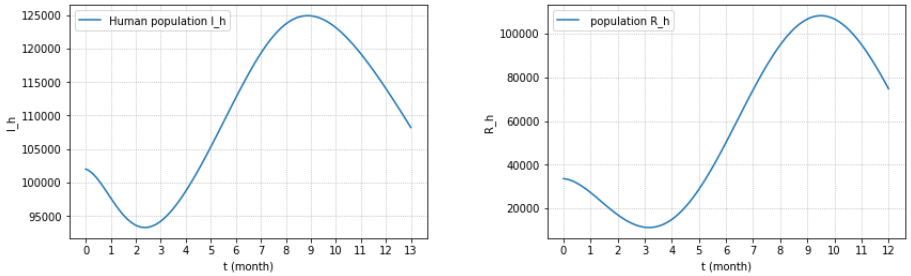

FIGURE 8. Seasonal evolution of infected (I_h) and recovered (R_h) persons with $R_0 > 1$.

In Figure 8, with these parameter values, we observe the evolution of the infected human population (I_h) and the recovered human population (R_h) on twelve (12) months. The graph of infected (I_h) cases in Figure 8 shows that in February, the number of malaria cases (I_h) is lower. This trend can be explained by the absence of infected mosquitoes (I_v) due to the drop in temperatures and lack of rainfall. Conversely, the number of malaria cases (I_h) significantly increases in September, which can be attributed to the massive presence of mosquitoes (I_v) that month due to rainfall. Therefore, we observe a minimum number of recoveries (R_h) in February and in September. The significant factors influencing this evolution are rainfall and temperature variations.

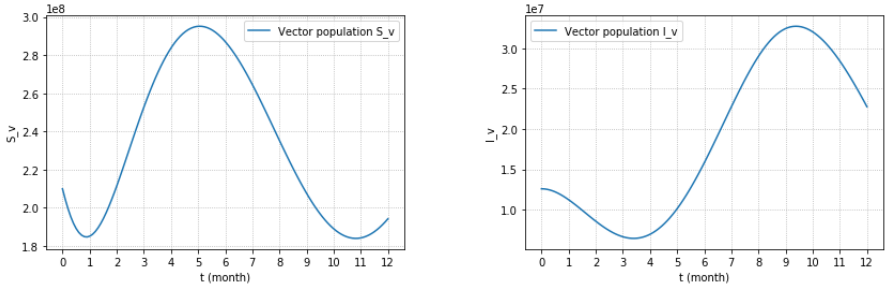

FIGURE 9. Seasonal evolution of susceptible and infected vectors with, $R_0 > 1$.

In Figure 9, with these parameter values, we observe the evolution of the susceptible vector population (S_v) and the infected vector population (I_v) for twelve months. Susceptible mosquitoes (S_v) decrease during the month of January, reaching their minimum number in February meanwhile, the number of infected vector (I_v) remains almost constant due to the transfer of (S_v) to (I_v). Moreover, every (S_v) that enters (I_v) stays in (I_v) until its death. In other words, no infected mosquito heals from the infection; instead, it remains infected until it dies. Following February, the population of susceptible mosquitoes increases and peaks in May. The peak of mosquitoes in May can be attributed to the presence of rainfall and favorable ambient temperatures for mosquito reproduction. This results in an increase in infected mosquitoes (I_h), reaching their maximum in

September due to the presence of malaria patients (I_h) and a wave of mosquitoes, the transition from (S_v) to (I_v) occurs massively in September. September and October are months during which the population should exercise caution and take measures to protect themselves from mosquito contact. The significant factors influencing this evolution of mosquitoes are rainfall and temperature variations. Therefore, malaria is a seasonal phenomenon. This determination of intense period of vector populations can assist the government in making decisions in the fight against malaria. The detection of periods of intense vector populations can depend on the data from the regions that is used for the study. In this paper, we use data from Burkina Faso.

