

# Stochastic age-structured malaria transmission model

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## Abstract

A stochastic age-structured malaria epidemic model is formulated and analysed. The proposed stochastic model is a perturbation of a deterministic age-structured model where the diffusion terms are driven by a multidimensional Brownian motion. Numerical simulations show that sample paths converge to deterministic trajectories when  $\mathcal{R}_0 < 1$  whereas a significant difference is observed for  $\mathcal{R}_0 > 1$ .

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## 1 Introduction

Since 1880, malaria is known as an infectious disease caused by the bites of infected female mosquitoes of the genus *Anopheles*[9]. With the current global

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warming phenomenon due to climate changes, mosquitoes multiplication is favoured. According to the World Health Organisation(W.H.O)[18], malaria is one the main cause of deaths in the world and essentially in sub-Saharan Africa. Actual estimates indicate that malaria is responsible of about 429 000 deaths annually world wide(2015). Among them, the African continent is leading with 92% of malaria deaths. High risk groups include pregnant women and children under five years. It is reported that malaria is responsible for death of a child each 30 seconds. It has become a major public health problem in many countries where a significant part of national budgets are oriented in the national malaria control programmes. The main target of preventive measures is to reduce the human-mosquito contact such as the use insecticide-treated mosquito nets, indoor residual spraying and jelly that are repellent to mosquitoes. There is no licensed malaria vaccines, potential candidates are undergoing evaluation.

Mathematical models have played a crucial role in understanding its transmission dynamic and are helping in designing control measures and eradication strategies. Since 1911, Ross proposed the first malaria epidemic model and suggested the reduction of mosquito population as the main control strategy. In 1957, MacDonald [14] did some modifications to Ross model and showed that reducing the mosquito population is not enough for eradication of the disease. More elaborated compartment models have then been used by different authors, including subdivisions of both human and mosquito population into classes such Susceptible-Latent-Infected-Recovered. Effects of important parameters influencing the dynamics of the disease have been in studied in different mathematical models in literature: models considering varying population sizes [16] through migration[6][20] are proposed. Effects of drugs resistance[5], acquired immunity in endemic areas[5][22] and duration of the incubation period[13].

Deterministic models using Ordinary Differential Equations(ODEs) have been extensively studied in modelling malaria transmission dynamic[5][7] [8][16] [17]. Recently, age-structured models using Partial Differential Equations(PDEs) have been used in modelling different diseases[1][12][19][24]. In the present works, we included two mains features in malaria modelling that are ignored in the existing models in literature: the age-structure and stochastic effects. Stochastic models are assumed to be more realistic while studying physical

phenomena, reason while we decided to include noise terms driven by a multi-dimensional Brownian motion in the malaria transmission dynamic.

The remainder of this work is structured as follows: in section 2, an age-structured model is formulated and parameters are explained. Details of the stochastic model formulation from the deterministic model are given. Section 3 deals with the existence of solutions whereas section 4 is devoted to the study of steady states of the model. In section 5, stability analysis of the disease free equilibrium point is investigated. Numerical simulations are presented in section 6 and a conclusion is given in section 7.

## 2 Model formulation

### 2.1 Deterministic model

We consider 4 distinct subclasses in the human population that are: Susceptible, Latent, Infected and Recovered denoted by:  $S_h, L_h, I_h, R_h$  respectively. The total human population is denoted by  $N_h$ . The mosquito population is classified into two compartments namely: Susceptible and Infected denoted by  $S_v, I_v$ . The mosquito total population is denoted by  $N_v$ . The recruitment of new susceptible individuals in the human population is at an age-dependent birth rate  $\alpha_h(a)$ . A natural death rate  $\mu_h(a)$  is imposed to all the human subclasses. With a constant biting rate  $\sigma$ , a proportion  $b$  of bites produces infection in human individuals. Among the bites, a proportion  $c$  infects susceptible mosquitoes. Latent individuals become infectious at an age-dependent rate  $\delta(a)$ . Infected host individuals recover at a rate  $\lambda(a)$  and an additional death rate  $\eta(a)$  due to the disease is imposed to the infected human subclass. The recovered individuals return to the susceptible subclass at an age-dependent rate  $\gamma(a)$ . Mosquito population changes through by natural death and birth rates,  $\lambda_v(a)$  and  $\mu_v(a)$ , respectively.

The following system of first order partial differential equations describes

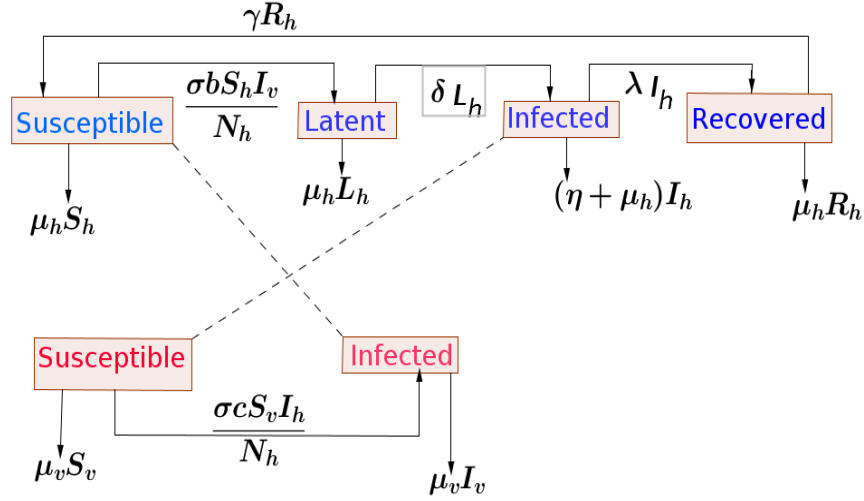


Figure 1: Flow diagram of malaria transmission dynamic

the dynamic of the malaria disease transmission.

