

# Tumour immunology: Understanding the interplay between cancer and the immune system.

Bhatnagar Schorey\*

Department of Biological Sciences, University of Notre Dame, Notre Dame, USA

## Introduction

Tumor immunology is a rapidly evolving field that investigates the complex interactions between cancer cells and the immune system. It focuses on understanding how tumors develop, evade immune surveillance, and explore innovative approaches to harness the power of the immune system for cancer treatment. This article provides an overview of tumor immunology, highlighting the key components and mechanisms involved in the interplay between cancer and the immune system. The immune system plays a crucial role in recognizing and eliminating cancer cells. The innate immune system provides an initial response through natural killer cells, dendritic cells, and macrophages, while the adaptive immune system mounts a targeted response through T cells and B cells. These immune cells recognize tumor-specific antigens and initiate immune responses to eliminate cancer cells. However, tumors have developed multiple mechanisms to evade immune recognition and suppression [1].

The tumor microenvironment is a dynamic ecosystem consisting of cancer cells, stromal cells, and immune cells. Immune cell infiltration within the tumor microenvironment is a hallmark of anti-tumor immune responses. Tumor-Infiltrating Lymphocytes (TILs), including cytotoxic T cells and natural killer cells, are key players in recognizing and eliminating cancer cells. However, the tumor microenvironment can create an immunosuppressive milieu through the secretion of immunosuppressive molecules, recruitment of regulatory T cells, and expression of inhibitory receptors, hindering effective anti-tumor immune responses [2].

## Immune evasion mechanisms

Tumors have developed various strategies to evade immune recognition and destruction. These mechanisms include down regulation of tumor antigens, disruption of antigen presentation pathways, expression of immune checkpoint molecules, and recruitment of immunosuppressive cells such as myeloid-derived suppressor cells and regulatory T cells. Understanding these immune evasion mechanisms is critical for developing strategies to overcome tumor resistance and enhance anti-tumor immune responses.

The process of cancer immuno editing involves the immune system interacting with tumour cells. Elimination, equilibrium, and escape are its three stages. The "three Es" of cancer

immuno editing are frequently referred to as these stages. Immuno editing involves both innate and adaptive immune systems. The immune response causes the tumour cells to be destroyed during the elimination phase, which suppresses the tumour. Certain tumour cells, however, may develop more mutations, alter their traits, and avoid the immune system. These cells may enter the equilibrium phase, in which the immune system does not recognise all tumour cells but the tumour does not enlarge at the same time [3].

Immune checkpoint inhibitors have revolutionized cancer treatment by restoring and enhancing anti-tumor immune responses. These inhibitors block inhibitory checkpoint molecules such as Programmed Death-1 (PD-1) and Cytotoxic T-Lymphocyte-Associated protein 4 (CTLA-4), enabling reactivation of T cell function and unleashing their anti-tumor activity. Checkpoint inhibitors have shown remarkable efficacy in various cancers, leading to long-lasting responses and improved patient outcomes.

It is reasoned that by acting as a "bystander effect," the immune system could contribute to the elimination of chemotherapy-resistant cancer cells. How the immune response is initiated against dying cancer cells, however, still requires a lot of study. According to experts in the area, necrotic cell death is truly immunogenic whereas apoptotic cell death is just marginally immunogenic. This may be due to the immune response being induced by the maturation of dendritic cells as a result of the inflammatory response being stimulated when cancer cells are eliminated via a necrotic cell death pathway [4].

An environment favourable to immunogenicity is created by anthrax cyclines. According to the researchers, this drug encourages antigen uptake and presentation by dendritic cells when killing cancer cells, triggering a T-cell response that can decrease tumours. Hence, the efficacy of immunotherapy depends on the activation of tumour-killing T-cells [5].

## References

1. Sugiyama D, Hinohara K, Nishikawa H. Significance of regulatory T cells in cancer immunology and immunotherapy. *Exp Dermatol.* 2022;2.
2. Botta C, Maia C, Garcés JJ, et al. FlowCT for the analysis of large immunophenotypic data sets and biomarker discovery in cancer immunology. *Blood Adv.* 2022;6(2):690-703.

\*Correspondence to: Bhatnagar Schorey, Department of Biological Sciences, University of Notre Dame, Notre Dame, USA, E-mail: bhatnagar@nd.edu

Received: 04-Apr-2023, Manuscript No. AACIR-23-97675; Editor assigned: 07-Apr-2023, Pre QC No. AACIR-23-97675 (PQ); Reviewed: 21-Apr-2023, QC No. AACIR-23-97675;

Revised: 24-Apr-2023, Manuscript No. AACIR-23-97675 (R); Published: 28-Apr-2023, DOI: 10.35841/aacir-6.2.140

3. Sánchez-Paulete AR, Teijeira A, Cueto FJ, et al. Antigen cross-presentation and T-cell cross-priming in cancer immunology and immunotherapy. *Ann Oncol.* 2017;28:44-55.
4. Dranoff G. Experimental mouse tumour models: what can be learnt about human cancer immunology?. *Nat Rev Immunol.* 2012;12(1):61-6.
5. Old LJ. Cancer immunology: the search for specificity—GHA Clowes Memorial lecture. *J Cancer Res.* 1981;41(2):361-75.