Mathematical Biosciences and Engineering

http://www.aimspress.com/journal/mbe

MBE, 19(10): 10316–10331.
DOI: 10.3934/mbe.2022482
Received: 11 April 2022
Revised: 26 June 2022
Accepted: 05 July 2022
Published: 21 July 2022

Research article

Stochastic dynamics of influenza infection: Qualitative analysis and numerical results

Jehad Alzabut[1,2,*], Ghada Alobaidi[3], Shah Hussain[4], Elissa Nadia Madi[4] and Hasib Khan[5]

[1] Department of Mathematics and Sciences, Prince Sultan University, Riyadh 11586, Saudi Arabia

[2] Department of Industrial Engineering, OSTİM Technical University, Ankara 06374, Türkiye

[3] Department of Mathematics and Statistics, American University of Sharjah, PO Box 26666, Sharjah, United Arab Emirates

[4] Faculty of Informatics and Computing, Universiti Sultan Zainal Abidin (UniSZA), Besut Campus, Terengganu, Malaysia

[5] Department of Mathematics, Shaheed Benazir Bhutto University, Sheringal, Dir Upper, Khyber Pakhtunkhwa, Pakistan

* **Correspondence:** Email: jalzabut@psu.edu.sa, jehad.alzabut@ostimteknik.edu.tr.

Abstract: In this paper, a novel influenza $\mathcal{SI}_N\mathcal{I}_R\mathcal{R}$ model with white noise is investigated. According to the research, white noise has a significant impact on the disease. First, we explain that there is global existence and positivity to the solution. Then we show that the stochastic basic reproduction $\dot{\mathrm{R}}_r$ is a threshold that determines whether the disease is cured or persists. When the noise intensity is high, we get $\dot{\mathrm{R}}_r < 1$ and the disease goes away; when the white noise intensity is low, we get $\dot{\mathrm{R}}_r > 1$, and a sufficient condition for the existence of a stationary distribution is obtained, which suggests that the disease is still there. However, the main objective of the study is to produce a stochastic analogue of the deterministic model that we analyze using numerical simulations to get views on the infection dynamics in a stochastic environment that we can relate to the deterministic context.

Keywords: influenza; stochastic modeling; white noise; persistence and extinction of disease

1. Introduction

Recently, Whitman and Jayaprakash [1] published a study of a simple, stochastic, agent-based model of influenza infection, Infectious diseases have been and continue to be a major public-health problem [2], disrupting people's quality of life and reducing their chances of survival. The appearance of new diseases and the repetition of mutant strains have added to their massive unhelpful blow. The

control of the influenza virus, a foremost international health condition poses a scientific challenge at many levels. Influenza is caused by a virus. According to [3], it is characterized by a severe cytopathogenic respiratory disease that is infectious in nature. Based on matrix protein and nucleoprotein differences, it can be subdivide into A, B and C [4].

Humans and animals are both infected with Types A and B. The most virulent type, i.e., the Type A virus which is now known as one of the most problematic viruses to tackle [5] is further classified based on the hemagglutinin and neuraminidase proteins located on the virus's surface. Hemagglutinin H1 to H16 and neuraminidase N1 to N9 are the two groups of proteins, the combination of which classifies the influenza subtypes. Influenza A's nomenclature is determined by a mix of hemagglutinin and neuraminidase. The H1N1 virus, for example, is influenza A with both H1 and N1 proteins; its spread is usually not epidemic, and that is why it is difficult to distinguish it from a regular cold [6].

Another way to characterize them is by the influenza A and B strains [7]. New influenza strains emerge as a result of genetic changes or drift [8]. Antigenic drift is caused by progressive changes in the virus over time. Antibodies have a difficult time recognizing new strains. Antigenic shift, on the other hand, is characterized by fast changes in the virus that result in the emergence of a completely new strain. Influenza A can go through either of the modifications, whereas Influenza B only goes through antigenic drift [9, 10].

Vaccination is incapable of protecting against the new influenza strain. The 2009 H1N1 influenza pandemic showed this. Antivirals are thus required to halt the spread of the influenza outbreak [11]. Recently, incidences of influenza virus resistance have been discovered. The H3N2 virus resistance to aminoadamantanes and the H1N1 virus resistance to oseltamivir are two examples. Resistance is lethal and has the potential to create numerous pandemics in the future [12].

When compared to the original strain, the transmission rate of a new strain is thought to be quite low. The fact that mutation reduces viral strength is linked to this phenomenon [13]. In the instance of H1N1 influenza virus resistance to oseltamivir, however, it was discovered that these alterations do not always affect virus transmission [14, 15].

For at least the last several centuries, the influenza virus has been responsible for periodic outbreaks of acute febrile illness every 1 to 4 years. The first influenza like sickness outbreak was documented in 1173 and 1174 [16], while the first true epidemic occurred in 1694. [17]. Between 1918 and 1919, the globe was struck by the worst epidemic in recorded history, with an estimated 21 million victims [18]. As indicated by records, it was one of the most terrible events in mankind's set of experiences. Three additional pandemics occurred in the 20th century, namely the 1957 H2N2 pandemic, the 1968 H3N2 pandemic and the 2009 flu A (H1N1) infection (pH1N1) pandemic.

In the most famous model, a flu strain was found in Mexico and later in the United States of America with a blend of various qualities not recently seen in pig or human flu infection strains [19]. The pandemic was announced in August 2010 after the involvement of different zones [20].

Young people have the highest rates of influenza infection, while older adults have the highest fatality rates. Mortality and morbidity are particularly high for those with certain high risk medical conditions, such as extreme aging, cardiovascular disorders and metabolic diseases such as diabetes mellitus. During the 2009 pandemic, there was an elevated risk of influenza morbidity and mortality in pregnant women [21]. Furthermore, evidence from prior pandemic and seasonal influenza outbreaks suggests that the risk of influenza complications is higher in the second half of pregnancy than in the first.

To investigate the influence of environmental conditions on influenza transmission and make the results more realistic, we first developed a stochastic mathematical influenza model. Following that, the factors required for extinction and persistence were analyzed. The threshold of the suggested stochastic influenza model has also been established. When there is tiny or large noise, it plays a critical role in the mathematical models as a backbone [22, 23]. Finally, we visualized the numerical simulations using MATLAB.

2. Stochastic influenza model

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

- The total inhabitant $\aleph(t)$ is distributed in four compartment, i.e., $\mathcal{S}_t$, $\mathcal{I}_{N_t}$, $\mathcal{I}_{R_t}$ and $\mathcal{R}_t$, which represent the susceptible and infected people with resistance, infected peoples with non-resistance and recovered people respectively.
- The variables and parameters of the proposed stochastic model are non-negative.
- We deliberate that the variability of μ and γ is subject to stochastic white noise disturbance, i.e., $\mu \to \mu + \sigma_1 B_1(t)$ and $\gamma \to \gamma + \sigma_2 B_2$; where $B_1(t) and B_2(t)$ represent the Brownian motion with the property $B_1(0) = 0 = B_2(0)$ and the intensities $\sigma_1^2 and \sigma_2^2$ are positive.

Remark 2.1. *The deterministic general epidemic study estimates that if $\dot{R}_r < 1$, a small outburst will arise, and if $\dot{R}_r > 1$, a large outbreak will occur, infecting a large chunk of the populace. 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, this 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 $\dot{R}_r > 1$ and the society is huge, if the outbreak is started by only one (or a few) early infectives, the epidemic may never take off by accident. The formulation of a related stochastic epidemic model is motivated by these two aspects. It allows parameter estimations from disease outbreak data to include standard errors, and the subject of disease extinction is better suited for stochastic models for researching epidemic diseases.*

In light of the above speculations, we established the following new stochastic influenza model;

$$\begin{aligned} d\mathcal{S} &= (b - d\mathcal{S} - \mathcal{S}\mathcal{I}_N\alpha - \frac{\mathcal{S}\beta\mathcal{I}_R}{k\mathcal{I}_R + 1})dt, \\ d\mathcal{I}_N &= (\alpha\mathcal{S}\mathcal{I}_N - (\mu + d)\mathcal{I}_N) \quad dt - \sigma_1\mathcal{I}_N dB_1(t), \\ d\mathcal{I}_R &= (\frac{\beta\mathcal{S}\mathcal{I}_R}{k\mathcal{I}_R + 1} - (\gamma + d)\mathcal{I}_R)dt - \sigma_2\mathcal{I}_R dB_2(t), \\ d\mathcal{R} &= (\mu\mathcal{I}_N + \gamma\mathcal{I}_R - d\mathcal{R})dt + \sigma_1\mathcal{I}_N dB_1(t) + \sigma_2\mathcal{I}_R dB_2(t). \end{aligned} \tag{2.1}$$

Table 1. Parametric description of the model.

