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Research Article

Mathematical Model of Cancer with Ordinary Differential Equations

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Abstract: The cancer cell starts dividing, infiltrating neighboring tissues and traveling throughout the lymphatic system. While there are ways to stop the spread of disease or get rid of infected cells, most of the approaches are unable to identify the early warning indicators of such an occurrence. Using diverse types of differential equations, especially ordinary differential equations (ODEs), is a helpful catalyst that experts employ. Using differential equations, researching resistance to chemotherapy, forecasting potential treatment failure, or evaluating the result and prognosis following various forms of therapy. Living things always include cancer cells, but a biological regulatory system keeps them from spreading to a dangerous degree (think overpopulation vs. natural resources). Therefore, the most efficient method of determining when to effectively intervene with the tumor growth is to use the cytokinetic method of quantitatively assessing the cancer cells' progression. Cancer Metabolism: One of the main characteristics of cancer is metabolic reprogramming, in which the metabolism of cancer cells is changed to fuel their explosive growth and multiplication. New models of cancer metabolism investigate the roles that dysregulated metabolic pathways play in the genesis and spread of tumors, providing prospective avenues for therapeutic intervention. The mathematical models of tumor growth modeling of ordinary differential equations (ODEs cancer). The tumor grows voraciously, and the scientists and mathematicians who tried to have a better understanding grow. The study of such treatments on models of tumor growth leads to one or more ODEs. Which gives some ideas on the relation between equations and tumor growth in cancer cells introduce ODEs to provide mathematical models of tumor growth. The dynamics of tumor cells and their growths through clinical, experimental, and theoretical approaches, new ideas for different cancer therapies are developed with the goal of controlling and reducing the death rate for earlier diagnosis. The kinetics of tumor cell proliferation and its treatment approach were covered in this research. In order to comprehend the proliferation of tumor cells, we expanded the study and xamined a feew basic mathematical models.

***Keywords*:** mathematical models, ordinary differential equations, tumor qrowth, simple models, continuous function

MSC: 34A30, 92B05, 92-10

1. Introduction

The tumor-growth and therapeutic interactions using the ordinary and delay differential equations. We investigate how adoptive cellular immunotherapy affects the model and outline the conditions that lead to tumor eradication.

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Cancer diseases and one of the most difficult to cure clinically is cancer. Consequently, there is a lot of studythe tumor cells and the immune system interact. The use of ordinary differential equations in mathematical models is crucial for comprehending the dynamics and monitoring tumor and immunological populations throughout time. Despite the fact that research on tumor immune dynamics.

The simple models of tumor growth and treatment on single nonlinear ODEs to the equation of exponential growth,

$$\frac{dM}{dt} = \lambda M \tag{1}$$

where, λ is a constant, and $M(t)$ is a continuous function on time t and represents the number of cells in tumor cells.

The generalized a nonlinear first order (ODEs) incorporate growth deceleration.

$$\frac{dM}{dt} = f(M) \tag{2}$$

The systems of ODEs consider two cell populations. The models are based on systems for ordinary differential equations.

2. Mathematical modeling of tumor growth and treatment

2.1 *Ordinary d equations of tumor growth*

Cancer is a disease that occurs when some body cells grow out of control and spread to other body regions. The human body is made up of trillions of cells [1]. Human cells often divide into new cells as needed in the body, a process called cell division.

A tumor is a mass of tissue that may resemble swelling. Not all tumors are cancerous.

Suppose that the rate of change for the population is proportional to the number of individuals at any time in the differential equation [2].

$$\frac{dY}{dt} = Ky. \tag{3}$$

with the initial condition $Y(t_0) = Y_0$. The population Y positive and increasing to different biological factors and mutation $k > 0$. The solution is [3]

$$Y = Y_0 e^{t-t_0}. \tag{4}$$

A generalization for the exponential model is given as:

$$\frac{dY}{dt} = aY^b. \tag{5}$$

Consider the tumor cell population by DE of the form

$$\frac{dY}{dt} = aY - bY^2,\ Y(t_0) = Y_0. \tag{6}$$

where $a > 0$ chooses $b > 0$ in order to inhibit the growth for y demanded by reality. This equation is termed a logistic equation, which is called the logistic [4] law of cancer tumor growth and can be solved by the separation of variables method. Let t_0 then

$$Y = Y_{max} = \frac{ab}{1 + (\frac{a/b}{Y_0} - 1)e^{-at}}. \tag{7}$$

$$Y_{max} = lim_{t \longrightarrow \infty} Y = \frac{a}{b}. \tag{8}$$

Consider $V(T)$ [5] it for the case of tumor volume. The growth of cancer can be modeled by

$$\frac{dV}{dt} = KV. \tag{9}$$

where $V(T)$ denotes the volume for dividing cells at time t, with initial volume $V(0) = V_0$.

