

A Two-Hidden-Layer Radial Basis Neural Surrogate for a Caputo Fractional-Order Schistosomiasis Model

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Abstract

A reproducible numerical framework is developed for a five-compartment Caputo fractional-order schistosomiasis model coupling susceptible, infected, and recovered humans with susceptible and infected snail populations. The compartmental equations are written in a population-balanced form, and their fundamental properties are established: nonnegativity of solutions, explicit bounds for the total human and snail populations, the disease-free equilibrium, and a threshold reproduction number. Reference trajectories are computed by the fractional Adams–Bashforth–Moulton predictor–corrector method. A two-hidden-layer radial basis function network, with 15 and 25 radial units, is then trained as a supervised trajectory surrogate. The output parameters are refined by the scaled conjugate-gradient algorithm, while a fixed 70–15–15 split is used for training, validation, and testing. Three Caputo orders, $\alpha = 0.5, 0.7$, and 0.9 , are studied on $0 \leq t \leq 10$. Across the held-out test sets, the root mean squared errors are 2.406×10^{-4} , 2.122×10^{-4} , and 1.793×10^{-4} , respectively, with coefficients of determination above 0.99996. The results show that the surrogate reproduces the reference trajectories accurately and inexpensively after training. The distinction between the predictor–corrector solver and the neural surrogate is maintained throughout: the network approximates validated numerical trajectories and is not presented as an independent replacement for the fractional differential equations.

Keywords: Caputo derivative; schistosomiasis; fractional Adams method; radial basis function network; scaled conjugate gradient; neural surrogate.

1 Introduction

Schistosomiasis is an acute and chronic parasitic disease caused by blood flukes of the genus *Schistosoma*. The transmission cycle couples human hosts, freshwater snails, and environmental exposure to cercariae. The World Health Organization estimated that at least 253.7 million people required preventive treatment in 2024 and reported transmission in 79 countries [1]. This burden motivates mathematical models capable of examining human–snail transmission, treatment, loss of immunity, and long-term control [2, 3, 4, 5].

Classical compartmental models are usually formulated with ordinary derivatives. Fractional derivatives provide a nonlocal alternative in which the current rate of change depends on the

history of the state. In epidemic models, this memory may represent heterogeneous waiting times, accumulated exposure, delayed behavioral response, or unresolved temporal structure. The Caputo derivative is especially convenient because it admits standard initial conditions and reduces continuously to the first derivative when $\alpha \rightarrow 1$ [6, 7, 8].

A five-state fractional schistosomiasis model and a neural approximation were previously considered by Botmart et al. [9]. That study established an important starting point but left several issues that must be resolved for a reproducible numerical investigation: the printed equations contain inconsistent transfer terms, the reference Adams method is not fully specified, and the neural approximation is sometimes described as though it were an independent differential-equation solver. In addition, a scientifically useful study should report initial conditions, integration interval, step size, data partition, normalization, error definitions, random seed, and executable code.

The present work reconstructs the model and numerical procedure in a population-balanced and reproducible form. The specific contributions are:

- (i) a coherent five-compartment Caputo model with explicit human and snail population balances;
- (ii) proofs of positivity and boundedness, together with the disease-free equilibrium and the threshold number $\mathcal{R}_0$;
- (iii) a complete fractional Adams–Bashforth–Moulton predictor–corrector implementation and a step-refinement check;
- (iv) a two-hidden-layer radial basis function (RBF) surrogate with 15 and 25 hidden units, trained by scaled conjugate gradients (SCG);
- (v) transparent training, validation, and test metrics for $\alpha \in \{0.5, 0.7, 0.9\}$; and
- (vi) a supplementary package containing the L^AT_EX source, figures, data, and Python code needed to regenerate the results.

The principal methodological distinction is that the Adams method generates the reference solution, whereas the neural network learns a compact surrogate of that solution. This terminology avoids attributing the discretization of the Caputo operator to a network trained only on trajectory data.

2 Fractional schistosomiasis model

2.1 Caputo derivative and integral representation

Definition 1. *For an absolutely continuous scalar function x and $0 < \alpha < 1$, the Caputo derivative is*

$${}^C D_t^\alpha x(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-s)^{-\alpha} x'(s) ds. \quad (1)$$

For $\alpha = 1$, it coincides with $x'(t)$.

The initial-value problem

$${}^C D_t^\alpha \mathbf{x}(t) = \mathbf{f}(t, \mathbf{x}(t)), \quad \mathbf{x}(0) = \mathbf{x}_0, \quad (2)$$

is equivalent to the weakly singular Volterra equation

$$\mathbf{x}(t) = \mathbf{x}_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} \mathbf{f}(s, \mathbf{x}(s)) ds. \quad (3)$$

This representation is the basis of the fractional Adams method used below.

2.2 Compartment definitions and equations

Let $W(t)$, $A(t)$, and $Q(t)$ denote susceptible, infected, and recovered humans, respectively. Let $E(t)$ and $S(t)$ denote susceptible and infected snails. The state vector is

$$\mathbf{x}(t) = (W(t), A(t), Q(t), E(t), S(t))^T.$$

The transmission structure is shown in figure 1.

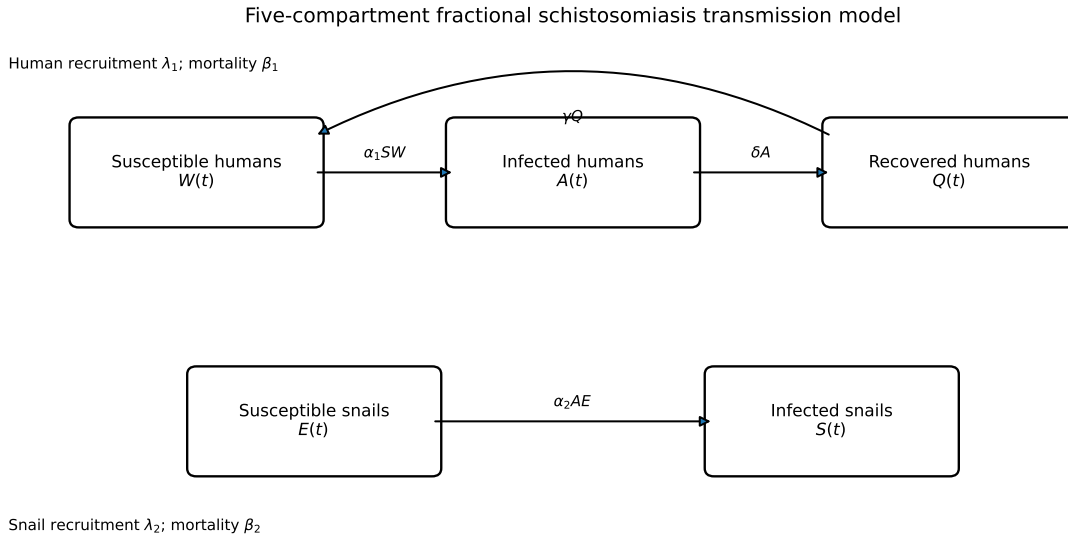


Figure 1: Five-compartment human–snail transmission structure. The infection flow from W to A is modulated by infected snails S , while the infection flow from E to S is modulated by infected humans A .

