PART II

Establishing the true costs of the problem of overweight and obesity

4. Health consequences of overweight and obesity in adults and children

4.1 Introduction

The health consequences of obesity are many and varied, ranging from an increased risk of premature death to several non-fatal but debilitating complaints that have an adverse effect on quality of life. Obesity is also a major risk factor for NCDs such as NIDDM, CVD and cancer, and in many industrialized countries is associated with various psychosocial problems. Abdominal obesity is of particular concern as it is associated with greater risks to health than is a more peripheral fat distribution.

The health consequences of overweight and obesity in both adults and children are considered here, while the effect of weight loss on these conditions is discussed in section 5.

The key issues covered are:

- The major health consequences associated with overweight and obesity, namely NIDDM, CHD, hypertension, gallbladder disease, psychosocial problems and certain types of cancer.
- The lack of detailed relative risk data for the various health problems associated with obesity. These are available only for a few industrialized countries, and show that the risks of suffering from NIDDM, gallbladder disease, dyslipidaemia, insulin resistance and sleep apnoea are greatly increased in the obese (relative risk (RR) much greater than 3). The risks of CHD and osteoarthritis are moderately increased (RR 2–3) and the risks of certain cancers, reproductive hormone abnormalities and low back pain are slightly increased (RR 1–2).
- Biases such as failure to control for cigarette smoking and unintentional weight loss. When these are removed from the analysis of mortality data, there is an almost linear relationship between BMI and death. The longer the duration of obesity, the higher the risk. Severe obesity is associated with a 12-fold increase in mortality in 25–35-year-olds compared with lean individuals. This highlights the importance of preventing weight gain throughout adult life.
- Excess abdominal fat. This is an independent predictor for NIDDM, CHD, hypertension, breast cancer and premature death.
- Weight gain during early adulthood. Most of this is body fat, which increases health risks.
- The many non-fatal but debilitating conditions that affect the obese. These are responsible for a much reduced quality of life in

overweight patients and are often the primary reason for contact with the health care system. Most of these conditions can be improved with modest weight loss.

- The psychosocial consequences of obesity. These have important implications for disease management, and are compounded by the fact that health professionals often view obese individuals as weak-willed and unlikely to benefit from counselling.
- The association between obesity and certain psychosocial consequences in adolescence, and the persistence of obesity into adulthood.

4.2 Obesity as a risk factor for noncommunicable diseases

Although obesity should be considered as a disease in its own right, it is also one of the key risk factors for other NCDs, such as NIDDM and CHD, together with smoking, high blood pressure and hypercholesterolaemia (*1*). The adverse health consequences of obesity are influenced to a greater or lesser extent by body weight, the location of body fat, the magnitude of weight gain during adulthood, and a sedentary lifestyle (*2*).

As a chronic disease, obesity has many similarities to hypertension and hypercholesterolaemia. Fig. 4.1 shows the positive relationship between relative risk of mortality and: (a) BMI (as an index of obesity); (b) cholesterol; and (c) diastolic blood pressure. In the "moderate-risk" category, which corresponds to the ranges between widely accepted cut-off points for lower and higher risk levels, an increase in any of the three variables greatly increases the risk of mortality. The increase is even steeper in the "high-risk" category, implying greater individual risk. However, from a population perspective, the middle range is of most concern as this encompasses the greatest number of people (*2*).

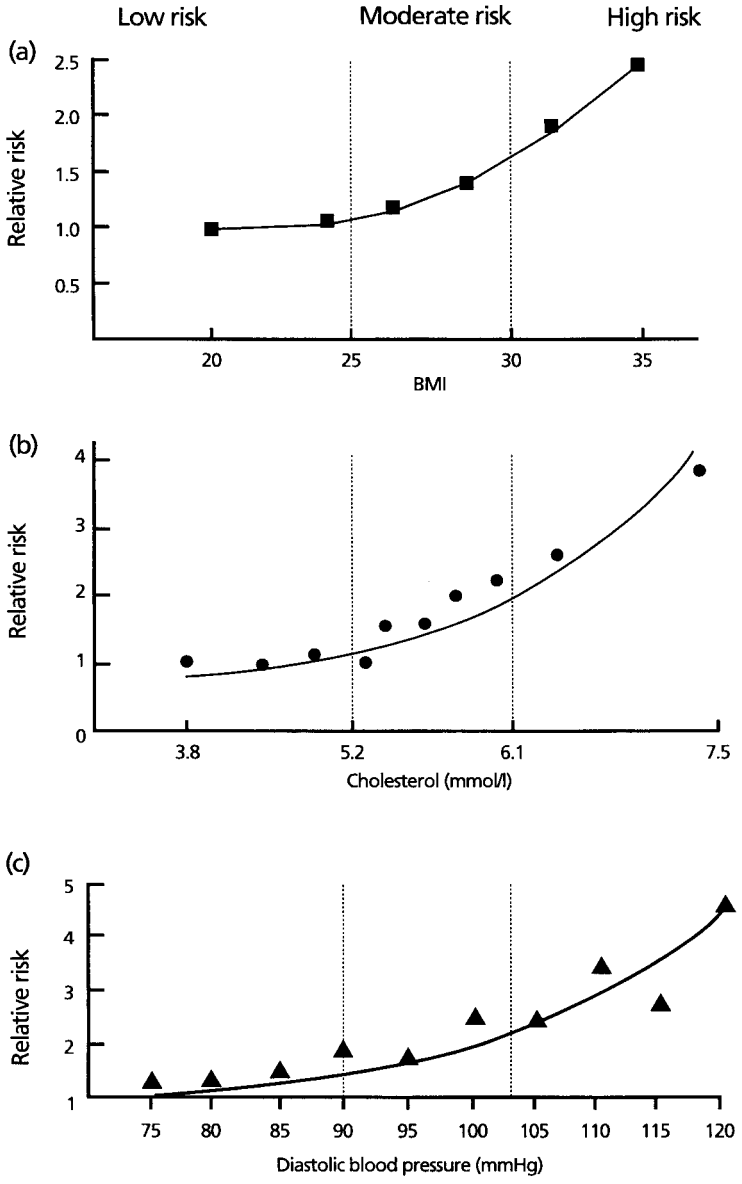
4.3 Difficulties in evaluating the health consequences of obesity

Most of the evidence linking health problems with obesity comes from prospective and cross-sectional population-based studies, although there is additional information from community interventions and clinical trials. Some confusion over the consequences of excess weight may arise because studies have used different BMI cut-off points for defining obesity, and because the presence of many medical conditions involved in the development of obesity may confound the effects of obesity itself.

Specific problems in evaluating the health consequences of obesity include:

Figure 4.1

Relationship between (a) BMI, (b) cholesterol and (c) diastolic blood pressure and relative risk of mortality[a]



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[a] Adapted from reference *2* with the permission of the publisher. Copyright John Wiley & Sons Ltd. Based on data from Stamler et al. (*3*, *4*) for the construction of the blood pressure and cholesterol plots, and from the Nurses' Health Study (*5*) for the BMI plot. There are similar continuous graded increases in the RR of mortality as BMI, blood pressure and cholesterol increase. However, the RR rises more rapidly for cholesterol and blood pressure than it does for BMI. The rise in the RR of mortality is notably steeper from BMI >30, cholesterol >6 mmol/litre, and diastolic blood pressure >100 mmHg (13.3 kPa).

- *The continuous relationship between gradations of excess weight and morbidity.* Individuals who have gained weight but still lie within the normal range will be assigned to a normal weight category even though they may be at increased risk of comorbidity because of excess weight gain.
- *Present health status and health behaviours (such as smoking).* These may have an impact on current weight and confuse its association with future health or even current well-being. For example, smoking is associated with a reduced BMI, so that the incidence of lung cancer caused by smoking appears to decrease with increased body weight.
- *The duration and design of epidemiological studies.* These will influence the strength of the association between weight and morbidity. Long-term monitoring is required to identify the range of health consequences of obesity, whereas studies of shorter duration with a large study population can be useful in identifying the major impact of obesity. Longer-term studies are also required where the outcome, e.g. cancer, is the result of a multistage process, with obesity having an effect on some but not necessarily all the stages. Most epidemiological studies measure prevalence rather than incidence, with the result that they are often confounded by survival bias and post-morbid modification of risk.
- *The age group studied.* This affects the relationship between obesity and health. For example, if the incidence of CHD in men is being analysed, obesity is a much more important predictor at younger than at older ages. The reverse is true, however, if total mortality is the end-point. The reason for this may be that obesity at an earlier age affects intervening risk factors much more strongly than in later life.
- *The use of initial weight criteria.* Most epidemiological studies adopt (by necessity) a static approach to classifying people by weight, i.e. subjects are generally placed in a weight group at the beginning of the study. The association with future illness or events is therefore based on that initial classification even if weight is subsequently gained or lost. This may give the impression that there is a risk-free zone up to BMI 27 or 28, which is misleading; weight gain independent of BMI is an important risk factor, as is the distribution of the fat gained.

4.4 Relative risk of obesity-associated health problems

The non-fatal but debilitating health problems associated with obesity include respiratory difficulties, chronic musculoskeletal problems, skin problems and infertility.

Table 4.1
Relative risk of health problems associated with obesity[a]

Greatly increased (relative risk much greater than 3)	Moderately increased (relative risk 2–3)	Slightly increased (relative risk 1–2)
NIDDM	CHD	Cancer (breast cancer in postmenopausal women, endometrial cancer, colon cancer)
Gallbladder disease	Hypertension	Reproductive hormone abnormalities
Dyslipidaemia	Osteoarthritis (knees)	Polycystic ovary syndrome
Insulin resistance	Hyperuricaemia and gout	Impaired fertility
Breathlessness		Low back pain due to obesity
Sleep apnoea		Increased risk of anaesthesia complications
		Fetal defects associated with maternal obesity

[a] All relative risk values are approximate.

The more life-threatening, chronic health problems associated with obesity fall into four main areas: (a) cardiovascular problems, including hypertension, stroke and CHD; (b) conditions associated with insulin resistance, e.g. NIDDM; (c) certain types of cancers, especially the hormonally related and large-bowel cancers; and (d) gallbladder disease.

It is important to recognize that ethnic differences have a bearing on the prevalence of a particular disease; some minority populations in the USA have a higher prevalence of certain obesity-related diseases (particularly NIDDM but, for black Americans, also CVD, stroke and osteoarthritis of the knee) compared with the white population (*6*). Nevertheless, although the absolute prevalence may vary, the relative risk of any particular disease (i.e. whether the risk is slightly, moderately or greatly increased for an obese person as compared with a lean person) is fairly similar throughout the world (Table 4.1).

4.5 Intra-abdominal (central) fat accumulation and increased risk

Compared with subcutaneous adipose tissue, intra-abdominal adipose tissue has:

— more cells per unit mass;
— higher blood flow;
— more glucocorticoid (cortisol) receptors;

— probably more androgen (testosterone) receptors;
— greater catecholamine-induced lipolysis.

These differences make intra-abdominal adipose tissue more susceptible to both hormonal stimulation and changes in lipid accumulation and metabolism. Furthermore, intra-abdominal adipocytes are located upstream from the liver in the portal circulation. This means that there is a marked increase in the flux of nonesterified fatty acid (NEFA) to the liver via the portal blood in patients with abdominal obesity.

There is good evidence that abdominal obesity is important in the development of insulin resistance (see section 4.8.1), and in the metabolic syndrome (hyperinsulinaemia, dyslipidaemia, glucose intolerance, hypertension) that links obesity with CHD (see section 4.8.2). Some non-Caucasian populations appear to be especially susceptible to this type of syndrome, in which lifestyle changes may play a particularly important etiological role (*7*).

