



## Aerobic Capacity and High Sensitivity C - reactive protein in Obese Men

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(Received 08 September, 2014, Accepted 21 October, 2014)

(Published by Research Trend, Website: [www.researchtrend.net](http://www.researchtrend.net))

**ABSTRACT:** Aerobic capacity or cardiorespiratory fitness is hypothesized to be related with systemic inflammation. The association of aerobic capacity ( $VO_2^{\max}$ ) as cardiorespiratory fitness with C - reactive protein (CRP) as pro-inflammatory cytokine and obesity determinants was determined in twenty eight inactive adult obese men aged  $37.1 \pm 4$  year and body mass index (BMI)  $32.7 \pm 3$   $kg/m^2$  was determined in present study. Pearson's correlation coefficient was run for testing of correlation analysis. There were no correlation between CRP concentration and any aerobic capacity in studied subjects ( $p = 0.94$ ). In contrast, aerobic capacity ( $VO_2^{\max}$ ) is strongly and inversely associated with anthropometrical markers as obesity determinants ( $p > 0.05$ ). This finding does not confirmed of CRP as a predictor of cardiorespiratory fitness on inactive obese subjects.

**Keywords:** C - reactive protein, Aerobic capacity, Obesity, Inflammation

### INTRODUCTION

Obesity and related diseases are known as the leading causes of death in both developed and developing countries [1]. Obesity is associated with chronic inflammation and increased secretion of inflammatory cytokine [2]. In addition to elevated circulating levels of cytokines, obese people have higher levels of intracellular cytokines [3]. Cytokines are pro-inflammatory and anti-inflammatory mediators produced by peripheral blood mononuclear cells, adipocytes, hepatocytes, and skeletal muscles [4].

Chronic inflammation occurs in response to increased inflammatory cytokines like C-reactive protein (CRP) [5]. CRP is an inflammatory cytokine produced by the liver in response to acute infection or inflammation, and its plasma concentration can increase up to 1000 times in response to an injury or infection [6]. CRP is mainly synthesized by hepatic ducts, and is regulated by IL-1b, IL-6, and TNF- . According to the scientific reports, as an inflammatory mediator, CRP is a better indicator than other cytokines in predicting cardiovascular diseases [7]. Several scientific sources suggest that CRP is the most important and useful clinical marker in identifying inflammation and evaluating cardiovascular diseases risk factors [8].

Scientific sources support this definition that obese people have higher levels of CRP than those with normal weight [9]. Low cardiorespiratory fitness or

aerobic capacity in obese people has also been reported frequently [10]. High fat diet, inactivity, and lack of exercise contribute to obesity and its related diseases. In this regard, scientific sources have always supported decreased aerobic capacity and levels of physical fitness in the presence of obesity [11]. The question is, whether low aerobic capacity or cardiorespiratory fitness is an independent outcome of physical inactivity and increased body fat of obese people, or is influenced by other factors such as hormonal components or inflammatory mediators? In response to the question, findings are limited and often conflicting. Some of the findings pointed out that cardiorespiratory fitness is associated with inflammatory components [12], and others pointed to the lack of relation between them [13]. Based on the existing evidence, this study is conducted to determine the relationship between  $VO_2^{\max}$  as a physiological determinant of cardiovascular fitness and CRP as a leading inflammatory cytokine in obese people.

### METHODS

#### A. Subjects

As mentioned above, in this study, we aimed to determine relationship between serum CRP and aerobic capacity in a group of non-trained obese men. Subjects were twenty eight inactive adult obese men aged  $37.1 \pm 4$  year and body mass index (BMI)  $32.7 \pm 3$   $kg/m^2$  that participated in study by accessible samples.

