In this paper, we study the dynamics of COVID-19 in the UAE with an extended SEIR epidemic model with vaccination, time-delays, and random noise. The stationary ergodic distribution of positive solutions is examined, in which the solution fluctuates around the equilibrium of the deterministic case, causing the disease to persist stochastically. It is possible to attain infection-free status (extinction) in some situations, in which diseases die out exponentially and with a probability of one. The numerical simulations and fit to real observations prove the effectiveness of the theoretical results. Combining stochastic perturbations with time-delays enhances the dynamics of the model, and white noise intensity is an important part of the treatment of infectious diseases.

1. Introduction

COVID-19 is a disease caused by SARS-CoV-2 that can trigger a respiratory tract infection. It spreads likewise other coronaviruses do, basically through person-to-person contact. Infections range from mild to deadly [1, 2]. To combat the spreading of all infectious diseases, vaccination is one of the most important procedures [3, 4]. Vaccines generally expose the immune system to harmless parts of the pathogen so that the immune system learns to recognize it and may be able to tamp down the infection before any symptoms appear [5, 6]. COVID-19 vaccines, such as Pfizer, AstraZeneca, and Sinopharm, are now widely available for people aged five years and older, and all the currently authorized COVID-19 vaccines are effective and reduce the risk of severe illness [7]. It is normal for a virus to mutate as it infects people, and SARS-CoV-2 has mutated so [8–10]. There are various variants which are now spreading, such as Alpha, Beta, Gamma, Delta, and Omicron. An initial study showed Omicron variant reduced the antibody protection by some vaccines, but a booster shot is likely to protect people from severe disease, and research works are still in proceedings in this field [11].

Up to date, more than 4.41 billion people worldwide have received a dose of the COVID-19 vaccine, equal to about 57.4 percent of the world population [12]. A vaccinated person refers to someone who has received at least one dose of a vaccine, and a fully vaccinated person has completed receiving the vaccine, whether that is one dose or two, and two weeks have passed. A COVID-19 booster shot is an additional dose of a vaccine given after the protection provided by the original shot(s) has begun to decline [13]. The booster is recommended to help people keep up their level of immunity for longer. In the UAE, more than 99 percent of the population at least have one dose of the vaccine, 91 percent of the population are fully vaccinated, and 32.3 percent of the population are booster given [14]; therefore, the number of confirmed cases of COVID-19 in the UAE has decreased significantly.

Modeling infectious diseases provides a controlled environment in which complex relationships between environmental and biological factors can be examined. In public health science, mathematical models of infectious diseases can be used to analyze various scenarios, and the results can inform policy, programs, and practices [15, 16].
Researchers are working to develop mathematical models that can be used to predict vaccination strategies for controlling epidemic diseases [3, 4, 17, 18].

Human virus diseases are highly affected by stochastic perturbations. Because human contact can change from one person to another, epidemic growth and spread in human disease are normally random, and the population is subject to factors that are either not fully understood or difficult to model precisely. A model that ignores these phenomena will negatively affect the analysis of the studied biological systems. Stochastic differential equation models (SDEs) are more suitable for modeling epidemic dynamics under certain conditions [19–21]. Increasingly, deterministic models need to be extended to stochastic models that can account for more complex variations in dynamics [22]. Furthermore, delay differential equations (DDEs) are extensively used to describe the dynamics of infectious diseases. Due to the fact that time-delay is relevant to hidden mechanisms such as the incubation period and the recovery of infected individuals [23–25].

In this paper, we study the dynamics of the COVID-19 epidemic in the UAE, using a modified stochastic delayed SEIRV (Susceptible-Exposed-Infected-Recovered-Vaccinated) model. The model incorporates white noise and time-delays. This model assumes that individuals can become infected during vaccination, but then become healthy afterward [28]. Incorporating time lags in epidemic models makes the systems much more realistic and enriches the dynamics of the model. Therefore, we include time-delays \( \tau_1 \) and \( \tau_2 \) to represent the incubation period; while \( \tau_3 \) stands for the time required for the infected individuals to become recovered. Hence, the deterministic SEIR model with vaccination and time-delays takes the form (see Figure 1).

### 2. The Model

For the dynamics of COVID-19 in the UAE, we propose an extended SEIR epidemic model with vaccination, time-delays, and random noise [26, 27]. The basic model categorized people into four classes: susceptible (S); individuals not yet infected; exposed (E); individuals experiencing incubation duration; infectious (I): confirmed cases; and removed (R): recovered individuals. We assume that the recovered individuals will remain in the class R \((t)\). Therefore, the SEIR model has the following equations system:

\[
\begin{align*}
\frac{dS}{dt} &= \Lambda - \beta_1 SE - aS, \\
\frac{dE}{dt} &= \beta_1 SE - \beta_2 EI - (\kappa + \alpha)E, \\
\frac{dI}{dt} &= \beta_2 EI - (d + r + \alpha)I, \\
\frac{dR}{dt} &= rI + \kappa E - aR, \\
\frac{dV}{dt} &= \beta_3 S - (\beta_6 + \alpha)V - \beta_4 VE - \beta_5 VI(t - \tau_2).
\end{align*}
\]
\( \beta_1, \beta_2, \beta_3, \) and \( \beta_4 \) are the transmission rates of susceptible into vaccinated class; vaccinated into exposed class; vaccinated into infected class; and \( \beta_5 \) vaccinated into recovered class, respectively.

The basic reproduction number,
\[
\mathcal{R}_0 = \frac{\beta_1 A}{(\beta_3 + \alpha)(\kappa + \alpha)} + \frac{\Lambda \beta_2 \beta_4}{(\beta_3 + \alpha)(\kappa + \alpha)(\beta_5 + \alpha)},
\]

of model (2), has a significant impact in epidemiology since it decides whether an epidemic occurs or the disease dies out [29]. If \( \mathcal{R}_0 < 1 \), then model (2) has only a disease-free equilibrium \( \mathcal{E}_0 = (S^*, 0, 0, R^*, V^*) = (\Lambda/\beta_3 + \alpha, 0, 0, \Lambda \beta_4/\alpha(\beta_6 + \alpha)(\beta_5 + \alpha) \) and it is globally asymptotically stable; while if \( \mathcal{R}_0 > 1 \), then, \( \mathcal{E}_0 \) is unstable and there is a unique endemic equilibrium \( \mathcal{E}^* = (S^*, E^*, I^*, R^*, V^*) \) which is globally asymptotically stable [28].

