

A MODEL OF THE DYNAMIC OF TRANSMISSION OF MALARIA, INTEGRATING SEIRS, SEIS, SIRS AND SIS ORGANIZATION IN THE HOST-POPULATION

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Abstract In this paper, we propose and analyse a model of dynamics transmission of malaria, incorporating varying degrees p of susceptible and π of infectious that makes the dynamic of the overall host population integrate SEIRS, SEIS, SIRS and SIS at the same time. For this model we compute a new threshold number ζ and establish the global asymptotic stability of the disease-free equilibrium when $\mathcal{R}_0 < \zeta < 1$. If $\zeta < \mathcal{R}_0 < 1$, the system admits a unique endemic equilibrium (EE) and if $\mathcal{R}_0 > 1$ depending on case the system admits one or two endemic equilibrium. Numerical simulations are presented for different value of \mathcal{R}_0 , based on data collected in the literature. Finally, the impact of parameters p and π of system dynamics are investigated.

Keywords Epidemiological model, malaria, basic reproduction number, global asymptotic stability, non-standard finite difference scheme (NFDS), simulation.

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1. Introduction

Malaria is a major cause of mortality and morbidity in the tropical and sub-tropical areas of the world. It is the worlds most prevalent vector-borne disease and remains among the most devastating diseases in human history. One of its main agent vector is the Plasmodium falciparum malariae, common in the tropical and sub-tropical areas of the globe. It is estimated that the number of cases of malaria rose from 233 million in 2000 to 244 million in 2005 but fell back to 225 million in 2009, and the number of deaths have decreased from 985 000 in 2000 to 781 000 in 2009 and 627000 in 2012 (WHO [13]). At the end of the 2013 rainy season, in the far north region in Cameroon, despite the higher advertisement relative to the used of the MILDA (“Moustiquaire Imprégnée à Longue Durée d’Action”) that have been largely distributed to peoples, there have been an epidemic of malaria which resulted to the death of a proportion of infectious cases (WHO [14]). This and many other fact to

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be numbered are mere justification of the fact that the deepness of the complexity of vector borne diseases is still to be reached. Starting from the basic Ross-MacDonald models (see, e.g., Ross [11] and Macdonald [9]) there are always model of the transmission of malaria that take into account host – population, structured by immune status (see, e.g., Zongo [15]), where the host are described by one population, divided, into a non-immune SEIS sub-population, a semi-immune SEIRS sub-population. The removed state of the semi-immune sub-population is joined, by materials of the non-immune infectious material that have acquired sufficient immune disposition. Chitnis [4] has considered one host population structured in SEIRS organization, with an account taken in the recruitment and the mortality that are linear function of the host and vector population; there is also another work of (see, e.g., Chitnis etc [5]) that makes open the way to the consideration of a dynamical model of the entomological inoculation rate (EIR); this paper has gain a hight improvement in the analysis by Angelov etc [2]. In this review, the vector population is in the SEI-compartment organization. Recent proposition has been made by Kamgang etc [7] working on the modeling of the use of the bed net as protecting measure against bites of mosquitoes in an endemic area. Their model is made up of one population of vectors organized in a way such as to take in account their activity as vertebrate parasite, and the host is divided into multiple sub-population, each in the *SIS* organization. In this work, we make a new proposition of the model of a dynamic transmission of vector borne disease that can enter the family of model of Chitnis [4] and Zongo [15], with a new description of the host population in the model. We integrate *SIS*, *SIRS*, *SEIS*, and *SEIRS* organization into a single host population. There are single compartment *S*, *E*, *I*, and *R* in the host population, and different rate of movement from one compartment to another such as to have the result of the co-existence of the organization numbered here above. The classical consideration would have been to introduce various sub-populations, each respecting one of the above organization. Our motivation in this modeling lays on difficult sub-division of host into sub-population.

The paper is organized as follows. Section 2 describes our model and gives the corresponding system of differential equations. Section 3 establishes the well-posedness of the model. The equilibriums of the system are calculated, and a threshold condition for the stability of the disease free equilibrium (DFE) is calculated. Section 4 analyze the global stability of DFE. Section 5 present the Non Standard Differential Scheme of the model and the graphs of trajectories.

2. Model description and mathematical specification

The human and mosquito populations are homogeneously mixed. In the following subsections, we provide a detailed description of the population structure and dynamics of hosts and vectors.

We propose a compartmental model of malaria transmission in which we have two populations namely hosts (humans) and the vectors (female Anopheles).

2.1. Host population structure and dynamics

The host population is subdivided into four compartments namely: susceptible, infected, infectious and immune. Let α_{hv} denote the incidence rate of infection of

susceptible humans, p the proportion of susceptible that becomes infected and π the proportion of infected that becomes immune, γ the rate of loss of immunity, δ_h the rate of transition for infected to infectious and π the rate of transition of recovery. When an infected mosquito bites a susceptible host, it can become infected with a rate $p\alpha_{hv}$ and after infectious at rate δ_h or directly infectious at rate $(1 - p)\alpha_{hv}$. Once it is infectious, it can become immune to a $\pi\xi$ rate and after susceptible at rate γ or become directly a susceptible at rate $(1 - \pi)\xi$.

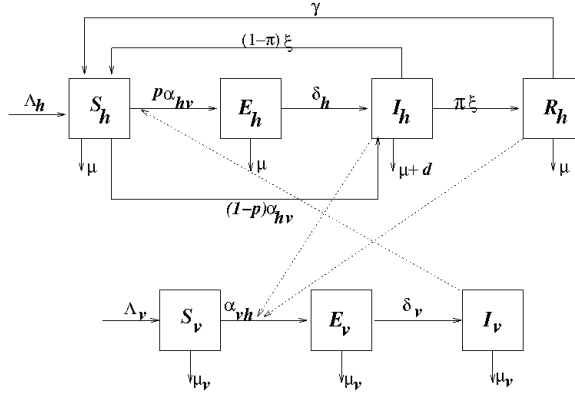


Figure 1. compartment flow diagram.

