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Acute and recovery responses of glucose and insulin resistance to running test in obese individuals

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Abstract

Accumulating evidence indicates that obesity and overweight are associated with insulin resistance; however, the precise mechanisms of any association between them have not yet been established. This study is designed to evaluate acute and recovery response of insulin resistance to an exercise test in adult obese men. For this purpose, twenty sedentary healthy obese men aged 37 – 44 years, 30 – 35 kg/m2 of BMI were enrolled for participation in this study. Venous blood samples were collected before, 0, 1 and 24 hours after single bout running test for 45 min in order to measuring glucose and insulin. Insulin and glucose levels were used for the homeostasis model assessment of insulin resistance. Repeated measures ANOVA method was used to analysis. Glucose concentration decreased immediately and 1 hour after exercise test and return to baseline levels after 24 hours. Serum insulin and insulin resistance decreased immediately after exercise, but return to baseline levels after 1 hour. Based on these data, we can say 45 min running for one session is associated with acute decreased in glucose and insulin resistance not recovery response in obese men.

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Introduction

Weight gain and obesity are major risk factors for conditions and chronic diseases such as type II diabetes, cardiovascular diseases, metabolic syndrome and insulin resistance (Steven *et al.,* 2007). Insulin resistance is known to decreased sensitivity to these metabolic actions of insulin (Kim *et al.,* 2008). The key actions of insulin maintain circulating glucose concentration by promoting glucose uptake in skeletal muscle and the tissues and inhibiting glucose production in the liver. Obesity is associated with increased blood glucose and insulin resistance, characterized by an impaired ability of insulin to inhibit glucose output from the liver and to promote glucose uptake in fat and muscle (Saltiel *et al.,* 2001; Nandi *et al.,* 2004). While obesity-linked insulin resistance was reported by many recent studies, the physiopathological mechanisms underlying these associations remain poorly understood. Today, several drugs have been introduced to reduce insulin resistance. On the other hand, accumulating evidence indicates that exercise training is a nonpharmologicl intervention for improve insulin resistance in obese or diabetic individuals (Perrini *et al.,* 2004; Dela *et al.,* 1990; Dela *et al.,* 2004).

In this are, numerous studies support beneficial effect of long-term exercise training on blood glucose of insulin resistance, but there are limited information about effects of short-time or one session exercise on these variables. This study is designed to investigation acute and recovery response of insulin resistance and glucose concentration to a session running test sedentary middle-aged obese men.

Materials and methods

In this study, we evaluated acute and recovery responses of insulin resistance and its determinative to one session running test in obese men. The methods and procedures used in this study were approved by Ethics Committee of Islamic Azad University, Parand branch, Iran. Subjects were sedentary adult men aged 37 - 44 years, obese (BMI 30–35 kg/m2, n=20) that participated in this study by accessible sampling. Written consent was obtained from each subject after the experimental procedures and possible risks and benefits were clearly explained. All of subjects had not participated in regular exercise for the preceding 6 months, nor did all subjects have stable body weight. All subjects were non-smokers. Participants had no evidence of coronary artery disease; tobacco use; or use of systemic steroids, ß-blockers, or thiazides. Subjects with a history or clinical evidence of impaired fasting glucose or diabetes, 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. In addition, exclusion criteria included supplementations that alter carbohydrate metabolism.

All of anthropometrical measurements were conducted by the same researcher when the participant had thin clothes on and was wearing no shoes. Weight and height were measured in the morning, in fasting condition, standing, wearing light clothing and no shoes. Body mass index (BMI) was calculated by dividing body mass (kg) by height in meters squared (m2). Abdominal-to-hip ratio was calculated as abdominal circumference divided by hip circumference as measured to the nearest 0.5 cm with a standard measuring tape. Percentage body fat was measured using body composition monitor (OMRON, Finland).

