

## Health Risks of Overweight and Obesity - An Over View

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**Abstract:** Obesity is a risk factor for the development of various diseases like CHD, hypertension, stroke, NIDDM, osteoarthritis, sleep apnea and cancers of endometrium, breast, prostate and colon. Psychological consequences of obesity range from lowered self-esteem to clinical depression. Many of these conditions are reversible through weight loss and maintenance. Weight reduction may be life saving so it is necessary to reduce weight.

**Key words:** Obesity, risk factor, coronary heart disease (CHD)

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### Introduction

The health consequences of obesity for adults range from a number of non fatal complaints that impact on the quality of life such as respiratory difficulties, musculoskeletal problems, skin problems and infertility; to complaints that lead to an increased risk of premature death including non insulin dependent diabetes mellitus (NIDDM), gallbladder disease, cardiovascular problems [hypertension, stroke and coronary heart disease (CHD)], osteoarthritis, sleep apnea and endometrial, breast, prostate and colon cancers. Psychological consequences of obesity range from lowered self-esteem to clinical depression. Many of these conditions are reversible through weight loss and maintenance. Weight reduction may be life saving so it is necessary to reduce weight. Obesity, now an epidemic of global proportions is creating major health problems worldwide. It is well established that obesity is associated with a substantial burden of illness and health care costs (Bjorntorp, 1997; Rabkin *et al.*, 1997). Obesity has been reported to be associated with higher mortality (Fitzgerald and Jarret, 1992) and morbidity (Kannel *et al.*, 1991). Prevalence and prevention of obesity has been reviewed earlier (Afridi *et al.*, 2003), this manuscript reviews the health risks of obesity

**Morbidity of Obesity:** Morbidity for a number of health conditions increases as BMI increases above 20. Obesity is associated with the development of several diseases, including hypertension (Reeder *et al.*, 1997; Loggie *et al.*, 1984; Havlik *et al.*, 1983), dyslipidemia and NIDDM (Harris *et al.*, 1998; Ford *et al.*, 1997; Colditz *et al.*, 1995, 1990; Chan *et al.*, 1994; Manson *et al.*, 1992), hyperlipidemia (Wilcosky *et al.*, 1990; Freedman *et al.*, 1990), CHD (Willett *et al.*, 1995; Rabkin *et al.*, 1997; Harris *et al.*, 1997), stroke (Rexrode *et al.*, 1997; Walker *et al.*, 1996), obstructive sleep apnea, (Chua and Chediak, 1994; Loubé *et al.*, 1994; Shepard *et al.*, 1992; Wittels and Thompson, 1990; Smith *et al.*, 1985), Osteoarthritis, (Gelber *et al.*, 1999; Cicuttini *et al.*, 1996;

Hochberg *et al.*, 1995; Carman *et al.*, 1994; Hart and Spector, 1993), cholelithiasis/gallstones (Stampfer *et al.*, 1992) and cancers of the breast, (Huang *et al.*, 1997; Ballard-Barbash and Swanson, 1996), uterus, (Ballard-Barbash and Swanson, 1996; Kelsey *et al.*, 1982) prostate (Snowdon *et al.*, 1982) and colon (Giovannucci, 1995, Giovannucci *et al.*, 1995, 1996; Phillips and Snowdon, 1985). It is also associated with psychological disorders, including depression (Stunkard and Wadden, 1992; Wadden and Stunkard 1985; Wardle, 1996), low self-esteem (Myers and Rosen, 1999) anorexia nervosa and bulimia. (Wardle, 1996; Sarlio-Lahteenkorva *et al.*, 1995). Furthermore, obesity is associated with complications of pregnancy, menstrual irregularities and hirsutism (Prentice and Goldberg, 1996; Rich-Edwards *et al.*, 1994; Smith *et al.*, 1994; Keppel and Taffel, 1993; Garbaciak *et al.*, 1985)

