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[Atherosclerosis](#). 2011 Feb 2. [Epub ahead of print]

High-density lipoprotein cholesterol and apolipoprotein A1 levels strongly influence the reactivity of small peripheral arteries.

Ferré R, Aragonès G, Plana N, Merino J, Heras M, Buixadera C, Masana L.

Vascular Medicine and Metabolism Unit and Research Unit on Lipids and Atherosclerosis, Sant Joan University Hospital, IISPV, Rovira i Virgili University, 43201 Reus, Spain; Spanish Biomedical Research Network in Diabetes and Associated Metabolic Disorders (CIBERDEM), Barcelona, Spain.

Abstract

OBJECTIVE: Reactive hyperaemia after shear stress is a surrogate marker of endothelial function. However, the mechanisms controlling the dilation capacity of small peripheral resistance arteries are not well characterised. We evaluated reactive hyperaemia by peripheral artery tonometry (PAT) in the acral arteries and studied their clinical and biochemical determinants.

METHODS: Eight hundred sixteen subjects at intermediate to high cardiovascular risk were recruited. The reactive hyperaemia index (RHI) of small digital arteries was measured by PAT. Clinical history data, anthropometry and biochemical parameters were also analysed. We studied the associations between clinical and biochemical factors and small artery RHI.

RESULTS: HDL cholesterol and apolipoprotein A1 levels were strongly and directly correlated with an increased dilation response. Metabolic syndrome components, such as increased waist circumference, hypertriglyceridaemia and smoking, were inversely associated with RHI as were serum markers of inflammation. The predictors of small peripheral artery RHI were HDL cholesterol, which had a protective effect, and smoking, which had a negative impact.

CONCLUSION: HDL cholesterol and apolipoprotein A1 levels had a strong, positive correlation with small artery reactive hyperaemia, whereas smoking, waist circumference and triglyceride levels were inversely associated. HDL cholesterol was the main determinant of RHI in small peripheral resistance arteries.

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PMID: 21367423 [PubMed - as supplied by publisher]

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