Modeling pyrethroids repellency and its role on the bifurcation analysis for a bed net malaria model

Berge Tsanou[a,c,1] **Jean C. Kamgang**[b] **Jean M.-S. Lubuma**[a] **Duplex E. Houpa Danga**[b]

[a] *Department of Mathematics and Applied Mathematics, University of Pretoria, Pretoria 0002, South Africa*
[b] *Department of Mathematics and Computer Science, University of Ngaoundéré, P.O. Box 454 Ngaoundéré, Cameroon*
[c] *IRD UMI 209 UMMISCO, University of Yaounde I, P.O. Box 337 Yaoundé, Cameroon*
and LIRIMA-EPITAG Team Project, University of Yaounde I, P.O. Box 812 Yaoundé, Cameroon

Abstract

We develop and analyze a simple mathematical model for malaria transmission where pyrethroids treated nets (PTNs) are used for control purposes, and in which knock-down/lethal, excito-repellent/deterrent effects are incorporated. We explicitly describe the contact rates between mosquitoes and humans by nonlinear functions of bed net usage and repellency rates. Using center manifold theory, we show that our model exhibits, saddle-node, transcritical forward and backward bifurcation when the reproduction number $\mathcal{R}_0$ crosses one. Our model reveals that repellency effect plays an important role for the existence of both the endemic equilibrium points and the occurrence of backward bifurcation and a threshold repellency rate is calculated. The epidemiological implication of the backward bifurcation is that, reducing $\mathcal{R}_0$ below one alone is not enough to eliminate malaria. We establish that, increasing either the rate of bed nets usage (i.e community protection) or their repellent effect (i.e personal protection) or the combination of both, decreases the contact rates between humans and mosquitoes. As a result, the disease burden metric $\mathcal{R}_0$ is reduced. The global asymptotic stability of equilibrium points are proven using the geometric approach and Lyapunov-LaSalle techniques. Furthermore, we show that neglecting repellency underestimates the basic reproduction number and hinders the control of malaria. The disease free equilibrium is shown to be a saddle-node of co-dimension 1 when $\mathcal{R}_0 = 1$. We also observe that $\mathcal{R}_0$ is mostly influenced by the bed net coverage rate, repellent effect of pyrethroids and the probability that mosquitoes target human hosts. Our results confirm that PTNs usage is an efficient control strategy to mitigate the malaria ability to spread, and suggest that the utilization of PTNs with high lethal rate, but low repellency rate is better than the use of those with high repellency and low lethal rates.

Keywords: Malaria; Pyrethroids; Lethal effect; Excito-repellent effect; Basic reproduction number; Backward bifurcation; Saddle-node bifurcation

1. Introduction

Malaria is an infectious disease caused by the plasmodium parasite and transmitted between humans through bites of the female Anopheles mosquitoes, sometimes called "malaria vectors", which bite mainly between dusk and dawn. In 2018 an estimated 228 million cases of malaria occurred worldwide and 405000 people died, mostly

[1] *Corresponding author permanent address:* Department of Mathematics and Computer Science, University of Dschang, P.O. Box 67 Dschang, Cameroon. E-mails: berge.tsanou@up.ac.za / berge.tsanou@univ-dschang.org

children in the African region [8]. The World Health Organization (WHO) estimates that every year 250 million people become infected and nearly one million die [1].

During the past decade, several interventions have been used to reduce malaria transmission. These include insecticide-treated nets (ITNs) and particularly PTNs, indoor residual spraying (IRS), intermittent preventive treatment in pregnant women and infants, larval control, and other vector control interventions. ITNs have proven to be one of the most effective intervention measures against malaria in reducing morbidity and mortality [21, 11, 24, 40, 26, 30]. It is only in the years 1970 with the development of pyrethroids of synthesis, less toxic for the vertebrates and environment that the use of ITNs gained more consideration [13]. The pyrethroids are today the only insecticide authorized for the impregnation of bed nets , because of their efficiency, their repulsive effect and their weak toxicity to humans[47, 48, 49]. Actually, they induce many effects on insects which we mention below.

- Knock-down effect: The direct contact with the impregnated material leads to a disruption of nerve impulse and causes a brief paralysis of the mosquito.
- Lethal effect: Depending on the duration of contact, the quantity of active material found on the bed net and the sensitivity of mosquito, an exposition can lead to the quick death of the latter.
- Excito-repellency effect: The direct contact of the mosquito with impregnated material causes a quick escape.
- Deterrent or dissuasive effect: Contrary to the above mentioned effects, this one does not involve any physical contact between the mosquito and bed net, rather the presence of a mosquito net in a home or in a bedroom can cause an avoidance behavior of the mosquito, dissuaded by the smell of insecticide.

Promising tests have revealed the efficiency of pyrethroids impregnated mosquito nets to limit the contact between human and mosquito [12, 14, 48]. Moreover, the protection offered by insecticide treated bed nets has been documented in many studies, ([16, 25, 28, 46, 20, 27], just to mention few) relying on the following three main mechanisms [24, 26, 30, 25],.

1- The nets create a physical barrier between the humans and mosquito vectors.

2- The insecticide used to treat the bed net repels mosquitoes (excito-repellency or deterrence, simply referred to as repellency), thus increasing the personal protection offered by the net.

3- If a mosquito fails to be repelled, it will often rest on the bed net, and may then be killed by lethal effect by contacting the insecticidal material found on the net.

Mathematical transmission models of infectious agents can be useful tools in understanding disease dynamics and assessing the impact of human behavior. As for the malaria disease, this human behavior incorporation had been taken care by considering the use of impregnated bed nets models in the following works [1, 5, 6, 35, 31, 37, 45] (just to mention few). on the mathematical modeling for the transmission of malaria focus only on the human behavior. Ngonghala et al. in [35] used a deterministic model for malaria spread that captures the decrease in ITNs effectiveness due to physical and chemical decay, as well as human behavior as a function of time.

Motivated by the well documented strong influence of behavioral factors in ITNs usage, in [6] Buonomo proposed a mathematical approach based on the idea of information-dependent epidemic models. The work by Mohammed and Numfor [31] studied a mathematical model for malaria-HIV co-infection transmission and control, in which insecticide-treated nets and malaria treatment are incorporated. More recently, Nwankwo et al. in [37] developed a mathematical model for malaria transmission that investigates the impact of temperature on the efficacy of ITNs. The work by Wang Xiunan et al. in [45] served to investigate the impact of bed net use on malaria control. |More precisely, they formulated a periodic vector-bias malaria model incorporating the juvenile stage of mosquitoes and the use of ITNs. The authors in [5] constructed a malaria model which includes the enhanced attractiveness of infectious humans to mosquitoes, as result of host manipulation by malaria parasites, and the human behavior, represented by insecticide-treated bed-nets usage. The researchers in [1] used a simple deterministic model that considers the transmission dynamics of malaria infection in mosquito and human populations, and investigate the impact of bed nets usage on its control. None of the above-mentioned works have explicitly modeled, incorporated and studied the role of excito-repellency/deterrence effect on the transmission dynamics of malaria. Moreover, they did consider the disease contact (human-to-mosquito and mosquito-to-human) rates as linear functions of the bed nets usage, which in reality can be strongly nonlinear functions of bed nets usage. In particular, the model by Agusto et al. in [1] reads as follows:

$$\begin{cases} \dot{S_h} = \Lambda_h - \lambda_h(b)S_h + \gamma_h I_h - \mu_h S_h \\ \dot{I_h} = \lambda_h(b)S_h - (\mu_h + \gamma_h + \delta)I_h \\ \dot{S_v} = \Lambda_v - \lambda_v(b)S_v - \mu_v(b)S_v \\ \dot{I_v} = \lambda_v(b)S_v - \mu_v(b)I_v. \end{cases} \tag{1.1}$$

In system (1.1), the human-to-mosquito and mosquito-to-human forces of infection are given by

$$\lambda_h(b) = \frac{m_h\beta(b)I_v}{N_h}, \quad \lambda_v(b) = \frac{m_v\beta(b)I_h}{N_h},$$

where, the average number of bites per mosquito per unit time (contact rate) $\beta(b)$, the mosquito mortality rate were described by linear decreasing functions of treated bed-net usage (b) as follows:

$$\beta(b) = \beta_{max} - b(\beta_{max} - \beta_{min}), \quad \mu_v(b) = \mu_{v0} + \mu_{v1}b, \quad 0 \leq b \leq 1,$$

where, the parameters β_{max} and β_{min} are the maximum and the minimum transmission rates, respectively, and (b) is the proportion of treated bed-net usage that could reduce the contact rate to a minimum level β_{min}. On the other hand, the human-to-mosquito and the mosquito-to-human contact rates are identical and modeled by the function $\beta(b)$. The natural mortality rate of mosquito is μ_{v0}, while $\mu_{v_1}b$ is an additional mortality rate due to ITNs utilization. The quantity m_h is the transmission probability per bite from infectious mosquitoes to humans and similarly, m_v is the transmission probability per bite from infectious humans to mosquitoes. The remaining parameters for the model (1.1) are recalled in Table (1). $N_h = S_h + I_h$ and $N_v = S_v + I_v$ are the total human and mosquito populations, respectively. This above-mentioned drawback in modeling the (human-to-mosquito and mosquito-to-human) contact rates, in the framework of insecticide or pyrethroids bed nets utilization, as identical linear functions of bed nets usage rate (b) is our main focus on this paper.

Therefore, the main purpose of this work is to model and do an in-depth analysis of a simple mathematical model of bed nets for malaria transmission, in which both lethal/knock-down and repellent/deterrent effects of ITNs/PTNs are explicitly modeled and incorporated in the model. For simplicity, we will address this by extending the model (1.1) above in a more realistic manner such that the mosquito-to-human and human-to-mosquito contact rates are distinct nonlinear functions of bed nets usage, and the repellent effect of pyrethroids explicitly considered. More importantly, the role of repellency on the model's bifurcation analysis will be insightfully assessed. Since, on the one hand, the knock-down and lethal effects of PTNs have similar consequences on the mosquitoes and, on the other hand, the excito-repellent and deterrent effects have the same consequences on mosquitoes behavior, we shall explicitly consider and model only two PTNs effects (repellency and lethality) on mosquitoes, and assess their influences on the dynamics of malaria in this manuscript. Precisely, we explicitly formulate the contact rates as nonlinear functions of bed net usage and repellency efficacy. We show that these functions are decreasing with respect to bed net usage and repellency effect. We perform a bifurcation analysis and highlight the occurrence of a backward bifurcation as a result of the repellency effect and/or the disease induced death rate. The consequence of the latter being the occurrence of a bistable situation where, a stable endemic equilibrium point co-exists with a stable disease-free equilibrium point whenever the reproduction number $\mathcal{R}_0$ is less than unity. The type of disease free equilibrium when $\mathcal{R}_0 = 1$ is also characterized. Using the geometric approach, we also establish the global asymptotic stability of the endemic equilibrium for $\mathcal{R}_0 > 1$. All these investigations are gathered in the remaining outline of the paper as follows. Section 2 deals with the details of model formulation. Asymptotic and bifurcation analyses of the model are presented in Section 3. Sensitivity analysis and simulations are performed in Section 4. Finally, Section 5 discusses and concludes the manuscript.

2. Model formulation and basic properties

2.1. Model formulation

We formulate a simple mathematical bed net model for the transmission dynamics of malaria in which, lethal/knock-down, and excito-repellency/deterrence effects are explicitly incorporated in disease contact rates as nonlinear functions. The human population is described by two classes such that at time $t \geq 0$, there are $S_h(t)$ susceptibles and $I_h(t)$ infectious. Similarly, the mosquito population has two components such that at time $t \geq 0$, there are $S_v(t)$ susceptibles and $I_v(t)$ infectious mosquitoes. Thus, the total human and mosquito populations at time t, are $N_h(t) = S_h(t) + I_h(t)$ and $N_v(t) = S_v(t) + I_v(t)$, respectively. In our model formulation, it is assumed that malaria is neither transmitted vertically nor horizontally. That is, all new human and mosquito births are susceptible and there is neither direct human-to-human nor mosquito-to-mosquito transmissions.

We emphasize that, though the forces of infection in model (1.1) originated from [1], as well as in many recent bed net models [5, 6, 31, 37, 45] have considered the lethal/knock-down effect of PTNs, they failed to explicitly incorporate the relevant excito-repellency/deterrence effect of pyrethroid impregnated nets. In addition to that, these recent works modeled the contact rates in the form of linear functions of bed net usage. It is worth mentioning that excito-repellency is important as it provides protection by diverting mosquitoes to non-human hosts, specially when all individuals in the population are PTN-users [3, 24, 25, 26, 30]. This is going to be carefully taken care

of in the modeling setting in this manuscript. Thus, we explicitly model the forces of infection for susceptible humans and vectors in the form of distinct nonlinear functions of bed net usage, and for the first time, as nonlinear functions of repellency effect.

2.1.1. Modeling the human-to-mosquito force of infection for susceptible humans

In this section, as well as in the next, as our main focus is the role played by the repellent and lethal effects of PTNs, we model the human-to-mosquito force of infection by explicitly quantifying the probability that a mosquito initiates bites on humans.

Since the female Anopheles mosquitoes answerable of the transmission of malaria bite mainly between dusk and dawn, we assume for simplicity that, the mosquito population under consideration is constituted of mosquitoes who target human beings when they are sleeping indoors (endophilic mosquitoes). Actually, if a mosquito enters a house where someone sleeps under a bed net, then it is repelled by the insecticide (or mechanically blocked by the net) with a probability r (note that the repellency parameter includes both, repellency caused by volatiles of the insecticide as well as repellency due to the sheer physical feature of the net); or if it is not repelled, it takes its bites and escapes with probability $(1-\mu_b)$; or it is killed by the insecticide on the net thanks to the lethal effect of the insecticide with probability μ_b. We assume that the killing probability μ_b depends as in [1], on the proportion b of PTNs usage in the linear simplistic form $\mu_b = \mu_b(b) = \mu_{v1}b,\ 0 \leq b \leq 1$, where μ_{v1} is the maximum PTN-induced death rate of mosquitoes. Notice that, when $b = 1$, the whole human population is protected by PTNs, while $b = 0$ models no PTN utilization.

