Obesity: Cardiovascular disease: Cancer: Quality of life

The prevalence of obesity has more than doubled in the last decade, and continues to increase, giving rise to some obvious problems, and also some problems which are not so plain. The average weight of Scottish men is 72 kg at age 16–24 years, and 84 kg at 55–64 years, the corresponding values being 62 and 70 kg for women (Scottish Health Survey; Scottish Office, Department of Health, 1995). These cross-sectional data suggest that most individuals gain about 10 kg between the ages of 20 and 50 years (on average 0·3 kg/year) and this value is probably an underestimate as younger individuals are now much fatter than they were 30 years ago. However, the rate of weight gain is much greater amongst those individuals who become obese, and is extreme in some cases. It can be assumed that this weight gain is mainly adipose tissue, which stores about 29·3 MJ (7000 kcal)/kg. About 20–30 % of 18 year olds are already overweight (BMI > 25 kg/m²; Scottish Intercollegiate Guidelines Network, 1996), and many of them will increase over adult life to exceed a BMI of 30 kg/m², contributing to the 25 % of 55–64 year olds currently ‘obese’ in the UK. As the incidence of smoking falls, the rising level of obesity is likely to become a more dominant cause of CHD and other diseases, perhaps with different clinical characteristics from those previously seen.

What is obesity?

The word obesity (from the Latin ob-esum, meaning on account of having been eaten) is a lay term which means the same as fatness but with moderately abusive overtones. Obesity is a disease with International Classification of Disease code E66 (World Health Organization, 1997). In

Abbreviations: NIDDM, non-insulin-dependent diabetes mellitus.
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adults ‘obesity’ is now defined by international convention to indicate the state of having a BMI of < 30 kg/m² (or ‘grade 2 obesity’), while a BMI of > 25 kg/m² is designated ‘overweight’ (or ‘grade 1 obesity’) and a BMI of 18.5–25 kg/m² is ‘normal’, so by definition a BMI of > 25 kg/m² is abnormal (World Health Organization, 1995). These BMI cut-offs were initially based on life-expectancy data from life assurance companies, but they match the overweight-related risks for a range of morbidities. Since the BMI (kg/m²) is conceptually complex and inaccessible to most of the general public, an alternative measure of overweight and obesity and its health risks was required for health promotion purposes. Waist circumference is a more recently standardized alternative (measured between lowest rib and iliac crest, with the subject standing), and it relates both to total fatness and specifically to the intra-abdominal fat without the need to adjust for height (Han et al. 1997).

The disease itself is the process of excess fat accumulation, and obesity is a progressive systemic disease process, with multiple organ-specific manifestations. Obesity brings a host of debilitating symptoms (physical, psychological, social and medical consequences; Table 1), plus secondary metabolic effects, many of which conspire to cause IHD. Ultimately obesity can kill, through its contribution to diabetes, CHD, cancers and more directly via complications such as Pickwickian syndrome, sleep apnoea, venous thrombosis embolus, or cellulitis. Many of the pathological consequences are manifestations of the ‘metabolic syndrome’, which is characterized by both central fat distribution and excess total body fat. These two components are under separate genetic influences, but both are aggravated by inactivity and a high-fat diet.

**Obesity and cardiovascular disease**

A scan of the world literature would suggest that obesity is almost exclusively perceived by clinical scientists as a risk factor (or ‘surrogate’) for cardiovascular disease. Most individuals with cardiovascular disease are overweight or obese (82 % of people with cardiovascular disease in 1995 in Scotland; Scottish Office, Department of Health, 1995). To give some indication of the scale of influence of obesity on cardiovascular disease, the Scottish Health Survey (Scottish Office, Department of Health, 1995) found the prevalence of any cardiovascular disorder was 37 % of adults with BMI > 30 kg/m² and 21 % for those with a BMI of 25–30 kg/m², compared with 10 % for individuals with BMI < 25 kg/m². It is not possible to state from these epidemiological data which are the dominant mechanisms relating weight gain to cardiovascular disease. There are four aspects to obesity in cardiovascular disease (Table 2).