The model is

$${}^C D_t^\alpha W = \lambda_1 - \alpha_1 SW + \gamma Q - \beta_1 W, \quad (4a)$$

$${}^C D_t^\alpha A = \alpha_1 SW - (\beta_1 + \delta) A, \quad (4b)$$

$${}^C D_t^\alpha Q = \delta A - (\gamma + \beta_1) Q, \quad (4c)$$

$${}^C D_t^\alpha E = \lambda_2 - \alpha_2 AE - \beta_2 E, \quad (4d)$$

$${}^C D_t^\alpha S = \alpha_2 AE - \beta_2 S. \quad (4e)$$

The recovery inflow in equation (4c) is δA , and the snail mortality rate in both snail equations is β_2 . These choices are required by the compartmental flow interpretation and make the total populations satisfy exact balance equations. They correct typographical inconsistencies in the printed system of Ref. [9].

Table 1: Model parameters and values used in the numerical experiments.

Symbol	Value	Interpretation	Units
λ_1	0.10	Human recruitment rate	population/time $^\alpha$
α_1	0.12	Snail-to-human transmission coefficient	population $^{-1}$ /time $^\alpha$
β_1	0.15	Human natural mortality/removal rate	time $^{-\alpha}$
δ	0.20	Human recovery rate	time $^{-\alpha}$
γ	0.23	Rate of immunity loss	time $^{-\alpha}$
λ_2	0.23	Snail recruitment rate	population/time $^\alpha$
α_2	0.30	Human-to-snail transmission coefficient	population $^{-1}$ /time $^\alpha$
β_2	0.18	Snail mortality rate	time $^{-\alpha}$

The initial condition is

$$(W(0), A(0), Q(0), E(0), S(0)) = (0.1, 0.2, 0.3, 0.4, 0.5). \quad (5)$$

The parameter values are nondimensional benchmark values inherited from the modeling scale used in Ref. [9]; they are not asserted to be a country-specific epidemiological calibration.

3 Qualitative properties

3.1 Positivity and boundedness

Theorem 1 (Positivity). *Let $0 < \alpha \leq 1$ and let the initial data in equation (5) be nonnegative. Then every solution of equation (4) that exists on $[0, T]$ remains in the nonnegative orthant.*

Proof. The vector field is quasi-positive. On the coordinate hyperplanes,

$$\begin{aligned} {}^C D_t^\alpha W \Big|_{W=0} &= \lambda_1 + \gamma Q \geq 0, \\ {}^C D_t^\alpha A \Big|_{A=0} &= \alpha_1 S W \geq 0, \\ {}^C D_t^\alpha Q \Big|_{Q=0} &= \delta A \geq 0, \\ {}^C D_t^\alpha E \Big|_{E=0} &= \lambda_2 \geq 0, \\ {}^C D_t^\alpha S \Big|_{S=0} &= \alpha_2 A E \geq 0. \end{aligned}$$

Hence no component can cross from zero into negative values; the standard fractional comparison principle gives invariance of $\mathbb{R}_+^5$. $\square$

Define the total human and snail populations

$$N_h = W + A + Q, \quad N_s = E + S.$$

Adding the corresponding equations gives

$${}^C D_t^\alpha N_h = \lambda_1 - \beta_1 N_h, \quad {}^C D_t^\alpha N_s = \lambda_2 - \beta_2 N_s. \quad (6)$$

Thus

$$N_h(t) = N_h(0)E_\alpha(-\beta_1 t^\alpha) + \frac{\lambda_1}{\beta_1} [1 - E_\alpha(-\beta_1 t^\alpha)], \quad (7)$$

$$N_s(t) = N_s(0)E_\alpha(-\beta_2 t^\alpha) + \frac{\lambda_2}{\beta_2} [1 - E_\alpha(-\beta_2 t^\alpha)], \quad (8)$$

where E_α is the one-parameter Mittag-Leffler function.

Theorem 2 (Bounded feasible region). *The set*

$$\Omega = \left\{ \mathbf{x} \in \mathbb{R}_+^5 : N_h \leq \max\left(N_h(0), \frac{\lambda_1}{\beta_1}\right), \quad N_s \leq \max\left(N_s(0), \frac{\lambda_2}{\beta_2}\right) \right\} \quad (9)$$

is positively invariant.

Proof. For $0 < \alpha \leq 1$ and $z \geq 0$, $0 < E_\alpha(-z) \leq 1$. Equations (7)–(8) therefore express each total as a convex combination of its initial value and its equilibrium level. Combined with positivity, this proves the result. $\square$

3.2 Disease-free equilibrium and reproduction number

The disease-free equilibrium (DFE) is

$$\mathcal{E}_0 = \left(\frac{\lambda_1}{\beta_1}, 0, 0, \frac{\lambda_2}{\beta_2}, 0 \right). \quad (10)$$

Following the next-generation construction [10], the infected variables are (A, S) . At the DFE, the new-infection and transition matrices are

$$F = \begin{pmatrix} 0 & \alpha_1 W_0 \\ \alpha_2 E_0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \beta_1 + \delta & 0 \\ 0 & \beta_2 \end{pmatrix}, \quad (11)$$

where $W_0 = \lambda_1/\beta_1$ and $E_0 = \lambda_2/\beta_2$. Therefore

$$\mathcal{R}_0 = \rho(FV^{-1}) = \sqrt{\frac{\alpha_1 \alpha_2 W_0 E_0}{(\beta_1 + \delta) \beta_2}}. \quad (12)$$

For the values in table 1,

$$\mathcal{R}_0 = 0.69769082 < 1. \quad (13)$$

Proposition 1 (Local stability of the DFE). *If $\mathcal{R}_0 < 1$, the DFE is locally asymptotically stable for every $0 < \alpha \leq 1$. If $\mathcal{R}_0 > 1$, it is unstable.*

Proof. The infected block of the Jacobian is

$$J_I = \begin{pmatrix} -(\beta_1 + \delta) & \alpha_1 W_0 \\ \alpha_2 E_0 & -\beta_2 \end{pmatrix}.$$

