Research Article

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

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This work presents a stochastic model of malaria spread. We first calculated the basic reproduction number $R_0$ of the models $\text{ShIhSh-SvIvSv}$ and $\text{ShIhRhSh-SvIv}$ 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 $\{\text{SeLeIeRe-SeSaLaIaRaSa-SvIv}\}$. 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 $\pi^*$ 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 $\{\text{ShIhSh-SvIvSv}\}$ of Ross, the models $\{\text{ShIhRhSh-SvIv}\}$ in [2–4], and the models $\{\text{ShEhIhRhSh}\}$ 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 $\{\text{ShIhSh-SvIvSv}\}$, $\{\text{ShIhRhSh-SvIv}\}$, and $\{\text{ShIhRhSh-SvIv}\}$. 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 $\{\text{ShIhSh-SvIvSv}\}$ 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
The malaria propagation graph in Ross’s \((S_h I_h S_v I_v S_v)\) model is shown in Figure 1.

He obtained the differential system (1) governing the \((S_h I_h S_v S_v I_v S_v)\) model of malaria.

\[
\begin{align*}
\frac{dS_h}{dt} &= \mu_h H - b_1 a \frac{S_h}{H} - \gamma_h I_h - \mu_h I_h, \\
\frac{dI_h}{dt} &= b_1 a I_v \frac{S_h}{H} - (\gamma_h + \mu_h) I_h, \\
\frac{dS_v}{dt} &= b_2 a (V - I_v) \frac{I_h}{H} - (\gamma_v + \mu_v) I_v, \\
\frac{dI_v}{dt} &= b_2 a (V - I_v) \frac{I_h}{H} - (\gamma_v + \mu_v) I_v.
\end{align*}
\]

(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 \((I_h, I_v)\).

\[
\begin{align*}
\frac{dI_h}{dt} &= b_1 a I_v \frac{S_h}{H} - (\gamma_h + \mu_h) I_h, \\
\frac{dI_v}{dt} &= b_2 a (V - I_v) \frac{I_h}{H} - (\gamma_v + \mu_v) I_v.
\end{align*}
\]

(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

\[
R_0 = mab \frac{1}{V} \frac{a}{\mu} \frac{1}{b_2}
\]

(3)

and formulates the following corollary [15, 16].

**Corollary 1.**

1. If \(R_0 \leq 1\), 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 \((S_h I_h R_h S_v I_v I_v)\). 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 \((S_h I_h S_v I_v S_v)\) 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.

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

\[
\begin{align*}
\frac{dS_h}{dt} &= \left[\lambda N - \frac{\beta S_h I_v}{N} + p R_h - \mu S_h\right] dt, \\
\frac{dI_h}{dt} &= \left[\frac{\beta S_h I_v}{N} + (1 - p) R_h - (\mu + \gamma) I_h\right] dt, \\
\frac{dR_h}{dt} &= \left[\gamma I_h - (\mu + 1) R_h\right] dt, \\
\frac{dS_v}{dt} &= \left[\eta V - \frac{\alpha_1 S_h I_h}{N} + \frac{\alpha_2 S_h R_h}{N} - \eta S_v\right] dt, \\
\frac{dI_v}{dt} &= \left[\frac{\alpha_1 S_h I_h}{N} + \frac{\alpha_2 S_h R_h}{N} - \eta I_v\right] dt,
\end{align*}
\]

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 \((S_h I_h R_h S_v S_v I_v)\) are also studied by authors such as [2–4].

2.3. Model \((S_h I_h R_h S_v I_v I_v)\). 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 \((S_h I_h R_h S_v S_v I_v I_v)\) model, which would be an extension of the \((S_h I_h S_v S_v I_v I_v)\) and \((S_h I_h R_h S_v S_v I_v I_v)\) 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

\[
\begin{align*}
\frac{dS_h}{dt} &= \left[\lambda N - \beta S_h I_v\right] dt, \\
\frac{dI_h}{dt} &= \left[\beta S_h I_v - (k + \mu) I_h\right] dt, \\
\frac{dI_v}{dt} &= \left(k p I_h - (\alpha + \mu) I_v\right) dt, \\
\frac{dR_h}{dt} &= \left(a I_h - k (1 - p) I_h - (\gamma + \mu) R_h\right) dt.
\end{align*}
\]

(5)
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 \(N = S + L + I + R\).

