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A STOCHASTIC MODEL FOR THE SPREAD OF HUMAN PLASMODIA

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Abstract

We use stochastic differential equations (SDEs) to model the spread of the malaria parasite through a compartmental model of the type $(S_hL_hI_hR_hS_h\text{-}I_v)$. Considering the transmission rates $\overline{\beta}(t)$ and $\overline{\nu}(t)$ by introducing standard Brownian motion in order to render the ordinary differential equation into SDE, we obtain the existence and uniqueness of the solution.

0. Introduction

Malaria is one of the infectious diseases transmitted to humans by a highly anthropophilic mosquito, the (infected) female Anopheles, which generally bites between dusk and dawn. Its mathematical modelling dates back to the beginning of the 20th century [1]. Several mathematical models have been developed to study the dynamics of the spread of malaria. These include Ross's $(S_hI_hS_h\text{-}S_vI_vS_v)$ models, the $(S_hI_hR_hS_h\text{-}S_vI_v)$ models in [3, 4], and the $(S_hE_hI_hR_hS_h)$ models in [5, 6]. However, none of these models take account of the random nature of the transmission of parasites. In this model, in addition to *Plasmodium falciparum* transmission in a given area, we introduce any other random form of contamination by other parasites. This article is structured as follows: first we present some preliminary results, necessary for the conduct of our work, then we present our results relating to the stochastic approach to the spread of plasmodia. Finally, we simulate the trajectories of our results.

1. Mathematical Preliminaries and Notations

1.1. Ross's $(S_hI_hS_h\text{-}S_vI_vS_v)$ model

In 1911, Ross [1] proposed a model which took into account both anopheline and human populations. This model is certainly the starting point for vector-host models. In his model, Ross assumes that both the human and Anopheles populations are constant and that one mosquito bites "*a*" humans

per unit time where "a" is a constant [7]. The scheme for the spread of malaria in Ross's $(S_hI_hS_h\text{-}S_vI_vS_v)$ model is shown in Figure 1.

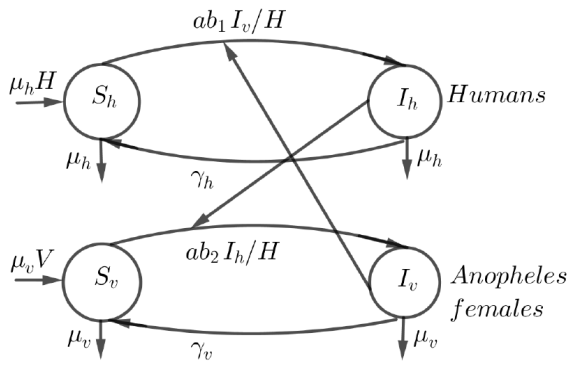


Figure 1. The final disease transmission graph.

He obtains the differential system (1) governing the $(S_hI_hS_h\text{-}S_vI_vS_v)$ model of malaria:

$$\begin{cases} \dfrac{dS_h}{dt} = \mu_h H - b_1 a I_v \dfrac{S_h}{H} - \gamma_h I_h - \mu_h I_h, \\ \dfrac{dI_h}{dt} = b_1 a I_v \dfrac{S_h}{H} - (\gamma_h + \mu_h) I_h, \\ \dfrac{dS_v}{dt} = b_2 a (V - I_v) \dfrac{I_h}{H} - (\gamma_v + \mu_v) I_v, \\ \dfrac{dI_v}{dt} = b_2 a (V - I_v) \dfrac{I_h}{H} - (\gamma_v + \mu_v) I_v. \end{cases} \tag{1}$$

After solving this system (1) by [7], we obtain the basic reproduction number R_0 defined by:

$$R_0 = mab_1 \frac{1}{\gamma} a \frac{1}{\mu} b_2 \tag{2}$$

and state the following theorem [8, 9]:

Theorem (Mosquito theorem)**.** (1) *If* $R_0 \leq 1$, *then disease disappears completely from the population after a certain time.*

(2) *If* $R_0 > 1$, *then disease remains endemic in the population.*

This so-called mosquito theorem can be used to determine the endemic character of epidemics in a given region.

1.2. Model $(S_hI_hR_hS_h\text{-}S_vI_v)$

Since malaria provides temporary immunity and is not lethal if treated, it is possible to use a SIRS (susceptible-infected-recovered-susceptible) model, since recovered individuals return to the S class with probability p $(p > 0)$ or relapsed individuals become infected again with probability $1 - p$. To Ross's $(S_hI_hS_h\text{-}S_vI_vS_v)$ model, we add the R reinstated compartment. These types of models are also solved by [10, 11]. Figure 2 illustrates the scheme of disease progression.

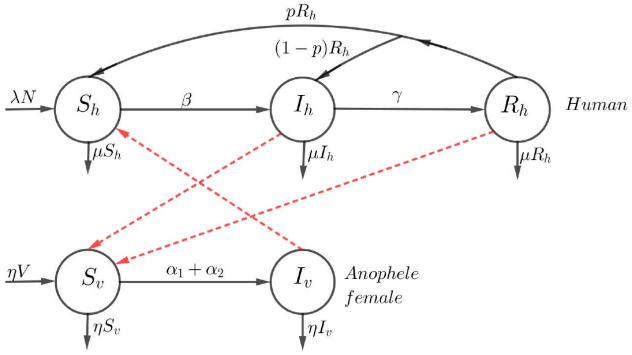


Figure 2. Malaria transmission diagram.

