Hindawi
Journal of Applied Mathematics
Volume 2024, Article ID 7555042, 16 pages
https://doi.org/10.1155/2024/7555042

Research Article

On a Stochastic Approach to Extensions of the Susceptible-Infected-Susceptible (SIS) Model Applied to Malaria

Abdoul Karim Drabo,[1] **Frédéric Bere**,[2] **and S. P. Clovis Nitiema**[1,3]

[1]*Laboratoire de sciences et technologies, Université Thomas SANKARA, 12, BP, 417 Ouagadougou, Burkina Faso*
[2]*Département de mathématiques, École Normale Supérieure, 01 BP 1757, Ouagadougou 01, Burkina Faso*
[3]*Département de mathématiques de décision, Université Thomas SANKARA, 12, BP, 417 Ouagadougou, Burkina Faso*

Correspondence should be addressed to Frédéric Bere; berefrederic@gmail.com

Received 12 August 2022; Revised 13 February 2024; Accepted 23 February 2024; Published 30 April 2024

Academic Editor: Oluwole D. Makinde

This work presents a stochastic model of malaria spread. We first calculated the basic reproduction number R_0 of the models $(S_hI_hR_hS_h\text{-}S_vI_v)$ and $(S_hL_hI_hR_hS_h\text{-}S_vL_vI_v)$ in order to show that the malaria-free equilibrium is asymptotically stable; then, we used a finite Markov chain model to describe the interactions between the different compartments of the model $(S_eL_eI_eR_e S_e\text{-}S_aL_aI_aR_aS_a\text{-}S_vI_v)$. We carried out numerical simulations of our results for two types of transmission zones: a zone with low malaria transmission and an endemic zone. Through these simulations, we first determined the invariant stationary distribution π^* of the model, and then, we found that the use of the indoor residual spraying (IRS) method by regular application of insecticides is more effective for the elimination of malaria than the use of long-acting impregnated mosquito nets (LLINs).

1. Introduction

Mathematical models of the spread of malaria date back to the beginning of the 20th century by Ross [1]. Several mathematical models have been developed to study the dynamics of malaria spread. We can cite the models $(S_hI_hS_h\text{-}S_vI_vS_v)$ of Ross, the models $(S_hI_hR_hS_h\text{-}S_vI_v)$ in [2–4], and the models $(S_hE_hI_hR_hS_h)$ in [5, 6]. The mathematical theory of the spread of malaria was solidified by Reiner et al. [7] in the 1960s. In 2018, Mbogo et al. [2] use the Galton-Watson branching processes to propose an extension of the model formulated by [4]. There are also results in the following works [8–13] that could well explain the literature. In this work, we first present in Section 2 some preliminary results, necessary for the conduct of our work by successively studying the models $(S_hI_hS_h\text{-}S_vI_vS_v)$, $(S_hI_hR_hS_h\text{-}S_vI_v)$, and $(S_hL_hI_hR_hS_h\text{-}S_vL_vI_v)$. Then, we present an extension of the Markovian SIS model in a hypoendemic and endemic area in Sections 3.2 and 3.3, respectively. And finally, Section 4 is devoted to the work of numerical simulations of our results.

2. Mathematical Preliminaries and Notations

2.1. Ross's $(S_hI_hS_h\text{-}S_vI_vS_v)$ Model. In 1911, Sir Ronald Ross proposed a model which took into account both anopheline and human populations. This model is certainly the starting point for vector-host models. Ross divided hosts (humans) and vectors (anopheles) into two classes, susceptible and infected, respectively. Let S_h be the population of susceptible humans and I_h the population of infected humans. Ross assumes that there is no latency period and that, consequently, an infected person is automatically an infectious person. Similarly, he refers to the population of susceptible anopheles as S_v and the population of infected anopheles as I_v. In his model, Ross assumes that both the human and Anopheles populations are constant and that one mosquito bites "a" humans per unit of time where "a" is constant

[14]. The malaria propagation graph in Ross's $(S_hI_hS_h\text{-}S_vI_vS_v)$ model is shown in Figure 1.

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

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

Assuming that the human and Anopheles populations are constant, Ross used two ordinary differential equations (2) to model the evolution of the fraction of individuals in the classes of infected (I_h, I_v).

$$\begin{cases} \dfrac{dI_h}{dt} = b_1 a I_v \dfrac{S_h}{H} - (\gamma_h + \mu_h) I_h, \\ \dfrac{dI_v}{dt} = b_2 a (V - I_v) \dfrac{I_h}{H} - (\gamma_v + \mu_v) I_v. \end{cases} \tag{2}$$

According to Ross, eradicating malaria requires reducing the number of infectious mosquitoes below a certain threshold. He determines the basic reproduction number R_0 per

$$R_0 = mab_1 \frac{1}{\gamma} a \frac{1}{\mu} b_2 \tag{3}$$

and formulates the following corollary [15, 16].

Corollary 1.

(1) If $R_0 \le 1$, then the disease completely disappears from the population after a certain time

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

2.2. 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 $1 - p$. So to Ross's $(S_hI_hS_h\text{-}S_vI_vS_v)$ model, we add the R recovered compartment. These types of model are also solved in [17, 18]. Figure 2 illustrates the scheme of disease progression.

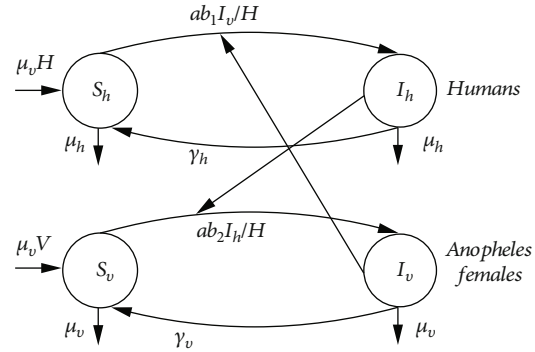

Figure 1: The final graph of disease transmission.

In this model, the differential equation system satisfies the following equation:

$$\begin{cases} dS_h(t) = \left[\lambda N - \dfrac{\beta S_h I_v}{N} + pR_h - \mu S_h\right] dt, \\ dI_h(t) = \left[\dfrac{\beta S_h I_v}{N} + (1-p)R_h - (\mu + \gamma) I_h\right] dt, \\ dR_h(t) = [\gamma I_h - (\mu + 1) R_h] dt, \\ dS_v(t) = \left[\eta V - \dfrac{\alpha_1 S_v I_h}{N} + \dfrac{\alpha_2 S_v R_h}{N} - \eta S_v\right] dt, \\ dI_v(t) = \left[\dfrac{\alpha_1 S_v I_h}{N} + \dfrac{\alpha_2 S_v R_h}{N} - \eta I_v\right] dt, \end{cases} \tag{4}$$

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 [2–4].

2.3. Model $(S_hL_hI_hR_hS_h\text{-}S_vL_vI_v)$. According to the literature, the average delay between exposure to parasitised blood and the first clinical signs of infection is 4 to 17 days for P. falciparum. There is therefore an additional class of exposed individuals (E) or latent individuals (L) who are not yet infectious. It is therefore necessary to model the dynamics of the spread of malaria using the $(S_hL_hI_hR_hS_h\text{-}S_vL_vI_v)$ model, which would be an extension of the $(S_hI_hS_h\text{-}S_vI_vS_v)$ and $(S_hI_hR_hS_h\text{-}S_vI_v)$ models previously studied. The disease progression diagram in this type of model can be represented in Figure 3.

The ordinary differential equations (ODEs) governing the deterministic SLIRS model are presented by the system of

$$\begin{cases} dS_h = \left(\lambda N - \beta \dfrac{S_h I_v}{N} - \mu S_h + \gamma R_h\right) dt, \\ dL_h = \left(\beta \dfrac{S_h I_v}{N} - (k + \mu) L_h\right) dt, \\ dI_h = (kpL_h - (\alpha + \mu) I_h) dt, \\ dR_h = (\alpha I_h - k(1-p) L_h - (\gamma + \mu) R_h) dt. \end{cases} \tag{5}$$

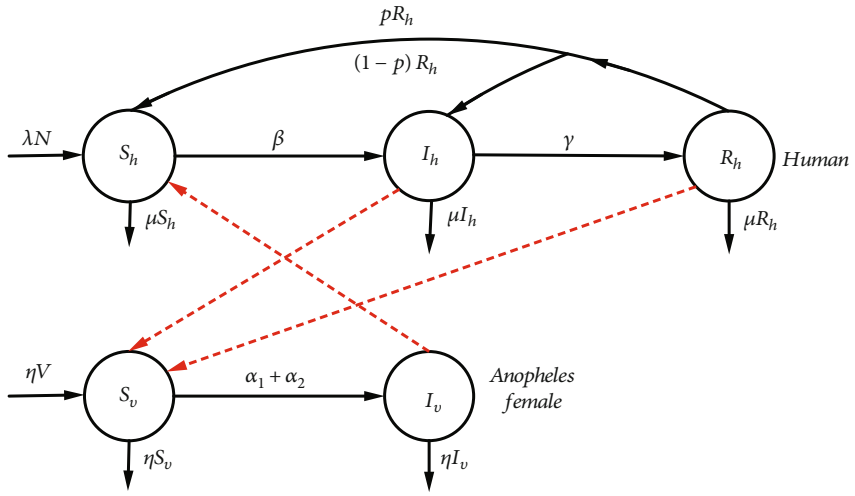

Figure 2: Malaria transmission diagram.

