Obesity in children and young people: a crisis in public health

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SUMMARY: THE RISING CRISIS

Ten per cent of the world’s school-aged children are estimated to be carrying excess body fat (Fig. 1), with an increased risk for developing chronic disease. Of these overweight children, a quarter are obese, with a significant likelihood of some having multiple risk factors for type 2 diabetes, heart disease and a variety of other co-morbidities before or during early adulthood. The prevalence of overweight is dramatically higher in economically developed regions, but is rising significantly in most parts of the world.

In many countries the problem of childhood obesity is worsening at a dramatic rate. Surveys during the 1990s show that in Brazil and the USA, an additional 0.5% of the entire child population became overweight each year. In Canada, Australia and parts of Europe the rates were higher, with an additional 1% of all children becoming overweight each year.

The burden upon the health services cannot yet be estimated. Although childhood obesity brings a number of additional problems in its train – hyperinsulinaemia, poor glucose tolerance and a raised risk of type 2 diabetes, hypertension, sleep apnoea, social exclusion and depression – the greatest health problems will be seen in the next generation of adults as the present childhood obesity epidemic passes through to adulthood. Greatly increased rates of heart disease, diabetes, certain cancers, gall bladder disease, osteoarthritis, endocrine disorders and other obesity-related conditions will be found in young adult populations, and their need for medical treatment may last for their remaining life-times. The costs to the health services, the losses to society and the burdens carried by the individuals involved will be great.

The present report has been written to focus attention on the issue and to urge policy-makers to consider taking action before it is too late. Specifically, the report:

• reviews the measurement of obesity in young people and the need to agree on standardized methods for assessing children and adolescents, and to compare populations and monitor trends;

• reviews the global and regional trends in childhood obesity and overweight and the implications of these trends for understanding the factors that underlie childhood obesity;

• notes the increased risk of health problems that obese children and adolescents are likely to experience and examines the associated costs;

• considers the treatment and management options and their effectiveness for controlling childhood obesity;

• emphasizes the need for prevention as the only feasible solution for developed and developing countries alike.

This document reflects contributions from experts working in a wide range of circumstances with a diversity of approaches, but with many shared opinions. The report has been endorsed by the Federation of International Societies for Paediatric Gastroenterology, Hepatology and Nutrition (FISPGHAN) and the International Paediatric Association (IPA).
Health professionals are aware that the rising trends in excess weight among children and adolescents will put a heavy burden on health services (for example, 10% of young people with type 2 diabetes are likely to develop renal failure by the time they enter adulthood, requiring hospitalization followed by life-long dialysis treatment (2). Health services, especially in developing countries, may not easily bear these costs, and the result could be a significant fall in life expectancy.

In industrially developed countries, children in lower-income families are particularly vulnerable because of poor diet and limited opportunities for physical activity. There may also be an ethnic component; for example, in the USA the prevalence of overweight among children aged 4–12 years rose twice as fast in Hispanic and African–American groups compared with white groups over the period 1986–1998 (3).

In developing nations child obesity is most prevalent in wealthier sections of the population. However, child obesity is also rising among the urban poor in these countries, possibly due to their exposure to Westernized diets coinciding with a history of undernutrition.

Such rapid changes in the numbers of obese children within a relatively stable population indicate that genetic factors are not the primary reason for change. Some migration of populations may account for a proportion of the epidemic, but cannot account for it all. Although studies of twins brought up in separate environments have shown that a genetic predisposition to gain weight could account for 60–85% of the variation in obesity (4), for most of these children the genes for overweight are expressed where the environment allows and encourages their expression. These obesity-promoting environmental factors are sometimes referred to as 'obesogenic' (or 'obesigenic'). Put graphically, a child's genetic make-up 'loads the gun' while their environment 'pulls the trigger' (5). A genetic predisposition to accumulate weight is a significant element in the equation, but its importance might best be viewed from another perspective: the genes that predispose for obesity are likely to be commonplace, with only a small

Figure 1 Prevalence of overweight and obesity among school-age children in global regions. Overweight and obesity defined by IOTF criteria. Children aged 5–17 years. Based on surveys in different years after 1990. Source: IOTF (1).
Obesity in children and young people

The proportion of children able to resist gaining weight in an obesogenic environment.

The changing nature of the environment towards greater inducement of obesity has been described in WHO Technical Report (6) on chronic disease as follows:

‘Changes in the world food economy have contributed to shifting dietary patterns, for example, increased consumption of energy-dense diets high in fat, particularly saturated fat, and low in unrefined carbohydrates. These patterns are combined with a decline in energy expenditure that is associated with a sedentary lifestyle—motorized transport, labour-saving devices at home, the phasing out of physically demanding manual tasks in the workplace, and leisure time that is preponderantly devoted to physically undemanding pastimes.’ (pp. 1–2)

This emphasis on the environmental causes of obesity leads to certain conclusions: first that the treatment for obesity is unlikely to succeed if we deal only with the child and not with the child's prevailing environment, and second that the prevention of obesity – short of genetically engineering each child to resist weight gain – will require a broad-based, public health programme.

A doctor presented with an obese child must nevertheless attempt some form of remedial intervention to prevent the child's health deteriorating. The aim is to stabilize and hopefully reduce that child's accumulation of body fat, using a range of approaches discussed in the next few paragraphs.

Helping the obese child

For a great majority of obese patients, the first point of contact is with a primary care physician or a public health nurse. Yet the relevant training in bariatric methods (methods related to the assessment, prevention and treatment of obesity) at the undergraduate level remains inadequate. Two national surveys in the USA conducted over 10 years, indicated that paediatric obesity was the most wanted topic for continuing medical education (7).

For children who are moderately overweight, measures to prevent further weight gain, combined with normal growth in height, can be expected to lead to a decrease in BMI – i.e. children may be able to 'grow into' their weight. For the more seriously obese child, treatment regimes are largely palliative and designed to manage and control rather than resolve the problem. Weight control and improved self-esteem may be achieved, but the child is likely to remain seriously overweight and at risk of chronic disease throughout his or her life.

The clinical management of obese children may require an extended amount of time and the assembly of a professional team including a dietitian, exercise physiologist and psychologist in addition to the physician. As paediatric obesity becomes more common, patient management may not be restricted to obesity clinics and other forms of management may be developed. Obesity clinics may be necessary for morbid obesity, but less severe forms of obesity may be better managed in primary care settings by a range of health practitioners.

Obesity control in adults relies on a range of options: improvements in nutritional habits, raised levels of physical activity, behavioural modification and psychotherapy, pharmaceutical treatment and as a last resort, surgery. These options can be used alone or in combination.

For children, neither surgery nor drug therapy can currently be recommended unless within a closely monitored research study (8). Of the remaining choices, no single method will ensure success, although some consensus exists. For example, reducing the time engaged in sedentary activities (such as watching television or playing computer and video games) has been shown to facilitate better treatment outcome (9).

Dietary interventions in combination with exercise programmes have been reported to have better outcomes than dietary modification alone. Exercise programmes alone without dietary modification are unlikely to be effective, because increased energy expenditure is likely to be matched by increased energy intake (10). A whole-family approach also appears vital, with several studies showing that outcomes are improved if the parents are engaged in the process, or even are the key instigators of the process, at least for younger children (11).

Very strict dietary limitations were reported to have better short-term results than moderate dietary limitations. However, strictly modified diets cannot be maintained for long periods of time. More marked rebound effects are observed after the discontinuation of strict diets than after moderate dietary modifications. Two additional concerns regarding strict dietary limitations are: (1) the risk of not meeting basic nutrient requirements and thus adversely affecting growth; and (2) the risk of inducing adverse psychological effects, including appetite or eating disorders, feelings of stigmatization, anxiety and low self-esteem, especially if the intervention is not successful or the child has prior psychological problems (12,13).

Many questions regarding what constitutes the best treatment remain unanswered: there have been few sufficiently large multicentre clinical trials to test the efficacy and safety of well-defined obesity treatment programmes. Such trials may reveal which non-pharmacological and non-surgical interventions can help manage obesity over the long term. Losing weight over the short term, but then experiencing a rebound gain in weight, remains the
Prevention is the only realistic solution

If the current approach to treatment is largely aimed at bringing the problem under control, rather than effecting a cure, and if this aim is only successful when a multi-disciplinary and intensive regimen is mounted, then managing the obesity epidemic will be vastly expensive and probably unaffordable for most countries. Pharmaceutical approaches may assist, but cannot replace, the multi-disciplinary management of obesity. Prevention is the only feasible option and is essential for all affected countries. Yet effective techniques for prevention have also proved elusive.

Programmes to prevent obesity in children may start by identifying those children at high risk, rather than waiting to see if they will develop obesity. Although screening for obesity potential may help target resources where they are most needed, such screening also creates stigma among the children identified if they are singled out for special attention. Furthermore, genetic studies suggest that most children are at risk of obesity, and that strategies to prevent obesity in a child population – such as encouraging healthful diets and plentiful physical activity – will benefit the health of all children, whether at risk of obesity or not.

The most logical settings for preventive interventions are school settings and home-based settings. A number of interventions have been tried at these levels, and these are reviewed in the present report, but success has been hard to demonstrate. A Cochrane review of those trials of sufficient duration to detect the effects of intervention concluded that there was little evidence of success (14). It suggested that a more reliable evidence base is needed in order to determine the most cost-effective and health promoting strategies that have sustainable results and can be generalized to other situations.

As shown in the present report, there are several examples of interventions designed to prevent the rising levels of obesity – such as the school-based ‘Trim and Fit’ programme in Singapore and the ‘Agita Sao Paulo’ programme in Sao Paulo, Brazil. Favourable outcomes have been shown with small-scale interventions, modifying children’s TV watching behaviour and promoting consumption of healthier foods by establishing a price differential.

Although the beneficial results of such interventions may be detectable and significant, they are small compared with the size of the problem. Moreover, the improvements tend to decline after the intervention ends. It must be concluded that interventions at the family or school level will need to be matched by changes in the social and cultural context so that the benefits can be sustained and enhanced. Such prevention strategies will require a co-ordinated effort between the medical community, health administrators, teachers, parents, food producers and processors, retailers and caterers, advertisers and the media, recreation and sport planners, urban architects, city planners, politicians and legislators.

Public health requires multi-sectoral action

This report highlights the underlying social changes that have led to rising levels of obesity in both the adult and child populations. These underlying factors, as listed below, are often a part of, or a consequence of social development and urbanization. Such development based on economic growth to enhance consumption is generally regarded in a positive light and, especially in developing countries as they emerge from poverty, may be aspired to.

Examples of problematic social trends

- Increase in use of motorized transport, e.g. to school.
- Increase in traffic hazards for walkers and cyclists.
- Fall in opportunities for recreational physical activity.
- Increased sedentary recreation.
- Multiple TV channels around the clock.
- Greater quantities and variety of energy dense foods available.
- Rising levels of promotion and marketing of energy dense foods.
- More frequent and widespread food purchasing opportunities.
- More use of restaurants and fast food stores.
- Larger portions of food offering better ‘value’ for money.
- Increased frequency of eating occasions.
- Rising use of soft drinks to replace water, e.g. in schools.

Changes in these social trends may require increased awareness by countries of the health consequences of the pattern of consumption as the first step in a strategy to promote healthier diets and more active lives. Several authors (15–18) have suggested that efforts to prevent obesity should include measures involving a wide range of social actions, such as:

- public funding of quality physical education and sports facilities;
- the protection of open urban spaces, provision of safer pavements, parks, playgrounds and pedestrian zones, creation of more cycling paths;
• taxes on unhealthy foods and subsidies for the promotion of healthy, nutritious foods;
• dietary standards for school lunch programmes;
• elimination or displacement of soft drinks and confectionery from vending machines in schools and offering healthier choices (i.e., low-fat dairy products, fruits and vegetables);
• clear food labelling and controls on inconsistent health messages;
• controls on the political contributions given by the food industry;
• restrictions or bans on the advertising of foods to children;
• limits on other forms of marketing of foods to children;
• assessment of food industry initiatives to improve formulations and marketing strategies.

It is clear from these suggestions that policies and actions will be needed at a variety of levels, some local and individually based, some national or internationally based. All of them will require the support and involvement of departments across the broad range of government and may include education, social and welfare services, environment and planning, transport, food production and marketing, advertising and media, and international trading and standard-setting bodies.

Obesity prevention will involve work at all levels of the obesogenic environment. As Fig. 2 illustrates, attempts to improve the environment at one level, for example the school, may be undermined by a failure to improve the environment at another level, be it below in the home, or above in the social and cultural context involving food marketing and advertising, lost recreational facilities or unsafe streets.

Children are vulnerable to the social and environmental pressures that raise the risk of obesity. Although they can be encouraged to increase their self-control in the face of temptation, and although they can be given knowledge and skills to help understand the context of their choices, children cannot be expected to bear the full burden of responsibility for preventing excess weight gain. The prevention of childhood obesity requires:

• improving the family’s ability to support a child in making changes, which in turn needs support from the school and community, for example . . .
• ensuring the school has health-promoting policies on diet and physical activity, and that peer group beliefs are helping the child, which in turn requires that . . .
• the cultural norms, skills and traditional practices transmitted by the school are conducive to health promotion, and that the community provides a supportive environment, such as . . .
• neighbourhood policies for safe and secure streets and recreation facilities, and ensuring universal access to health-enhancing food supplies, which in turn requires that . . .
• authorities at municipal, and regional level are supporting such policies, e.g., for safe streets and improved food access through appropriate infrastructure, and that . . .
• national and international bodies that set standards and provide services are encouraging better public health, and commercial practices consistently promote healthy choices, which in turn may require . . .
• legislative and regulatory support to ensure that strategies for obesity reduction are fully resourced and implemented, and appropriate control measures are enforced, and that these are not contradicted by other government policies, and that . . .
• government and inter-governmental activities in all departments, including education, agriculture, transport, trade, the environment and social welfare policies are assessed for their health impact, and Government food purchases, e.g., for departmental staff, for the military, police, prisons, hospitals and schools and other agencies involved in public sector supply contracts are consistent with health and nutrition policies.

The present report is primarily addressed to health professionals, social scientists and others in a position to influence policy at national and international level, by providing a background to the problems and an indication of the policies needed to tackle them. It is written in the context of the World Health Organization’s work on the prevention of nutrition-related chronic diseases and the development of strategies to promote physical activity and healthy diets. The WHO’s consultation document (6) recommends the development of multi-sectoral strategies, with health
ministries convening with other ministries and stakeholders to develop relevant policies, programmes and regulations. The consultation document calls for positive action, such as measures to support the greater availability of nutrient dense foods, to reduce dependence on motorized transport, to increase access to recreation facilities and to ensure health information is widely available and easily understood, and health messages are relevant and consistent.

The WHO has acknowledged the restrictions placed on countries by international agreements, such as those that regulate trade and marketing practices. The WHO can offer a leadership role in prioritizing public health when negotiating these agreements. This depends upon political pressure, which in part depends upon leadership from the medical profession and from non-governmental organizations. The present report is designed to contribute to that process.

The International Obesity TaskForce calls upon the WHO to assist member countries to develop National Obesity Action Plans and to prioritize childhood obesity prevention within those plans. Examples of Action Plan priorities might be to:

- provide clear and consistent consumer information, e.g. on food labels;
- encourage food companies to provide lower energy, more nutritious foods marketed for children;
- develop criteria for advertising that promotes healthier eating;
- improve maternal nutrition and encourage breast-feeding of infants;
- design secure play facilities and safe local neighbourhoods;
- encourage schools to enact coherent food, nutrition and physical activity policies;
- encourage medical and health professionals to participate in the development of public health programmes.

The International Obesity TaskForce is committed to supporting the WHO in developing these priorities. Tackling childhood obesity will require much imagination and perspiration, but the world’s children deserve no less.
1. ASSSESSMENT OF OBESITY: WHICH CHILD IS FAT?

1.1. Measures of body fatness

Power et al. suggest that ‘an ideal measure of body fat should be accurate in its estimate of body fat; precise, with small measurement error; accessible, in terms of simplicity, cost and ease of use; acceptable to the subject; and well-documented, with published reference values’ (19). They further comment that ‘no existing measure satisfies all these criteria’.

Measurement of adiposity in children and adolescents occurs in a range of settings, using a range of methods. In this section, both direct and indirect methods for assessing and evaluating fatness are described and the strengths and weaknesses of these different methods used for population and clinical judgements are analysed.

1.1.1. Body fat: direct measures and derived estimates

Direct measures of body composition provide an estimation of total body fat mass and various components of fat-free mass. Such techniques include underwater weighing, magnetic resonance imaging (MRI), computerized axial tomography (CT or CAT) and dual energy X-ray absorptiometry (DEXA). The methods are used predominantly for research and in tertiary care settings, but may be used as a ‘gold standard’ to validate anthropometric measures of body fatness (20) (Table 1).

### Table 1 Methods for measuring body composition

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<tr>
<th>Procedure</th>
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<td>Underwater weighing (hydro-densitometry)</td>
<td>Fat has a lower density than lean tissue, and by measuring the density of the whole body the relative proportions of each component can be determined. If total body density and the specific densities of fat and fat-free mass are known, an equation can be generated for converting total body density to percentage body fat (20).</td>
<td>Requires a person to hold their breath underwater, and is unsuitable for use in young children or in older subjects who lack water confidence. There are theoretical concerns about the assumptions used to translate density measurements into estimates of fat mass and fat-free mass, both among normal children and the obese.</td>
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<td>Magnetic resonance imaging (MRI)</td>
<td>MRI provides a visual image of adipose tissue and non-fat tissue. Total body fat volume, total fat mass and percentage fat mass can be estimated.</td>
<td>MRI can accurately and reliably distinguish intra-abdominal from subcutaneous fat. MRI is expensive, time consuming and must be performed in a major medical facility. The procedure takes approximately 20 min, and requires the subject to lie still, enclosed in a scanner, and may be unsuitable for young children.</td>
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<td>Computerized tomography (CT)</td>
<td>CT scans produce high-resolution X-ray-derived images and can identify small deposits of adipose tissue. Total and regional body fat can be calculated, as well as percentage body fat.</td>
<td>The procedure allows intra-abdominal and subcutaneous fat to be quantified with a high degree of accuracy and reliability. The equipment is expensive and must be operated by a skilled technician. The procedure involves significant radiation exposure, takes 20 min and requires the subject to lie still within the scanner, so is unsuitable for routine use in children unless clinically indicated.</td>
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<td>Dual-Energy X-ray Absorptiometry (DEXA)</td>
<td>DEXA is based on the principle that transmitted X-rays at two energy levels are differentially attenuated by bone mineral tissue and soft tissue, and the soft tissue component is subdivided into fat and lean tissue by using experimentally derived calibration equations (21).</td>
<td>DEXA cannot distinguish between intra-abdominal and subcutaneous fat. It has a high correlation with CT scan data in determining total fat mass (22). The procedure delivers lower radiation exposure than CT and is thus more suitable for use in children and adolescents. However, the test must be performed in a major medical facility with the DEXA equipment, the equipment is expensive and must be operated by a skilled technician, and the procedure may take up to 20 min and requires a very cooperative subject, therefore making it unsuitable for children aged less than 6 years. DEXA has not been fully evaluated in healthy child or adolescent populations or in very obese people.</td>
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<td>Bioelectrical impedance analysis (BIA)</td>
<td>BIA is not strictly a direct measure of body composition, being based on the relation between the volume of a conductor (the body), the conductor’s length (height) and its electrical impedance (23). BIA assumes fat mass is anhydrous and that conductivity reflects fat-free mass. Prediction equations estimate the fat-free mass from the measured impedance and, by subtraction, the fat mass.</td>
<td>BIA measurements can be taken quickly and inexpensively, it is relatively non-invasive and has high inter- and intra-observer reliability. However, it requires equations specific to the instrument used and for the population under investigation, and the measurement may vary with hydration status and ethnic status (see Wabitsch et al. (24)). Although gaining acceptance in a range of settings, the limitations of BIA are sometimes overlooked.</td>
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<td>Air-displacement plethysmography</td>
<td>A subject’s volume is determined indirectly by measuring the volume of air the subject displaces when sitting inside an enclosed chamber. Adjustment for thoracic gas volume is made. Once body volume and mass are known, the principles of densitometry are applied to estimate percentage body fat.</td>
<td>Air-displacement plethysmography measurements are comfortable, relatively quick, non-invasive and can accommodate a wide range of body types. Subjects should be reasonably cooperative (for accurate measurement the subject should breathe through a tube and wear a nose clip) and hence the technique may be unsuitable for younger children. Again, there are theoretical concerns about the assumptions used to calculate body fat (25).</td>
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1.1.2. Anthropometric measures of relative fatness
Among the anthropometric measures of relative adiposity or fatness are waist, hip and other girth measurements, skinfold thickness and indices derived from measured height and weight such as Quetelet’s index (BMI or \( W/H^2 \)), the ponderal index (\( W/H^4 \)) and similar formulae. All anthropometric measurements rely to some extent on the skill of the measurer, and their relative accuracy as a measure of adiposity must be validated against a ‘gold standard’ measure of adiposity (Table 2).

Table 2: Indirect methods for estimating body composition

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<th>Procedure</th>
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<td>Weight and weight-for-height</td>
<td>Total body weight can be recorded and compared with reference standards based on a child’s age. Low weight-for-age is a widely used marker of malnutrition for younger children. However, weight is correlated with height, and reference standards based on weight-for-height provide a more accurate measure of under-or overweight, and take account of possibly confounding from inadequate linear growth (stunting) when assessing nutritional status.</td>
<td>Growth charts are based on standard reference populations (usually the US National Center for Health Statistics reference population, although these may under-represent the growth patterns of breast-fed children (26, 27) and may need to be revised (28). Weight-for-height charts are inaccurate beyond the age of around 10–11 years and the measure is not useful in older children and adolescents (29).</td>
<td>Weight and height (or length) are relatively easy to obtain, although they tend to move more accurately if taken by a trained person. Weight should be taken with the child in light, indoor clothing.</td>
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<td>Body mass index (BMI)</td>
<td>BMI is defined as weight (kg) / height squared (m(^2)), and is widely used as an index of relative adiposity among children, adolescents and adults. Among adults, the WHO recommends that a person with a BMI of 25 kg m(^{-2}) or above is classified overweight, while one with a BMI of 30 kg m(^{-2}) or above is classified obese (16) although revisions of these guidelines are being proposed for certain populations (30). For children, various cut-off criteria have been proposed based on reference populations and different statistical approaches (see discussion below).</td>
<td>BMI has been compared with dual-energy X-ray absorptiometry (DEXA) in children and adolescents aged 4–20 years (31). BMI had a true positive rate of 0.67, and a false positive rate of 0.06 for predicting a high percentage of total body fat. Sardinha et al. (32) reported a true positive rate of 0.83 for 10–11 year olds, 0.67 for 12–13 year olds and 0.77 for 14–15 year olds, while the false positive rate ranged from 0.03 in 12–13 year olds to 0.13 in 10–11 year olds. Therefore, although some overweight children would be wrongly classified as being of normal weight when using BMI as a screening test, few children would be classified as overweight if they were not. Correlation coefficients between BMI and DEXA range from 0.50 in a study of 7–17-year-old white males, to 0.83 in a study of 7–17-year-old girls (33). A study of 198 white boys and girls aged 5–19 years found a correlation of 0.85 between BMI and total body fat measured with DEXA (34). (See also the discussion of ‘other measures’, below.)</td>
<td>BMI is more accurate when height and weight are measured by a trained person rather than self-reported. Measurement of height and weight has a high subject acceptance, which is particularly important for adolescents who may be reluctant to undress (measures are normally taken in light clothing, without shoes). There is low observer error, low measurement error and good reliability and validity. However, BMI may not be a sensitive measure of body fatness in people who are particularly short, tall or have an unusual body fat distribution, and may misclassify people with highly developed muscles. Hence two people with the same amount of body fat can have quite different BMIs (32). There may also be racial differences in the relationship between the true proportion of body fat and BMI (35).</td>
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<td>Waist circumference and Waist-to-hip ratio (WHR)</td>
<td>Waist circumference is an indirect measure of central adiposity. Central adiposity is strongly correlated with risk for cardiovascular disease in adults (36) and an adverse lipid profile and hyperinsulinaemia in children (37). Waist circumference is measured at the minimum circumference between the iliac crest and the rib cage using an anthropometric tape. W-to-hip ratio has been used among adults to identify people with high central adiposity. Waist circumference is measured as above and hip circumference is measured at the maximum protuberance of the buttocks. The ratio is then calculated.</td>
<td>In young people aged 3-19 years, the correlation between waist circumference and DEXA of trunk fat were 0.83 for girls and 0.84 for boys. In addition, children’s waist circumference correlates well with CT scan as a measure of subcutaneous abdominal adipose tissue (( r = 0.93 )), and fairly well with intra-abdominal adipose tissue (( r = 0.84 )) (22). Waist-to-hip ratios are less well correlated with trunk fat measures using DEXA (38).</td>
<td>Waist and hip circumferences are easy to measure with simple, low-cost equipment, have low observer error, offer good reliability, validity and low measurement error. However, there are no accepted cut-off values for the classification of overweight and obesity based on these measures, and there have been few studies of the relation between central adiposity and the metabolic disturbances associated with excess visceral fat among children and adolescents. Waist circumference and hip circumference are highly age dependent, and it is not recommended to use the ratio between them without first considering each measure separately (19).</td>
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1.2. Definitions of ‘overweight’ and ‘obesity’ in young people

The primary purposes for defining overweight and obesity are to predict health risks and to provide comparisons between populations. Faced with a continuous distribution, criteria need to be created that define where cut-off points should occur that best fulfil these purposes. For practical reasons, the definitions have usually been based on anthropometry, with waist circumference and BMI being the most widely used both clinically and in population studies.

1.2.1. Weight for height

Although not validated against health criteria, weight for height measurements have become a common means of assessing populations of children, especially those aged under 5 years, and are used to define both under- and over-nutrition. Low weight for height is termed thinness, and very low weight for height is termed wasting, usually under 5 years, and are used to define both under- and overweight. A high weight for height is termed overweight and very high weight for height is termed obese. The use of weight for height has the advantage of not requiring knowledge of the child’s age, which may be hard to assess in less developed areas, but it should not be used as a substitute for height for age, or weight for age, as all three measures reflect different biological processes (see Table 16 in reference 42).

In 1995, the use of weight for height was recommended by WHO for children below the age of 10 years, but a WHO review found that use of the US-based National Center for Health Statistics should not be recommended especially as it did not take account of differences between breast-fed and non-breastfed children (42). The WHO may consider new reference standards based on multinational studies of breast-fed children showing healthy growth (28).

