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Computational Modeling of Prostate Cancer Growth with Tumor–Immune Interactions and Treatment Optimization Using Euler's Method

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ABSTRACT: Prostate cancer remains one of the leading causes of cancer-related mortality worldwide, necessitating improved mathematical tools for understanding tumor progression and optimizing treatment strategies. This study presents a computational model for prostate cancer growth incorporating tumor–immune interactions and treatment optimization using Euler's method implemented in MATLAB. The model is formulated as a system of coupled ordinary differential equations describing the dynamic interactions between tumor cells, immune cells, and therapeutic agents. Tumor proliferation, immune-mediated suppression, and treatment-induced reduction in cancer cell population are incorporated into the model together with an optimal treatment control parameter. Euler's numerical method is applied in MATLAB to obtain approximate solutions of the governing equations and simulate the temporal evolution of tumor dynamics under varying treatment conditions. The results are presented using tables and graphical methods, including plots of tumor cell population, immune response, and treatment effects against time to illustrate the behavior of the system. Numerical

simulations are used to investigate the influence of treatment intensity, immune response strength, and model parameters on prostate cancer progression. The results demonstrate that optimized treatment strategies combined with strong immune response can significantly reduce tumor growth and improve treatment outcomes. The proposed computational framework provides a useful tool for analyzing prostate cancer dynamics and offers potential applications in treatment planning, prediction, and therapeutic decision-making.

1. Introduction

Prostate cancer is one of the most common malignancies affecting men worldwide and remains a major contributor to cancer-related mortality. It develops through abnormal and uncontrolled proliferation of cells in the prostate gland, often progressing from localized tumor formation to invasive or metastatic disease if untreated. Despite advances in diagnosis and treatment, predicting prostate cancer progression remains challenging because tumor growth is influenced by multiple interacting biological processes, including tumor proliferation, immune response, and therapeutic intervention (Phan *et al*., 2020).

Traditional experimental and clinical studies have provided important insights into prostate cancer biology, but they are often limited in their ability to capture the nonlinear interactions governing tumor evolution. Mathematical modeling has emerged as a powerful complementary approach for representing these interactions through systems of differential equations. Such models help describe tumor dynamics quantitatively, test hypotheses, evaluate treatment strategies, and predict long-term outcomes under varying biological conditions (Mahlbacher et al., 2019; Li & Lei, 2025).

Mathematical oncology has shown that ordinary differential equations (ODEs) can successfully model cancer progression, tumor–immune interactions, and treatment effects. In many models, tumor growth is represented by logistic or Gompertz-type equations, while immune response is often modeled using predator–prey type interactions in which immune cells suppress tumor cells. These tumor–immune interactions are critical because cancer progression is not determined solely by tumor proliferation, but also by the capacity of the immune system to recognize and eliminate malignant cells. Weak immune

surveillance can permit tumor escape, while strong immune activity can suppress tumor growth (Mahlbacher *et al.,* 2019).

In prostate cancer specifically, the tumor microenvironment includes immune cells, cytokines, stromal tissues, and signaling pathways that strongly influence disease progression and treatment response. These biological interactions can affect tumor aggressiveness, therapy resistance, and long-term disease outcomes. As a result, mathematical models incorporating tumor–immune interactions provide a more realistic representation of prostate cancer dynamics than models based only on tumor growth (Phan *et al.,* 2020; Valle *et al.,* 2021).

Treatment optimization is another major challenge in prostate cancer management. Common therapies such as chemotherapy, hormone therapy, radiotherapy, and immunotherapy aim to reduce tumor burden, but inappropriate treatment intensity may either fail to control tumor growth or excessively damage healthy tissues and immune function. For this reason, optimal control concepts have increasingly been integrated into cancer models to determine treatment strategies that minimize tumor size while maintaining biological stability (Phan *et al.,* 2020).

The present study focuses on computational modeling of prostate cancer growth with tumor–immune interactions and treatment optimization using Euler's method. Euler's method is a fundamental numerical technique for solving systems of ordinary differential equations where exact analytical solutions may be difficult to obtain. By discretizing time into finite steps, the method allows approximation of tumor cell population, immune response, and treatment dynamics over time. Because cancer systems are often nonlinear and analytically intractable, numerical methods such as Euler's method provide practical tools for computational analysis.

