RESEARCH ARTICLE

JENER Journal of Empirical and Non-Empirical Research
Volume 2, Issue 2, Page 205-213 | Article No. 27
DOI: 10.4898/jener.v2i2.a27
ISSN: 7895-9216
Journal homepage: https://jenerjournal.com
Journal email: editor@jenerjournal.com

JENER Journal
JENER Journal of Empirical and Non-Empirical Research

# Modeling the Spread of Leishmaniasis using Stochastic Differential Equation

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ARTICLE INFORMATION

**Article history:**
Published: February 2026

**Keywords:**
Leishmaniasis
Stochastic Differential quations
Epidemiological Modeling
Euler–Maruyama Method
Stochastic Runge–Kutta Method.

ABSTRACT

Leishmaniasis is a vector-borne infectious disease transmitted through the bite of infected female sandflies, posing significant public health challenges in many tropical and subtropical regions. In this study, we develop and analyze a stochastic differential equation (SDE) model for the transmission dynamics of Leishmaniasis to capture the inherent randomness and environmental fluctuations that influence disease spread. Starting from a deterministic compartmental model consisting of five interacting populations: susceptible humans, latent humans, infected humans, susceptible sandflies, and infected sandflies. We incorporate stochasticity by applying the procedure proposed by Allen et al. (2008). The resulting SDE was numerically solved and simulated using both the Euler–Maruyama and Stochastic Runge–Kutta methods using python to examine the dynamic behavior of the disease under uncertainty.

## 1. Introduction

Leishmaniasis is a vector borne diseases that consist of both the host and vector in its transmission and caused by the protozoan parasite Leishmania. This disease is one of the major tropical and subtropical diseases and transmitted by the bite of a sand fly specifically the female sand fly. Leishmaniasis has been classified as an endemic across the world affecting Asia, Africa, the Americas, and the Mediterranean region. Numerous environmental factors affect the distribution and dispersion of this disease (Desjeux 2004 and Shweta Khandibharad1 and Shailza Singh, 2023).

Understanding the geographical distribution and high-risk areas of diseases is a fundamental requirement for effective management, decision-making, and health system planning in any country. Today, Geographic Information Systems (GIS) serve as an important tool in health-related programs, allowing for cost-effective disease mapping, strategic planning, and analysis of factors influencing disease patterns. GIS also provides valuable data on meteorological and ecological conditions necessary for the survival of specific pathogens and their vectors. Consequently, the use of GIS can help predict seasonal variations in diseases by linking them to weather patterns and environmental conditions in different regions. (Yang G-J, Vounatsou P, and Xiao-Nong Z , 2005).

The life cycle of Leishmania consists of two distinct stages: one in the female sandfly and the other in the mammalian host (such as humans or dogs). When an infected sandfly takes a blood meal, it injects saliva that prevents clotting and simultaneously releases metacyclic promastigotes at the bite site. These promastigotes are slender, elongated, motile, and extracellular. Neutrophils are the first immune cells to arrive at the site, engulfing the promastigotes. However, because neutrophils are short-lived and undergo apoptosis, they are thought to act as “Trojan horses,” providing the parasites with a pathway to enter macrophages while evading immune activation. Inside macrophages, the promastigotes transform into amastigotes, which are small, round, and non-motile, and begin multiplying before spreading to nearby tissues. When another sand-fly feeds on an infected host, it ingests macrophages containing amastigotes. These cells rupture within the sand-fly’s midgut, releasing the parasites, which then develop further, migrate to the proboscis, and become ready to infect a new host. (Ty et al. 2019).

Traditionally, many mathematical models in disease epidemiology have relied on deterministic ordinary differential equations (ODEs), which do not capture the inherent uncertainties in disease transmission. Such uncertainties may arise from assumptions regarding disease parameters, population heterogeneity, behavioral dynamics, intervention strategies, external influences, and unforeseen events. In contrast, stochastic models explicitly incorporate the randomness associated with transmission processes, reflecting the probabilistic nature of events such as infection and recovery. This allows for a more realistic representation of variability and uncertainty in disease spread. While deterministic ODE models are suitable for large populations, stochastic models are particularly advantageous in describing dynamics within small populations or rare events (Ogwuche, 2023).

In this study, we address these uncertainties by introducing random perturbations modeled as a Wiener process, thereby transforming the deterministic ODE framework into a system of stochastic differential equations (SDEs). Specifically, we begin with the deterministic ODE model for Leishmaniasis and subsequently formulate its stochastic counterpart to better capture the dynamics of disease transmission.

## 2. Literature Review

Several works have been done on stochastic epidemic models. Ogwuche, Iortyer, Emonyi and Ali M. (2023) formulated an SDE model for the transmission of Tuberculosis (TB). In their work, a deterministic model for the transmission of TB was presented

and then transformed into a system of stochastic differential equation model. The Euler- Maruyama method was used for the simulation.

