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Effect of Three Months Aerobic Training on Glucose Concentration and Insulin Action in Obese Females

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ABSTRACT: It is generally accepted that obesity is associated with insulin resistance and metabolic syndrome. In this study, we aimed to determine beta cell function on response to three months exercise program in obese/overweight women. To achieve this outcome, twenty six adult obese/overweight women matched for age 38 ± 3 year and body weight 82 ± 7 kg were participated in this study and selected into exercise or control group by randomly. All participants were non-trained and non-pregnancy. Subjects in exercise group was completed a three months aerobic training program. Before and after exercise program, fasting glucose and insulin were measured of two groups. Anthropometrical markers were also measured before and after program. Paired-samples T tests were employed for comparison of corresponding variables within two groups. A P-value of < 0.05 was considered to be statistically significant. There were no statistically significant differences in all variables between exercise and control groups at baseline. Fasting glucose levels were significantly decreased after exercise program when compared to pretest in exercise subjects but not in control groups. Compared to pre-training, beta cell function increased significantly in exercise group but not in control subjects. Similar to improved insulin resistance that reported by many other studies, data of our study is also supports the beneficial property of long term aerobic training on beta cell function in obese/overweight individuals.

Keywords: Insulin action, Glucose homeostasis, obesity, Exercise training

INTRODUCTION

Over the past years, obesity has increasingly prevailed and is now one of the most serious public health problems in developed as well as developing countries. The incidence of obesity is associated with an increased incidence of some chronic diseases such as cardiovascular disease, metabolic syndrome, diabetes and diseases related to insulin resistance [1]. However, the mechanisms by which obesity effects chronic diseases are not yet fully understood. World Health Organization (WHO) mentions the rapid increase in the prevalence of obesity as an epidemic and considers obesity and its complications as a major health problem worldwide [2].

Throughout life, beta-cell function is impaired by certain reversible factors. Beta-cell dysfunction usually has a genetic basis [3] and accelerates the onset of diabetes [4]. In fact, the beta-cell function is impaired with aging [5], which comes along with impaired glucose tolerance and diabetes [6]. In addition, overweight and obesity accelerate the destruction of the beta cells [7]. As noted before, patients suffering from diabetes type 2, even those who are at a young age, are prone to cardiovascular risk factors. These findings have been observed in young men with a mean age of 22 years [8].

Studies on laboratory animals have shown that inadequate secretion of insulin leads to diabetes type 2 which is associated with decreased beta-cell function and levels caused by reduced proliferation and cell death [9]. Although temporary and moderate increase in insulin resistance leads to increased beta-cell mass [9, 10, 11], which is a result of cell hypertrophy, severe and long-term insulin resistance causes the reduction of beta-cell proliferation. So in response to prolonged insulin resistance, beta-cell mass levels are not be maintained [9]. Among external interventions, exercise has been introduced as an improving factor of insulin sensitivity and insulin function in insulin-resistant humans and animal models [12], although the effects of exercise on beta-cell mass and function has been less considered. Hence, the present study was conducted to determine the effect of a three-month program of aerobic exercise on blood glucose levels and beta-cell function in obese, non-athletic group of women.

MATERIAL AND METHODS

In present study, the effect of three months aerobic training on beta cell function and fasting glucose were measured in obese/overweight women. For this purpose, twenty six non-trained apparently healthy women matched for age (38 ± 3 years of old) and BMI ($26 \leq BMI \leq 36$) participated in this study by accidentally samples.

Participants were randomly assigned to either an aerobic training group (n = 13) or a control group (n = 13). Baseline physical characteristics are presented by group in Table 1. Participants were excluded if they had a chronic disease or had an orthopedic condition that would limit their ability to perform exercise.

Anthropometric measurements of height, weight, percent body fat, and circumference measurements were taken by the same trained general physician. Height was measured without shoes on standing while the shoulders were tangent with the wall. Body weight was measured in duplicate in the morning following a 12-h fast. Body mass index BMI was calculated as weight (kg) divided by squared height (m). Abdominal obesity and hip circumference were determined in a standing position at the end of normal expiration and ratio between them (AHO) was calculated for each subjects. Percentage body fat was measured using body composition monitor (OMRON, Finland).

A. Inclusion and exclusion criteria

All subjects were inactive, non-smoker and non-pregnancy. Subjects were currently participating in an organized physical activity training program over the previous 6 months, and were also excluded from the study. None of the subjects used drugs or therapies for obesity, and none had a past history of disease or injury that would prevent daily exercise. Potential participants were excluded from the study if they reported smoking or had a history of heart disease, stroke or were taking glucose-lowering medication.

