Effect of 8 weeks concurrent training on L-AFBP in obese men

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ABSTRACT

The aim of this study was to examine the effect of 8 weeks concurrent training on plasma liver fatty acid binding protein (L-AFBP) in obese men. Twenty three middle aged obese men (aged: 34.6 ± 8.8 years; ± SD) were randomly assigned to one of the concurrent training group (n=12) or control group (n=11). The concurrent group performed endurance and resistance training on the same days, 3 days a week for 8 weeks. Body mass, Body mass index (BMI) and waist hip ratio (WHR) were decreased (P<0.05) after 8 weeks concurrent training compared to the control group. The results also showed that L-AFBP was decreased (P<0.05) after 8 weeks concurrent training compared to the control group; however, for aspartate transaminase (AST), alanine transaminase (ALT) and C-reactive protein (CRP) no significant changes were observed after the intervention. In conclusion, serum L-AFBP levels were decreased by 8 weeks concurrent training in obese men.

KEYWORDS

Concurrent training, L-AFBP, AST, ALT, CRP

Introduction

Fatty acid-binding proteins (FABPs) are a class of cytoplasmic proteins that bind long chain fatty acids. FABPs are small intracellular proteins (~13-14 kDa) with a high degree of tissue specificity [1]. They are abundantly present in various cell types and play an important role in the intracellular utilization of fatty acids, transport and metabolism. There are at least nine distinct types of FABP, each showing a specific pattern of tissue expression. Due to its small size, FABP leaks rapidly out of ischemically damaged necrotic cells leading to a rise in serum levels. Ischemically damaged tissues are characterized histologically by absence (or low presence) of FABP facilitating recognition of such areas [2].

Liver-type fatty acid binding protein (L-FABP, FABP1) is predominantly expressed in liver. The L-FABP protein is derived from the human FABP1 gene. L-FABP is a sensitive marker for cell damage of liver cells in vitro and in vivo. L-FABP is also a marker for rapid hepatocyte lysis in vitro (as
for example in toxicology assays) and for detection of liver damage during and after transplantation [3].

Exercise is an important therapeutic strategy to reduce the metabolic effects of obesity, especially liver damage [4]. Although the changes in L-FABP levels might be an important clue for understanding the beneficial effects of exercise, a little data on exercise-induced changes of L-FABP have been reported. Hiraki et al. (2013), in an only available study on human, reported that L-FABP level did not significant changes after a single 20-min moderate-intensity exercise session [5], while Lira et al. (2010) noted that L-FABP level was decreased after 8 weeks endurance exercise with 60% \( \text{VO}_2\text{max} \) in rats [6].

Today serum tests of acute hepatocellular injury are commonly used to investigate the presence and monitor the progress of liver disease [7]. We hypothesized that exercise training would reduce the inflammatory markers and improves body composition and decrease the markers of liver damage such as L-FABP, AST and ALT concentrations; therefore, we investigated the effects of 8 weeks of concurrent training on body composition, CRP and L-FABP concentrations in obese men.

**Methods**

**Subjects**

Twenty three sedentary obese middle aged men with a mean (± SD) body mass index of 32.9 ± 2.4 kg/m², volunteered to participate in a 8 weeks concurrent training study. All the subjects were asked to complete a personal health and medical history questionnaire, which served as a screening tool. The subjects were given both verbal and written instructions outlining the experimental procedure, and written informed consent was obtained. The study was approved by the Islamic Azad University, Fars Science & Research branch Ethics Committee. The subjects were randomly assigned to one of the concurrent training group (n=12) or control group (n=11).

**Exercise protocol**

The concurrent training group performed 20-30 min endurance exercise at an intensity corresponding to 70-85% individual maximum heart rate and then performed circuit weight training per day. Resistance training was circularly performed in 8 stations and included 2-4 sets with 8-12 maximal repetitions at 65-80% of 1-RM in each station. Each circuit and set was separated by 2-3 min and 30 s rest respectively. The intervention was performed 3 days a week for 8 weeks.

**Measurements**

**Anthropometric and body composition measurements**

Height and weight were measured, and body mass index (BMI) was calculated by dividing weight (kg) by height (m²). Waist circumference was determined by obtaining the minimum circumference (narrowest part of the torso, above the umbilicus) and the maximum hip circumference while standing with their heels together. The waist to hip ratio (WHR) was calculated by dividing waist by hip circumference (cm) (ACSM, 2005).

**Biochemical analyses**

Fasted, resting morning blood samples (10 ml) were taken at the same time before and after 8 weeks intervention. All the subjects fasted at least for 12 hours and a fasting
blood sample was obtained by venipuncture. Serum obtained was frozen at -80 °C for subsequent analysis. The plasma L-FABP, CRP, AST and ALT levels were measured in duplicate using an enzyme-linked immunosorbent assay (ELISA) kits (Pars Azmoun, Tehran, Iran).

