INTRODUCTION

The prevalence of obesity is increasing globally, with nearly half a billion of the World’s population now considered to be overweight or obese (Rossner, 2002). The obesity epidemic is related both to dietary factors and to an increasingly sedentary lifestyle. Obesity has significant co-morbidities and these are associated with substantial health care and social costs. It is a complex disorder of appetite regulation and energy metabolism controlled by specific biological factors. It arises when the intake of food is in excess of physiological requirements.

Obesity can be defined as a disease in which excess body fat has accumulated such that health may be adversely affected (Kopelman, 2000). Obesity is a worldwide health problem and it does not only affect developed countries, as there is now a significant increase in overweight and obesity throughout the developing world. The prevalence of obesity has recently increased in Asian as well as Western countries (Ohzeki et al., 1990). Obesity is relatively common in Europe. It has been estimated (Speakman, 2003a) that nine people in the USA and one person in the UK die every 15 minutes as a direct consequence of obesity related illnesses. Current prevalence data from individual national studies collated by the International Obesity Task Force (IOTF) suggest that obesity ranges from 10 to 20% for men, and 10 to 25% for women. Kopelman (2000) suggests that obesity is now so common within the world’s population that it is beginning to replace undernutrition and infectious diseases as the most significant contributor to ill health. It is estimated that, at the beginning of this century, more people will die from complications of overnutrition than of starvation. The pandemic of obesity is so great that it has even spawned a new word ‘globesity’ (Speakman, 2003a).

The increasing prevalence of childhood obesity throughout the past two decades has been emphasized in many Western countries. The World Health Organization (1998) recognized obesity as a major public health epidemic worldwide, in developed as well as in some developing countries. In the UK, the prevalence of overweight in children was 14.7% in 1989 and 23.6% in 1998, while that of obesity increased from 5.4 to 9.2% during this period (Bundred et al., 2001). Between 1975 and 1995 the prevalence of overweight rose from 10.0 to 16.3% in German boys, and from 11.7 to 20.7% in German girls (Kromeyer-Hauschild et al., 1999). Between 1963 and 1991, the prevalence of overweight increased from 15 to 22.5% in 6-11 years old North American children, while that of obesity increased from 5 to 11% (Troiano et al., 1995). An excellent and exhaustive review of international trends in adolescent obesity has been given by Schneider (2000).

Obesity is caused by a combination of both genetic and environmental influences. It may develop at any age in either sex and in an increasing health problem. The excess of fat in men tends to accumulate in the upper abdomen. In women, the favoured sites for the accumulation of fat are the buttocks, hips and thighs (Bose, 1995). The site of fat accumulation is considered a predominant factor for metabolic disorders of obesity (Van Gaal et al., 1988). However, the incidence of obesity was found to be higher among women than men (Pi - Sunyer, 1994).

BACKGROUND

Adipose Tissue

In recent years, it has become increasingly clear that the distribution of adipose tissue is important when considering the risk of obesity (Sharma, 2002). Adipose tissue is a normal constituent of the human body that serves the important function of storing energy as fat for mobilization in response to metabolic demands. It is mostly located in the skin and subcutaneous tissue. In normal individuals, 65-70 per cent of adipose tissue is fat. Adipose tissue makes up 15-30 per cent of the total body weight of most adult humans.

Types of Adipose Tissues: There are two types of adipose tissues – White Adipose Tissue (WAT) and Brown Adipose Tissue (BAT), the latter being a special type of lipid which constitutes a very small part of total body fat. BAT performs a great thermogenic function especially in infants. In them, it is quite abundant and is located around clavicles, towards axilla, around kidneys and in the posterior peritoneum. BAT is different in being multilocular and in having abundant mitochondria. The mammals and
their young ones have abundant stores of BAT. WAT is the storage fat that is pressed into service to meet the metabolic needs of the body. The body uses WAT and it is stored for future use at the time of starvation.

**Adipose Tissue as an Endocrine Organ:** Until recently, the adipocyte was largely thought to be an inert storage cell whose main function was to store excess energy in the form of triglycerides. It is now apparent that adipocytes have a more complex role in the organism. Thus adipocytes produce a large number of hormones, peptides and smaller molecules that impact on metabolism and on cardiovascular regulation, not only in an autocrine and paracrine manner but also in an endocrine manner. This supports the view that adipose tissue must be regarded as an endocrine organ (Engeli and Sharma, 2000).

