Research Article
The Transmission Dynamics of Hepatitis B Virus via the Fractional-Order Epidemiological Model

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We investigate and analyze the dynamics of hepatitis B with various infection phases and multiple routes of transmission. We formulate the model and then fractionalize it using the concept of fractional calculus. For the purpose of fractionalizing, we use the Caputo–Fabrizio operator. Once we develop the model under consideration, existence and uniqueness analysis will be discussed. We use fixed point theory for the existence and uniqueness analysis. We also prove that the model under consideration possesses a bounded and positive solution. We then find the basic reproductive number to perform the steady-state analysis and to show that the fractional-order epidemiological model is locally and globally asymptotically stable under certain conditions. For the local and global analysis, we use linearization, mean value theorem, and fractional Barbalat’s lemma, respectively. Finally, we perform some numerical findings to support the analytical work with the help of graphical representations.

1. Introduction

Hepatitis B virus causes inflammation of the liver. It results from a noncytopathic virus which is called the hepatitis B virus (HBV). Characteristic of HBV is its high tissue and species specificity, as well as a unique genomic organization and replication mechanism. The infection of HBV has multiple phases: acute and chronic. The acute one refers to the first six months whenever there is an exposure of some one to the virus. Usually, in this period, the immune system has the capability to vanish the infection, while for some severe cases, it may also lead to the serious stage and so results in the lifelong illness. This is also known as the chronic stage. It could be noted that whenever HBsAg is positive for a person for a period of more than 6 months, it shows that it has a chronic illness. In case of the chronic stage, often, the individual has no history of the acute stage. This infection may also lead to the scarring of the liver, become liver failure, and produce liver cancer [1]. Hepatitis B virus is transferred by many ways: blood (razors, sharing of blades, toothbrushes, etc.) and semen and vaginal [2–5]. One of the other key sources of transmission is from the infected mother to her child called vertical transmission [6]. Worldwide, there are millions of infected population according to the WHO, in which only 93 millions are infected in China [7, 8]. Vaccines are available to immunize from the HBV which are very effective and almost provide permanent immunity [9, 10]. Mathematical modeling of infectious diseases has a vast field and has a rich literature, which plays a significant role to explore the dynamics and suggest the control mechanism. Since hepatitis B is one of the life-threatening and leading causes of death, it obtained the attention of various researches,
and consequently, many epidemiological models were developed (see [11–15]). Anderson and May presented a study in the form of a simple model to investigate the influence of carriers on the transmission of hepatitis B [16]. Williams et al. presented and analyzed the hepatitis B dynamics in the United Kingdom [17]. Moreover, a model was presented by Medley et al. to forecast a mechanism for eliminating hepatitis B from New Zealand [18]. In a similar way, a model that evaluates the effectiveness of the vaccination programme with the effect of age in China was presented by Zhao et al. [19]. Bakare et al. proposed the analysis of control by using an SIR epidemic model [20]. More epidemic models were investigated with control strategies by Kamyad et al. [21]. Onyango developed a model to study the multiple endemic solutions [22]. Similarly, Zhang et al. studied the dynamics of hepatitis B in Xinjiang [23]. Very recently, Khan et al. [24, 25] and Nana-Kyere et al. [26] formulated some epidemiological models to study different parameters' influences on the disease transmission and to suggest some control measures for the elimination of the infection. The study of fractional calculus obtained the attention of researchers and is growing rapidly. This analysis has been used to capture the axioms of inherited and the memory of different fields of science and technology. Numerous classical models have been proved with less accuracy in case of predicting the future dynamics of a system. However, models having fractional order are more useful to allocate and detain the missing information [27, 28]. It could also be stated that the classical derivative does not provide the dynamics between two different points [29, 30].

It is noted that hepatitis B virus transmission is influenced by different factors, i.e., various phases, routes of transmission, etc. Especially, the carriers are significant. The chronic carriers have no symptoms while transmitting the infection. Moreover, it could also be noted that the increased development of fractional calculus and fractional-order epidemiological models are more suitable than the classical order epidemic models and complex dynamics of hepatitis B; we therefore investigate a hepatitis B virus transmission epidemic model with various infection phases and multiple routes of transmission. Moreover, we also use the fractional calculus to fractionalize the model under consideration which has not yet been studied to the best of our knowledge. Once we formulate the model, we then study the existence analysis as well as uniqueness to prove the well-posedness and biological feasibility of the problem under consideration. For this analysis, the fixed point theory will be used. We also prove that the solutions of the proposed system are bounded and positive. We then discuss the steady state of the proposed model and investigate that the model under consideration is locally and globally asymptotically stable. For local stability analysis, we use the method of linearization, mean value theorem, and fractional Barbalat’s lemma. Finally, some numerical simulations will be performed to support the analytical work and show the difference between the classical and fractional order.

2. Preliminaries

Here, we describe the fundamental concepts related to the fractional calculus which are helpful to obtain our results.

Definition 1 (see [30]). Let us assume a function \( \phi(t) \) such that \( \phi \in H^1(0,T), T > 0; \) if \( \alpha > 0 \) and \( n - 1 < \alpha < n, n \in \mathbb{N} \), then the Caputo and Caputo–Fabrizio derivative of the fractional order \( \alpha \) are defined, respectively, as

\[
CD^\alpha_0\phi(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t (t-x)^{n-\alpha-1} \phi^{(n)}(x) dx \tag{1}
\]

and

\[
CFD^\alpha_0\phi(t) = \frac{K(\alpha)}{(1-\alpha)} \int_0^t \phi'(y) \exp\left(\frac{\alpha(y-t)}{1-\alpha}\right) dy. \tag{2}
\]

In equations (1) and (2), \( C \) and \( CF \) represent, respectively, Caputo and Caputo–Fabrizio, while \( t > 0 \) and \( K(\alpha) \) represent the normalization function such that \( K(1) = 0 = K(0) \).

Definition 2 (see [30]). If \( 0 < \alpha < 1 \) and \( \phi(t) \) varies with time \( t \), then the Riemann–Liouville integral of order \( \alpha \) is defined as

\[
R\!L_0^\alpha\phi(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-y)^{\alpha-1} \phi(y) dy, \tag{3}
\]

while the integral of order \( \alpha \) in the Caputo–Fabrizio-Caputo (CF) sense is defined by

\[
CFI_0^\alpha\phi(t) = \frac{2(1-\alpha)}{(2-\alpha)K(\alpha)} \int_0^t \phi(y) dy + \frac{2\alpha}{(2-\alpha)K(\alpha)} \phi(0). \tag{4}
\]

3. Model Formulation

We formulate the model keeping in view the characteristics of hepatitis B virus and so distribute the total population symbolized by \( T(t) \) into different compartmental population sizes, i.e., susceptible \( S(t) \), acute \( A(t) \), chronic \( C(t) \), recovered/immune \( R(t) \), and vaccinated \( V(t) \). We also define some constraints for the proposed problem:

- \( a_1 \): all the variables \( (S, A, C, R, \text{ and } V) \) and the parameters \( (\Pi, \zeta, \beta, \rho, \delta, \eta, \sigma, \rho , \gamma, \epsilon, \text{ and } \tau) \) are non-negative in the epidemic problem that is under consideration.

- \( a_2 \): the successfully vaccinated portion \( \eta \) of the susceptible individuals goes to the recovered class.