**Statistical analysis**

Results were expressed as the mean ± SD and distributions of all variables were assessed for normality. Paired t-test was used to compute mean (± SD) changes in the variables in control and concurrent training group pre and after the intervention. Differences among groups were assessed by using analysis of covariate (ANCOVA) test. The level of significance in all statistical analyses was set at P<0.05. Data analyses were performed using SPSS software for windows (version 17, SPSS, Inc., Chicago, IL).

**Results and Discussion**

Physical characteristics of the subjects at baseline and after training are presented in Table 1. Before the intervention, there were no significant differences in any of variables among the two groups. Body mass and BMI and WHR were increased (P<0.05) after 8 weeks concurrent training compared to the control group. The results also showed that L-FABP was decreased (P<0.05) after 8 weeks concurrent training compared to the control group; however, for AST, ALT and CRP no significant changes were observed after the intervention. No significant relationship was observed between A-FABP with BMI, body fat percentage and insulin resistance.

Excessive levels of free fatty acids are toxic to cells. The human body has evolved a defense mechanism in the form of small cytoplasmic proteins called fatty acid binding proteins (FABPs) that bind long-chain fatty acids, and then refer them to appropriate intracellular disposal sites (oxidation in mitochondria and peroxisomes or storage in the endoplasmic reticulum) [8,9]. It is postulated that FABPs play an important role in the pathogenesis of metabolic diseases. Elevated levels of L-FABP were associated with liver damage [10]. Our results demonstrated that L-FABP levels were decreased after 8 weeks concurrent training (P<0.05). Although previous studies demonstrated that increase of L-FABP concentration is a sensitive marker for cell damage of liver [3], a little data on exercise-induced changes of L-FABP have been reported. In agreement with our results, Lira et al. (2010) showed that L-FABP level was decreased in rats after 8 weeks endurance exercise on treadmill with 60% VO₂max for 60 min/day and 5 days/week [6]. However, Hiraki et al. (2013) reported that L-FABP level did not significant changes after a single 20-min moderate-intensity exercise session [5]. These discrepant results may be attributed to differences in subject populations and exercise protocol. Our subjects were obese men while adult outpatients with chronic kidney disease were participated in Hiraki et al. study. On the other hand, we examine the effect of 8 weeks concurrent training on L-AFBP while Hiraki et al. studies the changes of L-FABP concentrations after a single moderate-intensity exercise session. Previous study demonstrated that a significant positive relationship between FABPs levels with BMI and waist circumference in obese subjects [11]. Our results demonstrated that anthropometric and body composition improved after the intervention, thus exercise-induced changes in body mass, BMI and WHR may play a part in decreasing circulation L-FABP. The present study also demonstrated that AST,
ALT and CRP levels were not significant changes after the intervention and there were no significant relationships between these variables and L-FABP level. Therefore, it seems that other parameters such as exercise *per se* might decrease L-FABP concentrations. Additional research is needed to examine these mechanisms.

**Table 1** Anthropometric characteristics (mean ± SD) of the subjects before and after the training

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<th>Control (mean±SD)</th>
<th>Concurrent training (mean±SD)</th>
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<td>Pretraining</td>
<td>Posttraining</td>
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<tr>
<td>Body mass (Kg)</td>
<td>106.6 ± 4.7</td>
<td>106.5 ± 7.6</td>
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<td>98.08 ± 6.9</td>
<td>94.5 ± 7.4*†</td>
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<tr>
<td>BMI (Kg/m²)</td>
<td>34.1 ± 2.5</td>
<td>34.1 ± 3.3</td>
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<td>31.8 ± 1.7</td>
<td>30.6 ± 1.4*†</td>
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<td>WHR</td>
<td>0.95 ± 0.03</td>
<td>0.95 ± 0.04</td>
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<td></td>
<td>0.96 ± 0.04</td>
<td>0.94 ± 0.04*†</td>
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*: P<0.05 for between-group differences.
†: P<0.05, pretraining vs. posttraining values.

**Table 2** Biochemical characteristics (mean ± SD) of the subjects before and after the training

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<th>Control (mean±SD)</th>
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<td>Pretraining</td>
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<tr>
<td>L-FABP (ng/L)</td>
<td>371.03 ± 302.7</td>
<td>340.6 ± 277.2</td>
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<td></td>
<td>325.5 ± 208.8</td>
<td>219.0 ± 120.1*†</td>
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<td>AST (IU/L)</td>
<td>26.9 ± 12.4</td>
<td>28.6 ± 14.3</td>
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<td>25.6 ± 10.0</td>
<td>25.1 ± 5.0</td>
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<td>ALT (IU/L)</td>
<td>19.09 ± 8.6</td>
<td>17.4 ± 9.7</td>
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<td>17.1 ± 6.6</td>
<td>14.6 ± 4.2</td>
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<tr>
<td>CRP (mg/mL)</td>
<td>2.1 ± 0.3</td>
<td>2.7 ± 0.7</td>
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<td>2.0 ± 0.2</td>
<td>2.2 ± 0.5</td>
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*: P<0.05 for between-group differences.
†: P<0.05, pretraining vs. posttraining values.

**Acknowledgement**

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