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The effect of aerobic training program on some indicator markers of diabetes in smokers

Mirakhori Zahra, Mirakhori Fatemeh and Zand Alireza
Department of Physical Education and Sport Science,
Shahr- e -Qods Branch, Islamic Azad University, Tehran, IRAN

(Corresponding author: Mirakhori Zahra)
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ABSTRACT: Few studies have prospectively addressed the effects of exercise on inflammation profile and other abnormalities associated with diabetes in smokers. This study was also aimed to assess the effect of aerobic training program on fasting glucose and insulin resistance in smokers. For this purpose, fasting levels of insulin and glucose were measured before and after three months aerobic training in adult males with cigarette smoking (n=12) and control subjects matched for age (42 ± 5 year) and weight (92 ± 6 kg). The insulin resistance index was assessed by homoeostasis model assessment (HOMA-IR) by fasting glucose and insulin. Paired t test was used to determine the mean differences between pre and post-training values. The results were considered statistically significant for p<0.05. There were no statistically significant differences in all variables between exercise and control groups at baseline ($P > 0.05$). Compared to pre-training, fasting glucose concentration decreased significantly (p = 0.024) by aerobic in exercise group but not in control subjects. We also observed a significant decrease in insulin resistance in exercise (p = 0.008) group but this clinical variables was not changed in control subjects. From these data, we can say aerobic training for long time can be improve abnormalities associated with type II diabetes in smoker.

Keywords: Smoking, Insulin resistance, Aerobic training

INTRODUCTION

It is certain that the main and primary cause of type 2 diabetes is insulin resistance, and beta-cell dysfunction has the next degree of importance. However, some Asian studies reported that the beta-cell function is the primary factor in the pathogenesis of type 2 diabetes [1]. Apart from the main factors or the role of metabolic mediators in the incidence of diabetes, recent studies support the role of some external interventions, such as diet, physical inactivity, and tobacco use, especially in those who are susceptible to this disease. Despite the extensive evidence about the role of cigarette smoking in cardiovascular disease, the molecular mechanisms of its association with the severity and the incidence of type 2 diabetes is not well known. However, in a relatively close study, it was found that the glycosylated hemoglobin levels were significantly higher in smokers compared to non-smokers [2].

Increased blood glucose levels in smokers compared to non-smokers was reported by some studies [3]. The literature supports the increased insulin resistance in smokers compared to non-smokers [4, 5].

The importance of smoking in formation of oxidative stress and impaired beta-cell function has also been reported by some studies [6]. Clinical observations revealed that even those who are exposed to cigarette smoke may also become type 2 diabetics [7].

The literature reveals that among the external interventions, training increases insulin sensitivity in insulin-resistant obese patients or those with a family history of type 2 diabetes as well as the type 2 diabetics [8]. Some studies have reported that short-term exercise increased insulin sensitivity in healthy subjects [9]. However, in a study, training led to a significant increase in VO2max and a significant reduction of the non-esterified free fatty acids in non-diabetics [10]. No change was observed in diabetics. Despite these observations, there are limited studies about the effects of long-term training programs on determinant indicators of type 2 diabetes in smokers. Hence, the present study aimed at determining the effect of a 3-month aerobic training program on fasting glucose levels and insulin resistance in a group of male non-athlete smokers.

MATERIAL AND METHODS

A. Human subjects

This study included twenty four sedentary adult men with cigarette smoking aged 42 ± 5 year and body weight 92 ± 6 kg that selected by accessible samples then divided into exercise (n=12) and control group (n=12).

Exercise group was three months aerobic training for 3 times weekly. Inclusion criteria for the study groups were: healthy and a history of smoking at least for 3 years. Participants were non-athletes, non-smokers and non-alcoholics. Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months. We excluded people who had any self reported physician diagnosed chronic disease (arthritis, stroke, hypertension, cancer, heart attack and chronic cough). Furthermore patients with diabetic were also excluded from the study. After introduction and awareness of the subjects of the objectives of the study and once they had completed consent forms, the process of test implementation began.

B. Anthropometry

Before and after the aerobic training, anthropometrical markers were measured in the morning following a 12-h fast. 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. Weight was measured to the nearest 100 g using digital scales. Body mass index (BMI) was calculated by dividing body mass (kg) by height in meters squared (m2). Blood pressure was measured using the left arm after the subject had been sitting comfortably for 5 min, using an oscillometric device (Alpikado, japens).

