SANTA CRUZ BIOTECHNOLOGY, INC.

SOD-2 (24): sc-130346

BACKGROUND

The superoxide dismutase family is composed of three metalloenzymes (SOD-1, SOD-2 and SOD-3) that catalyze the oxido-reduction of reactive oxygen species (ROS) such as superoxide anion. The SOD-2 precursor is a 222 amino acid protein that is encoded by nuclear chromatin, synthesized in the cytosol and imported posttranslationally into the mitochondrial matrix. Unlike SOD-1, which is a homodimeric cytosolic Cu-Zn enzyme, SOD-2 is a homotetrameric manganese enzyme (also known as MnSOD) that functions in the mitochondrion. ROS are implicated in a wide range of degenerative processes, including Alzheimer's disease, Parkinson's disease and ischemic heart disease. Homozygous mutant mice, which lack SOD-2, exhibit dilated cardiomyopathy, accumulation of lipid in liver and skeletal muscle, metabolic acidosis, oxidative DNA damage and respiratory chain deficiencies in heart and skeletal muscle. Polymorphisms in the SOD-2 gene have also been implicated in nonfamilial, idiopathic, dilated cardiomyopathy in humans.

REFERENCES

1. Wispé, J.R., et al. 1989. Synthesis and processing of the precursor for human mangano-superoxide dismutase. Biochem. Biophys. Acta 994: 30-36.
2. Nishi, H., et al. 1995. DNA typing of HLA class II genes in Japanese patients with dilated cardiomyopathy. J. Mol. Cell. Cardiol. 27: 2385-2392.
3. Li, Y., et al. 1995. Dilated cardiomyopathy and neonatal lethality in mutant mice lacking manganese superoxide dismutase. Nat. Genet. 11: 376-381.
4. Borgstahl, G.E., et al. 1996. Human mitochondrial manganese superoxide dismutase polymorphic variant Ile58Thr reduces activity by destabilizing the tetrameric interface. Biochemistry 35: 4287-4297.
5. Hsieh, Y., et al. 1998. Probing the active site of human manganese superoxide dismutase: the role of glutamine 143. Biochemistry 37: 4731-4739.
6. Melov, S., et al. 1998. A novel neurological phenotype in mice lacking mitochondrial manganese superoxide dismutase. Nat. Genet. 18: 159-163.
7. Melov, S., et al. 1999. Mitochondrial disease in superoxide disumtase 2 mutant mice. Proc. Natl. Acad. Sci. USA 96: 846-851.
8. Hiroi, S., et al. 1999. Polymorphisms in the SOD-2 and HLA-DRB1 genes are associated with nonfamilial idopathic dilated cardiomyopathy in Japanese. Biochem. Biophys. Res. Commun. 261: 332-339.

CHROMOSOMAL LOCATION

Genetic locus: SOD2 (human) mapping to 6q25.3.

SOURCE

SOD-2 (24) is a mouse monoclonal antibody raised against recombinant SOD-2 of human origin.

PRODUCT

Each vial contains 200 µg IgG_1 kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

APPLICATIONS

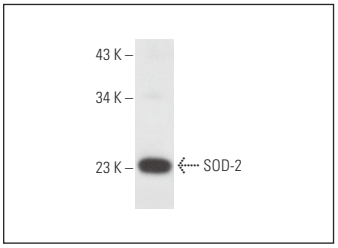
SOD-2 (24) is recommended for detection of SOD-2 of human origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

Suitable for use as control antibody for SOD-2 siRNA (h): sc-41655, SOD-2 shRNA Plasmid (h): sc-41655-SH and SOD-2 shRNA (h) Lentiviral Particles: sc-41655-V.

Molecular Weight of SOD-2: 25 kDa.

Positive Controls: DU 145 cell lysate: sc-2268, HISM cell lysate: sc-2229 or SK-N-MC cell lysate: sc-2237.

DATA



SOD-2 (24): sc-130346. Western blot analysis of SOD-2 expression in DU 145 whole cell lysate.

SELECT PRODUCT CITATIONS

1. Dahiya, S., et al. 2014. CCAAT enhancer binding protein and nuclear factor of activated T cells regulate HIV-1 LTR via a novel conserved downstream site in cells of the monocyte-macrophage lineage. PLoS ONE 9: e88116.
2. Sahlender, B., et al. 2022. Superoxide dismutase and catalase significantly improve the osteogenic differentiation potential of osteogenetically compromised human adipose tissue-derived stromal cells *in vitro*. Stem Cell Res. 60: 102708.

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.