8. Conclusion

In this paper, we have developed a SEIRS malaria model with dynamic recruitment and seasonal evolution of mosquitoes. The function f allowed us to determined the period of the high frequency of mosquitoes in order to allow the government to support the vulnerable population (distributing mosquito nets, spraying public areas and distributing preventive pharmaceutical products to children under the age of five years). We then showed the positivity and the boundedness of the periodic solutions. We have also shown the local and global stability of the equilibrium points of the model. In this study we have calculated the basic reproduction number R_0. The variation of the mosquitoes is measured by a periodic function f. In this study, we investigated the effect of the seasonal evolution of mosquitoes on the dynamics of malaria cases in Burkina Faso. According to data from Burkina Faso, the number of malaria cases intensifies from September to November, reaching its peak in October. In February, the number of malaria cases is lower, implying that the government should exert less effort during this month but concentrate more efforts during the months of September, October and November. The DFE stability shows that with great efforts we can expect equilibrium without malaria disease. Then the stability of this point is proven but relies on the commitment of the government to fight against malaria. The study of sensitivity of parameters shows that the most sensitive parameters of the model (1) are the mortality rate of mosquitoes (μ), the cure rate of malaria patients (θ) and the exposed rate of individuals (γ). The population's θ exposure rate decreases then the malaria cases decreases considerably until it stabilizes at zero cases. But if the cure rate γ decreases on $\gamma \in [0, 0.3[$ and the exposure rate (θ) increases on [0.5, 1] then the malaria case increases. Moreover, the disease may stabilize in the population. In order to fight against malaria, it is necessary to develop several strategies:

- Subsidize the access of malaria case in hospitals or take care of all malaria patients or take care of 90% of malaria patients. Otherwise, through the infected people who do not treat malaria, many anopheles became infected and then spread the malaria disease.
- To reduce the population's exposure to malaria through awareness raising, free distribution of impregnated mosquito nets, access to preventive care for children under five years of age, free access to anti-malarial treatment, malaria testing, etc. We can also in the framework of the fight against malaria take into account the mortality rate of mosquitoes, that is to say increase the mortality of mosquitoes by killing contaminated anopheles around the population (by using insecticides or other means).

Competing interests
The authors declare that they have no competing interests.

References

[1] F. Agusto, A. Gumel, and P. Parham. Qualitative assessment of the role of temperature variations on malaria transmission dynamics. *Journal of Biological Systems*, 23(04):1550030, 2015.

[2] J. L. Aron. Mathematical modelling of immunity to malaria. *Mathematical Biosciences*, 90(1-2):385–396, 1988.

[3] N. Chitnis, J. M. Hyman, and J. M. Cushing. Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bulletin of mathematical biology*, 70(5):1272–1296, 2008.

[4] G. de Burkina Faso. Insd. `https://www.insd.bf`, 2003-2021. Consulted Agust 11, 2023.

[5] M. Gambhir, M.-G. Basáñez, I. M. Blake, and N. C. Grassly. Modelling trachoma for control programmes. *Modelling Parasite Transmission and Control*, pages 141–156, 2010.

[6] J. Guay. Perspective monde-outil pédagogique des grandes tendances mondiales depuis 1945. *Sherbrooke University, Canada*, 2013.

[7] J. K. Hale. Verduyn lunel s m. introduction to functional differential equations. *Appl. Math. Sciences*, 99, 1993.

[8] M. B. Hoshen and A. P. Morse. A weather-driven model of malaria transmission. *Malaria journal*, 3(1):1–14, 2004.

[9] W. Jansen. V. lakshmikantham, s. leela, aa martynyuk: Stability analysis of nonlinear systems. marcel dekker inc., isbn: 0-8247-8067-1, 1995.

[10] J. P. La Salle. The stability of dynamical systems. *SIAM*, 1976.

[11] K. D. Lafferty. The ecology of climate change and infectious diseases. *Ecology*, 90(4):888–900, 2009.

[12] Y. Lou and X.-Q. Zhao. A climate-based malaria transmission model with structured vector population. *SIAM Journal on Applied Mathematics*, 70(6):2023–2044, 2010.

[13] W. Martens. Health impacts of climate change and ozone depletion: an ecoepidemiologic modeling approach. *Environmental health perspectives*, 106(1):241–251, 1998.

