

Numerical Analysis of Stochastic COVID-19 Epidemic Model with the Vaccine Intervention

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Abstract. The Pandemic, COVID-19, has left a great impact on every aspect of life around the globe. It paved the way for crises in all major sectors including health and economics. Therefore, that raise the necessity of an appropriate mathematical models to foresee the intensity and spread of COVID-19 variant. The study puts forward the mathematical investigation and examination of a stochastic COVID-19 model by considering viral latent period into account. The model consists eight compartments: susceptible, exposed, infected, recovered-exposed, recovered, dead population, quarantined, and vaccinated humans. The study compares the deterministic and stochastic COVID-19 models to examine their numerical behaviour. The results depict that the deterministic model does not constitutes the real world uncertainties while the stochastic model provides a more representation of COVID-19 transmission. To substantiate our results, real data of infected individuals after vaccination (from November 2022 to January 2023 in Spain) are used for model evaluation. Various numerical methods, including stochastic Runge-Kutta method, stochastic Non-standard Finite Difference (SNSFD) method and the stochastic Euler method, are applied. The results show, for the step sizes the SNSFD method portrays stable numerical behaviour and is less sensitive to step size variation as compared to other numerical methods (Stochastic Euler and Stochastic Runge-Kutta) considered. This study provides insight into the uncertain nature of pandemic outbreaks and may contribute to understanding of intervention and vaccination strategies.

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1. INTRODUCTION

Mathematical modeling is a very useful tool for understanding epidemiological prototypes of corresponding diseases. It helps in taking necessary public health intrusion control measures. The locals of Wuhan, in the Chinese province of Hubei, got sick with an unidentified pneumonia infection as of early December 2019 [21, 46]. The new coronavirus that caused this pneumonia was officially known as COVID-19 by the World Health Organization. It is also known as 2019-nCoV or SARS-CoV-2 by the International Committee on Taxonomy of Viruses. COVID-19 quickly spreads over all of China after Wuhan City [21]. According to the Chinese Health Authority [15], patients may first test negative for COVID-19, but later testing may reveal a positive result. It has been determined via study [15, 17] that the COVID-19 virus is transmitted from person to person and led to the virus's global proliferation, which is no longer limited to China. As of April, 2, 2020, there had been 900306 confirmed cases and 45693 confirmed fatalities, indicating that it was indeed a pandemic outbreak affecting 205 nations [43]. In order to contain the spread of this global epidemic, several researchers worked very hard to investigate and evaluate this illness from multiple perspectives [24, 16]. Hussain, et al. developed a stochastic model to discuss the dynamics of novel coronavirus disease [23]. Some researchers investigated on how to predict the spread of COVID-19 disease in different countries [36, 42, 45]. Yousefpour, et al. explored the design of control strategies of COVID-19 disease [44]. The only current chance in this pandemic period to stop or lessen the severity of the COVID-19 epidemic is vaccination, along with careful testing procedures and preventative measures. If a community is not completely vaccinated, then it is necessary to minimize interaction among individuals. The issue of lowering social alienation and lockdowns can profoundly negative impact on both the economy and mental health of the populace [9]. However, it is preferable to raise the vaccination rate and ensure that every citizen in the region is completely vaccinated. Analysing the data and using mathematical models to measure the impact of mitigation methods can be crucial to understanding the full dynamics of the illness [6]. Forecasting, monitoring, and limiting common outbursts may effectively be achieved through the use of mathematical frameworks built around the spread of infectious diseases [18]. A series of differential equations frequently used for examining the propagation of infectious disease [35]. In the epidemic mathematical models, the most prevalent compartments are susceptible, exposed, infectious, and recovered. This sort of model is also recognized as the SEIR model. On the basis of the scenario under investigation and the data availability, the underlying model can potentially be extended by incorporating new compartments. Regarding SEIR model, the rate of change of susceptible, exposed, infected, and recovered compartments are expressed by a collection of differential equations [7, 5]. Berkane et al. and Mahdi et al. investigate the effect of vaccinated population on

evolution of COVID-19 virus [30, 12]. Bushnaq et al. investigated the COVID-19 model under Mittag-Leffler power law [14].

Many mathematical models have been constructed and developed with the objectives of better understanding of dynamical behavior and the dynamics of the SARS-CoV-2 outbreak [39, 19]. Liu et al. established the work that is crucial to the data-driven model for COVID-19 propagation [29]. Khyar et al. implemented a multi strain SEIR model to explore the intricate behavior of the SARS-CoV-2 pandemic and proved that their model showed worldwide patterns [28]. It is determined that by employing the theory of suitable control strategies to examine the dynamics of the COVID-19 virus, optimal dosages are necessary to reduce the quantity of COVID-19 viruses [22, 41]. It has been investigated that different mathematical models display unique qualitative behaviour [26, 20, 27]. Allen and Burgin reviewed the comparison of deterministic and stochastic SIS and SIR models [4].

The COVID-19 pandemic has shown how important it is to have accurate and flexible mathematical models for predicting and controlling infectious diseases. Traditional deterministic models often do not fully capture real-life situations, as they ignore the random changes in transmission, vaccination, and human behaviour that can strongly affect the spread of a disease. To address this issue, this study develops a stochastic epidemic model that better represents these uncertainties. The key part of this work is extending the Fredj and Cherif model [11] by including the effect of vaccination, which helps provide a more complete understanding of disease dynamics. The results of this study can be useful for policymakers and healthcare professionals when making decisions about vaccination strategies, assessing different interventions, and preparing for future outbreaks. Furthermore, testing the model with real-world data improves its reliability, making it a valuable tool for predicting future epidemics and supporting public health planning.

Several studies have used stochastic models and nonstandard numerical methods to investigate the spread of infectious diseases [38, 8, 32]. Raza et al. [38] studied a stochastic dengue model with an incubation period and examined its numerical behaviour using stochastic Euler, stochastic Runge-Kutta and stochastic nonstandard finite difference methods. Arif et al. [8] considered a stochastic epidemic model for typhoid fever and analysed the performance of numerical schemes in the presence of random effects. For COVID-19, Tesfaye and Satana developed deterministic and stochastic epidemic models that include vaccination and investigated the reproduction number, equilibrium points, stability and the influence of stochastic effects. Recent studies have also considered vaccination, random variations in disease transmission and structure-preserving numerical techniques in epidemic models. These studies show the growing importance of stochastic modeling and numerical methods that can retain important properties of epidemic systems. Recent research show that random fluctuations can influence the system of biological disease. Stochastic effects examined in population models involving interactions between susceptible and infected individuals, show how environmental noise can effect the population dynamics. Similarly, stochastic diffusion modeling of HIV infection in CD4+ T cells was used to examine how random variation affect infection transmission. These studies highlight the important of introducing uncertainty into mathematical models when examine the unpredictable behaviour of infectious disease [1, 2].

The present study differs from these works in both the structure of the model and the numerical comparison considered. An eight-compartment COVID-19 model consisting

of susceptible, exposed, infectious, recovered-exposed, recovered, dead, quarantined and vaccinated populations is considered, and stochastic perturbations are incorporated into the model to represent uncertainty in disease dynamics. The numerical behaviour of the stochastic Euler, stochastic Runge-Kutta and stochastic NSFD schemes is compared for different step sizes. In addition, the deterministic and stochastic model predictions are compared with COVID-19 infection data from Spain during the post-vaccination period from November 2022 to January 2023. Therefore, the contribution of this study is not the development of the numerical methods themselves, but their comparative application to this extended COVID-19 vaccination model and the assessment of their numerical behaviour under stochastic effects.

Future research can further improve this model by including more real-world factors, such as new virus variants, changes in vaccine effectiveness over time, and the impact of human behaviour on disease spread. By refining epidemic models with stochastic elements, we move one step closer to developing more robust and adaptive public health strategies.

The arrangement of this article is as: In section 2, the preliminaries, Brownian motion and stochastic model is discussed. In section 3, formulation of deterministic and stochastic model is provided. In section 4, we provide different numerical schemes (stochastic Euler method, stochastic Runge-Kutta method and stochastic NSFD). Section 5 presents the discussions and results. In Section 6, a conclusion is presented.

2. PRELIMINARIES

In 1905, Einstein introduced the idea of stochastic differential equations (SDEs) by establishing a mathematical connection between the macro diffusion equation and the small-scale random movement of particles. Ordinary differential equations (ODEs) do not take into account random forcing and stochastic inputs, which is reason SDEs are becoming more and more popular these days due to physical advancements in real-world systems. The mathematical basis for solving stochastic differential equations (SDEs) is provided by a stochastic calculus. Generally speaking, the parameterized stochastic differential equation for a continuous time t , and variable Z_t can be written as follows:

$$dZ_t = m(t, Z_t) dt + n(t, Z_t) dB(t) \quad (2.1)$$

and the integral form is

$$Z(t) = c_1 + \int_{t_0}^t m(u, Z_u) du + \int_{t_0}^t n(u, Z_u) dB_u, \quad (2.2)$$

where $m(t, Z_t)$ and $n(t, Z_t)$ are drift and diffusion coefficients, respectively. The “ c_1 ” is called the initial value at instant time t_0 . The solution of Z_t of equation (2.1) or (2.2) is called stochastic process.

