Mathematical Analysis of a Generalized Fractional-Order Disease Model with Adaptive Immune Response

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Abstract: In this paper we develop and analyze a generalized Caputo fractional-order SEIR epidemic model including the effect of humoral and cellular adaptive immunity (the SEIR-HCI model). The model represents an autonomous system of dimension six that is determined by Caputo derivatives with orders $\alpha \in (0;1]$, which describe the time-dependent memory in the epidemic process. A detailed mathematical study has been carried out. In particular it is shown how solutions exist uniquely and are non-negative and uniformly bounded. The two equilibria are calculated analytically as well as the basic reproduction number R_0 using the next generation matrix method. It turns out that R_0 does not depend on the order of the derivative. The local asymptotic stability of the disease free equilibrium is investigated according to Matignon's fractional stability criterion. For the global asymptotic stability of the equilibria, suitable Lyapunov functions have been constructed. Furthermore, we show Ulam–Hyers stability and thus guarantee the robustness of all numerical methods that approximate the solution. Finally, simulations show that reducing α leads to a delay in the peak of the epidemic but has no influence on the final size of the outbreak. Additionally, a heatmap analysis of R_0 demonstrates the dependence of the basic reproduction number from several parameters.

Keywords: ***Caputo fractional derivative; SEIR epidemic model; adaptive immune response; basic reproduction number; next-generation matrix; Lyapunov stability; Ulam–Hyers stability; Mittag–Leffler function; disease-free equilibrium; endemic equilibrium.***

I. INTRODUCTION

Global public health faces significant challenges due to infectious disease; millions die each year, and there are large economic costs to families throughout the world [1]. Mathematical modeling of the spread of disease has become one of the few tools available to understand how pathogens are transmitted, predict future outbreaks, and develop evidence based interventions [2, 3]. The classic SIR model developed by Kermack & McKendrick [2] along with several of its variations have provided much insight into the process of disease spread. However, both are formulated using ordinary differential equations (ODEs). These ODEs are based upon integer order derivatives that imply Markovian or "memory less" dynamics.

There is increasing recognition that this may be an unrealistic assumption for many biological systems including those involving infectious disease. Biological systems such as those involved in epidemiology often involve complex interactions and time scales. Thus, the use of ODEs may not capture all of the complexity associated with many real-world systems. Fractional order differential equations (FODEs) represent an extension of ODEs where instead of using integer order derivatives, the system is represented by using fractional order derivatives. Fractional order derivatives are defined using non-integer order operators, thus representing the entire history of the system state via a convolution operator [6, 7]. As such, FODEs are ideally suited for modeling epidemiological processes where the current transmission rate is determined by factors such as the current state of the population and their collective past exposures [5, 10]. Of the various definitions used for fractional derivatives, the Caputo definition is preferred for applications since it retains the concept of initial conditions and approaches the standard derivative when α approaches unity [4, 6].

While often neglected in mathematical models of epidemics, the role of the adaptive immune response is critical to understanding how populations recover from infections. In humans, there are two layers of

defense against pathogens: innate immunity provides immediate, non-specific protection against pathogens while adaptive immunity confers long-term, specific immunity through the coordinated actions of B cells and T cells that produce antibodies and activate cytotoxic T cells (CTLs), respectively. When models fail to account for the dynamics of adaptive immunity, they can lead to inaccurate representations of recovery from infections, overestimate or underestimate the fraction of a population that will be immune after an outbreak and lead to suboptimal recommendations for enhancing immune function.

The motivation for this research is to develop a rigorous, mathematically-founded fractional epidemic model that includes components for immune compartment. The principal contributions of this study are as follows:

- A generalized Caputo fractional-order SEIR-HCI model with humoral (H) and cellular (C) immune-response compartments, yielding a six-dimensional autonomous fractional system.
- Rigorous proofs of existence, uniqueness, positivity, boundedness, local and global Lyapunov stability, and Ulam–Hyers stability for the proposed model.
- A closed-form expression for R_0 via the next-generation matrix, with a complete proof that R_0 is independent of the fractional order α.
- Numerical demonstrations showing how α modulates epidemic transient dynamics without altering equilibrium thresholds.

