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Prevalence and Etiology of Obesity - An Overview

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Abstract: Obesity is an excess body weight due to fat deposition as compared to set standards of body weight. Though it is determined by a number of methods, but body mass index (BMI) has become the measurement of choice for many obesity researchers and health professionals. BMI is a practical indicator of the severity of obesity. A more important aspect of obesity is the regional distribution of excess body fat. Mortality and morbidity ratio vary with the distribution of body fat, with the highest risk linked to excessive abdominal fat, usually called as Central obesity. Waist circumference is a useful measurement to the risks associated with obesity. Waist circumference and BMI are interrelated, waist circumference provides an independent prediction of risk over and above that of BMI. Prevalence of obesity varies amongst countries depending upon environmental and behavioral changes brought about by economic development, modernization and urbanization. The variation in prevalence of obesity epidemic in various races and communities of the world may be attributed to heredity, age, sex, diet, eating patterns, life style and/or behavior. The prevalence of obesity is minimum in China (3.8%) and Singapore (6.7%) and maximum in Micronesian Island of Naru (85% for male and 93% for female). In Pakistan, the prevalence is 7-8%. Obesity develops as a result of a complex interaction between a person's genes and the environment characterized by long-term energy imbalance due to excessive caloric consumption, insufficient energy out put (sedentary lifestyle, low resting metabolic rate) or both. Diet and life style play a significant role both in development and control of obesity. A virus Ad-36 found in obese individuals may be an additional factor to the escalating prevalence of obesity.

Key words: Obesity, prevalence, etiology, diet, lifestyle, virus

Introduction

Every individual needs a certain amount of body fat for stored energy, heat insulation, shock absorption and/or other functions. However, excessive deposition of fat in the body, which is usually referred as overweight or obesity in literature, is dangerous. Overweight refers to an excess body weight compared to set standards. The excess weight may come from muscles (lean body mass), bone, fat (adipose tissues), some time tumors and/or body water. Obesity specially refers to having an abnormally high proportion of total body fat (WHO, 1998; NHLBI, 1998). As a rule, women have more body fat than men. Most health care providers agree that men with more than 25% total body fat and women with more than 30% total body fat should be considered obese. (NIH, 2001)

Assessment of obesity: A number of methods are used to determine overweight and obesity. Some of them are based on weight for height tables and mathematical calculations of the relation between height and weight others are based on measurement of body fat. Even though accurate methods, to assess the exact amount of body fat exist, measuring body fat content by these techniques is often expensive and is not readily available clinically. The most accurate measures are to weigh a person underwater or to use an X-ray test called Dual Energy X-ray Absorptiometry (DEXA). These methods are not practical for average person and are done only in research centers with special equipments. The simpler methods to estimate body fat are, (1) to measure the thickness of the layer of fat just under the skin in several parts of the body (2) Sending a harmless amount of electricity through a person's body. Both methods are used at health clubs and commercial weight loss programs. Results from these methods however, can be inaccurate if done by an inexperienced person or on some one with severe obesity. (NIH, 1996a)

Because measuring the exact amount of a person's body fat is difficult and most expensive, health care providers rely on other means to diagnose if an individual is overweight or obese. Weight-for-Height tables/Metropolitan Life Insurance Tables, which have been used for decades, usually have a range of acceptable weights for a person of a given height (Metropolitan Life Insurance Company, 1983). One problem with these tables is that there are many versions, all with different weight ranges. In addition, separate tables are required for men and women. These tables are based on mortality outcomes and do not necessarily predict morbidity. Another problem is that they do not distinguish between excess fat and muscles. A very muscular person may appear obese, according to

the tables, when he or she is not (NIH, 2001).

