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IMPROVED FASTING GLUCOSE BY AEROBIC TRAINING PROGRAM IS INDEPENDENT OF INSULIN RESISTANCE

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ABSTRACT

Accumulating evidence indicates that obesity and overweight are associated with insulin resistance and type II diabetes. In present study, we aimed to evaluate the effect of an aerobic training program in adult obese men, for this purpose, twenty four sedentary adult men (age 34 ± 3.96 year, weight 101 ± 14 kg, height 177 ± 5 cm) were selected to participate in study and divided into exercise or control groups by randomly. Exercise group were completed three months aerobic exercise for 3 times per weeks. Pre and post training fasting blood samples were collected of all subjects of two groups. Blood samples used for measuring insulin and glucose concentration. Insulin resistance was assessed using the homeostasis model assessment for insulin resistance formula derived from fasting insulin and glucose levels. Data were evaluated using, paired t and Pearson's tests. Exercise program resulted significant decrease in anthropometrical and fasting glucose concentration in exercise group. No significant differences were found in serum insulin and insulin resistance by exercise program with compared to baseline. Benefit effects of exercise training on fasting glucose in obese subjects could not be only attributed to insulin resistance

Keywords: *Insulin Resistance, Aerobic Training, Glucose*

INTRODUCTION

Obesity is one of the most common diseases of this century and is associated with hypertension, atherosclerosis, hepatic and Billiary diseases, and respiratory and cardiovascular diseases (Maedler *et al.*, 2009). Among preventable diseases, obesity-induced deaths are next to tobacco-induced deaths, so that the overall lifetime of obese people is averagely 10.5 years less than non-obese individuals. Scientific resources have introduced obesity as a risk factor for type 2 diabetes; so that based on a credible report in recent years, more than 30-40 percent of people with type 2 diabetes are obese (Lazar, 2005). Apart from fat mass, increased accumulation of visceral fat and impaired function of adipose tissue is associated with insulin resistance (Klötting *et al.*, 2010). Literature has pointed out the fact that effective mechanisms in creating positive balance of calorie, such as inflammation and release of adipokine, are pathophysiologic indicators of metabolic abnormalities in diseases associated with obesity (Klötting *et al.*, 2010).

Obesity and increased body fat stores are the main causes of insulin resistance syndrome, type 2 diabetes, and atherosclerosis (Gustafson, 2010). Scientific resources have supported the role of exercise in improving blood glucose levels and insulin resistance and have mentioned that weight loss and reduction of body fat content are effective factors in improvement of blood glucose in obese people. But it is unclear whether the reduction or improvement in blood glucose in response to regular exercise is dependent only on changes in insulin resistance or improvements in blood glucose take place in the absence of changes in insulin resistance or insulin levels. This study was carried out to investigate the effect of three months of aerobic exercise on fasting glucose levels and insulin resistance and to determine their relationship after the exercise program.

MATERIALS AND METHODS

Study Subjects

The objective of present study was to determine effects of long term exercise training on fasting glucose, insulin and insulin resistance in obese men. Study included twenty four non-trained healthy males (age 34

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± 3.96 year, weight 101 ± 14 kg, height 177 ± 5 cm) that participated by voluntarily, then divided into exercise (n=12) or control (n=12) groups by randomly. All participants gave their informed written consent before participation in accordance with the ethical guidelines set by Islamic Azad University. Participants were non-smokers and non-alcoholics. All subjects were obese. Neither the control or exercise subjects had participated in regular exercise for the preceding 6 months, nor did all subjects have stable body weight. Participants had no evidence of coronary artery disease; impaired fasting glucose or diabetes, recent myocardial infarction, congestive heart failure, active liver or kidney disease, growth hormone deficiency or excess. Exclusion criteria also included medications that alter carbohydrate metabolism and inability to exercise.

Anthropometrical and biochemical variables were measured before exercise program and repeated 48 after lasted exercise session. Anthropometric measurements (body height and weight, waist and hip circumference) were performed with the subjects wearing light underwear and without shoes. Waist and hip circumferences were measured at the level of umbilicus and of trochanter major, respectively. Waist to hip circumference ratio was measured by dividing the abdominal circumference into that of the hip. Body mass index was measured for each individual by division of body weight (kg) by height (m²).

