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Research Article

A Numerical and Analytical Study of a Stochastic Epidemic SIR Model in the Light of White Noise

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This study examines a novel SIR epidemic model that takes into account the impact of environmental white noise. According to the study, white noise has a significant impact on the disease. First, we establish the solution's existence and uniqueness. Following that, we explain that the stochastic basic production $\mathfrak{R}_0$ is a threshold that determines the extinction or persistence of the disease. When noise levels are high, we acquire $\mathfrak{R}_0 < 1$, which causes the sickness to disappear. A sufficient condition for the existence of a stationary distribution is archived when the noise intensity is high, which suggests the infection is prevalent when $\mathfrak{R}_0 > 1$. Finally, numerical simulations are used to explain the key findings.

1. Introduction

The goal of this research is to show how challenging SIR models are for understanding the epidemic and to offer a useful model for establishing proposal insights into its spread. The traditional susceptible-infected-removed SIR model of Kermack and McKendrick is the progenitor of nearly all mathematical models for the transmission of infectious illnesses. Numerous researchers have thoroughly studied the dynamic behavior of various epidemic models and many of their expansions. The presence of the threshold values that determine whether a disease dies out, the stability of the disease-free and endemic equilibria, permanence, and extinction are the fundamental and essential study topics for contemporary studies.

For many years, the spread and transmission of illnesses have been questioned and examined. In reality, Graunt was the first scientist to attempt to scientifically quantify causes of mortality [1], and his investigation of causes of death resulted in a hypothesis that is now widely accepted among current epidemiologists. Bernoulli was the first mathematician to propose an infectious disease mathematical model. He modeled the transmission of smallpox [2], which was widespread at the time, and advocated for the benefits of variolation [3] in 1760. In 1927, McKendrick and Kermack proposed a basic deterministic (compartmental) model for forecasting the behavior of epidemic outbreaks [4]. SIR models are an extremely versatile modeling approach developed by the researchers. They are often used in the modeling of infectious illnesses using mathematics. People are divided into groups with the letters S, I, and R (susceptible, infectious, and recovered). ODE, which are deterministic, are most often applied to run the models, but they may also be used with a stochastic (random) framework, which is more realistic but trickier to evaluate.

According to John M. Last, epidemiology is the study of the spread and determinants of disease or well-being status in a population, or it is the outlet of medicine that deals with the occurrence, distribution, possible mechanism of malady, and other factors related to health. It is the foundation of common safety and nature's tactic varieties, as well as evidence-based preparation by distinct risk factors for illness and emphases on protective curative amenities. Syndrome diffusion experts assist by deliberating on proposals, variety,

and measurable investigation of evidence, altering understanding and spread of outcomes (calculating viscount inspection and periodic proficient review). As a result, epidemiology has generated techniques in scientific analysis, common safety education, and, less suggestively, fundamental surveys in the biological disciplines [5]. Disease causality, diffusion, epidemic analysis, disease observation, environmental epidemiology, forensic epidemiology, occupational epidemiology, screening, biomonitoring, and comparisons of cure effects, such as in clinical trials, are all important areas of epidemiological study. Further scientific castigations are used by epidemiologists, such as biology to better understand disease progressions, statistics to make actual use of data and advance appropriate outcomes, social sciences to better understand local and terminal grounds, and engineering to increase revelation.

The word "epidemiology" is usually used to describe and illuminate not only epidemics and infectious diseases, but disease in general, as well as associated circumstances. High blood pressure, mental disease, and obesity are objective insufficient of the concerns studied by epidemiology. As a consequence, this epidemiology is based on how the pattern of disease produces a change in human function. Mathematical research has generated great improvements in practical and theoretical fields [6–8].

To investigate the influence of environmental conditions on the epidemic model and make the results more realistic, we first developed a stochastic mathematical SIR model. Recent scientific advances with a focus on the transmission of numerous infectious illnesses (SDE) and (ODE) have been front and center. An SDE is a differential equation in which one or more of the terms are stochastic processes, with the solution likewise being a stochastic process. SDEs often include a variable that is calculated as the Wiener process or Brownian motion derivative and represents random white noise. Other kinds of random behavior, including jump processes, are indeed feasible. Stochastic differential equations and random differential equations are conjugate [9], while differential equation having one or more functions of one independent variable and their derivatives is known as an ordinary differential equation (ODE) in mathematics. Ordinary differential equations are applied in contrast to partial differential equations, which may refer to more than one independent variable [10].

The current paper will investigate the persistence and extinction of the epidemic, provide the system's threshold value, and be affected by motion brought on by white noise. Even though stochastic system perturbations have many more varied features, we still took into account how this system's threshold compares to those of other models that include the same motion [11]. Finally, we visualize the numerical simulations using MATLAB.

2. Stochastic Epidemic Model Description

In this section, we provide our new stochastic model in the form of differential equations.