$$\begin{cases}
 \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) S_h(t, a) = \alpha_h(a) N_h(t, a) - \frac{\sigma b S_h(t, a) I_v(t, a)}{N_h(t, a)} + \gamma(a) R_h(t, a) - \mu_h(a) S_h(t, a), \\
 \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) L_h(t, a) = \frac{\sigma b S_h(t, a) I_v(t, a)}{N_h(t, a)} - (\delta(a) + \mu_h(a)) L_h(t, a), \\
 \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) I_h(t, a) = \delta(a) L_h(t, a) - (\eta(a) + \lambda(a) + \mu_h(a)) I_h(t, a), \\
 \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) R_h(t, a) = \lambda(a) I_h(t, a) - (\gamma(a) + \mu_h(a)) R_h(t, a), \\
 \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) S_v(t, a) = \alpha_v(a) N_v(t, a) - \frac{\sigma c S_v(t, a) I_h(t, a)}{N_h(t, a)} - \mu_v(a) S_v(t, a), \\
 \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) I_v(t, a) = \frac{\sigma c S_v(t, a) I_h(t, a)}{N_h(t, a)} - \mu_v(a) I_v(t, a).
 \end{cases} \quad (1)$$

with initial and boundary conditions

$$\begin{cases}
 S_h(t, 0) = S_h^0; L_h(t, 0) = I_h(t, 0) = R_h(t, 0) = 0; S_v(t, 0) = S_v^0. \\
 S_h(0, a) = S_{0h}; L_h(0, a) = L_{0h}; I_h(0, a) = I_{0h}; R_h(0, a) = R_{0h}; \\
 S_v(0, a) = S_{0v}; I_v(0, a) = I_{0v}.
 \end{cases}$$

The total populations,  $N_h$  and  $N_v$  are given by  $N_h = S_h + L_h + I_h + R_h$  and  $N_v = S_v + I_v$ . Summing equations in (1), we get  $(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}) N_h(t, a) = (\alpha_h - \mu_h) N_h(t, a) - \eta I_h(t, a)$  and  $(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}) N_v(t, a) = (\alpha_v - \mu_v) N_v(t, a)$

By rescaling the state variables as

$$\begin{cases} s_h(t, a) = \frac{S_h(t, a)}{N_h(t, a)}, l_h(t, a) = \frac{L_h(t, a)}{N_h(t, a)}, i(t, a) = \frac{I_h(t, a)}{N_h(t, a)}, \\ r_h(t, a) = \frac{R_h(t, a)}{N_h(t, a)}, s_v(t, a) = \frac{S_v(t, a)}{N_v(t, a)}, i_v(t, a) = \frac{I_v(t, a)}{N(t, a)}, m = \frac{N_v}{N_h}, \end{cases}$$

system (1) is rewritten as

$$\begin{cases} \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) s_h(t, a) = \alpha_h(a) - \sigma b m s_h(t, a) i_v(t, a) + \gamma(a) r_h(t, a) - \mu_h(a) s_h(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) l_h(t, a) = \sigma b m s_h(t, a) i_v(t, a) - (\delta(a) + \mu_h(a)) l_h(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) i_h(t, a) = \delta(a) l_h(t, a) - (\eta(a) + \lambda(a) + \mu_h(a)) i_h(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) r_h(t, a) = \lambda(a) i_h(t, a) - (\gamma(a) + \mu_h(a)) r_h(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) s_v(t, a) = \alpha_v(a) - \sigma c s_v(t, a) i_h(t, a) - \mu_v(a) s_v(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) i_v(t, a) = \sigma c s_v(t, a) i_h(t, a) - \mu_v(a) i_v(t, a) \end{cases} \quad (2)$$

with initial and boundary conditions

$$\begin{cases} s_h(t, 0) = s_h^0; l_h(t, 0) = i_h(t, 0) = r_h(t, 0) = 0; s_v(t, 0) = s_v^0. \\ s_h(0, a) = s_{0h}; l_h(0, a) = l_{0h}; i_h(0, a) = i_{0h}; r_h(0, a) = r_{0h}; \\ s_v(0, a) = s_{0v}; i_v(0, a) = i_{0v}. \end{cases}$$

Let  $E$  be a Banach space defined as  $E := (L^1(0, \omega))^6$  (where  $\omega$  is the maximum age) endowed with the norm  $\|\phi\| = \sum_{j=1}^6 \|\phi_j\|$ ,  $\phi \in E$ . The state space is given by  $\Omega = \{(s_h, l_h, i_h, r_h, s_v, i_v) \in E_+, 0 \leq s_h + l_h + i_h + r_h \leq 1, 0 \leq s_v + i_v \leq 1\}$ , where  $E_+$  is the positive cone of  $E$ .

## 2.2 Stochastic model

We derive the stochastic model from the deterministic model using the approach found in [2], [3], [4] and [15] and references therein. We consider a small interval of time  $\Delta t$  in which at most one individual can enter or get out from a given subclass. Changes are denoted by  $+1, -1$  or  $0$ . The following table gives the possible changes and their corresponding probabilities.

Table 1: Table of probabilities associated to each possible transition

$(1, 0, 0, 0, 0, 0)$	$\alpha_h \Delta t$
$(-1, 1, 0, 0, 0, 0)$	$\sigma b m s_h i_v \Delta t$
$(1, 0, 0, -1, 0, 0)$	$\gamma r_h \Delta t$
$(-1, 0, 0, 0, 0, 0)$	$\mu_h s_h \Delta t$
$(0, -1, 1, 0, 0, 0)$	$\delta l_h \Delta t$
$(0, -1, 0, 0, 0, 0)$	$\mu_h l_h \Delta t$
$(0, 0, -1, 0, 0, 0)$	$\eta i_h \Delta t$
$(0, 0, -1, 1, 0, 0)$	$\lambda i_h \Delta t$
$(0, 0, -1, 0, 0, 0)$	$\mu_h i_h \Delta t$
$(0, 0, 0, -1, 0, 0)$	$\mu_h r_h \Delta t$
$(0, 0, 0, 0, 1, 0)$	$\alpha_v \Delta t$
$(0, 0, 0, 0, -1, 1)$	$\sigma c s_v i_h \Delta t$
$(0, 0, 0, 0, -1, 0)$	$\mu_v s_v \Delta t$
$(0, 0, 0, 0, 0, -1)$	$\mu_v i_v \Delta t$

We obtain the following covariance matrix.

$$V = \begin{pmatrix} V_{11} & -\sigma b m s_h i_v & 0 & -\gamma r_h & 0 & 0 \\ -\sigma b m s_h i_v & V_{22} & -\delta l_h & 0 & 0 & 0 \\ 0 & -\delta l_h & V_{33} & -\lambda i_h & 0 & 0 \\ -\gamma r_h & 0 & -\lambda i_h & V_{44} & 0 & 0 \\ 0 & 0 & 0 & 0 & V_{55} & -\sigma c s_v i_h \\ 0 & 0 & 0 & 0 & -\sigma c s_v i_h & V_{66} \end{pmatrix} \quad (3)$$

with  $V_{11} = \alpha_h + \sigma b m s_h i_v + \gamma r_h + \mu_h s_h$ ,  $V_{22} = \sigma b m s_h i_v + (\delta + \mu_h) l_h$ ,  $V_{33} = \delta l_h + (\eta + \lambda + \mu_h) i_h$ ,  $V_{44} = \lambda i_h + (\gamma + \mu_h) r_h$ ,  $V_{55} = \alpha_v + \sigma c s_v i_h + \mu_v s_v$ ,  $V_{66} = \sigma c s_v i_h + \mu_v i_v$ .

