

ARG83815 Rat SOD1 ELISA Kit

Package: 96 wells
Store at: 4°C

Summary

Product Description	ARG83815 Rat SOD1 ELISA Kit is an Enzyme Immunoassay kit for the quantification of Rat SOD1 in serum, plasma, tissue homogenates, cell lysates, cell culture supernatants and other biological fluids
Tested Reactivity	Rat
Tested Application	ELISA
Target Name	SOD1
Conjugation	HRP
Conjugation Note	Substrate: TMB and read at 450 nm.
Sensitivity	0.08 ng/mL
Sample Type	Serum, plasma, tissue homogenates, cell lysates, cell culture supernatants and other biological fluids
Standard Range	0.16-10 ng/mL
Sample Volume	100 µl
Alternate Names	homodimer; EC 1.15.1.1; SOD; HEL-S-44; Superoxide dismutase [Cu-Zn]; ALS1; Superoxide dismutase 1; IPOA; ALS; hSod1

Application Instructions

Assay Time	3.5 hours
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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	SOD (Superoxide Dismutase) is a well characterized cytosolic scavenger of oxygen free radicals that requires copper and zinc binding to potentiate its enzymatic activity. Enzymatically, SOD-1 facilitates the dismutation of oxygen radicals to hydrogen peroxide, and it also catalyzes prooxidant reactions, which include the peroxidase activity and hydroxyl radical generating activity. Defects in the gene encoding SOD-1 have been implicated in the progression of neurological diseases.
Function	Destroys radicals which are normally produced within the cells and which are toxic to biological systems. [UniProt]

PTM

Proteolytically processed under normal cellular conditions. Cleavage either by alpha-secretase, beta-secretase or theta-secretase leads to generation and extracellular release of soluble APP peptides, S-APP-alpha and S-APP-beta, and the retention of corresponding membrane-anchored C-terminal fragments, C80, C83 and C99. Subsequent processing of C80 and C83 by gamma-secretase yields P3 peptides. This is the major secretory pathway and is non-amyloidogenic. Alternatively, presenilin/nicastrin-mediated gamma-secretase processing of C99 releases the amyloid beta proteins, amyloid-beta 40 (Abeta40) and amyloid-beta 42 (Abeta42), major components of amyloid plaques, and the cytotoxic C-terminal fragments, gamma-CTF(50), gamma-CTF(57) and gamma-CTF(59). Many other minor beta-amyloid peptides, beta-amyloid 1-X peptides, are found in cerebral spinal fluid (CSF) including the beta-amyloid X-15 peptides, produced from the cleavage by alpha-secretase and all terminating at Gln-686.

Proteolytically cleaved by caspases during neuronal apoptosis. Cleavage at Asp-739 by either caspase-6, -8 or -9 results in the production of the neurotoxic C31 peptide and the increased production of beta-amyloid peptides.

N- and O-glycosylated. O-glycosylation on Ser and Thr residues with core 1 or possibly core 8 glycans. Partial tyrosine glycosylation (Tyr-681) is found on some minor, short beta-amyloid peptides (beta-amyloid 1-15, 1-16, 1-17, 1-18, 1-19 and 1-20) but not found on beta-amyloid 38, beta-amyloid 40 nor on beta-amyloid 42. Modification on a tyrosine is unusual and is more prevalent in AD patients. Glycans had Neu5AcHex(Neu5Ac)HexNAc-O-Tyr, Neu5AcNeu5AcHex(Neu5Ac)HexNAc-O-Tyr and O-AcNeu5AcNeu5AcHex(Neu5Ac)HexNAc-O-Tyr structures, where O-Ac is O-acetylation of Neu5Ac. Neu5AcNeu5Ac is most likely Neu5Ac 2,8Neu5Ac linked. O-glycosylations in the vicinity of the cleavage sites may influence the proteolytic processing. Appicans are L-APP isoforms with O-linked chondroitin sulfate.

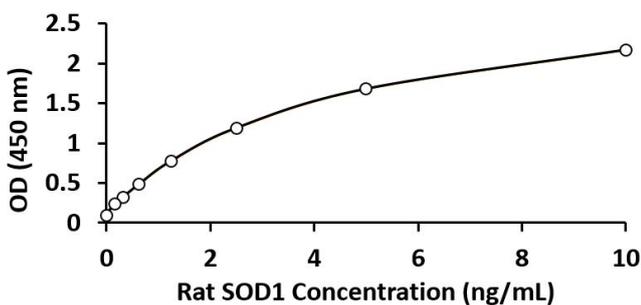
Phosphorylation in the C-terminal on tyrosine, threonine and serine residues is neuron-specific. Phosphorylation can affect APP processing, neuronal differentiation and interaction with other proteins. Phosphorylated on Thr-743 in neuronal cells by Cdc5 kinase and Mapk10, in dividing cells by Cdc2 kinase in a cell-cycle dependent manner with maximal levels at the G2/M phase and, in vitro, by GSK-3-beta. The Thr-743 phosphorylated form causes a conformational change which reduces binding of Fe65 family members. Phosphorylation on Tyr-757 is required for SHC binding. Phosphorylated in the extracellular domain by casein kinases on both soluble and membrane-bound APP. This phosphorylation is inhibited by heparin.

Extracellular binding and reduction of copper, results in a corresponding oxidation of Cys-144 and Cys-158, and the formation of a disulfide bond. In vitro, the APP-Cu(+) complex in the presence of hydrogen peroxide results in an increased production of beta-amyloid-containing peptides.

Trophic-factor deprivation triggers the cleavage of surface APP by beta-secretase to release sAPP-beta which is further cleaved to release an N-terminal fragment of APP (N-APP).

Beta-amyloid peptides are degraded by IDE.

Images



ARG83815 Rat SOD1 ELISA Kit standard curve image

ARG83815 Rat SOD1 ELISA Kit results of a typical standard run with optical density reading at 450 nm.