

TIM3

Purified Mouse Monoclonal Antibody Catalog # AO2703a

Specification

TIM3 - Product Information

Application E, WB, ICC
Primary Accession Q8TDQ0
Reactivity Human
Host Mouse
Clonality Monoclonal
Isotype Mouse IgG1
Calculated MW 33.4kDa KDa

Immunogen

Purified recombinant fragment of human TIM3 (AA: extra 22-202) expressed in HEK293 cells.

Formulation

Purified antibody in PBS with 0.05% sodium azide

TIM3 - Additional Information

Gene ID 84868

Other Names

HAVCR2; CD366; KIM-3; TIMD3; Tim-3; TIMD-3; HAVcr-2

Dilution

E~~ 1/10000

WB~~ 1/500 - 1/2000 ICC~~ 1/200 - 1/1000

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

TIM3 is for research use only and not for use in diagnostic or therapeutic procedures.

TIM3 - 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



Tel: 858.875.1900 Fax: 858.875.1999

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-alpha. 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

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 - Protocols

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

Western Blot



- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

TIM3 - Images

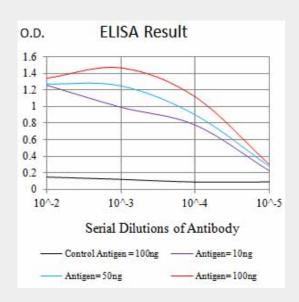


Figure 1:Black line: Control Antigen (100 ng); Purple line: Antigen (10ng); Blue line: Antigen (50 ng); Red line:Antigen (100 ng)

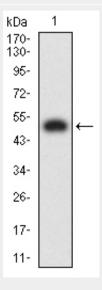


Figure 2:Western blot analysis using TIM3 mAb against human TIM3 (AA: extra 22-202) recombinant protein. (Expected MW is 50 kDa)



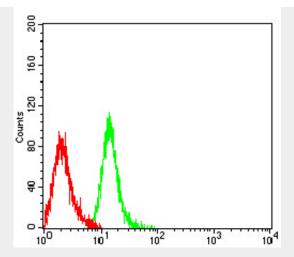


Figure 4:Flow cytometric analysis of Jurkat cells using TIM3 mouse mAb (green) and negative control (red).

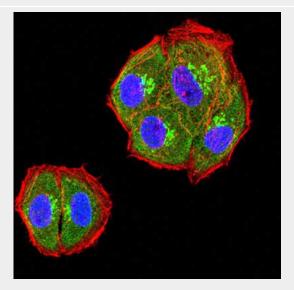


Figure 3:Immunofluorescence analysis of Hela cells using TIM3 mouse mAb (green). Blue: DRAQ5 fluorescent DNA dye. Red: Actin filaments have been labeled with Alexa Fluor- 555 phalloidin. Secondary antibody from Fisher (Cat#: 35503)

TIM3 - References

1.Tumour Biol. 2016 Jun;37(6):8209-18.2.Int Immunopharmacol. 2015 Dec;29(2):635-641.