Tawfiqullah Ayoubi and Ahmad shahed (2024) investigated an algorithm of stochastic Runge-Kutta method for numerical solution of stochastic differential equations (SDEs). Because, most SDEs do not have analytical solution. Hence, numerical solution is required to estimate the numerical solution of SDEs. In addition, they applied this algorithm for numerical solution of Logistic Stochastic Differential Equation for cell concentration

Allen and Linda (2017) presented a work on stochastic epidemic models, emphasizing their formulation, numerical simulation, and analytical exploration. The study developed models based on continuous-time Markov chains and stochastic differential equations, using well-known examples to illustrate key concepts. Furthermore, the authors also discussed analytical techniques for approximating the probability of disease outbreaks, thereby providing valuable insights into the application of stochastic methods in epidemiology.

Yoshihiro Maki and Hideo Hirose (2013) proposed a SDE version of the SIR simulation model with application to SARS (Severe Acute Respiratory Syndrome) case in 2003 in Hong Kong.

Ogwuche, O. I. and Emonyi, T. A formulated a stochastic model for the transmission of Lassa fever. In their work, they attempted to demonstrate the impact of uncertainties in the mode of transmission of Lassa fever by subjecting the dynamics to some white noise modeled by the Brownian motion as a Wiener process. An existing deterministic model involving the Susceptible, Exposed, Infected and Recovered (SEIR) individuals were transformed into a stochastic differential equation model by applying the procedure proposed by Allen et al (2008).

## 3. Methodology

The model formulated is based on the following assumptions:

- Susceptible individuals has equal chances to be infected when contact with by the infectious individuals is established;
- Recovered individuals can be re-infected;
- Leishmaniasis induced death can occur in the infected class.

Based on the assumptions above, the following parameters were use as shown in Table 1.

### *3.1 Deterministic Model of Transmission of Leishmaniansis*

The dynamics for the transmission of leishmaniasis is illustrated by Figure 1.

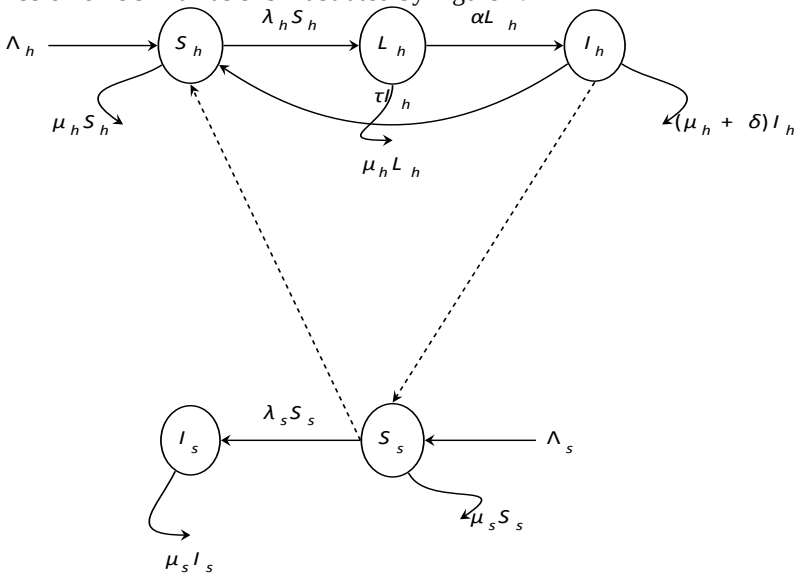


Figure 1: Schematic Diagram of the Leishmaniasis Model

### *3.2 Model Equations*

The assumptions in section 3.1 and the model flowchart together lead to the following system of ordinary differential equations which describe the transmission dynamics of the disease as:

| Parameter | Epidemiological interpretation |
|---|---|
| $S_h(t)$ | Susceptible human population at time t |
| $L_h(t)$ | Latent human population at time t |
| $I_h(t)$ | Infected human population at time t |
| $S_s(t)$ | Susceptible and – fly population at time t |
| $I_s(t)$ | Infected and – fly population at time t |
| $\lambda_h$ | Recruitment rate for human population |
| $\lambda_s$ | Recruitment rate for sand – fly population |

| | |
|---|---|
| $\tau$ | Disease recovery rate for human class |
| $\alpha$ | Movement rate from latent human class to infected human class |
| $\mu_h$ | Mortality rate for human population |
| $\mu_s$ | Mortality rate for sand – fly population |
| $\beta_1$ | Disease transmission rate for sand – fly population |
| $\beta_2$ | Disease transmission rate for human population |
| $\delta$ | Disease induced death rate for human class |

Table 1: Parameters of the model

$$\begin{cases} \frac{dS_h}{dt} = \Lambda_h - \lambda_h S_h - \mu_h S_h + \tau I_h \\ \frac{dL_h}{dt} = \lambda_h S_h - (\mu_h + \alpha) L_h, \\ \frac{dI_h}{dt} = \alpha L_h - (\mu_h + \delta + \tau) I_h, \\ \frac{dS_s}{dt} = \Lambda_s - \lambda_s S_s - \mu_s S_s, \\ \frac{dI_s}{dt} = \lambda_s S_s - \mu_s I_s, \end{cases} \quad \textbf{(1)}$$

subject to initial conditions

$S_h(0) > 0$, $L_h(0) \geq 0$, $I_h(0) \geq 0$, $S_s(0) > 0$, $I_s(0) \geq 0$,

where

$$\lambda_h = \frac{\beta_1 I_s}{N}, \quad \lambda_s = \frac{\beta_2 I_h}{N}.$$

The system (1) is assumed to have positive model parameters.

