PRDX3

PRDX3 is one member of Peroxiredoxins (PRDXs) localized in the mitochondria, and has been shown to be involved in antioxidant defense and redox signaling.

Hu et al. investigated the role of PRDX3 in neuronal trauma using a traumatic neuronal injury (TNI) model in primary cultured cortical neurons. They found that TNI significantly decreased the expression of PRDX3 at both mRNA and protein levels. Overexpression of PRDX3 by lentivirus (LV-PRDX3) transfection attenuated lactate dehydrogenase (LDH) release and neuronal apoptosis after TNI. The results of immunostaining showed that LV-PRDX3 transfection markedly reduced TNI-induced intracellular ROS production, protein radical formation and lipid peroxidation. In addition, overexpression of PRDX3 preserved mitochondrial membrane potential (MMP) levels and ATP generation, and inhibited mitochondrial cytochrome c release in TNI-injured neurons. The results of polymerase chain reaction (PCR) showed that PRDX3 overexpression also increased mitochondrial DNA (mtDNA) content and upregulated the expression of mitochondrial biogenesis-related factors. Taken together, our data demonstrate that PRDX3 protects against TNI insult by preserving mitochondrial function and mitochondrial biogenesis, and may have potential therapeutic value for traumatic brain injury (TBI) [1].

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Hu W, Dang XB, Wang G, Li S, Zhang YL. Peroxiredoxin-3 attenuates traumatic neuronal injury through preservation of mitochondrial function. Neurochem Int. 2018 Feb 7. pii: S0197-0186(17)30590-9. doi: 10.1016/j.neuint.2018.02.004. [Epub ahead of print] PubMed PMID: 29427714.