SANTA CRUZ BIOTECHNOLOGY, INC.

p-caspase-9 (Ser 196): sc-11755

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The Power to Question

BACKGROUND

A unique family of cysteine proteases has been described that differs in sequence, structure and substrate specificity from any previously described protease family. This family, designated Ced-3/caspase-1, is composed of caspase-1, caspase-2, caspase-3, caspase-4, caspase-6 and caspase-7 (also designated Mch3, ICE-LAP3 or CMH-1), caspase-9, caspase-10, and caspase-14. Ced-3/caspase-1 family members function as key components of the apoptotic machinery and act to destroy specific target proteins which are critical to cellular longevity. Caspase-3, caspase-7 and caspase-9, but not caspase-1, have been shown to cleave the nuclear protein PARP into an apoptotic fragment. Caspase-9 can be directly regulated by protein phosphorylation. The kinase Akt and p21-Ras, an Akt activator, induce phosphorylation of pro-caspase-9 in cells. Akt phosphorylates caspase-9 *in vitro* on Serine 196 and inhibits its protease activity. Caspase-14, also designated MICE (for mini-ICE), is highly expressed in embryonic tissues but appears to be absent from adult tissues. Procaspase-14 can be processed *in vitro* by caspase-8 and caspase-10 but not by other caspases.

REFERENCES

1. Duan, H., et al. 1996. ICE-LAP3, a novel mammalian homologue of the *Caenorhabditis elegans* cell death protein Ced-3 is activated during FAS- and tumor necrosis factor-induced apoptosis. J. Biol. Chem. 271: 1621-1625.
2. Fernandes-Alnemri, et al. 1996. *In vitro* activation of CPP32 and Mch3 by Mch4, a novel human apoptotic cysteine protease containing two FADD-like domains. Proc. Natl. Acad. Sci. USA 93: 7464-7469.
3. Duan, H., et al. 1996. ICE-LAP6, a novel member of the ICE/Ced-3 gene family, is activated by the cytotoxic T cell protease granzyme B. J. Biol. Chem. 271: 16720-16724.
4. Casciola-Rosen, L., et al. 1996. Apopain/CPP32 cleaves proteins that are essential for cellular repair: a fundamental principle of apoptotic death. J. Exp. Med. 183: 1957-1964.
5. Cardone, M.H., et al. 1998. Regulation of cell death protease caspase-9 by phosphorylation. Science 282: 1318-1321.
6. Hu, S., et al. 1998. Caspase-14 is a novel developmentally regulated protease. J. Biol. Chem. 273: 29648-29653.
7. Van de Craen, M., et al. 1998. Identification of a new caspase homologue: caspase-14. Cell Death Differ. 5: 838-846.
8. Ahmad, M., et al. 1998. Identification and characterization of murine caspase-14, a new member of the caspase family. Cancer Res. 58: 5201-5205.

CHROMOSOMAL LOCATION

Genetic locus: CASP9 (human) mapping to 1p36.21.

SOURCE

p-caspase-9 (Ser 196) is available as either goat (sc-11755) or rabbit (sc-11755-R) polyclonal affinity purified antibody raised against a short amino acid sequence containing phosphorylated Ser 196 of caspase-9 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-11755 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

p-caspase-9 (Ser 196) is recommended for detection of Ser 196 phosphorylated caspase-9 of 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for caspase-9 siRNA (h): sc-29931, caspase-9 shRNA Plasmid (h): sc-29931-SH and caspase-9 shRNA (h) Lentiviral Particles: sc-29931-V.

SELECT PRODUCT CITATIONS

1. Takeuchi, K., et al. 2004. Suppression of adriamycin-induced apoptosis by sustained activation of the phosphatidylinositol-3'-OH kinase-Akt pathway. J. Biol. Chem. 279: 892-900.
2. Dai. Y., et al. 2005. Farnesyltransferase inhibitors interact synergistically with the Chk1 inhibitor UCN-01 to induce apoptosis in human leukemia cells through interruption of both Akt and MEK/ERK pathways and activation of SEK1/JNK. Blood 105: 1706-1716.
3. Prueitt, R.L., et al. 2007. Inflammation and IGF-I activate the Akt pathway in breast cancer. Int. J. Cancer 120: 796-805.
4. Shin, Y.K., et al. 2007. SH3 binding motif 1 in influenza A virus NS1 protein is essential for PI3K/Akt signaling pathway activation. J. Virol. 81: 12730-1273
5. Glynn, S.A., et al. 2010. COX-2 activation is associated with Akt phosphorylation and poor survival in ER-negative, HER2-positive breast cancer. BMC Cancer 10: 626.
6. Xu, X.X., et al. 2010. Apoptosis of stomach cancer cell SGC-7901 and regulation of Akt signaling way induced by bovine lactoferrin. J. Dairy Sci. 93: 2344-2350.

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.

PROTOCOLS

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