



A Hilfer Fractional Vaccination Epidemic Model: Stability Analysis and a Physics-Informed Neural Network Validated on COVID-19 Data

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ABSTRACT

A Hilfer generalized fractional epidemic model with vaccination and asymptomatic transmission is proposed and analysed. The Hilfer operator interpolates between the Riemann-Liouville and Caputo derivatives through a type parameter. Therefore, the model captures hereditary memory and nonlocal temporal effects through both the fractional order and the type. An equivalent Volterra integral formulation is derived and used to prove the existence and uniqueness of solutions by fixed-point theory, together with positivity, boundedness, and a positively invariant region. The disease-free and endemic equilibria are obtained. The basic reproduction number is computed by the next-generation matrix method and local stability is established. Then, a fractional physics-informed neural network is developed on the Volterra residual and verified against a predictor-corrector scheme. The same network is used in inverse mode to calibrate the model against reported COVID-19 incidence for Italy during the autumn 2021 vaccination period, achieving a coefficient of determination of 0.99. Sensitivity analysis and a quantitative comparison with existing fractional approaches round out the study, which offers an accurate and flexible framework for memory-dependent epidemic systems.

1. Introduction

Mathematical epidemiology provides the quantitative language for understanding how infectious diseases spread and for judging which interventions work. The subject begins with Kermack & McK-

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endrick [1], whose threshold theorem introduced the compartmental view of an epidemic and remains the reference point for every model that follows. A modern account of that tradition is given by Hethcote [2], who surveys the deterministic theory of infectious diseases and explains why compartmental models are still central to outbreak prediction and policy design. The single number that governs the threshold behaviour of such models is the basic reproduction number, and its rigorous definition was established by Diekmann *et al.* [3]. The practical computation of this number for general compartmental systems was then made systematic by van den Driessche & Watmough [4] through the next-generation matrix, and the construction of that matrix was clarified further by Diekmann *et al.* [5]. These four works together supply the analytical machinery that we use in the equilibrium analysis of the present study.

Classical models built on ordinary derivatives describe a system whose future depends only on its present state, which ignores the memory that pervades biological processes. Fractional calculus removes this restriction, and the theory needed to work with it is documented in a sequence of monographs. Podlubny [6] presents the foundational operators and their Laplace analysis. Kilbas *et al.* [7] develop the existence and integral-transform theory in depth. Diethelm [8] develops the analysis of fractional differential equations with a view toward computation. The numerical side is treated by Li & Zeng in their first [9] and revised [10] texts on numerical methods for fractional calculus. The approximation of fractional integrals and derivatives is examined in depth by Li & Cai [11]. The related geometric viewpoint of fractal calculus, which we invoke when comparing operators, is collected by Golmankhaneh [12].

Within this landscape the Hilfer derivative is distinguished by an extra type parameter that connects the Riemann–Liouville and Caputo operators. The idea and its physical meaning were set out by Hilfer [13], who later reported experimental evidence for fractional time evolution in complex systems [14]. The mathematical theory of Hilfer problems was then built up in stages. Furati *et al.* first proved existence and uniqueness for a Hilfer initial value problem [15], and in a companion paper analysed the non-existence of global solutions [16]. Oliveira & de Oliveira [17] extended the operator to the Hilfer–Katugampola family. Almeida [18] introduced the derivative of a function with respect to another function that underlies many generalized formulations. More recently, Hilal *et al.* [19] established existence and stability for coupled Hilfer systems with nonlocal conditions, and Goedegebure and Marynets contributed a Bernstein-spline method for Hilfer initial value problems [20] and an analysis of Hilfer fractional-periodic boundary value problems [21]. These works supply the weighted spaces, the Volterra reformulation and the fixed-point tools on which our well-posedness proofs rely.

The use of nonlocal operators in epidemiology has grown rapidly, especially for COVID-19. Abdulwasaa *et al.* [22] used a fractal–fractional model to forecast the pandemic and showed the value of memory-dependent dynamics. El-Dessoky *et al.* [23] analysed an epidemic model built on the Atangana–Baleanu fractal–fractional derivative. Khan *et al.* [24] demonstrated that fractal–fractional operators fit observed data better than integer-order models.

Vaccination has been added to several of these frameworks; i.e., Alalhareth *et al.* [25] studied a fractal–fractional vaccination model for coronavirus transmission. Matouk *et al.* [26] examined a fractional spatio-temporal SEIR model, both finding that memory and vaccine parameters strongly shape the dynamics. On the analytical side, El Mamouni [27] proved well-posedness for a generalized fractal–fractional epidemic operator by fixed-point theory. Gunasekar *et al.* [28] modelled COVID-19 and tuberculosis co-infection. Amouch & Karim *et al.* [29] combined stability analysis with numerical simulation for a fractional COVID-19 model. For the numerical reference solution used throughout this paper we rely on the predictor–corrector method of Diethelm *et al.* [30].

A complementary and rapidly maturing tool is the physics-informed neural network. The framework was introduced by Raissi *et al.* [31], who embedded the residual of a differential equation into the training loss so that the network respects the governing physics. Karniadakis *et al.* [32] later sur-

veyed the broader field of physics-informed machine learning and its integration of models with data. The extension to fractional operators is due to Pang *et al.* [33], whose fractional physics-informed networks handle exactly the kind of nonlocal residual that a Hilfer model produces. The software realization of these ideas was provided by Lu *et al.* [34]. A current overview of the methodology and its open problems is given by Cuomo *et al.* [35]. For the real-data component of our study we use the publicly curated COVID-19 records assembled by Mathieu *et al.* [36], from which the Italian incidence series is taken.

The preceding survey exposes a clear gap. Although the Hilfer operator, the next-generation theory, the fractional numerical schemes and the physics-informed methodology are each well developed, they have not been brought together for a vaccination epidemic model with asymptomatic transmission. Most existing fractional epidemic studies use Caputo, Riemann–Liouville, Caputo–Fabrizio or Atangana–Baleanu operators, emphasise simulation rather than a thorough analysis, and are seldom validated on real observations through a learning method. This is the motivation for the present work: to build a single framework in which the Hilfer generalized fractional operator, a rigorous qualitative analysis, a fractional physics-informed neural network and validation on real data all appear together.

Accordingly, we propose a Hilfer generalized fractional $SEI_c I_\eta VR$ model with vaccination, asymptomatic and symptomatic infection. We derive its equivalent Volterra formulation and prove existence and uniqueness by fixed-point theory, we establish positivity, boundedness and a positively invariant region, we obtain the disease-free and endemic equilibria and compute the basic reproduction number by the next-generation matrix, and we prove local stability of both equilibria. We then construct a fractional physics-informed neural network on the Volterra residual, prove a consistency bound linking its error to the physics loss, verify it against the predictor–corrector reference, apply it in inverse mode to calibrate the model to reported COVID-19 incidence for Italy, and report a battery of training diagnostics for a below-threshold and an endemic regime. A sensitivity analysis of the reproduction number and a structured comparison with existing approaches round out the contribution.

The paper is organised as follows. Section 2 collects the mathematical preliminaries and Section 3 formulates the model and its compartmental diagram. Existence and uniqueness are proved in Section 4, positivity and boundedness in Section 5, and equilibria and stability in Section 6. The physics-informed neural network is developed in Section 7, numerical simulation and sensitivity analysis are presented in Section 8, real-data validation for Italy in Section 9, and the neural network training diagnostics in Section 10. A comparison with existing approaches is given in Section 11 and Section 12 concludes.

2. Mathematical Preliminaries

Throughout, let $0 < \alpha < 1$ be the fractional order and $0 \leq \nu \leq 1$ the Hilfer-type parameter, and set:

$$\omega = \alpha + \nu(1 - \alpha), \quad 0 < \omega \leq 1 \quad (1)$$

Definition 1. Let $x \in L^1([0, T], \mathbb{R})$. The Riemann–Liouville fractional integral of order $\alpha > 0$ is:

$$I_{0+}^\alpha x(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t - \xi)^{\alpha-1} x(\xi) d\xi, \quad t > 0 \quad (2)$$

as given in [6–8].

Definition 2. Let $0 < \alpha < 1$, $0 \leq \nu \leq 1$ and $x \in L^1([0, T], \mathbb{R})$. The Hilfer generalized fractional derivative of order α and type ν is:

$${}^H D_{0+}^{\alpha, \nu} x(t) = I_{0+}^{\nu(1-\alpha)} \frac{d}{dt} I_{0+}^{(1-\nu)(1-\alpha)} x(t) \quad (3)$$

following [13–15, 17]. For $\nu = 0$ it reduces to the Riemann–Liouville derivative and for $\nu = 1$ to the Caputo derivative.

Definition 3. For $0 < \omega \leq 1$, the weighted space of continuous functions is:

$$C_{1-\omega}([0, T], \mathbb{R}) = \left\{ x : (0, T] \rightarrow \mathbb{R} : t^{1-\omega} x(t) \in C([0, T], \mathbb{R}) \right\} \quad (4)$$

with norm $\|x\|_{1-\omega} = \max_{0 \leq t \leq T} |t^{1-\omega} x(t)|$, as in [15, 16, 19].

Lemma 4. Consider the Hilfer fractional initial value problem:

$${}^H D_{0+}^{\alpha, \nu} x(t) = f(t, x(t)), \quad I_{0+}^{1-\omega} x(0+) = x_0 \quad (5)$$

with $\omega = \alpha + \nu(1 - \alpha)$. Then, it is equivalent to the Volterra integral equation:

$$x(t) = \frac{x_0}{\Gamma(\omega)} t^{\omega-1} + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \xi)^{\alpha-1} f(\xi, x(\xi)) d\xi \quad (6)$$

in the sense of [15, 16, 19].

Proof. By Definition 2, ${}^H D_{0+}^{\alpha, \nu} = I_{0+}^{\nu(1-\alpha)} \frac{d}{dt} I_{0+}^{(1-\nu)(1-\alpha)}$, and the semigroup law $I_{0+}^a I_{0+}^b = I_{0+}^{a+b}$ together with $\omega = \alpha + \nu(1 - \alpha)$ gives the composition identity $I_{0+}^{\alpha} {}^H D_{0+}^{\alpha, \nu} x(t) = x(t) - \frac{t^{\omega-1}}{\Gamma(\omega)} (I_{0+}^{1-\omega} x)(0+)$ valid for $x \in C_{1-\omega}$. Applying I_{0+}^{α} to ${}^H D_{0+}^{\alpha, \nu} x = f(t, x)$ and using this identity with $(I_{0+}^{1-\omega} x)(0+) = x_0$ produces $x(t) - \frac{x_0}{\Gamma(\omega)} t^{\omega-1} = I_{0+}^{\alpha} f(t, x(t)) = \frac{1}{\Gamma(\alpha)} \int_0^t (t - \xi)^{\alpha-1} f(\xi, x(\xi)) d\xi$, which is the stated Volterra equation. Conversely, applying ${}^H D_{0+}^{\alpha, \nu}$ to that equation and using ${}^H D_{0+}^{\alpha, \nu} (t^{\omega-1}) = 0$ together with ${}^H D_{0+}^{\alpha, \nu} I_{0+}^{\alpha} = \text{Id}$ recovers both the differential equation and the weighted initial condition, so the two formulations are equivalent. $\square$

Lemma 5. Let $\mathcal{F} : [0, T] \times \mathbb{R}_+^6 \rightarrow \mathbb{R}^6$ be continuous. If there is a constant $L > 0$ with $\|\mathcal{F}(t, X) - \mathcal{F}(t, Y)\| \leq L\|X - Y\|$ for all $X, Y \in \mathbb{R}_+^6$ and $t \in [0, T]$, then $\mathcal{F}$ is Lipschitz continuous in the state variables [8, 10].

Lemma 6. Let $u(t) \geq 0$ be continuous on $[0, T]$ and satisfy:

$$u(t) \leq a + \frac{b}{\Gamma(\alpha)} \int_0^t (t - \xi)^{\alpha-1} u(\xi) d\xi, \quad a, b \geq 0 \quad (7)$$

Then, $u(t) \leq a E_{\alpha}(bt^{\alpha})$, where $E_{\alpha}(z) = \sum_{n=0}^{\infty} z^n / \Gamma(\alpha n + 1)$ is the Mittag–Leffler function [6, 8, 10].

Proof. Let $(Bu)(t) = \frac{b}{\Gamma(\alpha)} \int_0^t (t - \xi)^{\alpha-1} u(\xi) d\xi = b I_{0+}^{\alpha} u(t)$, a monotone operator on non-negative functions. Iterating $u \leq a + Bu$ gives, for every $n \geq 1$ that $u(t) \leq a \sum_{k=0}^{n-1} (B^k \mathbf{1})(t) + (B^n u)(t)$. Since $B^k \mathbf{1} = b^k I_{0+}^{k\alpha} \mathbf{1} = b^k t^{k\alpha} / \Gamma(k\alpha + 1)$ by the semigroup property of the Riemann–Liouville integral, and the remainder satisfies $(B^n u)(t) \leq \|u\|_{\infty} b^n t^{n\alpha} / \Gamma(n\alpha + 1) \rightarrow 0$ as $n \rightarrow \infty$, letting $n \rightarrow \infty$ yields $u(t) \leq a \sum_{k=0}^{\infty} (bt^{\alpha})^k / \Gamma(k\alpha + 1) = a E_{\alpha}(bt^{\alpha})$. $\square$

3. Model Formulation and Description

We extend the fractional $SEI_c I_{\eta} V R$ epidemic model with vaccination by means of the Hilfer generalized fractional derivative of Definition 2. The population is divided into six classes: susceptible S ,

exposed E , asymptomatic infected I_c , symptomatic infected I_η , vaccinated V and recovered R , with total size $N(t) = S(t) + E(t) + I_c(t) + I_\eta(t) + V(t) + R(t)$.

The associated power-law memory kernel $K_\alpha(t - \xi) = (t - \xi)^{\alpha-1}/\Gamma(\alpha)$ shows that the current epidemic state depends on the whole history of the disease, while the type parameter ν selects the memory structure between the Riemann–Liouville and Caputo cases. The proposed model reads

$$\begin{aligned} {}^H D_{0,t}^{\alpha,\nu} S &= \Delta - \beta_1 S I_c - \beta_2 S I_\eta - (\kappa + \rho) S \\ {}^H D_{0,t}^{\alpha,\nu} E &= \beta_1 S I_c + \beta_2 S I_\eta + \varepsilon \beta_1 V I_c + \varepsilon \beta_2 V I_\eta - (\gamma + \rho) E \\ {}^H D_{0,t}^{\alpha,\nu} I_c &= \theta \gamma E - (d_1 + \rho) I_c \\ {}^H D_{0,t}^{\alpha,\nu} I_\eta &= (1 - \theta) \gamma E - (d_2 + \rho) I_\eta \\ {}^H D_{0,t}^{\alpha,\nu} V &= \kappa S - \varepsilon \beta_1 V I_c - \varepsilon \beta_2 V I_\eta - (\varrho + \rho) V \\ {}^H D_{0,t}^{\alpha,\nu} R &= \varrho V + d_1 I_c + d_2 I_\eta - \rho R \end{aligned} \quad (8)$$

with weighted initial data:

$$I_{0,t}^{(1-\nu)(1-\alpha)} Z(0) = Z_0 \geq 0, \quad Z \in \{S, E, I_c, I_\eta, V, R\} \quad (9)$$

here, Δ is recruitment, β_1, β_2 are the transmission rates from asymptomatic and symptomatic infectives, $\varepsilon \in [0, 1]$ measures vaccine ineffectiveness, κ is the vaccination rate, γ is the progression rate out of the exposed class, θ is the fraction of new infectives that are asymptomatic, d_1, d_2 are recovery rates, ϱ is the rate at which vaccinated individuals gain lasting immunity and ρ is the renewal rate. The compartments are summarised in Table 1.

