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THE DYNAMICS OF HELICOBACTER PYLORI-ASSOCIATED STOMACH CANCER UNDER ψ-HILFER FRACTIONAL DERIVATIVE

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Abstract. Helicobacter pylori (HP) is an infection that affects approximately 50% of the world's population and is one of the main pathogenic risk factors for stomach cancer. Therefore, a rapid, non-invasive diagnostic approach is necessary to avert gastritis, peptic ulcer, and ultimately, stomach cancer associated with HP. HP accounts for the majority of the etiological burden associated with gastric cancer; however, the majority of in vivo studies have failed to adequately induce malignancy through HP infection alone with HP infection alone. This paper creates a new fractional-order differential equations (FODEs) model of HP-induced stomach cancer using the ψ-Hilfer fractional derivative. FODEs support real-world population dynamics so this model propagates a FODE approach to HP-induced stomach cancer relative to treatment of HP infection as transmission dynamics support direct and indirect transmission with exclusion and inclusion approaches - successful treatment works as a control (removal) factor. This model involves six populations-susceptibles, exposed, infected, treated, stomach cancer patients, and environmental bacterial concentration. The model's unique solution is established, and the properties of positivity and boundedness are verified. Local and global stability assessed for equilibrium point where coexistence is observed. The Adams-Bashforth-Moulton method (Predictor-Corrector scheme) is applied for numerical simulations to assess fractional dynamics and population behavior relative to parameters of interest. This study shows that by increasing the treatment rate of HP infection, the incidence rate of HP infection and stomach cancer significantly drop; thus, effective treatment alongside reduction in the contact rate is essential for population-induced elimination of stomach cancer.

Keywords: ψ-Hilfer fractional derivative, stomach cancer, Helicobacter pylori, Adams-Bashforth-Moulton method.

2010 Mathematics Subject Classification: Primary: 26A33; Secondary: 34A08, 92D30.

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1 Introduction

Helicobacter pylori (HP) was discovered in 1983 by Robin Warren and Barry Marshall and is a gram-negative, microaerophilic, spiral-shaped bacterium [1, 22]. It was first classified as Campylobacter pylori but in 1989, it was designated to a new genus Helicobacter; in 1990, it was named Helicobacter pylori. It is estimated to infect approximately 50% of the world's population [1, 22]. HP is the primary bacterial infectious etiology for a number of gastric disease. It is the primary causal factor for chronic gastritis, peptic ulcer disease, functional dyspepsia, MALT lymphoma, and gastric malignancies [10, 20, 37, 39]. Due to its strong relationship with gastric adenocarcinoma, the International Agency for Research on Cancer has classified HP as a Group 1 carcinogen [15, 31].

As a Group 1 carcinogen, HP is a major triggering factor that causes gastric carcinogenesis. It causes a stepwise histopathological pathway from chronic non-atrophic gastritis to atrophic gastritis to intestinal metaplasia to dysplasia to gastric cancer [12, 16, 57].

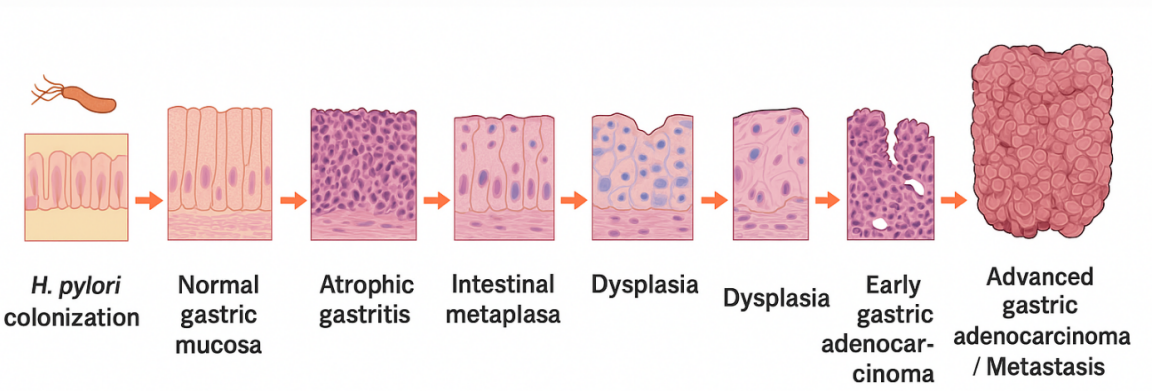

Figure 1: Progression of H. pylori-associated Stomach Cancer

Mechanistically, HP causes oxidative stress to gastric epithelial cells which causes inflammatory response and ultimately endoplasmic reticulum stress. This means HP causes DNA damage but also inhibits DNA damage repair response [27, 29, 38, 54]. Therefore, persistent inflammation exacerbates DNA damage and accelerates dysplasia to gastric carcinoma. In addition, even after HP eradication occurs (after intestinal metaplasia), histological studies show that gastric mucosa is rarely returned to normal tissue architecture due to non-reversible molecular changes [38]. Thus any known HPV-DNA damage relative to HPV-DNA, should be assessed to improve prevention, diagnostic and therapeutic strategies for HP-induced gastric disease [57]. The fact that it's one of the most important human bacterial pathogens suggests that HP colonizes the human stomach, and colonization typically persists into adulthood, since it is estimated that >50% of the world is colonized with HP by adulthood [21, 42].

HP is primarily colonized within the human body through its gastrointestinal tract epithelial surface residence as well as through mucosal penetration where it causes localized inflammation [4, 5, 26]. Since chronic HP infection occurs before most people reach adulthood, HP will lead to gastritis/atrophy/intestinal metaplasia which can ultimately lead to adenocarcinoma development relative to other risk factors as well [44]. According to epidemiological studies, 2.9% of people infected with HP will develop stomach cancer; meanwhile, no cases have been reported without HP positivity at diagnosis [53]. Furthermore, 62.18% of individuals with stomach cancer were noted to be positive for HP [21]. Yet the majority of experimental evidence indicates that HP cannot cause

gastric adenocarcinoma on its own without other mediating risk factors [6, 11, 28, 35, 41].

In 2012, approximately 952000 new cases of stomach cancer were diagnosed while approximately 720000 stomach cancer death recorded a mortality rate of ~75% [13, 23, 25]. It is highly prevalent in many countries as several risk factors exist which can lead to stomach cancer: prevalence of HP infection; smoking; dietary habits and preservation efforts [24]. However most literature suggests that HP infection is the most significant risk factor that initiates gastric cancer [9, 52, 55].

HP infection as determined by recent guidelines for treatment management and classified gastritis is the primary infectious disease with transmission occurring through (1) direct person-to-person; (2) oral-to-oral (kissing/vomiting/oral sex/breastfeeding); and (3) fecal-oral (contaminated food or water) means [17, 41]. Risk factors include household settings with overcrowding/low socioeconomic status/poor hygiene/contaminated food and water/immigration to places where HP prevalence is higher/home contacts who have become infected relative to other household members. Common associated symptoms include abdominal pain; fullness; early satiety; indigestion; nausea [31, 42].

Mathematical modeling in social sciences, engineering, and the natural sciences refers to the characterization of nonlinear system behavior with anticipatory characterization a common model exists called disease dynamics modeling across social science, engineering and natural science applications [2, 7, 30, 33] which explains how realistic growth can be modeled using real- world dynamics involving transmission which can be assessed using compartment modeling - mapping heterogeneous populations- and facilitating dynamic modeling for infection dynamics spread-potential - responsibilities for prone populations [8, 17, 32, 33].

The fractional order derivative was first established as a half order derivative in a letter by Gottfried Wilhelm Leibniz which he sent in 1695 to Guillaume de l'Hospital. This concept was discussed [46] but did not emerge as a formal mathematical theory until the nineteenth century until later in the nineteenth century when contributors (Bernhard Riemann/Leibniz's findings/Joseph Liouville) expanded upon those original thoughts in an era where newly established research suggests fractional derivatives/integrals produce real world movement phenomena increasingly better [46].

Thus fractional differential equations (FDEs) emerged [33] as an appropriate study model where concepts/classes of systems can be built without some contributors having implausible derivatives [33] whereby integer specific systems limited real world plausibilities. FODEs allow for extraordinary derivatives which create systems that longitudinally and non-locally represent memory better than classical equations [33] which have been established in other disciplines in math, engineering chemistry physics signals/images since the establishment of fractional calculus.

Many studies have taken place recently into the ψ-Hilfer derivative. Defined in the literature as a a suitably chosen kernel function ψ this linking power of flexibility became important because derivative connected to epidemic dynamics thus researchers became increasingly interested in Hilfer's work from 2018. The ψ-Hilfer fractional operator includes free choice over the selection of kernel function Ψ meaning it is especially strong over previously used systems with required derivatives. Thus many subsequent articles came out making this derivative important especially relative to properties established within. This is the only study to take use of the ψ-Hilfer fractional derivative as a generalized operator combining all previously derived definitions meaning FODEs are possible with uniqueness and properties proving existence through existence/uniqueness of solutions [46].

The remainder of this article is organized as follows: Section 2 introduces the ψ-Hilfer fractional derivative, Section 3 presents the mathematical model, Section 4 discusses existence and uniqueness

of solutions, Section 5 analyzes equilibria and stability, Section 6 describes the numerical scheme, Section 7 reports numerical simulations, and Section 8 concludes the paper.

2 Definitions of the ψ-Hilfer fractional derivative [1, 43]

2.1 ψ-Hilfer Fractional Integral and Derivative

Let $\psi \in C^1([a,b])$ be an increasing, positive, monotone function with $\psi'(t) \neq 0$.

