

Chapter 1.1

Definition, prevalence and historical perspectives of obesity in adults

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1.1.1 Definitions of *overweight* and *obesity*

In clinical practice, body fat is most commonly and simply estimated by using a formula that combines weight and height. The underlying assumption is that most variations in weight for persons of the same height is due to fat mass. The formula most frequently used in epidemiological studies is the *body mass index* (BMI), which is weight in kilograms divided by the square of the height in metres. BMI is strongly correlated with densitometry measurements of fat mass adjusted for height in middle-aged adults. The main limitation of BMI is that it does not distinguish fat mass from lean mass. Table 1.1.1 identifies the cut-off points applied by the World Health Organisation for BMI classification in adults [1].

Although BMI is used to classify individuals as 'obese' or 'overweight', it is only a proxy measure of the underlying problem of excess fat. As a person's body fat increases, both their BMI and their future risk of obesity-related illness also rise, although there is still some uncertainty about the exact nature of the relationship, especially in children.

Measurement of body circumference is an additional indicator of health risk in an overweight or obese person: excess visceral (intra-abdominal) fat is a risk factor for long-term conditions independent of total adiposity. Waist circumference and the ratio of waist circumference to hip circumference are practical measures for assessing upper-body fat distribution.

Skinfold thickness, measured with calipers, provides a more precise assessment of body fat, especially if taken at multiple sites. Skinfolts are useful in the estimation of fatness in children, for whom standards have been published. However, the measurements are more difficult to make in adults (particularly in the very obese), are subject to considerable variation between observers, require accurate calipers and do not provide any information on abdominal and intramuscular fat. In general, they are not superior to simpler measures of height and weight. Table 1.1.2 lists the practical measures for the assessment of an obese person.

Why use BMI?

BMI is an attractive measure because it is an easy, cheap and non-invasive means of assessing excess body fat. Prior to the application of BMI, clinicians referred to 'ideal' weight tables, which were derived from the weight–height tables provided by the Metropolitan Life Insurance Company (1959), based on subsequent mortality of insured adults in the USA and Canada [2]. However, prospective epidemiological data confirmed the impreciseness of the term 'ideal' even within a North American population (despite attempts to sharpen this with measures of body frame size), and its inapplicability when applied in a global context. BMI has been used widely around the world, permitting comparisons between areas, across population sub-groups and over a long period. Another advantage

Table 1.1.1 Cut-off points applied by the World Health Organisation for the classification of overweight and obesity

BMI*	WHO classification	Popular description
<18.5	Underweight	‘Thin’
18.5–24.9	Healthy weight	‘Healthy’
25.0–29.9	Overweight	‘Overweight’
30.0–34.9	Obesity I	‘Obese’
35.0–39.9	Obesity II	‘Obese’
40 or greater	Obesity III	‘Morbidly or seriously obese’

*BMI is the weight in kilograms divided by the square of the height in metres.

Data sourced from http://apps.who.int/bmi/index.jsp?introPage=intro_3.html

Table 1.1.2 Clinical methods for the assessment of an individual with obesity

Characteristic of obesity measured	Methods
Body composition	BMI Underwater weighing Dual-energy X-ray absorptiometry (DEXA) Isotope dilution Bioelectrical impedance Skinfold thickness
Regional distribution of fat	Waist circumference; waist-to-hip ratio Computerised axial tomography Ultrasound Magnetic resonance imaging (MRI)
Energy intake	Dietary recall or record ‘macronutrient composition’ by prospective dietary record or dietary questionnaire
Energy expenditure	Doubly labelled water Indirect calorimetry (resting) Physical activity level (PAL) by questionnaire Motion detector Heart rate monitor

of BMI is the availability of published thresholds and growth references to which children’s BMI can be compared. BMI in children varies with age and gender, which prevents the use of fixed thresholds as in adults [3,4]. Equivalent growth

references do not exist for other measures such as waist circumference.

Interpretation of BMI: defining a ‘healthy weight’ for a particular society

There are methodological problems that derive from a definition based on total mortality rates. People frequently lose weight as a consequence of illness that is ultimately fatal, which was unrecognised at the time of the survey. This gives the appearance of higher mortality among those with lower weights: reverse causation. The effect can be minimised by either excluding persons with diagnoses that might affect weight and/or those who report recent weight loss, or excluding those who die during the first years of follow-up. A second major concern is confounding factors that may distort the association between body weight and mortality – cigarette smoking is of particular importance. Overweight and obesity cause or exacerbate a large number of health problems, both independently and in association with other diseases, and are among the most significant contributors to ill health [5]. Unfortunately, many of the health risks associated with increasing body weight begin their manifestation in children and young people – of great current concern is the increasing prevalence of type 2 diabetes and associated medical complications in young overweight adults. There is a close relationship between BMI and the incidence of many long-term conditions caused by excess fat: type 2 diabetes, hypertension, coronary heart disease and stroke, metabolic syndrome, osteoarthritis (OA)

Table 1.1.3 Risks and diseases associated with increasing body weight

Metabolic syndrome	30% of middle-aged people in developed countries have features of metabolic syndrome
Type 2 diabetes	90% of people with type 2 diabetes have BMI >23 kg/m ²
Hypertension	5x risk in obesity 66% hypertension linked to excess weight 85% hypertension associated with BMI >25 kg/m ²
Coronary artery disease (CAD) and stroke	3.6x increase in risk of CAD for each unit change in BMI Dyslipidaemia progressively develops as BMI increases from 21 kg/m ² with rise in small-particle LDL 70% of obese women with hypertension have left ventricular hypertrophy Obesity a contributing factor to cardiac failure in >10% patients Overweight/obesity plus hypertension associated with increased risk of ischaemic stroke
Respiratory effects	Neck circumference >43 cm in men and >40.5 cm in women associated with obstructive sleep apnoea, daytime somnolence and development of pulmonary hypertension
Cancers	10% of all cancer deaths among non-smokers related to obesity (30% of endometrial cancers)
Reproductive function	6% of primary infertility in women attributable to obesity Impotency and infertility frequently associated with obesity in men
OA	Frequent association in the elderly with increasing body weight – risk of disability attributable to OA equal to heart disease and greater to any other medical disorder among elderly
Liver and gall bladder disease	Overweight and obesity associated with non-alcoholic fatty liver disease and non-alcoholic steatohepatitis (NASH); 40% of NASH patients are obese; 20% have dyslipidaemia 3x risk of gallbladder disease in women with BMI >32 kg/m ² ; 7x risk if BMI >45 kg/m ²

and cancer. An overview of the association between BMI and the development of a range of diseases is given in Table 1.1.3.

Risk factors for some conditions start to increase at relatively low BMIs (e.g. hypertension and type 2 diabetes). It is found that 85% of patients with hypertension have a BMI of >25 kg/m², and 90% of those with type 2 diabetes have a BMI of >23. In 2011, an estimated 62% of adults (aged 16 and over) were overweight or obese in the UK. The risk of developing type 2 diabetes is about two times more for people who are obese as compared to lean people. Abdominal obesity is a particular risk for the cluster of diseases that have become known as the *metabolic syndrome* – type 2 diabetes, hypertension and dyslipidaemia – and is strongly linked to the risk of cardiovascular disease [6]. Most of the medical complications will not present to a medical practitioner until the age of 40 years – hence, it is difficult

to discern the immediate benefits to younger generations from modest weight loss. In prevention terms, the benefit that an individual may feel may be relatively quick to manifest, but the overall benefit to society will take years [7].

1.1.2 Prevalence and trends for obesity in adults

In the UK, data on overweight and obesity among adults (16 years and older) are derived from the Health Survey for England [3]. Results from the 2011 survey are summarised in Box 1.1.1. A comparison with international findings is given in Figure 1.1.1.

A recent review of international comparisons has collated published and unpublished studies on BMI to provide a comprehensive global dataset covering

Box 1.1.1 Summary of the results from the Health Survey of England 2011 [3]

- An estimated 62% of adults (aged 16 and over) were overweight or obese.
- Around 2% were underweight, and 3% had severe obesity (BMI 40 or greater).
- Men and women have a similar prevalence of obesity, but men are more likely to be overweight (41%, compared to 33% in women).
- The prevalence of obesity in adults rose from 15% in 1993 to 25% in 2011. Many of those in the obese category have a BMI much higher than 30. There are more women than men with extremely high BMI values.
- The prevalence of obesity and overweight changes with age, being lowest in the 16–24 years age group, and generally higher in the older age groups among both men and women.
- Women living in more deprived areas have the highest prevalence of obesity, and those living in less deprived areas have the lowest. There is no clear pattern for men.
- Women from the African American racial category appear to have the highest prevalence of obesity, and men from Chinese and Bangladeshi groups the lowest, based on the most recent data (2004). However, research has shown that BMI may overestimate obesity among Africans and underestimate obesity in South Asians.
- Using adjusted thresholds for these ethnic groups could improve obesity estimates.