2.2. Mosquito population structure and dynamics

The population of vectors is divided into the three compartments namely: susceptible, infected and infectious. Let α_{vh} denote the incidence rate of infection of susceptible vectors, δ_v the rate of transition for infected to infectious. Following a bite of a healthy mosquito on an infectious or immune human, mosquitoes can become infected with a α_{vh} rate. Thereafter it becomes infectious to a rate δ_v .

The overall dynamics of the mosquito population and human population is depicted in the multi compartment diagram in 1 The fundamental model parameters are summarized in Table 1, while derived parameters are summarized in Table 2 and variables are summarized in Table 3.

2.3. Model equations

The diagram flow is resulting in the following equations:

$$\begin{cases} S'_h = \Lambda_h + \gamma R_h + (1 - \pi)\xi I_h - (\mu + \alpha_{hv})S_h, \\ E'_h = p\alpha_{hv}S_h - (\mu + \delta_h)E_h, \\ I'_h = \delta_h E_h + (1 - p)\alpha_{hv}S_h - (\mu + d + \xi)I_h, \\ R'_h = \pi\xi I_h - (\mu + \gamma)R_h, \\ S'_v = \Lambda_v - (\mu_v + \alpha_{vh})S_v, \\ E'_v = \alpha_{vh}S_v - (\delta_v + \mu_v)E_v, \\ I'_v = \delta_v E_v - \mu_v I_v. \end{cases} \tag{2.1}$$

Table 1. fundamental model parameter

Parameter	Description	Unity
human		
Λ_h	immigration	$h \times j^{-1}$
γ	rate of lost of immunity	j^{-1}
δ_h	rate of transition for infected to infectious	j^{-1}
ξ	rate of recovery	j^{-1}
μ	death rate	j^{-1}
d	disease-induced death rate	j^{-1}
a	number of bites on humans by a single female mosquito per unit time	$h \times j^{-1}$
m	probability of transmission of infection from infective mosquito	1
p	proportion of susceptible going to exposed state	1
π	proportion of infectious going to immune state	1
Mosquitoes		
Λ_v	immigration	$m \times j^{-1}$
μ_v	death rate	j^{-1}
δ_v	rate of transition for infected to infectious	j^{-1}
c	probability of transmission of infection from infective human	1
\tilde{c}	probability of transmission of infection from recovered human	1

Table 2. Derived model parameters

Param.	Formula	Description
α_{vh}	$\frac{a(cI_h + \tilde{c}R_h)}{N_h}$	incidence rate of susceptible mosquitoes
α_{vh}	$\frac{amI_v}{N_h}$	incidence rate of susceptible human

Table 3. Variable of model

Variable	Description
humans	
S_h	Number of susceptible human
E_h	Number of infected human
I_h	Number of infectious human
R_h	Number of immune human
mosquitoes	
S_v	Number of susceptible mosquitoes
E_v	Number of infected mosquitoes
I_v	Number of infective mosquitoes

3. Well-posedness, dissipativity and equilibriums of the system

In this section we demonstrate well-posedness of the model by demonstrating invariance of the set of non-negative states, as well as boundedness properties of the solution. We also calculate the equilibriums of the system.

3.1. Positive invariance of the non-negative cone in state space

The system (2.1) can be rewritten in the matrix form as

$$\dot{\mathbf{x}} = \mathbf{A}(\mathbf{x})\mathbf{x} + \mathbf{b}(\mathbf{x}) \iff \begin{cases} \dot{\mathbf{x}}_S = \mathbf{A}_S(\mathbf{x})(\mathbf{x}_S - \mathbf{x}_S^*) + \mathbf{A}_{SI}(\mathbf{x})\mathbf{x}_I, \\ \dot{\mathbf{x}}_I = \mathbf{A}_I(\mathbf{x})\mathbf{x}_I. \end{cases} \quad (3.1)$$

Equation (3.1) is defined for values of the state variable $\mathbf{x} = (\mathbf{x}_S; \mathbf{x}_I)$ lying in the non-negative cone of \mathbb{R}_+^7 . Here $\mathbf{x}_S = (S_h, S_v)$ represents the naive component and $\mathbf{x}_I = (E_h, E_v, I_h, I_v, R_h)$ the represents the infected and infectious components of the state of the system.

The matrix $\mathbf{A}_S(\mathbf{x})$, $\mathbf{A}_{SI}(\mathbf{x})$ and $\mathbf{A}_I(\mathbf{x})$ are define as

$$\mathbf{A}_S(\mathbf{x}) = \begin{pmatrix} -\mu & 0 \\ 0 & -\mu_v \end{pmatrix}, \quad \mathbf{A}_{SI}(\mathbf{x}) = \begin{pmatrix} 0 & 0 & (1 - \pi)\xi & -\frac{amS_h}{N_h} & \gamma \\ 0 & 0 & -\frac{acS_v}{N_h} & 0 & -\frac{a\tilde{c}S_v}{N_h} \end{pmatrix}$$

and

$$\mathbf{A}_I(\mathbf{x}) = \begin{pmatrix} -(\mu + \delta_h) & 0 & 0 & \frac{apmS_h}{N_h} & 0 \\ 0 & -(\delta_v + \mu_v) & \frac{acS_v}{N_h} & 0 & \frac{a\tilde{c}S_v}{N_h} \\ \delta_h & 0 & -(\mu + d + \xi) & \frac{(1 - p)amS_h}{N_h} & 0 \\ 0 & \delta_v & 0 & -\mu_v & 0 \\ 0 & 0 & \pi\xi & 0 & -(\mu + \gamma) \end{pmatrix} \quad (3.2)$$

For a given $\mathbf{x} \in \mathbb{R}_+^7$, the matrices $\mathbf{A}(\mathbf{x})$, $\mathbf{A}_S(\mathbf{x})$ and $\mathbf{A}_I(\mathbf{x})$ are Metzler matrices. The following proposition establishes that system (5.4) is epidemiologically well posed.

Proposition 3.1. *The non-negative cone \mathbb{R}_+^7 is positively invariant for the system (5.4).*

3.2. Boundedness and dissipativity of the trajectories

We have the following proposition.