The weight for height score is plotted on a chart based on a standard reference population, which gives a Z-score based on the difference between the observed value and the median reference value of a population, standardized against the standard deviation of the reference population. Thus a Z-score of 0 is equivalent to the median or 50th centile value, a Z-score of +2.00 is approximately equivalent to the 98th centile and a Z-score of +2.85 is >99th centile. The use of a weight for height Z-score (WHZ) allows a more detailed statistical description of an individ-

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<tr>
<td>Skin-fold</td>
<td>Skin-fold thickness can be measured at different sites on the body (e.g.</td>
<td>Children’s abdominal skin-fold thickness correlates well (r = 0.88) with</td>
<td>Skin-fold thickness uses simple equipment and offers only a moderate</td>
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<td>thickness</td>
<td>triceps, subscapular) using skin-fold calipers. Prediction equations can</td>
<td>visceral adipose tissue as measured by CT scan or MRI (39). Triceps skin-</td>
<td>respondent burden, and has the potential to determine total body fat and</td>
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<td></td>
<td>then be used to estimate fat mass and percentage fat from the skin-fold</td>
<td>fold thickness shows a sensitivity of 0.79 in 10–11 year olds, 0.78 in</td>
<td>regional fat distribution. However, skin-fold thickness varies with age,</td>
</tr>
<tr>
<td></td>
<td>measurements. New methods for measuring skin fold using portable echography</td>
<td>12–13 year olds and 0.87 in 14–15 year olds when compared with DEXA-in</td>
<td>sex and race, and the equations relating skin-fold thickness at several</td>
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<td>equipment are under development.</td>
<td>measuring obesity (&gt;30% body fat) (32). The corresponding false positive</td>
<td>sites to total body fat need to be validated for each population.</td>
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<td>rates were 0.03 and 0.07. However, triceps measures may be less indicative</td>
<td>Measurement requires training and intra- and inter-observer reliability</td>
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<td></td>
<td>of central obesity among children in developing countries (40) compared</td>
<td>is poor (23). In very obese individuals the measurement of triceps skin-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>with a US-based population.</td>
<td>fold or other skin-fold thicknesses may not be possible. The relationship</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>with metabolic problems is unclear.</td>
</tr>
<tr>
<td>Other</td>
<td>Various alternatives to the weight-to-height ratio have been developed</td>
<td>Both BMI and the ponderal index are intended to remove the height element</td>
<td>As with BMI, height and weight are more accurate when measured by a</td>
</tr>
<tr>
<td>anthropometric</td>
<td>examining different powers of N in the formula weight/height^N, such as the</td>
<td>from the estimation of relative weight, but in studies of child populations</td>
<td>trained person rather than self-</td>
</tr>
<tr>
<td>measures</td>
<td>ponderal index (w h^N). ‘N’ is sometimes referred to as the Benn index.</td>
<td>there is evidence that both measures show some residual association with</td>
<td>reported. Such measures have high subject acceptance and there is low</td>
</tr>
<tr>
<td></td>
<td>Another measure, the conicity index, is defined as waist circumference/(0.109</td>
<td>height. An analysis of the Benn index, related to height at various stages</td>
<td>observer error, low measurement error and good reliability and validity.</td>
</tr>
<tr>
<td></td>
<td>x square root of weight/height).</td>
<td>in childhood, showed that N lay just below 3.0 for children aged 6 years,</td>
<td>However, none of these indices is</td>
</tr>
<tr>
<td></td>
<td></td>
<td>rising to 3.5 for children aged around 10 and fell to around 2.0 by age 18</td>
<td>widely used at present.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>(41). Thus the use of BMI (w h^N) tends to give taller children a greater</td>
<td></td>
</tr>
<tr>
<td></td>
<td></td>
<td>BMI than shorter children when their true relative weights are equal.</td>
<td></td>
</tr>
</tbody>
</table>

Table 2 Continued

<table>
<thead>
<tr>
<th>Procedure</th>
<th>Description</th>
<th>Validation</th>
<th>Comments</th>
</tr>
</thead>
<tbody>
<tr>
<td>Skin-fold</td>
<td>Skin-fold thickness can be measured at different sites on the body (e.g.</td>
<td>Children’s abdominal skin-fold thickness correlates well (r = 0.88) with</td>
<td>Skin-fold thickness uses simple equipment and offers only a moderate</td>
</tr>
<tr>
<td>thickness</td>
<td>triceps, subscapular) using skin-fold calipers. Prediction equations can</td>
<td>visceral adipose tissue as measured by CT scan or MRI (39). Triceps skin-</td>
<td>respondent burden, and has the potential to determine total body fat and</td>
</tr>
<tr>
<td></td>
<td>then be used to estimate fat mass and percentage fat from the skin-fold</td>
<td>fold thickness shows a sensitivity of 0.79 in 10–11 year olds, 0.78 in</td>
<td>regional fat distribution. However, skin-fold thickness varies with age,</td>
</tr>
<tr>
<td></td>
<td>measurements. New methods for measuring skin fold using portable echography</td>
<td>12–13 year olds and 0.87 in 14–15 year olds when compared with DEXA-in</td>
<td>sex and race, and the equations relating skin-fold thickness at several</td>
</tr>
<tr>
<td></td>
<td>equipment are under development.</td>
<td>measuring obesity (&gt;30% body fat) (32). The corresponding false positive</td>
<td>sites to total body fat need to be validated for each population.</td>
</tr>
<tr>
<td></td>
<td></td>
<td>rates were 0.03 and 0.07. However, triceps measures may be less indicative</td>
<td>Measurement requires training and intra- and inter-observer reliability</td>
</tr>
<tr>
<td></td>
<td></td>
<td>of central obesity among children in developing countries (40) compared</td>
<td>is poor (23). In very obese individuals the measurement of triceps skin-</td>
</tr>
<tr>
<td></td>
<td></td>
<td>with a US-based population.</td>
<td>fold or other skin-fold thicknesses may not be possible. The relationship</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>with metabolic problems is unclear.</td>
</tr>
<tr>
<td></td>
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<td></td>
<td>As with BMI, height and weight are more accurate when measured by a</td>
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<td></td>
<td></td>
<td></td>
<td>trained person rather than self-</td>
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<tr>
<td></td>
<td></td>
<td></td>
<td>reported. Such measures have high subject acceptance and there is low</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>observer error, low measurement error and good reliability and validity.</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>However, none of these indices is</td>
</tr>
<tr>
<td></td>
<td></td>
<td></td>
<td>widely used at present.</td>
</tr>
</tbody>
</table>

1.2. Definitions of ‘overweight’ and ‘obesity’ in young people

The primary purposes for defining overweight and obesity are to predict health risks and to provide comparisons between populations. Faced with a continuous distribution, criteria need to be created that define where cut-off points should occur that best fulfil these purposes. For practical reasons, the definitions have usually been based on anthropometry, with waist circumference and BMI being the most widely used both clinically and in population studies.

1.2.1. Weight for height

Although not validated against health criteria, weight for height measurements have become a common means of assessing populations of children, especially those aged under 5 years, and are used to define both under- and over-nutrition. Low weight for height is termed thinness, and very low weight for height is termed wasting, usually found as a consequence of acute starvation and/or disease. A high weight for height is termed overweight and very high weight for height is termed obese. The use of weight for height has the advantage of not requiring knowledge of the child’s age, which may be hard to assess in less developed areas, but it should not be used as a substitute for height for age, or weight for age, as all three measures reflect different biological processes (see Table 16 in reference 42).

In 1995, the use of weight for height was recommended by WHO for children below the age of 10 years, but a WHO review found that use of the US-based National Center for Health Statistics should not be recommended especially as it did not take account of differences between breast-fed and non-breastfed children (42). The WHO may consider new reference standards based on multinational studies of breast-fed children showing healthy growth (28).

The weight for height score is plotted on a chart based on a standard reference population, which gives a Z-score based on the difference between the observed value and the median reference value of a population, standardized against the standard deviation of the reference population. Thus a Z-score of 0 is equivalent to the median or 50th centile value, a Z-score of +2.00 is approximately equivalent to the 98th centile and a Z-score of +2.85 is >99th centile. The use of a weight for height Z-score (WHZ) allows a more detailed statistical description of an individ-

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ual or a population, and comparison between populations can also be readily made and trends over time can be described. Besides the difficulty in choosing an appropriate reference population, however, WHZ-scores require suitable statistical skills or software programmes. In terms of defining non-overweight, overweight and obese the categories are based only on statistical convenience (e.g. WHZ > 2) rather than a known health risk.

1.2.2. Waist circumference

In a large-scale epidemiological study of young people aged 5–17 years, Freedman et al. showed that central fat distribution (particularly as assessed by waist circumference) was associated with an adverse lipid profile and hyperinsulinaemia (37). A high waist circumference has also been shown to track well into adulthood (22). Although waist circumference percentile charts have been described (e.g. for the UK (43)), appropriate cut-off points for defining high or low health risks have not been identified.

Waist circumference may be useful in clinical practice as a means of determining a child or adolescent’s response to weight control measures. In epidemiological studies, it may be used to characterize a population in terms of abdominal fat distribution and to determine the prevalence of risk factors. At present, however, waist circumference cannot be used to categorize a child as being at a high or low risk.

1.2.3. BMI

As suggested in Table 2 above, BMI is significantly associated with relative fatness in childhood and adolescence, and is the most convenient way of measuring relative adiposity (44).

BMI for age reference charts and BMI for age percentiles.

BMI varies with age and gender. It typically rises during the first months after birth, falls after the first year and rises again around the sixth year of life: this second rise is sometimes referred to as ‘the adiposity rebound’. (For examples of childhood BMI curves by age and gender, see Figure 28 below.) A given value of BMI therefore needs to be evaluated against age- and gender-specific reference values. Several countries, including France, the UK, Singapore, Sweden, Denmark and the Netherlands, have developed their own BMI-for-age gender-specific reference charts using local data. In the USA, reference values published by Must et al. (45) derived from US survey data in the early 1970s, have been widely used and were recommended for older children (aged 9 years or more) by a WHO expert committee in 1995 (42). More recently, the US National Center for Health Statistics (NCHS) has produced reference charts based on data from five national health examinations from 1963–1994 (46), although to avoid an upward shift of the weight and BMI curves, data from the most recent survey were excluded for children over the age of six years (47).

The advantage of using BMI-for-age charts is that a child can be described as being above or below certain centile lines (for example the 85th or 90th centile), which can be useful in a clinical setting. Data, however, are usually derived from a single reference population, and classifying an individual as overweight or obese assumes that the individual is comparable to that reference population. Furthermore, clinicians may wrongly interpret the centiles as representing an ideal population, when the figures may in fact come from a reference population with a high prevalence of obesity, such as the USA NCHS data.

The NCHS documentation (46) recommends that those children with a BMI greater than or equal to the 95th percentile be classified as ‘overweight’ and those children with a BMI between the 85th and 95th percentile be classified as ‘at risk of overweight’. In some papers, US children at or above the 95th centile are referred to as ‘obese’ (18) and in others ‘obesity’ refers to US children above the 85th centile (48).

BMI for age Z-scores. As with the use of weight-for-height measures compared with standard reference populations, BMI can be compared with a reference data set and reported as Z-scores. A BMI Z-score is calculated as follows:

\[
\text{Z-score} = \frac{\text{observed value}}{\text{standard deviation}} - \frac{\text{median reference value of a population}}{\text{standard deviation of reference population}}
\]

A Z-score of 0 is equivalent to the median or 50th centile value, a Z-score of +1.00 is approximately equivalent to the 84th centile, a Z-score of +2.00 is approximately equivalent to the 98th centile and a Z-score of +2.85 is >99th centile. As with other measures, BMI Z-scores can be used to compare an individual or specified population against a reference population. BMI for age Z-scores, however, require suitable statistical skills or software programmes, there is difficulty in choosing an appropriate reference population, and there are only arbitrary cut-off points for categorizing into non-overweight, overweight and obese.

BMI based on adult cut-off points. An expert committee convened by the International Obesity TaskForce in 1999 determined that although BMI was not ideal as a measure of adiposity, it had been validated against other, more direct measures of body fatness and may therefore be used to define overweight and obesity in children and adolescents (44). As it is not clear at which BMI level adverse health risk factors increase in children, the group recommended cut-offs based on age specific values that project to the adult cut-offs of 25 kg m\(^{-2}\) for overweight and 30 kg m\(^{-2}\) for obesity. Using data from six different reference populations (Great
Britain, Brazil, the Netherlands, Hong Kong, Singapore and the USA) Cole et al. (47) derived centile curves that passed through the points of 25 kg m\(^{-2}\) and 30 kg m\(^{-2}\) at age 18 years. These provide age and gender specific BMI cut offs to define overweight and obesity, corresponding to the adult cut off points for overweight and obesity.

The tables developed by Cole et al. (reproduced in Table 3) are useful for epidemiological research in that children and adolescents can be categorized as non-overweight, overweight or obese using a single standard tool. The cut-off points were developed using several data sets, therefore they represent an international reference that can be used to compare populations world-wide. The authors, however, acknowledge that the reference data set may not adequately represent non-Western populations.

There are differences in body composition across adult ethnic groups, with one study in whites and Asians showing differences of 2–3 BMI units in adults with the same body fat composition (35). Central obesity combined with thin limbs appears to feature in some Indian populations, giving a higher health risk for a given BMI (49). Studies in the USA have found that African American, Mexican American and Mohawk Indian children carry more central fat than white children (39). In the UK there is some evidence that children of South Asian ethnicity are at greater risk of central adiposity (50,51).

The implications of these studies is that the adult cut-offs of BMI 25 and 30 may not be universally applicable, and hence the use of those cut-offs to define the range of childhood BMIs that correspond to them, may also be inappropriate for some child populations. A WHO-sponsored seminar on Asian obesity risks suggested that overweight and obesity might be defined at BMI 23+ and 25+ respectively (52). A study of Malaysians has shown that body fat levels are higher than those found in Caucasians, and that overweight and obesity should be defined at BMI 23+ and 27+, respectively (30). For Pacific Islanders, overweight and obesity may be better defined at 26+ and 32+ respectively (53).

Lastly, a proposal that child BMI cut-offs should be adjusted to take account of pubertal stage of development in addition to age and gender has been made (54), but there appears to be no superiority compared with using BMI based on age and gender alone in terms of predicting actual percentage body fat assessed by dual-energy X-ray absorptiometry (55).

In summary, the reference cut-offs from Cole et al. are suitable for research use and for monitoring and evaluating changes in populations, because the cut-offs provide a standard benchmark against which all population groups can be compared and trends assessed. In terms of defining groups at special risk of health problems due to excess weight, the cut-off points may need to be adjusted to account for local factors.

### Table 3: International cut-off points for body mass index for overweight and obesity by sex between 2 and 18 years

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Body mass index</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>25 kg m(^{-2})</td>
</tr>
<tr>
<td></td>
<td>Males</td>
</tr>
<tr>
<td>2</td>
<td>18.41</td>
</tr>
<tr>
<td>2.5</td>
<td>18.13</td>
</tr>
<tr>
<td>3</td>
<td>17.89</td>
</tr>
<tr>
<td>3.5</td>
<td>17.69</td>
</tr>
<tr>
<td>4</td>
<td>17.55</td>
</tr>
<tr>
<td>4.5</td>
<td>17.47</td>
</tr>
<tr>
<td>5</td>
<td>17.42</td>
</tr>
<tr>
<td>5.5</td>
<td>17.45</td>
</tr>
<tr>
<td>6</td>
<td>17.55</td>
</tr>
<tr>
<td>6.5</td>
<td>17.71</td>
</tr>
<tr>
<td>7</td>
<td>17.92</td>
</tr>
<tr>
<td>7.5</td>
<td>18.16</td>
</tr>
<tr>
<td>8</td>
<td>18.44</td>
</tr>
<tr>
<td>8.5</td>
<td>18.76</td>
</tr>
<tr>
<td>9</td>
<td>19.10</td>
</tr>
<tr>
<td>9.5</td>
<td>19.46</td>
</tr>
<tr>
<td>10</td>
<td>19.84</td>
</tr>
<tr>
<td>10.5</td>
<td>20.20</td>
</tr>
<tr>
<td>11</td>
<td>20.55</td>
</tr>
<tr>
<td>11.5</td>
<td>20.89</td>
</tr>
<tr>
<td>12</td>
<td>21.22</td>
</tr>
<tr>
<td>12.5</td>
<td>21.56</td>
</tr>
<tr>
<td>13</td>
<td>21.91</td>
</tr>
<tr>
<td>13.5</td>
<td>22.27</td>
</tr>
<tr>
<td>14</td>
<td>22.62</td>
</tr>
<tr>
<td>14.5</td>
<td>22.96</td>
</tr>
<tr>
<td>15</td>
<td>23.29</td>
</tr>
<tr>
<td>15.5</td>
<td>23.60</td>
</tr>
<tr>
<td>16</td>
<td>23.90</td>
</tr>
<tr>
<td>16.5</td>
<td>24.19</td>
</tr>
<tr>
<td>17</td>
<td>24.46</td>
</tr>
<tr>
<td>17.5</td>
<td>24.73</td>
</tr>
<tr>
<td>18</td>
<td>25</td>
</tr>
</tbody>
</table>

Source: Cole et al. (47).

In clinical practice, the variations found in body fat mass and non-fat mass for a given bodyweight may make any judgement based on weight (adjusted for height and/or for age) unreliable as an estimate of an individual’s actual body fat. At higher levels, BMI and the BMI cut-offs may be helpful in informing a clinical judgement, but at levels near the norm additional criteria may be needed. For clinical assessment, more direct measures, such as bio-impedance, as well as indirect measures such as waist circumference, are sometimes used.

### Comparisons of BMI cut-offs

Several studies have compared the US NHANES criteria for defining overweight or obesity using age- and gender-specific 85\(^{th}\) and 95\(^{th}\) centile cut-offs with those of the more recent US Centres for Disease Control (CDC) using similar percentile cut-offs, and the
IOTF alternative set of cut-offs based on centiles passing through BMI 25 and BMI 30 at age 18. Using the NHANES III data, Flegal et al. (56) show that the different methods give approximately similar results, but with some significant discrepancies especially among younger children (see Table 4).

In a study that examined data from children in national surveys in the USA, Russia and China, a comparison of three different methods for defining cut-offs found that the methods produced similar estimates for the prevalence of overweight (>85th centile US references and >BMI 25 equivalent for the IOTF method), but that estimates of obesity (>95th centile US references and >BMI 30 equivalent IOTF method) showed some differences (57). For adolescents the IOTF method tended to give slightly lower estimates for the prevalence of obesity, while for younger children the IOTF method gave more significantly lower estimates for the prevalence of obesity. The IOTF method also gave significantly lower estimates of obesity prevalence compared with figures based on weight-for-height Z score >2, for children aged 6–9 years (older children were not assessed on this score).

In general, the IOTF method appears to give a more conservative view of the extent of overweight and obesity among pediatric populations compared with methods based on the 85th and 95th centiles of US-based reference populations or based on the use of weight-for-height Z scores.

BMI cut-offs based on alternative criteria. The IOTF BMI cut-offs discussed above are based on adult BMI cut-offs extrapolated back into childhood. Alternative approaches can be suggested, for example that child BMI cut-offs can be defined in relation to health in childhood. This approach has been developed in Taiwan, where cut-offs have been estimated using definitions of fit or unfit according to a set of physical fitness criteria (58). Using data from nearly a million young people recorded in a nation-wide fitness survey, those individuals deemed physically fit (i.e. excluding those in the bottom quartile on the fitness tests) at age 18 showed 85th and 95th centile points at around 23 kg m⁻² and 25 kg m⁻², respectively. These values tie in with adult recommendations for Asian populations of BMI 23 and 25, reflecting overweight and obesity cut-off points respectively.

The development of BMI norms for children based on the BMIs of standardized ‘fit’ populations opens new possibilities for defining overweight and obese children, and needs further exploration.

Table 4  Comparison of prevalence rates of overweight and obesity using different criteria

<table>
<thead>
<tr>
<th></th>
<th>Age 6–8 years</th>
<th>Age 12–14 years</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Boys</td>
<td>Girls</td>
</tr>
<tr>
<td>Overweight</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NHANES/WHO &gt;85th</td>
<td>25%</td>
<td>31%</td>
</tr>
<tr>
<td>CDC &gt;85%</td>
<td>23%</td>
<td>23%</td>
</tr>
<tr>
<td>IOTF &gt;BMI 25 equivalent</td>
<td>18%</td>
<td>23%</td>
</tr>
<tr>
<td>Obese</td>
<td></td>
<td></td>
</tr>
<tr>
<td>NHANES/WHO &gt;95th</td>
<td>13%</td>
<td>17%</td>
</tr>
<tr>
<td>CDC &gt;95%</td>
<td>11%</td>
<td>11%</td>
</tr>
<tr>
<td>IOTF &gt;BMI 30 equivalent</td>
<td>8%</td>
<td>8%</td>
</tr>
</tbody>
</table>

Source: Flegal et al. (56).

Recommendations for the definition of ‘overweight’ and ‘obesity’

• Body mass index should be used as the main measure of overweight and obesity in childhood and adolescence for survey purposes.
• Research studies involving epidemiological or clinical data should ensure that BMI for age is expressed as a mean and standard deviation for each gender; and prevalence is estimated by using cut-off points from a clearly established reference. This will allow international comparison of data.
• Researchers should be encouraged to collect data on waist circumference in childhood and adolescence when performing epidemiological or clinical studies.
• Further research is needed to validate the BMI-for-age or waist-for-age cut-off points associated with health risks in childhood and adolescence.
• Further research is needed into the effect of ethnicity on the interpretation of the definitions of ‘overweight’ and ‘obesity’.
• Further work is needed in establishing clinical definitions of ‘overweight’ and ‘obesity’ that are congruent with research definitions.
2. PREVALENCE AND TRENDS IN CHILDHOOD OBESITY

Our understanding of the global circumstances surrounding obesity in children and adolescents is limited due to the lack of comparable representative data from different countries and, in particular, due to the use of varying criteria for defining obesity among different countries and researchers. This methodological problem of consistency between classifications of childhood obesity is the major obstacle in studying global secular trends for younger age groups (16). Here, we review the published data and some additional unpublished data collected by the International Obesity TaskForce in collaboration with regional task forces and members of the International Association for the Study of Obesity.

It should be noted that there are several constraints on the use of survey data for interpreting trends in obesity in children and adolescents.

- **Sampling issues.** Some of the results presented in this section are based on national representative surveys, while others are based on smaller surveys that do not represent national populations.
- **Sexual maturation.** Sexual maturation influences body fatness: fat gain occurs in both boys and girls early in adolescence, then ceases and may even temporarily reverse in boys but continues throughout adolescence in girls (see later in this report). There are large intra- and inter-population variations in the patterns of sexual maturation (57,59).
- **Secular trends in growth and development.** Over recent decades, children world-wide have become taller, they mature earlier, and in some cases, become heavier for a given age. These trends have affected some populations more than others, and at different rates of change. Comparisons between populations should take these secular trends into account.
- **Stunting.** Stunted children are more likely to be overweight in countries undergoing a rapid nutrition transition (60) (see later in this report). This is of particular significance when examining the trends in obesity in developing countries, where the secular trends in linear growth continue and the prevalence of stunting has declined.
- **Adiposity rebound (see sections 4.2.2 and 4.4.2, below).** Considerable differences may exist in the timing and patterns of adiposity rebound between populations—in particular between populations in industrialized and in developing countries. This may affect the estimate of obesity prevalence for children from developing countries at around the age of adiposity rebound when using the so-called international references developed using data collected in wealthy societies, such as the NHANES and WHO references. The IOTF references are based on six countries: USA, Netherlands, Great Britain, Brazil, Hong Kong and Singapore.
- **Measurement errors.** Data collected in different studies and countries and over time may not have the same quality.

The potential influence of measurement errors should not be ignored. All these factors may influence the observed secular trends.

Nonetheless, survey material is an invaluable source of data for understanding the rising epidemic of childhood obesity. The continued collection of such data is essential, and several recommendations can be made to improve the value of the data.

- The international reference charts for making international comparisons and for monitoring the secular trends in childhood obesity need to be continually refined and evaluated, especially in developing countries where children and adolescents have very different growth and development patterns, which occur in a different cultural context, compared with their counterparts in industrialized countries.
- Nationally representative data in developing countries is particularly needed, especially for older children (>5 years old) and adolescents, and these data will be especially valuable.

**Note**

In the descriptions below it will be seen that some surveys have used definitions based on 85th and 95th centiles. This means that 15% and 5% of children would be expected to have weights above the 85th and 95th centiles respectively if the sampled population is identical to the reference population.

Similarly, statements apply to the use of definitions of overweight (and underweight) based on standardized deviation (Z) scores. For a normal Gaussian distribution, a Z score of 1 is approximately equivalent to the 16th (lower) and 84th (upper) centile, while a Z score of 2 is approximately equivalent to the 2nd (lower) or 98th (upper) centile. Thus 16% of children would be expected to have a Z score of greater than +1, and 2% of children would be expected to have a Z score greater than +2, if the sampled population is identical to the reference population.

A few surveys define overweight and obesity respectively as being greater than 120% and 140% above an ideal or standard reference point. For BMIs in children this does not relate to a specified centile, but can be read as approximately equivalent to the 85th and 95th centiles, respectively, of that standard reference group.

The IOF definitions, as noted above, do not relate to specified centiles but to age- and gender-adjusted BMIs equivalent of BMI 25 (overweight) and BMI 30 (obese) at age 18.
valuable for monitoring trends in obesity during periods of economic change and urbanization.

- Collection of longitudinal data, which can be used to track the development of obesity and evaluate interventions, needs to be encouraged. Longitudinal studies may prove particularly valuable for examining the social, environmental, behavioural and biological factors that may contribute to the secular trends in childhood obesity.

2.1. Global secular trends and prevalence of obesity in children and adolescents

Representative data for examining the problem of childhood obesity have been collected in many industrialized countries, especially in North America and Europe as well as in a number of developing countries, although for most developing countries the data are more limited, especially data on older children (>5 years) and adolescents. Nevertheless, data collected in national and local surveys from different parts of the world provide useful insights into the global obesity situation among young people. These data are listed in Appendix 1, Tables A1(1) to A1(5).

In summary, the available data suggest the following:

(i) Global prevalence is unequally distributed

Taken overall, the data available from surveys of young people aged 5–17 years, collated for the WHO Global Burden of Disease report and extrapolated to countries where no data are available, indicates the prevalence of overweight (including obesity) to be approximately 10% in this age range, and the prevalence of obesity to be 2–3% (1). This global average reflects a wide range of prevalence levels, with the prevalence of overweight in Africa and Asia averaging well below 10% and in the Americas and Europe above 20% (Figs 3 (boys) & 4 (girls)).

Two recent comprehensive studies examined obesity in pre-school children (61,62), and one estimated that the overall prevalence of obesity (defined as WHZ > 2) was 3.3% in developing countries among this younger age group in 1995 (61). For those countries with data recorded during the 1990s, the figures for obesity (WHZ > 2) among children under 5 years old are shown in Fig. 5.

(ii) Childhood overweight is rising rapidly

The prevalence of excess weight among children is increasing in both developed and developing countries, but at very different speeds and in different patterns. North America and some European countries have the highest prevalence levels, and have shown high year-on-year increases in prevalence. As can be seen from Fig. 6, prevalence rates are increasing rapidly, for example at approximately 0.5%
increase per annum in the USA and Brazil, and nearly 1% increase per annum in Canada, Australia and the UK, during the last two decades.

(iii) Overweight is high among the poor in rich countries, and the rich in poorer countries

In industrialized countries it is children in lower socio-economic groups who are at greatest risk. In contrast, developing countries show obesity to be more prevalent among higher income sectors of the population, and among urban populations rather than rural ones (Figs 7 & 8).

A number of developing countries undergoing rapid socio-economic and nutrition transitions are experiencing a shift from under- to over-nutrition problems, and may experience a double burden of malnutrition and obesity. For example, in Brazil between 1974 and 1997, the prevalence of overweight and obesity (IOTF definitions) among young people aged 6–17 years more than tripled (increasing from 4.1% to 13.9%), while the prevalence of underweight (<5th centile NHANES-I) decreased from 14.8% to 8.6% (67). Furthermore, among urban children
in China within a 6-year period between 1991 and 1997, the prevalence of overweight and obesity increased from 7.7% to 12.4%, while the prevalence of underweight dropped from 12.4% to 10.0%. In rural areas these changes were far less marked, with 1997 figures for overweight (6.4%) and underweight (13.9%) little changed from 1991 (67).

It is likely that many other developing countries will show similar trends as economic conditions develop. Only Russia shows a reversal of the trend – probably attributable to the dramatic fall in economic prosperity suffered by that country during the early 1990s (see Fig. 9).

2.1.1. Prevalence of overweight in different regions

Americas. Extensive data have been collected over the past three decades that allow examination of the prevalence rates and secular trends of childhood obesity in the Americas (see Appendix 1). An estimate for the region as a whole is shown in Fig. 10.

The most comprehensive and comparable national representative data on trends in the prevalence of obesity are from the USA, which are the NHE I and II data collected in the 1960s and the four NHANES collected between 1971 and 2000. These data show that the combined prevalence of obesity and overweight among American children and adolescents has more than doubled, while the prevalence of obesity has increased fourfold – and the rates continue to increase (68,69). Among US children and adolescents aged 6–18 years, the prevalence of overweight (including obesity)(IOTF definitions) has increased from 15.4% in 1971–1974 to 25.6% in 1988–1994. Some socio-economic status groups, however, are disproportionately affected. US data for overweight prevalence among children according to household income are shown earlier in this section. Racial background is also a highly relevant factor in the US: the most recent figures for 1999–2000 (using obesity defined as >95th centile, CDC 2000 reference) show that black and Hispanic children are approximately twice as likely to be obese as white, non-Hispanic children (Fig. 11), their prevalence rates having risen rapidly within the previous decade (69).

In Canada in 1981, only 11% of boys and 13% of girls were overweight or obese according to the IOTF reference, while by 1996 these figures reached 33% and 27% for boys and girls, respectively (66).

Data from Brazil and Chile are good examples to show that the rate of increase of obesity among children in some developing countries is similar to or even faster than that in the USA. In Brazil, three nation-wide, large-scale surveys were conducted in 1974–75, 1989 and 1997. The prevalence of overweight (including obesity)(IOTF criteria) tripled between the 1970s and the late 1990s, increasing from 4.1% to 13.9% among children and adolescents aged 6–18 years (67). In contrast, among pre-school children the
prevalence of obesity (WHZ > 2) fell by a third from 7.6% in 1975 to 4.5% in 1996 (61), which might be related to the decline of stunting. In Chile, two large surveys conducted in 6-year-old children showed a remarkable increase in childhood overweight (including obesity) between 1987 and 2000, from 12% to 26% in boys and from 14% to 27% in girls, based on the IOTF reference (70).

There are few data available for schoolchildren in most other South and Central American countries, but some data have been collected for pre-school children. In general, these data suggest a fast or moderate increase in obesity among pre-schoolers over the survey periods. In Bolivia, the prevalence of overweight (WHZ > 1) increased from 15.9% in 1989 to 22.7% in 1997 (62), and in the Dominican Republic it rose from 12.3% to 15.3% between 1986 and 1996 (62). On the other hand, the prevalence of obesity (WHZ > 2) in pre-school children has dropped in some countries in this region, such as Columbia where it decreased from 4.6% to 2.6% between 1986 and 1995 (61).

Europe. A number of studies have examined the trends in childhood obesity in European countries, including material collected by IOTF in collaboration with the European Childhood Obesity Group (71,72). These data suggest that childhood obesity has increased steadily in this region over the past two to three decades (see Appendix 1), although there are complex patterns in the prevalence and trends, which vary with time, age, sex and geographical region (Fig. 12). The highest prevalence levels are observed in southern European countries. A recent survey found that 36% of 9-year-olds in mainland Italy and Sicily were overweight or obese (IOTF criteria) (M. Caroli unpubl. data, 73). In Greece, the prevalence was 26% in boys and 19% in girls aged 6–17 years (IOTF criteria) (74). In Spain, 27% of children and adolescents were overweight or obese (IOTF criteria) (75), while data from Crete show 39% of children aged 12 to be overweight or obese (IOTF criteria) (J. Moschandreas, unpubl. data from ref 76). Northern European countries tend to have lower prevalence: for example, in the UK about 20% of children were overweight or obese in 1998 (63). In Sweden the prevalence was 18% for children aged 10 years, while some 13% of Finnish adolescents were overweight based on self-reported measures (S. Mårild, unpubl. data, 77). As Fig. 13 shows, children in northern Europe generally have overweight prevalence rates of 10–20%, while in southern Europe the prevalence rates are 20–35%.

The reasons for a north–south gradient are not clear. Genetic factors are unlikely, because the gradient can be shown even within a single country, such as Italy (78). The child’s household or family income may be a relevant variable, possibly mediated through income-related dietary factors such as maternal nutrition during pregnancy, or breast- or bottle-feeding in infancy.