We assume that, a mosquito targets human hosts with probability θ and non-human hosts with probability $(1-\theta)$. If it bites indoors (at a time when people are sleeping), it can target an PTN-user during its first biting attempt, during its second attempt (having been repelled once), during its third attempt (having been repelled twice), etc. If a mosquito is repelled by a bed net, it leaves the house and continues to search for alternate hosts to bite. Thanks to lethal effect, if each additional search of a host brings with it the risk μ_b of dying, then the probability of having been repelled n times is $[\theta rb(1-\mu_b)]^n$. Here, the product rb is referred to as "*the efficacy rate*" of PTNs usage. An unprotected human is defined here as a person who use a completely inefficient bed net. We model the probability of biting a human host after a single attempt by a decreasing function of the efficacy rate of PTNs usage in the form $\theta(1-rb)$. Hence, the following observations hold.

(i) When $b = 1$, the whole human population is protected by PTNs and this probability of biting a human host after a single attempt becomes the probability $\theta(1-r)$ of targeting a human host with probability θ when it is not repelled by the insecticide with probability $(1-r)$.

(ii) When $b = 0$ or $r = 0$, that is no bed net is used, or if any is used, it completely fails to repel the mosquitoes. Thus, $\theta(1-rb)$ reduces to the probability θ that the mosquito targets a human host and bites him after a single attempt.

(iii) When $r = 1$, this probability $\theta(1-rb)$ of biting a human host after a single attempt becomes the probability $\theta(1-b)$ of targeting an unprotected human host.

Thus, proceeding similarly as in [3], the probability that a mosquito finally bites a human host is

$$P_h(r,b) = \theta(1-rb)\sum_{n=0}^{+\infty}[\theta rb(1-\mu_b)]^n = \theta(1-rb)\sum_{n=0}^{+\infty}[\theta rb(1-\mu_{v1}b)]^n = \frac{\theta(1-rb)}{1-\theta rb(1-\mu_{v1}b)}.$$

As expected, one can notice that the probability that a mosquito initiates a bite on a human host actually depends on both the repellent probability r, and the treated bed-net usage rate b. The latter dependence also accounts for the impact of human behavior on PTNs effectiveness. Moreover, note that $P_h(r,b) = 0$ whenever $r = b = 1$. This stands for personal protection where mosquito repellency and lethality assume 100% maximal level and represents the ideal situation for which PTNs either divert mosquitoes to non-human hosts or kill them.

The parameter m_h denotes the transmission probability per bite from infectious mosquitoes to susceptible humans (actually, m_h is the product of the number of mosquitoes per person and the biting rate of mosquitoes on humans). A similar approach as in [1, 4, 33, 35, 42] yields the average number of bites per human per unit time in the form

$$\frac{P_h(r,b)N_v}{N_h}.$$

Thus, the human-to-mosquito force of infection for susceptible humans is finally quantified by

$$\lambda_h(r,b) = \frac{m_h P_h(r,b) I_v}{N_h}.$$

2.1.2. Modeling the force of infection for susceptible mosquitoes

As mentioned above, PTNs also provide protection by diverting mosquitoes to non-human hosts (animals) [10, 11, 24, 26]. Thus, proceeding similarly as above, it is straightforward to compute the probability that an endophilic mosquito initiates a bite on a non-human host as follows:

$$P_a(r,b) = (1-\theta)\sum_{n=0}^{+\infty}[\theta rb(1-\mu_{v1}b)]^n = \frac{(1-\theta)}{1-\theta rb(1-\mu_{v1}b)}.$$

Likewise, the probability that a mosquito initiates a bite indoors on a non-human host or a human host is

$$P_v(r,b) = P_a(r,b) + P_h(r,b) = \frac{(1-\theta)+\theta(1-rb)}{1-\theta rb(1-\mu_{v1}b)} = \frac{1-rb\theta}{1-\theta rb(1-\mu_{v1}b)}.$$

Note that m_v denotes the transmission probability per bite from infectious humans to susceptible mosquitoes. We assume that this probability is the same as the transmission probability per bite from infectious non-humans to susceptible mosquitoes. We also suppose that the proportion of infectious humans in human host population or in non-human host population is the same. Thus, the force of infection for susceptible vectors is modeled by

$$\lambda_v(r,b) = \frac{m_v P_v(r,b) I_h}{N_h}.$$

2.1.3. The model equations

Let's recall that, due to the treatment of bed-nets by pyrethroids, the female mosquitoes who quest for blood meals could die when they come into contact with a treated bed nets. For simplicity, we follow the approach in [1] and model the death rate of the mosquitoes as a linear function of the bed net efficacy in the form

$$\mu_v(b) = \mu_{v0} + \mu_{v1}b,$$

where, μ_{v0} is the natural death rate and, $\mu_{v1}b$ is the death rate due to the insecticides on the treated bed nets. However, a more complex (nonlinear) form of the function $\mu_v(b)$ could be used and explicitly calculated taking into account the role of bed nets in repelling and/or killing mosquitoes. Altogether, the proposed model that governs the dynamics of malaria transmission in this paper reads as follows:

$$\begin{cases} \dot{S_h} = \Lambda_h - \lambda_h(r,b)S_h + \gamma_h I_h - \mu_h S_h \\ \dot{I_h} = \lambda_h(r,b)S_h - (\mu_h + \gamma_h + \delta)I_h \\ \dot{S_v} = \Lambda_v - \lambda_v(r,b)S_v - \mu_v(b)S_v \\ \dot{I_v} = \lambda_v(r,b)S_v - \mu_v(b)I_v. \end{cases} \tag{2.1}$$

where,

$$\lambda_h(r,b) = \frac{m_h P_h(r,b) I_v}{N_h}, \quad \lambda_v(r,b) = \frac{m_v P_v(r,b) I_h}{N_h},$$

$$P_h(r,b) = \frac{\theta(1-rb)}{1-\theta rb(1-\mu_{v1}b)}, \quad P_v(r,b) = \frac{1-rb\theta}{1-\theta rb(1-\mu_{v1}b)},$$

$$\mu_v(b) = \mu_{v0} + \mu_{v1}b, \quad 0 \le b \le 1, \quad 0 \le r \le 1.$$

Parameters	Descriptions	Baseline values	References
Λ_h	Immigration rate for humans	$10^3/(50 \times 365)$	[9]
Λ_v	Immigration rate for mosquitoes	$10^4/21$	[41]
b	Proportion of PTNs usage	variable	
r	Repellent probability by the insecticide	variable	
θ	Probability that a mosquito targets human hosts	0.71(assumed)	
μ_{v1}	Maximum mosquito PTNs-induced death rate	1/4	[29, 35]
μ_h	Natural mortality rate in humans	$1/(50 \times 365)$	[35]
μ_{v0}	Natural mortality rate in mosquitoes	1/14	[15, 35]
δ	Disease-induced death rate in humans	1/100000	[9]
m_v	Human-to-mosquito probability of disease transmission	0.4	[35]
m_h	Mosquito-to-human probability of disease transmission	0.195	[15, 35]
γ_h	Recovery rate of infectious humans	1/4	[18]

Table 1: Description and baseline values for parameters of system (2.1)

2.2. *Basic properties*

2.2.1. *Impact of PTNs utilization on the contact rate between mosquitoes and humans*

First of all, we should prove that the utilization of bed nets and the explicit formulations of the contact rates taking into account the repellency and killing/knock-down effects are favorable to the control of malaria. In this regard, the following result serves the purpose.

Theorem 2.1. *PTNs usage reduces the contact between mosquitoes and humans. More specifically, the probability that a mosquito finally bites human beings $P_h(r,b)$ (resp. that a mosquito initiates bites on non-humans or humans $P_v(r,b)$) is a decreasing function of both the repellent probability r and the efficacy of bed nets utilization rate b.*

Proof : It suffices to prove that $\frac{\partial P_h(r,b)}{\partial b}, \frac{\partial P_h(r,b)}{\partial r}, \frac{\partial P_v(r,b)}{\partial b}, \frac{\partial P_v(r,b)}{\partial r} \leq 0.$

$$P_h(r,b) = \frac{\theta(1-rb)}{1-\theta rb(1-\mu_{v1}b)}.$$

If $r = 0$, then $P_h(r,b) = \Theta$. Thus, no mosquito is repelled and the bed nets are useless.

If $r > 0$, then

$$\frac{\partial P_h(r,b)}{\partial b} = \frac{-r\theta[1-r\theta b(1-\mu_{v1}b)] - \theta(1-rb)[-r\theta + 2r\theta\mu_{v1}b]}{[1-r\theta b(1-\mu_{v1}b)]^2},$$

$$= \frac{(r^2\theta^2\mu_{v1})b^2 - (2r\theta^2\mu_{v1})b - r\theta(1-\theta)}{[1-r\theta b(1-\mu_{v1}b)]^2}.$$

We focus on the sign of the quadratic polynomial

$$\mathcal{P}(b) = (r^2\theta^2\mu_{v1})b^2 - (2r\theta^2\mu_{v1})b - r\theta(1-\theta). \tag{2.2}$$

Since $r\theta(1-\theta) \geq 0$, Eq. (2.2) has two nonzero real roots $b_1 < 0$ and $b_2 > 0$, where,

$$b_2 = \frac{1}{r}\left(1 + \frac{\sqrt{\theta^2\mu_{v1}^2 + r^3\theta\mu_{v1}(1-\theta)}}{\theta\mu_{v1}}\right).$$

Clearly $b_2 > 1$ and $\mathcal{P}(b) \leq 0$ whenever $b \in [b_1, b_2]$. Consequently, for all $b \in [0,1]$, one always has

$$\frac{\partial P_h(r,b)}{\partial b} \leq 0.$$

Direct computations show that

$$\frac{\partial P_h(r,b)}{\partial r} = \frac{-\theta b\,(1-\theta+\theta\mu_{v1}b)}{[1-r\theta b(1-\mu_{v1}b)]^2} \leq 0,$$

$$\frac{\partial P_v(r,b)}{\partial b} = \frac{r\theta\mu_{v1}b\,(r\theta b-2)}{[1-r\theta b(1-\mu_{v1}b)]^2} \leq 0,$$

$$\frac{\partial P_v(r,b)}{\partial r} = \frac{-\theta\mu_{v1}b^2}{[1-r\theta b(1-\mu_{v1}b)]^2} \leq 0.$$

This achieves the proof.

2.2.2. Positivity and boundedness of solutions

The system of equations in (2.1) monitors human and mosquito populations, all parameters in the model are non-negative. It can be readily shown that, given non-negative initial conditions, the solutions of (2.1) remain non-negative. In order to analyze the system, we split it into two parts, namely the human sub-population model and mosquito sub-populations model, and consider the biologically feasible region

$$\Omega = \left\{(S_h(t), I_h(t), S_v(t), I_v(t)) \in \mathbb{R}_+^4 : 0 \leq N_h(t) \leq \frac{\Lambda_h}{\mu_h}, 0 \leq N_v(t) \leq \frac{\Lambda_v}{\mu_v(b)}\right\}.$$

On the other hand, once can easily establish that is Ω positively invariance of (i.e., solutions initiated in Ω remain in Ω for all $t \geq 0$), attracting and absorbing.

In fact, we have the relations

$$\frac{dN_h(t)}{dt} = \Lambda_h - \mu_h N_h(t) - \delta I_h(t),$$
$$\frac{dN_v(t)}{dt} = \Lambda_v - \mu_v(b) N_v(t),$$

from which it follows that

$$\frac{dN_h(t)}{dt} \leq \Lambda_h - \mu_h N_h(t),$$

$$\frac{dN_v(t)}{dt} = \Lambda_v - \mu_v(b) N_v(t).$$

A standard comparison theorem and a classical integration show respectively that

$$N_h(t) \leq \left(N_h(0) - \frac{\Lambda_h}{\mu_h}\right) e^{-\mu_h t} + \frac{\Lambda_h}{\mu_h},$$

$$N_v(t) = \left(N_v(0) - \frac{\Lambda_v}{\mu_v(b)}\right) e^{-\mu_v(b) t} + \frac{\Lambda_v}{\mu_v(b)}.$$

Now, if $N_h(0) \leq \frac{\Lambda_h}{\mu_h}$ and $N_v(0) \leq \frac{\Lambda_v}{\mu_v(b)}$; then $N_h(t) \leq \frac{\Lambda_h}{\mu_h}$ and $N_v(t) \leq \frac{\Lambda_v}{\mu_v(b)}$.
Hence, Ω is positively invariant and its attractiveness, absorbing properties follow readily. Therefore it is sufficient to consider the dynamics of the flow generated by (2.1) in Ω. This makes the model epidemiologically and mathematically well-posed [22] since we have proven the following result.

Theorem 2.2. *The subset Ω of $\mathbb{R}^4_+$ is a positively invariant, attracting and absorbing compact set for model (2.1). Hence, the model (2.1) is a dynamical system in Ω.*

3. Asymptotic and bifurcation analysis

3.1. Stability of the disease-free equilibrium

The disease-free equilibrium of the system (2.1) is $E_0 = \left(S_h^0, I_h^0, S_v^0, I_v^0\right) = \left(\frac{\Lambda_h}{\mu_h}, 0, \frac{\Lambda_v}{\mu_v(b)}, 0\right)$
Using the next generation operator approach as presented in [43], we calculate the reproduction number $\mathcal{R}_0$ of the system (2.1). In this regard, since the infected compartments are I_h and I_v, we define the function $\widetilde{F}$ for the rate of new infected cases

$$\widetilde{F} = \left(\frac{m_h P_h(r,b) I_v S_h}{N_h}, \frac{m_v P_v(r,b) I_h S_v}{N_h}\right),$$

and the function $\widetilde{V}$ for transfer terms between the disease infected compartments

$$\widetilde{V} = \left((\mu_h + \gamma_h + \delta) I_h, \mu_v(b) I_v\right).$$

We evaluate the Jacobian matrices F and V of $\widetilde{F}$ and $\widetilde{V}$ at the disease-free equilibrium E_0, respectively and obtain

$$F = \begin{pmatrix} 0 & m_h P_h(r,b) \\ m_v P_v(r,b) \frac{\Lambda_v}{\Lambda_h} \frac{\mu_h}{\mu_v(b)} & 0 \end{pmatrix}, \quad V = \begin{pmatrix} (\mu_h + \gamma_h + \delta) & 0 \\ 0 & \mu_v(b) \end{pmatrix}.$$

According to [43], the reproduction number $\mathcal{R}_0$ is the spectral radius of the next generation matrix

$$FV^{-1} = \begin{pmatrix} 0 & \frac{m_h P_h(r,b)}{\mu_v(b)} \\ \frac{m_v P_v(r,b)}{(\mu_h+\gamma_h+\delta)} \frac{\Lambda_v}{\Lambda_h} \frac{\mu_h}{\mu_v(b)} & 0 \end{pmatrix}.$$

That is,

$$\mathcal{R}_0 = \frac{1}{\mu_v(b)} \sqrt{\frac{m_v m_h P_v(r,b) P_h(r,b)}{(\mu_h+\gamma_h+\delta)} \frac{\mu_h \Lambda_v}{\Lambda_h}}.$$

We now proceed to study the local stability of the disease-free equilibrium. The Jacobian of system (2.1) at the disease-free equilibrium is given by

$$J = \begin{pmatrix} -\mu_h & \gamma_h & 0 & -m_h p_h(r,b) \\ 0 & -(\mu_h+\gamma_h+\delta) & 0 & m_h p_h(r,b) \\ 0 & -m_v P_v(r,b) \frac{\Lambda_v}{\Lambda_h} \frac{\mu_h}{\mu_v(b)} & -\mu_v(b) & 0 \\ 0 & m_v P_v(r,b) \frac{\Lambda_v}{\Lambda_h} \frac{\mu_h}{\mu_v(b)} & 0 & -\mu_v(b) \end{pmatrix}.$$

The eigenvalues of J are $\lambda_1 = -\mu_h$, $\lambda_2 = -\mu_v(b)$ plus the two solutions of the quadratic equation,

$$\lambda^2 + (\mu_h+\gamma_h+\delta+\mu_v(b))\,\lambda + \mu_v(b)(\mu_h+\gamma_h+\delta)\left(1-\mathcal{R}_0^2\right) = 0.$$

Clearly, for $\mathcal{R}_0 < 1$, the quadratic equation above has two roots with negative real parts, and for $\mathcal{R}_0 > 1$, it has one positive solution. Thus we have the following result.

