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Numerical Solution of Fractional order Hepatitis B Model Via the Generalized Fractional Adams-Bashforth-Moulton Approach

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ABSTRACT
In this paper we examine the epidemiological properties of Hepatitis B virus (HBV) infection based on the equation of a fractional-order mathematical model based on the Caputo derivative. The model uses interventions such as treatment and vaccination as control measures to examine the effect that they have on disease dynamics. We define the presence and uniqueness of solutions in the framework of the fractional order and test the stability of the endemic equilibrium point based on the theory of Lyapunov functions. The model is numerically solved with the help of the fractional Adams-Bashforth-Moulton method to indicate changes in the model parameters and their respective fractional orders into how each one of the above parameters affects the progress of the disease. The use of simulation shows that higher treatment and vaccination rates decreases the prevalence of Hepatitis B and shows the high level of flexibility and realism of the fractional-order models in contrast to the classical integer order equations. In the paper, the importance of fractional modeling in the representation of the effects of memory and nonlocal interaction among the biological systems is highlighted, which enhances the understanding and control of infectious diseases. The model however assumes that the population is homogeneous mixed, and hypothetical values of the parameters thus restrains empirical validation. To make the model more predictive and relevant in practical use in formulating effective control measures on Hepatitis B, future studies need to include spatial heterogeneity, stochastic effects.

INTRODUCTION

Hepatitis B virus (HBV) is known to cause liver cancer and is identified as a leading cause of the disease as it is known to cause about 80 percent of the reported cases. HBV infection is contracted via contaminated body fluids of blood, semen and vaginal fluid Mahon (2005), Lavanchy (2004). The virus is one of the major causes of liver morbidity and a significant health problem of the population that requires immediate consideration. Consequently, there is need to set effective preventive measures to counter the effects of this disease on the healthcare system and lessen the resulting health effects. Determining and applying effective measures in order to

forecast, manage, and eventually wipe out HBV infection is a serious issue to the societal health.

The mathematical models are necessary to study health-related issues in the society as they assist in determining and comprehending the main ecological and biological determinants that govern disease transmission. The transmission patterns of HBV have been studied through hundreds of studies conducted by researchers interested in the aspect of mathematical epidemiology. Conventionally, the majority of the literature on HBV dynamics has been based on integer-order systems of differential equations to further scientific knowledge of HBV spread (Khan et al. (2019), Mann. and Roberts (2011), Chang (2007), Thornley et al. (2008), Liu et al. (2011), Liu et al. (1987), Ren et al. (2012), Boukanjime and Fatini (2019).

Fractional differential equations are an extension of traditional integer-order models to a more general model, and allow a better description of complex dynamical systems. In this paper we construct a mathematical model of the behavior of the transmission of the Hepatitis B virus at fractional-order level including the parameters of treatment and vaccination campaigns. Due to the memory effect caused by the nature of the fractional calculus, this model offers a better representation of the process by which Hepatitis B spreads, by simulating the various situations of the interventions, we can find out the best ways to minimize the prevalence of Hepatitis B.

With their capacity to capture the memory and hereditary properties of biology, which implies that they consider multiple addictive agents of medical conditions, such as Hepatitis B, fractional derivatives possess a considerable level of effectiveness in the modeling of several infectious diseases. They allow a more detailed study of the evolution of infection throughout the process, considering the impact of historical experience of infection and treatment regimens on the present process of transmission. This improved vision helps to generate more realistic and effective control strategies to solve the challenges of drug resistance, re-infection as well as insufficient healthcare resources.

Another interesting area has been the history of fractional calculus and its notable advances, to which Atokolo et al. (2022) introduce new information, as they enable the modeling of the dynamic nature of complex systems. In contrast to classical integer-order models that only local characteristics of systems are considered, the fractional-order models include memory-effects and therefore are more likely to describe the global dynamics of systems. Such models are more realistic as well as more appropriate to depict real-life phenomena. They can therefore offer a strong guideline towards a better understanding the transmission process of infectious diseases like Hepatitis B and strategies can be developed to control their transmission.

The derivatives of the Caputo and Riemann-Liouville with singular kernels have been extensively applied in multiple biological applications. Non-singular kernel derivatives, including the Mittag-Leffler and the AtanganaBaleanu operators, have also become very popular in recent years. Atokolo et al. (2022) proposed one of the fractional-order Sterile Insect Technique (SIT) models to contain the propagation of the Zika virus infection. They used Laplace Adomian Decomposition Method (LADM) to get an infinite series solution to the model. Equally, Atokolo et al (2023) used a mathematical model involving the use of a power-law fractional derivative to develop a fractional order model that would help in the control of the spread of Lassa fever when vaccination and treatment are used. Yunus et al. (2023) have created a model based on the Caputo derivative and LADM to perform a study of preventing the spread of COVID-19 in Nigeria (fractional-order model). They found that, the recovery rate with the addition of fractional-order derivatives was better than that of the integer-order case especially with the addition of vaccination and treatment. In their study on the manifestations of helminth infection by soil, Omede et al. (2024) made use of the Caputo derivative to construct a fractional-order compartmental model. With the LADM, they were able to get infinite series solutions to converge to the exact values, and hence was more flexible than classical integer-order models. The mathematical model of prediction of Hepatitis C infection was put forward by Amos et al. (2024) based on the Adams-Bashford-Moulton method and the fractional-order mathematical model. They found that effective treatment was highly effective in reducing transmission of the disease and that the fractional-order model displayed a high level of adaptability as opposed to the classical models. James et al. (2024) have used an Adams-Bash-forth-Moulton method and implemented a fractional-order model to analyses the transmission dynamics of HIV/AIDS. They found that despite the fact that parameters like contact rates were lowered to indicate better approaches to treating the disease, the disease could be managed effectively, and this indicated the versatility and strength of the fractional-order models over conventional methods. In the works of Abah et al. (2024), the Adams-Bashforth-Moulton method was also used. They found that the fractional-order model was effective in capturing the reduction in disease transmission that was realized by the lowering rates of contact as well as the effectiveness of the treatment regime. This shows how fractional-order methods can be able to capture the complex dynamics of diseases as compared to the traditional integer-order models. The model predictive control of the co-epidemic dynamics of the HIV and COVID-19 is an ABC-fractional order derivative model Ahmed et al. (2021). The study by Smith et al. (2023) is a comprehensive review of the interaction of Hepatitis C and COVID-19 co-infections. The

authors were able to synthesize the recent research in mathematical modeling and outline the most frequently used methods and the fundamental findings as well as gaps that should be explored in further research.

The fractional-order models have different strengths because of their adaptability and the ability to track the non-local effects. In comparison to classical derivatives, fractional derivatives give a more accurate estimation of real-life results and improved flexibility. They include non-local interaction a feature that was not taken into consideration by the traditional models and they can cover memory effects, an ability that was not provided by integer-order derivatives. The reasons behind this have motivated the use of fractional differential equations by researchers as a way to address challenging issues. As an illustration, Das et al. (2020) was quoted discussing degenerate kernel fuzzy Volterra integral equations using a combination of Laplace transform and Adomian Decomposition Method by Ullah et al. (2024). This is a new tactic that has received focus on contributing to the hypothetical hypothesis of fuzzy analytical dynamic equations.

The study by Ali et al. (2017) examined whether a certain three-point boundary value problem has stable solutions. They used well-known non-linear fractional methods to analyze different kinds of stability, making a valuable contribution to the topic. This paper is aimed at achieving the following objectives: establish requirements so that the proposed fractional-order model has existence and uniqueness of solutions; use Lyapunov function to carry out a stability analysis of the endemic equilibrium point; calculate numerical solutions by making use of the fractional Adams-Bashforth-Moulton method; and perform numerical simulation in order to study the behavior of the model.

A review of literature concerning the mathematical models in Hepatitis B and the transmission dynamics showed that there are no studies that have explored the usage as well as the source of the Adams-Bashford-Moulton technique applied to the simulation and analysis of the transmission and control of Hepatitis B together with the fractional calculus.

Preliminary

In this section, we introduce the fundamental concepts and initial findings of fractional calculus. Our analysis incorporates both the right and left Caputo fractional derivatives, building on the models established by Milici et al. (2018) and Bonyah et al. (2020). We also explore the practical applications of this mathematics, demonstrating its use in solving real-world problems across diverse fields like physics, engineering, and bio-mathematics.