Section 2 presents the fractional calculus preliminaries. Section 3 formulates the SEIR-HCI model used in this study. Sections 4–5 establish the existence, uniqueness, positivity, and boundedness of the solution. Sections 6–7 analyze the equilibria and R_0. Sections 8–9 present the stability results. Section 10 presents the numerical illustrations. Section 11 concludes the paper.

II. FRACTIONAL CALCULUS PRELIMINARIES

Definition 2.1 *(Riemann–Liouville Fractional Integral).* Let $\alpha > 0$ and $f \in L^1([0,T],\mathbb{R})$. The Riemann–Liouville fractional integral of order α is

$$I_{0,t}^{\alpha} f(t) = \frac{1}{\Gamma(\alpha)} \int_0^t (t-s)^{\alpha-1} f(s) ds, \; t > 0. \quad (1)$$

Following the standard definition [7], this operator is used throughout the fractional preliminaries.

Definition 2.2 *(Caputo Fractional Derivative).* Let $n-1 < \alpha \le n$, $n \in \mathbb{N}$, and $f \in AC^n([0,T],\mathbb{R})$. The Caputo fractional derivative of order α is

$$^{c}D_{0,t}^{\alpha} f(t) = \frac{1}{\Gamma(n-\alpha)} \int_0^t (t-s)^{n-\alpha-1} f^{(n)}(s)\, ds, \quad (2)$$

For $0 < \alpha \le 1$, $^{c}D_{0,t}^{\alpha} f(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t (t-s)^{-\alpha} f'(s)\, ds$ [4,6]

Remark 2.3. The Caputo derivative satisfies $^{c}D_{0,t}^{\alpha} c = 0$ for any constant c, and reduces to f'(t) as $\alpha \to 1^{-}$.

Definition 2.4 *(Mittag–Leffler Function).*

$$E_a(z) = \sum_{k=1}^{\infty} \left(\frac{z^k}{\Gamma(\alpha k+1)}\right) E_{\alpha,\beta}(z) = \sum_{k=0}^{\infty} \frac{z^k}{\Gamma(\alpha k+\beta)}, \quad \alpha, \beta > 0. \quad (3)$$

This follow the classical Mittag –Leffler definition [17].

Lemma 2.5 *(Generalized Gronwall Inequality).* Let $\alpha > 0$, a(t) nonnegative nondecreasing on [0,T] , $b > 0$ constant, and $u \in C([0,T],\mathbb{R}+$ satisfy $u(t) \le a(t) + b\int_0^t (t-s)^{\alpha-1} u(s)\, ds$. Then $u(t) \le a(t)\, E_{\alpha}(b\, \Gamma(\alpha)\, t^{\alpha})$ [15].

Lemma 2.6 *(Fractional Comparison Principle).* If $^{c}D_{0,t}^{\alpha}$ $x(t) \ge 0$ for all $t > 0$ and $x(0) \ge 0$, then $x(t) \ge 0$ for all $t \ge 0$ [16].

Theorem 2.7 *(Existence and Uniqueness via Fixed Point).* Consider the Caputo fractional IVP $^{c}D_{0,t}^{\alpha}$ $x(t) = f(t, x(t))$, $x(0) = x_0$, $0 < \alpha \le 1$. If $f : [0,T] \times \mathbb{R}^n \to \mathbb{R}^n$ is continuous and Lipschitz in x uniformly in t, then there exists a unique solution $x \in C([0,T], \mathbb{R}^n)$ [7].

Definition 2.8 *(Caputo Derivative of a Lyapunov Function).* Let $V: \mathbb{R}^n \to \mathbb{R}_+$ be C^1. Along the trajectories of $^{c}D_{0,t}^{\alpha}$ $x = f(t, x)$:

$$^{c}D_{0,t}^{\alpha} \; V(x(t)) \le \nabla V(x(t))^{T} f(t, x(t)). \quad (4)$$

This inequality is used in the sense of the fractional Lyapunov theory [12].

III. MODEL FORMULATION

3.1. Compartmental Structure

We partitioned the total human population N(t) into six compartments:

- S(t): Susceptible individuals.
- E(t): Exposed individuals (infected, not yet infectious).
- I(t): Infectious individuals (symptomatic).
- R(t): Recovered individuals (temporarily immune).
- H(t): Humoral immune response level (circulating antibody concentration).
- C(t): Cellular immune response level (active CTL density).

