Chapter II

Review of Literature
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REVIEW OF LITERATURE

The available literature on overweight and obesity has been reviewed in the following pages under the headings given below:

1. THEORIES ON THE CAUSES OF OBESITY

2. ASSESSMENT OF OBESITY AND ITS GRADES
   1. Weight Height Indices
   2. Skin fold thickness as a measure of body fat.

3. PREVALENCE OF OBESITY

4. FACTORS INFLUENCING OBESITY
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5. MANAGEMENT OF OBESITY
   1. Dietary Management
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1. THEORIES ON THE CAUSES OF OBESITY

1. The settling point theory as a cause of obesity

This theory claims that "overeating" during childhood stimulates the body’s production of more fat cells for the storage of excess fats. As a person grows to adulthood, the fat cells continue to store more fats making adipose tissues become more massive. The theory says that compared to people of a healthy weight, obese people possess more fat cells, which increase their capacity to store fats.

The theory of the cause of obesity adds that the body mechanisms of obese people to inhibit fat intake (if there is already enough fats ingested) and to relieve hunger (if the person is already full) is slow. The body mechanisms might involve the brain’s delay or failure to send signals to the ‘hunger center’ of the brain to ‘tell’ the person that he is already full. The consequence of this problem is that the excess fats ingested are deposited to fat cells in adipose tissues.

Furthermore, the theory claims that the weight of obese people has a ‘settling point’ that any deviation from it is prevented by physiological means. The ‘settling point’ is comparable to the 37 degrees Celsius constant body temperature of human. This can be observed when an obese person’s metabolic rate drops as that person loses weight through diet. We know that low metabolic rate inhibits fat burning; this makes the
diet futile. When an obese person then begins to gain weight his
metabolic rate soars up and his appetite is enhanced. The lost in weight is
regained three times faster until the ‘settling point’ is reached Marieb,

2. **Fuel efficiency theory as a cause of obesity**

This theory claims that obese people are ‘more fuel efficient’ and
better ‘fat stores’ Marieb (2006). Perhaps everybody has the notion that
an obese people eat more food than normal persons that is why their body
‘ballooned’. This is not totally correct because some people who are
considered obese actually eat less food than normal weight people.

Molecular observation on the fat cells of obese people reveals that
they (fat cells) extend more alpha receptors from their plasma membranes
than what is observed in normal persons. This alpha receptors are the
sites where fatty acid and glycerol molecules bind, they are also
responsible in transferring these molecules toward the cytoplasm.
Therefore, the more alpha receptors, the more efficient are the entry of
fats towards the cell’s cytoplasm. Furthermore, fat cells of obese people
secrete high amount of lipoprotein lipase enzymes responsible in
unloading fats from the blood towards fat cells. The effect is again more
fat deposition to fat cells.

3. **Genetic predisposition theory as a cause of obesity**

This theory of the cause of obesity is based on genetics: the study
of heredity, genetically obese parents can transfer their obesity genes to
their children. When the obesity genes are expressed, the tendency for the
obese person is that any excess calorie he consumed is deposited as fat
instead of being processed for the formation of muscles (as observed in a
normal person). Scientists have already identified 250 obesity genes in mice and men (Foster, 2008) but their modes of expression are not yet understood. However, they believe that the environment has a strong influence in activating these genes to be expressed. One case study that supports this theory is an adopted child who grows to be like his obese parents even though his 'legal parents' did not fail to guide his eating habits. The child did inherit his 'real' parents' obesity genes.

2. ASSESSMENT OF OBESITY AND ITS GRADES

Deurenberg and Yap, (1999) fat is a normal component of the human body that is stored in adipose tissue. Obesity can be defined as a condition of excessive fat accumulation to the extent that health and well-being are affected. Body fat can be determined in vivo in different ways, using rather accurate laboratory techniques or using simple estimation techniques that can also be applied in field conditions. For population studies, the World Health Organization defines cut-off values for obesity based on the body mass index (BMI): weight/height squared (kg/m^2). Generally, for adults, if the BMI exceeds 25 kg/m^2, a subject is considered to be overweight, and if the BMI exceeds the value of 30 kg/m^2, a subject is considered obese.

1. *Weight Height Indices*

Many indices of obesity (weight/height, weight/height^2, weight/cube of height, cube root of weight/height etc.) have been used in the past.

The Quetlet's Index or Obesity Index or Body Mass Index [weight (kg)/ Height in meter^2] has been found to be the most appropriate one. Body Mass Index or BMI is simple and widely used method for
estimating body fat mass Mei et al. (2002). BMI was developed in the 19th century by the Belgian statistician and anthropometrist Adolphe Quetelet, (Quetelet, 1871). BMI is an accurate reflection of body fat percentage in the majority of the adult population. It however is less accurate in people such as body builders and pregnant women. A formula combining BMI, age and gender can be used to estimate a person’s body fat percentage to an accuracy of 4%.

BMI is calculated by dividing the subject’s mass by the square of his or her height, typically expressed either in metric or US ‘customary’ units.

Metric: BMI = Kilogram/meters$^2$

US/CUSTOMARY and imperial: BMI = Lb 703/in$^2$

Where Lb is the subjects weight in pounds and is the subjects height in inches.

The most commonly used definitions, established by the World Health Organization (WHO) in 1997 and published in 2000, provide the values lists in the table WHO, (2000).

<table>
<thead>
<tr>
<th>BMI</th>
<th>Classification</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 18.5</td>
<td>Underweight</td>
</tr>
<tr>
<td>18.5-24.9</td>
<td>Normal weight</td>
</tr>
<tr>
<td>25.0-29.9</td>
<td>Overweight</td>
</tr>
<tr>
<td>30.0-34.9</td>
<td>Class I obesity</td>
</tr>
<tr>
<td>35.0-39.9</td>
<td>Class II obesity</td>
</tr>
<tr>
<td>&gt; 40.0</td>
<td>Class III obesity</td>
</tr>
</tbody>
</table>
Some modifications to WHO definitions have been made by particular bodies. The surgical literature breaks down. Class III obesity into further categories (Gabriel, 2006).

- Any BMI > 40 is severe obesity
- A BMI of 40.0-49.9 is morbid obesity
- A BMI of > 50 is super obese.

As Asian populations develop negative health consequences at a lower BMI than Caucasians, some nations have redefined obesity. The Japanese have defined obesity as any BMI greater than 25, Kanazawa et al. (2002) while China uses a BMI of greater than 28, Bei-Fan (2002).

BMI is considered as a better index for assessing obesity because it does away with the need of height weight tables and is not dependent upon type of obesity frame.

In various studies, BMI has been used as screening method for obesity, but different authors have used different cut-off points, for screening purposes.

<table>
<thead>
<tr>
<th>Authors</th>
<th>Year</th>
<th>Cut-off points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sood</td>
<td>1982</td>
<td>≥ 25</td>
</tr>
<tr>
<td>National Centre for Health Statistics, USA.</td>
<td>1983</td>
<td>≥ 28</td>
</tr>
<tr>
<td>Young and Sevenhyen</td>
<td>1989</td>
<td>≥ 26</td>
</tr>
</tbody>
</table>
2. **Skin fold thickness as a measure of body fat**

The measurement of skin fold thickness using specially designed calipers offers a more direct assessment of fatness. Durnin and Womersley (1974) have measured skin fold thickness at 4 sites (biceps, triceps, suprailliac, and subscapular) and correlated it with total body fat as measured by under water weighting. The interpretation of the sum of skin fold measurement at four sites is as follows:

<table>
<thead>
<tr>
<th>Sum of 4 sites skin fold</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Up to 40 mm</td>
<td>Upper limit of normal</td>
</tr>
<tr>
<td>40 to 59.9 mm</td>
<td>Above normal</td>
</tr>
<tr>
<td>60 to 79.9 mm</td>
<td>Substantial excess of fat, equivalent to overweight (defined as body weight 110% or more)</td>
</tr>
<tr>
<td>80 mm and above</td>
<td>Markedly excess fat equivalent to obesity (defined as body weight 120% or more)</td>
</tr>
</tbody>
</table>

The measurement of the skin fold thickness can provide a useful technique for evaluating total body fat. Because the thickness of skin from site to site, usually varies from 0.8 mm to 1.1 mm. The most of the distance below a fold of skin represents subcutaneous fat. By measuring this, one can estimate body fat by consulting appropriate tables for Children, Seltzer and Mayer (1965) for adults, Durnin and Womersley (1974).
Study done by Khalid and Mohd. Yunus (2005) on the ‘prevalence of obesity in urban population based on Triceps skin-fold thickness in relation to body weight’ that showed that prevalence of obesity among male and female bases on triceps skin fold thickness using Seitzer and Meyer criterion was 2.2% and 10.4% respectively. Sood found the prevalence of obesity using same criterion in females and females to be 2.1% and 10.04% respectively. Sood found prevalence of obesity in males to be 9.4% according to relative body weight criterion, which was higher as compared to prevalence of 8.0% in the study. No significant difference was found in prevalence of obesity using triceps skin fold thickness in this study when compared with Sood. Prevalence of obesity was significantly higher by relative body weight than by triceps skin fold thickness in both males and females and this was consistent body weight criterion (metropolitan standard) is better tool for assessing the prevalence of obesity as compared to triceps skin fold thickness (Seltzer Mayer Criterion).