2.1. Stochastic Epidemic Model. The idea of stochastic epidemic models incorporates uncertainties into account, whereas deterministic epidemic models do not capture the intrinsic uncertainty of disease dynamics. Stochastic epidemic models are often developed in various ways for diffusing deterministic epidemic models [37, 25, 40]. The SDE theme offers a more beneficial way of transitioning from deterministic to stochastic models in epidemiology. Both parametric and non-parametric perturbation method can be utilized

to generate SDEs. We take a model parameter and modify it into random variables by applying the previous approach. For the latter, we propose adding an additional stochasticity parameter or incorporating Brownian motion into each differential equation. Allen [3] and Britton [13] suggested that non-parametric perturbation is more useful. We will simulate the process of implementing non-parametric perturbation into deterministic epidemic models and evaluate its efficacy using various numerical techniques on stochastic epidemic models. The main objective is to investigate the link between the stochastic and deterministic epidemic models.

3. FORMULATION OF MODEL

This section presents the mathematical formulation of the COVID-19 model considered in this study, including its deterministic and stochastic representations.

3.1. Deterministic COVID-19 Model. An equation representing COVID-19 was developed by authors in [9] using the compartments of susceptible humans (S_H), exposed humans (E_H), infectious humans (I_H), recovered-exposed humans (Er_H), recovered humans (R_H), dead population of humans (D_H), and quarantine humans (Q_H). By including the population of those who have received vaccinations (V_H) in their generalized fractional-order COVID-19 model, we have eight epidemiological components making up the model (Figure 1) with the total population of $H = S_H + E_H + I_H + Er_H + R_H + D_H + Q_H + V_H$. The governing equations from Figure 1 are described by:

$$\frac{dS_H}{dt} = -\frac{\mu_1^h}{H} S_H E_H - \frac{\mu_2^h}{H} S_H I_H - (\alpha^h + \mu_3^h) S_H - \beta_2^h S_H, \quad (3.3a)$$

$$\frac{dE_H}{dt} = \frac{\mu_1^h}{H} S_H E_H + \frac{\mu_2^h}{H} S_H I_H + \sigma^h \mu_1^h V_H I_H - (\alpha_1^h + \alpha_2^h + \beta_2^h) E_H, \quad (3.3b)$$

$$\frac{dI_H}{dt} = \alpha_1^h E_H - (\beta_1^h + \beta_2^h) I_H, \quad (3.3c)$$

$$\frac{dEr_H}{dt} = \alpha_2^h E_H - \beta_2^h Er_H, \quad (3.3d)$$

$$\frac{dR_H}{dt} = \beta_1^h I_H - \beta_2^h R_H, \quad (3.3e)$$

$$\frac{dD_H}{dt} = \beta_2^h I_H, \quad (3.3f)$$

$$\frac{dQ_H}{dt} = \mu_3^h S_H - \beta_2^h Q_H, \quad (3.3g)$$

$$\frac{dV_H}{dt} = \alpha^h S_H - \sigma^h \mu_1^h V_H I_H - \beta_2^h V_H, \quad (3.3h)$$

with the initial conditions:

$$\begin{aligned} S_H(0) &= S_{H_0} \geq 0, & E_H(0) &= E_{H_0} \geq 0, & I_H(0) &= I_{H_0} \geq 0, \\ Er_H(0) &= Er_{H_0} \geq 0, & R_H(0) &= R_{H_0} \geq 0, & D_H(0) &= D_{H_0} \geq 0, \\ Q_H(0) &= Q_{H_0} \geq 0, & V_H(0) &= V_{H_0} \geq 0. \end{aligned}$$

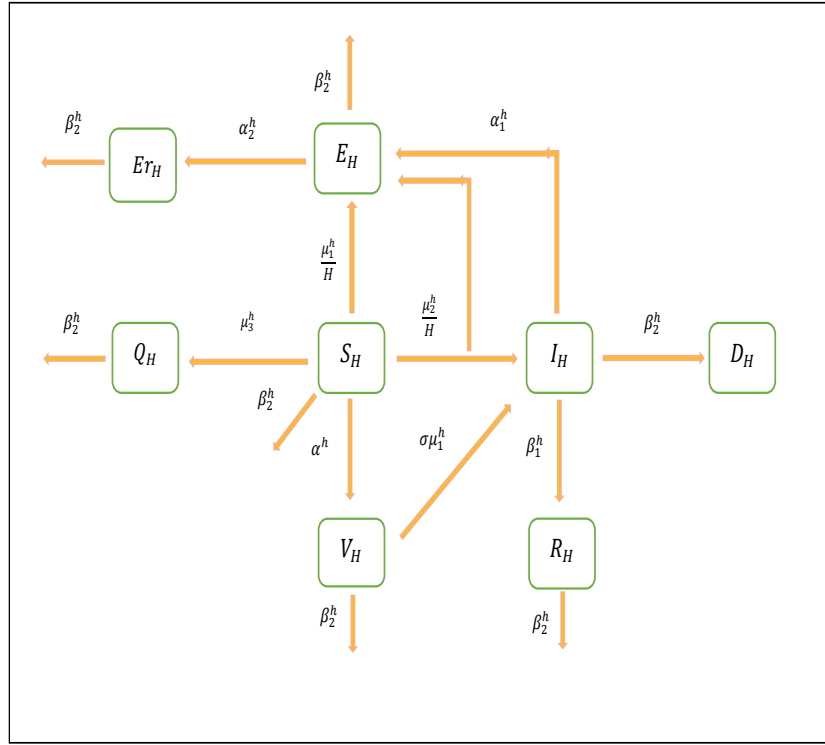


FIGURE 1. Flowchart of COVID-19 propagation in a population with control strategies.

Normalization form of equations (3.3a)-(3.3h) is described by:

$$\frac{dS_h}{dt} = -\mu_1^h S_h E_h - \mu_2^h S_h I_h - (\alpha^h + \mu_3^h) S_h - \beta_2^h S_h, \quad (3.4a)$$

$$\frac{dE_h}{dt} = \mu_1^h S_h E_h + \mu_2^h S_h I_h + \sigma^h \mu_1^h V_h I_h - (\alpha_1^h + \alpha_2^h) E_h - \beta_2^h E_h, \quad (3.4b)$$

$$\frac{dI_h}{dt} = \alpha_1^h E_h - (\beta_1^h + \beta_2^h) I_h, \quad (3.4c)$$

$$\frac{dEr_h}{dt} = \alpha_2^h E_h - \beta_2^h Er_h, \quad (3.4d)$$

$$\frac{dR_h}{dt} = \beta_1^h I_h - \beta_2^h R_h, \quad (3.4e)$$

$$\frac{dD_h}{dt} = \beta_2^h I_h, \quad (3.4f)$$

$$\frac{dQ_h}{dt} = \mu_3^h S_h - \beta_2^h Q_h, \quad (3.4g)$$

$$\frac{dV_h}{dt} = \alpha^h S_h - \sigma^h \mu_1^h V_h I_h - \beta_2^h V_h, \quad (3.4h)$$

where

$$S_h = \frac{S_H}{H}, \quad E_h = \frac{E_H}{H}, \quad I_h = \frac{I_H}{H}, \quad Er_h = \frac{Er_H}{H},$$

$$R_h = \frac{R_H}{H}, \quad D_h = \frac{D_H}{H}, \quad Q_h = \frac{Q_H}{H}, \quad V_h = \frac{V_H}{H},$$

and

$$\begin{aligned} S_h(0) = S_{h_0} \geq 0, & \quad E_h(0) = E_{h_0} \geq 0, & \quad I_h(0) = I_{h_0} \geq 0, \\ Er_h(0) = Er_{h_0} \geq 0, & \quad R_h(0) = R_{h_0} \geq 0, & \quad D_h(0) = D_{h_0} \geq 0, \\ Q_h(0) = Q_{h_0} \geq 0, & \quad V_h(0) = V_{h_0} \geq 0. \end{aligned}$$

3.2. Steady States Analysis of COVID-19 Model. Following the model formulation given in [9], the disease-free condition is considered when no exposed or infectious individuals are present in the population. Under this condition, the population is assumed to remain in the susceptible compartment, and the disease-free state used in this study is expressed as

Disease free equilibrium (DFE): $D_f = (H, 0, 0, 0, 0, 0, 0, 0)$.

Basic reproductive number: $R_1 = \frac{\mu_1^h(\beta_1^h + \beta_2^h) + \mu_2^h \alpha_1^h}{(\alpha_1^h + \alpha_2^h + \beta_2^h)(\beta_1^h + \beta_2^h)}$.

The basic reproduction number R_1 is used as a threshold quantity to describe the transmission potential of the disease. When $R_1 < 1$, disease transmission tends to decrease, whereas $R_1 > 1$ indicates that the infection may continue to spread within the population. Therefore, reducing R_1 below unity is important for controlling disease transmission.

Table 1 presents the parameter values used in the numerical simulations. Two parameter sets are considered to examine the numerical behaviour of the model under different transmission conditions. These parameter sets are referred to as Parameter Set I and Parameter Set II throughout the numerical analysis. Using the parameter values in Table 1, the reproduction number obtained from the above expression is approximately $R_1 = 7.265$ for Parameter Set I and $R_1 = 15.991$ for Parameter Set II. Thus, both parameter sets correspond to $R_1 > 1$, indicating the potential for continued disease transmission.