- \( a_3 \): the contact of susceptible with acute infected as well as with chronically infected is, respectively, denoted by \( \beta \) and \( \beta \), which lead to the acute portion with probability \( 1 - p \) and go to chronic with probability \( (1 - p) \), where this assumption is based on the hypothesis that some of the individuals have no history of acute illness.
Since some of the individuals got recovery in the acute stage and it leads to the chronic stage for some severe cases, therefore, a natural recovery with probability \( q \) has been proposed, while \((1 - q)\) leads to the chronic stage.

The recovery under treatment \((\tau)\) is taken of the chronic population.

\( a_6 \): the disease-induced death rate \((e)\) occurs in the chronic stage only.

\( a_7 \): the newborn rate is \( \Pi \) and assumed to be susceptible, while getting successful vaccination \((\zeta)\) leads to the vaccinated class.

In light of these assumptions, we develop a model as presented in the following:

\[
\begin{align*}
\frac{dS(t)}{dt} &= (1 - \zeta)\Pi - \beta S(t)A(t) - \rho \beta S(t)C(t) - (\vartheta + \eta)S(t) + \sigma V(t), \\
\frac{dA(t)}{dt} &= p[\beta S(t)A(t) + \rho \beta S(t)C(t)] - (\vartheta + \gamma)A(t), \\
\frac{dC(t)}{dt} &= (1 - p)[\beta S(t)A(t) + \rho \beta S(t)C(t)] + q\gamma A(t) - (\vartheta + \varepsilon + \tau)C(t), \\
\frac{dR(t)}{dt} &= (1 - q)\gamma A(t) + \eta S(t) + \tau C(t) - \delta R(t), \\
\frac{dV(t)}{dt} &= \zeta\Pi - (\vartheta + \sigma)V(t)
\end{align*}
\]  

with initial population sizes

\[
S(0) > 0, A(0) \geq 0, C(0) \geq 0, R(0) > 0, V(0) > 0,
\]  

where \( \zeta \) is the proportion of successful vaccination individuals and \( \Pi \) is the newborn rate. Similarly, the transmission rate of hepatitis B is denoted by \( \beta \), while the reduced transmission rate is \( \rho \). Moreover, \( \vartheta \) and \( \eta \) are, respectively, the natural death rate and permanent recovered individuals’ rate. We also symbolize the recovery rate of acute and chronic hepatitis B individuals by \( \gamma \) and \( \tau \), respectively. The disease-induced death rate is represented by \( \varepsilon \), while those individuals who lose their immunity are represented by \( \sigma \).

We extend the reported model by equation (5) to the associated fractional-order \((\alpha < 0 < \alpha < 1)\) version by taking into account the Caputo–Fabrizio–Caputo (CF) operator. We therefore replace the derivatives in the problem under consideration with a fractional derivative to maintain the dimension of both sides of the equations of the proposed model taking the \( \alpha \) power of each parameter which becomes

\[
\begin{align*}
\text{CF} D_0^\alpha S(t) &= (1 - \zeta^\alpha)\Pi^\alpha - \beta^\alpha S(t)A(t) - \rho^\alpha \beta^\alpha S(t)C(t) - (\vartheta^\alpha + \eta^\alpha)S(t) + \sigma^\alpha V(t), \\
\text{CF} D_0^\alpha A(t) &= p[\beta^\alpha S(t)A(t) + \rho^\alpha \beta^\alpha S(t)C(t)] - (\vartheta^\alpha + \gamma^\alpha)A(t), \\
\text{CF} D_0^\alpha C(t) &= (1 - p)[\beta^\alpha S(t)A(t) + \rho^\alpha \beta^\alpha S(t)C(t)] + q\gamma^\alpha A(t) - (\vartheta^\alpha + \varepsilon^\alpha + \tau^\alpha)C(t), \\
\text{CF} D_0^\alpha R(t) &= (1 - q)\gamma^\alpha A(t) + \eta^\alpha S(t) + \tau^\alpha C(t) - \delta^\alpha R(t), \\
\text{CF} D_0^\alpha V(t) &= \zeta^\alpha\Pi^\alpha - (\vartheta^\alpha + \sigma^\alpha)V(t).
\end{align*}
\]  

We now discuss the existence and uniqueness of the above fractional-order epidemiological model (7) in the following section.
4. Existence and Uniqueness

This section is devoted to the existence and uniqueness analysis of the solution of fractional-order epidemiological model (7). We use the concept of fixed point theory and prove the solution existence and uniqueness. For this analysis, transforming the proposed system into an integral equation, we obtain

\[
S(t) = S(0) + \frac{2(1-\alpha)}{K(\alpha)(2-\alpha)} \left\{ (1-\zeta^\alpha) \Pi^\alpha - \beta^\alpha S(t) A(t) - \rho^\alpha \beta^\alpha S(t) C(t) - (\theta^\alpha + \eta^\alpha) S(t) + \sigma^\alpha V(t) \right\},
\]

\[
A(t) = A(0) + \frac{2(1-\alpha)}{K(\alpha)(2-\alpha)} \left\{ \int_0^t \left[ (1-\zeta^\alpha) - \beta^\alpha S(y) A(y) - \rho^\alpha \beta^\alpha S(y) C(y) - (\theta^\alpha + \eta^\alpha) S(y) + \sigma^\alpha V(y) \right] dy, \right\}
\]

\[
C(t) = C(0) + \frac{2(1-\alpha)}{K(\alpha)(2-\alpha)} \left\{ (1-\zeta^\alpha) \Pi^\alpha - \beta^\alpha S(t) A(t) + \rho^\alpha \beta^\alpha S(t) C(t) + q\eta^\alpha A(t) - (\theta^\alpha + \epsilon^\alpha + \tau^\alpha) C(t) \right\},
\]

\[
R(t) = R(0) + \frac{2(1-\alpha)}{K(\alpha)(2-\alpha)} \left\{ (1-\zeta^\alpha) \Pi^\alpha - (\theta^\alpha + \epsilon^\alpha + \tau^\alpha) R(t) \right\},
\]

\[
V(t) = V(0) + \frac{2(1-\alpha)}{K(\alpha)(2-\alpha)} \left\{ \int_0^t \left[ \zeta^{\alpha} \Pi^\alpha - (\theta^\alpha + \epsilon^\alpha) V(t) \right] dy. \right\}
\]
Let $\ell_1$, $\ell_2$, $\ell_3$, $\ell_4$, and $\ell_5$ be the kernels, and they are defined by

\begin{align}
\ell_1(S(t), t) & = [(1 - \cdot^\alpha)\Pi^\beta - \beta^\alpha(S-t)A(t) - \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha)S(t) + \cdot^\alpha V(t))], \\
\ell_2(A(t), t) & = [p[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha)A(t)]], \\
\ell_3(C(t), t) & = [(1 - p)[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha)C(t)]], \\
\ell_4(R(t), t) & = [(1 - q)[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha)R(t)]], \\
\ell_5(V(t), t) & = [\cdot^\alpha\Pi^\beta - (\cdot^\alpha + \cdot^\alpha)V(t)].
\end{align}

**Theorem 1.** The above kernels $\ell_1$, $\ell_2$, $\ell_3$, $\ell_4$, and $\ell_5$ satisfy axioms of Lipschitz conditions.

