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## Research article

# A computational study of a stochastic fractal-fractional hepatitis B virus infection incorporating delayed immune reactions via the exponential decay 

Maysaa Al Qurashi ${ }^{1,2}$ Saima Rashid ${ }^{3, *}$, Fahd Jarad ${ }^{4,5,6,7, *}$<br>${ }^{1}$ Department of Mathematics, King Saud University, P. O. Box 22452, Riyadh 11495, Saudi Arabia<br>${ }^{2}$ Department of Mathematics, Saudi Electronic University, Riyadh, Saudi Arabia<br>${ }^{3}$ Department of Mathematics, Government College University, Faisalabad 38000, Pakistan<br>${ }^{4}$ Department of Physics, Government College University, Faisalabad 38000, Pakistan<br>${ }^{5}$ Department of Mathmatics, Cankaya University, Ankara, Turkey<br>${ }^{6}$ Department of Medical Research, China Medical University Hospital, China Medical University, Taichung, Taiwan<br>${ }^{7}$ Department of Mathematics, King Abdulaziz University, Jeddah, Saudi Arabia<br>* Correspondence: Email: saimarashid@gcuf.edu.pk, fahd@cankaya.edu.tr.


#### Abstract

Recently, researchers have become interested in modelling, monitoring, and treatment of hepatitis B virus infection. Understanding the various connections between pathogens, immune systems, and general liver function is crucial. In this study, we propose a higher-order stochastically modified delay differential model for the evolution of hepatitis B virus transmission involving defensive cells. Taking into account environmental stimuli and ambiguities, we presented numerical solutions of the fractal-fractional hepatitis B virus model based on the exponential decay kernel that reviewed the hepatitis B virus immune system involving cytotoxic T lymphocyte immunological mechanisms. Furthermore, qualitative aspects of the system are analyzed such as the existence-uniqueness of the non-negative solution, where the infection endures stochastically as a result of the solution evolving within the predetermined system's equilibrium state. In certain settings, infection-free can be determined, where the illness settles down tremendously with unit probability. To predict the viability of the fractal-fractional derivative outcomes, a novel numerical approach is used, resulting in several remarkable modelling results, including a change in fractional-order $\delta$ with constant fractal-dimension $\varpi, \delta$ with changing $\varpi$, and $\delta$ with changing both $\delta$ and $\varpi$. White noise concentration has a significant impact on how bacterial infections are treated.


Keywords: HBV model; Fractal-fractional Caputo-Fabrizio differential operators; existence and uniqueness; qualitative analysis; numerical solution

## 1. Introduction

Hepatitis B virus causes hepatitis B (HB), an extremely fatal liver disease, and is responsible for more than 2 billion chronic infections that have been discovered globally [1,2]. It is a significant issue for wellbeing promotion. It can lead to prolonged liver damage, persistent inflammation, and a significant chance of fatality through hepatocellular carcinoma and encephalopathy [3]. Hepatitis B infestations can only happen if the pathogen can get into the circulatory system and affect the liver (see Figure 1). Once inside the liver, the infection multiplies and emits a significant quantity of fresh pathogens into the interstitial fluid $[4,5]$.


Figure 1. Life cycle of HBV infection; see [3].

However, there are two potential stages of this infectious disease: acute and chronic. HB virus that is severe seems to last less than 6 months, typically. If the illness is severe, the defensive mechanism is expected to eradicate the organism of the pathogen, and the patient should fully recover in the next few weeks. The majority of individuals who develop HB have an active virus. The incubation period for persistent HB is 12 weeks or more. Most newborns who contract HBV during infancy, as well as several children between the ages of one and six, develop a degenerative disease. Individuals having
persistent HBV disease constitute a sizable $2 / 3$ of chronically transmitted individuals. Despite carrying and spreading germs, these individuals do not exhibit any indications of illness [6]. The surviving one-third experienced acute hepatitis, a liver condition that could potentially be incredibly dangerous (see Figure 2). Over 2.4 billion individuals worldwide suffer from recurrent liver problems. HB's severe or long-term effects cause over 600,000 deaths a year [6]. Cytotoxic T lymphocytes (CTLs) can deliberately threaten contaminated hepatocellular through severe HBV infections and contribute to the pathophysiology of liver failure by coordinating various immunological mechanisms; see [6].


Figure 2. Future medical approaches to get rid of HBV infection; see [6].
It is crucial to take into account the impact of temporal constraints on the HBV pathway because intercellular propagation and disease penetration are processes that require to be accomplished within a certain environment [7, 8]. Additionally, the criteria for development and contact are based on the disease's kind and phase, the defensive system's health, and the milieu of the organism wherein the association works [9]. The participant's general lack of well-being affects the environment of the body. One strategy for investigating the effects of internal environmental parameters on the evolution of HBV transmission could be to modify the determinism account of the bacteriophage association to incorporate the randomized pressure in both an incremental and random manner. Numerous studies, including [10-12], have investigated numerical simulations to look into the mechanisms of HBV spreading with sound intensity.

When trying to evaluate the analogous prediction systems, stochastic modelling of highly contagious infection agents has a vital influence and adds a sense of authenticity [13]. In general, various organisms respond in the equivalent habitat to virus-induced designs and highly infectious pathogens, and the consequences can vary. Because the environment is constantly changing, the system's attributes actually oscillate around certain optimum levels [14]. Wang et al. [15] created an insidiously contagious and randomized stochastic HIV infectious framework and studied the results of stationary transmission durability. The researchers [16] addressed how a randomized HBV candida
transmission concept with a significant delay in the propagation factor causes regular eruptions and the stagnant distribution and elimination consequences. In addition, the pathogens will be wiped out if the stochastic procreative quantity is below one, and if the stochastic procreative index is significantly higher than one, the infectious disease will be stochastically persistent with a perturbation theory model generated. According to Sun et al. [17], the presence of a solution for the stochastic highly contagious pathogenic framework of CTL feedback and decentralized delay was investigated. Rihan and Alsakaji [18] presented an investigation of an HBV infection framework, including a delayed immune reaction. For further details on epidemic systems; see [19-21] and the references cited therein.

Due to their involvement in assisting representatives in exploring the concealed characteristics of the complexities of interactive structures in rheological, permeation, machine design, electromagnetic fields, control, remote sensing, and thermodynamics, implementations of deterministic fractional differential equations (DEs) and fractional stochastic DEs motivated by Brownain motion (BM) have historically attracted the majority of attention in potential implementation. The basic differential and integral operators are unable to capture imperfections, but both fractional derivative/integral operators have been recognized as powerful computing tools [22-24]. Furthermore, when simulating physical and experimental events, F-F formulations exhibit diverse and distinct types of variability [25-27]. I think it seems that there are undoubtedly many real-world problems that neither fractal nor fractional interpretations are able to accurately reproduce on an individualized dimension. Researchers realized that novel mathematical procedures were required to replicate extremely complicated formations. Despite the assertion that there is hardly anything novel or transformative, it is difficult to believe that the combination of two existing concepts may produce a groundbreaking procedure. For added complexity, a unique differential formula was first implemented in [28]. This logical statement might be understood as the result of the accumulation of the fractal differentiation of a fractional derivative of a specific model. Evidently, there are three possible readings; it all depends on the kernels [29-32]. The concept was contested and applied to other challenges such as chaotic outgrowths, outbreaks, and diffusion, among others ( [33-36]), and the overwhelming majority of publications provided extremely impressive predicted findings.

In fact, random events are prevalent worldwide. Systems frequently experience random disturbances. Various studies have been conducted on stochastic dynamics; for example, a wide range of scientific theories, including meteorology, accounting, biology, and telecommunication systems, frequently exhibit randomized fluctuations with long-term dependency. In order to analyze fractional stochastic processes, fractional BM employing the Hurst index $H(1 / 2,1)$ has been proposed as an alternative to classical BM [37, 38]. Kerboua et al. [39] investigated the SFDEs with perturbed regulatory frameworks involving fractional BM. Pei and Xu [40] investigated the non-Lipschitz SDEs driven by fractional BM. In 2021, authors [41] presented a novel notion for analyzing and predicting the transmission of COVID-19 throughout Africa and Europe using stochastic and deterministic methods. Alkahtani and Koca [42] contemplated the fractional stochastic SIR system within the fractional calculus technique. Rashid et al. [43] expounded the stochastic F-F tuberculosis model via a nonsingular kernel with random densities.

In the research analysis, we examine the behaviour of fractional stochastic delay DEs of the HBV system involving cell-to-cell propagation and CTLs immunological responsiveness via the fractal-fractional operator based on the exponential decay kernel with random densities, which is
inspired by the aforementioned clinical and mathematical concerns. This biological model governed by fractional BM has not yet been demonstrated in the mainstream. As a result of this reality and in an effort to fill this discrepancy, we commence scientific work on one of these formulae in this article. We include the impact of several time delays (TD) and randomness inside a recipient to furnish a highly authentic scenario for the virus's design phase. In the meantime, existence-uniqueness, stochastic basic reproductive number, and the local stability of disease steady states are investigated in the stochastic context. Numerical results are presented by employing the revolutionary technique proposed by Atangana and Araz [41] in the F-F derivative sense. Graphical illustrations are presented with low random densities, incorporating the fractal-dimension and fractional-order. In a nutshell, we presented the simulation findings with and without control.

## 2. Preliminaries

Before advancing on to the formal description, it is imperative to study certain fundamental F-F operator concepts. Take into account the parameters provided in [28] as well as the functional $\mathbf{w}\left(\mathbf{t}_{1}\right)$, which is continuous and fractal differentiable on $[c, d]$ with fractal-dimension $\varpi$ and fractional-order $\delta$.