In this model, the differential equation system satisfies equation (3):

$$\begin{cases} dS_h(t) = \left[\lambda N - \dfrac{\beta S_h I_v}{N} + pR_h - \mu S_h\right]dt, \\ dI_h(t) = \left[\dfrac{\beta S_h I_v}{N} + (1-p)R_h - (\mu+\gamma)I_h\right]dt, \\ dR_h(t) = [\gamma I_h - (\mu+1)R_h]dt, \\ dS_v(t) = \left[\eta V - \dfrac{\alpha_1 S_v I_h}{N} + \dfrac{\alpha_2 S_v R_h}{N} - \eta S_v\right]dt, \\ dI_v(t) = \left[\dfrac{\alpha_1 S_v I_h}{N} + \dfrac{\alpha_2 S_v R_h}{N} - \eta I_v\right]dt, \end{cases} \tag{3}$$

where S_h, I_h, R_h, S_v and I_v represent the number of susceptible humans, infectious humans, recovered humans, susceptible mosquitoes and infectious mosquitoes, respectively. These types of models $(S_hI_hR_hS_h\text{-}S_vI_v)$ are also studied by authors such as [3, 4] and many others.

1.3. Model $(S_hL_hI_hR_hS_h\text{-}I_v)$

Studies have shown that the incubation period for plasmodia varies from one plasmodium to another. As this incubation period is long, it is necessary to extend the model $(S_hI_hR_hS_h\text{-}S_vI_v)$ by introducing an additional class of exposed or latent individuals L_h. At this latent stage, the individual has a probability p of being infectious and a probability $1-p$ of recovering, then becoming susceptible again. The disease progression diagram in this type of model is represented in Figure 3.

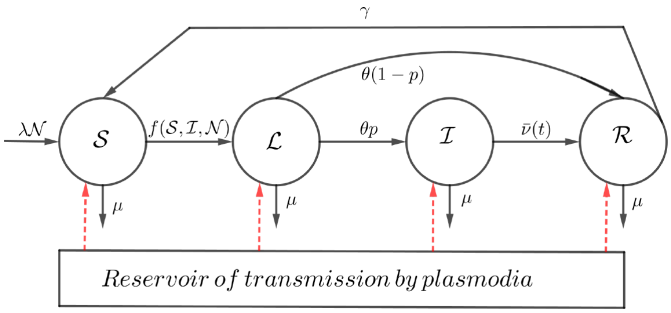


Figure 3. Plasmodia propagation diagram.

The ordinary differential equations (ODEs) governing the deterministic model $(S_hL_hI_hR_hS_h\text{-}I_v)$ are presented by the system of equations (4):

$$\begin{cases} d\mathcal{S} = [\lambda\mathcal{N} - f(\mathcal{S}, \mathcal{I}, \mathcal{N}) - \mu\mathcal{S} + \gamma\mathcal{R}]dt, & \mathcal{S}_0 > 0, \\ d\mathcal{L} = [f(\mathcal{S}, \mathcal{I}, \mathcal{N}) - (\theta + \mu)\mathcal{L}]dt, & \mathcal{L}_0 > 0, \\ d\mathcal{I} = [\theta p\mathcal{L} - (\bar{\nu}(t) + \mu)\mathcal{I}]dt, & \mathcal{I}_0 > 0, \\ d\mathcal{R} = [\bar{\nu}(t)\mathcal{I} + \theta(1-p)\mathcal{L} - (\gamma + \mu)\mathcal{R}]dt, & \mathcal{R}_0 > 0. \end{cases} \tag{4}$$

Mosquito ODEs are neglected here because susceptible humans have already received infecting bites. All the parameters and their biological interpretation are recorded in Table 1.

The dynamics of $\mathcal{N}$ is obtained by summing the equations of the system (4). As

$$\mathcal{N}(t) = \mathcal{S}(t) + \mathcal{L}(t) + \mathcal{I}(t) + \mathcal{R}(t),$$

we obtain

$$\frac{d\mathcal{N}(t)}{\mathcal{N}(t)} = \lambda - \mu \Leftrightarrow \mathcal{N}(t) = e^{(\lambda-\mu)t}.$$

- If $\lambda < \mu$, then $\mathcal{N}(t) \xrightarrow[t\to\infty]{} 0$, the population dies out.
- If $\lambda = \mu$, then the population remains constant for all t.
- If $\lambda > \mu$, then $\mathcal{N}(t) \xrightarrow[t\to\infty]{} \infty$, the population is exploding.

