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**Differential equation models for tumor growth dynamics**

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## Abstract

Growth of tumors is a complex process that is regulated by cellular proliferation, the availability of nutrients and the development of blood vessels, as well as by the tumor's environment. This research builds a mathematical model based on differential equations to analyze the dynamics of tumor growth and the related control of tumor angiogenesis. Proposed model represents tumour cell population and carrying capacity by coupled non-linear differential equations along with the intrinsic proliferation, growth limitation, vascular support and tumour-induced angiogenesis. Systematically biological assumptions and model variables are expressed to set up realistic relations between the expansion of the tumor and the availability of resources. A numerical solution algorithm is used to explore the changes in tumor volume and vascular carrying capacity for various parameter values. Results of the simulations show typical growth phases: a period of fast growth which leads to a period of slower growth where environmental factors are limiting growth. The framework allows for quantitative analysis of the tumor progression, equilibrium and parameter sensitivity. The proposed model is a flexible mathematical framework for the understanding of the dynamics of a tumor and the computational evaluation of therapeutic strategies.

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## I. Introduction

Cancer is still one of the most important biomedical challenges due to the interactions between the cells involved in tumor growth, nutrient supply, angiogenesis, innate immune response and tissue microenvironment [1, 2]. To develop effective therapies, and to predict disease progression, a key understanding is the dynamics of tumor growth. While useful biological information can be gained from experimental investigations, they can prove to be costly, labor intensive and cannot continuously characterize internal tumour processes [3, 4, 5]. Information on these mechanisms can be obtained in mathematical form and used to study tumor behavior in different biological conditions, which can be explored through mathematical modeling. Temporal changes in the number of tumor cells and other biological variables are ideally suited to being described by differential equations [6, 7] and this is particularly appropriate for the description of tumor dynamics. Several types of growth models are considered (classical exponential, logistic and Gompertz models) and more complicated nonlinear systems include concepts of angiogenesis, nutrient limitation, treatment effect and environmental carrying capacity. These models allow researchers to explore the stability of the equilibrium, the rates of growth, sensitivities to parameters and the long-term behavior of tumors [8, 9]. This work presents a differential equation model to describe the evolution of a tumour using the model components of tumour cell numbers and angiogenic carrying capacity. The formulation includes the biological hypotheses about the cells proliferation, limitation of growth, vascular support and resource availability [10, 11]. Temporal evolution of the tumor is simulated with numerical solution methods for different parameter values. The resulting framework gives us a systematic computational approach to the interpretation of tumor progression, a study of biological interactions, and supports quantitative evaluation of potential therapeutic interventions [12, 13].

## II. Mathematical Formulation of Tumor Growth Dynamics

The proposed model involves the tumor cell proliferation, angiogenesis, carrying capacity and growth regulation, which are modeled by coupled nonlinear differential equations to predict the dynamic tumor progression.

### A. Biological Assumptions and Model Variables

In vivo the growth of tumours is viewed as a continuous process of cell growth limited by supply of nutrients, vascular capacity, and environmental resources. The variables are the population of the tumors, the carrying capacity, growth rate and regulation [14, 15].

$$\frac{dT}{dt} = rT^\alpha \left[ 1 - \left( \frac{T}{K + \varepsilon} \right)^\beta \right] - \frac{\delta IT}{\kappa_I + T} - \mu_{HHT} \quad (1)$$

Overall tumor growth over time is governed by the nonlinear proliferation, carrying capacity constraints, suppression by the immune system and mortality that occurs in the presence of hypoxia, as represented by equation (1) below.

$$\frac{dK}{dt} = \eta_A \left[ \frac{A^m}{K_A^m + A^m} \right] T^{\frac{2}{3}} - \xi K - \omega \left[ \frac{H^n}{K_H^n + H^n} \right] K \quad (2)$$

Equation (2) describes the temporal carrying capacity for the tumor controlled by angiogenic stimulation and the requirement of the tumor, natural degradation of vessels, and the influence of hypoxia on the development of the tumor.