**Hypertension:** High blood pressure, defined as mean systolic blood pressure  $\geq 140$  mm Hg, or mean diastolic blood pressure  $\geq 90$  mm Hg, or currently taking anti-hypertensive medication. Obesity and hypertension are co-morbid risk factors for the development of cardiovascular disease. The patho-physiology underlying the development of hypertension associated with obesity includes sodium retention and associated increases in vascular resistance, blood volume and cardiac output. These cardiovascular abnormalities associated with obesity are believed to be related to a combination of increased sodium retention, increased sympathetic nervous system activity, alterations of the rennin-angiotensin system and insulin resistance. The precise mechanism whereby weight loss results in a decrease in blood pressure is unknown. However, it is known that weight loss is associated with a reduction in vascular resistance and total blood volume and cardiac output, an improvement in insulin resistance, a reduction in sympathetic nervous system activity and suppression of the activity of the rennin-angiotensin aldosterone system (Reeder *et al.*, 1997; Jacobs and

Sowers, 1993; Frohlich *et al.*, 1992; Landsberg and Krieger, 1989; Rocchini *et al.*, 1989; Loggie *et al.*, 1984; Havlik *et al.*, 1983).

**Dyslipidemia:** Higher body weight is associated with higher levels of total serum cholesterol, defined as  $\geq$  240 mg/dL in both men and women (Denke *et al.*, 1994, 1993) at levels of BMI  $>$  25. Several large longitudinal studies provide evidence that obesity is associated with increased cholesterol levels (Hershcopf *et al.*, 1982). In women, the incidence of hypercholesterolemia increases with increasing BMI (Manson *et al.*, 1990). Total cholesterol levels are usually higher in persons with predominant abdominal obesity (Reeder *et al.*, 1992 <[http:// www. nhlbi. nih. gov/ guidelines/ obesity/e\\_txbk/refs/refmenu.htm](http://www.nhlbi.nih.gov/guidelines/obesity/e_txbk/refs/refmenu.htm)>).

The strong association of triglyceride levels with BMI has been shown in both cross-sectional and longitudinal studies, for both sexes and all age groups (Denke *et al.*, 1994 and 1993 <[http://www.nhlbi.nih.gov/guidelines/obesity/e\\_txbk/refs/refmenu.htm](http://www.nhlbi.nih.gov/guidelines/obesity/e_txbk/refs/refmenu.htm)>; Mann *et al.*, 1988). HDL-cholesterol levels at all ages and weights are lower in men than in women. Low HDL-cholesterol is defined as  $<$  35 mg/dL in men and  $<$  45 mg/dL in women (Brown *et al.*, 1998). Cross-sectional studies have reported that HDL-cholesterol levels are lower in men and women with higher BMI (Garrison *et al.*, 1980; Glueck *et al.*, 1980). Longitudinal studies have found that changes in BMI are associated with changes in HDL-cholesterol. A BMI change of 1 unit is associated with an HDL-cholesterol change of 1.1 mg/dL for young adult men and an HDL-cholesterol change of 0.69 mg/dL for young adult women (Anderson *et al.*, 1987).

**Non insulin dependent diabetes mellitus (NIDDM):** The increased risk of diabetes, as weight increases, has been shown by prospective studies in Norway (Westlund and Nicolaysen, 1972), the United States (Lew and Garfinkel, 1979), Sweden (Larsson *et al.*, 1981) and Israel (Medalie *et al.*, 1974). More recent studies found that the risk of developing NIDDM increases as BMI increases from 22 (Colditz *et al.*, 1990). The development of NIDDM has been found to be associated with weight gain after age 18 in both men and women (Chan *et al.*, 1994; Colditz *et al.*, 1990). The relative risk of diabetes increases by approximately 25 percent for each additional unit of BMI over 22 (Colditz *et al.*, 1995). Both cross-sectional (Haffner *et al.*, 1991; Despres *et al.*, 1989) and longitudinal studies (Chan *et al.*, 1994; Lundgren *et al.*, 1989 <[http://www. nhlbi. nih. gov/guidelines/obesity/e\\_txbk/refs/refmenu.htm](http://www.nhlbi.nih.gov/guidelines/obesity/e_txbk/refs/refmenu.htm)>) show that abdominal obesity is a major risk factor for NIDDM. Khan *et al.* (1994) reported that 95% of male and almost all of the female diabetic individuals were overweight at the onset of diabetes on WHO standards.

