

High Fat Diet Induced Obesity Impairs Skeletal Muscle Glycogen and Lipid Preservation After Adiponectin Incubation

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ABSTRACT

High fat diets have been attributed as critical factors contributing to obesity and type 2 diabetes, characterized by increased lipid accumulation, impaired glucose uptake, and defects in glycogen storage in skeletal muscle. Adiponectin, an adipokine, has been shown to have antidiabetic effects by improving fat oxidation and glucose uptake. **PURPOSE:** To investigate the effects of adiponectin incubation, in high fat diet induced obese rats, on measures of skeletal muscle substrate metabolism including glycogen, glucose transporter 4 (GLUT4), lipid, and mitochondrial contents. **METHODS:** Male Sprague Dawley rats were fed a Western-style (21% fat by weight; 41% total energy) high fat diet for 9 weeks to induce obesity, then, for 6 weeks, either continued the Western Diet (WD) or were fed a standard Chow Diet (WCD) (4.8% fat; 0.74% saturated; 2% mono; 1.77% poly); a control group followed a 15-week chow diet (CD). Following the 15-week diet intervention, right and left hind-leg extensor digitorum longus (EDL) muscles were incubated in an organ bath (Krebs-Henseleit buffer containing 2000 mg/L glucose) with or without 0.1 mg/ml adiponectin for 30 minutes. Glycogen content was measured with periodic acid-schiff staining, GLUT4 (ab654), lipid (bodipy), and mitochondrial (ab14744) contents were measured using immunohistochemical techniques and quantified with imageJ software. **RESULTS:** There were no changes in substrate concentrations with adiponectin incubation in any diet group. However, the change (Δ) in glycogen, with adiponectin, was greater in animals fed a control chow diet compared to animals that followed a 15-week high fat Western-style diet (WD) (change (Δ); CD: 0.11 ± 0.07 vs. WD: -0.25 ± 0.14 ; one-way ANOVA $p=0.048$). When change in lipid content, after adiponectin treatment, was compared, animals fed high fat diets showed impaired preservation of lipid compared to the control group (change (Δ); CD: 25.9 ± 11.2 vs. WD: -21.1 ± 14.8 $p=0.02$; CD: 25.9 ± 11.2 vs. WCD: -18.8 ± 7.7 $p=0.02$). **CONCLUSION:** 9 weeks of a high fat Western-style diet is sufficient to induce defects in skeletal muscle substrate concentrations, including glycogen and lipid contents. Although adiponectin incubation did not increase substrate concentrations, a control chow diet demonstrated a better ability to preserve glycogen and lipid contents compared to high fat diet induced obese rats. Despite reverting to a standard chow diet, high fat diet induced obese rats did not demonstrate an insulin-sensitizing response from adiponectin incubation.