A STUDY ON SERUM C-REACTIVE PROTEIN LEVEL IN OBESITY

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BRANCH – I

COIMBATORE MEDICAL COLLEGE

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CERTIFICATE

Certified that this is the bonafide dissertation done by **Dr. T. Dhanalakshmi** and submitted in partial fulfillment of the requirements for the Degree of **M.D. General Medicine**, Branch I of **The Tamilnadu DR. M.G.R. Medical University Chennai.**

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DECLARATION

I solemnly declare that the dissertation titled **"A STUDY ON SERUM C-REACTIVE PROTEIN LEVEL IN OBESITY"** was done by me from AUGUST 2013 to JUNE 2014 under the guidance and supervision of Professor **Dr. S. MANOHARAN M.D.,**

This dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of the requirement for the award of MD Degree in General Medicine (Branch I).

Place : Coimbatore **Dr. T. Dhanalakshmi**

Date :

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DATE : **Dr. T. Dhanalakshmi**

PLACE :

AIM OF THE STUDY

Purpose of the Project

Study of C reactive protein level in obese person with BMI of more than 25 $kg/m^{2.}$

Objective

To test whether overweight and obesity are associated with low-grade systemic inflammation as measured by serum C-reactive protein (CRP) level.

To study the c reactive protein level in obesity using measures of body mass index.

To study CRP level in obese women and men.

Data Collection and the Source

Patient attending OPD ward is examined BMI. Body weight and height were measured using standardized procedures. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters and used as an indicator of body fat.

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ABBREVIATIONS

VPC	Ventricular premature complexes

APC	Atrial premature complexes
VT	Ventricular tachycardia
ST	Supraventricular tachycardia 1
OSA	Obstructive Sleep Apnea
OHS	Obesity hypoventilation syndrome
FFA	Free fatty acids
IGT	Impaired glucose tolerance
IR	Insulin resistance
DM	Diabetes mellitus
GH	Growth hormone
MS	Metabolic syndrome
SHT	Systemic hypertension
Bp	Blood pressure
SHT	System hyper tension A
IHD	Ischemic heart disease
SCAT	Subcutaneous adipose tissue
PCOD	Poly cystic ovary disease
OA	Osteoarthritis
Positive (HR)	High Risk - >3ml/lr
Positive (MR)	Moderate Risk- 1-3ml/lr
Negative	Low risk - <1mg/lr

INTRODUCTION

INTRODUCTION

Obesity is nothing but, a chronic common disease characterized by excess body fat. It develops gradually and often persists throughout life. As a preventable cause of death in the US, obesity is second only to smoking. Like other chronic condition, such as diabetes or hypertension, obesity worsens when strategies applied for weight reduction are withdrawn. If it is not treated, obesity emerges as a potent comorbid risk factor.

Nowadays obesity is emerging as an important health trouble in India. About 22 million Indians are obese especially abdominally obese. The nutrition foundation of India (NFI) study showed that 32.3 percent of middle class males and 50 percent of middle class females in New Delhi were obese. Overweight and obesity are escalating in Asian Indians especially those living in urban areas. Particularly vulnerable subgroups include children, women, and people living in suburban and rural areas. Body composition characteristic of Asian Indians (high body fat, abdominal adip17osity, truncal subcutaneous fat, and fatty liver) predispose them to develop insulin resistance and the metabolic syndrome early in life.

Obesity carries chronic health issues. The most seriousness of obesity may be completely understood by – **Epidemiologic transmission theory.** It traces the human history based on mortality.

I phase – femine and pestilence

II phase – receding pandemics

It is the later part of 19th and early years of 20th century.

III phase – middle of 20th century

It is mortality by cancer

In this phase death by cardiac illness are decreasing because of preventing medicine and advanced treatment.

IV phase – presenting era. Obesity and physical inactivity

Previous improvement in cardiovascular mortality is worsened by obesity hyperendemicity.

Over ten years 97 million people are in the classification of overweight and obesity. Nowadays it is over than 140 million.

Obesity influences staggering medical management and economical lose. It increases the death rate in SHT, DM, and IHD.

Excess abdominal adiposity directly influences the SHT, DM, IR, IHD and dyslipidemia. It is directly related to duration of obesity. In obese population, marked increase in inflammatory markers released by dysfunctional adipocytes. The main inflammatory markers are CRP, IL6, TNFα.

These markers predict the future outcome of the obese people. Maturation of preadipocyte to dysfunctional adipocyte was affected by chronic energy imbalance.

The role of inflammation in atherosclerosis proves that inflammatory mediators are taking part in endothelial injury plaque formation and unstable plaque. When the stimulus for production of inflammatory markers (energy imbalance and physical inactivity) are stopped, inflammatory drops dramatically to the normal.

In our study BMI was taken for obesity measurement and serum CRP level is used to measure the severity of the obesity.

REVIEW OF LITERATURE

DEFINITION

It is a state of excess adipose tissue mass [1]. Body weights are distributed continuously in human population. Medically definite difference between lean and obese is difficult.

PREVALENCE

Around 1.2 billion people in the world are overweight and at least 300 million of them are affected by obese. According to the WHO, obesity is one of the ten most preventable health risks. National health and nutritional examination services (NHANES) shows that American

adult with obesity (BMI >30) increased 14.5 % to 33.9%. The prevalence of obesity is more among poor and women, and blacks and Hispanics. Incidence and prevalence also rising among childrens.

SOCIETAL IMPACT

Obesity and its consequences increase economic costs of obesity hospital[2]. Visits due to obesity increased by 88% from 1988 to 1994 [3]. Hospital visit have been found to be 38% higher in obese population than normal weight persons [4]. Obesity has a major health and economic cost in world.

MORTALITY RATES AND OBESITY

Obesity and mortality rates depend on the following factors: 1. smoking 2. pre-existing disease and weight fluctuation 3. Physical inactivity 4. the lack of precision and accuracy in the use of BMI as an estimate of adiposity [5].

OBESITY MEASUREMENTS

Three anthropometric measures are used to evaluate the obesity. They are weight, height, and waist circumference.

Body mass index (BMI)

It is measured as weight in kg / height in m^2, or weight in pounds / height in $inches^2$ X 703. It is helpful to grade and it risks. It is commonly used indices in world wide. It gives and calculates approximately of body fat and its risk. Lower BMI has been proposed for Asian pacific region because these people at risk for glucose and lipid abnormalities in lower BMI.

Table 1. Classification of obesity with BMI level

	BMI (kilogram/ $meter^2$)	Obesity Class	Risk of Disease
Under weight	<18.5		
Healthy weight	18.5-24.9		
Overweight	25.0-29.9		Increased
Obesity	30.0-34.9	I	High
Obesity	35.0-39.9	II	Very high
Extreme obesity	≥40	III	Extremely high

BMI is lower for Asian Indians than western population.

Normal BMI : 18.0 – 22.9 kilogram/$meter^2$

Overweight : 23.0 – 24.9 kilogram/$meter^2$

Obesity : >25 kg/m^2. [6,7]

Ideal body weight (IBW)

Ideal body weight = 22.5 x height ($meters^2$)

Over weight – > 10% of IBW

Underweight - < 20% of IBW

Obesity – > 20% of IBW

Thickness of Skin-fold

It is measured by special pair of callipers over the triceps, biceps, subscapular and suprailiac region.

Normal triceps thickness of skin-fold

Adult males- 12.5 mm

Adult females – 16.5 mm

Broca's index

Height (inches) = weight (kg)

Height (cm) – 100 = desired body weight (kg)

Ratio of Waist-hip

It is helpful to measure the prognosis of obesity. Waist measurement of narrowest segment between ribcage and iliac crest. Hip-maximal measurement of the hip in gluteal region. Apple-shaped obesity is abdominal obesity, which is seen with type2 diabetesmelitus and ischaemic heart diseases, hyperlipidaemia and insulin resistance.

Waist to hip ratio	Obesity type	Prognosis
0.8 or <	Pear-shaped obesity	Good
0.9 or >	Apple-shaped obesity	poor

Circumference of Waist

It is useful to consider the prediction in obese population.

Waist circumference and risk of morbidity

Gender	Moderate	High
Male gender	> 94 cm (37”)	> 102 cm (40”)
Female gender	> 80 cm (32”)	> 88 cm (35”)

Other measure to quantify obesity include,

- Densitometry (under water weighing)
- CT or MRI
- Electrical impedance
- Dual energy X-ray Absorbtiometry (DEXA) is used to measure the percentage of body fat.

OBESITY PANDEMIC

Eating habits

Polices on production of food and the accessibility of high fat “fast food” or high glycemic foods are factors that contribute to the prevalence of obesity in society. The increase affordability for junk food leads to high energy consumption.

We need not to spend most of our time in hunting and gathering food as our ancestors did; instead we can get the food any corner like fast food stalls, restaurants. WHO recently reports that more mortality caused by obesity and physical inactivity. This could be prevented by healthy food and physical exercise[8].

Sedentary lifestyle

A change in food production and marketing leads to physical inactivity. Most of the jobs are sedentary [9]. Physical inactivity is associated with a risk of obesity and obesity and IHD [10]. Transport by walking is now replaced by vehicles. This is an important in sedentary lifestyle across all over the world. England health survey in 1998 reported that 70-80 percent of the persons lead sedentary live [11].

Psychological stress

Part of life starting from school education to job or business stress till end of life. Most of the case studies shows that job stress and cardiovascular disease (CVD) mortality among male [12]. Job stress also leads to physical inactivity, eating behaviours that may be contributed to BMI [13].

SECONDARY OBESITY

Obesity secondary to some pathology constitutes a minority of patients. This subset of patients includes various genetic syndromes like prader Willi syndrome, Lawrence Moon Biedl Syndrome etc.; hypothalamic lesions; various endocrinopathies like Cushing's syndrome, Hypothyroidism etc; and drug induced.

Homeostasis of body weight

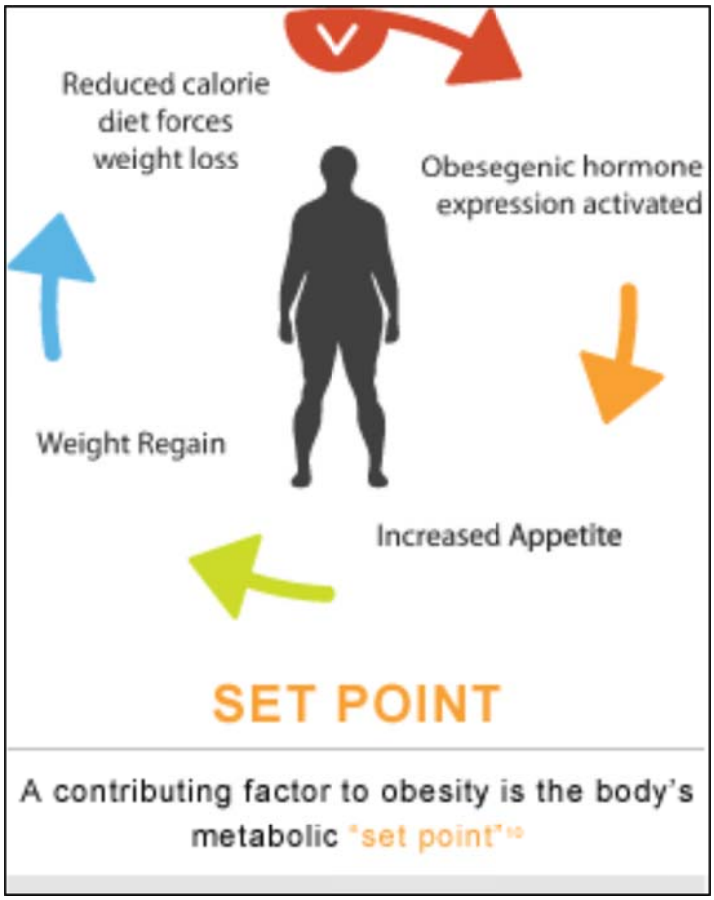


Fig. 1 Homeostasis of body weight

Physiological mechanism by which homeostasis of the body fat and energy stores are maintained. This mechanism is seen both in normal and obese individuals. When body weight is lost due to calorie deficit, homeostatic mechanisms come into play that results in restoration of weight by increasing appetite and decreasing energy expenditure; the opposite happens when body weight is gained. These homeostatic mechanisms act till the weight is brought to the **'set-point weight'** or the 'defended weight' of the particular individual.

Body weight or energy homeostasis occurs between the hypothalamus, adipose tissue, gastrointestinal tract, liver, pancreas and the nervous system, more specifically the sympathetic nervous system. Information about energy stores and energy needs is conveyed from the periphery to the hypothalamus.

The hypothalamus responds by sending signals resulting in hunger and energy conservation or satiety and energy expenditure, as the situation may demand. The satiety centre is located in the medical hypothalamus and includes the arcute nucleus. The hunger centre is present in the lateral hypothalamus. Lesions of the medial hypothalamus cause hyperphagia and obesity; whereas lesions of the lateral hypothalamus results in anorexia and cachexia.

Leptin secreted from the adipose tissue acts as a gauge for energy stores, with increased levels seen in obesity and decreased levels in seen in cachexia [14]. In states of calorie excess, leptin acts on the leptin receptors in hypothalamus to decreases appetite and to increase energy expenditure via the POMC (pro-opiomelanocortin) neurons.

The sympathetic nervous system acts via the β3 receptor in the skeletal muscles and brown adipose tissue, resulting in non-shivering (non-energy/adaptive) thermogenesis [15]. Signals are also conveyed from the gastrointestinal tract and the pancreas. Feeding and gastrointestinal

distension results in release of cholecystokinin, peptide YY, GLP-1 and insulin which decrease appetite; whereas when energy stores are low ghrelin is released from the stomach resulting in increased appetite [16-18].

Sex steroids also contribute to this homeostasis, with estrogen favoring subcutaneous fat accumulation and androgens favoring central fat accumulation [19]. This homeostatic mechanism of energy storage maybe modulated in certain conditions like fever, where cytokines acting on the hypothalamus may cause anorexia.

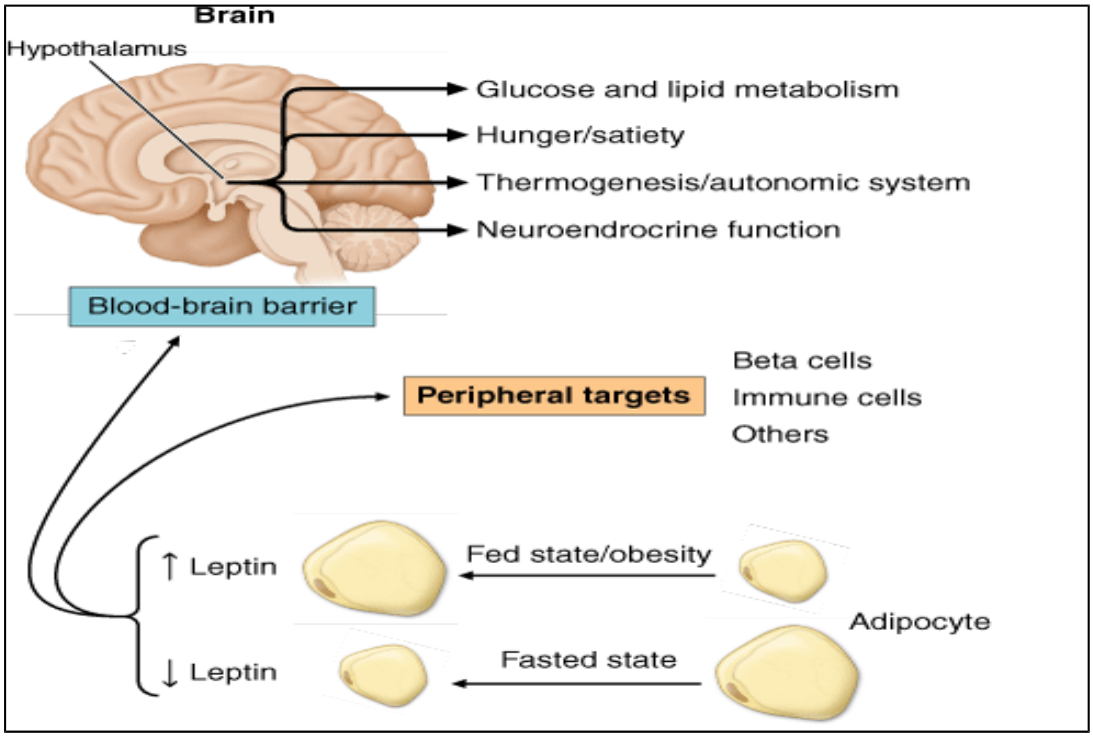


Fig. 2. Leptin and its regulated mechanism

OTHER HORMONES INVOLVED IN OBESITY

Neuropeptide Y (N-Y)

Leptin function and its effects on energy intake are the role of neuropeptide Y [20]. For food intake N-Y is the important stimulator. It is related to leptin dysfunction and enegy imbalance. Increase level of N-Y seen in SHT and DM who are obese [21].

Cortisol

It has very high metabolic action. It mobilizes FFA from stored TGL and takes part in glucose synthesis from amino acids and fat and has role in protein metabolism [22]. In Cushing disease people increase level of Cortisol is seen and it contribute visceral and abdominal adipocytes [23,24.]

Ghrelin

I t is a GH secretagogue mainly present in stomach; it was stimulated by fasting state and inhibited by feeding. The effect was controlled by central inhibition of leptin and other cytokines. The signal is transmitted by a neural pathway (vagus). It also mediated by hypothalamic N-Y. It also take part in GH regulation [25-27] .

Norepinephrine (NE), Serotonin, Interleukin-6 and TNFα

These factors have shown to have influence on energy intake and contribute to weight gain. NE increases and decreases the food intake by acting on specific receptor. if it acts on alpha-1 receptors in the PVN it inhibits food intake. In contrast, when NE acts on alpha-2 receptors in the PVN it inhibits food intake.

Serotonin action also in same fashion. It acts on the $5HT_{1A}$ and 5-HT_{2C} receptor [28]. Action on $5HT_{1A}$ stimulates, and 5-HT_{2C} action inhibits food intake. IL-6 is a inflammatory marker highly associated with high level of BMI. It modulate lipid metabolism in adipose tissue [29]. TNF alpha stimulate IL-6 synthesis from adipose tissue P80 receptors of adipose tissue. It also an inflammatory markers.

Testosterone

Testosterone decreases the LPL activity and stimulates lipolysis in men, in females it was overcome by female sexsteroid like estrogen. Testosterone influence s the leptin mRNA [30].

The growth hormone/insulin like growth factor axis .it is one of the regulatory mechanisms. It is take parts in lipid metabolism and synthesis of protein and prevent proteolysis by IGF-1. **thyrotropin stimulating hormone (TSH)** has main action on BMI and obesity [33].

TSH and BMI were positively related and TSH level correlated with leptin level.

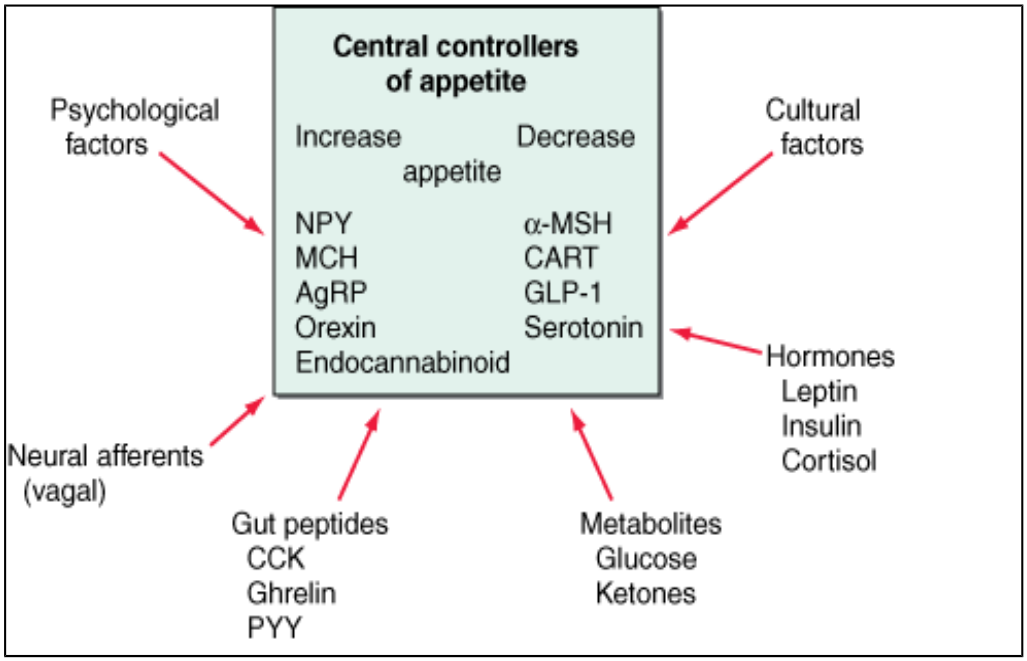


Fig. 3. The factors regulating food intake and energy expenditure

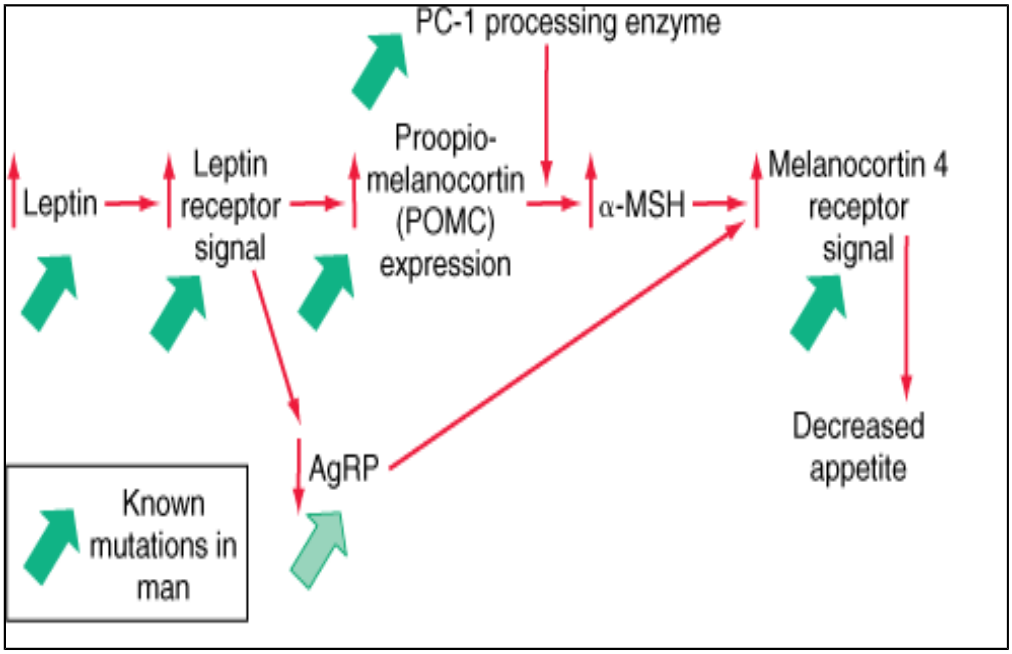


Fig. 4. Leptin action in various pathways

BASIC PATHOLOGY IN OBESITY

1. Hypothalamic dysfunction
2. Adipose tissue dysfunction
3. Alterations in gut signals-role of gut microbiome
4. Ineffectiveness of adaptive thermogenesis

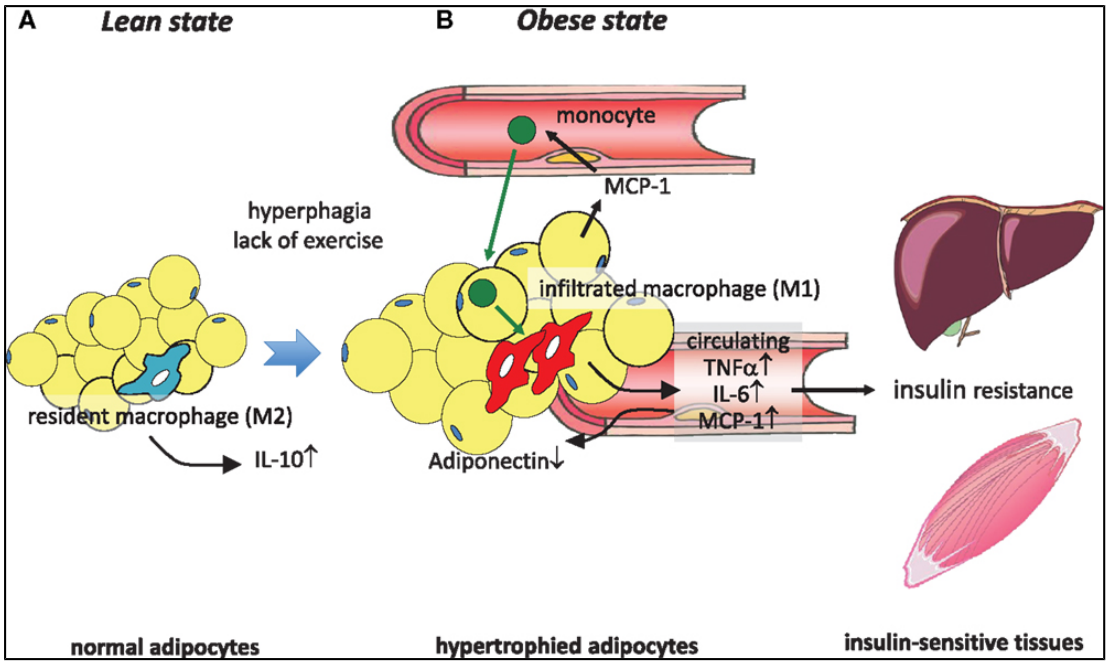


Fig. 5. Physiological dysregulation in obesity

1. Hypothalamic dysfunction

Hypothalamic dysfunction can result in loss of homeostasis resulting in cachexia or obesity. Experimental studies in mice have shown that high fat diet results in hypothalamic inflammation [34]. This inflammation results in the destruction of the POMC neurons and induces leptin resistance in the hypothalamus.

Janus kinase-signal was play role in Leptin signalling and transcription pathway activation was inhibited by inflammation. It results

in leptin resistance. So there is increasing body weight that leads to elevation in 'set-point-weight' [35]. These change in the difficulty in maintaining weight following a weight loss program.

Leptin decreases appetite by a number of mechanisms like direct action on the hypothalamus, increased POMC activity, increased GLP-1 secretion from the gut and increasing activity in the nucleus accumbens.

The nucleus acumens are described mainly in the context of drug addiction, where it acts as a 'reward' centre. Food intake can stimulate the nucleus accumbenes resulting in a sense of satiety ('reward'). This is known as the hedonic value of food. With leptin this sense of reward occurs even without food intake. Thus in leptin resistance of obesity satiety does not occur.

2. Adipose tissue dysfunction

Adipose tissue dysfunction causes not only the complications of obesity but may have a role in the pathogenesis of obesity. The adipose tissue inflammation arising out of adipose tissue hypoxia. Hypoxia occurs due to the expanding adipose tissue mass, as a result the adipose tissue is stripped of its vascular supply since the capillaries are unable to perfuse to such a large extent.

Hypoxia results in inflammation and the inflammatory cytokines result in macrophage infiltration and further inflammation [36]. The inflammation can cause leptin resistance and the resultant increase in weight. The inflamed adipose tissue is dysfunctional and insulin resistant with increased release of adiponection. Adiponection is anti-inflammatory and is protective against atherosclerotic cardiovascular disease.

3. Role of gut microbiome

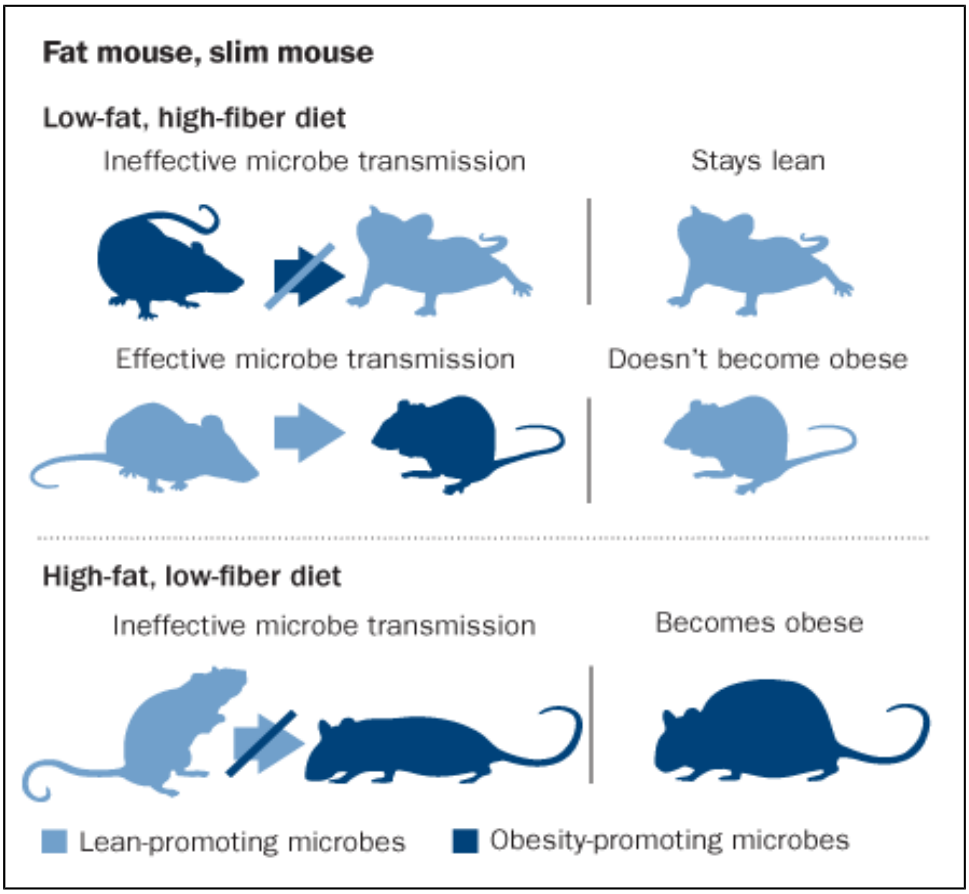


Fig.6 Role of gut microbiota

The revised view of adipose tissue inflammation, the gut microbiota has been implicated. The symbiotic gut microorganisms in the

human intestine (the microbiome) are a metabolically active organ that is now known to have a pathogenetic role in obesity. Germ free mice are known to be resistant to diet induced obesity, insulin resistance and diet induced obesity.

