Obesity as a disease

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Obesity is associated with the development of some of the most prevalent diseases of modern society. The greatest risk is for diabetes mellitus where a body mass index above 35 kg/m² increases the risk by 93-fold in women and by 42-fold in men. The risk of coronary heart disease is increased 86% by a 20% rise in weight in males, whereas in obese women the risk is increased 3.6-fold. Elevation of blood pressure, hyperlipidaemia and altered haemostatic factors are implicated in this high risk from coronary heart disease. Gallbladder disease is increased 2.7-fold with an enhanced cancer risk especially for colorectal cancer in males and cancer of the endometrium and biliary passages in females. Endocrine changes are associated with metabolic diseases and infertility, and respiratory problems result in sleep apnoea, hypoventilation, arrhythmias and eventual cardiac failure. Obesity is not a social stigma but an actual disease with a major genetic component to its aetiology and a financial cost estimated at $69 billion for the USA alone.

Obesity is not just a health risk but a disease. Estimates of the genetic contribution to weight gain in susceptible families range from 25–40% with a greater heritability for abdominal fat distribution of 50%\(^2\). Obviously there is a major environmental effect but this genetic susceptibility alone removes this condition from a social stigma to the disease category. The many associated conditions, such as diabetes, which are directly attributable to excessive weight make obesity a major influence on disease progression and, with its high prevalence, places obesity as the major nutritional disease of the Westernised world. This chapter describes those diseases associated with obesity by reference to mortality and morbidity and also by emphasising the advantage to health and co-morbid risk factors of weight loss.

Mortality

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There is now extensive evidence that links excessive body weight with overall mortality. The overall relationship between mortality and body mass index (BMI) adjusted for age shows a J shaped curve (Fig. 1) with an acceleration of the mortality risk above a BMI of 30\(^3\). This is well illustrated by the work of Manson et al.\(^4\) who examined the association...
between BMI and both overall mortality and mortality from specific diseases in a cohort of 115,195 women from the USA enrolled in their prospective Nurses' Health Study. These women were 30–55 years of age and healthy when enrolled in 1976. During 16 years of follow-up, there were 4726 deaths, some 881 from cardiovascular causes, 2586 from cancer and 1259 from other aetiologies. In the analysis of those women who had never smoked, the increased relative risk of death is not only seen in those with frank obesity but the risk rises with modest gains in weight. For instance, the increased relative risk of death was 1.3 in those with a BMI 25.0–26.9, 1.6 in those with a BMI 27.0–28.9 and was doubled (2.1) for those with a BMI of 29.0–31.9. Among women with a BMI above 32 who had never smoked, the risk of death from cardiovascular disease was 4.1 and from cancer was 2.1. In terms of attributable risk, some 53% of all deaths in this study among women with a BMI of 29 or greater could be attributed directly to their obesity.

Some have described the relationship between mortality and BMI as a J shaped curve with a rising mortality in the thinnest individuals. This apparent excess risk associated with leanness was found to be artifactual in the above nurses' study and was eliminated after accounting for smoking and subclinical disease. With these exclusions, lean women (BMI <19) had the lowest mortality. However, a weight gain of 10 kg or more from the age of 18 years was associated with an increased mortality in middle age. In contrast, those women who lost weight or gained less than 10 kg did not have a significant change in mortality. Also the BMI at age 18 years predicted overall and cardiovascular mortality in middle age.

Although coronary heart disease is a major cause of weight-related death, the obese often develop other conditions which further predispose to their mortality. This relative mortality is highest for diabetes mellitus and next for digestive diseases including cancer. Table 1 shows this relative mortality risk of obesity reported in a study of 750,000 men and women by Lew and Garfinkel. In this study, the mortality for cancer was highest for colorectal cancer in males (1.73 in males, 1.22 in females), whereas in females this was in endometrial cancer followed by cancer of the gallbladder and biliary passages. In addition, those with substantial obesity had an increased risk of cervix, breast and ovarian cancers.

Smoking appreciably elevates mortality (Fig. 1) such that those who smoke 20 or more cigarettes per day have double the risk of non-smokers throughout the weight range and this is especially apparent in males. Cessation of smoking often results in weight gain; in one study this averaged 2.8 kg in males and 3.8 kg in females. Weight gain results from a decrease in energy expenditure (each cigarette utilises 8 kcal by stimulation of the sympathetic system) and also by enhanced energy.
intake. The priority should always be to advise cessation of smoking for the health risk is greater. A weight gain of as much as 11–13 kg would be required to equate to the same mortality risk as a non-smoker. In one study this amount of weight was gained by 9.8% of males and 13.4% of females on cessation of smoking indicating that, although the majority will benefit, there is also a need for parallel dietary advice.

