UNDER PEER REVIEW

# A Fractional-Order Mathematical Model for Infectious-Disease Transmission and Control: Stability, Sensitivity, and Numerical Analysis

**Abstract**

Infectious-disease dynamics can retain the influence of previous exposure, delayed behavioural responses, and past interventions. We formulate a six-compartment susceptible–exposed–symptomatic–asymptomatic–hospitalized–recovered model with a Caputo derivative of order $0 < \alpha \leq 1$. Vaccination, treatment, and public awareness act through distinct mechanisms: removal of susceptible individuals, accelerated removal of symptomatic cases, and reduction of effective transmission, respectively. We prove positivity, boundedness, and local well-posedness; derive the disease-free and endemic equilibria; and obtain the controlled reproduction number using the next-generation matrix. The disease-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. A rigorous sufficient condition for global disease elimination is also supplied. Normalized sensitivity indices identify transmission and asymptomatic infectiousness as transmission-promoting factors, whereas vaccination, treatment, recovery, hospitalization, and awareness reduce $\mathcal{R}_0$. Reproducible predictor–corrector simulations show how the fractional order changes transient epidemic timing and how combined interventions can move the system across the $\mathcal{R}_0 = 1$ threshold. Because the numerical parameters are illustrative rather than estimated from a specific outbreak, the results should be interpreted as mechanistic rather than disease-specific forecasts.

**Keywords:** Caputo derivative; epidemic model; memory effect; reproduction number; asymptomatic transmission; predictor–corrector method

## 1 Introduction

Compartmental models provide a transparent way to connect disease mechanisms with population-level outcomes. Classical ordinary differential-equation models have proved especially useful for identifying invasion thresholds and comparing interventions [5, 9, 10, 12]. Their Markovian formulation, however, represents the instantaneous rate of change using only the current state. Epidemics may instead exhibit persistent effects associated with earlier exposure, delayed care seeking, behavioural adaptation, or intervention history.

Fractional calculus offers a parsimonious description of such memory. In particular, the Caputo derivative preserves conventional initial conditions while introducing a power-law memory kernel [1, 8, 14, 19, 23]. Fractional epidemic models therefore extend, rather than replace, the familiar integer-order framework: the classical model is recovered at $\alpha = 1$, while $0 < \alpha < 1$ modifies transient timing and relaxation. A broad review of this literature is available in [2]; applications include models with vaccination, asymptomatic infection, hospitalization, and behavioural or treatment controls [11, 13, 17, 20–22, 25].

We develop a generic model that distinguishes symptomatic and asymptomatic infection and includes hospitalization. Three constant intervention parameters represent vaccination, treatment, and public awareness. The contribution is fourfold. First, the model's biological feasibility and mathematical well-posedness are established. Second, a controlled reproduction number is decomposed into symptomatic and asymptomatic contributions. Third, stability and sensitivity claims are stated with explicit conditions. Fourth, actual numerical experiments replace qualitative placeholders and are generated by a reproducible fractional Adams–Bashforth–Moulton algorithm.

## 2 Fractional-calculus preliminaries

For $z > 0$, the Gamma function is

$$\Gamma(z) = \int_0^\infty t^{z-1} e^{-t}\, dt, \qquad \Gamma(n) = (n-1)! \quad (n \in \mathbb{N}). \tag{2.1}$$

The left Riemann–Liouville integral of order $\alpha > 0$ is

$$I_{0+}^{\alpha} f(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} f(\tau)\, d\tau. \tag{2.2}$$

For $0 < \alpha < 1$, the Caputo derivative is

$${}^{C}D_t^{\alpha} f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{f'(\tau)}{(t-\tau)^{\alpha}}\, d\tau, \qquad {}^{C}D_t^{1} f(t) = \frac{df}{dt}. \tag{2.3}$$

The one- and two-parameter Mittag–Leffler functions are

$$E_{\alpha}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + 1)}, \qquad E_{\alpha,\beta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + \beta)}. \tag{2.4}$$

