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Why Treat Obesity?

'Obesity poses a threat on a similar scale to climate change' [1]

What is the scale of the obesity problem?

Obesity is one of the most serious and complex health challenges faced by the UK, Europe and most of the rest of the world. There has been a dramatic doubling in its prevalence over the last 25 years with most adults in England now overweight, and 1 in 4 obese (24% men; 25% women) [2]. Alarming trends have been observed in children, with 14.4% of 2–10 year olds obese in 2009 [3]. If current trends continue, future prevalence predictions are dire, with suggestions that 9 in 10 adults in England could be obese by 2050 [4].

This will have profound cost implications for the NHS and the wider economy. Current estimates for the NHS suggest obesity costs £4.2 billion/year, with wider economic costs (reduced productivity, loss of earnings, increased benefits) of £16 billion/year. If future prevalence predictions are accurate, this may rise to £10 billion/year and £50 billion/year, respectively [4].

A strong social inequality exists in the prevalence of obesity, although the factors responsible are unclear: in men, 18% are obese in social class I versus 28% in social class V; in women, 10% are obese in social class I versus 25% in social class V [5]. Unfortunately, there is limited information on whether prevalence varies by ethnicity as most surveys only include small numbers from various ethnic groups. However, in 2004, a higher prevalence of obesity was found in black African, black Caribbean and Pakistani women compared to the general population [6].

Why does it matter?

Obesity is known to shorten life, is a risk factor for a range of major co-morbidities and can have profound effects on an individual's psychological and social well being. There are also wider economic and social consequences for society that make addressing obesity a compelling, albeit challenging, issue.

Obesity and early death

Obesity shortens life

Up to 13 years of life can be lost in obese men and up to 8 years in obese women [7]

Obesity increases the risk of dying early, particularly in men. In young adults there is a 50% greater risk of early death in those with body mass index (BMI) above 30 kg/m² compared to healthy-weight individuals [8]. Over the years there has been debate on the precise relationship between mortality risk and obesity. However, after appropriate adjustment for confounders, an elevated BMI is clearly linked with increased risk of premature death [9–11]. Obese white men aged 20–30 years with a BMI greater than 45 kg/m² are likely to lose 13 years of their life and for women with similar characteristics this can be up to 8 years [7]. The link between mortality risk and BMI is greatest up to the age of 50 but does continue through to old age [12]. Risk can be moderated depending on the level of physical fitness, with suggestions that being overweight and inactive may account for up to 31% of early all-cause mortality [13].

Obesity and type 2 diabetes

Diabetes risk

Rapid rise above BMI 25 kg/m²
Longer obesity duration = greater diabetes risk
Even small increases in weight increase risk

Of all the associated co-morbidities, type 2 diabetes is the most strongly linked. Increasing fat mass, particularly abdominal/visceral obesity, is well recognised as a risk factor for the development of type 2 diabetes, due to its contribution to insulin resistance and beta cell dysfunction. The BMI above which diabetes risk begins to rise rapidly is surprising low, with a 3.6-fold greater risk in women with a BMI of 23–24 kg/m² compared to those with a BMI <22 kg/m², highlighting that this association is not the sole reserve of the severely obese [14]. For those aged 40–49 years with BMI >35 kg/m², risk of developing diabetes has been found to be almost 80 times higher than in those with a BMI of <22 kg/m² [15].

The longer the duration of obesity and weight gain, the higher the level of risk, with a 3-fold elevated risk in those who have been overweight for less than 5 years versus a 5-fold risk in those who have been overweight for more than 5 years [16]. Several studies have shown that individuals with small weight gains in early adulthood of ~5–8 kg have twice the risk of diabetes compared to those who have minimised weight gain [15,17], emphasising the importance of preventing weight gain.

The risk of diabetes varies by ethnicity and is especially high in those of Asian origin. For each 5 kg weight gain, the risk of diabetes increased by 37% in whites, 38% in blacks but 84% in Asians.