The total population satisfies N(t) = S(t) + E(t) + I(t) + R(t).

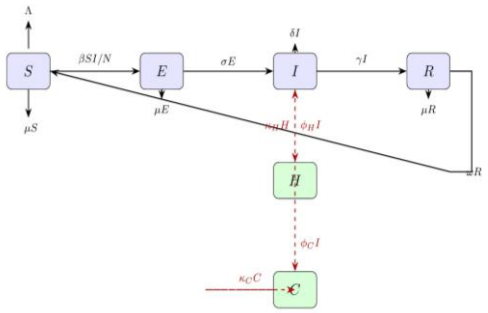

Figure 1: *Compartmental flow diagram of the SEIR-HCI model. Solid arrows represent population flows; dashed red arrows represent immune-response interactions.*

3.2. Model Assumptions

Assumption 3.1. The total population is recruited at a constant rate $\Lambda > 0$ and individuals die naturally at a rate $\mu > 0$.

Assumption 3.2. Disease transmission follows standard incidence $\beta SI/N$, with $\beta > 0$ being the effective contact rate.

Assumption 3.3. Humoral immunity (H) is stimulated by infected cells at a rate $\varphi_H > 0$ and suppresses infection at a rate $k_H > 0$; cellular immunity (C) is stimulated at a rate $\varphi_C > 0$ and suppresses infection at a rate $k_C > 0$. Both decay at rates d_H, d_C , > 0 .

Assumption 3.4. Recovered individuals lose immunity and return to the susceptible class at a rate $\omega \geq 0$.

Assumption 3.5. The fractional order $\alpha \in (0,1]$ is uniform across all the compartments.

3.3. SEIR-HCI Fractional System

Under Assumptions 3.1–3.5, the Caputo fractional-order SEIR-HCI model is:

$$^{c}D^{\alpha}_{0,t}\ S = \Lambda - \frac{\beta SI}{N} - \mu S + \omega R\,, \quad (5a)$$

$$^{c}D^{\alpha}_{0,t}\ E = \frac{\beta SI}{N} - (\sigma+\mu)E\,, \quad (5b)$$

$$^{c}D^{\alpha}_{0,t}\ I = \sigma E - (\gamma+\mu+\delta)I - k_H\, H\, I - k_C\, C\, I\,, \quad (5c)$$

$$^{c}D^{\alpha}_{0,t}\ R = \gamma I - (\mu+\omega)R\,, \quad (5d)$$

$$^{c}D^{\alpha}_{0,t}\ H = \varphi_H\ I - d_H H\,, \quad (5e)$$

$$^{c}D^{\alpha}_{0,t}\ t\, C = \varphi_C\ I - d_C\ C\ , \quad (5f)$$

with initial conditions $S(0) = S_0 > 0$, $E(0) = E_0 \geq 0$, $I(0) = I_0 > 0$, $R(0) = R_0 \geq 0$, $H(0) = H_0 \geq 0$, $C(0) = C_0 \geq 0$.

3.4. Effective Infection Rate with Immune Suppression

The net removal rate of infectious individuals is the immune-modulated rate:

$$\mu_I^{eff}(H, C) = \gamma + \mu + \delta + k_H\ H + k_C\ C, \quad (7)$$

which increases with immune activity, indicating that stronger immune responses accelerate recovery.

3.5. Parameter Description

Table 1: *Model parameters, biological interpretations, units, and baseline values.*

Parameter	Biological Meaning	Unit	Baseline
Λ	Recruitment rate into susceptible class	persons day^{-1}	100
β	Effective contact/transmission rate	day^{-1}	0.35
σ	Progression rate from exposed to infectious	day^{-1}	0.20
γ	Recovery rate of infectious individuals	day^{-1}	0.10

δ	Disease-induced mortality rate	day⁻¹	0.005
μ	Natural mortality rate	day⁻¹	0.0001
ω	Rate of waning immunity	day⁻¹	0.005
φ_H	Humoral immune stimulation rate	day⁻¹	0.08
φ_C	Cellular immune stimulation rate	day⁻¹	0.06
d_H	Humoral immune decay rate	day⁻¹	0.05
d_C	Cellular immune decay rate	day⁻¹	0.04
K_H	Antibody-mediated clearance rate	(persons day)⁻¹	0.03
K_C	CTL-mediated clearance rate	(persons day)⁻¹	0.025
α	Fractional order (memory index)	dimensionless	0.90

IV. EXISTENCE AND UNIQUENESS

Let x = (S, E,I,R,H,C)$(S, E, I, R, H, C)^T$ and F(t , x) denotes the right – hand side of (5), respectively.

