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Original Article

Inflammation-Driven Cancer Growth: A Mathematical Model

Palchhin Yadav[1], Paras Uchat[2]

[1,2]*Department of Mathematics, Indian Institute of Teacher Education, Gandhinagar, Gujarat, India.*

[2]*Corresponding Author : parasu@iite.ac.in*

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Abstract - *The objective of this study is to develop a mathematical model to understand cancer progression driven by chronic inflammation. The model is formulated using a system of nonlinear ordinary differential equations incorporating tumor growth, inflammation, immune suppression, and vascular support. Analytical techniques, including equilibrium analysis and Jacobian-based stability analysis, are used to examine system behavior. The results indicate that chronic inflammation increases tumor growth by enhancing the effective growth rate and promoting immune suppression, which reduces immune response effectiveness. Stability analysis shows that the system attains a locally asymptotically stable equilibrium, indicating persistent cancer progression. The study concludes that cancer progression is sustained by feedback interactions between tumor growth, inflammation, and immune suppression. These findings highlight the importance of targeting both inflammation and immune mechanisms in cancer treatment strategies.*

Keywords - *Chronic Inflammation, Immune suppression, Mathematical modeling, Stability analysis, Tumor dynamics.*

1. Introduction

Cancer progression is a complex and multiscale process involving uncontrolled cell growth, invasion of surrounding tissues, and eventual spread to distant organs. Although significant progress has been made in understanding cancer biology, the mechanisms that drive its development and progression are still not fully understood. Traditionally, cancer has been linked to genetic mutations, environmental exposures, and lifestyle factors. However, increasing attention has been given to the role of the tumor microenvironment, which actively influences tumor behaviour rather than acting as a passive background [4]. Among the components of the tumor microenvironment, chronic inflammation has emerged as an important factor in cancer development. While inflammation normally serves as a protective response to injury or infection, its prolonged presence can disrupt normal cellular regulation. Persistent inflammation can lead to DNA damage, promote abnormal cell proliferation, and alter immune responses. It also contributes to the formation of new blood vessels, which support tumor growth. Reports such as those from the National Cancer Institute (2015) indicate that long-term inflammation significantly increases the risk of cancer, highlighting its role not only in tumor progression but also in its initiation [3,6-8].

Mathematical modelling provides a useful framework for understanding such complex biological interactions. Classical models, including exponential, logistic, and Gompertz growth models, have been widely used to describe tumor growth under limited resources [5,9]. These models capture general growth behavior effectively but often consider tumor dynamics in isolation, without explicitly incorporating the influence of inflammation and immune-related processes. More recent studies have explored the role of inflammation, immune suppression, and vascular development in cancer progression. These studies show that inflammation can enhance tumor growth, suppress immune responses, and modify the tumor environment in ways that Favor disease progression [2,6,7]. Multiscale modelling approaches have further demonstrated that inflammation can exhibit both protective and tumor-promoting roles depending on its intensity and duration [1]. However, most existing approaches examine these processes separately or only partially combine them. As a result, the combined and interacting effects of inflammation-driven mechanisms are not fully understood within a single modelling framework. To address this limitation, the present study develops a mathematical model that integrates tumor growth, chronic inflammation, immune suppression and vascular support within a unified system. The model is formulated using nonlinear ordinary differential equations to represent the interactions among these factors over time. By considering these processes together, the study provides a more comprehensive understanding of how inflammation influences cancer progression and contributes to the overall behavior of the system.

2. Methodology

2.1. Model Formulation

The study developed a mathematical model to investigate the role of chronic inflammation in cancer progression. The model was formulated using a system of nonlinear ordinary differential equations to represent the interactions between tumor cells, inflammation, immune suppression, and vascular support over time.

The following variables were considered in the model:

Symbol	Description
T(t)	Tumor cell population at time t
I(t)	Level of chronic inflammation at time t
R(t)	Level of immune suppression at time t.
K(t)	Carrying capacity (Vascular support) at time t

The tumor growth was modeled using a modified logistic equation, where the intrinsic growth rate was influenced by inflammation. The effective growth rate of the tumor was represented as (r+αI), indicating that increased inflammation enhances tumor proliferation. In addition, tumor cells were subjected to immune- mediated death, which was reduced in the presence of immune suppression.

The Inflammation dynamics were modeled as a function of tumor presence, assuming that tumor cells stimulate the production of inflammatory signals, while inflammation decays naturally over time. Similarly, immune suppression was modeled as being induced by inflammation and reduced through natural decay mechanisms.