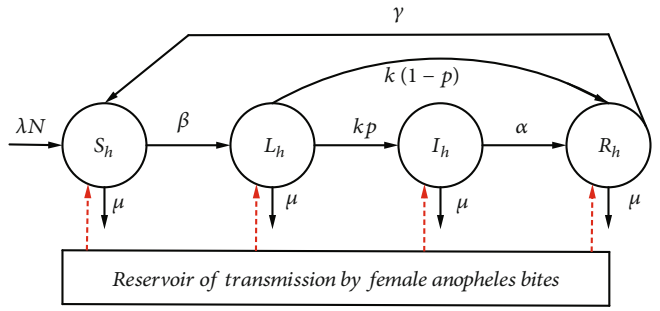

Figure 3: Malaria progression graph.

Mosquito ODEs are neglected for simplicity. All parameters and their biological interpretation are recorded in Table 1.

Once we know the triplet (S, L, I), we can work with the reduced system (6) and then deduce R because $N = S + L + I + R$.

$$\begin{cases} dS = \left(\lambda N - \beta\dfrac{SI}{N} - \mu S + \gamma R\right)dt, \\ dL = \left(\beta\dfrac{SI}{N} - (k+\mu)L\right)dt, \\ dI = (kpL - (\alpha+\mu)I)dt. \end{cases} \tag{6}$$

The disease-free equilibrium (DFE) of this model is therefore $(N, 0, 0)$. Authors such as [17, 19, 20] have also studied these types of models.

3. Main Results

3.1. Basic Reproduction Rate R_0

Theorem 2. *The basic reproduction rates R_0 of the models $(S_hI_hR_hS_h\text{-}S_vI_v)$ in Section 2.2 and $(S_hL_hI_hR_hS_h\text{-}S_vL_vI_v)$ in Section 2.3 are given by*

Table 1: Model parameters and their meanings.

Parameter	Biological interpretation
N	Size of the human population $(N = S + L + I + R)$
λ	Birth rate
μ	Death rate
k	Incubation rate
p	Probability of transition from latent to infectious state
$1-p$	Probability of transition from latent to recovered state
β	Transmission rate (susceptible to latent)
α	Transmission rate (infectious to removed)
γ	Immunity loss rate (removed to susceptible)

$$\begin{aligned} R_{01} &= \sqrt{\frac{\beta\alpha_1(1+\mu) + \beta\alpha_2\gamma}{\eta(\gamma+\mu)(1+\mu) + \eta\gamma(-1+p)}}, \\ R_{02} &= \frac{\beta kp}{(k+\mu)(\alpha+\mu)}. \end{aligned} \tag{7}$$

Proof. To determine the basic reproduction rate R_0, we apply the Van den Driessche method [15]. The nonlinear ordinary differential equation system (4) integrating the compartments of the model in Figure 2 can be expressed as

$$\frac{dX}{dt} = \mathscr{F}_j(X) - \mathscr{V}_j(X), \tag{8}$$

where $\mathscr{F}_j(X)$ represents new infections and $\mathscr{V}_j(X) = \mathscr{V}_j^+(X) - \mathscr{V}_j^-(X)$ represents the rate of individuals entering and exiting the j compartment, respectively [15]. The Jacobian matrices of $\mathscr{F}(X)$ and $\mathscr{V}(X)$ at equilibrium without disease E_0 are

$$\begin{aligned} D\mathscr{F}(E_0) = F &= \begin{pmatrix} 0 & 0 & \beta \\ 0 & 0 & 0 \\ \alpha_1 & \alpha_2 & 0 \end{pmatrix}, \\ D\mathscr{V}(E_0) = V &= \begin{pmatrix} \gamma+\mu & p-1 & 0 \\ -\gamma & \mu+1 & 0 \\ 0 & 0 & \eta \end{pmatrix}. \end{aligned} \tag{9}$$

The inverse matrix of V is

$$\begin{aligned} V^{-1} &= \begin{pmatrix} \dfrac{\mu+1}{(\gamma+\mu)(\mu+1)+\gamma(p-1)} & \dfrac{1-p}{(\gamma+\mu)(\mu+1)+\gamma(p-1)} & 0 \\ \dfrac{\gamma}{(\gamma+\mu)(\mu+1)+\gamma(p-1)} & \dfrac{\gamma+\mu}{(\gamma+\mu)(\mu+1)+\gamma(p-1)} & 0 \\ 0 & 0 & \eta^{-1} \end{pmatrix}, \\ FV^{-1} &= \begin{pmatrix} 0 & 0 & \beta \\ 0 & 0 & 0 \\ r_1 & r_2 & 0 \end{pmatrix}, \end{aligned} \tag{10}$$

with

$$\begin{aligned} r_1 &= \frac{\alpha_1(1+\mu)+\alpha_2\gamma}{(\gamma+\mu)(1+\mu)+\gamma(-1+p)}, \\ r_2 &= \frac{\alpha_1(1-p)+\alpha_2(\gamma+\mu)}{(\gamma+\mu)(1+\mu)+\gamma(-1+p)}. \end{aligned} \tag{11}$$

The set of eigenvalues of FV^{-1} is denoted $\mathrm{Sp}(FV^{-1})$ and is equal

$$\mathrm{Sp}\left(FV^{-1}\right) = \left\{0, \pm\sqrt{\frac{\beta\alpha_1(1+\mu)+\beta\alpha_2\gamma}{\eta(\gamma+\mu)(1+\mu)+\eta\gamma(-1+p)}}\right\}. \tag{12}$$

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

$$R_0 = \rho\left(FV^{-1}\right). \tag{13}$$

The basic reproduction number R_{01} of our model (4) in Section 2.2 is

$$R_{01} = \sqrt{\frac{\beta\alpha_1(1+\mu)+\beta\alpha_2\gamma}{\eta(\gamma+\mu)(1+\mu)+\eta\gamma(-1+p)}}. \tag{14}$$

Similarly for the model (6) in Section 2.3, we have

$$\begin{aligned} \mathscr{F}(S,L,I) = F &= \begin{pmatrix} \dfrac{\beta SI}{N} \\ 0 \end{pmatrix}, \\ \mathscr{V}(S,L,I) = V &= \begin{pmatrix} (k+\mu)L \\ kpL + (\alpha+\mu)I \end{pmatrix}. \end{aligned} \tag{15}$$

Hence,

$$\begin{aligned} F(\mathrm{DFE}) &= \begin{pmatrix} 0 & \beta \\ 0 & 0 \end{pmatrix}, \\ V(\mathrm{DFE}) &= \begin{pmatrix} k+\mu & 0 \\ kp & \alpha+\mu \end{pmatrix}. \end{aligned} \tag{16}$$

Therefore,

$$FV^{-1} = \begin{pmatrix} \dfrac{\beta kp}{(k+\mu)(\alpha+\mu)} & \dfrac{\beta}{\alpha+\mu} \\ 0 & 0 \end{pmatrix}. \tag{17}$$

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

Corollary 3. *The disease-free equilibrium of the system (6) is locally and asymptotically stable if $R_{01} < 1$ and unstable if $R_{01} > 1$.*

Corollary 4. *The malaria-free equilibrium E_0 of the system (4) is locally asymptotically stable if $R_{02} < 1$ and unstable if $R_{02} > 1$.*

3.2. Extension of the Markov SIS Model in a Hypoendemic Zone. In this section, we develop a stochastic model for malaria transmission by considering two types of host ("nonimmune" and "semi-immune") in the human population. The "nonimmune" group comprises human individuals who have never acquired any immunity to malaria. It is assumed that these hosts are vulnerable because they can suffer and/or die from malaria. "Semi-immune" people are those who have acquired or lost at least some immunity to malaria in their lifetime. It is assumed that these hosts are nonvulnerable, so they cannot die from malaria but can only suffer from it. We envisage a model of the type $(S_eL_eI_eR_eS_e)$ for nonimmunes until they enter the semi-immune category and then follow a model of the type $(S_aL_aI_aR_aS_a)$, where e and a denote the index for nonimmunes and semi-immunes, respectively. For the mosquito population, we use a model of the type (S_vI_v).

3.2.1. Elaboration of the Model. We consider that it is the gametocyte form of Plasmodium in humans that transmits the infection to mosquitoes and the sporozoite form in the mosquito that transmits the infection to humans. In our model, we adopt a number of operational definitions.

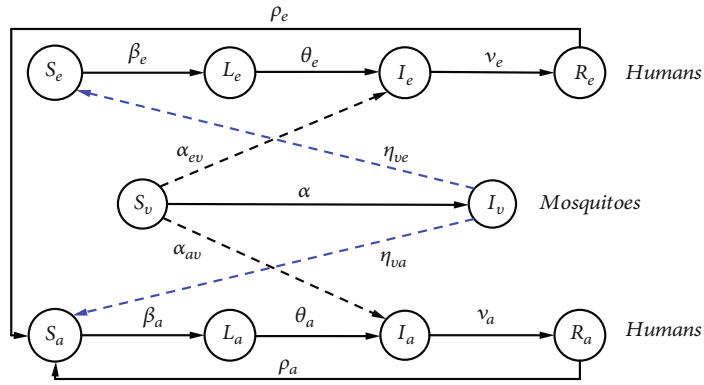

Figure 4: Diagram of malaria progression.