3. PREVALENCE OF OBESITY

Prevalence of obesity varies according to age, sex and region. It is more common in adults than in children and in females than in males. Though it is much more common in the west, its prevalence is gradually increasing the developing countries also. There are different method like weight/height, cube root of weight/height; estimation of total body fat and skin fold measurement for assessing obesity and different researchers have used different anthropometric criteria for this purpose.

Sahay et al. (1991) carried out on 236 female hostlers of B.H.U. campus aged 15-55 years excluding pregnant and lactating women with
the aim to find out the extent of obesity in the study group. Height weight, skin fold thickness (biceps, triceps, suprailliac, subscapular) were measured. Prevalence of obesity by two criteria was 11.4% and 26.3%, respectively. Since on two method of obesity determinative give comparable results, it is datable as to which method should be chosen for screening of obesity in the community.

Gopinath et al. (1994) studied urban women of Delhi and reported the prevalence rate of obesity as 33.4%. The nutrition foundation of India has just completed a study on the prevalence of obesity in urban Delhi and reported the prevalence rate of overweight (BMI 25+) and obesity (BMI > 30) as 50% and 14%, respectively, Gopalan (1998). The observations regarding the European populations reduce that WHO MONICA study, (1989) the prevalence of obesity and overweight is in the range of 50-70% in people aged between 35-64 years. The prevalence of overweight and obesity in the urban middle class women of Punjab as revealed from the data of the present study is no doubt less than what has been reported for the industrialized western countries.

Wang (2001) reported the prevalence of obesity and overweight was high in the US combined prevalence was (25.4%) low in China (combined prevalence, 16.0). These clearly suggest that national socio-economic development levels influence the epidemic of obesity. The prevalence of obesity and overweight and the combined prevalence of obesity and overweight by age, sex and SES groups. In the US prevalence of obesity and overweight among American adolescents was lowest in the high income group. By contrast, in China the high income groups generally were at a higher risk of obesity. The prevalence of obesity was higher in rural areas in Russia but higher in urban areas in China.
Interestingly, in both Russia and China, but not in the US, the prevalence of obesity and overweight was higher among children than among adolescents. The difference was especially remarkable for obesity.

Sidhu and Tatla (2002) on the 'prevalence of overweight and obesity among adult urban females of Punjab: A cross sectional study' the prevalence rate of malnutrition was calculated according to the critical limit of BMI as recommended by WHO (1998) out of 1000 females, only 45 females (4.5%) are underweight and 502 (50.2%) are normal while 200 females (20%) are overweight but 121 (12.1%) are in obesity grade I, 80 (8%) in obesity grade II and 52 (5.2%) are in obesity grade III or in other words, the prevalence of overweight and obesity in the present sample 20% and 25.3%, respectively. Thus, it is apparent from the present study that nearly half of the females belonging to upper middle class in Punjab are currently, overweight/obese.

Subramanyam et al. (2002) the prevalence of obesity and overweight in adolescent girls between 10-15 years of age, among the affluent families of Chennai-two studies are compare using body mass index (BMI) as a parameter. The first study done in the year 1981 (Group I) was compared with the second study in 1998 (Group II). Group I had 707 and group II had 610 girls. Overweight and obesity were denoted by BMI above 85th and 95th percentile respectively. Results showed a 9.6% prevalence of overweight and 6% prevalence of obesity in both studies. It was also observed that the BMI for the same age in the two study periods showed an increase from 1981 to 1998. BMI approximated the International reference value for BMI at age 13 years in the year 1998.
Mohammad Ali and Lindstrom (2005) studies 17.5% proportion of the women, aged 18-34 years were underweight (BMI < 20.0), 18.4% overweight, and 7.0% obese. The prevalence of underweight according to BMI < 18.5 definition was 5.8% among women aged 18-34 years, women who were overweight/obese were unemployed, had low education, low social participation, low emotional and instrumental support, were daily smokers, had a sedentary life style, had poor self reported global health, and had lack of internal locus of control compared with normal weight women.

Sidhu and Prabjot (2005) the prevalence of overweight and obesity was assessed during a community based epidemiological survey on a randomized sample of 1700 (900 urban and 800 rural) adult Punjabi females in the age group from 20-45 years. For the assessment of overweight and obesity, height and weight measurements were taken on each subject. The prevalence rate of malnutrition was calculated according to the critical limits of body mass index (BMI). The observations show that the combined overall prevalence rate of overweight/obesity in the present study is 43.88% and 22.26%, respectively for urban and rural females. The frequency of overweight and obesity is more among urban females than in their rural female counterparts.

Cynthia et al. (2006) in 2003-2004, 17.1% of US children and adolescents were overweight and 32.2% of adults were obese. Tests for trend were significant for male and female children and adolescents, indication an increase in the prevalence of overweight in female children and adolescents from 13.8% in 1999-2000 to 16.0% in 2003-2004 and an increase in the prevalence of overweight in male children and adolescents
from 14.0% to 18.2%. Among men, the prevalence of obesity increased significantly between 1999-2000 (27.5%) and 2003-2004 (31.1%). Among women, no significant increase in obesity was observed between 1999-2000 (33.4%) and 2003-2004 (33.2%). The prevalence of extreme obesity (body mass index-40) in 2003-2004 was 2.8% in men and 6.9% women. In 2003-2004 significant differences in obesity prevalence remained by race ethnicity and by age. Approximately 30% of non-Hispanic white adults were obese as were 45.0% of non-Hispanic black adults and 36.8% of Mexican Americans. Among adults aged 20 to 39 years, 28.5% were obese, while 36.8% of adults aged 40 to 59 years and 31.9% of those aged 60 years or older were obese in 2003-2004.

Toryila et al. (2009) the prevalence of overweight and obesity in Zaria, Kaduna state, Northern Nigeria and to establish the relationship between overweight and obesity with age, sex and blood pressure. 3250 healthy adults Nigerian civil servants were used in the study. The population composed of 1750 male and 1500 female with age range from 25 to 65 years, were examined for overweight and obesity using body mass index (BMI) and waist hip ratio, BMI was calculated as weight (kg) divided by height (m²). Blood pressure was measured by using sphygmomanometer. Pregnant women and all ill health persons were excluded from the study. The prevalence rate of overweight and obesity in Zaria was 29.6% (964). The prevalence rate of overweight and obesity was higher in female 16.1% (523), than in male 13.5% (441). The mean BMI was found to increase with age. The mean waist to hip ration was 1.08 for men and 0.967 for women.
4. FACTORS INFLUENCING OBESITY

Obesity is a multifactorial health problem. These factors are biological, social, dietary and nutritional, endocrine and psychological.

1. Biological factors

Biological factors may be described in terms of heredity, age, sex, parity, and also in terms of certain events in life of women such as pregnancy and menopause may be associated with weight gain.

Heredity

WHO (2000), the profile of fat distribution is also characterized by a significant heritability level of the order of about 50 per cent of the total human variation. Recent studies have shown that the amount of abdominal fat was influenced by a genetic component accounting for 50-60 per cent of the individual differences.

Joshi (2002) studies although genetically the child is not determined to be obese yet obesity in parents influences obesity in children since the food habits of parents mould those of the child.

Other genetically associated factors are the activities of the child such as fidgeting which is an important way of burning up calories. Some people who squirm and wriggle use up calories, which equal to those burnt on jogging several miles every day.

Dolson (2003-04) studied to a large extent, the weight are genetically determined probably more accurately, the weight range we can comfortably be in unless other factors intervene, is genetically determined. Before recent developments in DNA research, we don’t know much about which genes were contributing. We did know by
looking at twin, sibling and adoption studies that genetics plays a dominant role, for example, in general adoptive children have weights more similar to their biological than their adoptive parents, no matter how different the environments are. Identical twins reared in vastly different environments vis-à-vis food and activity level, but often end up looking almost similar in most cases, including weight.

Like height, weight is heritable, one recent review suggests that twin and adoption studies point to a genetic contribution for BMI of 40-70%. Farooqi (2005), while a more extensive, but earlier, review of familiar resemblance suggests that genetic factors explain 50-90% of BMI. Maes, Neale and Eaves (1997). The results of genetic studies, where presented separately for males and females, appear broadly similar, overall, the finding of such studies mean that genetic factors, determine individual susceptibility of gain weight. Such ‘thrifty’ genes provide an evolutionary advantage in time of famine when humans have to stockpile energy to survive, but a disadvantage when food is plentiful, Lev-Ran (2001). However, as several authors point out, while the propensity for obesity may have existed for a long time, the recent rapid rise in rates demonstrates the central role of environmental factors, Rennie et al. (2005).