Because some factors cannot be measured precisely, stochastic models always provide an estimate of these uncertainties based on approximate estimates [1, 30–32]. Therefore, we introduce randomness into model (2) by adding white noise to the state of the SEIR model with vaccination and time-delays. The modified model takes the form:

\[
\begin{align*}
\text{d}S &= [A - \beta_1 SE - (\beta_3 + \alpha)S] \text{d}t + \nu_1 S \text{d}W_1, \\
\text{d}E &= [\beta_2 VE + \beta_1 SE - \beta_2 EI (t - \tau_1) - (\kappa + \alpha)E] \text{d}t + \nu_2 E \text{d}W_2, \\
\text{d}I &= [\beta_5 V (t - \tau_2) + \beta_2 EI (t - \tau_1) - (d + r + \alpha)I] \text{d}t + \nu_3 I \text{d}W_3, \\
\text{d}R &= [\beta_4 S - (\beta_6 + \alpha)V - \beta_4 VE - \beta_3 V I (t - \tau_2)] \text{d}t + \nu_4 R \text{d}W_4, \\
\text{d}V &= [\beta_5 I (t - \tau_3) + \kappa E - \alpha R] \text{d}t + \nu_5 V \text{d}W_5,
\end{align*}
\]

with

\[
\begin{align*}
S(\theta) &= \phi_1(\theta), E(\theta) = \phi_2(\theta), I(\theta) = \phi_3(\theta), R(\theta) \\
&= \phi_4(\theta), V(\theta) = \phi_5(\theta), \\
\theta &\in [-r, 0], \quad \tau = \max\{\tau_1, \tau_2, \tau_3\}, \\
\phi_i(0) &> 0 \text{ and } \phi_i(\theta), i = 1, \ldots, 5, \text{ are non-negative continuous initial functions on } [-r, 0]. \quad W_i(t), \ i = 1, \ldots, 5, \text{ represent the independent Brownian motions defined on a complete probability space } (\Omega, \mathcal{F}, \{\mathcal{F}_t\}_{t \geq 0}, P) \text{ with a filtration } \{\mathcal{F}_t\}_{t \geq 0} \text{ satisfying the usual conditions (it is right continuous and } \mathcal{F}_0 \text{ contains all } P \text{- null sets}), \text{ where } \nu_i, i = 1, \ldots, 5 \text{ are the intensities of white noise.}
\end{align*}
\]

### 3. Stationary Distribution and Ergodicity

Among the most important and significant characteristics of the stochastic epidemic model (4) is its ergodic property. Under some conditions of white noise, the stochastic model fluctuates in the neighborhood of the infected equilibrium of the corresponding deterministic model for all time regardless of the starting conditions. First, we need to show that there is a global non-negative solution of model (4), which is as follows:

**Theorem 1.** For any given initial value (5), system (4) has a unique solution \( (S(t), E(t), I(t), R(t), V(t)) \) on \( t \geq -r \), and the solution will remain in \( \mathbb{R}_+^5 \) with probability one.

**Proof.** Since the system coefficients (4) satisfy linear growth and Lipschitzian conditions and based Khasminskii Lyapunov functional approach, we can show that system (4) has a global positive solution. The main challenge is to establish a Lyapunov function, so we define

\[
\mathcal{G}(S, E, I, R, V) = \mathcal{G}_1(S) + (E - 1 - \ln E) + (I - 1 - \ln I) + (V - 1 - \ln V) + (R - 1 - \ln R)
\]

\[
+ \left[ (1 - \ln I) + (R - 1 - \ln R) + (V - 1 - \ln V) + (R - 1 - \ln R) + r \right] \int_{t}^{t+\tau_1} I(s - \tau_1) \text{d}s + \beta_2 \int_{t}^{t+\tau_2} I(s - \tau_2) \text{d}s
\]

Therefore, we introduce randomness into model (2) by adding white noise to the state of the SEIR model with vaccination and time-delays. The modified model takes the form:
By Itô's formula on $\mathcal{G}$,

\[
\begin{align*}
\mathcal{G} (t) &= \mathcal{G} (0) + \int_0^t \mathcal{G} (s) \, ds + \int_0^t \mathcal{G} (s) \, \mathcal{B} (s) \, dW (s) \\
\mathcal{G} (t) &= \mathcal{G} (t) + \int_0^t \mathcal{G} (s) \, ds + \int_0^t \mathcal{G} (s) \, \mathcal{B} (s) \, dW (s),
\end{align*}
\]

where

\[\mathcal{G} = \Lambda - aS - \beta S + (\beta_3 + \alpha) - (\kappa + \alpha)E - \beta_4 V - \beta_5 I + (\kappa + \alpha)\]

\[-(d + \alpha)I - \beta_3 V (t - \tau) - \beta_5 E (t - \tau) I + (d + \alpha) + \beta_6 V + R + \kappa E - \alpha R \]

\[-\beta_3 V R \frac{r I (t - \tau)}{R} - \frac{\alpha (\beta_6 + \alpha) V - \beta_3 S}{R} + (\beta_6 + \alpha) + \beta_3 E + \beta_5 I \]

\[\frac{\tau^2 + \frac{\tau^2}{2} + \frac{\tau^2}{4} + \frac{\tau^2}{8}}{2} \]

\[\leq \Lambda + \beta_3 + \kappa + d + r + \beta_6 + 5\alpha - (\alpha + \beta_4) S + (\beta_1 + \beta_5 - \alpha) E - (\beta_2 + \beta_5 - d - \alpha) I \]

\[-\alpha R - (\beta_4 + \alpha) V + \frac{\tau^2 + \frac{\tau^2}{2} + \frac{\tau^2}{4} + \frac{\tau^2}{8}}{2} \]

\[\leq \mathcal{A},\]

where $\mathcal{A}$ is a positive constant. It follows that $\mathcal{G}$ is bounded. Hence, the rest of the proof is standard [33], so it is omitted.

