

## Acute Exercise-Induced Response of Platelet-Monocyte Complexes in Obese, Postmenopausal Women

MICHAEL M. LEVITT<sup>1</sup>, MARIA A. CARDENAS<sup>1</sup>, BRYAN RICHIE<sup>1</sup>, CARMEN A. COOK<sup>1</sup>, SHAOHAN LU<sup>1</sup>, KARA L. STECK<sup>1</sup>, JAY HAYNES<sup>2</sup>, ANDREAS KREUTZER<sup>1</sup>, JOEL B. MITCHELL<sup>1</sup>, and MELODY D. PHILLIPS<sup>1</sup>

<sup>1</sup>Exercise Physiology Lab; Kinesiology; Texas Christian University; Fort Worth, TX

<sup>2</sup>John Peter Smith Health Network; Fort Worth, TX

---

Category: Masters

Advisor / Mentor: Phillips, Melody D. (m.phillips@tcu.edu)

### ABSTRACT

Inactivity-related diseases such as cardiovascular disease (CVD) are linked to chronic low-grade, systemic inflammation. Platelet-monocyte complexes (PMCs) are markers of *in vivo* platelet activation and atherosclerosis, and may be early indicators of subclinical inflammation. **PURPOSE:** To examine the effects of an exercise bout on PMCs in those at risk for CVD. **METHODS:** Twenty-five overweight-obese (BMI  $32.7 \pm 5.2$  kg·m<sup>-2</sup>, 55-75 yr) women were randomly assigned to either the exercise (EX, n=13) or non-exercise control (CON, n=12) group. EX performed 2 sets of 8 resistance exercises and a 25-min treadmill walk at 70-80% HRR. Blood was obtained pre-exercise (PR), post- (PO), 1-hour and 2 hours post-exercise (1HR and 2HR). Blood was obtained at the same time points in CON. PMCs were identified via flow cytometry and analyzed in each monocyte phenotype. Monocyte phenotypes were defined as: Mon1 (CD14<sup>+</sup>CD16<sup>-</sup>CCR2<sup>+</sup>), Mon2 (CD14<sup>+</sup>CD16<sup>+</sup>CCR2<sup>+</sup>), and Mon3 (CD14<sup>+</sup>CD16<sup>+</sup>CCR2<sup>-</sup>). All events positive for both CD14 and CD42a (marker for platelets) were considered PMCs. **RESULTS:** A main effect for time revealed an increase in PMC number at PO ( $p=0.036$ ) which appears to have been driven by EX (EX = 61.5%; CON = 33.8% increase). PMCs formed with Mon1 and Mon2 followed a similar response. A significant group x time interaction for Mon3 PMC number ( $p=0.002$ ) indicated an increase from PR to PO (PR =  $5218 \pm 1170$ , PO =  $8195 \pm 1152$  cells ml<sup>-1</sup>), and a decrease from PO to 1HR and 2HR (1HR =  $3767 \pm 820$  cells ml<sup>-1</sup>, 2HR =  $3818 \pm 814$  cells ml<sup>-1</sup>) in EX. PMC number remained constant for CON at all timepoints. Estimated VO<sub>2</sub>max was negatively correlated with CD42a MFI (a marker of platelet density per monocyte) ( $r = -0.583$ ,  $p = 0.003$ ). Systolic blood pressure (SBP) positively correlated with percent PMC (% CD42a positive monocytes;  $r = 0.458$ ,  $p = 0.042$ ). **CONCLUSION:** Aerobic fitness appears to reduce platelet activation indicated by the negative relationship between VO<sub>2</sub>max and CD42a MFI. Chronic elevations in resting SBP are linked to PMC percentage, possibly due to sheer stress-induced platelet activation. It is possible that PMC elevation at PO is at least partially driven by exercise-induced increases in BP. These results support previous literature, indicating that PMCs are a CVD risk marker and may elucidate one mechanism by which physical fitness reduces risk for CVD.