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**MODELING AND ANALYSIS OF HEPATITIS B AND DIPHTHERIA
CO-INFECTION VIA FRACTAL-FRACTIONAL OPERATORS WITH
MITTAG-LEFFLER KERNEL**

M.SHARMILA * AND S.INDRAKALA

ABSTRACT. Combination infections of diphtheria and hepatitis B provide increasing global public health issues because of their intricate transmission patterns. In order to incorporate memory effects, non-local interactions, and fractal aspects of disease propagation, a unique fractal-fractional model with a Mittag-Leffler kernel is constructed in this paper. To represent actual co-infection scenarios, the model integrates important epidemiological characteristics such as transmission, development, treatment, recovery, immunization, and re-susceptibility rates. Variations in fractional order and fractal dimension have a substantial impact on disease dynamics, resulting in protracted outbreaks and delayed peaks, according to numerical models. The important impact of dual-pathogen interactions is shown by the results, which show magnified co-infection effects with higher infection and treatment levels under increased transmission.

1. Introduction

Hepatitis B is a potentially fatal liver infection that affects billions of people globally, with millions of chronic cases and a high death rate from liver complications. It continues to spread despite successful vaccinations due to reasons like declining immunity, various modes of transmission, and population movement. To comprehend HBV transmission and assess control measures, such as vaccination and treatment plans, numerous mathematical modeling studies have been carried out. Similar to this, diphtheria is still a dangerous and extremely contagious illness, and recent outbreaks have been connected to decreased immunity and dropping vaccination rates [1, 2, 3, 4, 5, 6, 7, 8, 9, 10].

Ongoing public health issues are highlighted by its serious repercussions and recurrence in some areas. Inspired by these worries, this study creates a thorough modeling framework that includes both asymptomatic and symptomatic infections as well as important control measures. In order to effectively manage the spread of these infectious diseases and reduce transmission, the work highlights the vital role that vaccination, awareness campaigns, and prompt medical intervention play [11, 12, 13, 14, 15, 16, 17, 18, 19].

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* Corresponding Author.

2. Mathematical Model Formulation and Assumptions

The total population of human at time t , denoted by $N_h(t)$, is divided into fifteen mutually exclusive compartments of susceptible individuals $S_h(t)$, exposed individuals to diphtheria only $E_D(t)$, individuals infected with diphtheria only $I_D(t)$, individuals on treatment with diphtheria only $T_D(t)$, recovered individuals from diphtheria only $R_D(t)$, vaccinated population against both diphtheria and hepatitis B $V(t)$, individuals exposed to both diphtheria and hepatitis B $E_{DB}(t)$, individual co-infected with both diphtheria and hepatitis B $I_{DB}(t)$, individual on treatment of both diphtheria and hepatitis B $T_{DB}(t)$, recovered individual from both diphtheria and hepatitis B $R_{DB}(t)$, exposed individuals to hepatitis B only $E_B(t)$, individuals infected with acute hepatitis B only $I_A(t)$, individuals infected with chronic hepatitis B only $I_C(t)$, individuals on treatment of hepatitis B $T_B(t)$, recovered individuals from hepatitis B $R_B(t)$, and bacteria population B . So that,

$$\begin{aligned} N_h(t) = & S_h(t) + E_D(t) + I_D(t) + T_D(t) + R_D(t) + V(t) + E_{DB}(t) \\ & + I_{DB}(t) + T_{DB}(t) + R_{DB}(t) + E_B(t) + I_B(t) + T_B(t) + R_B(t) + B. \end{aligned}$$

The susceptible human populations are recruited at the rate of Λ_h , the susceptible bacteria populations are recruited at the rate of Λ_B . Susceptible individuals are vaccinated against Diphtheria and Hepatitis B at the rate ω_1 and after some times the vaccine wane at the rate of ω_2 . Contact rate between the susceptible humans and infected human population with diphtheria, Contact rate between the susceptible humans and Humans on diphtheria only treatment, Contact rate between the susceptible humans and Bacteria population, Contact rate between the susceptible humans and infected human population with both diphtheria and Hepatitis B diseases, Contact rate between the susceptible humans and human population treatment of diphtheria and Hepatitis B diseases, Contact rate between the susceptible humans and infected human population with Hepatitis B, Contact rate between the susceptible humans and Humans on Hepatitis B only treatment are $\varphi_1, \varphi_2, \varphi_3, \varphi_4, \varphi_5, \beta_1, \beta_2$.

Exposed individuals to diphtheria only progresses to infected human population with diphtheria only at the rate of α_1 , exposed individuals to Hepatitis B only progresses to infected human population with Hepatitis B only at the rate of α_2 , exposed individuals to diphtheria only progresses to infected human population with diphtheria only at the rate of α_3 . Exposed individuals to hepatitis B only progresses to infected human population with both diseases at the rate of α_4 , exposed individuals to both hepatitis B and diphtheria progresses to infected human population with both diseases at the rate of α_5 , infected human population with diphtheria only are treated at the rate of θ_1 , infected human population with hepatitis B only are treated at the rate of θ_2 , infected human population with both diphtheria and hepatitis B are treated at the rate of θ_5 , infected human population with both diphtheria and hepatitis B receive diphtheria only at the rate of θ_4 , infected human population with both diphtheria and hepatitis B receive hepatitis B only at the rate of θ_5 , Human population on diphtheria only treatment recovered at the rate γ_1 , acute infected humans with hepatitis B only are treated at the rate θ_2 , exposed human population to hepatitis B only progresses to acute infected human population with hepatitis B only at the rate α_2 , acute hepatitis B only infected human population progresses to chronic infected human population with hepatitis B only at the rate α_6 , chronic hepatitis B infected humans are treated at the rate of θ_6 , Human population on

diphtheria only treatment recovered at the rate γ_2 , Human population on both diphtheria and hepatitis B treatment recovered at the rate γ_3 , Human populations die naturally at the rate of μ_h , Bacteria populations die naturally at the rate of μ_B , Disease induced death rate of diphtheria only infected humans, Disease induced death rate of hepatitis B only infected humans, Disease induced death rate of humans on diphtheria only treatment, Disease induced death rate of humans on hepatitis B only treatment, Disease induced death rate of humans infected with both diphtheria and hepatitis B, Disease induced death rate of humans on both diphtheria and hepatitis B treatment, are $\delta_1, \delta_2, \delta_3, \delta_4, \delta_5$, and δ_6 respectively. The recovered human populations from diphtheria only become susceptible again at the rate of σ_1 while the recovered human population from hepatitis B only becomes susceptible again at the rate of σ_2 .

I. We assume that recovered human population from both diseases can become susceptible again [25].

II. We assume an imperfect vaccine for Diphtheria and hepatitis B human population.

III. We assume disease induced death in human population [26, 31, 32, 33, 34, 35].

2.1. Model Flow Diagram. The figure 1 above shows how shared risk factors and transmission routes can lead to diphtheria and hepatitis B co-infection [27, 28]. Co-infection increases disease severity and complicates diagnosis and management. Early detection, appropriate treatment, isolation, and strong vaccination programs are key to preventing transmission and reducing complications.

