# A stochastic model for Malaria & its behavior under insecticide-treated nets

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#### Abstract

Stochastic model for Malaria transmission is introduced, and its behavior under insecticidetreated nets (which is used as a prevention) is considered. A white noise is introduced into the model, representing fluctuations in the environment that manifest them-selves naturally on the transmission coefficient rate. Existence and uniqueness of a global positive solution of the stochastic model is proved, as well as the conditions under which extinction and persistence in mean hold are establish. Numerical simulations are provided which illustrate the theoretical results and conclusions are derived on the impact of the fluctuations which are caused by the environment.

**Keywords:** Stochastic nonlinear model, malaria, Brownian motion, insecticide-treated nets, positivity, extinction, persistence, simulations.

#### Mathematics Subject Classification 2010: 34F05, 60H10, 92D30

# 1 Introduction

Malaria is a life threatening disease caused by *Plasmodium* parasites and transmitted from one individual to another by the bite of infected female anopheline mosquitoes [6, 37]. Malaria is a serious and sometimes fatal disease caused by a parasite that commonly infects a certain type of mosquito which feeds on humans. When the parasites get in to the the human body, they multiply in the liver, and then they infect red blood cells. Following World Health Organization (WHO) 2021 report, it is estimated that that in 2020, there were an estimated 241 million cases of malaria worldwide, with populations living in sub-Saharan Africa having the highest risk of acquiring malaria [41]. People who get Malaria are typically very sick with high fevers, shaking chills, and flu-like illness. Although malaria can be a deadly disease, illness and death from malaria can usually be prevented. Malaria is an entirely preventable and treatable disease, provided the currently recommended interventions are properly implemented. Following WHO, these interventions include (i) vector control through the use of insecticide-treated nets (ITNs), indoor residual spraying (IRS) and, in some specific settings, larval control, (ii) preventive chemotherapies for the most vulnerable populations, particularly pregnant women and infants, (iii) vaccine – from October 2021, WHO also recommends broad use of the RTS, S/AS01 malaria vaccine among children living in regions with moderate to high P. falciparum malaria transmission. The vaccine

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has been shown to significantly reduce malaria, and deadly severe malaria, among young children [41]. Furthermore, WHO recommends that all suspected cases of malaria be confirmed using parasite-based diagnostic testing (through either microscopy or a rapid diagnostic test), where this testing enables health providers to swiftly distinguish between malarial and non-malarial fevers, facilitating appropriate treatment.

The best available treatment, particularly for P. falciparum malaria, is artemisinin-based combination therapy (ACT). The primary objective of treatment is to ensure the rapid and full elimination of Plasmodium parasites to prevent an uncomplicated case of malaria from progressing to severe disease or death.

An ITN is a mosquito net that repels, disables and/or kills mosquitoes coming into contact with insecticide on the netting material. ITNs are considered one of the most effective interventions against malaria [20]. In 2007, WHO recommended full ITN coverage of all people at risk of malaria, even in high-transmission settings [39]. By 2011, 32 countries in the African region and 78 other countries worldwide, had adopted the WHO recommendation. A total of 89 countries, including 39 in Africa, distribute ITNs free of charge. Between 2004 and 2010, the number of ITNs delivered annually by manufacturers to malaria-endemic countries in sub-Saharan Africa increased from 6 million to 145 million. However, the numbers delivered in 2011 and 2012 are below the number of ITNs required to protect all population at risk. There is an urgent need to identify new funding sources, or to reduce expenses in some way, to maintain and expand coverage levels of interventions so that outbreaks of disease can be avoided and international targets for reducing malaria cases and deaths can be attained [41].

A number of studies reported that ITN possession does not necessarily translate into use. Human behavior change interventions, including information, education, communication (IEC) campaigns and post-distribution hang-up campaigns are strongly recommended, especially where there is evidence of their effectiveness in improving ITN usage (see [1], [3], [17], [23], [27], [30], [31], [41] etc.). In [9], [40] authors described that WHO suggests not co-deploying ITNs and IRS and that priority be given to delivering either ITNs or IRS at optimal coverage and to a high standard, rather than introducing the second intervention as a means to compensate for deficiencies in the implementation of the first intervention, where significant of vector control methods is emphasised. Recently in the report [41] from last year, it is stated that manufacturers delivered about 229 million ITNs to malaria endemic countries in 2020, 24 million fewer than in 2019, which is significant information about the efficiency and usage of ITNs.

All those measures of prevention are under influence of spread of malaria, ie in order to prevent infection caused by malaria and to predict measures of protection (which would decrease the expenses of prevention) it is necessary to have knowledge about its spreads and behaviour.

Many authors described spread of malaria by different mathematical models, all of them have different approaches and established different results. Paper [34] by Smith and other authors illustrate the behaviour of malaria with simulations. Recently, Mbogo, Luboobi and Odhiambo in [25] introduce stochastic model with a idealisation of constant number for the population and randomness is described with certain probabilities of getting infected. The way of introducing stochasticity in to the mathematical model for description of the spread of malaria was also presented in the paper of Pemberton-Ross, Chitnis, Pothin, Smith in [28]. On the other hand, Le, Kumar, Ruiz in [19] presented stochastic model for malaria by giving the probabilities under which number of individuals would move from susceptible to infected and similar situations. Jovanović and Krstić in [15] introduced stochastic model for malaria by introducing Brownian motion into fluctuations around steady state point. We refer to recent articles regarding models for Malaria [2], [7], [18], [26], [33], [35], [44], etc.

There is many other different approach of introducing randomness via stochasticity, but the one which is presented in this paper is different comparing to the mentioned ones.

As the real infectious disease contains some kinds of random fluctuations caused by changes in the environment (per example weather change, migrations etc), we consider to describe the spread of Malaria via perturbation by Brownian motion. Ie, our aim is to improve the deterministic epidemic model by Agusto, Del Valle, Blayneh, Ngonghala, Goncalves, Li, Zhao and Gong in [5] for the effects of ITNs on the transmission dynamics of malaria infection, by considering environmental white noise. Following [4, 36] we introduce a stochastic noise in the form of a two independent Brownian motions with positive intensity. To the best of our knowledge, it is the first time that a stochastic compartmental model for this type of nonlinear model for malaria is analysed. Moreover, it should be noted, that the impact of the fluctuations in the environment on the number of individuals who get infected with Malaria is an important fact, from the economic and social point of view, due to the costs of insecticide-treated nets and other measures of prevention.

The paper is organized as follows: Section 2 presents a compartmental stochastic model for Malaria transmission where the susceptible individuals that are under insecticide-treated nets are protected against infection of malaria and have smaller chances to get infected. Section 3 is devoted to existence and uniqueness of a global positive solution of the stochastic system (cf. Theorem 3.1). In Section 4 conditions for the extinction of the disease of Malaria within the population of humans as well as vectors are given (cf. Theorem 4.1). In Section 5 conditions for the persistence in mean of Malaria within the population of humans as well as vectors (cf. Theorem 5.1) are established. Numerical simulations are developed in Subsection 4.1 and Subsection 5.1, illustrating both theoretical results of extinction and persistence, respectively, and even more, in both cases, comparison with appropriate deterministic model is obtained. The paper ends with the Section 6 with discussion of the results and conclusions.

