

**A COMPARATIVE STUDY OF PULMONARY FUNCTION
TESTS IN OBESE & NON OBESE STUDENTS IN THE AGE
GROUP OF 18-25 YEARS**

By

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**A COMPARATIVE STUDY OF PULMONARY FUNCTION TESTS IN
OBESE & NONOBESE STUDENTS IN THE AGE GROUP OF 18-25 YEARS**

ABSTRACT

Background & objective:

A study was conducted to determine the effect of obesity on pulmonary functions and to compare the same between obese and non obese students in the age group of 18-25 years.

Material & methods:

A cross sectional study was conducted on obese (BMI ≥ 30 kg/m²) male (n=32) and female (n=18) students aged 18-25 years and compared with age matched non-obese (BMI 18.5–24.99 Kg/m²) male (n=23) and female subjects (n=27) as control. Detailed anthropometric and physiological data were collected. Pulmonary functions were recorded by using Computerized Spiro excel. Parameters recorded were Forced Vital Capacity (FVC in ml), Forced Expiratory Volume in 1st sec (FEV1 in ml), FEV1 %, Peak Expiratory Flow Rate [PEFR in L/min was recorded by mini Wright's Peak flow meter] and Maximum Expiratory Pressure [MEP in mmHg was recorded by Modified Black's apparatus].

Statistical analysis was done by calculating Mean \pm SD by using student's t-test. Correlation between degree of obesity and pulmonary functions was done by Pearson's correlation

Results:

Systolic Blood Pressure, Diastolic Blood Pressure, Pulse Rate and Respiratory Rate were significantly higher in obese students when compared to their respective control. We observed highly significant reduction in PEFR (p<0.001) and MEP

($p < 0.001$) in both obese male and female subjects compared to control. $FEV_{1\%}$ was significantly lower in obese female students.

In present study, FVC and FEV1 were negatively correlated with adiposity markers (BMI, WC, WHR, WHtR, Body fat %). WC {FVC ($r = -0.253$, $p < 0.01$), FEV_1 ($r = -0.236$, $p < 0.05$)}, WHtR {FVC($r = -0.357$, $p < 0.001$) and FEV_1 ($r = -0.319$, $p < 0.01$)} were statistically significant with FVC. Significant negative correlation of PEF and MEP was observed with all adiposity markers.

Interpretation & conclusion:

The study concludes that all the respiratory parameters were significantly reduced in obese subjects compared to non-obese subjects. Obesity and pattern of fat distribution have independent effect on pulmonary function

Key Words: Pulmonary functions, obesity.

ABBREVIATIONS

RR	-	Respiratory Rate
PR	-	Pulse Rate
BP	-	Blood Pressure
BMI	-	Body Mass Index
BSA	-	Body Surface Area
SBP	-	Systolic Blood Pressure
DBP	-	Diastolic Blood Pressure
bpm	-	beats per minute
cpm	-	cycles per minute
HR	-	Heart Rate
Ht	-	Height
Wt	-	Weight
FVC	-	Forced Vital Capacity (in ml)
FEV1	-	Forced Expiratory Volume at the end of first second (in ml)
FEV1%	-	Forced Expiratory Volume in one second (in %)
PEFR	-	Peak Expiratory Flow Rate (in L/min)
TV	-	Tidal Volume (in ml)
IRV	-	Inspiratory Reserve Volume (in ml)
ERV	-	Expiratory Reserve Volume (in ml)
IC	-	Inspiratory Capacity (in ml)
MEP	-	Maximum Expiratory Pressure (in mmHg)

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INTRODUCTION

Obesity is defined as “abnormal or excessive fat accumulation that may impair health”. W H O defines obesity as Body Mass Index (BMI) ≥ 30 kg/m².

Obesity has reached epidemic proportion in India in 21st century with morbid obesity affecting more than 5% population of country. Globally in 2005, nearly 400 million were obese .WHO estimates that by 2015, nearly 700 million people will be obese ¹. In India, the prevalence of obesity is 12.6% in women and 9.3% in men i.e. approximately more than 100 million individuals are obese. Industrialization and urbanization have led to decrease in physical activity, substantial change in dietary habit and overall change in the pattern of life style which are known to result in obesity¹⁻³. Numerous epidemiological studies have shown that obesity might increase the risk of cardiovascular diseases, hypertension, diabetes mellitus and cancers as well as other diseases such as arthritis, gout, kidney stone and gall bladder stone.^{1,4}

Weight and Body Mass Index (BMI) measure overall adiposity. They are used as predictors of pulmonary function in many epidemiologic studies. These measures are widely accepted as determinants of pulmonary function. Abdominal adiposity may influence pulmonary function through a mechanism that is distinct from that of overall adiposity. Truncal obesity reduces chest wall compliance, respiratory muscle function and peripheral airway size^{5,6}.

In obese adults, most frequently reported abnormalities are reduction in lung volumes and expiratory flow rates⁷. Pulmonary function tests permit an accurate assessment of functional state of respiratory system and allow quantification of the severity of disease, thereby enabling early detection, assessment of natural history and response to therapy. The pattern of pulmonary function is found to worsen with the degree of obesity moving from a restrictive pattern in mild to moderate obesity with

both FEV1 and FVC reduced and FEV1/FVC ratio being normal to an obstructive pattern in severe and morbid obesity with significant decrease in FEV1 as against FVC and FEV1/FVC ratio being decreased^{8,9}.

According to recent studies, reduction in lung volumes in obese people is not only directly related to the increase in BMI is also related to the distribution of fat which is different in men and women^{1,5,6}. Men are predominantly abdomino - thoracic breathers where as women are predominantly thoraco- abdominal. Y Chen et al reported that the effect of weight gain on pulmonary function is greater in men than in women⁴. Canoy et al reported that BMI is inversely related to respiratory function in both men and women.¹⁰

Considering the high pace of development in Vijayapur and the fact that this is the age group that would form the main work force in near future, a surveillance of pulmonary function tests in youth (in the age group of 18-25 years) studying in a degree college of Vijayapur city has been planned accordingly.

ADIPOCYTE BIOLOGY: ¹¹

Obesity is a state of increased adiposity. A lean adult has about 35 billion adipocytes each containing about 0.4 to 0.6 μ g of triglycerides (TG). An obese adult can have four times more adipocytes each containing 0.8 to 1.2 μ g of TGs. The adipocyte is a cell which is capable of secreting proteins that act as endocrine as well as paracrine cell. It will transmit metabolic signals to proximal and distant tissues and organs which have pathophysiological link between obesity and associated co-morbid conditions. Adipocytes secrete substances like Leptin, Resistin, Estrogen, Adiponectin and Visfatin. There is a direct relationship between plasma Leptin concentration and BMI or body fat percentage. Resistin levels are also increased in obesity. Plasma

concentration of adiponectin is decreased in obesity. Increased level of Visfatin is seen in obesity.

TYPES OF OBESITY:

- I. According to number or size of fat cells
 - II. According to fat accumulation and distribution.
- I. According to number or size of fat cells:** There are three types of obesity.
- A. Hyperplastic obesity: It is associated with an increase in number of fat cells and seen in children.
 - B. Hypertrophic obesity: It is associated with an increase in size of fat cells. It develops later in life associated with metabolic imbalance.
 - C. Hyperplastic - Hypertrophic obesity: It is a type of obesity in which there is an increase in number and size of fat cells.
- II. According to fat accumulation and distribution:** There are two types.
- A. Android obesity: It is male pattern of obesity in which fat deposits primarily in the abdomen and trunk (Apple shape).
 - B. Gynoid obesity: It is female pattern of obesity in which fat accumulates around the hips (Pear Shape).

MEASUREMENT OR ASSESSMENT OF OBESITY:¹²

The most widely used criteria in assessment of obesity are as follows:

1) BODY WEIGHT:

Though not an accurate measure of excess fat, body weight is a widely used index.

Following indicators are included:

- A. Body Mass Index (Quetelet's index) = Weight (kg) / Height (m²)
- B. Ponderal Index = Height (cm) / cube root of body weight (kg)
- C. Broca's Index = Height(cm) – 100 (for ideal weight)
- D. Corpulence Index = actual weight / desirable weight which should not exceed 1.2

The Body Mass Index and Broca's Index are widely used.

Classification of obesity based on BMI

BMI (kg/m²)	Class	Health risk
18.5 - 24.99	normal weight	very low
25 - 29.99	overweight	low
30 - 34.99	moderate obesity	moderate
35 - 39.99	severe obesity	high
> 40	morbid obesity	very high

2) SKIN FOLD THICKNESS:

Skin fold thickness is measured by several varieties of calipers. It is measured usually at four sites – mid triceps, biceps, subscapular and suprailiac regions. The main drawback of skin fold measurement is its poor repeatability and in extreme obesity, measurement may be impossible.

3) WAIST CIRCUMFERENCE AND WAIST : HIP RATIO

It is believed to be an approximate index of intra abdominal fat mass and total body fat. WHR >1 in men and > 0.85 in women indicate abdominal fat accumulation i.e. obesity.

CAUSES OF OBESITY: ¹¹

- 1) **INHERITED CAUSES:** Prader – Willi syndrome, Leptin deficiency, Leptin receptor mutations, Pro opiomelanocotin defects, Melanocortin – 4 receptor defects, Pro – hormone convertase 1 deficiency.
- 2) **ENVIRONMENTAL CAUSES:** High energy diet, Physical inactivity.
- 3) **OTHER CAUSES:** Endocrine disorders – Hypothyroidism, Cushing’s syndrome, Polycystic ovarian disease, Hypothalamic obesity, Hypogonadism. Drugs- Anticonvulsants, Antidepressants, Antipsychotics, β blockers, Corticosteroids, Insulin, Sex steroids, Oral hypoglycemic agents.

PATHOGENESIS: ¹¹

The increasing incidence of obesity is a major health issue faced by society. It is established that key components of central control of energy balance are located in Hypothalamus. In 21st century, it is taken for granted that hypothalamus is required for co-ordinate control of food intake and energy homeostasis. For some time, intimate interaction of hypothalamus and pituitary gland has been appreciated.

Recent studies showed the primary role of hypothalamus in controlling long term energy stores and thus adipose mass. Aschner demonstrated in dogs that mere removal of pituitary gland without damage to overlying hypothalamus did not result in obesity. The most definitive evidence of vital role of hypothalamus was provided by Hetherington and Ranson. They demonstrated that destruction of medial base of hypothalamus without damage to pituitary gland could result in morbid obesity. These studies established strongly that intact hypothalamus is required for normal energy homeostasis. Following these discoveries that hypothalamic lesions could cause obesity, it also became apparent that lesions in other regions of hypothalamus such as lateral hypothalamus could cause leanness thus suggesting that Feeding center is

located in the Lateral Hypothalamus (LH) and Satiety center in the Ventro Medial Hypothalamus (VMH). Regulation of food intake is an intricate phenomenon because of complex nature of control of eating behavior. The long term regulation of food intake is aimed at maintaining a balance between intake and expenditure of energy so that body weight is maintained within its normal range. Three major categories of factors are involved in regulating food intake in turn obesity. They are neural, hormonal and metabolic factors.^{11,12}

NEURAL FACTORS IN REGULATION OF FOOD INTAKE:

Hypothalamus is the major regulator of food intake. Feeding center is situated in lateral hypothalamus where as satiety center in Ventro Medial Hypothalamus. Hypothalamus receives sensory inputs from the external as well as internal environment. In addition, several hormones are known to play an important role in regulating food intake and metabolism. They directly act on neurons in hypothalamus. The hypothalamus integrates all these information and in turn provides motor output to regulatory sites such as anterior and posterior pituitary, cerebral cortex, pre motor and motor neurons in brainstem and spinal cord and autonomic preganglionic neurons. The patterned hypothalamic outputs of these effector sites ultimately result in coordinate endocrine and autonomic responses in energy balance.

A balanced interaction between two sets of neurons occurs within the arcuate nucleus which forms the medial group of nuclei in hypothalamus. They include:¹¹

1. **Neuropeptide Y (NPY) / Agouti Related Protein (AGRP)** – Its activation promotes food intake.
2. **Pro – opio Melanocortin (POMC) / Cocaine and Amphetamine Related Transcript (CART)** – Its activation inhibits food intake. Signals from these neurons are sent to other brain nuclei which ultimately result in alterations in

food intake and energy expenditure. In addition to inputs to hypothalamus, brain also receives signals from visceral organs including gut. This is known as Dorsal Vagal Complex which comprises of Nucleus Tractus Solitarius (NTS), Dorsal motor nucleus of vagus and area postrema. Sensory afferent signals carried by the glossopharyngeal and vagus nerves include taste, gastric stretch, levels of glucose and lipids in liver and portal vein. Nerve terminals carrying this information innervate NTS which in turn sends signals to dorsal motor nucleus of vagus that in turn innervates whole gut including pancreas. Extra hypothalamic structures in brain to control food intake include mesolimbic areas of amygdala, caudate nucleus, nucleus accumbens and septal nuclei.

The efferent controls include motor activities involved in seeking food as well as receptors innervated by the adrenergic nervous system and the hormonal system. A complex sequence of motor activities leads to the initiation of food seeking and identification of food. This system is integrated in lateral hypothalamus. Both sympathetic and parasympathetic nervous systems are involved in the development of obesity. But, reduction in sympathetic activity is producing noticeable obesity.

HORMONAL FACTORS IN REGULATION OF FOOD INTAKE: ¹²

- A.** Hormones that increase food intake (Orexigenic hormones): Neuropeptide Y, Orexin, Ghrelin, MCH (Melanin Concentrating Hormone), AGRP (Agouti Related Protein), Galanin and GHRH
- B.** Hormones that decrease food intake (Anorexigenic hormones): Leptin, Peptide YY, Estrogen, Dopamine, – MSH, CART, CCK and CRH

LEPTIN: It is secreted by adipose tissue and serves as a key hormone in regulation of food intake. Arcuate nucleus is the sensor to detect plasma Leptin concentration. Leptin directly depolarizes / activates POMC neurons and hyperpolarizes / inhibits AGRP / NPY neurons. High Leptin level occurs in obesity that decreases food intake and increases energy expenditure as well as sympathetic activity. Hypothalamus senses adiposity via Leptin. Accordingly, it controls food intake and energy expenditure of the body. Leptin also stimulates thyroid function, controls glucocorticoid secretion and increases activity of uncoupling protein in the adipose tissue cell. Thereby, it increases peripheral energy expenditure.

NEUROPEPTIDE Y (NPY): A polypeptide containing 36 amino acids which is a strong orexigen. NPY containing neurons are present abundantly in arcuate nucleus. It stimulates food intake by stimulating the feeding center. It also inhibits various orexigenic agents on hypothalamus.

GHRELIN: It is a polypeptide secreted from stomach and hypothalamus. It is considered to be a potent orexigen. It directly depolarizes AGRP/NPY neurons. It stimulates food intake and body weight gain. Fasting Ghrelin levels have shown to be inversely proportional to bodyweight.

ESTROGEN: It is a potent an orexigenic agent. It crosses blood brain barrier to act on hypothalamic centers. It inhibits feeding center and decreases release of NPY. In humans, obesity develops following menopause due to estrogen withdrawal.

CHOLECYSTOKININ: It is produced by GIT in response to food ingestion. Satiety effect of CCK is mediated via CCK – A receptors on afferent vagal nerves.

Other substances involved in food regulation are:

- a) **PYY:** It is released postprandially in ileum and colon activates POMC neurons inducing taste aversion and nausea.

- b) **GLP-1:** It has direct effect on satiety via inhibition of gastric emptying and induction of taste aversion.
- c) **Amylin:** It has an anorexic activity.
- d) **Glucocorticoids:** They stimulate food intake and weight gain.
- e) **Melanocortin 4 receptors:** They also regulate energy and glucose homeostasis by decreasing adipose mass, food intake and increasing energy expenditure.

The above said substances form the hormonal regulators of the Brain – Gut – Adipose axis and are also called Adipostatic factors.¹¹

Certain neurotransmitters are also involved in the transmission of information that regulates food intake and nutrient stores. They are as follows:

- a) Norepinephrine in ventromedial hypothalamus can increase fat stores by activating α -2 adrenergic receptors.
- b) Serotonin can decrease food intake.
- c) Tryptophan and 5- hydroxytryptophan can decrease food intake.
- d) β endorphin , dynorphin , galanin and GHRH can stimulate food intake.

METABOLIC FACTORS IN REGULATION OF FOOD INTAKE:

- 1) **PLASMA GLUCOSE:** It is an important metabolic factor in the regulation of feeding. Discharge of Ventro Medial hypothalamus i.e. satiety center partly depends on its glucose utilization. Increased plasma glucose concentration will in turn increase the activity of neurons of satiety center.
- 2) **MALONYL Co A:** It is produced from acetyl Co A. It will inhibit food intake by inhibiting hypothalamic NPY synthesis.
- 3) **AMINOACIDS AND FATTY ACIDS:** They give chemical signals to satiety center.

- 4) **BODY TEMPERATURE:** Decrease in body temperature increases food intake. Along with all the factors responsible for the regulation of food intake and body weight, a coordinated energy homeostasis includes a balance between energy intake and energy expenditure. The components of daily total energy expenditure (TEE) are: a) Resting Energy Expenditure (REE) which is approximately 70% of TEE , b) Energy expenditure during physical activity which accounts for 20% of TEE and c) Thermic Effect of Food (TEF) which accounts for 10% of TEE. The process of energy homeostasis is under the control of sympathetic nervous system and thyroid axis. Energy expenditure is increased by stimulation of β adrenergic receptor and elevation of thyroid hormone.

COMPLICATIONS OF OBESITY:¹¹

Obesity causes many serious complications that impair quality of life and lead to increased morbidity and premature death.

1. Endocrine and metabolic diseases: Metabolic or Insulin Resistance syndrome, Type2 Diabetes Mellitus and Dyslipidemia.
2. CVS diseases: Hypertension, Coronary Heart Disease and thromboembolic diseases.
3. Cerebrovascular diseases
4. Musculoskeletal diseases: Gout, Osteoarthritis
5. Cancers of stomach, prostate, breast, uterus, cervix and ovary
6. Genitourinary diseases in women
7. Neurologic diseases: Idiopathic intracranial hypertension
8. Cataract

9. Gastrointestinal diseases: Gastroesophageal reflux diseases, Gall stones, Pancreatitis and Liver diseases.

10. Pulmonary diseases: Restrictive lung diseases, Obesity hypoventilation syndrome and Obstructive sleep apnoea

LUNG/RESPIRATORY COMPLICATIONS OF OBESITY: ¹

Changes in lung function parameters in obesity:

Gas exchange: Increased PaCO₂, Reduced PaO₂ and Reduced DLco.

Respiratory mechanics: Reduced respiratory compliance and Increased airway resistance.

Respiratory muscle strength: Reduced maximal expiratory and inspiratory pressures.

Work of breathing: Increased work of breathing and Increased oxygen cost of breathing.

Breathing pattern: Increased Respiratory Rate.

Lung volumes: Reduced TV, FVC, ERV, FRC and TLC, Increased RV.

PULMONARY FUNCTION TESTS

The pulmonary function tests provide an assessment of respiratory function of an individual. The pulmonary function test is an age old but time tested parameter for assessing respiratory health of a person. With increased urbanization, increased population and indiscriminate industrialization, respiratory health of population is being affected.^{13 - 15}

There are various pulmonary function tests. These tests provide quantitative and objective assessment of respiratory function. They do not give a specific etiological or pathological diagnosis ^{16, 17}. The tests can be divisible into various categories. They are as follows: ¹⁸

A. Tests to assess ventilatory function of lungs-

1. Measurements of various lung volumes and capacities
2. Measurements of dead space
3. Measurements of lung compliance
4. Measurements of airway resistance

B. Tests for diffusion

C. Tests of ultimate purpose of respiration

D. Tests during exercise

VOLUMES AND CAPACITIES. : Volumes are basic entities while Capacity is derived from Volumes. Each Capacity is the sum of two or more Volumes.

LUNG VOLUMES:

- A. **Tidal Volume (TV):** It is the volume of air that is inspired or expired during normal quiet breathing. Normal value: 350 - 500ml.
- B. **Inspiratory Reserve Volume (IRV):** It is the maximum volume of air which can be inspired after normal tidal inspiration. Normal value: 2000 - 3200ml.
- C. **Expiratory Reserve Volume (ERV):** It is the volume of air which can be exhaled after normal tidal expiration. Normal value: 750 - 1000ml.
- D. **Residual Volume (RV):** It is the volume of air that is remaining in the lungs at the end of maximum exhalation. Normal value: 1000 - 1200ml.

LUNG CAPACITIES:

- A. **Inspiratory Capacity (IC):** It is the maximum volume of air which can be inspired after tidal expiration. Normal value: 2500 - 3700ml.

$$IC = TV + IRV$$

- B. **Expiratory Capacity (EC):** It is the maximum volume of air which can be exhaled after tidal inspiration. Normal value: 1250 - 1500ml.

$$EC = TV + ERV$$

C. **Functional Residual Capacity (FRC):** It is the volume of air that is remaining in the lungs at resting expiratory level. It is about 2200 - 2500ml.

$$FRC = ERV + RV.$$

D. **Vital Capacity (VC):** It is the maximum volume of air which can be exhaled from lungs by forceful effort followed by a maximal inspiration. Normal value: 4.6 - 4.9L in males and 3 - 3.3L in females.