(i) The total inhabitant $\mathfrak{N}_t$ is distributed in three compartments: $\mathcal{S}_t$, $\mathcal{I}_t$, and $\mathcal{R}_t$ represent the susceptible, infected peoples, and recovered people, respectively

(ii) The indicated stochastic model's variables and parameters are both nonnegative

(iii) We perturbed β and γ, i.e., $\beta \longrightarrow \beta + \sigma_1 \mathfrak{B}_1$ and $\gamma \longrightarrow \gamma + \sigma_2 \mathfrak{B}_2$. Where $\mathfrak{B}_1, \mathfrak{B}_2$ are the Brownian motion with the property $\mathfrak{B}_{1_0} = 0 = \mathfrak{B}_{2_0}$ and the intensity σ_1^2, σ_2^2 are positive

Remark 1. The deterministic general epidemic study estimates that if $\mathfrak{R}_0 < 1$, a small outburst will aries, and if $\mathfrak{R}_0 > 1$, a large outbreak will occur, infecting a large chunk of the population. The results are based on the assumption that the community is homogeneous and that individuals mingle evenly. However, if the hypothesis of an evenly mixed society is accepted, the model may not be appropriate in particular situations. When contemplating a tiny population, such as an epidemic outbreak in a daycare center or school, it appears logical to presume that the eventual number of infected will be unpredictable or random. Also, even if $\mathfrak{R}_0 > 1$ and the society is huge, if the outbreak is started by only one (or a few) early invective's, the plagues may never take off by accident. The formulation of a related stochastic epidemic model is motivated by these two aspects. It allows parameter estimation from disease outbreak data to include standard error, and the subject of the disease extinction is better suited for the stochastic model for researching epidemic diseases.

In the light of above speculations, we established the below new stochastic SIR epidemic model.

$$\begin{aligned} &d(S_t) = (\Lambda - \beta S_t I_t - dS_t)dt - \sigma_1 S_t I_t d\mathfrak{B}_1(t), \\ &, d(I_t) = (\beta S_t I_t - (d+\gamma)dI_t - \mathfrak{h}(I))dt + \sigma_1 S_t I_t d\mathfrak{B}_1(t) - \sigma_2 I_t d\mathfrak{B}_2(t), \\ &d(R_t) = (\gamma I + \mathfrak{h}(I) - dR)dt + \sigma_2 I_t d\mathfrak{B}_2(t). \end{aligned} \tag{1}$$

The above Table 1 represents the parameters and their values, while Table 2 represents the compartments and their values. Note that $h(I)$ is the elimination of the transferable entities due to the cure of the form:

$$h(I) = \begin{cases} \mathfrak{M} > 0 & \text{for} \quad I > 0\,; \\ \mathfrak{M} = 0 & I = 0. \end{cases} \tag{2}$$

The authors [12] have the following deterministic.

$$\begin{aligned} \frac{d(S_t)}{dt} &= \Lambda - \beta S_t I_t - dS_t, \\ \frac{d(I_t)}{dt} &= \beta S_t I_t - (d+\gamma)dI_t - \mathfrak{h}(I), \\ \frac{d(R_t)}{dt} &= \gamma I + \mathfrak{h}(I) - dR, \end{aligned} \tag{3}$$

Table 1: Parametric description of the model.

Symbol	Description	Value
Λ	Constitute the recruitment rate in the susceptible inhabitant	0.1
β	Is the diffusion rate	0.01
d	Is the usual passing away rate	0.006
γ	Is the impulsive salvage amount of the virulent entities	0.03

Table 2: Compartments and description.

Symbol	Description	Value
$\mathcal{S}$	Susceptible	20
I	Infected peoples	6
$\mathcal{R}$	Recovered peoples	1

and

$$d(\mathfrak{N}) = \Lambda - d\mathfrak{N}, \tag{4}$$

where $\mathfrak{N}_t = S_t + I_t + R_t$ indicates the entire constant residents for $\Lambda \approx \mu\mathfrak{N}$ and $\mathfrak{N}_0 = \mathcal{S}_0 + \mathcal{I}_0 + \mathcal{R}_0$. Equation (4) has the exact solution

$$\mathfrak{N}_t = e^{-dt}\left[\mathfrak{N}_0 + \frac{\Lambda}{d}e^{dt}\right]. \tag{5}$$

Also, we have

$$0 \geq \mathcal{S}_0, 0 \geq \mathcal{I}_0, \mathcal{R}_0 \geq 0 \Rightarrow S_t \geq 0, I_t \geq 0, 0 \leq R_t. \tag{6}$$

So that the result has positivity property. For the stability analysis of the model (3), we have the reproduction number, which is

$$\mathfrak{R}_0 = \frac{\beta\Lambda}{(d+\gamma)d}. \tag{7}$$

If $\mathfrak{R}_0 < 1$, the system (3) will be locally steady and will be unsteady if $\mathfrak{R}_0 \geq 1$ asymptotically stable. Further, the system (3) will be globally asymptotic if $\Lambda = 0$.

3. Preliminaries

Throughout this paper, we formulated the necessary assumptions. Suppose Rd_+ is the d-dimensional Euclidean space. $R_+^d = \{j \in R^d : 0 < j_i, d > 1\}$.

Let $(\mho, F, \mathfrak{P})$ a whole probability space that has been filtered by $\{F\}_{\mathfrak{t}\geq 0}$ and $\{\mathfrak{B}_\mathfrak{t}\}_{\mathfrak{t}\geq 0}$ is a 1-dimensional Brownian motion defined on it. Usually, we consider a SDE of n-dimension as

$$d\omega(\mathfrak{t}) = \mathfrak{F}(\mathfrak{y}(\mathfrak{t}), \mathfrak{t})d\mathfrak{t} + \mathfrak{G}(\mathfrak{y}(\mathfrak{t}), \mathfrak{t})d\mathfrak{B}(\mathfrak{t})), \quad \text{for} \quad \mathfrak{t} \geq \mathfrak{t}_0, \tag{8}$$

with initial value $\mathfrak{y}(\mathfrak{t}_0) = \mathfrak{y}_0 \varepsilon \mathfrak{R}^d$. By defining the dimensional operator £ with equation (8)

$$£ = \frac{\partial}{\partial\mathfrak{t}} + \sum_{i=1}^{d}\mathfrak{F}_i(\mathfrak{y}, \mathfrak{t})\frac{\partial}{\partial\mathfrak{y}_i} + \frac{1}{2}\sum_{i,j=1}^{d}\left[\mathfrak{G}^T(\mathfrak{y}, \mathfrak{t})\mathfrak{G}(\mathfrak{y}, \mathfrak{t})\right]_{ij}\frac{\partial^2}{\partial\mathfrak{y}_i\partial\mathfrak{y}_j}. \tag{9}$$

