CHAPTER-1

INTRODUCTION
INTRODUCTION

Obesity is the most common nutritional disorder of present era. Its significance requires constant emphasis because it is associated with increased mortality, predisposes to the development of Chronic diseases and diminishes the efficiency and happiness of those affected.

Obesity is a complex multifactorial disorder characterized by an excess of adipose tissue. Although obesity can be looked at simply as an excess of calories ingested compared with Calories expended. It involves complex interaction between genetic, metabolism and appetite regulation on the one hand and food availability behavior, physical activity and cultural factors on the other. Thus although the increasing prevalence of obesity is undoubtedly due to environmental factors such as eating more and exercising less, the role of other factors such as stress has been raised, chronic stress for example, although environment may effect metabolism by causing an increase in cortisol secretion. [Louis J. Aronne et al]¹

[Mathall & Jenesn et al]² Obesity is not simply the result of gluttony and a lack of willpower; similar to essential hypertension. Obesity results from the genetic predisposition combined with environmental factors both are necessary for obesity to occur but the genetic predisposition seems to be quite common based on the high prevalence of the disorders to become obese, the genese allowing obesity to occur must be present or an increase in metabolic person must have access to *enough* calories. The person must consume more calories than the body expends and the person must not be expending the excess calories with exercise.

According to [Armstrong Dubin (1951) and Strang (1959)]³. Increase of 20% weight above the standard height and weight chart constitute obesity.
[Macbride (1964)]⁴ says 15% increase of body weight above the standard height – weight chart constitute obesity.

According to (Cecil-text book of medicine)⁵ obesity is a risk factor of many illnesses Traditionally, obesity was believed to be associated with affluent life styles. However, obesity is a fast growing problem in developing countries. Several studies in Indian shown that changes in dietary pattern, physical activity levels, life styles associated with affluence and migration to urban areas are related to increasing frequencies of obesity.

According to [Shephard–1998]⁶ obesity is a chronic and increasingly common disease characterized by excess body fat. It develops gradually and often persists throughout life.

According to (Bengisson C, Larson B)⁷ Obesity is a complex multifactorial condition characterized by abnormal or excess body fat accumulation in adipose tissue to the extent that health may be impaired.

[According to Keys]⁸ A, Obesity is an era old problem. its historic roots can be traced up to paleolithic era more than 25000 years ago. Lately it has assumed a mammoth form as a major health problem.

[According to WHO]⁹ it is an global epidemic, obesity is a widespread disease of increasing prevalence and therefore is rapidly becoming a major health issue in modern society. An estimated 250 million adults worldwide are obese and many more are over weight.

According to [Bonetto L]¹⁰ Obesity may be defined as a disease, process in which excess body fat has accumulated to an extent that health may be adversely affected.
National health and nutrition examination surveys [NHANES]\textsuperscript{11} have been used BMI (Body Mass Index) to assess the prevalence of over weight and obesity and provided the statistics that are often cited.

Recently in 1998 the [US National Heart, Lung and Blood Institute]\textsuperscript{12} established guidelines of define "over weight" and obesity. The parameter used is Body Mass Index (also called Quetelet index) and is calculated using the formulae

\[
\text{Weight (Kg)} \quad \text{BMI} = \frac{\text{----------------------}}{[\text{Height (M)}]^2}
\]

[According to NHANES]\textsuperscript{13} survey over weight begins at a BMI greater than 27.3 in women form obesity and BMI greater than 32.3 form Morbidly Obese.

[NHANES II\textsuperscript{nd} survey says]\textsuperscript{14} although the prevalence of over weight above age of 20 has increased. The increased amount of fat and sugar in the diet and decreased amount of physical activity have been blamed for the increasing incidences of obesity.

[WHO expert committee]\textsuperscript{15} recommended the obesity is emerging as a world wide epidemic. Genetics of obesity led to the isolation of six genes that are associated with obesity and studies of energy metabolism clearly show that weight loss and gain are met with a change in energy metabolism that would tend to resist changes in body weight.

[According to Bonetto (1999)]\textsuperscript{17} waist hip ratio provides information about the distribution of body fat.

\[
\text{Waist Ratio} = \frac{\text{Desired Ratio}}{\text{Hips}}
\]

Women \( \leq 0.8 \)

Men \( \leq 1.0 \)

To find ratio – waist : Measure at narrowest point with stomach relaxed.

Hips : Measure at fullest point.
[WHO expert committee] has recommended the following classification of over weight and obesity which is applicable to all adult age group.

