INTRODUCTION AND REVIEW OF LITERATURE

Obesity is an excess body weight due to fat deposition. Obesity is a global pandemic and a major health concern because of the consequent morbidity and premature mortality. The prevalence of obesity is escalating at an alarming rate to epidemic proportions throughout the developed world. The prevalence data from individual national studies collected by the International Obesity Taskforce (2005) suggest that obesity ranges from 10 to 20 per cent for men and 10 to 25 per cent for women. According to Afridi and Khan (2004) obesity is no longer a concern for developed countries, but is also becoming an increasing problem in many developing countries. Although the prevalence of obesity is higher in the economically developed regions of the world (Haslam and James, 2005) compared to the developing regions. However, with increasing adoption of western lifestyles, the developing countries are also rapidly catching up with the obesity. Because of larger population size, the developing world is actually faced with larger burden of overweight and obesity (Gu et al., 2005 and Reddy et al., 2002).

Ironically, developing countries, which have been saddled with communicable diseases and under-nutrition for generations, are now facing an upsurge of obesity and its adverse health consequences. Afridi and Khan (2004) reported that the traditional societies undergoing the process of economic modernization demonstrate rapid increases in prevalence of obesity. It has been noted that one consequence of nutrition transition in developing countries is decline in under nutrition in association with increase in obesity.
El Rhazi et al., (2011) and Guerrero et al., (2008) found that in low and middle-income countries, malnutrition has become a double-headed monster. It is not uncommon to find under nutrition co-existing with obesity, especially in urban settings. It is estimated that at the beginning of this century, more people will die from complications of over nutrition than of starvation. The pandemic of obesity is so great that it has even spawned a new word ‘globesity’ (Speakman, 2003).

Obesity is an unhealthy excess of body fat and is a major contributor to the global burden of chronic disease and disability. Lee (2009), Victoria et al., (2009) and Bray (2004) reported that the health consequences of obesity are many and varied, ranging from an increased risk of premature death to several non-fatal but serious morbidities such as type 2 diabetes mellitus, nonalcoholic fatty liver disease, hypertension and coronary heart disease that adversely impact the quality of life. Andreyeva et al., (2004), Finkelstein et al., (2003) and Quesenberry et al., (1998) also observed that excess weight reduces the quality of life, raises medical expenditures, places stress on the health care system and results in productivity losses due to disability, illness and premature mortality. Kokiwar (2011) reported that obesity is unique with its own risk factors on one hand and is itself also a risk factor for many other important diseases on the other hand. It has also been reported by the National Institute of Diabetes and Digestive and Kidney Diseases (2004) that approximately 300,000 adult deaths in the United States each year are attributable to unhealthy dietary habits and physical inactivity or sedentary behavior. In a
population-based sample of 5 to 17 year old American children, 70 per cent of obese children had at least one cardiovascular disease risk factor while 39 per cent of obese children had two or more cardiovascular disease risk factors (CDC, 2010). There is also growing evidence connecting unhealthy weight with increased risk of developing cancer. The studies conducted by the American Institute for Cancer Research (2008) indicate that more than 100,000 cancers in the US each year are linked to excess body fat.

ETIOLOGY OF OBESITY

Obesity is a common but complex multifactorial disorder which develops from the interactions of multiple genes and environment characterized by long term energy imbalance due to excessive calorie consumption, insufficient energy output (Bray, 2004 and NHLBI, 1998) due to inadequate physical activity (WHO, 2000) or both (Afridi and Khan, 2004). Obesity is the first of the diseases of civilization to appear (Trowell and Burkitt’s, 1981). Obesity has a wide phenotypic variability ranging from mildly overweight to morbidly obese. Although the trend of increasing body girth is very much driven by the obesogenic environment, it is facilitated by the individual’s genetic susceptibility to excessive weight gain (Lake and Townshend, 2006). Marti et al., (2004); Bray and Bouchard (2004) and Poulsen et al., (2001) states that both genes and everyday life environmental factors such as cultural and social mediated food intake and reduced domestic and living work activities are involved in the obesity pandemic.
The size and shape of the human body is greatly influenced by heredity. Being fat is caused by a combination of hereditary traits and the body's natural response to the environment. Many studies have shown a consistent correlation between heredity and fat. Although rapid globalization of westernized way of life is also responsible for the large rise in the number of obesity cases, Obesity results from a complex interplay of environmental and genetic factors, as has been showed by numerous epidemiological studies carried out on twins, adopted children and nuclear families in several populations (Kopelman, 2000; Bouchard, 1991 and Stunkard et al., 1990). Loos and Bouchard (2003) reported that there is a synergistic relationship between genes and environment, in the presence of genetic predisposition to obesity the severity of disease is largely determined by lifestyle and environmental factors. Those with a high genetic predisposition for obesity will gain the most weight, whereas those resistant to obesity will gain little if any weight. Brook et al., (1975) also stated that obesity has a strong genetic component and tends to run in families.

The commonly observed coexistence of several obese members within a family also suggests the involvement of genetic factors in obesity (Bray, 2004). Cummings and Schwartz (2003); Rice et al., (1999); Bouchard et al., (1998) and Stunkard et al. (1986a,b) have reported that the risk of obesity increases when one has relatives who are obese and this risk is about two to three times higher and it further increases with the severity of obesity (Nirmala et al., 2008). The risk of excessive weight gain in children of some families with obese parents is increased two- to three-fold for moderate obesity and up to
eight times for severe obesity (Bouchard, 2001). The heritability of body weight is high and genetic variation plays a distinct role in determining the inter individual differences in susceptibility or resistance to obesogenic environment (Ramachandrappa and Farooqi, 2011 and Freeman et al., 2002). In most cases, genes involved in weight gain do not directly cause obesity but rather they increase the susceptibility to fat gain in subjects exposed to an environment characterized by an abundance of food and limited physical activity. Hill and Melanson (1999) and Hill and Peters (1998) argue that the culprit is an environment which promotes behaviors that cause obesity.