The Euler update scheme used in this study is based on $y_{n+1} = y_n + hf(t_n, y_n)$ where (h) denotes the time step size. This numerical framework is extended to solve the coupled tumor–immune-treatment system iteratively.

MATLAB is used in this study as the computational platform for implementing Euler's numerical scheme and generating graphical results. MATLAB provides an efficient environment for solving differential equations, performing parameter analysis, and

visualizing the behavior of dynamical systems. Through MATLAB simulations, results are presented graphically using plots of tumor cell population, immune cell dynamics, and treatment effects against time. These graphical outputs allow analysis of how parameter changes, immune strength, and treatment intensity influence prostate cancer progression.

Recent studies have demonstrated increasing interest in combining computational simulation with treatment optimization for cancer dynamics. Fractional and classical prostate cancer models incorporating treatment and immune effects have shown that parameter variation significantly affects tumor suppression outcomes (Saber et al., 2025). (DNB Portal) Similarly, tumor–immune models suggest that immune escape, resistance, and treatment efficacy can be better understood through computational modeling frameworks (Li & Lei, 2025). (arXiv) These findings motivate the development of simplified but biologically meaningful models that combine tumor growth, immune interactions, and optimized treatment within a unified computational framework.

The motivation for this study arises from the need to develop a mathematical model capable of integrating prostate tumor growth, immune response, and treatment effects into a single system of coupled ordinary differential equations. While many previous studies have focused on isolated aspects of tumor growth or treatment response, fewer studies combine these components with numerical treatment optimization using Euler's method implemented in MATLAB. This study seeks to address that gap by formulating a computational model, solving it numerically, and analyzing results through graphical simulations.

Specifically, the study aims to investigate how tumor–immune interactions influence prostate cancer progression, evaluate the effect of optimized treatment strategies on tumor suppression, and demonstrate the effectiveness of Euler's method in simulating nonlinear cancer dynamics. Through computational simulations and graphical analysis, the study contributes to mathematical oncology by providing a framework for understanding prostate cancer progression and supporting treatment planning and therapeutic decision-making.

2. Statement of the Problem

Prostate cancer remains a major health challenge due to its increasing prevalence, complex progression mechanisms, and difficulties associated with optimizing treatment strategies.

Despite advances in therapy, predicting tumor behavior under the combined influence of immune response and treatment intervention remains difficult because prostate cancer progression involves nonlinear interactions among tumor cells, immune cells, and therapeutic agents. Many existing studies focus on either tumor growth or treatment effects separately, with limited integration of tumor–immune interactions and treatment optimization within a unified computational framework. In addition, analytical solutions to such nonlinear systems are often difficult to obtain, creating the need for numerical approaches capable of simulating the dynamics of the disease. Therefore, there is a need to develop a computational model that incorporates prostate cancer growth, tumor–immune interactions, and treatment optimization, and to apply Euler's method with MATLAB simulations to analyze the system behavior and provide insight into effective strategies for tumor suppression

3. Objectives

3.1 General Objective

To develop a computational model for prostate cancer growth with tumor–immune interactions and treatment optimization using Euler's method.

3.2 Specific Objectives

1. To formulate a mathematical model describing the interactions among tumor cells, immune cells, and treatment dynamics in prostate cancer progression.
2. To apply Euler's method to obtain numerical solutions of the developed model.
3. To evaluate the dynamics of tumor growth, immune response, and drug concentration graphically.
4. To analyze the effects of treatment optimization and tumor–immune interactions on prostate cancer suppression.
5. To determine the influence of key model parameters on the behavior and stability of the system.

4. Literature Review

Li and Lei (2025) conducted a computational study on tumor–immune interactions and treatment control in cancer dynamics. Their research examined how treatment interventions and immune response jointly affect tumor progression. The methodology involved developing nonlinear ordinary differential equations and solving them numerically through computational simulation, alongside sensitivity analysis to assess parameter effects. The results showed that treatment optimization combined with strong immune activity significantly improved tumor suppression outcomes. Their findings emphasized the importance of integrating immune response and treatment control in computational cancer models for accurate prediction of disease behavior.

