Available online at http://scik.org
Commun. Math. Biol. Neurosci. 2023, 2023:130
https://doi.org/10.28919/cmbn/8223
ISSN: 2052-2541

A STOCHASTIC OPTIMAL CONTROL STRATEGY FOR MULTI-STRAIN COVID-19 SPREAD

AHMED ELQADDAOUI[1,*], AMINE EL BHIH[1], HASSAN LAARABI[1], MOSTAFA RACHIK[1], ABDELHADI ABTA[2]

[1]Laboratory of Analysis Modeling and Simulation, Department of Mathematics and Computer Science, Faculty of Sciences Ben M'Sik, Hassan II University, Po Box 7955, Sidi othman, Casablanca, Morocco

[2]Department of Mathematics and Computer Science, Poly-disciplinary Faculty, Cadi Ayyad University, P.O Box 4162, Safi, Morocco

Abstract. In this study, we propose an advanced stochastic mathematical model that delves into the intricate dynamics of multi-strain COVID-19 transmission. By accounting for environmental fluctuations, we introduce white noise into each compartment of the multi-strain system, enriching our understanding of its behavior. Rigorous proofs establish the system's existence and uniqueness, providing a robust foundation for further exploration. We investigate key control measures within the model, specifically focusing on vaccination and targeted treatment for each strain's compartment, as potent strategies to curtail the spread of multi-strain COVID-19. Moreover, our analysis extends to the realm of stochastic optimal control, where we examine the associated optimality conditions of the stochastic maximum Pontryagin. The ultimate goal is to reduce infections through precise control measures, paving the way for evidence-based policies that can effectively manage the pandemic's impact. By offering deep insights into multi-strain COVID-19 propagation, our innovative model contributes significantly to the fight against the virus, guiding the development of proactive strategies and public health interventions.

Keywords: stochastic optimal control; multi-Strain epidemic model; COVID-19; stochastic maximum principle.

2020 AMS Subject Classification: 92C60, 93E20.

*Corresponding author

E-mail address: ahelqaddaoui@gmail.com

Received September 17, 2023

1. INTRODUCTION

Coronaviruses encompass a family of viruses capable of inducing respiratory illnesses in humans. The name "coronavirus" originates from the distinctive crown-like spikes that adorn the surface of the virus. Among these viruses, a specific strain named SARS-CoV-2 is accountable for the development of the disease known as COVID-19. The emergence of this novel strain was initially documented in December 2019 in Wuhan, China, and swiftly disseminated across the globe. COVID-19 can manifest a spectrum of respiratory symptoms, ranging from mild to severe, with potentially fatal outcomes in certain cases. The primary mode of viral entry into the body is through direct contact with infected individuals, including activities such as sneezing, talking, singing, breathing, or through physical interactions like handshakes. Additionally, the virus can spread indirectly via contact with contaminated surfaces or objects that have been in contact with an infected person. Viral evolution is an inherent phenomenon that occurs as viruses disseminate within populations. As the virus undergoes alterations that deviate significantly from the original strain, these modifications are labeled as "variants". To detect and analyze these variants, scientists utilize genetic sequencing methods to scrutinize the viral genetic material. Through comparative analysis of the genetic sequences, researchers can discern noteworthy changes that have transpired in the virus's genetic composition. [Centers for Disease Control and Prevention. (2021). COVID-19: How it spreads. Retrieved from [16] Since the global spread of the SARS-CoV-2 virus, the causative agent of COVID-19, numerous variants have emerged and been identified in multiple countries worldwide [18]. One prominent example is the Omicron variant, scientifically known as variant B.1.1.529. The World Health Organization (WHO) was first notified of the Omicron variant on November 24, 2021, and subsequently classified it as a variant of concern on November 26, 2021, following guidance from the Technical Advisory Group on Virus Evolution [18]. This classification was primarily based on information originating from South Africa, which indicated that the Omicron variant carries a substantial number of mutations and has been associated with detrimental changes in the epidemiology of COVID-19 [1].

The Delta variant, designated as a variant of concern by the World Health Organization (WHO) on May 11, 2021, has become the prevailing strain circulating worldwide. It exhibits

heightened transmissibility compared to earlier virus strains and has contributed to a significant increase in the number of COVID-19 cases and fatalities on a global scale [1].

RELATED WORK

In recent years, there has been a notable upswing in research dedicated to the modeling of infectious diseases [2, 3, 4]. At the forefront of this field is the SIR model, a fundamental framework that sheds light on the intricate dynamics among three pivotal populations: the susceptible (S), the infected (I), and the recovered (R). First proposed by Kermack and Mc Kendricks in 1927 [5], the SIR model has garnered substantial recognition due to its aptitude in capturing the nuanced behavior of infectious diseases. In their research, Jaouad Danane et al. [6] delves into the intricate dynamics of a stochastic model that aims to understand the behavior of COVID-19 when an isolation strategy is implemented. Their study sheds light on the effectiveness of such a strategy in mitigating the spread of the disease. On a related note, R. Aboulaich et al. [7] conduct an in-depth investigation to identify optimal treatment regimens that minimize the overall tumor burden. Their research focuses on a diffusion process and proposes a novel stochastic optimal control model. The study specifically explores the potential of the Bacillus Calmette-Guerin (BCG) bacterium as an immunotherapeutic agent for combating superficial bladder cancer. The findings contribute to advancing our understanding of treatment strategies for this particular form of cancer. In a pioneering investigation by Hassan Laarabi et al. [8], an exceptional exploration was conducted on a mathematical model for a SIR epidemic, encompassing a saturated incidence rate. Their study operates within the realm of nonlinear optimal control, opening up new avenues for innovative approaches. Notably, they employ optimal vaccination strategies to minimize the count of susceptible and infected individuals, while simultaneously maximizing the population of recovered individuals. This comprehensive analysis provides invaluable insights into effective control measures and contributes significantly to the ongoing endeavors in combating epidemics. Abdelfatah Kouidere et al. [9] present an advanced continuous mathematical model that effectively captures the transmission dynamics of COVID-19 virus within the human population. The model comprehensively incorporates various stages, including susceptible, exposed, infected, quarantined, hospitalized, and recovered individuals, providing a holistic representation of the disease's spread. Kassahun Getnet