Symbol	Description	Value
α	The rate of infection by a non-resistant strain	0.15
β	The rate of infection by a resistant strain	0.10
γ	The rate at which resistant individuals are eliminated from the inhabitants	0.75
μ	Represents the pace at which non-resistant strains are evicted from the population	0.80
b	Denotes recruitment into the susceptible group	1.00
d	The death rate	0.20
k	The effect of mutation on the resistant strain	0.10

Also, we have the compartment table below:

Table 2. Compartments and description.

Symbol	Description	Value
$\mathcal{S}$	Susceptible	20
$\mathcal{I}_N$	Infected peoples with resistance	2
$\mathcal{I}_R$	Infected peoples with non-resistance	2
$\mathcal{R}$	Recovered peoples	1

The authors of [24] developed the following deterministic model:

$$\begin{aligned} \frac{d\mathcal{S}}{dt} &= b - d \quad \mathcal{S} - \alpha\mathcal{S}\mathcal{I}_N - \frac{\mathcal{S}\mathcal{I}_R\beta}{k\mathcal{I}_R + 1}, \\ \frac{d\mathcal{I}_N}{dt} &= \alpha\mathcal{S}\mathcal{I}_N - (d+\mu)\mathcal{I}_N, \\ \frac{d\mathcal{I}_R}{dt} &= \frac{\beta\mathcal{S}\mathcal{I}_R}{k\mathcal{I}_R + 1} - (d+\gamma)\mathcal{I}_R, \\ \frac{d\mathcal{R}}{dt} &= \mu\mathcal{I}_N + \gamma\mathcal{I}_R - d\mathcal{R}, \end{aligned} \tag{2.2}$$

and

$$d(\aleph_t) = b - d\aleph_t \tag{2.3}$$

where $\aleph_t = \mathcal{S}_t + \mathcal{I}_{N_t} + \mathcal{I}_{R_t} + \mathcal{R}_t$ indicates all the constant residents for $b \approx \mu\aleph$ and $\aleph_0 = \mathcal{S}_0 + \mathcal{I}_{N_0} + \mathcal{R}_0 + \mathcal{I}_{R_0}$. Equation (2.3) has the exact solution

$$\aleph_t = e^{-dt}[\aleph_0 + \frac{b}{d}e^{dt}] \tag{2.4}$$

Also we have $0 \geq \mathcal{I}_{N_0}, 0 \geq \mathcal{S}_0, \mathcal{I}_{R_0} \geq 0, \mathcal{R}_0 \geq 0 \Rightarrow \mathcal{I}_{N_t} \geq 0, \mathcal{S}_t \geq 0, \mathcal{I}_{R_t} \geq 0$ *and* $\mathcal{R}_t \geq 0$, so the result has a positivity property. If $\dot{\mathrm{R}}_r < 1$ then the model (2.2) will be locally stable and unstable otherwise. Similarly for $b = 0$ the model (2.2) will be globally asymptotically stable.

In this article, we will establish the computational and analytical aspects of the stochastic influenza model, and we will mathematically correlate our results with the deterministic model for future forecasts by using various parametric variables. As a result, this research can help the local community become more aware of the disease's spread.

3. Preliminaries

Here, we made the following speculations:

- $\mathbb{R}^{\mathrm{d}}_{+} = \{\varsigma \in \mathbb{R}^{\mathrm{d}} : 0 < \varsigma_i, \mathrm{d} > 1\}$.
- Suppose a complete probability space $(\Omega^{\circledast}, \Im^{\circledast}, \{\Im^{\circledast}_t\}_{t\geq 0}, \mathcal{P}^{\circledast})$ with filtration $\{\Im^{\circledast}_t\}_{t\geq 0}$ satisfies the usual condition.

We reflect a common four-dimensional stochastic differential equation for the existence of the solution of our model which is described by (2.1):

$$d\varsigma(t) \quad = \quad \Theta^{*}(\varsigma(t), t)dt) + \Theta(\varsigma(t), t)dB(t)), \quad for \quad t = t_0 \tag{3.1}$$

with the initial condition $\varsigma(t_0) = \varsigma_0 \in \mathbb{R}^{\mathrm{d}}$. By defining the differential operator $Ł^{\star}$ using (3.1), we get

$$Ł^{\star} \quad = \quad \frac{\partial}{\partial t} + \sum_{i=1}^{4} \Theta^{*}{}_{i}(\varsigma, t)\frac{\partial}{\partial \varsigma_{\,i}} + \frac{1}{2}\sum_{i,j=1}^{5}[\Theta^{\mathcal{T}}(\varsigma, t)\Theta^{\mathcal{T}}(\varsigma, t)]_{ij}(\frac{\partial^2}{\partial\varsigma_i\partial\varsigma_j}). \tag{3.2}$$

If $Ł^{\star}$ acts on the function $\mathcal{V}^{\star} = (\mathbb{R}^{\mathrm{d}} \times \tilde{\mathbb{R}}_{+}; \tilde{\mathbb{R}}_{+})$, then

$$Ł^{\star}\mathcal{V}^{\star}(\varsigma, t) \quad = \quad \mathcal{V}^{\star}_{t}(\varsigma, t) + \mathcal{V}_{\varsigma}(\varsigma, t)\Theta^{*}(\varsigma, t) + \frac{1}{2}trace[\Theta^{\mathcal{T}}(\varsigma, t)\mathcal{V}^{\star}_{\varsigma\varsigma}(\varsigma, t)\Theta(\varsigma, t)]. \tag{3.3}$$

4. Existence and uniqueness

In this section, our discussion will be on the solution of the stochastic influenza model (2.1).

Theorem 4.1. *There is a unique positive solution* $(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t)$ *of the system* (2.1) *for* $t \geq 0$ *with* $(\mathcal{I}_{N_0}, \mathcal{S}_0, \mathcal{I}_{R_0}, \mathcal{R}_0) \in \mathbb{R}^4_{+}$*, and the solution will be left in* $\mathbb{R}^4_{+}$ *, with a probability equal to one.*

Proof. Because (2.1) satisfies the local Lipschitz condition [25], formally for $(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t) \in \mathbb{R}^4_{+}$, we do have $(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t) \in \mathbb{R}^4_{+}$ a distinctive local solution on $t \in [0, \tau_e)$, where τ_e is the flare-up time. Next, our aim is to show $\tau_e = \infty$ for the global solution of (2.1). Assume $0 \leq \ell_0$ is very large so that $(\mathcal{I}_{N_0}, \mathcal{S}_0, \mathcal{I}_{R_0}, \mathcal{R}_0)$ lies in $[\frac{1}{\ell_0}, \ell_0]$. For $\ell_0 \leq \ell$, define

$$\tau_\ell \quad = \quad inf\{t \in [0, \tau_e) : \frac{1}{\ell} \geq min\{(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t)\} \text{ or } \ell \leq max\{(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t)\}\}.$$

Let $\inf\emptyset = \infty$ (because typically $\emptyset$ is the empty set). Since τ_ℓ is increasing for $\ell \to \infty$, let $\tau_\infty = lim_{\ell\to\infty}\tau_\ell$; then, we have $\tau_\infty \leq \tau_e$ almost surely. Next, we need to confirm $\tau_\infty = \infty$ a.s. If this assertion is incorrect, then there exist a constant $\mathcal{T} > 0$ and $\in (0, 1)$ such that $\mathbb{P}\{\tau_\infty \leq \mathcal{T}\} > \in$. As a consequence, we have $\ell_1 \geq \ell_0$ such that

$$\mathbb{P}\{\tau_\ell \leq \mathcal{T}\} \geq \in, \forall k \geq \ell_1 \quad \text{for} \quad t \leq \tau_\ell. \tag{4.1}$$