# 2 Malaria model

Agusto et al. in [5] introduced the model where they considered transmission of Malaria infection of mosquito (also referred as vector) and human (also referred as host) population. The host population is divided into two compartments, susceptible  $(S_h)$  and infectious  $(I_h)$ , with a total population  $(N_h)$  given by  $N_h(t) = S_h(t) + I_h(t)$  in each moment  $t \ge 0$ . Analogously, the vector population is divided into two compartments, susceptible  $(S_v)$  and infectious  $(I_v)$ , with a total population  $(N_v)$  given by  $N_v(t) = S_v(t) + I_v(t)$ , in each moment  $t \ge 0$ . The model is constructed under the following assumptions: all newborns individuals are assumed to be susceptible and no infected individuals are assumed to come from outside the community. The human and mosquito recruitment rates are denoted by  $\Lambda_h$  and  $\Lambda_v$ , respectively. The disease is fast progressing, thus the exposed stage is minimal and is not considered. Infectious individuals can die from the disease or become susceptible after recovery, the mosquito population does not recover from infection, ITNs contribute for the mortality of mosquitoes. The average number of bites per mosquito per unit of time (mosquito-human contact rate) is given by

$$\beta = \beta_{max}(1-b) \,,$$

where  $\beta_{max}$  denotes the maximum transmission rate and b the proportion of ITN usage. It is assumed that the minimum transmission rate is zero.

Note that the parameters  $\beta_{max}$  and  $\beta_{min}$  are the maximum and the minimum transmission rates, respectively, and b is the proportion of bed-net usage that could reduce the mosquito – human contact rate to a minimum level  $\beta_{min}$ .

Bed-nets are typically used at night, thus, we assume that even if the entire host population used bed-nets (b = 1), the transmission can only be reduced to a minimum value  $(\beta_{min})$ . Similarly, if no one uses bed-nets (b = 0), transmission would be at its maximum level  $(\beta_{max})$ .

The value of  $\beta$  is the same for human and mosquito population, so the average number of bites per human per unit of time is  $\beta N_v/N_h$  (see [5] and the references cited therein). Thus, the force of infection for susceptible humans ( $\lambda_h$ ) and susceptibles vectors ( $\lambda_v$ ) are given by

$$\lambda_h = rac{p_h eta I_v}{N_h} \quad ext{and} \quad \lambda_v = rac{p_v eta I_h}{N_h} \,,$$

where  $p_h$  and  $p_v$  are the transmission probability per bite from infectious mosquitoes to humans, and from infectious humans to mosquitoes, respectively. The death rate of the mosquitoes is modeled by  $\mu_{vb} = \mu_{v1} + \mu_{max}b$ , where  $\mu_{v1}$  is the natural death rate and  $\mu_{max}b$  is the death rate due to pesticide on ITNs.

Due to insecticide treatment of bed-nets, female mosquitoes questing for blood meal could die when they become in contact with a treated bed-net. Therefore, we have modeled the death rate of the mosquitoes as  $\mu_v(b) = \mu_{v1} + \mu_{max}b, 0 \leq b \leq 1$ , where  $\mu_{v1}$  is the natural death rate and  $\mu_{max}b$  is the death rate due to pesticide on treated bed-nets, taken as a linear function of b.

In the sequel the notation  $\beta$  will be use for  $\beta(b)$ , and  $\mu_{vb}$  for  $\mu_{vb}(b)$ .

In mentioned paper [5], authors described the state system of the deterministic malaria model in the following way:

$$\begin{cases} \dot{S}_{h}(t) = \Lambda_{h} - \frac{p_{h}\beta I_{v}(t)}{N_{h}(t)}S_{h}(t) + \gamma_{h}I_{h}(t) - \mu_{h}S_{h}(t) ,\\ \dot{I}_{h}(t) = \frac{p_{h}\beta I_{v}(t)}{N_{h}(t)}S_{h}(t) - (\mu_{h} + \gamma_{h} + \delta_{h})I_{h}(t) ,\\ \dot{S}_{v}(t) = \Lambda_{v} - \frac{p_{v}\beta I_{h}(t)}{N_{h}(t)}S_{v}(t) - \mu_{vb}S_{v}(t) ,\\ \dot{I}_{v}(t) = \frac{p_{v}\beta I_{h}(t)}{N_{h}(t)}S_{v}(t) - \mu_{vb}I_{v}(t) , \end{cases}$$
(1)

where the values of the parameters  $\delta_h$  is disease induced mortality rate in humans,  $\gamma_h$  is recovery rate of infectious humans to be susceptible,  $\mu_h$  is natural mortality rate in humans while other coefficients are already presented.

Since the system (1) represents human and mosquito populations, all parameters in the model are non-negative and it is shown in [5] that the solutions of the system are non-negative, given non-negative initial values.

Further, following [5], the biologically feasible region is given by:

$$\Omega = \Omega_h \times \Omega_v \subset \mathbb{R}^2_+ \times \mathbb{R}^2_+ \tag{2}$$

where

$$\Omega_h = \left\{ (S_h(t), I_h(t)) \in \mathbb{R}^2_+ : 0 \leqslant N_h(t) \leqslant \frac{\Lambda_h}{\mu_h} \right\}$$

and

$$\Omega_v = \left\{ (S_v(t), I_v(t)) \in \mathbb{R}^2_+ : 0 \leqslant N_v(t) \leqslant \frac{\Lambda_v}{\mu_{vb}} \right\} \,.$$

**Lemma 2.1** (Agusto, Del Valle, Blayneh, Ngonghala, Goncalves, Li, Zhao and Gong see [5]). The region  $\Omega = \Omega_h \times \Omega_v \subset \mathbb{R}^2_+ \times \mathbb{R}^2_+$  is positively invariant for the model (1) with non-negative initial conditions in  $\mathbb{R}^4_+$ .

Adding the first two equations and the last two equations of the system (1) gives

$$\begin{cases} \dot{N}_h(t) = \Lambda_h - \mu_h N_h(t) - \delta_t I_h(t) \\ \dot{N}_v(t) = \Lambda_v - \mu_{vb} N_v(t) . \end{cases}$$

Thus, it follows that

$$\begin{cases} \dot{N}_h(t) \leqslant \Lambda_h - \mu_h N_h(t) \\ \dot{N}_v(t) = \Lambda_v - \mu_{vb} N_v(t) \end{cases}$$

We propose a system of stochastic differential equations where the fluctuations in the environment are assumed to manifest themselves as fluctuations in the parameters  $p_h$  and  $p_v$ . This is natural way of introducing randomness from the environment, regarding that unpredictable influences of the environment have a reflection in probabilities of transmission of malaria. Probabilities are perturbed in the following way;

$$p_h \to p_h + \sigma_1 \dot{B}_1(t)$$
 and  $p_v \to p_v + \sigma_2 \dot{B}_2(t)$ 

where  $B_i(t)$ , i = 1, 2, are standard independent Brownian motions with intensity  $\sigma_i^2 > 0$ , respectively.

New stochastic nonlinear model for spread of Malaria is given by the following system of stochastic differential equations:

$$\begin{cases} dS_{h}(t) = \left[\Lambda_{h} - \frac{p_{h}\beta I_{v}(t)}{N_{h}(t)}S_{h}(t) + \gamma_{h}I_{h}(t) - \mu_{h}S_{h}(t)\right]dt - \sigma_{1}\frac{\beta I_{v}(t)}{N_{h}(t)}S_{h}(t)dB_{1}(t), \\ dI_{h}(t) = \left[\frac{p_{h}\beta I_{v}(t)}{N_{h}(t)}S_{h}(t) - (\mu_{h} + \gamma_{h} + \delta_{h})I_{h}(t)\right]dt + \sigma_{1}\frac{\beta I_{v}(t)}{N_{h}(t)}S_{h}(t)dB_{1}(t), \\ dS_{v}(t) = \left[\Lambda_{v} - \frac{p_{v}\beta I_{h}(t)}{N_{h}(t)}S_{v}(t) - \mu_{vb}S_{v}(t)\right]dt - \sigma_{2}\frac{\beta I_{h}(t)}{N_{h}(t)}S_{v}(t)dB_{2}(t), \\ dI_{v}(t) = \left[\frac{p_{v}\beta I_{h}(t)}{N_{h}(t)}S_{v}(t) - \mu_{vb}I_{v}(t)\right]dt + \sigma_{2}\frac{\beta I_{h}(t)}{N_{h}(t)}S_{v}(t)dB_{2}(t). \end{cases}$$
(3)

Another motivation for generalisation of the model (1) comes from the paper of Zhao and Jiang [42], where they discussed the stochastic *SIV* system with three  $B_i$ , i = 1, ..., 3, independent Brownian motions, and where  $\sigma_i$  are their intensities.