Mekonen et al. [10] have made a significant contribution by extending the deterministic mathematical model to shed light on the coinfection of COVID-19 and Tuberculosis (TB) through an optimal control framework. They enhance the deterministic model by incorporating four essential control measures: preventive efforts against TB, preventive techniques against COVID-19, infection treatments, and medical care for COVID-19. By integrating these measures, the study offers valuable insights and optimal strategies for effectively managing both diseases. In their discrete mathematical model, Amine El Bhih et al. [11] examined optimal control strategies for combating the spread of Covid-19. Their study focused on six population compartments (SEICWCR) representing susceptible, exposed, infected, infected with complications, infected multimorbidity with complications, and recovered individuals. They proposed an effective approach utilizing four controls: media and education for sensitizing and preventing the disease among susceptible individuals, home quarantine for infected individuals, hospital quarantine for infected individuals with complications, and hospital quarantine with necessary breathing assistance for infected individuals with multimorbidity and complications. In the realm of infectious disease modeling, Masaaki Ishikawa [12] conducted a notable study on stochastic models. His research delved into a population comprising four compartments: susceptible, infected, recovered, and vaccinated individuals. Ishikawa specifically explored the optimal vaccination strategy within the context of stochastic optimal control problems. By addressing the uncertainties inherent in disease dynamics, his work provides valuable insights into effective vaccination planning and management. Further models of problems of optimal control and population dynamics can be seen in [19, 20, 21, 22, 23].

PROBLEM STATEMENT

The new Corona virus appeared at the end of 2019 in China, and then spread throughout the world, and it was not long before several mutated strains appeared for this virus, for example, the mutated strain Delta and the mutated strain Omicron, etc. With the emergence of these mutated strains, the thing that made us predict the emergence of strains new mutant in the future. We assume in this paper that n mutated strains will appear denote by $I_1, I_2, I_3, ..., I_n$. and each strain I_i spreads with infection rate β_i. The propagation of these mutated strains is subject to several environmental fluctuations, so an infection rate β_i will be become $\beta_i + W(t)$ where w(t) wiener

process which describe environment fluctuation, the aim of this paper is controlling the spread of these mutant strains that reduce the number of people infected with each mutated strain and increasing the number of the recovered.

2. DETERMINISTIC MODEL

2.1. The deterministic model without controls. The following proportions $P(t), s(t), i_j(t)$, and $r(t)$ represent respectively the number of the entire population, the number of susceptible to infection, the number of infectious of mutant strain I_j, and the number of recovered at time t.

we assume that the population size is constant which denote by $N(t) = S(t) + \sum_{i=1}^{n} I_i(t) + R(t) = 1$, where $S(t) = \frac{s(t)}{P(t)}$ represent percentage of susceptible to infection at time t, $I_j(t) = \frac{i_j(t)}{P(t)}, j = 1,....,n$ represent the percentage of infectious of mutant strain I_j and $R(t) = \frac{r(t)}{P(t)}$ represent the percentage of recovered people.

The following model is the deterministic model witch describe interactions between the various compartments of this population.

$$\left\{ \begin{array}{l} \frac{dS(t)}{dt} = \Lambda - \Sigma_{i=1}^{n} \beta_i I_i(t) S(t) - \mu S(t). \\ \frac{dI_i(t)}{dt} = \beta_i I_i(t) S(t) - (\gamma_i + \omega_i + \mu) I_i \quad i = 1,2...,n. \\ \frac{dR(t)}{dt} = \Sigma_{i=1}^{n} \gamma_i I_i(t) - \mu R(t). \end{array} \right. \tag{1}$$

where β_i transmission rate the I_i mutant strain , μ death rate (we assume that natural death rate is equal to the birth rate), γ_i recovery rate from I_i mutant strain and ω_i presents the death rate due to I_i mutant strain.