If the growth is proportional [6] to the surface area and the death is proportional to the tumor size t [7].

$$\frac{dV}{dt} = aV^{2/3} - bV. \tag{10}$$

This model is applied to describe human tumor growth. The tumor doubling time t was introduced to measure and quantify how fast a tumor grows and quantify the growth rate, which is $\frac{ln2}{k}$ as the [8] tumor becomes larger, the doubling time t of the total tumor volume continuously increases. The exponential modeling predicts early growth well.

$$V(t) = V_0 exp[\frac{k}{a}(1 - e^{-at})]. \tag{11}$$

where k and a are positive constants. It states that tumors grow and more slowly with that passage [9] of time, the growth rate, given by the following nonlinear equation ODEs $V_0 e^{k/a}$ where k is constant.

Let model the growth of malignant tumors is given by

$$\frac{dp}{dt} = KP(lnP_x - lnP) \tag{12}$$

where P is the population of cancer cells, and k is constants.

Let's use nonlinear ODEs and assume that is real, and k is the host carrying capacity [10].

The value is to be understood as a limit; taking the limit gives a Gompertz equation.

$$f(M) = \frac{\mu M}{v}[1 - (\frac{M}{k})^{v}],\ \mu > 0,\ k > 0. \tag{13}$$

where $v > 0$ is the real and k is host carrying capacity. For the value $v = 0$ to be understood as a limit.

$$\frac{dM}{dt} = -\mu ln\frac{M}{K}. \tag{14}$$

The tumor treatments on cell killing by external agents, a very common assumption is the treatment modifies this equation [11]

$$\frac{dM}{dt} = -\varepsilon c(t) + f(M). \tag{15}$$

Here, ε is the positive constant, the strength for the chemotherapeutic agent, and $c(t)$ is the agent concentration at the location of the tumor cells. The mathematicians and scientists devoted their studies to such diseases using their mathematical modeling on [11] ODEs.

3. Modeling tumor treatment

The dynamics of the number of tumor cells at the time t, $M(t)$ is described by differential equation forms for the growth law, where $f(M)$ is the tumor cell growth dynamics, $G(t, M)$ describes the effects of the drug on the system, and an indicator function (= 1) if $t = t_{surgery}$ [12], and zero otherwise. Surgery is assumed to be instantaneous and remove [13] a fixed fraction of $exp(-K_s)$ of the tumor cells, where k is the fraction of removed cells during surgery [14].

$$\frac{dM}{dt} = f(M) - G(t, M) - K_s I(t = t_{surgery})M. \tag{16}$$

The mathematical model of [15] inhibition proposes is the following models:

$$\frac{d}{dt} = (\frac{k}{M}). \tag{17}$$

$$\frac{dk}{dt} = -\varepsilon c(t) + \beta M - \gamma K M^{2/3}. \tag{18}$$

where the first equation represents the endothelium compartment, and the second equation [16] represents the tumor compartment.

Differential equation models easily include the impacts of both cancer therapy options [17].

$$\frac{dc}{dt} = -\lambda c ln(\frac{c}{K}) - \zeta c. \tag{19}$$

$$\frac{dK}{dt} = \phi c - \phi K c^{2/3} - \Gamma K g(t). \tag{20}$$

In the simple case, anti-tumor treatment induces a continuous tumor cells kill with strength $0 \leq \zeta \geq 1$ conceivable as the chemotherapy or immunotherapy.

4. Ordinary differential equaition based model for tumor cell and analysis

4.1 *Model formulation*

The model formulation that is being developed is called the normal-tumors immune-unhealthy.

The following is required of the formulation:

$M(t) :=$ normal cells at the time t.

$T(t) :=$ tumor cells at the time t.