Table 1. Model parameters and their meanings

Parameter	Name	Unit	Meaning
$\mathcal{N}$	Population size	Number of individuals	At time t, $\mathcal{N}(t) = \mathcal{S}(t) + \mathcal{L}(t) + \mathcal{I}(t) + \mathcal{R}(t)$
λ	Birth rate	$\frac{(\text{Birth/person})}{\text{day}}$	Rate of newborns per year
μ	Death rate	$\frac{(\text{Deaths/person})}{\text{day}}$	Natural death rate of susceptible, latent, infectious and recovered individuals per year
θ	Incubation rate	day^{-1}	Rate of transition from latent to infectious state with probability p or from latent to recovered state with probability $1-p$
$f(\mathcal{S}, \mathcal{I}, \mathcal{N})$	Transmission rate (susceptible to latent)	jour^{-1}	Rate of change from susceptible to latent state
$\overline{\nu}$	Transmission rate (infectious to removed)	day^{-1}	Rate at which an infectious individual migrates into the recovered compartment
γ	Rate of immunity loss (recovered to susceptible)	day^{-1}	The rate at which recovered individuals lose their temporary immunity and become susceptible after infectious contact

We make the following changes to the variables in the model (4): $S = \mathcal{S}/\mathcal{N}$, $L = \mathcal{L}/\mathcal{N}$, $I = \mathcal{I}/\mathcal{N}$, and $R = \mathcal{R}/\mathcal{N}$ so that $S + L + I + R = 1$. Then we work with proportional incidences. The system of equations (4) then takes the form of the following relationship (5):

$$\begin{cases} dS = \left[\lambda(1 - S) - \dfrac{f(\mathcal{S}, \mathcal{I}, \mathcal{N})}{\mathcal{N}} + \gamma R\right]dt, & S_0 > 0, \\ dL = \left[\dfrac{f(\mathcal{S}, \mathcal{I}, \mathcal{N})}{\mathcal{N}} - (\theta + \lambda)L\right]dt, & L_0 > 0, \\ dI = [\theta pL - (\bar{\nu}(t) + \lambda)I]dt, & I_0 > 0, \\ dR = [\bar{\nu}(t)I + \theta(1 - p)L - (\gamma + \lambda)R]dt, & R_0 > 0. \end{cases} \tag{5}$$

The transmission function $g(S, I)$ defined by $g(S, I) = kSI$ (where k is a positive constant of proportionality) plays an important role in disease dynamics. Researchers [5, 6] have obtained a ratio-dependent non-linear incidence rate that takes the following form:

$$f(S, I) = \frac{k(I/S)^l S}{1 + \alpha(I/S)^h} = \frac{kS^{h-l+1}I^l}{S^h + \alpha I^l},$$

α being the parameter that measures the psychological or inhibitory effect. Similarly, the proportional impact function in our model is defined by

$$\frac{f(\mathcal{S}, \mathcal{I}, \mathcal{N})}{\mathcal{N}} = \bar{\beta}(t)S\frac{I_v}{\mathcal{N}}, \tag{6}$$

where I_v represents the population of infectious mosquitoes and $\bar{\beta}(t)$ is a random variable defined in Subsection 2.2. As in Ngwa and Shu's model [12], we define the force of infection from mosquitoes to humans by

$$k_v = c_v n_v \frac{I_v}{\mathcal{N}}, \tag{7}$$

where n_v represents the number of bites a mosquito makes on humans per unit of time; and c_v is the probability of the parasite entering the body of a

susceptible human bitten by an infectious mosquito I_v. By substituting $\frac{I_v}{\mathcal{N}} = \frac{k_v}{c_v n_v}$ in equation (6), the incidence function becomes

$$\frac{f(\mathcal{S}, \mathcal{I}, \mathcal{N})}{\mathcal{N}} = \frac{k_v}{c_v n_v}\overline{\beta}(t)S. \tag{8}$$

Also, equation (5) takes the following form (9):

$$\begin{cases} dS = \left[\lambda(1-S) - \dfrac{k_v}{c_v n_v}\overline{\beta}(t)S + \gamma R\right]dt, & S_0 > 0, \\ dL = \left[\dfrac{k_v}{c_v n_v}\overline{\beta}(t)S - (\theta+\lambda)L\right]dt, & L_0 > 0, \\ dI = [\theta pL - (\overline{\nu}(t)+\lambda)I]dt, & I_0 > 0, \\ dR = [\overline{\nu}(t)I + \theta(1-p)L - (\gamma+\lambda)R]dt, & R_0 > 0. \end{cases} \tag{9}$$

2. Main Results

In this section, we first determine the base reproduction rates R_0 of the models $(S_hI_hR_hS_h\text{-}S_vI_v)$ and $(S_hL_hI_hR_hS_h\text{-}I_v)$, then transform the deterministic model $(S_hL_hI_hR_hS_h\text{-}I_v)$ into a stochastic model and carry out a comparative study.

2.1. Basic reproduction rate R_0

Proposition. *The basic reproduction rates* R_0 *of the models* $(S_hI_hR_hS_h\text{-}S_vI_v)$ *of Subsection* 1.2 *and* $(S_hL_hI_hR_hS_h\text{-}I_v)$ *of Subsection* 1.3 *are given, respectively, by*

$$R_{01} = \sqrt{\frac{\beta\alpha_1(1+\mu)+\beta\alpha_2\gamma}{\eta(\gamma+\mu)(1+\mu)+\eta\gamma(-1+p)}} \quad \textit{and} \quad R_{02} = \frac{\overline{\beta}(t)\theta p}{(\theta+\lambda)(\overline{\nu}(t)+\lambda)}. \tag{10}$$