FIAF is suppressed in the gut mucosa by the microbiota. Gut microbiota promotes the lipoprotein lipase activity with increased uptake of FFA. Gut microbiota can also affect the secretion of gut hormones like peptide YY and GLP-1, thereby affecting gut transit time, energy harvest and satiety [37]. The gut microbiome increases the efficiency of fat absorption, decreases fat oxidation and interferes with the signalling of the satiety hormones from the gut.

Adipocytes, macrophages and cells of the adaptive immunity like T lymphocytes for producing adipose tissue inflammation. T lymphocytes first infiltrate the adipose tissue. The infiltrating T lymphocytes (T_H1) result in the phenotypic switch of macrophages in the adipose tissue, from the anti-inflammatory M2 macrophages to the pro-inflammatory M1 macrophages via the release of IFN γ and IL-17 from the T lymphocytes.

High fat diet results in temporary damage to the intestinal villi and increases the capillary density resulting in the increased absorption of antigens from the gut which can be inflammatory. The gut microbiome is

known to be increased in obesity. The lippolysaccharide of the gut bacteria is thus absorbed along with fat and gains access to the adipose tissue via chylomicrons, and thus inflammation is initiated in the adipose tissue [38].

IMPAIRMENT OF ADAPTIVE THERMOGENESIS

Hypothalamus controls energy expenditure is via the sympathetic nervous system where adaptive thermogenesis results in the dissipation of energy as heat rather than being stored. This is brought about by the uncoupling protein 1 in the inner mitochondrial membrane of the brown adipose tissue (BAT).

Brown adipose tissue developed as a protective mechanism for adaptive thermogenis. But, the use of PET scanning for the detection of malignancy changed this belief. During PET imaging, BAT was found in the supraclavicular, infrascapular, cervical, regions and also around the great vessels and adrenals.

Brown and white adipocytes, mytocytes, osteoblasts and chondrocytes all develop from the mesenchymal stem cell (MSC). BAT and white adipose tissue (WAT) are morphologically different. BAT adipocytes are smaller, polygonal, have a central nucleus and have multiple lipid droplets.

They have a brownish hue to the rich mitochondrial content (the mitochondrial content is comparable to the cardiac myocytes). The abundance of mitochondria which contains respiratory chain cytochrome enzymes with iron as a co-factor gives it a brown appearance. White adipocytes are rounded, larger with a single lipid droplet, have a peripherally located nucleus and have low mitochondrial content.

Recent evidence shows that the expression of *Myf5* gene in brown adipocytes mean that they share a common lineage with myocytes rather than with adipocytes. PGC1α increases the synthesis of UCP1 in the BAT by stimulating its promoter. Increased release of irisin from the skeletal muscle during exercise results in the browning of the white adipocytes, making them 'beige' or 'brite'.

This may be one of the mechanisms how exercise causes weight loss and improved insulin resistance. Another transcriptional factor FGF21 has been implicated in the browning of the white adipose tissue [39].

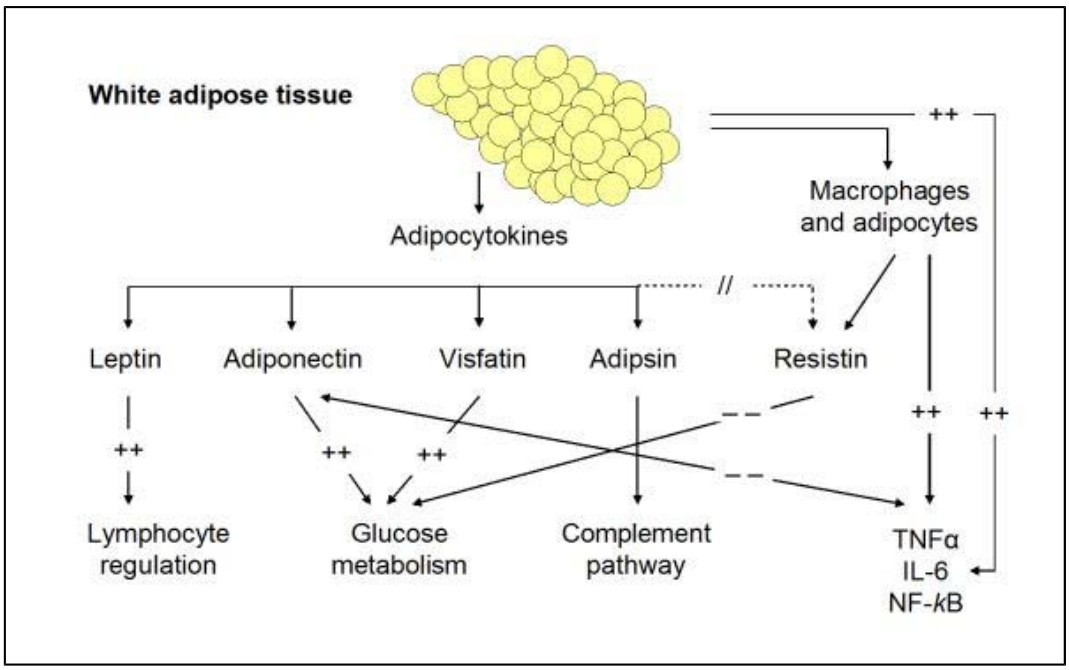


Fig. 7. White adipose tissue and its effects

NATRIURETIC PEPTIDES AND ADAPTIVE THERMOGENESIS

Natriuretic peptides are increased in heart failure; in fact they seem to play a role in the pathogenesis of cardiac cachexia. Natriuretic peptides cause natriuresis and dieresis, decrease preload and afterload in heart failure. Natriuretic peptides have been found to cause lipolysis, fatty acid oxidation and browning of the white adipose tissue. In obesity, the natriuretic peptide level is low. This has been proposed to be due to increased natriuretic peptide clearance receptor in the adipose tissue.

Natriuretic peptides also increase mitochondrial oxidative metabolism and fat metabolism and fat metabolism in the skeletal muscles [40.] Thus the high levels of natriureic peptides in heart failure may

explain the cardiac cachexia and the levels of natriuretic peptides in obesity may explain the high fat mass.

Thus there is a homeostatic mechanism for maintain the body weight within a set-point. However, in obesity there is perturbation of this inter-organ crosstalk in the form of hypothalamic and adipose tissue dysfunction; defective adaptive thermogenesis and increased gut microbiota.

Nonexercise activity thermogenesis (NEAT)

The new concept of thermogenesis is nonexercise activity thermogenesis (NEAT). It is linked to obese population. It accompanies physical activities like daily living, fidgeting, spontaneous muscle contraction, and posture maintaining. NEAT contributes 2-3 of daily energy expenditure by overfeeding. The fat storage seen in positive energy individual is predicted by NEA. This mechanism is unknown [1].

ETIOLOGY OF OBESITY

Obesity is seen in families, and the heritability of body weight is like height. Inheritance is not Mendelian. Not only genetic factors influence the obesity. The environment shared by the same family members eating habits and sedentary life also takes parts in obesity. Adoptees resemble their biologic than adoptive parents, indicates genetic

influences. There is one theory called hypothesis of **thirfty genotype.** In this genes that are useful in our ancestors survival are attenuated by environments in where food is available plenty in all time.

Specific genetic syndromes

Obesity caused by number of mutations in genome. *ob* gene mutation represented a major role. The ob/ob mouse develops overweight, ER, and hyperphagia. *ob* gene is the peptide leptin, a name derived from the Greek root *leptos*, meaning thin. The *ob* gene is present in humans. The most prominent of these is hypogonadotropic hypogonadism, which is reversed by leptin replacement in the leptin-deficient subset.

There is no evidence that mutations in the leptin or leptin receptor genes play a role in obesity. The absence of Proopio-melnanocortin causes secondary adrenal insufficiency due to absence of ACTH, pale skin and red hair due to absence of alpha-MSH. A-MSH binds to the type 4 melanocortin receptor (MC4R), a key hypothalamic receptor that inhibits eating.

Syndromes of obesity

At least 12 genetic loci have been identified, and most of the encoded proteins from two multiprotein complexes that are involved in ciliary function and microtubule-based intracellular transport. Mutations might disrupt leptin receptor trafficking in key hypothalamic neurons, causing leptin resistance. Syndromes of obesity is shown in Table 3 [1].

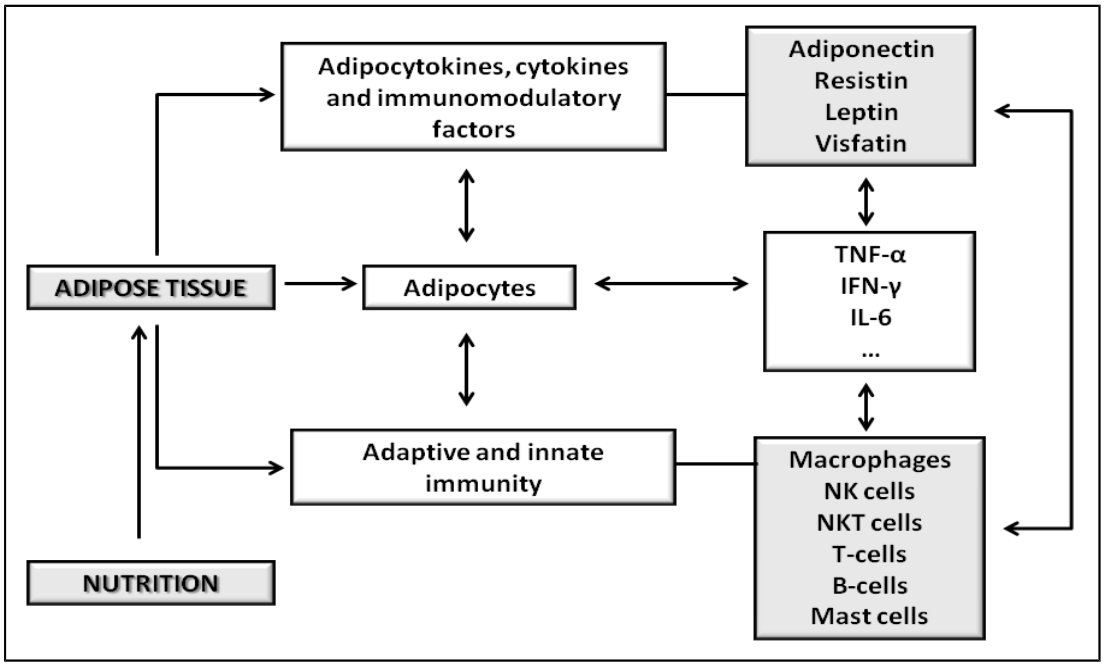


Fig. 8. Mechanism of adipose tissue and inflammation

Truncal and Abdominal Adiposity

Truncal obesity is an important cardiovascular risk factor. This can be due to excess abdominal adipose tissue, both intra-abdominal adipose tissues (IAAT) and subcutaneous adipose tissue (SCAT). Visceral adiposity is a metabolically active organ, strongly related to IR.

Adipocytes of visceral fat have a different histology and biology than subcutaneous tissue fat. It is contains large insulin resistant adipocytes, has a developed vasculaturity with the infiltration of inflammatory macrocytes.

Asians have more truncal SCAT. It was measured by skin fold thickness. high insulin resistance in a BMI and body fat matched Indian men could be explained by more truncal skinfolds.

Fatty liver is considered to be ‘innocuous’. In absence of alcohol intake of greater than 20 gram/day, it is called as non-alcoholic fatty liver disease (NAFLD).

Intra-myocellulor lipids (IMCL) are important in insulin resistance and measured non-invasively using proton magnetic resonance spectroscopy. Intra-myocellulor lipids is located in the mitochondria. The soleus muscle IMCL content is an important marker of Whole-body insulin sensitivity. Increase Intra-myocellulor lipids content is seen in soleus muscle in Indians with viceral obesity.

Soleus muscle Intra-myocellulor lipids content and C-reactive protein levels were higher in T2DM patients. Soleus muscle Intra-myocellulor lipids composition not correlated with IR and CRP levels. Acanthosis nigricans is a important marker for DM type 2 in Indians.

Truncal is seen in cushing diseases and lipodystrophy of HIV patients treated by ART. It is highly related with IR and metabolic syndrome.

Adipocyte Size

Visceral adipose tissue has 4 times the number of glucocorticoid receptors as SEAT adipose tissue with similar Kd values. Lipoprotein lipase activity in SCAT was 2 to 4 folds lower compared to visceral adipose tissue. Correlation between Lipoprotein lipase activity and glucocorticoid binding was seen.

In subcutaneous adipose tissue, whereas LPL activity was higher in omental than in subcutaneous adipose tissue in both male and female. These results show that there are differences between deposits of fat in their response to glucocorticoids and display wide differences in their biochemical properties.

The gluteal region fat cells from women has high insulin receptor binding and high rates of non-insulin-stimulated and maximally insulin-stimulated rates OF GLUCOSE transport and glucose metabolism and leptin mRNA is markedly expressed in abdominal subcutaneous adipocytes than with visceral adipocytes. There is inverse correlation between adipocytes, PPAR-gamma and Bodymassindex.

SCAT produces low interleukin-6 (IL-6) and coriticoterone and high TNF-alpha in comparison to mesenteric adipose tissue. Since PPAR-

gamma is involved in adipocytes development and insulin sensitivity and has a negative control on TNF-alpha synthesis indicating the indicate that complex local network of process in the regulation of adipocytes accumulation, metabolism and function. The functional differences in between various depots of adipose tissue indicate that they have role in MS.

Insulin resistance is the state where a given concentration of insulin gives below normal response on target organs like skeletal muscle, adipose tissue and liver. Stimulation of the rennin system in the vessels and cardio vascular system produces elevated level of Angiotensin II. It has atherogenic effects.

Elevated abdominal fat is a important marker of arterial hypertension. There is a correlation between weight and Bp, with 70 percent of hypertension in male and 60 percent in female being directly attribute to excess fat. Visceral fat is thought to be a metabolically active tissue, highly related to insulin sensitivity.

Adipose tissue is known to secrete a large number of adipokines such as leptin, adiponection, PAI-1, TNF-alpha, IL-6, angiotensin II, FFA and resistin. These factors contribute the process of inflammation endothelial malfunction, atherogenic and hypertension. Sympathetic system activity also contributes to the effect of hypertension.

In abdominal obesity there is deficiency of adiponection, “good guy adipokine”. Adiponection reduces the insulin resistance. This exerts antibiotic, anti-inflammatory and antiatherogenic effects. Thus there is an inverse relationship of adiponection and mean arterial pressure so, adiponection was a marker of hypertension.

INFLAMMATORY BIOMARKERS, ADIPOSE TISSUE, AND OBESITY

leptin and adiponectin as well as a number of other cytokines that are involved in inflammation [41].

With the onset of obesity, secretions of adipokines from adipose tissue change. The number and localization of immune, vascular, and structural cells within adipose tissue change with weight gain and loss. In addition, the location of the fat depot alters expression and secretion of adipokines with visceral fat generally having a higher secretion of adipokines.

Adipose tissue can also be present in organs including the heart, liver, and kidney, as well as in bone marrow and lungs. The ramifications of fat depots in these organs are not entirely known, but they appear to increase an individual’s health risk for morbidity and mortality.

Obesity results in rise of these pro-inflammatory adipokines as well as fall in the anti-inflammatory adipokine [42, 43]. It is the raised levels

of circulating hormones and adipocytokines in the body that has lead, obesity being as a state of chronic low-grade inflammation, further linking it to disease etiology [44, 45].

The process of inflammation is complex as it involves many cell types and molecules performing various functions, these include initiation, amplification, attenuation and resolution. Inflammation is assessed by biomarkers, with some released in part by adipose tissue, or by other tissues, with their release being stimulated by signalling molecules from adipose tissue. Many inflammatory molecules have multiple functions and contribute to both the increase and decrease of inflammation at various time points [46].

Commonly assessed biomarkers for inflammation include leptin, TNF-alpha, interleukin (IL)-6, adiponectin and serum C-reactive protein (CRP). Leptin, TNFα, IL-6 and CRP are known as pro-inflammatory cytokines as they induce inflammation whereas adiponectin is known as an anti-inflammatory cytokine as it acts to help reduce inflammatory pathways [47].

C-REACTIVE PROTEIN (CRP)

CRP PHYSIOLOGY

C-reactive protein (CRP) is a calcium-dependent ligand-binding plasma protein in the pentraxin family. The human CRP molecule is

synthesized by hepatocytes and is composed of 5 identical nonglycosylated polypeptide subunits, with 206 amino acid each [48].

Serum CRP binds with highest affinity to phosphocholine residues, though it binds with a number of other autologous and extrinsic ligands; it aggregates or precipitates the cellular, particular, or molecular structure of these ligands. Serum C-reactive protein was first detected by Tillet and Francis in 1930 with their identification of a substance in the serum of acutely infected by pneumococcal pneumonia inspection.

It forms PPT with polysaccharide capsule of St. Pneumonia. This indicates body chemical reaction to inflammation. It is not unique to pneumonia, also seen in other acute conditions [49]. From those days onwards serum CRP has been used as a screening device for occult inflammation, as a marker of disease activity, and as a diagnostic tool.

While systemic inflammation and the inflammatory cascade may have sources other than atherogenesis, increased recognition of the inflammatory component of the atherosclerotic process. It used as a marker of endothelial injury and atherogenesis.

In healthy individuals, levels of CRP are normally less than 10 milligram/dl: the median concentration of CRP is 0.8 milligram/litre; the 90^{th} centile is 3.0 milligram/litre; and the 99^{th} centile is 10 milligram/litre (Shine, 1981). In acute phase stimulus, however, CRP values can increase

from less than 50 microgram/dl to over 500 milligram/litre, a 10,000-fold increase.

Serum CRP level is not altered in other conditions of health and disease, circulating CRP level is directly the rate of synthesis [50]. This synthesis rate is a direct reflection of the pathological processes stimulating CRP production. The stimuli for synthesis disappear, serum CRP level drops at the rate of CRP clearance. CRP values tend to increase with age and this change is presumed to reflect an increase in subclinical pathologies (Hutchinson, 2000).

The people have constant CRP level for each person. Serum CRP indicates inflammatory process and tissue injury. In acute-state serum CRP values indicate no seasonal or diurnal variation and are not affected by eating habits. Liver failure is the only concurrent pathology that impairs serum CRP production, and few drugs reduce serum CRP levels unless they act upon the underlying pathology that provides the acute-phase stimulus for CRP production. Levels of serum CRP closely associated with inflammatory markers. It also predicts severity of coronary insult [51]. So the CRP levels are highly associated with cardiovascular diseases and its prognosis.

The American Heart Association and Centres for Disease Control released a Scientific Statement in 2003 indicating that inflammatory

markers used to classify risk assessment to identify persons. From this basis they are advised for lipid-lowering, anti-platelet, or cardiac drug therapies. Also included were those for whom there should be an increased emphasis on therapeutic lifestyle changes.

Cesari, et al studies shows serum levels of the inflammatory markers CRP, IL-6, and TNF-α are well correlated with strok, CAD, SHT and CHF. These 3 inflammatory markers indicate prognosis of the disease. Among this IL-6 being the strongest predictor for CHF and IHD. Because of the economic ease of testing for CRP, however, it maintains its importance the most suitable inflammatory marker for wide-spread testing.

Providing considerable evidence that CRP can offer striking predictive significance for cardiovascular disease[52]. The AHA/CDC shows that their dose response relationship between the level of serum CRP and incident of IHD. CRP is currently the best candidate for measures of inflammation.

Individuals with CRP levels of <1 are categorized as being at a low risk of cardiovascular disease, those with levels 1.0 to 3.0 are categorized as being at an average risk of cardiovascular disease, and individuals with CRP levels greater than 3 are categorized as being at a high risk of cardiovascular disease [53].

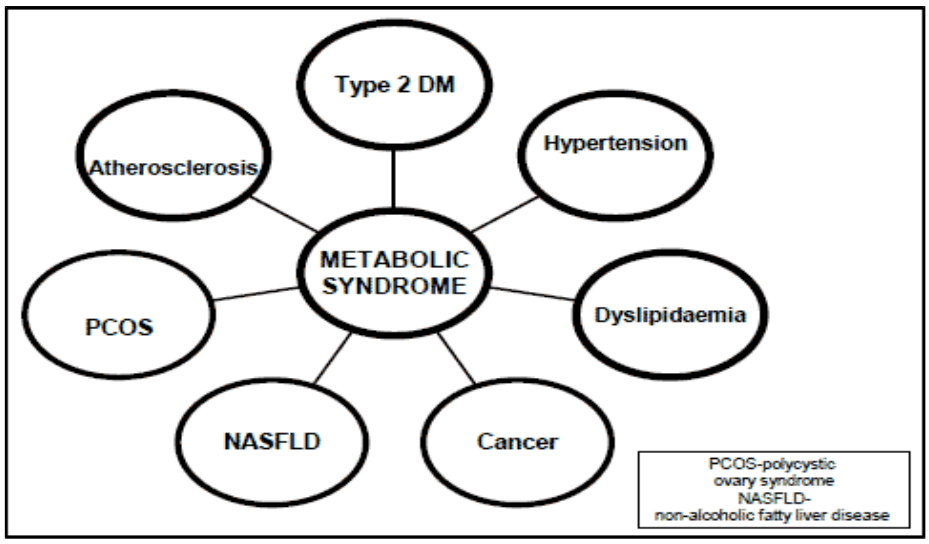


Fig. 9. Consequences of obesity

Cardiovascular System	**Respiratory SYSTEM**
Systemic hypertension	Breathlessness
CHF , CAD	OSP
Right heart failure	Hypoventilation syndrome
Varicose veins of legs	Pickwickian syndrome
Embolism of Pulmonary vessels	Bronchial Asthma
Endocrine system	**Gastrointestinal SYSTEM**
Metabolic syndrome [ms]	GERD
Type 2 DM	NAFLD
LIPID Abnormalities	Gall stones
PCOD	Carcinoma of colon
Musculoskeletal SYSTEM	**Genitourinary system**
High uricacid level	Stress incontinence of urine
Physical inactivity	Obesity-reated glomerulopathy
OA (knees and hips)	Male Hypogonadism
LBA	Cancer of Breastand uterus
Entrapment neuropathy	PIH
Psychological disorder	**Central nervous system**
Depression	Cerebrovascular accident
Body image disturbance	Intracranial hypertension
Social stigma	Meralgia paresthetica

Table 4. Obesity-related Organ Systems Review

PATHOLOGIC CONSEQUENCES OF OBESITY

There is strong association between obesity and CAD, DM type 2, and chronic illness; there is substantial increase in obesity associated diseases in future.

CARDIOVASCULAR DISORDER, SYSTEMIC, HYPERTENSION AND CCF

This occur due to rise in blood volume, which increase left ventricular filling, a rise in stroke volume and an increase in cardiac out increase in cardiac output is caused by structural change, and is not affected by heart rate (HR) and HR remains unchanged. The obesity affects IHD by arrhythmias, PVD and CVA, SCD. Atherogenesis forms by a progression of plaque formation and rupture. It leads to thrombus formation and fibrosis.

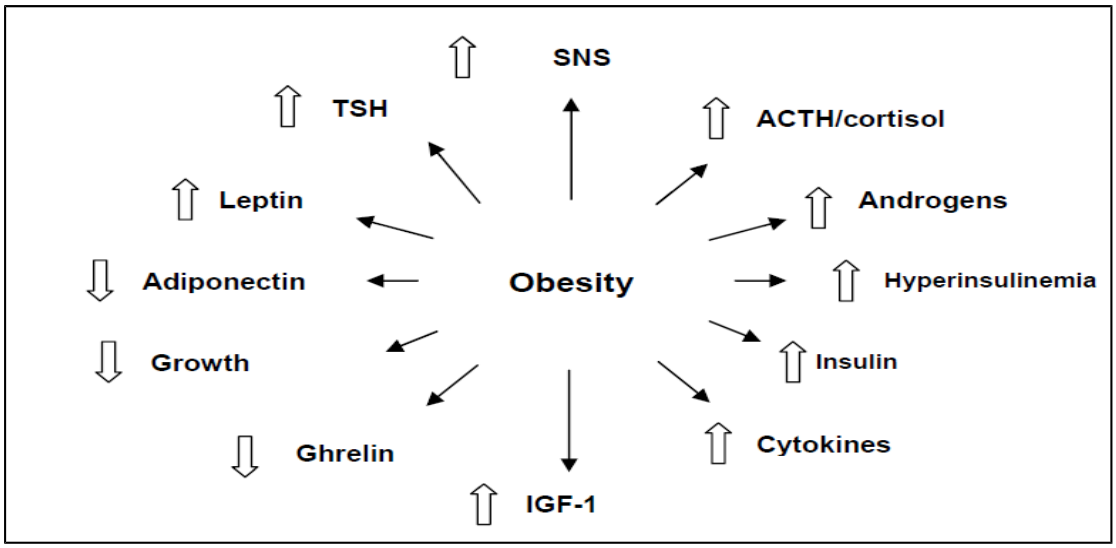


Fig. 10 Related hormones in Obese

Arrhythmias

Ventricular ectopics present 10 times greater in obese persons than normal weight persons. The common arrhythmias are sinus arrhythmia, PVC, APC, VT, and SVT.

CVA and Peripheral Vascular Disease (PVD)

In obesity there is increased risk of ischemia and atherosclerosis obesity is associated with increased intimal thickening of carotid arteries, and thus, overweight alone can increase the risk for stroke [54].

Sudden Death (SCD)

The study of Framingham Heart states that the sudden death is a predictive for overweight and obese male and female.

Gallbladder Disorders

In obese populations gallstones are having a high prevalence rate. The formation of gallstone needs 2 conditions: (1) secretion of supersaturated bile and stasis in gallbladder. Obesity affects bile content and motility of gallbladder. (2) Bile is more saturated with high cholesterol. This can cause gallbladder motor dysfunction 1. Recent studies show that leptin may cause the association between obesity and gallstones. The gallstones incidence in obese women is estimated at 2.6 per 100 person-years [55].

Sleep Disorders

There is strong associations are between obesity and OSA. Weight gain and loss can affect the severity of OSA depends on body weight. Approx 10-15 percent of severe obese persons develop OSA. In this CO^2 retention occur during wakefulness. This is called obesity-hypoventilation syndrome. The probable mechanism includes genetics in ventilatory responses and the metabolic effects of obesity, respiratory control. Cardiovascular or metabolic effects also involved in obesity on respiratory control [56].

Obesity and liver disease (NAFLD) Non-alcoholic fatty liver disease

FFA is thus increased in obesity due to increased release from the adipocytes and decreased oxidation. FFA impairs insulin signalling by two mechanisms. One is via the increased accumulation of the lipid intermediaries like diacylglycerol and ceramides. The other mechanism is by stimulating the NFκβ pathway via the TLR4.

However FFA cannot bind TLR4 directly, and recently it has been shown that the hepatokine fetuin A is an endogenous ligand for TLR4 and presents FFA to TLR4.

Fetuin A also decreases the release of adiponection in obesity. Cytokines released from the inflamed adipocytes stimulates the NFκβ

which impairs insulin signalling. Thus the above mentioned factors result to insulin resistance in obese people.

The gut micrboime inhibits the FIAF, a natural inhibitor of lipoprotein lipase. In absence of FIAF, the lipoprotein lipase activity is increased in obesity resulting in increased fatty acid. Increased fatty acid thus absorbed is delivered to the liver via portal veins and to the adipose tissue via chylomicrons.

Recently it was shown that IL-1β released during adipose expandability and thus favours ectopic fat deposition in the liver.

The fatty liver releases an inflammatory cytokine fetuin-A which has a pathogenic role in insulin resistance. Feutin A decreases the release of anti-inflammatory adipokine adiponection from the adipocyte. Adipose tissue dysfunction causes NAFLD and NAFLD further worsens the adipose tissue dysfunction.

Insulin resistance and NAFLD are associated with macrovascular complications like atherosclerotic cardiovascular disease. NAFLD may be related with the lipid abnormalities of MS characterized by a small dense LDL-C, with elevated triglycerides (TGL) and decreased in HDL.