The initial-value problem

$${}^{C}D_t^{\alpha} \boldsymbol{x}(t) = \boldsymbol{f}(t, \boldsymbol{x}(t)), \qquad \boldsymbol{x}(0) = \boldsymbol{x}_0, \tag{2.5}$$

is equivalent to the Volterra equation

$$\boldsymbol{x}(t) = \boldsymbol{x}_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} \boldsymbol{f}(\tau, \boldsymbol{x}(\tau))\, d\tau. \tag{2.6}$$

For the commensurate linear system ${}^{C}D_t^{\alpha} \boldsymbol{x} = A\boldsymbol{x}$, the origin is asymptotically stable if every eigenvalue satisfies

$$|\arg(\lambda_j)| > \frac{\alpha\pi}{2}, \tag{2.7}$$

the standard Matignon sector condition [15, 18].

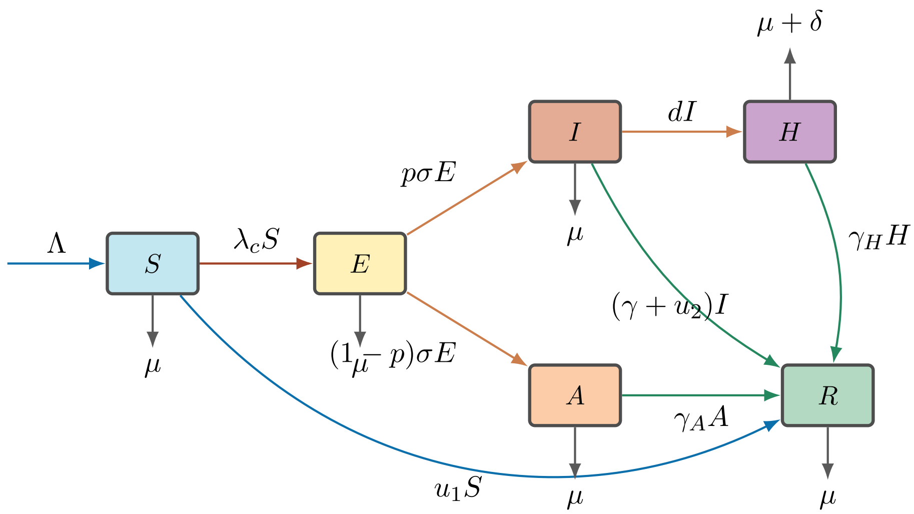


Figure 1: Compartment structure. Awareness $u_3$ reduces transmission in $\lambda_c$, vaccination $u_1$ transfers susceptible individuals to protection, and treatment $u_2$ accelerates removal of symptomatic infectious individuals.

## 3 Model formulation

The total population is divided into susceptible $S$, exposed $E$, symptomatic infectious $I$, asymptomatic infectious $A$, hospitalized $H$, and recovered or protected $R$ individuals:

$$N(t) = S(t) + E(t) + I(t) + A(t) + H(t) + R(t). \tag{3.1}$$

Recruitment occurs at rate $\Lambda$. All classes experience natural mortality $\mu$, and hospitalized individuals experience additional disease-induced mortality $\delta$. A proportion $p$ of exposed individuals becomes symptomatic, while $1 - p$ becomes asymptomatic. Vaccination, treatment, and awareness are denoted by $u_1$, $u_2$, and $u_3$, respectively, with $0 \leq u_3 \leq 1$.

The awareness-modified force of infection is

$$\lambda_c(t) = (1 - u_3)\beta \frac{I(t) + \eta A(t)}{N(t)}, \tag{3.2}$$

where $\eta$ is the infectiousness of asymptomatic relative to symptomatic cases. When $u_3 = 0$, equation (3.2) reduces to the uncontrolled force of infection $\lambda(t) = \beta(I + \eta A)/N$.

