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Logistic Growth and Relapse in the Stochastic Dynamics of SIRI Epidemics

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Abstract: This paper develops a stochastic SIRI epidemic model that incorporates nonlinear relapse dynamics, logistic population growth, and a bilinear incidence rate. We begin by establishing the existence and uniqueness of a positive global solution, ensuring the model's well-posedness. Subsequently, we derive sufficient conditions that determine whether the disease will persist in the population or eventually become extinct. These theoretical results are rigorously analyzed and validated through numerical simulations, which illustrate the interplay between key model parameters and epidemic outcomes. Our findings provide valuable insights into the complex dynamics of infectious diseases with relapse and population regulation.

Keywords: Stochastic *SIRI* model, Logistic growth, Extinction of disease, Persistence

1 Introduction

Millions of people die each year from cholera, respiratory infections, measles, malaria, and dengue fever in diverse parts of the world. This is enormously happening in many countries with weak health care systems. In 2011, WHO estimated there were 1.5 million deaths from tuberculosis, 1.2 million from HIV/AIDS, and nearly half a million mortalities from malaria. The high epidemic mortality rate has significant implications for life expectancy and the economic system in the concerned countries. For example, the ongoing Coronavirus (COVID-19) caused a deep international crisis with nearly seven million deaths resulting in global health problems and changes in lifestyle. Supporting public health authorities in making informed decisions during crises requires diverse scientific approaches. Mathematical modeling, both a methodological tool and a scientific discipline, is uniquely suited to this challenge. The classical methods employing experimental and statistical approaches may not be sufficient, but the use of the dynamic models may provide an additional understanding of the transmission mechanisms of an epidemic. In 1911, S. R. Ross, applied his discovery of malaria transmission between humans and mosquitoes to explore the effectiveness of different intervention methods through mathematical modeling. For this discovery, Ross received the Nobel Prize in 1902. The development of a model for infectious disease spread marked a turning point in mathematical epidemiology, opening doors to previously unimaginable levels of analysis and prediction. In 1927 [1], public health doctor W.O. Kermack and biochemist A.G. McKendrick collaborated to publish a groundbreaking system for modeling the spread of epidemics by direct contact. Their model, focusing on the dynamics of susceptible, infectious, and recovered individuals, provided a simple yet powerful framework that researchers have since analyzed and improved upon in various studies. These developments include intricate models such as $SIR, SIS, SEIR, SIRS, SVIS$, etc. (see for instance [2,3,4,5,6,7,8]), which allow for deeper investigation of the transmission dynamics of infectious diseases within a population. In certain diseases like tuberculosis and herpes, recovered individuals can become infectious again due to the reactivation of a lingering infection. This phenomenon, known as relapse, is caused by the reactivation of previously dormant pathogens [9]. Diseases with potential for relapse, can be modeled using compartmental models, including the *SIRI* model. These models, often composed of three basic compartments representing Susceptible S, Infected I, and Recovered R individuals, track disease dynamics and evaluate

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interventions. *SIRI* models are one specific type of compartmental model with strengths in understanding infectious diseases where recovered individuals can become infectious again. If a susceptible individual encounters an infectious person, they have a chance of becoming infected, moving from the S to the I group. After a certain period of infection, typically representing the duration of symptoms and infectivity, individuals transition from the I group to the R group, representing recovery. However, in diseases like tuberculosis and herpes, recovered individuals in the R group may lose their immunity over time and Relapse into the infectious I group As a result of the reawakening of a dormant pathogen. Pioneering work by Tudor [10] established the first compartmental models incorporating relapse, employing a bilinear incidence rate within a constant total population. Building on *SIRI* models, Sanchez et al. [11] presented a novel model for the spread of behaviors, specifically focusing on drinking habits. Their model incorporated social interactions within shared drinking environments and included a non linear relapse function. The model can be represented as

$$\begin{cases} dS = [\mu - \mu S - \beta SI]dt, \\ dI = [-(\mu+\lambda)I + \beta SI + \delta RI]dt, \\ dR = [-\mu R + \lambda I - \delta RI]dt, \end{cases} \tag{1}$$

in this system, the parameters can be interpreted as μ and β represent positive constants describing the birth rate and infection coefficient, respectively. λ and δ represents the recovery rate of the infected individuals and the loss of immunity rate (relapse rate), respectively. δRI captures the rate at which individuals transition from the recovered state R back to the infected state I. βSI signifies the bilinear rate of transmission from susceptible individuals S to infected individuals I. While natural populations may initially experience exponential growth, such expansion cannot last forever. However, resource limitations, stabilize populations at a sustainable level known as the carrying capacity. Studies like [12] and [13] demonstrate the utility of logistic growth models in forecasting COVID-19 cases, with the former focusing on Egypt and Oman, and the latter investigating Alpha variant transmission dynamics in England. Several researches have integrated epidemiological models incorporating logistic growth. Wang et al. [14] examined an SIR model incorporating a susceptible population characterized by logistic growth and bilinear incidence rate. In a similar vein, Xu et al. [15] delved into a delayed SEIS model featuring logistic growth and saturation incidence, proving the stability of both disease-free and persistent states. Additionally, Perez et al. [16] further explored an SIR epidemic model incorporating a saturated treatment rate, logistic growth, and a nonlinear incidence rate, while also exploring local stability and various bifurcation types. Verhulst developed a logistic growth model [17], which defines the equation for the rate of population growth in the following way

$$dN(t) = rN\left(1 - \frac{N}{K}\right)dt,$$

where r represents the intrinsic growth rate of the population and K denotes the carrying capacity of the environment. This equation expresses how the population size N changes over time using the growth rate r. The population size is proportional to K. The importance of this equation lies in its consideration of the negative feedback mechanism experienced when larger and smaller populations vie for identical resources. When populations approach their carrying capacity,the emergence of new offspring outpaces the available resources. For diseases with a high mortality rate, using logistic growth as a framework to estimate the influx of susceptible individuals is more effective (see, for instance, [18,19,20,21,22]). Considering this, our operational hypothesis assumes that the vulnerable population in any nation adheres to the logistic growth model. To support this hypothesis, we propose a *SIRI* epidemic model incorporating logistic growth and nonlinear relapse. This model, given by the following system, includes positive constant parameters, with $r > \mu$

$$\begin{cases} dS = \left[rS\left(1 - \frac{S}{K}\right) - \mu S - \beta SI\right]dt, \\ dI = [-(\mu+\lambda)I + \beta SI + \delta RI]\,dt, \\ dR = [-\mu R + \lambda I - \delta RI]\,dt. \end{cases} \tag{2}$$