Theorem 4.1 *(Existence and Uniqueness).* Let Ω = {x ∈ $\mathbb{R}^6$: ‖x‖ ≤ M} for some M > 0. The system (5) with initial conditions (6) has a unique solution x ∈ C ([0, T], Ω) for any T > 0.

Proof. Each component F_i is polynomial or rational in x. Since S (0) > 0 and I (0) > 0, we have N (0) > 0. By positivity (Theorem 5.1 below) and the continuity of solutions of the fractional IVP, N(t) ≥ N_{min} > 0 on [0,T] for some N_{min} depending on M and initial data. Hence, F is continuous on [0, T] × Ω. For any x, x̃ ∈ Ω, using N ≥ N_{min} ,

$$|F_1(X) - F(\bar{X})| \le \beta \left|\frac{SI}{N} - \frac{\bar{S}\bar{I}}{N}\right| + \mu|S - \bar{S}| + \omega|R - \bar{R}| \le K_1\left||X - \bar{X}\right||.$$

Components $F_2, \dots\dots, F_6$ are linear or bilinear in x, yielding Lipschitz constants $K_2, \dots\dots, K_6$,. With the global Lipschitz constant K = $\max_i K_i$, Theorem 2.7 guarantees a unique solution.

V. POSITIVITY AND BOUNDEDNESS

Theorem 5.1 *(Positivity).* If all initial conditions are non-negative, the solution of (5) remains non-negative for all t ≥ 0.

Proof. By applying Lemma 2.6 at each boundary, we obtain ${}^cD^{\alpha}_{0,t}$ S$|_{S=0}$= Λ + ωR ≥ 0; ${}^cD^{\alpha}_{0,t}$ E$|_{E=0}$ = βSI/N ≥ 0; ${}^cD^{\alpha}_{0,t}$ I$|_{I=0}$= σE ≥ 0; ${}^cD^{\alpha}_{0,t}$ R$|_{R=0}$= γI ≥ 0; ${}^cD^{\alpha}_{0,t}$ H$|_{H=0}$ = φ_H I ≥ 0; ${}^cD^{\alpha}_{0,t}$ C$|_{C=0}$= φ_c I ≥ 0. Hence, each state variable remains non-negative.

Theorem 5.2 *(Boundedness).* All solutions of (5) initiating in $\mathbb{R}^6_+$ are uniformly bounded. The biologically feasible region

$$\Xi = \{(S,E,I,R,H,C) \in \mathbb{R}^6_+ : N \le \Lambda/\mu,\ H \le \varphi_H\ \Lambda/(\mu d_H),\ C \le \varphi_C\Lambda/(\mu d_C),)\} \quad (8)$$

is positively invariant and attractive.

Proof. Adding Equations (5a)–(5d), we obtain: ${}^cD^{\alpha}_{0,t}$ N = Λ − μN − δI ≤ Λ − μN. By the fractional comparison principle, N(t) ≤ y(t) where y solves ${}^cD^{\alpha}_{0,t}$ y = Λ − μy.

The Mittag–Leffler solution is

y(t) = N (0) E_{α} $(-\mu t^{\alpha})$ + (Λ/μ)(1 − $E_{\alpha}(-\mu t^{\alpha})$).

Since E_{α} $(-\mu t^{\alpha}))$ → 0 as t → ∞, we obtain lim $sup_{t\to\infty}$ N(t) ≤ Λ/μ. From (5e), we have ${}^cD^{\alpha}_{0,t}$ H ≤ φ_H (Λ/μ) − d_H H, giving lim sup H(t) ≤ $\varphi_H\Lambda/(\mu d_H)$. Similarly for C. Hence, Ξ is positively invariant and attracting.