For $0 < \alpha \leq 1$ and $0 \leq \beta \leq 1$, the **left-sided ψ-Hilfer fractional derivative** of a function f is defined as

$$ {}^{H}\mathcal{D}_{a^+}^{\alpha,\beta;\psi} f(t) = I_{a^+}^{\beta(1-\alpha);\psi} (\frac{1}{\psi'(t)} \frac{d}{dt}) I_{a^+}^{(1-\beta)(1-\alpha);\psi} f(t). $$

Similarly, the **right-sided ψ-Hilfer fractional derivative** is

$$ {}^{H}\mathcal{D}_{b^-}^{\alpha,\beta;\psi} f(t) = I_{b^-}^{\beta(1-\alpha);\psi} (-\frac{1}{\psi'(t)} \frac{d}{dt}) I_{b^-}^{(1-\beta)(1-\alpha);\psi} f(t). $$

Here, the **left-sided ψ-Riemann–Liouville fractional integral** is

$$ I_{a^+}^{\alpha;\psi} f(t) = \frac{1}{\Gamma(\alpha)} \int_a^t \psi'(s) \, (\psi(t) - \psi(s))^{\alpha-1} \, f(s) \, \mathrm{d}s, $$

and the **right-sided ψ-Riemann–Liouville fractional integral** is

$$ I_{b^-}^{\alpha;\psi} f(t) = \frac{1}{\Gamma(\alpha)} \int_t^b \psi'(s) \, (\psi(s) - \psi(t))^{\alpha-1} \, f(s) \, \mathrm{d}s. $$

2.2 Choice of $\psi\,(\mathbf{t})$

The function ψ acts as a kernel generator that reparametrizes time. It must be monotone increasing and differentiable. Common choices include:

- $\psi(t) = t$ (standard fractional derivatives)
- $\psi(t) = lnt$ (logarithmic kernel)
- $\psi(t) = t^{\rho},\ \rho > 0$ (power-law kernel)
- $\psi(t) = sint,\ e^t$ (oscillatory or exponential time-scaling)

3 Description of the proposed mathematical model

Helicobacter pylori infection remains a global public health challenge, particularly in developing countries. Understanding its transmission pathways is critical for prevention and for reducing gastric cancer risk.

Earlier studies applied compartmental models to related infections. Cousins et al. [14] modeled Campylobacter dynamics through direct and indirect transmission. Rupnow et al. [47] used an SEIR framework to project H. pylori infection trends in the United States, while Siewe et al. [49] emphasized the role of asymptomatic carriers in sustaining infection.

Yet, few models have considered treatment as a control measure or examined the direct link between H. pylori and stomach cancer. This study addresses that gap by incorporating treatment into the model and analyzing its effects on gastric cancer dynamics through qualitative behavior, sensitivity analysis, and numerical simulations.

3.1 Helicobacter pylori transmission and progression to stomach cancer [34]

The total human population is divided into five compartments:

- $S(t)$: Susceptible individuals
- $E(t)$: Exposed individuals (infected but not symptomatic, can transmit)
- $I(t)$: Infected individuals (symptomatic with *H. pylori*)
- $T(t)$: Treated individuals (received treatment after infection)
- $C(t)$: Cancer individuals (progressed to stomach cancer)

3.1.1 Forces of Infection

Human-human transmission:

$$\lambda_1 = \beta_1 \left(E + \eta_1 I + \eta_2 T + \eta_3 C\right),$$

where β_1 is the ingestion rate of *H. pylori* through human contact, and $\eta_1, \eta_2, \eta_3 < 1$ are modification parameters for infectiousness.

Environment-human transmission:

$$\lambda_2 = \frac{(1-\varepsilon)\,\beta_2 H}{K+H},$$

where β_2 is the ingestion rate through contaminated environment, H is the concentration of *H. pylori* in water/food, K is the half-saturation constant, ε represents hygiene and sanitation, and $\frac{H}{K+H}$ is the probability of exposure to contaminated sources.

3.1.2 Model Assumption

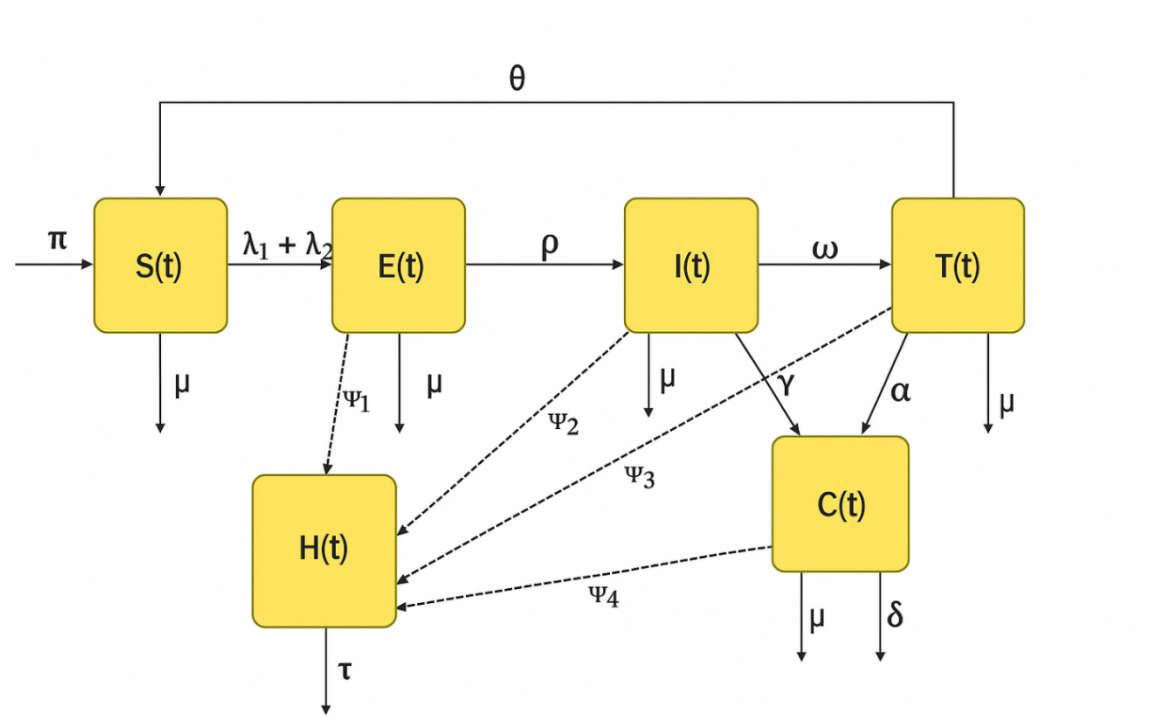

Figure 2: Flow diagram of the Helicobacter pylori transmission and progression to stomach cancer model [42]

- Strongest risk factor for stomach cancer is *H. pylori* infection.
- Human population is constant.
- All parameters are non-negative.
- All individuals are susceptible since *H. pylori* is widespread.
- Recovery does not provide permanent immunity.
- Birth and death rates are unequal.

In this study, the suggested ψ-Hilfer fractional-order model for Helicobacter pylori transmission and progression to stomach cancer is as follows: We consider the ψ-Hilfer fractional model of order $0 < \alpha \leq 1$, type $0 \leq \beta \leq 1$, with kernel $\psi(t)$, where $\gamma = \alpha + \beta(1-\alpha)$:

$$
\begin{aligned}
{}^{H}D_{a+}^{\alpha,\beta;\psi} S(t) &= \pi + \theta T - (\mu + \lambda_1 + \lambda_2)\, S, \\
{}^{H}D_{a+}^{\alpha,\beta;\psi} E(t) &= (\lambda_1 + \lambda_2)\, S - (\mu + \rho) E, \\
{}^{H}D_{a+}^{\alpha,\beta;\psi} I(t) &= \rho E - (\mu + \omega + \gamma) I, \\
{}^{H}D_{a+}^{\alpha,\beta;\psi} T(t) &= \omega I - (\alpha + \mu + \theta) T, \\
{}^{H}D_{a+}^{\alpha,\beta;\psi} C(t) &= \gamma I + \alpha T - (\mu + \delta) C, \\
{}^{H}D_{a+}^{\alpha,\beta;\psi} H(t) &= \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C - \tau H.
\end{aligned}
$$

Parameter	Description
π	Birth rate of individuals
μ	Natural mortality rate of individuals
λ_1	Force of infection due to human-to-human transmission
λ_2	Force of infection due to environmental transmission
ρ	Rate at which exposed individuals become infectious
ω	Treatment rate of infected individuals (I(t))
θ	Rate at which treated individuals return to the susceptible class
δ	Mortality rate induced by stomach cancer
γ	Rate at which infected individuals develop stomach cancer
α	Rate at which treated individuals develop stomach cancer
τ	Natural death rate of H. pylori bacteria
ψ_1	Shedding of H. pylori from the exposed class
ψ_2	Shedding of H. pylori from the infected class
ψ_3	Shedding of H. pylori from the treated class
ψ_4	Shedding of H. pylori from the stomach cancer class

Table 2: Parameter description [42]

with initial conditions

$$\left(I_{a+}^{1-\gamma;\psi}S\right)(a) = S_0, \quad \left(I_{a+}^{1-\gamma;\psi}E\right)(a) = E_0, \quad \left(I_{a+}^{1-\gamma;\psi}I\right)(a) = I_0,$$
$$\left(I_{a+}^{1-\gamma;\psi}T\right)(a) = T_0, \quad \left(I_{a+}^{1-\gamma;\psi}C\right)(a) = C_0, \quad \left(I_{a+}^{1-\gamma;\psi}H\right)(a) = H_0.$$

In the proposed ψ-Hilfer fractional-order model, the total population at time t is denoted as *N(t)*. It is defined as the sum of all compartments: *N(t) = S(t) + E(t) + I(t) + T(t) + C(t)* This equation ensures that the total population *N(t)* is the cumulative sum of all individuals in the system across the defined compartments. It represents population conservation under the assumption that no external births or deaths are explicitly included in the model.

