SANTA CRUZ BIOTECHNOLOGY, INC.

FAS (M-20): sc-716

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Cytotoxic T lymphocyte (CTL)-mediated cytotoxicity constitutes an important component of specific effector mechanisms in immuno-surveillance against virus-infected or transformed cells. Two mechanisms appear to account for this activity, one of which is the perforin-based process. Independently, a FAS-based mechanism involves the transducing molecule FAS (also designated APO-1) and its ligand (FAS-L). The human FAS protein is a cell surface glycoprotein that belongs to a family of receptors that includes CD40, nerve growth factor receptors and tumor necrosis factor receptors. The FAS antigen is expressed on a broad range of lymphoid cell lines, certain of which undergo apoptosis in response to treatment with antibody to FAS. These findings strongly imply that targeted cell death is potentially mediated by the intercellular interactions of FAS with its ligand or effectors, and that FAS may be critically involved in CTL-mediated cytotoxicity.

REFERENCES

1. Henkart, P.A. 1985. Mechanism of lymphocyte-mediated cytotoxicity. Annu. Rev. Immunol. 3: 31-58.
2. Young, J.D., et al. 1988. Perforin-dependent and independent pathways of cytotoxicity mediated by lymphocytes. Immunol. Rev. 103: 161-202.

CHROMOSOMAL LOCATION

Genetic locus: Fas (mouse) mapping to 19 C1.

SOURCE

FAS (M-20) is an affinity purified rabbit polyclonal antibody raised against a peptide mapping at the C-terminus of FAS 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-716 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

FAS (M-20) is recommended for detection of FAS of mouse and, to a lesser extent, 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for FAS siRNA (m): sc-29312, FAS shRNA Plasmid (m): sc-29312-SH and FAS shRNA (m) Lentiviral Particles: sc-29312-V.

Molecular Weight of FAS: 48 kDa.

Positive Controls: mouse thymus extract: sc-2406.

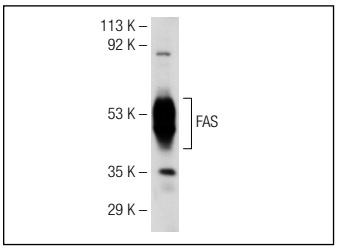
STORAGE

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

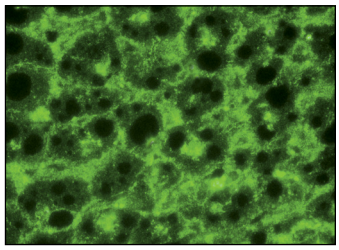
RESEARCH USE

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

DATA



FAS (M-20): sc-716. Western blot analysis of FAS expression in mouse thymus tissue extract.



FAS (M-20): sc-716. Immunofluorescence staining of normal mouse liver frozen section showing membrane staining.

SELECT PRODUCT CITATIONS

1. Micheau, O., et al. 1999. FAS ligand-independent, FADD-mediated activation of the FAS death pathway by anticancer drugs. J. Biol. Chem. 274: 7987-7992.
2. Yu, C.Z., et al. 2009. Neuroprotection against transient focal cerebral ischemia and oxygen-glucose deprivation by interference with GluR6-PSD95 protein interaction. Neurochem. Res. 34: 2008-2021.
3. Du, Y., et al. 2009. Neuroprotection of preconditioning against ischemic brain injury in rat hippocampus through inhibition of the assembly of GluR6-PSD95-mixed lineage kinase 3 signaling module via nuclear and non-nuclear pathways. Neuroscience 161: 370-380.
4. Li, T., et al. 2009. Inhibition of cerebral ischemia/reperfusion-induced injury by adenovirus expressed C-terminal amino acids of GluR6. Brain Res. 1300: 169-176.
5. Pan, J., et al. 2010. Small peptide inhibitor of JNKs protects against MPTP-induced nigral dopaminergic injury via inhibiting the JNK-signaling pathway. Lab. Invest. 90: 156-167.
6. Mandalari, G., et al. 2011. Natural almond skin reduced oxidative stress and inflammation in an experimental model of inflammatory bowel disease. Int. Immunopharmacol. 11: 915-924.
7. Tochitani, T., et al. 2011. 5-azacytidine, a chemotherapeutic drug, induces TRAIL-mediated apoptosis in mouse thymocytes *in vivo*. Exp. Toxicol. Pathol. 63: 237-242.
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9. Ferreira, N., 2012. Dietary curcumin counteracts extracellular transthyretin deposition: Insights on the mechanism of amyloid inhibition. Biochem. Biophys. Acta Rev. 1832: 39-45.
10. Ferreira, N., et al. 2012. Epigallocatechin-3-gallate as a potential therapeutic drug for TTR-related amyloidosis: *"in vivo"* evidence from FAP mice models. PLoS ONE 7: e29933.