Definition 2.1. ( [28]) The F-F operator of $\mathbf{w}\left(\mathbf{t}_{1}\right)$ involving the index kernel in terms of Riemann-Liouville (RL) can be presented as follows for $\delta \in[0,1]$ :

$$
\begin{equation*}
{ }^{F F P} \mathbb{D}_{0, \mathbf{t}_{1}}^{\delta, \pi}\left(\mathbf{w}\left(\mathbf{t}_{1}\right)\right)=\frac{1}{\Gamma(\mathbf{s}-\delta)} \frac{d}{d \mathbf{t}_{1}^{\sigma}} \int_{0}^{\mathbf{t}_{1}}\left(\mathbf{t}_{1}-\mathbf{u}\right)^{s-\delta-1} \mathbf{w}(\mathbf{u}) d \mathbf{u} \tag{2.1}
\end{equation*}
$$

where $\frac{d \mathbf{w}(\mathbf{u})}{d \mathbf{u}^{\bar{\sigma}}}=\lim _{\mathbf{t}_{1} \mapsto \mathbf{u}} \frac{\mathbf{w}\left(\mathbf{t}_{1}\right)-\mathbf{w}(\mathbf{u})}{\mathbf{t}_{1}^{\sigma}-\mathbf{u}^{\bar{\sigma}}}$ and $\mathbf{s}-1<\delta, \varpi \leq \mathbf{s} \in \mathbb{N}$.
Definition 2.2. ( [28]) The F-F operator of $\mathbf{w}\left(\mathbf{t}_{1}\right)$ involving the exponential function kernel in terms of $R L$ can be described as follows for $\delta \in[0,1]$ :

$$
\begin{equation*}
{ }^{F F E} \mathbb{D}_{0, \mathbf{t}_{1}}^{\delta, w}\left(\mathbf{w}\left(\mathbf{t}_{1}\right)\right)=\frac{\mathbb{M}(\delta)}{1-\delta} \frac{d}{d \mathbf{t}_{1}^{\sigma}} \int_{0}^{\mathbf{t}_{1}} \exp \left(-\frac{\delta}{1-\delta}\left(\mathbf{t}_{1}-\mathbf{u}\right)\right) \mathbf{w}(\mathbf{u}) d \mathbf{u} \tag{2.2}
\end{equation*}
$$

such that $\mathbb{M}(0)=\mathbb{M}(1)=1$ having $\delta>0, \varpi \leq \mathbf{s} \in \mathbb{N}$.
Definition 2.3. ( [28]) The corresponding F-F integral operator of (2.1) is stated as:

$$
\begin{equation*}
{ }^{F F P} \mathbf{J}_{0, \mathbf{t}_{1}}^{\delta}\left(\mathbf{w}\left(\mathbf{t}_{1}\right)\right)=\frac{\varpi}{\Gamma(\delta)} \int_{0}^{\mathbf{t}_{1}}\left(\mathbf{t}_{1}-\mathbf{u}\right)^{\delta-1} \mathbf{u}^{\pi-1} \mathbf{w}(\mathbf{u}) d \mathbf{u} \tag{2.3}
\end{equation*}
$$

Definition 2.4. ( [28]) The corresponding F-F integral operator of (2.2) is stated as:

$$
\begin{equation*}
{ }^{F F E} \mathbf{J}_{0, \mathbf{t}_{1}}^{\delta}\left(\mathbf{w}\left(\mathbf{t}_{1}\right)\right)=\frac{\delta \varpi}{\mathbb{M}(\delta)} \int_{0}^{\mathbf{t}_{1}} \mathbf{u}^{\varpi-1} \mathbf{w}(\mathbf{u}) d \mathbf{u}+\frac{\varpi(1-\delta) \mathbf{t}_{1}^{\boldsymbol{\pi}-1} \mathbf{w}\left(\mathbf{t}_{1}\right)}{\mathbb{M}(\delta)} \tag{2.4}
\end{equation*}
$$

## 3. Model configuration

In this study, we reveal a revolutionary system of stochastic delay DEs for Hepatitis B virus replication in a single recipient, despite the fact that the intracellular stage of over expression is not entirely appreciated. We surmise that throughout an HBV infestation, balanced (unexposed) metabolic enzymes can become infectious both by interaction with contaminated hepatocytes and by freshly generated complimentary pathogens. We furthermore suppose that the challenge-contaminated lymphocytes can be especially attacked by the cytotoxic T lymphocytes (CTLs). With the infiltration of a membrane and the ejection of retroviruses and the creation of viruses, there is still an unavoidably intracellular TD (significant-delay). Additionally, TD is necessary to reflect the gestation, which is the duration needed for the creation of fresh pathogens. Furthermore, we offer a delay differential framework to integrate the CTL community alongside infections predicated on the underlying framework of Nowak et al. [44]. A model is used to evaluate the structure

$$
\left\{\begin{array}{l}
\dot{\tilde{\mathbf{H}}}\left(\mathbf{t}_{1}\right)=\lambda-\phi_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)-\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)-\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right),  \tag{3.1}\\
\dot{\mathbf{I}}\left(\mathbf{t}_{1}\right)=\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{2} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \\
\dot{\mathbf{V}}\left(\mathbf{t}_{1}\right)=\omega \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{2}\right)-\phi_{3} \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \\
\dot{\tilde{\mathbf{D}}}\left(\mathbf{t}_{1}\right)=\vartheta-\phi_{4} \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)+\gamma_{4} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right),
\end{array}\right.
$$

where $\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)$, respectively, indicate the hepatocytes that are pure and productive of pathogens, infectious hepatocellular, HB infection pathogens as well as the CTLs. From the first component of the following formula, TD containing $\zeta_{1}$ is applied to estimate the length of period it takes from the first infections of a tissue and the generation of additional vesicles. The other component also incorporates the response time vital for proper hepatocytes to be invaded by malignant hepatocytes interactions before becoming contaminated hepatocellular, whilst $\zeta_{2}$ denotes the time that is required for recently generated particulates to develop before becoming contagious components. Either uncontrolled pathogens attack normal tissue at a pace of $\gamma_{1} \tilde{\mathbf{H}} \tilde{\mathbf{V}}$ (disease transmission mechanism), or viral proteins communicate directly normal tissues at a speed of $\gamma_{2} \tilde{\mathbf{H}} \tilde{\mathbf{I}}$ (cell-to-cell spread mechanism). As a result, the expression " $\gamma_{1} \tilde{\mathbf{H}} \tilde{\mathbf{V}}+\gamma_{2} \tilde{\mathbf{H}} \tilde{\mathbf{I}}$ " denotes the overall disease incidence of susceptible organisms. CTLs generate at a consistent rate of $\eta_{2}$ from the hypothalamus and at a pace of $\gamma_{4} \tilde{\mathbf{I}} \tilde{\mathbf{D}}$ as a consequence of stimulating invading pathogens, and they remove invading pathogens at a rate of $\gamma_{3} \tilde{\mathbf{I}} \tilde{\mathbf{D}}$, where $\omega$ is the rate at which viral proteins producing free radicals infections (see Figure 3).


Figure 3. schematic diagram of HBV infection.

In addition, the complexities of HBV transmission may be impacted by unpredictable perturbations in the mechanism of transmission within the recipient, including fluctuations in climate, emotion, and other endogenous rhythms. Because of this, many researchers have included randomization to determinism in studies of biologic processes to illustrate the influence of environmental heterogeneity, as seen in [44,45].
By incorporating nonlinear disturbance on the spontaneous mortality rate using white noise into every other component of the scheme, we capture the influence of randomness in the host for a more
reasonable position of the virus's progression (3.2). Although the HBV infection model's characteristics are not known in advance, the region to which they correspond can indeed be easily identified. So, we suggest a delayed probabilistic approach of the following:

$$
\left\{\begin{array}{l}
d \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)=\left(\lambda-\phi_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)-\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)-\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right) d \mathbf{t}_{1}+\sigma_{1} \tilde{\mathbf{H}} \mathbb{B}_{1}\left(\mathbf{t}_{1}\right),  \tag{3.2}\\
d \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)=\left(\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right) d \mathbf{t}_{1}+\sigma_{2} \tilde{\mathbf{I}} \mathbb{B}_{2}\left(\mathbf{t}_{1}\right), \\
d \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)=\left(\omega \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{2}\right)-\phi_{3} \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)\right) d \mathbf{t}_{1}+\sigma_{3} \tilde{\mathbf{V}} \mathbb{B}_{3}\left(\mathbf{t}_{1}\right), \\
d \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)=\left(\vartheta-\phi_{4} \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)+\gamma_{4} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right) d \mathbf{t}_{1}+\sigma_{4} \tilde{\mathbf{D}} \mathbb{B}_{4}\left(\mathbf{t}_{1}\right),
\end{array}\right.
$$

depending on the ICs $\tilde{\mathbf{H}}(\delta)=\mu_{1}(\delta), \tilde{\mathbf{I}}(\delta)=\mu_{2}(\delta), \tilde{\mathbf{V}}(\delta)=\mu_{3}(\delta), \tilde{\mathbf{D}}(\delta)=\mu_{4}(\delta)$.
Also, $\delta \in[-\zeta, 0], \zeta=\max \left\{\zeta_{1}, \zeta_{2}\right\}, \mu_{\kappa}(\delta) \in \mathbb{C}, \kappa=1, \ldots, 4 . \mathbb{C}\left([-\zeta, 0], \mathbb{R}_{+}^{4}\right)$ is the collection of Lebesgue integrable functions in this case including $\mathbb{B}_{\kappa}, \kappa=1, \ldots, 4$ is a real-valued standard BM specified on a complete probability space ( $\omega, \mathcal{A}, \mathbb{P}$ ) meeting the basic requirements [46] and $\sigma_{\kappa}, \kappa=1, \ldots, 4$ denote the concentrations of the white noise.

