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Manganese superoxide dismutase and oxidative stress modulation.

Bresciani G[1], da Cruz IB[2], González-Gallego J[3].

Author information

1 Facultad de Ciencias de la Salud, Universidad Autónoma de Chile, Temuco, Chile. Electronic address: guilhermebresciani@gmail.com.
2 Laboratório de Biogenômica, Departamento de Morfologia, Universidade Federal de Santa Maria, Santa Maria, Brazil.
3 Institute of Biomedicine (IBIOMED) and Centro de Investigación Biomédica en Red de Enfermedades Hepáticas y Digestivas (CIBERehd), University of León, León, Spain.

Abstract

Oxidative stress is characterized by imbalanced reactive oxygen species (ROS) production and antioxidant defenses. Two main antioxidant systems exist. The nonenzymatic system relies on molecules to directly quench ROS and the enzymatic system is composed of specific enzymes that detoxify ROS. Among the latter, the superoxide dismutase (SOD) family is important in oxidative stress modulation. Of these, manganese-dependent SOD (MnSOD) plays a major role due to its mitochondrial location, i.e., the main site of superoxide (O(2)(·-)) production. As such, extensive research has focused on its capacity to modulate oxidative stress. Early data demonstrated the relevance of MnSOD as an O(2)(·-) scavenger. More recent research has, however, identified a prominent role for MnSOD in carcinogenesis. In addition, SOD downregulation appears associated with health risk in heart and brain. A single nucleotide polymorphism which alters the mitochondria signaling sequence for the cytosolic MnSOD form has been identified. Transport into the mitochondria was differentially affected by allelic presence and a new chapter in MnSOD research thus begun. As a result, an ever-increasing number of diseases appear associated with this allelic variation including metabolic and cardiovascular disease. Although diet and exercise upregulate MnSOD, the relationship between environmental and genetic factors remains unclear.

KEYWORDS: Diseases; Environmental factors; MnSOD; Oxidative stress; SNP; Translational and posttranslational modulation

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