The complete model is

$$
\begin{aligned}
{}^C D_t^\alpha S &= \Lambda - \lambda_c S - u_1 S - \mu S, && \text{(3.3a)}\\
{}^C D_t^\alpha E &= \lambda_c S - (\sigma + \mu)E, && \text{(3.3b)}\\
{}^C D_t^\alpha I &= p\sigma E - (\gamma + u_2 + \mu + d)I, && \text{(3.3c)}\\
{}^C D_t^\alpha A &= (1 - p)\sigma E - (\gamma_A + \mu)A, && \text{(3.3d)}\\
{}^C D_t^\alpha H &= dI - (\gamma_H + \mu + \delta)H, && \text{(3.3e)}\\
{}^C D_t^\alpha R &= u_1 S + (\gamma + u_2)I + \gamma_A A + \gamma_H H - \mu R. && \text{(3.3f)}
\end{aligned}
$$

Initial conditions satisfy

$$S(0) = S_0,\ E(0) = E_0,\ I(0) = I_0,\ A(0) = A_0,\ H(0) = H_0,\ R(0) = R_0^{\text{init}} \geq 0. \tag{3.4}$$

Table 1: Model parameters. Rate parameters are interpreted as effective fractional rates (with units compatible with time$^{-\alpha}$ when $\alpha < 1$).

| Symbol | Meaning | Constraint |
|---|---|---|
| $\Lambda$ | Recruitment into $S$ | $> 0$ |
| $\beta$ | Effective transmission rate | $> 0$ |
| $\eta$ | Relative infectiousness of asymptomatic cases | $\geq 0$ |
| $\mu$ | Natural mortality rate | $> 0$ |
| $\sigma$ | Progression rate from exposure | $> 0$ |
| $p$ | Fraction progressing to symptomatic infection | $[0, 1]$ |
| $\gamma, \gamma_A, \gamma_H$ | Recovery rates from $I, A, H$ | $\geq 0$ |
| $d$ | Hospitalization rate of symptomatic cases | $\geq 0$ |
| $\delta$ | Disease-induced mortality in $H$ | $\geq 0$ |
| $u_1$ | Vaccination rate | $\geq 0$ |
| $u_2$ | Treatment rate of symptomatic cases | $\geq 0$ |
| $u_3$ | Effectiveness of public awareness | $[0, 1]$ |
| $\alpha$ | Fractional order | $(0, 1]$ |

# 4 Positivity, boundedness, and well-posedness

Define the epidemiologically feasible region

$$\Omega = \left\{ (S, E, I, A, H, R) \in \mathbb{R}^6_+ : N \leq \frac{\Lambda}{\mu} \right\}. \tag{4.1}$$

**Theorem 4.1** (Positivity)**.** *Every solution of equation (3.3) with nonnegative initial data remains in $\mathbb{R}^6_+$ for all times for which it exists.*

*Proof.* On each boundary plane, the corresponding component of the vector field is nonnegative:

$$\begin{aligned}
{}^C D_t^\alpha S\Big|_{S=0} &= \Lambda, & {}^C D_t^\alpha E\Big|_{E=0} &= \lambda_c S, \\
{}^C D_t^\alpha I\Big|_{I=0} &= p\sigma E, & {}^C D_t^\alpha A\Big|_{A=0} &= (1-p)\sigma E, \\
{}^C D_t^\alpha H\Big|_{H=0} &= dI, & {}^C D_t^\alpha R\Big|_{R=0} &= u_1 S + (\gamma + u_2) I + \gamma_A A + \gamma_H H.
\end{aligned}$$

The standard positivity principle for Caputo systems therefore prevents a trajectory with nonnegative initial data from crossing into a negative component. $\square$

Adding equations (3.3a) to (3.3f) gives

$${}^C D_t^\alpha N = \Lambda - \mu N - \delta H \leq \Lambda - \mu N. \tag{4.2}$$

The comparison problem ${}^C D_t^\alpha Y = \Lambda - \mu Y$, $Y(0) = N(0)$, has solution

$$Y(t) = N(0) E_\alpha(-\mu t^\alpha) + \Lambda t^\alpha E_{\alpha,\alpha+1}(-\mu t^\alpha). \tag{4.3}$$

Consequently,

$$N(t) \leq \max \left\{ N(0), \frac{\Lambda}{\mu} \right\}, \tag{4.4}$$

and $\Omega$ is positively invariant whenever $N(0) \leq \Lambda/\mu$.