First, obesity has a direct effect, causing or contributing to cardiac pathology via accelerated atheroma and also increased thrombotic risks. Second, obesity causes individuals with diseased (or even relatively normal) hearts to develop cardiac symptoms, i.e. disease as it presents. Third, obesity can mimic cardiac symptoms of breathlessness, oedema and chest pain; 40 % of obese patients with angina do not have demonstrable coronary artery disease (Bahadori et al. 1996). Fourth, obesity can compound and exaggerate the effect of other risk factors.

The interaction of overweight and obesity with conventional major risk factors for cardiovascular disease is very striking, as demonstrated in 12-year follow-up data from the Boston Nurses Study (Manson et al. 1990). In the absence of a risk factor (non-insulin-dependent diabetes mellitus (NIDDM), smoking, hypertension, hypercholesterolaemia) there was a highly significant doubling of risk as BMI increased from 21 to > 29 kg/m² (Fig. 1). This huge effect, however, was dwarfed by the colossal rise in CHD risk when one of the risk factors was also present. There was a particularly dramatic increase in risk for smokers who were overweight (or the overweight who smoke), which argues for prioritizing smoking cessation above weight loss.

The interaction between smoking and obesity is complicated. Both factors now occur mainly in more deprived population groups, thus compounding the cardiovascular risk that arises from poor diet composition. Smoking tends to reduce appetite and to elevate metabolic rate by its thermogenic effects; so historically, therefore, smoking has been associated with thinness. Now, however, the marketing of tobacco, especially to girls, as a way of controlling weight

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**Table 1. Medical consequences of overweight and obesity: a systemic disease with organ-specific manifestations**

<table>
<thead>
<tr>
<th>Physical symptoms</th>
<th>Metabolic problems</th>
<th>Endocrine</th>
<th>Inflammatory</th>
<th>Psychological</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathlessness</td>
<td>Hypertension</td>
<td>Endocrine</td>
<td>Menorrhagia</td>
<td>Tiredness</td>
</tr>
<tr>
<td>Varicose veins</td>
<td>NIDDM</td>
<td></td>
<td>Oestrogen-dependent cancers (breast, endometrium, prostate)</td>
<td>Low self esteem</td>
</tr>
<tr>
<td>Back pain</td>
<td>Hyperlipidaemia</td>
<td></td>
<td></td>
<td>Self deception and distortion of thought</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Hypercoagulation</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Oedema, cellulitis</td>
<td>IH and stroke</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sweating</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Stress incontinence</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

**Table 2. Four categories of mechanisms relating weight gain to cardiovascular disease**

<table>
<thead>
<tr>
<th>Obesity causes cardiac pathology:</th>
<th>Atheroma</th>
<th>Thrombosis</th>
<th>LV hypertrophy</th>
<th>Arrhythmia</th>
</tr>
</thead>
<tbody>
<tr>
<td>Obesity provokes cardiac symptoms:</td>
<td>Breathlessness</td>
<td>Oedema</td>
<td>Angina</td>
<td>Smoking</td>
</tr>
<tr>
<td>Obesity mimics cardiac symptoms:</td>
<td></td>
<td></td>
<td>NIDDM</td>
<td>Hyperlipidaemia</td>
</tr>
<tr>
<td>Obesity compounds other risk factors:</td>
<td></td>
<td></td>
<td>Hypertension</td>
<td></td>
</tr>
</tbody>
</table>

NIDDM, non-insulin-dependent diabetes mellitus.
has resulted in smoking taking a new position as a form of weight control. A consequence is that overweight individuals commonly also smoke. Fear of weight gain is an important obstacle to smoking cessation. A recent survey in Glasgow found that 80% of current smokers wanted to stop, and 45% cited fear of weight gain as a barrier (J Halim, MEJ Lean and S Morris, unpublished results). The interaction between smoking and being overweight, in terms of cardiac risk, is particularly frightening (Manson et al. 1990). At a BMI of > 29 kg/m² there is a doubling of coronary risk amongst non-smokers. The risk for overweight smokers rises more steeply with BMI, to reach a 12-fold risk at BMI > 29 kg/m².

