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International Journal of Recent Scientific Research Vol. 6, Issue, 2, pp.2608-2610, February, 2015 International Journal of Recent Scientific Research

# **RESEARCH ARTICLE**

## **CARDIO-RESPIRATORY FITNESS AND BODY MASS INDEX IN YOUNG MALE ADULTS**

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ARTICLE INFO	ABSTRACT		
Article History:	Background: Cardiovascular disorders have become the leading cause of death worldwide. In the past few		
Received 14 <sup>th</sup> , January, 2015 Received in revised form 23 <sup>th</sup> , January, 2015 Accepted 13 <sup>th</sup> , February, 2015 Published online, 28 <sup>th</sup>	decades, prevalence of cardiovascular disorder has increased substantially in younger population. Increased body fatness is an additional factor for developing cardiovascular diseases. One of the parameter for detecting fatness is body mass index. Maximal oxygen uptake (VO <sub>2</sub> max) has been regarded by majority of authors as the best indicator of aerobic capacity of an organism, and also internationally accepted parameter to evaluate cardio-respiratory fitness.		
February, 2015	<b>Objective:</b> The objective of this study was to determine the cardio-respiratory fitness in terms of $VO_2max$ in young healthy males and to study the relation between body mass index and cardio-respiratory fitness.		
Key words:	Methodology: 30 young healthy male subjects in the age group of 18 to 25 years were included in this		
VO <sub>2</sub> max, QCT, body mass index, cardiorespiratory fitness	study group. Body mass index was measured as weight (in kilograms) divided by square of height (in meters). Cardio-respiratory fitness in terms of VO <sub>2</sub> max was assessed by following the protocol of Queen's College Step Test (QCT).		
	RESULTS: The mean values for body mass index , mean predicted VO <sub>2</sub> max were $18.53 \pm 1.29 \text{ kg/m}^2$ was		
	37.01 $\pm$ 3.94 ml/kg/min respectively. There was a significant negative correlation between body mass index		
	(BMI) and VO <sub>2</sub> max.r=(02)		
	<b>Conclusion:</b> The results suggest that cardio-respiratory functions are affected by body fat. Excessive amount of body fat exerts an unfavourable burden on cardiac function and oxygen untake by working		

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developing cardiovascular comorbidities later in middle age.

## **INTRODUCTION**

In the last few decades there is global rise in cardiovascular disorder. Cardiovascular disorders have become the leading cause of death worldwide. With improvement of health care system the cause of death and disability have shifted from communicable diseases towards non communicable disorders.<sup>1</sup>

The change in life styles that is shifting more towards sedentary life is occurring throughout the developing world. According to world health report of WHO, 2003 out of all the deaths attributable to non communicable diseases (35 million) just over half of these (17 million) are result of cardiovascular diseases.<sup>2</sup> Cardiovascular diseases are responsible for about 25% of the DALY `S lost due to non communicable diseases in south east asia region in accordance with 2008 WHO update of the report "Global burden of disease". <sup>2</sup> India is experiencing an alarming increase in cardiovascular diseases accounting for 32% of all deaths in 2000.<sup>3</sup>According To Government Of India Annual Report 2006-7, Ministry Of Health & Family Welfare compared with all other countries India suffers the highest loss in potentially productive years of life due to death from

cardiovascular diseases in people aged 35-64 years by 2030 this loss is expected to rise 9.4 times greater loss in USA.<sup>2</sup>

There are several reports that indicate increased mortality in individuals with low cardio-respiratory fitness.<sup>4,5,6</sup> The prevalence of CVD has increased substantially in younger population because of development of CVD risk factors in young people.<sup>7</sup> The **aim** and **objectives** of the present study are to estimate the maximal oxygen uptake in youg males, to correlate it with body mass index.

## **MATERIAL AND METHOD**

muscles. Low cardio-respiratory fitness in young adults with increased body fat could be a factor for

30 male Individuals in the age group of 18 to 25 years, were selected. Individuals having history of any chronic disease or addiction, history of trauma or injury are excluded from studies. Subjects were having a sedentary lifestyle with no involvement in any athletic activity or yoga. All the subjects were well explained about the nature of the study and the detailed procedure of the study. The information was entered in a proforma given to each participant. Thorough general examination was done. Then a detailed systemic examination of the cardiovascular, respiratory, abdomen and central nervous system was also done. Written consent was taken from

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participants. The approval of the study protocol was obtained from the Institutional Ethics Committee.

The subjects and controls were called early in the morning. Body Weight was measured using a calibrated weighing machine with subjects in light clothing and bare feet. Height was measured using the height measuring scale fixed to the wall.

#### Assessment of VO<sub>2</sub>max

Queens's college step test was used for the indirect estimation of maximal aerobic capacity by the standard method of Mcardle et. al. 2001.<sup>8</sup>

Bench stepping sub maximal exercise was used according to the protocol. Prior to the test subjects performed 5 to 7 min. warm up consisting of lower limb stretching & brisk walking. Wooden bench of height 16.25 inches was used along with stop watch. A standardized system software Metronome was used to measure the stepping cadence which will be set at 96 beats per min. i.e. 24 complete steps for males. The step test began after a brief period of demonstration and practice period and the subjects performed the test for complete 3 min. After completion of the test subjects remained standing while their carotid pulse rate was measured from 5 to 20 sec. into recovery. Fifteen second Recovery heart rate was converted to be expressed as beats per minute (15 second Heart Rate x 4).

