

SOD-1 Polyclonal Antibody

Catalog No :	YT4364
Reactivity :	Human;Mouse;Rat
Applications :	WB;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
Rat Gene Id :	24786
Rat Swiss Prot No :	P07632
Immunogen :	The antiserum was produced against synthesized peptide derived from human SOD-1. AA range:36-85
Specificity :	SOD-1 Polyclonal Antibody detects endogenous levels of SOD-1 protein.
Formulation :	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% sodium azide.
Source :	Polyclonal, Rabbit,IgG

Dilution :	WB 1:500 - 1:2000. ELISA: 1:10000. Not yet tested in other applications.
Purification :	The antibody was affinity-purified from rabbit antiserum by affinity-chromatography using epitope-specific immunogen.
Concentration :	1 mg/ml
Storage Stability :	-15°C to -25°C/1 year(Do not lower than -25°C)
Observed Band :	18kD
Cell Pathway :	Amyotrophic lateral sclerosis (ALS);Huntington's disease;Prion diseases;
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-occurring 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,

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