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Caspase-4 Antibody

Catalog # ASC10294

Product Information

Application	WB, IF, E, IHC-P
Primary Accession	P49662
Other Accession	AAA86890, 886050
Reactivity	Human
Host	Rabbit
Clonality	Polyclonal
Isotype	IgG
Calculated MW	43262
Concentration (mg/ml)	1 mg/mL
Conjugate	Unconjugated
Application Notes	Casp-4 antibody can be used for the detection of Caspase-4 by Western blot at 1 and 2 □g/mL. Antibody can also be used for immunohistochemistry starting at 2 □g/mL. For immunofluorescence start at 10 □g/mL.

Additional Information

Gene ID	837
Other Names	Caspase-4 Antibody: TX, ICH-2, Mih1/TX, ICEREL-II, ICE(rel)II, ICH2, Caspase-4, Protease ICH-2, CASP-4, caspase 4, apoptosis-related cysteine peptidase
Target/Specificity	CASP4; Depending on cell lines or tissues used, other cleavage products may be observed.
Reconstitution & Storage	Caspase-4 antibody can be stored at 4℃ for three months and -20℃, stable for up to one year. As with all antibodies care should be taken to avoid repeated freeze thaw cycles. Antibodies should not be exposed to prolonged high temperatures.
Precautions	Caspase-4 Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

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:32109412, PubMed:34671164, PubMed:37001519, PubMed:37993712, PubMed:37993714). Also indirectly activates the NLRP3 and NLRP6 inflammasomes (PubMed:23516580, PubMed:26375003, PubMed:32109412, PubMed:7797510). Acts as a thiol protease that cleaves a tetrapeptide after an Asp residue at position P1: catalyzes cleavage of CGAS, GSDMD and IL18

(PubMed:15326478, PubMed:23516580, PubMed:26375003, PubMed:28314590, PubMed:32109412, PubMed:37993712, PubMed:37993714, PubMed:7797510). 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:25119034, PubMed:25121752, 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:25119034, PubMed:25121752, 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:25119034, PubMed:25121752, 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:25121752, PubMed:25964352). Activation of the non-canonical inflammasome in brain endothelial cells can lead to excessive pyroptosis, leading to blood-brain barrier breakdown (By similarity). Pyroptosis limits bacterial replication, while cytokine secretion promotes the recruitment and activation of immune cells and triggers mucosal inflammation (PubMed:25121752, PubMed:25964352, PubMed:26375003). 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: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.

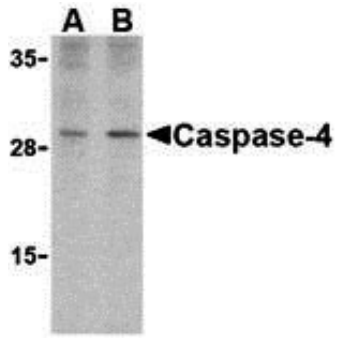
Background

Caspase-4 Antibody: Caspases are a family of cysteine proteases that can be divided into the apoptotic and inflammatory caspase subfamilies. Unlike the apoptotic caspases, members of the inflammatory subfamily are generally not involved in cell death but are associated with the immune response to microbial pathogens. Members of this subfamily include caspase-1, -4, -5, and -12. Activation of these caspases results in the cleavage and activation of proinflammatory cytokines such as IL-1β and IL-18. Caspase-4 was initially identified as a homologous protein to Caspase-1 and the C. elegans Ced-3 which could induce apoptosis in transfected cells. More recent studies have shown that it can be activated by ER stress and has been suggested to be involved in multiple neuronal pathologies such as Alzheimer's disease.

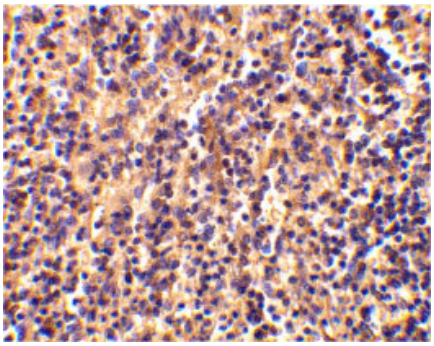
References

Martinon F and Tschopp J. Inflammatory caspases: linking an intracellular innate immune system to autoinflammatory diseases. Cell 2004; 117:561-74.
Kuida K, Lippke JA, Ku G, et al. Altered cytokine export and apoptosis in mice deficient in interleukin-1 β converting enzyme. Science 1995; 267:2000-3.
Gracie JA, Robertson SE, and McInnes IB. Interleukin-18. J. Leukoc. Biol. 2003; 73:213-224.
Kamens J, Paskind M, Hugunin M, et al. Identification and characterization of ICH-2, a novel member of the interleukin-1 β-converting enzyme family of cysteine proteases. J. Biol. Chem. 1995; 270:15250-6.

Images

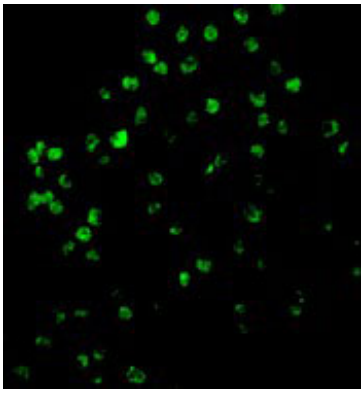


Western blot analysis of caspase-4 in human spleen cells with caspase-4 antibody at (A) 1 and (B) 2 µg/mL.



Immunohistochemical staining of human spleen tissue using caspase-4 antibody at 2 µg/mL.

Immunofluorescence of Caspase-4 in A20 cells with Caspase-4 antibody at 10 µg/mL.



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