Applied Mathematics & Information Sciences
An International Journal

http://dx.doi.org/10.18576/amis/190315

Exploring the Impact of Jump Perturbations on Stochastic SIRS Dynamics

*K. El Bakkioui, Y. El Khalfi, S. Boutouil, B. Harchaoui, M. El Idrissi**, *A. Settati, and A. Lahrouz*

Laboratory of Mathematics and Applications, FSTT, Abdelmalek Essaadi University, Tetouan, Morocco

Received: 12 Dec. 2024, Revised: 12 Jan. 2025, Accepted: 20 Feb. 2025
Published online: 1 May 2025

Abstract: This study develops a comprehensive stochastic framework to examine the impact of Lévy perturbations on the dynamics of the SIRS (Susceptible-Infectious-Recovered-Susceptible) model. Initially, we established the existence and uniqueness of the solution, ensuring a solid foundation for our analysis. We identified the critical conditions for disease persistence, which are essential for evaluating the applicability of the model in real-world scenarios. Additionally, we determined the criteria for disease extinction. To support our theoretical findings, we conducted extensive computer simulations.

Keywords: Stochastic SIRS model, Lévy jumps, Persistence, Extinction, Lyapunov Function, Threshold.

1 Introduction

During the early 20th century, epidemiology underwent a significant transformation, driven by the groundbreaking contributions of eminent scientists such as Anderson Gray McKendrick and Janet Leigh. Their pioneering work introduced the concept of mathematical modeling, which has since evolved into an indispensable tool in the field. This mathematical modeling has profoundly impacted the management of outbreaks and epidemics, playing a pivotal role in guiding evidence-based public health interventions. Epidemiology has evolved significantly, owing to notable physicians such as Quinto Tiberio Angelerio, who demonstrated remarkable proficiency in managing the plague outbreak in Alghero, Sardinia, in 1582. However, the actual emergence of modern epidemiology as a formal scientific discipline occurred during the 19th century. Often referred to as the ”father of modern epidemiology,” John Snow made a meaningful breakthrough when he meticulously traced a devastating cholera outbreak in London to water contamination from the Broad Street pump. This groundbreaking investigation is the pivotal moment that laid the cornerstone for contemporary epidemiology, shaping it into the scientific field we recognize today. Epidemiology is a scientific discipline investigating epidemics, diseases, and various health-related conditions, including those unrelated to diseases. Its roots can be traced to ancient Greece, notably through the influential work of Hippocrates of Kos, a renowned physician who made notable contributions by distinguishing between epidemic and endemic diseases. Epidemiology, in its broader scope, also encompasses the study of diseases affecting plants, domestic animals, and livestock. An epidemic is characterized by a significant and abnormal surge in the occurrence of a specific disease within a population, typically manifesting rapidly. Many factors influence the intricate disease transmission process [1–5], encompassing the infectious agent’s characteristics and the host population’s complex dynamics. Regarding the infectious agent, its inherent characteristics, such as its mode of transmission (e.g., respiratory droplets, direct contact), the duration of infectivity, and its responsiveness to medical interventions like treatments and vaccines, are crucial factors that determine its ability to spread among individuals. Equally important are the host population elements that influence the dynamics of an epidemic. Factors such as social interactions, demographics (e.g., age, gender), cultural practices, geographic distribution, and economic conditions are pivotal in determining a population’s susceptibility and resilience in the face of the disease. Across the pages of recorded history, human civilization has wrestled with recurrent epidemics and pandemics. These outbreaks of infectious diseases have inflicted significant human suffering, societal upheaval, and economic turbulence. Given the formidable nature of

* Corresponding author e-mail: mourad.elidrissi@etu.uae.ac.ma

these challenges, the precise prediction of outbreak progression becomes paramount to effectively mitigating their adverse impacts. Epidemiologic modeling is at the heart of this pursuit, a fundamental tool for comprehending the intricacies of disease transmission dynamics and formulating informed strategies for containment and prevention. Infectious diseases continue to exert substantial effects on communities worldwide, notwithstanding the era of modern medicine and advancements in medical science. These impacts extend to various facets, including public health, healthcare infrastructure, the economy, education, population dynamics, and international cooperation. The investigation of epidemic models holds considerable importance across multiple domains due to its numerous advantages. These benefits encompass enhanced comprehension and forecasting of epidemics, optimization of public health resource allocation, more effective planning of emergency responses, advancement of public health research, heightened public awareness, and the development of training programs in public health. This, in turn, facilitates informed public health decision-making, enhances epidemic readiness, and ultimately contributes to saving lives. This curious problem has attracted mathematicians since the 18th century. Kermack and McKendrick established the classical deterministic SIR model in 1927 [6], serving as a fundamental framework for analyzing epidemic dynamics within a closed population, categorizing individuals into three compartments: The SIR model, delineating individuals into susceptible S, infected I, and recovered R compartments, does not account for individuals who lose immunity post-recovery. To address this deficiency, an extended version known as the SIRS model has been introduced [7–12]. In the SIRS model, individuals who have previously recovered from an infection are susceptible to becoming infected again. Mathematically, this phenomenon is expressed as follows:

$$\begin{cases} \dfrac{dS_t}{dt} = [\rho(1-S_t) + \eta R_t - \alpha S_t I_t], \\ \dfrac{dI_t}{dt} = [\alpha S_t I_t - (\rho+\lambda) I_t], \\ \dfrac{dR_t}{dt} = [\lambda I_t - (\rho+\eta) R_t]. \end{cases} \tag{1.1}$$

In this context, the parameter ρ represents the rate of births and deaths in the population, α signifies the infection coefficient, λ represents the recovery rate of infected individuals, and η characterizes the rate at which recovered individuals lose their immunity, transitioning back to a susceptible state. Later, many studies used the stochastic approach to treat these models [13, 14]. Adding Lévy perturbations to the SIRS model can enrich epidemiological modeling by better capturing the complexity of real epidemic phenomena, including stochastic variations and rare events that significantly impact disease dynamics. This can provide valuable information for public health decision-making and epidemic management. The reader can refer to [15–19]. In this study, we examine a system modeled as a stochastic SIRS (Susceptible-Infectious-Recovered-Susceptible) model incorporating a jump perturbation:

$$\begin{cases} dS_t = [\rho(1-S_t) + \eta R_t - \alpha S_t I_t]\,dt \\ \qquad - \displaystyle\int_{\mathbb{D}} \varsigma_\upsilon S_{(t^-)} I_{(t^-)} \widetilde{\mathcal{N}}(dt, d\upsilon), \\ dI_t = [\alpha S_t I_t - (\rho+\lambda) I_t]\,dt \\ \qquad + \displaystyle\int_{\mathbb{D}} \varsigma_\upsilon S_{(t^-)} I_{(t^-)} \widetilde{\mathcal{N}}(dt, d\upsilon), \\ dR_t = [\lambda I_t - (\rho+\eta) R_t]\,dt, \end{cases} \tag{1.2}$$

where, dt is Lebesgue measure, and $\widetilde{\mathcal{N}}(dt, d\upsilon)$ is the compensated Poisson measure, such that

$$\widetilde{\mathcal{N}}(dt, d\upsilon) = \mathcal{N}(dt, d\upsilon) - \pi(d\upsilon)dt.$$

In this context, $\mathcal{N}(dt, d\upsilon)$ denotes a Poisson counting measure, while π represents a Lévy measure defined on $\mathbb{D} \subset \mathbb{R}^+$. Additionally, the continuously differentiable function $\varsigma_{(\cdot)}$ characterizes the impact of random jumps within the population. It is essential to ensure that $-1 < \varsigma_\upsilon < 1$ for every $\upsilon \in \mathbb{D}$. Additionally, $S_{(s^-)}$ and $I_{(s^-)}$ represent the left-hand limits of the functions S_s and I_s, respectively. Henceforth, we will refer to these limits as S_s and I_s for practicality. This work stems from the need to enhance traditional epidemiological models by incorporating Lévy perturbations, which account for sudden, random changes in disease dynamics. The research aims to understand better disease persistence and extinction by developing a more realistic stochastic SIRS model, leading to improved strategies for managing infectious diseases in unpredictable environments. The manuscript is organized systematically, beginning with Section 2, which discusses the positivity and existence of solutions for the system described in equation (1.2). In Section 3, we derive the necessary conditions for disease persistence. Section 4 delves into a detailed analysis of disease Extinction. Next, Section 5 presents a series of computer simulations to confirm the accuracy of the analytical results. Section 6 thoroughly analyzes the results and outlines potential directions. Finally, the paper concludes in Section 7 with a thorough discussion of our findings and possible avenues for future research.

