TACSM Abstract

Influence of Fitness and Adiposity on Melanocortin-1 and Melanocortin-3 Receptors on Monocytes

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ABSTRACT

Purpose: While it is known that exercise improves health by reducing systemic inflammation, potential mechanisms remain to be elucidated. The purpose of this study is to examine the influence of fitness and adiposity on the anti-inflammatory melanocortin system as a potential mechanism by which exercise reduces inflammation. **Methods:** Forty-one men and women (35-55yr), who were free from cardiovascular disease, inflammatory disorders, and not taking medications that affect inflammation, were recruited. Participants were questioned about exercise habits and medical history; then completed testing for body composition and aerobic fitness (VO_2max). Subjects were classified as lean/fit (LF, n=14), overweight/fit (OF, n=8), lean/sedentary (LS, n=5), or overweight/sedentary (OS, n=14). Overweight was defined as a body mass index (BMI) over 27.5 kg/m^{-2}, while lean was a BMI between 18.5-24.9 kg/m^{-2}. Fitness was defined by the accumulation of at least 4 hours of moderate-high intensity exercise every week, with a maximal oxygen consumption of at least 47 ml/kg lean mass/min. Sedentary individuals were defined as those who accumulate less that 1 hour of exercise every week, with maximal oxygen consumption less than 40 ml/kg lean mass/min. Those that met classification criteria returned to the lab at least 1 week later for a blood draw. Blood was analyzed using flow cytometry for cell surface expression of melanocortin receptor-1 (MC1R), and melanocortin receptor-3 (MC3R). Monocytes were gated using forward and side scatter. Four populations were differentiated based on CD14 and CD16 expression (classical monocytes = CD14$^{++bright}$/CD16$^{-negative}$, low pro-inflammatory= CD14^{+dim}/CD16^{+dim}, high pro-inflammatory I= CD14$^{++bright}$/CD16$^{++bright}$, and high pro-inflammatory II= CD14^{+dim} /CD16$^{++bright}$ **Results:** Mean fluorescence intensity (MFI) of MC1R and MC3R was significantly different among monocyte populations ($p \leq 0.01$). CD14$^{++bright}$/CD16$^{-negative}$ monocytes expressed the least MC1R (118.79±8.85 MFI) and MC3R (119.03±8.37 MFI), CD14^{+dim}/CD16^{+dim} expressed the second most MC1R (185.17±23.74 MFI) and MC3R (231.24±26.47 MFI), CD14$^{++bright}$/CD16$^{++bright}$ expressed the third greatest MC1R (335.44±51.6 MFI) and MC3R (631.75±62.03 MFI), and CD14^{+dim} /CD16$^{++bright}$ expressed the greatest MC1R (479.78±67.10 MFI) and MC3R (1178.03±86.34 MFI). Although there was no significant difference for MC1R or MC3R based upon fitness or body composition, there was a trend for greater MC1R expression on CD14^{+dim}/CD16$^{++bright}$ monocytes in LF compared to OS ($p=0.052$). **Conclusion:** Neither fitness or adiposity had a significant effect on melanocortin receptor expression. MC receptor expression, however, appears to be linked to that of CD16. Cells expressing higher levels of CD16 also expressed higher levels of MC1R and MC3R, indicating that as pro-inflammatory phenotype increases so do melanocortin-related anti-inflammatory regulators.

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