The unpredictable and stochastic nature of real-life events motivates the development of stochastic models. Consequently, numerous researchers have employed various parameter perturbation methods to develop stochastic epidemic models[23,24,25,26,27,28,29]. In line with this, Lahrouz and Settati [30,31] examined a model incorporating *SIRI* framework with nonlinear recovery rate to account for relapse behavior. To account for environmental fluctuations (see [32,33,34,35,36,37]), we introduce variability into parameters δ and β of the model (2), that is

$$\beta \longrightarrow \beta + \sigma_1 dB_1 \quad \text{and} \quad \delta \longrightarrow \delta + \sigma_2 dB_2,$$

where σ_1 and σ_2 representing the respective intensities of environmental white noises affecting the system. Here, $\sigma_1 > 0$ and $\sigma_2 > 0$ to ensure positive intensities. Additionally, $B_1(t)$ and $B_2(t)$ denote independent standard Brownian motions. By incorporating these noise terms into the previously defined system (2), we arrive at the following stochastic model

$$\begin{cases} dS = \left[rS\left(1 - \frac{S}{K}\right) - \mu S - \beta SI\right]dt - \sigma_1 SI dB_1, \\ dI = [-(\mu+\lambda)I + \beta SI + \delta RI]\,dt + \sigma_1 SI dB_1 + \sigma_2 RI dB_2, \\ dR = [-\mu R + \lambda I - \delta RI]\,dt - \sigma_2 RI dB_2. \end{cases} \tag{3}$$

The paper is structured as follows Section 2 demonstrates the existence of a unique global positive solution for system (3). In Section 3, we investigate the conditions under which the disease becomes extinct in our model. Section 4 delves into the concept of persistence in mean. Finally, Section 5 discusses our theoretical findings and provides numerical simulations to illustrate these results.

2 Existence and uniqueness of the solution

In this study section, we focus on validating the presence and uniqueness of a globally positive solution to the equation represented by (3) for all positive initial values. This involves investigating the existence and distinctiveness of such a solution in all possible scenarios. Denote the meaningful domain for system (3) by

$$\Delta = \left\{ x \in \mathbb{R}^3_+; x_1 + x_2 + x_3 < \frac{rK}{4\mu} \right\}.$$

The following theorem ensures the well-posedness of the model (3).

Theorem 1.*Let* $(S_0, I_0, R_0) \in \Delta$, *then the system* (3) *admits a unique solution* $(S(t), I(t), R(t))$ *for* $t \geq 0$ *and this solution remains in* Δ *with probability* 1.

Proof. Let $(S_0, I_0, R_0) \in \Delta$. The total population in system (3) verifies the equation

$$\mathrm{d}N = [-\mu N + rS - \frac{r}{K}S^2]dt.$$

Then, if $(S(s), I(s), R(s)) \in \mathbb{R}^3_+$ for all $0 \leq s \leq t$ almost surely, and using the following inequality $\frac{rK}{4} \geq rS - \frac{r}{K}S^2$, we get

$$dN(s) \leq (-\mu N + \frac{rK}{4})ds \quad \text{a.s.},$$

hence

$$N(t) \leq \frac{rK}{4\mu} + \left(N_0 - \frac{rK}{4\mu} \right) e^{-\mu t} \quad \text{a.s..}$$

Starting from $S(0) + I(0) + R(0) \leq \frac{rK}{4\mu}$, it can be readily demonstrated that

$$S(t) + I(t) + R(t) \leq \frac{rK}{4\mu}, \text{ for all } t \geq 0 \quad \text{a.s..} \tag{4}$$

Since the coefficients of the system (3) are locally Lipschitz continuous, for any given initial value $(S_0, I_0, R_0) \in \Delta$, there is a unique local solution $(S(t), I(t), R(t))$ on $t \in [0, \tau_e)$, where τ_e is the explosion time.
Let $\varepsilon_0 > 0$ such that $S_0, I_0, R_0 > \varepsilon_0$. For $\varepsilon \leq \varepsilon_0$ considering the stoping times

$$\tau_\varepsilon = \inf \{ t \in [0, \tau_e), S(t) \leq \varepsilon \text{ or} I(t) \leq \varepsilon \text{ or} R(t) \leq \varepsilon \}$$

and

$$\tau = \lim_{\varepsilon \to 0} \tau_\varepsilon.$$

Consider the function V defined for $(S, I, R) \in \left(0, \frac{rK}{4\mu}\right)^3$ by

$$V(S, I, R) = -\ln\left(\frac{4\mu S}{rK}\right) - \ln\left(\frac{4\mu I}{rK}\right) - \ln\left(\frac{4\mu R}{rK}\right).$$

Applying Ito's formula we obtain,
for all $t \geq 0, s \in [0, t \wedge \tau_\varepsilon]$

$$\begin{aligned}
dV &= \left[-r + \frac{rS(s)}{K} + \mu + \beta I + \frac{\sigma_1^2 I^2(s)}{2} \right] \mathrm{d}s \\
&\quad + \left[\mu + \lambda - \beta S(s) - \delta R(s) + \frac{\sigma_1^2 S^2(s)}{2} + \frac{\sigma_2^2 R^2(s)}{2} \right] \mathrm{d}s \\
&\quad + \left[\mu - \frac{\lambda I(s)}{R(s)} + \delta I(s) + \frac{\sigma_2^2 R^2(s)}{2} \right] \mathrm{d}s \\
&\quad + \sigma_1 (I(s) - S(s)) dB_1(s) + \sigma_2 (I(s) - R(s)) dB_2(s), \\
&\leq \left[3\mu + \lambda + \frac{rS(s)}{K} + (\beta + \delta) I(s) + \frac{\sigma_1^2 (I^2(s) + S^2(s))}{2} \right. \\
&\quad \left. + \frac{\sigma_2^2 (I^2(s) + R^2(s))}{2} \right] \mathrm{d}s + \sigma_1 (I(s) - S(s)) dB_1(s) \\
&\quad + \sigma_2 (I(s) - R(s)) dB_2(s).
\end{aligned}$$

Using (4) we affirm that

$$S(s), I(s), R(s) \in \left(0, \frac{rK}{4\mu}\right) \text{ for all} s \in [0, t \wedge \tau_\varepsilon] \quad \text{a.s..}$$

Therefore

$$\begin{aligned}
\mathrm{d}V &\leq k + \sigma_1 (I(s) - S(s)) dB_1(s) \\
&\quad + \sigma_2 (I(s) - R(s)) dB_2(s) \text{ a.s.},
\end{aligned}$$

where

$$k = 3\mu + \lambda + \frac{r^2}{4\mu} + \frac{(\beta + \delta) rK}{4\mu} + \frac{r^2 K^2 (\sigma_1^2 + \sigma_2^2)}{8\mu^2}.$$

Hence, by integration we obtain

$$V(S(s), I(s), R(s)) - V(S(0), I(0), R(0)) \leq ks + M_s \quad \text{a.s..}$$

Since

$$\begin{aligned}
M_s &= \int_0^s \sigma_1 (I(u) - S(u)) dB_1(u) \\
&\quad + \int_0^s \sigma_2 (I(u) - R(u)) dB_2(u)
\end{aligned}$$

is mean zero process, by taking the expectation of both parts of the above inequality, we deduce that for all $t \geq 0$

$$\begin{aligned} \mathrm{E}\left[V\left(S\left(t\wedge\tau_\varepsilon\right),I\left(t\wedge\tau_\varepsilon\right),R\left(t\wedge\tau_\varepsilon\right)\right)\right] &\leq k\left(t\wedge\tau_\varepsilon\right)+V_0, \\ &\leq kt+V_0. \end{aligned} \tag{5}$$