2 Positivity and Existence

This section will explore the global existence and positivity within the SDE system (1.2).

Definition 1(Meyer Angle Bracket). *Let $(M_t)_{t\geq 0}$ be a continuous local martingale. The Meyer's Angle-Bracket, or the predictable quadratic variation of M, denoted by*

$\langle M\rangle_t$, is a unique increasing predictable process such that $M_t^2-\langle M\rangle_t$ is a local martingale.

Lemma 1.*Let M_t be a local martingale that starts from zero at time 0. For $t\geq 0$, we define*

$$\varphi_{M_t}:=\int_0^t(1+s)^{-2}d\langle M\rangle_s.$$

Here, $\langle M\rangle_t$ represents the Meyer angle bracket process. Then we have,

$$\mathbb{P}\left(\lim_{t\to\infty}\frac{M_t}{t}=0\right)=1,$$

if we establish

$$\mathbb{P}\left(\lim_{t\to\infty}\varphi_{M_t}<\infty\right)=1.$$

*Proof.*See for example [20].

First, we aim to ensure both the existence and uniqueness of the solution to our model within the set Δ, defined as follows:

$$\Delta=\left\{(x_1,x_2,x_3)\in(0,1)^3;\,x_1+x_2+x_3=1\right\},$$

and $(\alpha,\eta,\lambda,\rho)\in(0,1)^4$. Thus, we rely on the following theorem:

Theorem 1.*Let $\upsilon\in\mathbb{D}$, $(S,I)\in(0,1)^2$, and we define*

$$\Psi(\upsilon,S,I)=[1-\varsigma_\upsilon I]\,[1+\varsigma_\upsilon S],$$

if

$$\sup_{0<S,I<1}\int_{\mathbb{D}}\ln\left[\Psi^{-1}(\upsilon,S,I)\right]\pi(d\upsilon)<\infty. \tag{2.1}$$

So for each initial value $(S_0,I_0,R_0)\in\Delta$, we have unique solution $(S_t,I_t,R_t)\in\Delta$ for equation (1.2).

*Proof.*Define $N=S+I+R$ the total population, and we identify the values taken (the numbers) by S, I, R and N with their frequencies, and by the equation (1.2),

$$dN_t=dS_t+dI_t+dR_t=-\rho(N_t-1)dt.$$

By integration for any $s\in[0,t]$, we obtain

$$N_s-1=[N_0-1]\exp(-\rho s)\quad a.s.,$$

from which for all $s\in[0,t]$,

$$(S_s,I_s,R_s)\in(0,1)^3\text{ and }N_s=1\ \ a.s.. \tag{2.2}$$

On the other hand, since the coefficients of the equations are locally Lipschitz continuous (see, e.g., [10, 21–25] for more information), it follows that there exists a unique local maximum solution (S_t,I_t,R_t) for $t\in[0,\tau_e)$, where τ_e denotes the timing of the explosion. Let $\varepsilon,\varepsilon_0>0$, for $\varepsilon\leq\varepsilon_0$, we define the stopping time:

$$\tau_\varepsilon=\inf\{t\in[0,\tau_e),\,(S_t\wedge I_t\wedge R_t)\leq\varepsilon\}. \tag{2.3}$$

Define for all $(S,I,R)\in\Delta$,

$$\Sigma_s=\Sigma_s(S_s,I_s,R_s)=-\ln(S_sI_sR_s).$$

By the Itô formula for every $t\geq 0$ and $s\in[0,t\wedge\tau_\varepsilon]$, we obtain

$$\begin{aligned}d\Sigma_s=&\left[3\rho+\lambda+\eta-\frac{\rho}{S_s}+\alpha I_s-\frac{\eta R_s}{S_s}-\alpha S_s-\frac{\lambda I_s}{R_s}\right]ds\\&-\int_{\mathbb{D}}\left\{\ln\left[(1+\varsigma_\upsilon S_s)(1-\varsigma_\upsilon I_s)\right]\right.\\&\left.+(I_s-S_s)\varsigma_\upsilon\right\}\pi(d\upsilon)ds\\&-\int_{\mathbb{D}}\ln\left[(1+\varsigma_\upsilon S_s)(1-\varsigma_\upsilon I_s)\right]\widetilde{\mathcal{N}}(ds,d\upsilon).\end{aligned}$$

Using equations (2.1) and (2.2), one obtains

$$\begin{aligned}d\Sigma_s\leq&\left[3\rho+\lambda+\eta+\alpha+\pi(\mathbb{D})\right.\\&\left.+\sup_{0<S,I<1}\int_{\mathbb{D}}\ln\left[\Psi^{-1}(\upsilon,S,I)\right]\pi(d\upsilon)\right]ds\\&-\int_{\mathbb{D}}\ln\left[(1+\varsigma_\upsilon S_s)(1-\varsigma_\upsilon I_s)\right]\widetilde{\mathcal{N}}(ds,d\upsilon).\end{aligned} \tag{2.4}$$

If we integrate (2.4) and the mathematical expectation property, this yields for each $t\geq 0$,

$$\begin{aligned}\mathbb{E}\left[\Sigma_t\left(S_{t\wedge\tau_\varepsilon},I_{t\wedge\tau_\varepsilon},R_{t\wedge\tau_\varepsilon}\right)\right]&\leq\Sigma_t\left(S_0,I_0,R_0\right)+\kappa t,\\&\leq\kappa t-3\ln(\varepsilon_0),\end{aligned} \tag{2.5}$$

where

$$\begin{aligned}\kappa=&3\rho+\lambda+\eta+\alpha+\pi(\mathbb{D})\\&+\sup_{0<S,I<1}\int_{\mathbb{D}}\ln\left[\Psi^{-1}(\upsilon,S,I)\right]\pi(d\upsilon).\end{aligned}$$

Suppose $\tau_e<\infty$, so there is $t>0$, where $\mathbb{P}(\{\tau_e<t\})>0$, implying $\mathbb{P}(\{\tau_\varepsilon<t\})>0$. For $\omega\in\{\tau_e<t\}$ and equation (2.3), one obtains

$$-\ln(\varepsilon)\leq\Sigma_t\left[S_{t\wedge\tau_\varepsilon}(\omega),I_{t\wedge\tau_\varepsilon}(\omega)R_{t\wedge\tau_\varepsilon}(\omega)\right].$$

Hence

$$\begin{aligned}-\ln(\varepsilon)\mathbb{P}(\{\tau_\varepsilon\leq t\})&\leq\mathbb{E}\left[\Sigma_t\left(S_{\tau_\varepsilon},I_{\tau_\varepsilon},R_{\tau_\varepsilon}\right)\mathbb{I}_{\{\tau_\varepsilon\leq t\}}\right],\\&\leq\mathbb{E}\left[\Sigma_t\left(S_{t\wedge\tau_\varepsilon},I_{t\wedge\tau_\varepsilon},R_{t\wedge\tau_\varepsilon}\right)\right].\end{aligned} \tag{2.6}$$

By using $\tau_\varepsilon\leq\tau_e$, (2.5) and (2.6), we get

$$\begin{aligned}\mathbb{P}(\{\tau_e\leq t\})&\leq\mathbb{P}(\{\tau_\varepsilon\leq t\}),\\&\leq\frac{3}{\ln(\varepsilon)}\left[\ln(\varepsilon_0)-\kappa t\right].\end{aligned}$$

Letting $\varepsilon\to 0$, obtaining the contradiction $\mathbb{P}(\{\tau_e\leq t\})=0$, we conclude that $\tau_e=\infty$.