Proposition 3.1. *The disease-free equilibrium point E_0 is locally asymptotically stable if $\mathcal{R}_0 < 1$ and it is unstable if $\mathcal{R}_0 > 1$.*

3.1.1. Endemic equilibrium

In this section, we investigate the existence of endemic equilibrium points. In simple epidemic models, typically, the bifurcation will be forward or super-critical, which means that there are no endemic states when $\mathcal{R}_0 < 1$. However, sometimes in more elaborated epidemic models, the phenomenon of backward or sub-critical bifurcation occurs when a stable disease-free equilibrium co-exists with two endemic equilibrium points, one of which is stable and the other one unstable in a region where the reproduction number is less than one [7, 17, 34]. From these studies, it appears that the backward bifurcation is connected to complex biological and social interactions of the infectious class. The latter phenomenon has important disease-control implications as it asserts that reducing the reproduction number to less than one is not enough for disease elimination. Furthermore, the presence of backward bifurcation in an epidemic model implies that additional control measures are needed to bring the epidemic under control. The endemic equilibrium of system (2.1) is denoted by $EE = (S_h^*, I_h^*, S_v^*, I_v^*)$, where,

$$S_h^* = \frac{(\mu_h+\gamma_h+\delta)\Lambda_h}{(\mu_h+\delta)\left(\lambda_h^*(r,b)+\mu_h\right)+\gamma_h\mu_h}, \quad I_h^* = \frac{\lambda_h^*(r,b)\Lambda_h}{(\mu_h+\delta)\left(\lambda_h^*(r,b)+\mu_h\right)+\gamma_h\mu_h},$$

$$S_v^* = \frac{\Lambda_v}{\lambda_v^*(r,b)+\mu_v(b)}, \quad I_v^* = \frac{\lambda_v^*(r,b)\Lambda_v}{(\lambda_v^*(r,b)+\mu_v(b))\,\mu_v(b)},$$

with,

$$\lambda_h^*(r,b) = \frac{m_h P_h(r,b) I_v^*}{N_h^*} = \frac{m_h P_h(r,b)\Lambda_v}{\mu_v(b)} \frac{(\mu_h+\delta)(\lambda_h^*(r,b)+\mu_h)+\gamma_h\mu_h}{\Lambda_h(\mu_h+\gamma_h+\delta+\lambda_h^*(r,b))} \frac{\lambda_v^*(r,b)}{\lambda_v^*(r,b)+\mu_v(b)},$$

$$\lambda_v^*(r,b) = \frac{m_v P_v(r,b) I_h^*}{N_h^*} = \frac{m_v P_v(r,b)\mu_v(b)\lambda_h^*(r,b)}{\mu_v(b)\left(\mu_h+\gamma_h+\delta+\lambda_h^*(r,b)\right)}.$$

Plugging $\lambda_v^*(r,b)$ into $\lambda_h^*(r,b)$ from the above expressions yields the quadratic equation

$$B_2[\lambda_h^*(r,b)]^2 + B_1\lambda_h^*(r,b) + B_0 = 0, \tag{3.1}$$

whose coefficients are given by

$$\begin{cases} B_2 = \mu_v(b)\Lambda_h[m_v P_v(r,b)+\mu_v(b)] > 0, \\ B_1 = (\mu_h+\gamma_h+\delta)\mu_v(b)\Lambda_h m_v P_v(r,b) + 2(\mu_h+\gamma_h+\delta)\mu_v^2(b)\Lambda_h - m_v m_h P_v(r,b)P_h(r,b)\Lambda_v(\mu_h+\delta), \\ B_0 = ((\mu_h+\gamma_h+\delta)\mu_v(b))^2\,\Lambda_h\left(1-\mathcal{R}_0^2\right). \end{cases}$$

If $\Delta := \Delta(\mathcal{R}_0) = B_1^2 - 4B_0B_2 \geq 0$ denotes the discriminant of (3.1), then its roots are $\lambda_{h\pm}^*(r,b) = \dfrac{-B_1 \pm \sqrt{\Delta}}{2B_2}$.
When $\mathcal{R}_0 < 1$, the number of positive roots (and consequently the number of endemic equilibrium points) for (3.1) are determined by the sign of B_1. Let's rewrite B_1 in the form

$$B_1 = (\mu_h+\gamma_h+\delta)\mu_v^2(b)\Lambda_h\frac{(\mu_h+\delta)}{\mu_h}(G-\mathcal{R}_0^2),$$

where,

$$G = \frac{m_v P_v(r,b)+2\mu_v(b)}{\mu_v(b)}\frac{\mu_h}{(\mu_h+\delta)}. \tag{3.2}$$

Theorem 3.2. *Let $\Delta(\mathcal{R}_0) = B_1^2 - 4B_0B_2$. If $G < 1$, then there exists a unique number $\mathcal{R}_0^c \in \left]\sqrt{G}, 1\right[$ satisfying $\Delta(\mathcal{R}_0^c) = 0$.*

Proof : Set $\mathcal{R}_0 \in \left]\sqrt{G}, 1\right[$, we have $B_1 < 0$. The derivative of $\Delta(\mathcal{R}_0)$ with respect to $\mathcal{R}_0$ gives,

$$\Delta'(\mathcal{R}_0) = 4(\mu_h+\gamma_h+\delta)\mu_v^2(b)\Lambda_h\mathcal{R}_0\left(2(\mu_h+\gamma_h+\delta)B_2 - B_1\frac{(\mu_h+\delta)}{\mu_h}\right) > 0.$$

Thus, $\Delta(\mathcal{R}_0)$ is strictly increasing in $\left]\sqrt{G}, 1\right[$. Moreover, $\Delta(\sqrt{G}) = -B_0B_2 < 0$ and $\Delta(1) = B_1^2 > 0$. Therefore, there is a unique $\mathcal{R}_0^c \in \left]\sqrt{G}, 1\right[$ such that $\Delta(\mathcal{R}_0^c) = 0$. This ends the proof.

Suppose $b = 0$ or $\theta = 0$ then, no bed net is used or no mosquito targets human hosts. That is, all the mosquitoes feed on non-human hosts.

Conversely, if $b \neq 0$ and $\theta \neq 0$, then looking at G as a function of r and solving for r the equation $G(r) = 1$ gives

$$r^* = \frac{1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)}{b\theta\left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)(1-\mu_{v1}b)\right)}. \tag{3.3}$$

Now, we can state the following result about the existence and the number of equilibrium points for (2.1). In order to emphasize on the importance of repellency, we use the repellency rate r as the bifurcation parameter such that the backward bifurcation occurs at the threshold value r^*.

Theorem 3.3. *: The malaria transmission model (2.1) has:*

1. *Assume $r > r^*$ and:*
   a) *Suppose $\sqrt{G} < \mathcal{R}_0 < 1$ and $B_1^2 - 4B_0B_2 = 0$, then system (2.1) has a unique endemic equilibrium.*
   b) *Suppose $\sqrt{G} < \mathcal{R}_0 < 1$ and $B_1^2 - 4B_0B_2 > 0$. Then for $\mathcal{R}_0^c < \mathcal{R}_0 < 1$, system (2.1) has two endemic equilibrium points, and no endemic equilibrium point whenever $\mathcal{R}_0^c > \mathcal{R}_0$.*
   c) *Suppose $\mathcal{R}_0 < \sqrt{G} < 1$, then system (2.1) has no endemic equilibrium.*
   d) *Suppose $\mathcal{R}_0 \geq 1$, then system (2.1) has a unique endemic equilibrium.*
2. *Assume $0 \leq r \leq r^*$ and:*
   a) *Suppose $\mathcal{R}_0 > 1$, then system (2.1) has a unique endemic equilibrium.*
   b) *Suppose $\mathcal{R}_0 \leq 1$, then system (2.1) has no endemic equilibrium.*

Proof : Since G is a strictly decreasing function of δ, we have $G < 1 \Leftrightarrow r > r^*$

1.a) For $\sqrt{G} < \mathcal{R}_0 < 1$ and $B_1^2 - 4B_0B_2 = 0$, we get $G - \mathcal{R}_0^2 < 0$. Thus $\lambda_h^*(r, b) = \frac{-B_1}{2B_2} > 0$ and the proof of 1.*a*) holds.

1.b) For $\sqrt{G} < \mathcal{R}_0 < 1$, since $\Delta(\sqrt{G}) = -B_0B_2 < 0$, $\Delta(\mathcal{R}_0^c) = 0$ and $\Delta(1) = B_1^2 > 0$. We get $\Delta(\mathcal{R}_0) < 0$ whenever $\mathcal{R}_0 < \mathcal{R}_0^c$, then system (2.1) has no endemic equilibrium. On the other hand, $\Delta(\mathcal{R}_0) > 0$ whenever $\mathcal{R}_0^c < \mathcal{R}_0 < 1$, then system (2.1) has two endemic equilibrium points, and 1.*b*) is established.

1.c) For $\mathcal{R}_0 < \sqrt{G} < 1$, we get $B_1 > 0$ and $B_0 > 0$. Then the conclusion for 1.*c*) holds.

1.d) For $r > r^*$ and $\mathcal{R}_0 \geq 1$, we get $B_1 < 0$ and $B_0 \leq 0$. Therefore 1.*d*) is proven.

2.a) For $0 \leq r \leq r^*$ and $\mathcal{R}_0 > 1$, we get $B_0 < 0$. Then system (2.1) has a unique endemic equilibrium.

2.b) For $0 \leq r \leq r^*$ and $\mathcal{R}_0 \leq 1$, we get $\mathcal{R}_0^2 \leq G$ and $B_0 \geq 0$. In this case, system (2.1) has no endemic equilibrium.

Corollary 3.4. *The necessary condition for the existence of backward bifurcation is $\delta > \mu_h$. Moreover, if*

$$\mathcal{R}_0 \leq \min\left\{1, 1/\sqrt{\mu_h + \delta}\right\}, \tag{3.4}$$

then the disease-free equilibrium point E_0 is globally asymptotically stable (GAS). In particular, if the disease-induced death rate δ is small enough such that $\mu_h + \delta \leq 1$, then E_0 is GAS whenever $\mathcal{R}_0 \leq 1$.

Proof : In fact it suffices to prove that, if $\delta \leq \mu_h$, there is no endemic equilibrium when $\mathcal{R}_0 \leq 1$. Indeed, for $\delta \leq \mu_h$, we have $G > 1$ or equivalently $r < r^*$. Moreover, if $\mathcal{R}_0 \leq 1$, condition 2.b) of Theorem 3.3 is satisfied and consequently no endemic equilibrium exists in this case. Hence, the backward bifurcation possibility is ruled out if $\delta \leq \mu_h$. First of all, observe that the assumptions in the second statement of Corollary 3.4 rule out the possibility of the existence of endemic equilibrium points. To prove that statement, Lyapunov-LaSalle techniques are used by considering the following Lyapunov function:

$$L = L(I_h, I_v, S_h, S_v) = \mu_v\mu_hI_h + m_h(\mu_h + \delta)P_hI_v + \mu_v\mu_h\left(S_h - S_h^0 \ln S_h\right) + m_h(\mu_h + \delta)P_h\left(S_v - S_v^0 \ln S_v\right).$$

Direct, yet simple computations show that

$$\begin{aligned}\frac{dL}{dt} &= -\frac{\mu_v\mu_h^2(S_h-S_h^0)^2}{S_h} - \frac{m_hP_h(\mu_h+\delta)\mu_v(S_v-S_v^0)^2}{S_v} + \left[\frac{\mu_v\mu_hm_hP_hS_h^0}{N_h} - m_hP_h(\mu_h+\delta)\mu_v\right]I_v \\ &\quad + \left[\frac{m_hP_h(\mu_h+\delta)m_vP_vS_v^0}{N_h} - \mu_v\mu_h\left(\mu_h+\delta+\gamma_h\frac{S_h^0}{S_h}\right)\right]I_h.\end{aligned}$$

Next, since $\frac{dN_h}{dt} \geq \Lambda_h - (\mu_h+\delta)N_h$, the application of Gronwall lemma yields a lower bound for N_h as follows:

$$N_h \geq \frac{\Lambda_h}{\mu_h+\delta}.$$

Using the above lower bound for N_h and the expression of $\mathcal{R}_0$, straightforward calculations and careful rearrangements lead to

$$\frac{dL}{dt} \leq -\frac{\mu_v\mu_h^2(S_h-S_h^0)^2}{S_h} - \frac{m_hP_h(\mu_h+\delta)\mu_v(S_v-S_v^0)^2}{S_v} + \mu_v\mu_h(\mu_h+\delta)\left(\mu_h+\delta+\gamma_h\right)\left(\mathcal{R}_0^2 - \frac{1}{\mu_h+\delta}\right)I_h.$$

Clearly, if $\delta \leq \mu_h$ and condition (3.4) holds, then, first there is no endemic equilibrium point and secondly, $\mathcal{R}_0^2 \leq 1/(\mu_h+\delta)$ such that $L(S_h, I_h, S_v, I_v)$ is a Lyapunov function for the disease-free equilibrium E_0.
Now, if $\mathcal{R}_0^2 < 1/(\mu_h+\delta)$, then $L(S_h, I_h, S_v, I_v)$ is a strict Lyapunov function and the GAS of E_0 follows. If $\mathcal{R}_0^2 = 1/(\mu_h+\delta)$ the LaSalle Invariance Principle applies easily in this case to L to prove that E_0 is globally attractive in Ω. Hence, E_0 is GAS. The particular case is straightforward since in that case, $\min\left\{1, 1/\sqrt{\mu_h+\delta}\right\} = 1$ and the condition $\mathcal{R}_0 \leq 1$ is enough for the disease-free equilibrium E_0 to be GAS.