**Proof.** Let us assume that $S$ and $S_1$, $A$ and $A_1$, $C$ and $C_1$, $R$ and $R_1$, and $V$ and $V_1$ are, respectively, the two functions for the kernels $\ell_1$, $\ell_2$, $\ell_3$, $\ell_4$, and $\ell_5$, so we establish the following system:

\begin{align}
\ell_1(S(t), t) - \ell_1(S_1(t), t) & = [(1 - \cdot^\alpha)\Pi^\beta - \beta^\alpha(S-S_1)A(t) - \cdot^\alpha\beta^\alpha(S-S_1)(C(t) - (\cdot^\alpha + \cdot^\alpha)(S-S_1) + \cdot^\alpha V(t)], \\
\ell_2(A(t), t) - \ell_2(A_1(t), t) & = [p[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha)(A-A_1)]], \\
\ell_3(C(t), t) - \ell_3(C_1(t), t) & = [(1 - p)[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha))C(t)]], \\
\ell_4(R(t), t) - \ell_4(R_1(t), t) & = [(1 - q)[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha)R(t)]], \\
\ell_5(V(t), t) - \ell_5(V_1(t), t) & = [\cdot^\alpha\Pi^\beta - (\cdot^\alpha + \cdot^\alpha)(V-V_1)].
\end{align}

Cauchy’s inequality application leads to the following system:

\begin{align}
\|\ell_1(S(t), t) - \ell_1(S_1(t), t)\| & = \|(1 - \cdot^\alpha)\Pi^\beta - \beta^\alpha(S-S_1)A(t) - \cdot^\alpha\beta^\alpha(S-S_1)(C(t) - (\cdot^\alpha + \cdot^\alpha)(S-S_1) + \cdot^\alpha V(t)], \\
\|\ell_2(A(t), t) - \ell_2(A_1(t), t)\| & = \|p[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha)(A-A_1)]], \\
\|\ell_3(C(t), t) - \ell_3(C_1(t), t)\| & = \|(1 - p)[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha))C(t)]], \\
\|\ell_4(R(t), t) - \ell_4(R_1(t), t)\| & = \|(1 - q)[\beta^\alpha(S-t)A(t) + \cdot^\alpha\beta^\alpha(S-t)(C(t) - (\cdot^\alpha + \cdot^\alpha)R(t)]], \\
\|\ell_5(V(t), t) - \ell_5(V_1(t), t)\| & = [\cdot^\alpha\Pi^\beta - (\cdot^\alpha + \cdot^\alpha)(V-V_1)].
\end{align}

Recursively, we obtain

\begin{align}
S(t) & = \frac{2(1 - \cdot^\alpha)}{(2 - \cdot^\alpha)K(\cdot)} \int_0^t \ell_1(S_{n-1}(y), y) dy, \\
A(t) & = \frac{2(1 - \cdot^\alpha)}{(2 - \cdot^\alpha)K(\cdot)} \int_0^t \ell_2(A_{n-1}(y), y) dy, \\
C(t) & = \frac{2(1 - \cdot^\alpha)}{(2 - \cdot^\alpha)K(\cdot)} \int_0^t \ell_3(C_{n-1}(y), y) dy, \\
R(t) & = \frac{2(1 - \cdot^\alpha)}{(2 - \cdot^\alpha)K(\cdot)} \int_0^t \ell_4(R_{n-1}(y), y) dy, \\
V(t) & = \frac{2(1 - \cdot^\alpha)}{(2 - \cdot^\alpha)K(\cdot)} \int_0^t \ell_5(V_{n-1}(y), y) dy.
\end{align}
The norm application with majorizing and the difference between successive terms imply

\[
\|U_n(t)\| = \|S_n(t) - S_{L,(n-1)}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \int_0^t \|\ell_1(S_{n-1}(y), y) - \ell_1(S_{L,(n-2)}(y), y)\| dy,
\]

\[
\|W_n(t)\| = \|A_n(t) - A_{L,(n-1)}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \int_0^t \|\ell_2(A_{n-1}(y), y) - \ell_2(A_{L,(n-2)}(y), y)\| dy,
\]

\[
\|X_n(t)\| = \|C_n(t) - C_{L,(n-1)}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \int_0^t \|\ell_3(C_{n-1}(y), y) - \ell_3(C_{L,(n-2)}(y), y)\| dy.
\]

\[
\|Y_n(t)\| = \|R_n(t) - R_{L,(n-1)}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \int_0^t \|\ell_4(R_{n-1}(y), y) - \ell_4(R_{L,(n-2)}(y), y)\| dy,
\]

\[
\|Z_n(t)\| = \|V_n(t) - S_{L,(n-1)}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \int_0^t \|\ell_5(V_{n-1}(y), y) - \ell_5(S_{L,(n-2)}(y), y)\| dy.
\]

where

\[
\sum_{i=0}^{\infty} U_i(t) = S_n(t),
\]

\[
\sum_{i=0}^{\infty} W_i(t) = A_n(t),
\]

\[
\sum_{i=0}^{\infty} X_i(t) = B_n(t),
\]

\[
\sum_{i=0}^{\infty} Y_i(t) = R_n(t),
\]

\[
\sum_{i=0}^{\infty} Z_i(t) = V_n(t).
\]

Since the kernels \(\ell_1, \ldots, \ell_5\) satisfy the Lipschitz conditions,
\[ \|U_n(t)\| = \|S_n(t) - S_{1,n-1}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \tau_1 \|S_{n-1}(t) - S_{1,n-2}(t)\| \\
+ \frac{2\alpha}{(2 - \alpha)K(\alpha)} \tau_2 \int_0^t \|S_{n-1}(y) - S_{1,n-2}(y)\| \, dy, \]

\[ \|W_n(t)\| = \|A_n(t) - A_{1,n-1}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \tau_3 \|A_{n-1}(t) - A_{1,n-2}(t)\| \\
+ \frac{2\alpha}{(2 - \alpha)K(\alpha)} \tau_4 \int_0^t \|A_{n-1}(y) - A_{1,n-2}(y)\| \, dy, \] (16)

\[ \|X_n(t)\| = \|C_n(t) - C_{1,n-1}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \tau_5 \|C_{n-1}(t) - C_{1,n-2}(t)\| \\
+ \frac{2\alpha}{(2 - \alpha)K(\alpha)} \tau_6 \int_0^t \|C_{n-1}(y) - C_{1,n-2}(y)\| \, dy, \|Y_n(t)\| \]

\[ \|Z_n(t)\| = \|V_n(t) - V_{1,n-1}(t)\| \leq \frac{2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \tau_7 \|V_{n-1}(t) - V_{1,n-2}(t)\| \\
+ \frac{2\alpha}{(2 - \alpha)K(\alpha)} \tau_9 \int_0^t \|V_{n-1}(y) - V_{1,n-2}(y)\| \, dy. \]

**Theorem 2.** The solution of fractional-order epidemiological model (7) exists.

**Proof.** The use of equation (15) with the recursive scheme implies

\[ \|U_n(t)\| \leq \|S(0)\| + \left\{ \frac{2\tau_1(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n + \left\{ \frac{2\tau_2(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n, \]

\[ \|W_n(t)\| \leq \|A(0)\| + \left\{ \frac{2\tau_3(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n + \left\{ \frac{2\tau_4(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n, \]

\[ \|X_n(t)\| \leq \|0\| + \left\{ \frac{2\tau_5(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n + \left\{ \frac{2\tau_6(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n, \] (17)

\[ \|Y_n(t)\| \leq \|R(0)\| + \left\{ \frac{2\tau_7(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n + \left\{ \frac{2\tau_8(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n, \]

\[ \|Z_n(t)\| \leq \|V(0)\| + \left\{ \frac{2\tau_9(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n + \left\{ \frac{2\tau_9(1 - \alpha)}{K(\alpha)(2 - \alpha)} \right\}^n. \]