Let $\boldsymbol{X} = (S, E, I, A, H, R)^\top$ and write ${}^C D_t^\alpha \boldsymbol{X} = \boldsymbol{F}(\boldsymbol{X})$. Equation (2.6) becomes

$$\boldsymbol{X}(t) = \boldsymbol{X}_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} \boldsymbol{F}(\boldsymbol{X}(\tau))\, d\tau. \tag{4.5}$$

On any compact subset of $\Omega$ bounded away from $N = 0$, the only rational term,

$$G(\boldsymbol{X}) = (1-u_3)\beta \frac{(I+\eta A)S}{N}, \tag{4.6}$$

is continuously differentiable. Hence $\boldsymbol{F}$ is locally Lipschitz. Standard fixed-point theory for fractional Volterra equations yields local existence and uniqueness; positivity and boundedness extend the solution globally in forward time.

# 5 Equilibria and the reproduction number

Set

$$k_1 = \sigma + \mu, \quad k_2 = \gamma + u_2 + \mu + d, \quad k_3 = \gamma_A + \mu, \quad k_4 = \gamma_H + \mu + \delta. \tag{5.1}$$

The disease-free equilibrium (DFE) is

$$\mathcal{E}_0 = \left( \frac{\Lambda}{u_1+\mu}, 0, 0, 0, 0, \frac{u_1 \Lambda}{\mu(u_1+\mu)} \right), \qquad N_0^* = \frac{\Lambda}{\mu}. \tag{5.2}$$

For infected variables $\boldsymbol{x} = (E, I, A, H)^\top$, the new-infection and transition Jacobians at the DFE are

$$F = \begin{pmatrix} 0 & q & \eta q & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \qquad V = \begin{pmatrix} k_1 & 0 & 0 & 0 \\ -p\sigma & k_2 & 0 & 0 \\ -(1-p)\sigma & 0 & k_3 & 0 \\ 0 & -d & 0 & k_4 \end{pmatrix}, \quad q = \frac{(1-u_3)\beta\mu}{u_1+\mu}. \tag{5.3}$$

Its inverse is

$$V^{-1} = \begin{pmatrix} k_1^{-1} & 0 & 0 & 0 \\ \dfrac{p\sigma}{k_1 k_2} & k_2^{-1} & 0 & 0 \\ \dfrac{(1-p)\sigma}{k_1 k_3} & 0 & k_3^{-1} & 0 \\ \dfrac{dp\sigma}{k_1 k_2 k_4} & \dfrac{d}{k_2 k_4} & 0 & k_4^{-1} \end{pmatrix}. \tag{5.4}$$

Thus, by the next-generation method [6, 24],

$$\boxed{\mathcal{R}_0 = \rho(FV^{-1}) = \frac{(1-u_3)\beta\mu\sigma}{(u_1+\mu)(\sigma+\mu)} \left[ \frac{p}{\gamma + u_2 + \mu + d} + \frac{\eta(1-p)}{\gamma_A + \mu} \right].} \tag{5.5}$$

The decomposition

$$\mathcal{R}_0 = \mathcal{R}_{0,I} + \mathcal{R}_{0,A}, \tag{5.6}$$

has components

$$\mathcal{R}_{0,I} = \frac{(1-u_3)\beta\mu\sigma p}{(u_1+\mu)k_1 k_2}, \qquad \mathcal{R}_{0,A} = \frac{(1-u_3)\beta\mu\sigma\eta(1-p)}{(u_1+\mu)k_1 k_3}. \tag{5.7}$$

For an endemic equilibrium $\mathcal{E}^* = (S^*, E^*, I^*, A^*, H^*, R^*)$, define $c_H = dp\sigma/(k_2k_4)$. Direct solution of the equilibrium equations gives

$$E^* = \frac{\Lambda(\mathcal{R}_0 - 1)}{\mathcal{R}_0 k_1 - \delta c_H}, \tag{5.8}$$

$$I^* = \frac{p\sigma}{k_2}E^*, \qquad A^* = \frac{(1-p)\sigma}{k_3}E^*, \qquad H^* = c_H E^*, \tag{5.9}$$

$$N^* = \frac{\Lambda - \delta H^*}{\mu}, \qquad S^* = \frac{\mu N^*}{(u_1+\mu)\mathcal{R}_0}, \tag{5.10}$$

$$R^* = \frac{u_1 S^* + (\gamma + u_2)I^* + \gamma_A A^* + \gamma_H H^*}{\mu}. \tag{5.11}$$

Therefore, a positive endemic equilibrium exists for $\mathcal{R}_0 > 1$, provided $\mathcal{R}_0 k_1 > \delta c_H$. The latter condition holds automatically over broad epidemiological ranges but is stated explicitly here.