In recent years, body mass index (BMI) also called Quetelet Index (initially described by Quetelet in 1869), has become the measurement of choice for many obesity researchers and health professionals, to measure overweight and obesity in adults. BMI is a practical indicator of the severity of obesity. BMI for various height and weight, for easy reference, is given in (Table 1). 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; Gallagher *et al.*, 1996; Revicki and Isreal, 1986; Garrow and Webster, 1985). There are however, some limitations with using BMI as a measure of total body fat that must be recognized. For example, BMI overestimates body fat in persons who are very muscular and/or have fluid retention (edema) and can underestimate body fat in persons who have lost muscle mass (e.g., the elderly). According to WHO (1997), BMI should be used to classify overweight and obesity and to estimate relative risk for diseases compared to normal weight. Calculating BMI is simple, rapid and inexpensive; moreover the classification can be applied generally to adults. BMI is calculated by taking the weight of the individual in kilograms (Kg) and dividing it by the square of his/her height taken in meters (m^2) or weight of an individual taken in pounds (lbs) divided by the square of his/her height taken in inches ($inches^2$) multiplied by 703, a factor.

$$BMI = \left[\frac{\text{Weight in kg}}{(\text{Height in meter})^2} = \frac{\text{kg}}{(\text{m})^2} \right]$$

or

$$\left[\frac{\text{Weight in lbs}}{(\text{Height in meter})^2} \times 703 = \frac{\text{lbs}}{(\text{inches})^2} \times 703 \right]$$

Classification of weight profile: WHO (1997), standards for classification of weight based on BMI is given in Table 2. The basis for this BMI classification scheme stems from observational and epidemiologic studies, which relate BMI to risk of morbidity and mortality (Colditz *et al.*, 1995 and 1990; Chan *et al.*, 1994; Lindsted *et al.*, 1991; Higgins *et al.*, 1988; Hubert *et al.*, 1983; Rabkin *et al.*, 1977).

BMI is a scale used for determining the weight status of an individual and the associated risks. It does not provide any clue for the distribution of fat in the various parts of the body. 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. Mortality and morbidity ratio vary with the distribution of body fat, with the highest risk linked to excessive abdominal fat, usually called as Central obesity or Android obesity (Sugerman *et al.*, 1997; Pouliot *et al.*, 1992; Ducimetiere and Richard, 1989; Despres *et al.*, 1989; Lapidus *et al.*, 1984; Larson *et al.*, 1984). Abdominal fat is described as having three compartments: visceral, retroperitoneal and subcutaneous (Abate *et al.*, 1996; Jensen *et al.*, 1995; Sjostrom *et al.*, 1986). Several studies suggest that the visceral fat component of abdominal fat is the most strongly correlated with risk factors (Abate *et al.*, 1995; Pouliot *et al.*, 1992; Bjorntorp, 1988; Fujioka *et al.*, 1987). Studies indicate that intra abdominal fat/android obesity (characterized by increased waist circumference) is associated with an increased risk for a number of diseases, including cardiovascular diseases (CVD), cardiac death, non insulin dependent diabetes mellitus (NIDDM), 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; Calle *et al.*, 1999; Ledoux *et al.*, 1997; Goodpaster *et al.*, 1997; Rabkin *et al.*, 1997; Abate *et al.*, 1995 and 1996; Chan *et al.*, 1994; Kannel *et al.*, 1991; Kissebah *et al.*, 1989). The presence of increased total abdominal fat appears to be an independent risk predictor when the BMI is not markedly increased (Lemieux *et al.*, 1996).

The importance of Central obesity is clear in populations (e.g. Asian), who tend to have relatively low BMI but high levels of abdominal fat and are particularly prone to NIDDM, hypertension and coronary heart disease (CHD). Recently an Indian study revealed that almost 20% of adults who were not overweight or obese still had Central obesity, putting them at a greater risk of developing these associated diseases (Gopalan, 1998). According to the American Heart Association, boys with chubby bellies are more likely to have high blood pressure than their slimmer counterparts (American Heart Association, 2002). In another prospective study, Oppert *et al.* (2002), found that Intra-abdominal fat appears to be the main body compartment involved in risk of cardiac death.

Relatively accurate measure of total abdominal fat can be made by magnetic resonance imaging (Abate *et al.*, 1996) or computed tomography (Albu *et al.*, 1997; Lean *et al.*, 1995). These methods, however, are expensive and not readily available for clinical practice. Research with these techniques, nonetheless, has shown that the waist circumference correlates with the amount of fat in the abdomen and thus is an indicator of the severity of abdominal obesity. The waist-to-hip ratio (WHR) 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). However, waist circumference has been found to be a better marker of abdominal fat content than WHR (Despres *et al.*, 1989). Therefore, in clinical practice, abdominal fat

Table 1: Body mass index (BMI)

