Journal of Advances in Mathematics and Computer Science

Volume 39, Issue 11, Page 1-28, 2024; Article no.JAMCS.124589
ISSN: 2456-9968
(Past name: British Journal of Mathematics & Computer Science, Past ISSN: 2231-0851)

Mathematical Analysis of Hepatitis B Virus Transmission Dynamics in the Absence of Therapy with Atangana-Baleanu Fractional -Order SPQWXY Model

Otugene Victor Bamigwojo [a*], Mbah Christopher Godwin Ezike [b], Paul Owhenagbo Alemoh [c], Idoko Peter Idoko(OP) [b], Jeremiah Damilola Adedoyin [d], Lawrence Anebi Enyejo [e] and Agina Precious Chikaedum [f]

[a] *School of Preliminary and Remedial Studies (SPRS), Federal University Lokoja (FUL), Nigeria.*
[b] *Faculty of Physical Sciences, University of Nigeria Nsukka (UNN), Nigeria.*
[c] *Department of Bioinformatics, University of Arkansas Little Rock, United State of American.*
[d] *Department of Mathematics, University of Nevada, Reno, USA.*
[e] *Department of Telecommunications, Enforcement Ancillary and Maintenance, National Broadcasting Commission, Aso-Villa, Abuja, Nigeria.*
[f] *Department of Mathematics, Chukwuemaka Odumegwu Ojukwu University Awka, Nigeria.*

Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

Article Information

DOI: https://doi.org/10.9734/jamcs/2024/v39i111937

Open Peer Review History:
This journal follows the Advanced Open Peer Review policy. Identity of the Reviewers, Editor(s) and additional Reviewers, peer review comments, different versions of the manuscript, comments of the editors, etc are available here: https://www.sdiarticle5.com/review-history/124589

**Corresponding author: E-mail: otugenevictorbamigwojo@gmail.com;*

Cite as: *Bamigwojo, Otugene Victor, Mbah Christopher Godwin Ezike, Paul Owhenagbo Alemoh, Idoko Peter Idoko(OP), Jeremiah Damilola Adedoyin, Lawrence Anebi Enyejo, and Agina Precious Chikaedum. 2024. "Mathematical Analysis of Hepatitis B Virus Transmission Dynamics in the Absence of Therapy With Atangana-Baleanu Fractional -Order SPQWXY Model". Journal of Advances in Mathematics and Computer Science 39 (11):1-28. https://doi.org/10.9734/jamcs/2024/v39i111937.*

Received: 02/08/2024

Accepted: 05/10/2024

Original Research Article

Published: 15/10/2024

Abstract

This paper presents an innovative fractional order network model aimed at elucidating the transmission dynamics of Hepatitis B Virus (HBV). Incorporating fractional calculus enables the model to capture the intricate, memory-dependent mechanisms inherent in HBV spread, thereby overcoming the constraints of conventional integer order models. The primary objective of the study is to develop a more precise depiction of HBV transmission, encompassing both vertical and horizontal routes in the absence of vaccination strategies. Furthermore, the paper assesses the existence and uniqueness of solutions utilizing the Banach fixed point theory with the Picard-Lindelf approach. Numerical simulations conducted across various fractional orders reveal that as the fractional order decreases from 1, the rate of endemic spread decelerates.

Keywords: SPQWXY HBV-virus model; atangana-baleanue fractional derivative; picard-linderlof approach; fixed point theory.

1 Introduction

Hepatitis B Virus (HBV) remains a significant global health concern, with approx- imately 257 million people infected worldwide and over 880,000 deaths annually at- tributed to HBV-related complications [1]. Understanding the intricate dynamics of HBV transmission is paramount for devising effective prevention and control strategies. Traditional mathematical models based on integer-order calculus have been instrumental in studying infectious disease dynamics, including HBV transmission. However, these models often overlook the inherent memory-dependent and non-local properties characteristic of many biological processes. Consequently, there is a growing recognition of the limitations of integer order models in capturing the complexity of HBV spread accurately.

To address these limitations, this paper introduces an innovative mathematical frame- work based on fractional calculus to model HBV transmission dynamics. Fractional calculus offers a powerful mathematical tool for describing phenomena with memory- dependent and non-local characteristics, making it particularly well-suited for modeling biological systems [2]. The proposed model, termed the Atangana-Baleanu Fractional- Order SPQWXY Model, integrates fractional calculus to capture the nuanced dynamics of HBV transmission. The primary objective of this study is to develop a comprehensive understanding of HBV transmission dynamics in the absence of therapy using the proposed fractional- order model. Specifically, we aim to investigate the impact of fractional-order dynamics on the spread of HBV, considering both vertical and horizontal transmission routes. Furthermore, we examine the existence and uniqueness of solutions for the model using rigorous mathematical analysis based on the Banach fixed point theory with the Picard- Lindelf approach [3]. The history of epidemiological mathematical modeling can be traced back to 1766 when Daniel Bernoulli published his seminal work on the effect of smallpox variolation on life expectancy [4]. This marked the inception of using mathematical language to understand the transmission dynamics of epidemic diseases. Building upon Bernoulli's foundational work, Kermack and McKendrick introduced a series of papers in 1927 that described disease transmission dynamics through systems of differential equations [5]. Traditionally, epidemiological mathematical models have relied on integer-order differential equations to characterize disease spread and assess control strategies [6]. However, recent advancements in mathematical analysis have revealed the potential of fractional calculus to model complex phenomena, including epidemiological dynamics [7]. Fractional calculus offers a powerful framework for describing systems with memory- dependent and non-local properties, making it

particularly well-suited for modeling bi- ological processes such as disease transmission [8]. Unlike integer-order derivatives, fractional derivatives incorporate past and present information, capturing the hereditary properties and memory efficacy essential for understanding biological mechanisms [9]. Key figures in the development of fractional calculus include Caputo, who intro- duced a fractional derivative with non-singular kernel in 1967 [10], and Atangana and Baleanu, who proposed new fractional derivatives and applied them to various models, including heat transfer [11]. Baleanu et al. investigated a fractional mathematical model for tumor-immune surveillance mechanisms and studied the effect of chemotherapy on the model [12]. 2 Recent studies by Kolade and Owolabi focused on the analysis and numerical sim- ulation of a fractional SEIR (Susceptible-Exposed-Infectious-Recovered) model with time delay, demonstrating the applicability of fractional calculus in epidemiological modeling [13]. Additionally, researchers have utilized fractional derivatives, such as the Atangana-Baleanu operator involving the Mittag-Leffler kernel, to analyze SEIRA (Susceptible-Exposed-Infectious-Recovered-Aware) mathematical models [14]. Fractional calculus, the generalization of traditional calculus to include non-integer order derivatives and integrals, has gained significant attention in recent years, particularly in the modeling of biological systems and disease dynamics. Its ability to describe memory effects, long-range interactions, and anomalous diffusion makes it a powerful tool for capturing the complexity of biological systems that traditional integer-order models struggle to handle. Here is a comprehensive review of fractional calculus in biological disease modeling.The origin of fractional calculus can be traced back to the 17th century when mathematicians like Leibniz and Euler discussed the possibility of extending the notion of derivatives and integrals to non-integer orders. However, its application to real-world problems, especially in biological sciences, only emerged in the late 20th and early 21st centuries. The motivation for applying fractional calculus in biological systems arises from the need to model processes that exhibit memory and hereditary properties—characteristics that are often present in biological systems and disease transmission dynamics. Fractional derivatives provide a way to incorporate the memory of past states into the current dynamics, which is crucial for accurately modeling diseases where the history of infection or immunity plays a significant role (e.g., immune response in viral infections). Biological systems, such as the spread of disease in heterogeneous populations, often exhibit diffusion that is not well-described by classical models. Fractional models can capture sub-diffusive or super-diffusive processes, which are common in epidemiology. Fractional models often fit experimental and epidemiological data better than their integer-order counterparts, particularly for complex diseases with delayed responses or long incubation periods. In classical epidemiological models such as SIR (Susceptible-Infectious-Recovered) or SEIR (Susceptible-Exposed-Infectious-Recovered), the dynamics of disease transmission are usually represented by integer-order differential equations. By extending these models using fractional derivatives, researchers have been able to:

- diseases with long latency periods or memory effects, where the present state depends on the entire history of the system (e.g., immune responses that depend on past exposure).
- Better represent diseases that spread in a heterogeneous population, where individuals may have different susceptibilities, contact rates, or recovery patterns.
- Address non-locality in space, where disease transmission is not limited to nearby individuals but can occur over long distances (e.g., air travel spreading infectious diseases globally).

Fractional calculus generalizes the concept of derivatives and integrals to non-integer orders, providing powerful tools to model systems exhibiting memory and hereditary properties. It extends beyond classical calculus to address complex phenomena found in various scientific and engineering disciplines. The primary fractional derivatives include:

- **Riemann-Liouville Derivative:** Defined using an integral representation with a singular kernel, often leading to singularity issues.
- **Caputo Derivative:** A modification of the Riemann-Liouville derivative that allows for better initial condition handling, particularly useful in boundary value problems.

Brauer and Castillo-Chavez's work on mathematical models in population biology and epidemiology provides foundational tools for modeling infectious diseases, including compartmental models that have been extended to

include fractional-order dynamics in recent studies [15]. Hu discusses occult Hepatitis B virus (HBV) infections, which are significant in understanding the persistence of HBV in patients without detectable serological markers [28]. Dietz and Heesterbeek revisited Bernoulli's model, which serves as a historical basis for modern infectious disease models, including fractional-order models used for HBV [29]. Abdelouahab and Hamri explore the Grünwald-Letnikov fractional-order derivative, which offers a discrete approximation critical for applying fractional calculus to epidemiological models [35]. Puri et al. examine how social media contributes to vaccine hesitancy, a relevant factor in the public's acceptance of HBV vaccination programs [36]. Wismans et al. provide insights into the immune response following Hepatitis B vaccination in diabetic patients, emphasizing the vaccine's efficacy in populations with compromised immune systems [37]. Roeder et al. discuss strategies for disease eradication, drawing parallels between rinderpest eradication and ongoing efforts for HBV control through vaccination [38]. Ayerbe et al. assess the long-term efficacy of Hepatitis B vaccines, essential for understanding long-term protection against HBV in vaccinated populations [39]. Yu et al. investigate anomalous diffusion in MRI, introducing mathematical tools that could inform more complex epidemiological models, including those for HBV transmission [40]. Rida et al. provide an approximate solution to a fractional-order model for Hepatitis C virus (HCV) infection, analogous to fractional-order models for HBV using similar mathematical approaches [41]. Otugene et al. analyze the existence and uniqueness of solutions in fractional-order network models, which supports the use of the Atangana-Baleanu derivative in understanding viral transmission dynamics [50]. Buckwold et al. investigate HBV genome mutations that impact viral replication and persistence, critical for understanding different stages of HBV infection [54]. Carman et al. discuss mutations in the HBV virus that prevent the formation of the e antigen, a marker of viral replication, which impacts infection modeling [55]. Swaddiwudhipong et al. highlight the importance of public health interventions in controlling infectious diseases, with parallels to HBV control measures [57]. Kermack and McKendrick's classic model forms the basis for modern epidemic models, including those adapted for fractional calculus in HBV studies [58]. Chen et al. review the application of fractional epidemic models, emphasizing the relevance of fractional calculus in epidemiological modeling [59]. Mukandavire et al. explore the effects of public health campaigns on HIV transmission, analogous to similar interventions in HBV control [60]. Wang et al. review disease-behavior dynamics on complex networks, which is crucial for understanding how individual behaviors, like vaccination, interact with HBV transmission [61].

The literature reviewed above underscores the broad spectrum of applications for fractional derivatives in mathematical modeling and the analysis of real-world phenomena. Particularly noteworthy is the recent emergence of the Atangana-Baleanu (A-B) fractional derivative, which has garnered widespread recognition and appreciation for its extensive utilization across various disciplines, including biology, physics, medical engineering, and nonlinear analysis. Motivated by the aforementioned considerations, this paper delves into the study of the SPQWXY model, which encompasses susceptible-exposed-subclinical infected- acute infected-chronic and fulminate cases of Hepatitis B Virus (HBV). The authors have structured the remainder of this paper as follows: In Section 2, we detail the formulation of the HBV virus model with fractional order, elucidating the mathematical framework underlying our analysis. Section 3 is dedicated to establishing the existence and uniqueness of solutions, employing the fixed-point theory and the Picard-Lindelf approach to rigorously validate our model. In Section 4, we demonstrate the positivity and boundedness of solutions in terms of the Atangana-Baleanu operator, providing further insights into the stability of our model. Finally, through numerical simulations conducted across various fractional orders, as discussed in Section 5, we reveal a notable trend: as the fractional order decreases from 1, the spread of the endemic proceeds at a slower pace. This finding highlights the critical role of fractional calculus in capturing the nuanced dynamics of HBV transmission.