ENDOTHELIAL DYSFUNCTION

Vascular endothelial dysfunction is a main cardiovascular risk predictor and is usually starter for the process of arterial atherosclerosis. The endothelium is one of the largest organs of the body and actively produces a huge variety of molecules which can act as agonists and antagonists nullifying their effects.

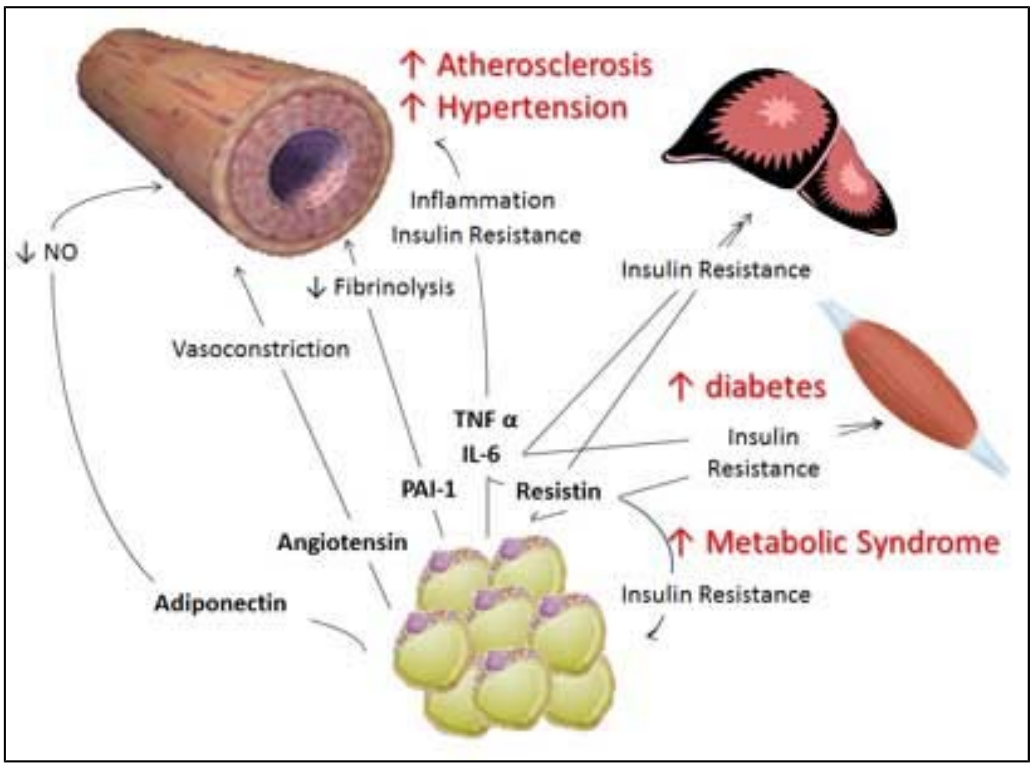


Fig. 12. Adipokines on endothelium

Eendothelial cell act as a receptor – It contains effect or structure which senses various physical or chemical stimus that present innerside of the vessel.

Table 5. Equilibrium of endothelial function
1. Endothelium –derived relaxing factors(EDRF) (Vasodilators)

Substances:

- NO - Nitric Oxide
- Endothelium –derived hyperpolarizing factor
- Bradykinin
- Adrenomadullin
- Atrial natriuretic peptide

Their overall effects:

- Vasodilatation
- Anti-thrombotic
- Anti-inflammatory
- Anti-oxidative stress

2. Endothelium – derived vasoconstrictors

Substances:

- Endothelin-1
- Angiotensin II
- Thromboxane A2
- Oxidant radicals
- Prostaglandin H2

Their overall effects:

- Vasoconstriction
- Increased thrombosis
- Increased inflammation
- Increased oxidative stress

"Endothelial dysfunction" (En D) means there is loss in normal physiology of endothelium. Normal properties of endothelium were dilation of the vessels increased in fibrinolysis and prevention of platelet aggregation.

Vascular endothelial cells are unable to find their ability to maintain the balance between the opposite forces and this result in dysregulated endothelium and disturbed cardiovascular homeostasis. Lipids and leucocytes (Monocytes & T-Lymphocytes) invade such an endothelium inciting an inflammatory response and appearance of fatty streaks.

Inflammation and Endothermal Dysfunction

Chronic low grade inflammation is associated with IR & En D. C-reactive Protein (CRP), IL-6, TNF-α and increased PAI-1 have been shown to be elevated in IR state. Microalbuminuria has also considered as urinary marker indicative of ch0ronic inflammation and is increased in MetS.

All of these are surrogate markers of En D. in patients with abdominal obesity, increased adipocyte expression of fatty acids and cytokines that include IR may be the underlying mechanism linking inflammation and MetS. Factors such as over nutrition, physical inactivity and ageing cause cytokine hypersecretion and eventually lead to IR & DM in persons with genetic or / and metabolic profile that predispose to these conditions.

Perivascular adipose tissue, endothelium and MetS

Perivaascular PAT (adipose tissue) means, Fatty tissue around the arteries large (aorta, coronaries) or small (vascular beds of the mesentry, smooth muscle, renal and fatty tissue depots). Perivaascular adipose tissue consists of fibroblasts, adipocytes, mast and stem cells, and neurons.

Most of the study shows that the Perivaascular adipose tissue (PAT) secretes a variety of adipokines like adiponectin and leptin among others which contribute regulation of vascular function. With CAD production of adiponectinis reduced in epicardial adipose tissue. Adipokines such as FFA, TNF-alpha and adiponectin are important in insulin resistance and inflammation.

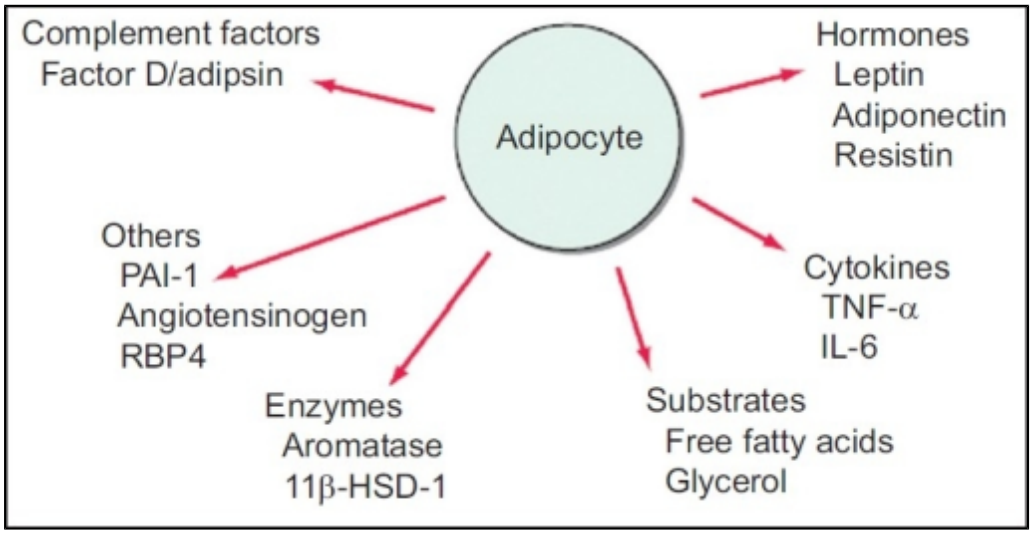


Fig. 13. Adipocytes and its substance action

Vascular endothelium is a main modifier of vascular tones. Perivaascular adipose tissue modifies the association between endothelium-dependent vasodilator (EDRF-NO) and vasoconstrictor substances such as endothelin-1.

Perivaascular adipose tissue around the micro vascular bed, derived from the subcutaneous fat around the gluteal region .Healthy lean people also has anticontractile properties of this bed. Leptin hormone and adiponection factor play a role in perivaascular adipose tissue associated endothelium-dependent vasodilation.

Inflammation: the link between obesity and diabetes

The family of NLR (Nod-like receptor), innate immunity cell sensors, like the leucine-rich-containg family, nucleotide-binding domain, pyrin domain-containing-3(Nlrp3), but alsoknownas Nalp3or cryopyrin) inflammasome are implicated inrecognizing certain non micro bial originate of ‘danger signals’. It leads to activation of caspase-1 and that activates interlukin-1beta (1l-1beta) and IL-18 synthesis.

Sl. No.	Risk	IDF (2005)	NCEP ATP III Criteria	WHO (1999)
I	Obese/ Visceral obesity	Waist circumference ≥ 90 centi metre in male, ≥ 80 centi metre in female,– In South Asians	Waist circumference ≥ 102 cm (M), ≥ 88 cm (F)	Body mass index ≥ 30 kilogram/m^2 and/or waist-to-hip ratio > 0.90 (Male), > 0.85 (Female)
II	Blood pressure(BP)			≥ 140/ ≥ 90 mm Hg
III.	Fasting glucose(FBS)	≥ 100 mg/dl or/and pre-existing DM	≥ 110 mille gram/dl	Diabetes, IGT or IR
IV	Triglycerides	≥ 150 mille gram /dl	≥ 150 mille gram /dl	≥ 150 mille gram /dl
	High density Cholesterol	< 40 mille gram /dl (Male), < 50mg/dl (Female)	< 40 mille gram /dl (M), < 50mg/dl (F)	
	Metabolic syndrome - definition		Atleast 3 risk factors	DM, IGT or IR plus any two or more risk factors

Table 6. Obesity and metabolic syndrome criteria

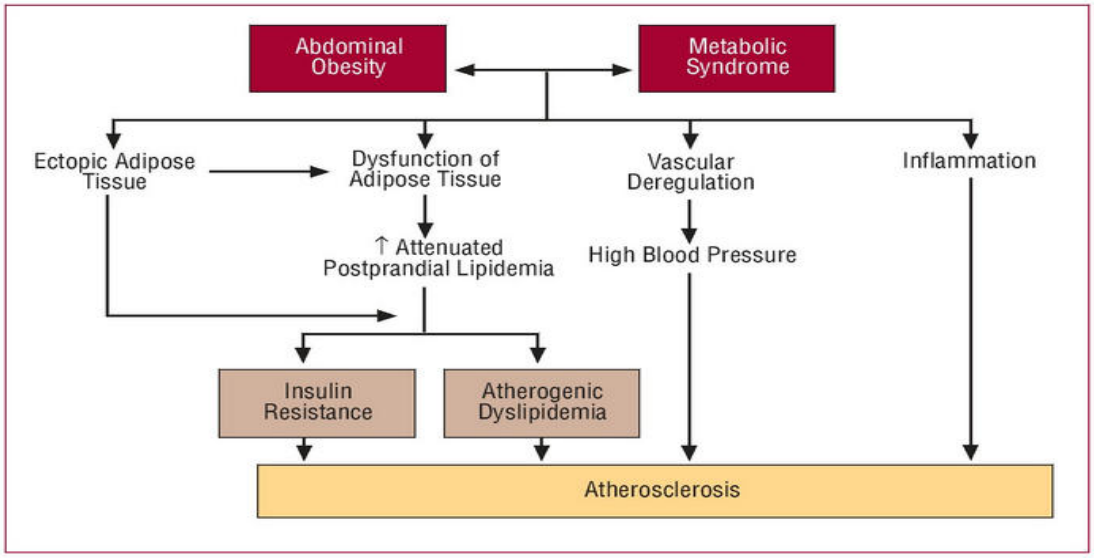


Fig. 14. Obesity and metabolic syndrome

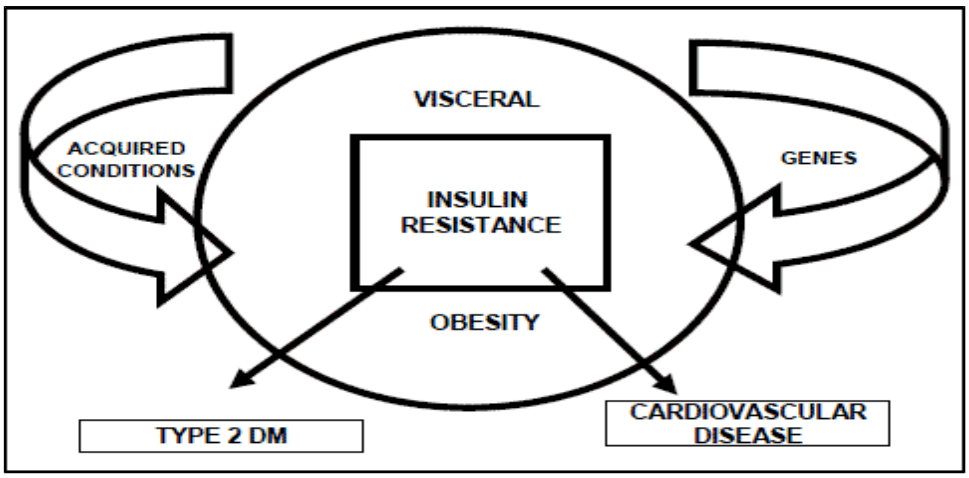


Fig. 15. Visceral fat and insulin resistance

INSULIN RESISTANCE

Insulin action: It is the effective anabolic hormone having a main role in carbohydrate, fatty acid and protein metabolism, growth and differentiation of cells, and vascular endothelial function.

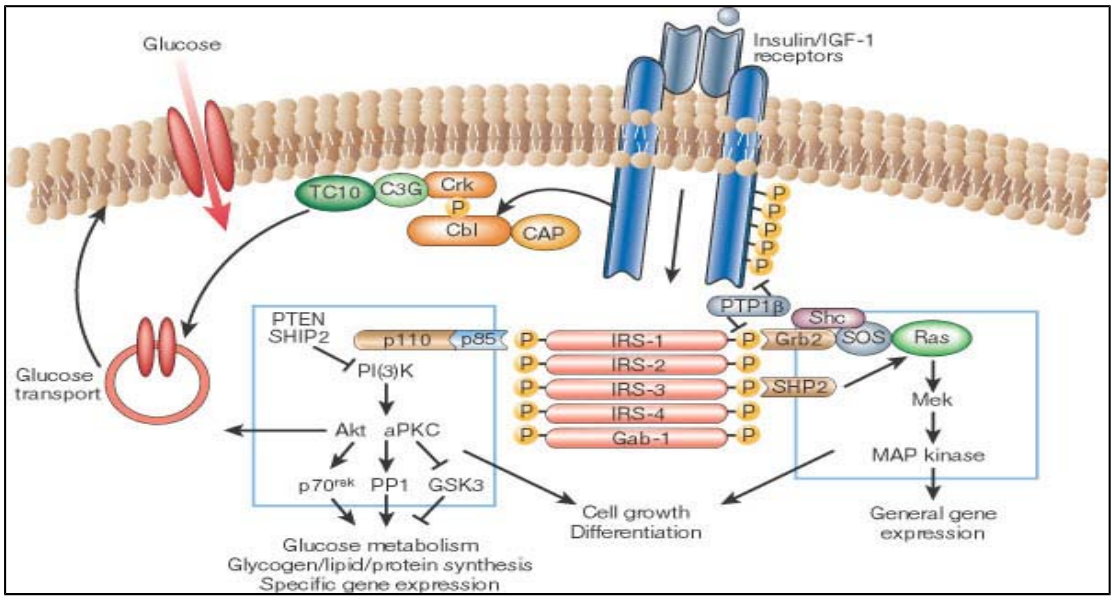


Fig. 16. Insulin signalling and the regulation of glucose and lipid metabolism

The glucoregulatory effects are by predominant action on three tissues: liver, muscle and adipose tissue.

The receptor of insulin can autophosphorylate and phosphorylate the substrates that is important for difficult cellular responses to insulin. Transphosphrylation of insulin receptor substrate (IRS) leads to activation of proteins 1-4. It leads to the activation of downstream signaling pathways. It can mediate insulin actions. IRS-1 has prominent action in the skeletal muscle and IRS-2 action in the liver. Path ways of two major signals activated by insulin binding to its receptor are the phosphatidylinositol-3-kinase (PI3K) pathway and mitogenic, or mitogen-activated protein kinase (MARK) pathway. Insulin resistance is states that the below normal biologic response to a given concentration of insulin.

Primary vs secondary insulin resistance

Intrinsic (primary) defects in insulin sensitivity caused by mutations of any protein and the final insulin-regulated proteins. Impairments in cellular events between insulin and its surface receptors leads to insulin resistance (IR) . It is called type A or secondary insulin resistance.

In secondary insulin resistance insulin sensitivity returns to normal after the removal of the factor or state. Cortisol, glucagon, catecholamines, and GH can promote insulin resistance (IR). In infection and stress conditions these hormones are secreted in excess leads to down-regulates insulin receptors. Increased levels of insulin down-regulates its receptors.

Another factor that affects sensitivity of insulin is auto antibodies to the insulin receptor. These antibodies are typically IgC and usually associated with other characteristics of autoimmunity.

Type A and type B resistance

In type A insulin resistance is inherited prototype. It is a syndrome of severe IR. It is categorized increased level of insulin .There may be associated with IGT, acanthosis nigicans and hyperandrogenism. Leprechaunism and lipodystrophy are classical examples. Type B resistance is due to antibodies to insulin receptor which may be associated with other autoimmune disease.

Obesity is associated with kidney disease via direct and indirect mechanisms. Indirect mechanisms include the association of obesity with hypertension, diabetes mellitus and atherosclerotic cardiovascular disease which are at risk factors for chronic kidney disease.

OBESITY AND HYPERTENSION

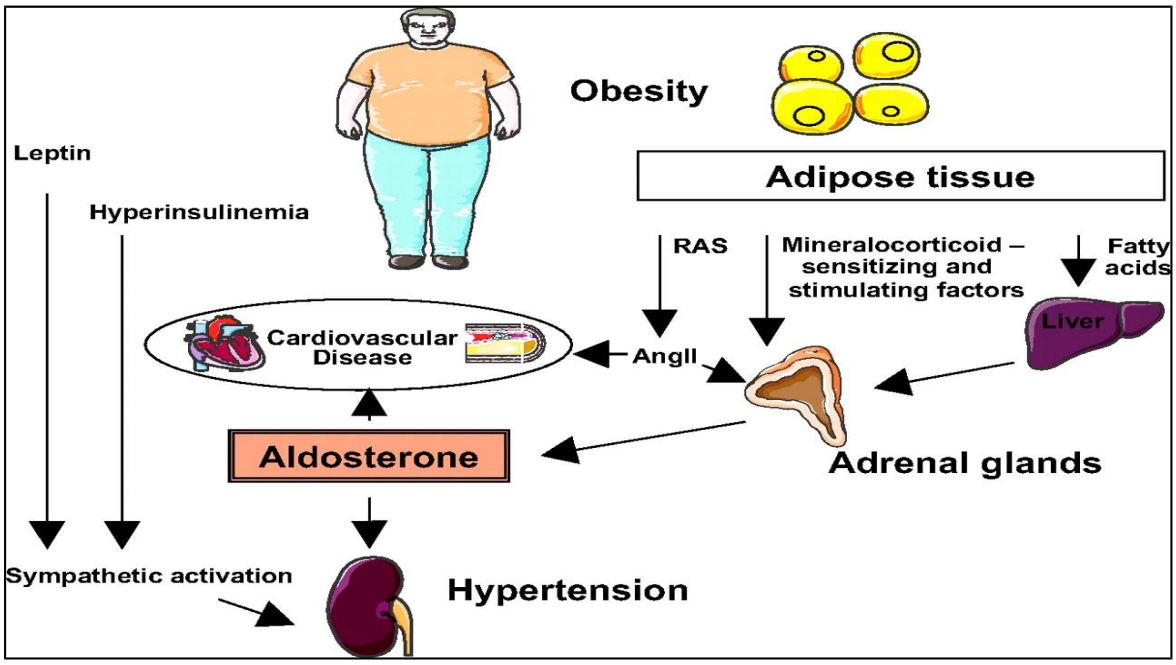


Fig. 17. Obesity and Hypertension

Obesity, kidney and liver

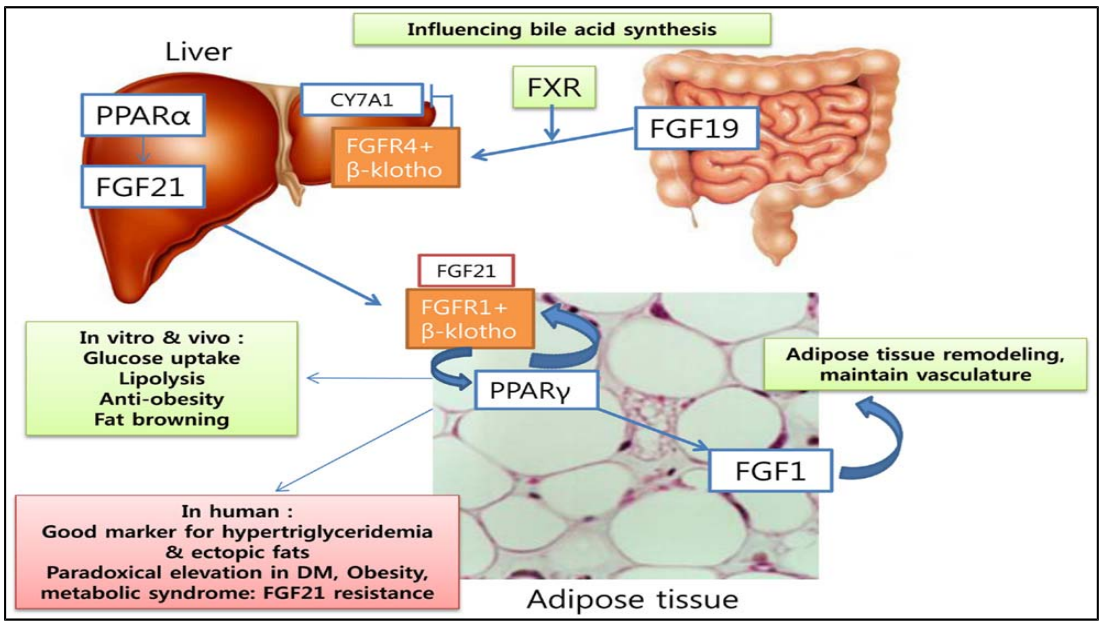


Fig. 18. Development of NAFLD and Obesity

Severely obese persons develop albuminuria with hypertrophy of podocyte, mesangial proliferation and glomerular hypertrophy enlargement, and focal segmental glomerular sclerosis in the absence of DM and SHT.

Fetuin-A and adiponection may have a role in linking obesity, chronic kidney disease and NAFLD. Feutin-A levels are high in obese, NAFLD and obese with CKD. Adiponection levels have been found to inversely correlative with proteinuria. Fetuin-A, a hepatokine released from the liver inhibits adiponection secretion from the adipose tissue. AMPK acts as a gauge for cellular energy reserve and is stimulated in states of energy depletion. AMPK also inhibits the generation of reactive oxygen species by suppressing an isoform of NADPH oxidase.

Normally adiponectin stimulates the AMPK activity resulting in stimulation of ATP producing catabolic pathway and inhibition of ATP consuming anabolic in obesity, a state of energy excess associated with low adiponection levels. When AMPK is chronically deactivated due to calorie excess there is both lipogenesis and increased cellular protein synthesis.

This along with oxidative stress results in podocyte hypertrophy with effacement of its foot process of the glomerulus, steatohepatitis and ultimately fibrosis due to reactive oxygen species and endoplasmic reticulum stress.

Osteo-sarcopenic obesity

Obesity believed to improve bone mass due to the mechanical effects of loading, increase in bone anabolic factors like hyperinsulinemia and increased estrogen due to increased aromatase activity. Bone mass has been found to be decreased in obesity.

Sarcopenia (loss of lean mass) is frequently seen with aging and is frequently associated with osteopenia. Obesity which may be considered a form of accelerated aging is frequently associated with loss of muscle mass and bone mass. The condition has been aptly termed 'Osteo-sarcopenic obesity'.

Inverse bone fat relationship. Both arise from the same mesenchymal stem cells, so when adipogenesis occurs, oteoblastogenesis is inhibited. Chronic inflammatory state of obesity results in the release of inflammatory cytokines that promote osteoclastic bone resorption by stimulating RANK/RANKL pathway. Obesity is associated with vitamin D deficiency which further impairs bone health.

The muscle-bone relationship is direct. Improved muscle mass improves bone health by the dynamic and static effect of loading. Contracting muscles are known to secrete 'myokines' which have beneficial effects on the bone, adipose tissue and improved metabolic

effects as opposed to adipokines. The important myokines include IL-6, myostatin, follistation, irisin etc.

Interleukin 6 (IL-6)

It is the first myokine described and is released in the circulation from the exercising muscles. It acts as an energy sensor and is released following muscle glycogen depletion. Sudden increasing levels of IL-6 increases fatty acid oxidation, glucose uptake and osteo protective and anti-inflammatory action.

This has somewhat paradoxical because IL-6 levels are known to be increased in type 2 diabetes and cardiovascular disease. Recent evidence points to the fact that IL-6 could be a marker of insulin resistance rather than a cause of it and obesity could be a state of IL-6 resistance resulting in increased levels.

Folistation

Following exercise a beneficial myokine follistatin is released which is a physiological antagonist of myostatin. Myostatin increases with physical inactivity, inhibits growth of muscles and promotes increase in adiposity. Thus exercise decreases the myostatin levels resulting in increase in muscle mass and decrease in fat mass.

Irisin

Another myokine released from the exercising muscle is irisin. Irisin has been responsible for the ’browing’ of the white adipose tissue resulting in’ beige’ or ‘brite’ adipocytes. These brite adipocytes are believed to have the same thermogenic potential as brown adipocytes and thereby result in decrease result in decrease in fat mass and weight loss.

Improvement in the bone mass results in release of factors like osteocalcin, osteoprotegerin and diminished osteopontin. These bone factors especiaaly osteocalcin has been found to improve insulin secretion and sensitivity. Osteopontin on the other hand has been linked to obesity and NAFLD.

Obesity and PCOS

Obesity and PCOS are closely related. In USA the prevalence of obesity in women with PCOS is 80%. Many women with PCOS have central obesity and upper abdominal obesity is associated with increased androgen production.

In PCOS an increase in the subcutaneous adipocyte size (which are more insulin resistant), decreased lipolytic effect of catecholamines on subcutaneous adipocytes and increased lipolytic activity on the visceral adipocytes. Increased delivery of FFA in the portal circulation

leading to hepatic insulin resistance. It acts on reproductive system and body metabolism. It action as a co-gonadotropin through its cognate receptor to modify steroidogenesis of ovary. Supra physiological doses of insulin, as seen in PCOS augment androgen production.

Insulin signals in the central nerves system in important for ovulation. This signaling is impaired in obesity and PCOS due to CNS insulin resistance. Hyperandrogenemia in PCOS can produce insulin resistance by directly affecting the insulin action on voluntary muscles and fatty tissue, by altering the adipokine secretions, and by raising the abdominal adiposity.

Obesity and cancer

Obesity is associated with colon cancer, CA breast, CA endometrial, renal malignancies, and adenocarcinoma of esophageal. Obesity has also been implicated in the occurrence of hematological malignancies like non-Hodgkin's lymphoma, leukemia, and multiple myeloma; thyroid cancer; pancreatic cancer; gallbladder cancer; high-grade prostate cancer; and ovarian cancer.

Insulin may promote mutagenesis by acting via the insulin or IGF-1 receptors. Low adiponection levels in obesity may be linked to cancer development and progression. Adiponection, the important protein

secreted by adipose tissue, exhibits insulin-sensitizing, anti-inflammatory, anti-atherogenic, pro-apoptotic, and anti-proliferative properties.

Obesity in south Asians

1. Urbanization
2. Less physical activity
3. High carbohydrate, high fat and low fiber food
4. Lower intake of w-3 PUFAs
5. Increased life expectancy of the elderly population
6. Migration from villages to cities
7. Stress and Alcohol and tobacco consumption

Metabolically obese Asian Indians

Asian and Indians could be classified as 'metabolically obese', i.e. they have several metabolic derangements but are 'non-obese, by usual BMI standards. These non-obese persons usually have elevated body fat, visceral adiposity, thick truncal SCAT and ectopic fat deposition features often seen South Asians. This pheno type contributes to IR, hyperglycemias, dyslipidemia, and excess pro-coagulant factors [57].

Relevance of Abdominal obesity and Asian Indian visceral fat

Distribution of fatty tissue is an important marker of obesity associated morbidity and mortality in humans. It can be associated with genetic cause, hormonal and environmental factors. Womens have more subcutaneous and gluteal-femoral region adipose tissue, which are essentially absent in men.

Men have a greater proportion of fatty tissue localized intra-abdominally. The gluteal-femoral adipose tissue is specifically enlarged in women. This has more lipoprotein lipase (LPL) activities. Women have the unique ability to protect visceral deposits from adipose tissue accumulation.

It is not seen in male. Whereas in males deposition of excess fat in this region parallel with other depots. This difference in the distribution of fat is attributed to the action of female sex steroid hormones on the regulation of adipocytes metabolism in concert with cortisol, which has a regularity role on LPL.

MNOW-Definition

Obesity is heterogeneous disorder with several possible etiologies, and obese persons do not show a clustering of metabolic and cardiovascular risk factors. Similarly, all lean persons do not present with a healthy metabolic and disease-free profile. “Metabolic Obesity”, means the reference to visceral fat deposition in either lean or obese individuals.

