SANTA CRUZ BIOTECHNOLOGY, INC.

caspase-3 p17 (S-19): sc-22139

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Caspase-3, also known as apopain, SCA-1, Yama and CPP32, is an aspartate-specific cysteine protease that belongs to the ICE subfamily of caspases. Caspase-3 is expressed in cells as an inactive precursor from which the p17 and p11 subunits of the mature caspase-3 are proteolytically generated during apoptosis. The caspase-3 precursor is first cleaved at Asp 175-Ser 176 to produce the p11 subunit and the p20 peptide. Subsequently, the p20 peptide is cleaved at Asp 28-Ser 29 to generate the mature p17 subunit. The active caspase-3 enzyme is a heterodimer composed of two p17 and two p11 subunits. At the onset of apoptosis, caspase-3 proteolytically cleaves PARP at a Asp 216-Gly 217 bond. During the execution of the apoptotic cascade, activated caspase-3 releases SREBP from the membrane of the ER in a proteolytic reaction that is distinct from their normal sterol-dependent activation. Caspase-3 cleaves and activates SREBPs between the basic helix-loop-helix leucine zipper domain and the membrane attachment domain. Caspase-3 also cleaves and activates caspase-6, -7 and -9. The human caspase-3 gene encodes a cytoplasmic protein that is highly expressed in lung, spleen, heart, liver, kidney and cells of the immune system.

REFERENCES

1. Nicholson, D., et al. 1995. Identification and inhibition of the ICE/CED-3 protease necessary for mammalian apoptosis. Nature 37: 37-43.
2. Cohen, G.M. 1997. Caspases: the executioners of apoptosis. Biochem. J. 326: 1-16.
3. Higgins, M.E., et al. 2001. Apoptosis-induced release of mature sterol regulatory element-binding proteins activates sterol-responsive genes. J. Lipid Res. 42: 1939-1946.
4. Vallender, E.J., et al. 2006. A primate-specific acceleration in the evolution of the caspase-dependent apoptosis pathway. Hum. Mol. Genet. 15: 3034-3040.

CHROMOSOMAL LOCATION

Genetic locus: CASP3 (human) mapping to 4q35.1; Casp3 (mouse) mapping to 8 B1.1.

SOURCE

caspase-3 p17 (S-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of caspase-3 p17 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-22139 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.

APPLICATIONS

caspase-3 p17 (S-19) is recommended for detection of caspase-3 p17 subunit and full length procaspase-3 of mouse, rat and 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).

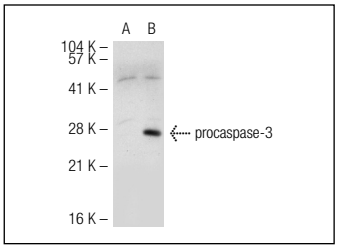
caspase-3 p17 (S-19) is also recommended for detection of caspase-3 p17 subunit and full length procaspase-3 in additional species, including equine, canine, bovine, porcine and feline.

Molecular Weight of procaspase-3: 32 kDa.

Molecular Weight of caspase-3 p17: 17 kDa.

Positive Controls: caspase-3 (h): 293T Lysate: sc-113427, Jurkat whole cell lysate: sc-2204 or CCRF-HSB-2 cell lysate: sc-2265.

DATA



caspase-3 p17 (S-19): sc-22139. Western blot analysis of caspase-3 expression in non-transfected: sc-117752 (**A**) and human caspase-3 transfected: sc-113427 (**B**) 293T whole cell lysates.

SELECT PRODUCT CITATIONS

1. Almonte-Becerril, M., et al. 2010. Cell death of chondrocytes is a combination between apoptosis and autophagy during the pathogenesis of Osteoarthritis within an experimental model. Apoptosis 15: 631-638.
2. Netsawang, J., et al. 2010. Nuclear localization of dengue virus capsid protein is required for DAXX interaction and apoptosis. Virus Res. 147: 275-283.
3. Humanes, B., et al. 2012. Cilastatin protects against cisplatin-induced nephrotoxicity without compromising its anticancer efficiency in rats. Kidney Int. 82: 652-663.

RESEARCH USE

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