# A Fractional-Order SEIARH Epidemic Model with Vaccination, Treatment, and Public-Awareness Controls


**Abstract**

This paper develops a fractional-order model for an infectious disease in a population divided into susceptible, exposed, symptomatic infectious, asymptomatic infectious, hospitalized, and recovered classes. A Caputo derivative is used to represent the influence of past epidemic states on present transmission and recovery. Vaccination moves susceptible people directly to the recovered class, treatment increases the recovery of symptomatic cases, and public-awareness activity reduces effective contact. We establish non-negativity, boundedness, and well-posedness of the model, derive the disease-free equilibrium and the basic reproduction number, and give threshold conditions for disease elimination. An optimal-control problem balances reductions in symptomatic infection and hospitalization against the costs of the three interventions. An illustrative parameter study shows that the uncontrolled reproduction number exceeds one, whereas treatment or awareness alone can move it below one; the largest reduction is obtained when the controls are combined. The formulation separates what follows analytically from the model from what must ultimately be checked against disease-specific data.



**Keywords:** fractional epidemic model; Caputo derivative; asymptomatic transmission; basic reproduction number; optimal control; public awareness.


## 1 Introduction

Compartmental models offer a transparent way to connect assumptions about infection, clinical progression, and intervention to population-level outcomes. The familiar SIR and SEIR frameworks remain useful because their parameters have direct epidemiological meanings and their thresholds can often be studied analytically [8, 2]. Their usual formulation, however, is local in time: once the current state is known, earlier states have no further influence on the trajectory.

That simplification may be restrictive when present behaviour depends on accumulated experience. Immune response, delays in seeking care, treatment history, and changes in risk perception can all leave effects that persist beyond a single instant. Fractional derivatives provide one way to represent such history dependence. In particular, the Caputo derivative combines a nonlocal memory kernel with initial conditions of the same form used for ordinary differential equations [3, 11, 7].

Two additional features motivate the structure considered here. First, people who remain asymptomatic can continue ordinary social activity and therefore contribute to transmission even when their infectiousness differs from that of symptomatic cases. Second, hospitalization changes both the clinical pathway and the demand placed on health services. A model that merges all infected people into one class cannot describe these distinctions.

We therefore study a fractional SEIARH system with six state variables. Three controls act at different points in the disease pathway: vaccination reduces the susceptible pool, treatment shortens symptomatic infection, and awareness lowers effective contact. The analysis addresses four questions. Are the model states biologically admissible? What threshold determines whether infection can invade? How do the controls alter that threshold? And how can their epidemiological benefits be weighed against implementation costs?

The remainder of the paper introduces the fractional-calculus notation, constructs the model and its flow diagram, establishes its main qualitative properties, derives the threshold quantity, and formulates an optimal-control problem. The final sections give an illustrative threshold analysis, discuss interpretation and limitations, and identify the data needed for disease-specific application.

## 2 Fractional-calculus preliminaries

Let $0 < \alpha \leq 1$. For an absolutely continuous function $x$ on $[0, T]$, its Caputo derivative is

$$ {}^{C}D_t^{\alpha} x(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-s)^{-\alpha} x'(s)\, ds, \qquad 0 < \alpha < 1, \tag{1} $$

where

$$ \Gamma(z) = \int_0^{\infty} e^{-s} s^{z-1}\, ds, \qquad z > 0. \tag{2} $$

At $\alpha = 1$, the operator in (1) reduces to the ordinary first derivative. Its integral form is

$$ x(t) = x(0) + \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} f(s, x(s))\, ds \tag{3} $$

when ${}^{C}D_t^{\alpha} x = f(t, x)$. This Volterra representation is useful for existence and uniqueness arguments [7].

The Mittag–Leffler function,

$$ E_\alpha(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k + 1)}, \tag{4} $$

plays the role occupied by the exponential function in first-order linear systems. For a commensurate linear fractional system ${}^{C}D_t^{\alpha} x = Ax$, Matignon's condition requires each eigenvalue $\lambda_j$ of $A$ to satisfy

$$ |\arg(\lambda_j)| > \frac{\alpha\pi}{2} \tag{5} $$

for asymptotic stability [10].

## 3 Modelling perspective

The model is intended as a general transmission framework rather than a calibrated description of one named infection. Its six classes retain the main clinical stages needed to distinguish hidden transmission, reported illness, and severe disease. The same structure can later be adapted to a particular pathogen by revising the force of infection, progression routes, and parameter interpretation.

