TACSM Abstract

The Effects of Physical Activity on Markers of Adipose Inflammation during Weight Cycling

RACHAEL E. HARTKE, MATTHEW P. HARRIS, MARCUS BREDEN, KAYLEE E. POOLE, ANDREW SEIJA, AND JOSHUA S. WOOTEN

Exercise Physiology and Biochemistry Laboratory; Department of Applied Health; Southern Illinois University Edwardsville; Edwardsville, IL

Category: Undergraduate

Advisor / Mentor: Wooten, Joshua (jwooten@siue.edu)

ABSTRACT

Weight loss using diet and exercise are the main treatment strategies for obesity; however, weight loss is rarely maintained resulting in weight regain or weight cycling. Obesity is associated with chronic low-grade inflammation resulting in the release of adipokines and activation of macrophages (M1) accelerating the development of insulin resistance. In contrast, the M2 macrophage phenotype is characterized by blocking inflammatory responses and promoting tissue repair. Despite the effectiveness of exercise on preventing comorbidities of obesity during weight-loss, the influence of physical activity during weight cycling on markers of adipose inflammation remains unclear. PURPOSE: The purpose of this study was to determine the role of physical activity on the expression of inflammatory markers in adipose tissue during weight cycling. METHODS: Male C57BL/6 mice were randomly assigned to one of three groups for 28 weeks: a high-fat diet obese control (HFD; 60% kcal from fat), an alternating high-low-high fat diet group (Diet; 60%/10%/60% kcal from fat) to simulate weight cycling, or a diet-matched weight cycling group that had unrestricted access to running wheels (Diet+PA). After weight regain, MCP-1, CD11c, CD163, F4/80, TLR4, and TNFα mRNA levels were quantified in perigonadal adipose tissue using qRT-PCR. A one-way ANOVA was used to identify significant differences between groups with significance set at $P<0.05$. RESULTS: Weight cycling without physical activity resulted in obesity and insulin resistance when compared to HFD obese controls. Interestingly, compared to the HFD control group, the Diet group demonstrated significantly greater expression of F4/80 (+50%), CD11c (+113%), TLR4 (+77%), and TNFα (+72%) mRNA, which may represent greater macrophage infiltration and M1 macrophage polarization. Physical activity during weight cycling resulted in lower weight regain compared to both HFD and Diet groups; however, mice still developed insulin resistance and increased expression of TLR4 (+76%), TNFα (+94%), and CD11c (+58%) suggesting increased M1 macrophage activation when compared to the HFD group. CONCLUSIONS: The data presented suggests weight cycling may accelerate the development of adipose dysfunction, and unrestricted physical activity appears to have minimal effects on the negative inflammatory effects of weight cycling.