The stochastic model is given as follows

$$\left\{ \begin{array}{l} (\frac{\partial}{\partial t} + \frac{\partial}{\partial a})s_h = \alpha_h - \sigma b m s_h i_v + \gamma r_h - \mu_h s_h + \sqrt{\alpha_h} \frac{dW_1}{dt} + \sqrt{\sigma b m s_h i_v} \frac{dW_2}{dt} \\ + \sqrt{\gamma r_h} \frac{dW_3}{dt} + \sqrt{\mu_h s_h} \frac{dW_4}{dt}, \\ (\frac{\partial}{\partial t} + \frac{\partial}{\partial a})l_h = \sigma b m s_h i_v - (\delta + \mu_h)l_h - \sqrt{\sigma b m s_h i_v} \frac{dW_2}{dt} + \sqrt{\delta l_h} \frac{dW_5}{dt} + \sqrt{\mu_h l_h} \frac{dW_6}{dt}, \\ (\frac{\partial}{\partial t} + \frac{\partial}{\partial a})i_h = \delta l_h - (\eta + \lambda + \mu_h)i_h - \sqrt{\delta l_h} \frac{dW_5}{dt} + \sqrt{\eta i_h} \frac{dW_7}{dt} \\ + \sqrt{\lambda i_h} \frac{dW_8}{dt} + \sqrt{\mu_h i_h} \frac{dW_8}{dt}, \\ (\frac{\partial}{\partial t} + \frac{\partial}{\partial a})r_h = \lambda i_h - (\gamma + \mu_h)r_h - \sqrt{\gamma r_h} \frac{dW_3}{dt} - \sqrt{\lambda i_h} \frac{dW_8}{dt} + \sqrt{\mu_h r_h} \frac{dW_{10}}{dt}, \\ (\frac{\partial}{\partial t} + \frac{\partial}{\partial a})s_v = \alpha_v - \sigma c s_v i_h - \mu_v s_v + \sqrt{\lambda_v} \frac{dW_{11}}{dt} + \sqrt{\sigma c s_v i_h} \frac{dW_{12}}{dt} + \sqrt{\mu_v s_v} \frac{dW_{13}}{dt}, \\ (\frac{\partial}{\partial t} + \frac{\partial}{\partial a})i_v = \sigma c s_v i_h - \mu_v i_v - \sqrt{\sigma c s_v i_h} \frac{dW_{12}}{dt} + \sqrt{\mu_v i_v} \frac{dW_{14}}{dt}. \end{array} \right.$$

with initial and boundary conditions

$$\left\{ \begin{array}{l} s_h(t, 0) = s_h^0; l_h(t, 0) = i_h(t, 0) = r_h(t, 0) = 0; s_v(t, 0) = s_v^0. \\ s_h(0, a) = s_{0h}; l_h(0, a) = l_{0h}; i_h(0, a) = i_{0h}; r_h(0, a) = r_{0h}; \\ s_v(0, a) = s_{0v}; i_v(0, a) = i_{0v}. \end{array} \right.$$

### 3 Existence and uniqueness of solutions

#### 3.1 Deterministic model

In order to investigate the existence of solutions for system (2), we rewrite it in a simplified form as follows.

We define a linear operator  $A$ , a generator of a  $C_0$ -semigroup, by

$$(A\phi)(x) := \left(-\frac{d\phi_1}{dx}, \dots, -\frac{d\phi_6}{dx}\right)^T, \phi_i \in D(A)$$

with  $D(A) := \{\phi \in E : \phi_i \in AC[0, \omega), \phi_i(0) = 0\}$ , where  $AC[0, \omega)$  denotes the space of absolutely continuous functions on  $[0, \omega)$ . We consider a nonlinear

and Frchet differentiable operator  $F$  defined by

$$F(\phi)(x) := \begin{pmatrix} \alpha_h(x) - \sigma b m \phi_1(x) \phi_6(x) + \gamma(x) \phi_4(x) - \mu_h(x) \phi_1(x) \\ \sigma b m \phi_1(x) \phi_6(x) - (\delta(x) + \mu_h(x)) \phi_2(x) \\ \delta(x) \phi_2(x) - (\eta(x) + \lambda(x) + \mu_h(x)) \phi_3(x) \\ \lambda(x) \phi_3(x) - (\gamma(x) + \mu_h(x)) \phi_4(x) \\ \alpha_v(x) - \sigma c \phi_5(x) \phi_3(x) - \mu_v(x) \phi_5(x) \\ \sigma c \phi_5(x) \phi_3(x) - \mu_v(x) \phi_6(x) \end{pmatrix} \quad (4)$$

Therefore, system (2) takes the following form

$$\begin{cases} \frac{dX}{dt} = AX(t) + F(X(t)), \\ X(0) = X_0, \end{cases} \quad (5)$$

where  $X(t) = (s_h(\cdot, t), l_h(\cdot, t), i_h(\cdot, t), r_h(\cdot, t), s_v(\cdot, t), i_v(\cdot, t))^T$ . It has been shown in [12] that the Cauchy problem (6) admits a unique positive mild solution with respect to positive initial conditions.