A growing consensus is that environmental factors have played a pivotal role in influencing people’s lifestyles and fueling the obesity epidemic in the United States and worldwide (WHO, 2000; Egger and Swinburn, 1997). Among environmental influences there are two major factors involved. The first is that among all populations children are targets of food advertisements which are potential health risks, as the consumption of these products become a habit and then a lifestyle. The excessive consumption of food products lead to eating more food than required in addition to acquiring an increasingly sedentary lifestyle (Klunder et al., 2011). The second is the known social networks (family, friends, neighbors and siblings) that have great influence on the individual’s food consumption. Thus, when two persons are perceived as friends and one is obese, the risk of other friend becoming obese is 17 per cent. Among siblings, if one is obese, the risk that the other sibling to become obese is 40 per cent (Christakis and Fowler, 2007). Today’s these social networks
family, friends, life partners and associates) have been recognized as an important factor affecting the incidence of obesity.

Parents being an important part of these social networks have a significant influence on children. Parents and children share the same socioecological environment because the home is the site where parents transmit habits and customs to their children. Parents provide food environments for their children's early experiences with food and eating. These family eating environments include parents' own eating behaviors and child-feeding practices. Results of the limited research on behavioral mediators of familial patterns of overweight indicate that parents' own eating behaviors and their parenting practices influence the development of children's eating behaviors, mediating familial patterns of overweight (Birch and Davison, 2001). A study by Johanssen et al., (2006) reported that mothers have a strong influence on the eating habits and thus weight of their children while Klunder et al., (2011) states that eating habits are passed on from fathers to children more strongly.

A positive linear relationship has also been observed between BMI and alcohol consumption (Tolstrup et al., 2005 and Breslow and Smothers, 2005). Alcohol is a significant source of calories and drinking may stimulate eating, particularly in social settings resulting in alcohol related overeating which could lead to weight gain. Experimental evidence from several metabolic studies showed that there is suppression of lipid oxidation by alcohol and thus the enhancement of a positive fat balance causing the deposition of unoxidized
fat preferentially in the abdominal area (Suter and Tremblay, 2005). Panagiotakos et al., (2004) observed that obese and overweight consume higher quantities of alcoholic beverages and are devoted to an unhealthier diet compared with those of normal weight. Smoking may also adversely alter the body fat distribution putting the smokers at a higher risk of cardiovascular diseases, cancer, diabetes and other metabolic consequences. Epidemiologic and clinical studies consistently show that smokers tend to weigh less than non-smokers and weight gain follows smoking cessation (Filozof et al., 2004 and Flegal et al., 1995). Nicotine has both thermogenic and appetite suppressant effects and its effects on appetite are enhanced by caffeine (Jessen et al., 2005). Centers for disease control and prevention scientists estimated that between 1978 and 1990, smoking cessation was responsible for about one-quarter of the increase in prevalence in overweight in men and for about one-sixth of the increase in women in US (Flegal et al., 1995). Mozaffarian et al., (2011) also reported that smoking cessation results in weight gain initially but in little weight gain thereafter.

PREVALENCE OF OBESITY

Although still considered more of a cosmetic problem by the general public, obesity has reached epidemic proportions worldwide. The International Obesity Taskforce (2005) estimated a total of 1.1 billion overweight including 320 million obese adults worldwide. It has been reported by Speakman (2003) that nine people in USA and one person in UK die every 15 minutes as a direct consequence of obesity related illnesses. Wang and Beydoun (2007) reported
that among US adults, obesity prevalence increased from 13 per cent to 32 per cent between 1960s and 2004. It has been found that 66 per cent of adults are overweight or obese, 16 per cent of children and adolescent are overweight and 34 per cent are at risk of overweight. US survey demonstrates that 65 per cent of Americans are overweight and 30.4 per cent are obese (Baron, 2006). It is further projected that by 2015, 75 per cent of adults will be overweight and 41 per cent will be obese (Wang and Beydoun, 2007). According to WHO, globally in 2005 approximately 1.6 million adults (age 15+) were overweight and at least 400 million were obese. WHO further projects that in 2015, approximately 2.3 billion adults will be overweight and more than 700 million will be obese.

Obesity is emerging as important health problem even in India. There is paucity of nationwide data on obesity in India however studies from different states of India suggest that the prevalence ranged from 10-50 per cent (Mohan and Deepa 2006). WHO (2006) had reported that in India there are 2.40 per cent obese, 13.50 per cent pre-obese males and 6.10 per cent obese and 17.40 per cent pre-obese females. Joshi and Joshi (2002) also reported that obesity, as an emerging problem is a major chronic disorder affecting 20-40 per cent adults in India. Dhurandhar and Kulkarni (1992) found prevalence of obesity to be 9.4 per cent among men and 19.3 per cent among women in urban community of South Delhi. Zarger et al., (2000) observed that among Kashmiri population the overall prevalence of obesity has been found to be 15.01 percent, the prevalence of obesity among males was 7.0 per cent and in females
23.69 per cent. Thus, obesity is a growing problem, more common in females and urban population. Kokiwar (2011), Pi-Sunyer (2002) and Pi-Sunyer (1994) also reported that the incidence of obesity has been found to be higher among females than males. Zhang et al., (2008) also found that the prevalence of overweight and obesity is significantly higher among Chinese women as compared to men which according to Bose (1995) may be due to higher body fat among females and the hormonal differences.