Proof. To determine the basic reproduction rate R_0, we apply the van den Driessche method [8]. The non-linear system of ordinary differential equations in the relationship (3) derived from the model in Figure 2 can be

expressed as

$$\frac{dX}{dt} = \mathcal{F}_j(X) - \mathcal{V}_j(X),$$

where $\mathcal{F}_j(X)$ represents new infections and $\mathcal{V}_j(X) = \mathcal{V}_j^+(X) - \mathcal{V}_j^-(X)$ represents the rate of individuals entering and leaving compartment j [8], respectively. The Jacobian matrices of $\mathcal{F}(X)$ and $\mathcal{V}(X)$ at equilibrium without disease E_0 are, respectively,

$$D\mathcal{F}(E_0) = F = \begin{pmatrix} 0 & 0 & \beta \\ 0 & 0 & 0 \\ \alpha_1 & \alpha_2 & 0 \end{pmatrix} \text{ and } D\mathcal{V}(E_0) = V = \begin{pmatrix} \gamma + \mu & p - 1 & 0 \\ -\gamma & \mu + 1 & 0 \\ 0 & 0 & \eta \end{pmatrix}.$$

The inverse matrix of V is

$$V^{-1} = \begin{pmatrix} \dfrac{\mu + 1}{(\gamma + \mu)(\mu + 1) + \gamma(p - 1)} & \dfrac{1 - p}{(\gamma + \mu)(\mu + 1) + \gamma(p - 1)} & 0 \\ \dfrac{\gamma}{(\gamma + \mu)(\mu + 1) + \gamma(p - 1)} & \dfrac{\gamma + \mu}{(\gamma + \mu)(\mu + 1) + \gamma(p - 1)} & 0 \\ 0 & 0 & \eta^{-1} \end{pmatrix},$$

$$FV^{-1} = \begin{pmatrix} 0 & 0 & \beta \\ 0 & 0 & 0 \\ r_1 & r_2 & 0 \end{pmatrix}$$

with

$$r_1 = \frac{\alpha_1(1 + \mu) + \alpha_2\gamma}{(\gamma + \mu)(1 + \mu) + \gamma(-1 + p)} \quad \text{and} \quad r_2 = \frac{\alpha_1(1 - p) + \alpha_2(\gamma + \mu)}{(\gamma + \mu)(1 + \mu) + \gamma(-1 + p)}.$$

The set $Sp(FV^{-1})$ of eigenvalues of the matrix FV^{-1} is given by

$$Sp(FV^{-1}) = \left\{0, \pm\sqrt{\frac{\beta\alpha_1(1 + \mu) + \beta\alpha_2\gamma}{\eta(\gamma + \mu)(1 + \mu) + \eta\gamma(-1 + p)}}\right\}.$$

The basic reproduction number R_0 is the spectral radius of the next generation matrix:

$$R_0 = \rho(FV^{-1}),$$

i.e., the basic reproduction number R_{01} of our model (3) (Subsection 1.2) is:

$$R_{01} = \sqrt{\frac{\beta\alpha_1(1+\mu)+\beta\alpha_2\gamma}{\eta(\gamma+\mu)(1+\mu)+\eta\gamma(-1+p)}}.$$

Similarly for the model (9) (Subsection 1.3), we have

$$\mathscr{F}(S, L, I) = F = \begin{pmatrix} \dfrac{\overline{\beta}(t)SI}{N} \\ 0 \end{pmatrix} \text{ and } \mathscr{V}(S, L, I) = V = \begin{pmatrix} (\overline{\nu}(t)+\lambda)L \\ \theta pL + (\overline{\nu}(t)+\lambda)I \end{pmatrix}.$$

Hence,

$$F(DFE) = \begin{pmatrix} 0 & \overline{\beta}(t) \\ 0 & 0 \end{pmatrix} \text{ and } V(DFE) = \begin{pmatrix} \overline{\nu}(t)+\lambda & 0 \\ \theta p & \overline{\nu}(t)+\lambda \end{pmatrix}.$$

Therefore,

$$FV^{-1} = \begin{pmatrix} \dfrac{\overline{\beta}(t)\theta p}{(\theta+\lambda)(\overline{\nu}(t)+\lambda)} & \dfrac{\overline{\beta}(t)}{\overline{\nu}(t)+\lambda} \\ 0 & 0 \end{pmatrix}.$$

Then the basic reproduction rate R_{02} is given by the spectral radius of FV^{-1}. □

Corollary 1. *The disease-free equilibrium of the system* (3) *is locally and asymptotically stable if* $R_{01} < 1$ *and unstable if* $R_{01} > 1$.

Corollary 2. *The malaria-free equilibrium* E_0 *of the system* (9) *is locally asymptotically stable if* $R_{02} < 1$ *and unstable if* $R_{02} > 1$.

2.2. Stochastic formulation of the $(S_hL_hI_hR_hS_h\text{-}I_v)$ model

In this subsection, we study the $(S_hL_hI_hR_hS_h\text{-}I_v)$ model which takes random effects into account. In her work, May [13] showed that all the parameters involved in a population model fluctuate randomly because the

factors controlling them are not constant. Suppose that such a system (9) is affected by random disturbances of the white noise type. In this situation, it is natural to describe its implementation by the corresponding system of stochastic differential equations. In the case of malaria, we thus assume that the transmission rates $\overline{\beta}(t)$ and $\overline{\nu}(t)$ can fluctuate around a certain value due to the continuous fluctuation of the environment. Thus the model (9) is made stochastic by the following result:

Theorem. *Consider the system of ordinary differential equations* (*ODEs*) (9) *governing the spread dynamics of Plasmodium falciparum in a given region. Then there are random effects of infection by other plasmodia rendering this ODE system into a system of stochastic differential equations* (*SDEs*). *Moreover, this SDE system exists and almost certainly admits in the domain* $]0, 1[^4$ *a unique solution* $X_t = (S_t, L_t, I_t, R_t)_{t\geq 0}$ *for any initial value* $X_0 \in]0, 1[^4$.

