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Product datasheet for AM05282PU-N

Caspase 3 (CASP3) Mouse Monoclonal Antibody [Clone ID: AM1-4]

Product data:

Product Type:	Primary Antibodies
Clone Name:	AM1-4
Applications:	WB
Recommended Dilution:	Western Blot.
Reactivity:	Human
Host:	Mouse
Isotype:	IgG
Clonality:	Monoclonal
Immunogen:	Hybridoma produced by the fusion of splenocytes from mice immunized with recombinant human Caspase-3 protein and mouse myeloma cells.
Specificity:	This antibody Detects human Caspase-3.
Formulation:	PBS containing 0.08% Sodium Azide as preservative. State: Purified State: Liquid (sterile filtered) purified IgG fraction.
Concentration:	lot specific
Conjugation:	Unconjugated
Storage:	Store the antibody at -20°C. Avoid repeated freezing and thawing.
Stability:	Shelf life: One year from despatch.
Gene Name:	caspase 3
Database Link:	Entrez Gene 836 Human P42574

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Background: Caspase-3 along with caspase 7 and 6 form the group of effector caspases that are responsible for the cleavage of multiple substrates including the cytokeratins, PARP, alpha fodrin, NuMA and others. Caspase-7 occurs in three varient forms.Caspase-3-like activities are required for Fas-mediated apoptosis. However, the role of caspase-1 and caspase-3 in mediating Fas-induced cell death is not clear. Although wild-type, caspase-1(-/-), and caspase-3(-/-) hepatocytes were killed at a similar rate when cocultured with FasL expressing NIH 3T3 cells, caspase-3(-/-) hepatocytes displayed drastically different morphological changes as well as significantly delayed DNA fragmentation. For both wild-type and caspase-1 (-/-) apoptotic hepatocytes, typical apoptotic features such as cytoplasmic blebbing and nuclear fragmentation are seen within 6 hr, but neither event was observed for caspase-3(-/-) hepatocytes. In thymocytes apoptotic caspase-3 (-/-) thymocytes exhibit similar abnormal morphological changes and delayed DNA fragmentation observed in hepatocytes. Cleavage of various caspase substrates implicates apoptotic events, including gelsolin, fodrin, laminB, and DFF45/ICAD are delayed or absent. The altered cleavage of these key substrates is likely responsible for the aberrant apoptosis observed in both hepatocytes and thymocytes deficient in caspase-3.

Synonyms: CASP-3, CASP3, CPP32, CPP-32, Yama protein, Apopain, SCA-1, SCA1