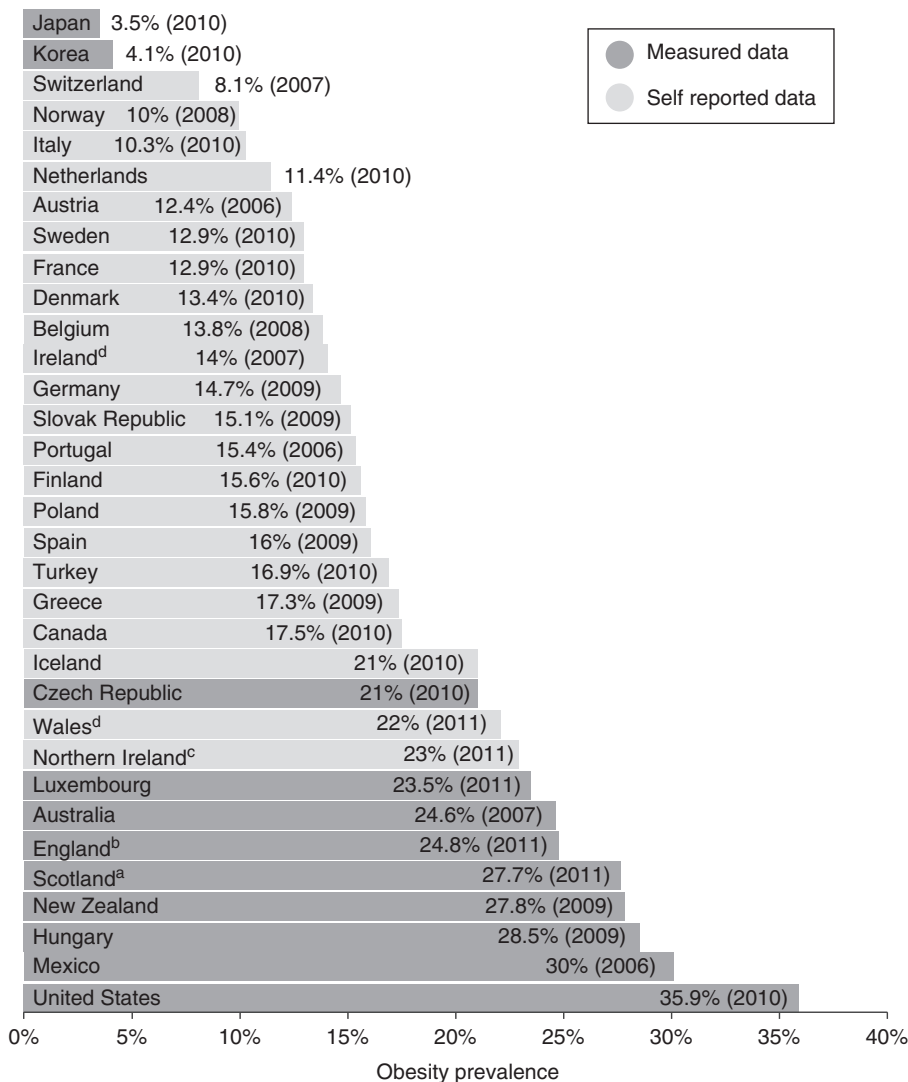


Figure 1.1.1 International comparisons of the prevalence of obesity (BMI 30 kg/m² and greater). Published with acknowledgment from Public Health England. Source: http://www.noo.org.uk/NOO_about_obesity/adult_obesity/international.

the years 1980–2008 [8]. The data was derived from both measured and self-reported estimates and included 199 countries and territories. Between 1980 and 2008, age-standardised mean global BMI increased by 0.4–0.5 kg/m² per decade in men and women. The reviewers noticed substantial differences across regions and gender. The region with the largest BMI rise was Oceania for both sexes, and the regions with almost flat trends or even potential decreases were Central and Eastern Europe for women and Central Africa and South Asia for men. The estimated value for the UK at 24.8% is high compared to most other countries, as is the rate of increase (almost double) during the past 25 years [3].

1.1.3 Recent history of obesity in adults

From an evolutionary point of view, excess body fat has served nature's purpose by providing a superbly efficient system for storing energy reserves. This system only became maladaptive when technological advances altered the balance between the availability of food and the body's expenditure of energy. During pre-historic times, the greatest burden for mankind was infection and famine. Natural selection rewarded the 'thrifty' genotypes of those who could store the greatest amount of fat from the least amount of food and release it as frugally as possible. Those who could store fat easily had an evolutionary advantage in the harsh environment of hunters and gatherers. This precarious food supply was gradually reduced with the advent of agriculture and domestication of animals – humankind developed the ability to grow its food, but supply still remained precarious due to the vagaries of nature and warfare.

Food shortage, famine and infection remained the biggest challenges to the world's population until the twentieth century. The current global epidemic of obesity is a problem of the late twentieth century, and now impacts across societies. Modern technological and scientific advances have resulted in an abundance of food in many countries, although many tragically still face chronic food shortages. However, it is only within the last 50 years that

overweight and obesity have come to be regarded as long-term conditions associated with well-defined comorbidities; prior to this, corpulence was often regarded by many societies as desirable and a reflection of well-being.

The increasing prevalence of obesity is closely associated with the emergence of modern lifestyles in the UK and other developed countries in Europe, North America and Asia. Towards the close of the nineteenth century, medical concerns were raised about obesity. In 1900, the UK was already the world's most urbanised and one of the wealthiest countries, with a large service sector, expanding public transport network and a developing consumer culture.

Living standards continued to rise during the years between World Wars I and II (1919–1939) despite high unemployment and economic depression. With falling prices and smaller families, real incomes per capita increased by about one-third. Britain's growing prosperity was not shared equally, and substantial sections of the working class continued to suffer from undernutrition.

Sir George Newman, the Chief Medical Officer at the Ministry of Health in the UK, held 'excessive and unsuitable food combined with lack of fresh air and exercise' responsible for sowing the 'seeds of degeneration' [9]. He accepted that some persons 'no doubt' were 'under-fed' in 1931, but argued that many were 'over-fed' – giving their poor bodies little rest, clogging them with yet more food'.

Doctors began to take interest in obesity, and *The Lancet* noted in 1933 that in 'these days of "slimming" there was no more popular subject of discussion among the laity than the reduction in weight' [10]. Such a preoccupation might appear incongruous at a time of economic depression, high unemployment, hunger marches and working-class poverty, but has been seen again in the present century. Between the wars, obesity was noted to be increasingly associated with the comforts of suburban middle-class life, plentiful food and a rapid rise in car ownership.

After 1939, the introduction of extensive rationing with a strict regulation on food supplies resulted in reductions of sugar, meat and fats, with consumption shifting to bread, potatoes and milk [11]. This substantial governmental intervention, which was only possible in the context of World War II,

amounted to a major turning point in the history of the British diet. The policy has been described as a revolutionary transformation because it largely eliminated the disparities of energy and nutrient intake between social classes. Energy consumption declined, and physical activity levels rose among the middle classes due to longer working hours, increased walking, reduced motoring (owing to petrol rationing) and schemes such as ‘Dig for Victory’ [12].

Nobel laureate Robert Fogel has examined health, nutrition and technology over three centuries, concluding that a synergy between improvements in productive technology and human physiology has enabled humans to more than double their longevity and to increase their average body size by more than 50% [13]. Fogel argues that larger and healthier humans have resulted in accelerated economic growth that, in turn, has led to reduced workload and increased leisure time. Increased longevity has also resulted in increased demands for healthcare. The erosion of class differentials in food intake persisted after the end of rationing in 1954, and weight gain was no longer confined to the highest-income groups. Since the 1950s, the British diet has been characterised by ever more abundant cheap food. In recent decades, the traditional three-meals-at-home pattern has been replaced by snacking, junk foods and takeaways, contributing towards weight gain. This has been compounded by a decline in physical activity, with expanding car ownership and new forms of home-based entertainment. The increasing problem of overweight and obesity in children and adults highlight the limits of personal responsibility when considering daily food consumption. However, policies aimed at addressing this imbalance have avoided any form of regulation.

The UK’s Foresight programme, in the Government Office for Science, was asked by government in 2005 to consider how society might deliver a sustainable response to obesity in the UK over the next 40 years [7]. Foresight works across government departments to analyse complex cross-cutting issues. The analysis of published evidence revealed that the causes of obesity are embedded in an extremely complex biological system, set within an equally complex societal framework. Furthermore, the scale of the challenge

to prevent obesity is substantially increased by the complex nature of the condition. The many causes of obesity argue against depending on a number of unconnected solutions to address the issue, and against focussing on single aspects because this is unlikely to bring about the scale of change required.

In 2010, a new coalition government in the UK prompted a rethink about social policies and public health. The new Secretary of State for Health launched a policy of ‘nudge’ when addressing the British Medical Association’s conference [14]. The Right Honourable Andrew Lansley cited evidence that ‘if we are constantly lecturing people and trying to tell them what to do, we will actually find that we undermine and are counterproductive in the results that we achieve’. Mr Lansley went on: ‘Behaviour change is the great challenge for public health – but too often it is ignored. Public health efforts, which only try to control supply, will fail. We have to impact on demand. That means we have to change behaviour, and change people’s relationships with each other and with drugs, alcohol, tobacco and food. The fact is, you can’t legislate for self-esteem from Westminster. We can’t pass the Elimination of Obesity Act 2010. Our Government will be a much smarter one, shunning the bureaucratic levers of the past and finding intelligent ways to encourage support and enable people to make better choices for themselves’.

Sadly, the historical evidence from public health initiatives and the complexity of the scientific basis of obesity suggests limited success from the policy of ‘nudge’. As Foresight concluded, a long-term comprehensive strategy needs to incorporate a range of policies. Importantly, the implementation of such strategies may have limited impact on the current generation of adults, but lead to long-term benefit in younger generations. Benefits from integrated and sustained strategies should lead to a progressive decline in obesity prevalence across all generations and an accompanying reduction in obesity-related diseases [7].

Lessons from the history of obesity

Too often, it seems that we concentrate on the present without reflecting on lessons from the past.

The following is a summary of knowledge about the growing prevalence of overweight and obesity.