3.3. Stochastic COVID-19 Model. In order to derive the SDEs for equations (4a)-(4h) let $X = [S_h, E_h, I_h, Er_h, R_h, D_h, Q_h, V_h]^T$ [8, 32], where $\hat{T}$ represents the transpose of the matrix. We have to determine the expectations $\hat{E}[\Delta X]$ and variance $\hat{E}[\Delta X \Delta X^T]$. Table 2 lists possible changes together with their associated transition probabilities to assist with estimate of these expectations.

The expectation and variance of COVID-19 model are defined, respectively, as follows:

$$\begin{aligned} \hat{E}[\Delta X] &= \sum_{i=1}^n P_i(\Delta X)_i, \\ \hat{E}[\Delta X] &= \begin{bmatrix} -\mu_1^h S_h E_h - \mu_2^h S_h I_h - (\alpha^h + \mu_3^h) S_h - \beta_2^h S_h \\ \mu_1^h S_h E_h + \mu_2^h S_h I_h + \sigma^h \mu_1^h V_h I_h - (\alpha_1^h + \alpha_2^h) E_h - \beta_2^h E_h \\ \alpha_1^h E_h - (\beta_1^h + \beta_2^h) I_h \\ \alpha_2^h E_h - \beta_2^h Er_h \\ \beta_1^h I_h - \beta_2^h R_h \\ \beta_2^h I_h \\ \mu_3^h S_h - \beta_2^h Q_h \\ \alpha^h S_h - \sigma^h \mu_1^h V_h I_h - \beta_2^h V_h \end{bmatrix} \Delta t \end{aligned}$$

TABLE 1. Parameter descriptions and numerical values used for the simulations under Parameter Set I and Parameter Set II.

Parameters	Description	Set I	Set II
μ_1^h	Contact rate with S_h and E_h	0.8	1.8
μ_2^h	Contract rate with S_h and I_h	0.02	0.02
μ_3^h	Home quarantine rate of S_h	0.166	0.166
α_1^h	Incubation rate	0.0109	0.0109
α_2^h	Recover rate of E_h	0.1	0.1
β_1^h	Recover rate of I_h	0.003	0.003
β_2^h	Death rate	0.0037	0.0037
α^h	Vaccine rate	0.00000035	0.00000035
σ^h	Vaccine inefficacy	0.005	0.005
σ_1^h	Intensity of noise in S_h	0.5	0.5
σ_2^h	Intensity of noise in E_h	0.1	0.1
σ_3^h	Intensity of noise in I_h	0.4	0.4
σ_4^h	Intensity of noise in Er_h	0.2	0.2
σ_5^h	Intensity of noise in R_h	0.4	0.4
σ_6^h	Intensity of noise in D_h	0.5	0.5
σ_7^h	Intensity of noise in Q_h	0.3	0.3
σ_8^h	Intensity of noise in V_h	0.1	0.1

and

$$\hat{E}[\Delta X \Delta X^{\hat{T}}] = \frac{1}{H} \sum_{i=1}^n P_i[(\Delta X)_i][(\Delta X)_i]^{\hat{T}},$$

$$\hat{E}[\Delta X \Delta X^{\hat{T}}] = \frac{1}{H} \begin{bmatrix} M_{11} & M_{12} & 0 & 0 & 0 & 0 & M_{17} & M_{18} \\ M_{12} & M_{22} & M_{23} & M_{24} & 0 & 0 & 0 & M_{28} \\ 0 & M_{23} & M_{33} & 0 & M_{35} & M_{36} & 0 & 0 \\ 0 & M_{24} & 0 & M_{44} & 0 & 0 & 0 & 0 \\ 0 & 0 & M_{35} & 0 & M_{55} & 0 & 0 & 0 \\ 0 & 0 & M_{36} & 0 & 0 & M_{66} & 0 & 0 \\ M_{17} & 0 & 0 & 0 & 0 & 0 & M_{77} & 0 \\ M_{18} & M_{28} & 0 & 0 & 0 & 0 & 0 & M_{88} \end{bmatrix}.$$

where

$$\begin{aligned} M_{11} &= q_1 + q_2 + q_3 + q_4 + q_5, & M_{12} &= -(q_1 + q_2), \\ M_{17} &= -q_4, & M_{18} &= -q_3, \\ M_{22} &= q_1 + q_2 + q_6 + q_7 + q_8 + q_9, & M_{23} &= -q_7, \\ M_{24} &= -q_8, & M_{28} &= -q_6, \\ M_{33} &= q_7 + q_{10} + q_{11}, & M_{35} &= -q_{10}, \\ M_{36} &= -q_{11}, & M_{44} &= q_8 + q_{12}, \\ M_{55} &= q_{10} + q_{13}, & M_{66} &= q_{11}, \\ M_{77} &= q_4 + q_{14}, & M_{88} &= q_3 + q_6 + q_{15}. \end{aligned}$$

where

$$\begin{aligned} q_1 &= \mu_1^h S_h E_h, & q_2 &= \mu_2^h S_h I_h, & q_3 &= \alpha^h S_h, & q_4 &= \mu_3^h S_h, & q_5 &= \beta_2^h S_h, \\ q_6 &= \sigma^h \mu_1^h V_h I_h, & q_7 &= \alpha_1^h E_h, & q_8 &= \alpha_2^h E_h, & q_9 &= \beta_2^h E_h, & q_{10} &= \beta_1^h I_h, \\ q_{11} &= \beta_2^h I_h, & q_{12} &= \beta_2^h E_r h, & q_{13} &= \beta_2^h R_h, & q_{14} &= \beta_2^h Q_h, & q_{15} &= \beta_2^h V_h. \end{aligned}$$

TABLE 2. Transitions and probabilities of expectation and variance.

Transitions	Probabilities
$(\Delta X)_1 = [-1, 1, 0, 0, 0, 0, 0, 0]^T$	$P_1 = \frac{\mu_1^h}{H} S_h E_h \Delta t$
$(\Delta X)_2 = [-1, 1, 0, 0, 0, 0, 0, 0]^T$	$P_2 = \frac{\mu_2^h}{H} S_h I_h \Delta t$
$(\Delta X)_3 = [-1, 0, 0, 0, 0, 0, 0, 1]^T$	$P_3 = \alpha^h S_h \Delta t$
$(\Delta X)_4 = [-1, 0, 0, 0, 0, 0, 1, 0]^T$	$P_4 = \mu_3^h S_h \Delta t$
$(\Delta X)_5 = [-1, 0, 0, 0, 0, 0, 0, 0]^T$	$P_5 = \beta_2^h S_h \Delta t$
$(\Delta X)_6 = [0, 1, 0, 0, 0, 0, 0, -1]^T$	$P_6 = \sigma^h \mu_1^h V_h I_h \Delta t$
$(\Delta X)_7 = [0, -1, 1, 0, 0, 0, 0, 0]^T$	$P_7 = \alpha_1^h E_h \Delta t$
$(\Delta X)_8 = [0, -1, 0, 1, 0, 0, 0, 0]^T$	$P_8 = \alpha_2^h E_h \Delta t$
$(\Delta X)_9 = [0, -1, 0, 0, 0, 0, 0, 0]^T$	$P_9 = \beta_2^h E_h \Delta t$
$(\Delta X)_{10} = [0, 0, -1, 0, 1, 0, 0, 0]^T$	$P_{10} = \beta_1^h I_h \Delta t$
$(\Delta X)_{11} = [0, 0, -1, 0, 0, 1, 0, 0]^T$	$P_{11} = \beta_2^h I_h \Delta t$
$(\Delta X)_{12} = [0, 0, 0, -1, 0, 0, 0, 0]^T$	$P_{12} = \beta_2^h E_r h \Delta t$
$(\Delta X)_{13} = [0, 0, 0, 0, -1, 0, 0, 0]^T$	$P_{13} = \beta_2^h R_h \Delta t$
$(\Delta X)_{14} = [0, 0, 0, 0, 0, 0, -1, 0]^T$	$P_{14} = \beta_2^h Q_h \Delta t$
$(\Delta X)_{15} = [0, 0, 0, 0, 0, 0, 0, -1]^T$	$P_{15} = \beta_2^h V_h \Delta t$

The stochastic differential equation is defined by,

$$\frac{dX(t)}{dt} = g_1(t, X(t)) + g_2(t, X(t)) \frac{dB(t)}{dt}.$$

Let

$$\text{drift} = g_1(t, X(t)) = \frac{\hat{E}[\Delta X]}{\Delta t}$$

and

$$\text{diffusion} = g_2(t, X(t)) = \sqrt{\frac{\hat{E}[\Delta X \Delta X^T]}{\Delta t}}.$$

Then the stochastic differential equation is,

$$dX(t) = g_1(X(t), t)dt + g_2(X(t), t)dB(t), \quad (3.5)$$

with initial conditions $X(0) = [0.4, 0.3, 0.3, 0, 0, 0, 0, 0]^T$, $0 \leq t \leq T$, and $B(t)$ is the Brownian motion. The SDEs satisfies the diffusion processes. The SDEs given in equation (3.5) of the COVID-19 model are solved numerically using the Euler-Maruyama method [10, 31]. This technique is commonly used to approximate solutions of stochastic models by extending the classical Euler method to include random fluctuations.