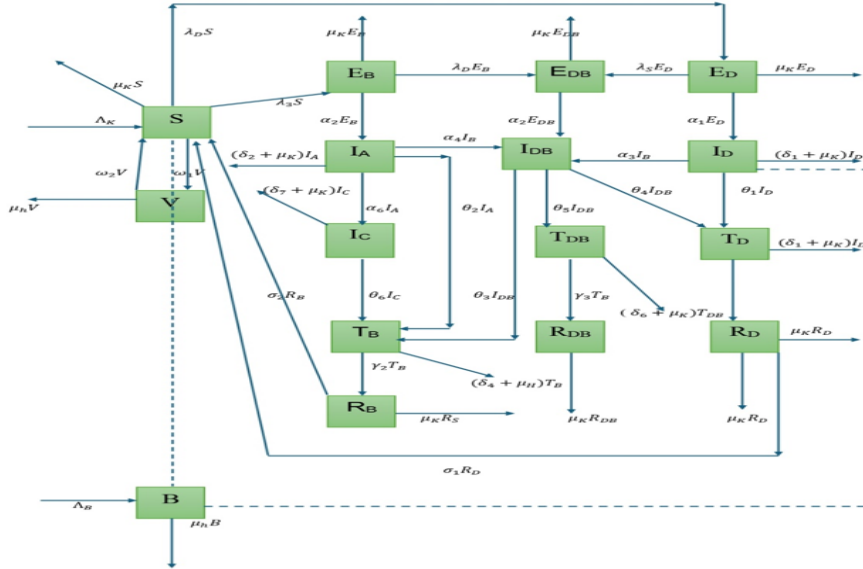


FIGURE 1. Diphtheria and Hepatitis B co-infection flow Diagram

2.2. Model Equations. Arising from the model formulation, flow diagram above, guided with assumptions, we obtain the model equations as follows:

$$\begin{aligned}
\frac{dS}{dt} &= \Lambda_h + \omega_2 V + \sigma_1 R_D + \sigma_2 R_B - \lambda_B S - \lambda_D S - (\omega_1 + \mu_h) S, \\
\frac{dE_D}{dt} &= \lambda_D S - \lambda_B E_D - (\alpha_1 + \mu_h) E_D, \\
\frac{dE_{DB}}{dt} &= \lambda_B E_D + \lambda_D E_B - (\alpha_5 + \mu_h) E_{DB}, \\
\frac{dE_B}{dt} &= \lambda_B S - \lambda_D E_B - (\alpha_2 + \mu_h) E_B, \\
\frac{dV}{dt} &= \omega_1 S - (\omega_2 + \mu_h) V, \\
\frac{dI_D}{dt} &= \alpha_1 E_D - (\theta_1 + \alpha_3 + \delta_1 + \mu_h) I_D, \\
\frac{dI_A}{dt} &= \alpha_2 E_B - (\theta_2 + \alpha_4 + \alpha_6 + \delta_2 + \mu_h) I_A, \\
\frac{dI_C}{dt} &= \alpha_6 I_A - (\theta_6 + \delta_7 + \mu_h) I_C,
\end{aligned} \tag{1}$$

$$\begin{aligned}
\frac{dI_{DB}}{dt} &= \alpha_3 I_D + \alpha_4 I_B + \alpha_5 E_{DB} - (\theta_3 + \theta_4 + \theta_5 + \delta_5 + \mu_h) I_{DB} \\
\frac{dT_D}{dt} &= \theta_1 I_D + \theta_4 I_{DB} - (\gamma_1 + \delta_3 + \mu_h) T_D \\
\frac{dT_B}{dt} &= \theta_3 I_{DB} + \theta_2 I_A + \theta_6 I_C - (\gamma_2 + \delta_4 + \mu_h) T_B \\
\frac{dT_{DB}}{dt} &= \theta_5 I_{DB} - (\gamma_3 + \delta_6 + \mu_h) T_{DB} \\
\frac{dR_D}{dt} &= \gamma_1 T_D - (\sigma_1 + \mu_h) R_D \\
\frac{dR_{DB}}{dt} &= \gamma_3 T_{DB} - \mu_h R_{DB} \\
\frac{dR_B}{dt} &= \gamma_2 T_B + \gamma_4 I_C - (\sigma_2 + \mu_h) R_B \\
\frac{dB}{dt} &= \Lambda_B - \mu_B B
\end{aligned}$$

Where

$$\begin{aligned}
\lambda_D &= \frac{(\psi_1 I_D + \psi_2 T_D + \psi_3 B + \psi_4 I_{DB} + \psi_5 T_{DB})}{N_h} \\
\lambda_B &= \frac{(\beta_1 I_A + \beta_2 T_B + \psi_4 I_{DB} + \psi_5 T_{DB} + \psi_6 I_C)}{N_h}
\end{aligned}$$

VARIABLES	DESCRIPTION
S	Susceptible human population
V	Population of human vaccinated against both disease
E_D	Exposed individuals to diphtheria only
I_D	Infected individuals with diphtheria only
T_D	Treated individuals with diphtheria only
R_D	Recovered individuals from diphtheria only
E_B	Exposed individuals to hepatitis B only
I_A	Infected individuals with acute hepatitis B only
I_C	Infected individuals with chronic hepatitis B only
T_B	Treated individuals with hepatitis B only

2.3. Model Variables and Parameters Descriptions.

R_B	Recovered individuals from hepatitis B only
E_{DB}	Exposed individuals to both diphtheria and hepatitis B
I_{DB}	Infected individuals with both diphtheria and hepatitis B
T_{DB}	Treated individuals with both diphtheria and hepatitis B
R_{DB}	Recovered individuals from both diphtheria and hepatitis B
Parameters	Description
Λ_h	Recruitment rate of human population
μ_h	Natural death rate of human population
ω_1	Vaccination rate
ω_2	Waning rate of vaccine
ψ_1	Contact rate between susceptible humans population and infected humans with diphtheria only
ψ_2	Contact rate between susceptible humans population and humans on treatment with diphtheria only
ψ_3	Contact rate between susceptible human population and bacteria population
ψ_4	Contact rate between susceptible human population and infected individual with both diphtheria and hepatitis B
ψ_5	Contact rate between susceptible humans population and humans on treatment with diphtheria and hepatitis B
ψ_6	Contact rate between susceptible humans population and chronic infected humans with hepatitis B only
β_1	Contact rate between susceptible humans population and acute infected humans with hepatitis B only
β_2	Contact rate between susceptible humans population and humans on treatment with hepatitis B only

α_1	Progression rate from exposed human population to diphtheria only to infected human population with diphtheria only
θ_2	Treatment rate of acute infected humans with hepatitis B only
α_2	Progression rate from exposed human population to hepatitis B only to acute infected human population with hepatitis B only
α_6	Progression rate from acute hepatitis B only infected human population to chronic infected human population with hepatitis B only
θ_6	Treatment rate of infected humans with hepatitis B only
θ_1	Treatment rate of infected humans with diphtheria only
γ_1	Recovery rate due to treatment of infected humans with diphtheria only
γ_3	Recovery rate due to treatment of infected humans with both diseases
σ_2	The rate at which recovered humans from hepatitis B only become susceptible again

σ_1	The rate at which recovered humans from diphtheria only become susceptible again
δ_1	Disease induced death rate of infected humans with diphtheria only
δ_3	Disease induced death rate of humans on treatment of diphtheria only
α_3	Progression rate of infected humans population with both diphtheria and hepatitis B to exposed human population with both diphtheria and hepatitis B
θ_4	The rate at which infected humans with diphtheria and hepatitis B receive diphtheria only treatment
θ_3	The rate at which infected humans with diphtheria and hepatitis B receive hepatitis B only treatment
δ_5	Disease induced death rate of infected humans with both diphtheria and hepatitis B
δ_6	Disease induced death rate of humans on treatment of both diphtheria and hepatitis B
θ_5	Treatment rate of infected humans with both diseases
α_2	Progression rate from exposed human population to hepatitis B only to acute infected human population
γ_2	Recovery rate due to treatment of infected humans with hepatitis B only

δ_2	Disease induced death rate of infected humans with hepatitis B only
δ_4	Disease induced death rate of humans on treatment of hepatitis B only
Λ_B	Recruitment rate of bacterial

Table 1 summarizes the key variables and parameters used in the study, including their definitions and roles in analyzing diphtheria and hepatitis B co-infection [27].

3. Preliminaries

3.1. Background materials. The manuscript relies on Fractal fractional operator definitions which we will explain first. Let us define (τ) to represent fractional-order while the symbol (ψ) stands for fractal-dimension. When $y(t)$ remain continuous it also has fractal differentiability on (a, b) with (ψ) , these definitions find their description in [21] as well as [22].