Memory is introduced through a common fractional order so that the first analysis remains tractable. This choice does not imply that all biological processes have identical memory. A data-driven extension could assign different orders to different compartments, but the additional flexibility would need to be justified through identifiable improvements in fit or prediction.

The interventions are likewise separated by mechanism. Vaccination changes population immunity, treatment changes the duration of symptomatic infection, and awareness changes contact behaviour. Keeping these actions distinct makes their influence on the reproduction number and objective functional easier to interpret.

# 4 Model construction

## 4.1 Population classes and assumptions

At time $t$, the total living population is

$$N(t) = S(t) + E(t) + I(t) + A(t) + H(t) + R(t). \tag{6}$$

Here $S$ denotes susceptible people, $E$ those who are infected but not yet infectious, $I$ symptomatic infectious cases, $A$ asymptomatic infectious cases, $H$ hospitalized cases, and $R$ recovered or effectively immunized people.

Recruitment enters $S$ at rate $\Lambda$, and natural death acts in every living class at rate $\mu$. Infection is caused by contact with $I$ and $A$. Exposed people leave $E$ at rate $\sigma$; a proportion $p$ develops symptoms, while $1 - p$ remains asymptomatic. Symptomatic cases are hospitalized at rate $\delta$. The baseline recovery rates from $I$, $A$, and $H$ are $\gamma$, $\gamma_A$, and $\gamma_H$, respectively. Disease-induced mortality is confined to $H$ and occurs at rate $d$.

The measurable controls satisfy $0 \leq u_i(t) \leq 1$. Vaccination $u_1$ transfers people from $S$ to $R$, treatment $u_2$ adds to the recovery rate from $I$, and public awareness $u_3$ reduces effective contact. Immunity is assumed not to wane during the period studied. For dimensional consistency in a fractional model, rate parameters may be interpreted as effective $\alpha$-order coefficients; disease-specific calibration should apply one convention consistently.

## 4.2 Compartmental flow diagram

Figure 1 shows every transfer used in the equations. Solid arrows represent movement between living classes. Dashed arrows show removal by natural or disease-induced death. The dotted influence arrows make explicit that both infectious classes contribute to the force of infection, while awareness acts on that transmission pathway.

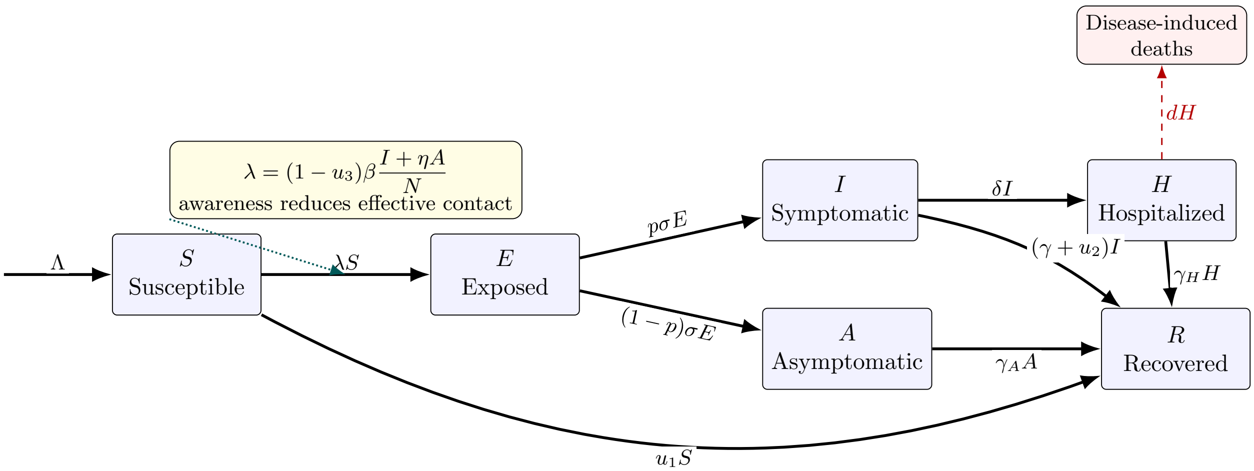


Figure 1: Compartmental flow of the controlled fractional-order SEIARH model. Dotted arrows indicate an influence on transmission rather than movement of people. Natural mortality at rate $\mu$ applies to every living compartment.