Its trace is negative, while

$$\det J_I = (\beta_1 + \delta) \beta_2 (1 - \mathcal{R}_0^2).$$

Thus, for $\mathcal{R}_0 < 1$, both infected eigenvalues are negative. The remaining linear modes have negative real eigenvalues associated with population relaxation and immunity loss. Negative

real eigenvalues satisfy Matignon's criterion $|\arg(\lambda)| > \alpha\pi/2$ for $0 < \alpha \leq 1$ [11]. If $\mathcal{R}_0 > 1$, the infected block has one positive eigenvalue, yielding instability. $\square$

4 Reference solver: fractional Adams predictor–corrector

Let $t_n = nh$, $n = 0, \dots, N$. For equation (2), the Diethelm–Ford–Freed PECE method [12, 13] uses the predictor

$$\mathbf{x}_{n+1}^P = \mathbf{x}_0 + \frac{h^\alpha}{\Gamma(\alpha + 1)} \sum_{j=0}^n b_{j,n+1} \mathbf{f}(t_j, \mathbf{x}_j), \quad (14)$$

where

$$b_{j,n+1} = (n + 1 - j)^\alpha - (n - j)^\alpha, \quad (15)$$

and the corrector

$$\mathbf{x}_{n+1} = \mathbf{x}_0 + \frac{h^\alpha}{\Gamma(\alpha + 2)} \left[\mathbf{f}(t_{n+1}, \mathbf{x}_{n+1}^P) + \sum_{j=0}^n a_{j,n+1} \mathbf{f}(t_j, \mathbf{x}_j) \right]. \quad (16)$$

The weights are

$$a_{j,n+1} = \begin{cases} n^{\alpha+1} - (n - \alpha)(n + 1)^\alpha, & j = 0, \\ (n - j + 2)^{\alpha+1} - 2(n - j + 1)^{\alpha+1} + (n - j)^{\alpha+1}, & 1 \leq j \leq n. \end{cases} \quad (17)$$

For $n = 0$, the first weight is interpreted as $a_{0,1} = \alpha$.

The computations use $0 \leq t \leq 10$, $h = 0.02$, and one corrector evaluation per step. The direct history summation is $O(N^2)$, which is acceptable for the 501-point benchmark and has the advantage of transparent reproducibility.

4.1 Step-refinement verification

A refinement study for $\alpha = 0.9$ compares solutions obtained with $h \in \{0.08, 0.04, 0.02\}$ against the solution with $h = 0.01$ at common grid points. The maximum componentwise discrepancies are reported in table 2 and displayed in figure 2. The monotone reduction confirms that the chosen reference grid is within the expected convergent regime.

Table 2: ABM step-refinement results for $\alpha = 0.9$.

Step size h	ℓ_∞ difference from the $h = 0.01$ solution
0.080	1.462×10^{-5}
0.040	3.679×10^{-6}
0.020	7.766×10^{-7}

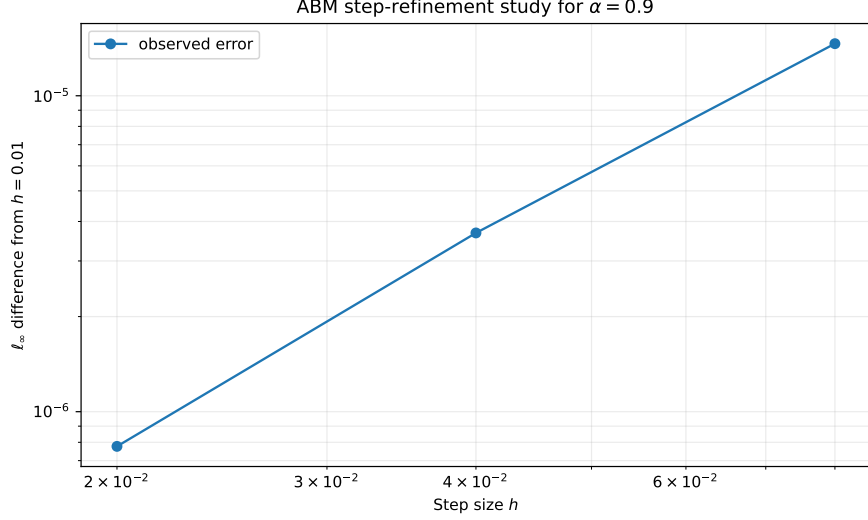


Figure 2: Step-refinement behavior of the fractional Adams predictor-corrector method for $\alpha = 0.9$.

5 Two-hidden-layer RBF neural surrogate

RBF networks are universal approximators and are particularly effective for smooth low-dimensional mappings [14, 15, 16]. Here, time is the scalar input and the five compartments are the outputs. The normalized input is

$$\xi = \frac{t - t_0}{T - t_0} \in [0, 1]. \quad (18)$$

5.1 First and second radial layers

The first hidden layer contains $m_1 = 15$ Gaussian units,

$$h_i^{(1)}(\xi) = \exp \left[-\frac{(\xi - c_i^{(1)})^2}{2(\sigma_1)^2} \right], \quad i = 1, \dots, m_1. \quad (19)$$

The centers $c_i^{(1)}$ are equally spaced across the training input range, and σ_1 equals 1.35 times the center spacing.

Let $\mathbf{h}^{(1)}(\xi) \in \mathbb{R}^{m_1}$. The second layer contains $m_2 = 25$ radial units,

$$h_j^{(2)}(\xi) = \exp \left[-\frac{\|\mathbf{h}^{(1)}(\xi) - \mathbf{c}_j^{(2)}\|_2^2}{2\sigma_2^2} \right], \quad j = 1, \dots, m_2. \quad (20)$$

The second-layer centers are sampled uniformly along the ordered training embeddings, and σ_2 is 1.8 times the median distance between adjacent centers. The five-output prediction is

$$\hat{\mathbf{x}}(t) = \boldsymbol{\mu}_x + \text{diag}(\mathbf{s}_x) \left(W^\top \begin{bmatrix} \mathbf{h}^{(2)}(\xi) \\ 1 \end{bmatrix} \right), \quad (21)$$

where $\boldsymbol{\mu}_x$ and $\mathbf{s}_x$ are the mean and standard deviation of the training targets.

5.2 Scaled conjugate-gradient training

The output matrix $W \in \mathbb{R}^{26 \times 5}$ minimizes

$$\mathcal{L}(W) = \frac{1}{2N_{\text{tr}}} \sum_{n \in \mathcal{I}_{\text{tr}}} \|W^\top \tilde{\mathbf{h}}_n - \mathbf{z}_n\|_2^2 + \frac{\eta}{2} \|W_{1:25,:}\|_F^2, \quad (22)$$

where $\mathbf{z}_n$ are standardized targets, $\tilde{\mathbf{h}}_n = [(\mathbf{h}_n^{(2)})^\top, 1]^\top$, and $\eta = 10^{-10}$. The SCG algorithm of Møller [17] avoids an explicit line search by using a finite-difference estimate of the Hessian along each conjugate direction. A lightly perturbed ridge-regression solution is used as a stable warm start, after which SCG refines the same regularized objective. This design keeps the experiment deterministic while retaining the requested SCG training mechanism.

