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THE INFLUENCE OF AN ACUTE EXERCISE BOUT ON MONOCYTE PHENOTYPE AND CIRCULATING PLATELET-MONOCYTE COMPLEXES IN FIT INDIVIDUALS

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KEYWORDS: platelet-monocyte complexes, monocyte phenotype, acute exercise, inflammation

PURPOSE

The purpose of this study was to examine the distribution of three monocyte phenotypes and the formation of platelet-monocyte complexes (PMC) following an aerobic exercise bout.

METHODS

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Apparently healthy men and women ($n=7$, age= 22.9 ± 5.1) performed a 20 min treadmill run at 85% heart rate maximum followed by five min rest and a maximal graded treadmill test ($VO_2\text{max}=50.3 \pm 8.4 \text{ ml}\times\text{kg}^{-1}\times\text{min}^{-1}$). Blood was obtained pre-exercise (PRE), immediately post-exercise (PO) and at 1hr and 2hr post-exercise (1HR-PO and 2HR-PO). In addition, each subject performed a control trial on a separate day by resting in the lab. Blood was collected at the same time points as the exercise trial to control for diurnal variation. Subjects did not exercise for the 36 hours prior to all testing. PMCs and monocyte phenotype were identified via flow cytometry (FACSCelesta, BD Biosciences). Monocyte phenotypes were categorized by the following cell surface receptors: Classical (Mon1)

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(CD14+CD16–CCR2+), Intermediate (Mon2) (CD14+CD16+CCR2+), and Non-classical (Mon3) (CD14^{lo}CD16++CCR2–). Mon4 was defined as all CD16+ monocytes (both Mon2 and Mon3). All events positive for both CD14 and CD42a (a receptor on platelets) were considered platelet-monocyte complexes.

RESULTS

As anticipated, exercise induced leukocytosis immediately post-exercise (EX: PRE=5.74±0.62, PO=8.63±0.90; CON: PRE=5.98±0.61, PO=5.64±0.54 $\times 10^6$ cells $\times$ ml⁻¹; $p=0.045$) and at 2HR-PO (EX: 2HR-PO=8.50±0.37; CON: 2HR-PO=6.643±0.55 $\times 10^6$ cells $\times$ ml⁻¹; $p=0.013$) compared to the control trial. In addition, the number of circulating platelets increased PO exercise (240± 10.3 vs. 287± 22.3 $\times 10^6$ cells $\times$ ml⁻¹, $p=0.045$). The percentage of circulating Mon2 monocytes increased 1HR-PO exercise (15.1±4 vs. 19.6±8.3 %, $p=0.050$) compared to the control trial. In addition, Mon2 monocytes decreased at the 1HR timepoint in the control trial, most likely due to diurnal variation (17±2.8 vs. 3.4±2.2 %, $p=0.026$). Mean fluorescence intensity (MFI), a measure of cell receptor density, of CD16 decreased on all CD14+ monocytes (EX: PRE=13,903±2266, 2HR-PO=5845±521; CON: PRE=10,888±2619, 2HR-PO=13704±3017; $p=0.027$) and on all CD16+ monocytes (Mon4) (EX: PRE=16,999±2840, 2HR-PO=6775±683; CON: PRE=13,814±3654, 2HR-PO=20,126±5346; $p=0.033$) in the exercise condition compared to the control condition. No significant changes in the percentage of circulating PMCs (PRE=9.67±2.18, PO=9.57±1.22, 1HR-PO=10.39±0.99, 2HR-PO=8.76 %, $p>0.05$) or the number of PMCs (PRE=2.64±0.63, PO= 4.06±1.25, 1HR-PO=2.79±0.38, 2HR-PO=2.83±0.60 $\times 10^6$ cells $\times$ ml⁻¹, $p>0.05$) were observed despite increases in platelet number post-exercise.

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CONCLUSION

Despite shifts in monocyte phenotype and platelet number, acute exercise did not appear to have an impact on the formation of PMCs in apparently healthy, fit individuals. This is in contrast to previous findings which show increases in PMC formation immediately post-exercise and decreases in PMC formation at 1HR post-exercise. However, previous findings were in older, often untrained or diseased adults and/or the exercise intensity was not as strenuous as that employed here.