### 4.3 Force of infection

The standard-incidence force of infection is

$$\lambda(t) = (1 - u_3(t))\beta\frac{I(t) + \eta A(t)}{N(t)}, \tag{7}$$

where $\beta$ is the effective contact rate and $0 < \eta < 1$ is the infectiousness of an asymptomatic case relative to a symptomatic case. Thus awareness changes exposure risk without moving people directly between compartments.

### 4.4 Governing equations

The diagram leads to the system

$$\begin{aligned}
{}^{C}D_t^{\alpha} S &= \Lambda - \lambda S - u_1 S - \mu S,\\
{}^{C}D_t^{\alpha} E &= \lambda S - (\sigma + \mu)E,\\
{}^{C}D_t^{\alpha} I &= p\sigma E - (\gamma + \delta + \mu + u_2)I,\\
{}^{C}D_t^{\alpha} A &= (1-p)\sigma E - (\gamma_A + \mu)A,\\
{}^{C}D_t^{\alpha} H &= \delta I - (\gamma_H + \mu + d)H,\\
{}^{C}D_t^{\alpha} R &= u_1 S + (\gamma + u_2)I + \gamma_A A + \gamma_H H - \mu R.
\end{aligned} \tag{8}$$

The initial data satisfy $S(0) > 0$ and $E(0), I(0), A(0), H(0), R(0) \geq 0$.

Table 1: Model parameters and controls.

| Symbol | Meaning |
|---|---|
| $\Lambda$ | Recruitment into the susceptible population |
| $\beta$ | Effective transmission coefficient |
| $\eta$ | Relative infectiousness of asymptomatic cases |
| $\mu$ | Natural mortality rate |
| $\sigma$ | Rate of progression out of the exposed class |
| $p$ | Proportion of exposed people who become symptomatic |
| $\delta$ | Hospitalization rate of symptomatic cases |
| $\gamma, \gamma_A, \gamma_H$ | Baseline recovery rates from $I$, $A$, and $H$ |
| $d$ | Disease-induced mortality rate among hospitalized cases |
| $u_1, u_2, u_3$ | Vaccination, treatment, and awareness controls |
| $\alpha$ | Fractional order |

# 5 Qualitative properties

Define

$$\Omega = \left\{ (S, E, I, A, H, R) \in \mathbb{R}^6_+ : N \leq \frac{\Lambda}{\mu} \right\}. \tag{9}$$

**Theorem 5.1** (Non-negativity)**.** *Every solution of* (8) *that begins in the non-negative orthant remains non-negative for as long as it exists.*

*Proof.* On a boundary where one state is zero, its inward production term is non-negative: these terms are $\Lambda$, $\lambda S$, $p\sigma E$, $(1-p)\sigma E$, $\delta I$, and $u_1 S + (\gamma + u_2)I + \gamma_A A + \gamma_H H$, respectively. The generalized mean-value property for Caputo derivatives then prevents a component with non-negative initial data from crossing into negative values. Hence the non-negative orthant is forward invariant. □

**Theorem 5.2** (Boundedness)**.** *If the initial population is finite, then every component of the solution is bounded. In particular,*

$$\limsup_{t\to\infty} N(t) \leq \frac{\Lambda}{\mu}. \tag{10}$$

*Proof.* Adding the six equations gives

$${}^C D_t^\alpha N = \Lambda - \mu N - dH \leq \Lambda - \mu N. \tag{11}$$

Comparison with the corresponding scalar equation yields

$$N(t) \leq N(0)E_\alpha(-\mu t^\alpha) + \frac{\Lambda}{\mu}\big[1 - E_\alpha(-\mu t^\alpha)\big]. \tag{12}$$

Because $E_\alpha(-\mu t^\alpha) \to 0$, the stated upper bound follows. □

**Proposition 5.3** (Existence and uniqueness)**.** *For admissible bounded controls and initial data with* $N(0) > 0$, *system* (8) *has a unique local solution. The non-negativity and population bound extend that solution throughout every finite study interval.*

*Proof.* On any positively invariant set bounded away from $N = 0$, the right-hand side is continuously differentiable in the state variables and therefore locally Lipschitz. Applying the fixed-point theorem to the equivalent Volterra integral system gives local existence and uniqueness. The a priori bounds rule out finite-time escape. □

# 6 Equilibria and transmission threshold

For equilibrium calculations, hold the controls at constant levels. To avoid confusing the recovered state with the reproduction number, equilibrium components are marked by superscripts rather than subscripts.

