Obesity: Cardiovascular disease: Cancer: Quality of life

The rapidly rising prevalence of obesity, worldwide, has prompted re-evaluations of the definitions and diagnostic criteria, and of the extent of the burden it contributes to health care services. Although categorized arbitrarily for epidemiological purposes according to BMI > 25 kg/m² (‘overweight’) and BMI > 30 kg/m² (‘obese’), the disease itself (ICD code E.66) is the process of excess fat accumulation. It leads to multiple organ-specific pathological consequences, particularly if there is a tendency to intra-abdominal fat accumulation. The simplest field method to identify obesity and risk of medical problems is the waist circumference, and this method has found a special role in health promotion. Risks begin with waist > 80 cm (women) or > 94 cm (men). As a broad generalization, obesity produces few symptoms below the age of 40 years, but then several symptoms often develop; tiredness, breathlessness, back pain, arthritis, sweatiness, poor sleeping, depression and menstrual disorders all being common. The symptoms are often attributed to diseases in other body systems. Metabolic diseases like diabetes, hyperlipidaemia and, hypertension develop later, but the mean BMI at diagnosis of diabetes is 28 kg/m². Ultimately, obesity increases the likelihood of myocardial infarction, stroke and several major cancers, but its biggest impact on health, especially in the elderly, is probably the multiplicity of effects on other body systems. The greatest challenge for public health is to develop effective preventive measures, recognizing that BMI > 25 kg/m² before the age of 20 years is a very strong predictor of obesity and ill health in adulthood.

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 disturbances</th>
</tr>
</thead>
<tbody>
<tr>
<td>Breathlessness</td>
<td>Hypertension</td>
<td>Hirsutism</td>
</tr>
<tr>
<td>Varicose veins</td>
<td>NIDDM</td>
<td>Menorrhagia</td>
</tr>
<tr>
<td>Back pain</td>
<td>Hyperlipidaemia</td>
<td>Oestrogen-dependent cancers</td>
</tr>
<tr>
<td>Arthritis</td>
<td>Hypercoagulation</td>
<td>(breast, endometrium, prostate)</td>
</tr>
<tr>
<td>Oedema, cellulitis</td>
<td>IHD and stroke</td>
<td></td>
</tr>
<tr>
<td>Sweating</td>
<td>Endocrine</td>
<td></td>
</tr>
<tr>
<td>Stress incontinence</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Anaesthetic and surgical hazards</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Sleep apnoea</td>
<td>Glikogenorrhea, infertility</td>
<td></td>
</tr>
<tr>
<td>Wound dehiscence</td>
<td>Menorrhagia</td>
<td></td>
</tr>
<tr>
<td>Hernia</td>
<td>Oestrogen-dependent cancers</td>
<td></td>
</tr>
<tr>
<td>Chest infections</td>
<td>(breast, endometrium, prostate)</td>
<td></td>
</tr>
<tr>
<td>Social</td>
<td>Psychological</td>
<td></td>
</tr>
<tr>
<td>Isolation</td>
<td>Tiredness</td>
<td></td>
</tr>
<tr>
<td>Agoraphobia</td>
<td>Low self esteem</td>
<td></td>
</tr>
<tr>
<td>Unemployment</td>
<td>Self deception and distortion of thought</td>
<td></td>
</tr>
<tr>
<td>Family, marital stress</td>
<td>Depression</td>
<td></td>
</tr>
</tbody>
</table>

NIDDM, non-insulin-dependent diabetes.

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

<table>
<thead>
<tr>
<th>Obesity causes cardiac pathology:</th>
<th>Atheroma</th>
</tr>
</thead>
<tbody>
<tr>
<td>Thrombosis</td>
<td>LV hypertrophy</td>
</tr>
<tr>
<td>Arrhythmia</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Obesity provokes cardiac symptoms:</th>
<th>Breathlessness</th>
<th>Oedema</th>
</tr>
</thead>
<tbody>
<tr>
<td>Angina</td>
<td></td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Obesity mimics cardiac symptoms:</th>
<th>Angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>Smoking</td>
<td></td>
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</tbody>
</table>

<table>
<thead>
<tr>
<th>Obesity compounds other risk factors:</th>
<th>Angina</th>
</tr>
</thead>
<tbody>
<tr>
<td>NIDDM</td>
<td></td>
</tr>
<tr>
<td>Hyperlipidaemia</td>
<td></td>
</tr>
<tr>
<td>Hypertension</td>
<td></td>
</tr>
</tbody>
</table>

LV, left ventricular; 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.

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
dermatologic 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
depletion 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
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 meta-
Table 3. Measures of health risk for use in health promotion
(Scottish Intercollegiate Guidelines Network, 1996; National
Institutes of Health, National Heart, Lung and Blood Institute, 1998)

<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 &lt; 94</td>
<td>18.5–25</td>
</tr>
<tr>
<td>F &lt; 80</td>
<td></td>
</tr>
<tr>
<td>Increasing risks:</td>
<td></td>
</tr>
<tr>
<td>M 94–102</td>
<td>25–30</td>
</tr>
<tr>
<td>F 80–88</td>
<td></td>
</tr>
<tr>
<td>High risks:</td>
<td></td>
</tr>
<tr>
<td>M &gt; 102</td>
<td>&gt; 30</td>
</tr>
<tr>
<td>F &gt; 88</td>
<td></td>
</tr>
</tbody>
</table>

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

Table 4. Prevalence (%) of coronary risk factors and of IHD or stroke
in adults (Scottish Office, Department of Health, 1995; Lean et al.
1999)

<table>
<thead>
<tr>
<th>BMI (kg/m²)</th>
<th>Normal wt &lt; 25</th>
<th>25–30</th>
<th>&gt; 30</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cholesterol ≥ 6.5 mmol/l:</td>
<td>M 9.4</td>
<td>17.2</td>
<td>24.6</td>
</tr>
<tr>
<td></td>
<td>F 9.4</td>
<td>19.1</td>
<td>19.8</td>
</tr>
<tr>
<td>HDL ≤ 0.9 mmol/l:</td>
<td>M 13.5</td>
<td>24.5</td>
<td>39.7</td>
</tr>
<tr>
<td></td>
<td>F 3.4</td>
<td>5.8</td>
<td>11.8</td>
</tr>
<tr>
<td>NIDDM:</td>
<td>M 0.6</td>
<td>1.8</td>
<td>4.7</td>
</tr>
<tr>
<td></td>
<td>F 0.4</td>
<td>1.0</td>
<td>3.8</td>
</tr>
<tr>
<td>IHD or stroke:</td>
<td>M 1.9</td>
<td>5.3</td>
<td>9.7</td>
</tr>
<tr>
<td></td>
<td>F 1.7</td>
<td>3.1</td>
<td>7.9</td>
</tr>
</tbody>
</table>

NIDDM, non-insulin-dependent diabetes mellitus.
appears to resolve after major weight loss (Sjostrom & Pieroni, 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 hyperphagia of obesity. The declared depression of the food intake are all secondary, rather than driving the hyperphagia of obesity. The declared depression of the 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 hyperphagia of obesity. The declared depression of the.

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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