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MODELING AND CONTROL OF HEPATITIS B VIRUS TRANSMISSION DYNAMICS USING FRACTIONAL ORDER DIFFERENTIAL EQUATIONS

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Abstract. Hepatitis B virus (HBV) continues to pose a significant global health burden, necessitating the development of accurate and effective mathematical models to understand its transmission dynamics and devise optimal control strategies. In this research paper, we present a fractional order model for Hepatitis B virus transmission, incorporating the complexities of memory effects and non-local interactions in disease spread. The proposed fractional order model is formulated as a system of differential equations, with distinct compartments. We employ fractional order derivatives to capture the long-term memory and non-local interactions inherent in HBV transmission, offering a more realistic representation of the epidemic dynamics. To assess the stability and control potential of the model, we conduct rigorous mathematical analysis. The basic reproduction number is computed using the next generation matrix approach to determine the disease's potential for spreading in the population. Critical points of the model are identified, and disease-free equilibrium points are obtained to assess their stability conditions. Furthermore, we derive endemic equilibrium points for the model, and their stability is analyzed using Jacobian transformation.To optimize control measures, sensitivity analysis of the model parameters is performed to identify influential factors affecting disease transmission. Numerical simulations of the fractional order model are implemented using the Adams-type Predictor-Corrector method, and the results demonstrate the effectiveness of the proposed control strategies in curbing the spread of HBV.

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1. INTRODUCTION

The hepatitis B virus causes hepatitis B, a potentially fatal liver infection. It is a significant issue for world health. It can result in chronic liver disease, chronic infection, and high mortality rates from liver cancer and cirrhosis [1]. Hepatitis B infections can only happen if the virus can get into the bloodstream and reach the liver. Once inside the liver, the virus multiplies and sends out a lot of fresh viruses into the bloodstream [2].

It has two stages of infection namely: Acute and chronic, According to the World Health Organization, the Hepatitis B virus is actively infecting more than one-third of the world's population. Additionally, more than 350 million of them have ongoing infections, and regrettably, 25 to 40 percent of them pass away from primary hepatocellular carcinoma (a form of liver cancer characterized by abnormal, dangerous growth(s) in the liver) or liver cirrhosis (scarring of the liver).[3]. Hepatitis B is the tenth leading cause of death worldwide . Hepatocellular cancer (HCC) is the third most common cause of cancer death worldwide since it accounts for more than five hundred per year [4].

To improve knowledge of the pathophysiology (creation and progression) of hepatitis B infection, Long and Qi, in 2008 [5] suggested mathematical models. Based on Nowak's population dynamics model of immune response to persistent viruses, their work uses mathematical equations to describe the interaction between HBV and the immunological response to the virus. Uninfected hepatocytes, infected hepatocytes, total host hepatocytes, free virus, and a CTL (Cytotoxic T lymphocyte utilized to kill virally infected or malignant cells) reaction are the five variables that make up the suggested model. Tahir khan et al in 2021 [6] looked into and evaluated the dynamics of hepatitis B, which has a number of different infection phases and transmission channels. The Caputo-Fabrizio operator and the idea of fractional calculus were used to fractionalize the model. The fixed point theory was used to discuss an extensive investigation of existence and uniqueness. To assist the analytical work with the aid of graphical representations, several numerical findings were made. Peijiang Liu et al in 2022 [7] formulated

a five compartmental model of hepatitis B model using the fractional Calculus in the Caputo sense and detailed analysis of the model was carried out. The result obtained also showed that the fractional model is best suited to model the viral infection than the classical model. Elif Demirci in 2022 [7] presented a fractional order mathematical model to explain the spread of Hepatitis B Virus (HBV) in a non-constant population. The model included both vertical and horizontal transmission of the infection and also vaccination at birth and vaccination of the susceptible class. A frequency dependent transmission rate was used. Numerical simulations of the model are presented. The approach presented in this paper differ from those presented and references therein. We present a fractional order $SVEI_1RI_2T$ (Susceptible-Exposed-Infected-Removed-Treated) model to discuss the dynamics of Hepatitis B and also show the impact of vaccination/Treatment on the population.

This paper is organized as follows: A brief review of the fractional calculus is presented in section 2 with definitions. Section 3 discusses fractional order models while section 4 presents model analysis involving equilibrium points and stability. Section 5 is devoted to numerical simulations and discussion of results. Section 6 gives the concluding remarks.

2. Fractional Order Calculus

The concept of fractional order calculus is as old as the concept of integer order calculus. The complexity and lack of application delayed its advancement until a few decades ago. Although there has been a considerable amount of work done in simulating the dynamics of epidemic diseases, it has been limited to integer-order differential equations. Most dynamical systems based on integer order calculus have recently been changed into fractional order due to the flexibility that can be used to precisely fit the experimental data much better than integer order modeling. [8].Because the fractional derivative is a generalization of the integer-order derivative, fractional modeling is an effective approach that has been used to investigate the behavior of diseases. Most vaccination models are based on ordinary differential equations (ODEs), however we characterize the behavior of these systems using fractional order differential equations in this work. The fractional derivative is defined in various ways. Gruwald-Letnikov, Riemann-Liouville, and Caputo's fractional derivatives have been employed more frequently than others, but they are not always equal. [9]. Comparing these three fractional derivatives, it is a fact that

Caputo's derivative of a constant is equal to zero, which is not true for the Riemann-Liouville derivative. The main advantage of Caputo's approach is that the initial conditions for fractional differential equations with Caputo derivatives take on the same form as for integer-order differential equations. Having this in mind, we restrict our attention to the Caputo derivative of order $\alpha > 0$, which is rather applicable to real world. For the purpose of this research work, we now gather some well-known definitions.

2.1. Definition of terms [9].

Definition 1. The Caputo Fractional derivative of order α of a function $f : \Re^+ \to \Re$ is given by

$$D_t^\alpha f(t) = \frac{1}{\Gamma(\alpha - n)} \int_\alpha^t \frac{f^{(n)}(\tau) d\tau}{(t-\tau)^{\alpha+1-n}} \qquad (n-1 < \alpha \leq n) \tag{2.1}$$

Definition 2. The formula for the Laplace transform of the Caputo derivative is given by

$$\int_0^\infty e^{-pt} \{D_t^\alpha f(t)\} dt = p^\alpha F(p) - \sum_{k=0}^{n-1} p^{\alpha-k-1} f^{(k)}(0), \qquad (n-1 < \alpha \leq n) \tag{2.2}$$

Definition 3. The Fractional integral of order α of a function $f : \Re^+ \to \Re$ is given by

$$J^\alpha (f(x)) = \frac{1}{\Gamma(\alpha)} \int_0^x (x-t)^{\alpha-1} f(t) dt, \qquad \alpha > 0, x > 0 \tag{2.3}$$

Definition 4. The fractional integral of the Caputo Fractional derivative of order α of a function $f : \Re^+ \to \Re$ is given by

$$J^\alpha \{D^\alpha f(t)\} = f(t) - \sum_{k=0}^{n-1} f^{(k)}(0) \frac{t^k}{k!}, \qquad t > 0 \tag{2.4}$$

Definition 5. A two-parameter funvtion of the Mittag-Leffler type is defined by the series expansion

$$E_{\alpha,\beta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + \beta)}, \qquad (\alpha, \beta > 0) \tag{2.5}$$

3. Model Assumptions and Formulation

The $SVEI_1RI_2T$ model is based on the following assumptions:

(1) The only way of entry into the population is through birth and the only way of exit is through death from natural causes or death from Hepatitis B-related causes.

(2) The the population mixes homogeneously. That is all individuals are equally likely to be infected by the infectious individuals in a case of contact except those who are Vaccinated or Removed.

(3) Any individual who recovers completely from the disease or who has been vaccinated receives a lifelong immunity from the disease.

(4) The proportion of people that moves from the susceptible to the removed class directly is assumed to have received the required three doses of Hepatitis B vaccine.