**Theorem 2.** Define

\[\mathcal{R}_0 = \frac{\beta_1 \Lambda}{v_1 v_2} + \frac{\Lambda \beta_3 \beta_4}{v_1^2 v_2 v_3} \]

where $v_1 = \beta_3 + \alpha + v_2^2 / 2$, $v_2 = \kappa + \alpha + v_2^2 / 2$, $v_3 = d + r + \alpha + v_3^2 / 2$, $v_4 = \beta_6 + \alpha + v_4^2 / 2$, and $v_5 = \beta_5 + \alpha + v_5^2 / 2$. If $\mathcal{R}_0 > 1$, then, system (4) has a unique stationary distribution $\pi (\cdot)$ and it admits the ergodic property.

**Proof.** Let $Y (t)$ is a regular time-homogenous Markov process in $\mathbb{R}^n$, defined by the stochastic delay differential equation:

\[dY (t) = f (Y (t), Y (t - \tau), t) dt + \sum_{i=1}^n g_i (Y (t), t) dW_i (t). \]

The diffusion matrix of the process $Y (t)$ is

\[\Pi (y) = \left( \delta_{ij} (y) \right), \]

\[\delta_{ij} (y) = \sum_{i=1}^n g_i (y) g_j (y). \]

**Lemma 1 ([34]).** The Markov process $Y (t)$ has a unique ergodic stationary distribution $\pi (\cdot)$ if there exists a bounded domain $\mathcal{B} \subset \mathbb{R}^n$ with regular boundary $\Delta$ and

(i) there is a positive number $\mathcal{X}$ such that $\sum_{i,j=1}^n \delta_{ij} (y) \xi_i \xi_j \geq \mathcal{X} |\xi|^2$, $5 \in \mathcal{B}$, $\xi \in \mathbb{R}^n$.

(ii) there exists a non-negative $\mathcal{C}^2$-function $V$ such that $\mathcal{L} V$ is negative for any $V \in \mathcal{B}$.

With a view to prove Theorem 2, we need to guarantee the validity of conditions (i) and (ii) of Lemma 1. Clearly, condition (i) satisfies; we need to check condition (ii). Define $\mathcal{F}_5 = \mathcal{F}_3 + \mathcal{F}_4$, where

\[\mathcal{F}_1 = -\ln \omega_1 \ln S + 1\frac{\tau^3}{\int_1^{\tau_1} I (s - \tau) ds}, \]

\[\mathcal{F}_2 = -\omega_1 \ln S + (\omega_1 \ln V + \omega_1) \frac{\tau^3}{\int_1^{\tau_1} I (s - \tau) ds}, \]

\[\mathcal{F}_3 = \mathcal{N} \mathcal{F}_2 + (-2 \ln S - \ln R - \ln I), \]

\[\mathcal{F}_4 = \frac{1}{\eta + 1} (S + E + R + V)^{\eta+1}. \]

$\omega_1 = \beta_1 \Lambda v_1^2$, $\omega_2 = \beta_3 \beta_4 \Lambda v_1^2 v_2^2$, and $\omega_3 = \beta_3 \beta_4 \Lambda v_1^2 v_3^2$, $0 < \eta < 4$,$v_1^2 v_2^2 v_3^2 v_4^2 v_5^2$, where $\mathcal{N}$ is a positive constant so that
\[-N \tilde{\nu}_3 (\tilde{\mathcal{R}}_0 - 1) + D \leq -2, \quad (13) \]

where \( D = \max \{ D_1, D_2 \} \) such that

\[
D_0 = \sup_{(S, E, I, R, V) \in \mathbb{R}_+^5} \left\{ \Lambda (S + E + I + R + V) - \frac{\alpha}{4} (S + E + I + R + V)^{\eta + 1} \right\},
\]

\[
D_1 = \sup_{(S, E, I, R, V) \in \mathbb{R}_+^5} \left\{ N \left( \beta_2 + \omega \beta_2 I + 2 \beta_1 E + 2 \tilde{\nu}_1 + \tilde{\nu}_3 + \tilde{\nu} - \frac{\alpha}{4} E^{\eta + 1} + D_0 \right) \right\},
\]

\[
D_2 = \sup_{(S, E, I, R, V) \in \mathbb{R}_+^5} \left\{ N \left( \omega_1 \beta_1 + \omega_2 \beta_2 I + 2 \beta_1 E + 2 \tilde{\nu}_1 + \tilde{\nu}_3 + \tilde{\nu} - \frac{\alpha}{4} E^{\eta + 1} + D_0 \right) \right\},
\]

\[
D_3 = \sup_{(S, E, I, R, V) \in \mathbb{R}_+^5} \left\{ N \left( \omega_1 \beta_1 + \omega_2 \beta_2 I + 2 \beta_1 E + 2 \tilde{\nu}_1 + \tilde{\nu}_3 + \tilde{\nu} - \frac{\alpha}{4} E^{\eta + 1} + D_0 \right) \right\}.
\]  

In addition, \( \mathcal{F}_5 \) is continuous and tends to \(+\infty\) as \((S, E, I, R, V)\) approaches the boundary of \( \mathbb{R}_+^5 \) and \( \| (S, E, I, R, V) \| \rightarrow \infty \). Hence, \( \mathcal{F}_5 \) must have a minimum point in the interior of \( \mathbb{R}_+^5 \).

We define a \( \mathcal{C}^2 \)-function \( \mathcal{F} (.) : \mathbb{R}_+^5 \longrightarrow \mathbb{R}_+ \) as

\[
\mathcal{L} \mathcal{F}_1 = -\beta_1 V - \beta_1 S + \beta_2 I + \tilde{\nu}_2 + \omega_1 \left( \frac{\Lambda}{3} + \beta_1 E + \tilde{\nu}_1 \right)
\]

\[
\mathcal{L} \mathcal{F}_2 = -\beta_2 \tilde{\nu}_2 + \tilde{\nu}_2 V + \beta_1 I + \omega_1 \beta_1 E,
\]

\[
\mathcal{L} \mathcal{F}_3 = -\beta_1 \tilde{\nu} + \tilde{\nu} - \beta_2 V + \beta_1 I + \omega_1 \beta_1 E + \omega_2 \beta_1 \left( \frac{\Lambda}{3} + \beta_1 E + \tilde{\nu}_1 \right) + \omega_3 \left( \frac{\beta_1 S}{V} + \tilde{\nu}_3 + \beta_1 E + \beta_1 I \right)
\]