There had been a worldwide increase in obesity among people of all ages. In more affluent countries, obesity is common not only in the middle-aged, but is also becoming increasingly prevalent among younger adults and children (Bindah and Othman, 2011). Pi-Sunyer (2002) states that obesity can occur at any age, older individuals are more likely to become obese. It has been found that obesity reaches its peak at around 55-64 years of age and decreases afterwards. Mohsen and Warsy (2002) also found significant increase in prevalence of obesity and overweight with age in both males and females of Saudi population. In all age groups, obesity was significantly more in females compared to males. Overweight was more prevalent in females 20-29 years of age as compared to males but the 30-49 years old males had a higher prevalence of overweight. Males and females aged more than 50 years had almost an equal prevalence of overweight. The prevalence has been found highest for age group 31-50 years among adults of Mumbai (Dhurandhar and Kulkarni, 1992). National Foundation of India (Joshi and Joshi 2002) reported higher incidence of obesity among people above 40 years. Zhang et al., (2008)
found that with increase in age the prevalence of overweight decreased in Chinese men however the prevalence in women gradually decreased after 45 years of age. Thus, the prevalence of obesity showed no obvious age differences among Chinese population.

**DETERMINANTS OF OBESITY**

Body mass index (BMI) defined as weight (kg) divided by height (m$^2$), is the most commonly used measure to determine obesity (WHO, 2000). BMI is a practical indicator of severity of obesity. In recent years, the body mass index has also become the medical standard used to measure overweight and obesity. BMI can be considered to provide the most useful population level measure of obesity. Most health organizations and published information on overweight and its associated risk factors use BMI to measure and define obesity. It may be used to estimate the prevalence of obesity within a population and the risk associated with it in cross-sectional comparisons. BMI does not directly measure the per cent of body fat but it provides a more accurate measure of overweight and obesity than relying on weight alone. BMI is a direct calculation that describes relative weight for height, is not gender specific (Matz, 1993) and is significantly correlated with total body fat content (NIH, 1998; Bray and Popkin, 1998). According to WHO (1997) BMI should be used to classify overweight and obesity compared to weight only.

A graded classification of overweight and obesity using BMI values provide valuable information about increasing body fatness although it does not provide any clue for the distribution of fat in the various parts of the body.
(Sugerman et al., 1997). It allows meaningful comparisons of weight status within and between populations and identification of individuals and groups at risk of morbidity and mortality (Bose, 1995). For meaningful comparison between or within the populations WHO (1998) advised the use of BMI cut-off points. For adults WHO (1998) recommended BMI 25.0kg/m² and 30.0kg/m² as the cut off points of overweight and obesity respectively. These values are age independent and the same for both sexes. Health care providers are concerned not only with how much fat a person has but also where the fat is located on the body. A more important aspect of obesity is the regional distribution of excess body fat. Morbidity and mortality due to obesity vary with the highest risk linked to excessive abdominal fat i.e. central obesity.

Waist circumference (WC) and waist hip ratio (WHR) are used for determination of central obesity (Bhadra et al., 2001). Studies indicate that central obesity, characterized by increased waist circumference is associated with an increased risk for a number of diseases including cardiovascular diseases, non insulin dependent diabetes mellitus, high blood pressure, gall bladder disease, stroke and certain cancers in patients with a BMI in the range between 25 and 34.9 and is associated with overall mortality, independent of BMI (Oppert et al., 2002). A study by Gopalan (1998) on Indian population revealed that almost 20 per cent of adults who were not overweight or obese still had central obesity, putting them at a greater risk of developing the associated diseases. According to American Heart Association (2002) boys
with chubby bellies are more likely to have high blood pressure than their slimmer counter parts.

Afridi and Khan (2004) reported that the waist circumference correlates with the amount of fat in the abdomen and thus is an indicator of severity of central obesity. The waist hip ratio also has been used in a number of epidemiologic studies to show increased risk for diabetes, coronary artery disease and hypertension (Albu et al., 1997). It has been reported by WHO (1998) that Waist circumference may be the preferred measure of abdominal obesity compared to waist hip ratio. Several investigators (Schneider et al., 2007; Lean et al., 1995; Pouliot et al., 1994 and Despres et al., 1989) have also reported that waist circumference has been found to be a better marker of abdominal fat content than BMI and waist hip ratio. Kurpad et al., (2003) states that waist circumference correlates better with body mass index than waist-to-hip ratio and the prevalence of central obesity has been found higher using waist circumference as determinant than that with waist-to-hip ratio. Neovius et al., (2005) found BMI and WC to be better diagnostic tests for body fat than WHR.

