

CASP8 Antibody

Purified Mouse Monoclonal Antibody Catalog # AO1629a

Specification

CASP8 Antibody - Product Information

Application E, WB, IHC, FC

Primary Accession <u>Q14790</u>

Reactivity
Host
Clonality
Isotype
Calculated MW
Human, Mouse, Rat, Monkey
Mouse
Monoclonal
IgG1
26kDa KDa

Description

This gene encodes a member of the cysteine-aspartic acid protease (caspase) family. Sequential activation of caspases plays a central role in the execution-phase of cell apoptosis. Caspases exist as inactive proenzymes composed of a prodomain, a large protease subunit, and a small protease subunit. Activation of caspases requires proteolytic processing at conserved internal aspartic residues to generate a heterodimeric enzyme consisting of the large and small subunits. This protein is involved in the programmed cell death induced by Fas and various apoptotic stimuli. The N-terminal FADD-like death effector domain of this protein suggests that it may interact with Fas-interacting protein FADD. This protein was detected in the insoluble fraction of the affected brain region from Huntington disease patients but not in those from normal controls, which implicated the role in neurodegenerative diseases. Many alternatively spliced transcript variants encoding different isoforms have been described, although not all variants have had their full-length sequences determined.

Immunogen

Formulation

Ascitic fluid containing 0.03% sodium azide.

CASP8 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

Dilution

E~~1/10000 WB~~1/500 - 1/2000 IHC~~1/200 - 1/1000 FC~~1/200 - 1/400



Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

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

CASP8 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, PubMed:8681376, PubMed:8681377, PubMed:9006941, PubMed:9184224, PubMed:8962078, PubMed:35446120, PubMed:35338844). 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:8681376, PubMed:8681377, PubMed:9006941, PubMed:9184224, PubMed:8962078, PubMed:35446120, PubMed:35338844). Cleaves and activates effector caspases CASP3, CASP4, CASP6, CASP7, CASP9 and CASP10 (PubMed: 8962078, PubMed:9006941, PubMed:16916640). 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: <a



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href="http://www.uniprot.org/citations/32929201" target=" blank">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:Q9|HX4}. Nucleus {ECO:0000250|UniProtKB:Q9|HX4}. Cell projection, lamellipodium. Note=Recruitment to lamellipodia of migrating cells is enhanced by phosphorylation at Tyr-380

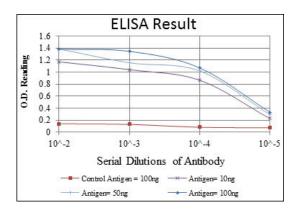
Tissue Location

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 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 Cvtometv
- Cell Culture





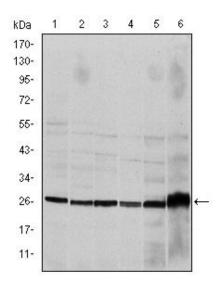


Figure 1: Western blot analysis using CASP8 mouse mAb against Hela (1), Jurkat (2), THP-1 (3), NIH/3T3 (4), Cos7 (5) and PC-12 (6) cell lysate.

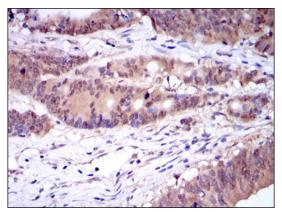


Figure 2: Immunohistochemical analysis of paraffin-embedded colon cancer tissues using CASP8 mouse mAb with DAB staining.

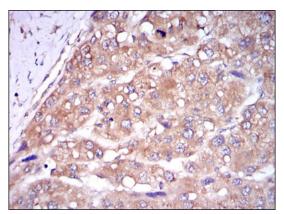


Figure 3: Immunohistochemical analysis of paraffin-embedded liver cancer tissues using CASP8 mouse mAb with DAB staining.



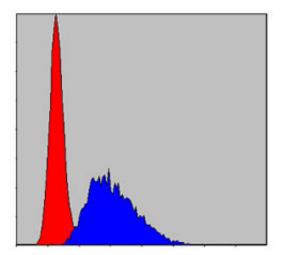


Figure 4: Flow cytometric analysis of NIH/3T3 cells using CASP8 mouse mAb (blue) and negative control (red).

CASP8 Antibody - References

1. Cancer Lett. 2009 Aug 28;281(2):128-33. 2. Cell Res. 2009 Mar;19(3):358-69.