They were in active and non-smoker. All participants reported being weight stable ( $\pm 1$ kg) for 6 months prior to the study and engaged in physical activity less than once per week. Subjects with a history or clinical evidence of impaired fasting glucose or diabetes, recent heart failure, active liver or kidney disease, or who were on medications were excluded. 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. Anthropometric measures

Body weight, height, waist circumference and % body fat measurements were obtained by standard methods. Weight and height were measured in the morning, in fasting condition, standing, wearing light clothing and no shoes. Body mass index (BMI) was calculated as weight (kg) divided by squared height (m). 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 of body fat was estimated by bioelectrical impedance method (Omron Body Fat Analyzer, Finland).

#### C. Blood analysis and $VO_2$ max calculation

CRP was measured after an overnight fast when subjects fasted for 10-12 hours. The Intra-assay coefficient of variation and sensitivity of the method were 8.3% and 10 pg/mL, respectively for CRP. Maximum volume consumption ( $VO_2$ max) was measured using a bicycle ergometer (Tuntury, F90,

Finland) in a stepwise fashion according to YMCA protocol [14].

#### D. Analyses

Statistical analysis was performed with the SPSS software version 16.0 using a Pearson correlation method to determine the relationship between  $VO_2$ max with CRP and anthropometrical markers. P value of less than 0.05 was regarded as indicative of a significant difference.

### RESULTS

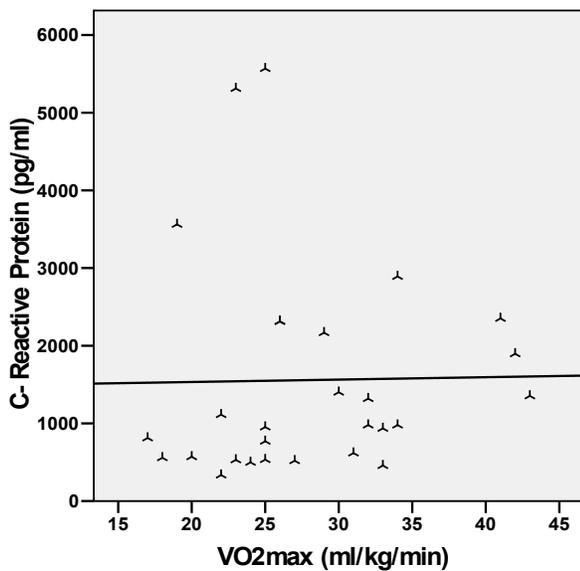
Relationship of  $VO_2$ max of a determinant marker of cardiorespiratory fitness with CRP as a inflammatory cytokine ere determined of adult obese men in present study. We also determined relation of  $VO_2$ max with obesity determinants in studied subjects. Table 1 shows the descriptive anthropometric features, aerobic capacity and CRP of the study groups. Data of Pearson's correlation coefficients showed that serum CRP was not associated with  $VO_2$ max in obese studied subjects ( $p = 0.94$ ,  $r = 0.02$ , Fig. 1).

Despite lack relation of  $VO_2$ max with CRP, this physiological marker was negatively correlated with body weight in studied subjects ( $p = 0.003$ ,  $r = 0.55$ , Fig. 2).

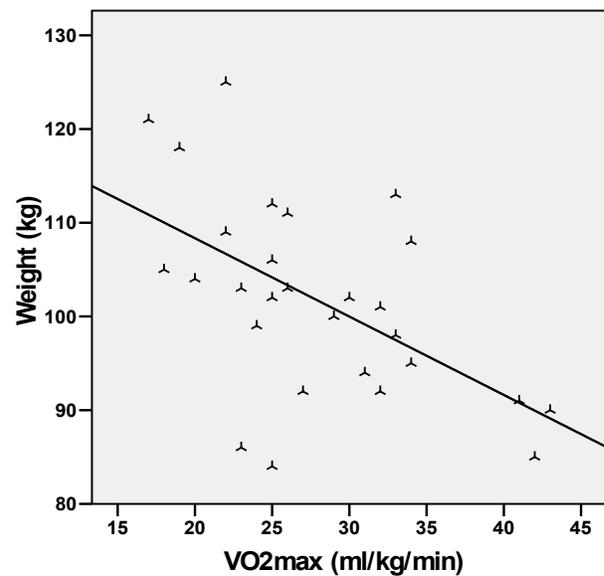
We found that this physiological marker negatively associated with body mass index ( $p = 0.01$ ,  $r = 0.48$ , Fig. 3). Cardiorespiratory fitness was also negatively correlated with abdominal circumference in obese subjects ( $p = 0.04$ ,  $r = 0.39$ , Fig. 4).