In the following section, we will examine the concept of disease persistence, focusing on determining the critical threshold conditions required for effective disease control and long-term management. This analysis will provide vital insights into the factors influencing the sustained presence of the disease within a population.

3 Persistence

It is essential in epidemiological studies of the disease to focus on cases where it persists and does not disappear spontaneously, as these cases can provide valuable information on its long-term impact and management. In what follows, we will demonstrate the persistence of the disease [26–28]. Let us define

$$\begin{cases} H(S) = -(\rho+\lambda)+\alpha S-\left[\frac{1}{4}\int_{\mathbb{D}}\varsigma_\upsilon^2\pi(d\upsilon)\right]S^2, \\ \mathscr{T}^1 = \alpha\left[\rho+\lambda+\frac{1}{4}\int_{\mathbb{D}}\varsigma_\upsilon^2\pi(d\upsilon)\right]^{-1}, \\ \Pi(S) = -(\rho+\lambda)+\alpha S-\left[\frac{1}{2}\int_{\mathbb{D}}\varsigma_\upsilon^2\pi(d\upsilon)\right]S^2, \\ \mathscr{T}^2 = \alpha\left[\rho+\lambda+\frac{1}{2}\int_{\mathbb{D}}\varsigma_\upsilon^2\pi(d\upsilon)\right]^{-1}. \end{cases} \tag{3.1}$$

Theorem 2. *Let* (2.1) *and*

$$\sup_{0<y<1}\int_{\mathbb{D}}\ln^2[1+\varsigma_\upsilon y]\,\pi(d\upsilon)<\infty, \tag{3.2}$$

hold. For $(S_0,I_0,R_0)\in\Delta$, *if* $\mathscr{T}^1>1$, $\mathscr{T}^2>1$ *and* $|\varsigma_\upsilon|<1$ *for each* $\upsilon\in\mathbb{D}$, *then*

$(i)\limsup_{t\to\infty} S_t \geq \rho$, *a.s.*,
$(ii)\liminf_{t\to\infty} I_t \leq (\rho+\eta)(\rho+\eta+\lambda)^{-1}(1-\rho)$, *a.s.*,
$(iii)\liminf_{t\to\infty} R_t \leq \lambda(\rho+\eta+\lambda)^{-1}(1-\rho)$, *a.s.*,
$(iv)\liminf_{t\to\infty} S_t \leq \rho'$, *a.s.*,
$(v)\limsup_{t\to\infty} I_t \geq (\rho+\eta)(\rho+\eta+\lambda)^{-1}(1-\rho')$, *a.s.*,
$(vi)\limsup_{t\to\infty} R_t \geq \lambda(\rho+\eta+\lambda)^{-1}(1-\rho')$, *a.s.*,

where, ρ *and* ρ' *denote the positive roots on the interval* $(0,1)$ *of equations* $H(S)=0$ *and* $\Pi(S)=0$, *respectively.*

Remark. Since $-1<\varsigma_v<1$ for all $v\in\mathbb{D}$, as a result, for all $S\in(0,1)$, $\Pi(S)<H(S)$, therefore, $\rho<\rho'$.

Proof. (i) From (1.2), and using Itô formula, one obtains

$$\begin{aligned}\ln(I_t) = \ln(I_0) &- \int_0^t[(\rho+\lambda)-\alpha S_s]ds \\ &+\int_{\mathbb{D}}\int_0^t\left[\ln(1+\varsigma_\upsilon S_s)-\varsigma_\upsilon S_s\right]\pi(d\upsilon) \\ &+\int_{\mathbb{D}}\int_0^t\ln\left[1+\varsigma_\upsilon S_s\right]\widetilde{\mathscr{N}}(ds,d\upsilon).\end{aligned} \tag{3.3}$$

Applying the following inequality:

$$\ln(1+x)-x<-\frac{x^2}{4},\quad -1<x\leq 1. \tag{3.4}$$

Hence

$$\begin{aligned}\ln(I_t) \leq \ln(I_0)+\int_0^t H(S_s)ds \\ +\int_{\mathbb{D}}\int_0^t\ln[1+\varsigma_\upsilon S_s]\widetilde{\mathscr{N}}(ds,d\upsilon).\end{aligned} \tag{3.5}$$

As $H(0)=-(\rho+\lambda)<0$, when $\mathscr{T}^1>1$ it follows that $H(1)>0$. Consequently, the equation $H(S)=0$ possesses a unique root $\rho\in(0,1)$, because if $\mathscr{T}^1>1$, we will have $H'(S_0)=0$ for a $S_0>1$. Therefore, H exhibits a monotonic increase on the interval $(0,1)$ in particular $(0,\rho)$. For any sufficiently small $\varepsilon>0$, when $0<S\leq\rho-\varepsilon$, we get

$$H(S)\leq H(\rho-\varepsilon)<0. \tag{3.6}$$

Now, we begin the proof of assertion (i). Assume the contrary, implying that there exists a sufficiently small strictly positive ε, where

$$\mathbb{P}\left[\limsup_{t\to\infty} S_t\leq\rho-2\varepsilon\right]>0.$$

Consider

$$\Omega_1=\left\{\limsup_{t\to\infty} S_t\leq\rho-2\varepsilon\right\}.$$

For every $\omega\in\Omega_1$, there exists $\tau(\omega)>0$, such that

$$S_t\leq\rho-\varepsilon<1,\ \text{ for each }\ t\geq\tau(\omega). \tag{3.7}$$

By (3.6) and (3.7), we get for each $s\geq\tau(\omega)$,

$$H(S_s)\leq H(\rho-\varepsilon)<0. \tag{3.8}$$

By equation (3.2) and Lemma (1), we can easily prove the existence of a set $\Omega_2\subset\Omega$, with $\mathbb{P}(\Omega_2)=1$, where for each $\omega\in\Omega_2$, one has

$$\lim_{t\to\infty}\int_{\mathbb{D}}\int_0^t\frac{\ln[1+\varsigma_\upsilon S_s]}{t}\widetilde{\mathscr{N}}(ds,d\upsilon)=0. \tag{3.9}$$

Now, fix any $\omega\in\Omega_1\cap\Omega_2$. Hence, by (3.5) and (3.8), for $t\geq\tau(\omega)$, we obtain

$$\begin{aligned}&\ln(I_t)-\ln(I_0) \\ &\leq\int_0^{\tau(\omega)}H(S_s)ds+\int_{\tau(\omega)}^t H(\rho-\varepsilon)ds \\ &\quad+\int_{\mathbb{D}}\int_0^t\ln[1+\varsigma_\upsilon S_s]\widetilde{\mathscr{N}}(ds,d\upsilon).\end{aligned} \tag{3.10}$$

Based on (3.9) and (3.10), we deduce

$$\begin{aligned}\limsup_{t\to\infty}\frac{\ln(I_t)}{t}&\leq H(\rho-\varepsilon), \\ &<0.\end{aligned} \tag{3.11}$$

Whence,

$$\lim_{t\to\infty} I_t=0. \tag{3.12}$$

By integrating the last equation of (1.2), one obtains

$$\begin{aligned}R_t &= R_0\exp\{-(\rho+\eta)t\} \\ &\quad+\lambda\int_0^t I_{(t-s)}\exp\{-(\rho+\eta)s\}ds.\end{aligned} \tag{3.13}$$

Using (3.13) and the Fatou lemma, we deduce

$$\limsup_{t\to\infty} R_t \leq \lambda(\rho+\eta)^{-1}\limsup_{t\to\infty} I_t. \tag{3.14}$$

Furthermore, in conjunction with (3.12), this implies $\lim_{t\to\infty} R_t = 0$ and thus $\lim_{t\to\infty} S_t = 1$. However, this contradicts (3.7).

