LETTERS IN BIOMATHEMATICS
An International Journal

Intercollegiate Biomathematics Alliance

RESEARCH ARTICLE

OPEN ACCESS

A Mathematical model of Malaria transmission dynamics with general incidence function and maturation delay in a periodic environment

Bakary Traoré, Ousmane Koutou, Boureima Sangaré

UFR/ST, Department of Mathematics, University Nazi BONI, Burkina Faso

ABSTRACT

In this paper, we investigate a mathematical model of malaria transmission dynamics with maturation delay of a vector population in a periodic environment. The incidence rate between vector and human hosts is modeled by a general nonlinear incidence function which satisfies a set of conditions. Thus, the model is formulated as a system of retarded functional differential equations. Furthermore, through dynamical systems theory, we rigorously analyze the global behavior of the model. Therefore, we prove that the basic reproduction number of the model denoted by $\mathcal{R}_0$ is the threshold between the uniform persistence and the extinction of malaria virus transmission. More precisely, we show that if $\mathcal{R}_0$ is less than unity, then the disease-free periodic solution is globally asymptotically stable. Otherwise, the system exhibits at least one positive periodic solution if $\mathcal{R}_0$ is greater than unity. Finally, we perform some numerical simulations to illustrate our mathematical results and to analyze the impact of the delay on the disease transmission.

ARTICLE HISTORY
Received November 25, 2018
Accepted February 23, 2020

KEYWORDS
Malaria transmission, delay differential equations, basic reproduction number, numerical simulations, periodic environment, general incidence function

1 Introduction

The spread of infectious diseases has always been a big concern and poses a threat to public health, as well as to the economic and social developments of the human society. Thus, its prevention and control become extremely important (Agusto, 2014; Anderson and May, 1982; Burnett and White, 1974). Quantitative studies of disease transmission mechanisms provide a foundation for such prevention and control, and the fundamental aim of epidemic dynamics is to investigate the transmission dynamics of infectious diseases. Hence, mathematical models based on the progressions of diseases are formulated, to analyze the origins of the diseases, the factors involved in their transmissions and to predict their prevalence and their patterns. Further, qualitative and quantitative studies, and sensitivity analysis of model parameters can help us make more realistic simulations and reliable transmission predictions which may not be feasible by experiments or field studies (Ma and Li, 2009). Thus, epidemiological models have been recognized as valuable tools in analyzing the spread and the control of infectious diseases. The standard technique for developing mathematical descriptions of diseases is to model the system as a set of ordinary differential equations (Bai and Zhou, 2012; Koutou et al., 2018a; Traoré et al., 2020). The earliest ordinary differential equations epidemic models were proposed by Kermack and McKendrick (1927). This is an immensely powerful approach which has led to many insights into the factors that affect disease prevalence and control.

In the study of epidemiological models, incidence rate plays an important role (Alexander and Moghadas, 2014; Roop-O et al., 2015). The incidence rate is the infection rate of susceptible individuals through their contacts with infectious individuals. The number of individuals contacted by an infective per unit of time is called a contact rate of the infection, denoted by $\mu(N)$. It depends on the population size N in a given environment (Burnett and White, 1974; Ma and Li, 2009). The function $p\mu(N)$ describes the force of infection of the infectious individuals and p represents the probability of infection per contact. This function usually depends on the toxicity of the virus or on the situation of the environment. Since diseases are transmitted to susceptible individuals (S) by contact with infective (I), then the incidence of the disease is given by $p\mu(N)SI/N$ where $p\mu(N)S/N$ is the infection rate and S/N is the fraction of susceptible in the population, (Anderson and May, 1982; Bai and Zhou, 2012). In the literature, there exist different types of incidence rate among which we quote: the bilinear incidence or simple mass action

CONTACT Boureima Sangaré mazou1979@yahoo.fr

incidence, denoted by $qpSI$ with $\mu(N) = qN$ and the standard incidence, denoted by $qpSI/N$ with $\mu(N) = q$ (Korobeinikov, 2006). In 1982, Anderson and May showed that the standard incidence is more suitable than the bilinear form for diseases transmitted in human populations. In 1978, Capasso and Serio used saturated incidence of the form $\beta SI/(1 + \kappa I)$. Further, in 1986 Liu et al. proposed nonlinear incidences of the form of $\beta SI^n/(1 + \kappa I^n)$. Some years later, the general form $(\beta h(I)S^n/N)$ of this incidence was introduced by certain authors. Due to the importance of the incidence rate in the mathematical modeling of infectious diseases, its choice is very important in the qualitative description of the diseases transmission. Hence, to avoid the use of a single incidence function, the use of a general incidence rate including a family of particular functions with similar properties has become a topic of interest for several authors (Alexander and Moghadas, 2014; Posny and Wang, 2014; Roop-O et al., 2015; Traoré et al., 2019; Traoré et al., 2020).

Malaria is a potentially deadly disease caused by protozoan parasites known as *Plasmodium* that infect and replicate within human blood cells. It is the most prevalent infectious disease in the world. Malaria parasites spread between humans via the bite of the infectious female adult *Anopheles*. There are five (previously four) *Plasmodium* species that have been reported to cause significant numbers of malaria infections in humans. Concerning the mathematical modeling for the spread of malaria transmission, significant breakthroughs have been made in recent years since the first model introduced by Ronald Ross in 1911 and Georges Macdonald in 1957. Since then, there has been a great deal of work about using mathematical models to study malaria transmission (Agusto, 2014; Chitnis et al., 2008; Chiyaka et al., 2008; Traoré et al., 2018). Nowadays, two major factors arouse scientist's interest. The first one is the life cycle of *Anopheles* in the dynamics of malaria transmission (Ai et al., 2011; Lutambi et al., 2013; Traoré et al., 2018) and the second one is the impact of climate on this life cycle (Lou and Zhao, 2010; Okuneye and Gumel, 2017; Wang and Zhao, 2018; Koutou et al., 2018b). Malaria is one of the diseases that exhibits seasonal fluctuation. Indeed, environmental and climatic factors play an important role in the geographical distribution and transmission of malaria. For example, in temperate climates and in tropical highlands, temperature restricts vector multiplication and the development of the parasite in the mosquito, while in arid climates, precipitations restrict mosquito breeding (Okuneye and Gumel, 2017; Traoré et al., 2017; Wang and Zhao, 2018; Traoré et al., 2020). Moreover, the population biology of the *Anopheles* vectors is crucial to understanding many aspects of the disease, as well as assessing control strategies and projecting future outcomes. Malaria models that do not incorporate the dynamics of the juvenile stages of the mosquito are known to give results that do not generally match with observed epidemiology.

As we have mentioned above, the incidence function is considered to play a vital role in ensuring that the model can give a reasonable qualitative description of the disease dynamics. Hence, we note that about malaria modeling, many models with different kinds of incidences have been proposed in order to analyze the dynamical properties of the disease. For instance, in one of their investigations, Lou and Zhao (2010) used a standard incidence rate to formulate a seasonal mosquito stage structure malaria model by including a delay in the immature mosquitoes maturation, and they proved that the introduction of the time delay has significant effect on the disease transmission. Further, using bilinear incidence, Wang et al. (2011) investigated a model of malaria transmission in a periodic environment and they showed that the existence of chaos in the periodic model may cause the disease to approach the uncontrollable state due to unpredictability. Olaniyi and Obabiyi (2013) introduced a new model of malaria transmission by using a saturated incidence. Thus, the use of different types of incidence function to model malaria transmission attracts many authors. However, since many forms of incidence functions are used to model the transmission of malaria, what type of incidence is more suitable and should be chosen when we investigate malaria transmission? That is a real challenge because its choice depends on many factors such as toxicity of parasite, situation of the environment and so on (Ma and Li, 2009; Hu and Sun, 2011). In view of these factors, it is very difficult to estimate the incidence rate. To contribute to the answer of the question, in this paper, we extend the work of Lou and Zhao (2010); Li et al. (2017) by using a general incidence function that includes a family of particular functions with similar properties to describe interaction between human hosts and vector in the periodic environment. The general incidence function used, contains all those existing in the literature of malaria modeling including that used by Lou and Zhao. Through mathematical analysis, we derive the epidemic threshold parameter $\mathcal{R}_0$, for predicting disease persistence or extinction in periodic environment. Further, by using Floquet theory (Tiana and Wang, 2015; Zhao, 2003; Traoré et al., 2020), we show that the global stability of the disease-free periodic equilibrium and the uniform persistence of the disease are strongly linked to the basic reproduction number.

The rest of the paper is organized as follows. In Section 2, we formulate the mathematical model. In Section 3, firstly, we introduce some basic results. Then, we compute the basic reproduction number $\mathcal{R}_0$ of the model in the periodic environment and we deduce the basic reproduction number $\hat{\mathcal{R}}_0$ of the associated autonomous model. The focus of Section 4 is on global behavior of our model. Numerical simulations are provided in Section 5 in order to illustrate our theoretical results. We conclude in Section 6.

