6 Obesity

John Garrow and Carolyn Summerbell

1 Summary

Statement of the problem

In 1976, a study group in the UK reported to the DHSS/Medical Research Council: 'We are unanimous in our belief that obesity is a hazard to health and a detriment to well-being. It is common enough to constitute one of the most important medical and public health problems of our time... '[1] In 1979, the US Department of Health published a similar report.[2] In the past twenty five years, many expert committees have issued the same warning, but the problem of obesity is rapidly becoming more serious in both developed and third world countries. The World Health Organization (WHO)[3] now calls it a 'global epidemic', and has concluded that the changing nature of the environment towards greater inducement of obesity (or the 'Obesogenic Environment') is mainly to blame.[4]

Obesity in adults is defined for epidemiological purposes as body mass index (BMI) > 30 kg/m.[2] The crude relationship of BMI (a convenient index of fatness) to all-causes mortality risk is shown in Figure 2: minimum risk is observed between BMI 20 and 25, and mortality increases both below and above this range. It is now known that the increased mortality among thinner subjects is largely related to cigarette smoking and pre-existing disease, and that in weight-stable non-smokers an increased risk of heart disease, diabetes and musculoskeletal disorders becomes evident even below BMI = 25 kg/m.[2] Obesity causes insulin insensitivity, which is an important causal factor in diabetes, heart disease, hypertension and stroke. Adipose tissue converts androgens to oestrogens, which probably explains the reproductive disorders and sex-hormone-sensitive cancers to which obese people are predisposed, and the increased cholesterol flux associated with obesity increases the risk of gallstones and associated diseases. The increased mechanical load increases liability to osteoarthritis and sleep apnoea. Obesity also carries psychosocial penalties. Thus there are many routes by which obesity is a detriment to well-being. All these penalties (except the risk of gallstones) decrease with weight loss.

The health consequences of obesity in children are less well defined, but the risks of diabetes, hypertension, heart disease and perceived poor health are greater among adults who were obese at age 18 years than in those who were of normal weight at age 18 years. Obesity also carries psychosocial penalties for fat children, who are likely to be teased, so there is good reason to try to prevent obesity in childhood.

It is difficult to explain why, when there have been repeated warnings from clinical scientists that obesity is a serious health hazard, there has been no effective control of the problem. We offer four possible explanations.

First, the academic view that obesity per se is not the problem. This arose because studies on cardiovascular risk factors in 50-year-old men showed that if age, cigarette smoking, blood pressure and serum cholesterol were already entered into a multiple regression equation, then adding weight status

did not improve prediction of which men would have a heart attack. This was interpreted to mean that if blood pressure and cholesterol were controlled, then obesity could be ignored. The reasoning was false, since (as indicated above) obesity itself predisposes to hypertension and dyslipidaemia, and also to many other important diseases as well as heart attacks.

The second argument is that obesity is determined by our genes, and therefore untreatable. Certainly there have been recent advances in our understanding of the genetics of obesity, but it is obvious that the rapid increase in prevalence in the past two decades does not reflect a change in the gene pool of the population, since genetic change can only occur between successive generations. A large part of the cause of the global epidemic must therefore be environmental (the 'Obesogenic Environment'), and hence (in principle) treatable.

A third factor is that obesity is a feminist issue. Social psychologists have rightly pointed out that many normal-weight young women try to lose weight to achieve an unrealistic female stereotype of excessive thinness. This is true, and regrettable, especially as some resort to cigarette smoking as a method for weight control. However, it is not an argument for neglecting the real health problem of true obesity.

The fourth problem is related to the third. There is frantic media interest in magic weight loss cures, so the public is bombarded with conflicting misinformation about the causes, consequences and cures for overweight.

For whatever reason, the National Health Service (NHS) and the Health Development Agency (HDA) have been very muted in their response to the obesity challenge, and have hoped that with general advice about healthy lifestyles, some help from the private sector, or perhaps the ever-hoped-for 'superdrug', the problem would go away. This policy has been a spectacular failure, as highlighted by a House of Commons Health Committee Report on Obesity published in May 2004.[5]

Sub-categories

Obesity is a condition that (like hypertension) begins at some arbitrary threshold and becomes increasingly severe. For a given level of obesity in adults the health hazards are greatest among those who are young with existing co-morbidities, or have family histories of obesity-related diseases (diabetes, heart disease, hypertension). For a given amount of fat, a central (intra-abdominal) distribution is more metabolically harmful than a peripheral distribution. The objective of treatment is to manage existing co-morbidities, and in all cases is to reduce fat mass to normal limits, maintain that loss and (if necessary) support the self-esteem of the patient. Healthcare needs are determined by an iterative process, which is described in section 7.

Among children, the objective is to prevent the development of obesity, rather than to achieve fat loss in children who have become obese. Children with obese parents, or who are showing excessively rapid weight gain during primary school years, are most in need of help.

Prevalence and incidence

The prevalence of obesity in adults in the UK has been rising steadily since the programme of national monitoring was started in 1980 (Table 5). The most recent figures available (2002) show that the prevalence of obesity in men was 23%, and in women 25%. In 2002 the prevalence of obesity in boys was 6%, and in girls 7%. The trend data shows how obesity has risen dramatically in the last 25 years, but it is not possible to estimate incidence with confidence since a large number of people are oscillating above and below the thresholds which define obesity and overweight.

Services available and their cost

In 2000 the National Audit Office (NAO) found that it was unusual for GPs and other health professionals to use a protocol for the management of obesity, although the majority of those asked reported that they would find a protocol useful.[6] It can be assumed that this situation has improved a little with time. The NAO survey also found that the primary care team wanted improved access to onward referral options for their overweight and obese patients, and training on obesity management. The variety and quantity of referral options remains limited and patchy across the UK.

The best available estimate of the direct and indirect costs of obesity were calculated for 1998 as part of the NAO report.[6] The NAO estimated that it cost at least £½ billion a year in treatment costs to the NHS, and possibly in excess of £2 billion to the wider community, in 1998. The direct cost is driven primarily by the cost of treating of the secondary disease attributable to obesity, and particularly coronary heart disease (CHD), hypertension and type 2 diabetes. A recent Health Technology Assessment (HTA) report on obesity published in 2004 concluded that the cost of diet and exercise together appear comparable to the cost of drug treatments in obese individuals with risk factors.[7]

Effectiveness of services and interventions

A convenient, but imperfect, measure of the effectiveness of obesity treatment is the weight loss achieved during treatment, and the extent to which it is maintained after active treatment ceases. Ideally, such measurements should be made over a period of several years. It is difficult to achieve high follow-up rates over long periods, so most trials of obesity treatment are characterised by a rather high drop-out rate, and a large variability in weight loss within a group of patients on the same treatment. This makes design of good randomised control trials (RCTs) very difficult. For example, in trials comparing orlistat with placebo, the standard deviation of weight loss within groups was of similar magnitude to the mean weight loss of the group, so it was necessary to recruit several hundred subjects to show statistically significant differences between the orlistat and placebo groups. The interventions which have been shown to be effective are analysed in section 6. It is evident that dietary treatments which are aimed at reducing total energy intake are most effective in causing weight loss in obese people. Decreasing dietary fat, or increasing fibre, are effective only to the extent that they reduce energy intake, which is a rather small effect when such diets are designed to be eaten ad libitum. Very-low-calorie diets cause greater weight loss than low calorie diets in the short term, but in the longer term this advantage disappears. Exercise alone does not cause significant weight loss, but causes a modest improvement in weight loss (and has other health advantages) when combined with a low-energy diet. Drugs licensed for the treatment of obesity cause a modest increased weight loss (effect size B) when added to a low-energy diet. Gastric surgery produces massive weight loss by enforcing a low-energy intake by the patient. The results of studies to manage obesity in children have yielded results that are too variable to summarise with confidence, but targeting a decrease in sedentary behaviour appears to be a useful strategy in both the prevention and treatment of obesity in children.

Quantified models of care

In section 7, a quantified model of care is proposed: the numbers of patients requiring care in a population of 100 000 is estimated in Table 10. For overweight and obese adults there are three levels of service provision: Level 1 is a slimming club in the community led by a non-medical healthcare worker. For those requiring further assessment, Level 2 is based on the primary care team, and Level 3, on a specialist in a

referral hospital. The process is iterative, so estimates of the workload at each level depend on the efficacy of management offered at the other levels, and that offered in the private sector.

The prevention of obesity in children is a different problem. Reasons are given for choosing the primary school years as an optimum stage at which to manage children who are in the highest tenth of BMI at age 5. The objective is not to cause weight loss, but to limit weight gain between the ages of 5 and 12 years.

Outcome measures and audit methods

A crude but robust audit measure is the BMI of the population served, and the weight loss achieved, and maintained, by those adults treated for overweight or obesity. The success of the programme for preventing obesity in children can be assessed by measuring the prevalence of overweight in schoolchildren at age 12 years. This does not address the other objective of improving the psychological status of weight-reduced adults, for which there is no simple audit method.

Information and research requirements

There are many questions to which we need answers. For example:

- the influence of dietary fat intake on obesity prevalence and treatment
- the efficacy of different preventative programmes, especially those involving exercise
- the relative influence of genetics and environment to the familial aggregation of obesity
- the efficacy of strategies for maintenance of weight loss
- the cost of preventing and treating obesity
- the relationship between the amount and rate of weight loss, and the reduction of co-morbidities such as hypertension, heart disease, diabetes, gallstones, osteoarthritis and sleep apnoea.

Introduction and statement of the problem

Introduction

The main purpose of this chapter is to help healthcare commissioners in England to develop purchasing plans for the prevention and treatment of obesity in both the primary and secondary care setting. In theory, this needs assessment activity should be easy. We have good data on the prevalence (but not incidence) of obesity in England (section 4) and the effect this has on health (sections 2 and 3), a number of systematic reviews on the efficacy of interventions for obesity (section 6), and information on costs (section 5). The main difficulty for healthcare commissioners is assessing the optimum field for service provision, i.e. where need, supply (section 5) and demand (section 2) are congruent. For obesity, there is poor overlap between need, supply and demand; we suggest that some of this difficulty is a result of the lack of training on weight management (and nutrition in general) given to medical students, and poor provision for catch-up courses for doctors once qualified (section 5). At the end of this chapter (section 7) we make recommendations of what we believe obesity health services should look like.

What is obesity?

Adults

Obesity is a condition in which body fat stores are enlarged to an extent that impairs health. Obese people tend to die young, and hence are not profitable subjects for life insurance. To guide doctors doing insurance examinations, life insurance companies have for many years published tables of 'desirable weight', based on the mortality experience of people they have insured. As more data become available for analysis it emerges that this desirable range corresponds closely to the range of BMI from 18.5 to 25. The index is calculated by dividing the individual's weight (kg) by the square of his or her height (m). Thus a person who weighed 65 kg and who was 1.73 m tall would have a BMI of $65/(1.73 \times 1.73) = 21.7$, which is in the desirable range. In practice, it is usually more convenient to use a chart such as that shown in Figure 1, which shows the boundaries of BMI 18.5, 25, 30, 35 and 40.

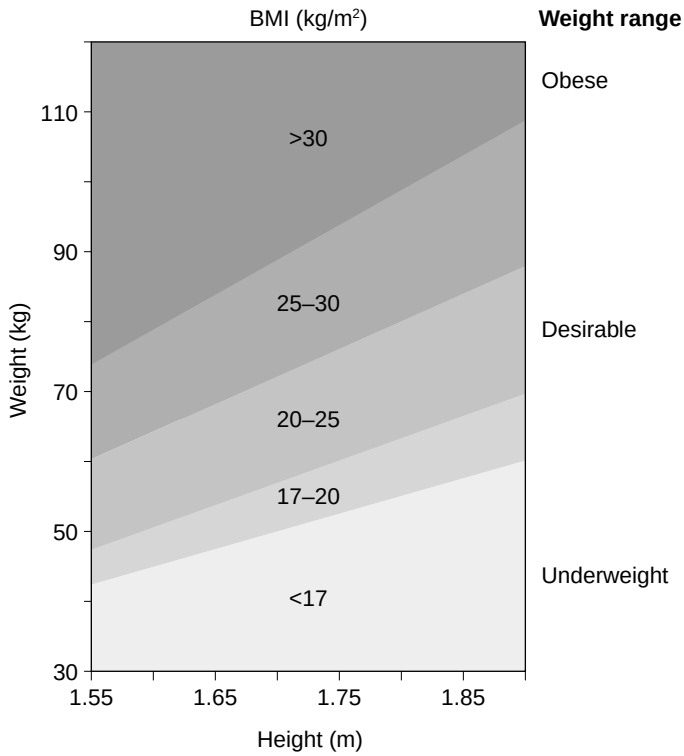

Figure 1: Weights and heights determining the boundaries between individuals who are underweight, normal weight, pre-obese or obese in grade I, grade II or grade III. A person on these boundaries would have a BMI of 18.5, 25.0, 30.0, 35.0 or 40.0 kg/m^2 respectively.[3]

It is arbitrary to choose a value for BMI above which a person is deemed obese: mortality starts to increase significantly somewhere between 25 and 30, and increases rapidly at values of BMI above 30, as shown in Figure 2.

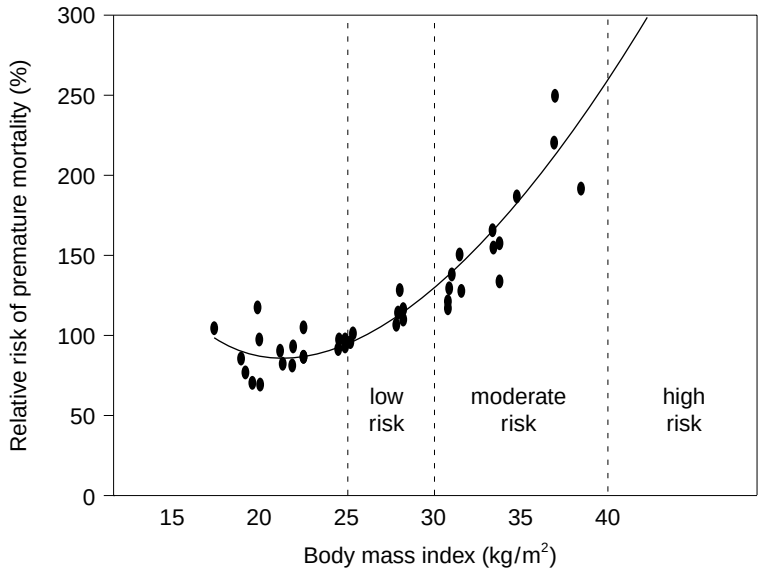

Figure 2: Relative risk of premature mortality with increasing BMI. The weight and height determining the grades of overweight and obesity are shown in Figure 1.
Source: Bray. *Ann Int Med* 1985; **104**: 1052–62.

Very thin people also show decreased longevity (but they usually die from cancer and chronic infectious diseases such as tuberculosis, rather than the non-communicable diseases associated with obesity), so below 18.5 there is increased mortality. At a WHO consultation meeting in 1997, it was proposed that the thresholds shown in Table 1 should be used to classify overweight in adults according to BMI.[8]

The weakness of BMI as a basis for defining obesity is that it does not take account of the distribution of body fat, which also affects health risks (see below). The most popular simple measure of abdominal adiposity is the ratio of circumferences of waist and hips (WHR, or waist/hip ratio). However, there are arguments for using simply waist circumference.[9] Suggested thresholds for waist circumference classification are shown in Table 2.

Table 1: Classification and risk of overweight in adults according to BMI.[3]

Classification	BMI (kg/m^2)	Risk of co-morbidities
Underweight	< 18.5	Low (but risk of other clinical problems increased)
Normal range	18.5–24.9	Average
Overweight	> 25	
Pre-obese	25–29.9	Increased
Obese class I	30.0–34.9	Moderate
Obese class II	35.0–39.9	Severe
Obese class III	> 40.0	Very severe

Table 2: Sex-specific waist circumferences that denote increased risk of metabolic complications of obesity in adult Caucasians.

	Risk of complications	
	Increased	**Substantially increased**
Men	> 94 cm (~37 in)	> 102 cm (~40 in)
Women	> 80 cm (~32 in)	> 88 cm (~35 in)

Children

The simple classification shown in Table 1 for obesity in adults is not applicable to children, since the ratio of velocity of weight gain to height gain changes during normal growth, especially around puberty. Many different methods are currently in use to estimate body fatness or relative weight, and for each method, various cut-off levels are used to describe overweight or obesity. These problems have been discussed in more detail elsewhere.[10] To encourage consistency in defining fatness, the International Task Force on Obesity has developed an international reference population and BMI standards to classify overweight and obesity, using age- and sex-specific curves.[11] BMI charts for boys and girls, based on these standards, should be used (published by the Child Growth Foundation, London). The BMI of a boy or girl on the 50th centile at age 1 year is 17.5 kg/m^2, falls to 15.5 kg/m^2 at age 6 years, and climbs to 21 kg/m^2 at age 18 years.

In the case of adults, the bands of BMI can be related to health risk as indicated in Figure 2. However, it is difficult to base a definition of obesity in children related to the health risks in adult life because there are too few longitudinal studies on which these judgements can be made. Even if these data were available, this task would be a tricky one since children often cross many centiles of weight-for-height, especially children under the age of 5 years. However, obesity in childhood is recognised as a significant health risk, as explained in more detail below, since obese adolescents are at increased risk of obesity in adult life[12] and at increased risk of mortality, independent of adult weight.[13]

Summary

The classification of obesity in both adults and children simply requires the accurate measurement of height, weight and waist circumference. However, as with most measurements there are potential errors involved. The most common error, particularly common in primary care, is a result of self-reported height and/or weight.[14] Tall, thin individuals are more likely to under-report their height, and shorter, fatter individuals to overestimate their height and underestimate their weight. It is important that weight and height are measured, and measured correctly; weight in light clothing and height without shoes. Weighing scales should be calibrated regularly, and height sticks should be checked to make sure that they are correctly placed. The good news for health commissioners is that the assessment of obesity is remarkably cheap!

Why is obesity important?

There is increasing recognition that obesity is important in causing many of the major non-communicable diseases with which it was previously classified merely as an associated condition. On 1st June 1998 the American Heart Association announced that it was upgrading obesity from a 'contributing risk factor' to a

'major risk factor for coronary heart disease'.[15] Obesity is a lifelong disease, not just a cosmetic issue nor a matter for moral judgement.

Adults

Obesity and total mortality

The main cause of the premature death rate among obese people is heart disease: hypertension, coronary thrombosis and congestive heart failure are all significantly more common among obese people than among normal-weight controls. Of course age and cigarette smoking are important contributors to the risk of heart disease in both obese and non-obese people, but obesity increases the risk (Figure 3). High blood pressure, raised concentration of plasma low-density cholesterol and a low concentration of high-density cholesterol fractions are all important risk factors, but weight gain makes these factors worse, and weight loss makes them better.

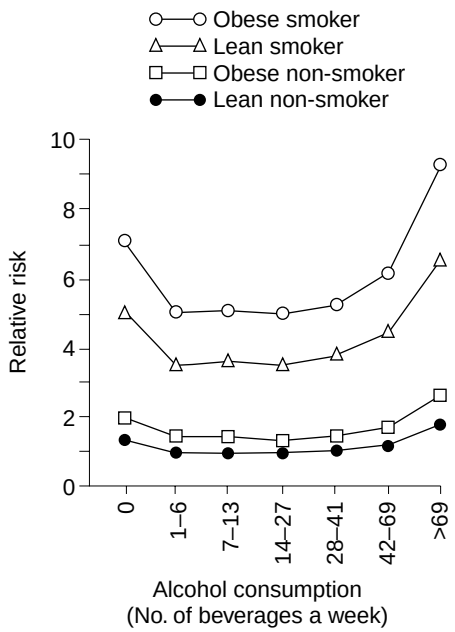

Figure 3: Relative risks (RR) of mortality in relation to alcohol intake at specified levels of smoking and BMI. (RR set at 1.00 for lean, non-smoker with alcohol intake of one to six beverages a week.) *Source*: Gronbaek *et al. BMJ* 1994; **308**: 302–6.

The way in which cigarette smoking and adult weight gain confuse the relationship between obesity and mortality is well illustrated in Table 3, which summarises results from a large study of nursing personnel by Manson *et al.* in 1995.[16]

On inspection of the relative risk of death during the 14-year follow-up of these women it appears that those in the whole group with a BMI between 19 and 27 kg/m^2 have the least mortality risk (0.8 relative to 1.0 for those with a BMI < 19). Only in the range 27.0–28.9 does the risk rise to the level of those < 19 kg/m^2, and above 29 the risk is definitely increased. However, when those women who had never smoked are analysed separately, a different story emerges. Among non-smokers the mortality risk starts to increase at

Table 3: Influence of BMI, history of cigarette smoking, and adult weight gain, on relative mortality risk in 30–55-year-old women followed for 14 years.

BMI	< 19	19.0–21.9	22.0–24.9	25.0–26.9	27.0–28.9	29.0–31.9	> 32.0
All women							
Adj. RR[a]	1.0	0.8	0.8	0.8	1.0	1.2	1.5
95% CI[b]		0.7–0.9	0.7–0.9	0.7–0.9	0.9–1.1	1.0–1.3	1.3–17
Women who never smoked							
Adj. RR	1.0	1.0	1.1	1.1	1.4	1.7	1.9
95% CI		0.8–1.3	0.9–1.3	0.8–1.3	1.1–1.8	1.4–2.2	1.5–2.5
Women who never smoked and had stable weight[c]							
Adj.RR	1.0	1.2	1.2	1.3	1.6	2.1	2.2
95% CI		0.8–1.6	0.9–1.7	0.9–1.9	1.1–2.5	1.4–3.2	1.4–3.4

[a] Relative risk of death from all causes adjusted for age, smoking, menopausal status, use of oral contraceptives and post-menopausal hormones, and parental history of myocardial infarction before age 60 years.
[b] 95% confidence interval for relative risk.
[c] Excluding first four years of follow-up and women with > 4 kg weight change during those four years.