- The history of consumption and living standards in Britain during the twentieth and early twenty-first century points towards the close relationship between obesity and modern affluent lifestyles characterised by abundant food and increasingly sedentary habits.
- Extensive rationing and controls on food reversed these trends during the 1940s and early 1950s. The policy was only possible in the context of war and does not offer a practical solution for the public health problems of the early twenty-first century.
- Inter-war weight loss manuals did not pay much attention to energy counting, but rather emphasised a holistic approach to transform lifestyles by adopting healthy habits as the key to successful, permanent weight reduction.

1.1.4 Genetics of obesity

The suspicion of an inherited background to obesity has existed over many generations. Carl Van Noorden in 1907 delineated two type of obesity – exogenous and endogenous [15]. He suggested that exogenous obesity, which accounted for the majority of cases, was the consequence of external elements, namely food consumption in excess of energy expenditure. Nevertheless, the group with endogenous obesity had an intrinsic problem that led to hypometabolism. Jules Hirsch observed in the 1950s that there is a ‘biochemical or biological element’ to will power, basing the observation on the life-long struggle faced by many obese persons to maintain weight loss over the longer term [16]. Ethan Sims demonstrated in a prison population that the majority of inmates (over)fed 10,000 kcal/day over 200 days had difficulty in maintaining their weight gain, whereas a small number, with family history of obesity, gained weight swiftly and struggled to lose the weight once the overfeeding had ceased [17].

The seminal studies of twins by Albert Stunkard and colleagues provide additional strong evidence

for an inherited basis to obesity. From the Danish Twin Registry, Stunkard identified twins who had been adopted early in life and therefore separated from their biological parents. Despite a shared environment with the adoptive parents, the adoptee twins’ BMIs were more closely associated to that of their biological rather than the adoptive parents. Accordingly, most adoptees inherited their biological parents’ obesity [18]. On reviewing identical twins registered by the Swedish registry, Stunkard found that identical twins had virtually the same body weight regardless of whether they had grown up together or separately [19]. A subsequent study by Bouchard and colleagues, which involved overfeeding 12 pairs of identical male twins over 100 days, demonstrated a wide variety of responses across the twin pairs; however, within each twin pair, there was little difference in the weight gained and even less difference in body fat distribution and visceral fat accumulation [20].

With the advent of molecular genetics, extensive mapping of obesity-gene mutations in rodents began in earnest. Jeffrey Friedman and colleagues cloned the gene *ob*, which encodes leptin [21]; their findings resulted in a transformation in obesity research. Thousands of articles have been published and substantial research funds invested in pursuing the relevance of leptin and related genes, including ghrelin, neuropeptide y and MCHC, in human obesity. However, the signalling pathways of molecules involved in appetite; and the genetic mutations that may interfere with these pathways. More recently, a common variant of the fat mass and obesity-associated gene *FTO* and genetic variants of *DYRK18* have been associated with human obesity [22]. This is evidence that provides fuel to the argument that body weight regulation is not simply governed by ‘energy in – energy out’.

Nevertheless, evidence remains that few are predestined to obesity, while many are predisposed to weight gain. Genetic predisposition in tandem with the development of food environments that encourage overeating and built environments that discourage energy expenditure are undoubtedly explanations for the increasing prevalence of obesity observed during the last four decades.

1.1.5 Summary box

Key points

- In clinical practice, body fat is most commonly and simply estimated by using a formula that combines weight and height – BMI. Measurement of body circumference is an additional indicator of health risk in an overweight or obese person: excess visceral (intra-abdominal) fat is a risk factor for long-term conditions independent of total adiposity.
- Overweight and obesity cause or exacerbate a large number of health problems, both independently and in association with other diseases, and are among the most significant contributors to ill health.
- Between 1980 and 2008, age-standardised mean global BMI has increased by 0.4–0.5 kg/m² per decade in men and women.
- The history of consumption and living standards during the twentieth and early twenty-first century points towards the close relationship between obesity and modern affluent lifestyles, characterised by abundant food intakes and increasingly sedentary habits.
- Evidence confirms that few individuals are predestined to obesity, while many are predisposed to weight gain. Genetic predisposition in tandem with the development of food environments that encourage overeating and built environments that discourage energy expenditure explain the increasing prevalence of obesity observed during the last four decades.

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Chapter 1.2

Definition, prevalence and historical perspectives of obesity in children

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An epidemic of childhood obesity occurred over the past few decades, affecting all age and social groups in the developed world. The prevalence of childhood obesity appears to have stabilised in some developed countries in recent years, but at an unacceptably high level. Therefore, obesity continues to be one of the most common child health problems, and a major challenge to public health. Developing countries have also experienced rising childhood obesity rates in recent years. The aim of this chapter is to summarise the evidence, using good-quality systematic reviews where available, on the worldwide prevalence of obesity among children; the development of the childhood obesity epidemic in the UK and USA; and the identification of subgroups of the population at greater risk of becoming obese. This chapter will also discuss how to define obesity in children for epidemiological purposes (i.e. to monitor and compare prevalence at the population level), whereas the criteria for diagnosing obesity in children for clinical management will be covered in Chapter 3.2.

1.2.1 Definition of obesity in children

Obesity is an excess of body fat, which is associated with increased health risks. Subjective assessment of obesity in children is inaccurate, and therefore obesity should be defined using objective methods [1]. Direct measures of adiposity, such as densitometry and dual-energy X-ray absorptiometry, are

more accurate than indirect methods (e.g. waist circumference, skinfolds, body mass index, bioelectrical impedance), but are not practical for epidemiological studies or clinical use [2]. A critique of the different methods to measure adiposity and define obesity in children is available elsewhere [2,3]. There is widespread international support for the use of body mass index (BMI; ratio of body weight to height squared – kg/m^2) to define childhood obesity in epidemiological studies and in clinical practice [1–7]. Although BMI is not a direct measure of adiposity in children, it correlates well with more accurate measures of body fatness, it is practical to measure and a high BMI for age is associated with both high fat mass and increased risk to health [1–3,7–10].

As BMI varies with age and gender in children, BMI values are compared with age- and gender-specific national reference data using cut-off points in the BMI distribution (BMI percentiles or standard deviation scores, SDSs) to identify obesity in children [4]. National BMI reference data are available in many countries including the UK [4] and the USA [11]. The UK 1990 reference data represents the BMI distribution of UK children in 1990, are widely available in the form of BMI centile charts and can be used as a baseline against which subsequent obesity trends can be compared [4]. Epidemiological studies commonly use the ≥ 85 th centile of BMI for age and gender and the ≥ 95 th centile of BMI for age and gender based on national (country-specific) reference data as the percentiles to identify overweight and obesity in children [1].

These percentiles define overweight and obesity reasonably well, and children with BMI in the ≥ 95 th percentile are at high risk of obesity-related conditions [1,5,7,10]. Age- and gender-specific waist circumference percentiles are available for the UK and elsewhere but have not been used in epidemiological studies to estimate obesity prevalence.

An alternative method for identifying obesity in children was developed by Cole et al. in 2000 [12]. This method provides age- and gender-specific BMI cut-offs that correspond to the adult cut-offs for obesity (i.e. BMI of 30 kg/m^2) and are based on international data collected from six countries (UK, Brazil, Hong Kong, the Netherlands, Singapore and the USA). These cut-offs, commonly referred to as the *International Obesity Task Force* (IOTF) *definition of obesity*, allow international comparisons of childhood obesity prevalence, but are not intended for clinical or national epidemiological use [12]. Detailed discussions of the ongoing debate surrounding the use of national and international methods to classify healthy weight, overweight and obesity in children are available elsewhere [1,12,13]. Briefly, both methods are widely used to define obesity in epidemiological studies; however a recent systematic review found that the IOTF method was much less sensitive (poorer accuracy) than national reference methods for detecting obesity in children [1]. The IOTF cut-offs for BMI are useful for worldwide comparisons of childhood obesity prevalence [2], but will give more conservative estimates of obesity prevalence, particularly in boys, compared to methods based on national (country-specific) reference data [1,2].

1.2.2 Trends in the prevalence of childhood obesity

Comparison of obesity rates among countries is difficult as different methods have been used to define obesity. Further differences can be due to different study designs, the range of ages of the samples included and the timing of the surveys [2]. Despite these problems, several systematic reviews have examined trends in the prevalence of childhood obesity in developed and developing countries over the past few decades [2,14–20].

The global situation

The prevalence of childhood obesity has increased markedly over the past few decades in most developed countries and in several developing countries [2,14,15,17]. Thus, obesity in children is a serious global public health problem. Prevalence is particularly high in the USA, Canada, Australia, Japan and parts of Europe, with obesity rates doubling or tripling from the 1970s to late 1990s in these countries [2,14,15]. In Europe, the highest levels are reported in the UK and many southern European countries, such as Greece, Spain, Malta, Portugal and Italy [2,14,15,18]. Parts of South East Asia and much of sub-Saharan Africa appear to have the lowest prevalence [2,14]. Some developing countries have experienced a marked increase in obesity prevalence, associated with rapid socioeconomic growth, including Brazil, Chile and urban regions of China [2,14]. Data from Brazil demonstrated a threefold increase in obesity prevalence among children and adolescents from 1974 to 1997 [14]. Obesity is more prevalent in wealthier groups and urban populations in developing countries, whereas children in the lowest SES groups are more at risk in developed countries [2,14].