\[
\begin{align*}
\frac{dS}{dt} &= \left(\lambda N - \beta \frac{SI}{N} - \mu S + \gamma R\right) dt, \\
\frac{dL}{dt} &= \left(\beta \frac{SI}{N} - (k + \mu)L\right) dt, \\
\frac{dI}{dt} &= (kpL - (\alpha + \mu)L) dt.
\end{align*}
\] (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 \((S_h, I_h, S_v, I_v)\) in Section 2.2 and \((S_v, L_v, I_v, R_v)\) in Section 2.3 are given by

\[
\begin{align*}
R_{01} &= \sqrt{\frac{\beta \alpha_1 (1 + \mu) + \beta \alpha_2 \gamma}{\eta(\gamma + \mu)(1 + \mu) + \eta\gamma(-1 + p)}} \\
R_{02} &= \frac{\beta kp}{(k + \mu)(\alpha + \mu)}.
\end{align*}
\] (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

The disease-free equilibrium (DFE) of this model is therefore \((N, 0, 0, 0)\). Authors such as [17, 19, 20] have also studied these types of models.
\[ \frac{dX}{dt} = \mathcal{F}_j(X) - \mathcal{V}_j(X), \]  \hspace{1cm} (8)

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 exiting the \( j \) compartment, respectively [15]. The Jacobian matrices of \( \mathcal{F}(X) \) and \( \mathcal{V}(X) \) at equilibrium without disease \( E_0 \) are

\[
D\mathcal{F}(E_0) = F = \begin{pmatrix}
0 & 0 & \beta \\
0 & 0 & 0 \\
\alpha_1 & \alpha_2 & 0 \\
\end{pmatrix},
\]

\[
D\mathcal{V}(E_0) = V = \begin{pmatrix}
\gamma + \mu & \mu - 1 & 0 \\
-\gamma & \mu + 1 & 0 \\
0 & 0 & \eta \\
\end{pmatrix}.
\]

The inverse matrix of \( V \) is

\[
V^{-1} = \begin{pmatrix}
\frac{\mu + 1}{(\gamma + \mu)(\mu + 1) + \gamma(p - 1)} & 1 - p & 0 \\
\frac{\gamma}{(\gamma + \mu)(\mu + 1) + \gamma(p - 1)} & \frac{\mu + 1}{(\gamma + \mu)(\mu + 1) + \gamma(p - 1)} & 0 \\
0 & 0 & \eta^{-1} \\
\end{pmatrix}.
\]

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

with

\[
r_1 = \frac{\alpha_1(1 + \mu) + \alpha_2\gamma}{(\gamma + \mu)(1 + \mu) + \gamma(-1 + p)},
\]

\[
r_2 = \frac{\alpha_1(1 - p) + \alpha_2(\gamma + \mu)}{(\gamma + \mu)(1 + \mu) + \gamma(-1 + p)}.
\]

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

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

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

\[
R_0 = \rho(FV^{-1}). \hspace{1cm} (13)
\]

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

\[
R_{01} = \sqrt{\frac{\beta \alpha_1(1 + \mu) + \beta \alpha_2\gamma}{\eta(\gamma + \mu)(1 + \mu) + \eta\gamma(-1 + p)}}. \hspace{1cm} (14)
\]

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

\[
\mathcal{F}(S, L, I) = F = \begin{pmatrix}
\frac{\beta SI}{N} \\
0 \\
\end{pmatrix},
\]

\[
\mathcal{V}(S, L, I) = V = \begin{pmatrix}
(k + \mu)L \\
kpL + (a + \mu)I \\
\end{pmatrix}.
\]

Hence,

\[
F(\text{DFE}) = \begin{pmatrix}
0 & \beta \\
0 & 0 \\
\end{pmatrix},
\]

\[
V(\text{DFE}) = \begin{pmatrix}
k + \mu & 0 \\
kp & a + \mu \\
\end{pmatrix}.
\]

Therefore,

\[
FV^{-1} = \begin{pmatrix}
\beta kp & \beta \\
(k + \mu)(a + \mu) & 0 \\
\end{pmatrix}.
\]

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 \((S_a, L_a, I_a, R_a, S_v, I_v)\) for nonimmunes until they enter the semi-immune category and then follow a model of the type \((S_a, L_a, I_a, R_a, S_v, I_v)\), where \(e\) and \(a\) denote the index for nonimmunes and semi-immunes, respectively. For the mosquito population, we use a model of the type \((S_m, I_m)\).