Recent evidence indicates that a weight loss of more than 9 kg in women is associated with a 25% reduction in all causes (diabetic, cardiovascular and cancer) of mortality. If the obese person has already developed a weight related disease, then intentional weight loss of any amount has been shown to reduce mortality by 20%. This is most marked for cancer (with a 40–50% reduction) and for diabetes (with a

Table 1 Mortality risk in obesity. Compares the risk of those weighing 140% or more above an ideal weight (100%) with those weighing 90–109% of ideal

<table>
<thead>
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<th>Males</th>
<th>Females</th>
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<tbody>
<tr>
<td>Diabetes mellitus</td>
<td>5.19</td>
<td>7.90</td>
</tr>
<tr>
<td>Digestive diseases</td>
<td>3.99</td>
<td>2.29</td>
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<tr>
<td>Coronary heart disease</td>
<td>1.85</td>
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<tr>
<td>Cerebral vascular disease</td>
<td>2.27</td>
<td>1.52</td>
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<tr>
<td>Cancer: all sites</td>
<td>1.33</td>
<td>1.55</td>
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<tr>
<td>Cancer: colorectal</td>
<td>1.73</td>
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<td>Cancer: prostate</td>
<td>1.29</td>
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<tr>
<td>Cancer: gallbladder/biliary</td>
<td>3.58</td>
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<tr>
<td>Cancer: endometrium</td>
<td>5.42</td>
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<tr>
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<td>2.39</td>
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<td>Cancer: ovary</td>
<td>1.63</td>
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30–40% fall in mortality). It is important to emphasise ‘intentional’ weight loss, for much confusion has arisen in the past from the inclusion of non-intentional, i.e. disease driven, weight loss which pre-empts death from many conditions.

**Morbidity**

Excess weight is associated with a multiplicity of problems as outlined in Table 2, hence the categorisation in many countries of obesity as a distinct disease. Equally, a 10 kg weight loss can confer significant health benefits (Table 3).

**Diabetes mellitus**

Most non insulin dependent diabetic patients are overweight, about 75% in most studies. Colditz et al. have reported on the development of diabetes in females in a 14 year prospective study of 114,281 nurses aged 30–55 years who did not have this condition or coronary heart disease, stroke or cancer at the outset. After adjustment for age, BMI was the dominant predictor of the risk for diabetes. In females the risk rises...
Table 3  Benefits of a 10 kg weight loss

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| **Mortality** | 20–25% fall in total mortality  
30–40% fall in diabetes related deaths  
40–50% fall in obesity related cancer deaths |
| **Blood pressure** | Fall of 10 mmHg systolic pressure  
Fall of 20 mmHg diastolic pressure |
| **Angina** | Reduced symptoms by 91%  
33% increase in exercise tolerance |
| **Lipids** | Fall by 10% in total cholesterol  
Fall by 15% in LDL cholesterol  
Fall by 30% in triglycerides  
Increase by 8% in HDL cholesterol |
| **Diabetes** | Reduces risk of developing diabetes by > 50%  
Fall of 30–50% in fasting blood glucose  
Fall of 15% in HbA1c |

above a BMI of 22 with a 5-fold increased risk at a BMI of 25, 28-fold risk at BMI of 30 and a 93-fold higher risk above a BMI of 35. Compared to those of stable weight, a gain of 8–10.9 kg increases the risk of diabetes by 2.7-fold. In contrast, those women who lose more than 5 kg, reduce their risk of developing diabetes by 50% or more. These results were also independent of any family history of diabetes.

The situation in males was reported by Chan *et al.* in a study of 51,529 men aged 40–75 years in 1986 and subsequently followed up for 5 years. Increased risk was seen for all BMI levels of 24 or above. Even men with a slight excess weight were more likely to develop diabetes than those with a BMI less than 23 kg/m². The risk adjusted for age was increased 2.2-fold in those with BMI 25–26.9, 6.7 in those with BMI 29–30.9 and 42 in those with BMI of 35 or more. The BMI at age 21 years and the absolute weight gain during adulthood were independent risk factors for diabetes development. A BMI of 27 at age 21 years increased the risk 6.4-fold whereas a gain in weight of 9 kg from the age of 21 years further raised the risk 3.5-fold. Fat distribution measured by the waist circumference was also independently associated with diabetes development. A waist above 40 inches (100 cm) alone increased the risk by 3.5-fold even after control for BMI.

