

DESCRIPTION

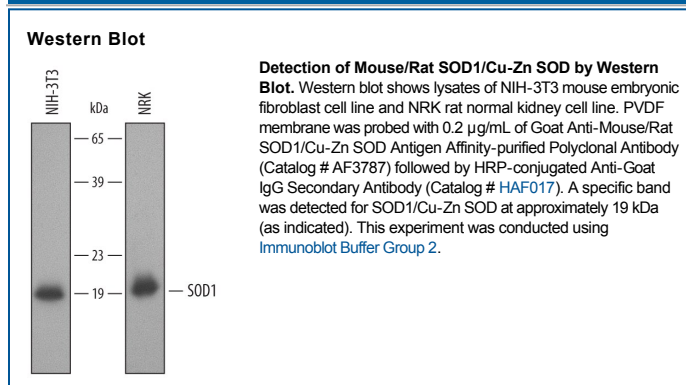
Species Reactivity	Mouse/Rat
Specificity	Detects endogenous mouse and rat SOD1 in Western blots. In Western blots, this antibody shows no cross-reactivity with recombinant human SOD2 or SOD3.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	<i>E. coli</i> -derived recombinant mouse SOD1 Met1-Gln154 Accession # P08228
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied as a 0.2 µm filtered solution in PBS.

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.

	Recommended Concentration	Sample
Western Blot	0.2 µg/mL	See Below

DATA



PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.2 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> ● 12 months from date of receipt, -20 to -70 °C as supplied. ● 1 month, 2 to 8 °C under sterile conditions after reconstitution. ● 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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 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. Mouse SOD1 is 97% aa identical to rat SOD1. Mutations in SOD1 have been suggested to be the cause 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 mutations in SOD1 result in the intracellular accumulation of the superoxide radical, leading to free radical-mediated damage, the release of cytochrome c, and apoptosis.