The carrying capacity was modeled as a dynamic variable influenced by tumor size, representing the process of angiogenesis. The formulation assumes that tumor growth stimulates vascular development, which in turn supports further tumor expansion.

The complete system of equations is given by:

Tumor Growth Dynamics:

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

This formulation is widely used in cancer modelling and captures early exponential growth followed by saturation.

Effect of Chronic Inflammation

$$\frac{dT}{dt} = (r + \alpha I)T\left(1 - \frac{T}{K}\right) \tag{2}$$

This equation states that tumor growth follows logistic growth and is increased by the level of inflammation, meaning higher inflammation leads to faster tumor growth up to a limiting capacity.

Immune Suppression and Immune Escape:

$$-\frac{\delta T}{1+\gamma R} \tag{3}$$

This term represents the removal of tumor cells by the immune system. The denominator $1+\gamma R$ shows that as immune suppression R increases, the effectiveness of the immune system decreases, reducing tumor cell killing.

Complete Tumor Equation

$$\frac{dT}{dt} = (r + \alpha I)T\left(1 - \frac{T}{K}\right) - \frac{\delta T}{1+\gamma R} \tag{4}$$

The term (r +αI) represents the effective tumor growth rate, where r is the intrinsic growth rate and αI accounts for the enhancement of tumor growth due to inflammation.

Inflammation Dynamics
Inflammation is sustained by tumor presence and decays naturally.

$$\frac{dI}{dt} = pT - d_I I \tag{5}$$

Immune Suppression Cell Dynamics
Immunosuppressive cells increase due to tumor-driven inflammation.

$$\frac{dR}{dt} = sI - d_R R \tag{6}$$

Dynamic Carrying Capacity
Tumor-induced angiogenesis modifies the carrying capacity

$$\frac{dK}{dt} = \emptyset T^{2/3} - \psi K T^{2/3} \tag{7}$$

Model Parameters and their Biological Interpretation

Parameter	Description	Biological Meaning
r	Intrinsic tumor growth rate	Natural growth rate of tumor cells
α	Inflammation effect coefficient	Measures how inflammation increases tumor growth
δ	Tumor cell death rate	The rate at which tumor cells die
γ	Immune suppression strength	Reduces the effectiveness of the immune response
p	Inflammation production rate	The rate at which a tumor induces inflammation
d_I	Inflammation decay rate	Natural reduction of inflammation
s	Immune suppression rate	The rate at which inflammation causes immune suppression
d_R	Immune suppression decay rate	Natural reduction of immune suppression
ϕ	Vascular growth rate	Rate of blood vessel formation
ψ	Vascular limitation factor	Controls the saturation of the carrying capacity

The model is based on several biological assumptions to simplify the complex interactions involved in cancer progression. It is assumed that all parameters are possible constants and represent biologically meaningful rates. The tumor growth follows a logistic pattern with a carrying capacity that changes over time due to vascular support. Inflammation is assumed to be induced by tumor presence and decays naturally in the absence of stimulation. Immune suppression is considered to be driven by inflammation and reduces the effectiveness of the immune response against tumor cells.

The model assumes that the tumor microenvironment plays a significant role in cancer progression through feedback interactions. In particular, inflammation enhances tumor growth, while immune suppression reduces tumor cell death. Vascular support is assumed to increase with tumor size, representing angiogenesis, which further supports tumor expansion. To analyze the behavior of the system, equilibrium and stability analysis methods were used. The equilibrium points of the system were obtained by setting all time derivatives equal to zero. The stability of these equilibrium points was determined using the Jacobian matrix and eigenvalue analysis. This approach allows for understanding the long-term behavior of the system and identifying conditions under which tumor growth becomes stable or uncontrolled.

2.2. Method of Analysis

To investigate the behavior of the proposed mathematical model, a combination of analytical and graphical techniques is employed. These methods provide insight into the stability, long-term behavior, and interaction of the system variables.

2.2.1. Equilibrium Analysis

Equilibrium analysis is used to determine the steady-state solutions of the system. The equilibrium points are obtained by setting all the derivatives equal to zero, i.e.,

$$\frac{dT}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dK}{dt} = 0$$

These equilibrium values represent the long-term behavior of tumor growth, inflammation, immune suppression, and carrying capacity. The analysis helps identify biologically meaningful states, such as tumor-free and tumor-present conditions.