3. A STOCHASTIC MODEL

3.1. The Stochastic model without controls. Because actual infectious diseases include random fluctuations induced by changes in the environment and weather, we evaluate the transmission rate with a random fluctuation. by substituting the transmission rate of each mutant strain with:

$$\beta_i \to \beta_i + \xi_i dW_i(t)$$

where $W_i(t)$ denote the wiener process, we propose the following stochastic model that describe the interactions between the different compartments of population.

$$(2)\quad \begin{cases} dS(t) = \left\{\Lambda - \Sigma_{i=1}^{n}\beta_i I_i(t)S(t) - \mu S(t)\right\}dt - \Sigma_{i=1}^{n}\xi_i I_i(t)S(t)dW_i(t). \\ dI_i(t) = \left\{\beta_i I_i(t)S(t) - (\gamma_i + \omega_i + \mu)I_i\right\}dt + \xi_i I_i(t)S(t)dW_i(t) \qquad i = 1,2...,n. \\ dR(t) = \left\{\Sigma_{i=1}^{n}\gamma_i I_i(t) - \mu R(t)\right\}dt. \end{cases}$$

The quantities $S(0) = S_0, I_i(0) = I_i^0, R(0) = R_0, (i = 1,2....n)$ are the initial conditions, where ξ_i $(i = 1,2,.....n)$ are constants that indicate the intensities of fluctuations caused by in the environment and weather, $(W_i(t))_{t\in[1,T]}$ is an independent Brownian motion.

3.2. Existence and Uniqueness of Solutions and Sufficient Conditions. The state system (2) can be rewritten as follows

$$(3)\quad \begin{cases} dX(t) = f(t,X(t))dt + \sigma(t.X_t)dW(t) \\ X(0) = X_0 \quad gevin. \end{cases}$$

where

$$f(t,X_t) = \begin{pmatrix} \Lambda - \Sigma_{i=1}^{n}\beta_i I_i(t)S(t) - \mu S(t) \\ \beta_1 I_1(t)S(t) - (\gamma_1 + \omega_1 + \mu)I_1 \\ \beta_2 I_2(t)S(t) - (\gamma_2 + \omega_2 + \mu)I_2 \\ \vdots \\ \beta_n I_n(t)S(t) - (\gamma_n + \omega_n + \mu)I_n \\ \Sigma_{i=1}^{n}\gamma_i I_i(t) - \mu R(t) \end{pmatrix} \quad X_t = \begin{pmatrix} S(t) \\ I_1(t) \\ I_2(t) \\ \vdots \\ I_n(t) \\ R(t) \end{pmatrix}$$

and

$$\sigma(t.X_t) = \begin{pmatrix} 0 & -\xi_1 I_1 S & -\xi_2 I_2 S & \dots & -\xi_n I_n S & 0 \\ 0 & \xi_1 I_1 S & 0 & \dots & 0 & 0 \\ 0 & 0 & \xi_2 I_2 S & 0 & \dots & 0 \\ \vdots & \vdots & \ddots & \ddots & \ddots & \vdots \\ 0 & 0 & \dots & 0 & \xi_n I_n S & 0 \\ 0 & 0 & \dots & 0 & 0 & 0 \end{pmatrix}$$

In this section, we prove the existence and uniqueness the solution of the stochastic differential equation (3).

Theorem 3.1. *For any initial condition $X(0) = X_0 \in [0,1]^{n+2}$ the stochastic differential equation (3) has a unique solution.*

Proof. We will make sure that the conditions of theorem [13, 17] are verified:

The function $F(.,.)$ is composed of measurable functions, therefore it is measurable. Similarly, for $\sigma(.,.)$.

1) let $X_t = (S(t), I_1(t), I_2(t), ...I_n(t), R(t)) \in [0,1]^{n+2}$, we have

$$|\sigma(t.X_t)| + \|f(t,X_t)\| = \sqrt{2\sum_{i=1}^{n} \xi_i^2 I_i^2(t) S^2(t)} \quad +$$

$$\sqrt{\left((\Lambda - \sum_{i=1}^{n} \beta_i I_i(t) S(t) - \mu S(t)\right)^2 + \sum_{i=1}^{n} [\beta_i I_i(t) S(t) - (\gamma_i + \omega_i + \mu) I_i]^2 + \left(\sum_{i=1}^{n} \gamma_i I_i(t) - \mu R(t)\right)^2}$$

Let's find a majoration for each of the terms under the radicals.

The first term:

$2\sum_{i=1}^{n} \xi_i^2 I_i^2(t) S^2(t) \ \leq \ 2\sum_{i=1}^{n} \xi_i^2$ because $I_i, S \in [0,1]$

The second term:

$$[(\Lambda - \textstyle\sum_{i=1}^{n} \beta_i I_i(t) S(t) - \mu S(t)]^2 \leq \Lambda^2 + [\sum_{i=1}^{n} \beta_i I_i(t) S(t) + \mu S(t)]^2$$

$$\leq \Lambda^2 + [\textstyle\sum_{i=1}^{n} \beta_i I_i(t) + \mu]^2 S(t)^2$$

$$\leq \Lambda^2 + (n+1)[\textstyle\sum_{i=1}^{n} \beta_i^2 I_i^2(t) + \mu^2] S(t)^2$$

$$\leq \Lambda^2 + (n+1)[\textstyle\sum_{i=1}^{n} \beta_i^2 + \mu^2] S(t)^2 \quad \text{because } I_i \in [0,1]$$

The third term: let $i \in \{, 2, ..., n\}$

$$[\beta_i I_i(t) S(t) - (\gamma_i + \omega_i + \mu) I_i]^2 \leq \beta_i^2 I_i^2(t) S^2(t) + (\gamma_i + \omega_i + \mu)^2 I_i^2$$

$$\leq [\beta_i^2 S^2(t) + (\gamma_i + \omega_i + \mu)^2] I_i^2$$

$$\leq [\beta_i^2 + (\gamma_i + \omega_i + \mu)^2] I_i^2 \quad \textit{where } S_i \in [0,1]$$

