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Present and future of immune checkpoint blockade: Monotherapy to adjuvant approaches

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Abstract

Immune regulation of aggressive tumor growth is often outpaced by tumor up-regulation of ligands that inhibit effector immune responses through the activation of immune checkpoints. A few of such checkpoints include programmed death-1 (PD-1), cytotoxic T lymphocyte associated antigen-4 (CTLA-4), lymphocyte activation gene-3 (LAG-3), T-cell immunoglobulin and mucin protein-3 (TIM-3), Glucocorticoid-induced TNFR family-related receptor (GITR), and killer cell immunoglobulin like receptor (KIR). With the exception of GITR, after binding to their respective ligands these checkpoints induce down-modulation of immune responses to prevent autoimmunity. However, such immune mechanisms are co-opted by tumors to allow rapid tumor cell proliferation. Pre-clinical studies in antibody blockade of PD-1 and CTLA-4 have led to promising augmentation of effector immune responses in murine tumor models, and human antibodies against PD-1 and CTLA-4 alone or in combination have

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