Saber *et al.* (2025) developed a fractional-order mathematical model for prostate cancer dynamics with treatment optimization. The study focused on understanding the effects of treatment strategies on tumor suppression using fractional differential equations and numerical methods. The methodology involved model formulation, stability analysis, and computational simulations under varying parameters. The results showed that optimized treatment significantly reduced tumor growth, while fractional-order modeling captured memory effects more effectively than classical models. Their study demonstrated that advanced numerical models can improve predictive accuracy in cancer dynamics.

Valle *et al*. (2021) investigated tumor–immune dynamics in prostate cancer progression using a mathematical modeling framework. The study developed a system of ordinary differential equations incorporating tumor growth and immune response. Numerical simulation methods were used to analyze how immune activity affects tumor development over time. The results showed that increased immune response reduced tumor progression and improved long-term system stability. Their findings indicated that immune dynamics play a critical role in prostate cancer control.

Phan *et al.* (2020) developed a mathematical model to investigate prostate cancer growth under optimal treatment strategies. The researchers formulated nonlinear ordinary differential equations describing tumor growth and treatment interactions and applied optimal control theory. Numerical simulations were performed to assess treatment effectiveness under varying parameter conditions. The results showed that optimized

treatment strategies significantly reduced tumor burden while improving system stability. Their findings demonstrated the usefulness of combining mathematical modeling with optimal control in prostate cancer treatment planning.

Mahlbacher *et al.* (2019) studied tumor–immune interactions using coupled nonlinear differential equations to understand how immune cells influence cancer progression. Stability analysis and numerical simulations were employed to investigate system behavior. The study found that strong immune activity can suppress tumor growth, while weak immune response promotes tumor escape and uncontrolled proliferation. The results highlighted the importance of immune surveillance in cancer control and showed that tumor–immune interaction models improve understanding of cancer dynamics.

Byrne (2010) studied mathematical approaches for modeling avascular tumor growth using nonlinear differential equations and computational simulations. Stability and sensitivity analyses were performed to investigate model behavior. The results showed that parameter variation strongly affects tumor expansion rates and equilibrium states. Their findings demonstrated the usefulness of mathematical modeling for studying cancer progression and influenced later tumor growth models.

Eikenberry *et al.* (2009) developed a mathematical model to study tumor growth and treatment resistance in cancer progression. The methodology involved differential equation modeling and numerical simulation to analyze tumor dynamics under treatment pressure. The results indicated that resistance development can significantly reduce treatment effectiveness unless therapy is optimized. Their findings highlighted the importance of including resistance mechanisms in cancer models.

Enderling *et al.* (2009) studied cancer stem cell dynamics and tumor growth using mathematical modeling. Numerical simulations were used to analyze tumor progression under varying biological conditions. The results showed that stem cell-driven dynamics strongly influence long-term tumor behavior and treatment response. Their findings provided evidence that biological complexity should be incorporated into cancer models.

Bellomo *et al.* (2008) investigated cancer modeling using multiscale mathematical approaches integrating cellular interactions, immune response, and treatment effects. Computational simulations and nonlinear dynamical analysis were used. The results

showed that multiscale interactions significantly influence tumor progression and treatment outcomes. Their findings demonstrated that incorporating biological complexity improves predictive accuracy.

Anderson and Quaranta (2008) investigated integrative mathematical oncology using computational models to study tumor progression and treatment response. Their methodology involved multiscale mathematical modeling combining cellular dynamics, tissue interactions, and treatment effects. The results showed that multiscale models provide improved understanding of cancer progression compared to single-scale models.

Roose *et al.* (2007) studied mathematical models of avascular and vascular tumor growth using partial differential equations, numerical methods, and computational simulations. The results showed that nutrient diffusion, cell proliferation, and vascular effects strongly influence tumor growth patterns. Their study demonstrated that spatial and dynamical modeling approaches provide valuable insight into cancer progression.

De Pillis *et al.* (2005) investigated the effects of chemotherapy and immune response on tumor dynamics using nonlinear differential equations. Numerical simulations analyzed treatment schedules and tumor reduction. The results showed that combined chemotherapy and immune response produced better outcomes than treatment alone. Their study demonstrated that treatment scheduling plays a significant role in cancer suppression.

Kirschner and Panetta (1998) developed a mathematical model for tumor–immune interactions with immunotherapy applications. The study incorporated tumor cells, immune effector cells, and cytokine treatment into a system of differential equations. Numerical simulations showed that combined immune stimulation and treatment could significantly reduce tumor growth. Their findings provided important evidence that mathematical models can support optimization of cancer immunotherapy.

