

Caspase-8 Polyclonal Antibody

Catalog Number: E-AB-62025

4 Publications



Note: Centrifuge before opening to ensure complete recovery of vial contents.

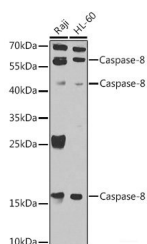
Description

Reactivity	Human, Mouse, Rat
Immunogen	Recombinant fusion protein of human Caspase-8 (NP_001073593.1).
Host	Rabbit
Isotype	IgG
Purification	Affinity purification
Conjugation	Unconjugated
Formulation	PBS with 0.02% sodium azide, 50% glycerol, pH7.3.

Applications Recommended Dilution

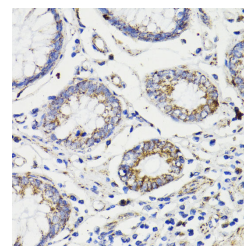
WB	1:500-1:2000
IHC	1:50-1:200
IF	1:50-1:200

Data

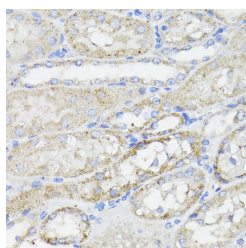


Western blot analysis of extracts of various cell lines using Caspase-8 Polyclonal Antibody at dilution of 1:1000.

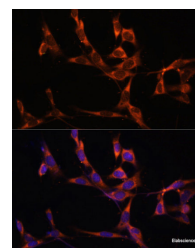
Observed Mw: 17kDa/44kDa/60kDa
Calculated Mw: 25-32kDa/45- 61kDa



Immunohistochemistry of paraffin-embedded Human colon carcinoma using Caspase-8 Polyclonal Antibody at dilution of 1:200 (40x lens).



Immunohistochemistry of paraffin-embedded Mouse kidney using Caspase-8 Polyclonal Antibody at dilution of 1:200 (40x lens).



Immunofluorescence analysis of NIH/3T3 cells using Caspase-8 Polyclonal Antibody at dilution of 1:100. Blue: DAPI for nuclear staining.

Preparation & Storage

Storage Store at -20°C. Avoid freeze / thaw cycles.

Background

For Research Use Only

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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 alternatively spliced transcript variants encoding different isoforms have been described, although not all variants have had their full-length sequences determined.

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