The fourth term:

$$[\sum_{i=1}^{n}\gamma_i I_i(t)-\mu R(t)]^2 \leq [\sum_{i=1}^{n}\gamma_i I_i(t)]^2+\mu^2 R(t)^2$$

$$\leq n\sum_{i=1}^{n}\gamma_i^2 I_i(t)^2+\mu^2 R(t)^2$$

$$\leq n\sum_{i=1}^{n}\gamma_i^2+\mu^2 R(t)^2 \quad \text{where } I_i\in[0,1]$$

So we have

$$|\sigma(t.X_t)|+\|f(t,X_t)\| \leq \sqrt{2\sum_{i=1}^{n}\xi_i^2}+$$

$$\sqrt{\Lambda^2+(n+1)[\sum_{i=1}^{n}\beta_i^2+\mu^2]S(t)^2+\sum_{i=1}^{n}[\beta_i^2+(\gamma_i+\omega_i+\mu)^2]I_i^2+n\sum_{i=1}^{n}\gamma_i^2+\mu^2R(t)^2}$$

We set

$$C=max\left\{(n+1)[\sum_{i=1}^{n}\beta_i^2+\mu^2]\,,\,[\beta_i^2+(\gamma_i+\omega_i+\mu)^2]\,,\,\mu^2\,,\,\left(\Lambda^2+n\sum_{i=1}^{n}\gamma_i^2\right)^{\frac{1}{2}}+2\sum_{i=1}^{n}\xi_i^2.\mid i=1,...,n\right\}$$

Then

$$|\sigma(t.X_t)|+\|f(t,X_t)\|\leq C(1+\|X_t\|).$$

We will investigate in following the other condition of theorems [13, 17]:

Let

$X(t)=(S(t),I_1(t),I_2(t),...,I_n(t),R(t))$, *and* $Y(t)=(S^{'}(t),I_1^{'}(t),I_2^{'}(t),...,I_n^{'}(t),R^{'}(t))$; $t\in[0,T]$.

We have

$$\|f(t,X_t)-f(t,Y_t)\|+|\sigma(t.X_t)-\sigma(t.Y_t)|=\left[\left(\sum_{i=1}^{n}\beta_i[I_i^{'}(t)S^{'}(t)-I_i(t)S(t)]+\mu[S^{'}(t)-S(t)]\right)^2\right.$$

$$\left.+\sum_{i=1}^{n}\left((\gamma_i+\omega_i+\mu)[I_i^{'}(t)-I_i]-\beta_i[I_i^{'}(t)S^{'}(t)-I_i(t)S(t)]\right)^2+\left(\mu[R^{'}(t)-R(t)]-\sum_{i=1}^{n}\gamma_i[I_i^{'}(t)-I_i(t)]\right)^2\right]^{\frac{1}{2}}$$

$$+\sqrt{2\sum_{i=1}^{n}\xi_i^2[I_i^{'}(t)S^{'}(t)-I_i(t)S(t)]^2}.$$

We put the expressions as follows, and getting the majoration of each one of them:

$A=\left(\sum_{i=1}^{n}\beta_i[I_i^{'}(t)S^{'}(t)-I_i(t)S(t)]+\mu[S^{'}(t)-S(t)]\right)$

$B_i=\left((\gamma_i+\omega_i+\mu)[I_i^{'}(t)-I_i]-\beta_i[I_i^{'}(t)S^{'}(t)-I_i(t)S(t)]\right).\quad i=1,2,...,n$

$C = \left(\mu[R^{'}(t) - R(t)] - \sum_{i=1}^{n} \gamma_i[I_i^{'}(t) - I_i(t)]\right)$
$E = 2\sum_{i=1}^{n} \xi_i^2[I_i^{'}(t)S^{'}(t) - I_i(t)S(t)]^2.$

For expression A:

$$A = \sum_{i=1}^{n} \beta_i[I_i^{'}(t)S^{'}(t) - I_i(t)S(t)] + \mu[S^{'}(t) - S(t)]$$

$$= \sum_{i=1}^{n} \beta_i[I_i^{'}(t)S^{'}(t) + S^{'}(t)I_i(t) - S^{'}(t)I_i(t) - I_i(t)S(t)] + \mu[S^{'}(t) - S(t)]$$

$$= \sum_{i=1}^{n} \beta_i S^{'}(t)[I_i^{'}(t) - I_i(t)] + \left(\mu + \sum_{i=1}^{n} \beta_i I_i^{'}(t)\right)[S^{'}(t) - S(t)]$$

Then by using the inequality of Cauchy-Schwarz[5] we get:

$$A^2 \leq 2\left[\left(\sum_{i=1}^{n} \beta_i S^{'}(t)[I_i^{'}(t) - I_i(t)]\right)^2 + \left(\left(\mu + \sum_{i=1}^{n} \beta_i I_i(t)\right)[S^{'}(t) - S(t)]\right)^2\right]$$

$$\leq 2\left[n\sum_{i=1}^{n} \beta_i^2 S^{'2}(t)[I_i^{'}(t) - I_i(t)]^2 + (n+1)\left(\mu^2 + \sum_{i=1}^{n} \beta_i^2 I_i^2(t)\right)[S^{'}(t) - S(t)]^2\right]$$

$$\leq 2\left[n\sum_{i=1}^{n} \beta_i^2[I_i^{'}(t) - I_i(t)]^2 + (n+1)\left(\mu^2 + \sum_{i=1}^{n} \beta_i^2)\right)[S^{'}(t) - S(t)]^2\right],$$

$$\textit{because } S^{'}(t),\ I_i(t) \in [0,1].$$

For expressions

$$(B_i\ :\ i = 1,2,...n),\ \ B_i = \left((\gamma_i + \omega_i + \mu)[I_i^{'}(t) - I_i(t)] - \beta_i[I_i^{'}(t)S^{'}(t) - I_i(t)S(t)]\right).$$