$I(t) :=$ immune system at the time t.

The model is based by following assumptions:

There is a high growth rate in the cells populations. Due to this.

5. The method solving differential equaition

Depending on the exact model being utilized, there are multiple ways to solve differential equations in mathematical modeling of cancer. This is a broad synopsis of the procedure:

Create the Model: You must create a mathematical model that explains how cancer cells behave and interact with their surroundings before you can begin to solve any equations. Differential equations are frequently used in this to account for changes over time.

Discretization: In order to solve continuous differential equations in your model numerically, you'll probably need to discretize them. Usually, this entails segmenting space and time into tiny grids or intervals.

Select a Numerical approach: To solve the discretized equations, choose a suitable numerical approach. Finite element, finite difference, and finite volume methods are common techniques. The decision is based on various considerations, including the model's complexity and the required level of accuracy.

Put the Numerical Solver into Practice: Write code to put the selected numerical technique into practice. Programming languages like Python, MATLAB, or C++ may be used for this.

Establish initial and boundary conditions. For each variable in your model, such as the initial cancer cell distribution, specify the initial circumstances.

Determine Parameters: Establish the parameters of your model, including diffusion coefficients, growth and death rates from the tissues normal cells to transition into tumor cells will take time [18].

Logistics when there is not a tumor and immune cells as:

$$\frac{dM}{dt} = rM(1\beta_1 M). \tag{21}$$

But with the interaction between tumor and immune cells, the equations [19] become

$$\frac{dM}{dt} = rM(1\beta_1 M) - \eta MI\gamma MT \tag{22}$$

The parameters

a. r suppose indicative the populations for the normal cells growth rate.

b. $\frac{\beta_1}{r}$ denote the cell saturations.

c. η indicative the rate of interaction between the normal cells and the immune cells.

d. γ indicative the rate of interaction between the normal cells and tumor cells.

The growth of the tumor cells is governed by the logistic population model when there is not an interaction with normal cells and immune cells.

$$\frac{dT}{dt} = \varepsilon_1 T(1\varepsilon_2 T). \tag{23}$$

and Including that the interaction of the cells,

$$\frac{dT}{dt} = \varepsilon_1 T(1\varepsilon_2 T) + \beta_2 MT - \varepsilon_3 TI. \tag{24}$$

The parameters

a. ε_1 indicative the growth rate of the tumor cells.

b. $\frac{\varepsilon_1}{\varepsilon_2}$ denotes the tumor cell saturation.

c. β_2 indicative of the interaction between normal cells and tumor cells.

d. ε_3 indicative of the interactions between the tumor and immune cells.

In modeling the immune system, we have to consider the constant source of immune system booster (σ). The immune system depletes at a natural rate [13] δ, s. The reaction rate is

$$R(s) = \frac{As}{K+s}. \tag{25}$$

[A, K constants]

$$\frac{dI}{dt} = \sigma - \delta I + \frac{\rho MI}{m+M} + \frac{\rho_1 TI}{m_1+T} + \mu MI + \mu_1 TI. \tag{26}$$

$m+M$ cell appearance stimulation the immune system to respond.

a. ρ indicative of this response rate.

b. m indicates the immune system rate.

c. μ indicates the reduction of the immune cells to the interaction with the normal cells.

d. μ_1 indicates the reduction of the immune cells to their interactions with the tumor cells.

The second term depicted by [20] $\frac{\rho_1 TI}{m_1+1}$ inhibits the tumor cells.

The following system expresses in the normal-tumor-immune-model unhealthy diet model.

$$\frac{dM}{dt} = rM(1-\beta_1 M) - \eta MT - \gamma MT. \tag{27}$$

$$\frac{dT}{dt} = \varepsilon_1 T(1-\varepsilon_2 T) + \beta_2 MT - \varepsilon_3 TI. \tag{28}$$

$$\frac{dI}{dt} = \sigma - \delta I + \frac{\rho MI}{m+M} + \frac{\rho_1 TI}{\mu_1 + T} + \mu MI + \mu_1 TI. \tag{29}$$

All parameters r, β_1, η, γ, ε_1, ε_2, β_2, ε_3, σ, δ, μ, μ_1, ρ, ρ_1, are positives.

