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Dysfunctional HDL - Bench to Clinic

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Abstract

High-density lipoproteins (HDLs) protect against atherosclerosis through multiple salutary mechanisms. Factors that impair the availability of functional apolipoproteins or the activities of macrophage cholesterol efflux pathways could markedly 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 proinflammatory enzyme myeloperoxidase renders HDL dysfunction by inducing both oxidative modification and nitrosylation of specific residues on plasma and arterial apolipoprotein A-I to render HDL dysfunctional, which results in impaired ABCA1 macrophage transport, the activation of inflammatory pathways, and an increased risk of coronary artery disease. Understanding the features of dysfunctional HDL or apolipoprotein A-I in clinical practice might lead to new diagnostic and therapeutic approaches to atherosclerosis.

Keywords

HDL function, HDL dysfunction, HDL proteome, HDL lipidome, macrophage cholesterol efflux, proinflammatory HDL