

Acute moderate intensity exercise induces vasodilation through an increase in nitric oxide bioavailability in humans.

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BACKGROUND: Long-term moderate-intensity exercise augments endothelium-dependent vasodilation through an increase in nitric oxide (NO) production. The purpose of this study was to determine the effects of different intensities of acute exercise on hemodynamics in humans. **METHODS:** We evaluated forearm blood flow (FBF) responses to different intensities of exercise (mild, 25% maximum oxygen consumption [VO₂max]; moderate, 50% VO₂max; and high, 75% VO₂max; bicycle ergometer, for 30 min) in eight healthy young men. The FBF was measured by using a strain-gauge plethysmography. **RESULTS:** After exercise began, moderate-intensity exercise, but not mild-intensity exercise, promptly increased FBF from 2.8±1.1 mL/min/100 mL to a plateau at 5.4±1.6 mL/min/100 mL at 5 min (P<.01) and increased mean arterial pressure from 84.7±11.8 mm Hg to a plateau at 125.7±14.3 mm Hg at 5 min (P<.01). Moderate-intensity exercise decreased forearm vascular resistance (FVR) from 29.2±5.4 to 16.8±3.2 mm Hg/mL/min/100 mL tissue (P<.01). The administration of NG-monomethyl-L-arginine, an NO synthase inhibitor, abolished moderate exercise-induced augmentation of vasodilation. Although we were not able to measure FBF during high-intensity exercise because of large body motion, high-intensity exercise markedly increased mean arterial pressure from 82.6±12.2 to 146.8±19.8 mm Hg. High-intensity exercise, but not mild-intensity or moderate-intensity exercise, increased plasma concentration of 8-isoprostane, an index of oxidative stress, from 24.1±10.8 to 40.2±16.7 pg/mL (P<.05) at 10 min after the end of exercise.

CONCLUSIONS: These findings suggest that acute moderate-intensity exercise induces vasodilation through an increase in NO bioavailability in humans and that high-intensity exercise increases oxidative stress.

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