These results are in keeping with a major study in Oslo by Westlund and Nicolayson on the 10 year follow up of 3751 men aged 40–49 years at the outset. The percentage developing diabetes was 0.6% in normal weight men but 23.4% in those 25% or more overweight. Weight loss improves this risk in males and females. In the nurses’ study reported above, a 5 kg weight loss reduced the risk of developing diabetes by 50% or more. Weight loss also improves mortality in those with established diabetes. A 9 kg weight loss reduces diabetes related
mortality by 30–40%, whereas a 5% weight loss reduces HbA1C by 7% and decreases fasting blood glucose by 15%. A loss of 10–20% in weight in non insulin dependent diabetic patients can normalise metabolic control and possibly life expectancy.

Cardiovascular disease, blood pressure, lipids and rheology

A number of cardiovascular risk factors are influenced by overweight including hypertension, impaired glycaemic control, dyslipidaemia and haemostatic and rheological factors. Until recently it was thought that only severe degrees of excess weight increased the risk of coronary heart disease but recent evidence shows a clear association with modest weight gain. In the nurses' study, Willett et al. controlled for age, smoking, menopausal status, post menopausal hormone use and family history. They found that the risk of coronary heart disease was increased 2-fold in those women of BMI 25–28.9 and 3.6 for a BMI of 29 or more. Weight gain from age 18 years increased the risk 1.6-fold for a 8–10.9 kg gain and 1.9-fold for a 11–19 kg gain. In males, a 10% increase in weight will increase the risk of coronary heart disease by 38% whereas a 20% weight rise corresponds with a 86% increased risk.

Blood pressure is increased by 6 mm systolic and 4 mm diastolic for a 10% gain in body fat with those genetically more susceptible showing the greater effect. Reisen et al. have demonstrated that a weight loss of 11 kg produced a 20% decrease in both systolic and diastolic pressure in hypertensive patients even when the sodium intake was kept constant. It would appear that, as a general rule, blood pressure is reduced by 1 mm-systolic and 2 mm diastolic for each 1% reduction in body weight.

The most characteristic lipid disorder in obesity is elevated total cholesterol and triglycerides, high LDL-cholesterol and low HDL-cholesterol. A meta-analysis by Datillo and Kris-Etherton of some 70 published studies and other work reviewed, has indicated that for every 1 kg of weight lost, there is a corresponding reduction by about 1% in total cholesterol and LDL, a rise by 1% in HDL and a reduction by 3% of triglycerides.

A number of haemostatic factors are associated with weight gain, particularly factors VII and X which may relate to thrombosis and the risk of fatal myocardial infarction. In a study by Ernst and Matrai using a 4.2 MJ diet, a 15% weight loss was associated with a decrease by 27% in blood viscosity, by 20% in red cell aggregation and by 5.5% in haematocrit; plasma fibrinogen remained unaffected. Hankey et al. have shown that a 5 kg weight loss reduced both factor VII coagulant
activity and red cell aggregability by 10%, whereas a weight loss of 5% can improve plasma activator inhibitor by 42%.

Such data on blood pressure, lipids and haemostatic factors most likely account for the reduction in morbidity and mortality from cardiovascular diseases with modest weight loss. In one study by Ornish et al.\textsuperscript{19} a 10 kg weight loss over 1 year reduced symptoms of angina by 91% with a 33% increase in exercise tolerance. The St Thomas Atherosclerosis Regression Study has shown the benefit of a reduced fat intake with or without cholestyramine on atheroma progression\textsuperscript{20}. The luminal diameter widened in 38% on a reduced fat intake diet, in 33% in those on diet and cholestyramine but in only 4% in the control group. The corresponding figures for coronary heart disease progression were 15% on reduced fat diet, 12% on diet and cholestyramine but 46% in the controls. An important feature of this trial was that weight loss was part of therapy in those with a BMI above 25. Ornish et al.\textsuperscript{19} also reported a 8.8% reduction in atheroma on polyvalent therapy including weight loss. These studies suggest that weight loss might reduce atheroma and may account for the 9% reduction in cardiovascular disease mortality reported with weight loss in the study of 43,457 women by Williamson et al.\textsuperscript{7}.