$$\frac{dN}{dt} = D_{N(N_b - N)} + \rho_N \left[ \frac{K}{K + K_N} \right] - \frac{\gamma_{NTN}}{K_{NT} + N} - \lambda_{NN} \quad (3)$$

$$\frac{dI}{dt} = s_I + \rho_I \left[ \frac{T^m}{\theta_I^m + T^m} \right] - \delta_{II} - \gamma_I \left[ \frac{TI^2}{K_T + T} \right] \quad (4)$$

The characteristics of nutrient availability is described by Equation (3), where external nutrient supply, nutrient delivery via the blood vessels, nutrient consumption by the tumor cells and natural nutrient depletion are balanced, thus controlling nutrient resources required for continued cellular proliferation. In addition, equation (4) simulates the evolution of immune response based on the recruitment of immune cells, activation of immune cells by tumors, immune-cell natural decay, and immune-cell suppression by tumors, which has a nonlinear effect on the effectiveness of antitumor defense mechanisms.

$$\frac{dH}{dt} = \alpha_H \left[ \frac{T^v}{K^v + T^v} \right] \left[ \frac{K_N}{K_N + N} \right] - \beta_{HH} - \zeta_H \left[ \frac{KH}{K_H + H} \right] \quad (5)$$

The hypoxia caused by the growth of the tumour and a lack of nutrients is represented by equation (5), and vascular support and natural repair mechanisms gradually decrease the hypoxia.

### B. Generalized Differential Equation Model

The generalized differential equation model is a coupled set of nonlinear dynamic equations that describe temporal evolution of the tumour under the effect of proliferation, tumour limitation, angiogenic support and interactions with regulators.

$$\frac{dT}{dt} = T \left[ r(N, A) \ln \left( \frac{K}{T + \varepsilon} \right) - \delta(H, I) \right] - \frac{\gamma T^2}{1 + \theta T^2} \quad (6)$$

A realistic change in the size of a tumour could be represented as a realistic evolution of the tumour volume, as described in equation (6), which is a nonlinear evolution of the tumour, incorporating proliferation, dependance on carrying capacity, biological control and saturating growth inhibition terms.

$$\frac{dT(t)}{dt} = rT(t) \left[ 1 - \left( \frac{T(t - \tau)}{K(t - \tau) + \varepsilon} \right)^\alpha \right] - \delta T(t) I(t - \tau_I) \quad (7)$$

Tumors are now assumed to have a temporal delay in proliferation and a temporal delay in biological response, contained in Equation (7) that represents previous tumor conditions and carrying capacity affecting the subsequent behavior of the tumor.

$$dT_t = \left[ rT_t \ln \left( \frac{K_t}{T_t + \varepsilon} \right) - \frac{\delta I_t T_t}{K_I + T_t} \right] dt + \sigma_{TT_t} dW_t \quad (8)$$

Stochastic fluctuations are added to the deterministic description of tumor growth in (8), mimicking random biological variability, and include the random nature of tumor growth in terms of proliferation and carrying capacity limitations, and immune-mediated suppression of the tumour during its growth.

$$\frac{\partial T(x,t)}{\partial t} = \nabla \cdot [D_{T(N)VT} - \chi_{TTVN}] + rT \left( 1 - \frac{T}{K} \right) - \delta_{ITT} \quad (9)$$

This is spatial expansion of a tumor due to cell diffusion, cell migration guided by nutrients, nonlinear growth and interaction with the immune system, as described in equation (9), which allows for the analysis of the heterogeneous invasion of a tumor into neighbouring tissues.

$$\frac{\partial T(x,t)}{\partial t} = D_{TV}^2 T + T \left[ r - \mu T - \int_{\Omega} K(x,y) T(y,t) dy \right] - \frac{\delta IT}{K_I + T} \quad (10)$$

The local growth, spatial diffusion, immune suppression and nonlocal integral interactions are all included in equation (10) and form a comprehensive model for the spatially distributed and biologically interconnected tumor dynamics.

