Although regular exercise reduces the risk of cardiovascular diseases, post-exercise recovery is thought to be a vulnerable phase for an increased susceptibility to sudden cardiovascular events. Cigarette smoking has been demonstrated to amplify sympathetic activation and cardiovascular stress. Paradoxically, many smokers tend to smoke immediately after leisure-time physical activity or exercise.

**PURPOSE:** We tested the hypothesis that smoking immediately after exercise would deteriorate autonomic and hemodynamic recovery following an acute bout of aerobic exercise compared with the sham smoking control.

**METHODS:** Ten healthy male smokers (ages 21-43 yrs; BMI = 24.7±3.9 kg/m²) participated in two trials in a randomized order: 1) cigarette smoking immediately after exercise (SM), 2) sham cigarette smoking after exercise (SHAM). All subjects exercised on a treadmill at a moderate intensity (40-60% of heart rate reserve) for 30 minutes and smoked one cigarette (0.6 mg nicotine) or sham cigarette immediately after exercise. We measured heart rate, brachial and central artery blood pressures, rate-pressure product, carotid-femoral pulse wave velocity (PWV), brachial artery flow-mediated dilation (FMD), and heart rate variability time domains. All variables were measured at baseline and at 10 minutes and 30 minutes after exercise, except for FMD measured at baseline and 30 minutes after exercise.

**RESULTS:** Rate-pressure product was significantly higher in the SM trial compared to the SHAM trial (interaction effect; p = 0.008). Central systolic and diastolic blood pressure increased in the SM trial (interaction effect; p = 0.026, p = 0.006, respectively). PWV was higher post–exercise in the SM trial, but this did not reach statistical significance (p = 0.116). FMD increased only in the SHAM trial (p = 0.008). SDNN decreased from baseline more so in the SM trial (SM: 51.5 ± 24.5 ms to 23.6 ± 15.7 ms to 36.3 ± 18.9 ms) compared with the SHAM trial (SHAM: 56.8 ± 28.1 ms to 43.1 ± 17.2 ms to 50.8 ± 22.8 ms; p = 0.041 for interaction).

**CONCLUSION:** Cigarette smoking immediately after exercise deteriorates autonomic and hemodynamic recovery in smokers, suggesting that smoking immediately after leisure-time physical activity or exercise should be avoided to reduce the susceptibility of sudden cardiovascular events.

**Postexercise Hypotension, Aortic Pressure, and AutonomicModulation In Men Living WithHIV**


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(A no relationships reported)

A single session of aerobic exercise reduces blood pressure vs. pre-exercise, which is referred as postexercise hypotension (PEH). Changes in cardiac autonomic control and local vasodilation contribute to PEH. HIV-infected patients present higher risk of hypertension, autonomic and endothelial dysfunction, which may influence the PEH. However, this phenomenon has never been studied in this population.

**PURPOSE:** To investigate the effects of acute aerobic exercise upon systemic blood pressure, aortic pressure, and cardiac autonomic modulation in men living with HIV.

**METHODS:** After cardiopulmonary exercise testing, 10 HIV-infected (HIV: 47.5 ± 9.7 yrs; 25.2 ± 3.0 kg/m²) and 14 healthy men (CTL: 40.1 ± 10.5 yrs; 25.8 ± 3.4 kg/m²) underwent cycling bout extending 150 kcal at 50% oxygen uptake reserve (time to achieve 150 kcal - HIV: 24.1 ± 5.5 and CTL: 23.1 ± 3.0 min) and control sessions (20 min), in a randomized counterbalanced order. Systolic blood pressure (SBP), aortic pressure, and heart rate variability were assessed 30 min before and 60 min after each session, by means of oscillometric digital monitor, pulse wave reflection (tonometry), and beat-to-beat heart rate intervals, respectively. Comparisons within-between sessions were made using 2-way ANOVA with repeated measures (p ≤ 0.05).

