

CARD9 Antibody (N-term)

Purified Mouse Monoclonal Antibody (Mab)
Catalog # AM8722b

Specification

CARD9 Antibody (N-term) - Product Information

WB.E Application **Primary Accession** O9H257 Reactivity Human Predicted Human Host Mouse Clonality monoclonal Isotype IgG1, ĸ Calculated MW 62241

CARD9 Antibody (N-term) - Additional Information

Gene ID 64170

Other Names

Caspase recruitment domain-containing protein 9, hCARD9, CARD9

Target/Specificity

This CARD9 antibody is generated from a mouse immunized with a recombinant protein from human CARD9.

Dilution

WB~~1:500

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

CARD9 Antibody (N-term) is for research use only and not for use in diagnostic or therapeutic procedures.

CARD9 Antibody (N-term) - Protein Information

Name CARD9 {ECO:0000303|PubMed:11053425, ECO:0000312|HGNC:HGNC:16391}

Function Adapter protein that plays a key role in innate immune response against fungi by forming signaling complexes downstream of C- type lectin receptors (PubMed: 26961233,



PubMed:33558980). CARD9- mediated signals are essential for antifungal immunity against a subset of fungi from the phylum Ascomycota (PubMed:24231284, PubMed:25057046, PubMed:25702837, PubMed:26521038, PubMed:26679537, PubMed:26961233, PubMed:27777981, PubMed:29080677, PubMed:33558980). Transduces signals in myeloid cells downstream of C-type lectin receptors CLEC7A (dectin- 1), CLEC6A (dectin-2) and CLEC4E (Mincl which detect pathogen- associated molecular pattern metabolites (PAMPs), such as fungal carbohydrates, and trigger CARD9 activation (By similarity). Upon activation, CARD9

downstream of C-type lectin receptors CLEC7A (dectin-1), CLEC6A (dectin-2) and CLEC4E (Mincle), carbohydrates, and trigger CARD9 activation (By similarity). Upon activation, CARD9 homooligomerizes to form a nucleating helical template that recruits BCL10 via CARD-CARD interaction, thereby promoting polymerization of BCL10 and subsequent recruitment of MALT1: this leads to activation of NF-kappa-B and MAP kinase p38 (MAPK11, MAPK12, MAPK13 and/or MAPK14) pathways which stimulate expression of genes encoding pro-inflammatory cytokines and chemokines (PubMed:11053425, PubMed:26488816, PubMed:26961233, PubMed:31296852, PubMed: 33558980). CARD9 signaling in antigen-presenting cells links innate sensing of fungi to the activation of adaptive immunity and provides a cytokine milieu that induces the development and subsequent of interleukin 17-producing T helper (Th17) cells (PubMed: 24231284). Also involved in activation of myeloid cells via classical ITAM- associated receptors and TLR: required for TLR-mediated activation of MAPK, while it is not required for TLR-induced activation of NF-kappa-B (By similarity). CARD9 can also be engaged independently of BCL10: forms a complex with RASGRF1 downstream of C-type lectin receptors, which recruits and activates HRAS, leading to ERK activation and the production of cytokines (By similarity). Acts as an important regulator of the intestinal commensal fungi (mycobiota) component of the gut microbiota (PubMed: 33548172). Plays an essential role in antifungal immunity against dissemination of gut fungi: acts by promoting induction of antifungal IgG antibodies response in CX3CR1(+) macrophages to confer protection against disseminated C.albicans or C.auris infection (PubMed: 33548172). Also mediates immunity against other pathogens, such as certain bacteria, viruses and parasites; CARD9 signaling is however redundant with other innate immune responses (By similarity). In response to L.monocytogenes infection, required for the production of inflammatory cytokines activated by intracellular peptidoglycan: acts by connecting NOD2 recognition of peptidoglycan to downstream activation of MAP kinases (MAPK) without activating NF- kappa-B (By similarity).

Cellular Location Cytoplasm

Tissue Location

Expression is restricted to several populations of phagocytes, such as macrophages, monocytes, and dendritic cells (PubMed:33548172). Highly expressed in spleen (PubMed:11053425). Also detected in liver, placenta, lung, peripheral blood leukocytes and in brain (PubMed:11053425).

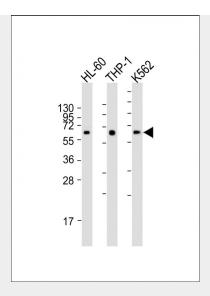
CARD9 Antibody (N-term) - Protocols

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

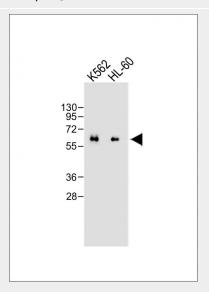
- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

CARD9 Antibody (N-term) - Images





All lanes: Anti-CARD9 Antibody (N-term) at dilution Lane 1: HL-60 whole cell lysate Lane 2: THP-1 whole cell lysate Lane 3: K562 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size: 62 kDa Blocking/Dilution buffer: 5% NFDM/TBST.



All lanes : Anti-CARD9 Antibody (N-term) at dilution Lane 1: K562 whole cell lysate Lane 2: HL-60 whole cell lysate Lysates/proteins at 20 μ g per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 62 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

CARD9 Antibody (N-term) - Background

Adapter protein that plays a key role in innate immune response to a number of intracellular pathogens, such as C.albicans and L.monocytogenes. Is at the crossroads of ITAM- tyrosine kinase and the Toll-like receptors (TLR) and NOD2 signaling pathways. Probably controls various innate immune response pathways depending on the intracellular pathogen. In response to L.monocytogenes infection, acts by connecting NOD2 recognition of peptidoglycan to downstream activation of MAP kinases (MAPK) without activating NF-kappa-B. Also involved in activation of myeloid cells via classical ITAM-associated receptors and TLR: required for TLR-mediated activation of MAPK, while it is not required for TLR-induced activation of NF-kappa-B (By similarity). Controls CLEC7A (dectin-1)-mediated myeloid cell activation induced by the yeast cell wall component zymosan, leading to cytokine production and innate anti-fungal immunity: acts by regulating





BCL10-MALT1-mediated NF-kappa-B activation pathway. Activates NF-kappa-B via BCL10. In response to the hyphal form of C.albicans, mediates CLEC6A (dectin-2)-induced I-kappa-B kinase ubiquitination, leading to NF-kappa-B activation via interaction with BCL10. In response to fungal infection, may be required for the development and subsequent differentiation of interleukin 17-producing T helper (TH-17) cells.

CARD9 Antibody (N-term) - References

Bertin J.,et al.J. Biol. Chem. 275:41082-41086(2000). Ota T.,et al.Nat. Genet. 36:40-45(2004). Humphray S.J.,et al.Nature 429:369-374(2004). Mural R.J.,et al.Submitted (JUL-2005) to the EMBL/GenBank/DDBJ databases. Zahedi R.P.,et al.J. Proteome Res. 7:526-534(2008).