2 Atangana-Baleanu Derivative

The Atangana-Baleanu (AB) derivative, introduced by Atangana and Baleanu in 2015, represents a novel approach to fractional calculus. It utilizes a non-singular kernel to address some of the limitations associated with traditional fractional derivatives.

Definition

The Atangana-Baleanu fractional derivative of order α for a function $f(t)$ is given by:

$$D_{AB}^{\alpha} f(t) = \frac{1}{\Gamma(m-\alpha)} \int_a^t (t-\tau)^{m-\alpha-1} e^{-\beta(t-\tau)} f(\tau)\, d\tau,$$

where:

- α is the order of differentiation.
- $m = \lceil \alpha \rceil$ is the smallest integer greater than or equal to α.
- Γ is the Gamma function, which generalizes the factorial function.
- β is a parameter that modulates the influence of the kernel's exponential term.
- a is the lower limit of integration, often taken as 0 or the initial condition.

Key Properties

- **Non-Singular Kernel:** Unlike the Riemann-Liouville derivative, which involves a singular kernel at $t = \tau$, the AB derivative uses a non-singular kernel $(t-\tau)^{m-\alpha-1} e^{-\beta(t-\tau)}$. This approach helps to avoid singularity issues and improves the stability of the derivative.
- **Memory Effects:** The AB derivative incorporates memory effects through the $e^{-\beta(t-\tau)}$ term, which accounts for the influence of past states on the current state. This feature is crucial for modeling processes with long-term dependencies.
- **Adjustable Parameter β:** The parameter β controls the impact of the kernel's exponential term, allowing for greater flexibility in modeling different types of memory effects and hereditary properties.
- **Fractional Differentiation Order:** The order of differentiation α can be a non-integer, providing a broader range of modeling capabilities compared to integer-order derivatives.

Mathematical Properties

- **Linearity:** The AB derivative is linear, meaning $D_{AB}^{\alpha}(af(t) + bg(t)) = aD_{AB}^{\alpha} f(t) + bD_{AB}^{\alpha} g(t)$, where a and b are constants.
- **Composition Rule:** For two functions $f(t)$ and $g(t)$, the derivative $D_{AB}^{\alpha}(f \cdot g)$ can be expressed using Leibniz's rule, extending to fractional derivatives.
- **Initial Conditions:** Initial conditions for AB derivatives are handled differently compared to integer-order derivatives, often requiring careful formulation to incorporate fractional orders.

Advantages over Other Fractional Derivatives

- **Avoidance of Singularities:** The non-singular kernel of the AB derivative eliminates the issues associated with singularities at $t = \tau$, leading to more stable numerical solutions and better representation of real-world processes.
- **Enhanced Flexibility:** The parameter β allows for customization of the memory effect, making it possible to model a wider range of phenomena with varying degrees of past influence.
- **Improved Stability:** Numerical simulations often show that the AB derivative provides improved stability and accuracy in solving fractional differential equations compared to traditional derivatives, especially for complex and nonlinear systems.

Applications

The Atangana-Baleanu derivative has been successfully applied in various fields, including:

- **Biological Systems:** Modeling of population dynamics, disease transmission (e.g., Hepatitis B Virus), and other biological processes where memory effects and long-range interactions are significant.
- **Engineering:** Control systems, signal processing, and systems with non-local interactions benefit from the AB derivative's flexibility in modeling complex dynamics.
- **Physics:** Used in models involving anomalous diffusion, viscoelastic materials, and other physical phenomena with memory effects.

The Atangana-Baleanu fractional derivative represents a significant advancement in fractional calculus by addressing limitations of traditional approaches with its non-singular kernel and adjustable parameters. It offers enhanced flexibility and stability, making it suitable for modeling complex systems with memory effects. However, challenges such as computational complexity, parameter sensitivity, and limited standardization need to be managed through careful application and further research.

3 Model Assumptions

Given a population size $N(t)$, we consider six population compartments corresponding to different stages of HBV (Hepatitis B Virus) infection:

- Susceptible ($S(t)$): Individuals at risk of HBV infection.
- Subclinical ($P(t)$): Infected but not yet infectious.
- Clinical ($Q(t)$): Infected and infectious.
- Acute ($W(t)$): Infectious with symptomatic HBV.
- Chronic ($Y(t)$): Long-term infection lasting months.
- Fulminant ($X(t)$): Severe, acute infection stage.

When HBV infects a cell and progresses to the acute stage $W(t)$, the viral DNA is converted into a single covalently closed circular DNA (cccDNA) molecule, followed by the accumulation of additional copies (up to 50) due to synthesis pathways or multiple infectious events. This amplification leads to the development of both chronic ($Y(t)$) and fulminant ($X(t)$) infection stages from the acute stage.

The disease spread occurs in a non-closed environment with emigration and immigration, altering the total population size. Therefore, the total population at any time is the sum of the subclinical stage of infection and the rate of new target cell production, which is not constant (i.e., $S(t) + P(t) + Q(t) + W(t) + Y(t) + X(t) \neq$ constant).

The rate of susceptible individuals becoming infected (force of infection ζ_1) is given by:

$$\zeta_1 = Q + W + X + Y$$

We assume that recovered individuals do not acquire permanent immunity, and demographic factors (age, sex, social status, race) do not affect the infection risk. The population mixes homogeneously, implying uniform interaction among individuals.

According to experimental evidence, individuals can transition from the subclinical compartment $P(t)$ to the susceptible class $S(t)$ naturally, especially with high immunity levels at a given time.

Key parameters influencing the model include:

- λ_i: Transmission rates to various compartments ($\lambda_1 \neq ... \neq \lambda_{11}$).
- δ_i: Disease-induced death rates ($\delta_1 \neq ... \neq \delta_8$).
- μ: Natural death rate.

- β: Rate of production of new target cells.

Individuals exit the infected compartments only through death (due to the disease δ_i) or natural causes (μ). The per capita birth and death rates in the absence of disease are β_i and μ, respectively, suggesting exponential population growth in the absence of disease.

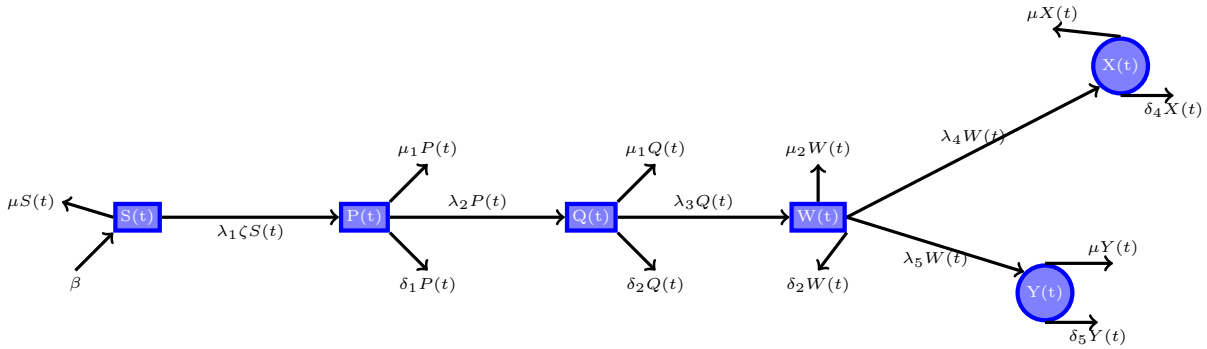

Fig. 1. Schematic diagram of HBV in the Absence of Therapy

The mathematical model with integer order used in this study is expressed by the equation:

$$\left.\begin{aligned} D_t^\alpha S(t) &= \beta - \mu S(t) - \alpha_1 \zeta S(t) \\ D_t^\alpha P(t) &= \alpha_1 \zeta S(t) - (\mu + \delta_1 + \lambda_2) P(t) \\ D_t^\alpha Q(t) &= \lambda_2 P(t) - (\mu + \delta_2 + \lambda_3) Q(t) \\ D_t^\alpha W(t) &= \lambda_3 Q(t) - (\mu + \delta_3 + \lambda_4 + \lambda_5) W(t) \\ D_t^\alpha X(t) &= \lambda_4 W(t) - (\mu + \delta_4) X(t) \\ D_t^\alpha Y(t) &= \lambda_5 W(t) - (\mu + \delta_5) Y(t) \end{aligned}\right\} \tag{3.1}$$

The natural death rate term is μ. In the absence of disease, the differential equation for the total population is given by: $\frac{dN}{dt} = \beta - \alpha_3 N$.The limit of the population size $N(t)$ as $t \to \infty$ is: $\lim_{t\to\infty} N(t) = \frac{\beta}{\mu}$ which represents the carrying capacity of the demographic structure under consideration. Therefore, the AB fractional order mathematical model, considering the assumptions and a saturating contact rate, is described by the following system of differential equations:

$$\left.\begin{aligned} {}_0^{ABC}D_t^\alpha S(t) &= G_1(t, S) \\ {}_0^{ABC}D_t^\alpha E(t) &= G_2(t, P) \\ {}_0^{ABC}D_t^\alpha I(t) &= G_3(t, Q) \\ {}_0^{ABC}D_t^\alpha I_T(t) &= G_4(t, W) \\ {}_0^{ABC}D_t^\alpha Q(t) &= G_5(t, X) \\ {}_0^{ABC}D_t^\alpha R(t) &= G_6(t, Y) \end{aligned}\right\} \tag{3.2}$$

where the kernels are given by:

$$\left.\begin{aligned} G_1(t,S) &= \beta - \mu S(t) - \alpha_1 \zeta S(t) \\ G_2(t,E) &= \lambda_2 P(t) - (\mu + \delta_2 + \lambda_3) Q(t) \\ G_3(t,I) &= \lambda_2 I(t) - (\mu + \alpha_3 + \lambda_1) Q(t) \\ G_4(t,I_T) &= \lambda_3 Q(t) - (\mu + \delta_3 + \lambda_4 + \lambda_5) W(t) \\ G_5(t,Q) &= \lambda_4 W(t) - (\mu + \delta_4) X(t) \\ G_6(t,R) &= \lambda_5 W(t) - (\mu + \delta_5) Y(t) \end{aligned}\right\} \tag{3.3}$$

The initial conditions for the compartments are given by: $S(0) = S_0, \quad P(0) = P_0, \quad Q(0) = Q_0, \quad W(0) = W_0, \quad X(0) = X_0, \quad Y(0) = Y_0$.In the presence of an endemic, the differential equation for the total population is:$\frac{dN}{dt} = \beta - \mu N - \alpha_3$. This differential equation indicates that the population size $N(t)$ is not constant over time.

4 Existence and Uniqueness of Solutions

Let's explore the presence and singular nature of the solution to the fractional order model (4). To illustrate this, we utilize the widely acknowledged Banach fixed point theorem. For a comprehensive examination of fixed points and contractions, we suggest referring to ([3] and its associated literature). Now, to affirm the presence and exclusivity of the solution, we take the following steps: Employing the AB fractional integral as delineated in [2] on model (4), we derive:

$$\left.\begin{aligned} S(t) - S(0) &= \frac{1-\alpha}{F(\alpha)} G_1(t,S) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_1(k,S)(t-k)^{\alpha-1} dk, \\ P(t) - P(0) &= \frac{1-\alpha}{F(\alpha)} G_2(t,P) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_2(k,P)(t-k)^{\alpha-1} dk, \\ Q(t) - Q(0) &= \frac{1-\alpha}{F(\alpha)} G_3(t,Q) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_3(k,Q)(t-k)^{\alpha-1} dk, \\ W(t) - W(0) &= \frac{1-\alpha}{F(\alpha)} G_4(t,W) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_4(k,W)(t-k)^{\alpha-1} dk, \\ X(t) - X(0) &= \frac{1-\alpha}{F(\alpha)} G_5(t,X) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_5(k,X)(t-k)^{\alpha-1} dk, \\ Y(t) - Y(0) &= \frac{1-\alpha}{F(\alpha)} G_6(t,Y) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t G_6(k,Y)(t-k)^{\alpha-1} dk \end{aligned}\right\} \tag{4.1}$$

where $\alpha \in (0,1)$, $F(\alpha)$ is a function of α, $\Gamma(\alpha)$ denotes the Gamma function, and $G_i(t,\cdot)$ are given functions.