Table 1

Description of the compartments of model (8)

Compartment	Meaning	Description
$S(t)$	Susceptible	Individuals at risk of infection
$E(t)$	Exposed	Infected individuals in the latent stage
$I_c(t)$	Asymptomatic infected	Infectious individuals without symptoms
$I_\eta(t)$	Symptomatic infected	Infectious individuals with symptoms
$V(t)$	Vaccinated	Individuals who have received vaccination
$R(t)$	Recovered	Individuals who have recovered from infection

The transfer of individuals between them is shown in Figure 1, which shows recruitment, infection, vaccination, breakthrough infection of vaccinated individuals and recovery flows between the six compartments. Each compartment also loses individuals at the renewal rate ρ . The overall workflow of the study is displayed in Figure 2.

For the analysis we write the state vector $X(t) = (S, E, I_c, I_\eta, V, R)^T$ and collect the right-hand sides of (8) into the map $\mathcal{F}(t, X) = (F_1, \dots, F_6)^T$, so that ${}^H D_{0+}^{\alpha,\nu} X(t) = \mathcal{F}(t, X(t))$ with $I_{0+}^{1-\omega} X(0+) = X_0$ and $\omega = \alpha + \nu(1 - \alpha)$.

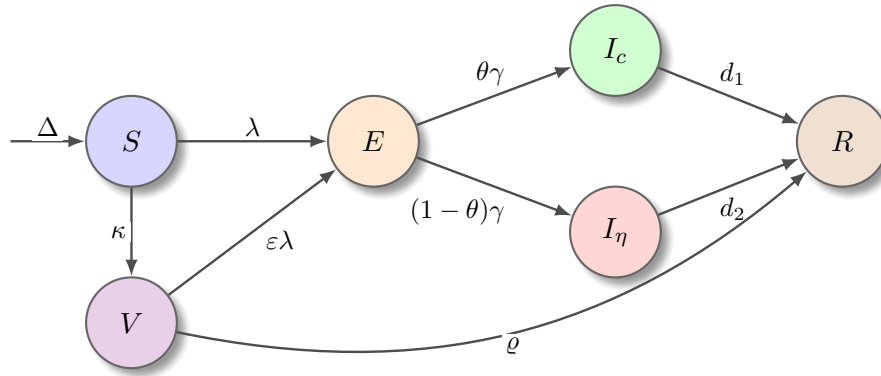


Fig. 1. Transfer diagram of the Hilfer generalized fractional $SEI_c I_\eta V R$ model (8) with force of infection $\lambda = \beta_1 I_c + \beta_2 I_\eta$

4. Existence and Uniqueness of Solutions

The components of $\mathcal{F}$ are:

$$\begin{aligned} F_1 &= \Delta - \beta_1 S I_c - \beta_2 S I_\eta - (\kappa + \rho) S, & F_2 &= \beta_1 S I_c + \beta_2 S I_\eta + \varepsilon \beta_1 V I_c + \varepsilon \beta_2 V I_\eta - (\gamma + \rho) E, \\ F_3 &= \theta \gamma E - (d_1 + \rho) I_c, & F_4 &= (1 - \theta) \gamma E - (d_2 + \rho) I_\eta, \\ F_5 &= \kappa S - \varepsilon \beta_1 V I_c - \varepsilon \beta_2 V I_\eta - (\varrho + \rho) V, & F_6 &= \varrho V + d_1 I_c + d_2 I_\eta - \rho R. \end{aligned}$$

By Lemma 4 the initial value problem is equivalent to the Volterra equation:

$$X(t) = \frac{X_0}{\Gamma(\omega)} t^{\omega-1} + \frac{1}{\Gamma(\alpha)} \int_0^t (t - \xi)^{\alpha-1} \mathcal{F}(\xi, X(\xi)) d\xi \quad (10)$$

and we define the operator $\mathcal{T} : C_{1-\omega}([0, T], \mathbb{R}^6) \rightarrow C_{1-\omega}([0, T], \mathbb{R}^6)$ by letting $(\mathcal{T}X)(t)$ equal the right-hand side of (10).

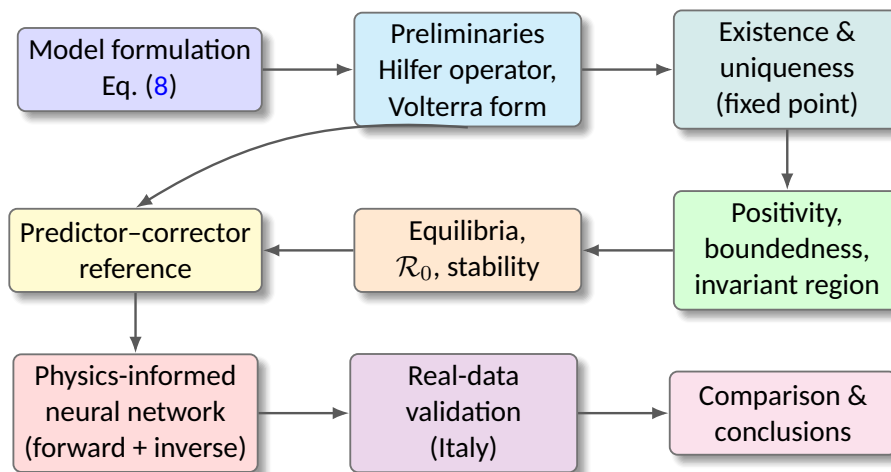


Fig. 2. Flow diagram, from model formulation and the Hilfer preliminaries through the qualitative analysis, the numerical reference scheme, the physics-informed neural network, and real-data validation to the concluding comparison

Theorem 7. Assume that $\mathcal{F} \in C([0, T] \times \mathbb{R}_+^6, \mathbb{R}^6)$, that $\mathcal{F}$ is Lipschitz in the state with constant $L > 0$ as in Lemma 5, and that:

$$\frac{L T^\alpha}{\Gamma(\alpha + 1)} < 1 \quad (11)$$

Then, Model (8) admits a unique solution in the weighted Banach space $C_{1-\omega}([0, T], \mathbb{R}^6)$.

Proof. The initial term $X_0 t^{\omega-1}/\Gamma(\omega)$ satisfies $t^{1-\omega} \cdot t^{\omega-1} = 1 \in C([0, T])$, and the convolution term is continuous with the same weight, so $\mathcal{T}$ maps $C_{1-\omega}([0, T], \mathbb{R}^6)$ into itself. The initial term is common to $\mathcal{T}X$ and $\mathcal{T}Y$ and cancels in the difference. Hence, for $X, Y \in C_{1-\omega}([0, T], \mathbb{R}^6)$ and each $t \in [0, T]$:

$$\|(\mathcal{T}X)(t) - (\mathcal{T}Y)(t)\| \leq \frac{1}{\Gamma(\alpha)} \int_0^t (t - \xi)^{\alpha-1} \|\mathcal{F}(\xi, X(\xi)) - \mathcal{F}(\xi, Y(\xi))\| d\xi.$$

Applying the Lipschitz bound of Lemma 5, $\|\mathcal{F}(\xi, X) - \mathcal{F}(\xi, Y)\| \leq L\|X(\xi) - Y(\xi)\| \leq L\|X - Y\|_{1-\omega}$, and evaluating the kernel integral in closed form $\frac{1}{\Gamma(\alpha)} \int_0^t (t - \xi)^{\alpha-1} d\xi = \frac{1}{\Gamma(\alpha)} \cdot \frac{t^\alpha}{\alpha} = \frac{t^\alpha}{\Gamma(\alpha+1)} \leq \frac{T^\alpha}{\Gamma(\alpha+1)}$, we obtain $\|\mathcal{T}X - \mathcal{T}Y\|_{1-\omega} = \sup_{t \in [0, T]} \|(\mathcal{T}X)(t) - (\mathcal{T}Y)(t)\| \leq \frac{LT^\alpha}{\Gamma(\alpha+1)} \|X - Y\|_{1-\omega}$, where we used $\Gamma(\alpha)\alpha = \Gamma(\alpha+1)$. Because $q := LT^\alpha/\Gamma(\alpha+1) < 1$, the operator $\mathcal{T}$ is a contraction, so by the Banach fixed point theorem it has a unique fixed point, which is the unique solution of model (8) on $[0, T]$. $\square$

The Lipschitz constant is finite because the nonlinearities in $\mathcal{F}$ are bilinear and the state variables remain bounded within the invariant region established in Section 5. Hence, the smallness condition holds on any finite horizon after a standard continuation argument based on Lemma 6.

5. Positivity and Boundedness of Solutions

We now show that solutions of Model (8) stay non-negative and bounded, so that the model is biologically meaningful.

Theorem 8. *If all parameters are non-negative and the weighted initial data (9) are non-negative, then every solution $X(t) = (S, E, I_c, I_\eta, V, R)^T$ of Model (8) satisfies $X(t) \geq 0$ for all $t > 0$.*

Proof. It suffices to check that the vector field is quasi-positive on the boundary of the non-negative orthant. On each boundary hyperplane:

$$\begin{aligned} {}^H D_{0+}^{\alpha, \nu} S|_{S=0} &= \Delta \geq 0, & {}^H D_{0+}^{\alpha, \nu} E|_{E=0} &= \beta_1 S I_c + \beta_2 S I_\eta + \varepsilon \beta_1 V I_c + \varepsilon \beta_2 V I_\eta \geq 0, \\ {}^H D_{0+}^{\alpha, \nu} I_c|_{I_c=0} &= \theta \gamma E \geq 0, & {}^H D_{0+}^{\alpha, \nu} I_\eta|_{I_\eta=0} &= (1 - \theta) \gamma E \geq 0, \\ {}^H D_{0+}^{\alpha, \nu} V|_{V=0} &= \kappa S \geq 0, & {}^H D_{0+}^{\alpha, \nu} R|_{R=0} &= \varrho V + d_1 I_c + d_2 I_\eta \geq 0. \end{aligned}$$

Thus, the field points inward on every face of $\mathbb{R}_+^6$, and by the positivity property of Hilfer fractional systems the solution remains non-negative for all $t > 0$. $\square$

Theorem 9. *Every non-negative solution of Model (8) is ultimately bounded and enters the positively invariant region:*

$$\Omega = \left\{ X(t) \in \mathbb{R}_+^6 : 0 \leq N(t) \leq \frac{\Delta}{\rho} \right\}, \quad N = S + E + I_c + I_\eta + V + R \quad (12)$$

Proof. Adding the six equations of (8) and using the linearity of the Hilfer operator, every transmission term $\beta_i S I_j$ cancels against its counterpart in the exposed equation, every breakthrough term $\varepsilon \beta_i V I_j$ cancels between the exposed and vaccinated equations, and the transfer terms combine as:

$$\begin{aligned} -(\kappa + \rho)S + \kappa S &= -\rho S, & -(\gamma + \rho)E + \theta \gamma E + (1 - \theta) \gamma E &= -\rho E, \\ -(d_1 + \rho)I_c + d_1 I_c &= -\rho I_c, & -(d_2 + \rho)I_\eta + d_2 I_\eta &= -\rho I_\eta, & -(\varrho + \rho)V + \varrho V &= -\rho V, \end{aligned}$$

together with $-\rho R$ from the recovered equation. Only the recruitment term survives among the couplings, so ${}^H D_{0+}^{\alpha,\nu} N(t) = \Delta - \rho(S + E + I_c + I_\eta + V + R) = \Delta - \rho N(t)$. By Lemma 4 this scalar linear Hilfer equation with $I_{0+}^{1-\omega} N(0+) = N_0$ has the closed-form solution $N(t) = \frac{N_0}{\Gamma(\omega)} t^{\omega-1} E_{\alpha,\omega}(-\rho t^\alpha) + \Delta t^\alpha E_{\alpha,\alpha+1}(-\rho t^\alpha)$, $N_0 = \sum Z_0$, where $E_{\alpha,\beta}(z) = \sum_{n \geq 0} z^n / \Gamma(\alpha n + \beta)$ is the two-parameter Mittag-Leffler function; this is verified by applying ${}^H D_{0+}^{\alpha,\nu}$ term by term and using ${}^H D_{0+}^{\alpha,\nu} [t^\alpha E_{\alpha,\alpha+1}(-\rho t^\alpha)] = 1 - \rho t^\alpha E_{\alpha,\alpha+1}(-\rho t^\alpha)$. For $\rho > 0$ the first term decays to zero and $t^\alpha E_{\alpha,\alpha+1}(-\rho t^\alpha) \rightarrow 1/\rho$, so $\limsup_{t \rightarrow \infty} N(t) \leq \Delta/\rho$. Consequently every non-negative solution eventually enters Ω , which is therefore positively invariant, and the model is biologically well posed. $\square$

6. Equilibria, Basic Reproduction Number and Stability

Because the Hilfer operator changes the memory structure but not the algebraic steady-state equations, the equilibria and the basic reproduction number are obtained from the right-hand side of Eq. (8).

At the disease-free equilibrium the infected classes vanish, $E = I_c = I_\eta = 0$. Setting the Hilfer derivatives in Eq. (8) to zero and substituting these values, the first, fifth and sixth balance equations reduce to:

$$\Delta - (\kappa + \rho)S^0 = 0, \quad \kappa S^0 - (\varrho + \rho)V^0 = 0, \quad \varrho V^0 - \rho R^0 = 0 \quad (13)$$

because every bilinear infection term contains a factor I_c or I_η and therefore drops out. Solving the first relation gives $S^0 = \Delta/(\kappa + \rho)$, inserting this into the second gives $V^0 = \kappa S^0/(\varrho + \rho)$, and the third then yields $R^0 = \varrho V^0/\rho$. Written out, $\mathcal{E}_0 = (S^0, 0, 0, 0, V^0, R^0)$ with:

$$S^0 = \frac{\Delta}{\kappa + \rho}, \quad V^0 = \frac{\kappa \Delta}{(\kappa + \rho)(\varrho + \rho)}, \quad R^0 = \frac{\varrho \kappa \Delta}{\rho(\kappa + \rho)(\varrho + \rho)} \quad (14)$$

For the baseline of Table 2 these evaluate to $\mathcal{E}_0 = (0.314, 0, 0, 0, 0.148, 0.539)$, and one checks that $S^0 + V^0 + R^0 = \Delta/\rho = 1$, consistent with the invariant total of Theorem 9. Taking the infected compartments (E, I_c, I_η) and applying the next-generation matrix method, the new-infection and transition matrices at $\mathcal{E}_0$ are:

$$F = \begin{pmatrix} 0 & \beta_1(S^0 + \varepsilon V^0) & \beta_2(S^0 + \varepsilon V^0) \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}, \quad \mathcal{V} = \begin{pmatrix} \gamma + \rho & 0 & 0 \\ -\theta\gamma & d_1 + \rho & 0 \\ -(1-\theta)\gamma & 0 & d_2 + \rho \end{pmatrix}$$

here, F collects the appearance of new infections in the exposed class and $\mathcal{V}$ collects the internal progression and removal of the infected subsystem. Because $\mathcal{V}$ is lower triangular, its determinant is $\det \mathcal{V} = (\gamma + \rho)(d_1 + \rho)(d_2 + \rho) > 0$ and its inverse is obtained by forward substitution:

$$\mathcal{V}^{-1} = \begin{pmatrix} \frac{1}{\gamma + \rho} & 0 & 0 \\ \frac{\theta\gamma}{(\gamma + \rho)(d_1 + \rho)} & \frac{1}{d_1 + \rho} & 0 \\ \frac{(1-\theta)\gamma}{(\gamma + \rho)(d_2 + \rho)} & 0 & \frac{1}{d_2 + \rho} \end{pmatrix}$$

Writing $M := S^0 + \varepsilon V^0$ for the effective susceptible pool and carrying out the product, only the

first row of $F\mathcal{V}^{-1}$ is nonzero:

$$F\mathcal{V}^{-1} = \begin{pmatrix} \frac{\gamma M}{\gamma + \rho} \left(\frac{\beta_1 \theta}{d_1 + \rho} + \frac{\beta_2(1 - \theta)}{d_2 + \rho} \right) & \frac{\beta_1 M}{d_1 + \rho} & \frac{\beta_2 M}{d_2 + \rho} \\ 0 & 0 & 0 \\ 0 & 0 & 0 \end{pmatrix}$$

The characteristic polynomial of this rank-one matrix is $\det(\lambda I - F\mathcal{V}^{-1}) = \lambda^2(\lambda - \text{tr } F\mathcal{V}^{-1})$, so two eigenvalues vanish and the third equals the trace. The basic reproduction number is the spectral radius $\mathcal{R}_0 = \rho(F\mathcal{V}^{-1}) = \text{tr } F\mathcal{V}^{-1}$. Hence:

$$\mathcal{R}_0 = \frac{\gamma(S^0 + \varepsilon V^0)}{\gamma + \rho} \left(\frac{\beta_1 \theta}{d_1 + \rho} + \frac{\beta_2(1 - \theta)}{d_2 + \rho} \right) \quad (15)$$

Substituting S^0 and V^0 from Eq. (14), and using $S^0 + \varepsilon V^0 = \frac{\Delta}{\kappa + \rho} \left(1 + \frac{\varepsilon \kappa}{\varrho + \rho} \right)$, yields:

$$\mathcal{R}_0 = \frac{\gamma \Delta}{(\gamma + \rho)(\kappa + \rho)} \left(1 + \frac{\varepsilon \kappa}{\varrho + \rho} \right) \left(\frac{\beta_1 \theta}{d_1 + \rho} + \frac{\beta_2(1 - \theta)}{d_2 + \rho} \right) \quad (16)$$

Moreover $\mathcal{R}_0 = \mathcal{R}_{0c} + \mathcal{R}_{0\eta}$, where:

$$\mathcal{R}_{0c} = \frac{\gamma(S^0 + \varepsilon V^0)}{\gamma + \rho} \frac{\beta_1 \theta}{d_1 + \rho}, \quad \mathcal{R}_{0\eta} = \frac{\gamma(S^0 + \varepsilon V^0)}{\gamma + \rho} \frac{\beta_2(1 - \theta)}{d_2 + \rho} \quad (17)$$

representing the contributions of the asymptomatic and symptomatic classes.