Premenopausal women have quantitatively more lipoprotein lipase (LPL) and higher LPL activity in the gluteal and femoral subcutaneous regions, which contain fat cells larger than those in men, but these differences disappear after the menopause (*8*). In contrast, men show minimal regional variations in LPL activity or fat cell size. These differences may explain the tendency for premenopausal women to deposit fat preferentially in lower body fat depots. The higher level of intra-abdominal adipose tissue found in men compared with premenopausal women seems to explain, in part, the greater prevalence of dyslipidaemia and CHD in men than in premenopausal women.

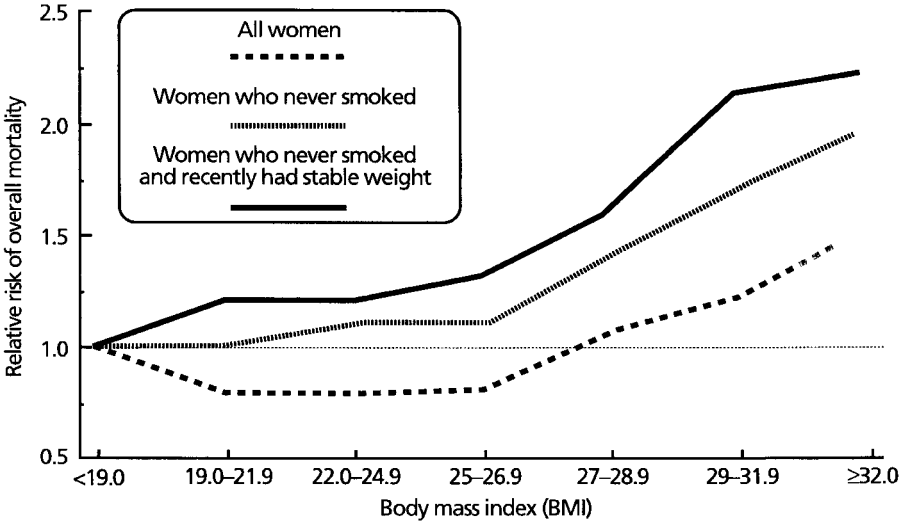
4.6 Obesity-related mortality

There has been much controversy about the relationship between obesity and mortality. While a number of studies have found a U- or J-shaped association, with higher mortality rates at both the upper and lower weight ranges, some have shown a gradual increase in mortality with increasing weight, while others have reported no association at all.

Many studies relating obesity and mortality have included biases in their design that have led to a systematic underestimate of the impact of obesity on premature mortality. These include the failure to control for cigarette smoking (producing an artificially high mortality in leaner subjects), inappropriate control for conditions such as hypertension and hyperglycaemia, which were assumed to be confounding

Figure 4.2

Relationship between BMI and relative risk of premature mortality[a]



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The relationship between BMI and all-cause mortality was examined using data from the Nurses' Health Study, which involved 115 195 middle-aged women. A total of 4726 deaths occurred during the 16-year follow-up. The apparent excess relative risks of mortality associated with leanness, suggested when the analysis included all women, were found to be artefacts as they were eliminated by accounting for smoking (leaving 1499 deaths) and subclinical disease (leaving 531 deaths). By excluding former and current smokers, women with BMI < 22 were found to have the lowest mortality among the remaining women. When disease-related health loss was also accounted for, the leanest women (BMI < 19) had the lowest mortality. This analysis is based on professional middle-aged women and so may not be representative of all population groups.

[a] Based on data from Manson et al. (*5*), with permission, and reproduced from Gill PG, Key issues in the prevention of obesity, *British Medical Bulletin*, 1997, **53**:359–388, with the permission of the publisher, Churchill Livingstone.

factors but are to a large degree the effects of obesity (hence some factorial analyses distort the true association between obesity and mortality), failure to control for weight loss associated with illness (leading to an underestimate of the impact of obesity on mortality), and failure to standardize for age (*9*, *10*).

The Nurses' Health Study (*5*) in the USA found that, when biases are removed from the analysis, an almost linear, continuous relationship between BMI and mortality is found, with no specific lower threshold (see Fig. 4.2). This is not surprising, given the largely linear relationship between body weight and conditions such as CHD, hypertension and NIDDM when BMI increases from 20 to 30 (*11–13*). Similar results and conclusions have been reached by others (*10*, *14*) but a

follow-up study of NHANES has continued to show U-shaped curves after control of the pertinent variables. Nevertheless, whatever the shape of the curve, it appears that the lowest mortality risk is associated with a BMI between 18 and 25. This conclusion was reached by the American Institute of Nutrition (*15*) after analysing numerous studies of obesity and mortality risk.

Although the increase in mortality rate with increased relative body weight is steeper for both men and women under age 50, the effect of overweight on mortality persists well into the ninth decade of life. The increased risk observed in younger people is linked to the duration of overweight, so that a special effort should be made to control the weight of younger adults (*14*, *16*, *17*).

Finally, if obesity is associated with an increased risk of premature mortality, it may seem paradoxical that obesity rates are rising in many countries at a time when overall death rates in these same countries are actually falling. However, the decline in overall death rates is essentially the consequence of the reductions in CVD. These, in turn, are the result largely of falling rates of smoking and the improvement in dietary quality (higher intake of fruits and vegetables and reduced intake of salt, saturated fat and cholesterol). The incidence of NIDDM, however, is increasing, and there is evidence that this is a consequence of the rise in the prevalence of obesity. Increased obesity cannot be completely explained by reduced rates of smoking, which appear to be associated with only small increases in the average body weight of the population. The expected effect over time is an increase in mean BMI worldwide that will lead to a further increase in NIDDM, gallbladder disease, hypertension and atherosclerosis. Although these effects may not be reflected in overall mortality rate figures, they will surely lead to a higher frequency of the debilitating and prolonged morbidity from NCDs that require expensive health care.

4.7 Chronic diseases associated with obesity

4.7.1 *Cardiovascular disease and hypertension*

Cardiovascular disease

CVD encompasses CHD, stroke and peripheral vascular disease. CHD and stroke account for a large proportion of deaths in men and women in most industrialized countries, and their incidence is increasing in developing countries.

Obesity predisposes an individual to a number of cardiovascular risk factors including hypertension, raised cholesterol and impaired glucose tolerance. However, longer-term prospective data now suggest

that obesity is also important as an independent risk factor for CHD-related morbidity and mortality (*18*). The Framingham Heart Study ranked body weight as the third most important predictor of CHD among males, after age and dyslipidaemia (*19*). Similarly, in women, a large-scale prospective study in the USA found a positive correlation between BMI and the risk of developing CHD. Weight gain substantially increased this risk (*20*). These findings are consistent with data from other countries. A 15-year follow-up study of 16000 men and women in eastern Finland concluded that obesity is an independent risk factor for CHD mortality in men and contributes to the risk of CHD in women (*21*).

On the basis of the Framingham Heart Study and other studies, it can be concluded that the degree of overweight is related to the rate of development of CVD (*22*). The CHD risk associated with obesity is higher in younger age groups and also in people with abdominal obesity than in those with excess fat around the hips and thighs (*23*) (see section 4.5). In addition, mortality from CHD has been shown to be increased in overweight individuals, even at body weights only 10% above the average (*24*).

Interestingly, Asian Indians have the highest rates of CHD of any ethnic group studied, despite the fact that nearly half this group are lifelong vegetarians. CHD occurs at an early age and generally follows a severe and progressive course. Although the prevalence of classic risk factors is relatively low, there is a substantial prevalence in this population of high triglyceride and low high-density lipoprotein (HDL) cholesterol levels, high lipoprotein (a) levels, hyperinsulinaemia and abdominal obesity (*25*). These appear to constitute weight-related risk factors in this population that may, in particular, reflect the central distribution of body fat.

Hypertension and stroke

The association between hypertension and obesity is well documented. Both systolic and diastolic blood pressure increase with BMI, and the obese are at higher risk of developing hypertension than lean individuals (*4*, *26*). Community-wide surveys in the USA (NHANES II) show that the prevalence of hypertension in overweight adults is 2.9-fold higher than that for non-overweight adults (*27*). The risk in those aged 20–44 years is 5.6 times greater than that in those aged 45–74 years (*28*), which in turn is twice as high as that for non-overweight adults (*29*). The risk of developing hypertension increases with the duration of obesity, especially in women, and weight reduction leads to a fall in blood pressure (see section 5.3.1).

A 1.00kPa (7.5mmHg) difference in diastolic pressure within the range 70–110mmHg (9.33–14.7kPa) is accompanied by a 29% difference in CHD risk and a 46% difference in the risk of stroke, irrespective of sex, age group or ethnicity (*30*).

While many large studies have examined the relationship between obesity and CHD, there has not been the same emphasis on stroke. One study in Honolulu, in which 1163 non-smoking men aged between 55 and 68 years were examined, found that elevated BMI was associated with increased risk of thromboembolic stroke (*31*). However, preliminary results obtained from women in the Swedish Obese Subjects (SOS) study were not conclusive (*32*). Other studies found that a high WHR rather than BMI was the risk factor associated with stroke and that this relationship was stronger than for any other anthropometric variable tested (*33*, *34*). It was suggested that a lifelong history of obesity rather than weight in middle age is more important in assessing risk of stroke (*13*).

The reason for the association between increased body weight and elevated blood pressure is unclear. One possibility is that obesity is associated with higher circulating levels of insulin (a consequence of insulin resistance) and consequently with enhanced renal retention of sodium, resulting in increased blood pressure (*35*). As exercise is known to improve insulin sensitivity, this would perhaps explain why exercise also reduces blood pressure. Other possible etiological factors include elevated plasma renin or enhanced catecholamine activity (*36*).

4.7.2 *Cancer*

A number of studies have found a positive association between overweight and the incidence of cancer, particularly of hormone-dependent and gastrointestinal cancers (Table 4.2).

Greater risks of endometrial, ovarian, cervical and postmenopausal breast cancer have been documented for obese women, while there is some evidence for an increased risk of prostate cancer among obese men. The increased incidence of these cancers in the obese is greater in those with excess abdominal fat and is thought to be a direct consequence of hormonal changes (*37*). The incidence of gastrointestinal cancers, such as colorectal and gallbladder cancer, has also been reported to be positively associated with body weight or obesity in some but not all studies, and renal cell cancer has consistently been associated with overweight and obesity, especially in women (*38*, *39*).

In addition to overall obesity, intra-abdominal fat distribution and adult weight gain have been independently associated with an

Table 4.2
Cancers with a higher reported incidence in obese persons

Hormone-dependent	Gastrointestinal/hepatic/renal
Endometrial	Colorectal
Ovarian	Gallbladder
Breast	Pancreatic
Cervical	Hepatic
Prostate	Renal

increased risk of breast cancer. For example, it has been reported that an increase in intra-abdominal fat accumulation increases the risk of postmenopausal breast cancer, independently of relative weight and particularly when there is a family history of the disease. Furthermore, weight gain during adulthood has consistently been associated with increased risk of breast cancer, even in cohort studies that showed no association between baseline relative weight and subsequent risk of breast cancer (*40*, *41*).

In one major prospective study, in which 750000 men and women were followed for 12 years, it was found that the mortality ratios[1] for any cancer were 1.33 and 1.55 for obese men and women, respectively (*42*). It should be noted, however, that in some studies of gastrointestinal and breast cancer, it has been difficult to determine whether it is the effect of dietary components that promote weight gain, such as a high fat content, or the effect of obesity per se that is important. Further research in this area is necessary.