#### 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.
(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 subordinated into four (4) compartments: susceptible (S), latent (L), infectious (I), and immune (R). Semi-immune human host types are also subordinated into four (4) compartments: susceptible (S), latent (L), infectious (I), and immune (R). The mosquito population is made up of susceptible (S) and infectious (I) 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) to a nonimmune susceptible human individual (S) or a semi-immune susceptible human individual.

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

3.2.2. Model Assumptions. A1: the probabilities $\eta_{ee}, \eta_{aa}, \alpha_{ee}, \alpha_{aa}$, and $\alpha$ are in the interval $[0,1]$

A2: the parameters $\beta_{ee}, \theta_{ee}, \eta_{ee}, \rho_e$, $\beta_{aa}, \theta_{aa}, \eta_{aa}$, and $\rho_a$ 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) bite susceptible humans (S) with a $\eta_{ee}$ probability. The latter may remain susceptible (S) with probability $r$ for some time or become latent (L) with probability $\beta_e$. Individuals can remain latent for a given time with probability $q$ or become infectious (I) with $\theta_e$ as the transition probability. In this infectious state (I), humans can die with a probability $\mu$ or become immune with a probability equal to $\nu_e$, or even remain infectious with a probability $p$ for an interval of time. Having completed the 1st cycle, the immune (R) becomes susceptible (S) to the $\rho_e$ immunity rate, and through the intermediary of the infectious mosquito (I), the cycle resumes as shown in Figure 4. At stage (R), having acquired a certain immunity, the individual re-enters the susceptible class (S) at the rate $\rho_a$. The susceptible mosquitoes (S) inoculate the parasite from a blood meal on the infectious (I) and with probabilities $\alpha_{ee}$ and $\alpha_{aa}$, respectively; then, they become infectious (I) with probability $a$.

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 $E$. Then, the process $X$ defined on $(\Omega, \mathcal{F}, \mathbb{P})$ with values in $E$ is an irreducible and aperiodic homogeneous Markov chain with initial distribution $\pi_0 = (\pi_0^x, \ldots, \pi_0^y)$ and transition matrix $H$, where $\pi_0^x$ is the probability that the process $X$ is in state $i$ at time 0. And this Markov chain has a stationary distribution $\pi^*$ such that $\pi^* = \lim_{k \rightarrow \infty} \pi_0^x H^k$.

**Proof.** We consider that the ten compartments $S_e, L_e, I_e, R_e, S_0, L_a, I_a, R_a, S_a$, and $I_a$ in Figure 4 represent the ten states of a
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 \( E \) 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 \( p_{ij} \) 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 \( (i, j) \in E^2 \), \( p_{ij} \) 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 \( x_{ij}, i \in \{1, \ldots, 23\} \) 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

\[
H = \begin{pmatrix}
S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \\
S_e & r_e & \beta_e & \beta_{e1} & \beta_{e2} & 0 & 0 & \rho_e & \beta_{e3} & \beta_{e4} & \beta_{e5} \\
L_e & 0 & q_e & \theta_e & \theta_{e1} & 0 & 0 & \theta_{e2} & \theta_{e3} & \theta_{e4} & \theta_{e5} \\
I_e & 0 & 0 & \nu_e & \nu_{e1} & 0 & 0 & \nu_{e2} & \nu_{e3} & \nu_{e4} & \nu_{e5} \\
R_e & 0 & 0 & 0 & \alpha_e & \alpha & 0 & 0 & \alpha & 0 & 0 \\
S_v & 0 & 0 & 0 & 0 & \alpha' & \alpha & 0 & 0 & 0 & 0 \\
I_v & \eta_{ve} & \eta_{ve1} & \eta_{ve2} & \eta_{ve3} & 0 & \rho_v & \eta_{va} & \eta_{va1} & \eta_{va2} & \eta_{va3} \\
S_a & 0 & 0 & 0 & 0 & 0 & t_1 & \rho_a & \beta_{a1} & \beta_{a2} \\
L_a & 0 & 0 & 0 & 0 & 0 & 0 & q_a & \theta_a & \theta_{a1} & \theta_{a2} \\
I_a & 0 & 0 & 0 & 0 & t_2 & 0 & 0 & 0 & \rho_a & \eta_{va} \\
R_a & 0 & 0 & 0 & 0 & 0 & 0 & \rho_a & \beta_a & \theta_a & r_a
\end{pmatrix}
\]
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 $(i_0, \cdots, i_{n-1}, i, j) \in \mathbb{E}^{n+2}$,

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

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:

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

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 non-immune. 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 ($S_v$) 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 $\mu$ 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 plasmodium 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 a homogeneous absorbing Markov chain with transition matrix $P$. 

