

# Phospho-Caspase 6(S257) Antibody Blocking peptide

Synthetic peptide Catalog # BP3043a

## **Specification**

## Phospho-Caspase 6(S257) Antibody Blocking peptide - Product Information

**Primary Accession** 

P55212

## Phospho-Caspase 6(S257) Antibody 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 <a href=/product/products/AP3043a>AP3043a</a> was selected from the region of human Phospho-Caspase 6-S257. 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.

## Phospho-Caspase 6(S257) Antibody 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/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

## Phospho-Caspase 6(S257) Antibody Blocking peptide - Protocols

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

• Blocking Peptides

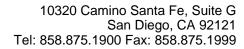
Phospho-Caspase 6(S257) Antibody Blocking peptide - Images

Phospho-Caspase 6(S257) Antibody Blocking peptide - Background

Caspase 6 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 2 subunits, large and small, that dimerize to form the active enzyme. This protein could be processed by caspases 7, 8 and 10, and is thought to function as a downstream enzyme in the caspase activation cascade.

## Phospho-Caspase 6(S257) Antibody Blocking peptide - References

Kalinin, A.E., et al., J. Invest. Dermatol. 124(1):46-55 (2005). Suzuki, A., et al., Oncogene





23(42):7067-7075 (2004).Horowitz, P.M., et al., J. Neurosci. 24(36):7895-7902 (2004).Schmeck, B., et al., Infect. Immun. 72(9):4940-4947 (2004).Mendez, E., et al., J. Virol. 78(16):8601-8608 (2004).