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Role of physical activity in obesity and type 2 diabetes-induced vascular dysfunction in heart

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The purpose of this study was to investigate whether physical activity initiated with the start of high-fat feeding would primordially prevent development of endothelial dysfunction, and if it does, then to determine some potential mechanisms. C57BL/6 female mice were randomly divided into three groups: 1) control low-fat diet (LF-SED; 15% of calories from fat), 2) high-fat diet (HF-SED; 45% of calories from fat), and 3) HF diet given access to a voluntary running wheel (HF-RUN). Our hypothesis was that HF-RUN would differ in multiple markers of endothelial dysfunction from HF-SED after 10 weeks of 45%-fat-diet, but would did not differ from LF-SED. HF-RUN differed from HF-SED in nine determinations in which HF-SED either had decreases in 1) Acetylcholine (ACh)-induced and flow-induced vasodilation in isolated, pressurized coronary arterioles, 2) heart phosphorylated endothelial nitric oxide synthase (p-eNOS/eNOS) protein, 3) coronary arteriole leptin (ob) receptor protein, 4) phosphorylated signal transducer and activator of transcription 3 (p-STAT3/STAT3) protein, and 5) coronary arteriole superoxide dismutase 1 protein; or had increases in 6) % body fat, 7) serum leptin, 8) coronary arteriole suppressor of cytokine signaling 3 (SOCS3) protein, and 9) coronary arteriole gp91phox protein. Higher endothelium dependent-vasodilation by ACh or leptin was abolished with incubation of NOS inhibitor NG-nitro-L-arginine-methyl ester (L-NAME) in LF-SED and HF-RUN groups. Further, impaired ACh-induced vasodilation in HF-SED was normalized by apocynin or TEMPOL to LF-SED and HF-RUN. These findings demonstrate multiple mechanisms (eNOS, leptin and redox balance) by which voluntary running opposes the development of impaired coronary arteriolar vasodilation during simultaneous high-fat feeding.

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