2 Mathematical model formulation

Malaria parasites are transmitted to human hosts through the bites of infectious female *Anopheles* mosquitoes (Koutou et al., 2018a,b; Traoré et al., 2020). Thus, in the model we consider two groups of populations: human population and mosquito population. The human population is divided into four epidemiological categories representing the state variables: susceptible humans denoted by $S_h(t)$, exposed humans denoted by $E_h(t)$, infectious humans denoted by $I_h(t)$ and immune humans (immune and asymptomatic, but slightly infectious) denoted by $R_h(t)$. The mosquito population is divided into two sub-groups: the juvenile mosquitoes and the adult mosquitoes. The adult mosquitoes are divided into three classes: susceptible mosquitoes denoted by $S_v(t)$, exposed mosquitoes denoted by $E_v(t)$ and infectious mosquitoes denoted by $I_v(t)$. The mosquito population does not include an immune class as mosquitoes never recover from infection because their infective period ends with their death due to their relatively short life-cycle. At any time, the total size of the human population and the mature mosquitoes population are respectively given by:

$$N_h(t) = S_h(t) + E_h(t) + I_h(t) + R_h(t),$$
$$N_v(t) = S_v(t) + E_v(t) + I_v(t).$$

If an infectious mosquito bites a susceptible human, then the human progresses through the exposed, infectious and immune classes before moving to the susceptible class if he loses his immunity. Similarly, when a susceptible mosquito bites an infectious or immune human, it moves through the exposed and infectious classes. The immature mosquitoes are divided into three classes: egg, larva and pupa. In each class we assume that we have the same development rate with periodic death rate $d_i(t)$, where $d_i(t)$ is determined by the climate profile. The juvenile mosquitoes maturation rate at time t, produced by female mosquitoes at time $t-\tau$ is given by

$$B\big(t-\tau, N_v(t-\tau)\big)\eta(t), \quad \text{with} \quad \eta(t) = \exp\left(-\int_{t-\tau}^{t} d_i(s)ds\right),$$

where τ is the average maturation period, and $B(t, N_v(t))$ the egg reproduction function.

Indeed, in the biological literature, there exists three types of time ω-periodic reproduction functions:

(i) $B_1(t,N) = \dfrac{p(t)N}{q(t)+N^n}$ with $p(t) > 0, q(t) > 0$ and $n > 0$.

(ii) $B_2(t,N) = a(t) + i(t)N$ with $a(t) \geq 0$ and $i(t) > 0$.

(iii) $B_3(t,N) = j(t)Ne^{-s(t)N}$ with $j(t) > 0$ and $s(t) > 0$.

Functions B_1 with $n = 1$ and B_3 are used in fisheries and are known respectively as the Ricker function and the Beverton-Holt function. In function B_2, quantity $a(t)$ represents a periodic immigration rate and $i(t)N$ is a periodic birth term.

Moreover, in this general model, the infection rates per susceptible mosquito and per susceptible human are respectively given by functions $g\big(t, I_h(t), R_h(t)\big)$ and $f\big(t, I_v(t)\big)$. We assume throughout this paper that:

(H1) : all vector population measures refer to densities of female mosquitoes,

(H2) : the mosquitoes bite only humans,

(H3) : all the new recruit humans and vectors are susceptible.

From the above assumptions, we obtain the diagram seen in Figure 1 where,

- Λ_h is the constant recruitment rate for humans,
- d_h is the natural death rate for humans,
- d_p is the disease-induced death rate for humans,
- α_h is the transfer rate of humans from exposed class to infectious class,
- r_h is the recovery rate of humans,
- γ is the per capita rate of loss of immunity for humans,
- α_v is the transfer rate of mosquitoes from exposed class to infectious class,
- τ is the maturation period of immature mosquitoes.

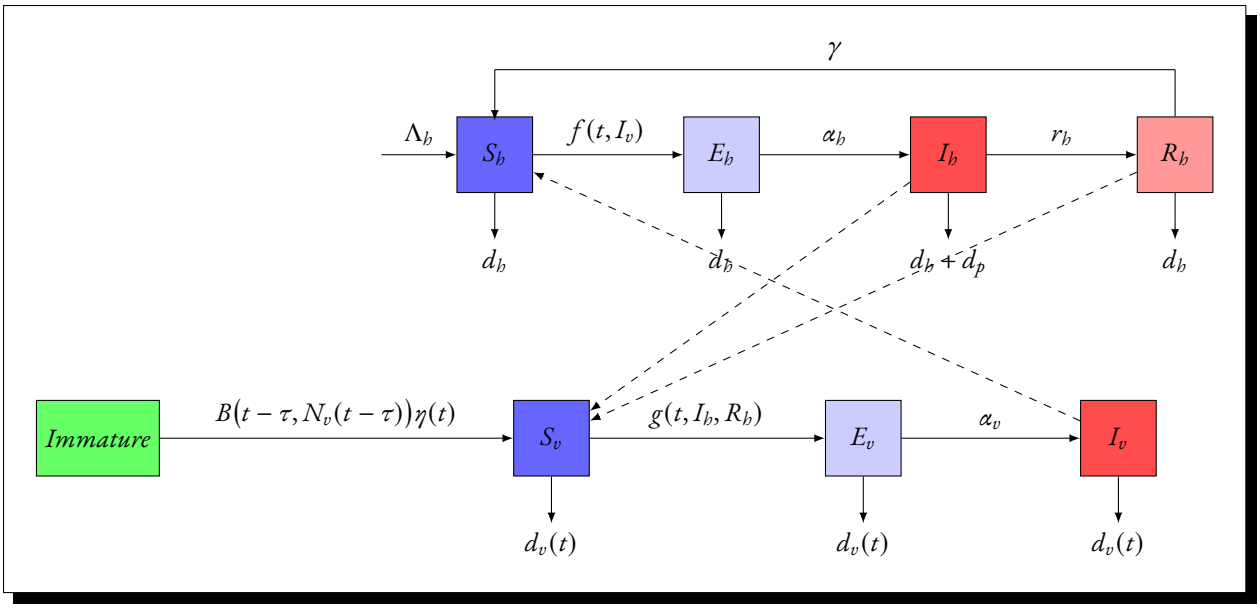


Figure 1: Transfer diagram: The dashed arrows indicate the direction of the infection and the solid arrows represent the transition from one class to another.

Furthermore, the number of individuals which survives from one class to the next (see Figure 1), is given by the following differential equations:

$$\begin{cases} \dot{S}_h(t) = \Lambda_h + \gamma R_h(t) - d_h S_h(t) - f\big(t, I_v(t)\big)S_h(t), \\ \dot{E}_h(t) = f(t, I_v(t))S_h(t) - (d_h + \alpha_h)E_h(t), \\ \dot{I}_h(t) = \alpha_h E_h(t) - (d_h + d_p + r_h)I_h(t), \\ \dot{R}_h(t) = r_h I_h(t) - (d_h + \gamma)R_h(t), \\ \dot{S}_v(t) = B\big(t - \tau, N_v(t - \tau)\big)\eta(t) - d_v(t)S_v(t) - g\big(t, I_h(t), R_h(t)\big)S_v(t), \\ \dot{E}_v(t) = g\big(t, I_h(t), R_h(t)\big)S_v(t) - (\alpha_v + d_v(t))E_v(t), \\ \dot{I}_v(t) = \alpha_v E_v(t) - d_v(t)I_v(t). \end{cases} \tag{1}$$

Moreover, at any time t, we have:

$$\dot{N}_h(t) = \Lambda_h - d_h N_h(t) - d_p I_h(t), \tag{2}$$

$$\dot{N}_v(t) = B\big(t - \tau, N_v(t - \tau)\big)\eta(t) - d_v(t)N_v(t). \tag{3}$$

3 Preliminaries and threshold dynamics

3.1 Preliminaries

Let C be the Banach space of continuous functions $\phi : [-\tau, 0] \to \mathbb{R}_+^7$ equipped with norm

$$\|\phi\| = \sup_{\vartheta \in [-\tau, 0]} |\phi(\vartheta)|,$$

and $\phi = (\phi_1, \phi_2, \phi_3, \phi_4, \phi_5, \phi_6, \phi_7)$ the initial function belongs to the space C. Then, system (1) can be written as follows:

$$\dot{x}(t) = K(t, x), \tag{4}$$

with

$$x(\vartheta) = \big(S_h(\vartheta), E_h(\vartheta), I_h(\vartheta), R_h(\vartheta), S_v(\vartheta), E_v(\vartheta), I_v(\vartheta)\big) \in C.$$

Theorem 3.1 (Lou and Zhao, 2009). *Equation (3) admits a positive ω-periodic solution $N_v^*(t)$ which is globally asymptotically stable in $C \setminus \{0\}$.*

In the particular case where $B(t-\tau, N_v(t-\tau)) = k > 0$, $d_i(t) = d_i > 0$ and $d_v(t) = d_v > 0$, then equation (3) reduces to an autonomous ordinary differential equation. The dynamics of the mosquito population is then governed by the following equation:

$$\dot{N}_v(t) = ke^{-\tau d_i} - d_v N_v(t). \tag{5}$$

It is obvious that equation (5) admits a positive solution

$$N_v^*(t) = \frac{ke^{-\tau d_i}}{d_v} + \left(N_v(0) + \frac{ke^{-\tau d_i}}{d_v}\right)e^{-d_v t}. \tag{6}$$

Moreover, it is clear that $N_v^*(t) \longrightarrow N_v^* = \dfrac{ke^{-\tau d_i}}{d_v}$ as $t \to +\infty$.