We outline a $\mathbb{C}^2$-function $U : \mathbb{R}^4_{+} \to \mathbb{R}_{+}$ by using the resulting formulation

$$U(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t) \quad = \quad (\mathcal{S}_t - \underline{\tilde{c}} - \underline{\tilde{c}}ln\frac{\mathcal{S}_t}{\underline{\tilde{c}}}) + (\mathcal{I}_{N_t} - (\frac{3}{4} + \frac{1}{4}) - ln\mathcal{I}_{N_t}) + (\mathcal{I}_{R_t} - (\frac{3}{4} + \frac{1}{4}) - ln\mathcal{I}_{R_t})$$

$$+ \quad (\mathcal{R}_t - (\frac{3}{4} + \frac{1}{4}) - ln\mathcal{R}_t). \tag{4.2}$$

Obviously the function U is non-negative which can follow from $z - (\frac{3}{4} + \frac{1}{4}) - \log z \geq 0 \quad \forall \quad z > 0$. Suppose $\ell \geq \ell_0$ and $\mathcal{T} \geq 0$ are arbitrary. Applying Ito's formula to (4.2) we get

$$\begin{aligned} dU(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t) &= (1 - \frac{1}{\mathcal{I}_{N_t}})d\mathcal{I}_{N_t} + \frac{1}{2\mathcal{I}_{N_t}^2}(d\mathcal{I}_{N_t})^2 + (1 - \frac{1}{\mathcal{I}_{R_t}})d\mathcal{I}_{R_t} + \frac{1}{2\mathcal{I}_{R_t}^2}(d\mathcal{I}_{R_t})^2 \\ &+ (1 - \frac{1}{\mathcal{R}_t})d\mathcal{R}_t + \frac{1}{2\mathcal{R}_t^2}(d\mathcal{R}_t)^2 + (1 - \frac{\tilde{c}}{\mathcal{S}_t})d\mathcal{S}_t \qquad (4.3) \\ &= \text{Ł}^\star U(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t) + \sigma_1(\mathcal{I}_{N_t} - \mathcal{S}_t)dB_1(t) + \sigma_2(\mathcal{I}_{R_t} - \mathcal{S}_t)dB_2(t), \qquad (4.4) \end{aligned}$$

where $\text{Ł}^\star U : \mathbb{R}_+^4 \to \mathbb{R}_+$ is defined by

$$\begin{aligned} \text{Ł}^\star U(\mathcal{I}_{N_t}, \mathcal{S}_t, \mathcal{I}_{R_t}, \mathcal{R}_t) &= (1 - \frac{\tilde{c}}{\mathcal{S}_t})(b - d\mathcal{S}_t - \alpha\mathcal{S}_t\mathcal{I}_{N_t} - \frac{\mathcal{S}_t\beta\mathcal{I}_R}{1 + k\mathcal{I}_{R_t}}) \\ &+ (1 - \frac{1}{\mathcal{I}_{N_t}})(\alpha\mathcal{S}_t\mathcal{I}_{N_t} - (d + \mu)\mathcal{I}_{N_t}) + \frac{1}{2}\sigma_1^2 \\ &+ (1 - \frac{1}{\mathcal{I}_{R_t}})(\frac{\beta\mathcal{S}_t\mathcal{I}_{R_t}}{1 + k\mathcal{I}_{R_t}} - (d + r)\mathcal{I}_{R_t}) + \frac{1}{2}\sigma_2^2 \\ &+ (1 - \frac{1}{\mathcal{R}_t})(\mu\mathcal{I}_{N_t} + \gamma\mathcal{I}_{R_t} - d\mathcal{R}_t) + \frac{1}{2}\sigma_1^2 + \frac{1}{2}\sigma_2^2 \\ &= b - d\mathcal{S}_t - \alpha\mathcal{S}_t\mathcal{I}_{N_t} - \frac{\beta\mathcal{S}_t\mathcal{I}_{R_t}}{1 + k\mathcal{I}_{R_t}} - \frac{\tilde{c}b}{\mathcal{S}_t} + \underline{\tilde{c}}d \\ &+ \underline{\tilde{c}}\alpha\mathcal{I}_{N_t} + \frac{\tilde{c}\beta\mathcal{I}_{R_t}}{1 + k\mathcal{I}_{R_t}} + \alpha\mathcal{S}_t\mathcal{I}_{N_t} + (d + \gamma)\mathcal{I}_{N_t} \\ &- \alpha\mathcal{S}_t + (d + \mu) + \frac{\mathcal{S}_t\beta\mathcal{I}_{R_t}}{1 + k\mathcal{I}_{R_t}} - (d + \gamma)\mathcal{I}_{R_t} + (d + \gamma) \\ &- \frac{\beta\mathcal{S}_t}{1 + k\mathcal{I}_{R_t}} + \mu\mathcal{I}_{N_t} + \gamma\mathcal{I}_{R_t} - d\mathcal{R}_t - \gamma + d - \frac{\mu\mathcal{I}_{N_t}}{\mathcal{R}_t} \\ &+ \sigma_1^2 + \sigma_2^2 \\ &\leq b - d\mathcal{S}_t - \alpha\mathcal{S}_t\mathcal{I}_{N_t} + \underline{\tilde{c}}d + \underline{\tilde{c}}\alpha\mathcal{I}_{N_t} + \alpha\mathcal{S}_t\mathcal{I}_{N_t} \\ &+ (d + \gamma)\mathcal{I}_{N_t} - \alpha\mathcal{S}_t + (2d + \mu + \gamma) - d\mathcal{I}_{R_t}\gamma\mathcal{I}_{R_t} \\ &+ \mu\mathcal{I}_{N_t} + \gamma\mathcal{I}_{R_t} - d\mathcal{R}_t - \gamma + d + \sigma_1^2 + \sigma_2^2 \\ &\leq b + \underline{\tilde{c}}d + \underline{\tilde{c}}\alpha\mathcal{I}_{N_t} + \alpha\mathcal{S}_t\mathcal{I}_{N_t} + (d + \gamma)\mathcal{I}_{N_t} + (d + \gamma) \\ &+ \gamma\mathcal{I}_{R_t} + \mu\mathcal{I}_{N_t} + d + \sigma_1^2 + \sigma_2^2 \triangleq B^*. \qquad (4.5) \end{aligned}$$

Thus, we have

$$\begin{aligned} \bar{E}^*[U(\mathcal{S}_t(\tau_J \wedge \curlywedge), (\mathcal{I}_{N_t}(\tau_J \wedge \curlywedge), (\mathcal{I}_{R_t}(\tau_J \wedge \curlywedge), (\mathcal{R}(\tau_J \wedge \curlywedge)] &\leq U(\mathcal{S}_0, \mathcal{I}_{N_0}, \mathcal{I}_{R_0}, \mathcal{R}_0 \\ &+ \bar{E}^*[\int_0^{\tau_J \wedge \curlywedge} \mathbb{K}dt] \\ &\leq U(\mathcal{S}_0, \mathcal{I}_{N_0}, \mathcal{I}_{R_0}, \mathcal{R}_0) + \mathbb{K} \curlywedge . \qquad (4.6) \end{aligned}$$

We consider that $\Omega_\jmath = \{\sigma_\jmath \leq \curlywedge\}$ for all $\jmath \geq \jmath_1$ and by (2.3) , $P(\Omega_\jmath) \geq \epsilon$. We comment that for every $\omega \in \Omega_\jmath$ there exist at least $\mathcal{S}_t(\tau_\jmath, \omega), \mathcal{I}_{N_t}(\tau_\jmath, \omega), \mathcal{I}_{R_t}(\tau_\jmath, \omega)$ *and* $\mathcal{R}(\tau_\jmath, \omega)$, equaling the value $\jmath$ or $\frac{1}{\jmath}$ then we get that $U(\mathcal{S}_t(\tau_\jmath), \mathcal{I}_{N_t}(\tau_\jmath), \mathcal{I}_{R_t}(\tau_\jmath), \mathcal{R}(\tau_\jmath))$ is not less than $\jmath - 1 - \log \jmath$ or $(\frac{1}{\jmath}) - 1 + \log \jmath$. Accordingly,

$$U(\mathcal{S}_t(\tau_\jmath), \mathcal{I}_{N_t}(\tau_\jmath), \mathcal{I}_{R_t}(\tau_\jmath), \mathcal{R}(\tau_\jmath)) \geq \bar{E}^*(\jmath - 1 - \log \jmath) \wedge (\frac{1}{\jmath}) - 1 + \log \jmath). \tag{4.7}$$