Setting the right-hand sides of Eq. (8) to zero and solving for an endemic state, the third and fourth equations give:

$$I_c = \frac{\theta \gamma E}{d_1 + \rho}, \quad I_\eta = \frac{(1 - \theta) \gamma E}{d_2 + \rho} \quad (18)$$

so the endemic force of infection is a multiple of the exposed level:

$$\lambda^* = \beta_1 I_c + \beta_2 I_\eta = \left(\frac{\beta_1 \theta \gamma}{d_1 + \rho} + \frac{\beta_2(1 - \theta) \gamma}{d_2 + \rho} \right) E =: A E \quad (19)$$

The susceptible balance $\Delta - \lambda^* S - (\kappa + \rho) S = 0$, then gives $S = \Delta / (A E + \kappa + \rho)$. The vaccinated balance $\kappa S - \varepsilon \lambda^* V - (\varrho + \rho) V = 0$ gives $V = \kappa S / (\varepsilon A E + \varrho + \rho) = \kappa \Delta / [(A E + \kappa + \rho)(\varepsilon A E + \varrho + \rho)]$, while the recovered balance yields $R = (\varrho V + d_1 I_c + d_2 I_\eta) / \rho$. Dividing the exposed balance $\lambda^* S + \varepsilon \lambda^* V - (\gamma + \rho) E = 0$ by $E > 0$ leaves $A S + \varepsilon A V = \gamma + \rho$. Substituting S and V and clearing the denominator $(A E + \kappa + \rho)(\varepsilon A E + \varrho + \rho)$, the endemic level $E^* > 0$ solves:

$$P(E) = (\gamma + \rho)(A E + \kappa + \rho)(\varepsilon A E + \varrho + \rho) - A \Delta(\varrho + \rho + \varepsilon A E + \varepsilon \kappa) = 0 \quad (20)$$

which is a quadratic in E with positive leading coefficient $\varepsilon A^2(\gamma + \rho)$. The endemic equilibrium is then $\mathcal{E}^* = (S^*, E^*, I_c^*, I_\eta^*, V^*, R^*)$ obtained by back substitution of E^* .

Lemma 10. *Model (8) always has the disease-free equilibrium $\mathcal{E}_0$. If $\mathcal{R}_0 > 1$, it has at least one positive endemic equilibrium $\mathcal{E}^*$.*

Proof. The disease-free equilibrium follows by setting $E = I_c = I_\eta = 0$ in Eq. (20), which was verified above. For the endemic case, evaluating Eq. (20) at $E = 0$ gives $P(0) = (\gamma + \rho)(\kappa + \rho)(\varrho + \rho) - A\Delta(\varrho + \rho + \varepsilon\kappa)$. Now $A = \gamma\left(\frac{\beta_1\theta}{d_1+\rho} + \frac{\beta_2(1-\theta)}{d_2+\rho}\right)$, so $\frac{A\Delta(\varrho+\rho+\varepsilon\kappa)}{(\gamma+\rho)(\kappa+\rho)(\varrho+\rho)} = \frac{\gamma\Delta}{(\gamma+\rho)(\kappa+\rho)}\left(1 + \frac{\varepsilon\kappa}{\varrho+\rho}\right)\left(\frac{\beta_1\theta}{d_1+\rho} + \frac{\beta_2(1-\theta)}{d_2+\rho}\right) = \mathcal{R}_0$, where the middle factor used $(\varrho + \rho + \varepsilon\kappa)/(\varrho + \rho) = 1 + \varepsilon\kappa/(\varrho + \rho)$. Therefore $P(0) = (\gamma + \rho)(\kappa + \rho)(\varrho + \rho)(1 - \mathcal{R}_0)$.

If $\mathcal{R}_0 > 1$ then $P(0) < 0$, while the positive leading coefficient of the quadratic gives $\lim_{E \rightarrow \infty} P(E) = +\infty$. By the intermediate value theorem there is a root $E^* > 0$, and back substitution then yields $I_c^*, I_\eta^*, S^*, V^*, R^* > 0$, so a biologically admissible endemic equilibrium exists. $\square$

Since R does not appear in the first five equations, the transmission dynamics are governed by the reduced state (S, E, I_c, I_η, V) . We use the following fractional stability criterion.

Lemma 11. For the linear Hilfer system ${}^H D_{0+}^{\alpha,\nu} x(t) = Bx(t)$ with $0 < \alpha < 1$, the equilibrium $x = 0$ is locally asymptotically stable if every eigenvalue λ_i of B satisfies $|\arg(\lambda_i)| > \alpha\pi/2$.

Theorem 12. The disease-free equilibrium $\mathcal{E}_0$ is locally asymptotically stable if $\mathcal{R}_0 < 1$ and unstable if $\mathcal{R}_0 > 1$.

Proof. The linearised infected subsystem at $\mathcal{E}_0$ is governed by $F - \mathcal{V}$, and by the next-generation theory $\rho(F\mathcal{V}^{-1}) = \mathcal{R}_0$. If $\mathcal{R}_0 < 1$ all infection-related eigenvalues lie in the stable sector of Lemma 11, so $\mathcal{E}_0$ is locally asymptotically stable; if $\mathcal{R}_0 > 1$ an eigenvalue leaves that sector and $\mathcal{E}_0$ becomes unstable. $\square$

Theorem 13. Assume $\mathcal{R}_0 > 1$ and that $\mathcal{E}^*$ exists. Then $\mathcal{E}^*$ is locally asymptotically stable provided every eigenvalue λ_i of the Jacobian $J(\mathcal{E}^*)$ satisfies $|\arg(\lambda_i)| > \alpha\pi/2$ for $i = 1, \dots, 6$.

Proof. The Jacobian of (8) at a general equilibrium is:

$$J = \begin{pmatrix} -\beta_1 I_c - \beta_2 I_\eta - (\kappa + \rho) & 0 & -\beta_1 S & -\beta_2 S & 0 & 0 \\ \beta_1 I_c + \beta_2 I_\eta & -(\gamma + \rho) & \beta_1 S + \varepsilon\beta_1 V & \beta_2 S + \varepsilon\beta_2 V & \varepsilon\beta_1 I_c + \varepsilon\beta_2 I_\eta & 0 \\ 0 & \theta\gamma & -(d_1 + \rho) & 0 & 0 & 0 \\ 0 & (1 - \theta)\gamma & 0 & -(d_2 + \rho) & 0 & 0 \\ \kappa & 0 & -\varepsilon\beta_1 V & -\varepsilon\beta_2 V & -\varepsilon\beta_1 I_c - \varepsilon\beta_2 I_\eta - (\varrho + \rho) & 0 \\ 0 & 0 & d_1 & d_2 & \varrho & -\rho \end{pmatrix}$$

Evaluating J at $\mathcal{E}^*$ and applying Lemma 11, the endemic equilibrium is locally asymptotically stable whenever the stated argument condition holds. $\square$

7. Physics-Informed Neural Network

We now construct a fractional physics-informed neural network for model (8). Rather than differentiating the network with a fractional operator, we build the physics residual directly on the Volterra formulation (10), which is the exact integral form of the Hilfer initial value problem. For the network experiments we use the Caputo realization of the Hilfer operator, $\nu = 1$ and $\omega = 1$, so the singular initial term in (10) reduces to the constant X_0 ; the general type ν enters through the analysis of Sections 2 and 8.

Let $\mathcal{N}_\theta : [0, T] \rightarrow \mathbb{R}^6$ be a densely connected feed-forward network with parameters θ . To respect the initial data exactly we use the hard-constraint trial solution:

$$\hat{X}_\theta(t) = X_0 + \frac{t}{T} g_\theta(t) \quad (21)$$

so that $\widehat{X}_\theta(0) = X_0$ for any θ . The map g_θ is a tanh network acting on the Fourier feature embedding $\phi(t) = (t, \sin(\pi t), \cos(\pi t), \dots, \sin(k\pi t), \cos(k\pi t))$, $t \in [0, 1]$, which lets the network represent the sharp epidemic peak without spectral bias. On a uniform grid $t_m = mh$, $m = 0, \dots, M$, the fractional integral in (10) is discretised by product-integration weights identical to those of the predictor-corrector scheme [30]:

$$(I^\alpha g)(t_m) \approx \sum_{j=0}^M W_{mj} g(t_j), \quad W_{mj} = \frac{h^\alpha}{\Gamma(\alpha + 2)} \tilde{a}_{j,m} \quad (22)$$

where $\tilde{a}_{m,m} = 1$, $\tilde{a}_{0,m} = (m-1)^{\alpha+1} - (m-1-\alpha)m^\alpha$, and for $1 \leq j \leq m-1$:

$$\tilde{a}_{j,m} = (m-j+1)^{\alpha+1} + (m-j-1)^{\alpha+1} - 2(m-j)^{\alpha+1}.$$

These coefficients are obtained by replacing g with its piecewise-linear interpolant on the grid and integrating the singular kernel against it exactly. On $[t_j, t_{j+1}]$ one writes $g(\xi) \approx g(t_j) + \frac{\xi-t_j}{h}(g(t_{j+1}) - g(t_j))$, and the elementary kernel moments:

$$\frac{1}{\Gamma(\alpha)} \int_{t_j}^{t_{j+1}} (t_m - \xi)^{\alpha-1} d\xi = \frac{h^\alpha}{\Gamma(\alpha+1)} [(m-j)^\alpha - (m-j-1)^\alpha], \quad \frac{1}{\Gamma(\alpha)} \int_{t_j}^{t_{j+1}} (t_m - \xi)^{\alpha-1} (\xi - t_j) d\xi$$

are evaluated in closed form; collecting the coefficient that multiplies each nodal value $g(t_j)$ produces the second difference $\tilde{a}_{j,m}$ above, while the two boundary intervals give $\tilde{a}_{0,m}$ and $\tilde{a}_{m,m} = 1$. Since these are precisely the corrector weights of [30], the discrete integral is consistent with the reference scheme, with quadrature error of order $h^{1+\alpha}$ uniformly on $[0, T]$ for $g \in C^2$. The physics residual of the network at the collocation nodes is:

$$r_i(t_m) = \widehat{X}_{\theta,i}(t_m) - X_{0,i} - \sum_{j=0}^M W_{mj} F_i(t_j, \widehat{X}_\theta(t_j)), \quad i = 1, \dots, 6 \quad (23)$$

and the physics loss is the scaled mean square residual:

$$\mathcal{L}_{\text{phys}}(\theta) = \frac{1}{6(M+1)} \sum_{m=0}^M \sum_{i=1}^6 \left(\frac{r_i(t_m)}{s_i} \right)^2 \quad (24)$$

where $s_i > 0$ are characteristic magnitudes of the compartments that balance the contributions of the large and small classes. In the forward problem the total loss is $\mathcal{L}_{\text{phys}}$ alone, since the initial condition is enforced by construction through Eq. (21).

For the inverse problem the transmission rates β_1, β_2 and a reporting fraction $\xi \in (0, 1)$ are treated as trainable parameters, and a data term is added. If $\{(\tau_k, y_k)\}$ are observed daily incidence values, the modelled incidence is $\xi(1-\theta)\gamma \widehat{E}_\theta(\tau_k)$ and the data loss is the relative mean square error:

$$\mathcal{L}_{\text{data}}(\theta, \beta_1, \beta_2, \xi) = \frac{1}{K} \sum_{k=1}^K \left(\frac{\xi(1-\theta)\gamma \widehat{E}_\theta(\tau_k) - y_k}{y_k} \right)^2 \quad (25)$$

so that the total objective is $\mathcal{L} = \mathcal{L}_{\text{phys}} + w_d \mathcal{L}_{\text{data}}$ with a weight $w_d > 0$. The network is trained first with the Adam optimiser and then refined with the limited-memory BFGS optimiser, which drives the residual to the level required for high accuracy.

The following consistency result links the physics loss to the true solution through the contraction established in Theorem 7.

Proposition 14. Let X^* be the unique solution of model (8) in the Caputo case $\nu = 1$, and let $\hat{X}_\theta$ be the trial solution (21). Denote by η the maximum residual $\max_{m,i} |r_i(t_m)|$ in (23) and by δ the quadrature error of the rule (22) on the exact solution. If $q := LT^\alpha/\Gamma(\alpha + 1) < 1$, then at the collocation nodes:

$$\max_m \|\hat{X}_\theta(t_m) - X^*(t_m)\| \leq \frac{\sqrt{6}\eta + \delta}{1 - q} \quad (26)$$

Proof. Write $\mathcal{T}$ for the Volterra operator of Eq. (10) with $\omega = 1$ and $\mathcal{T}_h$ for its product-integration discretisation (22). From Eq. (23), $\hat{X}_\theta = \mathcal{T}_h \hat{X}_\theta + r$ at the nodes, with $\|r\|_\infty \leq \sqrt{6}\eta$. Since $X^* = \mathcal{T}X^*$ and the rule has error δ , we also have $X^* = \mathcal{T}_h X^* + e$ with $\|e\|_\infty \leq \delta$. Subtracting $\hat{X}_\theta - X^* = \mathcal{T}_h \hat{X}_\theta - \mathcal{T}_h X^* + r - e$. By Lemma 5 the discrete operator inherits the Lipschitz constant, so $\|\mathcal{T}_h \hat{X}_\theta - \mathcal{T}_h X^*\|_\infty \leq q \|\hat{X}_\theta - X^*\|_\infty$. Hence $\|\hat{X}_\theta - X^*\|_\infty \leq q \|\hat{X}_\theta - X^*\|_\infty + \sqrt{6}\eta + \delta$, and solving for the error gives the stated bound. $\square$

Proposition 14 shows that once the physics loss is small, so that η is small, and the grid is fine, so that δ is small, the network solution is guaranteed to be close to the exact Hilfer solution, with a stability constant $1/(1 - q)$ controlled by the same contraction that guarantees uniqueness in Theorem 7. The numerical experiments of Section 8 confirm this behaviour.