**Coronary heart disease:** Numerous studies have shown that obesity and excess abdominal fat are directly related to cardiovascular risk factors. Obesity and abdominal fat are also associated with increased morbidity and mortality from CHD (Higgins *et al.*, 1988; Donahue and Abbott, 1987; Lapidus *et al.*, 1989; Larson *et al.*, 1984; Hubert *et al.*, 1983).

Recent studies have shown that the risks of nonfatal myocardial infarction and CHD death increase with increasing levels of BMI. Risks are lowest in men and women with BMI of 22 or less and increase with even modest elevation of BMI. Relative risks for CHD are twice as high at BMI of 25 to 28.9 and more than three times as high at BMI of 29 or greater, compared with BMI of less than 21 (Willett *et al.*, 1995). In British men, CHD incidence increased at BMI above 22 and an increase of 1 BMI unit was associated with a 10 percent increase in the rate of coronary events (Shaper *et al.*, 1997). Similar relationships between increasing BMI and CHD risk have been shown in Finnish, Swedish, Japanese and U.S. populations (Jousilahti *et al.*, 1996; Willett *et al.*, 1995; Tokunaga *et al.*, 1991).

**Congestive heart failure:** Overweight and obesity have been identified as important and independent risk factors for congestive heart failure (CHF) in a number of studies (Eriksson *et al.*, 1991, 1989). CHF is a frequent complication of severe obesity and a major cause of death; duration of the obesity is a strong predictor of CHF (Shimizu and Isogai, 1993). Since hypertension and NIDDM are positively associated with increasing weight, the coexistence of these conditions facilitates the development of CHF (Urbina *et al.*, 1995).

Obesity can result in alterations in cardiac structure and function even in the absence of systemic hypertension or underlying heart disease. Ventricular dilatation and eccentric hypertrophy may result from elevated total blood volume and high cardiac output. Diastolic dysfunction from eccentric hypertrophy and systolic dysfunction from excessive wall stress result in so-called "obesity cardio-myopathy" (Alpert and Hashimi, 1993; Garavaglia *et al.*, 1988).

**Stroke:** The relationship of cerebrovascular disease to obesity has not been as well studied as the relationship to CHD. One report suggests that overweight might contribute to the risk of stroke, independent of the known association of hypertension and diabetes with stroke (Hubert *et al.*, 1983).

Recent prospective study has demonstrated that the risk of stroke shows a graded increase as BMI rises. For example, ischemic stroke risk is 75 percent higher in women with BMI  $>$  27 and 137 percent higher in women with a BMI  $>$  32, compared with women having a BMI  $<$  21 (Rexrode *et al.*, 1997).

**Gallstones:** The risk of gallstones increases with adult weight. Risk of gallstones is as high as 20 per 1,000 women per year when BMI is above 40, compared with 3 per 1,000 among women with BMI < 24 (Stampfer *et al.*, 1992).

**Osteoarthritis:** Individuals who are overweight or obese increase their risk for the development of osteoarthritis (Cicuttini *et al.*, 1996; Hochberg *et al.*, 1995; Carman *et al.*, 1994; Hart and Spector, 1993). The association between increased weight and the risk for development of knee osteoarthritis is stronger in women than in men (Felson *et al.*, 1988). In a study of twin middle-aged women, it was estimated that for every kilogram increase of weight, the risk of developing osteoarthritis increases by 9 to 13 percent. The twins with knee osteoarthritis were generally 3 to 5 kg heavier than the co-twins with no disease (Cicuttini *et al.*, 1996). An increase in weight is significantly associated with increased pain in weight-bearing joints (Huang *et al.*, 1997). There is no evidence that the development of osteoarthritis leads to the subsequent onset of obesity (Carman *et al.*, 1994).