## 6.1 Disease-free equilibrium

At the disease-free equilibrium $E = I = A = H = 0$. Solving the remaining balance equations gives

$$\mathcal{E}^0 = \left(\frac{\Lambda}{\mu + u_1}, 0, 0, 0, 0, \frac{u_1\Lambda}{\mu(\mu + u_1)}\right), \qquad N^0 = \frac{\Lambda}{\mu}. \tag{13}$$

## 6.2 Basic reproduction number

Take $(E, I, A, H)^T$ as the infected subsystem. The Jacobians of new infection and all other transfers at (13) are

$$F = \begin{pmatrix} 0 & q & q\eta & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \\ 0 & 0 & 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} k_E & 0 & 0 & 0 \\ -p\sigma & k_I & 0 & 0 \\ -(1-p)\sigma & 0 & k_A & 0 \\ 0 & -\delta & 0 & k_H \end{pmatrix}, \tag{14}$$

where

$$q = (1 - u_3)\beta\frac{S^0}{N^0}, \quad k_E = \sigma + \mu, \quad k_I = \gamma + \delta + \mu + u_2, \quad k_A = \gamma_A + \mu, \quad k_H = \gamma_H + \mu + d. \tag{15}$$

The next-generation construction [4, 12] gives

$$\begin{aligned} \mathcal{R}_0 &= \rho(FV^{-1}) \\ &= \frac{(1 - u_3)\beta\mu\sigma}{(mu + u_1)(\sigma + \mu)}\left[\frac{p}{\gamma + \delta + \mu + u_2} + \frac{\eta(1-p)}{\gamma_A + \mu}\right]. \end{aligned} \tag{16}$$

The two terms in brackets are the expected symptomatic and asymptomatic contributions after survival through the exposed stage. Vaccination reduces $S^0/N^0$, treatment shortens symptomatic infectiousness, and awareness scales down both transmission routes.

**Theorem 6.1** (Local threshold)**.** *The disease-free equilibrium is locally asymptotically stable when $\mathcal{R}_0 < 1$ and unstable when $\mathcal{R}_0 > 1$.*

*Proof.* The linearization separates demographic directions from the infected block. The demographic eigenvalues are negative. The next-generation theorem implies that the infected block has eigenvalues with negative real parts when $\rho(FV^{-1}) < 1$, and it has an unstable direction when this spectral radius exceeds one. Negative-real-part eigenvalues also satisfy (5) for $0 < \alpha \leq 1$. □

### 6.3 Endemic equilibrium

Let $\mathcal{E}^* = (S^*, E^*, I^*, A^*, H^*, R^*)$ denote a positive equilibrium and let $\lambda^*$ be its force of infection. Its components satisfy

$$\begin{aligned} S^* &= \frac{\Lambda}{\lambda^* + u_1 + \mu}, & E^* &= \frac{\lambda^* S^*}{\sigma + \mu}, \\ I^* &= \frac{p\sigma E^*}{\gamma + \delta + \mu + u_2}, & A^* &= \frac{(1-p)\sigma E^*}{\gamma_A + \mu}, \\ H^* &= \frac{\delta I^*}{\gamma_H + \mu + d}, & R^* &= \frac{u_1 S^* + (\gamma + u_2) I^* + \gamma_A A^* + \gamma_H H^*}{\mu}. \end{aligned} \tag{17}$$

Together with $\lambda^* = (1 - u_3)\beta(I^* + \eta A^*)/N^*$, these relations reduce the equilibrium problem to one scalar consistency equation. A positive root corresponds to endemic persistence. The threshold $\mathcal{R}_0 = 1$ identifies the point at which infection can invade the disease-free state; uniqueness of an endemic root should be checked from that scalar equation rather than assumed without proof.

