Metabolic and Vascular Responses to Short, Disrupted Sleep and High-Intensity Interval Exercise in Healthy Men

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ABSTRACT

Exercise is known to impart transient blood lipid and vascular responses that appear consistent with reduced cardiovascular disease risk. It is unclear how short, disrupted sleep (SDS) modifies post-exercise blood lipid and conduit artery responses to a single episode of exercise. PURPOSE: To determine the influence of a single night of SDS on fasting and postprandial blood lipid and conduit artery responses after high-intensity interval exercise (HIIE). METHODS: Fifteen male participants (age 31.1 ± 5.3 yr; weight 83.5 ± 11.4 kg; BMI 25.8 ± 2.7 kg/m²; VO2max 49.1 ± 8.5 ml/kg/min) completed a non-exercise control trial after 9 to 9.5 hrs of reference sleep (REF), HIIE by treadmill running (90% and 40% of VO2 reserve in 3:2 min ratio) to expend 500 kcals after reference sleep (REF+EX) and HIIE after 3 to 3.5 hrs of short and disrupted sleep (SDS+EX) in a randomized crossover design. Blood samples were obtained by the same technician under standardized conditions just before, immediately after (IPE), 1 hr after exercise (1 HR) and just before a high-fat meal - 1240 kcals (56 g fat; 145 g carbohydrate; 38 g protein) and again 2, 4 and 6hrs after meal ingestion and at equal intervals during REF. Ultrasound measurements of brachial artery flow-mediated dilation (FMD) were obtained in a similar manner just before, 1 hr and 4 hrs after exercise. Total, high-density and low-density lipoprotein cholesterol (HDLC and LDLC) and paraoxonase-1 concentration were measured up to 1 hr post-exercise. Post-prandial triglyceride was measured and area under the curves - total (AUCT) and incremental (AUCi) were calculated. Lipid, lipid-related antioxidant, and FMD responses were analyzed using 3 (condition) by 3 (sample point) repeated measures ANOVAs. AUCT and AUCi were measured using one-way, 3 (condition) repeated measures ANOVAs. RESULTS: HDLC (+6.3%, p = 0.0023) and paraoxonase-1 (+10.8%, p <0.0001) increased and triglyceride (-18.5%, p <0.0001) decreased after REF+EX and SDS+EX. AUCT and AUCi remained refractory to exercise and short, disrupted sleep. FMD was augmented 1 hr after exercise in REF+EX (pre-exercise = 12.5 ± 0.9; 1 hr = 17.2 ± 1.5; 4 hr = 12.5 ± 0.9%) and SDS+EX (pre-exercise = 14.9 ± 1.7; 1 hr = 19.3 ± 2.2; 4 hr = 16.2 ± 2.4%) versus no change in REF (pre-exercise = 12.6 ± 1.4; 1 hr = 11.3 ± 1.0; 4 hr = 13.5 ± 2.1%) (p < 0.0494 condition by time interaction). CONCLUSION: Exercise transiently increased brachial artery FMD, fasting HDL cholesterol, and related antioxidant concentrations and reduced triglyceride levels, but did not modify total or incremental triglyceride AUC in response to a post-exercise high-fat meal. Exercise effects on blood lipids and vascular function were not influenced by a single night of short, disrupted sleep.