If the operator £ acts on the a function $\mathfrak{V} = (\mathbb{R}^d \times \mathbb{R}_+ ; \mathbb{R}_+)$ then

$$£\mathfrak{V} = \mathfrak{V}_t(\mathfrak{y}, \mathfrak{t}) + \mathfrak{V}_\mathfrak{y}(\mathfrak{y}, \mathfrak{t})\mathfrak{F}(\mathfrak{y}, \mathfrak{t}) + \frac{1}{2}\text{trace}\left[\mathfrak{G}^T(\mathfrak{y}, \mathfrak{t})\mathfrak{V}_{\mathfrak{y}\mathfrak{y}}\mathfrak{G}(\mathfrak{y}, \mathfrak{t})\right]. \tag{10}$$

4. Existence and Uniqueness

By utilizing the technique in [13–15], the following theorem can be proof with ease.

Theorem 2. *(S_t, I_t, R_t) is a unique positive solution of system (1)for $t \geq 0$ with $(\mathcal{S}_0, \mathcal{I}_0, \mathcal{R}_0) \in R_+^3$, and result will be left in R_+^3, with probability equals to one.*

We outline a $\mathfrak{C}^2$-function $\mathfrak{U} : \mathbb{R}_+^3 \longrightarrow \mathbb{R}_+$, by the resulting formulation

$$\mathfrak{U}(\mathcal{S}_t, \mathcal{I}_t, R_t) = (S_t - 1 - \ln S_t) + \left(I_t - \left(\frac{2}{3} + \frac{1}{3}\right) - \ln I_t\right) + \left(R_t - \left(\frac{2}{3} + \frac{1}{3}\right) - \ln R_t\right). \tag{11}$$

By applying Ito formula, we have

$$d\mathfrak{U}(S_t, I_t, R_t) = \left(1 - \frac{1}{S_t}\right)dS_t + \frac{1}{2S_t^2}(dS_t)^2 + \left(1 - \frac{1}{I_t}\right) \cdot dI_t + \frac{1}{2I_t^2}(dI_t)^2 + \left(1 - \frac{1}{R_t}\right)dR_t + \frac{1}{2R_t^2}(dR_t)^2, \tag{12}$$

$$= \mathfrak{L}^*\mathfrak{U}dt + \sigma_1(\mathcal{I}_t - \mathcal{S}_t)d\mathfrak{B}_1(t) + \sigma_2(\mathcal{I}_t - \mathcal{S})d\mathfrak{B}_2(t), \tag{13}$$

where $\mathfrak{L}^*\mathfrak{U} : \mathbb{R}_+^3 \longrightarrow \mathbb{R}_+$ is defined by

$$\begin{aligned}\mathfrak{L}^*\mathfrak{U} &= \left(1-\frac{1}{S_t}\right)(\Lambda-\beta S_t I_t - dS_t) + \frac{1}{2}\sigma_1^2 I^2 \\ &+ \left(1-\frac{1}{I_t}\right)(\beta S_t I_t - d(d+\gamma)I_t) + \frac{1}{2}\sigma_1^2 S^2 + \frac{1}{2}\sigma_2^2 I^2 \\ &+ \left(1-\frac{1}{R_t}\right)(\gamma I_t - dR) + \sigma_2^2, = \Lambda - \beta S_t I_t - dS_t - dS_t \\ &- \frac{\Lambda}{\mathcal{S}_t} + \beta I_t + d + \beta S_t I_t - (d+\gamma)d - \beta S_t + (d+\gamma)d \\ &+ \gamma I - dR_t - \gamma\frac{I_t}{R_t} + d + \frac{1}{2}\sigma_1^2 S^2 + \frac{1}{2}\sigma_2^2 I_+^2 \sigma_2^2. \leq \Lambda + d \\ &+ d^2 + \gamma d + d + + \frac{1}{2}\sigma_1^2 S^2 + \frac{1}{2}\sigma_2^2 I_+^2 \sigma_2^2 \coloneqq A.\end{aligned} \tag{14}$$

The rest of the proof can be followed from [16–18].

5. Extinction of the Disease

In this part, we will figure out when the sickness will wipe out, as well as when it will resurface. As a result, system's (1) basic reproduction number is provided. We may deduce the following lemmas from the proof in [19].