Now, however, the actual genes involved in obesity are being identified and the situation is incredibly complex. Weight is determined by many genes-some more important that others. More than 300 genes (so far) have been linked with obesity. Where our fat is deposited, how insulin is regulated, how much of various hormones our bodies secrete, and many, many more, for most of them, we don’t understand the
mechanisms yet. The next few years will be most interesting in that regard.

Clayson (2007) studied but let’s focus on one of the less obvious aspects of childhood obesity for a minute while the causes of obesity are well reported—namely lack of exercise and bad eating habits—there are also familiar and hereditary instances of obesity that are less often analyzed. Children whose parents are obese have a much greater chance of becoming obese themselves. Perhaps this has to do with genetic factors, but more often that not it has to do with the families sedentary lifestyle and eating habits. If parents do not take proper care of themselves, then their children can be born obese.

Michaellucas (2008) before commencing on the role of genetic obesity one must keep in mind that only a small percentage of children carry genetic defects for obesity and the factors other than genetic play a key role in making a child obese. Unfortunately, most of the people don’t realize the bad influence of these factors and keep emphasizing upon the genetic factors responsible for childhood obesity.

Age

The prevalence of obesity is linked with the age of the population studied and all statistics show that it increases with age.

International Children’s Centre, Paris (1984) reported that a study in USA to gain most weight between age of 29 and 35 years, while women gain most between 45 and 49 years of age.

Padma Kumari (1990) reported that prevalence of obesity increased with age. In her study, they found the maximum prevalence of obesity in women was seen between the age group of 40-49 year (66.7%) whereas
in another study the prevalence of obesity was about 90% among the women in the age group of 45-55 years, Young and Sevenhuayen (1989).

Brays (1994) shows that for a man aged 45 and increase of 12 kg above standard weight reduces his life expectancy by 25 per cent. The relative risk associated with obesity decrease with age over 75 years.

Seidell (1997) increasing with age atleast up till age 50-60 years in man and women.

Asthana et al. (1999) studies a direct relationship is observed between age and obesity. The prevalence of obesity showed an increasing trend over the successive age period, increasing from eight per cent in the age groups 15 to 20 years to 50 per cent in women aged 50 years and above. The mean age of obese and non-obese women was also calculated and it was significantly higher in obese (30.91 yrs.). This could be because of cumulative effect evident in late adulthood as compared to adolescent or early adulthood periods. In a study, Padma Kumari (1990) found maximum prevalence of obesity (66.77%) in women 40-49 years, so increase in the prevalence of obesity by age.

Park (2005) obesity can occur at any age, and generally increased with age, infants with excessive weight gain have one increased of obesity in later life. About one third of obese adult have been so since childhood. It has been well established that most adipose cells are formed early in life and the obese infant lays down more of these cells (hyperplastic obesity) than the normal infant. Hyperplastic obesity in adults is extremely difficult to treat with conventional method.

Haslam (2007) it was not until the 20th century that it became common, so much so that in 1997 the WHO formally recognized obesity
as a global epidemic Caballero (2007). As of 2005 the WHO estimates that at least 400 million adults (9.8%) are obese with higher rates among women than men WHO (2009). The rate of obesity also increases with age at least up to 50 or 60 year old. once considered a problem only of high-income countries obesity rates are rising world wide. These increases have been most dramatically in urban setting WHO (2009). The only remaining region of the world where obesity is not common is sub-sahara, Africa, James (2005).

Anjel Vahratian (2009) includes prevalence data on body mass for our study population, stratified by both age and race and Hispanic origin, overall, 24.5% of women 20-44 years of age were overweight and 23.0% were obese. Among those who were obese, 10.3% met the criteria for class II or III obesity. The prevalence of both overweight and obesity increase with age, the prevalence of overweight ranges from 20.4% in 20-29 years olds to 26.9% in 40-44 year olds. Similarly, the prevalence of obesity increased across age ranging from 19.1% in 20-29 year olds to 25.6% in 40-44 year olds. When stratified by race and Hispanic origin the prevalence of overweight and obesity respectively was highest among non-Hispanic black women (30.0% and 34.1%), followed closely by Hispanic (29.7% and 22.2%), non-Hispanic white (22.8% and 21.4%) and non-Hispanic other women (17.4% and 15.2%). Age trends persisted in this stratified analysis.

Sex

International Children’s Centre, Paris (1984) reported that women generally have higher rate of obesity than men, although men may have higher rates of overweight.
Kelly *et al.* (1984) examined the prevalence of obesity in males and females that 9% of male subject and 16% of female subjects aged 21 years or over were obese. The prevalence and degree of obesity were both greater in females than in males.

Sood *et al.* (1984) examined 509 males and 517 females of Delhi. They reported that 38.7% of male and 72.7% females were classified as obese on the basis of per cent body fat. Such high rates may be due to the equation use to calculate body density being derived from measurement on subject with different racial and ethnic backgrounds.

Retner *et al.* (1991) reported that current recommendation for appropriate weight gain in pregnancy suggest an optimum of 120% of ideal weight at delivery. This represents an increase of approximately 24 pounds in the normal weight women, and even the obese patients were observed to gain 16 pounds.

Women's BMI increases with successive pregnancies. The recent evidence suggested that this increase is likely to be on an average about 1 kg per pregnancy. On the other hand, in many developing countries consecutive pregnancies at short intervals are often associated with weight loss rather than weight gain WHO (2000).

Lobstein and Frelut (2003) the review of surveys conducted within Europe in the 1990s found the number of countries with higher prevalence for overweight and obesity among females was almost the same as that with higher prevalences for males. It is possible that gender differences may emerge in future, a study conducted in the US found significantly greater increases in rates among males between 1986-98 Strauss and Pollack (2001). It has therefore been suggested that
prevalence estimates for both males and females should always be presented Reilly (2005).

Schwarz (2007). Body fat percentage is total body fat expressed as a percentage of total body weight. It is generally agreed that men with more than 25% body fat and women with more than 33% body fat are obese.

Menopause

Weight gain might also be associated with menopause (Asthana et al., 1999) whether the women tend to gain weight near menopausal phase of life the prevalence of obesity was calculated around pre-menopausal age period. It is clearly evident that prevalence of obesity was high (46.84%) between 40 to 45 years age period and continues to remain high beyond 45 years (48.08%) as compared to 21.39 per cent under the age of 33 years and overall prevalence of 30.24 per cent. Thus, it could be clearly missed that prevalence of obesity started increasing after 35 years of age but become more pronounced after 40 years. Though no hormonal estimates were done, but one can speculate that these changes in the increased prevalence of obesity could be due to hormonal changes that occur in this period of women’s life.

Parity

Swaminathan (1988) has quoted that a healthy women gain on an average about 12.5 kg weight during pregnancy and about half of this consists of body fat stored in adipose tissue. The association between parity and obesity could be explained terms of certain hormonal changes in women during pregnancy like secretion of leutinising hormone coupled
with possible energy imbalance and mood variations, which may lead to deposition of adipose tissue and resultant obesity.

Asthana and Gupta (1999) studies the prevalence of obesity was found to be higher (51.28%) in women with parity more than three as compared to prevalence of 32.12% in women with parity three and less (P<0.01) high prevalence of obesity was associated with parity.

Alessandra et al. (2007) studied frequency of obesity was 28.4% a higher than the frequency of obesity observed in other Brazilian population samples. After multivariate adjusted, compared with women with less than 3 child births women with 3 childbirths or more were more overweight [odds ratio (OR) 3.4, 95%, confidence interval 95% CI, 1.2-9.6] and more obese [OR, 53, 95% CI, (1.3-17.5)]compared to black women, white women were also more obese (OR, 9.9, 95% CI, 1.5-64.6). In the sample of low income women, parity and race were the most important factors associated with obesity.

Koch et al. (2008) in MLAR (Multiple Linear and Logistic regression models) a modest parity-related increment in BMI and practically null increment in WC, WHR and WHIR was observed. Covariables that showed a statistically significant association with anthropometric measures of adiposity were age, law education, marital status, employment, smoking, smoking cessation, hypertension, diabetes, dislipidaemia, parent’s obesity. Menarche and fetal macrosomia. Crud Odds ratio (OR) showed a strong association between parity and anthropometric markers of obesity. All the measures of abdominal obesity related to parous women showed OR smaller than 1 (95%
confidence intervals 0.57 to 0.96) parity can increase adiposity but not necessarily following an abdominal patterns.

Pregnancy can have a significant impact on women's long term weight and risk of being overweight nulliparous women tend to be less overweight compared to multiparous women, and there is a dose-response effect the more children a women has the more likely she is to be overweight. In the past the primary focus of prenatal nutrition counseling was to prevent deficiencies. Today the growing epidemic of obesity and associated chronic illness has drawn attention to the problem of over nutrition during pregnancy.