Recent systematic reviews [16,18–20] have indicated that the prevalence of obesity in children has stabilised (i.e. no change in obesity prevalence) or levelled off (i.e. change in the trend from an increase towards stability or a slowing down in the increase) since the early 2000s in several developed countries including Australia, most of Europe, Japan and the USA. However, levels remain high in these countries, and childhood obesity continues to be a major public health issue. In addition, it is unclear if this stabilisation is a long-term change or a temporary phase that may be followed by a further increase [20]. Additionally, obesity prevalence has continued to rise in some countries (e.g. China and Vietnam), and recent trends for Africa, South America and much of the Middle East have not been reported [20].

Development of the obesity epidemic in the USA and UK

In the USA, the National Health and Nutrition Examination Survey (NHANES) has monitored

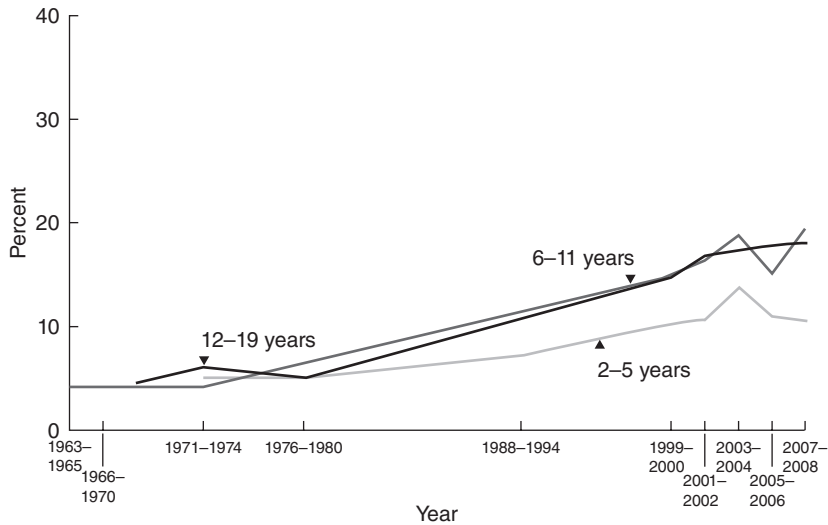


Figure 1.2.1 Trends in obesity among children and adolescents: United States, 1963–2008. Source: From reference [21].

trends in obesity prevalence among children from the 1960s (shown in Figure 1.2.1) using cross-sectional, nationally representative samples of US children [21]. The proportion of obese children and adolescents (defined as BMI ≥ 95 th percentile on US/CDC reference charts) remained stable at 5% from mid-1960s to 1976–1980, but then trebled between 1976–1980 and 2003–2004 [21]. By 2003–2004, 14% of 2–5-year-olds, 19% of 6–11-year-olds and 17% of 12–19-year-olds in the USA were obese [21,22]. The most recent estimate of obesity prevalence based on data from the 2007–2008 NHANES indicates that 10% of children aged 2–5 years, 20% of those aged 6–11 years and 18% of adolescents aged 12–19 years were obese, suggesting that prevalence has stabilised in the USA as previously discussed, although prevalence remains high [21,23].

Childhood obesity increased markedly in the UK from the late 1980s [24–26]. Chinn reported the prevalence of obesity (defined using the IOTF method) in representative samples of English and Scottish children aged 4–11 years using three cross-sectional surveys in 1974, 1984 and 1994 [24]. Obesity prevalence did not increase from 1974 to 1984, but increased substantially from 1984 to 1994 in both English and Scottish children. Similarly, in the 1996 Health Survey for England (HSE), 11% of

6-year-olds and 17% of 15-year-olds were classed as obese (BMI ≥ 95 th percentile). These figures were significantly higher than the 1990 UK reference standard of 5% [25], and indicated that obesity prevalence had doubled in children and trebled in adolescents (relative to the UK 1990 reference data) in a short period of time (shown in Figure 1.2.2). By the 2003 HSE, 14% of children aged 2–10 years and 21% of 11–15-year-olds were obese [27]. The most recent estimate of obesity prevalence based on the 2009 HSE indicated that 15% of 2–10-year-olds and 18% of 11–15-year-olds were obese [28], suggesting that prevalence has stabilised, albeit at a high level [26].

In addition to the rise in the proportion of children classed as ‘obese’, average BMI and BMI z scores have risen across paediatric populations in developed countries over the past few decades, suggesting that most children have been affected by the obesogenic environment (i.e. environments that promote obesity) [19,24,29,30]. In addition, data from measures of body composition and body fat distribution, such as skinfolds and waist circumference, indicate that total body fatness and central fatness has increased over the past few decades among children and adolescents [30–32]. A meta-analysis found significant increases in triceps and

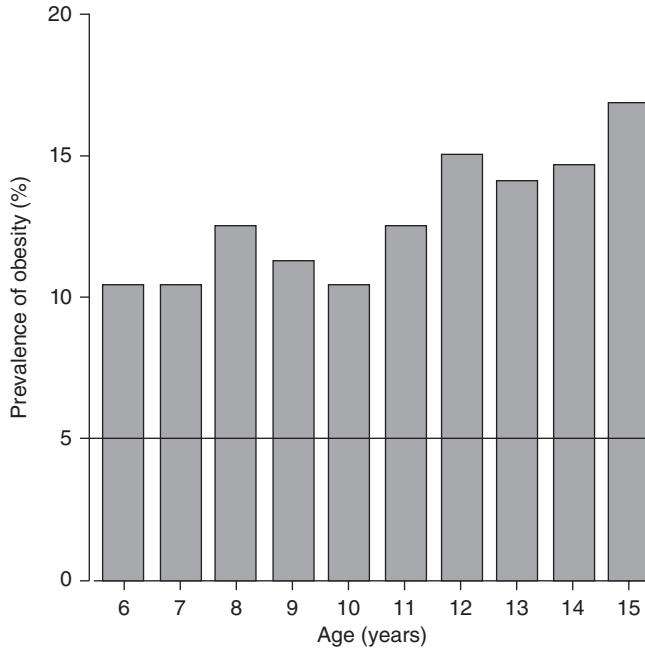


Figure 1.2.2 Prevalence of obesity (BMI > 95th centile) at each age compared with expected frequency (5%, horizontal line). Source: From reference [25].

subscapular skinfold thickness and percentage body fat (estimated from skinfold measures) between 1951 and 2003 in children aged 0–18 years in developed countries, with the rate of increase becoming much steeper in the 1980s [32]. McCarthy et al. used data from cross-sectional surveys conducted in 1977, 1987 and 1997 to examine changes in waist circumference for age (a measure of central fatness) and BMI of adolescents aged 11–16 years [29]. Average waist circumference for age and BMI had increased substantially over the 10–20-year period between the surveys. In addition, waist circumference for age had increased at a faster rate than BMI. The increase in central fatness among children is particularly worrying because central fatness is strongly associated with several cardiometabolic risk factors [1,10].

Obesity prevalence by age, gender, socioeconomic status (SES) and ethnicity

The obesity epidemic has affected children of all ages, both genders, most ethnic groups and all

socioeconomic backgrounds, although some groups have been affected more than others [2,30]. Several studies in the USA have observed higher obesity rates in African American and Hispanic children compared to non-Hispanic white children [22,33,34]. Hedley et al. [34] showed that the prevalence of obesity (BMI ≥ 95th percentile on US/CDC reference charts) among children aged 6–11 years was 20% in African Americans, 22% among Mexican Americans and 14% among non-Hispanic whites. Furthermore, over a 30-year period from 1971–1974 to 1999–2002, Mexican American and African American children experienced much greater increases in the prevalence of obesity than white children [33]. For example, among 6–11-year-olds, the prevalence of obesity (BMI ≥ 95th percentile) increased by 16% among Mexican Americans, by 15% among African Americans and by 10% among white children. Recent data from the 2007–2008 period indicate that ethnic differences in obesity prevalence are still evident [23].

The relationship between obesity and ethnicity in developed countries other than the USA is less clear, although there is some evidence to suggest that some

ethnic minority groups may be at greater risk of obesity [30,35]. A recent systematic review [35] of studies conducted in the UK between 1980 and 2010 concluded that Chinese children appeared to have lower risk for obesity than Caucasian children. In contrast, the literature on obesity prevalence among South Asian and black children relative to Caucasians in the UK was mixed. Several studies observed higher rates of obesity among South Asian and black children compared to Caucasians, whereas other studies observed either lower rates or no differences. The existing literature is limited by a lack of studies using UK representative samples, as well as the failure to consider differences in SES by ethnic group (ethnic minorities in the UK have lower SES than Caucasians). Also, categorising Bangladeshis, Indians and Pakistanis as 'South Asians' may mask the differences in obesity prevalence between these groups and the problems defining obesity using BMI cut-offs in some ethnic minority groups (at the same BMI level, children from some ethnic minority groups have higher fat mass) [35]. Despite these limitations, obesity prevalence appeared to be higher in South Asian boys and lower in South Asian girls relative to their Caucasian counterparts, whereas girls from black subgroups appeared to have higher risk, and boys from black subgroups lower risk, compared to Caucasians. The reasons for possible ethnic differences in obesity rates are not entirely clear, but may be *partly* explained by SES differences [35].

