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Oxidative stress drivers and modulators in obesity and cardiovascular disease: from biomarkers to therapeutic approach.

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Abstract

This review article is intended to describe how oxidative stress regulates cardiovascular disease development and progression. Epigenetic mechanisms related to oxidative stress, as well as more reliable biomarkers of oxidative stress, are emerging over the last years as potentially useful tools to design therapeutic approaches aimed at modulating enhanced oxidative stress "in vivo", thereby mitigating the consequent atherosclerotic burden. As a paradigm, we describe the case of obesity, in which the intertwining among oxidative stress, due to caloric overload, chronic low-grade inflammation induced by adipose tissue dysfunction, and platelet activation represents a vicious cycle favoring the progression of atherothrombosis. Oxidative stress is a major player in the pathobiology of cardiovascular disease (CVD). Reactive oxygen species (ROS)- dependent signaling pathways prompt transcriptional and epigenetic dysregulation, inducing chronic low-grade inflammation, platelet activation and endothelial dysfunction. In addition, several oxidative biomarkers have been proposed with the potential to improve current understanding of the mechanisms underlying CVD. These include ROS-generating and/or quenching molecules, and ROS-modified compounds, such as F2-isoprostanes. There is also increasing evidence that noncoding micro- RNA (mi-RNA) are critically involved in post- transcriptional regulation of cell functions, including ROS generation, inflammation, regulation of cell proliferation, adipocyte differentiation, angiogenesis and apoptosis. These molecules have promising translational potential as both markers of disease and site of targeted interventions. Finally, oxidative stress is a critical target of several cardioprotective drugs and nutraceuticals, including antidiabetic agents, statins, renin-angiotensin system blockers, polyphenols and other antioxidants. Further understanding of ROS-generating mechanisms, their biological role as well as potential therapeutic implications would translate into consistent benefits for effective CV prevention.

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