Proof. Let us first consider the following results:

Lemma 1. *The model* (9) *governing the dynamics of the spread of Plasmodium falciparum in a given region can be made stochastic.*

Proof. We define the transmission parameters $\overline{\beta}(t)$ and $\overline{\nu}(t)$ by:

$$\begin{cases} \overline{\beta}(t) = \beta(t) + \sigma_i \xi_i(t) 1/\sqrt{dt}, \; i = 1, 2, \\ \overline{\nu}(t) = \vartheta(t) + \sigma_i \xi_i(t) 1/\sqrt{dt}, \; i = 3, 4, \end{cases} \tag{11}$$

where σ_i, $i = \{1, ..., 4\}$ represents the noise intensities. In the following, we assume that σ_i, $i = \{1, ..., 4\}$ are constant. Let us say $\sigma_i = 0$ or 1 (the condition $\sigma_i = 0$ means that the disturbance does not exist); $\xi_i(t)$, $i = \{1, ..., 4\}$ are i.i.d white Gaussian noise terms of zero expectation such that $\xi_i(t)\sqrt{dt} = dB_i(t)$. $B_i(t)$, $i = \{1, ..., 4\}$ are independent standard Brownian motions defined on a filtered probability space

$(\mathbb{R}_+^4, \mathbb{B}(\mathbb{R}_+^4), (\mathbb{F}_t)_{t\geq 0}, \mathbb{P})$, where $\mathbb{B}(\mathbb{R}_+^4)$ denotes the Borelian tribe of $\mathbb{R}_+^4$. We assume that $\{S_0, L_0, I_0, R_0\}$ is $\mathbb{F}_{t_0}$-measurable and independent of $B_i(t) - B_i(t_0)$, $i = \{1, ..., 4\}$. By transferring the relationship (11) into the system of equations (9), we then obtain the system of stochastic differential equations (12) governing the dynamics of the transmission of plasmodia:

$$\begin{cases} dS = \left[\lambda(1-S) - \dfrac{k_v}{c_v n_v}\beta(t)S + \gamma R\right]dt - \sigma_1 \dfrac{k_v}{c_v n_v} S dB_1(t), & S_0 > 0, \\ dL = \left[\dfrac{k_v}{c_v n_v}\beta(t)S - (\theta+\lambda)L\right]dt + \sigma_2 \kappa L dB_2(t), & L_0 > 0, \\ dI = [\theta p L - (\nu(t)+\lambda)I]dt - \sigma_3 I dB_3(t), & I_0 > 0, \\ dR = [\nu(t)I + \theta(1-p)L - (\gamma+\lambda)R]dt + \sigma_4 R dB_4(t), & R_0 > 0. \end{cases} \tag{12}$$

This completes the construction of the SDE system. □

Let $\{X_1, ..., X_4\}$ be the pathological states corresponding respectively to the $\{S, L, I, R\}$ states of malaria, and let dX_i, $i = \{1, ..., 4\}$ be the small variation of the state X_i and dt a variation of time. Let $(X_t)_{t\geq 0} = (S_t, L_t, I_t, R_t)_{t\geq 0}$ be an Itô diffusion homogeneous in time, i.e., a Markov process $\{X_t(\omega)\}_{t\geq 0}$ whose state space is the probabilized space $(\mathbb{R}_+^4, \mathbb{B}(\mathbb{R}_+^4), \mathbb{P})$ satisfying a stochastic differential equation of the form

$$dX_i(t) = f(X_i(t))dt + g(X_i(t))dB_t, \quad X_i(t_0) = X_0, \quad i = \{1, ..., 4\}, \tag{13}$$

where B_t is a standard Brownian motion of dimension 4, $f : \mathbb{R}_+^4 \to \mathbb{R}_+^4$ the drift coefficient and $g : \mathbb{R}_+^4 \to \overline{R}_+^{4\times 4}$ the diffusion coefficient are fixed so that the SDE admits a single solution at all times. It is assumed that the functions f and g are locally Lipschitzian and are defined for any $i = \{1, ..., 4\}$ by

$$f(X_i(t)) = \begin{pmatrix} \lambda(1-S) - \dfrac{k_v}{c_v n_v}\beta(t)S + \gamma R \\ \dfrac{k_v}{c_v n_v}\beta(t)S - (\theta+\lambda)L \\ \theta pL - (\nu(t)+\lambda)I \\ \nu(t)I + \theta(1-p)L - (\gamma+\lambda)R \end{pmatrix},$$

$$g(X_i(t)) = \begin{pmatrix} -\sigma_1 \dfrac{k_v}{c_v n_v} S & 0 & 0 & 0 \\ 0 & \sigma_2 \kappa L & 0 & 0 \\ 0 & 0 & -\sigma_3 I & 0 \\ 0 & 0 & 0 & \sigma_4 R \end{pmatrix}$$

