ELK Biotechnology

Cleaved-Caspase-8 p18 (S217) rabbit pAb

Cat No.:ES8370

For research use only

Overview

Product Name	Cleaved-Caspase-8 p18 (S217) rabbit pAb
Host species	Rabbit
Applications	WB;ELISA
Species Cross-Reactivity	Human;Rat;Mouse;
Recommended dilutions	Western Blot: 1/500 - 1/2000. ELISA: 1/10000. Not yet tested in other applications.
Immunogen	Synthesized peptide derived from Cleaved-Caspase-8 p18 (S217) . at AA range: 170-250
Specificity	Cleaved-Caspase-8 p18 (S217) Polyclonal Antibody detects endogenous levels of Cleaved-Caspase-8 p18 (S217) protein.
Formulation	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Storage	Store at -20℃. Avoid repeated freeze-thaw cycles.
Protein Name	Caspase8
Gene Name	CASP8
Cellular localization	Cytoplasm . Nucleus .
Purification	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Clonality	Polyclonal
Concentration	1 mg/ml
Observed band	18 54kD
Human Gene ID	841
Human Swiss-Prot Number	Q14790
Alternative Names	CASP8; MCH5; Caspase-8; CASP-8; Apoptotic cysteine protease; Apoptotic protease Mch-5; CAP4; FADD-homologous ICE/ced-3-like protease; FADD-like ICE; FLICE; ICE-like apoptotic protease 5; MORT1-associated ced-3 homolog; MACH
Background	This gene encodes 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, a large protease subunit, and a 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 protein is involved in the programmed cell death induced by Fas and various apoptotic stimuli. The N-terminal FADD-like death effector domain of this protein suggests that it may interact with Fas-interacting protein FADD. This protein was detected in the insoluble fraction of the affected brain region from Huntington disease patients but not in those from normal controls, which implicated the role in neurodegenerative diseases. Many alt