(iv) In the same way, based on Itô formula as in (3.3) and applying

$$-\frac{x^2}{2} \leq \ln(1+x)-x, \quad \text{for each } x \geq 0.$$

Thus

$$\ln(I_t) \geq \ln(I_0) + \int_0^t \Pi(S_s)ds + \int_{\mathbb{D}}\int_0^t \ln\left[1+\varsigma_\upsilon S_s\right]\widetilde{\mathcal{N}}(ds,d\upsilon). \tag{3.15}$$

Suppose (iv) is contradicted, implying the existence of a sufficiently small $\varepsilon' > 0$ with $\mathbb{P}(\Omega_3) > 0$, where

$$\Omega_3 = \left\{\liminf_{t\to\infty} S_t \geq \rho' + 2\varepsilon'\right\}.$$

So, for each $\omega \in \Omega_3$, there exist $\tau'(\omega) > 0$ such that

$$S_t \geq \rho' + \varepsilon', \quad \text{for each } t \geq \tau'(\omega). \tag{3.16}$$

Similar (3.8), it is straightforward to confirm, by selecting $\varepsilon' > 0$ adequately small, that

$$\Pi(S_s) \geq \Pi(\rho'+\varepsilon') > 0, \quad \text{for } s \geq \tau'(\omega). \tag{3.17}$$

Using (3.9), (3.15), (3.17), and following a similar reasoning as in (3.10), we obtain

$$\limsup_{t\to\infty} \frac{\ln(I_t)}{t} \geq H(\rho'+\varepsilon') > 0.$$

Thus,

$$\lim_{t\to\infty} I_t = \infty.$$

This contradicts the condition $I < 1$.

(ii) By (i) and (2.2), one has

$$\liminf_{t\to\infty} I_t + \liminf_{t\to\infty} R_t \leq 1-\rho, \quad a.s.. \tag{3.18}$$

Using (3.13) and Fatou lemma, yields

$$\liminf_{t\to\infty} I_t \leq \lambda^{-1}(\rho+\eta)\liminf_{t\to\infty} R_t. \tag{3.19}$$

By combining equations (3.18) and (3.19), we establish the necessary assertion (ii).

(v) Similar to (ii), the conclusion follows from equations (3.14), (iv), and (2.2).

(iii)-(vi) These outcomes stem directly from equations (2.2), (i), (iv), (ii), and (v).

In the next section, we will analyze the extinction of the stochastic differential equation (SDE) (1.2) to determine the critical threshold necessary for achieving disease control or complete eradication.

4 Extinction

This section will examine the extinction phenomenon in the SDE system (1.2).

Theorem 3. *Let* $(S_0, I_0, R_0) \in \Delta$ *and* (2.1) *holds. Assume that*

$$\sup_{0<y<1}\int_{\mathbb{D}} \ln^2\left[1+\varsigma_\upsilon y\right]\pi(d\upsilon) < \infty. \tag{4.1}$$

We define the new threshold

$$\mathscr{T}^3 = \alpha\left[\rho + \frac{1}{4}\int_{\mathbb{D}} \varsigma_\upsilon^2 \pi(d\upsilon)\right]^{-1}, \tag{4.2}$$

and

$$\mathscr{T}^4 = \frac{1}{2}\int_{\mathbb{D}} \varsigma_\upsilon^2 \pi(d\upsilon). \tag{4.3}$$

If $\mathscr{T}^3 < 1$ *and* $\alpha \geq \mathscr{T}^4$*, then the system governed by* (1.2) *exhibits extinction with an exponential decay rate.*

Proof. Let

$$\Sigma_t(Z_t) = \ln(Z_t),$$

where, $Z_t = I_t + R_t$. Using the Itô formula, one obtains

$$\begin{aligned} d\Sigma_t &= \frac{1}{Z_t}\left[-\rho I_t - (\rho+\eta)R_t + \alpha S_t I_t\right]dt \\ &\quad + \int_{\mathbb{D}}\left[\ln\left(1+\varsigma_\upsilon\frac{S_tI_t}{Z_t}\right) - \varsigma_\upsilon\frac{S_tI_t}{Z_t}\right]\pi(d\upsilon)dt \\ &\quad + \int_{\mathbb{D}}\ln\left(1+\varsigma_\upsilon\frac{S_tI_t}{Z_t}\right)\widetilde{\mathcal{N}}(dt,d\upsilon). \end{aligned} \tag{4.4}$$

Using (3.4) and the following inequalities:

$$-1 < \varsigma_\upsilon < 1, \quad \frac{SI}{Z} \leq 1, \tag{4.5}$$

and

$$\frac{1}{Z}\left[-\rho I - (\rho+\eta)R\right] \leq -\rho.$$

Thus

$$\begin{aligned} d\Sigma_t &\leq \left[-\rho + \alpha\frac{S_tI_t}{Z_t} - \frac{1}{4}\int_{\mathbb{D}}\varsigma_\upsilon^2\pi(d\upsilon)\left(\frac{S_tI_t}{Z_t}\right)^2\right]dt \\ &\quad + \int_{\mathbb{D}}\ln\left(1+\varsigma_\upsilon\frac{S_tI_t}{Z_t}\right)\widetilde{\mathcal{N}}(dt,d\upsilon), \\ &\leq \sup_{0<\delta\leq 1}\Phi(\delta)dt + \int_{\mathbb{D}}\ln\left(1+\varsigma_\upsilon\frac{S_tI_t}{Z_t}\right)\widetilde{\mathcal{N}}(dt,d\upsilon), \end{aligned} \tag{4.6}$$

where

$$\Phi(\delta) = -\rho + \alpha\delta - \frac{1}{4}\int_{\mathbb{D}}\varsigma_\upsilon^2\pi(d\upsilon)\delta^2.$$

If $\alpha \geq \frac{1}{2}\int_{\mathbb{D}}\varsigma_\upsilon^2\pi(d\upsilon)$, then Φ is increasing on $(0,1)$. By integration, we get

$$\Sigma_t \leq \Sigma_0 + \int_0^t\left(-\rho+\alpha-\frac{1}{4}\int_{\mathbb{D}}\varsigma_\upsilon^2\pi(d\upsilon)\right)ds + M_t, \tag{4.7}$$

where, M_t is a real-valued local martingale such that

$$M_t = \int_{\mathbb{D}} \int_0^t \ln\left(1+\varsigma_\upsilon \frac{S_s I_s}{Z_s}\right) \widetilde{\mathcal{N}}(ds, d\upsilon).$$

Or

$$\langle M \rangle_t = \int_{\mathbb{D}} \int_0^t \ln^2\left(1+\varsigma_\upsilon \frac{S_s I_s}{Z_s}\right) \pi(d\upsilon) ds,$$
$$\leq \left[\sup_{0<y<1} \int_{\mathbb{D}} \ln^2(1+\varsigma_\upsilon y)\pi(d\upsilon)\right] t < \infty.$$

From (4.1) and (1), we get

$$\limsup_{t\to\infty} \frac{M_t}{t} = 0 \ \ a.s.. \tag{4.8}$$

The assertion is validated with (4.7) and (4.8).