Definition 3.1. [35]. The Caputo fractional differential operator makes use of the Mittag-Leffler Kernel to define the fractal fractional derivative of functions $y(t)$ at order (τ) while operating on dimension (ψ) as the follows:

$$FFHD_{0,t}^{\tau,\psi} y(t) = \frac{AB(\sigma)}{1-\sigma} \int_a^t E_\phi \left[\frac{-\tau}{1-\tau} (t-s)^\tau \right] \frac{dy(s)}{dS^\psi} ds$$

$$t \geq a \geq 0$$

Where $\tau, \psi \in (0, 1]$, $AB(\tau) = 1 - \tau + \tau/\Gamma(\tau)$ is the normalization function with $AB(0) = AB(1) = 1$ and

$$d_x(t)/d_t^\psi = \lim_{S \rightarrow t} (x(s) - x(t)) / (s^\psi - t^\psi).$$

Moreover, the Mittag-Leffler function is defined by;

$$E_\tau(Z) = \sum_{k=0}^{\infty} Z^k \Gamma(\tau k + 1), Z, \tau \in C, \tau > 0,$$

with C is the set the of complex number.

Definition 3.2

[21,22]. We present the equation for fractal fractional integration with the Mittag-Leffler Kernel in the Caputo sense of order (τ) for dimension (ψ) ;

$$FFHD_{0,t}^{\tau,\psi} y(t) = \frac{\psi(1-\tau)t^{\psi-1}x(t)}{AB(\tau)} + \frac{\tau\psi}{AB(\tau)} \int_a^t (t-s)^{\tau-1} S^{\psi-1} x(s) ds, t \geq a \geq 0 \tag{3}$$

Definition 3.3

[21]. $x \in C((a, b), R)$, The fractal Laplace transform of order (τ) is given as follows

$${}^F L_p^\psi x(t) = \int_0^\infty x(t) \exp(-pt)t^{\psi-1} dt \tag{4}$$

Lemma 3.2. [22] *Fractal fractional initial value problem is as follows:*

$$\begin{cases} FFHD_{0,t}^{\tau,\psi} x(t) = G(t, x(t)), t \in [0, T], \tau, \psi \in (0, 1] \\ x(0) = x_0 \end{cases} \tag{5}$$

The problem in (4) can be expressed as [50]

$$\begin{cases} FFHD_{0,t}^{\tau,\psi} x(t) = \psi t^{\psi-1} G(t, x(t)), t \in [0, T], \tau, \psi \in (0, 1] \\ x(0) = x_0. \end{cases} \tag{6}$$

The corresponding integral equation defines the solution of problem (5) is as follow:

$$x(t) = x(0) + \frac{\psi t^{\tau-1}(1-\sigma)G(t, x(t))}{AB(\tau)} + \tau \frac{\psi \tau}{AB(\tau)\Gamma(\sigma)} \int_0^t S^{\psi-1}(t-s)^{\tau-1} G(S, x(s)) dS \tag{7}$$

Several Lemmas became necessary for analyzing both uniqueness and existence in the proposed model.

Lemma 3.3 (Banach fixed point theorem). [23] *The Banach space X contains a closed subset $D \subset X, D \neq \emptyset$ within it.*

The operator P yields exactly one fixed point in D when it meets the contraction condition $P : D \rightarrow D$.

Lemma 3.4 (Krasnoselskii’s fixed point theorem). [24] *The set D consists of every element from Y forming both a non-empty subset and a closed as well as convex space.*

Let P and Q represent the two operators such that,

- $PU + QV \in D$, whenever $U, V \in D$,
- Q is compact and continuous
- P denotes the contraction mapping. then, there exist $k \in D$ such $Z = PK + QK$.

The new application utilizes the fractal fractional derivative operator to advance the Typhoid fever model (2.7) within the Mittag-Leffler Kernel framework $\tau \in (0, 1]$ and $\psi \in [0, 1]$, $FFHD_{0,t}^{\tau,\psi}$, as follows:

$$\begin{aligned} FFHD_{0,t}^{\tau,\psi} S(t) &= G_1 \left(t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \right. \\ &\quad \left. I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \right), \\ FFHD_{0,t}^{\tau,\psi} E_D(t) &= G_2 \left(t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \right. \\ &\quad \left. I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \right), \end{aligned}$$

$$\begin{aligned}
 FFHD_{0,t}^{\tau,\psi} E_{DB}(t) &= G_3 \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} E_B(t) &= G_4 \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} V(t) &= G_5 \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} I_D(t) &= G_6 \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} I_A(t) &= G_7 \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} I_C(t) &= G_8 \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} I_{DB}(t) &= G_9 \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} T_D(t) &= G_{10} \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} T_B(t) &= G_{11} \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} T_{DB}(t) &= G_{12} \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} R_D(t) &= G_{13} \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} R_{DB}(t) &= G_{14} \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} R_B(t) &= G_{15} \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 FFHD_{0,t}^{\tau,\psi} B(t) &= G_{16} \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}, \\
 \text{Where } G_i &= G_i \begin{pmatrix} t, S(t), E_D(t), E_{DB}(t), E_B(t), V(t), I_D(t), I_A(t), I_C(t), \\ I_{DB}(t), T_D(t), T_B(t), T_{DB}(t), R_D(t), R_{DB}(t), R_B(t), B(t) \end{pmatrix}.
 \end{aligned} \tag{8}$$

The functions G_i of the developed model follow the sequence for $i = 1, 2, 3, \dots, 16$ below:

$$\begin{aligned}
 G_1 &= \Lambda_h + \omega_2 V + \sigma_1 R_D + \sigma_2 R_B - \lambda_B S - \lambda_D S - (\omega_1 + \mu_h) S, \\
 G_2 &= \lambda_D S - \lambda_B E_D - (\alpha_1 + \mu_h) E_D, \\
 G_3 &= \lambda_B E_D + \lambda_D E_B - (\alpha_5 + \mu_h) E_{DB}, \\
 G_4 &= \lambda_B S - \lambda_D E_B - (\alpha_2 + \mu_h) E_B, \\
 G_5 &= \omega_1 S - (\omega_2 + \mu_h) V, \\
 G_6 &= \alpha_1 E_D - (\theta_1 + \alpha_3 + \delta_1 + \mu_h) I_D,
 \end{aligned}$$

$$\begin{aligned}
G_7 &= \alpha_2 E_B - (\theta_2 + \alpha_4 + \alpha_6 + \delta_2 + \mu_h) I_A, \\
G_8 &= \alpha_6 I_A - (\theta_6 + \delta_7 + \mu_h) I_C, \\
G_9 &= \alpha_3 I_D + \alpha_4 I_B + \alpha_5 E_{DB} - (\theta_3 + \theta_4 + \theta_5 + \delta_5 + \mu_h) I_{DB}, \\
G_{10} &= \theta_1 I_D + \theta_4 I_{DB} - (\gamma_1 + \delta_3 + \mu_h) T_D, \\
G_{11} &= \theta_3 I_{DB} + \theta_2 I_A + \theta_6 I_C - (\gamma_2 + \delta_4 + \mu_h) T_B, \\
G_{12} &= \theta_5 I_{DB} - (\gamma_3 + \delta_6 + \mu_h) T_{DB}, \\
G_{13} &= \gamma_1 T_D - (\sigma_1 + \mu_h) R_D, \\
G_{14} &= \gamma_3 T_{DB} - \mu_h R_{DB}, \\
G_{15} &= \gamma_2 T_B + \gamma_4 I_C - (\sigma_2 + \mu_h) R_B \\
G_{16} &= \Lambda_B - \mu_B B
\end{aligned} \tag{9}$$

With

$$\begin{aligned}
S_0 \geq 0, E_{D0} \geq 0, E_{DB0} \geq 0, E_{B0} \geq 0, V_0 \geq 0, \\
I_{D0} \geq 0, I_{A0} \geq 0, I_{C0} \geq 0, I_{DB0} \geq 0, T_{D0} \geq 0, T_{B0} \geq 0, T_{DB0} \geq 0, \\
R_{D0} \geq 0, R_{DB0} \geq 0, R_{B0} \geq 0, B_0 \geq 0.
\end{aligned}$$

The fractal fractional FF-Diphtheria and Hepatitis B co-infection model exists under the label (8).

We observed that if we let $\tau = 1$ in the FF-Diphtheria and Hepatitis B co-infection model transforms into the (7) to create an integer-order model system.

The model gets simplified into the fractional Diphtheria and Hepatitis B co-infection model by letting $\psi = 1$.

3.7. Model Analysis. Positivity of the model solution

$$\left(R_+^{16} = \left\{ G \in R_+^{16} : G \geq 0 \& (S, E_D, E_{DB}, E_B, V, I_D, I_A, I_C, I_{DB}, T_D, T_B, T_{DB}, R_D, R_{DB}, R_B, B)^T \right\} \right)$$

The vector transform is expressed by $(.)^T$.