Obesity is undoubtedly an important primary cause of IHD and stroke, operating through the effects of weight gain on hypertension (Dyer et al. 1994), hyperlipidaemia (Bjorntorp, 1990; Denke et al. 1993, 1994), NIDDM (Colditz et al. 1995) and increased blood coagulability (Hankey et al. 1997). These metabolic effects relate specifically to the intra-abdominal fat accumulation, so although elevated BMI brings increased cardiovascular risk, at least in younger individuals, more apple-shaped (‘android’) fat distribution is a stronger risk factor. Central fat distribution confers risk even in thin people, but it compounds the problems of being overweight. Thus, a large waist circumference is an important indicator of cardiovascular disease risk. The term ‘central obesity’ is a very confused one. In the past ‘waist : hip ratio’ was used and this value was assumed to be an indicator of fat distribution. However, a low hip circumference (reflecting low muscle mass at least in thinner individuals) is also a risk factor for NIDDM (Seidell et al. 1997; Han et al. 1998), and waist circumference alone is a better indicator of both total fatness and central fat accumulation (Lean et al. 1996; Han et al. 1997). The waist : hip value is a more complicated and rather contrived term, without any biological basis. There is an advantage for practical applications in health promotion in using the simple waist circumference (most men at least already know their waist) and the waist circumference is virtually unaffected by height, so no correction is needed for health promotion purposes (Han et al. 1997). ‘Action levels’ for waist circumference, initially developed for Scottish Intercollegiate Guidelines Network (1996), have now been adopted by National Institutes of Health, National Heart, Lung and Blood Institute (1998; Table 3).

The prevalences of CHD and stroke, and of risk factors for CHD are shown in Table 4, according to standard cut-offs of BMI and waist circumference. For all these health problems there is a progressive rise with BMI category. The relative risks, with reference to BMI < 25 kg/m² are 3–9. The effect of weight gain on NIDDM is dramatic; individuals with BMI < 21 kg/m² virtually never get NIDDM, but the relative risk (with reference to BMI < 21 kg/m²) over 14 years in middle-aged women is 8 at a BMI of 25 kg/m², 28 at a BMI of 30 kg/m², and 93 at a BMI of > 35 kg/m². A weight gain of as little as 8–10 kg doubles the risk of NIDDM, while age-adjusted relative risk is 15 with a weight gain > 20 kg (Colditz et al. 1995). Risks of death from CHD (and total mortality) are clearly related to quite modest weight changes in non-smoking women, with clearly increased mortality in those who gained more than 10 kg during 16 years follow up (Manson et al. 1995). There is a consistent trend to reduced mortality in those individuals who lose weight. The evidence is mounting gradually that

![Fig. 1. Relative risks of non-fatal myocardial infarction and fatal CHD for risk factors (a) smoking, (b) hypertension, (c) hypercholesterolaemia and (d) non-insulin-dependent diabetes mellitus, with increasing BMI in the Boston Nurses Study. (From Manson et al. 1990.)](https://doi.org/10.1017/S0029665100000379)
intentional weight loss reduces the mortality towards that of the general population (Lean et al. 1990; Williamson et al. 1995, 1999).

**Cardiac symptoms of obesity**

In general, symptoms of obesity are uncommon under age 40 years. However, several symptoms of CHD are aggravated by obesity and may be precipitated in patients who experience a rapid weight gain. These symptoms include angina and heart failure, and both are complicated. First, 40 % of obese patients with angina do not have demonstrable coronary artery disease (Bahadori et al. 1996), i.e. angina may be a direct symptom of obesity, with no need to blame ‘cardiac syndrome X’. Second, the major symptoms of congestive heart failure are commonly mimicked or compounded by exertional and nocturnal breathlessness as a direct effect of obesity, and leg oedema secondary to venous and lymphatic obstruction at the inguinal canal in obesity. Obesity also aggravates or precipitates heart failure through an increase in left ventricular mass. This, and potentially fatal arrhythmias, are particularly found in patients with obesity-related sleep apnoea syndrome.

