

Effects of Hemoconcentration and Sympathetic Activation on Serum Lipid Responses to Brief Mental Stress

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Effects of Hemoconcentration and Sympathetic Activation on Serum Lipid Responses to Brief Mental Stress

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Abstract

Objective: Previous studies suggest that hemoconcentration may be one mechanism by which acute psychological stress causes elevations of serum total cholesterol and its subfractions. Alternatively, such elevations may result from sympathetically mediated changes in lipid metabolism. This study evaluated these two hypotheses by manipulation of sympathetically mediated responses to stress using a nonselective adrenoceptor antagonist, labetalol.

Method: In a 2 × 2 factorial design, 52 healthy male participants were randomly assigned to a stress or no-stress condition and, within each condition, were administered either labetalol or saline. Participants assigned to stress completed three cognitive and evaluative tasks lasting a total of 18 minutes. Indices of hemoconcentration (hematocrit and hemoglobin), heart rate, blood pressure, and serum lipids (total, high-density lipoprotein (HDL), low-density lipoprotein (LDL), free fatty acids, and triglycerides) were assessed at preinfusion and infusion baselines and after mental stress (or rest).

Results: Labetalol reduced sympathetic activation, as shown by a substantial reduction in heart rate elevation during stress, but did not alter changes in blood pressure or in hemoconcentration, as indicated by equivalent increases in hematocrit and hemoglobin in the two stressed groups. Labetalol blocked stress-induced increases in free fatty acid concentrations and lowered triglyceride levels but did not influence rises in total, HDL, or LDL cholesterol among stressed subjects. However, arithmetic correction for

hemoconcentration eliminated the increases in total, HDL, and LDL cholesterol.

Conclusions: These findings suggest that elevations in total cholesterol and its HDL and LDL subfractions during acute stress are caused by accompanying hemoconcentration, whereas concomitant rises in free fatty acids and triglycerides result from the direct metabolic effects of sympathetic activation.

Key words: cholesterol, lipids, stress, hemoconcentration, sympathetic nervous system.

Reference

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