SANTA CRUZ BIOTECHNOLOGY, INC.

p-FADD (Ser 194): sc-12439

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

In contrast to growth factors which promote cell proliferation, FAS ligand (FAS-L) and tumor necrosis factor (TNF) 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-RI interacting proteins that contain a death domain homologous region (DDH). TRADD (TNF-RI-associated death domain) and FADD (FAS-associated death domain) are proteins that associate with the death domains of both FAS and TNF-RI 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 (human) mapping to 11q13.3.

SOURCE

p-FADD (Ser 194) is available as either goat (sc-12439) or rabbit (sc-12439-R) affinity purified polyclonal antibody raised against a short amino acid sequence containing Ser 194 phosphorylated FADD 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.

Blocking peptide available for competition studies, sc-12439 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p-FADD (Ser 194) is recommended for detection of Ser 194 phosphorylated FADD of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500).

Suitable for use as control antibody for FADD siRNA (h): sc-35352, FADD shRNA Plasmid (h): sc-35352-SH and FADD shRNA (h) Lentiviral Particles: sc-35352-V.

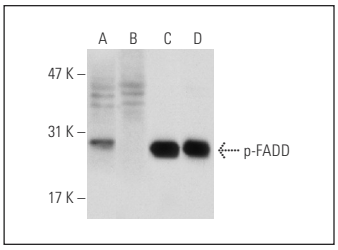
Molecular Weight of p-FADD: 30 kDa.

Positive Controls: A-431 whole cell lysate: sc-2201, HeLa whole cell lysate: sc-2200 or SW480 cell lysate: sc-2219.

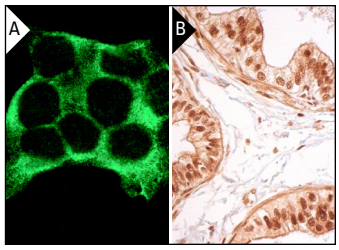
STORAGE

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

DATA



Western blot analysis of FADD phosphorylation in untreated (**A**,**C**), and lambda protein phosphatase treated (**B**,**D**) A-431 whole cell lysates. Antibodies tested include p-FADD (Ser 194): sc-12439 (**A**,**B**) and FADD (FD19): sc-56093 (**C**,**D**).



p-FADD (Ser 194): sc-12439. Immunofluorescence staining of methanol-fixed A-431 cells showing cytoplasmic staining (**A**). Immunoperoxidase staining of formalin fixed, paraffin-embedded human epididymis tissue showing nuclear staining of glandular cells (**B**).

SELECT PRODUCT CITATIONS

1. Uwagawa, T., et al. 2007. Mechanisms of synthetic serine protease inhibitor (FUT-175)-mediated cell death. Cancer 109: 2142-2153.
2. Garcia-Fuster, M.J., et al. 2008. Opioid receptor agonists enhance the phosphorylation state of Fas-associated death domain (FADD) protein in the rat brain: functional interactions with casein kinase Iα, $G_{\alpha i}$ proteins, and ERK1/2 signaling. Neuropharmacology 55: 886-899.
3. Ramos-Miguel, A., et al. 2009. Phosphorylation of FADD (Fas-associated death domain protein) at serine 194 is increased in the prefrontal cortex of opiate abusers: relation to mitogen activated protein kinase, phosphoprotein enriched in astrocytes of 15 kDa, and Akt signaling pathways involved in neuroplasticity. Neuroscience 161: 23-38.
4. Matsuda, N., et al. 2009. Silencing of fas-associated death domain protects mice from septic lung inflammation and apoptosis. Am. J. Respir. Crit. Care Med. 179: 806-815.
5. Couturier, J., et al. 2010. Interaction of double-stranded RNA-dependent protein kinase (PKR) with the death receptor signaling pathway in amyloid β (Aβ)-treated cells and in APPSLPS1 knock-in mice. J. Biol. Chem. 285: 1272-1282.
6. Gravina, G.L., et al. 2010. 5-Azacitidine restores and amplifies the bicalutamide response on preclinical models of androgen receptor expressing or deficient prostate tumors. Prostate 70: 1166-1178.
7. Alvaro-Bartolome, M., et al. 2011. Molecular adaptations of apoptotic pathways and signaling partners in the cerebral cortex of human cocaine addicts and cocaine-treated rats. Neuroscience 196: 1-15.
8. Gupta, K., et al. 2012. Green tea polyphenols induce p53-dependent and p53-independent apoptosis in prostate cancer cells through two distinct mechanisms. PLoS ONE 7: e52572.

RESEARCH USE

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