In order to simplify the notations, we introduce the following new functions:

$$f_i^v = \frac{\partial f}{\partial I_v}, \quad f_i^h = \frac{\partial f}{\partial I_h}, \quad f_r^h = \frac{\partial f}{\partial R_h}, \quad f_{ii}^v = \frac{\partial^2 f}{\partial I_v^2}, \quad g_i^v = \frac{\partial g}{\partial I_v},$$

$$g_i^h = \frac{\partial g}{\partial I_h}, \quad g_r^h = \frac{\partial g}{\partial R_h}, \quad g_{rr}^h = \frac{\partial^2 g}{\partial R_h^2}, \quad g_{ri}^h = \frac{\partial^2 g}{\partial R_h \partial I_h}, \quad g_{ir}^h = \frac{\partial^2 g}{\partial I_h \partial R_h}.$$

Furthermore, we assume that functions f and g satisfy the following assumptions:

(H4): f and g are assumed to be differentiable and periodic in time with a common positive period ω. That means that,

$$g\big(t+\omega, I_h(t), R_h(t)\big) = g\big(t, I_h(t), R_h(t)\big) \quad \text{and} \quad f\big(t+\omega, I_v(t)\big) = f\big(t, I_v(t)\big).$$

(H5): $f\big(t, I_v\big) \geq 0$ and $g\big(t, I_h, R_h\big) \geq 0$.

(H6): $f\big(t, 0\big) = 0 = g\big(t, 0, 0\big)$.

(H7): $f_i^v(t, I_v) \geq 0, g_i^h(t, I_h, R_h) \geq 0$ and $g_r^h(t, I_h, R_h) \geq 0$.

(H8): $f(t, I_v)$ and $g(t, I_h, R_h)$ are both concave for any $t \geq 0$; namely

$$\begin{pmatrix} g_{ii}^h & g_{ir}^h \\ g_{ri}^h & g_{rr}^h \end{pmatrix}$$

is negative semidefinite everywhere and f_{ii}^v is negative.

(H9): $f_i^h(t, 0) = 0 = f_r^h(t, 0)$ and $g_i^v(t, 0, 0) = 0$.

(H10): There exists a positive number $\hat{N}_v$ such as

$$B\big(t-\tau, N_v\big)\eta(t) - d_v(t)N_v < 0, \forall\, N_v \geq \hat{N}_v.$$

The conditions in assumption **(H5)** state that the infection occurs when there is contact between infectious mosquitoes and susceptible humans and also between susceptible mosquitoes and infectious humans. Assumption **(H7)** states that the rate of new infection increases with both the infected humans population size and the infected mosquitoes population size; and assumption **(H9)** ensures that there is no direct transmission of malaria in both populations. Condition **(H8)** is a common assumption in epidemic model, based on saturation effect.

Remark 3.1.

- The standard incidence function is of the form

$$f(t, I_v)S_h = \beta_1(t)\frac{I_v}{N_h}S_h \quad \text{and} \quad g(t, I_h, R_h)S_v = \beta_2(t)\frac{I_h}{N_h}S_v + \beta_3(t)\frac{R_h}{N_h}S_v \tag{7}$$

and has been used by Lou and Zhao (2010) and Roop-O et al. (2015).

- The saturation incidence function is of the form

$$f(t,I_v)S_h = \beta_1(t)\frac{I_v}{1+\mu_v I_v}S_h \quad \text{and} \quad g(t,I_h,R_h)S_v = \beta_2(t)\frac{I_h}{1+\mu_h I_h}S_v + \beta_3(t)\frac{R_h}{1+\mu_h I_h}S_v \tag{8}$$

has been used by Roop-O et al. (2015) and Olaniyi and Obabiyi (2013),

where β_i, $i = 1,2,3$ represent different rates of contact that lead to an infection. μ_h and μ_v determine the level at which the force of infection saturates.

Let us consider the following linear ordinary differential system

$$\dot{Q}(t) = \mathcal{B}(t)Q(t), \tag{9}$$

where $\mathcal{B}(t)$ is a continuous, cooperative, irreducible and ω-periodic $n\times n$ matrix function and $\Phi_{\mathcal{B}}(t)$ is the fundamental solution matrix of (9). Let $\rho\big(\Phi_{\mathcal{B}}(\omega)\big)$ be the spectral radius of the matrix $\Phi_{\mathcal{B}}(\omega)$. By the Perron-Frobenius theorem (see Smith, 1995, Theorem A.3), $\rho\big(\Phi_{\mathcal{B}}(\omega)\big)$ is the principal eigenvalue of $\Phi_{\mathcal{B}}(\omega)$ which is associated to a positive eigenvector. Hence, the following result is useful for our subsequent comparison arguments.

Lemma 3.1 (Bai and Zhou, 2012). *Let* $r = \frac{1}{\omega}\ln\rho(\Phi_{F-V(.)}(\omega))$, *then there exists a positive* ω*-periodic function* $v(t)$ *such that* $e^{rt}v(t)$ *is a solution of equation* (9).

3.2 Threshold dynamics

Now, we introduce the basic reproduction number for model (1) according to the theory developed in 2008 by Wang and Zhao, which is a generalization of the work in (Van den Driessche and Watmough, 2002) to the periodic case. Assumption **(H6)** and Theorem 3.1, yield that system (1) admits a unique disease-free periodic equilibrium

$$\mathcal{E}_t = \big(N_h^*, 0, 0, 0, N_v^*(t), 0, 0\big),$$

with

$$N_h^* = \frac{\Lambda_h}{d_h} = S_h^* \quad \text{and} \quad N_v^*(t) = S_v^*(t).$$

Hence, linearizing system (1) at the disease-free periodic state $\mathcal{E}_t$, we obtain the following system:

$$\begin{cases} \dot{E}_h(t) = S_h^* I_v(t) f_i^v(t,0) - (d_h+\alpha_h)E_h(t), \\ \dot{I}_h(t) = \alpha_h E_h(t) - (d_h+d_p+r_h)I_h(t), \\ \dot{R}_h(t) = r_h I_h(t) - (d_h+\gamma)R_h(t), \\ \dot{E}_v(t) = S_v^*(t)g_i^h(t,0,0)I_v(t) + S_v^*(t)g_r^h(t,0,0)R_h(t) - (\alpha_v + d_v(t))E_v(t), \\ \dot{I}_v(t) = \alpha_v E_v(t) - d_v(t)I_v(t). \end{cases}$$

This system can be written as follows:

$$\dot{z}(t) = \big(F(t) - V(t)\big)z(t), \tag{10}$$

where

$$z(t) = \big(E_h(t), I_h(t), R_h(t), E_v(t), I_v(t)\big)^T,$$

$$F(t) = \begin{pmatrix} 0 & 0 & 0 & 0 & S_h^* f_i^v(t,0) \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & S_v^*(t)g_i^h(t,0,0) & S_v^*(t)g_r^h(t,0,0) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

and

$$V(t) = \begin{pmatrix} d_h+\alpha_h & 0 & 0 & 0 & 0 \\ -\alpha_h & d_h+d_p+r_h & 0 & 0 & 0 \\ 0 & -r_h & d_h+\gamma & 0 & 0 \\ 0 & 0 & 0 & \alpha_v+d_v(t) & 0 \\ 0 & 0 & 0 & -\alpha_v & d_v(t) \end{pmatrix}.$$

Let $Y(t,s),\ t \geq s$ be the matrix solution of the linear ω-periodic system

$$\begin{aligned} \dot{Y}(t,s) &= -V(t)Y(t,s),\ \forall t \geq s, \\ Y(s,s) &= I_5, \end{aligned} \tag{11}$$

where I_5 is the 5×5 identity matrix and C_ω is the ordered Banach space of all ω-periodic functions from $\mathbb{R}$ to $\mathbb{R}^5$ which is equipped with the maximum norm $\|\cdot\|$. Suppose $\varphi(s) \in C_\omega$ is the initial distribution of infectious individuals in this periodic environment. Then $F(s)\varphi(s)$ is the rate of new infections produced by the infected individuals who were introduced at time s, and $Y(t,s)F(s)\varphi(s)$ represents the distribution of those infected individuals who were newly infected at time s and remain in the infected compartments at time t for $t \geq s$. Hence,

$$\psi(t) = \int_0^\infty Y(t, t-\sigma)F(t-\sigma)\varphi(t-\sigma)d\sigma, \quad \sigma \in [0, +\infty),$$

is the distribution of accumulative new infections at time t produced by all those infected individuals $\varphi(s)$ introduced at the previous time (Burnett and White, 1974; Capasso and Serio, 1978; Chitnis et al., 2008; Koutou et al., 2018a).

Thus, we define the next infection operator $\mathcal{L} : C_\omega \longrightarrow C_\omega$ by

$$(\mathcal{L}\varphi)(t) = \int_0^\infty Y(t, t-\sigma)F(t-\sigma)\varphi(t-\sigma)d\sigma, \quad \forall\, t \in \mathbb{R},\ \varphi \in C_\omega.$$

So, the basic reproduction number is $\mathcal{R}_0 = \rho(\mathcal{L})$, the spectral radius of $\mathcal{L}$.

In order to calculate $\mathcal{R}_0$, we consider the following linear ω-periodic system:

$$\dot{w}(t) = \left[\frac{1}{\lambda}F(t) - V(t)\right]w(t), \quad \forall\, t \in \mathbb{R}_+,\ \lambda \in (0, \infty). \tag{12}$$

Let $W(t,s,\lambda),\ t \geq s,\ s \in \mathbb{R}$, be the evolution operator of system (12) on $\mathbb{R}^5$. Clearly $W(t,0,1) = \Phi_{F-V}(t),\ \forall\, t \geq 0$. The following result will be used in our numerical calculation of the basic reproduction number.

Lemma 3.2 (Wang and Zhao, 2008)**.**

(i) If $\rho(W(\omega, 0, \lambda)) = 1$ has a positive solution λ_0, then λ_0 is an eigenvalue of $\mathcal{L}$, and hence $\mathcal{R}_0 > 0$.

(ii) If $\mathcal{R}_0 > 0$, then $\lambda = \mathcal{R}_0$ is the unique solution of $\rho(W(\omega, 0, \lambda)) = 1$.