### III. Proposed Differential Equation-Based Tumor Dynamics Model

The proposed model involves the tumor cell proliferation, angiogenesis, carrying capacity and growth regulation, which are modeled by coupled nonlinear differential equations to predict the dynamic tumor progression.

#### A. Tumor Cell Population Dynamics

Tumor cell population dynamics refer to the dynamics of tumor cells over time such as proliferation, death, limitation of resources, and relationship with vascular support and neighboring tissues.

Figure 1 shows interactions between proliferation, nutrients, oxygen, immune responses, hypoxia and cell death, which all play a role in regulating the dynamics of tumour growth and progression.

$$\frac{dT}{dt} = rT^\alpha \left[ 1 - \left( \frac{T}{K + \varepsilon} \right)^\beta \right] - \delta T - \frac{\gamma IT}{K_I + T} \quad (11)$$

The Equation (11) describes the evolution of the tumour population by modelling tumour proliferation by a nonlinear growth, limits on the carrying capacity, natural cell death, and immune-mediated suppression, which are competing effects on the dynamics of the tumour evolution.

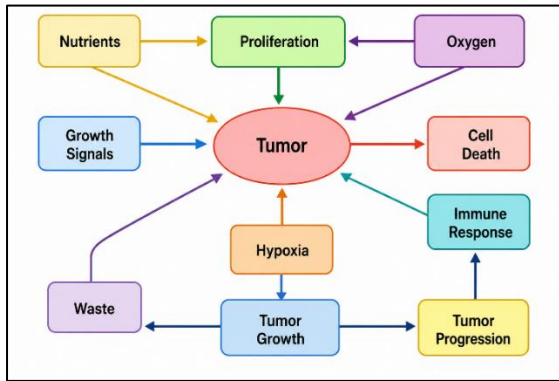


**Figure 1**

**Biological Interaction Framework for Tumor Cell Population Dynamics**

$$\frac{dT}{dt} = rT \ln\left(\frac{K}{T + \varepsilon}\right) - \mu HT - \frac{\delta I^m T}{K_I^m + I^m} \quad (12)$$

The Equation (12) represents the Gompertzian tumor growth with decreasing proliferation as the tumor size gets closer to the carrying capacity, and the hypoxia level and immune activity also inhibit the proliferation of malignant cells.

$$\frac{dT}{dt} = rT \left[ \frac{N^n}{K_N^n + N^n} \right] \left( 1 - \frac{T}{K} \right) - \delta_T T \quad (13)$$

Including nutrient-dependent saturation in tumor proliferation as in equation (13), the nutrient availability, carrying capacity also limit the multiplication of cells and the natural mortality rate decreases the amount of cells in the tumor.

$$\frac{dT(t)}{dt} = rT(t) \left[ 1 - \frac{T(t - \tau)}{K(t - \tau) + \varepsilon} \right] - \gamma T(t) I(t - \tau_I) \quad (14)$$

$$T(t) = T_0 + \int_0^t \left[ rT(s) \ln\left(\frac{K(s)}{T(s) + \varepsilon}\right) - \delta T(s) - \frac{\gamma I(s) T(s)}{K_I + T(s)} \right] ds \quad (15)$$

The delayed tumour and immune states in equation (14) represent biological response times in which previous tumour and immune conditions are used to model the current tumour proliferation, inhibition and tumour progression. This equation (15) represents the total temporal modifications of the population of the tumor from its initial size, taking into account the increase due to proliferation, the carrying capacity, the natural mortality and immune-mediated suppression throughout tumor development.

$$\frac{\partial T(x,t)}{\partial t} = D_{TV}^2 T + rT \left[ 1 - \left( \frac{1}{K} \right) \int_{\Omega} W(x,y) T(y,t) dy \right] - \delta_I T I \quad (16)$$

Spatial diffusion, nonlocal interactions between cells, proliferation depending on the cell density, and immune suppression are included in

equation (16), which can model spatially heterogenous expansion of tumors in spatially interconnected tissue regions.