In developed countries, children from families with low SES are at greater risk of obesity [2,30,36–38]. A recent systematic review of cross-sectional studies published between 1990 and 2005 concluded that there is an inverse association between SES (using parental education as the SES indicator) and obesity risk in children from developed countries [36]. The evidence suggested that children from all SES groups have been affected by the obesity epidemic; however, children whose parents (particularly mothers) have a low level of education were at greater risk. Stamatakis et al. [37] examined trends in obesity prevalence by SES in UK children aged 5–10 years using nationally representative cross-sectional data collected over several time periods to 2002–2003. A rapid increase in obesity prevalence was observed in all children, particularly from 1994 onwards; however,

prevalence had increased at a faster rate among children from lower SES groups [37]. A further study by Stamatakis et al. [38] showed that obesity rates had levelled off between 2002–2003 and 2006/2007 among UK children (aged 5–10 years), whereas obesity prevalence had continued to increase among children from lower socioeconomic groups, suggesting that the existing socioeconomic gap in childhood obesity in the UK may be expanding. Similarly, a study examining obesity trends between 2000 and 2006 in Australia reported that prevalence was only increasing in children attending low-SES schools [20]. Reasons for SES differences in obesity rates are complex and require further study, although poor diet and limited opportunities for physical activity may be important factors.

There is no obvious international trend towards systematic differences in obesity rates with age or between the sexes [30]. Although some studies have observed higher rates of obesity among older children (e.g. see prevalence figures in previous section 1.2.2), that may be due to greater exposure to the obesogenic environment with increasing age [30]. Apparent differences in obesity prevalence by gender may be a result of the method used to define obesity; the IOTF method underestimates prevalence in boys more than girls [1]. Thus, several studies have observed higher obesity rates in girls than boys when using the IOTF method, and no gender difference when national reference standards are used [1,2,18,30]. The plateau in prevalence described previously has been observed in all age groups and both genders [19,20,23,26], but this has not been shown in all studies [16].

1.2.3 Conclusion

The prevalence of childhood obesity is high and has increased rapidly over the past few decades in developed countries. Children of all ages, ethnic groups, socioeconomic backgrounds and both genders have been affected, although children from some ethnic minorities and lower socioeconomic groups are particularly at risk. Reasons for ethnic and SES differences are not entirely clear and require further study, although differences in dietary intake and physical activity may be important. Recent data indicate that prevalence of childhood obesity may have stabilised

or levelled off in some developed countries in recent years, although it is too soon to be certain if this change is long-term, and obesity rates have not appeared to stabilise among children from lower socioeconomic groups. Possible reasons for the stabilisation are unknown at present, although public health campaigns to improve dietary and physical activity habits may be at least partly responsible [20,26]. However, prevalence remains unacceptably high, and therefore obesity prevention and treatment strategies are needed for all children and should also target children and families from specific groups. Developing countries have also experienced rising obesity rates among children in recent years, particularly among wealthier groups and urban populations, and recent data suggest that these rates are not levelling off. Overall, childhood obesity is a serious global public health problem, and interventions to prevent and reduce obesity among children are needed worldwide.

1.2.4 Summary box

Key points

- Childhood obesity prevalence is high and has increased rapidly over the decades in developed countries.
- Children from some ethnic minorities and lower socioeconomic groups are particularly at risk. Reasons for ethnic and SES differences are unclear, although differences in dietary intake and physical activity may be important.
- Recent prevalence of childhood obesity has levelled off in developed countries, although obesity rates have not among children from lower socioeconomic groups.
- Prevalence remains unacceptably high; obesity prevention and treatment strategies are needed for all children, and should target children and families from specific at-risk groups.
- Prevalence of childhood obesity in developing countries reflects a rise in obesity among children, particularly among wealthier groups and urban populations.
- Childhood obesity remains a serious global public health problem, and interventions to prevent and reduce obesity among the young are required internationally.

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Chapter 1.3

Development of overweight and obesity across the life course

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Obesity and overweight are persistent conditions and are compounded by the gradual age-dependant involuntary weight gain that occurs in adulthood (approximately 0.5kg per year). In order to understand this unconscious weight gain, this chapter will address each of the key risk periods across the life course.

1.3.1 Childhood: birth to early school age

Childhood and adolescence are risk periods for excess weight gain. The Early Bird study identified the period from birth to 5 years of age as the stage when the majority of excess weight gain occurs, prior to puberty [1]. More recently, a considerable increase in weight gain has also been shown between children after 5 years of age, when formal schooling begins (school year one), and towards the end of primary/elementary school (school year six). The data in Figure 1.3.1 use information collected from the National Child Measurement programme in the UK to report the changes in school children's body mass index (BMI) [2]. For this report, the years 2006–2007 and 2014–2015 were considered [2]. Figure 1.3.1 highlights the recent trends in terms of obesity and overweight, and within that the trends evident in different subgroups of children. These changes in weight and growth rates reflect substantial changes in the environment and in the way children grow and live. Furthermore, weight gain may be occurring in children who are overweight and

moving towards obesity, but these changes are often known to be frequently overlooked by their parents [3]. This 'normalisation' of obesity and overweight may hamper approaches to challenge excess weight gain.

1.3.2 Adolescence and young adulthood

Weight gain is rapid in the translational period spanning adolescence to young adulthood [4]. Birth cohorts for surveys completed in 1998, 2003 and 2008 were used to examine weight and BMI changes over time in different age groups. Results highlighted the temporal increases in obesity rates between different age groups. Most cohorts were heavier during the period spanning 1998–2003 than in the 2003–2008 surveys. Overall increases between 1998 and 2003 were 1–1.5 kg/m² units in BMI and 2–7 cm in waist circumference, greater in the younger age cohorts. Famously, the Freshman 15 study in the USA highlighted relocation – in particular, a move away from home for education – as a key driver for weight gain. This included an average weight gain in the first semester ranging from 3.5 to 7.8 lbs (1.6 kg–3.5 kg) [5]. It may be that the additional freedom of food choice, lack of accountability and the opportunity to explore alternative dietary and lifestyle choices favours weight gain.

Inactivity has also been suggested as an important factor in weight gain at this developmental stage. A number of studies have explored possible

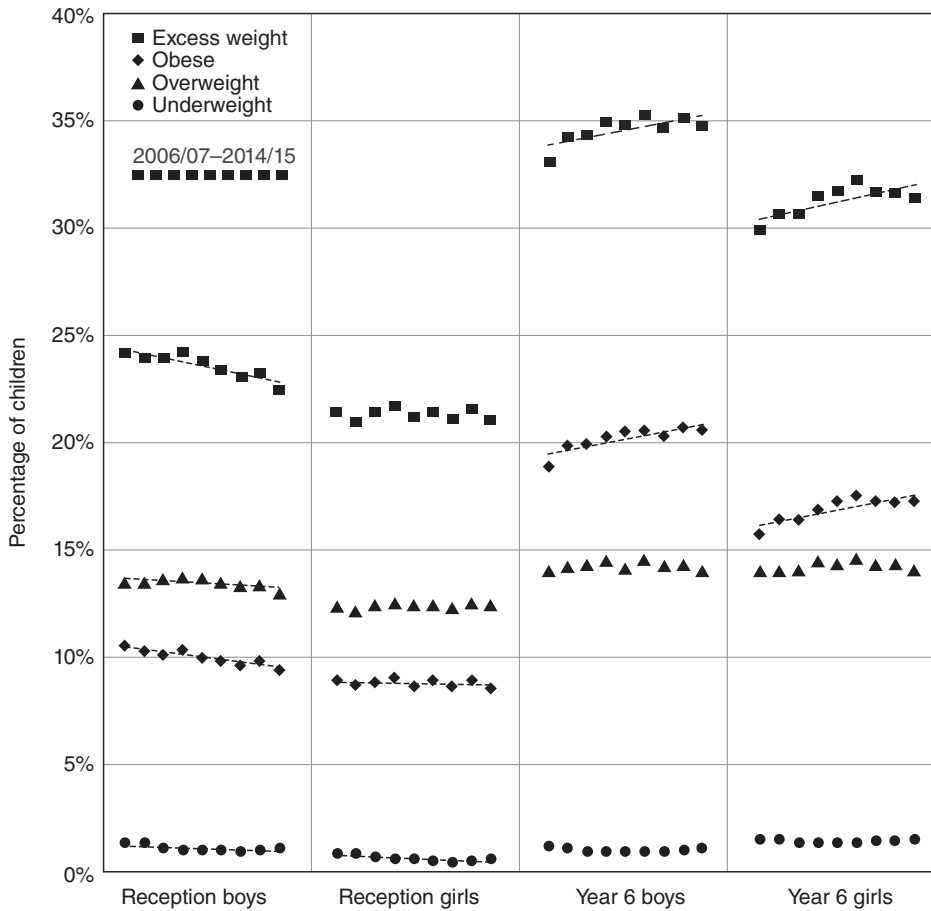


Figure 1.3.1 Prevalence of obesity, excess weight, overweight and underweight by year of measurement, school year and sex. NCMP 2006–2007 to 2014–2015. Significant upward or downward linear trends are shown with a dashed line [2]. Published with acknowledgment to Public Health England.

links with computer gaming/screen time as a proxy for inactivity. Cross-sectional and prospective associations between computer gaming and overweight ($\text{BMI} \geq 25 \text{ kg/m}^2$) have been observed in women, with adjustment for age, job and amount of activity, among other factors. For men, only cross-sectional associations could be seen [6]. The impact of screen time on weight gain has also been reported by the findings of a Swedish study. This impact is likely to persist across the life-course, given the habitual nature of the use of games or mobile devices.

1.3.3 Marriage or cohabitation and body weight

The formalisation of a relationship via marriage or as cohabitating partners, with or without financial commitment, may lead to changes in lifestyle patterns and behaviour. Being part of a settled relationship favours contentment, a situation that itself encourages weight gain [7]. Married respondents were usually heavier than both never-married and divorced respondents [8]. Observations have suggested that food becomes central to a relationship as

couples may have more time together to share mealtimes and make food purchases. Perhaps contentment and security reduce the importance of appearance or awareness of body weight, either subconsciously or otherwise.