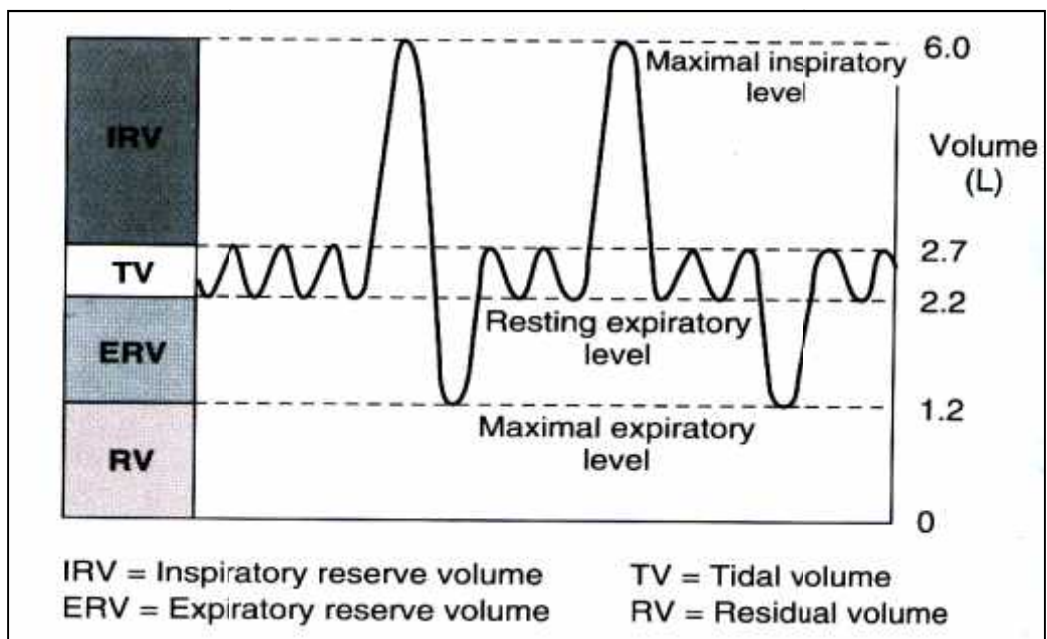
$$VC = TV + IRV + ERV$$

E. **Total Lung Capacity (TLC):** It is the amount air that can be present in the lungs at the end of maximum inspiration. Normal value: 5600 - 5800ml.

$$TLC = VC + RV.$$

Majority of lung volumes and capacities can be measured by Spirometry. In the present study, they are recorded by computerized spirometer with the exception of Residual Volume, Functional Residual Capacity and Total Lung Capacity.

FIGURE 1: Spirogram



DYNAMIC LUNG FUNCTION TESTS:

1. Forced Vital Capacity(FVC) :

This is the maximum volume of air which can be breathed out as forcefully and as rapidly as possible following a maximum inspiration. Thus, Forced Vital Capacity is exactly similar to Vital Capacity except that there is a special stress on rapid forceful and complete exhalation.

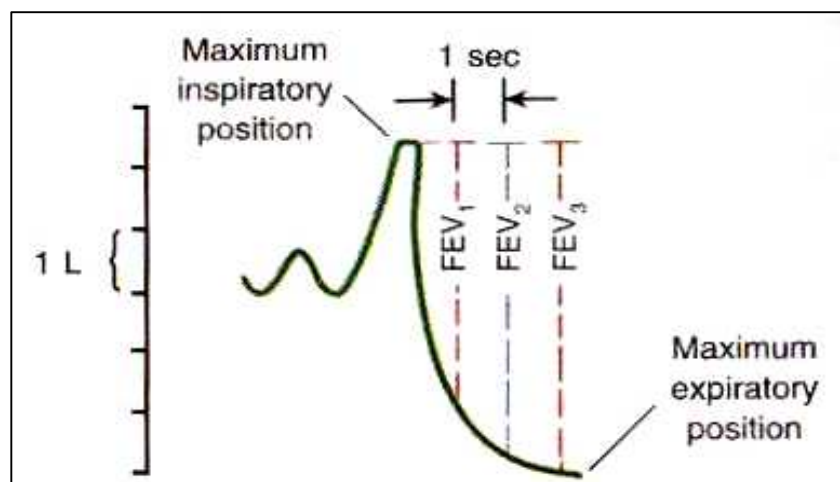
2. Forced Expiratory Volume or Timed Vital Capacity (FEV or TVC):

If the vital capacity is recorded on a kymograph (Spirograph) at a known speed, volume of air exhaled can be timed. This is TVC.

Components of TVC:

1. FEV₁: Forced Expiratory Volume at the end of 1st second i.e., volume of FVC exhaled in first second of exhalation. Normally 80% of FVC.
2. FEV₂: Forced Expiratory Volume at the end of 2nd second i.e., volume of FVC exhaled in first 2 seconds of exhalation. Normally 95% of FVC.
3. FEV₃: Forced Expiratory Volume at the end of 3rd second i.e., volume of FVC exhaled in first 3 seconds of exhalation. Normally 98-100% of FVC.

FIGURE 2: Record of Timed Vital Capacity



$$\text{FEV1\%} = \frac{\text{Volume of air exhaled in the first second}}{\text{Vital Capacity}} \times 100$$

4. FEV1 / FVC ratio (FEV1%):

This ratio in healthy adults should be approximately 75-80%. FEV₁% is more sensitive indicator of airway obstruction than FVC or FEV₁ alone. FEV₁ / FVC decreases in obstructive lung diseases. But, in the early phase of obstruction which originates in the small airways, this ratio may be normal.

5. Peak Expiratory Flow Rate (PEFR):

This is the expiratory flow rate during the peak of FVC. It is recorded with a mini Wright's Peak Flow Meter. PEFR measures efficiency of lungs by recording maximum flow of air. Peak Expiratory Flow Rate is dependent upon age, sex, build, etc. Normal value : 400 - 450 liters per minute. In a young adult, it is about 400L / min. It falls drastically in diseases such as COPD.

6. Maximum Expiratory Pressure (MEP):

Various respiratory symptoms are associated with respiratory muscle dysfunction. There are reports of progressive weakness of respiratory muscles in patients with multicore myopathy, multiple sclerosis, Motor Neuron disorder, Malnutrition and Congestive Heart Failure. Measurement of strength of respiratory muscles is useful in detection of weakness of respiratory muscles and to quantify its severity. In patients with severe weakness of respiratory muscles, Vital Capacity is reduced. But it is a non specific and relatively insensitive measurement. Conventionally, strength of respiratory muscles is evaluated by determining Maximum Expiratory Pressure (MEP).

However, studies showed that Maximal Expiratory Pressure alone can be used as a measuring tool for strength of respiratory muscles. MEP is useful in determining the ability of a person to cough effectively. It is reflecting the strength of the abdominal muscles and other expiratory muscles. It is measured by using a modified Black's apparatus.

Pictures of Mini Wright's Peak flow meter

Modified Black's apparatus



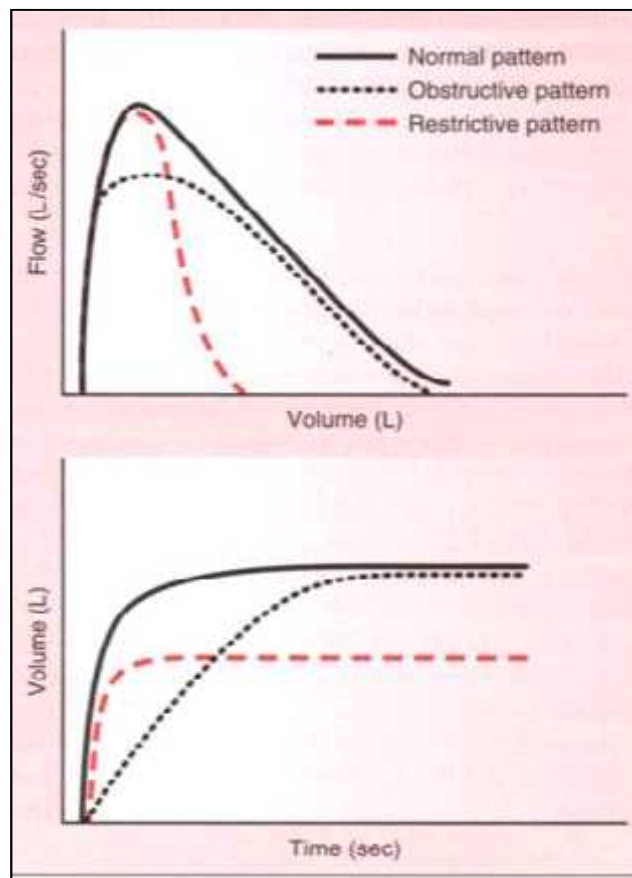
Spirometry:

Spirometry measures ventilation, the movement of air into and out of the lungs. The Spirogram will identify two different types of abnormal ventilatory patterns: obstructive and restrictive¹⁹.

Common causes of obstructive pattern are: cystic fibrosis, asthma, bronchiectasis, bronchitis, and emphysema. Chronic bronchitis, emphysema and asthma result in dyspnea (difficulty breathing) and deficient ventilation. They are regarded as Chronic Obstructive Pulmonary Diseases (COPD). COPD is the fourth leading cause of death among Americans¹⁹.

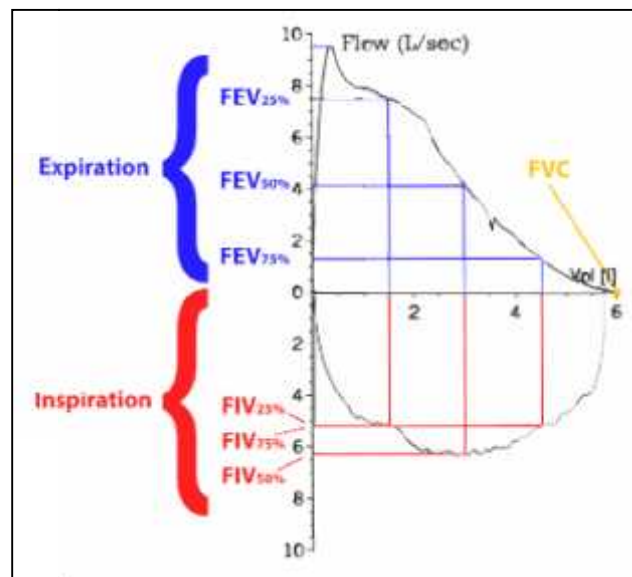
Common causes of restrictive pattern are: pregnancy, pneumonia, heart disease, fibrosis of lung, pneumothorax (collapsed lung) and pleural effusion (compression caused by chest fluid)¹⁹.

FIGURE 3: Spirograms showing obstructive & restrictive patterns.



Obstructive and restrictive patterns can be identified on spirogram using both "y" and "x" axes. Volume (liters) is plotted on the y-axis versus Time (seconds) on the x-axis. A restrictive pattern is characterized by a normal shape showing reduced values for all parameters. The reduction in volumes indicates the severity of the disease. An obstructive pattern produces a spirogram with an abnormal shape. Inspiratory volume is reduced. The volume of air expelled is normal but the air flow rate is slower causing an elongated tail to the FVC.

FIGURE 4: Flow-Volume Spirogram



A Flow-Volume loop spirogram is another way of displaying spirometric measurements. It requires FVC manoeuvre followed by a Forced Inspiratory Volume (FIV). Flow rate in liters per second is plotted on the y-axis and volume (liters) is plotted on the x-axis. The expiratory phase is shown on top and the inspiratory phase on the bottom.

The flow-volume loop spirogram is helpful in diagnosing upper airway obstruction and can differentiate some types of restrictive pattern¹⁹.

Spirometry is used to assess lung function over time and often to evaluate the efficacy of broncho dilating inhalers such as albuterol. Spirometry is contraindicated in patients whose condition will be aggravated by forced breathing such as²³: nausea or vomiting, hemoptysis, pneumothorax, recent heart attack, unstable angina, aneurysm (cranial, thoracic or abdominal), thrombotic condition (such as clotting within a blood vessel), recent thoracic or abdominal surgery.

The test should be terminated if the patient shows signs of significant head, chest or abdominal pain while the procedure is in progress.

Spirometry is dependent upon the patient's full compliance with breathing instructions, specially his or her willingness to extend a maximal effort at forced breathing. Therefore, the patient's emotional state must be considered¹⁹.

Table 1: Assessment of restrictive and obstructive ventilatory defects²⁰

Parameter	Obstructive lung disease	Restrictive lung disease
TLC	High	Decreased
FEF _{25-75%}	Low	Normal
VC	Normal / Increased	Decreased
FEV ₁	Decreased	Normal
FEV ₁ /FVC	Decreased	Normal
MVV	Decreased	Normal
RV	Increased	Decreased

AIMS AND OBJECTIVES OF STUDY

To compare pulmonary function tests between obese and non obese students in the age group of 18-25 years and to correlate them with degree of obesity.

REVIEW OF LITERATURE

Hippocrates (460-377 B.C) and Galen believed that breathing was for cooling the heart. Galen (130-211 A D) gave an idea that respiratory act was dependent upon the diaphragmatic contraction and wall movement.

Robert Boyle (1627-91) noted that air contained vital constituents required for life. In 1680, G. A. Borllely for the first time measured the Inspiratory Volume. He also mentioned Residual Volume (as quoted by Fenn 1976).

J. Black (1728-1799) discovered CO₂. In 1800, Sir Humphery Davy measured the lung volumes by using Hydrogen. Functional Residual Capacity was measured by N. Grehent in 1880. In 1846, John Hutchinson²¹ invented spirometer. He measured Vital Capacity. He believed it to be a powerful indicator of longevity. It is water spirometer which is still used with some modifications. In less than 10 years after Hutchinson, Wintrich developed a spirometer. It was easier to use. He stated that three parameters that determine Vital Capacity are body weight, height and age²². In 1859, E. Smith developed a portable spirometer. In 1866, Salter added a kymograph to the spirometer in order to record time while obtaining air volumes. In 1902, TG Brodie was the first to use a dry bellow wedge spirometer which is the precursor of Fleisch Spirometer used today. In 1904, Tissot introduced closed circuit spirometer²³.

Hermansen in 1933 recorded for the first time Maximum Breathing Capacity²¹. In 1950, the unanimously agreed nomenclature was given by United State Respiratory Physiologists Committee. In 1951, Tiffeneau , Pinelli and Gaensler developed the technique and procedure for measuring timed volumes. The procedure was referred to “Forced Vital Capacity ” which quantifies the air volume dynamics and shows the rates of air flow along the respiratory tree and useful for obtaining pulmonary function tests (Fenn 1965)²⁴.

Thus, the history of spirometry began in the 2nd century with simple measurement of ventilatory volumes. It progressed through time into more complex measurement of lung function using variety of techniques.

INDICATIONS OF SPIROMETRY: ^{25, 26}

- To evaluate potential hazard of occupation
- To identify smokers at risk of developing severe chronic airflow obstruction
- To evaluate fitness for specific surgical procedures
- To assist in monitoring the course of respiratory problems, response to therapy or change with growth and aging
- To quantify lung function impairment before and after government appointment
- To evaluate progression of a disease
- To evaluate lung functions for research purpose.

After paying tribute to these great scientists, we will now move on to review of literature proper.

In 2005, pulmonary function tests were studied in 373 obese individuals aged >18 years. The subjects selected for this study were accredited by the Alberta College of Physicians and Surgeons. Obese individuals showed significant reduction in FRC and ERV. The values showed an exponential decrease with increase in BMI.²⁷

In 2008, 80 healthy volunteers from both sexes in the age group of 20–40 years randomly selected from the employees of Himalayan Institute of Medical Sciences and community dwellers from the surrounding area of Bhaniyawala, Dehradun. The result indicated significantly lower values of FVC and FEV₁ in obese female individuals. The FVC and FEV₁ in female individuals were negatively

correlated with BMI. The values of Dynamic pulmonary function tests in male individuals showed negative correlation with BMI²⁸.

Another study was conducted in Merdional Hospital, Brazil in 2008. It comprised of 20 sedentary women who were non-smokers aged 20 to 35 years. A significant reduction in IRV, ERV, FEV₁ / FVC, MVV and % MVV was observed.²⁹

A study was conducted in the year 2009 Yeungnam University Hospital health promotion centre, Korea. It comprised of 152 obese male and 139 obese female individuals in the age group of 20-70years. FVC and FEV₁ were reduced in obese male and female individuals. They also noted that WHR and Body fat % were significantly negatively correlated with FVC in obese males and non significant with FEV₁ in obese females.³⁰

In 2010, a study was conducted on healthy young male volunteers from University population and nearby area of Bhaniyawala, Dehradun, Uttarakhand. 100 obese volunteers in the age group of 20 to 40 years were selected. Values of PEFR were reduced in obese individuals.³¹

Another study was conducted in general population of Amritsar in the year 2011. 100 obese and another 100 non obese female subjects were included in the study. It was observed that values of FEV₁/FVC, MVV and FVC were reduced in obese individuals.³²

A study was conducted in 2011 in Yenepoya Medical College, Mangalore. The study comprised of 35 obese & 51 non obese individuals. Reduced % FEV₁/FVC and ERV were observed in obese as compared to non obese individuals.³³

In 2012, a study was conducted in general population of Amritsar. 100 obese and another 100 non obese male individuals were included in the study. The values of

FVC, FEV₁ and MVV were shown to be decreased in obese as compared to non obese individuals.³⁴

In 2012, study was conducted by Department of Physiology, J.J.M. Medical College, Davangere. 60 obese and another 60 non-obese young individuals in the age group of 18-25 years were selected randomly from the general population of Davangere city. It was observed that values of FVC, FEV₁, FEV₂, FEV₃ and PEFV were reduced in obese as compared to non obese subjects.³⁵

A study was conducted in Mangalore in 2012. 40 obese males and 40 non obese males in the age group of 30-40 years participated in the study. It was observed that reduced FVC, FEV₁, FEV₁ / FVC, among them FEV₁ / FVC was significantly reduced in obese individuals³⁶.

In 2012, a study was conducted in Konaseema Institute of Medical Sciences, Amalapuram, Andhra Pradesh by Department of Physiology. 60 overweight as well as 60 normal weight male and female individuals in the age group of 16- 20 years included in the study. They observed significant reduction of FVC and FEV₁ in overweight male and female groups compared to normal weight. They also noted significant negative correlation of BMI, Body fat % with FVC, FEV₁ in overweight males and non significant in females.³⁷

A study was conducted in Amritsar in the year 2013. 200 obese male and 200 obese female individuals in the age group of 30-50 years participated in the study. It was observed that BMI had high significant correlation with systolic blood pressure whereas Waist to Height Ratio was significantly correlated with diastolic blood pressure in obese individuals.³⁸

In 2013, a study was conducted on 85 male and 65 female obese subjects in Department of Physiology, M.K.C.G Medical College, Brahmapur, Odisha. It was

observed that FVC, FEV₁, and FEV_{1%} were significantly reduced in both obese male and female subjects. They also observed that FVC and FEV_{1%} were having significant negative correlation with W / H and BMI in obese male subjects.³⁹

A study was conducted in Bangalore in the year 2014. 50 male subjects (50 in each of normal, overweight and obese categories of BMI) in the age group of 25-50 years, who were apparently healthy. It was observed that obese and overweight groups significantly differed from the normal group with their mean BMI. Among the Pulmonary function parameters recorded, FVC, FEV₁ and FEV_{1%} showed a significant decrease in the obese group compared with normal.⁴⁰

These reviews reveal that there is a definitive relationship between Body fat mass and pulmonary function tests in adults. However, a very few reports are available involving subjects from North Karnataka in particular Vijaypur. Hence, the present study was undertaken.

MATERIALS AND METHODS

Source of Data

The study was conducted on student volunteers of BLDEA's KCP Science College, Vijaypur City of North Karnataka.

Method of collection of Data

Study group: Group consisted of 50 obese student volunteers with BMI ≥ 30 kg/m² from BLDEA's KCP Science College, Vijaypur.

Control group: Group consisted of age matched 50 non obese student volunteers with BMI 18.5 - 24.99 kg/m² from BLDEA's KCP Science College, Vijaypur.

Duration of study: From December 2013 to April 2015

Age of subjects: 18 to 25 years in both groups.

Sample size: According to Shashi Mahajan et al, mean \pm SD of MVV in non obese male and female subjects were 111.19 ± 24.55 and 85.25 ± 10.82 respectively and obese male and female subjects were 92.04 ± 27.13 and 78.75 ± 12.98 .³⁴

Considering the average mean \pm SD of MVV in obese male and female individuals was 85.39 ± 20.45 and average mean \pm SD of MVV in non obese male and female individuals was 98.22 ± 17.68 and at 95% confidence interval with 90% power, the calculated sample size was 46 in each arm using the following statistical formula,

$$n = \frac{(Z_1 + Z_2)^2 \times 2 \times S^2}{d^2}$$

Z₁ was Z value for 95%.

Z₂ was Z value for 90%.

S was common standard deviation between two groups.

d was difference between mean values.

Hence, in each group 46 50 participants (students) were included.

Sampling technique: Subjects were selected using convenience sampling method.

Inclusion criteria:

- 1) Obese male and female individuals aged 18-25 years with BMI ≥ 30 kg/m² (study group)
- 2) Non obese male and female individuals aged 18-25 years with BMI 18.5-24.99 kg/m² (control group)

Exclusion criteria:

The following subjects were excluded from the study:

- Pre existing cardiovascular/ respiratory/ endocrine abnormalities
- Overweight
- History of smoking, alcohol
- Subjects with noticeable weight gain or weight loss over preceding 3 months
- Drugs causing weight gain or weight loss.
- Ribcage deformities like kyphosis and scoliosis
- Pregnancy

The following Parameters were recorded in the subjects:

I. Record of Physical Anthropometry:

1. **HEIGHT (cm):** This was measured in each subject standing without her / his footwear by Stadiometer.
2. **WEIGHT (kg):** Each subject was weighed in standard weighing machine with minimum clothing nearest 0.1 kg.
3. **BODY SURFACE AREA (square meters):** This was calculated by using Dubois nomogram.

4. **BODY MASS INDEX** (kilogram/m²): This was calculated for each subject from his / her height (meter) and weight (kg).
5. **WAIST CIRCUMFERENCE** (cm): It was measured at midpoint of outer border of 9th costal cartilage and highest point on iliac crest (approximate to the level of umbilicus) with minimum clothing with help of standard tailor tape.
6. **HIP CIRCUMFERENCE** (cm): It was measured at the largest posterior extension of the buttocks.
7. **SKIN FOLD THICKNESS** (mm): It was measured in subscapular, biceps, triceps and suprailiac areas using Harpenden calipers. Body fat percentage was calculated by using according to Durnin and Womersley (1974) formula.
8. **WAIST: HIP CIRCUMFERENCE RATIO** – WC / HC.
9. **WAIST: HEIGHT RATIO** – WC / Ht.

II. Record of Physiological Parameters ⁴¹⁻⁴⁴.

1. **PULSE RATE (PR)**: It was expressed as beats per minute by palpating right radial artery.
2. **BLOOD PRESSURE**: SBP and DBP were measured by mercurial sphygmomanometer in mm of Hg. Mean Arterial Pressure (MAP) was calculated in mm of Hg by using following formula $DBP \pm 1/3$ pulse pressure (PP).
3. **RESPIRATORY RATE (RR)**: It was expressed as cycles per minute by manual method.

III. Record of Pulmonary function Parameters⁴⁵⁻⁴⁷

The following pulmonary function parameters were recorded using Spiropac (MEDICAID) in each subject.