### 3.2 Stochastic model

System (4) can be rewritten as

$$\begin{cases} dX = (AX(t) + F(X(t)))dt + G(X(t))dW_t, \\ X(0) = X_0, \end{cases} \quad (6)$$

where  $G(X)$  is a  $6 \times 14$  matrix obtained from system(4). In [21], a proof of a unique mild solution to (7) is given. The existence and uniqueness are guaranteed by the global Lipschitz and linear growth conditions of both  $F$  and  $G$ . In what follows we show that there exist positive constants  $L$  and  $L'$  such that

$$\begin{aligned} \|F(X_1) - F(X_2)\|_1 &\leq L\|X_1 - X_2\|, \\ \|G(X_1) - G(X_2)\|_2 &\leq L'\|X_1 - X_2\|, \end{aligned}$$

and that there exist positive constants  $K$  and  $K'$  such that

$$\begin{aligned} \|F(X_1)\|_1 &\leq K(1 + \|X_1\|), \\ \|G(X_1)\|_2 &\leq K'(1 + \|X_1\|). \end{aligned}$$



Using Young's inequality for terms in  $s_h i_v$  and  $s_v i_h$ , and applying the triangle inequality, we obtain

$$\begin{aligned} \|F(X_1) - F(X_2)\|_1 &\leq \frac{3}{2}\mu_h |s_{1h} - s_{2h}| + (2\sigma + \mu_h)|l_{1h} - l_{2h}| + (\eta + 2\lambda + \frac{3}{2}\mu_h)|i_{1h} - i_{2h}| \\ &\quad + (\gamma + \mu_h)|r_{1h} - r_{2h}| + \frac{3}{2}\mu_v |s_{1v} - s_{2v}| + \frac{3}{2}\mu_v |i_{1v} - i_{2v}|. \end{aligned}$$

Taking  $L := \max(2\sigma + \mu_h, \eta + 2\lambda + \frac{3}{2}\mu_h, \gamma + \mu_h, \frac{3}{2}\mu_v)$ ,

$$\begin{aligned} \|F(X_1) - F(X_2)\|_1 &\leq L(|s_{1h} - s_{2h}| + |l_{1h} - l_{2h}| + |i_{1h} - i_{2h}| \\ &\quad + |r_{1h} - r_{2h}| + |s_{1v} - s_{2v}| + |i_{1v} - i_{2v}|) = L\|X_1 - X_2\|. \end{aligned}$$

Applying the same approximations, we arrive to similar result for the linear growth of  $F$ .

$$\begin{aligned} \|F(X)\|_1 &\leq \alpha_h + \alpha_v + \frac{3}{2}\mu_h |s_h| + (2\delta + \mu_h)|l_h| + (\eta + 2\lambda + \frac{3}{2}\mu_h)|i_h| + (2\gamma + \mu_h)|r_h| \\ &\quad + \frac{3}{2}\mu_v |s_v| + \frac{3}{2}\mu_v |i_v|. \end{aligned}$$

Setting  $K := \max(L, \alpha_h + \alpha_v)$

$$\|F(X)\|_1 \leq K(1 + |s_h| + |l_h| + |i_h| + |r_h| + |s_v| + |i_v|) = K(1 + \|X\|_1).$$

In the same way, by neglecting product terms  $s_h i_v$  and  $s_v i_h$ , we show the following for  $G$ .

$$\begin{aligned} \|G(X^1) - G(X^2)\|_2 &\leq [\mu_h (s_h^1 - s_h^2)^2 + (2\delta + \mu_h)(l_h^1 - l_h^2)^2 + (\eta + 2\lambda + \mu_h)(i_h^1 - i_h^2)^2 \\ &\quad + (2\gamma + \mu_h)(r_h^1 - r_h^2)^2 + \mu_v (s_v^1 - s_v^2)^2 + \mu_v (i_v^1 - i_v^2)^2]^{1/2}, \end{aligned}$$

Taking  $L' := \max(\mu_v, \eta + 2\lambda + \mu_h, 2\gamma + \mu_h, 2\delta + \mu_h)$ , we get

$$\begin{aligned} \|G(X^1) - G(X^2)\|_2 &\leq L'[(s_h^1 - s_h^2)^2 + (l_h^1 - l_h^2)^2 + (i_h^1 - i_h^2)^2 \\ &\quad + (r_h^1 - r_h^2)^2 + (s_v^1 - s_v^2)^2 + (i_v^1 - i_v^2)^2]^{1/2}, \end{aligned}$$

which shows that  $\|G(X^1) - G(X^2)\|_2 \leq L'\|X^1 - X^2\|_2$ . In the same way, the linear growth of  $G$  follows.

## 4 Steady state solutions

In this section, we deal with the deterministic model. The perturbation introduced to get the stochastic model is driven by a Brownian motion, a stochastic process which vanishes at  $t = 0$ . Numerical simulations of sample paths are used to study the behaviour of the system around the steady states.

### 4.1 Disease free equilibrium solutions

By setting  $l_h = i_h = r_h = i_v = 0$ , the steady state of system (2) is given by solution of the following system

$$\begin{cases} \frac{ds_h}{da} = \alpha_h(a) - \mu_h(a)s_h(a), \\ \frac{ds_v}{da} = \alpha_v(a) - \mu_v(a)s_v(a), \end{cases} \quad (7)$$

Thus, the disease free equilibrium point is  $(s_h^*, l_h^*, i_h^*, r_h^*, s_v^*, i_v^*) = (1, 0, 0, 0, 1, 0)$ .

### 4.2 Basic reproduction number

We compute the malaria reproduction number by the next generation matrix method. Let  $\mathcal{F}$  denotes the vector of terms corresponding to news infections,  $\nu = \nu_+ + \nu_-$ , where  $\nu_+$  is the vector of terms corresponding individuals entering a given compartment and  $\nu_-$  corresponds to individuals going out of a subclass in each population. We rewrite system (2) starting with equations with terms containing new infections in the two populations.

$$\begin{cases} \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) l_h(t, a) = \sigma b m s_h(t, a) i_v(t, a) - (\delta(a) + \mu_h(a)) l_h(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) i_h(t, a) = \delta(a) l_h(t, a) - (\eta(a) + \lambda(a) + \mu_h(a)) i_h(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) i_v(t, a) = \sigma c s_v(t, a) i_h(t, a) - \mu_v(a) i_v(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) s_h(t, a) = \alpha_h(a) - \sigma b m s_h(t, a) i_v(t, a) + \gamma(a) r_h(t, a) - \mu_h(a) s_h(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) r_h(t, a) = \lambda(a) i_h(t, a) - (\gamma(a) + \mu_h(a)) r_h(t, a), \\ \left( \frac{\partial}{\partial t} + \frac{\partial}{\partial a} \right) s_v(t, a) = \alpha_v(a) - \sigma c s_v(t, a) i_h(t, a) - \mu_v(a) s_v(t, a). \end{cases} \quad (8)$$