22 kg/m^2 and is significantly increased at 27 kg/m^2, so the increased mortality among the women < 19 kg/m^2 disappears when the smokers are removed from the analysis. An even greater change occurs if the analysis is restricted to those women who had never smoked, and who had not either died or gained > 4 kg in the first four years of follow-up. Now the J-shaped curve has disappeared, the minimum mortality is with the thinnest women, and a significant increase in mortality risk occurs above 27 kg/m^2. Some of the women who died within four years of enrolment in the survey probably had a disease at that time, which may explain why they were thin and also why they died. This example has been considered in some detail because crude data on mortality do not provide reliable information on the health risks of obesity if the confounding effects of cigarette smoking, adult weight gain and previous disease are not allowed for. If these factors are removed, a woman aged 30–55 years is more than twice as likely to die in the next 14 years if her BMI is > 29 kg/m^2 than if it was < 19 kg/m^2.

The effect of age on excess mortality from all causes associated with obesity is controversial. In a large cohort of obese persons (n=6193), obesity-related excess mortality declined with age at all levels of obesity.[17] This is not because obesity causes less ill health in older people, but because in older people death from causes unrelated to obesity becomes more common.

It is important to note that the obesity-related health risks cited above do not necessarily apply to populations of different ethnic origins. However, a systematic review[18] concluded that differences in absolute risk by ethnicity are not relevant to individuals in a clinical setting.

It is also important to note that there is evidence which suggests that being fit may reduce the hazards of obesity.[19]

Obesity: socioeconomic consequences

From the viewpoints of human suffering and healthcare expenditure, morbidity is as important as mortality. Obesity causes disability as well as death. Rissanen *et al.*[20] analysed data from the Finnish Social Security system and found that the risk of drawing a disability pension increased significantly with BMI, even within the 'desirable' range of 20–25 kg/m^2; one quarter of all disability pensions in women and half

as many in men were due to obesity. These disabilities arose mainly from cardiovascular and musculoskeletal disease.

The impact of obesity on a number of specific diseases is outlined below (e.g. Figure 4).

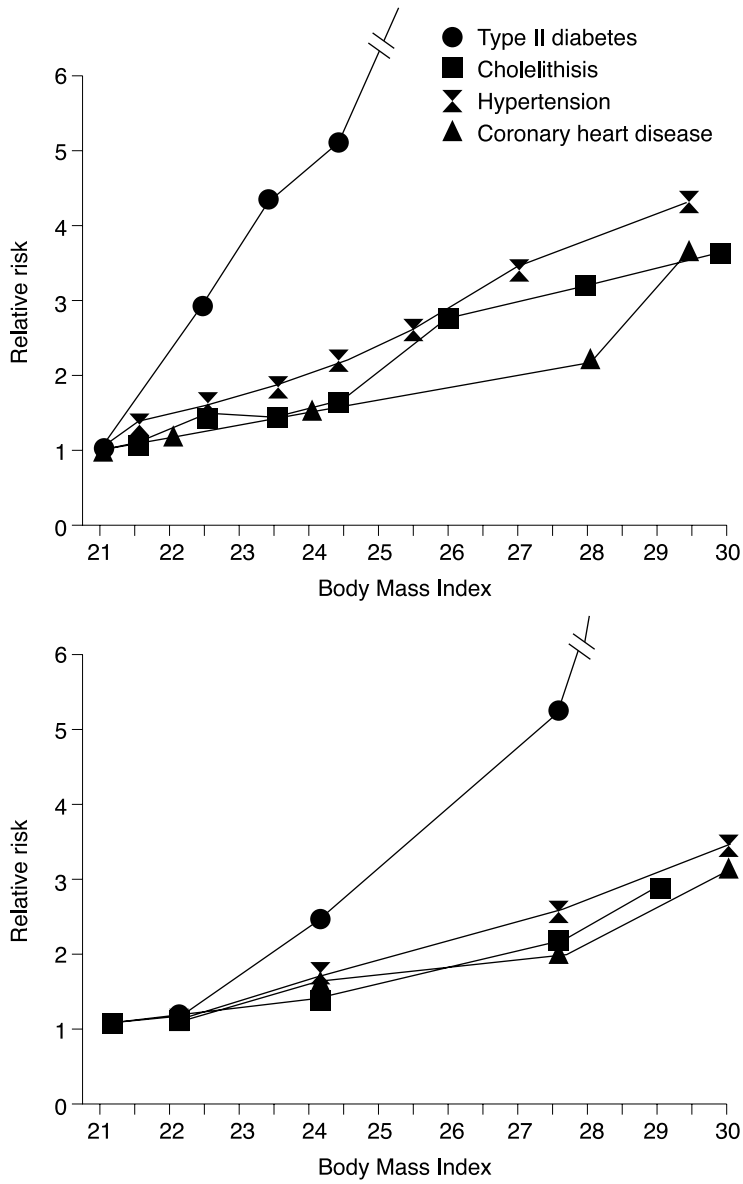

Figure 4: Relationship between BMI up to 30 and relative risk of type II diabetes, hypertension, CHD, and cholelithiasis. (a) Relations with women, initially 40–65 years old who were followed up for 18 years; (b) Relations with men, initially 40-65 years old, who were followed up for ten years. *Source*: Willett *et al. NEJM* 1995; **273**: 461–5.

Obesity, insulin sensitivity and diabetes

Type II diabetes is not as impressive a cause of mortality among obese people as heart disease, but it is itself a risk factor for heart disease, and also a very important cause of morbidity from neuropathy, nephropathy and eye disease.

The relationship between obesity and type II diabetes is very tight, and the risk of type II diabetes increases sharply with increasing BMI, particularly in men (Figures 4 and 5). A man more than 140% of average weight is 5.2 times more likely to die of type II diabetes than a normal-weight man, and for women the mortality ratio is 7.9 times for a similar degree of overweight.[21] A classic study of experimental obesity in Vermont has shown that the association between obesity and reduced insulin sensitivity (which is the primary problem in type II diabetes mellitus) is a causal one. Young male volunteers, with no family history of type II diabetes or obesity, overate for six months so they increased their weight by 21%, of which 73% was fat, and they then showed significant changes in biochemistry in the direction of type II diabetes. After weight loss to normal values these changes reverted to normal.[22] Even among pre-pubertal children obesity is associated with peripheral and hepatic insulin resistance.[23]

Although type II diabetes is not directly the cause of most of the excess mortality among obese people, the metabolic defect underlying type II diabetes is clearly the result of obesity,[24] which itself predisposes to hypertension and heart disease. These defects are reversible with weight loss, with corresponding improvement in mortality. A deliberate weight loss of 0.5–9.0 kg is associated with a 30–40% reduction in type II diabetes-related mortality.[25]

Coronary heart disease

The main cause of the excess mortality among obese people is coronary heart disease (CHD). Obesity is itself strongly related to hypertension and stroke (Figure 4), particularly in young people.[26] However, these risk factors improve when obese people lose weight.[27] After adjustment for age and smoking, the risk of a fatal or non-fatal myocardial infarction (MI) among women > 29 kg/m^2 is three times that among lean women.[16,28] High blood pressure, high triacylglycerol and high low-density lipoproteins favour the formation of atheromatous lesions, but obese people have the added hazard of abnormalities of blood clotting factors, which further increase the risk of thrombosis and MI.[29] These abnormalities improve with therapeutic weight reduction.[30]

Cancer

A very large survey by the American Cancer Society found that the mortality ratio for cancer among men who were 40% overweight was 1.33, and for women 1.55. The most important increase is for breast cancer in post-menopausal women, but there is also an increased risk of cancer of the endometrium, uterus, cervix, ovary and gall bladder in women, and of the colon, rectum and prostate in men.[31] Intentional weight loss of 0.5–9.0 kg is associated with a decrease of 40–50% in mortality from obesity-related cancers.

Osteoarthritis

Degenerative disease of weight-bearing joints is a very common complication of obesity, particularly in the knees of middle-aged women, and causes significant disability.[20] Unlike the risk of heart disease or diabetes, the risk of osteoarthritis is related to the total amount of fat, and not in particular to the amount of intra-abdominal fat.[32]

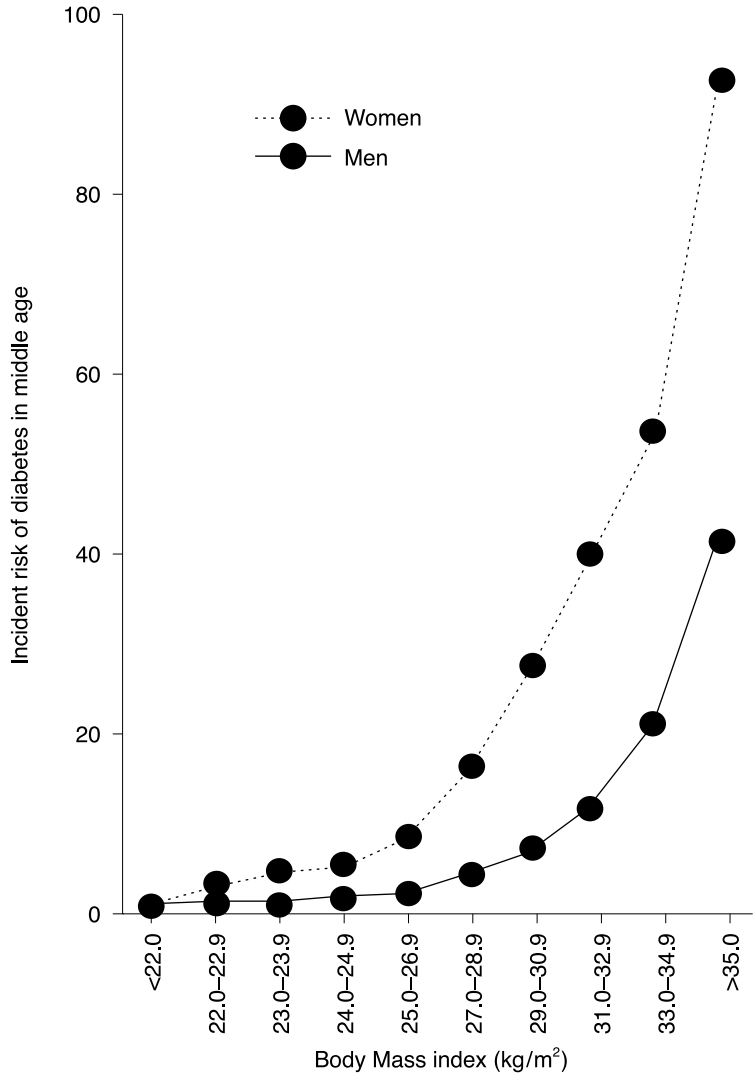

Figure 5: Relative risk of Type II diabetes with increasing weight.
Source: Colditz *et al. Ann Int Med* 1995; **122**: 481–6.

Gallstones

Obese people have a higher output of cholesterol in bile, with a lower concentration of bile salts, so their bile is constantly in danger of forming gallstones (Figure 4). Rapid weight loss increases the release of cholesterol from adipose tissue, and hence increases the load to be excreted in bile.[33] Bile stasis contributes to biliary infections, and also to the risk of gall-bladder cancer, mentioned above.

Reproductive disorders

Obesity is associated with disorders of menstrual function, fertility and childbirth. Examination of obese pregnant women is particularly difficult, whether by abdominal palpation, ultrasound or laparoscopy. The difficulty in monitoring fetal well-being in severely obese mothers partly explains their increased rate of caesarian section. However, even moderate degrees of obesity are associated with an increased incidence of hypertension, toxaemia, gestational diabetes, urinary tract infections and fetal macrosomia.[34] There is also an increased risk of neural tube defects in the children of obese mothers: among women over 70 kg in weight, dietary intake of folic acid does not have the same protective effect as it has in leaner women.[35,36]

Sleep apnoea

Obesity causes inefficiency of respiratory function by several mechanisms. The mechanical load of fat on the chest wall increases the mechanical work of inspiration, especially when the subject is recumbent, and a large mass of intra-abdominal fat tends to push the liver upwards, thus decreasing the intrathoracic space. There is also a mismatch of pulmonary ventilation and perfusion, so much of the blood flowing through the lung capillaries is at the base of the lung, where ventilation is poor. These problems may cause the Pickwickian syndrome of chronic hypoxia and carbon dioxide retention, which may manifest itself as inappropriate somnolence, vividly described by Dickens in the fat boy in *The Pickwick Papers*, and obstructive sleep apnoea (OSA). This is a serious condition, which is associated with pulmonary hypertension and right-sided heart failure. Data from the Swedish Obese Subjects (SOS) study show that OSA was an important contributor to morbidity in severe obesity, and contributed to cardiovascular mortality.[37] Respiratory function improves when obese people lose weight.[38]

Psychological and social disorders

The health hazards of the obese person, which have been listed above, become increasingly evident as the person becomes older: heart disease, hypertension, stroke, osteoarthritis, cancer and gallstones are all conditions which occur mainly in older people, so the obese young person does not experience these as a threat. However, the psychological and social penalties of obesity fall mainly on the child and young adult. Indeed, there is a view, often promoted in the media, that the penalties of obesity are mainly due to social discrimination, so if society treated obese people with respect and tolerance it would cease to be a problem.[39] This is wrong: the health hazards of obesity listed above would remain, however respectfully obese people were treated, but there is compelling evidence that our society discriminates against fat people.[40]

Social discrimination continues through adult life. Sonne-Holm and Sorensen[41] showed that, for a given parental social class, intelligence and education, severely obese people achieved less favourable social status than non-obese people. In the US, Gortmaker *et al.*[42] studied a nationally representative sample of 10 039 men and women who were 16 to 24 years old in 1981, and obtained follow-up data on 65–79% of the cohort seven years later. Women who were initially above the 95th centile for BMI had completed fewer years in school, were less likely to be married and had higher rates of household poverty than the women who had not been overweight, independent of their baseline socioeconomic status and aptitude-test scores. However, people with chronic conditions such as asthma and musculo-skeletal abnormalities did not differ from non-overweight people in these ways.

In the general population, those who are overweight or obese are not significantly more depressed than lean people.[40] Among those who are depressed, it is difficult to establish if this is caused by obesity or caused by dieting in an unsuccessful attempt to reverse the obesity. Among the severely obese, there have been numerous reports of psychopathology: for example volunteers for the SOS study, both men and

women, showed very poor ratings for mental well-being, and more symptoms of anxiety and depression than the reference population. The score on psychometric scales were as bad as, or worse than, those of patients with chronic pain, generalised malignant melanoma or tetraplegia after neck injury.[43] It is particularly important for doctors or dietitians who are treating obese patients to remember that their duty is to help to restore the self-esteem of obese patients, as well as to help them to lose their excess weight. This point is considered again when discussing treatment of obesity.

It is useful to note that there is some evidence that weight concerns vary with ethnic group.[44,45]

All of these penalties of obesity decrease with weight loss, with the exception of the risk of gallstone formation. During weight loss in an obese person, the cholesterol in adipose tissue is mobilised and the bile may become even more liable to form cholesterol stones.

Fat distribution and health risk

A study in Gothenburg, Sweden, showed that people with a high WHR (indicating that fat was largely in the abdominal cavity, rather than subcutaneously on the limbs) had a greater risk of heart disease and diabetes than people with a similar amount of fat distributed peripherally.[46] This probably relates to the insulin insensitivity which is caused by a high flux of free fatty acids in the portal circulation, because intra-abdominal fat cells can release fatty acids very rapidly. However, further studies have shown that the increased mortality among men was not significantly related to WHR when the follow-up period was extended to 20 years,[47] and the central distribution of fat is associated with both cigarette smoking and a high alcohol intake, which may have contributed to some of the observed excess mortality risk.

Children

Effect on adult morbidity and mortality

There is limited evidence for an association between adolescent obesity and increased risk of adult morbidity[10] and mortality.[13] Must *et al.*[13] showed that obesity in adolescence predicts a broad range of adverse health effects that are independent of adult weight after 55 years of follow-up: the risks of morbidity from CHD and atherosclerosis are increased among men and women who were overweight as adolescents.

There is better evidence that the childhood period is important for adult obesity (and all the associated health risks, as described above) because tracking of overweight, albeit moderate, is observed between childhood and adulthood. This topic has been reviewed.[10,48] The figures vary according to the definition of obesity and length of follow-up, but fat children have a high risk of going on to become fat adults. For example, in the 1958 British birth cohort, 38% of boys and 44% of girls above the 95th BMI centile at age 7, were obese at age 33.[49] Even so, only a small proportion of fat adults were fat in childhood. It is likely that there are factors operating in early adulthood that promote obesity, but there may also be factors operating in childhood that promote adult obesity. It is still a matter of debate whether there are particular stages in childhood, during which physiological alterations increase the risk of later obesity. These stages are termed critical periods, and may include the prenatal period, the adiposity rebound (second rise in adiposity occurring at about 6 years), and puberty.[50]

Parental fatness has also been identified by a systematic review as the most important predictor in childhood of adult obesity,[12] although the contribution of genes and inherited lifestyle factors to the parent–child fatness association remains largely unknown. Other important risk factors in childhood of adult obesity included social factors, birth weight, timing or rate of maturation, physical activity, dietary factors and other behavioural or psychological factors. The relationship between low socioeconomic status

(SES) in childhood and increased fatness in adulthood is remarkably consistent, but when fatness is measured in childhood, the association with SES is less consistent. Studies investigating SES were generally large, but very few considered confounding by parental fatness. Women who change social class (social mobility) show the prevalence of obesity of the class they join, an association which is not present in men, and the influence of other social factors such as family size, number of parents at home and childcare have been little researched. Parsons *et al.*[12] found good evidence from large and reasonably long-term studies, for an apparently clear relationship for increased fatness with higher birth weight, but in studies which attempted to address potential confounding by gestational age, parental fatness or social group, the relationship was less consistent. The relationship between earlier maturation and greater subsequent fatness was investigated in predominantly smaller, but also a few large studies. Again, this relationship appeared to be consistent, but in general, the studies had not investigated whether there was confounding by other factors, including parental fatness, SES, earlier fatness in childhood, or dietary or activity behaviours. Studies investigating the role of diet or activity were generally small, and included diverse methods of risk factor measurement. There was almost no evidence for an influence of activity in infancy on later fatness, and inconsistent but suggestive evidence for a protective effect of activity in childhood on later fatness. No clear evidence for an effect of infant feeding on later fatness emerged, but follow-up to adulthood was rare, with only one study measuring fatness after the age of 7 years. Again, confounding variables were seldom accounted for. A few, diverse studies investigated associations between behaviour or psychological factors and fatness, but mechanisms through which energy balance might be influenced were rarely addressed.

Effects during childhood

Obese children are more prone to physical ailments, and are also liable to underperform at school relative to their potential.[11] The health consequences of obesity in youth have been reviewed;[51,52] increased blood lipids, glucose intolerance, hypertension and increases in liver enzymes associated with fatty liver, have all been observed to be more common in obese children or adolescents. The diseases to which obese children are more liable are tabulated in Table 4.[8]

Table 4: Health consequences of childhood obesity.

High prevalence	Intermediate prevalence	Low prevalence
Faster growth	Hepatic steatosis	Orthopaedic complications
Psychosocial	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
Dyslipidaemia		Pseudotumour cerebri
Elevated blood pressure		Cholelithiasis
		Hypertension

The main penalties of obesity experienced by children are social isolation and peer problems.[53] Although there is little evidence to suggest that self-esteem is significantly affected in obese young children, on reaching the teenage years the effect is striking. Obese children are believed by their peers at school to be lazy, dirty, stupid, ugly, cheats and liars, and these perceptions have been reported by children as young as 9 years old.[54]

Overweight adolescent women have a lower educational attainment, lower incomes and are less likely to marry than those not overweight.[42] If these relationships are indeed causal, then they imply far-reaching consequences for costs to health services, and the total healthcare.

Why does the NHS think that obesity is not important?

The dramatic effect which obesity has on quality of life, morbidity and mortality (and equally how these can be reversed with weight loss) has resulted in repeated and strongly worded reports by national and international expert committees that 'obesity is one of the most important public health hazards of our time'.[8,18,55–57] There is a stark contrast between these authoritative warnings, and the absence in any country of any effective preventative or treatment programme to reverse the increasing prevalence of obesity. In the UK, a target for obesity was omitted from all current UK Government health policy documentation, including *Our Healthier Nation: Saving Lives*.[58] However, the Health Development Agency (HDA) produced an Evidence Briefing on 'The management of obesity and overweight'[59] which lists useful strategies, and NICE has produced guidance on the use of orlistat,[60] sibutramine[61] and surgery[62] for the management of obesity reviews and guidance on drugs and surgery. The difficulty for health care providers is how best to use these separate guidance strategies in the management of obesity. This was one of the many concerns highlighted by the House of Commons Health Committee.[5] An obesity strategy, and guidance on the management of obesity, are required to help tackle the problem of obesity in the UK. The good news is that NICE, in collaboration with the HDA, has started to work on these documents, and outputs were expected by summer 2006. In the interim, the guidance produced by the Scottish Intercollegiate Guidelines Network (SIGN) should be used.[63] So why has the health service refused to treat obesity seriously? We do not have hard evidence on which to base an answer to this crucial question, but believe there are several factors that, in varying proportion, inhibit health services from controlling obesity. Some of these obstacles have a factual basis, others are based in misapprehension of the facts. If progress is to be made these obstacles need to be removed. Their nature and basis is briefly reviewed below.

'Obesity per se is not a health hazard'

In the 1950s, international epidemiology was in its infancy. Keys and colleagues set up a prospective study of the factors which predisposed to CHD. They recruited healthy men aged 40–59 years in seven countries (USA, Japan and five European countries), and found after 15 years of follow-up that adiposity did not significantly predict mortality if age, cigarette smoking, blood pressure and cholesterol were already entered into a multiple regression equation.[64] This finding led public health policy makers to the view that obesity per se was benign, and that the real villains were smoking, blood pressure and cholesterol. The flaw in the argument, as described above, is that obesity itself predisposes to hypertension and hypercholesterolaemia, but even when these factors are allowed for, obesity remains an independent risk factor for heart disease and total mortality both in men[65] and women.[25]

'Obesity is genetically determined, and hence untreatable'

The genetics of obesity in some laboratory rodents has been extensively explored, and it has been shown that the genetically determined absence of 'leptin' entirely accounts for the obesity of some strains of mouse. The role of leptin in the aetiology of obesity in human subjects is not clear: obese people have high, not low, leptin concentrations. There is also good evidence from twin studies that the susceptibility to weight gain in a given environment is affected by hereditary factors, probably by the interaction of many

genes. However, it is obvious that the recent dramatic increase in the prevalence of obesity in the UK cannot be ascribed to a change in the genetic make-up of the population because genetic make-up can change over generations, not during the lifetime of an individual. Obesity is determined by the interaction between genes and environment, and is greatly influenced by psychosocial factors and cognitive actions. Environmental and lifestyle factors can certainly be changed, and it is by this route that obesity can be prevented or treated in the individual and in the community.

