SANTA CRUZ BIOTECHNOLOGY, INC.

PRX III (4G10): sc-59663

BACKGROUND

The peroxiredoxin (PRX) family comprises six antioxidant proteins, PRX I, II, III, IV, V and VI, which protect cells from reactive oxygen species (ROS) by preventing the metal-catalyzed oxidation of enzymes. The PRX proteins primarily utilize thioredoxin as the electron donor for antioxidation, although they are fairly promiscuous with regard to the hydroperoxide substrate. In addition to protection from ROS, peroxiredoxins are also involved in cell proliferation, differentiation and gene expression. PRX I, II, IV and VI show diffuse cytoplasmic localization, while PRX III and V exhibit distinct mitochondrial localization. The human PRX I gene encodes a protein that is expressed in several tissues, including liver, kidney, testis, lung and nervous system. PRX II is expressed in testis, while PRX III shows expression in lung. PRX I, II and III are overexpressed in breast cancer and may be involved in its development or progression. Upregulated protein levels of PRX I and II in Alzheimer's disease (AD) and Down syndrome (DS) indicate the involvement of PRX I and II in their pathogenesis. The human PRX IV gene is abundantly expressed in many tissues. PRX IV exists as a precursor protein, which is only detected in testis, and a processed secreted form. PRX V also exists as two forms, designated long and short. Like PRX IV, the long form of PRX V is highly expressed in testis. The short form of PRX V is more widely expressed, with high expression in liver, kidney, heart and lung. PRX VI, α 1-Cys peroxiredoxin (also known as antioxidant protein 2 or AOP2), is highly expressed in most tissues, particularly in epithelial cells. Localized to the cell cytosol, PRX VI functions independently of other peroxiredoxins and antioxidant proteins, specializing in antioxidant defense, lung phospholipid metabolism and protection of keratinocytes from cell death induced by reactive oxygen species.

REFERENCES

1. Iwahara, S., et al. 1995. Purification, characterization and cloning of a heme-binding protein (23 kDa) in rat liver cytosol. Biochemistry 34: 13398-13406.
2. Butterfield, L.H., et al. 1999. From cytoprotection to tumor suppression: the multifactorial role of peroxiredoxins. Antioxid. Redox Signal. 1: 385-402.
3. Mizusawa, H., et al. 2000. Peroxiredoxin I (macrophage 23 kDa stress protein) is highly and widely expressed in the rat nervous system. Neurosci. Lett. 283: 57-60.

CHROMOSOMAL LOCATION

Genetic locus: PRDX3 (human) mapping to 10q26.11.

SOURCE

PRX III (4G10) is a mouse monoclonal antibody raised against PRX III of human origin.

PRODUCT

Each vial contains IgG_1 in 100 µl of PBS with < 0.1% sodium azide, 0.1% gelatin, 50% glycerol and 0.01% stabilizer protein.

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

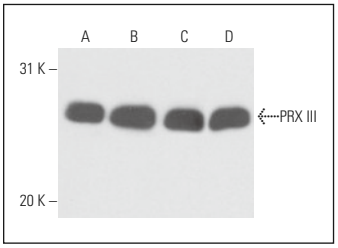
PRX III (4G10) is recommended for detection of PRX III of human origin by Western Blotting (starting dilution to be determined by researcher, dilution range 1:100-1:5000), immunoprecipitation [1-2 µl per 100-500 µg of total protein (1 ml of cell lysate)] and solid phase ELISA (starting dilution to be determined by researcher, dilution range 1:30-1:5000).

Suitable for use as control antibody for PRX III siRNA (h): sc-40833, PRX III shRNA Plasmid (h): sc-40833-SH and PRX III shRNA (h) Lentiviral Particles: sc-40833-V.

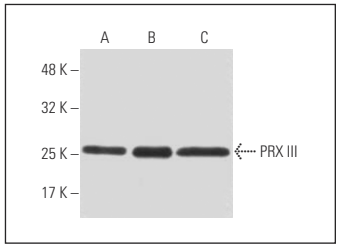
Molecular Weight of PRX III: 26 kDa.

Positive Controls: MCF7 whole cell lysate: sc-2206, HeLa whole cell lysate: sc-2200 or Jurkat whole cell lysate: sc-2204.

DATA



PRX III (4G10): sc-59663. Western blot analysis of PRX III expression in HeLa (**A**), NCI-H1688 (**B**), SHP-77 (**C**) and MCF7 (**D**) whole cell lysates.



PRX III (4G10): sc-59663. Western blot analysis of PRX III expression in HeLa (**A**), 293T (**B**) and Jurkat (**C**) whole cell lysates.

SELECT PRODUCT CITATIONS

1. Gimigliano, A., et al. 2012. Proteomic profile identifies dysregulated pathways in Cornelia de Lange syndrome cells with distinct mutations in SMC1A and SMC3 genes. J. Proteome Res. 11: 6111-6123.
2. Folda, A., et al. 2016. Mitochondrial thioredoxin system as a modulator of Cyclophilin D redox state. Sci. Rep. 6: 23071.
3. Caino, M.C., et al. 2016. A neuronal network of mitochondrial dynamics regulates metastasis. Nat. Commun. 7: 13730.
4. Caino, M.C., et al. 2017. Syntaphilin controls a mitochondrial rheostat for proliferation-motility decisions in cancer. J. Clin. Invest. 127: 3755-3769.
5. Elliott, B., et al. 2019. Essential role of JunD in cell proliferation is mediated via MYC signaling in prostate cancer cells. Cancer Lett. 448: 155-167.
6. Agrawal, S. and Fox, J.H. 2019. Novel proteomic changes in brain mitochondria provide insights into mitochondrial dysfunction in mouse models of Huntington's disease. Mitochondrion 47: 318-329.

RESEARCH USE

For research use only, not for use in diagnostic procedures.