(5) That first dose of vaccine does not confer lifelong immunity.

(6) The treated class consists of people who are undergoing treatment to remain stable

(7) The rate at which people die of the disease in the treated class is lesser than the rate at which people die of the disease in the chronically infectious class (that is $\delta_1 < \delta_2$).

(8) The population in the treatment compartment will not recover from the illness.

(9) The acutely infectious individuals do not undergo any form of treatment but recover naturally from the ailment.

(10) The population undergoing treatment can still die as a result of the disease.

The flow diagram of the model is as follows:

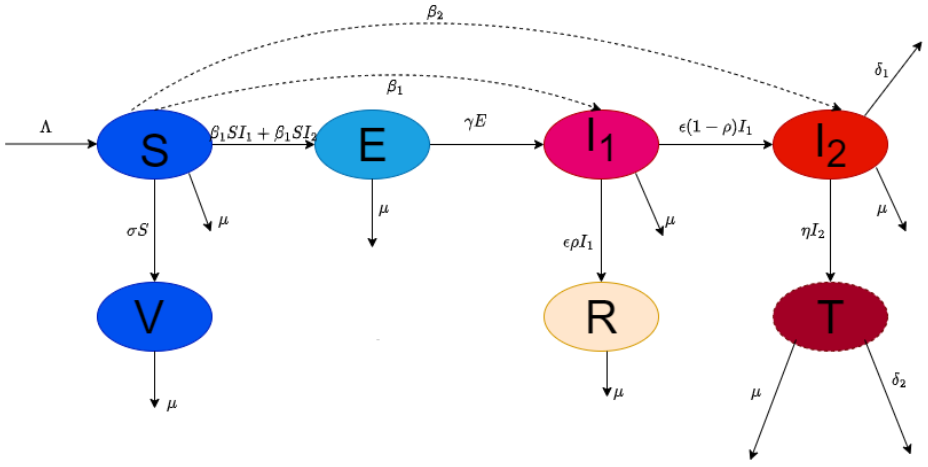

FIGURE 1. Flow chart

Taking into account the above descriptions and assumptions, the Fractional $SVEI_1RI_2T$ model is described by

$$\begin{cases} D^{\alpha}S(t) = \Lambda - \beta_1 SI_1 - \beta_2 SI_2 - \sigma S - \mu S \\ D^{\alpha}V(t) = \sigma S - \mu V \\ D^{\alpha}E(t) = \beta_1 SI_1 + \beta_2 SI_2 - \gamma E - \mu E \\ D^{\alpha}I_1(t) = \gamma E - \varepsilon\rho I_1 - \varepsilon(1-\rho)I_1 - \mu I_1 \\ D^{\alpha}R(t) = \varepsilon\rho I_1 - \mu R \\ D^{\alpha}I_2(t) = \varepsilon(1-\rho)I_1 - \eta I_2 - \delta_1 I_2 - \mu I_2 \\ D^{\alpha}T(t) = \eta I_2 - \delta_2 T - \mu T \end{cases} \tag{3.6}$$

By setting $\alpha = 1$, the system of equation 3.6 can be reduced to integer order system.

With the non-negative initial condition:

$S(0) = S_0, V(0) = V_0, E(0) = E_0, I_1(0) = I_{10}, I_2(0) = I_{20}, R(0) = R_0, T(0) = T_0$

3.1. Invariant region and the positivity of the model solutions.

Lemma 3.1. (Generalized Mean Value Theorem[10])

Suppose that $g(t)\,\varepsilon\, C[a,b]$ and $D^{\alpha}g(t)\,\varepsilon\, C(a,b]$ for $0 < \alpha \leq 1$, then

$$g(t) = g(a) + \frac{1}{\Gamma(\alpha)} D^{\alpha}g(\xi)(t-a)^{\alpha}, \tag{3.7}$$

where $a \leq \xi \leq t, \forall\, t\, \varepsilon\, (a,b]$.

Remark 3.1. Assume that $g(t)\,\varepsilon\, C[a,b]$ and $D^{\alpha}g(t)\,\varepsilon\, C(a,b]$ for $0 < \alpha \leq 1$. It follows from the lemma 3.1 that if $D^{\alpha}g(t) \geq 0$, $\forall\, t\, \varepsilon\, (a,b)$, then g(t) is non-decreasing $\forall\, t\, \varepsilon\, (a,b]$, and if $D^{\alpha}g(t) \geq 0$, $\forall\, t\, \varepsilon\, (a,b]$, then g(t) is non-increasing $\forall\, t\, \varepsilon\, [a,b]$.

Theorem 3.1. The closed set $\Omega = \{(S,V,E,I_1,I_2,R,T) \in R_+^7 : S+V+E+I_1+I_2+R+T \leq \frac{\Lambda}{\mu}\}$ is positively invariant with respect to model (3.6).

Proof. The fractional derivative of the total human population, obtained by adding all the human equations of model 3.6, is given by

$$D^{\alpha}N(t) = \Lambda - \mu N(t) \tag{3.8}$$

TABLE 1. Description of parameters and variables for model (3.6)

Variables	Description	Unit
S	Susceptible human population	people
V	Vaccinated human population	people
E	Exposed human population	people
I_1	Acutely infected human population	people
R	Recovered human poulation	people
I_2	Chronically infected human population	people
T	Human population under treatment	people
Parameters	**Description**	**Unit**
Λ	Recruitment rate	day^{-1}
σ	Vaccination rate	day^{-1}
β_1	Interaction rate between the Susceptible and the Acutely Infected population	day^{-1}
β_2	Interaction rate between the Susceptible and the Chronically Infected population	day^{-1}
γ	Progression rate from exposed class to Acutely infected class	day^{-1}
$\varepsilon\rho$	Proportion of Acutely infected population	day^{-1}
$\varepsilon(1-\rho)$	Proportion moving from the Acutely infected to the Chronically Infected	day^{-1}
δ_1	Death rate as a result of the Infection in the Chronically infected class	day^{-1}
η	Progression rate from the chronically Infected to the Treatment Class	day^{-1}
δ_2	Death rate as a result of the Infection in the treatment infected class	day^{-1}
μ	Natural death rate	day^{-1}

Taking the Laplace transform of (3.8) gives:

$$S^{\alpha}N(s) - S^{\alpha-1}N(0) = \frac{\Lambda}{S} - \mu N(s)$$

$$\Rightarrow N(s) = \frac{\Lambda}{S(S^{\alpha}+\mu)} + \frac{S^{\alpha-1}}{s^{\alpha}+\mu}N(0) \tag{3.9}$$

Taking the inverse Laplace transform of (3.9), we have:

$$N(t) = N(0)E_{\alpha,1}(-\mu t^{\alpha}) + \Lambda t^{\alpha}E_{\alpha,\alpha+1}(-\mu t^{\alpha}) \tag{3.10}$$

where $E_{\alpha,\beta}$ is the Mittag-Leffler function. But the fact that the Mittag-Leffler functions has an asymptotic behavior [9, 11], it follows that:

$$E_{\alpha,1}N(t) = \sum_{k=0}^{\infty} \frac{N^K(t)}{\Gamma(\alpha k+1)}, \alpha > 0 \tag{3.11}$$

$$E_{\alpha,\alpha+1}N(t) = \sum_{k=0}^{\infty} \frac{N^K(t)}{\Gamma(\alpha k+\alpha+1)}, \alpha > 0 \tag{3.12}$$

Expanding (1.6), we have

$$E_{\alpha,1}N(t) = \frac{1}{\Gamma 1} + \frac{N(t)}{\Gamma(\alpha+1)} + \frac{N^2(t)}{\Gamma(2\alpha+1)} + ...$$

Expanding (1.7), we have

$$E_{\alpha,\alpha+1}N(t) = \frac{1}{\Gamma(\alpha+1)} + \frac{N(t)}{\Gamma(2\alpha+1)} + \frac{N^2(t)}{\Gamma(3\alpha+1)} + ...$$

Since Mittag-Leffler function has an asymptotic property, we have

$$N(t) = 1 + O(N)$$

Taking limit as k$\longrightarrow \infty$, we have

$$N(t) \approx 1$$

Then, it is clear that Ω is a positive invariant set.Therefore, all solutions of the model with initial conditions in Ω remain in Ω for all t > 0. Then , $\Omega = N(t) > 0$ implies that it is feasible with respect to model (3.6).