The set $B = H(J) \times H(J) \times \cdots$ is a Banach space defined as the Cartesian product of spaces, where $H(J) = C[0,T]$ is the space of continuous real-valued functions defined on the interval $J = [0,T]$. The norm $||(\cdot)||$ on B is defined as:

$$||(S,E,I,I_T,Q,R)|| = ||S|| + ||E|| + ||I|| + ||I_T|| + ||Q|| + ||R||$$

Here, the norms $||S||$, $||P||$, $||Q||$, $||W||$, $||X||$, and $||Y||$ are defined as:

$$||S|| = \sup_{t\in J}|S(t)|, \quad ||P|| = \sup_{t\in J}|P(t)|, \quad ||Q|| = \sup_{t\in J}|Q|,$$
$$||W|| = \sup_{t\in J}|W(t)|, \quad ||X|| = \sup_{t\in J}|X(t)|, \quad ||Y|| = \sup_{t\in J}|Y(t)|$$

These norms measure the maximum absolute values of the respective functions over the interval $J = [0, T]$.

Theorem 1 (Lipschitz Condition and Contraction)
For each of the kernels, $G_1(t, S), G_2(t, P), \ldots, G_6(t, Y)$ in (2), there exist constants $L_i > 0$ for $i = 1, 2, 3, 4, 5, 6$ such that:

$$\begin{aligned}
\|G_1(t, S) - G_1(t, S_1)\| &\leq L_1\|S(t) - S_1(t)\|,\\
\|G_2(t, P) - G_2(t, P_1)\| &\leq L_2\|P(t) - P_1(t)\|,\\
\|G_3(t, Q) - G_3(t, Q_1)\| &\leq L_3\|Q(t) - Q_1(t)\|,\\
\|G_4(t, W) - G_4(t, W_1)\| &\leq L_4\|W(t) - W_1(t)\|,\\
\|G_5(t, X) - G_5(t, X_1)\| &\leq L_5\|X(t) - X_1(t)\|,\\
\|G_6(t, Y) - G_6(t, Y_1)\| &\leq L_6\|Y(t) - Y_1(t)\|
\end{aligned}$$

where $0 \leq L_i \leq 1$ for all $i = 1, 2, 3, 4, 5, 6$.

Proof:

$$\begin{aligned}
||G_1(t, S) - G_1(t, S_1)|| &= ||\beta - \mu S(t) - \alpha_1\zeta S(t) - (\beta - \mu S_1(t) - \alpha_1\zeta S_1(t))||\\
&\leq \Big(\alpha_1(m_2 + m_1)\Big)||(S_1(t) - S(t))||\\
&\leq L_1||(S_1(t) - S(t))||
\end{aligned}$$

Where $L_1 = \Big(\alpha_1(m_3 + m_5)\Big) + \mu$ $||S||$ is $\sup_{t\in J} = M_1$,$||P||$ is $\sup_{t\in J} = M_2$,$||Q||$ is $\sup_{t\in J} = M_3$,$||W||$ is $\sup_{t\in J} = M_4$,$||X||$ is $\sup_{t\in J} = M_5$,$||Y||$ is $\sup_{t\in J} = M_6$.

In a similar manner, one can demonstrate the existence of $L_i = 2, 3, 4, 5, 6$ and a contraction principle for $G_2(t, P), G_3(t, Q), G_4(t, W), G_5(t, X), G_6(t, Y)$, where $0 \leq L_i < 1$. The recursive form of (6) is now defined for $t = t_n$, where $n = 1, 2, 3, \ldots$.

$$\left.\begin{aligned}
S_n(t) &= \frac{1-\alpha}{F(\alpha)}G_1(t, S_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t G_1(k, S_{n-1})(t-k)^{\alpha-1}dk,\\
P_n(t) &= \frac{1-\alpha}{F(\alpha)}G_2(t, P_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t G_2(k, P_{n-1})(t-k)^{\alpha-1}dk,\\
Qn(t) &= \frac{1-\alpha}{F(\alpha)}G_3(t, Q_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t G_3(k, Q_{n-1})(t-k)^{\alpha-1}dk,\\
W_n(t) &= \frac{1-\alpha}{F(\alpha)}G_4(t, W_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t G_4(k, W_{n-1})(t-k)^{\alpha-1}dk,\\
X_n(t) &= \frac{1-\alpha}{F(\alpha)}G_5(t, X_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t G_5(k, X_{n-1})(t-k)^{\alpha-1}dk,\\
Y_n(t) &= \frac{1-\alpha}{F(\alpha)}G_6(t, Y_{n-1}) + \frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t G_6(k, Y_{n-1})(t-k)^{\alpha-1}dk,
\end{aligned}\right\} \tag{4.2}$$

The difference between successive terms in (7) are expressed as follows:

$$\left.\begin{aligned}
A_{1n}(t) = S_n - S_{n-1} = \frac{1-\alpha}{F(\alpha)}(G_1(t,S_{n-1}) - G_1(t,S_{n-2}))+ \\
\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t (G_1(t,S_{n-1}) - G_1(t,S_{n-1}))(t-k)^{\alpha-1}dk, \\
A_{2n}(t) = P_n - P_{n-1} = \frac{1-\alpha}{F(\alpha)}(G_2(t,E_{n-1}) - G_2(t,E_{n-2}))+ \\
\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t (G_2(t,P_{n-1}) - G_2(t,P_{n-1}))(t-k)^{\alpha-1}dk, \\
A_{3n}(t) = Q_n - Q_{n-1} = \frac{1-\alpha}{F(\alpha)}(G_3(t,I_{n-1}) - G_3(t,I_{n-2}))+ \\
\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t (G_3(t,Q_{n-1}) - G_3(t,Q_{n-1}))(t-k)^{\alpha-1}dk, \\
A_{4n}(t) = W_n - W_{n-1} = \frac{1-\alpha}{F(\alpha)}(G_4(t,W_{n-1}) - G_4(t,W_{n-2}))+ \\
\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t (G_4(t,W_{n-1}) - G_4(t,W_{n-1}))(t-k)^{\alpha-1}dk, \\
A_{5n}(t) = X_n - X_{n-1} = \frac{1-\alpha}{F(\alpha)}(G_5(t,X_{n-1}) - G_5(t,X_{n-2}))+ \\
\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t (G_5(t,X_{n-1}) - G_5(t,X_{n-1}))(t-k)^{\alpha-1}dk,
\end{aligned}\right\} \tag{4.3}$$

$$\left.\begin{aligned}
A_{6n}(t) = Y_n - Y_{n-1} = \frac{1-\alpha}{F(\alpha)}(G_6(t,Y_{n-1}) - G_6(t,Y_{n-2}))+ \\
\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t (G_6(t,Y_{n-1}) - G_6(t,Y_{n-1}))(t-k)^{\alpha-1}dk,
\end{aligned}\right\}$$

Norm: A norm in mathematics is a function that assigns a positive length or size to each vector in a vector space. Let V be a vector space over the field of real or complex numbers. A norm on V is a function $\|\cdot\| : V \to \mathbb{R}$ satisfying the following properties for all vectors $\mathbf{u}, \mathbf{v}$ in V and scalars α:

1. **Non-negativity:** $\|\mathbf{u}\| \geq 0$ and $\|\mathbf{u}\| = 0$ if and only if $\mathbf{u} = \mathbf{0}$, where $\mathbf{0}$ denotes the zero vector.

2. **Homogeneity:** $\|\alpha\mathbf{u}\| = |\alpha|\|\mathbf{u}\|$ for all scalars α.

3. **Triangle inequality:** $\|\mathbf{u} + \mathbf{v}\| \leq \|\mathbf{u}\| + \|\mathbf{v}\|$.

Taking the norm of both sides of (7), we have:

$$\left.\begin{aligned}
||A_{1n}(t)|| =&||S_n(t) - S_{n-1}(t)||\frac{1-\alpha}{F(\alpha)}||(G_1(t,S_{n-1}) - G_1(t,S_{n-2}))||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t ||(G_1(t,S_{n-1}) - G_1(t,S_{n-1}))||(t-k)^{\alpha 1}dk,\\
||A_{2n}(t)|| =&||P_n - P_{n-1}||= \frac{1-\alpha}{F(\alpha)}||(G_2(t,P_{n-1}) - G_2(t,P_{n-2}))||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t ||(G_2(t,P_{n-1}) - G_2(t,P_{n-1}))||(t-k)^{\alpha-1}dk,\\
||A_{3n}(t)|| =&||Q_n - Q_{n-1}||= \frac{1-\alpha}{F(\alpha)}||(G_3(t,Q_{n-1}) - G_3(t,Q_{n-2}))||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t ||(G_3(t,Q_{n-1}) - G_3(t,Q_{n-1}))||(t-k)^{\alpha-1}dk,\\
||A_{4n}(t)|| =&||W_n - W_{n-1}||= \frac{1-\alpha}{F(\alpha)}||(G_4(t,W_{n-1}) - G_4(t,W_{n-2}))||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t ||(G_4(t,W_{n-1}) - G_4(t,W_{n-1}))||(t-k)^{\alpha-1}dk,\\
||A_{5n}(t)|| =&||X_n - X_{n-1}||= \frac{1-\alpha}{F(\alpha)}||(G_5(t,X_{n-1}) - G_5(t,X_{n-2}))||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t ||(G_5(t,X_{n-1}) - G_5(t,X_{n-1}))||(t-k)^{\alpha-1}dk,
\end{aligned}\right\} \tag{4.4}$$

$$\left.\begin{aligned}
||A_{6n}(t)|| =&||Y_n - Y_{n-1}||= \frac{1-\alpha}{F(\alpha)}||(G_6(t,Y_{n-1}) - G_6(t,Y_{n-2}))||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t ||(G_6(t,Y_{n-1}) - G_6(t,Y_{n-1}))||(t-k)^{\alpha-1}dk,
\end{aligned}\right\}$$

The first equation in (9) is reduced to the following expression.

$$\begin{aligned}
||A_{1n}(t)|| =&||S_n(t) - S_{n-1}(t)||\leq \frac{1-\alpha}{F(\alpha)}||(G_1(t,S_{n-1}) - G_1(t,S_{n-2}))||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t ||(G_1(t,S_{n-1}) - G_1(t,S_{n-1}))||(t-k)^{\alpha-1}dk,\\
||A_{1n}(t)|| \leq& \frac{1-\alpha}{F(\alpha)}L_1||S_{n-1}(t) - S_{n-2}(t)||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}L_1\int_0^\tau ||S_{n-1}(t) - S_{n-2}(t)||(t-k)^{\alpha-1}dk,\\
||A_{1n}(t)|| \leq& \frac{1-\alpha}{F(\alpha)}L_1||S_{n-1}(t) - S_{n-2}(t)||+\\
&\frac{\alpha}{F(\alpha)\Gamma(\alpha)}L_1||S_{n-1}(t) - S_{n-2}(t)||\textstyle\int_0^t (t-k)^{\alpha-1}dk,\\
||A_{1n}(t)|| \leq& L_1||S_{n-1}(t) - S_{n-2}(t)||\left|\frac{1-\alpha}{F(\alpha)} + \frac{\alpha}{F(\alpha)\Gamma(\alpha)}\textstyle\int_0^t (t-k)^{\alpha-1}dk\right|\\
&||A_{1n}(t)|| \leq L_1||A_{1(n-1)}||\left|\frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}\right|
\end{aligned}$$

Therefore,

$$||A_{1n}(t)|| \leq L_1\left|\frac{1-\alpha}{F(\alpha)} + \frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}\right|||A_{1(n-1)}|| \tag{4.5}$$

Likewise, we streamlined the residual expression of (9) into the following configuration:

$$\left.\begin{aligned}
||A_{2n}(t)|| &\leq L_2\left|\frac{1-\alpha}{F(\alpha)}+\frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}\right|||A_{2(n-1)}(t)|| \\
||A_{3n}(t)|| &\leq L_3\left|\frac{1-\alpha}{F(\alpha)}+\frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}\right|||A_{3(n-1)}(t)|| \\
||A_{4n}(t)|| &\leq L_4\left|\frac{1-\alpha}{F(\alpha)}+\frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}\right|||A_{4(n-1)}(t)|| \\
||A_{5n}(t)|| &\leq L_5\left|\frac{1-\alpha}{F(\alpha)}+\frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}\right|||A_{5(n-1)}(t)|| \\
||A_{6n}(t)|| &\leq L_6\left|\frac{1-\alpha}{F(\alpha)}+\frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}\right|||A_{6(n-1)}(t)||
\end{aligned}\right\} \tag{4.6}$$

