

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human SOD1/Cu-Zn SOD in Western blots. In Western blots, no cross-reactivity with recombinant human (rh) SOD2 or rhSOD3 is observed.
Source	Monoclonal Mouse IgG _{2A} Clone # 348808
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	<i>E. coli</i> -derived recombinant human SOD1/Cu-Zn SOD Met1-Gln154 Accession # P00441
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide
*Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.	

APPLICATIONS

Please Note: Optimal dilutions should be determined by each laboratory for each application. [General Protocols](#) are available in the Technical Information section on our website.

Knockout Validated	Optimal dilution of this antibody should be experimentally determined.
Western Blot	Optimal dilution of this antibody should be experimentally determined.
Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

BACKGROUND

Superoxide Dismutases (SODs), originally identified as Indophenoloxidase (IPO), are enzymes that catalyze the conversion of naturally-occurring but harmful superoxide radicals into molecular oxygen and hydrogen peroxide. Superoxide Dismutases 1, SOD1, also known as Cu/Zn SOD, soluble SOD and IPO-A, 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 ALS-causing 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 radical-mediated damage, the release of cytochrome c, and apoptosis.

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