# **Product Name: Recombinant Human SOD1 (N-6His)**

Catalog #: PEH1546



### **Summary**

SOD1/Cu-Zn SOD/Cu/Zn Superoxide Dismutase Name

**Purity** Greater than 95% as determined by reducing SDS-PAGE

**Endotoxin level** <1 EU/µg as determined by LAL test.

Construction Recombinant Human Superoxide Dismutase [Cu-Zn] is produced by our E.coli

expression system and the target gene encoding Met1-Gln154 is expressed

with a 6His tag at the N-terminus.

Accession # P00441

Host E.coli

**Species** Human

**Predicted Molecular Mass** 18.1 KDa

**Formulation** Supplied as a 0.2 µm filtered solution of 20mM PB, 150mM NaCl, pH 7.2.

**Shipping** The product is shipped on dry ice/polar packs. Upon receipt, store it immediately

at the temperature listed below.

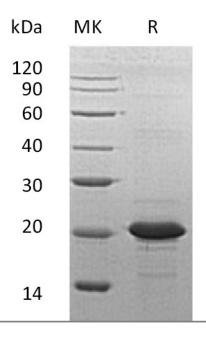
Stability&Storage Store at ≤-70°C, stable for 6 months after receipt. Store at ≤-70°C, stable for 3

months under sterile conditions after opening. Please minimize freeze-thaw

cycles.

Reconstitution

### **SDS-PAGE** image



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#### **Alternative Names**

Superoxide Dismutase [Cu-Zn]; Superoxide Dismutase 1; hSod1; SOD1

### **Background**

Superoxide Dismutase [Cu-Zn] (SOD1) is a soluble cytoplasmic and mitochondrial intermembrane space protein that belongs to the Cu-Zn superoxide dismutase family. SOD1 binds copper and zinc ions and is one of three isozymes responsible for destroying free superoxide radicals in the body. SOD1 neutralizes supercharged oxygen molecules, which can damage cells if their levels are not controlled. The enzyme protects the cell against dangerous levels of superoxide. Zinc binding promotes dimerization and stabilizes the native form. Mutations in SOD1 cause a form of familial amyotrophic lateral sclerosis. Defects in SOD1 are the cause of amyotrophic lateral sclerosis type 1 (ALS1) which is a familial form of amyotrophic lateral sclerosis, a neurodegenerative disorder affecting upper and lower motor neurons and resulting in fatal paralysis.

#### Note

For Research Use Only, Not for Diagnostic Use.

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