GENETICS OF OBESITY

Swarbrick and Vaisse (2003) reported that complex diseases such as obesity are likely to be based on a limited number of predisposing alleles, each conferring a small increase in the risk to the individual. Heterogeneity in complex phenotypes implies that the genetic predisposition may also result from any one of several rare variants in a number of genes. Loktionov (2003)
found that the role of a genetic predisposition in obesity has long been assumed to affect both energy intake and energy expenditure. The mutations in human genes coding for leptin (LEP) or ob gene, leptin receptor (LEPR), proopiomelanocortin (POMC), proconvertase (PCI) and melanocortin-4 receptor (MC4R) has solely implicated energy intake; however, Mendelian syndromes with obesity as a clinical feature (eg. Prader–Willi syndrome) have also revealed reductions in energy expenditure as contributing to obesity (Vaisee et al., 1998; Krude et al., 1998; Clement et al., 1998 and Montague et al., 1997). All the proteins coded by these genes are part of pathway regulating food intake. The leptin or ob gene codes for a hormone which is secreted by adipocytes in proportion to their fat content. In the hypothalamus, leptin binds the long form of its receptor and enhances the expression of POMC gene. The enzyme PCI cleaves POMC to yield ACTH and α-MSH which reduces food intake when it binds to brain-specific MC4R (Froguell and Boutin, 2001). Vaisse et al., (2000) reported that the MC4R gene is the most prevalent obesity gene being involved in 1 per cent to 4 per cent of very obese individuals. 176 human obesity cases due to single-gene mutations in 11 different genes have been reported, 50 loci related to Mendelian syndromes relevant to human obesity have been mapped to a genomic region, and causal genes or strong candidates have been identified for most of these syndromes (Rankinen et al., 2006). A population based study suggested that 35 per cent of the adjusted variation in BMI was accounted for by a single recessive locus while polygenic loci accounted for 42 per cent of variation (American Heart
Association, 1998). Genome-wide scans have been able to identify specific chromosomal regions such as those on chromosomes 2, 3, 5, 6, 7, 10, 11, 17 and 20 (Loos and Bouchard, 2003 and Clement and Ferre, 2003).

Genes may determine afferent and efferent signals as well as central mechanisms involved in body weight regulation (Loktionov, 2003). Thus, the transferable genetic information involved in short and long-term stable body weight regulation and diet composition maintenance (Palou et al., 2000 and Macho et al., 2000) is acting via different peptides and monoamines involved in the regulation of the appetite, variations in energy and nutrient utilization, resting metabolic rate or response to physical activity and individual differences in adipocyte metabolism. Loktionov (2003) and Palou et al., (2003) also stated that the possible mechanisms through which the genetic susceptibility could be acting include reduced rates of basal metabolism and macronutrients oxidation, alterations of adipogenesis and quantitative and qualitative deviations of food intake. Barsh et al., (2000) found that factors such as the hormonal profile, energy exercise efficiency and thermogenesis are specifically involved in the genetic processes affecting the energy balance equation. Thus, the genes predisposing to obesity also have an impact on the dietary intake as well as physical activity performance (Heitmann et al., 1997 and Heitmann et al., 1995).

In all societies and sub populations there are both obese and non-obese subjects. Thus, these differences are primarily a consequence of genetic factors (Speakman, 2004). Obesity is one of the most heritable traits in humans.
Studies suggest that 25 per cent to 70 per cent of obesity can be explained by genetics (Wu et al., 2003; Maffeis, 2000; Freidman, 2000 and Cardon et al., 1994). Barsh et al., (2000) suggested an estimated heritability of 50 per cent to 90 per cent for obesity.

Parental obesity more than doubles the risk of obesity in adulthood for both obese and non-obese children, especially those less than 10 years of age. Lawlor et al., (2008) and Whitaker et al., (1997) reported that obesity in one or both parents probably influences the risk of obesity in their offspring because of shared genes or environmental factors within the families. Sanderson and Faith (2010) found that the relationship between parental and children obesity follows a dose response such that children with one parent obese are more likely to be obese than are children with both parents non-obese. Moreover, children with both parents obese are even more likely to become obese than are children with one or no parent obese. Kumar et al., (2010) observed that children with parental obesity showed 25.2 times more chances of developing obesity. The data from National Health and Nutrition Survey reported that the children of overweight mothers are 1.9 times more likely to be obese and 3.4 times more likely to be obese if the mothers are obese (Danielzik et al., 2002). A recent study by Freeman et al., (2012) found that having an overweight or obese father significantly increased the odds of children obesity however the reverse i.e. having overweight or obese mother is not associated with increased risk of obesity in children. Klunder et al., (2011) also reported that if father is obese the risk of children to become obese increases 12.1 times while if mother
is obese the risk for obesity among children is increased by 6.5 times indicating a greater influence of father over mother for the child to be obese.

Whitaker et al., (2010) observed that obesity among children is considerably higher i.e. 35.30 per cent when both of their parents are obese while it has been found to be rare i.e. 2.3 per cent among children when both parents are non-obese. It has also been reported by Xi et al., (2009) that when both parents are non-obese only 3.29 per cent of children are obese and 27.01 per cent children are obese when both the parents are obese. Jacobson et al., (2007) also found that obesity prevalence among children is as high as 20.1 per cent when both the parents are obese while it is 8.20 per cent when one of the parents is obese and only 1.4 per cent children are obese when both of their parents are non-obese. Studies by Roberts et al., (1988) and Stunkard et al., (1986a) showed that where both parents are obese, 80 per cent of their children, even if not raised by their genetic parents, are also obese. 40 per cent are obese when one of the parents is obese and only 9 per cent are obese when both parents are lean.

Gupta and Kapoor (2011) reported that there is familial aggregation for obesity as well as gender differences in familial correlations of obesity in children with daughters being more likely than sons to be affected by parental obesity. Whitaker et al., (2010) and Davis et al., (2008) also reported that there is a strong association between parental obesity and children obesity and found this association to be significantly stronger between mother and children. It has been observed by Kumar et al., (2010) that maternal obesity passes mainly to
sons and paternal obesity to daughters while Xi et al., (2009) reported that maternal obesity has a greater influence on daughters than on sons and the paternal obesity showed a similar influence on sons.

