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Fractional-Order HBV Dynamics with Deep Neural Surrogates: A General Conformable Framework

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Abstract

The model suggested in this paper is a general conformable fractional-order HBV model with six compartments (susceptible, acute/chronic infected, hospitalized, recovered, vaccinated). The replacement of the classical derivative by a general conformable derivative adds a local time-scaling kernel which is flexible. The model maintains epidemiological structure and time-deformation behaviors are enriched. Theoretical analysis demonstrates positivity, boundedness, global existence, uniqueness and Ulam-Hyers stability, which establish biological and mathematical well-posedness. A predictor-corrector scheme which uses the local conformable structure gives almost second-order convergence (relative L_2 error from 8.01×10^{-5} to 1.21×10^{-6}). A deep neural network surrogate that is trained on 15,000 transitions generated by the solver in one step is evaluated using six optimizers; NAdam has the highest predictive performance. The choice of kernel has a great impact on both HBV dynamics and surrogate learnability: the rational kernel provides the lowest final infected burden and highest rollout accuracy (RMSE = 20.25, $R^2 = 0.896$). The framework shows that general conformable fractional modeling with neural surrogates is effective to analyze HBV flexibly and efficiently.

Keywords: Hepatitis B virus; general conformable fractional derivative; conformable kernels; positivity and boundedness; Ulam–Hyers stability; predictor–corrector scheme; deep neural network surrogate; optimizer benchmarking.

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1 Introduction

Hepatitis B virus (HBV) infection has been a significant global public health problem even though effective vaccines have been developed, and significant advances have been made in the diagnosis, prevention, and antiviral therapy. The world health organization states that HBV still has hundreds of millions of victims across the globe and still causes a significant amount of cirrhosis, hepatocellular carcinoma, and HBV-related mortality [1]. Besides vaccination and classical antiviral treatment regimens, recent biomedical research has placed significant importance on the viral lifecycle sensitivity, treatment effectiveness, and combination therapies that can lower the levels of the hepatitis B surface antigen, which also inspires the necessity of the advanced mathematical and computational frameworks of modeling as well [2–4].

It is well known that mathematical models are useful in the analysis of HBV transmission, intervention strategies, and the mechanism of disease persistence and control. To explore the vaccination, treatment, environmental changes, demographic fluctuation, and various ways of transmissions, classical and stochastic models have been put forward [5–8]. In more recent mathematical studies, dual transmission pathways, immune-response dynamics, and treatment-oriented HBV dynamics have also been studied, and reflect the increasing popularity of combining mechanistic epidemiological argument with more biological assumptions [3, 4, 9]. These advances attest to the fact that HBV modeling is a vibrant and dynamic field of study.

Fractional calculus has become an effective modeling tool in infectious disease in the past few years as it can model memory and hereditary effects that classical integer-order differential equations frequently overlook. Fractional-order formulations in the HBV setting have been applied to investigate the effect of vaccination, treatment, hospitalization, and nonlinear transmission effects and offer more dynamic behavior than the integer-order analogs [10, 11]. The recent studies on HBV have utilized diverse fractional operators such as conformable, Riemann, Liouville, fractal-fractional, Mittag, Leffler-type and various memory-dependent operators [12–15]. These publications demonstrate that the selection of fractional operator may have a strong impact on the

dynamics of epidemics predicted, the qualitative stability outcome, and the effectiveness of the numerical approximations.

The conformable fractional derivative is one of the numerous fractional operators proposed in the literature that have received a lot of attention due to its ability to maintain various classical differential rules, as well as being relatively straightforward to operate with [16, 17]. Based on this concept, Zhao and Luo proposed the so-called general conformable fractional derivative (GCFD) that extends the usual conformable derivative by substituting the factor $t^{1-\alpha}$ with a wider admissible conformable kernel [18]. This generalization increases the modeling flexibility preserving the local structure and the useful representation.

$$D_{\psi}^p f(t) = \psi(t, p) f'(t),$$

that the operator is especially appealing to analysis and numerical implementation. Generalized conformable derivatives have also been effectively used in nonlinear studies of stability, with practical arguments of stability and Lyapunov on systems with general conformable dynamics having been developed [19]. However, although the HBV and fractional-calculus literature is very rich, little has been done regarding the use of the general conformable framework in the HBV epidemic modeling.

Meanwhile, the numerical dynamics of a fractional and generalized-fractional models can be computationally intensive in the case where the numerical dynamics of the model requires a range of parameter values, kernel selections, and the fractional order to be considered. This difficulty has helped to increase the intersection of the scientific machine learning and the differential-equation modeling. Scientific machine learning reviews in the recent past have pointed out the growing importance of deep surrogates, neural operators and hybrid solver-learning systems in accelerating the approximation of complex dynamical systems solving differential equations [20, 21]. This move has already begun to be reflected in the HBV context: the feed-forward neural networks and neural-network-assisted models have been used recently to estimate stochastic and fractional HBV models, thus proving the feasibility of machine learning in epidemiological simulation and analysis [9, 22, 23]. These advances imply that it is timely and promising to combine conformable-fractional modeling and neural surrogates.

Based on these observations, this paper constructs a general conformable fractional HBV model that has six epidemiology compartments that comprise of susceptible, acutely infected, chronically infected, hospitalized, recovered and vaccinated groups. The proposed model is a generalization of the classical and standard conformable HBV formulations which includes a flexible admissible conformable kernel in the time derivative. In the theoretical perspective, we formulate positivity, boundedness, strict positivity of the total population, global existence, uniqueness and UlamHyers stability. In the numerical perspective, we take advantage of the local form of the general conformable derivative in order to obtain a stretched-time predictor-corrector scheme. Finally, scientific-machine-learning is a viewpoint, in which we construct a deep neural network surrogate, which is trained on the solver-generated trajectories and optimizer benchmarking over diverse admissible kernels.

The main innovation of the provided work is a combination of three complementary elements into one HBV modeling tool (i) the general conformable fractional epidemiological model, (ii) the mathematically consistent and efficient numerical approximation approach, and (iii) the deep-learning surrogate layer to give high-speed simulation and optimizer-aware prediction. In this respect, the paper will not only be used in modelling the HBV transmission, but also generalization of data-driven computational approaches of generalized fractional dynamical systems. The rest of the paper is structured in the following way.

In Section 2, the principal concepts of general conformable fractional calculus are reviewed.. The proposed HBV model is formulated in Section 3. Its qualitative properties are defined in Section 4. Section 5 displays the predictor-corrector solver of the stretched time. The deep neural surrogate as well as the solver-learning pipeline are presented in Sections 6 and 7. Lastly, in Section 8, the numerical experiments are described and the effect of kernels and optimizers on the dynamic and predictive behaviour of the model is discussed.

2 Preliminaries on General Conformable Fractional Calculus

Fractional calculus now forms a useful paradigm and framework in modeling complex dynamical systems, particularly where classical integer-order operators are too inflexible to be used to model anomalous or scale-dependent dynamics. Of the numerous possible fractional operators, the so-called conformable fractional derivative has received a lot of interest due to its ability to retain much of the classical differential properties, including that it is linear, the product rule, the quotient rule, and the chain rule, and yet it is simpler to operate with than a large number of the nonlocal operators [16, 17]. Zhao and Luo introduce the general conformable fractional derivative (GCFD), which is a generalization of the standard conformable derivative, where the factor $t^{1-\alpha}$ is replaced by a more general conformable function of the fractional conformable nature, which expands the modeling capabilities but still follows the same rules of local character and classical-like calculus [18]. This nonlinear generalization then has been applied in nonlinear systems, stability and control [19].

To be complete, we recall first the conformable fractional derivative proposed by Khalil et al. of a function $f : [0, \infty) \rightarrow \mathbb{R}$ with an order $\alpha \in (0, 1]$ by [16, 17].

$$T_\alpha(f)(t) = \lim_{\varepsilon \rightarrow 0} \frac{f(t + \varepsilon t^{1-\alpha}) - f(t)}{\varepsilon}, \quad t > 0. \quad (1)$$

If f is differentiable, then the above operator reduces to

$$T_\alpha(f)(t) = t^{1-\alpha} f'(t), \quad (2)$$

which explains why the conformable derivative preserves many classical differential identities [16, 17].

A key observation made by Zhao and Luo is that the factor $t^{1-\alpha}$ in (1) is not essential and can be replaced by a broader class of functions satisfying suitable consistency conditions. This leads to the *general conformable fractional derivative* (GCFD) [18]. Let $\psi(u, p)$ be a *fractional conformable function* and let $p \in (0, 1]$. Then the GCFD of a function f is defined by

$$D_\psi^p f(u) = \lim_{\varepsilon \rightarrow 0} \frac{f(u + \varepsilon \psi(u, p)) - f(u)}{\varepsilon}, \quad u > 0. \quad (3)$$

The function $\psi(u, p)$ is required to satisfy the consistency conditions

$$\psi(u, 1) = 1, \quad (4)$$

and

$$\psi(\cdot, p) \neq \psi(\cdot, q), \quad p \neq q, \quad p, q \in (0, 1], \quad (5)$$

so that the operator reduces to the classical first-order derivative when $p = 1$, while different orders correspond to genuinely different dynamics [18]. The standard conformable derivative is recovered as the special case

$$\psi(u, p) = u^{1-p}. \quad (6)$$

One of the most important features of the GCFD is that, for differentiable functions, it admits the simple representation

$$D_\psi^p f(u) = f'(u) \psi(u, p), \quad (7)$$

which follows directly from the general conformable construction [18]. Consequently, the operator preserves several classical rules. If f and g are p -differentiable, then [16–18]

$$D_\psi^p (af + bg) = a D_\psi^p f + b D_\psi^p g, \quad (8)$$

$$D_\psi^p (c) = 0, \quad (9)$$

$$D_\psi^p (fg) = f D_\psi^p g + g D_\psi^p f, \quad (10)$$

$$D_\psi^p \left(\frac{f}{g} \right) = \frac{g D_\psi^p f - f D_\psi^p g}{g^2}, \quad g \neq 0, \quad (11)$$

and, whenever f is differentiable,

$$D_\psi^p (f \circ g)(u) = f'(g(u)) D_\psi^p g(u). \quad (12)$$

These identities are especially useful in the derivation and analysis of compartmental epidemic models, where nonlinear incidence, vaccination, recovery, and transition terms frequently involve products and compositions of state variables.