4. Model Analysis

4.0.1. *The basic reproduction number R_0.* The basic reproduction number R_O is used in the study of disease transmission and control (epidemiology) to describe the average number of secondary infections caused by the introduction of one infectious person into a totally susceptible population. It has the following implications:

- If $R_0 < 1$, the the Disease free equilibrium is LAS (Locally asymptotically stable) and the disease cannot invade the population.
- If $R_(0) > 1$, implies that the DFE (Diseases free equilibrium) is unstable and invasion is possible.

Diekmann et all and Van Driessche et al [12, 13] provided a method for calculating R_0, which is the formation of the next-generation matrix. It is comprised of two parts: F and $V^{-}1$, where

$$F = \left| \frac{\partial f_i x_{(0)}}{\partial x_j} \right| , \qquad V = \left| \frac{\partial v_i x_{(0)}}{\partial x_j} \right|$$

The F_i are the new infections, while the V_i are transfer of infection from one compartment to another. x_0 is the disease free equilibrium point. R_0 is the spectral radius of the next generation matrix, which is the dominant Eigenvalue of the same matrix. To calculate this, we consider the infected compartments $E(t), I_1(t), I_2(t)$, and $T(t)$.

$$\begin{aligned} D^{\alpha}E(t) &= \beta_1 SI_1 + \beta_2 SI_2 - \gamma E - \mu E \\ D^{\alpha}I_1(t) &= \gamma E - \varepsilon\rho I_1 - \varepsilon(1-\rho)I_1 - \mu I_1 \\ D^{\alpha}I_2(t) &= \varepsilon(1-\rho)I_1 - \eta I_2 - \delta_1 I_2 - \mu I_2 \\ D^{\alpha}T(t) &= \eta I_2 - \delta_2 T - \mu T \end{aligned}$$

Define:

$$F_i = \begin{pmatrix} \beta_1 SI_1 + \beta_2 SI_2 \\ 0 \\ 0 \\ 0 \end{pmatrix}$$

$$V_i = \begin{pmatrix} -(\gamma+\mu)\mathrm{E} \\ -\gamma E - (\varepsilon\rho + \varepsilon(1-\rho) + \mu)I_1 \\ \varepsilon(1-\rho)I_1 - (\eta + \delta_1 + \mu)I_2 \\ \eta I_2 - (\delta_2 + \mu)T \end{pmatrix}$$

The Jacobian Matrices of F and V at DFE are given as

$$F = \begin{pmatrix} 0 & \beta_1 SI_1 + \beta_2 SI_2 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 00 & 0 & 0 & 0 \end{pmatrix}$$

$$-V = \begin{pmatrix} -(\gamma+\mu) & 0 & 0 & 0 \\ -\gamma & -(\varepsilon\rho+\varepsilon(1-\rho)+\mu) & 0 & 0 \\ 0 & \varepsilon(1-\rho) & -(\eta+\delta_1+\mu) & 0 \\ 0 & 0 & \eta & -(\delta_2+\mu)T \end{pmatrix}$$

Therefore, the dominant Eigenvalue of FV^{-1} is given as:

$$R_0 = \frac{\Lambda\gamma}{(\sigma+\mu)(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)}\left(\beta_1 + \frac{\beta_2\varepsilon(1-\rho)}{(\eta+\delta_1+\mu)}\right) \tag{4.13}$$

4.1. Equilibrium points and their stabilities.

4.1.1. *Disease-free equlibrium point.* The coordinates of an equilibrium (S,V,E,I_1,I_2,R,T) of system (3.6) satisfy the following equations:

$$\begin{cases} 0 = \Lambda - \beta_1 SI_1 - \beta_2 SI_2 - \sigma S - \mu S \\ 0 = \sigma S - \mu V \\ 0 = \beta_1 SI_1 + \beta_2 SI_2 - \gamma E - \mu E \\ 0 = \gamma E - \varepsilon\rho I_1 - \varepsilon(1-\rho)I_1 - \mu I_1 \\ 0 = \varepsilon\rho I_1 - \mu R \\ 0 = \varepsilon(1-\rho)I_1 - \eta I_2 - \delta_1 I_2 - \mu I_2 \\ 0 = \eta I_2 - \delta_2 T - \mu T \end{cases} \tag{4.14}$$

The disease-free equilibrium $P^0 = (\frac{\Lambda}{\sigma+\mu}, \frac{\sigma\Lambda}{\mu(\sigma+\mu)}, 0, 0, 0, 0, 0)$ alyways exists.

4.1.2. *Local Stability of the Disease-free equilibrium point P_0.* In the previous section, we have seen that the basic reproduction number serves as a threshold parameter in determining the number of equilibria in system (3.6). We will show in this section that R_0 also determines the local stability of the equilibria.

Theorem 4.1. The disease-free equilibrium $P^0 = (\frac{\Lambda}{\sigma+\mu}, \frac{\sigma\Lambda}{\mu(\sigma+\mu)}, 0, 0, 0, 0, 0)$ is locally asymptotically stable if $R_0 < 1$.

Proof. We shall apply the method of linearization. The Jacobian matrix of system 3.6 at $P^0 = (\frac{\Lambda}{\sigma+\mu}, \frac{\sigma\Lambda}{\mu(\sigma+\mu)}, 0, 0, 0, 0, 0)$ is given as:

$$J^0 = \begin{pmatrix} -\sigma-\mu & 0 & 0 & -\beta_1\frac{\Lambda}{(\sigma+\mu)} & 0 & -\beta_2\frac{\Lambda}{(\sigma+\mu)} & 0 \\ -\sigma & -\mu & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -\gamma-\mu & \beta_1\frac{\Lambda}{(\sigma+\mu)} & 0 & \beta_2\frac{\Lambda}{(\sigma+\mu)} & 0 \\ 0 & 0 & \gamma & -\varepsilon\rho-\varepsilon(1-\rho)-\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & \varepsilon\rho & -\mu & 0 & 0 \\ 0 & 0 & 0 & \varepsilon(1-\rho) & 0 & -\eta-\delta_1-\mu & 0 \\ 0 & 0 & 0 & 0 & 0 & \eta & -\mu-\delta_2 \end{pmatrix}$$

Next we find the characteristic equation which is given by$|J^0 - \lambda I| = 0$, where λ is the eigenvalue.

$|J^0 - \lambda I| =$

$$\begin{vmatrix} -(\sigma+\mu)-\lambda & 0 & 0 & -\beta_1\frac{\Lambda}{(\sigma+\mu)} & 0 & -\beta_2\frac{\Lambda}{(\sigma+\mu)} & 0 \\ -\sigma & -\mu-\lambda & 0 & 0 & 0 & 0 & 0 \\ 0 & 0 & -(\gamma+\mu)-\lambda & \beta_1\frac{\Lambda}{(\sigma+\mu)} & 0 & \beta_2\frac{\Lambda}{(\sigma+\mu)} & 0 \\ 0 & 0 & \gamma & -(\varepsilon\rho+\varepsilon(1-\rho)+\mu)-\lambda & 0 & 0 & 0 \\ 0 & 0 & 0 & \varepsilon\rho & -\mu-\lambda & 0 & 0 \\ 0 & 0 & 0 & \varepsilon(1-\rho) & 0 & -(\eta+\delta_1+\mu)-\lambda & 0 \\ 0 & 0 & 0 & 0 & 0 & \eta & -(\mu+\delta_2)-\lambda \end{vmatrix}$$

