

CASP4 Antibody (N-term) Blocking Peptide
Synthetic peptide
Catalog # BP6723a**Specification****CASP4 Antibody (N-term) Blocking Peptide -
Product Information**Primary Accession [P49662](#)**CASP4 Antibody (N-term) Blocking Peptide -
Additional Information****Gene ID 837****Other Names**Caspase-4, CASP-4, ICE(rel)-II, Protease
ICH-2, Protease TX, Caspase-4 subunit 1,
Caspase-4 subunit 2, CASP4, ICH2**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP6723a](/products/AP6723a) was selected from the N-term region of human CASP4. 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.

**CASP4 Antibody (N-term) Blocking Peptide -
Protein Information****Name** CASP4

{ECO:0000303|PubMed:15123740,

**CASP4 Antibody (N-term) Blocking Peptide
- Background**

CASP4 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 and a large and 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 caspase is able to cleave and activate its own precursor protein, as well as caspase 1 precursor. When overexpressed, it induces cell apoptosis.

**CASP4 Antibody (N-term) Blocking Peptide
- References**

Lakshmanan,U., J. Immunol. 179 (12),
8480-8490 (2007)

ECO:0000312|HGNC:HGNC:1505}

Function

Inflammatory caspase that acts as an essential effector of NLRP3 inflammasome-dependent CASP1 activation and IL1B and IL18 secretion in response to non-canonical activators, such as UVB radiation, cholera enterotoxin subunit B and cytosolic LPS (PubMed:23516580, PubMed:24879791, PubMed:25119034, PubMed:22246630, PubMed:26174085, PubMed:26173988, PubMed:26508369, PubMed:25964352). Thiol protease that cleaves a tetrapeptide after an Asp residue at position P1 (PubMed:7797510, PubMed:23516580). Independently of NLRP3 inflammasome and CASP1, promotes pyroptosis, through GSDMD cleavage and activation, followed by IL1A, IL18 and HMGB1 release in response to non-canonical inflammasome activators (PubMed:26375003, PubMed:32109412). Plays a crucial role in the restriction of Salmonella typhimurium replication in colonic epithelial cells during infection: in later stages of the infection, LPS from cytosolic Salmonella triggers CASP4 activation, which catalyzes cleavage of GSDMD, resulting in pyroptosis

of infected cells and their extrusion into the gut lumen, as well as in IL18 secretion (PubMed:25121752, PubMed:26375003, PubMed:25964352, PubMed:32109412). Cleavage of GSDMD is not strictly dependent on the consensus cleavage site but depends on an exosite interface on CASP4 that recognizes and binds the Gasdermin-D, C- terminal (GSDMD-CT) part (PubMed:32109412). Pyroptosis limits bacterial replication, while cytokine secretion promotes the recruitment and activation of immune cells and triggers mucosal inflammation (PubMed:25121752, PubMed:26375003, PubMed:25964352). Involved in LPS- induced IL6 secretion; this activity may not require caspase enzymatic activity (PubMed:26508369). Involved in cell death induced by endoplasmic reticulum stress and by treatment with cytotoxic APP peptides found Alzheimer's patient brains (PubMed:15123740, PubMed:22246630, PubMed:23661706). Activated by direct binding to LPS without the need of an upstream sensor (PubMed:25119034). Does not directly process IL1B (PubMed:<a href="htt

p://www.uniprot.org/citations/7743998" target="_blank">7743998, PubMed:7797510, PubMed:7797592). During non-canonical inflammasome activation, cuts CGAS and may play a role in the regulation of antiviral innate immune activation (PubMed:28314590).

Cellular Location

Cytoplasm, cytosol. Endoplasmic reticulum membrane; Peripheral membrane protein; Cytoplasmic side. Mitochondrion Inflammasome. Secreted
Note=Predominantly localizes to the endoplasmic reticulum (ER) Association with the ER membrane requires TMEM214 (PubMed:15123740) Released in the extracellular milieu by keratinocytes following UVB irradiation (PubMed:22246630).

Tissue Location

Widely expressed, including in keratinocytes and colonic and small intestinal epithelial cells (at protein level). Not detected in brain.

**CASP4 Antibody (N-term) Blocking Peptide
- Protocols**

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

- [Blocking Peptides](#)