(1) Terminology.

(i) Susceptible (S): an individual bitten by infectious mosquitoes and capable of contracting malaria

(ii) Latent (L): individual who has contracted malaria but does not transmit it initially

(iii) Infectious (I): an individual who harbours a high level of parasites in gametocyte form. They can suffer and/or die from malaria

(iv) Immune (R): an asymptomatic carrier of the parasite. This is a state of protection against the disease, but not against the parasite; it is a healing stage

A human being is said to be ill with malaria if he or she is either in a latent or infectious state. In order to write the model, the nonimmune human host types are subdivided into four (4) compartments: susceptible (S_e), latent (L_e), infectious (I_e), and immune (R_e). Semi-immune human host types are also subdivided into four (4) compartments: susceptible (S_a), latent (L_a), infectious (I_a), and immune (R_a). The mosquito population is made up of susceptible (S_v) and infectious (I_v) mosquitoes. We are studying two heterogeneous populations made up of several pathological classes, in which individuals can move from one class to another. There are ten different disease states. The pattern of disease progression in this model is illustrated in Figure 4.

The solid arrows indicate the direction of propagation of Plasmodium, thus creating states of health in the subjects.

The red dotted arrows indicating the direction of infection from an infectious mosquito (I_v) to a nonimmune susceptible human individual (S_e) or a semi-immune susceptible human individual.

The black dotted arrows indicate the direction of infection of a susceptible mosquito (S_v) on an infectious nonimmune human (I_e) or an infectious nonimmune human (I_a).

3.2.2. Model Assumptions. A1: the probabilities $\eta_{ve}, \eta_{va}, \alpha_{ev}, \alpha_{av}$, and α are in the interval $[0, 1[$.

A2: the parameters $\beta_e, \theta_e, \eta_e, \rho_e, \beta_a, \theta_a, \eta_a$, and ρ_a are assumed to be strictly positive in the interval $]0, 1[$ and represent the probabilities of moving from a subfund i to a subfund j.

A3: these passage probabilities and parameters designate the conditional probabilities $\mathbb{P}(X_1 = \text{compartment } j | X_0 = \text{compartment } i)$ satisfying the Markov property. These conditional probabilities linking two consecutive pathological states are between $]0, 1[$ if the transition has a biological meaning and 0 otherwise.

3.2.3. Interaction between Humans and Mosquitoes. Initially, infectious mosquitoes (I_v) bite susceptible humans (S_e) with a η_{ve} probability. The latter may remain susceptible (S_e) with probability r for some time or become latent (L_e) with probability β_e. Individuals can remain latent for a given time with probability q or become infectious (I_e) with θ_e as the transition probability. In this infectious state (I_e), humans can die with a probability μ or become immune with a probability equal to ν_e, or even remain infectious with a probability p for an interval of time. Having completed the 1st cycle, the immune (R_e) becomes susceptible (S_a) to the ρ_e immunity rate, and through the intermediary of the infectious mosquito (I_v), the cycle resumes as shown in Figure 4. At stage (R_a), having acquired a certain immunity, the individual re-enters the susceptible class (S_a) at the rate ρ_a. The susceptible mosquitoes (S_v) inoculate the parasite from a blood meal on the infectious (I_e) and I_a with probabilities α_{ev} and α_{av}, respectively; then, they become infectious (I_v) with probability α.

Tables 2–4 summarise the parameters that will be described in the model.

Proposition 5. *Let $(\Omega, \mathcal{F}, \mathbb{P})$ be a probability space and $X = \{X_n, n \geq 0\}$ the process of the mode of transmission of plasmodia with values in the discrete state space $\mathbb{E}$. Then, the process X defined on $(\Omega, \mathcal{F}, \mathbb{P})$ with values in $\mathbb{E}$ is an irreducible and aperiodic homogeneous Markov chain with initial distribution $\pi_0 = (\pi_0^1, \cdots, \pi_0^{10})$ and transition matrix H, where π_0^i is the probability that the process X is in state i at time 0. And this Markov chain has a stationary distribution π^* such that $\pi^* = \lim_{k \longrightarrow \infty} \pi_0 H^k$.*

Proof. We consider that the ten compartments $S_e, L_e, I_e, R_e, S_a, L_a, I_a, R_a, S_v$, and I_v in Figure 4 represent the ten states of a

Table 2: Plasmodium transmission parameters for the nonimmune host type.

Param.	Meaning	Unity
β_e	Rate of transition from the susceptible state S_e to the latent state L_e.	Without Dim.
θ_e	Rate of transition from latent state L_e to infectious state I_e.	Without Dim.
ν_e	Rate of transition from infectious state I_e to immune state R_e.	Without Dim.

Table 3: Contact parameters between humans and mosquitoes and their dimensions.

Param.	Meaning	Unity
n_a	Number of times a mosquito could bite a human being per unit of time, if human beings were freely available	Time^{-}1
η_{ev}	Probability of transmission of an infection from an infectious mosquito (I_v) to a susceptible nonimmune human (S_e)	Without Dim.
η_{va}	Probability of transmission of an infection from an infectious mosquito (I_v) to a susceptible nonimmune human (S_a)	Without Dim.
α_{ev}	Probability of transmission of infection from an infectious human being (I_e) to a susceptible mosquito (S_v), given that there has been contact between the two	Without Dim.
α_{av}	Probability of transmission of infection from an infectious human being (I_a) to a susceptible mosquito (S_a), given that there has been contact between the two	Without Dim.

Table 4: Plasmodium transmission parameters for the semi-immune host type.

Param.	Meaning	Unity
β_a	Rate of transition from the susceptible state S_a to the latent state L_a	Without Dim.
θ_a	Rate of transition from latent state L_a to infectious state I_a	Without Dim.
ν_a	Rate of transition from infectious state I_a to immune state R_a	Without Dim.
ρ_e	Rate of transition from the immune state R_e to the susceptible state S_a	Without Dim.
ρ_a	Rate of transition from the immune state R_a to the susceptible state S_a	Without Dim.

Markov chain. These ten states are denoted $\{i_1, i_2, i_3, i_4, i_5, i_6, i_7, i_8, i_9, i_{10}\}$, respectively. Let $\mathbb{E}$ be the set of ten states of the Markov chain. Let $X = \{X_n, n \geq 0\}$ be the process of the mode of transmission and evolution of Plasmodium. Let p_{ij}^n denote the probability that the process is in state j at time $n+1$ knowing that it is in state i at time n. Furthermore, the probability p_{ij}^n is assumed to be independent of n. For all $(i, j) \in \mathbb{E}^2$, p_{ij} is called the transition probability from state i to state j. The probabilistic transition graph of the Markov chain model is given in Figure 5, where the $x_i, i \in \{1, \cdots, 23\}$ correspond to the different parameters of the model and represent the transition probabilities of the chain. The chain is irreducible because its representative graph (Figure 5) is strongly connected. □

Under hypothesis A3, the p_{ij} are grouped together in a matrix H defined by the relation

$$H = \begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \end{array} \begin{array}{c} \begin{array}{cccccccccc} S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \end{array} \\ \begin{pmatrix} r_e & \beta_e & \beta_{e1} & \beta_{e2} & 0 & 0 & \rho_e & \beta_{e3} & \beta_{e4} & \beta_{e5} \\ 0 & q_e & \theta_e & \theta_{e1} & 0 & 0 & \theta_{e2} & \theta_{e3} & \theta_{e4} & \theta_{e5} \\ 0 & 0 & p_e & \nu_e & 0 & 0 & \nu_{e1} & \nu_{e2} & \nu_{e3} & \nu_{e4} \\ 0 & 0 & 0 & r_e & 0 & 0 & \rho_e & \rho_{e1} & \rho_{e2} & \rho_{e3} \\ 0 & 0 & 0 & 0 & \alpha' & \alpha & 0 & 0 & 0 & 0 \\ \eta_{ve} & \eta_{ve1} & \eta_{ve2} & \eta_{ve3} & 0 & p_v & \eta_{va} & \eta_{va1} & \eta_{va2} & \eta_{va3} \\ 0 & 0 & 0 & 0 & 0 & t_1 & r_a & \beta_a & \beta_{a1} & \beta_{a2} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & q_a & \theta_a & \theta_{a1} \\ 0 & 0 & 0 & 0 & t_2 & 0 & 0 & 0 & p_a & \eta_{va} \\ 0 & 0 & 0 & 0 & 0 & 0 & \rho_a & \beta_a & \theta_a & r_a \end{pmatrix} \end{array}. \quad (18)$$

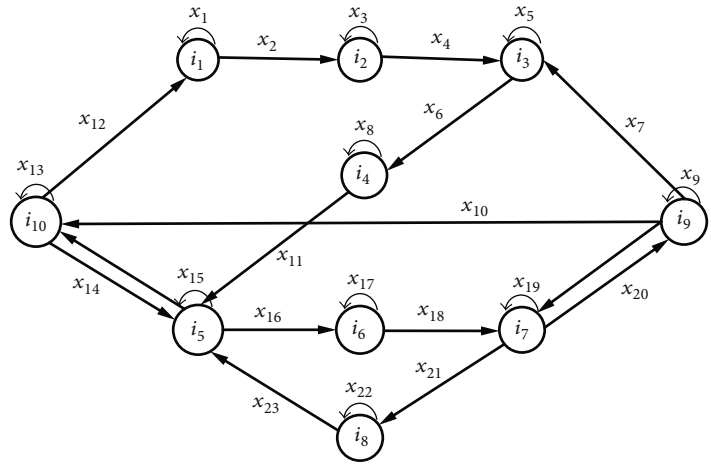

Figure 5: Transition graph of irreducible Markov chain.