Definition 1: Let $f \in \Lambda^{\infty}(R)$ then the left and right Caputo fractional derivative of the function f is given by

$$CD_t^{\eta} f(t) = \left(t^0 D_t^{-(n-\eta)} \left(\frac{d}{dt}\right)^n f(t) \right)$$

$$CD_t^{\eta} f(t) = \frac{1}{\Gamma(n-\eta)} \int_0^t \left((t-\lambda)^{n-\eta-1} f^n(\lambda) \right) d\lambda \quad (1)$$

The same way

$$CD_t^{\eta} f(t) = \left(D_T^{-(n-\eta)} \left(\frac{-d}{dt}\right)^n \right) f(t)$$

$${}^{C}D_T^{\eta} f(t) = \frac{(-1)^n}{\Gamma(n-\eta)} \int_t^T (\lambda - t)^{n-\eta-1} f^n(\lambda) d\lambda$$

Definition 2: The generalized Mittag-Leffler function $E_{\gamma,\beta}(x)$ for $x \in R$ is given by

$$E_{\gamma,\beta}(x) = \sum_{n=0}^{\infty} \frac{x^n}{\Gamma(\gamma n + \psi)}, \gamma, \psi > 0 \quad (2)$$

which can also be represented as

$$E_{\gamma,\psi}(x) = x E_{\gamma,\gamma+\psi(x)} + \frac{1}{\Gamma(\psi)} \quad (3)$$

$$E_{\gamma,\psi}(x) = L\left[t^{\psi-1} E_{\gamma,\psi(\pm\omega t^{\eta})} \right] = \frac{S^{\gamma-\psi}}{S^{\gamma} \pm \omega}. \quad (4)$$

Proposition 1.1

Let $f \in \Lambda^{\infty}(R) \cap C(R)$ and $\gamma \in R, n-1 < \gamma < n$, therefore, the conditions given below holds:

1. ${}_{t_0}^{C} D_t^{\eta} I^{\eta} f(t) = f(t)$
2. ${}_{t_0}^{C} D_t^{\eta} I^{\eta} f(t) = f(t) - \sum_{k-0}^{n-k} \frac{t^k}{K!} f^k(t_0)$ (5)

Model Formulation

The process of recruiting people into the susceptible group is represented as Λ_h, hence $\beta_1, \beta_2 and \beta_3$ are the effective rates at which the susceptible people are contacting the acute infected people, chronic infected population and population on the hepatitis B treatment respectively. The progression rate between exposed human population and acute infected humans with hepatitis B α_2, the progression rate between acute infected class with hepatitis B and chronic infected class are indicated by α_6 respectively, the acute and chronic infected human population are treated at the rate of θ_2 and θ_6. γ_4 is the natural recovery rate of chronic infected human population; γ_2 is the recovery rate of humans as a result of treatment of hepatitis B. The natural death rate of human beings has been represented asμ_h . The rate of death induced by the disease on acute infected humans with hepatitis B, chronic infected humans with hepatitis B and humans under treatment of hepatitis B are represented by $\delta_2, \delta_7 and \delta_4$. The rate of susceptible humankind against hepatitis B vaccination is referred to as ω_1 and the rate of vaccine failure as ω_2 and the rate at which recovered humans become susceptible again σ_2.

Model Assumptions

i. We assume that there is an imperfect vaccine in the human population.
ii. It is our assumption that recovered human beings from hepatitis B may be attacked by the disease after recovering.
iii. We assumed that the human population recover naturally.

Model Flow Chart

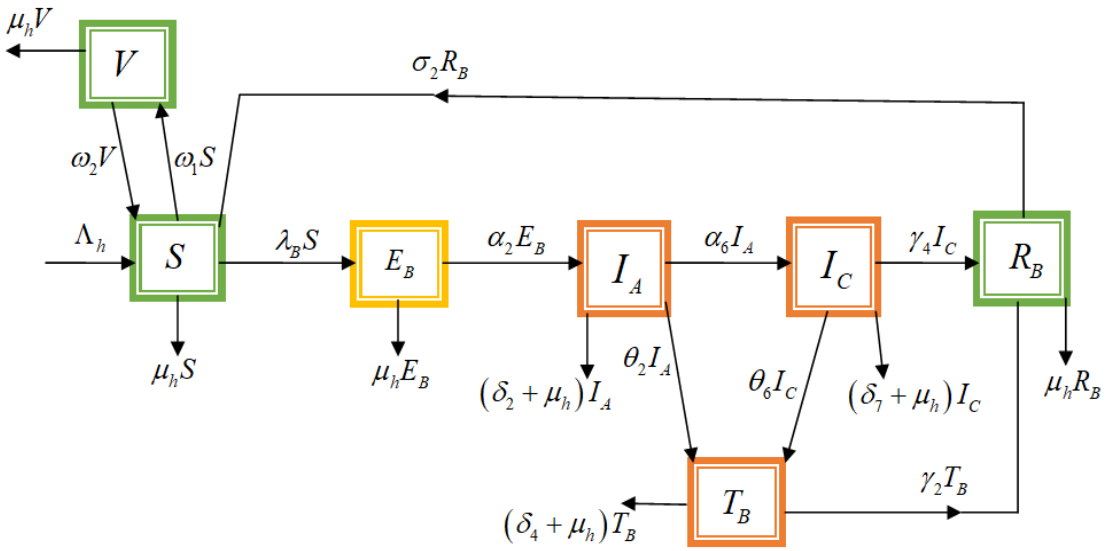

Figure 1: Hepatitis B Model Flow Chart

Model Equations

$$\frac{dS}{dt} = \boldsymbol{\Lambda}_h - \lambda_B S + \sigma_2 R_B + \omega_2 V - (\omega_1 + \mu_h)S,$$
$$\frac{dE_B}{dt} = \lambda_B S - (\alpha_2 + \mu_h)E_B,$$
$$\frac{dI_A}{dt} = \alpha_2 E_B - (\alpha_6 + \theta_2 + \delta_2 + \mu_h)I_A,$$
$$\frac{dI_C}{dt} = \alpha_6 I_A - (\theta_6 + \gamma_4 + \delta_7 + \mu_h)I_C, \quad (6)$$
$$\frac{dT_B}{dt} = \theta_2 I_A + \theta_6 I_C - (\gamma_2 + \delta_4 + \mu_h)T_B,$$
$$\frac{dV}{dt} = \omega_1 S - (\omega_2 + \mu_h)V,$$
$$\frac{dR_B}{dt} = \gamma_2 T_B + \gamma_4 I_C - (\sigma_2 + \mu_h)R_B.$$

Where $\lambda_B = \frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h}$.

Table 1: Model Variables and Parameters Descriptions

Variables	Descriptions
S	Humans who are susceptible to hepatitis B
E_B	Humans population who are Exposed to hepatitis B
I_A	Acute infected human infected with hepatitis B
I_C	Chronic infected human infected with hepatitis B
T_B	Human population on hepatitis B treatment
V	Vaccinated human population against hepatitis B
R_B	Recovered human population from hepatitis B
Parameters	**Descriptions**
Λ_h	Recruitment rate of Susceptible human population to hepatitis B
β_1	The rate of contact between the susceptible humans and the human acutely infected humans
β_2	Contact rate between the susceptible people and the chronically infected people
β_3	Contact rate between the susceptible human beings and those who are on hepatitis B treated
α_2	The levels of progression of exposed humans to hepatitis B and the hepatitis B class to acute infected class
α_6	The rate of progression of the acute infected hepatitis B class to chronic infected hepatitis B class
θ_2	Acute infected humans, rate of treatment
θ_6	The rate of chronic infected human being treatment
α_4	The natural recovery rate of the population that is chronically infected with the human race
γ_2	The recoverability of human beings on account of the treatment of hepatitis B

γ_4	Natural recovery rate hepatitis B.
μ_h	Death rate of human being which is natural
δ_2	Mortality rate of acute Infected humans with hepatitis B
δ_7	Death rate due to infection by hepatitis B of humans who had chronic infection
δ_4	The death rate of human beings on hepatitis B medication as a result of disease in humans
ω_1	Vaccinated human population.
ω_2	Waning rate of vaccine
σ_2	Rate at which recovered humans become susceptible again.