4 Existence and Uniqueness of the ψ-Hilfer Helicobacter pylori system

Let $0 < \alpha \leq 1$, $0 \leq \beta \leq 1$, $\gamma = \alpha + \beta(1-\alpha)$, and $\psi \in C^1([a,b])$ be strictly increasing with $\psi'(t) > 0$. Consider the Cauchy-type problem

$$^{H}D_{a+}^{\alpha,\beta;\psi}X(t) = F(t, X(t)), \qquad \left(I_{a+}^{1-\gamma;\psi}X\right)(a) = X_0,$$

where $X = (S, E, I, T, C, H)^\top$ and

$$F(t, X) = AX + f,$$

with $f = \left(\pi,\ 0,\ 0,\ 0,\ 0,\ 0\right)^{\top}$, and

$$A = \begin{pmatrix} -(\mu+\lambda_1+\lambda_2) & 0 & 0 & \theta & 0 & 0 \\ (\lambda_1+\lambda_2) & -(\mu+\rho) & 0 & 0 & 0 & 0 \\ 0 & \rho & -(\mu+\omega+\gamma) & 0 & 0 & 0 \\ 0 & 0 & \omega & -(\alpha+\mu+\theta) & 0 & 0 \\ 0 & 0 & \gamma & \alpha & -(\mu+\delta) & 0 \\ 0 & \psi_1 & \psi_2 & \psi_3 & \psi_4 & -\tau \end{pmatrix}.$$

Theorem 4.1 *(Existence and Uniqueness)*
Suppose F is continuous in t and Lipschitz continuous in X with constant L in a ball $B_R \subset \mathbb{R}^6$. Then there exists $h > 0$ such that the above ψ–Hilfer system admits a unique solution $X \in C_{1-\gamma;\psi}([a, a+h], \mathbb{R}^6)$, and, if the solution does not blow up, it extends uniquely to $[a, b]$. Moreover, solutions depend continuously on the initial data X_0.

Proof. Using the definition of the ψ-Hilfer derivative, the system is equivalent to the Volterra integral equation

$$X(t) = \frac{(\psi(t)-\psi(a))^{\gamma-1}}{\Gamma(\gamma)} X_0 + \frac{1}{\Gamma(\alpha)} \int_a^t \psi'(s)\,(\psi(t)-\psi(s))^{\alpha-1}\, F\,(s, X(s))\ \mathrm{d}s.$$

This defines an operator $\mathcal{T}$ on the weighted space

$$C_{1-\gamma;\psi}\left([a, a+h]\right) = \{X : [a, a+h] \to \mathbb{R}^6 \ \mid \sup_{t\in[a,a+h]} (\psi(t)-\psi(a))^{1-\gamma} \parallel X(t) \parallel < \infty\}.$$

A contraction estimate follows from the Lipschitz condition:

$$\parallel \mathcal{T}X - \mathcal{T}Y \parallel_{C_{1-\gamma;\psi}} \leq \frac{L\,(\psi(a+h)-\psi(a))^{\alpha}}{\Gamma(\alpha+1)} \parallel X - Y \parallel_{C_{1-\gamma;\psi}}.$$

For sufficiently small h, the factor is < 1, so Banach's fixed-point theorem yields a unique local solution. Global extension follows by continuation, and continuous dependence arises from a Grönwall inequality adapted to ψ. □

Remark 4.2 This result generalizes the classical Picard–Lindelöf theorem to the ψ–Hilfer setting. *[50]*

5 ψ-Hilfer Helicobacter pylori system: Equilibria, Positivity, Boundedness, and Stability with integral-type initial conditions

5.1 Equilibrium Point

Write $x = (S, E, I, T, C, H)^{\top}$ and A as

$$A = \begin{pmatrix} -(\mu+\lambda_1+\lambda_2) & 0 & 0 & \theta & 0 & 0 \\ (\lambda_1+\lambda_2) & -(\mu+\rho) & 0 & 0 & 0 & 0 \\ 0 & \rho & -(\mu+\omega+\gamma) & 0 & 0 & 0 \\ 0 & 0 & \omega & -(\alpha+\mu+\theta) & 0 & 0 \\ 0 & 0 & \gamma & \alpha & -(\mu+\delta) & 0 \\ 0 & \psi_1 & \psi_2 & \psi_3 & \psi_4 & -\tau \end{pmatrix},$$

$$b = \begin{pmatrix} \pi \\ 0 \\ 0 \\ 0 \\ 0 \\ 0 \end{pmatrix}.$$

Equilibria satisfy $Ax^* + b = 0$. Solving gives (let $k_1 = \frac{\lambda_1+\lambda_2}{\mu+\rho}$, $k_2 = \frac{\rho}{\mu+\omega+\gamma}$, $k_3 = \frac{\omega}{\alpha+\mu+\theta}$, $k_4 = \frac{1}{\mu+\delta}$ and $\kappa = \theta k_1 k_2 k_3$)

$$\begin{array}{lll} S^* & = \frac{\pi}{(\mu+\lambda_1+\lambda_2)-\kappa}, & E^* = k_1 S^*, \qquad I^* = k_2 E^*, \\ T^* & = k_3 I^*, & C^* = k_4\left(\gamma I^* + \alpha T^*\right) = k_4\left(\gamma + \alpha k_3\right) k_2 k_1 S^*, \\ H^* & & = \frac{\psi_1 E^* + \psi_2 I^* + \psi_3 T^* + \psi_4 C^*}{\tau}. \end{array}$$

This equilibrium is positive if $(\mu + \lambda_1 + \lambda_2) - \kappa > 0$.

5.2 Positivity Forward Invariance

On each coordinate face with a vanishing component, the corresponding right-hand side is nonnegative:

$$\begin{array}{llllll} S = 0: & \pi + \theta T \geq 0, & E = 0: & \left(\lambda_1 + \lambda_2\right) S \geq 0, & I = 0: & \rho E \geq 0, \\ T = 0: & \omega I \geq 0, & C = 0: & \gamma I + \alpha T \geq 0, & H = 0: & \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C \geq 0. \end{array}$$

Hence $\mathbb{R}^6_{\geq 0}$ is positively invariant for the ψ–Hilfer system (Metzler structure).

5.3 Boundedness

Let $k > 0$ be chosen so that $k\psi_1 \leq \mu$, $k\psi_2 \leq \mu$, $k\psi_3 \leq \mu + \theta$, $k\psi_4 \leq \mu + \delta$, and define $V = S + E + I + T + C + kH$. Using the equations,

$$^{H}D^{\alpha,\beta;\psi}_{a+} V(t) \leq \pi - m(S + E + I + T + C) - k\tau H \leq \pi - mV(t),$$

with $m = min\{\mu, \mu, \mu, \mu, \mu + \delta\}$. By the fractional comparison principle for the ψ–Hilfer operator,

$$V(t) \leq V(a) E_\alpha(-m\left(\psi(t) - \psi(a)\right)^\alpha) + \frac{\pi}{m}(1 - E_\alpha(-m\left(\psi(t) - \psi(a)\right)^\alpha)),$$

so V and all components remain bounded for $t \geq a$.

5.4 Stability (Local and Global)

The Jacobian at x^* equals A. For $0 < \alpha \leq 1$, the equilibrium x^* is Mittag–Leffler stable iff the eigenvalues of A satisfy the Matignon sector condition

$$|arg\left(\lambda_i\right)| > \frac{\alpha\pi}{2} \quad \text{for all } \lambda_i \in \sigma(A).$$

If this holds, the unique solution admits

$$x(t) = x^* + E_\alpha((\psi(t) - \psi(a))^\alpha A)\ (x(a) - x^*)\ \to\ x^* \quad (t \to \infty),$$

hence x^* is globally (Mittag–Leffler) asymptotically stable.

5.5 Comparison Principle Sketch for V

Let ${}^{H}D_{a+}^{\alpha,\beta;\psi}V \leq \pi - mV$ with $m > 0$ and $0 < \alpha \leq 1$. Consider the scalar inequality

$$ {}^{H}D_{a+}^{\alpha,\beta;\psi}y = \pi - my, \qquad \left(I_{a+}^{1-\gamma;\psi}y\right)(a) = \left(I_{a+}^{1-\gamma;\psi}V\right)(a).$$

Its solution is

$$y(t) = y(a)E_{\alpha}(-m\,(\psi(t)-\psi(a))^{\alpha}) + \frac{\pi}{m}(1 - E_{\alpha}(-m\,(\psi(t)-\psi(a))^{\alpha})).$$

Since the right-hand side of the V-dynamics is $\leq$ that of y, the comparison theorem for ψ–Hilfer systems yields $V(t) \leq y(t)$ for $t \geq a$, proving boundedness. The argument relies on monotonicity of ψ–fractional integrals and the resolvent positivity.