It can identify the high for cardiovascular disorder better than the usually used definitions of obesity. Metabolically healthy obesity (MHO) indicates the absence of any overt cardiometabolic disorder, in particular,

type 2 diabetes, dyslipidaemia and hypertension in an individual with a BMI>30 kg/m^2.

In contrast, MONW individuals are characterized by presence of these diseases, even though they are not obese. In the nonexistence of pathologies, the correlation and clustering of cardio metabolic risk factors or inflammatory markers have also been used to categorize subjects as metabolically normal or abnormal [58].

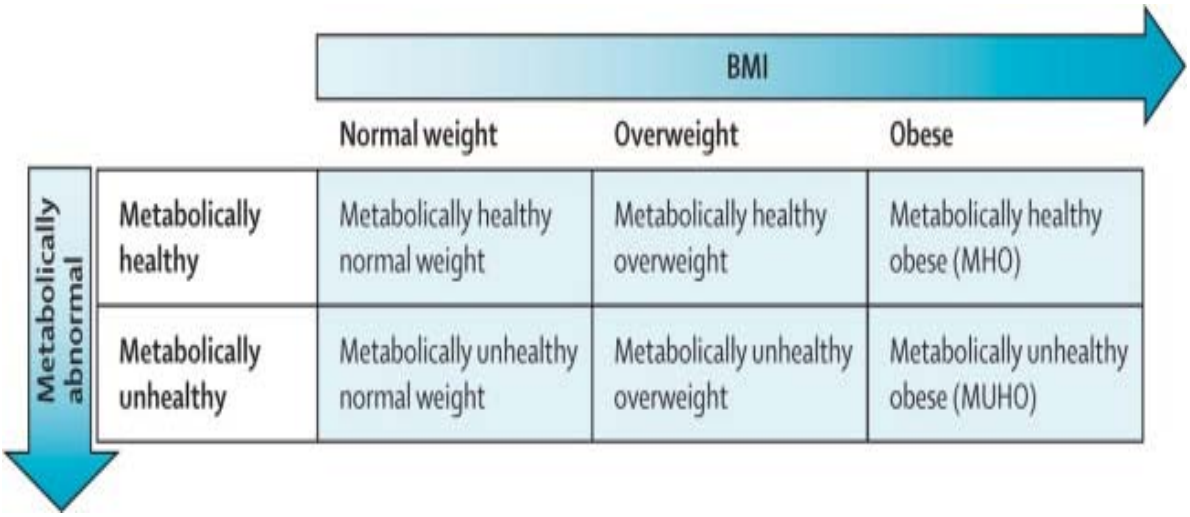


Fig. 19. Obesity spectrum (Four subtypes of obesity)

MONW-Metabolically Obese Normal Weight

In the recent past research has particular point on group of obese persons who are "metabolically healthy" (MHO) or "metabolically normal" (MNOB) despite raised adiposity. Some studies have shown that these individuals may not be at elevated risk for mortality, and treatment for obesity may be unnecessary.

Four categories of individuals may be identified in population based on BMI values (<30 or >30 kg/m^2) and presence or absence of metabolic derangements-Metabolically Abnormal Obese [MAO, also

termed Metabolically Resistant Obese (MRO) or Metabolically Obese Obese (MOO)], Metabolically Normal Obese [MNOB, also termed as Metabolically Healthy Obese (MHO)], Metabolically Obese Normal Weight [MONW, also termed as Metabolically Obese Non Obese (MONO)], and Metabolically Healthy Normal Weight [MHNW, also termed as Metabolically Healthy Non Obese (MHNO)].

Obesity in Asian Indians

Asians generally have a lower body-mass index as compared to their western counterparts, and they differ from European populations in relations between body-mass index and percentage of body fat and health risks. Indians have high percentage of body fat, abdominal obesity, hyperinsulinemia and low muscle mass. In particular, visceral obesity is common in South Asians, and evident even in non-obese people. That possibly explains why Asian Indians have a elevated risk of developing obesity related diseases and mortality at lesser levels of BMI.

Asian Indian Phenotype

Elevated predisposition to diabetes and premature coronary artery disease in Indian may be attributable to the so called ‘Asian Indian Phenotype’. Indian phenotype is characterized a larger central body obesity as shown by larger waist circumference (WC) and waist-to-hip

ratios (WHR) associated with a relatively smaller BMI, a measure of generalized obesity.

This cause to unique biochemical and hormonal changes including more plasma insulin levels, greater insulin resistance, lower HDL-cholesterol, higher triglyceride levels, increased small dense LDL-cholesterol, and decreased adiponection levels. Many Indians fit into the category of metabolically obese, normal weight persons [59].

Understanding MONW

In the year of 1980s, more investigators began to identify the existence of different subtypes of obesity. In 1981, Neil Rudderman reported the initial description of Metabolically Obese but Normal Weight (MNOW) phenotype, defined a subgroup of normal-weight individuals displaying obesity-related characteristic.

It was suggested that there are individuals who by standard weight tables are not obese or even overweight, but who have metabolic abnormalities characteristically associated with adult-onset obesity, are hyperinsulinemic, insulin-resistant, and have high predisposition for developing type 2 diabetes, hypertriglyceidemia, and premature coronary heart disease. It was also suggested that these MNOW individuals represent one end of spectrum of obesity and that some of them would be

difficult to detect by any criteria. And as in theor obese counterparts, associated metabolic disorders like hypertriglyceridemia and type 2 diabetes in these individuals also would benefit from caloric restriction.

Metabolically obese, normal weight pheno type persons are common in our population .It can represent the one end of the spectrum with insulin resistance syndrome (IRS).

MNOW in Indians

Limited data on prevalence of MNOW in India. A study assessing association of obesity and coronary risk factors in a North Indian community reported a high prevalence of diabetes (40/226, 17.7% in men and 33/232, 14.2% in women), overweight and obesity (BMI>25) in both men (123/226, 54.4%) and women (162/232, 69.8%).

Interesting to note is that both men and women with BMI<25kg/m^2 also had significant type 2 diabetes (16/103 men, 15.5%; 6/70 women, 8.6%), hypertension (men 35/103, 34%, women 19/70, 27.1%), high total cholesterol (29/103 men, 28.2%, 13/70 women, 18.6%), high triglycerides (15/103, 14.5% men, 18/70, 25.7% women), low HDL (79/103, 76.7% men, 29/70, 41.4% women) and metabolic syndrome (15/103, 14.6% men, 11/70, 15.7% women). This may be representative of prevalence of MONW phenotype in this study population [60].

Obesity paradox

When compared with normal subjects MONW individuals not only have an abnormal and more atherogenic metabolic profile, they are also predisposed to a higher risk of mortality as compared to their overweight and obese counterparts-the Obesity Paradox [61].

"Obesity paradox" means that, although obesity is a important risk factor for the development of cardiovascular and peripheral vascular disease, several studies has shown an inverse relationship between obesity and mortality.

When acute cardiovascular decompensation occurs, like, primary outcome in myocardial infarction, congestive heart failure, Post CABG, during dialysis, during sepsis: obese patients may have a survival benefit.

This benefit also observed in normal healthy obese elderly men and women, chronic hypertensives, diabetes and so on.

Major hypotheses for this apparent survival effect include

1. Obese patients may have good and aggressive medical care and improved observation than normal-weight persons.
2. Obese patients are more and better medical therapy than other individuals.

3. Obese patients tend to be young at the time of diagnosis of cardiovascular disorder, which can give an age benefit to them.
4. Some experts claim the sample size of the existing studies on obesity is still too small or too indecisive to make such determinations.
5. Other investigators suggest the way we measure obesity is unsatisfactory and may explain some of the paradoxical results seen.
6. Patients who lose weight involuntarily are likely to have severe underlying disease, and their life expectancy might be shorter, in contrast to patients who lose weight intentionally with lifestyle modifications, who have better outcomes.

These factors may lead to the false impression that obesity confers some survival advantage with acute cardiovascular stress and in some chronic conditions. Studies shows that obesity alone may confer a survival benefit independent of age, medical care, or therapy.

Positive inflammatory status correlated with metabolic health in obese and nonobese individuals, and these observations may have health and clinical significance in view of screening and stratification based on metabolic health phenotype to identify those at greatest cardiometabolic risk.

MATERIALS AND METHODS

MATERIALS AND METHODS

Design : Prospective study.

Sample Size : 100 Patients.

Duration of the study : August 2013 to July 2014

Inclusion Criteria

Adult of 17 to 45years with BMI of More than 25 kg/m^2 are selected. The persons are selected from OP and IP department. The people attending master health check-up are taken for our study group.

WHO criteria for obesity is used for the sample grouping.

Table 1. Classification of obesity with BMI level

	BMI (kg/m^2)	Obesity Class	Risk of Disease
Under weight	<18.5		
Healthy weight	18.5-24.9		
Overweight	25.0-29.9		Increased
Obesity	30.0-34.9	I	High
Obesity	35.0-39.9	II	Very high
Extreme obesity	≥40	III	Extremely high

Exclusion criteria

Patient with following history were excluded from our study

Chronic Smoker

On chronic drugs, Self medication

Connective tissue disease like rheumatic arthritis, SLE

Systemic Hypertension

Chronic illness like renal disorders and liver diseases

Cardiovascular diseases

Diabetes mellitus

Pregnancy, Menstrual abnormalities, patients with IUCD

Mentally Retarded person

Hypothyroidism

Written informed consent was taken from the patient. History regarding patient life style, eating habits, household works done by them is taken. Family history of obesity other chronic illness, occupation of the patient, residency of the patient (rural or urban) was taken in detail.

Detail physical examination of the patient was done. Patient weight, height, waist circumference of the patient was measured. From the weight and height, BMI was calculated.

Method of waist circumference measurement

By using non stretchable flexible inch tape in horizontal position, just above the iliac crest in normal end of the expiration while the patient is standing.

Blood sample was taken send for lipid profile, blood sugar, and serum C-reactive protein.

Serum C-reactive protein was measured by immunonephalometry method. Two ml of blood was taken for serum C-reactive level.

Patients are classified into three groups based on level of C-reactive protein. Those with less than 1 mg per dl were taken as negative group (low risk).

Patient with 1-2 mg per dl was grouped into moderate risk.

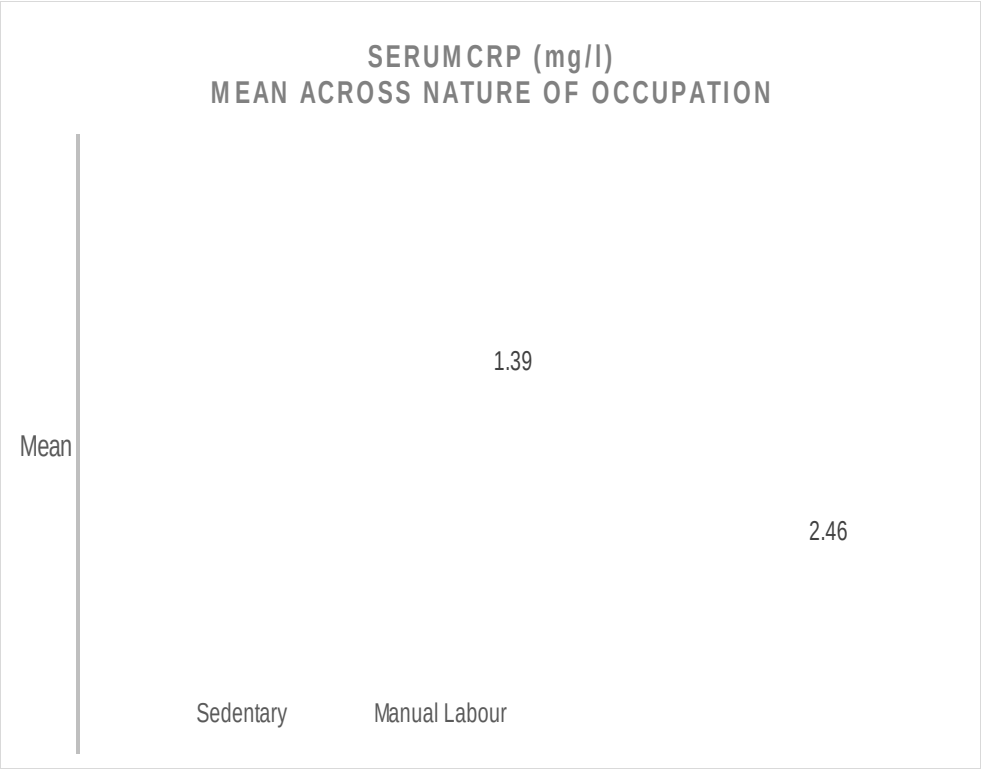
Those with high C-reactive protein more than 3 mg per dl was grouped in to high risk. These two groups taken as CRP positive group.

Risk level	**CRP (mg/L)**
Low	< 1.0
Average	1.0– 3.0
High	>3.0

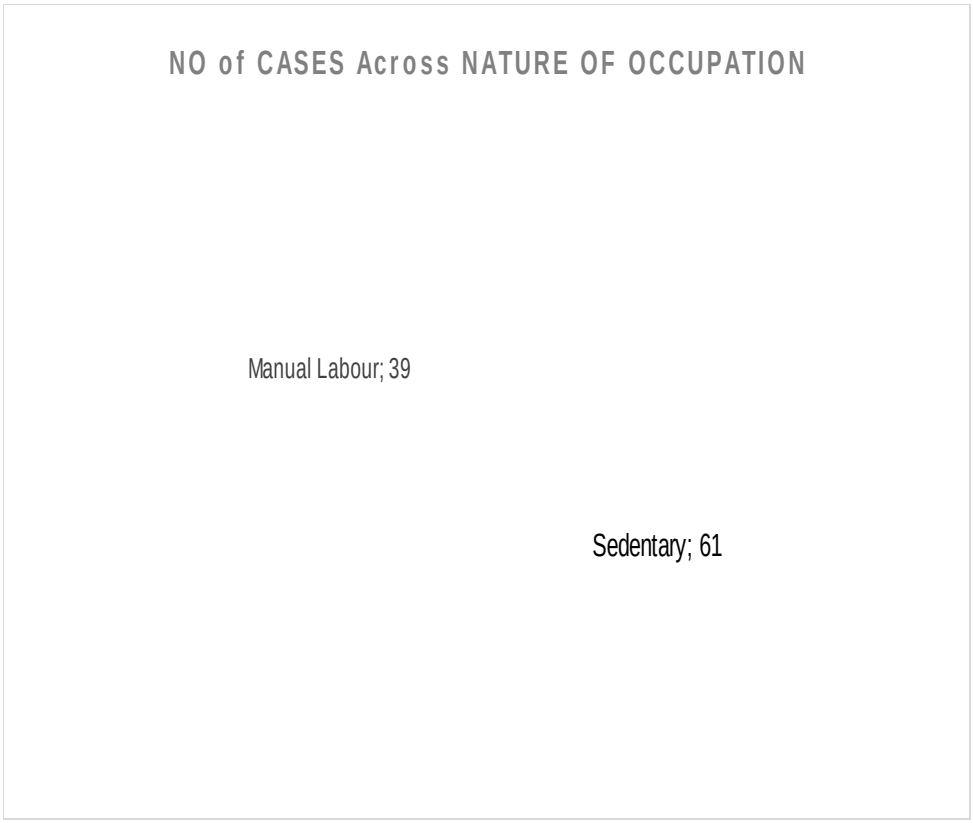
OBSERVATIONS

OBSERVATIONS

Average levels of SERUM CRP across Occupation				
	SERUM CRP (mg/L)			
Occupation	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Sedentary	2.46	0.00	5.20	61
Manual Labour	1.39	0.00	3.20	39
Total	2.05	0.00	5.20	100



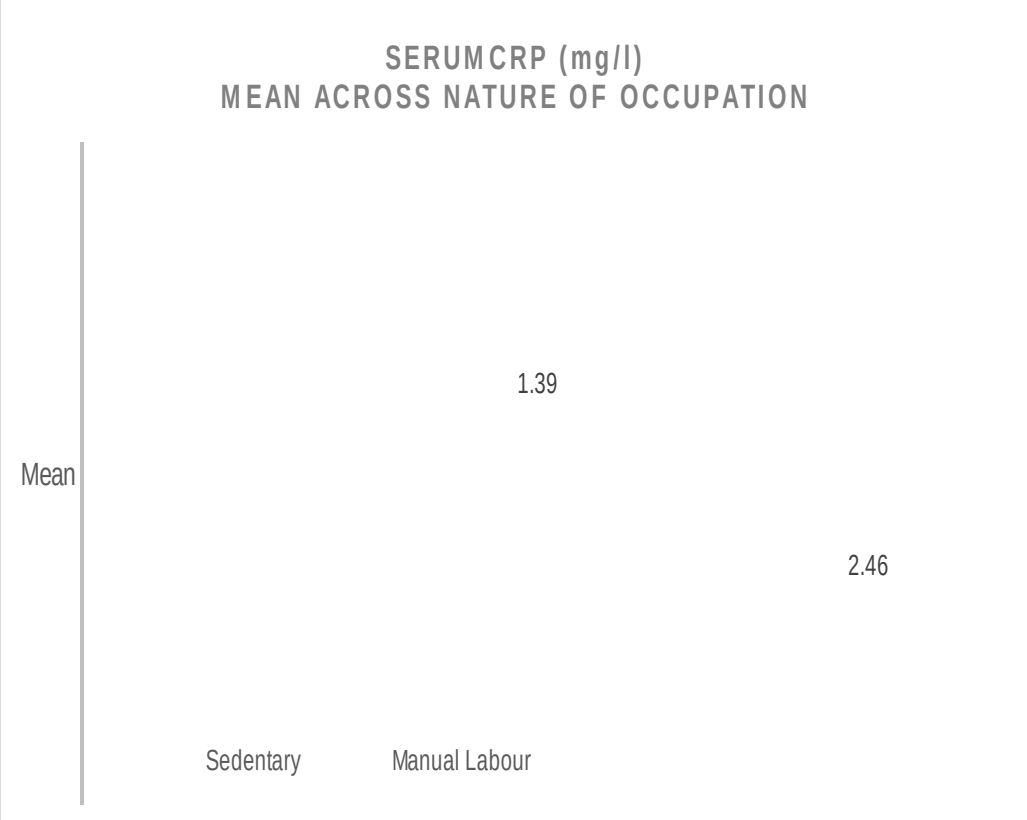
ANOVA					
SERUM CRP(mg/L)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	27.29498164	1.00	27.29	9.36	0.00
Within Groups	285.7970694	98.00	2.92		
Total	313.092051	99.00			



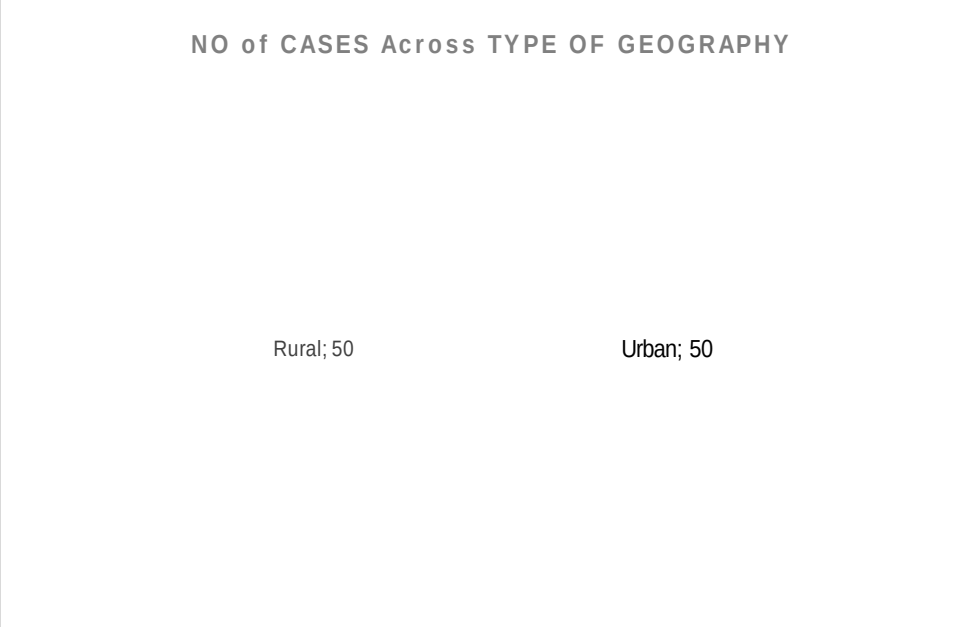
INTERPRETATION

- The mean of SERUM CRP in (mg/L) for sedentary occupation is 2.46 and 1.39 for manual labor.
- The Anova table indicates that the mean level between both the categories is significantly different.
- The conclusion is that SERUM CRP is highest for Sedentary Occupation as compared to manual labor occupation.

Average levels of SERUM CRP across Urban and Rural Areas				
	SERUM CRP (mg/L)			
Rural&Urban	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Urban	2.62	0	5.10	50
Rural	1.47	0	5.20	50
Total	2.05	0	5.20	100



ANOVA					
SERUM CRP (mg/L)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	33.373729	1.00	33.37	11.69	0.00
Within Groups	279.718322	98.00	2.85		
Total	313.092051	99.00			

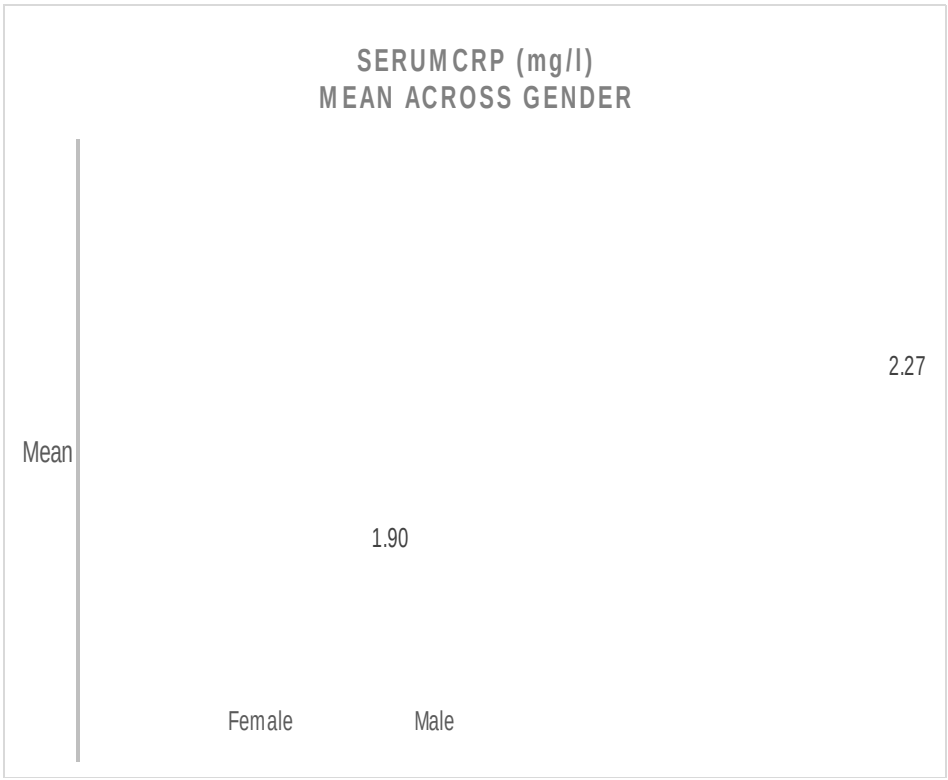


INTERPRETATION

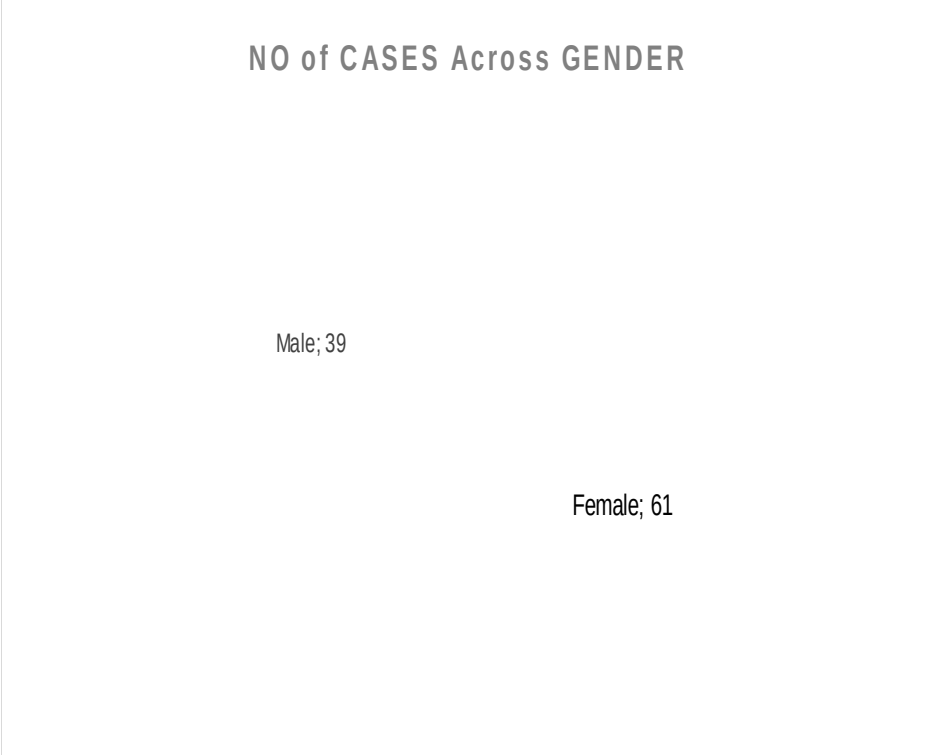
- The mean of SERUM CRP in (mg/l) for urban area is 2.62 and 1.47 for rural area.
- The Anova table indicates that the mean level between both the categories is significantly different.
- The conclusion is that SERUM CRP is highest for Urban area as compared to rural area

Average levels of SERUM CRP across Gender				
	SERUM CRP (mg/L)			
Gender	Mean	Minimum	Maximum	No of Cases

Female	1.90	0	5.2	61
Male	2.27	0	5.1	39
Total	2.05	0	5.2	100



ANOVA					
SERUM CRP (mg/L)	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	3.17	1.00	3.17	1.00	0.32
Within Groups	309.92	98.00	3.16		
Total	313.09	99.00			

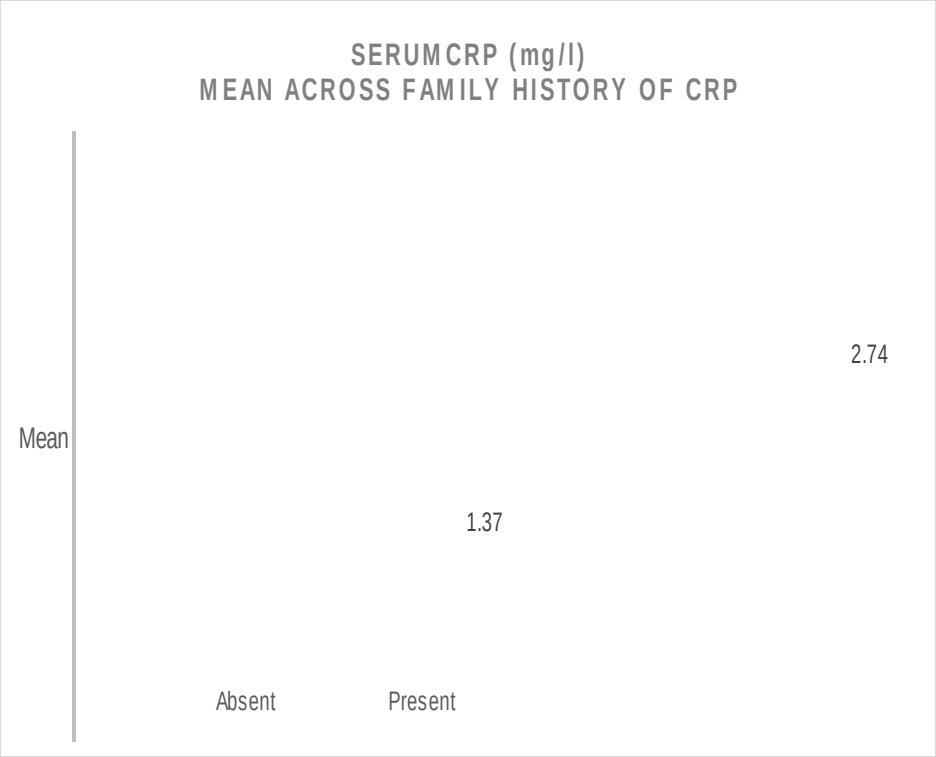


INTERPRETATION

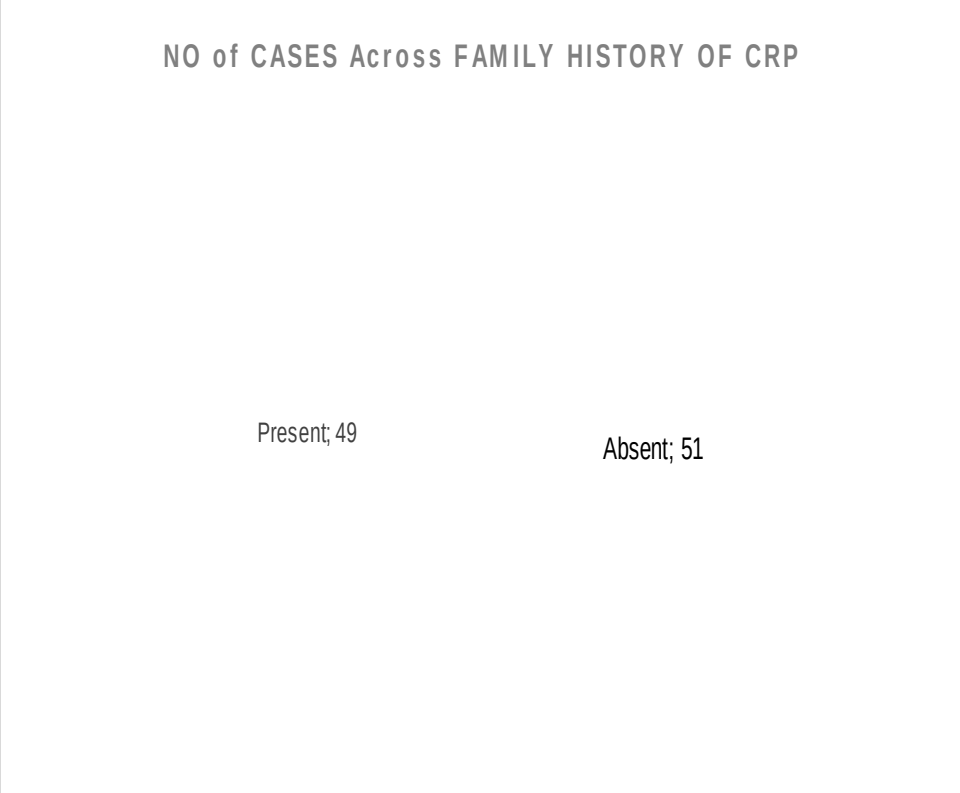
- The mean of SERUM CRP (mg/L) for Female Gender is 1.9 and 2.27 for Male Gender .
- The Anova table indicates that the mean level between both the categories is not significantly different .
- The conclusion is that SERUM CRP is not very different between the gender categories.