and

$$B = \begin{pmatrix} B_1 \\ B_2 \\ B_3 \\ B_4 \end{pmatrix}.$$

We denote by $P_t(X_0, A)$ the probability measure on the tribe generated by all the random variables $(X_t)_{t\geq 0}$ defined by

$$P_t(X_0, A) = \mathbb{P}\{X_{t_1} \in A_1, ..., X_{t_k} \in A_k\}$$

for any choice of time $0 \leq t_1 < t_2 < \cdots < t_k$, for any Borelian $A_1, A_2, ..., A_k \subset \mathbb{B}(\mathbb{R}_+^4)$ and $\forall X_0 \in \mathbb{R}_+^4$. We define the differential operator of the generator of the Itô diffusion of the relation (13) by

$$L = \sum_{i=1}^{4} f_i(X)\frac{\partial}{\partial X_i} + \frac{1}{2}\sum_{i,j=1}^{4} (gg')_{i,j}(X)\frac{\partial^2}{\partial X_i \partial X_j}, \tag{14}$$

where g' is the transpose matrix of g in $\mathbb{R}^{4\times 4}$.

Lemma 2. *The domain of L contains the set of twice continuously differentiable functions with compact support.*

Proof. Let φ be a function of class $C^2(\mathbb{R}_+^4)$, i.e., twice continuously differentiable with compact support, and apply Itô's formula to $Y_t = \varphi(X_t)$. In dimension 1, we obtain

$$Y_h = \varphi(X_0) + \int_0^h \varphi'(X_s)dX_s + \frac{1}{2}\int_0^h \varphi''(X_s)d\langle X\rangle_s$$

$$= \varphi(X_0) + \int_0^h \varphi'(X_s)f(X_s)ds + \int_0^h \varphi'(X_s)g(X_s)dB_s$$

$$+ \frac{1}{2}\int_0^h \varphi''(X_s)g(X_s)^2 ds.$$

Taking the expectation of $Y_h(\mathbb{E}(Y_h))$ evaluated at x such that $X_s = x$ for $0 < s \le t$, we obtain

$$\mathbb{E}(Y_h) = \varphi(x) + \mathbb{E}\left(\int_0^h \varphi'(X_s)f(X_s)ds + \frac{1}{2}\int_0^h \varphi''(X_s)g(X_s)^2 ds\right).$$

Hence

$$\frac{\mathbb{E}(Y_h) - \varphi(x)}{h} = \frac{1}{h}\int_0^h \mathbb{E}(\varphi'(X_s)f(X_s))ds + \frac{1}{2h}\int_0^h \mathbb{E}(\varphi''(X_s)g(X_s)^2)ds,$$

$$\lim_{h\to 0^+} \frac{\mathbb{E}(Y_h) - \varphi(x)}{h} = (L\varphi)(x) = \varphi'(x)f(x) + \frac{1}{2}\varphi''(x)g(x)^2.$$

The 4-dimensional case is treated in a similar way by applying the multidimensional Itô formula. □

The operator L defined in this way is uniformly elliptic in $\mathbb{R}_+^4$. That is, by posing $(gg')(X) = a_{ij}(X)$, there exists a positive constant c such that

$\sum_{i,\,j=1}^{4} a_{ij}(X)\varphi_i\varphi_j \geq c|\varphi|^2$ for all $X \in \mathbb{R}_+^4$ and for all non-zero vectors $\varphi = \varphi_1, ..., \varphi_4 \subset \mathbb{R}_+^4$, where $|\varphi| = \sqrt{\varphi_1^2 + \varphi_2^2 + \varphi_3^2 + \varphi_4^2}$.

Lemma 3. *The stochastic differential equation of the model* (12) *almost certainly admits a single positive local solution* $X_t = (S_t, L_t, I_t, R_t)'$ *for* $t \in [0, \tau_e]$ *and for any initial value* $X_{t_0} = X_0 \in]0, 1[^4$, *where* τ_e *denotes an extremely large finite time*.

Proof. The solution to this stochastic differential equation can take on negative values, which is not consistent with modelling disease states. To do this, we pose:

$$Y_t = \ln(X_t), \text{ with } Y_t = (u_t, v_t, w_t, z_t)' \text{ and } X_t = (S_t, L_t, I_t, R_t)'.$$

Then, *using Itô*'s *formula*,

$$\begin{cases} du = \left(\lambda e^{-u} - \lambda - \dfrac{k_v}{c_v n_v}\beta(t) + e^{q-u} - \dfrac{\sigma_1^2}{2}\dfrac{k_v^2}{c_v^2 n_v^2}\beta(t)\right)dt \\ \qquad\quad - \sigma_1 \dfrac{k_v}{c_v n_v}\beta(t)dB_1(t), \\ dv = \left(\dfrac{k_v}{c_v n_v}\beta(t)\beta(t)\dfrac{u}{v} - \theta - \lambda - \dfrac{\sigma_2^2}{2}\kappa^2\right)dt + \sigma_2\kappa dB_2(t), \\ dw = \left(\theta p\dfrac{v}{w} - \nu(t) - \lambda - \dfrac{\sigma_3^2}{2}\right)dt - \sigma_3 dB_3(t), \\ dz = \left(\nu(t)\dfrac{w}{z} + \theta(1-p)\dfrac{v}{z} + \gamma + \lambda - \dfrac{\sigma_4^2}{2}\right)dt + \sigma_4 dB_4(t) \end{cases} \tag{15}$$

at $t \geq 0$ with the initial value $Y_0 = \ln(X_0)$. According to Mao [14], we see that the coefficients of the model (15) are locally Lipschitzian, i.e., there is a unique local solution Y_t in $[0, \tau_e]$. Therefore, for any initial values S_0, L_0,