Remark 1. *Because the hidden centers and widths are fixed, equation (22) is convex in the output weights. The network is therefore a supervised surrogate of the ABM trajectories. It does not approximate the Caputo derivative inside the loss and should not be called a physics-informed or residual-based solver.*

6 Experimental protocol and error measures

For each $\alpha \in \{0.5, 0.7, 0.9\}$, the ABM method produces 501 samples on $[0, 10]$. A pseudorandom permutation generated with seed 20260716 assigns 70% of the points to training, 15% to validation, and 15% to testing. The same indices are used for all five outputs. Since points are sampled throughout the interval, these results measure held-out interpolation rather than strict future-time extrapolation.

For reference values $x_{n,k}$ and predictions $\hat{x}_{n,k}$, the reported measures are

$$\text{MAE} = \frac{1}{5N} \sum_{k=1}^5 \sum_{n=1}^N |\hat{x}_{n,k} - x_{n,k}|, \quad (23)$$

$$\text{RMSE} = \left[\frac{1}{5N} \sum_{k=1}^5 \sum_{n=1}^N (\hat{x}_{n,k} - x_{n,k})^2 \right]^{1/2}, \quad (24)$$

$$E_\infty = \max_{n,k} |\hat{x}_{n,k} - x_{n,k}|, \quad (25)$$

$$\text{NRMSE} = \frac{1}{5} \sum_{k=1}^5 \frac{[N^{-1} \sum_n (\hat{x}_{n,k} - x_{n,k})^2]^{1/2}}{\max_n x_{n,k} - \min_n x_{n,k}}, \quad (26)$$

$$R^2 = 1 - \frac{\sum_{n,k} (\hat{x}_{n,k} - x_{n,k})^2}{\sum_{n,k} (x_{n,k} - \bar{x}_k)^2}. \quad (27)$$

All calculations were performed in Python using NumPy, SciPy, pandas, and Matplotlib. The supplied script regenerates every CSV table and every figure.

7 Results

7.1 Reference dynamics and influence of the fractional order

The reference trajectories for the three fractional orders are compared in figure 3. All compartments remain nonnegative. Since $\mathcal{R}_0 < 1$, infected humans and infected snails decrease, while the

susceptible populations relax toward the disease-free levels. Smaller fractional order produces a stronger memory effect and slower long-time relaxation.

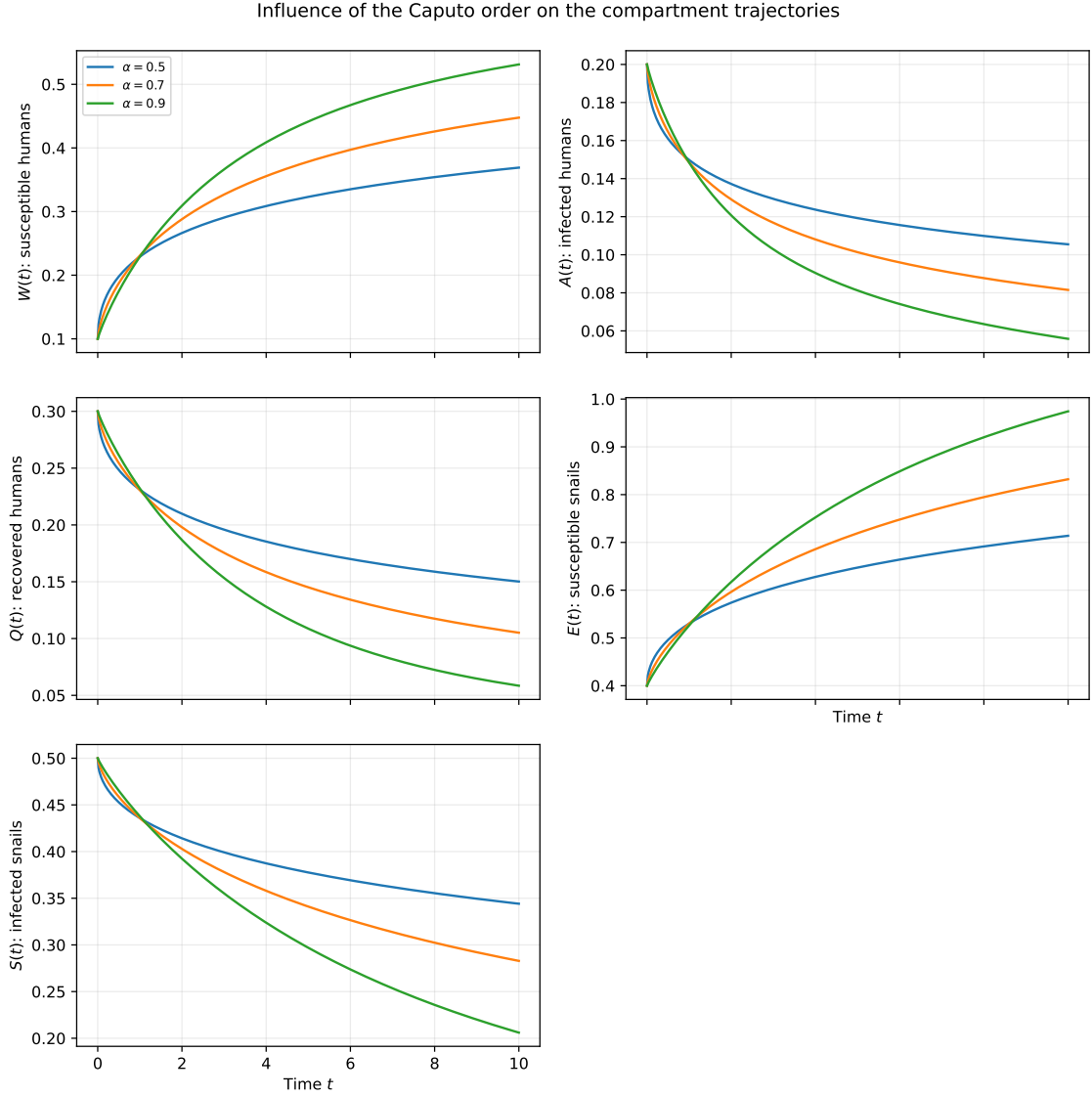


Figure 3: Effect of the Caputo order on the five reference trajectories.

The total populations in figure 4 approach $\lambda_1/\beta_1 = 2/3$ and $\lambda_2/\beta_2 \approx 1.2778$, in agreement with equations (7) and (8). This numerical balance is also a useful diagnostic: an implementation with the incorrect recovery or mortality terms would not reproduce these identities.