Remark 3.5. *It is worth noticeable that for the parameter values given in Table 1, the condition $\mu_h + \delta \leq 1$ is satisfied, while the condition $\mu_h > \delta$ is not. Thus, whenever $\mathcal{R}_0 < 1$ the backward bifurcation may occurs. On the other hand, since the natural mortality rate is a constant for a given population and the disease-induced death rate may vary depending on the health care conditions, there can be a favorable situation where $\mu_h \leq \delta$, and the disease will possibly die out if in addition, the reproduction number $\mathcal{R}_0$ is brought below one.*

3.2. Stability of the endemic equilibrium

Theorem 3.6. *The endemic equilibrium point EE is globally asymptotically stable if $\mathcal{R}_0 > 1$.*

The proof of the GAS of the endemic equilibrium *EE* uses the following instrumental result.

Theorem 3.7. *(Vidyasagar [44], Theorem 3.1). Consider the following C^1 system:*

$$\begin{cases} \dot{x} = f(x) & x \in \mathbb{R}^n \\ \dot{y} = g(x,y) & x \in \mathbb{R}^m, \end{cases} \tag{3.5}$$

with an equilibrium point (x^*, y^*) ie., $f(x^*) = 0$ and $g(x^*, y^*) = 0$.
If x^* is globally asymptotically stable (GAS) in $\mathbb{R}^n$ for the system $\dot{x} = f(x)$, and if y^* is (GAS) in $\mathbb{R}^m$ for the system $\dot{y} = g(x^*, y)$ then (x^*, y^*) is (locally) asymptotically stable for (3.5). Moreover, if all the trajectories of (3.5) are forward bounded, then (x^*, y^*) is GAS for (3.5).

Now, we are going to show that system (2.1) has the triangular structure in Theorem 3.7.
Let $N_v(t) = S_v(t) + I_v(t)$. We have $\frac{dN_v}{dt} = \Lambda_v - \mu_v(b)N_v$, so that system (2.1) is equivalent to

$$\begin{cases} \dot{N}_v = \Lambda_v - \mu_v(b)N_v \\ \dot{S}_h = \Lambda_h - \lambda_h(r,b)S_h + \gamma_h I_h - \mu_h S_h \\ \dot{I}_h = \lambda_h(r,b)S_h - (\mu_h + \gamma_h + \delta)I_h \\ \dot{I}_v = \lambda_v(r,b)\left(N_v(t) - I_v(t)\right) - \mu_v(b)I_v. \end{cases} \tag{3.6}$$

We have $\dot{N}_v = \Lambda_v - \mu_v(b)N_v$. Therefore, $N_v(t) \longrightarrow \frac{\Lambda_v}{\mu_v(b)}$ as $t \longrightarrow +\infty$. Thus, equilibrium $N_v^* = \frac{\Lambda_v}{\mu_v(b)}$ is globally asymptotically stable (GAS) in $\mathbb{R}$ for the system $\dot{N}_v = \Lambda_v - \mu_v(b)N_v$. Note that the endemic equilibrium point for system (2.1) translates to the endemic equilibrium point for system (3.6) which we denote by $\widehat{EE} = (N_v^*, S_h^*, I_h^*, I_v^*)$. Therefore, the proof of the GAS EE for system (2.1) is equivalent to the proof of the GAS of $\widehat{EE}$ for system (3.6). To achieve the latter, if we set

$$x = N_v, \quad y = (S_h, I_h, I_v),$$

then system (3.6) takes the desirable triangular form in Theorem 3.7, with

$$f(x) = \Lambda_v - \mu_v(b)x, \quad g(x,y) = \begin{pmatrix} \Lambda_h - \lambda_h(r,b)S_h + \gamma_h I_h - \mu_h S_h \\ \lambda_h(r,b)S_h - (\mu_h + \gamma_h + \delta)I_h \\ \lambda_v(r,b)\left(N_v(t) - I_v(t)\right) - \mu_v(b)I_v \end{pmatrix}.$$

Moreover, since $x^* = N_v^*$ is GAS for $\dot{x} = f(x)$, the GAS of $\widehat{EE}$ for $\mathcal{R}_0 > 1$ will be established as long as the GAS of the endemic equilibrium $\widetilde{EE} = (S_h^*, I_h^*, I_v^*)$ of the following reduced system (3.7) (corresponding to $\dot{y} = g(x^*, y)$ holds.

$$\begin{cases} \dot{S}_h = \Lambda_h - \lambda_h(r,b)S_h + \gamma_h I_h - \mu_h S_h, \\ \dot{I}_h = \lambda_h(r,b)S_h - (\mu_h + \gamma_h + \delta)I_h, \\ \dot{I}_v = \lambda_v(r,b)\left(N_v^* - I_v(t)\right) - \mu_v(b)I_v. \end{cases} \tag{3.7}$$

One can easily show that the set $\Omega_0 = \left\{(S_h(t), I_h(t), I_v(t)) \in \mathbb{R}_+^3 : S_h(t) + I_h(t) \leq \frac{\Lambda_h}{\mu_h}, I_v(t) \leq \frac{\Lambda_v}{\mu_v(b)}\right\}$ is positively invariant for the flow generated by (3.7) and that, thanks to Theorem (3.3), $\widetilde{EE} = (S_h^*, I_h^*, I_v^*)$ is the unique endemic equilibrium for (3.7) in the interior of Ω_0 when $\mathcal{R}_0 > 1$. On the other hand, since all the trajectories of (3.6) are forward bounded, then according to the Theorem 3.7, the GAS of $\widetilde{EE}$ for system (3.6) is complete if the GAS of $\widetilde{EE}$ for system (3.7) is established. This is done in the following theorem whose proof is given in the Appendix A.

Theorem 3.8. *The endemic equilibrium point $\widetilde{EE}$ of system (3.7) is GAS in the interior of Ω_0 whenever $\mathcal{R}_0 > 1$.*

3.3. Bifurcation analysis

3.3.1. Types of disease free equilibrium

The following result describes the topological type of the disease free equilibrium E_0.

Theorem 3.9. *For system (2.1), the disease free equilibrium $E_0 = \left(S_h^0, I_h^0, S_v^0, I_v^0\right) = \left(\frac{\Lambda_h}{\mu_h}, 0, \frac{\Lambda_v}{\mu_v(b)}, 0\right)$ is:*

(i) an attracting node whenever $\mathcal{R}_0 < 1$;

(ii) a hyperbolic saddle whenever $\mathcal{R}_0 > 1$;

(iii) a saddle-node of co-dimension 1 whenever $\mathcal{R}_0 = 1$.

Proof : See Appendix B.

3.3.2. Backward bifurcation and forward bifurcation analysis

The backward bifurcation possibility highlighted by 1.*b*) in Theorem 3.3 indicates that this phenomenon may occur for the values of $\mathcal{R}_0$ satisfying $\mathcal{R}_0^c < \mathcal{R}_0 < 1$.

Set

$$t_h = m_h P_h(r, b), \quad t_v = m_v P_v(r, b).$$

Recall that

$$\mathcal{R}_0 = \mathcal{R}_0(t_h) = \sqrt{\frac{t_v t_h}{(\mu_h + \gamma_h + \delta)} \frac{\mu_h}{(\mu_v(b))^2} \frac{\Lambda_v}{\Lambda_h}}.$$

Hence, the equation $\mathcal{R}_0(t_h) = 1$ is equivalent to:

$$t_h = \frac{(\mu_h + \gamma_h + \delta)(\mu_v(b))^2 \Lambda_h}{t_v \mu_h \Lambda_v} =: t_h^* \tag{3.8}$$

Let r^* be the unique solution of the equation $G(r) = 1$ given by (3.3). The following theorem describes the role that the repellent effect plays on the complete bifurcation analysis of system (2.1).

Theorem 3.10. *For the malaria transmission model (2.1), the following statements hold:*

1. *Assume* $r^* < 0$, *then system (2.1) exhibits a backward bifurcation at* $\mathcal{R}_0 = 1$.
2. *Assume* $0 < r^* < 1$, *we have:*
   - *a) If* $r > r^*$, *then system (2.1) exhibits a backward bifurcation.*
   - *b) If* $r < r^*$, *then the system exhibits a forward bifurcation at* $\mathcal{R}_0 = 1$.
3. *Assume* $r^* > 1$, *we have:*
   - *a) If* $\delta < \mu_h$, *then the system exhibits a forward bifurcation.*
   - *b) If* $\delta > \frac{m_v \mu_h}{\mu_v(b)} + \mu_h$, *then the system exhibits a backward bifurcation.*

Proof : See Appendix C.

Remark 3.11. *It is important to emphasize that* r^* *is a repellency threshold value not only for the existence of the endemic equilibrium points (see Theorem 3.3) but, also for the existence of various bifurcations (see Theorem 3.10). Moreover, the results of Theorem 3.10 are later discussed and commented in the captions of Figure 1, Figure 2 and Figure 3.*

The results in Theorem 3.10 are numerically confirmed by Figure 1, Figure 2 and Figure 3 below. In all the three figures, we have chosen to plot the infected mosquito component I_v^* of the endemic equilibrium point versus the reproduction number $\mathcal{R}_0$. Of course the illustrations are similar if one chooses to plot I_h^* (or any other component of the endemic equilibrium) against $\mathcal{R}_0$.

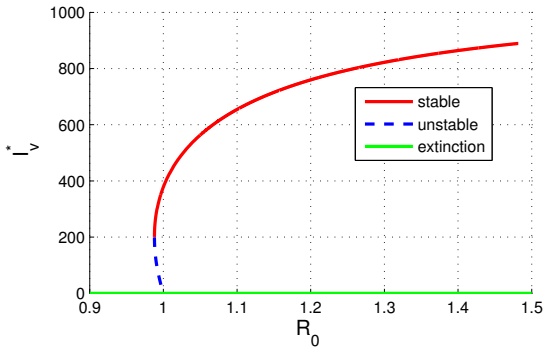

Figure 1: Illustration of the backward bifurcation phenomenon: The selected parameters are: $\delta = 1.58 \times 10^{-4}$, $\mu_h = \dfrac{1}{50 \times 365}$, $b = 0.75$, $\dfrac{m_v \mu_h}{\mu_v(b)} + \mu_h = 1.3944 \times 10^{-4} < \delta$, $r = 0.9$. Other parameters are given in Table (1). With these parameters, $r^* = -43.9113 < 0$, so that the backward bifurcation occurs. This result exemplifies the condition 1.) in Theorem 3.10. One can observe in this figure that, high value of repellency leads to large value of the threshold $\mathcal{R}_0^c \approx 1$ for the occurrence of backward, making the control of malaria easier to achieve.

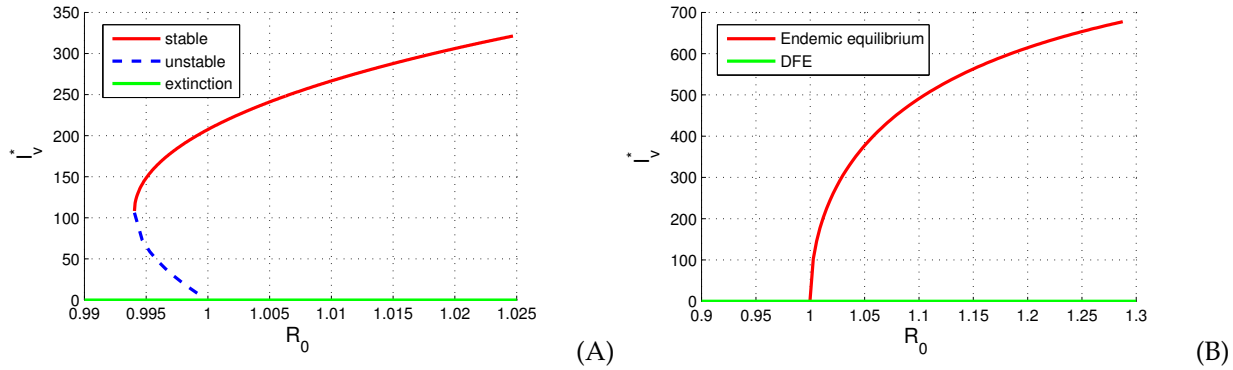

Figure 2: Illustration of the backward and forward bifurcation phenomena: (A) Backward bifurcation with the chosen parameters: $\delta = 1.28729 \times 10^{-4}$, $\mu_h = \dfrac{1}{50 \times 365}$, $b = 0.9$, $\dfrac{m_v \mu_h}{\mu_v(b)} + \mu_h = 1.2873 \times 10^{-4} > \delta > \mu_h$, $r = 0.95$. (B) Forward bifurcation with the chosen parameters: $\delta = 1.28729 \times 10^{-4}$, $\mu_h = \dfrac{1}{50 \times 365}$, $b = 0.9$, $\dfrac{m_v \mu_h}{\mu_v(b)} + \mu_h = 1.2873 \times 10^{-4} > \delta > \mu_h$, $r = 3 \times 10^{-4}$. The remaining parameters are given in Table 1. The computed value of r^* is $r^* = 4.81 \times 10^{-4}$, then we have, $0 < r^* < 1$. (A) $r = 0.95$, thus we have $r > r^*$, the backward bifurcation is occurs. (B) $r = 3 \times 10^{-4}$, we have $r < r^*$, the forward bifurcation is occurs. These results illustrate the items 2.*a*) and 2.*b*) in Theorem 3.10, respectively. The threshold value $\mathcal{R}_0^c \approx 0.992$ for the occurrence of backward bifurcation can be very high in this figure, making the control of malaria can be easier to achieve.