The integral operator associated with the GCFD is defined by Zhao and Luo as [18]

$$I_a^{p, \psi} f(u) = \int_a^u \frac{f(t)}{\psi(t, p)} dt, \quad u \geq a \geq 0. \quad (13)$$

When $\psi(u, p) = u^{1-p}$, the operator (13) reduces to the integral associated with the standard conformable derivative [16, 18].

The following fundamental relations hold [18]:

$$D_\psi^p (I_a^{p, \psi} f(u)) = f(u), \quad (14)$$

and, for differentiable f ,

$$I_a^{p, \psi} (D_\psi^p f(u)) = f(u) - f(a). \quad (15)$$

These two identities will be used to transform the proposed HBV model into an equivalent integral system, which is convenient for both theoretical analysis and numerical approximation.

Throughout this work, the fractional conformable function $\psi(\cdot, p)$ is assumed to be positive and sufficiently regular so that the derivative and integral operators are well defined. In particular, we assume that

$$\psi(t, p) > 0, \quad t > 0, \quad p \in (0, 1], \quad (16)$$

and that $1/\psi(\cdot, p)$ is locally integrable on the interval of interest. These assumptions ensure that the integral operator (13) is meaningful and that the generalized conformable dynamics remain well posed [18, 19]. In the numerical section, particular choices of ψ will be considered to illustrate how different conformable kernels modify the HBV dynamics.

The significant opportunity of the generalized conformable framework is that the scheme has a definite physical and geometrical interpretation. Specifically, Zhao and Luo demonstrate that generalized conformable derivative could be considered as the alteration of the classical derivative by the use of direction and scale dependent factor denoted by $\psi(u, p)$ through which offers a physically meaningful manner of deforming the local change of rate [18]. The latter characteristic renders the GCFD particularly appealing to epidemiological modeling, with the order p and conformable function ψ possibly being viewed as the tuning parameters, by which the implicit effective temporal scaling of the disease dynamics can be modified.

The above preliminaries present the analytical background necessary to establish the proposed general conformable fractional HBV model and then to establish its qualitative and quantitative aspects in the following sections.

3 General Conformable Fractional HBV Model

In this section, we formulate a general conformable fractional model for the transmission dynamics of hepatitis B virus (HBV). The proposed model is constructed by extending a six-compartment HBV structure that incorporates susceptible individuals, acutely infected individuals, chronically infected individuals, hospitalized patients, recovered individuals, and vaccinated individuals [6]. In the present work, the classical time derivative is replaced by the general conformable fractional derivative introduced in Section 2, thereby yielding a more flexible temporal scaling of the disease dynamics [18]. This formulation may be viewed as a generalized conformable extension of HBV models previously developed in the classical and conformable-fractional settings [6, 10]. Generalized conformable models have also been successfully considered in nonlinear systems and stability studies, which further supports the modeling framework adopted here [19].

Let

$$S(t), \quad I_1(t), \quad I_2(t), \quad H(t), \quad R(t), \quad V(t)$$

denote, respectively, the sizes of the susceptible, acutely infected, chronically infected, hospitalized, recovered, and vaccinated subpopulations at time $t \geq 0$ [6]. The total population is given by

$$N(t) = S(t) + I_1(t) + I_2(t) + H(t) + R(t) + V(t). \quad (17)$$

The model assumptions are summarized as follows:

- A recruitment flow enters the population at rate Ψ , with a proportion Π entering the susceptible class and the remaining proportion $1 - \Pi$ entering the vaccinated class.
- Susceptible individuals become acutely infected through effective contact with acute and chronic infectives at rates

$$\frac{\beta_1 S I_1}{N} \quad \text{and} \quad \frac{\beta_2 S I_2}{N},$$

respectively.

- Vaccinated individuals are not assumed to be perfectly protected; individuals with weak or waning immunity may become infected through contact with acute and chronic infectives at rates

$$\frac{\beta_3 V I_1}{N} \quad \text{and} \quad \frac{\beta_4 V I_2}{N},$$

respectively.

- Vaccinated individuals lose immunity at rate η and return to the susceptible class, while susceptible individuals are vaccinated at rate γ .
- Acute infectives progress to the chronic stage at rate α_1 , recover directly at rate α_2 , and move to the hospitalized compartment at rate ρ_1 .

- Chronically infected individuals recover at rate γ_1 , are hospitalized at rate ρ_2 , and experience disease-induced mortality at rate γ_2 .
- Hospitalized individuals recover at rate ρ_3 and experience disease-induced mortality at rate ρ_4 .
- All compartments are subject to natural mortality at rate χ .

Using the general conformable fractional derivative D_ψ^p of order $p \in (0, 1]$, the proposed HBV model is written as

$$D_\psi^p S(t) = \Psi\Pi - \frac{\beta_1 S(t)I_1(t)}{N(t)} - \frac{\beta_2 S(t)I_2(t)}{N(t)} + \eta V(t) - (\gamma + \chi)S(t), \quad (18)$$

$$D_\psi^p I_1(t) = \frac{\beta_1 S(t)I_1(t)}{N(t)} + \frac{\beta_2 S(t)I_2(t)}{N(t)} + \frac{\beta_3 V(t)I_1(t)}{N(t)} + \frac{\beta_4 V(t)I_2(t)}{N(t)} - (\alpha_1 + \alpha_2 + \rho_1 + \chi)I_1(t), \quad (19)$$

$$D_\psi^p I_2(t) = \alpha_1 I_1(t) - (\gamma_1 + \rho_2 + \gamma_2 + \chi)I_2(t), \quad (20)$$

$$D_\psi^p H(t) = \rho_1 I_1(t) + \rho_2 I_2(t) - (\rho_3 + \rho_4 + \chi)H(t), \quad (21)$$

$$D_\psi^p R(t) = \gamma_1 I_2(t) + \alpha_2 I_1(t) + \rho_3 H(t) - \chi R(t), \quad (22)$$

$$D_\psi^p V(t) = \Psi(1 - \Pi) + \gamma S(t) - \frac{\beta_3 V(t)I_1(t)}{N(t)} - \frac{\beta_4 V(t)I_2(t)}{N(t)} - (\eta + \chi)V(t). \quad (23)$$

The model is equipped with nonnegative initial conditions

$$S(0) = S_0 > 0, \quad I_1(0) = I_{10} > 0, \quad I_2(0) = I_{20} > 0, \quad H(0) = H_0 \geq 0, \quad R(0) = R_0 \geq 0, \quad V(0) = V_0 \geq 0. \quad (24)$$

The state variables and transitions in (18)–(23) preserve the epidemiological structure of the corresponding integer-order HBV model, while the temporal derivative is generalized through the conformable function $\psi(t, p)$.

For convenience, define the state vector

$$X(t) = [S(t) \quad I_1(t) \quad I_2(t) \quad H(t) \quad R(t) \quad V(t)]^\top \in \mathbb{R}_+^6.$$

Then the system (18)–(23) can be written compactly as

$$D_\psi^p X(t) = F(X(t)), \quad X(0) = X_0, \quad (25)$$

where $F : \mathbb{R}_+^6 \rightarrow \mathbb{R}^6$ is the nonlinear vector field induced by (18)–(23).

Using the general conformable integral introduced in Section 2, system (25) is equivalently represented by the Volterra-type integral equation

$$X(t) = X_0 + \int_0^t \frac{F(X(s))}{\psi(s, p)} ds, \quad t \geq 0. \quad (26)$$

This integral formulation will be the basis for the analysis of positivity, boundedness, existence, uniqueness, and Ulam–Hyers stability in the next section.

Moreover, since the general conformable derivative satisfies

$$D_\psi^p X(t) = \psi(t, p) X'(t)$$

for classically differentiable X , system (25) can also be written in the equivalent weighted ODE form

$$X'(t) = \frac{F(X(t))}{\psi(t, p)}, \quad t > 0. \quad (27)$$

Defining the stretched-time variable

$$\tau = \Theta_p(t) := \int_0^t \frac{1}{\psi(s, p)} ds, \quad (28)$$

the model further reduces to the standard first-order system

$$\frac{dX}{d\tau} = F(X), \quad X(0) = X_0. \quad (29)$$

This equivalent representation will play a central role in the numerical approximation developed later.

The proposed model includes several important limiting cases:

- If $p = 1$, then $\psi(t, 1) = 1$, and the general conformable derivative reduces to the classical first-order derivative. Consequently, system (18)–(23) reduces to the corresponding integer-order HBV model.
- If $\psi(t, p) = t^{1-p}$, then the model reduces to the standard conformable fractional HBV formulation associated with Khalil's derivative. In this sense, the present model generalizes the ordinary conformable derivative case.
- Different choices of the fractional conformable function $\psi(t, p)$ produce different temporal scalings of the HBV dynamics, which allows the model to interpolate between alternative local fractional time-deformation mechanisms.

Therefore, model (18)–(23) provides a unified general conformable framework for investigating HBV transmission under flexible local fractional dynamics.