Weight gain of 5 kg increases diabetes risk by 84% in Asians versus 37% in whites

Obesity and cancer

A BMI of ≥ 40 kg/m² has been associated with a 50–60% increased chance of developing cancer compared to healthy-weight individuals [9]. Obesity has been specifically implicated in cancer of the colon, endometrium and breast. A 1.5-fold greater risk of developing colorectal cancer has been found in women with a BMI greater than 29 kg/m² and in men with abdominal obesity (waist–hip ratio, WHR ≥ 0.99) [18,19]. Dietary factors (red and processed meats may exacerbate, while fibre and n-3 PUFA may protect) and physical inactivity (high activity levels may protect) have also been linked to the risk of colon cancer.

Obesity and cardiovascular disease

Obesity is a major modifiable risk factor for coronary heart disease. Its association with various atherogenic lipid and lipoprotein abnormalities is well recognised, including elevated total cholesterol and triglyceride, and lowered high-density lipoprotein cholesterol [20]. It is this link with atherogenesis, together with its negative impact on other coronary risk factors (hypertension, type 2 diabetes), that explains the strong positive association between the incidence of coronary heart disease and obesity [21]. It has been estimated that as much as 70% of the coronary heart disease in obese women is attributable to overweight [22]. The distribution of adipose tissue is also known to be important, with central obesity increasing metabolic risk via a greater predisposition to dyslipidemia [23].

Quality of life

Research clearly illustrates that obesity has an adverse effect on health-related quality of life, with the magnitude of impairment increasing with increasing severity of obesity [24]. Conversely, improvements are reported after weight loss, although most research has explored changes after surgery rather than changes related to lifestyle approaches [25]. Obesity affects many aspects of physical and social functioning, sexual function and satisfaction, public distress and the ability to engage fully in the workplace.

Whether obesity leads to or is a consequence of depression has been hotly debated and there is a need for greater understanding of this complex relationship. A recent meta-analysis concluded that depression and obesity were reciprocal, with an increased risk of depression in the obese, and with depression being predictive for obesity [26].

Factors that increase the risk of obesity

Smoking cessation

Giving up smoking is commonly associated with an average weight gain of 7kg [27], although this varies by age, lifestyle behaviours and socioeconomic status. There are a number of possible reasons for this link, including: the removal of the appetite suppressing effect of nicotine; an improved sense of taste and smell leading to altered food preferences; swapping oral gratification from smoking to food; and behaviourally using food in the same way as cigarettes – for example, to deal with stress, boredom, self-rewards or as a means of socialising.

Although over 80% of those quitting smoking will gain weight, the health benefits of smoking cessation far outweigh the health risks of gaining weight.

To reach the same health risk as smoking one packet of cigarettes a day, the average smoker would need to be 55kg overweight

As the evidence currently stands, the optimal timing of weight management and quit attempts is unclear. There is some concern that trying to control weight through lifestyle interventions while trying to quit smoking may negatively impact on the success of smoking cessation. Until it is clear that concurrent weight management does not lead to an increase in quit failure it may be wise to reserve weight-management interventions until smoking cessation has been successfully completed. However, there may be instances when an individual is so concerned about the possibility of weight gain that it adversely affects their motivation to stop smoking. Such situations require clinical judgment to determine whether individualised weight management alongside smoking cessation would be beneficial.

The provision of general advice ‘to avoid gaining weight’ while trying to quit smoking is generally ineffective and may hinder smoking cessation attempts. However, individualised weight-management interventions limit the extent of weight gain during the smoking cessation period, although the effect is small. The use of cognitive behavioural therapy and very low-calorie diets alongside smoking cessation treatments may be beneficial in reducing post-cessation weight gain. Longer-term studies are required and it is recommended that these strategies are reserved for use in research settings [27].

The role of physical activity in managing weight during and after smoking cessation is a little unclear, although it may be important for improved weight control over the longer term [27,28].

