

CASP7 Antibody (Center) Blocking Peptide
Synthetic peptide
Catalog # BP1328b**Specification**

CASP7 Antibody (Center) Blocking Peptide - Product InformationPrimary Accession [P55210](#)**CASP7 Antibody (Center) Blocking Peptide - Additional Information****Gene ID** 840**Other Names**

Caspase-7, CASP-7, Apoptotic protease Mch-3, CMH-1, ICE-like apoptotic protease 3, ICE-LAP3, Caspase-7 subunit p20, Caspase-7 subunit p11, CASP7, MCH3

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP1328b](/products/AP1328b) was selected from the Center region of human CASP7. 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.

CASP7 Antibody (Center) Blocking Peptide - Protein Information**Name** CASP7 {ECO:0000303|PubMed:9070923, ECO:0000312|HGNC:HGNC:1508}**Function**

Thiol protease involved in different programmed cell death processes, such as apoptosis, pyroptosis or granzyme-mediated programmed cell death, by proteolytically cleaving target proteins (PubMed: [8521391](http://www.uniprot.org/citations/8521391), PubMed: [8567622](http://www.uniprot.org/citations/8567622), PubMed: [8576161](http://www.uniprot.org/citations/8576161), PubMed: [9070923](http://www.uniprot.org/citations/9070923), PubMed: [16916640](http://www.uniprot.org/citations/16916640), PubMed: [17646170](http://www.uniprot.org/citations/17646170), PubMed: [18723680](http://www.uniprot.org/citations/18723680), PubMed: [19581639](http://www.uniprot.org/citations/19581639), PubMed: [11257230](http://www.uniprot.org/citations/11257230))

target="_blank">11257230, PubMed:11257231, PubMed:11701129, PubMed:15314233). Has a marked preference for Asp-Glu-Val-Asp (DEVD) consensus sequences, with some plasticity for alternate non-canonical sequences (PubMed:12824163, PubMed:19581639, PubMed:20566630, PubMed:15314233, PubMed:17697120, PubMed:23897474, PubMed:23650375, PubMed:27032039). Its involvement in the different programmed cell death processes is probably determined by upstream proteases that activate CASP7 (By similarity). Acts as an effector caspase involved in the execution phase of apoptosis: following cleavage and activation by initiator caspases (CASP8, CASP9 and/or CASP10), mediates execution of apoptosis by catalyzing cleavage of proteins, such as CLSPN, PARP1, PTGES3 and YY1 (PubMed:10497198, PubMed:16123041, PubMed:16374543, PubMed:16916640, PubMed:18723680, PubMed:20566630, PubMed:21555521, PubMed:22184066, PubMed:22451931, PubMed:28863261, PubMed:31586028, PubMed:34156061, PubMed:27889207, PubMed:35338844, PubMed:35446120). Compared to CASP3, acts as a minor executioner caspase and cleaves a limited set of target proteins (PubMed:18723680). Acts as a key regulator of the inflammatory response in response to bacterial infection by catalyzing cleavage and activation of the sphingomyelin phosphodiesterase SMPD1 in the extracellular milieu, thereby promoting membrane repair (PubMed:21157428). Regulates pyroptosis in intestinal epithelial cells: cleaved and activated by CASP1 in response to S.typhimurium infection, promoting its secretion to the extracellular milieu, where it catalyzes activation of SMPD1, generating ceramides that repair membranes and counteract the action of gasdermin-D (GSDMD) pores (By similarity). Regulates granzyme-mediated programmed cell death in hepatocytes: cleaved and activated by granzyme B (GZMB) in response to bacterial infection, promoting its secretion to the extracellular milieu, where it catalyzes activation of SMPD1, generating ceramides that repair membranes and counteract the action of perforin (PRF1) pores (By similarity). Following cleavage by CASP1 in response to inflammasome activation, catalyzes processing and inactivation of PARP1, alleviating the transcription repressor activity of PARP1 (PubMed:22464733). Acts as an inhibitor of type I interferon production during virus-induced apoptosis by mediating cleavage of antiviral proteins CGAS, IRF3 and MAVS, thereby preventing cytokine overproduction (By similarity). Cleaves and activates sterol regulatory element binding proteins (SREBPs) (PubMed:8643593). Cleaves phospholipid scramblase proteins XKR4, XKR8 and XKR9 (By similarity). In case of infection, catalyzes cleavage of Kaposi sarcoma-associated herpesvirus protein ORF57, thereby preventing expression of viral lytic genes (PubMed:20159985).

Cellular Location

Cytoplasm, cytosol. Nucleus. Secreted, extracellular space {ECO:0000250|UniProtKB:P97864}.
Note=Following cleavage and activation by CASP1 or granzyme B (GZMB), secreted into the extracellular milieu by passing through the gasdermin-D (GSDMD) pores or perforin (PRF1) pore, respectively {ECO:0000250|UniProtKB:P97864}

Tissue Location

Highly expressed in lung, skeletal muscle, liver, kidney, spleen and heart, and moderately in testis. No expression in the brain.

CASP7 Antibody (Center) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

CASP7 Antibody (Center) Blocking Peptide - Images**CASP7 Antibody (Center) Blocking Peptide - Background**

CASP7 is a protein which 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 which undergo proteolytic processing at conserved aspartic residues to produce two subunits, large and small, that dimerize to form the active enzyme. The precursor of this caspase is cleaved by caspase 3 and 10. It is activated upon cell death stimuli and induces apoptosis.

CASP7 Antibody (Center) Blocking Peptide - References

Xu,H.L., Cancer Epidemiol. Biomarkers Prev. 18 (7), 2114-2122 (2009)Gibot,L., Biochem. J. 420 (3), 473-483 (2009)Kim,Y.R., Hum. Pathol. 40 (6), 868-871 (2009)