The interplay between tumor cells and the immune system.

Wengen Yu*

Department of Dermatology, Sun Yat-sen University, China

Introduction

The relationship between tumor cells and the immune system is complex and dynamic, fundamentally influencing cancer progression, metastasis, and patient outcomes. Tumors have evolved intricate mechanisms to evade immune detection and destruction, leading to immune suppression and promoting tumor survival. Conversely, the immune system can recognize and eliminate tumor cells, highlighting the importance of understanding this interplay in developing effective cancer therapies. This article explores the mechanisms through which tumor cells evade immune responses, the role of the immune system in tumor surveillance, and current therapeutic strategies that leverage this interaction [1].

Tumors employ several strategies to evade the immune system, which can be categorized into immunoediting, immunosuppression, and antigen loss. This process refers to the selective pressure exerted by the immune system on tumor cells, leading to the emergence of variants that can survive immune attack. Initially, the immune system can recognize and eliminate tumor cells; however, through immunoediting, resistant variants can arise that may express lower levels of target antigens or other changes that diminish their visibility to the immune system [2]

Tumors can create a local immune-suppressive microenvironment by secreting cytokines and other factors that inhibit immune responses. For example, tumor-associated macrophages (TAMs) often adopt an immunosuppressive phenotype, secreting anti-inflammatory cytokines such as IL-10 and TGF-β, which can inhibit T cell activation and promote regulatory T cell (Treg) development [3].

Some tumor cells may lose or downregulate the expression of specific antigens recognized by the immune system, rendering them less detectable. This antigenic modulation can occur through genetic mutations or epigenetic changes, allowing tumor cells to escape immune surveillance. The immune system constantly patrols the body for abnormal cells, including those that may become cancerous. This process, known as immune surveillance, involves various immune cells, including cytotoxic T lymphocytes (CTLs), natural killer (NK) cells, and macrophages, which can recognize and destroy cancer cells [4].

Tumor cells can express unique or overexpressed antigens, known as tumor-associated antigens (TAAs) or tumor-specific antigens (TSAs), which can be recognized by the immune system. For instance, proteins derived from mutated oncogenes or tumor suppressor genes can serve as TSAs, prompting an immune response [5].

The complex interplay between tumor cells and the immune system has led to the development of several innovative therapeutic strategies aimed at enhancing anti-tumor immunity. Immune checkpoint inhibitors, such as anti-PD-1 and anti-CTLA-4 antibodies, have revolutionized cancer therapy by blocking inhibitory pathways that dampen T cell responses. By unleashing T cells from these checkpoints, these therapies have demonstrated significant efficacy in various cancers, including melanoma, lung cancer, and renal cell carcinoma [6].

Therapeutic cancer vaccines aim to enhance the immune response against tumor antigens. By stimulating the immune system to recognize and attack tumor cells, these vaccines can provide a more targeted and sustained immune response. For example, sipuleucel-T, a vaccine for prostate cancer, has shown effectiveness in clinical trials [7].

Chimeric antigen receptor T cell (CAR-T) therapy has emerged as a groundbreaking approach for treating hematologic malignancies. By engineering T cells to express receptors that specifically target tumor antigens, CAR-T therapy has achieved remarkable success in treating diseases like acute lymphoblastic leukemia and certain lymphomas [8].

Combining different immunotherapies or combining immunotherapy with conventional treatments, such as chemotherapy or radiation, is an active area of research. This approach aims to enhance anti-tumor responses and overcome the immunosuppressive microenvironment often created by tumors [9].

When tumor antigens are presented on major histocompatibility complex (MHC) molecules by antigen-presenting cells (APCs), they can activate T cells and generate a robust adaptive immune response. Helper T cells promote the activation of CTLs, which can directly kill tumor cells, while B cells produce antibodies targeting tumor antigens [10].

Conclusion

The interplay between tumor cells and the immune system is a dynamic and complex relationship that plays a critical role in cancer progression and treatment outcomes. Understanding the mechanisms of immune evasion and the various ways the immune system can recognize and eliminate tumor cells has

***Correspondence to:** Wengen Yu, Department of Dermatology, Sun Yat-sen University, China. E-mail: weng.yu@163.com

***Received:** 27-Sep-2024, Manuscript No. AAJCIT-24-150272; **Editor assigned:** 28-Sep-2024, Pre QC No. AAJCIT-24-150272(PQ); **Reviewed:** 14-Oct-2024, QC No AAJCIT-24-150272; **Revised:** 19-Sep-2024, Manuscript No. AAJCIT-24-150272(R); **Published:** 28-Sep-2024, DOI:10.35841/aajcit-7.5.232*

***Citation:** Yu W., The interplay between tumor cells and the immune system. J Cancer Immunol Ther. 2024;7(5):232*

led to significant advancements in cancer therapies. Immune checkpoint inhibitors, cancer vaccines, CAR-T cell therapy, and combination approaches are transforming the landscape of cancer treatment, offering hope to patients with previously limited options. Continued research into the intricacies of this interplay will pave the way for new therapeutic strategies and improved outcomes for cancer patients.

References

1. Sabbatino F, Liguori L, Polcaro G, Salvato I, Caramori G, Salzano FA, Casolaro V, Stellato C, Dal Col J, Pepe S. Role of human leukocyte antigen system as a predictive biomarker for checkpoint-based immunotherapy in cancer patients. Int J Mol Sci. 2020;21(19):7295.
2. Mahmoud F, Shields B, Makhoul I, Avaritt N, Wong HK, Hutchins LF, Shalin S, Tackett AJ. Immune surveillance in melanoma: From immune attack to melanoma escape and even counterattack. Cancer Biol Ther. 2017;18(7):451-69.
3. Sordo-Bahamonde C, Lorenzo-Herrero S, Granda-Díaz R, Martínez-Pérez A, Aguilar-García C, Rodrigo JP, García-Pedrero JM, Gonzalez S. Beyond the anti-PD-1/PD-L1 era: promising role of the BTLA/HVEM axis as a future target for cancer immunotherapy. Mol Can. 2023;22(1):142.
4. Fernando K, Kwang LG, Lim JT, Fong EL. Hydrogels to engineer tumor microenvironments in vitro. Biomat Sci. 2021;9(7):2362-83.
5. Kantoff PW, Higano CS, Shore ND, Berger ER, Small EJ, Penson DF, Redfern CH, Ferrari AC, Dreicer R, Sims RB, Xu Y. Sipuleucel-T immunotherapy for castration-resistant prostate cancer. N Eng J Med. 2010;363(5):411-22.
6. Maude SL, Laetsch TW, Buechner J, Rives S, Boyer M, Bittencourt H, Bader P, Verneris MR, Stefanski HE, Myers GD, Qayed M. Tisagenlecleucel in children and young adults with B-cell lymphoblastic leukemia. N Eng J Med. 2018;378(5):439-48.
7. Mellman I, Coukos G, Dranoff G. Cancer immunotherapy comes of age. Nat. 2011;480(7378):480-9.
8. Noy R, Pollard JW. Tumor-associated macrophages: from mechanisms to therapy. Immunity. 2014;41(1):49-61.
9. Pardoll DM. The blockade of immune checkpoints in cancer immunotherapy. Nat Rev Cancer. 2012;12(4):252-64.
10. Schreiber RD, Old LJ, Smyth MJ. Cancer immunoediting: integrating immunity's roles in cancer suppression and promotion. Sci. 2011;331(6024):1565-70.

Citation: *Yu W., The interplay between tumor cells and the immune system. J Cancer Immunol Ther. 2024;7(5):232*