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Acute alcohol consumption downregulates lipoprotein lipase activity *in vivo*

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ABSTRACT

Objective. Acute alcohol consumption can induce hypertriglyceridemia. Such an effect could be explained in part by the influence of alcohol on lipoprotein lipase (LPL) – the key enzyme responsible for triglyceride hydrolysis in circulation. Therefore, we have studied the effects of acute moderate alcohol consumption on LPL activity and on the concentrations of angiopoietin-like proteins 3 and 4 (ANGPTL3 and ANGPTL4), which are known to inhibit LPL.

Methods. Two experiments were carried out in 8 healthy volunteers. They received 25 g of alcohol (vodka) in one experiment and water in the other (control). The *in vivo* function of LPL was estimated using intravenous fat tolerance tests (IVFTT) carried out before, 2 and 4 hours after alcohol administration. At the end of each experiment, LPL activity and mass were measured in post-heparin plasma (PHP). The concentrations of ANGPTL3 and ANGPTL4 in blood were measured before alcohol consumption and at the end of the experiments.

Results. LPL activity, as estimated using the IVFTT, was reduced by 25% and 24% two and four hours after the administration of alcohol, respectively, and was not affected in the control experiment. At the end of the experiment, LPL activity in PHP was 23% lower after alcohol consumption than in the controls. The concentrations of ANGPTL3 and ANGPTL4 had dropped to 67% and 86% of baseline values, respectively, at 280 min after alcohol consumption. These levels were not affected in the control experiment. The levels of ANGPTL4 but not those of ANGPTL3 were increased in PHP compared to both baseline values and values at 280 min.

Conclusion. The capacity for triglyceride clearance seemed to be acutely reduced by alcohol consumption and the effect persisted for several hours. The levels of LPL activity in PHP were reduced to a similar extent. This reduction in LPL activity could not be explained by the changes in the levels of ANGPTL3 or ANGPTL4, which both decreased.

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Abbreviations: ANGPTL, angiopoietin-like protein; ANOVA, analysis of variance; apo B, apolipoprotein B; BMI, body mass index; ELISA, enzyme-linked immunosorbent assay; HDL, high density lipoprotein; HDL-C, high density lipoprotein cholesterol; IVFTT, intravenous fat tolerance test; LPL, lipoprotein lipase; NEFA, non-esterified fatty acid; PHP, post-heparin plasma; TG, triglyceride; VLDL, very low density lipoprotein.

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1. Introduction

Alcohol administration increases TG levels in blood [1], and alcohol consumed with a meal augments the magnitude of postprandial triglyceridemia [2–6]. These acute effects of alcohol consumption, especially on postprandial lipemia, seem to be accentuated in hypertriglyceridemic subjects [7]. Such an effect can be explained by a decrease in lipoprotein lipase (LPL) activity, a key enzyme responsible for intravascular TG lipolysis, after ethanol intake [2,8,9]. To clarify the role of LPL as a mediator of the acute effects of ethanol on triglyceridemia, we estimated LPL activity *in vivo* using an intravenous fat tolerance test (IVFTT) before and at 2 and 4 hours after consumption of 25 g of ethanol. LPL activity and mass were measured in post-heparin plasma obtained at the end of each experiment. In addition, we measured the levels of angiopoietin-like proteins (ANGPTL) 3 and 4, two proteins involved in the regulation of LPL activity [10].

2. Methods

Eight non-obese and normolipemic Caucasian young men (age: 24 ± 1 years, BMI: 24.5 ± 2.2 kg/m^2, cholesterol 3.86 ± 0.58 mmol/l, moderate drinkers) were included in the study, which adhered to the principles of the Declaration of Helsinki and which was approved by the Ethics Committee of the Institute for Clinical and Experimental Medicine and Thomayer Hospital in Prague. All participants gave their informed consent.