*3.3 Disease-free Equilibrium State*

Disease-free equilibrium points are steady-state solutions where there is no disease in the population. At equilibrium states the rate of change of the state varies with respect to time is Zero. That is the infected compartment of the model is equal to zero. Thus, $L_h = I_h = I_s = 0$. Then,

$$E_0 = (S_h, L_h, I_h, S_s, I_s) = (\tfrac{\Lambda_h}{\mu_h}, 0, 0, \tfrac{\Lambda_s}{\mu_s}, 0)_{\lambda}$$

*3.4 Basic Reproduction Number*

The basic reproduction number, denoted by $R_0$, represents the average number of secondary infections generated by a single infectious individual (e.g., a rodent or human) in a fully susceptible population.

If $R_0 = 1$, the disease is at a critical threshold where each case leads to one new case on average. This indicates that the infection is neither increasing nor decreasing in the population.

If $R_0 < 1$, it implies that an infected individual, on average, causes less than one new infection during their infectious period. In this case, the disease cannot sustain itself in the population and can potentially be eradicated.

Conversely, if $R_0 > 1$, it implies that each infected individual produces more than one new infection on average. As a result, the disease will spread within the population, making the disease-free equilibrium (DFE) unstable and invasion inevitable.

To compute $R_0$ for our model, we adopt the next-generation matrix method introduced by van den Driessche and Watmough (2002). The nextgeneration matrix $G$ is constructed using two components: $F$ and $V^{-1}$, defined as follows:

$$F = \left[\frac{\partial \mathcal{F}_i(X_0)}{\partial x_j}\right] \quad (2)$$

$$V = \left[\frac{\partial \mathcal{V}_i(X_0)}{\partial x_j}\right] \quad (3)$$

Where

$F_i$ =The new infections

$V_i$ =Transfers of infections from one compartment to another

$X_0$= The disease free equilibrium

$R_0$ = The dorminant eigenvalue of the matrix

$G = FV^{-1}$

The infection classes are $L_h$, $I_h$ and $I_s$. Hence,

$$F_i = \begin{bmatrix} \frac{\beta_1 I_s S_h}{N} \\ 0 \\ \frac{\beta_2 I_h S_s}{N} \end{bmatrix}$$

$$V_i = \begin{bmatrix} (\mu_h + \alpha)L_h \\ -\alpha L_h + (\mu_h + \delta + \tau)I_h \\ \mu_s I_s \end{bmatrix}$$

and using equation (2) and (3) we obtained:

$$F = \begin{bmatrix} 0 & 0 & \beta_1 \\ 0 & 0 & 0 \\ 0 & \frac{\beta_2 \lambda_s \mu_h}{\mu_s \lambda_h} & 0 \end{bmatrix}$$

and

$$V = \begin{bmatrix} \mu_h + \alpha & 0 & 0 \\ -\alpha & \tau + \mu_h + \delta & 0 \\ 0 & 0 & \mu_s \end{bmatrix}$$

Therefore,

$$V^{-1} = \begin{bmatrix} \frac{1}{\mu_h + \alpha} & 0 & 0 \\ \frac{\alpha}{(\mu_h + \alpha)(\tau + \mu_h + \delta)} & \frac{1}{\tau + \mu_h + \delta} & 0 \\ 0 & 0 & \frac{1}{\mu_s} \end{bmatrix}$$

The product of $FV^{-1}$ is

$$FV^{-1} = \begin{bmatrix} 0 & 0 & \frac{\beta_1}{\mu_s} \\ 0 & 0 & 0 \\ \frac{\beta_2 \lambda_s \mu_h \alpha}{\mu_s \lambda_h (\mu_h + \alpha)(\tau + \mu_h + \delta)} & \frac{\beta_2 \lambda_s \mu_h}{\mu_s \lambda_h (\tau + \mu_h + \delta)} & 0 \end{bmatrix}$$

The dominant eigenvalue of the matrix is

$$R_0 = \sqrt{\frac{\beta_1 \beta_2 \mu_s \lambda_s \alpha}{\lambda_h (\mu_h^2 + \mu_h \alpha + \mu_h \delta + \mu_h \tau + \alpha\delta + \alpha\tau)}}$$

*3.5 Formulation of the Stochastic Model for the Transmission of Leishmaniasis*

Using the first modeling procedure developed by Allen et al (2008) we derived the Stochastic model for the deterministic model of the Ordinary Differential Equation in (1).