6 ψ-Hilfer Helicobacter pylori system with Adams–Bashforth–Moulton Predictor–Corrector Scheme

We consider the following ψ-Hilfer fractional-order system:

$$ {}^{H}D_{a+}^{\alpha,\beta;\psi}\mathbf{X}(t) = \mathbf{F}\left(t, \mathbf{X}(t)\right), \qquad \left(I_{a+}^{1-\gamma;\psi}\mathbf{X}\right)(a) = \mathbf{X}_0,$$

where $\mathbf{X}(t) = (S, E, I, T, C, H)^{\top}$, $\gamma = \alpha + \beta(1-\alpha)$, and

$$\mathbf{F}\left(t, \mathbf{X}\right) = \begin{bmatrix} \pi + \theta T - (\mu + \lambda_1 + \lambda_2)\, S \\ (\lambda_1 + \lambda_2)\, S - (\mu + \rho)E \\ \rho E - (\mu + \omega + \gamma)I \\ \omega I - (\alpha + \mu + \theta)T \\ \gamma I + \alpha T - (\mu + \delta)C \\ \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C - \tau H \end{bmatrix}.$$

6.1 Integral Formulation

The equivalent Volterra integral formulation is given by

$$\mathbf{X}(t) = \frac{(\psi(t)-\psi(a))^{\gamma-1}}{\Gamma(\gamma)}\mathbf{X}_0 + \frac{1}{\Gamma(\alpha)}\int_a^t \psi'(s)\ (\psi(t)-\psi(s))^{\alpha-1}\ \mathbf{F}\left(s, \mathbf{X}(s)\right)\ \mathrm{d}s.$$

6.2 ψ-Uniform Grid

Let $\Psi_j = \psi\left(t_j\right) = \Psi_0 + jh_{\psi}$,$t_j = \psi^{-1}\left(\Psi_j\right)$, $j = 0, 1, \ldots$. We set $\mathbf{X}_j \approx \mathbf{X}\left(t_j\right)$, $\mathbf{F}_j = \mathbf{F}\left(t_j, \mathbf{X}_j\right)$. Weights:

$$\begin{aligned} b_k &= (k+1)^{\alpha} - k^{\alpha}, \\ a_k &= (k+2)^{\alpha+1} - 2(k+1)^{\alpha+1} + k^{\alpha+1}, \end{aligned}$$

and constants

$$C_{\alpha} = \frac{h_{\psi}^{\alpha}}{\Gamma(\alpha+1)}, \qquad D_{\alpha} = \frac{h_{\psi}^{\alpha}}{\Gamma(\alpha+2)}.$$

6.3 Predictor–Corrector Scheme

Predictor (Adams–Bashforth):

$$\mathbf{X}_{n+1}^{P} = \frac{(\Psi_{n+1} - \Psi_0)^{\gamma-1}}{\Gamma(\gamma)}\mathbf{X}_0 + C_\alpha \sum_{j=0}^{n} b_{\,n-j}\, \mathbf{F}_j.$$

Corrector (Adams–Moulton):

$$\mathbf{X}_{n+1} = \frac{(\Psi_{n+1} - \Psi_0)^{\gamma-1}}{\Gamma(\gamma)}\mathbf{X}_0 + D_\alpha \left(\sum_{j=0}^{n} a_{\,n-j}\, \mathbf{F}_j + a_{-1}\, \mathbf{F}\left(t_{n+1}, \mathbf{X}_{n+1}^{P}\right) \right),$$

with $a_{-1} = 1$.

6.4 Componentwise Form

For each variable, the right-hand side components are

$$\begin{aligned}
F_S &= \pi + \theta T - (\mu + \lambda_1 + \lambda_2)\, S, \\
F_E &= (\lambda_1 + \lambda_2)\, S - (\mu + \rho) E, \\
F_I &= \rho E - (\mu + \omega + \gamma) I, \\
F_T &= \omega I - (\alpha + \mu + \theta) T, \\
F_C &= \gamma I + \alpha T - (\mu + \delta) C, \\
F_H &= \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C - \tau H.
\end{aligned}$$

Thus, for example, the predictor for S is

$$S_{n+1}^{P} = \frac{(\Psi_{n+1} - \Psi_0)^{\gamma-1}}{\Gamma(\gamma)} S_0 + C_\alpha \sum_{j=0}^{n} b_{\,n-j}\, F_S\left(t_j, \mathbf{X}_j\right),$$

and similarly for E, I, T, C, H.

7 Numerical simulations predicting future dynamics of Helicobacter pylori-associated stomach cancer

Python was employed to perform numerical simulations in order to investigate the dynamical behavior of the ψ-Hilfer Helicobacter pylori system. The simulations were conducted using the specified initial conditions and parameter values summarized in Table 3, which were derived from the literature review and subsequently used for graphical presentation. The ψ-Hilfer fractional model of order $0 < \alpha \leq 1$, type $0 \leq \beta \leq 1$, with kernel $\psi(t)$, where $\gamma = \alpha + \beta(1 - \alpha)$ and *N(t) = S(t) +*

E(t) + I(t) + T(t) + C(t):

$$
\begin{aligned}
{}^{H}D_{a+}^{\alpha,\beta;\psi}S(t) &= \pi + \theta T - (\mu + \lambda_1 + \lambda_2)\, S,\\
{}^{H}D_{a+}^{\alpha,\beta;\psi}E(t) &= (\lambda_1 + \lambda_2)\, S - (\mu + \rho)E,\\
{}^{H}D_{a+}^{\alpha,\beta;\psi}I(t) &= \rho E - (\mu + \omega + \gamma)I,\\
{}^{H}D_{a+}^{\alpha,\beta;\psi}T(t) &= \omega I - (\alpha + \mu + \theta)T,\\
{}^{H}D_{a+}^{\alpha,\beta;\psi}C(t) &= \gamma I + \alpha T - (\mu + \delta)C,\\
{}^{H}D_{a+}^{\alpha,\beta;\psi}H(t) &= \psi_1 E + \psi_2 I + \psi_3 T + \psi_4 C - \tau H.
\end{aligned}
$$

with initial conditions

$$
\begin{aligned}
&\left(I_{a+}^{1-\gamma;\psi}S\right)(0) = 1500, \quad \left(I_{a+}^{1-\gamma;\psi}E\right)(0) = 1250, \quad \left(I_{a+}^{1-\gamma;\psi}I\right)(0) = 1000,\\
&\left(I_{a+}^{1-\gamma;\psi}T\right)(0) = 800, \quad \left(I_{a+}^{1-\gamma;\psi}C\right)(0) = 0, \quad \left(I_{a+}^{1-\gamma;\psi}H\right)(0) = 0.
\end{aligned}
$$

Parameter	**Value**
π	10
μ	0.000019
λ_1	0.02
λ_2	0.1075
ρ	0.65
ω	0.0073
θ	0.002485
δ	0.00839
γ	0.003
α	0.0066
τ	0.001
ψ_1	0.0009
ψ_2	0.0008
ψ_3	0.0004
ψ_4	0.0001

Table 3. Parameters and values of the model [17]

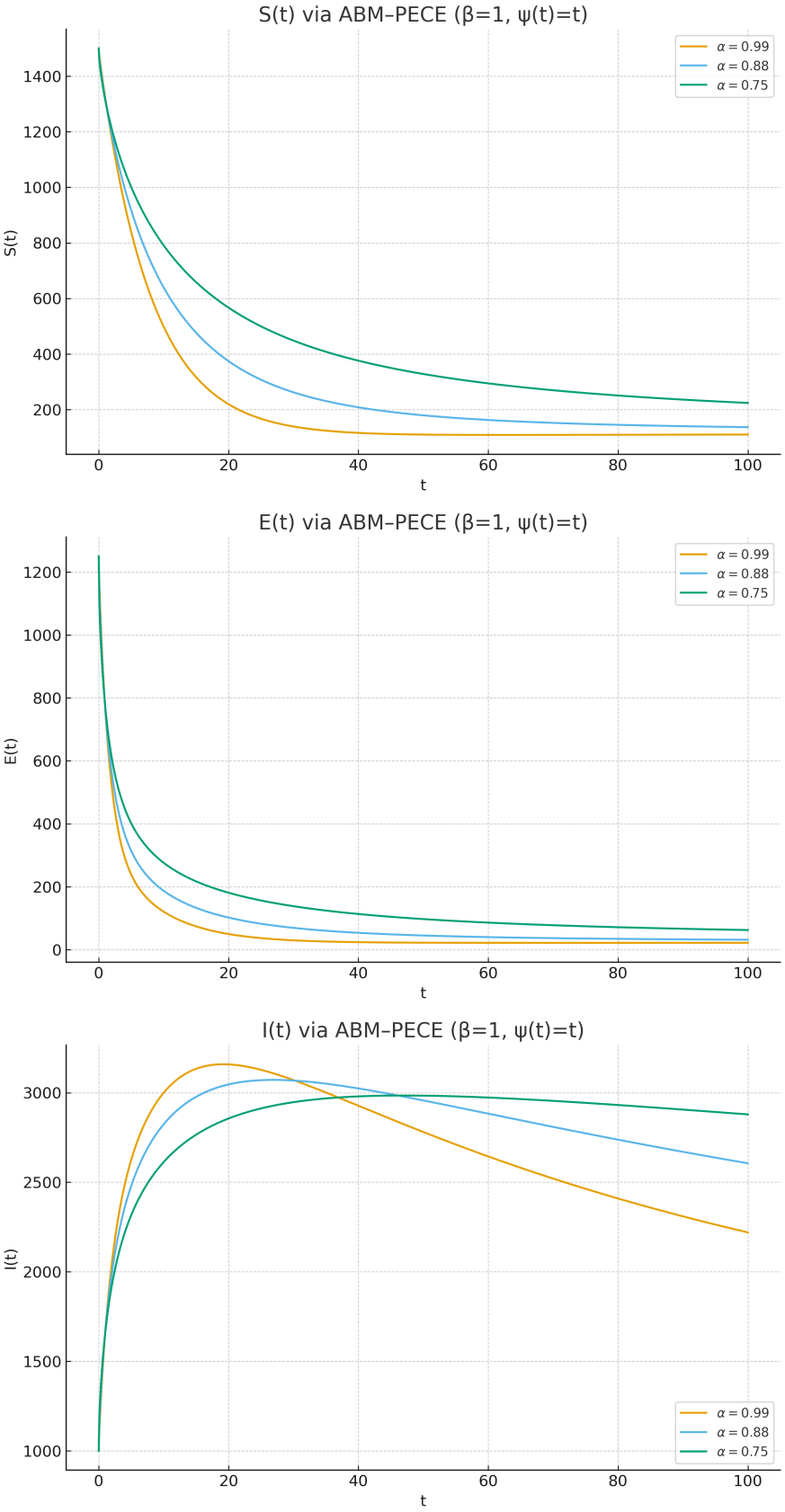

Figure 3: Numerical comparisons of ψ-Hilfer H. pylori system with $\psi(t) = t$.