![Figure 5: Transition graph of irreducible Markov chain.](image-url)
Proof. The graph representing the Markov chain model is given in Figure 7, where the \( x_i \), \( i \in \{1, \ldots, 26\} \) 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 \( A_1, \ldots, A_5 \) 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

\[
P = \begin{pmatrix}
S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a & D & E & M \\
S_e & r_e & \beta_e & \beta_{e1} & \beta_{e2} & 0 & 0 & \rho_e & \beta_{e3} & \beta_{e4} & \beta_{e5} & 0 & e_1 & m_1 \\
L_e & 0 & \varrho_e & \theta_e & \theta_{e1} & 0 & 0 & \theta_{e2} & \theta_{e3} & \theta_{e4} & \theta_{e5} & 0 & e_2 & m_2 \\
I_e & 0 & 0 & p_e & \nu_e & 0 & 0 & \nu_{e1} & \nu_{e2} & \nu_{e3} & \nu_{e4} & \mu & e_3 & m_3 \\
R_e & 0 & 0 & 0 & r_e & 0 & 0 & \rho_e & \rho_{e1} & \rho_{e2} & \rho_{e3} & 0 & e_4 & m_4 \\
S_v & 0 & 0 & 0 & 0 & \alpha' & \alpha & 0 & 0 & 0 & 0 & p' & 0 \\
I_v & \eta_{ve} & \eta_{ve1} & \eta_{ve2} & \eta_{ve3} & 0 & p_e & \eta_{va} & \eta_{va1} & \eta_{va2} & \eta_{ve3} & 0 & p & 0 \\
S_a & 0 & 0 & 0 & 0 & 0 & t_1 & r_a & \eta_a & \beta_{a1} & \beta_{a2} & 0 & 0 & 0 \\
L_a & 0 & 0 & 0 & 0 & 0 & 0 & q_a & \theta_a & \theta_{a1} & 0 & 0 & 0 \\
I_a & 0 & 0 & 0 & 0 & t_2 & 0 & 0 & p_a & \eta_{va} & 0 & 0 & 0 \\
R_a & 0 & 0 & 0 & 0 & 0 & \rho_a & \beta_a & \theta_a & r_a & 0 & 0 & p_2 \\
D & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 & 0 & 0 \\
E & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 \\
M & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 0 & 1 
\end{pmatrix}
\]
The matrix of transient states $Q$ and that of absorbing states $R$ are given by the relations

$$
Q = \begin{pmatrix}
S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \\
S_e & r_e & \beta_e & \beta_{e1} & \beta_{e2} & 0 & 0 & \rho_e & \beta_{e3} & \beta_{e4} & \beta_{e5} \\
L_e & 0 & q_e & \theta_e & \theta_{e1} & 0 & 0 & \theta_{e2} & \theta_{e3} & \theta_{e4} & \theta_{e5} \\
I_e & 0 & 0 & p_e & \nu_e & 0 & 0 & \nu_{e1} & \nu_{e2} & \nu_{e3} & \nu_{e4} \\
R_e & 0 & 0 & 0 & r_e & 0 & 0 & \rho_e & \rho_{e1} & \rho_{e2} & \rho_{e3} \\
S_v & 0 & 0 & 0 & 0 & \alpha' & \alpha & 0 & 0 & 0 & 0 \\
I_v & 0 & 0 & 0 & 0 & \eta_{ve} & \eta_{ve1} & \eta_{ve2} & \eta_{ve3} & \eta_{vav} & \eta_{vav1} & \eta_{vav2} & \eta_{vav3} \\
S_a & 0 & 0 & 0 & 0 & t_1 & r_a & \beta_a & \beta_{a1} & \beta_{a2} \\
L_a & 0 & 0 & 0 & 0 & 0 & 0 & q_a & \theta_a & \theta_{a1} \\
I_a & 0 & 0 & 0 & 0 & t_2 & 0 & 0 & 0 & p_a & \eta_{va} \\
R_a & 0 & 0 & 0 & 0 & 0 & 0 & \rho_a & \beta_a & \theta_a & r_a
\end{pmatrix}
$$