Following [21] and [43], there are two interesting questions yet to be answered for the system (3)

- In which sense and under what conditions will the disease be persistent?
- Is there a threshold for the noises to determine the extinction of the disease?

# 3 Global positive solution

In order to examine the properties of the model (3) which will describe the spread of malaria, existence of positive solution of the system of stochastic differential equations in necessary.

**Theorem 3.1.** For any initial value  $(S_h(0), I_h(0), S_v(0), I_v(0)) \in \mathbb{R}^4_+$ , there is a unique positive solution  $(S_h(t), I_h(t), S_v(t), I_v(t))$  of system (3) on  $t \ge 0$  and the solution will remain positive with probability one, that is to say,  $(S_h(t), I_h(t), S_v(t), I_v(t)) \in \mathbb{R}^4_+$  for all  $t \ge 0$  almost surely.

*Proof.* The proof is motivated by papers [10]-[13], [22] and [38].

Let  $k_0 > 0$  be sufficiently large such that values  $S_h(0), I_h(0), S_v(0), I_v(0)$  lie within the interval  $[1/k_0, k_0]$ . For each integer  $k > k_0$ , let us define the stopping time

$$\tau_k = \inf\left\{t \in [0, \tau_0) : \min\{S_h(t), I_h(t), S_v(t), I_v(t)\} \leqslant \frac{1}{k} \text{ or } \max\{S_h(t), I_h(t), S_v(t), I_v(t)\} \geqslant k\right\},$$

where in the sequel of this paper, we set  $\inf \emptyset = \infty$  (as usual  $\emptyset$  denotes the empty set). According to the definition,  $\tau_k$  is increasing as  $k \to +\infty$ . Set  $\tau_{\infty} = \lim_{k \to +\infty} \tau_k$ , from what follows  $\tau_{\infty} \leq \tau_{\epsilon}$ , for some  $\epsilon \in (0, 1)$  a.s. In order to complete the proof, we need to prove that  $\tau_{\infty} = \infty$ .

Let us suppose the opposite, that there exist a pair of constants  $T \ge 0$  and  $\epsilon \in (0, 1)$  such that

$$P(\tau_{\infty} \leqslant T) \geqslant \epsilon$$

Hence there exists  $k_1 \ge k_0$  such that

$$P(\tau_k \leqslant T) \ge \epsilon \text{ for all } k \ge k_1.$$
(4)

If we sum first two equations from system (3), we obtain

$$d(S_h(t) + I_h(t)) = \Lambda_h - \mu_h N_h(t) - \delta_t I_h(t)$$
  

$$\Rightarrow dN_h(t) \leq \Lambda_h - \mu_h N_h(t)$$
  

$$\Leftrightarrow dN_h(t) \leq -\mu_h \left[ N_h(t) - \frac{\Lambda_h}{\mu_h} \right]$$
  

$$\Leftrightarrow N_h(t) \leq \frac{\Lambda_h}{\mu_h} + C_1 e^{-\mu_h t} \left[ N_h(0) - \frac{\Lambda_h}{\mu_h} \right].$$

From last

$$N_h(t) \leqslant \begin{cases} \frac{\Lambda_h}{\mu_h}, & N_h(0) \le \frac{\Lambda_h}{\mu_h}, \\ N_h(0), & N_h(0) > \frac{\Lambda_h}{\mu_h}. \end{cases} := N_h.$$

$$(5)$$

Similarly, by summing last two equations from system (3), we have

$$\begin{split} d(S_v(t) + I_v(t)) &= \Lambda_v - \mu_{vb}(S_v(t) + I_v(t)) \\ \Leftrightarrow dN_v(t) &= \mu_{vb} \left[ \frac{\Lambda_v}{\mu_{vb}} - N_v(t) \right] \\ \Leftrightarrow N_v(t) &= \frac{\Lambda_v}{\mu_{vb}} + C_1 e^{-\mu_{vb}t} \left[ N_v(0) - \frac{\Lambda_v}{\mu_{vb}} \right]. \end{split}$$

From last,

$$N_v(t) \leqslant \begin{cases} \frac{\Lambda_v}{\mu_{vb}}, & N_v(0) \le \frac{\Lambda_v}{\mu_{ub}}, \\ N_v(0), & N_h(0) > \frac{\Lambda_v}{\mu_{ub}}. \end{cases} := N_v.$$
(6)

Because the coefficients of system (3) satisfy linear growth conditions, there is a unique local solution on  $[0, \tau_0)$  for any initial value  $(S_h(0), I_h(0), S_v(0), I_v(0))$ , where  $\tau_0$  is known in the literature as the *explosion time*. It is necessary to prove that the solution is global, i.e., that  $\tau_0 = +\infty$  almost surely (*a.s.*, for brevity). Let us define twice differentiable function  $V : R^4_+ \to R_+ \cup \{0\}$  in following way

$$V(S_{h}(t), I_{h}(t), S_{v}(t), I_{v}(t)) = \left(S_{h}(t) - a_{1} - a_{1}\log\frac{S_{h}(t)}{a_{1}}\right) + \left(I_{h}(t) - a_{2} - a_{2}\log\frac{S_{h}(t)}{a_{2}}\right) \\ + \left(S_{v}(t) - a_{3} - a_{3}\log\frac{S_{h}(t)}{a_{3}}\right) + \left(I_{v}(t) - a_{4} - a_{4}\log\frac{S_{h}(t)}{a_{4}}\right),$$

where  $a_1, a_2, a_3, a_4$  are positive constants to be determined later. Function V is nonnegative, because  $\log x \leq x - 1$  for every  $x \ge 0$ . Applying well known Ito formula on function V, we have

$$dV(S_{h}(t), I_{h}(t), S_{v}(t), I_{v}(t)) = L(S_{h}(t), I_{h}(t), S_{v}(t), I_{v}(t))dt + \left\{ -\left(1 - \frac{a_{1}}{S_{h}(t)}\right)\sigma_{1}\frac{\beta I_{v}(t)}{N_{h}(t)}S_{h}(t) + \left(1 - \frac{a_{2}}{I_{h}(t)}\right)\sigma_{1}\frac{\beta I_{v}(t)}{N_{h}(t)}S_{h}(t) \right\} dB_{1}(t) + \left\{ -\left(1 - \frac{a_{3}}{S_{v}(t)}\right)\sigma_{2}\frac{\beta I_{h}(t)}{N_{h}(t)}S_{v}(t) + \left(1 - \frac{a_{4}}{I_{v}(t)}\right)\sigma_{2}\frac{\beta I_{h}(t)}{N_{h}(t)}S_{v}(t) \right\} dB_{2}(t).$$
(7)