BMI	19	20	21	22	23	24	25	26	27	28	29
Height (inches)	Body weight										
	Normal weight						Over weight				
58	91	96	100	105	110	115	119	124	129	134	138
59	94	99	104	109	114	119	124	128	133	138	143
60	97	102	107	112	118	123	128	133	138	143	148
61	100	106	111	116	122	127	132	137	143	148	153
62	104	109	115	120	126	131	136	142	147	153	158
63	107	113	118	124	130	135	141	146	152	158	163
64	110	116	122	128	134	140	145	151	157	163	169
65	114	120	126	132	138	144	150	156	162	168	174
66	118	124	130	136	142	148	155	161	167	173	179
67	121	127	134	140	146	153	159	166	172	178	185
68	125	131	138	144	151	158	164	171	177	184	190
69	128	135	142	149	155	162	169	176	182	189	196
70	132	139	146	153	160	167	174	181	188	195	202
71	136	143	150	157	165	172	179	186	193	200	208
72	140	147	154	162	169	177	184	191	199	206	213
73	144	151	159	166	174	182	189	197	204	212	219
74	148	155	163	171	179	186	194	202	210	218	225
75	152	160	168	176	184	192	200	208	216	224	232
76	156	164	172	180	189	197	205	213	221	230	238

BMI	30	31	32	33	34	35	36	37	38	39	40
Height (inches)	Body Weight										
	Obesity					Morbid Obesity					
58	143	148	153	158	162	167	172	177	181	186	191
59	148	153	158	163	168	173	178	183	188	193	198
60	153	158	163	168	174	179	184	189	194	199	204
61	158	164	169	174	180	185	190	195	201	206	211
62	164	169	175	180	186	191	196	202	207	213	218
63	169	175	180	186	191	197	203	208	214	220	225
64	174	180	186	192	197	204	209	215	221	227	232
65	180	186	192	197	204	210	216	222	228	234	240
66	186	192	198	204	210	216	223	229	235	241	244
67	191	198	204	211	217	223	230	236	242	249	255
68	197	203	210	216	223	230	236	243	249	256	262
69	203	209	216	223	230	236	243	250	257	263	270
70	209	216	222	229	236	243	250	257	264	271	278
71	215	222	229	236	243	250	257	265	272	279	286
72	221	228	235	242	250	258	265	272	279	287	294
73	227	235	242	250	257	265	272	280	288	295	302
74	233	241	249	256	264	272	280	287	295	303	311
75	240	248	256	264	272	279	287	295	303	311	319
76	246	254	263	271	279	287	295	304	312	320	328

Table 1 Contd.

BMI	40	41	42	43	44	45	46	47	48	49	50
Height (inches)	Body Weight										
	Morbid Obesity										
58	191	196	201	205	210	215	220	224	229	234	239
59	198	203	208	212	217	222	227	232	237	242	247
60	204	209	215	220	225	230	235	240	245	250	255
61	211	217	222	227	232	238	243	248	254	259	264
62	218	224	229	235	240	246	251	256	262	267	273
63	225	231	237	242	248	254	259	265	270	278	282
64	232	238	244	250	256	262	267	273	279	285	291
65	240	246	252	258	264	270	276	282	288	294	300
66	247	253	260	266	272	278	284	291	297	303	223
67	255	261	268	274	280	287	293	299	306	312	319
68	262	269	276	282	289	295	302	308	315	322	328
69	270	277	284	291	297	304	311	318	324	331	338
70	278	285	292	299	306	313	320	327	334	341	348
71	286	293	301	308	315	322	329	338	343	351	358
72	294	302	309	316	324	331	338	346	353	361	368
73	302	310	318	325	333	340	348	355	363	371	378
74	311	319	326	334	342	350	358	365	373	381	389
75	319	327	335	343	351	359	367	375	383	391	399
76	328	336	344	353	361	369	377	385	394	402	410

Table 2: WHO standards for classification of weight (WHO, 1997)

Category	BMI	Risk of co-morbidities
Underweight	< 18.5	
Normal Weight	18.5-24.9	Average
Overweight	25.0-29.9	Increased
Obesity Class I	30.0-34.9	Moderate
Obesity Class II	35-39.9	Severe
Morbid Obesity Class III	≥40.0	Very severe

Table 3: Risk of obesity-associated metabolic complications

Metabolic Complication	Waist circumference	
	Men	Women
Increased	≥ 94 cm	≥ 80 cm
Substantially Increased	≥ 102 cm	≥ 88 cm

content should be assessed by measuring waist circumference (WHO, 1997).

