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# Effect Of Body Mass Index On Fev1

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***Abstract***
***Background:*** *The prevalence of obesity is rising at an astonishing rate over the last decade globally making the individual highly susceptible to various diseases. It has transformed into a major public health issues in India. These changes may reduce the compliance of the lungs and the thoracic cavity and increase the load on the respiratory muscles. This may end up with the reduction in forced expiratory volume in first second ($FEV_1$).*
***Aim and Objective:*** *The aim of the study is to see the effect of body mass index (BMI) on the FEV1 in adults.*
***Methodology:*** *This cross sectional analytical study was undertaken in the Physiology Department of Laxmi Chandrawanshi Medical College over a period of 6 months on 500 healthy individuals with age of 18–40 years old to determine the baseline anthropometric measurement followed by measurement of FEV1 using a computerized Spirometer. The subjects were divided in two groups based on their BMI. Group A with BMI <25 kg/m2 and Group B with BMI ≥25 kg/m2. Mean ± SD of BMI and FEV1 for both groups were calculated.*
***Results****: The FEV1 showed a statistically significant decline in overweight/obese individuals. The mean BMI in Group A (normal) was 20.80 ± 2.28, whereas in Group B (obese) was29.27 ± 2.77. The mean FEV1 was 3.54 ± 0.49 L in normal BMI group as compared to 3.47 ± 0.54L in obese BMI group.*
***Conclusion:*** *The FEV1 showed a compromised status in overweight/obese individuals. This difference can be attributed to the presence of adipose tissue around the ribcage, in the abdomen and visceral cavity loading the chest wall, leading to small airway collapse and subsequent lowering of the FEV1.*

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## I. Introduction

Globally, the epidemic of obesity in children and young adults is rapidly increasing, rising by more than 30% over the past decade with it's prevalence in India ranges from 10% to 40% [1]. It is classically characterized by increased body fat (BF), associated with dyslipidemia, insulin resistance, cell organelles stress, and vascular inflammation. Increased weight makes obese individual highly susceptible to various diseases.

Obesity is a worldwide epidemic; 66% of US adults are overweight or obese [2], and a prevalence of obesity up to 31% has been reported in Europe [3]. If current trends continue, the number of obese individuals is expected to rise to over 1 billion worldwide by 2030 [4]. It has transformed into a major public health issues in India.[5]

The etiology of obesity is complex and multifactorial and results from the interaction of genes with the environment, lifestyle, and emotional factors.[6] Rubinstein et al. stated that obesity impairs the respiratory functions by inducing airway hyperresponsiveness in adults,[7] whereas Brashier et al. linked it with the development of asthma.[8]

The multifactorial etiology comprising complex interaction between genetic, behavioral, environmental, and socioeconomic factors, leading to an imbalance in energy production and expenditure.[6]

This interaction comprising decreased energy expenditure leads to storage of excess energy in the form of fat. Pulmonary functional status was assessed by recording Forced Expiratory Volume in 1st sec (FEV1). FEV1 was selected because it is widely accepted as a reliable parameter of pulmonary functions and is simple to perform using a computerized spirometer. FEV1 is the maximum volume of air that can be expelled from the lungs in 1st second after taking the deepest possible breath followed by a forceful expiration.[9]. FEV1 is affected by several physiological factors, including age, gender, weight, height, and ethnicity. The truncal fat may compress the thoracic cavity and restrict the diaphragmatic movement resulting in reduced vertical diameter of the thoracic cavity.[10] These changes may reduce the compliance of the lungs and the thoracic cavity and increase the load on the respiratory muscles and may end up with the reduction in lung volumes, especially Vital Capacity(VC).[11] Presence of adipose tissue around the ribcage, in the abdomen and visceral cavity load the chest wall, leading to small airway collapse and subsequent lowering of the FEV1.[7]

The present study was undertaken to assess and correlate the obesity and pulmonary functional status in obese and nonobese young adult male and female subjects.