Obesity causes left ventricular hypertrophy which can co-exist with the cardiac changes associated with essential hypertension\textsuperscript{21}. In early stages of hypertension, cardiac output and peripheral resistance are both elevated, increasing blood pressure. Further increases in peripheral resistance caused by excess smooth muscle contraction and a contracted intravascular volume result in a progressive blood pressure rise with the result that the heart compensates for the increased afterload by left ventricular myocyte thickening and lengthening in a \textit{concentric} manner. In obesity, the increased lean mass, fat mass, body surface area and metabolic rate are associated with increased oxygen consumption resulting in an increased cardiac output by a rise in stroke volume. As total blood and plasma volume are also expanded, there is an increase in preload with an increased left ventricular end-diastolic volume resulting in left ventricular chamber dilatation. Cardiovascular reserve is preserved by compensatory hypertrophy of the left ventricle in an \textit{eccentric} manner so that the ratio between wall thickness and the chamber cavity radius is preserved (unlike hypertension where it is increased). If the obese become hypertensive then the resultant increased afterload can result in cardiac dilatation and hypertrophy and clinical cardiac failure. Left ventricular hypertrophy can be anticipated in more than 50% of patients who are more than 50% overweight\textsuperscript{22}.

\textit{Cerebrovascular disease} is common in the obese. In the Whitehall study involving 17,753 men aged 40–64 years, the risk associated with obesity for death from a stroke was more apparent in younger subjects.
Obesity and nonsmokers\textsuperscript{23}. In men aged 40–54 years, the most obese quintile had a mortality ratio some 2-fold higher than the thinnest. However, in men aged 55–64 years this ratio was only 1.2. The increased risk was more apparent in nonsmokers where the age adjusted ratio was 2.6. The Chicago stroke study of a biracial cohort aged 65–74 years did not show an association between body weight alone and stroke when potential confounders were controlled for in the analysis such as black race, female gender, age 70+, hypertension and diabetes all of which were independently associated with risk of stroke\textsuperscript{24}. Shinton \textit{et al.}\textsuperscript{25} have compared 125 men and women who had a first stroke and aged 35–74 years with 198 age and sex matched controls. They found an association between highest quartile for BMI and stroke with an odds ratio after multiple risk factor adjustment of 2.25. This lifelong pattern of risk appeared to be established early for the risk factor adjusted odds ratio for the BMI at age 21 years was 2.13.

**Digestive diseases**

\textit{Gallbladder disease} is the most common form of digestive disease in obese individuals. Rimm \textit{et al.}\textsuperscript{26}, in a study of 73,532 obese women in USA and Canada, reported a 2.7-fold increase in the prevalence of gallbladder disease. There is a progressive linear increased risk of gallstones from a BMI of 20 upwards which is twice as high in women as in men which further increases with age, and the number of pregnancies\textsuperscript{27}. Weight and age appear additive with obesity being 6-fold more important than age\textsuperscript{28}. Weight loss may actually exacerbate gallbladder disease. In the Boston nurses’ study, women who lost 4–10 kg had a 44% increased risk for detectable gallstone disease, whereas weight loss above 10 kg increased this risk to 94\textsuperscript{29}. The reason for this may be associated with the rise in the body pool of circulating cholesterol as adipose tissue stores are mobilised and the increase in rate at which cholesterol is excreted in the bile as obesity develops. The development of gallstones depends on the precipitation of cholesterol from a supersaturated bile. Dieting using inadequate dietary fibre further decreases the solubilisation of excreted cholesterol increasing gallstone formation.

\textit{Liver abnormalities} have been described in the obese mainly due to fatty infiltration. However, in the morbidly obese with BMI greater than 40, one study\textsuperscript{30} found only 2\% had normal livers, 56\% showed fatty infiltration alone, whereas 42\% had fatty infiltration associated with fibrosis or cirrhosis. In the American Cancer Society study\textsuperscript{5} of 750,000 men and women in the USA followed for 12 years, colorectal cancer was
the principal site of excess cancer mortality in obese males (relative rates: 1.73 in males, 1.22 in females), whereas in obese females the highest prevalence was from cancer of the gallbladder and biliary passages (relative mortality of 3.6). The relative mortality rates of cancer of the stomach and pancreas were higher in obese males (1.88 and 1.62, respectively) than in females where the mortality rate for stomach cancer (1.03) was reported similar to the non-obese and that for pancreatic cancer (0.61) significantly lower. The relative risk of endometrial cancer is more than doubled in women aged 60–69 years with a BMI of 25–29 kg/m\(^2\) and is increased 5.4-fold in those with significant obesity\(^5,31\).

