RESEARCH PAPER

Fractional Analysis of Brain Tumor–Immune System Interaction

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ABSTRACT

Brain tumors such as glioblastoma grow in complex ways that depend on previous conditions, which cannot be fully explained by traditional models. In this study, we use a fractional-order mathematical model to better understand the interaction between brain tumors and the immune system. The model incorporates past effects through Caputo fractional derivatives. The existence and uniqueness of the solution are established using the contraction principle. In addition, the stability of the system is analyzed under certain conditions. To support the theoretical results, numerical simulations are performed using the predictor–corrector method**.**

Keywords: Fractional order system, Caputo derivative, Tumor – immune interaction, Glioblastoma, Stability Analysis, Numerical simulation.

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INTRODUCTION

Fractional calculus offers a flexible way to describe systems where the present data depends on the past. This makes it especially useful in biology, where processes often show memory and gradual change. The concept took a major step forward when Caputo introduced a form of fractional derivative in 1967 that could be used with traditional starting values. This made it easier to apply in real-world problems. Later, in 2016, Atangana and collaborators proposed new types of fractional operators that avoided some of the limitations of earlier methods. These helped model smoother memory effects in biological systems. The groundwork for solving such equations was built earlier through detailed studies by Kilbas and colleagues in 2006, and the practical uses of fractional models were also shaped by Podlubny's in 1999.

Biological systems often rely on memory-based behaviour. Activities like how genes are expressed, how tissues heal, or how the immune system reacts are shaped by what has happened before. Back in 1993, Miller and others showed that fractional calculus offers a good way to represent these slow-changing processes like growth and decay over time[2]. Later, Li and Zeng in 2018 introduced improved numerical methods that enabled the simulation of complex biological processes governed by fractional dynamics with high precision [11]. These developments collectively opened new doors for applications in areas like epidemiology, neuroscience and oncology. Among these biological applications, cancer modelling—particularly of brain tumors—has gained significant interest. GBM stands out as one of the most aggressive and treatment-resistant forms of cancer. Characterized by rapid proliferation, invasive behaviour and immune evasion, glioblastoma often defies conventional therapy. Hanahan et al., in their landmark paper from 2000, described the "hallmarks of cancer," outlining the biological mechanisms that allow tumors like GBM to grow, resist cell death, and suppress immune surveillance [24]. Traditional integer-order differential models fail to account for the time-dependent and delayed nature of tumor–immune interactions. The immune response to tumors is far from instantaneous Joun et al., observed in 2020 that processes such as immune priming, recruitment, and activation evolve over extended periods, often days to weeks and are heavily influenced by memory effects [25]. In such cases, fractional-order models are better suited than classical models. These systems allow one to incorporate delays and nonlocality, reflecting the actual biological timelines of immune response. Losada and Nieto in their 2018 study, emphasized how fractional modelling of tumor–immune systems could effectively capture the memory-driven behaviour seen in clinical settings [20].

A tumor's behaviour is strongly shaped by its surrounding environment, which includes the immune system, structural tissues, and various chemical signals. In the case of glioblastoma, this environment is especially complex, making it hard to model using traditional methods. Back in 2014, Bai et al. showed that using fractional-order models can provide more stable and realistic descriptions of how tumor and immune cells interact[15]. Their work

highlighted that such models could even explain tumor dormancy, a state where cancer and immune cells live in balance. Additionally, the idea of immunoediting—where the immune system goes through phases of eliminating cancer, reaching balance, and then allowing it to escape—fits naturally within the flexible structure of fractional models. In a study from 2004, D'Onofrio et al. pointed out that delays in immune responses are crucial in understanding how tumors grow and survive over time[17]. The value of the fractional order, represented by α, acts like a memory setting: smaller values reflect deeper memory and slower reactions from the immune system, which can sometimes make responses more stable. Fractional-order systems also allow us to simulate different treatment strengths and lengths. For example, El-Saka and El-Mesiry in 2020 demonstrated how adjusting α can mimic different therapy strategies, helping researchers understand possible long-term effects[16].

