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CASP4 Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP6723b

Specification

CASP4 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession P49662

CASP4 Antibody (C-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 AP6723b was selected from the C-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 (C-term) Blocking Peptide - Protein Information

Name CASP4 {ECO:0000303|PubMed:15123740, ECO:0000312|HGNC:HGNC:1505}

Function
Inflammatory caspase that acts as the effector of the non- canonical inflammasome by mediating lipopolysaccharide (LPS)-induced pyroptosis (PubMed:25119034, PubMed:26375003, PubMed:34671164, PubMed:32109412, PubMed:37001519, PubMed:37993712, PubMed:37993714). Also indirectly activates the NLRP3 and NLRP6 inflammasomes (PubMed:7797510, PubMed:<a

href="http://www.uniprot.org/citations/23516580" target="_blank">23516580, PubMed:26375003, PubMed:32109412). Acts as a thiol protease that cleaves a tetrapeptide after an Asp residue at position P1: catalyzes cleavage of CGAS, GSDMD and IL18 (PubMed:7797510, PubMed:15326478, PubMed:23516580, PubMed:26375003, PubMed:28314590, PubMed:32109412, PubMed:37993712, PubMed:37993714). Effector of the non-canonical inflammasome independently of NLRP3 inflammasome and CASP1: the non-canonical inflammasome promotes pyroptosis through GSDMD cleavage without involving secretion of cytokine IL1B (PubMed:25121752, PubMed:25119034, PubMed:26375003, PubMed:31268602, PubMed:32109412, PubMed:37993712, PubMed:37993714). In the non-canonical inflammasome, CASP4 is activated by direct binding to the lipid A moiety of LPS without the need of an upstream sensor (PubMed:25121752, PubMed:25119034, PubMed:29520027, PubMed:32510692, PubMed:32581219, PubMed:37993712). LPS-binding promotes CASP4 activation and CASP4-mediated cleavage of GSDMD and IL18, followed by IL18 secretion through the GSDMD pore, pyroptosis of infected cells and their extrusion into the gut lumen (PubMed:25121752, PubMed:25119034, PubMed:37993712, PubMed:37993714). Also indirectly promotes secretion of mature cytokines (IL1A and HMGB1) downstream of GSDMD-mediated pyroptosis via activation of the NLRP3 and NLRP6 inflammasomes (PubMed:26375003, PubMed:32109412). Involved in NLRP3-dependent CASP1 activation and IL1B secretion in response to non-canonical activators, such as UVB radiation or cholera enterotoxin (PubMed:22246630, PubMed:23516580, PubMed:24879791, PubMed:25964352, PubMed:26173988, PubMed:26174085, PubMed:26508369). Involved in NLRP6 inflammasome- dependent activation in response to lipoteichoic acid (LTA), a cell- wall component of Gram-positive bacteria, which leads to CASP1 activation and IL1B secretion (PubMed:33377178). Involved in LPS- induced IL6 secretion; this activity may not require caspase enzymatic activity (PubMed:26508369). The non-canonical inflammasome is required for innate immunity to cytosolic, but not vacuolar, bacteria (By similarity). Plays a crucial role in the restriction of S.typhimurium replication in colonic epithelial cells during infection (PubMed:<a href="http://www.uniprot.org/citations/25121752"

target="_blank">25121752, PubMed:25964352). 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). May also act as an activator of adaptive immunity in dendritic cells, following activation by oxidized phospholipid 1-palmitoyl-2-arachidonoyl- sn-glycero-3- phosphorylcholine, an oxidized phospholipid (oxPAPC) (By similarity). Involved in cell death induced by endoplasmic reticulum stress and by treatment with cytotoxic APP peptides found in Alzheimer's patient brains (PubMed:15123740, PubMed:22246630, PubMed:23661706). 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). Catalyzes cleavage and maturation of IL18; IL18 processing also depends of the exosite interface on CASP4 (PubMed:15326478, PubMed:37993712, PubMed:37993714). In contrast, it does not directly process IL1B (PubMed:7743998, PubMed:7797592, PubMed:7797510). 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 (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

CASP4 Antibody (C-term) Blocking Peptide - Images

CASP4 Antibody (C-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.

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