Where

$$\begin{split} L(S_{h}(t), I_{h}(t), S_{v}(t), I_{v}(t)) \\ &\leqslant C + \left(1 - \frac{a_{1}}{S_{h}(t)}\right) \left[\Lambda_{h} - \frac{p_{h}\beta I_{v}(t)}{N_{h}(t)}S_{h}(t) + \gamma_{h}I_{h}(t) - \mu_{h}S_{h}(t)\right] \\ &+ \left(1 - \frac{a_{2}}{I_{h}(t)}\right) \left[\frac{p_{h}\beta I_{v}(t)}{N_{h}(t)}S_{h}(t) - (\mu_{h} + \gamma_{h} + \delta_{h})I_{h}(t)\right] \\ &+ \left(1 - \frac{a_{3}}{S_{v}(t)}\right) \left[\Lambda_{v} - \frac{p_{v}\beta I_{h}(t)}{N_{h}(t)}S_{v}(t) - \mu_{vb}S_{v}(t)\right] \\ &+ \left(1 - \frac{a_{4}}{I_{v}(t)}\right) \left[\frac{p_{v}\beta I_{h}(t)}{N_{h}(t)}S_{v}(t) - \mu_{vb}I_{v}(t)\right] \\ &\leqslant C_{1} + I_{h}(t) \left(a_{3}p_{v}\beta - \frac{a_{1}}{N_{h}}\gamma_{h}\right), \end{split}$$

for some generic constant  $C_1$ . We can choose constants  $a_2 = a_4 = 1$ , and  $a_1, a_3$  such that

$$a_3 p_v \beta - \frac{a_1}{N_h} \gamma_h \leqslant 0.$$

If we substitute last in (8), and afterwards substitute this bound in (7), we have

$$dV(S_{h}(t), I_{h}(t), S_{v}(t), I_{v}(t)) \leq C_{2} + \left\{ -\left(1 - \frac{a_{1}}{S_{h}(t)}\right) \sigma_{1} \frac{\beta I_{v}(t)}{N_{h}(t)} S_{h}(t) + \left(1 - \frac{a_{2}}{I_{h}(t)}\right) \sigma_{1} \frac{\beta I_{v}(t)}{N_{h}(t)} S_{h}(t) \right\} dB_{1}(t) + \left\{ -\left(1 - \frac{a_{3}}{S_{v}(t)}\right) \sigma_{2} \frac{\beta I_{h}(t)}{N_{h}(t)} S_{v}(t) + \left(1 - \frac{a_{4}}{I_{v}(t)}\right) \sigma_{2} \frac{\beta I_{h}(t)}{N_{h}(t)} S_{v}(t) \right\} dB_{2}(t),$$
(9)

for generic constant  $C_2$ . Integrating Eq. (9) from 0 to  $\tau_k \wedge T = \min\{\tau_k, T\}$  and then taking the expectation on both sides, we have

$$V(S_h(\tau_k \wedge T), I_h(\tau_k \wedge T), S_v(\tau_k \wedge T), I_v(\tau_k \wedge T)) \leq V(S_h(0), I_h(0), S_v(0), I_v(0)) + C_2 T.$$
(10)

Let  $A_k = \{\tau_k \leq T\}$  for  $k \geq k_1$ , and from (4) it follows that  $P(A_k) \geq \epsilon$ . Even more, for every  $\omega \in a_k$ , at least one of variables  $S_h, I_h, S_v, I_v$  or A is less than equal  $\frac{1}{k}$ , or it is greater or equal to k. Than, function  $V(S_h(\tau_k), I_h(\tau_k), S_v(\tau_k), I_v(\tau_k))$  is not less than

$$k - 1 - \log k$$
 or  $\frac{1}{k} - 1 - \log \frac{1}{k}$ ,

ie

$$V(S_h(\tau_k), I_h(\tau_k), S_v(\tau_k), I_v(\tau_k)) \ge \min\left\{k - 1 - \log k, \frac{1}{k} - 1 + \log k\right\}.$$

From (4) and (10) it follows that

$$E\Big(V(S_h(0), I_h(0), S_v(0), I_v(0)\Big) + C_2T \ge \epsilon \min\left\{k - 1 - \log k, \frac{1}{k} - 1 + \log k\right\},\$$

where  $I_{A_k}$  is usual notation for indicator of set  $A_k$ . If we let  $k \to +\infty$ , we obtain

$$+\infty > E\Big(V(S_h(0), I_h(0), S_v(0), I_v(0)\Big) + C_2T = +\infty,$$

which is a contradiction. This leads us that our assumption  $P(\tau_{\infty} \leq T) \geq \epsilon$  is wrong, it follows that  $\tau_{\infty} = \infty$  a.s.

Remark 3.1. It should be noted that from (5) and (6) the set

$$\Gamma^* = \left\{ (S_h(t), I_h(t), S_v(t), I_v(t)), S_h(t) > 0, I_h(t) > 0, S_v(t) > 0, I_v(t) > 0, \\ S_h(t) + I_h(t) \leqslant \frac{\Lambda_h}{\mu_h}, S_v(t) + I_v(t) \leqslant \frac{\Lambda_v}{\mu_{vb}} \right\}.$$
(11)

is a positively invariant set of system (3), which is similar to  $\Gamma$  of system (1).

## 4 Extinction

One of the most important issues, i.e. the problem which have to be solved, is the question under which conditions the Malaria will go to extinction. Extinction is necessary within the host and the vector also, because, in the case it is only extinct within the hosts, it will in some moment again be translated from vector to host.

Regarding that stochastic model which is here presented is influenced by two independent Brownian motions, boundaries for the intensities of Brownian motions will provide extinction of Malaria in both groups of populations.

In this section, we prove a condition for the extinction of the Malaria.

**Theorem 4.1.** For any initial value  $(S_h(0), I_h(0), S_v(0), I_v(0)) \in \mathbb{R}^4_+$ , such that solution of the system (3) is in  $\Gamma^*$  for every  $t \ge 0$ , if

1) 
$$\sigma_1^2 \ge \frac{p_h^2}{2(\mu_h + \gamma_h + \delta_h)}$$
, then  
 $I_h(t) \to 0, t \to +\infty \ a.s.,$ 

and

$$S_h(t) \to \frac{\Lambda_h}{\mu_h}, t \to +\infty \ a.s.$$

2) 
$$\sigma_2^2 \geqslant rac{p_v^2}{2\mu_{vb}}$$
 then

$$I_v(t) \to 0, t \to +\infty \ a.s.,$$

and

$$S_v(t) \to \frac{\Lambda_v}{\mu_{vb}}, t \to +\infty \ a.s.$$

*Proof.* Regarding that in every moment t we have that

$$N_h(t) = S_h(t) + I_h(t), \ N_v(t) = S_v(t) + I_v(t),$$

we can express

$$S_h(t) = N_h(t) - I_h(t), \ S_v(t) = N_v(t) - I_v(t),$$

for each  $t \ge 0$ .

So system (3) becomes following one

$$\begin{cases} dI_{h}(t) = \left[\frac{p_{h}\beta I_{v}(t)}{N_{h}(t)}(N_{h}(t) - I_{h}(t)) - (\mu_{h} + \gamma_{h} + \delta_{h})I_{h}(t)\right] dt + \sigma_{1}\frac{\beta I_{v}(t)}{N_{h}(t)}(N_{h}(t) - I_{h}(t))dB_{1}(t), \\ dI_{v}(t) = \left[\frac{p_{v}\beta I_{h}(t)}{N_{h}(t)}(N_{v}(t) - I_{v}(t)) - \mu_{vb}I_{v}(t)\right] dt + \sigma_{2}\frac{\beta I_{h}(t)}{N_{h}(t)}(N_{v}(t) - I_{v}(t))dB_{2}(t). \end{cases}$$
(12)

1) If we apply Ito formula on  $\ln I_h(t)$  we obtain,

$$\ln I_{h}(t) = \left\{ \frac{1}{I_{h}(t)} \left[ \frac{p_{h}\beta I_{v}(t)}{N_{h}(t)} (N_{h}(t) - I_{h}(t)) - (\mu_{h} + \gamma_{h} + \delta_{h})I_{h}(t) \right] - \frac{1}{2I_{h}^{2}(t)} \sigma_{1}^{2} \frac{\beta I_{v}^{2}(t)}{N_{h}^{2}(t)} (N_{h}(t) - I_{h}(t))^{2} \right\} dt$$

$$+ \frac{1}{I_{h}(t)} \sigma_{1} \frac{\beta I_{v}(t)}{N_{h}(t)} (N_{h}(t) - I_{h}(t)) dB_{1}(t)$$

$$= \dots$$

$$= \left\{ -\frac{1}{2} \left[ \frac{p_{h}}{\sigma_{1}} - \frac{\sigma_{1}\beta I_{v}(t)}{N_{h}(t)I_{h}(t)} (N_{h}(t) - I_{h}(t)) \right]^{2} + \frac{p_{h}^{2}}{2\sigma_{1}^{2}} - (\mu_{h} + \gamma_{h} + \delta_{h}) \right\} dt$$

$$+ \sigma_{1} \frac{\beta I_{v}(t)}{N_{h}(t)I_{h}(t)} (N_{h}(t) - I_{h}(t)) dB_{1}(t).$$