\[
\mathcal{L} \mathcal{F}_4 = -\beta_1 V(t - \tau_4) - \frac{\beta_2 \tilde{\nu}}{I} + \frac{\beta_1 E(t - \tau_4)}{I} + \gamma_3 - \frac{\beta_1 V}{R} - \frac{r I(t - \tau_3)}{R} - \frac{\kappa E}{R} + \tilde{\nu}_4,
\]

\[
\mathcal{L} \mathcal{F}_5 = (S + E + I + R + V)^\eta \left[ \Lambda - a S - a E - (d + a) I - a R - a V \right]
\]

\[
+ \frac{\eta}{2} (S + E + I + R + V)^{\eta + 1} \left( \frac{\gamma_1}{2} S^2 + \frac{1}{2} \beta_2^2 I^2 + \frac{1}{2} \omega^2 I^2 + \frac{1}{2} \gamma_1 R^2 + \frac{1}{2} \gamma_2 V^2 \right)
\]

\[
\leq \Lambda (S + E + I + R + V)^{\eta + 1} - \alpha (S + E + I + R + V)^{\eta + 1}
\]

\[
+ \frac{\gamma^2}{4} \left( \frac{\gamma_1}{2} V \sqrt{\gamma_2} \sqrt{\gamma_1} \sqrt{\gamma_2} V \right) (S + E + I + R + V)^{\eta + 1}
\]

\[
= \Lambda (S + E + I + R + V)^{\eta + 1} - \alpha (S + E + I + R + V)^{\eta + 1}
\]

By If ô’s formula, we obtain

\[
\mathcal{F}(S, E, I, R, V) = \mathcal{F}_5 (S, E, I, R, V)
\]

\[
\mathcal{F}_5 (S(0), E(0), I(0), R(0), V(0)).
\]
such that \( \tilde{\alpha} = \alpha - \eta/4 (\gamma_1^2 \gamma_2^2 \gamma_3^2 \gamma_4^2 \gamma_5^2) \). Hence,

\[
\mathcal{L} \mathcal{F}(S, E, I, R, V) \leq -\mathcal{N} \tilde{\gamma}_2 (\mathcal{R}_0 - 1) + \mathcal{N} \left( \omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_{1\epsilon} \right) E + \mathcal{N} \left( \beta_2 + \omega_3 \beta_{2\epsilon} \right) I - \frac{2\Lambda}{S} + 2\beta_1 E + 2\tilde{\gamma}_1 \\
+ \tilde{\gamma}_3 - \frac{\beta_2 V}{R} - \frac{r I}{R} + \tilde{\gamma}_4 + \Lambda (S + E + I + R + V)^\eta - \tilde{\alpha} (S + E + I + R + V)^\eta+1. 
\]  

(17)

Define a closed bounded set.

\[
\mathcal{B} = \left\{ (S, E, I, R, V) \in \mathbb{R}_+^5 : e \leq S \leq \frac{1}{e} e \leq E \leq \frac{1}{e} e \leq I \leq \frac{1}{e} e^2 \leq R \leq \frac{1}{e} e \leq V \leq \frac{1}{e} \right\}. 
\]  

(18)

By Lemma 1, we need to show that \( \mathcal{L} \mathcal{F} \leq -1 \) for \( (S, E, I, R, V) \in \mathbb{R}_+^5 \setminus \mathcal{B} \), such that \( \mathbb{R}_+^5 \setminus \mathcal{B} = \bigcup_{i=1}^{10} \mathcal{B}_i \), where

\[
\begin{align*}
\mathcal{B}_1 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : 0 < E \leq e \}, \\
\mathcal{B}_2 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : 0 < I < e \}, \\
\mathcal{B}_3 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : 0 < S < e \}, \\
\mathcal{B}_4 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : 0 < V < e, R > e^2 \}, \\
\mathcal{B}_5 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : 0 < R < e^2, V \leq \frac{1}{e} \}, \\
\mathcal{B}_6 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : R > \frac{1}{e} \}, \\
\mathcal{B}_7 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : I > \frac{1}{e} \}, \\
\mathcal{B}_8 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : S > \frac{1}{e} \}, \\
\mathcal{B}_9 &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : V > \frac{1}{e} \}, \\
\mathcal{B}_{10} &= \{ (S, E, I, R, V) \in \mathbb{R}_+^5 : E > \frac{1}{e} \}.
\end{align*}
\]  

(19)

Case 1. If \( (S, E, I, R, V) \in \mathcal{B}_1 \), then

\[
\mathcal{L} \mathcal{F} \leq -\mathcal{N} \tilde{\gamma}_2 (\mathcal{R}_0 - 1) + \mathcal{N} \left( \omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_{1\epsilon} \right) E \\
+ \mathcal{N} \left( \beta_2 + \omega_3 \beta_{2\epsilon} \right) I + 2\beta_1 E + 2\tilde{\gamma}_1 \\
+ \tilde{\gamma}_3 + \tilde{\gamma}_4 - \frac{\beta_2 V}{4} + D_0 \\
\leq -\mathcal{N} \tilde{\gamma}_2 (\mathcal{R}_0 - 1) + \mathcal{N} \left( \omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_{1\epsilon} \right) E + D_1 \\
\leq -\mathcal{N} \tilde{\gamma}_2 (\mathcal{R}_0 - 1) + \mathcal{N} \left( \omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_{1\epsilon} \right) e + D_1 \leq -1.
\]

from condition (10) and \(-\mathcal{N} \tilde{\gamma}_2 (\mathcal{R}_0 - 1) + \mathcal{N} \left( \omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_{1\epsilon} \right) e + D_1 \leq -1, \) we obtain \( \mathcal{L} \mathcal{F} \leq -1. \)