6. Model aalysis

6.1 *Postivity of solutions*

The model of the equation above displays the interaction between normal cells and tumor cells with the influence of the immune cell system. It is common sense. The M, T, and I are positive. This is because we do not have negative populations and values. In addition to the fact. All parameters are positive values; they are also values between zero and one. Suppose that,

$$\Gamma = (M, T, I) \in R$$

The next theorem shows the positivity of solutions.

7. Theorem 1

The dynamic system region of the mathematical model $\Gamma \subset R$, is positive invariant, and a positive solution exists t. From the mathematical model of the normal cells, we have this equaition,

$$\frac{dM}{dt} \leq \gamma M(t) \gamma \beta_1 M(t) \tag{30}$$

We have [21],

$$\frac{1}{M(t)2}\frac{dM(t)}{dt} - \frac{\gamma M(t)}{M(t)2} \leq -\gamma\beta_1. \tag{31}$$

Suppose that,

$$S = M(t)^{-1}, \tag{32}$$

then [22],

$$\frac{dZ(t)}{dt} = -M(t)^{-2}\frac{dM}{dt}. \tag{33}$$

This gives,

$$\frac{-dS(t)}{dt} - \gamma z \leq -\gamma\beta_1, \tag{34}$$

$$\frac{Z(t)}{dt} + \gamma z \leq \gamma\beta_1. \tag{35}$$

This has been reduced to a linear [23] first-order equation (ODEs), solving by using an integrating factor, we have,

$$I \cdot F = e^{\int \gamma dt} = e^{\gamma t}. \tag{36}$$

Multiply through by $e^{\gamma t}$.

$$e^{\gamma t}\frac{S(t)}{dt} + e^{\gamma t}\gamma s \leq e^{\gamma t}\gamma\beta_1. \tag{37}$$

Integrating equation [24],

$$Se^{\gamma t} \leq \frac{e^{\gamma t}\gamma\beta_1}{\gamma} + C. \tag{38}$$

$$S \leq \beta_1 + Ce^{-\gamma t}, \tag{39}$$

denote that $S = \frac{1}{M}$,

$$\frac{1}{M} \leq \beta_1 + Ce^{-\gamma t}. \tag{40}$$

The solution to this equation is given by

$$M(t) \leq \frac{1}{\beta_1 + Ce^{-\gamma t}}. \tag{41}$$

As $t \to \infty$, the solution of the equation is given by

$$M(t) \leq \frac{1}{\beta_1}. \tag{42}$$

From the (ODEs) describing the behavior of tumor cells, we obtain, that

$$\frac{dT}{dt} \leq \varepsilon_1 T(1\varepsilon_2 T). \tag{43}$$

By using Bernoulli's method,

$$\frac{dT}{dt} \leq \varepsilon_1 \varepsilon_1 \varepsilon_2 T^2. \tag{44}$$

$$\frac{dT}{dt} - \varepsilon_1 \leq \varepsilon_1 \varepsilon_2 T^2. \tag{45}$$

Divide through by T^2

$$\frac{1}{T^2}\frac{dT}{dt} - \frac{1}{T^2}\varepsilon_1 \leq \varepsilon_1 \varepsilon_2. \tag{46}$$

Let, $S = T^{-1}$,

$$\frac{dS}{dt} = T^{-2}\frac{dT}{dt}. \tag{47}$$

Inputting this,

$$\frac{-dS}{dt} - \varepsilon_1 z \leq -\varepsilon_1 \varepsilon_2. \tag{48}$$

$$\frac{dZ}{dt} + \varepsilon_1 z \leq +\varepsilon_1 \varepsilon_2. \tag{49}$$

The equation is reduced to a first-order linear equation (ODEs), using an integrating factor.

$$I \cdot F = e^{\int \varepsilon_1 dt} = e^{\varepsilon_1 t}. \tag{50}$$

Multiply both side by $e^{\varepsilon_1 t}$.

$$e^{\varepsilon_1 t}\frac{dZ}{dt} + e^{\varepsilon_1 t}\varepsilon_1 z \leq e^{\varepsilon_1 t}\varepsilon_1 \varepsilon_2. \tag{51}$$

$$e^{\varepsilon_1 t}\frac{d(Se^{\varepsilon_1 t})}{dt} \leq e^{\varepsilon_1 t}\varepsilon_1 \varepsilon_2. \tag{52}$$