High levels of physical activity have been shown to decrease the risk of colon cancer in men in the majority of studies, and in women in half the studies. However, the effect of physical activity on rectal cancer was not significant in most cases. Breast cancer and cancers of the reproductive system were less prevalent in women who had been athletes at college (*43*) compared with less active women. NHANES I data indicate that a high level of non-recreational activity is important in reducing the risk of cancer, but that there is only a weak relationship between recreational exercise and cancer, with the exception of prostate cancer (*44*).

4.7.3 *Diabetes mellitus*

A positive association between obesity and the risk of developing NIDDM has been repeatedly observed in both cross-sectional

[1] Ratio of premature deaths (<65 years) in a population with BMI ≥30 to premature deaths in a population with BMI <25.

(*45–57*) and prospective studies (*53, 58–66*). The consistency of the association across populations despite different measures of fatness and criteria for diagnosing NIDDM reflects the strength of the relationship. When women aged 30–55 years were monitored for 14 years, the additional risk of developing NIDDM for those who were obese was over 40 times greater than for women who remained slim (BMI <22) (*61*). The risk of NIDDM increases continuously with BMI and decreases with weight loss. Analysis of data from two recent large prospective studies illustrates the impact of overweight and obesity on NIDDM; about 64% of male and 74% of female cases of NIDDM could theoretically have been prevented if no one had had a BMI over 25 (*61, 66*).

Detailed analyses of the relationship between obesity and NIDDM have identified certain characteristics of obese persons that further increase the risk of developing this condition, even after controlling for age, smoking and family history of NIDDM. These include obesity during childhood and adolescence, progressive weight gain from 18 years, and intra-abdominal fat accumulation. In particular, intra-abdominal fat accumulation has been implicated as an independent risk factor for NIDDM in a variety of populations and ethnic groups around the world and, in some studies, has been an even stronger predictor of NIDDM than overall fatness (*52, 56, 60*).

Lack of physical activity and an unhealthy diet, both of which are associated with lifestyle in industrialized countries, are also important modifiable risk factors for overweight and obesity. The prevalence of NIDDM is 2–4-fold higher in the least physically active individuals compared with the most physically active (*67, 68*), an effect which is independent of the level of body mass, and a healthy diet can reverse the deterioration in glucose tolerance commonly seen with diets high in fat and low in carbohydrate and fibre (*69*).

Intra-abdominal fat accumulation, as well as obesity per se, are also associated with an increase in the risk of prediabetic conditions such as impaired glucose tolerance and insulin resistance. The benefits of weight loss in controlling NIDDM are discussed in section 5.

4.7.4 *Gallbladder disease*

In the general population, gallstones are more common in women and the elderly. However, obesity is a risk factor for gallstones in all age groups and, in both men and women, gallstones occur three to four times more often in obese compared with non-obese individuals, and the risk is even greater when excess fat is located around the abdomen. The relative risk of gallstones increases with BMI, and data from

the Nurses' Health Study suggest that even moderate overweight may increase the risk (*70*).

Supersaturation of the bile with cholesterol and reduced motility of the gallbladder, both of which are present in the obese, are thought to be factors underlying gallstone formation. Furthermore, since gallstones enhance the propensity to gallbladder inflammation, acute and chronic cholecystitis is also more common in the obese. Biliary colic and acute pancreatitis are other potential complications of gallstones.

Paradoxically, gallstones are also a common clinical problem in those losing weight (see section 5).

4.8 Endocrine and metabolic disturbances associated with obesity

4.8.1 *Endocrine disturbances*

Recent research has shown that adipocytes (fat cells) are more than just fat depots. They also function as endocrine cells, producing many locally and distantly acting hormones, and as target cells for a great many hormones. Altered hormonal patterns have been observed in obese patients, especially in those with intra-abdominal fat accumulation (*71*, *72*). Common hormonal abnormalities associated with intra-abdominal fat accumulation are listed in Table 4.3.

Insulin resistance

Sensitivity to insulin varies widely among any group of people, but insulin resistance is very often associated with obesity. It is especially pronounced with intra-abdominal fat accumulation and, since abdominal fat mass increases with increasing adiposity, is universally found in very severe obesity (BMI ≥40).

It has been suggested by some investigators that insulin resistance is an adaptation to obesity that tends to limit further fat deposition (*73*). In insulin resistance, the oxidation of fat tends to be favoured rather than its storage and the oxidation of glucose. Thus, if an individual

Table 4.3
Common hormonal abnormalities associated with intra-abdominal fat accumulation

Insulin resistance and increased insulin secretion
Increased free testosterone and free androstenedione levels associated with decreased sex hormone binding globulin (SHBG) in women
Decreased progesterone levels in women
Decreased testosterone levels in men
Increased cortisol production
Decreased growth hormone levels

who is gaining weight continues to eat the same amount, there will come a time at which net fat oxidation will, through insulin resistance, equal dietary fat intake and the individual will be in fat balance. A corollary, suggested by data from prospective studies (*74*), is that the more insulin resistant among a group of individuals of normal body weight will be protected from future weight gain. However, this is only a theory and is by no means universally accepted (*75*). In addition, insulin resistance is clearly maladaptive in terms of risk of CVD and other chronic diseases.

Insulin normally inhibits fat mobilization from adipose tissue and activates LPL. These are both metabolic processes that become insulin resistant in obesity. However, in contrast to the direct regulation of insulin secretion by plasma glucose concentration, the regulation of insulin secretion by fat metabolites is relatively weak. This means that oversecretion of insulin (due to insulin resistance) compensates for defects in glucose metabolism to a much greater degree than for defects in lipid metabolism. Disruption of the postprandial response by insulin leads to the dyslipidaemic state (section 4.8.2). Differential insulin resistance of specific organs or tissues may account for regional fat accumulation. For instance, the relative insulin sensitivity of intra-abdominal fat is thought to be required for central fat accumulation.

Physical activity improves insulin sensitivity through weight reduction and increased cardiorespiratory fitness. However, it also improves insulin sensitivity independently of these factors (*76*).

Hormones affecting reproductive function

Significant associations are seen in reproductive endocrinology between excess body fat, particularly abdominal obesity, and ovulatory dysfunction, hyperandrogenism and hormone-sensitive carcinomas (*77*). Changes in circulating sex hormones appear to underlie these abnormalities. Androstenedione and testosterone concentrations are commonly elevated whereas that of sex hormone binding globulin (SHBG) is reduced, while the plasma ratio of estrone to estradiol is also increased in obesity. A decrease in SHBG is associated with an increased clearance of free testosterone and estradiol, resulting in a disturbed sex hormone equilibrium.

Moderate obesity is frequently associated with polycystic ovary syndrome, which is the most common endocrine disorder of reproduction (*78*). Obesity contributes to or worsens, and weight loss generally improves, the associated hormonal abnormalities and menstrual function of obese women with polycystic ovary syndrome (*79*).

Adrenocortical function

Obese subjects have a normal circulating plasma cortisol concentration with a normal circadian rhythm, and normal urinary free cortisol. However, the cortisol production rate is increased in obesity to compensate for an accelerated rate of cortisol breakdown (*80*, *81*). Cortisol inhibits the antilipolytic effect of insulin in human adipocytes, an effect that may normally be particularly pronounced in abdominal fat because it contains a high density of glucocorticoid receptors. This mechanism may contribute to the manifestations of insulin resistance (*82*).

Studies have shown that patients with intra-abdominal fat accumulation have increased cortisol secretion, probably because they have increased activity of the hypothalamic–pituitary axis (HPA). Stress, alcohol and smoking have all been shown to stimulate the activity of the HPA (*83*).

4.8.2 *Metabolic disturbances*

Dyslipidaemia

Obese individuals are frequently characterized by a dyslipidaemic state in which plasma triglycerides are raised, HDL cholesterol concentrations are reduced and low-density lipoprotein apo B (LDL-apoB) levels are raised. This metabolic profile is most often seen in obese patients with a high accumulation of intra-abdominal fat and has consistently been related to an increased risk of CHD (*84*).

Excessive intra-abdominal fat accumulation is also associated with a greater proportion of small, dense low-density lipoprotein (LDL) particles. The high proportion of these small dense LDL particles may be the result of metabolic disturbances related to the accompanying high triglyceride or low HDL levels. Indeed, the hypertriglyceridaemic state may be the combined result of an increased production and a reduced breakdown of triglyceride-rich lipoproteins (*84*, *85*). This process results in lower HDL cholesterol levels and favours the triglyceride enrichment of LDL. The triglyceride-rich LDL is then enzymically degraded by hepatic lipase to produce small, dense LDL particles. A large proportion of these particles cannot be identified simply by the measurement of total or LDL cholesterol levels because these cholesterol levels are frequently in the normal range in obese individuals. A better indicator of small, dense LDL particle levels is an elevated ratio of LDL-apoB to LDL cholesterol.

Impaired fat tolerance (i.e. prolonged and/or exaggerated lipaemia following fat ingestion) is now also recognized as a component both of insulin resistance and of the atherogenic lipoprotein phenotype (*86*).

The metabolic syndrome and obesity

The common association of obesity with other CVD risk factors is well recognized. This clustering has been given several labels, including syndrome X and the insulin resistance syndrome, but the term metabolic syndrome is now favoured. There is no internationally agreed definition of the syndrome, but a suitable working definition would include two or more of the following:

— impaired glucose tolerance;
— elevated blood pressure;
— hypertriglyceridaemia and low HDL cholesterol;
— insulin resistance;
— central obesity.

Insulin resistance and/or hyperinsulinaemia have been suggested as the underlying cause(s) linking these conditions (*87*). Each individual component of the syndrome increases the CVD risk but, in combination, they interact to increase risk in a synergistic fashion.

Epidemiological studies confirm that the metabolic syndrome occurs commonly in a wide variety of ethnic groups including Caucasoids, Afro-Americans, Mexican Americans, Asian Indians and Chinese, Australian Aborigines, Polynesians and Micronesians. However, there is some evidence that the patterns of risk factors observed vary between and even within populations (*88*).

4.9 Debilitating health problems associated with obesity

Before chronic, life-threatening illness develops, overweight and obese patients usually present to primary care physicians with a range of conditions that adversely affect their quality of life, are often mechanical in origin, and are caused by the large amounts of excess weight that have to be carried. Though often perceived as less serious, these conditions are nonetheless debilitating and sometimes painful; they may also be costly in terms of the health resources consumed in their treatment and the absences from work that they cause. Sleep apnoea can have fatal consequences associated with cardiac arrhythmias. Unfortunately, few data are available on the economic costs of these conditions attributable to obesity.

4.9.1 *Osteoarthritis and gout*

Obesity is associated with the development of osteoarthritis and gout and, in obese middle-aged women at or after menopause, pain at the medial aspect of the knee (adiposa dolorosa juxta-articularis). Possible factors underlying the relationship between obesity and osteoarthritis include mechanical stresses related to the increased load

carried by the obese, metabolic changes associated with increased fatness, and dietary elements (e.g. high fat content) related to the development of obesity. The data indicate that mechanical damage is usually the cause. The increased risk of gout associated with obesity may be related to the accompanying hyperuricaemia, although central fat distribution may also be involved, particularly in women (*89–91*).