4 Qualitative Analysis: Positivity, Boundedness, Existence, Uniqueness, and Ulam–Hyers Stability

In this section, we investigate the well-posedness and qualitative properties of the proposed general conformable fractional HBV model (18)–(23). More precisely, we establish the positivity and boundedness of solutions, prove global existence and uniqueness, and derive an Ulam–Hyers stability result. These properties are fundamental for the biological consistency and mathematical reliability of the model.

Throughout this section, we impose the following assumptions:

- (A1) The fractional conformable function $\psi(\cdot, p)$ is continuous and positive on $[0, T]$ for every $T > 0$, and

$$\int_0^T \frac{1}{\psi(s, p)} ds < \infty.$$

- (A2) All epidemiological parameters are nonnegative, with

$$\Psi > 0, \quad \chi > 0, \quad \Pi \in [0, 1].$$

- (A3) The initial data satisfy

$$S_0 > 0, \quad I_{10} > 0, \quad I_{20} > 0, \quad H_0 \geq 0, \quad R_0 \geq 0, \quad V_0 \geq 0.$$

For convenience, define the state vector

$$X(t) = (S(t), I_1(t), I_2(t), H(t), R(t), V(t))^T \in \mathbb{R}_+^6,$$

and denote by $F(X)$ the nonlinear vector field of system (25). By the general conformable integral operator introduced in Section 2, the model is equivalent to

$$X(t) = X_0 + \int_0^t \frac{F(X(s))}{\psi(s, p)} ds. \quad (30)$$

We also define the generalized conformable time-stretching function

$$\Theta_p(t) := \int_0^t \frac{1}{\psi(s, p)} ds, \quad t \geq 0. \quad (31)$$

Under assumption (A1), $\Theta_p(t)$ is well defined, continuous, and nondecreasing.

Moreover, since the general conformable derivative satisfies

$$D_\psi^p f(t) = \psi(t, p) f'(t)$$

for differentiable functions and $\psi(t, p) > 0$, any conformably differentiable solution of (25) is classically differentiable on its interval of existence, and the sign of $D_\psi^p f$ is equivalent to the sign of f' . This observation will be used repeatedly in the sequel.

We begin with the positivity of solutions. This property is essential from the epidemiological viewpoint, since each state variable represents a population class and therefore must remain nonnegative for all times.

Theorem 4.1 (Positivity). *Let assumptions (A1)–(A3) hold. Then every solution of system (18)–(23) with initial condition (24) remains nonnegative for all $t \geq 0$. In particular, the positive orthant $\mathbb{R}_+^6$ is positively invariant.*

Proof. Because $\psi(t, p) > 0$, the sign of $D_\psi^p X_i(t)$ coincides with the sign of the classical derivative $X_i'(t)$. Therefore, to prove positive invariance, it suffices to verify that the vector field points inward on each boundary hyperplane of $\mathbb{R}_+^6$.

If $S(t) = 0$, then from (18),

$$D_\psi^p S(t) = \Psi\Pi + \eta V(t) \geq 0.$$

If $I_1(t) = 0$, then from (19),

$$D_\psi^p I_1(t) = \frac{\beta_2 S(t) I_2(t)}{N(t)} + \frac{\beta_4 V(t) I_2(t)}{N(t)} \geq 0.$$

If $I_2(t) = 0$, then from (20),

$$D_\psi^p I_2(t) = \alpha_1 I_1(t) \geq 0.$$

If $H(t) = 0$, then from (21),

$$D_\psi^p H(t) = \rho_1 I_1(t) + \rho_2 I_2(t) \geq 0.$$

If $R(t) = 0$, then from (22),

$$D_\psi^p R(t) = \gamma_1 I_2(t) + \alpha_2 I_1(t) + \rho_3 H(t) \geq 0.$$

If $V(t) = 0$, then from (23),

$$D_\psi^p V(t) = \Psi(1 - \Pi) + \gamma S(t) \geq 0.$$

Therefore, whenever a trajectory reaches the boundary of $\mathbb{R}_+^6$, the corresponding derivative is nonnegative, so the solution cannot cross the boundary toward negative values. Hence, $\mathbb{R}_+^6$ is positively invariant. $\square$

Remark 4.2. Theorem 4.1 guarantees the epidemiological admissibility of the model. In particular, once the initial state is biologically meaningful, all compartments remain nonnegative throughout the evolution, which confirms that the general conformable formulation preserves the population interpretation of the HBV system.

We next establish a boundedness property for the total population. This result is important because it prevents unphysical population blow-up and provides a natural invariant region for the qualitative analysis.

Theorem 4.3 (Boundedness). *Under assumptions (A1)–(A3), every nonnegative solution of system (18)–(23) is bounded on $[0, \infty)$. More precisely,*

$$N(t) \leq \max \left\{ N_0, \frac{\Psi}{\chi} \right\}, \quad t \geq 0, \quad (32)$$

where $N_0 = N(0)$.

Proof. Summing (18)–(23) yields

$$D_\psi^p N(t) = \Psi - \chi N(t) - \gamma_2 I_2(t) - \rho_4 H(t). \quad (33)$$

Since $I_2(t) \geq 0$ and $H(t) \geq 0$ by Theorem 4.1, we obtain

$$D_\psi^p N(t) \leq \Psi - \chi N(t). \quad (34)$$

Using the identity $D_\psi^p N(t) = \psi(t, p)N'(t)$ for differentiable N , inequality (34) becomes

$$N'(t) \leq \frac{\Psi - \chi N(t)}{\psi(t, p)}.$$

Let $Y(t)$ solve

$$D_\psi^p Y(t) = \Psi - \chi Y(t), \quad Y(0) = N_0.$$

Then, by the variation-of-constants formula associated with the generalized conformable integral,

$$Y(t) = N_0 e^{-\chi \Theta_p(t)} + \frac{\Psi}{\chi} \left(1 - e^{-\chi \Theta_p(t)} \right). \quad (35)$$

By the comparison principle, $N(t) \leq Y(t)$ for all $t \geq 0$. Since $0 < e^{-\chi \Theta_p(t)} \leq 1$, (35) immediately implies

$$N(t) \leq \max \left\{ N_0, \frac{\Psi}{\chi} \right\}, \quad t \geq 0.$$

This completes the proof. $\square$

Remark 4.4. Theorem 4.3 is an explicit upper bound on the total population as well as demonstrates that the model is in a biologically realistic regime. The bound is only based on the initial total population and the ratio of recruitment to mortality, Ψ/χ which is a natural threshold value in demographic epidemic models.

As a direct consequence of boundedness and positivity, we can define a positively invariant feasible region where all subsequent analysis will be carried out.

Corollary 4.5. *The set*

$$\Omega := \{X \in \mathbb{R}_+^6 : 0 \leq S + I_1 + I_2 + H + R + V \leq M\}, \quad M := \max \left\{ N_0, \frac{\Psi}{\chi} \right\}, \quad (36)$$

is positively invariant for system (18)–(23).

Remark 4.6. The biologically feasible set of the model is determined in Corollary 4.5. The region will be a key point of the existence, uniqueness and stability analysis, as it is a limited territory where the vector field is well defined and epidemiologically significant.

Before proving existence and uniqueness, it is useful to ensure that the total population never vanishes. This guarantees that the nonlinear incidence terms remain well defined for all times.

Lemma 4.7. *Let assumptions (A1)–(A3) hold. Then the total population satisfies*

$$N(t) > 0, \quad t \geq 0.$$

Proof. Since $N(0) = N_0 > 0$, it is enough to show that $N(t)$ cannot reach zero. Suppose, by contradiction, that there exists $t^* > 0$ such that $N(t^*) = 0$. By Theorem 4.1, all compartments are nonnegative, hence $I_2(t^*) \geq 0$ and $H(t^*) \geq 0$. Evaluating (33) at $t = t^*$ gives

$$D_\psi^p N(t^*) = \Psi - \gamma_2 I_2(t^*) - \rho_4 H(t^*) \geq \Psi > 0.$$

Because $\psi(t^*, p) > 0$, this implies $N'(t^*) > 0$, so the trajectory cannot cross toward the nonpositive region at t^* . This contradicts $N(t^*) = 0$ as a first hitting time. Therefore, $N(t) > 0$ for all $t \geq 0$. $\square$

Remark 4.8. Lemma 4.7 is a key technical result because it prevents singularity in the rational incidence terms. In particular, it guarantees that the denominator $N(t)$ never vanishes, so the vector field remains regular on the entire interval of existence.

We are now in a position to establish the global well-posedness of the model. The previous results ensure that the state remains in the feasible region and that the vector field is free of singularities.

Theorem 4.9 (Global existence and uniqueness). *Under assumptions (A1)–(A3), system (18)–(23) admits a unique global solution*

$$X \in C([0, \infty), \Omega).$$

Proof. By Lemma 4.7, the denominator $N(t)$ in the incidence terms remains strictly positive for all $t \geq 0$. Hence the vector field $F(X)$ is well defined on the positively invariant set Ω . Since Ω is bounded and the rational incidence functions have strictly positive denominator, F is locally Lipschitz on Ω . Therefore, for any finite interval $[0, T]$, the weighted vector field

$$G(t, X) := \frac{F(X)}{\psi(t, p)}$$

is continuous in t and locally Lipschitz in X , because $\psi(t, p) > 0$ and continuous on $[0, T]$.

Using the equivalent weighted ODE form

$$X'(t) = \frac{F(X(t))}{\psi(t, p)} = G(t, X(t)), \quad (37)$$

the classical Picard–Lindelöf theorem guarantees a unique local solution on some interval $[0, T_1]$. By Corollary 4.5, this solution remains in the bounded positively invariant set Ω , and by Lemma 4.7 the denominator $N(t)$ never vanishes. Hence no finite-time blow-up and no singularity can occur. Consequently, the local solution can be continued step by step to all of $[0, \infty)$.