We investigate that equation (17) is the solution of model (7); therefore, we make the following substitutions:

\[ S(t) = S_n(t) - Y_{1,n}(t), \]

\[ A(t) = A_n(t) - Y_{2,n}(t), \]

\[ B(t) = B_n(t) - Y_{3,n}(t), \]

\[ R(t) = R_n(t) - Y_{4,n}(t), \]

\[ V(t) = V_n(t) - Y_{5,n}(t), \] (18)
where \( Y_{1,n}(t), Y_{2,n}(t), Y_{3,n}(t), Y_{4,n}(t), \) and \( Y_{5,n}(t) \) denote the remainder terms of the series. So,

\[
S(t) - S_{n-1}(t) = \frac{2(1-a)\ell_1(S(t) - \Pi_1,(t))}{K(a)(2-a)} + \frac{2\alpha}{K(a)(2-a)} \int_0^t \ell_1(S(y) - Y_{1,n}(y))dy,
\]

\[
A(t) - A_{n-1}(t) = \frac{2\ell_2(A(t) - Y_{2,n}(t))(1-a)}{K(a)(2-a)} + \frac{2\alpha}{K(a)(2-a)} \int_0^t \ell_2(A(y) - Y_{1,n}(y))dy,
\]

\[
C(t) - C_{n-1}(t) = \frac{2\ell_3(C(t) - Y_{3,n}(t))(1-a)}{K(a)(2-a)} + \frac{2\alpha}{K(a)(2-a)} \int_0^t \ell_3(C(y) - Y_{1,n}(y))dy,
\]

\[
R(t) - R_{n-1}(t) = \frac{2\ell_4(R(t) - Y_{4,n}(t))(1-a)}{K(a)(2-a)} + \frac{2\alpha}{K(a)(2-a)} \int_0^t \ell_4(R(y) - Y_{1,n}(y))dy,
\]

\[
V(t) - V_{n-1}(t) = \frac{2\ell_5(V(t) - Y_{5,n}(t))(1-a)}{K(a)(2-a)} + \frac{2\alpha}{K(a)(2-a)} \int_0^t \ell_5(V(y) - Y_{1,n}(y))dy.
\]

Applying norm on both sides and the Lipschitz axiom,

\[
\begin{align*}
\|S(t) - S_{n-1}(t)\| &\leq \|Y_{1,n}(t)\| \left\{ 1 + \left( \frac{2(1-a)\tau_1}{K(a)(2-a)} + \frac{2\alpha t}{K(a)(2-a)} \right) \right\}, \\
\|A(t) - A_{n-1}(t)\| &\leq \|Y_{2,n}(t)\| \left\{ 1 + \left( \frac{2(1-a)\tau_2}{K(a)(2-a)} + \frac{2\alpha t}{K(a)(2-a)} \right) \right\}, \\
\|C(t) - C_{n-1}(t)\| &\leq \|Y_{3,n}(t)\| \left\{ 1 + \left( \frac{2(1-a)\tau_3}{K(a)(2-a)} + \frac{2\alpha t}{K(a)(2-a)} \right) \right\}, \\
\|R(t) - R_{n-1}(t)\| &\leq \|Y_{4,n}(t)\| \left\{ 1 + \left( \frac{2(1-a)\tau_4}{K(a)(2-a)} + \frac{2\alpha t}{K(a)(2-a)} \right) \right\}, \\
\|V(t) - V_{n-1}(t)\| &\leq \|Y_{5,n}(t)\| \left\{ 1 + \left( \frac{2(1-a)\tau_5}{K(a)(2-a)} + \frac{2\alpha t}{K(a)(2-a)} \right) \right\}.
\end{align*}
\]
Taking lim as $t$ approaches $\infty$, we get

\[
S(t) = \frac{2(1-a)\ell_1(S(t), t)}{K(a)(2-a)} + \frac{2\alpha}{K(a)(2-a)} \int_0^t \ell_1(S(y), y)dy + S(0),
\]
\[
A(t) = \frac{2(1-a)\ell_2(A(t), t)}{(2-a)K(a)} + \frac{2\alpha}{(2-a)K(a)} \int_0^t \ell_2(A(y), y)dy + A(0),
\]
\[
C(t) = \frac{2(1-a)\ell_3(C(t), t)}{(2-a)K(a)} + \frac{2\alpha}{(2-a)K(a)} \int_0^t \ell_3(C(y), y)dy + C(0),
\]
\[
R(t) = \frac{2(1-a)\ell_4(R(t), t)}{(2-a)K(a)} + \frac{2\alpha}{(2-a)K(a)} \int_0^t \ell_4(R(y), y)dy + R(0),
\]
\[
V(t) = \frac{2(1-a)\ell_5(V(t), t)}{(2-a)K(a)} + \frac{2\alpha}{(2-a)K(a)} \int_0^t \ell_5(V(y), y)dy + V(0),
\]

which proves the conclusion that the solution of the proposed epidemiological model as reported by equation (7) exists.

**Proof.** On the contradiction basis, we assume that $(S^+(t), A^+(t), B^+(t), R^+(t), V^+(t))$ is another solution of model (7); then,

**Theorem 3.** The proposed epidemiological model described by equation (7) possesses a unique solution.

\[
S(t) - S^+(t) = \frac{2(1-a)[\ell_1(S(t), t) - \ell_1(S^+(t), t)]}{K(a)(2-a)}
\]
\[
+ \frac{2\alpha}{K(a)(2-a)} \int_0^t [\ell_1(S(y), y) - \ell_1(S^+(y), y)]dy,
\]
\[
A(t) - A^+(t) = \frac{2(1-a)[\ell_2(S(t), t) - \ell_2(S^+(t), t)]}{K(a)(2-a)}
\]
\[
+ \frac{2\alpha}{K(a)(2-a)} \int_0^t [\ell_2(A(y), y) - \ell_2(A^+(y), y)]dy,
\]
\[
C(t) - C^+(t) = \frac{2(1-a)[\ell_3(S(t), t) - \ell_3(C^+(t), t)]}{K(a)(2-a)}
\]
\[
+ \frac{2\alpha}{K(a)(2-a)} \int_0^t [\ell_3(C(y), y) - \ell_3(C^+(y), y)]dy,
\]
\[
R(t) - R^+(t) = \frac{2(1-a)[\ell_4(R(t), t) - \ell_4(R^+(t), t)]}{K(a)(2-a)}
\]
\[
+ \frac{2\alpha}{K(a)(2-a)} \int_0^t [\ell_4(R(y), y) - \ell_4(R^+(y), y)]dy,
\]
\[
V(t) - V^+(t) = \frac{2(1-a)[\ell_5(V(t), t) - \ell_5(V^+(t), t)]}{K(a)(2-a)}
\]
\[
+ \frac{2\alpha}{K(a)(2-a)} \int_0^t [\ell_5(V(y), y) - \ell_5(V^+(y), y)]dy.
\]
Majorizing the above equations, we obtain

