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Relationship between the Changes Pattern of Aerobic Capacity and Fasting Glucose in Response to Chronic Exercise Training

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ABSTRACT
Obesity is related to chronic diseases such as syndrome metabolic and type II diabetic. The aims of present study were 1) to evaluate effect long term aerobic exercise on physical fitness and some marker indicator of type II diabetic in healthy obese men, 2) to determine relation between the change pattern of aerobic capacity and the other mentioned variables in response to this aerobic exercise program.
For this purpose, a total thirty adult obese males participated in this study and were randomly divided into experimental or control groups. The experimental group subjects were participated in an aerobic exercise program (3 months / 3 times weekly) and control group did not participate in any exercise session. Aerobic capacity, fasting insulin and glucose were measured in two separate occasions in each 2 groups (before and after aerobic program). Insulin resistance was calculated with fasting glucose and insulin. Statistical analysis was performed using an independent paired t-test. Pearson correlation was used to establish the relationship between the changes pattern between variables in response to exercise program.
Aerobic training program resulted in significant increase in aerobic capacity (VO2max) and beta cell function and a significant decrease in fasting glucose in experimental group. The change in VO2max was inversely associated with the change in fasting glucose and directly associated with the change in beta cell function.
These data highlight the role cardiovascular or physical fitness in obesity-induced abnormalities such as glucose homeostasis and insulin action.
***Keywords**: Aerobic capacity, Glucose, Exercise, Obesity*

INTRODUCTION

Studies conducted on adults have shown that insulin resistance is independent predictor of a hypertension, coronary heart disease, heart attacks, type 2 diabetes and some cancers, and augmented insulin function is a special protective factor against the incidence of this type of diseases [1]. On the other hand, scientific sources state that obesity is associated with impaired insulin function [2]. Studies on non-diabetic obese prove that in response to insulin resistance, insulin release from pancreatic beta cells is increased and the continuous adaptation leads to increased insulin resistance and hyperinsulinemia phenomenon in obese individuals [3, 4]. Although a temporary increase in insulin resistance leads to increased beta cell mass [5, 6], long-term insulin resistance brings about reduced beta cell proliferation. As a result, in response to long-term insulin resistance beta cell mass is not preserved [6]. Longitudinal studies have shown that the progression of beta cell damage is especially important in the prevalence of diabetes in obese individuals [7]. Impairment of beta cell sensitivity to glucose and the inability of cells to compensate for insulin resistance in the obese or type 2 diabetic patients have been frequently observed [8]. Obesity increases the risk of type 2 diabetes and other cardiovascular abnormalities, [1, 9] however, physical fitness level has proved be a more accurate predictive indicators of mortality caused by cardiovascular disease compared with the fat mass and body weight [9, 10]. Also, some studies have reported that physical activity affects insulin action and insulin sensitivity independently of changes in weight and body composition [11, 12]. Since, some studies have shown that cardiovascular fitness level affects insulin levels more than compared the percentage body fat, the importance of exercise to improve insulin function in people prone to the syndrome of insulin resistance or type 2 diabetics is increasing [13].

But despite extensive studies on the effect of exercise on blood glucose levels and insulin resistance, the role of physical activity or increased cardiovascular fitness resulting from exercise in insulin function or beta cell function has been given less deliberation and there is no general consensus in this context. Some studies in this context have indicated that regular exercise reduces insulin secretion [14], but some other studies have stated that prolonged exercise increases the glucose-dependent insulin secretion in humans and animal models resistant to insulin, particularly type-2 diabetes [15]. This study is conducted in order to determine the effects of long-term aerobic exercise on insulin

levels, glucose and beta cell function as the prevalence factors of diabetes as well as physiological factors of resting heart rate and maximal oxygen consumption (VO2max) as determinants of cardiovascular fitness on the one hand and to identify association between the pattern of changes of these variables in response to exercise program in none-diabetic obese men on the other hand.

MATERIALS AND METHODS

This semi-experimental study was conducted with the approval of the Ethics Committee of Islamic Azad University. This study was conducted in order to look into the impact of a long-term aerobic exercise training program on aerobic capacity and some indicator markers of type II diabetic in none-diabetic obese males as well as to determine relation between the change of VO2max with the change of fasting glucose and beta cell function in response to aerobic exercise program.

Subjects and Exclusion criteria: The studied subjects were 30 middle-aged males aged 37–48 years, sedentary, obese (BMI 30–36 kg/m2) that divided to experimental and control groups by randomly. A medical history to retrieve information about health status, current medications and activity/diet history were collected of all subjects. Nutritional status of two groups was similar during the time before the study. The participants of two groups were asked to remain their diet during the study. Each participant received written and verbal explanations about the nature of the study before signing an informed consent form. Subjects with a history or clinical evidence of impaired fasting glucose or diabetes, orthopedic abnormalities, recent myocardial infarction, congestive heart failure, active liver or kidney disease, growth hormone deficiency or excess, neuroendocrine tumor, anemia, or who were on medications known to alter insulin sensitivity were excluded. Subjects had neither used any medication 6 weeks prior to the study nor participated in any regular physical exercise. All subjects were non-smokers. In addition, exclusion criteria included inability to exercise and supplementations that alter carbohydrate-fat metabolism.

