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Special Issue

Lipoproteins and other Pathogenic Factors of Atherosclerosis

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Special Issue Introduction

Extra- and intracellular deposition of lipids, predominantly of cholesteryl esters, in arterial intima is one of the earliest manifestations of atherosclerosis. Formation of lipid laden foam cells is recognized as a trigger in the pathogenesis of atherosclerosis. Low-density lipoprotein (LDL) circulating in human blood is the source of lipids accumulated in arterial cells. However, numerous attempts to induce intracellular accumulation of cholesteryl esters by native LDL failed. In response to this challenge, intensive search for pro-atherogenic modified forms of circulating LDL have been started, and such forms were soon found. Among pro-atherogenic modified LDLs detected in blood, such forms as oxidized, small dense, desialylated, and electronegative have been described.

Large-scale epidemiological studies firmly established the association between low plasma levels of high-density lipoprotein (HDL) and elevated risk of cardiovascular disease. This relationship is thought to reflect the key biological function of HDL, which involves reserve cholesterol transport from the arterial wall to the liver for further excretion from the body. Factors that impair the activities of HDL strongly influence atherogenesis. HDL also inhibits lipid oxidation, restores endothelial function, exerts anti-inflammatory and antiapoptotic actions, and exerts anti-inflammatory actions in animal models. Such properties could contribute considerably to the capacity of HDL to inhibit atherosclerosis. Systemic and vascular inflammation has been proposed to convert HDL to a dysfunctional form that has impaired antiatherogenic effects. A loss of anti-inflammatory and antioxidative proteins, perhaps in combination with a gain of proinflammatory proteins, might be another important component in rendering HDL dysfunctional.

The purpose of the thematic issue is to collect current knowledge on the role of modified and dysfunctional lipoproteins as well as non-lipid factors in atherogenesis. Understanding the features of pathogenic factors might lead to new diagnostic and therapeutic approaches to atherosclerosis.

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