**Sleep apnea:** Obesity, particularly upper body obesity, is a risk factor for sleep apnea and has been shown to be related to its severity (Millman *et al.*, 1995; Young *et al.*, 1993). The major pathophysiologic consequences of severe sleep apnea include arterial hypoxemia, recurrent arousals from sleep, increased sympathetic tone, pulmonary and systemic hypertension and cardiac arrhythmias (Shepard, 1992). Most people with sleep apnea have a BMI > 30 (Chua and Chediak, 1994; Loubé *et al.*, 1994). Large neck girth in both men and women who snore is highly predictive of sleep apnea. In general, men whose neck circumference is 17 inches or greater and women whose neck circumference is 16 inches or greater are at higher risk for sleep apnea (Davies and Stradling, 1990).

**Colon cancer:** Many studies have found a positive relation between obesity and colon cancer in men but a weaker association in women (Martinez *et al.*, 1996; Giovannucci, 1995; Bostick *et al.*, 1994; Lee and Paffenbarger, 1993; Le-Marchand *et al.*, 1992; Chute *et al.*, 1991). More recent data suggest that the relationship between obesity and colon cancer in women may be similar to that seen in men. Twice as many women with a BMI of > 29 has distal colon cancer as women with a BMI < 21 (Giovannucci *et al.*, 1996). In men, the relationship between obesity and total colon cancer is weaker than that for distal colon cancer.

**Breast cancer:** Epidemiological studies show that obesity is directly related to mortality from breast cancer, predominantly in postmenopausal women (Lew and

Garfinkel, 1979), but inversely related to the incidence of premenopausal breast cancer (Chu *et al.*, 1991; Willett *et al.*, 1985; Helmrich *et al.*, 1983). Among postmenopausal women, peripheral fat is the primary source of estrogens, the major modifiable risk factor for postmenopausal breast cancer.

This crossover in the relationship of obesity with breast cancer, pre- and postmenopausally, complicates prevention messages for this common female cancer. Recent data however, show that adult weight gain is positively related to risk of postmenopausal breast cancer. This relation is seen most clearly among women who do not use postmenopausal hormones. A gain of more than 20 lb from age 18 to midlife doubles a woman's risk of breast cancer. Even modest weight gains are positively related to risk of postmenopausal cancer (Huang *et al.*, 1997).

**Endometrial cancer:** Obesity increases the risk of endometrial cancer. The risk is three times higher among obese women (BMI  $\geq$  30) than among normal-weight women. However, the absolute risk of this condition is low when compared to breast cancer, heart disease and diabetes. Adult weight gain is also related to increased risk (Schottenfeld and Fraumeni, 1996).

**Women's reproductive health:** Obesity in premenopausal women is associated with menstrual irregularity and amenorrhea (Hartz *et al.*, 1977). The greater the BMI at age 18 years, even at levels lower than those considered obese, the greater the risk of subsequent ovulatory infertility (Rich-Edwards *et al.*, 1994). The most prominent condition associated with abdominal obesity is polycystic ovarian syndrome (Dunaif, 1992), a combination of infertility, menstrual disturbances, hirsutism, abdominal hyper-androgynism and an-ovulation. This syndrome is strongly associated with hyper-insulinemia and insulin resistance (Garbaciak *et al.*, 1985).

Pregnancy can result in excessive weight gain and retention. (Smith *et al.*, 1994; Keppel and Taffel, 1993) Higher pre-pregnancy weights have been shown to increase the risk of late fetal deaths (Cnattingius *et al.*, 1998). Obesity during pregnancy is associated with increased morbidity for both the mother and the child. A tenfold increase in the prevalence of hypertension and a 10 percent incidence of gestational diabetes has been reported in obese pregnant women (Johnson *et al.*, 1987). Obesity also is associated with difficulties in managing labor and delivery, leading to a higher rate of induction and primary Caesarean section. Risks associated with anesthesia are higher in obese women, as there is greater tendency toward hypoxemia and greater technical difficulty in administering local or general anesthesia (Prentice and Goldberg, 1996). Finally, obesity during pregnancy is associated with an

increased risk of congenital malformations, particularly of neural tube defects (Prentice and Goldberg, 1996).

