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Extralipid effects of hypolipidemic drugs – why do clinical trials weakly support experimental data?

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Abstract:
There are many experimental and clinical data confirming the inflammatory cause of atherosclerosis. It is in agreement with the commonly accepted, pathomorphological theory described as: "reaction to injury". Connections between the concentration of proinflammatory cytokines, chemokines and serum lipoproteins are under current investigations. There are also attempts to compare amount of the above-mentioned molecules with atherosclerotic plaque dimentions and increased artery wall thickness. Much more promissing seems to be describing the role of inflammatory cell products in vascular risk stratification.
Carefully planned, prospective observations of patients are of greatest value for reliable information. The role of multicenter clinical studies is smaller because of their strict end-points and methodological restrictions. Hence, the question has arisen whether adherents of large population trials and evidence-based medicine trust in value of single-center studies. How to reconcile the effectiveness of therapy based on large population trials with complicated methods of determination of proinflammatory factors? Restrictive inclusion criteria requiring accurate diagnosis of inflammation raise doubts. For some investigators, it is only preselection, which reduces the real value of achieved results.
Anti-inflammatory and vasoprotective influence of hypolipidemic and hypotensive drugs is considered to be an important clinical supplementation to their basic mechanism of action. It cannot be ruled out that these additional effects of drugs are responsible for better outcomes in the treated patients.
Generally, precise distinguishing the effects of different groups of drugs is usually impossible in circumstances of clinical trials. However, we can measure different molecules, hs-CRP assay represents the best choice at this time.

Key words:
atherosclerosis, proinflammatory cytokines, adhesion molecules, hs-C-reactive protein (hs-CRP), hypolipemic therapy