\[
\| S(t) - S^*(t) \| = \frac{2(1 - \alpha)\| \ell_1(S(t), t) - \ell_1(S^*(t), t) \|}{K(\alpha)(2 - \alpha)}
\]

\[
+ \frac{2\alpha}{K(\alpha)(2 - \alpha)} \int_0^t \| \ell_1(S(y), y) - \ell_1(S^*(y), y) \| dy,
\]

\[
\| A(t) - A^*(t) \| = \frac{2(1 - \alpha)\| \ell_2(A(t), t) - \ell_2(A^*(t), t) \|}{K(\alpha)(2 - \alpha)}
\]

\[
+ \frac{2\alpha}{K(\alpha)(2 - \alpha)} \int_0^t \| \ell_2(A(y), y) - \ell_2(A^*(y), y) \| dy,
\]

\[
\| C(t) - C^*(t) \| = \frac{2(1 - \alpha)\| \ell_3(C(t), t) - \ell_3(C^*(t), t) \|}{K(\alpha)(2 - \alpha)}
\]

\[
+ \frac{2\alpha}{K(\alpha)(2 - \alpha)} \int_0^t \| \ell_3(C(y), y) - \ell_3(C^*(y), y) \| dy,
\]

\[
\| R(t) - R^*(t) \| = \frac{2(1 - \alpha)\| \ell_4(R(t), t) - \ell_4(R^*(t), t) \|}{K(\alpha)(2 - \alpha)}
\]

\[
+ \frac{2\alpha}{K(\alpha)(2 - \alpha)} \int_0^t \| \ell_4(R(y), y) - \ell_4(R^*(y), y) \| dy,
\]

\[
\| V(t) - V^*(t) \| = \frac{2(1 - \alpha)\| \ell_5(V(t), t) - \ell_5(V^*(t), t) \|}{K(\alpha)(2 - \alpha)}
\]

\[
+ \frac{2\alpha}{K(\alpha)(2 - \alpha)} \int_0^t \| \ell_5(V(y), y) - \ell_5(V^*(y), y) \| dy.
\]

Using Theorems 1 and 2, one may obtain

\[
\| S(t) - S^*(t) \| \leq \frac{2\tau_3 \psi_1(1 - \alpha)}{K(\alpha)(2 - \alpha)} + \left( \frac{2\tau_3 \alpha \phi_s t}{K(\alpha)(2 - \alpha)} \right)^\alpha,
\]

\[
\| A(t) - A^*(t) \| \leq \frac{2\tau_3 (1 - \alpha) \psi_3}{K(\alpha)(2 - \alpha)} + \left( \frac{2\tau_3 \alpha \phi_s t}{K(\alpha)(2 - \alpha)} \right)^\alpha,
\]

\[
\| C(t) - C^*(t) \| \leq \frac{2(1 - \alpha)\tau_5 \psi_5}{K(\alpha)(2 - \alpha)} + \left( \frac{2\alpha \tau_5 \phi_s t}{K(\alpha)(2 - \alpha)} \right)^\alpha,
\]

\[
\| R(t) - R^*(t) \| \leq \frac{2\tau_3 \psi_3 (1 - \alpha)}{K(\alpha)(2 - \alpha)} + \left( \frac{2\alpha \tau_3 \phi_s t}{K(\alpha)(2 - \alpha)} \right)^\alpha,
\]

\[
\| V(t) - V^*(t) \| \leq \frac{2\tau_5 \psi_5 (1 - \alpha)}{K(\alpha)(2 - \alpha)} + \left( \frac{2\alpha \tau_5 \phi_t t}{K(\alpha)(2 - \alpha)} \right)^\alpha.
\]
The inequalities as reported by equation (24) hold for every value of \( \kappa \); thus, we obtain

\[
\begin{align*}
S(t) &= S^*(t), \\
A(t) &= A^*(t), \\
B(t) &= B^*(t), \\
R(t) &= R^*(t), \\
V(t) &= V^*(t).
\end{align*}
\] (25)

We now discuss the positivity as well as the boundedness of model (7) to show the well-posedness of the problem.

Furthermore, we define a certain region for the dynamics of the proposed problem which is positively invariant. For this, the following lemmas have been explored.

\textbf{Lemma 1.} Since \((S(t), A(t), C(t), R(t), V(t))\) are the solutions of model (7), let us consider that the model possesses nonnegative initial conditions; then, the solutions \((S(t), A(t), C(t), R(t), V(t))\) are nonnegative for all \(t \geq 0\).

\textbf{Proof.} We assume a general fractional-order \((\omega)\) model of system (7) as

\[
\begin{align*}
G^\omega D_{0,t}^\omega (S(t))) &= (1 - \zeta(t))\Pi^\omega - \beta^\omega S(t)A(t) - \rho^\omega \beta^\omega S(t)C(t) - \left(3^\omega + \eta^\omega\right) S(t) + \sigma^\omega V(t), \\
G^\omega D_{0,t}^\omega (A(t))) &= \rho[\beta^\omega S(t)A(t) + \rho^\omega \beta^\omega S(t)C(t)] - \left(3^\omega + \eta^\omega\right) A(t), \\
G^\omega D_{0,t}^\omega (C(t))) &= (1 - \rho)[\beta^\omega S(t)A(t) + \rho^\omega \beta^\omega S(t)C(t)] + \eta^\omega A(t) - \left(3^\omega + \epsilon^\omega + \tau^\omega\right) C(t), \\
G^\omega D_{0,t}^\omega (R(t))) &= (1 - q)\gamma^\omega A(t) + \eta^\omega S(t) + \tau^\omega C(t) - \theta^\omega R(t), \\
G^\omega D_{0,t}^\omega (V(t))) &= \zeta^\omega \Pi^\omega - \left(3^\omega + \sigma^\omega\right) V(t),
\end{align*}
\]

where \( G \) represents the fractional-order operator under consideration and \( \omega \) is the order. So, equation (26) becomes

\[
\begin{align*}
G^\omega D_{0,t}^\omega (S(t))) &= (1 - \zeta(t))\Pi^\omega > 0, \\
G^\omega D_{0,t}^\omega (A(t))) &= \rho[\beta^\omega S(t)A(t) + \rho^\omega \beta^\omega S(t)C(t)] - \left(3^\omega + \eta^\omega\right) A(t) \geq 0, \\
G^\omega D_{0,t}^\omega (C(t))) &= (1 - \rho)[\beta^\omega S(t)A(t) + \rho^\omega \beta^\omega S(t)C(t)] + \eta^\omega A(t) - \left(3^\omega + \epsilon^\omega + \tau^\omega\right) C(t) \geq 0, \\
G^\omega D_{0,t}^\omega (R(t))) &= (1 - q)\gamma^\omega A(t) + \eta^\omega S(t) + \tau^\omega C(t) - \theta^\omega R(t) > 0, \\
G^\omega D_{0,t}^\omega (V(t))) &= \zeta^\omega \Pi^\omega > 0,
\end{align*}
\]

where \( \kappa(\xi) = \{\xi = 0\} \) and \( S, A, C, R, V \) contained in \( C(R_+ \times R_+) \) and \( \xi \in \{S, A, C, R, V\} \). By following [31], we conclude that the solutions \((S(t), A(t), C(t), R(t), V(t))\) are positive for all nonnegative \(t\). \(\square\)

