

## 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  $31.1 \pm 5.3$  yr; weight  $83.5 \pm 11.4$  kg; BMI  $25.8 \pm 2.7$  kg/m<sup>2</sup>; VO<sub>2</sub>max  $49.1 \pm 8.5$  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 VO<sub>2</sub>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 (AUC<sub>t</sub>) and incremental (AUC<sub>i</sub>) were calculated. Lipid, lipid-related antioxidant, and FMD responses were analyzed using 3 (condition) by 3 (sample point) repeated measures ANOVAs. AUC<sub>t</sub> and AUC<sub>i</sub> 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; AUC<sub>t</sub> and AUC<sub>i</sub> remained refractory to exercise and short, disrupted sleep. FMD was augmented 1 hr after exercise in REF+EX (pre-exercise =  $12.5 \pm 0.9$ ; 1 hr =  $17.2^* \pm 1.5$ ; 4 hr =  $12.5 \pm 0.9$ ) and SDS+EX (pre-exercise =  $14.9 \pm 1.7$ ; 1 hr =  $19.3^* \pm 2.2$ ; 4 hr =  $16.2 \pm 2.4$ ) versus no change in REF (pre-exercise =  $12.6 \pm 1.4$ ; 1 hr =  $11.3 \pm 1.0$ ; 4 hr =  $13.5 \pm 2.1$ ) ( $p < 0.0494$  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.