1. Forced Vital Capacity (FVC in L).
2. Forced Expiratory Volume at the end of first second (FEV₁ in L)
3. Percentage of Forced Expiratory Volume in one second (FEV_{1%}). FEV_{1%} was calculated mathematically using following formula: $FEV_{1\%} = FEV_1 / FVC \times 100$.
4. Peak Expiratory Flow Rate (PEFR in L/min) by using mini Wright's peak flow meter.
5. Maximum Expiratory Pressure (MEP in mmHg) by using Modified Black's apparatus.

Spirometry is the most widely used method to record pulmonary function tests. It records the amount of air breathed in and out and the rate at which this process takes place. The device used in this test is a Spirometer, a long piece of tubing with a mouth piece at one end and a recording device at the other. Spirometry reveals degree of obstruction and restriction of the airway.

Picture of Spiroexel instrument



Procedure: Spirometry requires nose to be pinched off as the subject breathes through a mouthpiece attached to the spirometer. The subject was instructed how to breathe during the procedure. Three breathing manoeuvres were practiced before recording the values. The highest of three trials was used for evaluation. This procedure measures air flow by electronic or mechanical displacement principles. It uses a microprocessor and recorder to calculate and plot air flow¹⁹.

Purpose: Spirometry is the most commonly done procedure to assess pulmonary functions. It can be performed at the bedside, in a physician's office or in a pulmonary laboratory. It is often the first test performed when a problem with lung function is suspected. Spirometry may also be suggested by an abnormal X- ray, arterial blood gas analysis or other diagnostic pulmonary test result. The National Lung Health Education Program recommends that regular spirometric tests are to be performed in people more than 45 years of age who have a history of smoking. Spirometric tests are also recommended for old people and persons with a family history of lung diseases and chronic respiratory ailments.

Precautions: The subject should inform the physician of any medications he or she is taking or of any medical conditions with which they are suffering. These factors may affect the validity of the test. The subject's smoking habits and history should be thoroughly documented. The subject must be able to understand and respond to instructions for the breathing manoeuvre. Therefore, the test may not be appropriate for very young, unresponsive or physically impaired people¹⁹.

Preparation: The subject's age and gender were recorded. Height and weight of each subject were measured before the procedure. The subject should not have eaten heavily within three hours of the test. He or she should be instructed to wear loose-fitting clothing over the chest and abdomen area. Breathing maneuver was explained

and demonstrated to the patient. The subject should practice breathing into the mouthpiece until he or she was able to duplicate the manoeuvre successfully on two consecutive attempts¹⁹.

Picture showing recording of Pulmonary Function Test in Male individual

(Control group)



Picture showing recording of Pulmonary Function Test in Female individual

(Control group)



Picture showing recording of Pulmonary Function Test in Male individual

(Study group)



Picture showing recording of Pulmonary Function Test in Female individual

(Study group)



Figure 5: Graph showing the recording of Pulmonary Function Test using Spiroexcel instrument in Male individual (Control group)

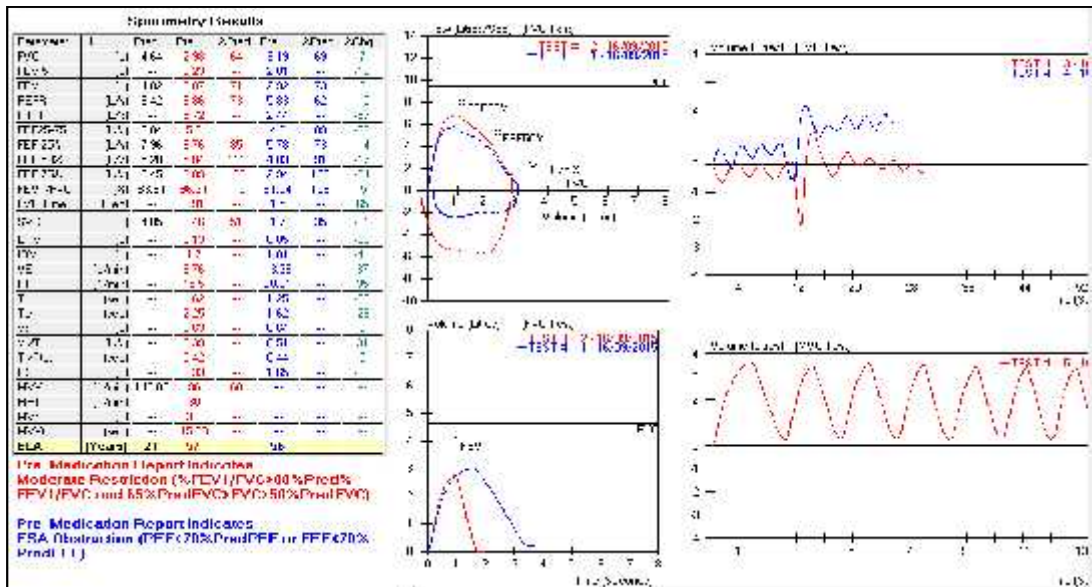


Figure 6: Graph showing the recording of Pulmonary Function Test using Spiroexcel instrument in Female individual (Control group)

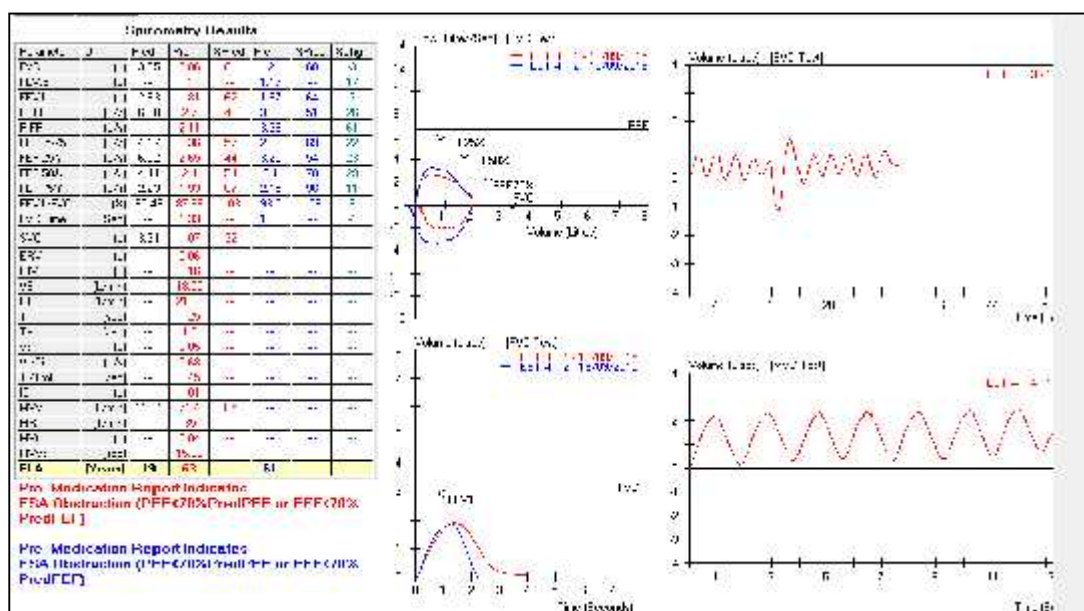


Figure 7: Graph showing the recording of Pulmonary Function Test using Spiroexcel instrument in Male individual (Study group)

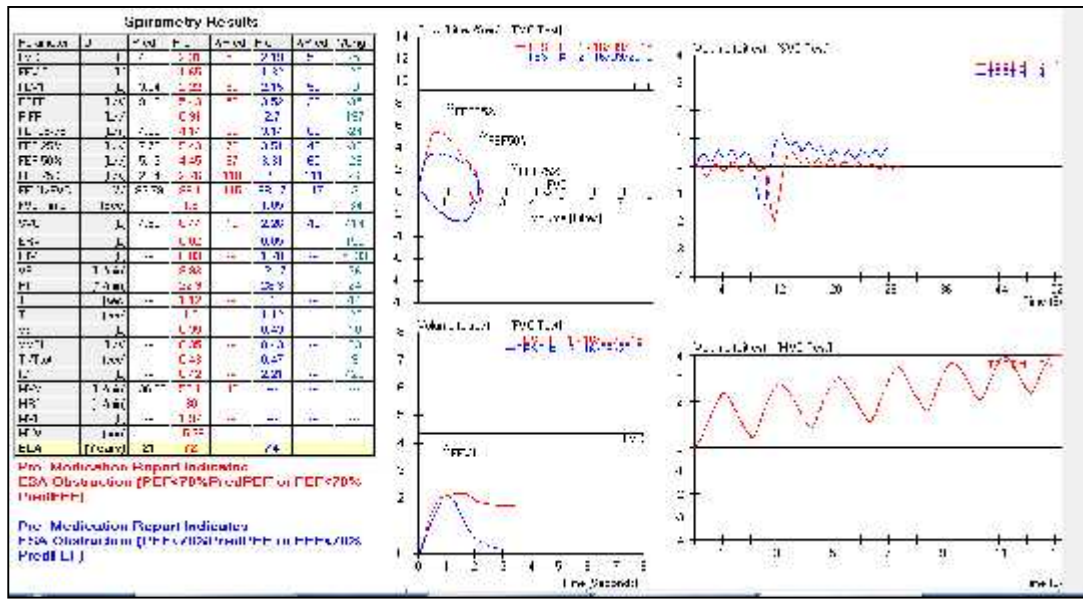
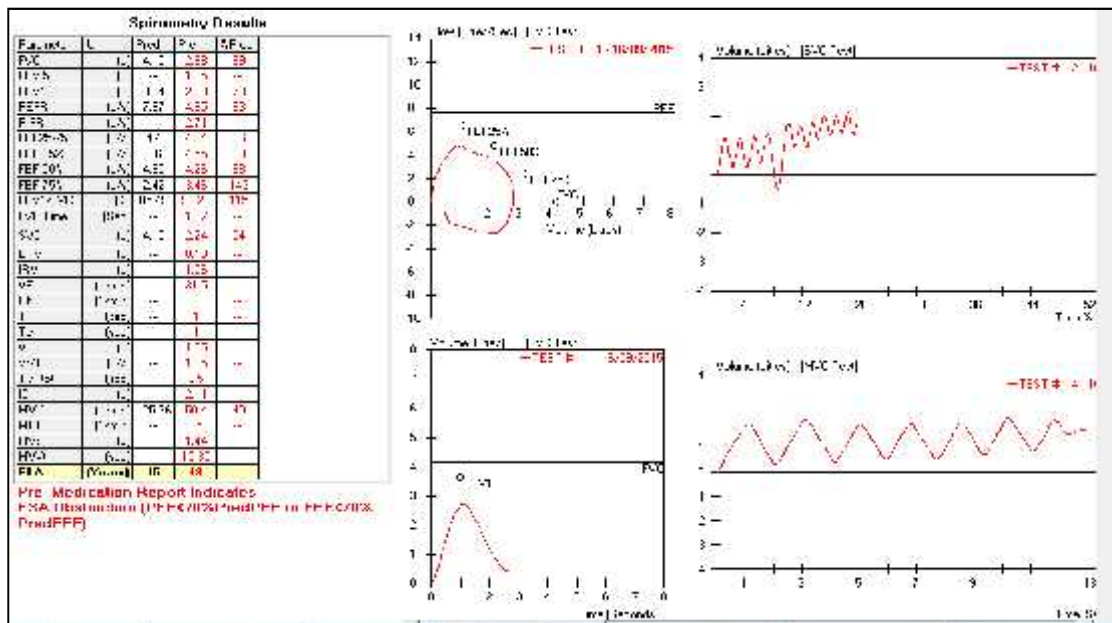


Figure 8: Graph showing the recording of Pulmonary Function Test using Spiroexcel instrument in Female individual (Study group)



STATISTICAL ANALYSIS: ^{48, 49}

The data obtained was analyzed in consultation with statistician.

Statistical measures used were

1. Mean, Standard deviation
2. Student's t test
3. Correlation

Pearson's correlation analysis was used to investigate the relationship between PFTs and the degree of obesity of study group.

RESULTS

The Anthropometric parameters recorded included Age (years), Height (centimeters), Weight (kilograms), Body Mass Index (kilograms / meters) and Body fat %. Total number of subjects in control group was 50. Total number of subjects in study group was 50.

I. Anthropometric Parameters

The mean and standard deviation, level of significance of each parameter were calculated and presented in table numbers: 2-5.

Table 2: Anthropometric Parameters of Control group Vs Study group (Values were Mean \pm SD).

Parameter	Control group(n=50)	Study group(n=50)	p value
Age (years)	20.34 \pm 2.09	20.7 \pm 1.94	0.375
Height (cm)	163.08 \pm 9.33	157.76 \pm 7.97	0.003
Weight (kg)	56.4 \pm 7.88	75.38 \pm 7.92	0.000*
BMI (kg/m ²)	21.05 \pm 1.42	30.39 \pm 1.06	0.000*
BSA (sqm)	1.61 \pm 0.15	1.76 \pm 0.13	0.000*
WC (cm)	82.88 \pm 8.20	100.74 \pm 8.20	0.000*
HC (cm)	105.50 \pm 11.33	109.90 \pm 7.53	0.25
WC/HC	0.78 \pm 0.039	0.91 \pm 0.035	0.000*
WC/Ht	0.50 \pm 0.055	0.61 \pm 0.054	0.000*
Body fat %	27.70 \pm 5.46	35.79 \pm 4.68	0.000**

BMI- Body Mass Index, BSA- Body Surface Area, WC- Waist Circumference, HC- Hip Circumference, Ht- Height

*indicates level of significance, p<0.05 Significant, p<0.01 highly significant, p<0.001 Very highly significant.

1. Age (years)

Mean \pm SD of age for study group was 20.7 ± 1.94 and control group was 20.34 ± 2.09 . There was no significant variation between two groups ($p = 0.375$). (Table 2)

2. Height (centimeters)

Mean \pm SD of height for study group was 157.76 ± 7.97 and for control group was 163.08 ± 9.33 . There was significant variation between two groups ($p = 0.003$). (Table 2)

3. Weight (kg)

Mean \pm SD of weight for study group was 75.38 ± 7.92 and for control group was 56.4 ± 7.88 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 2)

4. Body Mass Index (kg/ m²)

Mean \pm SD of BMI for study group was 30.39 ± 1.06 and for control group was 21.05 ± 1.42 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 2)

5. Body Surface Area(m²)

Mean \pm SD of BSA for study group was 1.76 ± 0.13 and for control group was 1.61 ± 0.15 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 2)

6. Waist Circumference (cm)

Mean \pm SD of WC for study group was 100.74 ± 8.20 and for control group was 82.88 ± 8.20 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 2)

7. Hip Circumference (cm)

Mean \pm SD of HC for study group was 109.90 ± 7.53 and for control group was 105.50 ± 11.33 . There was no significant variation between two groups ($p = 0.25$). (Table 2)

8. WC / HC

Mean \pm SD of WC / HC for study group was 0.91 ± 0.03 and for control group was 0.78 ± 0.03 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 2)

9. WC / Ht

Mean \pm SD of WC / Ht for study group was 0.61 ± 0.054 and for control group was 0.50 ± 0.055 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 2)

10. Body fat %

Mean \pm SD of Body fat % for study group was 35.79 ± 4.68 and for control group was 27.70 ± 5.46 . There was highly significant variation between two groups ($p = 0.007^*$). (Table 2)

FIGURE 9: Graph showing Anthropometric parameters of Control group Vs Study group

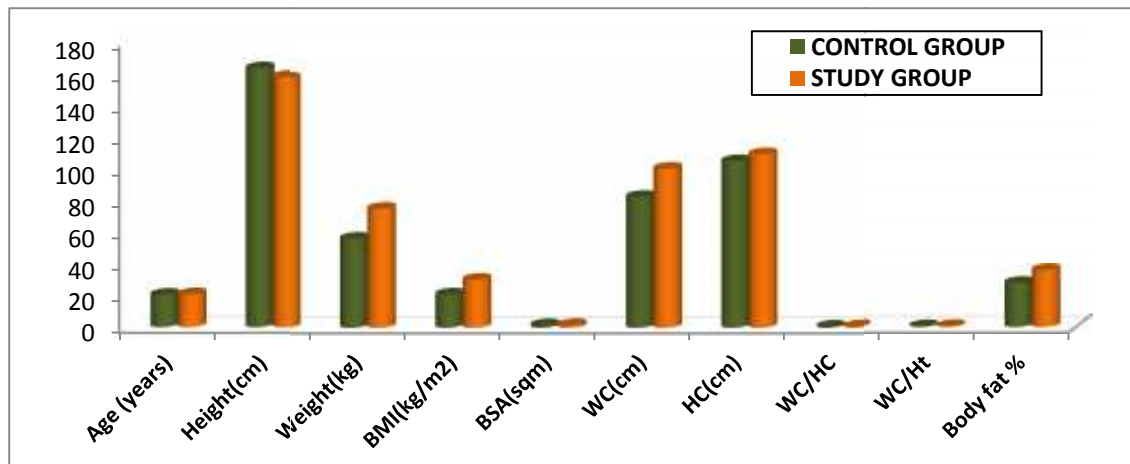


Table 3: Anthropometric Parameters of Female Control group Vs Study group (Values were Mean ± SD).

Parameter	Control group(n=18)	Study group(n=27)	p value
Age (years)	20.27 ± 2.42	20.14 ± 1.99	0.835
Height (cm)	159.05 ± 9.07	153.96 ± 7.09	0.54
Weight (kg)	53.77 ± 7.15	72.14 ± 6.56	0.000*
BMI (kg/m ²)	21.20 ± 1.23	30.37 ± 0.897	0.000*
BSA (sqm)	1.55 ± 0.14	1.70 ± 0.11	0.000*
WC (cm)	84.11 ± 6.12	102.37 ± 6.10	0.000*
HC (cm)	108.05 ± 9.50	112.51 ± 4.85	0.079
WC/HC	0.77 ± 0.002	0.90 ± 0.003	0.000*
WC/Ht	0.52 ± 0.004	0.63 ± 0.004	0.000*
Body fat %	33.50 ± 3.52	39.85 ± 1.58	0.007**

BMI- Body Mass Index, BSA- Body Surface Area, WC- Waist Circumference, HC-

Hip Circumference, Ht- Height

*indicates level of significance, $p < 0.05$ Significant, $p < 0.01$ highly significant, $p < 0.001$ Very highly significant.

1. Age (years)

Mean \pm SD of age for female study group was 20.14 ± 1.99 and for control group was 20.27 ± 2.42 . There was no significant variation between two groups ($p = 0.835$). (Table 3)

2. Height (centimeters)

Mean \pm SD of height for female study group was 153.96 ± 7.09 and for control group was 159.05 ± 9.07 . There was no significant variation between two groups ($p = 0.54$). (Table 3)

3. Weight (kg)

Mean \pm SD of weight for female study group was 72.14 ± 6.56 and for control group was 53.77 ± 7.15 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 3)

4. Body Mass Index (kg/m^2)

Mean \pm SD of BMI for female study group was 30.37 ± 0.897 and for control group was 21.20 ± 1.23 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 3)

5. Body Surface Area (m^2)

Mean \pm SD of BSA for female study group was 1.70 ± 0.11 and for control group was 1.55 ± 0.14 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 3)

6. Waist Circumference (cm)

Mean \pm SD of WC for female study group was 102.37 ± 6.10 and for control group was 84.11 ± 6.12 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 3)

7. Hip Circumference (cm)

Mean \pm SD of HC for female study group was 112.51 ± 4.85 and for control group was 108.05 ± 9.50 . There was no significant variation between two groups ($p = 0.079$). (Table 3)

8. WC / HC

Mean \pm SD of WC/HC for female study group was 0.90 ± 0.003 and for control group was 0.77 ± 0.002 . There was highly significant variation between two groups ($p=0.000^*$). (Table 3)

9. WC / Ht

Mean \pm SD of WC / Ht for female study group was 0.63 ± 0.004 and for control group was 0.52 ± 0.004 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 3)

10. Body fat %

Mean \pm SD of Body fat % for female study group was 39.85 ± 1.58 and for control group was 33.50 ± 3.52 . There was highly significant variation between two groups ($p = 0.007^*$). (Table 3)

FIGURE 10: Graph showing Anthropometric parameters of Female Control group Vs Study group.

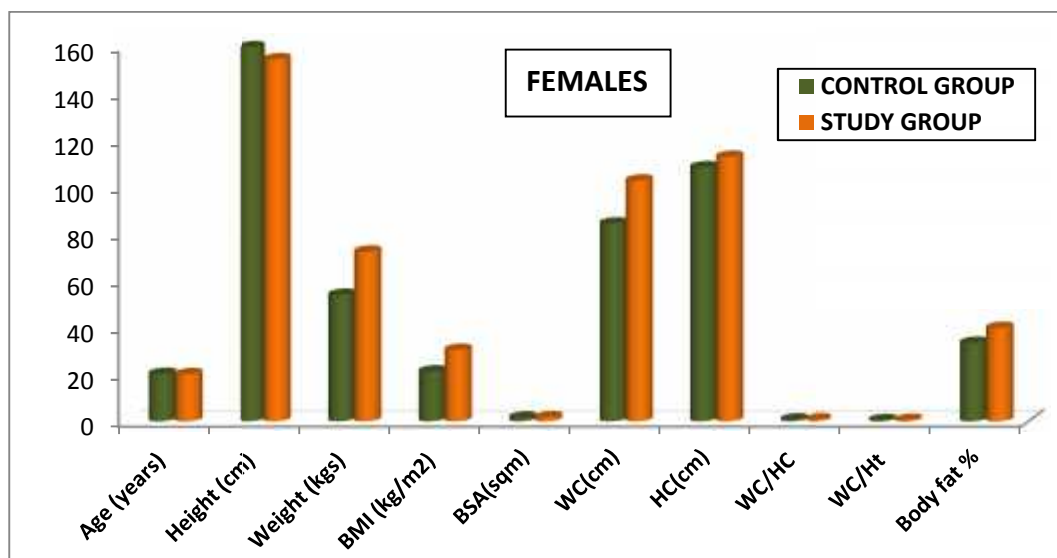


Table 4: Anthropometric Parameters of Male Control group Vs Study group (Values were Mean ± SD).