Although waist circumference and BMI are interrelated, waist circumference provides an independent prediction of risk over and above that of BMI. Waist circumference measurement is particularly useful in patients who are categorized as normal or overweight on the BMI scale. At BMIs 35, waist circumference has little added predictive power of disease risk beyond that of BMI. It is therefore not necessary to measure waist circumference in individuals with BMIs 35 (Table 4) (NHLBI, 1998).

Like BMI, WHO has set cutoff points for waist circumference, But unlike BMI, these are sex and population specific (Table 3) (WHO, 1997; Bouchard and Tremblay, 1990; Peiris *et al*., 1989).

Prevalence of obesity: Obesity prevails in various communities of the world. Its prevalence is escalating at an alarming rate to epidemic proportions through out the developed world. Furthermore, obesity is no longer just a concern for developed countries, but is also becoming an increasing problem in many developing countries. According to WHO report, there are more than 250 million obese adults and about 1.1 billion overweight people worldwide (WHO, 1998). Environmental and behavioral changes brought about by economic development, modernization and urbanization has been linked to the rise in global obesity. The variation in prevalence of obesity epidemic in various races and community of the world may be attributed to heredity, age, sex, diet, eating patterns, life style and/or behavior (Epstein and Higgins, 1992; Gurney and Gorstein, 1988).

Obesity is a serious and widespread health problem in only certain kind of societies, characterized by economic modernization, affluence, food surplus and social stratification. Numerous studies of traditional societies undergoing the process of economic modernization demonstrate rapid increases in the prevalence of obesity. Obesity is first of the "diseases of civilization" to appear. (Trowell and Burkitt's, 1981).

The highest reported prevalence of obesity is on the Micronesian island of Nauru, 85% for males and 93% for

Table 4: Classification of Overweight and Obesity by BMI, Waist Circumference and Associated Disease Risk*

Category	BMI	Obesity Class	Disease Risk* (Relative to Normal Weight and Waist Circumference)	
			Men < 102 cm Women < 88 cm	Men 102 cm Women 88 cm
Underweight	< 18.5		-----	-----
Normal†	18.5 - 24.9		-----	-----
Overweight	25.0 - 29.9		Increases	High
Obesity	30.0 - 34.9	I	High	Very High
	35.0 - 39.9	II	Very High	Very High
Extreme Obesity	≥40	III	Extremely High	Extremely High

*: Disease risk for type 2 diabetes, hypertension and CVD. †: Increased waist circumference can also be a marker for increased risk even in persons of normal weight.

females (Collins *et al.*, 1990). The second highest prevalence of obesity (75% of Adult population) has been reported in the urban Samoa (WHO, 1998).

In Europe, there are higher prevalence's of obesity in Southern European countries than Northern ones and within those countries, the risk of obesity is higher in rural than urban areas (Kluthe and Schubert, 1985). Current prevalence data from individual national studies suggests that the range of obesity prevalence in European countries is from 10 to 20% for men and 10 to 25% for women. Prevalence of obesity has increased by about 10-40% in the majority of European countries in the past 10 years. The most dramatic increase has been in the UK where it has more than doubled since 1980. In UK 61% of men and 52% of women are either overweight or obese. A greater proportion of men (45%) than women (34%) are overweight, but a slightly lower proportion of men (16%) than women (17%) is obese (WHO, 1998). In Finland 48% of men are overweight and 19% are obese, while in women these proportions are 32 and 19%. In Belgium, 12.1 and 18.4% of obesity has been reported in male and females respectively (Stam-Moraga *et al.*, 1999). In the middle Aged Scottish Population, Bolton-Smith and Woodward (1994) reported 43 and 11% overweight and obesity in men and 38 and 14% overweight and obesity in women.