4. Sensitivity analysis and simulations

4.1. Sensitivity analysis of the basic reproduction number

We assess the impact of parameters of the bed net model (2.1) on the reproduction number $\mathcal{R}_0$, by computing the elasticity indexes of $\mathcal{R}_0$ with respect to parameter values given in Table 1. According to the approach proposed in [9, 31, 32, 36], the elasticity index of $\mathcal{R}_0$ with respect to a parameter p, where p is any of the parameters in Table 1 reflected in the expression of $\mathcal{R}_0$, is given by

$$\frac{\partial \mathcal{R}_0}{\partial p} \times \frac{p}{\mathcal{R}_0}.$$

Since these indexes quantify the ratio of relative changes on $\mathcal{R}_0$ in response to corresponding changes in the parameters, they can identify critical parameters for disease control. This approach states that the reproduction

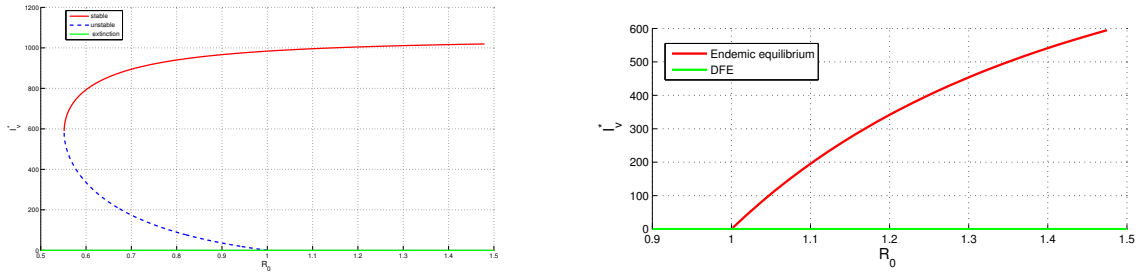

Figure 3: Illustration of the backward and Forward bifurcation phenomena: Backward bifurcation (Left figure) with the following parameters: $\delta = 1\times10^{-3}$, $\mu_h = \frac{1}{50\times365}$, $b = 0.75$, $\frac{m_v\mu_h}{\mu_v(b)} + \mu_h = 1.3944\times10^{-4} < \delta$, $r = 0.9$. Forward bifurcation (Right figure) with parameters: $\delta = 1\times10^{-5}$, $\mu_h = \frac{1}{50\times365}$, $b = 0.75$, $\mu_h > \delta$, $r = 0.9$. The remaining parameters are given in Table (1). With the latter set of parameters, $r^* = 2.008$. We have $r^* > 1$ and $\mu_h > \delta$, so that forward bifurcation is occurs. This result exemplifies the condition 3.*a*) in Theorem 3.10. For the (Left figure), The computing of r^* give $r^* = 2.3650$. Thus we have $r^* > 1$ and $\frac{m_v\mu_h}{\mu_v(b)} + \mu_h < \delta$, the backward bifurcation is occurs. This result illustrates the condition 3.*b*) in Theorem 3.10. The threshold value $\mathcal{R}_0^c = 0.55$, for the occurrence of backward bifurcation can be very low, suggesting that the control of malaria can be very difficult to achieve. On the other hand, even for high value of the repellency rate r, if the disease death rate δ is greater than the natural human mortality rate μ_h and $r^* > 1$, it becomes very difficult to control malaria.

number is most sensitive to the parameter with the largest elasticity index value and least sensitive to the parameter with the smallest elasticity index value. Table 2 displays the elasticity indexes of $\mathcal{R}_0$ to the 12 (twelve) parameters, arranged in decreasing magnitude order and hence decreasing sensitivity. As expected, the reproduction number is most sensitive to the bed-net coverage parameter b with an elasticity index of -1.33. It is also highly sensitive to the probability that a vector is repelled by the insecticide (or mechanically blocked by the net) r, and the probability that a mosquito targets a human host θ. Qualitatively, $\mathcal{R}_0$ decreases by 13.3% for an increase in bed-net coverage of 10%, $\mathcal{R}_0$ is reduced by 8.5% when repellency effect is increased by 10%, a 10% increase of the natural mosquito mortality rate decreases $\mathcal{R}_0$ by 5.55%; and a 10% increase of the probability that mosquito targets a human host increases $\mathcal{R}_0$ by 9.3%.

The public health implication of these results is that the use of insecticide-treated nets with high repellency and lethal effects, and of course vector control are important steps to take for the control of malaria.

Parameter	b	θ	r	μ_{v0}	Λ_h	Λ_v	m_v	m_h	μ_h	γ_h	μ_{v1}	δ
Elasticity	-1.33	0.93	-0.85	-0.55	-0.5	0.5	0.5	0.5	0.49	-0.49	-0.48	-0.001

Table 2: Elasticity indexes of the basic reproduction number $\mathcal{R}_0$.

4.2. **Impact of combined PTNs effects on the reproduction number**

Here, we assess both theoretically and numerically the role of PTNs usage on the reproduction number $\mathcal{R}_0$.

4.2.1. *Impact of PTNs on the basic reproduction number*

Let's recall that reproduction number

$$\mathcal{R}_0 = \sqrt{\frac{m_v m_h P_v(r,b) P_h(r,b)}{(\mu_h+\gamma_h+\delta)} \frac{\mu_h}{(\mu_v(b))^2} \frac{\Lambda_v}{\Lambda_h}}.$$

For $\mathcal{R}_0 \neq 0$, we have $P_v(r,b) \neq 0$ and $P_h(r,b) \neq 0$, thus

$$\frac{\partial \mathcal{R}_0}{\partial b} = \frac{m_v m_h \mu_h \Lambda_v}{2(\mu_h+\gamma_h+\delta)\Lambda_h \mathcal{R}_0 (\mu_v(b))^3}\left[\left(\frac{\partial P_v(r,b)}{\partial b}P_h(r,b) + \frac{\partial P_h(r,b)}{\partial b}P_v(r,b)\right)\mu_v(b) - 2\mu_{v1}P_v(r,b)P_h(r,b)\right].$$

$$\frac{\partial \mathcal{R}_0}{\partial r} = \frac{m_v m_h \mu_h \Lambda_v}{2(\mu_h+\gamma_h+\delta)\Lambda_h \mathcal{R}_0 (\mu_v(b))^2}\left(\frac{\partial P_v(r,b)}{\partial r}P_h(r,b) + \frac{\partial P_h(r,b)}{\partial r}P_v(r,b)\right).$$

Since, thanks to Theorem 2.1, $\frac{\partial P_h(r,b)}{\partial b}$, $\frac{\partial P_v(r,b)}{\partial b}$, $\frac{\partial P_v(r,b)}{\partial r}$, $\frac{\partial P_v(r,b)}{\partial r} \leq 0$, we conclude that

$$\frac{\partial \mathcal{R}_0}{\partial b} < 0, \ \frac{\partial \mathcal{R}_0}{\partial r} < 0.$$

We have shown the following important result and illustrated it Figure 4 and Figure 5 below.

Theorem 4.1. *The ITNs/PTNs lethal and repellent effects, considered either solely or combined, have a positive impacts on malaria control by decreasing its reproduction number $\mathcal{R}_0$.*

The Figure 4 shows the minimum level of PTNs feature required to contain malaria disease: In panel (i) the repellency rate is fixed at $r = 0.75$ and bed-net usage rate varies. One can see that the minimum level of bed net coverage b is 70% ($b = 0.70$), that is the sufficient bed net lethal effect rate required to bring $\mathcal{R}_0$ below one. Similarly in panel (ii), for the fixed value of the bed net coverage $b = 0.75$, one needs at least 65% of the repellent effect to bring $\mathcal{R}_0$ under unity. More precisely, from Figure 4 (i), if the bed net used in a given population can repel up to 75% ($r = 0.75$), then to contain malaria, the requirement is that 70% ($b = 0.70$) of this population should be protected by these bed-nets. On the other hand, from Figure 4 (ii), if 75% ($b = 0.75$) of the population used bed-nets, then these bed-nets should repel approximately 65%($r = 0.65$) of the mosquitoes in order to offer full protection against malaria. Furthermore, remark that as the bed-net usage or it its repellent effect decreases, $\mathcal{R}_0$ increases. Moreover, looking at the concavities of the plots in Figure 4, one realizes that $\mathcal{R}_0$ decreases faster as a function of bed net lethal effect (concave down) than it does as a function a of bed net repellent effect (concave up). This shows that, the influence of the lethal effect dominates the influence of repellency.
Figure 5 is the bifurcation diagram (contour plot) of $\mathcal{R}_0$ in the $(b-r)$ space by combining the plots in Figure 4. It further highlights that, if the lethal is below 0.70, irrespective of the value of the repellent effect rate, $\mathcal{R}_0$ will never be brought below one, and consequently, malaria will never be eliminated. On the other hand, if the repellent rate r is less 0.05, regardless the rate of the bed net lethal effect, $\mathcal{R}_0$ will remain greater that one, and malaria will never be controlled. Therefore, any couple (b,r) which is not in the set $[0,0.7]\times[0,1] \cup [0,1]\times[0,0.05]$ can be suitably chosen such that $\mathcal{R}_0$ is less that one (that is, malaria is eliminated).

4.2.2. Impact of the use of pyrethroid-treated bed net with single PTN effect on model (2.1)

The assumption in this paragraph is that the PTNs can either repel or kill the mosquitoes and not both. Note that this assumption leads to the new expressions of the probabilities P_h and P_v determined accordingly below, and that the equations and basic properties of model (2.1) remain unchanged. First, we consider that the bed nets can only kill, but do not repel the mosquitoes. Thus, the model takes into account the lethal effect of ITNs only, so that the contact rate between mosquitoes and humans depends only on the parameter b and becomes $P_h(b) = \theta(1-b)$.

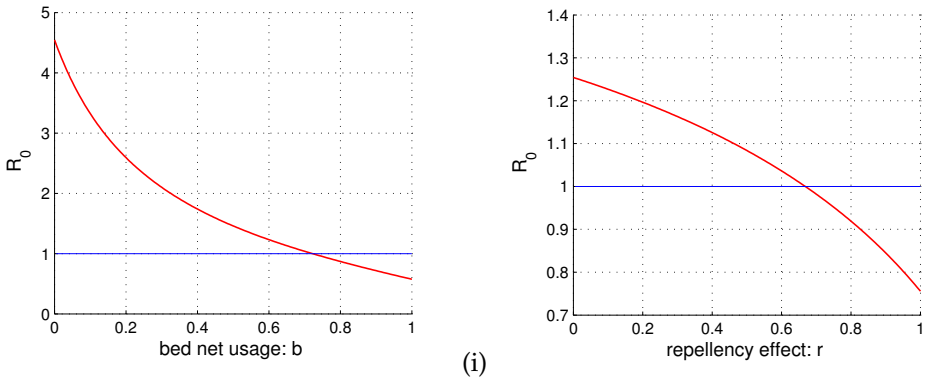

Figure 4: The minimum level of PTNs coverage required to contain malaria disease. $r = 0.75$ for panel (i), $b = 0.75$ for panel (ii) and the remaining parameters as in Table 1. The only effect of bed net efficacy or the repellency efficiency can help to bring the reproduction under the elimination threshold one. Bed net utilization efficacy does it faster than repellency.

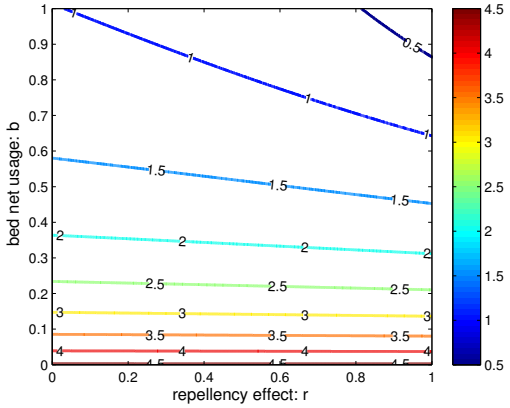

Figure 5: Contour plot of the reproduction number Versus bed net usage (b) and repellency (r). The remaining parameters are as in Table 1. We note that, for a fix value of repellency effect, the reproduction number $\mathcal{R}_0$ decreases when bed net usage increases. For a fix value of bed net usage, we also note that as the repellency effect decreases, $\mathcal{R}_0$ increases and vice versa.

In this particular setting, recalling the probability $(1-\theta)$ that mosquitoes target non-human hosts, it comes that $P_v(b) = \theta(1-b) + 1 - \theta = 1 - b\theta$.

Consequently, the reproduction number depends only on b and not on r and takes the form:

$$\mathcal{R}_0(b) = \sqrt{\frac{m_v m_h P_v(b) P_h(b)}{(\mu_h + \gamma_h + \delta)} \frac{\mu_h}{(\mu_v(b))^2} \frac{\Lambda_v}{\Lambda_h}}.$$

For $\mathcal{R}_0(b) \neq 0$, we have $\theta \neq 0$ and,

$$\frac{d\mathcal{R}_0(b)}{db} = \frac{m_v m_h \mu_h \Lambda_v}{2(\mu_h + \gamma_h + \delta)\Lambda_h (\mu_v(b))^3 \mathcal{R}_0(b)} \left[\left(\frac{dP_v(b)}{db} P_h(b) + \frac{dP_h(b)}{db} P_v(b) \right) \mu_v(b) - 2\mu_{v1} P_v(b) P_h(b) \right].$$

We have $\dfrac{dP_h(b)}{db} = \dfrac{dP_v(b)}{db} = -\theta < 0$ and conclude that $\dfrac{d\mathcal{R}_0(b)}{db} < 0$.