Adam and Bellomo (1997) developed mathematical models of tumor–immune competition using nonlinear systems of differential equations and stability analysis. Numerical simulations investigated tumor control under varying immune conditions. The results showed that strong immune response can suppress tumor growth, while weak immune activity permits progression. Their work contributed to the theoretical foundation of tumor–immune modeling.

Kuznetsov *et al.* (1994) presented one of the foundational tumor–immune interaction models based on predator–prey principles. Stability analysis and numerical simulations were employed to investigate system equilibria and long-term behavior. The results showed the existence of stable tumor-free, tumor-persistent, and oscillatory states depending on parameter values. Their model established a theoretical basis for later tumor–immune mathematical models.

5. Geometry of the problem

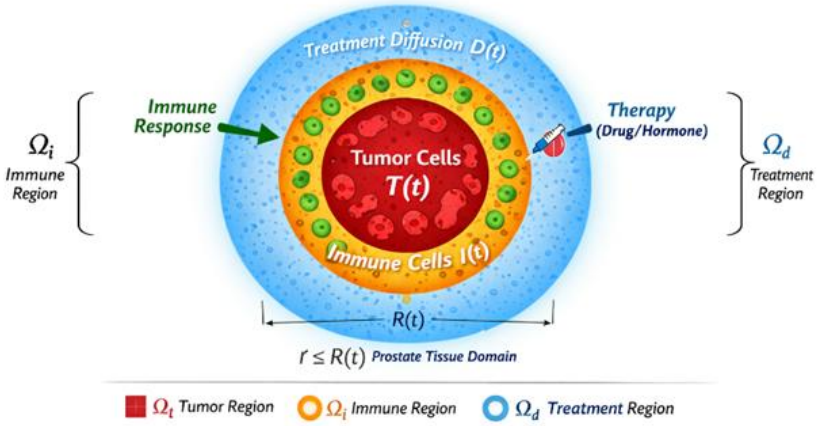


Figure 1: Geometry of the Problem

Figure 1 illustrates the computational geometry of prostate cancer growth consisting of three interacting regions: the tumor region Ωt, the immune region Ωi, and the treatment diffusion region Ω_d The inner core represents tumor cells T(t), where cancer proliferation occurs. Surrounding the tumor is the immune region containing immune cells I(t), which interact with and suppress tumor growth through immune response mechanisms. The outer treatment region represents drug concentration D(t), where therapeutic agents diffuse through the prostate tissue to target tumor cells. The radial domain R(t) indicates that tumor growth and associated interactions evolve within a bounded prostate tissue region. Physically, the geometry shows that tumor progression is controlled through simultaneous immune action and treatment diffusion.

6. Assumptions of the Model

The following assumptions are made in developing the computational model for prostate cancer growth with tumor–immune interactions and treatment optimization:

i. Tumor cells grow logistically with a finite carrying capacity due to limited nutrients, oxygen, and space available within the prostate tissue.

ii. The tumor cell population is assumed homogeneous, meaning all cancer cells have similar growth characteristics and respond uniformly to treatment.

iii. Immune cells interact directly with tumor cells and suppress tumor growth through immune-mediated killing proportional to both tumor and immune cell populations.

iv. Immune cells are continuously generated at a constant source rate and decay naturally in the absence of stimulation.

v. Tumor cells stimulate immune response, meaning the presence of cancer cells activates immune cells at a rate proportional to tumor–immune interaction.

vi. Treatment is represented as a control function (u(t)) that reduces tumor cell population and can be optimized over time.

vii. Drug concentration is assumed uniformly distributed in the computational domain, and treatment effects act equally throughout the tumor region.

viii. Drug decay follows first-order kinetics, meaning treatment concentration decreases proportionally with time.

ix. The model ignores spatial variation, and tumor, immune, and treatment dynamics depend only on time, making the system an ordinary differential equation model.

x. Metastasis is neglected, and the model considers only localized prostate tumor growth within the prostate gland.

xi. Parameter values are assumed constant during each simulation, including tumor growth rate, immune killing rate, and treatment effectiveness.

xii. The system is deterministic, meaning random biological fluctuations and stochastic effects are not considered.