Obesity currently affects about one third of all men and women in North America between the ages of 18 and 65 years (Flegal *et al.*, 1998; Mcdonald *et al.*, 1997). According to National Health and Nutrition Examination Survey NHANES-III Data about 55% of US adults of age 20 years and older are either overweight or obese, of these 33% are overweight and 22% are obese (Kuczmarski *et al.*, 1997, Gurney and Gorstein, 1988 and CDC, 2000). In Canada the prevalence has been reported as 35 and 27% in males and females (Mcdonald *et al.*, 1997; Reeder *et al.*, 1992). Gigante *et al.* (1997) reported 21% (25% women and 15% men) prevalence of obesity in Brazil.

The prevalence rate of obesity among adults of Arabian Peninsula is among the highest in the world. Musaiger *et al.* (2000) studied the prevalence of obesity in relation to lifestyle and social factors in Bahraini natives, aged 30-79 years. They found that prevalence of obesity was 56% and 79.6% in males and females and this higher incident was attributed to sedentary life style, dietary habits and history of diabetes and hypertension. Al-Mahroos and Al-Roomi (2001) reported 25 and 32% of obesity in Adult Bahraini (Aged 40-69 Years) men and women respectively. The prevalence of obesity was significantly higher among female subjects than males in all the age groups. Overweight and obesity were more prevalent among those with higher levels of education and people with high incomes. Obesity was inversely related to physical activity. In another survey prevalence of obesity in Al-khobar Saudi Arabia was 28.6% by BMI and 39.3% by triceps skinfold measurement in female students (Al-Abad and Al-Sowielem, 1998). Similarly, Al-Isa (1995) assessed prevalence of obesity in Kuwaiti adults and reported 70.2% overweight and 36.4% obesity. El-Hazmi and Warsy (1997) reported prevalence of 27 and 25% overweight and 13 and 20% obesity in Saudi males and females respectively.

Abbas *et al.* (2003) studied the prevalence of obesity in relation to diet and physical work in Mardan, Pakistan. He reported a prevalence of 7% obesity and 34% overweight in Tehsil Mardan, Pakistan. Khan *et al.* (2003) conducted a study on the prevalence of obesity and overweight in educational, research and health institutions of the University of Peshawar Campus, Pakistan. They reported that the prevalence of obesity and overweight was 8.0 and 29.6% respectively. Furthermore the prevalence of obesity and overweight in high and low income male groups was 7.5 and 34.9% and 6.5 and 24.9% respectively, while the prevalence of obesity and overweight in high and low income female groups was 13.6 and 31.8% and 7.4 and 30.5% respectively. Dhurandhar and Kulkarni (1992) studied the prevalence of obesity in 1784 adults of Bombay, India and found lowest prevalence (11%) of obesity in students and highest (53%) in male medical doctors. Prevalence was highest for the age group 31-50 years. The prevalence of obesity in urban community of South Delhi, India was 9.4 and 19.3% in men and women (Sood *et al.*, 1985). In Taiwan, Ding *et al.* (1995) reported 12.6% prevalence of obesity. Chiu *et al.* (2000) reported 27 and 35% prevalence of overweight and 3 and 6% obesity in Taiwanese men and women respectively. Arshad *et al.* (1996) reported 37.8% obesity in Kualalampur, Malaysia. According to 1998, National

Health Survey of Singapore, 24.4% of adults (Aged 18-69 years) were overweight and 6.0% were obese. Obesity ratio was slightly more in females (6.7%) than males (5.03%). Moreover obesity was more prevalent in Malays (16.2%), followed by Indians (12.2%) and Chinese (3.8%). The highest proportion of obesity was found in 50-59 years age group. In another study of 2636 adult males and 2111 adult females from three ethnic groups (Malays, Indians and Chinese) in urban areas, 29% of males were overweight, out of which 5% were obese. In females, 26% were overweight including 8% who were obese. The study revealed that in urban setting, obesity was more prevalent in both males (29%) and females (26%) while in rural areas, obesity was less prevalent in both male (15%) and females (20%) (Cheah, 2001).

According to National Nutrition Survey of Australia (1995) 29% of women and 45% of men (>19 yrs) were overweight having 18% of both men and women classified as obese. There is a steady increase in the proportion of men and women who are overweight or obese with increasing age. The age group most at risk of overweight and obesity, for both men and women, is the 45-64 year old age bracket with 50% of men and 36% of women overweight and 25% of both men and women classified as obese (Dietitian Association of Australia, 2002).