Case 2. If \( (S, E, I, R, V) \in \mathcal{B}_2 \), we have

\[
\mathcal{L} \mathcal{F} \leq -\mathcal{N} \tilde{\gamma}_2 (\mathcal{R}_0 - 1) + \mathcal{N} \left( \omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_{1\epsilon} \right) E \\
+ \mathcal{N} \left( \beta_2 + \omega_3 \beta_{2\epsilon} \right) I + 2\beta_1 E + 2\tilde{\gamma}_1 \\
+ \tilde{\gamma}_3 + \tilde{\gamma}_4 - \frac{\beta_2 V}{4} + D_0 \\
\leq -\mathcal{N} \tilde{\gamma}_2 (\mathcal{R}_0 - 1) + \mathcal{N} \left( \beta_2 + \omega_3 \beta_{2\epsilon} \right) I + D_2 \\
\leq -\mathcal{N} \tilde{\gamma}_2 (\mathcal{R}_0 - 1) + \mathcal{N} \left( \beta_2 + \omega_3 \beta_{2\epsilon} \right) \leq -1.
\]

(20)
which is obtained from (13) and 
\( N \gamma_2 (R_0 - 1) + N (\beta_2 + \omega_3 \beta_3) e + D_2 \leq -1. \)

**Case 3.** If \((S, E, I, R, V) \in B_3\), we get
\[
\mathcal{L}F \leq N (\omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_4) E + N (\beta_2 + \omega_3 \beta_3) I - \frac{2 \lambda}{S} + 2 \beta_1 E + 2 \gamma_1 \\
+ \gamma_3 + \gamma_4 + D_0 - \frac{\alpha}{4} (I^{\eta+1} + E^{\eta+1})
\]
\[
\leq - \frac{2 \lambda}{S} + D_3 \\
\leq - \frac{2 \lambda}{\epsilon} + D_3 \leq -1,
\]
such that \(0 < \epsilon \leq 2 \lambda / D_3 + 1\).

**Case 4.** Let \((S, E, I, R, V) \in B_4\), one may obtain
\[
\mathcal{L}F \leq - \frac{\beta_0 V}{R} + N (\omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_4) E \\
+ N (\beta_2 + \omega_3 \beta_3) I + 2 \beta_1 E + 2 \gamma_1 \\
+ \gamma_3 + \gamma_4 + D_0 - \frac{\alpha}{4} (I^{\eta+1} + E^{\eta+1}) \\
\leq D_3 - \frac{\beta_0}{\epsilon} \leq -1,
\]
where \(0 < \epsilon \leq \beta_0 / D_3 + 1\).

**Case 5.** If \((S, E, I, R, V) \in B_5\), we have
\[
\mathcal{L}F \leq - \frac{\beta_0 V}{R} + N (\omega_1 \beta_1 + \omega_2 \beta_1 + \omega_3 \beta_4) E \\
+ N (\beta_2 + \omega_3 \beta_3) I + 2 \beta_1 E + 2 \gamma_1 \\
+ \gamma_3 + \gamma_4 + D_0 - \frac{\alpha}{4} (I^{\eta+1} + E^{\eta+1}) \\
\leq D_3 - \frac{\beta_0}{\epsilon} \leq -1,
\]
where \(0 < \epsilon \leq \sqrt{\beta_0 / D_3} + 1\).

**Case 6.** If \((S, E, I, R, V) \in B_6\), we have
\[
\mathcal{L}F \leq - \frac{\alpha}{4} R^{\eta+1} + D_3 \\
\leq D_3 - \frac{\alpha}{4} e^{-2(\eta+1)} \leq -1,
\]
where \(0 < \epsilon \leq [\alpha / 4 (D_3 + 1)]^{1/(1+\eta)}\).

**Case 7.** If \((S, E, I, R, V) \in B_7\), one may obtain
\[
\mathcal{L}F \leq - \frac{\alpha}{4} R^{\eta+1} + D_3 \\
\leq D_3 - \frac{\alpha}{4} e^{-2(\eta+1)} \leq -1.
\]
where \(0 < \epsilon \leq [\alpha / 4 (D_3 + 1)]^{1/(1+\eta)}\).

Thus, condition (ii) of Lemma 1 holds; hence, system (4) identifies a unique stationary distribution \(\pi(\cdot)\).

### 4. Extinction of the Disease

In this section, we discuss conditions that predict the extinction of the disease. From the formula of the reproduction number, we can conclude that \(R_0 < R_0\). First, we go through the following Lemmas [21, 32]

**Lemma 2.** Let \((S(t), E(t), I(t), R(t), V(t))\) be the solution of (4) with initial conditions (5), then
\[
\lim_{t \to \infty} \frac{S(t)}{t} = 0,
\]
\[
\lim_{t \to \infty} \frac{E(t)}{t} = 0,
\]
\[
\lim_{t \to \infty} \frac{I(t)}{t} = 0,
\]
\[
\lim_{t \to \infty} \frac{R(t)}{t} = 0,
\]
\[
\lim_{t \to \infty} \frac{V(t)}{t} = 0.
\]

**Lemma 3.** Assume that \(\alpha > 1/2 (\gamma_1 + \gamma_2 + \gamma_3 + \gamma_1 + \gamma_2 + \gamma_3)\). Let \((S(t), E(t), I(t), R(t), V(t))\) be the solution of (4) with initial conditions (4), we have
\[
\lim_{t \to \infty} \frac{\int_0^t S(r) \, dr}{t} = 0, \quad \lim_{t \to \infty} \frac{\int_0^t E(r) \, dr}{t} = 0, \quad \lim_{t \to \infty} \frac{\int_0^t I(r) \, dr}{t} = 0,
\]
\[
\lim_{t \to \infty} \frac{\int_0^t R(r) \, dr}{t} = 0, \quad \lim_{t \to \infty} \frac{\int_0^t V(r) \, dr}{t} = 0.
\]
Theorem 3. Assume $\alpha > 1/2(\gamma_3^2 + \gamma_4^2 + \gamma_5^2 + \gamma_6^2)$. Let $(S(t), E(t), I(t), R(t), V(t))$ be the solution of (4) with initial conditions (5). If $\mathcal{R}_0 < 1$, then

$$\lim_{t \to \infty} \left[ \log \frac{E(t)}{t} + \beta_4 I(t) \right] \leq (\kappa + \alpha)(\mathcal{R}_0 - 1) < 0 \quad \text{a.s.} \quad (29)$$

which means $E(t)$ and $I(t)$ tend to zero exponentially almost surely. In other words the disease dies out with probability one. Moreover,

$$\lim_{t \to \infty} (S(t) + R(t) + V(t)) = \frac{\Lambda}{\alpha} = S^* + R^* + V^*. \quad (30)$$