<table>
<thead>
<tr>
<th>BMI (Kg/m²)</th>
<th>WHO Classification</th>
<th>Clinical Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 18.5</td>
<td>Under weight</td>
<td>Thin</td>
</tr>
<tr>
<td>18.5 to 24.9</td>
<td>Normal Weight</td>
<td>Healthy</td>
</tr>
<tr>
<td>25 to 29.9</td>
<td>Grade – I</td>
<td>Over weight</td>
</tr>
<tr>
<td>30 to 39.9</td>
<td>Grade – II</td>
<td>Obesity</td>
</tr>
<tr>
<td>&gt; 40.00</td>
<td>Grade – III</td>
<td>Morbid Obesity</td>
</tr>
</tbody>
</table>

Obesity – An imbalance in energy intake and energy expenditure.

According to Deybens (1921) Skinfold thickness from three separate sites (Biceps, Triceps and Subscapular area) also the method of accessing obesity.

(Medicine update) the regional distribution of fat plays an important role in determining the risk of a given level of obesity. There are different types of fat distribution in adult obesity.

The deposition of fat mostly in the abdomen (above the waist) is known as apple shaped or android or abdominal or central obesity this type of distribution is associated with a greater risk of most of the complications of obesity. Whereas collection of fat on hips and buttocks (below the waist) is known as pear shaped or Gynoid type obesity. [Dr. Shashank et al ]
Women with abdominal predominance fat pattern seem to smoke and use alcoholic beverages more have socioeconomic disadvantages, have psychosomatic diseases and have more psychiatric and psychological problems.

Bray (1976)\textsuperscript{20} reported that obesity can influence many aspects of life, serious diseases are most prevalent with obesity.

Excess fat accumulates because there is imbalance between energy intake and expenditure. This can arise in different ways and obesity is a clinical sign with several possible causes. There are no satisfactory etiology of obesity but a number of factors are known to be associated with its development.

Age :- obesity is most prevalent in middle age but can be occur at any stage of life. Adiposity increases with age with a redistribution of body fat towards the abdominal compartment.

Weight gain is also excessive in women after menopause.

Socioeconomic :- In affluent countries obesity in the lower socioeconomic groups and in developing countries it can occur only in the prosperous elite.

Heredity :- A familial tendency exists in many cases: Various other factors like

- Sex
- Sleeping Habit
- Psychological Factor
- Physical Activity
- Tension & Stress also affect body weight
- Smoking Habit
- Endocrine Factor
- Leaving Standard
- Climate
Obesity can influence many aspects of life Bray (1976) reported that serious diseases are most prevalent in overweight individuals and are the leading cause of successive mortality. It is important to remember that degree of overweight is not necessarily proportional to the severity of complication.

(Dr. Hakim et al) obesity affects practically all systems of body.

The obesity associated problems are:

Hyperinsulinemia and Diabetes Mellitus: Obesity is associated with hyperinsulinemia, insulin resistance in obesity is thought to be due increased free fatty acids. Which impair insulin action on skeletal muscle it inhibits the action of insulin. This leads to hyperinsulinemia and the development of type-II Diabetes Mellitus.

Hypertension: There is a significant positive relationship between free fatty acid and blood pressure in obesity. This occurs due to free fatty acid increasing the vascular sensitivity to adrenergic stimuli. Furthermore free fatty acid inhibit nitric oxide production causing vasoconstriction and blunt reflex vaso-relaxation.

Dyslipidemia: Obesity is associated with an increase in triglycerides, decreased HDL, and high LDL. This hyperlipidemia contribute so many diseases.

Cardiovascular disease: Obesity causes increased workload on the heart it is also associated with sudden death probably due to cardiac arrhythmias precipitated by atherosclerosis. This possibly is a reflection of an abnormal lipid profile which are commonly associated with obesity.

Gall Bladder disease: Gall bladder disease increases with obesity and age possibly related to increased excretion of biliary cholesterol. There is approximately a six fold increase in the incidence of gall stones in patients whose weight is 50% above ideal body weight.
Cholecystitis: Extreme dieting that are sometimes used in the treatment of obesity can precipitate cholecystitis.

Pulmonary Disease: Pulmonary abnormalities may occur in obese patients. These include decreased total lung capacity, functional residual capacity, reduced chest wall compliance and increased work load on breathing. This can lead to reduced effort tolerance and restriction of physical activity.

Bones and Joints: Increased trauma to weight bearing joints often leads to osteoarthritis. This commonly involves the knee and hip joints.