Integrating equation [25],

$$Se^{\varepsilon_1 t} \leq \varepsilon_1 \varepsilon_2 \int e^{\varepsilon_1 t} dt. \tag{53}$$

$$Se^{\varepsilon_1 t} \leq \varepsilon_2 e^{\varepsilon_1 t} + C. \tag{54}$$

$$S \leq \varepsilon_2 + Ce^{-\varepsilon_1 t}. \tag{55}$$

denotes that

$$S = \frac{1}{T}. \tag{56}$$

$$\frac{1}{T} \leq \varepsilon_2 + Ce^{-\varepsilon_1 t}. \tag{57}$$

$$T \leq \frac{1}{\varepsilon_2 + Ce^{-\varepsilon_1 t}}. \tag{58}$$

As $t \longrightarrow \infty$, we have that,

$$T(t) \leq \frac{1}{\varepsilon_2}. \tag{59}$$

Applying the integrating factor method [25] for first order ODE,

$$\frac{dI}{dt} \leq \sigma - \delta I. \tag{60}$$

The integrating factor is,

$$I \cdot F = e^{\int \delta t dt} = e^{\delta t}. \tag{61}$$

Multiply both sides by the integrating [26] factor,

$$e^{\delta t} \frac{dI}{dt} + e^{\delta t} \delta I \leq e^{\delta t} \sigma. \tag{62}$$

$$\frac{dIe^{\delta t}}{dt} \leq e^{\delta t} \sigma. \tag{63}$$

Integrating both sides give,

$$Ie^{\delta t} \leq \frac{e^{\delta t}}{\delta}\sigma + C. \tag{64}$$

Dividing both sides by, $e^{\delta t}$

$$I \leq \frac{\sigma}{\delta} + Ce^{-\delta t}. \tag{65}$$

The solutions are given by,

$$I(t) \leq \frac{\sigma}{\delta} + Ce^{-\delta t}. \tag{66}$$

As $t \longrightarrow \infty$. The solution is given by

$$I(t) \leq \frac{\sigma}{\delta}. \tag{67}$$

Then the solution $(M(t),\ T(t),\ I(t))$ is positive for all time t, [27]

$$\Omega = \Omega c := (M,\ T,\ I) \in R \tag{68}$$

$$M = \frac{1}{\beta 1} \tag{69}$$

$$T = \frac{1}{\alpha_2} \tag{70}$$

$$I = \frac{1}{\sigma}. \tag{71}$$

8. Equilibrium points

The steady state solutions is gotten when the left hand side of the equations is set to zero as follows.

1. $\frac{dN}{dt} = 0$

$$N(r\beta_1 N\gamma I\Gamma T) = 0. \tag{72}$$

2. $\frac{dT}{dt} = 0$

$$T(\alpha_1(1\alpha_2 T)+\beta_2 N\alpha_3 I)=0. \tag{73}$$

3. $\dfrac{dI}{dt}=0$

$$\sigma\delta I+\frac{\rho NI}{m+N}+\frac{\rho_1 TI}{m_1+T}+\mu NI+\mu_1 TI=0. \tag{74}$$

Solving for N, T, I we have the following based on either N or T are zero or both are zero.
1. When both N and T is zero then The third equation will reduce to

$$\sigma\delta I=0 \tag{75}$$

$$I=\frac{\sigma}{\delta}. \tag{76}$$

The equilibrium point is

$$E_o=(0,\,0,\,\frac{\sigma}{\delta}). \tag{77}$$

The resisting stage, ρ_2: This period comes into existence when abnormal cells begin to turn into tumor cells. The Equilibrium point at this stage is given as follows:

$$P_2=(0,\,T_1,\,I_1). \tag{78}$$

Where

$$T_1=\frac{1}{6\alpha_2}[6-\frac{\varepsilon_3+\sqrt{\varepsilon_3+4\varepsilon_4}+2\alpha_1\alpha_3\varepsilon_1}{\alpha_1\alpha_3\mu}+\frac{\alpha_3(-\alpha_1\varepsilon_1+\varepsilon_1)}{\mu+(\varepsilon_3+\sqrt{\varepsilon_3+4\varepsilon_4})}] \tag{79}$$