**Table 1: Mean and standard deviation of physical and physiological markers of studied subjects.**

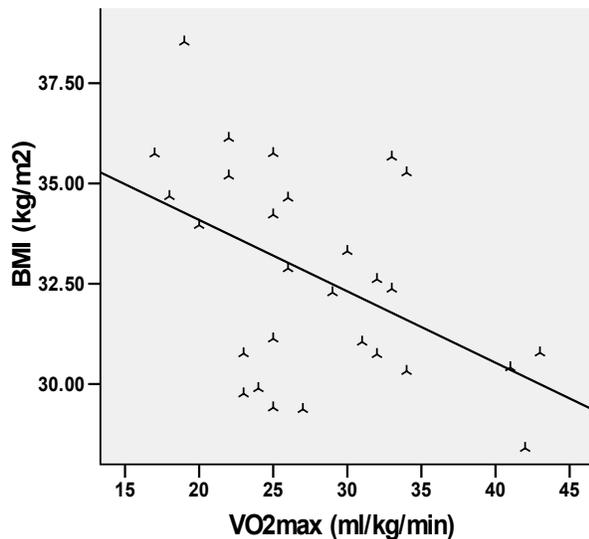
Variables	N	Minimum	Maximum	Mean	Std. Deviation
Age (year)	28	26	43	37.2	4.08
Height (cm)	28	169	186	176.3	4.05
Weight (kg)	28	84	125	101.8	10.6
Abdominal (cm)	28	93	130	108	8.3
Hip (cm)	28	90	133	106	8.5
AHO	28	.96	1.05	1.01	0.02
BMI (kg/m <sup>2</sup> )	28	28.4	38.5	32.7	2.60
Body fat (%)	28	28.9	36.4	32.6	2.01
CRP (pg/ml)	28	335	5568	1557	1375
$VO_2$ max (ml/kg/min)	28	17	43	27.9	6.96



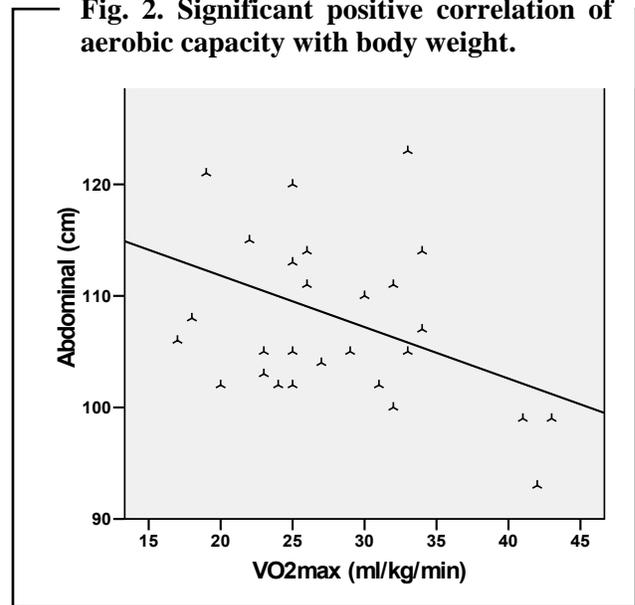
**Fig. 1. No Significant correlation between aerobic capacity and C - reactive protein.**



**Fig. 2. Significant positive correlation of aerobic capacity with body weight.**



**Fig. 3. Significant positive correlation of aerobic capacity with body mass index.**



**Fig. 4. Significant positive correlation of aerobic capacity with abdominal (cm).**

## DISCUSSION

Systemic inflammation is a condition that the levels of systemic inflammatory cytokines such as TNF- $\alpha$ , IL-6, IL-1 $\beta$  and CRP increase up to 2 or 3 times [15]. But it is not yet clear whether or not the presence of systemic inflammation contribute to aerobic capacity or cardiorespiratory levels in healthy obese people or

patients. There are different answers for these questions. Lack of relationship between serum levels of CRP and aerobic capacity was also detected in this study. Findings of the study indicated that elevated serum CRP levels are not associated with cardiorespiratory fitness in obese men, and their values are independent from each other.