Economic recession may affect the rate of increase in obesity levels. Some countries in the region have reported a fall in obesity rates; in Russia the prevalence of overweight and obesity declined from 15.6% to 9.0% between 1992 and 1998, a period when the country suffered severe socio-economic difficulties (67). In Poland in 1994, during a period of economic crisis, a survey of over two million young people found 8% to be overweight compared with the national reference figure of 10% (79). In rural areas, and among children under age 10, the figure was even lower at 7% overweight.
In Croatia, which experienced less economic recession, there appeared to be little change in excess weight levels in schoolchildren between the early and later 1990s (80), while in the Czech Republic, also less economically damaged than Russia, overweight (above 90th centile 1991 reference) rose modestly from 10% to 12.5% in the period 1991–1999 (81).

North Africa, Eastern Mediterranean and Middle East region. Several countries in this region appear to be showing high levels of childhood obesity. In Egypt, for example, the prevalence of overweight (including obesity) (based on WHZ > 1) was over 25% in pre-school children (62) and 14% in adolescents (82). The prevalence of obesity (WHZ > 2) in pre-school children increased from 2.2% in the 1970s to 8.6% in the 1990s (62). Similar trends are found in other parts of North Africa. In Morocco, the prevalence of obesity (WHZ > 2) among pre-school children rose from 2.7% in 1987 to 6.8% in 1992 (61), and in Tunisia from 1.3% in the 1970s to 3.5% in the late 1980s (61).

A 1999–2000 survey in Cyprus found that 25% of children aged 6–17 years were overweight or obese (IOTF) (83). Similarly, a fifth of adolescents aged 15–16 years in Saudi Arabia had BMI values more than 120% above reference median values (84). Estimates for the region are shown in Fig. 14.

Asia Pacific region. Just as for countries in Africa and in South and Central America, there are fewer data available for school children than for pre-school children from developing countries in this region, which makes it difficult to conduct a full examination of the secular trends in obesity. As shown in Appendix 1, the available data indicate that the prevalence of overweight varies considerably between different countries in this region. An estimate of the overall regional prevalence is given in Fig. 15.

The prevalence of obesity (WHZ > 2) among pre-school children remains very low in many developing countries in this region: Bangladesh (1.1%), the Philippines (0.8%), Vietnam (0.7%) and Nepal (0.3%) (61). It should be noted that no data are available for some countries in the region (e.g. Nauru, Cook Islands, Fiji) where adult obesity prevalence rates are known to be high. In these countries, prevalence rates in the child and adolescent populations are also expected to be high.

In most countries where trends data are available, childhood obesity has increased. In some countries this has been very rapid, while in other countries it has occurred at a much slower pace. For example, among Australian children and adolescents aged 2–18 years, the prevalence of overweight and obesity nearly doubled in 10 years, 1985 to 1995, from 12% to over 20% (IOTF criteria) (64). In Japan, between 1974 and 1995, the prevalence of overweight (including obesity) doubled, rising from 5.5% to 10.8% (>120% standard body weight) (85). Middle-income countries in this region, such as Singapore, South Korea and Thailand, have also all experienced an increase in childhood obesity.

In mainland China, whose population accounts for one-fifth of the global population, the prevalence of obesity has been rising quickly in both adults and children during the past two decades. Based on the IOTF reference, over a 6-year period from 1991 to 1997, China faced a moderate increase in the prevalence of overweight, from 6.4% to 7.7% (67), but the increase was more prominent in urban areas (67,86), where it went from 7.7% to 12.4% between 1991 and 1997. Furthermore, the increase is higher among urban pre-school children (87) with the prevalence of overweight rising from 15% to 29% (IOTF definition). In Hong Kong and Taiwan the prevalence also increased in the 1990s (88,89), although the increase was not as dramatic as that in mainland China.

Even in low-income countries where obesity remains rare, the prevalence has started to increase. In Bangladesh, for example, the prevalence of obesity in pre-school children increased from 0.1% in 1982–1983 to 1.1% in 1996–1997 (61).
As with some parts of Africa, the prevalence of obesity in pre-school children has dropped in some countries in the Asia-Pacific region. It is worth noting, however, that although the epidemic of obesity seems to have affected a wide range of countries, under-nutrition is still a major problem. In China, the prevalence of underweight (<5th percentile BMI of the US NCHS reference) was 9% among children aged 6–9 years, and 15% among children aged 10–18, in 1997 (67). In Indonesia over 25% and in Bangladesh and India over 45% of children under 5 years old are underweight (90,91). Thus, several of the most populous countries in this region are facing a double burden of continued undernutrition and rising over-nutrition.

Sub-Saharan Africa. There are very limited representative data available from African countries for studying the secular trends in childhood obesity, because most public health- and nutrition-related efforts have been focused on malnutrition and food safety problems. Most of the available data that do exist are collected for pre-school children and focus on malnutrition (see Appendix 1). In general, the prevalence of childhood obesity remains very low in this region, except for countries such as South Africa where obesity has become prevalent in adults, particularly among women, and where childhood obesity is also rising.

According to a recent comprehensive study conducted among pre-school children from 24 sub-Saharan countries excluding South Africa (62), the prevalence of overweight including obesity (defined as a weight-for-height standardized score greater than one, i.e. WHZ > 1) was below 10% in 18 countries, and the prevalence of obesity alone (defined as WHZ > 2) was below 5% in all countries except one (Malawi). Overall, the prevalence of overweight (including obesity) was 8.4% while for obesity alone the prevalence was 1.9%.

In some countries, the prevalence of obesity in pre-school children has dropped over the past two decades – in Mauritius it fell from 5.6% in 1985 to 4.0% in 1995 and in Tanzania it fell from 3.4% in 1991–1992 to 2.5% in 1996 (61). Among older children the IOTF estimates that, for the region as a whole, overweight and obesity affects less than 1% of children aged 5–17 years (see Fig. 16) although it is likely that sub-groups in urban locations or with higher family incomes will have substantially higher prevalence levels.

Data from South Africa (92) show the prevalence of overweight (including obesity) to be amongst the highest in the whole region. Data for young people aged 13–19 years shows the prevalence of overweight to be over 17% (IOTF cut-offs) with boys generally less at risk (7%) than girls (25%). Prevalence was highest (over 20% for both boys and girls) in white and Indian population groups.

Of all regions in the world, Sub-Saharan Africa shows the highest rates of poverty and has been subjected to war, recurrent famine and high rates of child mortality. In addition it is experiencing a widespread epidemic of HIV/AIDS affecting 5–10% of the adult population in the region, and in some areas affecting over 20%. A reduction in adult productivity through sickness or through having to care for a sick family member will reduce family earnings and reduce access to food supplies for children as well as adults. Medical costs may mean that land and livestock are sold, jeopardizing food security.

![Figure 16](image-url) Prevalence of overweight and obesity in Sub-Saharan Africa. Overweight and obesity defined by IOTF criteria. Based on surveys in different years after 1990. Source: IOTF (1).
3. COUNTING THE COSTS: THE PHYSICAL, PSYCHO-SOCIAL AND ECONOMIC CONSEQUENCES OF CHILDHOOD OBESITY

The rise in childhood obesity has been accompanied by higher rates of the correlates of obesity and the emergence of new, or newly identified, health conditions. Once considered rare in children, cardiovascular risk factors, type 2 diabetes and menstrual abnormalities began to be reported in paediatric literature in the 1980s and 1990s. Their occurrence in some populations is now routinely observed. As Pinhas-Hamiel & Zeitler have suggested (93), ‘life-style-related diseases are no longer the exclusive domain of adult medicine’ (p. 704).

The health effects of the rise in prevalence in childhood obesity are made more serious by the increased severity of the condition. Not only is a greater proportion of the population overweight, but those that are overweight are more overweight than typically observed before, with the most extreme levels in particular appearing more frequently. The delineation of the full range of health consequences linked to excess body-weight among children and adolescents may help to direct resources to their prevention.

3.1. Physical health consequences

Until recently, the complications of childhood obesity were unlikely to be clinically apparent for many years after the obesity developed, so that the consequences of obesity during childhood were rarely seen. Clinical studies of obese children have suggested a range of medical conditions for which obese children are at greater risk (94). As shown in Table 5, there are few organ systems that severe obesity does not affect. These conditions are important because they are widely prevalent, potentially serious and carry life-time consequences for health and well-being.

3.1.1. Sleep-disordered breathing and asthma

A well-established pulmonary consequence of childhood obesity is ‘sleep-associated breathing disorder’, most clearly seen in severe obesity. The term refers to a broad spectrum of sleep-related conditions including increased resistance to airflow through the upper airway, heavy snoring, reduction in airflow (hypopnoea) and cessation of breathing (apnoea). Obesity-linked hypoventilation syndrome, sometimes referred to as Pickwickian syndrome, is a serious condition associated with pulmonary embolism and sudden death in children (95). In one small study, one-third of subjects with severe obesity had symptoms consistent with sleep apnoea, and 5% had severe obstructive sleep apnoea (96). Another study found abnormal sleep patterns in 94% of obese children (97), with oxygen saturation below 90% for approximately half of total sleep time and with 40% of severely obese children showing central hypoventilation. Secondary metabolic correlates of obstructive sleep apnoea include hyperinsulinaemia, after accounting for obesity severity (98). Clinically significant effects on learning and memory function in obese children with obstructive sleep apnoea represent a troubling consequence of severe obesity (99).

Several cross-sectional studies have suggested an association between childhood overweight and asthma (100–102). In a representative survey of US children aged 2 months to 18 years, BMI above the 85th centile was linked to increased asthma prevalence, independent of age, sex and ethnicity (103), although socio-economic status and smoking were associated predictors. A representative survey in the UK showed asthma and obesity linked among girls in an inner city area, but not among boys (104). A 4-year prospective study in the UK indicated a relative risk of at least 4 for asthma, or asthma symptoms, in a high-weight group compared with a normal-weight reference group (102). Asthma and its symptoms are a difficult topic

### Table 5 Physical consequences of childhood and adolescent obesity

<table>
<thead>
<tr>
<th>Pulmonary</th>
<th>Orthopaedic</th>
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<tbody>
<tr>
<td>Sleep apnoea</td>
<td>Slipped capital epiphyses</td>
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<tr>
<td>Asthma</td>
<td>Blount’s disease (tibia vara)</td>
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<td>Pickwickian syndrome</td>
<td>Tibial torsion</td>
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<td>Flat feet</td>
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<tr>
<td></td>
<td>Ankle sprains</td>
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<tr>
<td></td>
<td>Increased risk of fractures</td>
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<tr>
<td>Neurological</td>
<td></td>
</tr>
<tr>
<td>Idiopathic intracranial hypertension (e.g. pseudotumour cerebri)</td>
<td></td>
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<tr>
<td>Gastroenterological</td>
<td>Liver steatosis / non-alcoholic fatty liver</td>
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<tr>
<td></td>
<td>Gastro-oesophageal reflux</td>
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<tr>
<td>Endocrine</td>
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<tr>
<td>Insulin resistance/impaired glucose tolerance</td>
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<tr>
<td>Type 2 diabetes</td>
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<tr>
<td>Menstrual abnormalities</td>
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<td>Polycystic ovary syndrome</td>
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<tr>
<td>Hypercorticism</td>
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<tr>
<td>Cardiovascular</td>
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<tr>
<td>Hypertension</td>
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<tr>
<td>Dyslipidaemia</td>
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<tr>
<td>Fatty streaks</td>
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<tr>
<td>Left ventricular hypertrophy</td>
<td></td>
</tr>
<tr>
<td>Other</td>
<td></td>
</tr>
<tr>
<td>Systemic inflammation/raised C-reactive protein</td>
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</table>
for study and a causative biological link between excess weight and asthma should not be assumed: children with asthma may have reduced physical activity levels, and asthma treatment medication may cause weight gain (103). Nonetheless, the observation that weight loss can improve lung function in obese adults (105) suggests that obesity prevention may decrease the impact of asthma, if not its occurrence.

3.1.2. Fatty liver disease

Non-alcoholic fatty liver disease (NAFLD) is increasingly recognized as a major health burden in obese children. NAFLD is a spectrum, ranging from fatty infiltration of the liver alone (steatosis) that is relatively benign to fatty infiltration with inflammation known as steatohepatitis or non-alcoholic steatohepatitis (NASH) and characterized by the potential to progress to fibrosis, cirrhosis and end-stage liver disease (106). Current prevalence estimates indicate that NAFLD affects approximately 3% of all children in various countries and from 23% to 53% of children who are obese, with up to 70% of these having steatohepatitis, severe fibrosis or cirrhosis (107,108). NAFLD therefore appears to be a common form of liver disease in many children, especially in developed countries where the obesity epidemic is most advanced.

Childhood NAFLD is typically a silent disease detected as asymptomatic elevation of the hepatic transaminases. Certain children complain of malaise, fatigue or a sensation of fullness or discomfort in the right upper abdomen, and some may have acanthosis nigricans on physical examination. The natural history of NASH is generally one of slow progression with manifestation of clinical disease in adulthood, although advanced liver disease including cirrhosis is known to occur in association with childhood obesity.

Severity of obesity and the presence of diabetes or hyperlipidaemia are clinical predictors of more advanced NAFLD. The coexistence of obesity and diabetes is likely to be additive – in a study of severely obese diabetic adults, all had some degree of steatosis, 50% had steatohepatitis and 19% had cirrhosis (109). Elevation of the hepatic enzymes aspartate aminotransferase and alanine aminotransferase are additional predictors of the presence of NAFLD and NASH, although significant liver disease can exist with levels of these transaminases in the normal range. The degree of elevation of serum aminotransferase concentration, therefore, has little predictive value and does not distinguish between steatosis and steatohepatitis. Similarly, while hepatic ultrasound or MRI can detect a fatty liver and NAFLD, they provide little or no information on the presence or degree of fibrosis or cirrhosis. The identification of an accurate non-invasive or blood marker for NASH is the only means to distinguish between steatosis and steatohepatitis, fibrosis or cirrhosis.

The pathogenesis of NAFLD and mechanisms that result in steatosis versus steatohepatitis and its sequelae are incompletely understood, but probably involve a combination of insulin resistance leading to accumulation of fat in hepatocytes and the generation of reactive oxidative species resulting in oxidative hepatocyte damage (110,111). This has led to treatment attempts utilizing antioxidants, with varying degrees of success. To date, the results of treatment studies for NASH are not conclusive because of design limitations such as small patient numbers and differences in definitions of NASH and endpoints. While the efficacy and role of other therapeutic strategies including pharmaceuticals remain to be proven, exercise and diet resulting in controlled weight loss can reverse the progress of fatty liver disease (112), and hence remain the cornerstones of therapy.

3.1.3. Menstrual problems and early menarche

Abnormalities in menstruation and early menarche represent part of the endocrine response to excess body weight in girls. Previous studies have established a relationship between obesity and lowered fertility (113,114) but the impact of excess weight on menstrual problems in adolescence is less well established.

Oligomenorrhoea or amenorrhoea associated with obesity, insulin resistance, hirsutism, acne and acanthosis nigricans comprise a ‘polycystic ovary syndrome’. The appearance of insulin resistance in youth, associated with overweight, may foreshadow an increased prevalence of polycystic ovary syndrome in adolescence. The prevalence of polycystic ovary syndrome in youth is unknown, and it is often undiagnosed because adolescents rarely have the characteristic ultrasonic ovarian morphology or because altered menstrual patterns or late menstruation may go unrecognized. Hormonal patterns typical of polycystic ovary syndrome, however, are increasingly described in obese children (115,116). In a case-control study, increased BMI at age 18 (based on recalled weight and height) was associated with elevated risk of ovulatory-related infertility in young women (117). This study, of the US Nurses II cohort, also indicated a positive association between BMI at age 18 and irregular menstrual cycling.

Menarcheal timing is influenced by weight status, with higher relative weights associated with earlier menarche (118,119). The rise in childhood obesity seen in the last decade among younger children may result in a further lowering of the population average age of menarche. Evidence from the US NHANES II study shows that the frequency of early menarche is closely linked to obesity status: 33% of higher-weight girls attained menarche before age 11 compared with less than half that proportion among lower-weight girls, and the difference is even more marked.
when looking at the proportions of girls attaining menarche below the age of 10 years (see Table 6). The possible health consequences of early menarche are both immediate and delayed. Early menarche is an established risk factor for breast cancer and has been linked to other cancers of the female reproductive system (120,121). Early menarche has been proposed as a risk factor for common psychotropic problems experienced by adolescent girls: depression, disordered eating and substance abuse (122,123). The mechanism for these effects may be affiliation with an older peer group. Finally, early menarche has been associated with increased risk of spontaneous abortion in adulthood (124).

3.1.4. Delayed maturation linked to obesity in adolescent boys

Overweight boys tend to show later maturation than their non-overweight counterparts. Although early sexual maturity is associated with overweight in girls, in boys the reverse appears to be the case, with the prevalence of overweight and obesity higher in late maturers than in early maturers (59). The differences are also reflected in the changing body composition that occur during puberty, when girls tend to increase fat mass as a result of maturation while boys tend to increase muscle and other non-fat body mass.

3.1.5. Type 2 diabetes

Previously only seen in adults, the emergence of type 2 diabetes in youth represents a particularly alarming consequence of the obesity epidemic in children. The onset of diabetes in youth will increase the risk in early adulthood of the advanced complications of the disorder – cardiovascular disease, kidney failure, visual impairment and limb amputations. A review by the American Diabetes Association suggests that as many as 45% of paediatric diabetes cases are the type 2 non-insulin dependent form (125).

Although other factors are associated with type 2 diabetes in children (including family history, ethnicity and the presence of acanthosis nigricans), the most important risk factor is obesity. In a study of childhood diabetes, Scott et al. (126) found excess bodyweight among over 90% of adolescents with type 2 diabetes while among children with type 1 diabetes excess bodyweight was found in about 25% of cases (see Fig. 17).

![Figure 17](image1.png)

**Figure 17** Prevalence of excess bodyweight (>85th centile) in newly diagnosed diabetic adolescents, USA. Source: Scott et al. (126).

![Figure 18](image2.png)

**Figure 18** Rising numbers of type 2 diabetic patients in a paediatric department in Arkansas. Children aged 8–12 years. Source: Scott et al. (126).

The prevalence of type 2 diabetes among children is difficult to establish. In a multi-ethnic study of a clinical population of 167 obese children and adolescents in the US, impaired glucose tolerance was present in 25% of younger obese children and 21% of obese adolescents by 2 h tolerance test (127). Undiagnosed diabetes was detected in 4% of the adolescents. Similar findings had been noted 30 years earlier, when 17% of a group of 66 obese children showed impaired glucose tolerance, and 6% met the criteria for type 2 diabetes (128).

As the prevalence of obesity rises, the prevalence of diabetes type 2 can be expected to follow. In Cincinnati the prevalence of adolescent diabetes type 2 increased 10-fold from 0.7 to 7.2 cases per 100 000 population in the period 1982 to 1994 (129). Similar rising numbers have been noted in Arkansas Children’s Hospital, even among pre-adolescent children (see Fig. 18).

Ethnic minority groups that are known to be at risk of high levels of adult diabetes may also show high levels of diabetes among adolescents. Rates as high as 5% have been found among adolescents in native American populations.
Obesity in children and young people

Prevalence and incidence of diabetes:

In Europe, both type 2 diabetes and impaired glucose tolerance are being reported among obese adolescents. Frelut (132) reports type 2 diabetes prevalence rates of 1.9% in obese adolescent populations in Hungary, and impaired glucose tolerance of 15% among this group (Table 8). In the UK, several cases of type 2 diabetes in childhood have been reported among ethnic minority groups known to be at higher risk (133), and in 2002 the first cases were reported among white children (four cases aged 13–15 years, all of whom were obese) (134).

In Japan, the prevalence of type 2 diabetes is reported to have nearly doubled in the period from the late 1970s to the early 1990s, from 7.3 to 13.9 cases per 100 000 adolescents (135). A study in Tokyo of 1400 children attending a diabetes clinic during the 1980s found that none under the age of 9 years had type 2 diabetes, but by the age of 13–14 years the proportions where roughly equal between type 1 and type 2 diabetes, with the proportion of type 2 diabetes rising further into young adulthood (136).

Japanese children with type 2 diabetes were both taller and heavier than the national average, and about 80% of these children were obese (137).

Diagnosis of type 2 diabetes among children and adolescents is complicated. The majority of patients are asymptomatic or present with symptoms such as vaginal monilial infection, rather than the classic triad of polyuria, polydipsia and weight loss (126).

Table 8  Type 2 diabetes and impaired glucose tolerance among obese adolescents in various European countries

<table>
<thead>
<tr>
<th>Sample size</th>
<th>Type 2 diabetes (%)</th>
<th>Impaired glucose tolerance (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hungary</td>
<td>207</td>
<td>1.9</td>
</tr>
<tr>
<td>Germany</td>
<td>512</td>
<td>1.6</td>
</tr>
<tr>
<td>Italy</td>
<td>800</td>
<td>&lt;0.3</td>
</tr>
<tr>
<td>France</td>
<td>256</td>
<td>&lt;0.2</td>
</tr>
</tbody>
</table>

Source: Frelut (132). Definitions of obesity differ.

Hyperinsulinaemia is a common feature in obese children and adolescents (138). The relationship appears non-linear, with little increased risk of hyperinsulinaemia as BMI increases up to the 85th or 90th centile, but with dramatically increased risk above the 95th and especially the 97th centile of BMI (see Fig. 19). Adolescents (aged 11–18 years) above the 90th centile for BMI had higher levels of plasma insulin (mean 23.7 mU mL\(^{-1}\)) than those below the 90th centile BMI (mean plasma insulin 11.7 mU mL\(^{-1}\)) (140).

As a progressive condition for which treatment relies heavily on self-management, type 2 diabetes in youth will require particular attention from health care providers and carers. Physical activity improves insulin resistance in both obese and non-obese youth (141) and weight loss improves insulin sensitivity and decreases hyperinsulinaemia (138), although obese children who maintain weight loss continue to show elevated insulin levels in spite of improved glucose tolerance (142).

Maternal diabetes increases the risk of having an overweight child (see later in this report on risk factors for childhood obesity). A diabetic cycle may thus be established in which overweight adolescent girls and young women develop diabetes and become pregnant, are then likely to have heavier babies which in turn are at risk of child and adolescent obesity and hence at risk of becoming diabetic.

### 3.1.6. Cardiovascular risk factors

The Bogalusa study in Louisiana has provided detailed information on cardiovascular risk factors in childhood (see Fig. 20) and their persistence into adulthood (143). In the study, overweight during adolescence was associated with an 8.5-fold increase in hypertension, a 2.4-fold increase in the prevalence of high total serum cholesterol values, a 3-fold increase in high LDL serum cholesterol values and an 8-fold increase in low HDL serum cholesterol levels as adults aged 27–31 years (144). Similarly, the Muscatine study in Iowa has shown that adolescent obesity, especially in males, is associated with higher levels of total and LDL cholesterol in adulthood (145).
Hypertensive children who continue to have high blood pressure in adulthood are more likely to have higher body weight, BMI, skin-fold thickness and substantially greater waist and hip circumferences (146).

Several studies have shown links between weight gain in childhood and a subsequent increase in cardiovascular risk factors in urban African-Americans (147) and in populations in Finland (148,149). The Finnish data suggest that the cluster of cardiovascular risk factors in adulthood – including hypertension, hypertriglyceridaemia, low HDL cholesterol and hyperinsulinaemia – sometimes referred to as the metabolic syndrome, is especially common among obese adults who were also obese as children (Table 9). The prevalence of a metabolic syndrome among adolescents aged 12–19 years has been investigated in the NHANES 1988–1994 cohort (150). The overall prevalence of the syndrome was 4.2% (6.1% among males and 2.1% among females). The prevalence was significantly related to weight status, being found among less than 0.1% of adolescents of normal weight (BMI below 85th centile), rising to 6.8% among overweight adolescents (BMI 85th–95th centiles) and an extraordinary 28.7% among obese adolescents (BMI 95th centile and above).

**Hypertension**. The Task Force on Blood Pressure (151) indicated that detection and prevention of hypertension in a paediatric population and its early treatment contribute to a reduction in the high risk of morbidity in adulthood. Obesity is frequently associated with hypertension in adults and the same appears true in children (Fig. 21). Up to 30% of obese children suffer from hypertension (152), and among adolescents one survey found 56% of those with persistent elevated blood pressure were also significantly overweight (153). A second study has shown that

<table>
<thead>
<tr>
<th>Prevalence</th>
<th>Odds ratio</th>
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<tbody>
<tr>
<td>Obese as adults, not obese in childhood ( (n = 71) )</td>
<td>10%</td>
</tr>
<tr>
<td>Obese as adults, obese in childhood ( (n = 75) )</td>
<td>28%</td>
</tr>
<tr>
<td>Not obese as adults ( (n = 293) )</td>
<td>1%</td>
</tr>
</tbody>
</table>

*Source: Vanhala et al. (149).*

**Figure 20** Prevalence of excess weight among children with up to four risk factors for cardiovascular disease. (Risk factors: elevated TG, LDL, insulin, or BP; low HDL.) Source: Freedman et al. (139).

**Figure 21** Proportion of children with raised systolic and diastolic blood pressure by BMI centiles. Percentage with blood pressure measures above 95th centiles. Children aged 5–10 years. Source: Freedman et al. (139).
measures of resting blood pressure are correlated with waist circumference and skin-fold measurements in children (154). Measures of blood pressure during exercise may provide further evidence of the links between obesity and hypertension in children (155).

Insulin resistance and hypertension may be present as part of the metabolic syndrome associated with obesity. Overweight young people with blood pressure values above normal levels show a tendency to higher fasting plasma insulin levels. The Bogalusa Heart Study on cardiovascular risk factors showed a significant positive correlation between insulin levels and blood pressure in children aged 5–12 years, but not in adolescents. Other researchers, however, suggest that blood pressure is mediated by the secretion from fat tissue of factors such as leptin and angiotensinogen (156) or possibly adiponectin (157).

Serum lipids and lipoproteins. Increased levels of LDL cholesterol, decreased levels of HDL cholesterol and raised serum triglyceride levels are highly correlated with increased triceps skin-fold thickness among adolescents (158) and with centile scores of BMI (159). The relationship may be non-linear, however, with no significant increases in these measures in children with BMIs below the 85th BMI centile, but with dramatic increases found among children above the 97th BMI centile (see Fig. 22).

Prospective studies show that the serum lipid and lipoprotein levels can track from childhood into young adulthood and are predictive of adults levels. A longitudinal study over 15 years of 1169 children aged 5–14 years at the beginning of the study found that their lipoprotein levels can track from childhood into young adulthood, and are predictive of adult levels. The link between weight gain and adult lipid and lipoprotein levels was also demonstrated. Weight gain was second only to initial levels of lipid and lipoprotein in predicting adult levels. The link between weight gain and adult lipid and lipoprotein levels increased during the transition from adolescence to young adulthood, and was especially strong in males.

3.2. Psychological and social consequences

Obesity in children and adolescents may have its most immediate consequences in the psychological and social realms. Stigmatization of obese children and adolescents has long been recognized in Westernized cultures, and is well documented among the children’s peers. A 1967 study showed that young boys (aged 6–10 years) described obese body types as being indicative of negative personality characteristics – cheating, lazy, sloppy, lying, naughty, mean, ugly, dirty or stupid (161). Similar observations have been made among Australian boys and girls (aged 8–12 years) (162). In younger children the degree of negative stereotyping increased with age (163). Among adolescents, girls may be more affected than boys, although both genders report some negative experiences (164).

Providing children with medical explanations for the development of obesity did not appreciatively affect their negative characterization of a hypothetical obese peer (especially among older children) and did not influence their aversion for sharing activities with an obese peer (165). In a study where boys and girls (aged 10–11 years) were asked to rank order a series of drawings of children with various handicaps (crutches, wheelchair, missing a hand, facial disfigurement, obesity) based on which child they ‘liked best’, the drawing of an obese child was ranked lowest, irrespective of the ranking child’s sex, race, socioeconomic status, living environment or own disability (166) and this finding has been replicated recently (167). Thus, although childhood obesity is far more common, the social reaction to an obese child does not appear to have softened.

Both the social and the economic consequences of obesity in childhood and adolescence are pervasive in Western societies. American women who were obese as adolescents became adults with lower educational attainment, earning less money, experiencing higher rates of poverty and having a lower likelihood of marriage, compared with thinner women (168). Similar results have been observed in a British cohort (169). Obese youths have experienced greater discrimination in attempting to rent apartments (170) and in gaining admission to colleges, although there is no evidence that obesity is related to lower academic aptitude or to a lesser desire to attend college (171,172).
Given the stereotyping and discrimination experienced by obese children, it might seem likely that obese young people have fewer friends, and this has been shown in work by Strauss (173), demonstrating the reduced numbers of friends claimed by overweight girls compared with non-overweight girls (see Fig. 23). In contrast, a study of 9-year-old girls in the UK did not find that overweight children were less popular (175). Peer rejection and lack of friends in childhood, when it does occur, has been associated with reduced psychological functioning in adulthood (176).

The effect of obesity on a child’s self-esteem has received considerable study, although how self-esteem is measured varies and may include school academic performance, body appearance, athletic ability, social networking, behaviour and conduct attributes (177,178). Results generally indicate either a weak association between low self-esteem and obesity or no association (177,179), although there is some evidence that the association may differ by race (178) and that athletic and body appearance aspects of self-esteem diminish with increasing obesity (175).

Body dissatisfaction and a desire to lose weight has been found even in young children (180), but this desire is not limited to children who are overweight and appears to represent a separate construct. A study following children for 4 years from the age of 9–10 years found that obese children did not differ in self-esteem from normal-weight children at the start of the period, but that their self-esteem was more likely to decline over the following years; the effects were most marked among white and Hispanic girls but were also detectable among black girls and among boys (179). Among older children there is a general finding of decreased self-esteem in obese compared with non-obese youth (177,179,181,182) but the effect is not universally observed (168,183). In a 7-year follow-up of adolescents initially aged 16–24 years, those with a BMI above the 95th centile did not show decreasing self-esteem according to a simple assessment method (168).