8. Numerical Simulation and Sensitivity Analysis

The simulations use the biologically motivated baseline parameters of Table 2, expressed in population-fraction units so that the demographic total satisfies $N^* = \Delta/\rho = 1$. With these values the reproduction number is $\mathcal{R}_0 = 1.71$, the disease-free equilibrium is $\mathcal{E}_0 = (0.314, 0, 0, 0, 0.148, 0.539)$ and a pronounced epidemic wave develops. The reference trajectories are computed with the fractional predictor-corrector scheme [30] on a grid of step $h = 0.25$, which reproduces the fine-grid solution to a relative L^2 error below 10^{-5} .

Table 2

Baseline parameters used in the simulations (population-fraction units, time in days)

Parameter	Value	Meaning
Δ	1/365	recruitment rate
β_1	0.45	transmission from asymptomatic
β_2	0.62	transmission from symptomatic
ε	0.30	vaccine ineffectiveness
κ	0.006	vaccination rate
ρ	1/365	renewal rate
γ	1/5.2	progression out of exposed
θ	0.40	asymptomatic fraction
d_1	1/7	asymptomatic recovery
d_2	1/10	symptomatic recovery
ϱ	0.010	immunity gain of vaccinated

Figure 3 shows the evolution of all six compartments: the susceptible class declines as infection and vaccination proceed, the infected classes rise to a peak and decay, and the recovered class grows monotonically.

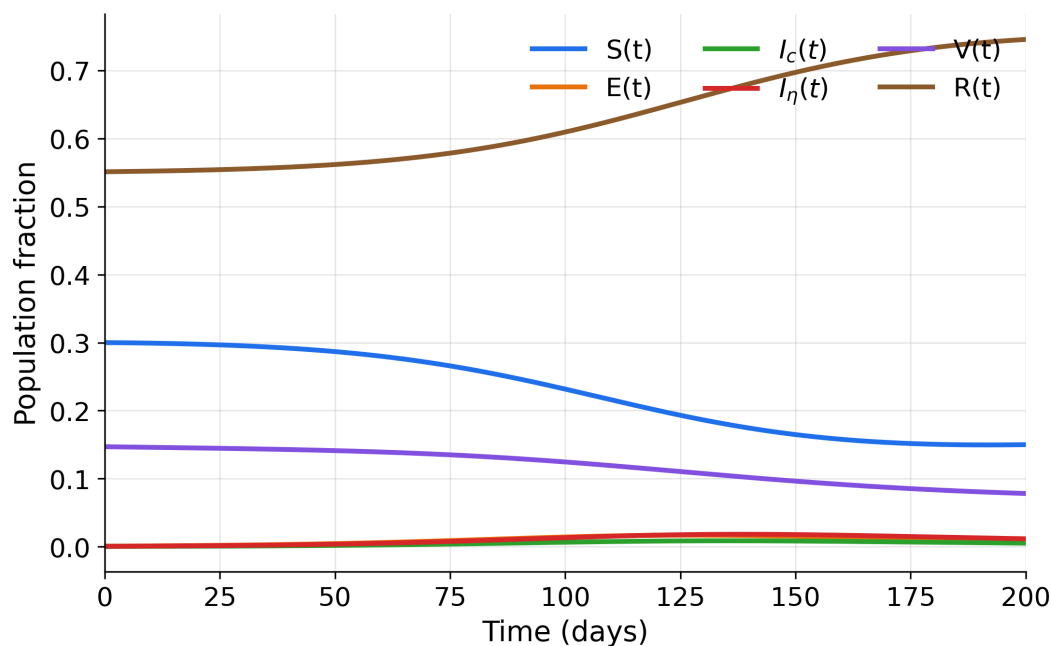


Fig. 3. Dynamics of all six compartments of Model (8) for the baseline parameters of Table 2 at $\alpha = 0.95$

The influence of the fractional order α is displayed in Figure 4. Smaller α corresponds to stronger memory, which lowers and broadens the infection peak and slows the return to equilibrium.

Figure 5 illustrates the effect of the Hilfer type parameter ν through the general solver of Section 2; as ν increases from the Riemann-Liouville case to the Caputo case the effective initial weighting and the transient amplitude change appreciably, confirming that the type parameter is an independent modelling degree of freedom.

The role of the control parameters is examined next. Figure 6 shows that increasing the vaccination rate κ substantially reduces the infection peak, whereas Figure 7 shows that increasing the vaccine ineffectiveness ε raises it. These trends are consistent with the closed-form Eq. (16): Figure 8 plots $\mathcal{R}_0$ as a decreasing function of κ , crossing the threshold $\mathcal{R}_0 = 1$ at a critical vaccination level, and Figure 9 plots $\mathcal{R}_0$ as an increasing function of ε . The joint dependence is summarised by the contour map of Figure 10, in which the white curve marks $\mathcal{R}_0 = 1$ and separates the eradication and persistence regions in the (κ, ε) plane.

The normalized sensitivity index of $\mathcal{R}_0$ with respect to a parameter p is $\Upsilon_p = (\partial \mathcal{R}_0 / \partial p)(p / \mathcal{R}_0)$. Several of these indices admit closed forms that can be read directly from Eq. (16). Writing:

$$\mathcal{R}_0 = C(\kappa) \left(1 + \frac{\varepsilon \kappa}{\varrho + \rho} \right) Q, \quad C(\kappa) = \frac{\gamma \Delta}{(\gamma + \rho)(\kappa + \rho)}, \quad Q = \frac{\beta_1 \theta}{d_1 + \rho} + \frac{\beta_2 (1 - \theta)}{d_2 + \rho}$$

the linear dependence on Δ gives $\Upsilon_\Delta = +1$. Since β_1 and β_2 enter only through Q :

$$\Upsilon_{\beta_1} = \frac{\beta_1 \theta / (d_1 + \rho)}{Q} = \frac{\mathcal{R}_{0c}}{\mathcal{R}_0}, \quad \Upsilon_{\beta_2} = \frac{\beta_2 (1 - \theta) / (d_2 + \rho)}{Q} = \frac{\mathcal{R}_{0\eta}}{\mathcal{R}_0}$$

so the two transmission indices are the fractional contributions of the asymptomatic and symptomatic routes and satisfy $\Upsilon_{\beta_1} + \Upsilon_{\beta_2} = 1$. Differentiating the recovery dependence:

$$\Upsilon_{d_2} = -\frac{\mathcal{R}_{0\eta}}{\mathcal{R}_0} \frac{d_2}{d_2 + \rho}, \quad \Upsilon_{d_1} = -\frac{\mathcal{R}_{0c}}{\mathcal{R}_0} \frac{d_1}{d_1 + \rho}$$

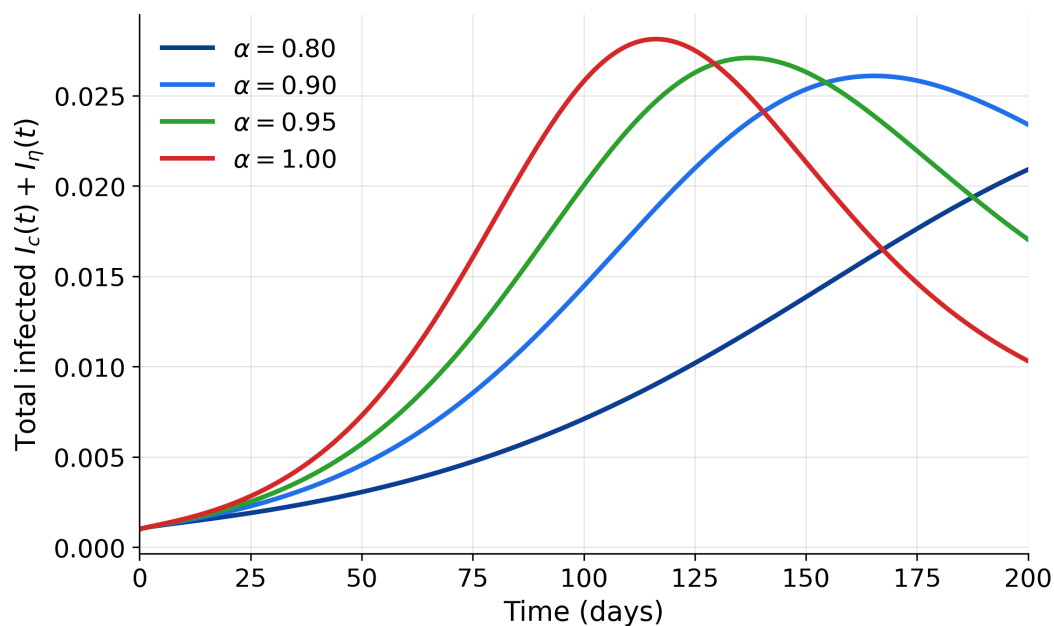


Fig. 4. Effect of the fractional order α on the total infected population $I_c(t) + I_\eta(t)$

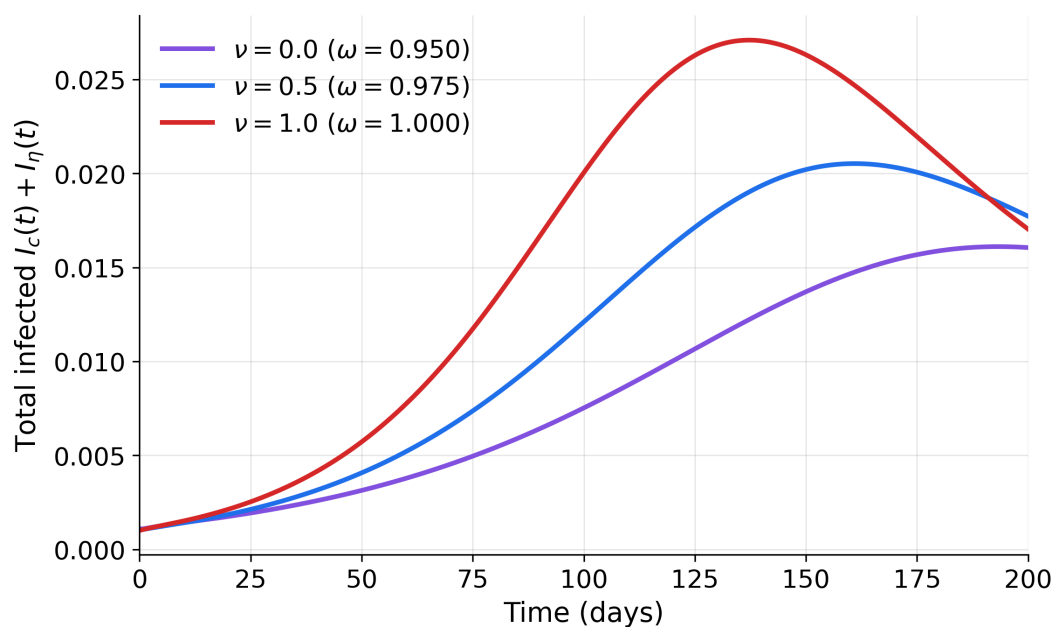


Fig. 5. Effect of the Hilfer type parameter ν on the total infected population, with the induced parameter $\omega = \alpha + \nu(1 - \alpha)$ reported in the legend

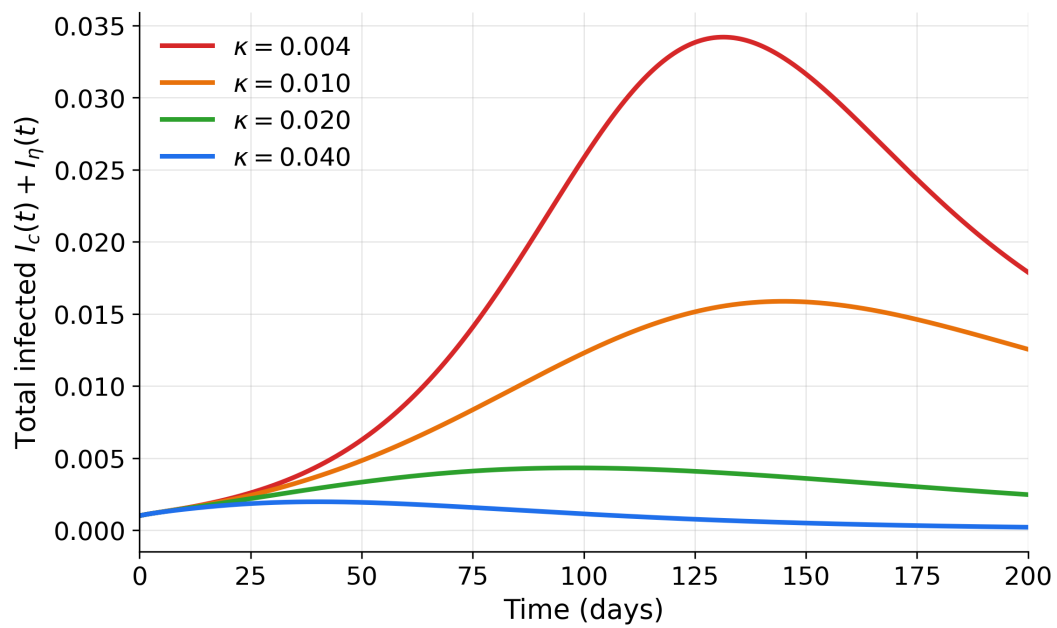


Fig. 6. Effect of the vaccination rate κ on the total infected population

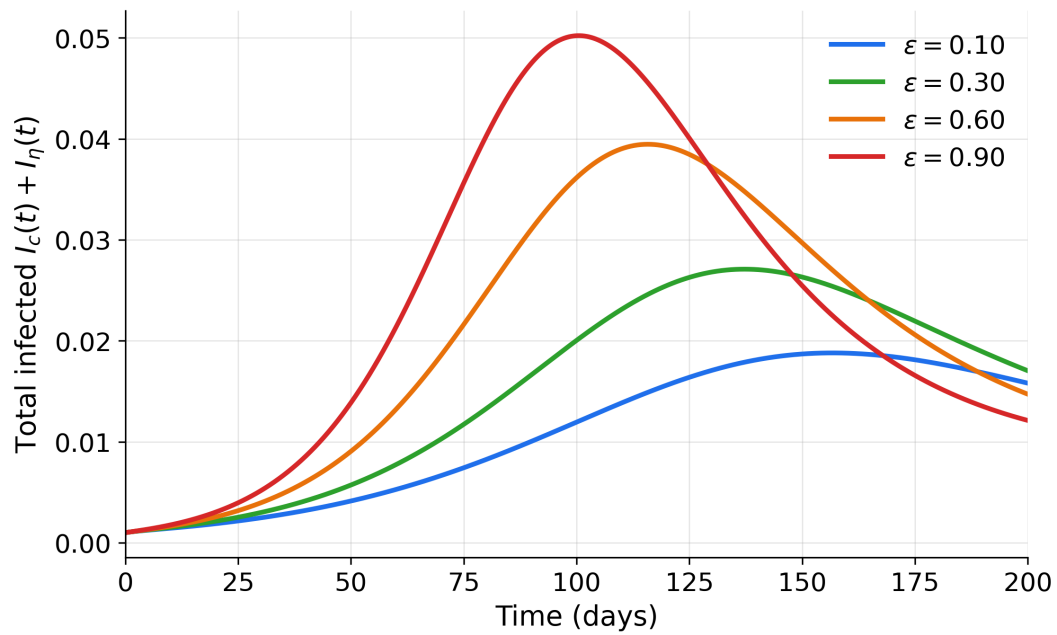


Fig. 7. Effect of the vaccine ineffectiveness parameter ε on the total infected population