## II. Materials And Methods

The sample size of the study was 500 subjects. The anthropometric data were recorded for the volunteer patients after obtaining an informed consent. Standing height was recorded without shoes, with light clothes on a wall by measuring tape. Weight was recorded without shoes and with light clothes on a weighing machine. Body mass index (BMI) was calculated by the formula BMI = Weight(kg)/ Height(cm)2 . Two separate groups were created, Group A consisted of normal BMI individuals, that is, BMI < 25 kg/m2 and Group B with BMI ≥25 kg/m2. All healthy subjects willing to participate in the study without any medical illness such as cardiorespiratory and neurological diseases or endocrinal and allergic disorders with no medication history for any ailments were included in the study. Subjects with habit of smoking and alcohol consumption were not included in the study. Ethical clearance was obtained from the Institution Ethics Committee and informed consent was obtained from all subjects. The representative samples were enrolled from age group 18–40 years comprising subjects from both genders. The subjects were instructed to take maximum inspiration and blow into the mouthpiece as forceful and as fast as they can. They were trained well to blow into the instrument maintaining a tight sealing between the lips and mouthpiece of the computerized spirometer. The procedure was repeated 3 times and the highest of the three readings was recorded.

## III. Results

The present study was done to study the effect of BMI on FEV1 in healthy young adults. The mean BMI in Group A was 20.80 with standard deviation of 2.28. The mean FEV1 in this group was 3.54 L with standard deviation of 0.49 [Table 1].

**Table 1**

| VARIABLES | n | minimum | maximum | mean | Standard deviation |
|---|---|---|---|---|---|
| BMI < 25 | 250 | 16.62 | 24.97 | 20.802 | 2.278 |
| FEV1 | 250 | 2.53 | 4.79 | 3.542 | 0.492 |

The mean BMI in Group B was 29.27 with standard deviation of 2.77. The mean VC in this group was 4.05 L with standard deviation of 0.64 [Table 2].

**Table 2**

| VARIABLES | n | minimum | maximum | mean | Standard deviation |
|---|---|---|---|---|---|
| BMI ≥ 25 | 250 | 25.03 | 38.32 | 29.272 | 2.772 |
| FEV1 | 250 | 2.23 | 4.27 | 3.47 | 0.54 |

Table 3 shows comparative analysis of FEV1 among non-obese and obese subjects using Independent 't' test. Obese subjects exhibit a significant decline in FEV1 ($p<0.01$) as compared to non-obese individuals.

**Table 3**

| PFT Parameter | Non obese (BMI < 25) (n=250) Mean ± SD | Obese (BMI ≥ 25) (n=250) Mean ± SD | 'p' value |
|---|---|---|---|
| FEV1 | 3.54 ± 0.49 | 3.47 ± 0.54 | <0.01 |

**Fig-1** compares dependency of forced expiratory volume in first second (FEV1) on BMI between non-obese and overweight/obese subjects using multiple linear regression analysis. Among overweight/obese subjects, the slope of best fitted regression line 'β' was estimated as -0.09 which means that average baseline

FEV1 deccreases by -0.09 L or 9ml for every 1 kg/m2 increase in BMI. Furthermore, a value of R2=0.28 vehemently explains 28% of FEV1 on BMI.