Just advising people planning to stop smoking to avoid gaining weight is unhelpful and may prevent the attempt to quit.

There is insufficient evidence to determine the optimal timing of weight-management interventions and smoking cessation.

It may be most prudent to wait until after a successful quit attempt has been completed before considering weight-management interventions.

The decision to offer individualised weight-management interventions concurrently with a quit attempt should be made on an individual basis using clinical judgment.

Certain medications

There are certain medications known to increase the risk of weight gain and some of those listed below have been associated with up to a 10 kg gain over 12 weeks [29]. It may be helpful to discuss weight-management options in instances where the prescribing of such medications is necessary and an alternative is not suitable.

- atypical antipsychotics, including clozapine;
- beta adrenergic blockers, particularly propranolol;
- insulin, when used in the treatment of type 2 diabetes mellitus;
- lithium;
- sodium valproate;
- sulphonylureas, including chlorpropamide, glibenclamide, glimepiride and glipizide;
- thiazolidinediones, including pioglitazone;
- tricyclic antidepressants, including amitriptyline.

To date there is no evidence to suggest a link between oral combined contraceptives or hormone replacement therapy and weight gain [30].

Obesity and its causes

Obesity is commonly misconstrued as a self-inflicted condition, the causes of which are simple: eating too much and exercising too little. This is far removed from the complex nature of obesity revealed by science, and such misunderstandings tend to fuel weight-related stigma and do little to enhance obesity treatments.

Why do practitioners need a good understanding of obesity causes?

Developing a broad understanding of the complex biological and environmental factors involved in the development of obesity may have a number of important benefits:

- 1 **Positive impact on the practitioner's attitudes to obese people (Chapter 2)** Acknowledging obesity is not self-inflicted, and patients are often pushing back against strong biological tendencies and a challenging environment helps practitioners understand the challenge of weight management.
- 2 **Improvement of the assessment process** A better understanding of the factors involved in obesity development may lead to a more sophisticated assessment of these elements.
- 3 **Improvement of the therapeutic relationship** Discussing issues of predisposition can convey understanding and optimism. The information that some people are more predisposed to obesity can be helpful for those who find it difficult to understand, and accept, why they find it harder to control their weight than others. It may allay feelings of self-blame, guilt and shame and is likely to convey a sense of support and understanding on the part of the practitioner.

Society views obesity as the result of personal failure (often lack of willpower) rather than influenced by environmental and genetic factors. Patients (and some health professionals) often view obesity in the same way. Summarising for patients what science tells us about the causes of obesity can counter these misconceptions, may allay guilt and self-blame, and can empower patients to address their obesity.

Consider this

If, as an obese person, a patient's starting point for managing their weight is this:

'I'm failing to lose weight because I don't have enough willpower. Why am I so useless at dieting when lots of my friends seem to have no difficulty? I know it's my fault and I need to do something about it but I don't know where to start. I suppose I just need to try harder but I'm not sure I can.'

Ask yourself:

- 1 What is the likely effect of their beliefs about the causes of their obesity on their self-esteem?
- 2 How confident are they likely to be about their ability to change behaviour and weight?
- 3 Is this a good place from which to begin a weight-management programme?

After discussion, the patient's attitude could be this:

'I've struggled with my weight for years and I've always blamed myself. Why couldn't I just eat less and do more exercise? It sounds so simple but I've failed at it time and time again. Now I understand that although some of it is clearly about me and the choices I make, it's not all my fault. That makes me feel so much better about myself and when I feel better about myself I eat less. Knowing that some people's bodies are much better set up to control weight is really helpful. I know my weight is always going to be something I need to take real care with, but I feel determined now to do something about this.'

What are the causes of obesity?

