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Immune checkpoint blockade plays a crucial role in mounting antitumor immunity thereby improving cancer survival

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Immune checkpoint blockade plays a crucial role in mounting antitumor immunity thereby improving cancer survival. Immune checkpoint inhibitors (ICIs) targeting programmed cell death protein 1 (PD-1) and cytotoxic lymphocyte T associated protein 4 (CTLA-4) boost antitumor T cell responses and improve survival in patients with cancer1.However, the overall response rate to existing ICI therapies remains low; therefore, the identification of alternative immune checkpoints as therapeutic targets is required.

V-domain immunoglobulin suppressor of T cell activation (VISTA) also known as Dies1, Gi24, PD-1H, or DD1α—is a B7 family immune checkpoint protein and a next-generation immunotherapy target2. Previous studies have indicated that VISTA inhibits T cell activation by two modes of action: Firstly, VISTA on the Antigen antigen-presenting cells (APCs) and tumor cells interacts "in-trans" with its cognate receptors on T cells thereby causing T cell suppression. Secondly, VISTA expression can be observed on the T cells itself thereby eliciting "in-cis" intrinsic suppression (Fig 1). Here, we identify leucine-rich repeats and immunoglobulin-like domains 1 (LRIG1) as a novel VISTA binding partner, which acts as an inhibitory receptor by interacting with VISTA resulting in T cell receptor signaling suppression3. Mice with T cell-specific LRIG1 deletion developed condescending antitumor responses because of the expansion of tumor-specific cytotoxic T lymphocytes (CTLs) which shows increased effector function and better survival. Sustained tumor control was associated with a reduction of quiescent CTLs (TCF1+ CD62Lhi PD-1low) and a reciprocal increase in progenitor and memory-like CTLs (TCF1+ PD-1+)3. In melanoma patients, elevated LRIG1 expression on tumor infiltrating CD8+ CTLs correlated with resistance to immunotherapies3. These results delineate the role of LRIG1 as an inhibitory immune checkpoint receptor and propose a rationale for targeting the VISTA/LRIG1 axis for cancer immunotherapy.

Biography

Dia Roy completed her Ph.D. on tumor immunology from Bose Institute, India and her expertise lie on evaluating the role of T cells in shaping the tumor microenvironment. Currently she is working as a Post doctoral researcher at Lerner Research Institute, Cleveland Clinic Foundation, USA. Her post doctoral work encompasses the role of inhibitory checkpoint molecules and their interaction with their partners to shape the TME.

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