Next, we will examine the SDE (1.2) computer simulations to validate and reinforce our theoretical findings.

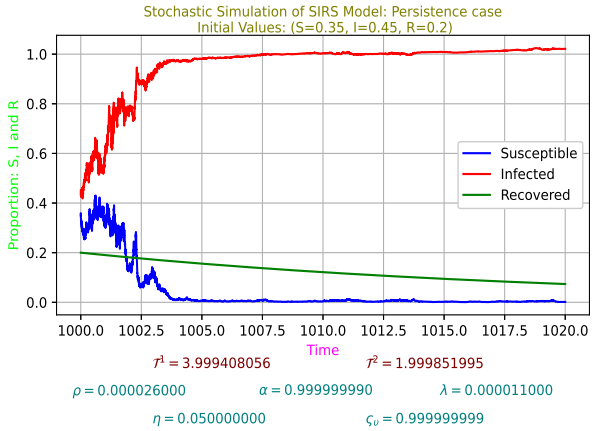

fig. 1. Trajectories of $S(t)$, $I(t)$, and $R(t)$ for system 1.2 with respect to Theorem 2.

5 Computer Simulations

In this section, we propose a few examples of numerical computer simulations to illustrate the theoretical results of Theorem 2 for persistence and Theorem 3, which prove the extinction of the disease by using the Maruyama Euler method (see, e.g., [29] for further information and the references cited therein).

5.1 Graphical representations in case of persistence theorem 2

The graphical representation plots the proportions of susceptible, infected, and recovered individuals over time. Each case specified in the cases list corresponds to a set of parameter values defining the transmission rate ρ, infection rate α, recovery rate λ, reintroduction rate η, and volatility parameter ς. For each case, the model is simulated, and the results are plotted on separate graphs, with the time on the **x-axis** and the proportion of individuals on the **y-axis**. The plots provide insights into how different parameter combinations affect the disease dynamics, allowing for comparative analysis.

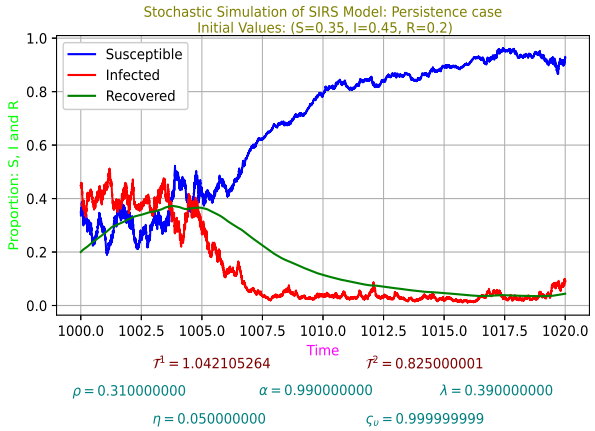

fig. 2. Trajectories of $S(t)$, $I(t)$, and $R(t)$ for system 1.2 with respect to Theorem 2.

5.2 Analysis, comparison and interpretation in case of persistence theorem 2

The variations in the dynamics of susceptible, infected, and recovered populations are analyzed concerning the values of $\mathcal{T}^1$ and $\mathcal{T}^2$, particularly when $\mathcal{T}^1 > 1$ and $\mathcal{T}^2 > 1$, see figures 1 and 4, as well as in the case of the negation of these conditions, see figures 2 and 3. The parameters $\mathcal{T}^1$ and $\mathcal{T}^2$ are derived from the model's equations and represent critical thresholds related to transmission, infection, and recovery rates. By comparing the simulations under different parameter conditions, insights are gained into how parameter changes impact the disease spread, including the emergence of endemic or epidemic behaviors.

5.3 Graphical representations in case of theorem extinction 3

The graphical representation plots the proportions of susceptible, infectious, and recovered individuals over time for each simulated case. Each case corresponds to a different set of parameter values defining transmission

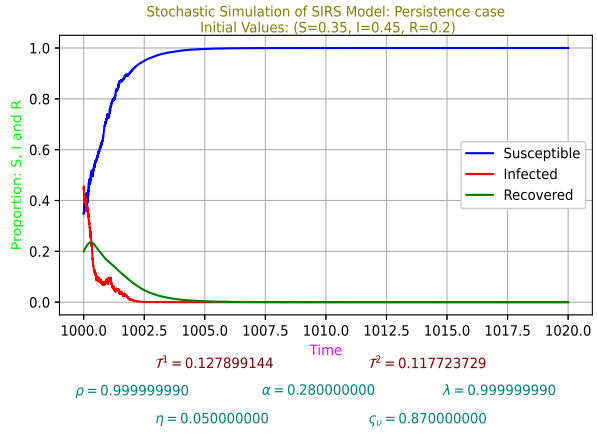

fig. 3. Trajectories of $S(t)$, $I(t)$, and $R(t)$ for system 1.2 with respect to Theorem 2.

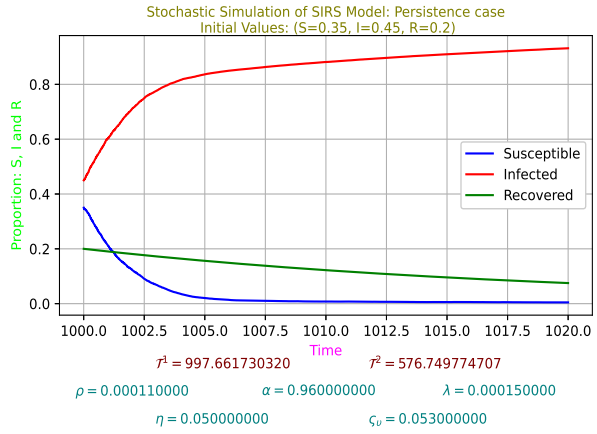

fig. 4. Trajectories of $S(t)$, $I(t)$, and $R(t)$ for system 1.2 with respect to Theorem 2.

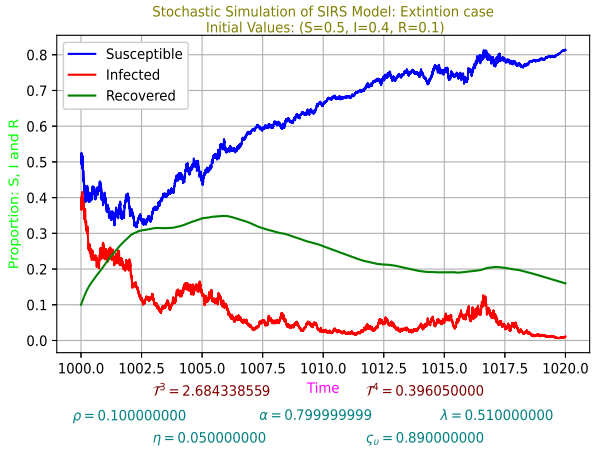

fig. 5. Trajectories of $S(t)$, $I(t)$, and $R(t)$ for system 1.2 with respect to Theorem 3.