Proposition 3.2. Let $N_h^* = \frac{N_h}{\mu}$, $N_v^* = \frac{N_v}{\mu_v}$, $N_h^\# = \frac{N_h}{\mu + d}$ and

$$\mathcal{G} = \left\{ (S_h, S_v, E_h, E_v, I_h, I_v, R_h) \in \mathbb{R}_+^7 \mid N_h^\# \leq N_h \leq N_h^*, S_v + E_v + I_v \leq N_v^* \right\}.$$

The set \mathcal{G} is GAS for the dynamical system of (2.1) defined on \mathbb{R}_+^7 .

Because \mathcal{G} is GAS we reduce the study of system (2.1) from \mathbb{R}_+^7 to \mathcal{G} .

3.3. Computation of the threshold condition

Theorem 3.1. The basic reproduction number is given by

$$\begin{aligned} \mathcal{R}_0 &= \frac{1}{N_h^*} \sqrt{\frac{(\mu(1-p) + \delta_h)(\tilde{c}\pi\xi + c(\gamma + \mu))\delta_v m a^2 S_h^* S_v^*}{\mu_v(\delta_h + \mu)(\gamma + \mu)(\delta_v + \mu_v)(d + \mu + \xi)}} \\ &= \sqrt{\frac{a^2 m \delta_v \mu \Lambda_v [\mu(1-p) + \delta_h] (\tilde{c}\pi\xi + c(\gamma + \mu))}{\mu_v^2 \Lambda_h (\delta_h + \mu)(\gamma + \mu)(\delta_v + \mu_v)(d + \mu + \xi)}}. \end{aligned} \tag{3.3}$$

Proof. To prove it we use the next generation operator method proposed by Diekmann & Heesterbeek [6] and Van Den Driessche & Watmough [12]. In the disease free equilibrium of the system (2.1), we have the following matrix

$$\begin{aligned} F &= \begin{pmatrix} 0 & 0 & 0 & \frac{ampS_h^*}{N_h^*} & 0 \\ 0 & 0 & \frac{S_v^*ac}{N_h^*} & 0 & \frac{S_v^*a\tilde{c}}{N_h^*} \\ 0 & 0 & 0 & \frac{(1-p)S_h^*am}{N_h^*} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}, \\ V &= \begin{pmatrix} -(\delta_h + \mu) & 0 & 0 & 0 & 0 \\ 0 & -(\delta_v + \mu_v) & 0 & 0 & 0 \\ \delta_h & 0 & -(d + \mu + \xi) & 0 & 0 \\ 0 & \delta_v & 0 & -\mu_v & 0 \\ 0 & 0 & \pi\xi & 0 & -(\gamma + \mu) \end{pmatrix}, \\ FV^{-1} &= \begin{pmatrix} 0 & -\frac{S_h^*a\delta_vmp}{(\delta_v + \mu_v)N_h^*\mu_v} & 0 & -\frac{S_h^*amp}{N_h^*\mu_v} & 0 \\ f_{21} & 0 & f_{23} & 0 & -\frac{S_v^*a\tilde{c}}{(\gamma + \mu)N_h^*} \\ 0 & \frac{(p-1)S_h^*a\delta_vm}{(\delta + \mu_v)N_h^*\mu_v} & 0 & \frac{(p-1)S_h^*am}{N_h^*\mu_v} & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \end{aligned}$$

where

$$f_{21} = -\frac{S_v^*a\tilde{c}\delta_h\pi\xi}{(\delta_h + \mu)(\gamma + \mu)(d + \mu + \pi\xi)N_h^*} - \frac{S_v^*ac\delta_h}{(\delta_h + \mu)(d + \mu + \xi)N_h^*}$$

and

$$f_{23} = -\frac{S_v^*a\tilde{c}\pi\xi}{(\gamma + \mu)(d + \mu + \xi)N_h^*} - \frac{S_v^*ac}{(d + \mu + \xi)N_h^*}$$

$$\mathcal{R}_0 = \rho(FV^{-1}) = \frac{1}{N_h^*} \sqrt{\frac{(\mu(1-p) + \delta_h)(\tilde{c}\pi\xi + c(\gamma + \mu))\delta_v m a^2 S_h^* S_v^*}{\mu_v(\delta_h + \mu)(\gamma + \mu)(\delta_v + \mu_v)(d + \mu + \xi)}}.$$

□

3.4. System equilibria

Steady states of the system are specified by the following proposition.

Proposition 3.3. *System (2.1) admit one disease free equilibrium given by $\mathbf{x}^* \in \mathbb{R}_+^7$ where $\mathbf{x}^* = \left(\frac{\Lambda_h}{\mu}, \frac{\Lambda_v}{\mu_v}, 0, 0, 0, 0, 0\right)$.*

Proposition 3.4. *Suppose that $a\mu - 2d\mu_v > 0$, the system (2.1) has:*

1. a unique biologically feasible endemic equilibrium \mathbf{x}^* if $\mathcal{R}_0 < 1$.
2. a unique biologically feasible endemic equilibrium \mathbf{x}^* if $\mathcal{R}_0 = 1$.
3. two biologically feasible endemic equilibrium \mathbf{x}_1^* and \mathbf{x}_2^* if $\mathcal{R}_0 > 1$ and $A_1^2 - 4A_2A_0 > 0$.
4. no endemic equilibrium otherwise.

