

**Research Article**

## **ACUTE RESPONSE OF GLUCOSE TO EXERCISE DOES NOT ATTRIBUTED TO INSULIN RESISTANCE**

**Hajirasouli Masoud\* and Hajirasouli Maral**

*Department of Physical Education and Sport Sciences, Islamshahr Branch, Islamic Azad University,  
Islamshahr, Iran*

*\*Author for Correspondence*

### **ABSTRACT**

A growing body of literature suggests an important contribution of abdominal obesity and inflammation to the development of insulin resistance and type II diabetes. The objective of this study was to compare fasting glucose, serum insulin and insulin resistance between non-trained obese and non-obese population matched age (35-45 year) and to determine acute response of these variables to a single bout exercise in obese subjects. For this purpose, fasting blood samples were collected after an overnight fast to measuring and compare mentioned variables between to groups. In addition, all dependent variables were measured immediately after a single bout cycling in obese subjects. Fasting glucose and serum insulin and insulin resistance were higher in obese men than in normal weight men ( $p = 0.05$ ). Exercise test resulted in significant decrease in glucose ( $p = 0.010$ ) and significant increase in serum insulin ( $p = 0.000$ ). Insulin resistance did not change by exercise in obese subjects ( $p = 0.44$ ). In conclusion, acute response of glucose to exercise can not attribute to insulin resistance. Further study will be needed to clarify the mechanism of the modulation of the glucose during acute exercise.

**Keywords:** *Glucose, Acute Exercise, Insulin Resistance*

### **INTRODUCTION**

Growing trend of mechanic life especially in the last two decades and reduced physical activity throughout the day underlie many chronic diseases. However, the presence of chronic diseases is not directly originated from this common model of life, rather it occurs due to some environmental and genetic factors such as obesity or increased body fat. Obesity is introduced as one of the first consequences of physical inactivity and high-calorie and high-fat nutritional patterns, and today is one of the most serious public health problems in developed societies, as well as developing countries. Obesity is associated with increased prevalence of chronic diseases such as cardiovascular disease, metabolic syndrome, diabetes, and diseases related to insulin resistance; although the basic mechanisms involved in direct association between obesity and these diseases are not yet fully known. World Health Organization recalls the rapid increase in the prevalence of obesity as an epidemic and introduces obesity and its complications as one of the major health problems in the world (Diabetes group, 2003).

The prevalence of obesity and its related diseases, particularly cardiovascular disease, is considered as one of the most important factors of mortality in developed countries; although its prevalence is also increasing rapidly in developing countries resulting in increased related diseases (Garrow, 1999). Obesity is mentioned as a risk factor for type 2 diabetes by researchers. According to statistics in recent years, more than 30-40 percent of people with type 2 diabetes are obese (Lazar, 2005). In fact, although obese diabetics secrete more insulin than people with normal weight, they secrete less insulin compared with same weight non-diabetic obese individuals (Sigal *et al.*, 2006). Scientific resources have supported the close relationship of diabetes type 2 with certain metabolic and chronic diseases resulting from obesity such as hyperglycemia, hypertension, atherosclerosis, insulin resistance, and coronary artery disease (Edward *et al.*, 2005). Besides heredity and inactivity, obesity and rise in body fat levels can be mentioned as important factors in the prevalence of insulin resistance phenomenon, because the effects of exercise- or diet-induced weight loss in reducing insulin resistance are frequently observed in obese people (Bjornholt *et al.*, 2000; Bennett, 2004). This study aimed to compare some key indicators of diabetes among obese and normal weight men and to assess the response of these variables to a short-term moderate hand-biking in obese men.

