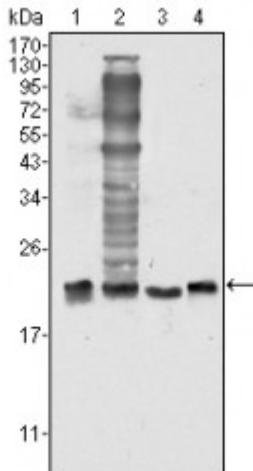


## Anti-SOD-1 antibody



<b>Description</b>	Mouse monoclonal to SOD-1.
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<b>Model</b>	STJ98392
<b>Host</b>	Mouse
<b>Reactivity</b>	Human, Mouse
<b>Applications</b>	ELISA, FC, IF, WB
<b>Immunogen</b>	Purified recombinant fragment of human SOD-1 expressed in E. Coli.
<b>Gene ID</b>	<a href="#">6647</a>
<b>Gene Symbol</b>	<a href="#">SOD1</a>
<b>Dilution range</b>	WB 1:500-1:2000IF 1:200-1:1000FC 1:200-1:400ELISA 1:10000
<b>Specificity</b>	SOD-1 Monoclonal Antibody detects endogenous levels of SOD-1 protein.
<b>Purification</b>	Affinity purification
<b>Clone ID</b>	6F5
<b>Note</b>	For Research Use Only (RUO).
<b>Protein Name</b>	Superoxide dismutase Cu-Zn Superoxide dismutase 1 hSod1
<b>Clonality</b>	Monoclonal
<b>Conjugation</b>	Unconjugated
<b>Isotype</b>	IgG1
<b>Formulation</b>	Ascitic fluid containing 0.03% sodium azide.

<b>Storage Instruction</b>	Store at -20°C, and avoid repeat freeze-thaw cycles.
<b>Database Links</b>	<a href="#">HGNC:11179</a> <a href="#">OMIM:105400</a>
<b>Alternative Names</b>	Superoxide dismutase Cu-Zn Superoxide dismutase 1 hSod1
<b>Function</b>	Destroys radicals which are normally produced within the cells and which are toxic to biological systems.
<b>Cellular Localization</b>	Cytoplasm Mitochondrion Nucleus. Predominantly cytoplasmic. the pathogenic variants ALS1 Arg-86 and Ala-94 gradually aggregates and accumulates in mitochondria.
<b>Post-translational Modifications</b>	Unlike wild-type protein, the pathogenic variants ALS1 Arg-38, Arg-47, Arg-86 and Ala-94 are polyubiquitinated by RNF19A leading to their proteasomal degradation. The pathogenic variants ALS1 Arg-86 and Ala-94 are ubiquitinated by MARCH5 leading to their proteasomal degradation. The ditryptophan cross-link at Trp-33 is responsible for the non-disulfide-linked homodimerization. Such modification might only occur in extreme conditions and additional experimental evidence is required. Palmitoylation helps nuclear targeting and decreases catalytic activity. Succinylation, adjacent to copper catalytic site, probably inhibits activity. Desuccinylation by SIRT5 enhances activity.

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