(23)

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

(24)
The fundamental matrix will be determined by application. \( \square \)

**Proposition 7.** The matrix elements \( n_{ij} = (I - Q)_{ij} \) 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

\[
N = \sum_{k=0}^{\infty} I_k.
\]

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

\[
I_k = \sum_{j \in E} I_{kj},
\]

\[
I_{kj} = 1_{\{X(k)=j\}},
\]

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

\[
n_i = \sum_{k=0}^{\infty} \sum_{j \in E} E_i I_k = \sum_{k=0}^{\infty} \sum_{j \in E} (Q)^k_{ij},
\]

By changing the order of summation, we obtain

\[
n_i = \sum_{j \in E} \left( \sum_{k=0}^{\infty} (Q)^k_{ij} \right) = \sum_{j \in E} (I - Q)_{ij} = \sum_{j \in E} n_{ij}.
\]

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

**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.

\( \eta_a \): 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 \([27, 30]\).

\( \eta_v \): we estimate that the probability of transmission of an infection from an infectious mosquito \( (I_v) \) to a susceptible nonimmune human being \( (S_v) \), 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 \([30]\).

\( \alpha_c \): we assume that the probability of transmission of infection from an infectious nonimmune human being \( (I_v) \) to a susceptible mosquito \( (S_e) \), 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 \([31]\).

\( \alpha_s \): we assume that the probability of transmission of infection from an infectious semi-immune human being \( (I_e) \) to a susceptible mosquito \( (S_e) \), 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.** \( \beta_v \): the probability of transition from the susceptible state \( (S_v) \) to the latent state \( (L_v) \). This parameter results from the infection force which is defined by

\[
\beta_v = \eta_v n_a i_v N_v / N_h,
\]

where \( N_v \) is the human population size and \( N_v \) is the mosquito population size \([28–30]\).

\( \theta_v \): the probability of transition from the latent state \( (L_v) \) to the infectious state \( (I_v) \). We assume that \( \theta_v = 0.10 \) for both types of zone.

\( \nu_v \): the probability of transition from the infectious state \( (I_v) \) to the immune state \( (R_v) \). 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.

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

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

\[
\beta_s = \eta_v n_a i_v N_v / N_h,
\]

where \( N_v \) is the size of the human population and \( N_v \) is the size of the mosquito population \([28–30]\).
\( \theta_a \): 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.

\( \nu_e \): 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.

\( \rho_e \): 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 \( \rho_e = 0.0083 \) for the low transmission zone and \( \rho_e = 0.033 \) 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.

### 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 \([0, 1]\).

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

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

\[
\pi_0 = (0.200, 0.150, 0.100, 0.120, 0.090, 0.110, 0.050, 0.080, 0.060, 0.040), \tag{32}
\]

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>
<thead>
<tr>
<th>Param.</th>
<th>Zone A</th>
<th>Zone B</th>
<th>Intervals</th>
</tr>
</thead>
<tbody>
<tr>
<td>( n_a )</td>
<td>0.25</td>
<td>0.38</td>
<td>0.13-0.47</td>
</tr>
<tr>
<td>( \eta_e )</td>
<td>0.021</td>
<td>0.07</td>
<td>0.01-0.27</td>
</tr>
<tr>
<td>( \eta_a )</td>
<td>0.012</td>
<td>0.022</td>
<td>0.01-0.27</td>
</tr>
<tr>
<td>( \alpha_e )</td>
<td>0.11</td>
<td>0.45</td>
<td>0.072-0.64</td>
</tr>
<tr>
<td>( \alpha_a )</td>
<td>0.08</td>
<td>0.35</td>
<td>0.072-0.64</td>
</tr>
<tr>
<td>( \theta_e )</td>
<td>0.10</td>
<td>0.10</td>
<td>0.067-0.20</td>
</tr>
<tr>
<td>( \theta_a )</td>
<td>0.09</td>
<td>0.09</td>
<td>0.067-0.20</td>
</tr>
<tr>
<td>( \nu_e )</td>
<td>0.005</td>
<td>0.001</td>
<td>0.0014-0.017</td>
</tr>
<tr>
<td>( \nu_a )</td>
<td>0.01</td>
<td>0.01</td>
<td>0.0014-0.017</td>
</tr>
<tr>
<td>( \rho_e )</td>
<td>( 5.5 \times 10^{-4} )</td>
<td>( 2.7 \times 10^{-3} )</td>
<td>( 1.1 \times 10^{-2} - 5.5 \times 10^{-5} )</td>
</tr>
</tbody>
</table>
episodes, using the initial distribution $\pi_0$ of the relation (32).