Theorem 3.5. *G satisfies boundedness in R_+^{16} in addition to being non-negative for the solution of the FF-Diphtheria and Hepatitis B co-infection model described by (8).*

Proof. For $t \in (0, \infty)$, Existence and uniqueness of the FF-Diphtheria and Hepatitis B co-infection model (8) is obtained.

Consequently, The non-negative region R_+^{16} represents a positive invariant region according to the presented content. Our research used the FF-Diphtheria and Hepatitis B co-infection model described in (8) as the basis for our analysis;

$$\begin{aligned}
FFHD_{0,t}^{\tau,\psi} S(t) &= \Lambda_h + \omega_2 v + \sigma_1 R_D + \sigma_2 R_B + \sigma_3 R_{DB} \geq 0 \\
FFHD_{0,t}^{\tau,\psi} E_D(t) &= \lambda_D S \geq 0 \\
FFHD_{0,t}^{\tau,\psi} E_{DB}(t) &= \lambda_B E_B + \lambda_D E_B \geq 0 \\
FFHD_{0,t}^{\tau,\psi} E_B(t) &= \lambda_B S \geq 0
\end{aligned}$$

$$\begin{aligned}
 FFHD_{0,t}^{\tau,\psi} V(t) &= \omega_1 S \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} I_D(t) &= \alpha_1 E_B \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} I_A(t) &= \alpha_2 E_B \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} I_C(t) &= \alpha_6 I_A \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} I_{DB}(t) &= \alpha_3 I_D + \alpha_4 I_B + \alpha_5 E_{DB} \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} T_D(t) &= \theta_1 I_D + \theta_4 I_{DB} \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} T_B(t) &= \theta_3 I_{DB} + \theta_2 I_A + \theta_6 I_C \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} T_{DB}(t) &= \theta_5 I_{DB} \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} R_D(t) &= \gamma_1 T_D \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} R_{DB}(t) &= \gamma_3 T_{DB} \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} R_B(t) &= \gamma_2 T_B + \gamma_4 I_C \geq 0 \\
 FFHD_{0,t}^{\tau,\psi} B(t) &= \Lambda_B \geq 0
 \end{aligned} \tag{10}$$

If $(S, E_{D0}, E_{DB}, E_{B0}, V, I_{D0}, I_{A0}, I_{C0}, I_{DB0}, T_{D0}, T_{B0}, T_{DB0}, R_{D0}, R_{DB0}, R_{B0}, B_0) \in R_+^{16}$, then, from the time interval from (10) solution G remains prevented from escaping through the hyper planes. Consequently, R_+^{16} is a positively invariant.

$N_h = S + E_D + E_{DB} + E_B + V + I_D + I_A + I_C + I_{DB} + T_D + T_B + T_{DB} + R_D + R_{DB} + R_B + B$ and $N_B = B$.

So from the FF-Diphtheria and Hepatitis B model (8) we obtained:

$$\left\{ FFHD_{0,t}^{\tau,\psi} N_h(t) = \Lambda_h - \mu_h N_h \right\} \text{ and } \left\{ FFHD_{0,t}^{\tau,\psi} N_B(t) = \Lambda_B - \mu_B N_B \right\} \tag{11}$$

Using the fractal Laplace transform Eq. (3), we conclude that,

$$N_h(t) \leq \frac{\Lambda_h}{\mu_h} \text{ as } t \rightarrow \infty, \quad N_B \leq \frac{\Lambda_B}{\mu_B} \text{ and } t \rightarrow \infty.$$

So we now have the biological range of values of the FF-Diphtheria and Hepatitis B co-infection model (8) as follows;

$$\{(S, E_D, E_{DB}, E_B, V, I_D, I_A, I_C, I_{DB}, T_D, T_B, T_{DB}, R_D, R_{DB}, R_B, B) \in R^{16} \geq 0\} \text{ and } N_h(t) \leq \frac{\Lambda_h}{\mu_h}$$

Similarly, $N_h \leq \frac{\Lambda_b}{\mu_b}$.

This shows that the above model is biologically feasible. \square

3.2. Equilibrium points of Hepatitis B and Diphtheria and their stabilities. The FF-Diphtheria model demonstrates two steady states which occur when the right side of equivalence (8) displays zero value. Then, we obtain;

3.3. The Disease free Equilibrium point. The disease-free equilibrium exists as a state with zero illness symptoms in human populations.

$$\begin{aligned} & (S^0, E_D^0, E_{DB}^0, E_B^0, V^0, I_D^0, I_A^0, I_C^0, I_{DB}^0, T_D^0, T_B^0, T_{DB}^0, R_D^0, R_{DB}^0, R_B^0, B^0) \\ &= \left(\frac{\Lambda_h (\omega_2 + \mu_h)}{\mu_h (\omega_2 + \omega_1 + \mu_h)}, 0, 0, 0, \frac{\omega_1 \Lambda_h}{\mu_h (\omega_2 + \omega_1 + \mu_h)}, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, 0, \frac{\Lambda_B}{\mu_B} \right). \end{aligned} \quad (12)$$

3.4. Hepatitis B sub-model Basic Reproduction Number. Basic Reproduction number defines the complete number of infections which spread from one infected human to susceptible population. The determination of Basic Reproduction number happens through next generation method $R_0^T = \rho FV^{-1}$ by using F as a non-negative matrix with other transition terms V and determining the largest Eigen value ρ .

$$\begin{aligned} F &= \begin{bmatrix} 0 & \frac{\beta_1(\omega_2+\mu_h)}{\omega_2+\omega_1+\mu_h} & \frac{\beta_2(\omega_2+\mu_h)}{\omega_2+\omega_1+\mu_h} & \frac{\beta_3(\omega_2+\mu_h)}{\omega_2+\omega_1+\mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad V = \begin{bmatrix} A_1 & 0 & 0 & 0 \\ -\alpha_2 & A_2 & 0 & 0 \\ 0 & -\alpha_6 & A_3 & 0 \\ 0 & -\theta_2 & -\theta_6 & A_4 \end{bmatrix} \\ V^{-1} &= \begin{bmatrix} \frac{1}{A_1} & 0 & 0 & 0 \\ \frac{\alpha_2}{A_2 A_1} & \frac{1}{A_2} & 0 & 0 \\ \frac{\alpha_6 \alpha_2}{A_3 A_2 A_4} & \frac{\alpha_6}{A_3 A_4} & \frac{1}{A_4} & 0 \\ \frac{\theta_2 \alpha_2}{A_3 A_2 A_5} & \frac{\theta_2}{A_3 A_5} & 0 & \frac{1}{A_5} \end{bmatrix} \\ R_0^B &= \frac{\alpha_2 (A_4 A_5 \beta_1 \mu_h + A_4 A_5 \beta_1 \omega_1 + A_4 \beta_3 \mu_h \theta_2 + A_4 \beta_3 \omega_1 \theta_2 + A_5 \alpha_6 \beta_2 \mu_h + A_5 \alpha_6 \beta_2 \omega_1)}{A_2 A_3 A_4 A_5 (\omega_2 + \omega_1 + \mu_h)}. \end{aligned} \quad (13)$$

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

3.5. Hepatitis B sub-model Endemic Equilibrium points. We examined the possibility of an endemic equilibrium point, signifying a stable condition where Hepatitis B persists within the population. At this equilibrium, the model's variables remain positive and retain non-zero values.