Fractional Hepatitis B Mathematical Model

In this section, the integer- order model of the hepatitis B in Eq. (5) through incorporation of Caputo fractional derivative operator. The flexibility of this adaptation is an improvement over any possible sample of the conventional model in Eq. (5), given that the fractional-order formulation has a greater output capacity to a wide array of dynamic outputs. The obtained fractional-order hepatitis B model is proposed the following way:

$$
\begin{aligned}
&CD_t^{\eta}S = \boldsymbol{\Lambda}_h - \lambda_B S + \sigma_2 R_B + \omega_2 V - (\omega_1 + \mu_h)S,\\
&CD_t^{\eta}E_B = \lambda_B S - (\alpha_2 + \mu_h)E_B,\\
&CD_t^{\eta}I_A = \alpha_2 E_B - (\alpha_6 + \theta_2 + \delta_2 + \mu_h)I_A,\\
&CD_t^{\eta}I_C = \alpha_6 I_A - (\theta_6 + \gamma_4 + \delta_7 + \mu_h)I_C,\\
&CD_t^{\eta}T_B = \theta_2 I_A + \theta_6 I_C - (\gamma_2 + \delta_4 + \mu_h)T_B,\\
&CD_t^{\eta}V = \omega_1 S - (\omega_2 + \mu_h)V,\\
&CD_t^{\eta}R_B = \gamma_2 T_B + \gamma_4 I_C - (\sigma_2 + \mu_h)R_B.
\end{aligned} \tag{6}
$$

Subject to the positive initial conditions

$$S(0) = S_0, E_B(0) = E_{B_0}, I_A(0) = I_{A0}, I_C(0) = I_{C0}, T_B(0) = T_{B0}, V(0) = V_0, R_B(0) = R_{B0}. \tag{7}$$

Positivity of Model Solution

We considered the non-negativity of the initial values

$$lim\, S\, upN_h(t) \leq \frac{\Lambda_h}{\mu_h},$$

Secondly, if $lim\, S\, upN_0(t) \leq \frac{\Lambda_h}{\mu_h}$,then our model feasible domain is given by:

$$\Omega = \left\{(S, E_B, I_A, I_C, T_B, V, R_B) \subset R_+^7 : S + E_B + I_A + I_C + T_B + V + R_B \leq \frac{\Lambda_B}{\mu_B}\right\},$$

so that

$$\Omega = \Omega_B \subset R_+^7,$$

hence, Ω is positively invariant.

In case of non-negative $(S_0, E_{B0}, I_{A0}, I_{C0}, T_{B0}\, V_0\, , R_{B0})$, the solution of model (6) will be non-negative for $t > 0$. Using (6), the first equation, we can get that:

$$
\begin{aligned}
&CD_t^{\eta}S = \boldsymbol{\Lambda}_h - \lambda_B S + \sigma_2 R_B + \omega_2 V - (\omega_1 + \mu_h)S,\\
&CD_t^{\eta}S + (\lambda_B + \omega_1 + \mu_h)S = \boldsymbol{\Lambda}_h + \sigma_2 R_B + \omega_2 V,
\end{aligned}
$$

But $\Lambda_h + \sigma_2 R_B + \omega_2 V \geq 0$then,

$$CD_t^{\eta}S + (\lambda_B + \omega_1 + \mu_h)S \geq 0 \tag{8}$$

By Laplace transform we get;

$$L\left[CD_t^{\eta}S\right] + L[(\lambda_B + \omega_1 + \mu_h)S] \geq 0$$

$$S(s) \geq \frac{S^{\eta-1}}{S^{\eta} + (\lambda_B + \omega_1 + \mu_h)}S(0),$$

The inverse of the Laplace transforms gave;

$$S(t) \geq E_{\eta,1}(-(\lambda_B + \omega_1 + \mu_h)t^{\eta})S_0, \tag{9}$$

Now that the word on the right of the Eq. We find that, in the case where (9) is positive, we can say that $(S \geq 0, E_B \geq 0, I_A \geq 0, I_C \geq 0, T_B \geq 0, V \geq 0, R_B \geq 0)$.

we are saying that are positives, and therefore, the solution will stay in R_+^7 for all $t > 0$ with positive initial situation.

Boundedness of Fractional Model Solution

The total population of individuals from our model is given by;

$$N_h(t) = S(t) + E_B(t) + I_A(t) + I_C(t) + T_B(t) + V(t) + R_B(t)..$$

So from our fractional model (6), we now obtain;

$$CD_t^{\eta}N(t) \leq \Lambda_h - \mu_h N_h(t) \tag{10}$$

Taking the Laplace transformation of (10) we now have;

$$L\left[CD_t^{\eta}N(t)\right] \leq L[\Lambda_h - \mu_h N_h(t)],,$$

$$N_h(s) \leq \frac{S^{\eta-1}}{(S^{\eta}+\mu_h)}N_h(0) + \frac{\Lambda_h}{S(S^{\eta}+\mu_h)}, \tag{11}$$

Taking the inverse Laplace transform of Eq. (11) we have:

$$N_h(t) \leq E_{\eta,1}(-\mu_h t^{\eta})N_h(0) + \Lambda_h E_{\eta,\eta+1}(-\mu_h t^{\eta}), \tag{12}$$

At$t \to \infty$,, the limit of Eq. (12) becomes

$$\underset{t\to\infty}{limSup}N_h(t) = \frac{\Lambda_h}{\mu_h}.$$

This means that, if $N_{h0} \leq \frac{\Lambda_h}{\mu_h}$ then $N_h \leq \frac{\Lambda_h}{\mu_h}$ which implies that, $N_h(t)$ is enclosed or bounded.

We now conclude that, this region $\Omega = \Omega_B$, is well posed and similarly feasible epidemiologically.

Existence and Uniqueness of our Model Solution

Let the real non-negative be H, we $Q = [0, H[]$

The set of all continuous function that is defined on M is represented by $N_{he}^0(Q)$ with norm as;

$$\|X\| = Sup\{|X(t)|, t \in Q\}. \tag{13}$$

Model (6) with initial conditions given in (8) may be taken into consideration and can be referred to as an initial value problem (IVP) as seen in (13).

$$
\begin{aligned}
&cD_t^{\eta}(t) = Z\big(t, X(t)\big), 0 < t < H < \infty,\\
&X(0) = X_0.
\end{aligned}
$$

Where

$$Y(t) = \big(S(t), E_B(t), I_A(t)\, , I_C(t), T_B(t)\, , V(t), R_B(t)\big)$$

represents the groups and Z be a continuous function defined as follows;

$$= \begin{pmatrix} \Lambda_h - \frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h} S + \sigma_2 R_B + \omega_2 V - (\omega_1 + \mu_h) S \\ \frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h} S - (\alpha_2 + \mu_h) E_B \\ \alpha_2 E_B - (\alpha_6 + \theta_2 + \delta_2 + \mu_h) I_A \\ \alpha_6 I_A - (\theta_6 + \gamma_4 + \delta_7 + \mu_h) I_C \\ \theta_2 I_A + \theta_6 I_C - (\gamma_2 + \delta_4 + \mu_h) T_B \\ \omega_1 S - (\omega_2 + \mu_h) V \\ \gamma_2 T_B + \gamma_4 I_C - (\sigma_2 + \mu_h) R_B \end{pmatrix} \quad (14)$$

Using proposition (2.1), we have that,

$$S(t) = S_0 + I_t^{\eta}\left[\Lambda_h - \frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h} S + \sigma_2 R_B + \omega_2 V - (\omega_1 + \mu_h) S\right],$$

$$E_B(t) = E_{B0} + I_t^{\eta}\left[\frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h} S - (\alpha_2 + \mu_h) E_B\right], \quad (15)$$

$$I_A(t) = I_{A0} + I_t^{\eta}[\alpha_2 E_B - (\alpha_6 + \theta_2 + \delta_2 + \mu_h) I_A],$$

$$I_C(t) = I_{C0} + I_t^{\eta}[\alpha_6 I_A - (\theta_6 + \gamma_4 + \delta_7 + \mu_h) I_C],$$

$$T_B(t) = T_{B0} + I_t^{\eta}[\theta_2 I_A + \theta_6 I_C - (\gamma_2 + \delta_4 + \mu_h) T_B],$$

$$V(t) = V_0 + I_t^{\eta}[\omega_1 S - (\omega_2 + \mu_h) V],$$

$$R(t) = R_0 + I_t^{\eta}[\gamma_2 T_B + \gamma_4 I_C - (\sigma_2 + \mu_h) R_B].$$

We have the Picard iteration of (15) as follows;

$$S(t) = S_0 + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{\eta - 1} Z_1\big(\lambda, S_{n-1}(\lambda)\big) d\lambda, \quad (16)$$

$$E_B(t) = E_{B0} + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{(\eta) - 1} Z_2\left(\lambda, E_{B(n-1)}(\lambda)\right) d\lambda,$$

$$I_A(t) = I_{A0} + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{\eta - 1} Z_3\left(\lambda, I_{(n-1)}(\lambda)\right) d\lambda,$$

$$I_C(t) = I_{C0} + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{\eta - 1} Z_4\left(\lambda, I_{(n-1)}(\lambda)\right) d\lambda,$$

$$T_B(t) = T_{B0} + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{\sigma - 1} Z_5\left(\lambda, T_{B(n-1)}(\lambda)\right) d\lambda,$$

$$V(t) = V_0 + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{\eta - 1} Z_6\left(\lambda, V_{(n-1)}(\lambda)\right) d\lambda,$$

$$R_B(t) = R_B + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{\eta - 1} Z_7\left(\lambda, R_{(n-1)}(\lambda)\right) d\lambda.$$