Given that marriage and in fact parenthood are associated with weight gain in general, the US CARDIA study data were used to try to understand the impact of marriage in differing environmental settings [9]. Data on neighbourhoods compared married adults living in more affluent areas – with lower population density, better environments for food purchasing and more public facilities for sport and activity – with their unmarried neighbours exposed to the same environment. There was a positive association between weight gain and marriage in white, affluent couples, in comparison to their unmarried neighbours. Marriage showed no such associations with the same comparisons made in less affluent couples, living in poorer settings [9].

Given the recognised variation in marriage duration, a review investigated the associations between marital transitions, BMI and body weight [10]. Fourteen studies described transitions both in and out of marriage. The positive association between marriage and weight gain was supported, while dissolving a marriage was associated with weight loss. The range of weight changes vary according to the duration of the study period [10]. A short-term study suggested a weight gain of around 1.5 kg after 3 months of cohabitation [11]. Although the lack of any comparator in this study meant that the impact of moving in together was unclear, more certainty was seen in analyses of national survey data, which allowed comparison between newly married adult couples and those already married. The newly wedded adults had gained an additional 2 kg after a year [12].

For a range of possible reasons, the impact of leaving a marital relationship was associated with weight loss, although the impact of divorce has been considered as only ‘temporary’ [8]. Using data from men who either divorced or remained married, the impact on BMI in each category after more than 20 years was limited. Both groups of men gained weight, and the BMI rose from a healthy weight to an overweight category. This mirrors the weight gain seen across most Western populations.

Despite being a complex setting, with many compounding factors, marriage or cohabitation, but not ceasing of a marriage, may in certain circumstances offer opportunities for interventions to promote healthy weight programmes.

1.3.4 Pregnancy

Pregnancy is often a time of great anticipation, especially for healthy mothers without any specific concerns about the health of their unborn child. Anecdotally, many pregnant women regard pregnancy as an opportunity to bloom, disregarding any restrictions they usually apply to the energy they consume. Pregnancy is probably the only period across the life course when positive encouragement for weight gain is given by many. Eating for two, although widely known to be a myth, appears attractive to many who choose to believe it. Increasingly, given the worldwide epidemic of obesity, excessive weight gain in pregnancy has been less widely accepted, but it does still have a cross-cultural impact. Excessive gestational weight gain is associated with adverse infant, childhood and maternal outcomes, and research to develop interventions to address this issue is ongoing [13].

In the UK, there are no data to assess how many pregnant women are obese or overweight. The Centre for Maternal and Child Enquiries [14] conducted a UK-wide audit of obesity during pregnancy for a 2-month period during 2009. The results disclosed that 5% of all women who had given birth (≥ 24 weeks’ gestation) were identified as having a BMI of $\geq 35 \text{ kg/m}^2$ at any time during pregnancy. Even more concerning was that the median BMI for these women was $>35 \text{ kg/m}^2$. This was 39.1 kg/m^2 , and 2% had a BMI of ≥ 40 (morbid obesity). Moreover, 0.2% of pregnant women reported having a BMI of ≥ 50 , categorized as super-morbid obese.

Recognizing the issue of obesity and excessive weight gain in pregnancy, the United States Institute of Medicine [15] produced some guidance material for weight gain, which differed according to individuals’ initial BMI. This issue was judged as important, given the demographic changes that had taken place in those women who were becoming

pregnant. They were older at conception, tended to gain more weight than in the past, were more likely to have a twin or triplet birth, and were more likely to be overweight or obese at conception. A recent pilot observational study aimed to determine the feasibility of implementing a programme within the UK National Health Service to limit maternal weight gain [16]. The findings demonstrated the difficulties in actually measuring body weight in women across their pregnancy, implying an unwillingness in the women or their health professionals to make weighing a routine practice. This has been demonstrated in other studies [17] involving the measurement of body weight. This highlighted the positive association between higher weight gain and adverse perinatal outcomes. Currently, UK clinical guidelines do not advocate regular weighing during pregnancy [18].

Awareness of the risks of excess maternal weight gain appears to be surprisingly low. The views of women on their gestational weight gain were sought in a survey of almost 500 pregnant women. Weight measurements were made at their 12th-week clinic

appointment, and their opinions of weight gain were sought [19]. Over half of all respondents were obese, and 62% were living in areas of mild to moderate deprivation. Over three-quarters of participants felt dissatisfied with their current weight, although a majority (60%) expressed limited concern about potential weight gain. Also, 39% were unconcerned about weight gain during their pregnancy, including 34 (19%) who reported having retained the weight gained in earlier pregnancies. These data suggest a lack of awareness among obese women regarding excessive gestational weight gain. A pilot study in pregnant women of low socioeconomic status suggested the ability of health professionals to reach and engage successfully with these women was poor. Despite them recognising the importance of the issue of obesity and excess gestational weight gain [20].

It is clear that opportunities exist to challenge the excessive weight gain observed in pregnancy. To date, the various approaches attempted have met with only limited success. While an opportunity is present, it remains unclear how best to reduce the

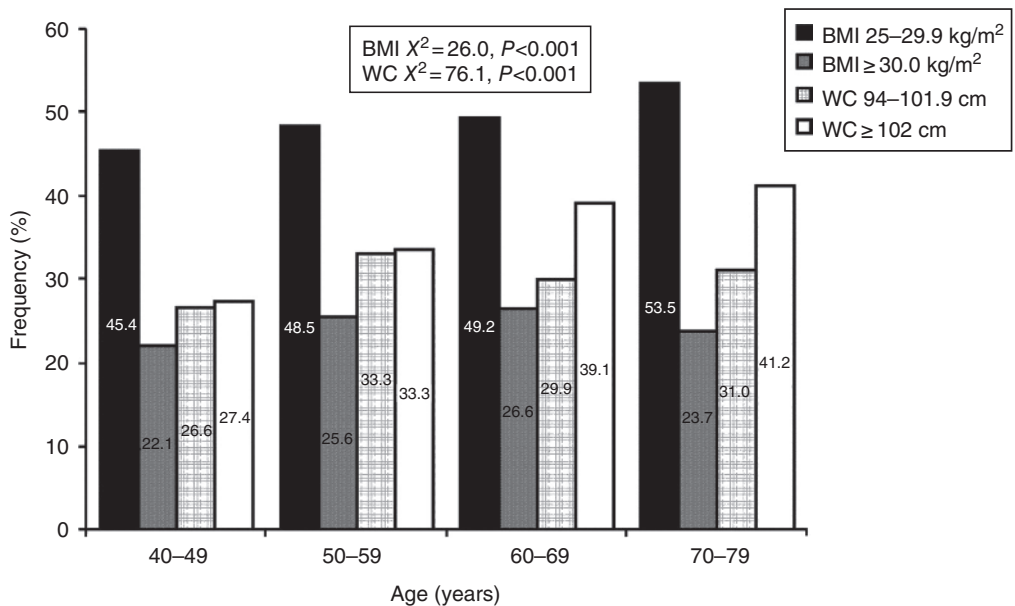


Figure 1.3.2 Distribution of subjects with high BMI (25–29.9 kg/m² or ≥30 kg/m²) or high waist circumference (94–101.9 cm or ≥102 cm) by 10-year age groups. Source: Taken from Han et al. 2015 [21].

elevated weight gain associated with pregnancy without incurring a detrimental impact on infant and maternal health.

1.3.5 Retirement and aging

Obesity is increasing in older adults, a category where obesity was traditionally considered unusual. The European Male Aging Study noted an unremitting increase in BMI and waist circumference from middle age until the seventh and eighth decades [21] (Figure 1.3.2).

A large US study highlighted a positive relationship between childhood poverty and downward socioeconomic status across the life course and an increase in older-age obesity and overweight [21]. This study demonstrates that increased body mass in middle and late adulthood is a likely consequence of the complex interplay among individuals' genes, dynamic socioeconomic experiences and historical context in which they live. While BMI changes after 55–60 years of age, waist circumferences continue to increase, indicating increasing body fat with reduced muscle mass [4]. It has been speculated that the loss of occupational role/status, leading to a lack of structure to the day, limited empowerment, a reduction in social contact and financial concerns may lead to depression, which is strongly associated with obesity and weight gain [21].

Retirement involves adapting to multiple new demands (reduced income, social isolation, grandchildren responsibilities, bereavement, etc.), but few people currently make structured forward preparations for life or health after retirement. However, some responsible employers and national pre-retirement agencies already provide courses on financial planning, adult education and keeping fit. The potential to deliver a more structured approach for diet and lifestyle changes, to preserve function and physical and mental health, is important for this high-risk group. Reducing or retarding chronic disease, compressing morbidity and optimising physical and mental health would improve functional capacity, quality of life and cognitive function to favour prolonged independent living, and enable continued contribution to domestic and social life. This would benefit the

individual, the family and the economy as a whole. To date, however, the opportunity has yet to be fully realised.

1.3.6 Conclusion

Weight change across particular phases of the life course are increasingly common. Many populations, both from the more economically developed and less economically developed nations, are becoming more affluent and therefore gaining weight. The epidemic of obesity, when considered in detail, appears to show that specific periods of life are particularly associated with weight gain. Opportunities for tailored and appropriate interventions to manage weight are available.