Anthropometric measurements: Body weight and height were measured with a standard physician's scale and a stadiometer, respectively when subjects were in a fasting state when the participant had thin clothes on and was wearing no shoes. Visceral fat and body fat percentage was determined using body composition monitor (OMRON, Finland). Systolic and diastolic blood pressure was measured using the left arm after the subject had been sitting comfortably for 5 min, using an oscillometric device (Alpikado, Japan). Two measurements were made every 1 minute and the average of two measurements was used for analysis. Body mass index (kg/m^2) was calculated as weight (kg) divided by squared height (m^2).

Blood sampling and exercise program: After anthropometric measurements, all subjects of experimental and control groups were asked to attend Hematology Lab following12 hours of overnight fasting, between the hours of 8 to 9 am for blood sampling. The subjects were advised to avoid any physical activity or exercise 48 hours before the blood sampling. This blood sampling was performed in order to measuring fasting glucose and insulin. Aerobic capacity (VO2max) was calculated by a YMCA standard protocol on cycle ergometer [16] (pre-test). In next stage, the experimental group subjects participated in aerobic training program lasted 3 months (3 days/wk) 60 to 80 percent of maximum heart rate. Each session started by 15 min of flexibility exercises, 30-40 min of aerobic exercise and 5–10 min of cool down activity. Aerobic exercises in each session included walking on a treadmill and stationary cycling. Initially, subjects exercised at low intensity and the intensity of exercise was gradually increased to 80% of peak heart rate in next sessions. The intensity of the activity of any person was controlled using the Polar heart rate tester (made in the US). In this 12-week period, participants in the control group were barred from participating in any exercise training. Finally, all measurements consist of fasting blood sampling, anthropometric measurements and VO2max, 48 h after last exercise session (post-test). Glucose was determined by the oxidase method (Pars Azmoon kit, Tehran). Serum insulin was determined by ELISA method. Beta cell function was assessed using the homeostasis model assessment by formula derived from fasting insulin and glucose levels [17].

Statistical analysis: Statistical analysis was performed with the SPSS software version 15.0 using an independent paired t-test. Pearson correlation method used to determine the relationship between the changes in VO2max with the change of fasting glucose and beta cell function. A p-value < 0.05 was considered to be statistically significant. All values are represented as mean ± SD.

RESULTS

Baseline and post training VO2max, anthropometrical indexes and clinical characteristics of two groups are shown in Table 1. All values are represented as mean ± SD. At baseline, there was a significant correlation between VO2max and body mass index or fasting glucose ($P < 0.05$). The data of independent T-test showed that baseline characteristics such as VO2max, body weight, BMI, insulin, glucose and beta cell function did not differ between experimental and control group (Table 1). Aerobic capacity (VO2max) and beta cell function were significantly increased in experimental group when compared with pre-exercise condition ($P < 0.05$). All anthropometrical markers were significant decrease in response to exercise program in experimental group ($P < 0.05$). Additionally, Exercise training resulted in significant decrease in fasting glucose in experimental group ($P = 0.009$). In experimental group, the change in aerobic capacity in response to exercise program was inversely correlated with the change in fasting glucose ($r = 0.56$, $P = 0.011$). Also, the change pattern in aerobic capacity was positive associated with the change pattern in beta cell function in response to aerobic exercise program ($r = 0.61$, $P = 0.021$).

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

Variable	Experimental group		Control group	
	Pre-test	Post-test	Pre-test	Post-test
Age (year)	44 ± 5	44 ± 5	43 ± 6	43 ± 6
Height (cm)	175 ± 7	175 ± 7	174 ± 6	174 ± 6
Weight (kg)	105 ± 11	98 ± 11	103 ± 13	103 ± 9
Rest heart rate (bpm)	81 ± 12	69 ± 9	79 ± 11	81 ± 14
VO2max (ml.kg.lit)	27.11 ± 5.12	32.06 ± 6.13	26.14 ± 5.12	27.21 ± 6.15
Abdominal circumference (cm)	107 ± 11	101 ± 14	106 ± 11	106 ± 24
Body mass index (m/kg^2)	34.28 ± 3.11	32 ± 3.14	34.02 ± 4.21	34.02 ± 4.12
Body fat (%)	34.11 ± 5.16	29 ± 3.12	33.5 ± 5.13	33.6 ± 6.11
Fasting glucose (mg/dl)	112 ± 14	89 ± 14	110 ± 13	112 ± 21
Insulin (μIU/ml)	8.11 ± 3.23	8.14 ± 3.21	9.01 ± 3.32	8.86 ± 2.11
Beta cell function	59.6 ± 13	113 ± 21	60 ± 15	65 ± 13