Skin: Obese people very often have thin friable skin increasing the incidence of fungal and yeast infection. Obese people often develop varicose, varicose veins and venous stasis.

Cancer: Obesity is linked with a higher incidences of cancer. In obese females there is a higher mortality from cancer of the breast, cervix, endometrium, Ovary and gall bladder.

Reproductive Disorders: Women with obesity often have menstrual abnormalities. This is due to increased androgen production and increased peripheral conversion of androgen to oestrogen. Obese women with oligomenorrhoea often have polycystic ovarian syndrome, which is associated with an ovulation. An increased incidence of uterine cancer is seen in post-menopausal women with lower-body obesity. The increased conversion of androstenedione to oestrogen that occurs to a greater degree in obesity is thought to be responsible.

Specific Syndromes associated with obesity: Cushings syndrome, hypothyroidism, insulinomas and disorders involving the hypothalamus are often associated with obesity.
There are various studies which give statistical significant result and show relation of obesity with associated diseases.

(Framingham heart studies)\(^{22}\) shows that among all the patients reached to heart clinics, 68.4% of them are obese. These figure show direct correlation between android obesity and increased risk of CHD, stroke, hypertension, cardiomyopathy. Congestive heart failure, diabetes and mortality.

(M. Shivatav et al)\(^{23}\) reported that obesity is an independent risk factor for chronic heart disease.

(Fragmigham heart studies)\(^{24}\) elucidate the risk of death within the age of 26 years increased by 1% for each extrapound (0.45kg) increased in the weight between age of 30-42 years and by 2% for corresponding weight. Increase between the age of 50 to 60 years.

(Peris AN and Mueller RA et al)\(^{25}\) Among the patient reaches to diabetic clinics, 69% of them are obese this figure show relation of obesity and diabetes. Obesity is associated with hyperinsulinemia. Over expression of TNFα gene (found in obese subject) inhibit the action of insulin, this leads to hyperinsulinemia. Increased level of free fatty acid (in obese) also leads to impaired insulin secretion by the process of β cell lipotoxicity.

(Huang et al. 1999)\(^{26}\) reported increased strain on weight bearing joints due to obesity leads to osteoarthritis or joint pain.

(Sheehan MT et al)\(^{27}\) reported that obesity is linked with higher incidences of breast cancer.

(Dr. Bajaj et al)\(^{28}\) reported obesity some times causes sterility.
All the obesity associated problems ultimately due to alteration of lipid levels or impairment in lipid metabolism.

(Shephared, J. Cobbe)\textsuperscript{29} reported that all the obesity associated problems are due to hyperlipidemia found in obese subject. Hyperlipidemia concern about Four Blood Lipids, Lipoproteins and Apoproteins, The normal level of these lipids are required for the normal health.

The Blood Lipids are

\begin{itemize}
  \item Cholesterol
  \item Triglyceride
  \item Phospholipids
  \item Esterified Cholesterol
\end{itemize}

Lipoprotein : is a complex macromolecule of lipid and protein which the non-polar lipid core is surrounded by a polar mono-layer of phospholipids, heads of free cholesterol and apolipoprotein. The major serum lipoproteins are (Harper, 1999)\textsuperscript{30}.

\begin{itemize}
  \item Chylomicrons
  \item VLDL
  \item IDL
  \item LDL
  \item HDL
  \item Lip(a)
\end{itemize}

Apolipoproteins : Apoliporoteins are the chief structural component of the lipoprotein. The major classes of apolipoprotein, are A, B, C and E. There are various type of Apoprotein AI, AII, AIV, B-48, B-100, E, C-I ,C-II, C-III.
Lipid metabolisms involves certain enzymes they are:

- Lipoprotein lipase (LPL)
- Hepatic lipase (H-PL)
- Cholesteryl ester
- Cholesterol ester Transfer protein
- LCAT
- PLIP (Phospholipid Transferase protein)
- apolipoproteins
- apo A-I
- apo B
- apo B editase
- Lecithin Cholesteryl acyl transferase

In obese subject there is increase production of VLDL and triglycerides leads to hypertriglyceridemia and defective clearance of LDL– cholesterol will leads to hypercholesterolemia. Both of these effect leads to hyperlipidemia.

Hyperlipidemia in obesity is considered to be elevated level of free acids. The increased level of free fatty acid stimulates VLDL secretion by increasing triglyceride synthesis which in turn leads to a greater secretion of VLDL APO β-100. Fatty acids in obese subject is thought to be due to either an increased release from adipose tissue or decreased uptake by other tissue. (MCNA)\textsuperscript{31}.