'Fat is a feminist issue'

Orbach[66] made the valid point that many young women struggled vainly to achieve unphysiological thinness in order to meet a feminine stereotype which was thought to be attractive, and they would be better if they gave up this futile endeavour. This is true, and it should remind healthcarers that it is important not to damage the self-esteem of obese people when trying to help them to lose weight. However, it is not a valid argument against providing help for those who are obese, so they can avoid the health hazards listed above.

Orbach's original thesis has been taken further by pressure groups who claim that health education campaigns designed to control obesity are a form of unfair discrimination, analogous to racism, and should be made illegal. Health educators who say (truthfully) that obesity is unhealthy, and can be avoided or reversed by lifestyle changes, may be accused of 'victim blaming'. No doubt this in part explains the very muted response of government organisations to the obesity problem.

Media misinformation

There is huge public demand (described below) for an easy solution to the problem of weight loss, so it is not surprising that an unlimited amount of bizarre advice, magic potions and pseudo-science is on offer to the bemused consumer. This is part of the price we pay for freedom of expression, but it is an obstacle to those who try to offer reliable guidance about the health hazards of obesity, and what can and should be done to avoid them.

Public demand and putting the NHS services for obesity into context

Demand may be defined as what the public would be willing to pay for, or might wish to use, in a system of free healthcare. The public demand for help to acquire the perfect body is immense; it is a national obsession, particularly among women. This is one of the key differences in the planning of obesity health services as compared with those for other diseases. The public demand is catered for by the many sources of help from outside the health service. Help in the form of specialist magazines and books, slimming clubs and slimming foods has soared in the past 20 years. The fact that so many people within England seek advice on obesity from outside the health service highlights two important issues for health services commissioners.

- Most individuals who would benefit from losing weight probably use non-NHS services, but only a minority also demand help from, or are offered help by, the NHS. Unfortunately the constant background information on dieting from other sources is usually more compelling. The patient may request advice from the health professional regarding a specific diet or food which they have heard will enhance their weight loss (or even cause weight loss without the tedium of dieting). Regardless of the credibility of reported claims for such diets or foods, it is important that the health professional addresses them seriously and does not dismiss them as simply ridiculous; if the patient thought that

these claims were ridiculous he or she would not have asked for advice on them in the first place. We suggest that effective care in this field requires provision of services by both the NHS and others working in close collaboration.

- Demand for obesity services is greater than the need for them since many individuals who seek advice on weight management are not overweight or obese. However, it is also important to recognise that these individuals might benefit from sound advice on lifestyle issues through the health promotion services.

Children are quite different from adults in terms of non-NHS sources of help on weight management. The sources mentioned above are targeted only at adults, and this provides the NHS with a niche on this childhood obesity 'market'. Earlier in this section we highlighted the risks of childhood obesity, and in section 7 we suggest ways in which the NHS may deliver services to this vulnerable group.

Prospect for new effective treatments

Readers of this chapter will be aware that the media are constantly heralding a 'breakthrough' in the treatment or prevention of obesity, since such items are effective in selling newspapers. The two developments that have received most attention recently are the discovery of the hormone leptin and the licensing of two anti-obesity drugs: orlistat and sibutramine. The evidence relating to these compounds is reviewed later in the chapter. The purpose of this short note is to consider the possibility that they will revolutionise the management of obesity and thus render the contents of this chapter obsolete.

Leptin is a hormone, released from adipose tissue, which has the effect of reducing food intake in genetically obese mice, and curing their obesity and infertility. Initially, therefore, there was great optimism that it would have similar effects in obese human subjects. It was found, however, that obese human subjects did not (like obese mice) have abnormally low levels of leptin, but abnormally high levels. So far (except in a single case of a rare genetic disorder) leptin has not been shown to be therapeutically effective in human obesity.

Orlistat is a drug that inactivates the enzymes that digest fat in the human small intestine, and thus reduces fat absorption by about 30%. The results of RCTs are reviewed in section 6. In very large, multicentre, international trials orlistat has been shown to cause greater weight loss in obese subjects on a low-fat diet than that observed in control subjects; this difference is statistically significant (since the trials involved about 1000 subjects) but clinically not very impressive.

Sibutramine promotes a sense of satiety through its action as a serotonin and noradrenaline re-uptake inhibitor. In addition, it may have an enhancing effect on thermogenesis through stimulation of peripheral noradrenergic receptors.

In a French trial,[67] patients with BMI > 30 were screened using a four-week treatment on a very-low-calorie diet; only those who lost > 6 kg in this phase entered the trial. An intention to treat analysis showed the mean weight change after one year among 81 patients on sibutramine (10 mg) was –5.2 kg, and among 78 patients on placebo was +0.5 kg. In an American trial,[68] 1463 patients were screened, 1047 were randomised and 683 completed the 24-week study. The weight loss at completion ranged from 1.2% in the placebo group to 9.4% among those on a dose of 30 mg/day. These results show that sibutramine causes weight loss which is statistically greater than placebo, but not impressive compared with the initial overweight of the volunteers, and with a large variation in response between individuals.

We are confident that, at least for the next decade, the health problems associated with obesity, and the methods available for effective treatment, will be little changed from the present situation. We do not expect that a new therapeutic 'breakthrough' will greatly affect the assumptions on which this chapter is based.

3 Sub-categories

Sub-categories of obesity related to risk of associated diseases

Health risk, in terms of mortality, increases progressively from normality to very severe obesity (section 2, Figure 2). At each weight, this health risk is greatest in young people, people with diabetes or hypertension, and people with a relatively high WHR. The costs of obesity, both to the individual and to the community, arise mainly from the co-morbidities rather than from obesity itself (*see* section 5).

From the viewpoint of healthcare planners there is a dilemma. One strategy (which has been widely adopted in the past) is to plan to treat the co-morbidities – heart disease, stroke, hypertension, diabetes, osteoarthritis, gallstones, certain cancers, reproductive disorders, sleep apnoea, psychological and social disorders – and ignore the underlying obesity. This strategy is superficially plausible, since patients with these co-morbidites clearly need treatment. However, experience shows that the strategy is expensive and ineffective if the underlying obesity is allowed to increase.

An alternative strategy is to seek to prevent the development of obesity by health education campaigns that promote physical activity and healthy diets of low energy density. This also fails for several reasons. First, people will not adopt healthier lifestyles unless the facilities are available to modify their diet and exercise more in conditions that are affordable and safe, and unless they have a clear understanding of the relationship between overweight and health risk. These requirements are not met at present. Second, a campaign to prevent overweight and obesity is inadequate to meet the needs of a population in which over half of all adults are already overweight, and one quarter of all adults are already obese. Third, campaigns that exhort adults to 'fight the flab' do not address the problem of increasing obesity among children. Government and health authorities potentially have some control over the diet and physical activity of schoolchildren, but this opportunity is not being effectively used to prevent obesity at its earliest stage.

The solution to the dilemma lies in the ability of healthcare planners to take a broader view of the problem of obesity, and integrate its three sub-categories. These are:

- **Obese patients with existing co-morbidities**. These are the most visible component of the problem, to which the most attention is at present paid. With better management of the other two components there will be fewer patients in this category, and so costs in this sector will decrease, and the level of public health will improve.
- **Obese individuals who do not yet have co-morbidities**. These are the people who are at present least well served by the NHS.
- **Adults and children who are not obese, but particularly at risk to become obese** (see below).

The evidence for categorising the health risk in children by degree of obesity is not available. Section 7 describes the evidence which suggests that primary school children, compared with younger or older children, are more effectively managed.

Sub-categories of the non-obese population who are most likely to become obese (predictors of obesity)

It is important to state at the outset that anybody is at risk of becoming obese, so long as they consume more energy than they use. Adults who are most likely to become obese are:

- those who were previously obese and who have lost weight
- smokers who have stopped smoking

- those who change from an active to an inactive lifestyle
- those with poor educational achievement.

Children at high risk of becoming obese adults (as detailed in section 2) are:

- children who have high weight-for-height
- children who have obese parents.

4 Prevalence and incidence

Adults

The prevalence of obesity, particularly in the developed world, has increased particularly rapidly over the past two decades. The WHO calls it a 'global epidemic'.[3] The prevalence of obesity in UK adults and children, compared with those living in other European countries, is shown in Figures 6, 7, and 8.

There are good data for England showing the prevalence of obesity in the UK (Table 5). The prevalence of overweight and obesity in the UK population was first determined in 1980, in a survey of a representative sample of 5000 men and 5000 women aged 16–64 years. At that time, the proportion of men in the 'pre-obese' stage was 34%, and 6% were obese; for women there were 24% in the 'pre-obese' stage and 8% were obese.

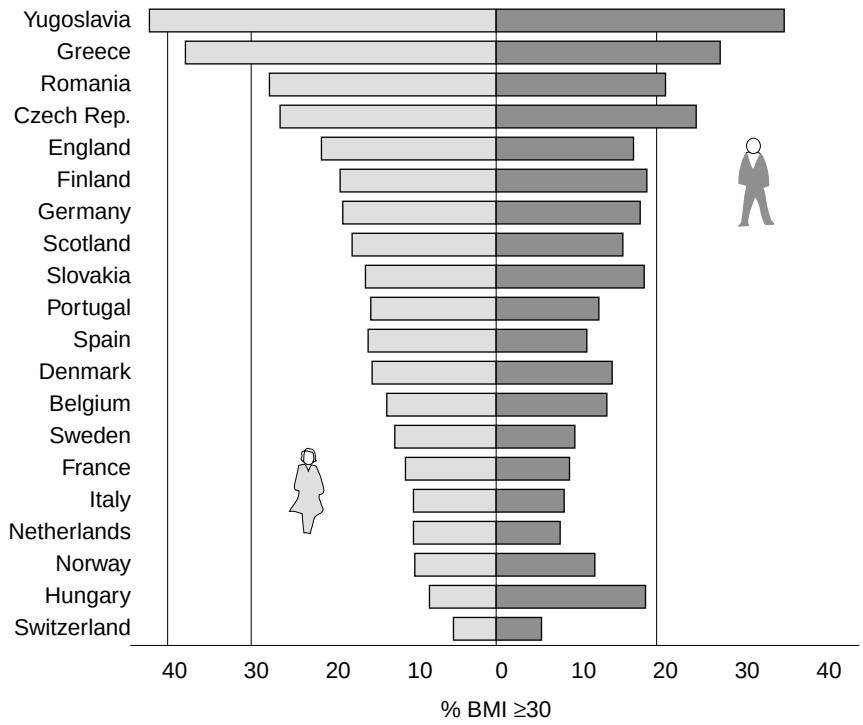

Figure 6: Prevalence of obesity (%) in adults in selected European countries in 2002 (IOTF data). *Source*: With kind permission of IOTF.

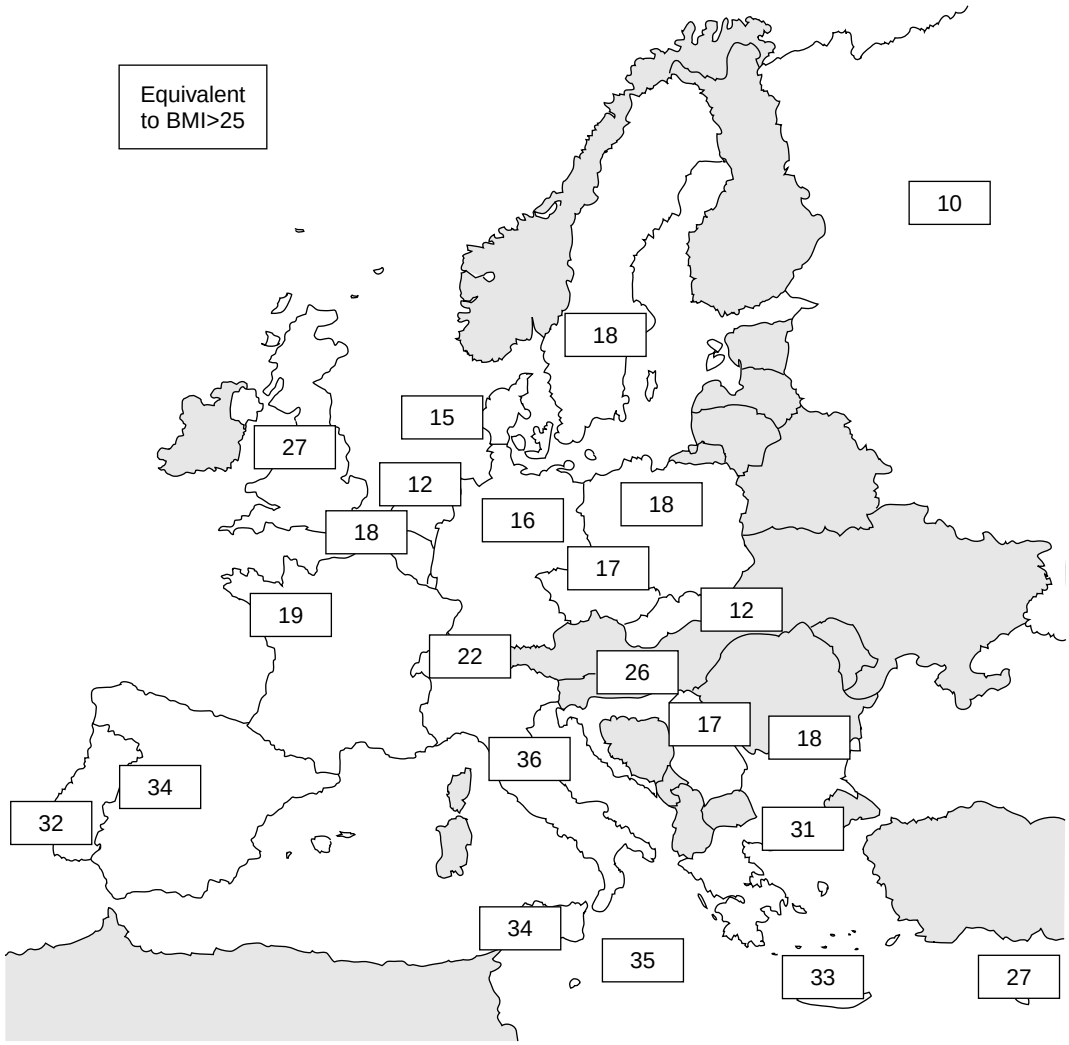

Figure 7: Prevalence of overweight (%) in children aged 7–11 years in selected European countries in 2002 (IOTF data).
Source: With kind permission of IOTF.

The prevalence was higher among the older subjects. Another survey carried out in 1987 using the same methodology showed an alarming increase in the prevalence of obesity (BMI > 30), which overall had increased from 6 to 8% in men and from 8 to 12% in women. The increase occurred in all age groups, but particularly among women aged 25–34 years, in whom the prevalence appears to have doubled over the seven-year interval. From 1990 there has been a regular cycle of surveys (Health Surveys for England) which continue to show an increasing prevalence of obesity: the trend up to 2002 (which is the latest year for which data are available at the time of writing is shown in Table 5. The data on prevalence become available about two years after the fieldwork is done, and the prevalence among adult men and women in the UK has increased by approximately one percentage point annually over the past decade. It is therefore

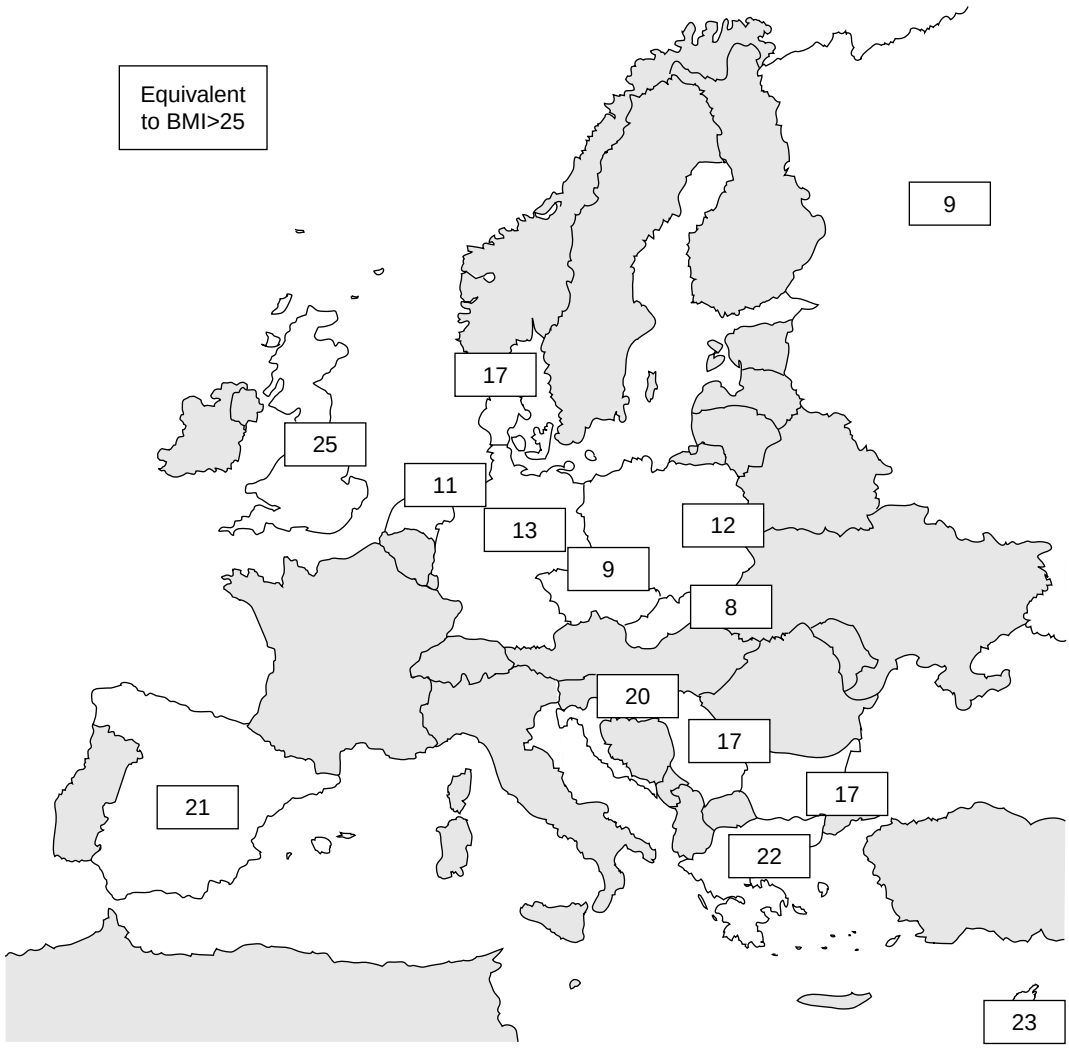

Figure 8: Prevalence of overweight (%) in children aged 14–17 years in selected European countries in 2002 (IOTF data).
Source: With kind permission of IOTF.

reasonable to estimate that the prevalence will be more than 25% among both men and women by the year 2005. Serial surveys in other countries show a similar trend.

It is evident that, since the first survey in 1980, the prevalence of obesity in the UK has more than tripled among both men and women. The general level of obesity in affluent countries ranges from the strikingly low values in Japan, through moderate levels in Netherlands and Sweden, to the high levels in USA and Australia. Among less affluent countries there is also a huge range from low levels in Brazil to astonishingly high levels in Samoa. Despite these differences the trend towards increasing obesity is evident everywhere, whatever the baseline level.

Table 5: Prevalence of obesity (%) in England 1980–2002.

BMI	1980	1993[a]	2000[a]	2002[a]
Men				
Healthy weight: 20–25	Not available	37.8	29.9	29.6
Overweight: 25–30	Not available	44.4	44.5	43.4
Obese: > 30	6	13.2	21.0	22.1
Morbidly obese: > 40	Not available	0.2	0.6	0.8
Women				
Healthy weight: 20–25	Not available	44.3	39.0	37.4
Overweight: 25–30	Not available	32.2	33.8	33.7
Obese: > 30	8	16.4	21.4	22.8
Morbidly obese: > 40	Not available	1.4	2.3	2.6
	1984[b]	1994[b]	2000[c]	2001/2[e]
Boys				
Healthy weight: 20–25	94.0[d]	89.3[d]	74.9[d]	72.0[d]
Overweight: 25–30	5.4	9.0	20.3	22.0
Obese: > 30	0.6	1.7	4.8	6.0
Morbidly obese: > 40	Not available	Not available	Not available	Not available
Girls				
Healthy weight: 20–25	89.4[d]	83.9[d]	66.6[d]	63.0[d]
Overweight: 25–30	9.3	13.5	26.6	29.0
Obese: > 30	1.3	2.6	6.8	8.0
Morbidly obese: > 40	Not available	Not available	Not available	Not available

Sources:
[a] Health Survey for England, various years.
[b] Data for children aged 4–12 in England only. Data extracted from POST report 199, September 2003, Improving children's diet – trends pp. 23–4.
[c] Data for children aged 2–19 in England only. Data extracted from the national office for statistics website: (www.statistics.gov.uk/cci/nugget.asp?id=718), diet and nutrition section.
[d] Healthy weights are calculated as 100 – (%obese + %overweight), and thus include those with BMI < 20.
[e] Data provided by IOTF (personal communication).

Relation to age, social class and region

Adults

Sex

In those aged 16–24 years, mean BMI in men is 23.8 in men, and 24.2 in women. Among those aged 16–64 years, mean BMI is 26.2 kg/m^2 in men and 25.8 kg/m^2 in women. In all adults, 65.5% of men and 56.5% of women are overweight or obese. A greater proportion of men (43.4%) than women (33.7%) are overweight, but slightly lower proportions of men (22.1%) than women (22.8%) are obese. Among those aged 16–64 years, 21.4% of men and 21.9% of women are obese.