After anthropometrical measurements, all participants asked to attend Hematology Lab between the hours of 8 to 9 am after overnight fast. Blood samples were collected prior to exercise, at the end of exercise, and at 1 and 24 hours following exercise. Blood samples were analyzed for serum insulin, glucose and insulin resistance. Subjects were asked to avoid doing any heavy physical activity for 48 hours before blood sampling. In exercise test, subjects underwent a running test for 45 min at intensity 75(%) of maximal heart rate after a 5 min warm-up at a speed. Blood samples were dispensed into EDTA-

coated tubes and centrifuged for 10 minutes in order to separate serum. The homoeostasis model assessment (HOMA) for estimating insulin resistance was calculated as serum glucose (mmol/L) ×serum insulin (mU/L)/22·5 (Matthews *et al.,* 1985).

Statistical analysis

Statistical analysis was performed with the SPSS software version 15.0. Experimental data are presented as means ± Sd and were analyzed by one-way analysis of variance with repeated measures over time. A p value less than 0.05 was considered statistically significant.

Results

Table 1 shows the descriptive anthropometric features of the study subjects. Data were expressed as individual values or the mean ± SD.

Table 1. Mean and standard deviation (SD) of anthropometric characteristics of studied subjects.

Variables	Mean	SD
Age (year)	38.2	2.11
Height (cm)	172	4.8
Weight (kg)	93.6	7.90
Abdominal circumference (cm)	104.4	4.87
Hip circumference (cm)	105.9	4.65
WHO	0.99	0.02
Body mass index (kg/m²)	31.64	1.58
Body Fat (%)	32.47	2.01
Visceral fat	13.20	2.18

Table 2. Acute and recovery responses of biochemical variables to exercise test.

Variables	Baseline	Acute response	Recovery response (1 hour)	Recovery response (24 hour)
Glucose (mg/dl)	92 ± 4	87 ± 3	89 ± 4	91 ± 4
Insulin (µIU/ml)	20.21 ± 1.87	13.38 ± 0.40	21.33 ± 2.78	19.39 ± 0.92
Insulin resistance (HOMA-IR)	4.69 ± 0.53	2.87 ± 0.14	4.87 ± 0.69	4.44 ± 0.36

Baseline and post exercise levels of insulin, glucose and insulin resistance are shown in Table 1. The statistical data showed that exercise test led to significantly decrease in glucose immediately and 1 hour after test but its value returned to baseline after 24 hour recovery (Fig 1). Serum insulin was also decreased immediately after exercise and returned to baseline after 1 or 24 hours recovery. Like the insulin, insulin resistance was also decreased significantly immediately after exercise but we did not change after 1 or 24 hours when compared to baseline (Fig 2).

Discussion

The findings of the study reveal that a relatively long-term exercise with moderate intensity decreases levels of glucose, insulin and insulin resistance in obese men studied. And blood glucose levels remained lower than baseline levels for hours following the implementation of the test. This difference was significant only after an hour from the test and then after 24 hours of recovery, although lower than baseline levels, it was not statistically significant. Exercise as an increasing factor of insulin sensitivity improves insulin function in people and

animals and leads to insulin resistance (Perrini *et al.,* 2004). In this context, some studies have indicated that physical activity reduces insulin secretion due to the impact on insulin stimulus (Dela *et al.,* 1990; Dela *et al.,* 2004). It appears that increased insulin performance despite its decreased levels immediately after exercise as well as insulin resistance reducing factor may play a significant role in this phenomenon, although the role of other factors in the glucose levels such as peptidic mediators or cytokine should not be ignored; because a close association is reported between inflammatory and non-inflammatory cytokine levels, and blood glucose before and after exercise of short or long duration (Tang *et al.,* 2005; Rubin *et al.,* 2008; Sheu *et al.,* 2008).