\textbf{Lemma 2.} Let \( \Omega \) be the region for dynamics of model (7) within it which is positively invariant; then,

\[
\Omega = \left\{ (S(t), A(t), C(t), R(t), V(t)) \in R^5_+ : S + A + C + R + V \leq \left(\frac{\Pi}{\theta}\right)^\omega \right\}.
\]

\textbf{Proof.} Since \( N \) represents the total population, therefore, it implies that

\[
G^\omega D_{0,t}^\omega (T(t)) + \theta^\omega T(t) \leq \Pi^\omega.
\] (29)

The solution of equation (29) gives

\[
T(t) \leq T(0)E_\omega (\Pi^\omega t^\omega) + \left(\frac{\Pi}{\theta}\right)^\omega (1 - E_\omega (\Pi^\omega t^\omega)),
\] (30)

where \( E(.) \) is the Mittag-Leffler function such that \( E_\omega (Z) = \sum_{n=0}^\infty Z^n / \Gamma(\omega n + 1) \). Note that, in equation (30), whenever times increase with no bound, \( T(t) \rightarrow (\Pi/\theta)^\omega \). Hence, if \( T(0) \leq (\Pi/\theta)^\omega \), then \( T(t) \leq (\Pi/\theta)^\omega \) for all \( t > 0 \), while if \( T(0) > (\Pi/\theta)^\omega \), then \( T \) goes into the feasible region \( \Omega \) and will never leave. So, it could be concluded that the dynamics of the fractional-order epidemiological model can be studied in the feasible region \( \Omega \). \(\square\)

5. Steady-State Analysis

The proposed epidemiological model (7) of the hepatitis B virus is examined for the equilibria: disease-free and endemic states. Let \( D_1 \) be the disease-free equilibrium of the proposed model; then, for analyzing this point, the population under consideration is assumed to be infection free. Thus, the system reported by equation (7) has a disease-free equilibrium \( D_1 = (S^0, A^0, C^0, R^0, V^0) \), where \( S^0 = q_1^0 (1 - \zeta(t)) + \sigma^\omega \zeta(t) q_1^0 q_2^0, A^0 = C^0 = 0, R^0 = \eta^\omega \Pi^\omega q_1^0 (1 - \zeta(t)) + \sigma^\omega \zeta(t) / \theta^\omega q_1^0 q_2^0, \) and \( V^0 = \zeta^\omega \Pi^\omega / q_1^0 \), and \( q_1 = \theta^\omega + \eta^\omega, q_2 = \theta^\omega + \epsilon^\omega + \tau^\omega, q_3 = \theta^\omega + \sigma^\omega \). Now, to calculate the basic reproductive number, we assume \( X = (A,C)^T \); then, system (7) yields
\[
\frac{dX}{dr} \bigg|_{D_1} = F - V, \quad (31)
\]

where
\[
F = \begin{bmatrix}
p\beta^aS^0 & p\beta^a\sigma_0 \\
(1 - p)\beta^aS^0 & (1 - p)\beta^a\sigma_0
\end{bmatrix},
\]
\[
V = \begin{bmatrix}
q_2 & 0 \\
-q\sigma^a & q_1
\end{bmatrix}. \quad (32)
\]

Therefore, the basic reproductive number is the spectral radius of \( \rho(FV^{-1}) \), i.e., \( R_0 = R_1 + R_2 + R_3 \), where
\[
R_1 = \frac{\beta^a\sigma_0}{q_2},
\]
\[
R_2 = \frac{\beta^a\sigma_0(1 - p)}{q_3},
\]
\[
R_3 = \frac{\beta^a\sigma_0(1 - p)q_3}{q_2d_3}. \quad (33)
\]

Let \( D_1 \) be the endemic equilibrium, and assume that \( S = S^* \), \( A = A^* \), \( C = C^* \), \( R = R^* \), and \( V = V^* \) at the steady state of the proposed model; then, the solution of the resultant algebraic equations will lead to the endemic equilibrium. Thus, regarding the local as well as global analysis of the proposed model, we have the following stability results.

**Theorem 4.** If \( R_0 < 1 \), then the disease-free equilibrium \( D_1 \) of the proposed model (7) is locally asymptotically stable, while if \( R_0 > 1 \), then the endemic equilibrium \( D_2 \) is locally asymptotically stable.

**Proof.** The linearizable version of the proposed hepatitis B model (7) around \( D_1 \) leads to a matrix given by
\[
J|_{D_1} = \begin{bmatrix}
-q_1 & -\beta^a\sigma_0 & -p\beta^a\sigma_0 & 0 & \sigma^a \\
0 & p\beta^a\sigma_0 - q_2 & p\beta^a\sigma_0 & 0 & 0 \\
0 & (1 - p)\beta^a\sigma_0 + q\sigma & (1 - p)\beta^a\sigma_0 - q_3 & 0 & 0 \\
\eta^a & (1 - q)\eta^a & \tau^a & -\sigma^a & 0 \\
0 & 0 & 0 & 0 & -q_4
\end{bmatrix}. \quad (34)
\]

The characteristic equation of the matrix \( J|_{D_1} \) takes the following form:
\[
\omega^5 + a_1\omega^4 + a_2\omega^3 + a_3\omega^2 + a_4\omega + a_5, \quad (35)
\]

where
\[
a_1 = q_1 + q_4 + \beta^a + q_2(1 - R_1) + q_3(1 - R_2),
\]
\[
a_2 = q_1q_4 + (q_1 + q_4)[\beta^a + q_2(1 - R_1) + q_3(1 - R_2)] \\
+ q_1q_3(1 - R_0) + q_3\beta^a(1 - R_2) + q_2\beta^a(1 - R_1),
\]
\[
a_3 = (q_1 + q_4)[q_2\beta^a(1 - R_1) + q_3\beta^a(1 - R_2) + q_2q_3(1 - R_3)] \\
+ q_1q_3[q_2\beta^a + q_3(1 - R_1) + q_3\beta^a(1 - R_0)],
\]
\[
a_4 = q_1q_4q_2q_3(1 - R_0) + q_2\beta^a(1 - R_1) + q_3\beta^a(1 - R_2) + (q_1 + q_4)q_2q_3\beta^a(1 - R_0),
\]
\[
a_5 = q_1q_4q_2q_3\beta^a(1 - R_0). \quad (36)
\]