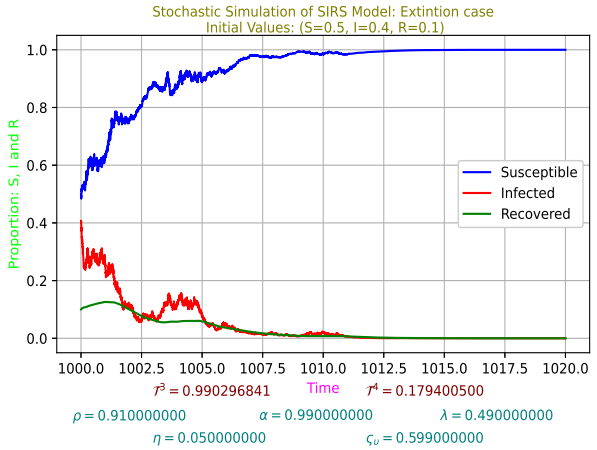

fig. 6. Trajectories of $S(t)$, $I(t)$, and $R(t)$ for system 1.2 with respect to Theorem 3.

rates ρ, infection α, recovery λ, reintroduction into the susceptible population η, and volatility ς. The plots provide visual insights into how changes in these parameters affect the dynamics of the disease spread, allowing for comparative analysis across different scenarios.

5.4 Analysis, comparison, and interpretation in case of theorem extinction 3

The variations in the susceptible, infectious, and recovered populations are analyzed concerning the values of $\mathcal{T}^3$ and $\mathcal{T}^4$, particularly when $\mathcal{T}^3 < 1$ and $\mathcal{T}^4 \leq \alpha$, see figures 6, as well as in the case of the negation of these conditions, see figures 5, 7, and 8. The parameters $\mathcal{T}^3$ and $\mathcal{T}^4$ are derived from the model's equations and represent critical thresholds related to the rates of transmission and infection. By comparing the simulations under different parameter conditions, insights are gained into how changes in these parameters impact the disease dynamics, including the emergence of endemic or epidemic behaviors.

6 Perspective

In recent years, the study of stochastic dynamics in epidemiology has gained significant traction, particularly

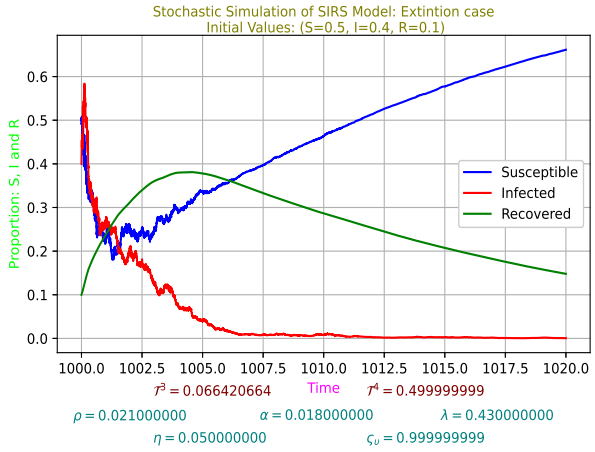

fig. 7. Trajectories of $S(t)$, $I(t)$, and $R(t)$ for system 1.2 with respect to Theorem 3.

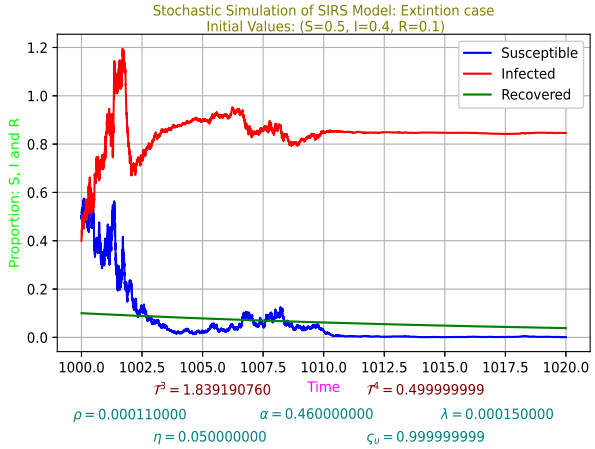

fig. 8. Trajectories of $S(t)$, $I(t)$, and $R(t)$ for system 1.2 with respect to Theorem 3.

in understanding the complexities of infectious disease transmission [38–62]. One such model, the stochastic model (SIRS), provides a valuable framework for investigating the dynamics of infectious diseases in populations where individuals move between susceptible, infectious, and recovered states. However, traditional SIRS models often assume continuous transitions between these states, overlooking the potential impact of sudden, large-scale perturbations or ”jumps.” Exploring the effects of jump perturbations on stochastic SIRS dynamics presents a novel avenue for understanding the behavior of infectious diseases in real-world scenarios. Unlike gradual transitions, jump perturbations can arise from various factors, such as sudden changes in environmental conditions, mass gatherings, or interventions like vaccination campaigns or policy changes. These perturbations can significantly alter disease transmission dynamics, leading to non-intuitive outcomes and challenging traditional modeling assumptions. From a scientific perspective, investigating jump perturbations in stochastic SIRS dynamics offers insights into the resilience and vulnerability of populations to abrupt changes in disease dynamics. Researchers can capture the inherent uncertainty and variability observed in real-world epidemiological data by incorporating stochasticity and jump processes into SIRS models. This allows for more accurate predictions of disease spread, improved assessment of intervention strategies, and a better understanding of the underlying mechanisms driving disease emergence and persistence. Professionally, this research has far-reaching implications for public health policy, infectious disease surveillance, and epidemic preparedness. Understanding how jump perturbations influence stochastic SIRS dynamics can inform the development of more effective disease control measures and response strategies. By identifying critical thresholds, tipping points, and high-risk scenarios, policymakers and public health officials can proactively mitigate the impact of sudden perturbations and minimize the risk of epidemic outbreaks or resurgence. In conclusion, exploring the impact of jump perturbations on stochastic SIRS dynamics represents a valuable interdisciplinary endeavor at the intersection of epidemiology, mathematics, and complex systems science [30–37]. Through rigorous scientific inquiry and collaboration, researchers can advance our understanding of infectious disease dynamics, enhance predictive capabilities, and ultimately contribute to more resilient and adaptive public health systems.

7 Conclusion

In conclusion, the stochastic SIRS model with jump perturbation provides a valuable framework for exploring and understanding the dynamics of infectious diseases with abrupt changes in transmission rates. This model offers insights into the potential impact of sporadic events or interventions on disease spread within a population. Its mathematical rigor and stochastic nature make it a valuable tool for researchers and policymakers in assessing and devising disease control and prevention strategies in dynamic and uncertain environments. Further research and refinement of this model may continue to contribute to our understanding of epidemiological processes and the development of effective public health measures.

Acknowledgement

The authors declare that they have no known conflicting financial interests or personal relationships that could have impacted the work presented in this article.