DISCUSSION

Failure to control obesity and blood glucose levels leads to certain chronic diseases such as dyslipidaemia, atherosclerosis and type-2 diabetes all of which represent the major constituents of metabolic syndrome [18]. By reducing visceral fat levels and decreasing body weight and fat mass, regular exercise improves insulin sensitivity and blood glucose levels [19]. Although the effect of exercise on insulin secretion is not permanent, exercise plays an important role in glucose homeostasis [20, 21]. The findings of this study showed that a three-month exercise program leads to increased aerobic capacity in obese men and the increase in aerobic capacity was associated with lower fasting glucose concentrations. It is hypothesized that improvement in cardiovascular fitness results in enhanced insulin function while reducing body fat mass [22]. Also, the statistical tests showed that the subsequent to a three-month training program, there would be a significant negative correlation between aerobic capacity and blood glucose concentration even after controlling the weight and body mass index as intermediary factors; in other words, increased aerobic capacity is associated with a lower fasting glucose concentration. In confirmation of these findings, some studies have reported that physical activity improves insulin function or blood glucose levels independently of changes in weight and body composition [11, 12]. The findings of a recent study also show that even in the absence of weight loss, aerobic exercise training would result in better control of glycemia and aerobic capacity plus lowered levels of blood fats [23]. It seems that exercise improves glucose homeostasis through increased glucose uptake in skeletal muscles and adipose tissue [24, 25, 26]. In addition, recent studies have reported that exercise training magnifies the symptoms of hepatic insulin by reducing the release of hepatic glucose under hyperinsulinemia conditions [27, 28].

Increasing age is associated with reduced beta cell mass [29]. On the other hand, enhanced beta cell function is associated with increased sensitivity of body cells to insulin as well as reduced

hyperglycemia [15, 30]. Although, very few studies have addressed to the response of beta cell function to exercise the present study showed that a three-month aerobic training would lead to enhanced beta-cell function. In this context, the findings of some recent studies also indicate enhancement of beta cell function in response to long-term exercise which would reduce blood glucose levels [15, 30]. In fact, both diet and exercise can affect the function of insulin while each one has its own independent mechanism of function. A high-fat diet to overcome insulin resistance increases beta cell mass by hypertrophy process whereas exercise increases beta cell through the process of hyperplasia. In fact exercise-induced hyperplasia occurs as a result of increased beta cell proliferation and decreased cell destruction (apoptosis) [19]. Also, the statistical findings showed that once the exercise program had been completed even after controlling weight and body mass index, there would be a significant relationship between aerobic capacity and the function of beta cells meaning that augmented aerobic capacity enhances the levels of beta cell function. In addition, statistical results showed that the pattern of resting heart rate decrease caused by physical activity has a significant correlation with blood glucose levels and enhanced beta cell function in obese subjects.

Although the effective mechanisms in reducing beta cell function in the obese or in diabetic patients as well as how exercise improves beta cell function cannot be described simply relying on such findings. But it is likely that exercise or improved aerobic capacity caused by exercise indirectly affect blood glucose or beta-cell function by balancing certain peptide hormones such as ghrelin, leptin or adiponectin, [31, 32]. For example, previous observations suggest that increased expression of adiponectin anti-diabetic hormone and GLUT4 reduce blood glucose levels [33]. This means that systemic increase of this peptide hormone as a result of exercise increases the ability of insulin to create maximal stimulation of glucose uptake by the expression of glucose transporter gene (GLUT4) and increases further beckoning of GLUT4 to plasma membrane [34]. In this regard, the findings of a recent study showed that 7 months of aerobic exercise would lead to concurrent increase of aerobic capacity, adiponectin and beta cell function together with reduced fasting glucose concentration [31].

CONCLUSION

In summary, our findings demonstrate that the increase in aerobic capacity in obese subjects after a three-month training program would bring about reduced blood glucose levels and enhanced beta cell function and that the pattern of changes in aerobic capacity has a significant correlation with the pattern of blood glucose decrease or increase in beta cell function. These findings highlight, to some extent, the role of cardiovascular fitness level in regulating marker indictors of type 2 diabetes in obese people. Thus, the aerobic capacity or physical fitness can be noted as predictive factors of obesity related chronic diseases. Understanding the mechanisms responsible for these correlations and hormonal factors or mediators of this relationship is of significant importance and requires further studies in this field.

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Conflict of interest

The authors declare that they have no Conflict of Interests