SANTA CRUZ BIOTECHNOLOGY, INC.

SOD-1 (N-19): sc-8636

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Cu-Zn superoxide dismutase-1 (SOD-1) is a well characterized cytosolic scavenger of oxygen free radicals that requires copper and zinc binding to potentiate its enzymatic activity. Enzymatically, SOD-1 facilitates the dismutation of oxygen radicals to hydrogen peroxide, and it also catalyzes prooxidant reactions, which include the peroxidase activity and hydroxyl radical generating activity. SOD-1 is ubiquitously expressed in somatic cells and functions as a homodimer. Defects in the gene encoding SOD-1 have been implicated in the progression of neurological diseases, including amyotrophic lateral sclerosis (ALS), a neurodegenerative disease characterized by the loss of spinal motor neurons, Downs syndrome and Alzheimer's disease. In familial ALS, several mutations in SOD-1 predominate, and they result in the loss of zinc binding and the loss of scavenging activity of SOD-1 and correlate with an increase in neurotoxicity and motor neuron death.

REFERENCES

1. Levanon, D., et al. 1985. Architecture and anatomy of the chromosomal locus in human chromosome 21 encoding the Cu/Zn superoxide dismutase. EMBO J. 4: 77-84.
2. Bewley, G.C. 1988. cDNA and deduced amino acid sequence of murine Cu-Zn superoxide dismutase. Nucleic Acids Res. 16: 2728.

CHROMOSOMAL LOCATION

Genetic locus: SOD1 (human) mapping to 21q22.11.

SOURCE

SOD-1 (N-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the N-terminus of SOD-1 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-8636 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

SOD-1 (N-19) is recommended for detection of SOD-1 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), 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).

Suitable for use as control antibody for SOD-1 siRNA (h): sc-36523, SOD-1 shRNA Plasmid (h): sc-36523-SH and SOD-1 shRNA (h) Lentiviral Particles: sc-36523-V.

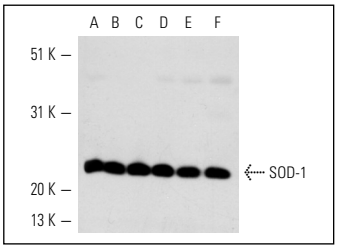
Molecular Weight of SOD-1: 23 kDa.

Positive Controls: HeLa whole cell lysate: sc-2200, Jurkat whole cell lysate: sc-2204 or Hs68 cell lysate: sc-2230.

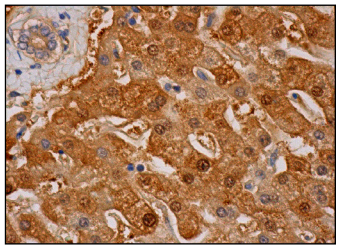
STORAGE

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

DATA



SOD-1 (N-19): sc-8636. Western blot analysis of SOD-1 expression in HeLa (**A**), Jurkat (**B**), Hep G2 (**C**), DU 145 (**D**), Hs68 (**E**) and CCD-1064Sk (**F**) whole cell lysates.



SOD-1 (N-19): sc-8636. Immunoperoxidase staining of formalin fixed, paraffin-embedded human liver tissue showing cytoplasmic and nuclear staining of hepatocytes.

SELECT PRODUCT CITATIONS

1. Raoul, C., et al. 2005. Lentiviral-mediated silencing of SOD-1 through RNA interference retards disease onset and progression in a mouse model of ALS. Nat. Med. 11: 423-428.
2. Yang, Y.S., et al. 2009. Reticulon-4A (Nogo-A) redistributes protein disulfide isomerase to protect mice from SOD1-dependent amyotrophic lateral sclerosis. J. Neurosci. 29: 13850-13859.
3. Towne, C. and Aebischer, P. 2009. Lentiviral and adeno-associated vector-based therapy for motor neuron disease through RNAi. Methods Mol. Biol. 555: 87-108.
4. Higashi, S., et al. 2010. TDP-43 physically interacts with amyotrophic lateral sclerosis-linked mutant CuZn superoxide dismutase. Neurochem. Int. 57: 906-913.
5. Rincheval, V., et al. 2012. Differential effects of Bcl-2 and caspases on mitochondrial permeabilization during endogenous or exogenous reactive oxygen species-induced cell death: a comparative study of H_2O_2, paraquat, t-BHP, etoposide and TNF-α-induced cell death. Cell Biol. Toxicol. 28: 239-253.
6. Van Hoecke, A., et al. 2012. EPHA4 is a disease modifier of amyotrophic lateral sclerosis in animal models and in humans. Nat. Med. 18: 1418-1422.

RESEARCH USE

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