Table 1. Explanation of system's feature.

| Symbols | Explanation |
| :---: | :---: |
| $\lambda$ | Rate of viral hepatocellular development via bone marrow and various tissues |
| $\vartheta$ | Rate at which CTLs are produced in the thymus |
| $\phi_{1}$ | Percentage of uninfectious hepatocytes that naturally die |
| $\phi_{2}$ | Percentage of infectious hepatocytes that naturally die |
| $\phi_{3}$ | Frequency of harmless pathogens dying |
| $\phi_{4}$ | Fatality rate of CTLs |
| $\gamma_{1}$ | Successful viral interaction incidence with healthy hepatocytes |
| $\gamma_{2}$ | Efficient proportion of interaction between healthy and diseased hepatocytes |
| $\gamma_{3}$ | CTLs' efficiency at eradicating infectious hepatocytes |
| $\gamma_{4}$ | CTL development speed as a result of contaminated cells' activation |
| $\omega$ | Speed of spontaneous viral activity in affected tissues |

## 4. Qualitative analysis

In previous decades, the idea of reproduction has been extensively used in epidemiological modelling since it has been recognized as a valuable mathematical tool for evaluating reproduction in a specific illness. According to the concept proposed by Atangana [48], one will identify two components $\mathbf{F}$ and $\tilde{\mathbf{V}}$, then

$$
(\mathbf{F} \tilde{\mathbf{V}}-\lambda \tilde{\mathbf{I}})=0
$$

will be analyzed to generate reproductive number [49]. The component $\mathbf{F}$ is particularly intriguing because it is derived from the nonlinear part of the infected classes.

$$
\frac{\partial}{\partial \tilde{\mathbf{I}}}\left(\frac{\tilde{\mathbf{I}}}{\mathbf{N}}\right)=\frac{[\mathbf{N}-\tilde{\mathbf{I}}]}{\mathbf{N}^{2}}
$$

and

$$
\begin{aligned}
\frac{\partial^{2}}{\partial \tilde{\mathbf{I}}^{2}}\left(\frac{(\mathbf{N}-\tilde{\mathbf{I}})}{\mathbf{N}^{2}}\right) & =-2 \frac{[\mathbf{N}-\tilde{\mathbf{I}}]}{\mathbf{N}^{3}} \\
& =\frac{-2(\tilde{\mathbf{H}}+\tilde{\mathbf{V}}+\tilde{\mathbf{D}})}{(\tilde{\mathbf{H}}+\tilde{\mathbf{I}}+\tilde{\mathbf{V}}+\tilde{\mathbf{D}})^{3}}
\end{aligned}
$$

At disease free equilibrium $\mathcal{E}_{0}=\left(\frac{\lambda}{\phi_{1}}, 0,0, \frac{\vartheta}{\phi_{4}}\right)$, we have

$$
\frac{\partial^{2}}{\partial \tilde{\mathbf{I}}^{2}}\left(\frac{(\mathbf{N}-\tilde{\mathbf{I}})}{\mathbf{N}^{2}}\right)=\frac{-2\left(\tilde{\mathbf{H}}_{0}+\tilde{\mathbf{D}}_{0}\right)}{\left(\tilde{\mathbf{H}}_{0}+\tilde{\mathbf{D}}_{0}\right)^{3}}
$$

Therefore, we have

$$
\mathbf{F}_{\mathcal{A}}=\left[\begin{array}{ll}
\frac{-2\left(\gamma_{1} \tilde{\mathbf{H}}_{+}+\tilde{\mathbf{D}}_{0}\right)}{\left(\tilde{\mathbf{H}}_{0}+\tilde{\mathbf{D}}_{0}\right)^{3}} & 0
\end{array}\right]=\left[\begin{array}{ll}
\frac{-2 \gamma_{1}\left(\frac{\lambda}{1_{1}}+\frac{\theta}{\phi_{4}}\right)}{\left(\frac{\lambda}{\phi_{1}}+\frac{\theta}{\phi_{4}}\right)^{3}} & 0
\end{array}\right]=\left[\begin{array}{ll}
\frac{-2 \gamma_{1}\left(\phi_{1} \phi_{\phi_{4}}\right)^{2}}{\left(\lambda \phi_{4}+\vartheta_{\phi_{1}}\right)^{2}} & 0
\end{array}\right]
$$

Then,

$$
\operatorname{det}\left(\mathbf{F}_{\mathcal{A}} \tilde{\mathbf{V}}^{-1}-\lambda \tilde{\mathbf{I}}\right)=0
$$

gives

$$
\mathcal{A}=\frac{-2 \gamma_{1} \phi_{1}^{2} \phi_{4}^{3}}{\left(\lambda \phi_{4}+\vartheta \phi_{1}\right)^{2}\left(\gamma_{3} \vartheta+\phi_{2} \phi_{4}\right)}<0 .
$$

Also, $\mathcal{A}$ indicates that the expansion will not repeat and will consequently have a single magnitude and wipe out. $\mathcal{A}>0$ indicates that there is sufficient intensity to initiate the regeneration phase, implying that the dispersion will have more than one cycle. Consequently, researchers will supply a strong insight of the aforesaid number.

### 4.1. Existence-uniqueness of the HBV model

In this part, we outline a few prerequisites that will ensure a non-negative solution of the stochastic delay DEs scheme presented in (3.2). This is feasible because a favourable result will exist if the system's coefficients satisfy the growth and Lipschitz assumptions.
Theorem 4.1. Suppose there is a system (3.2) $\left(\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right)$ with ICs having $\mathbf{t}_{1} \geq-\zeta$ and the solution will stay in $\mathbb{R}_{+}^{4}$, almost probably.

Proof. By means of the system (3.2), satisfies the Lipschitz continuous, there exists a peculiar solution $\left(\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right)$ defined on $\left[-\zeta, \zeta_{e}\right)$, where $\zeta_{e}$ signifies the explosion time. In order to demonstrate the solution, we need to to illustrate that $\zeta_{e}=\infty$. Assume that $\Lambda_{0}>0$ be a large enough, thus, we have $\left(\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right)=\left\{\left(\mu_{1}\left(\mathbf{t}_{1}\right), \mu_{2}\left(\mathbf{t}_{1}\right), \mu_{3}\left(\mathbf{t}_{1}\right), \mu_{4}\left(\mathbf{t}_{1}\right)\right): \mathbf{t}_{1} \in(-\zeta, 0)\right\} \in \mathbb{C}\left([-\zeta, 0] ; \mathbb{R}_{+}^{4}\right)$ contained in $\left[\frac{1}{\Lambda_{0}}, \Lambda_{0}\right]$. Introducing the stopping time, so for every $\Lambda \geq \Lambda_{0}$, we have

$$
\zeta_{\Lambda}:=\inf \left\{\mathbf{t}_{1} \in\left[-\zeta, \zeta_{e}\right): \min \left\{\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right\}\right\} \leq \frac{1}{\Lambda}
$$

or equivalently

$$
\zeta_{\Lambda}:=\inf \left\{\mathbf{t}_{1} \in\left[-\zeta, \zeta_{e}\right): \max \left\{\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right\}\right\} \geq \Lambda .
$$

Now letting $\inf \psi=\infty$ and increasing function $\zeta_{\Lambda}$ on $\Lambda$. Also, suppose $\zeta_{\infty}=\lim _{\Lambda \rightarrow \infty} \zeta_{\Lambda}$, then $\zeta_{\infty} \leq \zeta_{e}$ and need to prove $\zeta_{\infty}=\infty$ almost surely. We intend to find that $\zeta_{e}=\infty$ almost surely. If this claim is factually inaccurate, then $\exists$ some constants $\mathbb{T}>0$ and $\epsilon \in(0,1)$ such that $\mathbf{P}\left\{\zeta_{\infty} \leq \mathbb{T}\right\}>\epsilon$. Thus, for an integer $\Lambda_{1} \geq \Lambda_{0}$ such that $\mathbf{P}\left\{\zeta_{\Lambda} \leq \mathbb{T}\right\}>\epsilon, \forall \Lambda \geq \Lambda_{1}$. Introducing a mapping $U_{1}: \mathbb{R}_{+}^{4} \mapsto \mathbb{R}_{+}$as:

$$
\begin{aligned}
U_{1}\left(\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right): & =(\tilde{\mathbf{H}}-1-\ln \tilde{\mathbf{H}})+(\tilde{\mathbf{I}}-1-\ln \tilde{\mathbf{I}})+\Lambda_{2} \tilde{\mathbf{V}}+\Lambda_{2}(\tilde{\mathbf{D}}-1-\ln \tilde{\mathbf{D}}) \\
& +\int_{\mathbf{t}_{1}-\zeta_{1}}^{\mathbf{t}_{1}}\left(\gamma_{1} \tilde{\mathbf{H}}\left(s_{1}\right) \tilde{\mathbf{V}}\left(s_{1}\right)+\gamma_{2} \tilde{\mathbf{H}}\left(s_{1}\right) \tilde{\mathbf{I}}\left(s_{1}\right)\right) d s_{1}+\omega \Lambda_{2} \int_{\mathbf{t}_{1}-\zeta_{1}}^{\mathbf{t}_{1}} \tilde{\mathbf{I}}\left(s_{1}\right) d s_{1},
\end{aligned}
$$

where $\Lambda_{2}$ refer to be a non-negative constant to be determined. In view of the Itô's formula, we have

$$
\begin{aligned}
d U_{1}\left(\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right), \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right)= & \left(1-\frac{1}{\tilde{\mathbf{H}}}\right) d \tilde{\mathbf{H}}+\left(1-\frac{1}{\tilde{\mathbf{I}}}\right) d \tilde{\mathbf{I}}+\Lambda_{2}\left(1-\frac{1}{\tilde{\mathbf{D}}}\right) d \tilde{\mathbf{D}}+\Lambda_{2} \tilde{\mathbf{V}}+\frac{1}{2} \frac{1}{\tilde{\mathbf{H}}^{2}}(d \tilde{\mathbf{H}})^{2} \\
& +\frac{1}{2} \frac{1}{\tilde{\mathbf{I}}^{2}}(d \tilde{\mathbf{I}})^{2}+\Lambda_{2} \frac{1}{2} \frac{1}{\tilde{\mathbf{D}}^{2}}(d \tilde{\mathbf{D}})^{2}+\left(\gamma_{1} \tilde{\mathbf{H}} \tilde{\mathbf{V}}-\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)\right. \\
& \left.+\gamma_{2} \tilde{\mathbf{H}} \tilde{\mathbf{I}}-\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)+\omega \Lambda_{2} \tilde{\mathbf{I}}-\omega \Lambda_{2} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)\right) \\
= & (\tilde{\mathbf{H}}-1) \sigma_{1} \tilde{\mathbf{H}} d \mathbb{B}_{1}+(\tilde{\mathbf{I}}-1) \sigma_{2} \tilde{\mathbf{I}} d \mathbb{B}_{2}+\Lambda_{2}(\tilde{\mathbf{D}}-1) \sigma_{4} \tilde{\mathbf{D}} d \mathbb{B}_{4}+\Lambda_{2} \sigma_{3} \tilde{\mathbf{V}} d \mathbb{B}_{3} \\
& +\left\{\left(\lambda-\phi_{1} \tilde{\mathbf{H}}-\frac{\lambda}{\tilde{\mathbf{H}}_{\mathbf{H}}}+\phi_{1}+\gamma_{1} \tilde{\mathbf{V}}+\gamma_{2} \tilde{\mathbf{I}}+\frac{1}{2} \sigma_{1}^{2}\right)\right. \\
& +\left(\phi_{2}+\gamma_{3} \tilde{\mathbf{D}}-\gamma_{1} \frac{\tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)}{\tilde{\mathbf{I}}}-\gamma_{2} \frac{\tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)}{\tilde{\mathbf{I}}}+\frac{1}{2} \sigma_{2}^{2}\right) \\
& \left.+\Lambda_{2}\left(\omega \tilde{\mathbf{I}}-\phi_{3} \tilde{\mathbf{V}}\right)+\Lambda_{2}\left(\vartheta-\phi_{4} \tilde{\mathbf{D}}+\gamma_{4} \tilde{\mathbf{I}} \tilde{\mathbf{D}}-\frac{\vartheta}{\tilde{\mathbf{D}}}+\phi_{4}-\gamma_{4} \tilde{\mathbf{I}}+\frac{1}{2} \sigma_{4}^{2}\right)\right\} d \mathbf{t}_{1} .
\end{aligned}
$$

