

ICAD (FL-331): sc-9066

BACKGROUND

The Ced/ICE family of cysteine proteases plays a pivotal role in mediating apoptosis through the proteolysis of specific targets. Among the targets are poly(ADP-ribose) polymerase (PARP), gelsolin, DFF-45/ICAD and the nuclear lamins. PARP is a nuclear protein that is specifically cleaved by CPP32 and Mch2, but not by ICE, into a signature apoptotic fragment. Gelsolin is cleaved by CPP32 to an active form that severs actin filaments in a Ca²⁺-independent manner. In addition to binding actin, gelsolin can form complexes with fibronectin, which may be important for localizing gelsolin to inflammatory sites. DFF-45/ICAD, the subunit of DNA fragmentation factor, is cleaved by CPP32 to generate an active factor that induces DNA fragmentation. The nuclear Lamin A is cleaved by Mch2, but not CPP32. Nuclear Lamin B is fragmented as a consequence of apoptosis by an unidentified member of the ICE family.

CHROMOSOMAL LOCATION

Genetic locus: DFFA (human) mapping to 1p36.22; Dffa (mouse) mapping to 4 E2.

SOURCE

ICAD (FL-331) is a rabbit polyclonal antibody raised against amino acids 1-331 representing full length ICAD of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

ICAD (FL-331) is available conjugated to agarose (sc-9066 AC), 500 µg/0.25 ml agarose in 1 ml, for IP.

APPLICATIONS

ICAD (FL-331) is recommended for detection of ICAD and DFF-35 splice variant of mouse, rat and human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

ICAD (FL-331) is also recommended for detection of ICAD and DFF-35 splice variant in additional species, including equine.

Suitable for use as control antibody for ICAD siRNA (h): sc-35624, ICAD siRNA (m): sc-35625, ICAD shRNA Plasmid (h): sc-35624-SH, ICAD shRNA Plasmid (m): sc-35625-SH, ICAD shRNA (h) Lentiviral Particles: sc-35624-V and ICAD shRNA (m) Lentiviral Particles: sc-35625-V.

Molecular Weight of ICAD DFF-45 splice variant: 45 kDa.

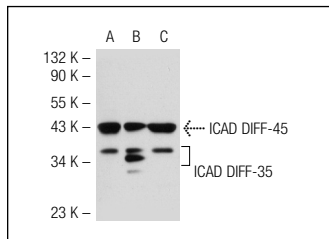
Molecular Weight of ICAD DFF-35 splice variant: 35 kDa.

Positive Controls: ICAD (m): 293T Lysate: sc-125479, U-937 cell lysate: sc-2239 or HeLa whole cell lysate: sc-2200.

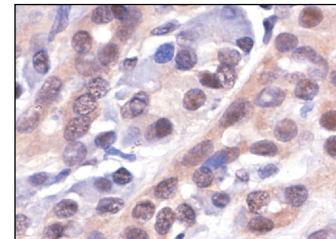
STORAGE

Store at 4° C, ****DO NOT FREEZE****. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

DATA



ICAD (FL-331): sc-9066. Western blot analysis of ICAD expression in non-transfected 293T: sc-117752 (A), mouse ICAD transfected 293T: sc-125479 (B) and HeLa (C) whole cell lysates.



ICAD (FL-331): sc-9066. Immunoperoxidase staining of formalin-fixed, paraffin-embedded human pancreas tissue showing nuclear and cytoplasmic staining.

SELECT PRODUCT CITATIONS

- Malina, H.Z., et al. 2001. Pathological apoptosis by xanthurenic acid, a tryptophan metabolite: activation of cell caspases but not cytoskeleton breakdown. *BMC Physiol.* 1: 7.
- Yu, F., et al. 2006. Involvement of BH3-only proapoptotic proteins in mitochondrial-dependent Phenoxodiol-induced apoptosis of human melanoma cells. *Anticancer Drugs* 17: 1151-1161.
- Ueda, E.K., et al. 2006. S179D prolactin primarily uses the extrinsic pathway and mitogen-activated protein kinase signaling to induce apoptosis in human endothelial cells. *Endocrinology* 147: 4627-4637.
- Kassi, E., et al. 2009. Ursolic acid triggers apoptosis and Bcl-2 downregulation in MCF-7 breast cancer cells. *Cancer Invest.* 27: 723-733.
- Marinov, M., et al. 2009. AKT/mTOR pathway activation and BCL-2 family proteins modulate the sensitivity of human small cell lung cancer cells to RAD001. *Clin. Cancer Res.* 15: 1277-1287.
- Ninios, Y.P., et al. 2010. Differential sensitivity of human leukemic cell lines to the histone deacetylase inhibitor, trichostatin A. *Leuk. Res.* 34: 786-792.
- Guerriero, J.L., et al. 2011. DNA alkylating therapy induces tumor regression through an HMGB1-mediated activation of innate immunity. *J. Immunol.* 186: 3517-3526.

RESEARCH USE

For research use only, not for use in diagnostic procedures.


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Try **ICAD (F-8): sc-17816** or **ICADL (C-4): sc-376044**, our highly recommended monoclonal alternatives to ICAD (FL-331).