From (4.1) and (4.6), we get the following relation

$$\begin{aligned} U(\mathcal{S}_0, \mathcal{I}_{N_0}, \mathcal{I}_{R_0}, \mathcal{R}_0) + \mathbb{K}\curlywedge &\geq \bar{E}^*[1_{\Omega_\jmath} U(\mathcal{S}_t(\tau_\jmath), \mathcal{I}_{N_t}(\tau_\jmath), \mathcal{I}_{R_t}(\tau_\jmath), \mathcal{R}_t(\tau_\jmath))] \\ &\geq \xi[(\jmath - 1 - \log \jmath) \wedge ((\frac{1}{\jmath}) - 1 + \log \jmath)], \end{aligned} \tag{4.8}$$

where $1_{\Omega_\jmath}$ denotes the indicator function. We observe that $k \to \infty$ leads to the ambiguity $\infty > U(\mathcal{S}_0, \mathcal{I}_{N_0}, \mathcal{I}_{R_0}, \mathcal{R}_0) + \mathcal{M}'\curlywedge = \infty$, which implies that $\tau_\infty = \infty$ a.s. □

5. Long time behavior of the system (2.1)

In this part, we determine when the sickness will be cured and when it will be revived. As a result, the system's (2.1) vital reproduction is demonstrated. Based on the proof in [26], we can deduce the subsequent lemmas:

Lemma 5.1. *Let* $(\mathcal{S}_t, \mathcal{I}_{N_t}, \mathcal{I}_{R_t}, \mathcal{R}_t)$ *be the solution of the model* (2.1) *with the initial values given by* $(\mathcal{S}_0, \mathcal{I}_{N_0}, \mathcal{I}_{R_0}, \mathcal{R}_0) \in \mathbb{R}^4_+$, *; then,* $\lim_{t\to\infty} \frac{\mathcal{I}_{N_t} + \mathcal{S}_t + \mathcal{I}_{R_t} + \mathcal{R}_t}{t} = 0$ *is almost certain.*

6. Remark

In fact, together with the positivity of the solution and the system (2.1), we have that $\lim_{t\to\infty} \frac{\mathcal{S}_t}{t} = 0$ $\lim_{t\to\infty} \frac{\mathcal{I}_{N_t}}{t} = 0$, $\lim_{t\to\infty} \frac{\mathcal{I}_{R_t}}{t} = 0$ and $\lim_{t\to\infty} \frac{\mathcal{R}_t}{t} = 0$ a.s.

Lemma 6.1. *Suppose* $d > (\frac{1}{2}\sigma_1^2 \vee \frac{1}{2}\sigma_2^2)$. *Assume* $(\mathcal{S}_t, \mathcal{I}_{N_t}, \mathcal{I}_{R_t}, \mathcal{R}_t)$ *is the solution of the model* (2.1) *with initial values given by* $(\mathcal{S}_0, \mathcal{I}_{N_0}, \mathcal{I}_{R_0}, \mathcal{R}_0) \in \mathbb{R}^4_+$*; then,*

$$\lim_{t\to\infty} \frac{\int_0^t \mathcal{I}_R(s) dB_2(s)}{t} = 0, \quad \lim_{t\to\infty} \frac{\int_0^t \mathcal{I}_N(s) dB_1(s)}{t} = 0. \tag{6.1}$$

Let

$$\mathcal{R}_r^\star = \frac{\alpha b}{d(d + \mu + \frac{1}{2}\sigma_1^2)} = \mathcal{R}_r - \frac{\alpha b}{2d(d + \mu)(d + \mu + \frac{1}{2}\sigma_1^2)} \sigma_1^2,$$

where

$$\mathcal{R}_r = \frac{\alpha b}{d(d + \mu)} \tag{6.2}$$

is the deterministic model's fundamental reproduction number.

7. Extinction of the disease

In this segment, we scrutinize the condition for the disappearance of the influenza model (2.1), we lead with the following representation and definition. Let $\langle \jmath(t)\rangle = \frac{1}{t}\int_0^t \jmath(r)dr$, then the following are the outcomes for the disease's termination.

Theorem 7.1. *Let $(\mathcal{S}_t, \mathcal{I}_{N_t}, \mathcal{R}, \mathcal{I}_{R_t})$ be the solution of the stochastic influenza model* (2.1)*, with the initial values given by $(\mathcal{S}_0, \mathcal{I}_{N_0}, \mathcal{R}_0, \mathcal{I}_{R_0}) \in \Omega^{\odot}$. If $R_r < 1$ then $\lim_{t\to\infty}(\frac{\log \mathcal{I}_{N_t}}{t}) < 0$ and $\lim_{t\to\infty}(\frac{\log \mathcal{I}_{R_t}}{t}) < 0$; almost surely, $\mathcal{I}_{N_t} \to 0$ and $\mathcal{I}_{R_t} \to 0$ exponentially a.s which means that the disease terminates with a probability of one). Also $\lim_{t\to\infty}\int_0^t \mathcal{S}_t = (\frac{b}{d})$, $\lim_{t\to\infty}\mathcal{I}_{N_t}(t)(t) = 0$, $\lim_{t\to\infty}\mathcal{I}_{R_t}(t) = 0$ and $\lim_{t\to\infty}\int_0^t \mathcal{R}_t(t) = 0$.*

Proof. After integrating (2.1), we can get the following system of equations

$$\begin{aligned}
\frac{\mathcal{S}_t - \mathcal{S}_0}{t} &= b - d\langle \mathcal{S}_t\rangle - \alpha\langle \mathcal{S}_t\mathcal{I}_{N_t}\rangle - \frac{\beta\langle \mathcal{S}_t\mathcal{I}_{R_t}\rangle}{1+k\langle \mathcal{I}_{R_t}\rangle},\\
\frac{\mathcal{I}_{N_t} - \mathcal{I}_{N_0}}{t} &= \alpha\langle \mathcal{S}_t\mathcal{I}_{N_t}\rangle - (d+\mu)\langle \mathcal{I}_{N_t}\rangle - \frac{1}{t}\sigma_1\int_0^t \mathcal{I}_{N_t}(r)dB_1(r),\\
\frac{\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t} &= \frac{\beta\langle \mathcal{S}_t\mathcal{I}_{R_t}\rangle}{1+k\langle \mathcal{I}_{R_t}\rangle} - (d+\gamma)\langle \mathcal{I}_{R_t}\rangle - \frac{1}{t}\sigma_2\int_0^t \mathcal{I}_{R_t}(r)dB_2(r),\\
\frac{\mathcal{R}_t - \mathcal{R}_0}{t} &= \mu\langle \mathcal{I}_{N_t}\rangle + \gamma\langle \mathcal{I}_{R_t}\rangle - d\langle \mathcal{R}\rangle + \frac{1}{t}\sigma_1\int_0^t \mathcal{I}_{N_t}(r)dB_1(r)\\
&+ \frac{1}{t}\sigma_2\int_0^t \mathcal{I}_{R_t}(r)dB_2(r),
\end{aligned} \tag{7.1}$$

$$\begin{aligned}
\frac{\mathcal{S}_t - \mathcal{S}_0}{t} + \frac{\mathcal{I}_{N_t} - \mathcal{I}_{N_0}}{t} + \frac{\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t} + \frac{\mathcal{R}_t - \mathcal{R}_0}{t} &= b - d\langle \mathcal{S}_t\rangle - d\langle \mathcal{I}_{N_t}\rangle\\
&- d\langle \mathcal{I}_{R_t}\rangle - d\langle \mathcal{R}\rangle,
\end{aligned} \tag{7.2}$$

$$\langle \mathcal{S}_t\rangle = \frac{b}{d} - \langle \mathcal{I}_{N_t}\rangle - \langle \mathcal{I}_{R_t}\rangle - \langle \mathcal{R}_t\rangle + \Phi(t), \tag{7.3}$$

where

$$\Phi(t) = -\frac{1}{d}\Big[\frac{\mathcal{S}_t - \mathcal{S}_0}{t} + \frac{\mathcal{I}_{N_t} - \mathcal{I}_{N_0}}{t} + \frac{\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t} + \frac{\mathcal{R}_t - \mathcal{R}_0}{t}\Big] \tag{7.4}$$