Simple computations yield

$$
d U_{1}\left(\mathbf{t}_{1}\right)=\mathcal{H} U_{1} d \mathbf{t}_{1}-\left\{\sigma_{1} d \mathbb{B}_{1}\left(\mathbf{t}_{1}\right)+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)+\sigma_{3} d \mathbb{B}_{3}\left(\mathbf{t}_{1}\right)+\sigma_{4} d \mathbb{B}_{4}\left(\mathbf{t}_{1}\right)\right\}
$$

where $\mathcal{H} U_{1}=\left(\lambda-\phi_{1} \tilde{\mathbf{H}}-\frac{\lambda}{\tilde{\mathbf{H}}}+\phi_{1}+\gamma_{1} \tilde{\mathbf{V}}+\gamma_{2} \tilde{\mathbf{I}}+\frac{1}{2} \sigma_{1}^{2}\right)+\left(\phi_{2}+\gamma_{3} \tilde{\mathbf{D}}-\gamma_{1} \frac{\left.\tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}} \mathbf{(} \mathbf{t}_{1}-\zeta_{1}\right)}{\tilde{\mathbf{I}}}-\gamma_{2} \frac{\tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)}{\mathbf{I}}+\right.$ $\left.\frac{1}{2} \sigma_{2}^{2}\right)+\Lambda_{2}\left(\omega \tilde{\mathbf{I}}-\phi_{3} \tilde{\mathbf{V}}\right)+\Lambda_{2}\left(\vartheta-\phi_{4} \tilde{\mathbf{D}}+\gamma_{4} \tilde{\mathbf{I}} \tilde{\mathbf{D}}-\frac{\vartheta}{\overline{\mathbf{D}}}+\phi_{4}-\gamma_{4} \tilde{\mathbf{I}}+\frac{1}{2} \sigma_{4}^{2}\right)=C$.
Thus, we have

$$
d U_{1}\left(\mathbf{t}_{1}\right) \leq C d \mathbf{t}_{1}-\left\{\sigma_{1} d \mathbb{B}_{1}\left(\mathbf{t}_{1}\right)+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)+\Lambda_{2} \sigma_{3} d \mathbb{B}_{3}\left(\mathbf{t}_{1}\right)+\Lambda_{2} \sigma_{4} d \mathbb{B}_{4}\left(\mathbf{t}_{1}\right)\right\}
$$

Performing integration from 0 to $\zeta_{\Lambda \wedge \Psi}$, it can be deduced that

$$
\begin{aligned}
\int_{0}^{\zeta \Lambda \wedge \psi} d U_{1}\left(\chi\left(\mathbf{t}_{1}\right)\right) \leq & \int_{0}^{\zeta \wedge \wedge \psi} C d \mathbf{t}_{1}-\left\{\int_{0}^{\zeta \Lambda \wedge \Psi} \sigma_{1} d \mathbb{B}_{1}\left(\mathbf{t}_{1}\right)+\int_{0}^{\zeta \Lambda \wedge \Psi} \sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)+\Lambda_{2} \int_{0}^{\zeta \wedge \wedge \psi} \sigma_{3} d \mathbb{B}_{3}\left(\mathbf{t}_{1}\right)\right. \\
& \left.+\Lambda_{2} \int_{0}^{\zeta \wedge \wedge \Psi} \sigma_{4} d \mathbb{B}_{4}\left(\mathbf{t}_{1}\right)\right\}
\end{aligned}
$$

using the fact that $\zeta_{\Lambda \wedge \mathbb{T}}=\min \left\{\zeta_{n}, \mathbf{t}_{1}\right\}$. Implementing the expectation on the aforesaid variants gives
$U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right) \leq U_{1}(\chi(0))+C \int_{0}^{\zeta \Lambda \Lambda T} d \mathbf{t}_{1}-\left\{\int_{0}^{\zeta_{\Lambda \Lambda T}} \sigma_{1} d \mathbb{B}_{1}\left(\mathbf{t}_{1}\right)+\int_{0}^{\zeta_{\Lambda \Lambda T}} \sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)+\Lambda_{2} \int_{0}^{\zeta_{\Lambda \Lambda T}} \sigma_{3} d \mathbb{B}_{3}\left(\mathbf{t}_{1}\right)\right.$

$$
\left.+\Lambda_{2} \int_{0}^{\zeta \Lambda \wedge \mathbb{T}} \sigma_{4} d \mathbb{B}_{4}\left(\mathbf{t}_{1}\right)\right\} .
$$

This implies that

$$
\begin{equation*}
\mathcal{E} U_{1}\left(\chi\left(\zeta_{\wedge \wedge \mathbb{T}}\right)\right) \quad \leq U_{1}(\chi(0))+\mathcal{C} \leq U_{1}(\chi(0))+\mathcal{C} \mathbb{T} . \tag{4.1}
\end{equation*}
$$

As $U_{1}\left(\chi\left(\zeta_{\Lambda \wedge T}\right)\right)>0$, then

$$
\begin{align*}
\mathcal{E} U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right) & =\mathcal{E}\left[U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right)_{\mathbf{x}\left(\zeta_{\Lambda} \leq \mathbb{T}\right)}\right]+\mathcal{E}\left[U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right)_{\mathbf{x}\left(\zeta_{\Lambda}>\mathbb{T}\right)}\right] \\
& \geq \mathcal{E}\left[U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right)_{\mathbf{x}\left(\zeta_{\Lambda} \leq \mathbb{T}\right)}\right] . \tag{4.2}
\end{align*}
$$

Further, for $\zeta_{\Lambda}$, since certain factors of $\chi\left(\zeta_{\Lambda}\right)$, say $\left(\tilde{\mathbf{H}}\left(\zeta_{\Lambda}\right)\right)$ including $0<\tilde{\mathbf{H}}\left(\zeta_{\Lambda}\right) \leq \frac{1}{\Lambda}<1$.
Thus, $U_{1}\left(\chi\left(\zeta_{\Lambda}\right)\right) \geq-\ln \left(\frac{1}{\Lambda}\right)$, this allow us to write $U_{1}\left(\chi\left(\zeta_{\Lambda}\right)\right)=\ln \left(\tilde{\mathbf{H}}\left(\zeta_{\Lambda}\right)\right) \leq \ln \left(\frac{1}{\Lambda}\right)$.
As a result, from (4.2) and the previous expression, we have

$$
\begin{align*}
\mathcal{E} U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right) & \geq \mathcal{E}\left[U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right)_{\mathbf{x}\left(\zeta_{\Lambda} \leq \mathbb{T}\right)}\right] \\
& \geq\left\{-\ln \left(\frac{1}{\Lambda}\right)\right\} . \tag{4.3}
\end{align*}
$$

Combining (4.1)-(4.3), we have

$$
\begin{equation*}
\mathcal{E} U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right) \geq-\ln \left(\frac{1}{\Lambda}\right) \mathbf{P}\left(\zeta_{\Lambda \wedge \mathbb{T}}\right) \tag{4.4}
\end{equation*}
$$

It follows that

$$
\begin{aligned}
\mathbf{P}\left(\zeta_{\Lambda \wedge \mathbb{T}}\right) & \leq \frac{\mathcal{E} U_{1}\left(\chi\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)\right)}{\ln \Lambda} \\
& \leq \frac{U_{1}(\chi(0))+C \mathbb{T}}{\ln \Lambda} .
\end{aligned}
$$

Applying limit sup $\Lambda \mapsto \infty$ on (4.4), $\forall \mathbb{T}>0$, we find

$$
\mathbf{P}\left(\zeta_{\Lambda \wedge \mathbb{T}}\right) \leq 0 \quad \Longrightarrow \lim _{\mathbf{t}_{1} \mapsto \infty} \mathbf{P}\left(\zeta_{\Lambda \wedge \mathbb{T}}\right)=0
$$

This is the desired result.

The average number of subsequent viral infections that a contagious individual causes while they are still dangerous is the primary reproductive component in this scenario. We also want to show that stochastic reproduction $\left(\mathbb{R}_{0}^{s}\right)$ is a special kind of basic reproduction number.