In Nigeria the prevalence of obesity in males and females, 20-60 years was 41, 54, 78 and 59% in civil servents, chiefs, business executives and market women (Okeke *et al.*,1983).

In developing countries, there is a strong and consistent positive association of social class and obesity for men, women and children. In heterogeneous and affluent societies like the U.S., there is a strong inverse correlation of social class and obesity for females (Sobal, 1991).

Etiology of obesity: Obesity is a chronic condition that develops as a result of a complex interaction between a person's genes and the environment characterized by long-term energy imbalance due to excessive caloric consumption, insufficient energy out put [sedentary lifestyle, low resting metabolic rate (RMR)] or both (Lindpainter, 1995; National Research Council, 1989; NHLBI, 1998; Astrup and Lundsgaard, 1998). It develops in a socio cultural environment characterized by mechanization, sedentary lifestyle and ready access to abundant food.

Genetic influence on the development of obesity: Although the development of obesity has genetic component, the mechanism is not known. Genetic influences are difficult to elucidate and identification of the genes is not easily achieved in familial or pedigree studies. Furthermore, whatever the influence the genotype has on the etiology of obesity, it is generally attenuated or exacerbated by non-genetic factors.

It has been long known that the tendency to gain weight runs in families. However, family members share not only genes but also diet and life style habits that may contribute to obesity. Separating these lifestyle factors from genetic one is often difficult, still, growing evidence points to heredity as a strong determinant factor of obesity (Stunkard, 1996). Obesity appears to be more prevalent in some families and ethnic groups.

Researchers vary in their opinion on the role genetics plays in energy regulation. 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 (Vogler *et al.*, 1995; Tambs *et al.*, 1991; Bouchard *et al.*, 1988). However, studies with identical twins reared apart suggest that the genetic contribution to BMI may be higher, i.e., about 70 percent (Stunkard *et al.*, 1990). There are several other studies of monozygotic twins reared apart that yielded remarkably consistent results (Allison *et al.*, 1996). Some of the reasons behind the different results obtained from twin versus family studies have been reported (Maes *et al.*, 1997; Allison, 1995; Allison and Pi-Sunyer, 1995; Allison *et al.*, 1996).

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. Some studies showed that where both parents were fat, 80% of their children, even if not raised by their genetic parents, were also fat. 40% were fat when one of the parent was obese and only 9% were fat when both parents were lean (Roberts *et al.*, 1988; Stunkard *et al.*, 1986). Another study found that twins, regardless of whether they were reared apart or in the same home environment, were about 70% likely to weigh the same (Stunkard *et al.*, 1990). Twin studies have demonstrated genetic influences on RMR, feeding behaviour, changes in energy expenditures in response to overfeeding, lipoprotein lipase activity and basal rate of lipolysis (Rosenbaum and Leibel, 1988; Bouchard *et al.*, 1990; Bogardus *et al.*, 1986; Brook 1977; American Heart Association, 1998) A recent population based study suggested that 35% of the adjusted variation in BMI was accounted for by a single recessive locus while polygenic loci accounted for 42% of variation. (American Heart Association, 1996).

In women, the hereditary advantage is even more important. For instance, we know that women have more fat than men and it is usually distributed on their bodies differently. Newborn girls in all ethnic groups weight less at birth than newborn boys but have a higher percentage of body fat (Beller, 1977). This trend continues as the child matures, with women having an average of two times the body fat of men (Bailey, 1982).

Support for a role of specific genes in human obesity of body fat content has been obtained from various studies. From the research currently available, several genes seem to have the capacity to cause obesity or to

increase the likelihood of becoming obese (Perusse *et al.*, 1996). In rare cases, human obesity results from a single gene disorder such as in the Bardet Biedl, Prader Willi, Ahlstrom and Cohen syndromes (Spiegelman and Flier, 1996). The rodent obesity gene for leptin, a natural appetite-suppressant hormone, has been cloned as has been its receptor (Tartaglia *et al.*, 1995; Zhang *et al.*, 1994). In addition, other single gene mutants have been cloned (Montague *et al.*, 1997; Tartaglia *et al.*, 1995). However, their relationship to human disease has not been established, except for one study describing two subjects with a leptin mutation (Montague *et al.*, 1997). This suggests that for most cases of human obesity, susceptibility genotypes may result from variations of several genes.