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

To analyze the endemic equilibrium point, the model equations are reformulated based on the infection rates within the populations [41, 42]. Utilizing the fractional Hepatitis B model (6), the endemic equilibrium state is defined as follows:

$$\begin{aligned} S^* &= \frac{\Lambda_h A_6 A_2 A_3 A_4 A_5 A_7}{((A_2 A_3 A_7 (\lambda_B + A_1) A_5 - \gamma_2 \sigma_2 \theta_2 \lambda_B \alpha_2) A_4 - \sigma_2 \lambda_B \alpha_2 \alpha_6 (A_5 \gamma_4 + \gamma_2 \theta_6)) A_6 - A_2 A_3 A_4 A_5 A_7 \omega_1 \omega_2}, \\ E_B^* &= \frac{\Lambda_h A_6 A_3 A_4 A_5 A_7 \lambda_B}{(((A_2 A_3 A_7 A_5 - \alpha_2 \gamma_2 \sigma_2 \theta_2) A_4 - \sigma_2 \alpha_2 \alpha_6 (A_5 \gamma_4 + \gamma_2 \theta_6)) \lambda_B + A_1 A_2 A_3 A_4 A_5 A_7) A_6 - A_2 A_3 A_4 A_5 A_7 \omega_1 \omega_2}, \\ I_A^* &= \frac{\Lambda_h A_6 A_4 A_5 A_7 \lambda_B \alpha_2}{(((A_2 A_3 A_7 A_5 - \alpha_2 \gamma_2 \sigma_2 \theta_2) A_4 - \sigma_2 \alpha_2 \alpha_6 (A_5 \gamma_4 + \gamma_2 \theta_6)) \lambda_B + A_1 A_2 A_3 A_4 A_5 A_7) A_6 - A_2 A_3 A_4 A_5 A_7 \omega_1 \omega_2}, \\ I_C^* &= \frac{\Lambda_h A_6 A_5 A_7 \lambda_B \alpha_2 \alpha_6}{(((A_2 A_3 A_4 A_7 - \alpha_2 \alpha_6 \gamma_4 \sigma_2) A_5 - \alpha_2 \gamma_2 \sigma_2 (A_4 \theta_2 + \alpha_6 \theta_6)) \lambda_B + A_1 A_2 A_3 A_4 A_5 A_7) A_6 - A_2 A_3 A_4 A_5 A_7 \omega_1 \omega_2}, \end{aligned}$$

$$\begin{aligned}
 T_B^* &= \frac{\Lambda_h A_6 A_7 \lambda_B \alpha_2 (A_4 \theta_2 + \alpha_6 \theta_6)}{(((A_2 A_3 A_7 A_5 - \alpha_2 \gamma_2 \sigma_2 \theta_2) A_4 - \sigma_2 \alpha_2 \alpha_6 (A_5 \gamma_4 + \gamma_2 \theta_6)) \lambda_B + A_1 A_2 A_3 A_4 A_5 A_7) A_6 - A_2 A_3 A_4 A_5 A_7 \omega_1 \omega_2}, \\
 V^* &= \frac{\Lambda_h A_2 A_3 A_4 A_5 A_7 \omega_1}{(A_7 A_2 ((\lambda_B + A_1) A_6 - \omega_1 \omega_2) A_3 A_5 - \gamma_2 \sigma_2 \theta_2 \lambda_B A_6 \alpha_2) A_4 - \sigma_2 \lambda_B A_6 \alpha_2 \alpha_6 (A_5 \gamma_4 + \gamma_2 \theta_6)}, \\
 R_B^* &= \frac{\Lambda_h \lambda_B (A_4 \gamma_2 \theta_2 + \alpha_6 (A_5 \gamma_4 + \gamma_2 \theta_6)) \alpha_2 A_6}{(((A_2 A_3 A_5 A_7 - \alpha_2 \gamma_2 \sigma_2 \theta_2) A_4 - \sigma_2 \alpha_2 \alpha_6 (A_5 \gamma_4 + \gamma_2 \theta_6)) \lambda_B + A_1 A_2 A_3 A_4 A_5 A_7) A_6 - A_2 A_3 A_4 A_5 A_7 \omega_1 \omega_2}.
 \end{aligned} \tag{14}$$

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:

$$Q_1 \lambda_B + Q_2 = 0 \tag{15}$$

Where

$$\begin{aligned}
 Q_1 &= \Lambda_h \left(\begin{aligned} &A_3 A_4 A_5 A_6 A_7 + A_4 A_5 A_6 A_7 \alpha_2 + A_4 A_6 A_7 \alpha_2 \theta_2 \\ &+ A_4 A_6 \alpha_2 \gamma_2 \theta_2 + A_5 A_6 A_7 \alpha_2 \alpha_6 + A_5 A_6 \alpha_2 \alpha_6 \gamma_4 \\ &+ A_6 A_7 \alpha_2 \alpha_6 \theta_6 + A_6 \alpha_2 \alpha_6 \gamma_2 \theta_6 \end{aligned} \right) \\
 Q_2 &= \Lambda_h (A_6 A_2 A_3 A_4 A_5 A_7 + (A_2 A_3 A_4 A_5 A_7 \omega_1) (1 - R_0^B))
 \end{aligned}$$

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

3.6. Sensitivity Analysis of Hepatitis B sub-model.

$$\begin{aligned}
 S_{\alpha_2}^{R_0^B} &= \frac{\mu_h}{\alpha_2 + \mu_h} = 0.000188, \\
 S_{\theta_6}^{R_0^B} &= - \frac{\alpha_6 \beta_2 (\gamma_2 + \delta_4 + \mu_h) \theta_6}{(\theta_6 + \gamma_4 + \delta_7 + \mu_h) \left(\frac{(\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h) \beta_1 + (\alpha_6 \beta_2 + \beta_3 \theta_2) \mu_h}{+ \alpha_6 \beta_2 \gamma_2 + \alpha_6 \beta_2 \delta_4 + \beta_3 \theta_2 (\theta_6 + \gamma_4 + \delta_7)} \right)} = -0.28352, \\
 S_{\gamma_4}^{R_0^B} &= - \frac{\alpha_6 \beta_2 (\gamma_2 + \delta_4 + \mu_h) \gamma_4}{(\theta_6 + \gamma_4 + \delta_7 + \mu_h) \left(\frac{(\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h) \beta_1 + (\alpha_6 \beta_2 + \beta_3 \theta_2) \mu_h}{+ \alpha_6 \beta_2 \gamma_2 + \theta_2 \beta_3 \gamma_4 + \alpha_6 \beta_2 \delta_4 + \theta_2 \beta_3 (\delta_7 + \theta_6)} \right)} = -0.141758, \\
 S_{\delta_7}^{R_0^B} &= - \frac{\alpha_6 \beta_2 (\gamma_2 + \delta_4 + \mu_h) \delta_7}{(\theta_6 + \gamma_4 + \delta_7 + \mu_h) \left(\begin{aligned} &(\theta_6 + \gamma_4 + \delta_7 + \mu_h) (\gamma_2 + \delta_4 + \mu_h) \beta_1 \\ &+ (\alpha_6 \beta_2 + \beta_3 \theta_2) \mu_h + \alpha_6 \beta_2 \gamma_2 \\ &+ \alpha_6 \beta_2 \delta_4 + \beta_3 \theta_2 (\theta_6 + \gamma_4 + \delta_7) \end{aligned} \right)} = -0.189011, \\
 S_{\delta_4}^{R_0^B} &= - \frac{\theta_2 \beta_3 (\theta_6 + \gamma_4 + \delta_7 + \mu_h) \delta_4}{(\gamma_2 + \delta_4 + \mu_h) \left(\begin{aligned} &(\theta_6 + \gamma_4 + \delta_7 + \mu_h) (\gamma_2 + \delta_4 + \mu_h) \beta_1 \\ &+ (\alpha_6 \beta_2 + \beta_3 \theta_2) \mu_h + \alpha_6 \beta_2 \gamma_2 + \alpha_6 \beta_2 \delta_4 \\ &+ \beta_3 \theta_2 (\theta_6 + \gamma_4 + \delta_7) \end{aligned} \right)} = -0.25055, \\
 S_{\gamma_2}^{R_0^B} &= - \frac{\beta_3 \theta_2 (\theta_6 + \gamma_4 + \delta_7 + \mu_h) \gamma_2}{(\gamma_2 + \delta_4 + \mu_h) \left(\begin{aligned} &(\theta_6 + \gamma_4 + \delta_7 + \mu_h) (\gamma_2 + \delta_4 + \mu_h) \beta_1 \\ &+ (\alpha_6 \beta_2 + \beta_3 \theta_2) \mu_h + \alpha_6 \beta_2 \gamma_2 + \alpha_6 \beta_2 \delta_4 \\ &+ \beta_3 \theta_2 (\theta_6 + \gamma_2 + \delta_7) \end{aligned} \right)} = -0.313188,
 \end{aligned}$$