Proof. Taking integration of the first and fifth equations of (4), we obtain

$$S(t) - S(0) = \Lambda - \beta_1 \langle S(t)E(t) \rangle - (\beta_3 + \alpha) \langle S(t) \rangle + \gamma_1 \int_0^t S(r) dW_1(r) / t,$$

$$V(t) - V(0) = \beta_5 \langle S(t) \rangle - (\beta_6 + \alpha) \langle V(t) \rangle - \beta_4 \langle V(t)I(t) \rangle + \gamma_3 \int_0^t V(r) dW_5(r) / t.$$ \quad (31)

Therefore,

$$\langle S(t) \rangle = \frac{1}{(\beta_3 + \alpha)} \left[ \Lambda - \beta_1 \langle S(t)E(t) \rangle + \gamma_1 \int_0^t S(r) dW_1(r) / t - \langle S(t) - S(0) \rangle \right]$$

$$\leq \frac{\Lambda}{(\beta_3 + \alpha)} + \frac{1}{(\beta_3 + \alpha)} \left( \gamma_1 \int_0^t S(r) dW_1(r) / t - \langle S(t) - S(0) \rangle \right) = \frac{\Lambda}{(\beta_3 + \alpha)} + \psi_1(t), \quad (32)$$

so that $\lim_{t \to \infty} \psi_1(t) = 0.$

Additionally, we have

$$\langle V(t) \rangle = \frac{1}{(\beta_6 + \alpha)} \left[ \beta_3 \langle S(t) \rangle - \beta_4 \langle V(t)E(t) \rangle - \beta_5 \langle V(t)I(t) \rangle + \gamma_5 \int_0^t V(r) dW_5(r) / t - \langle V(t) - V(0) \rangle \right]$$

$$\leq \frac{\beta_3 \langle S(t) \rangle}{(\beta_6 + \alpha)} + \frac{1}{(\beta_6 + \alpha)} \left( \gamma_5 \int_0^t V(r) dW_5(r) / t - \langle V(t) - V(0) \rangle \right)$$

$$\leq \frac{\beta_3 \Lambda}{(\beta_6 + \alpha)(\beta_3 + \alpha)} + \frac{\beta_3}{(\beta_6 + \alpha)} \psi_1(t) + \frac{1}{(\beta_6 + \alpha)} \left( \gamma_5 \int_0^t V(r) dW_5(r) / t - \langle V(t) - V(0) \rangle \right)$$

$$= \frac{\beta_3 \Lambda}{(\beta_6 + \alpha)(\beta_3 + \alpha)} + \frac{\beta_3}{(\beta_6 + \alpha)} \psi_1(t) + \phi_2(t), \quad (33)$$

where $\lim_{t \to \infty} \phi_2(t) = 0.$ Applying Itô’s formula to the second equation of system (4) yields

$$d\log \langle E(t) \rangle = \left( \beta_3 V(t) + \beta_1 S(t) - \beta_2 I(t) - \left( \kappa + \alpha + \frac{\gamma_4^2}{2} \right) \right) dt$$

$$+ \gamma_6 dW_2(t). \quad (34)$$

Integrating equation (34) from 0 to $t$ results in

$$\frac{\log E(t) - \log E(0)}{t} = \beta_4 \langle V(t) \rangle + \beta_1 \langle S(t) \rangle - \beta_2 \langle I(t) \rangle - \left( \kappa + \alpha + \frac{\gamma_4^2}{2} \right) + \frac{\gamma_6 dW_2(t)}{t}. \quad (35)$$
Then, from (32) and (33), we have

\[
\begin{align*}
\frac{\log E(t)}{t} + \beta_2 \langle I(t) \rangle & \leq \beta_4 \left[ \frac{\beta_3 \Lambda}{(\beta_6 + \alpha)(\beta_3 + \alpha)} + \frac{\beta_3}{(\beta_6 + \alpha)} \phi_1(t) + \phi_2(t) \right] \\
& + \beta_1 \left[ \frac{\Lambda}{(\beta_6 + \alpha)(\beta_3 + \alpha)} + \phi_1(t) \right] - (\kappa + \alpha) + \frac{\nu_2 dW_2(t)}{t} + \frac{\log E(0)}{t} \\
& = \frac{\beta_4 \beta_3 \Lambda}{(\beta_6 + \alpha)(\beta_3 + \alpha)} + \frac{\beta_1 \Lambda}{(\beta_3 + \alpha)} - (\kappa + \alpha) + \frac{\nu_2 dW_2(t)}{t} + \frac{\log E(0)}{t}
\end{align*}
\]

where

\[
\lim_{t \to \infty} \phi_3(t) = 0 \text{ a.s.}
\]

If \( R_0 < 1 \), from (36),

\[
\lim_{t \to \infty} \left[ \frac{\log E(t)}{t} + \beta_2 \langle I(t) \rangle \right] \leq \frac{\beta_4 \beta_3 \Lambda}{(\beta_6 + \alpha)(\beta_3 + \alpha)} + \frac{\beta_1 \Lambda}{(\beta_3 + \alpha)} - (\kappa + \alpha) = (\kappa + \alpha)(R_0 - 1) < 0.
\]

Therefore, \( \lim_{t \to \infty} E(t) = \lim_{t \to \infty} I(t) = 0 \). From model (4), we get

\[
d(S(t) + E(t) + I(t) + R(t) + V(t)) = [\Lambda - \alpha (S(t) + E(t)) + I(t) + R(t) + V(t)) - dI(t)]dr + \nu_1 S(t)dW_1(t) + \nu_2 E(t)dW_2(t) + \nu_3 I(t)dW_3(t) + \nu_4 R(t)dW_4(t) + \nu_5 V(t)dW_5(t).
\]