The drift vector is defined as:

$$\vec{F} = \sum_{j=1} p_j \vec{\lambda}_j$$

where $\lambda$ and $p_j$ are the random changes and the transition probabilities are defined in the table below:

| Change | Probability | Event |
|---|---|---|
| $[10000]^T$ | $P_1 = \Lambda_h \Delta t$ | Birth of Susceptible human |
| $[-10000]^T$ | $P_2 = \mu_h S_h \Delta t$ | Death of Susceptible human |
| $[-11000]^T$ | $P_3 = \beta_1 I_s S_h \Delta t$ | Susceptible becomes Latent |
| $[0-1000]^T$ | $P_4 = \mu_h L_h \Delta t$ | Death of Latent human |
| $[0-1100]^T$ | $P_5 = \alpha L_h \Delta t$ | latent becomes infected |
| $[00-100]^T$ | $P_6 = (\mu_h + \delta) I_h \Delta t$ | Death of infected human |
| $[10-100]^T$ | $P_7 = \tau I_h \Delta t$ | Infected human becomes susceptible |
| $[00010]^T$ | $P_8 = \Lambda_s \Delta t$ | Birth of susceptible sand fly |
| $[000-10]^T$ | $P_9 = \mu_s S_s \Delta t$ | Death of susceptible sand fly |
| $[000-11]^T$ | $P_{10} = \beta_2 I_h S_s \Delta t$ | Susceptible sand fly becomes infected |
| $[0000-1]^T$ | $P_{11} = \mu_s I_s \Delta t$ | infected sand fly dies naturally |

$$\mathcal{F} = p_1\begin{bmatrix}1\\0\\0\\0\\0\end{bmatrix} + p_2\begin{bmatrix}-1\\0\\0\\0\\0\end{bmatrix} + p_3\begin{bmatrix}-1\\1\\0\\0\\0\end{bmatrix} + p_4\begin{bmatrix}0\\-1\\0\\0\\0\end{bmatrix} + p_5\begin{bmatrix}0\\-1\\1\\0\\0\end{bmatrix} + p_6\begin{bmatrix}0\\0\\-1\\0\\0\end{bmatrix} + p_7\begin{bmatrix}1\\0\\-1\\0\\0\end{bmatrix} + p_8\begin{bmatrix}0\\0\\0\\1\\0\end{bmatrix} + p_9\begin{bmatrix}0\\0\\0\\-1\\0\end{bmatrix} + p_{10}\begin{bmatrix}0\\0\\0\\-1\\1\end{bmatrix} + p_{11}\begin{bmatrix}0\\0\\0\\0\\-1\end{bmatrix}$$

$$\vec{F} = \begin{bmatrix}\Lambda_h\\0\\0\\0\\0\end{bmatrix} + \begin{bmatrix}-\mu_h S_h\\0\\0\\0\\0\end{bmatrix} + \begin{bmatrix}-\beta_1 I_s\\\beta_1 I_s\\0\\0\\0\end{bmatrix} + \begin{bmatrix}0\\-\mu_h L_h\\0\\0\\0\end{bmatrix} + \begin{bmatrix}0\\-\alpha L_h\\\alpha L_h\\0\\0\end{bmatrix} + \begin{bmatrix}0\\0\\-(\mu_h+\delta)I_h\\0\\0\end{bmatrix} + \begin{bmatrix}\tau I_h\\0\\-\tau I_h\\0\\0\end{bmatrix} + \begin{bmatrix}0\\0\\0\\\Lambda_s\\0\end{bmatrix} + \begin{bmatrix}0\\0\\0\\-\mu_s S\\0\end{bmatrix} + \begin{bmatrix}0\\0\\0\\-\beta_2 I_h\\\beta_2 I_h\end{bmatrix} + \begin{bmatrix}0\\0\\0\\0\\-\mu_s I_s\end{bmatrix}$$

$$\vec{F} = \begin{bmatrix}\Lambda_h - \mu_h S_h - \beta_1 I_s + \tau I_h\\\beta_1 I_s - \mu_h L_h - \alpha L_h\\\alpha L_h - (\mu_h+\delta)I_h - \tau I_h\\\Lambda_s - \mu_s S_s - \beta_2 I_h\\\beta_2 I_h - \mu_s I_s\end{bmatrix}$$

Given $\Lambda_h = \frac{\beta_1 I_s}{N}$ and $\Lambda_s = \frac{\beta_2 I_h}{N}$ , the drift vector $\vec{F}$ is:

$$\vec{F} = \begin{bmatrix}\tau I_h - \mu_h S_h\\\beta_1 I_s - \mu_h L_h - \alpha L_h\\\alpha L_h - (\mu_h+\delta)I_h - \tau I_h\\-\mu_s S_s\\\beta_2 I_h - \mu_s I_s\end{bmatrix}$$