Parameter	Control group(n=32)	Study group(n=23)	p value
Age (years)	20.37± 1.93	21.34 ± 2.42	0.118
Height (cm)	165.34 ± 8.82	162.21 ± 6.61	0.139
Weight (kg)	57.87 ± 8.00	79.17± 7.81	0.000*
BMI (kg/m ²)	20.97 ± 1.53	30.41± 1.26	0.000*
BSA (sqm)	1.64 ± 0.15	1.83 ± 0.12	0.000*
WC (cm)	82.18 ± 9.19	98.82 ± 9.93	0.000*
HC (cm)	104.06 ± 12.15	106.82 ± 8.96	0.970
WC/HC	0.78 ± 0.004	0.91 ± 0.003	0.000*
WC/Ht	0.49 ± 0.005	0.60 ± 0.006	0.000*
Body fat %	24.44 ± 3.14	31.02 ± 1.39	0.000*

BMI- Body Mass Index, BSA- Body Surface Area, WC- Waist Circumference, HC- Hip Circumference, Ht- Height *indicates level of significance, p<0.05 Significant, p<0.01 highly significant, p<0.001 Very highly significant.

1. Age (years)

Mean \pm SD of age for male study group was 21.34 ± 2.42 and for control group was 20.37 ± 1.93 . There was no significant variation between two groups ($p=0.118$). (Table 4)

2. Height (centimeters)

Mean \pm SD of height for male study group was 162.21 ± 6.61 and for control group was 165.34 ± 8.82 . There was no significant variation between two groups ($p=0.139$). (Table 4)

3. Weight (kg)

Mean \pm SD of weight for male study group was 79.17 ± 7.81 and for control group was 57.87 ± 8.00 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 4)

4. Body Mass Index (kg/ m²)

Mean \pm SD of BMI for male study group was 30.41 ± 1.26 and for control group was 20.97 ± 1.53 . There was highly significant variation between two groups ($p=0.000^*$). (Table 4)

5. Body Surface Area (m²)

Mean \pm SD of weight for male study group was 1.83 ± 0.12 and for control group was 1.64 ± 0.15 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 4)

6. Waist Circumference (cm)

Mean \pm SD of WC for male study group was 98.82 ± 9.93 and for control group was 82.18 ± 9.19 . There was highly significant variation between two groups ($p=0.000^*$). (Table 4)

7. Hip Circumference (cm)

Mean \pm SD of HC for male study group was 106.82 ± 8.96 and for control group was 104.06 ± 12.15 . There was no significant variation between two groups ($p=0.970$). (Table 4)

8. WC / HC

Mean \pm SD of WC / HC for male study group was 0.91 ± 0.003 and for control group was 0.78 ± 0.004 . There was highly significant variation between two groups ($p=0.000^*$). (Table 4)

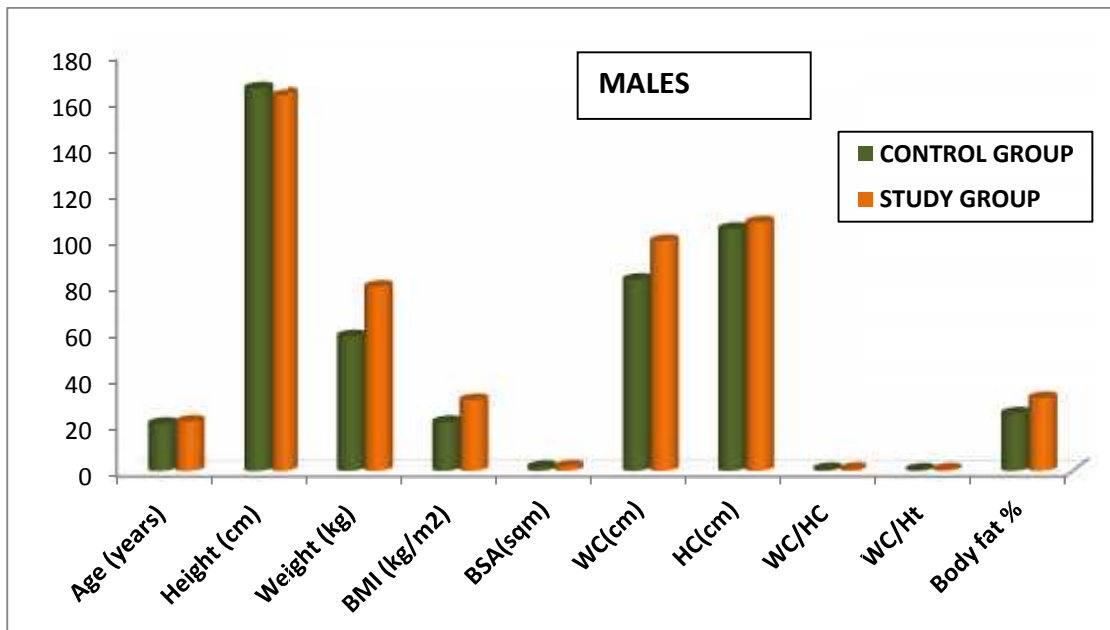
9. WC / Ht

Mean \pm SD of WC / Ht for male study group was 0.60 ± 0.006 and for control group was 0.49 ± 0.005 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 4)

10. Body fat %

Mean \pm SD of body fat % for male study group was 31.02 ± 1.39 and for control group was 24.44 ± 3.14 . There was highly significant variation between two groups ($p=0.000^*$). (Table 4)

FIGURE 11: Graph showing Anthropometric parameters of Male Control group Vs Study group



II. Physiological Parameters

Recording of various Physiological Parameters in control and study groups were represented in table numbers: 05-07. The Parameters recorded included Pulse Rate (beats/min), Respiratory Rate (cycles/min), Systolic Blood Pressure (mm Hg), Diastolic Blood Pressure (mm Hg) and Mean Arterial Blood Pressure (mmHg). The values were represented as Mean with Standard deviation of each Parameter in each group.

Table 5: Physiological Parameters of Control group Vs Study group (Values were Mean \pm SD)

Parameter	Control group(n=50)	Study group(n=50)	p value
Pulse rate (bpm)	73.92 \pm 5.00	85.46 \pm 6.13	0.000*
Respiratory rate(cpm)	13.76 \pm 2.24	21.56 \pm 1.97	0.000*
SBP (mmHg)	115.00 \pm 8.69	132.40 \pm 9.59	0.000*
DBP (mmHg)	74.04 \pm 6.05	84.80 \pm 6.46	0.000*
MAP (mmHg)	87.35 \pm 6.35	101.2 \pm 6.48	0.000*

SBP- Systolic Blood Pressure, DBP-Diastolic Blood Pressure, MAP- Mean Arterial Pressure. *indicates level of significance, $p < 0.01$

1. **Pulse Rate (beats/min)**

Mean \pm SD of Pulse Rate for study group was 85.46 \pm 6.13 and for control group was 73.92 \pm 5.00. There was highly significant variation between two groups ($p=0.000^*$). (Table 5)

2. **Respiratory Rate (cycles/min)**

Mean \pm SD of Respiratory Rate for study group was 21.56 \pm 1.97 and for control group was 13.76 \pm 2.24. There was highly significant variation between two groups ($p=0.000^*$). (Table 5)

3. **Systolic BP (mmHg)**

Mean \pm SD of Systolic BP for study group was 132.40 \pm 9.59 and for control group was 115.00 \pm 8.69. There was highly significant variation between two groups ($p=0.000^*$). (Table 5)

4. **Diastolic BP (mmHg)**

Mean \pm SD of Diastolic BP for study group was 84.80 \pm 6.46 and for control group was 74.04 \pm 6.05. There was highly significant variation between two groups ($p=0.000^*$). (Table 5)

4. Mean Arterial Blood Pressure (mmHg)

Mean \pm SD of Mean Arterial Blood Pressure for study group was 101.2 ± 6.48 and for control group was 87.35 ± 6.35 . There was highly significant variation between two groups ($p=0.000^*$). (Table 5)

FIGURE 12: Graph showing Physiological parameters of Control group Vs Study group

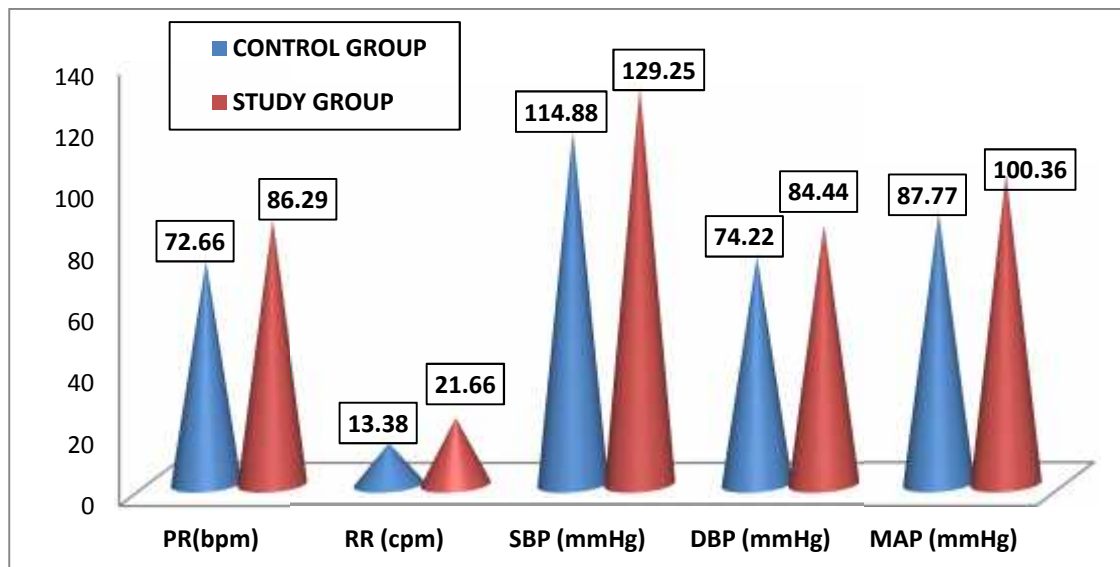


Table 6: Physiological Parameters of Female Control group Vs Study group (Values were Mean \pm SD).

Parameters	Control group(n=18)	Study group(n=27)	p value
Pulse rate (bpm)	72.66 ± 5.42	86.29 ± 5.84	0.000*
Respiratory rate(cpm)	13.38 ± 1.57	21.66 ± 1.81	0.000*
SBP (mmHg)	114.88 ± 8.37	129.25 ± 7.80	0.000*
DBP (mmHg)	74.22 ± 5.61	84.44 ± 6.40	0.000*
MAP (mmHg)	87.77 ± 5.94	100.36 ± 6.68	0.000*

SBP- Systolic Blood Pressure, DBP-Diastolic Blood Pressure, MAP- Mean Arterial Pressure. *indicates level of significance, $p<0.01$

1. Pulse Rate (beats/min)

Mean \pm SD of Pulse Rate for female study group was 86.29 ± 5.84 and for control group was 72.66 ± 5.42 . There was highly significant variation between two groups ($p=0.000^*$). (Table 6)

2. Respiratory Rate (cycles/min)

Mean \pm SD of Respiratory Rate for female study group was 21.66 ± 1.81 and for control group was 13.38 ± 1.57 . There was highly significant variation between two groups ($p=0.000^*$). (Table 6)

3. Systolic BP (mmHg)

Mean \pm SD of Systolic BP for female study group was 129.25 ± 7.80 and for control group was 114.88 ± 8.37 . There was highly significant variation between two groups ($p=0.000^*$). (Table 6)

4. Diastolic BP (mmHg)

Mean \pm SD of Diastolic BP for female study group was 84.44 ± 6.40 and for control group was 74.22 ± 5.61 . There was highly significant variation between two groups ($p=0.000^*$). (Table 6)

5. Mean Arterial Blood Pressure (mmHg):

Mean \pm SD of Mean Arterial Blood Pressure for female study group was 100.36 ± 6.68 and for control group was 87.77 ± 5.94 . There was highly significant variation between two groups ($p=0.000^*$). (Table6)

FIGURE 13: Graph showing Physiological parameters of Female Control group Vs Study group

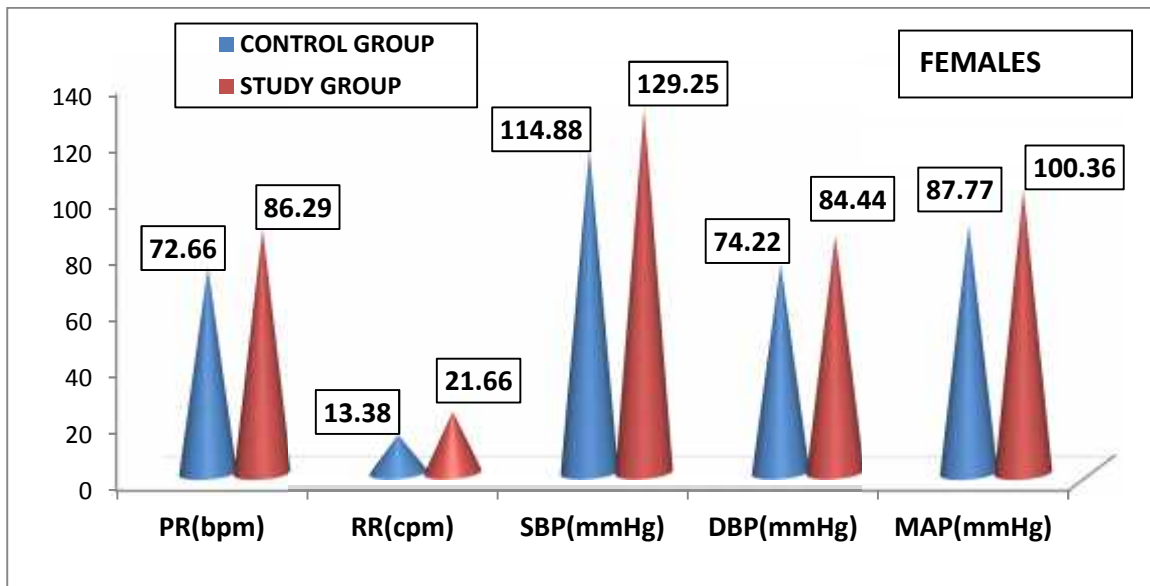


Table 7: Physiological Parameters of Male Control group Vs Study group. (Values were Mean \pm SD).

Parameter	Control group(n=32)	Study group(n=23)	p value
Pulse rate (bpm)	74.62 \pm 4.96	84.47 \pm 6.44	0.000*
Respiratory rate(cpm)	13.96 \pm 2.54	21.43 \pm 2.17	0.000*
SBP (mmHg)	115.06 \pm 9.00	136.08 \pm 10.33	0.000*
DBP (mmHg)	73.93 \pm 6.37	85.21 \pm 6.65	0.000*
MAP (mmHg)	87.12 \pm 6.62	102.16 \pm 6.66	0.000*

SBP- Systolic Blood Pressure, DBP-Diastolic Blood Pressure, MAP- Mean Arterial Pressure. *indicates level of significance, $p < 0.01$

1. Pulse Rate (beats/min)

Mean \pm SD of Pulse Rate for male study group was 84.47 ± 6.44 and for control group was 74.62 ± 4.96 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 7)

2. Respiratory Rate (cycles/min)

Mean \pm SD of Respiratory Rate for male study group was 21.43 ± 2.17 and for control group was 13.96 ± 2.54 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 7)

3. Systolic BP (mmHg)

Mean \pm SD of Systolic BP for male study group was 136.08 ± 10.33 and for control group was 115.06 ± 9.00 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 7)

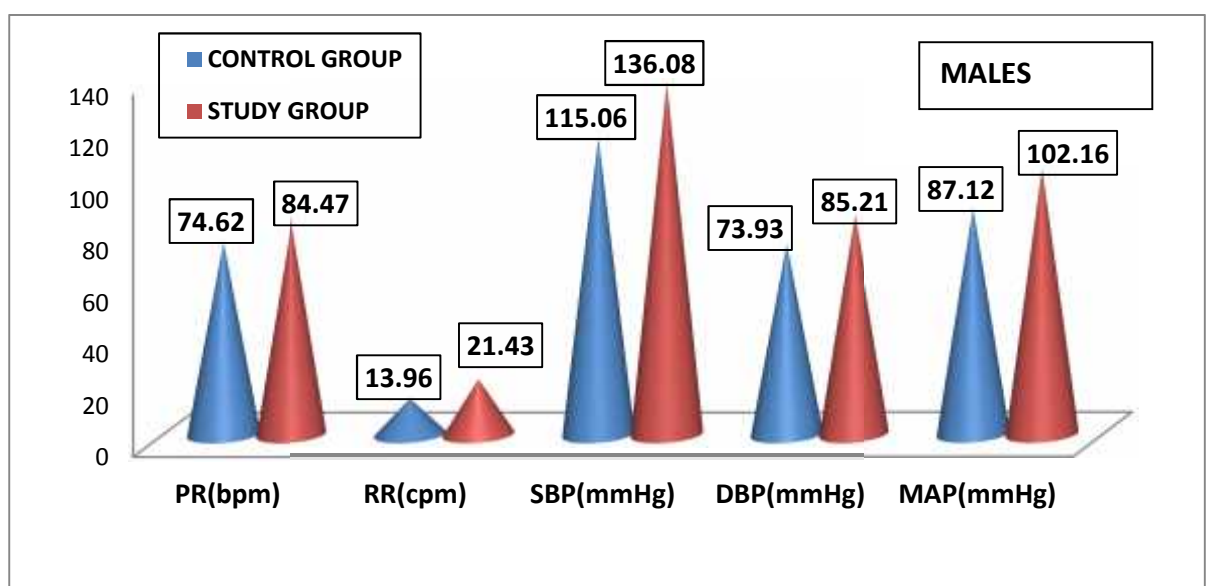
4. Diastolic BP (mmHg)

Mean \pm SD of Diastolic BP for male study group was 85.21 ± 6.65 and for control group was 73.93 ± 6.37 . There was highly significant variation between two groups ($p=0.000^*$). (Table 7)

5. Mean Arterial Blood Pressure (mmHg):

Mean \pm SD of Mean Arterial BP for male study group was 102.16 ± 6.24 and for control group was 87.12 ± 6.66 . There was highly significant variation between two groups ($p=0.000^*$). (Table 7)

FIGURE 14: Graph showing Physiological parameters of Male Control group Vs Study group.



III. Pulmonary function parameters in study and control groups

Recording of various pulmonary function parameters in control and study groups were represented in table numbers: 08-10. The Parameters recorded included Forced Vital Capacity (ml), Forced Expiratory Volume in 1st second (ml), Percentage of Forced Expiratory Volume in 1st second, Peak Expiratory Flow Rate in (L / min) and Mean Expiratory Pressure in mm Hg. The values were presented as Mean with Standard deviation of each Parameter in each group.

Table 8: Pulmonary Function Parameters of Control group Vs Study group. (Values were Mean \pm SD).

Parameter	Control group(n=50)	Study group(n=50)	p value
FVC (L)	2.64 \pm 0.70	2.48 \pm 0.62	0.222
FEV ₁ (L)	2.38 \pm 0.65	2.19 \pm 0.66	0.157
FEV ₁ %	92.07 \pm 7.69	88.50 \pm 12.84	0.09
PEFR (L/min)	466.0 \pm 98.68	316.4 \pm 103.20	0.000*
MEP (mmHg)	67.24 \pm 29.76	27 \pm 13.83	0.000*

FVC- Forced Vital Capacity, FEV₁- Forced Vital Capacity in first second, PEFR- Peak Expiratory Flow Rate, MEP- Maximum Expiratory Pressure

*indicates level of significance, p<0.01

1. Forced Vital Capacity (FVC) (Liters):

Mean \pm SD of FVC for study group was 2.48 \pm 0.62 and for control group was 2.64 \pm 0.70. There was no significant variation between two groups (p=0.222). (Table 8)

2. Forced Expiratory Volume at the end of 1st second (FEV₁) (Liters):

Mean \pm SD of FEV₁ for study group was 2.19 \pm 0.66 and for control group was 2.38 \pm 0.65. There was no significant variation between two groups (p=0.157). (Table 8)

3. Forced Expiratory Volume at the end of 1st second in percentage (FEV_{1%}):

Mean \pm SD of FEV_{1%} for study group was 88.50 ± 12.84 and for control group was 92.07 ± 7.69 . There was no significant variation between two groups ($p=0.09$). (Table 8)

4. Peak Expiratory Flow Rate (PEFR) (Liters/min):

Mean \pm SD of PEFR for female study group was 316.4 ± 103.20 and for control group was 466.0 ± 98.68 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 8)

5. Maximum Expiratory Pressure (MEP) (mmHg):

Mean \pm SD of MEP for female study group was 27 ± 13.83 and for control group was 67.24 ± 29.76 . There was highly significant variation between two groups ($p = 0.000^*$). (Table 8)

FIGURE 15: Graph showing FVC and FEV1 of Control group Vs Study group

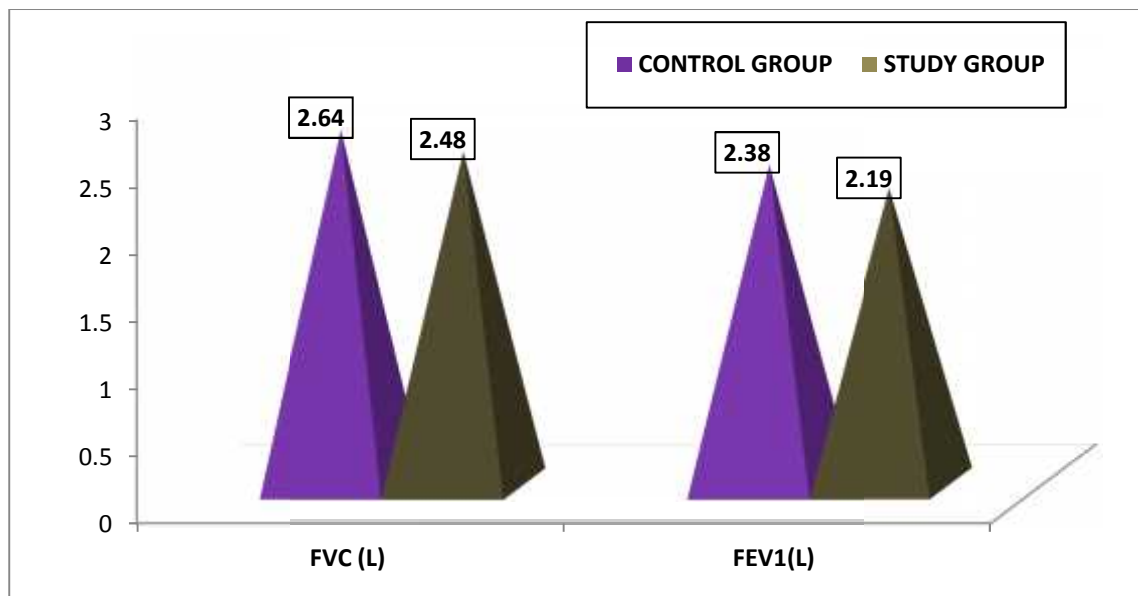


FIGURE 16: Graph showing FEV1 %, PEFR and MEP of Control group Vs Study group

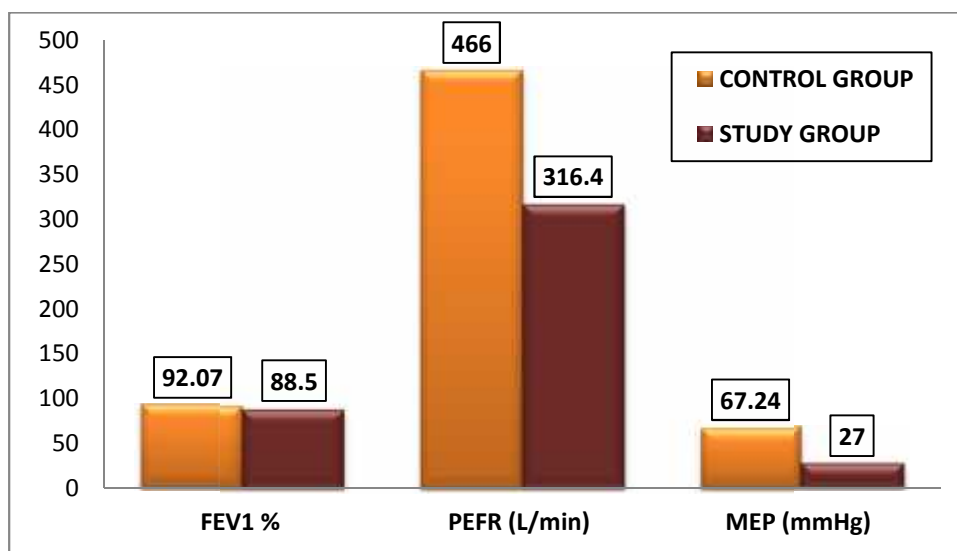


Table 9: Pulmonary Function Parameters of Female Study group Vs Control group. (Values were Mean \pm SD).