The first evidence about the important role of genetics in obesity came from the National Heart Lung and Blood Institute (NHLBI) Twin study in 1977, which indicated the possibility that the observed familial aggregation for obesity was due to genetic factors rather than environment (Feinleib et al., 1977). The genetic contribution to body weight has been further established through family studies, investigating parent-offspring relationships and the study of twins as well as adopted children (Maes et al., 1997 and Stunkard et al., 1990). Studies of twins suggest inheritance explains 25 per cent to 40 per cent of inter-individual differences in obesity (Stunkard et al., 1986a). Recent studies of individuals with a wide range of BMIs, together with information obtained on their parents, siblings and spouses, suggest that about 25 to 40 percent of the individual differences in body mass or body fat may depend on genetic factors (Bouchard et al., 1998; Vogler et al., 1995 and Tambs et al., 1991). Pietilainen et al., (1999) also estimated that 80 per cent of the inter individual variation in BMI was due to genetic effects, which was supported by a similar study from UK (Koeppen – Schomerus et al., 2001).

Grilo and Pogue-Geile (1991) and Price (1987) had observed that BMI is correlated among family members. In a study Price et al., (2000) reported the resemblance of BMI among families of obese women. Barsh et al., (2000) reported heritability estimates of 40-70 per cent for BMI. It has been reported
by several investigators (Adeyemo et al., 2003; McQueen et al., 2003; Platte et al., 2003; Pietilainen et al., 1999 and Allison et al., 1996a) that heritability estimates for body mass index ranged from 16 per cent to 85 per cent. Yang et al., (2007) also reported that heritability estimates for obesity related phenotypes varied from 6 per cent to 85 per cent among various populations. In a study of African-American adolescent twins Allison et al., (1994) reported that 90 percent of the variance in BMI was caused by genetic effects.

Twin studies provide a unique method for disentangling nature and nurture by taking advantage of the fact that monozygotic twins share all of their genes, whereas dizygotic twins on average share half of their segregating genes (Hooper et al., 2005). If genetic influence is important, monozygotic twins must be more similar than dizygotic twins. Twin studies can also estimate the extent to which the family environment makes family members more similar than would be expected from their genetic relatedness. A 1997 review of published adult twin and adoption studies also found that variation in BMI was largely due to heritable genetic differences (Maes et al., 1997). Studies published since 1997 have reached the same conclusion, with heritability estimates in adults ranging from 55 per cent to 85 per cent (Malis et al., 2005; Romeis et al., 2004; Schousboe et al., 2003 and Bulik et al., 2003). Twin studies also show that most of the nongenetic effect comes from environmental factors that are unique to each person (nonshared-environment effects) and not from the shared family context which has been confirmed by results from adoption studies (Grilo and Pogue-Geile, 1991 and Stunkard et al., 1990).
The studies conducted with monozygotic and dizygotic twins (Maes et al., 1997) or monozygotic twins reared apart (Stunkard et al., 1990) yielded the highest heritability levels with values clustering around 70 per cent indicating a higher genetic contribution to the BMI. Bouchard et al., (1998) reported even higher heritability estimates for BMI ranging from 50 per cent to 90 per cent among identical twins. However, adoption studies have generated the lowest heritability estimates, of the order of 30 per cent or less (Vogler et al., 1995 and Sorensen et al., 1992). Rice et al., (1999) reported that family studies have found levels of heritability intermediate between twin and adoption studies i.e. varying from 30 per cent to 70 per cent. Perusse (2000) also reported that twin studies have largely revealed that the heritability of BMI is 70 per cent to 80 per cent while family studies have most estimated the contribution of genetic factors at 25 per cent to 50 per cent.

Another study found that twins, regardless of the fact that they are reared apart or in the same home environment, are about 70 per cent likely to weigh the same (Segal and Allison, 2002 and Stunkard et al., 1990). Twin studies have demonstrated genetic influences on resting metabolic rate, feeding behavior, changes in energy expenditures in response to overfeeding, lipoprotein lipase activity and basal rate of lipolysis (American Heart Association, 1998; Bouchard and Tremblay, 1990; Rosenbaum and Leibel, 1988; Bogardus et al., 1986 and Brook 1977). Family clustering of obesity exists, with a relative risk among siblings of 3–7 (Allison et al., 1996a), and the concordance of body mass index is much higher between monozygotic
(74 per cent) than dizygotic twins (32 per cent), despite equally shared environments (Barsh et al., 2000 and Stunkard et al., 1990). The body mass index of adopted children is linearly related to that of both of their biological parents, even when there is no direct contact with them, and is unrelated to that of either adoptive parent, even though they are providing the daily menu (Stunkard et al., 1986b). A study of thousands of twins estimated that 77 per cent of the variation in their BMI and waist circumference was due to genetic variation. The rest of the variation is attributed to environmental differences (Wardle et al., 2008). Maes et al. (1997) also found that on average obesity is 67 per cent genetic and 33 per cent environmental.