**RESULTS:** No difference was detected between groups for maximal oxygen uptake (HIV: 27.3 ± 4.2 vs. CTL: 31.4 ± 6.8 mL·kg⁻¹·min⁻¹; p = 0.1) and SBP at rest (HIV: 117.2 ± 11.6 vs. CTL: 112.2 ± 8.9 vs. mmHg; P = 0.2). Resting aortic pressure was higher in HIV (107.0 ± 9.3 mmHg) vs. CTL (100.0 ± 4.3 mmHg; P = 0.03), while standard deviation of NN intervals (SDNN) was lower in HIV (28.3 ± 11.2 ms) vs. CTL (43.9 ± 20.8 ms; P = 0.04). In CTL, SBP (-9.3 ± 5.9 mmHg; P = 0.01), aortic pressure (-6.3 ± 4.6 mmHg; P = 0.03), and SDNN (-23.4 ± 44.5 ms; P = 0.05) decreased after submaximal exercise vs. control sessions. No significant change occurred in HIV for SBP (-4.2 ± 18.9 mmHg; P = 0.5), aortic pressure (-5.1 ± 13.0 mmHg; P = 0.2), or SDNN (+5.5 ± 25.6 ms; P = 0.4).

**CONCLUSION:** Healthy, but not HIV-infected men, exhibited acute blood pressure reduction after submaximal aerobic exercise. The higher central arterial stiffness and lower vagal modulation among HIV patients may help to explain the absence of PEH in this group. Supported by FAPERJ Grant.

**Fibromyalgia Patients Display Blunted Cardiovascular Responses During Repeated Exercise Stress**


(A no relationships reported)

Fibromyalgia syndrome (FMS) affects nearly 10 million people in the United States and an estimated 6% of the world’s population. FMS is idiopathic and characterized by severe pain (typically in joints and musculature), fatigue, and malaise. Pain and fatigue may limit physical activity, but other factors such as post-exertional malaise, may also contribute.

**PURPOSE:** To examine exertional and post-exertional cardiovascular responses.

**METHODS:** Thirty-five patients with fibromyalgia and 8 sedentary controls performed two cardiopulmonary exercise tests (CPET) to maximal exertion separated by 24 hours. Heart rate (HR) was measured continuously via ECG and blood pressure (BP) was recorded every two minutes. Independent samples T-tests compared differences between FMS patients and sedentary controls. Multiple linear regressions observed the effects of FMS on cardiovascular statistics (HR, SBP, Rate pressure product; RPP) at anaerobic threshold (AT) and VO2 max (peak), controlling for confounding variables (age, sex, BMI, workload, and any additional medical conditions).

**RESULTS:** Patients were 44.6±9.8 years old, 27.5±6.1 kg/m², and 88.4% female. FMS and sedentary controls did not differ in age or BMI. FMS and sedentary controls did not differ in VO2peak (p = 0.62), workload (p = 0.29), SBP (p = 0.44), DBP (p = 0.989), RPP (p = 0.05) during test 1. At AT, FMS did not influence HR (β = -3.71, p = 0.53), SBP (β = 3.94, p = 0.67), or RPP (β = -1.478, p = 0.35) at test 1, but did at test 2 for HR (β = -20.69, p = 0.003) and RPP (β = -5.035, p = 0.003). When comparing test 1 to test 2 with the same variables, FMS influenced both HR (β = -15.956, p = 0.001) and RPP (β = -3.2273, p = 0.01), but not SBP (β = -3.60, p = 0.686). At peak, FMS influenced HR (β = -23.80, p = 0.012) and RPP (β = -5.0786, p = 0.040) for test 1, but did not influence SBP (β = -2.86, p = 0.786). This was also observed during test 2 for HR (β = -23.30, p = 0.004), RPP (β = -7.373, p = 0.008), and SBP (β = -13.27, p = 0.294). When comparing test 1 to test 2 at peak, FMS did not influence HR (β = -0.31, p = 0.974), or RPP (β = -2.453, p = 0.175), but did influence SBP (β = -14.72, p = 0.029).

**CONCLUSION:** Post-exertional effects blunt the cardiovascular responses to exercise in FMS. This post-exertional effect has not been clearly elucidated in this illness and may help in understanding the illness.

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