Obviously $\Phi(t) \to 0$ as $t \to \infty$. Applying Ito's formula to the second equation of (2.1) gives

$$d\log \mathcal{I}_{N_t} = (\alpha\mathcal{S}_t - (d+\mu) + \frac{1}{2}\sigma_1^2)dt - \sigma_1 dB_1(t) \tag{7.5}$$

If we Integrate (7.5) from 0 to t and divide by t, we get

$$\frac{\log \mathcal{I}_{N_t} - \log \mathcal{I}_{N_0}}{t} = \alpha\langle \mathcal{S}_t\rangle - (d+\mu) + \frac{1}{2}\sigma_1^2 - \frac{1}{t}\sigma_1\int_0^t dB_1(r) \tag{7.6}$$

Substituting (7.3) in (7.6), we have

$$\begin{aligned}\frac{\log \mathcal{I}_{N_t} - \log \mathcal{I}_{N_0}}{t} &= \alpha\Big[\frac{b}{d} - \langle \mathcal{I}_{N_t}\rangle - \langle \mathcal{I}_{R_t}\rangle - \langle \mathcal{R}_t\rangle + \Phi(t)\Big] \\ &- (d+\mu) + \frac{1}{2}\sigma_1^2 - \frac{1}{t}\sigma_1 \int_0^t dB_1(r) \\ &\leq \frac{\alpha b}{d} - (d+\mu) + \frac{1}{2}\sigma_1^2 - \alpha\langle \mathcal{I}_{N_t}\rangle - \alpha\langle \mathcal{I}_{R_t}\rangle - \alpha\langle \mathcal{R}_t\rangle - \frac{1}{t}\sigma_1 \int_0^t dB_1(r) + \Phi(t) \\ &= -(d+\mu-\frac{1}{2}\sigma_1^2)(1-\mathcal{R}_r^*) - \alpha\langle \mathcal{I}_{N_t}\rangle - \alpha\langle \mathcal{I}_{R_t}\rangle - \alpha\langle \mathcal{R}_t\rangle + \urcorner, \end{aligned} \tag{7.7}$$

where

$$\urcorner = \Phi(t) - \frac{1}{t}\sigma_1 \int_0^t dB_1(r). \tag{7.8}$$

For $\urcorner = 0$ and $t \to \infty$, we have

$$\limsup_{t\to\infty} \frac{\log \mathcal{I}_{N_t}}{t} \leq -(d+\mu-\frac{1}{2}\sigma_1^2)(1-\mathcal{R}_r^\star) - \alpha\langle \mathcal{I}_{N_t}\rangle - \alpha\langle \mathcal{I}_{R_t}\rangle - \alpha\langle \mathcal{R}_t\rangle. \tag{7.9}$$

Equation (7.9) implies that

$$\lim_{t\to\infty} \mathcal{I}_{N_t} = 0. \tag{7.10}$$

Similarly, it may also be proved that

$$\lim_{t\to\infty} \mathcal{I}_{R_t} = 0. \tag{7.11}$$

Now for $\mathcal{S}_t$, we have the following from the first equation of the model (2.1)

$$\begin{aligned}\frac{\mathcal{S}_t - \mathcal{S}_0}{t} &= b - d\int_0^t \mathcal{S}_t dt - \alpha\int_0^t \mathcal{S}_t\mathcal{I}_{N_t} dt - \int_0^t \frac{\beta\mathcal{S}_t\mathcal{I}_{N_t}}{1+k\mathcal{I}_{R_t}}, \\ d\int_0^t \mathcal{S}_t dt &= b - \alpha\int_0^t \mathcal{S}_t\mathcal{I}_{N_t} dt - \int_0^t \frac{\beta\mathcal{S}_t\mathcal{I}_{N_t}}{1+k\mathcal{I}_{R_t}} - \frac{\mathcal{S}_t - \mathcal{S}_0}{t}, \\ \int_0^t \mathcal{S}_t dt &= \frac{b}{d} - \frac{\alpha}{d}\int_0^t \mathcal{S}_t\mathcal{I}_{N_t} dt - \int_0^t \frac{\frac{\beta}{d}\mathcal{S}_t\mathcal{I}_{N_t}}{1+k\mathcal{I}_{R_t}} - \frac{1}{b}(\frac{\mathcal{S}_t - \mathcal{S}_0}{t}). \end{aligned} \tag{7.12}$$

This implies that $\lim_{t\to\infty} \int_0^t \mathcal{S}_t = \frac{b}{d}$. Now from the fourth equation of the system (2.1), it follows that

$$\mathcal{R}_t = e^{-dt}\Big[\mathcal{R}_0 + \int_0^t \mu\mathcal{I}_N(r)e^{dt} + \int_0^t \gamma\mathcal{I}_R(r)e^{dt}\Big]. \tag{7.13}$$

By applying the L'Hospital's rule to the above result, we get

$$\lim_{t\to\infty} \int_0^t \mathcal{R} dt = 0, \tag{7.14}$$

which completes the proof. □

8. Persistence of the disease

In this section, we will investigate the necessary conditions for the persistence of the disease.

Theorem 8.1. *Assume $d > (\frac{1}{2}\sigma_1^2 \vee \frac{1}{2}\sigma_2^2)$. Let $(\mathcal{S}_t, \mathcal{I}_{N_t}, \mathcal{I}_{R_t}, \mathcal{R}_t)$ be the solution of the stochastic influenza model* (2.1)*, with the initial values given by $(\mathcal{S}_0, \mathcal{I}_{N_0}, \mathcal{I}_{R_0}, \mathcal{R}_0) \in \mathbb{R}_+^4$. If $R_r^\star > 1$, then*

$$\begin{aligned}
&\lim_{t\to\infty} \int_0^t \mathcal{S}(s)ds = \frac{b}{d\mathcal{R}_r^\star} \quad a.s,\\
&\lim_{t\to\infty} \int_0^t \mathcal{I}_N(s)ds = \frac{d(d+\mu+\frac{1}{2}\sigma_1^2)}{\alpha(d+\mu)}(\mathcal{R}_r^\star - 1) \quad a.s.,\\
&\lim_{t\to\infty} \int_0^t \mathcal{R}(s)ds = \frac{\mu(d+\mu+\frac{1}{2}\sigma_1^2)}{\alpha(d+\mu)}(\mathcal{R}_r^\star - 1) \quad a.s.,\\
&\lim_{t\to\infty} \int_0^t \mathcal{I}_R(s)ds = \left[\frac{d(d+\mu+\frac{1}{2}\sigma_1^2)((d+\mu+\frac{1}{2}\sigma_1^2) - (d+\gamma+\frac{1}{2}\sigma_1^2)\alpha d))}{\alpha dk(d+\gamma(b\alpha - d(d+\mu+\frac{1}{2}\sigma_1^2)))}\right](\mathcal{R}_r^\star - 1) \quad a.s.
\end{aligned}$$

Proof. If $\mathcal{R}_r^\star > 1$, then by (7.9) and Lemmas 5.1 and 5.2 in [27], we have

$$\lim_{t\to\infty} \int_0^t \mathcal{I}_N(s)ds = \frac{\frac{b\alpha}{d} - (d+\mu+\frac{1}{2}\sigma_1^2)}{\frac{\alpha(d+\mu)}{d}} = \frac{d(d+\mu+\frac{1}{2}\sigma_1^2)}{d+\mu}(\mathcal{R}_r^\star - 1). \tag{8.1}$$

$$\lim_{t\to\infty} \int_0^t \mathcal{S}(s)ds = \frac{b}{d} - \frac{d+\mu+\frac{1}{2}\sigma_1^2}{\alpha}(\mathcal{R}_r^\star - 1) = \frac{b}{d\mathcal{R}_r^\star}.$$

