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Caspase-8 subunit p10 Rabbit pAb

Catalog # AP96336

Product Information

Application	WB, IHC-P, IHC-F, IF
Primary Accession	O89110
Other Accession	O89110
Reactivity	Rat, Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Calculated MW	55357
Physical State	Liquid
Immunogen	KLH conjugated synthetic peptide derived from mouse Caspase-8 subunit p10
Epitope Specificity	351-420/480
Isotype	IgG
Purity	affinity purified by Protein A
Buffer	0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.
SIMILARITY	Belongs to the peptidase C14A family.Contains 2 DED (death effector) domains.
SUBUNIT	Heterotetramer that consists of two anti-parallel arranged heterodimers, each one formed by a 18 kDa (p18) and a 10 kDa (p10) subunit. Interacts with FADD, CFLAR and PEA15. Isoform 9 interacts at the endoplasmic reticulum with a complex containing BCAP31, BAP29, BCL2 and/or BCL2L1. Interacts with TNFAIP8L2.
DISEASE	Defects in CASP8 are the cause of caspase-8 deficiency (CASP8D) [MIM:607271]. CASP8D is a disorder resembling autoimmune lymphoproliferative syndrome (ALPS). It is characterized by lymphadenopathy, splenomegaly, and defective CD95-induced apoptosis of peripheral blood lymphocytes (PBLs). It leads to defects in activation of T-lymphocytes, B-lymphocytes, and natural killer cells leading to immunodeficiency characterized by recurrent sinopulmonary and herpes simplex virus infections and poor responses to immunization.
Important Note	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
Background Descriptions	Caspases are cysteine proteases, expressed as inactive precursors, that mediate apoptosis by proteolysis of specific substrates. Caspases have the ability to cleave after aspartic acid residues. There are two classes of caspases involved in apoptosis; initiators (activation by receptor cluster) and effectors (activation by mitochondrial permeability transition). Proapoptotic signals autocatalytically activate initiator caspases, such as Caspase 8 and Caspase 9. Activated initiator caspases then process effector caspases, such as Caspase 3 and Caspase 7, which in turn cause cell collapse.

Additional Information

Gene ID	12370

Other Names	ALPS2B; CAP4; Casp-8; FLICE; MACH; MCH5; CD20; CK-20; CK20; K20; KRT21; CASP8_HUMAN; CASP8; Apoptotic cysteine protease; Apoptotic protease Mch-5; FADD-homologous ICE/ced-3-like protease; FADD-like ICE (FLICE); ICE-like apoptotic protease 5; MORT1-associated ced-3 homolog (MACH); 3.4.22.61; CASP8_MOUSE; CASP8_RAT; caspase 8; caspase 8, apoptosis-related cysteine protease; caspase 8, apoptosis-related cysteine peptidase
Dilution	WB=1:500-2000,IHC-P=1:100-500,IHC-F=1:100-500,ICC/IF=1:100-500,IF=1:100-500,Flow-Cyt=1 □g/Test
Storage	Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.

Protein Information

Name	CASP8
Function	Thiol protease that plays a key role in programmed cell death by acting as a molecular switch for apoptosis, necroptosis and pyroptosis, and is required to prevent tissue damage during embryonic development and adulthood (PubMed:12065591, PubMed:18455983, PubMed:30361383, PubMed:30381458, PubMed:31511692, PubMed:31748744, PubMed:33397971, PubMed:9727491, PubMed:9727492). Initiator protease that induces extrinsic apoptosis by mediating cleavage and activation of effector caspases responsible for FAS/CD95-mediated and TNFRSF1A- induced cell death (PubMed:24813849, PubMed:24813850, PubMed:9654089, PubMed:9837723). Cleaves and activates effector caspases CASP3, CASP4, CASP6, CASP7, CASP9 and CASP10 (By similarity). Binding to the adapter molecule FADD recruits it to either receptor FAS/CD95 or TNFRSF1A (PubMed:29440439). The resulting aggregate called the death-inducing signaling complex (DISC) performs CASP8 proteolytic activation (By similarity). The active dimeric enzyme is then liberated from the DISC and free to activate downstream apoptotic proteases (By similarity). Proteolytic fragments of the N-terminal propeptide (termed CAP3, CAP5 and CAP6) are likely retained in the DISC (By similarity). Also cleaves and activates BID, thereby promoting cytochrome C release from mitochrondria (PubMed:9727491, PubMed:9727492). In addition to extrinsic apoptosis, also acts as a negative regulator of necroptosis: acts by cleaving RIPK1 at 'Asp-325', which is crucial to inhibit RIPK1 kinase activity, limiting TNF-induced apoptosis, necroptosis and inflammatory response (PubMed:31511692). Also able to initiate pyroptosis by mediating cleavage and activation of gasdermin-C and -D (GSDMC and GSDMD, respectively): gasdermin cleavage promotes release of the N-terminal moiety that binds to membranes and forms pores, triggering pyroptosis (PubMed:30361383, PubMed:30381458). Initiates pyroptosis following inactivation of MAP3K7/TAK1 (PubMed:30361383, PubMed:30381458). Also acts as a regulator of innate immunity by mediating cleavage and inactivation of N4BP1 downstream of TLR3 or TLR4, thereby promoting cytokine production (PubMed:32971525). May participate in the Granzyme B (GZMB) cell death pathways (By similarity). Cleaves PARP1 and PARP2 (PubMed:12065591).
Cellular Location	Cytoplasm. Nucleus. Note=Translocates into the nucleus during apoptosis.
Tissue Location	Expressed in a wide variety of tissues. Highest expression in spleen, thymus, lung, liver and kidney. Lower expression in heart, brain, testis and skeletal muscle

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