VI. EQUILIBRIUM ANALYSIS

6.1. Disease-Free Equilibrium

Setting E = I = 0, the disease-free equilibrium (DFE) is:

$$\mathscr{E}^0 = (S^0, 0, 0, 0, 0, 0), \quad S^0 = \Lambda/\mu. \quad (9)$$

6.2. Endemic Equilibrium

At the endemic equilibrium $\mathscr{E}^* = (S^*, E^*, I^*, R^*, H^*, C^*)$ with $I^* > 0$:

$$H^* = \varphi_H I^*/d_H, \quad C^* = \varphi_C I^*/d_C, \quad R^* = \gamma I^*/(\mu+\omega). \quad (10)$$

Setting (5c) to zero with the immune-modulated removal rate, we obtain:

$$E^* = \Phi(I^*) I^*/\sigma, \quad \Phi(I^*) = (\gamma+\mu+\delta) + \chi I^*, \quad \chi = K_H\varphi_H/d_H + K_C\varphi_C/d_C > 0. \quad (11)$$

Proposition 6.1. System (5) has (i) a unique DFE $\mathscr{E}^0$ for all parameter values; (ii) a unique endemic equilibrium $\mathscr{E}^*$ (with $I^* > 0$) if and only if $R_0 > 1$.

Proof. Part (i) is immediate. Part (ii) follows by defining $G(I) = R_0 \cdot (\gamma+\mu+\delta)/\Phi(I) \cdot S(I)/N(I) - 1$, showing $G(0) = R_0 - 1 > 0$ when $R_0 > 1$, $G \to -\infty$ as $I \to I^-_{max}$, and proving G is strictly decreasing (both factors $\frac{\gamma+\mu+\delta}{\Phi(I)}$ *and* $\frac{S(I)}{N(I)}$ are strictly decreasing in I). By the Intermediate Value Theorem, a unique zero $I^* \in (0, I_{max})$ exists. When $R_0 \le 1$, $G(0) \le 0$ everywhere, and no endemic equilibrium exists.

VII. BASIC REPRODUCTION NUMBER

We compute R_0 using the next-generation matrix (NGM) method [11]. The infected compartments are $X_I = (E, I)^T$ (the immune compartments H, C vanish at $\mathscr{E}^0$ but carry no new-infection flux and belong to the transition operator). The new-infection and transition matrices at $\mathscr{E}^0$ are:

$$F = \begin{pmatrix} 0 & \beta \\ 0 & 0 \end{pmatrix}, \quad V = \begin{pmatrix} \sigma+\mu & 0 \\ -\sigma & \gamma+\mu+\delta \end{pmatrix}. \quad (12)$$

Theorem 7.1 *(Basic Reproduction Number).* The basic reproduction number of system (5) is:

$$R_0 = \rho(FV^{-1}) = \beta\sigma / [(\sigma+\mu)(\gamma+\mu+\delta)]. \quad (13)$$

Proof. The characteristic polynomial of FV^{-1} is $\lambda(\lambda - R_0) = 0$, which gives $\rho(FV^{-1}) = R_0$.

Remark 7.2. The expression (13) is independent of α; the fractional order governs transient dynamics but not the threshold condition. The immune parameters $\varphi_H, \varphi_C, K_H, K_C, d_H d_C$ do not appear in R_0 because $H^* = C^* = 0$ at $\mathscr{E}^0$; they significantly modulate the endemic equilibrium $\mathscr{E}^*$ through $\Phi(I^*)$.

Remark 7.3 *(Epidemiological Factorisation).*

$$R_0 = [\beta/(\gamma+\mu+\delta)] \times [\sigma/(\sigma+\mu)],$$

where the two factors represent secondary infections per infectious period and the probability of survival through the latency period, respectively.

Figure (2) shows a heatmap of R_0 as a function of β and γ.