In this study, we build on those ideas by developing a simplified mathematical model that focuses on the relationship between glioblastoma cells and immune activity using fractional-order equations. We assume tumor cells grow following a logistic pattern, are targeted by immune cells, and also stimulate more immune action in return. These biological interactions are captured using a system of fractional differential equations that include memory effects. This model helps to better reflect how the disease behaves over time and may offer useful insights into treatment planning by revealing how therapies might influence tumor-immune balance in the long run.

MODEL FORMULATION

This part shows how brain tumor cells and immune cells interact over time. Using a fractional-order approach with Caputo derivatives, it includes memory effects to reflect how past states affect current behaviour. The parameter α (between 0 and 1) controls how strong this memory is. The system focuses on two parts: tumor cells T(t) and immune cells I(t), offering a basic but realistic view of their dynamics. The classical version of the system can be described as:

$$\frac{dT(t)}{dt} = rT(t)\left(1 - \frac{T(t)}{K}\right) - \delta T(t)I(t) - \mu T(t) \quad ---(1)$$

$$\frac{dI(t)}{dt} = \beta T(t) - \gamma I(t) \quad ---(2)$$

where the tumor population follows logistic growth, is suppressed by immune action and undergoes decay due to therapeutic or natural mechanisms. The immune response is stimulated by the tumor and decays over time. However, in biological reality immune memory and adaptive behaviour depend not just on the current tumor burden but also on cumulative exposure over time. To reflect this, we convert the model into the fractional domain using the Caputo derivative, which better accommodates the initial value formulation for physical systems. The fractional-order system is thus written as:

$$D_t^{\alpha} T(t) = rT(t)\left(1 - \frac{T(t)}{K}\right) - \delta T(t)I(t) - \mu T(t) \quad ---(3)$$

$$^{C}D_t^{\alpha} I(t) = \beta T(t) - \gamma I(t) \quad ---(4)$$

Where $^{C}D_t^{\alpha}$ denotes the Caputo fractional derivative of order $\alpha \in (0,1]$. T(t) and I(t) represents the tumor cell and immune cell population at time t. The structure of the model captures essential biological processes as follows:

Tumor Proliferation: The term $rT(t)\left(1 - \frac{T(t)}{K}\right)$ represents logistic growth, where r is the tumor proliferation rate and K is the tumor's carrying capacity, reflecting the maximum viable size within brain tissue.

Immune-Mediated Killing: The interaction term $-\delta T(t)I(t)$ reflects immune surveillance, where δ represents the rate at which immune cells attack and destroy tumor cells.

Tumor Cell Death: The decay term $-\mu T(t)$ captures natural or therapy-induced cell death in the tumor population governed by the parameter μ.

Immune Stimulation: The immune equation includes $\beta T(t)$, which models how the presence of tumor cells activates immune response via antigen recognition and cytokine release.

Immune Cell Decay: The term $-\gamma I(t)$ models the natural loss of immune effectors due to exhaustion.

The model incorporates several biologically relevant parameters that define the tumor–immune interaction. The fractional order $\alpha \in (0,1]$ represents the memory depth of the system, with lower values indicating a stronger influence of historical states on the current behaviour. The parameter $r>0$ denotes the intrinsic growth rate of tumor cells, while $K>0$ signifies the carrying capacity, representing the maximum tumor size that the brain environment can support. The immune system's effectiveness in eliminating tumor cells is captured by the killing rate $\delta>0$, whereas $\mu \geq 0$ accounts for tumor cell loss due to natural death or therapeutic interventions. The parameter $\beta>0$ reflects the rate at which tumor cells stimulate the immune response, indicating how aggressively the immune system is activated in the presence of tumor cells. Lastly, $\gamma>0$ characterizes the natural decline of immune cells over time, due to factors such as cellular exhaustion or programmed cell death. All these parameters

are assumed to be non-negative and fall within biologically realistic ranges, supported by experimental evidence and findings reported in existing literature.

PRELIMINARIES

Definition 1 [18]: The Caputo derivative is a widely used fractional operator for modelling systems with memory while maintaining compatibility with classical initial conditions. For a function h(t) that is differentiable in the classical sense, the Caputo derivative of order α ∈ (0,1) is defined as:

$$D_t^{\alpha} h(t) = \frac{1}{\Gamma(1-\alpha)} \int_0^t \frac{h^n(s)}{(t-s)^{\alpha}}\, ds$$

Where, $D_t^{\alpha} h(t)$ denotes the Caputo fractional derivative of f(t) of order $\propto$ and $h^n(s)$ is the classical nth order derivative of h(t).