2. **Social factors**

Socio-economic factors have a strong influence on the prevalence of obesity.

Gupta *et al.* (1998) study from Jaipur showed that among 237 children (13-17 years of age) from middle and upper middle class, 24 (10.1%) were obese as defined by BMI above the 95th percentile.

Martikainen and Marmot (1999). The increase in overweight and obesity has been extensively documented in industrialized societies, showing over the last quarter of a century a transition from a positive to a negative association between income and obesity. A similar phenomenon is new emerging in developing countries. Monteiro *et al.* (2000) and Vauy *et al.* (2001) and in some Latin American middle income countries, where greater levels of obesity among the poor are observed, especially in urban areas, conversely, less developed, societies and rural environment tend to display a more positive association between overweight and social status.
Reilly, Martorell (2000) and Bundred et al. (2001) studies based on relationship between obesity and socio-economic status have been conducted the world over. A rapid increase in the prevalence of obesity in children has been seen in England and United States of America and around the world.

Pena and Bacallao (2000), Latin America is in the middle of an epidemic of obesity that transcends socio-economic boundaries and affects the highest socio-economic level as well as the most disadvantaged.

Wang (2001) shows that child and adolescent obesity is related to SES, although the relationship differ among these three populations. We used family income as a primary indicator of SES, while rural urban residence might serve as an additional indicator. In the US low SES groups had a higher risk of obesity. By contrast in China high SES groups were at an increased risk. In Russia a transitional society that has experienced economic difficulties since the early 1990’s, both low income and high income groups were at an increased risk of obesity compared to the medium income group. One possible explanation for the different SES obesity relationship is developed countries such as the US and developing countries such as China is that the influence of SES on people’s lifestyles such as diet and physical activity may differ. Take food consumption patterns as an example. In China richer people have better access to meal and other energy-dense foods (which are much more expansive than other foods such as vegetables) than the poor. While is US higher SES groups usually consume more vegetables and fruits, which are less energy dense than low SES groups.
Ball et al. (2002) results demonstrated associations for women, after controlling for age between the employment domain and body mass index and waist to hip ratio. Low status employed women were 1.4 times as likely to be overweight as high status employed women. There were less consistent relationships observed among these factors for men, relationships between family unit and indicators of body weight and body fat distribution were observed for both men and women with those who were married.

Penny Gordon et al. (2003) keeping adolescents in their same environments and changing only family income and parental education had a limited effect of the disparities in overweight prevalence. Ethnicity-SES overweight differences were greater among females than males. Given that overweight prevalence decreased with increasing SES among white females and remained elevated and even increased among higher SES African-American females. African American/white disparity in overweight prevalence increased at the highest SES. Conversely, disparity was lessened at the highest SES for white, Hispanic and Asian females, Among males disparity was lowest at the average, SES level.

Mohammad Ali and Lindstrom (2005) obesity is a growing health problem in many countries. In Sweden an increase in the obesity prevalence was observed during the 1980’s and 1990’s in all socio-economic groups. Obesity is a risk factor for hypertension, cardiovascular diseases, diabetes, and total mortality. Obesity (BMI 30.0 or more) and overweight (BMI 25.0-29.9) are positively associated with increasing age, low level of leisure time physical activity, and in some groups differing dietary habits. In Sweden obesity and overweight are associated with lower levels of education and low socio-economic status.
In respect of socio-economic status (SES), studies suggest higher rates of obesity among low income groups in richer countries and high income groups in poorer ones (Lobstein et al., 2004). It is suggested that this is because in developing nations, higher SES individuals have become globalized, with easy access to relatively cheap, calories dense foods, while those of lower SES remain localized and undernourished. Sobal (2001) within the US, increases have also been greatest among children and adolescents from the lowest income families, so increasing SES disparities. Strauss and Pollack (2001) however, not all studies in developed countries find SES differences. Saxena et al. (2004) and Taylor (2005) and among those that report separately, there is some evidence that SES differences may be clearer among females than males Wardle et al. (2006).

Kaneria et al. (2006) study, it was observed that there has been a significant increase in overweight (4.85%) and obesity (3.73%) in children belonging to affluent and upper middle class income group. In affluent group children prevalence of overweight was relatively higher in the age group 14 and 16, but obesity was greater in 16 and 17 years age groups. Hence the present study has highlighted that obesity is an emerging health problem in adolescent children belonging to affluent families in Udaipur.

McLaren (2007), the correlation between social class and BMI varies globally. A review in 1989 found that in developed countries women of high social class were less likely to be obese. No significant differences were seen among men of different social classes. In the developing world, women, men, and children from high social classes had greater rates of obesity. Sobal and Stunkard (1989) an update of this
review carried out in 2007 found the same relationships, but they were weaker. The decrease in strength of correlation was felt to be due to the effects of globalization.

Many explanations have been put forth for associations between BMI and social class. It is thought that in developed countries, the wealthy are able to afford more nutritious food, they are under greater social pressure to remain slim, and have more opportunities along with greater expectations for physical fitness. In undeveloped countries the ability to afford food, high energy expenditure with physical labour, and cultural values favoring a larger body size are believed to contribute to the observed patterns (Mclaren, 2007). Attitudes towards body mass held by people in one’s life may also play a role in obesity. A correlation in BMI changes overtime has been found between friends, siblings and spouses (Christakis and Fowler, 2007).

**Marital status**

Asthana and Gupta (1999) analysis of marital status with reference to obesity was done to verify thus association. If this association is true then prevalence of obesity should be low in unmarried women as compared to married and widow women. Age adjusted prevalence rates were calculated. The result showed that age adjusted prevalence was lowest among unmarried (6.24%) as compared to married (35.28%) and widow (33.92%) women. The possible reasons for higher prevalence of obesity in married women could be intake of calorie dense food during each pregnancy and lactational period. Weight gain during each pregnancy, half of which is constituted by body fat and stress and strain of pregnancy and feeling of insecurity among widows, leading to anxiety,
depression and stress. All these situations start then to eat more in frequency and quantity, either to reduce tension or as a substitute gratification when more pertinent and realistic gratifications are unavailable.

Woaj Leug et al. (1999) higher educational level seems to be connected with a lower risk of being overweight, but this is only seen in women, according to research into the influence of marital status and education on consumption pattern of food groups, nutrient intake, blood pressure, waist-hip ratio and other laboratory parameters among Hong Kong Chinese subjects. Higher levels of education appear to be linked with a healthier dietary pattern in relation to chronic diseases. It is clear that there is a needed to consider the impact of individual socio-economic factors on dietary intake and cardiovascular risk factors.

3. **Dietary habits and nutritional factors**

Blundell et al. (1996) study dietary energy intake and excess body weight are positively associated. Among the various dietary factors, dietary fat and fiber have opposite effects. Dietary fat with high energy density and palatability with poor appetite control signals tends to increase energy intake and body fat, while the fiber limits energy intake by reducing density of fats. Subjects on high fat diet generally tend to over consume energy ‘passive over consumption’.

In Indian diet, the contribution of saturated fats like vanaspati and ghee to total fats is generally small. NFI, (1999) scientific report-15 study, the mean frequency of intake of refined oil, the most common cooking oil medium, was found to be significantly high among the overweight section. There was significant direct relationship with tertiles
of BMI and WHR especially in the males and BMI in females. However, it is difficult to draw meaningful conclusions from the frequency of food intake data alone without taking into consideration the actual quantity consumed. A positive trend of more frequent intake of ‘rich’ foods in subjects with higher tertiles of BMI and WHR may account for fat accumulation.

Srilakshmi (2005) nibbling between meals is common among housewives and is a potential cause for obesity and some may eat faster taking less time for chewing, therefore they tend to consume more food or sometimes housewives who do not want leftover foods to be thrown out may consume forcibly and put on weight WHO (2000). Nowadays television and print media is playing an important role in energy-dense micronutrient poor food and beverages (usually classified under the ‘eat least’, category in diet guidelines) of multinational corporations, which influence the daily eating habits. The consumer demand by itself may be influenced by advertising marketing, culture, fashion and convenience.

Regular consumption of high energy-dense fast foods and sugary drinks which are associated with less satiation and so insufficient compensation via subsequent reductions in intake, increased portion size, eating outside the home and snacking have been particularly implicated in promoting weight gain. This is especially the case among older children, who are less influenced by biological cues of satiety, Agras and Mascola (2005).

Obesity is related to an imbalance between energy input and output, the size of which may be very small if over a long period. Reilly (2007) one review suggests that in children, an imbalance of around 2%,
which is the equivalent of around 30 calories or 15 minutes of TV instead of play a day, may lead to obesity, Goran (2001). Behavioural determinants therefore include excess energy intake and inadequate energy expenditure. Prentice and Jebb (1995) although the emphasis given to these 'Big Two, and the neglect of other plausible contributors to the secular increase in obesity has recently been questioned, Keith et al. (2006).