For equation (3.5), the Euler-Maruyama scheme can be written as

$$X_{n+1} = X_n + g_1(t, X_n)\Delta t + g_2(t, X_n)\Delta B_n(t), \quad (3.6)$$

where $\Delta B_n = \sqrt{\Delta t} \xi_n$ and Δt is the time step, $B(t)$ is a standard Brownian motion and ξ_n is independent standard normal random variables.

3.4. Non-Parametric Perturbation Stochastic COVID-19 Model. To avoid the difficulty of calculating the expectation and variance matrices for each differential equation, an alternative approach is used. In this approach, random fluctuations are introduced directly into the deterministic COVID-19 model (3.4a)-(3.4h) through non-parametric perturbation terms [33, 34]. This new model is independent of transition stochastic model present in last section and it not calculated from its covariance matrix. So that these two stochastic models represent different mathematical approaches. The perturbed stochastic differential equation for system of equations (3.4a)-(3.4h) are described as:

$$\frac{dS_h}{dt} = -\mu_1^h S_h E_h - \mu_2^h S_h I_h - (\alpha^h + \mu_3^h) S_h - \beta_2^h S_h + \sigma_1^h dB S_h, \quad (3.7a)$$

$$\frac{dE_h}{dt} = (\mu_1^h E_h + \mu_2^h I_h) S_h + \sigma^h \mu_1^h V_h I_h - (\alpha_1^h + \alpha_2^h + \beta_2^h - \sigma_2^h dB) E_h, \quad (3.7b)$$

$$\frac{dI_h}{dt} = \alpha_1^h E_h - (\beta_1^h + \beta_2^h) I_h + \sigma_3^h dB I_h, \quad (3.7c)$$

$$\frac{dEr_h}{dt} = \alpha_2^h E_h - \beta_2^h Er_h + \sigma_4^h dB Er_h, \quad (3.7d)$$

$$\frac{dR_h}{dt} = \beta_1^h I_h - \beta_2^h R_h + \sigma_5^h dB R_h, \quad (3.7e)$$

$$\frac{dD_h}{dt} = \beta_2^h I_h + \sigma_6^h dB D_h, \quad (3.7f)$$

$$\frac{dQ_h}{dt} = \mu_3^h S_h - \beta_2^h Q_h + \sigma_7^h dB Q_h, \quad (3.7g)$$

$$\frac{dV_h}{dt} = \alpha^h S_h - \sigma^h \mu_1^h V_h I_h - \beta_2^h V_h + \sigma_8^h dB V_h, \quad (3.7h)$$

where $B(t)$ is Brownian motion. The parameters σ_j^h for $j = 1, 2, \dots, 8$ represent the intensity (or strength) of stochastic perturbations applied to each respective compartment. We have:

- σ_1^h : intensity of noise in the susceptible class;
- σ_2^h : intensity of noise in the exposed class;
- σ_3^h : intensity of noise in the infected class;
- σ_4^h : intensity of noise in the recovered-exposed class;
- σ_5^h : intensity of noise in the recovered class;
- σ_6^h : intensity of noise in the dead class;
- σ_7^h : intensity of noise in the quarantine class;
- σ_8^h : intensity of noise in the vaccinated class;

These noise intensities scale the impact of stochasticity in each compartment, allow the model to capture environmental or demographic randomness such as variability in transmission, reporting, or vaccine coverage.

It should be noted that the multiplicative perturbation in the death compartment may produce temporary decrease or negative numerical values for some stochastic realizations. Therefore, non-negativity and monotonicity of D_h are not guaranteed in this stochastic scheme. This is a limitation of the multiplicative Brownian perturbation used for this compartment.

The model predictions are compared with real-world COVID-19 infection data from Spain during the post-vaccination period from November 2022 to January 2023 are represented in Table 3, in each cell first entry show the real cases, second entry show the predicted values of infected humans from system (3.4a)-(3.4h) (deterministic COVID-19 model) and last entry show the absolute error between the observed and predicted values.

TABLE 3. Comparison of observed COVID-19 infection data and deterministic model predictions for Spain, with corresponding absolute errors.

Real	0.3167	0.3167	0.3167	0.3167	0.3167
Predicted	0.3167	0.3224	0.3277	0.3343	0.3383
Absolute error	0.0000	0.0057	0.0110	0.0176	0.0216
0.3167	0.3167	0.3364	0.3364	0.3364	0.3364
0.3406	0.3414	0.3410	0.3398	0.3379	0.3355
0.0239	0.0247	0.0046	0.0034	0.0015	0.0009
0.3364	0.3364	0.3364	0.3292	0.3292	0.3292
0.3326	0.3261	0.3226	0.3189	0.3113	0.3075
0.0038	0.0103	0.0138	0.0103	0.0179	0.0217
0.3292	0.3292	0.3292	0.3292	0.2663	0.2663
0.3008	0.2969	0.2902	0.2864	0.2790	0.2753
0.0284	0.0323	0.0383	0.0428	0.0127	0.0090
0.2663	0.2663	0.2663	0.2663	0.2663	0.2663
0.2681	0.2645	0.2575	0.2541	0.2474	0.2441
0.0018	0.0018	0.0088	0.0122	0.0189	0.0222
0.2663	0.2051	0.2051	0.2051	0.2051	0.2051
0.2376	0.2345	0.2283	0.2252	0.2193	0.2142
0.0287	0.0294	0.0232	0.0199	0.0142	0.0091
0.2051	0.2051	0.2051	0.2051	0.2051	0.1622
0.2085	0.2057	0.2009	0.1983	0.1930	0.1904
0.0034	0.0006	0.0042	0.0068	0.0121	0.0282
0.1622	0.1622	0.1622	0.1622	0.1622	0.1622
0.1854	0.1829	0.1781	0.1734	0.1711	0.1660
0.0232	0.0207	0.0159	0.0112	0.0089	0.0038
0.1622	0.1622	0.1622	0.1345	0.1345	0.1345
0.1632	0.1578	0.1552	0.1451	0.1403	0.1348
0.0010	0.0044	0.0070	0.0106	0.0058	0.0003
0.1345	0.1345	0.1345	0.1345	0.1345	0.1345
0.1295	0.1343	0.1308	0.1290	0.1248	0.1187
0.0050	0.0002	0.0037	0.0055	0.0097	0.0158
0.1345	0.0527	0.0527	0.0527	0.0527	0.0527
0.1125	0.1098	0.1013	0.0929	0.0874	0.0770
0.0220	0.0571	0.0485	0.0402	0.0347	0.0243
0.0527	0.0527	0.0527	0.0527	0.0527	
0.0687	0.0596	0.0550	0.0537	0.0525	
0.0160	0.0069	0.0023	0.0010	0.0002	

The model predictions are compared with real-world COVID-19 infection data from Spain during the post-vaccination period from November 2022 to January 2023 are represented in Table 4, in each cell first entry shows the real cases, second entry shows the predicted values of infected humans from system (3.7a)-(3.7h) (stochastic COVID-19 model) and last entry shows the absolute error between the observed and predicted values.

TABLE 4. Comparison of observed COVID-19 infection data and stochastic model predictions for Spain, with corresponding absolute errors.

Real Predicted Absolute error	0.3167 0.3167 0.0000	0.3167 0.3240 0.0073	0.3167 0.3496 0.0329	0.3167 0.3490 0.0323	0.3167 0.3504 0.0337
0.3167 0.3391 0.0224	0.3167 0.3441 0.0274	0.3364 0.3280 0.0084	0.3364 0.3223 0.0141	0.3364 0.3112 0.0252	0.3364 0.3219 0.0145
0.3364 0.3292 0.0072	0.3364 0.3156 0.0208	0.3364 0.3075 0.0289	0.3292 0.3100 0.0192	0.3292 0.3038 0.0254	0.3292 0.3047 0.0245
0.3292 0.2925 0.0367	0.3292 0.2771 0.0521	0.3292 0.2917 0.0375	0.3292 0.2709 0.0583	0.2663 0.2503 0.0160	0.2663 0.2594 0.0069
0.2663 0.2437 0.0226	0.2663 0.2470 0.0193	0.2663 0.2440 0.0223	0.2663 0.2505 0.0158	0.2663 0.2467 0.0196	0.2663 0.2325 0.0338
0.2663 0.2386 0.0277	0.2051 0.2492 0.0441	0.2051 0.2275 0.0224	0.2051 0.2320 0.0269	0.2051 0.2332 0.0281	0.2051 0.2220 0.0169
0.2051 0.2021 0.0030	0.2051 0.2058 0.0007	0.2051 0.1920 0.0131	0.2051 0.1938 0.0113	0.2051 0.1872 0.0179	0.1622 0.1843 0.0221
0.1622 0.1740 0.0118	0.1622 0.1798 0.0176	0.1622 0.1862 0.0240	0.1622 0.1746 0.0124	0.1622 0.1720 0.0098	0.1622 0.1727 0.0105
0.1622 0.1668 0.0046	0.1622 0.1566 0.0056	0.1622 0.1503 0.0119	0.1345 0.1451 0.0106	0.1345 0.1384 0.0039	0.1345 0.1466 0.0121
0.1345 0.1376 0.0031	0.1345 0.1332 0.0013	0.1345 0.1253 0.0092	0.1345 0.1193 0.0152	0.1345 0.1134 0.0211	0.1345 0.1083 0.0262
0.1345 0.1030 0.0315	0.0527 0.0983 0.0456	0.0527 0.0788 0.0261	0.0527 0.0815 0.0288	0.0527 0.0818 0.0291	0.0527 0.0792 0.0265
0.0527 0.0733 0.0206	0.0527 0.0668 0.0141	0.0527 0.0570 0.0043	0.0527 0.0598 0.0071	0.0527 0.0523 0.0004	

4. NUMERICAL SCHEMES

Due to the non-differentiability term of Brownian motion, the non-parametric perturbation stochastic COVID-19 model does not have explicit solutions. Therefore, we present some creative stochastic numerical techniques (Stochastic Euler, Stochastic Runge-Kutta (multi-stage numerical method) and Stochastic non-standard finite difference (SNSFD)) to solve non-parametric perturbation stochastic COVID-19 model.

