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Cleaved-CASP8 (D384) Polyclonal Antibody

Catalog No.E-AB-30009ReactivityHStorageStore at -20°C. Avoid freeze / thaw cycles.HostRabbitApplicationsWB,IHC-p,IF,ELISAIsotypeIgG

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

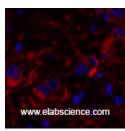
Images



Western Blot analysis of 293 cells with Cleaved-CASP8 (D384) Polyclonal Antibody



Immunohistochemistry of paraffinembedded Human kidney tissue using Cleaved-CASP8 (D384) Polyclonal Antibody at dilution of 1:200.



Immunofluorescence analysis of Human breast cancer tissue using Cleaved-CASP8 (D384) Polyclonal Antibody at dilution of 1:200.

Immunogen Information

Immunogen Synthesized peptide derived from the C-terminal

region of human Caspase-8

Swissprot Q14790

Synonyms 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

Product Information

Calculated MW 55kDa
Observed MW 47+55kDa

Buffer PBS with 0.02% sodium azide, 0.5% protective

protein and 50% glycerol, pH7.4

Purify Affinity purification

Dilution WB 1:500-2000, IHC 1:50-300, IF 1:50-300

Background

Most upstream protease of the activation cascade of caspases responsible for the TNFRSF6/FAS mediated and TNFRSF1A induced cell death. Binding to the adapter molecule FADD recruits it to either receptor. The resulting aggregate called death-inducing signaling complex (DISC) performs CASP8 proteolytic activation. The active dimeric enzyme is then liberated from the DISC and free to activate downstream apoptotic proteases. Proteolytic fragments of the N-terminal propeptide (termed CAP3, CAP5 and CAP6) are likely retained in the DISC. Cleaves and activates CASP3, CASP4, CASP6, CASP7, CASP9 and CASP10. May participate in the GZMB apoptotic pathways. Cleaves ADPRT. Hydrolyzes the small-molecule substrate, Ac-Asp-Glu-Val-Asp-AMC. Likely target for the cowpox virus CRMA death inhibitory protein. Isoform 5, isoform 6, isoform 7 and isoform 8 lack the catalytic site and may interfere with the pro-apoptotic activity of the complex.

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