**Breast cancer**

The relationship between breast cancer and obesity is not clear\(^32\). In the American Cancer Society study, the mortality ratio for breast cancer was not significantly raised for those 120–139% above ideal body weight (IBW = 100%), but was 1.63 for those greater than 140% IBW\(^5\). However, age has a significant impact on the risk of developing breast cancer. Premenopausal obese women have the same risk as lean women but in the postmenopause, obese women exhibit a higher risk\(^33\). Abdominal obesity and a positive family history increase this risk. It has been conjectured that this association with obesity, and especially central adiposity, is associated with enhanced conversion of androgens to oestrogen in the fat mass and a reduced sex-hormone binding globulin (SHBG) which adds to an increase in free oestradiol levels.

**Lung cancer**

This is one cancer which the obese are less prone to develop. The adjusted odds ratio for non smokers with BMI <22 was 2.9 in women and 0.9 in males compared to those with BMI <28. The odds ratio for smokers in the group with BMI <22 was 2.0 in both sexes\(^34\).

**Arthritis and bone mass**

It is not easy to find objective evidence for the improvement of arthritis with weight loss especially in the elderly. Whereas there is evidence that obesity is associated with osteoarthritis of the hip and knee, extensive literature does not indicate that weight loss in modest obesity had any measurable clinical benefit on this condition. In contrast McGoey et al.\(^35\).
have reported that in the morbidly obese there is relief of pain in the lower back, ankles and feet with 6–10 kg weight loss. Bone mass is increased in the obese but bone mass can decrease by 3–15% with weight loss. Some suggest that this loss recovers with weight regain, but not necessarily in postmenopausal women. Uric acid levels also increase with weight, precipitating gout, and also acutely rise with dieting. Musculoskeletal problems result in increased disability and the need for time off work, seen even in those with modest overweight.

**Endocrine abnormalities and psychological factors**

The production of sex steroids is altered in the obese. Obese women, especially those with central adiposity, have increased levels of free testosterone and are hyperandrogenic with marked insulin resistance, the latter implicated in the development of polycystic ovarian syndrome. In obese men, visceral adiposity is associated with a reduced testosterone level. Growth hormone is also reduced in obesity and insulin-like growth factor (IGF-1) is negatively associated with increasing visceral adiposity. Some obese women also show a reduced prolactin stimulatory response associated with a reduced catecholamine rise to insulin induced hypoglycaemia. It has been conjectured that these hormonal abnormalities may be secondary to an increased activity of the hypothalamic-pituitary axis. Corticotrophin-releasing hormone (CRH) which stimulates the secretion of ACTH from the pituitary has also been reported to inhibit both growth hormone releasing hormone and gonadotrophin releasing hormone which influence IGF-1 and sex steroids respectively as well as inducing insulin resistance.

Those who have visceral obesity exhibit a hyperdynamic ACTH secretion to CRH, as well as increased cortisol release from the adrenals, whereas those with gynaecoid obesity show a lesser response, identical to that observed in naturally thin individuals. Urinary free cortisol levels have also been reported to be positively associated with increasing visceral adiposity. As cortisol and insulin both increase fat accumulation, whereas growth hormone and testosterone promote lipid metabolism, the abnormalities noted in the obese would have the effect of promoting fat deposition, especially of visceral fat.

This overactivity of the hypothalamic pituitary axis has been conjectured as due to higher, cortical, influences possibly related to certain socioeconomic factors and psychological problems which appear to be more prevalent in the visceral obese. In population studies, men and women with a higher waist–hip ratio (more visceral obesity) report ill more often, have more frequent peptic ulcers and...
Obesity the disease

stomach bleeding and have more general health complaints\textsuperscript{38}. These individuals use more tranquillisers and antidepressant tablets, have more time off work for sickness, suffer stress related symptoms, sleeplessness and nightmares. They tend to be from the lower socio-economic groups, have poorer education, impaired personality, extraversion, low achievement, aggression, low dominance and have a need for sociability. Such inability to cope with life events in those with a genetic propensity for a certain fat distribution may produce a permanent hyperarousal response which stimulates certain brain peptides, especially CRH, which then drive the pituitary adrenal axis to produce a raised cortisol output\textsuperscript{43}. Such would also be implicated in neuropeptide Y release, one of the brain's most potent peptides for the stimulation of appetite especially for carbohydrates. This peptide could potentiate CRH release producing a vicious cycle driving the individual onwards to store visceral fat\textsuperscript{38}.