Duvigneaud et al. (2007) gives an overview of the difference in plausible dietary intake between BMI and WC (waist circumference) groups in women. Significantly higher values for carbohydrates, starch, fibre, fat (saturated, mono and polyunsaturated) and total energy intake were observed in overweight and obese women compared to normal weight women. Obese women also reported significantly higher intake of protein, sugar and iron intake than their normal weight counterparts. Similar findings were observed between the WC groups. Women with high risk abdominal obesity reported a significantly higher cholesterol intake and higher energy intake from alcohol compared to women with normal WC or moderate risk for abdominal obesity.

The widespread availability of nutritional guidelines have done little to address the problems of overeating and poor dietary choices, Marantz et al. (2008). In the period of 1971-2000, obesity rates in the United States increased from 14.5% to 30.9%, Flegal et al. (2002). During the same time period, an increase occurred in the average amount of calories consumed. For women, the average increase was 335 calories per day (1542 calories in 1971 and 1877 calories in 2004), while for men the average increase was 168 calories per day (2450 calories in 1971 and 2618 calories in 2004). Most of these extra calories come from an
increase in carbohydrate consumption rather than an increase in fat consumption, Wright et al. (2004). The primary sources of these extra carbohydrates are sweetened, beverages, which now accounts for almost 25 per cent of daily calories in young adults in America, Caballero (2007). Consumption of sweetened drinks is believed to be contributing to the rising rates of obesity, Malik et al. (2006) and Olsen et al. (2009).

A comparison of a cheeseburger 20 years age (left) which had 333 calories with a modern cheese burger right contains 590 calories.

As societies become increasingly reliant an energy dense, large portion, fast food meals, the association between fast food consumption and obesity becomes more concerning, Rosenheck (2008). In the United States consumption of fast food meal has tripled and calorie intake from fast food has quadrupled between 1977 and 1995.

4 Endocrine factors

Michaellucas (2008) studies that it must be keen in mind that all the genes for controlling different factors leading to obesity are mostly recessive and hence, express themselves intensely if they do so at all any how the most common hormonal cause hyperthyroidism in which thyroxin is not secreted profusely by the thyroid gland and the blood thyroxin level decreases which causes obesity. Another important hormonal cause is the Hypocartisolism, which is characterized by 24-hour free urinary cartisol level. This in turn leads to obesity primary Hyperinsulinism is still another hormonal cause of obesity due to genetic defects, which expresses itself. When plasma insulin increases in blood. This very factor increases the C-peptide level of the victim. Moreover pseudo hypothyroidism involves the increased PTH (Parathyroid
Hormone) level in the blood which causes the malfunctioning of many body organs and systems. All this leads to obesity.

5 **Obesity and Chronic diseases**

Obesity increases the risk of many physical and mental conditions. These co-morbidities are reflected predominantly in metabolic syndrome, James (2005). Metabolic syndrome being a combination of medical disorders, which includes diabetes mellitus type 2, high blood pressure, high blood cholesterol, and high triglyceride levels, Grundy (2004).

**Hypertension and obesity**

Aneja *et al.* (2004) study obesity is a common problem in much of the western world today in that is linked directly with several disease processes, notably, hypertension. It is becoming clear that the adipocyte is not interact with each other and may result in elevated blood pressure. Of particular importance is the putative role of leptin in the causation of hypertension via an activation of the sympathetic nervous system and a direct effect on the kidneys, resulting in increased sodium re-absorption leading to hypertension.

Bethesda (2004) study with the significant rise in obesity in this last decade comes a corresponding increase in the prevalence of hypertension. Almost 29 per cent of population is hypertensive. The relationship between obesity and BP appears to be linear and exists throughout the non-obese range. But the strength of the association of obesity with hypertension varies among different racial and ethnic groups. Generally, risk estimates suggest that approximately 75 and 65% of the cases of hypertension in men and women, respectively, are directly attributed to an overweight condition and obesity. It is important to
recognize that long-duration obesity does not appear necessary to elevate BP, as demonstrated by obesity in children without a condition of hypertension.

Obesity-related hypertension is commonly associated with further elements of the metabolic syndrome, such as insulin resistance and glucose intolerance. In particular, one should be aware that diabetes \textit{de novo} occurs in 2\% of treated hypertensive patients per year, Verdecchia \textit{et al.} (2004).

The relevance of both hypertension and obesity, as important public health challenges, in increasing worldwide, compared with the year 2000, the number of adults with hypertension is predicted to increase by 60\% to a total of 1.56 billion by the year 2025, Kearney \textit{et al.} (2005). The growing prevalence of obesity is increasingly recognized as one of the most important risk factors for the development of hypertension. This epidemic of obesity and obesity-related hypertension is paralleled by an alarming increase in the incidence of diabetes mellitus and chronic kidney disease. This editorial examines the evidence linking obesity with hypertension.

Excess body weight is the sixth most important risk factor contributing to the overall burden of disease worldwide, Haslam and James (2005). Obesity and in particular central obesity have been consistently associated with hypertension and increased cardiovascular risk. Based on population studies, risk estimates indicate that at least two-third of the prevalence of hypertension can be directly attributed to obesity, Krause \textit{et al.} (1998). Apart from hypertension, abdominal adiposity has also been implicated in the pathogenesis of coronary artery
disease, sleep apnoea, stroke and congestive heart failure, Haslam and James (2005). There is increasing evidence that obesity contributes to the development as well as to the progression of chronic kidney disease, De Jong et al. (2002).

Obesity is associated with an increased risk of cardiovascular, but this requires that obesity is combined with hypertension. In overweight and obese subjects, the cardiovascular risk is not significantly increased unless hypertension is present, Thomas et al. (2005). This observation underscores the role of hypertension as mediator through which obesity may cause cardiovascular disease while obese subjects are prone to hypertension, hypertensive subjects also appear to be prone to weight gain. Both the Framingham and Tecumseh studies have shown that future weight gain is significantly greater in hypertensive patients than in normotensive subjects, suggesting that even normal weight hypertensive are at a high risk of developing obesity, Julius et al. (2000). Therefore, the relationship between obesity and hypertension might be described as a 'two way street'. Julius et al. (2000) implying individual susceptibility to both conditions or common environmental factors. It is clear that obesity related hypertension is a multifactorial disorder. At this time, it is not possible to identity one single mechanism as the dominant a etiological factor. Genesis and evolution of obesity related co-morbidity presumably depend on several genetic and environmental factors. It is likely that obesity, hypertension and metabolic abnormalities interact and potentiate their individual impact on cardiovascular risk, Keller et al. (2003).

Rahmouni et al. (2005) study obesity is strongly associated with hypertension and cardiovascular disease. Several central and peripheral abnormalities that can explain the development or maintenance of high
arterial pressure in obesity have been identified. These include activation of the sympathetic nervous system and the renin-angiotensin-aldosterone system. Obesity is also associated with endothelial dysfunction and renal functional abnormalities that may play role in the development of hypertension. The continuing discovery of mechanisms regulating appetite and metabolism is likely to lead to new therapies for obesity-induced hypertension.

Diabetes Mellitus and Obesity

Increased risk posed by intra-abdominal fat for diabetes and other metabolic disease could be related to higher fat cell number in the abdominal adipose tissue, higher blood flow, increased receptors for cortisol and testosterone and greater catecholamine-induced lipolysis when compared with the subcutaneous adipose tissue, Must et al. (1992). In addition there is a marked increase in flux of non-esterified fatty acids to the liver in abdominally obese subjects. There is sufficient evidence to show that abdominal obesity causes insulin resistance and it is a key component of the metabolic syndrome. Racial susceptibility to insulin resistance and metabolic syndrome has been demonstrated and Indians are highly susceptible to both, Snehlata et al. (1999).

Banerjee and Chandalia et al. (1999) reported that a cluster of risk factors co-exists with central obesity including glucose intolerance, obesity, hyperinsulinaemia, hypertriglyceridaemia and hypertension, all of these are important risk factors for CHD. Studies in migrant Asians comparing body fat topography with that in Caucasians have confirmed similar findings. Mc Keigue et al. (1991) reported that in Asian Indian every 0.04 unit increase in WHR was associated with 4-fold increase in
diabetes (20% in Asians, 5% in Europeans), 2-fold higher post glucose insulin levels (41 μu/ml in Asians Vs. 19 μu/ml in Europeans) and significantly higher triglycerides and low high density lipoprotein (HDL).

A positive association between overweight and obesity and risk of type 2 diabetes has been established repeatedly in many cross-sectional and prospective studies, WHO (2000) and Ramchadran (2001). It was shown that the risk conferred by obesity for developing diabetes was higher by 40 times in obese women compared to those who remained slim, and the risk would be reduced significantly weight loss. The association of obesity with type 2 diabetes is complex and compounded by many heterogeneous factors. Obesity not only is a risk factor for development of diabetes, but also complicates the management of the disease.