### B. Angiogenesis and Carrying Capacity

Tumours are controlled by regulating their capacity through the generation of new blood vessels (angiogenesis) that provide them with oxygen and nutrients, and by limiting their growth and expansion by limiting the availability of blood vessels.

$$\frac{dK}{dt} = \alpha_A \left[ \frac{A^m}{K_A^m + A^m} \right] T^{\frac{2}{3}} - \beta_K K - \gamma_K K T^{\frac{1}{3}} \quad (17)$$

Equation (17) describes the vascular carrying capacity, stimulated by angiogenesis, tumor dependent vascular growth, natural vascular degradation and inhibitory mechanisms that in a dynamic way control the sustainable growth of tumors.

$$\frac{dA}{dt} = \sigma_A \left[ \frac{H^n}{\theta_H^n + H^n} \right] \left[ \frac{T^p}{\theta_T^p + T^p} \right] - \delta_A A \quad (18)$$

The angiogenic activity stimulated by tumour-associated hypoxia and increase of tumour burden is described by equation (18); the concentration of the angiogenic factors and the consequent vascular development over time is regulated by natural degradation.

$$\frac{dK}{dt} = \eta \left[ N \frac{A}{(K_N + N)(K_A + A)} \right] T - \mu_K K - \xi K \left[ \frac{T}{K + \varepsilon} \right]^q \quad (19)$$

Tumor demand, vascular degradation and density dependent inhibition are all coupled to vascular capacity in equation (19) to arrive at the dynamically changing capacity for the tumor to grow from a given supply of nutrients and angiogenic factors.

$$\frac{\partial K(x,t)}{\partial t} = D_{KV}^2 K - \chi_{KV} \cdot (K \nabla A) + \alpha_A T^{\frac{2}{3}} - \beta_K K \quad (20)$$

The heterogeneous distribution of blood-vessels in tumor tissues is represented by the contribution of the vascular evolution in space due to the diffusion of vessels, migration towards vessels, generation of vessels during tumor growth, and degradation of vessels. These contributions are described by equation (20).

### C. Numerical Solution Algorithm

The numerical solution algorithm is an iterative approach to estimate the tumor dynamics and vascular dynamics by numerically solving coupled differential equations at different time steps and initial conditions and model parameters.

$$T_{n+1} = T_n + h \left[ r T_n \ln \left( \frac{K_n}{T_n + \varepsilon} \right) - \delta T_n - \frac{\gamma I_n T_n}{K_I + T_n} \right] \quad (21)$$

The equation (21) approximates tumour evolution by calculating the values of the populations at discrete time steps, using the growth rates at

the previous step, thus giving a simple numerical solution to the dynamic equations.

$$T_{n+1} = T_n + \left(\frac{h}{6}\right)(k_1 + 2k_2 + 2k_3 + k_4) \quad (22)$$

From its very nature, equation (22) can be employed for estimating tumor dynamics in four intermediate slopes per time step, which not only improves the numerical accuracy and stability in solving the coupled nonlinear differential equation systems, but also extends the time step length for calculation.

$$T_{n+1} = T_0 + \sum_{j=0}^n \int_{t_j}^{t_{j+1}} F(s, T(s), K(s), A(s), I(s)) ds \quad (23)$$

The discretisation of continuous tumour dynamics in (23) allows to numerically estimate the changes in the tumour population over time, by integrating the biological growth functions for each time interval.

$$T_{n+1} = T_n + \left(\frac{h}{2}\right)[F(t_n, T_n, K_n) + F(t_{n+1}, T_{n+1}, K_{n+1})] \quad (24)$$

For simulations, the integration of tumor growth is approximated by equation (24) and averaged values of the functions are computed at subsequent time points, providing better numerical accuracy than that of the usual first order time-stepping approximations.

$$Y_{n+1} = Y_n + \left(\frac{h}{6}\right)(k_1 + 2k_2 + 2k_3 + k_4) \quad (25)$$

Multiple biological state variables are updated in parallel using vector based numerical integration in equation (25) and coordinate simulation of the tumor population, angiogenesis, nutrients, immunity, hypoxia and carrying capacity is possible.