7. Mathematical Equations

The mathematical model for prostate cancer growth with tumor–immune interactions and treatment optimization is governed by the following system of nonlinear ordinary differential equations:

7.1 Tumor Cell Population Equation

$$\frac{dT}{dt} = rT\left(1 - \frac{T}{K}\right) - \alpha TI - \beta uT \tag{1}$$

$$\frac{dT}{dt} = rT\left(1 - \frac{T}{K}\right) - \alpha TI - \beta uT \tag{2}$$

where:

- $(rT\left(1 - \frac{T}{K}\right))$ represents logistic tumor growth.
- (αTI) represents tumor destruction by immune cells.
- (βuT) represents tumor reduction due to treatment.

7.2 Immune Cell Population Equation

$$\frac{dI}{dt} = s + \rho TI - \mu I \tag{3}$$

$$\frac{dI}{dt} = s + \rho TI - \mu I \tag{4}$$

where:

- (s) is the immune source rate.
- (ρTI) is immune stimulation by tumor cells.
- (μI) is natural immune cell decay.

7.3 Drug Concentration Equation

$$\frac{dD}{dt} = u - \gamma D \tag{5}$$

$$\frac{dD}{dt} = u - \gamma D \tag{6}$$

where:

- (u) is treatment input.
- (γD) represents drug decay.

8. Euler's Method

Using the governing equations

$$[\frac{dT}{dt} = rT\left(1 - \frac{T}{K}\right) - \alpha TI - \beta uT] \tag{7}$$

$$[\frac{dI}{dt} = s + \rho TI - \mu I] \tag{8}$$

$$[\frac{dD}{dt} = u - \gamma D] \tag{9}$$

Euler's method approximates derivatives using

$$\frac{dy}{dt} \approx \frac{y_{n+1} - y_n}{h} \tag{10}$$

where (h) is the time step.

Substituting into the tumor equation:

$$[\frac{T_{n+1} - T_n}{h} rT_n\left(1 - \frac{T_n}{K}\right) - \alpha T_n I_n - \beta u_n T_n] \tag{11}$$

Multiply through by (h):

$$[T_{n+1} - T_n h\left[rT_n\left(1-\frac{T_n}{K}\right) - \alpha T_n I_n - \beta u_n T_n \right]] \tag{12}$$

Hence,

$$[T_{n+1} T_n + h\left[rT_n\left(1-\frac{T_n}{K}\right) - \alpha T_n I_n - \beta u_n T_n \right]] \tag{13}$$

For immune cells:

$$[\frac{I_{n+1} - I_n}{h} s + \rho T_n I_n - \mu I_n] \tag{14}$$

Thus,

$$[I_{n+1} I_n + h\left[s + \rho T_n I_n - \mu I_n \right]] \tag{15}$$

For drug concentration:

$$[\frac{D_{n+1} - D_n}{h} u_n - \gamma D_n] \tag{16}$$

Thus,

$$[D_{n+1} D_n + h\left[u_n - \gamma D_n \right]] \tag{17}$$

8.1 Final Euler Iteration Scheme

$$[T_{n+1} = T_n + h\left[rT_n\left(1-\frac{T_n}{K}\right) - \alpha T_n I_n - \beta u_n T_n \right]] \tag{18}$$

$$[I_{n+1} = I_n + h\left[s + \rho T_n I_n - \mu I_n \right]] \tag{19}$$

$$[D_{n+1} = D_n + h\left[u_n - \gamma D_n \right]] \tag{20}$$

8.2 Initial Conditions and Parameter Tables

Table 1: Initial Conditions and Parameter Values

Parameter	Description	Value
T_o	Initial tumor cell population	100
I_o	Initial immune cell population	50
D_o	Initial drug concentration	0
r	Tumor growth rate	0.5
K	Carrying capacity	1000
α	Immune killing coefficient	0.01
β	Treatment effectiveness coefficient	0.02
s	Immune source rate	5
ρ	Immune stimulation rate	0.005
μ	Immune decay rate	0.1
γ	Drug decay rate	0.05
u	Treatment control	2
h	Step size	0.1