Follows that $V\left(S\left(t\wedge\tau_\varepsilon\right),I\left(t\wedge\tau_\varepsilon\right),R\left(t\wedge\tau_\varepsilon\right)\right)>0$, hence

$$\begin{aligned} &\mathrm{E}\left[V\left(S\left(t\wedge\tau_\varepsilon\right),I\left(t\wedge\tau_\varepsilon\right),R\left(t\wedge\tau_\varepsilon\right)\right)\right] \\ &=\mathrm{E}\left[V\left(S\left(t\wedge\tau_\varepsilon\right),I\left(t\wedge\tau_\varepsilon\right),R\left(t\wedge\tau_\varepsilon\right)\right)\chi_{(\tau_\varepsilon\leq t)}\right] \\ &+\mathrm{E}\left[V\left(S\left(t\wedge\tau_\varepsilon\right),I\left(t\wedge\tau_\varepsilon\right),R\left(t\wedge\tau_\varepsilon\right)\right)\chi_{(\tau_\varepsilon>t)}\right], \\ &\geq\mathrm{E}\left[V\left(S\left(\tau_\varepsilon\right),I\left(\tau_\varepsilon\right),R\left(\tau_\varepsilon\right)\right)\chi_{(\tau_\varepsilon\leq t)}\right], \end{aligned}$$

here, χ_A represents the characteristic function of set A.
It's important to recognize that a component of $\left(S\left(\tau_\varepsilon\right),I\left(\tau_\varepsilon\right),R\left(\tau_\varepsilon\right)\right)$ equals ε, hence

$$V\left(X\left(S\left(\tau_\varepsilon\right),I\left(\tau_\varepsilon\right),R\left(\tau_\varepsilon\right)\right)\right)\geq-\ln\left(\frac{4\mu\varepsilon}{rK}\right).$$

Thereby

$$\mathrm{E}\left[V\left(S\left(\tau_\varepsilon\right),I\left(\tau_\varepsilon\right),R\left(\tau_\varepsilon\right)\right)\right]\geq-\ln\left(\frac{4\mu\varepsilon}{rK}\right)\mathrm{P}\left(\tau_\varepsilon\leq t\right). \tag{6}$$

By combining (5) with (6), we deduce for all $t \geq 0$

$$\mathrm{P}\left(\tau_\varepsilon\leq t\right)\leq\frac{-\left(kt+V_0\right)}{\ln\left(\frac{4\mu\varepsilon}{rK}\right)}.$$

Letting $\varepsilon \to 0$, one has $P(\tau \leq t) = 0$ for all $t \geq 0$. This implies that $P(\tau = \infty) = 1$. Since $\tau_e \geq \tau$, it follows that $\tau_e = \tau = \infty$ a.s.. Wich concludes the proof of the theorem.

3 Extinction of disease

When examining the dynamics of a stochastic epidemic model, a notable scenario arises when the threshold equals one, remaining untreated in all instances. To address this, we present an approach based on stopping times τ_ε and τ, introducing two positive numbers

$$\mathscr{R}_\delta=\frac{\delta rK}{4\mu\left(\mu+\lambda+\frac{1}{2}\left(\frac{\sigma_2 rK}{4\mu}\right)^2\right)},$$

$$\mathscr{R}_\beta=\frac{\beta rK}{4\mu\left(\mu+\lambda+\frac{1}{2}\left(\frac{\sigma_1 rK}{4\mu}\right)^2\right)}.$$

Theorem 2. *For any given initial values $(S(0),I(0),R(0)) \in \Delta$, if any of the followings assumptions*

(C1) $\mathscr{R}_\delta = 1, \quad \delta-\beta \geq \sigma_2^2\frac{rK}{4\mu}$ *and* $\beta \geq \sigma_1^2$

and

(C2) $\mathscr{R}_\beta = 1, \quad \beta-\delta \geq \sigma_1^2\frac{rK}{4\mu}$ *and* $\delta \geq \sigma_2^2$

holds, then we get

$$\lim_{t\to\infty} I(t) = 0 \ a.s..$$

Proof. We consider a positive constant ε satisfy the condition $\varepsilon \in (0,1)\cap(0,I_0)$.
Define two stopping times

$$\tau_1=\inf\{t\geq 0, I(t)\leq\varepsilon\},\quad \tau_1'=\inf\{t\geq\tau_1, I(t)\geq\varepsilon\}.$$

The first step in our proof involves establishing that $\mathbb{E}(\tau_1) < \infty$.
For all $T > 0$ and $t \leq T \wedge \tau_1$, we have

$$I(t)\geq\varepsilon \text{ a.s..} \tag{7}$$

Applying Ito's lemma, we can obtain the following equation

$$\begin{aligned} d\log(I) &= \left(-(\mu+\lambda)+\beta S+\delta R-\frac{1}{2}\left(\sigma_1^2S^2+\sigma_2^2R^2\right)\right)dt \\ &+\sigma_1 S dB_1+\sigma_2 R dB_2, \\ &=\phi(S,R)dt+\sigma_1 S dB_1+\sigma_2 R dB_2. \end{aligned}$$

Where ϕ is defined on $\left(0,\frac{rK}{4\mu}\right)$ by

$$\phi(x,y)=-(\mu+\lambda)+\beta x+\delta y-\frac{1}{2}\left(\sigma_1^2x^2+\sigma_2^2y^2\right). \tag{8}$$

If (C1) holds, in view of $\beta \geq \sigma_1^2$, the function $x \mapsto \phi(x,.)$ is clearly an increasing function. Since $S<\frac{rK}{4\mu}-R<\frac{rK}{4\mu}$, we can therefore conclude that

$$\begin{aligned} d\log(I) &\leq \left[\beta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_1^2\left(\frac{rK}{4\mu}\right)^2\right. \\ &+\left.\left(\delta-\beta+\sigma_1^2\frac{rK}{4\mu}\right)R-\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)R^2\right]dt \\ &+\sigma_1 S dB_1+\sigma_2 R dB_2, \\ &\triangleq F(R)dt+\sigma_1 S dB_1+\sigma_2 R dB_2. \end{aligned} \tag{9}$$

Since $\delta-\beta \geq \sigma_2^2\frac{rK}{4\mu}$, the function F is increasing on $(0,\frac{rK}{4\mu})$.
By (7) we have $R \leq \frac{rK}{4\mu}(1-\varepsilon)$, injecting this in (9) leads

to

$$\begin{aligned} d\log I \leq & \left[-\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)\left(\frac{rK}{4\mu}(1-\varepsilon)\right)^2\right. \\ & +\left(\delta-\beta+\sigma_1^2\frac{rK}{4\mu}\right)\left(\frac{rK}{4\mu}(1-\varepsilon)\right) \\ & \left.+\beta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_1^2\left(\frac{rK}{4\mu}\right)^2\right]dt \\ & +\sigma_1 S dB_1+\sigma_2 R dB_2, \\ = & \left[\left(\mu+\lambda+\frac{1}{2}\sigma_2^2\left(\frac{rK}{4\mu}\right)^2\right)(\mathscr{R}_\delta-1)\right. \\ & +\varepsilon\left(\frac{rK}{4\mu}\right)\left(\frac{rK}{4\mu}\sigma_2^2+\beta-\delta\right) \\ & \left.-\frac{\varepsilon^2}{2}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)\right]dt \\ & +\sigma_1 S dB_1+\sigma_2 R dB_2, \\ = & \left[\varepsilon\left(\frac{rK}{4\mu}\right)\left(\frac{rK}{4\mu}\sigma_2^2+\beta-\delta\right)\right. \\ & \left.-\frac{\varepsilon^2}{2}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)\right]dt \\ & +\sigma_1 S dB_1+\sigma_2 R dB_2. \end{aligned}$$