1.3.7 Summary box

Key points

- Specific key periods across the life course are often associated with weight gain.
- Obesity prior to pregnancy and obesity during pregnancy are increasingly common.
- Excess weight gain evident in young children is frequent but often goes unnoticed, or is not a concern to parents/carers.
- Awareness of weight gain may be limited with an increasing acceptance and normalisation of obesity.
- Obesity in older adults is increasingly common, bringing with it many of the comorbidities usually seen earlier in life.

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Chapter 1.4

Diagnostic criteria and assessment of obesity in adults

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Obesity is defined as a disease by the World Health Organisation. Its International Classification of Diseases code has subcategories of some historical interest, which do not correspond well to the clinical or public health concerns about obesity, or to management strategies. Most people think they know what is meant by 'obesity' in principle, but different criteria may be applied in relation to different intervention or treatment strategies, and between different populations or subgroups. The diagnostic criteria for other diseases such as hypertension or hyperlipidaemia similarly vary between genders and racial groups, or between children and adults, so different diagnostic criteria are used at different ages to initiate and assess treatment.

Modern obesity research has revealed complex genetic–environment interactions in its aetiology, and the interacting neural and endocrine components that modulate appetite and eating. New management packages, with better-defined goals and success criteria, have emerged, and so diagnostic criteria have come under new scrutiny. Importantly, in order to establish diagnostic criteria, a robust definition of obesity is required, to characterise the disease. In principle, obesity is obviously a disease of having too much body fat, but measuring body fat is not simple. Furthermore, any diagnosis based on measuring body fat, which is a component of both normal and obese individuals, will inevitably have to rely on arbitrary criteria. Body fat can only accumulate over time in people who are (for genetic and/or environmental reasons) predisposed. The

2010 Scottish Intercollegiate Guidelines Network (SIGN) guideline on obesity [1] took the important step of defining obesity as a disease process: '*Obesity* is defined as a disease process, characterised by excessive body fat accumulation, with multiple organ-specific consequences'. Accepting that there is a disease process operating, it becomes possible, if we have reliable markers of the process, to diagnose it before its full clinical manifestation becomes apparent.

1.4.1 Indicators of rising body fat content

Direct measures of body fat include computer tomography, magnetic resonance imaging, bio-electrical impedance analysis or dual-energy X-ray absorptiometry scan. These tools are primarily used in obesity research, whereas in routine clinical practice other anthropometric measures are used as a proxy measure of body fat.

1.4.2 Weight and body mass index (BMI)

The simplest indicator of obesity, or body fat content, is of course body weight. For monitoring patients over time, weight is the most reliable measure. However, body weight varies with stature as well as fatness. Many mathematical corrections have

Table 1.4.1 International classifications and sub-categories for BMI (adapted from references [2–4])

Classification	BMI (kg/m ²)	
	Principal cut-off points	Additional cut-off points
Underweight	<18.50	<18.50
Severe thinness	<16.00	<16.00
Moderate thinness	16.00–16.99	16.00–16.99
Mild thinness	17.00–18.49	17.00–18.49
Normal range	18.50–24.99	18.50–22.99 23.00–24.99
Overweight	≥25.00	≥25.00
Pre-obese	25.00–29.99	25.00–27.49 27.50–29.99
Obese	≥30.00	≥30.00
Obese class I	30.00–34.99	30.00–32.49 32.50–34.99
Obese class II	35.00–39.99	35.00–37.49 37.50–39.99
Obese class III	≥40.00	≥40.00

been applied to eliminate the influence of stature. The BMI (kg/m²) is not the best of the many indices proposed, but for adults it does eliminate most of the effects of stature, and it is simple enough for wide use in epidemiology. Women have more fat and less muscle than men at any given weight or BMI, so the sexes should always be separated in any analysis.

WHO adopted the now-conventional BMI categories for overweight and obesity in 1995, 2000 and 2004 (Table 1.4.1) for health monitoring in populations, and to establish associations between diet and lifestyle and health outcomes [2–4]. These are arbitrary BMI cut-offs, and the detailed health–outcome associations of BMI, or BMI categories, vary between genders, with age, and between social and racial groups. The conventional BMI categories were not intended for clinical diagnosis of individuals. They are not completely accurate in classifying individuals' body fat contents, so some people such as sportsmen, classified as 'obese' with BMI > 30 kg/m², actually have low body fat, but have expanded muscle masses. Muscle is more dense than fat, so variations in muscle mass introduce rather large errors in BMI classification.

It remains true that *most* people with BMI > 30 kg/m² in a population survey do in fact have excess body fat, to a degree that it is likely to affect health adversely. Almost nobody with BMI > 35 kg/m² has an acceptable amount of body fat. In the category with a BMI of 25–30 kg/m², a relatively large number are en route to, or at risk of, a BMI of > 30 kg/m². Hence, these conventional BMI categories are still useful in epidemiology. They were designed with European adult populations in mind and do need to be interpreted carefully when applied to groups with unusual features. Thus, a mean BMI of 27 kg/m² is of concern for the whole adult population of UK, with mean age about 45 years. However, a mean BMI of 27 kg/m² for a group of young people in their 20s would be of much greater concern. South Asian adults generally have more body fat, and less muscle, than Europeans at any given BMI, so a mean BMI of 27 kg/m² would also be of greater concern in a group of South Asians, implying more frequent attributable health problems [4]. Older BMI criteria had different cut-offs for men and women. This makes sense if the concern is the *absolute* level of risk attached to high body fat content: women can clearly tolerate great fat masses more than men

(being smaller, they need a greater proportion of body fat than men to survive long famines). The same BMI cut-offs are now applied to men and women to indicate similar *relative* excess of health impairment for both sexes.

The BMI is likely to remain a key indicator of body fat and obesity for surveys in the foreseeable future. It is essential for the measurements of weight and height to be made as reliably, accurately and precisely as possible. Equipment must be calibrated regularly, particularly stadiometers used to measure height, since squaring height to compute BMI also squares any errors and biases in its measurement.

1.4.3 Waist circumference

The waist circumference was introduced into public health thinking very specifically in 1996, in the SIGN obesity guideline, not as a diagnostic criterion, but as a tool for health promotion [5]. The BMI was too complex and conceptually problematic for health promotion among the general public, even with the weight vs. height charts to compute it. Research found that waist circumference was in fact a marginally better indicator of both total body fat and associated health risks than BMI, in addition to being simpler.

As with BMI, cut-offs are arbitrary. The waist cut-offs that prevailed, and which have now entered both public health and clinical guidelines worldwide, emerged from a pragmatic risk analysis of a large Dutch database to incorporate both BMI and the older marker ‘waist/hip ratio’, supported by correlations with risks of diabetes and cardiovascular risk factors. Waist >102 cm for men and >88 cm for women form ‘Action Level 2’ (broadly equivalent to BMI 30, obesity), above which risks are high and professional help is required for weight loss. The lower ‘Action Level 1’, of waist circumference >94 cm for men and >80 cm for women (similar to BMI 25 kg/m²), indicates a rising risk and the need for individuals to take personal action against weight gain [6]. Subsequent research has confirmed that waist circumference is at least as good as BMI, and in some cases marginally better, to predict obesity-related health risks, particularly type 2 diabetes.

The earlier term ‘waist–hip ratio’ (WHR) proved to be less accurate than waist alone, but does relate to type 2 diabetes in cross-sectional surveys [7], because the onset of diabetes incurs a loss of muscle mass, and hence a drop in hip circumference. (Subsequent research showed that hip circumference relates more to muscle mass than to fat.) For all practical purposes, it is unnecessary to adjust waist circumference for height.

There is potential for observer-error in measuring the waist, but this is not a major concern if it is done carefully. It should be measured according to the WHO guidelines – with the subject standing, using an inelastic tape, horizontal, between the iliac crest and lowest rib (which can be felt with a finger even in the most obese) [8]. An error of 1–2 cm in this position makes very little difference. Most people categorised as ‘obese’ on the strength of a waist greater than 88 or 102 cm would have excess body fat, even with relatively inexpert measurement. In extreme obesity, the belly becomes pendulous, hanging towards the ground, and so variations in body fat will no longer be detectable. Waist only correlates accurately with body fat content in people with BMI below about 40. However, this is not an obstacle to categorising, or diagnosing, people as obese.

At lower levels of total body fat, waist circumference might misclassify people as being overweight or obese, because it is specifically increased in people with larger-than-usual amounts of intra-abdominal fat. In large population surveys with a wide range of BMI, etc., most of the variance in waist circumference relates to variations in total body fat content: waist is primarily a marker of total body fat, and a little better than BMI for this purpose. In groups with similar BMI, variance in waist circumference is driven more by variations in intra-abdominal fat mass. In a group of individuals with exactly the same total body fat, different waist circumferences would indicate differences in intra-abdominal fat.

There are important differences in waist circumferences and fat distribution among different races (more so than ethnic differences). Thus, South Asian people generally have larger waists, with more intra-abdominal fat than Europeans, at the same

Table 1.4.2 Comorbidities risk associated with different levels of BMI and suggested waist circumference in adult South Asians (adapted from IDF-WPR and IOTF 2002)

Classification	BMI (kg/m ²)	Risk of comorbidities	
		Waist circumference	
		<90 cm (men) <80 cm (women)	≥90 cm (men) ≥80 cm (women)
Underweight	<18.5	Low (but risk of other clinical problems)	Average
Normal range	18.5–22.9	Average	Increased
Overweight	≥23		
At risk	23–24.9	Increased	Moderate
Obese I	25–29.9	Moderate	Severe
Obese II	≥30	Severe	Very severe

height and weight. For this reason, South Asians are at greater risk of metabolic complications from obesity at lower levels of waist circumference. In an attempt to equalise the level of risk at which intervention is indicated, a table of modified waist cut-offs has been produced (Table 1.4.2).