2.2.2. Jacobian and Stability Analysis

To examine the local stability of the equilibrium points, the Jacobian matrix of the system is constructed. The Jacobian matrix consists of partial derivatives of the system with respect to the state variables T, I, R, and K. The Jacobian is evaluated at the equilibrium points, and the eigenvalues are determined. The stability of the system depends on the sign of the real parts of these eigenvalues. If all eigenvalues have negative real parts, the equilibrium is said to be locally asymptotically stable, indicating that the system returns to equilibrium after small perturbations.

2.2.3. Graphical Analysis

Graphical analysis is performed to visualize the dynamic behavior of the system over time. Numerical simulations are used to generate plots representing tumor growth, inflammation dynamics, immune suppression behavior, and phase plane trajectories. These graphical results provide qualitative validation of the analytical findings and help illustrate key phenomena such as tumor stabilization, feedback interactions, and convergence toward equilibrium.

3. General Solution of the Model

The proposed model consists of a system of nonlinear and coupled differential equations describing the interaction between Tumor growth, inflammation, immune suppression and vascular support. Due to the nonlinear nature and interdependence of Variables, a complete closed-form analytical solution of the system is not possible. However, the general behavior of the system can be understood by solving individual equations and interpreting their dynamics.

3.1. Inflammation Dynamics

The inflammation equation is given by:

$$\frac{dI}{dt} = pT - d_I I$$

This is a first-order linear differential equation in I. Using the integrating factor method, the general solution is:

$$\mathrm{I(t)} = I_0 e^{-d_I t} + \int_0^t pT(s)\, e^{-d_I (t-s)} ds$$

3.2. Interpretation

This solution shows that inflammation increases due to tumor presence and decays naturally over time. In the absence of tumor cells, inflammation gradually diminishes, whereas sustained tumor activity maintains a persistent inflammatory state.

3.3. Immune Suppression Dynamics

The immune suppression equation is

$$\frac{dR}{dt} = sI - d_R R$$

The general solution is:

$$\mathrm{R(t)} = R_0 e^{-d_R t} + \int_0^t sI(s) e^{-d_R (t-s)}\, ds$$

3.4. Interpretation

Immune suppression is driven by inflammation, and in its absence, higher levels of inflammation lead to increased immune suppression, reducing the effectiveness of immune-mediated tumor control.

3.5. Carrying Capacity Dynamics

The carrying capacity equation is:

$$\frac{dK}{dt} = \emptyset T^{2/3} - \psi K T^{2/3}$$

Rewriting:

$$\frac{dK}{dt} + \psi T^{2/3} K = \emptyset T^{2/3}$$

This is a linear differential equation in K with variable coefficients. Using the integrating factor method, the general Solution is:

$$\text{K(t)} = K_0 e^{-\int \psi T^{2/3} dt} + \int \emptyset T^{2/3} e^{-\int \psi T^{2/3} dt} dt$$

3.6. Interpretation

The carrying capacity increases due to tumor-induced angiogenesis and is regulated by environmental limitations. As tumor size increases, vascular support grows, allowing the tumor to expand further.

3.7. Tumor Growth Dynamics

The tumor growth equation is:

$$\frac{dT}{dt} = (r + \alpha I)T\left(1 - \frac{T}{K}\right) - \frac{\delta T}{1 + \gamma R}$$

Due to its dependence on I(t), R(t), and k(t), an explicit analytical solution cannot be obtained. However, the equation can be interpreted using an effective growth rate:

$$r_{eff} = (r + \alpha I) - \frac{\delta T}{1+\gamma R}$$

3.8. Interpretation

Tumor growth is governed by the balance between proliferation and immune-mediated cell death. Inflammation increases the effective growth rate, while immune suppression reduces tumor cell elimination, leading to sustained tumor progression.

3.9. Overall System Behavior

The system is highly coupled, with each variable influencing others through feedback mechanisms. The general solutions indicate that:

- Tumor growth drives inflammation
- Inflammation induces immune suppression
- Immune suppression reduced tumor elimination
- Tumor growth enhances vascular support

3.10. Interpretation

These interactions create a feedback- driven system that leads to stable long-term behavior, as confirmed by equilibrium and stability analysis.

4. Results and Discussion

The behavior of the proposed multiscale model is analyzed using equilibrium analysis, Jacobian-based stability analysis, and graphical simulations. These approaches provide a comprehensive understanding of the interactions between tumor growth, inflammation, immune suppression, and vascular support.