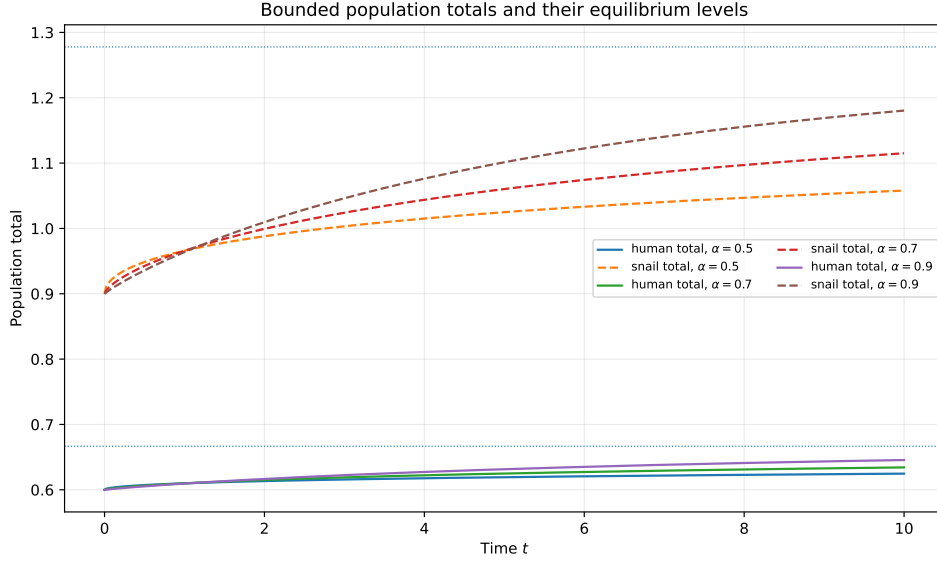


Figure 4: Human and snail population totals for the three fractional orders, together with their equilibrium levels.

7.2 Reference-surrogate agreement

Figures 5 to 7 compare the ABM trajectories with the RBF-SCG predictions. The curves are visually indistinguishable over most of the interval. The largest discrepancies occur close to the initial point, where fractional solutions exhibit the strongest curvature and where a random validation subset may contain only sparse neighboring training points.

Reference and neural-surrogate trajectories, $\alpha = 0.5$

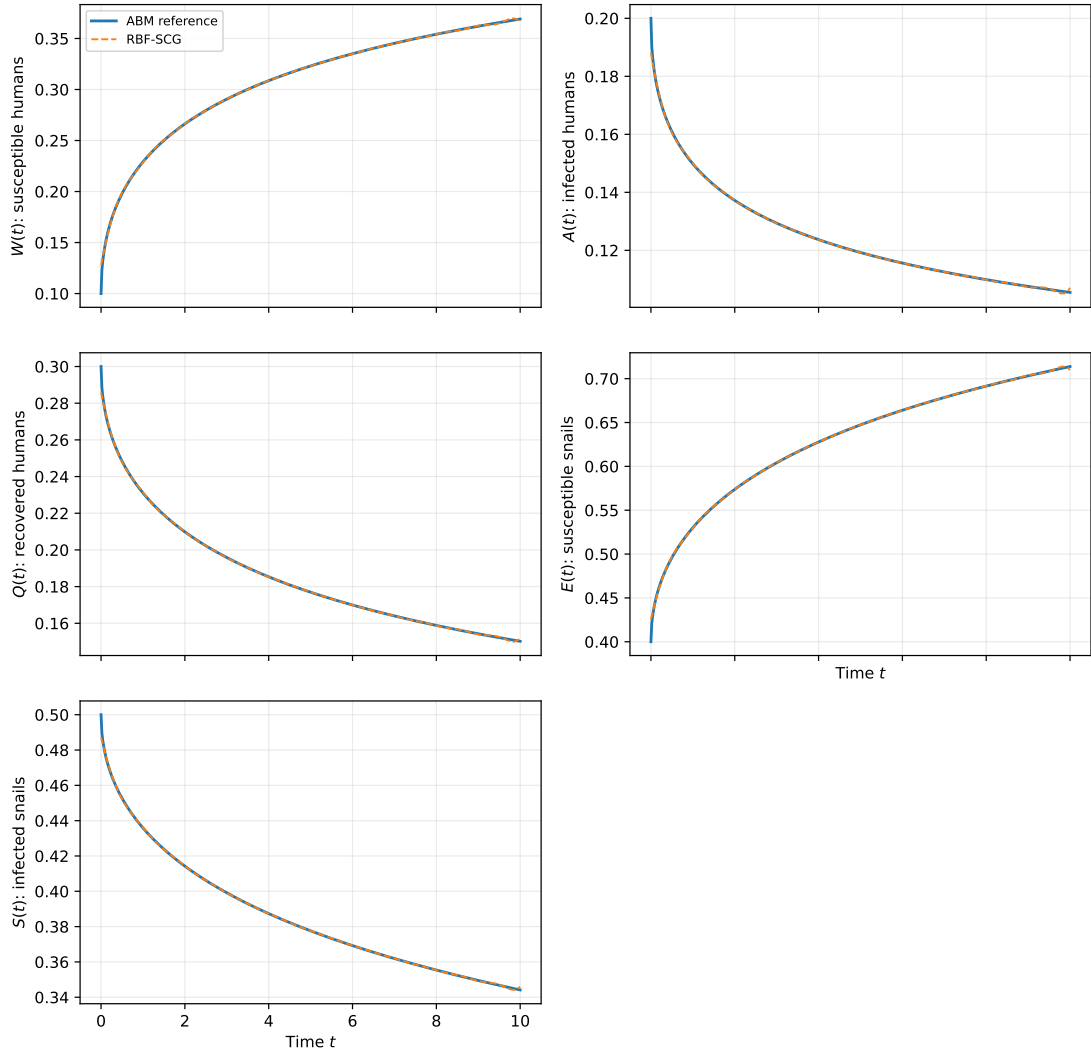


Figure 5: ABM reference and RBF-SCG trajectories for $\alpha = 0.5$.