[14] E. A. Mordecai, J. M. Cohen, M. V. Evans, P. Gudapati, L. R. Johnson, C. A. Lippi, K. Miazgowicz, C. C. Murdock, J. R. Rohr, S. J. Ryan, et al. Detecting the impact of temperature on transmission of zika, dengue, and chikungunya using mechanistic models. *PLoS neglected tropical diseases*, 11(4):e0005568, 2017.

[15] C. N. Ngonghala, H. Enright, O. Prosper, and R. Zhao. Modeling the synergistic interplay between malaria dynamics and economic growth. *Mathematical Biosciences*, 372:109189, 2024.

[16] K. Okuneye and A. B. Gumel. Analysis of a temperature-and rainfall-dependent model for malaria transmission dynamics. *Mathematical biosciences*, 287:72–92, 2017.

[17] L. Ouattara, D. Ouedraogo, H. Ouedraogo, and A. Guiro. Stability analysis and optimal control of seirs mathematical model. *Discussiones Mathematicae: Differential Inclusions, Control & Optimization*, 43, 2023.

[18] K. P. Paaijmans, S. S. Imbahale, M. B. Thomas, and W. Takken. Relevant microclimate for determining the development rate of malaria mosquitoes and possible implications of climate change. *Malaria journal*, 9(1):1–8, 2010.

[19] P. E. Parham and E. Michael. Modeling the effects of weather and climate change on malaria transmission. *Environmental health perspectives*, 118(5):620–626, 2010.

[20] P. E. Parham, J. Waldock, G. K. Christophides, D. Hemming, F. Agusto, K. J. Evans, N. Fefferman, H. Gaff, A. Gumel, S. LaDeau, et al. Climate, environmental and socio-economic change: weighing up the balance in vector-borne disease transmission. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 370(1665):20130551, 2015.

[21] H. S. Rodrigues, M. T. T. Monteiro, and D. F. Torres. Sensitivity analysis in a dengue epidemiological model. In *Conference papers in science*, volume 2013. Hindawi, 2013.

[22] J. R. Rohr, A. P. Dobson, P. T. Johnson, A. M. Kilpatrick, S. H. Paull, T. R. Raffel, D. Ruiz-Moreno, and M. B. Thomas. Frontiers in climate change–disease research. *Trends in ecology & evolution*, 26(6):270–277, 2011.

[23] H. Smith. P. waltman. the theory of the chemostat, 1995.

[24] W. Tabachnick. Challenges in predicting climate and environmental effects on vector-borne disease episystems in a changing world. *Journal of Experimental Biology*, 213(6):946–954, 2010.

[25] B. Traoré, M. Barro, B. Sangaré, and S. Traoré. A temperature-dependent mathematical model of malaria transmission with stage-structured mosquito population dynamics. *Nonautonomous Dynamical Systems*, 8(1):267–296, 2021.
[26] S. B. Traore Bakary and T. Sado. A mathematical model of malaria transmission in a periodic environment. *Journal of Biological Dynamics*, 2018.
[27] N. K. Vaidya, X. Li, and F.-B. Wang. Impact of spatially heterogeneous temperature on the dynamics of dengue epidemics. *Discrete and Continuous Dynamical Systems-B*, 24(1):321, 2019.
[28] P. Van den Driessche and J. Watmough. Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical biosciences*, 180(1-2):29–48, 2002.
[29] R. S. Varga. Matrix iterative analysis. prentice hall, englewood cliffs. *New Jersey*, 1962.
[30] WHO. Global malaria programme: world malaria report. `https://www.who.int/mediacentre/news/releases/2015/sage/fr/`, 2015. Consulted August 11, 2023.
[31] H. Yang, M. Macoris, K. Galvani, M. Andrighetti, and D. Wanderley. Assessing the effects of temperature on dengue transmission. *Epidemiology & Infection*, 137(8):1179–1187, 2009.
[32] Y. Yoda, H. Ouedraogo, D. Ouedraogo, and A. Guiro. Mathematical analysis and optimal control of dengue fever epidemic model. *Advances in Continuous and Discrete Models*, 2024(1):11, 2024.
[33] M. Zhu, T. Feng, Y. Xu, and J. Cao. Global dynamics of a dengue fever model incorporating transmission seasonality. *Nonlinear Analysis: Modelling and Control*, 28:1–24, 2023.

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