Parameter	Control group(n=18)	Study group(n=27)	p value
FVC (L)	2.183 \pm 0.67	2.180 \pm 0.45	0.987
FEV ₁ (L)	1.99 \pm 0.63	1.90 \pm 0.55	0.636
FEV ₁ %	93.20 \pm 4.89	86.57 \pm 14.90	0.039 *
PEFR (L/min)	452.7 \pm 91.51	297.03 \pm 78.48	0.000*
MEP (mmHg)	59.77 \pm 24.70	25.55 \pm 14.13	0.000*

FVC- Forced Vital Capacity, FEV₁- Forced Vital Capacity in first second, PEFR-

Peak Expiratory Flow Rate, MEP- Maximum Expiratory Pressure

*indicates level of significance, p<0.01

1. Forced Vital Capacity (FVC) (Liters):

Mean \pm SD of FVC for female study group was 2.180 ± 0.45 and for control group was 2.183 ± 0.67 . There was no significant variation between two groups ($p=0.987$). (Table 9)

2. Forced Expiratory Volume at the end of 1st second (FEV1) (Liters):

Mean \pm SD of FEV1 for female study group was 1.90 ± 0.55 and for control group was 1.99 ± 0.63 . There was no significant variation between two groups ($p=0.636$). (Table 9)

3. Forced Expiratory Volume at the end of 1st second in percentage (FEV1%):

Mean \pm SD of FEV1% for female study group was 86.57 ± 14.90 and for control group was 93.20 ± 4.89 . There was significant variation between two groups ($p=0.039^*$). (Table 9)

4. Peak Expiratory Flow Rate (PEFR) (Liters/min):

Mean \pm SD of PEFR for female study group was 297.03 ± 78.48 and for control group was 452.7 ± 91.51 . There was highly significant variation between two groups ($p=0.000^*$). (Table 9)

5. Maximum Expiratory Pressure (MEP) (mmHg):

Mean \pm SD of MEP for female study group was 25.55 ± 14.13 and for control group was 59.77 ± 24.70 . There was highly significant variation between two groups ($p=0.000^*$). (Table 9)

FIGURE 17: Graph showing FVC and FEV1 of Female Control group Vs Study group

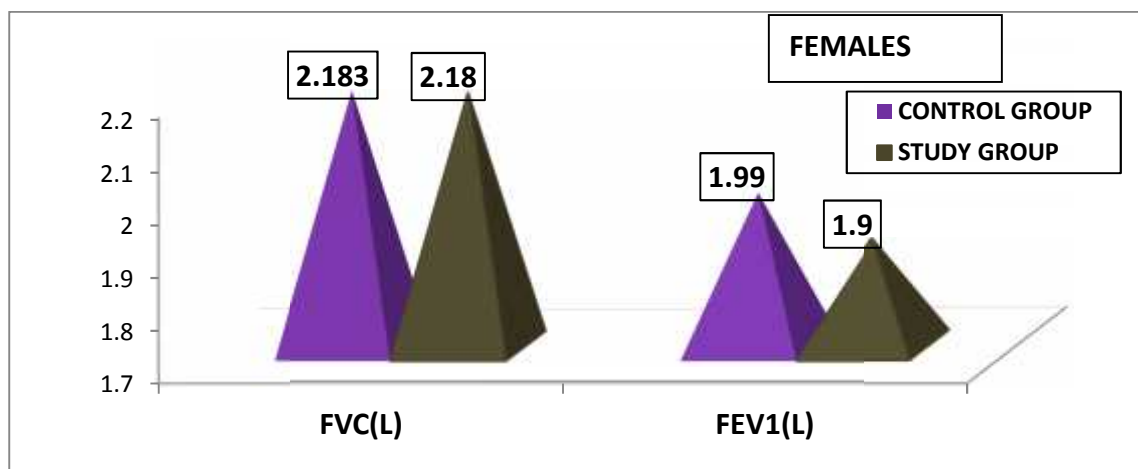


FIGURE 18: Graph showing FEV1%, PEFR and MEP of Female Control group Vs Study group

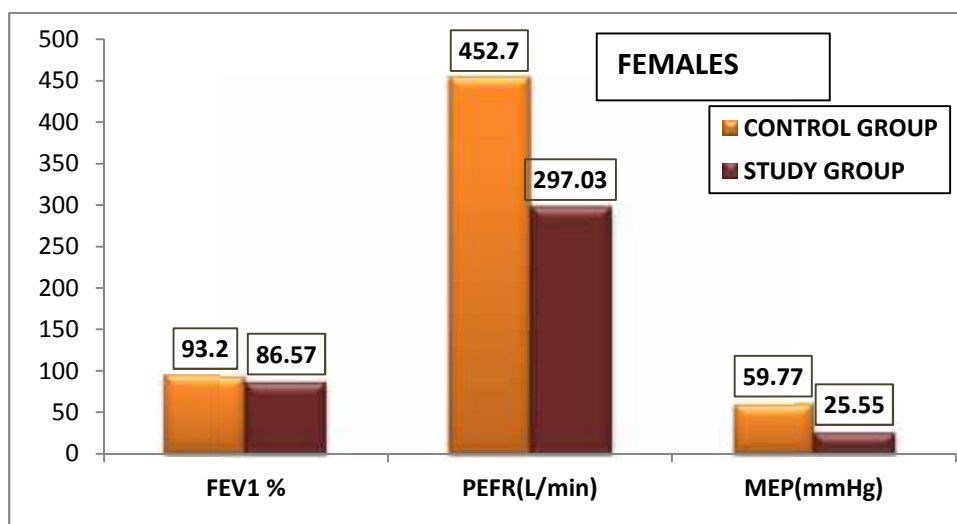


Table 10: Pulmonary Function Parameters of Male Control group Vs Study group. (Values were Mean \pm SD).

Parameter	Control group(n=32)	Study group(n=23)	p value
FVC (L)	2.90 \pm 0.58	2.83 \pm 0.63	0.654
FEV ₁ (L)	2.60 \pm 0.56	2.53 \pm 0.63	0.678
FEV ₁ %	91.44 \pm 8.00	90.77 \pm 9.74	0.790
PEFR (L/min)	473.43 \pm 103.16	339.13 \pm 124.27	0.000*
MEP (mmHg)	71.43 \pm 31.86	28.69 \pm 13.58	0.000*

FVC- Forced Vital Capacity, FEV1- Forced Vital Capacity in first second, PEFR- Peak Expiratory Flow Rate, MEP- Maximum Expiratory Pressure

*indicates level of significance, $p < 0.01$

1. Forced Vital Capacity (FVC) (Liters):

Mean \pm SD of FVC for male study group was 2.83 ± 0.63 and for control group was 2.90 ± 0.58 . There was no significant variation between two groups ($p=0.654$).

(Table 10)

2. Forced Expiratory Volume at the end of 1st second (FEV₁) (Liters):

Mean \pm SD of FEV₁ for male study group was 2.53 ± 0.63 and for control group was 2.60 ± 0.56 . There was no significant variation between two groups

($p=0.678$). (Table 10)

3. Forced Expiratory Volume at the end of 1st second in percentage (FEV_{1%}):

Mean \pm SD of FEV_{1%} for male study group was 90.77 ± 9.74 and for control group was 91.44 ± 8.00 . There was no significant variation between two groups ($p=0.790$).

(Table 10)

4. Peak Expiratory Flow Rate(PEFR) (Liters/min)

Mean \pm SD of PEFR for male study group was 339.13 ± 124.27 and for control group was 473.43 ± 103.16 . There was highly significant variation between two

groups ($p = 0.000^*$). (Table10).

5. Maximum Expiratory Pressure (MEP) (mmHg):

Mean \pm SD of MEP for male study group was 28.69 ± 13.58 and for control group was 71.43 ± 31.86 . There was highly significant variation between two

groups ($p=0.000^*$). (Table 10)

FIGURE 19: Graph showing FVC and FEV1 of Male Control group Vs Study group

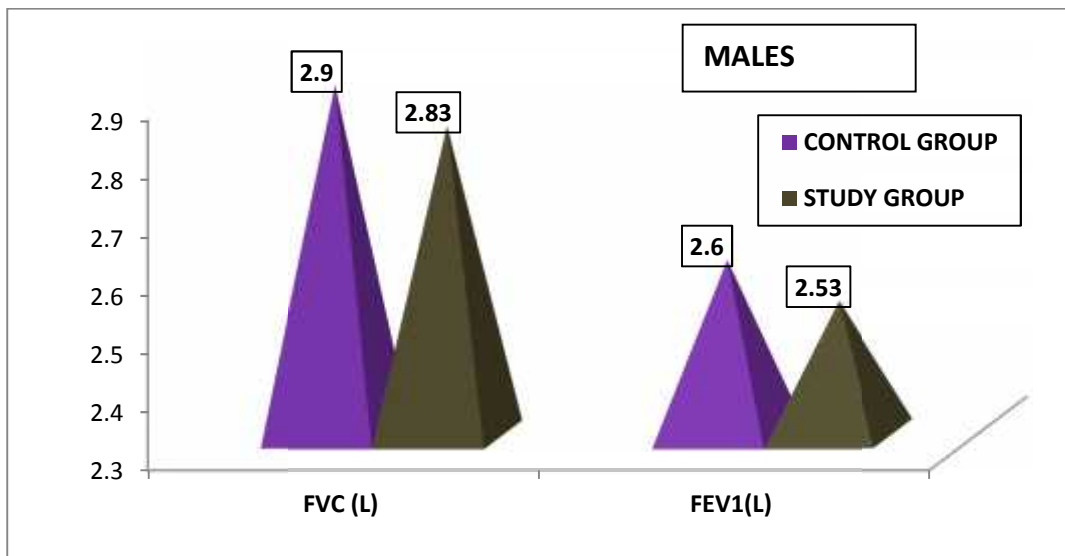
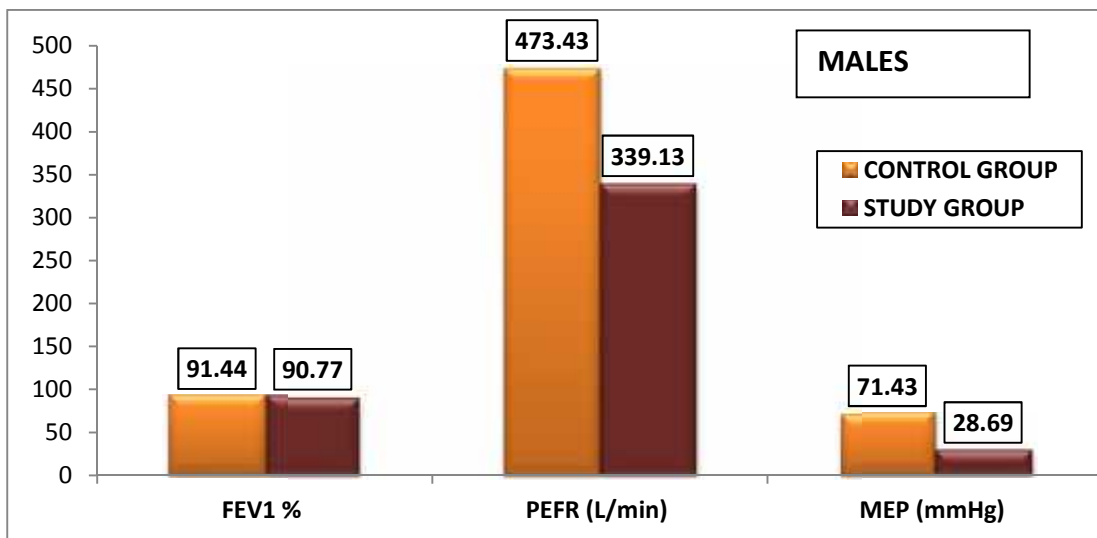


FIGURE 20: Graph showing FEV1%, PEFR and MEP of Male Control group Vs Study group



Coefficient of determination (r^2) was used to examine the degree of obesity with pulmonary functions.

Table 11: Bivariate analysis of adiposity markers with pulmonary functions

Parameter	FVC	FEV1	FEV1%	PEFR	MEP
	r (p value)	r (p value)	r (p value)	r (p value)	r (p value)
BMI	-0.120 (0.236)	-0.130 (0.198)	-0.168 (0.09)	-0.58* (0.000)	-0.66* (0.000)
WC	-0.253* (0.011)	-0.236* (0.018)	-0.163 (0.09)	-0.358* (0.000)	-0.455* (0.000)
WHR	-0.065 (0.518)	-0.039 (0.697)	-0.068 (0.501)	-0.585* (0.00)	-0.603* (0.000)
WHtR	-0.357* (0.000)	-0.319* (0.001)	-0.116 (0.250)	-0.386* (0.000)	-0.492* (0.000)
Body fat %	-0.247 (0.013)*	-0.249 (0.012)*	-0.270 (0.007)**	-0.520 (0.000)**	-0.482 (0.000)**

r is correlation, * indicates level of significance($p < 0.05$)

Adiposity markers (BMI, WC, WHR, WHtR and Body Fat %) were negatively correlated with pulmonary functions.

1. BMI was significantly negatively correlated with PEFR($r = -0.580$, $p = 0.000$) and MEP ($r = -0.66$, $p = 0.000$).
2. WC was significantly negatively correlated with FVC ($r = -0.253$, $p = 0.011$), FEV1($r = -0.236$, $p = 0.018$), PEFR($r = -0.358$, $p = 0.000$) and MEP($r = -0.455$, $p = 0.000$).
3. WHR was significantly negatively correlated with PEFR($r = -0.585$, $p = 0.000$) and MEP($r = -0.603$, $p = 0.000$).

4. WHtR was significantly negatively correlated with FVC($r = -0.357$, $p = 0.000$), FEV1($r = -0.319$, $p = 0.001$), PEFr($r = -0.386$, $p = 0.000$) and MEP($r = -0.492$, $p = 0.000$).
5. Body Fat % was significantly negatively correlated with FVC($r = -0.247$, $p = 0.013$), FEV1($r = -0.249$, $p = 0.012$), FEV1% ($r = -0.270$, $p = 0.007$), PEFr($r = -0.520$, $p = 0.000$) and MEP($r = -0.482$, $p = 0.000$).

FIGURE 21– Graph showing significant negative correlation between FVC and WC ($R^2 = 0.023$)*

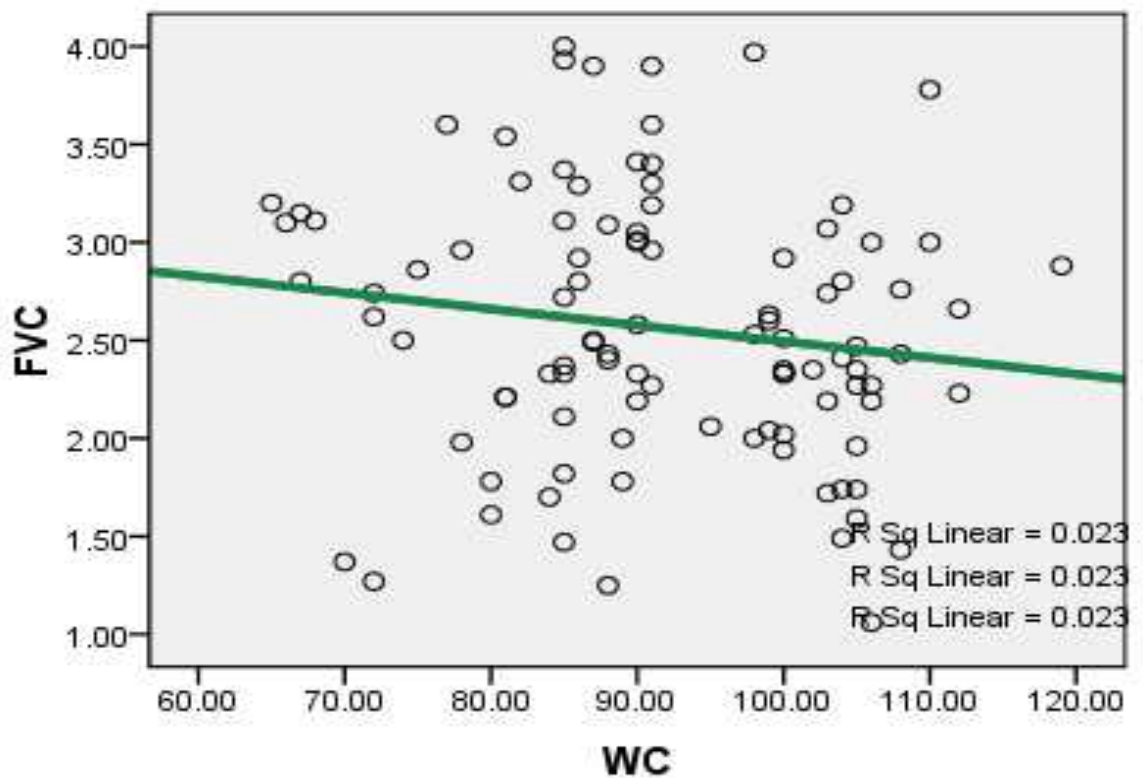


FIGURE 22- Graph showing significant negative correlation between FVC and WHtR ($R^2= 0.036$)*

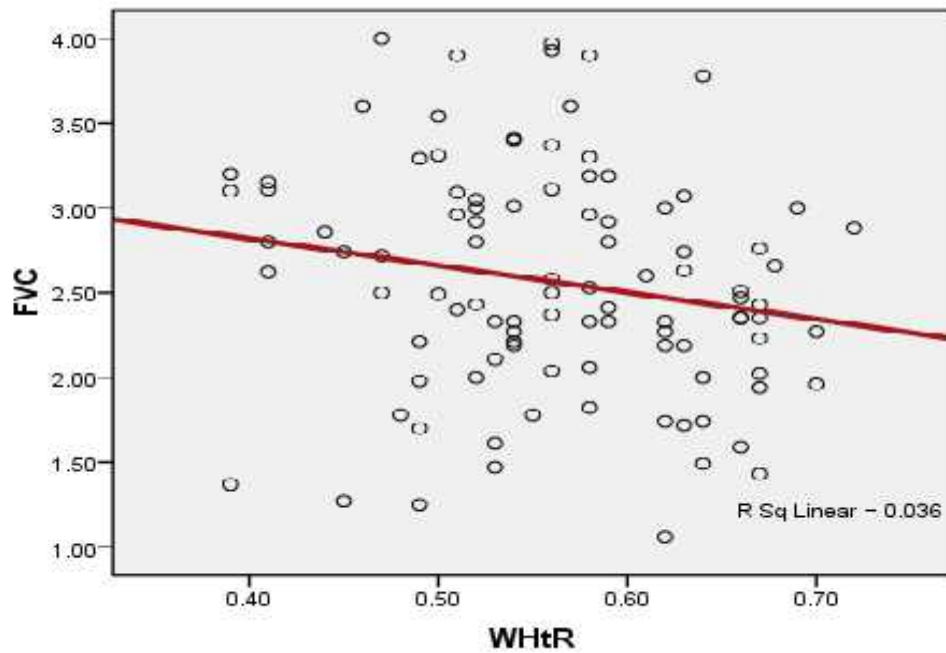
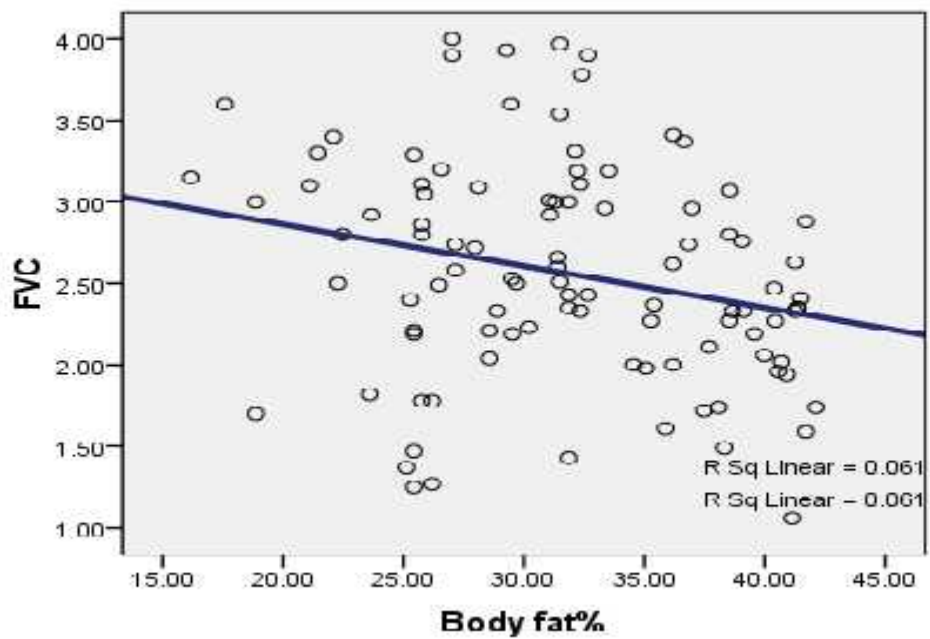
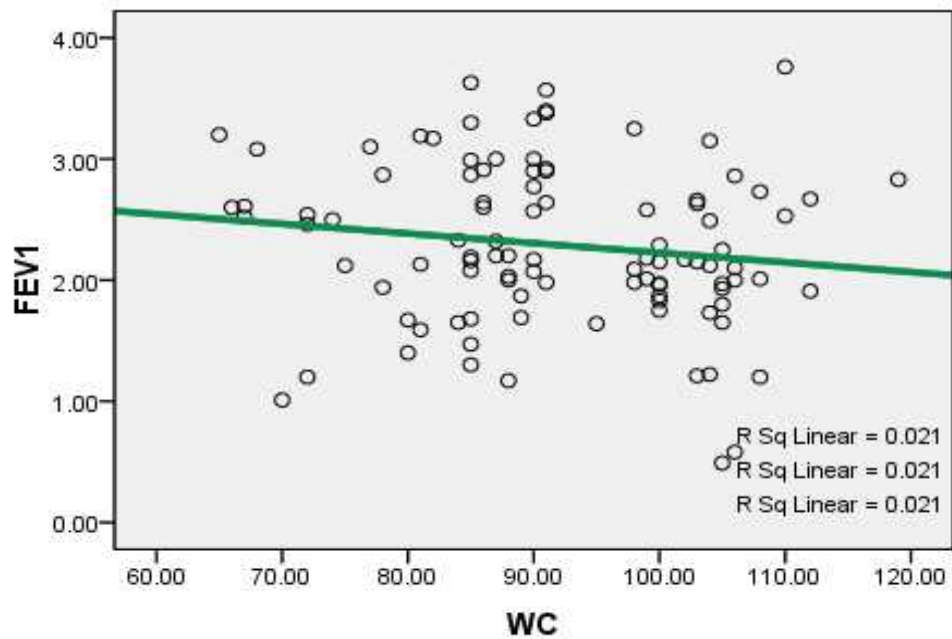


