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Classifiactions of Immunopharmacology and its immune system

Chaozhan Lin*

Department of Pharmacy, University of Chinese Medicine, Guangzhou, China

*Corresponding author. E-mail: Chaozhan@lin.cn

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DESCRIPTION

Combinatorial therapy, or the use of numerous antitumor medications with various mechanisms of action, is the focus of immunopharmacology.

Additionally, drug combinations that target various stages of the cell cycle are frequently used in combinatorial treatment regimens. The best treatment plans combine medications with distinct toxicities. By doing this, it is possible to reduce overall toxicity even when a synergistic group of cytotoxic medications is used.

The development of cancer cells that are resistant to these cytotoxic cocktails may be slowed or prevented while also killing as many cells as possible.

The scientific methods that shape the therapeutic landscape have been significantly impacted by the rapid technological advancement of recent decades.

Immunopharmacology is unquestionably a key player in the current shift toward precision medicine, which is largely characterised by the development of treatments tailored to individual patients. The International Union of Basic and Clinical Pharmacology's Immunopharmacology Section (ImmuPhar) states that immunopharmacology is the newest branch of pharmacology that deals with the selective modulation, primarily up- or down-regulation, of particular immune responses, which is frequently carried out by immune cell subsets with specialised functions. Although new drug classes with improved selectivity and/or specificity are now available thanks to recent biotechnological advancements, agents with immunomodulating properties have been used in clinical practise for more than 70 years. The counteraction of the inflammatory response following the administration of cortisone in patients with rheumatoid arthritis is a pertinent example from the late 1940s.

Though relatively few studies have examined the immunopharmacology of rexinoids in the context of cancer, documentation of rexinoid-induced immunomodulatory effects along with evidence of altered PD-L1 expression in tumours of mice following treatment with a rexinoid provides justification for considering the combination of rexinoids and checkpoint inhibitors for the treatment of cancer. Elevated levels of PD-L1 are associated with improved survival in breast cancer patients, and they enhance the tumours' response to checkpoint blockade. Given that rexinoids have demonstrated excellent safety profiles in numerous clinical trials conducted on cancer patients, this therapeutic combination has the potential to increase efficacy and is deserving of further study. Despite having numerous effects on the immune system, the immunotoxicity of BRMs has not been well studied. When immunopharmacology has unintended effects on the immune system, it can turn into immunotoxicity. For instance, when combined with indomethacin, soluble -glucans (also known as sonifian), which are clinically used as antitumor agents, can cause lethal toxicity in mice. The presence of significantly increased levels of IFN-, IL-6, and colony-stimulating factor in the sera points to a malfunction of the cytokine network. The cytotoxic T-lymphocyte-associated antigen 4 (CTLA-4) antagonist of CD28 costimulation further emphasises the distinction between immunopharmacology and immunotoxicity. Abatacept is a recombinant human fusion protein used to treat adult patients with moderately to severely active rheumatoid arthritis. It consists of the extracellular domain of CTLA-4 connected to the modified Fc portion of human IgG1. Abatacept inhibits the early stages of T-cell activation, including progression into the cell cycle, effector differentiation, and cell survival; promotes passive cell death; and restricts the clonal expansion of antigen-reactive T cells. However, other *in vivo* effects have also been reported, such as increasing the production of an intracellular enzyme that inhibits T-cell activation. In some cases, blocking the negative regulator CTLA-4 can also enhance immunity by reducing regulatory T cells, which are crucial for controlling autoimmune disease. Patients

who receive abatacept may therefore be at an increased risk of infection. Patients taking abatacept may face an increased risk of latent Mycobacterium tuberculosis reactivation, but abatacept administration to mice did not worsen chronic M. At this time, it is unclear what impact abatacept's inhibition of T-cell activation will have on the emergence of malignancy.

Abatacept was administered weekly for up to 84 weeks (for males) and 88 weeks (for females) in a mouse carcinogenicity study, and this caused an increase in the incidence of malignant lymphomas (at all doses) and mammary gland tumours (mid and high doses in females). The lymphomas and mammary tumours seen in this study were thought to be secondary to the long-term induced immunomodulation in the presence of these viruses in these mice, which were positive for murine leukaemia virus and mammary tumour virus. To understand the risk to humans, it will be necessary to monitor patient data over many years and determine the clinical relevance of these findings.