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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