Theorem 2: The fractional model given in (4) has a solution if we can find M_0 satisfying

$$\left(\frac{1-\alpha}{F(\alpha)}+\frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}\right)L_i \leq 1 i = 1,2,...,6. \tag{4.7}$$

From (10) and (11) we have:

$$\left.\begin{aligned}
||A_{1n}(t)|| &\leq ||S(0)||\left|\left(\frac{1-\alpha}{F(\alpha)}+\frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)}\right)L_1\right|^n, \\
||A_{2n}(t)|| &\leq ||P(0)||\left|\left(\frac{1-\alpha}{F(\alpha)}+\frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)}\right)L_2\right|^n, \\
||A_{3n}(t)|| &\leq ||Q(0)||\left|\left(\frac{1-\alpha}{F(\alpha)}+\frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)}\right)L_3\right|^n, \\
||A_{4n}(t)|| &\leq ||W(0)||\left|\left(\frac{1-\alpha}{F(\alpha)}+\frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)}\right)L_4\right|^n, \\
||A_{5n}(t)|| &\leq ||X(0)||\left|\left(\frac{1-\alpha}{F(\alpha)}+\frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)}\right)L_5\right|^n, \\
||A_{6n}(t)|| &\leq ||Y(0)||\left|\left(\frac{1-\alpha}{F(\alpha)}+\frac{M_0^\alpha}{F(\alpha)\Gamma(\alpha)}\right)L_6\right|^n
\end{aligned}\right. \tag{4.8}$$

The existence of the solution is confirmed by Theorem 1. Now, we need to demonstrate that the functions $S(t)$, $P(t)$, $Q(t)$, $W(t)$, $X(t)$, and $Y(t)$ are solutions of the model (4). To establish this, we assume that the following conditions are satisfied:

$$\left.\begin{aligned}
S(t)-S(0) &= S_n(t)-a_{1n}(t) \\
P(t)-P(0) &= P_n(t)-a_{2n}(t) \\
Q(t)-Q(0) &= Q_n(t)-a_{3n}(t) \\
W(0)-W(0) &= W_n(t)-a_{4n}(t) \\
X(t)-X(0) &= X_n(t)-a_{5n}(t) \\
Y(t)-Y(0) &= Y_n(t)-a_{6n}(t)
\end{aligned}\right\} \tag{4.9}$$

From (13),

$$||a_{1n}(t)|| = \frac{1-\alpha}{F(\alpha)}||(G_1(t,S_{n-1})-G_1(t,S_{n-2}))|| +$$

$$\frac{\alpha}{F(\alpha)\Gamma(\alpha)}\int_0^t ||(G_1(k,S_{n-1})-G_1(k,S_{n-1}))||(t-k)^{\alpha-1}dk$$

$$||a_{1n}(t)|| \leq \frac{1-\alpha}{F(\alpha)}L_1||S_n(t)-S_{n-1}(t)||\frac{t^\alpha}{F(\alpha)\Gamma(\alpha)}L_1||S_n(t)-S_{n-1}(t)||.$$

Continuing with recursive iterations, the connections can be articulated as follows:

$$||a_{1n}(t)|| \leq \left[\frac{1-\alpha}{F(\alpha)} + \frac{t^{\alpha}}{F(\alpha)\Gamma(\alpha)}\right]^{n+1} L_1^n ||S_n(t) - S_{n-1}(t)||^n$$

Which at $t = M_0^{\alpha}$ yields,

$$||a_{1n}(t)|| \leq \left[\frac{1-\alpha}{F(\alpha)} + \frac{M_0^{\alpha}}{F(\alpha)\Gamma(\alpha)}\right]^{n+1} L_1^n ||S_n(t) - S_{n-1}(t)||^n \tag{4.10}$$

Applying the limit to both sides of equation (15) as $n \to \infty$, we observe that:
The condition $\|a_{1n}(t)\| \to 0$ as $n \to \infty$ for values of t satisfying

$$\left[\frac{1-\alpha}{F(\alpha)} + \frac{t^{\alpha}}{F(\alpha)\Gamma(\alpha)}\right] L_1 \leq 1.$$

In a similar fashion, we demonstrate that

$$\|a_{2n}(t)\| \to 0, \quad \|a_{3n}(t)\| \to 0, \quad \|a_{4n}(t)\| \to 0, \quad \|a_{5n}(t)\| \to 0, \quad \|a_{6n}(t)\| \to 0$$

for values of t satisfying

$$\left[\frac{1-\alpha}{F(\alpha)} + \frac{t^{\alpha}}{F(\alpha)\Gamma(\alpha)}\right] L_i \leq 1 \quad \text{for } i = 2, 3, 4, 5, 6.$$

Theorems 1 and 2 provide assurance regarding the presence of a solution to model (3) through the Banach fixed point theorem. In Theorem 3, we shall substantiate the distinctiveness of this solution.

Theorem 3: Uniqueness of Solution
The fractional model (3) has a unique solution provided that

$$\left[\frac{1-\alpha}{F(\alpha)} + \frac{t^{\alpha}}{F(\alpha)\Gamma(\alpha)}\right] L_i \leq 1 \quad \text{for } i = 2, 3, 4, 5, 6. \tag{4.11}$$

Proof:
Assuming that $S_1(t)$, $P_1(t)$, $Q_1(t)$, $W_1(t)$, $X_1(t)$, and $Y_1(t)$ are solutions to model (3), then,

$$\begin{aligned} S(t) - S_1(t) = {} & \frac{1-\alpha}{F(\alpha)}(G_1 S(t) - G_1 S_1(t)) \\ & + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} \int_0^t (G_1 S(t) - G_1 S_1(t))(t-k)^{\alpha-1} dk, \end{aligned}$$

Taking the norm of both sides, we have:

$$\begin{aligned} ||S(t) - S_1(t)|| = {} & \frac{1-\alpha}{F(\alpha)} ||L_1||\, ||S(t) - S_1(t)|| \\ & + \frac{t^{\alpha}}{F(\alpha)\Gamma(\alpha)} ||L_1||\, ||S(t) - S_1(t)||. \end{aligned}$$

Since

$$\left(1 - ||L_1|| \left(\frac{1-\alpha}{F(\alpha)} + \frac{t^{\alpha}}{F(\alpha)\Gamma(\alpha)}\right)\right) > 0,$$

we obtain $||S(t) - S_1(t)|| = 0$. Thus, we have $S(t) = S_1(t)$.
Similarly, we can show that $P(t) = P_1(t)$, $Q(t) = Q_1(t)$, $W(t) = W_1(t)$, $X(t) = X_1(t)$, $Y(t) = Y_1(t)$. This completes the proof of Theorem 3.

4.1 Positivity and Boundedness

The model (3) will be meaningful epidemiologically , if the solution of system (4) with no negative initial data will remain non-negative for all time $t \geq 0$ [18-26].

Lemma (1) . For all time $t \geq 0$ and initial data $N(0) \geq 0$, where $N(t) = (S(t), E(t), I(t), I_T(t), Q(t), R(t))$. The solution of model (4) are non-negative for all $t \geq 0$r if there exist , furthermore, $\limsup N(t) \leq \frac{\beta}{\mu}$ The epidemiologically feasible region of model (4) is given by:

$\Omega =: \{(\mathrm{S(t)}\ ,\ \mathrm{P(t)}, \mathrm{Q(t)}, \mathrm{W}_T(t), X(t), Y(t)) \in \mathbb{R}^6_+ :$
$0 \leq S(t) + P(t) + Q(t) + W(t) + X(t) + Y(t) \leq \mathbb{N} \leq \frac{\beta}{\mu}\}$

Proof:
Applying the AB fractional integral(2) to the first equation of (3) we have:

$$I_t^\alpha D_t^\alpha \left(S(t) e^{\int_0^t (\mu + \alpha_1 \zeta) dS} \right) = \beta I_t^\alpha \left(e^{\int_0^t (\mu + \alpha_1 \zeta) dS} \right)$$

it is obvious that:

$$S(t) = \beta e^{-\int_0^t (\mu + \alpha_1 \zeta) dS} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t (\mu + \alpha_1 \zeta) dS} + \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t (\mu + \alpha_1 \zeta) dS} (t-w) dw \right] > 0 \tag{4.12}$$

The same rationale applies to the remaining equations within (3).

$$\left.\begin{aligned}
\mathbf{P(t)} &= \mu + \alpha_1(t) e^{-\int_0^t \lambda_2 dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t \lambda_2 dt} + \right. \\
&\left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t \lambda_2 dt} (t-w) dw \right] > 0, \\
\mathbf{Q(t)} &= \lambda_2 E(t) e^{-\int_0^t (\mu + \alpha_3 + \lambda_1) dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t (\mu + \alpha_3 + \lambda_1) dt} + \right. \\
&\left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t (\mu + \alpha_3 + \lambda_1) dt} (t-w) dw \right] > 0, \\
\mathbf{W(t)} &= (1-\rho)\alpha_3 I(t) e^{-\int_0^t (\mu + \lambda_2) dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t (\mu + \lambda_2) dt} + \right. \\
&\left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t (\mu + \lambda_2) dt} (t-w) dw \right] > 0, \\
\mathbf{X(t)} &= \rho\alpha_3 I(t) e^{-\int_0^t \Psi dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t \Psi dt} + \right. \\
&\left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t \Psi dt} (t-w) dw \right] > 0, \\
\mathbf{Y(t)} &= \alpha_4 I_T(t) e^{-\int_0^t (\mu + \alpha_5) dt} \left[\frac{1-\alpha}{F(\alpha)} e^{\int_0^t (\mu + \alpha_5) dt} + \right. \\
&\left. \frac{\alpha}{F(\alpha)\Gamma(\alpha)} e^{\int_0^t (\mu + \alpha_5) dt} (t-w) dw \right] > 0,
\end{aligned}\right\} \tag{4.13}$$

From equations (17) and (18), it follows that each solution of equation (4) is non-negative and remains in $\mathbb{R}^6_+$.

Next, we establish the boundedness of the solutions of the fractional model (4), considering that all parameters in the model, as mentioned earlier, are non-negative. We proceed by summing up all equations of model (4), yielding:

$$D_t^\alpha N = \beta - \mu N - \lambda_2 I_T - \Psi Q(t) - \alpha_5 R(t)$$
$$D_t^\alpha N(t) - \mu N(t) \leq \beta$$
$$\left(D_t^\alpha N(t) - \mu N(t\right) e^{\int_0^t \mu dt} \leq \beta e^{\int_0^t \mu dt}$$
$$N(t) \leq \frac{\beta}{\mu}$$

It is not difficult to observe that $N(t) \rightarrow \frac{\beta}{\mu}$ as t $\rightarrow \infty$. Hence $\Omega =: \{(\mathrm{S(t)}\ ,\ \mathrm{P(t)}, \mathrm{Q(t)}, \mathrm{W}_T(t), X(t), Y(t)) \in \mathbb{R}^6_+$ is the biologically feasible region of (4).

4.2 Existence of Equilibria of the Model

Meaning of Equilibrium: If a point X' is an equilibrium, then the constant vector $X(t) = X'$ is a solution of the system of ordinary differential equations (ODEs) because a constant function has zero derivatives ($\frac{d}{dt}X' = 0$). Since $F(X') = 0$ by definition of equilibrium, we have $\frac{d}{dt}X' = F(X')$. Conversely, if a constant vector $X(t) = X'$ is a solution of $\frac{d}{dt}X' = F(X')$, then in other words, an equilibrium is a point where the solution remains constant forever. The point may be stable or unstable. An equilibrium point is hyperbolic if none of the eigenvalues have zero real part. If all the eigenvalues have negative real parts, the point is stable. If at least one has a positive real part, the point is unstable. Any dynamical system may have none, one, or several equilibrium points, each of which may either be stable or unstable. Understanding these equilibrium points provides important insights into the system behavior that characterizes the model.

4.3 Disease-Free Equilibrium

The disease-free equilibrium (DFE) of equation (3) is given by:

$$(S_0(t), P_0(t), Q_0(t), W_0(t), X_0(t), Y_0(t)) = \left(\frac{\beta}{\mu}, 0, 0, 0, 0, 0\right)$$

The global stability of the disease-free equilibrium point will be shown after defining the basic reproduction number.