### 4.2. Basic reproduction number $\left(\mathbb{R}_{0}^{s}\right)$

Initially, considering the system's (3.2) second cohort, that is
$d \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)=\left(\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right) d \mathbf{t}_{1}+\sigma_{2} \tilde{\mathbf{I}} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)(4.5)$

Considering the Itô's technique for twice differentiation mapping $f_{1}(\tilde{\mathbf{I}})=\ln (\mathbb{I})$, the Taylor series representation is

$$
d f_{1}\left(\mathbf{t}_{1}, \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right)=\frac{\partial f_{1}}{\partial \mathbf{t}_{1}} d \mathbf{t}_{1}+\frac{\partial f_{1}}{\partial \tilde{\mathbf{I}}} d \tilde{\mathbf{I}}+\frac{1}{2} \frac{\partial^{2} f_{1}}{\partial \tilde{\mathbf{I}}^{2}}\left(d \tilde{\mathbf{I}}^{2}\right)^{2}+\frac{\partial^{2} f_{1}}{\partial \tilde{\mathbf{I}} \partial \mathbf{t}_{1}} d \mathbf{t}_{1} d \tilde{\mathbf{I}}+\frac{1}{2} \frac{\partial^{2} f_{1}}{\partial \mathbf{t}_{1}^{2}}\left(d \mathbf{t}_{1}\right)^{2} .
$$

This implies that

$$
\begin{aligned}
d f_{1}\left(\mathbf{t}_{1}, \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right)= & \frac{1}{\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)}\left\{\left(\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)\right.\right. \\
& \left.\left.-\gamma_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right) d \mathbf{t}_{1}+\sigma_{2} \tilde{\mathbf{I}} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right\} \\
& -\frac{1}{2 \tilde{\mathbf{I}}^{2}\left(\mathbf{t}_{1}\right)}\left\{\left(\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)\right.\right. \\
& \left.\left.-\gamma_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right) d \mathbf{t}_{1}+\sigma_{2} \tilde{\mathbf{I}} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right\}^{2} .
\end{aligned}
$$

It follows that

$$
\begin{aligned}
d f_{1}\left(\mathbf{t}_{1}, \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right)= & \left\{\left(\frac{\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)}{\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)}+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3}\right) d \mathbf{t}_{1}+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right\} \\
& -\frac{1}{2 \tilde{\mathbf{I}}^{2}\left(\mathbf{t}_{1}\right)}\left\{\mathcal{A}_{1}^{2}\left(d \mathbf{t}_{1}\right)^{2}+2 \mathcal{A}_{1} \mathcal{A}_{2} d \mathbf{t}_{1} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)+\mathcal{A}_{2}^{2}\left(d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right)^{2}\right\}
\end{aligned}
$$

where $\mathcal{A}_{1}=\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)$ and $\mathcal{A}_{2}=\sigma_{2} \tilde{\mathbf{I}}$, then (4.6) can be written as

$$
\begin{align*}
d f_{1}\left(\mathbf{t}_{1}, \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right)= & \left\{\left(\frac{\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)}{\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)}+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3}\right) d \mathbf{t}_{1}+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right\} \\
& -\frac{1}{2 \tilde{\mathbf{I}}^{2}\left(\mathbf{t}_{1}\right)}\left\{\mathcal{A}_{2}^{2}\left(d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right)^{2}\right\} \\
= & \left\{\left(\frac{\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)}{\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)}+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3}\right) d \mathbf{t}_{1}+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right\} \\
& -\frac{1}{2 \tilde{\mathbf{I}}^{2}\left(\mathbf{t}_{1}\right)}\left\{\left(\sigma_{2} \tilde{\mathbf{I}}\right)^{2}\right\} d \mathbf{t}_{1} . \tag{4.6}
\end{align*}
$$

As $d \mathbf{t}_{1} \mapsto 0,\left(d \mathbf{t}_{1}\right)^{2}, d \mathbf{t}_{1} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right) \mapsto 0$ and $\left(d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right)^{2}$ can be converted to $d \mathbf{t}_{1}$ (By the variance of Wiener technique), we have

$$
\begin{align*}
d f_{1}\left(\mathbf{t}_{1}, \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right) & =\left\{\left(\frac{\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)}{\left.\tilde{\mathbf{I}} \mathbf{t}_{1}\right)}+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3}\right) d \mathbf{t}_{1}+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right\}-\frac{1}{2}\left(\sigma_{2}\right)^{2} d \mathbf{t}_{1} \\
& =\left\{\left(\frac{\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)}{\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)}+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3}\right) d \mathbf{t}_{1}-\frac{1}{2} \sigma_{2}^{2}\right\} d \mathbf{t}_{1}+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right) . \tag{4.7}
\end{align*}
$$

Taking into consideration the next generation matrices [47] are as follows

$$
\mathbf{F}=\left[\begin{array}{cc}
\frac{\gamma_{2} \lambda}{\frac{\gamma_{1}}{\phi_{1}}} & \frac{\gamma_{1}}{\phi_{1}} \\
0 & 0
\end{array}\right] \text { and } \tilde{\mathbf{V}}=\left[\begin{array}{cc}
\frac{\gamma_{3} \vartheta}{\phi_{4}}+\phi_{2} & 0 \\
-\omega & \phi_{3}
\end{array}\right] \text {. }
$$

Therefore, $\mathbf{F}$ and $\tilde{\mathbf{V}}$ at disease-free equilibrium $\mathcal{E}_{0}=\left(\frac{\lambda}{\phi_{1}}, 0,0, \frac{\vartheta}{\phi_{4}}\right)$, we find

$$
\tilde{\mathbf{V}}^{-1}=\left[\begin{array}{cc}
\frac{\phi_{4}}{\gamma_{3} \vartheta_{4}+\phi_{2} \phi_{4}} & 0 \\
\frac{\phi_{3}\left(\phi_{2} \phi_{4}+\gamma_{3} \vartheta\right)}{} & 1 / \phi_{3}
\end{array}\right] .
$$

Thus, the basic reproduction number is $\mathbb{R}_{0}^{\mathbf{s}}=\rho\left(\mathbf{F} \tilde{\mathbf{V}}^{-1}\right)$ is

$$
\mathbb{R}_{0}^{s}=\frac{\lambda \phi_{4}\left(\omega \gamma_{1}+\phi_{3} \gamma_{2}\right)}{\phi_{1} \phi_{3}\left(\vartheta \gamma_{3}+\phi_{2} \phi_{4}\right)},
$$

which is the required stochastic fundamental reproduction number.

### 4.3. Local stability of diseases-free equilibrium point (DFEP) in stochastic sense

Theorem 4.2. For a community's infection to be eradicated, then $\mathbb{R}_{0}^{\mathbf{s}}<1$.
If $\mathbb{R}_{0}^{\mathbf{s}}<1$, then for any provided ICs $(\tilde{\mathbf{H}}(0), \tilde{\mathbf{V}}(0), \tilde{\mathbf{I}}(0), \tilde{\mathbf{D}}(0))=\left(\tilde{\mathbf{H}}_{0}, \tilde{\mathbf{V}}_{0}, \tilde{\mathbf{I}}_{0}, \mathbf{R}_{0}\right) \in \mathbb{R}_{+}^{4}$. Therefore, $\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)$ admits $\lim _{\mathbf{t}_{1} \mapsto \infty} \sup \frac{\left.\ln \left(\tilde{(T} \mathbf{t}_{1}\right)\right)}{\mathbf{t}_{1}} \leq \phi_{3}\left(\mathbb{R}_{0}^{s}-1\right)$ almost surely.
Proof. Taking into consideration (4.5), we have

$$
d f_{1}\left(\mathbf{t}_{1}, \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right)=\left\{\left(\gamma_{1} \tilde{\mathbf{H}} \tilde{\mathbf{V}}+\gamma_{2} \tilde{\mathbf{H}}-\gamma_{3} \tilde{\mathbf{D}}-\phi_{3}-\frac{1}{2} \sigma_{2}^{2}\right) d \mathbf{t}_{1}+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)\right\} .
$$

It follows that

$$
d \ln (\tilde{\mathbf{I}})=\left(\gamma_{1} \tilde{\mathbf{H}} \tilde{\mathbf{V}}+\gamma_{2} \tilde{\mathbf{H}}-\gamma_{3} \tilde{\mathbf{D}}-\phi_{3}-\frac{1}{2} \sigma_{2}^{2}\right) d \mathbf{t}_{1}+\sigma_{2} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right)
$$

After performing integration, we have

$$
\begin{align*}
\ln (\tilde{\mathbf{I}}) \quad & =\ln \left(\tilde{\mathbf{I}}_{0}\right)+\int_{0}^{\int_{1}}\left(\gamma_{1} \tilde{\mathbf{H}} \tilde{\mathbf{V}}+\gamma_{2} \tilde{\mathbf{H}}-\gamma_{3} \tilde{\mathbf{D}}-\phi_{3}-\frac{1}{2} \sigma_{2}^{2}\right) d \mathbf{t}_{1}+\sigma_{2} \int_{0}^{\mathbf{t}_{1}} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right) \\
& \leq \ln \left(\tilde{\mathbf{I}}_{0}\right)+\underbrace{\left(\gamma_{2} \frac{\lambda}{\phi_{1}}-\gamma_{3} \frac{\vartheta}{\phi_{4}}-\frac{1}{2} \sigma_{2}^{2}-\phi_{3}\right) \mathbf{t}_{1}}_{a t}+\sigma_{2} \int_{0}^{\mathbf{t}_{1}} d \mathbb{B}_{2}\left(\mathbf{t}_{1}\right) \\
& \leq \ln \left(\tilde{\mathbf{I}}_{0}\right)+\left(\gamma_{2} \frac{\lambda}{\phi_{1}}-\gamma_{3} \frac{\vartheta}{\phi_{4}}-\frac{1}{2} \sigma_{2}^{2}-\phi_{3}\right) \mathbf{t}_{1}+\Upsilon\left(\mathbf{t}_{1}\right), \tag{4.8}
\end{align*}
$$

where $\Upsilon\left(\mathbf{t}_{1}\right)=\sigma_{3} \int_{0}^{\mathbf{t}_{1}} d \mathbb{B}_{3}\left(\mathbf{t}_{1}\right)$ is the martingale. therefore, by the strong principal of large values for $\Upsilon\left(\mathbf{t}_{1}\right)$, see [50], we get $\lim _{\mathbf{t}_{1} \mapsto \infty} \sup \frac{\Upsilon\left(\mathbf{t}_{1}\right)}{\mathbf{t}_{1}}=0$ almost probably.
After dividing by $\mathbf{t}_{1}$ and applying limit $\mathbf{t}_{1} \mapsto \infty$, then (4.8) reduces to

$$
\lim _{\mathbf{t}_{1} \mapsto \infty} \sup \frac{\ln (\tilde{\mathbf{I}})}{\mathbf{t}_{1}} \quad \leq \gamma_{2} \frac{\lambda}{\phi_{1}}-\gamma_{3} \frac{\vartheta}{\phi_{4}}-\frac{1}{2} \sigma_{2}^{2}-\phi_{3}
$$

$$
\begin{aligned}
& =\phi_{3}\left(\frac{\lambda \phi_{4}\left(\omega \gamma_{1}+\phi_{3} \gamma_{2}\right)}{\phi_{1} \phi_{3}\left(\vartheta \gamma_{3}+\phi_{2} \phi_{4}\right)}-\frac{1}{2 \phi_{3}} \sigma_{2}^{2}\right) \\
& =\phi_{3}\left(\mathbb{R}_{0}^{s}-1\right)<0
\end{aligned}
$$

This indicates that $\mathbb{R}_{0}^{\mathbf{s}}<1$.
Finally, $\mathbb{R}_{0}^{\mathbf{s}}$ should be smaller than 1 for virus elimination in a population.
Remark 4.1. Under some settings, the solution of framework (3.2) oscillates all around endemic equilibrium of the undisturbed system (3.1) if $\mathbb{R}_{0}^{\mathbf{s}}>1$. This indicates that as long as the concentrations of white noise are low enough, the sickness will endure.