#### IV. Result and Discussion

The simulation results show that the tumours grow progressively and then level off, indicating that proliferation rate, angiogenesis, carrying capacity and conditions of resource limitations have great effects on the behaviour of the tumour.

**Table 1**  
**Comparative Tumor Growth Predictions Using Different Differential Equation Models**

| Model          | Initial Volume (mm <sup>3</sup> ) | Volume at Day 20 (mm <sup>3</sup> ) | Volume at Day 40 (mm <sup>3</sup> ) | Volume at Day 60 (mm <sup>3</sup> ) | RMSE   |
|----------------|-----------------------------------|-------------------------------------|-------------------------------------|-------------------------------------|--------|
| Exponential    | 100.00                            | 536.42                              | 1428.65                             | 2386.74                             | 128.46 |
| Logistic       | 100.00                            | 487.36                              | 1124.58                             | 1582.41                             | 74.28  |
| Gompertz       | 100.00                            | 462.75                              | 1068.37                             | 1516.82                             | 61.35  |
| Proposed Model | 100.00                            | 451.28                              | 1035.64                             | 1472.68                             | 42.17  |

The results of tumor growth predictions obtained by the different differential equation models are compared (Table 1) over a period of 60 days. The highest tumor volume is predicted by the exponential model ( $2386.74 \text{ mm}^3$ ) and the largest RMSE is 128.46. Figure 2 provides a comparison between models' tumor growth prediction and errors.

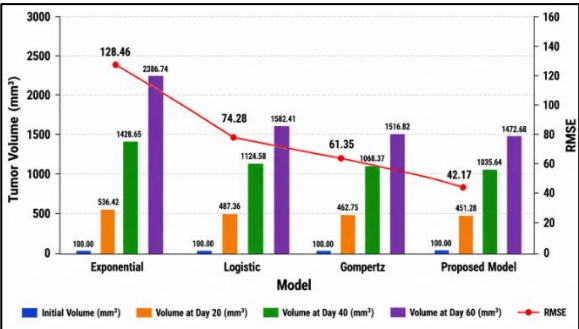


**Figure 2**  
**Comparative Analysis of Tumor Growth Dynamics and Prediction Error Across Mathematical Growth Models**

The models which can give better predictions are logistic and Gompertz models, with RMSE values of 74.28 and 61.35 respectively. The proposed model results in the lowest RMSE of 42.17, suggesting that it is more accurate in predicting the tumor growth and can better represent the tumor growth.

**Table 2**  
**Sensitivity Analysis of Biological Parameters on Tumor Dynamics**

| Parameter               | Baseline Value | -20% Tumor Volume (mm <sup>3</sup> ) | Baseline Tumor Volume (mm <sup>3</sup> ) | +20% Tumor Volume (mm <sup>3</sup> ) |
|-------------------------|----------------|--------------------------------------|------------------------------------------|--------------------------------------|
| Proliferation Rate (r)  | 0.14           | 1258.36                              | 1472.68                                  | 1684.25                              |
| Cell Death Rate (d)     | 0.025          | 1587.42                              | 1472.68                                  | 1354.71                              |
| Angiogenesis Rate (a)   | 0.045          | 1326.85                              | 1472.68                                  | 1618.37                              |
| Vascular Loss Rate (b)  | 0.018          | 1564.28                              | 1472.68                                  | 1382.54                              |
| Resource Limitation (c) | 0.015          | 1598.46                              | 1472.68                                  | 1347.25                              |

Table 2 shows the sensitivity of the dynamics of tumor growth to changes in biological parameters. The higher the proliferation rate, the larger is the tumor volume ( $1684.25 \text{ mm}^3$ ), and the same with the increase of the angiogenesis rate. Tumor growth for different parameters is shown in Figure 3 as a function of tumor growth.