$$\begin{aligned}
S_{\beta_1}^{R_0^B} &= \frac{(\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h)\beta_1}{(\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h)\beta_1 + (\alpha_6\beta_2 + \beta_3\theta_2)\mu_h + \alpha_6\beta_2\gamma_2 + \alpha_6\beta_2\delta_4 + \beta_3\theta_2(\theta_6 + \gamma_4 + \delta_7)} = 0.5219 \\
S_{\omega_1}^{R_0^B} &= \frac{\omega_1\omega_2}{(\mu_h + \omega_1)(\omega_2 + \omega_1 + \mu_h)} = 0.62484, \\
S_{\beta_3}^{R_0^B} &= \frac{\beta_3\theta_2(\theta_6 + \gamma_4 + \delta_7 + \mu_h)}{(\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h)\beta_1} = 0.033828, \\
&+ (\alpha_6\beta_2 + \beta_3\theta_2)\mu_h + \alpha_6\beta_2\gamma_2 + \alpha_6\beta_2\delta_4 \\
&+ \beta_3\theta_2(\theta_6 + \gamma_4 + \delta_7) \\
S_{\theta_2}^{R_0^B} &= -\frac{\theta_2 \left(\begin{array}{l} (\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h)\beta_1 - \mu_h^2\beta_3 \\ + ((-\theta_6 - \gamma_4 - \alpha_6 - \delta_2 - \delta_7)\beta_3 + \alpha_6\beta_2)\mu_h \\ - (\alpha_6 + \delta_2)(\theta_6 + \gamma_4 + \delta_7)\beta_3 + \beta_2\alpha_6(\gamma_2 + \delta_4) \end{array} \right)}{\left(\begin{array}{l} (\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h)\beta_1 + (\alpha_6\beta_2 + \beta_3\theta_2)\mu_h \\ + \beta_3\theta_2(\theta_6 + \gamma_4 + \delta_7) + \beta_2\alpha_6(\gamma_2 + \delta_4) \end{array} \right) (\alpha_6 + \theta_2 + \delta_2 + \mu_h)} = -0.208665, \\
S_{\alpha_6}^{R_0^B} &= -\frac{\alpha_6 \left(\begin{array}{l} (\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h)\beta_1 - \mu_h^2\beta_2 \\ + (-\beta_2\gamma_2 - \beta_2\delta_4 + (-\beta_2 + \beta_3)\theta_2 - \beta_2\delta_2)\mu_h \\ - \beta_2(\theta_2 + \delta_2)\gamma_2 - \beta_2(\theta_2 + \delta_2)\delta_4 + \beta_3\theta_2(\theta_6 + \gamma_4 + \delta_7) \end{array} \right)}{\left(\begin{array}{l} (\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h)\beta_1 + (\alpha_6\beta_2 + \beta_3\theta_2)\mu_h \\ + \alpha_6\beta_2\gamma_2 + \alpha_6\beta_2\delta_4 + \beta_3\theta_2(\theta_6 + \gamma_4 + \delta_7) (\alpha_6 + \theta_2 + \delta_2 + \mu_h) \end{array} \right)} = 0.44363, \\
&= -0.497488, \\
S_{\beta_2}^{R_0^B} &= \frac{(\gamma_2 + \delta_4 + \mu_h)\alpha_6\beta_2}{(\theta_6 + \gamma_4 + \delta_7 + \mu_h)(\gamma_2 + \delta_4 + \mu_h)\beta_1 + (\alpha_6\beta_2 + \beta_3\theta_2)\mu_h} = 0.42\theta_6 + \gamma_4 + \delta_7 \\
&+ \alpha_6\beta_2\gamma_2 + \alpha_6\beta_2\delta_4 + \beta_3\theta_2(\theta_6) \\
S_{\omega_2}^{R_0^B} &= -\frac{\omega_2}{\omega_2 + \omega_1 + \mu_h} = -0.624956, \\
S_{\mu_h}^{R_0^B} &= -0.167264.
\end{aligned}$$

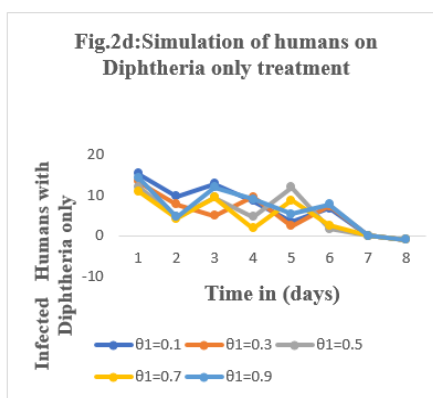
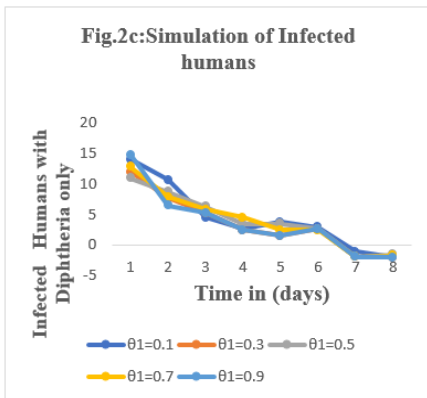
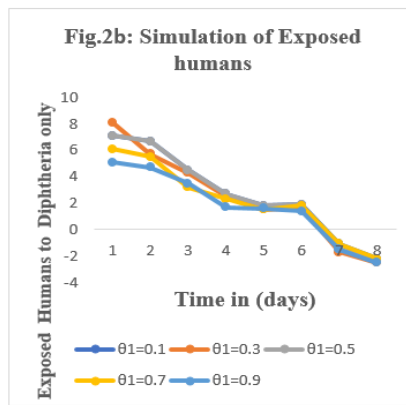
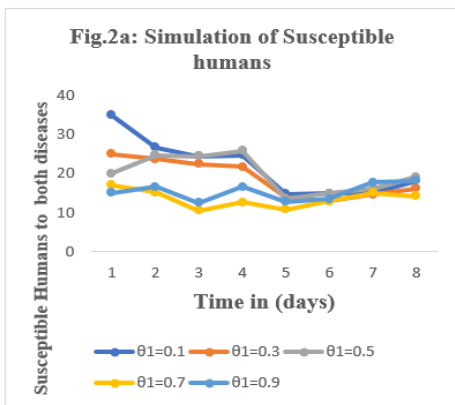
3.7. Diphtheria Sub-model Basic Reproduction Number. Basic Reproduction number defines the complete number of infections which spread from one infected human to susceptible population. The determination of Basic Reproduction number happens through next generation method $R_0^T = \rho FV^{-1}$ by using F as a non-negative matrix with other transition terms V and determining the largest Eigen value ρ .