**Obesity**

**Physiology of Obesity:** The amount of fat in the body (adiposity) is precisely regulated as part of the process of energy homeostasis, a process whereby energy intake (food intake) is matched to energy expenditure (metabolism and exercise) and the size of the body’s energy stores (the fat mass). Obesity is a problem of energy imbalance (Speakman, 2003b). Generally:

\[
\text{Food energy intake} = \text{Energy expenditure} + \text{Storage.}
\]

The major organ regulating this system is the brain, although multiple organ systems participate in the process. Adiposity signals enter the brain at the level of the hypothalamus. Neural signals from the gastro-intestinal system and the liver provide information about the food that is being eaten. These satiety signals are sent to the hindbrain. The brain responds to the hormone signals via integrated neuropeptide pathways, leading to a number of outputs that are directly related to the energy homeostasis.

There is strong evidence that key hormonal regulatory signals – the adiposity hormones – control both how much is eaten and how much energy is expended. These hormones circulate in the blood in direct proportion to body fat content. They enter the brain and act on receptors in areas of the hypothalamus known to regulate food intake and energy expenditure (Woods and Seeley, 2002).

**Types of Obesity:** French physician Jean Vague (1956) was the first to point out that there may be two types of human obesity (Android or visceral or upper body obesity and Gynoid or lower body obesity), depending on the bodily distribution of fat and their susceptibility to chronic diseases. Furthermore, he emphasized that individuals with **Android** (upper body) obesity had more risk of developing health problems compared to those with **Gynoid** (lower body) obesity (Fig. 1).

Visceral obesity refers to the accumulation of adipose tissue within the abdomen. Abdominal fat deposition is characterized by an increase in waist circumference. Abdominal or visceral fat (android obesity) is associated with the cardiovascular risk factors of the Metabolic Syndrome. These include Type 2 diabetes, impaired glucose tolerance, and hypertension and dyslipidaemia (high triglyceride, low LDL cholesterol). It is the mass of visceral adipose tissue which leads to these abnormalities. Visceral obesity is also associated with increased inflammatory response, increase in prothrombotic factors, renal hyperfiltration, endothelial dysfunction and several lipid abnormalities. All of these factors can contribute to the development of cardiovascular disease and end-organ damage in obese patients (Sharma, 2002).

Adipose tissue associated with upper body obesity contains large insulin-resistant adipocytes while adipose tissue associated with lower body obesity contains small insulin-sensitive adipocytes. Intra-abdominal adipose tissue is more metabolically active (Bjorntorp, 2000).

**Assessment of Obesity:** It is not just the amount of fat but also its distribution that determines the risk associated with obesity. Obesity can be measured in two main ways – generalised obesity and central obesity. Height and weight are the most simple and commonly used measures. A number of weight-for-height indices have been developed of which the body mass index (BMI) [defined as weight/height (kg/m²)] is the most commonly used measure of overall obesity (generalised obesity) while circumferences and skin-folds are measures of central obesity. Central body fat distribution is measured by waist
circumference (WC) and two commonly used indices, waist-hip ratio (WHR) and conicity index (CI) (Bhadra et al., 2001).

In recent years, the body mass index (BMI) has become the medical standard used to measure overweight and obesity. BMI can be considered to provide the most useful, albeit crude, population-level measure of obesity. It is the measurement of choice for many obesity researchers and other health professionals. Most health organizations and published information on overweight and its associated risk factors use BMI to measure and define overweight and obesity. It may be used to estimate the prevalence of obesity within a population and the risk associated with it. In cross-sectional comparisons, BMI does not directly measure percent of body fat, but it provides a more accurate measure of overweight and obesity than relying on weight alone. However, BMI values may be paraphrased with caution if estimates of body fat are required. It is more highly correlated with body fat than other indices of height and weight.