Age

Men and women tend to reach a maximum prevalence of obesity between 55–64 years, after which BMI starts to decline.

Social class

The prevalence of obesity is inversely related to social class, but there is no satisfactory explanation for this observed association.

Region

There are no marked regional differences in the prevalence of obesity in the UK when the effects of age, social class and smoking habit are allowed for. In men aged 16–24 years, the prevalence of being overweight is lowest in the South East (19.8%) and highest in Yorkshire and Humber (28.4%), while in women it is lowest in London (18.5%) and highest in East England (24.3%). The prevalence of obesity in men is lowest in the South West (5.9%) and highest in the North East (14.5%). In women, obesity is lowest in London (9.3%) and highest in the West Midlands (17.3%).

Smoking and ethnicity

Data on obesity by smoking or ethnicity in 2002 is not published in the *Health Survey for England*.

Incidence

Incidence of obesity is impossible to measure, since there is so much variability in weight with time in any individual. At any moment there will be quite a lot of people oscillating above and below any given threshold of severity.[69]

Children

Sex and age

Using BMI charts (published by the Child Growth Foundation, London), 5.5% of boys and 7.2% of girls aged 2–15 were obese in 2002. Mean BMI falls between age 2 and age 5 or 6. It then increases fairly rapidly to adulthood. Compared with males, females have a slightly higher BMI during adolescence.

Social class, region and ethnicity

Data on childhood obesity by social class, region, and ethnicity in 2002 are not published in the *Health Survey for England*.

Incidence

Incidence of obesity is even more difficult to measure in children since variability in weight and height is greater during growth.

5 Services available and their costs

What happens at present and why?

Obesity has features that do not apply to the other major causes of ill health and disability (e.g. hypertension, cancer or diabetes) with which the health service should cope. A patient with such an illness has probably been diagnosed by a doctor, and looks to medical aid (NHS or private or alternative) for treatment. The problem is one that is known only to those whom the patient chooses to inform, and it carries no social stigma.

In contrast, obesity is usually a condition the patient has self-diagnosed, it is obvious to the public and it does carry a social stigma in many cases. It is unusual for the obese person to seek medical aid in the first instance. The media is full of advice on what to do, so typically the obese person who eventually has a medical consultation gives a history of a series of self-help measures, which did not have a satisfactory outcome. This gives doctors the impression that the self-help measures are invariably ineffective, but this is not so: medical referrals are always self-help failures, but self-help successes do not register in the medical statistics. Field studies of unselected populations show that those who are now overweight were often previously normal weight, and those now of normal weight were often previously overweight.[69]

So, from the viewpoint of the primary care physician seeing an obese patient in surgery, the patient has typically 'tried everything' without satisfaction, and hopes that the GP will have a pill which will solve the problem. The patient already carries a large amount of misinformation about obesity: especially about the rate of weight loss which is to be expected, since this is routinely exaggerated by some commercial weight-loss organisations and magazines. The patient is a self-help failure who may well have been told by the previous therapist that his or her failure indicates a rare metabolic abnormality with which he or she expects the GP to deal. This is not a basis on which a successful consultation can be expected.

This unsatisfactory situation is made much worse by the fact that the GP has had little or no training on how to manage obesity unless, in exceptional circumstances, they attended a postgraduate course,[70] and has no clinical guidelines on which to anchor the consultation and help with decision making.

The next part of this section outlines, for doctors (GPs and consultants), practice nurses (PNs) and dietitians, information about treatments offered and delivery of this service. As there are no significant services that specifically tackle the prevention of obesity or childhood obesity in the UK, the information below is restricted to the treatment of adult obesity. The final part of this section deals with the cost of obesity.

How is obesity managed in primary care?

The first NHS port of call for the obese patient is the GP who can manage the patient him or herself, or refer them to a PN or a dietitian. Information is available on how GPs and PNs manage obesity from a survey conducted by the National Audit Office in 1999.[6] A sample of 1200 GPs and 1200 PNs were surveyed by post, and 20 GPs and 16 PNs took part in face-to-face interviews. The responses showed that management of obesity within general practice consists broadly of three types, depending on the degree of obesity and the extent of clinical complications:

- general lifestyle advice
- general lifestyle advice plus drug therapy
- onward referral to specialist.

Of note, only 4% of GPs used a protocol for managing overweight or obese patients, and many of those in use had been developed independently by the practice. Figure 9 shows the most frequent responses to the question 'What factors might help you in developing further your approach to managing your overweight and obese patients'. 63% of GPs and 85% of PNs believed that national clinical guidelines or a protocol would be 'useful' or 'very useful'. Since this NAO survey was carried out, many PCTs have now developed their own obesity strategy. However, these are variable in quality, and rarely evidence based. There is no formal system for logging local and regional obesity strategies, but the Association for the Study of Obesity website (www.aso.org.uk) provides a list of those made available to them, and links to the documents if possible. The good news is that the National Institute for Clinical Excellence (NICE) is committed to produce guidance for the prevention and treatment of obesity by the end of 2007.

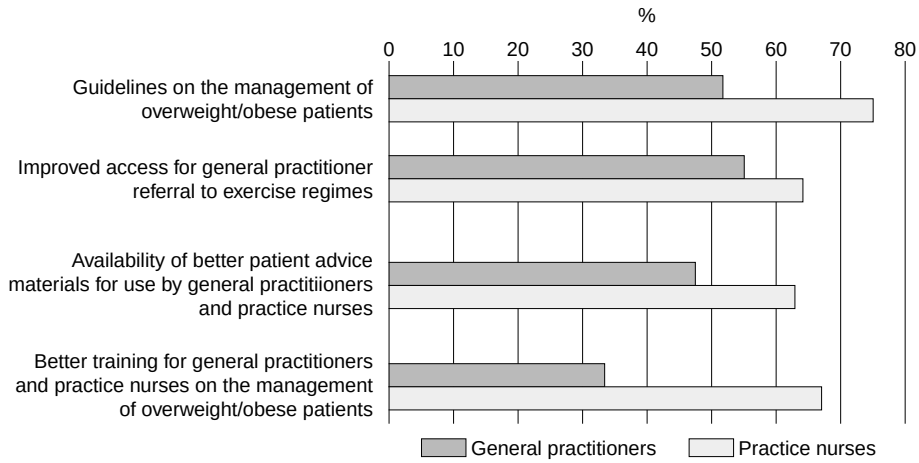

Figure 9: Factors which general practitioners and practice nurses said would assist them in advising and treating patients.[6]

66% of GPs felt that treating patients for overweight or obesity was the responsibility of the primary care team, and 75% thought they had a role to play in referring obese patients to appropriate specialists. [2% neither treated obese patients personally nor referred them to specialists.] Figure 10 shows the referral options most commonly used by GPs. Since this NAO survey was carried out, many PCTs have developed possible referral options to alleviate pressure on the primary care team. Some PCTs now have arrangements with commercial weight loss companies, some have helped develop community based programmes/self-help groups, and many have developed their exercise on prescription service. Indeed, improved access for referral to exercise regimes was a factor which GPs and PNs said would assist them in managing their overweight and obese patients (Figure 9). It is worth noting that access to the referral options mentioned above is variable by region.

68% of PNs and 33% of GPs stated that better training would assist them in managing their overweight and obese patients (Figure 9). This important point is picked up later in this section.

Referral to a hospital consultant who runs a specialist obesity clinic is unusual, simply because there are so few specialist obesity clinics. In the UK in 2004 there were 10 clinics for adults and 4 for children. Kopelman has argued that there should be more obesity clinics in the NHS.[71] Specialist obesity clinics are usually multidisciplinary, and often have a (limited) facility to refer patients on to a clinical psychologist or surgeon. An audit of one such specialist clinic (run by Prof. Kopelman at the Royal London Hospital)

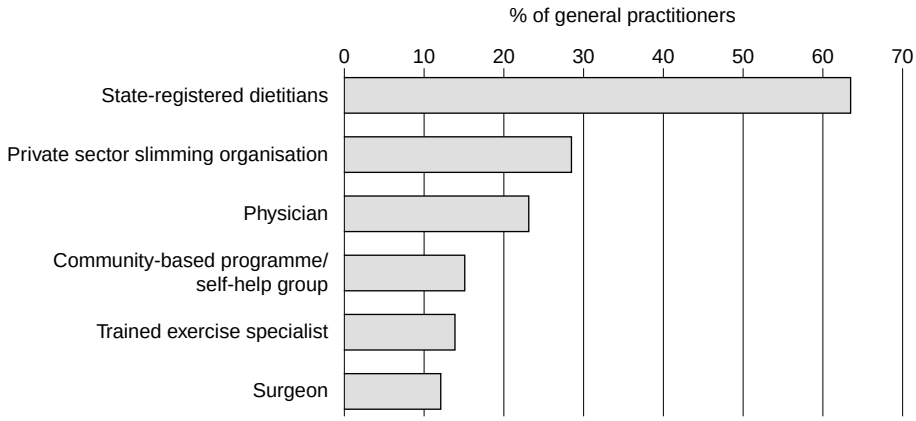

Figure 10: Referral options most commonly used by general practitioners.[6]

showed that 69% of patients were treated by diet alone, 23% by diet and anti-obesity drugs, and 7% by psychological interventions.[72,73] Surgical treatment for obesity in the UK is rare, and procedures are carried out at variable rates across the country.[74]

What do dietitians do?

From a survey conducted in 1998, only one out of 99 NHS dietetic services questioned did not accept overweight or obese client referrals for management.[73] The majority (87%) of respondents allocated up to 30 minutes for a new client appointment. Although 27% had no specific policy about the number of follow-up appointments during a 'course' of treatment, 20% offered one or two follow-up appointments; 35% offered three or four; 16% offered five or more, and only 2% offered no follow-up. Most follow-up appointments were every four weeks (8% were three weeks or more frequently; 50% were every four weeks; 35% were five weeks or less frequently, and data for 7% were missing). The duration of follow-up appointments was as follows: 24% were allocated less than 11 minutes; 73% 11–20 minutes; 2% 21–30 minutes).

The majority of dietetic services did not prioritise (or match) patients for different dietary treatments. Of those who did, criteria such as existing disease, other risk factors, diagnosed eating disorder, degree of fatness, weight history and mental health or learning difficulties were used to identify different approaches for management. However, no common strategies for matching patients to treatments could be identified. Most dietetic services (78%) saw overweight or obese clients only on an individual basis; 20% offered treatment on a combination of individual and group styles. Very few (only 23%) had a protocol, although some others (20%) stated that they currently had protocols under development.

The economic cost of obesity

The National Audit Office[6] conducted a survey to estimate the cost (direct and indirect) of obesity in England in 1998.

Direct costs

The direct costs of obesity were defined as the costs to the NHS of 1) treating obesity and 2) treating the associated disease that can be attributed to it. These direct costs were estimated by taking a prevalence-based, cost-of-illness approach based on extensive literature review and relying on published primary data.

1) The cost of treating obesity covered the cost of consultations with GPs related to obesity, the cost of hospital attendances, and the cost of drugs prescribed to help obese patients lose weight. Sources of data used for calculations are listed in the NAO report.[6] The direct costs of treating obesity in England in 1998 were estimated to be £9.4 million at 1998 prices (Table 6).

   Of note, this figure of £9.4 million is probably a gross underestimate for 2005 for a number of reasons. First, the number of GP consultations related to obesity probably matches the prevalence of obesity in the population, and as the prevalence of obesity has increased since 1998, one can assume that so has the number of GP consultations relating to obesity. Second, the data do not include the cost of consultations with practice nurses or dietitians. Since the amount of time spent by practice nurses on monitoring and advising patients exceeds that spent by most GPs, the cost of primary care interventions for obesity is likely to be significantly greater than that indicated by GP consultations alone. Third, at the time of the NAO survey (1998), orlistat had just been licensed in the UK (in Autumn of 1998), and sibutramine was yet to be licensed. Therefore, the annual cost of prescriptions for these anti-obesity medications in 1999 and beyond is likely to be considerably higher than £0.8 million.

Table 6: Total cost of obesity in England in 1998.[6]

Cost component	**Cost (£m)**
Cost of treating obesity	
General Practitioner consultations	6.8
Ordinary admission	1.3
Day cases	0.1
Outpatient attendances	0.5
Prescription	0.8
Total costs of treating obesity	9.5
Cost of treating the consequences of obesity	
General Practitioner consultations	44.9
Ordinary admissions	120.7
Day cases	5.2
Outpatient attendances	51.9
Prescription	247.2
Total costs of treating the consequences of obesity	**469.9**
Total direct costs	**479.4**
Indirect costs	
Lost earnings due to attributable mortality	827.8
Lost earnings due to attributable sickness	1,321.7
Total indirect costs	**2,149.5**
GRAND TOTAL	**2,628.9**

2) The cost of treating the consequences of obesity covered the cost of treating diseases such as CHD. The cost of treating these diseases was estimated by calculating the relevant population attributable risk proportion. The proportion of these diseases in the population that was attributable to obesity was then estabilised using systematic review. [Of note, there were a number of potentially important disease areas that were excluded from this analysis because of a lack of data, e.g. depression and back pain.] Further details of methodology used are provided in the NAO report.[6] The direct costs of treating the consequences of obesity in England in 1998 were estimated to be £469.9 million at 1998 prices (Table 7).

Table 7: The cost of treating the consequences of obesity in England in 1998.[6]

	Attributable cases (% of total cases)	Cost of general practitioners (£m)	Cost of hospital contacts (£m)	Cost of prescriptions (£m)	Total cost (£m)	Proportion of total costs (%)
Hypertension	794,276 (36)	25.5	7.7	101.6	134.8	29
Type II diabetes	270,504 (47)	7.9	36.7	78.9	123.5	26
Angina pectoris	90,776 (15)	2.8	35.3	46.6	84.7	18
Myocardial infarction	28,027 (18)	0.6	41.6	0.0	42.2	9
Osteoarthritis	194,683 (12)	4.7	14.5	15.6	34.8	7
Stroke	20,260 (6)	0.5	15.7	0.5	16.7	4
Gallstones	8,384 (15)	0.2	10.2	0.4	10.8	2
Colon cancer	7,483 (29)	0.4	10.0	0.0	10.4	2
Ovarian cancer	1,543 (13)	0.1	3.8	0.1	4.0	1
Gout	96,549 (47)	2.2	0.0	1.7	3.9	1
Prostate cancer	809 (3)	0.0	0.9	1.7	2.6	1
Endometrial cancer	834 (14)	0.0	1.1	0.1	1.2	0
Rectal cancer	126 (1)	0.0	0.2	0.1	0.3	0
Total		**44.9**	**177.7**	**247.3**	**469.9**	**100**

Indirect costs

Earnings lost due to premature mortality

The NAO estimated that over 31 000 deaths in England in 1998 were attributed to obesity; approximately 6% of all deaths. This represents over 275 000 life years lost due to obesity. Some 9000 of these deaths occurred before the age of 65, resulting in a loss of over 40 000 years of working life up to state retirement age alone. The associated lost earnings in England due to obesity in 1998 was estimated to be £827 million at 1998 prices.

Earnings lost due to sickness absence

The NAO estimated that there were over 18 million days of sickness attributable to obesity in 1998. Further details of methodology used are provided in the NAO report.[6] Of note, this is almost certainly an underestimate since the days of absence recorded were only based on medically certified days of incapacity where a claim to benefit was made (no data on self-certified days of sickness were available). On this basis, estimated lost earnings due to sickness absence attributable to obesity in England in 1998 was £1322 million at 1998 prices.

In summary, the direct cost of obesity to the NHS in England in 1998 was at least £480 million, equivalent to about 1.5% of NHS expenditure in that year (Table 6). The direct cost is driven primarily by the cost of treating the secondary diseases attributable to obesity, which accounted for 98% of the total. The most significant cost drivers by far are CHD, hypertension, and type II diabetes, followed by osteoarthritis and stroke. The indirect cost of obesity in England in 1998 represented by lost earnings was estimated to be £2149 million. Combining the direct and indirect costs, the total cost of obesity in England in 1998 was £2.6 billion, or 0.3% of UK Gross Domestic Product.

Cost of individual treatments

More detailed information on the costs of individual treatments for obesity are provided in a recently published report.[7] This report reviewed systematically health economic evaluations of obesity treatments and assessed the cost to the NHS of these treatments. The report concluded that:

Targeting high-risk individuals with drugs or surgery was likely to result in a cost per additional life-year or quality-adjusted life-year (QALY) of no more than £13 000. There was also suggestive evidence of cost-savings from treatment of people with type II diabetes with metformin. Targeting surgery at people with severe obesity and impaired glucose tolerance was likely to be more cost effective, at £2329 per additional life-year.

Economic modelling of diet and exercise over 6 years for people with impaired glucose tolerance was associated with a high initial cost per QALY, but by the sixth year the cost per QALY was £13 389. Results were sensitive to the quality of life weights, for which there were very limited data. Results did not include cost savings from disease other than diabetes, and therefore may be conservative.

The cost of diet and exercise together appear comparable to treatments, for example drugs, in obese individuals with risk factors, such as impaired glucose tolerance.

6 Effectiveness of services and interventions

Assessment of effectiveness

To assess the effectiveness of interventions for the treatment of obesity we have relied primarily on systematic reviews of RCTs. Randomised controlled trials are (almost) the best source of evidence for assessing the efficacy of healthcare interventions, including lifestyle interventions. Results from systematic reviews (and meta-analysis) of several RCTs which address the same question are even better.[75] A conclusion based on several RCTs, selected by previously defined criteria, is likely to be nearer the truth than one based on a group of RCTs which happen to support the reviewer's own prejudices.

There have been a wealth of good systematic reviews (and reviews of reviews) on the treatment of obesity published in the past few years, and a number of others were published in 2004/5 (Table 8a). [NICE is committed to produce guidance for the prevention and treatment of obesity by the end of 2007.] For the purpose of this chapter, we have chosen to use information and data from the most recent of the good systematic reviews listed in Table 8b. Thus, we have used the information and data from the Health Technology Assessment (HTA) review on obesity[7] for evidence of the efficacy of treatments for obesity in adults. We have used the information and data from two Cochrane reviews for evidence of the efficacy of interventions for the prevention[76] and treatment[77] of obesity in children. We have used the information and data from the Health Development Agency review of reviews on obesity[59] for evidence of the efficacy of interventions for the prevention of obesity in adults.

The findings from these four reviews are all based on sound methodology, but vary a little in their inclusion criteria; these differences are highlighted in Table 8b. Full reports of these four reviews contain details of the methodologies they employed and findings. All four reports are available free at the relevant websites (Table 8a).

Table 8a: Systematic reviews on the management of obesity published from 2002.

Source (country)	Referenced as	Population	Year published	Search date	URL
Systematic reviews					
Calgary (Canada)	Calgary	Children	2004	2004	www.calgaryhealthregion.ca
HTA (UK)	HTA	Adults	2004	Spring 2001	www.ncchta.org
NHMRC (Australia)	NHMRC	All	2003	May 2001	www.obesityguidelines.gov.au
US PSTF (US)	USPSTF	Children	2004	Feb 2003	www.ahrq.gov/clinic/uspstfix.htm
US PSTF (US)	USPSTF	Adults	2003	Feb 2003	www.ahrq.gov/clinic/uspstfix.htm
SBU (Sweden)	SBU	All	2002	2002	www.sbu.se
Cochrane (International)	Summerbell *et al.*	Children	2003	July 2002	www.nelh.nhs.uk
SIGN (Scotland)	SIGN	Children	2003	Dec 2001	www.sign.ac.uk
NHS CRD (UK)	NHS CRD	Children	2002	July 2002	www.york.ac.uk/inst/crd
Cochrane (International)	Campbell *et al.*	Children	2002	July 2002	www.nelh.nhs.uk
HTA-surgery (UK)	HTA-surgery	Adults	2002	Summer 2001	www.ncchta.org
HTA-sibutramine (UK)	HTA-sibutramine	Adults	2002	June 2000	www.ncchta.org
HTA-orlistat (UK)	HTA-orlistat	Adults	2001	June 2000	www.ncchta.org
Reviews of reviews					
CIHR (Canada)	CIHR	All	2003	May 2003	www.caphc.org/partnerships_obesity.html
HDA (UK)	HDA	All	2003	Oct 2002	www.hda.nhs.uk/evidence

The outcome presented in this chapter is weight loss. This is not, of course, the only measure of efficacy in a treatment for obesity, but it is one measure of outcome that is normally reported, and this enables us to make comparisons between treatments.

Estimates of the effectiveness, where known, are graded by the size of the effect where:

Mean weight loss attributable to intervention	
A* = large weight loss	>10 kg
A = strong beneficial effect	5–10 kg
B = moderate beneficial effect	2–5 kg
C = measurable beneficial effect	Significant difference
D = no measurable effect	No significant difference
E = harms of the treatment outweigh its benefits	

Table 8b: Inclusion criteria for systematic reviews from which information was used for this chapter.

Reference	Population and intervention	Study design	Minimum follow-up
HTA (2004)[7]	Treatment of obesity in adults	RCTs	At least 1 year
Campbell *et al.* (2004)[76]	Prevention of obesity in children	RCTs and CCTs	At least 3 months
Summerbell *et al.* (2004)[77]	Treatment of obesity in children	RCTs	At least 6 months
HDA (2003)[59]	Treatment and prevention of obesity in adults & children	various	various

and quality of the supporting evidence where:

I-1 = Evidence from several consistent or one large RCT(s)
I-2 = Evidence obtained from at least one properly designed RCT
II-1 = Evidence obtained from well-designed controlled trials without randomisation, or from well-designed cohort or case-control analytic studies
II-2 = Evidence obtained from multiple time series with or without the intervention
III = Opinions of respected authorities, based on clinical experience, descriptive studies, or reports of expert committees
IV = Evidence inadequate and conflicting

Effectiveness of treatments

Obesity inevitably follows a prolonged period during which energy intake (in food and drink) exceeds energy output (in resting metabolism, exercise, metabolic response to various thermogenic stimuli, and energy losses in excreta). Therefore effective treatment inevitably requires a reversal of energy balance, so output exceeds input, and body fat is burned off to meet the deficit. However it has recently been recognised that a large part of the cause of the 'global epidemic' of obesity is environmental. The availability and commercial promotion of cheap palatable snack foods tends to increase energy intake. The labour-saving devices that reduce the need for manual work, and the increasing tendency to watch, rather than participate in, vigorous leisure activities tends to decrease energy output. Together these tendencies create an 'obesogenic' environment.

