Review

Lipid peroxidation, isoprostanes and vascular damage

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Abstract:
Increased lipid peroxidation has been identified as a key mechanism for the development of atherosclerosis and inflammatory vascular damage. Determination of plasma concentration and urinary excretion of some F₂-isoprostanes (by immunometric assays or by mass-spectrometry), has been demonstrated to be a reliable approach to the assessment of lipid peroxidation, and therefore of oxidative stress in vivo. Several lines of evidence suggest that isoprostane generation may reflect oxidative stress in experimental and human atherosclerosis. Increased lipid peroxidation may precede the development of atherosclerosis. In fact, urinary excretion or plasma levels of an abundantly generated F₂-isoprostane, 8-iso-PGF₂α, have been found to be more elevated in subject with cardiovascular risk factors than in healthy subjects. Some isoprostanes, in particular 8-iso-PGF₂α, have been demonstrated to have biological activities that may contribute to the progression of vascular damage inducing endothelial and platelet activation and being powerful vasocostrictors. Increased lipid proxidation may be implicated in the bioactivity of angiotensin II. Experimental data indicate that increased oxidative stress due to activation of NAD(P)H oxidase is an obligatory step in its pro-hypertensive and pro-atherosclerotic effects. Increased generation of F₂-isoprostanes is observed in clinical and experimental conditions in which angiotensin II activity is increased. In conclusion, measurement of some F₂-isoprostanes in biological liquids represents a reliable marker of oxidative stress in vivo. The potential contribution of these compounds to the pathophysiology of the vascular damage and atherosclerosis has not yet been defined.

Key words:
isoprostanes, oxidative stress, atherosclerosis, cardiovascular risk factors, vascular damage, angiotensin II