Then, matrices  $\mathcal{F}$  and  $\nu$  are given by

$$\mathcal{F} = \begin{pmatrix} \sigma b m s_h i_v \\ \delta l_h \\ \sigma c s_v i_h \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad \nu = \begin{pmatrix} (\mu_h + \delta) l_h \\ (\eta + \lambda + \mu_h) i_h \\ \mu_v i_v \\ \sigma b m s_h i_v - \alpha_h - \gamma r_h + \mu_h s_h \\ (\gamma + \mu_h) r_h - \lambda i_h \\ \sigma c s_v i_h + \mu_v s_v - \alpha_v \end{pmatrix}$$

As we have three subclasses containing new infections, the partial derivatives of  $\mathcal{F}$  and  $\nu$  with respect to  $l_h, i_h$  and  $i_v$  are given by the following  $3 \times 3$  matrices  $F$  and  $V$ , respectively.

$$F = \begin{pmatrix} 0 & 0 & \sigma b m s_h \\ \delta & 0 & 0 \\ 0 & \sigma c s_v & 0 \end{pmatrix} \quad V = \begin{pmatrix} \mu_h + \delta & 0 & 0 \\ 0 & \eta + \lambda + \mu_h & 0 \\ 0 & 0 & \mu_v \end{pmatrix}$$

The spectral radius of the product  $FV^{-1}$  evaluated at the disease free equilibrium state, corresponds to the basic reproduction number denoted by  $\mathcal{R}_0$  and it is given by

$$\mathcal{R}_0 = \sqrt[3]{\frac{\sigma^2 \delta b c m}{[\mu_v (\mu_h + \delta) (\eta + \lambda + \mu_h)]^2}}. \quad (9)$$

### 4.3 Endemic steady state

We determine the endemic equilibrium state of system (2) when  $\mathcal{R}_0 > 1$  by solving the following system

$$\begin{cases} \frac{ds_h(a)}{da} = \alpha_h(a) - \sigma b m s_h(a) i_v(a) + \gamma(a) r_h(a) - \mu_h(a) s_h(a), \\ \frac{dl_h(a)}{da} = \sigma b m s_h(a) i_v(a) - (\delta(a) + \mu_h(a)) l_h(a), \\ \frac{di_h(a)}{da} = \delta(a) l_h(a) - (\eta(a) + \lambda(a) + \mu_h(a)) i_h(a), \\ \frac{dr_h(a)}{da} = \lambda(a) i_h(a) - (\gamma(a) + \mu_h(a)) r_h(a), \\ \frac{ds_v(a)}{da} = \alpha_v(a) - \sigma c s_v(a) i_h(a) - \mu_v(a) s_v(a), \\ \frac{di_v(a)}{da} = \sigma c s_v(a) i_h(a) - \mu_v(a) i_v(a) \end{cases} \quad (10)$$

with boundary conditions

$$\begin{cases} s_h(0) = s_{0h}, \\ l_h(0) = l_{0h}, \\ i_h(0) = i_{0h}, \\ r_h(0) = r_{0h}, \\ s_v(0) = s_{0v}, i_v(0) = i_{0v}. \end{cases}$$

which is rewritten as

$$\begin{cases} \frac{dX(a)}{da} = F(X(a)), \\ X(0) = (s_{0h}, l_{0h}, i_{0h}, r_{0h}, s_{0v}, i_{0v})^T. \end{cases} \quad (11)$$

The Cauchy problem (12) where  $F(X)$  is globally Lipschitz continuous as shown in section (2.2) admits a unique solution which corresponds to the endemic state of the deterministic model (2). Thus, we have

$$\begin{cases} s_h(a)^{**} = \int_0^a [\alpha_h(\tau) - \sigma b m s_h(\tau) i_v(\tau) + \gamma(\tau) r_h(\tau) - \mu_h(\tau) s_h(\tau)] d\tau, \\ l_h(a)^{**} = \int_0^a [\sigma b m s_h(\tau) i_v(\tau) - (\delta(\tau) + \mu_h(\tau)) l_h(\tau)] d\tau, \\ i_h(a)^{**} = \int_0^a [\delta(a) l_h(\tau) - (\eta(\tau) + \lambda(\tau) + \mu_h(\tau)) i_h(\tau)] d\tau, \\ r_h(a)^{**} = \int_0^a [\lambda(\tau) i_h(\tau) - (\gamma(\tau) + \mu_h(\tau)) r_h(\tau)] d\tau, \\ s_v(a)^{**} = \int_0^a [\alpha_v(\tau) - \sigma c s_v(\tau) i_h(\tau) - \mu_v(\tau) s_v(\tau)] d\tau, \\ i_v(a)^{**} = \int_0^a [\sigma c s_v(\tau) i_h(\tau) - \mu_v(\tau) i_v(\tau)] d\tau \end{cases} \quad (12)$$

## 5 Stability analysis

**Theorem 5.1.** *The disease-free equilibrium state is globally asymptotically stable if  $\mathcal{R}_0 \leq 1$  and  $(\delta + \mu_h)(\eta + \lambda + \mu_h)\mu_v < 1$ .*

*Proof:* We consider the lyapunov function  $V = Al_h + Bi_v + i_h$  and take its partial derivative with respect to  $(t, a)$ .

$$\begin{aligned}
\left(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}\right)V &= A\left(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}\right)l_h + B\left(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}\right)i_v + \left(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}\right)i_h, \\
&= A(\sigma b s_h i_v - (\delta + \mu_h)l_h) + B(\sigma c s_v i_h - \mu_v i_v) \\
&\quad + \delta l_h - (\eta + \lambda + \mu_h)i_h \\
&= [A\sigma b m s_h - B\mu_v]i_v + [B\sigma c s_v - (\eta + \lambda + \mu_h)]i_h + [-A(\delta + \mu_h) + \delta]l_h
\end{aligned}$$

We choose the values of  $A$  and  $B$  in such way that the coefficients of  $l_h$  and  $i_h$  are equal to zero. That implies,

$$\begin{cases} A = \frac{\delta}{\delta + \mu_h}, \\ B = \frac{\eta + \lambda + \mu_h}{\delta c s_v} \end{cases} \quad (13)$$

Replacing the values of  $A$  and  $B$  and evaluating the derivative at the disease free equilibrium, we obtain

$$\begin{aligned}
\left(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}\right)V_{DFE} &= \frac{1}{\sigma c(\delta + \mu_h)} \left[ \sigma^2 \delta b c m - (\delta + \mu_h)(\eta + \lambda + \mu_h)\mu_v \right] i_v, \quad (14) \\
&= \frac{1}{\sigma c(\delta + \mu_h)(\delta + \mu_h)(\eta + \lambda + \mu_h)\mu_v} \left[ \beta \mathcal{R}_0^3 - 1 \right] i_v
\end{aligned}$$

with  $\beta = (\delta + \mu_h)(\eta + \lambda + \mu_h)\mu_v$ . Thus, we have  $\left(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}\right)V_{DFE} \leq 0$  if  $\mathcal{R}_0 \leq 1$ . Therefore, by the LaSalle's invariance principle, the global asymptotic stability of the disease-free equilibrium point follows.