We have

$$\beta_i[I_i^{'}(t)S^{'}(t) - I_i(t)S(t)] = \beta_i S^{'}(t)[I_i^{'}(t) - I_i(t)] + \beta_i I_i(t)[S^{'}(t) - S(t)]$$

Then

$$B_i = (\gamma_i + \omega_i + \mu)[I_i^{'}(t) - I_i(t)] - \beta_i S^{'}(t)[I_i^{'}(t) - I_i(t)] - \beta_i I_i(t)[S^{'}(t) - S(t)]$$
$$= \left(\gamma_i + \omega_i + \mu - \beta_i S^{'}(t)\right)[I_i^{'}(t) - I_i(t)] - \beta_i I_i(t)[S^{'}(t) - S(t)]$$

Then

$$\begin{aligned}
B_i^2 &\leq \left(\gamma_i+\omega_i+\mu-\beta_i S^{'}(t)\right)^2 [I_i^{'}(t)-I_i(t)]^2+\beta_i^2 I_i^2(t)[S^{'}(t)-S(t)]^2 \\
&\leq \left((\gamma_i+\omega_i+\mu)^2+\beta_i^2 S^{'2}(t)\right)[I_i^{'}(t)-I_i(t)]^2+\beta_i^2 I_i^2(t)[S^{'}(t)-S(t)]^2 \\
&\leq \left(3(\gamma_i^2+\omega_i^2+\mu^2)+\beta_i^2 S^{'2}(t)\right)[I_i^{'}(t)-I_i(t)]^2+\beta_i^2 I_i^2(t)[S^{'}(t)-S(t)]^2 \\
&\leq \left(3(\gamma_i^2+\omega_i^2+\mu^2)+\beta_i^2\right)[I_i^{'}(t)-I_i(t)]^2+\beta_i^2[S^{'}(t)-S(t)]^2, \textit{ because } S^{'}(t), I_i(t)\in[0,1].
\end{aligned}$$

For expression C we have:

$$\begin{aligned}
C^2 &\leq \mu^2[R^{'}(t)-R(t)]^2+\left(\sum_{i=1}^{n}\gamma_i[I_i^{'}(t)-I_i(t)]\right)^2 \\
&\leq \mu^2[R^{'}(t)-R(t)]^2+n\left(\sum_{i=1}^{n}\gamma_i^2[I_i^{'}(t)-I_i(t)]^2\right)
\end{aligned}$$

For expression E we have

$$\begin{aligned}
E &= 2\sum_{i=1}^{n}\xi_i^2\left([I_i^{'}(t)S^{'}(t)-I_i(t)S^{'}(t)]+[I_i(t)S^{'}(t)-I_i(t)S(t)]\right)^2 \\
&\leq 4\sum_{i=1}^{n}\xi_i^2\left([I_i^{'}(t)S^{'}(t)-I_i(t)S^{'}(t)]^2+[I_i(t)S^{'}(t)-I_i(t)S(t)]^2\right) \\
&\leq 4\sum_{i=1}^{n}\xi_i^2 S^{'2}(t)\left([I_i^{'}(t)-I_i(t)]\right)^2+4\sum_{i=1}^{n}\xi_i^2 I_i^2(t)\left([S^{'}(t)-S(t)]\right)^2 \\
&\leq 4\sum_{i=1}^{n}\xi_i^2\left([I_i^{'}(t)-I_i(t)]\right)^2+4\sum_{i=1}^{n}\xi_i^2\left([S^{'}(t)-S(t)]\right)^2, \textit{ because } S^{'}(t), I_i(t)\in[0,1]
\end{aligned}$$

Let's find the majoration of:

$$\begin{aligned}
A^2+\sum_{i=1}^{n}B_i^2+C^2 &\leq 2n\sum_{i=1}^{n}\beta_i^2[I_i^{'}(t)-I_i(t)]^2+2(n+1)\left(\mu^2+\sum_{i=1}^{n}\beta_i^2\right)[S^{'}(t)-S(t)]^2 \\
&\quad+\sum_{i=1}^{n}\left(3(\gamma_i^2+\omega_i^2+\mu^2)+\beta_i^2\right)[I_i^{'}(t)-I_i(t)]^2+\sum_{i=1}^{n}\beta_i^2[S^{'}(t)-S(t)]^2 \\
&\quad+n\left(\sum_{i=1}^{n}\gamma_i^2[I_i^{'}(t)-I_i(t)]^2\right)+\mu^2[R^{'}(t)-R(t)]^2 \\
&\leq \left[2(n+1)\left(\mu^2+\sum_{i=1}^{n}\beta_i^2\right)+\sum_{i=1}^{n}\beta_i^2\right][S^{'}(t)-S(t)]^2 \\
&\quad+\sum_{i=1}^{n}\left(2n\beta_i^2+3(\gamma_i^2+\omega_i^2+\mu^2)+\beta_i^2+n\gamma_i^2\right)[I_i^{'}(t)-I_i(t)]^2+\mu^2[R^{'}(t)-R(t)]^2
\end{aligned}$$

$$\leq \left[(2n+3)\sum_{i=1}^{n}\beta_i^2+2(n+1)\mu^2\right][S^{'}(t)-S(t)]^2$$

$$+\sum_{i=1}^{n}\left((2n+1)\beta_i^2+(n+3)\gamma_i^2+3(\omega_i^2+\mu^2)\right)[I_i^{'}(t)-I_i(t)]^2+\mu^2[R^{'}(t)-R(t)]^2$$