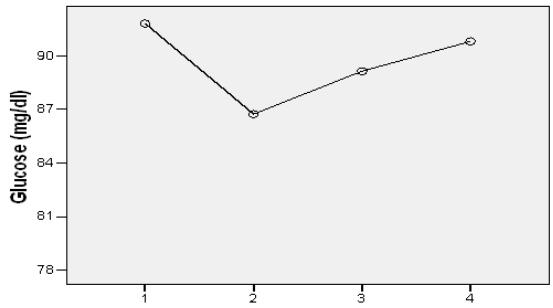

Fig. 1. Acute and recovery response of glucose concentration to exercise test.

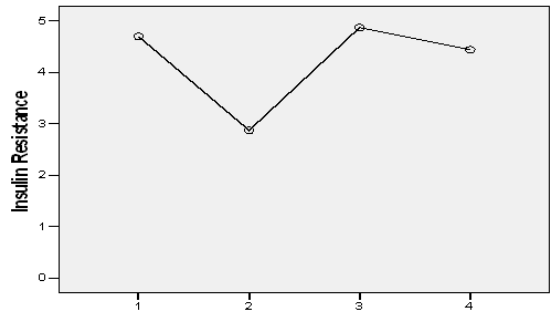

Fig. 1. Acute and recovery response of insulin resistance to exercise test.

It is known that among environmental interferences high fat diet leads to increased insulin resistance, whereas insulin resistance is associated with decreased physical activity. In addition to impacting on insulin resistance, regular diet or exercise program affect the mass and the performance of beta cells (Park *et al.,* 2007). In fact, regular physical activity improves glucose homeostasis not only by reducing insulin resistance but also by increasing beta-cells mass and function, however, the acute and delayed response of blood glucose levels to short-term or single-session exercise have been less frequently studied. Some recent studies have shown that exercise enhances glucose homeostasis by boosting glucose uptake in skeletal muscles and adipose tissue (Berggren *et al.,* 2005; Choi *et al.,* 2005; Cockram *et al.,* 2000). In addition, recent studies have reported that regular exercise decreases hepatic insulin symptoms by reducing glucose release liver in hyperinsuline conditions (Heled *et al.,* 2004; Perseghin *et al.,* 2007).

Long-term studies have been done before as to the effect of exercise on glucose and insulin levels or insulin resistance, but few studies have focused on the acute response as well as short-term delayed responses following a single session of exercise. Citing the findings of this study it can be concluded that a moderate-intensity yet relatively prolonged single session of exercise leads to an immediate and temporary decrease in blood glucose which lingers on for hours after the test however over time, its level decreases gradually to baseline levels. Confirming the findings of the present study, some previous studies have reported that even a single session of endurance exercise with moderate to high intensity also helps insulin sensitivity and leads to glucose homeostasis (Bell *et al.,* 2007; Yassine *et al.,* 2009). Hence, performing exercise several times a week is not ineffective in reducing glucose in obese or diabetic individuals.

References

Bell LM, Watts K, Siafarikas A. 2007. Exercise Alone Reduces Insulin Resistance in Obese Children Independently of Changes in Body Composition. Journal of Clinical Endocrinology & Metabolism **92(11),** 4230–5.
http://dx.doi.org/10.1210/jc.2007-0779

Berggren JR, Hulver MW, Houmard JA. 2005. Fat as an endocrine organ: influence of exercise. Journal of Applied Physiology **99,** 757–764.
http://dx.doi.org/10.1152/japplphysiol.00134.2005

Choi SB, Jang JS, Park S. 2005. Estrogen and exercise may enhance beta-cell function and mass via IRS2 induction in ovariectomized diabetic rats. Endocrinology **146,** 4786–4794.
http://dx.doi.org/10.1210/en.2004-1653

Cockram CS. 2000. The epidemiology of diabetes mellitus in the Asia-Pacific region. Hong Kong Medical Journal **6,** 43–52.

Dela F, Mikines KJ, Tronier B, Galbo H. 1990. Diminished arginine-stimulated insulin secretion in trained men. Journal of Applied Physiology **69,** 261–267.