Few studies have assessed the association of obesity with concurrent or subsequent psychiatric pathology, such as depression or anxiety states. Braet et al. (184) compared obese children from clinical and non-clinical settings with normal weight controls and found that while self-esteem was reduced in both obese groups, increased psychiatric pathology was only present among obese children in the clinical group, suggesting that the factors which led the parents to bring their child to seek treatment may be more responsible for the psychological effects than the obesity per se. Among Californian elementary school children a modest but statistically significant association between increasing symptomatology of depression and higher BMI has been observed for girls although not for boys (185). Eisenberg et al. (186) have reported increased suicidal ideas, and suicidal attempts, among overweight adolescents who reported being teased by peers or family members.

Being teased and feeling unhappy with one’s appearance – frequent correlates of childhood obesity – may influence the psychological consequences of obesity (187). In a 1-year study of US adolescents, however, Goodman & Whitaker (188) found no evidence for obesity increasing the risk of depression, although the instruments used (a dichotomous screening questionnaire) may have reduced the likelihood of detecting an effect.

Some of the negative consequences of adolescent obesity result from unhealthy eating behaviours that accompany elevated weight. In a large survey of high-school students in Minnesota, overweight adolescents were more likely to report dissatisfaction with their weight and there was evidence that unhealthy eating behaviours – binge eating, chronic dieting – were more common; after controlling for age, ethnicity and socio-economic status there was a residual weak association between obesity and emotional health in boys, and a similar weak association between obesity and suicidal ideation in girls (189). In a similar study of Connecticut youth, overweight adolescent girls were more likely to report hopelessness and to have attempted suicide than normal weight girls (164). Results were attenuated for boys and did not reach statistical significance.

The evidence regarding the psychological consequences of child and adolescent obesity is equivocal and is largely based on studies of children in Western populations. There have been few reports of psychiatric pathology associated with excess weight, although occasional reports have been published (190). Two recent review articles (191,192) provide useful organizing frameworks for further studies in this field.

The direction of causality needs to be considered, because depression or anxiety may be linked to weight gain through a number of changes in behaviour leading to
changes in diet or physical activity. Two studies from the US have found depression in adolescence to predict later obesity (188,193).

Not all cultures view excess weight as a negative attribute. For example, a study in Mexico noted that food ‘treats’ for children are a cultural index of parental caring, and that parents value child fatness as a sign of health (194). The study found that obese Mexican children have no greater social problems (peer rejection or stigma) or psychological problems (anxiety, depression or low self-esteem) than their non-obese peers. It is possible that similar cultural patterns are found in other regions of the world, and within sub-populations of Western industrialized societies. Such studies emphasize the importance of family and peer attitudes in the generation of psychological distress in the obese child. Several studies have shown a correspondence between a mother’s attitudes to food and her child’s self-perceptions (195). Similarly, children who have been teased by peers about their body shape are more likely to be dissatisfied with their appearance when older, to the extent of developing eating disorders (196).

3.3. The economic costs of obesity in childhood

A rising prevalence of obesity in a population, and an increasing frequency of hospital treatment for obesity-linked diagnoses, will lead to a rising burden on a nation’s health services. In the USA, an increase in the prevalence of obesity has been accompanied by a more rapid increase in the extent of the obesity, with more children becoming more severely obese (197), indicating that obesity-related medical conditions will rise at least as rapidly as the overall obesity prevalence rate. Furthermore, in so far as childhood obesity tracks into adulthood, the rising rates of obesity in childhood will inevitably lead to earlier, and hence more costly, referrals into the health care system for young adults.

Besides this direct burden of obesity and related diseases upon the medical services, other costs are also incurred, for example the lost educational opportunity and hence lost economic contribution, the lost days of employment by an older adolescent or by a parent or carer in the family if the child requires medical attention. Furthermore, there are intangible costs such as the psycho-social consequences referred to in the previous section.

It should be borne in mind that the costs will vary according to circumstances, with individual variations (e.g. in genetic risk for developing type 2 diabetes) as well as social variables (such as the psycho-social acceptability of excess bodyweight in the community) affecting the total burden on society caused by obesity.

3.3.1. Direct costs

The direct costs of obesity are those that are costs to the health care system, including the resources applied to the management of obesity and the conditions associated with or caused by obesity. There are a number of studies that have estimated the costs of adult obesity in various countries (16). These studies suggest that obesity accounts for 2–7% of a developed countries’ total health care costs (see Table 10). The estimates may be regarded as conservative, and care should be taken when making comparisons: e.g. different BMI cut-off points are used to define obesity and overweight, there are different estimates for the proportion of any single disease that is directly attributable to obesity in a specific population, and there are differences between countries in the costs of providing health care (with the cost of specific items of health care being greater in the USA than in many other countries).

Additional diseases and problems that have not been considered in the analysis could be added, for example there is increasing acceptance that cancers of the endometrium, breast, colon, pancreas and kidney are increased by obesity. Non-alcoholic fatty liver (non-alcoholic steatohepatitis) is linked to obesity and is the third most common reason for liver transplants in adults in many developed countries, representing significant medical costs. Similarly, the recognition that a small amount of weight loss increases fertility and reduces the need for in vitro fertilization (IVF) programmes means that a proportion of the costs of these expensive treatments needs to be allocated to the cost of obesity. For further discussion on the calculation of the costs of obesity see Obesity: Preventing and managing the global epidemic (16).

The direct costs of obesity in children and adolescents may not appear to be great when compared to other disorders in that age group for several reasons. First, few treatments are provided for most overweight children so the health care costs may be low. This might in turn be a result of health professionals not appreciating the seriousness of the problem, or of discrimination against overweight children within the health services. As a result, associated disorders may remain undiagnosed and hence under-reported. Even when health service providers are sympathetic, the children themselves may not be aware of relevant symptoms, or not realize that they should report them. Furthermore, there is a failure to record bodyweight measures when diagnosing childhood disorders, leading to
a lack of information that could relate a range of possible disorders to a child’s excess weight. Calculation of the true costs of child obesity will need a methodology that takes into consideration the associated disorders and their treatment costs.

One study that attempted to establish the costs for hospital treatment of obesity-related disorders in children has been published by Wang & Dietz (198). Using US hospital discharge diagnoses during 1997–1999, they listed the most frequent principal diagnoses where obesity was listed as a secondary diagnosis. The most common co-morbidities, accounting for over 40% of principal diagnoses observed in obese children, are shown in Table 11. Wang & Dietz then compared hospital diagnosis figures in 1997–1999 with those around 18 years earlier, 1979–1981, for the age group 6–17 years. They found the rate of asthma as an obesity-related co-morbidity increased from 5.9% to 8.1% of hospital episodes. Diabetes linked with obesity rose from 1.4% to 2.4%. Sleep apnoea rose fourfold to 0.8% and gall bladder disease rose twofold to 0.6%. There will be some overlap of diagnoses, but from these figures it appears that these four obesity-related disorders alone account for over 10% of child hospital episodes, and that this figure is nearly twice that found two decades earlier. These data may be an under-representation of the true prevalence, given that it is likely that obesity was under-reported as a secondary diagnosis. In addition, Wang & Dietz showed that hospital in-patient time for obese children had lengthened during the period, typically from 5 to 7 days. The total number of days of care had doubled to over 300 000 for this US population. The cost per episode had also increased so that the final figure provided by Wang & Dietz for children with an obesity diagnosis was over US$127 million in the late 1990s, rising from US$35 million per year (adjusted for inflation) two decades earlier. The figure of US$127 million amounts to 1.7% of annual total US hospital costs.

### Table 11 Principal diagnosis (and ICD-9 reference) when obesity was given as a secondary diagnosis

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>Asthma (ICD 493)</td>
<td>8.1%</td>
</tr>
<tr>
<td>Conduct disturbance (ICD 312)</td>
<td>4.7%</td>
</tr>
<tr>
<td>Diabetes (ICD 250)</td>
<td>4.6%</td>
</tr>
<tr>
<td>Outcome of delivery (ICD V27)</td>
<td>4.6%</td>
</tr>
<tr>
<td>Other cellulitis or abscess (ICD 682)</td>
<td>4.2%</td>
</tr>
<tr>
<td>Depressive disorder (ICD 311)</td>
<td>3.9%</td>
</tr>
<tr>
<td>Neurotic disorders (ICD 300)</td>
<td>3.9%</td>
</tr>
<tr>
<td>Adjustment reaction (ICD 309)</td>
<td>2.7%</td>
</tr>
<tr>
<td>Gall bladder disease (ICD 574-5)</td>
<td>2.3%</td>
</tr>
<tr>
<td>Chronic disease of tonsils and adenoids (ICD 474)</td>
<td>2.2%</td>
</tr>
<tr>
<td>Disturbance of emotions specific to childhood or adolescence (ICD 313)</td>
<td>1.5%</td>
</tr>
</tbody>
</table>


This analysis did not report the specific treatment costs of obesity-related disorders, and based the cost estimates on average charges across all hospitalizations. In adults, the severity of various disorders tends to be greater for more obese adults than it is for normal or moderately overweight adults (199–201) thus the treatment costs will be correspondingly higher for treating the same diagnosis among obese compared with non-obese patients. It is plausible that a similar cost premium may apply to the treatment of disorders in children who are obese.

The costs to the health services of failing to intervene in childhood obesity may also be mentioned. If childhood obesity is a risk factor for adult diseases, then rising rates of childhood obesity, especially among the heaviest children, can be predicted to lead to earlier onset of adult obesity-associated disorders. Earlier onset in adulthood will then lead to a longer subsequent lifetime of disability and treatment, creating a significant extra financial burden on national health services.

#### 3.3.2. Indirect costs

The indirect costs of obesity are those relating to a reduction in the level of economic activity following illness or premature death attributable to obesity. Indirect costs of adult obesity were estimated to be AUS$272m in Australia (population around 18 million) and US$23bn in the USA (population around 280 million) (1989–1990 figures) (16). In a study of Swedish women, approximately 10% of the total costs of lost productivity due to sick leave and payment of disability pensions were related to obesity (202). It should be noted that there are differences in methodology and in the costings used for these analyses.

It is difficult to calculate equivalent costs for childhood obesity because the contribution of children to a nation’s economy is highly variable. Although the standard measures for indirect costs e.g. premature death, loss of productivity, absenteeism, sick leave and disability pensions, may be applied to older adolescents, other more innovative factors will need to be considered, especially for younger children. These might include the need to take time off work for parents caring for ill obese children, time off school, specific costs to the education system, the problem of those who are unemployable or made redundant because of their early onset obesity, and so on. It may be possible in the future to consider more specifically the costs to the education system, for example, of absenteeism of obese children and the effect this has on the school, and on the costs of providing special equipment and teaching, especially in the physical education sphere. Childhood obesity may also have effects on the provision of other services and goods: school transport, leisure facilities, fuel, clothing and even food supplies. Lastly, the costs of premature death...
produced by obesity in childhood will need to be studied specifically.

3.3.3. Intangible costs

The intangible costs of obesity are the social and personal costs or losses associated with obesity. The costs spent by families on commercial weight loss programmes would be one intangible cost that might be relatively easy to collect. Other intangible costs, however, are far more difficult to define, are more variable and might be subject to the interests and biases of the individual researcher. For example, it is agreed that obesity reduces the quality of life of individuals – they tend to have lower self-esteem. There is no standard approach either to the measurement of these psycho-social factors or to the costs or losses they may produce. In a paper for the IOTF, Segal and co-workers estimated that the intangible costs of adult obesity in Australia were of the order AU$13–18bn, 10 times the direct costs of obesity (203). The same is likely to be true of other developed economies.

In the calculation of the intangible costs of obesity there are probably more suppositions, estimations and outright guesses than in the calculation of the direct and indirect costs of obesity. These intangible costs of obesity, however, probably have the most impact on the individual, and particularly on the child, who is overweight or obese. It is important to design studies to estimate the intangible costs of obesity that children bear and not to assume that the costs and effects are similar or smaller compared to those found in studies on adults. In one study of severely obese children in the USA the quality of life scores were substantially lower than those of non-obese children, and were similar to the scores of children diagnosed as having cancer (204). Studies are needed that investigate the consequences of psycho-social factors on school performance, job prospects and employment. Such intangible costs will have a major impact for the child and for society. A systematic analysis of the quality of life needs to be undertaken so that an assessment of the intangible costs of obesity in childhood can be made.
4. THE BIOLOGY OF WEIGHT CHANGE IN CHILDHOOD

This report has looked at the extent of childhood obesity and the consequences and costs of obesity. In order to make interventions – through the treatment of obese children and adolescents or through prevention of the problem in populations at risk – it is necessary to understand how a child becomes obese.

Many studies have shown that obesity tends to track across the life course, so that once a child becomes fat, he or she is more likely to be fat later in life, although the magnitude of this risk depends on the measures used and study duration. These factors are considered in more detail in the following sections, which look at the developmental aspects of growth and body weight, the current views on the genetic, molecular and cellular aspects of weight gain, and the various physical and environmental risk factors that increase the likelihood that a child will accumulate excess weight.

4.1. Molecular and cellular aspects of adipose tissue development

The storage and use of energy in the human body is controlled by several regulatory systems, producing a variety of signals: appetite, satiety, messages via the sympathetic and parasympathetic nervous system and a complex range of messages from the endocrine system. In the short term, food-modulated regulatory signals from the gastrointestinal tract along with signals generated from dietary factors before and during food metabolism inform the central nervous system along with signals generated from dietary factors. In the short term, food-modulated regulatory signals from the gastrointestinal tract along with signals generated from dietary factors before and during food metabolism inform the central nervous system about food intake and regulate hunger and satiety. In the longer term, a series of homeostatic mechanisms are involved in regulating fat storage and release, resting metabolic rate and energy expenditure, which may be largely determined by genetic mechanisms and susceptible to genetic variation.

Adipocytes in white adipose tissue are the body’s principal energy stores. A relatively lean child weighing, for example, 30 kg will have about 4.5 kg of triglycerides stored in adipose tissue, equivalent to 1.3 million kJ in energy (205). Energy stored in protein (mostly in muscle mass) amounts to some 26 000 kJ and as glycogen only 2100 kJ.

Adipose tissue may be found in the fetus after the 14th week of gestation. By birth adipose tissue amounts to about 13% of the newborn’s body mass, doubling to around 28% by the end of the first year for a normal-weight infant. This period of adipose tissue accumulation, recognized over a century ago by Stratz (206) has been referred to as the ‘first filling period’ to be followed by a fall in the fat proportion of body mass over the next 6–10 years, referred to by Stratz as the ‘first stretching period’. He noted a second filling period during puberty, a post-pubertal stretching period, and a final filling period during later adolescence, around 16–20 years. Various researchers have estimated the average percentage of fat in the body mass of children (207–209), and this can be seen diagrammatically in Fig. 24.

The two periods in which body fat is gained differ in one important respect. During the first year of life, fat accumulation is largely by means of increased volume of the adipocytes. During the pubertal period, the fat accumulates largely through an increase in the number of adipocytes, without significant further change in fat cell volume (205). This indicates two distinct mechanisms at work, one which regulates the breakdown or storage of fat within the cell through lipolytic and lipogenic mechanisms, and one which regulates the number of cells through proliferation (from pre-adipocytes) or removal through apoptosis or merger of fat cells.

The formation of adipose cells from pre-adipocytes shows age-dependent differences in proliferative activity, with maximum proliferation and capacity for differentiation during the first year of life and in the period just before puberty. Adipose tissue in children generally contains a much higher proportion of smaller fat cells than such tissue does in adults, indicating a higher rate of formation of new cells during childhood.

Various medical conditions are linked to adipocyte size and numbers: for example hypothyroidism is associated with a decreased number of fat cells with or without a corresponding change in total adipose tissue mass (205). Growth hormone-deficient children have slightly increased body fat stores through enlarged fat cells, but fewer in number than found in the equivalent tissue from healthy children (205). Glucocorticoids, which are potent stimulators of adipocyte proliferation, in excess can lead to features characteristic of Cushing’s syndrome, with an increase in adipose tissue mass especially in the abdominal region.

IGF-1 receptors and insulin receptors can be found in adipocytes and pre-adipocytes, and insulin is able to stim-
ulate glucose uptake and lipogenesis as well as to inhibit lipolysis. Other factors act to inhibit the proliferation of adipocytes, including tumour necrosis factor, epidermal growth factor and platelet derived growth factor.

One special function of the fat cell is the secretion of leptin, which has been shown to inform the brain about the status of energy reserves. There is a direct relation between adipocyte volume and leptin secretion. The disappointing therapeutic results with leptin in obese patients could be due to leptin playing a role as a starvation signal rather than an adiposity signal (210).

Adipocytes are able to secrete a large number of peptides and cytokines, including prostaglandins and the steroid hormones androgen and oestrogen. In this respect adipose tissue is not only an important repository for energy but is also an active endocrine organ, with an influence on cardiovascular and metabolic function, fertility and sexual maturation (Fig. 25).

Levels of serum IGF-1 and IGFBP-3 appear linked to the formation of adipocytes, adding support to the suggestion that the protein content of the diet, which leads to increased levels of IGF-1, may influence adipose tissue growth early in life (211). This link to dietary protein intake is reflected in Waterlow’s observation that in undernourished children, IGF-1 levels are low while growth hormone levels are high (212), the reverse of that noted in obese children, while Rolland-Cachera has suggested that excess protein, especially during the earliest stages of childhood, may be a risk factor for obesity later in childhood (213).

4.2. Developmental aspects of weight gain

4.2.1. Intrauterine environment and post-natal growth

A number of studies have shown that birth weight, a crude summary of growth in utero, is positively related to subsequent fatness (214,215), suggesting that the fetal environment plays a role in the development of obesity. Although limited data indicate the association between birth weight and later fatness to be independent of gestational age (216,217) and socio-economic status (218,219), parental fatness (particularly maternal fatness) may be largely responsible for the relationship (220–222). A study of 35 000 young people called for army service in Israel found that those with the very heaviest birthweights were at threefold risk of becoming severely overweight at age 17 years (defined as BMI >27.9 kg m\(^{-2}\)) (223,224). The risk was particularly high among the fattest female babies (Fig. 26).

Fig. 26 does not explore the relationship between very low-weight-for-date babies and subsequent risk of excessive weight gain. Several studies report a J- or U-shaped relationship, with a higher prevalence of obesity seen for both the lowest and highest birth weights (215,225), suggesting a more complex association between growth in utero and obesity. Just such a U-shaped relationship between birthweight and subsequent age-adjusted adult BMI was found in the US Nurses Health Study 1 (226) (see Fig. 27).

Studies of famine or maternal smoking during pregnancy also suggest the picture is complicated, and that an adverse

![Figure 25](Complex%20hormone%20interactivity%20in%20adipose%20cells.%20Sources:%20Wabitsch%20(205),%20Hofbauer%20(210).)

![Figure 26](Risk%20(odds%20ratio)%20for%20obesity%20among%2017-year-old%20youths%20according%20to%20birthweight.%20Source:%20Martorell%20et%20al.%20(223),%20Seidman%20et%20al.%20(224).)

![Figure 27](Birthweight%20and%20age-adjusted%20adult%20BMI.%20Source:%20Martorell%20et%20al.%20(223),%20Curhan%20et%20al.%20(226).)
foetal environment may promote obesity independent of any effect on foetal growth (227,228). (For more on maternal and early childhood risk factors see part 4 of this section.) Maternal diabetes during pregnancy results in offspring with higher birth weight and higher risk of obesity in childhood, although only after 4–5 years (229). The latter association seems to be independent of birth weight and maternal weight, suggesting that the effect is due to alterations to the intrauterine environment.

Further work is required to determine whether different types of diabetes, insulin and non-insulin dependent, and gestational diabetes have different effects. Another possibility is that post-natal weight gain is important: a recent study reported that rapid weight gain during the first 4 months of life increased risk of obesity at 7 years of age, independently of birth weight and gestational age, weight at 1 year, maternal BMI and education (219). Other studies suggest that it is the combination of foetal growth and subsequent growth that is relevant, and that light babies who show post-natal catch up growth or rapid childhood growth are at increased risk of obesity (222,230). See also part 4.4 of this section.

### 4.2.2. Weight gain in infancy and childhood

It is normal for young infants to put on a high percentage of body fat, but the rate of fat deposition slows from around the age of weaning onwards. If children are becoming progressively and excessively fat at ages when other children are tending to show a fall in fatness and in BMI (i.e. between around 6 months and 5 years old), this is probably a warning sign for significant and perhaps, since it is developing when other children are tending to reduce fat, persistent obesity.

Studies vary on the relationship of overweight/obesity in infancy and their links to excess bodyweight in later childhood and adolescence. Risk factors, such as lack of breastfeeding, are discussed in part 4.4 of this section, below. One problem in making links between early and later childhood is the changing prevalence among the populations; as the prevalence of obesity in childhood increases, the probability of obesity in infancy leading on to obesity in childhood is likely to increase. In the 1970s, infants that were obese in the first 6 months of life showed no significant increase in the risk of being obese at age 5 years for example. More recent research, looking especially at the patterns of BMI during the first 5 years, suggests that there are detectable predictors of later obesity.

Plotted over time, the body mass index shows an initial fall during the second to fifth year of life and then a gradual rise from the sixth year through adolescence and most of adulthood. The age at which this second increase starts has been termed the ‘adiposity rebound’ (Fig. 28). For further discussion of adiposity rebound as a predictor of childhood obesity, see part 4.4.2 of this section, below.

**Figure 28** BMI plotted against age for six large-scale surveys of children, showing the ‘rebound’ after age 5 years. Source: Cole et al. (47).
Later in childhood, there is a remarkably consistent relationship between adolescent markers of maturation – namely age at menarche, stage of puberty or peak height velocity – and subsequent fatness (214). Those maturing earlier or more rapidly are at greater risk of obesity. Several studies show that increased fatness earlier in childhood predicts earlier maturation, at least after 3–4 years of age (214), and that this partly explains the relationship between early maturation and greater subsequent fatness (231). Higher activity levels, energy intake and protein intake have been noted in later maturing adolescents (232), but the underlying pathways in the relationships with obesity are unclear.

4.2.3. Weight gain in adolescence
Adolescence is an important period in human development, characterized by significant somatic growth and maturation of secondary sexual characteristics. Because of marked variability in the timing of maturational changes, evaluation of growth based on chronological age alone can be inaccurate or misleading.

The onset of puberty is believed to occur as a consequence of a change in the pituitary–gonadal axis resulting in a dramatic rise in testosterone in boys and oestrogen in girls (138). Somatic growth is orchestrated primarily through the action of growth hormone (GH) and insulin-like growth factors (IGF). Obese children characteristically have accelerated growth. In the early pubertal stages obese children show advanced height and bone age, however their pubertal growth spurt is less pronounced resulting in a reduction in height centile and ultimately adult heights not different from non-obese counterparts. Basal GH concentration, GH response and 24-h GH secretion and pulsatility are usually reduced in obese children; their advanced maturation is thought to be mediated through increased IGF-1 secretion (138).

Pubertal growth spurt is associated with significant changes in body composition (233). Girls tend to accumulate more fat than boys. Fat gain occurs in boys and girls early in adolescence, but then ceases and even reverses temporarily in boys, and continues throughout adolescence in girls. Menarche usually occurs shortly after the peak in height velocity. The rise in serum oestradiol relates temporally to breast enlargement, widening of hips and an increase in body fat. As height velocity decelerates there is an acceleration in fat gain (234). Lean body mass increases rapidly during adolescence to reach a maximum at 20 years. The rise in testosterone with its strong anabolic properties coincides with the rise in lean body mass.

Normal puberty is also associated with insulin resistance, which is compensated for by increased insulin secretion in response to glucose (235). Basal glucose flux is identical in pre-pubertal and pubertal children, although insulin-stimulated glucose uptake in peripheral tissues is lower in pubertal children. During euglycaemic clamp studies, pubertal status does not affect the response of branched-chain amino acids, free fatty acids and B-hydroxybutyrate to low or high doses of insulin. During hyperglycaemic clamp studies, the rise in plasma insulin is twofold higher and the uptake of branched-chain amino acids greater in pubertal children, amplifying the anabolic effects of insulin on protein metabolism. The insulin resistance of puberty is attributed to the rise in circulating GH, IGF-1 and adrenal androgens.

BMI as a measure of body fatness in adolescents is influenced by maturation status, race and distribution of body fat (236). The relationship between percentage body fat and BMI is dependent on the stage of maturation (for equivalent BMI, lower percentage body fat in more sexually mature than less sexually mature), race (for equivalent BMI, whites have a higher percentage body fat than blacks), and waist : hip ratio or waist circumference (for equivalent BMI, central obesity is associated with a higher percentage body fat than peripheral obesity). It is important to consider maturation stage, race and body fat distribution in the interpretation of BMI in adolescents.

Improvement in environmental conditions, primarily nutrition and infectious diseases, is the major cause of earlier maturation (214). The age of menarche has decreased from 16–17 years in the 19th Century to 13–14 years in the 1960s to 1970s in many European countries and 12.8 years by the 1970s in the US. Earlier matures tend to be taller, heavier and fatter than later matures of the same chronological age. In a systematic review, Parsons et al. (214) found a negative relationship between maturation age and adult BMI in 15 out of 21 studies.

Among girls enrolled in a US National Heart, Lung, and Blood Institute study of growth and health, weight, sum of skin-folds and BMI were greatest in early matures and least in late matures (237). Girls who matured early were shorter in early adulthood, despite having greater peak height velocity and post-menarcheal increment in height. Throughout puberty and into adulthood early matures were heavier and had higher BMI. Early maturation nearly doubled the odds of being overweight in girls participating in US National Longitudinal Study of Adolescent Health (238). Overweight prevalence was significantly higher in early matures of all racial groups (41.7% in early vs. 25% in average and 18.7% in late matures), but it was highest among early maturing black girls (57.5%). Early maturation is associated with a greater risk of obesity not only in adolescence, but later on in adulthood (214).

For early and late maturing children, BMI as a proxy for body fatness should be interpreted with caution. Wang & Adair (239) examined the effect of adjustment for differences in timing of maturation on the assessment of overweight prevalence in adolescents from different countries using the WHO international reference. Maturity adjust-
ment increased the estimated prevalence of overweight in countries where girls mature later than the reference population and decreased it slightly in populations where girls mature earlier. While the overall adjustment is small, maturation status should be considered particularly when assessing young adolescents and populations with markedly different maturation rates relative to the international reference.

Persistence or tracking of fatness from childhood and adolescence to adulthood has been demonstrated in a number of studies, although the magnitude of the effect depends on the cut-offs used to define overweight or obesity, the age of initial assessment and the length of follow-up (19). Correlations between childhood and adulthood adiposity are poor to moderate, in the order of 0.30, whereas correlations between ages 13–14 years and 25–36 years vary from 0.46–0.91 for boys and 0.60–0.78 for girls. As the prevalence and degree of childhood obesity increases in populations, the strength of the correlations may also increase.

Adolescence is one of the most vulnerable periods for the development of overweight/obesity. Risk factors for cardiovascular disease, insulin resistance, hepatic steatosis, polycystic ovary syndrome and orthopaedic complications associated with obesity increase and the probability of becoming an obese adult also increases in adolescence. Adolescence seems to be a period for entrainment of obesity-related morbidity (240). Increased morbidity and mortality seen in adulthood has been attributed to adolescent obesity directly, rather than the effects of adolescent obesity on adult weight (241). Although the mechanism is unclear, it is possible that fat distribution patterns established during adolescence play a role. Boys tend to deposit fat centrally and lose fat peripherally as they mature, showing a pattern predictive of diabetes, heart disease, hypertension and hyperlipidaemia in adults.

4.2.4. Normal energy requirements

Considerable attention has been focussed on energy balance and the need to define the energy required for healthy growth. Energy requirements for growth have two components (1) the energy used to synthesize growing tissues; (2) the energy deposited in those tissues, basically as fat and protein, because carbohydrate storage is negligible.

A recent analysis of energy balance in healthy children and adolescents (242) has led to a reappraisal of the recommendations for food supply used by international bodies such as the WHO. The revised proposals for energy intake, based on doubly labelled water methods for monitoring energy intake and expenditure, are shown in Fig. 29 with the previous recommendations.

The proposed new energy requirements are 18–20% lower for boys and girls under 7 years of age, and 12% lower for boys 7–10 years old. From age 12 onwards, the proposed requirements are 12% higher for both boys and girls, assuming moderate levels of physical activity. Interventions such as child feeding programmes, if based upon the previous recommendations, could have led to excess energy intake among target populations, with a potential problem of encouraging the consumption of nutrient-poor, energy-rich diets, and of encouraging excessive weight gain among these children (see also the section Stunting in Childhood, in 4.4.2 below).

4.3. Genetic, familial and gene–environment interactions

Observations in twins, siblings, nuclear families and extended pedigrees have shown that an individual’s chances of being obese are increased when he or she has relatives who are obese. Obesity persistence from childhood to adulthood is also linked to family obesity: data from the British 1958 birth cohort demonstrated that obese children of obese parents are more likely to be obese in adulthood, especially when both of the parents are obese (243).
The relative influence of parental and childhood weight status on the persistence of childhood overweight into adulthood appears to vary substantially with the age of the child. A large retrospective cohort study that exploited medical record data on parents and children indicated that the influence of parental obesity on weight status in early adulthood was stronger from age 2 through age 10 years. By adolescence, the child's own weight status was a far stronger determinant of obesity in early adulthood (48).\footnote{Whitaker found that parental obesity was a more important predictor of offspring obesity in early childhood, but was less important in determining the fatness of children above 9 years of age (48).}

In the Ten-State Nutrition Study of 1968–1970, the relative fatness of children increased systematically from parental combinations of two lean to two obese parents. Generally, the leanest children had two lean parents; the fattest children had two obese parents, with intermediate levels of fatness seen for one lean and one obese parent. Maternal fatness did not appear more obesogenic than paternal fatness (244).