### 6.4 A sufficient global-elimination condition

The vaccination-dependent expression (16) is a local invasion threshold. A convenient global result follows from the stronger quantity

$$\mathcal{R}_g = \frac{(1-u_3)\beta\sigma}{\sigma + \mu}\left[\frac{p}{\gamma + \delta + \mu + u_2} + \frac{\eta(1-p)}{\gamma_A + \mu}\right]. \tag{18}$$

**Theorem 6.2** (Sufficient condition for global elimination)**.** *If $\mathcal{R}_g < 1$, then $E(t)$, $I(t)$, and $A(t)$ approach zero; consequently $H(t)$ also approaches zero.*

*Proof.* Write $c = (1 - u_3)\beta$ and consider

$$V = E + \frac{c}{k_I} I + \frac{c\eta}{k_A} A. \tag{19}$$

Because $S/N \le 1$, substitution of (8) gives

$$^{C}D_t^{\alpha} V \le (\sigma + \mu)(\mathcal{R}_g - 1)E. \tag{20}$$

Thus $V$ is non-increasing when $\mathcal{R}_g < 1$, and the only invariant set on which its fractional derivative can vanish has $E = I = A = 0$. The hospitalized equation is then a stable linear equation with vanishing input, so $H \to 0$. $\square$

# 7 Optimal-control formulation

Let the intervention period be $[0, T]$. We seek controls in

$$\mathcal{U} = \{(u_1, u_2, u_3) : u_i \text{ measurable and } 0 \le u_i(t) \le 1\}. \tag{21}$$

The objective functional is

$$J(u_1, u_2, u_3) = \int_0^T \left[ A_1 I + A_2 H + \frac{B_1}{2} u_1^2 + \frac{B_2}{2} u_2^2 + \frac{B_3}{2} u_3^2 \right] dt, \tag{22}$$

where $A_1, A_2 > 0$ value reductions in symptomatic infection and hospitalization, and $B_i > 0$ measure intervention cost or effort. Quadratic costs discourage unrealistically abrupt use of maximal controls.

Let $x = (S, E, I, A, H, R)^T$, let $f(x, u)$ denote the right-hand side of (8), and introduce costates $\psi = (\psi_1, \dots, \psi_6)^T$. The Hamiltonian is

$$\mathcal{H} = A_1 I + A_2 H + \frac{1}{2} \sum_{i=1}^{3} B_i u_i^2 + \psi^T f(x, u). \tag{23}$$

For a left-Caputo state equation, the fractional integration-by-parts formula produces a right-sided adjoint operator. In compact form, the necessary conditions are

$${}_tD_T^{\alpha} \psi(t) = \frac{\partial \mathcal{H}}{\partial x}(x^*, u^*, \psi), \tag{24}$$

with the corresponding zero terminal transversality condition for a free terminal state [1]. Using a left-sided Caputo derivative for both the forward state and backward costate equations would omit this distinction.

To retain the dependence of standard incidence on every population class, define

$$g(x, u_3) = (1 - u_3) \beta \frac{S(I + \eta A)}{N}. \tag{25}$$

The infection contribution to the Hamiltonian is $(\psi_2 - \psi_1) g$. Its state derivatives are

$$\begin{aligned}
g_S &= (1 - u_3)\beta \frac{(I + \eta A)(N - S)}{N^2}, & g_E &= -(1 - u_3)\beta \frac{S(I + \eta A)}{N^2}, \\
g_I &= (1 - u_3)\beta \frac{S[N - (I + \eta A)]}{N^2}, & g_A &= (1 - u_3)\beta \frac{S[\eta N - (I + \eta A)]}{N^2}, \\
g_H &= g_R = -(1 - u_3)\beta \frac{S(I + \eta A)}{N^2}.
\end{aligned} \tag{26}$$

Equations (24)–(26), together with the linear transition terms in (8), specify the complete costate system without treating $N$ as a constant.

Pointwise minimization of (23) gives the projected controls

$$\begin{aligned}
u_1^* &= \Pi_{[0,1]} \left( \frac{S(\psi_1 - \psi_6)}{B_1} \right), \\
u_2^* &= \Pi_{[0,1]} \left( \frac{I(\psi_3 - \psi_6)}{B_2} \right), \\
u_3^* &= \Pi_{[0,1]} \left( \frac{(\psi_2 - \psi_1)\beta S(I + \eta A)}{B_3 N} \right),
\end{aligned} \tag{27}$$

where $\Pi_{[0,1]}(z) = \min\{1, \max\{0, z\}\}$. The state equations are advanced from the initial data, the right-sided costate equations are solved from the terminal condition, and the controls are updated from (27) until the iteration converges. Convexity of the running cost and compactness of the control set support existence of an optimal triple under the regularity already established [9].