C. Biochemistry and training protocol

Fasting blood glucose (FBG) and insulin concentration were measured before and 48 hours after lasted exercise session in exercise or control groups. Blood samples were collected after an overnight fast between 8:00 and 9:00 a.m. Serum glucose was determined by enzymatic (GOD-PAP, glucose oxidase-amino antipyrine) colorimetric method (Pars Azmoun, Tehran, Iran), the Intra-assay and interassay coefficient of variation was 1.74% and 1.19% and sensitivity of the method were 5 mg/dL. Serum insulin was determined by ELISA method (Demedite, German). The Intra- assay coefficient of variation and sensitivity of the method were 2.6% and 2.88 µg/L, respectively. The HOMA1-IR index was calculated by the formula: HOMA1-IR = fasting plasma insulin (µU/ml) x fasting plasma glucose (mmol/L)/22.5 (11). Aerobic exercise program lasted three months for three times per week at 60-80% of maximal heart rate. Each exercise session lasted 45 – 60 min included 5-10 warm up, 30-45 min main exercise (walking or running on treadmill) then cool down. The intensity of the activity of any person was controlled using the Polar heart rate tester (made in the US).

D. Statistical Analysis

All data were tested for normal distribution by the Kolmogorov-Smirnov test. Independent student t test was used for between groups comparison at baseline. Paired t test was used to determine the mean differences between pre and post-training values on all metabolic and anthropometric variables. Statistical significance was accepted at p-value<0.05 or lower.

RESULTS

We previous mentioned that this study was conducted to assess the effect of aerobic training program on insulin resistance and fasting glucose as indicator markers of type II diabetes in smokers.

The baseline of anthropometric and clinical characteristics of the study participants in the exercise and control groups are shown in Table 1. At baseline there were no differences in the age, body weight and other anthropometrical markers between the two groups (Table 1). Fasting insulin, glucose and insulin resistance were also similar between two groups at baseline. Compared to pre-training, the glucose levels decreased ($p = 0.024$) in exercise group but not in the control groups. There was significant decrease in insulin resistance response to aerobic training program in exercise subjects ($p = 0.008$), but this variable remained without change in control subjects. After aerobic training intervention, serum insulin decreased in exercise group ($p = 0.025$) but not in control subjects (Table 2 and 3).

DISCUSSION

In the recent years, the prevalence of type 2 diabetes has increased in young and middle-aged adults. There is also considerable evidence that exercise delays or prevents the incidence of diabetes in susceptible individuals [12]. A significant reduction in the fasting glucose levels in male smokers was found in the present study. The reduction in fasting glucose concentrations in the studied smokers was observed while they had had a sedentary life style before the training program. Therefore, it can be concluded that a 3-month aerobic exercise is associated with a significant reduction in the blood glucose levels in smokers because the fasting glucose levels in the control group, who continued their sedentary life style, did not change significantly during the study. It was found that smokers were more susceptible to diabetes compared to non-smokers. A close association between tobacco consumption and the metabolic syndrome was also observed [13]. In this context, researchers have pointed out that smoking is associated with the reduced response of target cells to blood flow insulin levels [14].

ble 1: Mean and SD of anthropometrical and clinical markers in two groups at baselin

	Exercise group=1, Control group=2	Mean	Std. Deviation	Std. Error Mean
Age (year)	1	41.83	5.750	1.660
	2	42.42	4.274	1.234
Height (cm)	1	173.83	3.738	1.079
	2	174.92	2.610	.753
Weight (kg)	1	92.33	5.678	1.639
	2	92.33	6.184	1.785
Abdominal (cm)	1	102.08	6.895	1.990
	2	102.08	6.735	1.944
Hip (cm)	1	102.17	5.149	1.486
	2	102.33	5.516	1.592
AWHO	1	.9983	.03713	.01072
	2	.9975	.03596	.01038
BMI (kg/m2)	1	30.5883	2.19572	.63385
	2	30.1833	2.02214	.58374
Body Fat (%)	1	29.383	1.8717	.5403
	2	29.458	1.8238	.5265
Insulin (IU/ml)	1	13.49	6.341	1.830
	2	12.14	3.890	1.123
Insulin resistance (HOMA-IR)	1	3.3942	1.30028	.37536
	2	3.1758	.89260	.25767
Glucose (mg/dl)	1	111.17	45.075	13.012
	2	110.08	31.627	9.130

le 2: Pre and post training of anthropometrica and clinicla markers of exercise group

