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TIM-3

Catalog # PVGS1560

Product Information

Primary Accession	Q8TDQ0
Species	Human
Sequence	Ser22-Arg200
Purity	> 95% as analyzed by SDS-PAGE
Endotoxin Level	
Biological Activity	Assay #1: Immobilized TIM-3, hFc, Human at 0.5 μg/ml, the concentration of Anti-TIM3 mouse antibody () that produces 50% optimal binding response is found to be approximately 5.0 ng/ml. Assay #2: Immobilized Galectin-9, His, Human at 0.5 μg/ml (100 μl/well) can bind TIM-3, hFc, Human with a linear range of 0.78-6.25 μg/ml.
Expression System	HEK 293
Formulation	Lyophilized from a 0.2 μm filtered solution in PBS.
Reconstitution	It is recommended that this vial be briefly centrifuged prior to opening to bring the contents to the bottom. Reconstitute the lyophilized powder in ddH_2O or PBS up to 100 μg/ml.
Storage & Stability	Upon receiving, this product remains stable for up to 6 months at lower than -70°C. Upon reconstitution, the product should be stable for up to 1 week at 4°C or up to 3 months at -20°C. For long term storage it is recommended that a carrier protein (example 0.1% BSA) be added. Avoid repeated freeze-thaw cycles.

Additional Information

Gene ID	84868
Other Names	Hepatitis A virus cellular receptor 2, HAVcr-2, T-cell immunoglobulin and mucin domain-containing protein 3, TIMD-3, T-cell immunoglobulin mucin receptor 3, TIM-3, T-cell membrane protein 3, CD366, HAVCR2, TIM3, TIMD3
Target Background	T cell Ig- and mucin-domain-containing molecules (TIMs) are a family of transmembrane proteins expressed by various immune cells. TIM-3 is an inhibitory molecule that is induced following T cell activation. TIM-3 is expressed by exhausted T cells in the settings of chronic infection and cancer, and tumor-infiltrating T cells that co-express PD-1 and TIM-3 exhibit the most severe exhausted phenotype. Tumor-infiltrating dendritic cells also express TIM-3. TIM-3 expression on DCs was found to suppress innate immunity by reducing the immunogenicity of nucleic acids released by dying tumor cells.

Research studies show that heterodimerization of TIM-3 with CEACAM-1 is critical for the inhibitory function of TIM-3, and co-blockade of TIM-3 and CEACAM-1 enhanced antitumor responses in a mouse model of colorectal cancer. Its binding to Galectin-9 induces a range of immunosuppressive functions which enhance immune tolerance and inhibit anti-tumor immunity. TIM-3 ligation attenuates $CD8^+$ and Th1 cell responses and promotes the activity of Treg and myeloid derived suppressor cells. In addition, dendritic cell-expressed TIM-3 dampens inflammation by enabling the phagocytosis of apoptotic cells and the cross-presentation of apoptotic cell antigens.

Protein Information

Name HAVCR2

Synonyms TIM3, TIMD3

Function Cell surface receptor implicated in modulating innate and adaptive immune responses. Generally accepted to have an inhibiting function. Reports on stimulating functions suggest that the activity may be influenced by the cellular context and/or the respective ligand (PubMed:24825777). Regulates macrophage activation (PubMed:11823861). Inhibits T-helper type 1 lymphocyte (Th1)-mediated auto- and alloimmune responses and promotes immunological tolerance (PubMed:14556005). In CD8+ cells attenuates TCR-induced signaling, specifically by blocking NF-kappaB and NFAT promoter activities resulting in the loss of IL-2 secretion. The function may implicate its association with LCK proposed to impair phosphorylation of TCR subunits, and/or LGALS9-dependent recruitment of PTPRC to the immunological synapse (PubMed:24337741, PubMed:26492563). In contrast, shown to activate TCR-induced signaling in T-cells probably implicating ZAP70, LCP2, LCK and FYN (By similarity). Expressed on Treg cells can inhibit Th17 cell responses (PubMed:24838857). Receptor for LGALS9 (PubMed:16286920, PubMed:24337741). Binding to LGALS9 is believed to result in suppression of T-cell responses; the resulting apoptosis of antigen- specific cells may implicate HAVCR2 phosphorylation and disruption of its association with BAG6. Binding to LGALS9 is proposed to be involved in innate immune response to intracellular pathogens. Expressed on Th1 cells interacts with LGALS9 expressed on Mycobacterium tuberculosis- infected macrophages to stimulate antibactericidal activity including IL-1 beta secretion and to restrict intracellular bacterial growth (By similarity). However, the function as receptor for LGALS9 has been challenged (PubMed:23555261). Also reported to enhance CD8+ T-cell responses to an acute infection such as by Listeria monocytogenes (By similarity). Receptor for phosphatidylserine (PtSer); PtSer-binding is calcium-dependent. May recognize PtSer on apoptotic cells leading to their phagocytosis. Mediates the engulfment of apoptotic cells by dendritic cells. Expressed on T-cells, promotes conjugation but not engulfment of apoptotic cells. Expressed on dendritic cells (DCs) positively regulates innate immune response and in synergy with Toll- like receptors promotes secretion of TNF. In tumor-imfiltrating DCs suppresses nucleic acid-mediated innate immune repsonse by interaction with HMGB1 and interfering with nucleic acid-sensing and trafficking of nucleid acids to endosomes (By similarity). Expressed on natural killer (NK) cells acts as a coreceptor to enhance IFN-gamma production in response to LGALS9 (PubMed:22323453). In contrast, shown to suppress NK cell-mediated cytotoxicity (PubMed:22383801). Negatively regulates NK cell function in LPS-induced endotoxic shock (By similarity).

Cellular Location Cell membrane; Single-pass type I membrane protein. Cell junction. Cell membrane. Note=Localizes to the immunological synapse between CD8+

	T-cells and target cells
Tissue Location	Expressed in T-helper type 1 (Th1) lymphocytes. Expressed on regulatory T (Treg) cells after TCR stimulation. Expressed in dendritic cells and natural killer (NK) cells. Expressed in epithelial tissues. Expression is increased on CD4+ and CD8+ T-cells in chronic hepatitis C virus (HCV) infection. In progressive HIV-1 infection, expression is up-regulated on HIV-1-specific CD8 T-cells