## 8 Illustrative threshold analysis

The numerical values in Table 2 are retained as an illustrative, dimensionless parameter set. They are not estimates for a named disease and should not be interpreted as a fitted public-health forecast.

Table 2: Illustrative parameter values.

| $\Lambda$ | $\mu$ | $\beta$ | $\eta$ | $\sigma$ | $p$ | $\gamma$ | $\gamma_A$ | $\gamma_H$ | $\delta$ | $d$ | $u_1$ | $u_2$ | $u_3$ |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| 100 | .02 | .60 | .45 | .30 | .70 | .18 | .22 | .15 | .12 | .05 | .50 | .60 | .45 |

For these values, the uncontrolled threshold is $\mathcal{R}_0 = 1.547$. Table 3 evaluates (16) after one or more controls are switched on. The calculations isolate the structural effect of each intervention; they do not replace time-dependent optimal-control simulations.

Table 3: Reproduction number under constant-control scenarios.

| Scenario | $u_1$ | $u_2$ | $u_3$ | $\mathcal{R}_0$ |
|---|---|---|---|---|
| No intervention | 0 | 0 | 0 | 1.547 |
| Vaccination only | .50 | 0 | 0 | 0.059 |
| Treatment only | 0 | .60 | 0 | 0.744 |
| Awareness only | 0 | 0 | .45 | 0.851 |
| Combined controls | .50 | .60 | .45 | 0.016 |

The example yields three immediate observations. First, transmission is self-sustaining without intervention because $\mathcal{R}_0 > 1$. Second, each stated control level moves the local invasion threshold below one, although the strength of the effect depends on its assumed scale and unit. Third, acting at several points in the pathway produces the lowest threshold because vaccination changes susceptibility, treatment shortens symptomatic infectiousness, and awareness reduces exposure from both infectious classes.

For trajectory calculations, one may use the fractional Adams–Bashforth–Moulton predictor–corrector method [5, 6]. A defensible empirical application should report the time grid, convergence tolerance, fractional-order convention, initial conditions, and uncertainty analysis. It should also estimate parameters from data instead of choosing them solely to illustrate qualitative behaviour.

## 9 Discussion

The model makes the roles of asymptomatic infection and hospitalization visible without losing the threshold structure of an SEIR-type system. The decomposition in (16) is especially informative: one term measures infections arising through the symptomatic pathway and the other measures those arising through the asymptomatic pathway. A smaller value of $\eta$ does not make asymptomatic transmission negligible if the asymptomatic proportion or duration is large.

The fractional order changes transient behaviour rather than the algebraic location of equilibria. Values below one give greater weight to past states through the memory kernel in (1). Depending on calibration, this can delay peaks or lengthen epidemic tails. Such effects should be inferred from data, not interpreted automatically as proof that a fractional model is superior to an integer-order model.

The control analysis also highlights a practical distinction. Vaccination and treatment move people between disease states, whereas awareness modifies a behavioural transmission parameter. Their cost weights therefore cannot be compared meaningfully until a common economic or operational scale is chosen. The optimal profiles in (27) are mathematical recommendations conditional on those weights, the parameter estimates, and the assumed intervention bounds.

Several limitations remain. The system is homogeneous and has no age, spatial, or risk structure. Hospital transmission, waning immunity, reinfection, and imperfect vaccination are excluded. Recruitment and mortality are constant, and the fractional order is shared by all compartments. Finally, the illustrative values in Table 2 are not linked to observations. These limitations define clear directions for extension but also constrain the claims that can be made from the present formulation.

## 10 Conclusion

We formulated a fractional SEIARH model in which symptomatic and asymptomatic infection feed a common force of infection, hospitalization represents severe disease, and three controls act through distinct mechanisms. The model preserves non-negative, bounded states and admits a well-defined disease-free equilibrium. Its reproduction number separates the two infectious pathways and shows directly how vaccination, treatment, and awareness alter invasion risk. A stronger Lyapunov threshold supplies a sufficient condition for global elimination, while the optimal-control formulation balances disease burden against intervention effort and correctly retains the population dependence of standard incidence.

The principal policy message is conditional but clear: coordinated measures can outperform a single intervention because they interrupt different parts of the transmission process. Before the framework is used for decision-making, its parameters, fractional order, initial conditions, and cost weights must be estimated and validated for a specific disease and population.

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