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# Advances in Cancer Treatment

**Ilson Sepulveda***
Department of Oral and Maxillofacial Radiology, University of Concepción, Chile

**Abstract**

Schreiber and his group provided in experimental evidence supporting the concept of immune surveillance for cancer. However, they had also suggested that tumors developed in the presence of healthy immune system are less immunogenic compared to those that are developed in an immune-compromised host makes the immune system paradoxical in favouring the eventual growth of tumors leading to the escape of the immune response that is better able to escape the immune response.

**Keywords:** Immunogenic; Molecules; Mechanism; Cancer cells; Surgery; Conventional therapies

## Introduction

The immune system has four basic tumour eradication strategies; the host is protected from virus-induced tumors by immune shedding of viral load. In case of inflammation, the rapid clearance of pathogens and responseofinflammationpreventstheinflammatorymicroenvironment from advancing into the tumour. The immune system identifies explicitly TAAs or molecules secreted by cells under stress to kill tumors [1]. The immune system identifies precancerous and cancerous cells and eradicates them before the damage occurs. As we all know, nothing is perfect in this world likewise, our body's defense mechanism is not as perfect as it should be able to eradicate the cancer cells. As a result, some tumour cells take advantage and escape the immune surveillance to promote proliferation of the cancer cells. In addition, these tumors are less immunogenic to evade the immune response. The genetic and/or epigenetic alterations in a normal cell transform them into cancer cells. Whereas, it is important to understand the biology of cancer cells which has two standard characteristic features, an uncontrolled cell division and their invasive ability either locally or at distant sites. It is well established that if oncogenes regulate cancer initiation then their progression is further guided by tumour microenvironment. In addition, the inflammatory cells can also influence cancer progression in the tumour microenvironment by distorting the metastatic ability of tumour cells. The six known characteristic features of cancer are: unrestricted replication, predetermined growth signals, insensitivity to growth inhibitors, circumvents programmed cell death, blood vessel development, tissue invasion, and metastasis where in addition to these, cancer-related inflammation is now becoming seventh [2]. Recently, immunotherapy has shown positive patient outcomes in various clinical trials wherein various exogenously modified immune molecules are being manipulated to provide better immune response over conventional therapies, such as chemotherapy/radiotherapy or both along with surgery.

## Discussion

Immunotherapies are also recently used with adjuvants, which are termed as neo-adjuvant therapies. These therapies either encourage the activities of specific cells of the immune system or deactivate the signals produced by the cancer cells that help in suppressing the immune response. Therapies, including the endogenous immune mechanisms against cancer will act as a potent determinant to recognize the malignant cells as foreign agents. However, in order to achieve this, multiple immune pathways should be targeted simultaneously, which may offer better clinical outcomes [3]. Role of immune cells in cancer As described in the previous section, the immune cells play a crucial role in carcinogenesis. Both innate immune cells and adaptive immune cells participate either in cancer progression or cancer suppression. The innate immune cells act as the first line of defense against any pathogen which includes dendritic cells, macrophages, neutrophils, mast cells and natural killer cells, whenever, micro environment around the normal tissue gets disturbed these cells secrete various cytokines, chemokines, growth factors and proteases which hampers the cascade of events that leads to inflammation. Also, the adaptive immune cell-like T-cells and B-cells react to tumour micro environment, thus making a favorable environment for inflammatory response. These innate and adaptive immune cells in the tumour micro-environment communicate with the cancer cells and the surrounding stromal cells by auto-crine and paracrine mechanism [4]. In an aggressive and established tumour, the immune response generates towards the pro-inflammatory signalling which results in regression of these tumors very rarely. Both pro-tumour and anti-tumour immune responses co-exist with each other but which way the tumour has to progress is dependent on the tumour micro environment. Most of the immune cells are involved in the tumour micro environment, where tumour-associated macrophages and T cells are mainly present in the area where the tumour is present. TAMs mostly promote tumour growth, angiogenesis, invasion and migration and their increased infiltration leads to the poor prognosis of cancer. Mature T-cells are broadly categorized into two major types based on the presence of T cell receptors, γδ and αβ. αβ-T cells can be further divided into various subgroups like CD8+ cytotoxic T cells and CD4+ helper T cells. These Th cells further include Th1, Th2, Th17 and Treg, as well as NK cells. T cells can utilize both pro-tumour and anti-tumour effects, where an increase in T cell numbers can activate an increased population of Tc and Th cells which sometimes help in better survival of patients suffering from various types of cancers, melanoma, invasive colon cancer, multiple myeloma, and pancreatic cancers. Sometimes, the lower number of Tc cell involvement increases the susceptibility in experimental animal models towards spontaneous or chemical carcinogenesis. It has also been observed that in case of solid tumors, various T cell types cause tumour progression. Till now it has

