



Effect of an acute bout of aerobic exercise on chemerin levels in obese adults



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SUMMARY

Aims: Serum chemerin concentrations are elevated in obese individuals and may play a role in type 2 diabetes. Exercise improves insulin sensitivity, which may be related to changes in chemerin. This study explored how an acute bout of aerobic exercise affected chemerin levels in non-diabetic obese adults. **Methods:** Blood samples from 11 obese adults were obtained during two separate conditions: sedentary (SED) and exercise (EX; 60–65% $\text{VO}_{2\text{peak}}$). Samples were drawn at baseline, immediately following exercise and hourly for an additional 2 h. ANOVA was used to test for differences in chemerin between conditions.

Results: Unadjusted analysis showed no difference in overall change (baseline to 2 h post) in chemerin between conditions. During the 2-h post-exercise period, chemerin decreased to 12% below baseline, compared to a 2.5% increase above baseline during that time period on the sedentary day ($p = 0.06$, difference in post-to-2 h change between conditions). Controlling for homeostatic model assessment of insulin resistance (HOMA-IR), a significant difference existed between EX and SED in the change in chemerin from baseline to 2-h post ($p = 0.02$). Stratified analyses showed a consistent exercise-induced decrease in chemerin among non-insulin resistant subjects, while chemerin increased during exercise among insulin resistant subjects, and then decreased post-exercise.

Conclusion: An acute bout of exercise in obese individuals may elicit a drop in chemerin levels during the post-exercise period, and this response may vary based on insulin resistance.

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1. Introduction

Chemerin is an adipokine that has been shown to be elevated in obese individuals and associated with the metabolic impairments of type 2 diabetes mellitus (T2DM). The precise role of chemerin in the pathophysiology of diabetes remains unclear, although its positive association with BMI and obesity [1–3] and its regulatory

function in adipocyte differentiation *in vitro* [4] have been demonstrated.

Chemerin acts as a ligand for the G-protein coupled receptor CMKLR1, which is highly expressed in adipose tissue [1,4–6]. Chemerin was recently shown to regulate glucose uptake in both adipose tissue and skeletal muscle, which suggests its potential role in the development of T2DM [5–9]. It was found that Stimulation of cultured 3T3-L1 cells with the pro-inflammatory cytokine interleukin 1-Beta (IL1- β) enhanced chemerin expression and decrease insulin-stimulated glucose uptake [7]. Chemerin treatment in obese mice worsened glucose intolerance by decreasing serum insulin concentrations and glucose uptake in the liver and adipose tissue [10]. Further, Goralski et al. (2007) supported chemerin's necessary contribution to adipocyte development by showing that knocking down chemerin by adenoviral siRNA led to

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impaired 3T3-L1 pre-adipocyte differentiation, as well as reduced expression of adipocyte genes involved in glucose and lipid homeostasis. These findings suggest that adipocytes are both releasing and utilizing chemerin in an autocrine manner. Takahashi et al. (2008), on the other hand, found that chemerin incubation restored insulin sensitivity by 41% in cultured 3T3-L1 adipocytes via improved insulin-stimulated glucose uptake and insulin receptor substrate (IRS)-1 tyrosine phosphorylation. The implications of those findings, however, are debatable given that glucose uptake into adipocytes could be interpreted as both preventing hyperglycemia and facilitating lipogenesis.

Both acute and chronic exercise enhance glucose uptake and increase insulin action [11,12]. Several studies demonstrated an association between chronic exercise and both diminished chemerin levels and enhanced insulin action [13–17]. Acute bouts of exercise were also associated with immediate enhancements in insulin action, as evidenced by improvements in whole body glucose disposal [18], insulin-stimulated skeletal muscle glucose transport [19], and GLUT-4 translocation [20] following a single exercise session. We theorize that the acute effects of exercise on insulin sensitivity may be related to acute decreases in circulating chemerin following a single bout of exercise. Thus, the purpose of this study was to determine whether serum chemerin levels would decrease following an acute exercise bout in obese individuals.