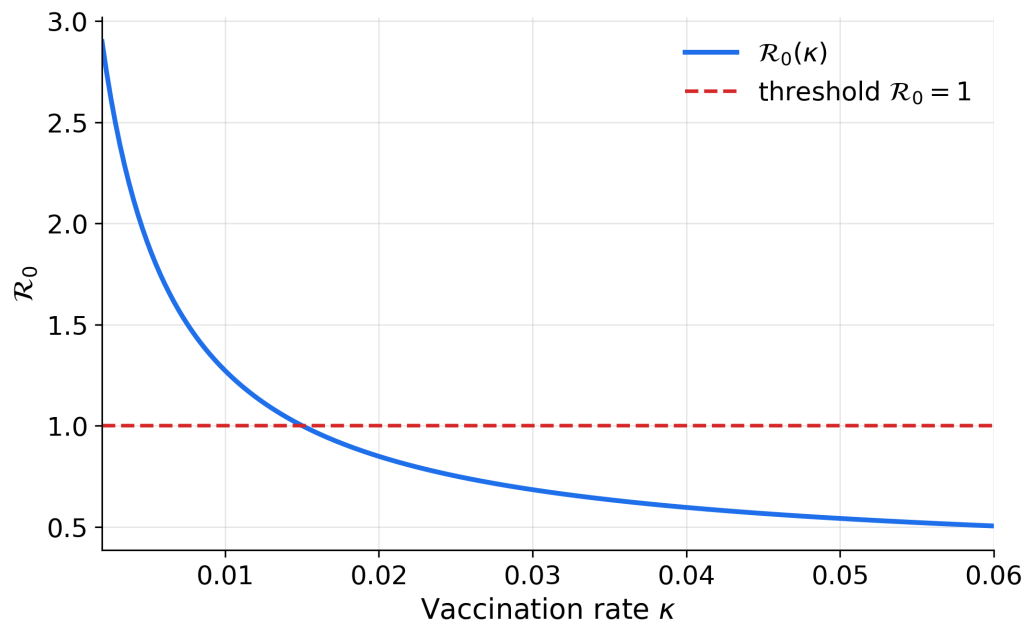


Fig. 8. Variation of the basic reproduction number $\mathcal{R}_0$ with the vaccination rate κ ; the dashed line is the threshold $\mathcal{R}_0 = 1$

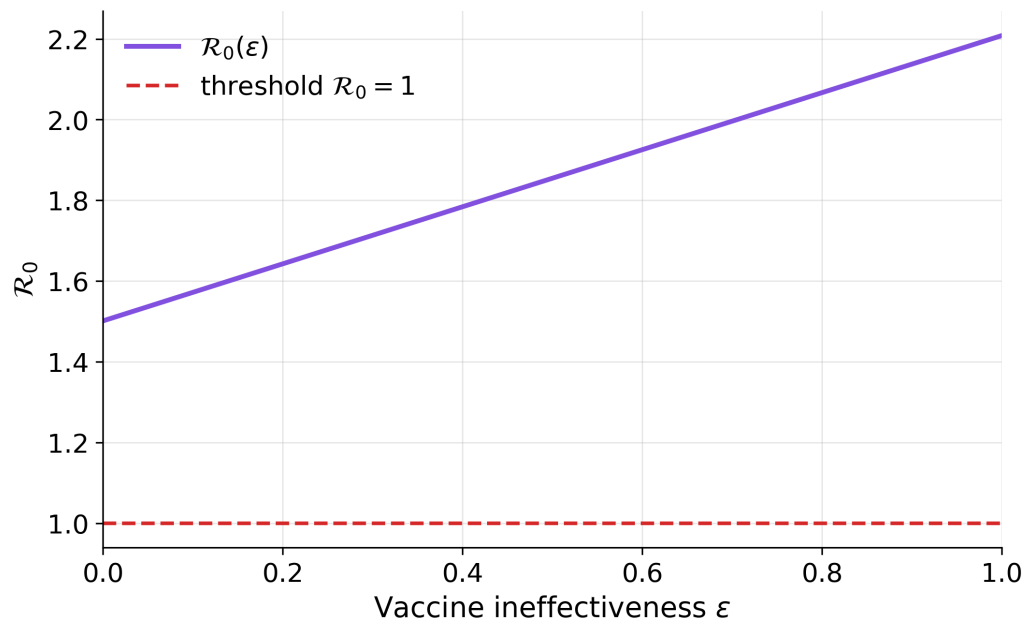


Fig. 9. Variation of the basic reproduction number $\mathcal{R}_0$ with the vaccine ineffectiveness parameter ϵ

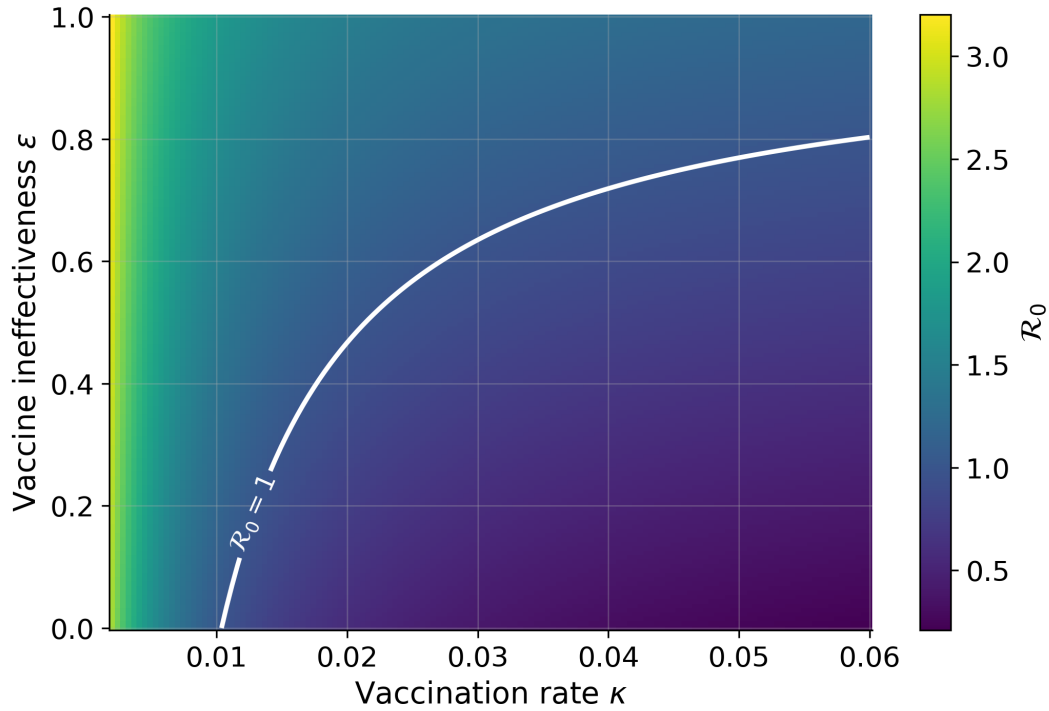


Fig. 10. Contour map of $\mathcal{R}_0$ over the (κ, ε) plane; the white curve is the critical threshold $\mathcal{R}_0 = 1$

and the vaccination rate, which acts through both $C(\kappa)$ and the bracket, gives:

$$\Upsilon_{\kappa} = \frac{\varepsilon\kappa}{(\varrho + \rho) + \varepsilon\kappa} - \frac{\kappa}{\kappa + \rho} \quad (27)$$

Evaluating at the baseline of Table 2 yields $\Upsilon_{\Delta} = +1$, $\Upsilon_{\beta_2} = +0.75$, $\Upsilon_{d_2} = -0.73$, $\Upsilon_{\beta_1} = +0.25$ and $\Upsilon_{\kappa} = -0.56$, so the symptomatic transmission and recovery rates dominate the transmission response while vaccination is the strongest control lever. Figure 11 ranks the indices, and the ordering agrees with Figures 6 and 8.

The parameter dependence summarised above is displayed as three-dimensional response surfaces in Figures 12–15, which give a geometric view of the same relationships. Figure 12 shows $\mathcal{R}_0$ as a surface over the pairs (κ, ε) , (β_1, β_2) and (θ, d_2) , with the critical curve $\mathcal{R}_0 = 1$ traced on the base plane; the decrease in κ and increase in ε found in Figures 8 and 9 reappear as the slope of the surface. Figure 13 follows the total infected population as a sheet in time against the fractional order α , the vaccination rate κ and the vaccine ineffectiveness ε , so that the lowering and broadening of the epidemic peak under stronger memory or stronger control is read from a single surface. Figure 14 places the influence of the Hilfer type ν beside two phase-space views of the trajectory, coloured by time and by vaccination rate, that show the approach to equilibrium in the $(S, E, I_c + I_{\eta})$ and (S, I_c, I_{η}) coordinates. Figure 15 gathers the peak infected level over (κ, ε) , the reproduction number over (κ, ϱ) and the signed sensitivity indices, reinforcing that vaccination and symptomatic transmission govern the threshold.

We now validate the physics-informed neural network of Section 7 on the forward problem. The network has five hidden layers of width 96 with six Fourier modes and is trained on the collocation grid with step $h = 0.25$ over the horizon $[0, 150]$. Figure 16 overlays the network solution on the predictor–corrector reference for all six compartments; the two are visually indistinguishable, with a global relative L^2 error of 8.8×10^{-4} and a maximum pointwise error of 1.2×10^{-3} . Figure 17 shows the training loss, which decreases through the Adam phase and is then driven to the level

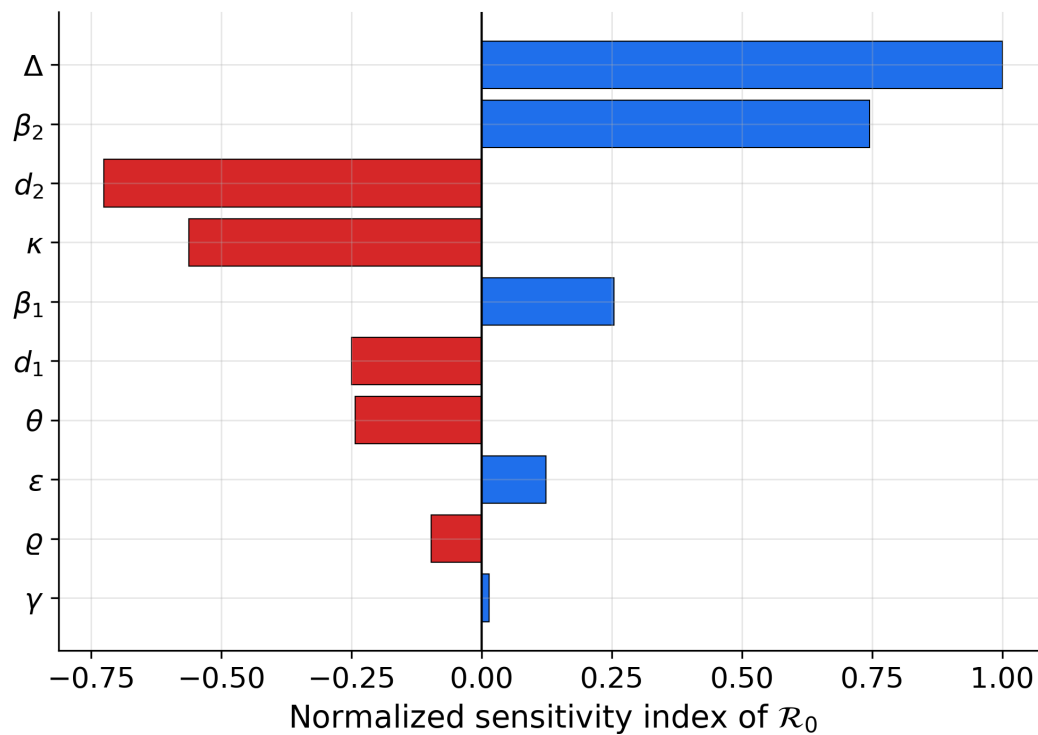


Fig. 11. Normalized sensitivity indices of the basic reproduction number $\mathcal{R}_0$ at the baseline parameters; positive bars increase and negative bars decrease $\mathcal{R}_0$

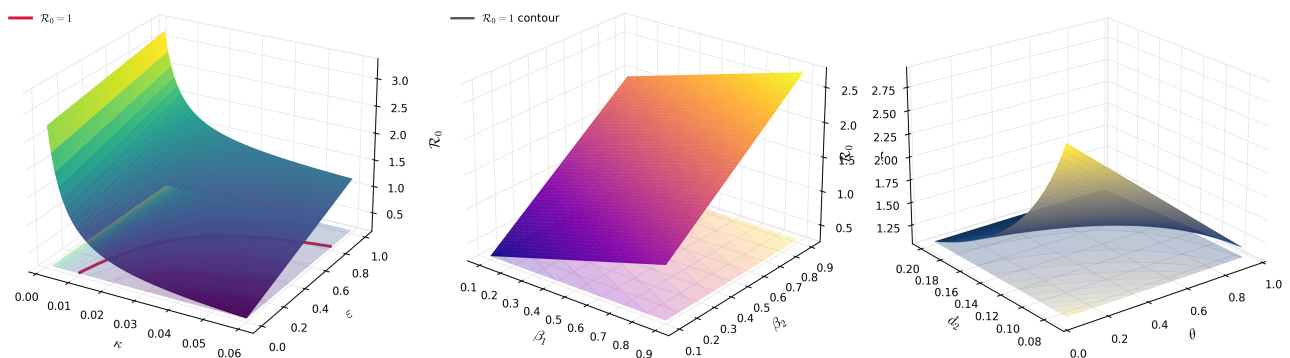


Fig. 12. Three-dimensional response surfaces of the basic reproduction number $\mathcal{R}_0$; left: dependence on the vaccination rate κ and the vaccine ineffectiveness ϵ , with the critical curve $\mathcal{R}_0 = 1$ traced on the base plane; centre: dependence on the asymptomatic and symptomatic transmission rates β_1, β_2 ; right: dependence on the asymptomatic fraction θ and the symptomatic recovery rate d_2

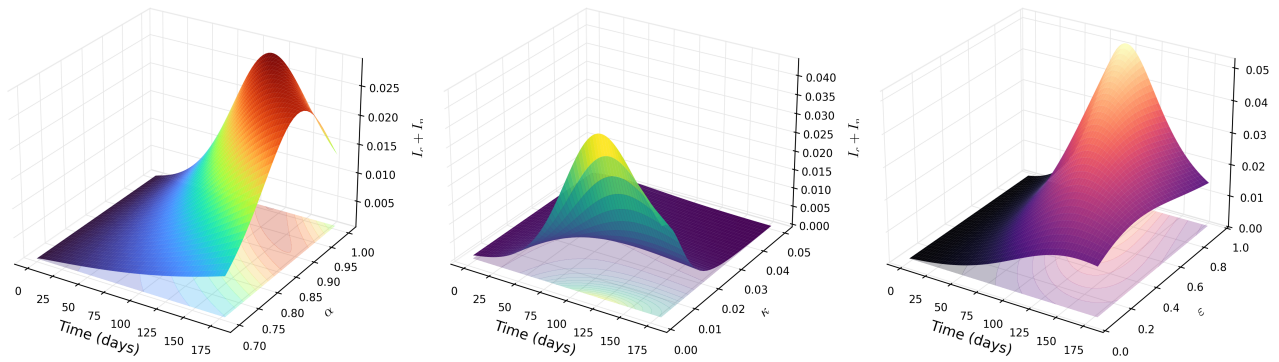


Fig. 13. Total infected population $I_c(t) + I_\eta(t)$ as a surface in time against, from left to right, the fractional order α , the vaccination rate κ and the vaccine ineffectiveness ε ; stronger memory or stronger control lowers and broadens the epidemic sheet