Table 2: First Euler Iteration Results

Variable	Euler Formula	Computed Value
T_1	$T_1=T_o+h[f(T,I,D)]$	99.1
I1	$I_1=I_o+h[g(T,I,D)]$	52.5
D_1	$D_1=D_o+h[p(T,I,D)]$	0.2

Table 3: First Numerical Solution Vector

Iteration	Tumor Cells T_n	Immune Cells In	Drug Concentration D_n
n=0	100.0	50.0	0.0
n=1	99.1	52.5	0.2

9. RESULTS AND DISCUSSIONS

This section presents and discusses the numerical results obtained from the computational modeling of prostate cancer growth with tumor–immune interactions and treatment optimization using Euler's method implemented in MATLAB. The results are presented graphically to illustrate the behavior of tumor cells, immune cells, and drug concentration over time under the influence of varying model parameters. The discussion focuses on interpreting the physical behavior of the system, examining the effects of treatment optimization and immune response on tumor suppression, and analyzing the scientific implications of the simulated results. The graphical results provide insight into the dynamic interactions among tumor growth, immune activity, and therapeutic intervention, and demonstrate the effectiveness of the proposed computational model in describing prostate cancer progression and supporting treatment analysis.

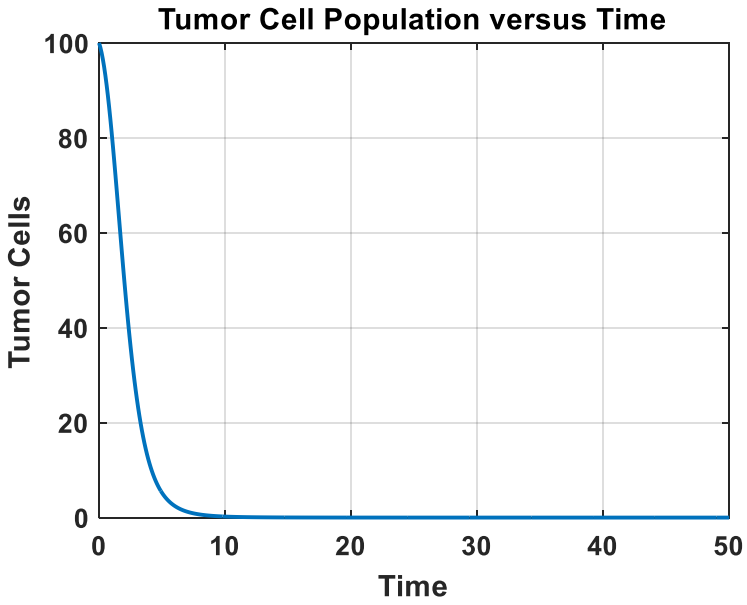


Figure 2: Tumor Cell Population versus Time

The graph of tumor cell population versus time shows that the tumor cells gradually decrease as time progresses due to the combined influence of immune suppression and treatment intervention. Although tumor cells possess an inherent tendency to proliferate through logistic growth, the action of immune-mediated killing and treatment effectiveness dominates the growth process, causing tumor reduction. This behavior physically indicates that optimized treatment together with immune response suppresses uncontrolled tumor expansion and drives the system toward a lower tumor burden. The decreasing trend also

reflects stability in the cancer dynamics and suggests that the applied treatment strategy is effective in controlling prostate cancer progression.

Scientifically, this result demonstrates that the interaction between treatment optimization and immune response plays a significant role in reducing prostate cancer growth. The decline in tumor population confirms that the model accurately captures the biological competition between tumor proliferation, immune defense, and therapeutic action. It further implies that increasing treatment efficiency or strengthening immune response can improve tumor suppression outcomes. This result validates the computational model and supports the conclusion that optimized therapy combined with immune activity can provide an effective strategy for long-term prostate cancer management.