Therefore, system (18)–(23) admits a unique global solution in $C([0, \infty), \Omega)$. $\square$

Remark 4.10. Theorem 4.9 confirms that the proposed GCFD-HBV model is globally well posed. This means that, for any biologically admissible initial condition, the model generates a unique trajectory that remains meaningful for all future times.

We finally investigate the robustness of the model with respect to small perturbations. For this purpose, we use the notion of Ulam–Hyers stability on a finite time interval.

Definition 4.11. System (25) is said to be *Ulam–Hyers stable* on $[0, T]$ if there exists a constant $C_T > 0$ such that, for every $\varepsilon > 0$ and every function $Y \in C([0, T], \Omega)$ satisfying

$$\left\| D_\psi^p Y(t) - F(Y(t)) \right\| \leq \varepsilon, \quad t \in [0, T], \quad (38)$$

there exists an exact solution X of (25) such that

$$\|Y(t) - X(t)\| \leq C_T \varepsilon, \quad t \in [0, T]. \quad (39)$$

Remark 4.12. Definition 4.11 formalizes the idea that approximate trajectories of the GCFD-HBV system remain close to true trajectories whenever the residual perturbation is sufficiently small. This notion is particularly relevant in the present work because numerical approximations and learning-based surrogates inherently introduce small modeling errors.

The next theorem shows that the proposed HBV model enjoys this robustness property under a standard Lipschitz assumption on the nonlinear vector field.

Theorem 4.13 (Ulam–Hyers stability). *Assume that F is Lipschitz continuous on Ω with constant $L > 0$. Then system (25) is Ulam–Hyers stable on every finite interval $[0, T]$. More precisely, if X and Y have the same initial condition and Y satisfies (38), then*

$$\|Y(t) - X(t)\| \leq \varepsilon \Theta_p(T) e^{L\Theta_p(T)}, \quad t \in [0, T]. \quad (40)$$

Proof. Let Y be an ε -approximate solution of (25). Then there exists a perturbation term $R(t)$ such that

$$D_\psi^p Y(t) = F(Y(t)) + R(t), \quad \|R(t)\| \leq \varepsilon, \quad t \in [0, T].$$

Using the generalized conformable integral, we can write

$$Y(t) = X_0 + \int_0^t \frac{F(Y(s))}{\psi(s, p)} ds + \int_0^t \frac{R(s)}{\psi(s, p)} ds. \quad (41)$$

Let X be the exact solution of (25) with the same initial condition X_0 . Subtracting (30) from (41), we obtain

$$Y(t) - X(t) = \int_0^t \frac{F(Y(s)) - F(X(s))}{\psi(s, p)} ds + \int_0^t \frac{R(s)}{\psi(s, p)} ds.$$

Taking norms and using the Lipschitz property of F , we get

$$\|Y(t) - X(t)\| \leq L \int_0^t \frac{\|Y(s) - X(s)\|}{\psi(s, p)} ds + \varepsilon \int_0^t \frac{1}{\psi(s, p)} ds.$$

Hence,

$$\|Y(t) - X(t)\| \leq L \int_0^t \frac{\|Y(s) - X(s)\|}{\psi(s, p)} ds + \varepsilon \Theta_p(t).$$

Applying Gronwall's inequality with respect to the stretched-time variable $\Theta_p(t)$ yields

$$\|Y(t) - X(t)\| \leq \varepsilon \Theta_p(t) e^{L\Theta_p(t)} \leq \varepsilon \Theta_p(T) e^{L\Theta_p(T)}, \quad t \in [0, T].$$

Therefore, Definition 4.11 holds with

$$C_T = \Theta_p(T) e^{L\Theta_p(T)}.$$

This completes the proof. $\square$

Remark 4.14. Theorem 4.13 shows that the proposed HBV model is robust with respect to small perturbations in the general conformable differential equation. In particular, approximate numerical or learning-based solutions remain close to the exact model trajectory, provided that the residual error is small. This property is particularly relevant for the numerical and deep-learning surrogate framework developed in the subsequent sections.

5 Numerical Method: General Conformable Predictor–Corrector Scheme

The attached HBV draft paper employs an integral reformulation of a fractional model together with a polynomial-interpolation-based numerical strategy tailored to the Atangana–Baleanu–Caputo derivative with Mittag–Leffler kernel [12]. In the present work, however, the proposed HBV system is governed by the *general conformable fractional derivative* (GCFD), whose structure is fundamentally different from the ABC formulation. In particular, the GCFD is a *local* operator and satisfies the identity

$$D_\psi^p X(t) = \psi(t, p) X'(t)$$

for differentiable state variables [18]. Consequently, the GCFD-HBV model can be reduced to an ordinary differential system after an appropriate time transformation, and this motivates the predictor–corrector scheme developed below. This approach preserves the simplicity of classical ODE discretizations while remaining fully consistent with the generalized conformable framework [18, 19].

Recall from Section 3 that the general conformable HBV model can be written compactly as

$$D_\psi^p X(t) = F(X(t)), \quad X(0) = X_0, \quad (42)$$

where

$$X(t) = [S(t) \quad I_1(t) \quad I_2(t) \quad H(t) \quad R(t) \quad V(t)]^\top$$

and $F(X)$ is the nonlinear vector field induced by (18)–(23). Since the GCFD satisfies

$$D_\psi^p X(t) = \psi(t, p) X'(t),$$

system (42) is equivalent to the weighted ODE

$$X'(t) = \frac{F(X(t))}{\psi(t, p)}, \quad t > 0. \quad (43)$$

Define the generalized conformable time-stretching function

$$\Theta_p(t) = \int_0^t \frac{1}{\psi(s, p)} ds, \quad t \geq 0. \quad (44)$$

Introducing the stretched-time variable

$$\tau = \Theta_p(t), \quad (45)$$

the chain rule yields

$$\frac{dX}{dt} = \frac{dX}{d\tau} \frac{d\tau}{dt} = \frac{1}{\psi(t, p)} \frac{dX}{d\tau}.$$

Substituting into (43), we obtain the standard first-order system

$$\frac{dX}{d\tau} = F(X), \quad X(0) = X_0. \quad (46)$$

Hence, the numerical approximation of the GCFD-HBV model reduces to the numerical integration of the classical ODE (46) in the stretched-time variable τ . This is the key simplification specific to the general conformable framework and does not hold for nonlocal operators such as the ABC derivative used in the attached draft paper [12, 18].

Let the physical time interval $[0, T]$ be partitioned uniformly as

$$0 = t_0 < t_1 < \cdots < t_N = T, \quad t_n = nh, \quad h = \frac{T}{N}.$$

The corresponding stretched-time nodes are defined by

$$\tau_n = \Theta_p(t_n) = \int_0^{t_n} \frac{1}{\psi(s, p)} ds, \quad n = 0, 1, \dots, N. \quad (47)$$

Denote the numerical approximation of $X(t_n)$ by X_n , and let

$$F_n := F(X_n), \quad n = 0, 1, \dots, N.$$

Then the effective step-size in stretched time is

$$\Delta\tau_n = \tau_{n+1} - \tau_n = \Theta_p(t_{n+1}) - \Theta_p(t_n) = \int_{t_n}^{t_{n+1}} \frac{1}{\psi(s, p)} ds. \quad (48)$$

A natural explicit predictor for (46) is obtained by the forward Euler method in stretched time:

$$\hat{X}_{n+1} = X_n + \Delta\tau_n F(X_n). \quad (49)$$

This predictor provides a first approximation of the solution at the next node.

To improve accuracy, we apply the trapezoidal rule to (46), which yields the corrector

$$X_{n+1} = X_n + \frac{\Delta\tau_n}{2} \left[F(X_n) + F(\hat{X}_{n+1}) \right]. \quad (50)$$

Equations (49)–(50) define a one-step general conformable predictor–corrector scheme. Unlike memory-based product-integration methods used for nonlocal fractional kernels, this method is local in time and depends only on the current state and the stretched-time increment $\Delta\tau_n$, which is consistent with the local nature of the GCFD [12, 18].

When the generalized conformable function is chosen as

$$\psi(t, p) = t^{1-p}, \quad p \in (0, 1], \quad (51)$$

the generalized conformable derivative reduces to the standard conformable derivative [18]. In this case,

$$\Theta_p(t) = \int_0^t s^{p-1} ds = \frac{t^p}{p}, \quad t > 0, \quad (52)$$

and therefore

$$\Delta\tau_n = \Theta_p(t_{n+1}) - \Theta_p(t_n) = \frac{t_{n+1}^p - t_n^p}{p}. \quad (53)$$

This explicit formula will be used in the numerical experiments whenever the canonical conformable kernel is selected.

Since the general conformable HBV model admits a unique solution under the assumptions established in Section 4, the numerical scheme (49)–(50) is consistent with the transformed ODE (46). The predictor step is first-order accurate in stretched time, while the trapezoidal corrector provides a second-order local correction in the variable τ . Hence, the method may be interpreted as a conformable extension of a classical predictor–corrector integrator applied after the generalized conformable time transformation [18, 19].

The complete numerical algorithm is summarized below.

