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Effects of concurrent training on insulin resistance and pancreatic-cells function in obese men

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Abstract

Introduction: The insulin resistance and subsequent pancreatic β cell failure that precedes the development of hyperglycemia is thus usually associated with obesity. Exercise training may improves these impairs, however, this is not well known. The aim of present study was to examine the effect of 8 weeks concurrent training on pancreatic β-cells function and insulin resistance in obese men.

Material & Methods: Twenty three 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. The subjects in the control group were instructed to maintain their normal physical activity throughout the study.

Results: The results indicated that fasting insulin (P=0.006), insulin resistance index (P=0.02) and pancreatic β-cells

function (P=0.04) were decreased in the training group compare to the control group; however, fasting blood sugar has no significant change after the intervention (P=0.3).

Conclusion: In summary, it seems that concurrent training utilized in this study improves insulin resistance and pancreatic β-cells function in obese men.

Keywords: Obesity, Concurrent training, Pancreatic β-cells function, Insulin resistance

1. Introduction

The explosive increase in obesity is one of the most pressing health-related problems anywhere in the world. According to the report from the world health organization (WHO), 1.4 billion adults in the world are overweight. 200 million male and 300 million female adults over 20 years of age are obese (1). However, the greater health concern with regard to obesity is the effects of obesity-related diseases such as diabetes and metabolic syndrome.

Diabetes mellitus is a major public health problem that is determined with impaired carbohydrate metabolism, protein, and fat due to unstable insulin secretion, insulin resistance secretion, or both (2). With an 8.5% global prevalence of diabetes in 2014; various estimates suggest that the number of affected people will be risen from 422 million to 642 million in the world by 2040 (3,4). In Iran, the prevalence of diabetes in adults aged 25–70 years was reported 11.9% (2011) which shows an increase of 35% compared to 2005. It is estimated that in the year 2030 nearly 9.2 million Iranians likely to have diabetes (5).

Insulin resistance indicates a failing physiological response to insulin. Even if insulin is normally secreted, the normal action of removal of glucose from the bloodstream to control blood sugar concentrations declines, and results in high blood sugar levels (6). The standard technique for assessment of insulin sensitivity is the hyperinsulinemic euglycemic clamp; however, it is far too invasive to be practical in most studies. The homeostatic model assessment (HOMA) method has been the widely used in clinical investigations. HOMA uses fasting insulin and

glucose levels to determine insulin resistance, and correlates well with the results of clamping. To avoid complex procedures or widely changing glucose levels, the homeostatic model assessment focuses on basal fasting glucose and insulin levels (7). Also the failing function and the decrease in number of β-cell is the common features of type 2 diabetes patients. The damage in β-cell which deters insulin secretion and inhibits the proper control of blood sugar levels is explained as a crucial feature of the occurrence of diabetes. Though there is a great deal of research using homeostatic model assessment insulin resistance (HOMA-IR) as an index of diabetic risk, research utilizing HOMA β-cell is less common (8).

Several factors may have a bearing on β-cell function, including healthy lifestyle – especially regular physical activity and exercise. Healthy life style provides clues to developing strategies to ameliorate the long-term management of Type 2 diabetes. Regular exercises improve those factors which relate to insulin resistance (9), and deter various complications such as cardiovascular disease (10); however, the data on the effects of exercise training on β-cell function are not well known. Ha et al. (2015), for example, reported that HOMA-IR and β-cell function improved after 12-week of supervised combined exercise (11). However, Omidi and Moghadasi (2017) showed that insulin resistance but not pancreatic β-cells function improved after 8 weeks of aerobic training in female patients with type 2 diabetes (12). The aim of present study was to examine the effect of 8 weeks concurrent training on pancreatic β-cells function and insulin resistance in obese men.