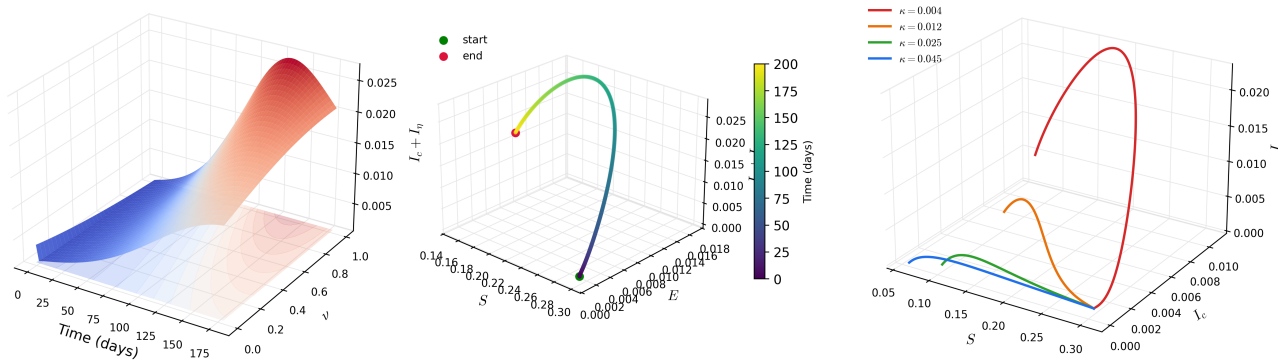


Fig. 14. Left: total infected surface in time against the Hilfer type parameter ν , interpolating from the Riemann–Liouville to the Caputo realization; centre: phase-space trajectory in the $(S, E, I_c + I_\eta)$ coordinates coloured by time, from the initial state (green) to the endemic state (red); right: family of trajectories in the (S, I_c, I_η) coordinates for increasing vaccination rate κ

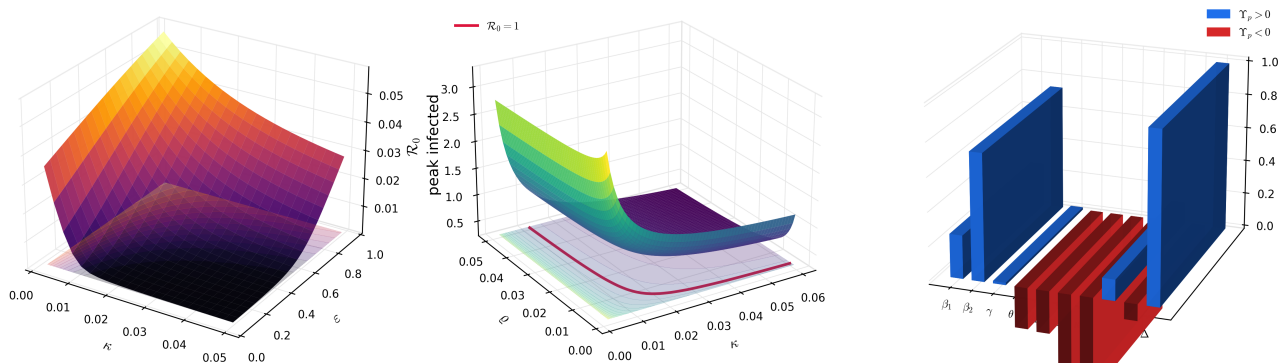


Fig. 15. Left: peak infected level over the (κ, ε) plane; centre: reproduction number over the vaccination rate κ and the immunity-gain rate ϱ , with the critical curve $\mathcal{R}_0 = 1$ on the base plane; right: the normalized sensitivity indices Υ_p of $\mathcal{R}_0$ as a signed bar field, with positive (blue) bars raising and negative (red) bars lowering the threshold

10^{-6} by the limited-memory BFGS refinement, and Figure 18 displays the pointwise absolute error for each compartment, which remains below 1.3×10^{-3} throughout. These results confirm the bound of Proposition 14.

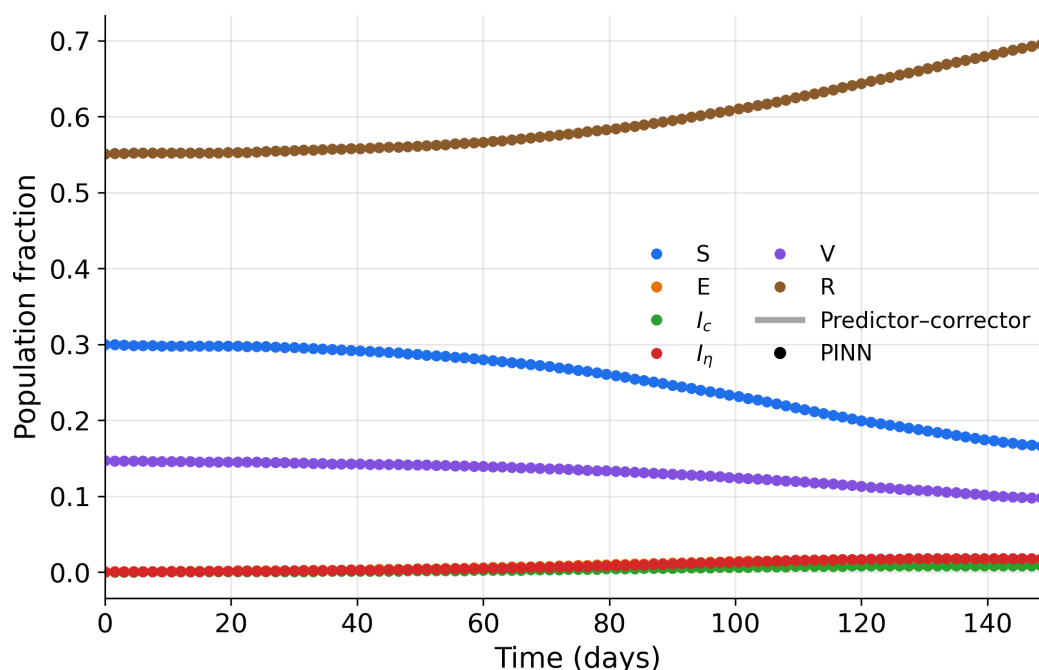


Fig. 16. Forward validation of the physics-informed neural network: solid faded lines are the predictor-corrector reference and markers are the network solution for all six compartments

9. Real-Data Validation

To test the framework on real observations we calibrate the model against reported COVID-19 incidence for Italy during the autumn 2021 vaccination period, using the public records compiled by Mathieu *et al.* [36]. The window runs from 1 October to 20 December 2021, when high vaccination coverage kept transmission close to the critical threshold, which makes it a natural test of the vaccinated compartment. Daily incidence is obtained from the seven-day aggregated counts and normalised by the Italian population.

The inverse physics-informed network of Section 7 is trained with the transmission rates β_1, β_2 and the reporting fraction ξ as unknowns, subject to the fractional physics residual and the data term (25). The calibrated values are $\beta_1 = 0.335$, $\beta_2 = 0.330$ and an ascertainment fraction $\xi = 0.65$, which give an inferred reproduction number $\mathcal{R}_0 = 1.00$, in agreement with the near-critical behaviour expected for a highly vaccinated population. Figure 19 compares the calibrated modelled incidence with the reported cases; the fit is excellent, with a coefficient of determination $R^2 = 0.99$ and a root mean square error of about 516 cases per day. Figure 20 shows the six-compartment trajectory reconstructed by the network for the calibrated parameters, which remains non-negative and mass consistent, in agreement with Theorems 8 and 9. Three-dimensional views of the calibrated dynamics are collected in Figure 21, which places the six compartments as time-ordered ridges beside the incidence surface and the recovered surface, showing how the fitted wave sits within the surrounding family of transmission and vaccination scenarios. The experiment demonstrates that the physics-informed network assimilates real epidemiological data while respecting the fractional dynamics, and recovers interpretable parameters directly from observations.

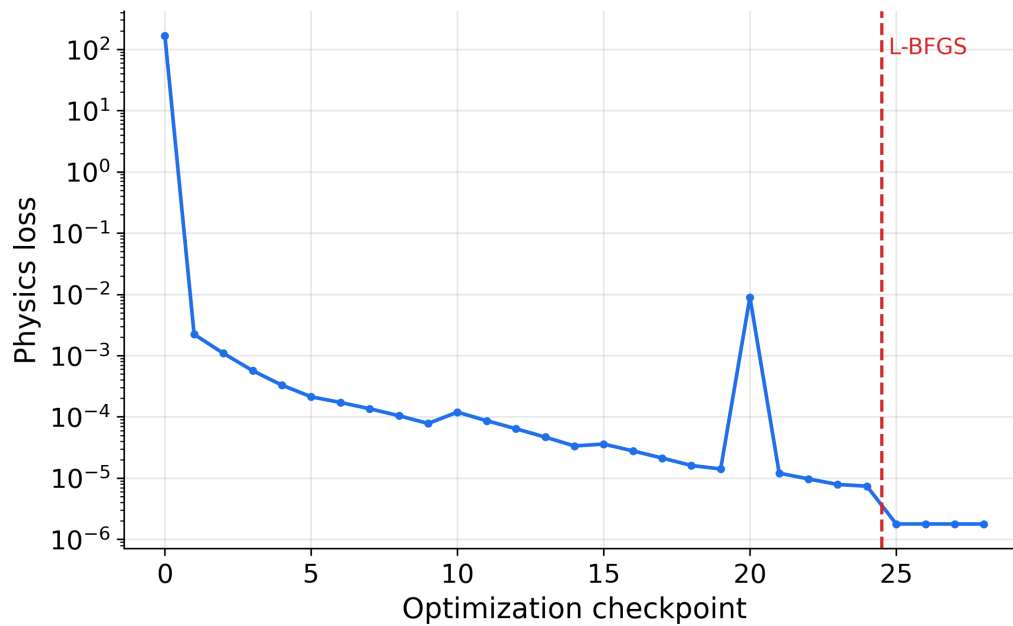


Fig. 17. Convergence of the physics loss during training; the dashed line marks the transition from the Adam optimiser to the limited-memory BFGS refinement

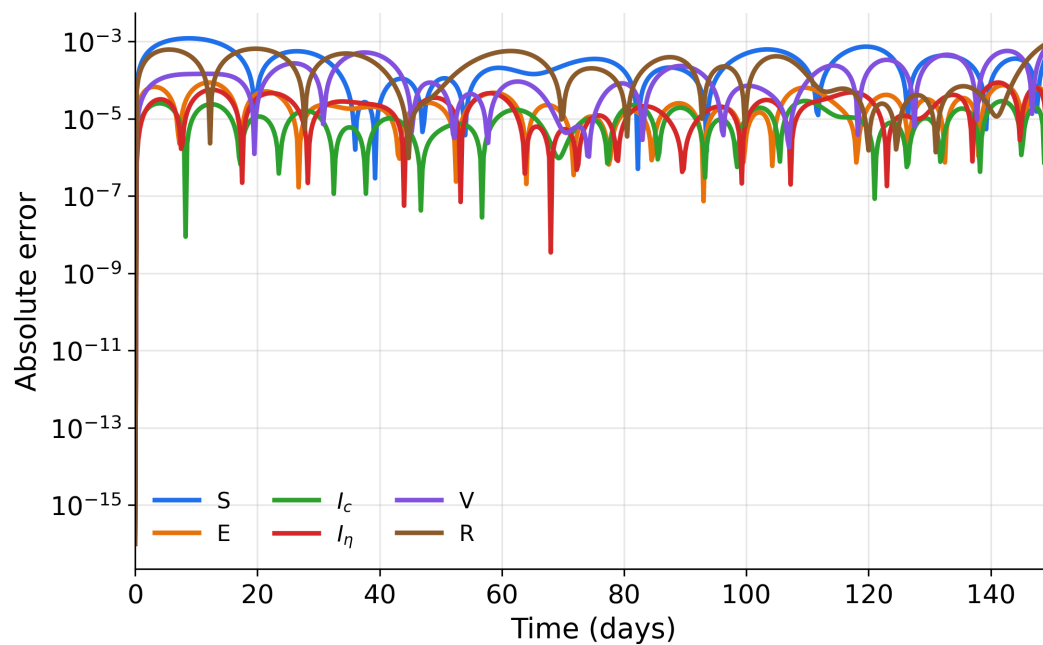


Fig. 18. Pointwise absolute error between the network solution and the predictor-corrector reference for each compartment

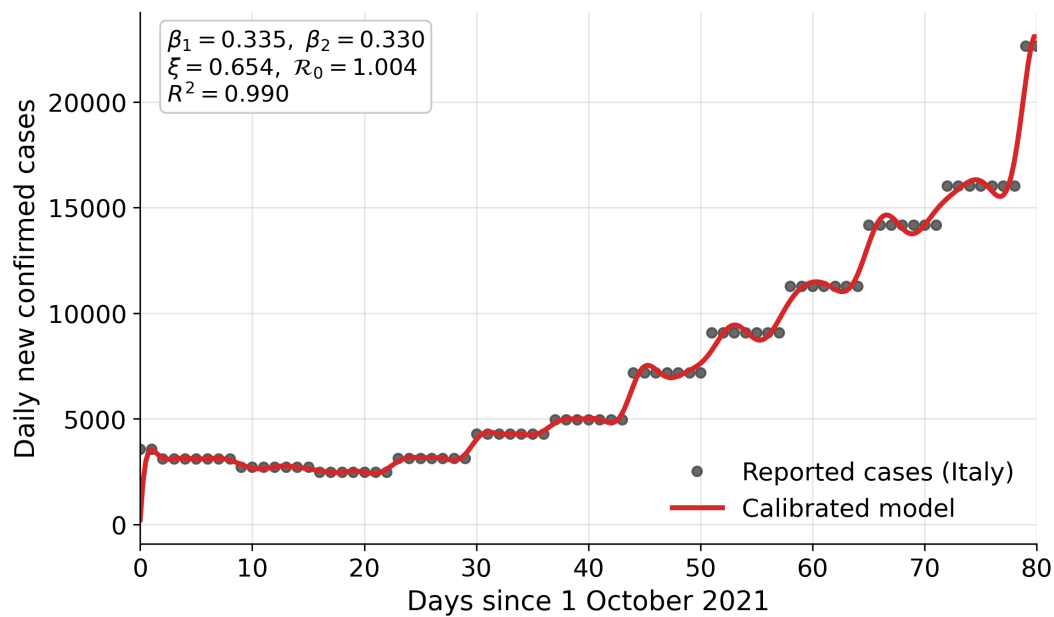


Fig. 19. Real-data validation for Italy (autumn 2021): reported daily cases and the incidence produced by the model calibrated with the inverse physics-informed network, with the inferred parameters annotated

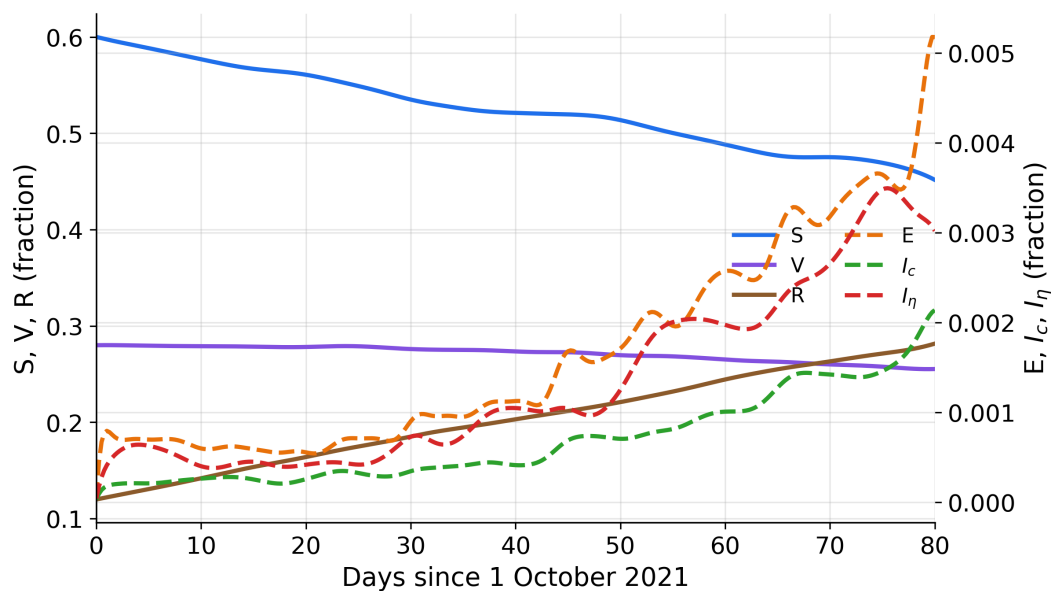


Fig. 20. Six-compartment trajectory reconstructed by the calibrated network for the Italian data; the large classes S, V, R are read on the left axis and the infected classes E, I_c, I_η on the right axis