By integrating the given inequality from 0 to $\tau_1 \wedge T$ and then taking the expectation on both sides, we obtain

$$\begin{aligned} & \varepsilon\left[\left(\frac{rK}{4\mu}\right)\left(\delta-\beta-\frac{rK}{4\mu}\sigma_2^2\right)+\frac{\varepsilon}{2}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)\right]\mathbb{E}(T\wedge\tau_1) \\ & \leq -\mathbb{E}(\log I(T\wedge\tau_1))+\log I(0). \end{aligned}$$

Letting $T \longrightarrow +\infty$ and applying Fatou's lemma, we get

$$\begin{aligned} \mathbb{E}(\tau_1) \leq & \frac{-\log\left(\frac{rK}{4\mu}\varepsilon\right)+\log I(0)}{\varepsilon\left[\left(\frac{rK}{4\mu}\right)\left(\delta-\beta-\frac{rK}{4\mu}\sigma_2^2\right)+\frac{\varepsilon}{2}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)\right]}, \\ & <\infty. \end{aligned}$$

Next, we define a sequence of stopping times τ_n and τ_n' for $n>1$.

$$\tau_n' = \inf\{t \geq \tau_n, I(t) \geq \varepsilon^n\},$$

$$\tau_{n+1} = \inf\left\{\tau_n < t < \tau_n', I(t) \leq \varepsilon^{n+1}\right\}.$$

By using similar reasoning as the initial step of the proof, we can establish the result.

$$\begin{aligned} & \mathbb{E}(\tau_{n+1})-\mathbb{E}(\tau_n) \\ & \leq \frac{-\log\left(\frac{rK}{4\mu}\varepsilon\right)+\log I(0)}{\varepsilon^{n+1}\left[\left(\frac{rK}{4\mu}\right)\left(\frac{rK}{4\mu}\sigma_2^2+\delta-\beta\right)+\frac{\varepsilon^{n+1}}{2}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)\right]}. \end{aligned}$$

By using induction, we can conclude that $\mathbb{E}(\tau_n)<\infty$ for all$n \in \mathbb{N}$. The sequence $(\tau_n)_{n\in\mathbb{N}}$ is, by definition, increasing. As a consequence $\tau_n \longrightarrow \tau_\infty$ and the family $(\tau_n<\infty)$ is decreasing. This implies that

$$\mathbb{P}\left(\bigcap_{n=1}^{\infty}(\tau_n<\infty)\right)=\lim_{n\to\infty}\mathbb{P}(\tau_n<\infty)=1.$$

We assert that $\mathbb{P}(\tau_\infty<\infty)=0$. To achieve a contradiction, let us consider that there exists $\omega \in (\tau_\infty<\infty)$. For such ω, we have $I(\tau_n(\omega))=\varepsilon^n$ for all $n\in\mathbb{N}^*$. By utilizing the continuity of $I(t)$ and taking the limit, we obtain

$$I(\tau_\infty(\omega))=0.$$

Define $\tau_0=\inf\{t>0,\ I(t)=0\}$. This implies that $(\tau_\infty<\infty)\subset(\tau_0<\infty)$. Consequently,

$$\mathbb{P}(\tau_0<\infty)\geq\mathbb{P}(\tau_\infty<\infty)>0.$$

This leads to a contradiction, as it conflicts with the fact that $\mathbb{P}(\tau_0=\infty)=1$. Therefore, we conclude that

$$\mathbb{P}(\tau_\infty<\infty)=0. \tag{10}$$

We can verify that

$$\mathbb{P}(\Omega')=1 \quad \text{where} \quad \Omega'=\bigcap_{n=1}^{\infty}(\tau_n<\infty). \tag{11}$$

From (10) and (11), it follows $\mathbb{P}((\tau_\infty=\infty)\cap\Omega')=1$. Consider $\omega\in(\tau_\infty=\infty)\cap\Omega'$, with $t>0,\eta>0$, and $n_0=\left[\frac{\log\eta}{\log\varepsilon}\right]$, where $[\cdot]$ denotes the integer part. For $t\geq\tau_{n_0}(w)$, there exists an n such that

$$n\geq n_0 \text{ and } \tau_n(w)\leq t\leq\tau_{n+1}(w),$$

this implies

$$\varepsilon^{n+1}\leq I(t,w)\leq\varepsilon^n\leq\varepsilon^{n_0}=\varepsilon^{\frac{\log\eta}{\log\varepsilon}}=\eta,$$

thus, we have

$$\lim_{t\to\infty}I(t,w)=0 \text{a.s.}.$$

Similarly, if conditions (C2) holds, it can be shown that $\lim_{t\to\infty}I(t,w)=0$ almost surely as well.

Now we will analyze the disease-free dynamics under these two assumptions

(C3) $\mathscr{R}_\delta<1, \delta-\beta\geq\sigma_2^2\frac{rK}{4\mu}$ and $\beta\geq\sigma_1^2$

and

(C4) $\mathscr{R}_\beta<1, \beta-\delta\geq\sigma_1^2\frac{rK}{4\mu}$ and $\delta\geq\sigma_2^2$.

Theorem 3.*Suppose that either (C3) or (C4) holds. Then, for any given initial values $(S(0),I(0),R(0))\in\Delta$, the disease-free equilibrium state $E^0(1,0,0)$ of system (3) is globally asymptotically stable in probability.*

*Proof.*Let us consider the Lyapunov function

$$\mathscr{V} = a_1(r-\mu-\frac{r}{K}S)^2 + kI^{1/k} + a_2R^2,$$

where $k>1$ and a_1, a_2 are positive constants that will be determined appropriately. The differential operator $\mathscr{L}$, acting on the function $\mathscr{V}$, is given by

$$\begin{aligned}\mathscr{L}\mathscr{V} =& -2a_1\frac{r}{K}(r-\mu-\frac{r}{K}S)[rS\left(1-\frac{S}{K}\right)-\mu S-\beta SI]\\ &+a_1\left(\frac{r}{K}\right)^2\sigma_1^2S^2I^2+I^{1/k}[-(\mu+\lambda)+\beta S+\delta R\\ &+\frac{1}{2}\left(\frac{1}{k}-1\right)(\sigma_1^2S^2+\sigma_2^2R^2)\Big]\\ &+2a_2R[-\mu R+\lambda I-\delta RI]+a_2\sigma_2^2R^2I^2.\end{aligned}$$