It could be noted that the real parts of the eigenvalues of the above matrix \( J|_{D_1} \) are negative whenever Routh–Hurwitz criteria, i.e., \( H : \{a_i > 0, \text{for } i = 1, 2, 3, 4, 5, a_1a_2a_3 - a_4a_5 > 0 \} \) hold. So,
\[
a_1a_2a_3 - a_4a_5 = \{q_1 + q_4 + \beta^a + q_2(1 - R_1) + q_3(1 - R_2)\}\{q_1q_4 + (q_1 + q_4)\beta^a + q_2(1 - R_1) \\
+ q_1q_3(1 - R_0) + q_3\beta^a(1 - R_2) + q_2\beta^a(1 - R_1)\}\{q_1q_4q_2q_3(1 - R_0) \\
+ q_1q_3q_2\beta^a + q_3(1 - R_1) + q_3\beta^a(1 - R_0)\} - (q_1 + q_4 + \beta^a \\
+ q_2(1 - R_1) + q_3(1 - R_2))^2\{q_1q_4q_2(1 - R_1) + q_3(1 - R_2) + q_2q_3(1 - R_0)\} + (q_1 + q_4)q_2q_3\beta^a(1 - R_0), \quad (37)
\]
and

\[
(a_1 a_4 - a_2) (a_1 a_2 a_3 - a_3^2 - a_1^2 a_4) - a_2^2 (a_1 a_2 - a_3)^2 - a_1 a_2^2
\]

\[
= \begin{cases} 
(q_1 + q_4 + q_3^2 + q_2 (1 - R_1) + q_1 (1 - R_2)) \\
(q_1 q_4 (q_2^3 (1 - R_1) + q_3^2 (1 - R_2) + q_1 (1 - R_0)) + (q_1 + q_4) q_1 q_3 (1 - R_0) + (q_1 + q_4) q_2 q_3 (1 - R_0)) \\
- q_1 q_2 q_3 q_4^2 (1 - R_0)
\end{cases} 
\]

\[
= \begin{cases} 
(q_1 + q_4 + q_3^2 + q_2 (1 - R_1) + q_1 (1 - R_2)) [q_1 q_4 + (q_1 + q_4) q_3^2 + q_1 (1 - R_0) + q_2 q_3 (1 - R_0)] \\
+ q_1 q_2 q_3 q_4^2 (1 - R_0)
\end{cases} 
\]

\[
- q_1 q_2 q_3 q_4^2 (1 - R_0)
\]

\[
- (q_1 + q_4 + q_3^2 + q_2 (1 - R_1) + q_1 (1 - R_2)) q_1 q_2 q_3 q_4^2 (1 - R_0)^2.
\]

Clearly, we observe that all the coefficients \(a_i\) for \(i = 1, 2, 3, 4, 5\) are positive whenever \(R_0 < 1\), and if \(a_1 a_2 a_3 - a_3^2 - a_1^2 a_4\) and \((a_1 a_4 - a_2) (a_1 a_2 a_3 - a_3^2 - a_1^2 a_4) - a_2 (a_1 a_2 - a_3)^2 - a_1 a_2^2\) are positive, then it implies that the Routh–Hurwitz criteria hold, and so, the disease-free state \(D_1\) is stable. In a similar fashion, it can proved that the disease endemic state \(D_2\) of the proposed model (7) is stable.

\[\square\]

**Theorem 5.** If \(R_0 \leq 1\), then the disease-free equilibrium \(D_1\) of the proposed model (7) is globally asymptotically stable, while if \(R_0 > 1\), then the endemic equilibrium \(D_2\) is globally asymptotically stable.

**Proof.** Let \(\chi(t) = (S(t), A(t), C(t), R(t), V(t))\), and we claim that it has a finite limit whenever \(t\) approaches to \(\infty\); then, the last equation of model (7) looks like

\[
\text{CF} D_{0+}^a V(t) = \zeta^2 \Pi^a - q_4 V(t). \tag{39}
\]

Since for \(t \geq 0\) and for any \(\varphi, \psi \leq \psi e^\psi\), by following the mean value theorem and the result as stated by Theorem 3.1 in [32], equation (39) implies that

\[
\|V(t)\| \leq a C \exp\left[-\frac{q_1}{\psi} \psi e^\psi\right] t, \tag{40}
\]

where \(a = \|V_0\| e^{-T} + KT^a e^{-T} / a \Gamma(a) + \zeta^2 \Pi^a, t \geq T\), and \(C\) is a positive constant, and consequently, we obtain

\[
\lim_{t \to \infty} V(t) \leq C (\zeta \Pi)^a. \tag{41}
\]

Similarly, the first equation of the proposed fractional-order model (7) can be rewritten as

\[
\text{CF} D_{0+}^a S(t) \leq \left(1 - \zeta^a\right) \Pi^a - q_1 S(t) + \sigma^a V(t). \tag{42}
\]

Let \(b = \|V_0\| e^{-T} + KT^a e^{-T} / a \Gamma(a) + \left(1 - \zeta^a\right) \Pi^a + \sigma^a V(t);\) then,

\[
\|S(t)\| \leq b C \exp\left[-\frac{q_1}{\psi} \psi e^\psi\right] t, \tag{43}
\]

which implies that

\[
\lim_{t \to \infty} S(t) \leq C \left(1 - \zeta^a\right) \Pi^a + \lim_{t \to \infty} \sigma^a V(t), \tag{44}
\]

or equivalently, equation (44) may take the form after using equation (41) in equation (44) such that

\[
\lim_{t \to \infty} S(t) \leq \text{C} \Pi^a. \tag{45}
\]

In a similar fashion, \(\lim_{t \to \infty} \chi(t) = (S_\infty, A_\infty, C_\infty, R_\infty, V_\infty)\),

\[
\tag{46}
\]

\[
\]
\[ \phi(\chi) = \begin{pmatrix} \phi_1(\chi) \\ \phi_2(\chi) \\ \phi_3(\chi) \\ \phi_4(\chi) \\ \phi_5(\chi) \end{pmatrix} \]

\[
\begin{aligned}
(1 - \zeta^\alpha)\Pi^\alpha - \beta^\alpha S(t)A(t) - \rho^\alpha \beta^\alpha S(t)C(t) - q_1S(t) + \sigma^\alpha V(t) \\
p[\beta^\alpha S(t)A(t) + \rho^\alpha \beta^\alpha S(t)C(t)] - q_2A(t) \\
(1 - p)[\beta^\alpha S(t)A(t) + \rho^\alpha \beta^\alpha S(t)C(t)] + qy^\alpha A(t) - q_3C(t) \\
(1 - q)y^\alpha A(t) + \eta^\alpha S(t) + r^\alpha C(t) - \delta^\alpha R(t) \\
\zeta^\alpha \Pi^\alpha - q_4V(t)
\end{aligned}
\]

Thus, in light of the mean value theorem, there exist positive constants C_1 and C_2 such that

\[
\|\phi(\chi)\| \leq C_1 + C_2\|\chi\|.
\]

So, Theorems 2.1 and 3.1 in [33] imply that

\[
\lim_{t \to \infty} D_{\infty}^\alpha (\chi(t)) = (0, 0, 0, 0).
\]

Consequently,

\[
\begin{aligned}
(1 - \zeta^\alpha)\Pi^\alpha - \beta^\alpha S(t)A(t) - \rho^\alpha \beta^\alpha S(t)C(t) - q_1S(t) + \sigma^\alpha V(t) = 0, \\
p[\beta^\alpha S(t)A(t) + \rho^\alpha \beta^\alpha S(t)C(t)] - q_2A(t) = 0, \\
(1 - p)[\beta^\alpha S(t)A(t) + \rho^\alpha \beta^\alpha S(t)C(t)] + qy^\alpha A(t) - q_3C(t) = 0, \\
(1 - q)y^\alpha A(t) + \eta^\alpha S(t) + r^\alpha C(t) - \delta^\alpha R(t) = 0, \\
\zeta^\alpha \Pi^\alpha - q_4V(t) = 0.
\end{aligned}
\]

Therefore, \((S_\infty, A_\infty, C_\infty, R_\infty, V_\infty)\) is an equilibrium point of the proposed fractional-order epidemiological model (7), and by a similar argument as stated by Theorem 3.1 in [35], we conclude that

\[
\begin{align*}
\lim_{t \to \infty} (\chi(t)) &= D_1, \\
\lim_{t \to \infty} (\chi(t)) &= D_2.
\end{align*}
\]