For estimates of effectiveness of treatments for obesity in this chapter we have relied primarily on a systematic review of RCTs in HTA 2004.[7] The authors of this review adopted the entry criteria that NICE suggest for drug trials, and therefore included only randomised controlled trials of people with BMI > 28 kg/m^2 at entry and a follow-up for at least 1 year. It is very difficult to conduct trials of dietary or lifestyle treatments that meet these criteria, since the control group are required to make no effort to control their obesity for a year, which is an unrealistic requirement. Having set these selection criteria the authors comment: 'Limitations in the evidence available for the reviews, particularly inadequate sample size and reporting, lack of long-term follow-up and few quality of life data, mean that most results should be interpreted with caution.' We have therefore used data from other sources that HTA 2004 excluded. For each type of treatment the effectiveness, and the quality of the supporting evidence, is graded as described in the above section.

Since dietary treatments are economical, widely used and suitable for the great majority of obese people, they will be discussed first, then exercise, drug treatment, behaviour therapy, surgery and various combinations of these treatments.

Effectiveness of patient-centred interventions

Dietary treatments

The largest intentional weight loss in an obese patient ever reported in a peer-reviewed medical journal was achieved on a diet supplying 800 kcal/day for 2 years.[78] The patient's weight fell from 310 kg to 90 kg as an inpatient in a metabolic unit. Such treatment today is economically and socially impractical, but this exceptional case shows that there is no limit to the amount of weight that can be lost by dieting, if the patient can actually keep to the diet.

Effects of 600 kcal/day deficit or low-fat diet versus control

This 'slightly hypocaloric' diet[79] was extensively used as a 'control' diet in RCTs of drug treatments (see below). Theoretically, if a person consumes a diet supplying 600 kcal/day less than expenditure their weight should decrease by 600 g/week, or 31 kg at 12 months, since the excess weight in obese people has an energy value of 7000 kcal/kg.[80] The observed weight losses in the trials below are much less than this theoretical value, indicating that the treatment subjects did not strictly observe the diet, or that the control group were not strictly non-dieting.

Listed below are meta-analyses of RCTs reviewed in HTA 2004,[7] in which can be found references for the individual trials. This review analyses the change in body weight (kg), total cholesterol, LDL cholesterol, HDL cholesterol, triglyceride, HbA_{1c}%, fasting plasma glucose and blood pressure. Only weight changes will be quoted here, since the beneficial changes in risk factors (where available) are closely proportionate to the weight change.

A meta-analysis of 12 RCTs found that, compared with non-dieting controls, the 600 kcal/day deficit or low-fat diets were associated with an overall weighted mean difference (WMD) weight change at 12 months of –5.31 kg (95% CI –5.86 to –4.77 kg). There was evidence of statistical heterogeneity, although the direction of effect was consistent across all studies. When 12-month weight changes from studies with imputed values were compared with studies with no assumed values, the weight changes were –4.52 kg (95% CI –5.67 to –3.36 kg) compared with –5.55 kg (95% CI –6.17 to –4.94 kg). When 12-month weight loss from RCTs with participants with cardiovascular risk factors was compared with RCTs with participants with no reported risk factors, a clearer difference between studies emerged (–4.19 kg, 95% CI –4.90 to –3.48 kg; compared with –6.98 kg, 95% CI –7.83 to –6.12 kg, respectively).

At 18 months weight change was –1.15 kg (95% CI –2.76 to 0.45 kg), 24 months –2.35 kg (95% CI –3.56 to –1.15 kg), 30 months 0.70 kg (95% CI –1.78 to 3.18 kg), 36 months –3.55 kg (95% CI –4.54 to –2.55 kg) and at 60 months –0.20 kg (95% CI –2.03 to 1.63 kg). After 12 months only a maximum of three studies provided data towards any one comparison.

Quality of supporting evidence = I-1 Size of effect = A

In the cluster RCT the weight change at 12 months was –0.88 kg (SD 4.0 kg) for the diet group and 1.3 kg (SD 3.0 kg) for the control group, which was not found to be a statistically significant difference.

The two studies that were associated with the least mean difference in weight change also had populations with the largest mean BMI of 34.0 kg/m.^{2}

One death occurred in the control arm of the Hypertension Prevention Trial (HPT) study and two deaths in the intervention arm of the TAIM Phase I study. Two deaths in the intervention group and one in

the control group occurred in the study by Hankey and colleagues, three deaths in the Hypertension Optimal Treatment (HOT) study and four deaths in the first year of the study by Swinburn and colleagues (group allocation not known). One diagnosis of cancer occurred in year 2 of the study by Wood and colleagues, and two cancers and one cardiac event in the Oslo Diet and Exercise Study (ODES) (allocation unknown).

Swinburn and colleagues found that 47% of the participants developed diabetes or impaired glucose tolerance, compared with 67% in the control group. After 1 year the investigators for the Dietary Intervention Study of Hypertension (DISH) reported that 59.5% of participants allocated to diet remained off medications, compared with 35.3% of controls (reported p=0.0015). The investigators for the HOT study also reported that people in the diet intervention arm required fewer medications between 1 year and 30 months, a difference that was consistently statistically significant. In the HPT 9% of intervention and control groups required drug treatment for hypertension during the 3 years of study.

One study reported no effect of diet counselling by doctor and dietitian or dietitian alone on subsequent use of medication. The same study reported that the cost of an extra kilogram weight loss was Aus$9.76 for the doctor/dietitian group and Aus$7.30 for the dietitian group.

Effects of low-calorie diet (LCD) versus control

An RCT on cancer patients showed that, compared with the control group, the LCD was associated with a WMD weight change at 12 months of –6.25 kg (95% CI –9.05 to –3.45 kg), at 24 months of –7.00 kg (95% CI –10.99 to –3.01 kg) and at 36 months of –6.10 kg (95% CI –10.71 to –1.49 kg).

Quality of supporting evidence I-2 Size of effect = B

Three breast cancers occurred in the intervention group and one in the control group. Three people died from breast cancer in the intervention group and five people in the control group. There were two deaths from other causes in each of the two groups.

Effects of very low-calorie diet (VLCD) versus control in an obese population with asthma

At 12 months VLCD compared with control was associated with a WMD weight change of –13.40 kg (95% CI –18.43 to –8.37 kg). After 1 year the difference in forced expiratory volume in 1 second between VLCD and control groups was 7.6% (95% CI 1.5 to 13.8%), forced vital capacity 7.6% (95% CI3.5 to 11.8%) and peak expiratory flow 6.2% (95% CI –1.4 to 13.7%).

Quality of supporting evidence = I-2 Size of effect = A*

During the year of follow-up 18 out of 19 participants in the control group and 16 out of 19 participants in the VLCD group had at least one exacerbation of asthma. The median number of exacerbations was 1 (range 0–7) in the control group and 1 (range 0–4) in the VLCD group (reported p=0.001). Overall reduction in rescue medication was 0.5 doses in the VLCD group and zero doses in the control group. Thirteen out of 19 participants in the control group needed a course of oral steroids during the year and ten out of 19 participants in the VLCD group.

Effects of low-calorie diet versus 600 kcal/day deficit or low-fat diet

At 12 months an LCD compared with a low-fat diet was associated with a WMD weight change of 1.63 kg (95% CI –1.26 to 4.52 kg).

Effects of very low-calorie diet versus low-calorie diet

Compared with LCD, VLCD was associated with an overall WMD weight change at 12 months of –0.15 kg (95% CI –2.73 to 2.43 kg) and at 18 months of –1.13 kg (95% CI –5.32 to 3.06 kg). Thus, there was no evidence to suggest that VLCD was associated with a significantly greater weight loss than LCD at any of the time-points.

Effects of protein-sparing modified fast (PSMF) versus low-calorie diet

At 12 months the PSMF compared with LCD was associated with an overall WMD weight change of –3.57 kg (95% CI –7.36 to 0.22 kg), at 18 months 0.69 kg (95% CI –1.58 to 2.96 kg), at 24 months of –2.17 kg (95% CI –4.88 to 0.54 kg), at 36 months of –1.51 kg (95% CI –5.43 to 2.41 kg) and at 60 months of 0.20 kg (95% CI –5.68 to 6.08 kg). There were no statistically significant changes in lipids at 18 months in one study in diabetics, although the same study found an association between the PSMF diet and reduced HbA_{1c} of –2.60% (95% CI –4.36 to –0.84%) and fasting plasma glucose of –4.5 mmol/l (95% CI –7.07 to –1.93 mmol/l) at 18 months.

Quality of evidence = I-2 Size of effect = B

Effects of 220 kcal vs 800 kcal diet for 24 weeks[81]

In the 20-week outpatient phase of this trial obese women on a diet supplying 220 kcal/day lost 19.9 kg, and those on 800 kcal/day lost 14.2 kg. However lean tissue contributed a larger proportion of the weight lost in the former group.

Quality of evidence II-1 Size of effect A*

Effectiveness of exercise

Effects of diet and exercise versus control

In these studies initial BMI was 27.9 to 31.3 kg/m^2, and the diet was either low fat, or one designed to cause a 600 kcal/day energy deficit. The exercise was up to three supervised sessions of 45–60 minutes weekly. Diet plus exercise versus no treatment was associated with an overall WMD weight change at 12 months of –4.78 kg (95% CI –5.41 to –4.16 kg). Weight loss at 24 months was still evident, with diet plus exercise associated with a WMD weight change of –2.70 kg (95% CI –3.60 to –1.80 kg).

Diet plus exercise compared with controls demonstrated a statistically significant effect on lipids, blood pressure and fasting plasma glucose at 12 months, and fasting plasma glucose at 24 months.

Quality of evidence = I-1 Size of effect = B

Effectiveness of drug treatments

The intervention which is most readily investigated by RCT is a drug trial, since either active drug or placebo can be administered in randomised double-blind conditions. Furthermore drug trials are usually very well managed, since the licensing of a new drug depends on the quality of evidence of efficacy presented to the licensing authority. In the UK at present (2004) only two drugs are licensed for the treatment of obesity.

Orlistat

This drug is from Roche Laboratories – trade name Xenical. It inhibits the activity of intestinal lipase, and thus reduces by about 30% the digestion and absorption of dietary fat from the gut.

Effects of orlistat 360 mg/day and diet versus placebo and diet

The added effect of orlistat 360 mg/day on weight reduction produced an overall WMD weight change at 12 months of –3.01 kg (95% CI –3.48 to –2.54 kg).

Quality of evidence = I-1 Size of effect = B

The added effect of orlistat 360 mg/day on weight maintenance produced an overall weight change after 12 months of –0.85 kg (95% CI –1.50 to –0.19 kg) with evidence of heterogeneity in these four studies.

All the risk factors for the 1-year weight reduction phase showed beneficial changes, except for HDL cholesterol, which showed a small decrease, and triglycerides (TGs). There was evidence of heterogeneity for HbA_{1c}, fasting plasma glucose and TGs for this 12-month weight reduction phase. This may have related to the inclusion of people with diabetes in two studies. After 12 months of orlistat in people with diabetes a change in HbA_{1c} of –0.27% (95% CI –0.38 to –0.15%) compared with the control group was observed, and –0.11% (95% CI –0.20 to 0.02%) for non-diabetics compared with controls. Similarly, for fasting glucose the observed change was –0.58 mmol/l (95% CI –0.80 to –0.36 mmol/l) for diabetics compared with controls and –0.16 mmol/l (95% CI –0.27 to –0.05 mmol/l) for non-diabetics. However, for TGs observed changes were less marked between diabetics compared with controls (–0.05 mmol/l, 95% CI –0.19 to 0.09 mmol/l) and non-diabetics (0.05 mmol/l, 95% CI –0.03 to 0.14 mmol/l). For the 12-month weight maintenance phase there were no added benefits on risk factors.

Two weight reduction studies produced an overall WMD weight change at 24 months of –3.26 kg (95% CI –4.15 to –2.37 kg). By 24 months the beneficial effects of orlistat on risk factors were still seen, with the exception of triglycerides and systolic blood pressure (SBP).

One death occurred in the orlistat arm of the study by Broom and colleagues from cancer and one death in the orlistat arm of the study by Hauptman and colleagues from acute myocardial infarction (MI). One death from brainstem infarction occurred in the orlistat arm of the study by Lindgarde and colleagues.

Davidson and colleagues reported four cases of breast cancer in year 1, three of these cases in participants treated with orlistat and one case in a participant treated with placebo (one in each group had evidence of breast cancer on mammograms before study entry). Rossner and colleagues reported one participant with cholelithiasis. Rossner also reported one participants with breast cancer in the placebo group and three participants with breast cancer in the 120-mg orlistat group (of whom two had mammogram evidence of cancer before recruitment). Sjostrom and colleagues reported one participant with gastrointestinal cancer in the placebo/placebo group during the 2 years of the study.

All the orlistat studies reported gastrointestinal adverse events, such as oily stools and faecal incontinence, to be more common in the orlistat groups than in the placebo groups.

In two studies vitamin supplementation was routinely given to all participants. Where reported, vitamin supplementation *per protocol* was always required more commonly in the orlistat groups than in the placebo groups.

Hollander and colleagues reported that the average dose of oral sulfonylureas decreased more in the orlistat than in the placebo group (–23% versus –9%, respectively, p=0.0019).

Effects of orlistat 360 mg/day for 52 weeks and diet versus placebo for 24 weeks and diet then orlistat 360 mg/day for 28 weeks and diet

At 12 months 52 weeks of orlistat 360 mg/day was associated with a WMD weight change of –0.69 kg (95% CI –2.85 to 1.47 kg) compared with the placebo/orlistat group. The orlistat group had changes at 12 months in

total cholesterol of –0.29 mmol/l (95% CI –0.65 to 0.07 mmol/l), LDL cholesterol of –0.51 mmol/l (95% CI –0.76 to –0.26 mmol/l) and fasting plasma glucose –0.30 mmol/l (95% CI –0.75 to 0.15 mmol/l). However, all these results are from only one study.

During the double-blind phase of 24 weeks 86.6% of participants on orlistat and 42.3% of participants on placebo experienced gastrointestinal side-effects. One participant required a cholecystectomy in the placebo/orlistat group and one participant developed a stroke in the 52-weeks orlistat 360 mg/day group.

Sibutramine

This drug from Abbott Laboratories – (trade name Reductil) is a serotoninergic and adrenergic re-uptake inhibitor that enhances satiation.

Effects of sibutramine (10 mg/day) and diet versus placebo and diet

Sibutramine and diet compared with diet in the three weight reduction studies was associated with an overall WMD weight change at 12 months of –4.12 kg (95% CI –4.97 to –3.26 kg). The weight reduction study by Apfelbaum and colleagues was associated with a weight change at 15 months of –3.70 kg (95% CI –5.71 to –1.69 kg). The STORM weight maintenance study was associated with a weight change at 18 months of –3.40 kg (95% CI –4.45 to –2.35 kg).

Quality of evidence I-1 Size of effect = B

At 12 months sibutramine in the weight reduction studies showed beneficial effects on HDL cholesterol and triglycerides, as did the sibutramine group in the STORM weight maintenance study. At 18 months in the STORM study HDL and triglycerides were still significantly improved.

At 12 months, SBP showed a WMD change of 1.16 mmHg (95% CI –0.60 to 2.93 mmHg) in two weight reduction studies. Diastolic blood pressure showed a WMD change of 2.04 mmHg (95% CI 0.89 to 3.20 mmHg).

One person required a cholecystectomy in the sibutramine group of the study by Apfelbaum and colleagues. One person was also withdrawn from the placebo group in this study because of the development of hypertension. Adverse events did not appear to differ between the treatment arms for this study, with the exception of constipation, which was more common with sibutramine (OR 4.14, 95% CI 1.31 to 13.10), although the confidence interval was wide.

In the study by Smith and colleagues one participant withdrew from the 10-mg sibutramine group owing to four drop attacks within 2 weeks of the start (history of epilepsy) and one participant withdrew from the 15-mg sibutramine group owing to palpitations due to frequent ventricular ectopic beats. Dry mouth was also significantly more frequent in both sibutramine groups than in participants on placebo (OR 11.42, 95% CI 2.72 to 47.87).

In the study by McMahon dry mouth and constipation were also the adverse events reported as being significantly more frequent in the group on sibutramine ($p < 0.05$). Eight out of 150 participants discontinued sibutramine as a result of hypertension, compared with one out of 74 participants on placebo (OR 4.11, 95% CI 0.50 to 33.52).

The STORM study found dry mouth, constipation, increased blood pressure, insomnia and nausea to be more than twice as frequent in the sibutramine participants. One participant in each of the sibutramine and placebo groups was withdrawn as a result of hypertension. Of the participants with hypertension taking sibutramine, two needed an increase in therapy and two a decrease.

Effects of SSRIs and diet versus placebo and diet

Other SSRIs (e.g. fluoxetine, femoxetine or sertraline) had no apparent added effect on weight loss or maintenance or any of the reported risk factors. At 12 months the added effect of SSRIs on weight reduction was associated with an overall WMD weight change of –0.33 kg (95% CI –1.49 to 0.82 kg).

The study by Bitsch and Skrumsager assessed weight at 12 months in 37 participants and reported a median change in weight of –6.6 kg for the femoxetine group and –8.8 kg for the placebo group.

O'Kane and colleagues reported one serious adverse event of colonic malignancy in the placebo group of a fluoxetine trial. Wadden and colleagues reported no difference in depression scores between participants on sertraline and placebo; other studies did not report on mood. Goldstein and colleagues, Wadden and colleagues and Bitsch and Skrumsager reported significantly more adverse events in the SSRI groups, which were expected side-effects of the drugs.

Metformin

Metformin is a biguanide that decreases glugoneogenesis and increases peripheral utilisation of glucose. It is mainly prescribed for the treatment of diabetes mellitus.

Effects of metformin (up to 1700 mg daily) and diet versus placebo and diet

Metformin for weight reduction was associated with a WMD effect on weight at 12 months of –1.09 kg (95% CI –2.29 to 0.11 kg) and at 24 months of –0.50 kg (95% CI –4.02 to 3.02 kg). At a median of 5 years metformin was associated with a WMD weight change of –0.12 kg (95% CI –1.13 to 0.89 kg), at 10 years of –0.37 kg (95% CI –1.67 to 0.93 kg) and at 15 years of –2.71 kg (95% CI –6.98 to 1.56 kg). The longer term data were only available for the UKPDS study.

Metformin had a beneficial effect on total cholesterol at 12 and 24 months and on fasting plasma glucose at 12 months. At a median of 5 years metformin was associated with a WMD in fasting plasma glucose of –1.30 mmol/l (95% CI –1.91 to –0.69 mmol/l), 10 years of –0.34 mmol/l (95% CI –1.10 to 0.42 mmol/l) and 15 years of –1.51 mmol/l (95% CI –3.76 to 0.74 mmol/l). The UKPDS was associated with a WMD in HbA_{1c} at a median of 5 years of –0.46% (95% CI –0.98 to 0.06%) and 15 years of –2.31% (95% CI –3.85 to –0.77%).

At 12 months the control arms were associated with a greater reduction in SBP and diastolic blood pressure (DBP) than the metformin arms and at 24 months this was statistically significant. The WMD effect on SBP at 24 months was 10.00 mmHg (95% CI 3.21 to 16.79 mmHg) and on DBP at 24 months was 5.00 mmHg (95% CI 0.56 to 9.44 mmHg). It should be noted these data at 24 months were derived from one study with small numbers of participants and that the confidence intervals are wide.

One death and no new cases of diabetes were reported in the metformin group of the BIGPRO 1 study and five new cases of diabetes occurred in the placebo group (OR for developing diabetes 0.09, 95% CI 0.00 to 1.64). Teupe and Bergis reported one MI in the treatment group at 1 year. Diarrhoea was more commonly reported for patients on metformin in BIGPRO 1 and the study by Teupe and Bergis.

The UKPDS reported outcomes of total mortality, and deaths for MI, stroke and all-cause cancers at a median period of 10 years. For all-cause mortality the OR was 0.62 (95% CI 0.42 to 0.91) in favour of metformin, for MI mortality the OR was 0.51 (95% CI 0.28 to 0.94). For cerebrovascular mortality the OR was 0.80 (95% CI 0.28 to 2.26) and for all-cause cancer mortality it was 0.73 (95% CI 0.36 to 1.49).

Acarbose

Acarbose inhibits the action of pancreatic amylase, and hence delays the digestion and absorption of carbohydrates from the gut. It is mainly prescribed for the treatment of diabetes mellitus.

Effects of acarbose (up to 600 mg/day) and diet versus placebo and diet in an obese population with type II diabetes

Over 12 months acarbose was associated with a WMD weight change of –0.79 kg (95% CI –1.53 to –0.05 kg). Over 12 months acarbose was associated with a WMD change in HbA_{1c} of –0.76% (95% CI –1.05 to –0.47%) and in fasting plasma glucose of –1.36 mmol/l (95% CI –1.96 to –0.75 mmol/l). The authors reported that lipids did not change in participants who received acarbose, but the data were not provided. Acarbose led to significant decreases in the doses of metformin, sulfonylurea and insulin prescribed. Acarbose was more frequently associated with gastrointestinal adverse effects, classified as mild. Four participants on insulin (one receiving acarbose and three receiving insulin) required correction of severe hypoglycaemia.

Effectiveness of behaviour therapy

Behaviour therapy is a treatment for obesity that was introduced by Stuart[81] who reported excellent results in 8 patients who lost 17.1 kg in 12 months (see below). Behaviour therapy does not in itself cause weight loss, but enables patients to adhere more strictly to the prescribed diet. The therapy is based on the assumption that behaviour is acquired and maintained by environmental events, so it can be relearned if the environment is altered to promote this change. Stuart's patients were therefore required to eat only in a special room in their house, at specified times, and with a specified place setting. Everything related to food and eating was recorded in a diary. They weighed themselves 4 times daily. They were taught how to cope with self-defeating thoughts about dieting.

