The Influence of Physical Activity and Monocyte Phenotype on Circulating Platelet-Monocyte Complexes in Overweight/Obese Persons

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ABSTRACT

Elevated platelet-monocyte complexes (PMC) promote atherosclerosis and are associated with cardiovascular disease. It is unknown whether consistent physical activity (PA) decreases circulating PMCs. Additionally, no one has determined the monocyte phenotype most associated with PMCs. PURPOSES: 1) to examine the influence of PA on PMCs and their association with inflammatory /prothrombotic markers such as C-reactive protein (CRP), L-selectin (LS), platelet factor 4 (PF4), von Willebrand Factor (vWF), and hemoglobin A1C (HbA1c) and 2) to determine the monocyte phenotype most likely to form PMCs. METHODS: Thirty-one overweight/obese subjects (44±5yr, BMI 34.2±5 kg·m^2) were divided into two groups: sedentary (SED, n=17) and physically active (PA, n=14) based on physical activity logs. SED participated in < 1 h of formal exercise while PA participated in moderate-high intensity exercise at least 3 h per week. Flow cytometry was used to identify PMCs on the monocyte phenotypes: classical (CD14+CD16-), intermediate (CD14+CD16+), and non-classical (CD14+CD16++). Platelets were identified using the marker CD42a. RESULTS: Percentage of circulating PMCs and median fluorescence intensity of CD42a (MedFI; marker of platelet density per monocyte) were not different between groups; however, monocyte phenotype significantly impacted PMC percentage and MedFI where the lower the CD16 expression, the greater the adhesion of platelets. Classical monocytes (CD16$^-$) had the highest % of PMC, etc. (Fig 1). HbA1c was greater (p=0.031) and LS (p=0.019) was lower in SED compared to PA (Fig. 2). There were no significant associations between any blood marker and PMC percentage, but PF4 was correlated with percent of CD16 - (r= -0.482, p=0.031) and CD16+ (r= 0.473, p=0.035) monocytes.

Fig. 1 Phenotype	**Percent PMC**	**MedFI**
CD16-	32 ± 3.2^a	184 ± 15^{abc}
CD16 + & ++	25 ± 2.6^a	159 ± 12^{ac}
CD16+	28 ± 3.1^a	163 ± 14^b
CD16++	17 ± 2.1^a	146 ± 11^c
Values with same letters are different from each other (p < 0.001)		

Fig. 2 Dependent Variable	**PA**	**SED**	***p* Value**
VO_2 max (ml·kg^{-1}·min^{-1})	30 ± 6	27 ± 6	0.307
HbA1c (%)	5.5 ± 0.9	5.8 ± 0.5	0.031*
CRP (mg·L^{-1})	8.1 ± 1.3	6.6 ± 1.3	0.347
L-Selectin (ng·mL^{-1})	1087 ± 89	844 ± 48	0.019*
PF4 (µg·mL^{-1})	1.52 ± 0.2	1.34 ± 0.2	0.584
vWF (µg·mL^{-1})	32 ± 3	24 ± 3	0.057

CONCLUSIONS: The absence of a separation between groups in VO_2max may partially explain the lack of a difference in PMCs between groups. Regarding our second aim, classical monocytes appear to be more involved in PMC formation than do CD16+ monocytes with CD16++ having the lowest percentage of cells with platelets adhered (PMC). This observation may be due to the shedding of adhesion molecules from platelets and monocytes during activation from classical (CD16-) to a more inflammatory state (ie. CD16+).