

AIF (N-19): sc-9417

BACKGROUND

A key event in the apoptotic process is the opening of the mitochondrial permeability transition pore, an event that is regulated by Bcl-2 family proteins, resulting in the release of several proteins from the mitochondrial intermembrane space. Several of these proteins participate in apoptosis, including cytochrome c, procaspases 2, 3, and 9, and AIF (apoptosis-inducing factor). AIF was shown to cause DNA fragmentation and chromatin condensation, and to induce the release of cytochrome c and caspase-9 from mitochondria. Bcl-2 overexpression was shown to prevent the release of AIF from mitochondria, but not to block its apoptogenic activity.

REFERENCES

1. Zamzami, N., et al. 1996. Mitochondrial control of nuclear apoptosis. *J. Exp. Med.* 183: 1533-1544.
2. Susin, S.A., et al. 1996. Bcl-2 inhibits the mitochondrial release of an apoptogenic protease. *J. Exp. Med.* 184: 1331-1341.
3. Kluck, R.M., et al. 1997. The release of cytochrome c from mitochondria: a primary site for Bcl-2 regulation of apoptosis. *Science* 275: 1132-1136.
4. Green, D.R., et al. 1998. Mitochondria and apoptosis. *Science* 281: 1309-1312.
5. Mancini, M., et al. 1998. The caspase-3 precursor has a cytosolic and mitochondrial distribution: implications for apoptotic signaling. *J. Cell Biol.* 140: 1485-1495.
6. Susin, S.A., et al. 1999. Mitochondrial release of caspase-2 and -9 during the apoptotic process. *J. Exp. Med.* 189: 381-394.
7. Susin, S.A., et al. 1999. Molecular characterization of mitochondrial apoptosis-inducing factor. *Nature* 397: 441-446.

CHROMOSOMAL LOCATION

Genetic locus: AIFM1 (human) mapping to Xq26.1; Aifm1 (mouse) mapping to X A6.

SOURCE

AIF (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of AIF 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-9417 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

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.

APPLICATIONS

AIF (N-19) is recommended for detection of AIF of human and, to a lesser extent, mouse 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).

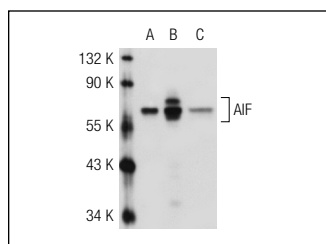
AIF (N-19) is also recommended for detection of AIF in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for AIF siRNA (h): sc-29193, AIF siRNA (m): sc-29194, AIF shRNA Plasmid (h): sc-29193-SH, AIF shRNA Plasmid (m): sc-29194-SH, AIF shRNA (h) Lentiviral Particles: sc-29193-V and AIF shRNA (m) Lentiviral Particles: sc-29194-V.

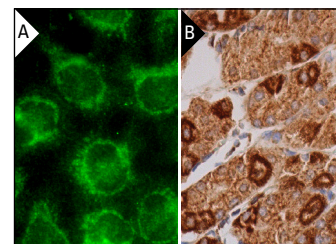
Molecular Weight of AIF: 57 kDa.

Positive Controls: AML-193 whole cell lysate: sc-364182, Jurkat whole cell lysate: sc-2204 or Hep G2 cell lysate: sc-2227.

DATA



AIF (N-19): sc-9417. Western blot analysis of AIF expression in non-transfected 293T: sc-117752 (A), mouse AIF transfected 293T: sc-118296 (B) and CTL-2 (C) whole cell lysates.



AIF (N-19): sc-9417. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human lower stomach tissue showing cytoplasmic staining of glandular cells (B).

SELECT PRODUCT CITATIONS

1. Carrozzo, R., et al. 2004. Maternally-inherited Leigh syndrome-related mutations bolster mitochondrial-mediated apoptosis. *J. Neurochem.* 90: 490-501.
2. Schoier, J., et al. 2006. *Chlamydia (Chlamydomonas) pneumoniae*-induced cell death in human coronary artery endothelial cells is caspase-independent and accompanied by subcellular translocations of Bax and apoptosis-inducing factor. *FEMS Immunol. Med. Microbiol.* 47: 207-216.
3. Son, Y.O., et al. 2006. Involvement of caspase activation and mitochondrial stress in trichostatin A-induced apoptosis of Burkitt's lymphoma cell line, Akata. *J. Cell. Biochem.* 99: 1420-1430.
4. Huang, T.Y., et al. 2012. Effect of sulforaphane on growth inhibition in human brain malignant glioma GBM 8401 cells by means of mitochondrial- and MEK/ERK-mediated apoptosis pathway. *Cell Biochem. Biophys.* 63: 247-259.