Reference and neural-surrogate trajectories, $\alpha = 0.7$

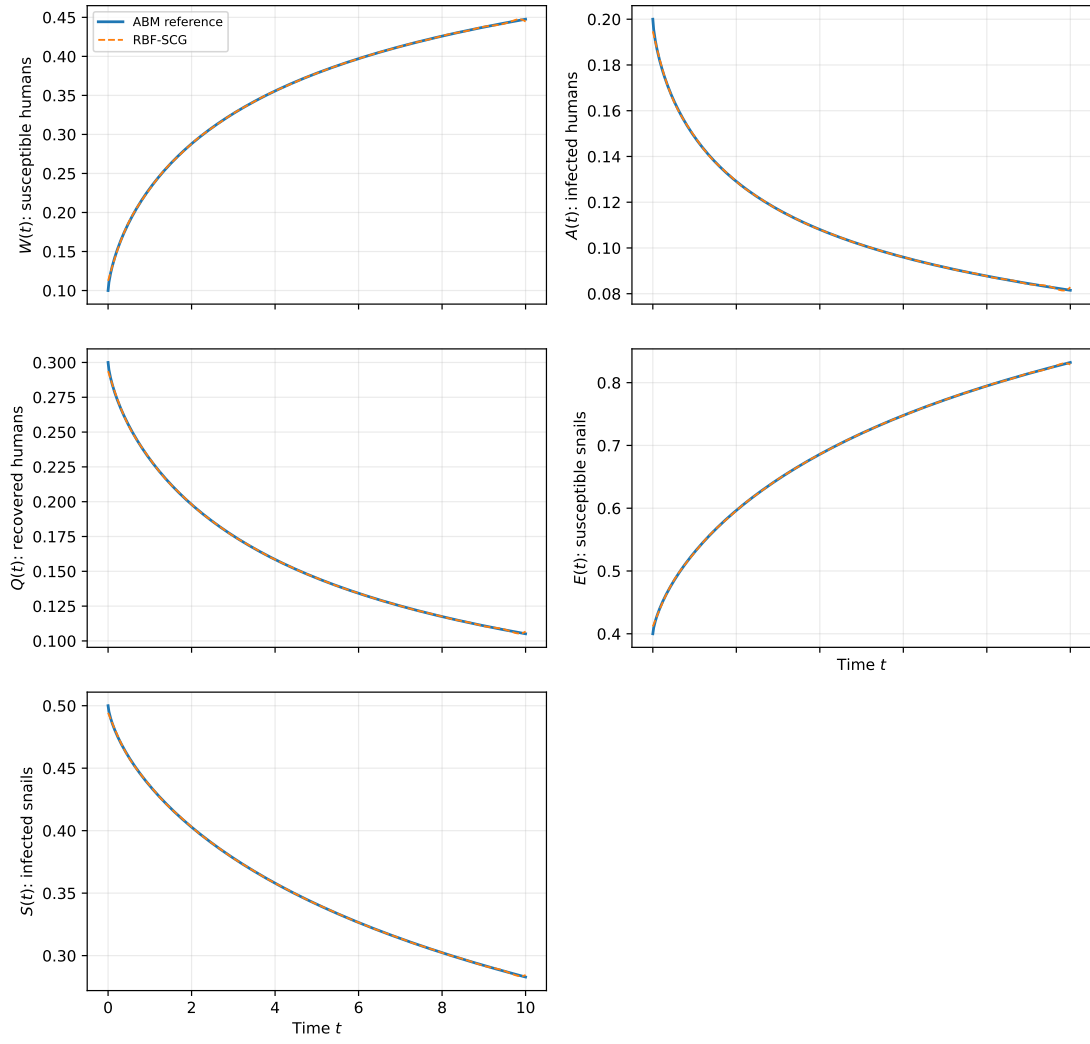


Figure 6: ABM reference and RBF-SCG trajectories for $\alpha = 0.7$.

Reference and neural-surrogate trajectories, $\alpha = 0.9$

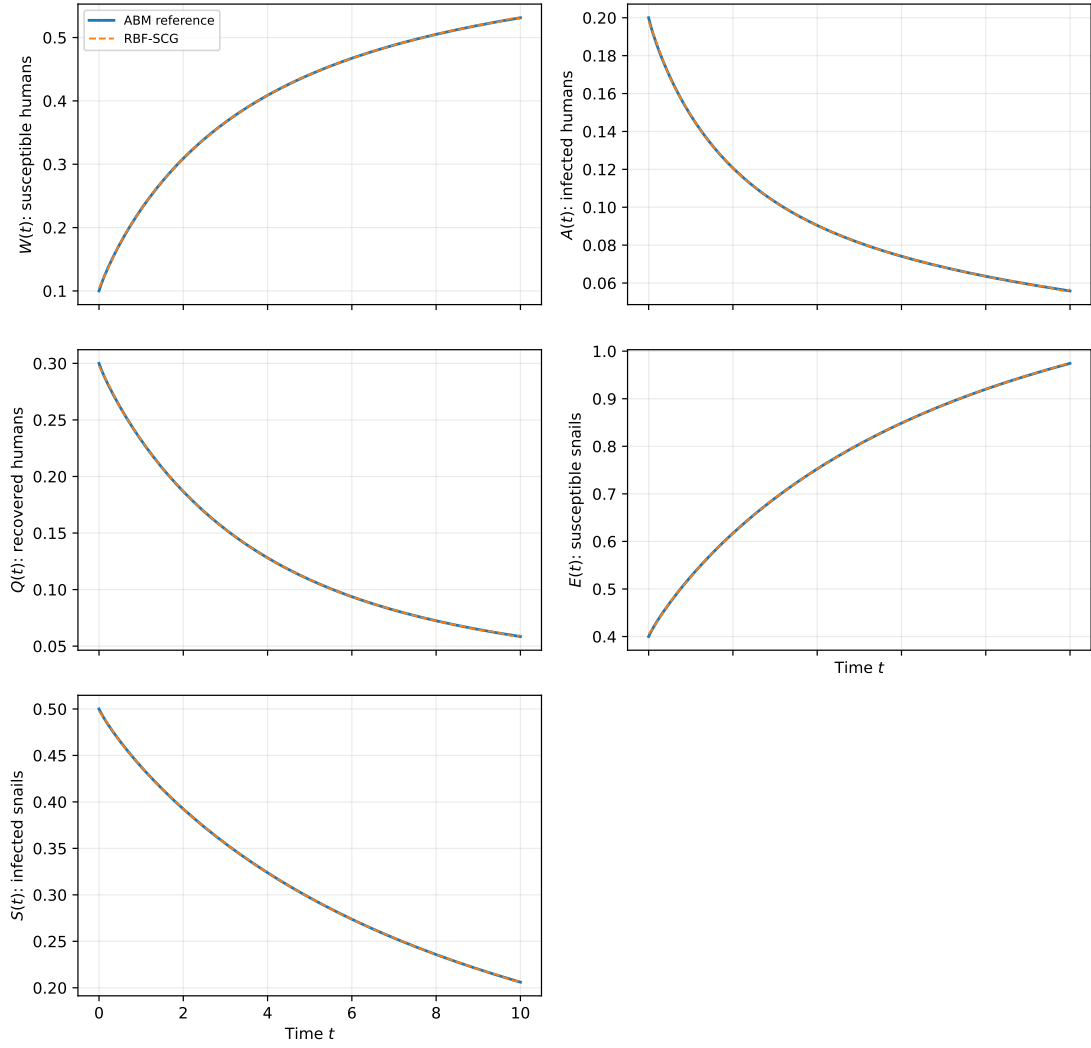


Figure 7: ABM reference and RBF-SCG trajectories for $\alpha = 0.9$.