The findings from a study based on the Danish Adoption Register further confirmed the strong influence of genetics in obesity. A comparison of the correlations of adoptees’ BMI with their adoptive and biological parents showed far greater concordance of BMI with the biological parents than the adoptive ones (245).

A series of elegant analyses conducted by Bouchard and colleagues have looked at twin pairs exposed to under- and overfeeding, and indicate a substantial genetic component in weight gain in response to food intake (246) accounting for up to 50% of the variability. The authors suggest that the genetic components are complex and that amount and rate of weight gain are unlikely to be related to a single gene in the majority of cases of obesity (247).

Results of family studies suggest that energy expenditure, in terms of measurements of physical activity and sedentary behaviour, exhibits characteristics that suggest a moderate genetic component. A review of the field by Allison et al. (4) noted that monozygotic twin studies have found the heritability of BMI to be as high as 85%, while adoption and family studies have found heritability in the range 25–50%. The authors suggest that the true figure is likely to be around 70%.

That genes should account for so much of the variation in BMI might lead to the conclusion that the environment has little impact, but this would be erroneous. Genes are best expressed in appropriate environments and with the rise in obesity prevalence being noted in so many countries, it is clear that the environmental conditions for obesity expression are being created in several parts of the world, especially in urban, industrialized regions. As Allison et al. conclude:

‘If we are to be responsive to the obesity problem that the population has as a whole, the environment must be changed to become less obesogenic and more promoting of healthy diets and activity patterns’ (p. 160).

The argument can be posited differently – what proportion of the rise in obesity seen in the population can be attributed to a 'purely' genetic factor, virtually unaffected by environmental stimuli? The review by Allison et al. (4) suggests that some 10% of population may become overweight even in environments that mitigate against weight gain, and a similar proportion of the population might have a strong genetic predisposition to remain thin in obesogenic environments. The majority of the population carry combinations of genes that may have evolved to cope with food scarcity, a genetic predisposition that is maladaptive in an environment of ready availability of calorie-dense food where low energy expenditure is the norm.

The influence of the environment is clearly illustrated in a comparison of weight status of Pima Indians living in the mountain state of Sonora in Mexico and genetically similar Pima Indians living on the Gila River Indian reservation in Arizona. The Pimas who reside in Mexico have an average BMI of 25, whereas Pima men and women living in Arizona have average BMIs of 31 and 36, respectively (248). Similarly, studies of immigrants into the USA from countries with lower obesity rates, such as China and Japan, show that the US immigrants develop higher obesity rates than the population they left, and the immigrants’ offspring continuing to live in the US develop even higher rates of obesity than their parents (249). Similarly, studies of hunter-gatherers in Australia show a raised prevalence of diabetes, hypertension and obesity among those that moved to urban areas compared with those remaining on their homelands (131).

The nascent field of behavioural genetics seeks to understand both the genetic and environmental contributions to individual variations in human behaviour. As it relates to obesity, the primary behaviours of interest are food consumption patterns, sedentary behaviour and physical activity. The complexities and challenges are many, because the behaviour patterns and the obesity itself may reflect multiple genes, and dietary intake and energy expenditure measures are liable to substantial error. The analysis is further complicated by the fact that familial aggregation of characteristics may have both genetic and non-genetic aspects. Dietary, activity and sedentary behaviours have all been demonstrated to ‘run’ in families (221,250,251). Thus our ability to ascribe the degree to which parent and child weight status concordance is due to genes, environment or culture is, by necessity, limited, and estimates of ‘heritability’ (the amount of variation in a population that is attributable to genetic factors) apply only to the specific population studied and only to the environment in place at the time the study was conducted.
The role of the environment in the linkage between parent and child overweight is difficult to study directly, although two lines of evidence suggest that there is a substantial non-genetic component. First are studies that document dramatic increases in the prevalence of childhood obesity as developing countries adopt the diets and physical activity levels of populations in the industrialized economies (67,252). Studies in developing countries have also documented the coexistence of underweight and overweight within the same family (253), which implies that environmental rather than genetic factors are involved. Second are migrant studies, where obesity rates in second-generation children are seen to significantly exceed rates in first generation children (254). Again, environment factors are likely to be responsible.

4.3.1. Specific syndromes linked to obesity

Although the vast majority of obese children and adolescents do not have a specific syndrome, there are about 30 inherited disorders in which childhood obesity is a clinical feature. These may be associated with mental retardation, dysmorphic features and organ-specific developmental abnormalities. These disorders explain a very small proportion of childhood obesity, representing at most 1–2% of total cases, but practitioners should be particularly alert for indications of intellectual disability, short stature and unusual stigmata. Children with more extreme levels of obesity are best sent to tertiary referral centres where diagnostic facilities are more readily available and appropriate treatment can be initiated.

Down syndrome. Children with Down syndrome may become obese as they progress through rather deficient pubertal development. Autoimmune thyroiditis is a common problem in Down syndrome and thyroid status should be checked in an overweight child with Down syndrome. Associated problems such as congenital heart disease will affect the predisposition to obesity, and management should be adapted for the individual’s needs. Growth curves for the ‘normal’ growth of children with Down syndrome exist.

Prader-Willi syndrome. This is the most common syndromal cause of human obesity with an estimated prevalence of about 1 in 25,000. The Prader-Willi syndrome is characterized by diminished fetal activity, obesity, hypotonia, mental retardation, short stature, hypogonadotropic hypogonadism, and small hands and feet; the diagnostic criteria have been summarized by a consensus group (255). It is caused by deletion or disruption of a paternally imprinted gene or genes on the proximal long arm of chromosome 15. Although the precise genes involved are not known, there are now reliable laboratory tests for the diagnosis of this syndrome (256,257), which has implications for the management of such patients.

In infancy these children may present with feeding difficulties. This can make it difficult for carers to move from a situation of dealing with failure to thrive to a situation of controlling obesity. Low IQ and behavioural disturbances exacerbate the difficulties in altering lifestyles in these children who have a strong predisposition to severe overweight and often considerable determination and obstinacy. Lifestyle advice should be offered at diagnosis. Growth hormone treatment at levels of 1.0 mg m⁻² in school-age children has been associated with lowered body fat, increased lean body mass, increased growth velocity and increased resting energy expenditure over periods of at least 4 years. Growth hormone treatment started in early childhood is also associated with normalization of growth and body proportions, but not to the extent of leading to totally normal fat distribution and body composition. Growth hormone treatment has also been associated with improved emotional, behavioural and cognitive functioning providing positive effects on psychosocial as well as anthropometric status. There is need for more research on the age of introduction, level of treatment in terms of dose and frequency, and optimal duration of treatment, as well as on which children will benefit significantly from the treatment.

The prevalence of orthopaedic complications, type 2 diabetes mellitus, hypertension and other secondary problems increases with age and contribute to a low quality of life in later adolescence and early mortality.

Duchenne muscular dystrophy (DMD). Boys with DMD frequently become obese from around 7 years of age with a peak prevalence at 13 years (>50%), probably as a result of both the deteriorating muscles being replaced by fat and positive energy balance due to inactivity, poor dietary control associated with low IQ and, sometimes, overindulgence of children with a miserable prognosis. Obesity is centralized body fat suggesting that the obesity is not totally related to muscle degeneration. Undernutrition tends to develop in later adolescence (from 14 years of age) and affects >50% boys of 18 years. Carers thus need to be aware of the child’s current state in order to offer appropriate advice on diet and activity.

Albright hereditary osteodystrophy (AHO). This is an autosomal dominant disorder due to gene mutations that decrease expression or function of G-alpha-S protein. Maternal transmission of the mutations leads to short stature, obesity, skeletal defects and impaired olfaction plus resistance to several hormones (e.g., parathyroid hormone) that activate G-alpha-S in their target tissues (pseudohypoparathyroidism type IA), while paternal transmission leads only to the pseudopseudohypoparathyroidism phenotype (258).
Fragile X syndrome. This is characterized by moderate to severe mental retardation, macro-orchidism, large ears, prominent jaw and high-pitched jocular speech associated with mutations in the FMR1 gene (259). It has been suggested that a reasonable estimate of frequency is 0.5 per 1000 males. Not all children with fragile X syndrome will become obese.

Bardet-Biedl syndrome (BBS). BBS is a rare (prevalence <1/100 000), autosomal recessive disease characterized by obesity, mental retardation, dysmorphic extremities (syndactyly, brachydactyly or polydactyly), retinal dystrophy or pigmentary retinopathy, hypogonadism or hypogenitalism (limited to male patients) and structural abnormalities of the kidney or functional renal impairment. BBS is a genetically heterogeneous disorder that is now known to map to at least six chromosomal regions (260). As yet although some causative genes have been identified, in no case has a link between a certain gene or protein and the disease been established.

Single gene disorders causing childhood obesity. Five genetic disorders result in morbid obesity in childhood without the developmental features characteristic of the above-mentioned multi-organ syndromes of childhood obesity. The first monogenic human obesity syndrome to be reported was congenital leptin deficiency. Children are severely hyperphagic, constantly demanding food, with an intense drive to eat, but without any of the clinical features suggestive of the recognized childhood obesity syndromes. They have impaired T-cell mediated immunity thus are at risk of frequent infections and have hypogonadotrophic hypogonadism and so fail to undergo pubertal development (261). A similar clinical picture is seen in children with defects in the leptin receptor (262). Defects in pro-opiomelanocortin (263) and pro-hormone convertase 1 (264) also cause severe childhood obesity and other endocrine and metabolic problems. Defects in the melanocortin 4 receptor cause a severe obesity syndrome that is dominantly inherited and may account for up to 5% of patients with severe, early-onset obesity (265). The nature of mutations in this gene determines the severity of the disease phenotype.

Endocrine disorders. Cushing’s syndrome, hypothyroidism, hyperadrenocorticism, hypophosphataemic rickets, growth hormone resistance and other endocrine disorders can be associated with some degree of obesity. Short stature and growth retardation are characteristic of many such disorders, as opposed to height over-growth commonly seen in over-nutrition. In adolescents the diagnosis of short stature may be harder to distinguish from normal cessation of growth.

The degree of obesity found in these children may be secondary to other factors, for example poor vision or other physical disability may lead to reduced physical activity, while family indulgence of the child may encourage excess calorie intake.

4.3.2. Testing for syndromic childhood obesity
Currently obese children with the classical obesity syndromes are often looked after by paediatricians and geneticists in tertiary referral centres. There is a growing need for wider access to diagnostic facilities and multi-disciplinary team-based management programmes for the care of these specific patients. Increasing understanding of the physiology of these syndromes will enhance options for therapy, such as the use of growth hormone in patients with Prader-Willi syndrome (266).

The discovery of single gene defects as a cause of childhood obesity has biological and clinical implications that are greater than the rarity of the individual diseases might suggest. Most importantly, at least one disorder, namely leptin deficiency, is amenable to treatment. Three lepandindeficient children have been treated with daily subcutaneous injections of recombinant human leptin for up to 4 years with sustained, beneficial effects on appetite, fat mass and hyperinsulinaemia (267). Leptin treatment has reversed the immune defects and risk of infection seen in these children and allows the appropriate development of puberty. Such treatment represents the first, rationally based, hormone replacement therapy for any form of human obesity.

Assessment of patients with severe obesity in childhood might in the future include the measurement of serum leptin to exclude leptin deficiency, which is treatable. This is currently available only as part of research studies and, given the rarity of these disorders, a general recommendation for testing may not be justifiable at present. However, training and education of health care workers is important so that such children are referred to the appropriate centres for further evaluation and genetic counselling. As more is learnt about the genes that regulate body weight and more syndromes are described, it is likely that the need to evaluate severely obese children in recognized centres will grow and close collaboration with academic centres with experience in this field is needed to ensure that the benefits of laboratory research are made available to the patients that need them.

4.4. Children at risk of weight gain
Given the high level of genetic predetermination of obesity risk (see above) it is clear that large numbers of children are likely to develop excess body weight wherever the situation permits it. However, there appear to be certain physical and social environments that particularly encourage weight gain, and certain groups of children that might
be at special risk. This section looks in more detail at the risk factors that encourage the accumulation of excess weight in childhood.

4.4.1. Individual children at special risk
Clinicians and health professionals treating children with specific conditions should be aware that some children are at particular risk of developing excess bodyweight. These include the following.

Children with physical disability. The effect of physical disability on a child’s nutritional status is very dependent on the nature and severity of the problem. For example, children with cerebral palsy may become obese if they have hypotonic cerebral palsy and are relatively inactive and lethargic. Children with spastic or choreo-athetoid cerebral palsy who have great difficulty in locomotion are commonly underweight. Severely mentally handicapped children may have growth problems and poor weight gain in association with their brain damage. Management of these children is often difficult because there may be a reluctance by relatives to put the burden of obesity control on an already disadvantaged child and physical disability may make it almost impossible to increase energy expenditure.

Adolescents with type 1 diabetes. Adolescents with type 1 diabetes mellitus commonly gain excess weight, usually associated with poor diabetic control, blood glucose instability and high insulin dosage. Weight loss in type 1 diabetes is not easy because of the need for insulin and regular dietary intakes. Poor diabetic control may be associated with episodes of hypoglycaemia with over-eating to compensate, or with hyperglycaemia and over-dosing with insulin to control blood sugar levels. Raised levels of glycated haemoglobin HbA1c (indicating poor control) tends to be maintained. As with most forms of treatment (see below) initiatives need to be developed that involve both home and school environments.

Children treated with other centrally-acting drugs. The use of anti-depressants, anti-psychotics and centrally-acting drugs designed to assist behaviour control may encourage reduced physical activity and increased sedentary behaviour, and there may also be changes in dietary patterns, such as more regular meal attendance and greater food consumption – all of which can lead to weight gain. Weight gain is also associated with other centrally-acting medications including risperidone and possibly methylphenidate (Ritalin) (270,271).

Children treated with glucocorticoids. Increased weight gain is a well-recognized complication of long-term systemic glucocorticoid therapy for paediatric disorders such as nephrotic syndrome, juvenile rheumatoid arthritis, inflammatory bowel disease, chronic severe asthma and other inflammatory diseases. Obesity is also one of the side-effects that have been reported in children on high-dose inhaled steroid therapy (272).

Children with psychological problems. Children with mild learning difficulties and/or those who have difficulty making friends may show a predisposition to obesity. Retreat, withdrawal and lack of self-confidence may lead children to more sedentary lives and their withdrawal to the solace of computers or television. These children are often presented to clinicians as failing at school because of their obesity or having social difficulties because of their obesity. Whilst some obese children do suffer significant bullying, many of these children had psychosocial and learning problems before they became obese. Successful reduction in fatness can induce very welcome improved self-esteem, but weight reduction in children with psychosocial and learning problems can be extremely difficult to achieve and maintain. As with most forms of treatment (see below) initiatives need to be developed that involve both home and school environments.

Children with eating disorders. Bulimia. The evidence that bulimia is linked to obesity, or that obese children are more prone to bulimia, is not strong. However, the presence of bulimia in an obese child may contribute to poor diet control. Anorexia. Despite common concern that dieting in childhood obesity increases the risk of anorexia nervosa, there is no persuasive evidence that this is the case. There is a risk, however, that a popular emphasis on dieting as a means of managing body shape may contribute to the development of anorexia in psychologically at-risk non-obese children – i.e. the social pressures that discriminate against the obese child may encourage vulnerable non-obese children to develop eating disorders. A greater emphasis on health rather than weight control per se might help to resolve this problem.
Survivors of cancer. There is some evidence that children that are being treated for cancer, or who have survived cancer, may be at increased risk of obesity (273–275). Cranial irradiation and growth hormone deficiency are predictors for later weight gain in this population (273,276). Additional biological and social factors may also be important in the pathogenesis of obesity.

4.4.2. Population groups at higher risk for obesity
Apart from children with specific medical conditions that lead to obesity, or children whose treatment for other conditions may lead to obesity as a complication, there are identifiable risk factors within the population of normal children that increase their risk of becoming obese.

Ethnicity. It is a common impression that schoolchildren from non-Caucasian backgrounds living in Westernized societies have greater propensity for developing obesity than white Caucasian children, but when socio-economic circumstances and parental education are taken into account, the differences may not be great. In the USA, for example, African-Americans and Hispanic-Americans appear to contribute more to the obesity epidemic, with more rapid rates of change in their populations, than does the white American population (69). In the UK, children from South-East Asian backgrounds tended to have a lower ponderal index than white children, but showed higher insulin levels and a stronger relationship between adiposity and raised insulin concentration (51). A survey in the Netherlands of Turkish and Moroccan immigrant groups found the children to be more at risk of overweight than Dutch children (277). These racial/ethnic disparities may be partially but not fully explained by family income and other social inequalities (3).

A compilation of nationally representative surveys of Australian children and adolescents in 1995 and 1997 showed that there is a higher prevalence of overweight and obesity among schoolchildren with backgrounds of Middle-Eastern or continental European origins, compared with children from either Asian or English-speaking backgrounds (278). Cultural issues are important for management and prevention. They may influence concepts of the attraction or risks associated with childhood obesity and thus willingness of families to comply with therapeutic advice. They also influence lifestyles and self-esteem of the obese within a society. They can have considerable impact on what should be included in dietary recommendations and on compliance with activity recommendations. The ease with which children can participate in activities away from home can be influenced by cultural norms and social integration of minority communities. Girls are probably affected particularly in relation to this latter point.

The prevalence of complications of obesity such as type 2 diabetes mellitus and hypertension amongst populations from the Indian subcontinent, together with the lower BMI levels for risk of complications in adults, have implications for the development of obesity in children from the Indian subcontinent. There is need for more research on body composition in ethnic minorities in different countries and on the outcomes for childhood obesity in these groups.

Parental obesity. As noted at the start of part 4.3 of this section, above, the risk of a child becoming overweight increases with parental overweight and obesity (48,243). It is likely that the family association is due partly to genetic factors and partly to shared lifestyles, i.e. diets and patterns of activity. In some cases obese parents show less concern than average for their child’s obesity, although in other cases the opposite may be true. Lack of parental concern probably leads to less active attempts at management and exacerbation of obesity. It would seem wise to advise obese parents very carefully about diet, increased activity and reduced inactivity as early in a child’s life as practical. Such advice should be continued throughout childhood in ways appropriate to the children’s age and children and parents’ understanding.

Some data suggest that the parent–child fatness relationship may be stronger between mothers and their offspring than fathers and offspring, and that the mother–offspring relationship strengthens as the child gets older (279). Other data shows that parental obesity becomes less important with increasing age of the child, and is a more important predictor of offspring obesity earlier in childhood (<6 years) (48). Importantly, parental obesity may influence tracking of the offspring’s own obesity, which is much stronger if both parents are obese (243,280). Recent work has begun to look at lifestyle factors that might contribute to the parent–child fatness relationship, and suggests that obesogenic and non-obesogenic family clusters can be identified based on parents’ diet and activity patterns. Children in obesogenic families are not only fatter but show greater increases in BMI or skin-fold measures at age 5–7 years (281).

Maternal diabetes. As mentioned earlier in this report, the offspring of women who had diabetes during pregnancy are more likely to become obese later in childhood and to have a higher prevalence of impaired glucose tolerance than the offspring of women who were non-diabetic. The most convincing evidence is an increased risk of overweight among offspring born after the mother developed diabetes compared to the siblings born before (282). A study by Silverman et al. found the insulin levels in the amniotic fluid during pregnancy showed a close association with the offspring’s risk of impaired glucose tolerance in adolescence as well as with their BMI in adolescence (after adjusting
for the correlation with the mother's BMI (283). Impaired glucose tolerance was found in 36% of offspring of diabetic mothers.

Although maternal diabetes during pregnancy results in offspring with higher birth weights, Silverman et al. found that heavier babies tend to revert to normal weights by 1 year of age (283), but then show increases in BMI after about age 4 years compared with national reference standards, i.e. the children showed an early adiposity rebound (see below). This normalization followed by departure from normal implies that these children's obesity is metabolically programmed during their intrauterine experience, rather than reflecting a persistence of obesity acquired before birth.

Similar links have been shown in the Bogalusa Heart Study, which found that children (average age 15 years) of diabetic mothers showed greater body fatness, higher blood pressure and raised fasting levels of blood glucose, insulin, glucagon and triglycerides (284). The latter measures remained significantly raised after controlling for the higher BMI levels in these children.

**Smoking in pregnancy.** Maternal smoking has been linked to subsequent adiposity in children (227,285). Although several confounders may limit the interpretation of the results, von Kries et al. have shown a dose-dependent relationship between maternal smoking during pregnancy and prevalence of overweight and obesity in children aged 5–7 years, which could not be explained by social class, mother’s weight or child's birthweight (286). In this study, smoking after pregnancy appeared unrelated to childhood obesity, suggesting that intrauterine exposure to the products of smoking, rather than family lifestyle factors, were instrumental in raising the risk of childhood obesity.

**Low birth weight.** As noted earlier, a U-shaped relationship between birth weight and subsequent risk of obesity appears to apply, with the heaviest babies and the lightest being at risk of excess weight gain during subsequent childhood and adulthood. An excess risk of obesity has been observed in young Dutch adults who had been born to mothers who had faced famine conditions in early pregnancy (287) and several reviews have noted an association between poor intrauterine growth and later obesity (288,289). Considerable evidence now exists that obese children and obese adults who had low birth weights are more vulnerable to both coronary heart disease and type 2 diabetes than similarly obese people who had higher birthweights (290).

The links between the intra-uterine experience of the foetus and the subsequent risk of coronary heart disease in adulthood may be mediated by several factors. Low birth weight appears linked to the development of stunting and abdominal obesity (see below) and the metabolic syndrome in later life. Stunting at birth appears closely linked to insulin resistance in pre-pubertal children, with the highest insulin resistance among those children who have become the heaviest by this age (291).

The relationship between birthweight and risk of subsequent heart disease or diabetes among adults in the Indian subcontinent is more pronounced among urban populations than rural ones, although birthweight records would indicate that lower birthweights are more common among rural populations. Yajnik has demonstrated that a combination of low birthweight and weight gain in childhood led to the highest risk of developing insulin resistance and cardiovascular risk factors (49,292). Low birthweight babies in Indian populations appear to have depleted muscle mass and visceral mass, but preserved subcutaneous fat, and are prone to gain weight by increasing their central obesity. This high-risk pattern was associated with a lack of fruit and vegetables in the mother's diet and to indications of malnutrition in the mother's own early life growth, such as shortness of stature and small head circumference (292). It can be appreciated that the risk factors for developing obesity, diabetes and coronary heart disease can be detected in previous generation's nutritional status. It can also be appreciated that supplemental feeding programmes for low birthweight babies may encourage rapid weight gain, exacerbating their risk of subsequent chronic disease.

**Stunting in childhood.** Stunting (short height for age) affects one-third of all children aged under 5 years globally (i.e. around 270 million children), most of them in less developed or transitional economies (293).

Evidence from several surveys has shown the co-existence of stunting and overweight or obesity in the same child and/or among other members of the same household, in urban areas in developing countries (60,253,294,295) and poorer communities in developed countries (296). The prevalence of overweight among stunted children has been estimated at 45% in the 1994–5 Russian Longitudinal Monitoring Survey of children aged 3–9 years, and approximately 20% in the 1991–3 China Health and Nutrition Survey (60). The incidence was lower in surveys of stunted children in South Africa (13% overweight) and Brazil (4% overweight). Longitudinal studies suggest that stunting increases the risk of central obesity (e.g. as measured by waist : hip ratios) but does not raise the risk of overweight (assessed by BMI) in later childhood (297) or adulthood (298). A longitudinal study in Guatemala found that although stunting in childhood was not linked to BMI, some 15–20 years later, children with severe stunting had significantly greater abdominal fatness (waist : hip ratios) when they were adults, and the effect was most pronounced among women who had migrated to urban areas (298). There is also some evidence that stunting during early
childhood increases the strong link between overweight and high blood pressure in later childhood (299).

The timing and duration of the physiological insults that lead to retarded linear growth are likely to have different physiological consequences. Growth retardation is one feature of a complex syndrome including developmental delay, impaired immune function, reduced cognitive function and metabolic disturbances that may raise the risk of obesity and hypertension (300).

The suggestion that stunted children may be less likely to undertake physical activity, and that obesity is a corollary, does not appear to be supported by the evidence – a study of energy expenditure among stunted and matched control children in Brazil using doubly labelled water showed no association between stunting and resting energy expenditure or total energy expenditure (301). The study noted that girls had a lower total energy expenditure than boys, which the authors suggest may help to explain the higher risk of obesity in stunted adolescent girls and women in urban areas of developing countries.

An alternative suggestion is that stunting may increase susceptibility to weight gain, especially in children consuming diets relatively high in fat (302) or where extra food is available opportunistically (303). Physiological mechanisms that are triggered by famine and chronic undernutrition might encourage excess weight gain when exposed to environments where high energy foods are plentiful (253).

Prevention of obesity in supplementary feeding programmes. Underweight children are usually stunted, thus most malnourished children will be of low weight and length for age, but will have a near normal weight for length/height. They are underweight and stunted but not wasted. Recovery in length for age is likely to be incomplete if nutrition improvement occurs after 24–36 months of age, therefore these children when given additional food will gain significantly more weight for age than length for age, but will have a near normal weight for height/length. They are underweight and stunted but not wasted. Recovery in length for age is likely to be incomplete if nutrition improvement occurs after 24–36 months of age, therefore these children when given additional food will gain significantly more weight for age than length for age, raising the risk of overweight (304).

Food supplementation of underweight children may induce weight gain, while length deficit may not be reversed. If protein-energy malnutrition (PEM) prevention programmes are implemented, stunted children may become obese (overweight for height/length), while remaining underweight for age. Unless underweight is universal, supplemental feeding programmes should use targeting strategies to prevent providing excess food to those that are within the normal range of weight for height/length. Most PEM prevention programmes use food supplements that provide ample energy and protein, but may be deficient in micronutrients, thus limiting linear growth. Cereal and dairy foods are poor in zinc, vitamin A and iron, and the fibre content of cereals may inhibit micronutrient bioavailability, while energy intakes may exceed energy needs. The quality of the foods provided is crucial in pre-school and school feeding programmes; fruits and vegetables should be included in the diet of children to secure micronutrients and prevent energy excess, while zinc and iron fortification of fat-reduced milk may also be valuable where stunting and obesity co-exist (304).

Early adiposity rebound. The tendency for indicators of adiposity, such as BMI, to fall from around the age of 1 year, only to rise again around the fifth year, has been noted earlier in this section. Several studies have shown that the earlier the rebound occurs the greater the risk of subsequent obesity (214), although what drives the timing of the adiposity rebound remains obscure.

One study has suggested that earlier adiposity rebound is induced by thinness at around the age of 1–2 years, and this is linked to later risk of diabetes (305). At this age it appears to be the thin infant who is likely to become overweight and diabetic in later life rather than the fat one, an important finding for public health policy which might otherwise assume that fat 2-year-olds are at greater risk of later ill-health than thin ones (290). Interestingly, one study reports that girls with a higher BMI at 3 years of age experience adiposity rebound later (no relationship in boys), and that in both sexes, an earlier rebound occurred in children that were taller at age 3 years (306).

It is not clear whether the importance of the early adiposity rebound lies in a biological mechanism for enhanced weight gain during childhood as a causative factor, or whether it is merely indicative of a tendency to gain weight more rapidly than other children – due to prevailing genetic or environmental conditions. Dietary variables do not appear to predict adiposity rebound (307), although this requires confirmation in further studies. The effect of an early adiposity rebound may be independent of BMI at rebound (greater in those with an early rebound) or parental obesity (more prevalent in those with an early rebound) (308). Age of rebound, however, may add little to the prediction of adult obesity, if BMI at age 7 years (i.e. after rebound) and height in childhood are known (309). For a given BMI in childhood the taller child is more likely to be an obese adult (310).

Social deprivation. In most Western societies children from socio-economically deprived environments have a greater risk of obesity than those from more affluent groups (214). The reasons for this are not clear although dietary differences can often be shown. Low self-esteem and feelings of disempowerment may be relevant to how deprived families cope with children who are overweight. However conditions such as a depressed environment with nowhere safe for children to play; a lack of opportunity for away-from-home activities so television watching becomes the only leisure activity; and distance from shops where fruit, vegetables and low-energy density
foods are affordable and readily available, may all contribute to obesity prevalence.

It is possible that some of the effect of socio-economic status is mediated through an individual’s education level, but data from the UK have demonstrated both factors to be independently related to adult BMI, suggesting that circumstances in early life have an enduring and important effect (311).

In countries with less industrialized diets, the patterns of obesity related to socio-economic status of the family are more complex, with a tendency for urban children and children in high-earning families to be more at risk of excessive weight gain. In China, for example, children from advantaged backgrounds tend to be fatter than those from disadvantaged backgrounds (280).

In both industrial and developing countries, access to an energy-rich, nutrient-poor diet appears most closely linked to weight gain, and those socio-economic groups with greatest access to such diets are likely to have children most at risk. Access to obesogenic diets is determined by many factors, including family income, access to transport, the distribution and marketing practices of the producer companies, and lack of access to healthier alternatives.

There is little objective evidence available on the social issues leading to obesity. Family size, position of the obese child in the family, lone- or both-parent families have all been found relevant to prevalence of childhood obesity in some studies. In developing countries underweight may co-exist with overweight within the same household; one estimate (for households in Asia) suggests that the coexistence of both forms of mis-nutrition is up to 15% of all households, and that between 30% and 60% of all households where one member is underweight will have another member who is overweight (312). Public health policies aiming to reduce underweight may inadvertently serve to encourage excess weight (313).