Definition 2 [19]**:** The Gamma function is a generalization of the factorial function to non-integer values. It is defined for all real numbers y > 0 by the improper integral:

$$\Gamma(y) = \int_0^{\infty} t^{y-1} e^{-t} dt$$

The Gamma function plays a fundamental role in fractional calculus, especially in defining fractional integrals and derivatives.

Definition 3 [20,21]**:** An equilibrium point y^* of a dynamical system is said to be *locally asymptotically stable* if solutions that start sufficiently close to y^* not only remain near y^* for all future times but also converge to y^* as t→∞. For fractional-order systems of order α∈(0,1], the equilibrium is locally asymptotically stable if all eigenvalues λ_i of the Jacobian matrix satisfy $|arg(\lambda i)| > \frac{\propto \pi}{2}$.

Definition 4 [22]**:** The predictor–corrector Adams–Bashforth–Moulton (ABM) method is a widely adopted numerical technique for solving fractional-order differential equations, particularly those involving Caputo derivatives. This method is designed to handle the memory-dependent structure inherent to fractional systems, allowing effective simulations for fractional orders 0 < α ≤ 1.

Consider the Caputo fractional differential equation:

$${}^{C}D_t^{\propto} z(t) = f(t, z(t)), \qquad z(t) = z_0$$

The ABM method proceeds in two phases: a predictor step (explicit) and a corrector step (implicit).

Predictor step (Adams–Bashforth type):

$$z_{n+1}^{p} = z_0 + \frac{1}{\Gamma(\alpha)} \sum_{j=0}^{n} b_{j,n+1} f(t_j, z_j)$$

Corrector step (Adams–Moulton type):

$$z_{n+1} = z_0 + \frac{1}{\Gamma(\alpha)} \left(\sum_{j=0}^{n} a_{j,n+1} f((t_j, z_j) + a_{n+1,n+1} f((t_{n+1}, z_{n+1}^{p}) \right)$$

Here, $b_{j,n+1}$ and $a_{j,n+1}$ are coefficients that depend on the step size h and the fractional order α, capturing the cumulative memory effect from the system's history. The predictor step yields a preliminary approximation z_{n+1}^{p}, which is refined in the corrector step to improve accuracy.

EXISTENCE AND UNIQUENESS

To ensure that the fractional-order tumor–immune model is mathematically well-posed, we analyse the existence and uniqueness of its solutions under biologically meaningful conditions. Let us consider the system (4,5) expressed in terms of the Caputo derivative of order α ∈ (0,1] with initial conditions T(0) = T₀ ≥ 0, I(0) = I₀ ≥ 0.

Let recast the system into the general form of a nonlinear fractional differential equation

$${}^{C}D_t^{\propto} Y(t) = G(Y(t)), \qquad Y(0) = Y_0 \qquad \text{----------(5)}$$

where Y(t) = [T(t), I(t)]^t and g: $\mathbb{R}^2 \to \mathbb{R}^2$ is defined by the right-hand sides of the system. Let f be continuous on a closed domain D ⊆ $\mathbb{R}^2$ and satisfy a Lipschitz condition in Y. Then the initial value problem of the system has a unique local solution on some interval [0, τ), τ > 0.

Letting $D = \frac{T^{\propto}}{\Gamma(\propto +1)}\, max\,(D_t, D_I)$

where $D_t = \left(1 - \frac{2T}{K}\right) - \delta I - \mu$ and $D_I = \beta - \gamma$.

Let define a closed and bounded subset D of $\mathbb{R}^2$ that contains all biologically feasible solutions, (i.e) positive values of T and I that do not exceed the carrying capacity or initial data. The function

$$G(Y) = [rT(t)\left(1 - \frac{T(t)}{K}\right) - \delta T(t) I(t) - \mu T(t), \quad \beta T(t) - \gamma I(t)] \qquad \text{---------(6)}$$

Define the norms as $||P|| = \bigcup_{t \in (0,T]} |P(t)|$, Then the norm of the matrix $A = [a_{ij}[t]]$ is defined by

$$||A|| = \sum_{t \in (0,T]} |a_{ij}[t]|$$

Now, we give the existence and uniqueness of the solution in the region $\Omega * (0, T]$.