Thus, the role of genetic factors in human obesity is complex, being determined by interaction of several genes each of which may work in combination with environmental factors such as nutrients, physical activity and smoking (Froguell and Boutin, 2001 and Tambs et al., 1991). The relative contribution of environment and genetic susceptibility towards the pathogenesis of obesity varied between different obese individuals, even within the same family and may contribute to this phenotypic variability. The environmental factors may be dominant contributing factor in the development of late onset obesity in an adult while genetic factors may exert a greater influence in a young child who developed early obesity in the obesogenic environment. Study of twins provides the clearest evidence for genes and environment both having a role. Thus, environmental factors also play a critical role in the development of obesity by unmasking genetic susceptibilities.
ENVIRONMENTAL FACTORS

Obesity can be described as the ‘New World Syndrome” as it has emerged as the most prevalent serious public health problem. Obesity is a polygenic disease whose development can be modulated by various genes and by environmental influences (Froguel and Boutin, 2001). Since most often genetic factors account only a third of the variance in body weight, environmental influences must therefore account for the balance. The variation in prevalence of obesity epidemic in various communities of the world may be attributed to environmental factors such as age, sex, diet patterns, lifestyle (Kopelman, 2000; Epstein and Higgins, 1992; Gurney and Gorstein, 1988), socioeconomic status and education level (Pi-Sunyer, 2002). Friedman (2003) stated that all environmental factors act in concert with individual genetic susceptibilities for obesity.

Hill and Peters (1998) states that the modern obesogenic environment of industrialized countries developed over past few decades in our bid to reduce work and improve efficiency and quality of life, is characterized by easy access to highly palatable, highly caloric food, sedentary vocations, and leisure-time activities dominated by television, videogames and computers which led to a reduction of energy expenditure and increased energy intake. Consequently the proportion of overweight people has risen steadily over the years (Lee, 2009). Obesogenic environment predisposes to diabetes, cardiovascular disease, osteoarthritis, sleep apnea, cancer, and other ailments (Zheng, 2011; Berrington and Gonzalez, 2010; Flegal et al., 2007; Kopelman, 2000; and Lean et al.,
Type 2 diabetes and cardiovascular diseases, once thought to occur only in adults, are now commonly seen in obese children (Tirosh, 2011 and Bhargava, 2004).

The global epidemic of obesity has resulted from societal factors that promote sedentary lifestyles and the consumption of high fat energy dense diets (WHO, 2000). Reddy (1998) also reported that changes in dietary patterns, physical activity levels and lifestyles associated with diet and urbanization are related to increasing incidence of obesity in India. According to Bray and Champagne (2005) large portion sizes, high fat intakes, easy access to calorically sweetened beverages, and lack of any need to be physically active all play a role in the toxic environment that leads to obesity.

Industrialization and modernization is rightly blamed for increasing obesity prevalence all over the world, as it encouraged sedentary lifestyles (Bell et al., 2005 and French et al., 2001a) resulting in the net deposition of calories as fat. Of the multiple causal factors associated with the rise in obesity in developing countries, perhaps the two most important are urbanization and globalization of food production and marketing. Marketing campaigns and price incentives have an important impact on food purchasing pattern in developing countries where as much as 60 per cent of household income is spent on food. One experimental study of young children offered them identical pairs of foods and beverages, the only difference being that some of the foods were in McDonald’s packaging. Robinson et al., (2007) found that children were significantly more likely to choose items perceived to be from
McDonald’s indicating that there is significant effect of exposure to fast food advertising on overweight and obese children.

**Socioeconomic Status**

There is bidirectional causal relation between socioeconomic status and obesity because obesity may adversely affect one’s opportunities for education, occupation and marriage (Gortmaker *et al.*, 1993). The emergence of obesity in developing countries initially affected primarily the higher socioeconomic strata of the population but more recent trend show a shift in prevalence from higher to the lower socioeconomic level (Monteiro *et al.*, 2004). Studies by several investigators (Sarlio-Lahteenkorva *et al.*, 2004; Zhang and Wang, 2004; Wardle *et al.*, 2002; Galobardes *et al.*, 2000; Sundquist and Johansson, 1998; Jeffery *et al.*, 1991 and Sobal and Stunkard, 1989) reported a strong inverse relationship between socioeconomic status and obesity among women. In low-income countries, obesity is more common in middle-aged women, people of higher socioeconomic status and those living in urban communities. In more affluent countries it tends to be associated with lower socioeconomic status, especially in women, and the urban–rural differences are diminished or even reversed (Bindah and Othman, 2011).

Prevalence of obesity declines with income and education. It has been reported by Mokdad *et al.*, (2001) that 26 per cent of high school drop outs were obese in 2000 in US versus 22 per cent of high school completers and 15 per cent of college completers. On the basis of NHANES 1999-2000 data overall, less educated persons in United States (those with less than a high
school education) have a higher prevalence of obesity. The prevalence of obesity has been found 27.4 per cent, 23.2 per cent, 21.0 per cent and 15.7 per cent for individuals with less than high school education, a high school degree, some college and college or above respectively (Mokdad et al., 2003). Zhang and Wang (2004) also found a decreasing inverse association between obesity and education. Zhang et al., (2008) found that people of china with higher levels of education had a risk of gaining weight as the well educated people usually have a good income and can afford foods which are supposed to lead to obesity. However, Panagiotakos et al., 2004 found that the obese are less educated than those of normal weight.