The H matrix thus constructed is stochastic by extension of the SIS model.

Furthermore, during the clinical manifestations of malaria, the future state of the patient does not depend on the previous state, but rather on the current state (the system has no "memory"). All the information needed to predict the future is contained in the current state of the process. So we can say that the spread and evolution of malaria satisfy the weak Markov property (discrete time, discrete space): for any $n \geq 0$, for any sequence of states $(i_0, \cdots, i_{n-1}, i, j) \in \mathbb{E}^{n+2}$,

$$\mathbb{P}(X_{n+1} = j | X_0 = i_0, X_1 = i_1, \cdots, X_{n-1} = i_{n-1}, X_n = i) = \mathbb{P}(X_{n+1} = j | X_n = i), \tag{19}$$

as soon as $\mathbb{P}(X_0 = i_0, X_1 = i_1, \cdots, X_{n-1} = i_{n-1}, X_n = i) > 0$. This property expresses the fact that the law of X_{n+1} depends on $X_0, \cdots, X_n$ only through the value of X_n: the "present" (X_n) gives as much information about the "future" (X_{n+1}) as if we knew all the "past" $X_0, \cdots, X_n$. By hypothesis, the transition mechanism does not change over time. The weak Markov property then takes the following form:

$$\forall n \geq 0, (i_0, \cdots, i_{n-1}, i, j) \in \mathbb{E}^{n+2},$$
$$\mathbb{P}(X_{n+1} = j | X_0 = i_0, X_1 = i_1, \cdots, X_{n-1} = i_{n-1}, X_n = i) = \mathbb{P}(X_1 = j | X_0 = i), \tag{20}$$

i.e.,

$$\forall (i, j) \in \mathbb{E}^2 \; \mathbb{P}(X_{n+1} = j | X_n = i) = \mathbb{P}(X_1 = j | X_0 = i), \tag{21}$$

which expresses the homogeneity of the process in time.

3.3. Extending the SIS Markov Model to an Endemic Area. Children under 5 years old are the most vulnerable to malaria in endemic areas [21–24]. They have not yet developed their own immunity and are therefore considered nonimmune. We do not take into account the age of the individual but rather his or her immunological status, because according to studies [25, 26], children and adults have the same probability of being infected by malaria. It is assumed that there has been one case of death in the infectious nonimmune human compartments. Then, the Plasmodium propagation diagram takes the form of Figure 6.

The solid arrows indicate the direction of propagation of Plasmodium, thus creating states of health in the subjects.

The blue dotted arrows indicate the direction of infection from an infectious mosquito (I_v) to a nonimmune susceptible human individual (S_e) or a semi-immune susceptible human individual.

The black dotted arrows indicate the direction of infection of a susceptible mosquito (S_v) on an infectious nonimmune human (I_e) or an infectious nonimmune human (I_a).

The red dotted arrows indicate a case of death:

(i) of infectious nonimmune human (I_e) thus entering compartment D, where μ represents the probability of dying from malaria knowing that the nonimmune human is infectious

(ii) mosquitoes by intradomiciliary spraying

The green dotted arrow indicates the use of long-lasting impregnated mosquito nets.

3.3.1. Model Assumptions. We supplement the list of assumptions in the previous Section 3.2 with two more:

A4: infectious mosquitoes I_v are eliminated by intradomiciliary insecticide spraying with a probability p, $(p \in]0, 1[)$.

A5: humans use long-acting impregnated mosquito nets with a probability of $1 - p$, and for reasons of model simplification, environmental factors that could have more or less significant effects are neglected.

Proposition 6. *Let $(\Omega, \mathcal{F}, \mathbb{P})$ be a probability space and $X = \{X_n, n \geq 0\}$ the process of the mode of transmission of plasmodia with values in the discrete state space $\mathbb{E}$. Then, the process X defined on $(\Omega, \mathcal{F}, \mathbb{P})$ with values in E is a homogeneous absorbing Markov chain with transition matrix P.*

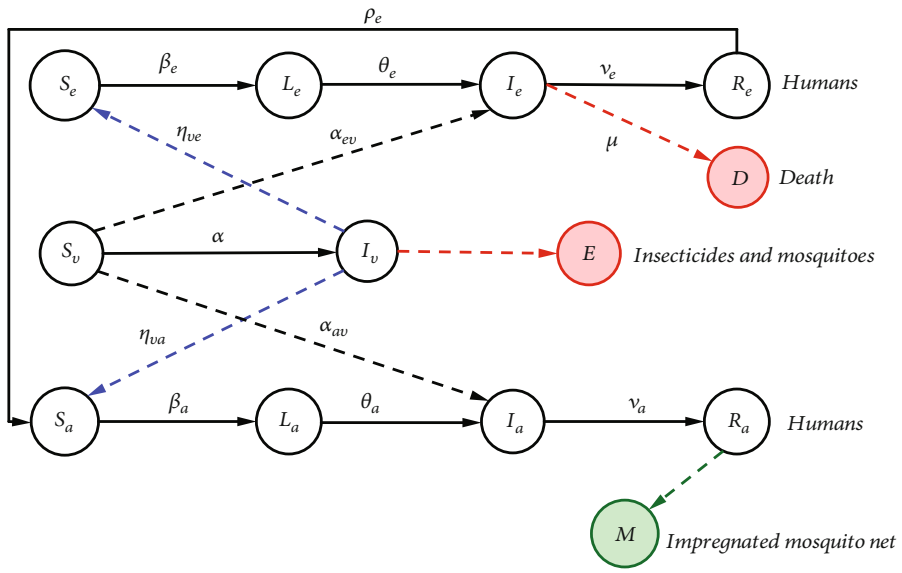

Figure 6: Diagram showing the progression of malaria in an endemic area.

Proof. The graph representing the Markov chain model is given in Figure 7, where the $x_i, i \in \{1, \cdots, 26\}$ correspond to the different parameters of the model and represent the transition probabilities of the chain.

There are three absorbing states (i_{11}, i_{12}, i_{13}) on the graph. As a result, the chain is absorbent. And under the assumptions A1,...,A5 and under the same interactions between humans and mosquitoes described in the previous section, we group the p_{ij} in a matrix P whose canonical form is represented by the relation

$$P = \begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \\ D \\ E \\ M \end{array} \begin{array}{c} \begin{array}{cccccccccccc} S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a & D & E & M \end{array} \\ \begin{pmatrix} r_e & \beta_e & \beta_{e1} & \beta_{e2} & 0 & 0 & \rho_e & \beta_{e3} & \beta_{e4} & \beta_{e5} & 0 & e_1 & m_1 \\ 0 & q_e & \theta_e & \theta_{e1} & 0 & 0 & \theta_{e2} & \theta_{e3} & \theta_{e4} & \theta_{e5} & 0 & e_2 & m_2 \\ 0 & 0 & p_e & \nu_e & 0 & 0 & \nu_{e1} & \nu_{e2} & \nu_{e3} & \nu_{e4} & \mu & e_3 & m_3 \\ 0 & 0 & 0 & r_e & 0 & 0 & \rho_e & \rho_{e1} & \rho_{e2} & \rho_{e3} & 0 & e_4 & m_4 \\ 0 & 0 & 0 & 0 & \alpha' & \alpha & 0 & 0 & 0 & 0 & 0 & p' & 0 \\ \eta_{ve} & \eta_{ve1} & \eta_{ve2} & \eta_{ve3} & 0 & p_v & \eta_{va} & \eta_{va1} & \eta_{va2} & \eta_{ve3} & 0 & p & 0 \\ 0 & 0 & 0 & 0 & 0 & t_1 & r_a & \eta_a & \beta_{a1} & \beta_{a2} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & q_a & \theta_a & \theta_{a1} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & t_2 & 0 & 0 & 0 & p_a & \eta_{va} & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & \rho_a & \beta_a & \theta_a & r_a & 0 & 0 & p_2 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \end{pmatrix} \end{array}. \quad (22)$$

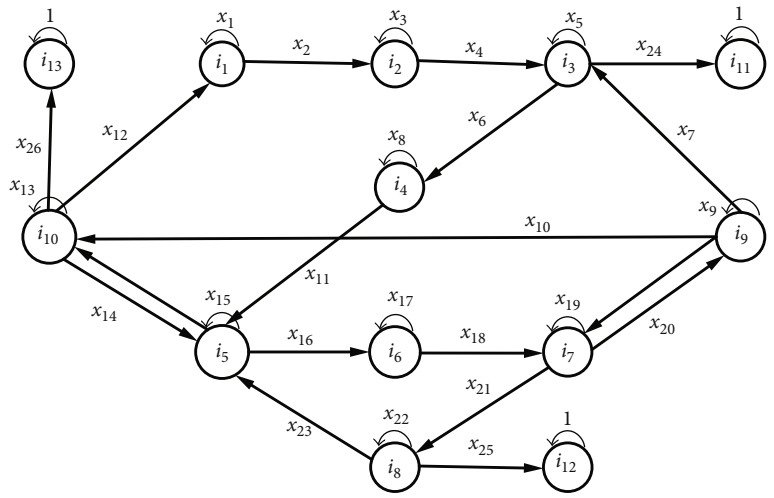

Figure 7: Transition graph of the absorbing Markov chain.