Hence,

$$\lambda_1 = -(\mu+\delta_2), \lambda_2 = -\mu, \lambda_3 = -\mu, \lambda_4 = -(\sigma+\mu)$$

and matrix reduces to:

$$\begin{vmatrix} -(\gamma+\mu)-\lambda & \beta_1\frac{\Lambda}{(\sigma+\mu)} & \beta_2\frac{\Lambda}{(\sigma+\mu)} \\ \gamma & -(\varepsilon\rho+\varepsilon(1-\rho)+\mu)-\lambda & 0 \\ 0 & \varepsilon(1-\rho) & -(\eta+\delta_1+\mu)-\lambda \end{vmatrix}$$

The characteristics cubic equation is given as:

$$\lambda^3 + a_1\lambda^2 + a_2\lambda + a_0 = 0$$

where

$$a_1 = \gamma+\mu+\varepsilon\rho+\varepsilon(1-\rho)+\mu+\eta+\delta_1+\mu$$

$$a_2 = (\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho))+(\gamma+\mu)(\eta+\delta_1+\mu)+(\varepsilon\rho$$
$$+\varepsilon(1-\rho))(\eta+\delta_1+\mu)+(\frac{\beta_1\Lambda\gamma}{\sigma\mu})$$

$$a_3 = \frac{-1}{(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)(\eta+\delta_1+\mu)}(R_0-1)$$

Hence, a_3 is positive when $R_0 < 1$. By Routh-Hourwitz criterion [1], the Eigenvalues have negative real parts. Therefore, the disease-free equilibrium is locally asymptotically stable if $R_0 < 1$.

4.1.3. *The Global Stability of the Disease-Free Equilibrium.* The model equation is given as:

$$\begin{cases} {}^cD^{\alpha}_{0+}S(t) = \Lambda-\beta_1 SI_1-\beta_2 SI_2-\sigma S-\mu S \\ {}^cD^{\alpha}_{0+}V(t) = \sigma S-\mu V \\ {}^cD^{\alpha}_{0+}E(t) = \beta_1 SI_1+\beta_2 SI_2-\gamma E-\mu E \\ {}^cD^{\alpha}_{0+}I_1(t) = \gamma E-\varepsilon\rho I_1-\varepsilon(1-\rho)I_1-\mu I_1 \\ {}^vD^{\alpha}_{0+}R(t) = \varepsilon\rho I_1-\mu R \\ {}^cD^{\alpha}_{0+}I_2(t) = \varepsilon(1-\rho)I_1-\eta I_2-\delta_1 I_2-\mu I_2 \\ {}^cD^{\alpha}_{0+}T(t) = \eta I_2-\delta_2 T-\mu T \end{cases}$$

The disease-free equilibrium of the model is

$$P^0 = (S^0,V^0,E^0,I_1^0,R^0,I_2^0,T^0) = (\frac{\Lambda}{\sigma+\mu},\frac{\sigma\Lambda}{\mu(\sigma+\mu)},0,0,0,0,0)$$

Following the notation from theorem 2 [14], we have

$$A = \begin{bmatrix} -(\sigma+\mu) & -\beta_1\frac{\Lambda}{(\sigma+\mu)} & -\beta_2\frac{\Lambda}{(\sigma+\mu)} & 0 \\ \gamma & -(\varepsilon\rho+\varepsilon(1-\rho)+\mu) & 0 & 0 \\ 0 & \varepsilon(1-\rho) & -(\eta+\delta_1+\mu) & 0 \\ 0 & 0 & \eta & -(\mu+\delta_2) \end{bmatrix}$$

The matrix A can be written as $A = M - D$ with

$$M = \begin{bmatrix} 0 & -\beta_1 \frac{\Lambda}{(\sigma+\mu)} & -\beta_2 \frac{\Lambda}{(\sigma+\mu)} & 0 \\ \gamma & 0 & 0 & 0 \\ 0 & \varepsilon(1-\rho) & 0 & 0 \\ 0 & 0 & \eta & 0 \end{bmatrix}$$

and

$$D = \begin{bmatrix} -(\sigma+\mu) & 0 & 0 & 0 \\ 0 & -(\varepsilon\rho + \varepsilon(1-\rho) + \mu) & 0 & 0 \\ 0 & 0 & -(\eta + \delta_1 + \mu) & 0 \\ 0 & 0 & 0 & -(\mu + \delta_2) \end{bmatrix}$$

The point $P^0 = (\frac{\Lambda}{\sigma+\mu}, \frac{\sigma\Lambda}{\mu(\sigma+\mu)}, 0)$ is globally asymptotically stable for the system of uninfected individuals:

$$\begin{cases} {}^cD^{\alpha}_{0+} S(t) = \Lambda - \sigma S - \mu S \\ {}^cD^{\alpha}_{0+} V(t) = \sigma S - \mu V \\ {}^vD^{\alpha}_{0+} R(t) = -\mu R \end{cases}$$

It is easy to show that

$$R(t) = R_0 E_{\alpha}(-\mu t^{\alpha})$$

satisfies the third equation. Also,

$$V(t) = V_0 E_{\alpha}(-\mu t^{\alpha}) + \frac{\Lambda\sigma}{\mu(\sigma+\mu)}(1 - E_{\alpha}(-\mu t^{\alpha}))$$

satisfies the second equation. The solution of equation one is given as

$$S(t) = S_0 E_{\alpha}(-(\sigma+\mu)t^{\alpha}) - \Lambda E_{\alpha}(-(\sigma+\mu)t^{\alpha}) + \frac{\Lambda}{(\sigma+\mu)}$$

Hence,

$$(S(t), V(t), R(t)) \to (\frac{\Lambda}{\sigma+\mu}, \frac{\sigma\Lambda}{\mu(\sigma+\mu)}, 0) \, as t \to \infty$$

In addition, by Lemma 4[14], $E_{\alpha\alpha}$ is nonnegative and so, by Theorem 2 [14], the disease-free equilibrium of the model (3.6) is globally asymptotically stable.

4.1.4. *Endemic Equilibrium Point.* The endemic equilibrium point

$P^* = (S^*, V^*, E^*, I_1^*, R^*, I_2^*, T^*)$ with $S^*, V^*, E^*, I_1^*, R^*, I_2^*, T^* > 0$ satisfies

$$S^* = \frac{(\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\gamma+\mu)(\eta+\delta_1+\mu)}{\gamma\beta_2\varepsilon(1-\rho)+\gamma\beta_1(\eta+\delta_1+\mu)}$$

$$V^* = \frac{\sigma}{\mu}\left(\frac{(\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\gamma+\mu)(\eta+\delta_1+\mu)}{\gamma\beta_2\varepsilon(1-\rho)+\gamma\beta_1(\eta+\delta_1+\mu)}\right)$$

$$E^* = \frac{\varepsilon\rho+\varepsilon(1-\rho)+\mu}{\gamma}\left(\frac{\Lambda\gamma}{\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\gamma+\mu)(\eta+\delta_1+\mu)} - \frac{(\sigma+\mu)(\eta+\delta_1+\mu)}{\beta_1(\eta+\delta_1+\mu)+\beta_2\varepsilon(1-\rho)}\right)$$

$$I_1^* = \frac{\Lambda\gamma}{\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\gamma+\mu)(\eta+\delta_1+\mu)} - \frac{(\sigma+\mu)(\eta+\delta_1+\mu)}{\beta_1(\eta+\delta_1+\mu)+\beta_2\varepsilon(1-\rho)}$$

$$I_2^* = \frac{\varepsilon(1-\rho)}{(\eta+\delta_1+\mu)}\left(\frac{\Lambda\gamma}{\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\gamma+\mu)(\eta+\delta_1+\mu)} - \frac{(\sigma+\mu)(\eta+\delta_1+\mu)}{\beta_1(\eta+\delta_1+\mu)+\beta_2\varepsilon(1-\rho)}\right)$$

$$R^* = \frac{\varepsilon\rho}{\mu}\left(\frac{\Lambda\gamma}{\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\gamma+\mu)(\eta+\delta_1+\mu)} - \frac{(\sigma+\mu)(\eta+\delta_1+\mu)}{\beta_1(\eta+\delta_1+\mu)+\beta_2\varepsilon(1-\rho)}\right)$$

$$T^* = \frac{\eta}{(\delta_2+\mu)}\left(\frac{\varepsilon(1-\rho)}{(\eta+\delta_1+\mu)}\left(\frac{\Lambda\gamma}{\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\gamma+\mu)(\eta+\delta_1+\mu)} - \frac{(\sigma+\mu)(\eta+\delta_1+\mu)}{\beta_1(\eta+\delta_1+\mu)+\beta_2\varepsilon(1-\rho)}\right)\right)$$