Flegal et al., (1988) found decreasing inverse relationship between obesity and income among women and a stable positive association among men. Although obesity is frequently associated with poverty but the obesity has increased at all levels of income. Moreover, it is typically not the poor who have experienced the largest gains the absolute increase in obesity among US women is 27.00 per cent for those at middle incomes but only 14.5 per cent for the poor. However among the US men, the increase in obesity is 21 per cent for those at the highest level of income but only 5.4 per cent for the poor (Chang and Lauderdale, 2005). Obesity is associated with a clear income disadvantage particularly among women with higher socioeconomic status. However, excess body weight is not associated with income disadvantages in men (Sarlio-Lahteenkorva et al., 2004).
Dietary Habits

Dietary habits have a significant positive association with obesity (Bindah and Othman, 2011). The development of overweight and obesity has been thought to be a consequence of positive energy balance, where energy consumption from food and beverages is more than the energy spend through basal metabolism and physical activity resulting in an accumulation of energy stored as fat in the adipose tissue (Reddy et al., 2002). In most of the developed and many of developing countries there is an overall abundance of palatable and caloric-dense food. Additionally, the abundance of food in supermarket, the availability of food sold at fast food restaurants and vending machines and the large portions of food served outside the home, promote high caloric consumption. Many of our socio-cultural traditions, especially at holidays or special occasions, promote overeating and preferential consumption of high caloric foods. For many people, even when caloric intake is not above the recommended level, the number of calories expended in physical activity is insufficient to offset consumption. All this lead a person to be obese (French et al., 2001b and NIH, 1996).

Diets that have been hypothesized as influencing energy intake and therefore weight status include fat intake (Liu et al., 2003 and Jequier, 2001), sugar-sweetened beverages (Ello-Martin et al., 2005; Berkey et al., 2004 and Ludwig et al., 2001), sweets (Mozaffarian et al., 2011; Buijsse et al., 2009; Wolff and Dansinger, 2008 and Van and Seidell, 2007), non-vegetarian foods (Mozaffarian et al., 2011), snack foods (Ledikwe et al., 2007; Field et al., 2004...
and Francis et al., 2003) and fast foods (Micha and Mozaffarian, 2009; French et al., 2001b and McNutt et al., 1997). Ello-Martin et al., (2007), Rolls et al., (2005) and Bray et al., (2004) also reported that several cross-sectional studies have demonstrated significantly positive correlations between fat intake and various measures of obesity. Galson (2008) identifies poor attention to nutrition, increased access to fast food, high fat content processed snacks, and high calorie sugary beverages as major contributors to the growing obesity crisis. However, Hoerr (2009) reported that the fast foods and eating away from home is not necessarily problematic for everyone, but rather for those who have a stronger genetic predisposition to develop obesity.

The shift from traditional diets to high fat and high energy diets has contributed to the changes seen in people’s nutritional status (Popkin et al., 2001). Epidemiologic data indicates that chronic intake of fatty diets and fructose and frequent consumption of fast foods increase the risk of obesity (Malik et al., 2010; Bray, 2010; Kestens and Daniel, 2010; Sacks, 2009; Gross et al., 2004 and Wing, 1995). This is evident in developing countries where indigenous diets rich in vegetables, fruits, and fish have been replaced by cheaper and more energy-dense Western diets (Mehio et al., 2010 and Mavoa and McCabe, 2008). Higher intakes of energy and carbohydrates by both genders as well as high intakes of protein and fat by men are related to greater incidence of being overweight (Hu et al., 2002). Increased consumption of total energy, soft drinks, snacks foods and more frequent eating at fast food or other
restaurants and inadequate consumption of vegetables and fruits contributes to the increased risk of obesity (Briefel and Johnson, 2004). Regarding fast food availability, although some studies showed associations with obesity, Burdette and Whitaker (2004) found no association between being overweight and proximity to fast food restaurants in over 7000 children in US. Studies by Berkey et al., (2004), Field et al., (2004), Newby et al., (2004) and Ludwig et al., (2001) reported no association between soft drink consumption and BMI indicating that there is no independent effect of calories from soft drinks on body weight other than its pleasant taste possibly leading to the potential increase in total caloric intake as would any food. However, Malik et al., (2006) reported strong association between soft drinks consumption and weight gain. Bes-Rastrollo et al., (2006) found that sweetened soft drinks and fast food intake and to lesser extent consumption of sweetened fruit drinks is associated with a yearly weigh gain of 0.4 kg. They reported that the energy obtained from soft drinks does not fully displace that consumed from solid foods and may encourage an increase in the consumption of other foods. They also pointed out that consumption of soft drinks and fast foods exert adverse effects on satiety, glucose metabolism and the rate of ingestion and gastric emptying. Pereira et al., (2005) observed that consumption of soft drinks and fast foods displace the consumption of dairy products which could increase the weight. Rosenheck (2008) also reported positive link between obesity and fast food consumption. Lakdawalla and Thomas (2002) found that 40 per cent of the increase in
obesity is attributable to increased energy consumption. Bleich et al., (2007) and Cutler et al., (2003) also reported that increased calories intake is the main contributor to obesity.

A study by David et al., (2008) reported that lower body weights are associated with a lower percentage of calorie intakes from protein and greater occupational physical activity levels only among men with higher education level among women. Meal frequency has also been found to be associated with obesity. Studies by Toschke et al., (2005), Kant et al., (1995) and Edelstein et al., (1992) have demonstrated an inverse relationship between meal frequency and prevalence of overweight and obesity in both children and adults. Vik et al., (2010) found that number of meals eaten is associated with being overweight and observed that eating four meals per day is significantly negatively related to being overweight or obese suggesting that lesser number of meals consumed results in increased body weight. Franko et al., (2008) also reported that increased meal frequency is related to decreased likelihood of being obese.