The matrix of transient states Q and that of absorbing states R are given by the relations

$$Q = \begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \end{array} \begin{array}{c} \begin{array}{cccccccccc} S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \end{array} \\ \begin{pmatrix} r_e & \beta_e & \beta_{e1} & \beta_{e2} & 0 & 0 & \rho_e & \beta_{e3} & \beta_{e4} & \beta_{e5} \\ 0 & q_e & \theta_e & \theta_{e1} & 0 & 0 & \theta_{e2} & \theta_{e3} & \theta_{e4} & \theta_{e5} \\ 0 & 0 & p_e & \nu_e & 0 & 0 & \nu_{e1} & \nu_{e2} & \nu_{e3} & \nu_{e4} \\ 0 & 0 & 0 & r_e & 0 & 0 & \rho_e & \rho_{e1} & \rho_{e2} & \rho_{e3} \\ 0 & 0 & 0 & 0 & \alpha' & \alpha & 0 & 0 & 0 & 0 \\ \eta_{ve} & \eta_{ve1} & \eta_{ve2} & \eta_{ve3} & 0 & p_v & \eta_{va} & \eta_{va1} & \eta_{va2} & \eta_{ve3} \\ 0 & 0 & 0 & 0 & 0 & t_1 & r_a & \beta_a & \beta_{a1} & \beta_{a2} \\ 0 & 0 & 0 & 0 & 0 & 0 & 0 & q_a & \theta_a & \theta_{a1} \\ 0 & 0 & 0 & 0 & t_2 & 0 & 0 & 0 & p_a & \eta_{va} \\ 0 & 0 & 0 & 0 & 0 & 0 & \rho_a & \beta_a & \theta_a & r_a \end{pmatrix} \end{array}. \tag{23}$$

$$R = \begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \end{array} \begin{array}{c} \begin{array}{ccc} D & E & M \end{array} \\ \begin{pmatrix} 0 & e_1 & m_1 \\ 0 & e_2 & m_2 \\ \mu & e_3 & m_3 \\ 0 & e_4 & m_4 \\ 0 & p' & 0 \\ 0 & p & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & 0 \\ 0 & 0 & p_2 \end{pmatrix} \end{array}. \tag{24}$$

The fundamental matrix will be determined by application. □

Proposition 7. *The matrix elements $n_{i,j} \coloneqq (I-Q)_{i,j}$ give us the expectation of the total number $N_i(j)$ of stays in j before passing beyond the transient states, and the i line of $(I-Q)^{-1}$ gives us the "expected life balance," with a given initial state i.*

Proof. Let I_k be the indicator of being present in the transient state at time k. We pose

$$N = \sum_{k=0}^{\infty} I_k. \tag{25}$$

We have $n_i = \sum_{k=0}^{\infty} E_i I_k$. Considering also the breakdown into indicators

$$\begin{aligned} I_k &= \sum_{j \in \mathbb{E}} I_{k,j}, \\ I_{k,j} &= 1_{\{X(k)=j\}}, \end{aligned} \tag{26}$$

where $I_{k,j}$ is the indicator of being in position $j \in \mathbb{E}$ at time k. As a result,

$$n_i = \sum_{k=0}^{\infty} \sum_{j \in \mathbb{E}} E_i I_k = \sum_{k=0}^{\infty} \sum_{j \in \mathbb{E}} (Q)_{i,j}^{k}. \tag{27}$$

By changing the order of summation, we obtain

$$n_i = \sum_{j \in \mathbb{E}} \left(\sum_{k=0}^{\infty} (Q)_{i,j}^{k} \right) = \sum_{j \in \mathbb{E}} (I-Q)_{i,j} = \sum_{j \in \mathbb{E}} n_{i,j}, \tag{28}$$

where $n_{i,j} = (I-Q)_{i,j}$ is the total expected time spent in j starting from state i. □

4. Numerical Simulations

4.1. Values Assigned to Transition Probabilities. In this section, we numerically analyse the stochastic matrices (18) and (22). We consider two malaria transmission zones: zone A corresponding to a low transmission zone and zone B corresponding to a high transmission zone. We begin by determining the values of the biological parameters equivalent to the transition probabilities in each zone. Most of these data are obtained from the literature, in particular from models developed by [27–31].

4.1.1. The Transition Probabilities between the Three Types of Host. The probability of effective transition between the three host types is the average number of contacts per unit of time (in this case the day) that could lead to infection of a specific host type.

n_a: we estimate that the average number of mosquito bites a human being can suffer per day is 0.28 for the low transmission zone and 0.38 for the high transmission zone. This average number n_a is a function of the exposed surface area of the human and any vector control interventions used by humans to reduce exposure to mosquitoes [27, 30].

η_{ve}: we estimate that the probability of transmission of an infection from an infectious mosquito (I_v) to a susceptible nonimmune human being (S_e), knowing that there has been contact between the two, is 0.021 for the low transmission zone and 0.07 for the high transmission zone [30].

η_{va}: we estimate that the probability of transmission of an infection from an infectious mosquito (I_v) to a susceptible semi-immune human being (S_a), knowing that there has been contact between the two, is 0.012 for the low transmission zone and 0.022 for the high transmission zone [28, 31].

α_{ev}: we assume that the probability of transmission of infection from an infectious nonimmune human being (I_e) to a susceptible mosquito (S_v), given that there has been contact between the two, is 0.11 for a low transmission zone and 0.45 for a high transmission zone [31].

α_{av}: we assume that the probability of transmission of infection from an infectious semi-immune human being (I_a) to a susceptible mosquito (S_v), given that there has been contact between the two, is 0.08 for an area of low transmission and 0.35 for an area of high transmission.

4.1.2. Transitions in Nonimmune Host Compartments. β_e: the probability of transition from the susceptible state (S_e) to the latent state (L_e). This parameter results from the infection force which is defined by

$$\beta_e = \eta_{ve} n_a i_v \frac{N_v}{N_h}, \tag{29}$$

where N_h is the human population size and N_v is the mosquito population size [28–30].

θ_e: the probability of transition from the latent state (L_e) to the infectious state (I_e). We assume that $\theta_e = 0.10$ for both types of zone.

ν_e: the probability of transition from the infectious state (I_e) to the immune state (R_e). We have assumed that this rate is 0.005 for the zone of low transmission and 0.001 for the zone of high transmission zone.

ρ_e: the probability of transition from the immune state (R_e) to the susceptible state (S_a). This is a phase of change of status from nonimmune to semi-immune hosts. We have assumed that this probability is 5.5×10^{-4} for the low transmission zone and 2.7×10^{-3} for the high transmission zone [27, 29, 31].

4.1.3. Transitions in the Compartments of Semi-immune Hosts. β_a: the probability of transition from the susceptible state (S_a) to the latent state (L_a). This parameter results from the strength of infection of the semi-immunes, which is defined by

$$\beta_a = \eta_{va} n_a i_v \frac{N_v}{N_h}, \tag{30}$$

where N_h is the size of the human population and N_v is the size of the mosquito population [28–30].

θ_a: the probability of transition from the latent state (L_a) to the infectious state (I_a). We assume that $\theta_a = 0.09$ for both types of zone.

ν_a: the probability of transition from the infectious state (I_a) to the immune state (R_a). We have assumed that this rate is 0.01 for both types of zone.

ρ_a: the probability of transition from the immune state (R_a) to the susceptible state (S_a). Here, the immune cells heal and return to the susceptible compartment. We assume that $\rho_a = 0.0083$ for the low transmission zone and $\rho_a = 0.033$ for the high transmission zone [30].

For $i = j$, the p_{ii} are obtained by stochastic effect. The other transition probabilities not quoted here are obtained by using the Markov property.

All these estimated probabilities are summarised in Table 5.

Table 5: Transition probabilities in zones A and B.