## 5. Numerical illustration of the fractal-fractional stochastic HBV model

In what follows, the framework is further expanded to $\mathrm{F}-\mathrm{F}$ derivative operators.
Firstly, we present the Caputo-Fabrizio F-F derivative for the classical derivative formulation. As a result, the stochastic F-F framework will be quantitatively determined employing the numerical technique described previously. This type of system is presented by

$$
\begin{align*}
& \left({ }_{0}^{\mathbf{F F E}} \mathbb{D}_{\mathbf{t}_{1}}^{\delta, \sigma} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)=\left(\lambda-\phi_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)-\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)-\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right)+\sigma_{1} \mathbb{G}_{1}\left(\mathbf{t}_{1}, \tilde{\mathbf{H}}\right) \mathbb{B}_{1}\left(\mathbf{t}_{1}\right),\right. \\
& { }_{0}^{\mathbf{F F E}} \mathbb{D}_{\mathbf{t}_{1}}^{\delta_{\varpi} \varpi} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)=\left(\gamma_{1} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{V}}\left(\mathbf{t}_{1}-\zeta_{1}\right)+\gamma_{2} \tilde{\mathbf{H}}\left(\mathbf{t}_{1}-\zeta_{1}\right) \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{1}\right)-\gamma_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)-\phi_{3} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)\right) \\
& +\sigma_{2} \mathbb{G}_{2}\left(\mathbf{t}_{1}, \tilde{\mathbf{I}}\right) \mathbb{B}_{2}\left(\mathbf{t}_{1}\right),  \tag{5.1}\\
& { }_{0}^{\mathbf{F F E}} \mathbb{D}_{\mathbf{t}_{1}}^{\delta, \sigma} \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)=\left(\omega \tilde{\mathbf{I}}\left(\mathbf{t}_{1}-\zeta_{2}\right)-\phi_{3} \tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)\right)+\sigma_{3} \mathbb{G}_{3}\left(\mathbf{t}_{1}, \tilde{\mathbf{V}}\right) \mathbb{B}_{3}\left(\mathbf{t}_{1}\right), \\
& { }_{0}^{\mathbf{F F E}}{\underset{D}{\mathbf{D}_{1}}}_{\delta, \varpi}^{\mathbf{D}_{1}}\left(\mathbf{t}_{1}\right)=\left(\vartheta-\phi_{4} \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)+\gamma_{4} \tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right) \tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)\right)+\sigma_{4} \mathbb{G}_{4}\left(\mathbf{t}_{1}, \tilde{\mathbf{D}}\right) \mathbb{B}_{4}\left(\mathbf{t}_{1}\right) .
\end{align*}
$$

For $t_{\mathfrak{m}+1}=(\mathfrak{m}+1) \Delta \mathbf{t}_{1}$, then we the aforementioned system can be integrated as follows

$$
\begin{aligned}
& \tilde{\mathbf{H}}_{\mathfrak{m}+1} \quad=\tilde{\mathbf{H}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \varpi \mathbf{t}_{1}{ }_{\mathfrak{m}+1}^{\varpi-1}\left[\begin{array}{l}
\tilde{\mathbf{H}}^{*}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{H}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{V}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{D}}_{\mathfrak{m}+1}^{\mathbf{q}}\right) \\
+\sigma_{1} \mathbb{G}_{1}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{H}}_{\mathfrak{m}+1}^{\mathbf{q}}\right)\left(\mathbb{B}_{1}\left(\mathbf{t}_{1 \mathfrak{m}+1}\right)-\mathbb{B}_{1}\left(\mathbf{t}_{1 \mathfrak{m}}\right)\right)
\end{array}\right] \\
& +\frac{\delta \varpi}{\mathbb{M}(\delta)} \sum_{\ell=0}^{\mathfrak{m}}\left[\int_{\mathbf{t}_{1 \ell}}^{\mathbf{t}_{1 \ell+1}} \varrho^{\delta-1} \tilde{\mathbf{H}}^{*}(\varrho, \tilde{\mathbf{H}}, \tilde{\mathbf{I}}, \tilde{\mathbf{V}}, \tilde{\mathbf{D}}) d \varrho+\int_{\mathbf{t}_{1 \ell}}^{\mathbf{t}_{\ell+1}} \varrho^{\delta-1} \sigma_{1} \mathbb{G}_{1}(\varrho, \tilde{\mathbf{H}}) d \mathbb{B}_{1} \varrho\right], \\
& \tilde{\mathbf{I}}_{\mathfrak{m}+1} \quad=\tilde{\mathbf{I}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \varpi \mathbf{t}_{1 \mathfrak{m}+1}^{\varpi-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{I}}^{*}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{H}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{V}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{D}}_{\mathfrak{m}+1}^{\mathbf{q}}\right) \\
+\sigma_{2} \mathbb{G}_{2}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathbf{q}}\right)\left(\mathbb{B}_{2}\left(\mathbf{t}_{1 \mathfrak{m}+1}\right)-\mathbb{B}_{2}\left(\mathbf{t}_{1 \mathfrak{m}}\right)\right)
\end{array}\right]\right. \\
& +\frac{\delta \varpi}{\mathbb{M}(\delta)} \sum_{\ell=0}^{\mathfrak{m}}\left[\int_{\mathbf{t}_{1 \ell}}^{\mathbf{t}_{1 \ell+1}} \varrho^{\delta-1} \tilde{\mathbf{I}}^{*}(\varrho, \tilde{\mathbf{H}}, \tilde{\mathbf{I}}, \tilde{\mathbf{V}}, \tilde{\mathbf{D}}) d \varrho+\int_{\mathbf{t}_{1 \ell}}^{\mathbf{t}_{1 \ell+1}} \varrho^{\delta-1} \sigma_{2} \mathbb{G}_{2}(\varrho, \tilde{\mathbf{I}}) d \mathbb{B}_{2} \varrho\right], \\
& \tilde{\mathbf{V}}_{\mathfrak{m}+1}=\tilde{\mathbf{V}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \varpi \mathbf{t}_{1}{ }_{\mathfrak{m}+1}^{\varpi-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{V}}^{*}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{V}}_{\mathfrak{m}+1}^{\mathbf{q}}\right) \\
+\sigma_{3} \mathbb{G}_{3}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{V}}_{\mathfrak{m}+1}^{\mathbf{q}}\right)\left(\mathbb{B}_{3}\left(\mathbf{t}_{1 \mathfrak{m}+1}\right)-\mathbb{B}_{3}\left(\mathbf{t}_{1 \mathfrak{m}}\right)\right)
\end{array}\right]\right.
\end{aligned}
$$

$$
\begin{aligned}
& +\frac{\delta \varpi}{\mathbb{M}(\delta)} \sum_{\ell=0}^{\mathbf{m}}\left[\int_{\mathbf{t}_{1 \ell}}^{\mathbf{t}_{1+1+1}} \varrho^{\delta-1} \tilde{\mathbf{V}}^{*}(\varrho, \tilde{\mathbf{I}}, \tilde{\mathbf{V}}) d \varrho+\int_{\mathbf{t}_{1 \ell}}^{\mathbf{t}_{1+1}} \varrho^{\delta-1} \sigma_{3} \mathbb{G}_{3}(\varrho, \tilde{\mathbf{V}}) d \mathbb{B}_{3} \varrho\right], \\
& \tilde{\mathbf{D}}_{\mathrm{m}+1}=\tilde{\mathbf{D}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \varpi \mathbf{t}_{1 \mathrm{~m}+1}^{\tau-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{D}}^{*}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathrm{q}}, \tilde{\mathbf{D}}_{\mathrm{m}+1}^{\mathrm{q}}\right) \\
+\sigma_{4} \mathbb{G}_{4}\left(\mathbf{t}_{1 m+1}, \tilde{\mathbf{D}}_{\mathrm{m}+1}^{\mathrm{q}}\right)\left(\mathbb{B}_{4}\left(\mathbf{t}_{1 \mathrm{~m}+1}\right)-\mathbb{B}_{4}\left(\mathbf{t}_{1 m}\right)\right)
\end{array}\right]\right. \\
& +\frac{\delta \varpi}{\mathbb{M}(\delta)} \sum_{\ell=0}^{\mathfrak{m}}\left[\int_{\mathbf{t}_{\ell \ell}}^{\mathbf{t}_{1+1}} \varrho^{\delta-1} \tilde{\mathbf{D}}^{*}(\varrho, \tilde{\mathbf{I}}, \tilde{\mathbf{D}}) d \varrho+\int_{\mathbf{t}_{\ell \ell}}^{\mathbf{t}_{1+1}} \varrho^{\delta-1} \sigma_{4} \mathbb{G}_{4}(\varrho, \tilde{\mathbf{D}}) d \mathbb{B}_{4} \varrho\right] .
\end{aligned}
$$