So we have

$$\|f(t,X_t)-f(t,Y_t)\|+|\sigma(t.X_t)-\sigma(t.Y_t)|=\left(A^2+\sum_{i=1}^{n}B_i^2+C^2\right)^{\frac{1}{2}}+\sqrt{E}.$$

We put

$D=\left[\left((2n+3)\sum_{i=1}^{n}\beta_i^2+2(n+1)\mu^2\right)\vee\left((2n+1)\beta_j^2+(n+3)\gamma_j^2+3(\omega_j^2+\mu^2)\right):j=1,...n\right]^{\frac{1}{2}}$
$\vee\left(2\sqrt{\sum_{i=1}^{n}\xi_i^2}\right)$.

Then

$$\|f(t,X_t)-f(t,Y_t)\|+|\sigma(t.X_t)-\sigma(t.Y_t)\leq D\|X_t-Y_t\|\|.$$

According to the Existence and Uniqueness Theorem [13, 17], the EDS (3) admits a unique solution.

□

3.3. The Stochastic model with controls. So the objective of this paper is controlling the spread of these mutated strains and not letting them get out of control, otherwise there will be an unprecedented environment. to avoid the latter, we add controls to this model. and the previous model will become as shown in the following:

$$(4)\quad \begin{cases} dS(t)=\left\{\Lambda-\Sigma_{i=1}^{n}\beta_iI_i(t)S(t)-u(t)S(t)-\mu S(t)\right\}dt-\Sigma_{i=1}^{n}\xi_iI_i(t)S(t)dW_i(t). \\ dI_i(t)=\{\beta_iI_i(t)S(t)-(\gamma_i+\omega_i+\mu)I_i-v_i(t)I_i(t)\}dt+\xi_iI_i(t)S(t)dW_i(t) \qquad i=1,2...,n. \\ dR(t)=\left\{\Sigma_{i=1}^{n}\gamma_iI_i(t)-\mu R(t)+\Sigma_{i=1}^{n}v_i(t)I_i(t)\right\}dt. \end{cases}$$

where $u(t)$ presents the percentage of vaccinated at time t, and $v_i(t)$ represents the treatment for infected people of mutant strain I_i the role of control $u(t)$ is to protect as many people as possible from infection, and roles of controls $v_i, i=1,...,n$ are to provide treatments for the infected people of each mutant strain I_i.

3.4. A Stochastic Optimal Control Approach.

3.4.1. *The Hamiltonian function and Cost function.* we consider the cost function $J(u,v)$ such that

$$J(u,v) = E\left\{\int_0^{t_f}\left(\sum_{i=1}^{n} A_i I_i(t) - BR(t) + \frac{C}{2}u^2(t) + \frac{1}{2}\sum_{i=1}^{n} D_i v_i^2(t)\right)dt + \frac{1}{2}\sum_{i=1}^{n} K_i I_i^2(t_f)\right\}$$

where A_i, B, D_i, K_i are positive constants. Our objective is to reduce the number of infected persons while also reducing systemic expenses by striving to maximize the number of people recovered from each I_i mutant strain. In other words, we are looking for the optimal control (u^*, v^*) such that:

$$J(u^*, v^*) \leq J(u,v),\ \forall u \in U,\ \forall v \in V.$$

where U and V are admissible controls sets defined as;

$U[0,t_f] = \{u(t) : u(t)\ \mathscr{F}_t - progressively\ measurable,\ u(t) \in [0, u_{max}]\ \forall t \in [0,t_f]\}$

$V[0,t_f] = \{v_i(t) : v_i(t)\ \mathscr{F}_t - progressively\ measurable,\ v_i(t) \in [0, v_i^{max}]\ \forall t \in |0,t_f],\ i = 1,2,.....n\}$

Then we define the Hamiltonian function H as

$H = p_0\left[\Lambda - \sum_{i=1}^{n}\beta_i I_i(t)S(t) - u(t)S(t) - \mu S(t)\right] + \sum_{i=1}^{n} p_i\left[\beta_i I_i(t)S(t) - (\gamma_i + \omega_i + \mu)I_i - v_i(t)I_i(t)\right] +$
$p_{n+1}\left[\sum_{i=1}^{n}\gamma_i I_i(t) - \mu R(t) + \sum_{i=1}^{n} v_i(t)I_i(t)\right] + \left[\sum_{i=1}^{n} A_i I_i(t) + BR(t) + \frac{C}{2}u^2(t) + \frac{1}{2}\sum_{i=1}^{n} D_i v_i^2(t) +\right.$
$\left.\frac{1}{2}\sum_{i=1}^{n} K_i I_i^2(t_f)\right] - q_0\sum_{i=1}^{n}\xi_i I_i(t)S(t) + \sum_{i=1}^{n} q_i \xi_i I_i(t)S(t)$

where $(p_0, p_1, ..., p_{n+1})$ and $(q_0, q_1, ..., q_{n+1})$ are adjoint vectors

we set as $x^*(t) = [S^*(t), I_1^*(t), I_2^*(t), ..., I_n^*(t), R^*(t)]^t$ and $v^* = [v_1^*, v_2^*, ..., v_n^*]^t$.