Studies into the effects of stress on hormonal levels in monkeys replicate the above scenario\textsuperscript{44}. If the social hierarchy of the monkey colony is deliberately disrupted to induce a helplessness reaction to stress, then the monkeys accumulate visceral fat with increased cortisol secretion, large adrenals, low sex steroid levels, insulin resistance and coronary heart disease.

Respiratory problems

There are a number of ways in which obesity affects lung function\textsuperscript{45}. An increased amount of fat in the chest wall and abdomen limits respiratory excursion reducing lung volume. This is accentuated in the supine position increasing the mechanical work of breathing by 30% in modest obesity and by a 3-fold increase in obesity-hypoventilation syndrome (sometimes called the Pickwickian syndrome). A ventilation/perfusion disturbance resulting in abnormal gas exchange is often observed in extremes of obesity, with underventilated but overperfused lower portions of the lungs. This results in hypoxia with normal arterial carbon dioxide levels, the degree of hypoxia worsening in the supine position. This hypoxia expands the pulmonary blood volume which adds strain to ventricular function. These changes in respiratory function are most important during sleep. During rapid eye movement (REM) sleep, there are decreases in voluntary muscle tone with a reduction in arterial oxygen saturation and a rise in carbon dioxide tension in all individuals, but especially marked in the obese. Irregular respiration and occasional apnoeic episodes often occur in lean people during REM sleep, but obesity with its influence on respiratory mechanics increases...
Obesity

their frequency and may result in severe hypoxia with resultant arrhythmias and cardiac dysfunction.

In many with uncomplicated obesity, these ventilation perfusion defects are countered by increased ventilation which restores blood gases to normal, but in some there develops depression in both hypercapnic and hypoxic respiratory drives and with an accompanying irregular pattern of breathing with apnoeic episodes can result in obesity-hypoventilation syndrome. As their obesity worsens, so do the apnoeic episodes resulting in frequent awakening and the resultant sleep deprivation produces daytime somnolence. Persistent hypoxia further blunts the hypoxic drive resulting in a deteriorating cycle with the development of pulmonary hypertension and eventual right ventricular failure worsening the hypoxia. 24 h monitoring of arterial oxygen saturation using an ear lobe oximeter will help in the diagnosis of those at risk.

Pregnancy

Obese women have a higher risk of obstetric complications. The relative risk of antenatal complications in women whose prepregnancy weight was at least 135% of IBW was reported increased 6.6-fold for the development of diabetes, 1.9-fold for pregnancy induced hypertension and 1.4-fold for urinary tract infections. Other complications include pre-eclampsia (1.5-fold risk), thrombophlebitis, post partum haemorrhage and wound or episiotomy infections. Obese women have an increased risk of Caesarean delivery due to a variety of factors, such as fetal size especially macrosomia (birth weights >4000 g), an increase in maternal pelvic soft tissue narrowing the birth canal, late deceleration of the fetal heart rate, intrapartum meconium staining, prolonged labour, malpresentations and cord incidents. This increased prevalence of Caesarean section occurs in pregnancies with or without antenatal complications. In the latter, the rate has been reported as high as 19.6% in the morbidly obese compared to 10.1% in normal weight and 12.4% in moderate obesity. Fetal weight appears to be directly proportional to maternal size with more than 50% of obese women having babies who weigh greater than 3600 g. Maternal weight when not associated with antenatal complications is not associated with an increased perinatal mortality, but this is increased if there is an antenatal complication by as much as 3-fold. Recently, an increased risk of neural tube defects, especially spina bifida, has been reported in women with BMI greater than 29 (odds ratio 1.9).
Conclusion

Obesity is a disease associated with extensive human suffering and also a massive financial cost to society. It has been estimated that for the year 1990, the direct cost of obesity associated disease in the USA alone was $45.8 billion and the indirect costs $23 billion\textsuperscript{50}. Hence the total economic cost of obesity in USA was estimated as $68.8 billion\textsuperscript{50}. At such a cost to society, obesity is not a social condition but a rampant disease. Obesity is not simply a matter of overeating and lack of will power but a disease with a major genetic aetiology modified by the environment and should be treated vigorously in the same manner that we now apply to its associated co-morbid conditions such as diabetes, coronary heart disease, hypertension and hyperlipidaemia.

References

2 Sorensen TIA. The genetics of obesity. Metabolism 1995; 44 Suppl 3: 4–6
50 Wolf AM, Colditz GA. The cost of obesity; the US perspective. Pharmacoeconomics 1994; 5 Suppl 1: 34–7