

**Caspase-8 Antibody**  
**Catalog # ASC10303****Specification**

---

**Caspase-8 Antibody - Product Information**

Application	IHC
Primary Accession	<a href="#">Q14790</a>
Other Accession	<a href="#">NP_001219</a> , <a href="#">841</a>
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	55 kDa KDa
Application Notes	Caspase-8 antibody can be used for the detection of caspase-8 by Western blot at 0.5 to 2 µg/mL. Antibody can also be used for immunocytochemistry starting at 2 µg/mL and immunohistochemistry starting at 5 µg/mL. For immunofluorescence start at 20 µg/mL.

**Caspase-8 Antibody - Additional Information**Gene ID **841****Other Names**

Caspase-8 Antibody: CAP4, MACH, MCH5, FLICE, ALPS2B, Casp-8, Caspase-8, Apoptotic cysteine protease, CASP-8, caspase 8, apoptosis-related cysteine peptidase

**Target/Specificity**

Caspase-8 antibody was raised against a 16 amino acid synthetic peptide from near the carboxy terminus of human Caspase-8 isoform A. <br><br>The immunogen is located within the last 50 amino acids of Caspase-8.

**Reconstitution & Storage**

Caspase-8 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**

Caspase-8 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

**Caspase-8 Antibody - Protein Information**

**Name** CASP8 {ECO:0000303|PubMed:9931493, ECO:0000312|HGNC:HGNC:1509}

**Function**

Thiol protease that plays a key role in programmed cell death by acting as a molecular switch for

