Data Sheet (Cat.No.TMPY-01585)

TargetMol

SOD1 Protein, Human, Recombinant (His)

General Information

Synonyms:	SOD;homodimer;ALS;hSod1;IPOA;superoxide dismutase 1, soluble;HEL-S-44;ALS1
Protein Construction:	A DNA sequence encoding the human SOD1 (NP_000445.1) (Ala 2-Gln 154) was expressed, with a polyhistide tag at the N-terminus. Predicted N terminal: Met
Species:	Human
Expression Host:	E. coli
Accession:	P00441
Molecular Weight:	16.8 kDa (predicted); 20 kDa (reducing conditions)

QC Testing

Biological Activity:	Activity testing is in progress. It is theoretically active, but we cannot guarantee it. If you require protein activity, we recommend choosing the eukaryotic expression version first.
Purity:	> 95 % as determined by SDS-PAGE
Endotoxin:	Please contact us for more information.
Formulation:	Lyophilized from a solution filtered through a 0.22 μm filter, containing 20 mM Tris, 500 mM NaCl, pH 8.0.Typically, a mixture containing 5% to 8% trehalose, mannitol, and 0.01% Tween 80 is incorporated as a protective agent before lyophilization.

Preparation and Storage

Reconstitution:

A Certificate of Analysis (CoA) containing reconstitution instructions is included with the products. Please refer to the CoA for detailed information.

Stability & Storage:

It is recommended to store recombinant proteins at -20°C to -80°C for future use. Lyophilized powders can be stably stored for over 12 months, while liquid products can be stored for 6-12 months at -80°C. For reconstituted protein solutions, the solution can be stored at -20°C to -80°C for at least 3 months. Please avoid multiple freeze-thaw cycles and store products in aliquots.

Actual storage temperature shall be subject to the COA.

Shipping:

In general, lyophilized powders are shipped with blue ice, while solutions are shipped with dry ice.

Protein Background

SOD1 belongs to the Cu-Zn superoxide dismutase family. It 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. 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.

Reference

Murakami K,et al.(2011) SOD1 (copper/zinc superoxide dismutase) deficiency drives amyloid β protein oligomerization and memory loss in mouse model of Alzheimer disease. J Biol Chem. 286(52):44557-68.
Thompson M,et al.(2012) Paradoxical roles of serine racemase and D-serine in the G93A mSOD1 mouse model of amyotrophic lateral sclerosis. J Neurochem. 120(4):598-610.
Magrané J,et al.(2012) Mitochondrial dynamics and bioenergetic dysfunction is associated with synaptic alterations in mutant SOD1 motor neurons. J Neurosci. 32(1):229-42.
Gertz B,et al.(2012) Nuclear localization of human SOD1 and mutant SOD1-specific disruption of survival motor neuron protein complex in transgenic amyotrophic lateral sclerosis mice. J Neuropathol Exp Neurol. 71(2):162-77.