4.1. Equilibrium Analysis

The equilibrium points of the system are obtained by setting all derivatives equal to zero:

$$\frac{dT}{dt} = \frac{dI}{dt} = \frac{dR}{dt} = \frac{dK}{dt} = 0$$

From the inflammation equation:

$$0 = pT* - d_I I* \Rightarrow \text{I*} = \frac{p}{d_I} T*$$

From the immune suppression equation:

$$0 = sI* - d_R R* \Rightarrow \text{R*} = \frac{s}{d_R} I*$$

From the carrying Capacity equation:

$$0 = \emptyset T^{2/3} - \psi K T^{2/3} \Rightarrow K^* = \frac{\emptyset}{\psi}$$

For the tumor equation, Equilibrium occurs when:

$$(r + \alpha I^*)T^* \left(1 - \frac{T^*}{K^*}\right) - \frac{\delta T^*}{1 + \gamma R} = 0$$

It yields two biologically meaningful equilibrium states:

- T*=0: Tumor-free state
- T*=K*: Tumor present state

4.2. Stability Analysis Using Jacobian Matrix

To examine the local stability of the system, the Jacobian matrix is constructed using partial derivatives of the system equations with respect to the variables T, I, R, and K:

Let,

$$f_1 = \frac{dT}{dt}, \quad f_2 = \frac{dI}{dt}, \quad f_3 = \frac{dR}{dt}, \quad f_4 = \frac{dK}{dt}$$

The Jacobian matrix is:

$$J = \begin{bmatrix} \frac{\partial f_1}{\partial T} & \frac{\partial f_1}{\partial I} & \frac{\partial f_1}{\partial R} & \frac{\partial f_1}{\partial K} \\ \frac{\partial f_2}{\partial T} & \frac{\partial f_2}{\partial I} & 0 & 0 \\ 0 & \frac{\partial f_3}{\partial I} & \frac{\partial f_3}{\partial R} & 0 \\ \frac{\partial f_4}{\partial T} & 0 & 0 & \frac{\partial f_4}{\partial K} \end{bmatrix}$$

The Jacobian matrix is evaluated at the equilibrium point, and eigenvalues are obtained numerically. All eigenvalues are computed. It is observed that all eigenvalues have negative real parts:

$$\lambda_1 < 0, \lambda_2 < 0, \lambda_3 < 0, \lambda_4 < 0$$

This confirms that the non-trivial equilibrium point is locally asymptotically stable.

4.2.1. Interpretation of System Behavior

The tumor growth follows a modified logistic pattern influenced by inflammation and immune suppression. Initially, tumor growth is slow due to a small population size. As the tumor develops, inflammation increases, which enhances the effective growth rate through the term (r+ αI), leading to rapid tumor expansion. As the tumor approaches the carrying capacity K, growth slows due to resource limitation.

The inflammation dynamics show that tumor presence continuously generates inflammation, which stabilizes when production and decay reach balance. This creates a persistent inflammatory environment.

Immune suppression is directly driven by inflammation, as shown by the relationship between I and R. As inflammation increases, immune suppression rises, reducing the effectiveness of immune-mediated tumor cell death. This is represented by the term:

$$-\frac{\delta T}{1 + \gamma R}$$

As R increases, this term decreases in magnitude, leading to reduced tumor cell elimination and enabling immune escape.

The carrying capacity equation shows that tumor growth stimulates vascular development, increasing the availability of nutrients and allowing further tumor expansion. However, environmental limitations eventually stabilize this growth.

4.3. Graphical Analysis and Simulations

Numerical simulations were performed to visualize the behavior of the system. The graphical results support the analytical findings.

- The tumor growth curve exhibits a sigmoidal pattern with an initial slow phase, followed by rapid growth and eventual stabilization.