Lemma 3 (see [20]). *Let (S_t, I_t, R_t) be the solution of system (1) with initial value $(S_0, I_0, R_0) \in \mathbb{R}_+^3$. Then*

$$Lim_{t\longrightarrow\infty}\frac{S_t + I_t + R_t}{t} = 0 \quad a.s. \tag{15}$$

6. Remark

In fact, combine with solution positivity and equation (15), we have by [20]

$$\mathrm{Lim}_{t\longrightarrow\infty}\left(\frac{\mathcal{S}_t}{t}\right) = 0, \mathrm{Lim}_{t\longrightarrow\infty}\left(\frac{I_t}{t}\right) = 0, \mathrm{Lim}_{t\longrightarrow\infty}\left(\frac{R_t}{\mathfrak{t}}\right) = 0\ a.s., \tag{16}$$

and according to lemma 2.2 of [20], we have

Lemma 4. *Assume $d > 1/2(\sigma_1^2 \vee \sigma_2^2)$. Let (S_t, I_t, R_t) be the solution of system (1) with initial value $(S_0, I_0, R_0) \in \mathbb{R}_+^3$, then*

$$\begin{aligned}Lim_{t\longrightarrow\infty}\frac{\int_0^t S(r)}{t} &= 0, \\ Lim_{t\longrightarrow\infty}\frac{\int_0^t S(r)}{t} &= 0, \\ Lim_{t\longrightarrow\infty}\frac{\int_0^t S(r)}{t} &= 0.\end{aligned} \tag{17}$$

$\mathfrak{R}_0 = \beta\Lambda/d(d+\gamma)$ is the basic reproduction of the system (3) in [12].

and

$$\mathfrak{R}_0^{\bullet} = \frac{\beta\Lambda}{d\left(d+\gamma+(1/2)\sigma_2^2\right)} = \mathfrak{R}_0 - \frac{\beta\Lambda}{2d(d+\gamma)\left(d+\gamma+(1/2)\sigma_2^2\right)}\sigma_2^2. \tag{18}$$

We will study the results in the next part based on Lemma 3 and 4.

Theorem 5. *Suppose $d > 1/2(\sigma_1^2 \vee \sigma_2^2)$. Let (S_t, I_t, R_t) be the solution of the system (1) with any initial value $(S_0, I_0, R_0) \in \mathbb{R}_+^3$. If $1 > \mathfrak{R}_0^{\bullet}$, then*

$$Lim_{t\longrightarrow\infty}\ \sup\ \frac{\log I_t}{t} \leq \left(d+\gamma+\frac{1}{2}\sigma_2^2\right)(\mathfrak{R}_0^{\bullet} - 1) < 0\ a.s. \tag{19}$$

I_t approaches to 0 exponentially almost sure. In other words, the illness will most likely die out.

Proof. From system (1), we have

$$\begin{aligned}\frac{-S_0 + S_t}{t} &= -(-\Lambda) - \frac{d}{t}\int_0^t S(s)ds - \frac{\beta}{t}\int_0^t I(s)S(s) \\ &\cdot ds - \frac{\sigma_1}{t}\int_0^t S(s)I(s)d\mathfrak{B}_1(s), \\ \frac{-I_0 + I_t}{t} &= \beta\frac{\int_0^t S(s)I(s)ds}{t} - d(d+\gamma)\frac{\int_0^t I(s)ds}{t} \\ &- \frac{\int_0^t h(I)ds}{t} + \sigma_1\frac{\int_0^t S(s)I(s)}{t}d\mathfrak{B}_1(s) \\ &- \sigma_2\frac{\int_0^t I(s)d\mathfrak{B}_2(s)}{t}, \\ \frac{-R_0 + R_t}{t} &= \gamma\frac{\int_0^t I(s)ds}{t} + \frac{\int_0^t h(I)ds}{t} - d\frac{\int_0^t R(s)ds}{t} + \sigma_2\frac{\int_0^t I(s)d}{t}\mathfrak{B}_2(s),\end{aligned} \tag{20}$$

then

$$\begin{aligned}d\frac{\int_0^t S(s)ds}{t} + d(d+\gamma)\frac{\int_0^t I(s)ds}{t} &= \Lambda - \frac{I_t - S_t}{t} + \frac{I_0 - S_0}{t} \\ &- \frac{\int_0^t h(I)ds}{t} - \sigma_2\frac{\int_0^t I(s)d\mathfrak{B}_2(s)}{t} = \Lambda + \xi(t),\end{aligned} \tag{21}$$

where $\xi(t)$ possesses the property that

$$\mathrm{Lim}_{t\longrightarrow\infty}\xi(t) = 0. \tag{22}$$

According to (15) and (17), we have

$$\mathrm{Lim}_{t\longrightarrow\infty}\frac{d\int_0^t S(s)ds + d(d+\gamma)\int_0^t I(s)ds}{t} = \Lambda. \tag{23}$$

Furthermore

$$\begin{aligned}\log \mathrm{I}_t - \log I_0 &= \beta\int_0^t \mathrm{S}(s)ds - \left(d+\gamma+\frac{1}{2}\sigma_2^2\right)\\ &\quad\cdot t + \sigma_1 \mathrm{S}(s)\mathfrak{B}_1(t) - \sigma_2\mathfrak{B}_2(t),\\ \log \mathrm{I}_t &= \log I_0 + \beta\frac{\Lambda}{d}t - \beta(d+\gamma)\int_0^t I(s)ds + \frac{\beta}{d}t\xi(t)\\ &\quad - \left(d+\gamma+\frac{1}{2}\sigma_2^2\right)t + \sigma_1\mathrm{S}(s)\mathfrak{B}_1(t) - \sigma_2\mathfrak{B}_2(t),\\ &= \left[t\beta\frac{\Lambda}{d} - \left(\gamma+\frac{1}{2}\sigma_2^2+d\right)\right]t - \beta(d+\gamma)\int_0^t I(s)ds\\ &\quad + \log I_0 + \frac{\beta}{d}t\xi(t) + \sigma_1\mathrm{S}(s)\mathfrak{B}_1(t) - \sigma_2\mathfrak{B}_2(t).\\ &\le \left[\beta\frac{\Lambda}{d}t - \left(d+\gamma+\frac{1}{2}\sigma_2^2\right)\right]t + \log I_0\\ &\quad + \frac{\beta}{d}t\xi(t) - \sigma_2\mathfrak{B}_2(t),\end{aligned} \tag{24}$$