Average levels of SERUM CRP across Family History	
	SERUM CRP (mg/L)

Family History	Mean	Minimum	Maximum	No of Cases
Absent	1.37	0	3.7	51
Present	2.74	0	5.2	49
Total	2.05	0	5.2	100



ANOVA					
SERUM CRP (mg/L)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	46.89	1.00	46.89	17.26	0.00
Within Groups	266.20	98.00	2.72		
Total	313.09	99.00			

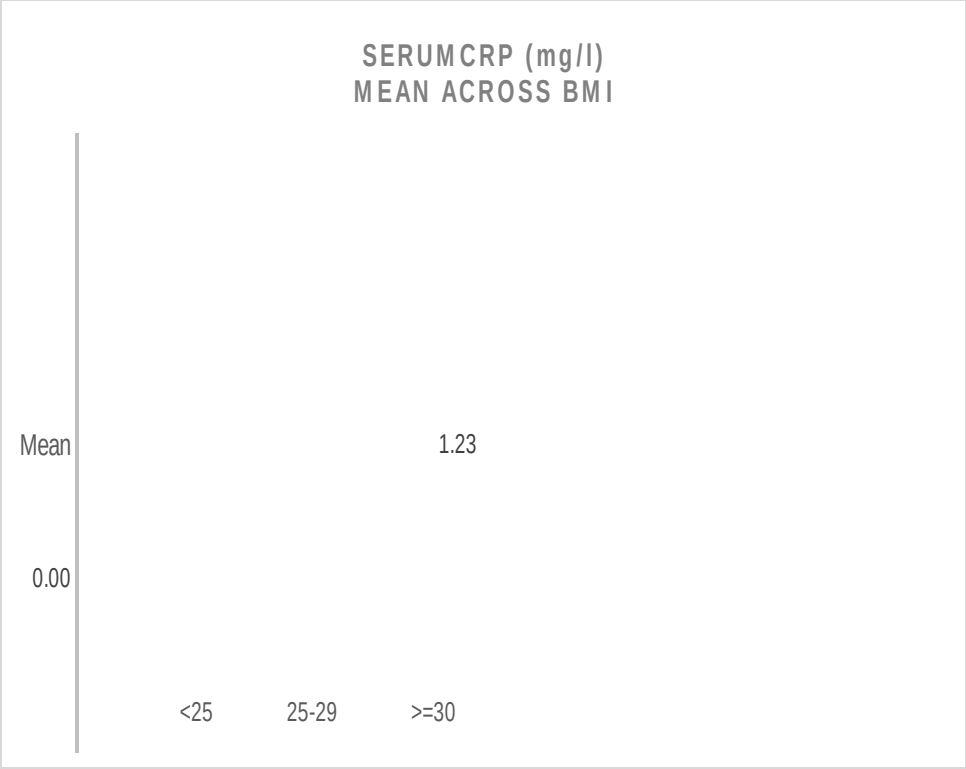


INTERPRETATION

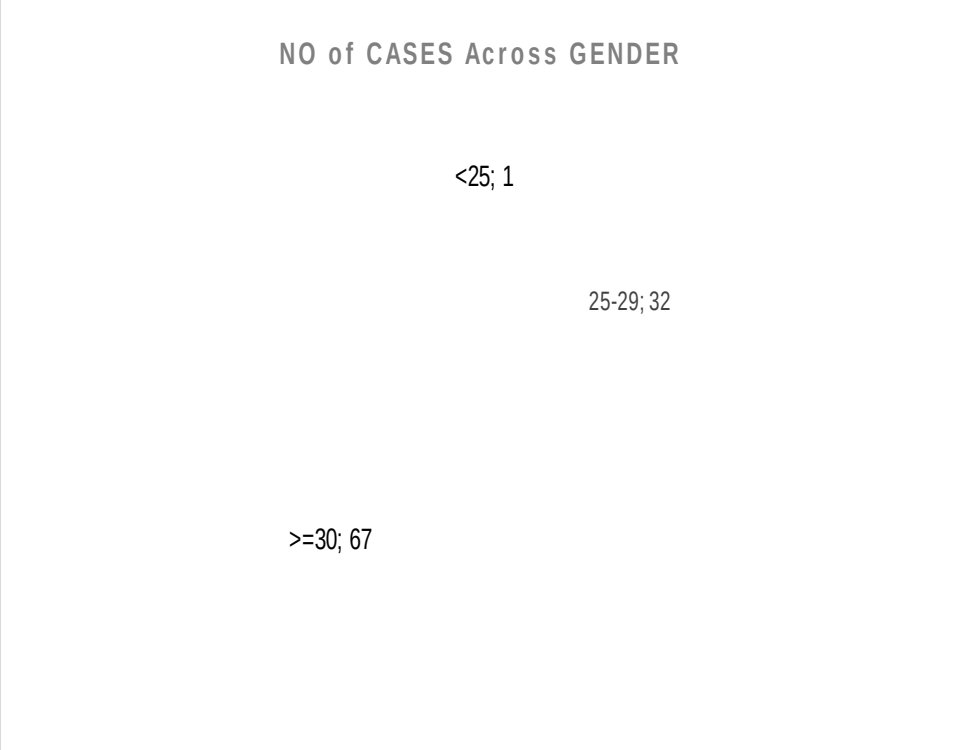
- The mean of SERUM CRP in (mg/L) for absent family history is 1.37 and 2.74 for present family history .
- The Anova table indicates that the mean level between both the categories is significantly different.
- The conclusion is that SERUM CRP is highest for Present family history as compared to absent family history.

Average levels of SERUM CRP across BMI	
	SERUM CRP (mg/L)

BMI Cut off (Kg/m$^{2)}$	Mean	Minimum	Maximum	No of Cases
<25	0.00	0	0	1
25-29	1.23	0	5.1	32
>=30	2.47	0	5.2	67
Total	2.05	0	5.2	100



ANOVA					
SERUM CRP (mg/L)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	37.45	2.00	18.73	6.59	0.00
Within Groups	275.64	97.00	2.84		
Total	313.09	99.00			



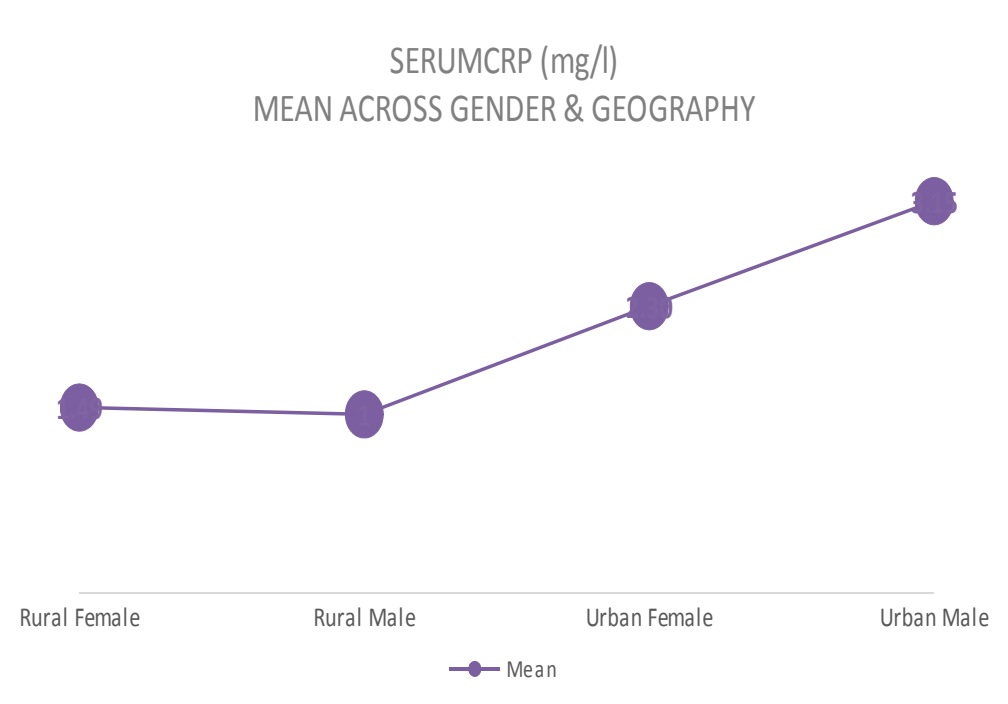
INTERPRETATION

- The mean of SERUM CRP in (mg/L) for 25 to 29 BMI cutoff is 1.23 and greater than 30 BMI cutoff is 2.47.
- The Anova table indicates that the mean level between both the categories is significantly different.
- The conclusion is that SERUM CRP is highest for BMI cutoff greater than 30 as compared to BMI cutoff for 25 to 29.

Average levels of SERUM CRP across Rural Urban Areas and Gender				
	SERUM CRP (mg/L)			
Area and Gender	Mean	Minimum	Maximum	No of Cases

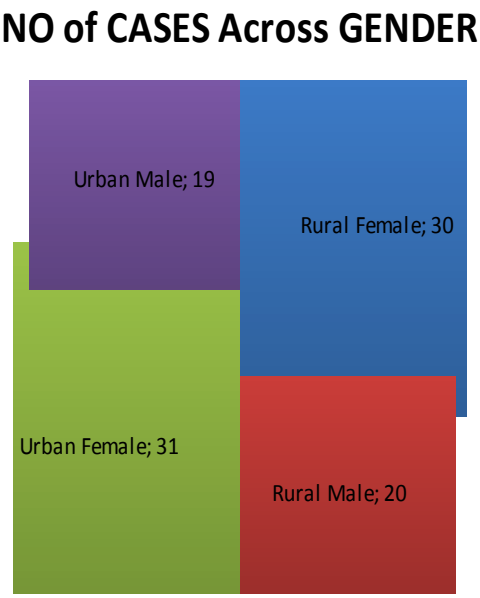
Rural Female	1.49	0	5.20	30
Rural Male	1.44	0	3.90	20
Urban Female	2.30	0	5.10	31
Urban Male	3.15	0	5.10	19
Total	2.05	0	5.20	100

ANOVA					
SERUM CRP (mg/L)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	41.77	3.00	13.92	4.93	0.00
Within Groups	271.32	96.00	2.83		
Total	313.09	99.00			



Multiple Comparisons						
SERUMCRP **(mg/L)**						

Tukey HSD						
(I) R_U_SEX	(J) R_U_SEX	Mean Difference (I-J)	Std. Error	P Value	95% Confidence Interval	
					Lower Bound	Upper Bound
Rural Male	Urban Male	-1.71	0.54	0.01	-3.12	-0.30
	Rural Female	-0.06	0.49	1.00	-1.32	1.21
	Urban Female	-0.87	0.48	0.28	-2.13	0.39
Urban Male	Rural Male	1.71	0.54	0.01	0.30	3.12
	Rural Female	1.66	0.49	0.01	0.37	2.94
	Urban Female	0.84	0.49	0.32	-0.44	2.12
Rural Female	Rural Male	0.06	0.49	1.00	-1.21	1.32
	Urban Male	-1.66	0.49	0.01	-2.94	-0.37
	Urban Female	-0.81	0.43	0.24	-1.94	0.31
Urban Female	Rural Male	0.87	0.48	0.28	-0.39	2.13
	Urban Male	-0.84	0.49	0.32	-2.12	0.44
	Rural Female	0.81	0.43	0.24	-0.31	1.94
*. The mean difference is P Value significant at the 0.05 level.						

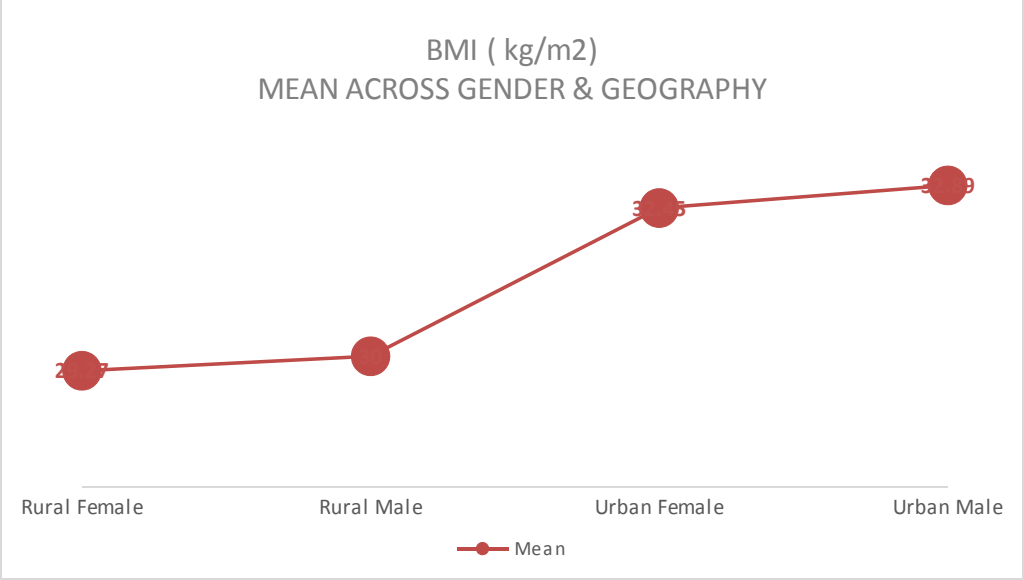


INTERPRETATION

- The mean of SERUM CRP (mg/L) for Rural Female is 1.49, Rural Male is 1.44, InUrban Female is 2.30 and Urban Male is 3.15.
- The Anova table indicates that the mean level between one of the categories are significantly different.
- The multiple comparison tables indicates that mean SERUM CRP is significantly different between Rural Male and Urban Male and between Urban Male and Rural Female.
- However between rural male and rural female there is no significant difference between SERUM CRP.
- Also no significance difference between urban male and urban female there is no significant difference between SERUM CRP.

Average levels of BMI across Rural Urban Areas and Gender				
	BMI (Kg/m^2)			
Area and Gender	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Rural Female	29.27	24.00	40.00	30.00
Rural Male	29.55	26.00	37.00	20.00
Urban Female	32.45	26.00	39.00	31.00
Urban Male	32.89	26.00	38.00	19.00
Total	31.00	24.00	40.00	100.00

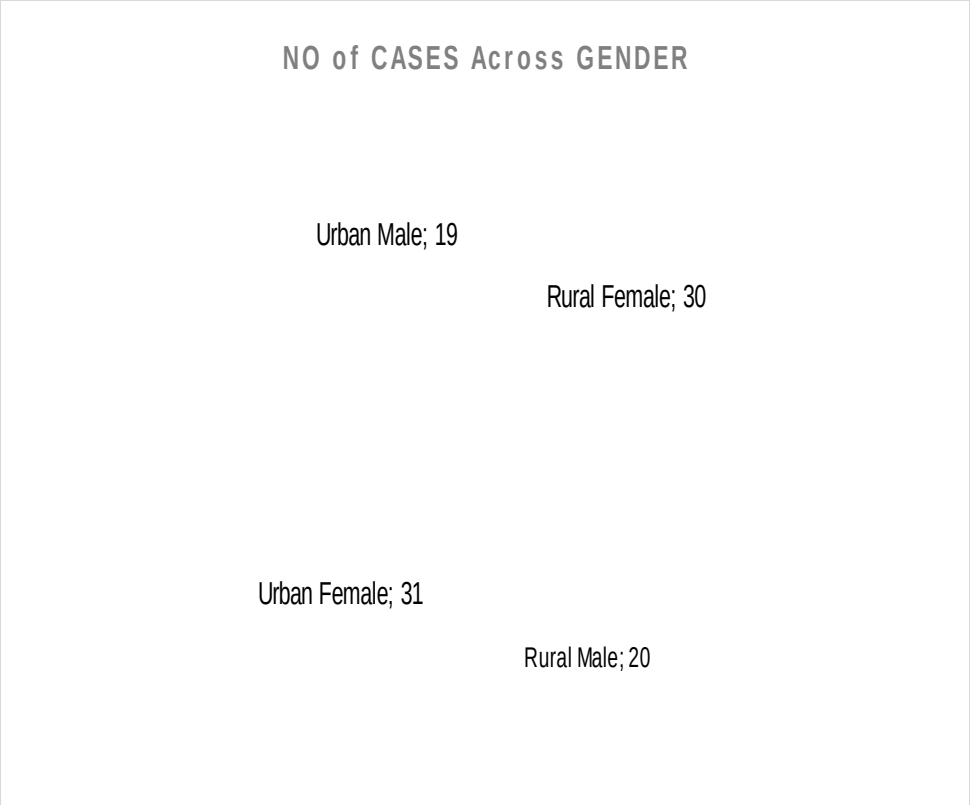
ANOVA					
BMI (Kg/m^2)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	265.72	3	88.57	7.63	0.00
Within Groups	1114.28	96	11.61		
Total	1380	99			



*. The mean difference is P Value significant at the 0.05 level.

Multiple Comparisons						
BMI (Kg/m^2)						

Tukey HSD						
(I) R_U_SEX	(J) R_U_SEX	Mean Difference (I-J)	Std. Error	P Value	95% Confidence Interval	
					Lower Bound	Upper Bound
Rural Male	Urban Male	-3.34	1.09	0.01	-6.20	-0.49
	Rural Female	0.28	0.98	0.99	-2.29	2.85
	Urban Female	-2.90	0.98	0.02	-5.46	-0.35
Urban Male	Rural Male	3.34	1.09	0.01	0.49	6.20
	Rural Female	3.63	1.00	0.00	1.02	6.24
	Urban Female	0.44	0.99	0.97	-2.15	3.04
Rural Female	Rural Male	-0.28	0.98	0.99	-2.85	2.29
	Urban Male	-3.63	1.00	0.00	-6.24	-1.02
	Urban Female	-3.18	0.87	0.00	-5.47	-0.90
Urban Female	Rural Male	2.90	0.98	0.02	0.35	5.46
	Urban Male	-0.44	0.99	0.97	-3.04	2.15
	Rural Female	3.18	0.87	0.00	0.90	5.47



INTERPRETATION

- The mean of BMI (Kg/m^2) for Rural Female is 29.27, Rural Male is 29.55, Urban Female is 32.45 and Urban Male is 32.89.

- The Anova table indicates that the mean level between one of the categories are significantly different .
- The multiple comparison tables indicate that there is significant difference in BMI levels between urban male and rural male, also between rural male and urban female.
- Also the mean levels are different between urban female and rural female.
- Conclusion, mean levels of BMI is highest for urban female as compared to rural female.
- Mean levels of BMI is highest for urban male as compared to rural male.
- Mean levels of BMI is highest for urban female as compared to rural male .

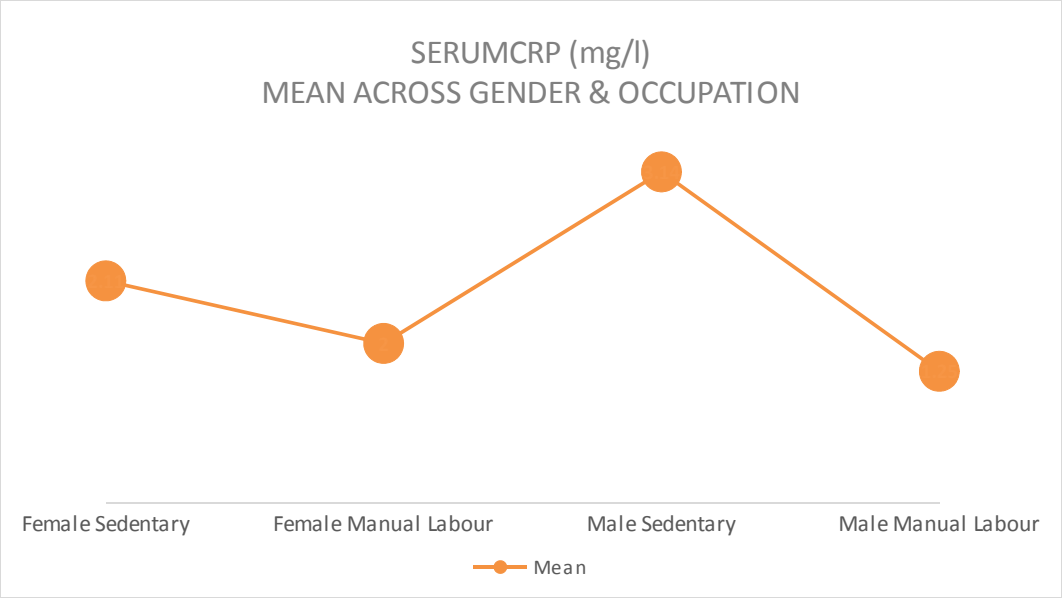
BMI_AGE_Cutoff vs CRP Level

Case Summaries				
BMI_AGE_Cutoff	SERUM CRP (ml/L)			
	Mean	Minimum	Maximum	No of Cases
20	0.00	0.00	0.00	1
22	0.81	0.00	3.90	11
23	1.41	0.00	5.10	15
24	1.53	0.00	4.10	6
32	2.22	0.00	4.80	15
33	2.23	0.00	5.10	35
34	3.16	0.00	5.20	17
Total	2.05	0.00	5.20	100

ANOVA					
SERUM CRP **(mg/L)**					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	51.56	6.00	8.59	3.06	0.01
Within Groups	261.54	93.00	2.81		
Total	313.09	99.00			

Average levels of SERUMCRP across Gender and Occupation				
	SERUMCRP (mg/L)			
Sex_Occupation	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Female Sedentary	2.11	0.00	5.20	40
Female Manual Labour	1.51	0.00	2.90	21
Male Sedentary	3.14	0.00	5.10	21
Male Manual Labour	1.25	0.00	3.20	18
Total	2.05	0.00	5.20	100

ANOVA					
SERUMCRP (mg/L)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	42.69272957	3.00	14.23	5.05	0.00
Within Groups	270.3993214	96.00	2.82		
Total	313.092051	99.00			


SERUMCRP (mg/l)
MEAN ACROSS GENDER & OCCUPATION
Female Sedentary
Female Manual Labour
Male Sedentary
Male Manual Labour
Mean

Multiple Comparisons						
SERUMCRP **(mg/L)** Tukey HSD						
(I) Sex_Occupation	(J) Sex_Occupation	Mean Difference (I-J)	Std. Error	P Value	95% Confidence Interval	
					Lower Bound	Upper Bound
Female Sedentary	Female Manual Labour	0.59	0.45	0.56	-0.59	1.78
	Male Sedentary	-1.03	0.45	0.11	-2.22	0.15
	Male Manual Labour	0.86	0.48	0.28	-0.39	2.10
Female Manual Labour	Female Sedentary	-0.59	0.45	0.56	-1.78	0.59
	Male Sedentary	-1.63	0.52	0.01	-2.98	-0.27
	Male Manual Labour	0.26	0.54	0.96	-1.15	1.67
Male Sedentary	Female Sedentary	1.03	0.45	0.11	-0.15	2.22
	Female Manual Labour	1.63	0.52	0.01	0.27	2.98
	Male Manual Labour	1.89	0.54	0.00	0.48	3.30
Male Manual Labour	Female Sedentary	-0.86	0.48	0.28	-2.10	0.39
	Female Manual Labour	-0.26	0.54	0.96	-1.67	1.15
	Male Sedentary	-1.89	0.54	0.00	-3.30	-0.48
*. The mean difference is P Value sinnificant at the 0.05 level.						

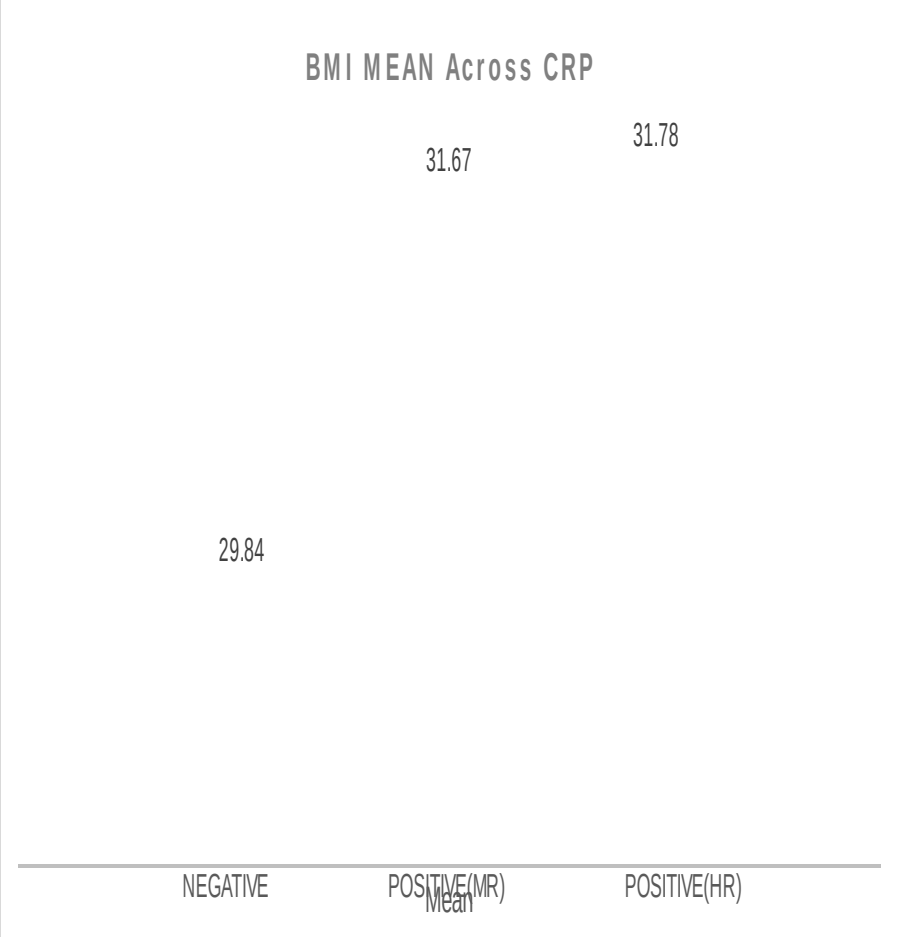
INTERPRETATION

- The mean of SERUM CRP **(mg/L)** is 2.11 for Female Sedentary occupation, 1.51 for Female Manual Labor, 3.13 for Male Sedentary occupation and 1.25 for Male manual labor
- The anova table indicates that there is significant difference in average SERUM CRP at least between one of the categories
- The multiple comparison table indicates that mean SERUM CRP is significantly different between the following categories
- Female Manual Labour and Male Sedentary occupation

- ❖ Male Sedentary and Male Manual Labour
- ❖ Conclusion is that SERUM CRP is highest for Male Sedentary occupation as compared to Female Sedentary Occupation
- ❖ Also, SERUM CRP is highest for Male Sedentary occupation as compared to Female Sedentary Occupation

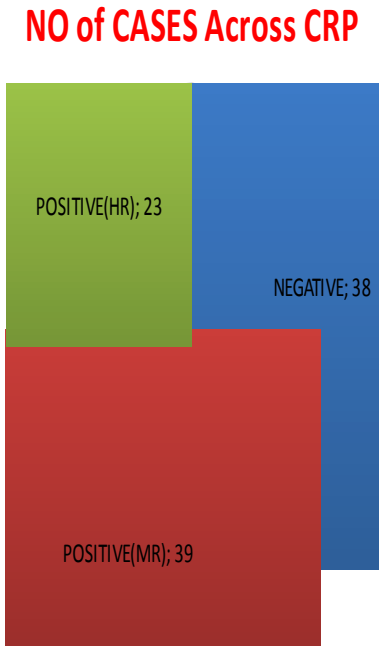
BMI Average Across CRP Groups				
	BMI (mg/L)			
CRP (mg/L)	**Mean**	**Minimum**	**Maximum**	**No of Cases**
NEGATIVE	29.84	24.00	39.00	38.00
POSITIVE(MR)	31.67	26.00	40.00	39.00
POSITIVE(HR)	31.78	25.00	38.00	23.00
Total	31.00	24.00	40.00	100.00

ANOVA					
BMI (Kg/m^2)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	82.37	2	41.18	3.08	0.05
Within Groups	1297.63	97	13.38		
Total	1380	99			



Multiple Comparisons						
BMI (Kg/m^2) Tukey HSD						
(I) CRP	(J) CRP	Mean Difference (I-J)	Std. Error	**P Value**	95% Confidence Interval	
					Lower Bound	Upper Bound
NEGATIVE	POSITIVE(MR)	-1.82	0.83	0.08	-3.81	0.16
	POSITIVE(HR)	-1.94	0.97	0.12	-4.24	0.36
POSITIVE(MR)	NEGATIVE	1.82	0.83	0.08	-0.16	3.81
	POSITIVE(HR)	-0.12	0.96	0.99	-2.40	2.17
POSITIVE(HR)	NEGATIVE	1.94	0.97	0.12	-0.36	4.24
	POSITIVE(MR)	0.12	0.96	0.99	-2.17	2.40

Correlations			
		CRP_Cal	
BMI **(Kg/m^2)**	Pearson Correlation	0.22	
	P Value (2-tailed)	0.03	
	No of Cases	100	
*. Correlation is P Valuenificant at the 0.05 level (2-tailed).			

