ATAGENIX LABORATORIES

Catalog Number:ATMP00025HU

Recombinant Human TIGIT protein ,C- His Tag

Product Details

Summary

English name	Recombinant Human TIGIT protein ,C- His Tag
Purity	>90% as determined by SDS-PAGE
Endotoxin level	<1.0 EU per μg of the protein as determined by the LAL method.
Construction	A DNA sequence encoding the human TIGIT(Met1~Pro141) was fused with the C-terminal His Tag
Accession #	Q495A1
Host	Mammalian cells
Species	Homo sapiens (Human)
Predicted Molecular Mass	13.99kDa
Formulation	Supplied as solution form in PBS, pH7.5 or lyophilized from PBS, pH7.5.
Shipping	In general, proteins are provided as lyophilized powder/frozen liquid. They are shipped out with dry ice/blue ice unless customers require otherwise.
Stability &Storage	Use a manual defrost freezer and avoid repeated freeze thaw cycles. Store at 2 to 8 °C for one week . Store at -20 to -80 °C for twelve months from the date of receipt.
Reconstitution	Reconstitute in sterile water for a stock solution.A copy of datasheet will be provided with the products, please refer to it for details.

Background

Background	T-cell immunoreceptor with Ig and ITIM domains (TIGIT) is also known as V-set and immunoglobulin domain-containing protein 9 (VSIG9), V-set and transmembrane domain-containing protein 3 (VSTM3),which belongs to single-pass type I membrane protein containing an immunoglobulin variable domain, a transmembrane domain and an immunoreceptor tyrosine-based inhibitory motif (ITIM). TIGIT is expressed at low levels on peripheral memory and regulatory CD4+ T-cells and NK cells and is up-regulated following activation of these cells (at protein level). TIGIT binds with high affinity to the poliovirus receptor (PVR) which causes increased secretion of IL10 and decreased secretion of IL12B and

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	suppresses T-cell activation by promoting the generation of mature immunoregulatory dendritic cells.
Alternative Names	TIGIT,VSIG9,VSTM3
References	Zhang, Liu (2020) Targeting NK Cell Checkpoint Receptors or Molecules for Cancer Immunotherapy Frontiers in immunology 11() 1295

Frontier progress

Checkpoint blockade therapy, for example using antibodies against CTLA-4 and PD-1/PD-L1, relieves T cells from the suppression by inhibitory checkpoints in the tumor microenvironment; thereby achieving good outcomes in the treatment of different cancer types. Like T cells, natural killer (NK) cell inhibitory receptors function as checkpoints for NK cell activation. Upon interaction with their cognate ligands on infected cells, tumor cells, dendritic cells and regulatory T cells, signals from these receptors severely affect NK cells' activation and effector functions, resulting in NK cell exhaustion. Checkpoint inhibition with antagonistic antibodies (Abs) can rescue NK cell exhaustion and arouse their robust anti-tumor capacity. Most notably, the response to anti-PD-1 therapy can be enhanced by the increased frequency and activation of NK cells, thereby increasing the overall survival of patients with multiple types of cancer. In addition, rescue of NK cell activity could enhance adaptive T cells' anti-tumor activity. Some antagonistic Abs (e.g., anti-TIGIT and anti-NKG2A monoclonal Abs) have extraordinary potential in cancer therapy, as evidenced by their induction of potent anti-tumor immunity through recovering both NK and T cell function. In this review, we summarize the dysfunction of NK cells in the tumor microenvironment and the key NK cell checkpoint receptors or molecules that control NK cell function. We particularly focus on recent advances in the most promising strategies through blockade of NK cell checkpoints or their combination with other approaches to more effectively reject tumors.