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# Cardiorespiratory Fitness in Relation to Fasting Glucose and Insulin Action in Obese Females

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**ABSTRACT: Obesity is a major health problem in people and related with insulin resistance and lower physical fitness. In pre sent study were conducted to test whether cardiorespiratory fitness is associated with beta cell function or glucose in obese women. For these purpose, twenty nine adult females aged 38 ± 4.4 year and body mass index (BMI) 31.7 ± 2.48 kg/m2 selected for this study by accessible sampling. Subjects were non-athletes and non-pregnancy. Fasting blood samples were collected to calculating glucose, insulin and beta cell function. Cardiorespiratory fitness (VO2max) was measured using a Rockport Walking Test. Pearson's correlation coefficients were used to evaluate the correlations between VO2max and other variables. Statistical significance was accepted at p-value<0.05 or lower. Data showed that VO2max is positively correlated with beta cell function and negatively correlated with fasting glucose in studied subjects. These data suggest that cardiorespiratory can be affect glucose concentration may be through beta cell function.**

**Keywords:** Maximal oxygen consumption, Glucose, Obesity

## INTRODUCTION

Access to high-fat and high-calorie foods in one hand, and genetic factors and reduced physical activity on the other hand, contribute to the incidence of obesity. Literature supports a strong positive relationship between the level of fat cells and obesity-related disorders, such as hypertension, atherosclerosis, and insulin resistance. It was found that adipose tissue plays an important role in inflammation and reduced immune system function, especially in the case of obesity [1], so that the increased levels of adipose tissue or subcutaneous fat is associated with increased inflammation in obese patient or healthy obese individuals.

Impaired glucose tolerance and type 2 diabetes increase with age in obese patients [2), where more than 45 percent of the elderly people possess the criteria of type 2 diabetes [3,4]. Progress of type 2 diabetes, particularly in obese patients, is associated with the failure of beta-cells to compensate for insulin resistance [5]. In obese and type 2 diabetic patients, adipokines profile and energy metabolism and insulin function is significantly impaired compared to healthy control individuals [6]. Some studies have supported the increased insulin resistance and decreased insulin sensitivity in obesity [5,7,8]. Reduction in beta-cell function can be observed in some obese subjects, particularly those susceptible to type 2 diabetes [5].

It was found that physical activity increases insulin sensitivity in insulin-resistant obese patients or those with a family history of type 2 diabetes or those with type 2 diabetes. However, these studies have not assessed the effect of exercise on beta-cell function.

On the other hand, obese and diabetic obese individuals have lower levels of cardiorespiratory fitness compared to those with normal weight [11,12]. Reduced cardiorespiratory fitness in obese individuals is often rooted in the lack of activity and exercise. Based on this evidence, it appears that there is a relationship between insulin resistance or beta cell function and cardiorespiratory fitness, especially in those who have a sedentary lifestyle. Since the studies in this area are limited, the present study aimed at determining the association between beta-cell function and cardiorespiratory fitness (VO2max) in a group of obese non-athlete women.

## METHOD AND METHODS

*A. Study Subjects and Recruitment*

Twenty nine healthy adult obese women (38 ± 4.4 years, BMI 31.7 ± 2.48 kg/m2. M ± SD) participated in the study by accessible sampling. All subjects were otherwise in good health were taking no medications. Participants were non-athletes, non-smokers and non-pregnancy. Participants were included if they had not been involved in regular physical activity/diet in the previous 6 months.

We also excluded people who had any self reported physician diagnosed chronic disease (arthritis, stroke, diabetes, hypertension, cancer, heart attack, chronic cough, or bronchitis). After the nature of the study was explained in detail, informed consent was obtained from all participants.