and

$$\mathrm{Lim}_{t\longrightarrow\infty}\frac{1}{t}\left[\log I_0 + \frac{\beta}{d}t\xi(t) - \sigma_2\mathfrak{B}_2(t)\right] = 0 \quad a.s. \tag{25}$$

By (22) and the property of Brownian motion. If $1 > \mathfrak{R}_0^{\bullet}$, then, from (24), we have

$$\begin{aligned}\mathrm{Lim}_{t\longrightarrow\infty} \quad \sup\ \frac{\log I_t}{t} &\le \frac{\beta\Lambda}{d} - \left(d+\gamma+\frac{1}{2}\sigma_2^2\right)\\ &= \left(d+\gamma+\frac{1}{2}\sigma_2^2\right)(\mathfrak{R}_0^{\bullet}-1) < 0,\end{aligned} \tag{26}$$

as required. □

7. Persistence of the Disease

In this section, we will look at the infection's persistence in the pandemic context (1), with the following theorem introducing our main result.

Theorem 6. *Suppose $d > 1/2(\sigma_1^2 \vee \sigma_2^2)$. Let (S_t, I_t, R_t) be the solution of the system (1) with any initial value $(S_0, I_0, R_0) \in \mathbb{R}_+^3$. If $1 < \mathfrak{R}_0^{\bullet}$, then*

$$\begin{aligned}&Lim_{t\longrightarrow\infty}\frac{1}{t}\int_0^t S(s)ds = \frac{\Lambda}{d\mathfrak{R}_0^{\bullet}},\\ &Lim_{t\longrightarrow\infty}\frac{1}{t}\int_0^t \mathscr{I}(s)ds = \frac{d\left(\gamma+(1/2)\sigma_2^2+d\right)}{(d\beta+\gamma\beta)}(\mathfrak{R}_0^{\bullet}-1),\\ &Lim_{t\longrightarrow\infty}\frac{1}{t}\int_0^t \mathscr{R}(s)ds = \frac{\gamma\left(d+\gamma+(1/2)\sigma_2^2\right)}{\beta(d+\gamma)}(\mathfrak{R}_0^{\bullet}-1).\end{aligned} \tag{27}$$

Proof. If $\mathfrak{R}_0^{\bullet} > 1$, then, by (24) and by Lemma 3 and 5.2 in [21].

$$\begin{aligned}\mathrm{Lim}_{t\longrightarrow\infty}\frac{1}{t}\int_0^t I(s)ds &= \frac{(1/d)(\beta\Lambda) - \left(d+(1/2)\sigma_2^2+\gamma\right)}{(d\beta+\gamma\beta)/d},\\ &= \frac{\gamma\left(d+\gamma+(1/2)\sigma_2^2\right)}{\beta(d+\gamma)}(\mathfrak{R}_0^{\bullet}-1).\end{aligned} \tag{28}$$

Along with (23)

$$\mathrm{Lim}_{t\longrightarrow\infty}\frac{1}{t}\int_0^t S(s)ds = \frac{\Lambda}{d} - \left(\frac{\gamma+d+(1/2)\sigma_2^2}{\beta}\right)(\mathfrak{R}_0^{\bullet}-1), = \frac{\Lambda}{d\mathfrak{R}_0^{\bullet}}. \tag{29}$$

Further, integrating from 0 to t the last equation of system (1), we get

$$\frac{R_t - R_0}{t} = \frac{\gamma}{t}\int_0^t I(s)ds + \int_0^t h(I)ds - \frac{d}{t}\int_0^t R(s)ds + \frac{\sigma_2^2}{t}\int_0^t R(s)d\mathfrak{B}_2(s), \tag{30}$$

now (17) and (28) ⇒

$$\mathrm{Lim}_{t\longrightarrow\infty}\frac{1}{t}\int_0^t \mathscr{R}(s)ds = \frac{\left(\gamma+d+(1/2)\sigma_2^2\right)\gamma}{\beta(d+\gamma)}(\mathfrak{R}_0^{\bullet}-1). \tag{31}$$

□

Remark 7. Theorems 5 and 6 reveal that the illness's ability to die out or endure is highly influenced by the strength of white noise disturbances, with tiny white noise disturbances promoting long-term disease prevalence and big white noise disturbances causing the epidemic disease to die out.