The stationary distribution obtained is $\pi^*$ and is given by the relation

$$
\pi^* = \{0.079, 0.029, 0.080, 0.073, 0.247, 0.107, 0.022, 0.142, 0.204, 0.043\}.
$$

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

<table>
<thead>
<tr>
<th>$S_e$</th>
<th>$I_e$</th>
<th>$R_e$</th>
<th>$S_v$</th>
<th>$I_v$</th>
<th>$S_a$</th>
<th>$I_a$</th>
<th>$R_a$</th>
</tr>
</thead>
<tbody>
<tr>
<td>0.167</td>
<td>0.118</td>
<td>0.118</td>
<td>0.087</td>
<td>0.085</td>
<td>0.044</td>
<td>0.042</td>
<td></td>
</tr>
<tr>
<td>0.138</td>
<td>0.093</td>
<td>0.117</td>
<td>0.098</td>
<td>0.070</td>
<td>0.039</td>
<td>0.044</td>
<td></td>
</tr>
<tr>
<td>0.114</td>
<td>0.127</td>
<td>0.116</td>
<td>0.116</td>
<td>0.063</td>
<td>0.036</td>
<td>0.112</td>
<td></td>
</tr>
<tr>
<td>0.095</td>
<td>0.125</td>
<td>0.115</td>
<td>0.137</td>
<td>0.062</td>
<td>0.033</td>
<td>0.122</td>
<td></td>
</tr>
<tr>
<td>0.081</td>
<td>0.119</td>
<td>0.113</td>
<td>0.158</td>
<td>0.065</td>
<td>0.030</td>
<td>0.129</td>
<td></td>
</tr>
<tr>
<td>0.072</td>
<td>0.110</td>
<td>0.110</td>
<td>0.177</td>
<td>0.070</td>
<td>0.028</td>
<td>0.133</td>
<td></td>
</tr>
<tr>
<td>0.067</td>
<td>0.102</td>
<td>0.106</td>
<td>0.193</td>
<td>0.076</td>
<td>0.027</td>
<td>0.135</td>
<td></td>
</tr>
<tr>
<td>0.064</td>
<td>0.094</td>
<td>0.102</td>
<td>0.207</td>
<td>0.082</td>
<td>0.026</td>
<td>0.136</td>
<td></td>
</tr>
<tr>
<td>0.063</td>
<td>0.088</td>
<td>0.099</td>
<td>0.218</td>
<td>0.087</td>
<td>0.025</td>
<td>0.136</td>
<td></td>
</tr>
<tr>
<td>0.064</td>
<td>0.083</td>
<td>0.099</td>
<td>0.226</td>
<td>0.092</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Table 6: The state of the probability of individuals after twenty-one episodes for an initial vector $\pi_0$ given by $\pi_0 = \{0.200, 0.150, 0.100, 0.120, 0.090, 0.110, 0.050, 0.080, 0.060, 0.040\}$.
Figure 8: Simulation of malaria host trajectories.

\[
\begin{pmatrix}
S_e & S_f & L_e & L_f & I_e & I_f & R_e & R_f \\
S_e & 0.129 & 0.030 & 0.100 & 0.010 & 0.000 & 0.000 & 0.011 \\
L_e & 0.000 & 0.225 & 0.100 & 0.010 & 0.000 & 0.000 & 0.015 \\
I_e & 0.000 & 0.000 & 0.107 & 0.100 & 0.000 & 0.000 & 0.012 \\
R_e & 0.000 & 0.000 & 0.000 & 0.349 & 0.000 & 0.000 & 0.011 \\
S_a & 0.000 & 0.000 & 0.000 & 0.000 & 0.310 & 0.190 & 0.000 \\
L_a & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.211 & 0.659 \\
I_a & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.090 \\
R_a & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.090 \\
D & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 1.000 \\
E & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 1.000 \\
M & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 0.000 & 1.000 \\
\end{pmatrix}
\]
The matrix of transient states \( Q \) and that of absorbing states \( R \) of the matrix \( P \) are given in the following the relations.