7.3 Training, validation, and testing metrics

Table 3 reports aggregate metrics for each data subset. The test RMSE decreases from 2.406×10^{-4} at $\alpha = 0.5$ to 1.793×10^{-4} at $\alpha = 0.9$. All test R^2 values exceed 0.99996. Validation errors are larger than test errors because the fixed random split places several early high-curvature points in the validation subset. This behavior is reported rather than hidden by selecting a favorable split.

Table 3: Aggregate neural-surrogate metrics.

α	Subset	MAE	RMSE	E_∞	NRMSE	R^2
0.5	Train	2.153×10^{-4}	3.856×10^{-4}	4.581×10^{-3}	2.123×10^{-3}	0.999924
0.5	Validation	6.100×10^{-4}	2.462×10^{-3}	2.794×10^{-2}	1.215×10^{-2}	0.997401
0.5	Test	1.656×10^{-4}	2.406×10^{-4}	1.160×10^{-3}	1.675×10^{-3}	0.999962
0.7	Train	1.928×10^{-4}	3.204×10^{-4}	3.201×10^{-3}	1.243×10^{-3}	0.999978
0.7	Validation	4.100×10^{-4}	1.215×10^{-3}	1.243×10^{-2}	4.547×10^{-3}	0.999724
0.7	Test	1.504×10^{-4}	2.122×10^{-4}	9.686×10^{-4}	9.647×10^{-4}	0.999989
0.9	Train	1.627×10^{-4}	2.528×10^{-4}	2.195×10^{-3}	7.325×10^{-4}	0.999993
0.9	Validation	2.758×10^{-4}	6.027×10^{-4}	5.144×10^{-3}	1.724×10^{-3}	0.999966
0.9	Test	1.285×10^{-4}	1.793×10^{-4}	7.221×10^{-4}	5.743×10^{-4}	0.999996

The state-by-state metrics in table 4 show the same trend. The largest maximum error, 2.794×10^{-2} , occurs for W when $\alpha = 0.5$, while the global R^2 for that variable remains 0.999290. For $\alpha = 0.9$, all statewise maximum errors are below 5.2×10^{-3} .

Table 4: Statewise metrics evaluated on all 501 grid points.

α	State	MAE	RMSE	E_∞	NRMSE	R^2
0.5	A	1.581×10^{-4}	5.991×10^{-4}	1.187×10^{-2}	6.339×10^{-3}	0.998829
0.5	E	3.862×10^{-4}	1.329×10^{-3}	2.578×10^{-2}	4.234×10^{-3}	0.999610
0.5	Q	2.052×10^{-4}	7.245×10^{-4}	1.414×10^{-2}	4.836×10^{-3}	0.999439
0.5	S	1.915×10^{-4}	6.532×10^{-4}	1.267×10^{-2}	4.190×10^{-3}	0.999620
0.5	W	3.938×10^{-4}	1.424×10^{-3}	2.794×10^{-2}	5.291×10^{-3}	0.999290
0.7	A	1.273×10^{-4}	3.168×10^{-4}	5.290×10^{-3}	2.674×10^{-3}	0.999849
0.7	E	3.227×10^{-4}	7.357×10^{-4}	1.170×10^{-2}	1.702×10^{-3}	0.999953
0.7	Q	1.643×10^{-4}	3.860×10^{-4}	6.262×10^{-3}	1.981×10^{-3}	0.999930
0.7	S	1.630×10^{-4}	3.627×10^{-4}	5.727×10^{-3}	1.671×10^{-3}	0.999955
0.7	W	3.176×10^{-4}	7.592×10^{-4}	1.243×10^{-2}	2.184×10^{-3}	0.999911
0.9	A	9.059×10^{-5}	1.611×10^{-4}	1.877×10^{-3}	1.118×10^{-3}	0.999979
0.9	E	2.729×10^{-4}	4.671×10^{-4}	5.144×10^{-3}	8.133×10^{-4}	0.999991
0.9	Q	1.257×10^{-4}	2.167×10^{-4}	2.453×10^{-3}	8.973×10^{-4}	0.999988
0.9	S	1.445×10^{-4}	2.368×10^{-4}	2.507×10^{-3}	8.055×10^{-4}	0.999991
0.9	W	2.389×10^{-4}	4.155×10^{-4}	4.727×10^{-3}	9.636×10^{-4}	0.999986

7.4 Pointwise errors, regression, and SCG convergence

The absolute-error histories are displayed in figures 8a to 8c. The errors remain small across the full interval and decay rapidly after the initial high-curvature region. The regression diagrams in figure 9 place the held-out predictions close to the identity line. The SCG objective and gradient histories in figure 10 decrease monotonically after the warm start, confirming stable refinement of the output weights.

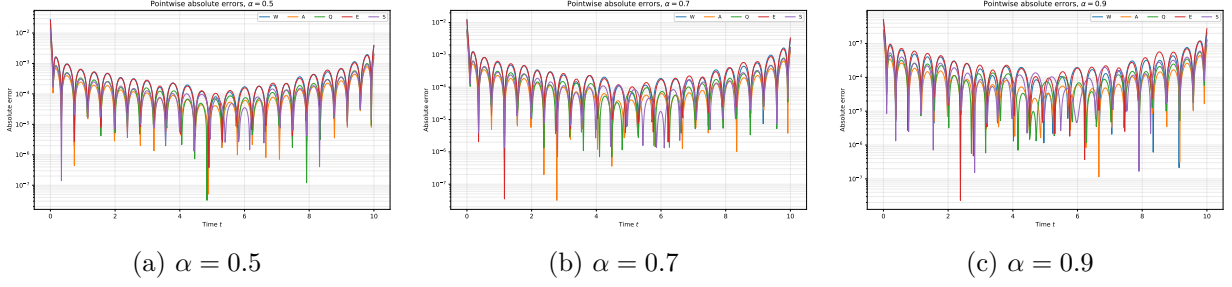


Figure 8: Pointwise absolute errors of the five neural outputs.

