

# CASP8 Antibody (C-term)

Purified Mouse Monoclonal Antibody (Mab)

Catalog # AM2113b

## Product Information

|                   |                        |
|-------------------|------------------------|
| Application       | WB, FC, E              |
| Primary Accession | <a href="#">Q14790</a> |
| Reactivity        | Human                  |
| Host              | Mouse                  |
| Clonality         | monoclonal             |
| Isotype           | IgG1                   |
| Clone Names       | 550CT8.5.2             |
| Calculated MW     | 55391                  |

## Additional Information

|                    |  |
|--------------------|--|
| Gene ID            | 841  |
| Other Names        | Caspase-8, CASP-8, 3.4.22.61, 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, Caspase-8 subunit p18, Caspase-8 subunit p10, CASP8, MCH5 |
| Target/Specificity | This CASP8 antibody is generated from a mouse immunized with a KLH conjugated synthetic peptide between 427-461 amino acids from the C-terminal region of human CASP8.   |
| Dilution           | WB~~1:2000 FC~~1:25 E~~Use at an assay dependent concentration.  |
| Format             | Purified monoclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein G column, followed by dialysis against PBS.  |
| Storage            | Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.  |
| Precautions        | CASP8 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.  |

## Protein Information

|          |  |
|----------|--|
| Name     | CASP8 {ECO:0000303 PubMed:9931493, ECO:0000312 HGNC:HGNC:1509}   |
| 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:[23516580](#), PubMed:[35338844](#), PubMed:[35446120](#), PubMed:[8681376](#), PubMed:[8681377](#), PubMed:[8962078](#), PubMed:[9006941](#), PubMed:[9184224](#)). 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:[23516580](#), PubMed:[35338844](#), PubMed:[35446120](#), PubMed:[8681376](#), PubMed:[8681377](#), PubMed:[8962078](#), PubMed:[9006941](#), PubMed:[9184224](#)). Cleaves and activates effector caspases CASP3, CASP4, CASP6, CASP7, CASP9 and CASP10 (PubMed:[16916640](#), PubMed:[8962078](#), PubMed:[9006941](#)). Binding to the adapter molecule FADD recruits it to either receptor FAS/TNFRSF6 or TNFRSF1A (PubMed:[8681376](#), PubMed:[8681377](#)). The resulting aggregate called the death-inducing signaling complex (DISC) performs CASP8 proteolytic activation (PubMed:[9184224](#)). The active dimeric enzyme is then liberated from the DISC and free to activate downstream apoptotic proteases (PubMed:[9184224](#)). Proteolytic fragments of the N-terminal propeptide (termed CAP3, CAP5 and CAP6) are likely retained in the DISC (PubMed:[9184224](#)). In addition to extrinsic apoptosis, also acts as a negative regulator of necroptosis: acts by cleaving RIPK1 at 'Asp-324', which is crucial to inhibit RIPK1 kinase activity, limiting TNF-induced apoptosis, necroptosis and inflammatory response (PubMed:[31827280](#), PubMed:[31827281](#)). 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:[32929201](#), PubMed:[34012073](#)). Initiates pyroptosis following inactivation of MAP3K7/TAK1 (By similarity). Also acts as a regulator of innate immunity by mediating cleavage and inactivation of N4BP1 downstream of TLR3 or TLR4, thereby promoting cytokine production (By similarity). May participate in the Granzyme B (GZMB) cell death pathways (PubMed:[8755496](#)). Cleaves PARP1 and PARP2 (PubMed:[8681376](#)). Independent of its protease activity, promotes cell migration following phosphorylation at Tyr-380 (PubMed:[18216014](#), PubMed:[27109099](#)).

#### Cellular Location

Cytoplasm {ECO:0000250|UniProtKB:Q9JHX4}. Nucleus {ECO:0000250|UniProtKB:Q9JHX4}. Cell projection, lamellipodium. Note=Recruitment to lamellipodia of migrating cells is enhanced by phosphorylation at Tyr-380

#### Tissue Location

Isoform 1, isoform 5 and isoform 7 are expressed in a wide variety of tissues. Highest expression in peripheral blood leukocytes, spleen, thymus and liver. Barely detectable in brain, testis and skeletal muscle

## Background

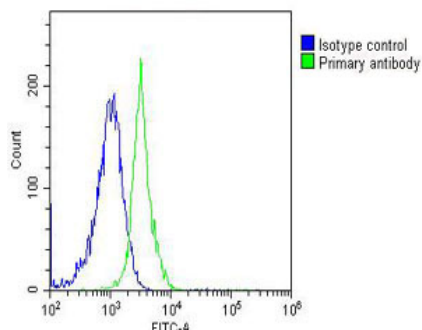
Most upstream protease of the activation cascade of caspases responsible for the TNFRSF6/FAS mediated and TNFRSF1A induced cell death. Binding to the adapter molecule FADD recruits it to either receptor. The resulting aggregate called death- inducing signaling complex (DISC) performs CASP8 proteolytic 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.

## References

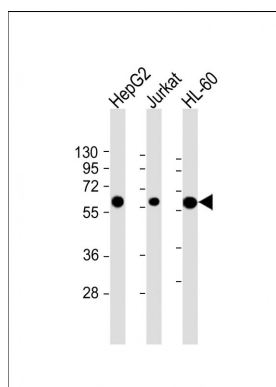
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 Fernandes-Alnemri T.,et al.Proc. Natl. Acad. Sci. U.S.A. 93:7464-7469(1996).  
 Srinivasula S.M.,et al.J. Biol. Chem. 272:18542-18545(1997).  
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## Images



Overlay histogram showing Jurkat cells stained with AM2113b(green line). The cells were fixed with 2% paraformaldehyde (10 min) and then permeabilized with 90% methanol for 10 min. The cells were then incubated in 2% bovine serum albumin to block non-specific protein-protein interactions followed by the antibody (AM2113b, 1:25 dilution) for 60 min at 37°C. The secondary antibody used was Goat-Anti-Mouse IgG, DyLight® 488 Conjugated Highly Cross-Adsorbed(OJ192088) at 1/200 dilution for 40 min at 37°C. Isotype control antibody (blue line) was mouse IgG1 (1µg/1x10<sup>6</sup> cells) used under the same conditions. Acquisition of >10, 000 events was performed.



All lanes : Anti-CASP8 Antibody (C-term) at 1:2000 dilution  
 Lane 1: HepG2 whole cell lysate Lane 2: Jurkat whole cell lysate Lane 3: HL-60 whole cell lysate Lysates/proteins at 20 µg per lane. Secondary Goat Anti-mouse IgG, (H+L), Peroxidase conjugated at 1/10000 dilution. Predicted band size : 55 kDa Blocking/Dilution buffer: 5% NFDM/TBST.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.