Following formula was used for determining VO<sub>2</sub>max. 9, 10

For males:  $VO_2max = 111.33 - (0.42 \text{ X PR})^8$ 

For each parameter, the mean value and standard deviation were calculated. All the calculations and statistics were done using Microsoft Excel 2010. Correlation of VO<sub>2</sub>max with BMI was done using Pearson's correlation coefficient (r). Pearson's correlation coefficient (r) indicates the strength of correlation of VO<sub>2</sub>max with BMI. Positive r value indicates positive correlation & negative r value indicates negative correlation. Higher the r value greater is the correlation. Statistical significance is denoted by p value. A 'p' value of less than 0.05(p < 0.05) was considered to be statistically significant.

## RESULT

The mean values for body weight, height and body mass index were  $55.13 \pm 3.13$ kg,  $1.72 \pm 0.02$  m and  $18.53 \pm 1.29$  kg/m<sup>2</sup>

respectively. Mean predicted VO<sub>2</sub> max was  $37.01 \pm 3.94$  ml/kg/min. (Table 1 ) When VO<sub>2</sub>max was correlated with BMI

using Pearson's correlation coefficient maximal oxygen uptake  $(VO_2max)$  showed strong negative correlation with, body mass index (r value is -0.2) (p value <0.05) statistically significant.(Table 2)



Graph 1Scatter plot showing negative correlation of VO2max with BMI

#### DISCUSSION

Present study shows negative correlation between  $VO_2max$  and BMI. These are the probable reasons why, BMI, have negative correlation with maximal oxygen uptake ( $VO_2max$ .)

Measurements of body weight and body dimensions (anthropometry) are used to reflect body fat in large (epidemiological) studies or in clinical settings. Body mass index (BMI) has traditionally been used to identify individuals who are the most likely to be overweight or obese. Body mass index (BMI) is employed globally to classify humans as normal, overweight and obese <sup>11</sup>. Compared with assessment methods of body fat percent (BF %), it is inexpensive and easy to administer. Obesity is an epidemic disease. Body weight depends on balance between calorie intake and utilization of calories. Effects of obesity result from two factors, increased mass of adipose tissue, and increased secretion of pathogenetic products from enlarged fat cells.

Increased free fatty acids from fat cells results in insulin resistance. The release of cytokines particularly IL-6 stimulate pro-inflammatory state that characterizes obesity. Increased secretion of prothrombin activator inhibitor-1 from fat cells plays a role in procoagulant state of obesity and along with changes in endothelial function increases risk of cardiovascular disease and hypertension. Combined effects of these consequences of increased fat stores in an increase risk of shortened life expectancy.<sup>12</sup> Overweight subjects have much greater increase in sympathetic nerve firing rate than normal subjects.

parameter	Weight	Height	Body Mass	Recovery Heart Rate	VO <sub>2</sub> max
	(kilograms)	(meters)	Index(kg/m <sup>2</sup> )	(per minute)	(ml/kg/min)
Subjects (n=30) (Mean <u></u> SD)	55.13 <u>+</u> 3.13	1.72 ± 0.02	18.53 ± 1.29	176.93 🛨 9.37	37.01 ± 3.94

Table 1Baseline characteristics of the Study Group

<b>Table 2</b> Correlation between VO <sub>2</sub> max and BMI in subjects
by Pearson's Correlation

Variat	ole	VO <sub>2</sub> max
DMI	r	-0.298
DIVII	р	0.021 *

\*statistically significant (p value <0.05)

There is disproportionate increase in cardiac output due to increased sympathetic activity which increases blood pressure. Combination of overweight and hypertension leads to thickening of ventricular wall and larger heart volume and thus a greater likelihood of cardiac failure. <sup>13</sup> Cardiac weight increases with increasing body weight suggesting increased cardiac work. Increased cardiac work results in

cardiomyopathy and heart failure in absence of diabetes mellitus and hypertension. Weight loss decreases heart weight. The duration of obesity is important while assessing effects of BMI on heart. Those with early onset of obesity had major effects.<sup>8</sup>Our study have been supported by studies of Shazia et al (2015)<sup>15</sup>, Chatterjee et al (2005)<sup>14</sup>, Ozcelik et al (2003)<sup>16</sup>, Salbatori et al (1999)<sup>17</sup>, Watanabe et al (1994)<sup>18</sup>. The data of the present study clearly demonstrated, that a strong negative correlation existed between aerobic fitness and body-fat. Excessive amount of body fat exerts an unfavorable burden as well as hindering action towards cardiac function particularly during exhaustive exercise when excessive hyperactive body musculature fails to uptake sufficient amount of oxygen due to deposition of proportionately high amount of fat mass. Loss of weight during weight reduction program of obese increased their VO<sub>2</sub>max(ml/kg/min) due to withdrawal of fat induced inhibitory action towards oxygen utilization by body musculature.<sup>14</sup>

# CONCLUSION

There was a significant negative correlation between BMI and  $VO_2max$  (ml/kg/min). This suggests the possibility of effect of body fat on cardio-respiratory functions. Excessive amount of body fat exerts an unfavorable burden as well as hindering action towards cardiac function during exercise. Reduced cardiac performance during progressive exercise results in decreased oxygen uptake. Due to deposition of proportionately high amount of fat mass, body musculature fails to uptake sufficient amount of oxygen. These findings demonstrate the importance of low cardio-respiratory fitness in young adults with increasing BMI which could be a factor for developing cardiovascular comorbidities later in middle age.

**Limitation**. The limitation of the present study was the small sample size of subjects. So, it is important to replicate and extend our observations to large population. Future scope;

- 1. To evaluate, and compare the cardio-respiratory fitness in terms of  $VO_2max$  in males and females.
- 2. To evaluate correlation of  $VO_2max$  and BMI in females.

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## How to cite this article:

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Afshan Kausar and Syed Mudassir: Cardio-respiratory fitness and body mass index in young male adults. International Journal of Recent Scientific Research, Vol. 6, Issue, 2, pp.2608-2610, February, 2015