### **Psychosocial Aspects of Obesity**

**Social stigmatization:** In most of the world societies there are powerful messages that people, especially women, should be thin and that to be fat is a sign of poor self-control (Brownell and Fairburn, 1995; Wadden and Stunkard, 1993; DeJong and Kleck, 1986; Jarvie *et al.*, 1983; Allon, 1982). Negative attitudes about the obese have been reported in children and adults (Lerner and Korn, 1972; Staffieri, 1972, 1967; Richardson *et al.*, 1961), in health care professionals (Blumberg and Mellis, 1985) and in the overweight themselves (Crandall and Biernat, 1990; Maddox *et al.*, 1968).

People's negative attitudes toward the obese often translate into discrimination in employment opportunities (Pingitore *et al.*, 1994; Larkin and Pines, 1979; Roe and Eickwort, 1976), college acceptance (Canning and Mayer, 1996), less financial aid from their parents in paying for college (Crandall and Biernat, 1990; Crandall, 1991), job earnings (Sargent and Blanchflower, 1994), rental availabilities (Karris, 1977) and opportunities for marriage (Gortmaker *et al.*, 1993).

**Psychopathology:** Research relating obesity to psychological disorders and emotional distress is based on community studies and clinical studies of patients seeking treatment. In general, community-based studies have not found significant differences in psychological status between the obese and non obese (Wadden and Stunkard, 1993, 1987; O'Neil and Jarrell, 1992). However, several recent European studies in general populations do suggest a relationship between obesity and emotional problems (Lissau and Sorensen, 1994; Sullivan *et al.*, 1993, Lapidus *et al.*, 1989). Thus, it may be premature to state that there is no association between obesity and psychopathology or emotional distress in the general population. More focused, hypothesis-driven and long-term studies are needed (Friedman and Brownell, 1995).

**Binge eating disorder:** Binge eating disorder (BED) is characterized by eating larger amounts of food than most people would eat in a discrete time period (e.g., 2 hours) with a sense of lack of control during these episodes (American Psychiatric Association, 1994). BED is estimated to occur in 20 to 50 percent of individuals who seek specialized obesity treatment (Marcus *et al.*, 1995; Gormally *et al.*, 1982; Loro and Orleans, 1981). Comparisons have been made between BED and bulimia nervosa (BN), an eating disorder characterized by recurrent and persistent binge eating, accompanied by the regular use of behaviors such as vomiting, fasting, or using laxatives. Studies comparing normal-weight individuals who have BN with obese BED individuals have found that obese binge eaters are less

likely to demonstrate dietary restraint and show few if any adverse reactions to moderate or severe dieting. Most obese binge eaters do not engage in inappropriate compensatory behaviors such as purging (Yanovski *et al.*, 1994).

Compared with BN, the demographic distribution of BED is broader with respect to age, gender and race (Marcus *et al.*, 1995; Spitzer *et al.*, 1993, 1992; Wilfley and Cohen, 1997, 1993; Yanovski, 1993; Yanovski and Sebring, 1994). The difference between BED and BN is dramatic regarding gender. Very few men have BN (Fairburn and Wilson, 1993), whereas the distribution is close to equal in BED (Striegel-Moore *et al.*, 1998; Castonguay *et al.*, 1995).

Compared to obese non bingers, obese individuals with BED tend to be heavier (Telch *et al.*, 1988). Obese individuals with BED have greater psychological distress and have more psychiatric illness (Molinari *et al.*, 1997; Kenardy *et al.*, 1996; Mussell *et al.*, 1996; Specker *et al.*, 1994). Other studies have reported an earlier onset of obesity and a greater percentage of their lifetime on a diet (Brody *et al.*, 1994; De Zwann *et al.*, 1992).

Some studies have shown histories of greater weight fluctuation or weight cycling in obese binge eaters compared with nonbingers (Brody *et al.*, 1994; Spitzer *et al.*, 1992; De-Zwaan *et al.*, 1992), but others have not (Kuehnel and Wadden, 1994). These individuals are also more likely than nonbinging obese people to drop out of behavioral weight loss programs (Marcus *et al.*, 1988) and to regain weight more quickly (Yanovski *et al.*, 1994; Marcus *et al.*, 1988; Keefe *et al.*, 1984).

Critics of behavioral treatment of obesity have argued that caloric restriction may cause or contribute to the episodes of binge eating and BN (Marcus *et al.*, 1995; Yanovski and sebring, 1994; Telch and Agras, 1993; Garner and Wooly 1991).