4.1.5. *Local Stability of the Endemic Equilibrium when $R_0 > 1$.* The Jacobian Matrix of the model 3.6 is given as

$$J = \begin{pmatrix} -\sigma-\beta_1 I_1-\beta_2 I_2\mu & 0 & 0 & -\beta_1 S & 0 & -\beta_2 S & 0 \\ -\sigma & -\mu & 0 & 0 & 0 & 0 & 0 \\ \beta_1 I_1+\beta_2 I_2 & 0 & -\gamma-\mu & \beta_1 S & 0 & \beta_2 S & 0 \\ 0 & 0 & \gamma & -\varepsilon\rho-\varepsilon(1-\rho)-\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & \varepsilon\rho & -\mu & 0 & 0 \\ 0 & 0 & 0 & \varepsilon(1-\rho) & 0 & -\eta-\delta_1-\mu & 0 \\ 0 & 0 & 0 & 0 & 0 & \eta & -\mu-\delta_2 \end{pmatrix}$$

The Jocobian Matrix of the Endemic Equilibrium point is

$$J(P^*) = \begin{pmatrix} -\sigma-\beta_1 I_1^*-\beta_2 I_2^*-\mu & 0 & 0 & -\beta_1 S & 0 & -\beta_2 S^* & 0 \\ -\sigma & -\mu & 0 & 0 & 0 & 0 & 0 \\ \beta_1 I_1^*+\beta_2 I_2^* & 0 & -\gamma-\mu & \beta_1 S^* & 0 & \beta_2 S^* & 0 \\ 0 & 0 & \gamma & -\varepsilon\rho-\varepsilon(1-\rho)-\mu & 0 & 0 & 0 \\ 0 & 0 & 0 & \varepsilon\rho & -\mu & 0 & 0 \\ 0 & 0 & 0 & \varepsilon(1-\rho) & 0 & -\eta-\delta_1-\mu & 0 \\ 0 & 0 & 0 & 0 & 0 & \eta & -\mu-\delta_2 \end{pmatrix}$$

Hence, $\lambda_{1,2} = -\mu, \lambda_3 = -(\delta_2+\mu)$ and the matrix reduces to

$$|J(P^*)-\lambda I| = \begin{vmatrix} -\sigma-\beta_1 I_1^*-\beta_2 I_2^*-\mu-\lambda & 0 & -\beta_1 S & -\beta_2 S^* \\ \beta_1 I_1^*+\beta_2 I_2^* & -\gamma-\mu-\lambda & \beta_1 S^* & \beta_2 S^* \\ 0 & \gamma & -\varepsilon\rho-\varepsilon(1-\rho)-\mu-\lambda & 0 \\ 0 & 0 & \varepsilon(1-\rho) & -\eta-\delta_1-\mu-\lambda \end{vmatrix}$$

For simplicity, let $a = \sigma + \beta_1 I_1^* + \beta_2 I_2^* + \mu$, $b = \beta_1 S$, $c = \beta_2 S$, $d = \beta_1 I_1^* + \beta_2 I_2^*$, $e = \gamma + \mu$, $f = \varepsilon\rho + \varepsilon(1-\rho) + \mu$,$g = \varepsilon(1-\rho)$,$h = \eta + \delta_1 + \mu$.

Therefore, we have

$$|J(P^*) - \lambda I| = \begin{pmatrix} -a-\lambda & 0 & -b & -c \\ d & -e-\lambda & b & c \\ 0 & \gamma & -f-\lambda & 0 \\ 0 & 0 & g & -h-\lambda \end{pmatrix}$$

The characteristics equation becomes

$$-(a+\lambda)[-(e+\lambda)(f+\lambda)(h+\lambda) + b\gamma(h+\lambda) + \gamma cg] + \gamma b(h+\lambda)d + \gamma cgd = 0$$

Further simplification yields

$$\lambda^4 + (e+f+h+a)\lambda^3 + (ef+eh+fh+ae+af+ac-b\gamma)\lambda^2$$
$$+ (aeh+afh+efh+aef+b\gamma d - b\gamma h - c\gamma g - b\gamma a)\lambda$$
$$+ aefh + bdh\gamma + cdg\gamma - bhg\gamma - acg\gamma = 0$$

If the coefficients are given as

$$a_3 = (e+f+h+a)$$

$$a_2 = (ef+eh+fh+ae+af+ac-b\gamma)$$

$$a_1 = (aeh+afh+efh+aef+b\gamma d - b\gamma h - c\gamma g - b\gamma a)$$

$$a_0 = aefh + bdh\gamma + cdg\gamma - bhg\gamma - acg\gamma$$

According to Routh Hurwitz's criterion [1], all the roots of the equation will be less than zero if the following conditions are met:

- If all the coefficients and the constant term are greater than zero
- If $a_3a_2 > a_1$ and $a_1 > \frac{a_3^2 a_0}{a_3a_2 - a_1}$

Then it follows that all the eigenvalues satisfy the condition $|arg(\lambda)| > \frac{\alpha\pi}{2}$ [1].

4.1.6. *Global Stability of the Endemic Equilibrium Point when $R_0 > 1$.*

Theorem 4.2. Suppose that $R_0 > 1$. Then the endemic equilibrium point P^* is globally asymptotically stable in the interior of Ω.

Proof. To prove the global stability of P^*, we use the Volterra type Lyapunov function approach [15].

Let $P^* = (S^*, V^*, E^*, I_1^*, R^*, I_2^*, T^*)$ be the endemic equilibrium.

Consider the function

$V(S,V,E,I_1,R,I_2,T) = (S-S^*) - S^* log\frac{S}{S^*} + (V-V^*) - V^* log\frac{V}{V^*} + (E-E^*) - E^* log\frac{E}{E^*} + (I_1 - I_1^*) - I_1^* log\frac{I_1}{I_1^*} + (R-R^*) - R^* log\frac{R}{R^*} + (I_2 - I_2^*) - I_2^* log\frac{I_2}{I_2^*} + (T-T^*) - T^* log\frac{T}{T^*}$

We first show that $V(S,V,E,I_1,R,I_2,T) > 0$ in the interior of Ω and $V(S,V,E,I_1,R,I_2,T)=0$ only at P^*. For $y^* > 0$, let $f(y) = y - y^* - y^* log\frac{y}{y^*}$. Then $f(y^*) = 0$ and $f'(y) = 1 - \frac{y^*}{y}$. Therefore, $f'(y) < 0$ if $y < y^*$ and $f'(y) > 0$ if $y > y^*$. This means that $f(y)$ has an absolute minimum 0 at $y = y^*$ in the interval $(0,\infty)$. This property tells us that $V(S,V,E,I_1,R,I_2,T)$ is positive definite with respect to point P^*.