(iii) $\mathcal{R}_0 = 0$ if and only if $\rho(W(\omega, 0, \lambda)) < 1$, for all $\lambda > 0$.

Remark 3.2. In the case where model (1) is reduced to an autonomous system, the basic reproduction number can be computed as follows:

$$\hat{\mathcal{R}}_0^{\,2} = \frac{\alpha_h \alpha_v S_h^*}{Q}\left(r_h g_r^h(0,0) + (d_h + \gamma)g_i^h(0,0)\right)S_v^* f_i^v(0). \tag{13}$$

with

$$Q = d_v(\alpha_v + d_v)(d_h + \gamma)(d_h + d_p + r_h)(d_h + \alpha_h).$$

Let $S_v^* = \dfrac{ke^{-\tau d_i}}{d_v}$.

- Using the standard incidence function given in (7), we obtain

$$\hat{\mathcal{R}}_{0,1}^{\,2} = \frac{\alpha_h \alpha_v}{Q}\left(\frac{r_h \beta_3}{S_h^*} + \frac{(d_h + \gamma)\beta_2}{S_h^*}\right)\beta_1 k e^{-\tau d_i} \tag{14}$$

  with

$$Q = d_v^2(\alpha_v + d_v)(d_h + \gamma)(d_h + d_p + r_h)(d_h + \alpha_h).$$

- Using the saturation incidence function given in (8), we obtain

$$\hat{\mathcal{R}}_{0,2}^{\,2} = \frac{\alpha_h \alpha_v S_h^*}{Q}\left(r_h \beta_3 + (d_h + \gamma)\beta_2\right)\beta_1 k e^{-\tau d_i}. \tag{15}$$

  with

$$Q = d_v^2(\alpha_v + d_v)(d_h + \gamma)(d_h + d_p + r_h)(d_h + \alpha_h).$$

From the above results, one can see that setting all the parameters of the model (1) it is clear that $\hat{\mathcal{R}}_{0,1}$ is always less than $\hat{\mathcal{R}}_{0,2}$. Hence, the choice of incidence function is crucial when determining the transmission dynamics of malaria. Moreover, we note that the larger τ is, the smaller the basic reproduction number becomes (Chiyaka et al., 2008; Tian and Song, 2017). Besides, it is obvious that incidence functions defined in (7) and (8) satisfy assumptions **(H4)–(H9)**.

4 Global behavior of the model

4.1 Positivity and boundedness of solutions

Lemma 4.1 (Ouedraogo et al., 2018, 2019). *Let **(HI0)** hold. For any $\phi \in C$, system (1) has a unique nonnegative solution. Moreover, all the solutions are ultimately and uniformly bounded.*

Proof. For any $\phi \in C$, the function

$$K(t,\phi) = \begin{pmatrix} \Lambda_h + \gamma\phi_4(0) - d_h\phi_1(0) - f\big(t,\phi_7(0)\big)\phi_1(0) \\ f(t,\phi_7(0))\phi_1(0) - (d_h+\alpha_h)\phi_2(0) \\ \alpha_h\phi_2(0) - (d_h+d_p+r_h)\phi_3(0) \\ r_h\phi_3(0) - (d_h+\gamma)\phi_4(0) \\ B\Big(t-\tau, \sum_{i=5}^{7}\phi_i(-\tau)\Big)\eta(t) - d_v(t)\phi_5(0) - g\big(t,\phi_2(0),\phi_4(0)\big)\phi_5(0) \\ g\big(t,\phi_2(0),\phi_4(0)\big)\phi_5(0) - (\alpha_v + d_v(t))\phi_6(0) \\ \alpha_v\phi_6(0) - d_v(t)\phi_7(0), \end{pmatrix}$$

is continuous and Lipschitzian in ϕ in each compact set in $\mathbb{R}\times C$. Hence, by fundamental theory of functional differential equations (Hale, 1977; Ouedraogo et al., 2018, 2019; Koutou et al., 2018a,b), system (1) admits a unique solution. Therefore, note that if ϕ is nonnegative and $\phi_i(0) = 0$, for $i = 1,2,\dots,7$ then $K(t,\phi)$ is nonnegative. So, thanks to Remark 5.2.1 in (Smith, 1995), the set C is positively invariant.

From equations (2) and (3), we have:

$$\begin{aligned} \dot{N}_h(t) &\le \Lambda_h - d_h N_h(t), \\ \dot{N}_v(t) &\le B\big(t-\tau, N_v(t-\tau)\big)\eta(t) - d_v(t)N_v(t). \end{aligned}$$

Thus, using the standard comparison theorem, it yields that

$$\limsup_{t\to\infty} N_h(t) \le \limsup_{t\to\infty}\left[\frac{\Lambda_h}{d_h} + \left(N_h(0) - \frac{\Lambda_h}{d_h}\right)e^{-d_h t}\right] = N_h^*$$

and

$$\limsup_{t\to\infty}\big(N_v(t) - N_v^*(t)\big) \le 0.$$

It yields that, all the solutions are ultimately bounded.

Moreover, if $N_h(t) > N_h^*$ and $N_v(t) > \dot{N}_v$, then $\dot{N}_h(t) < 0$ and $\dot{N}_v(t) < 0$. It then follows that all the solutions are uniformly bounded and that completes the proof. □

4.2 Extinction of disease

In this section, we investigate the global stability of the disease-free periodic equilibrium, $\mathcal{E}_t$ which also provides a condition for the extinction of the disease. We give the following results, which will be used in the proof of our main results.

Lemma 4.2 (Wang and Zhao, 2008). *The following statements are valid:*

(i) $\mathcal{R}_0 = 1$ *if and only if* $\rho(\Phi_{F-V}(\omega)) = 1$.

(ii) $\mathcal{R}_0 < 1$ *if and only if* $\rho(\Phi_{F-V}(\omega)) < 1$.

(iii) $\mathcal{R}_0 > 1$ *if and only if* $\rho(\Phi_{F-V}(\omega)) > 1$.

Hence, the disease-free periodic equilibrium $\mathcal{E}_t$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$, where $\Phi_{F-V}(t)$ is the monodromy matrix of linear ω-periodic system (12).

Theorem 4.1. *If $\mathcal{R}_0 < 1$ and $d_p = 0$, then the disease-free periodic equilibrium, $\mathcal{E}_t$ is globally asymptotically stable.*

Proof. If $d_p = 0$, then we have,

$$\begin{aligned}\dot{N}_h(t) &= \Lambda_h - d_h N_h(t),\\ \dot{N}_v(t) &= B\big(t-\tau, N_v(t-\tau)\big)\eta(t) - d_v(t)N_v(t).\end{aligned}$$

Thus, there exists a period $\omega_1(\varepsilon)$ such that $\forall t \geq \omega_1(\varepsilon)$,

$$N_h(t) \leq S_h^* + \varepsilon \quad \text{and} \quad N_v(t) \leq S_v^*(t) + \varepsilon, \quad \forall\, \varepsilon > 0.$$

Since f and g are differentiable, then, from assumptions (**H6**) and (**H8**), and using the Taylor-Young formula, it yields that

$$\begin{aligned}S_h(t)f\big(t, I_v(t)\big) &\leq (S_h^* + \varepsilon)I_v(t)f_i^v(t,0)\\ S_v(t)g\big(t, I_h(t), R_h(t)\big) &\leq (S_v^*(t) + \varepsilon)\Big(I_h(t)g_i^h(t,0,0) + R_h(t)g_r^h(t,0,0)\Big).\end{aligned}$$

Thus, from model (1) we have

$$\begin{aligned}\dot{E}_h(t) &\leq (S_h^* + \varepsilon)f_i^v(t,0)I_v(t) - (d_h + \alpha_h)E_h(t),\\ \dot{E}_v(t) &\leq (S_v^*(t) + \varepsilon)g_i^h(t,0,0)I_v(t) + (S_v^*(t) + \varepsilon)g_r^h(t,0,0)R_h(t) - (\alpha_v + d_v(t))E_v(t).\end{aligned}$$

We obtain the following system:

$$\begin{pmatrix}\dot{E}_h(t)\\ \dot{I}_h(t)\\ \dot{R}_h(t)\\ \dot{E}_v(t)\\ \dot{I}_v(t)\end{pmatrix} \leq \Big(F_\varepsilon(t) - V(t)\Big)\begin{pmatrix}E_h(t)\\ I_h(t)\\ R_h(t)\\ E_v(t)\\ I_v(t)\end{pmatrix}, \tag{16}$$

with

$$F_\varepsilon(t) = \begin{pmatrix}0 & 0 & 0 & 0 & (S_h^* + \varepsilon)f_i^v(t,0)\\ 0 & 0 & 0 & 0 & 0\\ 0 & 0 & 0 & 0 & 0\\ 0 & (S_v^*(t) + \varepsilon)g_i^h(t,0,0) & (S_v^*(t) + \varepsilon)g_r^h(t,0,0) & 0 & 0\\ 0 & 0 & 0 & 0 & 0\end{pmatrix}.$$

Now, let us consider the following auxiliary system:

$$\dot{\tilde{z}}(t) = \Big(F_\varepsilon(t) - V(t)\Big)\tilde{z}(t), \tag{17}$$

where

$$\tilde{z}(t) = \big(\tilde{E}_h(t), \tilde{I}_h(t), \tilde{R}_h(t), \tilde{E}_v(t), \tilde{I}_v(t)\big)^T.$$

If $\mathcal{R}_0 < 1$, then, from Lemma 4.2, $\rho(\Phi_{F-V}(\omega)) < 1$. Moreover, by the continuity of the spectral radius, we have

$$\lim_{\varepsilon \to 0^+} \rho\big(\Phi_{F_\varepsilon - V}(\omega)\big) = \rho\big(\Phi_{F-V}(\omega)\big) < 1.$$

Thus, there exists $\varepsilon^* > 0$ such that $\rho\big(\Phi_{F_\varepsilon - V}(\omega)\big) < 1$, $\forall \varepsilon \in [0, \varepsilon^*]$. Further, Lemma 3.1 implies that there exists a positive ω-periodic function $v(t)$ such that $\tilde{z}(t) = e^{rt}v(t)$ is a solution of (17), with $r = \frac{1}{\omega}\ln\rho(\Phi_{F_\varepsilon - V}(\omega))$. Since $\rho\big(\Phi_{F_\varepsilon - V}(\omega)\big) < 1$, then, $r < 0$ and $\tilde{z}(t) \to 0$ as $t \to \infty$. Then, by using the comparison theorem (Lakshmikantham et al., 1989), it yields that

$$\lim_{t\to\infty}\big(E_h(t), I_h(t), R_h(t), E_v(t), I_v(t)\big) = (0,0,0,0,0).$$

Hence, by the first equation of system (1) we get $\lim_{t\to\infty} S_h(t) = S_h^*$.