4.9.2 *Pulmonary diseases*

Obesity impairs respiratory function and structure, leading to physiological and pathophysiological impairments. The work of breathing is increased in obesity, mainly as a result of the extreme stiffness of the thoracic cage consequent on the accumulation of adipose tissue in and around the ribs, abdomen and diaphragm (*92*). Hypoxaemia is common, partly because the low relaxation volume causes ventilation to occur at volumes below the closing volume (*93*, *94*), and is exacerbated when lying down because of the reduced functional residual capacity (*95*).

Sleep apnoea occurs in more than 10% of men and women with a BMI of 30 or above, and 65–75% of individuals with obstructive sleep apnoea are obese. In one study, sleep apnoea occurred in 77% of those with a BMI above 40. In addition to BMI, however, obstructive sleep apnoea is related to central obesity and to neck size, probably as a result of the narrowing of the upper airway when lying down. The nocturnal disruption of sleep is associated with daytime somnolence, hypercapnia, morning headaches, pulmonary hypertension and, eventually, right ventricular failure (*96*, *97*).

4.10 Psychological problems associated with obesity

The SOS study found that the proportion of individuals receiving pensions for medical reasons was more than twice as high in obese patients as in population controls. Psychological problems in the obese (with women more affected than men) were found to be worst in those who were also chronically ill or injured, e.g. suffering from rheumatoid arthritis, cancer or spinal injury (*98*). The true social and economic costs of the non-fatal health consequences of obesity may therefore be seriously underestimated.

Other data on the psychosocial aspects of obesity relate mainly to the USA, and reflect cultural differences that may be irrelevant to other countries, especially as there appear to be ethnic differences in attitudes towards obesity. Black women in the USA, for instance, are 2–3 times more likely than white women to be obese, yet black women

have been shown to experience less social pressure to reduce their weight, start dieting later in life, and be significantly less likely to diet at each developmental milestone (*99*). Nevertheless, as the prevalence of obesity rises in developing countries, and populations are increasingly affected by the cultural values prevailing in industrialized countries, psychosocial problems are likely to become an increasingly common feature of the overall health profile of the obese.

It is important to note that the mechanisms leading to impaired psychological health are different from those underlying physical illness. The psychosocial problems associated with obesity are not the inevitable consequences of obesity but rather of the culture-bound values by which people view body fat as "unhealthy" and "ugly". Stunkard & Sobal (*100*) noted that ". . . obesity does not create a psychological burden. Obesity is a physical state. People create the psychological burden."

4.10.1 *Social bias, prejudice and discrimination*

Obesity is highly stigmatized in many industrialized countries, in terms both of the perceived undesirable bodily appearance and of the character defects that it is supposed to indicate. Even children as young as 6 years of age describe the silhouette of an obese child as "lazy", "dirty", "stupid", "ugly", "liar" and "cheat" more often than drawings of other body shapes (*101*).

Obese people have to contend with discrimination. Analyses of large surveys have shown that, compared with their non-obese peers, those who are obese are likely to complete fewer years at school, and less likely to be accepted by prestigious schools or to enter desirable professions. Furthermore, overweight young women in the United Kingdom and the USA earn significantly less than healthy women who are not overweight or than women with other chronic health problems (*102*).

The negative stereotypes and attitudes of health professionals (including doctors, medical students, nutritionists and nurses) towards obesity are of particular importance. Awareness of these negative attitudes may make the obese reluctant to seek medical assistance for their condition (*103*). Doctors may be less interested in managing overweight patients, believing that they are weak-willed and less likely to benefit from counselling. British general practitioners were less likely to prescribe lipid-lowering agents to overweight people (or to smokers), and doctors explicitly stated that this was their policy (*104*). Although little has been done so far to improve the stereotypes and attitudes of health professionals, Wiese et al. (*105*) found that

educational intervention was associated with a more positive attitude to the obese among first-year medical students.

4.10.2 *Psychological effects*

Research in this area has produced inconclusive results. Scores on standard psychological tests have been shown to differ little, if at all, between obese and non-obese people, and the evaluation of self-esteem in obese children and adolescents has not given consistent results (*106*). However, the implication that obesity has no psychological consequences is in conflict with the experience of overweight individuals and with the literature, in which strong cultural bias and negative attitudes towards obese people are consistently reported. Friedman & Brownell (*107*) suggest that this "paradox" can be explained by the manner in which these first-generation studies have been conducted and that new studies should be carried out to examine risk factors within the obese population.

4.10.3 *Body shape dissatisfaction*

Many obese people have an altered body image, i.e. they see their bodies as ugly and believe that others wish to exclude them from social interaction. This occurs most often in young women of middle and upper socioeconomic status, among whom obesity is less prevalent, and in those who have been obese since childhood.

4.10.4 *Eating disorders*

Binge-eating disorder is a recognized psychological condition (*108*) that occurs with increased frequency among obese persons, approximately 30% of whom seek medical help in dealing with it. In particular, the disorder is associated with severe obesity, a high frequency of weight cycling, and pronounced psychiatric comorbidity. It is characterized mainly by uncontrolled binge-eating episodes, usually in the early evening or at night.

Obese binge-eaters have worse moods and more severe psychological problems than obese people who do not binge-eat, and are more likely to drop out of weight-control programmes based on behaviour modification. Although binge-eaters may regain weight faster than non-binge-eaters, both short- and long-term weight loss among binge-eaters and non-binge-eaters appears to be similar (*109*).

The night-eating syndrome is characterized by the consumption of at least 25% — although more recent opinion suggests up to 50% — of total energy intake after the evening meal. This syndrome seems to be more common in morbidly obese patients and is related to sleep

disturbances such as sleep apnoea. It is thought to be due to alterations in the circadian rhythm, affecting both food intake and mood.

Nocturnal sleep-related disorder is a newly delineated night-eating pattern characterized by eating on arousal from sleep. It may be a variant of binge-eating disorder but its relationship with night-eating syndrome is unclear.

There is no clear evidence that these eating disorders are the primary cause of weight gain. It has been suggested that the increasing incidence of eating disorders is associated with the psychological pressure to slim (*110*, *111*). The fact that these disorders do not exist in societies where obesity is accepted as normal strongly supports the view that they have a cultural basis. Once established in patients, however, they are serious medical conditions and are difficult to cure.

4.11 Health consequences of overweight and obesity in childhood and adolescence

4.11.1 *Prevalence*

Obesity-related symptoms in children and adolescents include psychosocial problems, increased CVD risk factors, abnormal glucose metabolism, hepatic–gastrointestinal disturbances, sleep apnoea and orthopaedic complications (Table 4.4).

The most important long-term consequence of childhood obesity is its persistence into adulthood, with all the associated health risks. Obesity is more likely to persist when its onset is in late childhood or adolescence and when the obesity is severe (*112*, *113*). Overweight in adolescence has also been shown to be significantly associated with long-term mortality and morbidity (*114*).

Table 4.4
Health consequences of childhood obesity

High prevalence	Intermediate prevalence	Low prevalence
Faster growth	Hepatic steatosis	Orthopaedic complications
Psychosocial problems	Abnormal glucose metabolism	Sleep apnoea
Persistence into adulthood (for late-onset and severe obesity)	Persistence into adulthood (depending on age of onset and severity)	Polycystic ovary syndrome
		Pseudotumour cerebri
		Cholelithiasis
		Hypertension
Dyslipidaemia		

4.11.2 *Psychosocial effects*

The most common consequence of obesity in children in industrialized countries is poor psychosocial functioning. Preadolescent children associate the shape (or silhouette) of an overweight body with poor social functioning, impaired academic success and reduced fitness and health (*115*), as well as with character defects (see p. 56). However, there is little evidence to suggest that self-esteem is significantly affected in obese young children (*106*, *116*).

Among teenagers, however, cross-sectional studies consistently show an inverse relationship between body weight and both overall self-esteem and body image (*106*). A marked self-awareness of body shape and physical appearance develops during adolescence so that it is perhaps not surprising that the pervasive, negative social messages associated with obesity in many communities have a major impact at this stage. Overweight in adolescence may also be associated with later social and economic problems. A large prospective study conducted in the USA has shown that women who were overweight during adolescence and young adulthood were more likely to have lower family incomes, higher rates of poverty and lower rates of marriage than women with various other forms of chronic physical disability during adolescence (*102*).

4.11.3 *Cardiovascular risk factors*

Dyslipidaemia, hypertension and insulin resistance are frequently seen in obese children (*117*, *118*) and dyslipidaemia appears to be related to increased abdominal fat accumulation (*119*). Caprio and coworkers (*120*) suggest that insulin resistance in children may also be associated with abdominal obesity.

Although NIDDM is very rare, it accounts for one-third of all new cases of diabetes seen in some institutions in the USA (*121*).

Elevated serum lipid and lipoprotein levels, blood pressure and plasma insulin in childhood are all carried over into young adulthood, obesity status in childhood at baseline being a significant predictor of adult values (*122*, *123*).

4.11.4 *Hepatic and gastric complications*

Hepatic complications in obese children have been reported, particularly hepatic steatosis characterized by raised serum transaminase levels (*124*). Abnormal liver enzymes may be associated with cholelithiasis, but this condition is rare in children and adolescents.

Gastro-oesophageal reflux and gastric emptying disturbances, which affect a minority of obese children, may be a consequence of raised intra-abdominal pressure due to increased abdominal fat.

4.11.5 *Orthopaedic complications*

It is well documented that obese children can suffer from orthopaedic complications. The more serious of these include slipped capital femoral epiphysis (*125*) and Blount disease (a bone deformity resulting from overgrowth of the tibia) (*126*, *127*), while more minor abnormalities include knock knee (genu valgum) and increased susceptibility to ankle sprains.

4.11.6 *Other complications of childhood obesity*

Other serious complications to have been reported in obese children include obstructive sleep apnoea and pseudomotor cerebri. Obstructive sleep apnoea can cause hypoventilation and even sudden death in severe cases (*128*, *129*). Pseudomotor cerebri is a rare condition linked to raised intracranial pressure; it requires immediate medical attention.

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5. Health benefits and risks of weight loss

5.1 Introduction

While the effects of obesity on the functioning, health, and quality of life of obese subjects have been studied in great detail, the impact of weight loss is less well documented. Short-term studies have demonstrated clear benefits from modest weight loss on most of the associated consequences of obesity but there are very few well designed studies on the benefits of long-term weight loss.

The health benefits and risks of weight loss and of maintaining the new lower weight in the long term are considered here with particular reference to mortality, general health, and obesity-related comorbidities including chronic diseases, endocrine and metabolic disturbances, and poor psychosocial functioning. Two distinct hazards of weight loss, namely gallstones and reduced bone density, are also considered, as is weight cycling. Finally, a brief account is given of the effects of weight loss in obese children and adolescents.

The following should be noted:

- Well designed studies of the effects of long-term (>2 years) weight loss are few in number. Difficulties associated with such studies include that of maintaining long-term weight loss, and the need to distinguish intentional from unintentional weight loss.
- Intentional weight loss results in marked improvements in NIDDM, dyslipidaemia, hypertension, cardiovascular risk and ovarian function. There are also improvements in breathlessness, sleep quality, sleep apnoea, back and joint pain, and osteoarthritis.
- The only distinct hazards of weight loss are an increased incidence of gallstones (when weight loss is rapid) and possibly a reduction in bone density.

5.2 Problems in evaluating the effects of long-term weight loss

Problems in evaluating the benefits of long-term weight loss include:

— the difficulty of maintaining weight loss in adults over a long period;
— whether weight cycling is taken into account, and how it is defined when the outcome of a study is assessed;
— distinguishing "unintentional" weight loss, which may reflect underlying disease, from "intentional" weight loss;
— distinguishing the beneficial effects of weight loss *per se* from those of the changes in diet and physical activity necessary to achieve it.