Algorithm 1 General conformable predictor–corrector scheme

- 1: **Input:** final time T , number of steps N , initial state X_0 , fractional order p , conformable function $\psi(\cdot, p)$
 - 2: Set $h = T/N$ and $t_n = nh$ for $n = 0, 1, \dots, N$
 - 3: Compute $\tau_n = \Theta_p(t_n) = \int_0^{t_n} \frac{1}{\psi(s, p)} ds$ for $n = 0, 1, \dots, N$
 - 4: **for** $n = 0, 1, \dots, N-1$ **do**
 - 5: Compute $\Delta\tau_n = \tau_{n+1} - \tau_n$
 - 6: Predictor:
 $\hat{X}_{n+1} = X_n + \Delta\tau_n F(X_n)$
 - 7: Corrector:
 $X_{n+1} = X_n + \frac{\Delta\tau_n}{2} \left[F(X_n) + F(\hat{X}_{n+1}) \right]$
 - 8: **end for**
 - 9: **Output:** $\{X_n\}_{n=0}^N$
-

The numerical trajectories generated by Algorithm 1 will serve two purposes in the sequel: first, to analyze the general conformable HBV dynamics under different parameter settings; and second, to generate high-fidelity simulation data for the training of the deep neural network surrogate introduced in the next sections.

6 Deep Neural Network Surrogate Framework

To accelerate repeated simulations of the proposed general conformable fractional HBV model, we construct a deep neural network (DNN) surrogate trained on numerical trajectories generated by the predictor–corrector

scheme introduced in Section 5. The surrogate is designed to learn one-step state transitions of the GCFD-HBV dynamics under different fractional orders and different admissible conformable kernels. In this way, the learning component is fully integrated with the numerical solver and does not rely on synthetic trajectories unrelated to the mathematical model.

The training dataset is generated directly from the numerical trajectories produced by Algorithm 1. For each simulated trajectory, the numerical solver provides a sequence of states

$$X_n \approx X(t_n), \quad n = 0, 1, \dots, N,$$

together with the stretched-time increments

$$\Delta\tau_n = \Theta_p(t_{n+1}) - \Theta_p(t_n).$$

Each supervised learning pair is then formed as a one-step transition

$$(\mathcal{Z}_n, X_{n+1}),$$

where the input feature vector $\mathcal{Z}_n$ consists of the current state, the local stretched-time increment, the fractional order, the kernel encoding, and a reduced set of informative epidemiological parameters. In the implementation used for the present study, the resulting dataset contains 15,000 samples, with input dimension 15 and output dimension 6.

In order to enhance diversity, the data is made based on trajectories of a variety of GCD kernels, i.e., the canonical-power, shifted-power, rational and exponential kernels, and different fractional orders. This design allows the surrogate to be informed of the baseline HBV dynamics and also the impact of the temporal deformation that exists depending on the kernel on the epidemiology paths. The application of more than one kernel is especially relevant to the current work since the results of the rollout later reveal that the selection of kernels has a significant effect on the accuracy of the surrogate.

The surrogate model is used in the form of a multilayer perceptron (MLP) regressor. The feature vector of the current numerical state in the form of the 15-dimensional input layer is fed to the input layer, and the next six-dimensional HBV state is predicted by the output layer.

$$\hat{X}_{n+1} = (\hat{S}_{n+1}, \hat{I}_{1,n+1}, \hat{I}_{2,n+1}, \hat{H}_{n+1}, \hat{R}_{n+1}, \hat{V}_{n+1})^\top.$$

The network is trained using one-step transitions instead of full dynamic time trajectories, as it is preferable since the underlying dynamics of the numerical solver being corrected moves the state by one-step updates in the stretched-time variable. This design option also does not require the unwanted sequential structure in case the numerical integrator is already local in the transformed variable, τ . The train/validation/test protocol applied during the implementation is a typical random split and the benchmark of the optimizer reported indicates that such a setup can be used to attain very high one-step predictive accuracy.

The histories of numerical training also indicate that there are a number of adaptive optimizers that are converging fast in the environment. Specifically, the loss curves show that NAdam, AdamW, and Adam are the most favorable ones in terms of convergence, and Adagrad and, in particular, SGD converge much slower and achieve significantly worse final errors. The trends are in line with the quantitative benchmark that is stated in Subsection 8.4.

The risk of the surrogate being trained is reduced by minimizing the mean squared error (MSE) of the predicted and numerically generated one-step state. Where X_{n+1} refers to the target state obtained using GCFD numerical solver and $\hat{X}_{n+1}$ refers to network prediction. We have:

$$\mathcal{L}_{\text{MSE}} = \frac{1}{M} \sum_{m=1}^M \left\| \hat{X}_{n+1}^{(m)} - X_{n+1}^{(m)} \right\|_2^2, \quad (54)$$

where M is the batch size.

Three common regression measures are used to measure the predictive performance and they include the root mean squared error (RMSE), mean absolute error (MAE), and the coefficient of determination R^2 . They can be applied in benchmark evaluation of a single step and in a rollout evaluation that is multi-step. The one-step optimization results reveal that the optimal optimizers have RMSE values that are near to 3 and R^2 values are near to 0.998, and this implies that the surrogate has a very high accuracy in capturing the local GCFD-HBV transition map. Measures of the same are taken at the rollout level to compare full surrogate-generated trajectories with the ones obtained through the numerical solver. The errors are bound to be higher in such a challenging environment since errors in prediction one-step are compounded by time. The summary of rollout validates the fact that the rational kernel provides the best kernel-specific surrogate performance as it achieves $\text{RMSE} = 20.2479$, $\text{MAE} = 10.7776$ and $R^2 = 0.8961$ at $p = 0.85$. As a measure of

the sensitivity of surrogate training to the optimization strategy, six standard optimizers are compared, i.e., Adam, AdamW, RMSprop, SGD, Adagrad, and NAdam. These optimizers have a clear delineation based on their results at the benchmark. NAdam scores the highest in one-step test, RMSE = 2.9698, MAE = 1.4705 and $R^2 = 0.9980$, and closely AdamW and Adam follow. RMSprop is also a competitive algorithm, but it is significantly less precise, whereas Adagrad and SGD are significantly less effective. To be more specific, the ranking received as the result of the benchmark when ranked by RMSE as the ones with the lowest values are NAdam > AdamW > Adam > RMSprop > Adagrad > SGD. Such a minor gap among the three leading approaches suggests that the surrogate-learning task is conditioned and that there are multiple adaptive optimizers that can be used to learn the one-step transition map. Nevertheless, the gap between the best adaptive techniques and SGD is large indicating that adaptive learning-rate techniques are especially useful to this multi-feature, multi-output GCFD surrogate problem. The optimizer benchmark thus justifies the application of NAdam as the training strategy of choice in the further rollout experiments. It also offers a practical conclusion that is useful in practice in scientific machine learning to conformable-fractional epidemiological models: although the physical dynamics are defined by the numerical solver, the optimizer selection can play an important role in determining the performance and ultimate quality of the learned surrogate approximation.

7 Integrated Learning Algorithm

This part gives a summary of the entire computational process to integrate the general conformable fractional HBV model solver with the deep neural network surrogate. The offered architecture is aimed at maintaining the mathematical format of the GCFD-HBV system and allowing the data-driven acceleration of repeat simulations. The general process is divided into four processes that are interconnected, i.e. generation of numerical trajectories, construction of supervised dataset, training and selection of surrogate and evaluation of generalization by using rollouts. The framework begins with the general conformable HBV model which is given in Section 3, and the stretched-time predictor–corrector scheme which is presented in Section 5. Given a prescribed kernel $\psi(\cdot, p)$, fractional order p , parameter set and initial condition, the Algorithm 1 produces a trajectory $\{X_n\}_{n=0}^N$, with $X_n \approx X(t_n)$ being the same, and increments of the stretched-time that are denoted as $\Delta\tau_n = \Theta_p(t_{n+1}) - \Theta_p(t_n)$. Such numerical outputs are then converted to one-step samples of supervised learning of the type (Z_n, X_{n+1}) , where Z_n is the present state, $\Delta\tau_n$ is the increment, a kernel encoding and a smaller number of chosen epidemiological parameters. By so doing, it is possible to train the surrogate on directly the data produced by the mathematically sound GCFD solver, and not on a set of arbitrary synthetic signals. The resulting dataset that is used in the experiments consists of 15,000 transition samples and input dimension 15 and output dimension 6.