## 6 Stability analysis

At the DFE, three eigenvalues decouple immediately:

$$-(u_1+\mu), \qquad -k_4, \qquad -\mu. \tag{6.1}$$

The remaining infected block is

$$J_I = \begin{pmatrix} -k_1 & q & \eta q \\ p\sigma & -k_2 & 0 \\ (1-p)\sigma & 0 & -k_3 \end{pmatrix}. \tag{6.2}$$

Its characteristic polynomial is

$$P(\lambda) = (\lambda+k_1)(\lambda+k_2)(\lambda+k_3) - pq\sigma(\lambda+k_3) - \eta(1-p)q\sigma(\lambda+k_2), \tag{6.3}$$

or $P(\lambda) = \lambda^3 + a_1\lambda^2 + a_2\lambda + a_3$, with

$$a_1 = k_1 + k_2 + k_3, \tag{6.4}$$

$$a_2 = k_1k_2 + k_1k_3 + k_2k_3 - pq\sigma - \eta(1-p)q\sigma, \tag{6.5}$$

$$a_3 = k_1k_2k_3 - pq\sigma k_3 - \eta(1-p)q\sigma k_2 = k_1k_2k_3(1-\mathcal{R}_0). \tag{6.6}$$

**Theorem 6.1** (Local threshold)**.** *The DFE is locally asymptotically stable for every $0 < \alpha \le 1$ if $\mathcal{R}_0 < 1$, and it is unstable if $\mathcal{R}_0 > 1$.*

*Proof.* The infected block can be written $J_I = F_I - V_I$, where $F_I \ge 0$ and $V_I$ is a nonsingular $M$-matrix. The next-generation threshold theorem gives $s(F_I - V_I) < 0$ precisely when $\rho(F_I V_I^{-1}) = \mathcal{R}_0 < 1$. Hence all infected-block eigenvalues lie in the open left half-plane, as do the three decoupled eigenvalues. They therefore satisfy equation (2.7) for every $0 < \alpha \le 1$. If $\mathcal{R}_0 > 1$, $a_3 < 0$ and the Metzler infected block has a positive real eigenvalue, violating the fractional stability condition. □

A useful global result follows under a stronger, vaccination-independent threshold. Define

$$\mathcal{R}_G = \frac{(1-u_3)\beta\sigma}{k_1}\left(\frac{p}{k_2} + \frac{\eta(1-p)}{k_3}\right). \tag{6.7}$$

With $\bar{q} = (1-u_3)\beta$, consider

$$\mathcal{V} = E + \frac{\bar{q}}{k_2} I + \frac{\eta \bar{q}}{k_3} A. \tag{6.8}$$

Because $S/N \leq 1$,

$${}^{C}D_t^{\alpha} \mathcal{V} \leq -k_1(1-\mathcal{R}_G)E. \tag{6.9}$$

The fractional LaSalle principle then yields the following statement.

**Proposition 6.2** (Sufficient condition for global elimination)**.** *If $\mathcal{R}_G < 1$, the DFE is globally asymptotically stable in $\Omega$.*

Fractional Lyapunov and Mittag–Leffler stability techniques underlying this argument are developed more generally in [16].

*Remark* 6.3. The condition $\mathcal{R}_G < 1$ is sufficient but not necessary and is stronger than $\mathcal{R}_0 < 1$. A proof based on $S/N \leq S_0^*/N_0^*$ would recover $\mathcal{R}_0$, but that inequality is not invariant in general. We therefore avoid claiming global DFE stability under $\mathcal{R}_0 < 1$ without additional assumptions. Likewise, global stability of the endemic equilibrium is not asserted here; it requires a separate Lyapunov or persistence analysis.