Many attempts have been made to make this therapy less onerous, while retaining its efficacy, so now the term 'behaviour therapy' is used to describe many different combinations of psychological strategies to enable the patient to achieve greater control over their eating behaviour. Behaviour is placed after drug therapy in this chapter because, like sibutramine, behaviour therapy does not in itself cause weight loss, but makes it easier for patients to follow a prescribed diet, and thus lose weight.

Effects of diet and behaviour therapy versus control

In these studies the mean BMI at baseline was 34 kg/m^2 . The control group received 'minimal' treatment, and the diet and behaviour therapy group were contacted 13 to 40 times in the initial year.

The meta-analysis of diet and behaviour therapy compared with no treatment showed a WMD weight change at 12 months of –7.21 kg (95% CI –8.68 to –5.75 kg) and at 24 months of –1.80 kg (95% CI –4.77 to 1.17 kg).

Quality of evidence = I-1 Size of effect = A*

At 12 months diet and behaviour therapy demonstrated beneficial effects on HDL cholesterol, with a weighted mean difference of 0.11 mmol/l (95% CI 0.06 to 0.17 mmol/l), triglycerides –0.58 mmol/l (95% CI –0.98 to –0.17 mmol/l), SBP –3.39 mmHg (95% CI –5.91 to –0.86 mmHg) and DBP –3.37 mmHg (95% CI –5.16 to –1.58 mmHg). At 24 months the study by Wing and colleagues showed significant beneficial effects on total cholesterol only, WMD –0.30 mmol/l (95% CI –0.58 to –0.02 mmol/l), but the number of participants in this study was small.

In the cluster RCT by Kaplan and colleagues, mean body weight in the groups ranged from 89.9 to 92.2 kg. All participants received an equal number of contacts and an active initial treatment period of 10 weeks. LCD plus behaviour therapy was associated with a mean weight change at 18 months of –1.68 kg, but weight change was not reported for the control group. At 18 months the diet and behaviour group was associated with a mean change in HbA_{1c} of –0.46% compared with 0.36% in the control group. Quality of

well-being was also assessed, and was increased by 0.03 units in the diet and behaviour group and decreased by 0.04 units in the control group at 18 months.

No deaths or serious adverse events were reported in any of the studies. Wing and colleagues reported that the risk of developing diabetes was 7% in the control group and 30.3% in the diet and behaviour therapy group.

Effects of diet and behaviour therapy and exercise versus control

Diet, behaviour therapy and exercise compared with control from 11 studies was associated with an overall WMD weight change at 12 months of –4.00 kg (95% CI –4.47 to –3.54 kg).

Quality of evidence = I-1 Size of effect = B

Diet, behaviour therapy and exercise was associated with a WMD weight change at 30 months of –4.68 kg (95% CI –6.08 to –3.28 kg, two trials) and at 36 months of –2.00 kg (95% CI –2.66 to –1.34 kg, one trial).

In the cluster RCT by Kaplan and colleagues, the authors reported that at 18 months participants' weight was 'essentially constant' in the LCD, behaviour therapy and exercise group. Change in weight was not reported for the control group at 18 months. At 18 months the diet, behaviour therapy and exercise group was associated with a mean change in HbA_{1c} of –1.48% compared with 0.36% in the control group. The authors reported that for 100 participants receiving the diet, exercise and behaviour therapy programme 4.7 well-years would be produced, compared with the control (0.047 well-years for each participants, where 0 = death and 1 = optimal function).

In the study by Wing and colleagues, two out of 40 control participants and five out of 40 participants assigned diet, exercise and behaviour therapy developed type II diabetes mellitus at 2 years.

Two participants with MI were reported in the active treatment group and four in the control group (which includes non-obese participants) of the Trial of Non-pharmacological Interventions in the Elderly (TONE) study. Two participants with cerebrovascular accident were also reported in the control group (which includes non-obese participants), and none in the intervention group. In the TONE study the hazard ratio for the primary end-point (recurrence of hypertension and cardiovascular events) was 0.65 (95% CI 0.50 to 0.85) for those randomised to weight loss alone compared with controls. One participant with breast cancer and one with pancreatic cancer were reported, but it was unclear which groups these people came from.

One death was reported in the intervention group and one in the control group of Trials of Hypertension Prevention (TOHP) I. The relative risk for developing hypertension for the intervention group was 0.66 (95% CI 0.46 to 0.94). In TOHP II five people randomised to weight loss died (three cardiovascular disease deaths) and two people in the usual care group died. The relative risk of developing hypertension for the weight loss group was 0.87 (p=0.06) at 48 months.

Effects of family versus individual treatment

The family-based intervention from four studies was associated with an overall WMD weight change at 12 months of –2.96 kg (95% CI –5.31 to –0.60 kg).

Quality of evidence = I-1 Size of effect = B

At 18 months two family-based intervention studies were associated with an overall WMD weight change of –1.08 kg (95% CI –3.04 to 0.87 kg). At 24 months one family-based study was associated with an overall WMD weight change of –5.61 kg (95% CI –10.98 to –0.24 kg). At 43 months one family-based study was associated with an overall WMD weight change of –0.75 kg (95% CI –6.95 to 5.45 kg) and at 48 months

–1.55kg (95% CI –7.88 to 4.78 kg); however after 18 months the number of participants contributing to this comparison was very small.

At 18 months Wing and colleagues were unable to demonstrate any difference between family and individual approaches for weight change, HbA_{1c} or fasting plasma glucose, in a study with a small number of participants.

There were no reported deaths or serious adverse events in any of the included studies.

Effects of group versus individual treatment

Compared with individual treatment the group administered intervention for three studies was associated with an overall WMD weight change at 12 months of 1.59 kg (95% CI –1.81 to 5.00 kg).

Effects of diet and behaviour therapy versus diet

The additional effect of behaviour therapy on diet was associated with an overall WMD weight change at 12 months of –7.67 kg (95% CI –11.97 to –3.36 kg), at 18 months of –4.18 kg (95% CI –8.32 to –0.04 kg), at 36 months of –2.91 kg (95% CI –8.60 to 2.78 kg) and at 60 months of 1.90 kg (95% CI –3.75 to 7.55 kg).

Quality of evidence = I-2 Size of effect = A

Thus, there was a significant added effect of behaviour therapy on weight change at 12 and 18 months, but not at 36 or 60 months. The number of participants contributing to the comparisons decreased over time so the sustained effect of behaviour therapy cannot readily be assessed. In the cluster RCT by Phenix, where meeting time was the unit of randomisation, mean body weight in the groups ranged from 79 to 86 kg. Phenix evaluated the added effects to diet of two forms of behaviour therapy, which were overt behaviour therapy and cognitive behaviour therapy. The added effect of overt behaviour therapy to an LCD was associated with a weight change at 12 months of –3.26 kg compared with –4.82 kg in the diet-only group. The added effect of cognitive behaviour therapy to an LCD was associated with a weight change at 12 months of –6.68 kg compared with –4.82 kg in the diet-only group.

No deaths or serious adverse events were reported in any of the included studies.

Added effect of any intervention over diet

Comparing all treatments assessed as an adjunct to diet at 12 months, behaviour therapy was associated with the greatest WMD weight change of –7.67 kg (95% CI –11.97 to –3.36 kg).

Sibutramine was associated with a WMD weight change of –4.12 kg (95% CI –4.97 to –3.26 kg), orlistat –3.01 kg (95% CI –3.48 to –2.54 kg), exercise –1.95 kg (95% CI –3.22 to –0.68 kg) and metformin –1.09 kg (95% CI –2.29 to 0.11 kg), acarbose –0.79 kg (95% CI –1.53 to –0.05 kg) and behaviour therapy plus exercise –0.67 kg (95% CI –4.22 to 2.88 kg).

At 18 months, exercise was associated with improved weight loss when added to diet, and the additional behaviour therapy was just significant. At 24 months, orlistat was associated with enhanced weight loss when added to diet, and exercise enhanced weight loss when added to diet and behaviour therapy.

The effect of exercise was similar at 36 months. At 60 months behaviour therapy as an adjunct to diet could not be shown to prevent weight gain.

At 12 months orlistat added to diet was associated with lowered DBP and SBP, HbA_{1c}, total cholesterol and glucose, whereas sibutramine increased DBP. At 12 months acarbose added to diet was associated with lowered HbA_{1c} and glucose.

Only one study assessed the added effect of behaviour therapy and exercise to diet and was unable to demonstrate any significant effect on weight or any risk factor at 12 months and 24 months.

The addition of exercise to diet and behaviour therapy was associated with significantly increased weight loss at 12 and 24 months.

Comparisons of treatments versus controls

Few studies compared LCD or VLCD with control, but there was a trend for these diets to produce more weight loss at 1 year than the 600 kcal/day deficit or low-fat diet. One VLCD study was associated with the greatest WMD weight change at 12 months of -13.40 kg (95% CI –18.43 to –8.37 kg). At 24 and 36 months there was some suggestion that LCDs were more effective than 600 kcal/day deficit diets.

Diet and exercise, diet and behaviour therapy, and diet, behaviour therapy and exercise were all associated with significantly greater weight loss than control at 12 months.

In terms of mode of delivery, participants appeared to lose less weight in a group setting than when receiving treatment on an individual basis at all time-points, and this reached statistical significance at 24 months. Participants also appeared to lose more weight when accompanied by their spouse or a friend than when unaccompanied, and this was statistically significant at 12 and 24 months.

Surgery

The HTA 2004 review makes little mention of the efficacy of surgery in the treatment of obesity. In this case also there are problems about entry criteria. By far the largest trial of the effectiveness of surgical treatment is the Swedish Obese Subjects (SOS) trial, that started in 1987.[83] It is intended to recruit 2000 matched patient pairs, and to follow them for 10 years. It is not a randomised trial (the patient having surgery is matched (on 18 variables) with a similar patient who is having non-surgical treatment) therefore it is not included in any Cochrane review. So far 1879 patient pairs have been recruited. After 8 years the weight loss in the surgical group was 28±15 kg, and 0.5±8.9 kg among controls. The weight loss in the surgical patients (as percent body weight) was 33±10% after gastric bypass, 23±10% after vertical banded gastroplasty, and 21±12% after gastric banding. The two-year incidence of diabetes in the surgical patients is 32 times less than in the matched controls, and with hypertension it is 2.6 times less than in controls.

Effects of surgery and diet and behaviour therapy versus surgery

One RCT reported weight change in patients after bariatric surgery. The mean BMI at baseline was 48.9 kg/m^2 for the diet and behaviour group, and 47.6 kg/m^2 for the minimal intervention group. At 12 months diet and behaviour therapy compared with the minimal intervention was associated with a WMD weight change of –10.03 kg (95% CI –22.29 to 2.23 kg) and at 24 months of –10.56 kg (95% CI –23.17 to 2.05 kg). The number of participants in the study was small. The dropout rate was 47% in 2 years, and it was unclear if intention-to-treat (ITT) analysis had been used.

Critical evaluation of the HTA review[7]

The HTA 2004 systematic review presents the weight loss and change in risk factors, compared with control groups, of obese patients who were initially BMI > 28 kg/m^2, and who consented to be randomised to active or control groups, and were followed for at least 12 months. These entry criteria are reasonable for comparing drug trials of orlistat or sibutramine (with which the review is mainly concerned) but not for assessing the effectiveness of diet, exercise or surgery as treatments for obesity. There have been some RCTs comparing diet with 'control', but it is very difficult to recruit obese people who are randomised to the

non-diet control group who will remain for 12 months making no effort to obtain some help elsewhere to lose weight. RCTs of exercise very rarely are able to recruit individuals with BMI > 28 kg/m^2, because such people are incapable of prolonged vigorous exercise.[82]

Run-in periods, drop-outs, and ITT analysis

Ideally, patients in an RCT should be typical of all patients with the condition under study, and they should all complete the protocol as planned. In real life these ideal conditions are rarely achieved, so the trial protocols are designed to cope with patients who enrol in the study, but do not comply with the protocol, or who drop out before the trial is designed to end. For example, in a trial of orlistat,[81] all 743 entrants had a run-in period of 4 weeks on placebo and diet, and the 688 patients who took > 75% of the capsules prescribed were then randomised to orlistat or placebo. Therefore the patients who entered the trial were not typical of all the patients screened – they were those selected for being more compliant.

In all long-term drug trials some patients drop out, and these are usually those who are making poor progress. If the analysis is confined to those who complete the trial the results are falsely favourable to the treatment, especially if more patients drop out from the active than from the control group. To avoid this error ITT (intention to treat) analysis is used, but in the case of weight loss trials this also involves bias, since when a patient drops out the last recorded weight is assumed to be the weight at termination of the trial. We know that this is unduly favourable, because when patients drop out of trials their weight does not usually remain constant, but they tend to regain the weight they have lost.

For these reasons, when comparing trials of different interventions for weight loss, we must consider factors such as the starting weight of participants, the selection of compliant responders, and the analysis of drop-outs. Unfortunately this important information is often not provided in the HTA 2004 review.

Prevention of obesity in adults

Three reviews have investigated the prevention of obesity and overweight in adults.[84,85,86] Two of these reviews[84,85] looked at the same three community-based studies, but there was a subtle difference in their conclusions. One review[84] stated that '*community-based education programmes linked with financial incentives may be effective (based on one study) but more research is required*', whereas the other review[85] concluded that, based on the very limited evidence to date, '*community-based obesity prevention methods have not been proven effective. There is insufficient evidence to recommend in favour of, or against, community-based obesity prevention programmes.*' In all the studies, the mean weight of the intervention and control communities did not differ significantly during a three to seven year follow-up period. However, one review[85] also concluded that, given the health risks and financial costs associated with obesity, '*priority should be given to the prevention of obesity over weight loss interventions*'.

A systematic review of interventions to prevent weight gain in both children and adults[86] identified 11 papers describing five distinct interventions in schools and four in the wider community. Five of the studies were RCTs and half targeted individuals with a low income or a socially disadvantaged background. All the studies had a follow-up of at least one year, except one which followed up after six weeks. Due to the variability of study designs, samples and outcome variables, the authors found it difficult to identify effective types of interventions. The review reported positive changes in half of the interventions that measured diet and physical activity by self-report. Effects on observed weight were mixed, with two studies finding no significant differences, two studies reporting less weight gain in the intervention group and one study finding less weight gain only in sub-groups; the other four studies did not report on the effect on weight. Only one of the five RCTs reported a significant effect on weight. This intervention involved a correspondence programme and a mix of behaviour change methods including goal setting, self-monitoring

and contingencies. Overall, the authors concluded that *'future interventions might be more effective if they were explicitly based on methods of behaviour change that have been shown to work in other contexts. Effective interventions would be more easily replicated if they were explicitly described. Effectiveness might be more precisely demonstrated if more objective measures of physical activity and diet were used, and if the follow-up was over a longer period.'*

In summary, the evidence from the three identified reviews[84,85,86] was found to be mixed and inconclusive in terms of effectiveness. Considering the potential scale of obesity and overweight and the associated health, economic and social consequences, the development of effective strategies to prevent obesity is a priority. Therefore, there is an urgent need for further research.

There is inconclusive evidence regarding the effectiveness of community-based interventions (for example, seminars, mailed educational packages and mass media participation) for the prevention of obesity and overweight in adults.

Quality of supporting evidence = I-2 Size of effect = too variable to summarise (due to the different types and combinations of interventions used)

Obesity in children

Treatment of obesity in children[77]

Eighteen randomised controlled trials, with 975 participants, were included in this review. Five studies (n=245 participants) investigated changes in physical activity and sedentary behaviour. Two studies (n=107 participants) compared problem-solving with usual care or behavioural therapy. Nine studies (n=399 participants) compared behavioural therapy with varying degrees of family involvement, with no treatment or usual care or mastery criteria and contingent reinforcement. Two studies (n=224 participants) compared cognitive behavioural therapy with relaxation.

These 18 studies shared similar goals and objectives. However, there were multiple differences in terms of study design (particularly comparisons) and quality (particularly sample size and thus power), and outcome measures. Most of the studies included in this review were too small to have the power to detect the effects of the treatment. A meta-analysis wasn't conducted since so few of the trials included the same comparisons and outcomes. Therefore, results are synthesised in a narrative format.

The results suggest that there may be some additional benefit to behaviour therapy where parents, rather than the child, are given the primary responsibility for behaviour change. In addition, relaxation may be as effective as behavioural therapy. Although there were many trials which focused on changing levels of physical activity and/or sedentary behaviour, these trials were too small to draw any conclusions from with confidence. However, there are some data from these trials in favour of a reduction in sedentary behaviour. Compliance to lifestyle advice was assessed in a minority of the included studies.

Physical activity is recommended for everybody regardless of their weight because of the proven health benefits, although these are not as clear in childhood. Therefore, children should be encouraged to increase their levels of physical activity, even if there is no great benefit in terms of weight reduction.

Most studies were generated in the United States of America among children aged between 7 and 12 years. Interventions to reduce obesity may vary in effect depending on the age of the child since children are metabolically, developmentally, emotionally and nutritionally different in each of the three childhood phases (i.e. infancy, childhood and adolescence). Most studies were underpowered (15/18 randomised fewer than 23 children to at least one group). Seven of the included studies were carried out by the same research team in the US, which has implications for generalising the results of this review to other contexts. Many of the 18 included trials were run from a specialist obesity clinic within a hospital setting.

The proposed relationship between treating obesity and eating disorders, particularly in young populations, may limit the enthusiasm of healthcare workers to treat obesity in children. However, while eating disorders are clearly important public health issues and while dieting may be a risk factor for eating disorders in some people, the literature about this relationship remains equivocal. It is important to acknowledge that the proportion of the population who are obese far exceeds the proportion of the population who have eating disorders.

In summary, the limited evidence makes it difficult to conclude that one strategy or combination of strategies is more important than others in the treatment of childhood obesity. However, the existing literature provides some useful insights regarding 1) the potential of a reduction in children's sedentary behaviours, 2) the delivery of behaviour therapy, and 3) the use of relaxation as effective treatments for childhood obesity. The practicalities of delivering effective advice on lifestyle changes to overweight children will vary with the wide span of social, ethnic and economic circumstances. However, obesity is known to cluster in families, and interventions should be targeted to children whose parents are overweight as a priority.

Quality of supporting evidence = I-1 Size of effect = too variable to summarise (due to the many different types and combinations of interventions used)

NOTE: The SIGN guideline (2003)[63] on childhood obesity provides practical guidance for healthcare workers who treat overweight and obese children.

Prevention of obesity in children[76]

Ten studies were included in this review, seven of which were long-term (follow-up at least 1 year from baseline) and three of which were shorter term (follow-up at least 3 months from baseline). These ten studies shared similar goals and objectives, but multiple differences in terms of study design and quality, target population, theoretical underpinning of intervention approach, and comparable outcome measures, precludes the opportunity to combine results. However, it seems reasonable to suggest that a concentration on strategies that encourage a reduction in sedentary behaviours and an increase in physical activity may be fruitful.

Most studies were generated in the United States of America among children aged between 7 and 12 years. The diversity of the studies limits the generalisability and reproducibility of the findings. Documentation of the characteristics of non-participants and those who are lost to follow-up, whilst difficult to achieve, would be extremely useful in broadening our understanding of the implications of these study findings. Clearly, generalisability would be improved by designing interventions that target a range of communities.

The small number and diverse nature of studies in this area is likely to reflect a number of conceptual, methodological and possibly ethical challenges facing researchers. The environment in many countries, in which driving physiological goals to be sedentary and well fed are overwhelmingly supported by an increasingly complex socio-political environment, may make any attempt to change the status quo seem unrealistic. Therefore, interventions in this area may be considered to be too difficult to pursue. A further conceptual challenge is posed by our limited understanding of the interface between individuals' behaviours and the environment.

This review highlights a paradoxical situation. At a time in which we see obesity prevention nominated as a public health priority, we find a research environment that currently lacks the power to set clear directions for obesity prevention activity across a range of groups at risk.

In summary, there is limited quality data on the effectiveness of obesity prevention programmes and as such no generalisable conclusions can be drawn. Concentration on strategies that encourage reduction of sedentary behaviours and an increase in physical activity may be fruitful.

Quality of supporting evidence = I-2 Size of effect = D

NOTE: The SIGN guideline (2003)[63] on childhood obesity provides practical guidance for healthcare workers who are involved in preventing children from becoming overweight and obese.

Effectiveness of healthcare professional-centred interventions[87]

Eighteen studies were included involving 446 providers and 4158 patients. Five were professional-oriented interventions (the use of reminders and training) and the sixth was a study of professional and organisational interventions of shared care. Twelve studies compared either the deliverer of weight loss interventions or the setting of interventions. The studies identified for this review are heterogeneous in terms of participants, interventions, outcomes, and settings. In addition, only a small number of different interventions have been evaluated rigorously. Along with small sample-sizes and reasonably high drop-out rates among patients, it is difficult to draw firm conclusions on how the management of obesity might be improved from the available evidence.

The two reminder studies indicate that this may be a promising approach to changing doctors' practice. More information is necessary to be able to indicate whether this finding is generalisable across other settings and health professionals. It is not possible to say whether the change in practice may result in a reliable change in patient outcomes.

It is not possible to tell from the evidence whether training might be a useful approach to changing the behaviour of practice nurses. Two studies assessed the effect of a brief educational intervention on obesity management to GPs and both showed that a cheap and quick intervention of this type may be promising in terms of changing practice, at least in the short term. However, a recent study that involved the entire primary care team found no change in patient outcomes at one year.[88]

One study indicated some positive effects in the short term from encouraging shared care between GPs and a hospital service, but these were not sustained over the long term. It seems that additional strategies might be necessary to attempt to ensure the maintenance of improvements among patients.