There is no single cause of obesity and no one dominant causal factor. Rather, obesity develops as a consequence of a complex mix of genetic and environmental factors, the contributions and relevancies of which vary from one individual to another. This degree of causal complexity is elegantly illustrated in the Foresight Report: (http://www.bis.gov.uk/assets/bispartners/foresight/docs/obesity/obesity_final_part2.pdf) (see diagram on page 84).

At the most simplistic level, energy balance explains why weight gain occurs. If energy consumed from food/drink exceeds that expended through physical activity (and metabolism), a positive energy balance occurs and weight gain is the result. However, this explanatory model provides an incomplete picture and does not highlight the many complicated mechanisms that influence why someone might consume more energy, or do little physical activity.

It is now widely accepted that the rapid rise in the prevalence of obesity has been driven by technological advances which have dramatically changed the way we live. This has impacted on work patterns, modes of transport, food production,

meal preparation and shopping practices, and this changed environment has revealed an underlying tendency for many people to gain weight. The Foresight report [1] describes this as ‘passive obesity’: an almost involuntary process of weight gain where the environmental drivers of obesity have been so overwhelming that weight gain is inevitable for many people. This highlights the inaccuracy of considering obesity a self-inflicted condition in which sole responsibility for change lies with the individual.

At an individual level the causes of obesity are numerous and will vary from person to person. Across an individual’s life span the causes of weight gain may also change. This underlines the importance of comprehensive assessment (Chapter 6) so that the specific factors that have contributed to weight gain over time can be identified, and modifiable elements addressed through management.

Biology and genes

Food intake and appetite regulation

Substantial advances have occurred in our understanding of the specific mechanisms involved in appetite regulation, many of which have come from animal models of obesity. The body attempts to regulate weight and food intake through a complex biological system of hormonal and neural pathways and feedback loops. Appetite regulation is extremely complex and centres on the hypothalamus and brain stem as the key regulators of energy balance in the brain. They receive information from the gastrointestinal tract, adipose tissue and circulation about current fat stores and nutritional status, and analyse and then modify responses accordingly. There are numerous complex pathways and signals involved and much has been learnt about signals and systems that stimulate (Neuropeptide Y, Agouti protein, melanin concentrating hormone) or terminate (POMC, corticotrophin-releasing factor) feeding. Leptin, secreted by adipose tissue, is probably the best-known signal and seems to function as a trigger to increase feeding when fat stores are low. Leptin levels fall as fat stores decline, leading to an increase in hunger and food-seeking behaviour. Conversely, as fat stores increase higher leptin levels signal the hypothalamus, satiety occurs, food intake subsequently falls and equilibrium is achieved. However, there is a great deal still to be learnt about the functioning of this complex system and the interactions between the many signals and pathways.

As the obesity epidemic illustrates, this control system has struggled to adapt to our rapidly changing environment and ‘the pace of technological progress has outstripped human evolution’ [1]. It seems that appetite regulation may be more tightly controlled in naturally thin people, with greater precision in the matching between energy consumed and energy expended. In those predisposed to obesity, the appetite control system may be less proficient at matching, and unless eating and activity behaviours are consciously controlled, weight gain occurs. Research has illustrated that, appetite control mechanisms can be easily overridden by the sight, smell, palatability and availability of foods [31] – sensory factors that are so abundant in today’s environment.

Energy expenditure

Research has explored the ‘energy sparing’ aspects of metabolism to determine whether there might be components of energy expenditure which make substantial contributions to the aetiology of obesity. There is no evidence to support the idea that obesity is caused by a slow metabolism. Indeed, research suggests that resting energy expenditure tends to be higher in obese people because of the higher metabolic cost of larger body sizes. After adjustment for body weight and composition there is hardly any difference between resting metabolism in obese and lean people [32]. Indeed, no physiological differences have been found that might explain why lean people avoid weight gain in the current environment, and in studies where overfeeding has occurred similar rates of weight gain or loss have been seen.