Two experiments with the same design were carried out (Fig. 1A). In one, the subjects drank alcohol (vodka, 40% ethanol); in the other, they drank the same volume of water. The order of the experiments was randomized and they were carried out with an interval between them of at least 2 weeks. The subjects were advised not to consume any alcohol for 48 hours preceding the experiments. During the experiments the subjects consumed no food and were allowed only water. One hour before alcohol administration

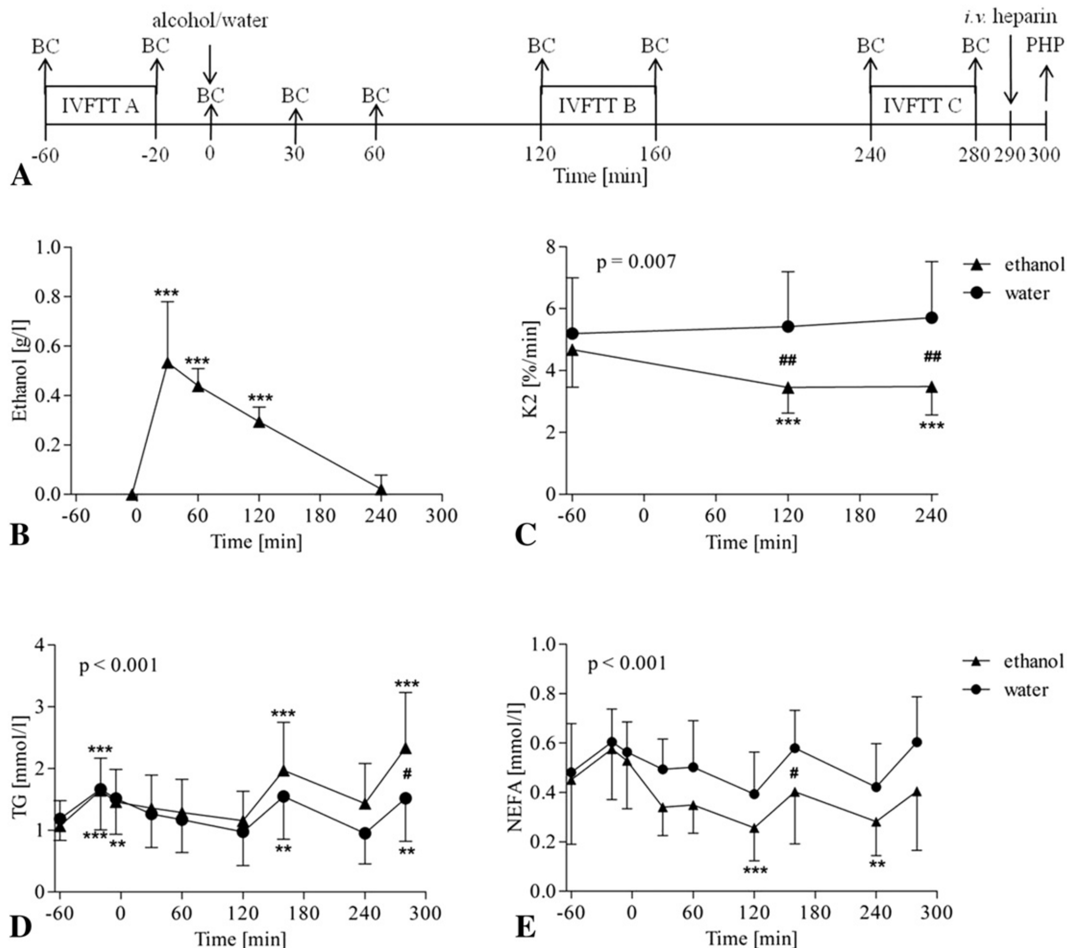

Fig. 1 – (A) Design of the experiment. IVFTT – intravenous fat tolerance test; alcohol/water – alcohol or water consumption; PHP – post-heparin plasma. Ethanol concentration (B), k2 rate constant for clearance of fat emulsion in IVFTT (C), TG (D) and NEFA (E) concentrations after alcohol (EtOH) and water administration at 0 min. Data are presented as mean ± SD., *** p < 0.01, p < 0.001 vs the first measurement in the series; #, ## p < 0.05, p < 0.01, alcohol vs water administration.**

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