10320 Camino Santa Fe, Suite G
San Diego, CA 92121
Tel: 858.875.1900 Fax: 858.875.1999

TIM-3 Antibody

Catalog # ASC12111

Product Information

Application	WB, IHC-P, IF, E
Primary Accession	Q8TDQ0
Other Accession	NP_116171
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Clone Names	HAVCR2
Calculated MW	33394

Additional Information

Gene ID	84868
Alias Symbol	HAVCR2
Other Names	TIM-3 Antibody: Hepatitis A virus cellular receptor, HAVCR2, TIM-3, CD366, KIM-3, TIMD3, TIMD-3
Target/Specificity	At least two antibodies are known to exist; this antibody will detect both isoforms.
Reconstitution & Storage	TIM-3 antibody can be stored at 4˚C for three months and -20˚C, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	TIM-3 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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-infiltrating 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 and 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

Background

The immune checkpoint protein TIM-3 is a member of the immunoglobulin superfamily and TIM family of proteins that was initially identified as a specific marker of fully differentiated IFN-γ producing CD4 T helper 1 (Th1) and CD8 cytotoxic cells. It is a Th1-specific cell surface protein that regulates macrophage activation and negatively regulates Th1-mediated auto- and alloimmune responses, and is also highly expressed on regulatory T cells, monocytes, macrophages, and dendritic cells (1). TIM-3 and PD-1 are co-expressed on most CD4 and CD8 T cells infiltrating solid tumors or in hematologic malignancy in mice; blocking TIM-3 in conjugation with a PD-1 blockade increases the functionality of exhausted T cells and synergizes with to inhibit tumor growth (2,3).

References

Monney L, Sabatos CA, Gaglia JL, et al. Th1-specific cell surface protein Tim-3 regulates macrophage activation and severity of autoimmune disease. Nature 2002; 415:536-41.Sakuishi K, Apetoh L, Sullivan JM, et al. Targeting Tim-3 and PD-1 pathways to reverse T cell exhaustion and restore anti-tumor immunity. J Exp Med 2010; 207:2187–94.Zhou Q, Munger ME, Veenstra RG, et al. Coexpression of Tim-3 and PD-1 identifies a

CD8+ T-cell exhaustion phenotype in mice with disseminated acute myelogenous leukemia. Blood 2011; 117:4501–10.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.