Now from the fourth equation of model (2.1), we obtain

$$\begin{aligned}
\frac{\mathcal{R}_t - \mathcal{R}_0}{t} &= \frac{\mu}{t}\int_0^t \mathcal{I}_N(s)ds + \frac{\gamma}{t}\int_0^t \mathcal{I}_R(s)ds - \frac{d}{t}\int_0^t \mathcal{R}(s)ds\\
&+ \frac{\sigma_1}{t}\int_0^t \mathcal{I}_N(s)dB_1(s) + \frac{\sigma_2}{t}\int_0^t \mathcal{I}_R(s)dB_1(s),
\end{aligned}$$

$$\frac{1}{t}\int_0^t \mathcal{R}(s)ds = \frac{\mu}{t}\int_0^t \mathcal{I}_N(s)ds + \eth, \tag{8.2}$$

where

$$\begin{aligned}
\eth &= \frac{\gamma}{t}\int_0^t \mathcal{I}_R(s)ds - \frac{d}{t}\int_0^t \mathcal{R}(s)ds + \frac{\sigma_1}{t}\int_0^t \mathcal{I}_N(s)dB_1(s)\\
&+ \frac{\sigma_2}{t}\int_0^t \mathcal{I}_R(s)dB_1(s) - \frac{\mathcal{R}_t - \mathcal{R}_0}{t},
\end{aligned}$$

$\eth(t)$ has the property that

$$\lim_{t\to\infty} \eth = 0. \tag{8.3}$$

By substituting (8.1) in (8.2), we have

$$\lim_{t\to\infty}\int_0^t \mathcal{R}(s)ds = \frac{\mu(d+\mu+\frac{1}{2}\sigma_1^2)}{\alpha(d+\mu)}(\mathcal{R}_r^\star - 1).$$

Now from the third equation of the model (2.1), using Ito's formula yields

$$d(ln\mathcal{I}_{R_t} - k\mathcal{I}_{R_t}) = \left[\beta\mathcal{S}_t - (d+\gamma) - k(d+\gamma)\mathcal{I}_{R_t} - \frac{1}{2}\sigma_2^2\right]dt + \sigma_2 dB_2(s). \tag{8.4}$$

Integrating (8.4) from 0 to t, we have

$$\begin{aligned}
\frac{\ln\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t} + k(\frac{\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t}) &= \frac{\beta}{t}\int_0^t \mathcal{S}(s)ds - (d+\gamma+\frac{1}{2}\sigma_2^2) \\
&- \frac{k(d+\gamma)}{t}\int_0^t \mathcal{I}_R(s)ds + \frac{1}{t}\int_0^t \sigma_2 dB_2(s) \\
&= \frac{b\beta}{d\mathcal{R}_r^\star} - \frac{k(d+\gamma)}{t}\int_0^t \mathcal{I}_R(s)ds + \frac{1}{t}\int_0^t \sigma_2 dB_2(s) \\
&- (d+\gamma) - \frac{1}{2}\sigma_2^2 \\
\frac{k(d+\gamma)}{t}\int_0^t \mathcal{I}_R(s)ds &= \frac{b\beta}{d\mathcal{R}_r^\star} + \frac{1}{t}\int_0^t \sigma_2 dB_2(s) - (d+\gamma) \\
&- \frac{1}{2}\sigma_2^2 - \left[\frac{\ln\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t} + k(\frac{\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t})\right],
\end{aligned}$$

$$\frac{1}{t}\int_0^t \mathcal{I}_R(s)ds = \frac{1}{k(d+\gamma)}\left[\frac{b\beta}{d\mathcal{R}_r^\star} - \frac{1}{2}\sigma_2^2 - (d+\gamma) + \wedge(s)\right], \tag{8.5}$$

where $\wedge(s) = \frac{1}{t}\int_0^t \sigma_2 dB_2(s) - (\frac{\ln\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t} + k(\frac{\mathcal{I}_{R_t} - \mathcal{I}_{R_0}}{t})$; $\eth(t)$ has the property that

$$\lim_{t\to\infty}\wedge(s) = 0. \tag{8.6}$$

Taking the limit of (8.5) and incorporating the value $\mathcal{R}_r^\star$ we have

$$\lim_{t\to\infty}\int_0^t \mathcal{I}_R(s)ds = \left[\frac{d(d+\mu+\frac{1}{2}\sigma_1^2)((d+\mu+\frac{1}{2}\sigma_1^2) - (d+\gamma+\frac{1}{2}\sigma_1^2)\alpha d))}{\alpha dk(d+\gamma(b\alpha - d(d+\mu+\frac{1}{2}\sigma_1^2)))}\right](\mathcal{R}_r^\star - 1) \quad a.s.$$

This completes the proof. □

9. Numerical scheme and results

We have accomplished our analysis of disease extinction and persistence. We will now perform some numerical simulations of (2.1) to illustrate the applicability of our findings. The Milstein technique [28] was used to generate the numerical simulations. Consider the model's discretization equation:

$$\mathcal{S}_{k+1} = \mathcal{S}_k + (b - d\mathcal{S}_k - \alpha\mathcal{S}_k\mathcal{I}_{N_k} - \frac{\beta\mathcal{S}_k\mathcal{I}_{R_k}}{1+k\mathcal{I}_{R_k}})\Delta t,$$

$$
\begin{aligned}
\mathcal{I}_{N_{k+1}} &= \mathcal{I}_{N_k} + (\alpha \mathcal{S}_k \mathcal{I}_{N_k} - (d+\mu)\mathcal{I}_{N_k})\Delta t - \sigma_1 \mathcal{I}_{N_k}\sqrt{\Delta t}\tau_k - \frac{\sigma_1^2}{2}\mathcal{I}_{N_k}(\tau_k^2 - 1)\Delta t,\\
\mathcal{I}_{R_{k+1}} &= \mathcal{I}_{R_k} + (\frac{\beta \mathcal{S}_k \mathcal{I}_{R_k}}{1 + k\mathcal{I}_{R_k}} - (d+\gamma)\mathcal{I}_{R_k})\Delta t - \sigma_2 \mathcal{I}_{R_k}\sqrt{\Delta t}\tau_k - \frac{\sigma_2^2}{2}\mathcal{I}_{R_k}(\tau_k^2 - 1)\Delta t,\\
\mathcal{R}_{k+1} &= \mathcal{R}_k + (\mu \mathcal{I}_{N_k} + \gamma \mathcal{I}_{R_k} - d\mathcal{R}_k)\Delta t + \sigma_1 \mathcal{I}_{N_k}\sqrt{\Delta t}\tau_k + \frac{\sigma_1^2}{2}\mathcal{I}_{N_k}(\tau_k^2 - 1)\Delta t,\\
&+ \sigma_2 \mathcal{I}_{R_k}\sqrt{\Delta t}\tau_k + \frac{\sigma_2^2}{2}\mathcal{I}_{R_k}(\tau_k^2 - 1)\Delta t.
\end{aligned}
$$

Here, we shall discuss the graphical description of the model (2.1). In Figure 1, we have illustrated a numerical solution of the model (2.1) and those obtained in comparative studies of all the classes for the white noise values $\sigma_1 = \sigma_2 = 0.0, 0.05, 0.10, 0.12$ and $S(0) = 20$, $I_N(1) = 2$, $I_R(1) = 2$, $R(1) = 1$, $b = 1.00$, $d = 0.20$, $\alpha = 0.15$, $\mu = 0.80$, $\beta = 0.10$, $\gamma = 0.75$, *and* $k = 0.10$. In Figure 2, we have