References

[1] X. Zhang, K. Wang, Stochastic SIR model with jumps. Applied Mathematics Letters, 26, 8, 867-874, (2013).
[2] D. Applebaum, M. Siakalli, Asymptotic stability of stochastic differential equations driven by Lévy noise, J. Appl. Probab, 46, (2009).
[3] J. Bao, C. Yuan, Stochastic population dynamics driven by Lévy noise, J. Math. Anal. Appl, 391, 363–375, (2012).
[4] A. Settati, A. Lahrouz, A. Assadouq, M. El Fatini, M. El Jarroudi, K. Wang, The impact of nonlinear relapse and reinfection to derive a stochastic threshold for SIRI epidemic model, Chaos, Solitons & Fractals, 137, 109897, (2020).
[5] A. Lahrouz, A. Settati, M. El Fatini, A. Tridane, The effect of a generalized nonlinear incidence rate on the stochastic SIS epidemic model, Mathematical Methods in the Applied Sciences, 44(1), 1137-1146, (2021).
[6] W. O. Kermack, A.G. McKendrick, Contribution to Mathematical Theory of Epidemics, P. Roy. Soc. Lond. A Mat, 115, 700-721, (1927).
[7] H. W. Hethcote, Qualitative analyses of communicable disease models, Math. Biosci, 28, 335-356, (1976).
[8] A. Settati, A. Lahrouz, M. El Fatini, A. El Haitami, M. El Jarroudi, M. Erriani, A Markovian switching diffusion for an SIS model incorporating Lévy processes, Discrete and Continuous Dynamical Systems-B, 28 (1), 209–229, (2023).
[9] X. Zhang, C. H. E. N. Fu, W. A. N. G. Ke, D. U. Hong, Stochastic SIRS model driven by Lévy noise, Acta Mathematica Scientia, 36, 3, 740-752, (2016).
[10] S. Boutouil, B. Harchaoui, A. Settati, A. Lahrouz, A. Nait, M. El Jarroudi, M. Erriani, Analyzing Stochastic SIRS Dynamics Under Jump Perturbation. International Journal of Applied and Computational Mathematics, 10, 1, 3, (2024).
[11] M. El Idrissi, B. Harchaoui, A. N. Brahim, I. Bouzalmat, A. Settati, A. Lahrouz, A sufficient condition for extinction and stability of a stochastic SIS model with random perturbation, WSEAS Transactions on Systems, 21, 367-371, (2022).
[12] A. N. Brahim, B. Harchaoui, S. Boutouil, M. El Idrissi, S. Aznague, A. Settati, A. Lahrouz, M. EL Jarroudi, Investigating Stochastic Dynamics of the Gilpin-Ayala Model in Dispersed Polluted Environments, WSEAS Transactions on Mathematics, 22, 607-620, (2023).
[13] Q. Lu, Stability of SIRS system with random perturbations, Physica A, 388, 3677-3686, (2009).
[14] S. Aznague, M. El Idrissi, A. N. Brahim, B. Harchaoui, S. Boutouil, A. Settati, A. Lahrouz, M. El Merzguioui, J. El Amrani, A Probabilistic SIRI Epidemic Model Incorporating Incidence Capping and Logistic Population Expansion, Appl. Math. Inf. Sci, 17, 5, 773–789, (2023).
[15] I. Bouzalmat, M. El Idrissi, A. Settati, A. Lahrouz, Stochastic SIRS epidemic model with perturbation on immunity decay rate, Journal of Applied Mathematics and Computing, 1-26, (2023).
[16] A. Settati, A. Lahrouz, M. Zahri, A. Tridane, M. El Fatini, H. El Mahjour, M. Seaid, A stochastic threshold to predict extinction and persistence of an epidemic SIRS system with a general incidence rate, Chaos, Solitons & Fractals, 144, 110690, (2021).
[17] B. Harchaoui, M. El Idrissi, A. El Haitami, A. Nait Brahim, A. Settati, A. Lahrouz, M. El Jarroudi, M. Er-Riani, T. Amtout, Examining the Relationship Between Infection Power Rate and the Critical Threshold in Stochastic SIS Epidemic Modeling, WSEAS Transactions on Biology and Biomedicine, 2224-2902, 20, 8, 73-79, (2023).
[18] T. Caraballo, A. Settati, A. Lahrouz, S. Boutouil, B. Harchaoui, On the stochastic threshold of the COVID-19 epidemic model incorporating jump perturbations, Chaos, Solitons & Fractals, 180, 114521, (2024).
[19] T. Caraballo, A. Settati, M. E. Fatini et al., Global stability and positive recurrence of a stochastic SIS model with Lévy noise perturbation, Physica A, 523 677–690, (2019).
[20] R. Lipster, A strong law of large numbers for local martingales, Stochastics, 3, 217-228, (1980).
[21] D. Zhou, X. Shi, X. Zhou, Dynamic Analysis of a Stochastic Delayed SEIRS Epidemic Model with Lévy Jumps and the Impact of Public Health Education, Axioms, 12(6), 560, (2023).
[22] I. Ali, S. U. Khan, Analysis of stochastic delayed SIRS model with exponential birth and saturated incidence rate. Chaos, Solitons & Fractals, 138, 110008, (2020).
[23] M. El Idrissi, A. Settati, A. Lahrouz, A. Assadouq, M. El Jarroudi, M. Er-Riani, M. Boujoudar, Stochastic Modeling of Nonlinear SIS Epidemic Dynamics with Jump Perturbations, New Mathematics and Natural Computation, 26, 1793-0057, (2024).
[24] S. Aznague, B. Harchaoui, A. N. Brahim, A. Settati, A. Lahrouz, T. Amtout, M. El Idrissi, A Stochastic SIR Epidemic with Non-Linear Power Functions and Logistic Growth, New Mathematics and Natural Computation, 26, 1793-0057, (2024).
[25] Adel Settati, Aadil Lahrouz, Mourad el Idrissi, An Introduction to Stochastic Integration and Itô Calculus, LAP LAMBERT Academic Publishing, Czialor, 978-620-7-65302-7, 88, (2024).
[26] A. Settati, A. Lahrouz, Stability and ergodicity of a stochastic Gilpin–Ayala model under regime switching on patches, International Journal of Biomathematics, 10(06), 1750090, (2017).
[27] A. Assadouq, A. Settati, A. El Myr, Qualitative Analysis of a SIR Epidemic Model with a Nonlinear Relapse and Incidence Rate Stochastically Perturbed, Appl. Math, 13(6), 999-1007, (2019).
[28] T. Caraballo, I. Bouzalmat, A. Settati, A. Lahrouz, A. N. Brahim, B. Harchaoui, Stochastic COVID-19 epidemic model incorporating asymptomatic and isolated compartments, Mathematical Methods in the Applied Sciences, (2024).
[29] P. E. Kloeden, E. Platen, P. E. Kloeden, E. Platen, Stochastic differential equations, Springer Berlin Heidelberg, 103-160, (1992).
[30] A. El Haitami, A. Settati, A. Lahrouz, M. El Idrissi, M. El Merzguioui, A stochastic switched SIRI epidemic model integrating nonlinear relapse phenomena, International Journal of Dynamics and Control, 12(5), 1287-1301, (2024).

