

HAVCR2 Antibody

Purified Mouse Monoclonal Antibody (Mab)

Catalog # AM8632b

Product Information

Application	WB, FC, E
Primary Accession	Q8TDQ0
Reactivity	Human
Host	Mouse
Clonality	monoclonal
Isotype	IgG2b,k
Clone Names	1827CT151.74.68
Calculated MW	33394

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, HAVCR2, TIM3, TIMD3
Target/Specificity	This HAVCR2 antibody is generated from a mouse immunized with a recombinant protein of human HAVCR2.
Dilution	WB~~1:4000 FC~~1:25 E~~Use at an assay dependent concentration.
Format	Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.
Storage	Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.
Precautions	HAVCR2 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-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

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

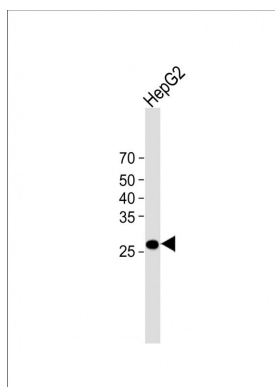
Regulates macrophage activation. Inhibits T-helper type 1 lymphocyte (Th1)-mediated auto- and alloimmune responses and promotes immunological tolerance. May be also involved in T-cell homing. Receptor for LGALS9.

References

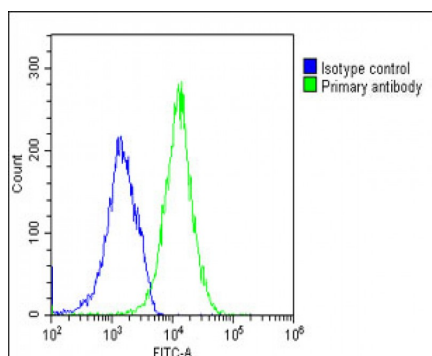
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Zhang W.,et al.Submitted (APR-2000) to the EMBL/GenBank/DBJ databases.

Kuehn E.W.,et al.Submitted (DEC-2001) to the EMBL/GenBank/DDBJ databases.
Ota T.,et al.Nat. Genet. 36:40-45(2004).
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Images



All lanes: Anti-HAVCR2 Antibody at 1:1000 dilution + HepG2 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary: Goat Anti-Mouse IgG, (H+L), Peroxidase conjugated (ASP1613) at 1/8000 dilution. Observed band size: 28 KDa Blocking/Dilution buffer: 5% NFDM/TBST.



Overlay histogram showing U-2 OS cells stained with AM8632b(green line). The cells were fixed with 2% paraformaldehyde (10 min) and then permeabilized with 90% methanol for 10 min. The cells were then incubated in 2% bovine serum albumin to block non-specific protein-protein interactions followed by the antibody (AM8632b, 1:25 dilution) for 60 min at 37°C. The secondary antibody used was Goat-Anti-Mouse IgG, DyLight® 488 Conjugated Highly Cross-Adsorbed(NH174309) at 1/200 dilution for 40 min at 37°C. Isotype control antibody (blue line) was mouse IgG2b (1µg/1x10⁶ cells) used under the same conditions. Acquisition of >10, 000 events was performed.

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