with I_h^* the positive solution of the equation $A_2x^2 + A_1x + A_0 = 0$ and the other components of \mathbf{x}^* is given by:

$$\begin{aligned} R_h^* &= \frac{\pi\xi}{\mu + \gamma} I_h^*, & S_v^* &= \frac{\Lambda_v(\mu + \gamma)(\Lambda_h - dI_h^*)}{\mu_v(\mu + \gamma)(\Lambda_h - dI_h^*) + a\mu I_h^*(c(\mu + \gamma) + \tilde{c}\pi\xi)}, \\ E_v^* &= \frac{a\mu(cI_h^* + \tilde{c}R_h^*)}{(\Lambda_h - dI_h^*)(\delta_v + \mu_v)} S_v^*, & I_v^* &= \frac{\delta_v}{\mu_v} E_v^*, \\ S_h^* &= \frac{(\Lambda_h - dI_h^*)[\Lambda_h + \gamma R_h^* + (1 - \pi)\xi I_h^*]}{\mu[\Lambda_h - dI_h^* + a\mu I_v^*]}, & E_h^* &= \frac{p\mu a m}{(\Lambda_h - dI_h^*)(\mu + \delta_h)} I_v^* S_h^*, \end{aligned}$$

Proof. The endemic equilibrium point is all point $(S_h^*, S_v^*, E_h^*, E_v^*, I_h^*, I_v^*, R_h^*)$ of Ω that satisfy:

$$\begin{cases} \Lambda_h + \gamma R_h^* + (1 - \pi)\xi I_h^* - (\mu + \alpha_{hv})S_h^* = 0, \\ p\alpha_{hv}S_h^* - (\mu + \delta_h)E_h^* = 0, \\ \delta_h E_h^* + (1 - p)\alpha_{hv}S_h^* - (\mu + d + \xi)I_h^* = 0, \\ \pi\xi I_h^* - (\mu + \gamma)R_h^* = 0, \\ \Lambda_v - (\mu_v + \alpha_{vh})S_v^* = 0, \\ \alpha_{vh}S_v^* - (\delta_v + \mu_v)E_v^* = 0, \\ \delta_v E_v^* - \mu_v I_v^* = 0. \end{cases} \quad (3.4)$$

$$\left\{ \begin{array}{l} \Lambda_h + \gamma R_h^* + (1 - \pi)\xi I_h^* - \left(\mu + \frac{amI_v^*}{N_h^*} \right) S_h^* = 0, \\ p \frac{amI_v^*}{N_h^*} S_h^* - (\mu + \delta_h) E_h^* = 0, \\ \delta_h E_h^* + (1 - p) \frac{amI_v^*}{N_h^*} S_h^* - (\mu + d + \xi) I_h^* = 0, \\ \pi \xi I_h^* - (\mu + \gamma) R_h^* = 0, \\ \Lambda_v - \left(\mu_v + \frac{acI_h^* + a\tilde{c}R_h^*}{N_h^*} \right) S_v^* = 0, \\ \frac{acI_h^* + a\tilde{c}R_h^*}{N_h^*} S_v^* - (\delta_v + \mu_v) E_v^* = 0, \\ \delta_v E_v^* - \mu_v I_v^* = 0. \end{array} \right. \quad (3.5)$$

$$\left\{ \begin{array}{l} R_h^* = \frac{\pi \xi}{\mu + \gamma} I_h^*, \\ S_v^* = \frac{\Lambda_v(\mu + \gamma)(\Lambda_h - dI_h^*)}{\mu_v(\mu + \gamma)(\Lambda_h - dI_h^*) + a\mu I_h^*(c(\mu + \gamma) + \tilde{c}\pi\xi)}, \\ E_v^* = \frac{a\mu(cI_h^* + \tilde{c}R_h^*)}{(\Lambda_h - dI_h^*)(\delta_v + \mu_v)} S_v^*, \\ I_v^* = \frac{\delta_v}{\mu_v} E_v^*, \\ S_h^* = \frac{(\Lambda_h - dI_h^*) [\Lambda_h + \gamma R_h^* + (1 - \pi)\xi I_h^*]}{\mu [\Lambda_h - dI_h^* + amI_v^*]}, \\ E_h^* = \frac{p\mu am}{(\Lambda_h - dI_h^*)(\mu + \delta_h)} I_v^* S_h^*, \\ \delta_h E_h^* + \frac{(1 - p)a\mu m I_v^* S_h^*}{\Lambda_h - dI_h^*} - (\mu + d + \xi) I_h^* = 0. \end{array} \right. \quad (3.6)$$

Let us set the following parameters

$$\begin{aligned} A_2 &= \mu_v d(\mu + \delta_h)(\mu_v + \delta_v)(\gamma + \mu)(\mu + d + \xi) [(\gamma + \mu)(a\mu c - d\mu_v) + a\mu\tilde{c}\pi\xi], \\ A_1 &= -(\mu + \delta_h)[\Lambda_h\mu_h\xi(\gamma + \mu)(\mu_v + \delta_v)((\gamma + \mu)(ac\mu - 2d\mu_v) + a\tilde{c}\mu\pi(d + 1)) \\ &\quad + (\mu + d)(\mu + \gamma)^2(\Lambda_h\mu_v(\mu_v + \delta_v)(ac\mu - 2d\mu_v) + a^2c\delta_v m\mu\Lambda_v) \\ &\quad + a^2\delta_v m\pi\mu\xi\Lambda_v(\tilde{c}d(\gamma + \mu) + \tilde{c}\gamma + \tilde{c}(1 + \mu) + c(1 + \mu))] \\ &\quad + a^2\delta_v m\mu^2 p\xi [c(\mu^2(1 - p) + \mu(2 + \pi) + \gamma) + \tilde{c}\pi\Lambda_v(\mu(1 - p) + \gamma)], \\ A_0 &= \Lambda_h^2\mu_v^2(\gamma + \mu)^2(\delta_h + \mu)(\delta_v + \mu_v)(d + \mu + \xi)(\mathcal{R}_0^2 - 1) = A'_0(\mathcal{R}_0^2 - 1), \\ B_2 &= -\mu_v d(\mu_v + \delta_v) [(\gamma + \mu)(a\mu c - d\mu_v) + a\mu\tilde{c}\pi\xi], \\ B_1 &= \Lambda_h\mu_v(\mu_v + \delta_v)[(\gamma + \mu)(ac\mu - 2d\mu_v) + a\tilde{c}\pi\mu\xi] + a^2\delta_v m\mu\Lambda_v[c(\gamma + \mu) + \tilde{c}\pi], \\ B_0 &= \Lambda_h^2\mu_v^2(\mu_v + \delta_v)(\gamma + \mu), \end{aligned}$$

$$\begin{aligned}
 & \delta_h E^* + \frac{(1-p)a\mu m I_v^* S^*}{\Lambda_h - dI^*} - (\mu + d + \xi)I^* = 0 \\
 \iff & \frac{I^*(A_2 I^{*2} + A_1 I^* + A_0)}{(\gamma + \mu)(\mu + \delta_h)(B_2 I^{*2} + B_1 I^* + B_0)} = 0 \\
 \iff & I^* = 0 \text{ or } A_2 I^{*2} + A_1 I^* + A_0 = 0 \\
 \iff & I^* = 0 \text{ or } I^{*2} + \frac{A_1}{A_2} I^* + \frac{A_0}{A_2} = 0.
 \end{aligned} \tag{3.7}$$