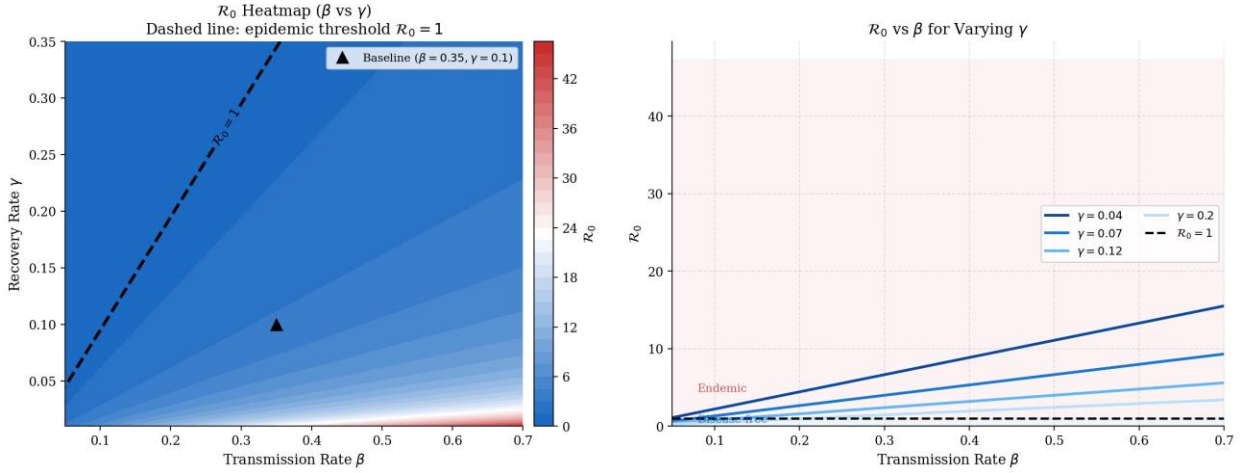

Figure 2: *Heatmap of $\mathfrak{R}_0$ as a function of transmission rate β and recovery rate γ. The dashed contour marks the epidemic threshold $\mathfrak{R}_0 = 1$. Right panel shows $\mathfrak{R}_0$ vs. β for varying γ; increasing γ (via treatment) drives $\mathfrak{R}_0$ below unity.*

VIII. STABILITY ANALYSIS

8.1. Local Stability of the Disease-Free Equilibrium

Theorem 8.1 *(Local Stability of DFE).* The DFE $\mathscr{E}^0$ is locally asymptotically stable (LAS) if $R_0 < 1$ and unstable if $R_0 > 1$.

Proof. The Jacobian at $\mathscr{E}^0$ is the 6×6 matrix $J(\mathscr{E}^0)$

$$J(\mathcal{E}^0) = \begin{pmatrix} -\mu & 0 & -\beta & \omega & 0 & 0 \\ 0 & -(\sigma+\mu) & \beta & 0 & 0 & 0 \\ 0 & \sigma & -(\gamma+\mu+\delta) & 0 & 0 & 0 \\ 0 & 0 & \gamma & -(\mu+\omega) & 0 & 0 \\ 0 & 0 & \phi_H & 0 & -d_H & 0 \\ 0 & 0 & \phi_C & 0 & 0 & -d_C \end{pmatrix}. \quad (14)$$

The characteristic polynomial factors as $p(\lambda) = p_1(\lambda) \cdot p_2(\lambda)$ where:

$$p_1(\lambda) = (\lambda+\mu)(\lambda+\mu+\omega)(\lambda+d_H)(\lambda+d_C), \quad (15)$$

$$p_2(\lambda) = \lambda^2 + [(\sigma+\mu)+(\gamma+\mu+\delta)]\lambda + (\sigma+\mu)(\gamma+\mu+\delta)(1-R_0). \quad (16)$$

All roots of p_1 are real and negative. For p_2: when $R_0 < 1$, both roots have strictly negative real parts and satisfy Matignon's stability criterion $|\arg(\lambda_i)| > \alpha\pi/2$ [20, 21]. When $R_0 > 1$, p_2 has a root with a positive real part, yielding instability.

8.2. Lyapunov Global Stability

Lemma 8.2 *(Volterra–Lyapunov Term).* Let $g(x) = x - 1 - \ln x \geq 0$ for $x > 0$. For $u(t) > 0$ with equilibrium $u^* > 0$ [12]: ${}^cD^{\alpha}_{0,t}\ [u^* g(u/u^*)] \leq (1 - u^*/u)\ {}^cD^{\alpha}_{0,t}\ u.$ (17)

Theorem 8.3 *(Global Stability of DFE).* If $R_0 \leq 1$, the DFE $\mathscr{E}^0$ is globally asymptotically stable (GAS) in Ξ.