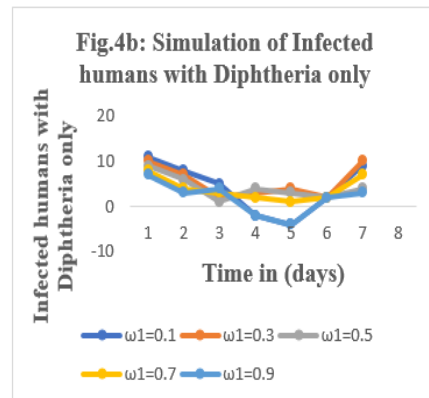
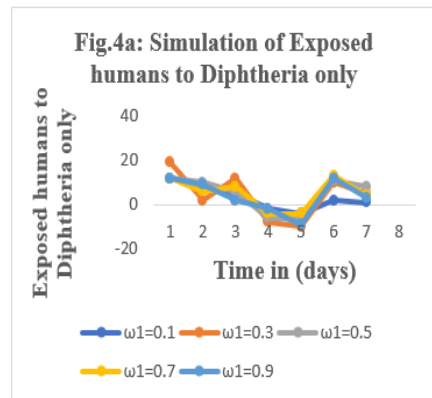
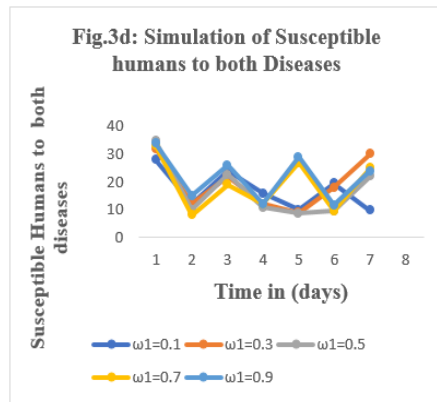
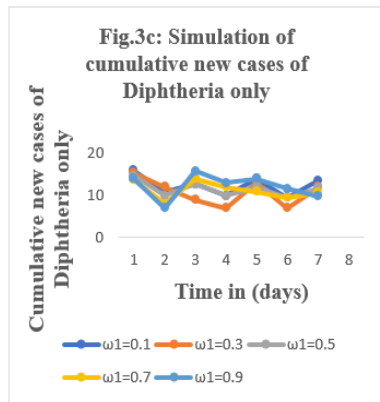
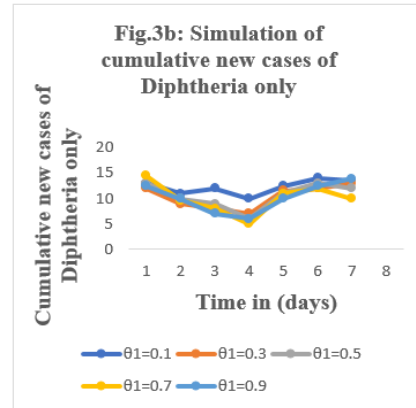
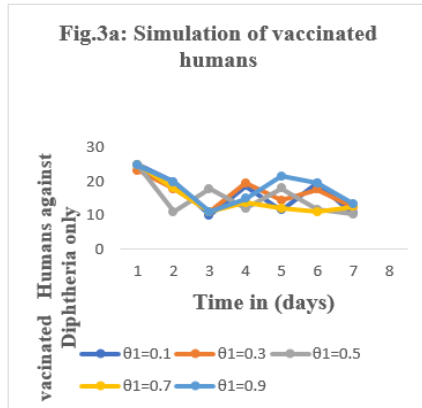
$$\begin{aligned}
F &= \begin{pmatrix} 0 & \frac{\psi_1(\omega_2 + \mu_h)}{\omega_2 + \omega_1 + \mu_h} & \frac{\psi_2(\omega_2 + \mu_h)}{\omega_2 + \omega_1 + \mu_h} & \frac{\psi_3(\omega_2 + \mu_h)}{\omega_2 + \omega_1 + \mu_h} \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix} \\
V &= \begin{pmatrix} K_2 & \frac{\psi_1(\omega_2 + \mu_h)}{\omega_2 + \omega_1 + \mu_h} & \frac{\psi_2(\omega_2 + \mu_h)}{\omega_2 + \omega_1 + \mu_h} & \frac{\psi_3(\omega_2 + \mu_h)}{\omega_2 + \omega_1 + \mu_h} \\ -\alpha_1 & K_3 & 0 & 0 \\ 0 & -\theta_1 & K_4 & 0 \\ 0 & 0 & 0 & K_7 \end{pmatrix} V^{-1} = \begin{pmatrix} \frac{1}{K_2} & 0 & 0 & 0 \\ \frac{\alpha_1}{K_2 K_3} & \frac{1}{K_3} & 0 & 0 \\ \frac{\theta_1 \alpha_1}{K_3 K_2 K_4} & \frac{\theta_1}{K_3 K_4} & \frac{1}{K_4} & 0 \\ 0 & 0 & 0 & \frac{1}{K_7} \end{pmatrix}
\end{aligned}$$

This is the dominant Eigen value:

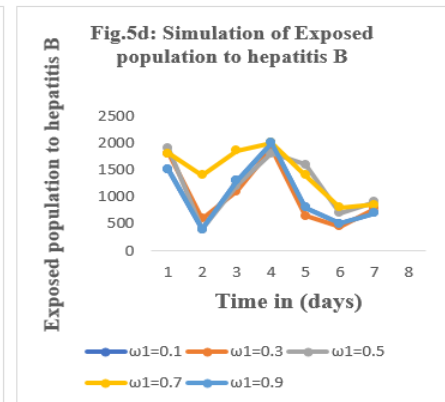
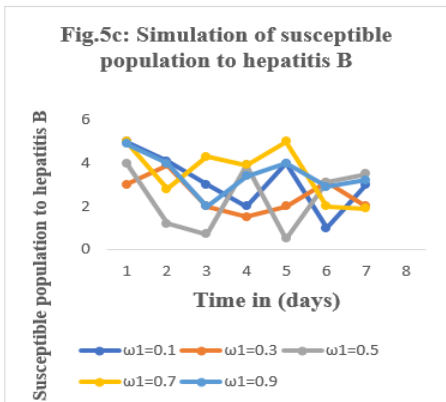
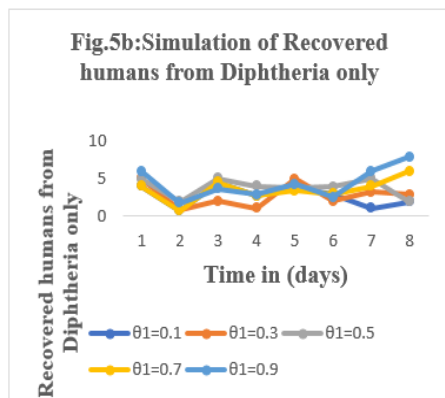
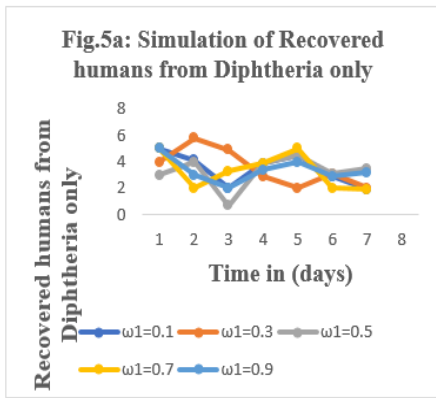
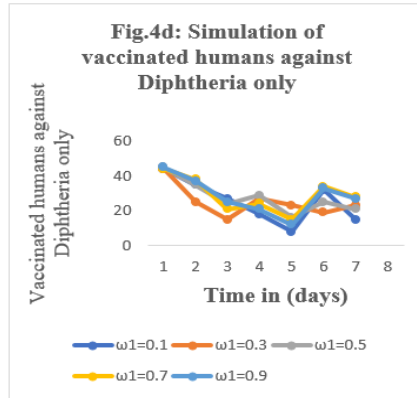
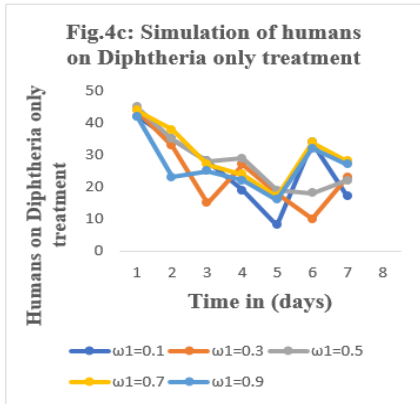
$$R_0 = \frac{\alpha_1 (K_4 \mu_h \psi_1 + K_4 \omega_2 \psi_1 + \mu_h \psi_2 \theta_1 + \omega_2 \psi_2 \theta_1)}{K_2 K_3 K_4 (\omega_2 + \omega_1 + \mu_h)} \quad (16)$$