Laboratory Measurements and Exercise Program

Before and 48 hours after the aerobic training program, venous blood samples were obtained after 10-12 overnight fast in order to measuring insulin and glucose concentration in two groups. Insulin resistance was assessed using the homeostasis model assessment for insulin resistance formula derived from fasting insulin and glucose levels (Matthews *et al.*, 1985). Glucose was determined by the oxidase method (Pars Azmoon, Tehran). After sampling in EDTA- or serum-tubes, blood was immediately chilled on ice, centrifuged and aliquots were frozen at -80°C until assayed. Insulin was determined by ELISA method (Demeditec, Germany) and the intra- assay and inter-assay coefficient of variation of the method were 2.6% and 2.88 respectively. Aerobic exercise training was performed for three months (3 times per week, %60-80HRmax). Each session started flexibility exercises, then exercise for 30-45 min included running on treadmill and 5–10 min of cool down activity.

Statistical Methods: Statistic analysis was done with SPSS 16.0 for Windows. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. After calculation of the mean and the standard deviation, the statistical analysis was conducted using unpaired T-test to compare all variables in exercise with the control group. Paired t test was used to determine the mean differences between pre and post-training values on all metabolic and anthropometric variables. All statistical tests were performed and considered significant at a $P \leq 0.05$.

RESULTS AND DISCUSSION

Pre taining and post training of the descriptive anthropometric and biochemical features of the study groups are shown in Table 1. At baseline (pre-training), there were no differences in the age, body weight or other anthropometrical markers between the two groups (see Table 1). Fasting insulin and glucose and insulin resistance were also similar in exercise and control groups at baseline ($p \geq 0.05$).

Table 1: Baseline and post training levels of anthropometrical and clinical characteristics of two groups

Variables	Exercise group		Control group	
	pre-training	post-training	Pre-training	post-exercise
Weight (kg)	101 ± 14	96 ± 15	100 ± 13	101 ± 9
AC (cm)	108 ± 10	103 ± 10	107 ± 11	107 ± 8
Hip (cm)	108 ± 9	104 ± 9	107 ± 12	108 ± 9
AHO (Ratio)	1.0 ± 0.027	0.99 ± 0.031	1.0 ± 0.032	1.0 ± 0.086
BMI (kg/m ²)	32.17 ± 3.41	30.58 ± 3.53	31.89 ± 2.64	31.69 ± 3.22
Body fat (%)	31.92 ± 4.08	27.62 ± 2.42	32.06 ± 3.61	32.69 ± 4.21
Insulin (-)	9.08 ± 1.80	9.63 ± 1.89	9.23 ± 2.11	9.14 ± 1.69
Insulin resistance	2.26 ± 0.44	2.15 ± 0.51	2.34 ± 0.36	2.51 ± 0.42
Glucose (mg/dl)	101 ± 9	90 ± 10	103 ± 14	100 ± 10

AC, Abdominal circumference; BMI, body mass index; AHO, Abdominal to hip circumference ratio

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Compared to pre-training, BMI, body fat (%), body weight, abdominal circumference and other anthropometrical markers decreased significantly ($p < 0.05$) after aerobic training program but these variables were not changed in control group. Aerobic exercise program resulted significant decrease ($p = 0.004$) in exercise group (Figure 1). Serum insulin ($p = 0.216$) and insulin resistance ($p = 0.356$) did not change by exercise program in exercise group.