4.4 Basic Reproduction Number R_0

The basic reproduction number R_0 is defined as the expected number of secondary infections produced when one infected individual is introduced into a completely susceptible population [33-32]. Computation of R_0 typically involves the product of infection rates and the duration of infection. The basic reproduction number is obtained using the next-generation matrix and is given by the spectral radius $\Phi(-FV^{-1})$, where:

$$F = \begin{pmatrix} 0 & \frac{b_0 a_1 \beta}{\mu k(N)} & \frac{b_0 a_2 \beta}{\mu k(N)} & \frac{b_0 a_3 \beta}{\mu k(N)} & \frac{b_0 a_4 \beta}{\mu k(N)} \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 & 0 \end{pmatrix}$$

$$V^{-1} = \begin{pmatrix} \frac{1}{K_1} & 0 & 0 & 0 & 0 \\ \frac{\lambda_2}{K_1K_2} & \frac{1}{K_2} & 0 & 0 & 0 \\ \frac{\lambda_2\lambda_3}{K_1K_2K_3} & \frac{\lambda_3}{K_2K_3} & \frac{1}{K_3} & 0 & 0 \\ \frac{\lambda_2\lambda_3\lambda_4}{K_1K_2K_3K_4} & \frac{\lambda_3\lambda_4}{K_2K_3K_4} & \frac{\lambda_4}{K_3K_4} & \frac{1}{K_4} & 0 \\ \frac{\lambda_2\lambda_3\lambda_5}{K_1K_2K_3K_5} & \frac{\lambda_3\lambda_5}{K_2K_3K_5} & \frac{\lambda_5}{K_3K_5} & 0 & \frac{1}{K_5} \end{pmatrix}$$

$\mathrm{R}_0 = \frac{\beta b_0\lambda_2(a_2\lambda_3K_4K_5+a_3\lambda_3\lambda_4K_5+a_4\lambda_3\lambda_5K_4+a_1K_3K_4K_5)}{K_1K_2K_3K_4K_5\mu}$.*Where,*
$K_1 = \mu+\delta_1+\lambda_2, K_2 = \mu+\delta_2+\lambda_3, K_3 = \mu+\delta_2+\lambda_4+\lambda_5, K_4 = \mu+\delta_4, K_5 = \mu+\delta_5$

Global Asymptotic Stability (GAS) of HBV in the Absence of Therapy at DFE

Theorem 4.1. *The disease-free equilibrium (DFE) state: $S(t)_0, P(t)_0, Q(t)_0, W(t)_0, X(t)_0, Y(t)_0 \equiv (\beta, 0, 0, 0, 0, 0)$ is globally asymptotically stable when $R_0 \leq -1$ [42-49].*

Proof. From equation (3), we construct a Lyapunov function of the form:

$$V(P,Q,W,X,Y) = A_1P + A_2Q + A_3W + A_4X + A_5Y$$

where $A_1 > 0$, $A_2 > 0$, $A_3 > 0$, $A_4 > 0$, $A_5 > 0$ are constants whose values will be determined. Taking the time derivatives of V, we have:

$$\frac{dV}{dt}\frac{dP}{dt} = A_1\frac{dP}{dt} + A_2\frac{dQ}{dt} + A_3\frac{dW}{dt} + A_4\frac{dX}{dt} + A_5\frac{dY}{dt} \tag{4.14}$$

Substituting $\frac{dP}{dt}$, $\frac{dQ}{dt}$, $\frac{dW}{dt}$, $\frac{dX}{dt}$ into equation (3), we have:

$$\begin{aligned} \frac{dV}{dt} = {} & A_1(\alpha_1\zeta S(t) - (\mu+\delta_1) + \lambda_2)P(t)) \\ & + A_2(\lambda_2P(t) - (\mu+\delta_2+\lambda_3)Q(t)) \\ & + A_3(\lambda_3Q(t) - (\mu+\delta_2+\lambda_4+\lambda_5)W(t)) \\ & + A_4(\lambda_4W(t) - (\mu+\delta_4)X(t)) \\ & + A_5(\lambda_5W(t) - (\mu+\delta_5)Y(t)) \end{aligned} \tag{4.15}$$

Solving for the constants A_1, A_2, A_3, A_4, and A_5 gives:

$$\left.\begin{aligned} A_1 &= \tau_1R_0 \\ A_2 &= \frac{K_1\tau_1R_0}{\lambda_2} \\ A_3 &= \frac{\tau_0}{K_3R_0} \\ A_4 &= \frac{\tau_0}{K_4R_0} \\ A_5 &= \frac{\tau_0}{K_5R_0} \end{aligned}\right\} \tag{4.16}$$

Where: $\tau_0 = b_0\lambda_2\left(a_2\lambda_3K_4K_5 + a_3\lambda_3\lambda_4K_5 + a_4\lambda_3\lambda_5K_4 + a_1K_3K_4K_5\right.$ and $\tau_1 = K_1K_2K_3K_4K_5\mu$ Substituting [22] into [21] and simplifying the resultant equation we have:

$$\begin{aligned} \frac{dV}{dt} = {} & \alpha_1\zeta S(t)\frac{\tau_1R_0}{\alpha_1} - K_1\frac{\tau_1R_0}{\alpha_1} - \lambda_1)P(t))\frac{K_1\tau_1R_0}{\alpha_1\lambda_2} + \\ & \frac{\tau_0}{K_3R_0}\lambda_3 - K_1)Q(t)\frac{K_1\tau_1R_0}{\alpha_1\lambda_2}) + \frac{\tau_0}{K_4R_0}\lambda_4 + \\ & (\frac{\tau_0}{K_5R_0}\lambda_5 - \frac{\tau_0}{R_0})W(t)) - \frac{\tau_0}{R_0}X(t) - \frac{\tau_0}{R_0}Y(t) \end{aligned} \tag{4.17}$$

Since all parameters and variables of model equations (1)-(6) are non-negative, it follows that $\frac{dV}{dt} \leq 0$ for $R_0 \leq 1$ in equation (1.34) with $\frac{dV}{dt} = 0$ if and only if $S(t) = P(t) = Q(t) = W(t) = X(t) = Y(t) = 0$. Hence, V is a Lyapunov function on $\mathcal{D}$.

Further, the largest compact invariant set in $\{(S(t), P(t), Q(t), W(t), X(t), Y(t)) \in \mathcal{D} : \dot{V} = 0\}$ is the singleton $\{\varepsilon\}$. Therefore, it follows from LaSalle's invariant principle that $(P(t), Q(t), W(t), X(t), Y(t)) \longrightarrow (0, 0, 0, 0, 0)$ as $t \to \infty$. Thus, every solution of the equations of the model (1)-(6) with initial conditions in $\mathcal{D}$ approaches $\{\varepsilon\}$ as $t \to \infty$ (whenever $R_0 \leq 1$), so that $\{\varepsilon\}$ is globally asymptotically stable (GAS) in $\mathcal{D}$ if $R_0 \leq 1$. The epidemiological implication of this theorem is that if R_0 can be made to a value less than unity, a small influx of individuals into the community will not generate large outbreaks of the disease, and it will die out in time.

4.5 Endemic Equilibrium State of HBV in the Absence of Therapy

At the endemic equilibrium state, the disease exists, and as such, $S(t) \neq P(t) \neq Q(t) \neq W(t) \neq X(t) \neq Y(t) \neq 0$, but $\frac{D}{dt}S(t) = \frac{D}{dt}P(t) = \frac{D}{dt}Q(t) = \frac{D}{dt}W(t) = \frac{D}{dt}X(t) = \frac{D}{dt}Y(t) = 0$. Our interest here is to determine the expression of equation (3) such that $S(t) = S^*(t)$, $P(t) = P^*(t)$, $Q(t) = Q^*(t)$, $W(t) = W^*(t)$, $X(t) = X^*(t)$, $Y(t) = Y^*(t)$ at the endemic equilibrium state. Solving the six equations of equation (3) algebraically in steps for the state variables gives:

$$\left.\begin{aligned} S^* &= \frac{\beta}{\mu(1+\alpha_1 R_0)} \\ P^* &= \frac{\alpha_1 \beta \mu R_0}{k_1 k_0} \\ Q^* &= \frac{\lambda_2 \alpha_1 \beta \mu R_0}{k_2 k_1 k_0} \\ W^* &= \frac{\lambda_3 \lambda_2 \alpha_1 \beta \mu R_0}{k_3 k_2 k_1 k_0} \\ X^* &= \frac{\lambda_4 \lambda_3 \lambda_2 \alpha_1 \beta \mu R_0}{k_4 k_3 k_2 k_1 k_0} \\ Y^* &= \frac{\lambda_5 \lambda_3 \lambda_2 \alpha_1 \beta \mu R_0}{k_5 k_3 k_2 k_1 k_0} \end{aligned}\right\} \tag{4.18}$$

4.6 Endemic Equilibrium State of HBV in the Absence of Therapy

Theorem 4.2. *If $R_0 > 1$, the endemic equilibrium point of model (4) is globally asymptotically stable.*

Proof To establish the global stability of the endemic equilibrium J^*, we construct a Lyapunov function of the form:.

$$\begin{aligned} F = S - \ddot{S} - \ddot{S} log\frac{S}{\ddot{S}} + \left(P - \ddot{P} - \ddot{P} log\frac{P}{\ddot{P}}\right) + \left(Q - \ddot{Q} - \ddot{Q} log\frac{Q}{\ddot{Q}}\right) + \\ \left(W - \ddot{W} - \ddot{W} log\frac{W}{\ddot{W}}\right) + \left(X - \ddot{X} - \ddot{X} log\frac{X}{\ddot{X}}\right) + \\ \left(Y - \ddot{Y} - \ddot{Y} log\frac{Y}{\ddot{Y}}\right) \end{aligned} \tag{4.19}$$

Differentiating F (i.e., equation (24)), we have:

$$\begin{aligned} \dot{F} = \dot{S} - \frac{\ddot{S}}{S}\dot{S} + \left(\dot{P} - \frac{\ddot{P}}{P}\dot{P}\right) + \left(\dot{Q} - \frac{\ddot{Q}}{Q}\dot{Q}\right) + \left(\dot{W} - \frac{\ddot{W}}{W}\dot{W}\right) + \\ \left(\dot{X} - \frac{\ddot{X}}{X}\dot{X}\right) + \left(\dot{Y} - \frac{\ddot{Y}}{Y}\dot{Y}\right) \end{aligned} \tag{4.20}$$

Substituting the corresponding right-hand side of equations (4) into (25) and simplifying, we have

$$\begin{aligned}\dot{F} = \beta - \mu S(t) - \alpha_1\zeta S(t) - \frac{\ddot{S}}{S}\big(\beta - \mu S(t) - \alpha_1\zeta S(t)\big) + \\ \left(\alpha_1\zeta S(t) - K_1P(t) - \frac{\ddot{P}}{P}(\alpha_1\zeta S(t) - K_1P(t)\right) + \\ \left(\lambda_2P(t) - (\mu + \delta_2 + K_2Q(t) - \frac{\ddot{Q}}{Q}(\lambda_2P(t) - K_2Q(t))\right) + \\ \left(\lambda_3Q(t) - K_3W(t) - \frac{\ddot{W}}{W}(\lambda_3Q(t) - K_3W(t))\right) + \\ \left(\lambda_4W(t) - K_4X(t) - \frac{\ddot{X}}{X}(\lambda_4W(t) - K_4X(t))\right) + \\ \left(\lambda_5W(t) - K_5Y(t) - \frac{\ddot{Y}}{Y}(\lambda_5W(t) - K_5Y(t))\right)\end{aligned} \tag{4.21}$$

At steady state, we observe from model 4 that:

$$\beta = \alpha_1\ddot{S}(\ddot{Q} + \ddot{W} + \ddot{X} + \ddot{Y}) + \mu\ddot{S} \tag{4.22}$$