FIGURE 23- Graph showing significant negative correlation between FVC and Body fat % ($R^2= 0.061$)*



**FIGURE 24- Graph showing significant negative correlation between
FEV1 and WC ($R^2= 0.021$)***



**FIGURE 25- Graph showing significant negative correlation between
FEV1 and WHtR ($R^2= 0.03$)***

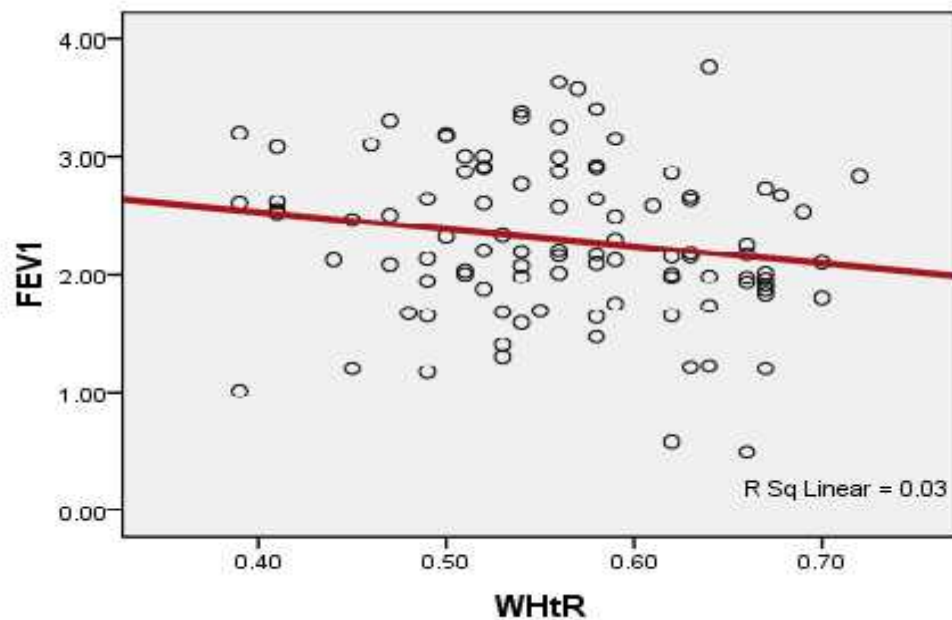


FIGURE 26- Graph showing significant negative correlation between FEV1 and Body fat% ($R^2= 0.062$)*

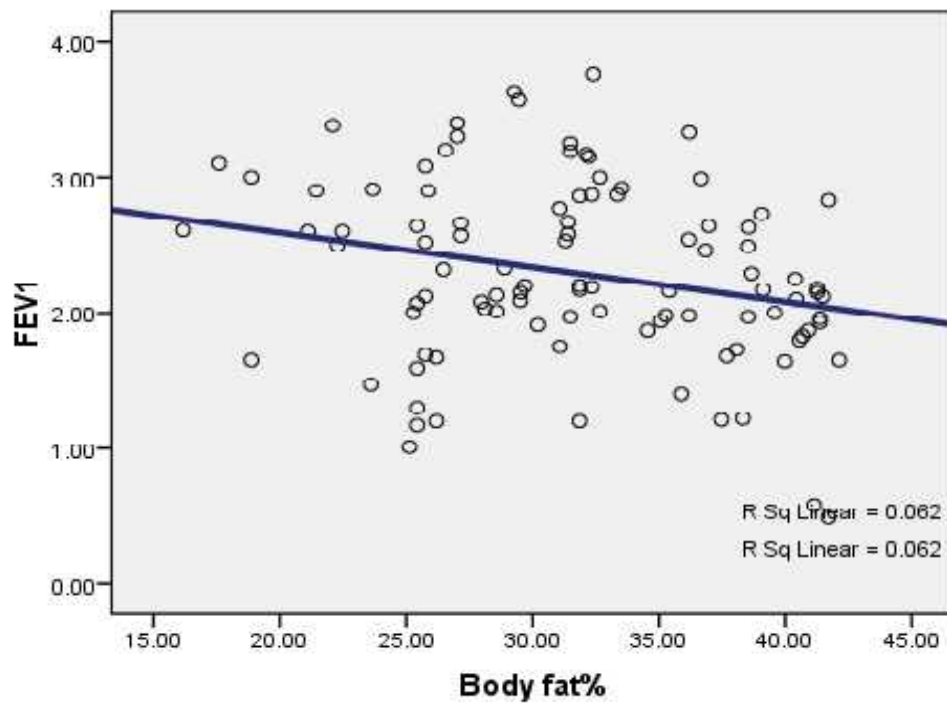


FIGURE 27- Graph showing significant negative correlation between FEV1% and WC ($R^2= 0.022$)*

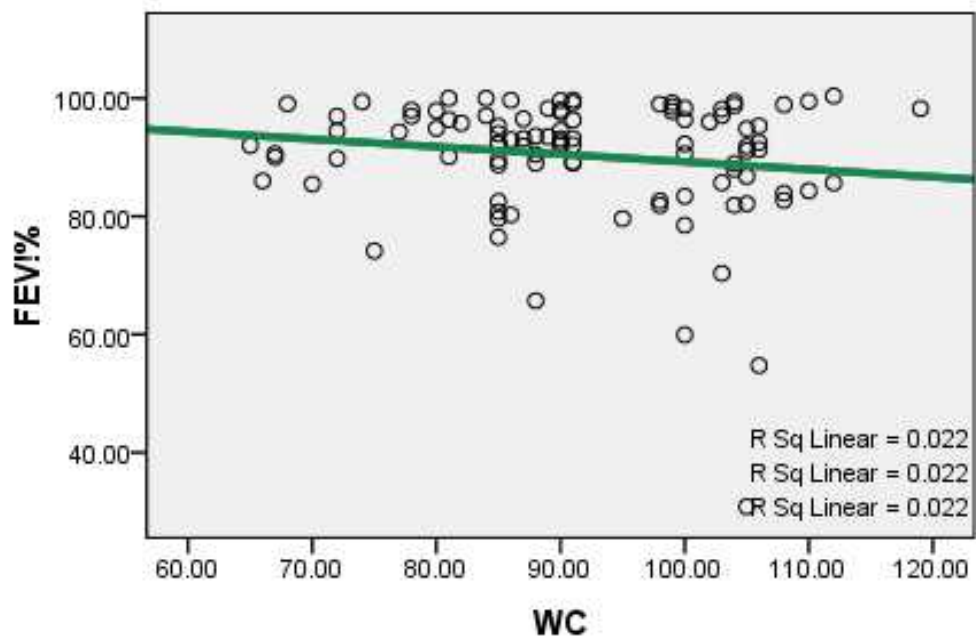


FIGURE 28- Graph showing significant negative correlation between FEV1% and Body fat % ($R^2= 0.03$)*

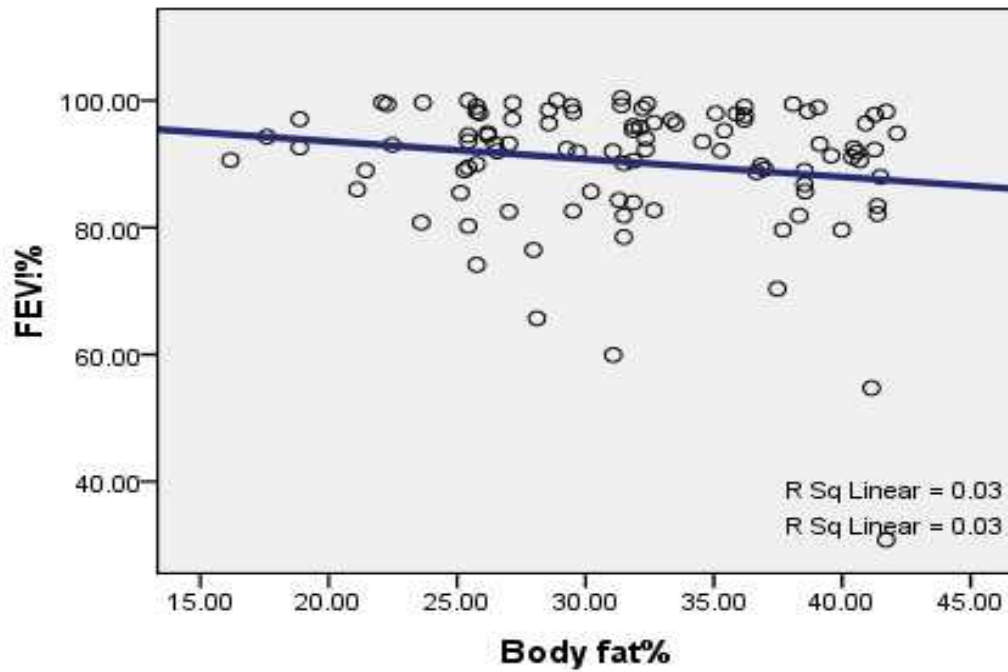
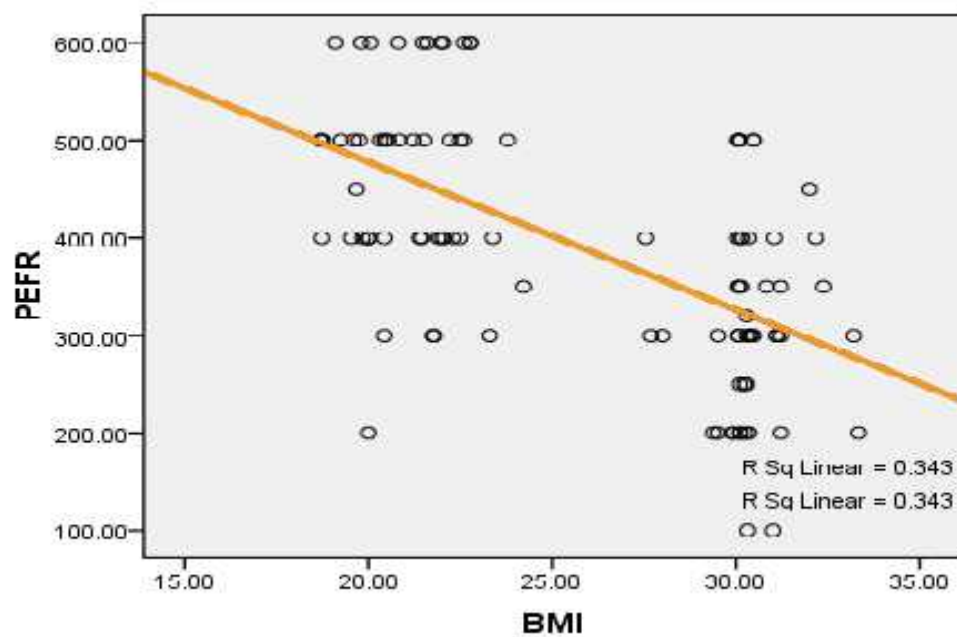
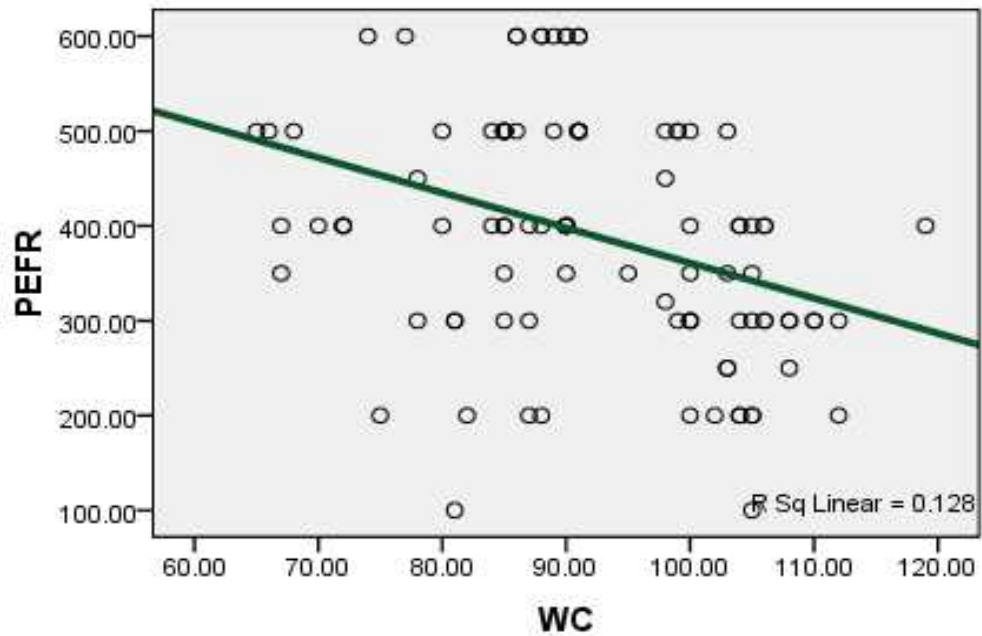


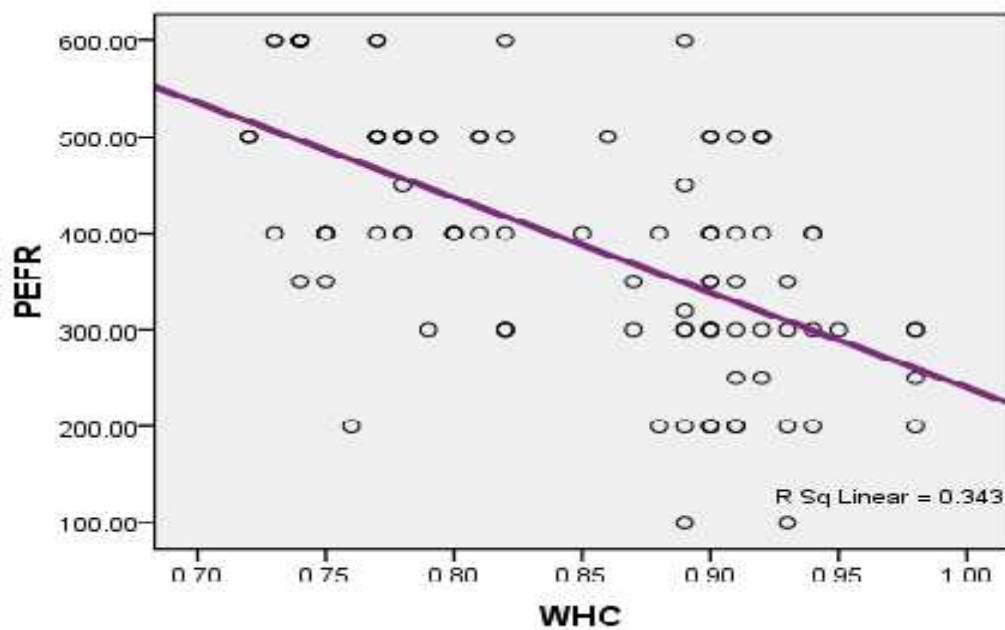
FIGURE 29- Graph showing significant negative correlation between PEFR and BMI ($R^2= 0.343$)*



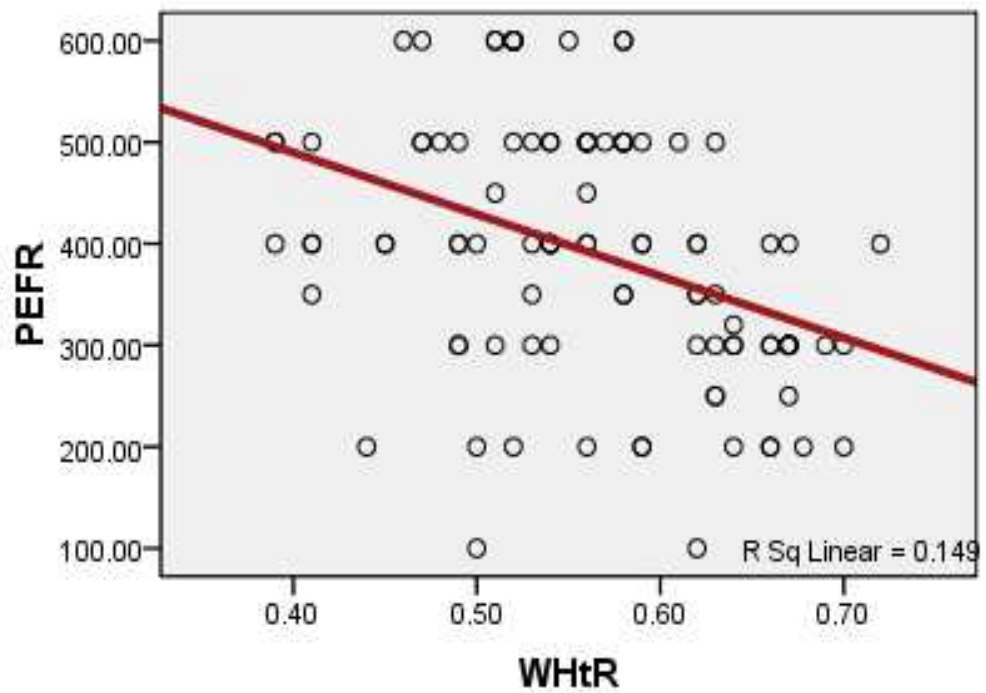
**FIGURE 30- Graph showing significant negative correlation between
PEFR and WC ($R^2= 0.128$)***



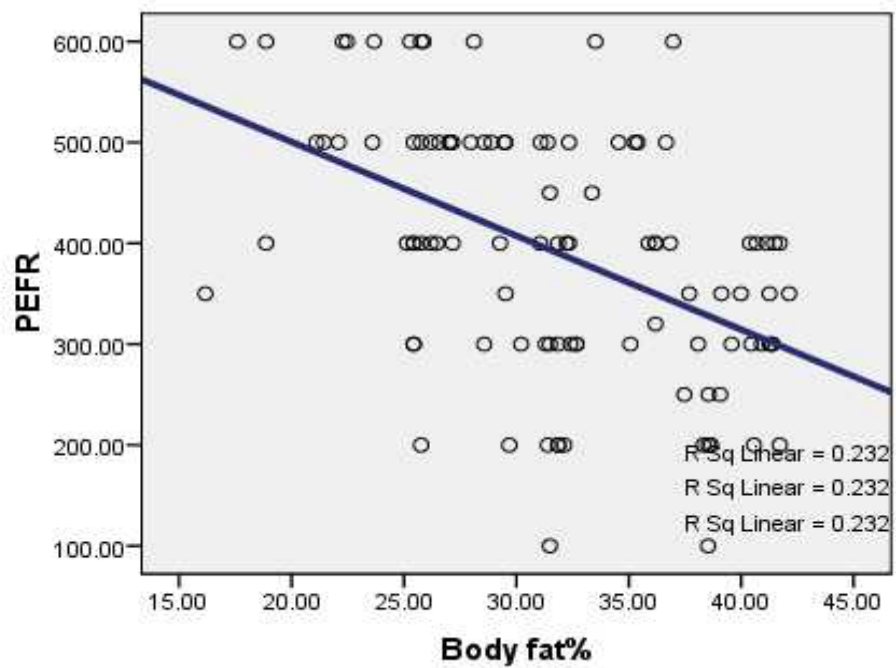
**FIGURE 31- Graph showing significant negative correlation between
PEFR and WHC ($R^2= 0.343$)***