## Research Article

### MATERIALS AND METHODS

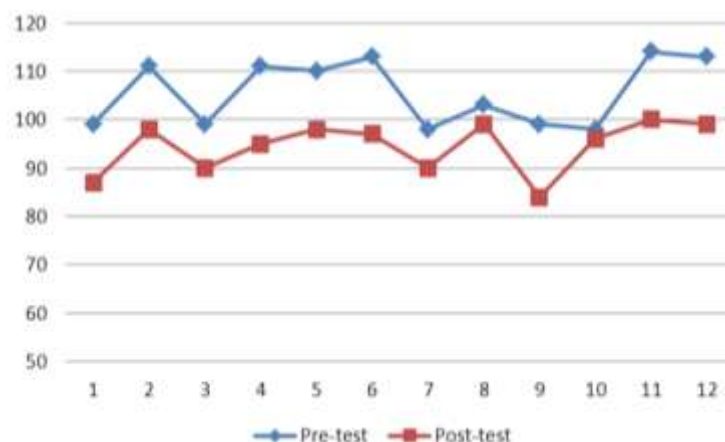
**Study population and design:** First aim of this study was to compare fasting glucose, insulin and insulin resistance between obese and normal weight groups. To achieve this aim, thirteen healthy untrained obese men and thirteen healthy untrained normal weight men were participated in this study. Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months. Participants were non-athletes, non-smokers and non-alcoholics. Subjects included individuals with no cardiovascular diseases, gastrointestinal diseases, kidney and liver disorders or diabetes. Exclusion criteria included medications that alter carbohydrate metabolism and inability to exercise. Written consent was obtained from each subject after the experimental procedures and possible risks and benefits were clearly explained.

**Anthropometric Measurements:** All anthropometrical markers were measured in two group subjects. The weight and height of the participants were measured by the same person when the participant had thin clothes on and was wearing no shoes by using the standard hospital scales. Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. Visceral fat and body fat percentage was determined using body composition monitor (OMRON, Finland). BMI was calculated as weight (in kilograms) divided by the square of height (in meters).

**Blood Biochemistry Examination:** All subjects of two groups were asked to attend hematology Lab following 12 hours of overnight fasting, between the hours of 8 to 9 am for blood sampling. Fasting blood samples were analyzed to determine glucose, insulin and insulin resistance. Obese subjects were also completed a stepwise test according to YMCA protocol included leg cycling. Blood samples were repeated at 0 min after exercise test. All blood samples were collected in order to measuring glucose, insulin and insulin resistance. Glucose was determined by the oxidase method (Pars Azmoon kit, Tehran). Serum insulin was determined by ELISA method. Insulin resistance (HOMA-IR) = [fasting insulin ( $\mu$ /ml)  $\times$  fasting glucose (mmol/l)] / 22.5

**Statistical Analysis:** Data are presented as mean  $\pm$  S.D. Analyses were performed using SPSS (version 15.00). The Kolmogorov-Smirnov test was applied to determine the variables with normal distribution. Multivariate analysis of variance was used to examine baseline differences between groups. Pre-exercise and post-exercise values were determined with the Student t test for obese group. A probability level of  $p < 0.05$  was used to indicate statistical significance.

### RESULTS



**Figure 1: The changes pattern of glucose by exercise test. Glucose concentration decreased by exercise test in studied obese subjects**

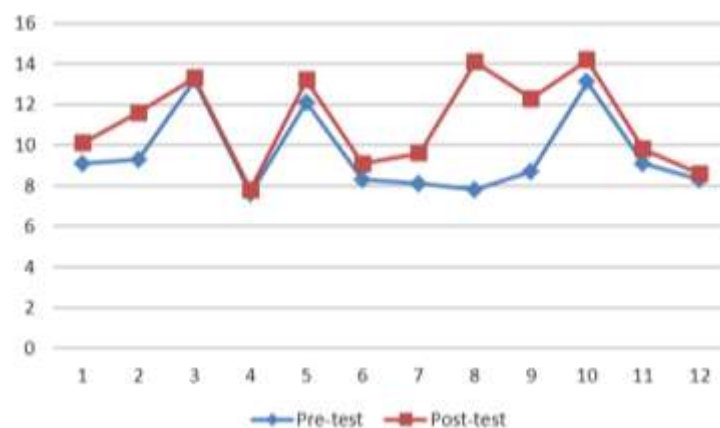
Anthropometric and metabolic characteristics of the study participants in the normal weight and obese groups are shown in Table 1. Fasting glucose concentration, serum insulin and insulin resistance were significant higher in obese in comparison to normal weight subjects ( $p < 0.001$ ). Glucose concentration

## Research Article

was significantly ( $p = 0.010$ ) decreased (from a mean  $(106 \pm 7$  to  $94 \pm 5$ , mg/dl, Figure 1) and serum insulin significantly ( $p = 0.000$ ) increased (from  $9.6 \pm 2.04$  to  $11.1 \pm 2.25$ ,  $\mu\text{IU/ml}$ , Figure 2) by exercise test in obese subjects. Significant differences were not found in insulin resistance between pre and post test in obese subjects (pre;  $2.48 \pm 0.49$ , post;  $2.60 \pm 0.55$ ,  $p = 0.44$ ).

