Best Tools for immunology Research

SOD-1 Monoclonal Antibody

Catalog No :	YM0590
Reactivity :	Human;Mouse
Applications :	WB;IF;FCM;ELISA
Target :	SOD-1
Fields :	>>Peroxisome;>>Longevity regulating pathway - multiple species;>>Parkinson disease;>>Amyotrophic lateral sclerosis;>>Huntington disease;>>Prion disease;>>Pathways of neurodegeneration - multiple diseases;>>Chemical carcinogenesis - reactive oxygen species
Gene Name :	SOD1
Protein Name :	Superoxide dismutase [Cu-Zn]
Human Gene Id :	6647
Human Swiss Prot No :	P00441
Mouse Gene Id :	20655
Mouse Swiss Prot No :	P08228
Immunogen :	Purified recombinant fragment of human SOD-1 expressed in E. Coli.
Specificity :	SOD-1 Monoclonal Antibody detects endogenous levels of SOD-1 protein.
Formulation :	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source :	Monoclonal, Mouse
Dilution :	WB 1:500 - 1:2000. IF 1:200 - 1:1000. Flow cytometry: 1:200 - 1:400. ELISA: 1:10000. Not yet tested in other applications.
Purification :	Affinity purification

Storage Stability : -15°C to -25°C/1 year(Do not lower than -25°C)

Molecularweight : 16kD

Cell Pathway : Amyotrophic lateral sclerosis (ALS);Huntington's disease;Prion diseases;

P References :
1. Apoptosis. 2005 May;10(3):499-502.
2. Hum Mol Genet. 2008 Nov 1;17(21):3303-17.

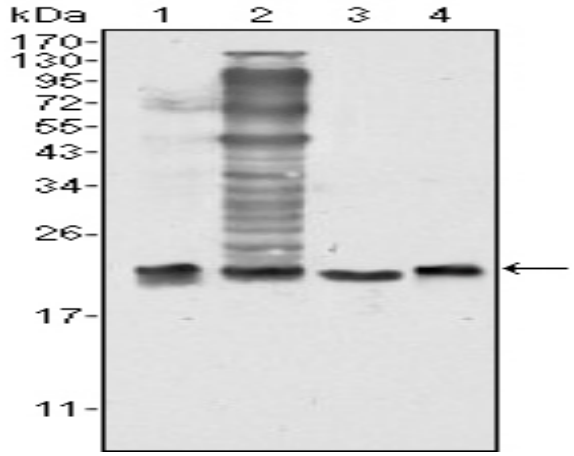
Background : The protein encoded by this gene binds copper and zinc ions and is one of two isozymes responsible for destroying free superoxide radicals in the body. The encoded isozyme is a soluble cytoplasmic protein, acting as a homodimer to convert naturally-occuring but harmful superoxide radicals to molecular oxygen and hydrogen peroxide. The other isozyme is a mitochondrial protein. Mutations in this gene have been implicated as causes of familial amyotrophic lateral sclerosis. Rare transcript variants have been reported for this gene. [provided by RefSeq, Jul 2008],

Function : catalytic activity:2 superoxide + 2 H(+) = O(2) + H(2)O(2).,cofactor:Binds 1 copper ion per subunit.,cofactor:Binds 1 zinc ion per subunit.,disease:Defects in SOD1 are the cause of amyotrophic lateral sclerosis type 1 (ALS1) [MIM:105400]. ALS1 is a familial form of amyotrophic lateral sclerosis, a neurodegenerative disorder affecting upper and lower motor neurons and resulting in fatal paralysis. Sensory abnormalities are absent. Death usually occurs within 2 to 5 years. The etiology of amyotrophic lateral sclerosis is likely to be multifactorial, involving both genetic and environmental factors. The disease is inherited in 5-10% of cases leading to familial forms.,function:Destroys radicals which are normally produced within the cells and which are toxic to biological systems.,miscellaneous:The protein (both wild-type and ALS1 variants) has a tendency to form fibrillar aggregates in the

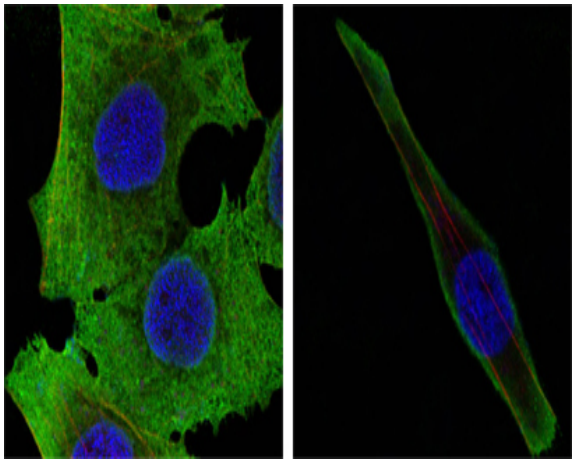
Subcellular Location : Cytoplasm . Mitochondrion . Nucleus . Predominantly cytoplasmic; the pathogenic variants ALS1 Arg-86 and Ala-94 gradually aggregates and accumulates in mitochondria. .

Expression : Colon,Fetal brain cortex,Placenta,

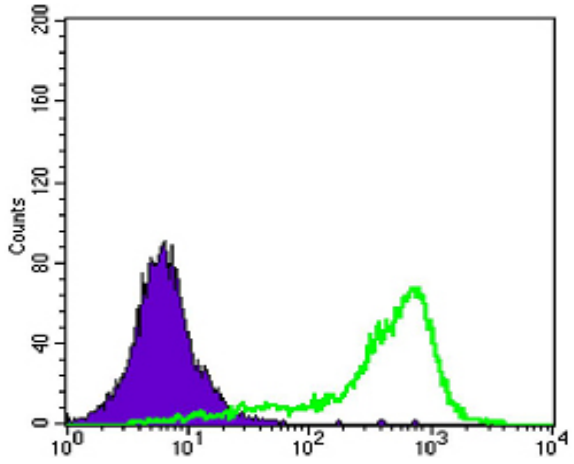
Products Images



Western Blot analysis using SOD-1 Monoclonal Antibody against HeLa (1), NIH/3T3 (2), A549 (3) and A431 (4) cell lysate.



Confocal immunofluorescence analysis of PANC-1 (left) and SKBR-3 (right) cells using SOD-1 Monoclonal Antibody (green). Red: Actin filaments have been labeled with DY-554 phalloidin. Blue: DRAQ5 fluorescent DNA dye.



Flow cytometric analysis of A431 cells using SOD-1 Monoclonal Antibody (green) and negative control (purple).