*B. Anthropometry*

Each subject's anthropometrical markers were measured. Anthropometric measurements were performed in all study participants before breakfast, with the subject wearing light clothing without shoes. Standing height of the barefoot subjects was measured to the nearest 0.1 cm with the use of a wall-mounted stadiometer. Weight was measured to the nearest 100 g using digital scales. Obesity was measured by body mass index (BMI). Body mass index (BMI) was calculated by dividing body mass (kg) by height in metres squared ($m^2$). Abdominal circumference and hip circumference were measured in the most condensed part using a non-elastic cloth meter. Percentage body fat was measured using body composition monitor (OMRON, Finland).

***C****. Laboratory and Training exercise*

Cardiorespiratory fitness (VO2max) was measured using a Rockport Walking Test [13]. In this exercise test, after a brief warm up, the subject walks as briskly as possible for one mile (1609 meters) with a heart rate monitor. Tester records heart rate (beats per minute) and time of completion. Evaluates cardiovascular fitness for adults. It is important to accurately measure exercise heart rate. Heart rate was electronically monitored in the original study.

Venous blood was collected from subjects after an overnight fast. Plasma insulin was determined using ELISA method. Glucose was determined by the oxidase method (Pars Azmoon, Tehran). The homoeostasis model assessment (HOMA) for estimating beta cell function was calculated by fasting glucose and insulin [14].

*D. Data analysis*

Normal distribution of data was analyzed by the Kolmogorov-Smirnov normality test. Data were analyzed by computer using the Statistical Package for Social Sciences (SPSS) for Windows, version 15.0. Pearson's correlation coefficients were used to evaluate the correlations between VO2max with glucose concentration and beta cell function. All statistical tests were performed and considered significant at a $P \leq 0.05$.

## RESULTS

In present study, VO2max as cardiorespiratory fitness in response to fasting glucose and beta cell function were determined in adult obese women. Body weight, all anthropometrical markers and blood chemistry parameters are shown in Table 1.

Data of Pearson's correlation coefficients showed that cardiorespiratory fitness was negatively correlated with fasting glucose ($p= 0.030$, $r = 0.40$, Fig. 1). A significant positive correlation was also observed between VO2max and beta cell function ($p = 0.007$, $r = 0.49$, Fig. 2).

## DISCUSSION

The main finding of this study was the significant relationship between VO2max, as an indicator of cardiovascular fitness, with beta-cell function. In other words, increase in cardiovascular fitness is associated with increase in beta-cell function. Although some earlier studies measured the relationship between cardiorespiratory fitness with some other biochemical markers, such as inflammatory or anti-inflammatory cytokine, this study was one of the few to investigate the relationship between VO2max and beta-cell function in obese women.

**Table 1: The descriptive anthropometric of studied patients.**

| Variable | Mean | Standard deviation | Range |
|---|---|---|---|
| **Age** (years) | 38 | 4.4 | 28 – 46 |
| **Weight** (kg) | 81.7 | 6.9 | 67 – 96 |
| **Height** (cm) | 161 | 5.4 | 152 – 172 |
| **Body mass index** ($kg/m^2$) | 31.7 | 2.48 | 29 – 39 |
| **Body Fat (%)** | 45.1 | 3.72 | 36 – 54 |
| **Abdominal Circumference** (cm) | 109 | 8.2 | 96 – 124 |
| **Hip circumference** (cm) | 111 | 7.5 | 97 – 123 |
| **Fasting glucose** (mg/dl) | 93 | 10 | 76 – 114 |
| **Serum insulin** (μIU/ml) | 7.3 | 4.39 | 3 – 18.8 |