Three stochastic numerical schemes are considered for comparative purposes. The stochastic Euler scheme provides a simple baseline discretization, the stochastic Runge-Kutta scheme provides a multi-stage comparison, and the stochastic NSFD scheme is considered to investigate the preservation of qualitative properties and numerical behaviour under variations in the step size. These schemes follow the numerical framework adopted in [38, 8], while in this study a common Brownian motion is used. In the numerical implementation, the stochastic random term at the n -th time step is generated as $dB_n = \sqrt{h}\xi_n$, where $\xi_n \sim N(0, 1)$, and the same dB_n is used in all compartment equations at that time step.

The stochastic Euler method [38, 8] with common Brownian motion for non-parametric perturbation COVID-19 model (3.7a)-(3.7h) are described by:

$$S_h^{n+1} = S_h^n + h[-\mu_1^h S_h^n E_h^n - \mu_2^h S_h^n I_h^n - (\alpha^h + \mu_3^h) S_h^n - \beta_2^h S_h^n + \sigma_1^h dB_n S_h^n],$$

$$E_h^{n+1} = E_h^n + h[\mu_1^h S_h^n E_h^n + \mu_2^h S_h^n I_h^n + \sigma^h \mu_1^h V_h^n I_h^n - (\alpha_1^h + \alpha_2^h + \beta_2^h) E_h^n + \sigma_2^h dB_n E_h^n],$$

$$I_h^{n+1} = I_h^n + h[\alpha_1^h E_h^n - (\beta_1^h + \beta_2^h) I_h^n + \sigma_3^h dB_n I_h^n],$$

$$Er_h^{n+1} = Er_h^n + h[\alpha_2^h E_h^n - \beta_2^h Er_h^n + \sigma_4^h dB_n Er_h^n],$$

$$R_h^{n+1} = R_h^n + h[\beta_1^h I_h^n - \beta_2^h R_h^n + \sigma_5^h dB_n R_h^n],$$

$$D_h^{n+1} = D_h^n + h[\beta_2^h I_h^n + \sigma_6^h dB_n D_h^n],$$

$$Q_h^{n+1} = Q_h^n + h[\mu_3^h S_h^n - \beta_2^h Q_h^n + \sigma_7^h dB_n Q_h^n],$$

$$V_h^{n+1} = V_h^n + h[\alpha^h S_h^n - \sigma^h \mu_1^h V_h^n I_h^n - \beta_2^h V_h^n + \sigma_8^h dB_n V_h^n],$$

where "h" is called step size.

The stochastic Runge-Kutta (SRK) scheme adopted from Raza, Arif and Rafiq [38] is used here as a multi-stage numerical scheme with common Brownian motion is used. The scheme employs a four-stage Runge-Kutta structure with stochastic terms incorporated at each stage. No specific strong or weak convergence order is assumed in the present study.

The non-parametric perturbation stochastic COVID-19 model (3.7a)-(3.7h) can be used to generate the stochastic Runge-Kutta method (multi-stage numerical method) in the following way:

Step 1:

$$A_{11} = h[-\mu_1^h S_h^n E_h^n - \mu_2^h S_h^n I_h^n - (\alpha^h + \mu_3^h) S_h^n - \beta_2^h S_h^n + \sigma_1^h dB_n S_h^n],$$

$$B_{21} = h[\mu_1^h S_h^n E_h^n + \mu_2^h S_h^n I_h^n + \sigma^h \mu_1^h V_h^n I_h^n - (\alpha_1^h + \alpha_2^h) E_h^n - \beta_2^h E_h^n + \sigma_2^h dB_n E_h^n],$$

$$C_{31} = h[\alpha_1^h E_h^n - (\beta_1^h + \beta_2^h) I_h^n + \sigma_3^h dB_n I_h^n],$$

$$D_{41} = h[\alpha_2^h E_h^n - \beta_2^h Er_h^n + \sigma_4^h dB_n Er_h^n],$$

$$E_{51} = h[\beta_1^h I_h^n - \beta_2^h R_h^n + \sigma_5^h dB_n R_h^n],$$

$$F_{61} = h[\beta_2^h I_h^n + \sigma_6^h dB_n D_h^n],$$

$$G_{71} = h[\mu_3^h S_h^n - \beta_2^h Q_h^n + \sigma_7^h dB_n Q_h^n],$$

$$H_{81} = h[\alpha^h S_h^n - \sigma^h \mu_1^h V_h^n I_h^n - \beta_2^h V_h^n + \sigma_8^h dB_n V_h^n].$$

Step 2:

$$A_{12} = h[-\mu_1^h(S_h^n + \frac{A_{11}}{2})(E_h^n + \frac{B_{21}}{2}) - \mu_2^h(S_h^n + \frac{A_{11}}{2})(I_h^n + \frac{C_{31}}{2}) \\ - (\alpha^h + \mu_3^h)(S_h^n + \frac{A_{11}}{2}) - \beta_2^h(S_h^n + \frac{A_{11}}{2}) + \sigma_1^h dB_n(S_h^n + \frac{A_{11}}{2})],$$

$$B_{22} = h[\mu_1^h(S_h^n + \frac{A_{11}}{2})(E_h^n + \frac{B_{21}}{2}) + \mu_2^h(S_h^n + \frac{A_{11}}{2})(I_h^n + \frac{C_{31}}{2}) + \sigma^h \mu_1^h(V_h^n + \frac{H_{81}}{2}) \\ (I_h^n + \frac{C_{31}}{2}) - (\alpha_1^h + \alpha_2^h)(E_h^n + \frac{B_{21}}{2}) - \beta_2^h(E_h^n + \frac{B_{21}}{2}) + \sigma_2^h dB_n(E_h^n + \frac{B_{21}}{2})],$$

$$C_{32} = h[\alpha_1^h(E_h^n + \frac{B_{21}}{2}) - (\beta_1^h + \beta_2^h)(I_h^n + \frac{C_{31}}{2}) + \sigma_3^h dB_n(I_h^n + \frac{C_{31}}{2})],$$

$$D_{42} = h[\alpha_2^h(E_h^n + \frac{B_{21}}{2}) - \beta_2^h(E_h^n + \frac{B_{21}}{2}) + \sigma_4^h dB_n(E_h^n + \frac{B_{21}}{2})],$$

$$E_{52} = h[\beta_1^h(I_h^n + \frac{C_{31}}{2}) - \beta_2^h(R_h^n + \frac{E_{51}}{2}) + \sigma_5^h dB_n(R_h^n + \frac{E_{51}}{2})],$$

$$F_{62} = h[\beta_2^h(I_h^n + \frac{C_{31}}{2}) + \sigma_6^h dB_n(D_h^n + \frac{F_{61}}{2})],$$

$$G_{72} = h[\mu_3^h(S_h^n + \frac{A_{11}}{2}) - \beta_2^h(Q_h^n + \frac{G_{71}}{2}) + \sigma_7^h dB_n(Q_h^n + \frac{G_{71}}{2})],$$

$$H_{82} = h[\alpha^h(S_h^n + \frac{A_{11}}{2}) - \sigma^h \mu_1^h(V_h^n + \frac{H_{31}}{2})(I_h^n + \frac{C_{31}}{2}) - \beta_2^h(V_h^n + \frac{H_{31}}{2}) \\ + \sigma_8^h dB_n(V_h^n + \frac{H_{31}}{2})].$$