Moreover, we have

$$\lim_{t\to\infty}(S_v(t) - N_v^*(t)) = \lim_{t\to\infty}(N_v(t) - E_v(t) - I_v(t) - N_v^*(t)) = 0.$$

It then follows that $\mathcal{E}_t$ is globally attractive if $\mathcal{R}_0 < 1$. This completes the proof. □

4.3 Disease persistence

Here, we study the behavior of model (1) when the basic reproduction number, $\mathcal{R}_0$ is greater than unity.

Let us consider the following sets:

$$\begin{aligned} X &:= C\big([-\tau, 0], \mathbb{R}_+^7\big), \\ X_0 &:= \big\{\phi \in X : \phi_i(0) > 0,\ \forall i \in \{2, 3, 4, 6, 7\}\big\}, \\ \partial X_0 &:= X \setminus X_0. \end{aligned}$$

Let $u(t, \phi)$ be the unique solution of (1) such that $u(0, \phi) = \phi$ the periodic semiflow generated by periodic system (1) and $P\colon X \to X$ the Poincaré map associated with system (1), namely:

$$\begin{aligned} P(\phi) &= \Phi(\omega)\phi = u(\omega, \phi), \quad \forall\, \phi \in X. \\ P^n(\phi) &= \Phi(n\omega)\phi = u(n\omega, \phi), \quad \forall\, n \geq 0. \end{aligned}$$

We see that both X and X_0 are positively invariant with respect to model (1), and ∂X_0 is a relatively closed set in X. Moreover, P is point dissipative from Lemma 4.1 and P^{n_0} is compact whenever $n_0\omega > \tau$. Thus, following Theorem 2.4 in (Magal and Zhao, 2006), P admits a global attractor in X.

Lemma 4.3. *If $\mathcal{R}_0 > 1$, there exists $\xi > 0$ such that when $\|\phi - \mathcal{E}\| \leq \xi$, for any $\phi \in X_0$, one has $\limsup_{k\to\infty} d\big(P^k(\phi), \mathcal{E}\big) \geq \xi$ where $\mathcal{E} = \big(N_h^*, 0, 0, 0, N_v^*(\vartheta), 0, 0\big)$ for all $\vartheta \in [-\tau, 0]$.*

Proof. Suppose by contradiction that

$$\limsup_{k\to\infty} d\big(P^k(\phi), \mathcal{E}\big) < \xi \quad \text{for some} \quad \phi \in X_0. \tag{18}$$

Then, there exists an integer $k_1 \geq 1$ such that for all $k \geq k_1$, $d\big(P^k(\phi), \mathcal{E}\big) < \xi$. By the continuity of solutions with respect to initial values, if $\|\phi - \mathcal{E}\| \leq \xi$, then

$$\Big\|u\big(t, P^k(\phi)\big) - u\big(t, \mathcal{E}\big)\Big\| < \varepsilon_1, \quad \text{for all} \quad t \in [0, \omega] \quad \text{and} \quad \varepsilon_1 > 0. \tag{19}$$

Let $t = k\omega + t'$, where $t' \in [0, \omega]$ and $k = [\frac{t}{\omega}]$. $[\frac{t}{\omega}]$ is the greatest integer less than or equal to $\frac{t}{\omega}$. Hence, we have

$$\big\|u\big(t, \phi\big) - u\big(t, \mathcal{E}\big)\big\| = \big\|u\big(t' + k\omega, \phi\big) - u\big(t' + k\omega, \mathcal{E}\big)\big\| = \Big\|u\big(t', P^k(\phi)\big) - u\big(t', \mathcal{E}\big)\Big\| < \varepsilon.$$

It then follows that

$$S_h(t) \geq S_h^* - \varepsilon_1,\ S_v(t) \geq S_v^*(t) - \varepsilon_1,\ 0 \leq I_v(t) \leq \varepsilon_1,\ 0 \leq I_h(t) \leq \varepsilon_1,\ 0 \leq R_h(t) \leq \varepsilon_1.$$

Moreover, assuming that f and g are concave, then we have

$$\begin{aligned} S_h(t) f\big(t, I_v(t)\big) &\geq \big(S_h^* - \varepsilon_1\big)\Big(f(t, 0) + I_v(t) f_i^v(t, 0) + \frac{1}{2}\varepsilon_1 I_v(t) f_{ii}^v(t, 0)\Big) \\ S_v(t) g\big(t, I_h(t), R_h(t)\big) &\geq \big(S_v^*(t) - \varepsilon_1\big)\Big(g(t, 0, 0) + I_h(t) g_i^h(t, 0, 0) + R_h(t) g_r^h(t, 0, 0) \\ &\qquad + \frac{1}{2}\varepsilon_1 I_h(t) g_{ii}^h(t, 0, 0) - \varepsilon_1 I_h(t)\big|g_{ir}^h(t, 0, 0)\big| + \frac{1}{2}\varepsilon_1 R_h(t) g_{rr}^h(t, 0, 0)\Big). \end{aligned}$$

Further, we obtain the following system:

$$\begin{pmatrix} \dot{E}_h(t) \\ \dot{I}_h(t) \\ \dot{R}_h(t) \\ \dot{E}_v(t) \\ \dot{I}_v(t) \end{pmatrix} \geq \Big(F_{\varepsilon_1}(t) - V(t)\Big) \begin{pmatrix} E_h(t) \\ I_h(t) \\ R_h(t) \\ E_v(t) \\ I_v(t) \end{pmatrix}, \tag{20}$$

with

$$F_{\varepsilon_1}(t) = \begin{pmatrix} 0 & 0 & 0 & 0 & (S_h^* - \varepsilon_1)\tilde{G}_1(t,0) \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & (S_v^*(t) - \varepsilon_1)\tilde{G}_2(t,0,0) & (S_v^*(t) - \varepsilon_1)\tilde{G}_3(t,0,0) & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix},$$

where

$$\begin{aligned} \tilde{G}_1(t,0) &= f_i^v(t,0) + \tfrac{1}{2}\varepsilon_1 f_{ii}^v(t,0), \\ \tilde{G}_2(t,0,0) &= g_i^h(t,0,0) + \tfrac{1}{2}\varepsilon_1 g_{ii}^h(t,0,0) - \varepsilon_1\left|g_{ir}^h(t,0,0)\right|, \\ \tilde{G}_3(t,0,0) &= g_r^h(t,0,0) + \tfrac{1}{2}\varepsilon_1 g_{rr}^h(t,0,0). \end{aligned}$$

Let us consider again the following auxiliary system:

$$\dot{\tilde{z}}(t) = \left(F_{\varepsilon_1}(t) - V(t)\right)\tilde{z}(t), \tag{21}$$

where

$$\tilde{z}(t) = \left(\tilde{E}_h(t), \tilde{I}_h(t), \tilde{R}_h(t), \tilde{E}_v(t), \tilde{I}_v(t)\right)^T.$$

If $\mathcal{R}_0 > 1$, once again, Lemma 4.2 implies that $\rho(\Phi_{F-V}(\omega)) > 1$. By the continuity of the spectral radius, we have

$$\lim_{\varepsilon_1 \to 0^+} \rho\left(\Phi_{F_{\varepsilon_1}-V}(\omega)\right) = \rho\left(\Phi_{F-V}(\omega)\right) > 1.$$

Thus, there exists $\varepsilon_1^* > 0$ such that $\rho\left(\Phi_{F_{\varepsilon_1}-V}(\omega)\right) > 1$, $\forall \varepsilon_1 \in [0, \varepsilon_1^*]$. Hence, from Lemma 3.1, there exists a positive ω-periodic function $v(t)$ such that $\tilde{z}(t) = e^{rt}v(t)$ is a solution of (21), with $r = \dfrac{1}{\omega}\ln\rho(\Phi_{F_{\varepsilon_1}-V}(\omega))$. Since $\rho(\Phi_{F_{\varepsilon_1}-V}(\omega)) > 1$, then $r > 0$ and $\lim\limits_{t\to\infty} \tilde{z}(t) = \infty$. It then follows from the comparison principle that

$$\lim_{t\to\infty} \left|\left(E_h(t), I_h(t), R_h(t), E_v(t), I_v(t)\right)\right| = \infty$$

which is in contradiction with (18). □

Theorem 4.2. *If $\mathcal{R}_0 > 1$, there exists $\delta > 0$ such that any solution of system (1) with initial condition, $\phi \in X_0$ satisfies*

$$\begin{aligned} &\liminf_{t\to\infty} S_h(t) \geq \delta, \quad \liminf_{t\to\infty} E_h(t) \geq \delta, \quad \liminf_{t\to\infty} I_h(t) \geq \delta, \quad \liminf_{t\to\infty} R_h(t) \geq \delta, \\ &\liminf_{t\to\infty} S_v(t) \geq \delta, \quad \liminf_{t\to\infty} E_v(t) \geq \delta, \quad \liminf_{t\to\infty} I_v(t) \geq \delta, \end{aligned}$$

and system (1) admits at least one positive periodic solution.