Integrating from 0 to t both sides of the last expression and then dividing by t, we obtain

$$\frac{\ln I_h(t)}{t} \leqslant \frac{\ln I_h(0)}{t} + \frac{p_h^2}{2\sigma_1^2} - (\mu_h + \gamma_h + \delta_h) + \frac{M_h(t)}{t},$$
(13)

where

$$M_h(t) = \int_0^t \sigma_1 \frac{\beta I_v(t)}{N_h(t)I_h(t)} (N_h(t) - I_h(t)) dB_1(t).$$

Regarding that  $M_h(t)$  is integral with respect to the Brownian motion, this is local continuous martingale. Also, if we replace upper bound with t = 0 in  $M_h(t)$ , we have that  $M_h(0) = 0$ . Further we can find quadratic variation and obtain next limit,

$$\limsup_{t \to +\infty} \frac{\langle M_h, M_h \rangle_t}{t} \leqslant \sigma_1^2 \frac{\beta^2 I_v^2(t)}{N_h^2(t) I_h^2(t)} (N_h(t) - I_h(t))^2 < +\infty.$$

Last expression is finite, because

$$\left(\frac{N_h(t) - I_h(t)}{I_h(t)}\right)^2 = \left(\frac{I_h(t) - N_h(t)}{I_h(t)}\right)^2 \leqslant 1.$$

Applying the large number theorem for martingales (see [24]), we have that

$$\lim_{t \to +\infty} \frac{M_h(t)}{t} = 0 \quad a.s.$$

If we substitute condition of the theorem,

$$\frac{p_h^2}{2\sigma_1^2} - (\mu_h + \gamma_h + \delta_h) \leqslant 0 \Leftrightarrow \sigma_1^2 \geqslant \frac{p_h^2}{2(\mu_h + \gamma_h + \delta_h)},$$

in (13), we obtain that

$$\lim_{t \to +\infty} I_h(t) = 0, \ a.s.$$

If we sum first two equations from system (3), we obtain

$$d(S_h(t) + I_h(t)) = \Lambda_h - \mu_h(S_h(t) + I_h(t)) - \delta_t I_h(t).$$

Solving the last equation, we obtain that

$$S_h(t) + I_h(t) = e^{-\mu_h t} \left[ S_h(0) + I_h(0) + \int_0^t (\Lambda_h - \delta_t I_h(s)) e^{\mu_h s} \, ds \right].$$

Applying L'Hospital's rule, it follows that

$$\lim_{t \longrightarrow \infty} (S_h(t) + I_h(t)) = \frac{\Lambda_h}{\mu_h}.$$
 As  $I_h(t) \longrightarrow 0$ , a.s.,  $t \longrightarrow \infty$ , it follows that  $S_h(t) \longrightarrow \frac{\Lambda_h}{\mu_h}$  a.s.,  $t \longrightarrow \infty$ .

This completes first part of the theorem.

2) If we apply Ito formula on  $\ln I_v(t)$  we obtain,

$$\begin{aligned} \ln I_v(t) &= \left\{ \frac{1}{I_v(t)} \left[ \frac{p_v \beta I_h(t)}{N_h(t)} (N_v(t) - I_v(t)) - \mu_{vb} I_v(t) \right] - \frac{1}{2I_v^2(t)} \sigma_2^2 \frac{\beta^2 I_h^2(t)}{N_h^2(t)} (N_v(t) - I_v(t))^2 \right\} dt \\ &+ \frac{1}{I_v(t)} \sigma_2 \frac{\beta I_h(t)}{N_h(t)} (N_v(t) - I_v(t)) dB_2(t) \\ &= \dots \\ &= \left[ -\frac{1}{2} \left( \frac{p_v}{\sigma_2} - \frac{\sigma_2 \beta I_h(t)}{N_h(t) I_v(t)} (N_v(t) - I_v(t)) \right)^2 + \frac{p_v^2}{2\sigma_2^2} - \mu_{vb} \right] dt \\ &+ \frac{\sigma_2 \beta I_h(t)}{N_h(t) I_v(t)} (N_h(t) - I_v(t)) dB_2(t). \end{aligned}$$

Integrating from 0 to t both sides of the last expression and then dividing by t, we obtain

$$\frac{\ln I_v(t)}{t} \leqslant \frac{\ln I_v(0)}{t} + \frac{p_v^2}{2\sigma_2^2} - \mu_{vb} + \frac{M_v(t)}{t},\tag{14}$$

where

$$M_{v}(t) = \int_{0}^{t} \frac{\sigma_{2}\beta I_{h}(t)}{N_{h}(t)I_{v}(t)} (N_{h}(t) - I_{v}(t))dB_{2}(t).$$

Regarding that  $M_v(t)$  is integral with respect to the Brownian motion, this is local continuous martingale. Also, if we replace upper bound with t = 0 in  $M_v(t)$ , we have that  $M_v(0) = 0$ . Further we can find quadratic variation and obtain next limes,

$$\limsup_{t \to +\infty} \frac{\langle M_v, M_v \rangle_t}{t} \leqslant \left( \frac{\sigma_2 \beta I_h(t)}{N_h(t) I_v(t)} (N_v(t) - I_v(t)) \right)^2 < +\infty.$$

Last expression is finite, because

$$\left(\frac{N_v(t) - I_v(t)}{I_v(t)}\right)^2 = \left(\frac{I_v(t) - N_v(t)}{I_v(t)}\right)^2 \leqslant 1.$$

Applying the large number theorem for martingales (see [24]), we have that

$$\lim_{t \to +\infty} \frac{M_v(t)}{t} = 0 \quad a.s.$$

If we substitute condition of the theorem,

$$\frac{p_v^2}{2\sigma_2^2}-\mu_{vb}\leqslant 0\Leftrightarrow \sigma_2^2\geqslant \frac{p_v^2}{2\mu_{vb}},$$

in (14), we obtain that

$$\lim_{t \to +\infty} I_v(t) = 0, \ a.s.$$

If we sum last two equations from system (3), we obtain

$$d(S_{v}(t) + I_{v}(t)) = \Lambda_{v} - \mu_{vb}(S_{v}(t) + I_{v}(t)).$$

Solving the last equation, we obtain that

$$S_{v}(t) + I_{v}(t) = e^{-\mu_{vb}t} \left[ S_{v}(0) + I_{v}(0) - t\Lambda_{v} \right].$$

Applying L'Hospital's rule, it follows that

$$\lim_{t \to 0} (S_v(t) + I_v(t)) = \frac{\Lambda_v}{\mu_{vb}}.$$
  
As  $I_v(t) \longrightarrow 0$ , a.s.,  $t \longrightarrow \infty$ , it follows that  $S_v(t) \longrightarrow \frac{\Lambda_v}{\mu_{vb}}$  a.s.,  $t \longrightarrow \infty$ 

This completes second part of the theorem.

### 4.1 Numerical simulation: example for extinction

In order to illustrate numerically Theorem 4.1 (and compare the behaivour of stochastic model with appropriate deterministic one), the values for parameters  $\Lambda_h$ ,  $\Lambda_\mu$ ,  $\mu_h$ ,  $\mu_{v1}$ ,  $\gamma_h$ ,  $\delta_h$ , b,  $\beta_{max}$  are taken from [5, 32]. The values of parameter b vary depending of the level of protection using bet-nets, and this influences the probabilities of infection  $p_h$  and  $p_v$ . Furthermore, the intensities of the noises and initial values for the latitude of the each group of populations for host and vector, depend on the concrete ambience, situation and case that won't be demonstrated within this paper.