BMI generally correlates highly with adiposity, although it can sometimes misclassify total body fat content. For example, athletes who are muscular have a high BMI, due to muscle weighing more than fat, and will have BMIs within the overweight and obesity range even though they are not fat. The shortest and tallest subjects also tend to be misclassified as obese. It may be noted that BMI is a height-weight system of measurement that applies to both sexes. It’s not a perfect system, because (e.g.) very muscular people may fall into the “overweight” category when they are actually healthy and fit. But it’s a useful pointer for most people.

A graded classification of overweight and obesity using BMI values provide valuable information about increasing body fatness. It allows meaningful comparisons of weight status within and between populations and the identification of individuals and groups at risk of morbidity and mortality (Bose, 1995). For meaningful comparison between or within populations, WHO (1998) advised the use of BMI cut-off points. A BMI value of 30 or more is now widely accepted as denoting obesity. It allows a firm basis for evaluating interventions. For adults, WHO (1998) has recommended 25.0 kg/m² and 30.0 kg/m² as the cut-off points of overweight and obesity, respectively. These BMI values are age-independent and the same for both sexes. The table shows a simplistic relationship between BMI and the risk of morbidity, which can be affected by a range of factors, including the nature of diet, ethnic group and activity level (Table 1).

WHO (2000) recommended different ranges for the Asia-Pacific region based on risk factors and morbidities (Table 2). In Asians, the cut-offs for overweight (23.0 kg/m²) and obesity (25.0 kg/m²) are lower than the international WHO (1998) criteria. These provisional recommendations will need to be revised in the light of further validation of studies and clinical experience. Some support for these cut-offs comes from data on Chinese living in Hong Kong (Ko et al., 1999). Similar data have been published from the Chinese in Singapore (Deurenberg-Yap et al., 1999) and in Indian Asians living in Mauritius, where there is a significantly increased risk of Type 2 diabetes and hypertension among those with a BMI between 23 to 24.9 kg/m² compared to those within the normal range. Clearly these cut-offs do not apply to Pacific Islanders. In these populations, higher cut-offs are required to define overweight and obesity of BMI 26 kg/m² and BMI 32 kg/m² respectively (Swinburn et al., 1999). However, sparse data exists at present to make definitive recommendations.

<table>
<thead>
<tr>
<th>Table 1: Classification of adults obesity according to BMI</th>
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<tr>
<td>Classification</td>
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<td>Underweight</td>
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<tr>
<td>Normal range</td>
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<tr>
<td>Overweight</td>
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<td>Preobese</td>
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<td>Obese class I</td>
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<td>Obese class II</td>
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| Table 2: Proposed classification of weight by BMI in adult Asians
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<tr>
<td>Classification</td>
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<tr>
<td>Underweight</td>
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<td>Normal range</td>
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<td>Overweight</td>
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<td>At risk</td>
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<td>Obese II</td>
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It has now been well established that the prevalence of childhood obesity is increasing rapidly worldwide. Because of their public health importance, the trends in childhood obesity have been
closely monitored. The measurement of overweight and obesity in children and adolescents poses particular problems due to different rates of maturation and growth. As children are still growing, the adult BMI cut-offs are not considered appropriate for children. Adiposity measures are linked to a child’s stage of maturation at the time of measurement and there are two periods when adiposity increases - about the age 5-7 years, and in early puberty. Although a fixed cut-off can be used to define obesity in adults, these need to be adjusted for age in childhood (and additionally for maturation in adolescence). Some countries have their own charts for calculating weight, height and obesity for age (e.g. Singapore). However, recent agreement has been reached on appropriate measures of adiposity which allow classification and comparison. The BMI-for-age chart is recommended. Those greater than the 95th percentile are considered obese, whilst those greater than the 85th percentile of BMI for age are “at risk” (WHO, 1995).

Although BMI-for-age charts have been developed for other countries, many of these are outdated or are only applicable to a narrow age range. However, BMI-for-age charts have recently been developed for Swedish, Italian and British children (WHO, 2000).

Cole et al. (2000) recommended an internationally suitable definition of child overweight and obesity, specifying the measurement, the reference population, and the age and sex specific cut off points. The proposed cut off points, which are less arbitrary and more internationally based than current alternatives, should help to provide internationally comparable prevalence rates of overweight and obesity in children. In this chart the 85th percentile and the 95th percentile roughly correspond to BMIs of 25 kg/m² and 30 kg/m², respectively in 18 year olds. This international cut off points for body mass index for overweight and obesity by sex between 2 and 18 years, defined to pass through body mass index of 25 and 30 kg/m² at age 18, obtained by averaging data from Brazil, Great Britain, Hong Kong, Netherlands, Singapore, and United States (Table 3). These are unlikely to be appropriate for Asian and Pacific children; and in any case, they artificially confine the prevalence of overweight to 15% and of obesity to 5% of the population.