Proof. Let us define the set

$$M_\partial := \left\{\phi \in \partial X_0 : P^n(\phi) \in \partial X_0, \forall n \geq 0\right\}.$$

We claim that

$$M_\partial = \left\{\phi \in X : \phi_i(0) = 0,\ \forall i \in \{2,3,4,6,7\}\right\}. \tag{22}$$

If

$$\left(\phi_1(0), \phi_2(0), \phi_3(0), \phi_4(0), \phi_5(0), \phi_6(0), \phi_7(0)\right) = \left(\phi_1(0), 0, 0, 0, \phi_5(0), 0, 0\right)$$

with $\phi_1(0) > 0$ and $\phi_5(0) > 0$ then,

$$(S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t), I_v(t)) \equiv (S_h(t), 0, 0, 0, S_v(t), 0, 0)$$

with $S_h(t) > 0$ and $S_v(t) > 0$. So, $M_\partial \supseteq \left\{\phi \in X : \phi_i(0) = 0,\ \forall i \in \{2,3,4,6,7\}\right\}$.

Now, we show that $M_\partial \subseteq \left\{\phi \in X : \phi_i(0) = 0,\ \forall i \in \{2,3,4,6,7\}\right\}$. That is, for all $n \geq 0$ and $\phi \in \partial X_0$, we have:

$$E_h(n\omega)I_h(n\omega)R_h(n\omega)E_v(n\omega)I_v(n\omega) = 0.$$

Suppose that there exists an integer n_1 such that,

$$\big(E_h(n_1\omega), I_h(n_1\omega), R_h(n_1\omega)\big), E_v(n_1\omega), I_v(n_1\omega)\big)^T > 0.$$

Then, solving the equations of system (1), we derive that

$$\big(S_h(t), E_h(t), I_h(t), R_h(t), S_v(t), E_v(t), I_v(t)\big) \in X_0, \quad \forall\, t \geq n_1\omega,$$

which contradicts that $\phi \in \partial X_0$. That requires $P^n(\phi) \in \partial X_0$, $\forall\, n \geq 0$. Hence, claim (22) holds.

Furthermore, claim (22) implies that $\mathcal{E}$ is the only fixed point of P and acyclic in ∂X_0. In addition, Lemma 4.3 implies that $\mathcal{E}$ is an isolate invariant set in X and $W^s(\mathcal{E}) \cap X_0 = \emptyset$, where $W^s(\mathcal{E}) = \big\{x \in X : \limsup_{n\to\infty} d\big(P^n(x), \mathcal{E}\big) = 0\big\}$ is the stable set of $\mathcal{E}$. By the acyclicity theorem on uniform persistence for maps (see Zhao, 2003, Theorem 1.3.1 and Remark 1.3.1), it then follows that P is uniformly persistent with respect to $(X_0, \partial X_0)$. So, the periodic semiflow $\Phi(t)$ is also uniformly persistent with respect to X_0. Hence system (1) is uniformly persistent.

Furthermore, Theorem 3.1 in (Magal and Zhao, 2006) implies that system (1) has at least one ω-periodic solution $u(t, \phi^*)$ with $\phi^* \in X_0$ for all $t \geq 0$.

Now, let us prove that $S_h^*(0)$ and $S_v^*(0)$ are positive. If $S_h^*(0) = S_v^*(0) = 0$ then, we obtain that $S_h^*(t) > 0, S_v^*(t) > 0$ for all $t > 0$. But using the periodicity of solution, we have $S_h(0) = S_h(n\omega) = 0$ and $S_v(0) = S_v(n\omega) = 0$, that is a contradiction. □

5 Numerical simulations

In this section, we perform some numerical simulations to support our theoretical analysis given in Sections 3 and 4. To illustrate our results, we use the standard incidence rate as in (Traoré et al., 2018; Traoré et al., 2020).

$$f\big(t, I_v(t)\big) = a\beta(t)\frac{I_v(t)}{N_h(t)}$$
$$g\big(t, I_h(t), R_h(t)\big) = \beta(t)\frac{bI_h(t) + cR_h(t)}{N_h(t)},$$

where

- $\beta(t)$ is the periodic biting rate of mosquitoes to humans,
- a is the probability of infection from infectious mosquitoes to susceptible humans,
- b is the probability of infection from infectious humans to susceptible mosquitoes,
- c is the probability of infection from immune humans to susceptible mosquitoes.

It is obvious that functions f and g satisfy assumptions **(H5)**, **(H6)**, **(H7)**, **(H8)** and **(H9)**.

5.1 Estimation of model parameters

We suppose that the total human population for the concerned region is 1,500,000 and the life expectancy is 50 years. Then, the human recruitment rate Λ_h and natural death rate d_h can be respectively estimated as follows:

$$d_h = \frac{1}{12 \times 50} = 0.0016 \text{ per month},$$
$$\Lambda_h = d_h \times 1{,}500{,}000 + 2600 = 5000 \text{ humans per month}.$$

Moreover, the values of some constant parameters for malaria transmission model (1) are listed in Table 1.

Moreover, assuming that the average number of mosquito bites depends on their gonotrophic cycle which is also a function of temperature, then according to Lou and Zhao (2010), the duration of mosquito gonotrophic cycles can be fitted by Tejerina et al. (2008):

$$G(\theta) = \frac{30.4}{107.204 - 13.3523\theta + 0.677509\theta^2 - 0.0159732\theta^3 + 0.000144876\theta^4} \text{ per month},$$

where θ represents the temperature.

Then, the temperature-dependent biting rate of mosquitoes is given by:

$$\beta(\theta) = \frac{107.204 - 13.3523\theta + 0.677509\theta^2 - 0.0159732\theta^3 + 0.000144876\theta^4}{30.4} \text{ per month}.$$

Parameters	Values	References	Dimensions
Λ_h	5000	estimated	month^{-1}
d_h	0.0016	estimated	month^{-1}
d_p	0.0028	Lou and Zhao, 2010	month^{-1}
α_h	3.04	Lou and Zhao, 2010	month^{-1}
r_h	0.0159	Lou and Zhao, 2010	month^{-1}
γ	0.0167	Lou and Zhao, 2010	month^{-1}
α_v	2.523	Chitnis et al., 2008	month^{-1}
a	0.022	Chitnis et al., 2008	-
b	0.48	Chitnis et al., 2008	-
c	0.048	Chitnis et al., 2008	-
τ	0.49	estimated	month^{-1}

Table 1: Parameters used for numerical simulation.

Similarly, we have:

$$d_v(\theta) = 3.04 + 29.564 \exp\left(\frac{75,935.7 - \theta}{2.7035}\right) \text{ per month}$$

and

$$d_i(\theta) = \frac{30.4}{-4.4 + 1.31\theta - 0.03\theta^2}.$$

Assuming that the temperature varies as a function of time, then we can write functions β and d_v in the following general form:

$$\beta(t) = \beta_0 + \tilde{\beta}_0 \cos\left(\frac{\pi t}{6}\right), \qquad d_v(t) = \alpha_0 + \tilde{\alpha}_0 \cos\left(\frac{\pi t}{6}\right), \qquad d_i(t) = \gamma_0 + \tilde{\gamma}_0 \cos\left(\frac{\pi t}{6}\right).$$

For our computation, we use the reproduction function $B(t, N_v(t)) = i(t)N_v(t)$, where $i(t)$ is the egg-laying rate per adult female per month. In addition, since the ω-periodic function $i(t)$ is proportional to the biting rate $\beta(t)$ (Wang and Zhao, 2018), then we have $i(t) = e\beta(t)$. Finally, function $B(t, N_v)$ is read as follows:

$$B(t, N_v(t)) = e\beta(t)N_v(t).$$

5.2 Numerical results

Using the values in Table 1, we simulate model (1) with different values of the basic reproduction number, $\mathcal{R}_0$, in order to illustrate our theoretical results and analyze its behavior.

5.2.1 Persistence of malaria

Considering the initial conditions $S_h(0) = 1{,}000{,}000$, $E_h(0) = 30$, $I_h(0) = 20$, $R_h(0) = 499{,}950$, $S_v(0) = 2{,}950{,}000$, $E_v(0) = 30{,}000$, $I_v(0) = 20{,}000$, we obtain what appears in Figure 2(a)–(f).

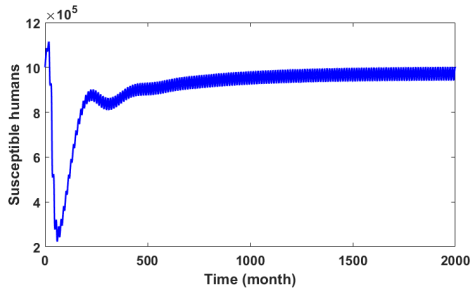
Figure 2 shows that malaria persists in both populations and system (1) converges toward a positive periodic solution. This numerical result is obtained with $\mathcal{R}_0 = 1.10 > 1$ that illustrates the result of our Theorem 4.2.