Asian Indians generally have lower BMI than many other races but the association of BMI with glucose intolerance is as strong as in any other population, Ramchandran et al. (2001). It was shown for urban Indian population that at a BMI of > 23kg/m² the risk for diabetes was significant for both genders, Snehlata et al. (2003). Therefore the healthy BMI for an Indian is definitely below 23 kg/m². This has been confirmed by studies from other parts of India. Banerjee and Chandalia et al. (1999) and also from other Asian populations. According to the WHO recommendations, a BMI of 18.5-22 kg/m² has been considered as healthy for Asian populations.

6 Psychological factors

Psychological factors in the development of obesity are widely recognized. Ryden (1981) found that obesity tended to occur in subject
with elevated levels of anxiety, tension, impulsiveness and aggression. Flangan et al. (1991) also indicated that obese subjects showed more anxiety than did control subjects.

Black et al. (1992) examined the prevalence of mental disorder in morbidly obese women. The morbidly obese subjects were more likely than the non-obese group to have a lifetime history of mood disorders, anxiety disorders, and tobacco dependency. The morbid subjects were also more likely to meet diagnostic criteria for one or more personality disorders.

Overweight people generally consume more calories than people of normal weight but this does not mean they are overeating, as they are consuming an appropriate amount of calories for their height body weight (assuming weight stability). This distinction is important, as the idea that obese individuals bring about or maintain their obesity by inappropriate overeating underlies many of our cultures negative stereotypes about obesity. Obese patient who reported recurrent uncontrolled binge eating. Many of these patients would meet the criteria for binge eating disorder, regular binge eating in the absence of the extreme weight loss behaviour characteristic of bulimia nervosa. Compared with non-binge eaters eat significantly more food in laboratory studies when instructed to binge or eat normally, report an earlier onset of obesity and greater percentage of lifetime on a diet; overeat more in response to negative emotional states report lower levels of self-esteem, and display significantly greater levels of psychopathology, especially, depression and personality disorders, Kuehnel, Waddden (1994) and Mussell et al. (1996).
Another abnormal eating pattern that exist most commonly in obese individuals has become known as the "night eating syndrome". First described in 1955, its key features are morning anorexia, evening hyperphagia and insomnia. More recent clinical reports have suggested that many of these patients suffer from sleep disorders such as somnambulism, restless syndrome, and obstructive sleep apnea, Schenck and Mahowald (1994) and the most patients report some degree of amnesia for the eating episode, Winkelman (1998). In addition, night eating syndrome is associated with neuro-endocrine abnormalities including attenuated nocturnal rise in leptin and melatonin increased plasma.

Simon (2004) a psychiatrist and researcher at the University of Washington's Center of Health Studies-Group Health, said in an interview. However, the association appears to be generally stronger for women than for men, he pointed out. Indeed, in his own recent study of some 2,300 women, which he reported at APA's 2005 annual meeting in May, he found that major depression was twice as prevalent among women with a body mass index (BMI) greater than 30 than in women with a BMI less than 30. A BMI of 30 or more indicates obesity.

Treichel (2005) study the obesity epidemic is firing scientist up to learn more about the psychological ramifications of obesity. This is an area that psychologists, and particularly psychiatrists, did not pay much attention to until the past decade or so, 'Grow told psychiatric News'. "In the field of psychiatry we have really started to get the message about this epidemic and are working to try to understand it'. 'I know that researchers are looking a lot more at the psychological aspects of not just binge eating disorder, but of obesity in general'.

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5 MANAGEMENT OF OBESITY

The goal of any reducing programme should not be merely to lose weight but to maintain normal limit once it is achieved. The role of diets or calories restriction, physical exercise and yoga, psychotherapy, behaviour modification, drugs and surgery are described below.

1. Dietary management

Diets to promote weight loss are generally divided into four categories, low fat, low carbohydrate, low calorie, and very low calorie, Strychar (2006). A meta-analysis of six randomized controlled trials found no difference between the main diet types (low calorie, low carbohydrate and low fat), with a 2-4 kilogram (4.4-8.8 lb) weight loss in all studies, Strychar (2006). A two years, all diet methods resulted in similar weight loss irrespective of the macronutrients emphasized, Sacks et al. (2009).

Low fat diets

Samaha et al. (2003) provided 33% of total calories intake as fat, which is more than the 20-30% energy intake normally indicative of a low fat diet. In addition, dietary compliance was not assessed by Foster et al. (2003). The three studies are important, but are not evidence that low carbohydrate diet in the long term are superior to the energy restricted low fat diet.

Strychar (2006) studies low fat diets involve the reduction of the percentage of fat in one's diet. Calorie consumption is reduced but not purposely so, diets of this type include NCEP step I and II. A meta analysis of 16 trials of 2-12 months' duration found that low fat diets resulted in weight loss of 3.2 kg (7.1 lb) over eating as normal.
Low carbohydrate diets

Bravata et al. (2003) studied how carbohydrate diet such as Atkins and protein power are relatively high in fat and protein. They are very popular in the press but are not recommended by American Heart Association. A review of 107 studies did not find that low carbohydrate diets cause weight loss, except when caloric intake was restricted, Hession et al. (2009). No adverse effects from low carbohydrate diets were detected.

Daniels (2003) the conventional dietary approach for obesity is high carbohydrate, low fat, energy deficient diet but weight loss is not substantial as processed starchy foods and sugars in these diets prevent effective weight loss. Low carbohydrate diets have been popular since 1860 when William Barting claimed to lose weight without feeling hunger, Krauss et al. (2000) and Freedman et al. (2001). The most popular diet among them is Atkins diet which is the most widely prescribed low carbohydrate diet. Whereas the Atkins diet permits no more than 5-10% of calorie intake from carbohydrate, Wilett’s new food pyramid (with which the Atkins diet is often confused) allow 40-45% of calorie intake from wholegrain foods, fruit, and vegetables.


Eating plan in new diet revolution describes not simply a diet but a ‘Lifetime nutritional philosophy’ with vitamin and mineral supplementation and regular exercise. It consists of four phases:

1) *Induction*

In induction phase there is carbohydrate restriction to 20 gm/day in from of salads and non-starchy vegetable.
2) **Ongoing weight loss**

Calories are added in from of nutrient dense and fiber rich food by increasing carbohydrate to 25 g/day for first week, 30 g/day next week and so on until weight loss stops then subtract 5 g from the earlier so that continued sustained moderate weight loss occurs.

3) **Pre-maintenance**

Transfer from weight loss to weight maintenance phase. Increase intake in 10 g increment each week till gradual weight loss is maintained.

4) **Life time maintenance**

Select from wide variety of food while controlling carbohydrate to ensure weight maintenance and sense of well being.

Putative mechanism of weight loss on low carbohydrate diet

Krauss et al. (2000). A systematic review of low carbohydrate diet reported that the weight loss is associated with only the duration of diet and restriction of energy intake, not with carbohydrate restriction itself. Bravata et al. (2003) on review of 107 articles prior to 2003 of it was found that only 56 studies had follow up to more than 90 days and none were randomized control trials and had no control group. This constituted insufficient evidence to make recommendations.

Samaha et al. (2003) studies 132 severely obese individuals (39% had type 2 diabetes, and 43% had metabolic syndrome) were randomized to either an ad-libitum low carbohydrate diet or an energy restricted low-fat diet for 6 months. Those on the low carbohydrate diet had lost 3-9 kg more weight after 6 months (95% CI 1-6 to 6-3 kg) but at 12 months the difference was no longer significant, (1.9 kg-1.0 to 4.9) Stern et al.
(2004). In another 6 month study, 53 obese women were again randomized to comparative diets Foster et al. (2003), and the low carbohydrate group again lost more weight [8.5 (SD 1.00) Vs. 3.9 (SD 1.0) kg after 6 months].

**Low calorie diets**

Strychar (2006) studies that low calorie diet usually produce an energy deficit of 500-1000 calories per day, which can result in a 0.5 kilogram (1.1 Lb) weight loss per week. They include the DASH diet and weight watcher among others. The National Institutes of Health reviewed 34 randomized controlled trials to determine the effectiveness of low-calorie diet. They found that these diets lowered total body mass by 8% over 3-12 months.

**Very low calorie diets**

Strychar (2006) very low calorie diets provide 200-800 Kcal/day, maintaining protein intake but limiting calories from both fat and carbohydrates. They subjects the body to starvation and produce an average weekly weight loss of 1.5-2.5 kg (3.3 -5.5 lb). These diets are not recommended for general use as they associated with adverse side effects such as loss of lean muscle mass, increased risk of gout, and electrolyte imbalances. People attempting these diets must be monitored closely by a physician to prevent complications.