The distinction between intentional and unintentional weight loss is of major importance in studies of the relationships between weight loss and morbidity or mortality. If weight loss occurs unintentionally as a result of underlying disease or serious illness, the association between weight loss and morbidity or mortality will be artificially increased. A bias resulting from misclassification may also occur if only two weight measurements are made, especially if weight loss is temporary and due to a minor acute illness. For this reason it is recommended that a minimum of three — and preferably more — weight measurements should be made throughout the study period.

5.3 Weight loss and general health

5.3.1 *Modest weight loss*

Data from a number of studies have shown that modest weight loss (defined as a weight loss of up to 10%) improves glycaemic control, and reduces both blood pressure and cholesterol levels (*1*). Modest weight loss also improves lung function and breathlessness, reduces the frequency of sleep apnoea, improves sleep quality, and reduces daytime somnolence. However, the degree of improvement often depends on the length of time that the condition has been present. Modest weight loss will also alleviate osteoarthritis, depending on the degree of structural damage, as well as back and joint pain.

5.3.2 *Extensive weight loss*

Following vertical-banded gastroplasty, severely obese patients who lose 20–30kg in weight, at a rate of 4.5kg per month for the first 6 months, gain substantial health benefits. They show a marked fall in blood lipids within the first 2 years of follow-up, and the condition of 43% of hypertensive patients and 69% of NIDDM patients is improved. Furthermore, at the population level, the incidences of hypertension, hyperlipidaemia and NIDDM are reduced to about one-sixth of those seen in obese patients who maintain their excess weight (*2*, *3*).

5.4 Weight loss and mortality

Unfortunately, most studies on weight loss and mortality have not controlled for unintentional weight loss or for cigarette smoking. In one large study of overweight white women in the USA in which these variables were evaluated, intentional weight loss consistently reduced mortality in women with obesity-related comorbidities such as NIDDM or CVD. However, the effects in women without comorbidities were not consistent with an association between intentional weight loss and reduction in mortality. Thus the benefit of

intentional weight loss was best seen in those of poorer health status (*4*).

In a randomized controlled dietary intervention trial of post-infarct patients in India, the effect of dietary intervention on cardiac mortality was greatest among patients who had also lost around 10% of their body weight (*5*). Further longer-term, well controlled studies are thus clearly needed to define accurately the beneficial effects of weight loss on mortality.

5.5 Impact of weight loss on chronic disease, and on endocrine and metabolic disturbances

5.5.1 *Cardiovascular disease and hypertension*

A number of cardiovascular risk factors related to blood clotting (haemostatic, rheological and fibrinolytic) have been associated with overweight (*6–8*). In particular, coagulation factors VII and X, which are directly associated with BMI, are involved in thrombosis (*9*) and increased risk of myocardial infarction (*10*). Weight loss in overweight subjects has been shown to reduce red blood cell aggregation and to improve fibrinolytic capacity.

Weight loss induces a fall in blood pressure. Short trials lasting a few weeks show that each 1% reduction in body weight leads, on average, to a fall of 1 mmHg (0.133 kPa) systolic and 2 mmHg (0.267 kPa) diastolic pressure (*11–14*). Marked falls in blood pressure can occur with very-low-energy diets, although modest dietary restrictions are also beneficial. Antihypertensive drug therapy, reducing a high alcohol intake, and lowering both dietary salt intake (*15*, *16*), and saturated fat intake (*17*, *18*) all contribute to further blood-pressure reduction independently of weight loss. It is estimated that a 10-kg weight loss can produce a fall of 10 mmHg (1.33 kPa) in systolic blood pressure and of 20 mmHg (2.67 kPa) in diastolic pressure (*19*).

Longer trials, with a 10-year follow-up of patients identified originally as mildly hypertensive, show that positive dietary change, together with smoking cessation and an increase in isotonic exercise (e.g. running), reduces both body weight and blood pressure. These levels can be sustained for 10 years and the need for drug therapy is significantly reduced (*12*).

5.5.2 *Diabetes mellitus and insulin resistance*

Studies of weight loss in NIDDM patients have consistently shown that a weight reduction of 10–20% in obese individuals with NIDDM results in marked improvements in glycaemic control and insulin sensitivity. These improvements can last from 1 to 3 years even if the

weight is subsequently regained. In the 75% of newly diagnosed NIDDM patients who are overweight, a 15–20% weight loss in the first year after diagnosis seems to reverse the elevated mortality risk of NIDDM (*20*). However, not all NIDDM patients respond to weight loss with metabolic improvements: the loss of abdominal adipose tissue may be more important in improvements in diabetic control than loss of weight per se.

Hyperglycaemia frequently decreases as soon as a low-energy diet is initiated, suggesting that dietary energy restriction has a beneficial effect independently of weight loss. Exercise training also improves glucose tolerance and insulin sensitivity independently of weight loss. The American Diabetes Association (*21*) recommends that aerobic exercise should be performed at moderate intensity for 20–45 minutes, 3 days per week. However, although epidemiological studies have emphasized the value of vigorous activity, mainly because it is easy to assess, total energy expenditure may be the important factor in limiting NIDDM rather than periods of intense physical activity (*22*).

5.5.3 *Dyslipidaemia*

The levels of blood lipids associated with obesity, namely high triacylglycerides, high cholesterol and low HDL cholesterol, can also be expected to return to normal after modest weight loss. For every 1 kg lost, LDL cholesterol has been estimated to decrease by 1% (*23*).

A 10-kg weight loss can produce a fall of 10% in total cholesterol levels, a 15% decrease in LDL levels, a 30% decrease in triacylglycerides and an 8% increase in HDL cholesterol (*19*). In addition, it has been found that serum triglyceride and HDL cholesterol levels show the most favourable changes after weight loss in those with a high waist:hip ratio (*24*).

5.5.4 *Ovarian function*

A weight loss of 5% or more during dietary treatment can improve insulin sensitivity and ovarian function in overweight and obese women with hirsutism and polycystic ovaries (*25*). In some obese women with amenorrhoea, normal menstrual function may be restored after weight loss (*26*).

5.6 Weight loss and psychosocial functioning

Most studies on the quality of life of obese patients before and after weight loss have been conducted on patients following surgery for obesity, and all show dramatic improvements in the overall quality of

life. The SOS study in Sweden (*27*), for example, showed significant improvements in social interaction, anxiety, depression and mental well-being that were sustained for 2 years after surgery for obesity. Although it is unclear whether these improvements will be seen with modest weight loss following non-surgical intervention, Klem et al. (*28*) recently reported that formerly obese subjects who had lost weight through diet and/or exercise modification found their quality of life to be substantially improved. While this is based on self-reporting by individuals who were maintaining weight losses of at least 13.6 kg for periods of over 1 year, it provides additional evidence of the benefits of weight loss.

Dieting is often perceived to have untoward psychological effects, including depression, nervousness and irritability. However, studies have shown that weight loss is associated with a decrease in depression score, particularly when it is achieved by behaviour modification (*29*, *30*).

A dramatic example of how extremely overweight individuals perceive their disorder has been provided by studies of a group of severely obese patients before and after losing weight as a result of gastric surgery (*31*, *32*). Before surgery, all the patients felt unattractive and the great majority felt that people talked about them behind their backs at work. They also felt that they had been discriminated against when applying for jobs and treated disrespectfully by the medical profession. After having achieved a weight loss of 50 kg, all the patients said that they would prefer to be deaf, dyslexic or diabetic or to suffer from severe heart disease or acne than to return to their previous weight. Given a hypothetical choice, they all preferred to be of normal weight than have "a couple of million dollars" — a choice that they made in less than a second.

5.7 Hazards of weight loss

Weight loss from "crash" dieting may result in acute attacks of gout. However, for intentional and controlled weight loss resulting from medical intervention, only two distinct hazards have emerged from a variety of prospective studies:

- *Gallbladder disease.* Women who lose 4–10 kg have a 44% increased risk of clinically relevant gallstone disease, and greater weight loss increases this risk. Mobilization of cholesterol from adipose tissue stores is increased during weight loss, so that the risk of supersaturation of bile with cholesterol is greater than when weight is stable. Premenopausal women are at particular risk because of an estrogen-induced enhanced biliary secretion of cholesterol.

- *Reduced bone density.* Bone density is typically increased in obese patients and reduced after weight loss. In white women, weight loss beginning at age 50 was found to increase the risk of hip fracture (*33*). Whether there is restitution of bone mass with weight regain following slimming, however, is uncertain; Compston et al. (*34*) found this to be the case whereas Avenell et al. (*35*) did not. There is little information on the impact of weight cycling on bone density.

It should also be noted that, in societies in which overweight and obesity are seen as a sign of affluence, weight loss may be interpreted as an indication of financial disaster.

5.8 Weight cycling

Weight cycling refers to the repeated loss and regain of weight that can occur as a result of recurrent dieting. However, there is no standard definition of weight cycling so that comparison between different studies is difficult (*36*).

It has been suggested that weight cycling is associated with negative health outcomes, makes future weight loss more difficult and results in a decrease in lean-to-fat tissue ratio (*37*). However, the evidence is conflicting; weight variability was associated with increased risk of CVD and all-cause mortality in men, particularly in those who continued to smoke, but the association between weight change and death was not seen in the heaviest men (*38*). Recently in the USA, the National Task Force on the Prevention and Treatment of Obesity (*39*) concluded that the evidence available at the time was that the increased risk was not sufficient to outweigh the potential benefits of moderate weight loss in obese patients.

5.9 Effects of weight loss in obese children and adolescents

Weight loss of only 3% significantly decreased blood pressure in obese adolescents, and blood pressure was further improved if exercise was added to the weight-loss programme (*40*). A weight loss of nearly 16% in obese children resulted in a parallel decrease in serum triacylglycerides and plasma insulin in the first year, with an increase in HDL cholesterol. These changes remained stable in the second year of the study; after 5 years, body weight was still 13% below the initial value, peripheral hyperinsulinaemia was reduced and HDL cholesterol remained higher (*41*).

The symptoms of hepatic steatosis in obese children eventually disappear when excess weight is lost (*42*).

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6. Economic costs of overweight and obesity

6.1 Introduction

The economic costs of overweight and obesity are important issues for health care providers and policy-makers alike. To date, there have been only a few attempts to quantify the economic burden of obesity-related morbidity and mortality. This is in marked contrast to smoking and alcohol consumption, where a large number of international studies have been undertaken to determine the magnitude of the economic burden that they impose on the community. In addition, few studies have assessed the relative cost-effectiveness of alternative interventions aimed at either preventing or treating obesity.

The limited information available on the economics of overweight and obesity is reviewed in this section. The use and limitations of cost-of-illness studies on obesity-related disease are summarized and the basic steps required in undertaking such a study are then outlined. A brief overview of the few studies in different countries that have provided estimates of the economic costs of obesity follows; key findings as well as the limitations of the methods used are highlighted, after which the cost-effectiveness of alternative interventions aimed at either preventing or treating obesity is reviewed. Finally, the implications of current understanding of the economics of obesity for public policy decision-making are considered and priorities for future research in this area discussed.