Dela F, von Linstow ME, Mikines KJ, Galbo H. 2004. Physical training may enhance beta-cell function in type 2 diabetes. American Journal of Physiology - Endocrinology and Metabolism **287,** 1024–1031.
http://dx.doi.org/10.1152/ajpendo.00056.2004

Heled Y, Shapiro Y, Shani Y, Moran DS, Langzam L, Barash V, Sampson SR, Meyerovitch J. 2004. Physical exercise enhances hepatic insulin signaling and inhibits phosphoenolpyruvate carboxykinase activity in diabetes-prone Psammomys obesus. Metabolism **53,** 836–841.
http://dx.doi.org/10.1016/j.metabol.2004.02.001

Kim J, Wei Y, James R. 2008. Role of Mitochondrial Dysfunction in Insulin Resistance. American Heart Association **102,** 401-414.

Matthews DR, Hosker JP, Rudenski AS, Naylor BA, Treacher DF, Turner RC. 1985. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. Diabetologia **28(7),** 412-419.
http://dx.doi.org/10.1007/BF00280883

Nandi A, Kitamura Y, Kahn CR, Accili D. 2004. Mouse models of insulin resistance. Physiological Reviews **84(2),** 623-47.
http://dx.doi.org/10.1152/physrev.00032.2003

Park S, Hong SM, Lee JE, Sung SR. 2007. Exercise improves glucose homeostasis that has been impaired by a high-fat diet by potentiating pancreatic B- cell function and mass through IRS2 in diabetic rats. Journal of Applied Physiology **103(5),** 1764-71.
http://dx.doi.org/10.1152/japplphysiol.00434.2007

Perrini S, Henriksson J, Zierath JR, Widegren U. 2004. Exercise-induced protein Kinase C isoform-specific activation in human skeletal muscle. Diabetes **53,** 21–24.
http://dx.doi.org/10.2337/diabetes.53.1.21

Perseghin G, Lattuada G, De Cobelli F, Ragogna F, Ntali G, Esposito A, Belloni E, Canu T, Terruzzi I, Scifo P, Del Maschio A, Luzi L. 2007. Habitual physical activity is associated with intrahepatic fat content in humans. Diabetes Care **30,** 683–688.
http://dx.doi.org/10.2337/dc06-2032

Rubin DA, McMurray RG, Harrell JS, Hackney AC, Thorpe DE, Haqq AM. 2008. The association between insulin resistance and cytokines in adolescents: the role of weight status and exercise. Metabolism **57(5),** 683-90.
http://dx.doi.org/10.1016/j.metabol.2008.01.005

Saltiel AR, Kahn CR. 2001. Insulin signalling and the regulation of glucose and lipidmetabolism. Nature **414,** 799–806.
http://dx.doi.org/10.1038/414799a

Sheu WH, Chang TM, Lee WJ, Ou HC, Wu CM, Tseng LN ET AL. 2008. Effect of weight loss on proinflammatory state of mononuclear cells in obese women. Obesity (Silver Spring) **16(5),** 1033-8.
http://dx.doi.org/10.1038/oby.2008.37

Steven E, Laura H, Afia N. 2007. Obesity, Inflammation, and Insulin Resistance. Gastroenterology **132,** 2169–2180. http://dx.doi.org/10.1053/j.gastro.2007.03.059

Tang Z, Yuan L, Gu C, Liu Y, Zhu L. 2005. Effect of exercise on the expression of adiponectin mRNA and GLUT4 mRNA in type 2 diabetic rats. Journal of Huazhong University of Science and Technology **25(2),** 191-3, 201.

Yassine HN, Marchetti CM, Krishnan RK. 2009. Effects of exercise and caloric restriction on insulin resistance and cardiometabolic risk factors in older obese adults – Arandomized clinical trial. Journal of Genontolog **64,** 90 –5.