8. Numerical Scheme and Results

Our study of disease extinction and persistence has now concluded. We will now perform some numerical simulations of (1) to illustrate the applicability of our findings. The Milstein technique [22] is used to generate numerical simulations. Consider the model's discretization equation:

$$\begin{aligned}S_{k+1} &= S_k + (\Lambda - \beta S_k I_k - dS_k)\Delta t - \sigma_1 S_k I_k\sqrt{\Delta t}\tau_k - \frac{\sigma_1^2}{2}S_k I_k\left(\tau_k^2-1\right)\Delta t,\\ I_{k+1} &= I_k + (\beta S_k I_k - d(d+\gamma)I_k - \mathfrak{M})\Delta t + \sigma_1 S_k I_k\sqrt{\Delta t}\tau_k\\ &\quad + \frac{\sigma_1^2}{2}S_k I_k\left(\tau_k^2-1\right)\Delta t - \sigma_2 I_k\sqrt{\Delta t}\tau_k - \frac{\sigma_2^2}{2}I_k\left(\tau_k^2-1\right)\Delta t,\\ R_{k+1} &= R_k + (\gamma I_k + \mathfrak{M} - dR_k)\Delta t + \sigma_2 I_k\sqrt{\Delta t}\tau_k + \frac{\sigma_2^2}{2}I_k\left(\tau_k^2-1\right)\Delta t.\end{aligned} \tag{32}$$

8.1. Numerical Data. Here, we highlight the numerical data for the stochastic model (1). For the parametric and initial values, we refer to [12].

Figures 1(a) and 1(b) are the comparison of S class in the deterministic system and in the stochastic system, with

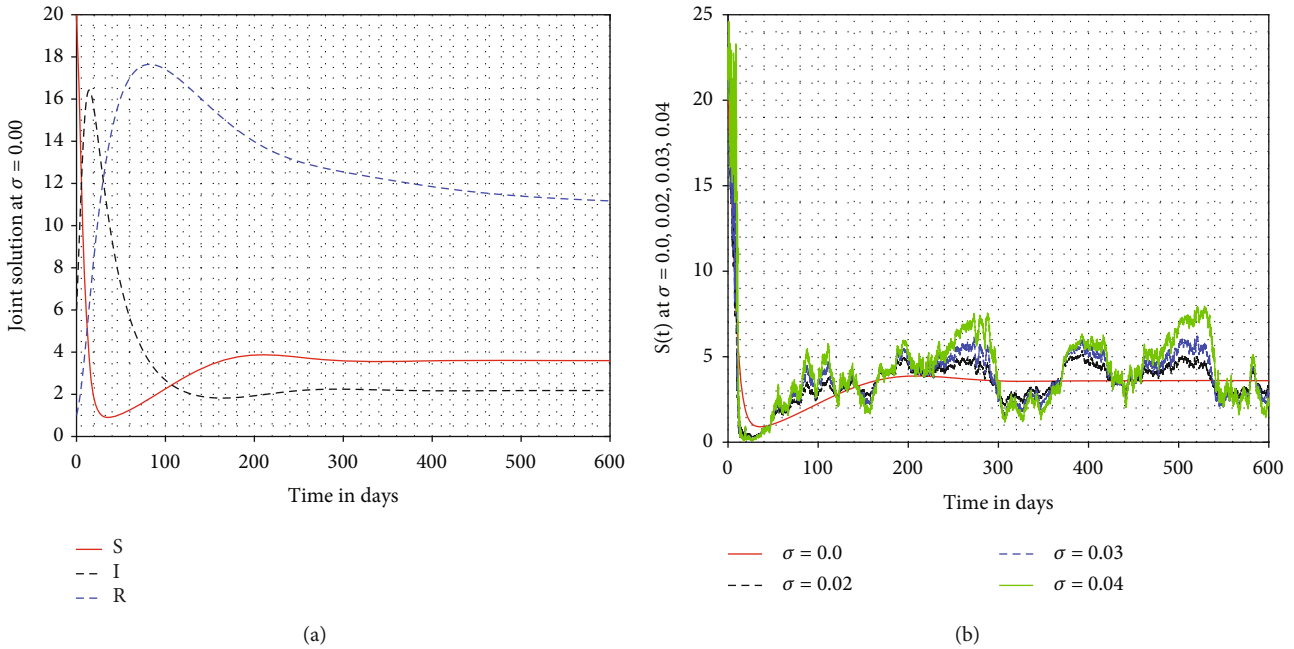

Figure 1: (a, b) Joint solution of (1) at $\sigma = 0.0$ and $S(t)$.