Param.	Zone A	Zone B	Intervals
n_a	0.25	0.38	0.13-0.47
η_{ve}	0.021	0.07	0.01-0.27
η_{va}	0.012	0.022	0.01-0.27
α_{ev}	0.11	0.45	0.072-0.64
α_{va}	0.08	0.35	0.072-0.64
θ_e	0.10	0.10	0.067-0.20
θ_a	0.09	0.09	0.067-0.20
ν_e	0.005	0.001	0.0014-0.017
ν_a	0.01	0.01	0.0014-0.017
ρ_e	5.5×10^{-4}	2.7×10^{-3}	$1.1 \times 10^{-2} - 5.5 \times 10^{-5}$

4.2. Study of the H Matrix in a Zone A. In this section, we numerically analyse the H matrices derived from the relationship (18) using the parameter values obtained in Table 5 (zone A), which correspond to a stable transmission zone. We obtain the H matrix of the relationship (31) with values in $[0, 1[$.

$$H = \begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \end{array} \begin{array}{c} \begin{array}{cccccccccc} S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \end{array} \\ \begin{pmatrix} 0.719 & 0.030 & 0.100 & 0.010 & 0.000 & 0.000 & 0.011 & 0.030 & 0.090 & 0.010 \\ 0.000 & 0.725 & 0.100 & 0.010 & 0.000 & 0.000 & 0.015 & 0.060 & 0.080 & 0.010 \\ 0.000 & 0.000 & 0.748 & 0.100 & 0.000 & 0.000 & 0.012 & 0.030 & 0.090 & 0.020 \\ 0.000 & 0.000 & 0.000 & 0.859 & 0.000 & 0.000 & 0.011 & 0.030 & 0.090 & 0.010 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.810 & 0.190 & 0.000 & 0.000 & 0.000 & 0.000 \\ 0.210 & 0.030 & 0.100 & 0.010 & 0.000 & 0.518 & 0.012 & 0.020 & 0.090 & 0.010 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.211 & 0.649 & 0.030 & 0.090 & 0.020 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.900 & 0.090 & 0.010 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.229 & 0.000 & 0.000 & 0.000 & 0.759 & 0.012 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.080 & 0.030 & 0.090 & 0.800 \end{pmatrix} \end{array}. \quad (31)$$

We consider a heterogeneous population of humans and mosquitoes made up of eighty people and twenty mosquitoes assumed to be under normal conditions of temperature and reproduction. The human population is made up of fifty-seven nonimmune host types and twenty-three semi-immune host types. This gives the initial distribution given by the relationship

$$\pi_0 = (\,0.200,\quad 0.150,\quad 0.100,\quad 0.120,\quad 0.090,\quad 0.110,\quad 0.050,\quad 0.080,\quad 0.060,\quad 0.040\,), \quad (32)$$

that is, 20% of susceptible nonimmune, 15% of latent nonimmune, 10% of infectious nonimmune, 12% of recovered nonimmune, 9% of susceptible mosquitoes, 11% of infectious mosquitoes, 5% of susceptible semi-immune, 8% of latent semi-immune, 6% of infectious semi-immune, and 4% of recovered semi-immune. We use R version 4.2.1 (2022-06-23 ucrt)-"Funny-Looking Kid" for the simulation results. Tables 6 and 7 give the probabilities $\pi_n = \pi_0 H^n$ for $n = \{1, 2, \cdots, 21\}$. They illustrate the evolution of the different stages of malaria over a period of twenty-one malaria

Table 6: The state of the probability of individuals after twenty-one episodes for an initial vector π_0 given by $\pi_0 = (0.200,\ 0.150,\ 0.100,\ 0.120,\ 0.090,\ 0.110,\ 0.050,\ 0.080,\ 0.060,\ 0.040)$.

	1	2	3	4	5	6	7	8	9	10
S_e	0.167	0.138	0.114	0.095	0.081	0.072	0.067	0.064	0.063	0.064
L_e	0.118	0.093	0.074	0.059	0.047	0.039	0.032	0.028	0.024	0.022
I_e	0.121	0.127	0.125	0.119	0.110	0.102	0.094	0.088	0.083	0.080
R_e	0.118	0.117	0.116	0.115	0.113	0.110	0.106	0.102	0.099	0.095
S_v	0.087	0.098	0.116	0.137	0.158	0.177	0.193	0.207	0.218	0.226
I_v	0.085	0.070	0.063	0.062	0.065	0.070	0.076	0.082	0.087	0.092
S_a	0.044	0.039	0.036	0.033	0.030	0.028	0.027	0.026	0.025	0.024
L_a	0.099	0.112	0.122	0.129	0.133	0.135	0.136	0.136	0.136	0.135
I_a	0.121	0.162	0.188	0.205	0.214	0.219	0.220	0.219	0.218	0.216
R_a	0.042	0.044	0.046	0.047	0.048	0.048	0.048	0.048	0.047	0.047

Table 7: The state of the probability of individuals after twenty-one episodes for an initial vector π_0 given by $\pi_0 = (0.200, 0.150, 0.100, 0.120, 0.090, 0.110, 0.050, 0.080, 0.060, 0.040)$.

	11	12	13	14	15	16	17	18	19	20	21
S_e	0.065	0.067	0.069	0.071	0.072	0.074	0.075	0.076	0.077	0.078	0.078
L_e	0.021	0.020	0.019	0.019	0.019	0.019	0.019	0.019	0.020	0.020	0.020
I_e	0.077	0.076	0.075	0.075	0.076	0.076	0.077	0.078	0.078	0.079	0.079
R_e	0.091	0.088	0.085	0.082	0.080	0.078	0.077	0.076	0.075	0.074	0.074
S_v	0.233	0.238	0.241	0.243	0.245	0.246	0.246	0.247	0.247	0.247	0.247
I_v	0.096	0.099	0.101	0.103	0.104	0.105	0.106	0.106	0.107	0.107	0.107
S_a	0.023	0.023	0.023	0.022	0.022	0.022	0.220	0.022	0.022	0.022	0.022
L_a	0.134	0.133	0.132	0.130	0.130	0.129	0.128	0.127	0.127	0.126	0.126
I_a	0.214	0.212	0.210	0.209	0.208	0.207	0.206	0.205	0.205	0.205	0.205
R_a	0.046	0.045	0.045	0.044	0.044	0.044	0.044	0.043	0.043	0.043	0.043

episodes, using the initial distribution π_0 of the relationship (32).

The stationary distribution obtained is π^* and is given by the relation

$$\pi^* = \{0.079, 0.020, 0.080, 0.073, 0.247, 0.107, 0.022, 0.125, 0.204, 0.043\}. \quad (33)$$

Graphical representations of the results of Tables 6 and 7 are listed in Figure 8.

For a given initial distribution, the population of susceptible Anopheles mosquitoes increases and stabilises at around 25%. There has also been an increase in the number of infectious mosquitoes. The rapid decline in the susceptible nonimmune curve led to rapid growth in the susceptible semi-immune curve, creating a peak of probability 0.22 which stabilised after the twentieth episode. After several episodes of malaria, nonimmune individuals gradually migrate to the semi-immune class to acquire a certain level of immunity, leading to a high level of semi-immune individuals, which stabilises from the twentieth episode onwards.

4.3. Study of the P Matrix in an Endemic Area. Using the parameters obtained in Table 5 (zone B), we obtain the numerical values of the absorbing matrix P of the relation (22) described by the relation