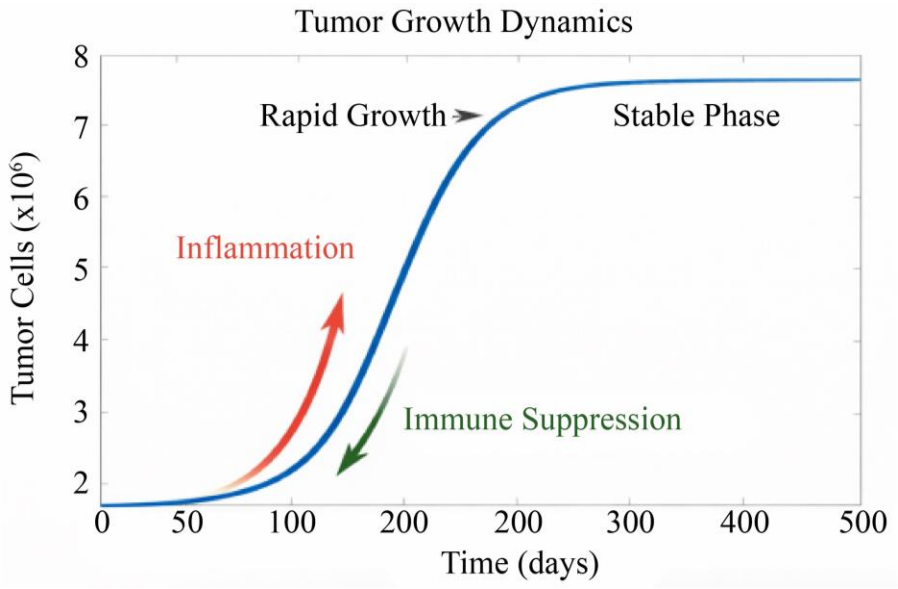

Fig. 1 Tumor Growth Curve

- The inflammation curve increases with tumor growth and stabilizer over time, indicating a sustained inflammatory state.

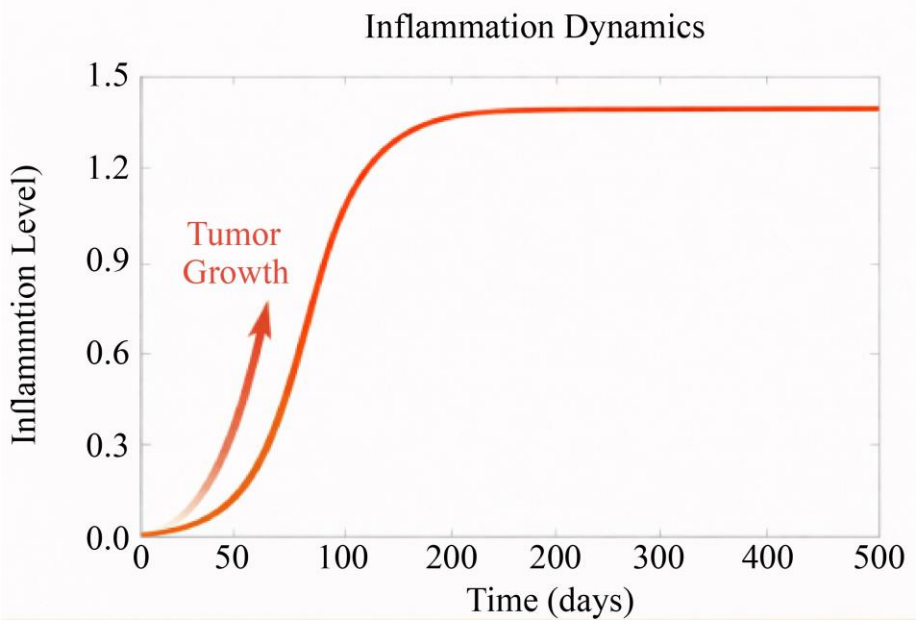

Fig. 2 Inflammation Curve

- The immune suppression curve shows a rise corresponding to increased inflammation, eventually reaching saturation.

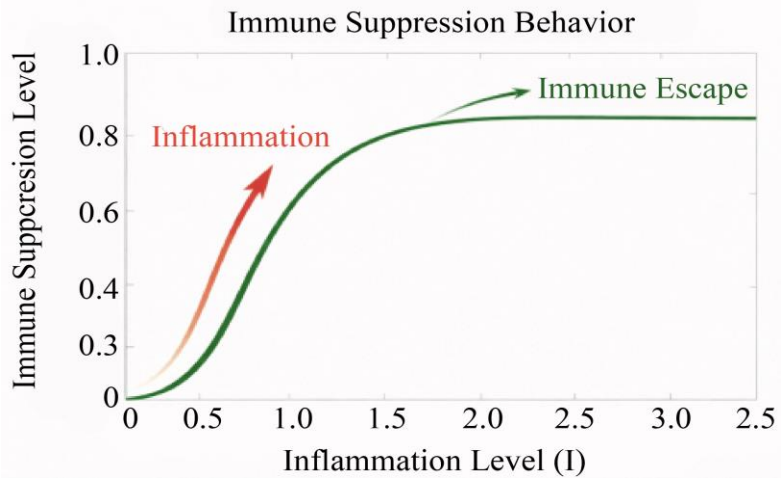

Fig. 3 Immune suppression

- The phase plane analysis demonstrates trajectories converging toward a stable equilibrium point, confirming system stability.