B. Blood analysis and protocol

Blood samples were collected after a overnight fast before and after exercise program. The exercise program involved 45-60 min of aerobic exercise training, three times per week for 12 weeks. Each exercise session was supervised by an exercise physiologist or one of the study physicians. In each session, subjects completed a 5-10 min warm-up, followed by 60 min of aerobic exercise at 60-80%VO2max (with continuous heart rate monitoring). Each exercise sessions finished by 5-min cool down. Aerobic exercise involved running on a flat surface with no slope or treadmill. Adherence to the exercise prescription was documented through the use of Polar heart rate monitors, and subjects received feedback if training intensities were either too high or low in comparison with desirable intensities.

Blood samples were analyzed for fasting glucose and insulin. 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. Glucose was determined by the oxidase method (Pars Azmoon kit, Tehran). Beta cell function was assessed using the homeostasis model assessment formula derived from fasting insulin and glucose levels [13].

C. Data analysis

All analyses in the statistical evaluation were carried out with SPSS-13.0 software. Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. Comparisons between the means of each group were done using the independent t-test. Student's t-tests for paired samples were performed to determine whether there were signigcant within-group changes in the outcomes. The results were considered statistically significant for $p<0.05$.

RESULTS

We previous mentioned that this study was produced with aim to assess that effect of three month aerobic training for three time per week on beta cell function and fasting glucose in non-trained obese/overweight women. Pre and post training of anthropometrical markers are showed in Table 1. All values are reported as mean and standard deviation. At baseline, there were no differences in the age, body weight and other anthropometrical indexes between the two groups.

We observed a significant decline for body weight ($p = 0.003$) and other anthropometrical or body composition markers after exercise program when compared with pre test. Compared to pre-training, beta cell function increased significantly ($p = 0.015$) in exercise group but this variables was not changed in control group ($p = 0.913$) (Fig. 1). Fasting glucose levels were significantly decreased by aerobic training intervention when compared with pre test ($p = 0.001$), but not in control subjects ($p = 0.356$) (Fig. 2).

Table 1: Pre and Post training of anthropometrical markers in exercise and control group (M ± SD).

Variables	**Height** (cm)	**Weight** (kg)	**BMI** (kg/m^2)	**BF** (%)	**Abdominal** (cm)	**Hip** (cm)
Exercise group (*pre)*	159 ± 4	81.8 ± 6.79	32.3 ± 2.65	46.2 ± 3.68	110 ± 9.3	114 ± 7.1
Exercise group *(post)*	159 ± 4	79.4 ± 7.88	31.3 ± 3.13	44.7 ± 3.88	108 ± 8.3	110 ± 6.3
Control group *(pre)*	159 ± 5	81.8 ± 7.51	32.2 ± 2.87	46.3 ± 3.14	110 ± 7.1	114 ± 5.6
Control group *(post)*	159 ± 5	81.9 ± 7.44	32.2 ± 2.90	46.4 ± 2.97	111 ± 6.9	114 ± 5.2

BMI, body mass index; **BF,** Body fat percentage;

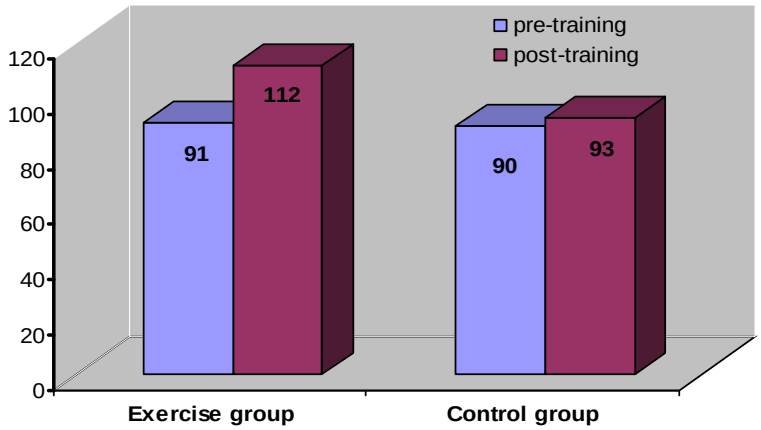

Fig 1: Pre and post training of Beta cell function of two groups.