Obese individuals with excess fat in the intra-abdominal depots are at particular risk of the adverse health consequences of obesity. Abdominal obesity is of particular concern as it is associated with greater risks to health than a more peripheral fat distribution. Abdominal or visceral fat (android obesity) is associated with the cardiovascular risk factors of the Metabolic Syndrome. These include Type 2 diabetes, impaired glucose tolerance, and hypertension and dyslipidaemia (high triglyceride, low LDL cholesterol). It is the mass of visceral adipose tissue which leads to these abnormalities.

<table>
<thead>
<tr>
<th>Age (in years)</th>
<th>Overweight (BMI ≥ 25 kg/m²)</th>
<th>Obesity (BMI ≥ 30 kg/m²)</th>
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<tbody>
<tr>
<td></td>
<td>Males</td>
<td>Females</td>
</tr>
<tr>
<td>2.0</td>
<td>18.41</td>
<td>18.02</td>
</tr>
<tr>
<td>2.5</td>
<td>18.13</td>
<td>17.76</td>
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<tr>
<td>3.0</td>
<td>17.89</td>
<td>17.56</td>
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<tr>
<td>3.5</td>
<td>17.69</td>
<td>17.40</td>
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<tr>
<td>4.0</td>
<td>17.55</td>
<td>17.28</td>
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<tr>
<td>4.5</td>
<td>17.47</td>
<td>17.19</td>
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<tr>
<td>5.0</td>
<td>17.42</td>
<td>17.15</td>
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<tr>
<td>5.5</td>
<td>17.45</td>
<td>17.20</td>
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<tr>
<td>6.0</td>
<td>17.55</td>
<td>17.34</td>
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<tr>
<td>6.5</td>
<td>17.71</td>
<td>17.53</td>
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<td>7.0</td>
<td>17.92</td>
<td>17.75</td>
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<tr>
<td>7.5</td>
<td>18.16</td>
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<td>8.0</td>
<td>18.44</td>
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<tr>
<td>8.5</td>
<td>18.76</td>
<td>18.69</td>
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<tr>
<td>9.0</td>
<td>19.10</td>
<td>19.07</td>
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<tr>
<td>9.5</td>
<td>19.46</td>
<td>19.45</td>
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<tr>
<td>10.0</td>
<td>19.84</td>
<td>19.86</td>
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<tr>
<td>10.5</td>
<td>20.20</td>
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<td>11.0</td>
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<tr>
<td>11.5</td>
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<td>12.5</td>
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<tr>
<td>13.5</td>
<td>22.27</td>
<td>22.98</td>
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<td>14.5</td>
<td>22.96</td>
<td>23.66</td>
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<td>15.0</td>
<td>23.29</td>
<td>23.94</td>
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<tr>
<td>15.5</td>
<td>23.60</td>
<td>24.17</td>
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<td>16.0</td>
<td>23.90</td>
<td>24.37</td>
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<td>16.5</td>
<td>24.19</td>
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<tr>
<td>17.0</td>
<td>24.46</td>
<td>24.70</td>
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<tr>
<td>17.5</td>
<td>24.73</td>
<td>24.85</td>
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<tr>
<td>18.0</td>
<td>25.00</td>
<td>25.00</td>
</tr>
</tbody>
</table>

Source: Cole et al. (2000).
cardiovascular disease (CVD) and other forms of chronic disease (diabetes mellitus, gout, osteoarthritis, cancers etc.) even though the risks seem to vary in different populations. Moreover, sex differences in central body fat distribution exists among different populations (Bhadra et al., 2002).

When used in a ratio with the hip circumference, waist circumference is an indicator of the degree of masculine distribution of adipose tissue. Over the past ten years or so, it has become accepted that a high waist - hip ratio (waist - hip ratio > 0.95 in men and > 0.85 in women) indicates abdominal fat accumulation. However recent evidence suggests that waist circumference alone may provide a more functional correlate of abdominal fat distribution and associated ill health (Lean et al., 1995). Thus, waist circumference may be the preferred measure of abdominal obesity compared to the WHR (WHO, 1998).