Consider the parameter values from Table 1 from [5, 32].

Symbol	Description	Value
$\Lambda_h$	Recruitment rate in humans	$10^3/(70 \times 365)$
$\Lambda_v$	Recruitment rate in mosquitoes	$10^4/21$
$\mu_h$	Natural mortality rate in humans	$1/(70 \times 365)$
$\delta_h$	Disease induced mortality rate in humans	$10^{-3}$
$\gamma_h$	Recovery rate of infectious humans to be susceptible	1/4
$\mu_{v_1}$	Natural mortality rate of mosquitoes	1/21
$\mu_{max}b$	Mortality rate of mosquitoes due to treated net	1/21b
$\beta_{max}$	Maximum mosquito-human contact rate	0.1

Table 1: Parameter values for extinction, taken from [5, 32].

In the sequel, it will be shown that under different initial circumstances (different noises and initial values), the number of infected hosts and vectors will eventually go to zero (extinction), and number of susceptible hosts will tend to  $\frac{\Lambda_h}{\mu_h}$ , while number of susceptible vectors will tend to  $\frac{\Lambda_v}{\mu_{vb}}$ .

Remark 4.1. In the sequel in all the examples following notations will be used:

SH – susceptible hosts in stochastic model,	SHd – susceptible hosts in deterministic model
IH – infected hosts in stochastic model,	IHd – infected vectors in deterministic model
SV – susceptible vectors in stochastic model,	SVd – susceptible vectors in deterministic model
IV – infected vectors in stochastic model,	IVd – infected vectors in deterministic model

	-	-	

*Remark 4.2.* All numerical calculation and simulations presented in the sequel were done using Python.

**Example 1.** In order to presented that stochastic model is describing well the conditions from the environment, and that it behaves in the long-run distance as deterministic one, the initial state conditions  $(S_h(0), I_h(0), S_v(0), I_v(0)) \in \mathbb{R}^4_+$  are taken as in [5, 32]

$$S_h(0) = 800, \quad I_h(0) = 200, \quad S_v(0) = 4000, \quad I_v(0) = 1000.$$
 (15)

Also, probabilities are taken as in mention papers to be low in this case,  $p_h = p_v = 0.05$  because the intention is to present extinction of the Malaria, in the case as bed-nets usages is reasonable high, taken  $\beta = 0.05$ .

Further, the intensities  $\sigma_1, \sigma_2$  are chosen such that conditions of Theorem 4.1 are fulfilled, ie let

$$\sigma_1^2 = 0.4 \ge \frac{p_h^2}{2(\mu_h + \gamma_h + \delta_h)} \sim 0.00497, \qquad \sigma_1^2 = 0.2 \ge \frac{p_h^2}{2(\mu_h + \gamma_h + \delta_h)} \sim 0.014583.$$

therefore, it holds

$$I_h(t) \to 0, t \to +\infty \ a.s., \qquad S_h(t) \to \frac{\Lambda_h}{\mu_h} = 10^3, t \to +\infty \ a.s$$

Also,

$$I_v(t) \to 0, t \to +\infty \ a.s., \qquad S_v(t) \to \frac{\Lambda_v}{\mu_{vb}} = 5.5556 \times 10^3, t \to +\infty \ a.s.$$

This result can be illustrated in Figure 1, where the time scale is taken to be 10, and 100, to presented that in long time distance, variables will tend to estimated values.



Figure 1: Existnction of Malaria within host and vectors for  $\sigma_1^2 = 0.4, \sigma_2^2 = 0.2$ .

On Figure 2 it has been easily seen how stochastic models describes better randomness, but it fluctuates around deterministic values (time scale are different for the groups, because interaction between the vectors is bigger which influences spread between them to be fast).  $\star$ 

**Example 2.** For another initial values (such that they are in positively invariant set, i.e. taking

$$S_h(0) = 1000$$
,  $I_h(0) = 400$ ,  $S_v(0) = 5000$ ,  $I_v(0) = 2000$ . (16)

Also, probabilities are taken as in mention papers to be different,  $p_h = 0.06, p_v = 0.08$ , and with a stronger usage of bed-nets, ie  $\beta = 0.03$ .



Figure 2: Existnction of Malaria within host and vectors for  $\sigma_1^2 = 0.4, \sigma_2^2 = 0.2$ .

Then for

$$\sigma_1^2 = 0.1 \ge \frac{p_h^2}{2(\mu_h + \gamma_h + \delta_h)} \sim 0.00497, \qquad \sigma_1^2 = 0.4 \ge \frac{p_h^2}{2(\mu_h + \gamma_h + \delta_h)} \sim 0.014583$$

therefore, it holds

$$I_h(t) \to 0, t \to +\infty \ a.s., \qquad S_h(t) \to \frac{\Lambda_h}{\mu_h} = 10^3, t \to +\infty \ a.s.$$

Also,

$$I_v(t) \to 0, t \to +\infty \ a.s., \qquad S_v(t) \to \frac{\Lambda_v}{\mu_{vb}} = 5.5556 \times 10^3, t \to +\infty \ a.s.$$

This result can is illustrated in Figure 3, while on Figure 3 comaparison with deterministic model is obtained.



Figure 3: Existnction of Malaria within host and vectors for  $\sigma_1^2 = 0.1, \sigma_2^2 = 0.4$ .



Figure 4: Existnction of Malaria within host and vectors for  $\sigma_1^2 = 0.1, \sigma_2^2 = 0.4$ .

 $\star$ 

This is very useful and interesting result, because if the noises are big enough (in order to the environment is unpredictable which is realistic premise), if we start observing some habitat with both populations, hosts and vectors, starting with what ever population size, in time, Malaria will go to extinction.

# 5 Persistence

This section is dedicated to determine the conditions for the noises, in order to Malaria be persistence within humans and vectors.

**Definition 5.1.** System (3) is said to be persistent in mean if

$$\lim_{t \to \infty} \frac{1}{t} \int_0^t I_h(s) ds > 0 \ a.s., \ \lim_{t \to \infty} \frac{1}{t} \int_0^t I_v(s) ds > 0 \ a.s.$$

Let us introduce the notation  $\langle x(t) \rangle = \frac{1}{t} \int_0^t x(s) ds > 0.$ 

**Theorem 5.1.** The conditions for persistence of humans and mosquitos are given in the sequel. If:

1.

$$\sigma_1^2 \leqslant 2 \frac{p_h \beta \Lambda_h - \mu_h (\mu_h + \gamma_h + \delta_h)}{\mu_h \beta^2 N_u^2} \tag{17}$$

then

$$\liminf_{t \to +\infty} \langle I_h(t) \rangle \geq \frac{p_h \beta \frac{\Lambda_h}{\mu_h} - (\mu_h + \gamma_h + \delta_h) - \frac{\sigma_1^2 \beta^2 \Lambda_v^2}{2\mu_{vb}^2}}{p_h \beta \frac{\mu_h + \delta_h}{\mu_h} + \mu_h + \gamma_h + \delta_h}$$

2.

$$\sigma_2^2 \leqslant 2 \frac{p_v \beta \frac{\Lambda_v}{\mu_{vb}} - \mu_{vb}}{\beta^2 N_v^2},\tag{18}$$

then

$$\liminf_{t \to +\infty} \langle I_v(t) \rangle \ge \frac{p_v \beta \frac{\Lambda_v}{\mu_{vb}} - \mu_{vb} - \frac{\sigma_2^2 \beta^2 N_v^2}{2}}{p_v \beta + \mu_{vb}}.$$

*Proof.* 1. If we sum first two equations of system (3), we obtain

$$dS_h(t) + dI_h(t) = [\Lambda_h - (\mu_h + \delta_h)I_h(t) - \mu_h S_h(t)] dt.$$