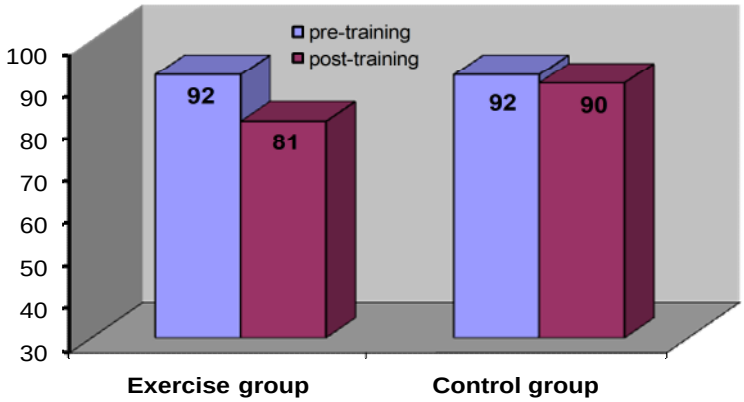

Fig. 2. Pre and post training of fasting glucose in two groups.

DISCUSSION

In this study, a three-month aerobic exercise significantly increased beta-cell function in obese adults. In support of these findings, the literature has raised the hypothesis that regular physical activity has a protective effect on pancreatic beta-cell function [14], though there are few studies on the subject and the direct effect of exercise on beta-cell adjustment to insulin resistance is still unclear.

It is known that a diet high in fat increases insulin resistance which leads to the incidence and severity of diabetes type 2 [15]. A fat diet which consists of 40 percent of fat leads to Hypertrophy of insulin and finally to an increase in insulin secretion to compensate for insulin resistance and is able to maintain the blood glucose levels at a normal rate [16, 17]. Although keeping a high-fat diet for a long course of time leads to an insufficient release of insulin due to beta-cell sensitivity and eventually leads to diabetes type 2 [16], yet, a diet moderate on fat maintains proper levels of insulin-secretion and proper beta-cell functioning [16]. Changes in insulin levels and insulin resistance are the same in both types of diabetes and metabolic syndrome which is, in most cases, resulted from a response to the consumption of foods high on sugar, in a way that, when blood glucose levels rise, the pancreas cells increase insulin secretion at doses higher than normal in order to lower blood glucose concentrations, the result of which is a sudden drop in blood sugar. The continuous bombardment of the target receptors by excessive secretion of insulin causes, in the long run, these receptors to become dependent on high insulin levels and accordingly decreases their sensitivity to insulin and finally increases insulin resistance [18].

Regular exercise reduces levels of visceral fat and body weight without reducing non-fat mass and increases insulin sensitivity and improves blood glucose levels [19]. In the present study, too, significant increase in beta-cell function was observed following three months of training which was associated with a significant reduction in fat indicators such as weight and body fat percentage or body-mass indicator.

As a result of exercise, not only insulin resistance but also glucose-dependent insulin secretion plays an important role in homeostasis Galuzi [20, 21], though the effects of exercise on insulin secretion are not permanent.

In response to obesity, especially visceral obesity, insulin sensitivity is reduced and is closely related to systemic hypertension and dyslipidaemia. These features are associated with metabolic syndrome which is all associated with the increased cardio-vascular risk factors [22]. But the present study shows that, in addition to increased beta-cell function and reduction of fasting glucose levels, weight loss induced by exercise in obese women is also associated with significant increases in high level lipoprotein (HDL). In obese mice, limited calorie [23], reduction in fat mass [24], and increased physical activity [25, 26], improve insulin sensitivity and inhibit the spread of hyperglysymy. These interventions that increase insulin sensitivity or function lead to a reduction in insulin demand from the pancreas and to the reduction of apoptosis of pancreas insulin beta cells [14].

Despite the fact that the effect of exercise on beta-cell function in diabetes type 2 has been in more focus, it has been less studied in healthy obese people. However, it is shown that beta cells in type 2 diabetic subjects and healthy individuals react in response to exercise [27], though the effects of exercise varies depending on the initial capacity of the beta cells. According to this evidence, it is expected that depending on the capacity of insulin secretion from Pancreas beta-cells, the effect of exercise on beta-cell function be different in different obese and diabetic populations and it is not necessarily expected that beta-cell response to exercise in non-diabetics with relatively balanced blood glucose be similar to that in diabetic individuals. Scientific studies have shown that exercise-induced muscles can increase beta-cell functions [28]. In this respect, also other scientific studies have shown that insulin-resistant muscles secrete peptides that can increase beta-cell mass or secretion [29, 30]. It is also possible that in the present study, the change in the secretion of these peptides in response to exercise has directly increased beta-cell function.

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