5.2.2 Extinction of disease

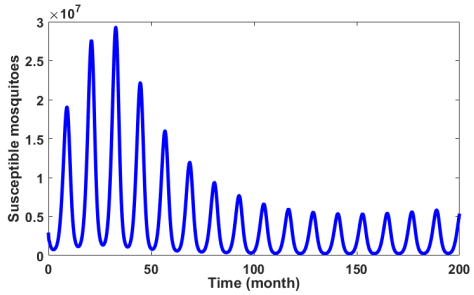
To study the extinction of the disease, we assume that after several years of infection, humans realize the seriousness of the infection and decide to implement some measures to control the expansion of the disease. These measures are intended to prevent mosquito bites and to reduce the number of adult mosquitoes in the city through the use of bed nets and insecticides. Thus, using the values in Table 1 and considering the following initial conditions $S_h(0) = 250{,}050$, $E_h(0) = 250{,}000$, $I_h(0) = 500{,}000$, $R_h(0) = 499{,}950$, $S_v(0) = 20{,}000$, $E_v(0) = 30{,}000$, $I_v(0) = 2{,}950{,}000$, we obtain what appears in Figure 3(a)–(d).

Figure 3 shows that despite the high initial proportion of infected humans and mosquitoes, the disease disappears from both populations in the long run and the solution of system (1) converges to the periodic disease-free equilibrium $(N_h^*, 0, 0, 0, N_v^*(t), 0, 0)$ which is globally asymptotically stable. That illustrates the result of our Theorem 4.1.

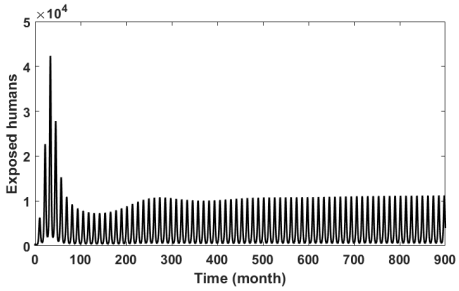
Moreover, since functions B, d_v, β and d_i are temperature dependent, then a simple variation in temperature has significant effect on the dynamics of the disease transmission. That situation means that malaria resurgence can happen in certain regions due to climate change.



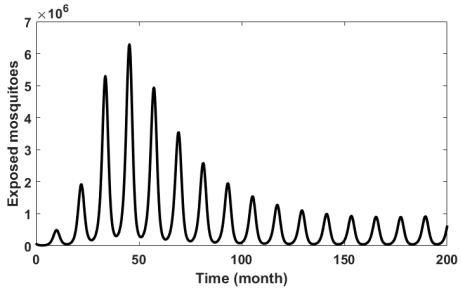
(a) Density of susceptible humans.



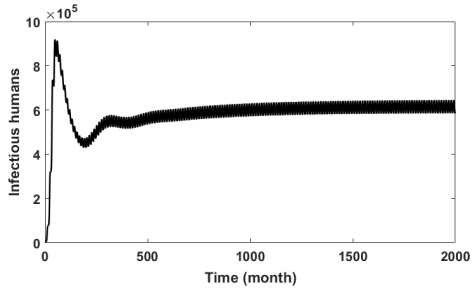
(b) Density of susceptible mosquitoes.



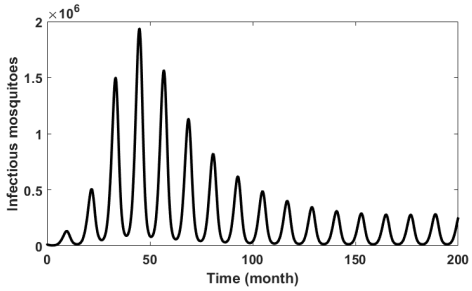
(c) Density of exposed humans.



(d) Density of exposed mosquitoes.

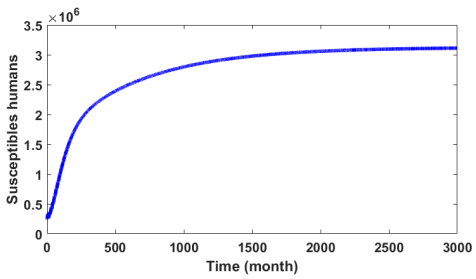


(e) Density of infectious humans.

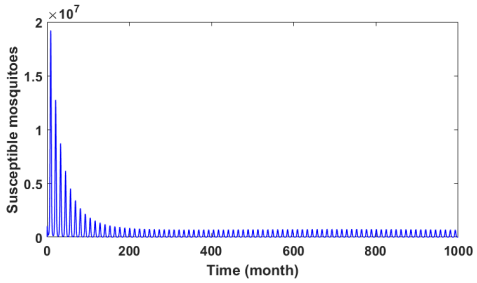


(f) Density of infectious mosquitoes.

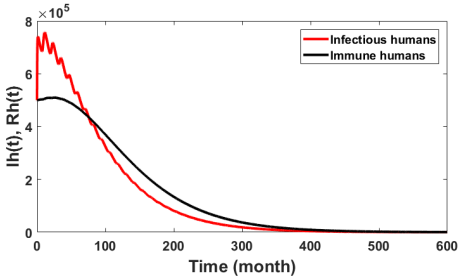
Figure 2: Persistence of malaria for $\beta_0 = 8, \tilde{\beta}_0 = 4, \alpha_0 = 4.5, \tilde{\alpha}_0 = 1.5, \gamma_0 = 2.85, \tilde{\gamma}_0 = 1.5, e = 2.5$ and $\mathcal{R}_0 = 1.10$.



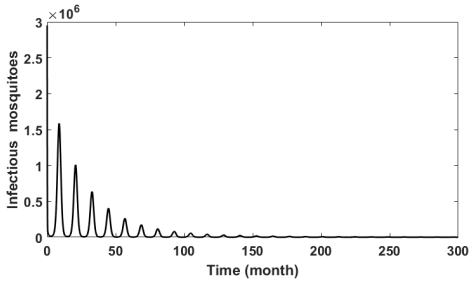
(a) Density of susceptible humans.



(b) Density of susceptible mosquitoes.

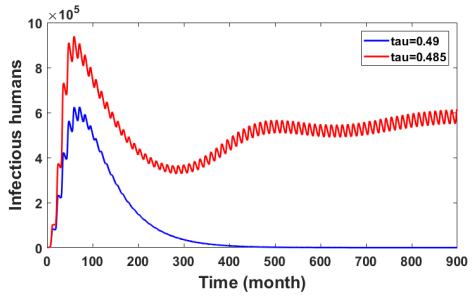


(c) Density of infectious and immune humans.

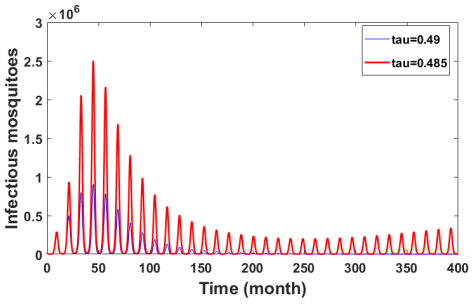


(d) Density of infectious mosquitoes.

Figure 3: Extinction of malaria for $\beta_0 = 6.95, \tilde{\beta}_0 = 3, \alpha_0 = 5, \tilde{\alpha}_0 = 3, \gamma_0 = 2.5, \tilde{\gamma}_0 = 1.5, d_p = 0, e = 2.5$ and $\mathcal{R}_0 = 0.91$.



(a) Density of infectious humans.



(b) Density of infectious mosquitoes.

Figure 4: Evolution of infectious humans and mosquitoes for $\beta_0 = 8, \tilde{\beta}_0 = 4, \alpha_0 = 4.5, \tilde{\alpha}_0 = 1.5, \gamma_0 = 2.85, \tilde{\gamma}_0 = 1.5, e = 2.5$.

5.2.3 Effect of the maturation period on the dynamics

In order to reduce the malaria-induced death rate for humans, it is necessary to know the relative importance of the different factors responsible for its transmission. Thus, we will analyze the impact of the duration of the immature state on the transmission. Considering the following initial conditions $S_h(0) = 1{,}000{,}000$, $E_h(0) = 30$, $I_h(0) = 20$, $R_h(0) = 499{,}950$, $S_v(0) = 2{,}950{,}000$, $E_v(0) = 30{,}000$, $I_v(0) = 20{,}000$, we obtain what appears in Figure 4(a)–(b).

Figure 4 shows that a small perturbation of the maturation delay has a large impact on the transmission of malaria. Indeed, the number of infected humans and mosquitoes increases (resp. decreases) if the delay is low (resp. is high). Thus, the maturation delay of immature mosquitoes is an important factor in the dynamics of malaria transmission. Hence, it can be used to control the transmission.

6 Conclusion

In this paper we have presented a mathematical model of malaria transmission with seasonal fluctuation by using a general periodic incidence function and a general reproduction function. The immature mosquitoes have been considered in the model and their maturation period has been incorporated too. The basic reproduction number, $\mathcal{R}_0$ of the non-autonomous periodic model has been determined, and we have shown that it is the threshold parameter between the persistence and the extinction of

the disease. It emerges from our study that under some assumptions, the periodic disease-free equilibrium is globally stable if $\mathcal{R}_0 < 1$, whereas the disease is persistent if $\mathcal{R}_0 > 1$.