If we integrate last expression and divide it with t, we obtain

$$\frac{S_h(t)-S_h(0)}{t} + \frac{I_h(t)-I_h(0)}{t} = \Lambda_h - (\mu_h + \delta_h)\langle I_h(t)\rangle - \mu_h\langle S_h(t)\rangle$$
$$\iff \langle S_h(t)\rangle = \frac{1}{\mu_h} \left(\Lambda_h - \frac{S_h(t)-S_h(0)}{t} - \frac{I_h(t)-I_h(0)}{t} - (\mu_h + \delta_h)\langle I_h(t)\rangle\right).$$

From last we have

$$\langle S_h(t) \rangle = \frac{\Lambda_h}{\mu_h} - \frac{K_1(t)}{\mu_h} - \frac{\mu_h + \delta_h}{\mu_h} \langle I_h(t) \rangle, \tag{19}$$

where

$$K_1(t) = \frac{S_h(t) - S_h(0)}{t} + \frac{I_h(t) - I_h(0)}{t}$$

Applying Ito's formula on  $d(\ln I_h(t) + I_h(t))$ , we obtain

$$d\ln I_h(s) + dI_h(s) = \left[\frac{p_h\beta I_v(s)S_h(s)}{N_h(s)} \left(\frac{1}{I_h(s)} + 1\right) - (\mu_h + \gamma_h + \delta_h)(I_h(s) + 1) - \frac{\sigma_1^2\beta^2 I_v^2(s)S_h^2(s)}{2I_h^2(s)N_h^2(s)}\right]dt + M_h(s),$$

 $\operatorname{for}$ 

$$M_{h}(t) = \frac{\sigma_{1}\beta I_{v}(t)S_{h}(t)}{I_{h}(t)N_{h}(s)}dB_{1}(t) + \frac{\sigma_{1}\beta I_{v}(t)S_{h}(t)}{N_{h}(s)}dB_{1}(t).$$

Further if we use the assumptions that for every  $t \ge 0$  we have  $1 \le S_h(s), I_h(s) \le N_h, I_v(s) \le N_v$ it follows that

$$d\ln I_h(s) + dI_h(s) \ge \left[\frac{p_h \beta S_h(t)}{N_h(s)I_h(s)} - (\mu_h + \gamma_h + \delta_h)I_h(t) - \frac{\sigma_1^2 \beta^2 N_v^2}{2}\right] dt + M_h(s).$$

If we integrate last expression from 0 to t and divide it with t, we obtain

$$\frac{\ln I_h(t) - \ln 0}{t} + \frac{I_h(t) - I_h(0)}{t} \ge p_h \frac{\beta \langle S_h(t) \rangle}{N_h^2} - (\mu_h + \gamma_h + \delta_h)) \langle I_h(t) \rangle - \frac{\sigma_1^2 \beta^2 N_v^2}{2} + \frac{\int_0^t M_h(s) ds}{t}.$$

If we substitute (19) in last expression, we than have

$$K_{2}(t) \geq p_{h}\beta \left(\frac{\Lambda_{h}}{\mu_{h}} - \frac{K_{1}(t)}{\mu_{h}} - \frac{\mu_{h} + \delta_{h}}{\mu_{h}} \langle I_{h}(t) \rangle \right) - (\mu_{h} + \gamma_{h} + \delta_{h}) \langle I_{h}(t) \rangle - \frac{\sigma_{1}^{2}\beta^{2}N_{v}^{2}}{2} + \frac{\int_{0}^{t} M_{h}(s)ds}{t},$$

$$(20)$$

where

$$K_2(t) = \frac{\ln I_h(t) - \ln I_h(0)}{t} + \frac{I_h(t) - I_h(0)}{t}$$

Regarding that  $M_h(t)$  is a local martingale, and  $M_h(0) = 0$ , it follows that

$$\lim_{t \longrightarrow +\infty} \frac{\int_0^t M_h(s) ds}{t} = 0 \ a.s.$$

Also,

$$\lim_{t \to +\infty} K_1(t) = \lim_{t \to +\infty} \left( \frac{S_h(t) - S_h(0)}{t} - \frac{I_h(t) - I_h(0)}{t} \right) = 0,$$
$$\lim_{t \to +\infty} K_2(t) = \lim_{t \to +\infty} \left( \frac{\ln I_h(t) - \ln I_h(0)}{t} + \frac{I_h(t) - I_h(0)}{t} \right) = 0.$$

Substituting estimates for  $M_h, K_1$  and  $K_2$  in (20), we obtain

$$\liminf_{t \to +\infty} \langle I_h(t) \rangle \geq \frac{p_h \beta \frac{\Lambda_h}{\mu_h} - (\mu_h + \gamma_h + \delta_h) - \frac{\sigma_1^2 \beta^2 \Lambda_v^2}{2\mu_{vb}^2}}{p_h \beta \frac{\mu_h + \delta_h}{\mu_h} + \mu_h + \gamma_h + \delta_h}.$$
(21)

If we apply condition (17) of the theorem in (21), we have that the desies is persistant within humans, ie the theorem is proved.

2.) If we sum last two equations of system (3), we obtain

$$dS_v(t) + dI_v(t) = \left[\Lambda_v - \mu_{vb}(I_v(t) + S_v(t))\right] dt.$$

If we integrate last expression and divide it with t, we obtain

$$\frac{S_v(t)-S_v(0)}{t} + \frac{I_v(t)-I_v(0)}{t} = \Lambda_v - \mu_{vb} \langle I_h(t) \rangle - \mu_{vb} \langle S_h(t) \rangle$$
$$\iff \langle S_v(t) \rangle = \frac{1}{\mu_{vb}} \left( \Lambda_v - \frac{S_v(t)-S_v(0)}{t} - \frac{I_v(t)-I_v(0)}{t} - \mu_{vb} \langle I_v(t) \rangle \right).$$

From last we have

$$\langle S_v(t) \rangle = \frac{\Lambda_v}{\mu_{vb}} - \frac{J_1(t)}{\mu_{vb}} - \langle I_v(t) \rangle, \qquad (22)$$

where

$$J_1(t) = \frac{S_v(t) - S_v(0)}{t} + \frac{I_v(t) - I_v(0)}{t}$$

Similarly as it was than for the group of humans, in the sequel we will proof persistence for the vectors.

Applying Ito's formula on  $d(\ln I_v(t) + I_v(t))$ , we obtain

$$d\ln I_v(s) + dI_v(s) = \left[ p_v \beta I_h(t) S_v(t) \left( \frac{1}{I_v(s)} + 1 \right) - \mu_{vb} - \mu_{vb} I_v(s) - \frac{\sigma_2^2 \beta^2 I_h^2(t) S_v^2(t)}{2N_h^2(t) I_v^2(t)} \right] dt + M_v(s)$$

for

$$M_{v}(t) = \frac{\sigma_{2}\beta I_{h}(t)S_{v}(t)}{N_{h}(t)I_{v}(t)}dB_{2}(s) + \frac{\sigma_{2}\beta I_{h}(t)S_{v}(t)}{N_{h}(t)}dB_{2}(s).$$

Further if we use the assumptions that for every  $t \ge 0$  we have  $1 \le S_v(s), I_v(s) \le N_v, I_h(s) \le N_h$ it follows that

$$d\ln I_{v}(s) + dI_{v}(s) \ge \left[ p_{v}\beta S_{v}(t) - \mu_{vb} - \mu_{vb}I_{v}(s) - \frac{\sigma_{2}^{2}\beta^{2}N_{v}^{2}}{2} \right] dt + M_{v}(s)$$

If we integrate last expression from 0 to t and divide it with t, we obtain

$$\frac{\ln I_v(t) - \ln I_v(0)}{t} + \frac{I_v(t) - I_v(0)}{t} \ge p_v \beta \langle S_v(t) \rangle - \mu_{vb} - \mu_{vb} \langle I_v(s) \rangle - \frac{\sigma_2^2 \beta^2 N_v^2}{2} + \frac{\int_0^t M_v(s) ds}{t}.$$