Integrated HBV-DNN Surrogate Framework

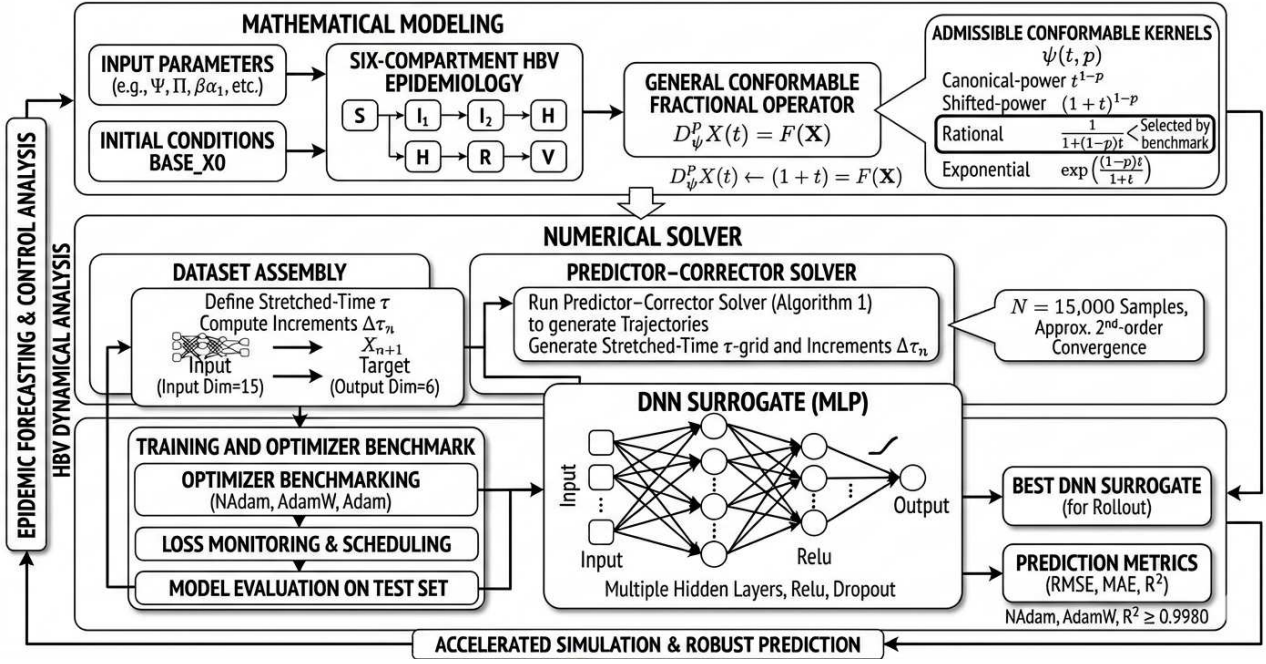


Figure 1: Integrated workflow of the proposed GCFD-HBV solver–surrogate framework. The diagram summarizes the interaction between the general conformable HBV model, the admissible kernel selection stage, the stretched-time predictor–corrector solver, the construction of supervised learning data, the DNN surrogate training and optimizer benchmark, and the final rollout-based predictive evaluation.

Figure 1 shows the combined process of the suggested HBV-DNN surrogate model. The top component of the diagram depicts the mathematical modeling phase where the six-compartment HBV epidemiological structure is integrated together with the general conformable fractional operator, as well as admissible conformable kernel. This step determines the HBV dynamics that is kernel-dependent and offers the modeling framework used in all the subsequent calculations. The numerical solver is the central section during which the transformation of the stretched time and the predictor–corrector integration algorithm are implemented in order to introduce high-fidelity GCFD-HBV system trajectories. These paths are subsequently translated into unit step supervised input-outputs to the surrogate-learning step. The bottom of the figure gives the description of the DNN surrogate and the training process, the process of optimizer benchmarking, loss monitoring, and test-set evaluation. Lastly, the surrogate is trained on a rollout mode which is utilized to provide fast predictions and its performance is evaluated by conventional regression measures like RMSE, MAE and R^2 . In general, the figure addresses how the suggested framework integrates mechanistic fractional modelling, robust numerical approximation, and information-supported surrogate learning into an end-to-end computational paradigm of computational accelerated HBV simulation and prediction.

The integrated learning workflow can be summarized by Table 1 :

Table 1: Integrated GCFD-HBV solver-surrogate workflow.

Step	Stage	Description	Key Outcome
1	Kernel and order selection	Choose a conformable kernel $\psi(\cdot, p)$ from the admissible family considered in this work (canonical-power, shifted-power, rational, or exponential) and select a fractional order $p \in (0, 1]$.	The selected kernel and order determine the temporal deformation mechanism of the HBV dynamics. The numerical experiments show that these choices strongly influence the model behavior. In particular, the rational kernel yields the smallest final infected population and the best rollout performance for the DNN surrogate.
2	Trajectory generation	For each selected kernel and order, simulate the GCFD-HBV system using the predictor-corrector solver in stretched time. This step produces the trajectory states and the local increments $\Delta\tau_n$ required for supervised training.	High-fidelity numerical trajectories are generated for the learning stage. The prior validation study confirms that the underlying solver exhibits approximately second-order convergence.
3	Dataset assembly	Convert the numerical trajectories into one-step training pairs. Each input vector includes the current HBV state, the local stretched-time step, the fractional order, the kernel identifier, and selected parameter features, while the output vector is the next six-dimensional state.	A structured supervised dataset is constructed, allowing the surrogate to learn both epidemiological transitions and kernel-dependent temporal deformations.
4	Surrogate training	Train the MLP surrogate using the MSE loss and benchmark multiple standard optimizers.	The optimizer benchmark shows that NAdam attains the best one-step test performance, followed closely by AdamW and Adam, whereas SGD and Adagrad are substantially less effective.
5	Rollout evaluation	Starting from an unseen initial condition and parameter configuration, recursively apply the trained surrogate over multiple time steps and compare the resulting rollout against the numerical solver.	The generalization ability of the surrogate is assessed over long horizons. The rollout benchmark demonstrates that the rational kernel yields the most accurate surrogate trajectories, with RMSE = 20.2479, MAE = 10.7776, and $R^2 = 0.8961$ at $p = 0.85$.

The end-to-end workflow used in the experiments is summarized in Algorithm 2.

Algorithm 2 Integrated GCFD-HBV learning framework

```
1: Input: kernel family  $\mathcal{K}$ , fractional-order set  $\mathcal{P}$ , baseline parameter set  $\Theta$ , initial-state generator  $\mathcal{I}$ , simulation horizon  $T$ , and number of time steps  $N$ 
2: for each kernel  $\psi \in \mathcal{K}$  do
3:   for each fractional order  $p \in \mathcal{P}$  do
4:     Generate numerical trajectories of the GCFD-HBV model using Algorithm 1
5:     Compute the stretched-time increments  $\Delta\tau_n$ 
6:     Construct one-step supervised samples  $(\mathcal{Z}_n, X_{n+1})$ 
7:   end for
8: end for
9: Aggregate all generated samples into a global supervised dataset
10: Split the dataset into training, validation, and test subsets
11: for each optimizer in  $\{\text{Adam, AdamW, RMSprop, SGD, Adagrad, NAdam}\}$  do
12:   Train the surrogate model on the training subset
13:   Monitor the validation loss and retain the best model state
14:   Evaluate one-step prediction performance on the test subset
15: end for
16: Select the optimizer with the lowest test RMSE
17: Evaluate multi-step rollout performance of the selected surrogate on unseen scenarios
18: Output: trained surrogate model, optimizer ranking, rollout performance metrics, and kernel performance summary
```

The solver-surrogate framework is an integrated framework, which offers a consistent approach to scientific machine learning of generalized conformable epidemiological systems. The mathematical fidelity of the HBV model to the numerical solver and the neural surrogate provide the computationally efficient approximation of the learned transition operator. Optimizer benchmark shows that adaptive approaches work incredibly better than the classical SGD to this problem, where NAdam has the highest one-step generalization. In addition, the rollout findings indicate that the rollout long-horizon surrogate prediction is not only dependent on the optimizer, but also on the selected conformable kernel. Rational kernel comes out to be the best scenario of the current experiments, not only dynamically, but also regarding the accuracy of the surrogate rollouts.

In the following section, this unified framework will be employed in discussing the numerical behavior of the GCFD-HBV model, quality of the trained surrogate and how the choice of the optimizer and kernel can impact the predictive behavior of the learned approximation.

8 Numerical Results and Discussion

This section presents the numerical validation of the proposed general conformable fractional HBV framework, the dynamic effects induced by different admissible conformable kernels, the predictive quality of the DNN surrogate, and the comparative performance of the optimizers used during training. Overall, the experiments confirm that the stretched-time predictor–corrector scheme is highly accurate, that kernel choice significantly affects both the epidemic dynamics and the learnability of the surrogate, and that adaptive optimizers clearly outperform non-adaptive alternatives in the present learning problem.

8.1 Validation of the Numerical Method

To determine the consistency of the proposed numerical solver, a study of mesh-refinement was conducted by comparing the approximation of the predictor–corrector with a reference solution of high accuracy in the transformed ODE system. It is observed that the relative L_2 error fall monotonically with increase in the number of time steps, as indicated in Table 2. This proves the consistency and stability of the numerical scheme.

Table 2: Validation of the proposed predictor–corrector solver under mesh refinement.

N	h	Relative L_2 error	Estimated order
200	0.200	8.0114×10^{-5}	–
400	0.100	1.9678×10^{-5}	2.0255
800	0.050	4.8764×10^{-6}	2.0127
1600	0.025	1.2138×10^{-6}	2.0063

The approximate convergence orders are quite near to 2 and this means that the stretched-time predictor–corrector method is practically a second-order scheme. Such accuracy is adequate both to directly simulate the

GCFD-HBV system and to produce high-fidelity data to train the surrogate.

8.2 Dynamic Behavior of the HBV-GCD Model

The dynamic behaviour of HBV-GCD model was studied in the four admissible conformable kernels, which include canonical-power, shifted-power, rational and exponential. At each kernel, simulations were done at the representative fractional orders $p = 0.65$, $p = 0.80$, and $p = 0.95$. These numerical findings indicate that the numerical results show that the kernel mainly alters the temporal scaling of the HBV dynamics, but the initial epidemic peak is almost the same in all the cases tested.