Although mutations in the Ob gene have not been found in humans, there is evidence suggesting a linkage between the Ob gene and some obese populations (Clement *et al.*, 1996; Reed *et al.*, 1996). Persons with mutations in the gene that encodes for adipose beta-3 receptors appear prone to weight gain (Clement *et al.*, 1995). Two other genes that have been implicated in the development of human obesity are the genes that encode for the gluco corticoid receptor and Na-K-ATPase (Clement *et al.*, 1996; Deriaz *et al.*, 1994) Severely or morbidly obese persons are, on the average, about 10 to 12 BMI units heavier than their parents and siblings. Several studies have reported that a single major gene for high body mass was transmitted from the parents to their children. The trend implies that a major recessive gene, accounting for about 20 to 25 percent of the variance, is influenced by age and has a frequency of about 0.2 to 0.3% (Bouchard *et al.*, 1998). However, no gene(s) has (have) yet been identified. Evidence from several studies has shown that some persons are more susceptible to either weight gain or weight loss than others (Bouchard and Trembly, 1990; Bouchard *et al.*, 1990).

Environmental influences on the development of obesity: Since most often genetic factors account for only a third of the variance in body weight, environmental influences must therefore account for the balance. Several environmental factors, involving both energy intake and energy output, contribute to obesity. Environmental influences on overweight and obesity are primarily related to food intake and physical activity behaviors. Most evidence suggests that the main reason for the rising prevalence is a combination of changes in eating patterns and less active lifestyles. According to WHO (1997), the fundamental causes of the obesity epidemic are sedentary lifestyles and high-fat, energy-dense diets.

Diet: Diet plays a significant role both in development and control of obesity. For years, doubt has persisted about the contribution of excessive food intake to obesity. However, a recent study erased this doubt and made it clear that obesity is associated with increased food consumption (Lichtman *et al.*, 1992). Intake of excess dietary fat has been implicated as a major cause of obesity for decades (Lissner and Heitmann, 1995). Fat provides more energy than protein and carbohydrate per unit weight and contribute to obesity. It can also influence food intake, energy metabolism and substrate oxidation. High fat foods are also preferentially selected by individual because of their high palatability and a weak satiety effect. A consistent body of evidence links dietary fat intake to body fat, likely because of the energy density of fat; high-fat diets tend to be high-energy diets (Bray and Popkin, 1998; WIN, 1998). There is no evidence, linking carbohydrate intake to obesity risk (Bolton-Smith, 1996) on a population basis. Satiety relates to appetite control and consequently to weight regulation. Available evidence suggests that protein is more satiating than carbohydrate or fat; high-fiber carbohydrate has a satiety index nearly comparable to protein. In their natural state, fruits and vegetables provide fewer calories than other choices, especially if they replace foods high in fat (US Department of Health and Human Services, 1995 and 1998).

According to Ding *et al.* (1995), obese individual have higher serum cholesterol, triglycerides and Lipoprotein and lower HDL cholesterol. According to Bolton-Smith and Woodward (1994) composition of diet has a significant effect on relative weight. Arshad *et al.* (1996) examined the energy, carbohydrate, protein and fat intakes in relation to nutritional status in government office workers In Kualalampur, Malaysia. They reported 37.8% prevalence of obesity and 1709±637 kcal/day energy intake. The mean intakes of carbohydrate, fat and protein were 55.7±7.6, 29.7±21.7 and 15.6±3.8% respectively.

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 the 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 (NIH, 1996b; French *et al.*, 2001).

Lifestyle: Lifestyle is an important factor to be considered in obesity development and control. A strong link exists between physical inactivity and weight gain. Multiple cohort and cross-sectional studies have shown an association between obesity and 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. Physical

inactivity enhances the severity of other risk factors, but it also has been shown to be an "independent" risk factor for all-cause mortality or CVD mortality (Leon, 1997; Paffenbarger *et al.*, 1993). Although physical inactivity is not listed as a risk factor that modifies the intensity of therapy required for elevated cholesterol or blood pressure, increased physical activity is indicated for the management of these conditions. The presence of physical inactivity in an obese person warrants intensified efforts to remove excess body weight, because physical inactivity and obesity both heighten disease risks (Paffenbarger *et al.*, 1993).