Employing $(S,I,R)\in\Delta$ and the inequality $I^j \le I^{1/k}\left(\frac{rK}{4\mu}\right)^{j-1/k}$ for $j\in\{1,2\}$ yields to

$$\begin{aligned}\mathscr{L}\mathscr{V} \le& -2a_1\frac{r}{K}S(r-\mu-\frac{r}{K}S)^2-2a_2\mu R^2-2a_2\delta R^2I\\ &+I^{1/k}\Bigg[2a_1\beta\frac{r}{K}\left(\frac{rK}{4\mu}\right)^{2-1/k}(r+\mu+\frac{r^2}{4\mu})\\ &+a_1\left(\frac{r}{K}\right)^2\left(\frac{rK}{4\mu}\right)^{4-1/k}\sigma_1^2+2a_2\lambda\left(\frac{rK}{4\mu}\right)^{2-1/k}\\ &+a_2\left(\frac{rK}{4\mu}\right)^{4-1/k}\sigma_2^2+\frac{1}{2k}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)\\ &+\left(-(\mu+\lambda)+\beta S+\delta R-\frac{1}{2}\left(\sigma_1^2S^2+\sigma_2^2R^2\right)\right)\Bigg] \qquad (12)\\ =& -2a_1\frac{r}{K}S(r-\mu-\frac{r}{K}S)^2-2a_2\mu R^2-2a_2\delta R^2I\\ &+I^{1/k}\Bigg[a_1\frac{r}{K}\left(\frac{rK}{4\mu}\right)^{2-1/k}\left(2\beta\left(r+\mu+\frac{r^2}{4\mu}\right)\right.\\ &\left.+\left(\frac{r}{K}\right)\left(\frac{rK}{4\mu}\right)^2\sigma_1^2\right)+\frac{1}{2k}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)\\ &+a_2\left(\frac{rK}{4\mu}\right)^{2-1/k}\left(2\lambda+\left(\frac{rK}{4\mu}\right)^2\sigma_2^2\right)+\phi(S,R)\Bigg].\end{aligned}$$

Suppose that (C3) holds. Given that $\beta\ge\sigma_1^2$, the function ϕ defined in (8) has the property that for any $y\in(0,\frac{rK}{4\mu})$, the function $x\to\phi(x,y)$ is increasing. Therefore, for any $(x,y)\in\{x+y\le\frac{rK}{4\mu}\}$, we can conclude that

$$\begin{aligned}\phi(x,y) \le& \phi(\frac{rK}{4\mu}-y,y),\\ =& \beta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_1^2(\frac{rK}{4\mu})^2\\ &+\left(\delta-\beta+\sigma_1^2\frac{rK}{4\mu}\right)y-\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)y^2,\\ \triangleq& F(y). \qquad (13)\end{aligned}$$

Given that $\delta-\beta\ge\sigma_2^2\frac{rK}{4\mu}$, the function F is increasing on $(0,\frac{rK}{4\mu})$.
This implies $F(y)<F(\frac{rK}{4\mu})$.
Substituting this inequality into (13) yields

$$\phi(x,y)\le\left(\delta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_2^2\left(\frac{rK}{4\mu}\right)^2\right).$$

By combining this inequality into (12), we obtain

$$\begin{aligned}\mathscr{L}\mathscr{V}(x) \le& -2a_1\frac{r}{K}S(r-\mu-\frac{r}{K}S)^2\\ &-2a_2\mu R^2-2a_2\delta R^2I+AI^{1/k}. \qquad (14)\end{aligned}$$

where

$$\begin{aligned}A =& a_1\frac{r}{K}\left(\frac{rK}{4\mu}\right)^{2-1/k}\left(2\beta\left(r+\mu+\frac{r^2}{4\mu}\right)+\left(\frac{r}{K}\right)\left(\frac{rK}{4\mu}\right)^2\sigma_1^2\right)\\ &+a_2\left(\frac{rK}{4\mu}\right)^{2-1/k}\left(2\lambda+\left(\frac{rK}{4\mu}\right)^2\sigma_2^2\right)\\ &+\frac{1}{2k}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)\\ &+\left(\delta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_2^2\left(\frac{rK}{4\mu}\right)^2\right).\end{aligned}$$

Since the threshold $\mathscr{R}_\delta<1$, we can find a sufficiently large positive integer value for k such that

$$k>\frac{\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)}{-\delta\frac{rK}{2\mu}+2(\mu+\lambda)+\sigma_2^2\left(\frac{rK}{4\mu}\right)^2}.$$

We can now strategically choose positive values for a_1 and a_2 to be small enough to ensure that

$$a_2<-\frac{\frac{1}{2k}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)+\left(\delta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_2^2\left(\frac{rK}{4\mu}\right)^2\right)}{\left(\frac{rK}{4\mu}\right)^{2-1/k}\left(2\lambda+\left(\frac{rK}{4\mu}\right)^2\sigma_2^2\right)},$$

and

$$a_1<-\frac{a_2\left(\frac{rK}{4\mu}\right)^{2-1/k}\left(2\lambda+\left(\frac{rK}{4\mu}\right)^2\sigma_2^2\right)+\frac{1}{2k}\left(\frac{rK}{4\mu}\right)^2\left(\sigma_1^2+\sigma_2^2\right)+\left(\delta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_2^2\left(\frac{rK}{4\mu}\right)^2\right)}{\frac{r}{K}\left(\frac{rK}{4\mu}\right)^{2-1/k}\left(2\beta\left(r+\mu+\frac{r^2}{4\mu}\right)+\left(\frac{r}{K}\right)\left(\frac{rK}{4\mu}\right)^2\sigma_1^2\right)}.$$

This manipulation in equation (14) results in all the coefficients associated with $(1-S)^2, I^{\frac{1}{k}}$ and R^2 being negative. Furthermore, assuming condition (C4) is satisfied, we can leverage the fact that $\delta\ge\sigma_2^2$. This property implies that the function $y\mapsto\phi(.,y)$ is monotonically increasing with respect to its second argument y. Consequently, for any pair of values (x,y) satisfying the constraint $x+y\le\frac{rK}{4\mu}$, we can conclude that

$$\begin{aligned}\phi(x,y) &\leq \phi(x,\frac{rK}{4\mu}-x),\\ &= \delta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_2^2(\frac{rK}{4\mu})^2\\ &\quad+\left(\beta-\delta+\sigma_2^2\frac{rK}{4\mu}\right)x-\frac{1}{2}\left(\sigma_1^2+\sigma_2^2\right)x^2,\\ &\triangleq G(x). \end{aligned} \tag{15}$$

Since $\beta-\delta\geq\sigma_1^2\frac{rK}{4\mu}$, the function G is increasing on $(0,\frac{rK}{4\mu})$, which means $G(y)<G(\frac{rK}{4\mu})$. Substituting this inequality in (15) yields

$$\phi(x,y)\leq\left(\beta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_1^2\left(\frac{rK}{4\mu}\right)^2\right).$$