**Obesity and cancer**

There are well established links between obesity and elevated risk of several major cancers (Department of Health, 1998). Several of these (uterus, prostate, breast) are endocrine cancers, and the process appears to be mediated by elevation of free oestrogen, partly through altered adipose tissue aromatase activity, and partly through the suppression of sex hormone-binding globulin in obesity. In breast cancer, excess body fat probably delays detection of small tumours, with greater likelihood of metastases at the time of diagnosis.

There may be additional common components related to a high-fat diet. Colon cancer is also substantially more frequent in the obese. Here the mechanisms are more obscure, but physical inactivity appears to play a specific role and a high-fat diet may have specific effects. High fruit and vegetable consumption protect against colonic cancer, but it is difficult to be sure if overweight people eat less fruits and vegetables than others.

**Obesity and the whole patient**

Obese patients may present with one symptom, or a related set of problems that impinge on an aspect of life, but it is important to recognize that the disease produces pathology in many organs and systems concurrently. The list in Table 1 is long, but most medical consequences are probably directly, causally and reversibly related to weight gain; thus, they often develop together, gradually over the same time period. This development of secondary consequences of obesity is always slow, over many years, and the insidious debilitation may go unrecognized or ignored. From the clinical aspect, therefore, it is important to undertake a full evaluation of patients, and to enquire about other systems which may still be affected, but sub-clinically. In that way it may be possible to check the progression of a range of problems.

There are no detailed longitudinal surveys available to examine the full complexity of obesity symptomatology. Virtually all the symptoms and consequence of obesity are multifactorial and also age-related. Thus, establishing thresholds is difficult, and particularly so because body fat and BMI commonly increase with age. As a very broad generalization, direct specific ‘medical’ symptoms of obesity are rare below the age of 40 years, and then tend to appear in clusters. It is apparent, however, that pathological consequences start at much younger ages; therefore, effects such as elevated blood pressure, hyperlipidaemias or insulin resistance can be detected in young individuals. Endocrine consequences, such as infertility and polycystic ovarian syndrome present in obese young women. Hazards of obesity in pregnancy are well documented, including metabolic disturbances such as gestational diabetes, or a striking increase in pre-eclampsia in overweight women (Sattar et al. 1996), and increased likelihood of mechanical difficulties during labour. The commonest major symptom of obesity is probably breathlessness, often attributed to asthma and treated ineffectively with bronchodilators. The progression to sleep apnoea syndrome and Pickwickian syndrome is one of the least understood hazards. It is most common with

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**Table 3. Measures of health risk for use in health promotion**

<table>
<thead>
<tr>
<th>Waist circumference* (cm)</th>
<th>BMI (kg/m²)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Healthy or normal:</td>
<td></td>
</tr>
<tr>
<td>M:F</td>
<td>&lt;94: 18·5–25</td>
</tr>
<tr>
<td>Increasing risks:</td>
<td></td>
</tr>
<tr>
<td>F:F</td>
<td>80–88:</td>
</tr>
<tr>
<td>High risks:</td>
<td></td>
</tr>
<tr>
<td>M:F</td>
<td>&gt;102: &gt;30</td>
</tr>
<tr>
<td>F:F</td>
<td>&gt;88:</td>
</tr>
</tbody>
</table>

*Waist is measured midway between the lowest rib and the iliac crest: BMI measurement requires calibrated scales and stadiometer.

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**Table 4. Prevalence (%) of coronary risk factors and of IHD or stroke in adults**

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Normal wt &lt; 25</th>
<th>25–30</th>
<th>&gt; 30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol</td>
<td>6·5 mmol/l:</td>
<td>9·4</td>
<td>17·2</td>
</tr>
<tr>
<td>HDL</td>
<td>≤0·9 mmol/l:</td>
<td>13·5</td>
<td>24·5</td>
</tr>
<tr>
<td>NIDDM:</td>
<td>M:F</td>
<td>0·6</td>
<td>1·8</td>
</tr>
<tr>
<td>IHD or stroke:</td>
<td>M:F</td>
<td>1·9</td>
<td>5·3</td>
</tr>
</tbody>
</table>

NIDDM, non-insulin-dependent diabetes mellitus.
central fat distribution and has mechanical as well as central (medullary) components. An early warning system is snoring, and this condition can cause death, both through respiratory failure and through road accidents which result from falling asleep while driving.