The following follows from the stochastic maximum principle:

$$dx^*(t) = \frac{\partial H(x^*, u^*, v^*, p, q)}{\partial p}dt + \sigma(x^*(t))dW(t)$$

$$dp(t) = \frac{-\partial H(x^*, u^*, v^*, p, q)}{\partial x}dt + q(t))dW(t)$$

$$H(x^*, u^*, v^*, p, q)) = \min_{(u,v)\in(U,V)} H(x^*, u, v, p, q))$$

After calculation we obtain the fallowing:

$dS^*(t) = \frac{\partial H(x^*,u^*,v^*,p,q)}{\partial p_0}dt + \sigma_0(x^*(t))dW(t)$

$$= \left[\Lambda - \sum_{i=1}^{n}\beta_i I_i^*(t)S(t)^* - u^*(t)S^*(t) - \mu S^*(t)\right]dt - \sum_{i=1}^{n}\xi_i I_i^*(t)S^*(t)dW(t)$$

$$dI^*(t) = [\beta_i I_i^*(t)S(t)^* - (\gamma_i + \xi_i + \mu)I_i^*(t) - v_i^* I_i^*(t)]\,dt + \xi_i I_i^*(t)S(t)^* dW(t)$$

$$(i = 1, 2, ..., n)$$

$$dR^*(t) = [\textstyle\sum_{i=1}^n \gamma_i I_i(t) - \mu R(t) + \sum_{i=1}^n v_i(t)I_i(t)]\,dt$$

$$dp_0(t) = \tfrac{-\partial H(x^*,u^*,v^*,p,q)}{\partial S}dt + q_0(t))dW(t)$$

$$= -\left[p_0\left(-\sum_{i=1}^n \beta_i I_i(t) - u(t) - \mu\right) + \sum_{i=1}^n p_i[\beta_i I_i(t)] - q_0\sum_{i=1}^n \xi_i I_i(t) + \sum_{i=1}^n q_i\xi_i I_i(t)\right]dt$$

$$+q_0(t))dW(t)$$

$$dp_i(t) = \tfrac{-\partial H(x^*,u^*,v^*,p,q)}{\partial I_i}dt + q_i(t))dW(t)$$

$$= -\{-p_0\beta_i S(t) + p_i[\beta_i S(t) - (\gamma_i + \omega_i + \mu) - v_i(t)] + p_{n+1}(\gamma_i + v_i(t)) + A_i - q_0\xi_i S(t) + q_i\xi_i S(t)\}\,dt$$

$$+q_i(t))dW(t),\ i = 1.....n$$

$$dp_{n+1}(t) = \tfrac{-\partial H(x^*,u^*,v^*,p,q)}{\partial R}dt + q_{n+1}(t))dW(t) = (p_{n+1}\mu + B)dt + q_{n+1}(t))dW(t)$$

3.4.2. *Optimal Control Characterization and Necessary Conditions.* We derivative the Hamiltonian function with respect to $u, v_i\ \ i = 1.....n$

$$\frac{\partial H(x^*,u^*,v^*,p,q)}{\partial u} = 0. \qquad \frac{\partial H(x^*,u^*,v^*,p,q)}{\partial v_i} = 0.$$

We obtain $u = \frac{p_0 S^*(t)}{C}$. Therefore

$$u^*(t) = max\left\{min\left\{\frac{p_0 S^*(t)}{C}, u_{max}\right\}, 0\right\}$$

and $v_i(t) = \frac{(p_i - p_{n+1})I^*(t)}{D_i}$. Therefore

$$v_i^*(t) = max\left\{min\left\{\frac{(p_i - p_{n+1})I^*(t)}{D_i}, v_i^{max}\right\}, 0\right\}$$

CONCLUSION

In conclusion, our study introduces a cutting-edge stochastic mathematical model that unravels the intricate dynamics of multi-strain COVID-19 transmission. The incorporation of environmental fluctuations through white noise enriches the model's accuracy and realism, bolstering the credibility of our findings. The rigorous proofs of existence and uniqueness establish a solid foundation for further exploration in this critical area of research. Our analysis of control

measures, including vaccination and targeted treatment, underscores their indispensable role in curbing the spread of multi-strain COVID-19, offering practical solutions for public health officials and policymakers.

Moreover, our investigation into stochastic optimal control provides invaluable insights for designing evidence-based policies that effectively reduce infections. These findings have significant implications for managing the pandemic's impact and safeguarding global health. In summary, our innovative stochastic model makes a substantial contribution to the battle against multi-strain COVID-19, serving as a vital resource for shaping public health strategies and guiding policy development to protect communities worldwide. As the pandemic continues to evolve, the knowledge gained from this study remains crucial in empowering efforts to combat the virus and build resilient healthcare systems for a more secure future.

DATA AVAILABILITY

The disciplinary data used to support the findings of this study have been deposited in the Network Repository (http://www.networkrepository.com).

CONFLICT OF INTERESTS

The authors declare that there is no conflict of interests.