Similarly, the covariance matrix which is the volatility coefficient is de- fined as:

$$V = \sum_{j=1} p_j \vec{\lambda}_j (\lambda_j)^T$$

$$V = p_1\begin{bmatrix}1\\0\\0\\0\\0\end{bmatrix}[1\,0\,0\,0\,\,0] + p_2\begin{bmatrix}-1\\0\\0\\0\\0\end{bmatrix}[-1\,0\,0\,0\,\,0] + p_3\begin{bmatrix}-1\\1\\0\\0\\0\end{bmatrix}[-1\,1\,0\,0\,\,0] + p_4\begin{bmatrix}0\\-1\\0\\0\\0\end{bmatrix}[0-1\,0\,0\,\,0] + p_5\begin{bmatrix}0\\-1\\1\\0\\0\end{bmatrix}[0-1\,1\,0\,\,0]$$
$$+ p_6\begin{bmatrix}0\\0\\-1\\0\\0\end{bmatrix}[0\,0-1\,0\,\,0] + p_7\begin{bmatrix}1\\0\\-1\\0\\0\end{bmatrix}[1\,0-1\,0\,\,0] + p_8\begin{bmatrix}0\\0\\0\\1\\0\end{bmatrix}[0\,0\,0\,1\,\,0] + p_9\begin{bmatrix}0\\0\\0\\-1\\0\end{bmatrix}[0\,0\,0-1\,\,0]$$
$$+ p_{10}\begin{bmatrix}0\\0\\0\\-1\\1\end{bmatrix}[0\,0\,0-1\,1] + p_{11}\begin{bmatrix}0\\0\\0\\0\\-1\end{bmatrix}[0\,0\,0\,0\,\,-1]$$

Multiplying the covariance matrix we have:

$$V = \begin{bmatrix}\tau I_h + 2\beta_1 I_s + \mu_h S_h & -\beta_1 I_s & -\tau I_h & 0 & 0\\-\beta_1 I_s & \alpha L_h + \beta_1 I_s + \mu_h L_h & -\alpha L_h & 0 & 0\\-\tau I_h & -\alpha L_h & \alpha L_h + (\mu_h+\delta)I_h + \tau I_h & 0 & 0\\0 & 0 & 0 & 2\beta_2 I_h + \mu_s S_s & -\beta_2 I_h\\0 & 0 & 0 & -\beta_2 I_h & \beta_2 I_h + \mu_s I_s\end{bmatrix}$$

The resulting stochastic differential equation is given by :

$$dX(t) = \vec{F}(t, X(t))dt + V(t, X(t)dW(t))$$
$$X(0) = [X_1(0), X_2(0), X_3(0), X_4(0), X_5(0)]^T$$

*3.6 Numerical Simulation*

The Euler-Maruyama and Stochastic Runge-Kutta methods for of SDEs were used for the simulation. These methods are numerical schemes used for approximating the solutions of stochastic differential equations (SDEs).

*3.6.1 Euler- Maruyama Method*

The Euler–Ito SDE-Maruyama method is used for the simulation. For

$$dX_t = a(t, X_t)dt + b(t, X_t)dW_t$$

the Euler–Maruyama method is given by:

$$Yn+1 = Yn + a(\tau n, Yn)(\tau n+1 - \tau n) + b(\tau n, Yn)(W\tau n+1 - W\tau n)$$

According to Kloeden and Platen (2007), one of the simplest time-discrete approximations of an Itoˆ process is the Euler approximation, or the Euler–Maruyama approximation as it is sometimes called.

Consider an Itoˆ–Maruyama process $X = \{X_t, t_0 \le t \le T\}$ satisfying the scalar stochastic differential equation:

$$dX_t = a(t, X_t)dt + b(t, X_t)dW_t$$

On $t_0 \le t \le T$, with the initial value $X_{t0} = X_0$, and for a given discretization

$$t_0 = \tau_0 < \tau_1 < \cdots < \tau_n < \cdots < \tau_N = T$$

of the time interval $[t_0, T]$, an Euler approximation is a continuous-time stochastic process

$$Y = \{Y(t), t_0 \le t \le T\}$$

Satisfying the iterative scheme:

$$Yn+1 = Yn + a(\tau n, Yn)(\tau n+1 - \tau n) + b(\tau n, Yn)(W\tau n+1 - W\tau n)$$