Where $\Omega = (T, I)$: max $(|T|, |I| \leq A)$ Therefore, the solution of system is given as follows:

$$B(Y) = Y = Y_0 + \frac{1}{\Gamma(\propto)} \int_0^t (t - \theta)^{\alpha - 1}\, G\,(Y(\theta)) d\theta$$

$$B(Y_1) - B(Y_2) = \frac{1}{\Gamma(\propto)} \int_0^t (t - \theta)^{\alpha - 1}\, G\left(Y_1(\theta)\right) - G(Y_2(\theta)) d\theta$$

We can easily get the following inequality,

$$|B(Y_1) - B(Y_2)| \leq \frac{1}{\Gamma(\propto)} \left| \int_0^t (t-\theta)^{\alpha-1} G\left(Y_1(\theta)\right) - G\left(Y_2(\theta)\right) d\theta \right|$$

$$||B(Y_1) - B(Y_2)|| \leq \frac{T^{\propto}}{\Gamma(\propto +1)} max\,(D_t, D_I)||Y_1 - Y_2|| \leq D||Y_1 - Y_2||$$

If $D < 1$, then the mapping $Y = B(Y)$ is a contraction mapping and this gives the sufficient condition for existence and uniqueness of the solution of the system.

Hence, by the existence and uniqueness theorem for Caputo-type fractional differential equations, our tumor–immune system has a unique solution $Y(t) = [T(t), I(t)]^t$ on a finite interval $[0, \tau)$ for some $\tau > 0$. This result ensures that the model dynamics are deterministic and reproducible under specified initial conditions, which is crucial for both theoretical analysis and numerical simulations.

STABILITY ANALYSIS

To investigate the dynamical behaviour of the fractional-order tumor–immune system near its equilibrium states, we analyse the local stability properties using the Jacobian matrix method. In fractional-order systems, stability is not solely determined by the sign of the real parts of the eigenvalues, but also by their location in the complex plane relative to the fractional order. Specifically, for systems governed by Caputo derivatives of order $0 < \alpha \leq 1$, the system is locally asymptotically stable at an equilibrium point if all eigenvalues λ_i of the Jacobian satisfy the condition $|arg(\lambda i)| > \frac{\propto \pi}{2}$.

To locate equilibrium points, the right-hand sides of the system equations are set to zero. This yields a system of algebraic equations, leading to two steady states. The first is the tumor-free equilibrium $E_0 = (0,0)$ and the second is a positive equilibrium $E_1 = (T^*, I^*)$, where both cell populations are nonzero. From the immune equation, we immediately obtain $I^* = \frac{\beta}{\gamma} T^*$. Substituting into the tumor equation and simplifying gives a quadratic in T^*.

$$\frac{\delta\beta}{\gamma}(T^*)^2 + \left(\frac{r}{K} + \mu\right) T^* - r = 0$$

A positive solution of this equation yields the biologically meaningful steady state E_1, provided the parameters support positivity. The Jacobian matrix J of the system, linearized around any equilibrium (T,I) is given by:

$$J = \begin{bmatrix} r\left(1 - \frac{2T}{K}\right) - \delta I - \mu & -\delta T \\ \beta & -\gamma \end{bmatrix}$$

Evaluating the Jacobian at the tumor-free equilibrium $E_0 = (0,0)$, we obtain:

$$J(E_0) = \begin{bmatrix} r - \mu & 0 \\ \beta & -\gamma \end{bmatrix}$$

Which leads to two eigenvalues $\lambda_1 = r - \mu$ and $\lambda_2 = -\gamma$. The fractional-order stability condition for this system requires that the arguments of both eigenvalues lie outside a sector of angle $\alpha\pi$. In practical terms, this means that both eigenvalues must have negative real parts, and their phases must lie outside the instability sector centered around the real axis. If $r<\mu$, both eigenvalues are negative real numbers, satisfying the condition for local asymptotic stability when $\alpha \in (0,1]$. Therefore, the tumor-free equilibrium is stable when the tumor proliferation rate is lower than the natural or therapy-induced death rate.