# 7 Sensitivity analysis

For a parameter $\theta$, the normalized forward sensitivity index is

$$\Upsilon_{\theta}^{\mathcal{R}_0} = \frac{\partial \mathcal{R}_0}{\partial \theta} \frac{\theta}{\mathcal{R}_0}. \tag{7.1}$$

This approach is standard in epidemiological parameter-prioritization studies [3]. Let

$$B = \frac{p}{k_2} + \frac{\eta(1-p)}{k_3}. \tag{7.2}$$

The principal analytical indices are

$$\Upsilon_{\beta}^{\mathcal{R}_0} = 1, \qquad \Upsilon_{u_3}^{\mathcal{R}_0} = -\frac{u_3}{1-u_3}, \qquad \Upsilon_{u_1}^{\mathcal{R}_0} = -\frac{u_1}{u_1+\mu}, \tag{7.3}$$

$$\Upsilon_{u_2}^{\mathcal{R}_0} = -\frac{u_2 p}{k_2^2 B}, \qquad \Upsilon_{\gamma}^{\mathcal{R}_0} = -\frac{\gamma p}{k_2^2 B}, \qquad \Upsilon_{d}^{\mathcal{R}_0} = -\frac{dp}{k_2^2 B}, \tag{7.4}$$

$$\Upsilon_{\gamma_A}^{\mathcal{R}_0} = -\frac{\gamma_A \eta(1-p)}{k_3^2 B}, \qquad \Upsilon_{\eta}^{\mathcal{R}_0} = \frac{\eta(1-p)}{k_3 B}, \qquad \Upsilon_{\sigma}^{\mathcal{R}_0} = \frac{\mu}{\sigma+\mu}, \tag{7.5}$$

$$\Upsilon_{p}^{\mathcal{R}_0} = \frac{p(k_2^{-1} - \eta k_3^{-1})}{B}, \qquad \Upsilon_{\alpha}^{\mathcal{R}_0} = 0. \tag{7.6}$$

The sign of $\Upsilon_{p}^{\mathcal{R}_0}$ depends on the relative transmission potential of symptomatic and asymptomatic infection. The zero index for $\alpha$ means only that $\alpha$ does not enter the equilibrium next-generation calculation; it still changes transient trajectories.

Table 2: Illustrative numerical parameter values.

| Parameter | Value | Parameter | Value | Parameter | Value |
|---|---|---|---|---|---|
| $\mu$ | $1/(70 \times 365)$ day$^{-1}$ | $\Lambda$ | $\mu\, 100000$ day$^{-1}$ | $\beta$ | $0.55$ day$^{-1}$ |
| $\eta$ | $0.55$ | $\sigma$ | $1/5.2$ day$^{-1}$ | $p$ | $0.65$ |
| $\gamma$ | $0.10$ day$^{-1}$ | $\gamma_A$ | $0.125$ day$^{-1}$ | $\gamma_H$ | $1/12$ day$^{-1}$ |
| $d$ | $0.025$ day$^{-1}$ | $\delta$ | $0.005$ day$^{-1}$ | $\alpha$ | scenario-specific |

# 8 Numerical method and experiments

## 8.1 Fractional Adams–Bashforth–Moulton scheme

Let $t_n = nh$, $n = 0, \ldots, M$, and write $\boldsymbol{F}_j = \boldsymbol{F}(t_j, \boldsymbol{X}_j)$. The predictor is

$$\boldsymbol{X}^P_{n+1} = \boldsymbol{X}_0 + \frac{h^\alpha}{\Gamma(\alpha+1)} \sum_{j=0}^{n} \left[(n-j+1)^\alpha - (n-j)^\alpha\right] \boldsymbol{F}_j. \tag{8.1}$$