The Lyapunov derivative of V along solutions of (3.6) is

$\dot{V} = S' - \frac{S^*}{S}S' + V' - \frac{V^*}{V}V' + E' - \frac{E^*}{E}E' + I_1' - \frac{I_1^*}{I_1}I_1' + R' - \frac{R^*}{R}R' + I_2' - \frac{I_2^*}{I_2}I_2' + T' - \frac{T^*}{T}T'.$

Now, set $\dot{V}_1 = S' - \frac{S^*}{S}S'$, $\dot{V}_1 = V' - \frac{V^*}{V}V', \dot{V}_3 = E' - \frac{E^*}{E}E', \dot{V}_4 = I_1' - \frac{I_1^*}{I_1}I_1', \dot{V}_5 = R' - \frac{R^*}{R}R', \dot{V}_6 = I_2' - \frac{I_2^*}{I_2}I_2', \dot{V}_7 = T' - \frac{T^*}{T}T'.$

Therefore,

$$\begin{aligned}
\dot{V}_1 &= \frac{S-S^*}{S}S' \\
&= \frac{S-S^*}{S}(\beta_1 S^* I^* + \beta_2 S^* I_2^* + (\sigma+\mu)S^* - (\beta_1 S I_1 + \beta_2 S I_2 + (\sigma+\mu)S) \\
&= -(\sigma+\mu)\frac{(S-S^*)^2}{S} + \beta_1 S^* I_1^*(1 - \frac{S^*}{S} - \frac{SI_1}{S^* I_1^*} + \frac{I_1}{I_1^*}) \\
&+ \beta_2 S^* I_2^*(1 - \frac{S^*}{S} - \frac{SI_2}{S^* I_2^*} + \frac{I_2}{I_2^*}) \\
&\leq \beta_1 S^* I_1^*(1 - \frac{S^*}{S} - \frac{SI_1}{S^* I_1^*} + \frac{I_1}{I_1^*}) + \beta_2 S^* I_2^*(1 - \frac{S^*}{S} - \frac{SI_2}{S^* I_2^*} + \frac{I_2}{I_2^*})
\end{aligned}$$

Similarly, $\dot{V}_2 \leq \mu V^*(2 - \frac{V}{V^*} - \frac{V^*}{V})$

$\dot{V}_3 \leq \beta_1 S^* I_1^*(1 + \frac{SI_1}{S^* I^*} - \frac{E}{E^*} - \frac{E^* SI_1}{ES^* I_1^*}) + \beta_2 S^* I_2^*(1 + \frac{SI_2}{S^* I_2^*} - \frac{E}{E^*} - \frac{E^* SI_2}{ES^* I_2^*})$

$\dot{V}_4 \leq \gamma E^*(1+\frac{E}{E^*}-\frac{I_1^*E}{I_1E^*}-\frac{I_1}{I_1^*})$
$\dot{V}_5 \leq \mu R^*(1+\frac{I_1}{I_1^*}-\frac{I_1R^*}{I_1^*R}-\frac{R}{R^*})$
$\dot{V}_6 \leq \varepsilon(1-\rho)I_1^*(1+\frac{I_1}{I_!^*}-\frac{I_2}{I_2^*}-\frac{I_1I_2^*}{I_1^*I_2})$
$\dot{V}_7 \leq \eta I_2^*(1+\frac{I_2}{I_2^*}-\frac{T}{T^*}-\frac{T^*I_2}{TI_2^*})$

Hence, $\dot{V}=\dot{V}_1+\dot{V}_2+\dot{V}_3+\dot{V}_4+\dot{V}_5+\dot{V}_6+\dot{V}_7 \leq 0$ for all $(S^*,V^*,E^*,I_1^*,R^*,I_2^*,T^*)$ in the interior of Ω. The inequality implies that we must have $S=S^*, V/V^*=E/E^*=I_1/I_1^*=R/R^*=I_2/I_2^*=T/T^*$ if $\dot{V}=0$. Letting $S=S^*$ in the equation of system 3.6, we obtain $V=V^*, E=E^*, I=I_1^*, R=R^*, I_2=I_2^*$ and thus $T=T^*$. This implies that V is negative definite with respect to P^*. According to the LaSalle's invariance principle[15], if $R_0>0$, the endemic equilibrium P^* is globally asymptotically stable.

4.1.7. *Sensitivity Analysis.* The effect of changing parameter values on the perceived usefulness of the reproduction number,R_0, is demonstrated in this section. It is necessary to identify the important parameter, which may be a vital threshold to manage the illness.

The following are the mathematical representations of $R_0's$ sensitivity index towards the parameters β_1,β_2,Λ,γ,σ,ε,ρ,μ,η,δ_1:

$$\frac{\partial R_0}{\partial \beta_1}=\frac{\Lambda\gamma}{(\sigma+\mu)(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)}, \quad \frac{\partial R_0}{\partial \beta_2}=\frac{\Lambda\gamma\varepsilon(1-\rho)}{(\sigma+\mu)(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\eta+\delta_1+\mu)},$$
$$\frac{\partial R_0}{\partial \Lambda}=\frac{\beta_1\gamma}{(\sigma+\mu)(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)}+\frac{\beta_2\gamma\varepsilon(1-\rho)}{(\sigma+\mu)(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\eta+\delta_1+\mu)},$$
$$\frac{\partial R_0}{\partial \sigma}=-\left(\frac{\beta_1\gamma\Lambda}{(\sigma+\mu)^2(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)}+\frac{\beta_2\gamma\varepsilon(1-\rho)\Lambda}{(\sigma+\mu)^2(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\eta+\delta_1+\mu)}\right),$$
$$\frac{\partial R_0}{\partial \gamma}=\frac{\beta_1\mu\Lambda}{(\sigma+\mu)(\gamma+\mu)^2(\varepsilon\rho+\varepsilon(1-\rho)+\mu)}+\frac{\beta_2\mu\varepsilon(1-\rho)\Lambda}{(\sigma+\mu)(\gamma+\mu)^2(\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\eta+\delta_1+\mu)}$$
$$\frac{\partial R_0}{\partial \eta}=-\frac{\beta_2\Lambda\varepsilon(1-\rho)}{(\sigma+\mu)(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\eta+\delta_1+\mu)^2},$$
$$\frac{\partial R_0}{\partial \delta_1}=-\frac{\beta_2\Lambda\varepsilon(1-\rho)}{(\sigma+\mu)(\gamma+\mu)(\varepsilon\rho+\varepsilon(1-\rho)+\mu)(\eta+\delta_1+\mu)^2},$$
$$\frac{\partial R_0}{\partial \varepsilon}=-\frac{\beta_1\mu\Lambda}{(\sigma+\mu)(\gamma+\mu)(\varepsilon+\mu)^2}+\frac{\beta_2\mu(1-\rho)\Lambda}{(\sigma+\mu)(\gamma+\mu)(\varepsilon+\mu)^2(\eta+\delta_1+\mu)},$$
$$\frac{\partial R_0}{\partial \rho}=-\frac{\beta_2\Lambda\gamma\varepsilon}{(\sigma+\mu)(\gamma+\mu)(\varepsilon+\mu)(\eta+\delta_1+\mu)},$$
$$\frac{\partial R_0}{\partial \mu}=-(A+Z)$$

where

$$A=\frac{\beta_1\Lambda\gamma[3\mu^2+2\mu(\mu+\sigma+\varepsilon)+(\sigma\gamma+\gamma\varepsilon+\sigma\varepsilon)]}{[(\sigma+\mu)(\gamma+\mu)(\varepsilon+\mu)]^2}$$

$$Z = \beta_2\Lambda\gamma\varepsilon(1-\rho)[4\mu^3+3\mu^2(1+\varepsilon+(\eta+\delta_1)+2\mu(\sigma\gamma+\sigma\varepsilon+\sigma\eta+\sigma\delta_1$$
$$+\sigma+\varepsilon\gamma+\gamma\eta+\gamma\delta_1+1+\varepsilon(\eta+\delta_1))+\varepsilon\sigma\gamma+(\eta+\delta_1)(\sigma\gamma+\varepsilon\sigma+\gamma\varepsilon+)]$$
$$/[(\sigma+\mu)(\gamma+\mu)(\varepsilon+\mu)(\eta+\delta_1+\mu)]^2$$

It may be deduced that some derivatives appear positive and that as any of the positive value parameters β_1,β_2,Λ,γ, described above is increased, the basic reproductive number,R_0, increases. The proportionate reaction to the proportion stimulation is used to calculate the elasticity.