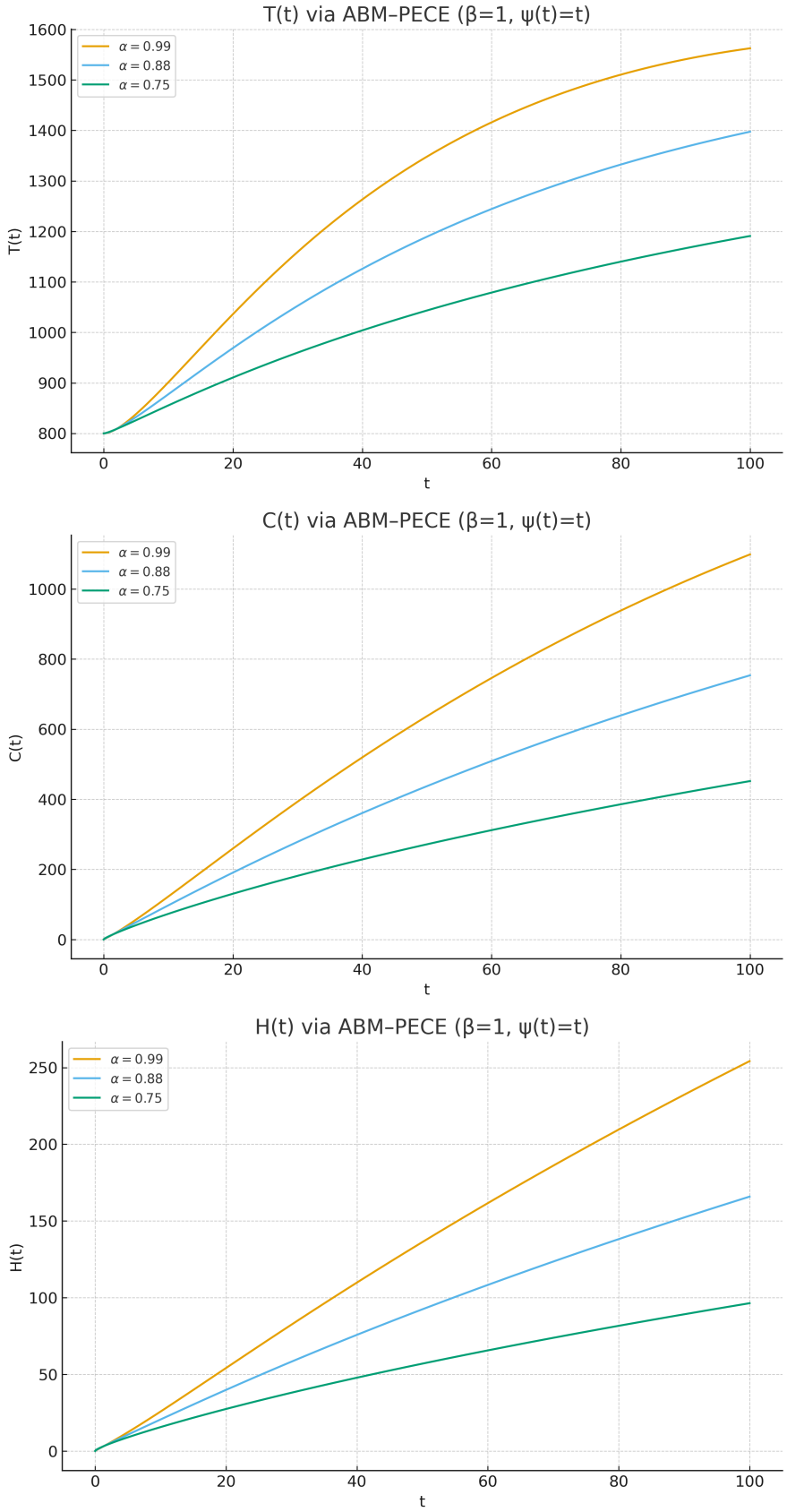

Fig. 3 (cont'd): Numerical comparisons of ψ-Hilfer H. pylori system with $\psi(t) = t$.

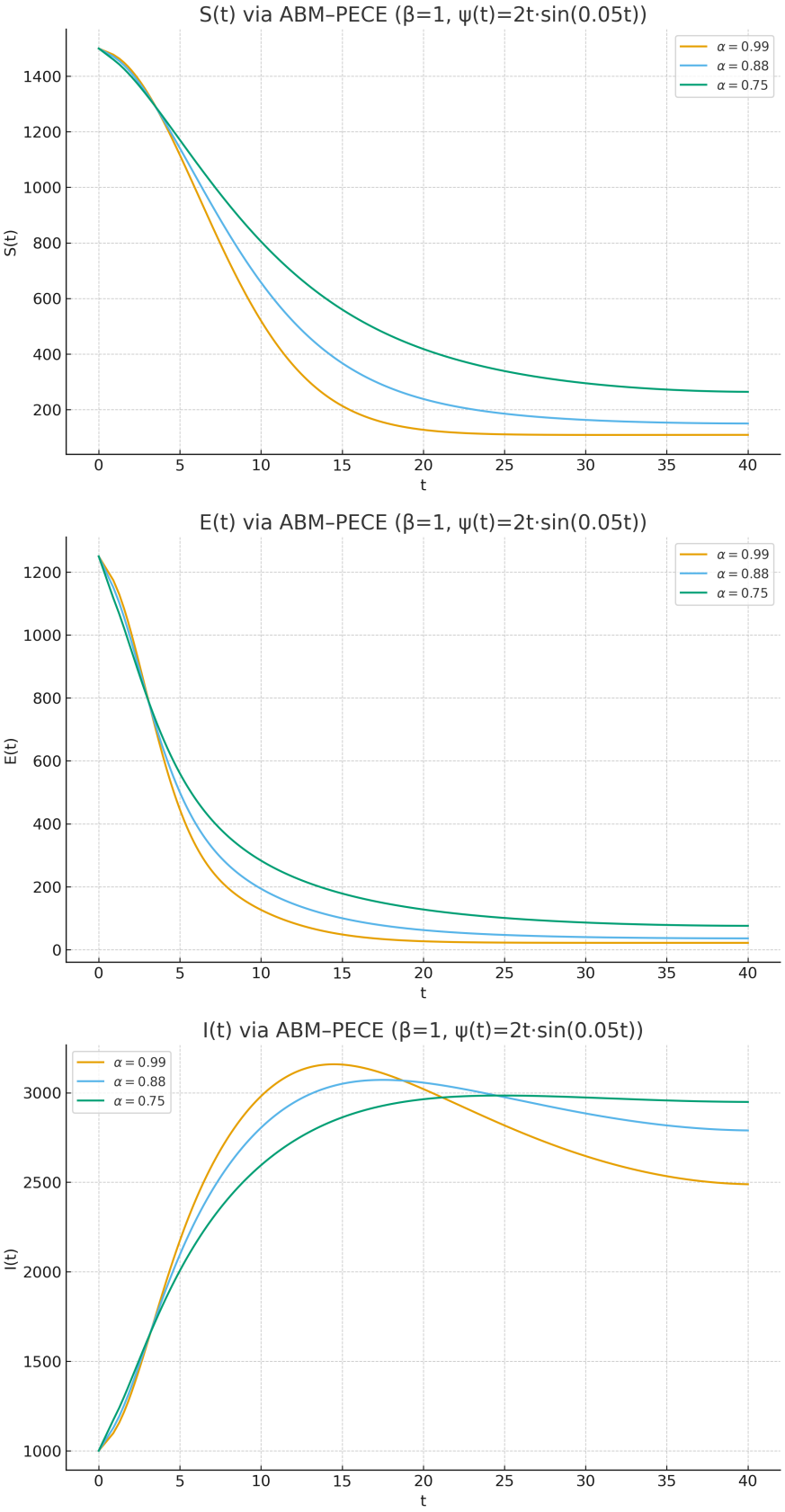

Figure 4: Numerical comparisons of ψ-Hilfer H. pylori system with $\psi(t) = 2t\sin(0.05t)$