STABILITY ANALYSIS WITH SPECIFIC PARAMETERS

To illustrate the application of the stability analysis in a real biological context, consider the fractional-order tumor–immune interaction system with the parameters set to biologically meaningful values: tumour growth rate $r=0.6$, carrying capacity $K=1000$, immune attack rate $\delta=0.001$, therapy-induced death rate $\mu=0.2$, immune stimulation rate $\beta=0.3$, immune decay rate $\gamma=0.1$, and fractional order $\alpha=0.9$. To determine the equilibrium point, the second equation yields $I^* = \frac{\beta}{\gamma} T^*$ Substituting this expression into the first equation provides a quadratic equation in T^*:

$$r\left(1 - \frac{T^*}{K}\right) - 3\delta T^* - \mu = 0$$

Plugging in the numerical values results in:

$$0.6\left(1 - \frac{T^*}{1000}\right) - 0.003T^* - 0.2 = 0$$

which simplifies to:

$0.4 = (0.0006 + 0.003)T^* = 0.0036T^* \Rightarrow T^* \approx 111.11.$

Thus, $I^* = 3 \times 111.11 \approx 333.33$, and the equilibrium point is approximately $(T^*, I^*) = (111.11, 333.33)$. Next, the Jacobian matrix at this equilibrium is computed using:

$$J = \begin{bmatrix} r\left(1 - \frac{2T^*}{K}\right) - \delta I^* - \mu & \delta T^* \\ \beta & -\gamma \end{bmatrix}$$

With $T^* = 111.11$ and $I^* = 333.33$, $We\ Complit$:

- $\frac{2T^*}{K} = \frac{222.22}{1000} = 0.2222,$
- $\delta T^* = 0.001\ X\ 333.33 = 0.3333$
- $-\delta T^* = -0.001\ X\ 111.11 = 0.1111.$

Then the Jacobian becomes:

$$J = \begin{bmatrix} 0.6\,(1 - 0.2222) - 0.3333 - 0.2 & -0.1111 \\ 0.3 & -0.1 \end{bmatrix} = \begin{bmatrix} -0.0666 & -0.1111 \\ 0.3 & -0.1 \end{bmatrix}$$

The characteristic equation is given by:

$$\lambda^2 - (a+b)\lambda + (ad - bc) = 0$$

With $\alpha = -0.0666$, $d = -0.1$, $b = -0.1111$ & $c = 0.3$

calculating the coefficients

- $a + d = -0.1666.$
- $ad - bc = (-0.0666)(-0.1)(-0.1111)(0.3) = 0.03999.$

The resulting equation $\lambda^2 + 0.1666\lambda + 0.03999 = 0$ Yields complex roots,

$$\lambda = -0.0833 \pm 0.1819i.$$

These eigenvalues have a negative real part, suggesting damping behaviour. However, stability for fractional-order systems requires satisfying the angle condition $|\arg(\lambda)| > \frac{\alpha\pi}{2}$. The argument of the complex eigenvalue is:

$$\theta = \tan^{-1}\left(\frac{0.1819}{0.0833}\right) \approx \tan^{-1}(2.183) \approx 65.24^{\circ},$$

While the threshold for stability under $\propto = 0.9$ is $\frac{0.9\pi}{2} \approx 81^{\circ}$. Since $65.42^{\circ} < 81^{\circ}$, the system is not stable under the current parameters set.

To enhance stability, the immune cell decay rate is increase to $\gamma = 0.3$ resulting in $I^* = T^*$. Assuming $T^* \approx 80$, then $I^* = 80$ and recalculating the Jacobian entries

- $a = 0.6\left(1 - \frac{160}{1000}\right) - 0.002 \times 80 - 0.25 = 0.094,$
- $d = 0.3, b = -0.002 \times 80 = -0.16$ & $c = 0.3.$

Now the trace becomes $Tr(J) = 0.094 - 0.3\ is$ -0.206, and the determination is:

$$Det(J) = 0.094 \times (-0.3) - (-0.16)(0.3) = 0.0198.$$

These values suggest complex eigenvalues with smaller imaginary part and stronger damping. In this setup, the eigenvalues are $\lambda = -0.15 \pm 0.08i$, With $\arg(\lambda) = \tan^{-1}\left(\frac{0.08}{0.15}\right) \approx 28^{\circ}$. For $\propto = 0.25$, the fractional angle threshold is $\frac{0.25\pi}{2} \approx 22.5°$, which is satisfied since $28° > 22.5°$.