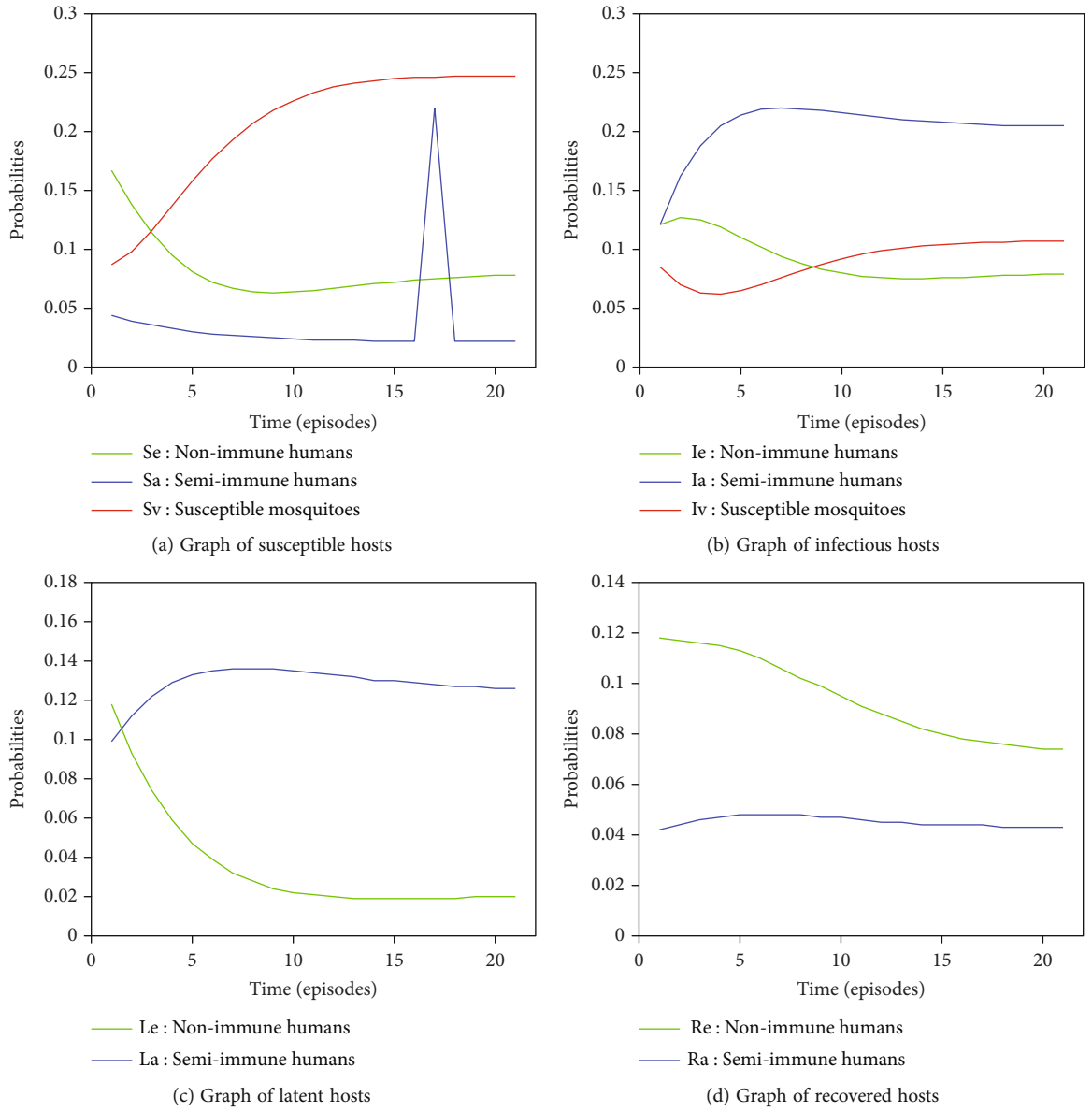

Figure 8: Simulation of malaria host trajectories.

$$
\begin{array}{c}
\\
S_e \\
L_e \\
I_e \\
R_e \\
S_v \\
I_v \\
S_a \\
L_a \\
I_a \\
R_a \\
D \\
E \\
M
\end{array}
\begin{array}{c}
\begin{array}{cccccccccccc}
S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a & D & E & M
\end{array} \\
\begin{pmatrix}
0.129 & 0.030 & 0.100 & 0.010 & 0.000 & 0.000 & 0.011 & 0.030 & 0.090 & 0.010 & 0.000 & 0.350 & 0.240 \\
0.000 & 0.225 & 0.100 & 0.010 & 0.000 & 0.000 & 0.015 & 0.060 & 0.080 & 0.010 & 0.000 & 0.240 & 0.260 \\
0.000 & 0.000 & 0.107 & 0.100 & 0.000 & 0.000 & 0.012 & 0.030 & 0.090 & 0.020 & 0.041 & 0.250 & 0.350 \\
0.000 & 0.000 & 0.000 & 0.349 & 0.000 & 0.000 & 0.011 & 0.030 & 0.090 & 0.010 & 0.000 & 0.260 & 0.250 \\
0.000 & 0.000 & 0.000 & 0.000 & 0.310 & 0.190 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.500 & 0.000 \\
0.210 & 0.030 & 0.100 & 0.010 & 0.000 & 0.018 & 0.012 & 0.020 & 0.090 & 0.010 & 0.000 & 0.500 & 0.000 \\
0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.211 & 0.659 & 0.030 & 0.090 & 0.020 & 0.000 & 0.000 & 0.000 \\
0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.900 & 0.090 & 0.010 & 0.000 & 0.000 & 0.000 \\
0.000 & 0.000 & 0.000 & 0.000 & 0.229 & 0.000 & 0.000 & 0.000 & 0.759 & 0.012 & 0.000 & 0.000 & 0.000 \\
0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.080 & 0.030 & 0.090 & 0.300 & 0.000 & 0.000 & 0.500 \\
0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 1.000 & 0.000 & 0.000 \\
0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 1.000 & 0.000 \\
0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 1.000
\end{pmatrix}
\end{array}. \tag{34}
$$

The matrix of transient states Q and that of absorbing states R of the matrix P are given in the following the relations.

$$Q = \begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \end{array} \begin{array}{c} \begin{array}{cccccccccc} S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \end{array} \\ \begin{pmatrix} 0.129 & 0.030 & 0.100 & 0.010 & 0.000 & 0.000 & 0.011 & 0.03 & 0.090 & 0.010 \\ 0.000 & 0.225 & 0.100 & 0.010 & 0.000 & 0.000 & 0.015 & 0.06 & 0.080 & 0.010 \\ 0.000 & 0.000 & 0.107 & 0.100 & 0.000 & 0.000 & 0.012 & 0.03 & 0.090 & 0.020 \\ 0.000 & 0.000 & 0.000 & 0.349 & 0.000 & 0.000 & 0.011 & 0.03 & 0.090 & 0.010 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.310 & 0.190 & 0.000 & 0.00 & 0.000 & 0.000 \\ 0.210 & 0.030 & 0.100 & 0.010 & 0.000 & 0.018 & 0.012 & 0.02 & 0.090 & 0.010 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.211 & 0.659 & 0.03 & 0.090 & 0.020 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.90 & 0.090 & 0.010 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.229 & 0.000 & 0.000 & 0.00 & 0.759 & 0.012 \\ 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.080 & 0.03 & 0.090 & 0.300 \end{pmatrix} \end{array}. \tag{35}$$

$$R = \begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \end{array} \begin{array}{c} \begin{array}{ccc} D & E & M \end{array} \\ \begin{pmatrix} 0.000 & 0.350 & 0.240 \\ 0.000 & 0.240 & 0.260 \\ 0.041 & 0.250 & 0.350 \\ 0.000 & 0.260 & 0.250 \\ 0.000 & 0.500 & 0.000 \\ 0.000 & 0.500 & 0.000 \\ 0.000 & 0.000 & 0.000 \\ 0.000 & 0.000 & 0.000 \\ 0.000 & 0.000 & 0.000 \\ 0.000 & 0.000 & 0.500 \end{pmatrix} \end{array}. \tag{36}$$

4.3.1. Fundamental Matrix. The fundamental matrix of an absorbing Markov chain can be used to extract many properties of this chain. In particular, it can be used to determine the average number of visits to a given state before absorption, the time expectancy until absorption starting from a given state, and the probabilities of being absorbed in a given state k, starting from a state i. The fundamental matrix resulting from the P matrix is given by the relation

$$\begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \end{array} \begin{array}{c} \begin{array}{cccccccccc} S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \end{array} \\ \begin{pmatrix} 1.163 & 0.047 & 0.142 & 0.041 & 0.251 & 0.061 & 0.058 & 0.475 & 0.757 & 0.044 \\ 0.019 & 1.294 & 0.156 & 0.045 & 0.307 & 0.077 & 0.080 & 0.898 & 0.926 & 0.056 \\ 0.014 & 0.003 & 1.128 & 0.175 & 0.241 & 0.060 & 0.061 & 0.444 & 0.725 & 0.056 \\ 0.016 & 0.003 & 0.010 & 1.539 & 0.281 & 0.068 & 0.064 & 0.519 & 0.846 & 0.047 \\ 0.072 & 0.014 & 0.043 & 0.013 & 1.522 & 0.298 & 0.018 & 0.116 & 0.219 & 0.013 \\ 0.261 & 0.052 & 0.156 & 0.045 & 0.264 & 1.084 & 0.067 & 0.421 & 0.795 & 0.047 \\ 0.188 & 0.037 & 0.113 & 0.033 & 0.687 & 0.780 & 3.010 & 1.228 & 2.069 & 0.157 \\ 0.066 & 0.013 & 0.040 & 0.012 & 1.348 & 0.275 & 0.067 & 10.185 & 4.063 & 0.223 \\ 0.070 & 0.014 & 0.042 & 0.012 & 1.462 & 0.290 & 0.035 & 0.140 & 4.406 & 0.085 \\ 0.033 & 0.007 & 0.020 & 0.006 & 0.324 & 0.138 & 0.351 & 0.595 & 0.977 & 1.467 \end{pmatrix} \end{array}. \tag{37}$$

Thus, for an individual in the state S_e, the average number of months before absorption by D or M is $1.163 \approx 35$ days in S_e, $0.047 \approx 2$ days in L_e, $0.142 \approx 4$ days in I_e, $0.475 \approx 14$ days in L_a, and 0.757 in I_a. For infectious mosquitoes I_e, the average number of months before absorption by E (eliminated by insecticides) or M (eliminated by LLINs) is $0.261 \approx 7$ days in S_e, $0.052 \approx 2$ days in L_e, and $0.156 \approx 5$ days in I_e. With absorbing Markov chains, all equilibrium distributions will be limited to state absorbents, here D, E, and M. Also, we can determine the probabilities of absorption of states S_e to R_a by states D, E, and M. To do this, let us consider the matrix $B = NR$ defined by the relation

$$B = \begin{array}{c} \\ S_e \\ L_e \\ I_e \\ R_e \\ S_v \\ I_v \\ S_a \\ L_a \\ I_a \\ R_a \end{array} \begin{array}{c} \begin{array}{ccc} D & E & M \end{array} \\ \begin{pmatrix} 0.006 & 0.621 & 0.374 \\ 0.006 & 0.560 & 0.435 \\ 0.046 & 0.483 & 0.471 \\ 0.000 & 0.584 & 0.417 \\ 0.002 & 0.953 & 0.046 \\ 0.006 & 0.829 & 0.166 \\ 0.005 & 0.845 & 0.181 \\ 0.002 & 0.851 & 0.148 \\ 0.002 & 0.918 & 0.081 \\ 0.001 & 0.251 & 0.752 \end{pmatrix} \end{array}. \quad (38)$$