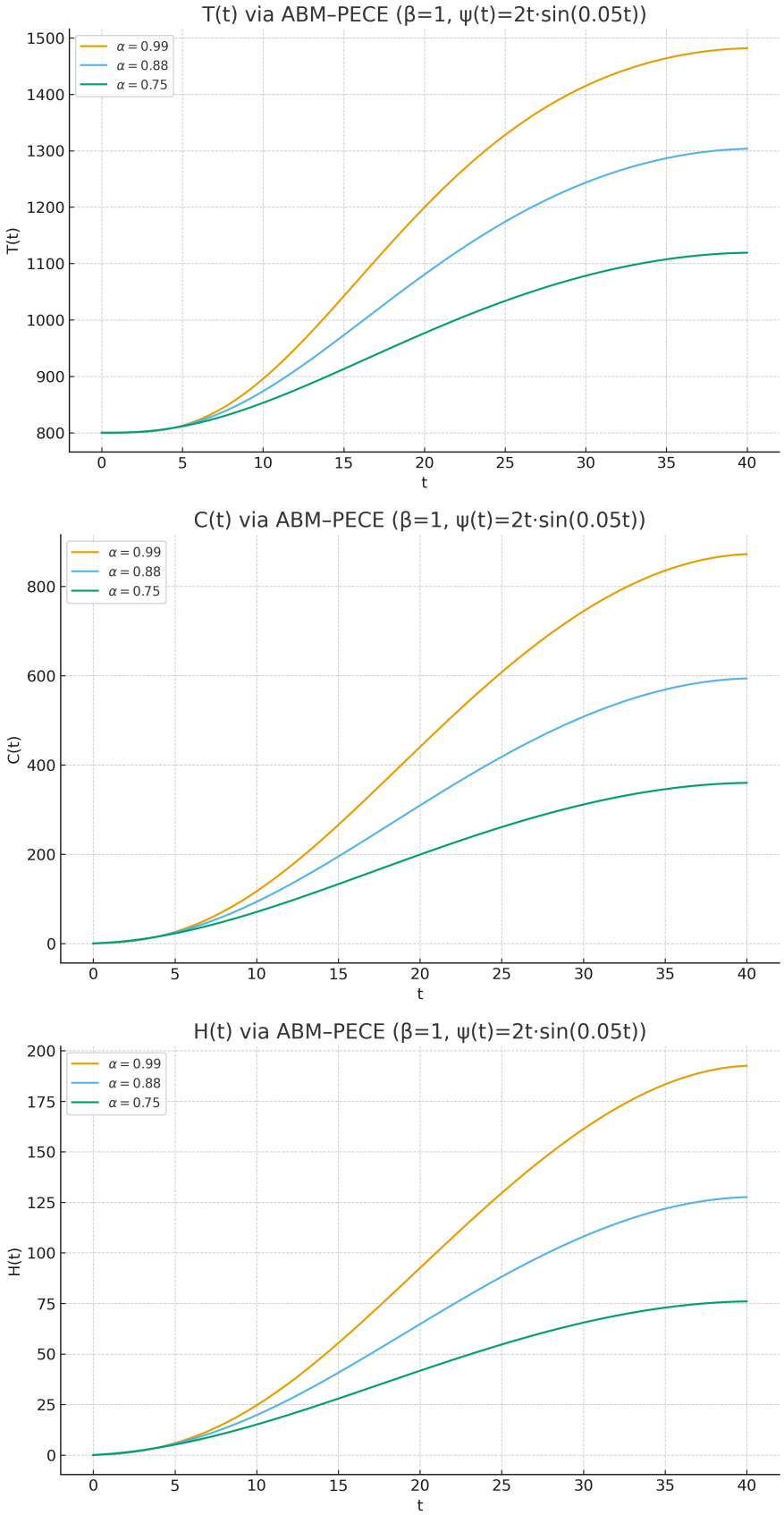

Figure 4 (cont'd): Numerical comparisons of ψ-Hilfer H. pylori system with $\psi(t) = 2t\sin(0.05t)$

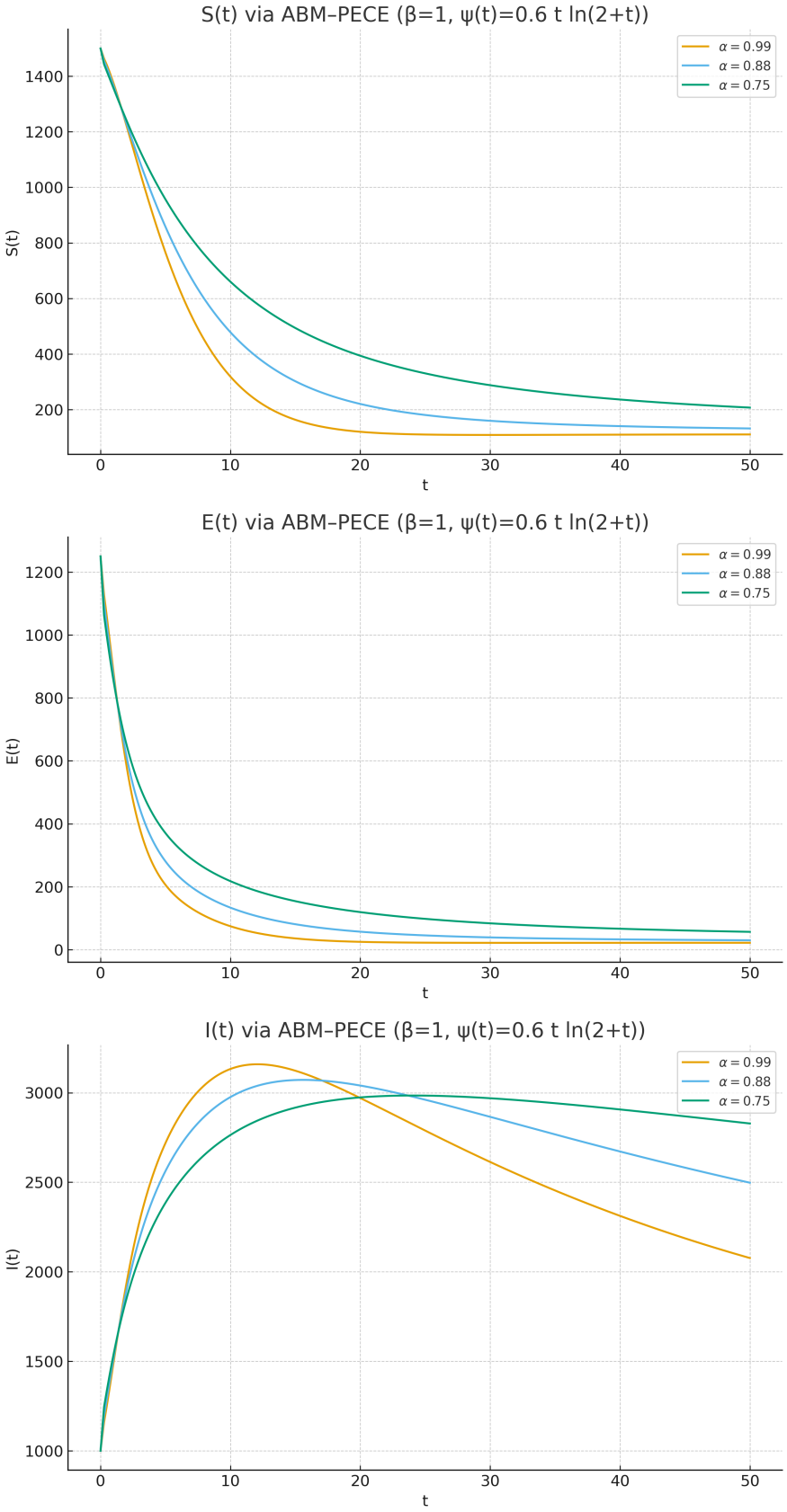

Figure 5: Numerical comparisons of ψ-Hilfer H. pylori system with $\psi(t) = 0.6t\ln(2+t)$

