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

Abstract

Infectious-disease dynamics may retain effects associated with previous exposure, delayed behavioural responses, and earlier interventions. This study formulates a six-compartment susceptible–exposed–symptomatic–asymptomatic–hospitalised–recovered model governed by a Caputo derivative of order $0 < \alpha \leq 1$. Vaccination, treatment, and public awareness are represented through distinct mechanisms: removal of susceptible individuals, accelerated removal of symptomatic infectious individuals, and reduction of effective transmission, respectively. The analysis establishes positivity, boundedness, and local well-posedness; derives the disease-free and endemic equilibria; and obtains 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 $\mathcal{R}_0 > 1$, while a stronger sufficient condition is provided for global disease elimination. The reproduction number is decomposed into symptomatic and asymptomatic contributions, clarifying the distinct roles of the two infectious pathways. Normalised sensitivity indices identify transmission and asymptomatic infectiousness as factors that increase $\mathcal{R}_0$, whereas vaccination, treatment, recovery, hospitalisation, and awareness reduce the threshold. Reproducible predictor–corrector simulations show that the fractional order affects transient epidemic timing and peak behaviour, while combined interventions can move the illustrative system across the $\mathcal{R}_0 = 1$ threshold. Because all numerical parameters are illustrative rather than estimated from a specific outbreak, the numerical findings should be interpreted as mechanistic demonstrations rather than disease-specific forecasts or empirical intervention-effect estimates.

Keywords: Caputo derivative; fractional-order model; infectious-disease transmission; memory effect; reproduction number; asymptomatic transmission; stability analysis; sensitivity analysis; vaccination; treatment; public awareness; 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 **(Brauer et al., 2019; Heffernan et al., 2005; Hethcote, 2000; Kermack & McKendrick, 1927)**. 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 **(Caputo, 1967; Diethelm, 2010; Kilbas et al., 2006; Podlubny, 1999; Samko et al., 1993)**. 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 **Chen et al. (2021)**; applications include models with vaccination, asymptomatic infection, **hospitalisation**,

and behavioural or treatment controls **(Higazy, 2020; Khan et al., 2020; Mahata et al., 2022; Pan et al., 2021; Rezapour et al., 2020; Rostamy & Mottaghi, 2016; Xu & Hu, 2025)**.

Recent studies continue to use Caputo-based fractional formulations to examine memory-dependent epidemic dynamics, threshold and stability behaviour, intervention effects, and reproducible numerical solution strategies (Calatayud et al., 2024; Jangir et al., 2026; Nwokike et al., 2026; Pathak & Kota, 2026; Thakur et al., 2026).

Accordingly, the methodological gap addressed here is the integration, within one generic fractional-order framework, of symptomatic and asymptomatic transmission, hospitalisation, vaccination, treatment, and public awareness, together with explicit threshold, stability, sensitivity, and reproducible numerical analyses. The framework remains mechanistic rather than disease-specific because its parameters are illustrative.

We develop a generic model that distinguishes symptomatic and asymptomatic infection and includes **hospitalisation**. 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 **(Almeida, 2017)**

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

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

$${}^{C}D_t^{\alpha} x(t) = f(t, x(t)), \qquad x(0) = x_0, \tag{2.5}$$

is equivalent to the Volterra equation

$$x(t) = x_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} f(\tau, x(\tau))\, d\tau. \tag{2.6}$$

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

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

the standard Matignon sector condition **(Li & Zhang, 2011; Matignon, 1996)**.

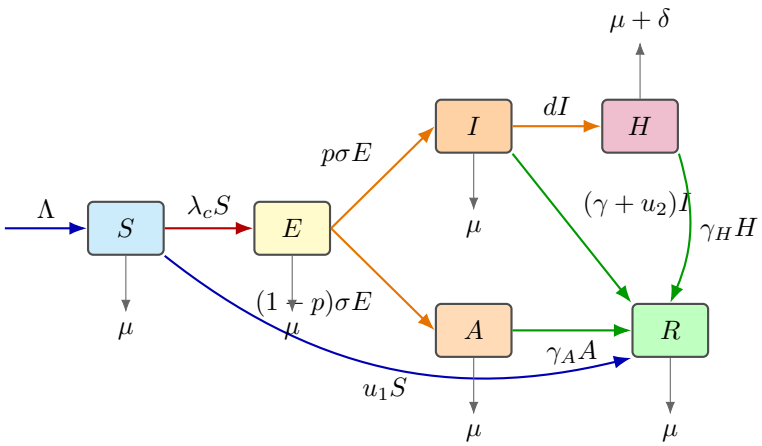

Figure 1: Compartment structure. Awareness u_3 reduces transmission in λ_c, vaccination u_1 transfers susceptible individuals to protection, and treatment u_2 accelerates removal of symptomatic infectious individuals.

3 Model formulation

The total population is divided into susceptible S, exposed E, symptomatic infectious I, asymptomatic infectious A, **hospitalised** 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 Λ. All classes experience natural mortality μ, and **hospitalised** individuals experience additional disease-induced mortality δ. 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 $0 \le u_3 \le 1$.