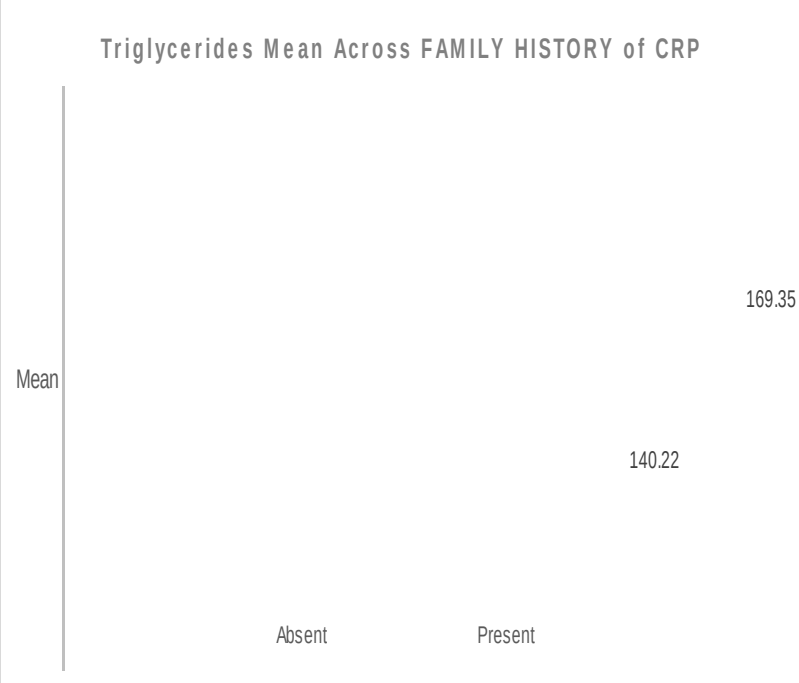


INTERPRETATION

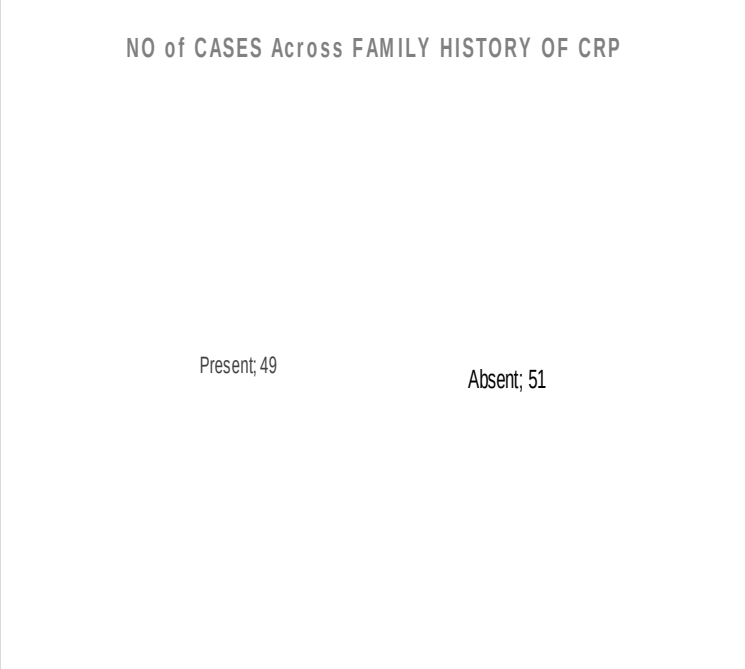
- The mean of BMI (Kg/m^2) for negative CRP is 29.84 and it is 31.67 and 31.78 for POSITIVE (MR) and POSITIVE (HR) respectively.
- The ANOVA test indicates that the Average BMI is not highly significant between the CRP Categories as the P value is 0.51.

- ❖ Therefore the conclusion is that BMI levels are not different between either of the CRP categories.

Triglycerides Average Across Family History				
	triglycerides			
Family_History	Mean	Minimum	Maximum	No of Cases
Absent	140.22	121.00	178.00	51
Present	169.35	133.00	189.00	49
Total	154.49	121.00	189.00	100



ANOVA					
triglycerides					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	21207.26051	1	21207.26	100.26	0.00
Within Groups	20729.72949	98	211.53		
Total	41936.99	99			

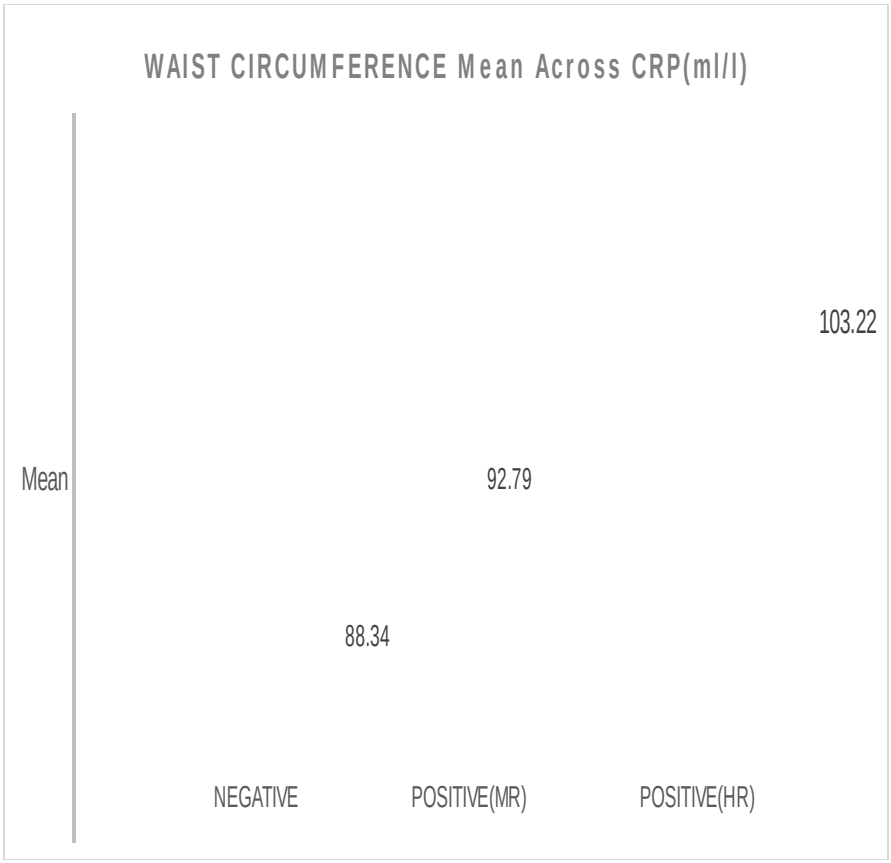


INTERPRETATION

- The mean of Triglycerides for absent family history is 140.22 and 169.35 for present family history.
- The Anova table indicates that the mean level between both the categories is highly significantly different.
- The conclusion is that the average level of Triglycerides for present family history is significantly higher than absent family history.

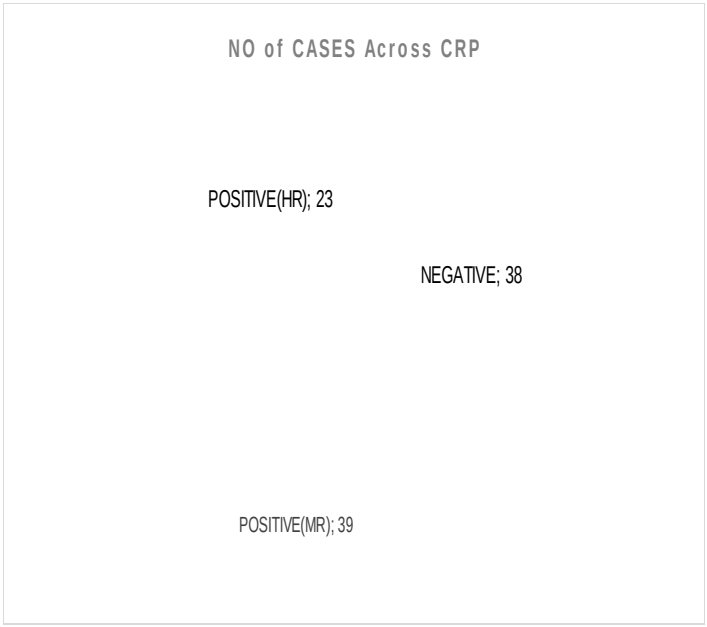
WC Average Across CRP

	WC (cm)			
CRP (ml/L)	**Mean**	**Minimum**	**Maximum**	**No of Cases**
NEGATIVE	88.34	81.00	103.00	38
POSITIVE(MR)	92.79	78.00	104.00	39
POSITIVE(HR)	103.22	98.00	112.00	23
Total	93.50	78.00	112.00	100



ANOVA					
WC (cm)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	3202.175351	2	1601.09	53.99	0.00
Within Groups	2876.824649	97	29.66		
Total	6079	99			

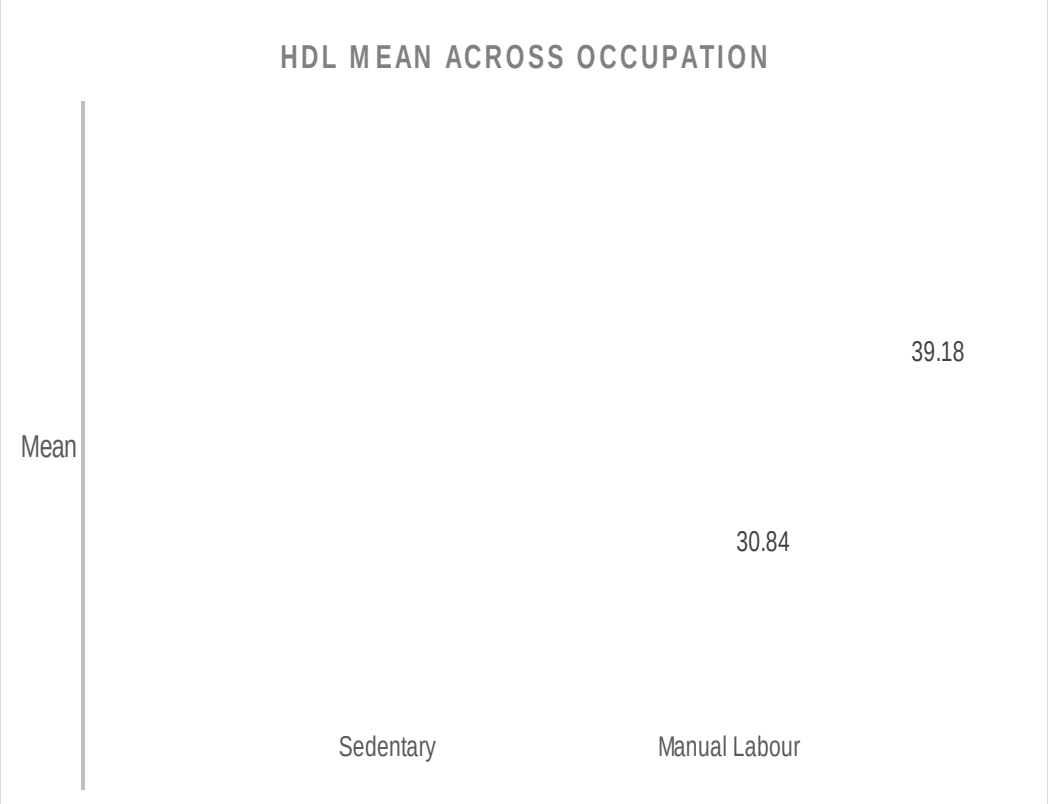
Multiple Comparisons						
WC (cm) Tukey HSD						
(I) CRP (ml/L)	(J) CRP	Mean Difference (I-J)	Std. Error	P Value	95% Confidence Interval	
					Lower Bound	Upper Bound
NEGATIVE	POSITIVE(MR)	-4.45	1.24	0.00	-7.41	-1.50
	POSITIVE(HR)	-14.88	1.44	0.00	-18.30	-11.45
POSITIVE(MR)	NEGATIVE	4.45	1.24	0.00	1.50	7.41
	POSITIVE(HR)	-10.42	1.43	0.00	-13.83	-7.01
POSITIVE(HR)	NEGATIVE	14.88	1.44	0.00	11.45	18.30
	POSITIVE(MR)	10.42	1.43	0.00	7.01	13.83
*. The mean difference is P Value significant at the 0.05 level.						



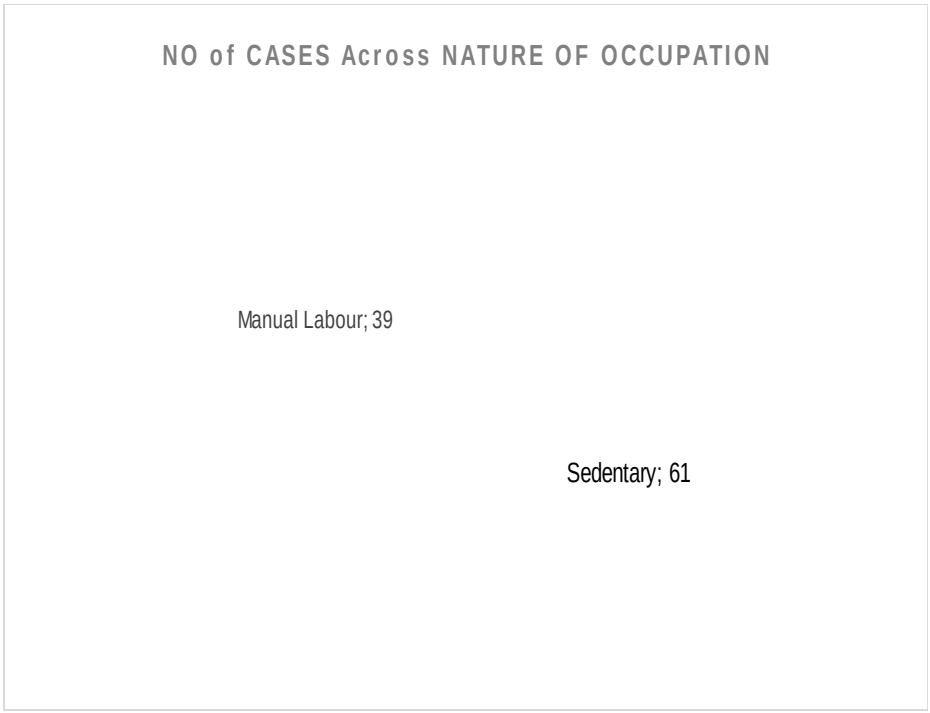
INTERPRETATION

- The average of WC for Negative CRP (mg/L) is 88.34 and 92.79 and 103.22 for POSITIVE MR and POSITIVE HR categories respectively.
- The Anova table indicates that the mean level between one of the categories are highly significantly different.
- The Multiple Comparison tukey test confirms that the average levels between all three categories are significantly different.
- The conclusion is that the average levels of WC are highest for POSITIVE HR CRP, second highest for POSITIVE MR CRP and Least for NEGATIVE CRP.

HDL Average Across Occupation				
	HDL (mg/dl)			
Occupation	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Sedentary	30.84	21	41	61
Manual Labour	39.18	26	54	39
Total	34.09	21	54	100



ANOVA					
HDL(mg/dl)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	1656.085755	1	1656.085755	56.86421728	0.00
Within Groups	2854.104245	98	29.12351271		
Total	4510.19	99			

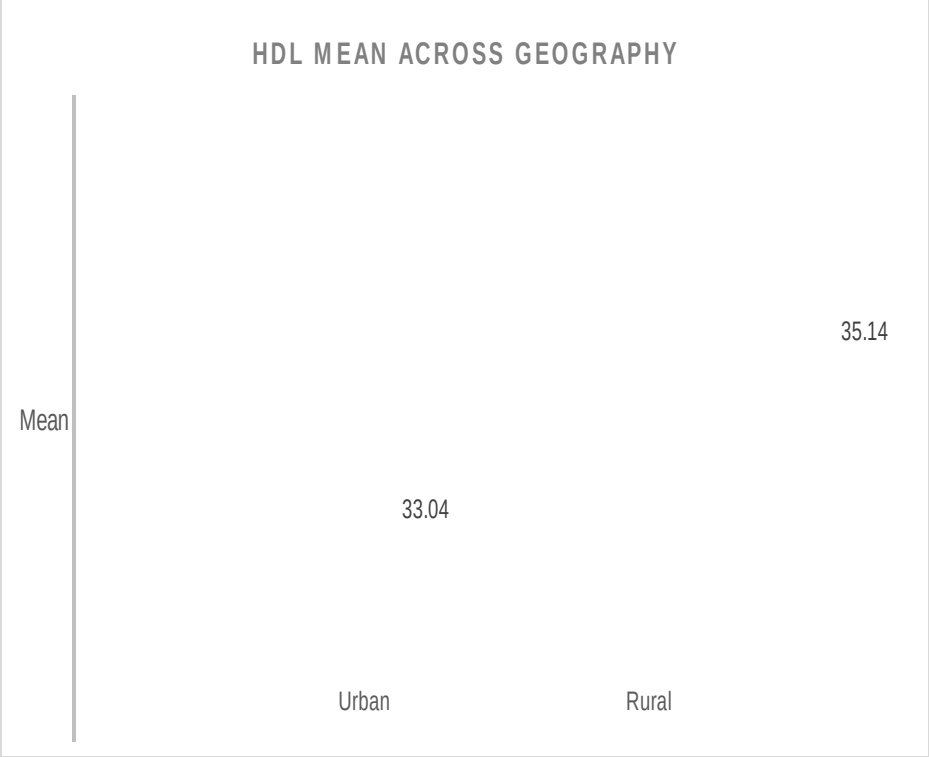


INTERPRETATION

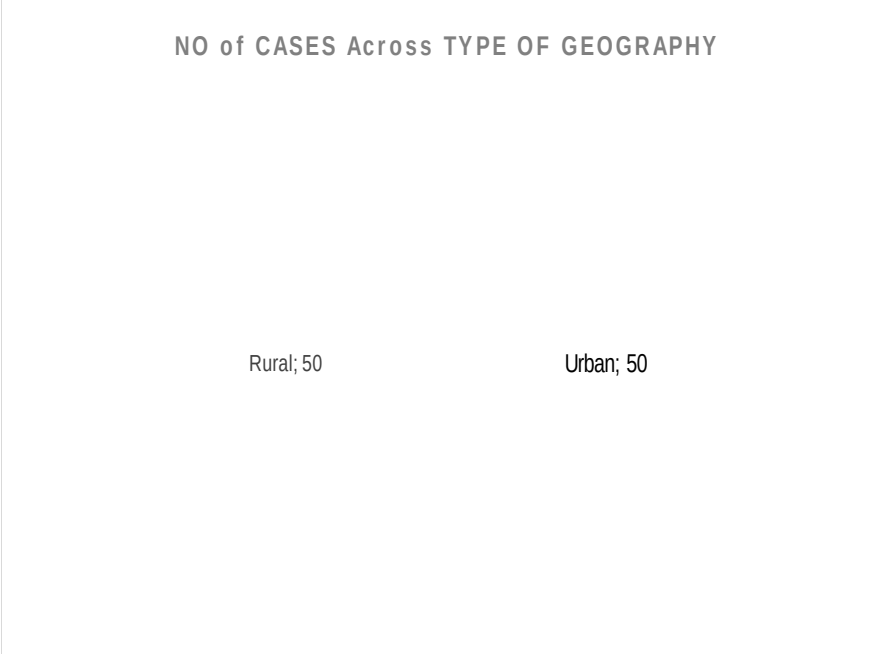
- The mean of HDL for Sedentary Occupation is 30.84 and 39.18 for Manual Labor.
- The Anova table indicates that the mean level between both the categories is highly significantly different.
- The conclusion is that the average level of HDL for Manual Labor is significantly higher than Sedentary Occupation

HDL Average Across Rural and Urban Areas	
	HDL (mg/dl)

Rural_Urban_Cal	Mean	Minimum	Maximum	No of Cases
Urban	33.04	21	54	50
Rural	35.14	21	45	50
Total	34.09	21	54	100



ANOVA					
HDL (mg/dl)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	110.25	1	110.25	2.455601667	0.12
Within Groups	4399.94	98	44.89734694		
Total	4510.19	99			



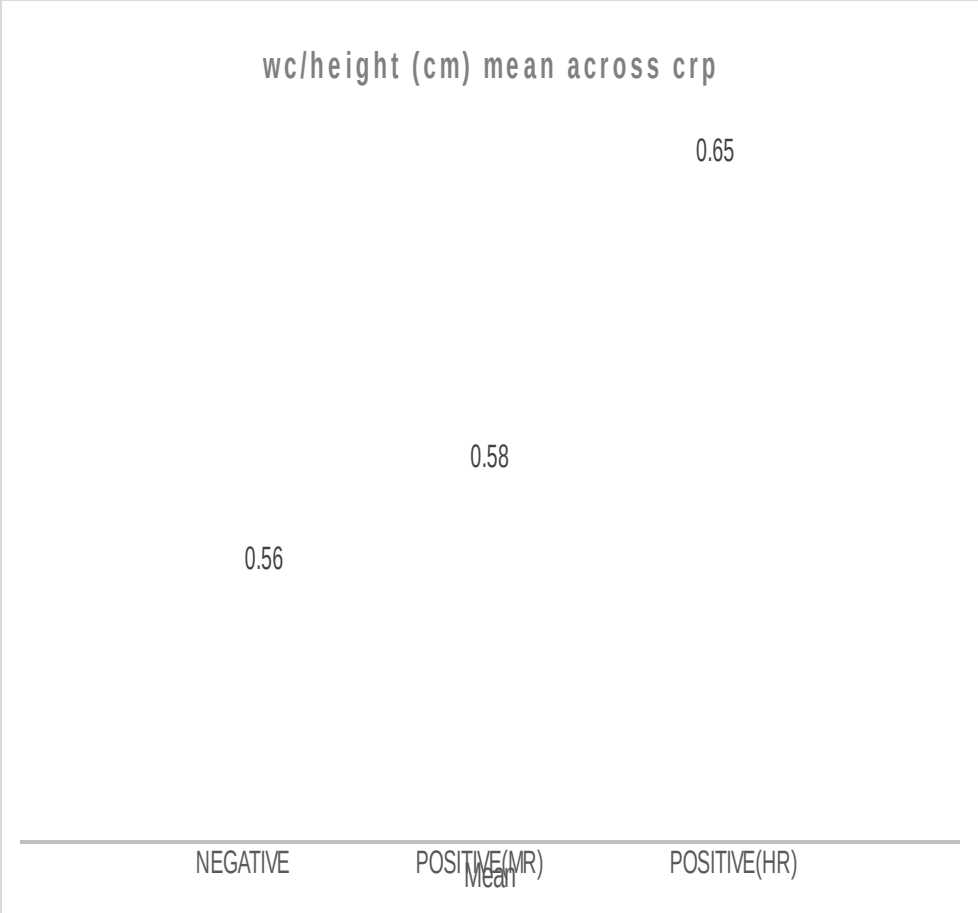
INTERPRETATION

- The mean of HDL (mg/dl) for Urban area is 33.04 and 35.14 for Rural Area.
- The Anova table indicates that the mean level between both the categories is not significantly different.
- The conclusion is that the average level of HDL for Urban area is not significantly different from rural area.

WC/Height Average Across CRP				
	WC/HEIGHT			
CRP (mg/L)	Mean	Minimum	Maximum	No of Cases
NEGATIVE	0.56	0.48	0.65	38
POSITIVE(MR)	0.58	0.47	0.66	39
POSITIVE(HR)	0.65	0.61	0.71	23
Total	0.59	0.47	0.71	100

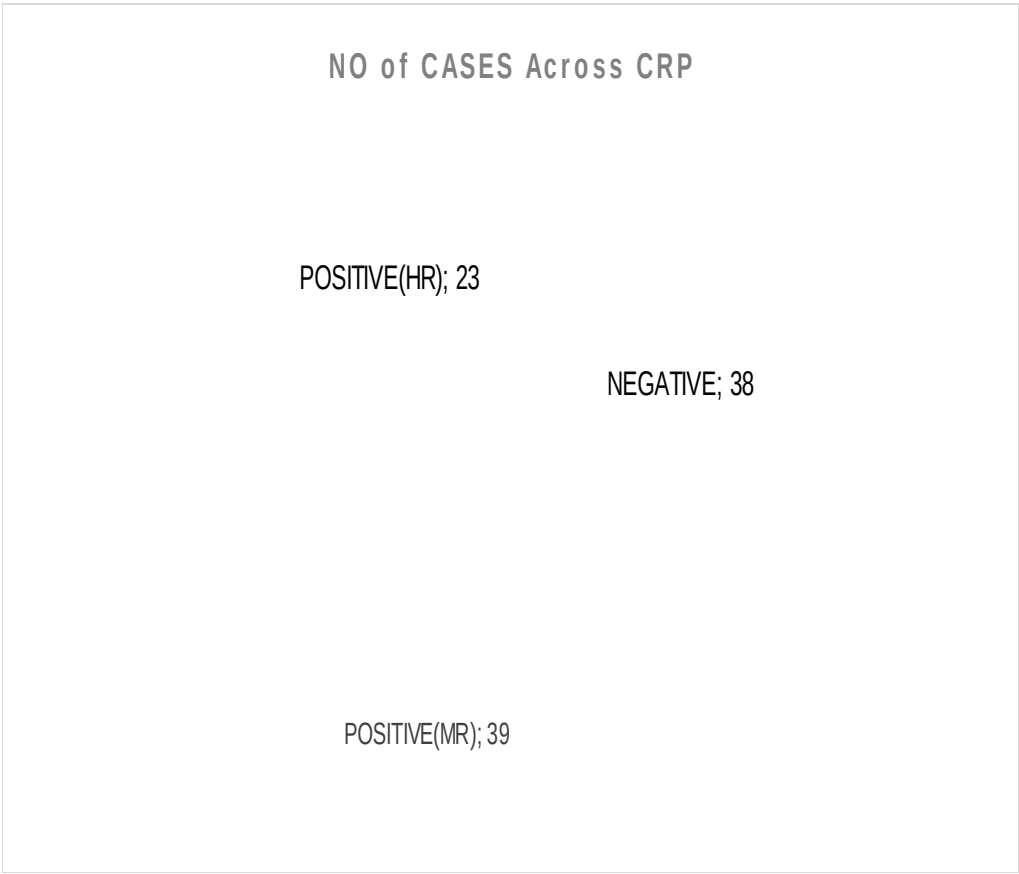
ANOVA					
WC/HEIGHT (cm)					

	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	0.119172382	2	0.059586191	37.02864112	0.00
Within Groups	0.156091618	97	0.001609192		
Total	0.275264	99			



Multiple Comparisons						
WC_BY_HEIGHT						

(cm) Tukey HSD						
(I) CRP (mg/L)	(J) CRP (mg/L)	Mean Difference (I-J)	Std. Error	P Value	95% Confidence Interval	
					Lower Bound	Upper Bound
NEGATIVE	POSITIVE(MR)	-0.02	0.01	0.04	-0.04	0.00
	POSITIVE(HR)	-0.09	0.01	0.00	-0.12	-0.06
POSITIVE(MR)	NEGATIVE	0.02	0.01	0.04	0.00	0.04
	POSITIVE(HR)	-0.07	0.01	0.00	-0.09	-0.04
POSITIVE(HR)	NEGATIVE	0.09	0.01	0.00	0.06	0.12
	POSITIVE(MR)	0.07	0.01	0.00	0.04	0.09
* The mean difference is P Value significant at the 0.05 level.						

