Human/Mouse SOD1/Cu-Zn SOD Antibody

Antigen Affinity-purified Polyclonal Goat IgG
Catalog Number: AF3418

DESCRIPTION

Species Reactivity	Human/Mouse
Specificity	Detects human and mouse SOD1/Cu-Zn SOD in Western blots. Detects rat SOD1, but Catalog # AF3787 is recommended.
Source	Polyclonal Goat IgG
Purification	Antigen Affinity-purified
Immunogen	*E. coli*-derived recombinant human SOD1/Cu-Zn SOD Met1-Gln154 Accession # P00441
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 either lyophilized or as a 0.2 μm filtered solution in PBS.

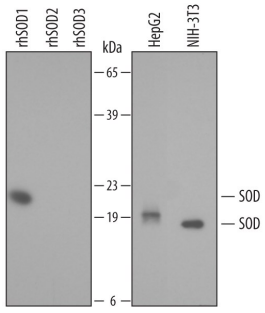
APPLICATIONS

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
Simple Western	10 µg/mL	See Below

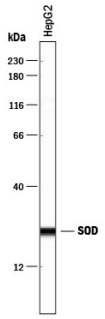
DATA

Western Blot



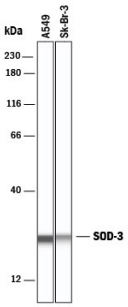
Detection of Human/Mouse SOD1/Cu-Zn SOD by Western Blot. Western blot shows lysates of HepG2 human hepatocellular carcinoma cell line and NIH-3T3 mouse embryonic fibroblast cell line. PVDF membrane was probed with 0.2 µg/mL Goat Anti-Human/Mouse SOD1/Cu-Zn SOD Antigen Affinity-purified Polyclonal Antibody (Catalog # AF3418) followed by HRP-conjugated Anti-Goat IgG Secondary Antibody (Catalog # HAF109). For additional reference, recombinant human SOD1, SOD2, and SOD3 (1 ng/lane) were included. A specific band for SOD1/Cu-Zn SOD was detected at approximately 16-19 kDa (as indicated). This experiment was conducted under reducing conditions and using Immunoblot Buffer Group 2.

Simple Western



Detection of Human SOD1/Cu-Zn SOD by Simple Western™. Simple Western lane view shows lysates of HepG2 human hepatocellular carcinoma cell line, loaded at 0.2 mg/mL. A specific band was detected for SOD1/Cu-Zn SOD at approximately 25 kDa (as indicated) using 10 µg/mL of Goat Anti-Human/Mouse SOD1/Cu-Zn SOD Antigen Affinity-purified Polyclonal Antibody (Catalog # AF3418) followed by 1:50 dilution of HRP-conjugated Anti-Goat IgG Secondary Antibody (Catalog # HAF109). This experiment was conducted under reducing conditions and using the 12-230 kDa separation system.

Simple Western



Detection of Human SOD1/Cu-Zn SOD by Simple Western™. Simple Western lane view shows lysates of A549 human lung carcinoma cell line and SK-BR-3 human breast cancer cell line, loaded at 0.2 mg/mL. A specific band was detected for SOD1/Cu-Zn SOD at approximately 24 kDa (as indicated) using 10 µg/mL of Goat Anti-Human/Mouse SOD1/Cu-Zn SOD Antigen Affinity-purified Polyclonal Antibody (Catalog # AF3418) followed by 1:50 dilution of HRP-conjugated Anti-Goat IgG Secondary Antibody (Catalog # HAF109). This experiment was conducted under reducing conditions and using the 12-230 kDa separation system.

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.** • 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.

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BACKGROUND

Superoxide Dismutases (SODs), originally identified as Indophenoloxidase (IPO), are enzymes that catalyze the converversion of naturally-occuring 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 Cu^{2+} and Zn^{2+} 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 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.

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