Since $\mathscr{R}_\beta<1$, then, by applying the same arguments as before, we can easily verify that

$$\begin{aligned}\mathscr{L}V(x) &\leq -2a_1'\frac{r}{K}S(r-\mu-\frac{r}{K}S)^2-2a_2'\mu R^2\\ &\quad-2a_2'\delta R^2I+A'I^{1/k'}.\end{aligned}$$

where

$$\begin{aligned}A' &= a_1'\frac{r}{K}\left(\frac{rK}{4\mu}\right)^{2-1/k}\left(2\beta\left(r+\mu+\frac{r^2}{4\mu}\right)+\left(\frac{r}{K}\right)\left(\frac{rK}{4\mu}\right)^2\sigma_1^2\right)\\ &+a_2'\left(\frac{rK}{4\mu}\right)^{2-1/k'}\left(2\lambda+\left(\frac{rK}{4\mu}\right)^2\sigma_2^2\right)+\frac{1}{2k'}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)\\ &+\left(\beta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_1^2\left(\frac{rK}{4\mu}\right)^2\right),\end{aligned}$$

$$k'>\frac{\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)}{-\beta\frac{rK}{2\mu}+2(\mu+\lambda)+\sigma_1^2\left(\frac{rK}{4\mu}\right)^2},$$

$$0<a_2'<-\frac{\frac{1}{2k'}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\left(\beta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_1^2\left(\frac{rK}{4\mu}\right)^2\right)}{\left(\frac{rK}{4\mu}\right)^{2-1/k'}\left(2\lambda+\left(\frac{rK}{4\mu}\right)^2\sigma_2^2\right)},$$

and

$$0<a_1'<-\frac{a_2'\left(\frac{rK}{4\mu}\right)^{2-1/k'}\left(2\lambda+\left(\frac{rK}{4\mu}\right)^2\sigma_2^2\right)+\frac{1}{2k'}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\left(\beta\frac{rK}{4\mu}-(\mu+\lambda)-\frac{1}{2}\sigma_1^2\left(\frac{rK}{4\mu}\right)^2\right)}{\frac{r}{K}\left(\frac{rK}{4\mu}\right)^{2-1/k'}\left(2\beta\left(r+\mu+\frac{r^2}{4\mu}\right)+\left(\frac{r}{K}\right)\left(\frac{rK}{4\mu}\right)^2\sigma_1^2\right)}.$$

Consequently, the coefficients of $(1-S)^2, I^{\frac{1}{k}}$ and R^2 are all negatives. As a consequence, based on the properties established in Theorem 3, we can conclude that the proof is complete.

4 Persistence

The most interesting topics in analyzing infectious disease models often revolve around two key outcomes: extinction and persistence. Section 3 addressed the concept of extinction. In this section, we will focus on demonstrating that the disease is persistent in mean. To address this, we define a positive number

$$\mathscr{R}=\frac{r(\beta K+4\mu)}{4\mu\left(\mu+\lambda+\frac{1}{2}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)}.$$

For analytical convenience, we introduce the following notation $<x>=\frac{1}{t}\int_0^t x(s)ds$.

Theorem 4.*If $\mathscr{R}>1$ and $\beta\leq\frac{r}{K}$. Then for any given initial value $(S(0),I(0),R(0))\in\Delta$, the solution of system (3) has the property that*

$$(i)\ \liminf_{t\to\infty}<I>\geq\frac{\left(\mu+\lambda+\frac{1}{2}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)(\mathscr{R}-1)}{\mu+\lambda+\beta}\ a.s.,$$

$$(ii)\ \liminf_{t\to\infty}<R>\geq\frac{\lambda\left(\mu+\lambda+\frac{1}{2}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)(\mathscr{R}-1)}{(\mu+\delta\frac{rK}{4\mu})(\mu+\lambda+\beta)}\ a.s..$$

*Proof.*Let consider the second equation of the system (3). By integrating both sides of this equation, we get

$$\begin{aligned}\frac{I-I_0}{t} &=-(\mu+\lambda)<I>+\beta<SI>+\delta<RI>\\ &\quad+\frac{\sigma_1}{t}\int_0^t SIdB_1+\frac{\sigma_2}{t}\int_0^t RIdB_2.\end{aligned} \tag{16}$$

Applying Ito's formula to model (3) yields the following equations

$$d\ln S=[r-\frac{r}{K}S-\mu-\beta I]dt-\sigma_1IdB_1+\frac{\sigma_1^2}{2}I^2dt, \tag{17}$$

and

$$\begin{aligned}d\ln I &=[-(\mu+\lambda)+\beta S+\delta R-\frac{\sigma_1^2}{2}S^2-\frac{\sigma_2^2}{2}R^2]dt\\ &\quad+\sigma_1SdB_1+\sigma_2RdB_2.\end{aligned} \tag{18}$$

Taking the integrals of both sides of equations (17) and (18) over the time interval from 0 to t, and then dividing both sides by t, leads to

$$\begin{aligned}\frac{\ln S(t)-\ln S(0)}{t} &=r-\frac{r}{K}<S>-\mu-\beta<I>\\ &\quad+\frac{\sigma_1^2}{2}<I^2>-\frac{\sigma_1}{t}\int_0^t IdB_1,\end{aligned} \tag{19}$$

$$\begin{aligned}\frac{\ln I(t)-\ln I(0)}{t} &=-(\mu+\lambda)+\beta<S>+\delta<R>\\ &\quad-\frac{\sigma_1^2}{2}<S^2>-\frac{\sigma_2^2}{2}<R^2>\\ &\quad+\frac{\sigma_1}{t}\int_0^t SdB_1+\frac{\sigma_2}{t}\int_0^t RdB_2.\end{aligned} \tag{20}$$

We combine (16), (19), and (20) and derive that

$$\begin{aligned}
&\frac{\ln S(t)-\ln S(0)}{t}+\frac{\ln I(t)-\ln I(0)}{t}+\frac{I-I_0}{t}\\
=&r-(\mu+\lambda)-(\mu+\lambda+\beta)<I>+(\beta-\frac{r}{K})<S>\\
&+\delta<R>-\frac{\sigma_1^2}{2}<S^2>+\frac{\sigma_1^2}{2}<I^2>-\frac{\sigma_2^2}{2}<R^2>\\
&+\beta<SI>+\delta<RI>-\frac{\sigma_1}{t}\int_0^t IdB_1+\frac{\sigma_1}{t}\int_0^t SdB_1\\
&+\frac{\sigma_2}{t}\int_0^t RdB_2+\frac{\sigma_1}{t}\int_0^t SIdB_1+\frac{\sigma_2}{t}\int_0^t RIdB_2,\\
\geq&\frac{r}{4\mu}(\beta K+4\mu)-\left(\mu+\lambda+\frac{1}{2}(\frac{rK}{4\mu})^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)\\
&-(\mu+\lambda+\beta)<I>+\frac{\sigma_1}{t}\int_0^t(S+SI-I)dB_1\\
&+\frac{\sigma_2}{t}\int_0^t(R+RI)dB_2,
\end{aligned}\tag{21}$$

then

$$\begin{aligned}
<I>\geq&\frac{\left(\mu+\lambda+\frac{1}{2}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)(\mathscr{R}-1)}{\mu+\lambda+\beta}\\
&+\frac{1}{\mu+\lambda+\beta}\left(\frac{1}{t}M_1(t)+\frac{1}{t}M_2(t)-\frac{\ln S(t)-S(0)}{t}\right.\\
&\left.-\frac{\ln I(t)-I(0)}{t}-\frac{I-I_0}{t}\right).
\end{aligned}$$