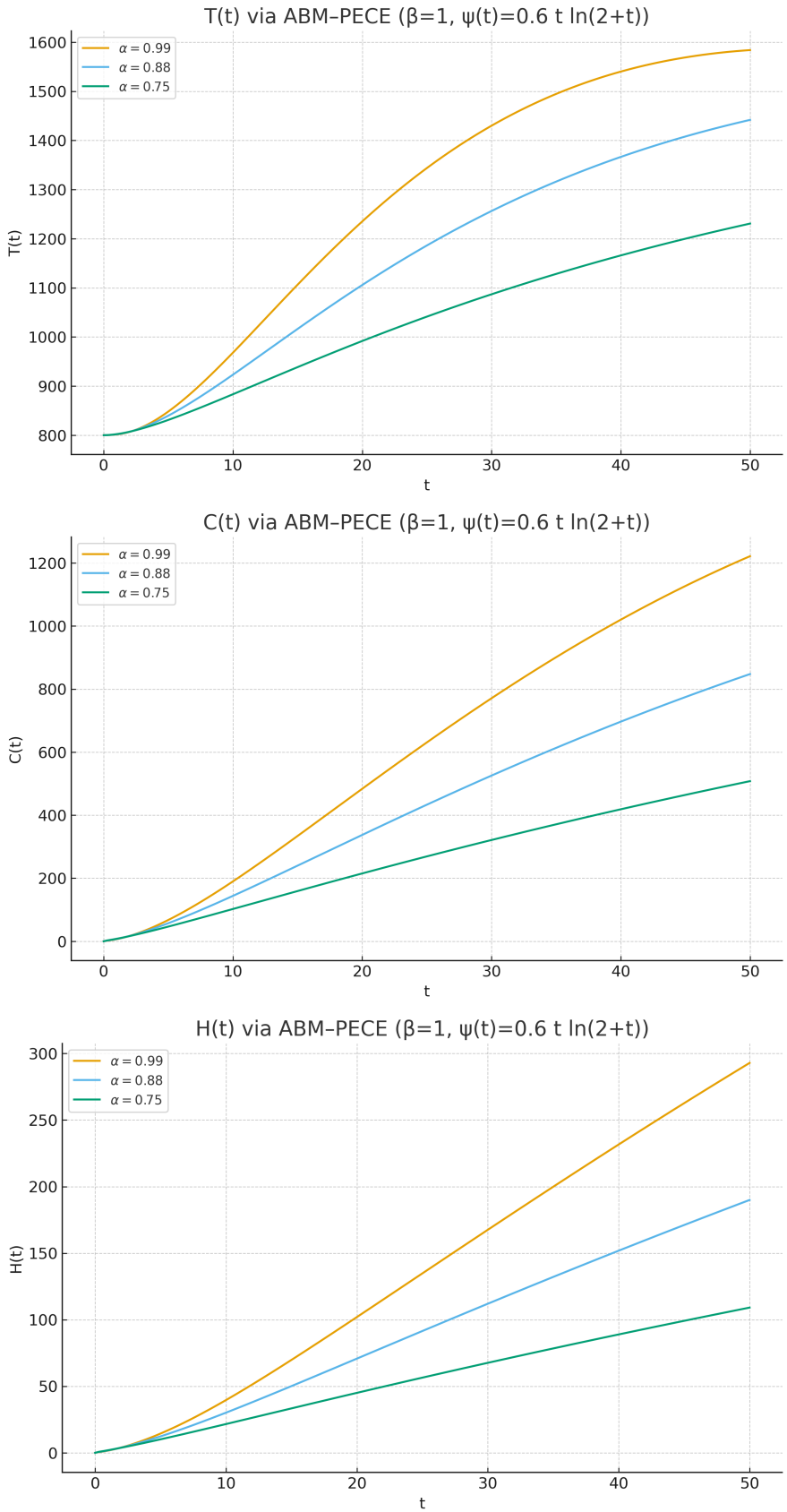

Fig. 5 (cont'd: Numerical comparisons of ψ-Hilfer H. pylori system with $\psi(t) = 0.6t\ln(2+t)$

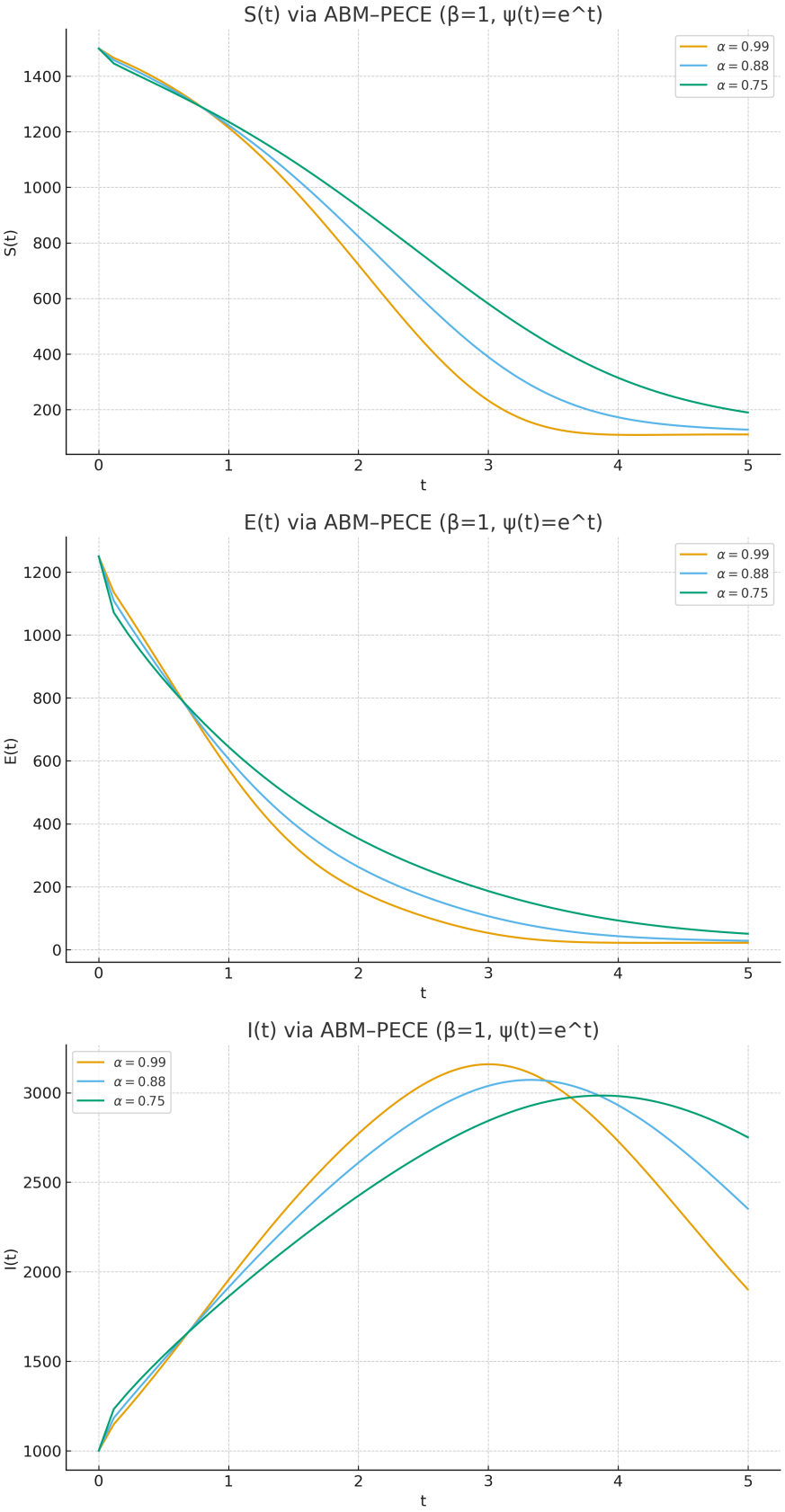

Figure 6: Numerical comparisons of ψ-Hilfer H. pylori system with $\psi(t) = e^t$

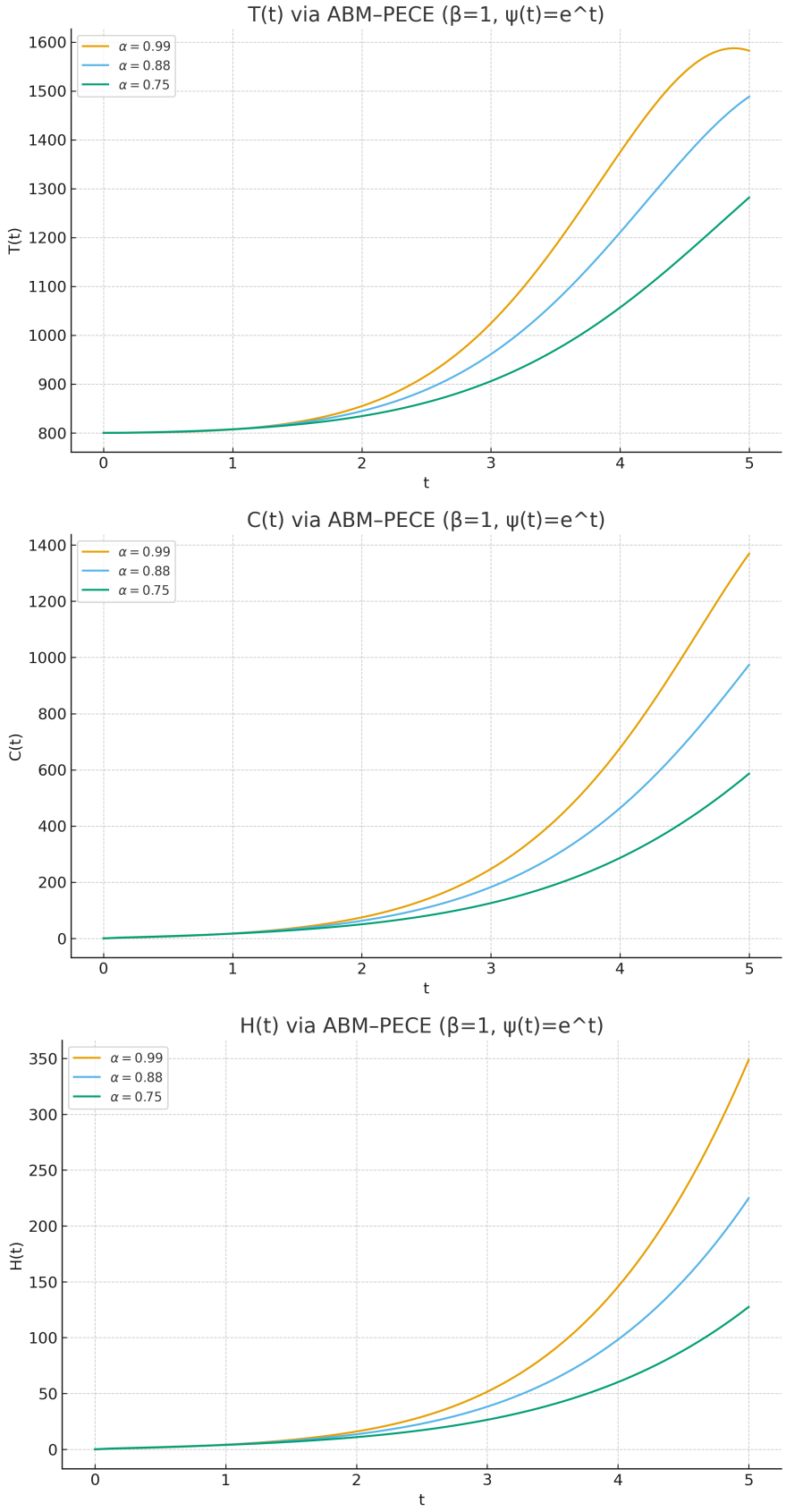

Fig. 6 (cont'd): Numerical comparisons of ψ-Hilfer H. pylori system with $\psi(t) = e^t$

7.1 Figure Captions

Figure 3: Numerical simulation of the ψ-Hilfer Helicobacter pylori system with $\psi(t) = t$. The classical kernel yields baseline epidemic dynamics, with rapid initial infection spread, a steady rise in cancer cases, and stabilization of the environmental bacterial load.