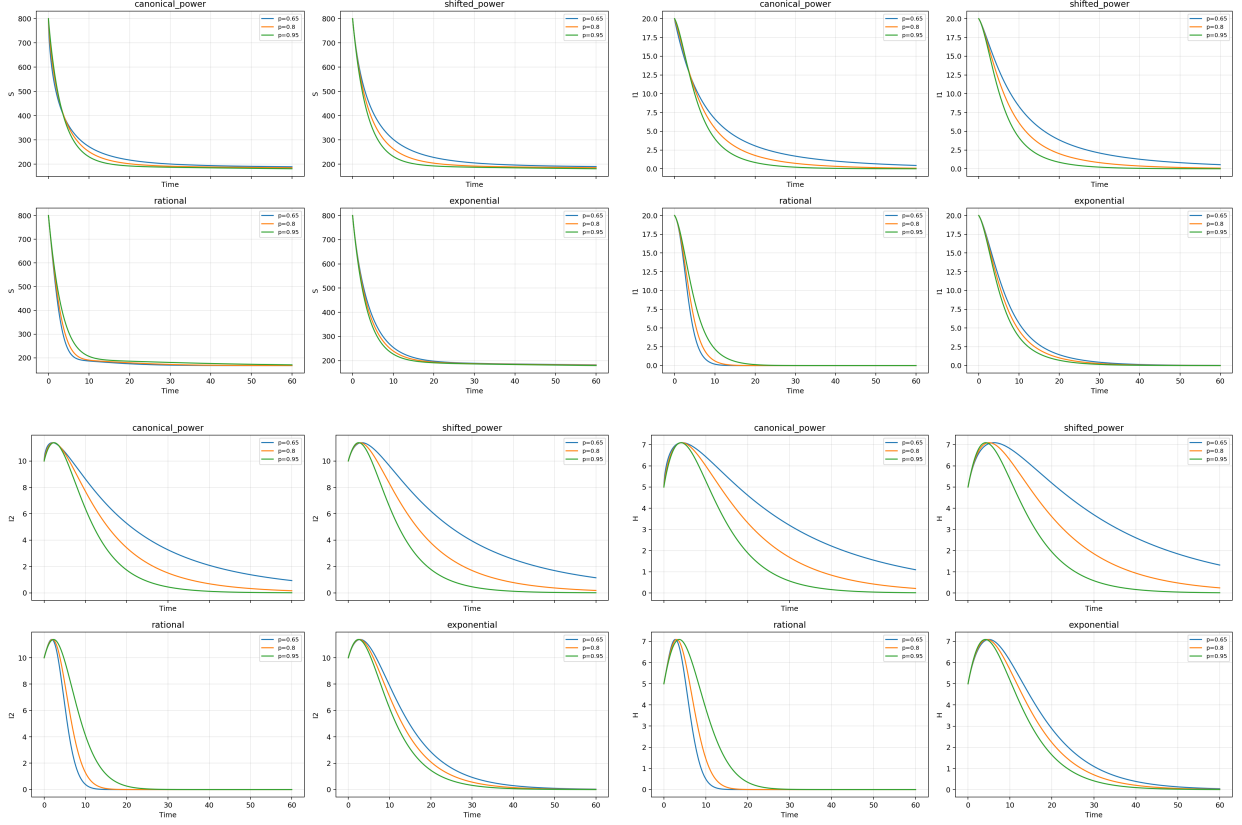


Figure 2: Effect of the general conformable kernels on the susceptible, acute infected, chronic infected, and hospitalized compartments for different fractional orders.

Figure 2 reveals that the vulnerable category will decrease exponentially under any kernel, yet the temporary decay speed is more vulnerable to the kernel and the order in the infected sections. Specifically, the acute and chronic infected populations irrevocably degenerate under the rational kernel than under the canonical-power and shifted-power kernels. This means that the conformable role can be a major change in the effective temporal scaling of infection progression.

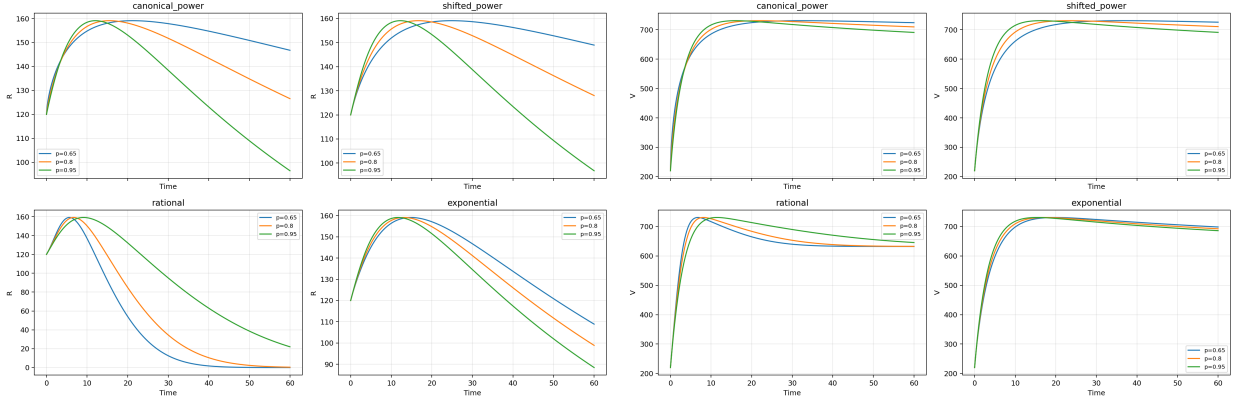


Figure 3: Effect of the general conformable kernels on the recovered and vaccinated compartments for different fractional orders.

Figure 3 shows significant variations in the long term balance between recovery and vaccination. In the case of canonical-power, shifted-power, and exponential kernel, the recovered population diminishes drastically with the fractional order but the vaccinated population is prevalent at long times. In comparison, the rational kernel has a significantly different behavior, lower terminal recovered population and moderate level of vaccinated, which shows a more rapid decrease in the classes of the infected.

Table 3 is a more quantitative summary of the final epidemic burden. The final infected population is estimated to be significantly different in all kernels and orders though the maximum infected population is constant at 35.535. The rational kernel leads the overall infected total to be virtually zero at all tested values of p and the other kernels have small, yet nonzero, infected tails.

Table 3: Selected numerical summaries of the GCFD-HBV dynamics for the tested conformable kernels and fractional orders.

Kernel	p	Peak infected total	Final infected total	Final vaccinated
Canonical-power: t^{1-p}	0.65	35.5344	2.4753	724.0533
Canonical-power: t^{1-p}	0.80	35.5352	0.4554	709.7527
Canonical-power: t^{1-p}	0.95	35.5350	0.0268	690.8928
Shifted-power: $(1+t)^{1-p}$	0.65	35.5353	3.0136	725.7615
Shifted-power: $(1+t)^{1-p}$	0.80	35.5351	0.5159	710.7251
Shifted-power: $(1+t)^{1-p}$	0.95	35.5351	0.0278	691.1030
Rational: $\frac{1}{1+(1-p)t}$	0.65	35.5351	≈ 0	632.2848
Rational: $\frac{1}{1+(1-p)t}$	0.80	35.5350	≈ 0	632.5136
Rational: $\frac{1}{1+(1-p)t}$	0.95	35.5353	1.41×10^{-8}	645.6455
Exponential: $\exp\left(\frac{(1-p)t}{1+t}\right)$	0.65	35.5354	0.0934	698.5302
Exponential: $\exp\left(\frac{(1-p)t}{1+t}\right)$	0.80	35.5353	0.0345	692.3682
Exponential: $\exp\left(\frac{(1-p)t}{1+t}\right)$	0.95	35.5350	0.0110	685.9964

These observations substantiate the claim that the choice of the kernels is not only a quantitative tool, but it is also a real modeling decision in the GCFD-HBV framework. The rational kernel is the best of the tested kernels in the current simulations in terms of epidemic suppression.

8.3 Surrogate Prediction Performance

The DNN surrogate was trained on one step transition dynamics produced by numerical solver and tested in recursive rollout mode. The obtained data consists of 15,000 samples, where the dimensions of inputs are 15 and the dimensions of outputs are 6. On the one-step level, the surrogate is highly accurate when optimized using the most effective adaptive optimizers. The prediction of long-horizon rollout is however more difficult due to the fact that local prediction errors are compounded with time.