Where $M_1(t)=\sigma_1\int_0^t(S+SI-I)dB_1$ and $M_2(t)=\sigma_2\int_0^t(R+RI)dB_2$.
By the strong law of large number theorem for martingales (Lipster [38]) and the fact that $S(t),I(t),R(t)\in\left(0,\frac{rK}{4\mu}\right)$, which yields that

$$\begin{aligned}
&\lim_{t\to\infty}\frac{S(t)}{t}=0,\quad \lim_{t\to\infty}\frac{I(t)}{t}=0,\\
&\lim_{t\to\infty}\frac{M_1(t)}{t}=0\quad a.s.,\quad \lim_{t\to\infty}\frac{M_2(t)}{t}=0\quad a.s.,
\end{aligned}$$

therefore,

$$\liminf_{t\to\infty}<I>\geq\frac{\left(\mu+\lambda+\frac{1}{2}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)(\mathscr{R}-1)}{\mu+\lambda+\beta}a.s.\tag{22}$$

This validates the necessary assertion (i).
(ii) Integrating the third equation of system (3) yields

$$\begin{aligned}
\frac{R-R_0}{t}=&-\mu<R>+\lambda<I>-\sigma<RI>-\frac{\sigma_1}{t}\int_0^t RIdB_2,\\
\geq&-(\mu+\delta\frac{rK}{4\mu})<R>+\lambda<I>-\frac{\sigma_1}{t}\int_0^t RIdB_2.
\end{aligned}\tag{23}$$

Then

$$\begin{aligned}
<R>\geq&\frac{1}{\mu+\delta\frac{rK}{4\mu}}\left(\lambda<I>-\frac{R(t)-R(0)}{t}\right.\\
&\left.-\frac{\sigma_2}{t}\int_0^t R(u)I(u)dB_2(u)\right).
\end{aligned}$$

According to the strong law of large number theorem for martingales and the fact that $R\in\left(0,\frac{rK}{4\mu}\right)$, we derive

$$\lim_{t\to\infty}\left(\frac{R(t)-R(0)}{t}-\frac{\sigma_2}{t}\int_0^t R(u)I(u)dB_2(u)=0\right)\text{ a.s..}$$

Then, applying (i), we obtain

$$\liminf_{t\to\infty}<R>\geq\frac{\lambda\left(\mu+\lambda+\frac{1}{2}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)(\mathscr{R}-1)}{(\mu+\delta\frac{rK}{4\mu})(\mu+\lambda+\beta)}a.s..$$

This completes the proof of the Theorem 4.

5 Simulations

This section presents numerical simulations to visualize the behavior predicted by our main results. We will employ a numerical method specifically designed for stochastic differential equations to approximate the solutions of system (3). This method, known as the Milstein method. By applying this method to system (3), we obtain a discretized equation that can be used to generate numerical solutions.

$$\begin{cases}
S_{k+1}=&S_k+\left[rS_k\left(1-\frac{S_k}{K}\right)-\mu S_k-\beta S_kI_k\right]\Delta t\\
&-\sigma_1S_kI_k\sqrt{\Delta t}\tau_k-\frac{\sigma_1^2}{2}S_kI_k\left(\tau_k^2-1\right)\Delta t,\\
I_{k+1}=&I_k+[-(\mu+\lambda)I_k+\beta S_kI_k+\delta R_kk_k]\Delta t\\
&+\sigma_1S_kI_k\sqrt{\Delta t}\tau_k+\frac{\sigma_2^2}{2}S_kI_k\left(\tau_k^2-1\right)\Delta t\\
&+\sigma_2R_kI_k\sqrt{\Delta t}\xi_k+\frac{\sigma_2^2}{2}R_kI_k\left(\xi_k^2-1\right)\Delta t,\\
R_{k+1}=&R_k+[-\mu R_k+\lambda I_k-\delta\beta R_kI_k]\Delta t\\
&-\sigma_2R_kI_k\sqrt{\Delta t}\xi_k-\frac{\sigma_2^2}{2}R_kI_k\left(\xi_k^2-1\right)\Delta t.
\end{cases}$$

Here , τ_k and ξ_k $(k=1,2,...)$ represent independent random variables. These variables follow a normal distribution $N(0,1)$.

*Example 1.*For system (3), we choose $K=1.6$, $r=0.3$, $\mu=0.1$, $\lambda=0.4$ and initial value $(S(0),I(0),R(0))=(0.7,0.3,0.1)$.
Case 1: Let $\sigma_1=0.3,\sigma_2=0.35,\delta=0.4902$ and $\beta=0.3$. Then $\mathscr{R}_\delta=1$, $\beta\geq\sigma_1^2$ and $\delta-\beta\geq\sigma_2^2\frac{rK}{4\mu}$.

Case 2: Let $\sigma_1 = 0.45, \sigma_2 = 0.2, \delta = 0.1$ and $\beta = 0.5382$. Then $\mathscr{R}_\beta = 1, \delta \geq \sigma_2^2$ and $\beta - \delta \geq \sigma_1^2 \frac{rK}{4\mu}$.

From upon Theorem 2, we can establish that the disease-free equilibrium state, denoted by $E^0(1,0,0)$, of the system described by equation (3) is globally asymptotically stable in probability. This implies that with probability one (almost surely), solutions to the system will converge to this disease-free state over time.
Furthermore, as illustrated in Figure 1, the trajectories of the solution paths for the system (3) visually demonstrate their convergence towards the equilibrium point $E^0(1,0,0)$.

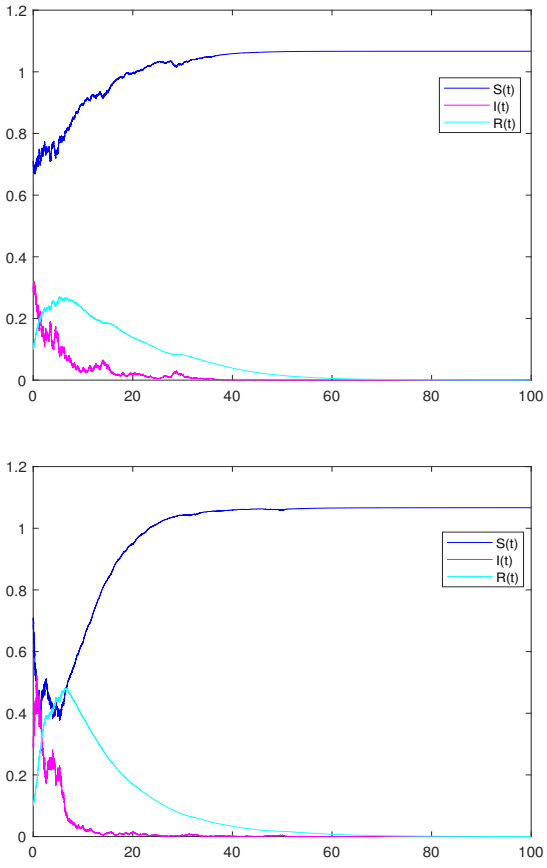

Fig. 1: Trajectories of $S(t)$, $I(t)$, and $R(t)$ for the scenario presented in Example 1. The upper panel shows the path of these variables over time in Case 1, while the lower panel displays the corresponding paths in Case 2.