Figure 4: Numerical simulation of the ψ-Hilfer Helicobacter pylori system with $\psi(t) = 2t\sin(0.05t)$. The oscillatory kernel introduces periodic fluctuations across all compartments, mimicking seasonal or behavioral cycles of infection and transmission.

Figure 5: Numerical solution of the ψ-Hilfer Helicobacter pylori system with $\psi(t) = 0.6tln(2+t)$. A logarithmic kernel makes for slower dynamics, higher cancer totals over time and lower infection totals as larger concerns after longer time periods, higher totals over time and greater reduction in environmental bacterial load from a preventable concern based upon unwanted stomach irritants.

Figure 6: Numerical solution of the ψ-Hilfer Helicobacter pylori system with $\psi(t) = e^t$. An exponential kernel makes for faster dynamics, lower cancer totals over time and lower infection totals over time as well as greater reduction in environmental bacterial load from a proactive approach.

7.2 General Observations

This section exists as a general commentary relative to all the numerical results found in Section 7 regarding dynamics per each selected kernel component for the ψ-Hilfer Helicobacter pylori system. Therefore, for organization, we will separate commentary by figure.

All results in Section 7 are based upon the Adams-Bashforth-Moulton predictor-corrector method of a solution for the ψ-Hilfer fractional model. The only difference in considerations relative to the integrated form is the selected kernel function $\psi(t)$. This is an effective means to validate kernels for memory and scaling-type fractional system concerns.

The initial conditions discussed are relative and realistic epidemiological parameter findings discussed relative to Table 3 with $S = 1500$, $E = 1250$, $I = 1000$, $T = 800$, $C = 0$, $H = 0$.

For each run, it is clear there is a stable equilibrium where each value compartment at equilibrium equals the same constant value suggesting that S, E, I, T, C, H are sustainable for each other no matter where they start (as long as they are realistic) and in a predictable fashion toward a stable value determined for medical practical application.

However, it is clear that kernel $\psi(t)$ makes a difference regarding how relatively easily this equilibrium is reached:

- linearized means more than desired to determine maximum attributable infection values and cancer results.

- Treated values ultimately less infected overall values from treated means relative to untreated populations; however $C(t)$ and $H(t)$ still exist to negative values suggesting caution in the stomach means cancer.

7.3 Figure 3: $\psi(t) = t$ (Classical Case)

- This represents the fractional derivative classically; thus, this is the most baseline study off which to draw more complicated occurrences going forward.
- Susceptibles (S) decrease somewhat expectedly but relatively quickly relative and significantly before stabilization occurs.
- Exposed (E) and Infected (I) expected to grow exponentially to epidemic proportions before dying down.
- Treated (T) has expected time due to treatment possibilities but not strong enough to prevent lifelong treatment improbabilities.
- Cancer (C) accumulates over time but relatively slowly as expected.
- Environmental (H) reaches infectious proportions but exits relatively quickly.

Remark: This major trajectory can serve as a control parameter for oscillatory dynamics or limited/extended dynamics.

7.4 Figure 4: $\psi(t) = 2t\sin(0.05t)$ (Oscillatory Kernel)

- The oscillatory kernel provides oscillatory proceedings for all compartments.
- Exposed (E) and Infected (I) seem to occur almost simultaneously three times.
- Susceptibles (S) ultimately decrease due to treatment but essentially occurs always due to ultimately exposed infection down the road.
- Cancer (C) climbs more slowly than Figure 3 but establishes oscillations.
- Environmental (H) fluctuates due to sinusoidal function but realistically only on the way up.

Remark 7.1 *Represents this population to be exposed potentially due to seasonal exposure or behavioural change.*

7.5 Figure 5: $\psi(t) = 0.6t\ln(2+t)$ (Logarithmic Kernel)

- The logarithmic kernel makes for slower dynamics.
- Infected (I) and Exposed (E) occur slowly with an exponential thrust which means these people transmit at exponential growth rates to others due to ultimately inevitable Infection dynamics at play regardless of precaution.
- Susceptibles (S) effectively clear relatively slowly as transmission was sustained; treatment was not final effectively but accelerated over time.
- Cancer (C) explodes much more quickly and subsequently more significantly as it becomes a larger visible concern over time.

- Environmental concentration (H) explodes quickly and stays up high as it is assumed people re-expose their preexisting irritants they've had over time.

Remark 7.2 *Suggests concern over delayed response to control measures.*

7.6 Figure 6: $\psi(t) = e^t$ (Exponential Kernel)

- The exponential kernel makes for overwhelming high dynamics and susceptibility in a negative way.
- Exposed (E) and Infected (I) grow extremely fast but die down.
- Treated (T) occurs immediately but drops quickly as treatment powers are not sustainable after initial diagnostic recommendations.
- Cancer (C) grows relatively low compared to other cases but based on other statistics, still appears over time.
- Environmental concentrations (H) rapidly rise but quickly decline.

Remark 7.3 *Suggests very high rates of temporal scaling and too aggressive of decay of memory for medicine purposes.*

7.7 Insights from the Numerical Results

1. The type of kernel makes a distinct difference relative to how quickly they reach endemic equilibrium: The higher demand the kernel operates at the lower health burden occurs down the road:
    - Classical = moderate dynamics;
    - Oscillatory kernels = cyclical dynamics;
    - Logarithmic kernels = prolonged infection;
    - Exponential kernels = suppressed prevalence.
2. Cancer occurs worst relative to $\psi(t) = 0.6t\ln(2+t)$ and best under $\psi(t) = e^t$.
3. Real world policy implications where:
    - Oscillatory $\psi(t)$: seasonality reinfection concerns;
    - Logarithmic $\psi(t)$: delay in treatment shows risks;
    - Exponential $\psi(t)$: treatment shows potential for clearance and death prevention; prolonged symptoms pose a significant clinical burden.

8 Conclusion

This manuscript presents a novel $\psi(t)$-Hilfer fractional-order model for Helicobacter pylori (HP), one of the most significant bacterial carcinogens worldwide, which seeks to establish stomach cancer dissemination through an indirectly transmitted population at large that cannot necessarily caution treatment precautions relative.

Thus, by creating direct transmission—where typical epidemiological models are found and subsequent indirect means of transmission and progression reliant upon gastric tolerability and maintenance/prevention compounded by treatment as a controlling contingency through which maintenance/existing dynamics may be used otherwise a modeling framework needs to be established that exceeds Hilfer's classical order option for practicality needs.

Thus, through theoretical results support existence/uniqueness/positivity/solution boundedness while stability analyses suggest the endemic equilibrium is globally asymptotically stable.

Therefore, numerical analysis with solutions based upon the Adams-Bashforth-Moulton predictor-corrector method through which the recognized attraction of surviving fractional parameters where bounded solutions were found bounded on spatial/temporal data integrity found in Table 3 practicality lends itself to believable findings of population values justifiable for practical study implementation when common parameters changed in other formulations based upon memory reactive consideration as opposed to linear simple summation/equation.

Thus, different fractional potentialities assessed relative to $\psi(t)$-the kernel choice - effectively serve as a situational variability component for otherwise longitudinally relatable systems for usability since they feature otherwise conservative at face value.

From $\psi(t)$–classical versus oscillatory versus logarithmic versus exponential impacted both a transient level incremental differences based upon time and subsequently longitudinal concerns cancer prevention/symptom existence through mortality effectiveness or lack thereof. Higher numbers treated mean lower infected populations and high gastric cancer prevention means males under extensive population dynamics average higher $\sum 1$ generations while relatively low findings under extensive hematological proportions occur.

Thus, compounded with modern psychological risk considerations derived through long treatment/treatment needs if only avoidant so long as assessed by avoidance effects derived from bacterial mortality—it seems no surprise that this specific bacterial is appealing to priority populations suggesting caution should be interjected with effective treatment if cross-contamination extends exposure according to intergenerational suggested implied risk from increased generations.

This study would benefit from continued HP-related carcinogenesis down the line with randomly determined disturbances based on today's treatment relative stochastic observation, or an epidemiological study fractionalized through multiple strains or even heterogeneous population components assessed where fractionalized intervention proves useful across longitudinally dynamic complicated modeling studies over time.

Ultimately this study serves as a practical foundational assessment for how fractional orderings operate not only as interactive findings but their lifetime relationships cumulatively provide dynamic stress for multiple compartment relationships holistically where millions known to suffer from HP complications implore appreciation sooner rather than later for common benefits worth understanding.

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