[31] A. Settati, S. Hamdoune, A. Imlahi, A. Akharif, Extinction and persistence of a stochastic Gilpin–Ayala model under regime switching on patches, Applied Mathematics Letters, 90, 110-117, (2019).
[32] B. Harchaoui, S. Boutouil, A. Settati, A. Lahrouz, M. El Idrissi, M. El Jarroudi, Stochastic SIRI epidemic model with global incidence rate and relapse, International Journal of Applied and Computational Mathematics, 10(6), 179, (2024).
[33] A. Lahrouz, A. Settati, H. El Mahjour, M. El Jarroudi, M. El Fatini, Global dynamics of an epidemic model with incomplete recovery in a complex network, Journal of the Franklin Institute, 357(7), 4414-4436, (2020).
[34] A. Settati, A. Lahrouz, M. El Jarroudi, M. El Jarroudi, Dynamics of hybrid switching diffusions SIRS model, Journal of Applied Mathematics and Computing, 52, 101-123, (2016).
[35] A. Assadouq, H. El Mahjour, A. Settati, Qualitative behavior of a SIRS epidemic model with vaccination on heterogeneous networks, Ital. J. Pure Appl. Math, 43, 958-974, (2020).
[36] A. Assadouq, A. Settati, A. El Myr, Qualitative Analysis of a SIR Epidemic Model with a Nonlinear Relapse and Incidence Rate Stochastically Perturbed, Appl. Math, 13(6), 999-1007, (2019).
[37] A. Settati, Gaussian approximation of the empirical process under random entropy conditions, Stochastic processes and their Applications, 119(5), 1541-1560, (2009).
[38] Adel Settati, Mohamed Boujoudar, Mourad el Idrissi, Fundamentals and Principles of Differential Calculus, LAP LAMBERT Academic Publishing, 978-620-7-65412-3, 81, (2024).
[39] A. Settati, A. Lahrouz, Stationary distribution of stochastic population systems under regime switching, Applied Mathematics and Computation, 244, 235-243, (2014).
[40] A. Lahrouz, A. Settati, Necessary and sufficient condition for extinction and persistence of SIRS system with random perturbation, Applied Mathematics and Computation, 233, 10-19, (2014).
[41] M. El Fatini, A. Lahrouz, R. Pettersson, A. Settati, R. Taki, Stochastic stability and instability of an epidemic model with relapse, Applied Mathematics and computation, 316, 326-341, (2018).
[42] A. Lahrouz, A. Settati, Asymptotic properties of switching diffusion epidemic model with varying population size, Applied Mathematics and Computation, 219(24), 11134-11148, (2013).
[43] B. Berrhazi, M. El Fatini, A. Lahrouz, A. Settati, R. Taki, A stochastic SIRS epidemic model with a general awareness-induced incidence, Physica A: Statistical Mechanics and its Applications, 512, 968-980, (2018).
[44] A. Lahrouz, A. Settati, A. Akharif, Effects of stochastic perturbation on the SIS epidemic system, Journal of mathematical biology, 74, 469-498, (2017).
[45] A. Settati, A. Lahrouz, On stochastic Gilpin–Ayala population model with Markovian switching, Biosystems, 130, 17-27, (2015).
[46] A. Lahrouz, A. Settati, P. S. Mandal, Dynamics of a switching diffusion modified Leslie–Gower predator–prey system with Beddington–DeAngelis functional response, Nonlinear Dynamics, 85, 853-870, (2016).
[47] A. Lahrouz, A. Settati, Qualitative study of a nonlinear stochastic SIRS epidemic system, Stochastic Analysis and Applications, 32(6), 992-1008, (2014).
[48] A. Lahrouz, L. Omari, A. Settati, A. Belmaâti, Comparison of deterministic and stochastic SIRS epidemic model with saturating incidence and immigration, Arabian Journal of Mathematics, 4(2), 101-116, (2015).
[49] A. Settati, A. Lahrouz, M. El Jarroudi, M. El Fatini, K. Wang, On the threshold dynamics of the stochastic SIRS epidemic model using adequate stopping times, Discrete & Continuous Dynamical Systems, Series B, 25, (2020).
[50] M. El Fatini, M. El Khalifi, A. Lahrouz, R. Pettersson, A. Settati, The effect of stochasticity with respect to reinfection and nonlinear transition states for some diseases with relapse, Mathematical Methods in the Applied Sciences, 43(18), 10659-10670, (2020).
[51] J. El Amrani, T. Amtout, M. Er-Riani, A. Lahrouz, A. Settati, On the positive self-similar solutions of the boundary-layer wedge flow problem of a power-law fluid, Journal of Engineering Mathematics, 148(1), 9, (2024).
[52] A. Settati, A. Lahrouz, Stability and ergodicity of a stochastic Gilpin–Ayala model under regime switching on patches, International Journal of Biomathematics, 10(06), 1750090, (2017).
[53] A. El Myr, A. Assadouq, L. Omari, A. Settati, A. Lahrouz, A stochastic SIR epidemic system with a nonlinear relapse. Discrete Dynamics in Nature and Society, 2018(1), 5493270, (2018).
[54] B. Harchaoui, M. Esseroukh, B. El Khatib, A. Settati, A. Lahrouz, S. Boutouil, M. Erriani, Impact of Polluted Environments on Stochastic Gilpin–Ayala Population Dynamics with Dispersal, Appl. Math, 18(6), 1179-1188, (2024).
[55] M. El Jarroudi, A. Lahrouz, A. Settati, Asymptotic behavior of a random oscillating nonlinear reactive transport in thin turbulent layers, Mathematical Methods in the Applied Sciences, 44(18), 14849-14873, (2021).
[56] A. Settati, A. Lahrouz, M. El Idrissi, Probability Analysis of Selected Population Models, LAP LAMBERT Academic Publishing, 978-6208222802, 100, (2024).
[57] B. E. Berrhazi, M. El Fatini, A. Laaribi, R. Pettersson, R. Taki, A stochastic SIRS epidemic model incorporating media coverage and driven by Lévy noise. Chaos, Solitons & Fractals, 105, 60-68, (2017).
[58] A. Settati, M. El Idrissi, A. Lahrouz, Foundations and Applications of Statistical Theory, LAP LAMBERT Academic Publishing, (2025).
[59] A. Settati, M. El Idrissi, A. Lahrouz, Applied Probability: Concepts and Applications of Random Variables, LAP LAMBERT Academic Publishing, (2025).
[60] M. El Idrissi, Mhamed El Merzguioui, A. Settati, Theory of Distributions, LAP LAMBERT Academic Publishing, 978-6208065997, 120, (2024).
[61] A. Lahrouz, T. Caraballo, I. Bouzalmat, A. Settati, Randomness suppress backward bifurcation in an epidemic model with limited medical resources, Communications in Nonlinear Science and Numerical Simulation, 140, 108380, (2025).
[62] A. Lahrouz, H. El Mahjour, A. Settati, A. Bernoussi, Dynamics and optimal control of a non-linear epidemic model with relapse and cure, Physica A: Statistical Mechanics and its Applications, 496, 299-317, (2018).

Khalid El Bakkioui obtained a Diploma of Advanced Graduate Studies (DESA) in Functional Analysis from the Faculty of Sciences Dhar El Mahraz in Fez City (FSDMF), Morocco. He has pursued his doctoral studies at the Mathematics Department of Abdelmalek Essaâdi University in Morocco (UAE).

Youssef EL Khalfi obtained a Master's degree in modeling and scientific computing from the Faculty of Multidisciplinary (FPN) in Nador, Morocco. Since 2021, he has pursued a Ph.D. at the Department of Mathematics at Abdelmalek Essaâdi University in Morocco (UAE).

Saloua Boutouil holds a Master's in Mathematical Engineering from the Faculty of Sciences and Technology (FSTT) in Tangier, Morocco. Since 2018, he has been actively pursuing a Ph.D. degree at the Department of Mathematics at Abdelmalek Essaâdi University (UAE).

Bilal Harchaoui received a Master of Mathematical Engineering from the Faculty of Sciences and Technology (FSTT), Tangier (Morocco). He has been enrolled for a Ph.D. degree since 2018 at the Department of Mathematics at Abdelmalek Essaâdi University (UAE).

Mourad El Idrissi an esteemed scholar, obtained a Master's in Mathematical Engineering from the prestigious Faculty of Sciences and Technology (FSTT) in Tangier, Morocco. Since 2019, he has been engaged in his Ph.D. studies at the distinguished Department of Mathematics at Abdelmalek Essaâdi University in the UAE, demonstrating unwavering dedication to advancing knowledge in the field.

Adel Settati is an esteemed full professor of mathematics at the Faculty of Sciences and Technology (FSTT) in Tangier, Morocco. He completed his doctoral studies in probability at the renowned University of Rennes 1 in France. Dr. Settati's primary research interests revolve around dynamic systems, applied probability, and stochastic epidemic systems. His research contributions in these fields have been significant, and he has authored numerous research papers in high-impact scientific journals.

Aadil Lahrouz is a distinguished full professor of mathematics at the Faculty of Sciences and Technology (FSTT) located in Tangier, Morocco. Dr. Lahrouz's primary research interests revolve around dynamic systems, applied probability, and stochastic epidemic systems. His research contributions in these fields have been significant, and he has authored numerous research papers in high-impact scientific journals.