Because $I_h^* \neq 0$, we are interested in the function $f(I_h^*) = I_h^{*2} + \frac{A_1}{A_2} I_h^* + \frac{A_0}{A_2}$.

$f(I_h)$ is a polynomial function to find its roots, let us use the concept of sum and product. Indeed,

$$\begin{aligned}
 f(I_h^*) &= I_h^{*2} - S I_h^* + P, S = -\frac{A_1}{A_2} \text{ and } P = \frac{A_0}{A_2}, (A_2 > 0 \text{ and } A_1 < 0), \\
 f(I_h^*) = 0 &\iff I_h^{*2} - S I_h^* + P = 0.
 \end{aligned}$$

1. If $\mathcal{R}_0 < 1$ then $A_0 < 0$. In this case $S^2 - 4P > 0$ and $P < 0$ therefore there exists a unique positive solution to the equation $f(I_h^*) = 0$.
2. If $\mathcal{R}_0 = 1$ then $A_0 = 0$. In this case, $P = 0$ then the equation $f(I_h^*) = 0$ has a unique positive solution if $A_1 < 0$.
3. If $\mathcal{R}_0 > 1$ then $A_0 > 0$. In this case, $P > 0$. So there are two positive solutions to the equation $f(I_h^*) = 0$ if $S^2 - 4P > 0$.

□

4. Global asymptotic stability of the disease free equilibrium (DFE)

In this section we analyze the stability of the system equilibria given in Proposition 3.3.

We have the following result for the global asymptotic stability of the disease free equilibrium:

Theorem 4.1. *Let $\zeta = \left(\frac{\mu}{\mu+d}\right)^{\frac{1}{2}}$, and $\tilde{\mathcal{G}} = \{\mathbf{x} \in \mathcal{G} : \mathbf{x} \neq 0\}$ a positively invariant space. When $\mathcal{R}_0 \leq \zeta$, then the DFE for system (2.1) is GAS in the sub-domain $\{\mathbf{x} \in \tilde{\mathcal{G}} : \mathbf{x}_I = 0\}$.*

Proof. Our proof is based on Theorem 4.3 of Kamgang & Sallet [8], which establishes global asymptotic stability for epidemiological systems that can be expressed in the matrix form (5.4). We need only establish for the system (2.1) that the five conditions (h1–h5) required in Theorem 4.3 of Kamgang & Sallet [8] are satisfied when $\mathcal{R}_0 \leq \zeta$.

(h1) The system (2.1) is defined on a positively invariant set \mathbb{R}_+^7 of the non-negative orthant. The system is dissipative on $\tilde{\mathcal{G}}$.

(h2) The sub-system $\dot{\mathbf{x}}_S = \mathbf{A}_S(\mathbf{x}_S, 0)(\mathbf{x} - \mathbf{x}_S^*)$ is express like: $\begin{cases} \dot{S}_h = \Lambda_h - \mu S_h \\ \dot{S}_v = \Lambda_v - \mu_v S_v \end{cases}$ is

the linear system which is GAS at the DFE $\left(\frac{\Lambda_h}{\mu}, \frac{\Lambda_v}{\mu_v}\right)$. The DFE, satisfying the hypotheses H_2 .

- (h3) The matrix $\mathbf{A}_I(\mathbf{x})$ given by (3.2) is Metzler. The graph shown in the figure below, whose nodes represent the various infected disease states is strongly connected, which shows that the matrix \mathbf{A}_I is irreducible.

In this case, the two properties required for condition (h3) follow immediately: off-diagonal terms of the matrix $\mathbf{A}_I(\mathbf{x})$ are non-positive; and Figure 2 shows the associated direct graph $G(\mathbf{A}_I(\mathbf{x}))$, which is evidently connected, thus establishing irreducibility.

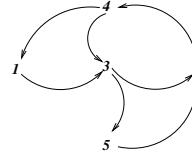


Figure 2. graph associated to the matrix $\mathbf{A}_I(\mathbf{x})$

- (h4) Knowing that $\frac{1}{N_h^\#} > \frac{1}{N_h}$, $S_h^* > S_h$ and $S_v^* > S_v$ we obtain the upper bound $\bar{\mathbf{A}}_I$ of $\mathbf{A}_I(\mathbf{x})$ given by:

$$\bar{\mathbf{A}}_I = \begin{pmatrix} M & N \\ P & Q \end{pmatrix}$$

with

$$M = \begin{pmatrix} -(\mu + \delta_h) & 0 & 0 \\ 0 & -(\delta_v + \mu_v) & \frac{a\tilde{c}S_v^*}{N^\#} \\ \delta_h & 0 & -(\mu + d + \xi) \end{pmatrix},$$

$$N = \begin{pmatrix} 0 & 0 \\ apm & \frac{a\tilde{c}S_v^*}{N^\#} \\ 0 & 0 \end{pmatrix},$$

$$P = \begin{pmatrix} 0 & \delta_v & 0 \\ 0 & 0 & \pi\xi \end{pmatrix} \text{ et } Q = \begin{pmatrix} -\mu_v & 0 \\ 0 & -(\mu + \gamma) \end{pmatrix},$$

$\mathbf{A}_I(\mathbf{x}) < \bar{\mathbf{A}}_I$ for all $\mathbf{x} \in \mathcal{G}$ and $\mathbf{A}_I(\mathbf{x}^*) = \bar{\mathbf{A}}_I$ for all $\mathbf{x} \in \tilde{\mathcal{G}}$ condition (h4) is satisfied.