(Sheehan et al)\textsuperscript{32} reported elevated free fatty acids have multitude of effects one of the effect is elevated level of serum lipoproteins.

(Stone 1984)\textsuperscript{33} reported that obese subjects often have increased triglycerids and decreased HDL–cholesterol.

Groundy and Burnett (1990)\textsuperscript{34} reported that many obese people, but certainly not all have elevated triglyceride. In most over weight individual, caloric restriction reduces serum triglyceride level early with delayed effect of rise of
HDL-cholesterol. When obese people loose weight to nearly an ideal level HDL-cholesterol shows no change.

According to [Comfort et al]^{166}, Antioxidant level reduces by formation of free radical with aging, this leads to increase in weight through the increase in lipid level.

(Gordon et al)\textsuperscript{35} Analysis Studies demonstrated that for every 1 mg/dl rise in HDL-cholesterol, the risk of CHD decreases by 2% in men and 3% in women and this effect was independent of LDL-cholesterol levels.

(Wolf and Grundy 1983)\textsuperscript{36} indicating that why obesity contribute hypertriglyceridemia in some people but not in all ? The question is still unanswered.

(Zimmerman et al 1984 )\textsuperscript{37} reported that obese subjects have increased synthetic rate for cholesterol, triglyceride, bile acids. VLDL, LDL. Its because of increased level of free fatty acid in obese subject most probably due to increased release adipose tissue or decreased uptake by other tissue.

(Caggiula et al 1981)\textsuperscript{38} reported that serum total cholesterol and in some instances LDL-cholesterol has been shown to change in direct proportion to weight gain or loss. A high input of LDL-cholesterol can originate from increased secretion of VLDL or a high fractional conversion of VLDL to LDL.

(Grundy et al 1987)\textsuperscript{39} over nutrition can effect metabolism of LDL in several ways that might contribute to raise LDL level.

(Zimmerman et al 1984)\textsuperscript{40} reported that obese patients lose weight to nearly an ideal level, LDL-cholesterol shows no change.
(According to Garrison et al)\textsuperscript{41} it is clear that obese people having 8 to 10 mg/dl HDL–cholesterol below normal. The mechanism why obesity reduces HDL-cholesterol is now clear. In obese elevated triglyceride level stimulated the transfer of cholesterol-esters from HDL particles to triglyceride rich lipoprotein and this action results lowering of HDL-cholesterol concentration in obese.

(Anjali Manocha 2001)\textsuperscript{42} demonstrated the inverse relationship of triglyceride and HDL–cholesterol is strongly only up to distinct elevated range beyond which any further rise in triglyceride is not associated with a further fall in HDL.

(Denke et al 1993)\textsuperscript{43} reported that total cholesterol and LDL–cholesterol levels correlated positively with body weight several studies have observed that when people gained weight their serum cholesterol level rise conversely when they lost weight serum cholesterol levels fall. Obese individual either men or women have 5 to 10 mg/dl constantly lower HDL-cholesterol level as compared with lean counterparts.

(The Franningham studies)\textsuperscript{44} estimated for every 10(lb) weight gain total cholesterol level increased 7 mg/dl in men and 5 gm/dl in women.

In population studies there were weak but statistically significant direct correlation between body fatness and either total cholesterol or triglyceride.

In both men and women under age 40 body fatness is correlated inversely with HDL–cholesterol level and directly with LDL-cholesterol level. Although these correlation were weak (Garrison et al 1980)\textsuperscript{45}.

But according to (National Cholesterol education programme 1919)\textsuperscript{46} the increase of triglycerides and cholesterol and decrease of HDL–cholesterol is directly related to obesity.
Albrinck et al 1962 Triglyceride level were lowest in the leanest men and highest in the men who gained excess weight during adult life. Not all fat men and women had high triglycerides level in-fact the fattest men had normal or only slightly increased lipid levels. The highest triglyceride level were found in women who were only moderately obese and had acquired their adiposity during adult life. This suggested that acquired obesity that attained after Maturity even though moderate in degree might be different from natural obesity and might have the stronger association with hyper triglyceridemia in middle age obese subject have high synthetic rate for cholesterol and bile acid. They have increased turn over of apo-LDL but this is not necessarily associated with high LDL cholesterol level (Kesatini and Grundy 1983).