In addition to the anthropometric assessment, various other sophisticated tools [bioelectrical impedance analysis (BIA), magnetic resonance imaging (MRI), dual-energy x-ray absorptiometry (DEXA), isotope dilution, computed tomography (CT), ultrasound etc.] are useful for measuring body fat in certain clinical situations specially medical treatment and in obesity research. These tools are particularly useful when trying to identify the genetic and environmental determinants of obesity and their interactions, as they enable the variable and complex nature of obesity to be split up into separate components (WHO, 1998). Thus, obese individuals can be characterized by measuring body composition, anatomical distribution of fat and energy intake, among others.

Causes of Obesity: Several factors may contribute to the development of obesity. It is not a single disorder but a heterogeneous group of conditions with multiple causes. Georges et al. (1991) suggest a larger role for socio-cultural factors in the patterning of body fat distribution. Muller and Reid (1979) suggested that environmental factors such as nutrition, stress and exercise have significant effect on subcutaneous fatness (body fat). The prevalence of obesity was inversely associated with the level of education and physical activity. Socio-economic status (SES) and behavioral factors are important determinants of weight gain and overweight (Rissanen et al., 1991). Recent epidemiological trends in obesity indicate that the primary cause of the global obesity problem lies in environmental and behavioural changes. The world-wide obesity problem can be viewed as a consequence of the substantial economic, social and cultural problems now confronting developing and newly industrialized countries.

Despite living in similar environments not everyone gets obese. These differences potentially have at their cause a large genetic component (Speakman, 2000). Research continues into the genetics of obesity and an update of the human obesity gene map has been published. More than 300 genes, markers, and chromosomal regions have been associated or linked with human obesity phenotypes. The number of genes and other markers associated or linked with human obesity phenotypes continues to expand (Chagnon et al., 2003). Of course, some of these loci will turn out to be more important than others, and many will eventually be proven to be false positives. The main goals remain to identify the combination of genes and mutations that are contributing to the predisposition to human obesity and to determine the environmental circumstances under which these gene combinations and mutations occur. As we near the completion of the final draft of the human genome sequence and, with it, a more exhaustive annotation of the genome, we expect continuous advances in the understanding of the genetic basis of the predisposition to human obesity (Rossner, 2002).

Consequences of Obesity: Regardless of its cause, obesity may be associated with a variety of risks. Obesity causes or exacerbates many health problems, both independently and in association with other diseases. In particular, obesity is associated with the development of type-II diabetes mellitus or non-insulin dependent diabetes mellitus [NIDDM] (Gordon et al., 1977; Bose, 1992), coronary heart disease [CHD] (Foster and Burton, 1985; Ghosh et al., 2003, 2004), respiratory complications, dyslipidaemia, gout, osteoarthritis of large and small joints, sleep apnoea and other degenerative conditions associated with higher mortality (Seidell and Bouchard, 1997). Obesity has been implicated as a risk factor in the development of hypertension [HT] (Bose and Mascie-Taylor, 1998; Vague et al., 1988). It is also associated with an increased incidence of certain forms of cancer. In women, the higher mortality rate is associated with endometrial, gallbladder, cervical, ovarian and breast cancers (Garfinkel, 1985).

Economic Costs of Obesity: The costs of obesity to a community and individuals may be divided into the direct costs to the health system and the indirect or social costs to the individual and community (e.g. sick days, individual expenditure on weight loss). The direct costs depend
in the main part on the diseases caused by obesity and the cost of these diseases. One of the problems in this assessment is the relative risk of the disease in different communities and ethnic groups. Several methods have been used to calculate these costs and there have been a wide range of results on the cost of obesity in different countries. Some diseases which have been included in the calculations are Type 2 diabetes, heart disease, hypertension, endometrial cancer, arthritis and colorectal cancer. Indirect costs also vary widely. One of the latest estimates in the United States for indirect costs was US $47.6 billion per year. Another more recent estimate (Speakman, 2003b) has put the figure at $97 billion annually. Obesity currently costs the UK economy around £2.5 billion every year and leads to the premature death of 30,000 people and a shortening of lifespan in the obese by around nine years (Speakman, 2003c). Little data is yet available for the Asia-Pacific region. It is necessary to develop a standard way of calculating the costs of obesity so that various countries’ health expenditure can be compared and for the benefits of treatment to be calculated. For these reasons, suggested standard formulae are being prepared (WHO, 2000).