- (h5) $\alpha(\bar{\mathbf{A}}_I) < 0 \iff \alpha(Q - PM^{-1}N) < 0$.
After one iteration we have

$$T = Q - PM^{-1}N = \begin{pmatrix} \frac{a^2\delta_v cm S_v^* (\mu(1-p) + \delta_h)}{(\mu + \delta_h)(\mu_v + \delta_v)(\xi + \mu + d)N^\#} - \mu_v & \frac{a\delta\tilde{c}S_v^*}{(\mu_v + \delta_v)N^\#} \\ \frac{am\pi\xi(\mu(1-p) + \delta_h)}{(\mu + \delta_h)(\mu + d + \xi)} & -(\gamma + \mu) \end{pmatrix}$$

The second iteration gives

$$\alpha(\bar{\mathbf{A}}_I) < 0 \iff \mathcal{R}_0 < \left(\frac{\mu}{\mu + d}\right)^{\frac{1}{2}}.$$

Since the five conditions for Theorem 4.3 of Kamgang & Sallet [8] are satisfied, the DFE is GAS when $\mathcal{R}_0 < \left(\frac{\mu}{\mu + d}\right)^{\frac{1}{2}}$ \square

5. Numerical Simulation

5.1. A Nonstandard finite difference scheme

For the numerical approximation of our model, we use the nonstandard finite difference (NSFD). We replace the continuous time variable t by discrete nodes $t_n = n\Delta t, n \in \mathbb{Z}$ where Δt is the step size. We wish to find approximate solutions of $S_h, E_h, I_h, R_h, S_v, E_v$ and I_v denote $S_h^n, E_h^n, I_h^n, R_h^n, S_v^n, E_v^n$ and I_v^n at the time $t = t_n$. A major advantage of having an exact scheme for a differential equation is that questions related to the usual considerations of consistency, stability and convergence do not arise (see, e.g., Mickens [10]). Our NSFD scheme reads as:

$$\left\{ \begin{array}{l} \frac{S_h^{n+1} - S_h^n}{\phi(\Delta t)} = \Lambda_h + \gamma R_h^n + (1 - \pi)\xi I_h^n - \mu S_h^n - \alpha_{hv}^n S_h^{n+1}, \\ \frac{E_h^{n+1} - E_h^n}{\phi(\Delta t)} = p\alpha_{hv}^n S_h^{n+1} - (\mu + \delta_h)E_h^n, \\ \frac{I_h^{n+1} - I_h^n}{\phi(\Delta t)} = \delta_h E_h^n + (1 - p)\alpha_{hv}^n S_h^{n+1} - (\mu + d + \xi)I_h^n, \\ \frac{R_h^{n+1} - R_h^n}{\phi(\Delta t)} = \pi\xi I_h^n - (\mu + \gamma)R_h^n, \\ \frac{S_v^{n+1} - S_v^n}{\phi(\Delta t)} = \Lambda_v - \mu_v S_v^n - \alpha_{vh}^n S_v^{n+1}, \\ \frac{E_v^{n+1} - E_v^n}{\phi(\Delta t)} = \alpha_{vh}^n S_v^{n+1} - (\delta_v + \mu_v)E_v^n, \\ \frac{I_v^{n+1} - I_v^n}{\phi(\Delta t)} = \delta_v E_v^n - \mu_v I_v^n. \end{array} \right. \quad (5.1)$$

Since in (5.2) the non linear terms are approximated in a non local way by using more than one mesh point and the standard denominator Δt of the discrete derivatives is replaced by a more complex positive function $\phi(\Delta t)$ which satisfies the condition:

$$\begin{aligned} \phi(\Delta t) &= \Delta t + O((\Delta t)^2), \\ \left(\phi(\Delta t) = \frac{1 - e^{-h\Delta t}}{h} \text{ with } h = \max(\mu, \mu + \delta_h, \mu + d + \xi, \mu + \gamma, \mu_v, \mu_v + \delta_v) \right). \end{aligned}$$

(5.2) is called a nonstandard finite difference method.(see, e.g., Anguelov & Lubuma [1, 3], Mickens [10]).

This system can be rewrite as follow

$$\left\{ \begin{array}{l} S_h^{n+1} = \frac{\phi(\Delta t)}{1 + \alpha_{hv}^n \phi(\Delta t)} [\Lambda_h + \gamma R_h^n + (1 - \pi)\xi I_h^n] + \frac{1 - \mu\phi(\Delta t)}{1 + \alpha_{hv}^n \phi(\Delta t)} S_h^n, \\ E_h^{n+1} = \phi(\Delta t)p\alpha_{hv}^n S_h^{n+1} + [1 - \phi(\Delta t)(\mu + \delta_h)]E_h^n, \\ I_h^{n+1} = \phi(\Delta t)[\delta_h E_h^n + (1 - p)\alpha_{hv}^n S_h^{n+1}] + [1 - \phi(\Delta t)(\mu + d + \xi)]I_h^n, \\ R_h^{n+1} = \phi(\Delta t)\pi\xi I_h^n + [1 - \phi(\Delta t)(\mu + \gamma)]R_h^n, \\ S_v^{n+1} = \frac{\phi(\Delta t)\Lambda_v}{1 + \phi(\Delta t)\alpha_{vh}^n} + \frac{1 - \mu_v\phi(\Delta t)}{1 + \phi(\Delta t)\alpha_{vh}^n} S_v^n, \\ E_v^{n+1} = \phi(\Delta t)\alpha_{vh}^n S_v^{n+1} + [1 - \phi(\Delta t)(\delta_v + \mu_v)]E_v^n, \\ I_v^{n+1} = \phi(\Delta t)\delta_v E_v^n + [1 - \phi(\Delta t)\mu_v]I_v^n. \end{array} \right. \quad (5.2)$$