In the 1-dimensional case, $d = m = 1$, the Euler scheme has the form

$$Y_{n+1} = Y_n + a\Delta + b\Delta W$$

where

$$\Delta = \tau_{n+1} - \tau_n = I(0) = J(0)$$

is the length of the time discretization subinterval $[\tau_n, \tau_{n+1}]$, and

$$\Delta W = W\tau n+1 - W\tau n.$$

For $k = 1,\ldots,d$, the scheme can be written as

$$Y_{n+1}^k = Y_n^k + a^k\Delta + b^k\Delta W,$$

where the drift and diffusion coefficients are $d$-dimensional vectors

$$a = (a_1,\ldots,a_d), \qquad b = (b_1,\ldots,b_d).$$

For the general multi-dimensional case with $d,m = 1,2,\ldots$, the $k$-th component of the Euler scheme has the form

$$Y_{n+1}^k = Y_n^k + \alpha^k\Delta + \sum_{j=1}^{m} b_{kj}\Delta W_j$$

where

$$\Delta W_j = W_{\tau_{n+1}}^j - W_{\tau_n}^j = I(j) = J(j)$$

is the N(0,Δ)-distributed increment of the $j$-th component of the $m$-dimensional standard Wiener process $W$ on $[\tau_n, \tau_{n+1}]$, and $\Delta W_{j1}$ and $\Delta W_{j2}$ are independent for $j_1 \neq j_2$. The diffusion coefficient

$$b = [b_{k,j}]$$

is a $d \times m$ matrix.

*3.6.2 Stochastic Runge-Kutta Method*

Stochastic Runge-Kutta (SRK) method. This method was introduced by Rumellin (1982). SRK was developed based on the increment of Wiener process,

$$J_1(t) = \int_{t_n}^{t_{n+1}} dW(t)$$

The general form of SRK for numerical approximation SDEs is:

$$Y_t = Y_n + \Delta\sum_{i=1}^{s}\alpha_{i,j}\, f(Y_j^n) + J_1\sum_{i=1}^{s} b_{i,j}\, g(Y_j), \quad i = 1,2,\ldots s$$

$$Y_{n+1} = Y_n + \Delta\sum_{i=1}^{s}\alpha_{i,j} f(Y_i^n) + J_1\sum_{i=1}^{s}\gamma_i\, g(Y_j),$$

where $A = (a_{i,j})_{sxs}$ and $B = (b_{i,j})_{sxs}$ are matrices of real element,

$\alpha^T = (\alpha_1 \ldots \alpha_s)$ and $\gamma^T = (\gamma_1 \ldots \gamma_s)$ are row vectors.

**4. Numerical Implementation and Results**

The resulting model in equation (1) was simulated using the Euler-Maruyama and Stochastic Runge-kutta methods using the parameter and initial condition:

| Parameter | Value |
|---|---|
| $\tau$ | 0.2 |
| $\alpha$ | 0.1 |
| $\mu_h$ | 0.01 |
| $\mu_s$ | 0.02 |
| $\beta_1$ | 0.05 |
| $\beta_2$ | 0.04 |
| $\delta$ | 0.1 |