apoptosis, necroptosis and pyroptosis, and is required to prevent tissue damage during embryonic development and adulthood (PubMed: [>23516580</a>, PubMed: \[>35338844</a>, PubMed: \\[>35446120</a>, PubMed: \\\[>8681376</a>, PubMed: \\\\[>8681377</a>, PubMed: \\\\\[>8962078</a>, PubMed: \\\\\\[>9006941</a>, PubMed: \\\\\\\[>9184224</a>\\\\\\\\). Initiator protease that induces extrinsic apoptosis by mediating cleavage and activation of effector caspases responsible for FAS/CD95-mediated and TNFRSF1A-induced cell death \\\\\\\\(PubMed: \\\\\\\\[>23516580</a>, PubMed: \\\\\\\\\[>35338844</a>, PubMed: \\\\\\\\\\[>35446120</a>, PubMed: \\\\\\\\\\\[>8681376</a>, PubMed: \\\\\\\\\\\\[>8681377</a>, PubMed: \\\\\\\\\\\\\[>8962078</a>, PubMed: \\\\\\\\\\\\\\[>9006941</a>, PubMed: \\\\\\\\\\\\\\\[>9184224</a>\\\\\\\\\\\\\\\\). Cleaves and activates effector caspases CASP3, CASP4, CASP6, CASP7, CASP9 and CASP10 \\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\[>16916640</a>, PubMed: \\\\\\\\\\\\\\\\\[>8962078</a>, PubMed: \\\\\\\\\\\\\\\\\\[>9006941</a>\\\\\\\\\\\\\\\\\\\). Binding to the adapter molecule FADD recruits it to either receptor FAS/TNFRSF6 or TNFRSF1A \\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\[>8681376</a>, PubMed: \\\\\\\\\\\\\\\\\\\\[>8681377</a>\\\\\\\\\\\\\\\\\\\\\). The resulting aggregate called the death-inducing signaling complex \\\\\\\\\\\\\\\\\\\\\(DISC\\\\\\\\\\\\\\\\\\\\\) performs CASP8 proteolytic activation \\\\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\\\[>9184224</a>\\\\\\\\\\\\\\\\\\\\\\). The active dimeric enzyme is then liberated from the DISC and free to activate downstream apoptotic proteases \\\\\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\\\\[>9184224</a>\\\\\\\\\\\\\\\\\\\\\\\). Proteolytic fragments of the N-terminal propeptide \\\\\\\\\\\\\\\\\\\\\\\(termed CAP3, CAP5 and CAP6\\\\\\\\\\\\\\\\\\\\\\\) are likely retained in the DISC \\\\\\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\\\\\[>9184224</a>\\\\\\\\\\\\\\\\\\\\\\\\). In addition to extrinsic apoptosis, also acts as a negative regulator of necroptosis: acts by cleaving RIPK1 at 'Asp-324', which is crucial to inhibit RIPK1 kinase activity, limiting TNF-induced apoptosis, necroptosis and inflammatory response \\\\\\\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\\\\\\[>31827280</a>, PubMed: \\\\\\\\\\\\\\\\\\\\\\\\\[>31827281</a>\\\\\\\\\\\\\\\\\\\\\\\\\\). Also able to initiate pyroptosis by mediating cleavage and activation of gasdermin-C and -D \\\\\\\\\\\\\\\\\\\\\\\\\\(GSDMC and GSDMD, respectively\\\\\\\\\\\\\\\\\\\\\\\\\\): gasdermin cleavage promotes release of the N-terminal moiety that binds to membranes and forms pores, triggering pyroptosis \\\\\\\\\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\\\\\\\\[>32929201</a>, PubMed: \\\\\\\\\\\\\\\\\\\\\\\\\\\[>34012073</a>\\\\\\\\\\\\\\\\\\\\\\\\\\\\). Initiates pyroptosis following inactivation of MAP3K7/TAK1 \\\\\\\\\\\\\\\\\\\\\\\\\\\\(By similarity\\\\\\\\\\\\\\\\\\\\\\\\\\\\). Also acts as a regulator of innate immunity by mediating cleavage and inactivation of N4BP1 downstream of TLR3 or TLR4, thereby promoting cytokine production \\\\\\\\\\\\\\\\\\\\\\\\\\\\(By similarity\\\\\\\\\\\\\\\\\\\\\\\\\\\\). May participate in the Granzyme B \\\\\\\\\\\\\\\\\\\\\\\\\\\\(GZMB\\\\\\\\\\\\\\\\\\\\\\\\\\\\) cell death pathways \\\\\\\\\\\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\\\\\\\\\\[>8755496</a>\\\\\\\\\\\\\\\\\\\\\\\\\\\\\). Cleaves PARP1 and PARP2 \\\\\\\\\\\\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\\\\\\\\\\\[>8681376</a>\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\). Independent of its protease activity, promotes cell migration following phosphorylation at Tyr-380 \\\\\\\\\\\\\\\\\\\\\\\\\\\\\\(PubMed: \\\\\\\\\\\\\\\\\\\\\\\\\\\\\\[>18216014</a>, PubMed: \\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\[>27109099</a>\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\).\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/27109099\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/18216014\\\\\\\\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/8681376\\\\\\\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/8755496\\\\\\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/34012073\\\\\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/32929201\\\\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/31827281\\\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/31827280\\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/9184224\\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/9184224\\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/9184224\\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/8681377\\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/8681376\\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/9006941\\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/8962078\\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/16916640\\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\\]\\\\\\\\\\\\\\\(http://www.uniprot.org/citations/9184224\\\\\\\\\\\\\\\)\\\\\\\\\\\\\\]\\\\\\\\\\\\\\(http://www.uniprot.org/citations/9006941\\\\\\\\\\\\\\)\\\\\\\\\\\\\]\\\\\\\\\\\\\(http://www.uniprot.org/citations/8962078\\\\\\\\\\\\\)\\\\\\\\\\\\]\\\\\\\\\\\\(http://www.uniprot.org/citations/8681377\\\\\\\\\\\\)\\\\\\\\\\\]\\\\\\\\\\\(http://www.uniprot.org/citations/8681376\\\\\\\\\\\)\\\\\\\\\\]\\\\\\\\\\(http://www.uniprot.org/citations/35446120\\\\\\\\\\)\\\\\\\\\]\\\\\\\\\(http://www.uniprot.org/citations/35338844\\\\\\\\\)\\\\\\\\]\\\\\\\\(http://www.uniprot.org/citations/23516580\\\\\\\\)\\\\\\\]\\\\\\\(http://www.uniprot.org/citations/9184224\\\\\\\)\\\\\\]\\\\\\(http://www.uniprot.org/citations/9006941\\\\\\)\\\\\]\\\\\(http://www.uniprot.org/citations/8962078\\\\\)\\\\]\\\\(http://www.uniprot.org/citations/8681377\\\\)\\\]\\\(http://www.uniprot.org/citations/8681376\\\)\\]\\(http://www.uniprot.org/citations/35446120\\)\]\(http://www.uniprot.org/citations/35338844\)](http://www.uniprot.org/citations/23516580)