## 6 Numerical simulation

In this section, the numerical method used in our simulations is based on the finite difference method. Forward in time-backward in age numerical scheme is used as in[1]. Each equation in system (2) can be rewritten as

$$\left(\frac{\partial}{\partial t} + \frac{\partial}{\partial a}\right)f(t, a) = g(t, a),$$

and can be approximated by

$$\frac{f(t_{k+1}, a_i) - f(t_k, a_i)}{\Delta t} + \frac{f(t_k, a_i) - f(t_k, a_{i-1})}{\Delta a} = g(t_k, a_i).$$

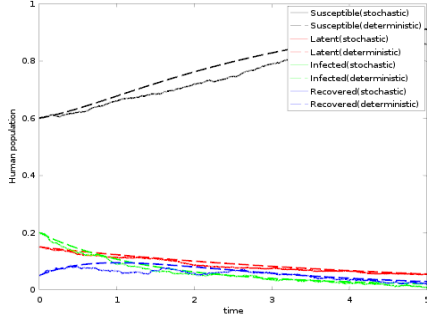
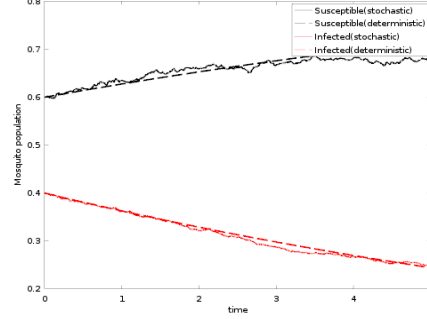
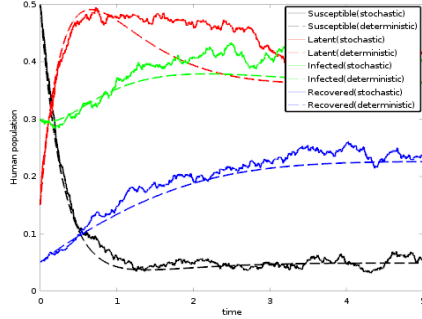
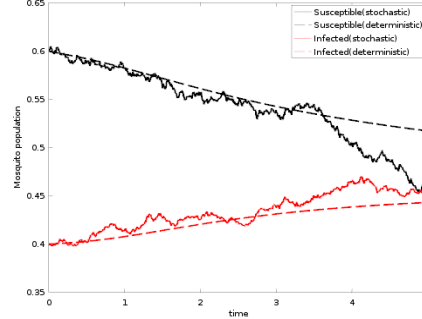
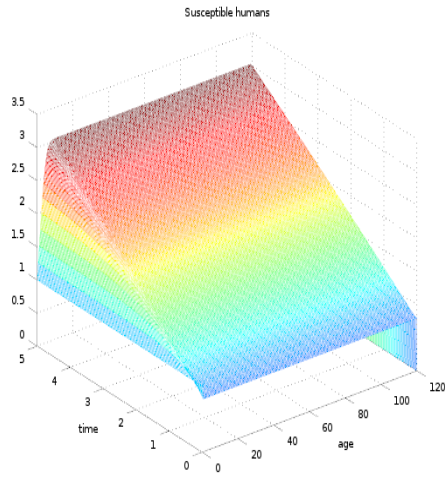
(a) Case where  $\mathcal{R}_0 < 1$  for the human population(b) Mosquito population when  $\mathcal{R}_0 < 1$ (c) Human population when  $\mathcal{R}_0 > 1$ (d) Mosquito population when  $\mathcal{R}_0 > 1$ 

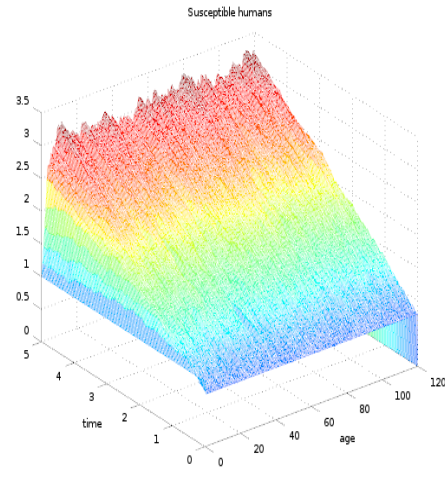
Figure 2: We compare the deterministic and stochastic evolution over time when  $\mathcal{R}_0 < 1$  ( $\mathcal{R}_0 = 0.10528$ ) and  $\mathcal{R}_0 > 1$  ( $\mathcal{R}_0 = 11.885$ ). Sub-figures 2(a)-2(b) Show an increasing number of susceptible both for humans and mosquitoes over time. In the other compartments, populations are decreasing, which implies that the disease will disappear even without intervention. In sub-figures 2(c)-2(d), we observe that infected individuals are increasing. An intervention is need to stop the disease progression.

In what follows, we present simulations of the deterministic model (2) and the stochastic one (4). In figures 2(a)-2(d), the evolution in time of the state variables shows that the effects of the perturbation are more remarkable when  $\mathcal{R}_0 > 1$ . In Figures 3-6, we present an age-time evolution of both the deterministic and stochastic case. We observe that stochastic evolution approaches

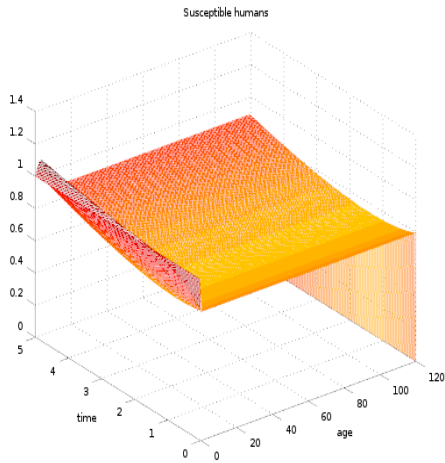
the deterministic one when  $\mathcal{R}_0 < 1$ .