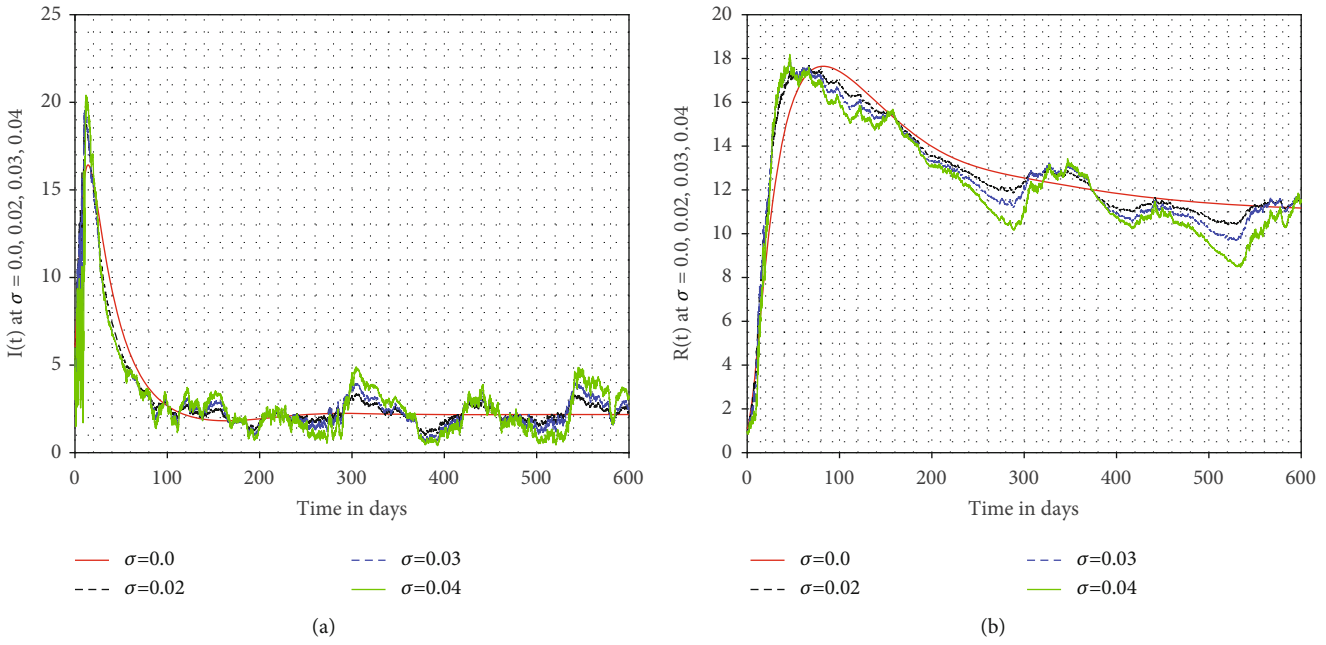

Figure 2: (a, b) $I(t)$ and $R(t)$ for different values of σ.

$\Lambda = 0.1, d = 0.006, m = 0.00001, \beta = 0.01, \gamma = 0.03$ and initial values $S(0) = 20, I(0) = 6, R(0) = 1$. In Figures 1(a) and 1(b), we have presented the joint solution of the model (1) for $\sigma = 0.0$, S, and different values of σ. Comparing the first figure, the noise getting smaller, the fluctuation of the system of model (1) is getting weaker. If we increase the value of $\sigma = 0.02, 0.03, 0.04$, respectively, the amplitude of fluctuation becomes stronger. That is to say, noise intensities have great effect on the solution of S.

Figures 2(a) and 2(b) are the comparison of I, R classes in the deterministic system and in the stochastic system, with $\Lambda = 0.1, d = 0.006, m = 0.00001, \beta = 0.01, \gamma = 0.03$ and initial values $S(0) = 20, I(0) = 6, R(0) = 1$. In Figure 2, we presented the dynamics of I and R of the model (1) for $\sigma = 0.0$, S, and different values of σ. Then, I will tend to zero exponentially with probability one. That is to say, an event distinct from its corresponding deterministic model might cause the illness to become extinct when there are enormous noises (3). The role of parameters on the stochastic model (1) has an importance. For observing this, we have modified the parametric values and observed a change in the dynamics of all the classes. Even, there is a change in the dynamics as a whole

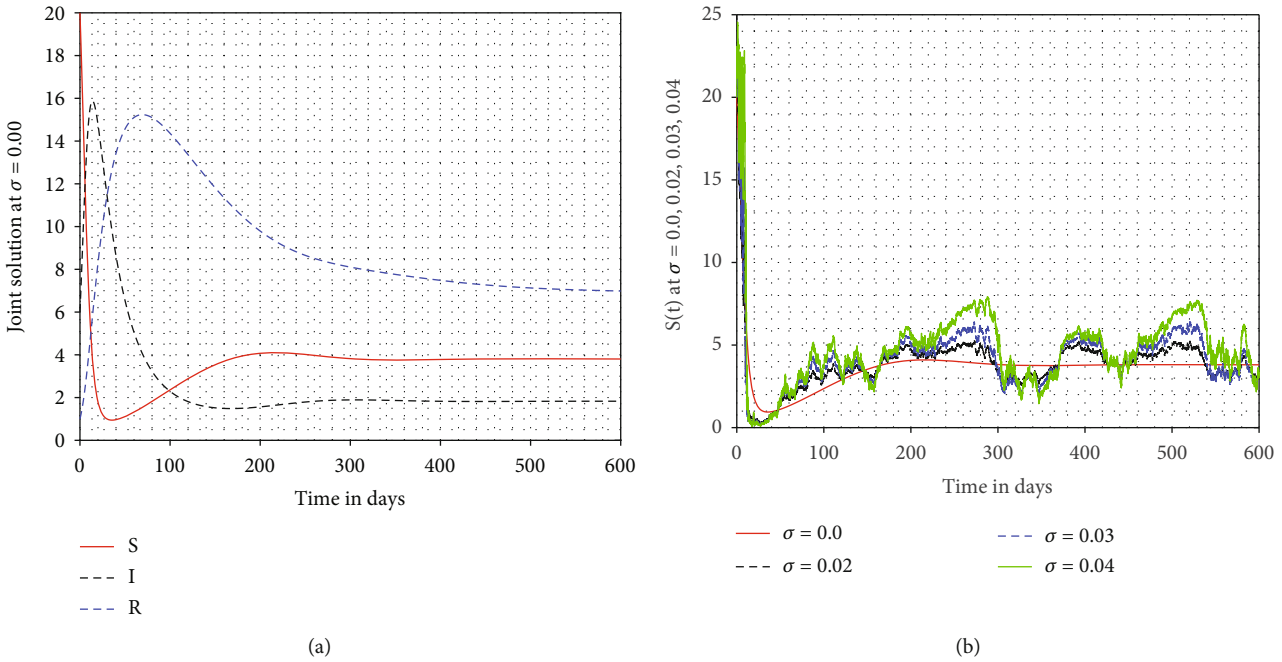

FIGURE 3: (a, b) Joint solution of (1) at $\sigma = 0.0$ and $S(t)$.