2. Materials and methods

Subjects

Twenty three obese men participated in the present study as the subject. The subjects were sedentary obese men aged 23–58 years, who had not previous been diagnosed with abnormal glucose metabolism and had body mass index percentage greater than 30. Participants were non-smokers and had not participated in regular exercise/diet programs for the preceding 6 months. The exclusion criteria were as follows: Patients with known history of acute or chronic respiratory infections, neuromuscular disease, and cardiopulmonary disease. In addition,

exclusion criteria included inability to exercise and supplementations that alter carbohydrate and fat metabolism. The subjects were given both verbal and written instructions outlining the experimental procedure, and written informed consent was obtained. Characteristics of the subject's measurements included for age, height (cm), weight (kg), BMI, and WHR. Blood variables measurements included for fasting insulin and glucose. All variables were measured 2-day before the pre- and post- of the study. The study was approved by the Islamic Azad University, Marvdasht branch Ethics Committee.

Measurements

Age was self-reported via a questionnaire height, weight, body mass index (BMI), Hip circumstance, waist circumference and waist to hip ratio (WHR) were assessed using standard methods. The BMI (kg/m^2) of the subject was calculated on the basis of their height and weight. The waist to hip ratio (WHR) was calculated by dividing waist by hip circumference (cm). Glucose was determined by the oxidase method. Insulin concentration was measured using ELISA method (Mercodia, Sweden). The intra and inter-assay coefficients of variation for glucose were $<1.3\%$ and a sensitivity of 1 mg/dl. Utilizing the analyzed glucose and insulin concentration level in the blood, HOMA-IR, which is the indicator for insulin resistance, was estimated and HOMA β-cell, the indicator for insulin secretion level, was also assessed. HOMA-IR and HOMA β-cell were estimated as follows (13):

$$\text{HOMA-IR} = [\text{fasting insulin } (\mu\text{IU/ml}) \times \text{fasting glucose (mmol/l)}]/22.5$$

$$\text{HOMA } \beta\text{-cell} = [20 \times \text{fasting insulin } (\mu\text{IU/ml})]/[\text{fasting glucose (mmol/l)} - 3.5]$$

Exercise training

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 5 stations (Chest press, lateral rise hand, barbell curl, leg press and hamstring with machine) 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.

Statistical analysis

Results were expressed as the mean ± SD and distributions of all variables were assessed for normality. Data were analyzed using independent and paired sample t-test. The level of significance in all statistical analyses was set at $P<0.05$. Data analysis was performed using SPSS software for windows (version 17, SPSS, Inc., Chicago, IL).

3. Results

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 decreased ($P<0.05$) after 8 weeks concurrent training compared to the control group.

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

	Control (mean±SD)		Concurrent training (mean±SD)	
	Pre-training	Post-training	Pre-training	Post-training
Body mass (Kg)	106.6 ± 4.7	106.5 ± 7.6	98.08 ± 6.9	94.5 ± 7.4*,
BMI (Kg/m^2)	34.1 ± 2.5	34.1 ± 3.3	31.8 ± 1.7	30.6 ± 1.4*,
WHR	0.95 ± 0.03	0.95 ± 0.04	0.96 ± 0.04	0.94 ± 0.04*,

*:$P<0.05$ for between-group differences.

†:$P<0.05$, pre-training *vs.* post-training values.

Biochemical parameters of the subjects are presented in Table 2. The results indicated that fasting insulin ($P=0.006$), insulin resistance index (HOMA-IR) ($P=0.02$) and pancreatic β-cells function (HOMA-B) ($P=0.04$) were decreased in the training group compare to the control group; however, fasting blood sugar has no significant change after the intervention ($P=0.3$).

Table 2. Changes of the biochemical parameters of the subjects before and after training

	Control (mean ± SD)		Training (mean ± SD)	
	Pre-training	Post-training	Pre-training	Post-training
Fasting glucose(mmol/l)	5.6 ± 0.4	5.6 ± 0.6	5.2 ± 0.7	5.4 ± 0.8
Fasting insulin (IU/ml)	12.7 ± 6.7	14.3 ± 8.4	11.4 ± 8.9	6.8 ± 6.2*,
HOMA-IR	3.2 ± 1.9	3.5 ± 2.0	2.7 ± 2.3	1.7 ± 1.6*,
HOMA-B	119.5 ± 50.1	167.7 ± 185.1	132.5 ± 101.1	71.4 ± 56.6*,

*: P<0.05 for between-group differences.