Hence, the disease endemic state \(D_1\) does not exist whenever \(R_0 < 1\), and so, \(\lim \chi(t) = D_1\) as \(t\) approaches \(\infty\), and if \(R_0 = 1\), then \(D_2 = D_1\) and \(\lim \chi(t) = D_1\) as \(t\) approaches \(\infty\), while on the contrary, if \(R_0 > 1\), then \(D_2\) exists, and thus, \(\lim \chi(t) = D_2\) as \(t\) tends to \(\infty\).

\[\square\]

6. Numerical Simulation

In this section, the numerical simulations are carried out to understand the temporal dynamical behavior corresponding with hepatitis B virus fractional-order epidemiological model (7). This is very important to show the feasibility of the reported work and investigate the validity of the analytical work using large-scale numerical simulation. It is important to point out that, unlike traditional numerical analysis, there are not as many options to choose schemes for the numerical analysis of the fractional-order epidemiological model simulations [36]. Thus, there is a need of extensive research in order to develop new schemes and techniques that are both convergent and robust in the field of
fractional calculus. By following the numerical schemes as reported in [37, 38], we assume $[0, t]$ interval of simulation and $h = 10^{-3}$ is the time step for integration, and $n = T/h$,

\[\begin{align*}
\text{CF} S_{u+1} &= S(0) + (1 - \alpha) \left[ (1 - \zeta^u) \Pi^u - \beta^u S(t) A(t) - \rho^u \beta^u S(t) C(t) - (\delta^u + \eta^u) S(t) + \sigma^u V(t) \right] \\
&+ ah \sum_{k=0}^{u} \left[ (1 - \zeta^k) \Pi^k - \beta^k S(t) A(t) - \rho^k \beta^k S(t) C(t) - (\delta^k + \eta^k) S(t) + \sigma^k V(t) \right], \\
\text{CF} A_{u+1} &= A(0) + (1 - \alpha) \left[ p [\beta^u S(t) A(t) + \rho^u \beta^u S(t) C(t)] - (\delta^u + \gamma^u) A(t) \right] \\
&+ ah \sum_{k=0}^{u} \left[ p [\beta^k S(t) A(t) + \rho^k \beta^k S(t) C(t)] - (\delta^k + \gamma^k) A(t) \right], \\
\text{CF} C_{u+1} &= C(0) + (1 - \alpha) \left[ (1 - p) [\beta^u S(t) A(t) + \rho^u \beta^u S(t) C(t)] + q y^u A(t) - (\delta^u + \epsilon^u + \tau^u) C(t) \right] \\
&+ ah \sum_{k=0}^{u} \left[ (1 - p) [\beta^k S(t) A(t) + \rho^k \beta^k S(t) C(t)] + q y^k A(t) - (\delta^k + \epsilon^k + \tau^k) C(t) \right], \\
\text{CF} R_{u+1} &= (1 - \alpha) \left[ (1 - q) y^u A(t) + \eta^u S(t) + \tau^u C(t) - \theta^u R(t) \right] \\
&+ ah \sum_{k=0}^{u} \left[ (1 - q) y^k A(t) + \eta^k S(t) + \tau^k C(t) - \theta^k R(t) \right] + R(0), \\
\text{CF} V_{u+1} &= (1 - \alpha) \left[ (\zeta^u \Pi^u - (\theta^u + \sigma^u) V(t) \right] + V(0) \\
&+ ah \sum_{k=0}^{u} \left[ (\zeta^k \Pi^k - (\theta^k + \sigma^k) V(t) \right].
\end{align*}\]

Furthermore, the parameters’ value is assumed with biological feasibility; that is, $\zeta = 0.4$, $\Pi = 0.0975$, $\delta = 0.00000456$, $\epsilon = 0.3454$, $\beta = 0.022$, $\rho = 0.048$, $\rho = 0.5$, $q = 0.5$, $y = 0.45$, $\eta = 0.8613$, $\tau = 0.1428$, and $\sigma = 0.06$, and
the initial sizes of compartmental populations are $(100, 90, 80, 70, 60)$. The graph visualizes the dynamics of the acutely infected population $(A(t))$ for different values of the fractional-order parameter $(\alpha)$, and the initial population sizes are $(100, 90, 80, 70, 60)$. By the execution of the above scheme with the stated parameters’ value as above along the initial sizes of populations, we obtain the results as depicted in Figures 1–5. These graphs visualize the dynamical behaviors of the susceptible, the acutely and chronically infected, the recovered, and the vaccinated groups of populations. More precisely, the dynamics of the susceptible individuals for different values of the fractional-order parameter $(\alpha)$ is shown in Figure 1, which demonstrates that if the value of $\alpha$ increases, then the ratio of the susceptible individuals decreases. The graph demonstrates the dynamics of the acutely infected population against different values of the fractional-order parameter $(\alpha)$, and the initial sizes of the population are $(100, 90, 80, 70, 60)$. The graph demonstrates the dynamics of the chronically infected population against different values of the fractional-order parameter $(\alpha)$, and the initial sizes of the population are $(100, 90, 80, 70, 60)$. The graph demonstrates the dynamics of the recovered population against different values of the fractional-order parameter $(\alpha)$, and the initial sizes of the population are $(100, 90, 80, 70, 60)$. The graph demonstrates the dynamics of the vaccinated population against different values of the fractional-order parameter $(\alpha)$, and the initial sizes of the population are $(100, 90, 80, 70, 60)$. The initial sizes of compartmental populations are $(100, 90, 80, 70, 60)$.
decreases. This shows that the fractional-order parameter and the susceptible population are inversely proportional to each other. Similarly, the acutely and chronically infected population are also inversely proportional to the fractional-order parameter \( \alpha \) as shown in Figures 2 and 3, respectively, while the dynamics of recovered individuals reveals that there is a direct relation between the fractional-order parameter \( \alpha \) and the recovered population, i.e., whenever the value of \( \alpha \) increases, the size of the recovered population also increases as depicted in Figure 4. The dynamics of the vaccinated group of population is described in Figure 5, which demonstrates that the fractional-order parameter \( \alpha \) has a negative impact on the dynamics of the vaccinated population, i.e., whenever the value of \( \alpha \) increases, the size of the population group \( V(t) \) decreases. This analysis reveals that the CF fractional-order model presents more valuable outputs regarding the behavior of compartmental populations which usually could not be obtained in case of the classical model.

7. Conclusion

The work carried out in this study consists of a new epidemiological model related to dynamics of hepatitis B virus transmission. We used the CF operator and investigated the dynamics of hepatitis B virus. We formulated the proposed model first and then fractionalized by using the Caputo–Fabrizio operator with dimensional balance in respect of involved epidemic parameters. We used the fixed point theory and rigorously showed that the model under the CF operator possesses a unique solution. We also discussed biological as well as mathematical feasibility of the proposed model by proving that the solutions of the model are bounded and positive. Moreover, the basic reproductive number is calculated, and the stability analysis of the steady states of the proposed fractional-order epidemiological model is shown. At the end, we presented some numerical simulations to show the relation between compartmental populations and the fractional-order operator. Thus, the major findings of this study show that the CF fractional-order operator is the best choice instead of the classical order.

Data Availability

No data were used to support this study.

Conflicts of Interest

The authors declare that they have no conflicts of interest.

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