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ORIGINAL ARTICLE

Serum chemerin and insulin sensitivity alterations due to exercise training below and above lactate thresholds in streptozocin-induced diabetic rats

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Abstract The aim of this study was to investigate serum chemerin and insulin sensitivity alterations due to exercise training below and above lactate thresholds in streptozocin-induced diabetic rats. Twenty-four rats (190 ± 14 g) were randomly assigned to three groups of control (CON), above lactate thresholds (ALT) and below lactate thresholds (BLT) ($n = 8$). Then after induction of diabetes, BLT and ALT performed an 8-week exercise program on treadmill running (15 and 27 m/min, respectively) at 0° slope, 1 h/day, and 5 days/week. SPSS version 16 was used for data analysis. The findings showed that, BLT serum chemerin decreased −27 % compared to CON that was statistically significant ($p = 0.042$), fasting Glucose level of ALT was significantly decreased 19 % compared to CON after 8-week training protocol ($p = 0.02$). Also 24 % significant reduction was observed in BLT in comparison to CON ($p = 0.005$). Insulin sensitivity of BLT and ALT improved 7 and 5 %, respectively, compared to CON and both were not significant. Also, positive correlationship was observed between fasting plasma glucose and serum chemerin but it was not significant statistically. Negative correlationship was seen between plasma insulin level and chemerin concentrations and it was not significant too. Compared with CON, BLT weight was significantly reduced 12 % ($p = 0.012$). Also 9 % weight loss was observed in ALT due to 8-week exercise training which was not statistically significant ($p = 0.058$). In summary, 8-week exercise training below lactate thresholds may reduce serum chemerin and improve fasting glucose levels.

Keywords Lactate thresholds · Training · Chemerin · Insulin sensitivity

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Introduction

Chemerin is a protein playing a role in chemotaxis and originates from the liver and the adipocytes that induce their differentiation such as chemotaxis and triggering dendritic cells and macrophages [1]. Chemerin regulates activation of dendritic cells and macrophages by some intracellular factors such as the G protein-coupled receptors CMKLR1 (ChemR23), GPR1, and CCRL2 [2]. Nevertheless, it has been proved that chemerin modulates adipocyte differentiation in an autocrine/paracrine manner and stimulates the expression of adipocyte genes participating in lipid and glucose metabolism [3]. In muscle cells, chemerin accelerates insulin resistance (IR) by inducing lack in insulin receptor signaling and glucose uptake [4]. Furthermore, Becker et al. [5] recently reported that elevation in expression of chemerin induces IR in the skeletal muscle of low-density lipoprotein receptor knockout mice on high-fat diet. Utilization of exogenic chemerin exacerbates glucose tolerance consequently; decrease insulin concentrations; and belittle glucose uptake of tissue in ob/ob, db/db, and diet-induced obese but not lean and normoglycemic mice [5]. Exercise training is main and prevalent methods of negative energy balance that impresses glucose metabolism, lessens fat storage, and ameliorates IR [6]. In animal studies, it has been indicated that exercise causes impressive improvement in insulin sensitivity. This action is carried out by