I_0 and R_0, the unique positive local solutions of the model (15) are given by $S_t = e^{u(t)}$, $L_t = e^{v(t)}$, $I_t = e^{w(t)}$ and $R_t = e^{z(t)}$. □

Lemma 4. *The solution of the stochastic differential equation system* (12) *is global in the sense that* $\tau_e = \infty$.

Proof. For each integer $n > n_0$, we define the stopping time as follows:

$$\tau_n = \inf\left\{t \in [0, \tau_e] : X(t) \in \left]\frac{1}{n}, n\right[^c\right\} \tag{16}$$

such that τ_n is increasing when n tends towards ∞ and $\inf \varnothing = \infty$, where $\varnothing$ denotes the empty set:

$$\tau_\infty = \limsup_{n\to+\infty} \tau_n \leq \tau_e \text{ a.s.}$$

We want to show that $\tau_\infty = \infty$ a.s. Let us proceed by absurdity, i.e.,

$$\exists T > 0 \text{ and } \varepsilon \in \left]0, 1\right[\text{ such that } \mathbb{P}\{\tau_\infty \leq T\} > \varepsilon.$$

In other words, there exists an integer $n_1 \geq n_0$ such that $\Omega_n = \mathbb{P}\{\tau_n \leq T\} \geq \varepsilon$, for all $n \geq n_1$.

Consider the positive function V of class C^2 defined by

$$V : \mathbb{R}_+^4 \to \mathbb{R}_+$$

$$(S, L, I, R) \mapsto (S - \ln S) + (L - \ln L) + (I - \ln I) + (R - \ln R).$$

Applying Itô's lemma to the function V, we obtain:

$$dV = LVdt - \sigma_1 \frac{k_v}{c_v n_v}\beta(t)(S-1)dB_1(t) + \sigma_2\kappa(L-1)dB_2(t)$$

$$+ \sigma_3(I-1)dB_3(t) + \sigma_4(R-1)dB_4(t),$$

where

$$
\begin{aligned}
LV &= \left(1-\frac{1}{S}\right)\left(\lambda(1-S)-\frac{k_v}{c_v n_v}\beta(t)S+\gamma R\right)\\
&\quad+\left(1-\frac{1}{L}\right)\left(\frac{k_v}{c_v n_v}\beta(t)S-(\theta+\lambda)L\right)+\left(1-\frac{1}{I}\right)(\theta pL-(\nu(t)+\lambda)I)\\
&\quad+\left(1-\frac{1}{R}\right)(\nu(t)I+\theta(1-p)L-(\gamma+\lambda)R)\\
&\quad+\frac{\frac{k_v^2}{c_v^2 n_v^2}\beta^2(t)\sigma_1^2+\kappa^2\sigma_2^2+\sigma_3^2+\sigma_4^2}{2}\\
&= 4\lambda+\theta+\frac{k_v}{c_v n_v}\beta(t)+\nu(t)+\gamma-\frac{\lambda}{S}-\frac{k_v}{c_v n_v}\beta(t)\frac{S}{L}-\gamma\frac{R}{S}-\theta p\frac{L}{I}\\
&\quad-\nu(t)\frac{I}{R}-\theta(1-p)\frac{L}{R}+\frac{\frac{k_v^2}{c_v^2 n_v^2}\beta^2(t)\sigma_1^2+\kappa^2\sigma_2^2+\sigma_3^2+\sigma_4^2}{2}\\
&< 4\lambda+\theta+\frac{k_v}{c_v n_v}\beta(t)+\nu(t)+\gamma+\frac{\frac{k_v^2}{c_v^2 n_v^2}\beta^2(t)\sigma_1^2+\kappa\sigma_2^2+\sigma_3^2+\sigma_4^2}{2}
\end{aligned}
$$

for all $t \geq 0$, $\beta(t), \nu(t) \in \,]0, 1[$. Therefore, there exists $M > 0$ independent of S, L, I and R such that $LV(S, L, I, R) \leq M$. Consequently,

$$
\begin{aligned}
dV(S, L, I, R) \leq Mdt &- \sigma_1\frac{k_v}{c_v n_v}\beta(t)(S-1)dB_1(t)\\
&+\sigma_2\kappa(L-1)dB_2(t)+\sigma_3(I-1)dB_3(t)\\
&+\sigma_4(R-1)dB_4(t).
\end{aligned}
$$