2. **Physical activity and practice of Yoga and Pranayam**

**Physical activity**

Ferro-Luzzi and Martino (1996) in the present study, obese individuals were engaged in sedentary occupations. However, it is
difficult to draw conclusions as to cause and effect relationship between BMI and physical activity. It is difficult to indicate whether individuals prefer desk jobs because of their obesity or the more sedentary work style caused the obesity. More prospective data will help to clarify this relationship. The assumption that the sedentary life style is the case of overweight, however, seems to be justified and reasonable.

WHO (1998) physical activity levels (PAL) are the universally accepted way of expressing energy expenditure. In this study, the obese with desk jobs and with higher BMI and WHR were found to take regular walking exercise. In this case exercising was apparently restored to as a way-albeit inadequate-of combating the obesity that has already set in. Relatively less obese individuals were involved in household chores like cleaning, cooking, marketing and gardening. People may be likely to overestimate their physical activity levels and this may be even more true for overweight individuals, Waish et al. (2004).

This could lead to results suggesting that obesity is more important than physical activity as a cause of chronic disease due to the misclassification of physical activity. Using the objective measure of cardio-respiratory fitness probably lead to less misclassification and results in finding stronger associations with health outcomes we found that measured cardio-respiratory fitness predicted mortality better than self reported physical activity, Wei et al. (2000).

NFI (1999)- Scientific Report-15 traditional lifestyles are generally associated with gain in body weight with age. However, modernization apparently has profound effects on body weight. Rapid urbanization and industrialization change in lifestyles and eating habits, and sedentary
occupations contribute to energy imbalance. Easily availability of processed, ready to eat fast foods, which tend to be relatively less in complex carbohydrates, high in fat, and rich in sugars and cream, increase energy intake. Thus social and economic pressure in the environment contribute to obesity through changes in dietary habits and physical activities.

Both work time and leisure time activity may change considerably throughout the world due to modern gadgets such as computers and television. A sedentary lifestyle, therefore, favour a positive energy balance and weight gain. The relationship between physical inactivity and obesity however complex, several confounders are likely to complicate the picture such as physical fitness, opportunities for exercise, diet and temporal relationships between exercise and meals. It is yet not clear whether long-term low-cost activity is superior to short-term high cost activity in the maintenance of energy balance.

A sedentary lifestyle plays a significant role in obesity Seidell (2005) worldwide there has been a large shift towards less physically demanding work. WHO (2009) and Ness-Abramof or Apovian (2006) and currently at least 60% of the world's population does not get sufficient exercise. This is primarily due to increasing use of mechanized transportation and a greater prevalence of labour saving technology in the home, WHO (2009) and Aramof or Apovian. World trends in active leisure time physical activity are controversial. The World Health Organization indicates that worldwide people are taking up less active recreation pursuits however a study from Finland, Barodulin et al. (2008) found an increase and a study from the United States found leisure time physical activity has not changed significantly, Brownson et al. (2005).
With use, muscles consume energy derived from both fat and glycogen. Due to large size of leg muscles, walking, running, and cycling are the most effective means of exercise to reduce body fat. Exercise affects macronutrient balance. During moderate exercise, there is a shift to greater use of fat as a fuel, Sahlin et al. (2008).

A meta-analysis of 43 randomized controlled trials by the Cochrane collaboration with diet, however, it resulted in a 1 kilogram weight loss over dieting alone. A 1.5 kilogram (3.3 lb) loss was observed with a greater degree of exercise Shaw et al. (2006). Even though exercise as carried out in the general population has only modest effects, a dose response curve is found, and very intense, exercise can lead to substantial weight loss. During 20 weeks of basic military training with no dietary restriction, obese military recruits lost 12.5 (27.6 lb) Lee, Kumar and Leong (1994). High levels of physical activity seems to be necessary to maintain weight loss, Bessesen (2008).

A systematic review found that people, who use pedometers during an average an 18-week period, increased their physical activity by 27% and subsequently decreased their BMI by 0.38, Bravata et al. (2007).

Practice of Yoga and Pranayam

Yoga is gentle way to bring a balanced attitude to all aspects of life. Yoga can help you control your weight more effectively, whether you need to lose weight or to gain it.

Many a times the glands are sluggish in our body. These sluggish glands are stimulated by Yoga asana to increase their hormonal secretions. Especially the thyroid glands pays important role in our weight because it affects body metabolism, Alan et al. (2006).
Mandlik and Dorle (2008) study Yoga has considered all aspects of obesity (Physical, emotional and mental).

- Regular practice of yoga and controlled life style reduces obesity (weight is reduced).
- Yoga makes human being agile, efficient and slim.
- Yoga is suitable for people in any age group.
- Yoga helps achieve control over mind and behaviour (one can easily control food habit and change life style to reduce the obesity).
- Yoga has different effect on obesity, which is permanent in nature than other techniques for obesity reduction. Weight loss is permanent but one needs to practice few important techniques regularly.

Mandlik and Dorle (2008) study practice of yoga and various exercises.

- Regular exercise like running, swimming etc.
- Yogasanas like Paschimotannasana; Saral Hasta Bhujangasana, Sarvangasna, Halasana, Dhanurasana, Veerasana, Trikonasana, Arotha Matsyendrasana etc.
- Along with yogasanas Sun salutation is very effective for obesity reduction, also pranayama, cleansing processes like agnisar, Uddiyan bandha etc helps.

Regular practice of yoga can certainly reduce the obesity and weight loss, but one needs to learn specific yoga techniques, which are more effective for reducing the weight, we will be studying such
techniques in this article, Yoga Vidya Dham has a separate obesity reduction program, more than 5000 people have been benefited by this program.

Hunger and thirst control by Yoga asana and Pranayama?

According to Alan et al. (2006) - Yes; Hunger, thirst, sleep, behaviour, thought can be control by yogasanas and pranayama.

Bhujangasana reduces hunger and Shalabhasana increases hunger.

By practicing both these one after the other hunger can be equipoise practicing Shitali and Shitkari pranayama, one can live with least consumption of food, water and sleep.

Many a times the glands are sluggish in our body. These sluggish glands are stimulated by yoga asana to increase their hormonal secretions. Especially the thyroid glands pays important role in our weight because it affects body metabolism.

In asanas like the shoulder stand and the fish posture thyroid gland is stimulated. It also increases fat metabolism so the fat is burnt and energy results in to better muscle tone and a higher vitality level.

Pranayama increases the oxygen intake to the body cells, including the body cells. This causes increased oxidation or burning up of fat cells. Yogic exercise induce more continuous and deeper breathing which gradually burns, sometimes forcefully, many of the calories already ingested.

The relaxation and meditation aspect of yoga has a broad spectrum. Regular practice of meditation improves concentration and will power. Meditation also may help stimulate insight into the unconscious
motivators of your eating behaviour. So the yogic practices reduce anxiety tend to reduce anxious eating. When under nervous strain we tend to gulp our food without attaining much genuine satisfaction.

The weight you gain will be healthy firm tissue, not fat. That is, yoga will tend to produce the ideal weight for you. This is due to yoga’s effect of ‘normalizing’ glandular activity, Alan et al. (2006).

3. Psychotherapy

Psychotherapy should not be considered a primary treatment for obesity. However, this does not mean that psychotherapy has no role. Both cognitive behavioral therapy and interpersonal therapy have been found to be effective in normalizing eating and reducing distress in obese patients with binge eating disorder, although neither intervention is associated with significant weight loss Devlin (1996). Psychotherapy may be helpful in enhancing self-acceptance in obese patient who have learned to feel ashamed about their weight and may help patients to cope with the effects of prejudice and ‘weightism’ that are pervasive in our culture often greater self-acceptance and resulting increase in overall self-esteem are key steps in developing motivation for working toward a heal their lifestyle and/or for undertaking weight control treatment, Wilson G.T. (1996). Body image therapy programs have been developed to help obese individual alter the way they perceive and evaluate their bodies Rosen et al. (1995). These interventions are crucial for many obese patients. Even after successful weight loss treatment remain at a higher than normal weight. Self help organizations that promote size acceptance provide recognition and support for obese individuals and serve as a forum for addressing determination and altering harmful cultural stereotypes.
Marian Tanofsky et al. (2007) studies the most prevalent disordered eating pattern described in overweight youth is loss of control (LOC) eating, during which individuals experience an inability to control the type or amount of food they consume. LOC eating is associated cross-sectionally with greater adiposity in children and adolescents and seems to predispose youth to gain weight or body fat above that expected during normal growth, thus likely contributing to obesity in susceptible individuals. No prior studies have examined whether LOC eating can be decreased by interventions in children or adolescents without full syndrome eating disorders or whether programs reducing LOC eating prevent inappropriate weight gain attributable to LOC eating. Interpersonal psychotherapy, a form of therapy that was designed to treat depression and has been adopted for the treatment of eating disorders, has shown efficacy in reducing binge eating episodes and inducing weight stabilization among adults diagnosed with binge eating disorder. In this paper, author propose a theoretical model of excessive weight gain in adolescents at high risk for adult obesity who engage in LOC eating and associate overeating patterns. A rational is provided for interpersonal psychotherapy as an intervention to slow the trajectory of weight gain in at risk youth, with the aim of preventing or ameliorating obesity in adulthood.

