

TREM1 Antibody

Catalog # ASC11600

Specification

TREM1 Antibody - Product Information

Application Primary Accession Other Accession Reactivity Host Clonality Isotype Calculated MW Application Notes

WB, E <u>O9NP99</u> <u>NP_061113</u>, <u>8924262</u> Human Rabbit Polyclonal IgG Predicted: 26 kDa KDa TREM1 antibody can be used for detection of TREM1 by Western blot at 1 - 2 μg/mL.

TREM1 Antibody - Additional Information

Gene ID Target/Specificity TREM1; 54210

Reconstitution & Storage

Antibody can be stored at 4°C up to one year. Antibodies should not be exposed to prolonged high temperatures.

Precautions TREM1 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

TREM1 Antibody - Protein Information

Name TREM1

Function

[Isoform 1]: Cell surface receptor that plays important roles in innate and adaptive immunity by amplifying inflammatory responses (PubMed:10799849, PubMed:21393102). Upon activation by various ligands such as PGLYRP1, HMGB1 or HSP70, multimerizes and forms a complex with transmembrane adapter TYROBP/DAP12 (PubMed:25595774, PubMed:25595774, PubMed:25595774, PubMed:29568119). In turn, initiates a SYK-mediated cascade of tyrosine phosphorylation, activating multiple downstream mediators such as BTK, MAPK1, MAPK3 or phospholipase C-gamma (PubMed:21659545). This cascade promotes the neutrophil- and macrophage-mediated release of pro-inflammatory cytokines and/or chemokines, as well as their migration and thereby amplifies inflammatory responses that are



triggered by bacterial and fungal infections (PubMed: 17098818, PubMed:17568691). By also promoting the amplification of inflammatory signals that are initially triggered by Toll-like receptor (TLR) and NOD-like receptor engagement, plays a major role in the pathophysiology of acute and chronic inflammatory diseases of different etiologies including septic shock and atherosclerosis (PubMed:11323674, PubMed:21393102).

Cellular Location

[Isoform 1]: Cell membrane; Single-pass type I membrane protein. Note=Recruited to lipid rafts when activated.

Tissue Location

Mostly expressed by immune cells of the myeloid lineage, such as monocytes, macrophages, neutrophils and dendritic cells (PubMed:10799849). Expression is associated with a mature stage of myeloid development (PubMed:11922939). Highly expressed in adult liver, lung and spleen than in corresponding fetal tissue. Also expressed in the lymph node, placenta, spinal cord and heart tissues lsoform 2 was detected in the lung, liver and mature monocytes

TREM1 Antibody - Protocols

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

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

TREM1 Antibody - Images



Western blot analysis of TREM1 in 293 cell lysate with TREM1 antibody at (A) 1 and (B) 2 μ g/mL.

TREM1 Antibody - Background



TREM1 Antibody: TREM1 (triggering receptor expressed on myeloid cells 1) is a receptor belonging to the Ig superfamily that amplifies neutrophil and monocyte-mediated inflammatory responses triggered by bacterial and fungal infections by stimulating release of pro-inflammatory chemokines and cytokines, as well as increased surface expression of cell activation markers. It is thought that TREM1 other related TREM proteins such as TREM2 act to modulate the inflammatory response. TREM1 has also been linked to inflammatory bowel disease resulting in enhanced production of TNF, IL-6, IL-8, and MCP-1.

TREM1 Antibody - References

Bouchon A, Facchetti F, Weigand MA, et al. TREM-1 amplifies inflammation and is a crucial mediator of septic shock. Nature 2001; 410:1103-7.

Ford JW and McVicar DW. TREM and TREM-like receptors in inflammation and disease. Curr. Opin. Immunol. 2009; 21:38-46

Schenk M, Bouchon A, Seibold F, et al. TREM-1-expressing intestinal macrophages crucially amplify chronic inflammation in experimental colitis and inflammatory bowel diseases. J. Clin. Invest. 2007; 117:3097-106.