Step 3:

$$A_{13} = h[-\mu_1^h(S_h^n + \frac{A_{12}}{2})(E_h^n + \frac{B_{22}}{2}) - \mu_2^h(S_h^n + \frac{A_{12}}{2})(I_h^n + \frac{C_{32}}{2}) - (\alpha^h + \mu_3^h)(S_h^n \\ + \frac{A_{12}}{2}) - \beta_2^h(S_h^n + \frac{A_{12}}{2}) + \sigma_1^h dB_n(S_h^n + \frac{A_{12}}{2})],$$

$$B_{23} = h[\mu_1^h(S_h^n + \frac{A_{12}}{2})(E_h^n + \frac{B_{22}}{2}) + \mu_2^h(S_h^n + \frac{A_{12}}{2})(I_h^n + \frac{C_{32}}{2}) + \sigma^h \mu_1^h(V_h^n \\ + \frac{H_{32}}{2})(I_h^n + \frac{C_{32}}{2}) - (\alpha_1^h + \alpha_2^h)(E_h^n + \frac{B_{22}}{2}) - \beta_2^h(E_h^n + \frac{B_{22}}{2}) + \sigma_2^h dB_n(E_h^n + \frac{B_{22}}{2})],$$

$$\begin{aligned}
C_{33} &= h[\alpha_1^h(E_h^n + \frac{B_{22}}{2}) - (\beta_1^h + \beta_2^h)(I_h^n + \frac{C_{32}}{2}) + \sigma_3^h dB_n(I_h^n + \frac{C_{32}}{2})], \\
D_{43} &= h[\alpha_2^h(E_h^n + \frac{B_{22}}{2}) - \beta_2^h(Er_h^n + \frac{D_{42}}{2}) + \sigma_4^h dB_n(Er_h^n + \frac{D_{42}}{2})], \\
E_{53} &= h[\beta_1^h(I_h^n + \frac{C_{32}}{2}) - \beta_2^h(R_h^n + \frac{E_{52}}{2}) + \sigma_5^h dB_n(R_h^n + \frac{E_{52}}{2})], \\
F_{63} &= h[\beta_2^h(I_h^n + \frac{C_{32}}{2}) + \sigma_6^h dB_n(D_h^n + \frac{F_{62}}{2})], \\
G_{73} &= h[\mu_3^h(S_h^n + \frac{A_{12}}{2}) - \beta_2^h(Q_h^n + \frac{G_{72}}{2}) + \sigma_7^h dB_n(Q_h^n + \frac{G_{72}}{2})], \\
H_{83} &= h[\alpha^h(S_h^n + \frac{A_{12}}{2}) - \sigma^h \mu_1^h(V_h^n + \frac{H_{82}}{2})(I_h^n + \frac{C_{32}}{2}) - \beta_2^h(V_h^n + \frac{H_{32}}{2}) \\
&\quad + \sigma_8^h dB_n(V_h^n + \frac{H_{32}}{2})].
\end{aligned}$$

Step 4:

$$\begin{aligned}
A_{14} &= h[-\mu_1^h(S_h^n + A_{13})(E_h^n + B_{23}) - \mu_2^h(S_h^n + A_{13})(I_h^n + C_{33}) - (\alpha^h + \mu_3^h)(S_h^n \\
&\quad + A_{13}) - \beta_2^h(S_h^n + A_{13}) + \sigma_1^h dB_n(S_h^n + A_{13})], \\
B_{24} &= h[\mu_1^h(S_h^n + A_{13})(E_h^n + B_{23}) + \mu_2^h(S_h^n + A_{13})(I_h^n + C_{33}) + \sigma^h \mu_1^h(V_h^n + H_{83})(I_h^n \\
&\quad + C_{33}) - (\alpha_1^h + \alpha_2^h)(E_h^n + B_{23}) - \beta_2^h(E_h^n + B_{23}) + \sigma_2^h dB_n(E_h^n + B_{23})], \\
C_{34} &= h[\alpha_1^h(E_h^n + B_{23}) - (\beta_1^h + \beta_2^h)(I_h^n + C_{33}) + \sigma_3^h dB_n(I_h^n + C_{33})], \\
D_{44} &= h[\alpha_2^h(E_h^n + B_{23}) - \beta_2^h(Er_h^n + D_{43}) + \sigma_4^h dB_n(Er_h^n + D_{43})], \\
E_{54} &= h[\beta_1^h(I_h^n + C_{33}) - \beta_2^h(R_h^n + E_{53}) + \sigma_5^h dB_n(R_h^n + E_{53})], \\
F_{64} &= h[\beta_2^h(I_h^n + C_{33}) + \sigma_6^h dB_n(D_h^n + F_{63})], \\
G_{74} &= h[\mu_3^h(S_h^n + A_{13}) - \beta_2^h(Q_h^n + G_{73}) + \sigma_7^h dB_n(Q_h^n + G_{73})], \\
H_{84} &= h[\alpha^h(S_h^n + A_{13}) - \sigma^h \mu_1^h(V_h^n + H_{83})(I_h^n + C_{33}) - \beta_2^h(V_h^n + H_{83}) \\
&\quad + \sigma_8^h dB_n(V_h^n + H_{83})].
\end{aligned}$$

Step 5:

$$\begin{aligned}
S_h^{n+1} &= S_h^n + \frac{1}{6} [A_{11} + 2A_{12} + 2A_{13} + A_{14}], \\
E_h^{n+1} &= E_h^n + \frac{1}{6} [B_{21} + 2B_{22} + 2B_{23} + B_{24}], \\
I_h^{n+1} &= I_h^n + \frac{1}{6} [C_{31} + 2C_{32} + 2C_{33} + C_{34}], \\
Er_h^{n+1} &= Er_h^n + \frac{1}{6} [D_{41} + 2D_{42} + 2D_{43} + D_{44}], \\
R_h^{n+1} &= R_h^n + \frac{1}{6} [E_{51} + 2E_{52} + 2E_{53} + E_{54}], \\
D_h^{n+1} &= D_h^n + \frac{1}{6} [F_{61} + 2F_{62} + 2F_{63} + F_{64}], \\
Q_h^{n+1} &= Q_h^n + \frac{1}{6} [G_{71} + 2G_{72} + 2G_{73} + G_{74}], \\
V_h^{n+1} &= V_h^n + \frac{1}{6} [H_{81} + 2H_{82} + 2H_{83} + H_{84}],
\end{aligned}$$

where "h" is called step size.

The preceding non-parametric perturbation stochastic COVID-19 model (3.7a)-(3.7h) can be used to create the suggested framework of the SNSFD method [38, 8] in the following way:

$$\begin{aligned}
S_h^{n+1} &= \frac{S_h^n + \varphi(h)[\sigma_1^h dB_n S_h^n]}{1 + \varphi(h)(\alpha^h + \mu_3^h + \beta_2^h + \mu_1^h E_h^n + \mu_2^h I_h^n)}, \\
E_h^{n+1} &= \frac{E_h^n + \varphi(h)[\mu_1^h S_h^n E_h^n + \mu_2^h S_h^n I_h^n + \sigma^h \mu_1^h V_h^n I_h^n + \sigma_2^h dB_n E_h^n]}{1 + \varphi(h)(\alpha_1^h + \alpha_2^h + \beta_2^h)}, \\
I_h^{n+1} &= \frac{I_h^n + \varphi(h)[\alpha_1^h E_h^n + \sigma_3^h dB_n I_h^n]}{1 + \varphi(h)(\beta_1^h + \beta_2^h)}, \\
Er_h^{n+1} &= \frac{Er_h^n + \varphi(h)[\alpha_2^h E_h^n + \sigma_4^h dB_n Er_h^n]}{1 + \varphi(h)(\beta_2^h)}, \\
R_h^{n+1} &= \frac{R_h^n + \varphi(h)[\beta_1^h I_h^n + \sigma_5^h dB_n R_h^n]}{1 + \varphi(h)(\beta_2^h)}, \\
D_h^{n+1} &= D_h^n + \varphi(h)[\beta_2^h I_h^n + \sigma_6^h dB_n D_h^n], \\
Q_h^{n+1} &= \frac{Q_h^n + \varphi(h)[\mu_3^h S_h^n + \sigma_7^h dB_n Q_h^n]}{1 + \varphi(h)(\beta_2^h)}, \\
V_h^{n+1} &= \frac{V_h^n + \varphi(h)[\alpha^h S_h^n + \sigma_8^h dB_n V_h^n]}{1 + \varphi(h)(\sigma^h \mu_1^h I_h^n + \beta_2^h)},
\end{aligned}$$

where $\varphi(h) = 1 - e^{-h}$, " h " is called step size and values of parameters are given in Table 1.

Using MATLAB, we simulate the solution of the proposed framework of these techniques.

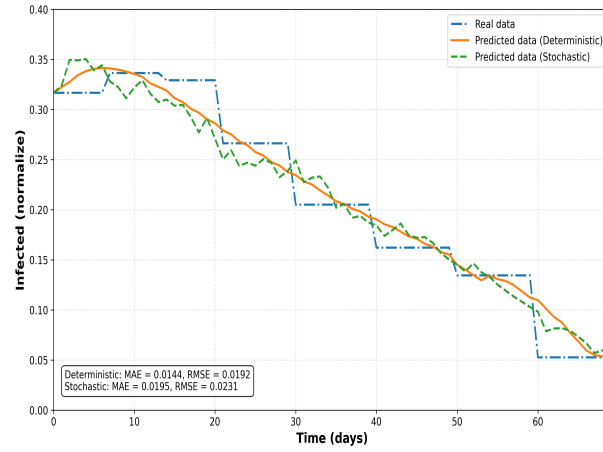


FIGURE 2. Comparison of observed COVID-19 infection data from Spain and the corresponding deterministic and stochastic model predictions during the post-vaccination period from November 2022 to January 2023.