Becker et al. (2007) study obesity is a serious chronic disease, associated with severe sequelae and increased mortality rates, and therefore requires long-term care. This article gives an overview of the current state of research on psychotherapeutic treatment of obesity, focusing on behavioral approaches. Systematic well controlled studies on humanistic and psychodynamic therapies are not available. A small
number of studies on psychotherapy-related approaches, e.g.- relaxation therapy or hypnotherapy, failed to demonstrate any decisive positive outcomes, while weight loss programs using methods of behaviour therapy and lifestyle modification approaches result, on the average in a short-term weight loss of 10% of the initial weight, long term effects of such programs are disappointing. Further evidence suggests, however, that long term maintenance programs may facilitate lasting behavioral changes of patients in their daily lives and work against weight regain. More research on effective maintenance programs is called for to further improve care of obese patients, it should lay stronger emphasis on internet-based weight maintenance programs.

Menard (2007) reported that empirical evidence and anecdotal clinician reports clearly suggest that negative affects is a primary trigger for binge eating and that repetitive binging can result in obesity. Many interventions, therefore appropriately target both negative affect and associated negative cognitions (thoughts). In fact, cognitive behavioral therapy (CBT) one of the most ubiquitous and well supported psychological approaches for treatment of obesity and binge eating focuses on correcting negative and or distorted cognitions. This general treatment approach can be challenging, however, when clinicians are presented with a vastly different set of triggers.

This case study present just such a scenario—that of a ‘happy eater’ unlike patients who use food to manage, stress, distress, loneliness or boredom, this subgroup of the clinical population eats out of joy and identifies, food, eating and overeating with community, family, love and well being, often, there is a cultural emphasis on the importance of food
and this trends to impact motivation, likewise, the happy eater tends to experience little distress and few negative cognitions.

Happy eaters typically have strong incentives-both external (family pressure) and internal (positive-affect)-to maintain their eating behaviors despite desiring weight loss. Treatment providers will find this type of patient present unique challenges and ultimately, considerable rewards.

4. **Behaviour modification**

Viegener *et al.* (1990) testes whether the efficiency of behaviour therapy for obesity might be improved by the use of an intermittent, low fat, low calorie diet. In a study, 60 obese women were arranged either to (i) behaviour therapy plus continuous 1200 calorie per day balance deficit diet, or (ii) behaviour therapy plus an intermittent low fat, 800 calorie per day diet, used 4 days per week. Subjects in intermittent diet condition demonstrated significantly greater weight losses than subjects in the standard treatment.

Behavioral treatment combined with a very low calorie diet of up to 800 Kcal/day, often in the form of a liquid nutritional supplement, was highly touted and widely used several years ago. Typical programs used a very low calorie diet for 12-16 weeks, followed by reintroduction self selected 1000-1500 Kcal/day diet. This approach produces more rapid weight loss initially but relapse occurs more quickly, so that treated individuals, after 1 year, show similar weight regain to those on more moderate calorie restriction, National Task Force (1993).

A comprehensive behavioral weight control program, comprising components of improved eating habits, lifestyle change, and increased exercise, is widely viewed as the treatment of choice for overweight and
moderately obese individuals, with 5 months of treatment, behavioral treatment combined with moderate dietary restriction e.g. 1000-1500 Kcal/day (self selected foods) results in a mean weight loss of 15-20 pounds Wing (1998). Behavioral weight loss programs are also associated with significant decreases in depression and body image dissatisfaction, together with increases in self-esteem and interpersonal functioning French and Jeffery (1994) problem is that these treatment effects are not maintained overtime.

Swaminathan (2002) investigated that overweight can be prevented excessively by avoiding excessive and frequent eating of food rich in caloriesviz. fried, nuts, sweets etc. and by taking mild and moderate exercise daily. The body weight should be maintained constant at the normal level by adjusting the calorie intake. Fiber diet and green leafy vegetable should be taken.

Srilakshmi (2005) diet therapy is one of the most important strategies for weight loss and weight maintenance. The person should be put in negative energy balance ideally 500-1000 calories less than recommended daily allowance. And ideal reduction of 500gm-1000gm per week is approved once the target is fixed progress should be checked once a month usually 3 kg are lost is the first month largely due to utilization of carbohydrate store and water. Reducing diet should provided adequate amounts of proteins, vitamins and minerals. Reducing excessive food sources of fat and sugars can dramatically reduce calories for many overweight people and general guidelines to accomplish such as change may be as effective as a specific meal plan. Regardless of the method chosen to produce weight loss, the patient must eventually learn
to eat a healthy diet to maintain body weight. This is when behaviour modification techniques and exercise programmes are useful.

5. **Drugs**

Only two anti-obesity medications are currently approved by FDA for long term use. One is *orlistat* (Xenical), which reduces intestinal fat absorption by inhibiting pancreatic lipase the other is sibutramine (Meridia), which acts in the brain to inhibit deactivation of the neurotransmitters norepinephrine, serotonin, and dopamine very similar to some (anti depressants), therefore decreasing appetite. Rimonabant (Acomplia), a third drug, works via a specific blockade of the endocannabinoid system. It has been developed from the knowledge that cannabis smokers often experience hunger, which is often referred to as 'the munchies'. It has been approved in Europe for the treatment of obesity has not yet received approval in the United States or Canada due to safety concerns. Food and Drug Administration (2007).

Weight loss these drugs is modest, over the longer term, average weight loss an orlistat is 2.9 kg (6.4 lb), sibutramine is 4.2 kg (9.3 lb) and rimonabant is 4.7 kg (10.4 lb). Orlistat and rimonabant lead to a reduced incidence of diabetes, and all three drugs have some effect on cholesterol. There is however little data on how these drugs affect the longer-term complications or outcomes of obesity, Rucker *et al.* (2007).

Certain drugs are useful depending on the comorbities present. Metformin (Glucophage) is preferred in overweight diabetics as it may lead to mild weight loss in comparison to sulfonylureas or insulin (UKPDS) Group (1998). The thiazolidinediones, on the other hand, may cause weight gain, but decrease central obesity. Fonseca (2003) diabetics
also achieve modest weight loss with fluoxetine (prozac), orlistat and sibutramine over 12-57 weeks.

Health effects of appetite suppressants may include, headache or dizziness, restlessness tremors, nervousness or anxiety, insomnia, dry mouth, diarrhoea or constipation. There have been cases of raised blood pressure, seizures, strokes and heart-damage but these are rare events. Side effects of lipase-inhibitors such as xenical® include unpredictable and increased bowel movement. However, since all obesity drugs are subject to stringent testing prior to launch, and because after (approval) their health effects are carefully recorded by manufactures and the FDA, side effects are typically well publicised.

6 Surgery

*Bariatric surgery* (Weight loss surgery) is use of surgical interventions in the treatment of obesity. As every operation may have complications, surgery is only recommended for severely obese people (BMI > 40), who have failed to lose weight with dietary modification and pharmacological treatment. Weight loss surgery relies on various principles, the most common approaches area reducing the volume of the stomach, producing an earlier sense of satiation (e.g. by adjustable gastric banding and vertical banded gastro-plasty) and reduce the length of bowel that food will be in contact with, directly reducing absorption (gastric bypass surgery). Band surgery is reversible, while bowel shortening operations are not. Some procedures can be performed laparoscopically. Complications from weight loss surgery are frequent, Encinosa *et al.* (2006).
Surgery for severe obesity is associated with long-term weight loss and decreased overall mortality. One study found a weight loss of between 14% and 25% at 10 years depending on the type of procedure performed and a 29% reducing in all cause mortality when compared to standard weight loss measures Sijostrom et al. (2007). A marked decrease in the risk of diabetes mellitus, cardiovascular disease and cancer has also been found after bariatric surgery Sijostrom and Adams et al. (2007) weight loss is marked in the first few months after surgery and is sustained in the long-term. In one study there was an unexplained increase in death from accidents and suicide but this did not outweigh the benefit in terms of disease prevention. Adams et al. (2007) when the two main techniques are compared gastric bypass procedures are found to lead to 30% more weight loss than banding procedures one year after surgery, Tice et al. (2008).

The effects of liposuction on obesity are less well determined. Some small studies show benefits Giugliano et al. (2004) while others show none. Klein et al. (2004). A treatment involving the placement of an intragastric balloon via gastroscopy has shown promise. One type of balloon lead to a weight loss of 5.7 BMI units over 6 months or 14.7 kg (32.4 lb). Regaining of lost weight is however, common after removal and 4.2% of people were intolerant of the device Imaz et al. (2008).