We now transformed the initial value problem of Eq. (13) to obtain;

$$X(t) = X(0) + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{\eta - 1} Z\big(\lambda, X(\lambda)\big) d\lambda. \quad (17)$$

Lemma 1, The Lipchitz condition described from Eq. (14) is satisfied by vector $Z\big(t, X(\lambda)\big)$ on a set $[0, H[]_+^7]$ with the Lipchitz constant given as:

$$\omega = max\begin{pmatrix} \big(\beta_1^* + \beta_2^* + \beta_3^* + \omega_1 + \mu_h\big), (\alpha_2 + \mu_h), (\alpha_6 + \theta_2 + \delta_2 + \mu_h), \\ (\theta_6 + \gamma_4 + \delta_7 + \mu_h), (\gamma_2 + \delta_4 + \mu_h), (\omega_2 + \mu_h), (\sigma_2 + \mu_h) \end{pmatrix}.$$

Proof:

$$\|Z_1(t, S) - Z_1(t, S_1)\|$$

$$= \left\| \begin{matrix} \Lambda_h - \frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h} S + \sigma_2 R_B + \omega_2 V - (\omega_1 + \mu_h) S \\ -\Lambda_h - \frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h} S + \sigma_2 R_B + \omega_2 V - (\omega_1 + \mu_h) S_1 \end{matrix} \right\|,$$

$$= \left\| -\Lambda_h - \frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h} S + \sigma_2 R_B + \omega_2 V - (\omega_1 + \mu_h) S(S - S_1) + \mu(S - S_1) \right\|,$$

$$\leq -\left(\big(\beta_1^* + \beta_2^* + \beta_3^*\big)\right) \|(S - S_1)\| + \|\mu_h (S - S_1)\|$$

$$\therefore \|Z_1(t, S) - Z_1(t, S_1)\| \leq \left(\left(\big(\beta_1^* + \beta_2^* + \beta_3^*\big) + \omega_1 + \mu\right)\right) \|(S - S_1)\| + \|\mu_h (S - S_1)\|$$

Similarly we obtained the following;

$$\|Z_2(t, E_B) - Z_2(t, E_{B1})\| \leq (\alpha_2 + \mu_h) \|(E_B - E_{B1})\|,$$

$$\|Z_3(t, I_A) - Z_3(t, I_{A1})\| \leq (\alpha_6 + \theta_2 + \delta_2 + \mu_h) \|(I_A - I_{A1})\|,$$

$$\|Z_4(t, I_C) - Z_4(t, I_{C1})\| \leq (\theta_6 + \gamma_4 + \delta_7 + \mu_h) \|(I_C - I_{C1})\|,$$

$$\|Z_5(t, T_B) - Z_5(t, T_{B1})\| \leq (\gamma_2 + \delta_4 + \mu_h) \|(T_B - T_{B1})\|,$$

$$\|Z_6(t, V) - Z_6(t, V_1 \quad)\| \leq (\omega_2 + \mu_h) \|(V - V_1)\|, \quad (18)$$

$$\|Z_7(t, R_B) - Z_7(t, R_{B1})\| \leq (\sigma_2 + \mu_h) \|(R_B - R_{B1})\|.$$

Where we obtained

$$\big\|Z_1\big(t, X_1(t)\big) - Z\big(t, X_2(t)\big)\big\| \leq \beta \|X_1 - X_2\|,$$

$$\omega = max\begin{pmatrix} \big(\beta_1^* + \beta_2^* + \beta_3^* + \omega_1 + \mu_h\big), (\alpha_2 + \mu_h), (\alpha_6 + \theta_2 + \delta_2 + \mu_h), \\ (\theta_6 + \gamma_4 + \delta_7 + \mu_h), (\gamma_2 + \delta_4 + \mu_h), (\omega_2 + \mu_h), (\sigma_2 + \mu_h) \end{pmatrix}. \quad (19)$$

Lemma 2. The first value problem (6), (7) in Eq. It exists and is unique.

$X(t) \in A_B^0(f)$.

Using Picard-Lindelof and fixed point theory, we estimate the solution of

$X(t) = S\big(X(t)\big)$,

where S is defined as the Picard operator articulated as ;

$S: A_B^0(f, R_+^7) \to A_B^0(f, R_+^7)$

Therefore

$$S\big(X(t)\big) = X(0) + \frac{1}{\Gamma(\eta)} \int_0^t (t - \lambda)^{\eta - 1} Z_1\big(\lambda, X(\lambda)\big) d\lambda, \quad (20)$$

which becomes :

$$\big\|S\big(X_1(t)\big) - S\big(X_2(t)\big)\big\|$$

$$= \frac{1}{\Gamma(\eta)}\left[\int_0^t (t-\lambda)^{\eta-1} Z\big(\lambda, X_1(\lambda)\big) - Z\big(\lambda, X_2(\lambda)\big) d\lambda\right],$$
$$\leq \frac{1}{\Gamma(\eta)}\left[\int_0^t (t-\lambda)^{\eta-1} Z\big(\lambda, X_1(\lambda)\big) - Z\big(\lambda, X_2(\lambda)\big) d\lambda\right],$$
$$\leq \frac{\omega}{\Gamma(\eta)}\left[\int_0^t (t-\lambda)^{\eta-1} \|X_1 - X_2\| d\lambda\right],$$
$$\big\|S\big(X_1(t) - SX_2(t)\big)\big\| \leq \frac{\omega}{\Gamma(\eta+1)S}. \quad (21)$$

When, $\frac{\omega}{\Gamma(\eta+1)} S \leq 1$. then the Picard operator gives a negation, so Eq. (6), (7) solution is unique.

The Basic Reproduction Number (R_0) and Model Equilibrium Points:

The disease-free equilibrium points of the model (5) is expressed as:

$$(S^0, E_B^0, I_A^0, I_C^0, T_B^0, V, R_B^0) = \left(\frac{\Lambda_h(\omega_2+\mu_h)}{\mu_h(\omega_2+\omega_1+\mu_h)}, 0,0,0,0, \frac{\omega_1\Lambda_h}{\mu_h(\omega_2+\omega_1+\mu_h)}, 0\right) \quad (22)$$

Basic Reproduction number:

In infectious disease modeling, the most critical number is the Basic Reproduction Number denoted by R_0^H, In simple terms, it measures the disease's potential to spread by calculating how many people one infected person will likely pass the illness to in a fully vulnerable population.

In computing the basic reproduction number, we apply the next generation method.

$$R_0^B = \frac{(\omega_2+\mu_h)\alpha_2(A_3A_4\beta_1 + A_3\beta_3\theta_2 + A_4\alpha_6\beta_2 + \alpha_6\beta_3\theta_6)}{A_1A_2A_3A_4(\omega_2+\omega_1+\mu_h)}. \quad (23)$$

Where $A_1 = (\alpha_2 + \mu_h)$, $A_2 = (\alpha_6 + \theta_2 + \delta_2 + \mu_h)$, $A_3 = (\theta_6 + \gamma_4 + \delta_7 + \mu_h)$, $A_4 = (\gamma_2 + \delta_4 + \mu_h)$.

Endemic Equilibrium Point

We also studied what happens if Hepatitis B becomes a permanent, ongoing presence in the community. In this scenario, the infection never completely disappears, but instead settles into a stable, long-term pattern where the disease continues to circulate at a constant level.

$\big(S^* \neq 0, E_B^* \neq 0, I_A^* \neq 0, I_C^* \neq 0, T_B^* \neq 0, V^* \neq 0, R_B^* \neq 0\big)$.