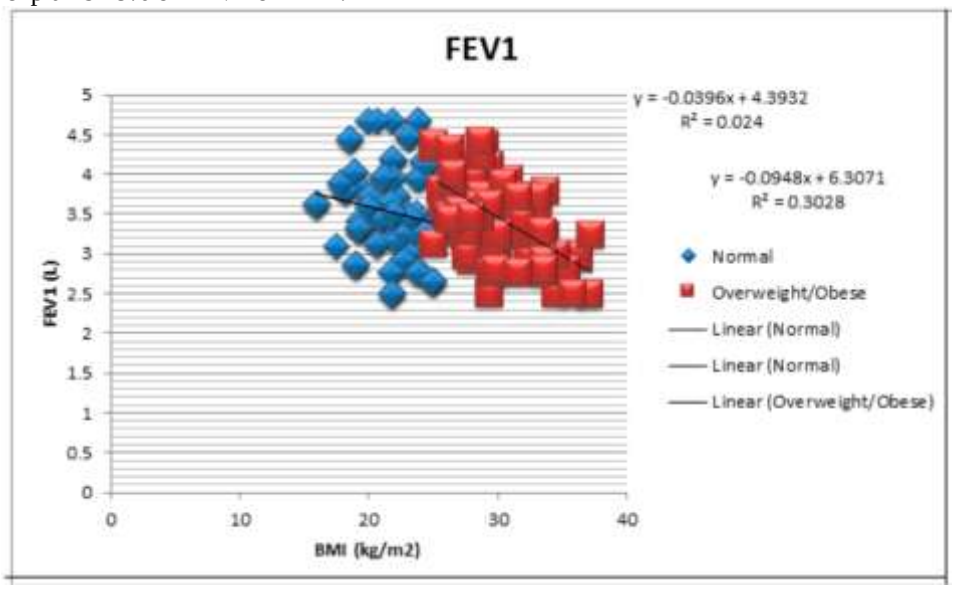


**Figure 1**

## IV. Discussion

Our study shows that increase BMI decrease FEV1. Obesity is a condition, in which a person has excess body weight relative to other people of the same gender and height. Respiratory problems are associated with obesity and these occur when the extra weight of the chest wall squeezes the lungs and causes restricted breathing. It is generally accepted that increased body mass loading of the respiratory apparatus (chest and the lungs) plays a role in the development of respiratory failure by causing either an overwhelming load to the respiratory muscle or substantial ventilation perfusion disparities. Huang et al. showed that abdominal fat is negatively and consistently associated with pulmonary function.[12] Kamal et al. found that obese subjects are at a risk of lung function weakening, based on the criteria followed for BMI and percentage of body fat.[13] Kumari et al. showed that increase in BMI and Waist Hip Ratio (WHR) had an inverse relationship with PEFR in obese when compared to the normal weight subjects.[14] Parameswaran et al. concluded from their study on obese women that the body mass appears to exert a greater influence on inspiratory reserve volume, whereas a greater BMI is associated with a decline in expiratory reserve volume.[11] Sutherland et al. studied that increased adiposity is associated with impairment in lung function across a broad range of static and dynamic lung volumes and that these values do not appear to depend whether the body fat is centrally or peripherally distributed.[15] Our results are in concordance with the findings of many other studies that have shown various lung volumes and capacities decrease with increasing BMI. Our study is an attempt to bring understanding about deviation of lung function with increase BMI. The evidence may help to recognize the pulmonary health risks that crop up with increasing BMI and fat accumulation.

Hence, our study is an effort to bring understanding about the difference of lung function with increase in BMI. The data may help to acknowledge the pulmonary health risk that crop up with increasing BMI and fat accumulation.

## V. Limitations

This study is limited by the small number of subjects, preventing further investigation such as the possible role of gender or adiposity distribution. Future study with larger sample size and comparing other pulmonary function parameters relation with the obesity will give more insight into effect of obesity on pulmonary function.

Even though the distribution of obesity can play a role in respiratory mechanics[10] the collective effect of total chest wall size, rather than changes in any specific regional chest wall fat may be involved, as shown in moderately obese men.[16] Neither the WHR nor the body fat distribution was measured in our study.

## VI. Conclusion

The FEV1 showed a compromised status in overweight/ obese individuals. Mechanically, this can be ascribed to the occurrence of adipose tissue all around the ribcage, in the abdomen and visceral cavity packing

the chest wall, leading to small airway collapse and consequent lowering of the FEV1. Early identification of risk individuals before the onset of disease is imperative in a resource limited country like ours.

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