We have

$E_{\beta_1} = \frac{\partial R_0}{\partial \beta_1} \times \frac{\beta_1}{R_0} = \frac{1}{1+\frac{\beta_2\varepsilon(1-\rho)}{\beta_1(\eta+\delta_1+\mu)}}$, $\quad E_{\beta_2} = \frac{\partial R_0}{\partial \beta_2} \times \frac{\beta_2}{R_0} = \frac{1}{1+\frac{\beta_1(\eta+\delta_1+\mu)}{\beta_2\varepsilon(1-\rho)}}$,

$E_\Lambda = \frac{\partial R_0}{\partial \Lambda} \times \frac{\Lambda}{R_0} = 1, \qquad E_\gamma = \frac{\partial R_0}{\partial \gamma} \times \frac{\gamma}{R_0} = \frac{\mu}{(\gamma+\mu)}$

Consequently, we see that E_{β_1},E_{β_2},E_Λ, and E_γ are positive. This implies that raising the values of these parameters,β_1,β_2,Λ,γ, will increase the value of the basic reproduction number.R_0.

4.1.8. *Adam-Bashforth-Moulton Predictor–Corrector Scheme for the $SVEI_1RI_2T$ Model.* The most frequently employed numerical method for solving fractional order initial value issues is the Adams-Bashforth-Moulton strategy.

Consider the fractional differential equation below:

$$^cD_t^\nu G_i(t) = f_j(t, G_j(t)), \quad G_j^r(0) = G_{j0}^r \tag{4.15}$$

$$r = 0, 1, 2, ..., \nu, j \in \mathbb{N}$$

where G_{j0}^r is the arbitrary real number, $\nu > 0$ and the fractional differential operator D_t^ν is similar to the well-known Volterra integral equation in the Caputo sense.

$$G_j(t) = \sum_{n=0}^{\nu-1} G_{j0}^r \frac{t^n}{n!} + \frac{1}{\Gamma(\nu)} \int_0^t (t-u)^{\nu-1} f_j(u, G_j(u))du, \quad j \in \mathbb{N} \tag{4.16}$$

In this study, we investigate the numerical solution of a fractional order $SVEI_1RI_2T$ model with vaccination using the Adam's-Bashforth-Moulton predictor-corrector scheme. The algorithm is described below:

Let $h = \frac{T}{\hat{m}}, \quad t_n = nh, \quad n = 0,1,2,...,\hat{m}.$

Corrector formulae:

$$S_{n+1} = S_0 + \frac{h^\nu}{\Gamma(\nu+2)}(\Lambda - \beta_1 S^p_{n+1} I_1{}^p_{n+1} - \beta_2 S^p_{n+1} I_2{}^p_{n+1} - \sigma S^p_{n+1} - \mu S^p_{n+1}) + \frac{h^\nu}{\Gamma(\nu+2)} \sum_{j=0}^{n} \alpha_{j,n+1}((\Lambda - \beta_1 S_j I_{1j} - \beta_2 S_j I_{2j} - \sigma S_j - \mu S_j)$$

$$V_{n+1} = V_0 + \frac{h^\nu}{\Gamma(\nu+2)}(\sigma S^p_{n+1} - \mu V^p_{n+1}) + \frac{h^\nu}{\Gamma(\nu+2)} \sum_{j=0}^{n} \alpha_{j,n+1}(\sigma S_j - \mu V_j)$$

$$E_{n+1} = E_0 + \frac{h^\nu}{\Gamma(\nu+2)}(\beta_1 S^p_{n+1} I_1{}^p_{n+1} + \beta_2 S^p_{n+1} I_2{}^p_{n+1} - \gamma E^p_{n+1} - \mu E^p_{n+1}) + \frac{h^\nu}{\Gamma(\nu+2)} \sum_{j=0}^{n} \alpha_{j,n+1}((\beta_1 S_j I_{1j} + \beta_2 S_j I_{2j} - \sigma E_j - \mu E_j)$$

$$I_{1n+1} = I_{10} + \frac{h^\nu}{\Gamma(\nu+2)}(\gamma E^p_{n+1} + \varepsilon\rho I_1{}^p_{n+1} - \varepsilon(1-\rho) I_1{}^p_{n+1} - \mu I_1{}^p_{n+1}) + \frac{h^\nu}{\Gamma(\nu+2)} \sum_{j=0}^{n} \alpha_{j,n+1}((\gamma E_j + \varepsilon\rho I_{1j} - \varepsilon(1-\rho) I_{1j} - \mu I_{1j})$$

$$R_{n+1} = R_0 + \frac{h^\nu}{\Gamma(\nu+2)}(\varepsilon\rho I_1{}^p_{n+1} - \mu R^p_{n+1}) + \frac{h^\nu}{\Gamma(\nu+2)} \sum_{j=0}^{n} \alpha_{j,n+1}(\varepsilon\rho I_{1j} - \mu R_j)$$

$$I_{2n+1} = I_{20} + \frac{h^\nu}{\Gamma(\nu+2)}(\varepsilon(1-\rho) I_1{}^p_{n+1} - (\eta + \delta_1 + \mu) I_2{}^p_{n+1}) + \frac{h^\nu}{\Gamma(\nu+2)} \sum_{j=0}^{n} \alpha_{j,n+1}((\varepsilon(1-\rho) I_{1j} + \varepsilon\rho I_{1j} - (\eta + \delta_1 + \mu) I_{2j})$$

$$T_{n+1} = T_0 + \frac{h^\nu}{\Gamma(\nu+2)}(\eta I_2{}^p_{n+1} - \mu T^p_{n+1} - \delta_2 T^p_{n+1}) + \frac{h^\nu}{\Gamma(\nu+2)} \sum_{j=0}^{n} \alpha_{j,n+1}(\eta I_{2j} - \mu T_j - \delta_1 T_j)$$

Predictor formulae:

$$S^p_{n+1} = S_0 + \frac{1}{\Gamma(\nu)} \sum_{j=0}^{n} \zeta_{j,n+1}((\Lambda - \beta_1 S_j I_{1j} - \beta_2 S_j I_{2j} - \sigma S_j - \mu S_j)$$

$$V^p_{n+1} = V_0 + \frac{1}{\Gamma(\nu)} \sum_{j=0}^{n} \zeta_{j,n+1}(\sigma S_j - \mu V_j)$$

$$E^p_{n+1} = E_0 + \frac{1}{\Gamma(\nu)} \sum_{j=0}^{n} \zeta_{j,n+1}((\beta_1 S_j I_{1j} + \beta_2 S_j I_{2j} - \sigma E_j - \mu E_j)$$

$$I_1{}^p_{n+1} = I_{10} + \frac{1}{\Gamma(\nu)} \sum_{j=0}^{n} \zeta_{j,n+1}((\gamma E_j + \varepsilon\rho I_{1j} - \varepsilon(1-\rho) I_{1j} - \mu I_{1j})$$

$$R^p_{n+1} = R_0 + \frac{1}{\Gamma(\nu)} \sum_{j=0}^{n} \zeta_{j,n+1}(\varepsilon\rho I_{1j} - \mu R_j)$$

$$I_2{}^p_{n+1} = I_{20} + \frac{1}{\Gamma(\nu)} \sum_{j=0}^{n} \zeta_{j,n+1}((\varepsilon(1-\rho) I_{1j} + \varepsilon\rho I_{1j} - (\eta + \delta_1 + \mu) I_{2j})$$

$$T_{n+1}^{p} = T_0 + \frac{1}{\Gamma(\nu)} \sum_{j=0}^{n} \zeta_{j,n+1}(\eta I_{2j} - \mu T_j - \delta_1 T_j)$$

where

$$\nu_{j,n+1} = \begin{cases} n^{\nu+1} - (n-\nu)(n+1)^{\nu}, & if\, j = 0 \\ (n-j+2)^{\nu+1} + (n-j)^{\nu+1} - 2(n-j+1)^{\nu} + 1 & 0 \leq j \leq n, \\ 1, & if\, j = 1 \end{cases}$$

and

$$\zeta = \frac{h^{\nu}}{\nu}[(n+1-j)^{\nu} - (n-j)^{\nu}], \quad 0 \leq j \leq n.$$

5. Numerical Simulation and Discussion

In this section, we run rigorous numerical simulations to evaluate and validate our model system's analytical results 3.6. To achieve a numerical solution to the system 3.6, we used the mathematical software MATLAB (2018a version) and Adam's-Bashforth-Moulton predictor-corrector scheme.

