

CASP8 Antibody (C-term) Blocking Peptide

Synthetic peptide Catalog # BP6559b

Specification

CASP8 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession <u>Q14790</u>

CASP8 Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 841

Other Names

Caspase-8, CASP-8, 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

The synthetic peptide sequence used to generate the antibody AP6559b was selected from the C-term region of human CASP8. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format

Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage

Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions

This product is for research use only. Not for use in diagnostic or therapeutic procedures.

CASP8 Antibody (C-term) Blocking Peptide -

CASP8 Antibody (C-term) Blocking Peptide - Background

CASP8 is 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.

CASP8 Antibody (C-term) Blocking Peptide - References

Ji,G., Hum. Reprod. 24 (10), 2439-2446 (2009)



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 (By similarity). Initiator protease that induces extrinsic apoptosis by mediating cleavage and activation of effector caspases responsible for the TNFRSF6/FAS mediated and TNFRSF1A induced cell death (PubMed: <a h ref="http://www.uniprot.org/citations/23516" 580" target=" blank">23516580, PubMed:8681376, PubMed:8681377, PubMed:9006941, PubMed:9184224, PubMed:8962078). Cleaves and activates effector caspases CASP3, CASP4, CASP6, CASP7, CASP9 and CASP10 (PubMed:8962078, PubMed: 9006941). Binding to the adapter molecule FADD recruits it to either receptor TNFRSF6/FAS mediated or TNFRSF1A (PubMed:8681376, PubMed:8681377). The resulting aggregate called 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-D (GSDMD): GSDMD cleavage promoting release of the N-terminal moiety (Gasdermin-D, N-terminal) that binds to membranes and forms pores, triggering pyroptosis (By similarity). 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 (PubMed: 8681376).

Cellular Location

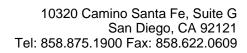
Cytoplasm

{ECO:0000250|UniProtKB:Q9JHX4}. Nucleus {ECO:0000250|UniProtKB:Q9JHX4}

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 (C-term) Blocking Peptide - Protocols





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

• Blocking Peptides