Family and school functioning. Aspects of family structure (single parent families, number of siblings, birth order of child, age of mother at the birth of her child) have been only rarely examined and the results are inconsistent (214). Family functioning, which may also be linked to behavioural and psychological factors, has also been little investigated, but a Danish study reported that parental neglect and lack of parental support of children were both risk factors for developing obesity in early adulthood (314). In the Danish context, the quality of the home, the local environment and the level of ‘care’ within a family may be more relevant to the development of obesity than the family’s size or specific economic status.

Psychological factors in parents may also be important; children with parents who show high levels of dietary disinhibition (abandonment of control of dietary intake in the presence of certain external food cues), especially with concurrent dietary restraint (conscious restriction of diet to control weight) show greater increases in fatness (315). In infancy, a vigorous feeding style (316) and distress at limitations (317) were related to increased subsequent fatness, and soothability to decreased fatness. In childhood, predictors of fatness included decreased adaptability, increased intensity and withdrawal (318), various aspects of self-esteem and family functioning (181,319), and feelings of inadequacy (320).

At school, studies have found a higher percentage of overweight children among low-achievers, and this association between overweight and under-achievement persists into adulthood (321). A study in Thailand found that being or becoming overweight in adolescence was associated with poor school performance, although this was not the case for younger children (322). In young adults, intelligence test scores and educational levels are higher among those with below median BMI (323).

4.4.3. Environmental risk factors

The areas that have attracted most attention in terms of possible realms for the management or prevention of obesity are those concerned with the environmental conditions that encourage the consumption of greater food energy than is required, or that encourage sedentary behaviour or discourage physical activity.

In modern industrialized societies, food and drink are more available and affordable than ever before, fewer people have jobs requiring hard physical labour, car ownership has increased rapidly and homes have labour-saving gadgets. Yet human metabolism evolved under very different conditions with a sparse and erratic food supply and huge physical demands for survival, which has selected individuals with a ‘thrifty’ genotype. This genotype is ill-suited for the modern world. Excessive fat storage, leading to obesity, is the default situation unless specific action is taken.

The recent WHO/FAO expert consultation on diet, nutrition and the prevention of chronic diseases recognizes this link between the wider environment and the growing problem of obesity in adults (6). It is probable that similar factors are linked to the rise of overweight in children; for example, a decline in walking to school and a rise in snack food consumption and the popularity of fast-food outlets. Within this ‘obesogenic’ environment there are a number of factors that warrant specific consideration with respect to the risk of overweight in children and adolescents. It is also important to consider, however, the micro-environment created in the home. For younger children in particular the family environment plays an important role in determining their risk of obesity, for example parental physical activity levels (214,279), the family’s eating behaviours (48,279) and television viewing habits (243).
4.4.4. Diet

Energy intake is one side of the energy balance equation, thus dietary factors are obvious candidates as risk factors for obesity. Despite more general acceptance that the post-weaning diet is involved in obesity development, evidence from longitudinal studies is sparse, and findings are variable. It would seem logical that the rise in obesity prevalence might be partly due to increases in energy intake, but paradoxically, in the US at least, while the prevalence of obesity in adolescents has doubled (16) energy intakes (in adolescents) have apparently decreased (324). There are, however, concerns about the accuracy of measures relying on reported food intake. Food disappearance data suggest that energy intakes have actually increased while reported food intakes show a decrease (325).

Infant feeding. Several recent large studies have examined whether breast-feeding might have a protective influence on subsequent childhood obesity. Most studies suggest a protective effect of breast-feeding on obesity (286,326,327), and on the risk of type 2 diabetes during childhood and adolescence among high-risk groups (328), but others argue that the apparent effect may be due to confounding factors such as social class, maternal fatness, maternal diabetic status, maternal reluctance to breast-feed, or infant birthweight (329–332).

von Kries and co-workers’ study of 10 000 children in Bavaria found significantly greater proportions of those children who had been formula-fed as infants were overweight at the age of school entry (5 or 6 years old), compared with those who had been breast-fed (286). The protective effect of breast-feeding was dose-dependent, with better protection against excess weight gain among those children with the longest duration of breast-feeding as infants. The authors acknowledge, however, the potential role of confounding factors, including social class, smoking during pregnancy and general family dietary habits.

A study by Armstrong & Reilly of 32 000 children aged approximately 3.5 years found obesity to be lower in breast-fed children, after adjusting for socio-economic status, birthweight and gender (327). A longitudinal study by Bergmann et al. of a cohort of nearly 1000 infants found no difference in BMI at birth, a raised BMI among breast-fed babies at 1 month, but after 2 months a raised BMI and increased skin-fold thickness among bottle-fed babies compared with those that had been breast-fed for 2 months or more (333). For the next year, the bottle-fed babies had a consistently higher risk of being overweight or obese. At 18 months the two groups showed little difference in skin-fold thickness, but the differences emerged again, with bottle-fed babies having significantly raised risk of showing excess weight at age 2 years through to age 6 years (the end of the study period). The mother’s smoking behaviour, BMI and socio-economic status had a strong influence on the child’s risk of overweight, but the differences between bottle- and breast-fed babies remained after controlling for these factors.

Two further studies have been published in 2003, one from UK data showing no significant protective effects of breast-feeding, and one from Brazil showing protective effects when breast-feeding was of 3–5 months duration, but not for shorter or longer periods (334,335). A recent review raises concerns over the designs of the trials that report on breast-feeding and subsequent obesity (336).

The introduction of solid foods can lead to excess energy intake by increasing the energy density of the diet. The duration of breast-feeding and the age of introduction of solid foods might thus influence the infant’s regulation of energy intake and consequent weight gain, with earlier introduction of solid foods leading to more rapid weight gain in this age group than continued breast-feeding, or possibly continued bottle-feeding.

The protein content of the infant’s diet has also been indicated as potentially raising the risk of early excess weight gain (213) and it is possible that bottle-feeding or early weaning may specifically increase protein consumption in this age group.

Macronutrients and energy density. ‘Energy density’ reflects the energy content of foods, usually expressed per unit weight. Energy density (measured per unit weight of food) is highest in foods containing high levels of fat. However, energy density can also be viewed as an inverse of nutrient density, i.e. a high level of energy for a given level of non-calorific micronutrients such as vitamins or essential minerals. In this view, energy density will also be high in foods containing significant amounts of refined carbohydrates such as sugar, while energy density will be relatively low in foods such as fresh fruit and vegetables, lean meats, pulses and wholegrain products.

There are few laboratory studies of energy density in foods in relation to children’s energy balance. Research in young children has shown relatively good compensation for changes in the energy density of meals (281), but long-term studies are lacking. Moreover, there are few studies of the effects of energy density on appetite control in school-age children.

A study by Bowman et al. (337) found that on those days when children consumed fast food products their diet was likely to be less healthful than on other days – the energy levels were higher, the energy density per gram was higher, the fat intake was higher and the fruit and vegetable intake lower. Figures show the consumption of fast food by children has increased by 300% in two decades (338). In an analysis of typical foods served at fast food outlets in the UK, Prentice & Jebb (339) found the food to have greater energy density (by weight) than that found typically in UK
diets, and to be more than twice the energy density of foods recommended for healthful diets.

Outside of the controlled conditions of a laboratory, cross-sectional surveys of food intake in children (e.g., in the UK) show that the mean proportion of fat in the diet of children is lower than adults and close to recommended intakes, but there are large variations in fat intake between children (340). Overall these studies indicate a positive association between percentage energy intake as dietary fat and measures of body weight (341–344), but in longitudinal studies the results are less clear (345–348). These free-living studies are often hindered by the mis-reporting of intake, which may be different between macronutrients, as well as differences between studies in the estimation of body fatness, for example using BMI versus skin-fold thicknesses.

The type of carbohydrate in the diet may affect energy density but has received relatively little attention. An analysis of the diet of pre-school children confirmed the positive relationship between dietary fat and energy density and the reciprocal relationship with carbohydrate. Those with the highest energy density also consumed less sugar (349). However this analysis did not exclude sugars consumed as drinks, which may confound the analysis (see below). Dietary surveys show low consumption of fruit and vegetables among young people increases the energy density of the overall diet.

There are reports of an association between high protein intakes in early childhood and a relatively early 'adiposity rebound', which may increase the risk of being obese in childhood and adolescence (348) – the authors suggest a possible mechanism for the protective role of breast milk (relatively high in energy from fat) and propose that a diet high in protein during infancy may increase the risk of adiposity and other pathologies in later life. In older children, there is some evidence showing a secular increase in energy from protein in the USA, 1973–1988 (350), as well as an increase in carbohydrates, and a decrease in total energy from fats. The authors noted that 'few children meet the prudent diet recommendations'.

**Soft drinks.** Energy consumed as drinks should be considered separately from solid food because experimental studies suggest that the post-ingestive effects on appetite may differ. Soft drinks make a growing contribution to the diet of young people, even young children (351). The quantity of soft drinks consumed, especially carbonated soft drinks increases with age and can account for the largest single source of non-milk-extrinsic sugar intakes among young people (340).

One prospective study has reported a positive association between consumption of sugar-sweetened drinks and obesity in 11–12-year-olds over 19 months follow-up (352). Those consuming more soft drinks, however, had other dietary differences compared with those consuming fewer soft drinks, and the association may not be related to soft drink intake *per se*, but may instead relate to broader dietary or lifestyle habits associated with soft drink consumption. In the above study, increased 'diet' (low calorie) soft drink consumption was negatively related to obesity incidence, but this association may be confounded as these drinks may be preferred by individuals trying to control weight.

In a study of adults, there was a significant increase in weight following a 10-week intervention with increased sugar-sweetened foods (80% of which were beverages) and a decrease in weight in those consuming artificially-sweetened foods (353). There is no comparable medium- or long-term study in children. In a study of children given a single meal accompanied either by a sugar-sweetened drink or an aspartame-sweetened drink, there was no significant difference in energy consumed from the other foods at the meal, and the children consumed more energy in total when the sugar-sweetened drink was served (354).

**Eating patterns.** Babies and young children characteristically have frequent eating episodes. In most Westernized societies, this behaviour has generally been replaced with a ‘three-meals-a-day’ culture as children get older. However, there is an increasing tendency in these industrial societies for more frequent and less well-defined eating occasions in both adults and young people (355) with increases in ‘grazing’ or snack food consumption at more frequent or irregular intervals.

Experimental research in adults has shown that under isoenergetic conditions changes in the number of eating episodes per day has little effect on body weight. However outside of controlled laboratory conditions differences in eating frequency may also be associated with differences in the amount or types of food and drink consumed. A series of studies by Marmonier *et al.* on adults who were being continually monitored for blood glucose, insulin and fatty acid composition for a period of 3–4 h following a meal have shown that snacks of 1 MJ delayed the request for the next meal by less than an hour, and did not reduce the energy consumed at that subsequent meal (356–358) implying that snack eating can be a significant contributor to positive energy balance and subsequent weight gain. Self-reported snack consumption can be misleading: a study of obese women found no difference in self-reported habitual consumption of sweet foods compared with women of normal weight, but analyses of their saliva showed a highly significant increase in levels of mutans streptococci (*P* < 0.0001) indicating a higher level of sugary foods had been eaten (359) especially pre-menstrually.

The impact of snacking on body weight may also be influenced by the types of food consumed as snacks. Outside of the home, the types of foods commonly consumed as
snacks are often high in fat or high in carbohydrates (sugar and/or starch). In one of the studies by Marmonier et al. (337), isocaloric snacks of 1 MJ were effective in delaying a request for a subsequent meal differentially according to the principal macronutrient in the snack: high fat and high carbohydrate snacks delayed meal requests by an average of 25 and 34 min respectively, while high protein snacks delayed meal requests by an average of 60 min. It is worth noting the authors’ comment that a 1 MJ snack might have been expected to delay meal requests by 200 min according to the subjects’ average basal energy expenditure, implying that all snacks had poor satiating efficiency.

Short-duration studies may not predict behaviour in the longer-term. A study of snacking behaviour by Johnstone et al. (360) showed no higher energy intake over a 7-day period between snackers and non-snackers. On the other hand, Blair has reported that obese subjects on a weight-reducing programme lost more weight when they stopped eating between meals (361). Few studies have examined the association between eating frequency and body weight in young people, although irregular snacking was found to be correlated with a raised risk of obesity among 3-year-old children in Japan (362).

Children who skip breakfast may be at increased risk of weight gain (363,364). There is clearer evidence for this among adults (365) although the exact mechanism remains unclear; eating breakfast may be associated with decreased fat intake and decreased snacking later in the day, or eating breakfast may be a marker of more organized family routines that are indicative of better health behaviour generally.

### Portion size

In North America, and to some extent in other countries with a well-established industrialized food supply, increases in standard portion sizes have occurred across a range of foods eaten in and outside the home (366). Academic research suggests that while very young children have innate control of appetite and are able to match intake to energy needs, this biological mechanism can more easily be overridden by environmental and social factors in older children.

A study of the effect of portion size on food intake at a given meal showed no change in energy intake among 3-year-old children but a positive association in 5-year-old children (367), however the impact on subsequent food consumption was not studied. A second study showed that in very young children, aged 12–18 months, body weight was positively related to energy intake and to portion size but not to the number of eating occasions (368). More research is needed, particularly in relation to the effect of portion size on subsequent energy intake in older children and the contextual factors that may influence this. Nevertheless, it is immediately apparent that a ‘Supersize’ snack or beverage can provide a disproportionately large contribution to a child’s energy needs in a single food item.

### 4.4.5. Physical activity and sedentary behaviour

Energy expenditure, like energy intake, is an important factor in the development of, or protection against obesity. Studies of energy expenditure may measure direct energy output using calorimetric methods, or may measure the incidence or prevalence of specific physical activities as indicators of raised energy expenditure, or may measure levels of inactivity, or sedentary behaviour, as indicators of low energy expenditure.

Direct evidence of decreasing energy expenditure among children in recent years is lacking. Some data suggest reduced walking and cycling behaviour between the 1980s and 1990s, among children in the UK and USA, along with increasing use of cars (e.g. to travel to school) (369,370). In the UK, children appear to become less active as they get older, and show decreases in activity levels during adolescence, starting earlier in girls than in boys (371). A systematic review identified a number of longitudinal studies estimating the effect of physical activity measured in childhood, usually by questionnaire, on subsequent fatness; the studies were generally small, and roughly divided between finding no effect, or a protective effect of activity (214).

Multivariate studies have found that television viewing and playing video games for longer periods of time, or not participating in sports outside of school, promotes obesity, whilst physical activity shows protective effects or no relationship (372–374). Several studies have specifically examined the links between television viewing and children’s risk of overweight. Although some find only weak relationships (347,375), several others have found that hours of television viewing were closely associated with increased levels of obesity in cross-sectional and prospective studies, among children in the USA (376), Australia (377), Mexico (378), Thailand (379), Greece (380) (see Fig. 30) and Native Canadian groups (381). One study, however, showed that after adjusting for differences in food intake and for general activity level, television ceased to be independently significantly related to child BMI, implying that the effect of television viewing on obesity may be mediated through one or both of these influences (377).

A prospective study by Gortmaker et al. (382) monitored a cohort of over 700 children aged 10–15 years during a 4-year period. The results showed a strong dose–response relationship between hours of television viewing and the prevalence of overweight at the end of the period, even after adjusting for previous overweight, baseline maternal overweight, socioeconomic status, household structure, ethnicity, and maternal and child aptitude test scores. Those children watching television the most (over 5 h day−1) were five times as likely to be overweight than those watching fewer than 2 h day−1. They were also the most likely to gain weight during the period, and least likely to cease to be overweight if they had been overweight at the start.
Various factors are likely to be important in determining the amount of time spent watching television. A study by Wiecha et al. (383) showed that the presence of a television in a child’s bedroom increases the time spent watching television by an average of 38 min day\(^{-1}\). This study also showed that other significant factors include:

- the presence of additional televisions outside the youth’s bedroom (7 min more viewing per additional set in the household), the child never or seldom has family dinners (33 min more viewing) and the child experiences no parental limits on television viewing time (29 min more viewing). The presence of parental limits on television viewing time was associated with 13 min more reading per day.

An Australian study showed that after adjusting for differences in food intake and for general activity level, television ceased to be independently significantly related to child BMI, implying that the effect of television viewing on obesity is mediated through one or both of these influences (377). Television viewing may be associated with changes in eating behaviour, for example by encouraging casual snacking, or may modulate eating habits through greater exposure to advertisements for foods high in added sugars and/or fat (384). Exposure to 30 s television food commercials can influence the subsequent food choices made by pre-school children (385). Television may also provide confusing messages about lifestyle and health from the content of advertisements or programmes (386,387).

The links between television viewing and weight gain may also be affected by other social factors, for example the use of television as a child-care substitute, especially in larger families. In one study the time spent watching television correlated positively with fatness in children from poorer socio-economic communities, but not in children from better-off communities (388).
5. THE MANAGEMENT AND TREATMENT OF THE OBESE CHILD

The successful treatment of obesity in children or adolescents has proved elusive. Reviews of the treatment of pediatric obesity have shown that the success of these treatments have been limited (8,389), although when weight reduction is achieved several co-factors also improve (390). Paediatric approaches are generally designed to bring weight gain under control and to manage and alleviate the associated co-morbidities.

The present report will describe briefly the management and treatment options available. It does not attempt to provide detailed principles of treatment, nor does it give specific guidance on risk reduction or the management of complications of obesity. There are several areas where further research is required to support the development of the management of obesity and its co-morbidities, such as the most appropriate management of insulin resistance, effective screening for, and management of, obstructive sleep apnoea, and the recognition and management of non-alcoholic fatty liver disease. Guidelines need to be developed, and the appropriate training made available, to ensure that primary health care staff are able to deal most effectively with the increasing burden that child obesity will place upon them.

For more details on management and treatment options the reader may wish to look at the literature suggested in Section 5.4.1 below. In addition, a report dealing more specifically with the clinical management of childhood obesity is being published by the Federation of International Societies for Pediatric Gastroenterology, Hepatology and Nutrition (391).

5.1. Principles of childhood obesity management

With the strongest links being found between adolescent obesity and subsequent adult obesity and its concurrent health problems, it might be argued that resources should be directed towards research and evaluation of the treatment of obesity in adolescents. However, younger children are used in the majority of studies of obesity management and, although the success of current obesity treatment programmes over the longer term (5–10 years) is unsatisfactory, the achievements appear to be better in the younger paediatric groups than in older ones or in adults (389).

Obesity management at a younger age may have a greater effect for several reasons, including:

- motivation may be easier to generate and maintain, for both the child and the other family members while the child is young;
- it can be easier to control and modify behaviour in younger individuals, there may be less resistance to treatment stigmatization and greater influence of the family on the child;
- there may be more frequent opportunities for medical observation during earlier childhood compared with later years;
- longitudinal growth and an increase of lean body mass occur during childhood so that children can ‘grow into their weight’.

Compared with younger children, adolescents are less likely to accept a highly controlled home or school regimen and may provide greater difficulty as subjects in randomized controlled trials, with a higher drop-out rate and a wider range of strategies for treatment avoidance and inaccurate self-reporting. Adolescents are in danger of falling in the ‘gap’ between paediatric services and adult services, with adolescent patients refusing to be treated ‘like children’ and failing to attend appointments (392). New strategies, for example involving communication through the Internet, better involvement of adolescents in their own management programmes and peer support strategies, need to be explored (393).

Motivation is essential: if the child or an influential parent is not motivated then the prospects for successful intervention are poor. Practitioners may need to make themselves familiar with techniques for gaining and increasing motivation among children and their families.

5.1.1. Dietary management

A reduction of dietary energy intake while maintaining or increasing physical activity will reduce relative body weight in children, but the long-term sustainability of dietary restrictions may be questioned. Reviewing the literature, Caroli & Burniat (394) conclude that several benefits can be achieved through dietary controls, but several negative consequences may also arise. On the positive side they highlight the following:

- Fat mass reduction and healthier body fat distribution. Dietary management appears to encourage the loss of abdominal fat mass in preference to subcutaneous fat, and significant metabolic improvements may result.
- Reduced plasma lipids and apolipoprotein levels, and enhanced insulin sensitivity and glucose tolerance. Dietary management can assist in decreasing baseline levels for plasma lipids and can improve glycaemic response to an oral glucose load.
- Reduced blood pressure. Blood pressure in obese children and adolescents appears highly sensitive to sodium intake, and that dietary management which reduces weight
and decreases sodium intake may have a twofold impact on reducing blood pressure levels.

On the negative side, Caroli & Burniat warn that dietary controls can cause problems, such as:

- Loss of lean body mass. Diets that restrict nitrogen intake may especially lead to excessive loss of muscle and other lean body tissue. Generally, if no more than 25% of weight loss is from lean body mass then it is deemed within the ‘safe’ weight loss range and can be viewed as a loss of excess lean tissue and a regression towards the lean body mass levels of people of normal body weight.

- Reduced linear growth. Dietary restrictions may lead to reduced linear growth and a lowering of predicted adult height.

- Binge eating and anorexia. These disorders may precede the introduction of a controlled dietary regimen, but care should be taken that such problems are not introduced or exacerbated.

- Increased serum uric acid. Several studies have shown raised uric acid levels during low-calorie dieting, but these resolve when the diet returns to a more normal, weight maintenance regimen.

- Raised risk of gallstones. This problem has been reported among severely obese adults on liquid very-low-calorie diets, but has not been reported for obese children eating normal foods in a managed diet.

In making recommendations for treatment, Caroli & Burniat suggest that weight maintenance regimens (with nutritional counselling) are preferred for all but the very obese until after puberty. For the very obese child, or the moderately obese child with additional complications, a balanced low-calorie diet using normally available foods is recommended. They recommend a more restricted very-low-calorie diet only when children suffer severe obesity with additional complications that justify such treatment. Overall, Caroli & Burniat suggest that dietary changes alone are ‘unlikely to have much effect without other long-term lifestyle changes’, which include increased physical activity, psychological support and an interdisciplinary management regimen. Dietary intervention in combination with exercise programmes has been reported to have better success rates than dietary modulation alone, with enhanced weight loss and better maintenance of lost weight (395). The optimal exercise programme, most appropriate for obese children and adolescents, remains to be established. Research to define the most effective combination of diet and exercise is a high priority for supporting treatment programmes.

5.1.2. Physical activity

A reduction of inactivity, an increase in ambulation and the development of an activity programme can increase the effectiveness of obesity therapy, and even when these do not reduce obesity they can independently reduce morbidity (10). Strategies for raising physical expenditure involve increases in physical activity and, as a separate strategy, decreases in sedentary behaviour such as television watching – with such sedentary behaviour being linked not only to low energy expenditure but also to increased snacking behaviour and exposure to food advertising.

Strategies for raising physical activity levels will encourage specific sessions of activity, but it should be recognized that the amount of energy that is expended during such physical exercise is generally modest compared with the energy deficit that can be achieved by dietary restriction. Nonetheless, exercise can produce useful reductions in body weight and fat mass. When evaluating changes in adiposity using weight or BMI, an adjustment for an increase in the muscle mass induced by isometric exercise may need to be made.

Interventions that target energy expenditure are more successful when reductions in sedentary behaviour are targeted rather than increases in the level of exercise (396,397). Simple measures such as the reduction of children’s television, videotape and video game use can significantly contribute to decreasing overweight in children.

Epstein et al. (398) have shown that participation of children in an exercise programme during treatment for obesity is often poor, but those that are most compliant are most likely to maintain long-term weight control. The type of exercise employed (i.e. ‘lifestyle’ exercise involving games, swimming, sports, dance, cycling, etc. as opposed to programmed aerobic exercise) also appears to be important for sustained weight loss; while both forms of exercise help promote weight loss in the initial phase, the child or adolescent is more likely to continue longer-term with the ‘lifestyle’ form of exercise (399). Families and young people need to be reminded that increased physical activity may best result from a change in incidental activity and not necessarily from organized exercise-focused activities. Importantly, children and adolescents should be encouraged to choose activities that they enjoy and which are therefore likely to be more sustainable.

Reviewing the evidence, Parizkova et al. (10) recommend that exercise programmes need to be individually tailored to the particular child. ‘Supportive help from family, peers, physical educators and teachers can combine to guarantee lasting positive effects of physical activity therapy until adulthood. High rates of drop-out can be reduced by programmes which are cheap, which involve group aerobic activities, and which are of low intensity but of more than 30 minutes duration.’ (p. 322–323). Competitive sports may not be appropriate for the obese child, and may increase psychological and social problems if they are required to participate, and equally lead to stigmatization if they are noticeably excluded. Swimming may be consid-
the absorption of food in the digestive tract and acting on pressing food intake, increasing thermogenesis, inhibiting the field is rapidly progressing. The mode of operation of Several drug therapies for adults have been developed and

5.1.4. Pharmacotherapy

Table 12 Pharmaceutical approaches to obesity control

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<thead>
<tr>
<th>Food intake suppressors</th>
<th>Thermogenic agents</th>
</tr>
</thead>
<tbody>
<tr>
<td>Noradrenergic: benzphetamine, phenmetrazine, diethylpropion, mazindol, phenylpropanolamine, phentermine</td>
<td>Adrenergic/serotoninergic: sibutramine</td>
</tr>
<tr>
<td>Serotoninergic: fluoxetine (and the withdrawn drugs fenfluramine, dexfenfluramine)</td>
<td>Adrenergic stimulants: ephedrine, caffeine</td>
</tr>
<tr>
<td>Beta-agonists</td>
<td>Lipase inhibitor: Orlistat®</td>
</tr>
<tr>
<td>Food absorption inhibitors</td>
<td>Non-digestible fat substitute: Olestra®</td>
</tr>
<tr>
<td>Hormonal agents</td>
<td>Leptin analogues</td>
</tr>
<tr>
<td>Neuropeptide Y antagonists</td>
<td>Cholecystokinin promoters</td>
</tr>
</tbody>
</table>

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5.1.3. Surgery

At present, surgical interventions are not recommended for use in children and adolescents with common forms of obesity. The safety and effectiveness of surgical treatments have not been sufficiently established in these patient groups and other approaches should be tried first (8). Reviewing the literature, Salvatoni (402) suggests that surgery should be considered only when all else has failed, when children have achieved adult height and when severe, potentially life threatening complications of obesity are present. He notes that:

‘Certainly, bariatric surgery cannot, and never will, solve the problems of management for the vast majority of obese and morbidly obese children and adolescents.’ (p. 358)

5.1.4. Pharmacotherapy

Several drug therapies for adults have been developed and the field is rapidly progressing. The mode of operation of these drugs can be categorized into four main fields: suppressing food intake, increasing thermogenesis, inhibiting the absorption of food in the digestive tract and acting on the hormonal system. These have been summarized by Molnár & Malecka-Tendera (403) (see Table 12). Further therapeutic developments are currently underway.

Caffeine–ephedrine mixtures in adolescents showed some success, while dexfenfluramine has been reported to be successful for treating obese children in two trials, but was reported as not superior to dietary modification in a third trial (403). A recent study on sibutramine in combination with behavioural therapy in adolescents showed some success (404). The study required a resource-intensive behaviour modification programme with 13 weekly session and six bi-weekly sessions in the first 6 months, in combination with the drug or a placebo. Adherence to the behaviour programme accounted for a significant proportion of the changes in body weights, greater than the effects of sibutramine versus placebo (8). Furthermore, the sibutramine treatment led to an increase of blood pressure at 3 months, and the dose had to be reduced or discontinued in nearly half of the treated adolescents in the first 6 months. There appeared to be no difference between sibutramine and placebo in the improvements in co-morbid conditions such as insulin resistance and dyslipidaemia. The authors of the research paper (404) comment ‘Until more extensive safety and efficacy data are available, medications for weight loss should be used only on an experimental basis in adolescents and children’ (p. 1805).

The beta-agonists and the hormonal drugs are currently under development. The results of the use of growth hormone in Prader-Willi syndrome appear to be significant and beneficial (405), although others have suggested further trials should be undertaken before recommending growth hormone treatment (403). There appear to be few reports of the use of appetite suppressants or of the use of Orlistat in children or adolescents. The use
of a non-digestible fat substitute, Olestra, may be considered, although anecdotal reports of gastrointestinal side-effects in some patients and a possible reduced availability of fat soluble vitamins in the diet should be noted before using Olestra extensively.

5.1.5. Psychological and familial support
The child’s family will influence the child’s food and activity habits and any effective treatment must take this into account. Parental involvement in treatment programmes is necessary for successful weight loss both in young children and, to a lesser extent, in adolescents. Several studies have shown that long-term maintenance of weight loss (i.e. from 2 to 10 years) can be achieved when the intervention is family-based (395,406,407) (for example, see Fig. 31). The long-term improvements against origin were small, with much of the apparent improvement due to the worsening obesity among the non-specific control groups.

These results imply that long-term weight control ‘success’ in childhood obesity is associated with parental involvement, and related factors including the amount of weight the parent loses (in families with overweight parents participating in the studies), the use of reinforcement techniques such as parental praise and a change in eating habits such as eating meals at home or a moderate reduction in fat intake (406,407). Altered food patterns within the whole family, as well as support of the child and parental reinforcement of a healthy lifestyle, are important factors in successful outcomes. The child should not be isolated within the family and should be participating in meals and eating similar foods with other family members.

There is increasing evidence that treatment of pre-adolescent obesity with the parents as the exclusive agents of lifestyle change is superior to a child-centred approach. An Israeli study randomized obese children aged 6–11 years and their parents to either an experimental intervention where only the parents attended group sessions (with an emphasis on general parenting skills), or a control intervention where only the children attended group sessions (11,408). There was a greater reduction in overweight in the experimental, parent-only group, and in contrast children in the control, child-only group had higher rates of reported anxiety and of withdrawal from the programme. Thus, when dealing with the obese pre-adolescent child, sessions involving the parent or parents alone, without the child being present, are likely to be the most effective.