We investigate numerical simulations of the model system 3.6 in the Caputo sense, using the parameters listed in Table 1. In the scenario, Table 1 is utilized for simulation. The following figures were produced to examine the behavior of the model 3.6 under various initial conditions.

TABLE 2. Estimated values of parameters and Varibles

Variables	Value	Source
S	100	Assumed
V	15	Assumed
E	5	Assumed
I_1	6	Assumed
R	10	Assumed
I_2	3	Assumed
T	4	Assumed

Parameters	Value	Source
Λ	0.0260	[1]
σ	0.8	Estimated
β_1	0.095	Estimated
β_2	0.25	[2]
γ	0.03	[1]
$\varepsilon\rho$	3.6	Estimated
$\varepsilon(1-\rho)$	0.4	Estimated
δ_1	0.0063	Assummed
η	0.025	Assumed
δ_2	0.051	Assumed
μ	0.01890	Estimated

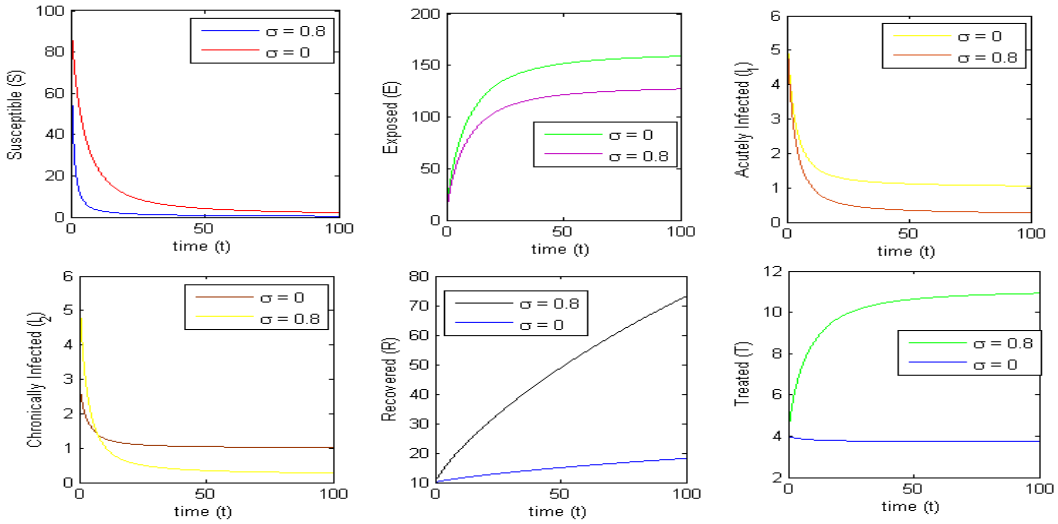

FIGURE 2. Comparison of dynamical behaviour of all individuals with respect to time for fractional order $\alpha = 0.82$, $\sigma = 0$ and $\sigma = 0.8$

Figure 2 shows the dynamical behavior of all individuals for fractional order$\alpha = 0.82$. The comparison of the number of susceptible, infected, exposed, recovered and treated individuals

in case of the vaccination parameter $\sigma = 0$ and $\sigma = 0.8$ is quite obvious.The number of susceptible individuals is less for $\sigma = 0.8$ compared to $\sigma = 0$. Similar is the case with exposed individuals and infected individuals. However, in the case of recovered individuals and individuals under treatment, the situation is exactly the opposite, for obvious reasons. Now, the recovered individuals will be more in case of $\sigma = 0.8$ than in case of $\sigma = 0$.

Vaccination is an important component in protecting people from Hepatitis B, and several concepts have been offered in which vaccination rates are seen as extremely advantageous. As a result, the addition of the vaccine parameter σ reduces the basic reproduction number R_0.

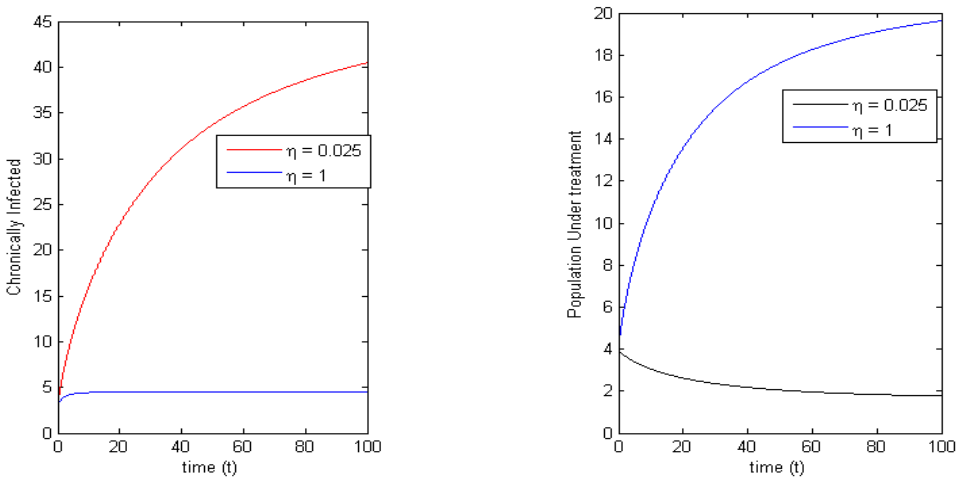

FIGURE 3. Comparison of dynamical behaviour between individuals who are chronically infected and individuals under treatment with respect to time for fractional order $\alpha = 0.82$, $\eta = 0.025$ and $\sigma = 1.0$

Figure 3 shows the dynamical behavior of between the chronically infected individuals and individuals under treatment for fractional order$\alpha = 0.82$. The number of chronically infected indidvuals is more for $\eta = 0.025$ than $\eta = 1$. It is exactly the opposite for the population under treatment because treatment is also a control in this study with $\delta_2 < \delta_1$. Although there is no cure for chronically infected people, receiving therapy reduces the number of deaths caused by the virus and lessens the risk of contracting other infections.

6. Conclusion

In this paper we have discussed the fractional order $SVEI_1RI_2T$ model with vaccination and treatment as control strtegies. Based on the data collected, we estimated the basic reproduction number. The fractional-order derivatives are typically more suitable in modeling because the option of derivative order allows one more degree of freedom, resulting in a better fit to real-time data with less inaccuracy than the integer-order model. The model shows that the Hepatitis B virus propagation is mostly determined by the population's contact rates with affected people. It has been observed that when the proportion of the population that is vaccinated increases, the spread of the virus is drastically reduced. Thus, if this is accomplished through widespread vaccination or making vaccine mandatory, the virus can be avoided.Treatment was also shown to be a control strategy for the spread of Hepaptitis B virus. Sensitivity analysis reveals that R_0 is directly proportional to the recruitment rate of susceptible individualsΛ, the rate of infection of susceptible individuals β_1, β_2 and the rate of progression from exposed to infected individuals γ, all of which can be controlled through the effective implementation of vaccination drives. To obtain numerical solutions to the system, the Adam-Bashforth-Moulton predictor-corrector technique was applied. To validate the efficacy and influence of the control parameters, numerical simulations using MATLAB are given.

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Conflict of Interests

The authors declare that there is no conflict of interests.

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