Proof. Define $V_0 = (\sigma / (\sigma+\mu))E + I$. Because V_0 is linear, equality holds in (4) as follows:

${}^cD^{\alpha}_{0,t}\ V_0 = \sigma\beta SI/[(\sigma+\mu)N] - (\gamma+\mu+\delta)\, I - K_H\ HI - K_C\ CI.$

Using $S/N \leq 1$ and dropping non-negative immune terms: ${}^cD^{\alpha}_{0,t}\ V_0 \leq (\gamma+\mu+\delta)(R_0-1)I \leq 0$ when $R_0 \leq 1$. By LaSalle's invariance principle for Caputo systems [18], $\mathscr{E}^0$ is GAS.

Theorem 8.4 *(Global Stability of Endemic Equilibrium).* If $R_0 > 1$, the endemic equilibrium $\mathscr{E}^*$ is GAS in $\Xi \setminus \{E=I=0\}$.

Proof. Define the Volterra-type Lyapunov function $V^* = S^* g(S/S^*) + E^* g(E/E^*) + I^* g(I/I^*) + R^* g(R/R^*) + (K_H\ S^*/2\varphi_H\ H-H^*)^2 + (K_C\ S^*/2\varphi_C\varphi C)(C-C^*)^2$. (18).

Applying Lemma 8.2 and substituting the model equations and endemic equilibrium conditions, one obtains after collecting terms via the AM–GM inequality that ${}^cD^{\alpha}_{0,t}\ V^* \leq -\frac{\mu(S-S^*)^2}{S} - \frac{K_H d_H S^*}{\varphi_H}(H - H^*)^2 - \frac{K_C d_C S^*}{\varphi_C}(C-C^*)^2 - (\sigma+\mu)E^*\left[g\left(\frac{S^*}{S}\right) + g\left(\frac{SIN^*}{S^*I^*N}\right) + g\left(\frac{E^*I}{EI^*}\right)\right] - \gamma I^*\left[g\left(\frac{I}{I^*}\right) + g\left(\frac{IR^*}{I^*R}\right)\right] - \frac{XI^*(I-I^*)}{I} \leq 0.$ (19). Every term is non-positive ($g\left(g \geq 0, \mu, \frac{K_H d_H S^*}{\varphi_H}, \frac{K_C d_C S^*}{\varphi_C} > 0\right)$. . Equality holds iff $x = \mathscr{E}^*$. By LaSalle's principle [18], all solutions converge to $\mathscr{E}^*$.

IX. ULAM–HYERS STABILITY

Definition 9.1 *(Ulam–Hyers Stability).* System (5) is Ulam–Hyers (UH) stable if there exists $\theta > 0$ such that for every $\varepsilon > 0$ and every ε-approximate solution $\tilde{x}$ (satisfying $\| {}^cD^{\alpha}_{0,t}\ \tilde{x} - F(t,\tilde{x})\| \leq \varepsilon$), there exists an exact solution x with $\|\tilde{x}(t) - x(t)\| \leq \theta\varepsilon$ for all $t \in [0,T]$.

Theorem 9.2 *(Ulam–Hyers Stability).* If F is globally Lipschitz with constant K and $\frac{KT^{\alpha}}{\Gamma(\alpha+1)} < 1$, (20), then system (5) is UH stable with

$$\theta = \frac{\frac{T^{\alpha}}{\Gamma(\alpha+1)}}{1 - KT^{\alpha}/\Gamma(\alpha+1)}. \quad (21)$$

Proof. Converting the Caputo IVP to its Volterra integral form, subtracting the exact solution from an ε-approximate one, and applying the Lipschitz condition yields $M \leq \frac{\epsilon T^{\alpha}}{\Gamma(\alpha+1)} + \frac{KT^{\alpha}}{\Gamma(\alpha+1)} \cdot M$, where $M = sup_t \|\tilde{x}(t) - x(t)\|$. Rearranging yields $M \leq \theta\varepsilon$.

Corollary 9.3. Under the conditions of Theorem 9.2, the system is generalized Ulam–Hyers–Rassias stable with $\varphi(t) = E_{\alpha}(t^{\alpha})$ and constant $\theta\varphi = \theta$.

