Product Name: SOD-1 Rabbit Polyclonal Antibody
Catalog #: APRab18097

Summary

Production Name	SOD-1 Rabbit Polyclonal Antibody
Description	Rabbit Polyclonal Antibody
Host	Rabbit
Application	WB,ELISA
Reactivity	Human,Mouse,Rat

Performance

Conjugation	Unconjugated
Modification	Unmodified
Isotype	IgG
Clonality	Polyclonal
Form	Liquid
Storage	Store at 4°C short term. Aliquot and store at -20°C long term. Avoid freeze/thaw cycles.
Buffer	Liquid in PBS containing 50% glycerol, 0.5% BSA and 0.02% New type preservative N.
Purification	Affinity purification

Immunogen

Gene Name	SOD1
Alternative Names	SOD1; Superoxide dismutase [Cu-Zn]; Superoxide dismutase 1; hSod1
Gene ID	6647.0
SwissProt ID	P00441.The antiserum was produced against synthesized peptide derived from human SOD-1. AA range:36-85

Application

Dilution Ratio	WB 1:500 - 1:2000. ELISA: 1:10000
Molecular Weight	18kD

Background

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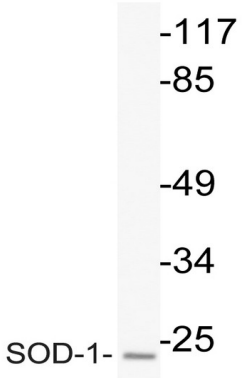
The protein encoded by this gene binds copper and zinc ions and is one of two isozymes responsible for destroying free superoxide radicals in the body. The encoded isozyme is a soluble cytoplasmic protein, acting as a homodimer to convert naturally-occuring but harmful superoxide radicals to molecular 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],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 absence of the intramolecular disulfide bond or of bound zinc ions. These aggregates may have cytotoxic effects. Zinc binding promotes dimerization and stabilizes the native form.,online information:ALS genetic mutations db,online information:Superoxide dismutase entry,PTM:Unlike wild-type protein, the pathogenics variants ALS1 Arg-38, Arg-47, Arg-86 and Ala-94 are polyubiquitinated by RNF19A; which leads to their proteasomal degradation.,similarity:Belongs to the Cu-Zn superoxide dismutase family.,subunit:Homodimer. The pathogenics variants ALS1 Arg-38, Arg-47, Arg-86 and Ala-94 interact with RNF19A, whereas wild-type protein does not.,

Research Area

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

Image Data



Western blot analysis of lysate from Jurkat cells, using SOD-1 antibody.

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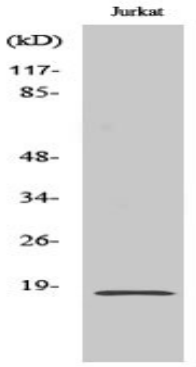

Western Blot analysis of various cells using SOD-1 Polyclonal Antibody diluted at 1： 1000

Note

For research use only.