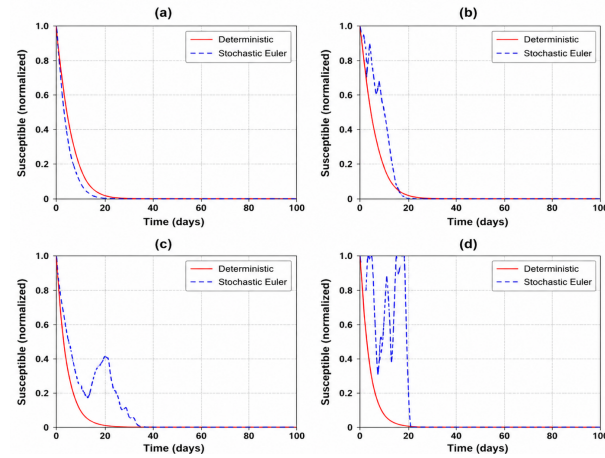


FIGURE 3. Susceptible humans for (a) $h = 0.1$, (b) $h = 0.3$, (c) $h = 0.5$, and (d) $h = 0.9$, comparing the deterministic Euler scheme (red line) and stochastic Euler scheme (blue line).

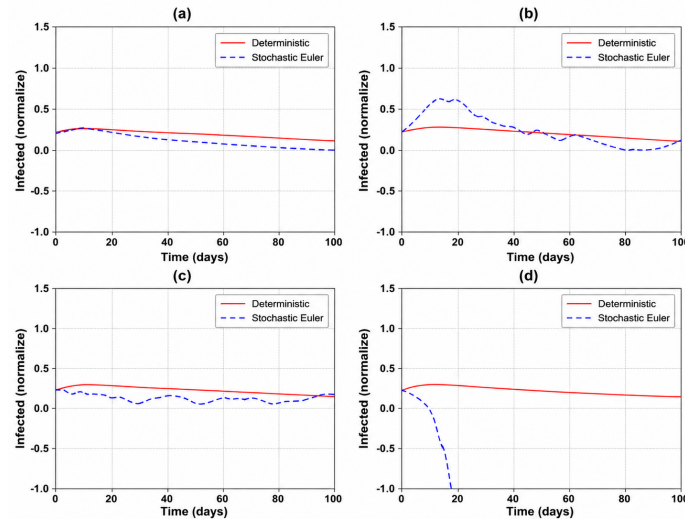


FIGURE 4. Infected humans for (a) $h = 0.1$, (b) $h = 0.7$, (c) $h = 1.5$, and (d) $h = 2$, comparing the deterministic Euler scheme (red line) and stochastic Euler scheme (blue line).

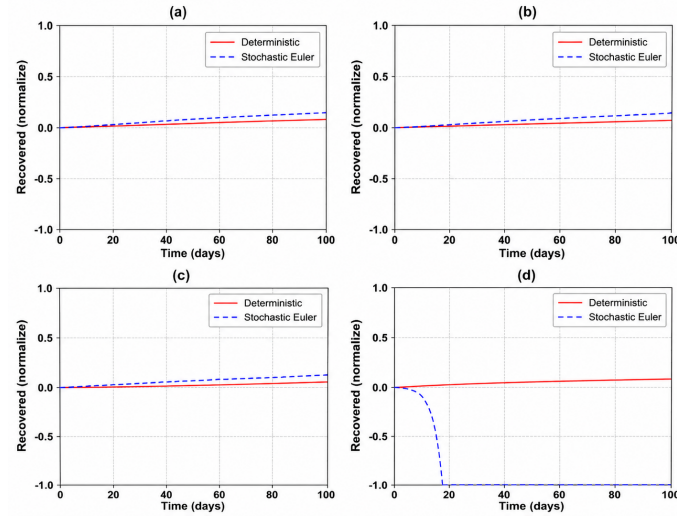


FIGURE 5. Recovered humans for (a) $h = 0.1$, (b) $h = 0.8$, (c) $h = 1.5$, and (d) $h = 2$, comparing the deterministic Euler scheme (red line) and stochastic Euler scheme (blue line).

5. RESULTS AND DISCUSSION

The numerical experiments illustrate that the proposed stochastic COVID-19 framework maintains the fundamental qualitative structure of the deterministic model while introducing realistic fluctuations caused by environmental and epidemiological uncertainty. The

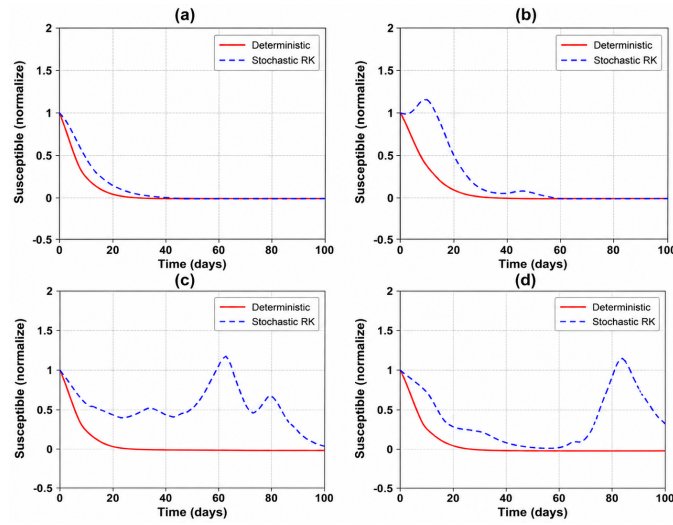


FIGURE 6. Susceptible humans for (a) $h = 5.5$, (b) $h = 6.5$, (c) $h = 7.8$, and (d) $h = 8.5$, comparing the deterministic Runge–Kutta scheme (red line) and stochastic Runge–Kutta scheme (blue line).

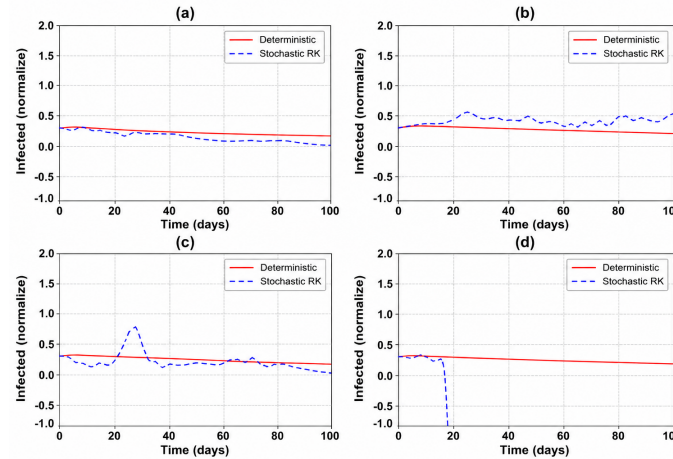


FIGURE 7. Infected humans for (a) $h = 0.1$, (b) $h = 1.1$, (c) $h = 2.1$, and (d) $h = 3.5$, comparing the deterministic Runge–Kutta scheme (red line) and stochastic Runge–Kutta scheme (blue line).

validation using actual infection data from Spain (November 2022 - January 2023) offers empirical evidence for the formulations reliability. Figure 2 illustrates that both deterministic and stochastic trajectories show the same global trend of fewer infected people after large-scale vaccination. The deterministic solution gives a strictly smooth evolution curve

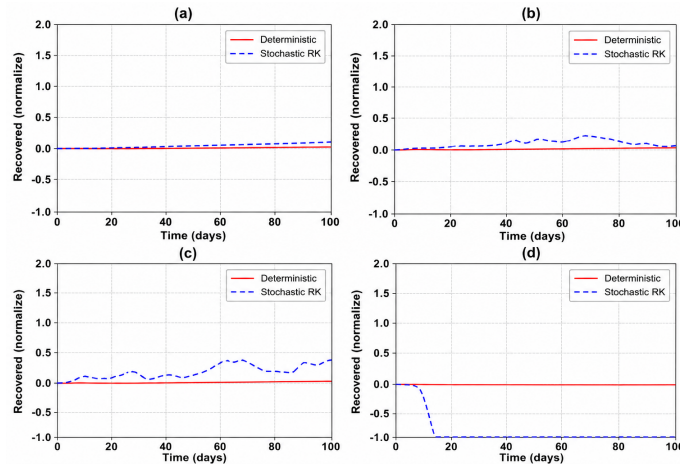


FIGURE 8. Recovered humans for (a) $h = 0.5$, (b) $h = 1.5$, (c) $h = 2.5$, and (d) $h = 3.5$, comparing the deterministic Runge–Kutta scheme (red line) and stochastic Runge–Kutta scheme (blue line).

