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Anti-mouse TIGIT (Vstm3) Antibody

Catalog Number:	209101, 209102
Size:	25 µg, 100 µg
Target Name:	TIGIT, VSTM3, T-cell immunoreceptor with Ig and ITIM domains, VSIG9
Regulatory Status:	RUO

PRODUCT DETAILS

Clone:	mTIGIT-ARg2a
Application:	Flow Cytometry
Reactivity:	Mouse
Format:	Purified
Isotype:	Rat IgG2a
Antibody Type:	Monoclonal
Formulation:	Phosphate-buffered solution, pH 7.2, containing 0.09% sodium azide
Protein Concentration:	0.5 mg/mL
Storage&Handling:	The antibody solution should be stored between 2°C and 8°C
Isotype Controls:	303501
Antibody Family:	Mouse Antibodies
Storage & Handling:	The antibody solution should be stored between 2°C and 8°C
Isotype Control:	303501

BACKGROUND INFORMATION

Mouse TIGIT, also known as T-cell immunoreceptor with Ig and ITIM domains, is an inhibitory immune checkpoint receptor encoded by the Tigit gene. It is primarily expressed on activated T cells, regulatory T cells (Tregs), and natural killer (NK) cells. TIGIT plays an important role in regulating immune-cell activation, maintaining immune tolerance, and preventing excessive inflammatory responses. By suppressing T-cell and NK-cell activity, it helps maintain immune homeostasis but can also limit effective immune responses against tumors and chronic infections.

Mouse TIGIT is a type I transmembrane glycoprotein belonging to the immunoglobulin superfamily. Its structure consists of an extracellular immunoglobulin variable-like (IgV) domain, a single transmembrane region, and a cytoplasmic tail containing an immunoreceptor tyrosine-based inhibitory motif (ITIM) and an immunoglobulin tail tyrosine (ITT)-like motif. These intracellular motifs recruit inhibitory signaling molecules, including SHIP-1, to suppress immune-cell activation. Its principal ligands are CD155 (PVR) and CD112 (nectin-2), which are members of the nectin and nectin-like adhesion molecule families. TIGIT competes with the activating receptor CD226 (DNAM-1) for ligand binding, thereby shifting the balance toward immune inhibition.

TIGIT contributes to immune dysfunction in cancer and chronic viral infections. Increased TIGIT expression on tumor-infiltrating T

cells and NK cells is frequently associated with impaired cytotoxic activity and an exhausted immune phenotype. Its immunosuppressive activity can also support regulatory T-cell function and facilitate tumor immune evasion. In autoimmune and inflammatory disease models, TIGIT signaling may help restrain excessive immune activation and tissue inflammation.

Therapeutically, TIGIT is an important target for cancer immunotherapy. Blocking antibodies against TIGIT aim to restore T-cell and NK-cell function and enhance antitumor immunity. Combining TIGIT blockade with PD-1 or PD-L1 inhibitors is being investigated to improve treatment responses. Mouse TIGIT is also a valuable research target for studying immune checkpoints, tumor immunity, and preclinical immunotherapy strategies.

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