Prevention of Obesity: Prevention of obesity is much more desirable than treatment of the condition once it is established. Prevention of overweight and obesity is supposed to begin early in life. Generally, there are three approaches to prevention. Universal prevention is based on a total population approach, whereas selective and targeted prevention strategies are directed at high-risk groups.

Universal Prevention: Measures directed at the whole population should aim to stabilise the level of obesity and eventually lower the incidence and hence the prevalence of obesity. A reduction in weight-related disease by lifestyle modification including improved diet and physical activity levels are objectives, as well as a reduction in smoking and alcohol consumption.

Selective Prevention: Selective prevention aims to educate sub-groups of the population with a high-risk of obesity so that they can deal effectively with the risk factors, which may be genetic and which predispose them to obesity (WHO, 1998). Such strategies can be initiated in appropriate settings which allow access to these high-risk groups, including schools, community centers and primary care venues.

Targeted Prevention: Targeted prevention aims to prevent weight gain and reduce the number of people with weight-related disorders in those individuals that are already overweight or those with biological markers associated with excess adiposity, who are not yet obese (WHO, 1998). This group has a particularly high risk of developing obesity and obesity-related disorders.

Management of Obesity in Adults

Lifestyle Approaches: Weight management strategies should include modification of diet and physical activity, and of daily habits and thoughts. Specific behaviours conducive to over-eating or under-activity need to be identified and corrected. Weight loss is more likely to be achieved and maintained by behaviour modification techniques that focus on lifestyle and attitude.

Dietary Measures: Many countries have dietary guidelines based on local experience and data. In some parts of Asia, diets are high in carbohydrates and fat, and low in protein. Modifications need to be made to reduce the total calories by lowering the intake of highly refined carbohydrates and replacing these with complex carbohydrates, and reducing fat intake. All weight management strategies need to educate patients about food and healthy eating habits. Dietary therapy essentially involves instructing patients on changing their diet in order to restrict caloric intake. It has been reported (Speakman, 2003c) that the most beguiling aspect of diets is that, in the short term, they produce significant weight loss. However, the only way to permanently lose weight on a diet is to stay permanently on the diet.

Physical Activity: Another important factor in a successful weight management programme is increasing daily physical activity (Speakman, 2003b). If physical activity or exercise is used alone to treat obesity, only a moderate weight loss of between 4-5 kg over a three month period is likely. Physical activity advice should be tailored for age and cultural climate and emphasize increased daily activity such as walking and climbing stairs. It is not necessary for the obese patient to participate in strenuous activity; low to medium intensity is sufficient.

Pharmacotherapy: Pharmacotherapy should be part of a long-term management strategy for obesity that is specific to a given patient. In certain cases pharmacological treatment may need to be considered in addition to diet, exercise and behav- iour modification. To date, there is little published scientific evidence reporting the long-term safety and efficacy of currently available anti-obesity drugs, thus no particular drug can be recom- mended for routine use. There is also no available data on the effects of combining two or more
types of these anti-obesity drugs (WHO, 2000).

**Very Low Calorie Diets (VLCD):** The phrase VLCD generally refers to commercially available package diets that provide daily requirements of first grade protein, vitamins and essential minerals, together with sufficient carbohydrate to prevent electrolyte loss (‘Modifast’, ‘Optifast’). VLCDs usually allow between 400-800 calories/day and can induce an immediate weight loss of 1.0-1.5 kg per week that is predominantly fat loss with minimization of negative nitrogen balance. However, they do not change eating habits or result in weight loss over the long-term. VLCDs are effective weight loss treatments and may be used in those with severe obesity or who have another medical reason for needing rapid weight loss. In clinical trials, weight loss due to low calorie diets was just as effective as VLCDs after a one year period (Wadden et al., 1994; Richman et al., 1992). Although they are effective therapy, they should only be used under medical supervision and with a weight management and weight maintenance programme.