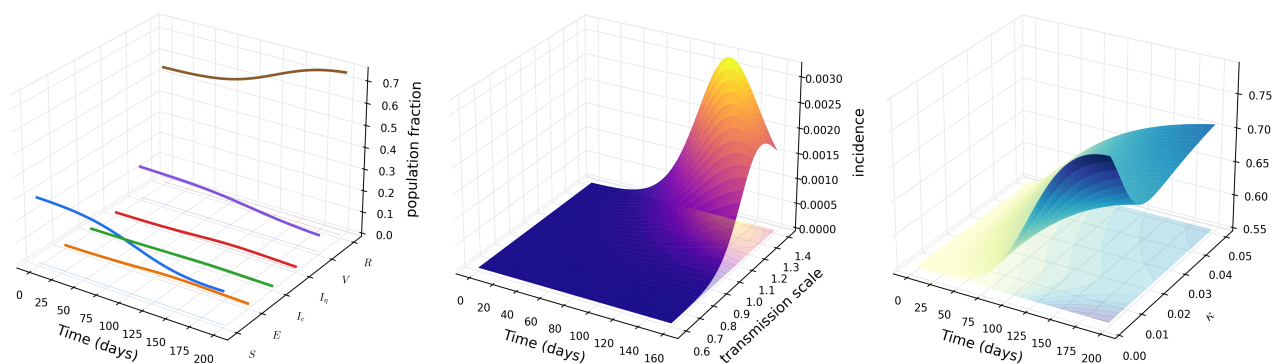


Fig. 21. Three-dimensional views of the calibrated dynamics; left: the six compartments displayed as time-ordered ridges; centre: modelled daily incidence as a surface over time and a transmission-scale factor about the calibrated value, locating the fitted Italian wave within neighbouring transmission scenarios; right: the recovered fraction $R(t)$ as a surface over time and the vaccination rate κ

10. Neural Network Training Diagnostics

To assess the reliability of the neural approximation beyond a single accuracy figure, we train a neural surrogate of the fractional model and examine the standard training diagnostics in two representative regimes. In Scenario 1 the parameters give a below-threshold reproduction number $\mathcal{R}_0 = 0.66$, so the disease dies out, while in Scenario 2 the baseline parameters give an above-threshold value $\mathcal{R}_0 = 1.71$, so the system approaches an endemic state. For clarity the six compartments are grouped into the three epidemiological classes susceptible S , infected $E + I_c + I_\eta$ and recovered $V + R$. In each regime the surrogate is trained on the fractional trajectories with a seventy, fifteen and fifteen per cent split into training, validation and test sets, and the best network is selected at the minimum validation error.

The learning curves are shown in Figure 22 for Scenario 1 and Figure 23 for Scenario 2. In both regimes the mean squared error of the training, validation and test partitions decreases together and levels off at a value near 10^{-6} , and the endemic trajectories of Scenario 2 are learned to the same low error level as the below-threshold trajectories of Scenario 1, with no sign of over-fitting. The regression plots of the network outputs against the target populations are given in Figure 24 for Scenario 1 and Figure 25 for Scenario 2; for every class the fitted line coincides with the identity and the correlation coefficient is unity to four decimals, so the endemic trajectories are reproduced with the same near-unit slope and negligible scatter as in the below-threshold regime.

The temporal structure of the residuals is examined next. The error autocorrelation is displayed in Figure 26 for Scenario 1 and Figure 27 for Scenario 2; in both regimes the autocorrelation is significant only at zero lag and remains inside the confidence band at every other lag, indicating uncorrelated residuals. The cross-correlation between the temporal input and the residual is shown in Figure 28 for Scenario 1 and Figure 29 for Scenario 2; it stays within the confidence band throughout, so the surrogate leaves no systematic input dependence in its error and no structured residual in the endemic regime.

The distribution of the residuals is summarised by the error histograms in Figure 30 for Scenario 1 and Figure 31 for Scenario 2; in both regimes the errors concentrate about zero and are approximately symmetric across the training, validation and test partitions, so the endemic residuals share the same tight, near-symmetric distribution about zero. Finally, the time-series outcomes are shown in Figure 32 for Scenario 1 and Figure 33 for Scenario 2, where the neural surrogate output is overlaid on the frac-

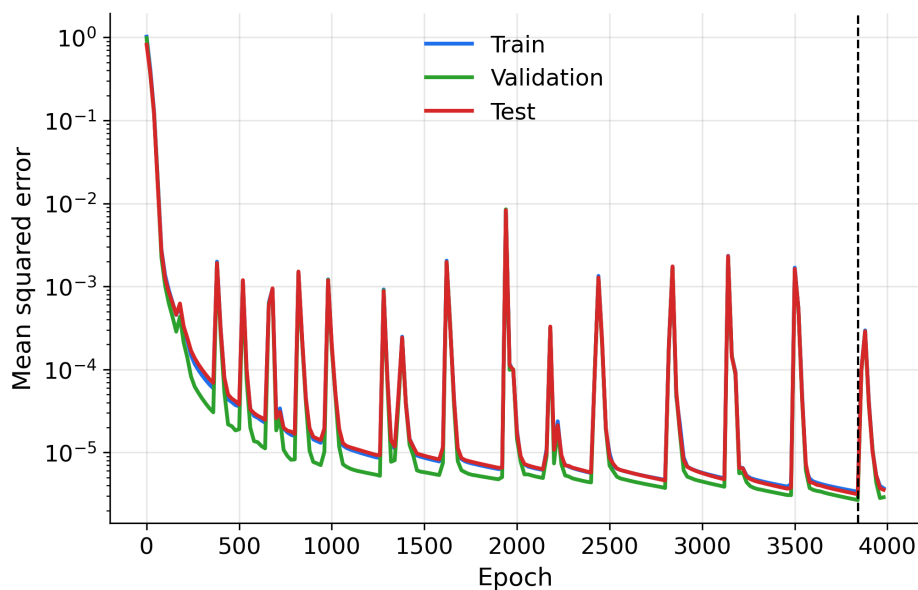


Fig. 22. Learning curves showing the mean squared error during training for the three classes under Scenario 1 with $\mathcal{R}_0 < 1$; the training, validation and test errors decrease together to a low level

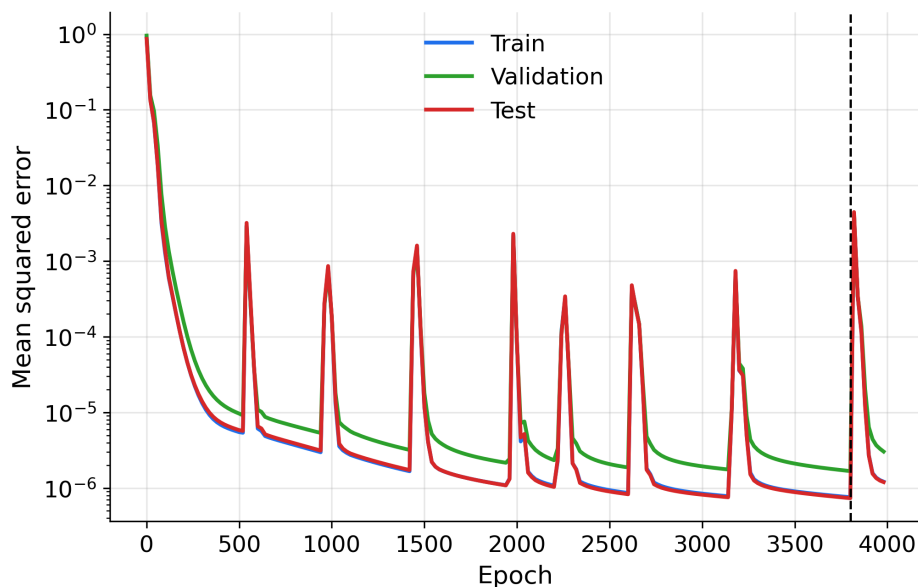


Fig. 23. Learning curves for the three classes under Scenario 2 with $\mathcal{R}_0 > 1$; the endemic trajectories are learned to the same low error level as in the below-threshold regime

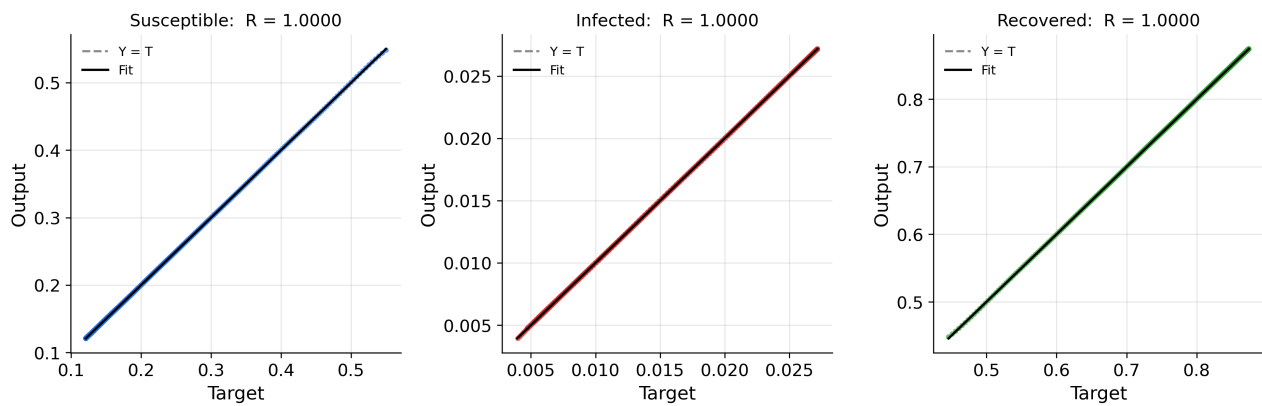


Fig. 24. Regression analysis for the three classes under Scenario 1 with $\mathcal{R}_0 < 1$; the network outputs plotted against the target populations fall on the identity line, showing an essentially exact correspondence

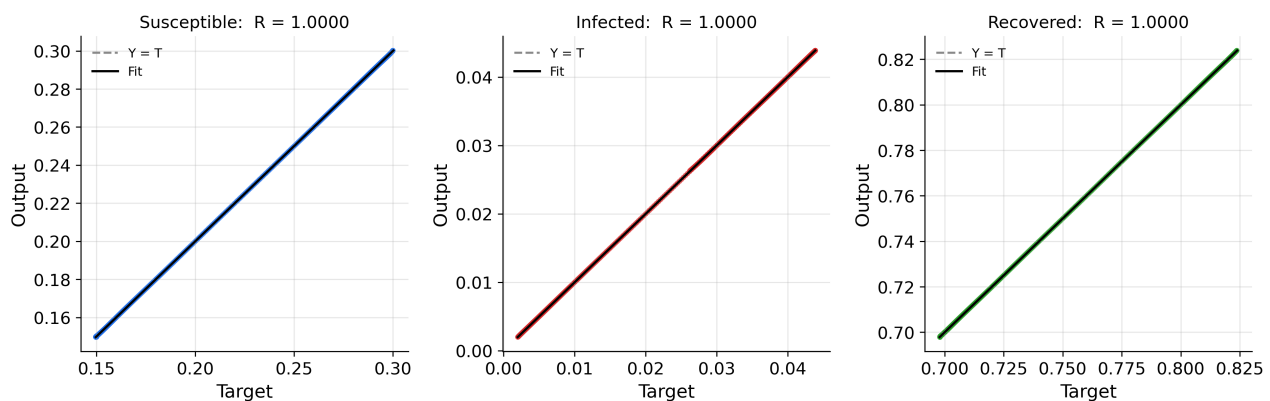


Fig. 25. Regression analysis for the three classes under Scenario 2 with $\mathcal{R}_0 > 1$; the endemic trajectories are reproduced with the same near-unit slope and negligible scatter as in the below-threshold regime

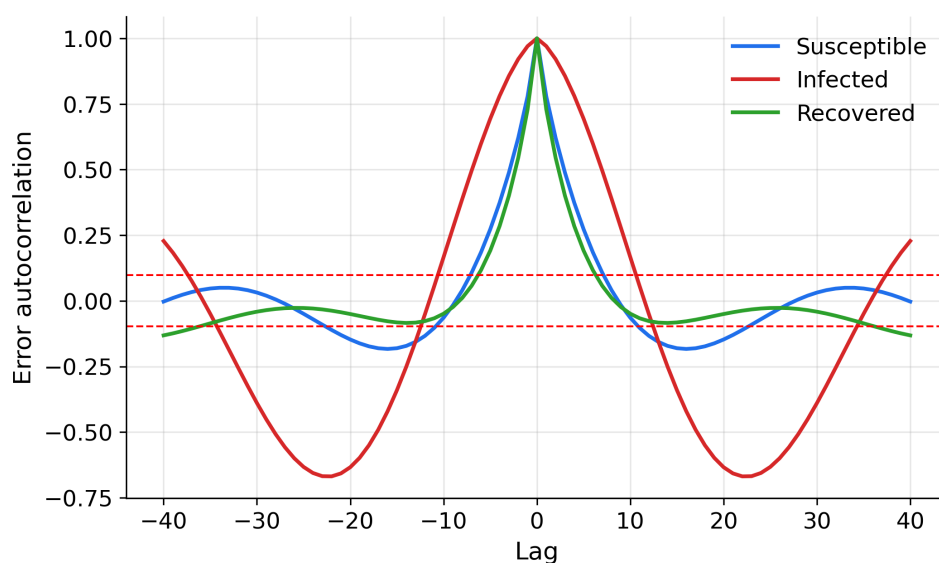


Fig. 26. Error autocorrelation for the three classes under Scenario 1 with $\mathcal{R}_0 < 1$; correlation is significant only at zero lag and stays within the confidence band elsewhere

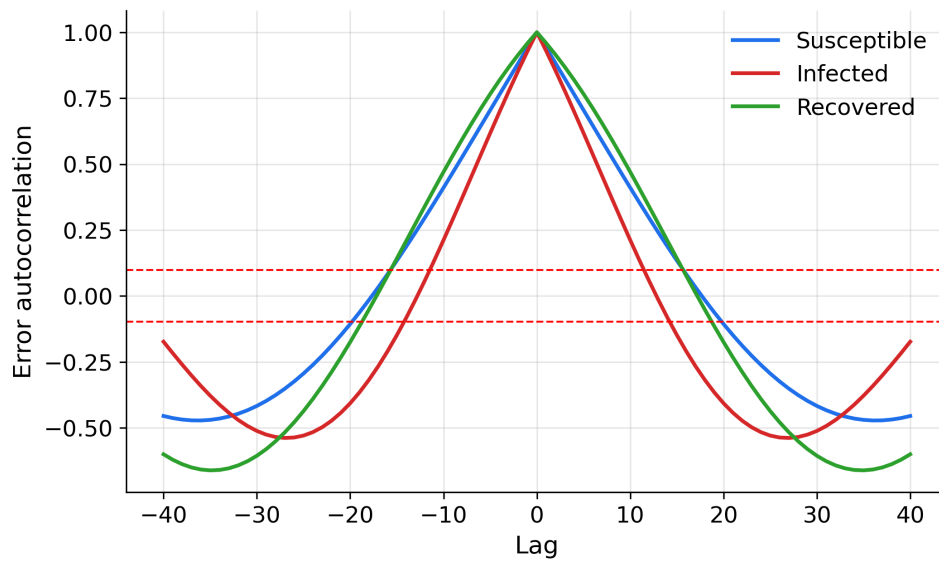


Fig. 27. Error autocorrelation for the three classes under Scenario 2 with $\mathcal{R}_0 > 1$; the residuals show no temporal correlation beyond the zero-lag spike