Taking integration of (38) from 0 to \( t \), one obtains

\[
\langle S(t) + E(t) + I(t) + R(t) + V(t) \rangle = \frac{\Lambda}{\alpha} + \phi_4(t),
\]

where

\[
\phi_4(t) = \frac{1}{\alpha} \int_0^t \left( S(0) + E(0) + I(0) + R(0) + V(0) \right) - \frac{1}{t} \langle S(t) + E(t) + I(t) + R(t) + V(t) \rangle \\
- d\langle I(t) \rangle + \frac{\nu_1}{t} \int_0^t S(r)dW_1(r) + \frac{\nu_2}{t} \int_0^t E(r)dW_2(r) \\
+ \frac{\nu_3}{t} \int_0^t I(r)dW_3(r) + \frac{\nu_4}{t} \int_0^t R(r)dW_4(r) + \frac{\nu_5}{t} \int_0^t V(r)dW_5(r).
\]
One can easily obtain that $\lim_{t \to -\infty} \varphi(t) = 0$, and since $\lim_{t \to -\infty} E(t) = \lim_{t \to -\infty} I(t) = 0$, we have
\[ \limsup_{t \to -\infty} (S(t) + E(t) + I(t) + R(t) + V(t)) = \frac{\Lambda}{\alpha} \] (41)
which implies that $\lim_{t \to -\infty} (S(t) + R(t) + V(t)) = \Lambda/\alpha = S^* + R^* + V^*$, as required; hence, the proof is completed. \hfill \Box

5. Numerical Simulations

In this section, we numerically simulate the solution of the stochastic system (4) using Milstein’s higher-order method [35]. The discretization transformation takes the form:

\[ S_{j+1} = S_j + \left[ \Lambda - \beta_S S_j E_j - (\beta_3 + \alpha) S_j \right] + \nu_1 S_j \xi_{1,j} \sqrt{\phi} + \frac{\nu^2}{2} S_j \left[ \xi_{1,j}^2 - 1 \right], \]
\[ E_{j+1} = E_j + \left[ \beta_4 V_j E_j + \beta_S S_j E_j - \beta_2 E_j I_{(j-n)} - (\kappa + \alpha) E_j \right] + \nu_2 E_j \xi_{2,j} \sqrt{\phi} + \frac{\nu^2}{2} E_j \left[ \xi_{2,j}^2 - 1 \right], \]
\[ I_{j+1} = I_j + \left[ \beta_3 V_j I_{(j-n)} + \beta_2 E_j I_{(j-n)} - (d + r + \alpha) I_j \right] + \nu_3 I_j \xi_{3,j} \sqrt{\phi} + \frac{\nu^2}{2} I_j \left[ \xi_{3,j}^2 - 1 \right], \]
\[ R_{j+1} = R_j + \left[ \beta_6 V_j + r I_{(j-n)} + \kappa E_j - \alpha R_j \right] + \nu_4 R_j \xi_{4,j} \sqrt{\phi} + \frac{\nu^2}{2} R_j \left[ \xi_{4,j}^2 - 1 \right], \]
\[ V_{j+1} = V_j + \left[ \beta_1 S_j - (\beta_4 + \alpha) V_j - \beta_5 V_j - \beta_3 V_j I_{(j-n)} \right] + \nu_5 V_j \xi_{5,j} \sqrt{\phi} + \frac{\nu^2}{2} V_j \left[ \xi_{5,j}^2 - 1 \right], \]

where $\xi_{i,j}$, $i = 1, \ldots, 5$ are mutually independent $N(0, 1)$ random variables, $n_1, n_2$, and $n_3$ are integers such that the time-delays can be expressed in terms of the step-size as $\tau_1 = n_1 h$, $\tau_2 = n_2 h$, and $\tau_3 = n_3 h$. We choose a set of parameters $\Lambda = 0.5, \beta_1 = 0.9905, \beta_2 = 1.9, \beta_3 = 0.6, \beta_4 = 0.928, \beta_5 = 1.93, \beta_6 = 0.9092$, $a = 0.5$, $\kappa = 0.003$, $d = 0.01$, and $r = 0.001$, with $\tau_1 = \tau_2 = 3$ and $\tau_1 = 4$.

Figure 2 indicates that the system has a unique stationary distribution and the disease is persistent by Theorem 2, such that the intensities of white noise are relatively small where $\lambda_0 > 1$ with $\nu_1 = 0.01, \nu_2 = 0.01, \nu_3 = 0.02, \nu_4 = 0.02, \nu_5 = 0.02$ and $\nu_6 = 0.01$ with the same set of parameters as in Figure 2, that is the positive equilibrium is globally asymptotically stable such that the stochastic solution fluctuates around the deterministic steady state value and the disease still persist. However, we increase the intensities of white noise $\nu_1 = 0.1, \nu_2 = 0.5$, and $\nu_3 = \nu_4 = \nu_5 = 0.2$ such that $\lambda_0 < 1$. Figure 4 implies that the disease will ultimately tend to extinction under the relatively strong white noises $\nu_1 = 0.1, \nu_2 = 0.5, \nu_3 = 0.3, \nu_4 = 0.4$, and $\nu_5 = 0.2$ confirmed by Theorem 3. In Figure 5, we investigate the impact of the transition rate from susceptible into vaccinated class with different values of $\beta_5$, which indicates that the number of susceptible, exposed, and infected individuals decrease as $\beta_5$ increases, while the recovered individuals increase as $\beta_5$ increases, other parameter values are the same as in Figure 2.

Remark 1. Under certain criteria with a large magnitude of white noises, the disease can be eradicated, whereas the small intensity of white noises can preserve a stationary distribution.

5.1. Fitting the DDEs Model to Real Data. To investigate the reality of the deterministic model (2), we fit real data for the number of the confirmed cases of COVID-19 in the UAE during June 22, 2021, to August 11, 2021 [36] with model (2) using least-square approach [37, 38].

Given a set of real data in Table 1 and a mathematical model (2), the objective function (weighted least squares function) is as follows:
\[ \Phi_H(p) = \sum_{i=1}^{5} \sum_{j=1}^{M} [x'(t_j, p) - X_j]^2 h_{ij}. \] (43)

Here, $x', i = 1, \ldots, 5$ represents the variables $S, E, I, R, V$; $p$ is the model parameter to be estimated. Thus, we then try to attain the optimum parameter $\hat{p}$ that satisfies $\Phi(\hat{p}) \leq \min_{p} \Phi(p) \equiv \max_{\Phi} \mathcal{S}(p)$, where $\mathcal{S}(\hat{p})$ is the likelihood function [37, 38]. However, the estimation of the parameters that appear in the undisturbed model (2) is considered as an optimization problem. Herein, the data are scaled in ten thousands.