**Surgery:** Gastric-partition surgery can be an effective therapy to treat those with BMI > 40 kg/m². Current surgical methods include gastric banding or stapling, laparoscopic placement of an adjustable inflatable band and gastro-jejunal bypass surgery. Interim results from the Swedish Obesity Study indicate weight loss of the order of 35-55 kg, maintained for 4 years, with greatly improved quality of life measures. Liposuction is not a treatment for generalised obesity, but may be used for unsightly local collections of fat or for a specific medical purpose.

**Management of Obesity in Children**

Management of obesity in children differs from that in adults in that the prevention of weight gain is of importance rather than focusing on weight loss. Lean body mass increases as children get older; thus, keeping fat mass constant will eventually help to normalise body weight. The best and most effective way to treat children with obesity is to treat the family, and not the child alone, by encouraging increased daily activity and healthy eating habits. Higher energy expenditure can be achieved more effectively through increased general activity in schools and play rather than competitive or structured sports. In developed countries, the recent increase in the prevalence of childhood obesity appears to be related to the time spent in sedentary behaviour associated with non-active leisure pursuits. Television viewing, computer and video games are principal sources of inactivity among children. The role of pharmacotherapy in children and adolescents is undefined but may be considered in extreme cases. There has been little or no efficacy or outcomes data on pharmacotherapy in children <18 years. In the diets of obese children, only small reductions in energy-intake should be made so that sufficient energy and nutrients are available to ensure normal growth and development. VLCDs are not generally recommended in childhood (WHO, 2000).

**FUTURE RESEARCH**

The following research areas are identified as being of particular importance –

- The sex and ethnic specific cut-off points of obesity.
- The validity and tracking of simple measures of excess weight, e.g. BMI – for age and sex in children and adolescents from different societies and ethnic groups.
- The relationship between BMI and adiposity in stunted children.
- BMI standards for the elderly (> 60 years or > 80 years).
- The interaction between measures of fitness and both dietary factors and physical activity in determining obesity co-morbidities.
- Standardised studies need to be performed to determine the prevalence of obesity, and its co-morbidities such as Type 2 diabetes, hypertension, cancers and dyslipidaemia.
- These prospective epidemiological studies need to be repeated on the same populations to determine the relative risk of developing these co-morbidities with obesity.
- Studies need to be performed in both Asian and Pacific Island countries to determine the relationship between BMI, waist circumference and risk of development of co-morbidities.
- The above studies will enable the development of waist circumference cut-offs to classify abdominal obesity on the same basis as those for BMI and should include the effectiveness of other simple clinical measures of obesity for determining risk in various populations e.g. waist to height, waist to sitting height, waist circumference and waist to hip ratio.
- Studies which investigate the attitudes to obesity are needed as the results of such studies will guide planned interventions.
- Together with this, intervention studies on the prevention of obesity-related chronic diseases are needed.
REFERENCES


HUMAN OBESITY: A BACKGROUND

Key Words: Obesity. Adiposity. Body Mass Index. Nutritional Status. Chronic Diseases

Abstract

Obesity has emerged as the most prevalent serious public health problem of our time. It is a condition of excessive fat accumulation in adipose tissue, to the extent that health may be impaired. Certain detrimental effects to health are attributed to obesity. Obesity may develop at any age in either sex. Several factors may contribute to the development of obesity. Obesity develops over time and, once it develops, is difficult to treat. Obesity should not be regarded simply as a cosmetic problem affecting certain individuals, but a crisis that threatens global well being. An estimated 300 million people around the world are obese. The International Obesity Task Force (IOTF) conservative estimates, based on current trends, show that obesity levels will continue to rise in the early 21st century - with severe health consequences unless urgent action is taken now. It is half a century since obesity was introduced into the international classification of diseases. In the 21st century it has become an epidemic. The links between overweight and obesity and a range of other serious diseases from diabetes and heart disease to cancer are now more clearly understood, yet little is being done to tackle this world-wide problem effectively. Obesity levels in some countries have doubled in recent years and are rising in developing countries like India too. The consequences of ignoring obesity are increasing levels of serious illness and rising health costs.

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