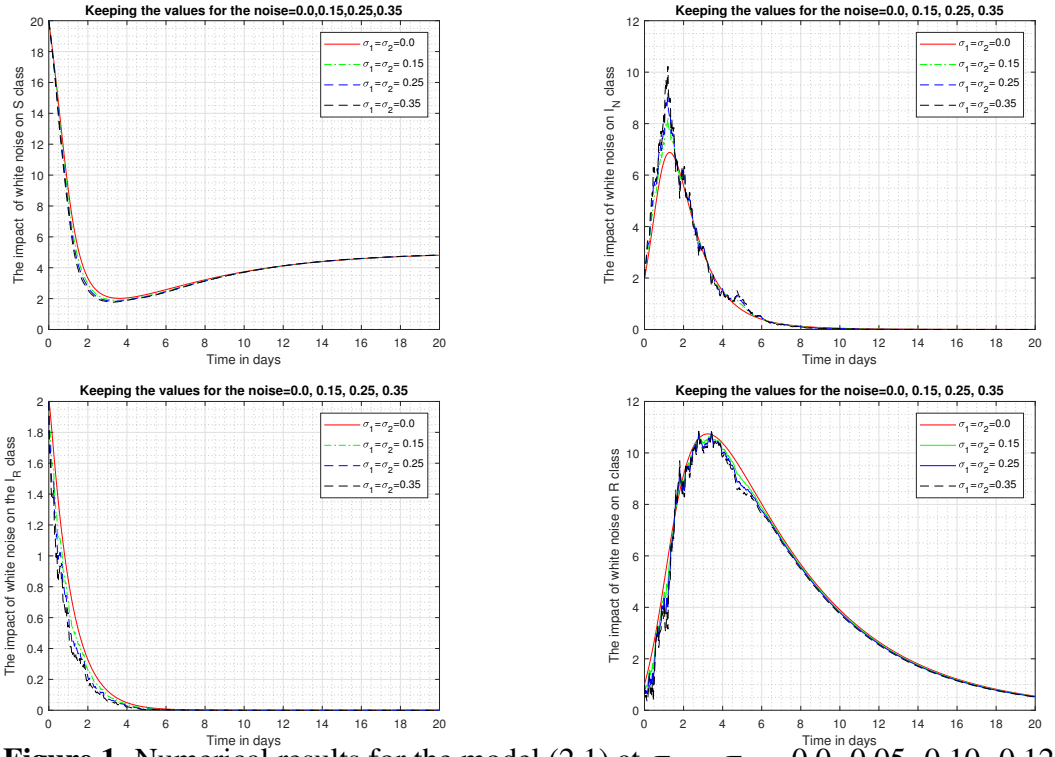

Figure 1. Numerical results for the model (2.1) at $\sigma_1 = \sigma_2 = 0.0, 0.05, 0.10, 0.12$.

illustrated a numerical solution of the model (2.1) and those obtained in comparative studies of all the classes for the white noise values $\sigma_1 = \sigma_2 = 0.0, 0.05, 0.10, 0.12$ and $S(0) = 20$, $I_N(1) = 2$, $I_R(1) = 2$, $R(1) = 1$, $b = 1.00$, $d = 0.20$, $\alpha = 0.60$, $\mu = 0.20$, $\beta = 0.10$, $\gamma = 0.75$, *and* $k = 0.10$. We have observed that there is an important role in the dynamics of the values for α and μ. As α was increased from 0.15 to 0.60 and μ was decreased from 0.80 to 0.20, we observed a rapid fall in the susceptible class and I_N increased for the cases with the white noise and without white noise. In this case the recovery rate also increased. The role of white noise has presented a change in the dynamics more accurately. Figure 3 represents the joint solutions for the model (2.1) at zero noise for different values of μ and α. The left side corresponds to $\mu = 0.80$ and $\alpha = 0.15$ while the right side graphs show the

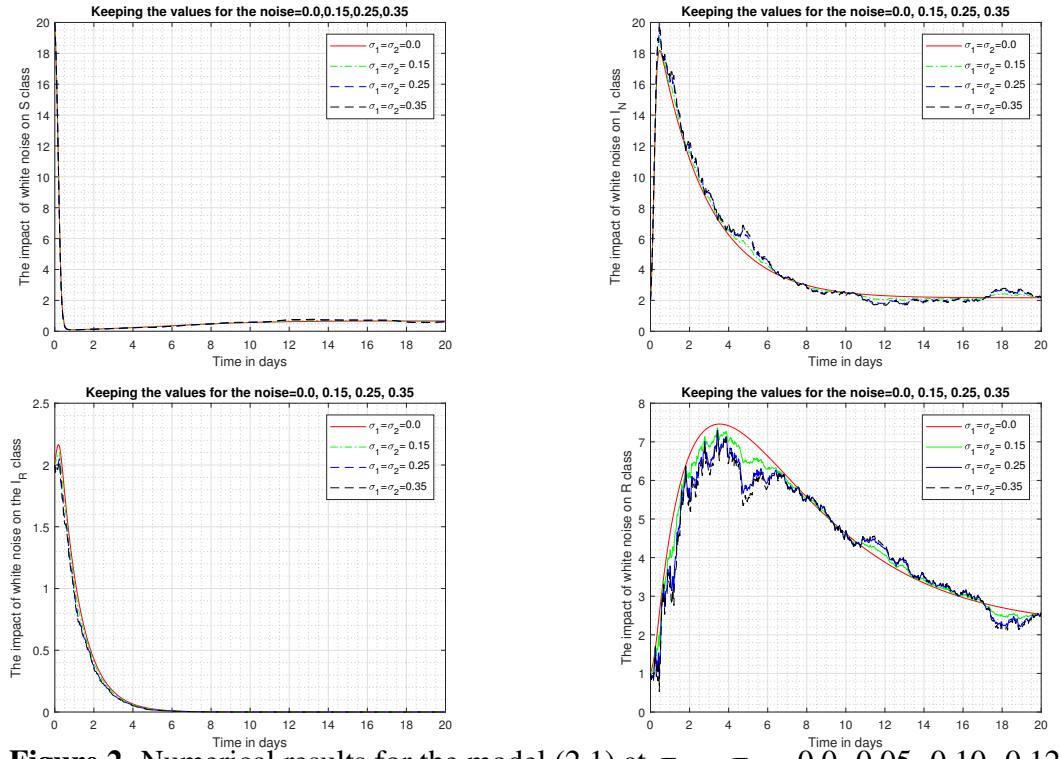

Figure 2. Numerical results for the model (2.1) at $\sigma_1 = \sigma_2 = 0.0, 0.05, 0.10, 0.12$.

joint solution at $\mu = 0.15$ and $\alpha = 0.60$. This change shows a clear difference in the dynamics.

10. Conclusions

In this work, we have explored the dynamic behavior of an $\mathcal{SI}_N\mathcal{I}_R\mathcal{R}$ influenza stochastic model that ponders the effects of information interference and environmental noise. Information interventions and white noise have been discovered to have a significant impacts on the condition. Hereafter, we present our primary findings.

We have measured the effects of environmental white noise on the disease. We have shown that $\dot{\mathrm{R}}_r$ is a threshold of the model (2.1) for the disease to die out or persist, and that noise strength can change the value of the stochastic reproduction number $\dot{\mathrm{R}}_r$. If $\dot{\mathrm{R}}_r < 1$, the disease will die out with a probability of one. On the other hand, if $\dot{\mathrm{R}}_r > 1$, there is a stationary distribution for the model (2.1), which means that the disease will prevail. The discretization approach was used to construct a numerical scheme for the model simulations. The results of the simulations are presented throughout the article in the form of graphs that are divided into three sections. In Figure 2, we have shown a numerical solution of the model (2.1) as well as those obtained in comparative studies of all classes for the white noise values $\sigma_1 = \sigma_2 = 0.0, 0.05, 0.10, 0.12$ and $S(0) = 20$, $I_N(0) = 2$, $I_R(0) = 2$, $R(0) = 1$, $b = 1.00$, $d = 0.20$, $\alpha = 0.60$, $\mu = 0.20$, *and* $\beta = 0.10$. We have seen that the dynamics of the values for α and μ play an essential role. We noticed a rapid fall in the susceptible class as the α value was increased from 0.15 to 0.60 and μ dropped from 0.80 to 0.20; additionally, I_N was increased for both instances with and without white noise. The healing rate was also boosted in this instance. White noise has played a larger role in influencing the dynamics.

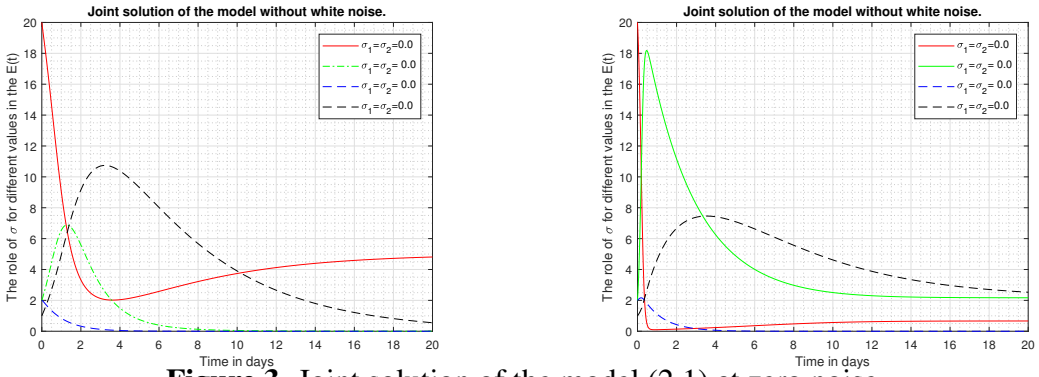

Figure 3. Joint solution of the model (2.1) at zero noise.