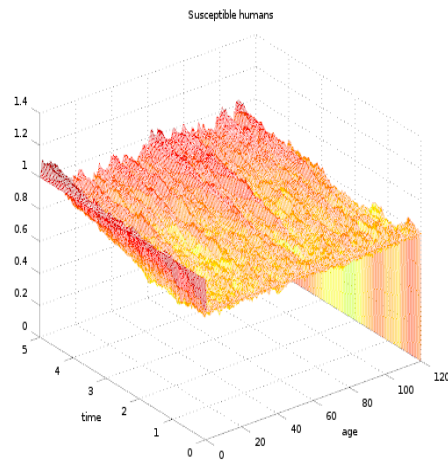
(a) Case where  $\mathcal{R}_0 < 1$  for the deterministic model



(b) Stochastic model when  $\mathcal{R}_0 < 1$



(c) Deterministic model when  $\mathcal{R}_0 > 1$



(d) Stochastic model when  $\mathcal{R}_0 > 1$

Figure 3: The age-time evolution of the deterministic and stochastic models for the susceptible humans shows that when  $\mathcal{R}_0 < 1$ , the population will continue to increase. The disease has life effect in lowering susceptible individuals. When  $\mathcal{R}_0 > 1$ , the susceptible individuals decrease and attain a stable state: the endemic equilibrium state.

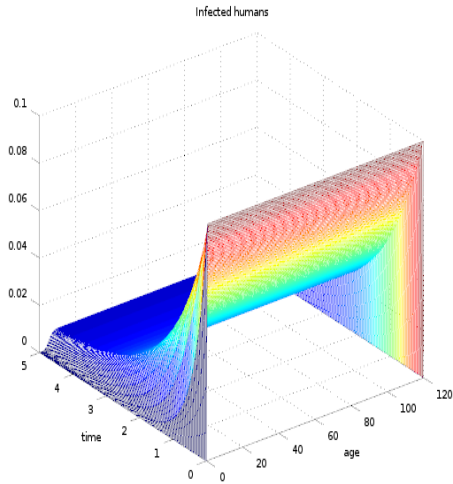
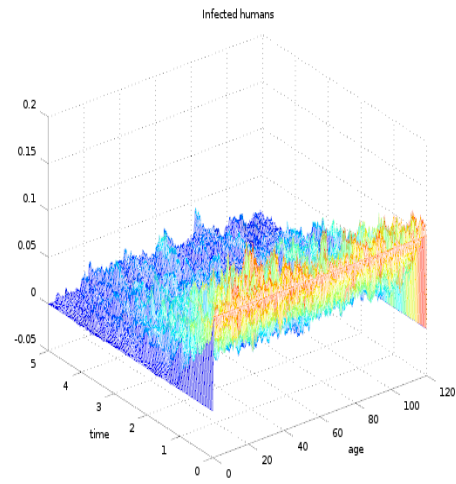
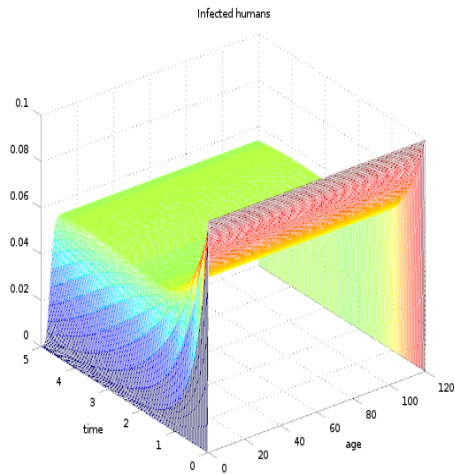
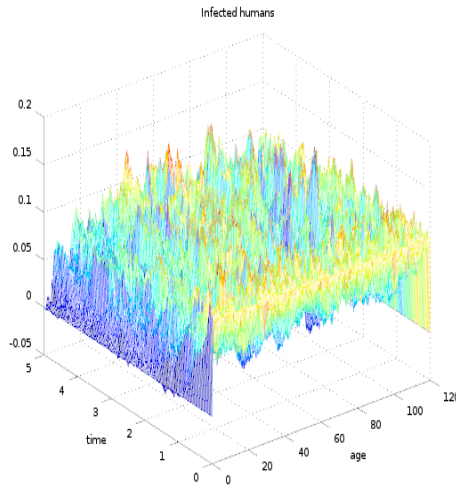
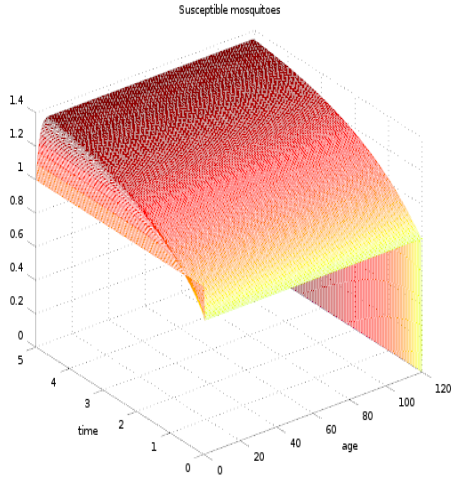
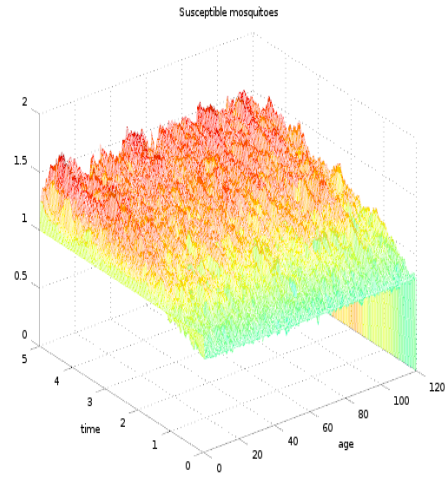
(a) Deterministic model when  $\mathcal{R}_0 < 1$ (b) Stochastic model when  $\mathcal{R}_0 < 1$ (c) Deterministic model when  $\mathcal{R}_0 > 1$ (d) Stochastic model when  $\mathcal{R}_0 > 1$ 

Figure 4: Figure 4 compare infected human individuals for both deterministic and stochastic models. In the case of  $\mathcal{R}_0 < 1$ , the infected individuals disappear over time whereas an endemic state is reached when  $\mathcal{R}_0 > 1$ .