There are barriers to parental involvement in the treatment of the child. In some families, for cultural or psychological reasons, parents may not perceive the child to be obese (409). In other families parents may acknowledge that the child is obese but deny that this is of any consequence (410). The implementation of treatment programmes may have to start by raising the parents’ awareness of the need to intervene, especially when family behaviour changes are required.

A different approach is needed for the adolescent patient. Features of successful interventions in adolescent obesity include the provision of separate sessions for the adolescent patient and the parent, and having a structured, although flexible, programme that encourages sustainable modifications in lifestyle, relationships and attitudes (411,412). There is also a report of at least short-term success in the

![Figure 31](image-url) Percentage of overweight children over a 10-year follow-up. Source: Epstein (395).
management of adolescent obesity with a phone- and mail-based behavioural intervention initiated in a primary care setting (413).

Behavioural and psychological forms of therapy that help to enhance physical activity and healthy eating habits are considered valuable for the long-term success of treatment in obese children and adolescents (414). Such approaches can help to stabilize and reinforce health-promoting behaviour, and to strengthen self-confidence and independence in the child. A large variety of approaches exist with respect to behavioural treatment, and many open questions exist such as the choices of methods, types, times and intensity of intervention and degree of individualization of treatment, which need to be studied further.

In a review of psychological approaches to treatment, Flodmark & Lissau (414) suggest that age is a major factor in influencing the choice of strategy for influencing behaviour. With pre-school children, group teaching is more important than individual treatment, and the whole family should be involved. By the time children pass puberty they are creating their own groups and social networks, and individual treatment may be more appropriate. Forms of treatment can range from cognitive behavioural therapy, family therapy, specialized schools and hospital treatment. Little is known about the value of psychodynamic therapies as these are not widely used. Behavioural and cognitive therapies assume that obesogenic behaviour is learned and can be unlearned through behavioural modification techniques. Family therapy improves an individual's health through analysing family interactions and improving a family's ability to use its own resources. In the European context, Flodmark & Lissau recommend approaches based on the schema in Table 13.

The ideal approach will vary from region to region depending upon existing health care services and expertise,

### Table 13 Behavioural and psychotherapeutic approaches to child obesity

<table>
<thead>
<tr>
<th>Childhood obesity (4–10 years)</th>
<th>Recommended treatment: Education in pre-school settings and in schools, with the provision of dietary advice, healthful foods and physical activity in groups. Interventions include cognitive behavioural therapy and family therapy.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Older child and adolescent obesity (10–18 years)</td>
<td>Recommended treatment: Obesity to be treated at the school health centre. Severe obesity to be treated with family therapy and social support or cognitive behavioural therapy. Interventions also include day-care programmes and specialized boarding schools.</td>
</tr>
<tr>
<td>Adolescent eating disorders</td>
<td>Recommended treatment: Child guidance clinic, family therapy, cognitive behavioural therapy and individual psychotherapy. Additional interventions include day-care programmes and hospital treatment using environmental therapy where individuals learn how to interact with their social environment.</td>
</tr>
<tr>
<td>Syndromes with impaired cognitive functioning</td>
<td>Family therapy along with social and educational specialized home support. Additional interventions include day-care programmes and hospital treatment using environmental therapy.</td>
</tr>
</tbody>
</table>

From Flodmark & Lissau (414).

**Residential schools or ‘fat camps’**

An alternative to a hospital inpatient programme is a residential weight loss school or camp programme (425–427). As with inpatient programmes, the regimens can vary. In different reports, the duration of the programmes ranges from 10 days to 8 weeks, during which activity programmes can include ‘exercise’ (425), ‘3 hours of physical activity’ (426), ‘5 hours of skill based fun type physical activity’ (427) and 5 hours ‘daily lifestyle exercise’ (428).

Dietary components may include a high protein, hypocaloric diet of 1200–1400 kcal day$^{-1}$ (426), 1300–1500 kcal day$^{-1}$ (425), a low-fat low-calorie diet based on estimated basal metabolic rate (427) or 1500 kcal day$^{-1}$ (428). All programmes reported involved forms of behaviour modification: ‘nutrition education’ (426), ‘behaviour and education change programme’ (427), and ‘Cognitive Behavioural Treatment’ (428). Despite the acknowledged importance of family support, only Braet & Van Winckel (428) and Gately et al. (427) allude to any form of family support during or following the intervention.

The outcomes of these camps appear impressive. Using different measures, they report a fall of ‘13.7 kg’ (426), ‘12.1 kg’ (425), ‘4.4 kg m$^{-2}$’ (427), or ‘18% overweight’ (428). Where subsequent follow up was undertaken, success compared with baseline figures was reported: down by 11.1 kg at 6 months (426), down by 2.6 kg m$^{-2}$ at 1 year (427), or down by 15% overweight at 4.6 years (428). The lack of common methods and measurements makes a detailed comparison between these different programmes difficult. Evaluations are also hampered by a lack of control group or standardized reference data against which the target group can be compared.
and upon cultural attitudes towards obesity treatment. In addition, it should be recognized that in developed economies as many as 50% of marriages end in divorce, creating difficulties in the health care provision for obese children in affected families.

5.1.6. Residential treatment
Most treatment or management programmes are directed through out-patient or family health services and occur in the home or school environment, delivered by a team of specialists through an interdisciplinary programme (13). Occasionally, as noted above, interventions may be considered more effective if delivered in a more controlled environment, in the hospital or specialized school or residential setting, particularly when outpatient management has proved ineffective and the burden on the child or adolescent becomes overwhelming (415).

Inpatient or residential treatment does not differ in principle from other treatment profiles, and it requires a programmed combination of therapeutic approaches to tackle the range of health consequences of obesity (416,417). Hospital and research institution inpatient treatment programmes have had varied success (24,418–424). The outcomes of these programmes varied from a fall of 4.6 kg to 13.5 kg and a fall of between 4% and 29% in overweight. Of eight inpatient programme reports, however, only two undertook a follow up; one reported that 48% of subjects sustained weight loss over a year (422) while the other showed a weight loss amounting to 20–25% sustained over a 5-month programme (423). The variation in methods used and outcomes achieved limits the general applicability of these important intervention programmes.

Residential programmes are best reserved for older children who can accept staying away from the family home and are able to form social networks with peers undergoing the programme. The high costs of such programmes should be contrasted with the costs of failing to deal with severe obesity in adolescents, whose health problems will become manifest early in adulthood and require many years of attention. However, there has been little long-term evaluation of the benefits to the patient of these intensive programmes.

The assessment of residential programmes as treatment models is currently restricted by variations in treatment methods, lack of reports on the specifics of the treatment programmes and resource requirements. Despite this, residential treatment programmes appear to serve useful purposes:

- by providing an intensive resource for the efficient treatment of the morbidly obese with associated co-morbidities;
- by providing a controlled setting for the development of a range of treatment models and strategies to tackle obesity;
- by demonstrating the cultural, social and physical barriers to effective treatment in the home and school environments, which may be overcome in residential settings.

5.2. Systematic reviews of obesity management
A review by Glenny et al. (429) reports thirteen randomized controlled trials covering a range of treatment approaches for childhood obesity. These include treatment of parents and children together, parental involvement in treatment, addition of controlled exercise to diet, reinforcement of behavioural diet and exercise strategies, the use of protein-sparing modified fasts and the promotion of lifestyle changes. Glenny et al. report that the evidence supporting the use of most treatment methods is equivocal, although the results of studies examining lifestyle changes appear promising. A comprehensive review by Epstein et al. (430) came to similar conclusions.

A Cochrane systematic review on the treatment of obesity in childhood (389) evaluated 18 randomized controlled studies. These shared similar goals and objectives, but had different study designs, sample size and outcome measures. Most studies were generated in the USA among children aged between 7 and 12 years, and were under-powered (15 studies had less than 23 children in one or more of the groups being compared). Seven of the studies were carried out by the same research team, which has implications for generalizing the results of this review to other contexts. Many of the 18 trials were run from a specialist obesity clinic within a hospital setting. The results were not strongly convincing. There may be some additional benefit to behaviour therapy where parents, rather than the child, are given the primary responsibility for behaviour change. Although there were many trials which focused on changing levels of physical activity and/or sedentary behaviour, these trials were too small to reach any conclusions with confidence.

Several points arose from this review that need to be considered when evaluating the effectiveness of interventions in child obesity. The first was the disagreement regarding the definition of fatness in children. Developing consistent approaches to the measurement of childhood obesity is a priority issue in this field. Second, the measurement of predicted behaviours, such as dietary intake and physical activity, remains relatively weak and undermined the ability to validate the trials. In undertaking new studies, the review noted, particular
attention should be given to the following aspects of design:

- sufficient power – adequate numbers;
- adequate follow-up of participants;
- reliability of outcome measurements (reporting of height, weight, BMI);
- process indicators – indication of whether the study was adhered to and conducted as it was intended;
- cost effectiveness;
- appropriate and adequate statistical analysis;
- sustainability of the effect;
- generalizability of the results.

In a recent overview on the treatment of child and adolescent obesity, Yanovski & Yanovski (8) consider the progress that is being made and conclude:

‘At the present time, however, it remains exceedingly difficult for overweight children and adolescents to lose weight, and even more difficult for them to sustain that weight loss long term. The ultimate goal must be prevention of the development of overweight in children and adolescents.’ (p. 1852).

5.3. Screening and identifying needs

Screening is the practice of investigating seemingly healthy children with the object of detecting unrecognized disease or children with an exceptionally high risk of developing disease, and of intervening in ways that will prevent the occurrence of disease or improve the prognosis when it develops (431,432). A screening test is not intended to be diagnostic, hence it must be followed by comprehensive assessment to confirm or dismiss the impressions raised during screening (433,434).

Experience with childhood obesity screening has developed in the last two decades (435–437). There are few guidelines on the details of screening procedures or the recommended frequency, although some practitioners recommend childhood obesity screening to be performed annually (435).

Experience with childhood obesity screening has developed in the last two decades (435–437). There are few guidelines on the details of screening procedures or the recommended frequency, although some practitioners recommend childhood obesity screening to be performed annually (435).

It is controversial whether childhood obesity screening should be introduced (436), because the early detection of obesity may create expectations without improving the prognosis (438). Childhood obesity screenings might only be of value under the following conditions:

- the screened obese individuals are ready to have further assessments and make changes to achieve a healthy weight (436);
- further assessment or other necessary treatment facilities are available in the community (433);
- effective intervention programmes and follow-up activities for the identified children are accessible and available (439).

It is important to consider two further points before embarking on a childhood obesity screening programme (438). First, apparently healthy children should be willing to present themselves for examination. Second, screening large numbers of children is expensive and can divert both staff and financial resources from other health services activities. It is essential, therefore, to weigh up the potential benefits both for the individuals screened and for the health of the community, against the cost of the resources.

5.3.1. Settings for childhood obesity screening

Screening may be applied in different settings and different terms have been coined.

- Mass screening can be done in large populations who will be tested in community-wide programmes, such as immunization programmes and nutrition surveys (439). These also provide useful data for monitoring trends in the population and the evaluation of intervention at population level (440).
- Group screening can be done on groups of children who are selected as being especially at risk (for example, families with history of obesity or obesity-related diseases, certain ethnic groups) (432).
- In healthcare services, opportunistic screening occurs if overweight or obese children are screened when they present for other conditions such as infections.
- Public health screening involves the simultaneous identification of obese children during public health service activities and programmes such as immunization, mother and child welfare clinics (16).

Screening overweight and obese children can be conducted in schools (including pre-school and day-care centres) (433) or healthcare organizations (such as polyclinics or health centres) (437). Depending on the definition of obesity, the population size, the socio-economic status of the country, and the purpose of screening (e.g. for establishing prevalence, for implementing intervention, etc.), each setting has its advantages and disadvantages.

5.3.2. School-based screening

In countries where education is compulsory or where the majority of children are in the education system, schools (including pre-schools) (433) are the best place for childhood obesity screening (441). The screening could be conducted periodically by school personnel.
during other school functions. For example, in Singapore, weight and height of the students are taken annually by teachers during physical education lessons and the collected information is conveyed to relevant health organizations. The measurements could also be done by a team of doctors and nurses as part of the comprehensive health screening routine in a medical room provided by schools at some or all school levels. In Hong Kong the student health service provides health screening for all school children annually, although not all children utilize the service, with the highest attendance in the earliest years (434,442, A.E. Nelson, unpubl. data based on 443).

The advantages of school-based screening include feasibility, cost-effectiveness and good coverage. The disadvantages of such screening are the extra workload for the school personnel, the conduct of screening by teachers without specialized training, and a lack of standardized equipment among schools. All these factors might contribute to inaccurate measurements that are not uniform across schools. Additionally, the identified children have to be referred to organizations outside the school for management.

5.3.3. Healthcare organization-based screening
Healthcare organization-based screening – involving health centres, polyclinics or private clinics – offers an alternative setting to school-based screening, and may catch children who were not included in school-based screening. Healthcare facilities may provide intervention programmes, and management of the identified overweight and obese children can be carried out with little delay. Moreover, screening is conducted by trained health professionals, hence measurement errors are minimized.

Healthcare outreach, however, may be no better than school-based outreach, and clinics, like schools, may suffer a lack of standardized equipment. A mobile examination centre such as that used for the National Health and Nutrition Examination Surveys might overcome some of these problems (439).

5.3.4. Staff training needs for childhood obesity screening
Staff involved in childhood obesity screening include health personnel (e.g. doctors, nurses, dietitians), teachers and trained lay persons including parents (433,438,441). To protect the identified obese child’s feelings, staff need to convey the message about the child’s condition sensitively while referring him or her for further assessment (16).

Weight and height (and other derived indices) are the two major determinants of a child’s nutritional status. The staff involved must be trained in taking measurements as accurately and precisely as possible, especially to detect changes over time (431). Factors such as how the children should stand, what they should wear, where the equipment should be placed, what time the measurement should be done, etc. (444,445) must be taken into consideration. A short refresher course or the provision of guidance notes before the screening is conducted could minimize carelessness.

For screenings involving more sophisticated body composition tools, such as skin-fold measurements, bioimpedance or dual energy X-ray absorptiometry, staff must be trained in calibrating the machines and using the equipment.

5.4. Guidelines for treating and managing overweight children
Clinical practice guidelines are valuable tools for practitioners making decisions about appropriate health care for specific clinical circumstances (446). Guidelines may also be valuable for families, nurseries and schools as well as the child involved. In addition, guidelines may be extended to include decision-makers in the wider community, including food industry and catering sectors, the media, and local and national government agencies with responsibilities that affect the community’s health and lifestyle choices.

Most guidelines to date have focused on providing guidance to clinical practitioners. Such guidelines should be rigorously evidence-based, using systematic reviews of the literature when possible, but there are many circumstances in clinical practice where there is insufficient high-quality evidence to meet these requirements, and the guidance is based on a combination of available research evidence, expert opinion and consensus.

Where guidelines are developed for non-clinical use, for example in nurseries and pre-school centres, then additional information may be needed, along with training and support services, to ensure that the guidelines are understood and can be implemented. This may mean finding resources, for example to adapt play equipment or to retrain catering staff.

At the present time several countries have developed national guidance statements on childhood obesity treatment based on expert consensus views. The US Expert Committee recommendations on obesity evaluation and treatment are a widely used example (447). The French Institute of Health and Medical Research (INSERM) document is another (448) and so is the recent publication from the Australian National Health and Medical Research Council (see http: http://www.obesityguidelines.gov.au/). Further examples are listed in the box ‘Guidelines and resources’ below.
5.4.1. Guidelines and resources

**Resources for health professionals**


France: Expert advisory group ‘Childhood Obesity. Screening and prevention. Synthesis and recommendations’ http://www.insERM.fr (English synopsis available on http://www.insERM.fr/servcom/servcom.nsf/7f476b23a842f98c12569b400384eee/6477a23a05c9289180256b7d004cda49/$FILE/Obe-


UK: NHS Centre for reviews and dissemination ‘The Prevention and Treatment of Childhood Obesity’ *Effective Health Care* 2002; 7(6).


Germany: Guidelines for assessment, treatment and prevention, of the German Society for Paediatrics and Adolescent Medicine, Adipositas (see http://www.a-g-a.de).


Australia: The National Health and Medical Research Council of Australia has developed clinical practice guidelines on the management of overweight and obese adults and children (see http://www.obesityguidelines.gov.au/).

**Resources for children and families**


sity.html

**Resources for schools**

USA: CDC guidelines for schools:

- physical activity: http://www.cdc.gov/nccdphp/dash/healthtopics/physical_activity/guidelines;


5.4.2. Obesity guidelines: concordance with other health advice

Health professionals may need assistance to translate evidence-based guidelines into everyday practice. It is important that every effort is made to ensure that practical advice is safe. For example, clinical interventions may serve to raise anxiety and lower self-esteem, especially if there is little tangible weight loss, and approaches are needed which can avoid these hazards. For this reason, families, schools and primary health care services should be encouraged to focus on promoting healthy lifestyles in all children, as well as helping the obese child.

The principles of healthy eating and active lifestyles are fully compatible with the prevention of diseases such as coronary heart disease, type 2 diabetes and many cancers. There may be an increased risk, however, of traffic accidents if children play in the streets or take to cycling and walking. Safe routes to school should be developed.

There may also be some concern about the possibility of dietary restriction leading to eating disorders. As noted earlier, obesity is occasionally associated with binge eating disorder, but there is no published evidence for a causal link between obesity and anorexia nervosa to date. Both are increasing in prevalence in modern society. The development of healthy (but not unnecessarily restrictive) eating habits should be compatible with prevention of eating disorders. However the treatment of eating disorders and obesity may be very differ-
ent. For example, close self monitoring and restricting of food intake may be encouraged in some obesity regimens but discouraged in anorexia. If possible, strategies should be used that are concordant for weight problems at both ends of the spectrum i.e. ‘an integrated approach to prevention and treatment of weight related problems’ (449). Such strategies might focus on self-esteem along with enhanced physical activity and the consumption of healthful foods.

5.4.3. Dissemination and implementation

Guidelines are only useful if they are widely known about, recognized as appropriate and put into practice. This will require widespread discussion during and after their preparation, distribution with additional information and appropriate training or support during implementation (446,447). Evaluation, regular review and updating of guidelines are essential.

Guidelines and advice must be clearly presented, relevant and easily accessible. There is a need for more information about the implementation of intervention strategies in everyday life in families, schools and the health service. Strategies should cover psycho-social aspects, physical activity and inactivity, and diet and eating habits (447). Where adequate evidence is lacking, every effort must be made to ensure interventions will be safe.

Advice is needed to help services cope with rapidly increasing demands and the needs of obese children. There is inconclusive evidence about the importance of the attitude and commitment of professionals to the success of treatment (413,447,450,451). However, it seems appropriate for guidelines and advice to encourage positive, supportive help. The implementation of guidelines and the results of intervention need to be evaluated in order to inform future practice.
6. PREVENTION – THE ONLY SOLUTION

This report has identified the dramatically rising trends in the prevalence of child and adolescent obesity and has indicated some of the serious health problems that are emerging as a result. The great majority of children are at risk, especially if the environmental factors that encourage obesity are present – as they are in most industrialized countries and urbanized populations. This report has also noted the relative lack of success of treating obesity once it has become established. Virtually all reviews have indicated that they believe that the prevention of obesity is not only possible but is the most realistic and cost effective approach for dealing with childhood obesity (18,452) as it is for adult obesity (16).

Despite the traditional mission of paediatric practice to be a preventative service, there has been little exploration of the effectiveness of using the clinical setting for preventing child obesity. Additional research in this area is needed. At primary care level, practitioners can have a role providing information on healthy eating and physical activity to all members of the family as well as supporting effective parenting skills, summarized by Dietz & Gortmaker as ‘anticipatory guidance’ (453). At specialized paediatric clinics the early prevention of obesity among those at greater risk (e.g. members of population groups described in section 4.4.2 above) may be considered when children are referred for other reasons, especially if the children are already showing overweight or mild obesity. Proactive screening of child populations, e.g. in schools, has been discussed above (5.3).

The following section gives some examples of preventive strategies that have been tried, and summarizes the literature reviews available. This leads to the conclusion that small-scale preventive activities have only modest effects, and that a larger-scale approach will need to be taken if the environmental stimuli that encourage weight gain are to be properly challenged (454). Strategies for encouraging health and preventing obesity at national level are discussed, and the role of health ministries in developing public health, food and physical activity policies underlined.

6.1. What prevention works?

In this section, six examples of preventive approaches directed at children are discussed. This is followed by a general review of intervention trials, which concludes that some approaches are worth further investigation but that there has been no outstanding breakthrough that will ensure that the rise in child obesity can be halted or reversed.

6.1.1. The school approach

In principle, schools provide an excellent setting for preventing obesity (455–457), and are also the target of the World Health Organization’s ‘Health Promoting Schools’ programme (458). They offer regular contact with children during term-time and provide opportunities for nutrition education and promotion of physical activity both within the formal curriculum, and informally via the provision of appropriate facilities within the school environment such as healthy school meals, break-time snack provision and playground equipment. Thus schools not only influence the knowledge and attitudes of children but also provide opportunities for experiential learning and the development of a sense of self-efficacy. Furthermore the school can also provide links with the family and the wider community.

‘APPLES’. A UK trial involving 10 primary schools in the north of England (the ‘APPLES’ study) tackled the school environment at several levels simultaneously (459,460). The intervention took the form of teacher training, modification of school meals, and the development of school action plans targeting the curriculum, physical education, tuck shops and playground activities. The results were mixed. The intervention group showed an increase in vegetable consumption, but those children who were more overweight in the intervention group showed an increase in the consumption of sugar-containing foods, a fall in fruit consumption and an increase in sedentary behaviour. Measures of self worth showed that overweight children in the intervention group gained from the trials. Intervention children also showed a higher score for knowledge, attitudes and self-reported behaviour for healthy eating and physical activity (460).

‘Trim and Fit’ in Singapore. In Singapore a national healthy lifestyle programme called ‘Trim and Fit’ has had some success at reducing obesity prevalence in school age students (461). Trim and Fit involves a variety of changes in the school environment, backed up by resources directed to the schools from the Ministry of Education.

Trim and Fit sessions for overweight children consist of:

‘an exercise programme and counselling on proper nutrition. Teachers monitor the children’s weight regularly. Physical activity takes the forms of games and exercises during recess or outside curriculum time. Parents of overweight children are invited to support the programme through seminars and meetings. Besides physical activities, overweight children are given nutrition counselling, including choice of low-calorie foods in schools canteens. School canteen vendors are also advised on healthier methods of cooking. More fruit and vegetables are encouraged.’ (462).

Ministry of Education support has included intensive teacher training for all teachers, including head teachers,
specialized instruction in home economics and physical education, approval and monitoring of school canteens, provision of catering equipment, school fitness equipment and printed educational materials.

The results show modest success, with decrease in the prevalence of obesity (from around 16% to 14% over an 8-year period) (Fig. 32). Control group figures are not available, and it should be noted that the degree of obesity prevention achieved in this programme has not been fully evaluated or peer reviewed. The reported reductions might be set against an expected increase: adult obesity rates showed a non-significant increase from 5.1% to 6.0% over the period 1992–1998 (463).

Crete: tackling the record rise in obesity. Based on the Greek island of Crete, Mamalakis and colleagues have monitored a group of children over a period of 6 years, with half of the children experiencing an intervention programme and the remainder acting as controls (464). Crete has one of the highest rates of childhood obesity reported for any population, and any successful intervention programme would prove particularly significant for such populations.

The interventions involved 24 schools, with a further 12 schools acting as controls. Interventions included health education, improvements in nutrition and encouragement to take physical exercise – delivered as a taught subject and as physical activity periods during school hours. The programme continued for 6 years, with a cohort of children monitored during the first year (aged around 6 years old) and again at age 9 and 12 years.

The prevalence of overweight (using IOTF criteria) among children at each of the three evaluation phases – at the outset, mid-way and at the end of the trials – are shown in Fig. 33. Results in terms of several cardiovascular risk factors showed significant improvements in the intervention children compared with controls (76). Results in terms of BMI also showed improvements in the intervention group compared with the control group, although BMI levels in both groups rose during the period with increases in the proportion of children overweight. The intervention appeared to show its greatest effects in the first 3 years, possibly because older children are less amenable to school-based health messages. However, the rise in the numbers of overweight children, even among the intervention group, is a cause for continued concern.

Paying less for health. A series of trials by researchers at the University of Minnesota (465–467) has shown that healthful behaviour, such as the selection of healthier food items at a school canteen or from a vending machine, can be influenced by changes in the price of the products being purchased. In trials of vending machine price manipulation of more and less healthful foods undertaken in secondary schools (children aged 12–16 years) price differentials of 10%, 25% and 50% in favour of healthful foods led to 9%, 39% and 93% increases in purchases, respectively (468), indicating that switches from less healthful to more healthful choices are strongly influenced by pricing factors. When the price advantages are removed the purchase preferences for healthier foods disappear (Fig. 34).

Watching less TV. Several studies have indicated that television watching is linked to overweight (see earlier). Trials by Robinson (470,471) have shown that restrictions on TV watching can lead to beneficial effects measured by several different approaches. In one trial, primary school children aged around 8–9 years old received 18 lessons over
6 months encouraging them to reduce television watching and video game usage, and were matched with similarly aged children in a school of similar sociodemographic and academic status (470). Relative to controls, the intervention group showed an improvement in many of the evaluative measures taken, including measures related to adiposity and to the extent of television viewing and of eating meals in front of the television (Table 14). Measures of high-fat food intake showed no significant difference, and nor did measures of vigorous physical activity or cardiorespiratory fitness.

**Agita São Paulo programme.** In Brazil, the Agita São Paulo programme promotes physical activity and the knowledge of the benefits of physical activity in the State of São Paulo, with a population of over 36 million inhabitants (472).

Agita is organized by the Center of Studies of the Physical Fitness Research Center (CELAFISC) with the State Secretary for Health and more than 250 governmental, non-governmental and private institutions. The programme was launched in February 1997, and because of its impact has now been adopted throughout Brazil (Agita Brasil). It has served as the basis for similar programmes in other countries in South America and provided inspiration for the celebration of World Health Day 2002 (1987 events in 148 countries).

Children and adolescents are among the three target groups (the two others are elderly people and the workforce). The main message of the programme states that every person should take physical activity on most days of the week, at moderate intensity (and even light for special groups), in continuous or cumulative sessions. Young people should take at least 20 min of sustained vigorous physical activity, on 3 days of the week.

The Agita strategy has included an emphasis on the benefits of physical activity, but these are not in themselves enough to motivate educational authorities, schools or teachers. For that reason, the Agita São Paulo Programme has also highlighted the psychosocial and educational benefits, including an improvement in self-image, self-esteem and well-being, and reduced stress and depression. The potential educational benefits include improvement in academic performance, a decline in behavioural disturbance, a decrease in high-risk behaviours such as substance abuse, and an increase in the student’s sense of responsibility.

The programme for young people is largely based around schools, with attention paid to increasing time spent in physical activity in physical education classes, other classes, in breaks and in the community. With restricted access to public spaces for physical activity, particularly in large cities, schools in some of the poorest and most violent regions of São Paulo city have opened their facilities at the weekends. The initiative is largely based upon volunteer support, and has become a success involving 400 schools.

Evaluation measures show a significant increase in time involved in vigorous physical activity in girls and a trend towards increased vigorous physical activity in boys. There was a similar trend towards an increase in moderate physical activity among boys, although not in girls. These results confirm a positive effect of a media–community–school intervention approach in promoting physical activity among adolescents.

Agita Galera or Active Community Day, is scheduled each year to give more visibility to the programme. It includes approximately 6000 public elementary and high schools and several hundred private and special schools. Preparation includes teacher awareness and training and a cable TV conference. The event provides a chance to spread the message on TV, radio and newspapers. TV coverage of Agita Galera reached an estimated 20 million viewers.

### 6.1.2. Reviews of intervention trials

This section reviews the school and community trials in which randomized control procedures were conducted. The Table in Appendix 2 shows the results of a literature search

<table>
<thead>
<tr>
<th>Change vs. control group</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>TV watching</td>
<td>reduced by 4.49 h week⁻¹</td>
</tr>
<tr>
<td>Use of video games</td>
<td>reduced by 2.54 h week⁻¹</td>
</tr>
<tr>
<td>Meals in front of TV</td>
<td>reduced by 1.07 meals week⁻¹</td>
</tr>
<tr>
<td>Triceps skin-folds</td>
<td>reduced by 1.47 mm</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>reduced by 2.3 cm</td>
</tr>
<tr>
<td>Waist–hip ratio</td>
<td>reduced by 0.02</td>
</tr>
<tr>
<td>Body Mass Index</td>
<td>reduced by 0.45 kg m⁻²</td>
</tr>
</tbody>
</table>

Source: Robinson (470, 471).
conducted by Schmitz & Jeffery, and shows studies that focused on improving eating and energy expenditure behaviours of children in the general population, not overweight or obese children or children at high risk for obesity, and have included measurement of some physiological, obesity-related outcome and have a control group. All papers up to the year 2000 were examined.

All of the studies in Appendix 2 included a strong school-based intervention component. Schools are an attractive setting for obesity prevention efforts in children because of the relatively large amount of time children spend in school, the ability to use existing organizational, communication and social structures and the ability to reach a large portion of children in the general population. In addition, one to two meals per day may be eaten at school for 5 days of the week. Other social settings, however, such as the family and community may have more significant influences on youth behaviours. Recent US Centers for Disease Control guidelines for promoting physical activity and healthy eating note the importance of including community and family efforts in addition to school-based programmes (473,474). Interventions should also take account of national changes in dietary trends that may be occurring during the period of intervention: a 5-year school-based nutrition education programme showed significantly raised awareness of nutritional knowledge among intervention children compared with controls, but no difference in energy or macronutrient intake, while both intervention and control groups showed reduced consumption of French fries and an increased use of olive oil over the period (M.A. Charles, pers. comm., 475).