Following a procedure provided in [30-34], Equation (26) simplifies to:

$$\begin{aligned}\dot{F} = \mu\ddot{S}\left(2 - \frac{S}{\ddot{S}} - \frac{\ddot{S}}{S}\right) + \alpha_1\ddot{S}\ddot{Q}\left(3 - \frac{1}{S} - \frac{\ddot{Q}P}{\ddot{P}}\right) + \\ \alpha_1\ddot{S}\ddot{W}\left(3 - \frac{1}{S} - \frac{P}{\ddot{P}} + \frac{Q}{\ddot{Q}} - \frac{\ddot{X}Q}{\ddot{Q}W} - \frac{\ddot{Y}Q}{\ddot{Q}W} - \frac{\ddot{W}Q}{\ddot{Q}W}\right) + \\ \alpha_1\ddot{S}\ddot{X}\left(4 - \frac{1}{S} - \frac{P}{\ddot{P}} + \frac{Q}{\ddot{Q}} - \frac{\ddot{W}Q}{\ddot{Q}W} - \frac{\ddot{X}Q}{\ddot{Q}X}\right) + \\ \alpha_1\ddot{S}\ddot{X}\left(4 - \frac{1}{S} - \frac{P}{\ddot{P}} + \frac{Q}{\ddot{Q}} - \frac{\ddot{W}Q}{\ddot{Q}W} - \frac{\ddot{Y}W}{\ddot{Q}Y}\right) - \\ \left(\frac{\alpha_1\ddot{P}}{P} + 2\alpha_1\ddot{S}\right)(Q + W + X + Y)\end{aligned} \tag{4.23}$$

Given that the arithmetic mean consistently surpasses the geometric mean, as inferred from (42), we can deduce the ensuing inequality:

$2 - \frac{S}{\ddot{S}} - \frac{\ddot{S}}{S} \leq 0$

$3 - \frac{1}{S} - \frac{\ddot{Q}P}{\ddot{P}} \leq 0, 3 - \frac{1}{S} - \frac{P}{\ddot{P}} + \frac{Q}{\ddot{Q}} - \frac{\ddot{X}Q}{\ddot{Q}W} - \frac{\ddot{Y}Q}{\ddot{Q}W} - \frac{\ddot{W}Q}{\ddot{Q}W} \leq 0$

$4 - \frac{1}{S} - \frac{P}{\ddot{P}} + \frac{Q}{\ddot{Q}} - \frac{\ddot{W}Q}{\ddot{Q}W} - \frac{\ddot{X}Q}{\ddot{Q}X} \leq 0, 4 - \frac{1}{S} - \frac{P}{\ddot{P}} + \frac{Q}{\ddot{Q}} - \frac{\ddot{W}Q}{\ddot{Q}W} \leq 0$

Further, since all the model parameters are non-negative, it follows that $\dot{F} < 0$ for $R_0 > 1$, and F is a Lyapunov function on D. The biological implication of this theorem is that when R_0 is greater than unity, a small influx of infected individuals into a community will generate large outbreaks of the disease, allowing it to invade the population.

5 Numerical Simulation (Absence of Control)

The aim of this section is to simulate and document the influence of various values of the fractional order α within the model. To achieve this goal, we perform numerous numerical simulations utilizing Python along with

its libraries(Simpy 4.1.1).The parameters, including their descriptions, values, and sources, are summarized in Table 1. Following Table 1, the numerical simulations and their corresponding descriptions are presented.

Table 1. Parameters of the Model, their Descriptions,Values and Source

Parameters	Description of parameter	Values	Source
β	level of production of new target cells	500	Assumed
μ	Natural death rate	0.021	[52]
α_1	Transition rate from S to P	0.50	[52]
δ_1	Disease induse rate at the subclinical state	0.03	[51]
λ_2	Fraction of subclinical persons that were clinical	0.50	[52]
λ_3	Fraction of clinical that migrate to acute	0.048	[52]
λ_4	Fraction of Acute that developed chronic HBV	0.025	[53]
λ_5	Fraction of Acute that developed fulminant HBV	0.08	[53])
δ_2	Disease induse rate at the clinical state	0.068	[52]
δ_3	Disease induse rate at the acute state	0.0068	[52]
δ_4	Disease induse rate due to chronic infection	0.068	[52]
δ_5	Disease induse rate due to fulminant infection	0.03	[53]

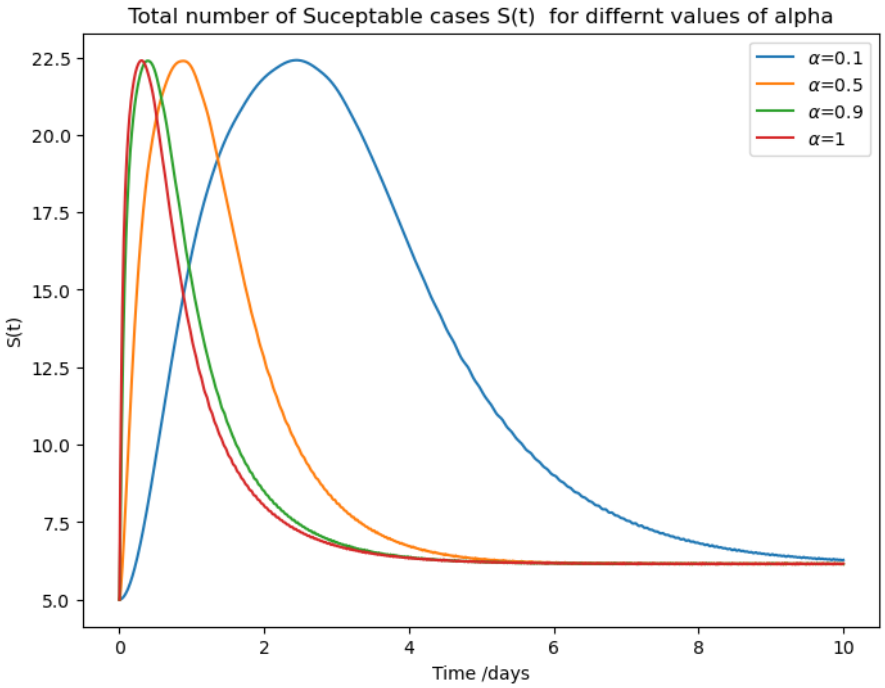

Fig. 2. Susceptible Population

Observations:
1. Impact of alpha on Susceptible Population:

- As alpha increases from 0.1 to 1, the peak of S(t) shifts to the left, indicating that the maximum number of susceptible cases is reached more quickly.

- The height of the peak decreases with increasing alpha, showing that higher values of alpha lead to a lower number of susceptible individuals at the peak.

2. Behavior Over Time:
- For alpha = 0.1, the curve peaks later and higher, meaning that the disease spreads more slowly, and a larger number of individuals remain susceptible for a longer period.
- As alpha increases, the curve not only peaks earlier but also returns to the baseline level more quickly, suggesting that the disease spreads faster and the susceptible population decreases more rapidly.

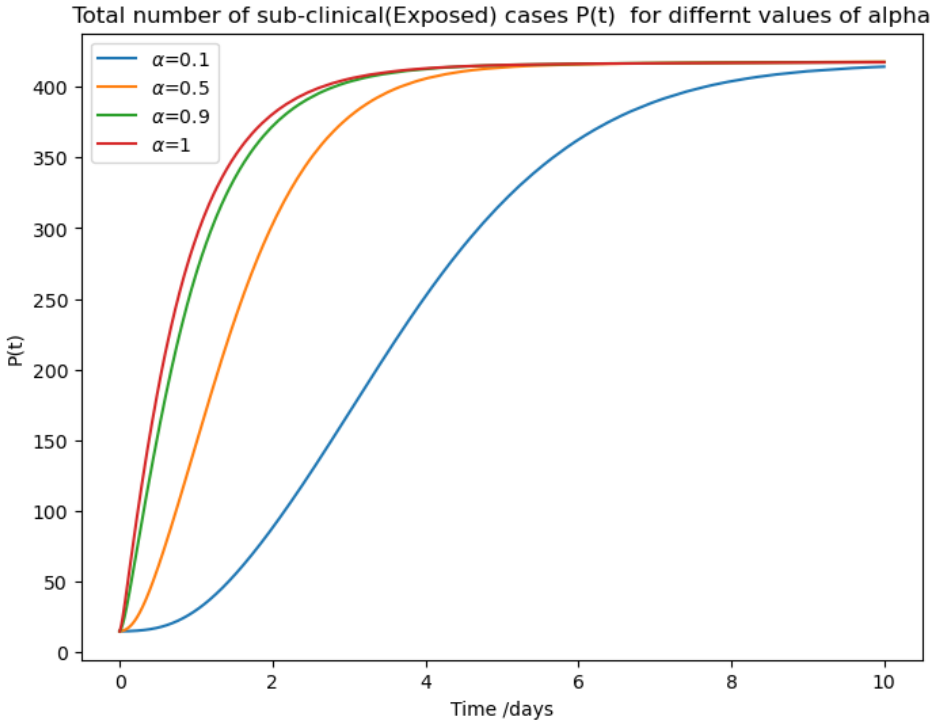

Fig. 3. Exposed Humans Population

Observations:
1. Effect of alpha on the Rate of Increase:
- As alpha increases from 0.1 to 1, the curve becomes steeper, indicating a faster increase in the number of sub-clinical cases P(t) over time.
- For alpha = 1 , which corresponds to the classical first-order derivative, the number of cases rapidly increases and quickly approaches its peak value
- As alpha decreases, the growth of P(t) becomes slower, indicating that the system's response to the infection is more gradual.

2. Long-Term Behavior:

- Regardless of the value of alpha , all curves appear to approach the same asymptotic value, suggesting that the total number of sub-clinical cases stabilizes at a similar final value. This reflects the system reaching a steady state where the number of new cases levels off.

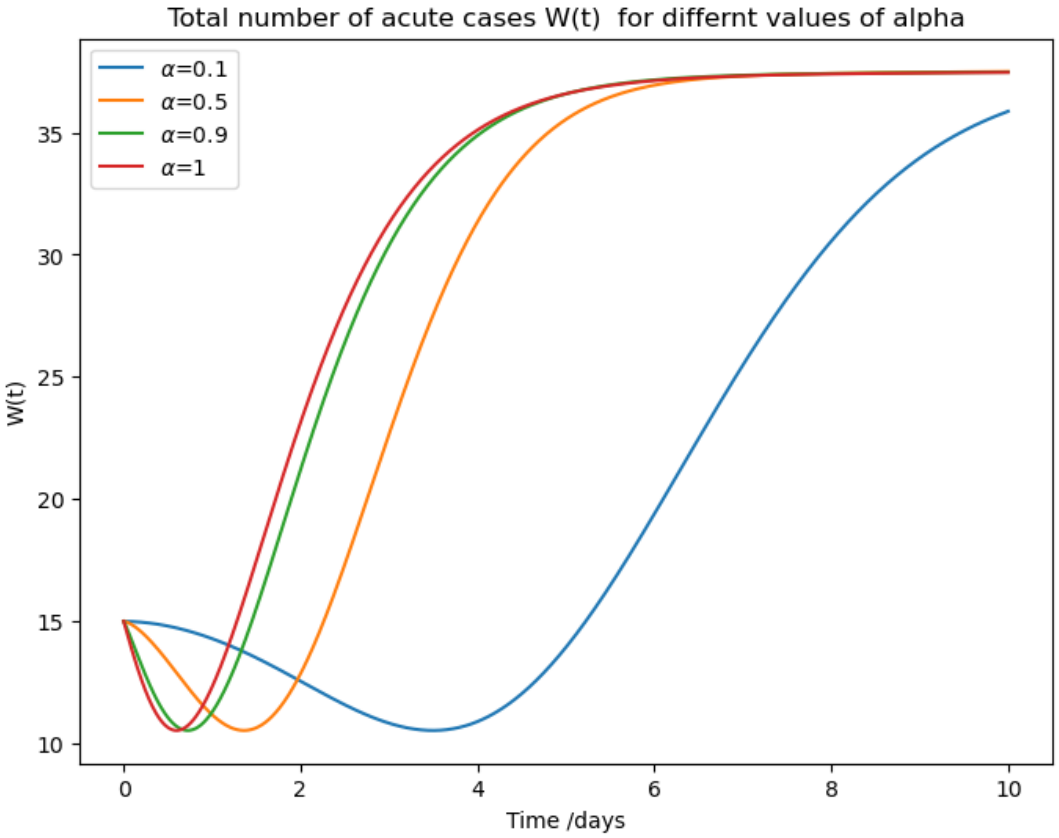

Fig. 4. Acute Infected Humans Population

Observations:
The graph illustrates the impact of varying the fractional order α in modeling the acute human population $W(t)$ during Hepatitis B virus (HBV) transmission. Lower α values introduce a memory effect, leading to delayed and oscillatory dynamics, which biologically may represent the influence of past infections and interventions on current disease spread. This suggests that the population takes longer to stabilize, reflecting the complexities of immune responses and chronic carrier states in HBV. In contrast, higher α values result in a more rapid and smooth rise in acute cases, indicating a quicker stabilization, possibly corresponding to populations with less complex immune histories or more uniform responses to the infection. This highlights the fractional-order model's ability to capture the nuanced transmission dynamics of HBV, offering insights that can improve public health strategies.