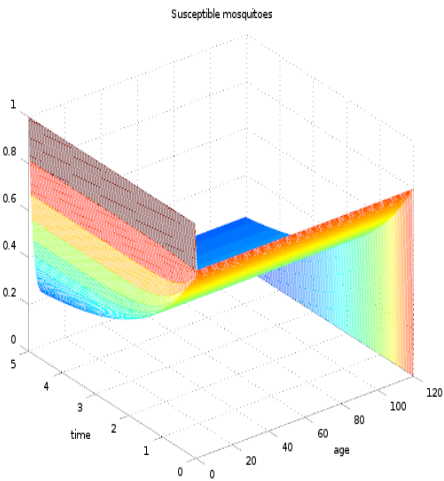




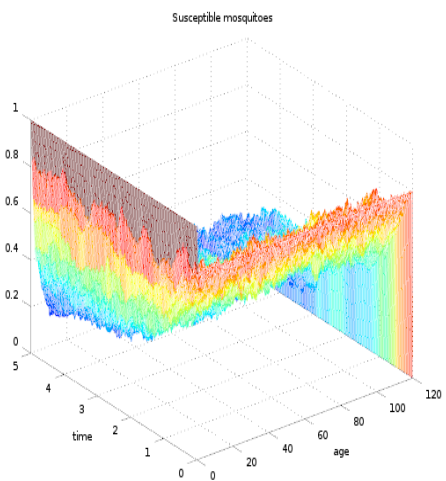
(a) Susceptible mosquitoes when  $\mathcal{R}_0 < 1$  for the deterministic model



(b) Stochastic model when  $\mathcal{R}_0 < 1$



(c) Deterministic model when  $\mathcal{R}_0 > 1$



(d) Stochastic model when  $\mathcal{R}_0 > 1$

Figure 5: Susceptible mosquito population compared in the deterministic and stochastic models

## 7 Conclusion

In this work, we considered an age-structured model and introduced a per-

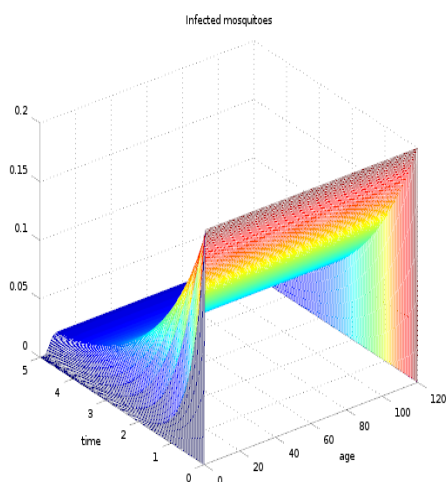
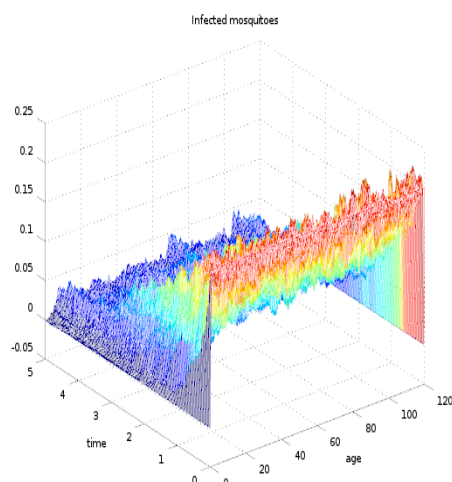
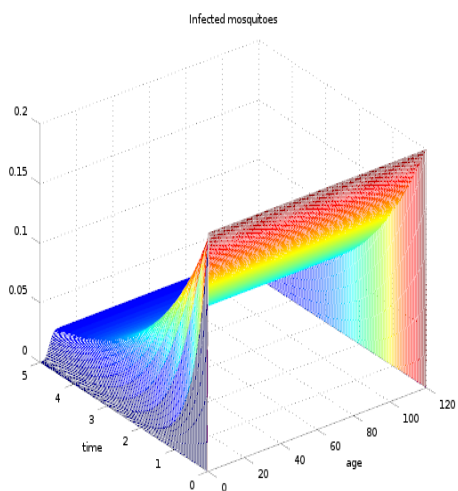
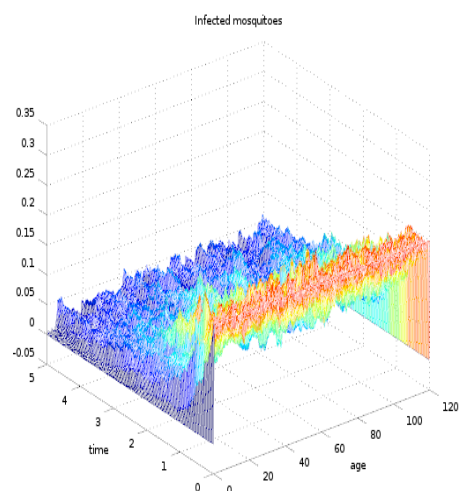
(a) Deterministic model when  $\mathcal{R}_0 < 1$ (b) Stochastic model when  $\mathcal{R}_0 < 1$ (c) Infected mosquitoes when  $\mathcal{R}_0 > 1$ (d) Stochastic case when  $\mathcal{R}_0 > 1$ 

Figure 6: Comparison between the deterministic and stochastic cases for infected mosquitoes

turbation to the deterministic model. A comparison between sample paths and deterministic trajectories has been presented. We observed that, when  $\mathcal{R}_0 < 1$ , the perturbation is inducing small variations in deterministic system

whereas remarkable differences appears when  $\mathcal{R}_0 > 1$ . The parameters of the basic reproduction number show that reducing the contact human-mosquito has large effect in controlling the disease. Therefore, much effort is required to reduce mosquito biting rate by using protectives tools such insecticide treated bed nets and indoor residual spraying. In areas of high mosquitoes concentration, reducing the number of biting producing infections in humans is to be included for an eradication of the disease; more efforts to obtain effective vaccine are necessary. Effects of migrations on the present model are to be investigated for future works.

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