		Mean	Std. Deviation	Std. Error Mean
Pair 1	Weight (pre)	92.33	5.678	1.639
	Weight (post)	88.33	5.581	1.611
Pair 2	Abdominal (pre)	102.08	6.895	1.990
	Abdominal (post)	97.42	5.977	1.725
Pair 3	Hip (pre)	102.17	5.149	1.486
	Hip (post)	97.17	4.745	1.370
Pair 4	BMI (pre)	30.5883	2.19572	.63385
	BMI (post)	29.2567	2.06363	.59572
Pair 5	%fat (pre)	29.383	1.8717	.5403
	%fat (post)	27.750	1.8263	.5272
Pair 6	Insulin (pre)	13.492	6.3406	1.8304
	Insulin (post)	9.217	2.6788	.7733
Pair 7	Insulin resistance (pre)	3.3942	1.30028	.37536
	Insulin resistance (post)	2.2283	.74897	.21621
Pair 8	Glucose (pre)	111.17	45.075	13.012
	Glucose (post)	99.75	32.491	9.379

Table 3: Paired Samples Test of anthropometrical and clinical merkers of between pre and post training of exercis

		Paired Differences				t	df	Sig. (2-tailed)
		Mean	Std. Deviation	95% Confidence Interva of the Difference Lower	Upper			
Pair 1	Weight (pre) - Weight (post)	4.000	2.923	2.143	5.857	4.740	11	.001
Pair 2	Abdominal (pre) - Abdominal (post)	4.667	1.497	3.715	5.618	10.795	11	.000
Pair 3	Hip (pre) - Hip (post)	5.000	1.128	4.283	5.717	15.353	11	.000
Pair 4	BMI (pre) - BMI (post)	1.33167	.99273	.70092	1.96242	4.647	11	.001
Pair 5	%fat (pre) - %fat (post)	1.6333	.4942	1.3193	1.9473	11.449	11	.000
Pair 6	Insulin (pre) - Insulin (post)	4.2750	5.7132	.6450	7.9050	2.592	11	.025
Pair 7	Insulin resistance (pre) - Insu resistance (post)	1.16583	1.25092	.37103	1.96063	3.228	11	.008
Pair 8	Glucose (pre) - Glucose (pos	11.417	15.066	1.844	20.989	2.625	11	.024

The literature suggests that smokers are less able to control their diabetes compared to non-smokers. Some studies have reported that smokers undergo metabolic disorders characterized by the insulin resistance syndrome which increases the postprandial triglycerides, and subsequently increases LDL particles [15]. A study in the American populations reported the higher incidence of hyperinsulinemia in smokers compared to non-smokers [16]. Researchers have also noted the increased levels of cardiovascular risk factors in smokers compared to non-smokers are rooted in the insulin resistance phenomenon [17]. Despite these observations, some other studies have reported no relationship between smoking and diabetes [18].

Exercise has been reported as a factor of insulin sensitivity enhancement and insulin function improvement in the insulin-resistant humans and animal models [19]. Some studies indicated that regular exercise reduces insulin secretion through its stimuli [20]. However, other studies suggested that long-term exercise increases the secretion of glucose-dependent insulin in humans and animal models with type 2 diabetes [21]. Some studies showed that exercise improves glucose homeostasis by increasing glucose absorption in skeletal muscle and adipose tissue [22, 23]. Some studies also reported that exercise reduces the hepatic insulin symptoms through reducing the release of hepatic glucose in hyperinsulinemia [24, 25].

The positive effects of exercise on blood levels in both diabetics and non-diabetics individuals is somehow associated with the insulin function response or the response of target cells to insulin so that the reduced insulin resistance somehow reduces blood glucose levels in diabetic and non-diabetic populations. In the present study, the fasting glucose levels were reduced following the training programs.

The reduced insulin resistance was a main finding of this study. Based on these findings, it can be concluded that in smokers, aerobic exercise program reduces hyperinsulinemia through reducing insulin resistance in target cells. It is also possible that exercise program affect the blood glucose levels though affecting other hormonal components. For example, a significant inverse relationship between adiponectin, as an inflammatory cytokine, and insulin resistance was observed by some researchers [26, 27]. It was also found that the increase in adiponectin leads to the reduced insulin resistance in smokers which is associated with lower baseline levels of blood sugar and is due to reduced release of hepatic glucose through increasing adiponectin levels [28]. The literature also supports the significant increase in adiponectin in response to long-term training programs [29, 30].

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