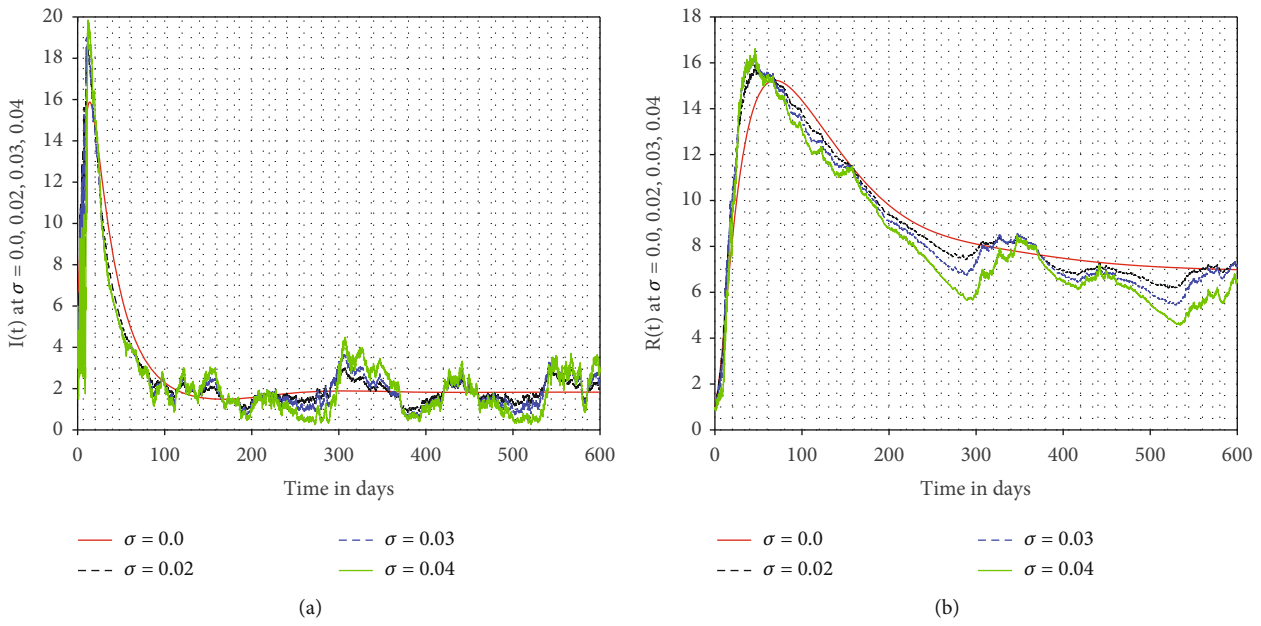

FIGURE 4: (a, b) $I(t)$ and $R(t)$ for different values of σ.

which can be observed in Figure 3(a). In Figures 3 and 4, the considered parametric values are $\Lambda = 0.1, d = 0.008, r = 0.1, m = 0.0001, \beta = 0.01, \gamma = 0.03$ and while keeping the initial values unchanged and σ is changed as mentioned in the graphs.

9. Conclusions

In this research, we explored the dynamic behavior of a novel SIR epidemic model that takes into account the impact of information intervention and environmental noise. Information intervention and white noise have been demonstrated to have significant effects on the condition.

The following are the key findings:

(i) We have thought about how white noise in the environment affects the condition

We have proven that the $\mathfrak{R}_0^{\bullet} = \mathfrak{R}_0 - \beta\Lambda/2d(d+\gamma)(d+\gamma+(1/2)\sigma_2^2)\sigma_2^2$ is a model (1) threshold for the illness to die out or endure, and noise intensities can modify the value of the stochastic reproduction number $\mathfrak{R}_0^{\bullet}$.

(ii) If $\mathfrak{R}_0^{\bullet} < 1$, the illness will be eradicated with a strong probability

(iii) If $\mathfrak{R}_0^{\bullet} > 1$, on the other hand, model (2) has a stationary distribution, indicating that the illness will dominate

(iv) Additionally, we have examined the numerical simulation of both deterministic and stochastic models that give a reasonable level of support for our examined technique

10. Remark

Comparing with the results in [23, 24], we observed that stochastic dynamics of fractional order are commonly demonstrated as nonrandom differential equation driven by fractional Brownian motion. On the other hand, our stochastic models are likely to provide various outcomes each time they are performed. Using random variables, our stochastic system indicates the probability of various outcomes under various circumstances. Our stochastic model offers information and forecasts results that take into account various degrees of randomness or inconsistency, and an abrupt change can be observed in (1).

There are still a number of intriguing aspects that we will discuss later. For instance, rapid climate change, weather warming or cooling, and wetness or evaporation may all have an impact on disease propagation. As a result, when a discontinuous random process, like variational noise, is added to model (1), how does it affect disease spread? This is something we will look into later.

Data Availability

No data were used to support this study.

Additional Points

Preprint Statement. This manuscript is not reposited on any preprint server.

Conflicts of Interest

The authors have no conflicts of interest regarding the publication of this paper.

Authors' Contributions

All the authors have equal contributions in this article.

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