***Corresponding author:** Ilson Sepulveda, Department of Oral and Maxillofacial Radiology, University of Concepción, Chile, E-mail: isepulvedaa@udec.cl

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been reported that NK cells lack pro-tumorigenic role [5]. Similarly, TAMs and lymphocytes also play a major role in tumour progression including Treg cells which act in a pro tumorigenic manner by suppressing the anti-tumour immune responses. Leucocytes forming the major group of the immune cells due to which these can be one of the important determinants among hallmarks of cancer as cancer-related inflammation is also considered as the seventh hallmark of cancer. Previously, it was believed that the leucocytes help in immune surveillance to eradicate the tumour, but their diverse role has changed the concept in carcinoma-induced sarcomas and spontaneous epithelial carcinomas where they have shown protection against lymphocytes and IFN-γ [6]. In breast cancer, the occurrence of TILs with a high number of CD4+/CD8+ and the Th2/Th1 ratio is one of the indicators of poor cancer prognosis. Progression and metastasis of mammary cancer is stimulated by Th2 CD4+ T cells by targeting TAMs, giving rise to pro-angiogenic and pro-metastatic factors. Similarly like these immune cells, breast cancer cells also produce several pro-tumorigenic cytokines and chemokines like IL-4, IL-6, IL-8 and CXCR-4, CCL-2, CCL-5 respectively, which cause tumour progression. Till now, the degeneracy of T cell is not clear which arises various queries regarding the factors that determine the fate of T cell whether it will act as anti- or pro-tumorigenic in different types of cancers. As a consequence, these factors are one of the significant factors in immune-therapeutics [7]. The above-mentioned phenomena may be collectively called as tumour-immuno printing strategy, where both the innate and adaptive immune cells infiltrate the tumour-stroma and making it more favorable for tumour progression and escape from a further immune response in the tumour micro-environment, and which may have a beneficial impact on both the diagnostic and prognostic approach for cancer management. Recent studies have highlighted that the immune system may promote the emergence of primary tumour tissues and evade the immune selection process, rather than acting as a suppressor of the disease that might lead to the progression of cancer. Immune surveillance is known to regulate not only in host protection but also the advancement of the tumour in three major steps including elimination, equilibrium, and escape. The process starts when the normal cells are induced to change into transformed cells [8]. The first phase-elimination helps the cancer immune surveillance using extrinsic tumour suppressor response to clear out those transformed cells, thus, giving protection against cancer which is mostly T-cell dependent. If the elimination process fails to clear the transformed cells, then the second phase, i.e., equilibrium comes in action, where cancer persistence occurs due to the genetic instability and immune response [9]. In which the transformed cells maintain their favorable microenvironment to expand their number for the maintenance of cancerous condition. Tumour cells having reduced immunogenicity can survive better in an immune-competent host however maintenance leads to the escape from immunological surveillance which allows third phase to act resulting in growth of the cancer. During this phase, immune-edited cells grow uncontrollably through immune pressure determining as invasive tumors whereas in other models, a tumour-mediated active immunosuppression is found to enhance the tolerance level of tumour-specific T cell as a dominant immune escape mechanism. In cancer patients, immune-editing shows the main effect of the triple E theory where clinically seeming tumours inherit the immune response resistance by escaping the adaptive immunity. It can help the process of complete inalterability of most immunotherapies and vaccines for cancer therapy, in a small population of patients with even immunogenic diseases such as melanoma. Neoplastic cells also have capability to enter the inflammatory pathways leading to tumour development by recruiting leukocytes, however, the underlying mechanisms in tumour-mediated inflammatory responses is still unclear [10].

## Conclusion

The innate immune cells belonging to myeloid lineage composed of TAMs and immature myeloid cells are found to be involved intrinsically. These cells produce various chemokines, cytokines, proteases and several growth factors, which may promote tumour growth; and mediate local or systemic immune-suppression by inducing angiogenesis and tissue remodelling.

## Acknowledgement

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## Conflict of Interest

None

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