**Lifestyle**

Lifestyle influences promoting excessive caloric intake and sedentary patterns are known to induce a positive energy balance leading to weight gain (Swinburn and Egger, 2004). A strong link exists between physical inactivity and weight gain. Multiple cohort and cross-sectional studies have shown an association between obesity and physical inactivity (Williamson et al., 1993). There is the possibility that this relationship is bidirectional, with obesity
discouraging physical activity and inactivity promoting weight gain (DiPietro, 1995). A lack of physical activity imparts an increased risk for both CVD and NIDDM. The drop in physical activity among children has been linked to their sedentary behaviors, including the increased amounts of time spent engaged with media sources such as television, video games, and computers. It has been found that time spent watching TV, videos, DVDs, and movies averaged slightly over 3 hours per day among children aged 8–18 years (CDC, 2010). A positive association between time spent viewing television and increased prevalence of obesity in children has been reported by CDC (2010). This connection appears rooted in the fact that watching television not only takes away from time children could be spending being physically active, but also places children in the home where snacks and food advertisements are more readily available.

Hu et al., (2003), Robinson (1999) and Williamson et al., (1993) reported that sedentary lifestyle is a major contributor to weight gain. Panagiotakos et al., (2004) also found that overweight and obese are more frequently sedentary. Data from the National Health and Nutrition Examination Survey (NHANES) show a close relationship between low levels of physical activity and weight gain in both men and women (Smith et al., 1994). Zhang et al., (2008) and Hu et al., (2002) reported that lower levels of physical activity are related to greater incidence of being overweight and obese. Martinez-Gonzalez et al., (1999) found a strong association of obesity and higher body weight with a sedentary lifestyle and lack of physical activities in
adult population. Similarly Vik et al., (2010) and Hernandez et al., (1999) also investigated the relation of obesity with physical activity and reported prevalence of obesity to be positively related with physical inactivity and television viewing/computer usage.

A statistically significant relationship exists between TV viewing and body weight among US adults (Hu et al., 2001 and Tucker and Bagwell, 1991). Prolonged television watching is highly predictive of obesity risk in adults and children (Hu et al., 2003). Mozaffarian et al., (2011) observed that duration of television viewing influence the weight gain in adults as it is mediated by changes in diet, physical activity and sleep duration. Epstein et al., (2008) reported that reducing television viewing and computer use may have an important role in preventing obesity and in lowering BMI which may be related more to changes in energy intake than to changes in physical activity. Robinson (2001) also stated that reducing television viewing can prevent obesity among children as it reduces the meals consumed in front of television. Several investigators including Manios et al., (2009), Dubois et al., (2008), Temple et al., (2007), Utter et al., (2006), Vereecken et al., (2006), Coon et al., (2001) and Escobar-Chaves et al., (2001) found that television watching encourage snacking during viewing and also influences the food choices both during viewing and at other times. Deckelbaum and Williams (2001) also reported that physical inactivity, high frequency of television viewing or computer usage, over consumption of high calorie foods, snacking while watching television or doing homework and over exposure to advertisements of high calorie foods are
modifiable risk factors for obesity among children. According to Marshall et al., (2004) the global changes in diet and physical inactivity have been fueled by changes in agricultural practices, food processing, marketing and distribution, transportation, and other aspects of urban planning and TV based physical inactivity may be unfairly implicated in recent epidemiologic trends of overweight and obesity among youth.

The hours of sleep per night are also inversely related to BMI and Obesity (Gangwisch et al., 2005 and Von Kries et al., 2002). Short sleep duration is associated with obesity and weight gain among children and adults. Sleep deprivation results in increased hunger and appetite (Spiegel et al., 2004a) as well as endocrine changes including decreased leptin and thyroid stimulating hormone secretion, increased ghrelin levels and decreased glucose tolerance leading to increased risk of obesity (Spiegel et al., 2004b; Taheri et al., 2004 and Spiegel et al., 1999). Lopez- Garcia et al., 2008 reported that among Spanish adult population sleeping less than 5 hours is associated with obesity. In western societies, obesity has raised in parallel with decrease in hours of sleep (Patel and Redline, 2004) because of biological factors such as sleep and other medical disorders and because of behavioral and social factors such as child care, shift work and round the clock entertainment. Several cross sectional studies have reported that obesity is more common among those who sleep less (Kohatsu et al., 2006; Gangwisch et al., 2005 and Kripke et al., 2002). Studies by various investigators (Hasler et al., 2004; Taheri et al., 2004 and Patel et al., 2004) reported sleeping less than 7 hours is
in long term associated with obesity in young adults. Patel and Hu (2008) and Van et al., (2008) also reported that short sleep duration is strongly and consistently associated with concurrent obesity especially in younger age groups. Mozaffarian et al., (2011) suggested that the association between sleep duration and weight gain is characterized by a U shaped curve i.e. weight gain is lowest among individuals who sleep 6 to 8 hours a night and is higher among those who sleep less than 6 hours or more than 8 hours.

Going through the literature available, obesity has been studied in the light of many factors including genetical from twin, adoption as well as family studies and environmental. On the basis of the literature available to the present investigator it is revealed that there is lack of family study on obese individuals in India and in particular Punjab. The present study has been planned to fulfill this gap and is targeted at observing the heritability of obesity from selected sample of obese and non-obese families on the basis of BMI. An attempt has also been made to study and compare the genetics of WHR, WC and BMI among obese and non-obese family members. In the present study an attempt has also been made to observe on the basis of selected genetical markers any similarity if present between obese parents and their obese children. Lastly, the present study also aims to observe the possible role of selected environmental factors contributing in obesity.