

The Effects of Combined Exercise Training on Flow-Mediated Dilation and C-Reactive Protein in Overweight, Postmenopausal Women

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ABSTRACT

Obesity-related inflammation, especially coupled with a decline in estrogen post menopause, leads to endothelial dysfunction where the endothelium is unable to adequately dilate in response to sheer stress. C-Reactive Protein (CRP) may cause endothelial dysfunction through superoxide production and downregulation of endothelial nitric oxide synthase leading to decreased nitric oxide production. Exercise training improves endothelial function through adaptations to increase nitric oxide bioavailability and regulation of superoxide production. **PURPOSE:** To determine the influence of acute and chronic combined resistance and aerobic exercise on flow-mediated dilation (FMD) and CRP in overweight to obese, postmenopausal women, as well as determining potential relationships between these variables. **METHODS:** Overweight to obese (BMI $33.01 \pm 4.60 \text{ kg}\cdot\text{m}^2$), postmenopausal women ($64.3 \pm 5.3 \text{ yr}$) were randomized into either an exercise (EX, $n = 20$) or education control (ED, $n = 18$) group for a 12-week intervention where EX underwent moderate intensity aerobic and resistance exercise training (25 minute treadmill walking 70-80% VO_2max ; 8 resistance exercises, 2 sets at 8-12 repetition maximum, respectively) 3 days per week and ED attended education sessions (talks on health, CPR certification, etc.) twice per week. Before (BT) and after (AT) the intervention, both groups underwent an experimental trial day where blood was collected before (PRE), immediately after (PO), 1 hour (1HR), and 2 hours (2HR) post exercise in EX and at the same time points for resting ED. FMD was performed PRE and 2HR for EX and at the same time points for ED. FMD was analyzed blinded using Brachial Analyzer for Research (Medical Imaging Applications, LLC; Coralville, IA). EDTA-plasma was used for CRP analysis via Quantikine® ELISA (R&D Human C-Reactive Protein/CRP Immunoassay, catalog no. DCRP00). **RESULTS:** Acute exercise improved % FMD by 2HR with BT and AT collapsed and with no change in ED (EX: PRE 9.72 ± 0.48 , 2HR 11.2 ± 0.51 %; ED: PRE 9.46 ± 0.51 , 2HR: 8.83 ± 0.53 %; $p = 0.04$). There was no significant effect of exercise training in EX (BT PRE 9.54 ± 0.70 , AT PRE 9.86 ± 0.71 %) or education session in ED (BT PRE 9.88 ± 0.73 , AT PRE 9.04 ± 0.75 %; $p = 0.33$). CRP did not change in response to exercise training (EX BT PRE 3.87 ± 0.59 , AT PRE $3.51 \pm 0.62 \text{ ng/mL}$; ED BT PRE 4.37 ± 0.62 , AT PRE $4.63 \pm 0.65 \text{ ng/mL}$; $p = 0.112$). There was a significant positive correlation between FMD and CRP both at BT and AT PRE ($p = 0.035$, $r = 0.37$; $p = 0.038$, $r = 0.37$, respectively). **CONCLUSION:** Acute exercise increased FMD 2 hours after exercise, suggesting that there is a residual effect of exercise on FMD up to at least 2 hours in our population. Resting FMD and CRP did not significantly improve in response to the 12-week moderate intensity aerobic and resistance exercise training program. The duration and intensity of the exercise training program may not have been adequate to result in significant changes in CRP and endothelial function as determined via FMD.