

Cleaved-CASP8 (D384) Polyclonal Antibody

Catalog No. E-AB-30009

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

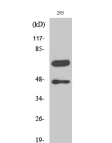
Description

| | |
|---------------------|--|
| Reactivity | Human |
| Immunogen | Synthesized peptide derived from the C-terminal region of human Caspase-8 |
| Host | Rabbit |
| Isotype | IgG |
| Purification | Affinity purification |
| Conjugation | Unconjugated |
| Buffer | PBS with 0.02% sodium azide, 0.5% protective protein and 50% glycerol, pH7.4 |

Applications Recommended Dilution

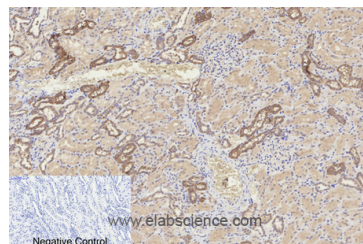
| | |
|------------|------------|
| WB | 1:500-2000 |
| IHC | 1:50-300 |
| IF | 1:50-300 |

Data

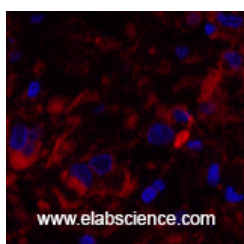


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Western Blot analysis of 293 cells with Cleaved-CASP8 (D384) Polyclonal Antibody
Observed Mw:47+55kDa
Calculated Mw:55kDa



Immunohistochemistry of paraffin-embedded 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.

Preparation & Storage

For Research Use Only

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

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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