Secondly, we consider the model with bed nets that repel only and do kill mosquitoes. That is the lethal effect of bed net wanes completely (i.e. $\mu_b = \mu_{v1} b = 0$). The reproduction number can be written:

$$\mathcal{R}_0(r) = \sqrt{\frac{m_v m_h P_h(r,b)}{(\mu_h + \gamma_h + \delta)} \frac{\mu_h}{(\mu_{v0})^2} \frac{\Lambda_v}{\Lambda_h}}.$$

With $\mathcal{R}_0(r) \neq 0$ and $b \neq 0$, then we have $\theta \neq 0$ and the derivative of $\mathcal{R}_0(r)$ is

$$\frac{d\mathcal{R}_0(r)}{dr} = \frac{m_v m_h \mu_h \Lambda_v}{2(\mu_h + \gamma_h + \delta)\Lambda_h(\mu_{v0})^2 \mathcal{R}_0(r)} \frac{b\theta\left(\theta - 1 - \theta\mu_{v1}b\right)}{\left(1 - r\theta b(1 - \mu_{v1}b)\right)^2}.$$

Thus $\frac{d\mathcal{R}_0(r)}{dr} < 0$. These results are summarized in Theorem 4.2 below and illustrated in Figure 6:

Theorem 4.2. *The utilization of PTNs with either the lethal or the repellent effect has a positive impact on the control of malaria by decreasing the reproduction number.*

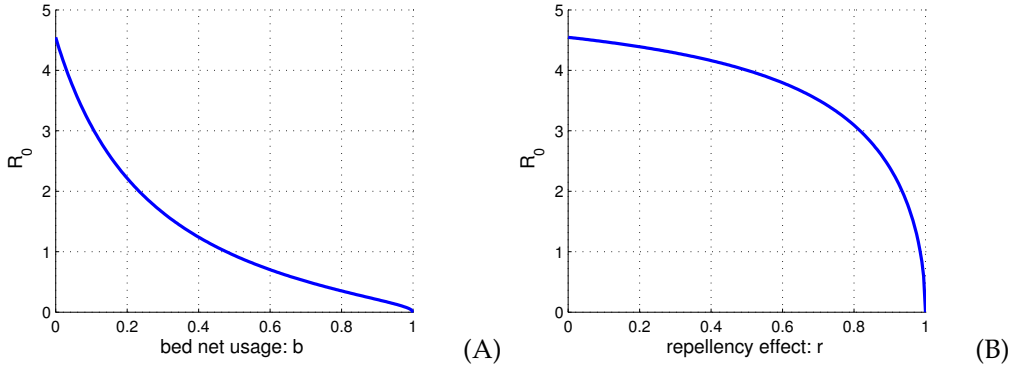

Figure 6: (A): $\mathcal{R}_0$ Versus bed net lethal effect b and full repellency (i.e. r= 1). (B): $\mathcal{R}_0$ Versus bed net repellent effect r with complete bed net usage (i.e. $b = 1$). Other parameters are given in Table 1. Note that as the bed net usage decreases, $\mathcal{R}_0(b)$ increases and vice-versa. This figure highlights the relative prominence of bed net usage rate to the repellent effect, since the reproduction number drops faster and earlier below one for variable lethal effect (Figure 6(A)) than it does for variable repellency effect (Figure 6(B)).

One can remark that $\mathcal{R}_0(b)$ is decreasing and concave down (fast decrease), while $\mathcal{R}_0(r)$ is decreasing and concave up (slow decrease), indicating that $\mathcal{R}_0$ decreases faster as a function of the lethal effect b as compared to its reduction with respect to the repellent effect. This suggests that the utilization of ITNs with the high lethal effect is better than those with high repellent effect.

4.2.3. Neglecting repellency underestimates the basic reproduction number and hinders malaria elimination

In this paragraph, we investigate whether the explicit involvement of repellent effect of pyrethroid in a dynamical model of malaria overestimates or underestimates the basic reproduction number. To do, we denote the Agusto et al.'s model (1.1) reproduction number by $\mathcal{R}_0^{(a)}$ and compare it with the reproduction number $\mathcal{R}_0$ obtained in our model (2.1). This comparison is possible only if the two models share some basic and relevant parameters. Since model (1.1) did not considered the non-human host population, we fix (for comparison purpose) $\theta = 1$ in our PTNs' model (2.1). Let's recall that reproduction number in model (1.1)

$$\mathcal{R}_0^{(a)} = \sqrt{\frac{m_v m_h \beta(b)\beta(b)}{(\mu_h + \gamma_h + \delta)} \frac{\mu_h}{(\mu_v(b))^2} \frac{\Lambda_v}{\Lambda_h}},$$

where,

$$\beta(b) = \beta_{max} - b(\beta_{max} - \beta_{min}); \quad 0 \leq b \leq 1$$

The simulations in Figure 7 depicts the comparison between the reproduction number for our PTNs model (2.1) and the reproduction number for Agusto et al.' model (1.1). They suggest that the value of the reproduction

number can be underestimated if the repellent effect of the PTNs is neglected. The consequence of this is that the elimination of malaria might be more difficult to achieve, if the PTNs with low/zero repellent rate are distributed for utilization to a given malaria affected population.

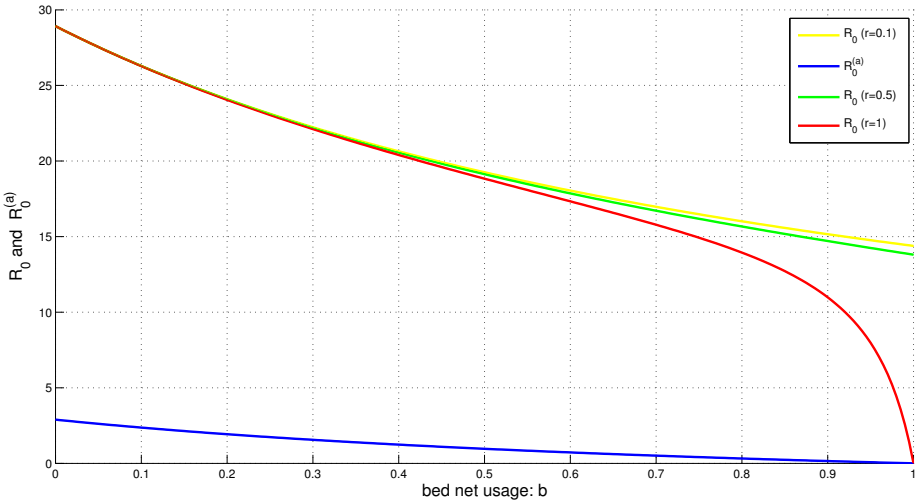

Figure 7: Neglecting repellency underestimates the reproduction number: $\mathcal{R}_0^{(a)}$ and $\mathcal{R}_0$ versus the PTNs lethal effect b, with different value of repellency rate r. All shared parameters are those from Agusto et al. model [1]. Repellency regulates the reproduction number and the lesser value for the repellency r, the larger the gap between the reproduction number without repellency effect $\mathcal{R}_0^{(a)}$ and the reproduction number with repellency $\mathcal{R}_0$.

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4.2.4. Numerical illustrations of the impacts of bed net usage on contact rates

In this sub-subsection, we present some simulations to illustrate the impacts of bed net coverage b and repellent effect r on the contact rates between mosquitoes and humans. Parameters values as in Table 1. These impacts are shown and commented the captions of Figure 8 and Figure 9 below. The illustrations are presented for $P_h(r, b)$, but the reader should notice the conclusions hold for the plot and contour plot of $P_v(r, b)$. For that reason we did not display them in the manuscript. The common feature of these figures is that the contact rates $P_h(r, b)$ and $P_v(r, b)$ are decreasing functions of the lethal effect or the repellency effect.

5. Conclusion and discussions

Pyrethroid-treated bed nets protect individuals against malaria by blocking and repelling mosquitoes, and they protect the community by killing mosquitoes thanks to lethal effect. We have proposed and analyzed a dynamical PTNs model for malaria transmission, in which lethal and excito-repellency/deterrence effects are incorporated and their role on the long run of the system studied. The results obtained have revealed the existence of a backward bifurcation for certain parameter values which implies that the reduction of $\mathcal{R}_0$ below unity alone is not enough to mitigate malaria evolution. Therefore, additional control strategies such as indoor residual spraying and treatment might be necessary to reduce malaria burden and control the disease. Furthermore, the occurrence

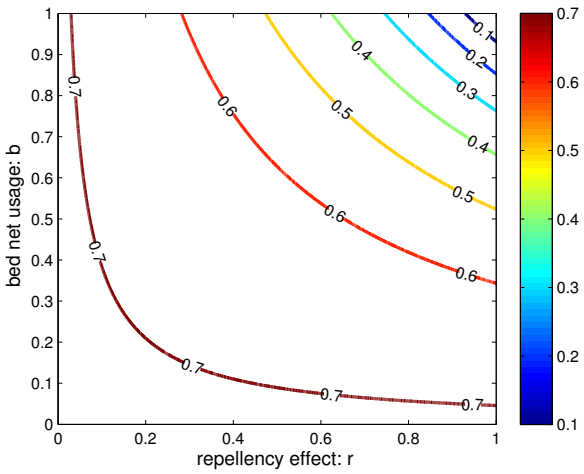

Figure 8: Contour plot of the contact rate $P_h(r, b)$ between mosquitoes and humans. For a fixed value of repellency effect, the contact rate decreases when bed net usage increases. For a fix value of bed net usage, we also note that as the repellency effect decreases, the contact rate increases and vice versa.

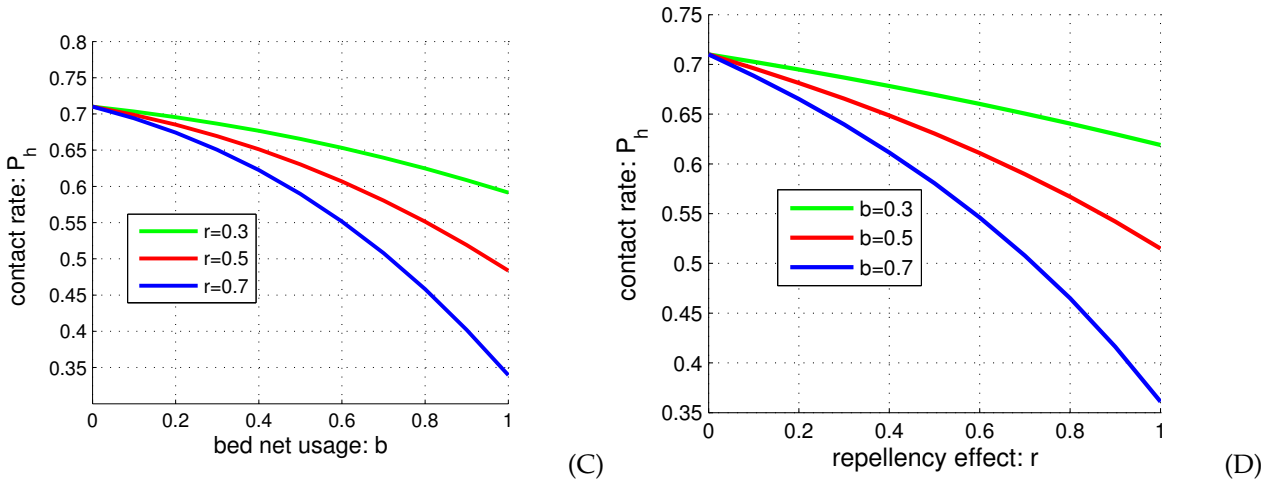

Figure 9: Impacts of lethal and repellent effects on the malaria contact rates. (C): $P_h(r, b)$ versus the bed net usage efficacy b and three (mild-moderate-high) levels of repellent effect. (D)-$P_h(r, b)$ versus the bed net repellent effect r and and three (mild-moderate-high) levels of bed net utilization efficacy.

of a backward bifurcation has been shown to depend on the range of the repellent effect rate. Precisely, we have computed the repellency threshold value r^* necessary to study the existence of both the endemic equilibrium points and backward bifurcation when the basic reproduction number $\mathcal{R}_0$ is less than one.

We have shown that the reproduction number is highly sensitive to the bed-net coverage parameter (b) and the repellency parameter (r), and demonstrated that although the disease contact rates are decreasing functions of the bed net usage b and repellency effect r, they are more sensitive to b than r. The global asymptotic stability of the endemic equilibrium whenever $\mathcal{R}_0 > 1$ has been established, using the geometric approach. We have demonstrated that the disease free equilibrium is a saddle-node of co-dimension 1 when $\mathcal{R}_0 = 1$, and established that the PTNs utilization (lethal or repellent or combined effect) has a positive impact in reducing the reproduction number.

This allowed us to conclude that the (PTNs) utilization decreases reproduction number, thus reducing the disease burden and help to control malaria. We have shown that the elimination of malaria might be more difficult to achieve, if bed nets with low/zero repellent rate is distributed to the population. As a whole, the following results summarize the theoretical and numerical analyses of our model:

(i) The consideration of bed net repellency enriches the dynamics of malaria evolution by making disease contact rates highly nonlinear functions of lethal and repellent effects.

(ii) Bed net repellency is one of the causes of backward bifurcation, which impedes the classical requirement that, bringing the reproduction number below unity is sufficient to control malaria.

(iii) Both low bed net repellency and high disease-induced mortality rates can hinder the efficient control of malaria.

(iv) The utilization of bed nets with high lethal effect is better than bed nets with high repellency effect.

(v) Both bed net repellency and lethal effects decrease the disease contact rates, and the lethal effect rate does it faster than the repellency effect.

(vi) Bed net repellency effect regulates the disease burden and its negligence might underestimates that burden.

(vii) Malaria dies out completely if the malaria-induced death rate is less than the natural mortality rate or the sum of these two mortality rates is less than unity.

Our recommendation is that that public health authorities provide pyrethroid-treated bed nets with high lethal and repellent effects and organize the sensitization campaign to inform population to use them properly.

Although our relatively simple model (2.1) with the new nonlinear bed nets contact rates functions displays rich and complex dynamical behaviors, we acknowledge that the dynamics of PTNs might be more complicated if some modeling hypotheses are relaxed and many other relevant disease features taking into consideration. For instance, if the assumption that mosquito PTN-induced mortality is a linear function of bed net usage is relaxed, one should expect the mosquito mortality rate to become a highly nonlinear function of bed net lethal effect b, and consequently, a more difficult and richer dynamics of the disease evolution to studied. This complication will be empowered if additionally, that new model explicitly incorporates the repellent effect r, making the PTN-induced mortality rate a nonlinear function of r as well. On the other hand, if one considers explicitly the mosquito preference [2, 38] to our modeling framework in this manuscript, the resulted model might more harder to study and the result expectations might not be predictable. All these new modeling settings might not lead to monotone disease contact rates and constitute to the new directions of our work that need to be investigated and on which we are already working.