Martinez-Gonzalez *et al.* (1999) examined physical activity, sedentary lifestyle and obesity in a sample of 15 member countries of European Union and found a strong association of obesity and higher body weight with a sedentary lifestyle and lack of physical activities in adult population. Similarly Hernandez *et al.* (1999) investigated the relation of obesity with physical activity, Television programs and other forms of video viewing among 712 children's, 9-16 years old, in Mexico City. They reported that prevalence of obesity was related with physical inactivity and television viewing.

Virus: According to Dhurandhar *et al.* (1997), a virus Ad-36 might be an additional factor for the escalating epidemic of obesity. For the last few years Dhurandher and his colleagues are researching a virus Ad-36 found almost exclusively in obese human beings. Of the subjects who have been examined so far, almost no normal weight individuals have antibodies for the virus, whereas 15-60 percent of obese patients test positive. They are not suggesting that this virus is the only cause of weight gain, but rather that it may be an additional factor to the escalating prevalence of obesity. One question remains. Does obesity predispose patients to the virus, or does the virus cause the weight gain?

The possibility that virus could be a cause of obesity goes back to late 1980's when Dr. Dhurandhar found a similar virus SMAM-1, killing hundreds of thousands of chicken. To establish a connection between the chicken epidemic and his obese patients, he took blood samples from 52 patients and found that 10 of them tested positive for SMAM-1 antibodies. Those 10 were heavier than the other 42 patients and were having lower blood cholesterol levels, like the chickens killed in the epidemic (Dhurandher *et al.*, 1997). Dhurandher wanted to pursue his findings but, realized that neither the finances nor the facilities were available in India. After closing up his practice and heading for the US he interested Dr. Richard Atkinson, in his theory, but the road block to them was the US Department of Agriculture, not allowing importing SMAM-1 virus. Thus they started to look for a similar virus and succeeded to discover Ad-36 virus in the feces of a young German girl with Diarrhea. Afterward to show the connection between Ad-36 and obesity, they tested hundreds of human subjects and performed experiments on a wide variety of animals including chickens, mice and primates. To determine whether Ad-36 would produce obesity in chickens as SMAM-1 had in India, Dhurandher inoculated one day old broilers with the virus and found that they gained more weight and fat than controls. Chickens infected with Ad-36 had increased visceral fat, lower serum cholesterol and triglycerides even though they did not eat any more than the controlled birds (Dhurandher *et al.*, 2000)

Dhurandher next inoculated 35 mice with Ad-36 and when the mice responded similarly to the chickens, he tested mammals (marmosets and monkeys) more similar to human. In marmosets 2 animals that tested positive for the virus, gained significantly more weight than controls (42.8±6.5 gm versus 14.4±5.9gm). The infected marmosets also had substantially lower cholesterol, although triglycerides were not affected (Atkinson, 2000; Dhurandher *et al.*, 1997). To prove that Ad-36 could infect humans as well as primates posed a problem. Dhurandher could not infect human subjects with an active virus; therefore he tested human subjects for antibodies for the Ad-36. Eventually Dhurandher and Atkinson tested 400 subjects in three cities. Of the first 154 obese volunteers and 45 lean controls, 15% of the obese subjects tested positive, but non of the lean subjects (Atkinson, 1998). By 1999, 313 obese and 92 lean human volunteers had been tested for the presence of the antibodies to Ad-36. 100 (32%) of the obese subjects tested positive, but only 4 of the lean controls. Also these subjects had lower serum cholesterol and triglycerides. To investigate environmental versus genetic contributions to obesity, Dhurandher studied 86 pairs of identical twins and found the twin exposed to Ad-36 was significantly heavier than the twin without the antibody, proving that non genetic cause was responsible for the obesity (Dhurandher, 1999).

The mechanism by which Ad-36 causes obesity is unclear. It can be hypothesized that hypothalamic damage caused by viruses might be a cause, but Dhurandher found that this did not turn out to be the case with Ad-36, because brain of chicken infected with Ad-36 was having no damage to hypothalamus or any other location of the brain, eliminating the possibility that central mechanism was responsible. Therefore he turned his attention to the fat cells. Ad-36 infected animals had larger fat cells, more of them and reduced serum cholesterol and triglycerides.

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