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CASP4 Blocking Peptide (Center)

Synthetic peptide
Catalog # BP21390c

Specification

CASP4 Blocking Peptide (Center) - Product Information

Primary Accession P49662

CASP4 Blocking Peptide (Center) - 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 is selected from aa 163-177 of HUMAN CASP4

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 Blocking Peptide (Center) - Protein Information

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

Function
Inflammatory caspase that acts as an essential effector of NLRP3 inflammasome-dependent CASP1 activation

CASP4 Blocking Peptide (Center) - Background

Involved in the activation cascade of caspases responsible for apoptosis execution. Involved in ER-stress induced apoptosis. Cleaves caspase-1.

CASP4 Blocking Peptide (Center) - References

Faucheu C.,et al.EMBO J. 14:1914-1922(1995).
Munday N.A.,et al.J. Biol. Chem. 270:15870-15876(1995).
Kamens J.,et al.J. Biol. Chem. 270:15250-15256(1995).
Fernandes-Alnemri T.,et al.Submitted (JUN-1995) to the EMBL/GenBank/DDBJ databases.
Taylor T.D.,et al.Nature 440:497-500(2006).

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:<a href="http://www.uniprot.org/ci

tations/26375003"
target="_blank">26375003,
PubMed:<a href="http://www.uniprot.org/ci
tations/25964352"
target="_blank">25964352,
PubMed:<a href="http://www.uniprot.org/ci
tations/32109412"
target="_blank">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:<a href="http://
www.uniprot.org/citations/32109412"
target="_blank">32109412).
Pyroptosis limits bacterial replication, while
cytokine secretion promotes the
recruitment and activation of immune cells
and triggers mucosal inflammation
(PubMed:<a href="http://www.uniprot.org/c
itations/25121752"
target="_blank">25121752,
PubMed:<a href="http://www.uniprot.org/ci
tations/26375003"
target="_blank">26375003,
PubMed:<a href="http://www.uniprot.org/ci
tations/25964352"
target="_blank">25964352). Involved
in LPS- induced IL6 secretion; this activity
may not require caspase enzymatic activity
(PubMed:<a href="http://www.uniprot.org/c
itations/26508369"
target="_blank">26508369). Involved
in cell death induced by endoplasmic
reticulum stress and by treatment with
cytotoxic APP peptides found Alzheimer's
patient brains (PubMed:<a href="http://ww
w.uniprot.org/citations/15123740"
target="_blank">15123740,
PubMed:<a href="http://www.uniprot.org/ci
tations/22246630"
target="_blank">22246630,
PubMed:<a href="http://www.uniprot.org/ci
tations/23661706"
target="_blank">23661706).
Activated by direct binding to LPS without
the need of an upstream sensor
(PubMed:<a href="http://www.uniprot.org/c
itations/25119034"
target="_blank">25119034). Does not
directly process IL1B (PubMed:<a href="htt
p://www.uniprot.org/citations/7743998"
target="_blank">7743998,
PubMed:<a href="http://www.uniprot.org/ci
tations/7797510"
target="_blank">7797510,
PubMed:<a href="http://www.uniprot.org/ci

tations/7797592"
target="_blank">7797592). During
non-canonical inflammasome activation,
cuts CGAS and may play a role in the
regulation of antiviral innate immune
activation (PubMed:<a href="http://www.un
iprot.org/citations/28314590"
target="_blank">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 Blocking Peptide (Center) - Protocols

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

- Blocking Peptides