Utilizing the fact of polynomials in the aforesaid system, we find

$$
\begin{aligned}
& \tilde{\mathbf{I}}_{\mathfrak{m}+1}=\tilde{\mathbf{I}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \varpi \mathbf{t}_{\mathbf{t}_{m+1}(\boldsymbol{m - 1}}\left[\left\{\begin{array}{l}
\tilde{\mathbf{I}}^{*}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{H}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{V}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{D}}_{\mathfrak{m}+1}^{\mathbf{q}}\right) \\
+\sigma_{2} \mathbb{G}_{2}\left(\mathbf{t}_{1 m+1}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathrm{q}}\right)\left(\mathbb{B}_{2}\left(\mathbf{t}_{1 m+1}\right)-\mathbb{B}_{2}\left(\mathbf{t}_{1 m}\right)\right)
\end{array}\right]\right.
\end{aligned}
$$

$$
\tilde{\mathbf{D}}_{\mathfrak{m}+1} \quad=\tilde{\mathbf{D}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \varpi \mathbf{t}_{1_{\mathrm{m}+1}}^{\omega-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{D}}^{*}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathbf{q}}, \tilde{\mathbf{D}}_{\mathfrak{m}+1}^{\mathrm{q}}\right) \\
+\sigma_{4} \mathbb{G}_{4}\left(\mathbf{t}_{1 \mathrm{~m}+1}, \tilde{\mathbf{D}}_{\mathrm{m}+1}^{\mathrm{q}}\right)\left(\mathbb{B}_{4}\left(\mathbf{t}_{1 m+1}\right)-\mathbb{B}_{4}\left(\mathbf{t}_{1 m}\right)\right)
\end{array}\right]\right.
$$

Furthermore, we can write

$$
\begin{aligned}
& \tilde{\mathbf{H}}_{m+1}=\tilde{\mathbf{H}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \varpi \mathbf{t}_{1 \mathrm{~m}+1}^{\tau-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{H}}^{*}\left(\mathbf{t}_{1 \mathrm{~m}+1}, \tilde{\mathbf{H}}_{\mathrm{m}+1}^{\mathrm{q}}, \tilde{\mathbf{I}}_{\mathrm{m}+1}^{\mathrm{q}}, \tilde{\mathbf{V}}_{\mathrm{m}+1}^{\mathrm{q}}, \tilde{\mathbf{D}}_{\mathrm{m}+1}^{\mathrm{q}}\right) \\
+\sigma_{1} \mathbb{G}_{1}\left(\mathbf{t}_{1 m+1}, \tilde{\mathbf{H}}_{\mathrm{m}+1}^{\mathrm{q}}\right)\left(\mathbb{B}_{1}\left(\mathbf{t}_{1 m+1}\right)-\mathbb{B}_{1}\left(\mathbf{t}_{1 m}\right)\right)
\end{array}\right]\right.
\end{aligned}
$$

$$
\tilde{\mathbf{V}}_{\mathfrak{m}+1}=\tilde{\mathbf{V}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \varpi \mathbf{t}_{1 \mathrm{~m}+1}^{\pi-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{V}}^{*}\left(\mathbf{t}_{1 \mathrm{~m}+1}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathrm{q}}, \tilde{\mathbf{V}}_{\mathfrak{m}+1}^{\mathbf{q}}\right) \\
+\sigma_{3} \mathbb{G}_{3}\left(\mathbf{t}_{1 \mathrm{~m}+1}, \tilde{\mathbf{V}}_{\mathfrak{m}+1}^{\mathrm{q}}\right)\left(\mathbb{B}_{3}\left(\mathbf{t}_{1 \mathrm{~m}+1}\right)-\mathbb{B}_{3}\left(\mathbf{t}_{1 \mathrm{~m}}\right)\right)
\end{array}\right]\right.
$$

where

$$
\begin{aligned}
& \tilde{\mathbf{H}}_{\mathrm{m}+1}^{\mathbf{q}} \quad=\tilde{\mathbf{H}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \mathbf{t}_{1_{\mathrm{m}+1}}^{\omega-1}\left[\begin{array}{l}
\tilde{\mathbf{H}}^{*}\left(\mathbf{t}_{1 \mathrm{~m}}, \tilde{\mathbf{H}}_{\mathrm{m}}, \tilde{\mathbf{I}}_{\mathfrak{m}}, \tilde{\mathbf{V}}_{\mathrm{m}}, \tilde{\mathbf{D}}_{\mathrm{m}}\right) \\
+\varpi \sigma_{1} \mathbb{G}_{1}\left(\mathbf{t}_{1 \mathrm{~m}+1}, \tilde{\mathbf{H}}_{\mathfrak{m}+1}\right)\left(\mathbb{B}_{1}\left(\mathbf{t}_{1 \mathrm{~m}+1}\right)-\mathbb{B}_{1}\left(\mathbf{t}_{1 \mathrm{~m}}\right)\right)
\end{array}\right] \\
& +\frac{\delta \varpi}{\mathbb{M}(\delta)} \sum_{\ell=0}^{m-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{H}}^{*}\left(\mathbf{t}_{1 m}, \tilde{\mathbf{H}}_{m}, \tilde{\mathbf{I}}_{\mathfrak{m}}, \tilde{\mathbf{V}}_{\mathfrak{m}}, \tilde{\mathbf{D}}_{\mathfrak{m}}\right) \frac{\mathbf{h}^{\sigma}}{\sigma}\left((\ell+1)^{\sigma}-\ell^{\sigma}\right) \\
+\sigma_{1} \mathbb{G}_{1}\left(\mathbf{t}_{1 m}, \tilde{\mathbf{H}}_{\mathrm{m}}\right)\left(\mathbb{B}_{1}\left(\mathbf{t}_{1 m+1}\right)-\mathbb{B}_{1}\left(\mathbf{t}_{1 m}\right)\right) \frac{\mathbf{h}^{\sigma}}{\sigma}\left((\ell+1)^{\sigma}-\ell^{\sigma}\right)
\end{array}\right],\right. \\
& \tilde{\mathbf{I}}_{\mathfrak{m}+1}^{\mathbf{q}} \quad=\tilde{\mathbf{I}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \mathbf{t}_{1_{m+1}^{\pi-1}}\left[\left\{\begin{array}{l}
\left(\tilde{\mathbf{I}}^{*}\left(\mathbf{t}_{1 m}, \tilde{\mathbf{H}}_{\mathfrak{m}}, \tilde{\mathbf{I}}_{\mathfrak{m}}, \tilde{\mathbf{V}}_{\mathfrak{m}}, \tilde{\mathbf{D}}_{\mathfrak{m}}\right)\right. \\
+\varpi \sigma_{2} \mathbb{G}_{2}\left(\mathbf{t}_{1 m+1}, \tilde{\mathbf{I}}_{\mathfrak{m}+1}\right)\left(\mathbb{B}_{2}\left(\mathbf{t}_{1 m+1}\right)-\mathbb{B}_{2}\left(\mathbf{t}_{1 m}\right)\right)
\end{array}\right]\right. \\
& +\frac{\delta \varpi}{\mathbb{M}(\delta)} \sum_{\ell=0}^{m-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{I}}^{*}\left(\mathbf{t}_{1 m}, \tilde{\mathbf{H}}_{\mathfrak{m}}, \tilde{\mathbf{I}}_{\mathfrak{m}}, \tilde{\mathbf{V}}_{\mathfrak{m}}, \tilde{\mathbf{D}}_{\mathfrak{m}}\right) \frac{\mathbf{h}^{\sigma}}{\sigma}\left((\ell+1)^{\sigma}-\ell^{\sigma}\right) \\
+\sigma_{2} \mathbb{G}_{2}\left(\mathbf{t}_{1 \mathrm{~m}}, \tilde{\mathbf{I}}_{\mathfrak{m}}\right)\left(\mathbb{B}_{2}\left(\mathbf{t}_{1 \mathrm{~m}+1}\right)-\mathbb{B}_{2}\left(\mathbf{t}_{1 \mathrm{~m}}\right)\right) \frac{\mathbf{h}^{\sigma}}{\sigma}\left((\ell+1)^{\sigma}-\ell^{\sigma}\right)
\end{array}\right],\right.
\end{aligned}
$$

$$
\begin{aligned}
& +\frac{\delta \varpi}{\mathbb{M}(\delta)} \sum_{\ell=0}^{\mathfrak{m}-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{V}}^{*}\left(\mathbf{t}_{1 \mathrm{~m}}, \tilde{\mathbf{I}}_{\mathfrak{m}}, \tilde{\mathbf{V}}_{\mathfrak{m}}\right) \frac{\mathbf{h}^{\pi}}{\sigma}\left((\ell+1)^{\sigma}-\ell^{\sigma}\right) \\
+\sigma_{3} \mathbb{G}_{3}\left(\mathbf{t}_{1 \mathrm{~m}}, \tilde{\mathbf{V}}_{\mathrm{m}}\right)\left(\mathbb{B}_{3}\left(\mathbf{t}_{1 \mathrm{~m}+1}\right)-\mathbb{B}_{3}\left(\mathbf{t}_{1 \mathrm{~m}}\right)\right) \frac{\mathbf{h}^{\sigma}}{\sigma}\left((\ell+1)^{\sigma}-\ell^{\sigma}\right)
\end{array}\right],\right. \\
& \tilde{\mathbf{D}}_{\mathfrak{m}+1}^{\mathbf{q}} \quad=\tilde{\mathbf{D}}_{0}+\frac{(1-\delta) \delta \varpi}{\mathbb{M}(\delta)} \mathbf{t}_{1_{\mathfrak{m}+1}{ }^{\sigma-1}}\left[\begin{array}{l}
\tilde{\mathbf{D}}^{*}\left(\mathbf{t}_{1 m}, \tilde{\mathbf{I}}_{\mathfrak{m}}, \tilde{\mathbf{D}}_{\mathfrak{m}}\right) \\
+\varpi \sigma_{4} \mathbb{G}_{4}\left(\mathbf{t}_{1 \mathfrak{m}+1}, \tilde{\mathbf{D}}_{\mathfrak{m}+1}\right)\left(\mathbb{B}_{4}\left(\mathbf{t}_{1 \mathfrak{m}+1}\right)-\mathbb{B}_{4}\left(\mathbf{t}_{1 m}\right)\right)
\end{array}\right] \\
& +\frac{\delta \varpi}{\mathbb{M}(\delta)} \sum_{\ell=0}^{\mathfrak{m}-1}\left[\left\{\begin{array}{l}
\tilde{\mathbf{D}}^{*}\left(\mathbf{t}_{1 \mathrm{~m}}, \tilde{\mathbf{I}}_{\mathrm{m}}, \tilde{\mathbf{D}}_{\mathrm{m}}\right) \frac{\mathbf{h}^{\pi}}{\sigma}\left((\ell+1)^{\sigma}-\ell^{\sigma}\right) \\
+\sigma_{4} \mathbb{G}_{4}\left(\mathbf{t}_{1 \mathrm{~m}}, \tilde{\mathbf{D}}_{\mathrm{m}}\right)\left(\mathbb{B}_{4}\left(\mathbf{t}_{1 \mathrm{~m}+1}\right)-\mathbb{B}_{4}\left(\mathbf{t}_{1 \mathrm{~m}}\right)\right) \frac{\mathbf{h}^{\pi}}{\sigma}\left((\ell+1)^{\sigma}-\ell^{\sigma}\right)
\end{array}\right] .\right.
\end{aligned}
$$

### 5.1. Results and discussion

To illustrate the aforementioned mathematical findings, we will provide a few simulation studies in this part. We find the system's stochastic F-F derivative in the Caputo-Fabrizio context using a
revolutionary numerical approach introduced in [41]. An evolutionary algorithm was developed to numerically predict outcomes since state formulas possess ICs. Table 2 is an overview of the primary attribute settings. The outcomes with no controls, just vaccine restrictions, just therapy regulation, and both vaccination and medication regulations are compared.