$$\left\{ \begin{array}{l} S_h^{n+1} = \frac{1 - \mu\phi(\Delta t)}{1 + \alpha_{hv}^n \phi(\Delta t)} (S_h^n - S_h^*) + \frac{1}{1 + \alpha_{hv}^n \phi(\Delta t)} (\phi(\Delta t)[\gamma R_h^n + (1 - \pi)\xi I_h^n] + \frac{\Lambda_h}{\mu}), \\ S_v^{n+1} = \frac{1 - \mu_v\phi(\Delta t)}{1 + \alpha_{vh}^n \phi(\Delta t)} (S_v^n - S_v^*) + \frac{\Lambda_v}{\mu_v(1 + \phi(\Delta t)\alpha_{vh}^n)}, \\ E_h^{n+1} = \phi(\Delta t)p\alpha_{hv}^n S_h^{n+1} + [1 - \phi(\Delta t)(\mu + \delta_h)]E_h^n, \\ I_h^{n+1} = \phi(\Delta t)[\delta_h E_h^n + (1 - p)\alpha_{hv}^n S_h^{n+1}] + [1 - \phi(\Delta t)(\mu + d + \xi)]I_h^n, \\ R_h^{n+1} = \phi(\Delta t)\pi\xi I_h^n + [1 - \phi(\Delta t)(\mu + \gamma)]R_h^n, \\ E_v^{n+1} = \phi(\Delta t)\alpha_{vh}^n S_v^{n+1} + [1 - \phi(\Delta t)(\delta_v + \mu_v)]E_v^n, \\ I_v^{n+1} = \phi(\Delta t)\delta_v E_v^n + [1 - \phi(\Delta t)\mu_v]I_v^n. \end{array} \right. \quad (5.3)$$

$$\left\{ \begin{array}{l} \mathbf{x}_S^{n+1} = \mathbf{A}_S(\mathbf{x}^n)(\mathbf{x}^n - \mathbf{x}^*) + \mathbf{A}_{SI}(\mathbf{x}^n)\mathbf{x}_I^n, \\ \mathbf{x}_I^{n+1} = \mathbf{A}_I(\mathbf{x}_S^{n+1})\mathbf{x}_I^n. \end{array} \right. \quad (5.4)$$

with

$$\begin{aligned} \mathbf{x}_S^{n+1} &= \left(S_h^{n+1} - \frac{\Lambda}{\mu(1 + \sigma\alpha_{hv}^n)}, S_v^{n+1} - \frac{\Lambda_v}{\mu_v(1 + \sigma\alpha_{vh}^n)} \right)^T, \\ \mathbf{x}_I^{n+1} &= (E_h^{n+1}, E_v^{n+1}, I_h^{n+1}, I_v^{n+1}, R_h^{n+1})^T, \\ \mathbf{A}_S(\mathbf{x}^n) &= \begin{pmatrix} \frac{1 - \mu\sigma}{1 + \alpha_{hv}^n \sigma} & 0 \\ 0 & \frac{1 - \mu_v\sigma}{1 + \alpha_{vh}^n \sigma} \end{pmatrix}, \\ \mathbf{A}_{SI}(\mathbf{x}^n) &= \frac{\sigma}{1 + \alpha_{hv}^n \sigma} \begin{pmatrix} 0 & 0 & (1 - \pi)\xi & 0 & \gamma \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix} \end{aligned}$$

and

$$\mathbf{A}_I(\mathbf{x}_S^{n+1}) = \text{diag}(\mathbf{v}(\mathbf{x}_S^{n+1})) + \tilde{\mathbf{A}}_I(\mathbf{x}_S^{n+1})$$

with

$$\mathbf{v}(\mathbf{x}_S^{n+1}) = (1 - \sigma(\mu + \delta_h), 1 - \sigma(\delta_v + \mu_v), 1 - \sigma(\mu + d + \xi), 1 - \sigma\mu_v, 1 - \sigma(\mu + \gamma))$$

and

$$\tilde{\mathbf{A}}_I(\mathbf{x}_S^{n+1}) = \begin{pmatrix} 0 & 0 & 0 & \frac{\sigma p a m}{N_h^n} S_h^{n+1} & 0 \\ 0 & 0 & \frac{\sigma a c}{N_h^n} S_h^{n+1} & 0 & \frac{\sigma a \tilde{c}}{N_h^n} S_h^{n+1} \\ \sigma \delta_h & 0 & 0 & \frac{\sigma(1-p) a m}{N_h^n} S_h^{n+1} & 0 \\ 0 & \sigma S_v^{n+1} & 0 & 0 & 0 \\ 0 & 0 & \sigma \pi \xi & 0 & 0 \end{pmatrix}$$

where $\sigma = \phi(\Delta t)$

We will use the data in the following table for our simulations.

Parameter	Range
humans	
Λ_h	$10^3/(60 \times 365) - 10^3/(50 \times 365)$
γ	0.00055 – 0.0027
δ_h	0.08 – 0.1
ξ	0.0035 – 0.0037
μ	$1/(60 \times 365) - 1/(50 \times 365)$
d	$1.8 \times 10^{-5} - 3.4892 \times 10^{-4}$
a	0.30 – 0.56
m	0.0018
p	<i>variable</i>
π	<i>variable</i>
Mosquitoes	
Λ_v	$10^4/30 - 10^4/21$
μ_v	$1/30 - 1/21$
c	0.24 – 0.8333
\tilde{c}	0.024 – 0.08333

5.2. Figures of trajectories of significatives components of the states

To illustrate results in this work, the system (2.1) is simulated using parameters value/range in the following table 5.1 We see by observing the figures (3) and (4) that when π is fixed the variation of p does not influence the level of endemicity in the population. This means that when we increase the proportion of susceptible becoming infected, the level of endemicity does not vary, so this increase does not affect the level of endemicity. Value of I_h to equilibrium for $\pi = 0.8$ is lower than that for $\pi = 0.2$. So the change π influences the level of endemicity.