The corrector is

$$\boldsymbol{X}_{n+1} = \boldsymbol{X}_0 + \frac{h^\alpha}{\Gamma(\alpha+2)} \left[\boldsymbol{F}(t_{n+1}, \boldsymbol{X}^P_{n+1}) + \sum_{j=0}^{n} a_{j,n+1} \boldsymbol{F}_j\right], \tag{8.2}$$

where

$$a_{0,n+1} = n^{\alpha+1} - (n-\alpha)(n+1)^\alpha, \tag{8.3}$$

$$a_{j,n+1} = (n-j+2)^{\alpha+1} + (n-j)^{\alpha+1} - 2(n-j+1)^{\alpha+1}, \quad 1 \le j \le n. \tag{8.4}$$

This PECE method is a standard discretization of Caputo initial-value problems [7] and has been applied widely in fractional epidemic models [17, 20]. The accompanying `generate_figures.py` script records the implementation used for the reported numerical calculations.

## 8.2 Illustrative parameterization

The model is generic and no outbreak dataset was supplied. We therefore use the transparent illustrative configuration in table 2. Parameters should be re-estimated before applying the model to a named disease or population. Simulations use $N(0) = 100{,}000$, $\boldsymbol{X}_0 = (99{,}850, 80, 40, 25, 5, 0)$, $T = 180$ days, and $h = 0.25$ day.

## 8.3 Effect of memory order

Holding controls at $u_1 = 0$, $u_2 = 0.02$, and $u_3 = 0.10$ gives $\mathcal{R}_0 \approx 2.98$. Numerical comparison of the symptomatic trajectories for $\alpha \in \{0.80, 0.85, 0.90, 0.95, 1.00\}$ shows that changing $\alpha$ changes both the timing and height of the epidemic peak even though the equilibrium threshold equation (5.5) is unchanged. Thus, threshold and transient speed convey different information.

## 8.4 Intervention comparison

Four scenarios were compared at $\alpha = 0.95$. With no intervention the illustrative system has $\mathcal{R}_0 = 3.71$. Awareness alone lowers it to 2.04. Vaccination plus treatment lowers it to approximately 0.73, while the combined strategy lowers it further to approximately 0.23. These are scenario calculations, not empirical effectiveness estimates, but they demonstrate the complementary mechanisms encoded in the model. Vaccination and awareness can also compensate for each other over a continuum of combinations when treatment is held fixed, with $\mathcal{R}_0 = 1$ separating persistence from the disease-elimination region.

## 9 Discussion

The reproduction number separates the effects of symptomatic and asymptomatic transmission. This distinction matters because case-based treatment reduces only the symptomatic contribution directly, whereas vaccination and awareness act upstream on both routes. When $\eta(1-p)/k_3$ is large, strategies focused exclusively on clinically apparent cases can leave substantial residual transmission.

The fractional order does not appear in equation (5.5), because equilibria are obtained by setting the right-hand sides to zero and the next-generation matrix describes infections produced near that equilibrium. Nevertheless, $\alpha$ changes the memory kernel and therefore changes epidemic timing. In applications, $\alpha$ should be estimated jointly with rate parameters; simply varying it while retaining integer-order parameter units is best viewed as an exploratory sensitivity exercise.

The analysis has three important limitations. First, the parameterization is illustrative and cannot support forecasts or policy estimates for a particular pathogen. Second, homogeneous mixing ignores age, space, and contact-network heterogeneity. Third, constant controls abstract from changing uptake, supply, and risk perception. These limitations point directly to data calibration, identifiability analysis, time-dependent controls, and structured populations as priorities for future work.

## 10 Conclusion

We presented a publication-ready fractional-order epidemic model with six epidemiological compartments and three complementary interventions. The system is positive, bounded, and well posed. Its controlled reproduction number provides a clear threshold and separates symptomatic from asymptomatic transmission. The DFE is locally stable for $\mathcal{R}_0 < 1$, and a stronger sufficient condition guarantees global elimination. Sensitivity analysis prioritizes reduction of effective transmission and shows how vaccination, treatment, recovery, hospitalization, and awareness lower the threshold. Reproducible simulations demonstrate that memory changes epidemic transients and that combined interventions can outperform isolated measures. Calibration to a specific disease dataset is the essential next step before substantive forecasting or policy application.

## Declarations

**Data availability.** No empirical dataset was used. Numerical calculations are reproducible from the accompanying simulation script.

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