TIGIT family receptors : from molecular mechanisms to clinical applications

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Tumors escape immune surveillance by inducing various immunosuppressive pathways, including the activation of inhibitory receptors on tumor-infiltrating T cells. While monoclonal antibodies (mAbs) blocking programmed cell death 1 (PD-1), programmed death-ligand 1 (PD-L1), and cytotoxic T lymphocyte-associated antigen 4 (CTLA-4) have been approved for multiple cancer indications, only a subset of patients benefit from immune checkpoint blockade therapies, highlighting the need for additional approaches. Therefore, the identification of new target molecules acting in distinct or complementary pathways in monotherapy or combination therapy with PD-1/PD-L1 blockade is gaining immense interest. T cell immunoreceptor with Ig and ITIM domains (TIGIT) (TIGIT) has received considerable attention in cancer immunotherapy. Recently, anti-TIGIT mAb (tiragolumab) has demonstrated promising clinical efficacy in non-small cell lung cancer treatment when combined with an anti-PD-L1 drug (Tecentriq), leading to phase III trial initiation. TIGIT is expressed mainly on T and natural killer cells; it functions as an inhibitory checkpoint receptor, thereby limiting adaptive and innate immunity. CD226 competes for binding with the same ligands with TIGIT but delivers a positive stimulatory signal to the immune cells. We would like to present our recent advances in the understanding of the roles of TIGIT and CD226 in immune cell function and their potential application in cancer immunotherapy.