

RAIDD Antibody
Catalog # ASC10009

Specification

RAIDD Antibody - Product Information

Application	IHC
Primary Accession	P78560
Other Accession	AAB42217 , 8738
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	22 kDa KDa
Application Notes	RAIDD antibody can be used for detection of RAIDD by Western blot at 0.5 - 1 µg/mL. A 22 kDa band should be detected. Antibody can also be used for immunocytochemistry starting at 5 µg/mL. For immunofluorescence start at 20 µg/mL.

RAIDD Antibody - Additional Information

Gene ID **8738**

Other Names

RAIDD Antibody: MRT34, RAIDD, Death domain-containing protein CRADD, Caspase and RIP adapter with death domain, CASP2 and RIPK1 domain containing adaptor with death domain

Target/Specificity

RAIDD antibody was raised against a 19 amino acid peptide near the center of human RAIDD. The immunogen is located within amino acids 90 - 140 of RAIDD.

Reconstitution & Storage

RAIDD antibody can be stored at 4°C for three months and -20°C, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.

Precautions

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

RAIDD Antibody - Protein Information

Name CRADD

Synonyms RAIDD

Function

Adapter protein that associates with PIDD1 and the caspase CASP2 to form the PIDDosome, a complex that activates CASP2 and triggers apoptosis (PubMed:<a

[15073321](http://www.uniprot.org/citations/15073321), PubMed:<[16652156](http://www.uniprot.org/citations/16652156)>, PubMed:<[17159900](http://www.uniprot.org/citations/17159900)>, PubMed:<[17289572](http://www.uniprot.org/citations/17289572)>, PubMed:<[9044836](http://www.uniprot.org/citations/9044836)>). Also recruits CASP2 to the TNFR-1 signaling complex through its interaction with RIPK1 and TRADD and may play a role in the tumor necrosis factor-mediated signaling pathway (PubMed:<[8985253](http://www.uniprot.org/citations/8985253)>).

Cellular Location

Cytoplasm {ECO:0000250|UniProtKB:O88843}. Nucleus {ECO:0000250|UniProtKB:O88843}

Tissue Location

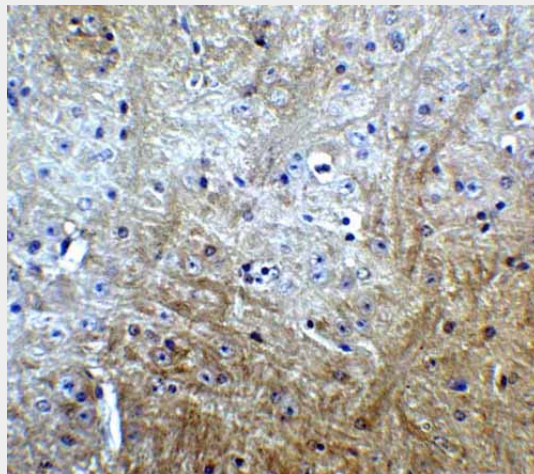
Constitutively expressed in most tissues, with particularly high expression in adult heart, testis, liver, skeletal muscle, fetal liver and kidney.

RAIDD Antibody - 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](#)

RAIDD Antibody - Images



Immunohistochemistry of Slitrk1 in mouse brain tissue with Slitrk1 Antibody at 5 µg/mL.

RAIDD Antibody - Background

RAIDD Antibody: Apoptosis, or programmed cell death, occurs during normal cellular differentiation and development of multicellular organisms. Apoptosis is induced by certain cytokines including TNF and Fas ligand of the TNF family through their death domain (DD)-containing receptors, TNFR1 and Fas. The death signals are transduced by a group of DD-containing adapter molecules. A novel

cell death adapter was recently identified by two independent groups and designated RAIDD (RIP-associated ICH-1/CED-3-homologous protein with DD) and CRADD (caspase and RIP adapter with DD)¹, RAIDD contains a DD and a CARD (for caspase recruitment domain) which interact with RIP and caspase, respectively, to transduce death signals^{1, 3}. RAIDD is constitutively expressed in many tissues and mediates apoptosis caused by Fas and TNFR-1.

RAIDD Antibody - References

Duan H, Dixit VM. RAIDD is a new 'death' adaptor molecule. *Nature* 1997;385:86-89
Ahmad M, Srinivasula SM, Wang L, Talanian RV, Litwack G, Fernandes-Alnemri T, Alnemri ES. CRADD, a novel human apoptotic adaptor molecule for caspase-2, and FasL/tumor necrosis factor receptor-interacting protein RIP. *Cancer Res* 1997 57:615-619
Hofmann K, Bucher P, Tschopp J. The CARD domain: a new apoptotic signalling motif. *Trends Biochem Sci* 1997;22:155-156 (RD1299)