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An Intimate Relationship between ROS and Insulin Signalling: Implications for Antioxidant Treatment of Fatty Liver Disease

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Abstract

Oxidative stress damages multiple cellular components including DNA, lipids, and proteins and has been linked to pathological alterations in nonalcoholic fatty liver disease (NAFLD). Reactive oxygen species (ROS) emission, resulting from nutrient overload and mitochondrial dysfunction, is thought to be a principal mediator in NAFLD progression, particularly toward the development of hepatic insulin resistance. In the context of insulin signalling, ROS has a dual role, as both a facilitator and inhibitor of the insulin signalling cascade. ROS mediate these effects through redox modifications of cysteine residues affecting phosphatase enzyme activity, stress-sensitive kinases, and metabolic sensors. This review highlights the intricate relationship between redox-sensitive proteins and insulin signalling in the context of fatty liver disease, and to a larger extent, the importance of reactive oxygen species as primary signalling molecules in metabolically active cells.

Figures

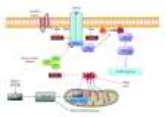

Figure 1 Molecular mechanisms linking ROS, antioxidants,...

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