

SOD-1 (FL-154): sc-11407

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, Down 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.

CHROMOSOMAL LOCATION

Genetic locus: SOD1 (human) mapping to 21q22.11; Sod1 (mouse) mapping to 16 C3.3.

SOURCE

SOD-1 (FL-154) is a rabbit polyclonal antibody raised against amino acids 1-154 representing full length 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.

APPLICATIONS

SOD-1 (FL-154) is recommended for detection of SOD-1 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), 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 siRNA (m): sc-36522, SOD-1 shRNA Plasmid (h): sc-36523-SH, SOD-1 shRNA Plasmid (m): sc-36522-SH, SOD-1 shRNA (h) Lentiviral Particles: sc-36523-V and SOD-1 shRNA (m) Lentiviral Particles: sc-36522-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.

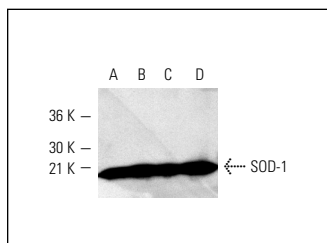
PROTOCOLS

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

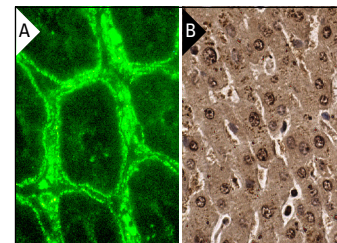
RESEARCH USE

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

DATA



SOD-1 (FL-154): sc-11407. Western blot analysis of SOD-1 expression in HeLa (A), Jurkat (B), Hs68 (C) and DU 145 (D) whole cell lysates.



SOD-1 (FL-154): sc-11407. Immunofluorescence staining of normal mouse intestine frozen section showing cytoplasmic staining (A). Immunoperoxidase staining of formalin fixed, paraffin-embedded human liver tissue showing nuclear and cytoplasmic staining of hepatocytes (B).

SELECT PRODUCT CITATIONS

- Golab, J., et al. 2003. Antitumor effects of photodynamic therapy are potentiated by 2-methoxyestradiol. A superoxide dismutase inhibitor. *J. Biol. Chem.* 278: 407-414.
- Bertinato, J., et al. 2003. Copper modulates the degradation of copper chaperone for Cu,Zn superoxide dismutase by the 26 S proteasome. *J. Biol. Chem.* 278: 35071-35078.
- Blander, G., et al. 2003. Superoxide dismutase 1 knock-down induces senescence in human fibroblasts. *J. Biol. Chem.* 278: 38966-38969.
- Bhaskaran, N., et al. 2012. Chamomile confers protection against hydrogen peroxide-induced toxicity through activation of Nrf2-mediated defense response. *Phyther. Res.* 27: 118-125.
- Baluchamy, S., et al. 2012. Reactive oxygen species mediated tissue damage in high energy proton irradiated mouse brain. *Mol. Cell. Biochem.* 360: 189-195.
- Cao, J.Y., et al. 2012. Changes in the nasopharyngeal carcinoma nuclear proteome induced by the EBNA1 protein of Epstein-Barr virus reveal potential roles for EBNA1 in metastasis and oxidative stress responses. *J. Virol.* 86: 382-394.
- St-Louis, R., et al. 2012. Reactive oxygen species are required for the hypothalamic osmoregulatory response. *Endocrinology* 153: 1317-1329.


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