The following important points should be noted:

- The economic cost is made up of three main components:
  - "direct costs", i.e. the costs, to the individual and the service provider, associated with treating obesity itself;
  - the "opportunity cost" to the individual, i.e. the social and personal loss associated with obesity, generally arising from premature death or attributable morbidity;
  - "indirect costs", usually measured as lost production due to absenteeism from work and to premature death.
- The economic impact of obesity-related disease is usually estimated from cost-of-illness studies. These are useful in the development of public health policy but their limitations should be recognized: intangible costs and many of the direct costs of disease management and prevention, especially those incurred outside the formal health care system, tend to be ignored. A number of studies have therefore focused on the impact of obesity on broader social and economic issues, including the frequency of long-term sick leave.

- The economic costs of obesity have been assessed in several developed countries and are in the range 2–7% of total health care costs. These are conservative estimates based on variable criteria but clearly indicate that obesity represents one of the largest items of expenditure in national health care budgets.
- Although there have been no studies of the economic impact of obesity in developing countries, the escalating economic burden of adult NCDs in such countries has already been recognized by a number of international agencies including WHO and the World Bank. The real costs of therapy in developing countries exceed those in developed countries because of the extra burden associated with the use of scarce foreign exchange to pay for imports of expensive equipment and drugs, as well as the need for the specialized training of staff. In view of the existing burdens of endemic deficiency disorders and infectious diseases, obesity prevention is not only crucial but also the only sensible approach to planning public health policies in developing countries.
- Preliminary data suggest that a large proportion of the economic costs of obesity can be avoided by efficient prevention or intervention strategies.

6.2 Cost-of-illness studies

"Cost-of-illness" or "disease-costing" is a technique used to estimate the financial impact of disease on a community. The economic costs of obesity include:

- *Direct costs*: the cost to the *community* resulting from the diversion of resources to the diagnosis and treatment of diseases directly related to obesity, as well as from the cost of obesity treatment itself (including the cost of providing health care services to patients and their families, and the cost of service providers).

- *Intangible costs*: the cost to the *individual* arising from the impact of obesity on quality of life generally and on health specifically.

- *Indirect costs*: the welfare and economic benefits lost to *other members of society* through a reduction in the goods and services produced i.e. the impact of the reduced quality of life of the obese individual on the productive potential available to the rest of society. These costs are usually measured as the production lost through work-related absenteeism and premature death.

Most cost-of-illness studies focus on measuring direct and indirect costs, while less attention is given to the more difficult task of quantifying the intangible costs.

6.2.1 *Uses of cost-of-illness studies*

Cost-of-illness studies are useful in the development of public policy because they can:

- Identify and analyse how resources are currently being allocated to different types of costs, services and diseases.
- Help to identify potential improvements in health status, in the case of a specific disease, that can be achieved by the application of effective prevention programmes, or to identify a risk factor for a disease. A knowledge of the incidence and prevalence of the disease, the consequent use of health services, and costs can allow a calculation of the potential savings to a community that can be achieved through effective prevention programmes, which may (or may not) be greater than the costs of prevention.
- Assist health planners to make comparisons between the relative economic burden of different diseases that may assist in setting priorities for prevention, if taken together with information on the costs and effectiveness of prevention strategies.
- Provide data on the cost side of the cost–effectiveness ratio for subsequent economic appraisal.
- Be used to demonstrate to policy-makers and politicians the magnitude of the health problem in financial terms.

6.2.2 *Limitations of cost-of-illness studies*

The major criticism of cost-of-illness studies is that they can be misused. A cost-of-illness study may indicate that a disease is costly to treat. It may also suggest that a disease has a high social cost relative to other diseases or social problems, implying that society would be relatively better off without it. While this is obviously true, it does not mean that a higher priority should be given to treating that disease. Treatment (or prevention) may be relatively ineffective or expensive, so that priority-setting should be based on the relative cost-effectiveness of interventions and not on the cost of the disease alone. This criticism is best explained by Davey & Leeder (*1*):

> *". . . Instead of answering the question, 'Where should I put the next health care dollar to achieve the greatest health gain?' cost-of-illness studies provide information only about the burden of illness. They concentrate on cost and say nothing about the effectiveness of treatment and value for money invested."*

Some economists have argued that, while cost-of-illness studies do not indicate where resources should be allocated in the short term, they do indicate where the greatest potential health improvements

and savings in health care resources could be made if effective interventions were available.

A further criticism concerns the focus of cost-of-illness studies on direct health care costs and the indirect costs of lost production, less emphasis being placed on the burden of disease, premature death and reduced quality of life. Because these latter intangible costs are less easy to measure in monetary terms, they tend to be ignored. Diseases associated with high health care costs but relatively low morbidity and mortality (such as dental disease) may therefore be seen as imposing a far greater burden than other diseases associated with high costs in terms of premature death and reduction in quality of life but low health care costs (such as youth suicide).

The definition of health care incorporated in cost-of-illness studies tends to be narrow and ignores many of the direct costs of disease management and prevention, especially those arising outside the formal health care system. This is particularly true of obesity, as the highest direct cost category is most likely to be the personal expenditure on weight-loss programmes incurred by overweight and obese individuals. The impact of the narrow range of direct costs included in studies is likely to vary across disease types and risk factors.

6.2.3 *Steps in undertaking a cost-of-illness study*

The following basic steps need to be taken in carrying out a cost-of-illness study on obesity-related disease where, in accordance with the WHO criteria, overweight is defined as BMI 25–29.9 and obesity as BMI $\geq$ 30:

- identify those diseases related to overweight and obesity;
- quantify the relationship between obesity and the associated disease morbidity and mortality using standard criteria (i.e. the population-attributable fractions (PAFs); for more information on PAFs, see below);
- identify the relevant economic cost categories to be estimated;
- quantify the total costs associated with diet-related disease;
- use the PAFs to apportion that share of total costs directly attributable to overweight and obesity;
- undertake a sensitivity analysis of key epidemiological and economic parameters (or assumptions) to provide a range of cost estimates.

Population-attributable fraction

The epidemiological statistic needed to quantify the direct relationship between a risk factor of interest and a disease (and thus quantify its associated economic costs) is the population-attributable fraction.

This has been defined as the proportion of total events (e.g. deaths or morbidity) in a population that could be prevented if a particular risk factor (e.g. obesity) could be eliminated.

The PAF reflects the overall impact of the morbidity and mortality associated with a factor (e.g. obesity) in the specified population. It can be interpreted from an etiological standpoint (causal outcomes attributed to a particular risk factor) or from a prevention standpoint (the maximum number of events that could be prevented). Many epidemiologists use the concept of "preventable proportion" as a useful generalization of the PAF concept.

Where only one category of exposure (e.g. obese or non-obese) is concerned, PAF is given by:

$$\text{PAF} = \frac{p(\text{RR}-1)}{1+p(\text{RR}-1)}$$

where p = prevalence of risk factor (e.g. obesity) in a population
RR = relative risk
= incidence of disease in an obese person (I_e) divided by the incidence of disease in a non-obese person (I_o) = I_e/I_o

PAF can be expressed either as a fraction or as a percentage. Thus a PAF of 0.73 means that 73% of the incidence of the disease could be eliminated by removal of the risk factor (or conversely, that the risk factor is responsible for 73% of the incidence of the disease).

A number of epidemiological studies have assessed the relative risk of specific diseases associated with excess body weight. Most have used BMI as the risk factor; in only a few studies has the risk of disease been quantified in terms of body fat distribution (e.g. by the use of the waist circumference). Such studies have shown a positive relationship between BMI and the development of CHD (*2–4*), hypertension (*5*), stroke (*6*), NIDDM (*2*), gallbladder disease (*7*), sleep apnoea (*8*), and a number of cancers including breast cancer (*9*, *10*) and colon cancer (*11*). In addition, further studies have shown a relationship between excess body weight and obstetric complications in women (*12*), progression of osteoarthritis (*13*), and rheumatoid arthritis (*14*).

There is a need for a comprehensive systematic review (e.g. a meta-analysis) to provide a clearer understanding of the relationships found in such studies between excess weight and the diseases. Once these data are available, relative risk estimates can be combined with country-specific overweight and obesity prevalence data to determine PAFs for use in cost-of-illness studies.

6.2.4 *The disability-adjusted life year*

An alternative to the cost-of-illness study for use in the economic evaluation of the consequences of obesity and overweight is the disability-adjusted life year (DALY) (*15*). This can provide estimates of the burden imposed by death and disability due to any disorder and makes it possible to compare populations in different geographical and social settings. Both the proportion of chronic diseases attributable to overweight and obesity and the costs of their management vary across populations and between social classes within populations. The use of a combined measure of the loss of life expectancy and prolonged morbidity in national, regional and global estimates of the economic effects of overweight and obesity is therefore desirable.

Obesity and overweight, in the same way as tobacco use, contribute to several NCDs. Thus, the total DALY loss attributable to obesity and overweight would represent the attributable fraction of the total loss of DALYs due to NCDs associated with excess body weight. A number of estimates of the attributable fraction associated with tobacco use have been made, thus facilitating national and regional comparisons. Efforts should therefore be made to generate similar estimates of the attributable fraction associated with obesity and overweight.

6.3 International estimates of the cost of obesity

6.3.1 *Studies in developed countries*

At present, the economic burden of obesity-related diseases has been estimated in only a few studies. Some of the data available for developed countries are reviewed below and summarized in Table 6.1. The scope and methodology of the various studies vary considerably in terms of the diseases costed, the definition of obesity, the cost

Table 6.1
Economic costs of obesity[a]

Country	Year	Study	BMI	Estimated direct costs	National health care costs
Australia	1989–1990	NHMRC (*16*)	>30	A$ 464 million	>2%
France	1992	Lévy et al. (*17*)	≥27	FF 12000 million	2%
Netherlands	1981–1989	Seidell & Deerenberg (*18*)	>25	NLG 1000 million	4%
USA	1994	Wolf & Colditz (*19*)	>29	US$ 45800 million	6.8%

[a] As defined by cut-off point of BMI.

categories used and the epidemiological assumptions as to the relationship between obesity and disease. This makes it difficult to compare costs across countries and to extrapolate the results from one country to another. The limited data available suggest that, as previously mentioned, some 2–7% of total health care expenditure in a country may be directly attributable to overweight and/or obesity.

Australia

The National Health and Medical Research Council (NHMRC) replicated the 1992 Colditz study (*20*), using the same obesity-related diseases and the same estimates of relative risk but applying Australian estimates of obesity prevalence (based on BMI >30). The NHMRC estimated the direct cost of obesity to be A$ 464 million (1989–1990), indirect costs amounting to an additional A$ 272 million. Hypertension and CHD combined accounted for approximately 60% of the total economic costs of obesity. For hypertension, the largest costs were those for medical services and pharmaceuticals, whereas for CHD, hospital costs and the indirect costs associated with premature mortality were the most significant (*16*).

As part of the total cost-of-obesity estimate, the NHMRC also estimated the costs of obesity treatment within the formal health care system in Australia. These accounted for approximately 10% of the total economic cost of obesity.

The estimate provided by the NHMRC should be considered conservative for the same reasons as the Colditz study in the USA. Of interest is the fact that, while the costs of obesity treatment within the health care sector amounted to less than A$ 80 million, a 1992 survey by the Consumer Advocacy and Financial Counselling Association of Victoria (*21*) estimated that 300000 consumers purchased a weight-loss programme in Australia each year from a variety of weight-loss centres, and that the industry turnover was in excess of A$ 500 million per annum. This shows that a substantial proportion of the economic cost of obesity is incurred outside the formal health care sector.

