Editorial

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Targeting Immune-Mediated Suppressive Mechanisms as a Novel Cancer Immunotherapy Strategy

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Editorial Note

Over the last decade, new types of cancer treatments, generally known as cancer immunotherapies, have developed and shown amazing outcomes, but only in a small percentage of cancer patients. Immunotherapies stimulate or restore effective anti-tumor immune responses by mobilizing the immune system. Immune Checkpoint Inhibitors (ICIs) that target the CTLA-4/B7 and PD1/PDL1 immune checkpoints (so-called ICIs) are currently widely used in clinical trials. Although ICIs frequently provide long-term benefits, full or virtually complete tumor responses are rare, and resistance is shown in a significant proportion of patients. Primary or acquired resistance to ICIs is widespread, and identifying predictive indicators of effectiveness or resistance is challenging. The search for new alternative or complementary targets that activate, release, or augment anticancer immune responses is presently in full swing.

ICIs fine-tune the immune response and control hyperactivation by acting as negative regulators of T cell activation that have evolved through time. The most powerful T cell immunological checkpoint molecules are Cytotoxic T Lymphocyte Antigen 4 (CTLA4) and Programmed Cell Death 1 (PD1). They have biological effects in different parts of the body and at different periods during the T cell's lifetime. As a result, they functionally complement each other, ensuring that T cell responses maintain self-tolerance while successfully defending the body against infections and neoplasia. Several pioneering research groups have successfully targeted CTLA4 and PD1 as therapies for a range of refractory malignancies.

In this context, developing strategies to target the immunosuppressive Tumor Micro Environment (TME) is of utmost importance. The current challenges are to identify the immunosuppressive mechanisms that most significantly contribute to cancer cells' primary or acquired resistance to anti-tumor immune responses at the molecular and cellular levels, as well as to demonstrate that targeting these mechanisms has therapeutic anti-tumor activity. To create innovative ways to enhance the result and raise the proportion of patients responding to cancer immunotherapy, a deeper knowledge of the interaction between cancer cells and immune cells is necessary. Regulatory T cells (Tregs), for example, are well-known to promote local immunosuppression within tumors. They are key modulators of adaptive immunity and are required to maintain self-tolerance. Because of the variety of immunosuppressive mechanisms used by these cells to carry out their functions, the number of different Treg subpopulations identified to date, and the existence of many other cell types endowed with immunosuppressive functions, new research is needed to identify and better define novel therapeutic targets that could aid in the development of effective anti-tumor immunity.

The goal of this issue was to give a view of the immunosuppressive mechanisms that appear to predominate within the TME, as well as to identify novel, therapeutically targetable immunosuppressive processes. The different articles demonstrate how far the discipline has progressed while also emphasizing its intricacy.