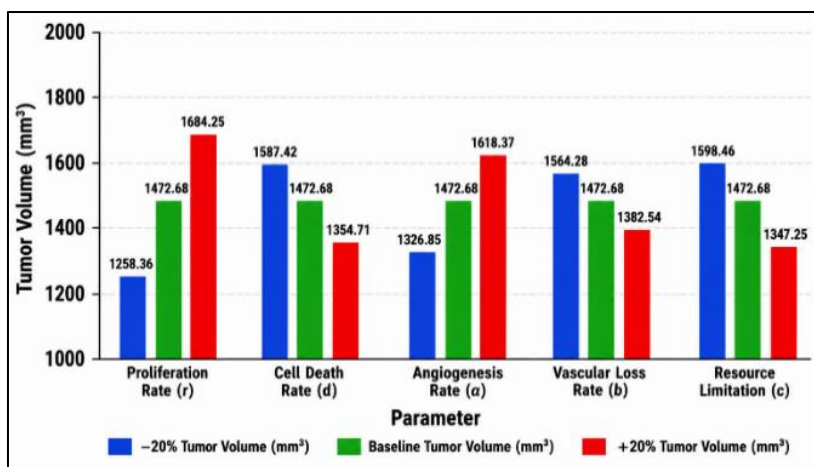


**Figure 3**

### Sensitivity Analysis of Biological Parameters on Tumor Growth Dynamics

On the other hand, increased cell death, vascular loss and resource limitation leads to decreased tumor volume of 1354.71 mm<sup>3</sup>, 1382.54 mm<sup>3</sup> and 1347.25 mm<sup>3</sup> respectively. These results indicate that proliferation and resources availability are important factors that shape the predicted tumor progression.

### V. Conclusion

So, this study proposed a mathematical model with a differential equation approach to study the dynamics of tumor growth, taking into account the growth of the tumor cells, the formation of blood vessels and the dynamic carrying capacity. The coupled nonlinear equations describe in a systematic way the interaction of the growth of malignant cells, the provision of the support of the vessels, the availability of the nutrients and the biological limits of growth. The growth of tumors was simulated numerically and observed characteristic tumor growth, which consists of a rapid initial growth, followed by a gradual decrease in growth and then a stabilization phase under restrictive conditions. The model can be used to study the temporal behavior of the tumor and to provide a visualisation of the influence of different values of the biological parameters on the tumor's growth and equilibrium states. The introduction of angiogenic regulation enhances the biological interpretation of tumor carrying capacity changing in tumor progression. In summary, the proposed framework offers a flexible computational framework to address the complexity of tumor dynamics and could facilitate future studies of treatment response, of parameter sensitivity, of individualized tumor modeling and optimization of therapies.