$$\varepsilon_1=2\mu_1+\alpha_2(\delta+m_1\mu_1-\rho_1)\geq 0 \tag{80}$$

$$\varepsilon_2=3\mu_1({}_1(1+m_1\alpha_2)(\alpha_2\delta+\mu_1)-\alpha_1\alpha_2\rho_1+\alpha_1\alpha_3\sigma)\geq 0 \tag{81}$$

$$\varepsilon_3=\alpha_1^2\alpha_3^2(\alpha_1\varepsilon_1\mu(\varepsilon_1\mu)+\alpha_2^2(2\delta_2^2+2(-m_1\mu_1+\rho_1)^2)-\delta(5m_1\mu_1+4\rho_1)$$

$$+9(\mu_1+\alpha_2(\delta+2m_1\mu_1+\rho_1)\alpha_1^2\alpha_2\alpha_3^4\mu_1\sigma\geq 0 \tag{82}$$

$$\varepsilon_4 = -\alpha_1^3\alpha_3^6(\alpha_1(\mu_1(\varepsilon_1\mu_1)+\alpha_2^2(\delta^2+(-m_1\mu_1+\rho_1)^2\delta_2(m_1\mu_1+2\rho_1)))-32\alpha_3\mu_1\sigma_2)^3 \geq 0. \tag{83}$$

8.1 *Remark*

From the positivity of solutions and the biological meaning of the equi- librium point ρ_2, we deduced that: The tumor cells compete for survival at the resisting stage by rapidly dividing and growing. Therefore,

$$1 \leq T_1 \leq \frac{1}{\alpha_2} \text{ where } 0 \geq \alpha_2 \geq 1. \tag{84}$$

There is a suppression to the activity of immune cells because of rapid growth and division of tumors cells . Therefore,

$$1_1 \leq \frac{\sigma}{\delta} \text{ where } 0\frac{\sigma}{\delta} = I(0) = 1.22 \tag{85}$$

9. Remark

According to the physiological meaning of cell cycle life, we can deduce that the rate of division normal cell to abnormal cells is very small compared with the rate of the natural divide of cell. Thus, $0 \leq \beta_1 \leq 0$.

10. Vascular adaptation

Secomb and Pries' series of articles, which simulate vessels in the rat mesentry, come to the following conclusion: $R(t) =$ radius at time t:

$$\begin{aligned} R(t+dt) &= R(t)+RdtS \\ &= M+Mes+C. \end{aligned} \tag{86}$$

$M =$ mechanical input (stress on the wall), $Me =$ metabolic requirements, $s =$ shrinkage.

11. Model development

Hybrid cellular automata: Separate cells functioning separately Development, decline, and adaptation. The fields that are continuous are $H+$, glucose, and oxygen. After each generation, find the steady-state metabolite fields traits depending on genes. Hyperplasticity: growth away from the wall of the basement. Glycolytic: improved glucose uptake and utilization. Acid-resistant: To inflict damage, lower the extracellular pH.

12. Reult and discussion

Tumor Growth Dynamics: By modeling the growth of cancer cells and their interactions with surrounding tissue, ODE models can replicate the evolution of a tumor over time. Predictions regarding the tumor's size, growth rate, and geographic dispersion could be among the outcomes.

Response to Treatment: Using ODE models, one can replicate the outcomes of several cancer therapies, including radiation, chemotherapy, and targeted therapy. Predictions of tumor regression, recurrence risk, and the emergence of treatment resistance are possible outcomes.

Tumor Heterogeneity: ODE models can take into account variations in the proliferative rates, migratory capacities, and therapeutic responsiveness of cancer cells. The findings could provide light on how treatment outcomes and the formation of drug-resistant subpopulations are impacted by tumor heterogeneity.

ODE models can include immune surveillance, tumor evasion strategies, and therapeutic therapies, among other interactions between cancer cells and the immune system. The findings might clarify how the immune system affects tumor growth and prognosis after treatment.

We presented a family of models (ODEs and DDEs) to characterize the dynamics of the tumor immune system in this work. With varying values of the parameters (the rate of tumor cells preceded by the effector cells) and (the maximum growth rate of the tumor cells population), the models' evolution and quality have been presented.