To understand what happens when Hepatitis B becomes a long-term presence, we reworked the model's equations to focus on how the infection spreads. Starting with our discrete Hepatitis B model (Equation 6), we found that the steady state where the disease persists at a constant level is defined by the following values:

$$S^* = \frac{\Lambda_h A_6 A_2 A_3 A_4 A_5 A_7}{\big((A_2A_3A_7(\lambda_B + A_1)A_5 - \gamma_2\sigma_2\theta_2\lambda_B\alpha_2)A_4 - \sigma_2\lambda_B\alpha_2\alpha_6(A_5\gamma_4 + \gamma_2\theta_6)\big)A_6 - A_2A_3A_4A_5A_7\omega_1\omega_2},$$
$$E_B^* = \frac{\Lambda_h A_6 A_3 A_4 A_5 A_7 \lambda_B}{\big(((A_2A_3A_7A_5 - \alpha_2\gamma_2\sigma_2\theta_2)A_4 - \sigma_2\alpha_2\alpha_6(A_5\gamma_4+\gamma_2\theta_6))\lambda_B + A_1A_2A_3A_4A_5A_7\big)A_6 - A_2A_3A_4A_5A_7\omega_1\omega_2},$$
$$I_A^* = \frac{\Lambda_h A_6 A_4 A_5 A_7 \lambda_B \alpha_2}{\Big(\big(\big(A_2A_3A_7A_5 - \alpha_2\gamma_2\sigma_2\theta_2\big)A_4 - \sigma_2\alpha_2\alpha_6\big(A_5\gamma_4 + \gamma_2\theta_6\big)\big)\lambda_B + A_1A_2A_3A_4A_5A_7\Big)A_6 - A_2A_3A_4A_5A_7\omega_1\omega_2},$$
$$I_C^* = \frac{\Lambda_h A_6 A_5 A_7 \lambda_B \alpha_2 \alpha_6}{\big(((A_2A_3A_4A_7 - \alpha_2\alpha_6\gamma_4\sigma_2)A_5 - \alpha_2\gamma_2\sigma_2(A_4\theta_2 + \alpha_6\theta_6))\lambda_B + A_1A_2A_3A_4A_5A_7\big)A_6 - A_2A_3A_4A_5A_7\omega_1\omega_2},$$
$$T_B^* = \frac{\Lambda_h A_6 A_7 \lambda_B \alpha_2 \big(A_4\theta_2 + \alpha_6\theta_6\big)}{\Big(\big(\big(A_2A_3A_7A_5 - \alpha_2\gamma_2\sigma_2\theta_2\big)A_4 - \sigma_2\alpha_2\alpha_6\big(A_5\gamma_4 + \gamma_2\theta_6\big)\big)\lambda_B + A_1A_2A_3A_4A_5A_7\Big)A_6 - A_2A_3A_4A_5A_7\omega_1\omega_2},$$
$$V^* = \frac{\Lambda_h A_2 A_3 A_4 A_5 A_7 \omega_1}{\big(A_7A_2\big((\lambda_B + A_1)A_6 - \omega_1\omega_2\big)A_3A_5 - \gamma_2\sigma_2\theta_2\lambda_B A_6\alpha_2\big)A_4 - \sigma_2\lambda_B A_6\alpha_2\alpha_6(A_5\gamma_4 + \gamma_2\theta_6)},$$
$$R_B^* = \frac{\Lambda_h\lambda_B\big(A_4\gamma_2\theta_2 + \alpha_6(A_5\gamma_4+\gamma_2\theta_6)\big)\alpha_2 A_6}{\big(((A_2A_3A_5A_7 - \alpha_2\gamma_2\sigma_2\theta_2)A_4 - \sigma_2\alpha_2\alpha_6(A_5\gamma_4+\gamma_2\theta_6))\lambda_B + A_1A_2A_3A_4A_5A_7\big)A_6 - A_2A_3A_4A_5A_7\omega_1\omega_2}. \quad (24)$$

Substituting into the force of infection

$$\lambda_B = \frac{(\beta_1 I_A + \beta_2 I_C + \beta_3 T_B)}{N_h},$$

We obtained;

$$Q_1\lambda_B + Q_2 = 0. \quad (25)$$

Where

$$Q_1 = \Lambda_h \begin{pmatrix} A_3A_4A_5A_6A_7 + A_4A_5A_6A_7\alpha_2 + A_4A_6A_7\alpha_2\theta_2 \\ +A_4A_6\alpha_2\gamma_2\theta_2 + A_5A_6A_7\alpha_2\alpha_6 + A_5A_6\alpha_2\alpha_6\gamma_4 \\ +A_6A_7\alpha_2\alpha_6\theta_6 + A_6\alpha_2\alpha_6\gamma_2\theta_6 \end{pmatrix},$$

$$Q_2 = \Lambda_h\left(A_6A_2A_3A_4A_5A_7 + (A_2A_3A_4A_5A_7\omega_1)(1 - R_0^B)\right).$$

This implies that the model has a stable endemic equilibrium point.

Global Stability of Hepatitis B Disease

Theorem 1: Prove that the system (5) is globally asymptotically stable at Disease free equilibrium, moreover, at $R_0 < 1$.

Proof:

We construct the Lyapunov function to prove the results,

$$L = u_1(S - S_0) + u_2(E_B - E_{B0}) + u_3(I_A - I_{A0}) + u_4(I_C - I_{C0}) + u_5(T_B - T_{B0}) + u_6(V - V_0) + u_7(R_B - R_{B0}). \quad (26)$$

Where $u_1, u_2, u_3, u_4, u_5, u_6, u_7$ are positive constants.

Taking the derivative of a Lyapunov function, we obtained;

$$L' = \Lambda_h u_1 + \omega_2 S E_B(u_2 - u_1) + (1 - \omega_1)(u_3 - u_1) + \alpha_2(u_3 - u_2) + \omega_1(u_4 - u_2)$$
$$+\theta_2(u_4 - u_3) + \omega_1(u_5 - u_4) + \alpha_2(u_6 - u_4) + \alpha_1(u_7 - u_4) - \mu_h u_1 S - \mu_h u_2 E_B$$
$$-\mu_h u_3 I_A - \mu_h u_4 I_C - \mu_h u_5 V - \mu_h u_6 T_B - \mu_h u_7 R_B.$$

Choosing the positive constants $u_1 = u_2 = u_3 = u_4 = u_5 = u_6 = u_7 = 1$

And $N_h > \frac{\Lambda_h}{\mu_h}$ then, we obtained;

$$L' = \Lambda_h - \mu_h N_h$$
$$L' = -[\mu_h N_h - \Lambda_h] < 0. \quad (27)$$

Hence the system (5) is globally asymptotically stable at the Disease-free equilibrium and at $R_0^B < 1$.

Fractional Order Model Numerical Results

The fractional-order Hepatitis B model was numerically solved using the generalized fractional Adams–Bashforth–Moulton method as described by Bonyah et al.(2020). Table 1 presents the parameter values used in the model, while Table 2 displays the different fractional-order values applied and simulated in the analysis.

Implementation of Fractional Adams–Bashforth–Moulton Method

The technique described by Baskonus. and Bulut (2015), and Ren et al.(2012) was employed in the present study. The approximate solution for the fractional-order Hepatitis B model in Equation (6) was developed using the fractional Adams–Bashforth–Moulton method. The fractional form of Equation (6) is presented as follows:

$${}^cD_t^\eta H(t) = Q(t, q(t)), 0 < t < \omega, \quad (28)$$

$$H^{(n)}(0) = H_0^{(n)}, n = 1,0,\ldots, q, q = [\alpha].$$

The $H = \left(S^*, E_B^*, I_A^*, I_C^*, T_B, V^*, R_B^*\right) \in R_+^7$ and $V(t, q(t))$ is a continuous function of a real value. Equation (27) can hence be expressed in terms of the idea of fractional integral as follows:

$$H(t) = \sum_{n=0}^{m-1} H_0^{(n)} \frac{t^n}{n!} + \frac{1}{\Gamma(\eta)} \int_0^t (t - y)^{\eta - 1} R\left(k, m(k)\right) dk \quad (29)$$

Using the method described in [43], we let the step size $g = \frac{\omega}{N}, N \in \mathrm{N}$ with a grid that is uniform on $[0, \omega]$. Where $t_c = cr, c = 0,1,1,\ldots N$. Thus, and fractional order model of Hepatitis B model could be well approximated as (6) creates:

$$S_{k+1}(t) = S_0 + \frac{g^\eta}{\Gamma(\eta+2)}\left\{\Lambda_h - \left(\beta_1 I_A^n + \beta_2 I_C^n + \beta_3 T_B^n\right)\frac{S^n}{N_h^n} + \sigma_2 R_B^n + \omega_2 V^n - \left(\omega_1 + \mu_h\right)S^n\right\} +$$

$$\frac{g^\eta}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\left\{\Lambda_h - \left(\beta_1 I_{Ay} + \beta_2 I_{Cy} + \beta_3 T_{By}\right)\frac{S_y}{N_{hy}} + \sigma_2 R_{By} + \omega_2 V_y - \left(\omega_1 + \mu_h\right)S_y\right\},$$

$$E_{B(k+1)}(t) = E_{B0} + \frac{g^\eta}{\Gamma(\eta+2)}\left\{(\beta_1 I_A^n + \beta_2 I_C^n + \beta_3 T_B^n)\frac{S^n}{N_h^n} - (\alpha_2 + \mu_h)E_B^n\right\} + \frac{g^\eta}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\left\{\left(\beta_1 I_{Ay} + \beta_2 I_{Cy} + \beta_3 T_{By}\right)\frac{S_y}{N_{hy}} - (\alpha_2 + \mu_h)E_{By}\right\}, \quad (30)$$