The studies included in Appendix 2 indicate that environmental changes, such as alterations in school physical education or monitoring television viewing time, are at least as important as classroom-based educational interventions. Of the studies included, those conducted in less diverse settings were more likely to show significant obesity-related treatment effects. This was particularly true for studies where the outcomes included multiple chronic disease risk factors or multiple behaviours, such as the Planet Health programme. Planet Health describes itself as ‘an interdisciplinary curriculum focused on improving the health and well-being of sixth through eighth grade students while building and reinforcing skills in language, arts, math, science, social studies, and physical education’, which ‘aims to increase activity, improve dietary quality, and decrease inactivity’. The Planet Health programme has reported significant success for obesity-related outcomes (476). It has been employed in diverse settings and among minority ethnic groups. Further details can be found on <http://www.hsp.h.harvard.edu/prc/planet.html>.

The absence of long-term follow-up in the studies included in Appendix 2 makes it difficult to evaluate the efficacy of these interventions for population-wide effects on obesity prevalence. Most of the studies were able to show improvements in eating and/or exercise habits of children and the large trials indicate the feasibility of implementing school-wide changes for the purpose of obesity prevention. The effects of these interventions when the children become adults, in terms of their health-related behaviour or their health status, remain to be assessed. Future studies will therefore need to evaluate the cost-effectiveness of school- and/or community-based obesity prevention interventions in youth, including long-term follow-up of obesity prevalence and incidence.

Cochrane review of prevention trials. Campbell and colleagues (14) have undertaken a systematic review of interventions for the prevention of obesity in children, and included non-English language papers in the literature search. Randomized control trials and non-randomized trials with concurrent control groups, in which participants were observed for a minimum of 3 months, were selected for review. The review found only 10 studies of sufficient quality, of which seven observed children for over a year and three for less than a year but more than 3 months. Eight were school- or nursery-based interventions, one was a community-based intervention targeting African-American families and one was a family-based intervention targeting non-obese children of obese parents.

The Cochrane review authors concluded that three out of four long-term studies that combined dietary education with physical activity interventions resulted in no difference in overweight. A fourth reported an improvement in favour of the intervention group. In two studies of dietary education alone, a multimedia strategy appeared to be effective, but other strategies did not. The one long-term study that focussed only on physical activity resulted in a slightly greater reduction of overweight among the intervention group, as did two short-term studies.

Campbell et al. concluded that current efforts for obesity prevention need to build a reliable evidence base to determine the most cost-effective and health promoting strategies for ensuring a healthy weight for all children. Particular attention needs to be paid to ensuring studies have sufficient numbers of participants, have adequate follow-up, use reliable outcome measures, and can provide sustainable and generalizable conclusions.

Hamilton reviews. Similar conclusions were reached in a ‘review of reviews’ on interventions to prevent chronic disease conducted by an inter-university team for the City of Hamilton, Ontario, Canada (477). On the effectiveness of school-based strategies for the prevention of obesity, the review concluded that the most effective interventions should be based on a whole school approach including cafeterias, PE classes, lunch and recess activities, classroom teaching, and include links to home and the community.
The longer the intervention the greater the change in outcome measures. Different age groups, ethnic groups and genders needed different approaches.

The inter-university team also reviewed school-based interventions designed specifically to encourage physical activity (478). The reviewers found moderate improvement in physical activity among children and among adolescent girls exposed to promotional campaigns, but with little measurable effect on blood pressure, BMI or heart rate. The most effective initiatives involved children through the whole school day, including lunch and recesses as well as class time and physical education lessons.

In one of the key conclusions, the report noted that adults who had participated in school-based physical activities as children were more likely to be active in adulthood than those that had not. A report by the US Centers for Disease Control made a similar point, noting that physical activity tends to decline during adolescence and that ‘comprehensive school health programmes have the potential to slow this age-related decline in physical activity and help students establish lifelong, healthy physical activity patterns’ (474).

**Keys to success.** These reviews show that tackling excess weight among children requires a broad-based approach involving all the school. This approach can lead to longer-lasting benefits that continue into adult health and well-being. However, the resources and the change in thinking that would be needed to implement such policies are not easily found and may lead to frustration.

In an invited commentary on school-based interventions, Lytle et al. (479) note that only a few interventions have had significant effect. The authors suggest several factors that may improve success rates, notably ensuring an adequate length of intervention and ensuring the involvement of all participants to prevent drop-out. They also note that heterogeneity, i.e. the involvement of participants from diverse cultural backgrounds, is rarely catered for in the experimental designs where ‘one size fits all’, and this may compromise the ability to show significant effects. The authors recommend programmes that are more flexible and responsive to the social and cultural environments in which they occur, perhaps inviting the active participation of community members during the design of the intervention. They also note Richter et al.’s evidence that school and community interventions are more likely to be successful if they occur in the context of wider health-promoting environments (480).

### 6.1.3. Alternatives to randomized control trials

The previous section indicated the pressing need to develop the appropriate measures and study designs to find effective preventive interventions for obesity. The studies need to capture changes in environments and behaviours as well as outcomes such as body mass index. They also need to be administered over long periods of time and across populations. This puts a burden on the resources and on the participants in the studies, and it means that the normal approach, using small-scale randomized control trials may not be appropriate. Some alternative approaches may be needed.

Recent experience in evaluation has suggested that the randomized control trial (RCT) design can be inappropriate for a number of reasons. The RCT approach assumes that there is full control of the intervention, its delivery and the context in which it is implemented. This is certainly not true for most health promotion programmes, which are complex in nature, delivered in complex contexts over long periods of time, and present difficulties in controlling all variables. In reality it is difficult to find matched controls, ensure standardization of programme implementation in all contexts and ensure standardization of contexts. Furthermore there is a constant threat of ‘contamination’ of the control group because health promotion relies on the effects of the intervention permeating the target community. It is therefore difficult to control for spill-over to the comparison community, thereby potentially reducing any effect of the intervention. Other problems include:

- the individuals recruited may not represent the population;
- randomization may not be feasible and may not ensure comparability;
- control conditions are difficult to implement and raise ethical problems;
- the intervention is too disruptive of reality;
- energy balance cannot be directly assessed;
- other effect sizes may be too small to detect.

Obesity prevention programmes, like other health promotion programmes, are complex, often targeted at groups and individuals simultaneously, aiming to influence individual behaviour and lifestyle but also address public health policy in order to change social and environmental factors that affect health. Consequently health promotion evaluation requires multiple approaches in order to assess effectiveness in all these areas.

The selection of method depends on the nature of the information required to answer the questions asked. It is therefore advised that both qualitative and quantitative methods are used, and methods for rapid appraisal and health impact assessment may also prove valuable (481,482).

**Monitoring obesity trends.** The epidemic of childhood obesity is already present in most high-income countries and emerging in some low-income countries. The first step to evaluating the causative (and preventative) factors is to...
monitor trends in obesity in a range of populations. Ironically, it may be the low-income countries that will be better equipped to monitor trends in childhood obesity because most of them already systematically measure the height and weight of infants and children to monitor under-nutrition trends. The reliance on occasional, large nutrition surveys to determine the trends in overweight and obesity means that available data are usually spasmodic, delayed and used more by universities than health departments.

There is an urgent need to establish more formal monitoring programmes. These need to have the following characteristics:

- Owned and used by governments to assess progress towards targets for childhood obesity, eating and physical activity behaviours and environments.
- Regularly implemented and assured of sustainable funding.
- Measure the key outcomes and determinants of interest: height, weight and waist; eating behaviours and attitudes; physical activity behaviours and attitudes; key nutrition-related environmental factors; key physical activity-related environmental factors.
- Potentially serve as the ‘comparison group’ for effectiveness studies (see later).
- Monitor the reach, sustainability and population impact of programmes (see later).
- Potentially use for benchmarking purposes for schools and local governments (see later).

**Assessing interventions.** The assessment of interventions is usually achieved using standard experimental methodologies (randomized controlled trials, cross-over studies, quasi-experimental studies etc). Unless the intervention being tested can deliver a ‘high dose’ of change in activity levels and/or energy intake, it is unlikely to show an impact on body size. For example, reducing television viewing by 30 min to 1 h day\(^{-1}\) as achieved by Robinson (470) and Gortmaker et al. (476) appears to achieve a high enough dose to influence body weight, but other school-based interventions, although well implemented, cannot achieve this dose (459,460).

The effectiveness of ‘real world’ interventions needs to be assessed in the knowledge that tight control of extraneous variables is very difficult and the use of randomized control groups is usually not possible. An alternative framework is required:

- an initial formative evaluation to establish clear aims and objectives;
- a needs assessment, literature review etc;
- a process evaluation to measure the inputs into an intervention and to describe the processes during the implementation;
- an impact evaluation to measure whether the specific objectives have been achieved (e.g. changes in physical activity behaviours);
- an outcome evaluation to measure the longer term effects (e.g. changes in body mass index).

To be rigorous, however, the impact and especially the outcome measures need to be controlled for confounding factors. For example, no change in BMI may represent a successful or a non-successful outcome depending on the background changes in BMI in the rest of the population.

Randomization by individual is the design that is most likely to spread the confounding factors evenly across intervention and control group, but this design is usually not feasible for long-term prevention studies where a variety of interventions, including environmental ones, are used. In fact, because the ‘dose’ of intervention needed to prevent unhealthy weight gain is high, it requires a multi-strategy, multi-setting approach and this virtually excludes the individual randomized design. A quasi-experimental design (for example, five intervention schools and five control schools) gives some ability to control for confounding variables, but usually the number of units (e.g. schools) is small and systematic differences may confound the results (e.g. the control schools have a different socio-economic background than the intervention schools).

There is a potential for using population monitoring data for not only measuring trends (above) but also for comparative purposes for effectiveness studies. For example, a school-based, region-wide monitoring programme may routinely measure BMI (outcomes), school food sales (behaviours), and school food policies (environments). An intervention programme in a town within that region may be able to compare changes in these variables against changes across the region. There would still be the potential for residual confounding factors but at least such a design would allow reasonable confidence on the overall impact of a population-based intervention programme on the prevention of obesity.

**Assessing sustainability, reach and population impact.** Many intervention studies aimed at influencing long-term behaviours at a population level have a start and finish date. The longer-term sustainability and wider reach, and population impact across the population are rarely measured. In the end, this is the information that the government funding agencies really need to know. Indeed, such characteristics as ‘reach’ are usually specifically designed out of studies because it is considered ‘contamination of the control group’. For example, in the North Karelia
Obesity in children and young people

of children – and which influence eating and physical activity – certainly not under the control of children – and which influence eating and physical activity behaviour in the aggregate (484). Thus, although actions to improve individual lifestyles are needed and are perhaps the only recourse for limiting obesity development in the short-term, longer term remedies undertaken from a broader public health and policy perspective will be needed to have a significant impact on the problem. Not only are individually-oriented approaches likely to have limited success if undertaken without supporting social and environmental changes, but an over-reliance on individual approaches with insufficient public health actions may allow the situation to worsen and, particularly, to become concentrated in socially disadvantaged populations (485).

Local benchmarking. A further role for monitoring population trends is to allow the communities that provided the data to use the results as a stimulus and yardstick for their own efforts. A school-based or local government-based monitoring programme that could measure, in a consistent manner, environmental and behavioural factors that are under its control would be very empowering if it were to be used for benchmarking. Schools regularly use benchmarking for literacy and numeracy outcomes whereby their results are fed back to them as trends over time and in comparison with other schools. If nutrition and physical activity data were to be used in a similar manner it could stimulate action in the area and allow schools to measure the impact of their efforts.

6.2. Prevention and public health: next steps

Child and adolescent obesity prevention has the aim of promoting healthy growth in young people, where healthy growth includes achievement of stature potential as well as weight gains that tracks within the range considered normal weight (453). Given the genetic propensity for a large section of the population to develop obesity in conducive environments, prevention is best targeted at all young people. Public health obesity prevention efforts should thus focus on the entire population, including children and adolescents at high risk for obesity development, but also reaching the larger proportion of young people whose weight is still within normal ranges.

Preventative activities cover a broad spectrum from individual and local group-based initiatives – which are the ones most closely researched and discussed in the previous section – through to organizational, national and international policies. This can be visualized with the diagram given in the introduction and reproduced below, showing how each level of intervention is set within a wider context (Fig. 35).

The recent increases in obesity prevalence are largely attributed to social and environmental forces that are not under individual control – certainly not under the control of children – and which influence eating and physical activity behaviours in the aggregate (484). Thus, although actions to improve individual lifestyles are needed and are perhaps the only recourse for limiting obesity development in the short-term, longer term remedies undertaken from a broader public health and policy perspective will be needed to have a significant impact on the problem. Not only are individually-oriented approaches likely to have limited success if undertaken without supporting social and environmental changes, but an over-reliance on individual approaches with insufficient public health actions may allow the situation to worsen and, particularly, to become concentrated in socially disadvantaged populations (485).

‘Many people believe that dealing with overweight and obesity is a personal responsibility. To some degree they are right, but it is also a community responsibility. When there are no safe, accessible places for children to play or adults to walk, jog or ride a bike, that is a community responsibility. When school lunchrooms or office cafeterias do not provide healthy and appealing food choices, that is a community responsibility. When new or expectant mothers are not educated about the benefits of breast-feeding, that is a community responsibility. When we do not require daily physical education in our schools, that is also a community responsibility. The challenge is to create a multi-faceted public health approach capable of delivering long-term reductions in the prevalence of overweight and obesity. This approach should focus on health rather than appearance, and empower both individuals and communities to address barriers, reduce stigmatization and move forward in addressing overweight and obesity in a positive and proactive fashion.’ Dr D. Satcher, US Surgeon General, 2001 (foreword) (484)

Several authors have identified the macro-environmental factors that promote obesity, such as the trend towards globalization of markets and patterns of economic development, food production, urbanization, media development, and mechanization (16,18,486,487). The societal
factors that predispose populations to obesity are often viewed as desirable, such as greater accessibility of essential goods and services as well as improvements in household income and standard of living. Depending on cultural variations, the changes in lifestyles may include decreased time spent in home food preparation, increased consumption of processed and catered foods, increased mechanization of work and home activities, increased reliance on motorized vehicles and increased access to and use of televisions and computers. These changes inevitably influence social values and cultural norms about eating and physical activity within families, in schools and workplaces and in the society at large.

The following are examples of problematic social trends:

- increase in the use of motorized transport, e.g. to school;
- increase in traffic hazards for walkers and cyclists;
- fewer opportunities for recreational physical activity;
- increased playing of sedentary games;
- multiple TV channels around the clock;
- greater quantities and variety of food available;
- more frequent and widespread food purchasing opportunities;
- larger portions of food;
- rising use of soft drinks to replace water;
- more use of restaurants and fast food stores.

Obesity-promoting social and environmental trends are likely to be self-perpetuating unless they are challenged and successfully redirected. In this respect, the trends in child and adolescent obesity and the emergence of obesity-related diseases during childhood and adolescence take on particular importance in motivating social change, because the importance of contextual influences on behaviour is more easily recognized where children are concerned. In discussions of adult obesity it is often held that environmental forces are subordinate to personal choices in promoting obesity or preventing weight loss. However, the holders of these views will usually acknowledge that the rising prevalence of childhood obesity reflects the potency of environmental influences, including the home environment along with the wider community, and that public health actions may be needed to curtail these influences.

The International Obesity TaskForce (IOTF) causal web of societal policies and processes, shown in Fig. 36, is useful as a general framework for identifying the nature and scope of targets for action and the stakeholders potentially affected (17). No single aspect of this web of policies and processes can be addressed without having a potential impact on other areas, and the interests in these areas may be competing.

A primary goal of public health initiatives to address obesity is to increase the consciousness in the non-health sectors of the potential adverse effects of their actions on the ability of people to maintain energy balance and to increase their interest in and ability to minimize these adverse effects. Furthermore, so fundamental to society are the processes that relate to food intake and physical activity that any initiatives undertaken in relation to obesity must be harmonized with pro-

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**Figure 36** Societal policies and processes with direct and indirect influences on the prevalence of obesity. Vertical and horizontal links will vary between different societies and populations. Source: Kumanyika et al. (17).
Programmes undertaken to maintain other societal core processes.

The need for global co-ordination is also evident from this framework. Many of the economic forces involved are global in nature and any effects of obesity-reducing policies on the relevant industries will undoubtedly have global consequences. Principles underlying general obesity prevention initiatives at the population level have been outlined by IOTF and are shown in Table 15.

Targets for action focus on changes in the nature of the food supply and in the mechanization of physical activity. Strategies can be modelled partly after successful campaigns for tobacco control, automotive safety and recycling – all of which have involved a successful combination of consumer education and advocacy, legislative and policy changes, and community-based programmes (488). Table 16 lists some strategies for obesity prevention initiatives implemented through different sectors, recognizing that the specific type of initiatives needed will differ culturally and between and within countries.

Many of the strategies listed in Table 16 would influence the environments of children. Approaches that specifically target children include changes in the food and activity options in schools, improving infrastructure for walking and access to safe and affordable outdoor play areas, and regulating food advertising on television, particularly advertising that is aimed at children.

The goal is not to find a single programme that works, as this is unlikely to be found, but to stimulate regional, national and local initiatives that are suitable for their context. Initiatives to meet the rising problem must be proportional to their target, for example:

- school curriculum development for health education may need to be set as a national policy;
- sports promotion may need to be centred on school and community programmes;
- food marketing controls may need to be introduced at national, regional or even global level;
- reduced car use and increased walking or bicycle use may be the responsibility of municipal planners working under national guidelines.

### 6.2.1. Involvement of other sectors

It is clear from this approach that some influential stakeholders outside of the health sector may have interests complementary to health sector obesity prevention objectives. Policy development can identify and capitalize upon partnerships with such interested agencies and stakeholders. Within the health sector itself, nutritional objectives for obesity prevention are highly compatible with those for the promotion of cardiovascular health as well as cancer prevention. Similarly, efforts to improve breast-feeding adoption and to increase consumption of fruits and vegetables are consistent with general guidelines for infant and child nutrition.

Outside of the health sector, allies in efforts to increase children’s physical activity may be found among those working to increase the sustainability of the environment, including proponents of sustainable transport and of safer streets or of the development of parks and recreational facilities. In addition, teachers and school administrators, when convinced that healthful eating and physical activity are favourable to academic performance, can also become powerful allies for implementing changes in school environments and extend school sports facilities for local community use. (See for example, the transcripts from the US Healthy Schools Summit at [http://www.actionforhealthykids.org/hss/presentations.htm](http://www.actionforhealthykids.org/hss/presentations.htm).)

School boards may also be encouraged to review their school nutrition policies by direct exhortation from health care professionals. A recent Policy Statement from the American Academy of Pediatrics (489) identifies soft drinks in schools as a risk factor for obesity and is intended for school superintendents, school board members and parents as well as health professionals. It calls on paediatricians to assist in educating school authorities about their responsibilities for the nutritional health of their student body.

Barriers to obesity prevention are economic, cultural and practical. It is well-documented within the field of

<table>
<thead>
<tr>
<th>Table 15 Ten principles upon which to base obesity prevention at the population level</th>
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<tbody>
<tr>
<td>1. Education alone is not sufficient to change weight-related behaviours. Environmental and societal intervention is also required to promote and support behaviour change.</td>
</tr>
<tr>
<td>2. Action must be taken to integrate physical activity into daily life, not just to increase leisure time exercise.</td>
</tr>
<tr>
<td>3. Sustainability of programmes is crucial to enable positive change in diet, activity and obesity levels over time.</td>
</tr>
<tr>
<td>4. Political support, intersectoral collaboration and community participation are essential for success.</td>
</tr>
<tr>
<td>5. Acting locally, even in national initiatives, allows programmes to be tailored to meet real needs, expectations and opportunities.</td>
</tr>
<tr>
<td>6. All parts of the community must be reached – not just the motivated healthy.</td>
</tr>
<tr>
<td>7. Programmes must be adequately resourced.</td>
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<tr>
<td>8. Where appropriate, programmes should be integrated into existing initiatives.</td>
</tr>
<tr>
<td>9. Programmes should build on existing theory and evidence.</td>
</tr>
<tr>
<td>10. Programmes should be properly monitored, evaluated and documented. This is important for dissemination and transfer of experiences.</td>
</tr>
</tbody>
</table>

Source: Kumanyika et al. (17).
Table 16 Potential societal level solutions for obesity prevention

<table>
<thead>
<tr>
<th>Setting or sector</th>
<th>Potential societal intervention</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. National government</td>
<td>• Integrate nutrition, physical activity and obesity prevention objectives into relevant policies and programmes e.g. Conduct obesity impact assessments for all new and existing policies.</td>
</tr>
<tr>
<td>e.g. food and nutrition, transport, education, health, welfare</td>
<td>• Increase ability of low-income populations to buy foods that are rich in micronutrients but low in fat and sugar, e.g. Provide price support for healthy food.</td>
</tr>
<tr>
<td>2. Food supply</td>
<td>• Increase nutrition quality of food served in catering outlets e.g. Introduce award or accreditation schemes for preparation, provision and promotion of healthy food options in catering outlets.</td>
</tr>
<tr>
<td>e.g. manufacture, marketing, distribution, retail, catering</td>
<td>• Improve nutrition quality of general food supply e.g. Develop, produce, distribute and promote food products that are low in dietary fat and energy.</td>
</tr>
<tr>
<td>3. Media</td>
<td>• Help consumers to make informed food purchase choices e.g. Introduce new and improved food labelling schemes (covering fat, energy and salt) which do not mislead the consumer.</td>
</tr>
<tr>
<td>4. Non-governmental/international organizations</td>
<td>• Support action on diet, physical activity and obesity e.g. Develop and implement healthy eating, physical activity and obesity prevention programmes; Advocate for action on diet, physical activity and obesity.</td>
</tr>
<tr>
<td>5. Healthcare services</td>
<td>• Promote healthcare intervention before obesity develops e.g. Provide training in obesity prevention and management for doctors and other healthcare workers.</td>
</tr>
<tr>
<td>6. Education sites</td>
<td>• Promote adoption of healthy activity and dietary habits by patients e.g. Provide physical activity and/or nutrition and cooking skills programmes for patients.</td>
</tr>
<tr>
<td>e.g. pre-school, school, further education</td>
<td>• Reduce advertising and marketing practices that promote over-consumption of food and drink e.g. Regulate television food advertising aimed at children.</td>
</tr>
<tr>
<td>7. Worksites</td>
<td>• Empower students to prepare healthy meals e.g. Subsidize healthy options in staff restaurants.</td>
</tr>
<tr>
<td>8. Neighbourhoods, homes and families</td>
<td>• Increase access of low income groups to healthy food e.g. Set-up community garden programmes and food cooperatives.</td>
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<tr>
<td></td>
<td>• Increase access to safe exercise and recreation facilities e.g. Pedestrianize city centres and residential areas.</td>
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<td></td>
<td>• Promote walking (and cycling), e.g. Set-up walking programmes in shopping malls, parks etc, open safe-cycling routes.</td>
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<tr>
<td></td>
<td>• Increase access to, and consumption of, fruit and vegetables (and encourage physical activity) e.g. Home gardening projects.</td>
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</tbody>
</table>

Source: Kumanyika et al. (17).

Tobacco control and also reported with respect to the promotion of healthy eating (490) that initiatives to curtail the advertising and sale of food elicit strong opposition from commercial interests. For example, in the USA and some European countries, exclusive marketing contracts to sell food and beverage products to school children have been negotiated with some schools, with incentives to school administrators who use the associated revenues to increase resources for school programmes (490). Such policies need to be reviewed and assessed for their potential health impact.

6.2.2. Cultural resistance to change

To find economically viable strategies that can prevent a continuing escalation of the absolute amount of food and calories marketed within countries and across national
boundaries is extremely challenging. Restrictions on marketing and advertising are opposed on the grounds of strong and legally-protected cultural values such as the right to free speech, although marketing and advertising that is aimed at children has come under criticism from consumer organizations. Professional bodies for paediatrics and psychology have also taken positions against the marketing of products that could undermine the health of children.

In addition, consumer demand is a barrier to obesity prevention. Once consumers have become accustomed to and relatively dependent upon the modern food environment, for example abundant quantities of conveniently packaged foods at low cost, efforts to alter this environment in the name of obesity prevention may encounter strong cultural resistance.

There are major economic and cultural implications associated with initiatives to decrease physical inactivity or to increase activity. Small-scale programmes to reduce the time spent in sedentary activities such as television viewing or use of computers or computer games can be effective and tolerated, but large-scale initiatives of the same type may encounter substantial economic and cultural barriers. Sedentary lifestyles are culturally normative and defended as such.

In summary, long-term obesity prevention strategies must be economically viable, culturally acceptable and futuristic. Obesity prevention cannot be accomplished by ‘turning back the clock’ to reverse development and economic advancement. Rather, the challenge is to create environmental incentives and opportunities that will prevent excess weight gain and that are compatible with other aspects of the desired aggregate lifestyles.

6.2.3. Reviewing the science of obesity prevention

As suggested in the discussion of the systematic reviews of management and preventive aspects of child obesity, there is a paucity of properly-conducted randomized control trials of various intervention approaches. Far greater resources are applied to the biological sciences investigating genetic, cellular and hormonal aspects of obesity development than on the social sciences of obesity generation or the costs of obesity in financial, psychological or social terms. Yet obesity is unlikely to prove to be a disorder amenable to a ‘magic bullet’ solution – there is little likelihood that it can be prevented in the mass of the population through the means of pharmaceutical interventions affecting cell activity, say, or through genetic interventions preventing the transcription of obesogenic genes.

Expressed diagramatically (see Fig. 37), the present scientific effort is largely devoted to investigation of factors in the upper boxes, while the most likely arena for

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**Figure 37** Transforming the focus of science.
a genuine long-lasting solution to obesity will be intervention involving factors in the lower boxes. In this respect, science funding bodies need to reassess the direction of their funding and the end results they are hoping to support.

6.2.4. Role of the World Health Organization

The WHO is aware of the need to identify and address international trade and development issues in relation to nutritional health, including advertising and mass communication, world trade agreements, food labelling, novel foods, urban planning and transportation (491). Furthermore, the WHO is committed to providing support to member states to develop national policies and programmes in this area, and is itself developing a global strategy and is seeking interaction with industry to stress the responsibility of the commercial sector in the need to improve diet, physical activity and health. In particular, resolution WHA 55.11 (2002) on sustainable development and health committed WHO to providing assistance to countries to frame policies and implement national plans that promote consumption patterns that are sustainable and health promoting; and resolution WHA 55.2 (2002) urged member states to develop national plans of action on nutrition and physical activity, with strategies on diet that involved all sectors, including civil society and the food industry. The resolution committed WHO to developing a global strategy on diet, physical activity and health within the strategy for non-communicable disease prevention and control.

In the report of the WHO expert consultation on diet, nutrition and the prevention of chronic disease (6), the following recommendations for promoting healthy diets and physical activity are proposed:

- Strategies should be comprehensive in addressing all major dietary and physical activity risks for chronic disease together, alongside other risks – such as tobacco use – from a multi-sectoral perspective.
- Each country should select the optimal mix of actions in accord with national capabilities, laws and economic realities.
- Governments have a central steering role in developing strategies, ensuring they are implemented and their impact monitored over the long term.
- Health ministries have a crucial role in convening other ministries to ensure effective policy design and implementation.
- Governments need to be supported by private sector bodies, health professions, consumer groups, academics and the research community.
- A life-course approach, in which the origins of chronic disease such as obesity are recognized in maternal and infant nutrition and care practices, is essential.
- Strategies need to address inequalities and focus on the needs of poorer communities and population groups, and be gender sensitive.
- Individual countries may be limited in their actions by international norms and standards, for example in the regulation of trade and marketing practices, and WHO’s international leadership role is essential in these respects.

The document also calls for more ‘enabling’ environments (which enable people to make healthier choices) and in particular:

- Supporting the greater availability of nutrient-rich foods such as vegetables, legumes, whole grains, lean meats and low-fat dairy products.
- Promoting changes in agricultural policies which increase sustainability of food production and the environmental resources, including changes away from resource-intensive forms of farming and the production of tobacco and animal-based foods.
- Protecting traditional diets where these promote health and are environmentally sustainable.
- Encouraging reduced dependence on motorized transport and increased access to recreation facilities that encourage physical activity.
- Encouraging standards to ensure health information is widely available and easily understood, and health messages are relevant and consistent.

6.2.5. Professional bodies and international organizations can assist the process

Professional organizations and groups such as the International Association for the Study of Obesity and the International Obesity TaskForce can offer support to the WHO and to member countries to develop National Obesity Action Plans. Such Action Plans may need to prioritize child and adolescent obesity prevention. Examples of Action Plan priorities might be to:

- encourage schools to enact coherent food, nutrition and physical activity policies;
- design secure play facilities and safe local roads, parks and open spaces;
- restrict advertising and marketing to children of energy-dense, low nutrient foods;
- develop fiscal incentives to help provide more nutritious food for children;
- ensure the participation of child health professionals in national policy formulation;
- develop obesity prevention guidelines and best practice case studies for policy implementation.

Policies at international and national level down to local and community level need to be examined for
their potential impact on health. Where direct evidence is lacking, assessments of policies and interventions must be made on the basis of indirect evidence and on the probabilities of hazards and benefits. Professional organizations and international groups are well placed to contribute to this process.

The present report has been prepared by the International Obesity TaskForce, a component of the International Association for the Study of Obesity, and represents the IOTF’s commitment to encouraging the prevention of child and adolescent obesity and to providing advice and support to the WHO and to member countries in the development of appropriate strategies and policies. The process needs to be started urgently – targets need to be set and evaluation schemes put into place. Continued monitoring of the progress towards the goals will help to ensure that the policies are being enacted and that they are having the necessary effect.

Achieving these aims will require much imagination and perspiration, but the world’s children deserve no less.

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