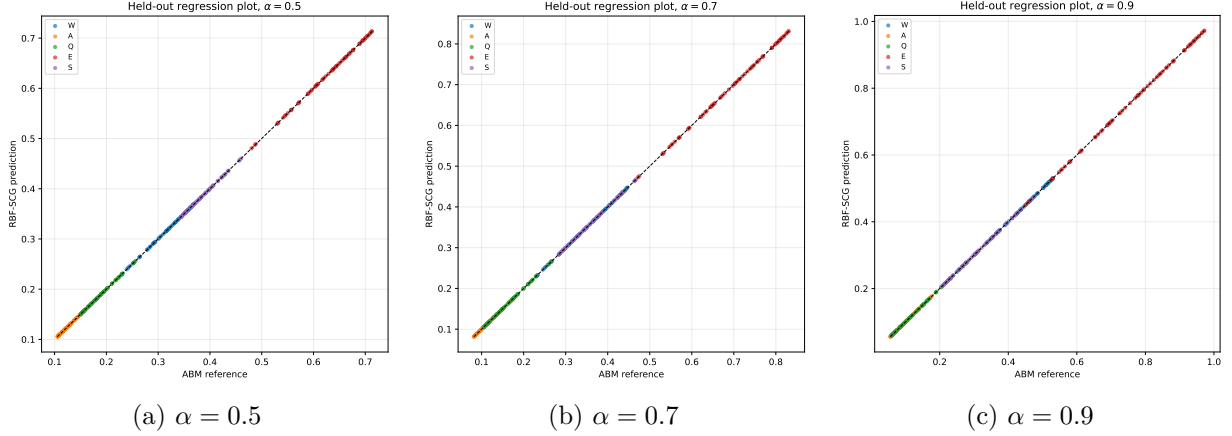


Figure 9: Held-out RBF-SCG predictions versus ABM reference values.

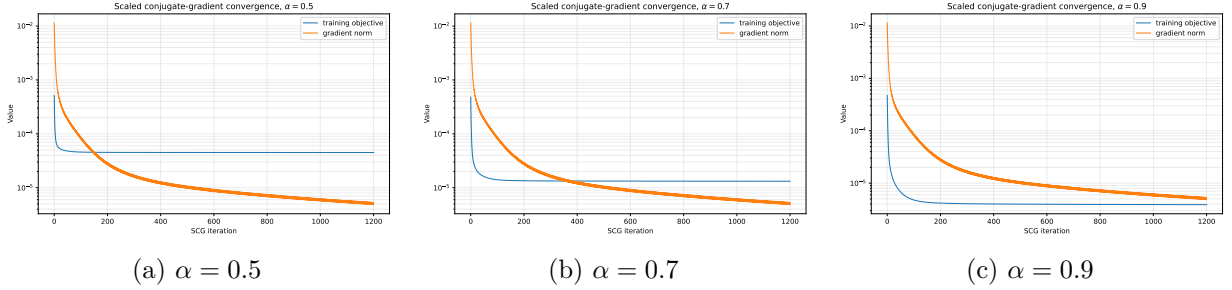


Figure 10: Scaled conjugate-gradient objective and gradient histories.

8 Discussion

The reconstructed model has three advantages over the incomplete formulation. First, every transfer is attached to a source compartment: recovery is proportional to infected humans, and snail mortality acts consistently on both snail classes. Second, the exact total-population equations provide analytical bounds and implementation checks. Third, the threshold number equation (12) connects the observed decay of infection to a standard epidemiological criterion.

The numerical results support the RBF network as a compact trajectory surrogate. Once trained, evaluating the network requires only two radial feature maps and a small matrix multiplication, whereas direct evaluation of the classical ABM history sum becomes increasingly costly as the time grid is refined. This makes the surrogate useful for repeated visualization, parameter-screening prototypes, inverse-problem initialization, or embedding within a larger optimization loop.

Several limitations must be emphasized. The parameter set is a nondimensional benchmark rather than a fit to surveillance data. The network is trained separately for each fractional order and does not generalize automatically to new parameter vectors. Randomly held-out time points assess interpolation, not long-horizon extrapolation. Moreover, the surrogate inherits any discretization error present in the ABM reference data. A future physics-informed version would include the discretized Caputo residual and the initial-condition residual in the training objective, but such a method would require a distinct validation study and should not be conflated with the supervised construction used here.

A stronger epidemiological study should calibrate the model to a specific endemic region, quantify parameter uncertainty, compare alternative memory kernels, and investigate intervention controls. On the computational side, fast convolution, sum-of-exponentials, or short-memory approximations could replace the $O(N^2)$ history summation for long simulations.

9 Conclusions

A complete and reproducible analysis of a five-compartment fractional schistosomiasis model has been presented. The corrected model preserves nonnegativity, admits explicit bounds for the human and snail totals, and has a disease-free threshold governed by $\mathcal{R}_0$. For the benchmark parameters, $\mathcal{R}_0 \approx 0.6977$, and the numerical trajectories consistently approach the disease-free state.

The fractional Adams–Bashforth–Moulton method was used as the reference solver. A two-hidden-layer RBF network with 15 and 25 radial units was trained as a supervised surrogate using scaled conjugate-gradient refinement. Across three fractional orders, the held-out test RMSE remained between 1.793×10^{-4} and 2.406×10^{-4} , with $R^2 > 0.99996$. These results demonstrate accurate approximation of the validated trajectories while preserving the methodological distinction between a numerical solver and a neural surrogate.

Author contributions

G.C.A. contributed to conceptualization, epidemiological modeling, and the initial manuscript. A.H.S. contributed to mathematical analysis, numerical methodology, software, validation, visualization, and manuscript revision. Both authors reviewed and approved the final manuscript.

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Conflicts of interest

The authors declare no conflicts of interest.

Data Availability

All data generated or analysed during this study are included in this article and its Supplementary Information files. The Supplementary Information contains the Python source code, CSV files, figure files, and the complete LaTeX source required to reproduce the numerical results.

Ethics statement

This work uses no individual-level human or animal data and therefore requires no ethics approval.

A Reproducibility algorithm

The following steps reproduce the reported experiment:

1. Set the parameters in table 1, the initial condition in equation (5), and the seed to 20260716.
2. For each $\alpha \in \{0.5, 0.7, 0.9\}$, solve equation (4) on $[0, 10]$ with $h = 0.02$ using equations (14) and (16).
3. Normalize time and standardize each output using training-set statistics.
4. Build 15 first-layer and 25 second-layer Gaussian radial features.
5. Partition the 501 samples into 70% training, 15% validation, and 15% testing using the fixed permutation.
6. Refine the output weights by SCG for at most 1200 iterations.
7. Evaluate equations (23) to (27) and export all figures and CSV files.

B Software structure

main.tex

Complete manuscript source.

references.bib

Bibliographic database.

code/generate_results.py

ABM solver, RBF–SCG surrogate, tables, and figures.

data/

Reference trajectories, predictions, errors, metrics, and metadata.

figures/

Vector PDF and 300-dpi PNG versions of all figures.

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