ARG80928
Human SOD1 ELISA Kit

Package: 96 wells
Store at: 4°C

Summary

Product Description	ARG80928 Human SOD1 ELISA Kit is a Enzyme Immunoassay kit for the quantification of Human SOD1 (Cu/Zn-SOD) in Serum, Plasma and Cell culture supernatants.
Tested Reactivity	Hu
Tested Application	ELISA
Target Name	SOD1
Conjugation	HRP
Sensitivity	40 pg/ml
Sample Type	Serum, Plasma and Cell culture supernatants.
Standard Range	78 - 5000 pg/ml
Alternate Names	homodimer; EC 1.15.1.1; SOD; HEL-S-44; Superoxide dismutase [Cu-Zn]; ALS1; Superoxide dismutase 1; IPOA; ALS; hSod1

Properties

Form	96 well
Storage instruction	Store the kit at 2-8°C. Keep microplate wells sealed in a dry bag with desiccants. Do not expose test reagents to heat, sun or strong light during storage and usage. Please refer to the product user manual for detail temperatures of the components.
Note	For laboratory research only, not for drug, diagnostic or other use.

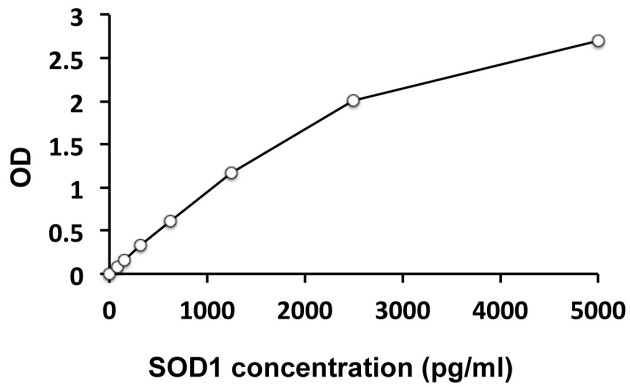
Bioinformation

Gene Symbol	SOD1
Gene Full Name	superoxide dismutase 1, soluble
Background	Superoxide Dismutases (SODs), originally identified as Indophenoloxidase (IPO), are enzymes that catalyze the conversion of naturallyoccuring but harmful superoxide radicals into molecular oxygen and hydrogen peroxide. Superoxide Dismutases 1, SOD1, also known as Cu/Zn SOD, soluble SOD and IPOA, is a soluble, cytoplasmic 16 kDa homodimer. Each SOD1 monomer binds one Cu2+ and one Zn2+ ion. Three isozymes of SOD have been identified and are functionally related but have very modest sequence homology. SOD1 shares 23% and 27% sequence identity with SOD2 and SOD3, respectively. Mutations in SOD1 have been implicated as causes of familial amyotrophic lateral sclerosis (ALS). The ALScausing mutations of SOD1 are scattered throughout the protein and provide no clear functional or structural clues to the underlying disease mechanism. The oligomerization hypothesis suggests that mutant SOD1 proteins become misfolded and consequently oligomerize into high molecular weight aggregates that result in the death of motor neurons. The oxidative damage hypothesis suggests that loss of function mutation in SOD1 protein results in the accumulation of cellular superoxide radical, leading to free radicalmediated damage, the release of cytochrome c, and apoptosis.
Function	Destroys radicals which are normally produced within the cells and which are toxic to biological systems. [UniProt]
Highlight	Related products:

SOD1 antibodies; SOD1 ELISA Kits; SOD1 Duos / Panels;
New ELISA data calculation tool:
Simplify the ELISA analysis by GainData

Research Area	Cancer kit; Cell Biology and Cellular Response kit; Cell Death kit; Gene Regulation kit; Metabolism kit; Microbiology and Infectious Disease kit; Neuroscience kit; Signaling Transduction kit
PTM	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.

Images



ARG80928 Human SOD1 ELISA Kit standard curve image

ARG80928 Human SOD1 ELISA Kit results of a typical standard run with optical density reading at 450nm.