REFERENCES

[1] World Health Organization (WHO), https://www.who.int.

[2] M. Choisy, J. F. Guégan, P. Rohani, Mathematical modeling of infectious diseases dynamics, in: M. Tibayrenc (Ed.), Encyclopedia of Infectious Diseases, 1st ed., Wiley, 2007: pp. 379–404. https://doi.org/10.1002/9780470114209.ch22.

[3] M.Y. Li, An introduction to mathematical modeling of infectious diseases, Springer International Publishing, Cham, 2018. https://doi.org/10.1007/978-3-319-72122-4.

[4] K. Dietz, D. Schenzle, Mathematical models for infectious disease statistics, in: A.C. Atkinson, S.E. Fienberg (Eds.), A Celebration of Statistics, Springer New York, New York, NY, 1985: pp. 167–204. https://doi.org/10.1007/978-1-4613-8560-8_8.

[5] W.O. Kermack, A.G. McKendrick, A contribution to the mathematical theory of epidemics, Proc. R. Soc. Lond. A. 115 (1927), 700–721. https://doi.org/10.1098/rspa.1927.0118.

[6] J. Danane, K. Allali, Z. Hammouch, et al. Mathematical analysis and simulation of a stochastic COVID-19 Lévy jump model with isolation strategy, Results Phys. 23 (2021), 103994. https://doi.org/10.1016/j.rinp.2021.103994.

[7] R. Aboulaich, A. Darouichi, I. Elmouki, et al. A stochastic optimal control model for BCG immunotherapy in superficial bladder cancer, Math. Model. Nat. Phenom. 12 (2017), 99–119. https://doi.org/10.1051/mmnp/201712507.

[8] H. Laarabi, E.H. Labriji, M. Rachik, A. Kaddar, Optimal control of an epidemic model with a saturated incidence rate, Nonlinear Anal.: Model. Control. 17 (2012), 448–459. https://doi.org/10.15388/na.17.4.14050.

[9] A. Kouidere, B. Khajji, A. El Bhih, et al. A mathematical modeling with optimal control strategy of transmission of COVID-19 pandemic virus, Commun. Math. Biol. Neurosci. 2020 (2020), 24. https://doi.org/10.28919/cmbn/4599.

[10] K.G. Mekonen, L.L. Obsu, Mathematical modeling and analysis for the co-infection of COVID-19 and tuberculosis, Heliyon. 8 (2022), e11195. https://doi.org/10.1016/j.heliyon.2022.e11195.

[11] A. El Bhih, Y. Benfatah, A. Kouidere, et al. A discrete mathematical modeling of transmission of COVID-19 pandemic using optimal control, Commun. Math. Biol. Neurosci. 2020 (2020), 75. https://doi.org/10.28919/cmbn/4780.

[12] M. Ishikawa, Stochastic optimal control of an SIR epidemic model with vaccination, in: Proceedings of the 43rd ISCIE International Symposium on Stochastic Systems Theory and Its Applications, 2011, Shiga. https://doi.org/10.5687/sss.2012.57.

[13] B. Øksendal, Stochastic differential equations, in: Stochastic Differential Equations, Universitext, Springer, Berlin, Heidelberg, 2003: pp. 65–84. https://doi.org/10.1007/978-3-642-14394-6_5.

[14] S.S. Dragomir, Discrete inequalities of the Cauchy-Bunyakovsky-Schwarz type, Nova Science Publishers Inc, New York, (2004).

[15] J. Yong, X.Y. Zhou, Stochastic controls: Hamiltonian systems and HJB equations, Springer, New York, 1999.

[16] CDC, How COVID-19 spreads, https://www.cdc.gov/coronavirus/2019-ncov/prevent-getting-sick/how-covid-spreads.html.

[17] X. Mao, Stochastic differential equations and applications, Woodhead Publishing, Chichester, 1997.

[18] WHO, Tracking SARS-CoV-2 variants, https://www.who.int/activities/tracking-SARS-CoV-2-variants.

[19] A. El Bhih, Y. Benfatah, S. Ben Rhila, et al. A spatiotemporal prey-predator discrete model and optimal controls for environmental sustainability in the multifishing areas of Morocco, Discr. Dyn. Nat. Soc. 2020 (2020), 2780651. https://doi.org/10.1155/2020/2780651.

[20] A. El Bhih, Y. Benfatah, M. Rachik, Exact determinantions of maximal output admissible set for a class of semilinear discrete systems, Arch. Control. Sci. 30 (2020), 523–552. https://doi.org/10.24425/ACS.2020.134676.

[21] A. El Bhih, R. Ghazzali, S. Ben Rhila, et al. A discrete mathematical modeling and optimal control of the rumor propagation in online social network, Discr. Dyn. Nat. Soc. 2020 (2020), 4386476. https://doi.org/10.1155/2020/4386476.

[22] A. El Bhih, Y. Benfatah, M. Rachik, et al. Regional optimal harvesting controls of a spatiotemporal prey-predator three species fishery model, Commun. Math. Biol. Neurosci. 2022 (2022), 54. https://doi.org/10.28919/cmbn/7387.

[23] A. El Bhih, Y. Benfatah, A. Ghazaoui, M. Rachik, On the maximal output set of fractional-order discrete-time linear systems, Math. Model. Comput. 9 (2022), 262–277. https://doi.org/10.23939/mmc2022.02.262.