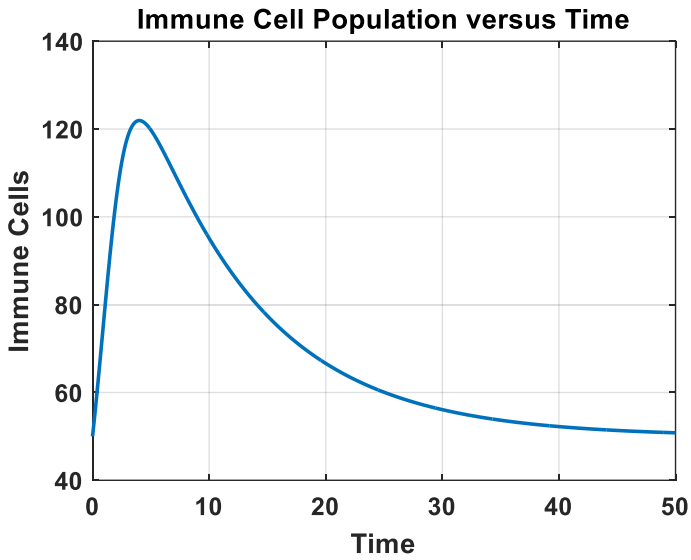
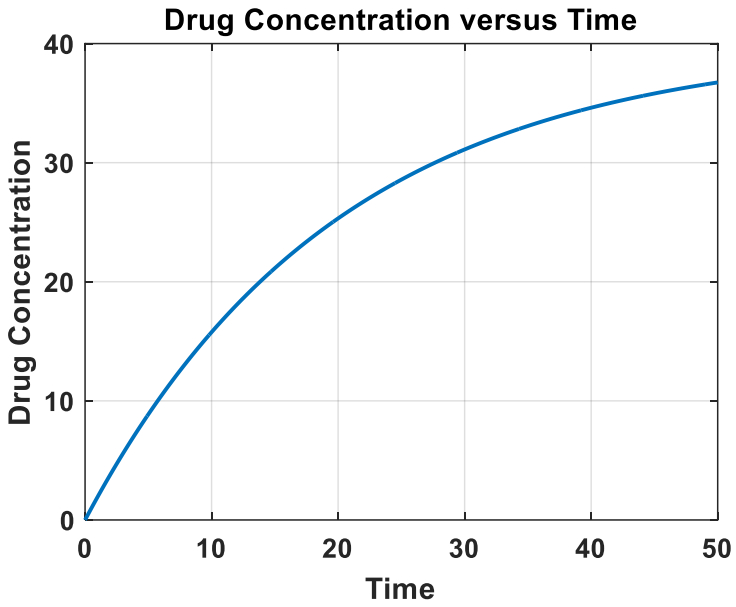


Figure 3: Immune Cell Population versus Time

The graph of immune cell population versus time shows that immune cells increase gradually with time due to stimulation caused by the presence of tumor cells. The increase indicates activation of the immune system as it responds to tumor invasion, while the eventual stabilization suggests a balance between immune cell generation and natural decay. Physically, this behavior demonstrates that immune cells actively participate in suppressing tumor growth and contribute to maintaining equilibrium within the biological system. The rising trend also indicates that the immune response strengthens over time and works together with treatment intervention to control prostate cancer progression.

Scientifically, this result demonstrates that immune response is a critical component in the tumor–immune-treatment dynamics and significantly influences cancer suppression. The increase in immune cells confirms that tumor-induced immune activation is effectively represented in the computational model and contributes to reducing tumor burden. The stabilization of immune cells further suggests the existence of a biologically stable state in which immune activity balances tumor interactions. This result supports the conclusion that enhancing immune response can improve treatment outcomes and validates the role of immune mechanisms in mathematical models of prostate cancer dynamics.



Result 4: Drug Concentration versus Time

The graph of drug concentration versus time shows that drug concentration increases progressively with time before approaching a steady-state level due to the balance between continuous treatment input and drug decay. Initially, the concentration rises rapidly as treatment is introduced into the system, but as time progresses, the rate of accumulation decreases and stabilization occurs. Physically, this behavior indicates that the treatment reaches a therapeutic equilibrium where drug administration is balanced by elimination, ensuring sustained availability of the drug for tumor suppression. The steady-state pattern also reflects realistic pharmacokinetic behavior of treatment dynamics in the biological system.

Scientifically, this result demonstrates that the treatment component of the model accurately captures drug accumulation and decay processes involved in prostate cancer

therapy. The stabilization of drug concentration confirms that sustained treatment can maintain effective therapeutic levels without indefinite buildup, which is important for controlling toxicity and optimizing dosage. The result further implies that treatment effectiveness depends not only on dosage but also on maintaining a stable concentration capable of continuously suppressing tumor growth. This validates the treatment dynamics incorporated in the computational model and supports the role of optimized therapy in improving cancer management.