Acknowledgments

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Appendices

Appendix A: Proof of Theorem 3.8

We use the geometric approach to establish the global asymptotic stability of $\widetilde{EE}$. First, note that Ω_0 is simply connected in $\mathbb{R}^3_+$ and system (3.7) has a unique endemic equilibrium in the interior of Ω_0 whenever $\mathcal{R}_0 > 1$. Moreover, the instability of the disease-free equilibrium implies the uniform persistence of system (3.7) see [19], i.e. there exists a constant $c > 0$ such that any solution $x(t, x_0) = (S_h(t), I_h(t), I_v(t))$ of (3.7) with the initial condition $x_0 = (S_h(0), I_h(0), I_v(0))$ in the interior of Ω_0 satisfies the inequality

$$\min\left\{\lim_{t\to+\infty} inf S_h(t), lim_{t\to+\infty} inf I_h(t), lim_{t\to+\infty} inf I_v(t)\right\} > c.$$

The uniform persistence together with boundedness of Ω_0 is equivalent to the existence of a compact absorbing set K in the interior of Ω_0 [23]. Therefore, it remains to find conditions for which the Bendixson's criterion are verified. The Jacobian matrix J of System (3.7) is

$$J = \begin{pmatrix} -\frac{\partial \lambda_h}{\partial S_h}S_h - \lambda_h - \mu_h & -\frac{\partial \lambda_h}{\partial I_h}S_h + \gamma_h & -\frac{\partial \lambda_h}{\partial I_v}S_h \\ \frac{\partial \lambda_h}{\partial S_h}S_h + \lambda_h & \frac{\partial \lambda_h}{\partial I_h}S_h - (\gamma_h + \mu_h + \delta) & \frac{\partial \lambda_h}{\partial I_v}S_h \\ \frac{\partial \lambda_v}{\partial S_h}(N_v^* - I_v) & \frac{\partial \lambda_v}{\partial I_h}(N_v^* - I_v) & -\lambda_v - \mu_v \end{pmatrix}.$$

For a 3×3 matrix

$$M = \begin{pmatrix} a_{11} & a_{12} & a_{13} \\ a_{21} & a_{22} & a_{23} \\ a_{31} & a_{32} & a_{33} \end{pmatrix},$$

the second additive compound matrix is given by

$$M^{[2]} = \begin{pmatrix} a_{11} + a_{22} & a_{23} & -a_{13} \\ a_{32} & a_{11} + a_{33} & a_{12} \\ -a_{31} & a_{21} & a_{22} + a_{33} \end{pmatrix}.$$

Hence, the second additive compound matrix $J^{[2]}$ of J is

$$J^{[2]} = \begin{pmatrix} -b_{11} & \frac{\partial \lambda_h}{\partial I_v}S_h & \frac{\partial \lambda_h}{\partial I_v}S_h \\ \frac{\partial \lambda_v}{\partial I_h}(N_v^* - I_v) & -b_{22} & -\frac{\partial \lambda_h}{\partial I_h}S_h + \gamma_h \\ -\frac{\partial \lambda_v}{\partial S_h}(N_v^* - I_v) & \frac{\partial \lambda_h}{\partial S_h}S_h + \lambda_h & -b_{33} \end{pmatrix},$$

with,

$$\begin{aligned} b_{11} &= \gamma_h + 2\mu_h + \delta + \lambda_h + \left(\frac{\partial \lambda_h}{\partial S_h} - \frac{\partial \lambda_h}{\partial I_h}\right)S_h, \\ b_{22} &= \mu_h + \mu_v(b) + \lambda_h + \lambda_v + \frac{\partial \lambda_h}{\partial S_h}S_h, \\ b_{33} &= \gamma_h + \mu_h + \delta + \mu_v(b) + \lambda_v - \frac{\partial \lambda_h}{\partial I_h}S_h. \end{aligned}$$

Let $x = (S_h, I_h, I_v)$. Choose now the matrix $P = P(S_h, I_h, I_v) = \text{diag}(1, I_h/I_v, I_h/I_v)$ and define the matrix P_f by

$$(P_{ij}(x))_f = \left(\frac{\partial P_{ij}(x)}{\partial x}\right)^T . f(x) = \nabla_{P_{ij}(x)} . f(x).$$

Then, $P_f P^{-1} = \text{diag}\left(0, \frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v}, \frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v}\right)$, and the matrix $N = P_f P^{-1} + PJ^{[2]}P^{-1}$ can be re-written in the block form

$$N = \begin{pmatrix} N_{11} & N_{12} \\ N_{21} & N_{22} \end{pmatrix},$$

where,

$$N_{11} = -\gamma_h - 2\mu_h - \delta - \lambda_h - \left(\frac{\partial \lambda_h}{\partial S_h} - \frac{\partial \lambda_h}{\partial I_h}\right) S_h, \quad N_{12} = \begin{pmatrix} \frac{S_h I_v}{I_h}\frac{\partial \lambda_h}{\partial I_v} & \frac{S_h I_v}{I_h}\frac{\partial \lambda_h}{\partial I_v} \end{pmatrix},$$

$$N_{21} = \begin{pmatrix} \frac{\partial \lambda_v}{\partial I_h}\frac{I_h}{I_v}(N_v^* - I_v), & -\frac{\partial \lambda_v}{\partial S_h}\frac{I_h}{I_v}(N_v^* - I_v) \end{pmatrix}, \quad N_{22} = \begin{pmatrix} \frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v} - b_{22} & -\frac{\partial \lambda_h}{\partial I_h} S_h + \gamma_h \\ \frac{\partial \lambda_h}{\partial S_h} S_h + \lambda_h & \frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v} - b_{33} \end{pmatrix}.$$

Now, define a vector norm $|.|$ in $\mathbb{R}^3_+$ by

$$|(x, y, z)| = \max\{|x|, |y| + |z|\}.$$

Let $\sigma(.)$ denote the Lozinskii measure with respect to the above defined norm given by

$$\sigma(N) = \lim_{h \to 0^+} \frac{|I + hN| - 1}{h}.$$

Using a similar argument as in [39], we have the following estimate

$$\sigma(N) \leq \sup\{g_1, g_2\} = \sup\{\sigma_1(N_{11}) + |N_{12}|, \sigma_1(N_{22}) + |N_{21}|\},$$

where $|N_{21}|$, $|N_{12}|$ are matrix norms with respect to the L^1 vector norm defined for a generic matrix $A = (a_{ij})$, $|A| = \max_{1 \leq k \leq n} \sum_{j=1}^{n} |a_{jk}|$ and σ_1 is the Lozinskii measure of A with respect to that L^1 norm. Since N_{11} is scalar, its Lozinskii measure with respect to any norm in $\mathbb{R}_+$ is equal to N_{11} . Therefore,

$$\sigma_1(N_{11}) = -\gamma_h - 2\mu_h - \delta - \lambda_h - \left(\frac{\partial \lambda_h}{\partial S_h} - \frac{\partial \lambda_h}{\partial I_h}\right) S_h,$$

$$\begin{aligned} \sigma_1(N_{22}) &= \max\left\{\frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v} - b_{22} + \frac{\partial \lambda_h}{\partial S_h} S_h + \lambda_h, \quad \frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v} - b_{33} - \frac{\partial \lambda_h}{\partial I_h} S_h + \gamma_h\right\}, \\ &= \max\left\{\frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v} - \mu_h - \mu_v(b) - \lambda_v, \quad \frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v} - b_{33} - \delta - \mu_h - \mu_v(b) - \lambda_v\right\}, \\ &= \frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v} - \mu_h - \mu_v(b) - \lambda_v, \end{aligned}$$

and

$$|N_{12}| = \frac{S_h I_v}{I_h}\frac{\partial \lambda_h}{\partial I_v}, \quad |N_{21}| = \left(\frac{\partial \lambda_v}{\partial I_h} - \frac{\partial \lambda_v}{\partial S_h}\right)\frac{I_h}{I_v}(N_v^* - I_v).$$

Thus,

$$g_1 = -\gamma_h - 2\mu_h - \delta - \lambda_h - \left(\frac{\partial \lambda_h}{\partial S_h} - \frac{\partial \lambda_h}{\partial I_h}\right) S_h + \frac{S_h I_v}{I_h}\frac{\partial \lambda_h}{\partial I_v}, \tag{5.1}$$

and

$$g_2 = \frac{\dot{I_h}}{I_h} - \frac{\dot{I_v}}{I_v} - \mu_h - \mu_v(b) - \lambda_v + \left(\frac{\partial \lambda_v}{\partial I_h} - \frac{\partial \lambda_v}{\partial S_h}\right)\frac{I_h}{I_v}(N_v^* - I_v). \tag{5.2}$$

From system (3.7), we have

$$\lambda_h \frac{S_h}{I_h} = \frac{\dot{I}_h}{I_h} + (\gamma_h + \mu_h + \delta), \tag{5.3}$$

and

$$\frac{\lambda_v}{I_v}(N_v^* - I_v) = \frac{\dot{I}_v}{I_v} + \mu_v(b), \tag{5.4}$$

On the other hand, we know that

$$I_v \frac{\partial \lambda_h}{\partial I_v} = \lambda_h. \tag{5.5}$$

Thus,

$$\frac{S_h I_v}{I_h} \frac{\partial \lambda_h}{\partial I_v} = \lambda_h \frac{S_h}{I_h}, \tag{5.6}$$

and the substitution of (5.3) into (5.1) and (5.4) into (5.2) yields

$$g_1 = \frac{\dot{I}_h}{I_h} - \mu_h - \lambda_h - \left(\frac{\partial \lambda_h}{\partial S_h} - \frac{\partial \lambda_h}{\partial I_h}\right) S_h, \quad g_2 = \frac{\dot{I}_h}{I_h} - \mu_h - \lambda_v + \left[\left(\frac{\partial \lambda_v}{\partial I_h} - \frac{\partial \lambda_v}{\partial S_h}\right)\frac{I_h}{I_v} - \frac{\lambda_v}{I_v}\right](N_v^* - I_v).$$

Now, after some computations it comes that

$$g_1 = \frac{\dot{I}_h}{I_h} - \mu_h - \lambda_h, \tag{5.7}$$

and

$$g_2 = \frac{\dot{I}_h}{I_h} - \mu_h - \lambda_v. \tag{5.8}$$

The relations in (5.7) and (5.8) imply

$$\sigma(N) \leq \frac{\dot{I}_h}{I_h} - \mu_h. \tag{5.9}$$

The above relations hold alongside each solution $(S_h(t), I_h(t), I_v(t))$ to system (3.7) corresponding to the initial condition $(S_h(0), I_h(0), I_v(0)) \in K$, with K being a compact and absorbing in ω_0 shown earlier. Moreover, $\forall\ t > T$,

$$\frac{1}{t}\int_0^t \sigma(N)\,\mathrm{d}s \leq \frac{1}{t}\int_0^T \sigma(N)\,\mathrm{d}s + \frac{1}{t} ln \frac{I_h(t)}{I_h(T)} - \mu_h \frac{t-T}{t}.$$

Therefore,

$$\bar{q} = \lim_{t \to +\infty} \sup \sup_{x_0 \in K} \frac{1}{t}\int_0^t \sigma(N(x(s, x_0)))\,\mathrm{d}s \leq -\mu_h < 0.$$

This proves the GAS of $\widetilde{EE}$.

Appendix B: Proof of Theorem 3.9

We have $\dot{N}_v = \Lambda_v - \mu_v(b)N_v$. Therefore, $N_v(t) \longrightarrow \dfrac{\Lambda_v}{\mu_v(b)} = S_v^0$ as $t \longrightarrow +\infty$. Thus, the type of disease free equilibrium E_0, is the same as that of the equilibrium $E_1 = \left(S_h^0, I_h^0, I_v^0\right) = \left(\dfrac{\Lambda_h}{\mu_h}, 0, 0\right)$ for the following system (5.10).

$$\begin{cases} \dot{S}_h = \Lambda_h - \lambda_h(r,b)S_h + \gamma_h I_h - \mu_h S_h \\ \dot{I}_h = \lambda_h(r,b)S_h - (\mu_h + \gamma_h + \delta)I_h \\ \dot{I}_v = \lambda_v(r,b)\left(S_v^0 - I_v(t)\right) - \mu_v(b)I_v. \end{cases} \tag{5.10}$$

The eigenvalues of Jacobian matrix of system (5.10), at E_1 are $-\mu_h$ and the two roots of the quadratic equation

$$\lambda^2 + (\mu_h + \gamma_h + \delta + \mu_v(b))\,\lambda + \mu_v(b)(\mu_h + \gamma_h + \delta)\left(1 - \mathcal{R}_0^2\right) = 0.$$

If $\mathcal{R}_0 < 1$, the quadratic equation has two negative real roots. Thus E_1, and hence E_0 is also an attracting node.

If $\mathcal{R}_0 > 1$. It is clear that E_1 is a hyperbolic saddle. Thus, E_0 is a hyperbolic saddle.