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 η 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

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

Initial conditions satisfy

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

4 Positivity, boundedness, and well-posedness

Define the epidemiologically feasible region

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

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

Symbol	Meaning	Constraint
Λ	Recruitment into S	> 0
β	Effective transmission rate	> 0
η	Relative infectiousness of asymptomatic cases	≥ 0
μ	Natural mortality rate	> 0
σ	Progression rate from exposure	> 0
p	Fraction progressing to symptomatic infection	$[0, 1]$
$\gamma, \gamma_A, \gamma_H$	Recovery rates from I, A, H	≥ 0
d	Hospitalization rate of symptomatic cases	≥ 0
δ	Disease-induced mortality in H	≥ 0
u_1	Vaccination rate	≥ 0
u_2	Treatment rate of symptomatic cases	≥ 0
u_3	Effectiveness of public awareness	$[0, 1]$
α	Fractional order	$(0, 1]$

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:

$$ {}^{C}D_t^{\alpha} S\Big|_{S=0} = \Lambda, \quad {}^{C}D_t^{\alpha} E\Big|_{E=0} = \lambda_c S, \quad {}^{C}D_t^{\alpha} I\Big|_{I=0} = p\sigma E,$$

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

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

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

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

$$ X(t) = X_0 + \frac{1}{\Gamma(\alpha)} \int_0^t (t-\tau)^{\alpha-1} F(X(\tau))\, d\tau. \tag{4.5}$$

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

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

is continuously differentiable. Hence 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

$$E_0 = \left(\frac{\Lambda}{u_1 + \mu}, 0, 0, 0, 0, \frac{u_1 \Lambda}{\mu(u_1 + \mu)} \right), \qquad N_0^* = \frac{\Lambda}{\mu}. \tag{5.2}$$

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

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

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 **(Diekmann et al., 1990; van den Driessche & Watmough, 2002)**,

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

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 $E^* = (S^*, E^*, I^*, A^*, H^*, R^*)$, define $c_H = dp\sigma/(k_2 k_4)$. 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 $0 < \alpha \leq 1$ 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 $F_I \geq 0$ 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 $0 < \alpha \leq 1$. 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

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

Because $S/N \leq 1$,

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

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 Ω.

Fractional Lyapunov and Mittag–Leffler stability techniques underlying this argument are developed more generally in **Li et al. (2010)**.

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 θ, the **normalised** 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-prioritisation** studies **(Chitnis et al., 2008)**. 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 α means only that α 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
μ	$1/(70 \times 365)$ day^{-1}	Λ	$\mu\, 100000$ day^{-1}	β	0.55 day^{-1}
η	0.55	σ	$1/5.2$ day^{-1}	p	0.65
γ	0.10 day^{-1}	γ_A	0.125 day^{-1}	γ_H	$1/12$ day^{-1}
d	0.025 day^{-1}	δ	0.005 day^{-1}	α	scenario-specific

8 Numerical method and experiments

8.1 Fractional Adams–Bashforth–Moulton scheme

Let $t_n = nh$, $n = 0, \ldots, M$, and write $F_j = F(t_j, X_j)$. The predictor is

$$X_{n+1}^{P} = X_0 + \frac{h^{\alpha}}{\Gamma(\alpha+1)} \sum_{j=0}^{n} \left[(n-j+1)^{\alpha} - (n-j)^{\alpha} \right] F_j. \tag{8.1}$$

The corrector is

$$X_{n+1} = X_0 + \frac{h^{\alpha}}{\Gamma(\alpha+2)} \left[F(t_{n+1}, X_{n+1}^{P}) + \sum_{j=0}^{n} a_{j,n+1} F_j \right], \tag{8.2}$$

where

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

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

This PECE method is a standard **discretisation** of Caputo initial-value problems **(Diethelm et al., 2002)** and has been applied widely in fractional epidemic models **(Mahata et al., 2022; Pan et al., 2021)**. The accompanying `generate_figures.py` script records the implementation used for the reported numerical calculations.

8.2 Illustrative parameterisation

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 $N(0) = 100{,}000$, $X_0 = (99{,}850, 80, 40, 25, 5, 0)$, $T = 180$ 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 α 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, α changes the memory kernel and therefore changes epidemic timing. In applications, α should be estimated jointly with rate parameters; simply varying it while retaining integer-order parameter units is best viewed as an exploratory sensitivity exercise.

10 Limitations

The analysis has three principal limitations. First, the parameterisation is illustrative and therefore cannot support forecasts or policy estimates for a particular pathogen. Second, the assumption of homogeneous mixing does not represent heterogeneity in age, space, or contact networks. Third, constant intervention parameters do not capture changes in uptake, supply, treatment access, or risk perception over time. These limitations indicate that disease-specific calibration, identifiability analysis, time-dependent controls, and structured-population extensions are required before the framework can be used for substantive forecasting or policy assessment.

11 Conclusion

This study developed a six-compartment fractional-order epidemic model that

incorporates symptomatic and asymptomatic infection, hospitalisation, recovery, vaccination, treatment, and public awareness. The model was shown to be positive, bounded, and locally well posed, and its disease-free and endemic equilibria were characterised. The controlled reproduction number separates symptomatic and asymptomatic transmission and provides the principal local threshold: the disease-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$. A stronger sufficient condition was also established for global disease elimination. Sensitivity analysis indicates that transmission and asymptomatic infectiousness increase the reproduction number, whereas vaccination, treatment, recovery, hospitalisation, and awareness reduce it. The numerical experiments further show that fractional order affects transient epidemic timing and peak behaviour, while combined interventions can move the illustrative system below the threshold. Because the parameter values are illustrative, the results support mechanistic interpretation rather than disease-specific prediction. Calibration against an identified outbreak dataset is therefore necessary before 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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