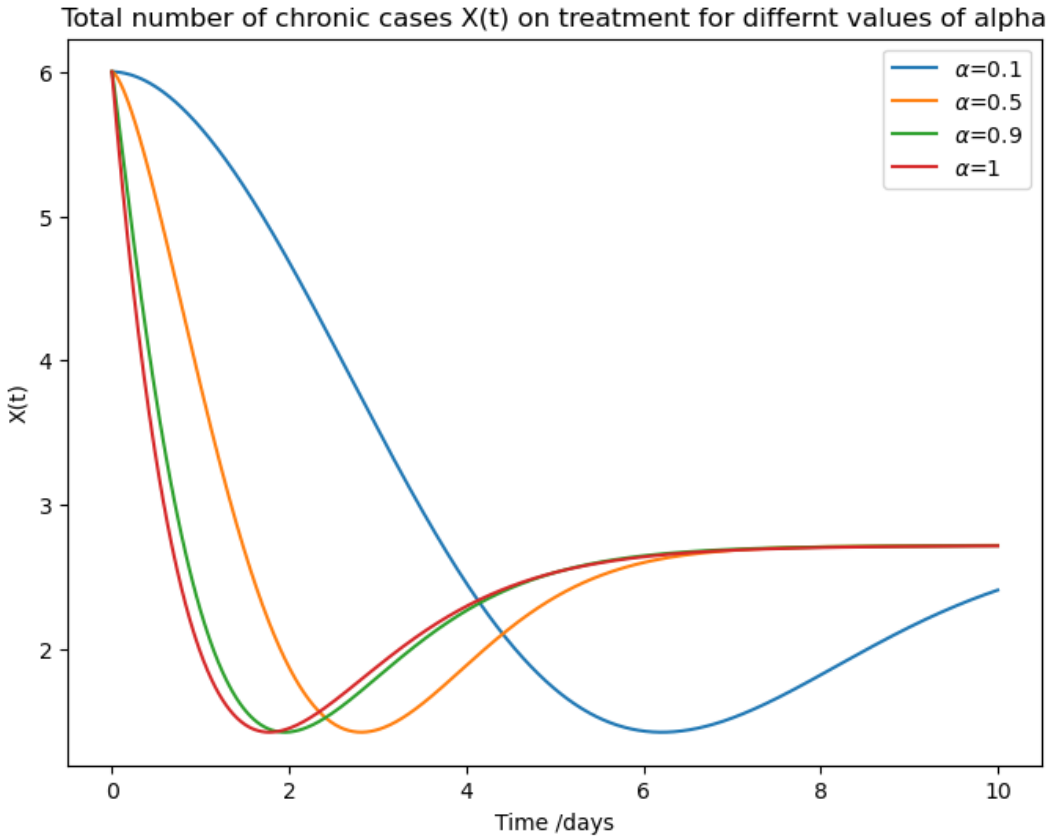

Fig. 5. Chronic Humans Population

Observations:
The graph represents the evolution of the chronic human population X(t) in the context of Hepatitis B virus (HBV) transmission, modeled using a fractional-order differential system with varying values of alpha. The parameter alpha influences how past states of the system (e.g., prior infections, immune responses) affect the current rate of chronic case accumulation. Biologically, lower values of alpha (e.g., alpha = 0.1 indicate a stronger memory effect, which slows the initial increase in chronic cases and introduces a more gradual rise over time. This suggests that when memory effects are significant, the progression from acute to chronic infection is more gradual, possibly reflecting the prolonged time it takes for the immune system to react to the infection, or the delayed effects of interventions like antiviral treatments or vaccinations. As alpha increases, the memory effect diminishes, and the rise in chronic cases becomes quicker and more immediate. For alpha = 1, the population rapidly reaches a higher number of chronic cases, reflecting a scenario where the transition from acute to chronic infection is more direct and less influenced by past infection history. This could represent populations with a more uniform or immediate immune response to HBV or where the disease progression is faster due to other factors.

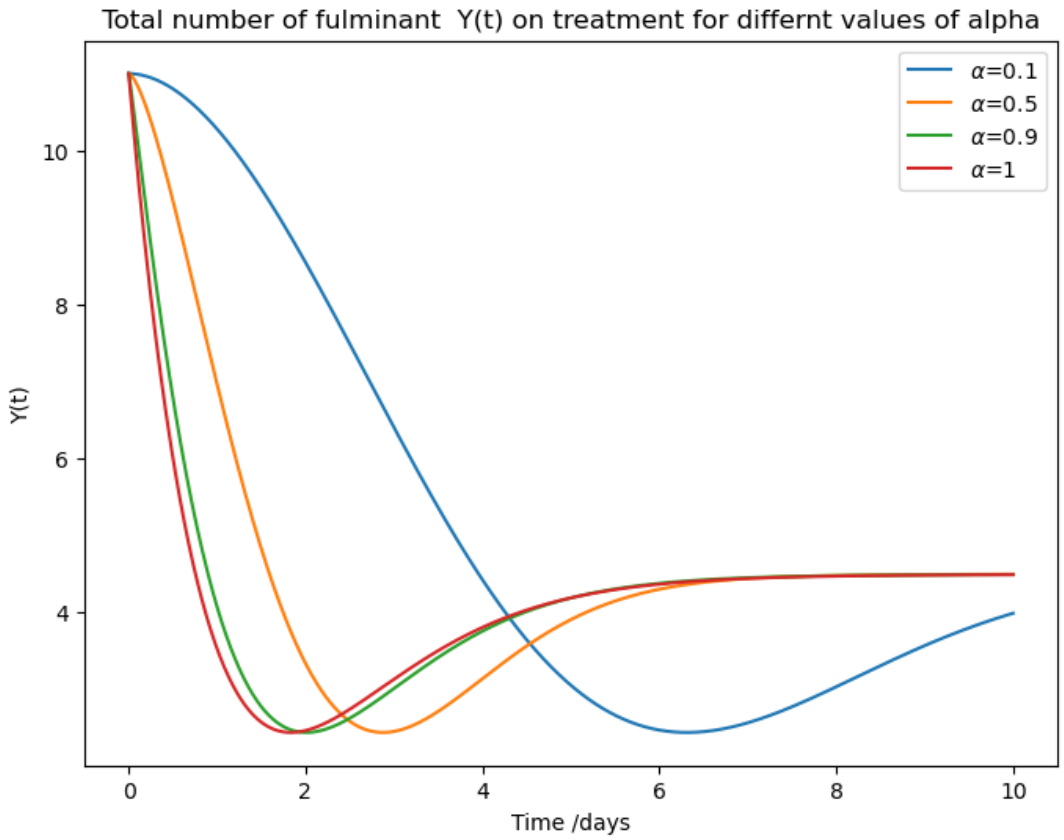

Fig. 6. Fulminant Humans Population

Observations:
The graph displays the time evolution of the fulminant hepatitis B cases Y(t) under different fractional orders alpha over a 10-day period. Each curve represents a different value of alpha, from alpha = 0.1 to alpha = 1.
1. Impact of alpha on (Y(t) :
For alpha = 0.1, the initial decrease in fulminant cases is slower, and the system exhibits notable oscillations before reaching a steady state. As alpha increases, the initial decrease becomes steeper, and the oscillations dampen more quickly, leading to a faster stabilization of fulminant cases.
Biological Interpretation:
Lower alpha (e.g., alpha = 0.1) suggests a strong memory effect, meaning the current state of the fulminant cases is heavily influenced by past states. This can represent the biological scenario where the history of infection, immune response, and treatment has a prolonged influence on the progression and resolution of fulminant hepatitis. The oscillatory behavior for lower alpha values indicates a more complex dynamic where patients might experience fluctuating symptoms or treatment responses before stabilization.
2. Disease Progression and Treatment:
- For higher alpha values, the fulminant cases decline rapidly and stabilize more quickly, implying a more immediate response to treatment and less influence from past states. This could reflect scenarios where effective treatment is quickly administered, leading to a rapid decline in severe cases without significant fluctuation. The different trajectories also suggest that the progression from fulminant to recovery or chronic states can vary significantly based on the population's historical exposure and treatment efficacy.

3. Epidemiological Implications:

The variation in the trajectories of Y(t) with different alpha values highlights the importance of considering fractional-order models in capturing the nuanced dynamics of fulminant HBV cases.

6 Summary of Findings

HBV remains a significant global health concern, with transmission dynamics influenced by various factors. Traditional models have limitations in capturing the complexities of HBV transmission dynamics. Hence, the study utilized a fractional order model to provide a more accurate representation. The study elucidated the dynamics of HBV transmission using a fractional order model and highlighted the effectiveness of enlightenment intervention as a control strategy. The findings underscore the importance of considering complex dynamics and intervention strategies in combating infectious diseases like HBV. Further research is warranted to refine the model and assess the long-term impact of enlightenment interventions on HBV transmission rates. Fractional Order Model Formulation: The formulation of a fractional order model of Hepatitis B virus (HBV) transmission dynamics using the Atangana -Baleanu operator represents a significant contribution to the field of infectious disease modeling. By leveraging fractional calculus principles, we have developed a model that accounts for memory effects and long-range dependencies, providing a more accurate representation of HBV transmission dynamics compared to traditional models. Insights into Complex Dynamics: The fractional order model offers insights into the complex dynamics of HBV transmission, elucidating the interplay between various factors such as population demographics, vaccination coverage, and intervention strategies. By capturing the intricate temporal dependencies inherent in infectious disease dynamics, the model enhances our understanding of HBV transmission patterns and informs targeted control measures.

Disclaimer(Artifical Intelligence)

This research paper, titled "Mathematical Analysis of Hepatitis B Virus Transmission Dynamics in the Absence of Therapy with Atangana-Baleanu Fractional-Order SPQWXY Model," was developed using traditional mathematical modeling techniques and did not rely on artificial intelligence (AI) or machine learning algorithms for the primary research, data analysis, or conclusions drawn. The theoretical framework, model formulation, and validation were based on well-established mathematical methodologies, including fractional calculus and numerical simulations, without the use of AI-driven automation tools. Any tools, software, or platforms utilized during the preparation of this manuscript, such as text editing software or statistical packages, were limited to manual inputs by the authors and did not involve AI-generated content, analysis, or automated decision-making processes. The responsibility for the accuracy, integrity, and scientific rigor of the results rests solely with the authors.

Competing Interests

Authors have declared that no competing interests exist.

References

[1] Atangana A, Goufo EFD. Modern and generalized analysis of exogenous growth models. Chaos, Solitons and Fractals. 2022;163:112605. Available:https://doi.org/10.1016/j.chaos.2022.112605

[2] Huppert A, Katriel G. Mathematical modelling and prediction in infectious disease epidemiology. Clinical Microbiology and Infection. 2013;19(11):999–1005. Available:https://doi.org/10.1111/1469-0691.12308

[3] Misra AK, Sharma A, Shukla JB. Modeling and analysis of effects of awareness programs by media on the spread of infectious diseases. Mathematical and Computer Modelling. 2011;53(5–6):1221–1228.
Available:https://doi.org/10.1016/j.mcm.2010.12.018

[4] Da Costa ALP, Neto OAR, Silva-Júnior ACS. Conditioners of the infectious diseases dynamics. Estação Científica (UNIFAP). 2019;8(3):09–23.

[5] Mack A, Choffnes ER, Relman DA. (Eds.). Infectious disease Movement in a borderless world: Workshop summary. National Academies Press; 2010.