2. Materials and methods

2.1. Subjects

This study was a secondary analysis of data and biological samples from a completed crossover study of the effects of exercise on satiety and circulating hormone levels [21]. The original study and the current analysis were approved by the Syracuse University Institutional Review Board and all subjects ($n = 11$) signed an informed consent document prior to participation in the study. Inclusion criteria were: age between 18 and 35 years (mean = 25.3 ± 4.3) and BMI $>30 \text{ kg/m}^2$ (mean = 33.8 ± 4.0). Exclusion criteria included weight loss or gain of ≥ 5 lbs. three months prior to the study, diagnosed gastrointestinal problems, or diagnosed orthopedic limitations to normal walking activity. Subjects could not be using any glucose-lowering medications or other medications that may affect glucose metabolism (e.g. antidepressants, hormonal contraceptives, steroid hormones). Indices of resting blood pressure (Omron HEM automatic blood pressure monitor, Omron, Kyoto, Japan), cardiovascular disease (medical questionnaire), and lipid profile (Cholestech LDX, Cholestech Corporation, Hayward, CA) were evaluated in order to ensure a healthy subject cohort. Specifically, resting blood pressure of 140/90 mm/Hg, total cholesterol of 200 mg/dL, and low density lipoprotein cholesterol of 160 mg/dL were used as cut-offs for exclusion. Female subjects ($n = 3$) were tested within the first eight days of their menstrual cycle to minimize possible hormonal effects on glucose and chemerin levels.

2.2. $\text{VO}_{2\text{peak}}$ assessment

Aerobic capacity was assessed as previously described [22]. Briefly, $\text{VO}_{2\text{peak}}$ was measured during a gradual treadmill exercise stress test using breath by breath analysis performed with a metabolic cart (True One 2400, ParvoMedics, Sandy, UT). Expired gases and heart rate were analyzed during the test and $\text{VO}_{2\text{peak}}$ was calculated. Subjects initially walked at a pace of 2.5 miles per hour (mph) and a 0% grade, with speed increased by increments of 0.5 mph per stage until a speed of 3.5 mph was achieved at minute six. Starting at minute eight, the workload increased 2% per stage until the subject reached volitional fatigue. Criteria for a successful test were determined in accordance with [23].

During the initial visit, each subject's habitual dietary intake as well as general health and physical activity were recorded using Institutional Review Board (IRB) approved questionnaires. Individuals were measured for height and weight and body composition was assessed using air-displacement plethysmography (BODPOD system, Life Measurement, Inc., Concorde, CA) according to manufacturer's specifications.

2.3. Study protocol

This study was a crossover design in which each subject served as their own control. For the original study, each subject reported to the Syracuse University Human Performance Lab on two separate occasions for twelve hours of testing, beginning at 0700 h. [21]. Subjects participated in the sedentary (SED) and exercise (EX) protocols in random order with a minimum 7-day period between conditions.

Subjects were fasted and had abstained from caffeine consumption for 12 h, and abstained from alcohol consumption and structured exercise for at least 24 h prior to testing. Upon arrival to the lab on testing days the subjects had a Teflon catheter inserted in their antecubital vein. On the exercise day, blood samples were drawn immediately prior to exercise for baseline reference, immediately following the cessation of exercise (post), one hour post-exercise (1 h), and two hours post-exercise (2 h). The exercise protocol consisted of one hour of walking at an intensity corresponding to 60–65% of $\text{VO}_{2\text{peak}}$. Blood samples were drawn at the same one-hour intervals on the sedentary day.

As part of the original study, liquid meals were administered immediately after the first and third blood draws during both conditions. All meals were matched for energy content (15% protein, 65% carbohydrate, 20% fat; Wegmans Nutritional Beverage, Wegmans, Rochester, NY), with the majority of carbohydrate coming from sucrose and corn syrup, and the majority of protein from soy and whey. All of the subjects were required to remain in the laboratory and participated in quiet, sedentary activities including reading, studying, playing board games, and watching movies, with extremely limited physical activity (e.g. walking to restroom) outside of the designated exercise period.

2.4. Blood analysis

Blood samples were obtained and transferred to serum separator tubes (BD Vacutainer, Franklin Lakes, NJ), separated by centrifugation, divided into two sets of polypropylene tubes, and stored at -80°C for subsequent analysis. Samples were assayed in duplicate for serum glucose using a commercially available glucose oxidase assay (Sigma-Aldrich Corp., St. Louis, MO). A second set of samples were briefly centrifuged ($3000 \times g$, 5 min, 4°C) and assayed for serum insulin concentrations using Luminex xMap Technology (Linco Research, St. Charles, MO) on a Luminex 100/200 platform (Luminex Corporation, Austin, TX). All procedures followed manufacturer's instructions (Millipore, Billerica, MA) with quality controls assayed within expected ranges. Inter-assay and intra-assay coefficients for insulin were 4.9% and 8.5%, respectively. The lowest limits of detection in this assay were 137 pg/mL.

Blood plasma collected from all subjects was analyzed in duplicate to assess circulating chemerin levels. The Enzyme-linked Immunosorbent Assay (ELISA) was performed according to the manufacturer's instructions (BioVendor LLC, Candler, NC). Briefly, unbound sights were blocked to prevent false positive results with BSA. The chemerin antibody was added to the appropriate wells, followed by a secondary IgG conjugated antibody. The colorimetric

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