But the high synthetic rate for cholesterol and bile acid is only because of increased availability of fatty acid due to lypolysis of adipose tissue. (Dr. Hakim)

Albrink and Man 1959 reported that 5% of normal young men in their twenties had serum triglyceride above normal limits and between age of 40-60 years 35% exceeded the limit. The differences in the cholesterol level though less marked than those for triglyceride were in same direction showing an increase with age. Total serum lipids as well as individual lipids were as a rule some what higher in obese than in lean-persons. Although triglyceride appears more strongly related to obesity than cholesterol.

Golfman & Johnes 1952 Studies concluded that serum level of lipoprotein was significantly associated with weight where as the serum cholesterol estimation had no significant relation ship.

Diet and exercise in men have produced lower triglyceride and higher HDL-cholesterol level but in women after menopause have actually been little change in HDL- cholesterol level (Bernard 1991).
In women of pre menopause have sufficient concentration of oestrogen this hormone is known to be antiatherogenic factor because oestrogen hormone protect HDL-cholesterol to fall. (Journal of MCNA—women health)\textsuperscript{57} So that HDL-cholesterol level fall in obese women is lower in comparison to obese men.

(Kannel—1998)\textsuperscript{52} In cross sectional studies LDL-C levels rise in women after age of 55 years.

(Burnard — 1990)\textsuperscript{53} HDL cholesterol level maintained at normal due to oestrogen hormone in premenopouse women.

(Mathews et al 1989)\textsuperscript{54} HDL–cholesterol level falls in post menopausal women.

Lipid research clinics population studies and [La Rasa et al 1986]\textsuperscript{55} demonstrated that the elevation of LDL–cholesterol was lower in pre-menopausal women than in aged matched men.

NCEP guidelines place greater emphasis on low HDL-cholesterol level than the previous version and have revised the level below which HDL-cholesterol is considered to be a CHD risk factor. The guidelines recommended the reduction of weight and rising of HDL–cholesterol.

Data from the Framingham population on study indicated that at any given level of total cholesterol in obese the relative risk of CHD increases with decreasing level of HDL-Cholesterol. The incidences of myocardial Infarction was increased almost two fold / in men with 1000 HDL-Cholesterolal level ≤ 52 mg/ dl as compared with men who had higher HDL-cholesterol level. This relation ship was even more apparent in obese women with (HDL-C ≤ 52 mg/dl were associated with a fourfold to six fold increase risk of MI.
The prospective cardiovascular muster (PROCAM) study demonstrated a link between low HDL-C and increased risk of atherosclerosis.

In obese subject increase in level of Triglyceride and cholesterol and decrease in level of HDL-cholesterol results risk of heart problems.

(Gorden et al) analysis studies demonstrated that for every 1 mg / dl rise in HDL-cholesterol the risk of CHD decreased by 2% in men and 3% in women and this effect was independent of LDL-cholesterol level.

Gordon et al studies demonstrated that the risk of CHD is higher in individual with low level of HDL-cholesterol and that this relationship is independent of the total cholesterol level. These data support the involvement of HDL within in the current treatment guidelines as a risk factor for the development of CHD.

There are so many clinical evidences for the benefits of raising HDL-cholesterol level.

Studies conducted to date have been unable to demonstrate clearly a causal link between low level of HDL-cholesterol and atherosclerosis.

Animal studies suggests that HDL-cholesterol is anti-atherogenic.

Veterans affairs cooperative studies program. High density intervention trial (VAHIT) assessed the effect of raising HDL-cholesterol levels on CHD risk in patient with low levels of HDL-cholesterol and increased triglyceride level.

(VA-HIT studies) conducted in women with CHD and low levels of HDL-cholesterol with normal LDL-cholesterol and triglyceride levels below those recommended for the initiation of lipid modifying therapy showed that raising HDL-cholesterol levels significantly reduced the rate of CHD events.
Diet and exercise had significant beneficial effects upon the level of HDL-cholesterol and Total cholesterol but not on LDL-cholesterol and was associated with a reduction of 22% in non fatal myocardial Infarction or death due to CHD. For every 5 mg/dl increase in HDL-cholesterol, risk of CHD death or myocardial infarction decreased by 11%. Although baseline mean triglyceride levels were with in the reference range (161 mg/dl), the observed decreased of 31% in triglyceride may have contributed to the reduction in CHD events by improving the distribution of LDL-cholesterol sub-fractions. (Michail And Jenson) 2

The Air Force/Texas coronary Atherosclerosis prevention [AFCAPS/Texas]59 study in asymptomatic subjects with below average. HDL-cholesterol levels and mild to moderate elevation in LD2-cholesterol and total cholesterol result Hypertension.

