

CASP8 / Caspase 8 Antibody
Rabbit Polyclonal Antibody
Catalog # ALS12970**Specification**

CASP8 / Caspase 8 Antibody - Product Information

Application	WB, IHC-P
Primary Accession	Q14790
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	55kDa KDa
Dilution	WB~~1:1000 IHC-P~~N/A

CASP8 / Caspase 8 Antibody - Additional Information**Gene ID** 841**Other Names**

Caspase-8, CASP-8, 3.4.22.61, Apoptotic cysteine protease, Apoptotic protease Mch-5, CAP4, FADD-homologous ICE/ced-3-like protease, FADD-like ICE, FLICE, ICE-like apoptotic protease 5, MORT1-associated ced-3 homolog, MACH, Caspase-8 subunit p18, Caspase-8 subunit p10, CASP8, MCH5

Target/Specificity

15 amino acid peptide from near the middle of human caspase-8 isoform E

Reconstitution & Storage

Short term 4°C, long term aliquot and store at -20°C, avoid freeze thaw cycles. Store undiluted.

Precautions

CASP8 / Caspase 8 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

CASP8 / 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](http://www.uniprot.org/citations/23516580) target="_blank">23516580, PubMed: [35338844](http://www.uniprot.org/citations/35338844) target="_blank">35338844, PubMed: [35446120](http://www.uniprot.org/citations/35446120) target="_blank">35446120, PubMed: [8681376](http://www.uniprot.org/citations/8681376) target="_blank">8681376, PubMed: [8681377](http://www.uniprot.org/citations/8681377) target="_blank">8681377, PubMed: [8962078](http://www.uniprot.org/citations/8962078) target="_blank">8962078)

target="_blank">8962078, PubMed:9006941, PubMed:9184224). 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, PubMed:35338844, PubMed:35446120, PubMed:8681376, PubMed:8681377, PubMed:8962078, PubMed:9006941, PubMed:9184224). Cleaves and activates effector caspases CASP3, CASP4, CASP6, CASP7, CASP9 and CASP10 (PubMed:16916640, PubMed:8962078, PubMed:9006941). Binding to the adapter molecule FADD recruits it to either receptor FAS/TNFRSF6 or TNFRSF1A (PubMed:8681376, PubMed:8681377). The resulting aggregate called the death-inducing signaling complex (DISC) performs CASP8 proteolytic activation (PubMed:9184224). The active dimeric enzyme is then liberated from the DISC and free to activate downstream apoptotic proteases (PubMed:9184224). Proteolytic fragments of the N-terminal propeptide (termed CAP3, CAP5 and CAP6) are likely retained in the DISC (PubMed:9184224). 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, PubMed:31827281). 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, PubMed:34012073). 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). Cleaves PARP1 and PARP2 (PubMed:8681376). Independent of its protease activity, promotes cell migration following phosphorylation at Tyr-380 (PubMed:18216014, PubMed:27109099).

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

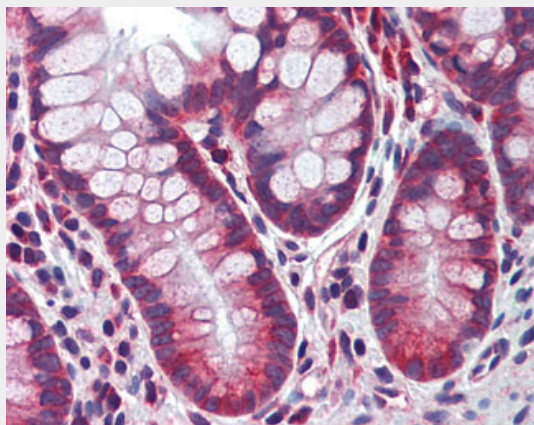
Isoform 1, isoform 5 and isoform 7 are expressed in a wide variety of tissues. Highest expression in peripheral blood leukocytes, spleen, thymus and liver. Barely detectable in brain, testis and skeletal muscle

CASP8 / Caspase 8 Antibody - Protocols

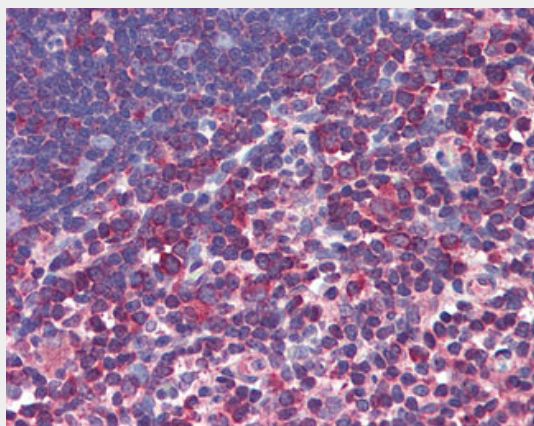
Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

CASP8 / Caspase 8 Antibody - Images



Anti-Caspase 8 antibody IHC of human small intestine.



Anti-Caspase 8 antibody IHC of human thymus.

CASP8 / Caspase 8 Antibody - Background

Most upstream protease of the activation cascade of caspases responsible for the TNFRSF6/FAS mediated and TNFRSF1A induced cell death. Binding to the adapter molecule FADD recruits it to either receptor. The resulting aggregate called death-inducing signaling complex (DISC) performs CASP8 proteolytic activation. The active dimeric enzyme is then liberated from the DISC and free to activate downstream apoptotic proteases. Proteolytic fragments of the N-terminal propeptide (termed CAP3, CAP5 and CAP6) are likely retained in the DISC. Cleaves and activates CASP3,

CASP4, CASP6, CASP7, CASP9 and CASP10. May participate in the GZMB apoptotic pathways. Cleaves ADPRT. Hydrolyzes the small-molecule substrate, Ac-Asp-Glu-Val-Asp-|-AMC. Likely target for the cowpox virus CRMA death inhibitory protein. Isoform 5, isoform 6, isoform 7 and isoform 8 lack the catalytic site and may interfere with the pro-apoptotic activity of the complex.

CASP8 / Caspase 8 Antibody - References

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Muzio M.,et al.Cell 85:817-827(1996).
Fernandes-Alnemri T.,et al.Proc. Natl. Acad. Sci. U.S.A. 93:7464-7469(1996).
Srinivasula S.M.,et al.J. Biol. Chem. 272:18542-18545(1997).
Grenet J.,et al.Gene 226:225-232(1999).