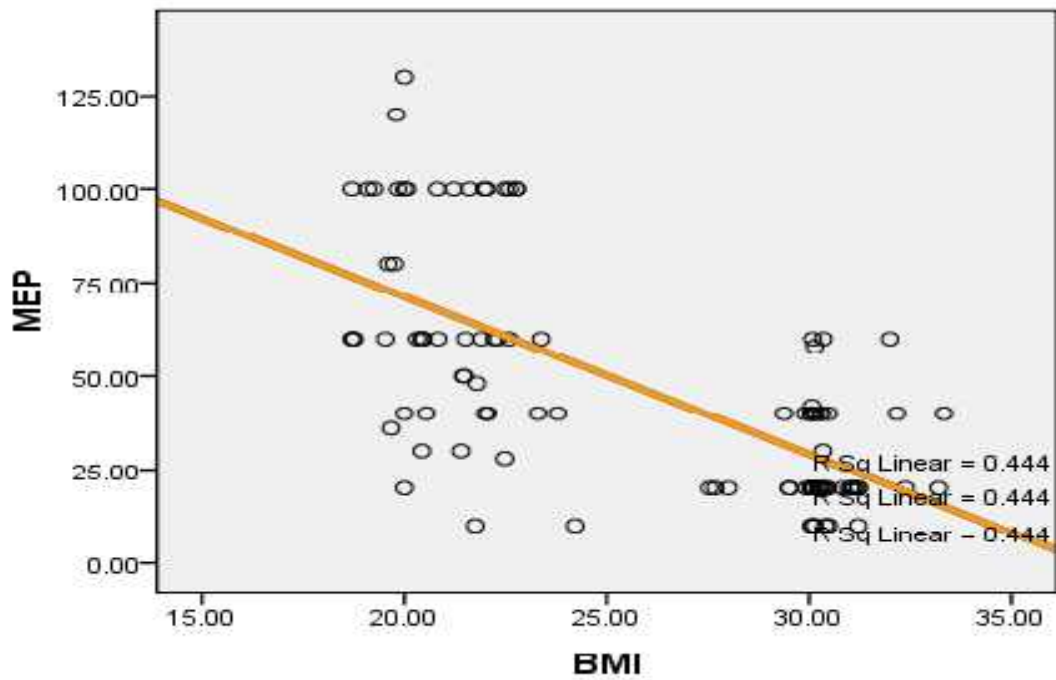
**FIGURE 32- Graph showing significant negative correlation between
PEFR and WHtR ($R^2= 0.149$)***



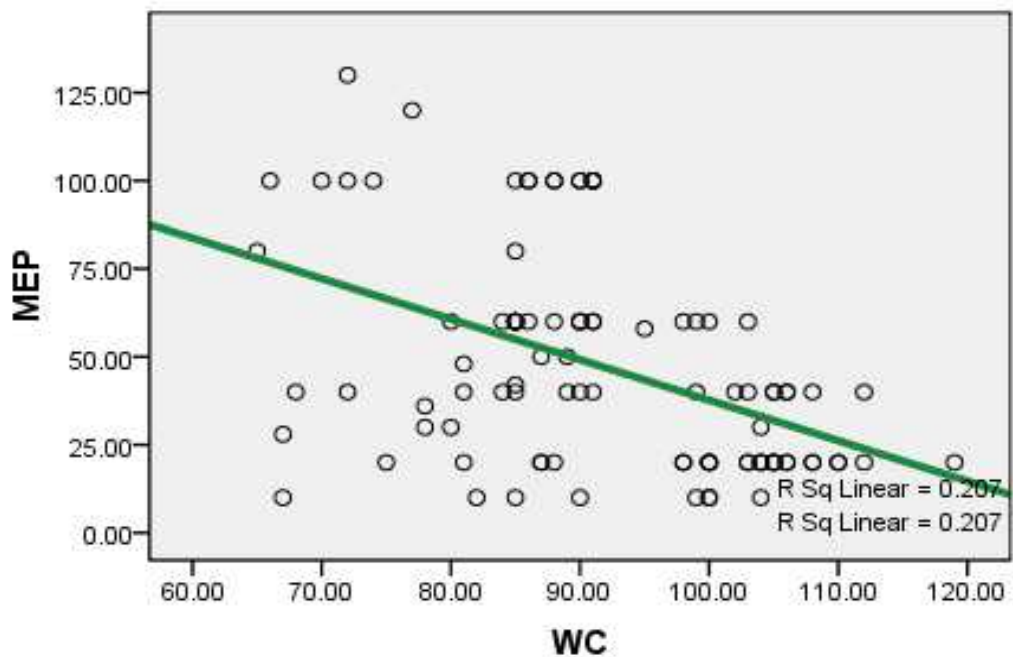
**FIGURE 33- Graph showing significant negative correlation between
PEFR and Body fat % ($R^2= 0.232$)***



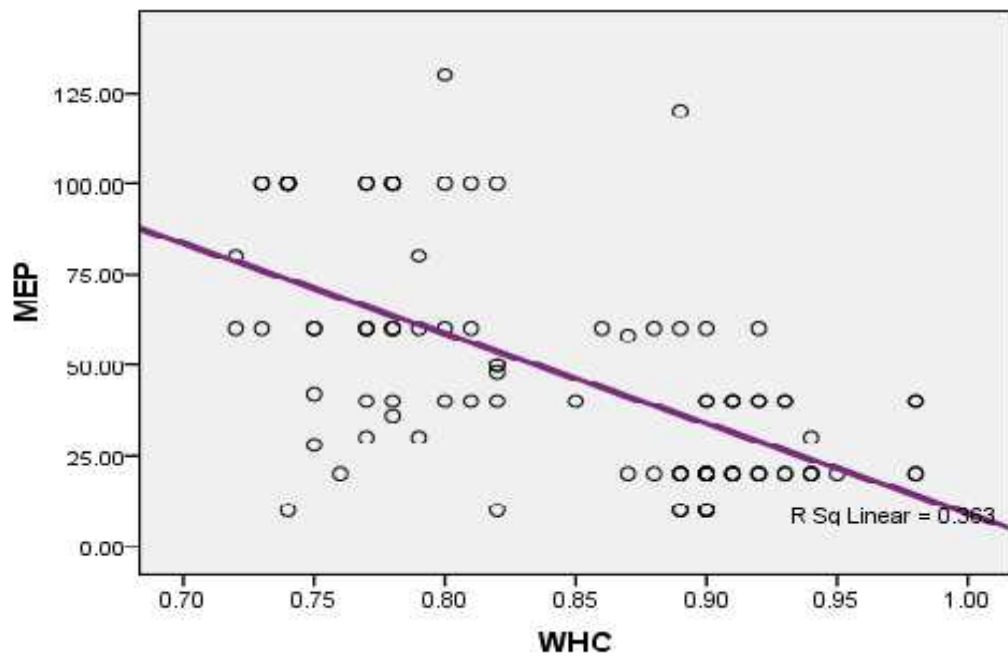
**FIGURE 34- Graph showing significant negative correlation between
MEP and BMI ($R^2= 0.444$)***



**FIGURE 35- Graph showing significant negative correlation between
MEP and WC ($R^2= 0.207$)***



**FIGURE 36- Graph showing significant negative correlation between
MEP and WHC ($R^2 = 0.363$)***



**FIGURE 37- Graph showing significant negative correlation between
MEP and WHtR ($R^2 = 0.242$)***

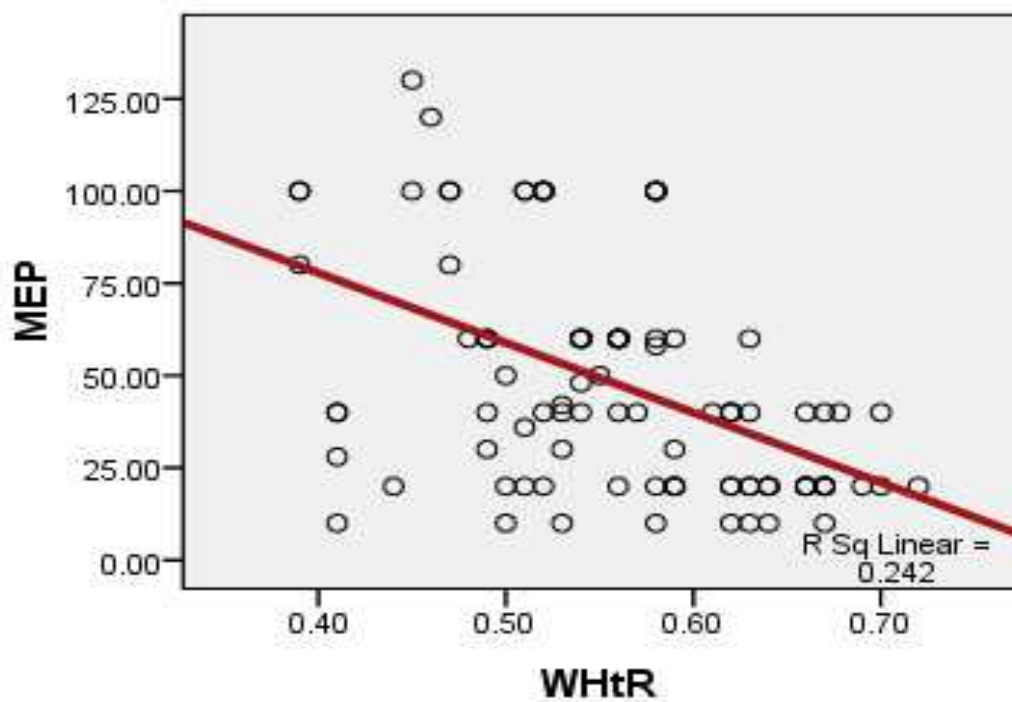
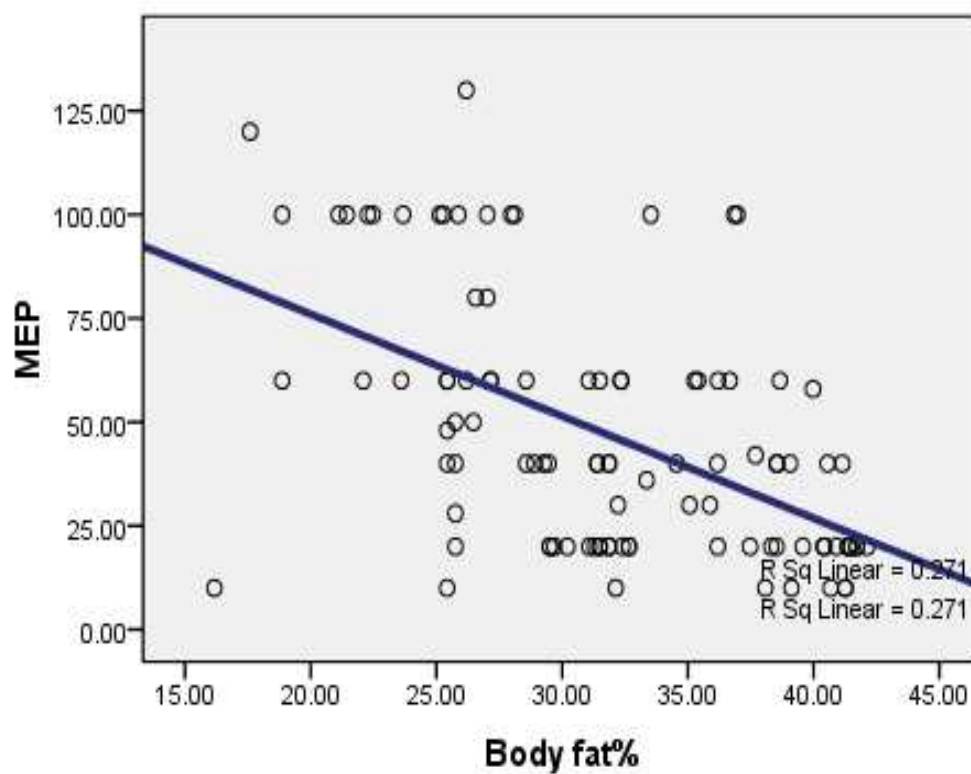


FIGURE 38- Graph showing significant negative correlation between MEP and Body fat % ($R^2 = 0.271$)*



DISCUSSION

The present study was undertaken on 50 obese student volunteers with BMI $\geq 30 \text{ kg/m}^2$ compared with age matched 50 non obese student volunteers with BMI 18.5 - 24.99 kg/m^2 from BLDEA's KCP Science College, Vijaypur applying necessary inclusion and exclusion criteria as mentioned earlier. They were subjected to detailed clinical examination.⁵⁰

Obesity is a state of excessive adiposity influenced by various neural, hormonal and metabolic factors. The common method of measurement of adiposity is determination of BMI. However, BMI does not adequately express the distribution of fat in different parts of the body nor does it directly measure body composition and muscle mass. Despite these limitations, National Institute of Health's Clinical guidelines for the identification, evaluation and treatment of overweight and obesity in adults defines obesity as BMI $\geq 30 \text{ kg/m}^2$. It has long been noted that there is a gender difference with respect to the location of fat where men tend to have more abdominal fat giving them the android pattern and women tend to have greater amount of gluteal fat giving them a gynoid pattern of fat distribution. The complications of obesity are associated with increased abdominal fat. Although, this fat distribution pattern is more common in men, both men and women show increased cardiovascular risk with greater abdominal fat.

From puberty onward, women have more fat than men and women also tend to gain more fat during adult life than men. Yet, women have a lower risk associated with any degree of extra fat. Increasing abdominal or visceral fat depots are affected by Corticosteroids and sympathetic activity. An increase in lower body or gluteal fat deposits is promoted by low concentration of Cortisol and high concentration of Estradiol, especially in women.

Obesity increases the pressure placed on the chest wall and thoracic cage which restricts pulmonary functions by decreasing respiratory compliance, increasing the work of breathing and restricting ventilation measured as decreased Total Lung Capacity, Forced Vital Capacity and Maximum Ventilatory Volume.¹¹

Anthropometric Parameters

Significant changes were observed in anthropometric parameters like weight, BMI, BSA, WC, HC, WC / HC, WC / Ht and Skin fold thickness among female and male study groups compared to that of respective control groups.

Physiological Parameters

Significant changes were observed in physiological parameters among study group compared to control group. Another important observation in the present study was significantly higher Systolic Blood Pressure in subjects with higher BMI. It is established that the sympathetic function increases with increase in adiposity and blood pressure of obese subjects are found to be higher compared to their non obese controls that contributes to the organ damage in young obese individuals. Therefore, one may think that increased sympathetic activity has contributed to changes in lung functions. However, increase in BP in obese subjects contributing to changes in lung function appears to be remote as the level of BP was much below hypertensive and prehypertensive range. Nevertheless, adipokines released from adipose tissue in obesity contribute to alteration in lung functions, which should be investigated in future studies.

Respiratory Parameters:

RR:

Significant changes were observed in respiratory rate among study group compared to control group. It may be due to increase in resistance as well as

mechanical load which in turn increases the work of breathing. Hence, obese individuals have an increased demand for ventilation.⁹

FVC:

No significant changes were observed in Forced Vital Capacity (FVC) among study group compared to control group. Dirceu Costa et al and Anupama et al observed similar results.^{29, 51}

In present study, adiposity markers (BMI, WC, WHR, WHtR and Body fat %) negatively correlated with FVC. Among them WC, WHtR and Body fat % were significant. Helala et al in their study observed significant negative correlation between WC and FVC in obese group. With an average of 1 cm increase in WC was associated with 20ml reduction in FVC.⁵²

Robert et al observed that obese men had more impairment of FVC with weight gain than obese women, estimated reduction of FVC was 17ml / Kg weight gain for men and 10ml / kg for women⁵³. Park et al in their study observed that with a 1% increase in body fat percentage, there was a decrease of FVC by 13 ml⁵⁴. According to Singh et al, overweight and obese children showed 13- 44% reduction in FVC depending upon their degree of obesity.⁵⁵

This may be due to the related increase in muscle strength and pulmonary functions initially increase in parallel with weight gain, although subsequent impairment of chest wall mobility results in reduced FVC. Obesity is the common cause for reduced FVC as it may interfere with movement of diaphragm and excursion of chest wall.⁴

Distribution of body fat also affects pulmonary function: the larger the waist measure or WHR were meaning the more fat distribution in upper body, the less the pulmonary function was. With an increasing abdominal fat deposition, thoracic

volume reduces and consequently, not only Vital Capacity but also power of respiratory muscles decrease deteriorating mechanical efficiency of thorax.

FEV1:

No significant changes were observed in Forced Expiratory Volume in 1st second among study group compared to control group. Dirceu Costa et al, Anupama et al and Yogesh Saxena et al showed similar results.^{28, 29, 50}

In present study, adiposity markers were negatively correlated with FEV1. Among them, WC, WHtR and Body Fat % were significant. Increase of WC by 1cm is associated with reduction in FEV1 by 13ml⁵¹. BMI was negatively correlated with FEV₁ but not significant. Helela et al study showed similar finding but was significant in obese group. According to Singh et al, overweight and obese children showed 20% to 46% reduction in FEV1 depending upon their degree of obesity⁵⁴. A reduction in absolute value of FEV1 indicates airway narrowing.

A possible mechanism could be large WC usually results in a high risk of developing visceral abdominal obesity. Levels of systemic inflammatory cells such as cytokine including Leptin, C-reactive protein and Fibrinogen are high in adipose cells. Systemic inflammation is considered to be associated with deteriorating pulmonary functions⁵⁶.

FEV1%:

We observed significant reduction in FEV_{1%} only in obese females compared to controls. No significant reduction in FEV_{1%} in obese males compared to controls. Mahajan et al and Saxena et al showed similar results that there was no significant reduction in FEV_{1%} in obese males. Costa et al and Kalpana et al showed significant reduction in FEV_{1%} only in obese females.^{29, 57}

In present study, BMI, WC, WHR and WHtR showed no significant and body fat % showed significant negative correlation with FEV1%. Similar findings were observed by Chen et al and Canoy et al^{5, 10}. There was no significant negative correlation of WC with FEV1%.

Women have less respiratory muscle strength. Therefore, lower dynamic compression will be produced. Hyperventilation caused by the effect of Progesterone on the bulbar respiratory neurons, airways and diaphragm may also explain these alterations. Reduction in FEV1% indicates the early part of small airway diseases.⁵⁸

PEFR:

The remarkable change was decrease in the values of PEFR in both female and male study groups as compared to their respective control groups.

Lower values of PEFR could be linked to mechanical effect of obesity on diaphragm and also because of fat deposition between the muscles and ribs that can lead to increase in metabolic demands and work of breathing⁵⁹⁻⁶².

As PEFR is more effort dependent and an index of expiratory airway resistance, it reflects the caliber of the bronchi and large bronchioles. The primary factors that affect PEFR are the strength of the expiratory muscles generating the force of contraction, elastic recoil pressure of the lungs and airway size.⁶³

Abdominal adiposity may influence pulmonary functions by restricting the descent of the diaphragm and limiting lung expansion as compared to overall adiposity which may compress the chest wall. In our study, PEFR was negatively correlated with adiposity markers. Sonu Ajmani, Shenoy et al and Mahajan et al observed similar findings. Viger et al showed strong negative correlation between BMI and PEFR.^{33, 34, 54}

MEP:

In our study, MEP was significantly reduced in both female and male study groups compared to their respective control groups. Choudary et al showed reduced MEP in females than males⁶⁴.

There was a significant negative correlation of MEP with adiposity markers. According to recent studies, the reduction in lung volume in obese people is not only directly related to the increase in the BMI, but it is also related to the distribution of body fat⁶⁵. Choudary et al in their study found that MEP was found to be positively correlated in both sexes.

Ochs-Balcom et al stated that abdominal adiposity contributes to impairment of lung function and is even more important than general adiposity markers such as weight and BMI⁵⁶. Once body fat is more peripherally distributed in women and more centrally distributed in men, pulmonary function is also affected according to gender as verified by some authors.⁶⁶

PREVENTIVE ASPECT¹

By virtue of following means, lung functions of an individual may be improved:

1. Regular exercise and physical activity.
2. Breathing exercises may help in strengthening the respiratory muscles.

CONCLUSION

The decline in FVC and FEV₁ in the present study is suggestive of obesity that significantly affects the function of large airways. Decrease in respiratory functions in obesity may be due to decrease in distensibility of chest wall or limited expansion of thoracic cavity.

These changes in parameters and respiratory effect could be possibly due to an increase in abdominal fat deposition and reduction in thoracic volume, which in turn results in airway obstruction.

The study demonstrated significant pulmonary dysfunction in obese individuals, thereby suggesting that obesity led to pulmonary impairment. Higher the obesity will lead to further decrease in pulmonary functions.

Based on the present study, we conclude that mechanical effect of obesity on diaphragm and also because of fat deposition between the muscles and ribs that can lead to increase in metabolic demands and work of breathing which adversely affect the pulmonary function parameters such as FVC, FEV₁, FEV₁/FVC, PEF_R and MEP and cause an restrictive pattern of lung diseases. Values of PEF_R were significantly reduced as compared to that of control group. We attribute this reduction in lung function test to respiratory muscle weakness.

Hence, we propose repeated recording of simple, non invasive and dynamic lung function tests like PEF_R in subjects. It may help to assess the efficiency of lungs in clinical practice.

Breathing exercises may help in strengthening the respiratory muscles and will improve the lung functions.

SUMMARY

A study was carried out to determine the effect of obesity on pulmonary functions. The study group consisted of 50 obese individuals. The control group comprised of 50 age matched non obese individuals.

Detailed anthropometric and physiological data were recorded. Pulmonary functions were recorded by using Computerized Spiro excel. The parameters pertinent to the study were Forced Vital Capacity (FVC), Forced Expiratory Volume in 1st sec (FEV1) , FEV1 % , Peak Expiratory Flow Rate (PEFR) and Maximum Expiratory Pressure (MEP).

Statistical analysis was done by calculating Mean \pm SD by using Student's t-test. Correlation between degree of obesity and pulmonary functions was done by Pearson's correlation.

Significant difference was observed in anthropometric and physiological parameters between the study and control groups.

There was a reduced pulmonary function in study group compared to control group. FEV1% was significantly reduced in female study group as compared to control. Values of PEFR and MEP were significantly reduced in study group as compared to control.

A negative correlation was observed between adiposity markers (BMI, WC, WHR, WHtR and Body Fat %) and pulmonary function. WC, WHtR and Body Fat % were significantly negatively correlated with FVC and FEV1. Body Fat % was significantly negatively correlated with FEV1%. Adiposity markers were significantly negatively correlated with pulmonary functions.

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ANNEXURE - 1

B.L.D.E.UNIVERSITY'S
SHRI.B.M.PATIL MEDICAL COLLEGE, BIJAPUR – 586103
INSTITUTIONAL ETHICAL COMMITTEE



INSTITUTIONAL ETHICAL CLEARANCE CERTIFICATE

The Ethical Committee of this college met on 13-11-2013 at 3-30pm scrutinize the synopsis of Postgraduate Students of this college from Ethical Clearance point of view. After scrutiny the following original/corrected and revised version synopsis of the Thesis has accorded Ethical Clearance.