Table 2. List of parameters.

| Symbols | Values | References | Units |
| :---: | :---: | :---: | :---: |
| $\lambda$ | 6 | [18] | cell $\mathrm{ml}^{-1}$ day $^{-1}$ |
| $\vartheta$ | 0.2 | [9] | cell $\mathrm{ml}^{-1}$ day $^{-1}$ |
| $\phi_{1}$ | 0.01 | [55] | $d a y^{-1}$ |
| $\phi_{2}$ | 0.1 | [51] | day ${ }^{-1}$ |
| $\phi_{3}$ | 0.1 | [18] | day ${ }^{-1}$ |
| $\phi_{4}$ | 0.3 | [9] | day ${ }^{-1}$ |
| $\gamma_{1}$ | 0.01 | [57] | virions ${ }^{-1}$ day $^{-1}$ |
| $\gamma_{2}$ | 0.1 | [18] | cell $^{-1}$ day $^{-1}$ |
| $\gamma_{3}$ | 0.2 | [56] | cell $^{-1}$ day $^{-1}$ |
| $\gamma_{4}$ | 0.015 | [18] | $\mathrm{cell}^{-1} \mathrm{day}^{-1}$ |
| $\omega$ | 0.4 | [56] | $\mathrm{cell}^{-1} \mathrm{day}^{-1}$ |

Figures 4-5 presents the dynamics of the F-F HBV model (5.1) with attributed values of Table 2 involving TDs $\zeta_{1}=1, \zeta_{2}=2$, assuming $\sigma_{1}=0.003, \sigma_{2}=0.004, \sigma_{3}=0.006$ and $\sigma_{4}=0.004$. Straightforward computations result in $\mathbb{R}_{0}^{s}<1$, satisfying the requirements of Theorem 4.2. Figures $4-5$ demonstrate that there are fewer individuals who have been treated by untreated adults and that the proportion of acutely and chronically contaminated youngsters is declining, respectively. Therefore, we observe that controlling the preponderance of individuals who are HBV-positive may significantly lower or reduce the amount of contaminated neonates who are released during the distribution process when fractional-order $\delta$ decreasing and fractal-dimension $\varpi$ remains fixed. It is concluded that the F-F HBV model (5.1) generated in this research is perfectly accurate. It is thought that it can highlight several important characteristics that are also true in more simulation designs of HBV infection.


Figure 4. Graphical illustrations of the stochastic F-F HBV model (5.1) for healthy cells $\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)$ and infectious cells $\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)$ when there is a significant decrease in $\delta$ and $\varpi=1$..


Figure 5. Graphical illustrations of the stochastic F-F HBV model (5.1) for HBV cells $\tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)$ and CTL cells $\tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)$ when there is a significant decrease in $\delta$ and $\varpi=1$..

Figures 6-7 define the complexities of the F-F HBV model (5.1) with aforementioned parametric descriptions considering $\operatorname{TDs} \zeta_{1}=1, \zeta_{2}=2$, assuming $\sigma_{1}=0.003, \sigma_{2}=0.004, \sigma_{3}=0.006$ and $\sigma_{4}=0.004$. This also predicts that, through cytolytic and non-cytolytic processes, CTL cells are essential for the prevention and treatment of HBV infection. Infectious colonies are killed by cytolytic control by manipulating, while noncytolytic regulatory processes "treat" the intracellular pathogens [51]. According to F-F investigations, the infectious equilibrium state is robust and the pace at which pathogens produce free viral infection is low. If a medicine with a profound impact is discovered, the value of the rate of the viral bacteria's item will decrease, and other immunization collaborators should step up their efforts to reduce interaction rates to a meaningful scale when $\delta$ is
fixed and $\varpi$ declines. The HBV infection can be treated.


Figure 6. Graphical illustrations of the stochastic F-F HBV model (5.1) for healthy cells $\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)$ and infectious cells $\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)$ when there is a significant decrease in $\varpi$ and $\delta=1$..


Figure 7. Graphical illustrations of the stochastic F-F HBV model (5.1) for HBV cells $\tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)$ and CTL cells $\tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)$ when there is a significant decrease in $\varpi$ and $\delta=1$..

Figures 8-9 illustrate the complexities of the F-F HBV model, (5.1) with aforementioned parametric descriptions, considering TDs $\zeta_{1}=1, \zeta_{2}=2$, assuming $\sigma_{1}=0.1, \sigma_{2}=0.2, \sigma_{3}=0.3$ and $\sigma_{4}=0.4$. The likelihood that the sickness will disappear is, therefore, one. It has been demonstrated that a white noise setting with a higher intensity may aid in curing the illness more quickly than a simulation sans noise when $\delta$ decreases and $\varpi$ increased. Our analysis's clear and fundamental goal is to minimize the harm inflicted by HBV by reducing the population's infection rate while increasing the population's rate of recovery. The authorities have a mission to ensure that people are notified of
the proliferation of the HBV.


Figure 8. Graphical illustrations of the stochastic F-F HBV model (5.1) for healthy cells $\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)$ and infectious cells $\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)$ when $\delta$ falls significantly and $\mu$ increases.


Figure 9. Graphical illustrations of the stochastic F-F HBV model (5.1) for HBV cells $\tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)$ and CTL cells $\tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)$ when $\delta$ falls significantly and $\mu$ increases.

Figures 10-11 show the intricacies of the F-F HBV model, (5.1) utilizing the parameterized representations stated above and TDs $\zeta_{1}=1, \zeta_{2}=2$, assuming $\sigma_{1}=0.1, \sigma_{2}=0.2, \sigma_{3}=0.3$ and $\sigma_{4}=0.4$. The aforementioned configurations $10-11$ makes it evident that the basic reproduction ratio $\mathbb{R}_{0}^{\mathbf{s}}$ in framework (3.1) does not incorporate the index of CTL cells, which means that $\mathbb{R}_{0}^{s}$ is unable to accurately represent the function of immunogenicity. However, formula (5.1) includes the $\delta$ and $\varpi$, which helps illustrate the function of the CTL cells. As a result, the model (5.1) ought to be more feasible.


Figure 10. Graphical illustrations of the stochastic F-F HBV model (5.1) for healthy cells $\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)$ and infectious cells $\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)$ when $\varpi$ falls significantly and $\delta$ increases.


Figure 11. Graphical illustrations of the stochastic F-F HBV model (5.1) for HBV cells $\tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)$ and CTL cells $\tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)$ when $\varpi$ falls significantly and $\delta$ increases.

Figures 12-13 presents the solution for low densities of $\sigma_{\kappa}, \kappa=1,2,3,4$ using the Matlab package. Therefore, if a treatment with a huge influence can be discovered, the HBV infection can be treated provided that the value of $\vartheta$ is sufficiently diminished. According to the above mechanism, since many people are uneducated and unaware of viral diseases associated with them, particularly HBV, it indicates that the current regime should encourage good hygiene precautions in the general populace. Both the recycling of syringes on minors in rural areas and inadequate sanitary standards in public health sectors, which are major causes of HBV, should be prohibited. Blood donations must follow a specified protocol and must first be tested and approved by an officially recognized lab.

In a nutshell, the presence of an F-F derivative operator with stochastic disturbance (noise) and intracellular TDs in the framework (3.1) is hypothesized to deliver a better awareness in the presentation of the presented data, which has serious repercussions for treating alternatives and targeted therapies.


Figure 12. Graphical illustrations of the stochastic F-F HBV model (5.1) for healthy cells $\tilde{\mathbf{H}}\left(\mathbf{t}_{1}\right)$ and infectious cells $\tilde{\mathbf{I}}\left(\mathbf{t}_{1}\right)$ without treatment (red zigzag line) and with treatment (blue zigzag line).


Figure 13. Graphical illustrations of the stochastic F-F HBV model (5.1) for HBV cells $\tilde{\mathbf{V}}\left(\mathbf{t}_{1}\right)$ and CTL cells $\tilde{\mathbf{D}}\left(\mathbf{t}_{1}\right)$ without treatment (red zigzag line) and with treatment (blue zigzag line).

## 6. Conclusion

In the current study, we examined the effects of high-order stochastic disturbances on the complexities of the delay differential framework of HBV disease, which includes internalized latency,
immunotherapy, disease-to-organ and their transmissions via the F-F derivative within the exponential decay kernel. We also presented the non-negative solution pertaining to the unit probability, stochastic reproduction number, and local stability, which is calculated for the steady states. Under certain assumptions, the sickness may eventually go away under certain circumstances, with probability 1 . A revolutionary approach is used in a handful of simulated findings to demonstrate the viability of the outcomes. In the therapy of HBV and other viral disorders, the volume of Gaussian white noise is crucial. The presence of random disturbances (noise) and biochemical TDs in the framework is predicated to provide a greater understanding of the quantitative results, which has significant consequences for antibiotic compounds and regenerative medicine. Several additional intriguing subjects need to be looked into more thoroughly. Other types of environmental noise, including Lévy noise, may be taken into consideration [52]. Additionally, the deterministic system can be expanded to incorporate fractional derivatives in the framework to be able to take into account long-run memory of the bacteria's behavior, which is suggested by the research in [53,54].

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## Conflict of interest

The authors declare there is no conflict of interest.

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