According to these results, the probability of an individual S_e, L_e, I_e, R_e, S_a, L_a, I_a, and R_a dying from malaria is equal to 0.006, 0.006, 0.046, 0.000, 0.005, 0.002, and 0.001, respectively. It can be seen that the probability of an infectious individual I_e dying from malaria is consistent with the clinical results. Susceptible mosquitoes S_v (infectious I_v, respectively) are absorbed either by insecticides E at 95.3% (at 82.9%, respectively) or by LLINs at 04.6% (at 16.6%, respectively). The results show that to eliminate malaria, it is preferable to use indoor residual spraying (IRS) by regular application of insecticides rather than long-acting impregnated mosquito nets (LLINs). Susceptible individuals S_e and S_a are absorbed by IRS at 62.1% and 84.5% and by LLIN at 37.4% and 14.8%, respectively. This means that susceptible humans prefer IRS rather than LLINs to protect themselves against parasite infection.

5. Conclusion

In this study, we proposed a Markovian stochastic approach to a compartment model regarding malaria transmission. This Markovian model $(S_eL_eI_eR_eS_e\text{-}S_aL_aI_aR_aS_a\text{-}S_vI_v)$ generalizes the host-vector compartment models of types $(S_hI_h\text{-}S_vI_v)$, $(S_hI_hR_hS_h\text{-}S_vI_v)$, and $(S_hL_hI_hR_hS_h\text{-}S_vL_vI_v)$. The study of this model made it possible to identify a technique suitable for combating malaria. Our model constitutes a valuable tool for the stochastic modeling of epidemics: it is used to predict the evolution of the dynamics of malaria in a human and anopheline population. In the forthcoming paper, we will study the geometric V-ergodicity of the model by including the effect of abiotic factors such as temperature.

Data Availability

All the data is in the article.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

References

[1] R. Ross, *The Prevention of Malaria*, John Murray, 1911.

[2] R. W. Mbogo, L. S. Luboobi, and J. W. Odhiambo, "A stochastic model for malaria transmission dynamics," *Journal of Applied Mathematics*, vol. 2018, Article ID 2439520, 13 pages, 2018.

[3] R. I. Gweryina and A. R. Kimbir, "Dynamical behavior of a malaria relapse model with insecticide treated nets (ITNs) as protection measure," *Applications and Applied Mathematics: An International Journal (AAM)*, vol. 15, no. 1, p. 9, 2020.

[4] H.-F. Huo and G.-M. Qiu, "Stability of a mathematical model of malaria transmission with relapse," *Abstract and Applied Analysis*, vol. 2014, Article ID 289349, 9 pages, 2014.

[5] Y. Cai, Y. Kang, and W. Wang, "A stochastic SIRS epidemic model with nonlinear incidence rate," *Applied Mathematics and Computation*, vol. 305, pp. 221–240, 2017.

[6] X. Ren and T. Zhang, "Global analysis of an SEIR epidemic model with a ratio-dependent nonlinear incidence rate," *Journal of Applied Mathematics and Physics*, vol. 5, no. 12, pp. 2311–2319, 2017.

[7] R. C. Reiner Jr., T. A. Perkins, C. M. Barker et al., "A systematic review of mathematical models of mosquito-borne pathogen transmission: 1970–2010," *Journal of The Royal Society Interface*, vol. 10, no. 81, article 20120921, 2013.

[8] D. Arku, G. Kallah-Dagadu, and G. Debrah, "A stochastic H-U model for malaria transmission via Markov theory," *Journal of Biostatistics*, vol. 18, no. 3, pp. 459–473, 2021.

[9] P. Bedi, A. Kumar, T. Abdeljawad, and A. Khan, "Existence of mild solutions for impulsive neutral Hilfer fractional evolution equations," *Advances in Difference Equations*, vol. 2020, no. 1, 16 pages, 2020.

[10] A. Khan, Y. Li, K. Shah, and T. S. Khan, "On coupled p-laplacian fractional differential equations with nonlinear boundary conditions," *Complexity*, vol. 2017, Article ID 8197610, 9 pages, 2017.

[11] R. Begum, O. Tunç, H. Khan, H. Gulzar, and A. Khan, "A fractional order Zika virus model with Mittag–Leffler kernel," *Chaos, Solitons & Fractals*, vol. 146, article 110898, 2021.

[12] A. Devi, A. Kumar, D. Baleanu, and A. Khan, "On stability analysis and existence of positive solutions for a general nonlinear fractional differential equations," *Advances in Difference Equations*, vol. 2020, no. 1, 16 pages, 2020.

[13] A. Devi, A. Kumar, T. Abdeljawad, and A. Khan, "Stability analysis of solutions and existence theory of fractional Lagevin equation," *Alexandria Engineering Journal*, vol. 60, no. 4, pp. 3641–3647, 2021.

[14] D. Bichara, "*Study of epidemiological models: stability, observation and estimation of parameters, [Ph.D. thesis]*," University of Lorraine, 2013.

[15] P. Van den Driessche and J. Watmough, "Reproduction numbers and sub-threshold endemic equilibria for compartmental

models of disease transmission," *Mathematical Biosciences*, vol. 180, no. 1-2, pp. 29–48, 2002.

[16] J. M. M. Ondo, "*Spatial aspects in epidemiological modelling, [Ph.D. thesis]*," University of Grenoble, 2012.

[17] J. Banasiak, R. Ouifki, and W. A. Woldegerima, "Some mathematical tools for modelling malaria: a subjective survey," *Biomath*, vol. 10, article 2110029, 2021.

[18] F. Mugabi, K. J. Duffy, J. Y. T. Mugisha, and O. C. Collins, "Determining the effects of transplacental and direct transmission on the probability of persistence in a bluetongue virus model in temperate and tropical regions," *Results in Applied Mathematics*, vol. 7, article 100120, 2020.

[19] M. A. Khan and A. Atangana, "Modeling the dynamics of novel coronavirus (2019-nCov) with fractional derivative," *Alexandria Engineering Journal*, vol. 59, no. 4, pp. 2379–2389, 2020.

[20] A. Atangana and S. I. Araz, "Advanced analysis in epidemiological modeling: detection of wave," medRxiv, 2021.

[21] E. Kalipeni, "Determinants of infant mortality in Malawi: a spatial perspective," *Social Science & Medicine*, vol. 37, no. 2, pp. 183–198, 1993.

[22] T. A. Smith, R. Leuenberger, and C. Lengeler, "Child mortality and malaria transmission intensity in Africa," *Trends in Parasitology*, vol. 17, no. 3, pp. 145–149, 2001.

[23] K. Marsh and R. W. Snow, "Host—parasite interaction and morbidity in malaria endemic areas," *Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences*, vol. 352, no. 1359, pp. 1385–1394, 1997.

[24] D. Payne, B. Grab, R. E. Fontaine, and J. H. G. Hempel, "Impact of control measures on malaria transmission and general mortality," *Bulletin of the World Health Organization*, vol. 54, no. 4, pp. 369–377, 1976.

[25] E. Andersen, "Assessment of age-dependent immunity to malaria in transmigrants," *The American Journal of Tropical Medicine and Hygiene*, vol. 56, no. 6, pp. 647–649, 1997.

[26] M. J. Barcus, Krisin, I. R. Elyazar et al., "Primary infection by *Plasmodium falciparum* or *P. vivax* in a cohort of Javanese migrants to Indonesian Papua," *Annals of Tropical Medicine & Parasitology*, vol. 97, no. 6, pp. 565–574, 2003.

[27] H. M. Yang, "Malaria transmission model for different levels of acquired immunity and temperature-dependent parameters (vector)," *Revista de Saude Publica*, vol. 34, no. 3, pp. 223–231, 2000.

[28] H. M. Yang and M. U. Ferreira, "Assessing the effects of global warming and local social and economic conditions on the malaria transmission," *Revista de Saude Publica*, vol. 34, pp. 214–222, 2000.

[29] G. A. Ngwa and W. S. Shu, "A mathematical model for endemic malaria with variable human and mosquito populations," *Mathematical and Computer Modelling*, vol. 32, no. 7-8, pp. 747–763, 2000.

[30] P. Zongo, "*Mathematical modeling of malaria transmission dynamics, [Ph.D. thesis]*," University of Ouagadougou, 2009.

[31] N. Chitnis, J. M. Hyman, and J. M. Cushing, "Determining important parameters in the spread of malaria through the sensitivity analysis of a mathematical model," *Bulletin of Mathematical Biology*, vol. 70, no. 5, pp. 1272–1296, 2008.