Despite the simplicity of the underlying models, they exhibit extremely rich dynamics and provide a clear representation of the phenomena of the actual interaction between immunotherapy and tumor growth. As a consequence of the glycolytic cycle, lactic acid lowers the extracellular pH, which damages healthy cells while simultaneously encouraging the growth of malignant cells.

Over the last few decades, research on cancer has incorporated an increasing amount of mathematical models. Here, we've shown how simple quantitative models can be developed, compared to experimental data, and used to illustrate complex biological processes and interactions.

The suggested course of treatment involved applying specific medications in a precisely scheduled order to achieve the intended result.

Determining this time-dependent method required an understanding of signaling pathway dynamics, or how protein concentrations vary over time. Without the need for time-consuming trials until a viable solution was found, the development of the ODE dynamical system model made it easier to investigate novel treatment approaches in simulation. So, the model made it possible to create intelligent experiments, whereas biological research that lacks mathematical models is forced to use guessing and intuition to direct the creation of new tests models have been included into cancer research. Here, we've demonstrated how basic quantitative models can be used to simulate intricate biological processes and interactions by demonstrating how they're produced and contrasted with experimental data. For the sake of simplicity, we have selected seminal publications as examples and have had to omit a substantial amount of great literature on mathematical modeling. Recent review articles and books that provide a more thorough summary of the state-of-the-art in cancer modeling are recommended reading for interested readers.

13. Cancer treatments

Cancer therapies (drugs, immunotherapy, or chemotherapy). The addition of a cancer treatment to the model will impact the prediction.

Chemotherapy: The delivery of chemotherapeutic medicines to target cancer cells can be simulated mathematically. The model can be enhanced by including parameters related to drug pharmacokinetics, dose scheduling, and drug resistance mechanisms, which can help maximize treatment plans and reduce adverse effects.

Targeted Therapy: Growth factor receptors and signaling pathways are two examples of molecular targets that can be precisely inhibited by targeted medicines. These effects can be modeled. These models aid in understanding resistance mechanisms, predicting the effectiveness of targeted medicines, and determining the best dose regimens.

Immunotherapy: ODE models of immunological responses to cancer can mimic the actions of immunotherapeutic treatments such as cancer vaccines, adoptive cell therapy, and immune checkpoint inhibitors. These models assist in clarifying the processes of immune cell activation, immune evasion by tumors.

Radiation therapy: Targeting and eliminating cancer cells while preserving healthy tissue can be replicated using mathematical models. The model incorporates parameters pertaining to tissue radiobiology, fractionation schedules, radiation dose, and treatment plan optimization in order to minimize radiation-induced toxicity.

Combination Therapies: Models can evaluate the effectiveness of multimodal treatment regimens, like chemotherapy with immunotherapy or targeted therapy. To optimize therapy outcomes, these models assist in determining the best drug combinations, synergistic effects, and treatment sequencing techniques.

14. The best model for cancer growth

The three models $\prime V = f(V)$ for V are the Gompertz, logistic, and Bertalanffy models at Table 1. Utilize the model equations. Remember that $K = a/b$ in the logistic model. Determine the optimal values for parameters a and b by minimizing the $NMSE$. Which model best captures the progression of cancer? Using Matlab, Excel's "Solver" feature, or any other program of your choice.

When the differential equations are numerically solved in Excel using Euler's approach, the model estimate can be evaluated: $V_i = V_{i1} + f(V_{i1})(t_i t_{i1})$, $i = 1, 2, ..., 45$.

15. Modeling cancer growth

With reference to the Excel "Solver" function, the following optimal parameter values at 1 and $NMSE$ are obtained:
Bertalanffy $a = 0.4340$, $b = 0.2158$, $NMSE = 0.0089$.
Logistic $a = 0.3389$, $b = 0.0489$, $NMSE = 0.0138$.
Gompertz $a = 0.2375$, $b = 0.1179$, $NMSE = 0.0049$.
The Gompertz model provides the most accurate fit to the data on tumor growth with an NMSE of 0.49.