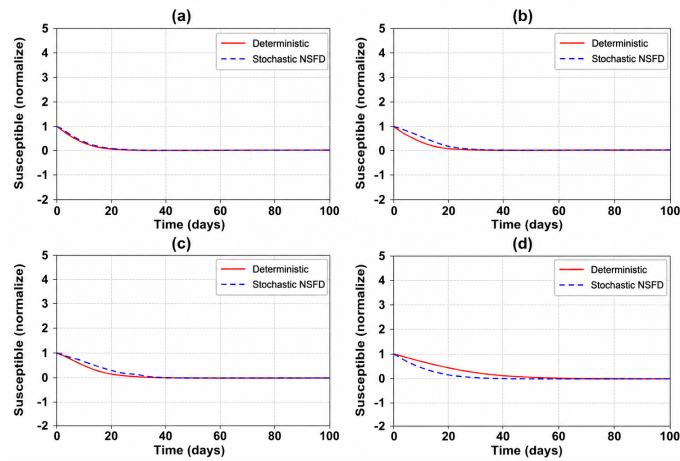


FIGURE 9. Susceptible humans for (a) $h = 0.1$, (b) $h = 1.1$, (c) $h = 2.1$, and (d) $h = 3.1$, comparing the deterministic nonstandard finite difference scheme (red line) and stochastic nonstandard finite difference scheme (blue line).

that shows its average dynamic structure and lack of random perturbations. However, the stochastic solution shows controlled oscillatory deviations around the empirical data. This captures the meantime transformations that occur naturally in the real transmission processes. The changes are not undefined or random values. They come from multiplicative diffusion terms that are based on Brownian motion. In simple terms help to show the

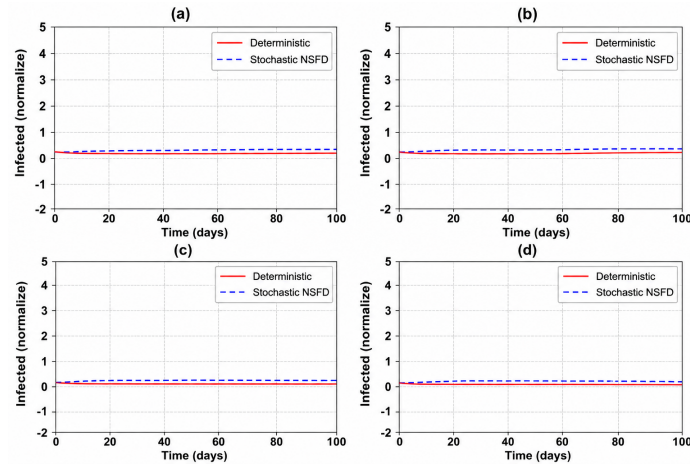


FIGURE 10. Infected humans for (a) $h = 0.1$, (b) $h = 1.1$, (c) $h = 2.1$, and (d) $h = 3.1$, comparing the deterministic nonstandard finite difference scheme (red line) and stochastic nonstandard finite difference scheme (blue line).

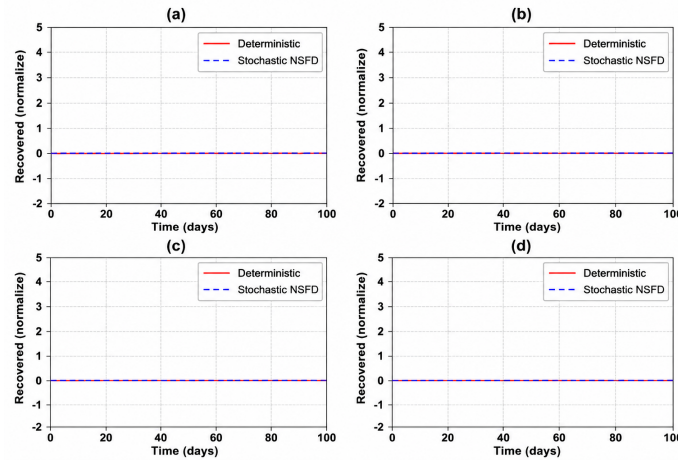


FIGURE 11. Recovered humans for (a) $h = 0.1$, (b) $h = 1.1$, (c) $h = 2.1$, and (d) $h = 3.1$, comparing the deterministic nonstandard finite difference scheme (red line) and stochastic nonstandard finite difference scheme (blue line).

uncertainty in the model, such as differences in how often people come into contact and natural biological variations. The stochastic approach is very close to the standard empirical curve, which suggests that the level of diffusion used is appropriate. It also shows that these random effects do not change the main deterministic trend of the model.

The stability characteristics of the stochastic Euler scheme are illustrated in Figures 3 - 5. For sufficiently small discretization size, particularly " $h = 0.1$ ", the numerical solutions exhibit comparatively stable behaviour with bounded stochastic fluctuation. The susceptible, infected and recovered compartments show consistency, as illustrated in Figures 3 - 5. These results show that the Euler approximation performs comparatively well under small step size. However, when the time step increases, the method starts to lose its stability. In such cases, noticeable oscillations appear, and the solution may move away from its equilibrium, especially for moderate and large values of " h ". This behaviour happens because the Euler method is only conditionally stable in nonlinear stochastic systems that include multiplicative noise. Since the diffusion term depends on the state variables, using large time steps increases the effect of random changes, which can lead to numerical instability. Therefore, although the Euler scheme is simple and easy to use, its accuracy and reliability strongly depend on choosing an appropriate step size. For this reason, it may not be suitable when using large time steps, particularly in long-term epidemic simulations.

The stochastic Runge-Kutta method provides a multi-stage numerical approximation, as seen in Figures 6-8. When the step size is small or moderate, both the deterministic and stochastic Runge-Kutta trajectories show comparatively similar and stable numerical behaviour. In these cases, the solutions remain stable and keep the main characteristics of the continuous model. However, when the step size becomes large, instability starts to appear again. Oscillations can be observed, especially in the infected and recovered groups, and the solutions may begin to diverge. This suggests that even multi-stage numerical methods do not always guarantee stability when multiplicative stochastic effects are present, particularly for large step sizes. This happens because the nonlinear interaction between the drift and diffusion terms increases the numerical error as the step size grows. As a result, although the Runge-Kutta method shows comparatively stable numerical behaviour for small step sizes, it is still sensitive to the choice of discretization in stochastic epidemic models.

A different type of stability behaviour can be seen in the stochastic non-standard finite difference (SNSFD) scheme shown in Figures 9-11. This method is designed to preserve the important properties of the original continuous system by using nonlocal denominator functions and the transformation $\varphi(h) = 1 - e^{-h}$. The numerical results indicate that the SNSFD method is less sensitive to variations in h than the other methods considered. For the tested step sizes, the susceptible, infected, and recovered trajectories exhibit comparatively stable numerical behaviour without the pronounced oscillations observed for the other schemes at larger step sizes. Therefore, the results in Figures 9-11 suggest that the SNSFD method provides comparatively robust numerical behaviour under the simulation conditions considered in this study.

From a dynamical point of view, the stochastic simulations show that the basic reproduction number is still the main factor that determines whether a disease will die out or continue to spread. When the reproduction number is less than one, the results suggest that the system tends to move towards the disease-free state over time, although small random fluctuations may still appear. On the other hand, when the reproduction number is greater than one, the number of infected individuals remains at a positive level, while with ongoing random variations caused by stochastic effects. Vaccination plays an important role by lowering the transmission rate, as it reduces the number of susceptible individuals and changes

how the infected spreads. However, the simulations also show that even with high vaccination coverage, short-term increase in cases can still occur due to random disturbances. This suggests that it is important to consider uncertainty in disease modeling, since relying only on deterministic models may not fully capture the possible variations in infection patterns.

Overall, the findings show that by adding stochastic elements makes the model more realistic while still keeping its basic structure unchanged. The results also indicate that the accuracy of the model depends strongly on the choice of discretisation method. Among the methods tested, the SNSFD approach performs best, as it gives more stable results and better reflects the behaviour of the original continuous stochastic system.

6. CONCLUSION

This study developed a stochastic version of an eight-compartment COVID-19 model that also includes vaccination. The aim was to study both its numerical results and how the disease spreads under uncertainty. Random effects were added to the model using multiplicative diffusion, which helps to represent real-life variations in disease transmission. At the same time, the basic structure of the original deterministic model was kept the same. The model was then tested using real COVID-19 data from Spain. The results show that both deterministic and stochastic trajectories show the same global trend of fewer infected people after large-scale vaccination.

A detailed numerical comparison shows that common stochastic methods, such as the stochastic Euler and stochastic Runge-Kutta schemes, are not always stable. Their performance depends a lot on the choice of time step. When the step size is small, these methods can give accurate results near equilibrium. However, for the large step sizes, the stochastic non-standard finite difference (SNSFD) method remains stable compared with the other methods considered in this study. It keeps the results within limits. These numerical results indicate that the SNSFD method is less sensitive to step size variation under the simulation conditions considered in this study.

The analysis shows that the basic reproductive number is still the most important factor in understanding how the disease spreads, both in deterministic and stochastic models. Vaccination helps to reduce the rate of transmission, but random effects show that the spread of the disease is still uncertain and cannot be predicted exactly. Because of this, including uncertainty in epidemic models makes them more realistic. It also helps policymakers make better decisions and plan more effective public health strategies.

Future research can improve this model by adding more real-world factors. For example, the transmission rate could be allowed to change over time, and different virus variants could be included with their own characteristics. The model can also be extended to study how the disease spreads across different locations and regions. In addition, control strategies can be tested under uncertain conditions to see what works best. These improvements would make stochastic epidemic models more useful for supporting flexible and strong public health policies.

CREDIT AUTHORSHIP CONTRIBUTION'S STATEMENT

Quratulain Malik, Sana Javed, Muhammad Yousaf, Naima Amin, and Cemil Tunç: Conceptualization, Data curation, Formal analysis, Validation, Visualization. Muhammad

Yousaf and Cemil Tunç: Project administration. Muhammad Yousaf and Sana.Javed: Supervision. Quratulain Malik: Writing-original draft, Writing-review and editing. All authors have read and agreed to the published version of the manuscript.

DECLARATIONS

Conflicts of interest. The authors declare that they have no conflicts of interest.

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Data availability. The data used in this study were obtained from Our World in Data that covered Spain from November 2022 to January 2023.
(<https://ourworldindata.org/coronavirus>)

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