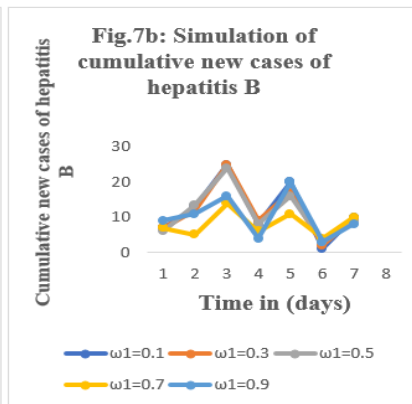
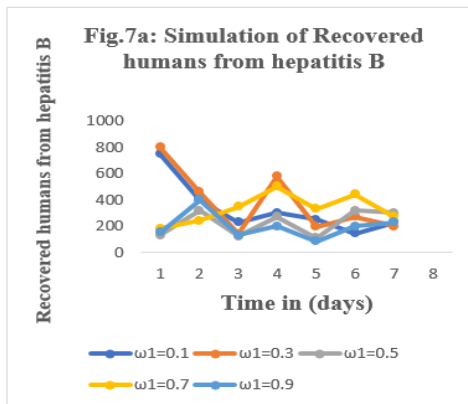
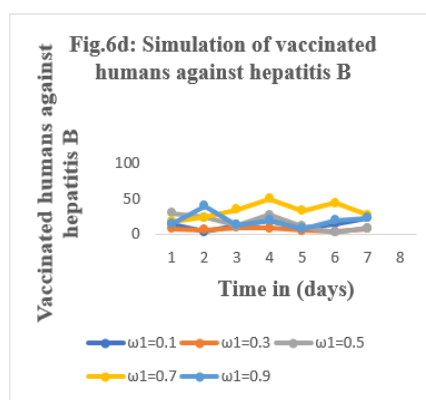
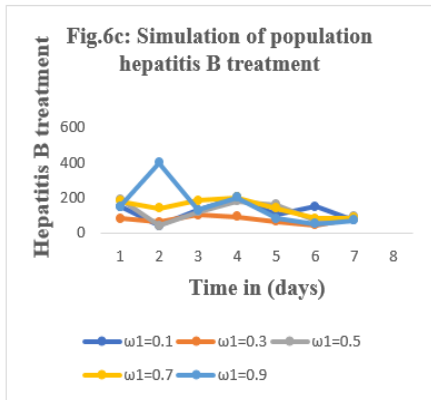
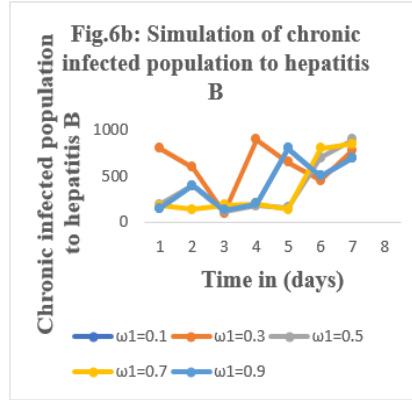
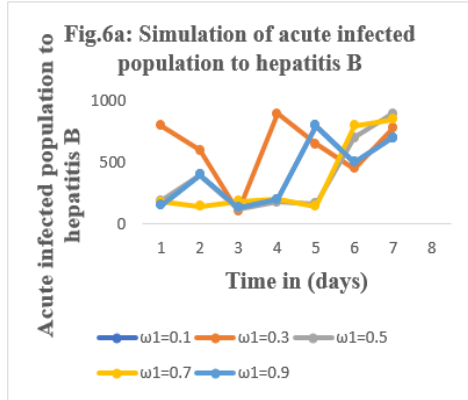
4. Numerical Simulation

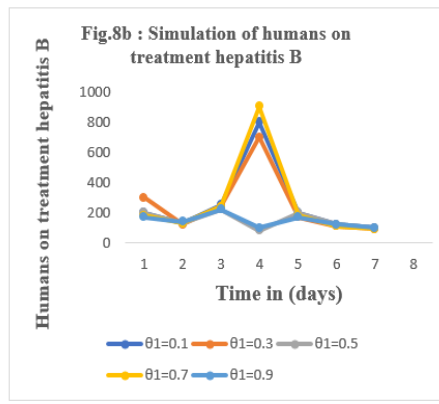
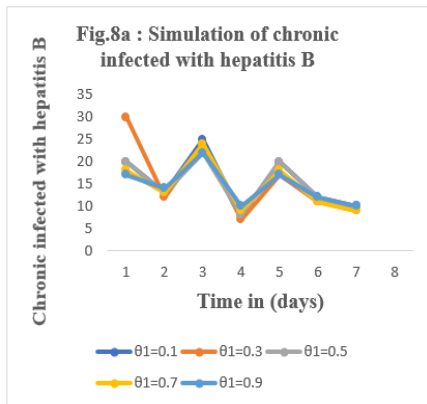
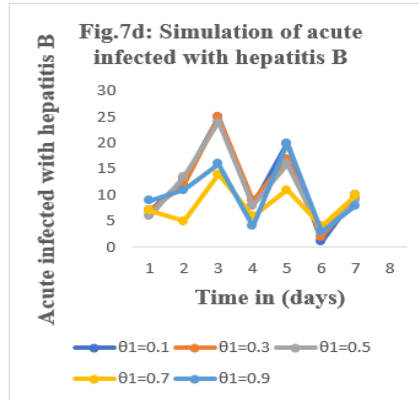
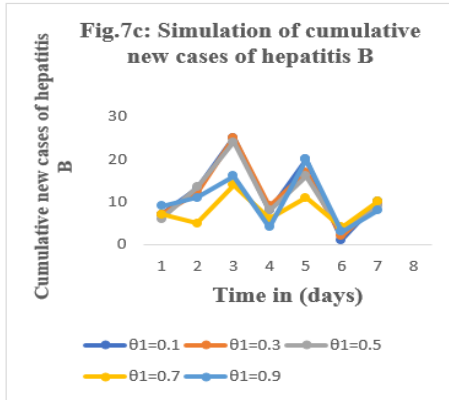




HEPATITIS B AND DIPHTHERIA VIA FRACTAL-FRACTIONAL







5. Discussion

The simulation findings show how treatment and vaccination rates affect the dynamics of infections with only hepatitis B and diphtheria. Raising the rate of diphtheria treatment increases the vulnerable and treated populations while drastically lowering the number of exposed and infected people. In a similar vein, increased vaccination rates reduce vulnerable, exposed, and infected populations, but they also increase the vaccinated class and may initially increase reported cumulative cases because of better detection.

Increasing vaccination rates for Hepatitis B increases the vaccinated group while decreasing susceptible, exposed, acute, and chronic infected populations. Increasing the number of people receiving treatment and recovering from Hepatitis B successfully lowers acute and chronic infections as well as cumulative instances. Overall, the findings show that lowering the burden of illness and preventing the spread of disease depend heavily on increased treatment and vaccination rates.

6. Conclusion

In order to understand the intricate dynamics of Hepatitis B and Diphtheria co-infection, this work provides a novel fractal-fractional mathematical model that incorporates several compartments, including susceptible, vaccinated, exposed, infectious, treated, recovered populations, and bacterial load. To capture memory effects, non-local interactions, and fractal features in disease transmission, the model uses a fractal-fractional derivative with a Mittag-Leffler kernel. Numerical models show that differences in fractional order and fractal dimension have a substantial impact on the course of the disease, resulting in longer infection peaks and longer epidemic durations, especially in co-infected and chronic compartments.

The findings demonstrate that, due to interactions between bacterial dynamics and viral persistence, co-infection increases the burden of disease in comparison to single infections. Additionally, while high transmission and susceptibility maintain epidemics, higher treatment, recovery, and immunization rates successfully lower infection levels. Sensitivity analysis of the basic reproduction number highlights the fractal-fractional framework's ability to capture co-infection dynamics and demonstrates that improving healthcare treatments and lowering transmission are crucial for successful disease control.

7. Competing Interests

The authors declare that they have no competing interests.

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M.SHARMILA : DEPARTMENT OF MATHEMATICS, KUNTHAVAI NAACCHIYAAR GOVERNMENT ARTS COLLEGE FOR WOMEN (AUTONOMOUS), (AFFILIATED TO BHARATHIDASAN UNIVERSITY, TIRUCHIRAPPALLI-620 024),THANJAVUR - 613007,TAMIL NADU, INDIA.

Email address: schitramohan@gmail.com

S.INDRAKALA : DEPARTMENT OF MATHEMATICS, KUNTHAVAI NAACCHIYAAR GOVERNMENT ARTS COLLEGE FOR WOMEN (AUTONOMOUS), (AFFILIATED TO BHARATHIDASAN UNIVERSITY, TIRUCHIRAPPALLI-620 024), THANJAVUR - 613007, TAMIL NADU, INDIA.

Email address: s.indirakala@yahoo.com