X. NUMERICAL ILLUSTRATIONS

All simulations used the ABM predictor–corrector scheme [19] with baseline parameters from Table 1, step size h = 0.1 days, and T = 200 days. The baseline reproduction number is:

$$R_0 = (0.35 \times 0.20) / [(0.2001)(0.1051)] \approx 3.33. \quad (22)$$

10.1. Effect of Fractional Order on Epidemic Dynamics

Figure 3 illustrates I(t) for α ∈ {0.7, 0.8, 0.9, 1.0}. Decreasing α delays the epidemic peak and reduces the peak infection size due to the memory effect, while the endemic equilibrium level is independent of α (consistent with the α-independence of R_0). This supports the theoretical finding that α governs transient dynamics but not epidemic thresholds.

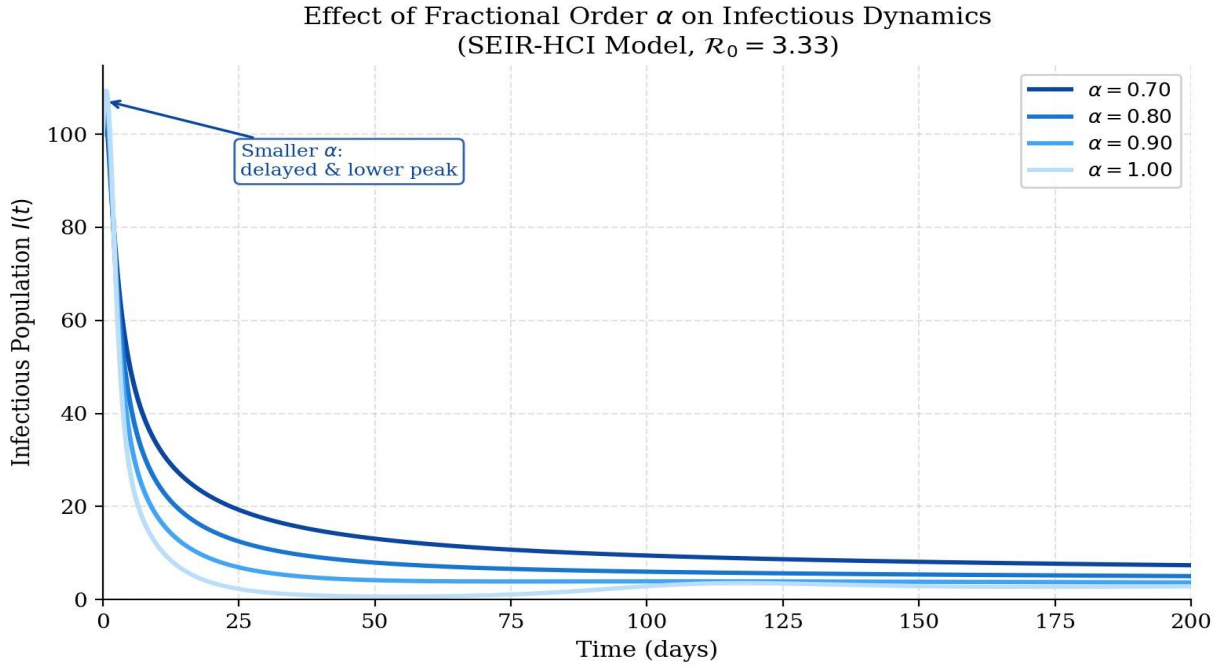

Figure 3: *Effect of fractional order α ∈{0.70, 0.80, 0.90, 1.00} on infectious population dynamics. Smaller α values delay the epidemic peak and reduce peak prevalence due to memory effects inherent in the Caputo fractional derivative.*

XI. CONCLUSION

We developed and rigorously analyzed the SEIR-HCI model, a Caputo fractional-order epidemic system with humoral and cellular immune response compartments. The model is mathematically well-posed; solutions exist, are unique, remain non-negative, and are uniformly bounded in Ξ. The basic reproduction number $R_0 = \beta\sigma/[(\sigma+\mu)(\gamma+\mu+\delta)]$ defines a sharp threshold for disease free equilibrium (DFE): when $R_0 \leq 1$ the DFE is globally asymptotically stable (GAS); whereas when $R_0 > 1$ there exists a unique endemic equilibrium which is also GAS. The Ulam–Hyers stability of solutions implies the reliability of numerical methods to capture these properties. The fractional parameter α controls the time scale of transients in terms of peak amplitude and timing; however, it does not affect the value of R_0, thus consistently with the biological intuition that memory can affect how fast an infection spreads but cannot modify the long term persistence criteria of R_0.

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