Adipose tissue is a great source of stored body fat. As a dynamic endocrine organ, adipose tissue secretes a number of peptide mediators which are capable of controlling fat stores (lipid metabolism), sensitivity to insulin, regulation of blood pressure, and energy balance [16, 17]. Excessive body fat contributes to systemic disorders of pro-inflammatory and anti-inflammatory mediators which may ultimately lead to prevalence of cardiovascular disease, type 2 diabetes and other obesity-related diseases in obese people [18]. The link between inflammatory components and insulin resistance has been detected in previous studies [19]. CRP is an effective inflammatory mediator to determine the levels of inflammation in most diseases. So that, CRP measurement is believed to be the only factor to detect inflammation; however, measurement of other inflammatory markers along with CRP may provide complimentary information about the mechanisms involved in inflammation [20]. Higher levels of CRP are associated with coronary heart diseases, obesity, diabetes, smoking, and sedentary lifestyle [21]. A recent study indicated that CRP along with age, blood pressure, and diabetes are the leading factors contributing to cardiovascular diseases in obese people [22].

Lack of association between CRP and aerobic capacity was observed in this research, while some of the previous studies supported the significant relationship between inflammatory cytokines and physiological factors associated with cardiorespiratory fitness. For example, in a recent study, a significant relationship between  $VO_2\text{max}$  and serum levels of other inflammatory cytokines such as TNF- $\alpha$  and IL-6 was detected in overweight children [12]. In another study, higher levels of IL-6 and CRP were associated with lower levels of  $VO_2\text{max}$  in asymptomatic men [23].

It is possible that not all increased inflammatory cytokines contribute to cardiorespiratory fitness. In this regard, a recent study indicated that despite a significant relationship between  $VO_2\text{max}$  and IL-6 in overweight young men, no significant relationship was observed between  $VO_2\text{max}$  and TNF- $\alpha$  [12]. Another study also indicated that regardless of the association between  $VO_2\text{max}$  and serum levels of IL-6 and TNF- $\alpha$ , no relationship was detected between  $VO_2\text{max}$  and other proinflammatory or anti-inflammatory factors such as CRP, IL-1b and IL-10 [24]. In another study on Swedish children, no significant relationships were noted between CRP and fibrinogen [13]. In this study, when fat mass was entered into the regression model, no association was observed between levels of physical activity, cardiorespiratory fitness, and inflammatory markers [13]. Despite the lack of association between

$VO_2\text{max}$  and CRP serum levels among obese men in this study, it is likely that levels of cardiorespiratory fitness are associated with CRP receptors in skeletal muscle IL-6 and TNF- $\alpha$  gene expression. It is also possible that CRP levels indirectly affect cardiorespiratory fitness levels through other pathways such as other hormonal factors.

Despite the lack of significant association between  $VO_2\text{max}$  and CRP serum levels, the research findings indicated an association between  $VO_2\text{max}$  levels as a determinant factor of cardiorespiratory fitness and other indicators of obesity such as body weight and body mass index (BMI). So that, there was an adverse correlation between  $VO_2\text{max}$  change patterns and weight and BMI patterns in obese subjects. In other words, people with higher BMI had lower levels of cardiorespiratory fitness. Based on the existing evidences, it can be concluded that aerobic capacity or cardiorespiratory fitness in inactive obese men is dependent on weight and BMI rather than inflammatory factors. However, further studies will be required to better understand the mechanisms responsible for reduced cardiorespiratory fitness in healthy obese people or patients.

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