



Product name:	SOD-1 Rabbit Polyclonal Antibody
Cat number:	ABN18097
Conjugate:	Unconjugated
Size:	100 ug
Concentration:	1mg/ml
Host:	Rabbit
Isotype:	IgG
Immunogen:	The antiserum was produced against synthesized peptide derived from human SOD-1. AA range:36-85
Reactivity:	Human,Mouse,Rat
Applications:	WB 1:500-1:2000,ELISA 1:5000-1:20000
Molecular Weight:	~15kDa
Purification:	Affinity purification
Form:	Liquid
Buffer:	PBS, 50% glycerol, 0.05% Proclin 300, 0.05% protective protein.
Storage:	Aliquot and store at -20°C (valid for 12 months). Avoid freeze/thaw cycles.
Synonyms:	SOD1; Superoxide dismutase [Cu-Zn]; Superoxide dismutase 1; hSod1
Source:	Rabbit
Background:	<p>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],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</p>

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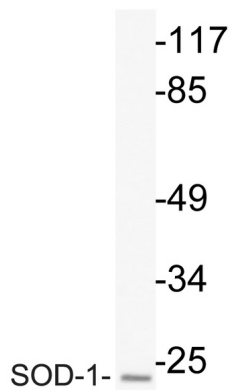
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intramolecular disulfide bond or of bound zinc ions. These aggregates may have cytotoxic effects. Zinc binding promotes dimerization and stabilizes the native form.,online information:ALS genetic mutations db,online information:Superoxide dismutase 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.

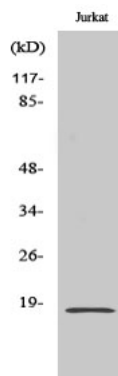
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Web-site: <https://immunologicalsciences.com> - E-mail: info@immunologicalsciences.com



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



Western Blot analysis of various cells using SOD-1 Polyclonal Antibody diluted at 11000

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