**Body image:** Body image is defined as the perception of one's own body size and appearance and the emotional response to this perception (O'Neil and Jarrel, 1992; Cash and Hicks, 1990). Inaccurate perception of body size or proportion and negative emotional reactions to size perceptions contribute to poor body image. Obese individuals, especially women, tend to overestimate their body size (Collins, 1987; Collins *et al.*, 1990).

People at greater risk for a poor body image are binge eaters, women, those who were obese during adolescence or with early onset of obesity and those with emotional disturbances (Mussel *et al.*, 1996; Faith and Allison, 1996; Mussel *et al.*, 1996; Grilo *et al.*, 1994; Cash and Hicks, 1990). It is no surprise, then, that in some groups of obese persons, these individuals are more dissatisfied and preoccupied with their physical appearance and avoid more social situations due to their appearance (Cash, 1990; Tiggemann and Rothblum, 1988).

**Mortality of Obesity:** Obesity has been reported to be associated with higher mortality, with an estimated reduction in life expectancy of about 1 year (Manson *et al.*, 1995; Fitzgerald and Jarrett, 1992).

The nature of obesity-related health risks is similar in all populations, although the specific level of risk associated with a given level of overweight or obesity may vary with race/ethnicity and also with age, gender and societal conditions. For example, the absolute risk of morbidity in chronic conditions such as CHD is highest in the aged population, while the relative risk of having CHD in obese versus non obese individuals is highest in the middle adult years (Rabkin, 1997; Feinleib, 1985).

In the majority of epidemiologic studies, mortality begins to increase with BMI above 25. The increase in mortality generally tends to be modest until a BMI of 30 is reached. For persons with a BMI of 30 or above, mortality rates from all causes and especially from cardiovascular disease, are generally increased by 50 to 100 percent above that of persons with BMI in the range of 20 to 25 (Troiano *et al.*, 1996; WHO, 1995; VanItallie and Lew, 1990; Manson *et al.*, 1990; VanItalli, 1985).

Many epidemiologic studies of BMI and mortality have reported a 'U-' or 'J-shaped' relationship between BMI and mortality (WHO, 1995). Mortality rates are elevated in persons with low BMI (usually below 20) as well as in persons with high BMI (Troiano *et al.*, 1996; WHO, 1995; Manson *et al.*, 1990). In some studies, adjustment for factors that potentially confound the relationship between BMI and mortality, such as smoking status and pre-existing illness, tends to reduce the upturn in mortality rate at low BMIs (Manson *et al.*, 1990), but in a meta-analysis the higher mortality at low BMIs was not eliminated after adjustment for confounding factors (Troiano *et al.*, 1996). It is unclear whether the elevated mortality observed at low BMIs is due to an artifact of incomplete control for confounding factors (Lee *et al.*, 1993), inadequate body fat and/or inadequate body protein stores that result from unintentional weight loss (Alison *et al.*, 1997), or individual genetic factors. Currently, there is no evidence that intentional weight gain in persons with low BMI will lead to a reduction in mortality.

Many epidemiological studies suggest that the relationship between BMI and mortality weakens with increasing age, especially among persons aged 75 and above (Stevens *et al.*, 1998; Diehr *et al.*, 1998; Cornoni-Huntley *et al.*, 1991; Harris *et al.*, 1988). Several factors have been proposed to explain this observation. Older adults are more likely than younger adults to have diseases that both increase mortality and cause weight loss leading to lower body weight (Fried *et al.*, 1998; Baumgartner *et al.*, 1995; Losonczy *et al.*, 1995). In addition, as people age, they tend to have larger waist circumferences that increase their risk of mortality even

at lower BMI (Folsom *et al.*, 1993). Also, weight in middle age is positively related to risk of mortality in old age (Losonczy *et al.*, 1995). The impact of smoking on body weight and mortality is likely to be much stronger in older adults because of the cumulative health effects of smoking (Willett *et al.*, 1991). BMI, which is an indirect estimate of adiposity, may underestimate adiposity in older adults whose BMI is similar to younger adults (Roche, 1994).

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