### Cellular Location

Cytoplasm {ECO:0000250|UniProtKB:Q9JHX4}. Nucleus {ECO:0000250|UniProtKB:Q9JHX4}. Cell projection, lamellipodium. Note=Recruitment to lamellipodia of migrating cells is enhanced by

phosphorylation at Tyr-380

#### **Tissue Location**

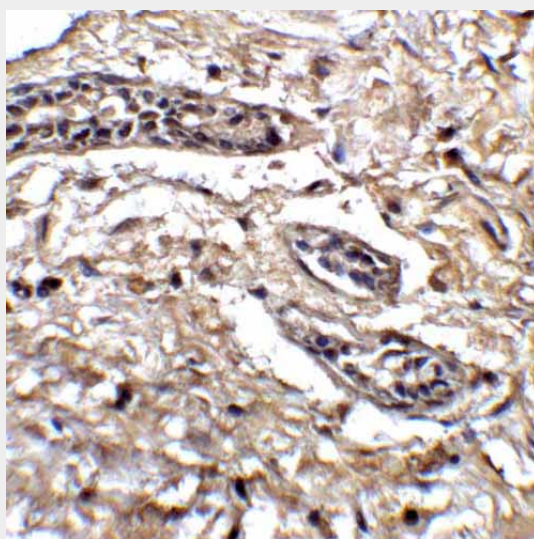
Isoform 1, isoform 5 and isoform 7 are expressed in a wide variety of tissues. Highest expression in peripheral blood leukocytes, spleen, thymus and liver. Barely detectable in brain, testis and skeletal muscle

#### **Caspase-8 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](#)

#### **Caspase-8 Antibody - Images**



Immunohistochemistry of Bcl9L in human breast tissue with Bcl9L antibody at 5 µg/ml.

#### **Caspase-8 Antibody - Background**

**Caspase-8 Antibody:** Caspases are a family of cysteine proteases that can be divided into the apoptotic and inflammatory caspase subfamilies. Unlike the apoptotic caspases, members of the inflammatory subfamily are generally not involved in cell death but are associated with the immune response to microbial pathogens. The apoptotic subfamily can be further divided into initiator caspases, which are activated in response to death signals, and executioner caspases, which are activated by the initiator caspases and are responsible for cleavage of cellular substrates that ultimately lead to cell death. Caspase-8 is an initiator caspase that was identified as a member of the Fas/APO-1 death-inducing signaling complex. The adaptor molecule FADD couples procaspase-8 to the Fas receptor death domain; subsequent oligomerization promotes procaspase-8 autoactivation. FLIP, a catalytically inactive caspase-8-like molecule inhibits these interactions and thus can inhibit apoptosis.

## **Caspase-8 Antibody - References**

Martinon F and Tschopp J. Inflammatory caspases: linking an intracellular innate immune system to autoinflammatory diseases. *Cell* 2004; 117:561-74.

Zhivotovsky B and Orrenius S. Caspase-2 function in response to DNA damage. *Biochim. Biophys. Res. Comm.* 2005; 331:859-67.

Wolf BB and Green DR. Suicidal tendencies: apoptotic cell death by caspase family proteinases. *J. Biol. Chem.* 1999; 274:20049-52.

Muzio M, Chinnaiyan AM, Kischkel FC, et al. FLICE, a novel FADD-homologous ICE/CED-3-like protease, is recruited to the CD95 (Fas/APO-1) death-inducing signaling complex. *Cell* 1996; 85:817-27.