Table 3: Table of value for the parameters

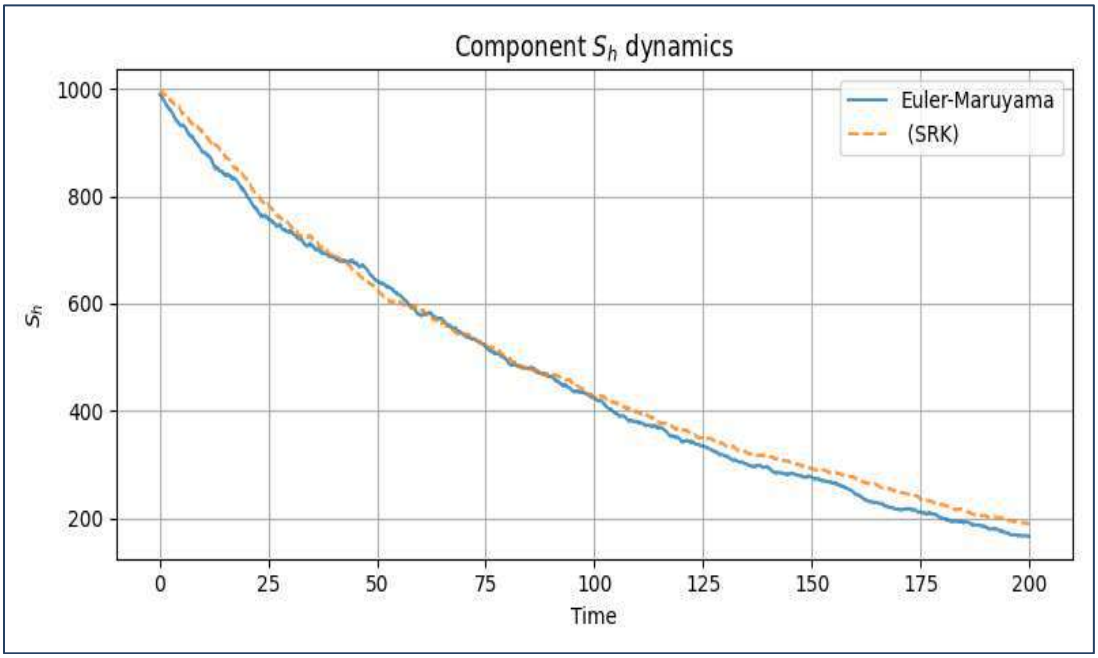


Figure 2: susceptible human

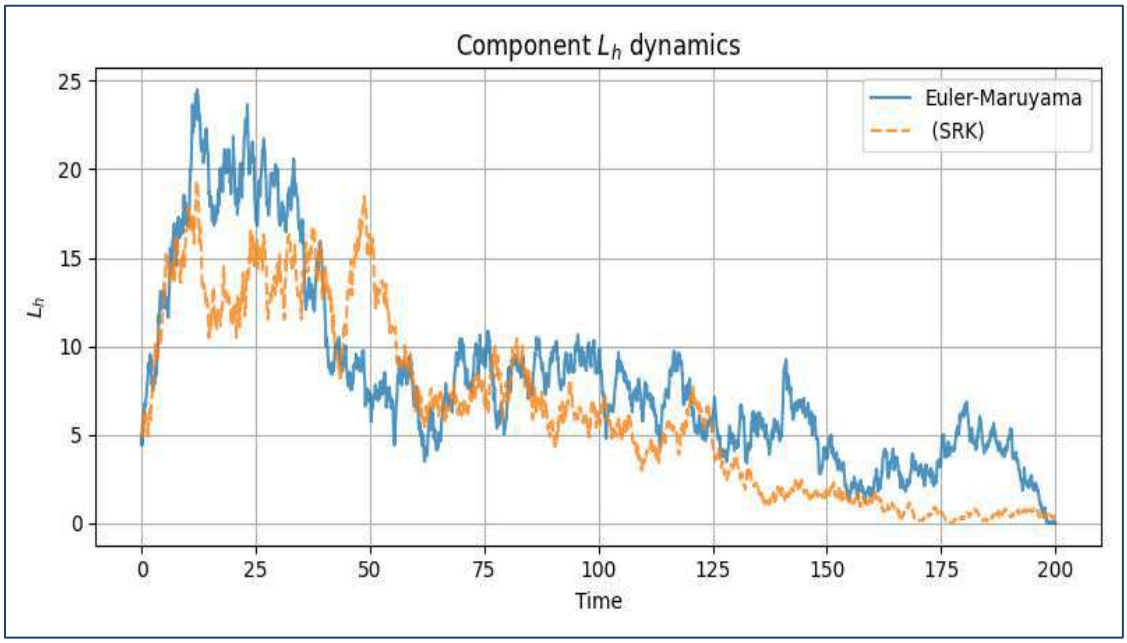


Figure 3: latent human

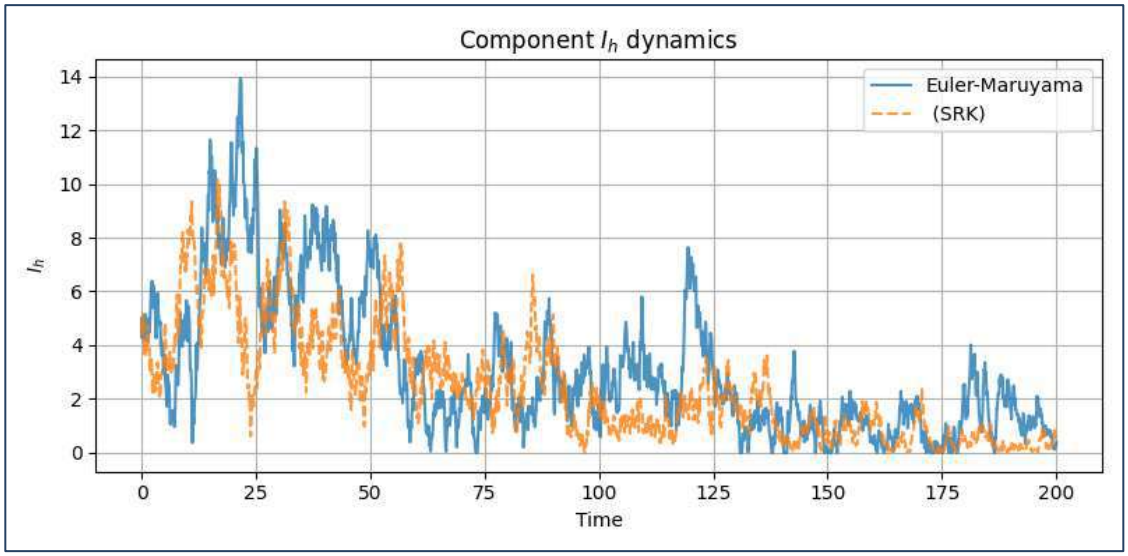


Figure 4: infected human

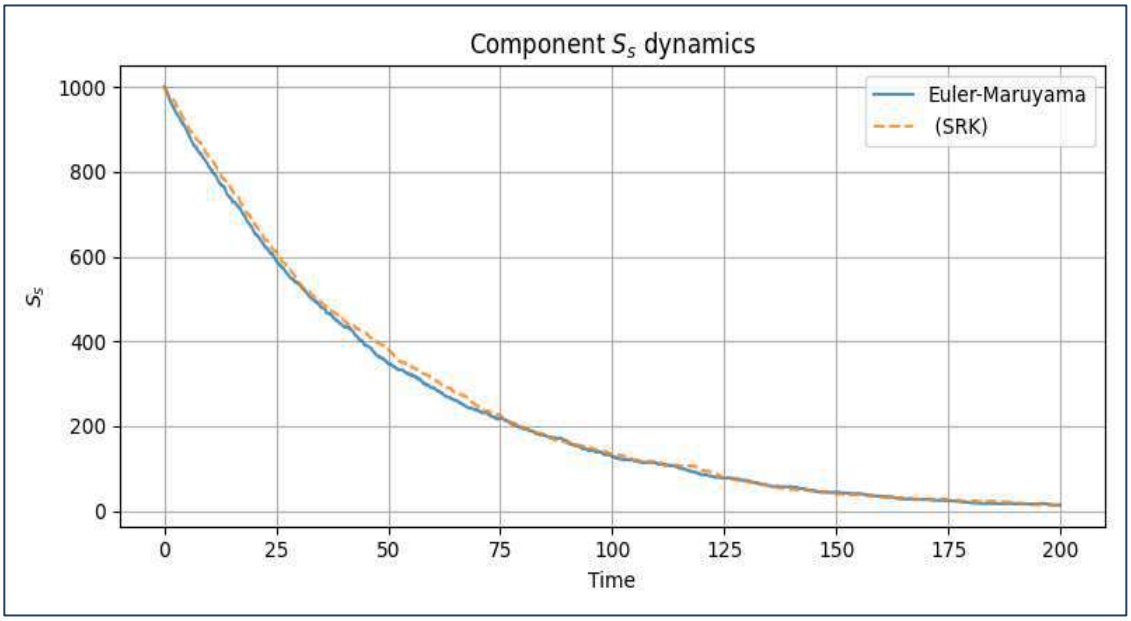


Figure 5: Susceptible sun-fly

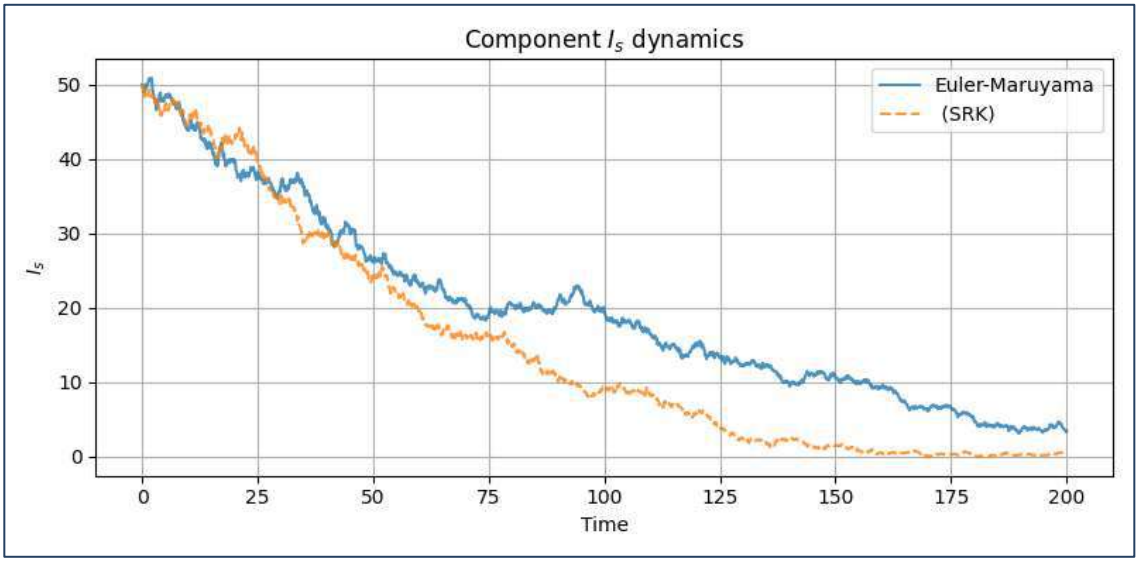


Figure 6: Infected sun-fly

**5. Conclusion and Recommendations**

The results demonstrate that while both methods can capture the basic epidemic dynamics, the Runge-Kutta method is significantly more accurate for moderate step sizes. Euler's method shows qualitative agreement but accumulates numerical error with larger step sizes. Runge-Kutta provides more stable and accurate solutions.

Numerical methods are essential tools for simulating complex nonlinear systems like the Leishmaniasis model. The choice of method affects the accuracy and stability of the solution. For most applications, the Runge-Kutta method provides a good balance between computational cost and precision.

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