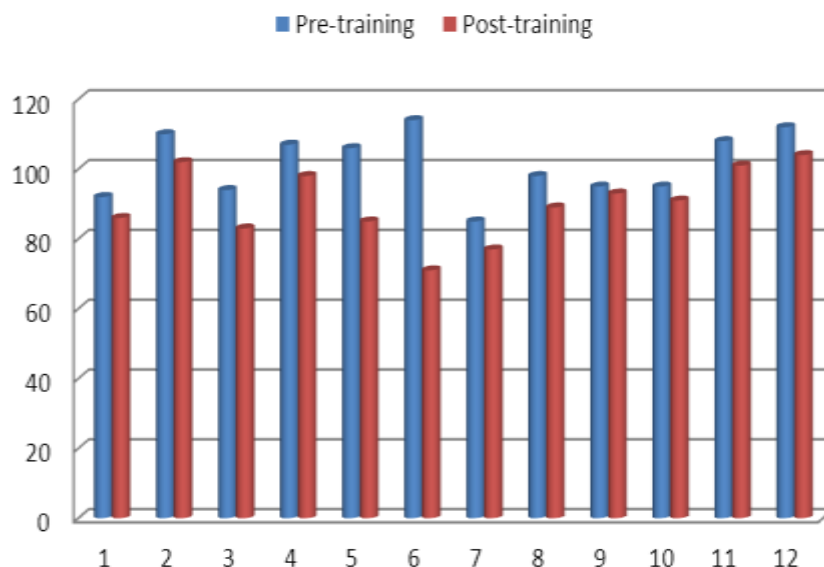


Figure 1: Glucose levels in pre and post training of exercise group. Aerobic training resulted significant decrease in glucose in exercise group

Discussion

In the present study it was found that three months of aerobic exercise has significantly reduced fasting glucose levels compared to baseline in obese adult males, although no significant changes occurred in insulin levels and insulin resistance. Exercise program has also reduced significantly body fat percent and any of the anthropometric indices such as BMI. Obesity is associated with increased stores of fatty acid in adipose tissue and has been closely associated with development of insulin resistance in tissues such as liver and skeletal muscles (Galic *et al.*, 2010). Recent epidemiological studies have shown that the pathophysiologic mechanisms, such as increased inflammation, endothelial dysfunction, and imbalance in homeostasis of peptide mediators, result in damage to beta cells of the pancreas and diseases associated with obesity such as type 2 diabetes, insulin resistance, and cardiovascular disease (Goldberg, 2009).

It is well known that obese people, either healthy or patient, have higher levels of fasting glucose and insulin resistance than normal weight people (Sigal *et al.*, 2006). Previous studies have always mentioned that beta cells of the pancreas secrete more insulin in response to increased resistance of target cells or decreased sensitivity of cells to insulin in order to cope with insulin resistance and to maintain blood glucose level (Kriketos *et al.*, 2004). It should also be noted that increased production and secretion of insulin to compensate for insulin resistance is a temporary adjustment and continuation of this situation will lead to hyperactivity of beta cells and their decreased function in long-term (Kriketos *et al.*, 2004). Loss of mass or decreased function of beta cells in response to long-term overcompensation of insulin secretion for maintaining blood glucose levels during increased insulin resistance is far more visible in type 2 diabetes. However, the literature has revealed that an appropriate pattern of food intake and regular and continuous exercise is associated with improved blood glucose levels, particularly in obese or diabetic individuals (Pradhan *et al.*, 2003; Pan *et al.*, 1997). In support of these observations, a three-month aerobic exercise in present study has led to a significant reduction in blood glucose in obese men.

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On the other hand, although blood glucose levels were significantly decreased in response to aerobic exercise, no significant changes were observed in insulin resistance. Lack of improvement in insulin resistance was observed while the three-month exercise program led to significant decrease in anthropometric indices and body fat content. Based on these observations, it seems that apart from insulin resistance, improvement in blood glucose levels after the exercise program is related to other unknown mechanisms. For example, although pre- or anti-inflammatory cytokines were not measured in the present study, which is one of its limitations, recent scientific resources have mentioned the effective role of these cytokines including adiponectin or some interleukins in maintaining the homeostasis of blood glucose (Halle *et al.*, 1999; Meuller *et al.*, 1998). Some other studies have also supported the importance and potential role of certain cytokines such as leptin, adiponectin, and other anti-inflammatory cytokines in energy homeostasis and fat and glucose metabolism (Hu *et al.*, 1996; Van Gaal *et al.*, 1999; Snehalatha *et al.*, 1999; Trujillo *et al.*, 2005). In addition, some scientific resources have attributed the improvement and reduction in blood glucose levels following a regular exercise program to the improvement in the levels of these cytokines in response to exercise (Kim *et al.*, 2007). Indeed, the main limitation of this study was lack of measurement of the mentioned cytokines. Furthermore, some previous studies have suggested that the response of inflammatory cytokines and some other mediators subsequent to exercise is independent of changes in insulin function or insulin resistance (Marcell *et al.*, 2005; Fatouros *et al.*, 2005).

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