The findings from studies evaluating different settings and deliverers are inconclusive. Most are small and of limited quality, and do not appear to demonstrate any consistent setting or deliverer effects. However, one study comparing inpatient and outpatient treatments is interesting in that it offers a novel approach to obesity management. In this study, benefits were seen in the inpatient group, including in the longer term. It would be useful to know whether these findings can be replicated on a larger scale across different settings. However, the cost of such an approach to obesity management may prove prohibitive. Without good quality studies including reliable cost effectiveness analyses, it is not possible to say whether the health benefits are worth the additional financial outlay.

Given the large number of commercially run weight loss programmes in some countries, it would be interesting to know whether interventions delivered by health professionals are more effective than those delivered by lay people. We identified one such study. This study found that mean weight loss at one year in weight loss clinics run by a professional therapist was greater than that in weight loss clinics run by a self-help weight clinic leader who had previously been trained by the professional therapist in behavioural therapy.

It would also be interesting to know if less resource-intensive interventions (such as programmes delivered in the home) are cost effective relative to more intensive face to face treatments, but based on the available evidence we cannot say whether this might be so.

There were no studies assessing whether negative attitudes amongst providers were impinging on good practice and whether interventions to change attitudes might result in improved clinical decisions. Given that much commentary has been passed on the possible implications of negative views toward this group of patients, it is surprising there have been no rigorous evaluations of strategies to improve negative attitudes and related practices.

There were no studies comparing whether organisational interventions designed to change the structure of services for overweight and obese people are more effective than educational or behavioural interventions for health professionals. The rationale behind this comparison was that changes in the provision of weight loss services may be more effective than attempting to change health professionals' practice on an individual basis. That is, health professionals could utilise a service rather than think about what to do with overweight and obese patients themselves, thereby overcoming negative perceptions of patients and treatment efficacy, as well as knowledge and time barriers. Along with more general evaluations of interventions to implement obesity services, such comparisons would be of interest.

In summary, health professionals, and in particular primary care providers, have the potential to access large numbers of patients. We currently have little information about how practice or the organisation of care in this area might be improved, although reminder systems, brief training interventions, shared care, inpatient care and dietitian-led treatments may all be worth further investigation.

Effectiveness and cost

In section 7 we suggest how the NHS could most effectively spend the resources allocated for obesity.

7 Models of care and recommendations

Objectives of treatment

In the treatment of patients with established obesity there are three objectives:

- to assist the patient to achieve a weight at which the health risks of obesity are reduced to the lowest possible level for this patient
- to help the patient to maintain this weight loss indefinitely
- to maintain, or restore if necessary, the patient's self-esteem.

Target weight and rate of weight loss

Some guidelines emphasise that 'priority in obesity management should be on weight maintenance and modest weight loss, rather than a return to ideal or normal weight'.[56] The reasoning behind this advice is that there can be a substantial reduction in mortality and risk factors in overweight people who lose 5–10 kg in one year, and that repeated failures to achieve normal weight amplify a patient's depression and lack of self-esteem. These are valid arguments, but there is no evidence that a patient who is, say, 40 kg overweight does better if advised to lose 5–10 kg than if advised to lose 30–40 kg. If the patient who is 40 kg overweight is aged 65 years, short in stature and crippled with osteoarthritis of hips or knees, it is quite

likely that a loss of 5–10 kg is as much as can reasonably be achieved. In this situation, the modest weight loss may reduce the health risks to the lowest possible level for this patient, and thus achieve the first objective above.

On the other hand, if the 40 kg-overweight patient is aged 25 years and otherwise fit, but has a family history of diabetes or heart disease, a loss of only 5–10 kg is unlikely to be the optimum for reduction of health risk. Thus the appropriate target weight loss should be assessed in the light of the individual circumstances, rather than by the application of any fixed rule.

Concerning the optimum rate of weight loss there is consensus that a rate greater than 1 kg/week is undesirable, since it is likely to cause excessive loss of lean tissue.

Priority for treatment: obesity or co-morbidity?

In section 3, three sub-categories of obesity were identified: obese patients with existing co-morbidities, obese patients without co-morbidities and non-obese individuals with special risk factors for the development of obesity. At present the usual policy is to treat the co-morbidities of the first group, and to ignore the remainder.

This policy may not be appropriate. French *et al.*[89] studied the effect of weight change between the ages of 18 and 50 years on disease prevalence later in life. They obtained by postal questionnaire reported weight change between the ages of 18 and 50 years in 41 837 women, and were able to classify the weight change pattern of 17 252 of these women into one of five classes. The pattern of the remaining 23 710 women (mostly weight gainers) would not fit into these patterns, and they were excluded from the series. Between age 18 and 30 years those in whom weight changed by less than 5% were classified 'weight stable'. Those in whom weight increased, or decreased, by 5 kg or more were classified as 'weight gainers' or 'weight losers'. A similar designation was used for weight change between the age of 30 and 50 years. The number of women who showed each of the five patterns of weight gain, and their subsequent odds ratio of developing diabetes, high blood pressure, heart attack, other heart disease, cancer or perceived poor health, is shown in Table 9. The data are also analysed separately for those who were, or were not, overweight at age 18 years. Three important messages emerge from this table.

1 Weight loss of 5 kg or more between the age of 18 and 30 years is uncommon: it was observed in only 8% of the population, half of whom regained this lost weight during the period between 30 and 50 years of age. The largest group (47%) are those who gain at least 5 kg between 18 and 30, and again between 30 and 50 years.
2 The odds ratio for diabetes, hypertension, heart attack, other heart disease and perceived poor health (but not cancer) is significantly increased in those women who gained weight compared with those whose weight was stable. The diseases most strongly associated with weight increase are diabetes and hypertension.
3 Eight percent (712 of 9153) of the women were overweight at age 18. In virtually every instance, the odds ratio of the diseases analysed (except cancer) is markedly higher among those who were overweight at 18 compared with those who were not.

This indicates that a policy of laissez faire is not justified. Individuals who are overweight at age 18 years, or who gain more than 5 kg during adult life, have a significantly increased risk of developing the co-morbidities of obesity. Both medically and economically it may be better to prevent this weight gain than to treat the co-morbidity when it arises.

Table 9: Pattern of reported weight change in 17 252 women between age 18 and 50 years, and subsequent health. 95% confidence interval for odds ratio includes 1.0; values are shown in parentheses.[89]

Proportion of women		**28%**	**4%**	**4%**	**17%**	**47%**
Weight change*						
Age 18–30 years		**stable**	**loss**	**loss**	**gain**	**gain**
Age 30–50 years		**stable**	**stable**	**gain**	**stable**	**gain**
Odds ratio of reporting disease at age 62 years						
Diabetes	(a)	1.0	(0.6)	2.3	3.8	6.6
	(b)	5.5	(1.4)	7.5	8.9	19.1
Hypertension	(a)	1.0	(0.8)	1.8	1.8	3.2
	(b)	2.0	(1.2)	3.1	6.3	7.6
Heart attack	(a)	1.0	(1.3)	1.9	1.6	2.0
	(b)	(1.5)	(1.6)	2.0	5.4	3.5
Other heart disease	(a)	1.0	(1.3)	1.8	1.7	1.8
	(b)	(1.3)	2.0	(1.1)	2.6	2.9
Cancer	(a)	1.0	(1.2)	(1.6)	(0.8)	(0.9)
	(b)	(1.0)	(0.8)	(0.6)	(1.0)	(1.1)
Perceived poor health	(a)	1.0	(1.1)	1.7	1.3	2.1
	(b)	(1.4)	1.5	2.4	2.1	4.5

*Stable = < 5 kg change, loss = 5 kg+ loss, gain = 5 kg+ gain.
(a) not overweight at age 18 years, (b) overweight at age 18 years.

Health gain from weight loss

The data presented above show that weight gain in adult life is a common event, and is associated with greater risks of ill health. In adults, there is conflicting evidence about the health effects of weight loss, or fat loss. This evidence is reviewed at length in a systematic review.[18] Most of the evidence comes from prospective cohort studies. In particular, there are strong epidemiological data, which show that weight loss is associated with *increased* total mortality. This has led some commentators to conclude that, although weight gain is bad for health, losing the excess weight does not confer any benefit.

This paradox has been resolved by a secondary analysis of data from the Tecumseh and the Framingham studies.[90] In these two studies, change in body weight, and also in fat mass (by skinfolds) was recorded. They found that each standard deviation in weight loss *increased* the mortality hazard by 29% and 39% respectively in the two studies. Contrarily, each standard deviation of fat loss *reduced* the hazard rate by 15% and 17% respectively. This supports the interpretation that unintentional weight loss in the general population is a sign of ill health, but that intentional fat loss in obese people is beneficial.

The reason why evidence relating intentional weight loss to health outcomes comes from cohort studies is obvious; although one can randomise individuals to different types of treatments for obesity, one cannot randomise them to weight-loss or no weight-loss groups. Indeed, this problem was the topic of a workshop convened in 1997 by the National Institutes of Health and Centres for Disease Control and Prevention.[91] The workshop participants agreed that a well-designed RCT should be undertaken to estimate the magnitude and direction of the long-term health benefits of intentional weight loss. The results of this study, and those from the SOS study will provide much better evidence. Data on sick leave and disability pension from the SOS study at four years have been published.[92] Severely obese patients who underwent surgical treatment

for obesity lost more weight and experienced a reduction in sick leave and disability pension compared with controls, particularly patients aged 47–60 years.

Matching patients to resources

It is obvious that the number of people involved, and type of care needed, varies greatly between the sub-groups identified in section 3. The relatively small number of obese people with established co-morbidities urgently need medical care, whereas the large number of non-obese people at high risk to develop obesity do not. This suggests that the appropriate model of care should have different levels ranging from low-cost advice centres, which are easily accessible to concerned members of the public and capable of coping with large numbers, to tertiary referral centres capable of providing medical care for the most complex problems.

- Level 1 is essentially a self-help group, which may be guided by a health professional – either a state registered dietitian[93] or a specially trained public health nurse.[94] It is aimed at management of overweight people for whom the SIGN[56] target of weight maintenance or modest weight loss is entirely suitable. It is also suitable for the long-term follow-up of patients who have been assessed and/or treated at Level 2 or 3. The GP of the individual attending a Level 1 centre should be so informed, to ensure that there is no medical contraindication to the proposed weight management plan.
- Level 2. For those individuals who do not achieve satisfactory results at Level 1, or whose obesity or co-morbidity is sufficiently severe to justify it, the next step is to Level 2. This is based on a primary care doctor who may, having assessed the patient, refer back to Level 1. The assessment of current disease and risk factors at Level 2, which is suggested by the SIGN[56] guidelines includes the following:
  - measurement of weight, height and waist: BMI calculated
  - risk factors assessed (e.g. smoking)
  - blood pressure measured
  - urine tested for glucose
  - plasma γ-glutamyl transferase
  - total plasma cholesterol
  - thyroid-stimulating hormone.

  Advice on risk factor management and dietary advice may be offered at level 2, or the patient may be sent back with a referring letter to level 1 for this advice and further follow-up.
- Level 3. The last step, which is appropriate for a small minority of difficult cases, is Level 3, based on a specialist in a tertiary referral centre. Any attempt to quantify the workload at each of these levels involves assumptions about the efficacy of each level (especially Level 1) and the criteria that are used to make the judgement that a change to another level is indicated. Furthermore, if the system works well the prevalence of obesity will decrease, and hence the load at every level will decrease also (see below).

Integration within the NHS and relationship with private facilities

The components of the above model, which involve NHS resources (Levels 2 and 3), should be integrated with NHS activities elsewhere. Clearly the existing facilities for managing diabetes, heart disease, osteoarthritis, etc., should be available to obese patients entering through the Level 1 route, and conversely patients who enter treatment for a co-morbidity, and who are also overweight, should have access to the Level 1 facilities.

Any model of obesity management based on the NHS will run in parallel with other non-NHS facilities, such as commercial clinics, slimming clubs, health farms and leisure centres. At present there is justifiable

suspicion and even hostility between NHS and non-NHS healthcare providers in the field of obesity, since there are many charlatans who are untrained and unregulated in the private sector. Another problem is that under rule 51.3 of the Advertising Standards Agency, slimming clubs are not allowed to advertise themselves as treating obesity (BMI > 30 kg/m^2) because this is a medical condition which should be treated 'under the supervision of a suitably qualified health professional'. This is a commendable attempt to protect the public from exploitation by charlatans, but it also limits the ability of well-run private facilities to provide much-needed Level 1 facilities.

Logistics of the proposed model

Since a very large proportion of the population need help from this model it is logistically necessary to prioritise services to those who would derive most benefit from weight control. The only health *benefit* associated with obesity is a protection against osteoporosis: probably this is partly because adipose tissue contains aromatase, which converts androgens to oestrogens, and hence tends to preserve skeletal mass in post-menopausal women. The health hazards of obesity are greater in young people than in older ones, and weight loss is easier to achieve in younger people, so there are grounds for offering help primarily to younger adults.[95] Management of obesity in children requires a different approach. The implications of this policy related to demographic patterns are discussed below.

Age structure of a model population of 100 000, and of those requiring obesity management services

The age structure of the population of England and Wales is shown in Table 10, divided into five categories. For three of these (pre-school children, secondary school children and retired adults) the case for providing obesity management services is much weaker than for the remaining two – primary school children and pre-retirement adults. The reasons for these priorities are indicated above.

Table 10: Age structure of a model population of 100 000 people in the UK, and the numbers requiring obesity management services.

Category	Age (years)	Number	Annual recruits intake	Requiring management	
				Annual number	Total
Pre-school	0–4	6,400	1,280	nil	nil
Primary school	5–11	8,900	1,270	120	840
Secondary school	12–15	5,000	1,270	nil	nil
Pre-retirement	16–64	63,600	1,270	NK	38,000
Retired	> 65	16,100	NK	nil	nil
Total	100,000				840 primary school 38,000 adults

NK = not known.

For purposes of the following calculation, the population is assumed to be of a constant size, and numbers have been rounded to give no more than three significant figures. In the model population of 100 000 with a typical age structure there will be 8900 primary school children aged 5–11 years, with an annual intake of 1270. Among each year's entry 9% will be above the 91st centile, giving an annual intake of 120 children, who are in this category for seven years, giving a total of 840 primary school children requiring management of their overweight. Facilities for children are discussed later in this section.

The other sector requiring help are the pre-retirement adults of which there are 63 600. With the 2002 prevalence of overweight and obesity in the UK population there will (at any given time) be about 38% or 24 000 who are overweight (BMI 25–30), and about 23% or 14 000 who are obese (BMI >30). To achieve the second objective of treatment given at the start of this section, help should be available to 38 000 young adults (aged 16–64 years) who are overweight or obese.

Weight management clinics required to help 38 000 overweight adults

Although typically a population of 100 000 people will at any given moment have about 38 000 who need help with weight management, it will not be the same 38 000 from one year to the next. In Table 10, the annual intake of overweight adults is shown as 'not known'. Within a population individuals are constantly passing above and below whatever threshold is used to define an 'overweight' category[96] so it is not possible to identify a subsection of the population who are particularly at risk for obesity. The facility must therefore be some form of clinic capable of dealing with those individuals who are overweight, and who perceive themselves to be in need of help at this time.

For reasons given above, it is not possible to estimate how many people in a population of 100 000 would wish to use a Level 1 facility. Some indication can be gained from the experience of Bush *et al.*[93] in Harrow, and of Karvetti and Hakala[94] in Finland. The Harrow Slimming Club operated in a northern suburb of London, and registered 1090 members in 50 courses over a period of ten years. On average, therefore, each year there were five 10-week courses, which were attended by 109 people. Karvetti and Hakala recruited 243 overweight people in six weeks in the city of Turku, which has a population of about 160 000 and 'several' health centres. If a district with a population of 100 000 had five clinics, and each clinic ran ten courses per year for 20 people per course, this would provide facilities for 1000 members per year. This would probably be an adequate facility, because a family member (usually a mother of children) who attended the course would often be able to transmit what she had learned to other members of the family.

Level 2 help should be available by referral of Level 1 members who have additional problems. If this is estimated to be 10% of those attending at Level 1, then there would be 100 referrals per year to GPs of overweight people with additional problems. This would not be a great burden, since it would be fewer than ten patients per year to individual GPs.

If there were ten specialist centres in the UK, then each Level 3 facility would serve about 600 districts. If practitioners at Level 2 referred 98% of the patients back to Level 1 for management, and 2% on to Level 3, then each specialist unit would receive about 1200 referrals per year. Assessment at Level 3 would initially involve an outpatient consultation with a specialist (and preferably also a dietitian), for which about 30 minutes would be required, so a clinic session of 2.5 hours would provide slots for five new patients. At this input, the Level 3 facility would have to run about five clinics per week. At present there is no experience of a national Level 3 facility such as that suggested, so the estimate of workload is a guess which would have to be modified in the light of experience, and in the light of the confounding factors mentioned at the start of this section.

Facilities required to manage 38 000 overweight adults

The facilities needed at Level 1 are easily provided by local authority school health clinics, which are not used in the evenings. The most important requirement is a good leader – preferably a state registered dietitian or specially trained health nurse. A room in which it is possible to conduct seminar-type teaching with a group of up to 25 members, and facilities for weighing the members of the group and for showing audiovisual teaching aids is also required.

The facilities required at Level 2 are normally found in the surgery premises of GPs. These are for a clinical consultation, physical examination and assessment according to the SIGN[56] guidelines. These are weight, height and waist measurement, blood pressure, urine glucose, plasma γ glutamyl transferase, plasma cholesterol and thyroid-stimulating hormone.

The facilities for Level 3 care will normally be in a teaching hospital. Apart from the advantage of the facilities mentioned below, it is important that the management of obesity at this level should be part of the education of healthcare students. In addition to the normal clinical and diagnostic facilities, there should be a physician with a special interest in obesity, full dietetic support, and preferably facilities for measuring resting metabolic rate by indirect calorimetry and for measuring body composition by dual emission x-ray absorptiometry (DEXA) or bioimpedance. The consultant physician needs to have the option of referral for advice to a surgeon or clinical psychologist, and to be familiar with the indications for drug treatment. It is very helpful to have the option to use up to three hospital beds, so that patients who have been referred from a distance can be admitted for investigation if necessary.

Hospital admission 'to get the patient started on a diet' is a waste of resources. Of course it is possible to initiate weight loss in a metabolic ward, but that has no long-term benefit because if the patient is totally unable to diet outside hospital the prognosis is hopeless. However a brief admission (up to three weeks) may be justified in some cases to establish the type of diet and exercise programme which a given patient can reasonably be expected to maintain in the long term. Even the most highly motivated patients do not adhere to dietary restrictions as rigidly at home as they do in a well-run metabolic ward.[97]

Management of obesity in children

There is good evidence that childhood obesity is a risk for ill health in adulthood (*see* section 2), but data on the effects of intentional weight control in childhood on health outcomes are lacking. Table 9 shows that the subsequent health problems of women who are already overweight at the age of 18 years are significantly greater than of those who are not overweight at age 18 years, so it is evidently necessary to prevent excessive weight gain in childhood.

The change in BMI from birth to age 20 years is shown in Figure 11. At birth the average baby is approximately 50 cm long and weighs 3.4 kg, giving a BMI of 13.6. In the first year of life, the normal child trebles its birth weight and grows to 75 cm, giving a BMI of 17.5. Over the next four years height increases more rapidly than weight, so BMI decreases, and the nadir of the 50th centile line is below 16 kg/m^2 at age 5 years. During this period of rapid height growth there are potential dangers of causing permanent stunting if energy intake is restricted. In fact, fat babies often do not stay fat,[98] so it seems prudent to delay weight control measures until after the age of 5 years. By age 12, the 50th centile line has risen to 18 kg/m.^{2} The broken arrow in Figure 11 shows that if a child who was on the 91st centile at age 5 years *did not increase BMI* over the next seven years, he or she would then be on the 50th centile of weight-for-height. A strategy by which this might be achieved is discussed below. The rationale for ceasing weight control measures at age 12 years is that children during and after puberty become much more economically and emotionally independent, and some rebellion against adult authority is a normal phenomenon. If the proposed

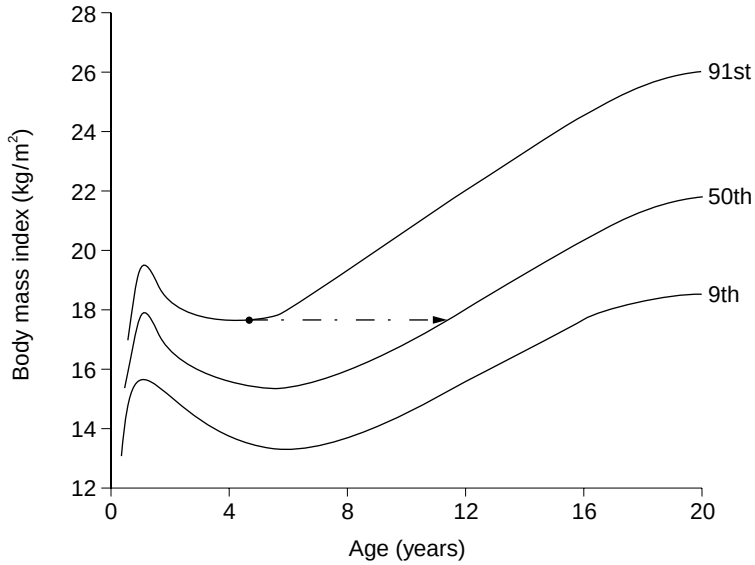

Figure 11: The relationship of BMI to age in a child on the 9th, 50th or 91st centile between birth and age 20 years. The broken horizontal arrow shows that if a child aged 5 years, who is then on the 91st centile, maintains a constant BMI over the next seven years, then by age 12 years he or she would be on the 50th centile of weight-for-height. The years of primary schooling offer an opportunity to prevent obesity in adolescents.[11]

programme has achieved its objective by age 12 years there will be little need for weight control in the secondary school years.