Moreover, we have computed the basic reproduction number of the autonomous model associated to our periodic model and then we have shown that the choice of incidence function has a large impact on the dynamics of malaria transmission. For example, we have shown that the basic reproduction number calculated with standard incidence function (used by Lou et al.) is smaller than basic reproduction number calculated with saturated incidence function (used by Roop et al.) Thus, to avoid the use of several incidence functions in the modeling of malaria transmission, we have constructed a general function which include a large class of incidence function. In addition, we have shown that maturation period has significant effect on the disease transmission dynamics. More precisely, the length of the maturation period determines how fast or how slow the disease will progress within an area. That result has been numerically illustrated by using specific initial values and different lengths of the maturation period (see Figure 4). Thus, numerical simulations have shown that the maturation period τ of juvenile mosquitoes is an efficient parameter in fighting against malaria transmission. However, in practice it is challenging to control the duration of the juvenile stage. Nevertheless, we can avoid this challenge by incorporating the compartment of juvenile mosquitoes in the model. That will allow elimination of the disease through the juvenile population reduction.

Moreover, we note that the global stability of the positive ω-periodic solution has not been established due to the complexity of the model (Traoré et al., 2020; Li et al., 2017; Hu and Sun, 2011). So, we keep this problem for future investigations.

References

Agusto, F. (2014). Malaria drug resistance. the impact of human movement and spatial heterogeneity. *Bulletin of Mathematical Biology 76*(7), 1607–1641. 37, 38

Ai, S., J. Lu, and J. Li (2011). Mosquito stage-structured malaria models and their global dynamics. *SIAM Journal on Applied Mathematics 72*(4), 1213–1237. 38

Alexander, M. E. and S. M. Moghadas (2014). Periodicity in an epidemic model with a generalized nonlinear incidence. *Mathematical Biosciences 189*(1), 75–96. 37, 38

Anderson, R. M. and R. M. May (1982). *Population biology of infectious diseases*. Spring-Verlag, Berlin, Heidelberg, New York. 37, 38

Bai, Z. and Y. Zhou (2012). Global dynamics of an seirs epidemic model with periodic vaccination and seasonal contact rate. *Nonlinear Analysis: Real World Applications 13*(3), 1060–1068. 37, 42

Burnett, M. and D. O. White (1974). *Natural history of infectious disease*. 4th ed, Cambridge University Press, Cambridge, UK. 37, 43

Capasso, V. and A. Serio (1978). A generalization of the kermack mckendrick deterministic epidemic model. *Mathematical Biosciences 42*(1–2), 41–61. 38, 43

Chitnis, N., J. M. Hyman, and J. M. Cushing (2008). Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model. *Bulletin of Mathematical Biology 70*(5), 1272–1296. 38, 43, 49

Chiyaka, C., J. M. Tchuenche, W. Garira, and S. Dube (2008). A mathematical analysis of the effects of control strategies on the transmission dynamics of malaria. *Applied Mathematics and Computation 195*(2), 641–662. 38, 43

Hale, J. K. (1977). *Theory of functional differential equations*. Springer, Heidelberg. 44

Hu, X. and F. Sun (2011). Threshold dynamics for an epidemic model with acute and chronic stages. *International Journal of Nonlinear Science 12*(1), 105–111. 38, 52

Kermack, W. O. and A. G. McKendrick (1927). A contribution to the mathematical theory of epidemics. *Proceedings of the Royal Society of London A: Mathematical, Physical and Engineering Sciences 115*(772), 700–721. 37

Korobeinikov, A. (2006). Lyapunov functions and global stability for sir and sirs epidemiological models with non-linear transmission. *Bulletin of Mathematical Biology 68*(3), 615–626. 38

Koutou, O., B. Traoré, and B. Sangaré (2018a). Mathematical model of malaria transmission dynamics with distributed delay and a wide class of nonlinear incidence rates. *Cogent Mathematics & Statistics 5*(1), 1–25. 37, 39, 43, 44

Koutou, O., B. Traoré, and B. Sangaré (2018b). Mathematical modeling of malaria transmission global dynamics: taking into account the immature stages of the vectors. *Advances in Difference Equations 2018*(220), 1–34. 38, 39, 44

Lakshmikantham, V., S. Leela, and A. A. Martynyuk (1989). *Stability analysis of Nonlinear systems*. Marcel Dekker Inc., New York, Basel. 45

Li, L., Z. Teng, S. Ruan, M. Li, and X. Feng (2017). A mathematical model for the seasonal transmission of schistosomiaisis in the lake and marshland regions of china. *Mathematical Biosciences and engeneering 14*(5-6), 1279–1299. 38, 52

Liu, W., S. A. Levin, and Y. Iwasa (1986). Influence of nonlinear incidence rates upon the behavior of sirs epidemiologic models. *Journal of Mathematical Biology 23*(2), 187–204. 38

Lou, Y. and X.-Q. Zhao (2009). Threshold dynamics in a time-delayed periodic sis epidemic model. *Discrete & Continuous Dynamical Systems Ser. B 12*(1), 169–186. 41

Lou, Y. and X.-Q. Zhao (2010). A climate-based malaria transmission model with structured vector population. *SIAM Journal on Applied Mathematics 70*(6), 2023–2044. 38, 41, 48, 49

Lutambi, A. M., M. A. Penny, T. Smith, and N. Chitnis (2013). Mathematical modelling of mosquito dispersal in a heterogeneous environment. *Mathematical Biosciences 241*(2), 198–216. 38

Ma, Z. and J. Li (2009). *Dynamical modeling and analysis of epidemics*. World Scientific Publishing Co. Pte. Ltd, Singapore. 37, 38

Macdonald, G. (1957). *The epidemiology and control of malaria*. Oxford University press, London. 38

Magal, P. and X.-Q. Zhao (2006). Global attractors and steady states for uniformly persistent dynamical systems. *SIAM Journal on Mathematical Analysis 37*(1), 251–275. 46, 48

Okuneye, K. and A. B. Gumel (2017). Analysis of a temperature-rainfall-dependent model for malaria transmission dynamics. *Mathematical Biosciences 289*, 72–92. 38

Olaniyi, S. and O. S. Obabiyi (2013). Mathematical model for malaria transmission dynamics in human and mosquito populations with nonlinear forces of infection. *International Journal of Pure and Applied Mathematics 88*(1), 125–156. 38, 42

Ouedraogo, H., W. Ouedraogo, and B. Sangaré (2018). A self-diffusion mathematical model to describe the toxin effect on the zooplankton dynamics. *Nonlinear Dynamics and Systems Theory 18*(4), 392–408. 44

Ouedraogo, H., W. Ouedraogo, and B. Sangaré (2019). A mathematical model with a trophic chain predation based on the odes to describe fish and plankton dynamics. *Annals of the University of Craiova - Mathematics and Computer Science Series 46*(1), 164–177. 44

Posny, D. and J. Wang (2014). Modelling cholera in periodic environments. *Journal of Biological Dynamics 8*(1), 1–19. 38

Roop-O, P., W. Chinviriyasit, and S. Chinviriyasit (2015). The effect of incidence function in backward bifurcation for malaria model with temporary immunity. *Mathematical Biosciences 265*, 47–64. 37, 38, 41, 42

Ross, R. (1911). *The prevention of malaria*. John Murray, London. 38

Smith, H. L. (1995). Monotone dynamical systems: an introduction to the theory of competitive and cooperative systems. *Mathematical Surveys and Monographs 41, AMS, Providence, RI*. 42, 44

Tejerina, R., V. Quispe, and T. Chavez (2008). A physiological time analysis of the duration of the gonotrophic cycle of anopheles pseudopunctipennis and its implications for malaria transmission in bolivia. *Malaria Journal 7*(141), 1–17. 48

Tian, D. and H. Song (2017). Global dynamics of a vector-borne disease model with two delays and nonlinear transmission rate. *Mathematical Methods in Applied Sciences 40*(18), 6411–6423. 43

Tiana, J. and J. Wang (2015). Some results in floquet theory, with application to periodic epidemic models. *Applicable Analysis 94*(6), 1128–1152. 38

Traoré, B., O. Koutou, and B. Sangaré (2020). A global mathematical model of malaria transmission dynamics with structured mosquito population and temperature variations. *Nonlinear Analysis: Real World Applications 53*, 1–33. 37, 38, 39, 48, 52

Traoré, B., B. Sangaré, and S. Traoré (2017). A mathematical model of malaria transmission with structured vector population and seasonality. *Journal of Applied Mathematics 2017*(ID 6754097), 1–15. 38

Traoré, B., O. Koutou, and B. Sangaré (2019). Global dynamics of a seasonal mathematical model of schistosomiasis transmission with general incidence function. *Journal of Biological Systems 27*(1), 19–49. 38

Traoré, B., B. Sangaré, and S. Traoré (2018). A mathematical model of malaria transmission in a periodic environment. *Journal of Biological Dynamics 12*(1), 400–432. 38, 48

Van den Driessche, P. and J. Watmough (2002). Reproduction numbers and sub-threshold endemic equilibria for compartmental models of disease transmission. *Mathematical Biosciences 180*(1–2), 29–48. 42

Wang, J., Z. Teng, and T. Zhang (2011). Thresholds dynamics of a malaria transmission model in periodic environment. *Communications in Nonlinear Science and Numerical Simulation 18*(5), 1288–1303. 38

Wang, W. and X.-Q. Zhao (2008). Threshold dynamics for compartmental epidemic models in periodic environments. *Journal of Dynamics and Differential Equations 20*(3), 699–717. 42, 43, 44

Wang, X. and X.-Q. Zhao (2018). A climate-based malaria model with the use of bed nets. *Journal of Mathematical Biology 77*(1), 1–25. 38, 49

Zhao, X.-Q. (2003). *Dynamical Systems in Population Biology*. CMS books Math./ ouvrages Math, SMC, 16 Springer-Verlag, New York. 38, 48