SANTA CRUZ BIOTECHNOLOGY, INC.

FADD (M-19): sc-6036

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

In contrast to growth factors which promote cell proliferation, FAS ligand (FAS-L) and the tumor necrosis factors (TNFs) rapidly induce apoptosis. Cellular response to FAS-L and TNF is mediated by structurally related receptors containing a conserved "death domain" and belonging to the TNF receptor superfamily. TRADD, FADD and RIP are FAS/TNF-R1 interacting proteins that contain a death domain homologous region (DDH). TRADD (TNF-R1-associated death domain) and FADD (FAS-associated death domain) associate with the death domains of both FAS and TNF-R1 via their DDH regions. Overexpression of TRADD leads to NFκB activation and apoptosis in the absence of TNF. Overexpression of FADD causes apoptosis, which can be blocked by the bovine pox protein CrmA, suggesting that FADD lies upstream of ICE and possibly other serine proteases. The receptor interacting protein, RIP, associates with FAS exclusively via its DDH and this association is abrogated in lpr mutants. Unlike TRADD and FADD, RIP contains a putative amino-terminal kinase domain.

REFERENCES

1. Smith, C.A., et al. 1994. The TNF receptor superfamily of cellular and viral proteins: activation, costimulation and death. Cell 76: 959-962.
2. Nagata, S., et al. 1995. The FAS death factor. Science 267: 1449-1456.

CHROMOSOMAL LOCATION

Genetic locus: Fadd (mouse) mapping to 7 F5.

SOURCE

FADD (M-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of FADD of mouse origin.

PRODUCT

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

Blocking peptide available for competition studies, sc-6036 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

FADD (M-19) is recommended for detection of FADD of mouse and rat 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).

Suitable for use as control antibody for FADD siRNA (m): sc-35351, FADD shRNA Plasmid (m): sc-35351-SH and FADD shRNA (m) Lentiviral Particles: sc-35351-V.

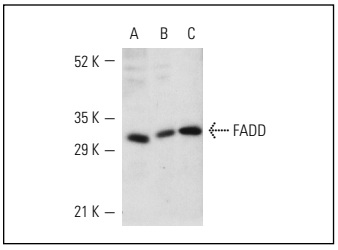
Molecular Weight of FADD: 27 kDa.

Positive Controls: mouse embryo extract: sc-364239, mouse lung extract: sc-2390 or NIH/3T3 whole cell lysate: sc-2210.

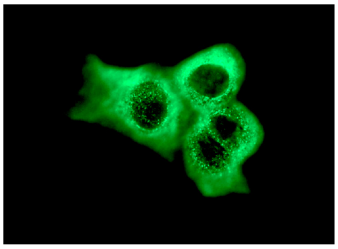
STORAGE

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

DATA



FADD (M-19): sc-6036. Western blot analysis of FADD expression in NIH/3T3 whole cell lysate (**A**) and mouse embryo (**B**) and lung (**C**) extracts.



FADD (M-19): sc-6036. Immunofluorescence staining of methanol-fixed NIH/3T3 cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Mueller, C.M., et al. 2000. Distinct molecular mechanisms of FAS resistance in murine B lymphoma cells. J. Immunol. 165: 1854-1862.
2. Panahian, N., et al. 2001. Site of injury-directed induction of Heme Oxygenase 1 and 2 in experimental spinal cord injury: differential functions in neuronal defense mechanisms? J. Neurochem. 76: 539-554.
3. Gilot, D., et al. 2005. A role for caspase-8 and c-FLIP$_L$ in proliferation and cell-cycle progression of primary hepatocytes. Carcinogenesis 26: 2086-2094.
4. Schile, A.J., et al. 2008. Regulation of apoptosis by XIAP ubiquitin-ligase activity. Genes Dev. 22: 2256-2266.
5. Tourneur, L., et al. 2008. Adenosine receptors control a new pathway of Fas-associated death domain protein expression regulation by secretion. J. Biol. Chem. 283: 17929-17938.
6. Ramakrishnan, P., et al. 2011. Sam68 is required for both NFκB activation and apoptosis signaling by the TNF receptor. Mol. Cell 43: 167-179.
7. Vanlangenakker, N., et al. 2011. cIAP1 and TAK1 protect cells from TNF-induced necrosis by preventing RIP1/RIP3-dependent reactive oxygen species production. Cell Death Differ. 18: 656-665.
8. Rodrigue-Gervais, I.G., et al. 2014. Cellular inhibitor of apoptosis protein cIAP2 protects against pulmonary tissue necrosis during influenza virus infection to promote host survival. Cell Host Microbe 15: 23-35.

RESEARCH USE

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

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.