[6] Fauci AS. Infectious diseases: Considerations for the 21st century. Clinical Infectious Diseases. 2001;32(5):675–685.
Available:https://doi.org/10.1086/319235

[7] Scherer A, McLean A. Mathematical models of vaccination. British Medical Bulletin. 2002;62(1):187–199.
Available:https://doi.org/10.1093/bmb/62.1.187

[8] Mounts AW, Holman RC, Clarke MJ, Bresee JS, Glass RI. Trends in hospitalizations associated with gastroenteritis among adults in the United States, 1979–1995. Epidemiology and Infection. 1999;123(1):1–8.
Available:https://doi.org/10.1017/s0950268899002675

[9] Brechot C, Degos F, Luggassy C, Thiers H, Zafrani S, Franco D, et al. Hepatitis B virus DNA in patients with chronic liver disease and negative tests for hepatitis B surface antigen. New England Journal of Medicine. 1985;312:270.
Available:https://doi.org/10.1056/NEJM198501313120501

[10] Brechot C, Thiers V, Kremsdorf D, Nalpas B, Pol S, Paterlini-Brechot P. Persistent hepatitis B virus infection in subjects without hepatitis B surface antigen. Hepatology. 2001;34(1):194-203.
Available:https://doi.org/10.1053/jhep.2001.26632

[11] Chen DS, Hsu NH, Sung JL, Hsu TC, Hsu ST, Kuo YT. A mass vaccination program in Taiwan against hepatitis B virus infection in infants of hepatitis B surface antigen-carrier mothers. Journal of the American Medical Association. 1987;257(19):2597–2603.
Available:https://doi.org/10.1001/jama.1987.03390190103031

[12] Martin DL, Gustafson TL, Pelosi JW, Suarez L, Pierce GV. Contaminated produce—a common source for two outbreaks of Shigella gastroenteritis. American Journal of Epidemiology. 1986;124(2):299–305.
Available:https://doi.org/10.1093/oxfordjournals.aje.a114407

[13] European Consensus Group on Hepatitis B Immunity. Are booster immunisations needed for lifelong hepatitis B community? The Lancet. 2000;355(9203):561–565.
Available:https://doi.org/10.1016/S0140-6736(00)02095-6

[14] Bosch FX, Ribes J, Borras J. Epidemiology of primary liver cancer. Seminars in Liver Disease. 1999;19(3):271–285.
Available:https://doi.org/10.1055/s-2007-1007111

[15] Brauer F, Castillo-Chavez C. Mathematical models in population biology and epidemiology. Springer. 2012;2.

[16] Samonis G, Elting L, Skoulika E, Maraki S, Tselentis Y. An outbreak of diarrhoeal disease attributed to Shigella sonnei. Epidemiology and Infection. 1994;112(2):235–245.
Available:https://doi.org/10.1017/S0950268800057764

[17] Mandell GL, Bennett JE, Dolin R. Plasmodium species. In Mandell, Douglas and Bennet's principles and practice of infectious diseases. Churchill Livingstone. 2000;2817–2830.

[18] Chen HL, Chang MH, Ni YH, Hsu HY, Lee PI, Lee CY. Seroepidemiology of hepatitis B virus infection in children: Ten years of mass vaccination in Taiwan. Journal of the American Medical Association. 1996;276(12):906–908.
Available:https://doi.org/10.1001/jama.1996.03540120058032

[19] Chan HL, Leung NW, Hussain M, Wong ML, Lok AS. Hepatitis B e antigen-negative chronic hepatitis B in Hong Kong. Journal of Hepatology. 2000;31(4):763–768.
Available:https://doi.org/10.1016/S0168-8278(00)80127-6

[20] Hethcote HW. The mathematics of infectious diseases. SIAM Review. 2000;42(4):599–653.
Available:https://doi.org/10.1137/S0036144500371907

[21] Hethcote HW. Three basic epidemiological models. In Levin S. (Ed.). Applied Mathematics in Ecology. Springer. 1989;119–144.
Available:https://doi.org/10.1007/978-1-4612-3472-0_8

[22] Nåsell I. Stochastic models of some endemic infections. Mathematical Biosciences. 2002;179(1):1–19.
Available:https://doi.org/10.1016/S0025-5564(02)00098-7

[23] Glasser J, Meltzer M, Levin B. Mathematical modeling and public policy: Responding to health crises. Emerging Infectious Diseases. 2004;10(11):2050–2051.
Available:https://doi.org/10.3201/eid1011.040698

[24] Huo J, Zhao H, Zhu L. The effect of vaccines on backward bifurcation in a fractional order HIV model. Nonlinear Analysis: Real World Applications. 2015;26:289–305.
Available:https://doi.org/10.1016/j.nonrwa.2015.01.006

[25] Taubenberger JK, Morens DM. 1918 influenza: The mother of all pandemics. Revista Biomedica. 2006;17(1):69–79.

[26] Zhou JJ, Wu WS, Sun CM, Dai HQ, Zhou N, Liu CB. Long-term evaluation of immune efficacy among newborns 16 years after HBV vaccination. Chinese Journal of Vaccines and Immunization. 2003;9:129–133.

[27] Ikeda K, Arase Y, Kobayashi M, Someya T, Saitoh S, Suzuki Y. Consistently low hepatitis B virus DNA saves patients from hepatocellular carcinogenesis in HBV-related cirrhosis: A nested case-control study using 96 untreated patients. Journal of Intervirology. 2003;46:96–104.
Available:https://doi.org/10.1159/000069540

[28] Hu KQ. Occult hepatitis B virus infection and its clinical implications. Journal of Viral Hepatitis. 2002;9:243–257.
Available:https://doi.org/10.1046/j.1365-2893.2002.00337.x

[29] Dietz K, Heesterbeek JAP. Daniel Bernoulli's epidemiological model revisited. Mathematical Biosciences. 2002;180(1–2):1–21.
Available:https://doi.org/10.1016/S0025-5564(02)00107-0

[30] Du M, Wang Z, Hu H. Measuring memory with the order of fractional derivative. Scientific Reports. 2013;3(1):3431.
Available:https://doi.org/10.1038/srep03431

[31] Glomski M, Ohanian E. Eradicating a disease: Lessons from mathematical epidemiology. The College Mathematics Journal. 2012;43(2):123–132.
Available:https://doi.org/10.4169/college.math.j.43.2.123

[32] Jit M, Brisson M.Modelling the epidemiology of infectious diseases for decision analysis: A primer. PharmacoEconomics. 2011;29(5):371–386.
Available:https://doi.org/10.2165/11585590-000000000-00000

[33] Chen M, Chen CJ, Lai MS. Universal hepatitis B vaccination in Taiwan and the incidence of hepatocellular carcinoma in children. The Taiwan Childhood Hepatoma Study Group. The New England Journal of Medicine. 1997;336:1855–1859.
Available:https://doi.org/10.1056/NEJM199706263362602

[34] Funk ML, Rosenberg DM, Lok ASF. Worldwide epidemiology of HBeAg-negative chronic hepatitis B and associated precore and core promoter variants. Journal of Viral Hepatitis. 2002;9:52–61.
Available:https://doi.org/10.1046/j.1365-2893.2002.00329.x

[35] Abdelouahab MS, Hamri NE. The Grünwald-Letnikov fractional-order derivative with fixed memory length. Journal Details Missing; 2002.

[36] Puri N, Coomes EA, Haghbayan H, Gunaratne K. Social media and vaccine hesitancy: New updates for the era of COVID-19 and globalized infectious diseases. Human Vaccines and Immunotherapeutics. 2020;16(11):2586–2593.
Available:https://doi.org/10.1080/21645515.2020.1780846

[37] Wismans P, Van Hattum J, De Gast GC. A prospective study of in vitro anti-HBs producing B cells (spot-ELISA) following primary and supplementary vaccination with recombinant hepatitis B vaccine in insulin-dependent diabetic patients and matched controls. Journal of Medical Virology. 1991;5:216–222.

[38] Roeder P, Mariner J, Kock R. Rinderpest: The veterinary perspective on eradication. Philosophical Transactions of the Royal Society B: Biological Sciences. 2013;368(1623):20120139.

Available:https://doi.org/10.1098/rstb.2012.0139

[39] Ayerbe MC, Perez-Rivilla A, ICOVAHB Group. Assessment of long-term efficacy of hepatitis B vaccine. European Journal of Epidemiology. 2001;17:151–156.
Available:https://doi.org/10.1023/A:1017904900541

[40] Yu Q, Reutens D, O'Brien K, Vegh V. Tissue microstructure features derived from anomalous diffusion measurements in magnetic resonance imaging. Human Brain Mapping. 2017;38(2):1068–1081.
Available:https://doi.org/10.1002/hbm.23436

[41] Yu Q, Reutens D, Vegh V. Can anomalous diffusion models in magnetic resonance imaging be used to characterise white matter tissue microstructure? Neuroimage. 2018;175:122–137.

[42] Baker R, Mahmud A, Miller I. Infectious disease in an era of global change. Nature, Mediterr. J. Math. 2021;13(2):557–572.

[43] Beasley RP, Hwang LY, Lin CC, Chien CS. Hepatocellular carcinoma and hepatitis B virus: A prospective study of 22,707 men in Taiwan. 1981;2:1129–1133.

[44] Anderson RM, May RM. Infectious diseases of humans: Dynamics and control. Oxford University Press; 1992.

[45] May RM. Uses and abuses of mathematics in biology. Science. 2004;303(5659):790–793.

[46] Hadziyannis S. Hepatitis B e antigen-negative chronic hepatitis B: From clinical recognition to pathogenesis and treatment. Viral Hepatitis Review. 1995;1:7–36.

[47] Goldstein ST, Alter MJ, Williams IT, Moyer LA, Judson FN, Mottram K. Incidence and risk factors for acute hepatitis B in the United States, 1982–1998: Implications for vaccination programs. Journal of Infectious Diseases. 2002;185:713–719.

[48] Panda SK, Atangana A, Abdeljawad T. Existence results and numerical study on novel coronavirus 2019-NCoV/SARS-CoV-2 model using differential operators based on the generalized Mittag-Leffler kernel and fixed points. Fractals. 2022;30(08):2240214.

[49] Qin S, Liu F, Turner I, Vegh V, Yu Q, Yang Q. Multi-term time-fractional Bloch equations and application in magnetic resonance imaging. Journal of Computational and Applied Mathematics. 2017;319:308–319.

[50] Rida SZ, Rady AA, Arafa AAM, Khalil M. Approximate solution of a fractional order model of HCV infection with drug therapy effect. International Journal of Applied Mathematics. 2012;1(2):108–116.

[51] Straif-Bourgeois S, Ratard R. Infectious disease epidemiology. In Handbook of Epidemiology. 2005;1327–1362.

[52] Ishikawa T, Ichida T, Yamagiwa S, Sugahara S, Uehara K, Okoshi S. High viral loads, serum alanine aminotransferase, and gender are predictive factors for the development of hepatocellular carcinoma from viral compensated liver cirrhosis. Journal of Gastroenterology and Hepatology. 2001;16:1274–1281.

[53] Wu T, Perrings C, Kinzig A, Collins JP, Minteer BA, Daszak P. Economic growth, urbanization, globalization, and the risks of emerging infectious diseases in China: A review. Ambio. 2017;46:18–29.

[54] Otugene VB, Agina PC, Edogbaya H, Digwo DC. Existence and uniqueness analysis of Atangana-Baleanu fractional order network model of Noro virus. International Journal of Mathematical and Applied Mathematics. 2022;5:2682–5708.

[55] Buckwold VE, Xu Z, Chen M, Yen TS, Ou JH. Effects of a naturally occurring mutation in the hepatitis B virus basal core promoter on precore gene expression and viral replication. Journal of Virology. 1996;70:5845–5851.

[56] Carman WF, Jacyna MR, Hadziyannis S, Karayiannis P, McGarvey MJ, Makris A, et al. Mutation preventing formation of hepatitis B e antigen in patients with chronic hepatitis B infection. Lancet Infectious Diseases. 1989;8663:588–591.

[57] Swaddiwudhipong W, Karintraratana S, Kavinum S. A common-source outbreak of shigellosis involving a piped public water supply in northern Thai communities. Journal of Tropical Medicine and Hygiene. 1995;98:145–145.

[58] Kermack WO, McKendrick AG. A contribution to the mathematical theory of epidemics. Proceedings of the Royal Society of London. Series A, Containing Papers of a Mathematical and Physical Character. 1927;115(772):700–721.

[59] Chen Y, Liu F, Yu Q, Li T. Review of fractional epidemic models. Applied Mathematical Modeling. 2021;97:281–307.

[60] Mukandavire Z, Garira W, Tchuenche JM. Modelling effects of public health educational campaigns on HIV/AIDS transmission dynamics. Applied Mathematical Modeling. 2009;33(4):2084–2095.

[61] Wang Z, Andrews MA, Wu ZX, Wang L, Bauch CT. Coupled disease–behavior dynamics on complex networks: A review. Physics of Life Reviews. 2015;15:1–29.

Peer-review history:
The peer review history for this paper can be accessed here (Please copy paste the total link in your browser address bar)
https://www.sdiarticle5.com/review-history/124589