Finland

The impact of obesity on several indicators of health care utilization was assessed among 10000 adult Finns in the National Survey on Health and Social Security in 1987 (*22*). The costs of medicines, physician consultations and hospital inpatient stays increased with increasing BMI. The excess health care utilization was due mainly to an increased need for medication, the cost of which rose by about 12% when BMI increased from 25 to 40. On the basis of these data it was estimated that, if all Finns were of normal weight, the annual

savings would be of the same order of magnitude as if all smokers in Finland were to stop smoking permanently.

France

To estimate the direct cost of obesity-related diseases in France in 1992, Lévy et al. (*17*) identified the direct costs of personal health care, hospital care, physician services and drugs for diseases with a well established relationship with obesity. These included NIDDM, hypertension, hyperlipidaemia, CHD, stroke, venous thromboembolism, osteoarthritis of the knee, gallbladder disease and certain cancers. The proportion of these diseases attributable to obesity (defined by the cut-off point of BMI ≥27) ranged from about 25% for hypertension and stroke to about 3% for breast cancer. The direct costs of obesity were estimated to be almost 12000 million francs, or approximately 2% of total health care expenditure in 1992. The costs of hypertension represented 53% of the total direct costs of obesity.

Netherlands

The cost of the excess use of medical care and associated costs due to obesity in the Netherlands were estimated using the data on 58000 participants in the Health Interview Surveys carried out from 1981 to 1989 (*18*). The health care costs included those for consultations with general practitioners and medical specialists, hospital admissions and the use of prescribed drugs. Obese (BMI ≥30) and overweight (BMI 25–30) individuals were more likely to have consulted a general practitioner. The total general practitioner costs attributable to obesity/overweight were equivalent to 3–4% of the country's total general practitioner expenditure. For hospitalizations, the fraction attributable to obesity was 3% and for overweight 2%. The excess use of medications by obese and overweight people, however, was very striking: compared with the non-obese, obese individuals were 5 times more likely to use diuretics and 2.5 times more likely to take drugs for CVD. It was estimated from these data that the direct costs of overweight and obesity were about 4% of total health care costs in the Netherlands. This is of the same order of magnitude as the health care costs attributable to all forms of cancer.

While the study did not cover all potential cost categories relevant to obesity, it was the first cost estimate to include the impact of overweight, and this category accounted for about 48% of the total costs of excess weight gain.

United States of America

The first national study undertaken on the economic cost of obesity was that by Colditz (*20*) in the USA. The diseases included in the cost

estimate were NIDDM, CVD, hypertension, gallbladder disease, and colon and postmenopausal breast cancer. Obesity was defined as a BMI greater than 29. Total costs attributable to obesity in 1986, including both direct and indirect costs, were estimated to be US$ 39300 million, representing 5.5% of the overall costs of illness for the USA in that year. The PAFs used for particular diseases were NIDDM 0.57, CVD 0.19, hypertension 0.26, breast cancer 0.06 and colon cancer 0.02. However, the estimates of relative risks used by Colditz are currently being revised by a number of groups to bring PAF and economic cost estimates into line with agreed classification criteria for overweight and obesity.

Colditz's original estimate should be considered conservative because estimates for many obesity-related diseases and for several relevant economic cost categories were excluded. Colditz points out that the addition of musculoskeletal disorders to his estimate would have raised the figure to US$ 56300 million, or 7.8% of the cost of illness for the USA in 1986.

In 1994, Wolf & Colditz (*19*) published a revised estimate of the economic costs of obesity in the USA, extending the range of obesity-associated diseases included in the analysis and updating their calculations. They estimated that the total cost of obesity in 1990 was US$ 68800 million, of which US$ 45800 million was due to the direct cost of obesity-associated disease. The remaining US$ 23300 million was an estimate of the indirect costs of obesity due to lost productivity (about US$ 4000 million, or 25591480 annual workdays) and premature mortality from diseases associated with obesity (about US$ 19000 million). These figures should still be considered to be conservative.

6.3.2 *Studies on the broader economic issues*

Methods other than cost-of-illness studies have been used to determine the economic impact of obesity-related diseases, e.g. studies on the influence of obesity either on attainment of social class (see below) or on pension and disability payments.

It is important to note that indirect costs of disease relate to the loss of worker productivity due to worker absenteeism, staff turnover and reduced worker productivity as a result of obesity-related morbidity, together with lost earnings due to premature death from an obesity-related disease. A common misconception among health professionals is that sickness, unemployment and other social welfare benefits should be included in the indirect costs of diseases. Economists do not

include such benefits in cost-of-illness studies as they are viewed as a transfer payment from the tax-paying population to the recipients. There is a continuing debate among health economists about whether to include indirect costs in a study and how to measure these costs reliably.

Attainment of social class

Cross-sectional studies in many affluent societies show an inverse relationship between educational level and the prevalence of obesity. However, in addition to indications that low socioeconomic status leads to obesity, there are also indications that the reverse may also be true. Obese subjects may also be subject to economic disadvantages such as higher premiums for life insurance policies.

One study of Danish draftees showed that, after adjustment for parental social class, level of education and intelligence, fewer obese men attained a relatively high social class compared with non-obese men (*23*). Similarly, a prospective study of young women in the USA showed that those who were obese were less likely to marry, and had fewer years of schooling, as well as a lower income compared with non-obese women (*24*). These results are supported by those of a number of other prospective studies showing that obese young adults do not attain the same social class as their non-obese peers. Although such data should be interpreted with caution, it has been suggested that societal discrimination may limit the socioeconomic potential of the obese.

Frequency of long-term sick-leave

In the SOS study (*25*) in Sweden, the frequency of long-term sick-leave (over 6 months) was reported to be 1.4 and 2.4 times higher in obese men and women, respectively, compared with the general Swedish population. Similarly, the rate of premature disability pensions was reported to be increased by a factor of 1.5–2.8 among participants in the study. The total loss of productivity due to obesity was estimated to be about 7% of the total cost of losses of productivity due to sick-leave and disability pensions in Sweden.

Premature work disability

In a large prospective Finnish study (*26*), obesity was associated with a twofold increased risk of premature work disability in men and a 1.5-fold greater risk in women. Most of the premature pensions attributable to obesity were due to cardiovascular and musculoskeletal diseases. A quarter of all disability pensions for these diseases in women were solely attributable to overweight and obesity.

6.3.3 *Studies in developing countries*

Although there have not been any comparable studies of the economic impact of obesity in developing countries, both WHO and the World Bank have recently highlighted the increasing burden associated with the rapidly emerging adult NCDs in these countries (*15*, *27*), where they have now replaced infectious diseases as the leading cause of death. In developing countries, about 50% of deaths in 1990 were caused by NCDs, but by 2020 that proportion is expected to rise to almost 77%. In 1990, some 42% of deaths were attributed to infectious and reproductive conditions, while by 2020 that proportion is expected to decline to about 12%. In contrast, in developed countries 87% of deaths in 1990 were from NCDs and the proportion is expected to rise only slightly—to 90%—by 2020.

The treatment needs of the rapidly expanding urban populations and increasingly affluent middle classes in developing countries are already overwhelming many medical services. Furthermore, as previously mentioned, the real costs of therapy associated with NCDs in developing countries exceed those in developed countries; the need to use scarce foreign exchange to pay for imports of expensive equipment and drugs and for the training of specialized staff creates an extra burden.

In recent World Bank studies, e.g. in Chile (*28*), the burden of disease has been expressed in terms of numbers of DALYs lost. NCDs account for a 5- and 9-fold greater rate of premature death than communicable diseases in men and women, respectively, and 10- and 5-fold greater rates of disability. The numbers of DALYs lost in men are 15-fold, and in women 20-fold, greater for NCDs than for infections. So far, the burden of disease attributable to excess weight gain and obesity has not been calculated, but cancers impose a substantial burden as do NIDDM and CVD. There is thus a need in developing countries to apply the new economic methods of determining the proportion of these diseases attributable to excess weight gain so that the impact of one of the principal contributors to NCDs can be assessed.

6.3.4 *Conclusions*

International studies on the economic costs of obesity have shown that they account for between 2% and 7% of total health care costs, the level depending on the range of diseases and cost categories included in the analysis. The figures are based mainly on cross-sectional data, and should be considered a conservative estimate of the true cost of obesity-related diseases for a number of reasons:

- In most studies, only a limited number of obesity-related diseases have been costed.
- Most studies have excluded some relevant direct-cost categories from the analysis.
- In the majority of cases only the economic costs associated with obesity (BMI ≥30) have been included in the analysis. The inclusion of costs associated with overweight (i.e. BMI 25–29.9) would substantially increase the attributed cost because the number of overweight individuals in a community is generally greater by a factor of 3–4 than those who are obese; the economic cost of drug use, for example, was increased by 65% if the overweight category was included (*18*).

Although there have not been any comparable studies of the economic impact of obesity in developing countries, the real costs of therapy associated with NCDs in such countries are likely to exceed those in developed countries.

6.4 Economic costs and benefits of obesity treatment

6.4.1 *Analyses of obesity-control trials*

Unfortunately, very little information is available on the economic benefits of treatment, but some extrapolations can be made from the preliminary and early data from the large-scale SOS intervention study of 1743 obese men and women in Sweden (*25*).

After 2 years of follow-up, Sjöström and his colleagues found a number of benefits in the subjects who were surgically treated and who individually lost between 30kg and 40kg. Thus quality of life was markedly improved and several cardiovascular risk factors were substantially decreased. The prevalence of NIDDM — 13% in controls and 16% in the intervention group before treatment — decreased by 68% in the intervention group and by only 16% in the controls. In other words, two-thirds of NIDDM was "cured" by the obesity intervention. Furthermore, the incidence of NIDDM was only 0.5% in the intervention group as compared with 7% in the controls. A 4–5-fold risk reduction was observed in the development of hypertension, hypertriglyceridaemia and the lowering of HDL cholesterol. During 2 years of follow-up, the incidence of NIDDM was very low in the intervention group but 30-fold higher in controls. Data on other disease end-points are not yet available.

In order to try to estimate the economic consequences of this controlled study, the results of treatment and the associated costs may be compared with the estimated costs of non-treated obese subjects. If

NIDDM is taken as an example, the 14-fold risk reduction in the treatment group suggests that NIDDM was prevented to a large extent. In addition, two-thirds of patients with established NIDDM were "cured". Applying these results to the estimated cost of obesity-related NIDDM in France would decrease the total costs of obesity in that country by approximately 3%, while in the USA costs could be reduced by almost 20%. Similar calculations with respect to change in cardiovascular risk factors are not easy, but a large proportion of the obese subjects who would usually be eligible for treatment for hypertension and hyperlipidaemia would not need such treatment. In France this would result in a 25% reduction in costs.

Little published information is so far available on the potential impact of obesity treatment in the SOS trial on sick-leave and pensions, which constitute the other major component of the costs of obesity. This is difficult to evaluate because treated patients have not been followed up for a long enough period, but the initial data indicated that the number of lost working days increased more quickly in the controls than in the intervention groups (*25*). Furthermore, the marked improvements in the quality of life of treated patients are not only an important outcome in themselves but also suggest that other major benefits that would reduce health costs can be expected after longer follow-up.

However, it is important to include in the costs those of the intervention itself (i.e. surgery) and of the follow-up review. The actual cost of the surgical procedure is not available, but follow-up figures suggest that, in spite of the surgical intervention, the frequency of visits to a doctor was the same in controls and intervention subjects by the second year after surgery.

The SOS study is the only fully controlled, large-scale, long-term study of the effects of the radical treatment of obesity and substantial weight loss. The results of this study will provide valuable information on the medical and economic consequences of effective intervention in obesity within a limited period. Preliminary results are very promising.