$$I_{A(k+1)}(t) = I_0 + \frac{g^\eta}{\Gamma(\eta+2)}\alpha_2 E_B^n - (\alpha_6 + \theta_2 + \delta_2 + \mu_h)I_A^n + \frac{g^\eta}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\left\{\alpha_2 E_{By} - (\alpha_6 + \theta_2 + \delta_2 + \mu_h)I_{Ay}\right\},$$

$$I_{C(k+1)}(t) = I_0 + \frac{g^\eta}{\Gamma(\eta+2)}\{\alpha_6 I_A^n - (\theta_6 + \gamma_4 + \delta_7 + \mu_h)I_C^n\} + \frac{g^\eta}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\left\{\alpha_6 I_{Ay} - (\theta_6 + \gamma_4 + \delta_7 + \mu_h)I_{Cy}\right\},$$

$$T_{B(k+1)}(t) = T_{B0} + \frac{g^{\eta}}{\Gamma(\eta+2)}\{\theta_2 I_A^n + \theta_6 I_C^n - (\gamma_2 + \delta_4 + \mu_h)T_B^n\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\{\theta_2 I_{Ay} + \theta_6 I_{Cy} - (\gamma_2 + \delta_4 + \mu_h)T_{By}\},$$

$$V_{k+1}(t) = V_0 + \frac{g^{\eta}}{\Gamma(\eta+2)}\{\omega_1 S^n - (\omega_2 + \mu_h)V^n\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\{\omega_1 S_y - (\omega_2 + \mu_h)V_y\},$$

$$R_{B(k+1)}(t) = I_0 + \frac{g^{\eta}}{\Gamma(\sigma+2)}\{\gamma_2 T_B^n + \gamma_4 I_C^n - (\sigma_2 + \mu_h)R_B^n\} + \frac{g^{\eta}}{\Gamma(\eta+2)}\sum_{y=0}^{k} dy, k+1\{\gamma_2 T_{By} + \gamma_4 I_{Cy} - (\sigma_2 + \mu_h)R_{By}\}.$$

Where

$$S_{k+1}^n(t) = S_0 + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\left\{\Lambda_h - \left(\beta_1 I_{Ay} + \beta_2 I_{Cy} + \beta_3 T_{By}\right)\frac{S_y}{N_{hy}} + \sigma_2 R_{By} + \omega_2 V_y - (\omega_1 + \mu_h)S_y\right\},$$

$$E_{B(k+1)}^n(t) = E_{B0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\left\{\left(\beta_1 I_{Ay} + \beta_2 I_{Cy} + \beta_3 T_{By}\right)\frac{S_y}{N_{hy}} - (\alpha_2 + \mu_h)E_{By}\right\}, \quad (31)$$

$$I_{A(k+1)}^n(t) = I_0 + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\alpha_2 E_{By} - (\alpha_6 + \theta_2 + \delta_2 + \mu_h)I_{Ay}\},$$

$$I_{C(k+1)}^n(t) = I_0 + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\alpha_6 I_{Ay} - (\theta_6 + \gamma_4 + \delta_7 + \mu_h)I_{Cy}\},$$

$$T_{B(k+1)}^n(t) = T_{B0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\theta_2 I_{Ay} + \theta_6 I_{Cy} - (\gamma_2 + \delta_4 + \mu_h)T_{By}\},$$

$$V_{k+1}^n(t) = V_0 + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\omega_1 S_y - (\omega_2 + \mu_h)V_y\},$$

$$R_{B(k+1)}^n(t) = R_{B0} + \frac{1}{\Gamma(\eta)}\sum_{y=0}^{k} f_{y,k+1}\{\gamma_2 T_{By} + \gamma_4 I_{Cy} - (\sigma_2 + \mu_h)R_{By}\}.$$

From (29) and (30) obtained;

$dy_{,K+1} = K^{\eta+1} - (k-\eta)(k+\eta)^{\eta}, y = 0.$

$(k-y+2)^{\eta+1} + (k-\eta)^{\eta+1} - 2(k-y+1)^{\eta+1}, 1 \le y \le k$

and $f_{y,k+1} = \frac{g^{\eta}}{\eta}[(k-y+1)^{\eta}(k-y)^{\eta}], 0 \le y \le k.$

Importance of using the Fractional Adam-Bashforth-Moulton Method in Obtaining the Numerical Solutions of the Model

i. The fractional Adams-Bashforth-Moulton scheme strictly just needs one extra function evaluation per step and has high-order of accuracy.
ii. This approach has the advantage of automatic error control, and can often be applied to ODE solvers to accomplish integration.
iii. This means that it has wide applicability in other fields such as engineering, chemistry and medicine and as such, it is a useful method in numerically solving partial and fractional-order differential equations.

Table 2: Parameter Values used for Numerical Simulation

Parameters	Values	Sources
Λ_h	16540000	CDC (2023)
β_1	1×10^{-9}	Boukanjime and Fatini (2019)
β_2	0.8328	Assumed
β_3	0.8214	Assumed
α_2	0.058426	Fitted
α_4	0.143597	Fitted
γ_2	0.278267	Fitted
γ_4	0.5	Fitted
θ_2	0.032	Boukanjime and Fatini (2019)
σ_2	0.05	Fitted
μ_h	0.07	CDC (2023)
δ_2	0.3	Assumed
δ_7	0.0200	Assumed
δ_4	0.02	Assumed
ω_1	0.5521	Assumed
ω_2	0.1	Granas and Dugundji (2003)

Table 3: Number of Cases of Potential Hepatitis B Virus Infections K and the Proportion of K in the Compartment of Diseased Individuals from China

YEAR	CASES
2004	368,566
2005	432,541
2006	454,624
2007	462,366
2008	465,379
2009	466,907
2010	468,028
2011	469,102
2012	470,246
2013	471,495
2014	472,866
2015	474,361
2016	475,983
2017	477,732
2018	479,609
2019	481,614
2020	483,749
2021	486,013