Table 1. Modeling cancer growth

Value 1	Value 2	Value 3	Value 4	Value 5	Value 6	Value 7	Value 8	Value 9	Value 10
3.46	0.0158	12.39	0.4977	04977	3.2046	5.9668	48.29	3.2046	5.9668
4.58	0.0264	13.42	0.6033	0.6033	4.5241	6.6945	49.24	4.5241	6.6945
5.67	0.0326	15.19	0.8441	0.8441	4.3459	6.6395	50.19	4.3459	6.6395
6.64	0.0445	16.24	1.2163	1.2163	5.1374	6.8971	51.14	5.1374	6.8971
7.63	0.0646	17.23	1.4470	1.4470	5.5376	7.2966	52.10	5.5376	7.2966
8.41	0.0933	18.18	2.3298	2.3298	4.8946	7.2268	54.00	4.8946	7.2268
9.32	0.1454	19.29	2.5342	2.5342	5.0660	6.8815	56.33	5.0660	6.8815
10.27	0.2183	21.23	3.0064	3.0064	6.1494	8.0993	57.33	6.1494	8.0993
11.19	0.2842	21.99	3.4044	3.4044	6.8548	7.2112	59.38	6.8548	7.2112

16. Conclusions

Early detection of the cancer allows for the application of chemo or immunotherapy when necessary, preventing the disease from spreading further.

In cancer biology, ordinary differential equation-based models are helpful for examining the evolution of biological systems. It is necessary to gather data at many intervals in order to estimate models with adequate accuracy. A more

thorough grasp of the underlying dynamics can be attained by qualitatively analyzing models. Here, we demonstrated a number of uses of ODEs in cancer biology. ODEs and mathematical models in general have the ability to validate experimental results and open up new directions for scientific research.

Since employing ODEs at Table 2 to simulate a biological mathematical model necessitates that there be only one independent variable (such as time) and that all others (such as space) can be omitted, the mathematical modeling in the cancer study uses ODEs.

We stress that, in general, mathematical models and ODEs [28] can validate experimental results and open up new lines of inquiry for science [29].

We did not evaluate the models, but we did remember that in order to have a greater knowledge of the underlying dynamic system, all mathematical models should be analyzed from a qualitative point of view.

Table 2. Parmeters description mathematical model of cancer with ordinary differential equaitions

Parmeter	Description
$M(t)$	continuous function on time t
$Y(t)$	positive and increasing to different biological factors
ODEs	Ordinary Differential Equaitions
$V(T)$	the volume for dividing cells at time
p	the population of cancer cells
k	host carrying capacity
$M(t)$	normal cells at the time
$T(t)$	tumor cells at the time
$I(t)$	immune system at the time
r	indicative the populations for the normal cells growth rate
β_1	the cell saturations
η	indicative the rate of interaction between the normal cells and the immune cells
γ	indicative the rate of interaction between the normal cells and tumor cells
ε_1	indicative the growth rate of the tumor cells
β_2	indicative of the interaction between normal cells and tumor cells
ε_2	the tumor cell saturation
ε_3	indicative of the interactions between the tumor and immune cells
R	positive invariant
ρ	indicative of this response rate
m	indicates the immune system rate
μ	indicates the reduction of the immune cells to the interaction with the normal cells
μ_1	indicates the reduction of the immune cells to their interactions with the tumor
M	mechanical input (stress on the wall)
Me	metabolic requirements
s	shrinkage
$a,\ b$	minimizing the NMSE

17. Future studies

Computational science and mathematics have collaborated to improve our understanding of biological systems. The immune systems was developed in this work to provide a dynamical, analytical, and numerical analysis of the effect of a compromised immune system on the development of cancer. There was no doubt that the biological and mathematical mechanisms were related. We can infer from the analytical results that the development of abnormal cells in the tissue was made possible by the instability of the reaction to the abnormal cells. Because there was an instance of semi-stability

in the coexistence stage, which meant that tumor cells could arise at any time, this led to malignancies. This suggests that the cells are unable to coexist.

In conclusion, the immune system's dynamic response when aberrant cells were able to settle in the tissue and started to develop into tumor cells. It was clear that there was a connection between the biological mechanisms underlying the various stages of cancer progression and the mathematical principles of the immune systems. To validate the outcomes of our mathematical model and provide more accurate results, we suggest carrying out additional experimental research to clinically examine the findings of this study and add to the analysis of actual instances. In subsequent research, we will refine this model by examining the impacts of variables that are associated with an elevated risk of cancer, like dietary factors.

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Conflict of interest

The authors declare no competing financial interest.

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