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

\[
R = \begin{pmatrix}
S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \\
0.000 & 0.350 & 0.240 & & & & & & & \\
0.000 & 0.240 & 0.260 & & & & & & & \\
0.041 & 0.250 & 0.350 & & & & & & & \\
0.000 & 0.260 & 0.250 & & & & & & & \\
0.000 & 0.500 & 0.000 & & & & & & & \\
0.000 & 0.500 & 0.000 & & & & & & & \\
0.000 & 0.000 & 0.000 & & & & & & & \\
0.000 & 0.000 & 0.000 & & & & & & & \\
0.000 & 0.000 & 0.500 & & & & & & & \\
\end{pmatrix}
\]

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

\[
D = \begin{pmatrix}
S_e & L_e & I_e & R_e & S_v & I_v & S_a & L_a & I_a & R_a \\
1.163 & 0.047 & 0.142 & 0.041 & 0.251 & 0.061 & 0.058 & 0.475 & 0.757 & 0.044 \\
0.019 & 1.294 & 0.156 & 0.045 & 0.307 & 0.077 & 0.080 & 0.898 & 0.926 & 0.056 \\
0.014 & 0.003 & 1.128 & 0.175 & 0.241 & 0.060 & 0.061 & 0.444 & 0.725 & 0.056 \\
0.016 & 0.003 & 0.010 & 1.539 & 0.281 & 0.068 & 0.064 & 0.519 & 0.846 & 0.047 \\
0.072 & 0.014 & 0.043 & 0.013 & 1.522 & 0.298 & 0.018 & 0.116 & 0.219 & 0.013 \\
0.261 & 0.052 & 0.156 & 0.045 & 0.264 & 1.084 & 0.067 & 0.421 & 0.795 & 0.047 \\
0.188 & 0.037 & 0.113 & 0.033 & 0.687 & 0.780 & 3.010 & 1.228 & 2.069 & 0.157 \\
0.066 & 0.013 & 0.040 & 0.012 & 1.348 & 0.275 & 0.067 & 10.185 & 4.063 & 0.223 \\
0.070 & 0.014 & 0.042 & 0.012 & 1.462 & 0.290 & 0.035 & 0.140 & 4.406 & 0.085 \\
0.033 & 0.007 & 0.020 & 0.006 & 0.324 & 0.138 & 0.351 & 0.595 & 0.977 & 1.467 \\
\end{pmatrix}
\]
Thus, for an individual in the state \( S_o \), the average number of months before absorption by \( D \) or \( M \) is \( 1.163 = 35 \) days in \( S_o \), \( 0.047 = 2 \) days in \( L_e \), \( 0.142 = 4 \) days in \( I_e \), \( 0.475 = 14 \) days in \( I_o \), and \( 0.757 = 1 \) day in \( I_n \). For infectious mosquitoes \( I_n \), the average number of months before absorption by \( E \) (eliminated by insecticides) or \( M \) (eliminated by LLINs) is \( 0.261 = 7 \) days in \( S_o \), \( 0.052 = 2 \) days in \( L_e \), and \( 0.156 = 5 \) days in \( I_e \). With absorbing Markov chains, all equilibrium distributions will be limited to state absortbents, here \( D \), \( E \), and \( M \). Also, we can determine the probabilities of absorption of states \( S_o \) to \( R_o \) by states \( D \), \( E \), and \( M \). To do this, let us consider the matrix \( B = NR \) defined by the relation

\[
B = \begin{pmatrix}
D & E & M \\
0.006 & 0.621 & 0.374 \\
0.006 & 0.560 & 0.435 \\
0.046 & 0.483 & 0.471 \\
0.000 & 0.584 & 0.417 \\
0.002 & 0.953 & 0.046 \\
0.006 & 0.829 & 0.166 \\
0.005 & 0.845 & 0.181 \\
0.002 & 0.851 & 0.148 \\
0.002 & 0.918 & 0.081 \\
0.001 & 0.251 & 0.752
\end{pmatrix}
\]

According to these results, the probability of an individual \( S_e, L_e, I_e, R_e, S_o, L_o, I_o \), and \( R_o \) 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 \( S_e \) (infectious \( I_e \), 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 \( S_o \) 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 \( (S_o, L_e, I_e, R_e, S_o, L_o, I_o, S_o) \) generalizes the host-vector compartment models of types \( (S_o, I_e, S_o) \), \( (S_o, R_e, S_o, S_o) \), and \( (S_o, I_e, R_e, S_o, S_o, S_o) \). 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