6.4.2 *Potential cost savings associated with a reduction in the prevalence of obesity*

A small number of studies have provided estimates of the potential impact on health care costs of a reduction in the population prevalence of obesity.

In a study in the USA (*29*), obese patients with NIDDM were assigned to a 12-week weight-loss programme involving an 800-$kcal_{th}$

diet (1 $kcal_{th}$ = 4.18 kJ). Subjects lost an average of 15.3 kg over the 12 weeks, but at 1-year follow-up had regained 9.0 kg. The authors estimated that the average saving in prescription costs per subject over 1 year was US$ 442.80. While the study showed a significant prescription cost saving, sample sizes were small and the energy intake associated with the weight-loss programme was very small. It would thus be unwise to assume that these results would be reproduced under different conditions.

As an extension of a cost-of-obesity study discussed earlier in this section (see p. 84), the NHMRC estimated the potential annual saving to the Australian health care system that would result if the prevalence of obesity were reduced by 20% by the year 2000 (baseline 1989), as specified by the National Health Goals and Targets (*30*). The method used in this study was to recalculate the PAF based on the target prevalence of obesity (and on the assumption that relative risk estimates remain constant) for each obesity-related disease. The 1989–1990 estimated cost for each obesity-related disease was then multiplied by the change in PAF to estimate the potential annual saving. The NHMRC estimated that an annual saving of A$ 59 million in health care expenditure and a potential 2300 life-years could be gained if the obesity target was achieved.

While the NHMRC calculation shows the potential cost saving that might be achieved if the target obesity prevalence were achieved, it does not provide information on the public and private expenditure that would be required to fund programmes to achieve this target. The analysis therefore does not help decision-makers to decide whether investing scarce community resources in preventing or treating overweight and obesity represents an efficient use of such resources. Such decisions should be based on an evaluation of the costs and outcomes (effectiveness) of alternative interventions for the prevention and treatment of overweight and obesity.

6.4.3 *Cost–effectiveness of obesity prevention and treatment*

Few studies have addressed the economic evaluation of the prevention and treatment of overweight and obesity and, of these, most have been concerned with treatment rather than primary prevention. A limited number of studies on the cost-effectiveness of non-drug versus drug treatment of hypertension have been conducted and have included measurements of weight loss. In addition, a number of studies have focused on the financial benefits of workplace fitness programmes (including the benefit of weight loss) in reducing employee absenteeism, but the methods used in these studies have been criticized (*31*, *32*). Authors have sometimes overgeneralized and used

optimistic estimates of the health benefits of risk factor modification. In other studies, the relevant programme cost categories have been incorrectly specified, and rather dubious methods of valuing and measuring these costs have been used. The results seem in some instances to be biased in favour of finding workplace health promotion to be a good investment.

The results of two studies on the cost–effectiveness of alternative interventions for weight control are discussed below.

Cost–effectiveness of obesity management in the prevention of NIDDM

In a recent study by Segal et al. (*33*), an attempt was made to model the potential cost–effectiveness of a range of interventions for the prevention and treatment of NIDDM in Australia. These interventions included a population approach using mass media programmes focusing on lifestyle changes (including diet and exercise); a behaviour modification programme for the seriously obese; a group programme targeting overweight men (based on an established programme called GutBusters); gastric surgery for the morbidly obese; and a behaviour modification programme for women who had gestational NIDDM.

Both the costs and outcomes of the various interventions were estimated. Net costs (or savings) were derived by adding together programme costs and the potential savings in future health care costs from the prevention of cases of NIDDM. Outcomes were expressed as NIDDM years deferred and life-years saved. Costs were based on reports in the literature, discussions with service providers and published health service cost data. Epidemiological data reported in the literature were used in assessing the effectiveness of various programmes in preventing NIDDM. A range of estimates was calculated based on different assumptions as to programme success, programme costs and other important variables.

The most cost-effective interventions were found to be the GutBusters Programme (a commercial 6-week group session programme for men) and the mass media lifestyle modification programme. It was estimated that both would lead to future cost savings resulting from the reduced incidence of NIDDM, and these savings would be greater than the programme costs. Table 6.2 summarizes the main results of the study.

Although the results presented in Table 6.2 depend very much on the assumption of long-term success in the various weight-loss programmes, the wide range of cost–effectiveness estimates indicate that they are robust. Indeed, while the analysis incorporates the esti-

Table 6.2
Summary of the estimated cost–effectiveness of a range of interventions for the prevention of NIDDM[a]

Intervention[a]	Net cost per NIDDM year avoided (A$)	Net cost per life-year gained (A$)
Surgery for seriously obese:		
all IGT[b]	1200	4600
10% IGT, 90% normal[c]	3500	12300
Diet/behaviour modification for seriously obese:		
all IGT	saving	saving
10% IGT, 90% normal	1600	2600
Group programme for overweight men:		
all IGT	saving	saving
10% IGT, 90% normal	saving	saving
Diet/behavioural programme for women with previous gestational NIDDM:		
all IGT	800	1200
30% IGT, 70% normal	2100	2400
Media programme	saving	saving

[a] Reproduced from reference *33* with the permission of the publisher.
[b] "IGT" refers to programmes targeted at those with impaired glucose tolerance.
[c] "normal" relates to normal glucose tolerance.

mated effect of weight loss on all-cause mortality, and not just that associated with NIDDM, the probable impact of a successful prevention programme on other risk factors (such as cholesterol or blood pressure) has not been taken into account. In addition, the expected savings in future health care costs relate to NIDDM only, ignoring possible savings in the management of other obesity-related diseases. For these reasons the results may well be conservative.

The authors of the study concluded that the prevention of NIDDM, through appropriate interventions, can represent a highly efficient use of community resources. Such programmes can achieve a substantial improvement in health status at little cost or indeed with the possibility of a net saving in the use of health care resources.

Cost–effectiveness of commercial weight-loss programmes

A study by Spielman et al. (*34*) analysed the cost of losing weight in commercial weight-loss programmes in the Boston metropolitan area. It reviewed 11 commercial diet programmes and estimated for each programme the out-of-pocket cost to the participant (over a 12-week

period) of losing 1 kg. The diet programmes were divided into three groups:

1. Medically supervised very-low-calorie diets (VLCDs) that provide <800 $kcal_{th}$/per day.[1]
2. Nutrient-balance reduced-energy diet programmes (REDPs), the client consuming 800–1200 $kcal_{th}$/day[1] (50% carbohydrate, 15–20% protein, <30% fat).
3. Support groups that may or may not offer individual dietary advice and act as a self-help programme with volunteer staff.

It was found that the cost of a 12-week commercial weight-loss programme varied enormously, from US$ 2120 for the most expensive VLCD to US$ 108 for the least expensive REDP. The data are summarized in Table 6.3.

This short-term analysis suggests that support groups and lower-cost REDPs were the most cost-effective interventions. Dietitians were only marginally better value for money than REDPs, particularly when the expected reduction in usual supermarket expenditures is subtracted. However, the study can be criticized on a number of grounds:

- Weight loss in a sample of programme participants was not measured. Instead, the "expected" weight loss based on the literature was used and excellent compliance was assumed throughout the duration of the programme. Weight loss may thus be substantially overestimated and in practice there would be significant differences in the weight loss achieved by the different programmes. Potential drop-out rates are also ignored.
- The study was based on a 12-week programme, did not take into account the costs and impacts of weight-maintenance programmes, and could not take account of the longer-term impact of the competing programmes.
- The financial "costs" measured were restricted to programme initiation fees, the cost of any food supplements purchased as a result of the programme (e.g. liquid protein formulae for VLCDs, or preprepared foods for REDPs) and of medical monitoring and/or associated behaviour modification programmes. Additional costs (or savings) associated with daily food purchases, reduced-energy beverages, etc., were not taken into account. Thus, for a

[1] 1 $kcal_{th}$ = 4.18 kJ.

Table 6.3
Cost in US$ per kilogram of active weight loss (12 weeks)[a]

Programme type	Initial weight	
	80 kg	136 kg
Nutrient-balanced REDP:		
Jenny Craig	23.00	13.50
Nutri-System	19.00	12.00
Registered dietitian	15.00	9.00
Weight Watchers	2.50	1.50
VLCD programmes:		
Health Management Resources (HMR)	17.50	10.00
Medifast	14.00	8.00
Support groups:		
Taking Off Pounds Sensibly (TOPS)	0.07	0.04

[a] Reproduced from reference *34* with the permission of the publisher.

commercial programme, the cost of prepared food plus additional staple items purchased from the supermarket have to be compared with usual food bills.

- The "time" costs of attending the programmes were also not taken into account. These costs may be significant and would differ from one programme to another.

If all the costs had been included and the effectiveness of the interventions measured, the costs of the programmes might well have been different.

Economic costs and benefits of obesity treatment in developing countries
No analyses have been made of the economic costs of obesity treatment in developing countries. However, other analyses of the costs of health interventions show that prevention is more cost-effective than treatment once disease is diagnosed. In Table 6.4, the costs of a variety of public health packages (including education, information, surveillance and monitoring) are compared with those of some primary care clinical services in developing countries where the major needs are the treatment of trauma and infection. Low-income developing countries do not have the resources to provide anything other than public health and essential clinical services. In middle-income developing countries, the high costs of discretionary clinical services (see Table 6.4) mean that the cost of dealing with chronic diseases exceeds that of all other forms of health care. Thus, it would appear to be more cost-effective for money spent on obesity and other NCDs to

Table 6.4
Allocation of public expenditure on health in developing countries, 1990[a]

Type of service	Allocation in developing countries (US$ per person per year)[b]		Contents of health-related packages	Cost per DALY (US$)
	Actual	Proposed		
Public health package	1	5	Immunizations; school health programmes; tobacco and alcohol control; health, nutrition and family planning information; vector control; STD prevention; monitoring and surveillance.	25
Essential clinical services	4-6[c]	10	Treatment of tuberculosis, STDs; infection and minor trauma; management of the sick child; prenatal and delivery care; family planning; assessment, advice, and minor pain alleviation.	25–75
Discretionary clinical services[d]	13–15	6	All other health services, including low-cost treatment of cancer, CVD, other chronic conditions, major trauma, and neurological and psychiatric disorders.	>1000
Total	21	21		

[a] Source: references *35* and *36*.
[b] Estimates are for all developing countries, i.e. an average of costs in low-income countries (annual income US$ 350 per capita) and middle-income countries (annual income US$ 2500 per capita). The figures shown should be regarded as approximate.
[c] Based on estimates in World Bank health sector reports, current spending on essential clinical services is estimated to be 20–30% of total public expenditure on health.
[d] Estimated as total cost of overall health packages minus cost of public health and essential clinical services packages.

be used for prevention rather than for expensive treatments during the advanced stage of disease.

Public health action to prevent obesity has the added benefit of involving the establishment of new or improved physical and social structures within a community, many of which can have long-term positive effects for both current and future generations. Treatment systems, however, are likely to demand recurrent expenditure as new cases of obesity emerge, together with the need for either long-term or repeated treatment. At present, most individuals with excess

weight gain in developing countries are not treated, and the demand for medical and dietetic help is expected to rise rapidly. In addition, limited resources will be diverted to pay for slimming diets and other aids to weight loss.

In developing countries where NCD epidemics are emerging or accelerating, a large proportion of NCD deaths occur in the productive middle years of life, at ages much younger than those seen in developed countries. The health burdens attributable to excess weight gain in societies in transition are likely to be huge because of the absolute numbers at risk, the large reduction in life expectancy and the fact that the problem affects, in particular, individuals with a key role in promoting economic development.

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