

TIM3 Antibody [9G1]
Catalog # ASC12183**Specification****TIM3 Antibody [9G1] - Product Information**

Application	IHC-P, IF, ICC, E
Primary Accession	Q8TDQ0
Other Accession	NP_116171
Host	Mouse
Clonality	Monoclonal
Isotype	IgG2b,k

TIM3 Antibody [9G1] - Additional Information

Gene ID	84868
Alias Symbol	HAVCR2
Other Names	
TIM-3 Antibody: Hepatitis A virus cellular receptor, HAVCR2, TIM3, CD366, KIM-3, TIMD3, TIMD-3	

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

TIM3 Antibody [9G1] is for research use only and not for use in diagnostic or therapeutic procedures.

TIM3 Antibody [9G1] - 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:<http://www.uniprot.org/citations/24825777>)

TIM3 Antibody [9G1] - Background

The immune checkpoint protein TIM3 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). TIM3 and PD-1 are co-expressed on most CD4 and CD8 T cells infiltrating solid tumors or in hematologic malignancy in mice; blocking TIM3 in conjugation with a PD-1 blockade increases the functionality of exhausted T cells and synergizes with to inhibit tumor growth (2,3).

TIM3 Antibody [9G1] - 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.

target="_blank">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-alpha. In tumor-infiltrating DCs suppresses nucleic acid-mediated innate immune response by interaction with HMGB1 and interfering with nucleic acid-sensing and trafficking of nucleic 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

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

TIM3 Antibody [9G1] - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)

- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)