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Superoxide Dismutase 1 (SOD-1) Antibody

Rabbit Anti Human Polyclonal Antibody
Catalog # ABV11695

Product Information

Application	WB
Primary Accession	P00441
Reactivity	Human, Mouse, Rat
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Calculated MW	15936

Additional Information

Gene ID	6647
Positive Control	Western Blot: 3T3 cell lysate, rat kidney lysate
Application & Usage	Western blot: 1:200
Other Names	Superoxide Dismutase 1, SODC
Target/Specificity	SOD1
Antibody Form	Liquid
Appearance	Colorless liquid
Formulation	30 □g (0.5 mg/ml) of antibody in PBS containing 0.01 % BSA, 0.01 % thimerosal, and 50 % glycerol, pH 7.2
Handling	The antibody solution should be gently mixed before use.
Reconstitution & Storage	-20 °C
Background Descriptions	
Precautions	Superoxide Dismutase 1 (SOD-1) Antibody is for research use only and not for use in diagnostic or therapeutic procedures.

Protein Information

Name	SOD1 (HGNC:11179)
Function	Destroys radicals which are normally produced within the cells and which are toxic to biological systems (PubMed:18948262, PubMed:24140062). Catalyzes the oxidation of hydrogen sulfide (H2S) to sulfate, playing an important role in detoxifying H2S and limiting the accumulation of reactive

	sulfur species (RSS) such as persulfides and polysulfides (PubMed:36630448).
Cellular Location	Cytoplasm. Nucleus. Note=Predominantly cytoplasmic; the pathogenic variants ALS1 Arg-86 and Ala-94 gradually aggregates and accumulates in mitochondria.

Background

Superoxide Dismutase (SOD) is an oxidoreductase that catalyzes the reaction between superoxide anions and hydrogen to yield molecular oxygen and hydrogen peroxide. The enzyme protects the cell against dangerous levels of superoxide. It belongs to the Cu-Zn superoxide dismutase family. It binds copper and zinc ions. 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. SOD1 destroys radicals which are normally produced within the cells and which are toxic to biological systems. Defects in SOD1 are the cause of amyotrophic lateral sclerosis type 1 (ALS1). 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.