

MR1 Antibody (C-term)
Affinity Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP17332B**Specification**

MR1 Antibody (C-term) - Product Information

Application	WB,E
Primary Accession	O95460
Other Accession	NP_001181929.1 , NP_001181928.1
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Antigen Region	312-341

MR1 Antibody (C-term) - Additional Information**Gene ID** 3140**Other Names**

Major histocompatibility complex class I-related gene protein, MHC class I-related gene protein, Class I histocompatibility antigen-like protein, MR1

Target/Specificity

This MR1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 312-341 amino acids from the C-terminal region of human MR1.

Dilution

WB~~1:1000

Format

Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

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

MR1 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

MR1 Antibody (C-term) - Protein Information**Name** MR1

Function Antigen-presenting molecule specialized in displaying microbial pyrimidine-based metabolites to alpha-beta T cell receptors (TCR) on innate-type mucosal-associated invariant T

(MAIT) cells (PubMed:[23051753](#), PubMed:[26795251](#), PubMed:[12794138](#), PubMed:[19416870](#), PubMed:[22692454](#), PubMed:[23846752](#)). In complex with B2M preferentially presents riboflavin-derived metabolites to semi-invariant TRAV1-2 TCRs on MAIT cells, guiding immune surveillance of the microbial metabolome at mucosal epithelial barriers (PubMed:[26795251](#), PubMed:[24695216](#), PubMed:[20581831](#)). Signature pyrimidine-based microbial antigens are generated via non-enzymatic condensation of metabolite intermediates of the riboflavin pathway with by-products arising from other metabolic pathways such as glycolysis. Typical potent antigenic metabolites are 5-(2-oxoethylideneamino)-6-D-ribitylaminouracil (5-OE-RU) and 5-(2-oxopropylideneamino)-6-D-ribitylaminouracil (5-OP-RU), products of condensation of 5-amino-6-D-ribitylaminouracil (5-A-RU) with glyoxal or methylglyoxal by-products, respectively (PubMed:[24695216](#)). May present microbial antigens to various TRAV1-2-negative MAIT cell subsets, providing for unique recognition of diverse microbes, including pathogens that do not synthesize riboflavin (PubMed:[27527800](#), PubMed:[31113973](#)). Upon antigen recognition, elicits rapid innate-type MAIT cell activation to eliminate pathogenic microbes by directly killing infected cells (PubMed:[24695216](#), PubMed:[27527800](#), PubMed:[23846752](#)). During T cell development, drives thymic selection and post-thymic terminal differentiation of MAIT cells in a process dependent on commensal microflora (By similarity). Acts as an immune sensor of cancer cell metabolome (PubMed:[31959982](#)). May present a tumor-specific or -associated metabolite essential for cancer cell survival to a pan-cancer TCR consisting of TRAV38.2-DV8*TRAJ31 alpha chain paired with a TRBV25.1*TRBJ2.3 beta chain on a non-MAIT CD8- positive T cell clone (MC.7.G5), triggering T cell-mediated killing of a wide range of cancer cell types (PubMed:[31959982](#)).

Cellular Location

Cell membrane; Single-pass type I membrane protein. Endoplasmic reticulum membrane; Single-pass type I membrane protein. Golgi apparatus membrane; Single-pass type I membrane protein. Early endosome membrane; Single-pass type I membrane protein. Late endosome membrane; Single-pass type I membrane protein. Note=In the absence of antigen remains within the endoplasmic reticulum where it acts as a metabolite sensor. Antigen binding triggers trafficking of the ternary complex to the plasma membrane. After presentation, most of these complexes are rapidly internalized and degraded via endocytosis. A small subset recycles via endosomes back to the plasma membrane and may thus acquire and present new antigens that do not efficiently reach the endoplasmic reticulum. [Isoform 3]: Cell membrane; Single-pass type I membrane protein. Endoplasmic reticulum membrane; Single-pass membrane protein. Note=The larger proportion remains in the ER in an immature state. The subset that reach cell surface does it through a B2M-independent pathway.

Tissue Location

Ubiquitous (PubMed:7624800, PubMed:9780177). Low expression is detected in peripheral blood B cells, T cells, monocytes and in bronchial epithelial cells (at protein level) (PubMed:27043408) Expressed in plasmablasts or plasma B cells in the lamina propria of ileum, appendix and colon (at protein level) (PubMed:19760593). Highly expressed on a subset of CD45-positive CD3-positive thymocytes (at protein level) (PubMed:22692454).

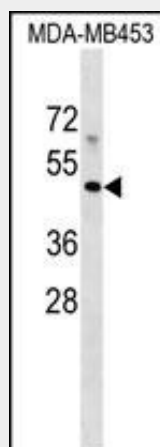
MR1 Antibody (C-term) - Protocols

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](#)

MR1 Antibody (C-term) - Images



MR1 Antibody (C-term) (Cat. #AP17332b) western blot analysis in MDA-MB453 cell line lysates (35ug/lane). This demonstrates the MR1 antibody detected the MR1 protein (arrow).

MR1 Antibody (C-term) - Background

MR1 has antigen presentation function. Involved in the development and expansion of a small population of T cells expressing an invariant T cell receptor alpha chain called mucosal-associated invariant T cells (MAIT). MAIT cells are preferentially located in the gut lamina propria and therefore may be involved in monitoring commensal flora or serve as a distress signal. Expression and MAIT cell recognition seem to be ligand-dependent.

MR1 Antibody (C-term) - References

Gozalbo-Lopez, B., et al. *Histol. Histopathol.* 24(11):1439-1449(2009)
Stumpf, A.N., et al. *Blood* 114(17):3684-3692(2009)
Huang, S., et al. *Proc. Natl. Acad. Sci. U.S.A.* 106(20):8290-8295(2009)
Aldemir, H. *Biochem. Biophys. Res. Commun.* 366(2):328-334(2008)
Miley, M.J., et al. *J. Immunol.* 170(12):6090-6098(2003)