

## ARG81353 SOD Activity Assay Kit

Package: 100 assay Store at: -20°C

Summary	
Product Description	ARG81353 SOD Activity Assay Kit is a detection kit for the quantification of Superoxide Dismutase in serum, plasma and cell / tissue lysates.
Tested Reactivity	Hu, Ms, Rat, Mamm
Tested Application	FuncSt
Target Name	SOD
Conjugation Note	Read at 490 nm.
Sample Type	Serum, plasma and cell / tissue lysates.
Standard Range	0.0012 - 5 U/µl (Unit Definition: One unit will inhibit the rate of reduction of cytochrome c by 50% in a coupled system, using xanthine and xanthine oxidase, at pH 7.8 at 25°C in a 3.0 ml reaction volume.)
Sample Volume	10 - 70 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**

Application Note	Please note that this kit does not include a microplate.
Assay Time	1 hour

## Properties

Form	Liquid
Storage instruction	Store the kit at -20°C. 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	The protein encoded by this gene 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. [provided by RefSeq, Jul 2008]
Function	Destroys radicals which are normally produced within the cells and which are toxic to biological systems. [UniProt]

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.

Succinvlation, adjacent to copper catalytic site, probably inhibits activity. Desuccinvlation by SIRT5 enhances activity. [UniProt]