†: P<0.05, pre-training vs. post-training values.

4. Discussion

Type 2 diabetes comprises the majority of people with diabetes around the world, and is largely the result of excess body weight and physical inactivity. Participation in regular and consistent exercise improves the health of the general population, but also has significant beneficial effects on obesity-related diseases (14). Previous studies indicated that exercise training is a useful therapy for improving insulin resistance (15-17). The results of the current study in line with previous studies demonstrated that fasting insulin and insulin resistance determined by HOMA-IR and pancreatic β-cells function (HOMA-B) improve after 8 weeks concurrent training. In line with the result of present study, Ha et al. (2015) reported that HOMA-IR and β-cell function improved after 12-week of supervised combined exercise (11). The result of reductions in HOMA-IR for this study is also supported by previous research (18,19). However, there is still an issue regarding whether insulin resistance or HOMA β-cell precedes one another in the occurrence of diabetes. In the case where the insulin resistance is argued to be the primary cause, decline in the function of β-cell is the later response to the gradual increase in the insulin secretion due to the insulin resistance. Those who argue that the malfunction of β-cell is the primary cause of the diabetes state that the reduction in the insulin secretion is the necessary course for the normal level of blood sugar increase without adequate control (20).

Exercise increases insulin-mediated GLUT4 translocation to the sarcolemma and subsequent glucose uptake, which may reflect a transient elevation as a consequence of the "last bout" (21). The underlying increase in GLUT4 transcription and expression of GLUT4 mRNA has been shown to persist for 3 to 24 hours after exercise (22,23).

In this way, regular exercise translates into a steady-state increase of GLUT4 protein expression, and subsequent improvement in glucose control over time (22). Similarly, enhanced whole-body insulin sensitivity has been shown to occur in the hours immediately following exercise, and evidence from a limited number of studies using hyperinsulinaemic-euglycaemic clamp and oral glucose tolerance test suggests that this may persist for up to 24 to 72 hours after the last bout (23-25).

There is still the issue of which between insulin resistance and HOMA β-cell precedes the occurrence of diabetes (20). In the case where insulin resistance is argued to be the primary cause, decline in the function of β-cell is the later response to the gradual increase in insulin secretion due to the insulin resistance. However, those who argue that the malfunction of β-cell as the primary cause of diabetes state that the reduction in the insulin secretion is the reason that normal blood sugar levels would increase (26).

Haffnet et al. (1996) noted that the β-cell malfunction is the primary cause of diabetes (27). Through regular exercise, less insulin can carry the same amount of glucose to muscles and the liver. Therefore, β-cells in the pancreas do not have to excessively secrete insulin, resulting in decreased HOMA-IR and HOMA β-cell indexes (28). The mechanisms behind the improved pancreatic β-cell function to increase insulin action could be multifaceted, including perhaps an improved coordinated feedback loop between liver (decreased hepatic gluconeogenesis), muscle (attenuated insulin resistance) and pancreas (slowly wakening of β islets to secrete insulin). However, given that type 2 diabetes is a disease characterized by perturbations in several organs, anti-inflammatory cytokines secreted by both adipocytes (e.g. adiponectin) and myocytes (e.g. IL-6) could be involved in the improvement of pancreatic β-cell function (29). Although we had not measured the inflammatory and anti-inflammatory cytokines, not significant change in β-cell function might due to unchanged on these cytokines in the present study.

5. Conclusion

We conclude that 8 weeks of concurrent training affected, insulin, HOMA-IR and HOMA β-cell in obese men.

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Conflict of interests: authors declare that there is no conflict of interest.

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