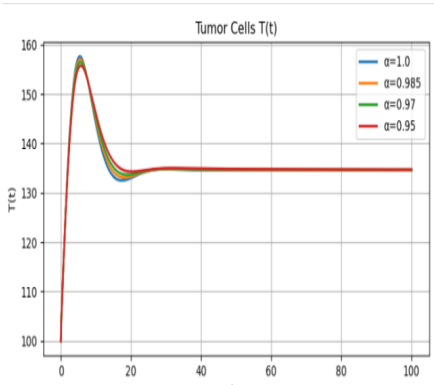
Hence, by tuning the fractional order and biological parameters such as decay rate and immune attack rate, the system can be steered into a locally asymptotically stable. This example highlights the sensitivity of fractional systems to parameter changes and demonstrates how fractional calculus provides flexible control. over the dynamic behaviour of tumour–immune models.

NUMERICALLY SIMULATE

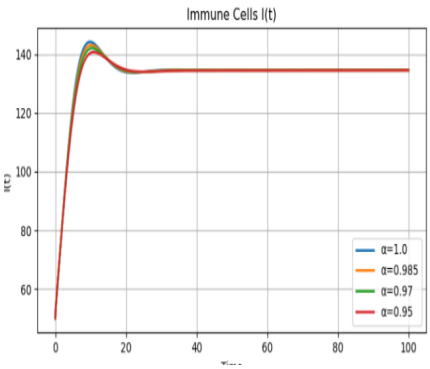
To examine the long-term dynamics of the tumor–immune interaction, we carried out a numerical simulation using the predictor–corrector Adams–Bashforth–Moulton scheme, which is especially suitable for handling fractional-order differential equations with Caputo derivatives. This approach effectively balances computational precision with the ability to capture memory-dependent behaviour in biological systems.

The parameter values used for the simulation were chosen based on existing biological evidence and match those employed in the earlier stability analysis. Specifically, the tumor growth rate was set to 0.6, with a carrying capacity of 1000. The immune cell killing rate was 0.001, while natural or therapy-induced tumor death was modelled with a rate of 0.2. To represent the immune system's activation, a stimulation rate of 0.3 was used, accompanied by a decay rate of 0.1 to capture immune cell exhaustion over time.

We began with an initial tumor population of 100 and an immune cell count of 50. The simulation was conducted over a time span from 0 to 100 with a step size of 0.1 to capture subtle variations in system behaviour. Implementing the scheme in Python allowed us to adjust parameters and fractional orders flexibly. Depending on the chosen memory level (i.e., the fractional order α), the system either exhibited damped oscillations or settled into a stable steady state, revealing the significant role memory plays in the progression and control of tumor dynamics.



(a)Tumor cell



(b) Immune cell

Observation: The simulation results provided a clear view of the system's behaviour under different memory conditions. For tumor cell dynamics, all trajectories ultimately approached a steady state, indicating stability in the system. Interestingly, lower values of α, which correspond to stronger memory effects, led to slower convergence and more gradual oscillations. In contrast,

higher values of α resulted in quicker stabilization, suggesting that memory modulates the speed of the system's response rather than its outcome. A similar pattern was observed in the immune cell population. Regardless of the memory depth, the immune response remained stable, though its peak activity and rate of decline varied. With lower α, the immune response exhibited delayed but smoother changes over time. This reinforces the robustness of the model's stability and shows how fractional-order dynamics influence the system's timing rather than its overall behaviour.

CONCLUSION

This work introduced a fractional-order model to describe the interaction between tumor cells and the immune system, with an emphasis on memory effects captured through Caputo derivatives. Through analytical techniques, we established that the model admits a unique solution and examined its stability characteristics. Both the tumor-free and positive equilibria were analysed using a Jacobian-based approach tailored for fractional systems. Numerical simulations supported the theoretical results and revealed how variations in memory depth influence system dynamics. Altogether, the findings suggest that incorporating fractional calculus into tumor modelling not only enhances biological realism but may also inform therapeutic strategies that depend on long-term immune adaptation.

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