By integrating the two members of the above inequality from 0 to $\tau_n \wedge T$, where $\tau_n \wedge T = \min(\tau_n, T)$ and taking the expectations, we obtain

the following:

$$\begin{aligned}
&\int_0^{\tau_n\wedge T} dV(S(s),\, L(s),\, I(s),\, R(s))\\
&\leq \int_0^{\tau_n\wedge T} Mdt - \int_0^{\tau_n\wedge T} \sigma_1 \frac{k_v}{c_v n_v}\beta(t)(S(s)-1)dB_1(t)\\
&\quad + \int_0^{\tau_n\wedge T} \sigma_2\kappa(L(s)-1)dB_2(t) + \int_0^{\tau_n\wedge T} \sigma_3(I(s)-1)dB_3(t)\\
&\quad + \int_0^{\tau_n\wedge T} \sigma_4(R(s)-1)dB_4(t),\\
&\mathbb{E}[V(S(\tau_n\wedge T),\, L(\tau_n\wedge T),\, I(\tau_n\wedge T),\, R(\tau_n\wedge T))]\\
&\leq V(S(0),\, L(0),\, I(0),\, R(0)) + MT. \qquad (17)
\end{aligned}$$

We know that $\Omega_n = \mathbb{P}\{\tau_n \leq T\} \geq \varepsilon$, for any $n \geq n_1$ and according to the relationship (16), for any $\omega \in \Omega_n$, there exists a certain k such that $X_k(\tau_n, \omega)$ is equal to either n or $\frac{1}{n}$ for $k = 1, 2, 3, 4$. Hence,

$$V(S(\tau_n, \omega),\, L(\tau_n, \omega),\, I(\tau_n, \omega),\, R(\tau_n, \omega)) \geq \min\left\{(n - \ln n), \left(\frac{1}{n}, \ln\frac{1}{n}\right)\right\}.$$

It follows from the relationship (17) that

$$\begin{aligned}
&V(S(0),\, L(0),\, I(0),\, R(0)) + MT\\
&\geq \mathbb{E}[\chi_{\Omega_n(\omega)} V(S(\tau_n),\, L(\tau_n),\, I(\tau_n),\, R(\tau_n))]\\
&\geq \varepsilon \min\left\{(n - \ln n), \left(\frac{1}{n}, \ln\frac{1}{n}\right)\right\},
\end{aligned}$$

where $\chi_{\Omega_n(\omega)}$ is the indicator function of Ω_n. Since $n \to \infty$, we get

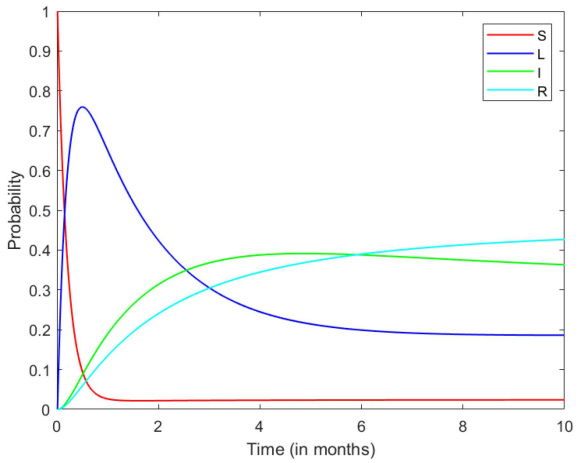
$$\infty > V(S(0),\, L(0),\, I(0),\, R(0)) + MT = \infty \text{ a.s.},$$

which is a contradiction, because we must have $\tau_\infty = \infty$. Consequently, the solution of the stochastic differential equation system (12) is global.

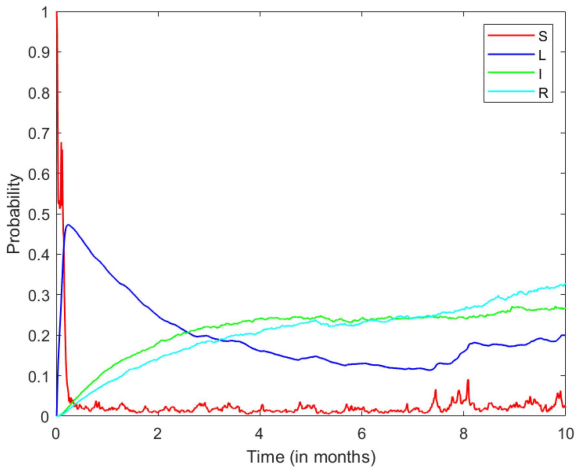
With the demonstration of these lemmas, the proof of the theorem is complete. □

2.3. Numerical simulations and interpretations

In this subsection, we make a comparative study of the trajectories of solutions obtained by simulation in MATLAB R2022b.



(a) Deterministic case



(b) Stochastic case

Figure 4. Simulation of ODE and SDE solution trajectories.

The introduction of Brownian motion induced a disturbance in the latent individuals. This could be due to the variability in the latency period of the different plasmodia. The spike of latent individuals in Figure 4(a) indicates that 80% of these individuals are almost infected by the same parasite. Similar variability between infected and recovered individuals can be observed in Figures 4(a) and 4(b). The differences in probabilities observed between the deterministic and stochastic cases mean that Figure 4(a) reflects the image of an area where the only virulent plasmodium is present, whereas Figure 4(b) illustrates the image of an area where several parasite strains are present, making malaria diagnosis complex.

3. Conclusion and Perspectives

In this paper, we have proposed a stochastic approach to model the random propagation of plasmodia through stochastic differential equations. Presenting prototypes of existing models for the development of our model, we have shown the existence and uniqueness of the differential equations governing our model. Finally, we simulated the trajectories of the solutions.

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