Early growth patterns

Nutrition and growth in the womb and early childhood seem to influence obesity risk in later life, although the mechanisms for this are unclear. Low-birth-weight babies have a higher risk of heart disease and diabetes in adulthood, and this may be linked with rapid weight gain and feeding practices in the first few months of life [33]. Metabolism may be plastic in the first few months of life and the nutritional environment may play a role in ‘setting’ the baby’s metabolic pathway. This highlights the importance of healthy nutrition and lifestyle choices in early life.

Eating and activity behaviours

These are clearly critical elements of influence on obesity; however, attempts to understand their precise contribution are hampered by the methods available to measure food intake and physical activity. Measuring dietary intake relies on reported rather than actual intake and obesity is known to be associated with substantial under-reporting of energy intake. Nonetheless, a number of dietary risk factors have been identified, including high-energy dense foods, diets high in fat and low in fibre, sugary drinks and large portion sizes.

The obesogenic environment

Recent environmental changes are commonly linked with the increasing prevalence of obesity, although the magnitude of their involvement isn’t fully understood. Technological advances (cars, washing machines, dishwashers) have reduced the effort required for many everyday activities and together with reduced occupational activity are likely to encourage obesity. Likewise the increased access and availability of relatively cheap, but not necessarily healthy food, and increasingly unstructured meal patterns make the healthy choice more challenging.

Health benefits of modest weight loss

Although many patients will strive to achieve an 'ideal' body weight, the inherent challenges in weight management may mean this isn't feasible (depending on baseline BMI) with dietary intervention alone. In fact, it may not be necessary. There is good evidence to support the value of modest weight loss in improving health and psychological well being. Indeed, it can be argued that losing and sustaining modest amounts of weight is better than losing and then regaining substantial quantities of weight.

It is recommended that weight-loss targets are not based simply on weight alone but include the patient's existing co-morbidities and risks. In those with a BMI between 25 and 35 kg/m², where co-morbidities are less likely, a 5–10% weight loss is often sufficient to reduce cardiovascular and metabolic risk. However, in those with a BMI greater than 35 kg/m², where co-morbidities may be present, weight loss of 15–20% may be necessary before sustained improvements to co-morbidities occur [29].

BMI 25–35 kg/m ²	5–10% target weight loss
BMI >35 kg/m ²	15–20% target weight loss

Modest sustained weight loss (5–10%) has been associated with the following:

- **Mortality** Reduced all-cause mortality in those with diabetes and lower cancer- and diabetes-related mortality in obese women with some obesity-related co-morbidity [34].
- **Asthma** Improved lung function (if more than 10 kg lost) [35].
- **Arthritis** Reduced osteoarthritis-related disability (5% weight loss associated with improved function and reduced pain) [36]. Improved mobility and reduced pain reported in adults over 60 years with established osteoarthritis [37].
- **Blood pressure** A reduction of 3.8–4.4 mmHg in systolic and 3–3.6 mmHg in diastolic blood pressure at 12 months with a 5 kg loss, and a 6 mmHg fall in systolic and 4.6 mmHg in diastolic blood pressure at 2 years with a 10 kg loss [38,39].
- **Diabetes** In those with impaired glucose tolerance, lifestyle treatments and modest weight loss can prevent or delay the onset of type 2 diabetes (58% reduction in diabetes incidence) [40]. In obese patients with type 2 diabetes, a 5 kg loss has been found to reduce HbA1c by 0.28% at 12 months [34,39].
- **Lipid profile** Modest weight loss is associated with reductions in low-density lipoprotein, total cholesterol and triglycerides and with increased levels of high-density lipoprotein [34,41].

Conclusion

If current predictions are correct, in 40 years' time the majority of UK adults will be obese. This will have overwhelming cost implications for health care and the wider economy. Given the profound effect of obesity on morbidity and mortality

and the substantial health benefits achievable with modest weight loss, delivering comprehensive lifestyle management, ideally at the pre-morbid stage of the condition, is critical.

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