**Table 1: Anthropometric and biochemical characteristics of the study participants**

|                          | Obese=1, Normal=2 | Mean    | Std. Deviation |
|--------------------------|-------------------|---------|----------------|
| Age (year)               | 1                 | 38.08   | 2.429          |
|                          | 2                 | 37.08   | 2.610          |
| Height (cm)              | 1                 | 176.58  | 3.232          |
|                          | 2                 | 174.17  | 2.657          |
| Weight (kg)              | 1                 | 101.33  | 11.276         |
|                          | 2                 | 71.17   | 4.407          |
| Abdominal (cm)           | 1                 | 107.33  | 7.843          |
|                          | 2                 | 87.17   | 3.326          |
| Hip (cm)                 | 1                 | 104.92  | 8.062          |
|                          | 2                 | 87.83   | 4.218          |
| WHO                      | 1                 | 1.0242  | .02429         |
|                          | 2                 | .9933   | .01923         |
| BMI (kg/m <sup>2</sup> ) | 1                 | 32.4492 | 2.90799        |
|                          | 2                 | 23.4432 | 1.03279        |
| Body Fat (%)             | 1                 | 33.717  | 2.6003         |
|                          | 2                 | 21.850  | 1.4016         |
| Insulin                  | 1                 | 9.558   | 2.0385         |
|                          | 2                 | 6.550   | .8970          |
| Insulin Resistance       | 1                 | 2.4842  | .48874         |
|                          | 2                 | 1.4858  | .24310         |
| Glucose                  | 1                 | 105.67  | 6.814          |
|                          | 2                 | 91.75   | 6.398          |



**Figure 2: The changes pattern of insulin by exercise test. Insulin concentration increased by exercise test in studied obese subjects**

## DISCUSSION

The fact that levels of blood glucose is higher in obese individuals than normal weight individuals is not a new finding, because most previous studies have pointed out high levels of glucose in obese patients

## Research Article

compared with healthy or normal weight people (Sigal *et al.*, 2006). However, insulin resistance has been also reported by some previous studies.

The present study showed that the studied obese men had significantly higher levels of glucose, insulin, and insulin resistance than normal weight men. In support of some scientific references and based on the findings of this study, higher levels of insulin in obese men may be also attributed to over-response of beta cells of the pancreas for increasing insulin secretion in order to overcome insulin resistance; in this regard, it was pointed out in literature that in response to increased resistance of target cells or decreased sensitivity of cells to insulin, beta cells of the pancreas secrete more insulin in order to cope with insulin resistance and to maintain blood glucose level (Kriketos *et al.*, 2004).

It should also be noted that this is a temporary adjustment and does not work in long-term, because the continuation of this situation is associated with hyperactivity of beta cells and their decreased function in long-term (Kriketos *et al.*, 2004). This phenomenon is far more visible in type 2 diabetic patients. It was well known that proper diet and short- or long-term exercise can lead to improved blood glucose levels, especially in obese or diabetic populations (Pradhan *et al.*, 2003; Pan *et al.*, 1997). In support of these observations, we showed that a relatively short one-session exercise significantly reduced blood glucose in obese men, while, serum insulin levels increased after exercise. It may be concluded from these findings that exercising can lead to an immediate response of insulin. In fact, these results pointed out that one-session exercise had resulted in a significant increase in insulin immediately after the test; this seems to have a potential contribution in reduction of blood glucose after exercise which led to simultaneous reduction in blood glucose and increase in insulin.