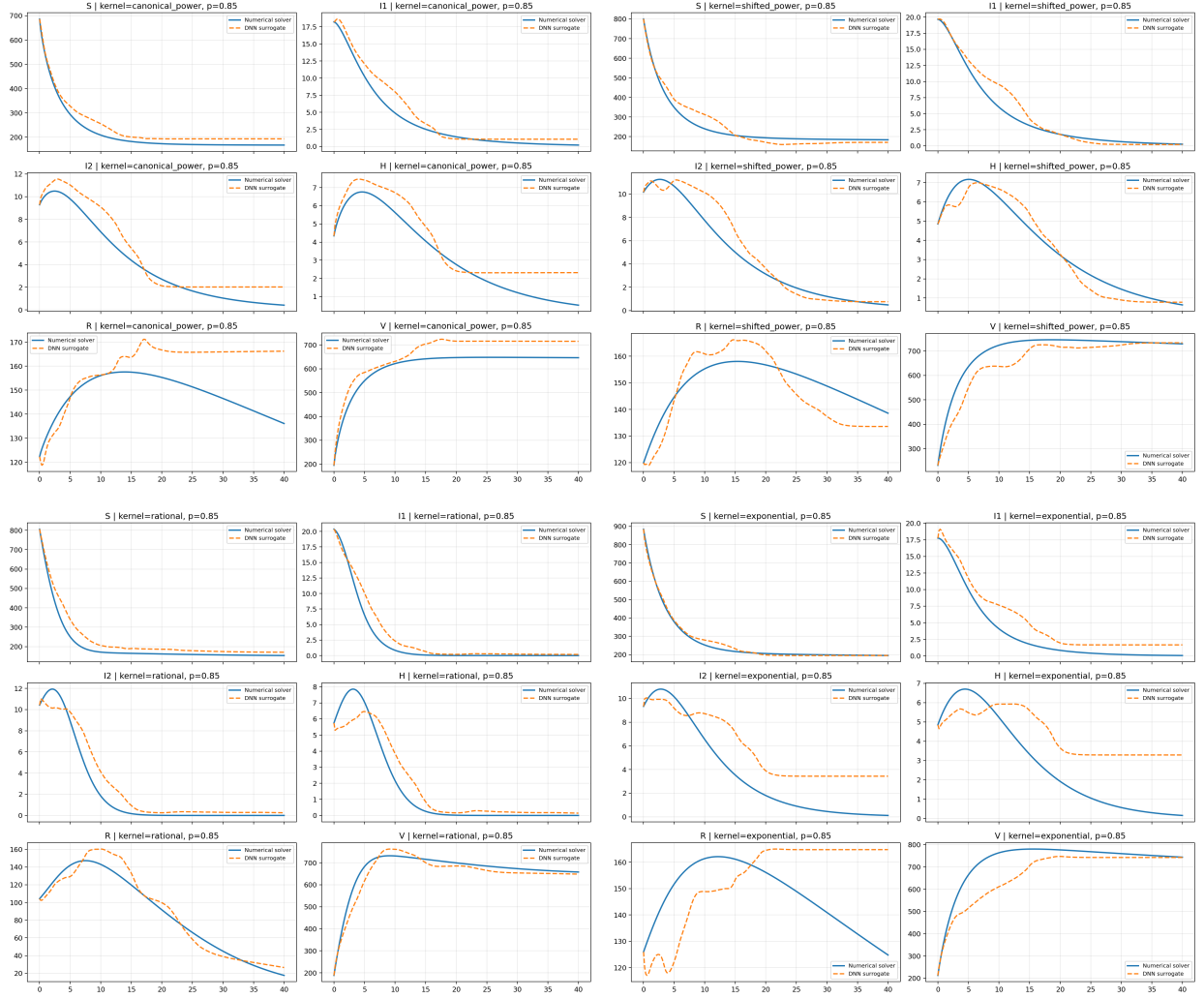


Figure 4: Comparison between the numerical solver and the DNN surrogate in multi-step rollout mode for the four tested conformable kernels at $p = 0.85$.

Figure 4 indicates that the surrogate is globally consistent with the solver trajectories but the extent of the mismatch is highly dependent on the kernel. The rational kernel has the greatest agreement across the entire time horizon, whereas the canonical-power and exponential kernels have a higher rollout error accretion in certain compartments. It is in accord with the quantitative indices presented in Table 4.

Table 4: Rollout performance of the DNN surrogate for the tested conformable kernels at $p = 0.85$.

Kernel	RMSE	MAE	R^2
Canonical-power	27.3980	16.0813	0.7137
Shifted-power	25.7280	12.8594	0.8287
Rational	20.2479	10.7776	0.8961
Exponential	34.3284	15.3257	0.7556

It is evident that a rational kernel will give the highest rollout performance with the lowest RMSE and MAE and the highest coefficient of determination. This implies that, the rational kernel, besides generating the strongest epidemic suppression in the direct solver, results in trajectories that are more susceptible to learning by the neural surrogate.

8.4 Training and Optimizer Comparison

Six standard optimizers were benchmarked to measure the sensitivity of surrogate training to the optimization strategy, which were Adam, AdamW, RMSprop, SGD, Adagrad, and Nadam. Figure 5 shows the training and validation loss curves and Table 5 presents the ultimate test measurements.

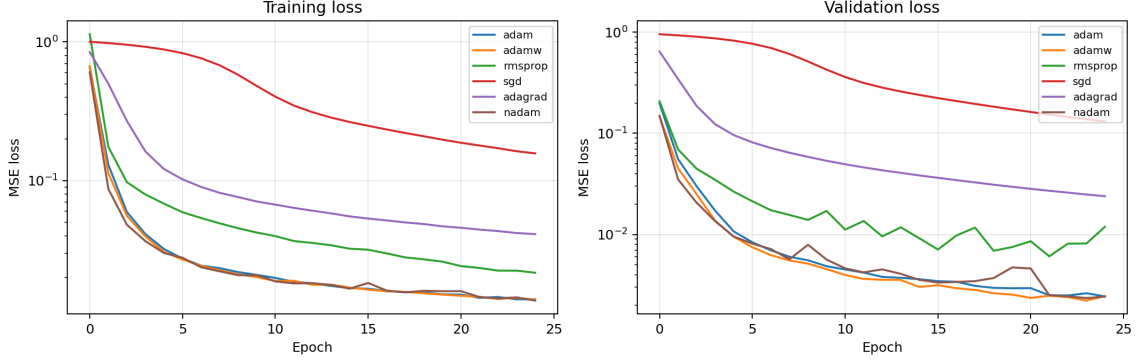


Figure 5: Training and validation loss histories for the tested optimizers.

Figure 5 indicates that NAdam, AdamW, and Adam quickly converge to low error rates of validation, and RMSprop levels at a larger loss and both Adagrad and SGD converge more slowly and less precisely. This qualitative observation is substantiated in Table 5 quantitatively.

Table 5: Optimizer benchmark for one-step surrogate prediction.

Optimizer	RMSE	MAE	R^2
NAdam	2.9698	1.4705	0.9980
AdamW	3.0535	1.5580	0.9980
Adam	3.2495	1.6047	0.9975
RMSprop	5.0766	2.5375	0.9941
Adagrad	12.9570	6.5860	0.9612
SGD	26.6052	14.1399	0.8417

NAdam, AdamW and Adam are the closest performers in terms of the best overall performance. The distance between these adaptive techniques and SGD is significant, and this suggests that adaptive learning-rate techniques are especially useful in this multi-feature, multi-output surrogate-learning task. According to these findings, NAdam is chosen as the default optimizer of the final surrogate model.

On the whole, the learning and numerical experiments allow making three key conclusions. First, the proposed stretched-time predictor–corrector solver is accurate and empirically second-order. Second, the conformable kernel has a significant impact on the HBV dynamics and the quality of surrogate rollout prediction. Third, adaptive optimizers, in particular, NAdam, are the most suitable option to train DNN surrogates of the current GCFD-HBV system.

9 Conclusion

A general conformable fractional HBV model is proposed in this work in the effort to describe the interaction between the susceptible, acute infected, chronic infected, hospitalized, recovered, and vaccinated populations. The model constructs the classical and standard conformable HBV models with a local time-deformation mechanism that is deformed with more flexibility by introducing the general conformable fractional derivative. The formulation maintains the epidemiological sense of the state variables and increases the modeling space on which the HBV dynamics have access to. The theoretical explanation proves that the suggested system is well posed mathematically and biologically. Particularly, positivity, limited positivity, strict positivity of the total population, global existence, uniqueness, and UlamHyers stability were proved. Such conclusions ensure that at least the model trajectories are meaningful at all times in the future and that approximate solutions are even close to the exact ones as long as the residual perturbation is sufficiently small. The significance of this theoretical strength is particularly due to the fact that it justifies the numerical approximation phase as well as the surrogate construction based on learning. Numerical predictorcorrector scheme was then obtained in the stretched time variable belonging to a general conformable kernel. The conventional test indicated that the procedure is acting as a scheme of approximate second-order and the convergence orders are estimated as near to 2 as mesh refinement. This establishes the fact of the transformed-time solver giving a good high-fidelity estimate of the given GCFD-HBV system. On the data-driven part, a deep neural network surrogate was trained on 15,000 one-step transition samples obtained using the numerical solver.

It has been shown by the optimizer benchmark that adaptive algorithms are much dominant in this context, with NAdam giving the highest one-step test accuracy, followed by AdamW and Adam. This demonstrates that

the choice of optimizers is not just a technical aspect of scientific machine learning of generalized conformable epidemiological models. Another finding of the experiments was that the choice of the kernel affects the dynamics of HBV as well as the performance of long horizons of the learned surrogate heavily. Of the kernels that were tested, the rational kernel

$$\psi(t, p) = \frac{1}{1 + (1 - p)t}$$

produced the smallest terminal infected burden in the direct numerical simulations and yielded the best surrogate rollout performance, with RMSE = 20.2479 and $R^2 = 0.8961$ at $p = 0.85$. This finding illustrates the significance of choosing the kernel in the overall conformable environment and that some of the kernels can prove beneficial both epidemiological and computational perspectives. Altogether, the current research indicates that general conformable fractional modeling, coupled with neural surrogate learning, is an effective framework to use in the analysis of HBV. The suggested methodology provides mathematical freedom as well as efficiency in computing the model that remains viable to repeated run of the simulations, sensitivity-related studies, and optimization-aware surrogate modeling in the future. Following on this paper, there are several directions, such as an extension to the derivation of the basic reproduction number to the general conformable setting, optimal control when the dynamics are governed by a kernel, identification of parameters given real epidemiological data, and the generalisation to stochastic or multi-group HBV models.

Author Contributions

- **Essia Ben Alaia:** Conceptualization, methodology, software, formal analysis, investigation, data curation, writing – original draft preparation.
- **Slim Dhahri:** Methodology, software, validation, formal analysis, data curation, visualization, writing – review and editing.
- **Sahar Almashaan:** Validation, resources, funding acquisition, writing – review and editing, project administration, corresponding author.
- **Hatem Alwardi:** Validation, resources, writing – review and editing.
- **Omar Naifar:** Conceptualization, methodology, supervision, validation, resources, writing – review and editing, project administration.

Conflict of Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Data Availability Statement

The datasets supporting the findings of this study were generated numerically from the proposed general conformable fractional HBV model using the stretched-time predictor-corrector scheme described in the manuscript. The computational code used to produce the numerical simulations, surrogate-training dataset, and optimizer-benchmarking results is publicly available at:

<https://github.com/vegaws1/FGD/blob/main/HBV>

Additional generated datasets and computational results are available from the corresponding author upon reasonable request.

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