Indeed, a primary school-based intervention study in two towns in northern France is currently assessing the impact of an educational programme in all 6–12-year-olds. The study began in 1992, and body weight evolution will be reported after the ten-year follow-up.[99]

Facilities required to manage overweight among 840 primary school children

A child on the 50th centile for BMI at age 5 years has a weight of 19 kg, height 1.15 m, BMI = 14.4 kg/m.^{2} By age 12 years the corresponding values are weight 40 kg, height 1.5 m, BMI = 17.8 kg/m.^{2} A child with the same height but who is travelling along the 91st centile for BMI will weigh 23 kg, BMI = 17.4 kg/m^2 at 5 years, and by 12 years will weigh 50 kg, BMI = 22.2 kg/m.^{2} Therefore the 50th-centile child will gain 21 kg over the seven years in primary school, while the 91st centile child will gain 27 kg. However, if the 91st-centile child gained only 17 kg (instead of 27 kg) and continued to achieve the same height growth, the result would be that the two children would converge to the same value of BMI, as shown by the broken arrow in Figure 11. In terms of energy balance, an increase of weight by 21 kg indicates a storage of 147 Mcal (600 MJ), and an increase of 17 kg indicates a storage of 119 Mcal (500 MJ). The difference in rate of energy storage between the two over a period of seven years is approximately 11 kcal (46 kJ)/day.

The purpose of this calculation is to show that the degree of average daily energy restriction that is required to convert an overweight 5-year-old into a normal-weight 12-year-old is very small. It can be achieved by substituting fruit and low-energy drinks for sweets and high-energy drinks, and by encouraging extra exercise. It should be noted that weight *loss* is not part of the objective: what is required is a slowing of weight gain.

The problem with this lies not in the thermodynamics, but in the social implications of weight control of young children. Great care is required that well-intentioned schemes do not make matters worse by causing social stigmatisation of the overweight child. It is for this reason that Figure 11 has the 9th centile line also, in order that underweight children are also selected as having special healthcare needs, as would children with problems of hearing or vision. It seems likely that the ideal model would be for children at entry to primary school entry to have their BMI determined. Those who fell below the 9th, or above the 91st, centile line in Figure 11 would be referred to a special needs nurse, who would undertake monitoring of weight and height growth of these children during the next seven years. This should be done with the minimum of fuss, and with the help of the child's parents, teachers and school physical education and catering staff, with referral to a paediatrician if necessary.

8 Outcome measures

The simplest criterion of the prevalence of obesity among adults is measured height and weight so that BMI can be calculated. BMI can be audited at any level, and a cross-sectional sample is measured annually by the Health Surveys for England. The success of a programme to manage obesity will also be indicated by the decrease in co-morbidities such as heart disease, diabetes, gallstones, osteoarthritis, sleep apnoea, some sex-hormone-sensitive cancers and the psychopathologies associated with severe obesity. These criteria, though important, are difficult to interpret, since changes in the prevalence of these diseases may be confounded by changes not associated with obesity status, such as cigarette smoking.

The success of a programme for preventing obesity in children can be assessed by measuring the prevalence of overweight in children at age 12 years.

9 Information and research requirements

There are many questions to which we need answers. For example:

- the influence of dietary fat intake on obesity prevalence and treatment
- the efficacy of different preventative programmes, especially those involving exercise
- the relative influence of genetics and environment to the familial aggregation of obesity
- the efficacy of strategies for maintenance of weight loss
- the cost of preventing and treating obesity.

A more extensive list of topics that require further research is set out in recent reports.[7,11,59]

References

1 Waterlow JC (chairman). *Research on obesity: report of a DHSS/MRC group*. London: HMSO, 1976.
2 Bray GA (ed.). *Obesity in America*. NIH Publication No 79–358. Washington: US Dept of Health Education and Welfare, 1979.
3 World Health Organization. *Obesity: Preventing and Managing the Global Epidemic*. Report of a WHO Consultation. WHO Technical Report Series no. 894. WHO: Geneva, 2000.
4 World Health Organization. *Report of a Joint WHO/FAO Expert Consultation. Diet, Nutrition and the Prevention of Chronic Diseases*. WHO Technical Report Series no. 916. WHO: Geneva, 2002.
5 House of Commons Health Committee. *Obesity*. Third Report of Session 2003–2004. Volume 1. House of Commons. The Stationery Office, London, 2004.
6 National Audit Office. *Tackling obesity in England*. NAO. London: The Stationery Office, 2001.
7 Avenell A, Broom J, Brown TJ, Poobalan A, Aucott L, Stearns C *et al*. Systematic review of the long-term effects and economic consequences of treatments for obesity and implications for health improvement. *Health Technol Assess* 2004; **8**/21. Available at www.ncchta.org
8 World Health Organization (WHO), International Obesity Task Force (IOTF). *Obesity: preventing and managing the global epidemic*. Report of a WHO consultation on obesity, Geneva, June 3–5 1997. Geneva: WHO, 1998.
9 Seidell JC, Flegal KM. Assessing obesity: classification and epidemiology. *Br Med Bull* 1997; **53**: 238–52.
10 Power C, Lake JK, Cole TJ. Measurement and long-term health risks of child and adolescent fatness. *Int J Ob* 1997; **21**: 507–26.
11 IOTF. Obesity in children and young people: a crisis in public health. *Obesity Reviews* 2004; **5** (suppl 1): 1–104.
12 Parsons T, Power C, Logan S, Summerbell CD. Childhood predictors of adult obesity: a systematic review. *Int J Ob* 1999; **23** (suppl 8): S1–107.
13 Must A, Jacques PF, Dallai GE, Bajema CJ, Dietz WH. Long term morbidity and mortality of overweight adolescents. *N Engl J Med* 1992; **327**: 1350–5.
14 Crawley HF, Portides G. Self-reported versus measured height, weight and body mass index amongst 16–17 year old British teenagers. *Int J Ob* 1995; **19**: 579–84.
15 Eckel RH, Krauss RM, for the AHA Nutrition Committee. American Heart Association call to action: obesity as a major risk factor for coronary heart disease. *Circulation* 1998; **97**: 2099–100.
16 Manson JE, Willett WC, Stamfer MJ, Colditz GA, Hunter DJ, Hankinson SE, Hennekins CH, Speizer FE. Body weight and mortality among women. *N Engl J Med* 1995; **333**: 677–85.
17 Bender R, Jockel K-H, Trautner C, Spraul M, Berger M. Effect of age on excess mortality in obesity *JAMA* 1999; **281**: 1498–504.
18 The National Heart, Lung, and Blood Institute (NIH). *Clinical guidelines on the identification, evaluation, and treatment of overweight and obesity in adults*. Bethesda, Maryland: National Institutes of Health, 1998. Available at website: www.nhlbi.nih.gov/guidelines/obesity/ob_gdlns.pdf
19 Lee CD, Blair BN, Jackson AS. Cardiorespiratory fitness, body composition, and all-cause and cardiovascular disease mortality in men. *Am J Clin Nutr* 1998; **69**: 373–80.
20 Rissanen A, Heliovaara M, Knekt P, Reunanen A, Aromaa A, Maatela J. Risk of disability and mortality due to overweight in a Finnish population. *BMJ* 1990; **301**: 835–6.
21 Lew EA, Garfinkel L. Variations in mortality by weight among 750,000 men and women. *J Chronic Dis* 1979; **32**: 563–76.
22 Sims EAH, Danforth E Jr, Horton ES, Bray GA, Glennon JA, Salans LB. Endocrine and metabolic effects of experimental obesity in man. *Recent Progress Hormone Res* 1973; **29**: 457–96.

23 Hoffman RP, Armstrong PT. Glucose effectiveness, peripheral and hepatic insulin sensitivity in obese and lean prepubertal children. *Int J Ob* 1996; **20**: 521–5.
24 Scheen AJ, Paquot N, Letiexhe MR, Paolisso G, Castillo MJ, Lefebvre PJ. Glucose metabolism in obese subjects: lessons from OGTT, IVGTT and clamp studies. *Int J Obes* 1995; **19** (suppl 3): S14–S20.
25 Williamson DF, Pamuk E, Thun M, Flanders D, Byers T, Heath C. Prospective study of intentional weight loss and mortality in never-smoking overweight US white women aged 40–64 years. *Am J Epid* 1995; **141**: 1128–41.
26 Chen Y, Rennie DC, Reeder BA. Age-related association between body mass index and blood pressure: the Humbolt study. *Int J Obe*s 1995; **19**: 825–31.
27 Dattilo AM, Kris-Etherton PM. Effects of weight reduction on blood lipids and lipoproteins: a meta-analysis. *Am J Clin Nutr* 1992; **56**: 320–8.
28 Manson JE, Colditz GA, Stamfer MJ, Wuillett WC, Rosner B, Monson RR, Speizer FE, Hennekens CH. A prospective study of obesity and risk of coronary heart disease in women. *N Engl J Med* 1990; **322**: 822–9.
29 Meade TW, Ruddock V, Stirling Y, Chakrabati R, Miller GJ. Fibrinolytic activity, clotting factors and long-term incidence of ischaemic heart disease in the Northwick Park study. *The Lancet* 1993; **342**: 1076–9.
30 Ernst E, Matrai A. Normalisation of hemorheological abnormalities during weight reduction in obese patients. *Nutrition* 1987; **3**: 337–9.
31 Garfinkel L. Overweight and cancer. *Ann Intern Med* 1985; **103**: 1034–6.
32 Davis MA, Neuhaus JM, Ettingrer WH, Mueller WH. Body fat distribution and osteoarthritis. *Am J Epid* 1990; **132**: 701–7.
33 Spirt BA, Graves LW, Weinstock R, Bartlett SJ, Wadden TA. Gallstone formation in obese women treated by a low-calorie diet. *Int J Ob* 1995; **19**: 593–5.
34 Galtier-Dereure F, Montpeyroux F, Boulot P, Bringer J, Jaffiol. Weight excess before pregnancy: complications and cost. *Int J Ob* 1995; **19**: 443–8.
35 Shaw GM, Velie EM, Schaffer D. Risk of neural tube defect-affected pregnancies among obese women. *JAMA* 1996; **275**: 1093–6.
36 Werler MM, Louik C, Shapiro S, Mitchell AA. Pre-pregnant weight in relation to risk of neural tube defects. *JAMA* 1996; **275**: 1089–92.
37 Grunstein RR, Stenlof K, Hedner J, Sjostrom L. Impact of obstructive apnoea and sleepiness on metabolic and cardiovascular risk factors in the Swedish Obese Subjects (SOS) study. *Int J Ob* 1995; **19**: 410–18.
38 Harman EM, Block AJ. Why does weight loss improve the respiratory insufficiency of obesity? *Chest* 1986; **90**: 153–4.
39 Chernin K. *The Tyranny of Slenderness*. London: The Women's Press, 1981.
40 Wadden TA, Stunkard AJ. Social and psychological consequences of obesity. *Ann Intern Med* 1985; **103**: 1062–7.
41 Sonne-Holm S, Sorensen TI. Prospective study of attainment of social class of severely obese subjects in relation to parents social class, intelligence and education. *BMJ* 1986; **292**: 586–9.
42 Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *N Engl J Med* 1993; **329**: 1008–12.
43 Sullivan M, Karlsson J, Sjostrom L, Backman L, Bengtson C, Bouchard C, Dahlgren S, Jonsson E, Larsson B, Lindstedt S, Naslund I, Olbe L, Wedel H. Swedish obese subjects (SOS) – an intervention study of obesity. Baseline evaluation of health and psychosocial functioning in the first 1743 subjects examined. *Int J Ob* 1993; **17**: 503–12.
44 Ogden J, Chanana A. Explaining the effect of ethnic group on weight concern: finding a role for family values. *Int J Ob* 1998; **22**: 641–7.

45 Striegel-Moore RH, Wilfley DE, Caldwell MB, Needham ML, Brownell KD. Weight-related attitudes and behaviors of women who diet to lose weight: a comparison of black dieters and white dieters. *Ob Res* 1996; **4**: 109–16.
46 Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E. Sjostrom L. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow-up of participants in the population study of women in Gothenburg, Sweden. *BMJ* 1984; **289**: 1257–61.
47 Larsson B. Regional obesity as a health hazard in men – prospective studies. *Acta Med Scand Suppl* 1988; **723**: 45–51.
48 Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, Byers T. Do obese children become obese adults? A review of the literature. *Prev Med* 1993; **22**: 167–77.
49 Power C, Lake JK, Cole TJ. Body mass index and height from childhood to adulthood in the 1958 British born cohort. *Am J Clin Nutr* 1997; **66**: 1094–101.
50 Dietz WH. Critical periods in childhood for the development of obesity. *Am J Clin Nutr* 1994; **59**: 955–9.
51 Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics* 1998; **101** (suppl): 518–25.
52 Weiss R, Dziura J, Burgert TS, Tamborlane WV, Taksali SE, Yeckel CW, Allen K, Lopes M, Savoye M, Morison J, Sherwin RS, Caprio S. Obesity and the metabolic syndrome in children and adolescents. *N Engl J Med* 2004; **350**: 2362–74.
53 Stunkard A, Burt V. Obesity and the body image. II. Age at onset of disturbances in the body image. *Am J Psychiatry* 1967; **123**: 1443–7.
54 Hill AJ, Silver EK. Fat, friendless and unhealthy: 9-year-old children's perception of body shape stereotypes. *Int J Ob* 1995; **19**: 423–30.
55 Royal College of Physicians (RCP). *Clinical Management of Overweight and Obese Patients with Particular Reference to the Use of Drugs*. London: RCP, 1998.
56 Scottish Intercollegiate Guidelines Network (SIGN). *Obesity in Scotland: integrating prevention with weight management*. Edingburgh: SIGN, 1996. Also available (updated version) at website: http://show.cee.hw.ac.uk/sign/home.htm
57 British Nutrition Foundation (BNF). *Obesity*. Report of the British Nutrition Foundation Task Force (J Garrow, chairman). Oxford: Blackwell Science, 1999.
58 Department of Health. *Our Healthier Nation: saving lives*. London: HMSO, 1999.
59 Health Development Agency. The management of obesity and overweight: An analysis of reviews of diet, physical activity and behavioural approaches. Evidence Briefing. October 2003.
60 NICE, National Institute for Clinical Excellence (2001) Orlistat for the treatment of obesity in adults. Guidance No 22. Available at: www.nice.org.uk
61 NICE, National Institute for Clinical Excellence (2001) Guidance on the use of Sibutramine for the treatment of obesity in adults. Guidance No 31. Available at: www.nice.org.uk
62 NICE, National Institute for Clinical Excellence (2002) Guidance on the use of surgery to aid weight reduction for people with morbid obesity. Guidance No 46. Available at: www.nice.org.uk
63 Scottish Intercollegiate Guidelines Network Obesity in Scotland. Management of obesity in children and young people. Guideline No 69, Published by Scottish Intercollegiate Guidelines Network, Edinburgh, 2003. Also available at website: www.sign.ac.uk/
64 Keys A, Menotti A, Aravanis C, Blackburn H, Djordevic BS, Buzina R, Dontas AS, Fidanza F, Karvonen MJ, Kimura N, Mohacek I, Nedeljkovic S, Puddu V, Punsar S, Taylor HL, Conti S, Kromhout D, Toshima H. The seven countries study: 2289 deaths in 15 years. *Prev Med* 1984; **13**: 141–54.
65 Hubert HB. The nature of the relationship between obesity and cardiovascular disease. *Int J Cardiol* 1984; **6**: 268–74.
66 Orbach S. *Fat is a Feminist Issue*. London: Hamlyn, 1978.

67 Apfelbaum M, Vague P, Ziegler O, Hanotin C, Thomas F, Leutenegger E. Long-term maintenance of weight loss after a very-low-calorie diet: a randomized blinded trial of the efficacy and tolerability of sibutramine. *Am J Med* 1999; **106**: 179–84.
68 Bray GA, Blackburn GL, Ferguson JM, Greenway FL, Jain AK, Mendel CM, Mendels J, Ryan DH, Schwartz SL, Scheinbaum ML, Seaton TB. Sibutramine produces dose-related weight loss. *Ob Res* 1999; **7**(2): 189–98.
69 Jeffery RW, Folsom AR, Leupker RV, Jacobs DR, Gillum RF, Taylor HL, Blackburn H. Prevalence of overweight and weight loss behaviour in a metropolitan adult population: the Minnesota heart survey experience. *Am J Public Health* 1984; **74**: 349–52.
70 Summerbell CD. Teaching nutrition to medical doctors: the potential role of the State Registered Dietitian. *J Hum Nutr Diet* 1996; **9**: 349–56.
71 Kopelman, P. Place of obesity clinics in the NHS. *Brit J Hosp Med* 1993; **49** (8); 533–5.
72 Grace C, Summerbell C, Kopelman P. An audit of dietary treatment modalities and weight loss outcomes in a specialist obesity clinic. *J Hum Nutr Diet* 1998; **11**(3): 197–202.
73 Cowburn G, Summerbell C. A survey of dietetic practice in obesity management. *J Hum Nutr Diet* 1998; **11**: 191–6.
74 Wilkinson J, Summerbell C, Macknight N, Bailey K, Chappel D. Use of Surgery to Aid Weight Reduction – Experience of Two Regions of Northern England – a database study. *Int J Ob* (in press, 2004).
75 Summerbell CD, Higgins JPT. Systematic reviews and meta-analysis. *BNF Bulletin* 1997; **22**: 111–8.
76 Campbell K, Waters E, O'Meara S, Kelly S, Summerbell C. Interventions for preventing obesity in children (Cochrane Review). In: The Cochrane Library, Issue 2, 2004. Chichester, UK: John Wiley & Sons, Ltd.
77 Summerbell CD, Ashton V, Campbell KJ, Edmunds L, Kelly S, Waters E. Interventions for treating obesity in children (Cochrane Review). In: The Cochrane Library, Issue 2, 2004. Chichester, UK: John Wiley & Sons, Ltd.
78 Bortz WM. A 500 pound weight loss. *Amer J Med* 1969; **47**: 325–31.
79 Sjostrom L, Rissanen A, Andersen T, Boldrin M, Golay A, Koppeschaar HP *et al.* Randomised placebo-controlled trial of orlistat for weight loss and prevention of weight regain in obese patients. European Multicentre Orlistat Study Group. *The Lancet* 1998; **352**: 167–72.
80 Webster JD, Hesp R, Garrow JS. The composition of excess weight in obese women estimated by body density, total body water and total body potassium. *Hum Nutr Clin Nutr* 1984; **38C**: 299–306.
81 Stuart RB. Behavioural control of overeating. *Behav Res Ther* 1967; **5**: 357–65.
82 Garrow JS, Summerbell CD. Meta-analysis: effect of exercise, with or without dieting, on the body composition of overweight subjects. *Eur J Clin Nutr* 1995; **49**: 1–10.
83 Torgerson JS, Sjostrom L. The Swedish Obese Subjects (SOS) study – rationale and results. *Int J Obes* 2001; **25**: 52–4.
84 NHS CRD (Centre for Reviews and Dissemination). A systematic review of the interventions for the prevention and treatment of obesity, and the maintenance of weight loss. CRD Report 10, 1997, University of York. Also available at website: http://www.york.ac.uk/inst/crd/obesity.htm
85 Douketis JD, Feightner JW, Attia J, Feldman WF, with the Canadian Task Force on Preventative Health Care. Periodic health examination, 1999 update: 1. Detection, prevention and treatment of obesity. *J Can Med Ass* 1999; **160**: 513–25.
86 Hardeman W, Griffin S, Johnston M, Kinmonth AL, Wareham NJ. Interventions to prevent weight gain: a systematic review of psychological models and behaviour change methods. *Int J Ob* 2000; **24**: 131–43.
87 Harvey EL, Glenny A-M, Kirk SFL, Summerbell CD. Effective Professional Practice: a systematic review of health professionals' management of obesity. *Int J Ob* 1999; **23**(12): 1213–22.

88 Moore H, Summerbell CD, Greenwood D, Tovey P, Griffiths J, Henderson M, Hesketh K, Woolgar S, Adamson AJ. Improving the management of obesity in primary care: a cluster randomised trial. *BMJ* 2003; **327**: 1085–9.
89 French SA, Jeffery RW, Folsom AR, McGovern P, Williamson DF. Weight loss maintenance in young adulthood: prevalence and correlations with health behaviour and disease in population-based sample of women aged 55-69 years. *Int J Ob* 1996; **20**: 303–10.
90 Allison DB, Zannolli R, Faith MS, Heo M, Pietrobelli A, Van Itallie TB, Pi-Sunyer FX, Heymsfield SB. Weight loss increases and fat loss decreases all-cause mortality rate: results from two independent cohort studies. *Int J Ob* 1999; **23**: 603–11.
91 Yanovski SZ, Bain RP, Williamson DF. Report of a National Institute of Health – Centers for Disease Control and Prevention Workshop on the feasibility of conducting a randomised clinical trial to estimate the long-term health effects of intentional weight loss in obese persons. *Am J Clin Nutr* 1999; **69**: 366–72.
92 Narbro K, Agren G, Jonsson E, Larsson B, Naslund I, Wedel H, Sjostrom L. Sick leave and disability pension before and after treatment for obesity: a report from the Swedish Obese Subjects (SOS) study. *Int J Ob* 1999; **23**: 619–24.
93 Bush A, Webster J, Chalmers G, Pearson M, Penfold P, Brereton P, Garrow JS. The Harrow slimming club: Report on 1090 enrolments in 50 courses, 1977–1986. *J Hum Nutr Diet* 1988; **1**: 429-36.
94 Karvetti RL, Hakala P. A 7-year follow-up of a weight reduction programme in Finnish primary health care. *Eur J Clin Nutr* 1992; **46**: 743–52.
95 Andres R, Elahi D, Tobin JD, Muller DC, Brant L. Impact of age on weight goals. *Ann Int Med* 1985; **103**: 1030–3.
96 French SA, Jeffery RW, Folsom AR, Williamson DF, Byers T. Weight variability in a population-based sample of older women: reliability and intercorrelation of measures. *Int J Ob* 1995; **19**: 22–9.
97 Garrow JS, Webster JD, Pearson M, Pacy PJ, Harpin G. Inpatient-outpatient randomised comparison of Cambridge diet versus milk diet in 17 obese women over 24 weeks. *Int J Obes* 1989; **13**: 521–9.
98 Poskitt EME, Cole TJ. Do fat babies stay fat? *BMJ* 1977; **1**: 7–9.
99 Basedevant A, Boute D, Borys JM. Who should be educated? Education strategies: could children educate their parents? *Int J Ob* 1999; **23** (suppl 4): S10–S13.