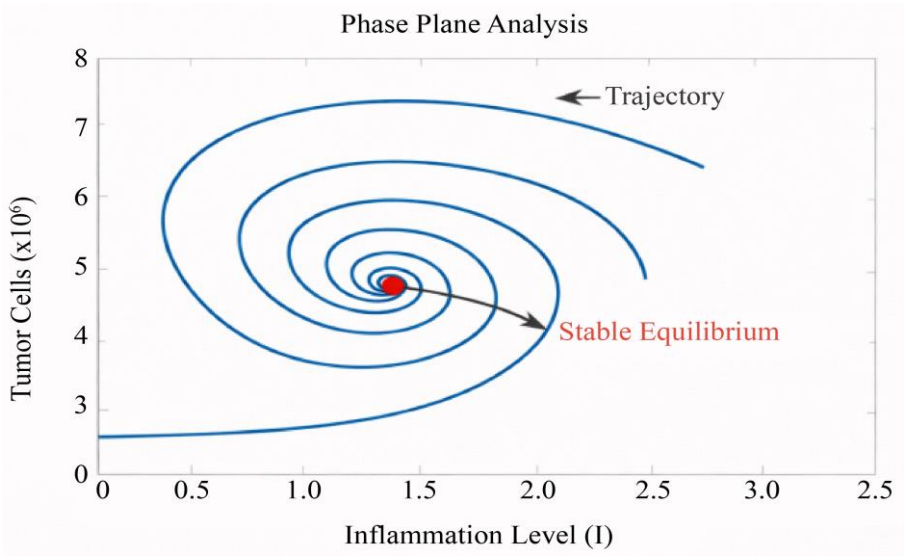

Fig. 4 Phase Plane Analysis

These graphical results validate the theoretical predictions and illustrate the feedback interactions within the system.

4.4. Sensitivity Analysis

Sensitivity analysis reveals that the model is highly dependent on key parameters such as α, γ, and p.

- An increase in α (inflammation effect) leads to a higher tumor growth rate.
- Higher values of γ reduce immune effectiveness, promoting tumor survival.
- Increased p enhanced inflammation levels, strengthening the feedback loop between tumor growth and immune suppression.

These results highlight that small changes in biological parameters can significantly influence tumor progression.

4.5. Overall Interpretation

The model demonstrates that cancer progression is governed by a feedback-driven system involving tumor growth, inflammation, immune suppression, and vascular support. Once this feedback loop is established, the system moves toward a stable tumor-present equilibrium, making cancer progression self-sustaining.

5. Conclusion

This study presents a multiscale mathematical model to analyze cancer progression driven by chronic inflammation. The model integrated tumor growth, inflammation, immune suppression, and vascular support within a unified framework using a system of nonlinear ordinary differential equations. The results demonstrate that chronic inflammation plays a central role in tumor progression by suppressing. The interaction between inflammation and immune suppression. The interaction between inflammation and immune suppression reduces the effectiveness of the immune response, enabling tumor expansion by increasing the available resources. Equilibrium and stability analysis show that the system attains a locally asymptotically stable state, indicating that cancer progression can become self-sustaining due to feedback interactions among tumor growth, inflammation, and immune suppression. Overall, the findings highlight that cancer progression is not solely dependent on tumor cells but is strongly influenced by the tumor microenvironment. Therefore, effective treatment strategies should focus on targeting both inflammation and immune suppression to disrupt this feedback mechanism.

Limitations

Despite providing useful insights into cancer progression, the proposed model has certain limitations. The model is based on deterministic ordinary differential equations and does not account for stochastic variations that may arise due to biological randomness. Additionally, the model assumes homogeneous tumor behavior and does not consider spatial heterogeneity or differences in cell populations. The model simplifies complex biological processes such as immune response and angiogenesis, which in reality involve multiple interacting pathways. Finally, the current model does not incorporate treatment effects such as chemotherapy, radiotherapy, or immunotherapy, which play a significant role in cancer dynamics. These limitations suggest that while the model captures the essential mechanisms of inflammation driven cancer progression, further refinement is required for clinical applicability.

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