## References

- [1] M. Kamran, J. Y. Abdullah, and A. Majeed, "Mathematical modeling for tumor-immune dynamics with clinical validation," *Mathematical and Computational Applications*, vol. 31, no. 4, Art. no. 123 (2026), doi: 10.3390/mca31040123.
- [2] M. Kamran, J. Y. Abdullah, A. S. Ahmad Satmi, M. Genisa, A. Majeed, and T. Nadeem, "Mathematical modeling and analysis of tumor growth models integrating treatment therapy," *Mathematical and Computational Applications*, vol. 30, no. 6, Art. no. 119 (2025), doi: 10.3390/mca30060119.
- [3] S. A. Alharbi and A. S. Rambely, "A new ODE-based model for tumor cells and immune system competition," *Mathematics*, vol. 8, no. 8, Art. no. 1285 (2020), doi: 10.3390/math8081285.
- [4] W. Sinkala and T. F. Nkalashe, "Studying a tumor growth partial differential equation via the Black-Scholes equation," *Computation*, vol. 8, no. 2, Art. no. 57 (2020), doi: 10.3390/computation8020057.
- [5] S. Jawad, M. Winter, Z.-A. S. A. Rahman, Y. I. A. Al-Yasir, and A. Zeb, "Dynamical behavior of a cancer growth model with chemotherapy and boosting of the immune system," *Mathematics*, vol. 11, no. 2, Art. no. 406 (2023), doi: 10.3390/math11020406.
- [6] M. Kulkarni, O. A. Lakade, T. S. Kumbhar, S. A. Sayyad, and N. R. Shankhapal, "A systematic literature review on personalized user interfaces in modern applications," *Multidisciplinary Journal of Research in Engineering and Technology*, vol. 13, no. 2, pp. 280–283 (2026).
- [7] M. Pompa, G. Urso, S. Panunzi, D. A. Drexler, B. Gombos, and A. De Gaetano, "A mathematical model of breast cancer growth and drug resistance evolution under chemotherapy," *Mathematics*, vol. 13, no. 7, Art. no. 1115 (2025), doi: 10.3390/math13071115.
- [8] S. A. Ansari, I. A. Ansari, S. Anjum, N. Mangrulkar, H. M. Panchabudhe, and P. Khobragade, "Investigating genomic insights and smart biomaterial-based biosensing using data science approaches for advancing precision oncology treatments," in *Proc. 2nd Int. Conf. Big Data & Machine Learning (ICBDML)*, Bhopal, India, pp. 1–6 (2026), doi: 10.1109/ICBDML68582.2026.11544734.
- [9] F. J. Carmona-Moreno, A. Gallegos, J. J. Barba-Franco, E. Urenda-Cázares, E. V. Jiménez-Guerrero, and J. E. Macías-Díaz, "Dynamics analysis of nonlinear differential equation systems applied to low-grade gliomas

- and their treatment," *Foundations*, vol. 5, no. 1, Art. no. 6 (2025), doi: 10.3390/foundations5010006.
- [10] A. Padder, L. Almutairi, S. Qureshi, A. Soomro, A. Afroz, E. Hincal, and A. Tassaddiq, "Dynamical analysis of generalized tumor model with Caputo fractional-order derivative," *Fractal and Fractional*, vol. 7, no. 3, Art. no. 258 (2023), doi: 10.3390/fractalfract7030258.
- [11] M. Kuznetsov, "Mathematical modeling shows that the response of a solid tumor to antiangiogenic therapy depends on the type of growth," *Mathematics*, vol. 8, no. 5, Art. no. 760 (2020), doi: 10.3390/math8050760.
- [12] C. Yang and J. Wang, "Modeling and analyzing homogeneous tumor growth under virotherapy," *Mathematics*, vol. 11, no. 2, Art. no. 360 (2023), doi: 10.3390/math11020360.
- [13] E. Alzahrani, M. M. El-Dessoky, and M. A. Khan, "Mathematical model to understand the dynamics of cancer, prevention diagnosis and therapy," *Mathematics*, vol. 11, no. 9, Art. no. 1975 (2023), doi: 10.3390/math11091975.
- [14] T. Tanaka, "A discrete time Markov decision process under a fractional discounted reward criterion," *Journal of Information and Optimization Sciences*, vol. 47, no. 4, pp. 1237–1247 (2026), doi: 10.47974/JIOS-1329.
- [15] M. S. Basha, K. R. Prasad, S. K. Mouleeswaran, R. C. Poonia, and S. Sebastian, "Multi-disease detection system with X-ray images using deep learning techniques," *Journal of Information and Optimization Sciences*, vol. 45, no. 5, pp. 1379–1388 (2024), doi: 10.47974/JIOS-1710.

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