Title "A Comparative Study of Pulmonary function tests in Obese and Nonobese students in the - Age group of 18-25 years"

Name of P.G. Student: Dr Sowmya Timmanra Koraddi
Department of Physiology

Name of Guide/Co-investigator: Dr Manjunatha Aithkole
Prof & HOD Dept of physiology

DR. TEJASWINI VALLABHA
CHAIRMAN
CHAIRMAN

Following documents were placed before E.C. for Scrutiny:
1) Copy of Synopsis/Research Project
2) Copy of informed consent form.
3) Any other relevant documents.

Institutional Ethical Committee
B.L.D.E.U's Shri B.M. Patil
Medical College, BIJAPUR-586103.

ANNEXURE - 2

**B. L. D. E. U SHRI B.M. PATIL MEDICAL COLLEGE, HOSPITAL AND
RESEARCH CENTRE, VIJAYPUR**

RESEARCH INFORMED CONSENT FORM

Title of the project

**A COMPARATIVE STUDY OF PULMONARY FUNCTION TESTS IN
OBESE & NON OBESE STUDENTS IN THE AGE GROUP OF 18-25 YEARS**

Principal investigator/ P.G. Guide's name: **DR. MANJUNATHA AITHALA MD**

PROF AND HEAD, DEPARTMENT OF PHYSIOLOGY

1. PURPOSE OF RESEARCH: I have been informed that this study will assess the pulmonary function tests in obese young students. This study will be useful academically as well as to find out pulmonary function tests in obese young students
2. PROCEDURE: I understand that, the procedure of the study will involve recording of various physiological & physical parameters. The procedure will not interfere with any of my physiological parameters and they are non invasive.
3. RISK AND DISCOMFORTS: I understand this study which tests the effect of obesity on pulmonary function will not cause any discomfort to me and do not involve any risk to my health.
4. BENEFITS: I understand that my participation in the study may not have a direct benefit to me but this may have a potential beneficial effect in the field of pulmonary function testing and obesity in future.
5. CONFIDENTIALITY: I understand that medical information produced by this study will become part of institutional records and will be subject to the

confidentiality and privacy regulation of the said institute. Information of a sensitive personal nature will not be a part of medical record, but will be stored in investigator's research file and identified only by a code number. The code key connecting name two numbers will be kept in a separate secured location.

If the data to be used for publication in the medical literature and for teaching purpose no names will be used and other identities such as photographs, audio and video tapes will be used only with my special written permission. I understand I may see the photographs and the video tapes and have the audio tapes before giving this permission.

6. REQUEST FOR MORE INFORMATION: I understand that I may ask more questions about the study at any time. Concerned researcher is available to answer my questions or concerns. I understand that I will be informed of any significant new findings discovered during the course of this study which might influence my continued participation. If during the study or later, I wish to discuss my participation in all concerns regarding this study with a person not directly involved, I am aware that the social worker of the hospital is available to talk with me. A copy of this consent form will be given to me to keep for careful readings.
7. REFUSAL OR WITHDRAWAL OF PARTICIPATION: I understand that my participation is voluntary and may refuse to participate or may withdraw my consent and discontinue participation in the study at any time without prejudice to my present or future care at this hospital. I also understand that researcher may terminate my participation in this study at any time after

she/he has explained the reasons for doing so and had helped arrange for my continued care by my physician or physical therapist if this is appropriate.

8. INJURY STATEMENT: I understand that in unlikely event of injury to me resulting directly from my participation in this study, if such injury is reported promptly, then medical treatment will be available to me, but no further compensation would be provided. I understand that by my agreement to participate in this study I am not waiving any of my legal rights.

I have explained to _____ (Name of subject) the purpose of the research, the procedure required and the possible risk and benefits to the best of my ability.

Investigator/PG (Guide)

Date

I confirm that _____ (Name of the P.G. Guide/ Chief researcher) has explained to me the purpose of research, the study procedure that I will undergo, and the possible risk and discomforts as well as benefits that I may experience. Alternative to my participation in the study, I have also been to give my consent form. Therefore, I agree to give consent to participate as a subject and this research project.

Participant

Date:

Signature of witness

Date:

Modified from Portney L.G. Watkins M.P., in Foundation of Clinical Research, Second Edition, New Jersey, Prentice Hall Health 2000.

ANNEXURE-3

QUESTIONNAIRE⁶⁶.

Respiratory Questionnaire to exclude any respiratory function deterioration.

1. Previous illness yes /no

Asthma, chronic bronchitis, pneumonia, tuberculosis, Pleurisy and heart problems.

2. Symptoms

a. **Cough:** yes/no

Do you usually cough first time in the morning?

Do you usually cough at other times during day or night?

Do you cough on most days for as much as three months of the year?

For how many years have you had this cough?

- 2years
- 2-5 years
- 5 years or more.

Do you cough more on any particular day of the week?

Do you cough during any particular season of the year?

b. **Sputum:** yes/no

Do you usually bring of phlegm, sputum or mucus from your chest first time in the morning?

Do you usually bring of phlegm or mucus from your chest at other times of the day or night?

Do you usually bring of phlegm, sputum or mucus from your chest on most days for as much as three months of the year?

From how many years have raised phlegm sputum or mucus from your chest?

- 2years
- 2-5 years
- 5 years or more.

a. **Wheezing:** yes/no

Does your breathing is wheezy?

Have you ever had a feeling of tightness in your chest?

Have you ever had attack of shortness of breath with wheezing?

At what age, did wheezing first occur?

How frequently does wheezing occur?

- Day or night
- A few times per week
- A few times per month
- Is it worse on any particular day of the week? which day

b. **Breathlessness:** yes/no

- Do you get short of breath when walking on ground?
- Do you get short of breath while walking up stairs?
- How many flights of stairs can you climb up without stopping?

≥ 1-2

≥ 1-3

More than 3

c. **Haemoptysis** yes/no

- Have your coughed blood from your chest?
- If yes, when was this last time happened?

B.L.D.E.U'S Shri B.M. Patil Medical College, Vijaypur

DEPARTMENT OF PHYSIOLOGY

CLINICAL PROFORMA

**Title: A COMPARATIVE STUDY OF PULMONARY FUNCTION TESTS IN
OBESE & NONOBESE STUDENTS IN THE AGE GROUP OF 18-25 YEARS**

Name:

Age/Sex:

Address & phone no:

General physical examination

PR:

BP:

MAP:

Wt:

Ht:

Temperature:

RR:

WC:

HC:

WC/HC:

WC/Ht:

Systemic Examination:

Cardiovascular system:

Respiratory system:

Central nervous system:

Per abdomen

PARAMETERS FOR STUDY

Pulmonary function tests:	I	II	III	Best
1. FVC (in ml)				
2. FEV1.				
3. FEV 1%.				
4. PEF (in L/min).				
5. MEP (in mmHg).				

Signature of PG student

Signature of Guide and HOD

ANNEXURE 4a: MASTER CHART - CONTROL GROUP

SI.	AGE	SEX	HT	WT	BMI	BSA	WC	HC	WHC	WHtR	Body fat %	PR	RR	SBP	DBP	MAP	FVC	FEV1	FEV1%	PEFR	MEP
1	18	F	150	45	20	1.38	72	90	0.8	0.45	36.84	80	16	120	80	93.33	2.74	2.46	89.78	400	100
2	18	F	150	45	20	1.38	72	92	0.78	0.41	36.18	78	17	110	70	83.33	2.62	2.54	96.95	400	40
3	21	F	150	50	22	1.46	85	105	0.8	0.56	29.27	70	12	110	70	83.33	3.93	3.63	92.37	400	40
4	19	F	161	51	19.67	1.5	78	99	0.78	0.51	33.36	69	14	110	70	83.33	2.96	2.87	96.96	450	36
5	18	F	155	56	23.3	1.54	81	98	0.82	0.49	28.57	68	14	100	70	80	2.21	2.13	96.38	300	40
6	21	F	167	57	20.43	1.62	78	98	0.79	0.49	35.07	68	14	120	80	93.33	1.98	1.94	97.98	300	30
7	20	F	145	45	21.4	1.34	80	103	0.77	0.53	35.88	71	16	110	70	83.33	1.61	1.4	97.9	400	30
8	23	F	168	56	20.54	1.66	89	109	0.81	0.52	34.55	72	14	110	70	83.33	2	1.87	93.5	500	40
9	18	F	150	46	20.44	1.4	85	110	0.77	0.56	32.34	68	13	110	70	83.33	3.11	2.87	92.28	500	60
10	18	F	155	45	18.73	1.42	85	115	0.73	0.54	32.34	70	12	110	70	83.33	2.33	2.19	93.99	400	60
11	20	F	150	50	22.22	1.46	85	110	0.77	0.56	36.66	71	13	100	70	80	3.37	2.99	88.72	500	60
12	21	F	164	60	22.3	1.68	90	120	0.75	0.54	36.2	73	12	120	80	93.33	3.41	3.33	97.65	400	60
13	18	F	167	60	21.51	1.7	91	116	0.78	0.54	35.27	72	12	120	80	93.33	2.27	1.98	92.09	500	60
14	18	F	155	50	20.81	1.48	91	117	0.77	0.58	33.52	70	12	120	80	93.33	3.19	2.92	96.31	600	100
15	23	F	168	62	21.9	1.72	90	120	0.75	0.54	31.06	74	12	128	86	100	3.01	2.77	92.03	400	60
16	21	F	165	62	22.77	1.7	91	117	0.77	0.58	36.97	70	13	130	70	90	2.96	2.64	89.19	600	100
17	25	F	165	62	22.77	1.7	86	116	0.74	0.52	23.67	74	12	120	70	86.66	2.92	2.91	99.66	600	100
18	25	F	178	66	20.83	1.84	85	110	0.77	0.56	35.4	90	13	120	80	93.33	2.37	2.16	95.26	500	60
19	19	M	150	45	20	1.38	75	98	0.76	0.44	25.76	72	12	110	70	83.33	2.86	2.12	74.13	200	20
20	21	M	160	50	19.53	1.52	90	110	0.8	0.56	27.16	73	15	100	70	80	2.58	2.57	99.61	400	60
21	20	M	167	57	20.43	1.62	88	100	0.88	0.49	25.43	72	12	110	70	83.33	1.25	1.17	93.6	400	60
22	21	M	155	53	22.06	1.5	90	105	0.85	0.54	25.43	72	12	110	70	83.33	2.19	2.07	94.52	400	40
23	19	M	150	45	20	1.38	72	90	0.8	0.45	26.19	80	20	120	80	93.33	1.27	1.2	94.49	400	130
24	22	M	174	60	21.79	1.82	81	98	0.82	0.54	25.43	80	20	120	80	93.33	2.21	1.59	100	300	48
25	19	M	162	59	22.48	1.6	67	89	0.75	0.41	25.76	81	17	110	70	83.33	2.8	2.52	90	400	28
26	18	M	164	64	23.79	1.68	68	88	0.77	0.41	25.76	79	15	120	80	93.33	3.11	3.08	99.04	500	40
27	22	M	170	72	24.22	1.8	67	90	0.74	0.41	16.17	81	17	120	80	83.33	3.15	2.61	90.62	350	10
28	20	M	168	56	19.84	1.62	70	89	0.78	0.39	25.13	69	15	100	70	80	1.37	1.01	85.47	400	100

29	21	M	183	73	21.75	1.96	85	103	0.82	0.53	25.43	79	16	120	80	93.33	1.47	1.3	89.47	300	10
30	20	M	173	70	23.38	1.84	84	103	0.81	0.49	18.87	68	17	110	70	83.33	1.7	1.65	97.06	400	60
31	18	M	163	57	21.45	1.6	87	106	0.82	0.5	26.47	84	20	120	80	96.66	2.49	2.32	93.17	400	50
32	18	M	160	55	21.48	1.58	89	108	0.82	0.55	25.76	73	13	110	70	83.33	1.78	1.69	98.26	600	50
33	18	M	146	40	18.76	1.3	85	108	0.78	0.58	23.6	70	12	110	70	83.33	1.82	1.47	80.77	500	60
34	18	M	166	56	20.32	1.64	80	110	0.72	0.48	26.19	70	12	110	70	83.33	1.78	1.67	94.89	500	60
35	20	M	174	62	20.47	1.76	86	108	0.79	0.49	25.43	72	12	110	70	83.33	3.29	2.64	80.24	500	60
36	22	M	176	70	22.59	1.86	99	115	0.86	0.56	28.57	72	13	110	70	83.33	2.04	2.01	98.53	500	60
37	22	M	178	61	19.25	1.8	85	103	0.82	0.47	27.97	72	12	110	70	83.33	2.72	2.08	76.47	500	100
38	19	M	170	58	20.06	1.7	88	118	0.74	0.51	28.11	72	12	120	80	83.33	3.09	2.03	65.7	600	100
39	25	M	172	65	21.97	1.8	90	122	0.73	0.52	25.87	73	13	120	70	86.66	3.05	2.9	98	600	100
40	18	M	155	51	21.22	1.5	91	116	0.78	0.58	27.02	72	13	120	70	86.66	3.9	3.4	93.2	500	100
41	20	M	165	52	19.1	1.6	86	116	0.74	0.52	22.47	70	14	110	70	83.33	2.8	2.6	93	600	100
42	19	M	180	64	19.75	1.86	85	117	0.72	0.47	27.02	71	13	100	70	80	4	3.3	82.5	500	80
43	24	M	155	62	21.6	1.5	74	99	0.74	0.47	22.28	80	12	120	60	80	2.5	2.5	99.4	600	100
44	22	M	168	53	18.7	1.6	66	81	0.81	0.39	21.11	76	12	130	80	96.66	3.1	2.6	86	500	100
45	23	M	165	54	19.8	1.58	77	86	0.89	0.46	17.59	84	12	130	90	103.33	3.6	3.1	94.3	600	120
46	21	M	163	52	19.6	1.5	65	82	0.79	0.39	26.56	76	12	120	70	86.66	3.2	3.2	92	500	80
47	22	M	160	48	18.7	1.52	91	116	0.78	0.54	22.09	67	13	100	70	80	3.4	3.38	99.7	500	60
48	20	M	164	63	22.6	1.68	90	122	0.73	0.52	18.87	79	14	120	80	93.33	3	3	92.6	600	100
49	18	M	170	65	22.49	1.78	91	116	0.78	0.58	21.44	73	13	136	80	98.66	3.3	2.9	89	500	100
50	23	M	165	60	22.03	1.7	88	118	0.74	0.51	25.28	76	12	126	86	99.33	2.4	2	89	600	100

ANNEXURE 4b: MASTER CHART - STUDY GROUP

Sl.	AGE	SEX	HT	WT	BMI	BSA	WC	HC	WHC	WHtR	Body fat %	PR	RR	SBP	DBP	MAP	FVC	FEV1	FEV1%	PEFR	MEP
1	19	F	152	70	30.29	1.68	98	109	0.89	0.64	36.1886	84	20	130	80	96.66	2	1.98	99	320	20
2	18	F	167	86	30.83	1.94	105	112	0.93	0.62	42.13	79	20	120	80	93.33	1.74	1.65	94.83	350	20
3	19	F	162	80	30.48	1.86	104	116	0.89	0.64	38.08	98	24	140	80	100	1.74	1.73	99.43	300	10
4	20	F	160	77	30.07	1.8	85	112	0.75	0.53	37.69	77	19	120	80	93.33	2.11	1.68	79.62	350	42
5	20	F	156	74	30.4	1.74	99	111	0.89	0.63	41.26	77	20	120	70	86.66	2.63	2.18	97.76	300	10
6	19	F	162	80	30.14	1.84	95	108	0.87	0.58	39.98	81	21	110	70	80	2.06	1.64	79.61	350	58
7	20	F	159	76	30.06	1.78	100	110	0.9	0.62	41.26	78	24	130	80	96.66	2.33	2.15	92.27	350	10
8	19	F	155	75	31.21	1.74	90	100	0.9	0.58	39.12	88	25	120	80	93.33	2.33	2.17	93.13	350	10
9	21	F	148	66	30.13	1.6	100	110	0.9	0.67	40.68	71	20	120	80	93.33	2.02	1.83	90.59	400	10
10	20	F	149	67	30.17	1.6	106	117	0.9	0.62	41.14	91	22	140	90	106.66	1.06	0.58	54.72	400	40
11	23	F	148	68	31.04	1.62	104	110	0.94	0.59	41.48	91	21	130	90	103.33	2.41	2.12	87.97	400	20
12	19	F	158	75	30.04	1.78	105	115	0.91	0.66	40.39	93	20	130	90	103.33	2.47	2.25	91.09	400	20
13	22	F	144	63	30.38	1.58	100	110	0.9	0.67	40.91	88	20	130	90	103.33	1.94	1.87	96.39	300	20
14	22	F	144	63	30.38	1.58	100	110	0.9	0.67	41.37	90	21	130	90	103.33	2.35	1.96	83.4	300	20
15	21	F	152	70	30.29	1.7	105	115	0.91	0.66	41.37	88	20	130	90	103.33	2.35	1.93	82.13	300	20
16	22	F	165	75	27.54	1.84	119	129	0.92	0.72	41.71	90	22	140	80	100	2.88	2.83	98.26	400	20
17	20	F	150	75	33.33	1.7	105	115	0.91	0.7	40.56	88	23	130	90	103.33	1.96	1.8	91.84	200	40
18	20	F	150	70	31.11	1.66	106	115	0.92	0.7	40.44	90	24	130	90	103.33	2.27	2.1	92.51	300	20
19	20	F	154	72	30.35	1.7	100	110	0.9	0.59	38.66	86	20	140	90	106.66	2.33	2.29	98.28	200	60
20	20	F	143	62	30.31	1.5	105	112	0.93	0.62	38.53	84	20	120	80	103.33	2.27	1.97	86.78	100	40
21	19	F	150	68	30.22	1.6	103	111	0.92	0.63	37.47	87	21	130	80	106.66	1.72	1.21	70.35	250	20
22	19	F	142	61	30.25	1.48	104	116	0.89	0.64	38.32	90	25	140	90	106.66	1.49	1.22	81.88	200	20
23	19	F	162	79	30.1	1.84	103	113	0.91	0.63	38.55	88	22	130	90	103.33	3.07	2.63	85.67	250	40
24	20	F	154	70	29.51	1.68	106	117	0.9	0.62	39.57	86	21	130	90	103.33	2.19	2	91.32	300	20
25	21	F	152	70	30.29	1.66	108	110	0.98	0.67	39.06	88	22	130	90	103.33	2.76	2.73	98.91	250	40
26	21	F	154	71	29.93	1.7	104	110	0.94	0.59	38.53	90	24	130	80	106.66	2.8	2.49	88.93	200	20
27	21	F	165	85	31.22	1.92	105	115	0.91	0.66	41.71	89	24	140	90	106.66	1.59	0.49	30.82	200	20
28	18	M	167	85	30.47	1.94	98	108	0.9	0.58	29.51	85	21	150	70	96.66	2.53	2.09	82.61	500	20

29	18	M	159	76	30.06	1.78	91	98	0.92	0.57	29.46	80	20	160	90	113.33	3.6	3.57	99.17	500	40
30	18	M	169	86	30.11	1.96	100	110	0.9	0.59	31.08	81	21	140	90	106.66	2.92	1.75	59.93	500	20
31	19	M	161	78	30.09	1.8	103	111	0.92	0.63	27.15	88	22	130	80	96.66	2.74	2.66	97.08	500	60
32	21	M	162	85	32.38	1.9	103	113	0.91	0.63	29.52	84	23	140	80	100	2.19	2.15	98.17	350	20
33	18	M	158	75	30.04	1.76	84	91	0.92	0.53	28.89	84	22	140	90	106.66	2.33	2.33	100	500	40
34	20	M	162	80	30.48	1.84	99	108	0.91	0.61	31.4	84	23	130	90	103.33	2.6	2.58	99.23	500	40
35	21	M	170	93	32.17	2.04	106	117	0.9	0.62	31.86	81	25	160	90	113.33	3	2.86	95.33	400	40
36	20	M	160	77	30.07	1.8	108	110	0.98	0.67	31.86	84	21	130	90	103.33	1.43	1.2	83.92	300	20
37	22	M	176	94	30.34	2.06	104	110	0.94	0.59	32.22	91	22	140	90	106.66	3.19	3.15	98.75	400	30
38	20	M	150	72	32	1.72	98	109	0.89	0.56	31.49	89	20	130	90	103.33	3.97	3.25	81.86	450	60
39	22	M	160	85	33.2	1.88	108	110	0.98	0.67	32.66	89	20	130	90	103.33	2.43	2.01	82.72	300	20
40	20	M	150	70	31.11	1.68	100	110	0.9	0.66	31.49	84	22	130	90	103.33	2.51	1.97	78.49	300	20
41	21	M	170	80	27.68	1.92	110	115	0.95	0.64	32.4	90	24	140	90	106.66	3.78	3.76	99.47	300	20
42	23	M	159	76	30.06	1.82	110	118	0.93	0.69	31.3	90	25	140	90	106.66	3	2.53	84.33	300	20
43	25	M	165	85	31.22	1.94	112	118	0.94	0.67	30.21	92	23	140	90	106.66	2.23	1.91	85.65	300	20
44	25	M	165	80	29.38	1.86	112	120	0.93	0.678	31.4	90	22	140	90	106.66	2.66	2.67	100.38	200	40
45	24	M	168	85	30.11	1.86	88	100	0.88	0.52	31.86	94	23	120	80	93.33	2.43	2.2	90.5	200	20
46	25	M	168	79	28	1.9	87	99	0.87	0.51	32.66	88	22	130	70	90	3.9	3	96.5	300	20
47	23	M	153	70	29.9	1.68	102	104	0.98	0.66	31.86	70	17	130	80	96.66	2.35	2.17	96	200	40
48	25	M	154	70	29.51	1.62	87	96	0.9	0.56	29.69	71	17	120	80	93.33	2.5	2.2	91.9	200	20
49	23	M	162	60	31	1.64	81	91	0.89	0.5	31.49	80	20	130	80	96.66	3.54	3.19	90.1	100	20
50	20	M	163	80	30.11	1.72	82	91	0.9	0.5	32.13	74	18	130	80	96.66	3.31	3.17	95.77	200	10