

# CASP6 Antibody (N-term) Blocking peptide

Synthetic peptide Catalog # BP13835a

### **Specification**

## CASP6 Antibody (N-term) Blocking peptide - Product Information

Primary Accession

P55212

## CASP6 Antibody (N-term) Blocking peptide - Additional Information

Gene ID 839

#### **Other Names**

Caspase-6, CASP-6, Apoptotic protease Mch-2, Caspase-6 subunit p18, Caspase-6 subunit p11, CASP6, MCH2

## **Target/Specificity**

The synthetic peptide sequence used to generate the antibody AP13835a was selected from the N-term region of CASP6. 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.

## CASP6 Antibody (N-term) Blocking peptide - Protein Information

Name CASP6 (HGNC:1507)

#### **Function**

Cysteine protease that plays essential roles in programmed cell death, axonal degeneration, development and innate immunity (PubMed:<a href="http://www.uniprot.org/citations/8663580" target="\_blank">8663580</a>, PubMed:<a href="http://www.uniprot.org/citations/19133298" target="\_blank">19133298</a>, PubMed:<a href="http://www.uniprot.org/citations/22858542" target="\_blank">22858542</a>, PubMed:<a href="http://www.uniprot.org/citations/27032039" target="\_blank">27032039</a>, PubMed:<a href="http://www.uniprot.org/citations/28864531" target="\_blank">28864531</a>, PubMed:<a href="http://www.uniprot.org/citations/30420425" target="\_blank">30420425</a>, PubMed:<a href="http://www.uniprot.org/citations/32298652" target="\_blank">32298652</a>). Acts as a non- canonical executioner caspase during apoptosis: localizes in the nucleus and cleaves the nuclear structural protein NUMA1 and lamin A/LMNA thereby inducing nuclear shrinkage and fragmentation (PubMed:<a href="http://www.uniprot.org/citations/8663580" target="\_blank">8663580</a>, PubMed:<a href="http://www.uniprot.org/citations/8663580" target="\_blank">8663580</a>, PubMed:<a



href="http://www.uniprot.org/citations/9463409" target=" blank">9463409</a>, PubMed:<a href="http://www.uniprot.org/citations/11953316" target=" blank">11953316</a>, PubMed:<a href="http://www.uniprot.org/citations/17401638" target="blank">17401638</a>). Lamin-A/LMNA cleavage is required for chromatin condensation and nuclear disassembly during apoptotic execution (PubMed: <a href="http://www.uniprot.org/citations/11953316" target=" blank">11953316</a>). Acts as a regulator of liver damage by promoting hepatocyte apoptosis: in absence of phosphorylation by AMP-activated protein kinase (AMPK), catalyzes cleavage of BID, leading to cytochrome c release, thereby participating in nonalcoholic steatohepatitis (PubMed:<a href="http://www.uniprot.org/citations/32029622" target=" blank">32029622</a>). Cleaves PARK7/DJ-1 in cells undergoing apoptosis (By similarity). Involved in intrinsic apoptosis by mediating cleavage of RIPK1 (PubMed:<a href="http://www.uniprot.org/citations/22858542" target=" blank">22858542</a>). Furthermore, cleaves many transcription factors such as NF-kappa-B and cAMP response element-binding protein/CREBBP (PubMed:<a href="http://www.uniprot.org/citations/10559921" target=" blank">10559921</a>, PubMed:<a href="http://www.uniprot.org/citations/14657026" target="blank">14657026</a>). Cleaves phospholipid scramblase proteins XKR4 and XKR9 (By similarity). In addition to apoptosis, involved in different forms of programmed cell death (PubMed:<a href="http://www.uniprot.org/citations/32298652" target=" blank">32298652</a>). Plays an essential role in defense against viruses by acting as a central mediator of the ZBP1-mediated pyroptosis, apoptosis, and necroptosis (PANoptosis), independently of its cysteine protease activity (PubMed:<a href="http://www.uniprot.org/citations/32298652" target=" blank">32298652</a>). PANoptosis is a unique inflammatory programmed cell death, which provides a molecular scaffold that allows the interactions and activation of machinery required for inflammasome/pyroptosis, apoptosis and necroptosis (PubMed: <a href="http://www.uniprot.org/citations/32298652" target="\_blank">32298652</a>). Mechanistically, interacts with RIPK3 and enhances the interaction between RIPK3 and ZBP1, leading to ZBP1-mediated inflammasome activation and cell death (PubMed:<a href="http://www.uniprot.org/citations/32298652" target=" blank">32298652</a>). Plays an essential role in axon degeneration during axon pruning which is the remodeling of axons during neurogenesis but not apoptosis (By similarity). Regulates B-cell programs both during early development and after antigen stimulation (By similarity).

**Cellular Location** Cytoplasm. Nucleus

## CASP6 Antibody (N-term) Blocking peptide - Protocols

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

#### Blocking Peptides

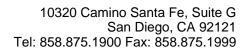
CASP6 Antibody (N-term) Blocking peptide - Images

## CASP6 Antibody (N-term) Blocking peptide - Background

This gene encodes a protein which is a member of thecysteine-aspartic acid protease (caspase) family. Sequentialactivation of caspases plays a central role in the execution-phaseof cell apoptosis. Caspases exist as inactive proenzymes whichundergo proteolytic processing at conserved aspartic residues toproduce two subunits, large and small, that dimerize to form theactive enzyme. This protein is processed by caspases 7, 8 and 10, and is thought to function as a downstream enzyme in the caspaseactivation cascade. Alternative splicing of this gene results intwo transcript variants that encode different isoforms. [providedby RefSeq].

## CASP6 Antibody (N-term) Blocking peptide - References

Wurstle, M.L., et al. J. Biol. Chem. 285(43):33209-33218(2010)Lee, S.Y., et al. J Thorac Oncol





5(8):1152-1158(2010) Halawani, D., et al. J. Neurosci. 30(17):6132-6142(2010) Kim, M.S., et al. APMIS 118(4):308-312(2010) Yoo, N.J., et al. Tumori 96(1):138-142(2010)