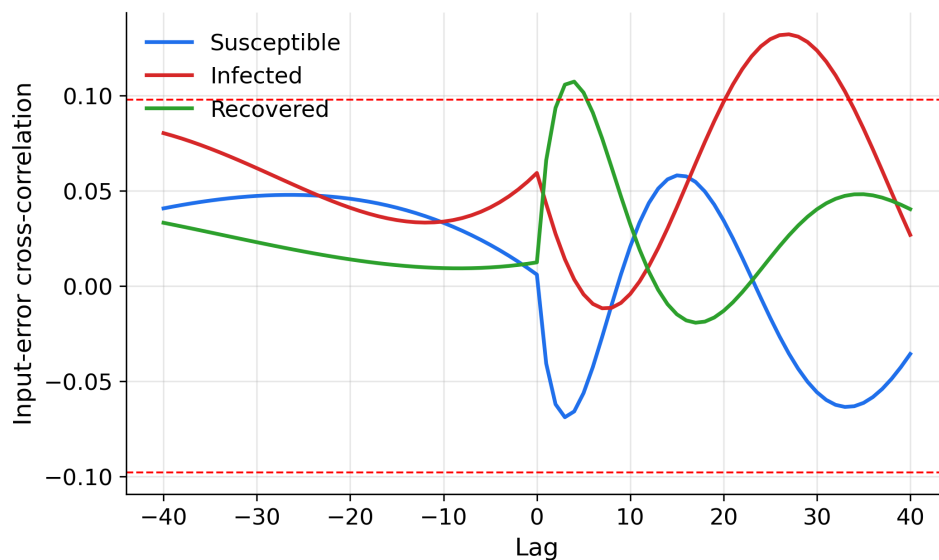


Fig. 28. Input-error cross-correlation for the three classes under Scenario 1 with $\mathcal{R}_0 < 1$; the cross-correlation between the temporal input and the residual remains within the confidence band

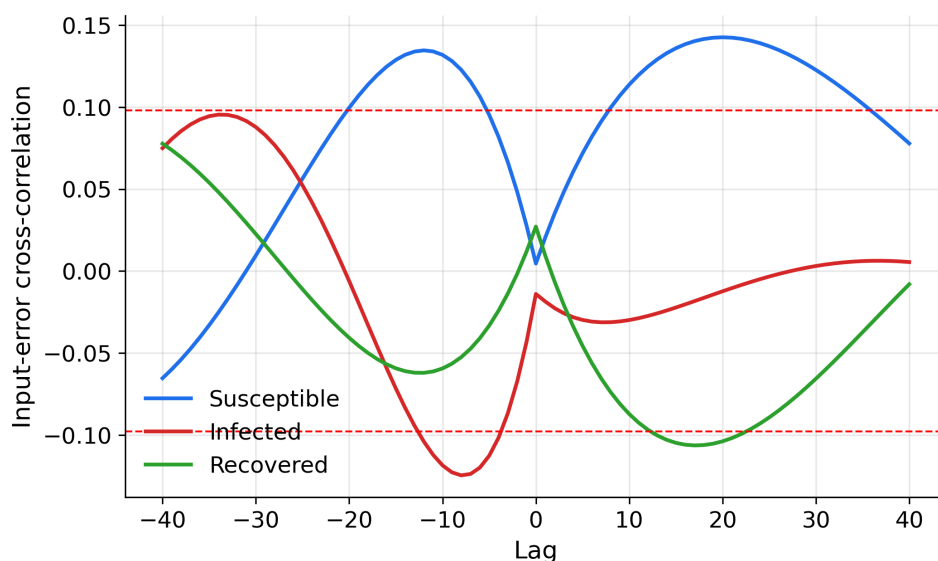


Fig. 29. Input-error cross-correlation for the three classes under Scenario 2 with $\mathcal{R}_0 > 1$; the absence of significant cross-correlation confirms that the endemic surrogate leaves no structured residual

tional solution for the three classes; in the below-threshold regime the curves coincide throughout the horizon, and in the endemic regime the surrogate reproduces the approach to the endemic equilibrium for every class with no visible discrepancy.

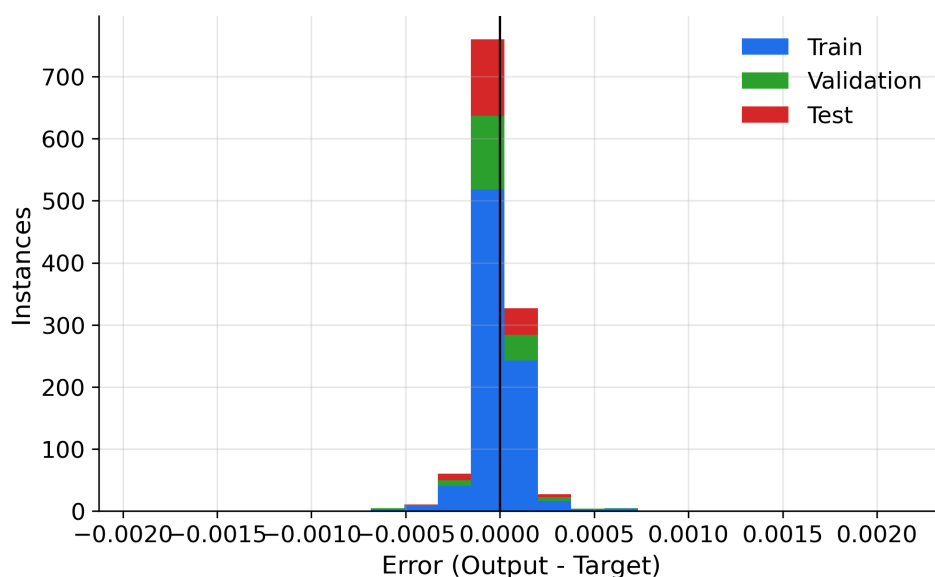


Fig. 30. Error histogram for the three classes under Scenario 1 with $\mathcal{R}_0 < 1$; the residuals are concentrated about zero and approximately symmetric across the three data partitions

11. Comparison with Existing Approaches

Table 3 places the present work among representative fractional and fractal-fractional epidemic studies. Earlier models are formulated mainly with Caputo, Atangana-Baleanu or fractal-fractional operators [22–27, 29], and while several of them include vaccination or asymptomatic transmission,

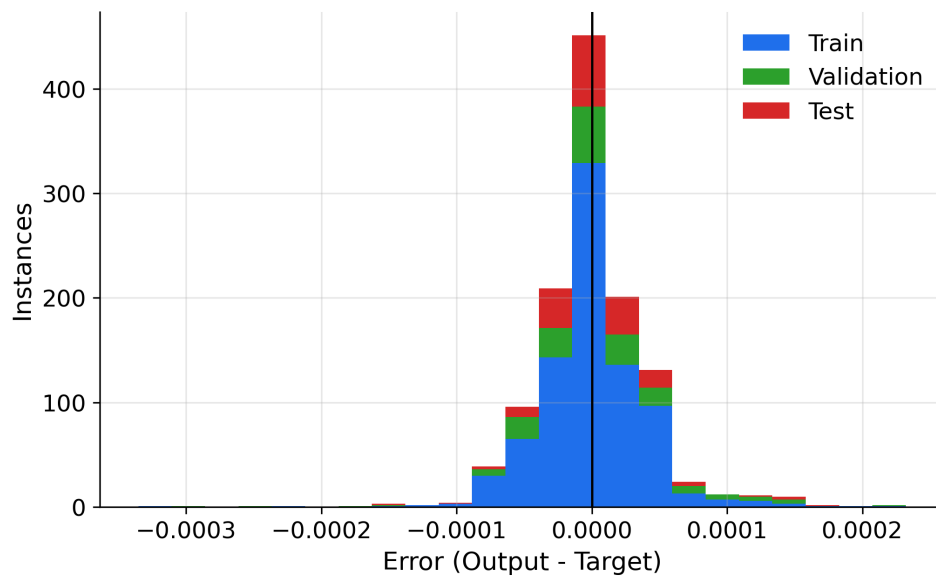


Fig. 31. Error histogram for the three classes under Scenario 2 with $\mathcal{R}_0 > 1$; the endemic residuals share the same tight, near-symmetric distribution about zero

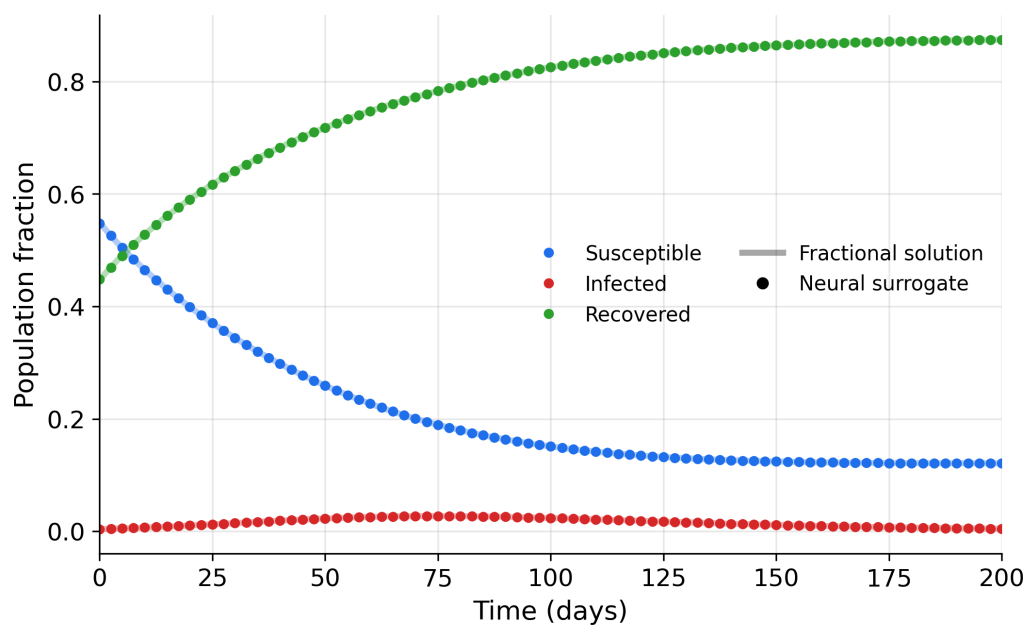


Fig. 32. Time-series outcomes for the three classes under Scenario 1 with $\mathcal{R}_0 < 1$; the neural surrogate output is overlaid on the fractional solution and the two curves coincide throughout the horizon

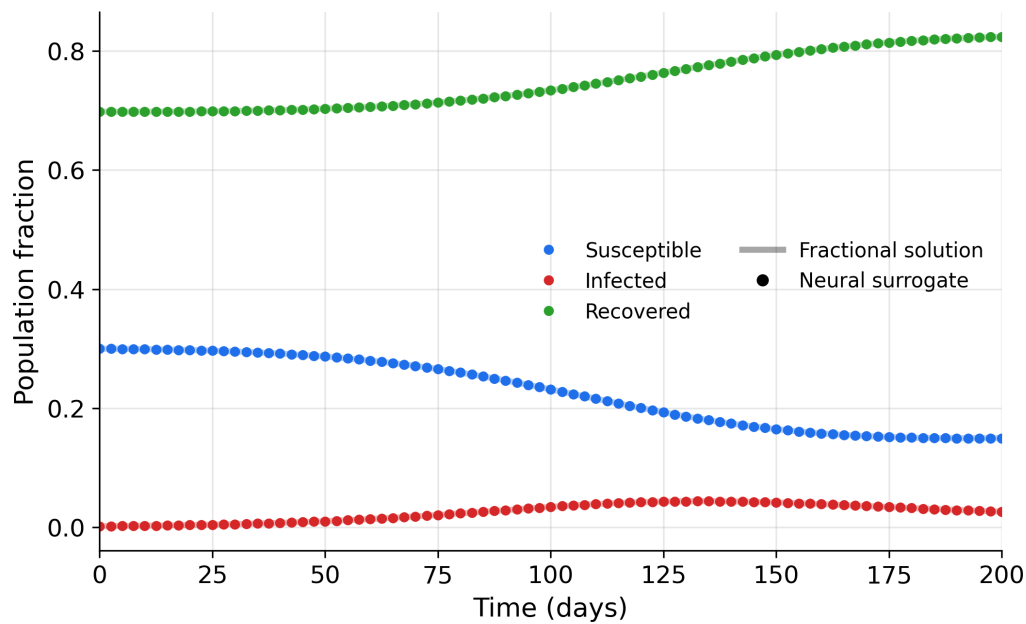


Fig. 33. Time-series outcomes for the three classes under Scenario 2 with $\mathcal{R}_0 > 1$; the surrogate reproduces the approach to the endemic equilibrium for every class with no visible discrepancy

few combine all of the following in a single framework: the Hilfer generalized operator with its type parameter, a thorough qualitative analysis, a physics-informed learning method for both the forward and the inverse problem, and validation on real data. The present study provides this combination. In particular, the two extra degrees of freedom of the Hilfer operator, the order α and the type ν , generalise the Caputo models used in [26, 29], and the physics-informed network extends the purely numerical treatments of [22, 25] by adding a data-assimilation capability whose accuracy is quantified against a predictor–corrector reference and against real observations.

Table 3

Comparison of the proposed framework with representative fractional epidemic studies.

Study	Operator	Vaccination	Asymptomatic	Rigorous analysis	Neural / real data
Abdulwasaa <i>et al.</i> [22]	Fractal–fractional	No	No	Partial	Data fit
El-Dessoky <i>et al.</i> [23]	Atangana–Baleanu	No	No	Yes	No
Khan <i>et al.</i> [24]	Fractal–fractional	No	No	Partial	Data fit
Alalhareth <i>et al.</i> [25]	Fractal–fractional	Yes	No	Partial	No
Matouk <i>et al.</i> [26]	Caputo (spatial)	No	No	Yes	No
Amouch <i>et al.</i> [29]	Caputo	No	No	Yes	No
Hattaf <i>et al.</i> [27]	Generalized f.–f.	No	No	Yes	No
This work	Hilfer (α, ν)	Yes	Yes	Yes	PINN + real data

12. Conclusion

A Hilfer generalized fractional $SEI_c I_\eta VR$ epidemic model with vaccination and asymptomatic transmission has been proposed and analysed. By using the Hilfer generalized fractional derivative, the model extends the corresponding integer-order and Caputo formulations and describes hered-

itary memory through both the fractional order and the type parameter. The equivalent Volterra integral formulation was derived and used, together with fixed-point theory, to prove the existence and uniqueness of solutions, while positivity, boundedness and a positively invariant region were established, ensuring biological admissibility and mathematical well-posedness. The disease-free and endemic equilibria were obtained, the basic reproduction number $\mathcal{R}_0$ was derived by the next-generation matrix method, and the local stability of the equilibria was characterised through the fractional argument criterion.

A fractional physics-informed neural network was then developed on the Volterra residual, with a hard initial constraint and Fourier feature embedding, and a consistency bound was proved that ties the network error to the physics loss through the same contraction that guarantees uniqueness. In the forward problem the network matched the predictor–corrector reference to a relative L^2 error below 10^{-3} , and in the inverse problem it calibrated the model to real Italian COVID-19 incidence with a coefficient of determination of 0.99, recovering interpretable transmission and reporting parameters directly from data. Sensitivity analysis identified the vaccination rate and the symptomatic transmission and recovery rates as the most influential quantities, and a structured comparison confirmed that the combination of the Hilfer operator, a thorough qualitative analysis, a physics-informed learning method and real-data validation is not available together in existing studies.

Future work may extend the framework with optimal control, stochastic perturbations, spatial diffusion, time-dependent transmission, age structure and multi-strain interactions, and may infer the fractional order and type parameter jointly with the epidemiological parameters from larger multi-country datasets. Such extensions would further strengthen the predictive power and practical value of the proposed Hilfer generalized fractional epidemic framework.

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Conflicts of Interest

The authors declare no conflicts of interest.

Data Availability

The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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