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Serum lipids and cardiovascular reactivity to stress

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Abstract

Several studies have reported an association between serum lipid levels and cardiovascular reactivity to laboratory stressors. Their findings, however, are equivocal. The inconsistencies may be due to shortcomings such as the small number of subjects, the inclusion of patient groups, no control for medication, and no control for age effects. Two studies are presented investigating the relationship in large groups of adolescent and middle-aged males and females. Cholesterol, triglycerides and HDL were measured. Subjects were exposed to mental stressors, and in one study also to a cold pressor test. In addition to heart rate and blood pressure, in one study impedance cardiography was used to measure pre-ejection period, stroke volume and total peripheral resistance. Canonical correlation analysis suggested an association between triglycerides and decreased cardiac reactivity to mental stressors in middle-aged females. Trends in the same direction were found in both middle-aged males and females with respect to reactivity to the cold pressor. These associations, however, were not confirmed when the extreme deciles of the triglyceride distributions were compared with respect to stress reactivity. The fact that associations were completely absent in youngsters but sometimes showed up in older persons suggested an age dependency of the association. In post hoc analyses, indeed, some evidence was found for stronger cardiac responsivity being associated with cholesterol specifically in relatively older males. In females, in contrast to this, the association between triglycerides and cardiac responsivity was stronger in the younger group. More detailed measurement techniques, of specifically vascular processes, may be needed to explore further the effects of sex and age on the association between lipids and stress reactivity.

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1. Introduction

The association between serum lipid levels and the risk of coronary heart disease (CHD) is well established. Lipids, including serum cholesterol and triglycerides, are transported in the circulation bound to (apo)proteins, the cluster together forming the lipoproteins. The different lipoproteins can be separated on the basis of their densities (lipid/protein ratios). The major classes of lipoproteins are very low-density lipoprotein (VLDL), mainly consisting of triglycerides, low-density lipoprotein (LDL), the main transporter of cholesterol, and high-density lipoprotein (HDL). HDL exhibits a reverse relationship with CHD risk. Serum lipid and lipoprotein levels have a substantial genetic/constitutional component (Heller et al., 1993; Snieder et al., 1997) and are also influenced by behavioral factors like diet (Hopkins, 1992), exercise (Tran et al., 1983) and smoking (Freeman et al., 1993). Periods of episodic or chronic stress have shown to be associated with elevated cholesterol levels. This has, for example, been demonstrated for undergoing an earthquake (Trevisan et al., 1992), job loss (Kasl et al., 1968), threat of unemployment (Mattiasson et al., 1990), perceived job insecurity (Siegrist et al., 1988), perceived workload (McCann et al., 1990) or examinations (van Doornen and van Blokland, 1987). A rise in cholesterol level during stress is, however, not a universal finding (Niaura et al., 1991).

The mechanism underlying a stress-induced cholesterol elevation is supposed to be the sympatho-adrenergic system, because adrenaline is a potent lipolytic agent. Exaggerated cardiovascular reactivity to stress has been proposed as a risk factor for future CHD (Matthews et al., 1986; van Doornen, 1991). The propensity of individuals to be sympathetically reactive to stress is generally assessed by exposing subjects to short-term laboratory stressors. Large individual differences in cardiovascular reactivity are generally observed, which seem to reflect differences in adrenergic mobilisation (Eisenhofer et al., 1985). Because the sympatho-adrenergic system is both involved in lipolysis and in stress reactions several studies have explored the association between cardiovascular reactivity to stress and serum lipids. According to this idea lipid levels and stress reactivity are associated because of a common mechanism: sympatho-adrenal activity.

There is yet another mechanism that might be responsible for a possible association. Hypercholesteremia has been shown to be associated with an augmented response to vasoconstrictors (Heistad et al., 1984) and impaired endothelial-dependent vascular relaxation (Casino et al., 1993). This points to a more peripheral vascular explanation for the association between stress reactivity and lipids.

The idea of a common sympathetic mechanism would show up as a relationship between cardiac responsivity and lipids, the more peripheral explanation would predict lipids to be associated with a stronger rise in peripheral resistance in response to stress.