Operative and post-operative surgical complications are more frequent in obese subjects of all ages, particularly chest infections, wound infections and venous thrombosis which seems resistant to prophylaxis. With increasing age, obesity is the strongest predictor for venous thrombosis (Goldhaber et al. 1983; Lowe et al. 1999). An interesting new finding in this context is the increased adipose tissue synthesis of plasminogen activator inhibitor in the obese (Bastard & Pieroni, 1999).

The highly-publicised relationship between obesity and cerebrovascular and cardiovascular diseases (stroke, myocardial infarction) is very real, but related to age in a complicated way. Prospective studies hitherto have not been able to delineate clearly whether relative risks of coronary risk factors change with age. Absolute risk increases dramatically, but there is some suggestion that relative risk is little changed with age (Lean et al. 1998). On the other hand, the evidence from other large epidemiological studies suggests that the impact of obesity on BMI or CHD declines with age. The prospect of accelerated CHD is a major worry for young men, but the added risk from obesity appears to be lost by the age of years 50 years (Royal College of Physicians, 1983). For older individuals, therefore, the burden of ill health from obesity is dominated by musculo-skeletal, metabolic and socio-psychological problems, and CHD is less important as a result of obesity.

Psychological disturbances are common amongst obese and overweight individuals, and even in normal-weight individuals who are ‘restrained eaters’ in order to control an underlying genetic predisposition to weight gain. It is considered that the disturbances of thought, and beliefs about body image and the systematic under-reporting of food intake are all secondary, rather than driving the hyper-phagia of obesity. The declared depression of obesity is a very real problem, but with rather different characteristics from the classical ‘endogenous depression’. First, it is associated with overeating, often in a planned but surreptitious bingeing pattern, whereas endogenous depression is characterized by weight loss. Second, it appears to resolve after major weight loss (Sjostrom et al. 1999). Binge eating in obesity is frequent, but little understood. It is itself probably a secondary problem, but may in fact underlie at least the maintenance of obesity. Regulating eating patterns by behavioural (or surgical) means can reverse the problem.

**Conclusion**

Over history, there have been times when obesity has been treated as a mark of success or stature in society, as an object of humour, and as a sensitive personal problem. Somewhat grudgingly, it seems, obesity has become accepted as a risk factor for CHD, with a major influence on other risk factors. However, just listening to patients makes it clear that obesity is a major disease, the debilitating consequences of a relentless disease process. It certainly does cause heart disease, but this disease is only one of many consequences, and a rather distant one. The reality of life with obesity is steady accumulation of problems affecting every aspect of life and almost every system of the body.

Various attempts to estimate the economic costs of obesity have produced misleading information, because data relating BMI category and age to costable disease consequences are very incomplete, and some relative risk values are very misleading. The value calculated by Seidell (1995) of 4% of the total health care budget attributable to BMI > 25 kg/m² (i.e. above the costs if everyone had BMI < 25 kg/m²) is enormous, but possibly conservative. Also, as the prevalence of overweight and obesity rise, not only will these costs rise, but the whole profile of some clinical practice will change. For example, as smoking declines, heart disease will fall, but the pathological and clinical pattern of CHD will also change as obesity becomes responsible for a greater proportion of CHD.

The conventional BMI and waist cutoffs (Table 3) give good indicators of multiple health risks, independent of age. They can form the basis for epidemiology, health promotion and clinical screening, although interpretations will vary to some extent between racial and ethnic groups.

**References**


Han TS, Seidell JC, Currall JEP, Morrison CE, Deurenberg P, Lean MEJ (1997) The influences of height and age on waist

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Seidell JC, Han TS, Feskens EJM & Lean MEJ (1997) Narrow hips and broad waist circumferences independently contribute to increased risk of NIDDM. *Journal of Internal Medicine* 42, 401–406.