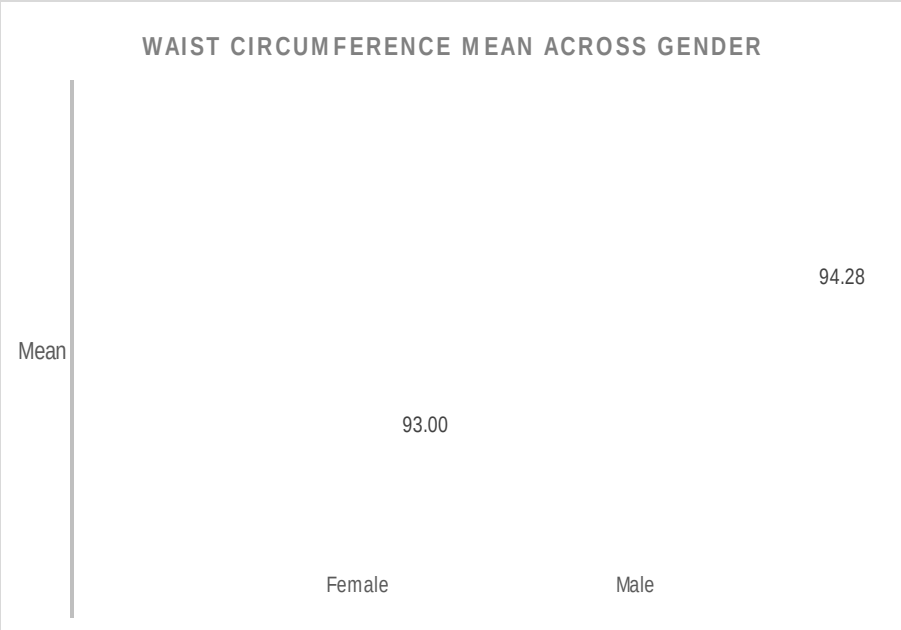


INTERPRETATION

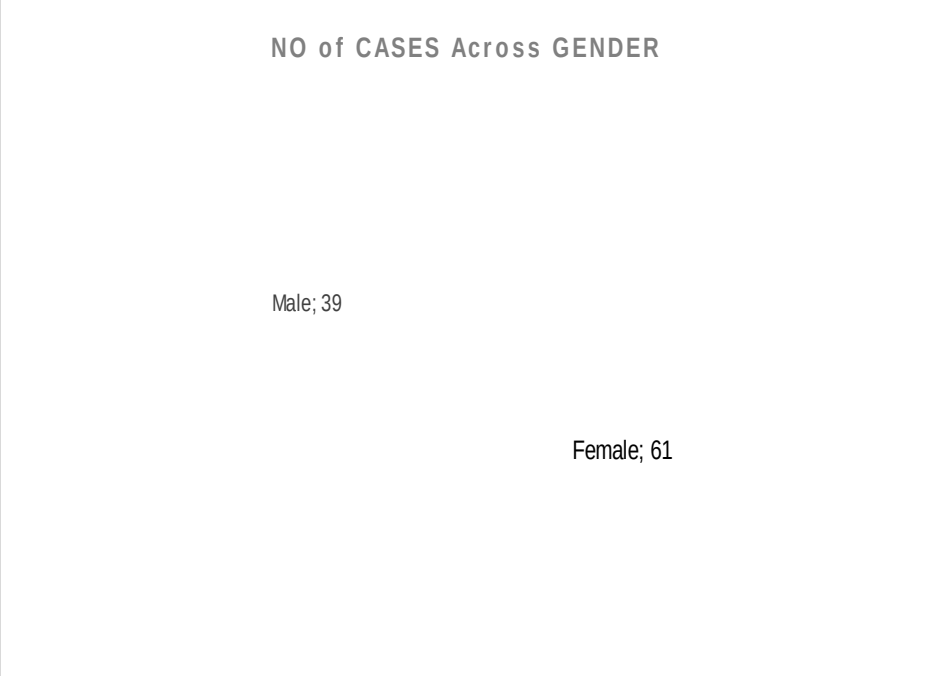
- The mean of WC/Height (cm) for NEGATIVE CRP Level is 0.56, and 0.58 and 0.65 for POSITIVE (MR) and POSITIVE (HR) respectively.
- The Anova table indicates that the mean level between one of the three categories are significantly different.
- The multiple comparison tukey tests indicate that mean levels between all the three categories are significantly different.
- The conclusion is that the mean level of WC/Height is highest for POSITIVE (HR) CRP, second highest for POSITIVE (MR) CRP and least for NEGATIVE CRP.

WC Average Across Gender				
	WC (cm)			
Gender	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Female	93.00	81.00	112.00	61
Male	94.28	78.00	106.00	39

Total	93.5	78	112	100



ANOVA					
WC (cm)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	39.10	1	39.10	0.63	0.43
Within Groups	6039.90	98	61.63		
Total	6079	99			

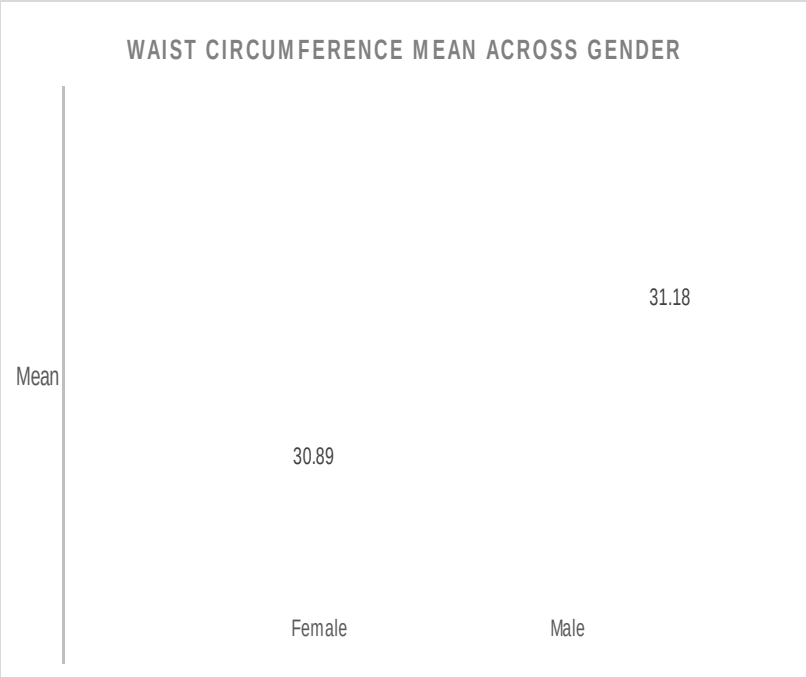


INTERPRETATION

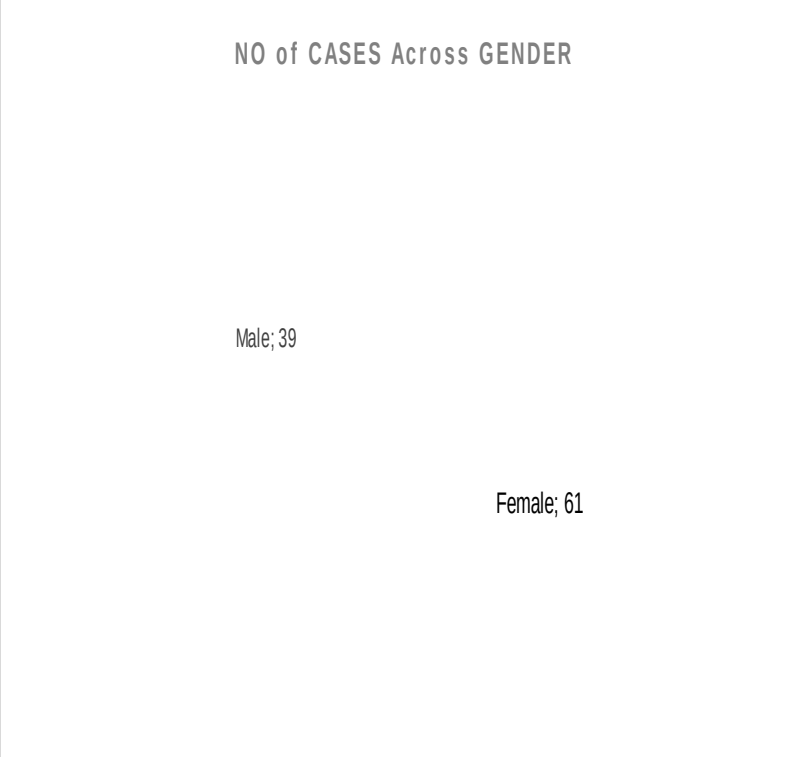
- The mean of WC (cm)for Female Gender is 93 and for Male Gender is 94.28.
- The Anova table indicates that the mean level between both the categories are not significantly different.
- The conclusion is that the average level of WC is not different between Female and Male and that both are almost the same.

BMI Average Across Gender				
	BMI			
Gender	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Female	30.89	24.00	40.00	61
Male	31.18	26.00	38.00	39

Total	31	24	40	100



ANOVA					
BMI (Kg/m^2)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	2.06	1.00	2.06	0.15	0.70
Within Groups	1377.94	98.00	14.06		
Total	1380	99			

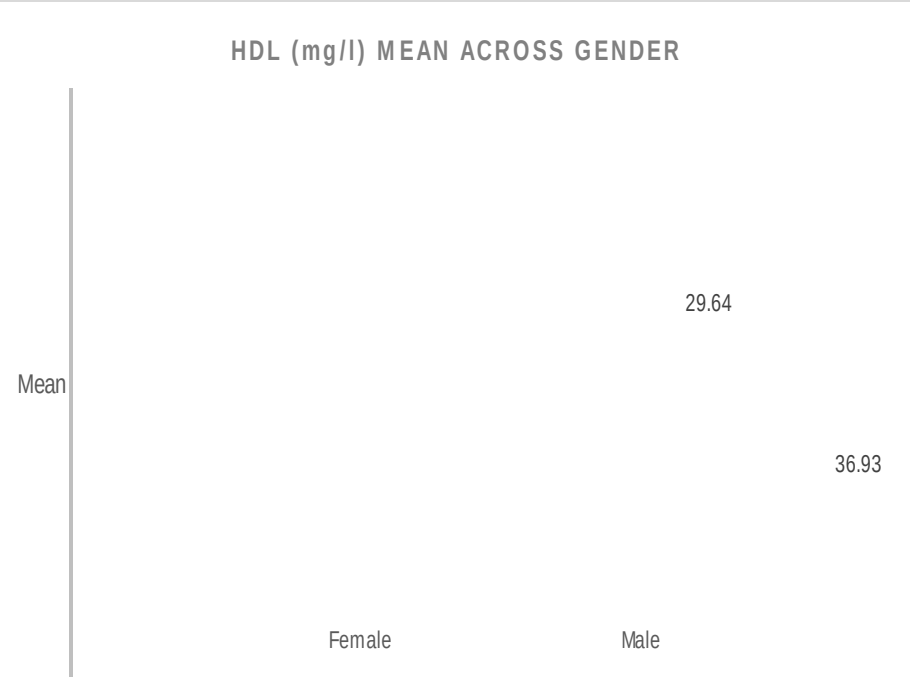


INTERPRETATION

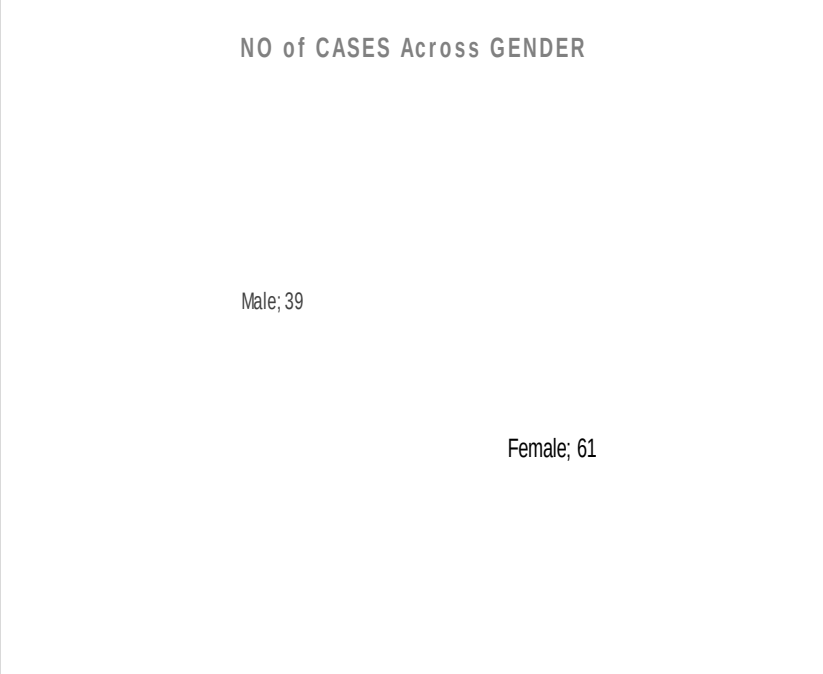
- The mean of BMI (kg/m^2) for Female Gender is 30.89 and for Male Gender is 31.18.
- The Anova table indicates that the mean level between both the categories is not significantly different.
- The conclusion is that the average level of BMI is not different between Female and Male and that both are almost the same.

HDL Average Across Gender	
	HDL (mg/dl)

Gender	Mean	Minimum	Maximum	No of Cases
Female	36.93	30.00	54.00	61
Male	29.64	21.00	42.00	39
Total	34.09	21	54	100



ANOVA					
HDL (mg/L)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	1265.48	1.00	1265.48	38.22	0.00
Within Groups	3244.71	98.00	33.11		
Total	4510.19	99			

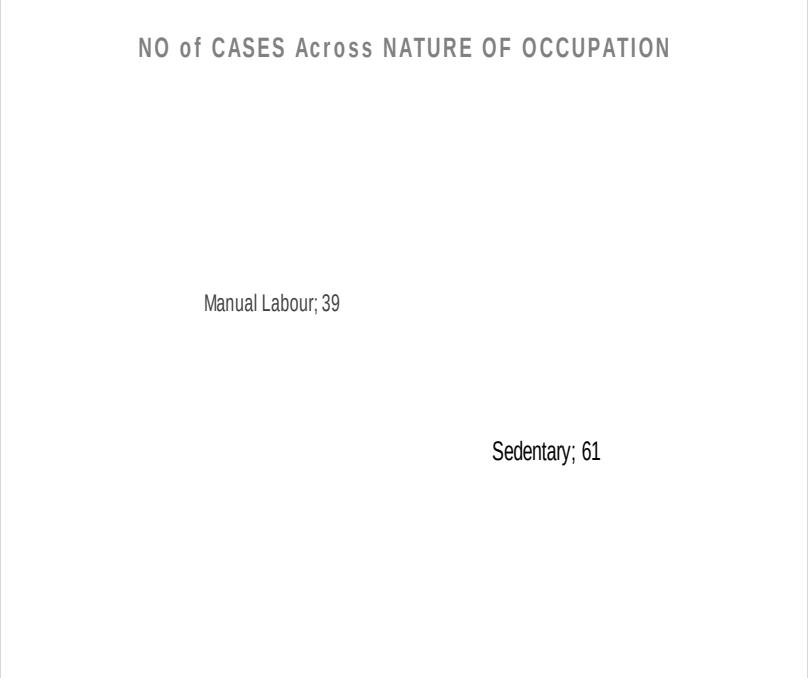


INTERPRETATION

- The mean of HDL (mg/L) for Female Gender is 36.93 and for Male Gender is 29.64.
- The Anova table indicates that the mean level between both the categories are significantly different.
- The conclusion is that the average level of HDL is highest for Female and as compared to Male .

WC Average Across Occupation				
	WC (cm)			
Occupation	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Sedentary	97.07	78.00	112.00	61
Manual Labour	87.92	81.00	102.00	39
Total	93.5	78	112	100

ANOVA					
WC					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	1988.49	1.00	1988.49	47.64	0.00
Within Groups	4090.51	98.00	41.74		
Total	6079	99			

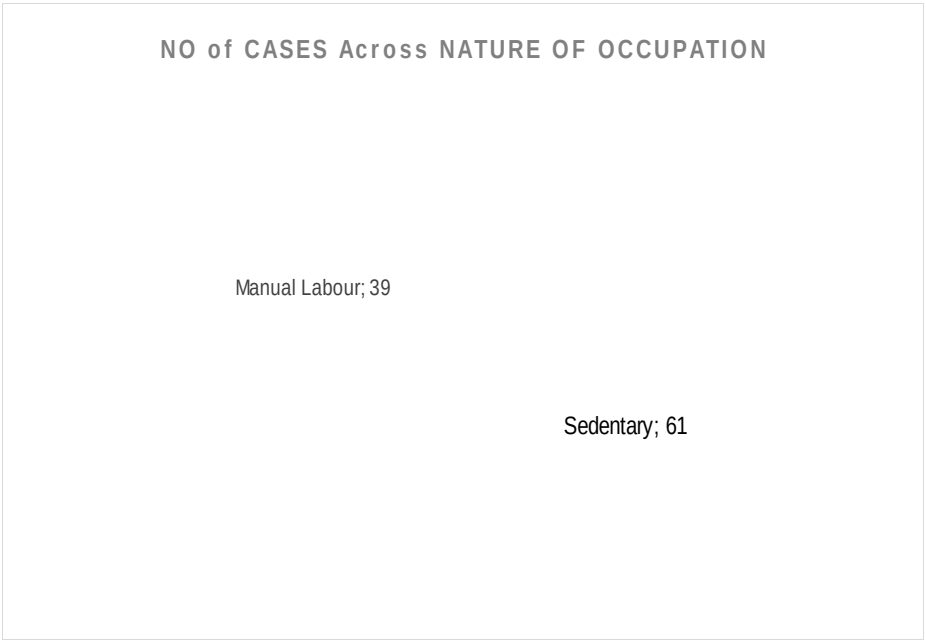


INTERPRETATION

- The mean of WC (cm) for Sedentary Occupation is 97.07 and for Manual Labor is 87.92.
- The Anova table indicates that the mean level between both the categories are significantly different.
- The conclusion is that the average level of WC is highest for Sedentary occupation as compared to Manual Labor

.	BMI (Kg/m^2) Average Across Occupation			
	BMI (Kg/m^2)			
Occupation	**Mean**	**Minimum**	**Maximum**	**No of Cases**
Sedentary	31.16	24.00	38.00	61
Manual Labour	30.74	26.00	40.00	39
Total	31	24	40	100

ANOVA					
BMI (Kg/m^2)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	4.20	1.00	4.20	0.30	0.59
Within Groups	1375.80	98.00	14.04		
Total	1380	99			

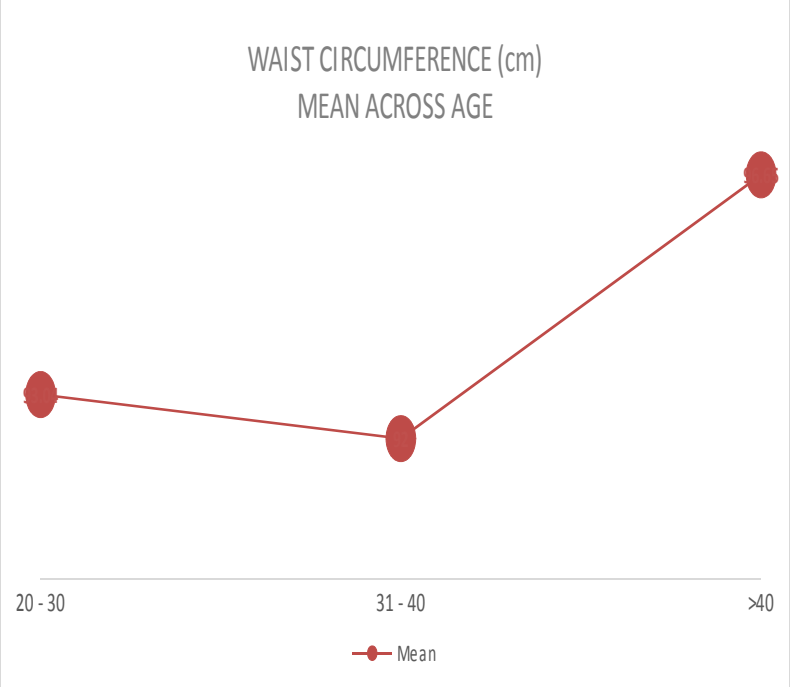


INTERPRETATION

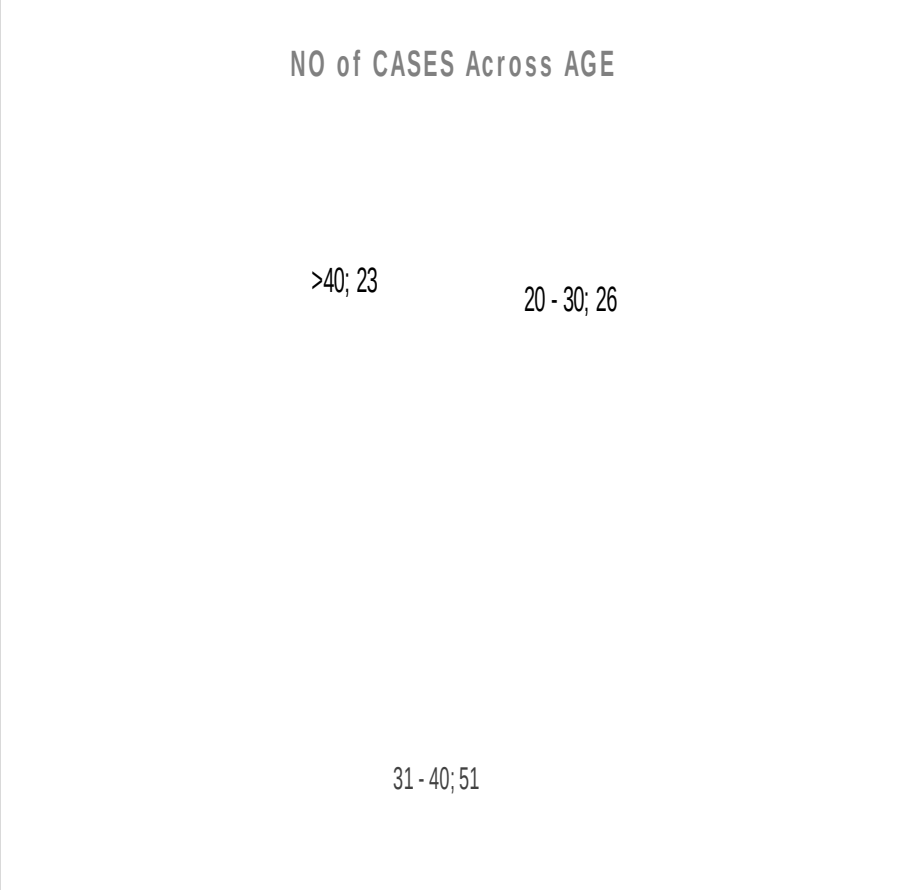
- The mean BMI **(Kg/m²)** for Sedentary Occupation is 31.16 and for Manual Labour is 30.74.
- The Anova table indicates that the mean level between both the categories are not significantly different .
- The conclusion is that the average level of BMI is not different between either of the occupation.

WC (cm) Average Across Age				
	WC (cm)			
Age	**Mean**	**Minimum**	**Maximum**	**No of Cases**
20 - 30	93.04	81.00	106.00	26
31 - 40	92.31	78.00	112.00	51
>40	96.65	82.00	108.00	23
Total	93.50	78.00	112.00	100

ANOVA					
WC					**P Value**
	Sum of Squares	df	Mean Square	F	0.08
Between Groups	305.84	2.00	152.92	2.57	
Within Groups	5773.16	97.00	59.52		
Total	6079.00	99.00			



Multiple Comparisons						
WC (cm) Tukey HSD						
(I) Age	**(J) Age**	Mean Difference (I-J)	Std. Error	P Value	95% Confidence Interval	
					Lower Bound	Upper Bound
20 - 30	31 - 40	0.72	1.86	0.92	-3.70	5.15
	> 40	-3.61	2.21	0.24	-8.87	1.64
31 - 40	20 - 30	-0.72	1.86	0.92	-5.15	3.70
	> 40	-4.34	1.94	0.07	-8.95	0.27
> 40	20 - 30	3.61	2.21	0.24	-1.64	8.87
	31 - 40	4.34	1.94	0.07	-0.27	8.95

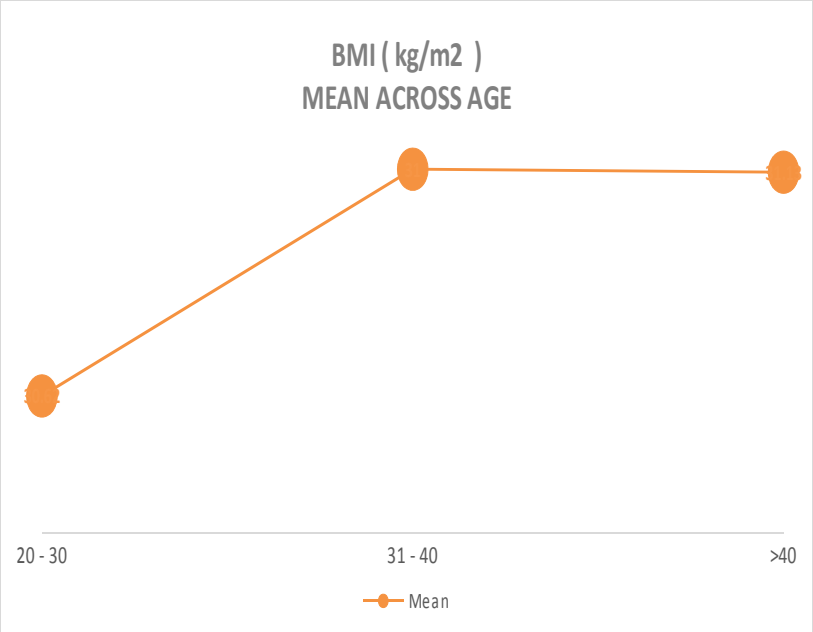


INTERPRETATION

- The mean WC (cm) in the age group of 20 to 30 is 93.04, 31 to 40 is 92.31 and greater than 40 is 96.65.
- The Anova table indicates that the mean level between both the categories are not significantly different
- The conclusion is that the average level of WC is not different between the age categories

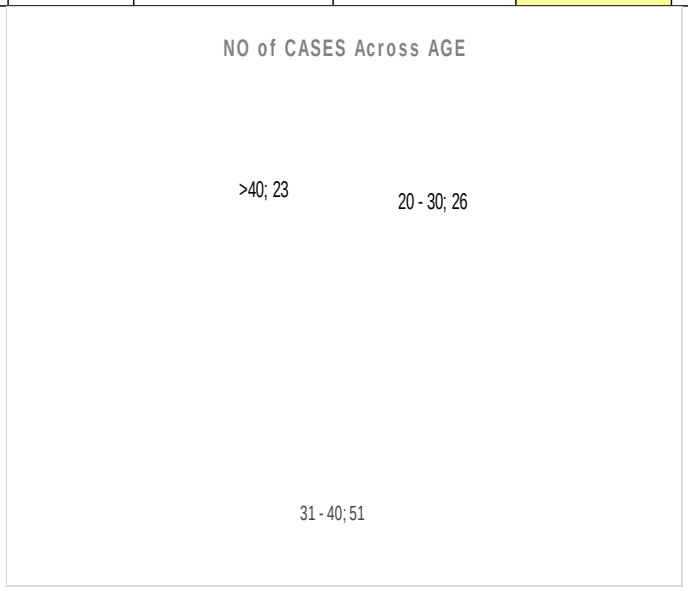
Average BMI Across Age				
	BMI (Kg/m^2)			
Age	**Mean**	**Minimum**	**Maximum**	**No of Cases**
20 - 30	30.62	25.00	40.00	26
31 - 40	31.14	24.00	39.00	51
>40	31.13	25.00	38.00	23
Total	31	24	40	100

ANOVA					
BMI (Kg/m^2)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	5.20	2.00	2.60	0.18	0.833
Within Groups	1374.80	97.00	14.17		
Total	1380	99			



Multiple Comparisons						
BMI (Kg/m^2)						

Tukey HSD						
(I) Age	(J) Age	Mean Difference (I-J)	Std. Error	P Value	95% Confidence Interval	
					Lower Bound	Upper Bound
20 - 30	31 - 40	-0.52	0.91	0.83	-2.68	1.64
	> 40	-0.52	1.08	0.88	-3.08	2.05
31 - 40	20 - 30	0.52	0.91	0.83	-1.64	2.68
	> 40	0.01	0.95	1.00	-2.24	2.26
> 40	20 - 30	0.52	1.08	0.88	-2.05	3.08
	31 - 40	-0.01	0.95	1.00	-2.26	2.24

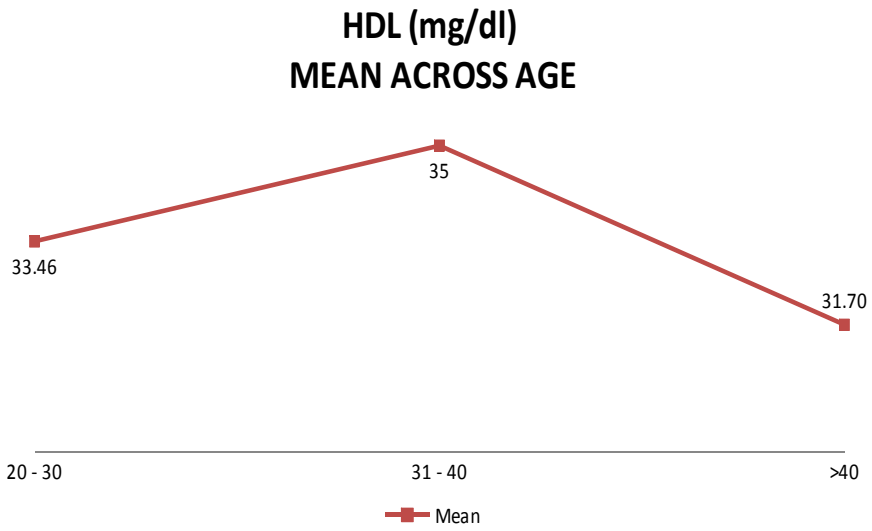


INTERPRETATION

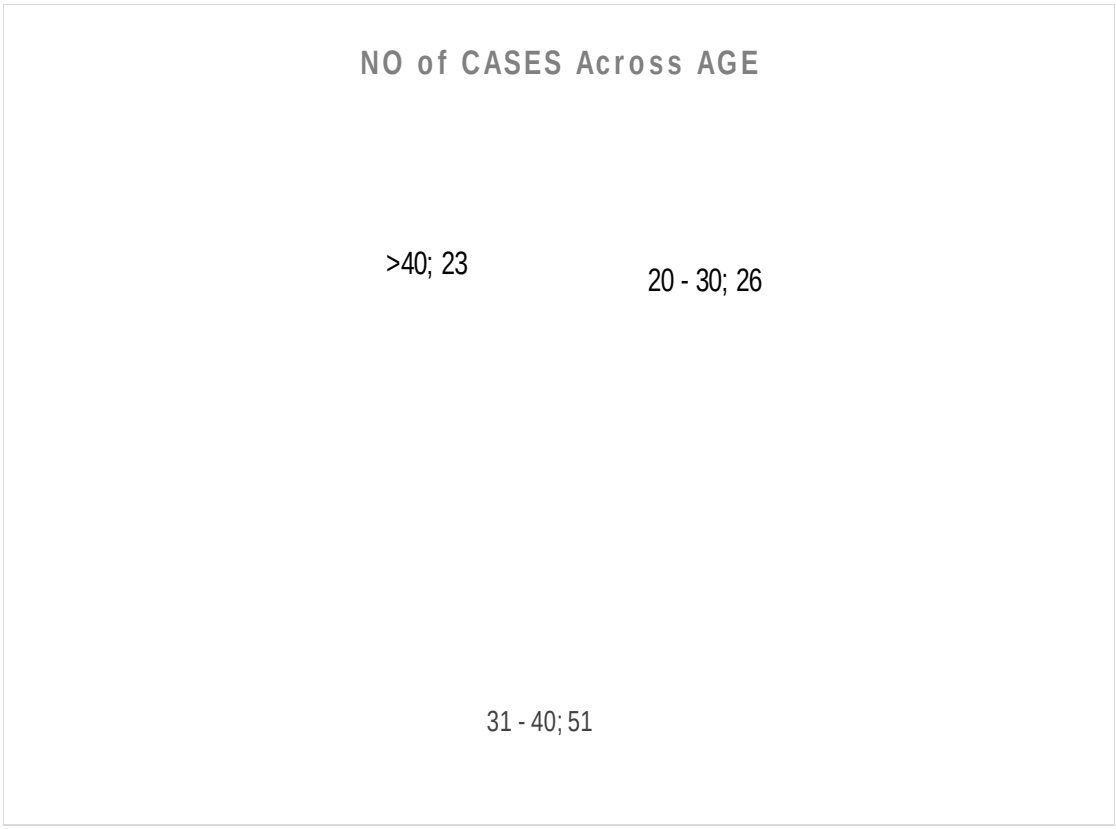
- The mean BMI **(Kg/m²)** in the age category of 20 to 30 is 30.62 and 31.14 and 31.13 for 31 to 40 and greater than 40 respectively.
- The Anova table indicates that the mean level between both the categories are not significantly different .
- The conclusion is that the average level of BMI is not different between the age categories .