On the other hand, blood glucose levels have significantly decreased in response to exercise, but no significant changes were observed in insulin resistance. Deriving a general conclusion from these findings is somewhat difficult, because based on what was observed in the literature, several molecular mechanisms are involved in maintaining glucose homeostasis, including balance in inflammatory or anti-inflammatory cytokines (Halle *et al.*, 1999; Meuller *et al.*, 1998). Significant reduction in blood glucose in the absence of change in insulin resistance in the current study, reemphasize this point that other factors are also effective in improvement of glucose balance in response to exercise; because some other studies support the importance and potential role of cytokines such as leptin, adiponectin, or other anti-inflammatory cytokines in energy homeostasis and lipid and glucose metabolism (Hu *et al.*, 1996; Van Gaal *et al.*, 1999; Snehaltha *et al.*, 1999; Trujillo *et al.*, 2005).

## REFERENCES

- Bennett PH (2004).** Epidemiology of Diabetes Mellitus. In: *Diabetes Mellitus: A Fundamental and Clinical Text* edited by LeRoith D, Talor SI and Olefsky JM 3rd edition (Lippincott William & Wilkins).
- Bjornholt JV, Erikssen G, Liestol K, Jervell J, Thaulow E and Erikssen J (2000).** Type 2 diabetes and maternal family history: an impact beyond slow glucose removal rate and fasting hyperglycemia in low risk individuals: Results from 22.5 years of follow-up of healthy no diabetic men. *Diabetes Care* 23(9) 1255-59.
- Diabetes Prevention Program Research Group (2003).** Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *New England Journal of Medicine* 346 393-403.
- Edward P, Feener and Victor J Dzau (2005).** Pathogenesis of cardiovascular disease in diabetes. In: *Joslin's Diabetes Mellitus* edited by Kahn CR, Weir GC, King GL, Jacobson AM, Moses AC and Smith RJ 14th edition (Lippincott Williams & Wilkins) 867-75.
- Garrow JS (1999).** Obesity: definition, Aetiology and Assessment. *Encyclopedia of Human Nutrition* (Academic press) 3 1430-34.
- Halle M, Berg A, Garwers U, Grathwohl D, Knisel W and Keul J (1999).** Concurrent reductions of serum leptin and lipids during weight loss in obese men with type II diabetes. *American Journal of Physiology* 277 277-282.
- Hu E, Liang P and Spiegelman BM (1996).** AdipoQ is a novel adipose-specific gene dysregulated in obesity. *Journal of Biological Chemistry* 271 10697-703.

### Research Article

**Kriketos AD, Gan SK, Poynten AM, Furler SM, Chisholm DJ and Campbell LV (2004).** Exercise increases adiponectin levels and insulin sensitivity in humans. *Diabetes Care* **27** 629–630.

**Lazar MA (2005).** How obesity causes diabetes: not a tall tale. *Science* **307** 373–375.

**Meuller WM, Gregoire FM and Stanhope KL (1998).** Evidence that glucose metabolism regulates leptin secretion from cultured rat adipocytes. *Endocrinology* **139** 551 – 558.

**Pan XR, Li GW and Hu YH (1997).** Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. *Diabetes Care* **20** 537–544.

**Pradhan AD, Cook NR, Buring JE, Manson JE and Ridker PM (2003).** C-reactive protein is independently associated with fasting insulin in nondiabetic women. *Arteriosclerosis, Thrombosis, and Vascular Biology* **23** 650-5.

**Sigal RJ, Kenny GP, Wasserman DH, Castaneda-Sceppa C and White RD (2006).** Physical activity/exercise and type 2 diabetes: a consensus statement from the American diabetes association. *Diabetes Care* **29** 1433–1438.

**Snehalatha C, Ramachandran A, Satyavani K, Sivasankuri S and Vijay V (1999).** Difference in body fat percentage does not explain the gender dimorphism in leptin in Asian Indians. *Journal of Association of Physicians of India* **47** 1164- 1167.

**Trujillo ME and Scherer PE (2005).** Adiponectin- journey from an adipocyte secretory protein to biomarker of the metabolic syndrome. *Journal of Internal Medicine* **257** 167-75.

**Van Gaal LF, Wauters MA, Mertens IL, Considine RU and De Leeuw IH (1999).** Clinical endocrinology of human leptin. *International journal of obesity and related metabolic disorders* **23**(suppl 1) 29–36.