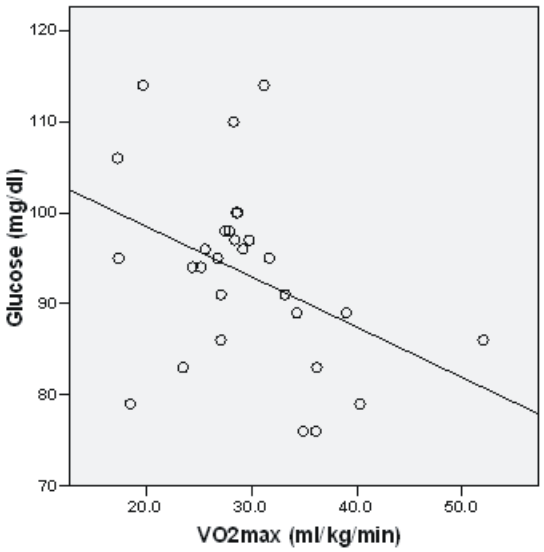


**Fig. 1.** Significant negative correlation between VO2max and fasting glucose.

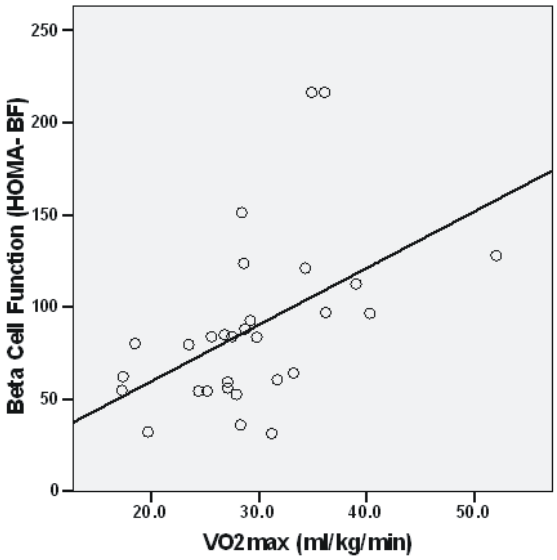


**Fig. 2.** Significant positive correlation between VO2max and Beta cell function.

The findings of this study supports the hypothesis that reduced physical activity or sedentary, which is characterized by a decreased cardiorespiratory fitness, is associated with increased blood glucose levels, especially in obese healthy or patient populations because in the present study, in addition to direct and significant relationship with beta cells, VO2max also had a negative significant correlation with fasting glucose levels. Regarding the relationship between VO2max and other biochemical markers that are affected by obesity, findings of a recent study support the effect of improved aerobic fitness (VO2max) in of the prevention of systemic inflammation [15]. In another study, a significant relationship was observed between VO2max and inflammatory markers such as CRP, IL-1B, IL-10, and TNF-α in overweight children [16]. A strong relationship was observed between VO2max and factors determining obesity and inflammatory profile with emphasis on CRP [11].

Findings of the present study, which suggest a direct relationship between VO2max and beta-cell function and an inverse relationship between VO2max with blood glucose, conclude that a dynamic lifestyle and participating in long-term aerobic training, which is associated with increased cardiorespiratory fitness, reduce blood glucose levels and improve insulin sensitivity as well as beta cell function. It was found that three months of exercise training increases VO2max in adults with type 2 diabetes [17]. In this regard, the findings of a study showed that insulin resistance and fasting fatty acid levels are independent predictor of VO2max [12].

Literature have pointed out that diet or exercise affects beta-cell function and mass as well as the insulin resistance [18]. Diet and exercise both increase insulin secretion while having mechanism of action independent of each other. A high fat diet increases beta-cell mass trough hypertrophy to overcome insulin resistance while aerobic training increases beta-cell mass trough hyperplasia. This hyperplasia is induced by beta cell proliferation and reduced apoptosis [18].

Some studies have shown that homeostasis exercise improves glucose by enhancing glucose uptake in skeletal muscle and adipose tissue [19-21]. Moreover, some studies reported that aerobic training reduces liver insulin signals by reducing hepatic glucose release in hyperinsulinemia [7-8]. Although some studies have shown that exercise improves the mass and function of beta-cells in type 2 diabetic patients [18]. Improved beta-cell function in type 2 diabetic obese in response to weight loss strongly supports the hypothesis that obesity and its associated metabolic disorders are a factor in destruction of the beta cells.

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