Figures (5) and (6) when they show to us is that if p is fixed and π varies, the level of endemicity decreases when π increases which means that the increasing the proportion of individuals who become removed positively influence on the lower level of endemicity.

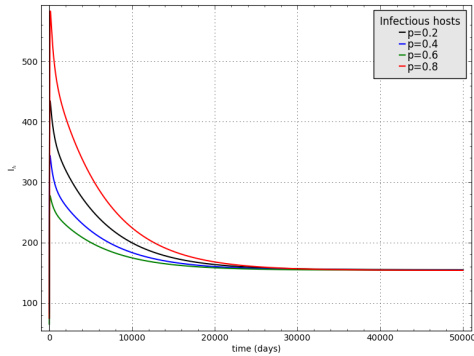


Figure 3. infected host for various values of p and $\pi = 0.2$

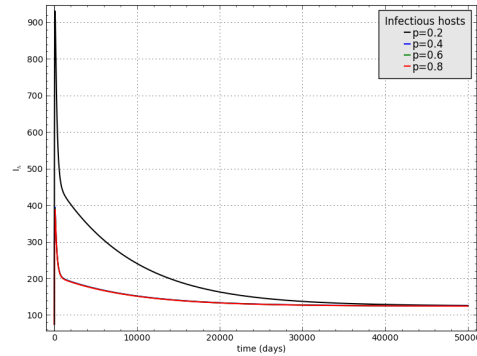


Figure 4. infected host for various values of p and $\pi = 0.8$

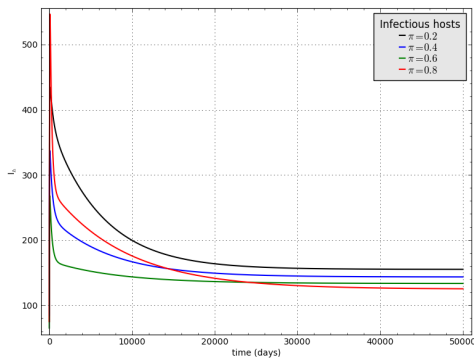


Figure 5. infected host for various values of π and $p = 0.2$

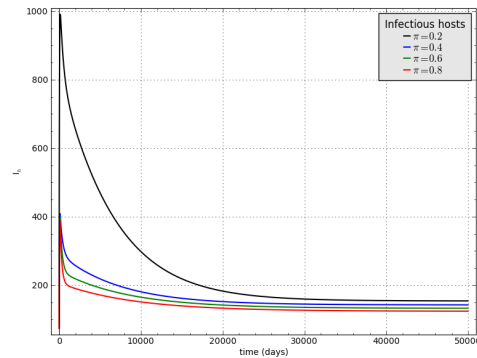


Figure 6. infected host for various values of π and $p = 0.8$

In the following figures, we have fixed all parameters and varies p and p_i in order to make our human population predominantly *SIS*, *SIRS*, *SEIS*, and *SEIRS*; this to identify the dynamics that would provide a minimum level of endemicity.

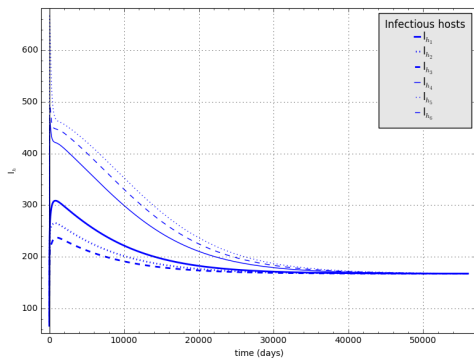


Figure 7. Infected human in SIS dominant population ($p = 0.1$ and $\pi = 0.1$)

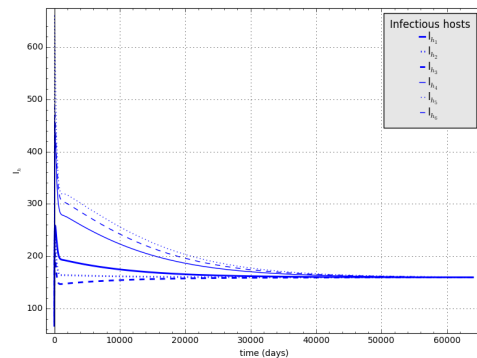


Figure 8. Infected human in SIRS dominant population ($p = 0.1$ and $\pi = 0.9$)

By observing (7), (8), (9) and (10), we find that the smallest value of I_h equilibrium is reached when the population is predominantly *SIRS* or *SEIRS* which

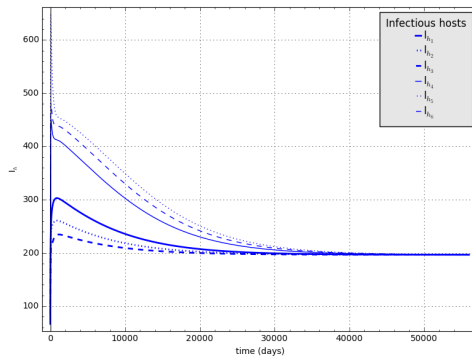


Figure 9. Infected human in SEIS dominant population ($p = 0.9$ and $\pi = 0.1$)

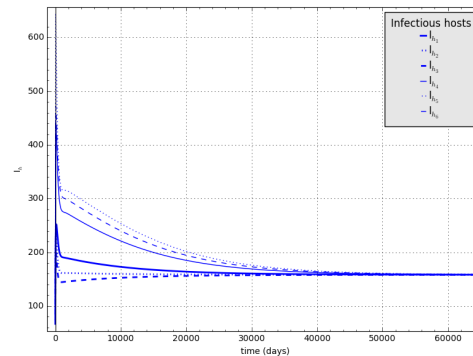


Figure 10. Infected human in SEIRS dominant population ($p = 0.9$ and $\pi = 0.9$)

shows once again the importance of premunition in the fight against malaria. Since this premunition is obtained after exposure to repeated bites of *Anopheles* females and the trend is to advise the hoods of using impregnated bed nets, so we advise all users to the very serious not to be rather more vulnerable.

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