The joint solutions for the model (2.1) at zero noise are shown in figure 3 for different values of μ and α . The left side graphs correspond to $\mu = 0.80$ and $\alpha = 0.15$, whereas the right side graphs correspond to $\mu = 0.15$ and $\alpha = 0.60$ for the combined solution. This shift reveals a significant shift in the dynamics.

Acknowledgments

J. Alzabut is thankful to Prince Sultan University and OSTİM Technical University for their endless support. G. Alobaidi was supported by Faculty research grant from the American University of Sharjah (Project number FRG21-S-S05).

Conflict of interest

The authors declare there is no conflict of interest.

References

1. J. Whitman, C. Jayaprakash, Stochastic modeling of influenza spread dynamics with recurrences, *Plos One*, **15** (2020), e0231521. https://doi.org/10.1371/journal.pone.0231521
2. P. Brachman, Infectious diseasespast, present, and future, *Int. J. Epidemiol.*, **32** (2003), 684–686. https://doi.org/10.1093/ije/dyg282
3. C. Peteranderl, S. Herold, C. Schmoldt, Human influenza virus infections, *Semin. Respir. Crit. Care Med.*, **37** (2016), 487–500. https://doi.org/10.1055/s-0036-1584801
4. F. RAM, F. Smith, M. Peiris, K. Kedzierska, P. Doherty, Palese P. Shaw ML Treanor J. Webster RG Gracia-Sastre A, *Nat. Rev. Dis. Primers*, **4** (2018), 3.
5. R. Eccles, Understanding the symptoms of the common cold and influenza, *Lancet Infect. Dis.*, **5** (2005), 718–725. https://doi.org/10.1016/S1473-3099(05)70270-X
6. L. Mohler, D. Flockerzi, H. Sann, U. Reichl, Mathematical model of influenza A virus production in large-scale microcarrier culture, *Biotechnol. Bioeng.*, **90** (2005), 46–58. https://doi.org/10.1002/bit.20363

7. A. Mosnier, S. Caini, I. Daviaud, E. Nauleau, T. Bui, E. Debost, et al., Clinical characteristics are similar across type A and B influenza virus infections, *Plos One*, **10** (2015), e0136186. https://doi.org/10.1371/journal.pone.0136186
8. M. Martcheva, M. Iannelli, X. Li, Subthreshold coexistence of strains: the impact of vaccination and mutation, *Math. Biosci. Eng*., **4** (2007), 287. https://doi.org/10.3934/mbe.2007.4.287
9. W. Shao, X. Li, M. Goraya, S. Wang, J. Chen, Evolution of influenza a virus by mutation and re-assortment, *Int. J. Mol. Sci*., **18** (2017), 1650. https://doi.org/10.3390/ijms18081650
10. Y. Kanegae, S. Sugita, A. Endo, M. Ishida, S. Senya, K. Osako, et al., Evolutionary pattern of the hemagglutinin gene of influenza B viruses isolated in Japan: cocirculating lineages in the same epidemic season. *J. Virol*., **64** (1990), 2860–2865. https://doi.org/10.1128/jvi.64.6.2860-2865.1990
11. A. Fiore, A. Fry, D. Shay, L. Gubareva, J. Bresee, T. Uyeki, Centers for Disease Control and Prevention (CDC) Antiviral agents for the treatment and chemoprophylaxis of influenza recommendations of the Advisory Committee on Immunization Practices (ACIP), *MMWR Recomm. Rep*., **60** (2011), 1–24.
12. A. Monto, J. McKimm-Breschkin, C. Macken, A. Hampson, A. Hay, A. Klimov, et al., Detection of influenza viruses resistant to neuraminidase inhibitors in global surveillance during the first 3 years of their use, *Antimicrob. Agents Chemother*., **50** (2006), 2395–2402. https://doi.org/10.1128/AAC.01339-05
13. J. Carr, J. Ives, L. Kelly, R. Lambkin, J. Oxford, D. Mendel, et al., Influenza virus carrying neuraminidase with reduced sensitivity to oseltamivir carboxylate has altered properties in vitro and is compromised for infectivity and replicative ability in vivo, *Antivir. Res*., **54** (2002), 79–88. https://doi.org/10.1016/S0166-3542(01)00215-7
14. M. Rameix-Welti, V. Enouf, F. Cuvelier, P. Jeannin, S. vanderWerf, Enzymatic properties of the neuraminidase of seasonal H1N1 influenza viruses provide insights for the emergence of natural resistance to oseltamivir, *PLoS Pathog*., **4** (2008), e1000103. https://doi.org/10.1371/journal.pcbi.1000103
15. M. Moghadami, A. Moattari, H. Tabatabaee, A. Mirahmadizadeh, A. Rezaianzadeh, J. Hasanzadeh, et al., High titers of hemagglutination inhibition antibodies against 2009 H1N1 influenza virus in Southern Iran, *Iran. J. Immunol*., **7** (2010), 39–48.
16. A. Hirsch, Handbook of geographical and historical pathology, New Sydenham Society, 1883.
17. D. Molineux, Molineux's historical account of the late general coughs and colds; with some observations on other epidemick distempers, *Philos. Trans*., (1694), 105–111.
18. N. Johnson, J. Mueller, Updating the accounts: global mortality of the 1918-1920" Spanish" influenza pandemic, *Bull. Hist. Med*., **1** (2002), 105–115.
19. *Novel Swine-Origin Influenza A (H1N1) Virus Investigation Team*, Emergence of a novel swine-origin influenza A (H1N1) virus in humans, *N. Engl. J. Med*., **361** (2009), 1–10. https://doi.org/10.1056/NEJMoa0903810
20. *World Health Organization*, Report of the WHO pandemic influenza A (H1N1) vaccine deployment initiative, 2012.

21. A. Siston, S. Rasmussen, M. Honein, A. Fry, K. Seib, W. Callaghan, et al., Pandemic 2009 influenza A (H1N1) virus illness among pregnant women in the United States, *J. Am. Med. Assoc*., **303** (2010), 1517–1525.

22. S. Hussain, E. Nadia, H. Khan, S. Etemad, S. Rezapour, T. Sitthiwirattham, et al., Investigation of the stochastic modeling of COVID-19 with environmental noise from the analytical and numerical point of view, *Mathematics*, **9** (2021), 3122. https://doi.org/10.3390/math9233122

23. S. Hussain, E. Nadia, H. Khan, H. Gulzar, S. Etemad, S. Rezapour et al., On the stochastic modeling of COVID-19 under the environmental white noise, *J. Funct. Spaces*, **2022** (2022). https://doi.org/10.1155/2022/4320865

24. I. Baba, H. Ahmad, M. Alsulami, K. Abualnaja, M. Altanji, A mathematical model to study resistance and non-resistance strains of influenza, *Results Phys*., **26** (2021), 104390. https://doi.org/10.1016/j.rinp.2021.104390

25. Y. Zhao, D. Jiang, D. Regan, The extinction and persistence of the stochastic SIS epidemic model with vaccination, *Phys. A: Stat. Mech. Appl*., **392** (2013), 4916–4927. https://doi.org/10.1016/j.physa.2013.06.009

26. R. Webster, A. Kendal, W. Gerhard, Analysis of antigenic drift in recently isolated influenza A (H1N1) viruses using monoclonal antibody preparations, *Virol. J*., **96** (1979), 258–264. https://doi.org/10.1016/0042-6822(79)90189-2

27. C. Ji, D. Jiang, Threshold behaviour of a stochastic SIR model, *Appl. Math. Model*., **38** (2014), 5067–5079. https://doi.org/10.1016/j.apm.2014.03.037

28. D. Higham, An algorithmic introduction to numerical simulation of stochastic differential equations, *SIAM Rev.*, **43** (2001), 525–546. https://doi.org/10.1137/S0036144500378302