Parameters estimates are $\hat{\beta}_2 = 1.99854$, $\hat{\beta}_5 = 1.9813$, $\bar{\beta} = 0.219092$, $\bar{\kappa} = 0.01099$, and $\bar{r} = 0.047$; therefore, $\lambda = 1.54 > 1$, see Figure 6; while Figure 7 illustrates the
Figure 2: Numerical simulations of model (4), which shows that model (4) has a unique ergodic stationary distribution where the disease is persistent and $R_0 > 1$ with $y_i = 0.01$, $i = 1, \ldots, 5$.

Figure 3: The solutions of the stochastic system (4) and the undisturbed system (2) left banners such that $R_0 > 1$. While right banners show the density function diagram of $S(t)$, $R(t)$, and $V(t)$. 
Figure 4: Numerical simulations of model (4) shows that the disease dies out when the white noise is relatively large such that $\hat{R}_0 < 1$.

Figure 5: The impact of the transition rate from susceptible into vaccinated class with different values of $\beta_3$, which indicates that the number of susceptible, exposed, and infected individuals decrease as $\beta_3$ increases, while the recovered individuals increase as $\beta_3$ increases.
The steps of parameter estimations are summarized as follows:

1. Guess an initial parameter estimate $p_0$;
2. We then solve the system using a deterministic model (2) using the current parameters;
3. A minimization routine, such as OPTIMTOOL in Matlab, is then used to adjust the parameter values;
4. When the value $\Phi(p)$ cannot be further reduced, the best fit parameter values have been determined;
5. Determine if the chosen set of parameters is acceptable or not.

### 6. Concluding Remarks

In this paper, we extended the classical SEIR epidemic model to include vaccination and time-delays that incorporate randomness into the equations by including white noise perturbations on some parameters. The model has been examined by fitting to real observations in UAE, during June 22, 2021, to August 11, 2021. The study found that disease extinction is more likely if the noise intensity is high, and this

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**Table 1:** Number of recorded COVID-19 cases in the UAE, from June 21, 2021, to August 11, 2021 [36].

| Time (days)       | June 21 | June 22 | June 23 | June 24 | June 25 | June 26 | June 27 |
|-------------------|---------|---------|---------|---------|---------|---------|---------|
| Infected cases    | 1,850   | 1,964   | 2,167   | 1,988   | 2,161   | 2,223   | 2,282   |
| Time (days)       | June 28 | June 29 | June 30 | July 1  | July 2  | July 3  | July 4  |
| Infected cases    | 2,122   | 1,747   | 1,675   | 1,663   | 1,632   | 1,599   | 1,573   |
| Time (days)       | July 5  | July 6  | July 7  | July 8  | July 9  | July 10 | July 11 |
| Infected cases    | 1,552   | 1,513   | 2.04    | 2,184   | 1,539   | 1,529   | 1,52    |
| Time (days)       | July 12 | July 13 | July 14 | July 15 | July 16 | July 17 | July 18 |
| Infected cases    | 1,518   | 1,542   | 1,522   | 1,529   | 1,508   | 1,541   | 1,506   |
| Time (days)       | July 19 | July 20 | July 21 | July 22 | July 23 | July 24 | July 25 |
| Infected cases    | 1,547   | 1,521   | 1,507   | 1,529   | 1,549   | 1,539   | 1,55    |
| Time (days)       | July 26 | July 27 | July 28 | July 29 | July 30 | July 31 | August 1|
| Infected cases    | 1,52    | 1,537   | 1,519   | 1,537   | 1,548   | 1,52    | 1,537   |
| Time (days)       | August 2| August 3| August 4| August 5| August 6| August 7| August 8|
| Infected cases    | 1,519   | 1,537   | 1,548   | 1,519   | 1,508   | 1,52    | 1,545   |
| Time (days)       | August 9| August 10| August 11|
| Infected cases    | 1,411   | 1,321   | 1,334   |

**Figure 6:** The fitted curve of DDEs model (2) and the confirmed COVID-19 cases in the UAE from June 22, 2021 to August 11, 2021. The estimated parameters are $\hat{\beta}_2 = 1.99854$, $\hat{\beta}_5 = 1.9813$, $\hat{\beta}_6 = 0.219092$, $\hat{\kappa} = 0.01099$, and $\hat{\rho} = 0.047$.

**Figure 7:** The response of the stochastic model (4) with the estimated parameters $\hat{\beta}_2 = 1.99854$, $\hat{\beta}_5 = 1.9813$, $\hat{\beta}_6 = 0.219092$, $\hat{\kappa} = 0.01099$, and $\hat{\rho} = 0.047$, such that the intensities of white noises are $\nu_1 = 0.03$, $\nu_2 = 0.02$, $\nu_3 = 0.02$, $\nu_4 = 0.04$, and $\nu_5 = 0.04$.

The response of the stochastic model (4) with the estimated parameters; therefore, the stochastic fluctuations enhance the consistency of the model with the real data.
can be used to develop some effective control strategies. Biological systems models should include random influences as they deal with real-life subsystems, which cannot be adequately isolated from factors outside the system. The addition of white noise and time-delays adds complexity to the model and enriches its dynamics.

Our conclusions are as follows.

(i) When the intensity of white noise is relatively low, the disease will persist as long as $\mathcal{R}_0 > 1$ (see Figures 2 and 3) and will die out with greater white noise; see Figure 4.

(ii) The stochastic fluctuations improve the consistency of the model with the real data; see Figure 7.

(iii) It is shown that the disease can be controlled efficiently if the level of vaccination is increased. Therefore, as $\beta_1$ is increased, the solution of model (4) fluctuates around the disease-free equilibrium.

(iv) If the stochastic perturbations $\gamma_i = 0$, $i = 1, \ldots, 5$, then, the threshold of the stochastic model (4) can be reduced to that of the deterministic counterpart. Therefore, $\mathcal{R}_0 > 1$ is a generalized result indicating the persistence of the disease.

(v) Using mathematical models to develop, manufacture, and deliver vaccines is more efficient and results in safer and more efficient vaccines.

Future research will focus on stochastic epidemic models with Markovian switching and time-delays.

Data Availability

The authors confirm that the data supporting the findings of this study are available within the article.

Conflicts of Interest

The authors declare no conflicts of interest.

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