If we substitute (22) in (23), we than have

$$J_2(t) \ge p_v \beta \left(\frac{\Lambda_v}{\mu_{vb}} - \frac{J_1(t)}{\mu_{vb}} - \langle I_v(t) \rangle\right) - \mu_{vb} - \mu_{vb} \langle I_v(s) \rangle - \frac{\sigma_2^2 \beta^2 N_v^2}{2} + \frac{\int_0^t M_v(s) ds}{t}, \quad (23)$$

where

$$J_2(t) = \frac{\ln I_v(t) - \ln I_v(0)}{t} + \frac{I_v(t) - I_v(0)}{t}$$

Regarding that  $M_v(t)$  is a local martingale, and  $M_v(0) = 0$ , it follows that

$$\lim_{t \to +\infty} \frac{\int_0^t M_v(s) ds}{t} = 0 \ a.s. \ .$$

Also,

$$\lim_{t \to +\infty} J_1(t) = \lim_{t \to +\infty} \left( \frac{S_v(t) - S_v(0)}{t} - \frac{I_v(t) - I_v(0)}{t} \right) = 0,$$
$$\lim_{t \to +\infty} J_2(t) = \lim_{t \to +\infty} \left( \frac{\ln I_v(t) - \ln I_v(0)}{t} + \frac{I_v(t) - I_v(0)}{t} \right) = 0.$$

Substituting estimates for  $M_v, J_1$  and  $J_2$  in (23), we obtain

$$\liminf_{t \to +\infty} \langle I_v(t) \rangle \ge \frac{p_v \beta \frac{\Lambda_v}{\mu_{vb}} - \mu_{vb} - \frac{\sigma_2^2 \beta^2 N_v^2}{2}}{p_v \beta + \mu_{vb}}.$$

Proof of the theorem is complete.

## 5.1 Examples: numerical simulation for persistence

In order to illustrate persistence result, we will take a smaller population size (because for big ones it is almost impossible to predict persistence of the Malaria, it is dependent from the change of other parameters also)  $\Lambda_h = 100/(70 \times 365), \Lambda_v = \frac{1000}{21}$ . Further, we suppose that the transmission of the Malaria is on the highest level (from the literature refer)  $\beta - 0.1$ , and that usages of bed-nets is very small, which induces that rate of death of vectors is small,  $\mu_{vb} = 1/21$ .

**Example 3.** In order to presented that stochastic model is describing well the conditions from the environment, and that it behaves in the long time distance as deterministic one, the initial state conditions

$$S_h(0) = 50$$
,  $I_h(0) = 40$ ,  $S_v(0) = 400$ ,  $I_v(0) = 290$ . (24)

Also, probabilities are taken to be high  $p_h = p_v = 1$ , because the intention is to present persistence of the Malaria.

Further, the intensities  $\sigma_1, \sigma_2$  are chosen such that conditions of Theorem 5.1 are fulfilled, ie let

$$\sigma_1^2 = 0.0019 \leqslant 2 \frac{p_h \beta \Lambda_h - \mu_h (\mu_h + \gamma_h + \delta_h)}{\mu_h \beta^2 N_v^2} \sim 0.001975, \quad \sigma_2^2 = 0.019 \leqslant 2 \frac{p_v \beta \frac{\Lambda_v}{\mu_{vb}} - \mu_{vb}}{\beta^2 N_v^2} \sim 0.01999$$

therefore, it holds

$$\liminf_{t \longrightarrow +\infty} \langle I_h(t) \rangle \ge K_1$$

and

$$\liminf_{t \to +\infty} \langle I_v(t) \rangle \geq K_2,$$

where  $K_1, K_2$  are concrete constants.

This result can is illustrated in Figure 5, where the time scale is taken to be 100, and 150, to presented that in long time distance, variables will tend to estimated values.



Figure 5: Persistence of Malaria within host and vectors for  $\sigma_1^2 = 0.0019, \sigma_2^2 = 0.019$ .

On Figure 6 it has been easily seen how stochastic models describes better randomness, but it fluctuates around deterministic values (time scale are different for the groups, because interaction between the vectors is bigger which influences spread between them to be fast).  $\star$ 



Figure 6: Persistence of Malaria within host and vectors for  $\sigma_1^2 = 0.0019, \sigma_2^2 = 0.019$ .

**Example 4.** In order to presented that stochastic model is describing well the conditions from the environment, and that it behaves in the long time distance as deterministic one, the initial state conditions are

$$S_h(0) = 40$$
,  $I_h(0) = 50$ ,  $S_v(0) = 30$ ,  $I_v(0) = 49$ . (25)

Also, probabilities are taken to be high  $p_h = 0.8, p_v = 0.6$ .

Further, the intensities  $\sigma_1, \sigma_2$  are chosen such that conditions of Theorem 5.1 are fulfilled, i.e. let

$$\sigma_1^2 = 0.0009 \leqslant 2 \frac{p_h \beta \Lambda_h - \mu_h (\mu_h + \gamma_h + \delta_h)}{\mu_h \beta^2 N_v^2} \sim 0.001975, \quad \sigma_2^2 = 0.009 \leqslant 2 \frac{p_v \beta \frac{\Lambda_v}{\mu_{vb}} - \mu_{vb}}{\beta^2 N_v^2} \sim 0.01999$$

therefore, it holds

$$\liminf_{t \longrightarrow +\infty} \langle I_h(t) \rangle \geq K_1$$

and

$$\liminf_{t \longrightarrow +\infty} \langle I_v(t) \rangle \geq K_2,$$

where  $K_1, K_2$  are concrete constants.

This result can is illustrated in Figure 7, where the time scale is taken to be 10, and 100, to presented that in long time distance, variables will tend to estimated values.



Figure 7: Persistence of Malaria within host and vectors for  $\sigma_1^2 = 0.0009, \sigma_2^2 = 0.009$ .

On Figure 8 it has been easily seen how stochastic models describes better randomness, but it fluctuates around deterministic values (time scale are different for the groups, because



Figure 8: Persistence of Malaria within host and vectors for  $\sigma_1^2 = 0.0009, \sigma_2^2 = 0.009$ .

interaction between the vectors is bigger which influences spread between them to be fast).  $\star$ 

It should note that the lower bound for infected humans is a small number but strictly positive, and it will never go to zero. The fact that it is small is expected because now we are observing smaller groups of populations.

## 6 Conclusion and remarks

It should be noted that stochastic model introduce a certain realism in to the description of the spread of Malaria comparing with its deterministic compartment. Constant changes coming from the environment (weather changes such as floats, heavy rains and similar, migrations, economical crisis etc.) can be described with a stochastic model, while deterministic compartment can not catch this probable fluctuations of the values in the model. Size of the intensity of introduced noise describes the range of the values in stochastic model which is a reflection of how big those changes can be.

In subsections 4.1 and 5.1 numerical illustrations for two extreme scenarios are given, ie comparison of stochastic model and its deterministic compartment in case when Malaria goes to extinction or stay persistent. On Figures 2,4,6, and 8 it can be seen how trajectories of stochastic model oscillates over the deterministic path by which it allows certain range, and by this add realism, in the description of the spread of the Malaria.

It should emphasized that by taking  $\beta(b)$  to be dependent of bed-bet usage b, we somehow "control" the system and the populations. For different levels of bed-net usage, i.e. for different values for b, we have a different spread of the system values.

The next problem which would be realistic to observe is to take in to account the dependence of Brownian motions  $B_1$  and  $B_2$ . This has sense because there are a lot of natural influences, which affect both, humans and vectors, such as per example floods, earthquakes etc. By this, further work could be a stochastic model for Malaria (3) with noises  $\sigma_1$  and  $\sigma_2$ . Furthermore, jump can be introduce in the model via which big changes in the values which rise unpredictable could be described.

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