Average HDL Across Age				
	HDL (mg/dl)			
Age	**Mean**	**Minimum**	**Maximum**	**No of Cases**
20 - 30	33.46	21.00	44.00	26
31 - 40	35.49	21.00	54.00	51
>40	31.70	21.00	45.00	23
Total	34.09	21	54	100

ANOVA					
HDL (mg/dl)					
	Sum of Squares	df	Mean Square	F	**P Value**
Between Groups	242.11	2.00	121.06	2.75	0.07
Within Groups	4268.08	97.00	44.00		
Total	4510.19	99.00			



Multiple Comparisons						
HDL (mg/dl) Tukey HSD						
(I) Age_Cutoff	(J) Age_Cutoff	Mean Difference (I-J)	Std. Error	P Value	95% Confidence Interval	
					Lower Bound	Upper Bound
20 - 30	31 - 40	-2.03	1.60	0.42	-5.83	1.78
	> 40	1.77	1.90	0.62	-2.75	6.29
31 - 40	20 - 30	2.03	1.60	0.42	-1.78	5.83
	> 40	3.79	1.67	0.06	-0.17	7.76
> 40	20 - 30	-1.77	1.90	0.62	-6.29	2.75
	31 - 40	-3.79	1.67	0.06	-7.76	0.17



INTERPRETATION

- The mean HDL (mg/dl) in the age category of 20 to 30 is 33.46 and 35.49 and 31.70 for 31 to 40 and greater than 40 respectively.
- The Anova table indicates that the mean level between both the categories is not significantly different.
- The conclusion is that the average level of HDL is not different between the age categories .

DISCUSSION

DISCUSSION

Obesity is a chronic inflammatory disorder which is associated with increase cardio metabolic morbidity and mortality.

It is defined with various anthropometric measurements like body mass index (BMI), weight circumference, waist height ratio and etc.

In obese population dysfunctional adipocyte was associated with increase level of inflammatory markers. This marker indicates future outcome of obesity related disorders. In our study serum C-reactive protein is taken as an inflammatory marker. In our present study 100 sample population was taken. CRP level less than 1 mg/l was taken as

CRP negative. More than 1 mg/l was positive. In hundred patients, 62 persons are having CRP level more than 1 mg/l.

In our study, the mean of SERUM CRP for 25Kg/m^2 to 29Kg/m^2 BMI cutoff is 1.23 and greater than 30 BMI cutoff is 2.47. The SERUM CRP is highest for BMI cutoff greater than 30 as compared to BMI cutoff for 25 to 29.

In present study the mean of BMI for Rural Female is 29.27, Rural Male is 29.55, Urban Female is 32.45 and Urban Male is 32.89. Mean levels of BMI is highest for urban female as compared to rural female. Mean levels of BMI is highest for urban male as compared to rural male. Mean levels of BMI is highest for urban female as compared to rural male. Ghosh A [62] study states that obesity prevalence not affected with particular residence. Study by Shah Ebrahim [63] states that in Urban Migration population Obesity rate was high.

The mean of SERUM CRP for Urban area is 2.62 and 1.47 for rural area. The SERUM CRP is highest for urban area as compared to rural area.

In our study the mean of SERUM CRP is 2.11 for Female Sedentary occupation, 1.51 for Female Manual Labor, 3.13 for Male Sedentary occupation and 1.25 for Male manual labor. Also, SERUM

CRP is highest for Male Sedentary occupation as compared to Female Sedentary Occupation. Choi. J et al study also shows that increase level of CRP is stronger in female than male.

In our study, the mean of SERUM CRP for Rural Female is 1.49, Rural Male is 1.44, Urban Female is 2.30 and Urban Male is 3.15. However between rural male and rural female there is no significant difference between SERUM CRP. Also no significance difference between urban male and urban female there is no significant difference between SERUM CRP.

The average of WC for Negative CRP is 88.34 and 92.79 and 103.22 for POSITIVE MR and POSITIVE HR categories respectively. The conclusion is that the average levels of WC are highest for POSITIVE HR CRP, second highest for POSITIVE MR CRP and Least for NEGATIVE CRP. Increase in waist circumference was associated with increase CRP level. It is an important predictor to access the rick for IHD and DM.

In the article of Hirofumi Nakamura [64] and Emanuela Lapice [65], studies states that Waist circumference is the main determinant in obesity to identify high risk group. It is independent of BMI. In our study also

increase Waist circumference in more than 90cm was associated with increase CRP level.

The mean of WC for Female Gender is 93 and for Male Gender is 94.28. The average level of WC is not different between Female and Male and that both are almost the same. The mean of WC for Sedentary Occupation is 97.07 and for Manual Labor is 87.92 . The average level of WC is highest for sedentary occupation as compared to Manual Labor.

The mean of WC/Height for NEGATIVE CRP Level is 0.56, and 0.58 and 0.65 for POSITIVE (MR) and POSITIVE (HR) respectively. The mean level of WC/Height is highest for POSITIVE (HR) CRP, second highest for POSITIVE (MR) CRP and least for NEGATIVE CRP. Waist height ratio also very important measurement to predict cardio metabolic risk in obese people.

The mean of SERUM CRP for absent family history is 1.37 and 2.74 for present family history. The SERUM CRP is highest for positive family history as compared to absent family history. In the article of CDC features study on Obesity & Genetics and Lisa Y Gibson [66] studies states that no clear evidence for family history and obesity. But it also states that not only genetic influence, environmental factor the family

member shared, eating habits, and physical inactivity influence the obesity of the particular person. In our study family style is a significance impact on obesity.

The mean of SERUM CRP for sedentary occupation is 2.46 and 1.39 for manual labor. The SERUM CRP is highest for Sedentary Occupation as compared to manual labor occupation.

The mean of SERUM CRP for Female Gender is 1.9 and 2.27 for Male Gender. The conclusion is that SERUM CRP is not very different between the gender categories.

SUMMARY

SUMMARY

In our study the SERUM CRP is highest for BMI cutoff greater than 30 as compared to BMI cutoff for 25Kg/m^2 to 29Kg/m^2

Mean levels of BMI is highest for urban female as compared to rural female.

Mean levels of BMI is highest for urban male as compared to rural male.

Mean levels of BMI is highest for urban female as compared to rural male.

No significant difference between SERUM CRP of rural male and rural female, urban male and urban female.

The SERUM CRP is highest for urban area as compared to rural area.

SERUM CRP is highest for Male Sedentary occupation as compared to Female Sedentary Occupation .

The average levels of WC are highest for POSITIVE (HR) CRP, second highest for POSITIVE (MR) CRP and Least for NEGATIVE CRP.

In our study increase waist circumference in more than 90cm was associated with increase CRP level.

The average level of WC is highest for sedentary occupation as compared to Manual Labor.

The mean level of WC/Height is highest for POSITIVE (HR) CRP, Second highest for POSITIVE (MR) CRP and least for NEGATIVE CRP.

The SERUM CRP is highest for positive family history as compared to absent family history.

The conclusion is that SERUM CRP is not very different between the gender categories.

CONCLUSION

CONCLUSION

Obesity is a slowly progressive inflammation Serum CPR level is most powerful marker for Cardio metabolic risk. Abdominal obesity was associated with high level of Serum CRP. Early life style modification like regular physical exercise and avoiding high calorie diet will help to improve the quality of life, Family members also to be included in the life style modification. Statin has both lipid lowering effect and Anti inflammatory effect. The use of Statin in obese person with normal lipid profile with elevated CRP level can be evaluated by further studies.

PROFORMA

PROFORMA

Serial No :

Patient information

Name : Patient No :
Income :

Age : Sex : Address:

Occupation : Menstrual history :

Past History

Diabetes :

Hypertension :

Past drug History :

Cardiac disease :

Associated illness :

Family History :

General physical examination

Pulse : Pallor : Emaciation : Pedal edema :

BP : Icterus : Oral thrush : Facial puffiness:

RR : Cyanosis : Clubbing : Lymphadenopathy:

Temp : Weight in Kg: Height in cm: Waist circumference in cm :

CVS :

RS :

PA :

CNS :

INVESTIGATIONS

Blood sugar :

Lipid Profile :

Serum C-reactive Protein :

CONSENT FORM

CONSENT FORM

Yourself Mr./Mrs./Ms. …… are being asked to be a participant in the research study titled "A STUDY ON SERUM C-REACTIVE PROTEIN LEVEL IN OBESITY" in CMC hospital, Coimbatore , conducted by Dr. T. Dhanalakshmi, Post Graduate Student, Department of General Medicine, Coimbatore Medical College. You are eligible after looking into the inclusion criteria .You can ask any question which you may have before agreeing to participate.

Research Being Done

A STUDY ON SERUM C-REACTIVE PROTEIN LEVEL IN OBESITY

Purpose of Research

- To test whether overweight and obesity are associated with low-grade systemic inflammation as measured by serum C-reactive protein (CRP) level.
- To study the c reactive protein level in obesity using measures of body mass index.
- To study CRP level in obese women and men.

Decline from Participation

You have the option to decline from participation in the study existing protocol for your condition.

Privacy and Confidentiality

Privacy of individuals will be respected and any information about you or provided by you during the study will be kept strictly confidential.

Authorization to publish Results

Results of the study may be published for scientific purposes and/or presented to scientific groups, however you will not be identified.

Statement of Consent

I volunteer and consent to participate in this study. I have read the consent or it has been read to me. The study has been fully explained to me, and I may questions at any time.

…….

……...

Signature/Left thumb impression Date

(Volunteer)

…….

……

Signature of witness

Date

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Gene	Gene Product	Mechanism of Obesity	In Human	In Rodent
Lep (ob)	Leptin, a fat-derived hormone	Mutation prevents leptin from delivering satiety signal; brian perceives starvation	Yes	Yes
LepR (db)	Leptin receptor	Same as above	Yes	Yes
POMC	Proopiomelanocortin, a precursor of several hormones and neuropeptides	Mutation prevents synthesis of melanocyte-stimulating hormone (MSH), a satiety signal	Yes	Yes
MC4R	Type 4 receptor for MSH	Mutation prevents reception of satiety signal from MSH	Yes	Yes
AgRP	Agouti-related peptide, a neuropeptide expressed in the hypothalamus	Overexpression inhibits signal through MC4R	No	Yes
PC-1	Prohormone convertase 1, a processing enzyme	Mutation prevents synthesis of neuropeptide, probably MSH	Yes	No
Fat	Carboxypeptidase E, a processing enzyme	Same as above	No	Yes
Tub	Tub, a hypothalamic protein of unknown function	Hypothalamic dysfunction	No	Yes
TrkB	TrkB, a neurotrophin receptor	Hyperphagia due to uncharacterized hypothalamic defect	Yes	Yes

Table 2. Some Obesity Genes in Humans and Mice

Table 3. A Comparison of Syndrome of Obesity-Hypogonadism and Mental Retardation

Feature	Syndrome				
	Prader-Willi	Laurence-Moon-Biedl	Ahlstrom's	Cohen's	Carpenter's

Inheritance	Sporadic; two-thirds have defects	Autosomal recessive	Autosomal recessive	Probably autosomal recessive	Autosomal recessive
Stature	Short	Normal; infrequently short	Normal; infrequently short	Short or tall	Normal
Obesity	Generalized Moderate to severe Onset 1-3 years	Generalized Early onset, 1-2 years	Truncal Early onset, 1-2 years	Truncal Mid-chidhood, age 5	Truncal, gluteal
Craniofacies	Narrow bifrontal diameter Almond-shaped eyes Strabismus V-shaped mouth High-arched palate	Not distinctive	Not distinctive	High nasal bridge Arched palate Open mouth Short philtrum	Acrocephaly Flat nasal bridge High-arched palate
Limbs	Small hands and feet Hypotonia	Polydactyly	No abnormalities	Hypotonia Narrow hands and feet	Polydactyly Syndactyly Genu valgum
Reproductive status	1º Hypogonadism	1º Hypogonadism	Hypogonadism in males but not in females	Normal gonadal function or hypogonadotrophic hypogonadism	2º Hypogonadism
Other features	Enamel hypoplasia Hyperphagia Temper tantrums Nasal speech			Dysplastic ears Delayed puberty	
Mental retardation	Mild to moderate		Normal intelligence	Mild	Slight

Thesis Title : A STUDY ON SERUM C-REACTIVE PROTEIN LEVEL IN OBESITY

S.No	OP No	Name	Age (Yrs)	Sex	Weight (Kg)	Height (cm)	Ht (m)	BMI	WC	SERUM CRP LEVEL (mg/l)	Triglycerides	HDL	Occupation	Rural/Urban	Family History
1	50320	Ganesh	30	M	81	176	1.76	26	89	1.8	140	36	Manua	R	
2	50544	Kumar	41	M	71	159	1.59	28	98	2.2	123	32	Manua	R	
3	51567	Veeran	40	M	78	164	1.64	29	88	2.6	168	30	sedent	R	Present
4	51346	Vimalan	36	M	72	160	1.60	28	102	3.8	175	24	sedent	R	Present
5	52888	Murugan	30	M	77	166	1.66	28	88	2.1	134	37	Manua	R	
6	52899	Nandhu	32	M	73	167	1.67	26	89	1.9	141	40	Manua	R	
7	53012	Kalimuthu	29	M	82	171	1.71	28	106	3.9	167	24	sedent	R	Present
8	53120	Kabilan	40	M	78	164	1.64	29	104	3.7	156	21	sedent	R	Present
9	53155	Rajan	30	M	83	164	1.64	31	99	2.9	176	26	sedent	R	Present
10	53179	Maruthamuthu	36	M	92	160	1.60	36	104	3.8	159	26	sedent	R	Present
11	53226	Maran	42	M	86	169	1.69	30	98	4.1	186	25	sedent	U	Present
12	53477	Cheran	38	M	92	175	1.75	30	95	3.7	183	26	sedent	U	Present
13	53568	Raman	22	M	85	158	1.58	34	102	3.8	169	25	sedent	U	Present
14	53678	Ezhil Muthalvan	38	M	90	165	1.65	33	78	3.97	189	22	sedent	U	Present
15	54234	Balu	32	M	92	172	1.72	31	98	2.6	125	38	Manua	U	
16	54321	Vasanth	30	M	84	162	1.62	32	93	3.5	143	21	sedent	U	
17	54456	Kannan	42	M	87	158	1.58	35	104	3.7	132	24	sedent	U	
18	54789	Chinnasamy	40	M	90	160	1.60	35	102	3.1	126	35	Manua	U	
19	54880	Raja	42	M	82	163	1.63	31	90	3.2	133	42	Manua	U	Present
20	55000	Rajappan	40	M	81	159	1.59	32	89	2.8	122	37	Manua	U	
21	55057	Ghunasekaran	40	M	96	163	1.63	36	103	4.2	179	23	sedent	U	Present
22	55109	Kesavan	40	M	96	159	1.59	38	102	5.1	182	22	sedent	U	Present
23	55167	Rasu	25	M	97	174	1.74	32	106	4.3	185	21	sedent	U	Present
24	55198	Dharman	24	M	96	173	1.73	32	88	2.8	137	35	Manua	U	
25	55220	Maran	32	M	95	165	1.65	35	104	4.1	176	25	sedent	U	Present
26	55255	Gurusamy	36	M	90	165	1.65	33	102	4.8	171	24	sedent	U	Present
27	55289	Raja	30	M	73	167	1.67	26	103	<1	165	24	sedent	U	Present
28	55358	Narayanasamy	40	M	83	175	1.75	27	86	<1	135	32	Manua	R	Present
29	55398	Rajamani	20	M	75	164	1.64	28	89	<1	167	26	Manua	R	Present
30	55410	Karuppusamy	25	M	69	163	1.63	26	81	<1	132	35	Manua	R	

31	55433	Mathavan	25	M	76	168	1.68	27	84	<1	138	38	Manua	R	
32	55477	Selvaraj	30	M	87	165	1.65	32	89	<1	165	25	sedent	R	
33	55490	Rajarajan	38	M	96	168	1.68	34	81	<1	131	39	Manua	R	
34	55523	Muthusamy	42	M	88	159	1.59	35	101	<1	169	21	sedent	U	Present
35	55589	Sivakumar	38	M	98	163	1.63	37	88	<1	130	40	Manua	R	
36	55610	Senthil	38	M	96	163	1.63	36	89	<1	136	39	Manua	R	
37	55689	Ravi	29	M	94	164	1.64	35	92	<1	165	23	sedent	U	Present
38	55723	Chandran	38	M	73	164	1.64	27	88	<1	134	37	Manua	R	
39	55756	Selvarasu	29	M	88	177	1.77	28	85	<1	133	36	Manua	R	
40	55822	Kaliswari	26	F	83	156	1.56	34	86	2.3	186	41	Manua	R	Present
41	55845	Malliga	30	F	79	157	1.57	32	84	2.5	177	43	Manua	R	Present
42	55878	Dhaiva	28	F	97	156	1.56	40	89	2.6	140	40	Manua	R	
43	55911	Chandra	28	F	85	158	1.58	34	101	2.7	134	32	sedent	R	
44	55912	Kamalammal	33	F	87	162	1.62	33	82	2.1	138	40	Manua	R	
45	56001	Kavitha	29	F	85	163	1.63	32	102	3.7	162	32	sedent	R	Present
46	56053	Murugathal	36	F	87	153	1.53	37	102	3.8	165	36	sedent	R	Present
47	56121	Narmatha	32	F	73	153	1.53	31	89	2.1	136	44	Manua	R	
48	56145	Rathiga	40	F	75	153	1.53	32	101	5.2	173	36	sedent	R	Present
49	56189	Noornisha	36	F	61	156	1.56	25	104	3.9	170	41	sedent	R	Present
50	56221	Lakshmiammal	32	F	82	158	1.58	33	85	2.9	166	40	Manua	R	Present
51	56256	Padma	33	F	81	154	1.54	34	89	2.8	131	43	Manua	R	
52	56290	Menaga	30	F	66	159	1.59	26	88	2.6	133	41	Manua	R	Present
53	56311	Rajeshwari	40	F	67	158	1.58	27	89	2.9	136	45	Manua	R	
54	56329	Rajammal	25	F	61	153	1.53	26	89	2.6	138	44	Manua	R	
55	56377	Marriammal	25	F	76	154	1.54	32	100	4.2	182	35	sedent	U	Present
56	56390	Sasikala	28	F	74	160	1.6	29	99	4.8	164	40	Seden	U	Present
57	56456	Saraswathy	30	F	67	158	1.58	27	102	5.1	140	37	sedent	U	Present
58	56478	Indhira	35	F	89	157	1.57	36	98	2.2	175	52	Manua	U	Present
59	56199	Susi	40	F	81	159	1.59	32	102	4.9	168	33	sedent	U	Present
60	56505	Maruthal	32	F	83	159	1.59	33	103	2.8	169	35	sedent	U	
61	56589	Essakki	26	F	91	159	1.59	36	92	2.1	178	44	Manua	U	
62	56612	Dhanya	42	F	64	157	1.57	26	105	4.1	162	36	sedent	U	Present
63	56619	Lakshmi	43	F	92	156	1.56	38	98	2.9	171	34	Seden	U	
64	56687	Meena	38	F	78	159	1.59	31	99	2.7	132	35	Seden	U	
65	56723	Sridevi	33	F	90	163	1.63	34	104	4.8	173	36	sedent	U	Present
66	56756	Ananthammal	34	F	87	158	1.58	35	112	5.1	167	38	sedent	U	Present
67	56790	Suganya	40	F	71	156	1.56	29	108	4.9	162	36	sedent	U	Present

68	56811	Latha	38	F	76	157	1.57	31	98	2.1	178	54	Manua	U	Present
69	56834	Maral	38	F	69	149	1.49	31	98	2.5	131	36	Seden	U	
70	56899	Rafia	40	F	76	154	1.54	32	98	3.4	169	35	sedent	U	Present
71	56900	Ponnammal	34	F	91	161	1.61	35	93	2.2	130	34	Seden	U	
72	57023	Shifa	32	F	86	157	1.57	35	94	2.7	136	41	sedent	U	
73	57122	Kannaki	32	F	83	159	1.59	33	103	2.9	134	36	sedent	U	
74	57156	Monisha	28	F	72	160	1.6	28	92	2.4	138	37	sedent	U	
75	57289	Sherine	32	F	71	156	1.56	29	94	2.6	135	32	sedent	U	
76	57390	Ponni	28	F	78	159	1.59	31	96	<1	176	34	edenta	U	PRESENT
77	57412	Mariyammal	29	F	90	160	1.6	35	94	<1	179	35	edenta	U	PRESENT
78	57434	Kaliammal	42	F	79	157	1.57	32	82	<1	172	31	edenta	U	PRESENT
79	57456	Kuppammal	34	F	87	149	1.49	39	85	<1	125	39	ual Lal	U	
80	57678	Indhirani	37	F	78	159	1.59	31	84	<1	121	37	ual Lal	U	
81	57699	Mangalm	39	F	77	153	1.53	33	81	<1	178	32	edenta	U	PRESENT
82	57789	Bhuvaneshwari	38	F	78	151	1.51	34	83	<1	125	38	ual Lal	U	
83	57890	Mahalakshmi	30	F	73	149	1.49	33	88	<1	173	33	edenta	U	PRESENT
84	57920	Fathima	28	F	85	158	1.58	34	90	<1	166	31	edenta	U	
85	58109	Rafiabeham	24	F	79	157	1.57	32	86	<1	169	33	edenta	U	
86	58120	Nithya	36	F	66	156	1.56	27	92	<1	164	31	edenta	R	
87	58234	Jayamarie	42	F	70	158	1.58	28	94	<1	168	30	edenta	R	
88	58345	Kamalambal	39	F	71	159	1.59	28	92	<1	171	33	edenta	R	PRESENT
89	58467	Mariya	22	F	64	157	1.57	26	92	<1	165	31	edenta	R	
90	58567	Roshini	32	F	68	159	1.59	27	84	<1	123	41	ual Lal	R	
91	58690	Valli	28	F	66	156	1.56	27	88	<1	166	32	edenta	R	
92	58745	Mailathal	30	F	49	143	1.43	24	86	<1	173	35	edenta	R	PRESENT
93	58934	Meenachi	32	F	63	156	1.56	26	84	<1	126	37	ual Lal	R	
94	59034	maheswari	32	F	76	162	1.62	29	83	<1	128	36	ual Lal	R	
95	59167	Ranjini	36	F	54	142	1.42	27	83	<1	123	40	ual Lal	R	
96	59256	Aandal	37	F	69	157	1.57	28	83	<1	126	35	ual Lal	R	
97	59321	Latha	40	F	59	153	1.53	25	89	<1	165	35	edenta	R	
98	59432	Baby	40	F	79	162	1.62	30	92	<1	172	34	edenta	R	PRESENT
99	59576	Devi	21	F	63	159	1.59	25	88	<1	177	32	edenta	R	PRESENT
100	60123	Nalini	24	F	61	156	1.56	25	102	<1	162	34	edenta	R	

BMI - Body Mass Index (kg/m^2)

WC- waist circumference or Circumference of Waist

Coimbatore Medical College

COIMBATORE, TAMILNADU, INDIA - 641 014

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ETHICS COMMITTEE

CERTIFICATE

Name of the Candidate : DHANALAKSHMI. T.

Course : M.D. GENERAL MEDICINE

Period of Study : 2012 - 2015

College : COIMBATORE MEDICAL COLLEGE

Dissertation Topic : A STUDY ON SERUM C-REACTIVE PROTEIN LEVEL IN OBESITY

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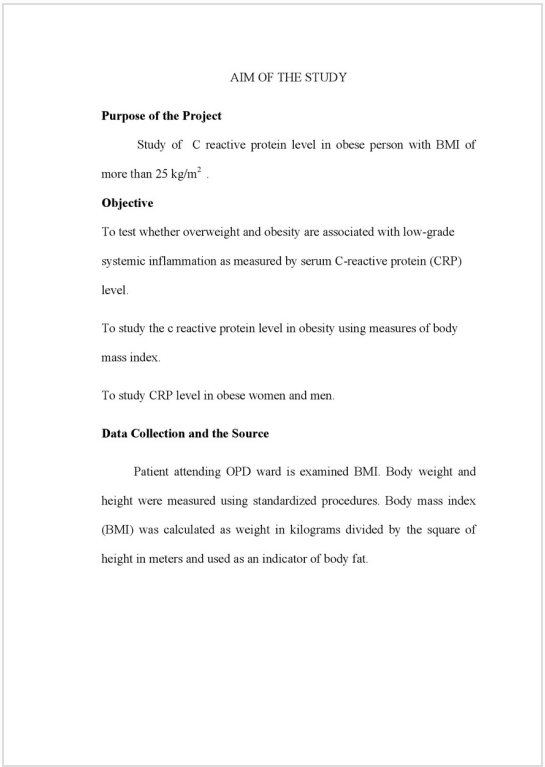

AIM OF THE STUDY

Purpose of the Project

Study of C reactive protein level in obese person with BMI of more than 25 kg/m^2 .

Objective

To test whether overweight and obesity are associated with low-grade systemic inflammation as measured by serum C-reactive protein (CRP) level.

To study the c reactive protein level in obesity using measures of body mass index.

To study CRP level in obese women and men.

Data Collection and the Source

Patient attending OPD ward is examined BMI. Body weight and height were measured using standardized procedures. Body mass index (BMI) was calculated as weight in kilograms divided by the square of height in meters and used as an indicator of body fat.