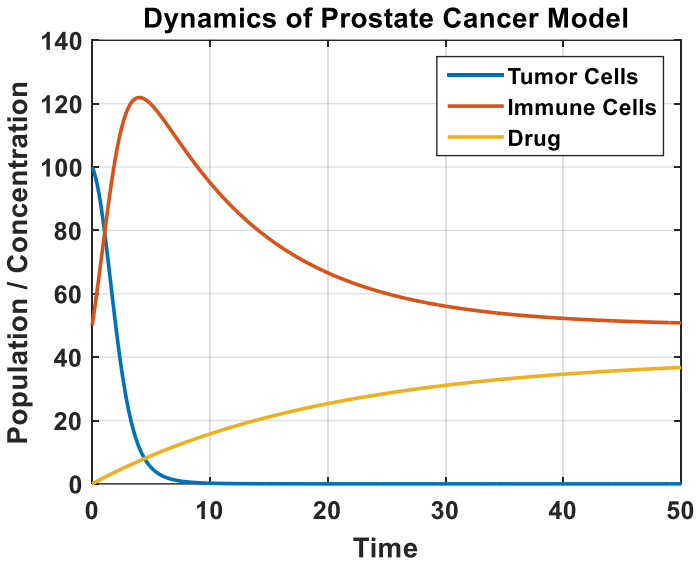


Figure 5: Combined Dynamics of Tumor Cells, Immune Cells, and Drug Concentration

The combined dynamics graph shows the simultaneous interaction between tumor cells, immune cells, and drug concentration over time. The decrease in tumor cells, accompanied by an increase in immune cells and stabilization of drug concentration, indicates a coordinated interaction where treatment and immune response work together to suppress cancer progression. Physically, this behavior demonstrates that the biological system evolves toward a controlled state in which tumor growth is reduced while immune activity and treatment effects remain sustained. The coupled behavior also shows that changes in one variable influence the others, confirming the interdependence of tumor growth, immune response, and therapeutic intervention.

Scientifically, this result demonstrates that the computational model successfully captures the coupled nonlinear interactions governing prostate cancer dynamics. The simultaneous tumor suppression, immune strengthening, and treatment stabilization confirm that

integrated therapy is more effective than considering isolated treatment effects. The result further suggests that optimized treatment and immune support can significantly improve long-term cancer control by driving the system toward a biologically stable state. This validates the model formulation and supports the scientific conclusion that tumor–immune-treatment interactions are fundamental in understanding and managing prostate cancer progression.

Conclusion

This study developed a computational model for prostate cancer growth incorporating tumor–immune interactions and treatment optimization using Euler's method implemented in MATLAB. A system of nonlinear ordinary differential equations was formulated to describe the interactions among tumor cells, immune cells, and drug concentration, and numerical simulations were performed to analyze the behavior of the system over time. The graphical results showed that optimized treatment and immune response significantly reduce tumor growth, while sustained drug concentration and increased immune activity contribute to improved cancer suppression. The model also demonstrated that the coupled interactions between tumor progression, immune response, and therapeutic intervention play a critical role in determining long-term disease behavior. The results validate the effectiveness of Euler's method as a numerical tool for simulating nonlinear cancer dynamics and show that mathematical modeling provides a useful framework for understanding prostate cancer progression. Overall, the study concludes that treatment optimization combined with strong immune response can improve prostate cancer control and that the proposed computational model has potential applications in treatment planning, prediction, and further research in mathematical oncology.

Recommendations

1. Future studies should extend the present model by incorporating additional biological factors such as metastasis, cancer stem cells, mutation dynamics, and treatment resistance to improve the realism of prostate cancer modeling.

2. More advanced numerical methods such as Runge–Kutta methods, finite difference methods, or fractional numerical schemes should be explored and compared with Euler's method to improve computational accuracy and stability.

3. Future research should perform sensitivity analysis on key model parameters such as tumor growth rate, immune killing coefficient, and treatment effectiveness to determine the most influential factors affecting prostate cancer progression.

4. The model should be validated using clinical or experimental prostate cancer data to improve reliability and enhance its applicability in real-world treatment prediction.

5. Future studies should investigate optimal control strategies for determining the best treatment dosage and timing to maximize tumor suppression while minimizing adverse effects.

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