If $\mathcal{R}_0 = 1$ the third eigenvalue is zero. To determine the type of E_1. First, we translate E_1 to origin by the change of variables $x = S_h - S_h^0$, $y = I_h$ and $z = I_v$. Observe that, 0, $-\mu_h$ and $-(\mu_h + \gamma_h + \delta + \mu_v(b))$ are the three eigenvalues of Jacobian matrix evaluated at E_1, and the corresponding associated to the eigenvectors V_1, V_2 and V_3 are:

$$V_1 = \begin{pmatrix} -\dfrac{\mu_v(b)^2\,(\mu_h+\delta)\,\Lambda_h}{\mu_h^2 m_v P_v(r,b)\Lambda_v} \\ \\ \dfrac{\mu_v(b)^2\Lambda_h}{m_v P_v(r,b)\mu_h\Lambda_v}; \\ \\ 1 \end{pmatrix}, \quad V_2 = \begin{pmatrix} 1 \\ \\ 0 \\ \\ 0 \end{pmatrix}, \quad V_3 = \begin{pmatrix} -\dfrac{(\gamma_h+\mu_v(b))}{\gamma_h+\mu_v(b)+\delta} \\ \\ 1 \\ \\ -\dfrac{m_v P_v(r,b)\mu_h\Lambda_v}{\Lambda_h\mu_v(b)\,(\mu_h+\gamma_h+\delta)} \end{pmatrix}.$$

Let's define the following linear transformation

$$\begin{pmatrix} x \\ y \\ z \end{pmatrix} = P \begin{pmatrix} X \\ Y \\ Z \end{pmatrix},$$

where P is the matrix whose respective columns are V_1, V_2, V_3. Under the transformation P which diagonalizes the Jacobian matrix at E_1, system (5.10) is transformed to

$$\begin{cases} \dot{X} = \displaystyle\sum_{i,j,k\in\mathbb{N}}^{i+j+k=2} l_{ijk} X^i Y^j Z^k + O(|X,Y,Z|^3), \\ \dot{Y} = -\mu_h Y + \displaystyle\sum_{i,j,k\in\mathbb{N}}^{i+j+k=2} m_{ijk} X^i Y^j Z^k + O(|X,Y,Z|^3), \\ \dot{Z} = -(\mu_h+\gamma_h+\delta+\mu_v(b))Z + \displaystyle\sum_{i,j,k\in\mathbb{N}}^{i+j+k=2} n_{ijk} X^i Y^j Z^k + O(|X,Y,Z|^3). \end{cases} \tag{5.11}$$

The coefficients l_{ijk}, m_{ijk} and n_{ijk} of system (5.11) are computed as follows. For the non-vanishing coefficients l_{ijk}, m_{ijk} and n_{ijk}, direct computations and algebraic simplifications give

$$\begin{cases} l_{200} = \dfrac{m_h P_h(r,b)\mu_v(b)^2}{m_v P_v(r,b)\Lambda_v}, \\ l_{101} = \dfrac{m_h P_h(r,b)\mu_h}{\Lambda_h}\left(1 - \dfrac{\mu_v(b)}{\mu_h+\gamma_h+\delta}\right), \\ l_{002} = -\dfrac{m_v P_v(r,b) m_h P_h(r,b)\mu_h^2\Lambda_v}{\Lambda_h^2\,(\mu_h+\gamma_h+\delta)\,\mu_v(b)}. \end{cases} \qquad \begin{cases} m_{200} = -\dfrac{m_h P_h(r,b)\mu_v(b)^2}{m_v P_v(r,b)\Lambda_v}, \\ m_{101} = -\dfrac{m_h P_h(r,b)\mu_h}{\Lambda_h}\left(1 - \dfrac{\mu_v(b)}{\mu_h+\gamma_h+\delta}\right), \\ m_{002} = \dfrac{m_v P_v(r,b) m_h P_h(r,b)\mu_h^2\Lambda_v}{\Lambda_h^2\,(\mu_h+\gamma_h+\delta)\,\mu_v(b)}. \end{cases}$$

$$\begin{cases} n_{200} = \dfrac{\mu_v(b)^3}{m_v P_v(r,b)\Lambda_v}(\mu_h + \delta - \dfrac{2\Lambda_h\mu_v(b)}{\Lambda_v\mu_h} - 2), \\ n_{002} = -2\dfrac{m_v P_v(r,b)\mu_h}{\Lambda_h}(1 + \dfrac{\Lambda_v\mu_h}{\Lambda_h\mu_v(b)}) + \dfrac{m_v P_v(r,b)\mu_h^2\Lambda_v}{\Lambda_h^2\mu_v(b)} \times \dfrac{(\gamma_h + \mu_v(b))}{(\gamma_h + \mu_v(b) + \delta)}, \\ n_{101} = -4\dfrac{\mu_v(b)^2}{\Lambda_v}\left(1 + \dfrac{\Lambda_v\mu_h}{\Lambda_h\mu_v(b)}\right) + \dfrac{\mu_v(b)}{\Lambda_h}(\mu_h + \delta) + \dfrac{\mu_v(b)\mu_h(\gamma_h + \mu_v(b))}{\Lambda_h(\gamma_h + \mu_v(b) + \delta)}, \\ n_{110} = -\dfrac{\mu_v(b)\mu_h}{\Lambda_h}, \\ n_{011} = -\dfrac{m_v P_v(r,b)\mu_h^2\Lambda_v}{\Lambda_h^2\mu_v(b)}. \end{cases}$$

Thereafter, the system (5.11) on the center manifold is locally topologically equivalent to

$$\dot{X} = A_1 X^2 + O(X^3),$$

where,

$$A_1 = \frac{m_h P_h(r,b)\mu_v(b)^2}{m_v P_v(r,b)\Lambda_v}.$$

Since $A_1 > 0$, we conclude that E_0 is a saddle-node of co-dimension 1.

Appendix C: Proof of Theorem 3.10

The Jacobian matrix of system (2.1) at the disease-free equilibrium DFE with $t_h = t_h^*$ is

$$J(E_0) = \begin{pmatrix} -\mu_h & \gamma_h & 0 & -t_h^* \\ 0 & -(\mu_h + \gamma_h + \delta) & 0 & t_h^* \\ 0 & -t_v\dfrac{\Lambda_v}{\Lambda_h}\dfrac{\mu_h}{\mu_v(b)} & -\mu_v(b) & 0 \\ 0 & t_v\dfrac{\Lambda_v}{\Lambda_h}\dfrac{\mu_h}{\mu_v(b)} & 0 & -\mu_v(b) \end{pmatrix}.$$

Hence, its eigenvalues are $\lambda_1 = -\mu_h$, $\lambda_2 = -\mu_v(b)$, $\lambda_3 = 0$ and $\lambda_4 = -\mu_h - \gamma_h - \mu_v(b)$.

Now, we denote by $W = (w_1, w_2, w_3, w_4)^T$ a right eigenvector corresponding to the zero eigenvalue. Then,

$$W = \left(-\frac{\mu_h + \delta}{\mu_h}w_2, \quad w_2, \quad -t_v\frac{\Lambda_v}{\Lambda_h}\frac{\mu_h}{\mu_v(b)^2}w_2, \quad t_v\frac{\Lambda_v}{\Lambda_h}\frac{\mu_h}{\mu_v(b)^2}w_2\right)^T.$$

Furthermore, the left eigenvector $V = (v_1, v_2, v_3, v_4)$ corresponding to the zero eigenvalue such that $V.W = 1$ solves the linear system:

$$\begin{cases} -\mu_h v_1 & = 0, \\ \gamma_h v_1 - (\mu_h + \gamma_h + \delta)v_2 - t_v\dfrac{\Lambda_v}{\Lambda_h}\dfrac{\mu_h}{\mu_v(b)}v_3 + t_v\dfrac{\Lambda_v}{\Lambda_h}\dfrac{\mu_h}{\mu_v(b)}v_4 & = 0, \\ -\mu_v(b)v_3 & = 0, \\ -v_1 t_h^* + v_2 t_h^* - \mu_v(b)v_4 & = 0. \end{cases}$$

Thus,

$$V = \left(0, \quad t_v\frac{\Lambda_v}{\Lambda_h}\frac{\mu_h}{\mu_v(b)(\mu_h + \gamma_h + \delta)}v_4, \quad 0, \quad v_4\right).$$

Let $w_2 = \dfrac{\Lambda_h}{\Lambda_v}\dfrac{\mu_v(b)}{\mu_h t_v}$, $V.W = 1$ equivalent to $v_2w_2 + v_4w_4 = 1$. Thus $v_4 = \dfrac{(\mu_h+\gamma_h+\delta)\mu_v(b)}{\mu_v(b)+\mu_h+\gamma_h+\delta}$.
Let $f_i(i = 1,2,3,4)$ be the vector on the right hand side of system (2.1). The non-vanishing second-order partial derivatives of $f_i(i = 1,2,3,4)$ at the disease-free equilibrium E_0 are:

$$\frac{\partial^2 f_2}{\partial I_h \partial I_v}(E_0,t_h^*) = \frac{-t_h^*}{S_h^0}; \quad \frac{\partial^2 f_4}{\partial S_h \partial I_h}(E_0,t_h^*) = \frac{-t_v S_v^0}{(S_h^0)^2}; \quad \frac{\partial^2 f_4}{\partial I_h \partial S_v}(E_0,t_h^*) = \frac{t_v}{S_h^0}; \quad \frac{\partial^2 f_4}{\partial I_h^2}(E_0,t_h^*) = \frac{-2t_v S_v^0}{(S_h^0)^2}.$$

Using t_h^* given by equation (3.8), we obtain:

$$\frac{\partial^2 f_2}{\partial I_h \partial I_v}(E_0,t_h^*) = -\frac{(\mu_h+\gamma_h+\delta)(\mu_v(b))^2\Lambda_h}{t_v\mu_h\Lambda_v} \times \frac{\mu_h}{\Lambda_h} = -\frac{(\mu_h+\gamma_h+\delta)(\mu_v(b))^2}{t_v\Lambda_v}.$$

According to definitions of the coefficients $\mathcal{A}$ and $\mathcal{B}$ in [7] (Theorem 4.1), it follows that:

$$\begin{aligned}\mathcal{A} &= \frac{1}{2}\sum_{k,i,j=1}^{4} v_k w_i w_j \frac{\partial^2 f_k}{\partial x_i \partial x_j}(E_0,t_h^*), \quad x = (S_h, I_h, S_v, I_v),\\ &= 2v_2w_2w_4\frac{\partial^2 f_2}{\partial I_h \partial I_v}(E_0,t_h^*) + 2v_4w_1w_2\frac{\partial^2 f_4}{\partial S_h \partial I_h}(E_0,t_h^*) + 2v_4w_2w_3\frac{\partial^2 f_4}{\partial I_h \partial S_v}(E_0,t_h^*) + v_4w_2^2\frac{\partial^2 f_4}{\partial I_h^2}(E_0,t_h^*),\end{aligned}$$

and

$$\mathcal{B} = \sum_{k,i=1}^{4} v_k w_i \frac{\partial^2 f_k}{\partial x_i \partial t_h}(E_0,t_h^*) = v_2w_2.$$

Substituting the eigenvectors and the above non-zero partial derivatives into $\mathcal{A}$ and $\mathcal{B}$ leads us to

$$\begin{aligned}\mathcal{A} &= \frac{2(\mu_h+\gamma_h+\delta)(\mu_v(b))^2}{t_v\Lambda_v\mu_h\left(\mu_h+\gamma_h+\delta+\mu_v(b)\right)}\left(\delta-\mu_h-\frac{t_v\mu_h}{\mu_v(b)}\right)\\ &= \frac{2(\mu_h+\gamma_h+\delta)(\mu_v(b))^2}{m_vP_v(r,b)\Lambda_v\mu_h\left(\mu_h+\gamma_h+\delta+\mu_v(b)\right)}\left(\delta-\mu_h-\frac{m_vP_v(r,b)\mu_h}{\mu_v(b)}\right),\\ &= \frac{2(\mu_h+\gamma_h+\delta)(\mu_v(b))^2}{m_vP_v(r,b)\Lambda_v\mu_h\left(\mu_h+\gamma_h+\delta+\mu_v(b)\right)}b\theta\left(1-\frac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)(1-\mu_{v1}b)\right)\frac{m_v\mu_h}{\mu_v(b)\left(1-\theta rb(1-\mu_{v1}b)\right)}\left(r-r^*\right).\end{aligned}$$

and

$$\mathcal{B} = t_v\frac{\Lambda_v}{\Lambda_h}\frac{\mu_h}{\mu_v(b)\left(\mu_h+\gamma_h+\delta+\mu_v(b)\right)}. \tag{5.12}$$

Obviously, the coefficient $\mathcal{B}$ is positive. Now,

1. Assume $r^* < 0$. In fact it suffices to prove that $\left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)(1-\mu_{v1}b)\right) > 0$.
   First, we show that $\delta > \frac{m_v\mu_h}{\mu_v(b)} + \mu_h$.
   a) If $\delta \le \mu_h$, we get $0 < \left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)(1-\mu_{v1}b)\right) \le \left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)\right)$. Thus, $r^* \ge 1$. Absurd.
   b) If $\delta > \mu_h$ and $\delta \le \dfrac{m_v\mu_h}{\mu_v(b)} + \mu_h$, then $\left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)(1-\mu_{v1}b)\right) > \left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)\right)$ and $\left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)\right) \ge 0$. Thus $r^* \ge 0$. Absurd.

   Secondly, for $\delta > \dfrac{m_v\mu_h}{\mu_v(b)} + \mu_h$ and $r^* < 0$, we have $\left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)\right) < 0$ and $r^* < 0$. Thus $\left(1-\dfrac{\mu_v(b)}{m_v\mu_h}(\delta-\mu_h)(1-\mu_{v1}b)\right) > 0$. Hence, model (2.1) undergoes a backward bifurcation.

2. Assume $0 < r^* < 1$. In fact it suffices to prove that $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) > 0$.

   First, we show that: $\mu_h < \delta < \frac{m_v\mu_h}{\mu_v(b)} + \mu_h$.

a) If $\delta \leq \mu_h$, we get $r^* \geq 1$. Absurd.

b) If $\delta \geq \frac{m_v\mu_h}{\mu_v(b)} + \mu_h$, then $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) > \left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)\right)$ and $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)\right) \leq 0$.

   i) If $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) < 0$, then $r^* > 1$. Absurd.

   ii) If $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) > 0$, then $r^* \leq 0$. Absurd.

   Secondly, for $\mu_h < \delta < \frac{m_v\mu_h}{\mu_v(b)} + \mu_h$, we get $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) > \left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)\right)$ and $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)\right) > 0$. Thus, $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) > 0$.

3.a) Assume $r^* > 1$ and $\delta < \mu_h$, we have $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) > 0$. Hence, the system presents a forward bifurcation at $\mathcal{R}_0 = 1$.

3.b) Assume $r^* > 1$ and $\delta > \frac{m_v\mu_h}{\mu_v(b)} + \mu_h$. For $\delta > \frac{m_v\mu_h}{\mu_v(b)} + \mu_h$, we have $\delta > \mu_h$ and $\delta > \frac{m_v\mu_h}{\mu_v(b)} + \mu_h$, then $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) > \left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)\right)$ and $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)\right) < 0$.

   Thus for $r^* > 1$ and $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)\right) < 0$, we get $\left(1 - \frac{\mu_v(b)}{m_v\mu_h}(\delta - \mu_h)(1 - \mu_{v1}b)\right) < 0$. Therefore, model (2.1) undergoes a backward bifurcation.

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***Declaration of Interest Statement**

Declaration of interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

***Credit Author Statement**

Berge Tsanou: Conceptualization, Methodology, Formal analysis, Writing-Review & Editing. **Jean Claude Kamgang**: Methodology, Writing- Original draft preparation, Formal analysis, Software. **Jean M.-S. Lubuma***:* Conceptualization, Supervision, Methodology, Funding acquisition, Writing-Review & Editing**: Duplex Elvis Houpa Danga**: Methodology, Writing-Review & Editing, Formal analysis.

Conflict of Interest

The authors have no conflict of interest to declare.