(Medicine Science Jan–02 Vol.5)60 studies on obese women with hypercholesterolemia aged 34 to 60 years concluded that a modified vegetarian diet rich in omega 3-fatty acid efficiently potentiated the cholesterol lowering effect.

According to (JMS vol.5)61 studies show increasingly supporting the view that hypertriglyceridemia is an important independent cardiac risk factor.

Different studies to prove that triglyceride are an independent risk factors in CHD.

Framingham study duration 25 years follow up number of patients/age [5209 men and women 30-62 years] his observation is high triglyceride levels are considered high risk for obesity and CHD with exception of those with a very low total HDL-cholesterol ratio. Elevated triglyceride are a highly significant risk factor for CHD in women and important in men with low HDL-C (≤ 40 mg/dl).
(Paus Prospective)\textsuperscript{74} 11 years study on 7038 men (43-54 years). Univariate analysis of plasma, triglyceride, cholesterol, insulin levels in both fasting and post glucose load were significantly higher in subjects who died from CHD as compared to those who did not multivariate regression analysis. Plasma triglyceride level was the only factor positively and significantly associated with coronary death.

Meta analysis by (Hokanson and Austin)\textsuperscript{63} 8.4 years study in men and 11.4 years study in women. Number of male patients is 46413 and female patients is 10,804. Triglyceride is an independent risk factor for cardiac disease. Elevated triglyceride were associated with a 30\% increase in women and a 75\% increase in men in the risk for CHD.

Copenhagen males 8 years studies on 2906 men fasting triglycerides are a strong risk factors of CHD independent of other major risk factors including HDL cholesterol.

These studies reveal that TG; on their own, have an important role to play in predicting atherogenesis.

(PROCAM study)\textsuperscript{64} done between 1979 & 1985 which involved 19,698 person in the age groups 16-65 years. In a multi variate analysis after 8 years, this study stated that elevate triglycerides were a significant and independent risk factor for CHD even after adjustment for other factors like LDL & HDL cholesterol; age; systolic BP; cigarette smoking ; diabetes mellitus, and family history of diabetes mellitus and angina pectoris, In addition, this study also stated that increase in total and LDL-cholesterol and triglycerides as well as decrease in HDL cholesterol levels were directly proportional to the increase in the risk of CHD.
Prevalence of CHD due to obesity is 80% according (Christian Medical College, Vellore)

According to (Prof. K. Srianth) Low HDL–cholesterol seems to be hallmark of most Asian-urban population.

Studies by K. Srinath et al says, if one look at the BMI cumulative frequency distribution curves of the rural and urban areas of Delhi represented by low socioeconomic urban and high income group respective. Shows prevalence of obesity is high in urban part and high income group.

Survey done by (Dr. Ramchandran and Dr. Parvesh and Associate) In Indian population the findings were : Obesity found to be more prevalent in urban population, CHD risk due to obesity is very high, Prevalence of Type II diabetes was higher in obese and The obesity due to smoking was more prevalent in rural area.

(ICMR survey Study) conducted in Delhi on the prevalence of obesity in 2001 gives following data :-

• The incidences of developing morbid obesity are greater in women with comparison to men.

• Mainly the less physical activity and eating habits develop obesity.

• The incidences of obesity increases in urban areas.

• The incidences of obesity is much more greater in Histectomic women.

By seeing these studies we can understand that obesity is the most dangerous problem of present era. It is very necessary to do work on it. This
Problem is most common in adult women of urban part. The increasing prevalence of obesity and associated diseases among adult women of urban part is alarming.

It is observed that no significant work has been reported on obesity and associated diseases in relation to lipid metabolism in various parts of a nation and whatever the work done on obesity, provided conflicting reports.

No work has been done on obesity and associated diseases in relation to lipid metabolism in adult women of urban part of Madhya Pradesh where physical activity, life style, dietary habit and socioeconomic status are entirely different and have definite impact on obesity.

In obesity there will be increase in free radical production and so antioxidant level is disturbed. So, study of change in anti-oxidant level in obesity will definitely help in treating obesity and associated diseases.

Keeping into consideration all these general observations about obesity and its associated diseases is decided to investigate lipid profile and anti-oxidant level in various grade of obesity and associated diseases in adult women as well as control group and find out their relation with obesity, obesity associated diseases, dietary habit, life style and physical activity.