*Example 2.*The parameters K, r, μ and λ are identical to those used in Example 1.
Case 1 : Let $\sigma_1 = 0.35, \sigma_2 = 0.3, \delta = 0.4$ and $\beta = 0.25$. Then $\mathscr{R}_\delta = 0.8499 < 1$, $\delta - \beta \geq \sigma_2^2 \frac{rK}{4\mu}$ and $\beta \geq \sigma_1^2$.
Case 2 : Let $\sigma_1 = 0.35, \sigma_2 = 0.25, \delta = 0.2$ and $\beta = 0.4$. Then $\mathscr{R}_\beta = 0.816 < 1$, $\beta - \delta \geq \sigma_1^2 \frac{rK}{4\mu}$ and $\delta \geq \sigma_2^2$. From Theorem 3, we can establish that the disease-free equilibrium state, denoted by $E^0(1,0,0)$, of the system described by equation (3) is globally asymptotically stable in probability. This implies that with probability one (almost surely), solutions to the system will converge to this disease-free state over time.
Furthermore, as illustrated in Figure 2, the trajectories of the solution paths for the system (3) visually demonstrate their convergence towards the equilibrium point $E^0(1,0,0)$.

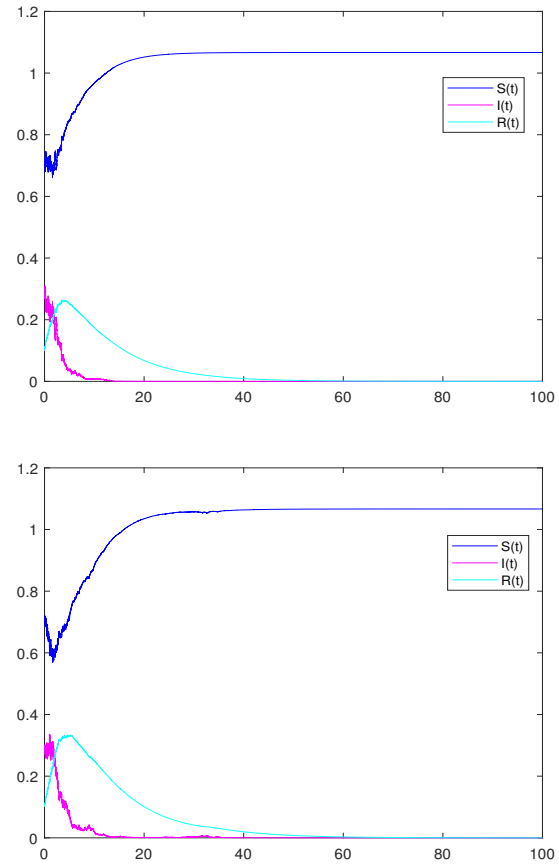

Fig. 2: Trajectories of $S(t)$, $I(t)$, and $R(t)$ for the scenario presented in Example 2. The upper panel shows the path of these variables over time in Case 1, while the lower panel displays the corresponding paths in Case 2.

*Example 3.*The parameters K, r and μ are the same as those specified in Example 1. Let's select the parameters

in system (3) as follows $\lambda = 0.1$, $\sigma_1 = 0.15$, $\sigma_2 = 0.1$,$\delta = 0.3$, $\beta = 0.18$ and initial value $(S(0),I(0),R(0)) = (0.7,0.3,0.1)$. Then $\mathscr{R} = 1.1694 > 1$, $(4\mu)^2 - r(4\mu + \frac{\sigma_2^2}{2}K^2) \geq 0$ and $\beta \leq \frac{r}{K}$. Theorem 4 implies that the disease being persistent in mean. Figure 3 supports this conclusion.

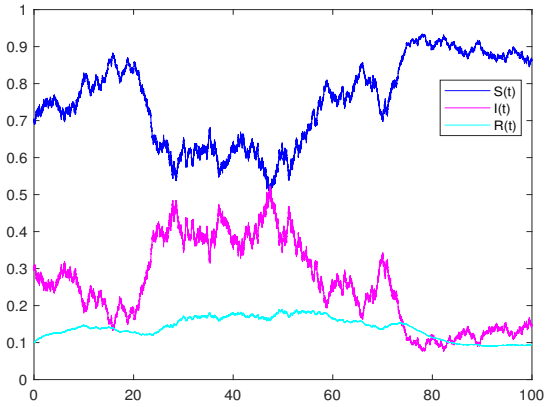

Fig. 3: Trajectories of $S(t)$, $I(t)$, and $R(t)$ for the scenario presented in Example 3.

6 Conclusion

In conclusion, this paper has successfully developed and analyzed a novel stochastic SIRI epidemic model incorporating nonlinear relapse dynamics, logistic population growth, and a bilinear incidence rate. This model represents a significant contribution to the field of mathematical epidemiology, offering a more nuanced understanding of disease transmission by considering the complexities of relapse and population regulation. Through rigorous mathematical analysis, we established the existence and uniqueness of a global positive solution, ensuring the model's well-posedness and biological relevance. Furthermore, our theoretical analysis provided explicit conditions for disease extinction and persistence. Theorem 2 states that if either of the conditions

$$(C1) \quad \mathscr{R}_\delta = 1, \quad \delta - \beta \geq \sigma_2^2 \frac{rK}{4\mu}, \quad \beta \geq \sigma_1^2$$

or

$$(C2) \quad \mathscr{R}_\beta = 1, \quad \beta - \delta \geq \sigma_1^2 \frac{rK}{4\mu}, \quad \delta \geq \sigma_2^2$$

holds, then the infection will almost surely die out. Conversely, Theorem 4 establishes that if $\mathscr{R} > 1$ and $\beta \leq \frac{r}{K}$, the disease persists with the following lower bounds on the long-term averages of the infected and recovered populations:

$$\liminf_{t\to\infty}\langle I\rangle \geq \frac{\left(\mu+\lambda+\frac{1}{2}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)(\mathscr{R}-1)}{\mu+\lambda+\beta} \quad \text{a.s.}$$

$$\liminf_{t\to\infty}\langle R\rangle \geq \frac{\lambda\left(\mu+\lambda+\frac{1}{2}\left(\frac{rK}{4\mu}\right)^2(\sigma_1^2+\sigma_2^2)+\frac{r^2}{4\mu}\right)(\mathscr{R}-1)}{(\mu+\delta\frac{rK}{4\mu})(\mu+\lambda+\beta)} \text{a.s.}$$

These theoretical findings were rigorously validated and illustrated through numerical simulations, which showcased the dynamic interplay between key model parameters and epidemic outcomes. The numerical results not only support our theoretical findings but also provide valuable insights into the practical implications of the model, allowing for a better understanding of how different factors influence the spread and control of infectious diseases with relapse. Future work could extend this model by incorporating vaccination, treatment strategies, or spatial heterogeneity. This work provides a solid foundation for further investigations into the dynamics of infectious diseases and offers valuable tools for public health decision-making.

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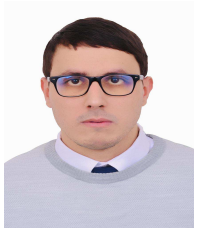

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