Numerical Simulation

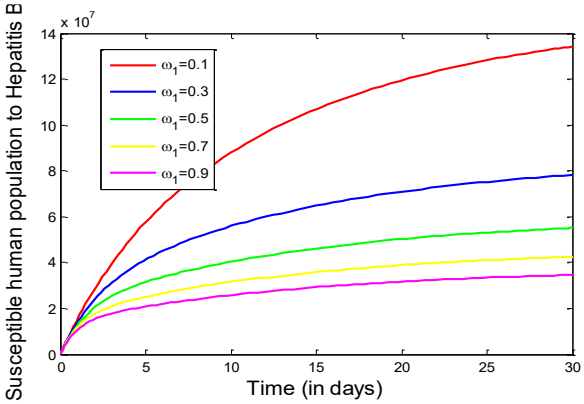

Figure 3a: Simulation of susceptible population to hepatitis B

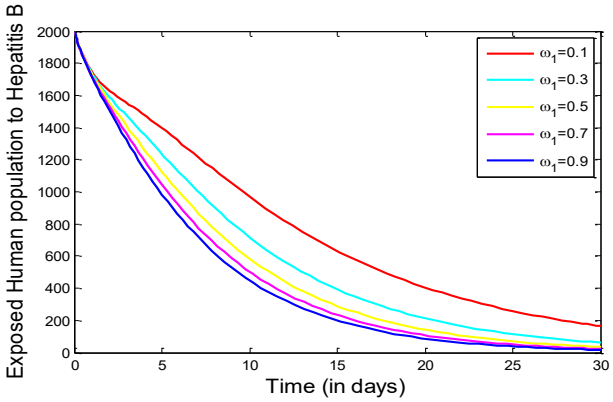

Figure 3b: Simulation of Exposed population to hepatitis B

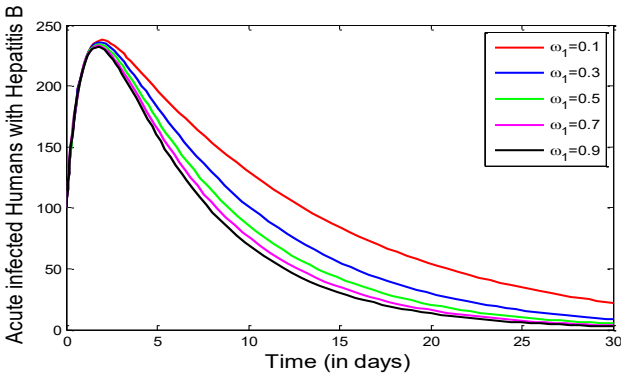

Figure 3c: Simulation of acute infected population to hepatitis B

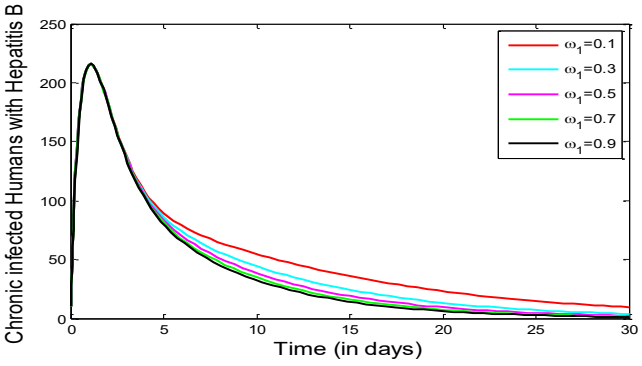

Figure 3d: Simulation of acute infected population to hepatitis B

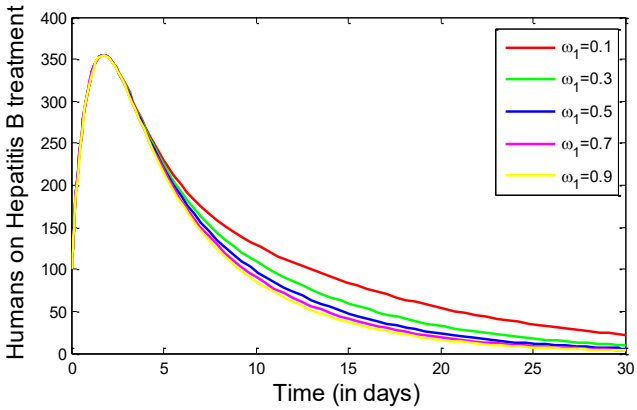

Figure 3e: Simulation of population hepatitis B treatment

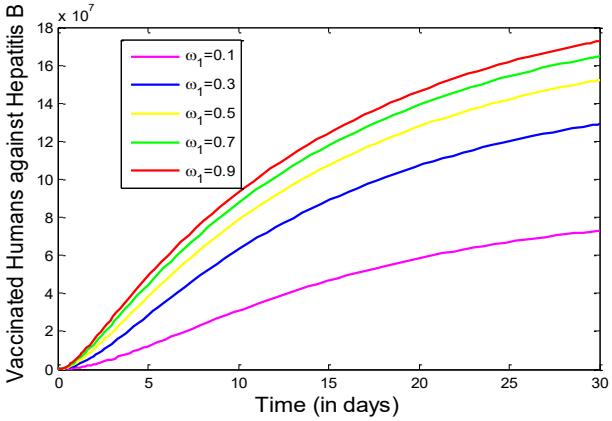

Figure 3f: Simulation of vaccinated humans against hepatitis B

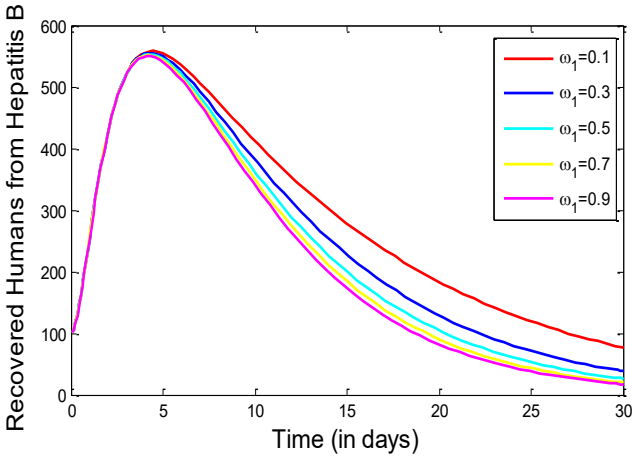

Figure 3g: Simulation of Recovered humans from hepatitis B

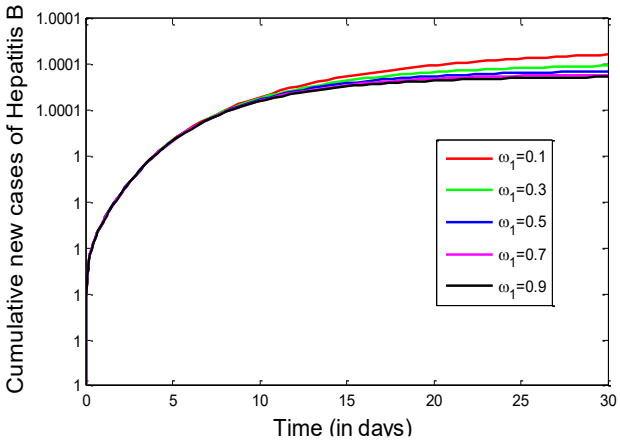

Figure 3h: Simulation of cumulative new cases of hepatitis B

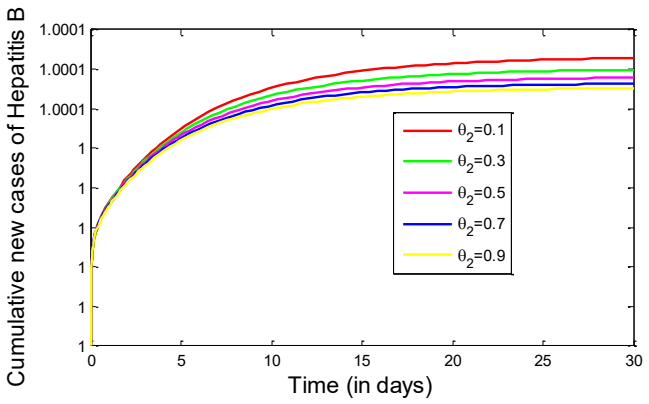

Figure 3i: Simulation of cumulative new cases of hepatitis B

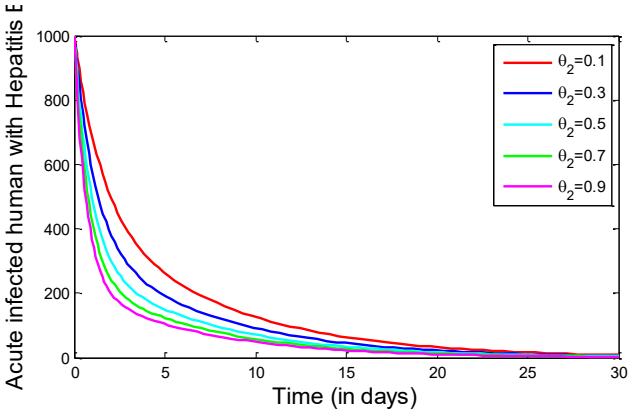

Figure 3j: Simulation of acute infected with hepatitis B

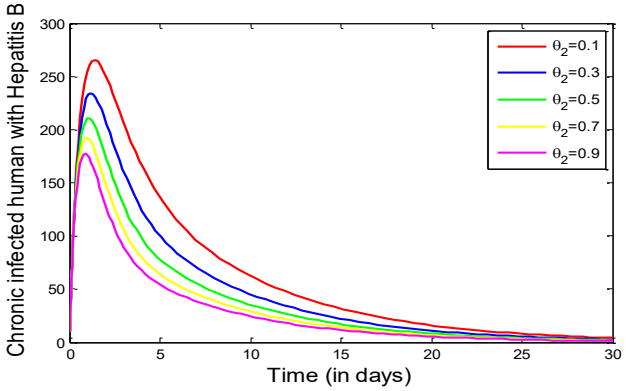

Figure 3k: Simulation of chronic infected with hepatitis B

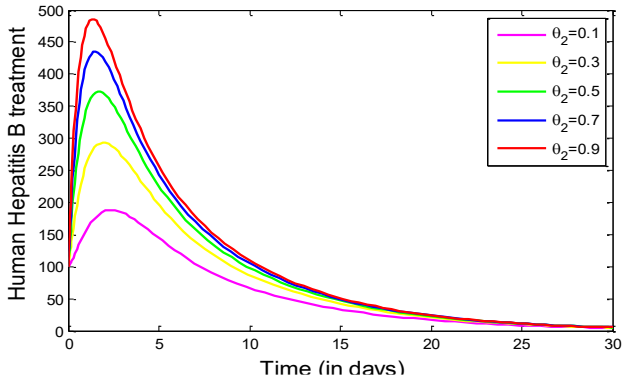

Figure 3l: Simulation of humans on treatment hepatitis B

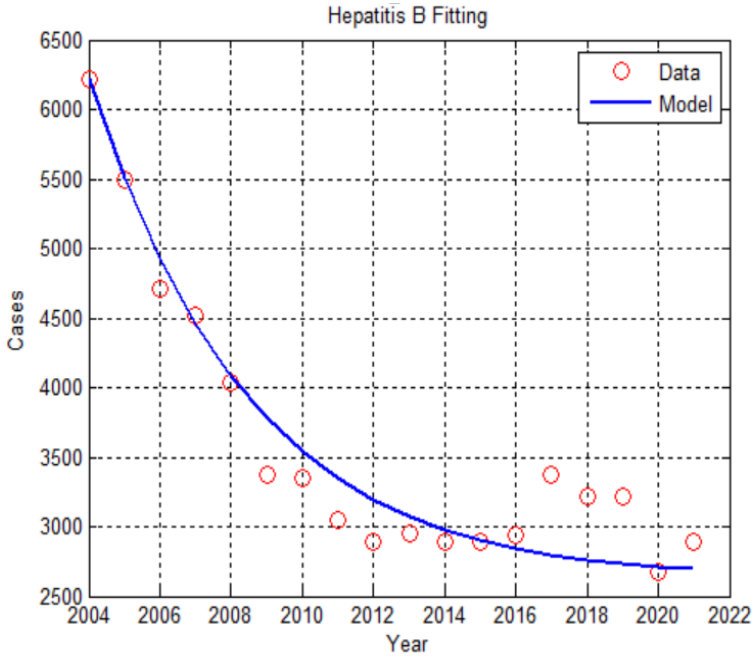

Figure 4: Hepatitis B Data Fitting

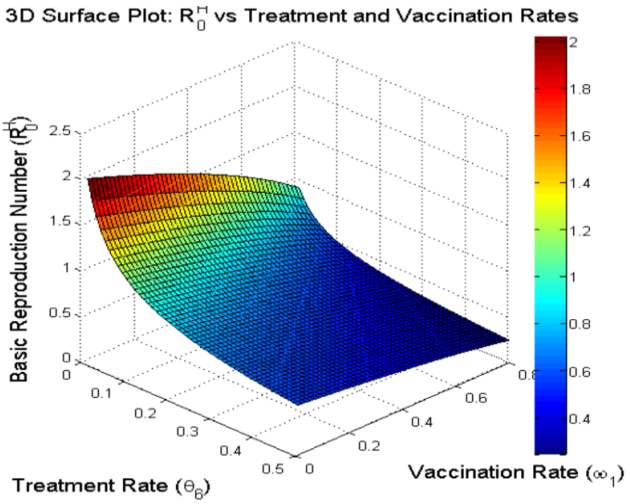

Figure 5a: Contour plot showing the impact of θ_6 and ω_1 on R_0^H

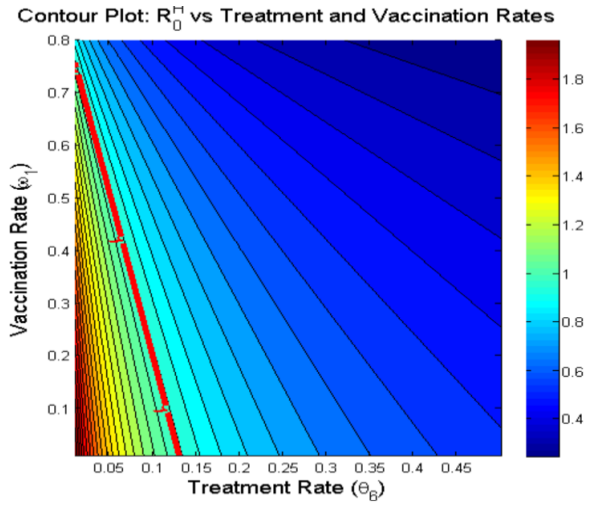

Figure 5b: Contour plot showing the impact of θ_6 and ω_1 on R_0^H

(3a) shows simulation of the impact of vaccination rate (ω_1)among people with Hepatitis B infection on the susceptible human population. It can be observed that higher vaccination rate (ω_1)leads to the reduction of susceptible human to the two diseases. (3b) shows simulation of the impact of vaccination rate (ω_1)among people with Hepatitis B infection on the exposed human population. It can be observed that higher vaccination rate (ω_1)leads to the reduction of exposed human to the two diseases. (3c) reveals the simulation of influence of vaccination rate (ω_1) of humans that are vulnerable to Hepatitis B on acute population people that are affected by Hepatitis B. This demonstrates that the increasing of vaccination rate (ω_1) contributed towards the reduction of the number of acute infected human of Hepatitis B. (3d) reveals the simulation of influence of vaccination rate (ω_1) of humans that are vulnerable to Hepatitis B on chronic infected population with Hepatitis B. This demonstrates that the increasing of vaccination rate (ω_1) contributed towards the reduction of the number of chronic infected human of Hepatitis B. (3e) shows the simulation of the impact of the vaccination rate (ω_1)of infected individuals with the Hepatitis B on the human population in regard to the Hepatitis B treatment. It has been observed that the more the vaccination rate (ω_1)is enhanced, the lower the number of human beings of Hepatitis B disease who are on treatment as depicted by (3f). the effect of the vaccination rate (ω_1)of individuals susceptible to Hepatitis B on vaccinated human population against Hepatitis B is simulated. This demonstrates that the more the vaccination rate(ω_1), the more the humans that are vaccinated against the Hepatitis B disease. (3g) illustrates the model of the impact of vaccination rate (ω_1)that is placed on individuals susceptible to Hepatitis B on Recovered human population that is infected by hepatitis B. This indicates that the higher the level of vaccination activities (ω_1)the lesser the human population is found to be recovered from Hepatitis B disease. (3h). displays the simulation of the influence of the rate at which humans are vaccinated against Hepatitis B (ω_1) on the total new cases of Hepatitis B. This indicates that the high level of vaccination (ω_1) contributes to the rise of the new cases of the Hepatitis B disease. In (3i)., the influence of the rate of treatment (θ_2) of Hepatitis B infected people on the total new cases of Hepatitis B is simulated. This indicates that it is important to raise the rate of treatment (θ_2) which will promote the decline of the incident cases of Hepatitis B disease. (3j). demonstrates how the rate of the spread of Hepatitis B (θ_2) affects the treatability of acute infected humans with Hepatitis B. This indicates that higher rate of treatment (θ_2)would contribute to the reduction in the acute population infected with Hepatitis B by human beings.

(3k) describes the simulation of effect of the treatment rate (θ_2) of those infected with Hepatitis B on chronically infected human with Hepatitis B. This indicates that the rise in treatment rate (θ_2) will result in the reduction in the number of people who have been chronically infected with Hepatitis B. (3l) The effect of the treatment rate (θ_2)of people infected with Hepatitis B on the people on the Hepatitis B treatment can be seen in the figure. This indicates that rise in treatment (θ_2) gives increases the human population on Hepatitis B treatment. (4a) shows the contour plot of $\theta_6 and \omega_1$ onR_0^H . In the graph under discussion, the numerical data curve starts at the value of 0.6 which is the maximum value of the data to be and that the correlation between the variations of $\theta_6 and \omega_1$in the measurement of the transmission rate would be less than one (1). Higher values of $\theta_6 and \omega_1$indicate that the outbreak of Hepatitis B among the population is decreasing. (4b) indicated that when the values ofR_0^H reach a minimum of less than one (1), there is an indication of reduction of the basic reproduction number. $\theta_6 and \omega_1$should be reduced to ensure that the effects of Hepatitis B on the population are reduced. The lack of appropriate measures undertaken including will enhance the current prevalence of Hepatitis B.

CONCLUSION

In this research, we set out to better understand the spread of Hepatitis B using a more nuanced type of mathematics known as fractional calculus. Think of it as an upgrade from a simple on/off switch to a dimmer switch it allows for more gradual and realistic transitions, which is crucial for modeling complex processes like disease transmission. By building a fractional-order model, we were able to simulate how Hepatitis B progresses through a community and how key interventions, like vaccination and treatment, can change its course. Our simulations revealed a clear and hopeful finding: when we increase vaccination efforts among healthy individuals and improve treatment access for those who are infected, the overall burden of the disease drops significantly. The real power of this approach is its ability to capture the "memory" of biological systems meaning past conditions can influence future outcomes in a way traditional models often miss. This makes our model not just a theoretical exercise, but a more flexible and realistic tool that could one day help guide public health strategies. Of course, our study is a starting point, not a final answer. To keep things manageable, we made some simplifying assumptions for instance, we modeled the population as a single, uniform group, without accounting for geographical differences or the random chance events that affect real-world outbreaks. We also used real case data. To build on this work, the next steps are exciting. Future researchers could: Add a sense of place by incorporating geography to see how the disease moves across different regions**,** By tackling these challenges, we can transform this promising

theoretical framework into a powerful, practical tool for the ongoing battle against Hepatitis B.

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