‘Waist-deniers’

Confusion about the associations of waist circumference has dissuaded some guideline-writers from accepting it as an alternative, or equivalent, to BMI for predicting body fat and health risks. Its major advantage – its very simplicity – may have engendered suspicion, and tables have been constructed combining cut-offs of BMI and waist. This approach valuably identifies people at risk despite a low BMI; however, risk assessment is most accurate using continuous variables without categorisation. This is not the same as diagnosis, and is unhelpful for health promotion.

Several large surveys have suggested that WHR is better than waist circumference. The Interheart study is one. However, the waist measurement methods did not use the standard WHO method; data came from a large number of different countries, so different methods were used (shortest circumference) [9]. Others have suggested introducing ratios of waist with height to generate an index. This tends to eliminate differences between the sexes, but

at the expense of a more complicated, less immediate message.

1.4.4 Diagnosis of obesity by genetic markers

All disease arises through a conjunction of genetic and environmental factors, many mediated, as with obesity, by the behavioural patterns of individuals. To become obese, an individual must consume more energy than he or she expends. An increase in BMI from 23 to 30 kg/m² requires the accumulation of about 160,000 kcal of excess fat. This has to be consumed over a period of time as energy above the need for energy balance. There are large numbers of physiological mediators of appetite and eating, and of energy expenditure, all of which may be disrupted by genetic factors, either at the level of a gene mutation or through altered expression of a gene. It has long been recognised that there are familial, and probably genetic, factors responsible for obesity, and very large numbers of gene variants have been shown over the years to have weak associations with greater BMI, and effect sizes are generally very small. Evolutionary success has depended on the body weight and appetite being under complex physiological and ultimately genetic control, such that weight loss is opposed by a range of mechanisms all directed at

increasing food consumption and limiting non-essential energy expenditure. Obesity is usually considered to be polygenic, that is, there need to be 'defects', or altered expression, in several regulatory systems simultaneously. These 'defects' can be considered simply exaggerated normal mechanisms for evolutionary survival. None of the genetic variants so far identified, or even combinations, are sufficient to explain the obesity epidemic. The main influence is environmental, but there is growing interest in the role of epigenetics, for example, by studying the effects of diet and stresses in pregnancy to program the expression of genes related to appetite and energy balance (refer to Chapter 3.1). Susceptibility to weight gain can be exaggerated, or unmarked, by treatment with various obesogenic drugs [10].

1.4.5 Obesity diagnosis with clinical staging

A more functional diagnosis of obesity, based not solely on criteria from BMI or other static measure of body fat, includes criteria of clinical staging. This approach is used routinely in cancer diagnosis, where the staged diagnosis defines treatment. The Edmonton system combines conventional BMI categories with a descriptive term to denote the degree of functional impairment. This system was loosely directed towards treatment, and importantly drew attention that there could be an 'end-stage' in obesity – for example, with severe arthritis – in which even successful weight loss would not improve function significantly, and so only symptomatic palliative care is indicated [11] (Figure 1.4.1).

A simplified diagnostic staging was adopted in the 2010 SIGN clinical guideline, more overtly linked to the very limited range of evidence-based treatments available.¹ It again recognised that, for some patients, only symptomatic palliative care would be indicated. Within the SIGN 2010 guideline, the concept and diagnostic category of 'severe and complicated obesity' was recognised. In the past, the term 'morbid obesity' was used by WHO and others for use in epidemiology, to denote the category with BMI $>35\text{ kg/m}^2$. The word *morbid*

(latin, *morbus*=disease) was used in recognition that many people with BMI $>35\text{ kg/m}^2$ were functionally affected and unwell because of their obesity, but many are not, so this is clearly inadequate for diagnostic staging. The term 'severe and complicated obesity' has thus been introduced to identify individuals who require more aggressive intervention, either because their obesity has progressed to an extreme level (e.g. BMI $>35\text{ kg/m}^2$), or because, at a lower level of BMI, they already have serious secondary medical and functional complications, such as diabetes or arthritis. 'Complicated obesity' (which points to clinical complications and treatment indications) is preferable to 'complex obesity' (which can be misinterpreted as referring to aetiology).

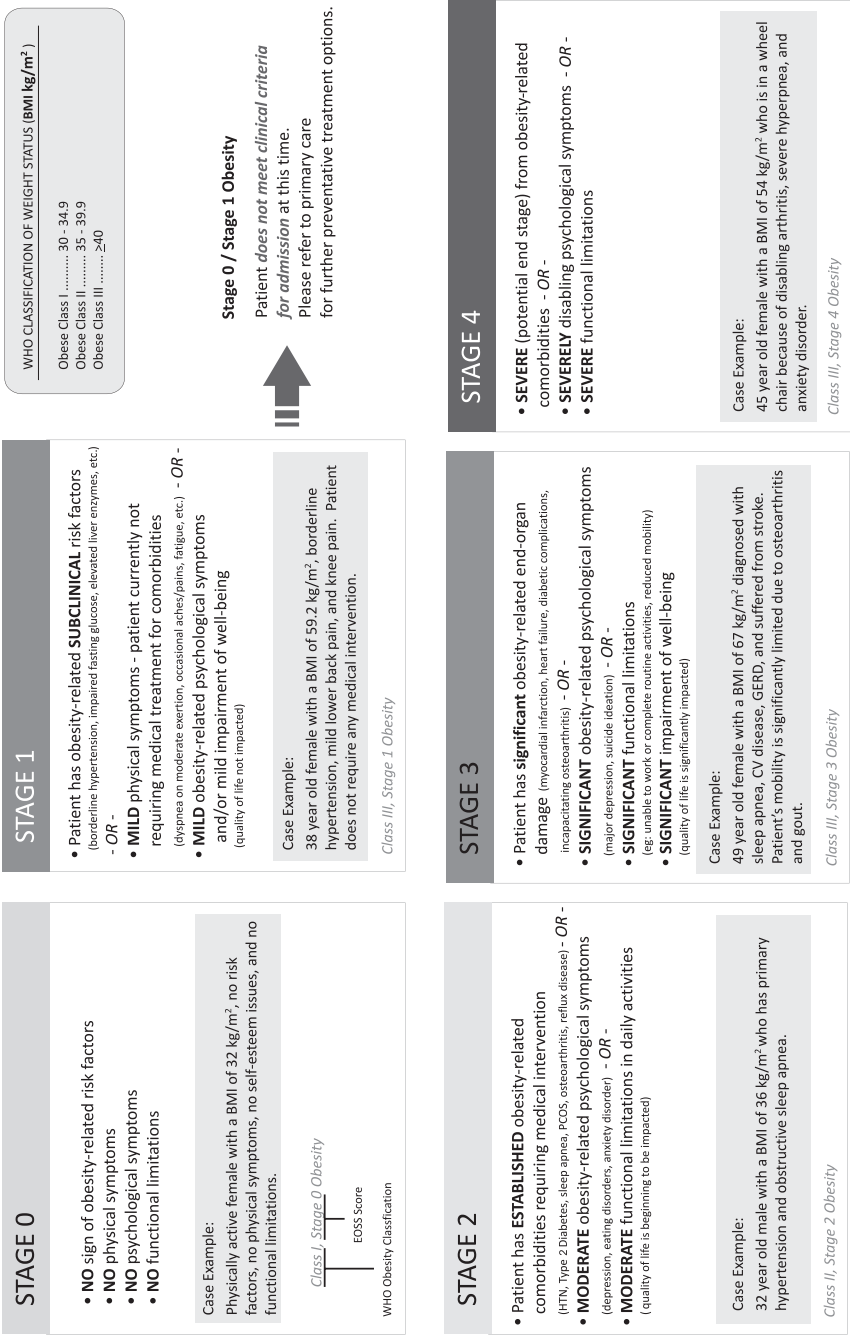
The principle of clinical and functional staging for the diagnosis of obesity is a valuable advance, both for epidemiological classification and to determine clinical action. Adding clinical staging for diagnosis makes it less necessary to modify the BMI or waist categories for different races or ethnic groups, which can be difficult to define in mixed populations.

1.4.6 Summary box

Key points

- Obesity is a disease, defined as the disease process of excess body fat accumulation, with multiple organ-specific clinical and public health consequences.
- A BMI of 30 kg/m^2 is the most widely used criterion for the classification of obesity, in epidemiology, but not for clinical or diagnostic use in individuals.
- For health promotion, waist circumference is the simplest and most robust criterion: $>80\text{ cm}$ (women) or $>94\text{ cm}$ (men) marks a need for self-determined action to avoid further gain; $>88\text{ cm}$ (women) or $>102\text{ cm}$ (men) indicates high health risks and a need for professional support for sustained weight loss.
- For clinical decision-making, clinical assessment of co-morbid conditions must be added to BMI or waist level to determine the appropriate intervention.

EOSS: EDMONTON OBESITY STAGING SYSTEM - Staging Tool



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Chapter 1.5

Diagnostic criteria and assessment of obesity in children

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1.5.1 Introduction

The effective management of childhood obesity is dependent on accurately identifying and assessing the weight status of a child or young person [1]. In addition to the need to tackle the lack of parental awareness and their inability to recognise their child's overweight status results in low levels of weight concern [2]. A health professional is tasked with the role of ensuring that the issue of excess weight is raised in a sensitive manner, in order to help parents understand the potential health risks of their child carrying excessive weight but also motivate them to engage in treatment [3,4]. This chapter provides key aspects of accurately assessing childhood overweight and obesity [3–6]. While there is a constant need to ensure dietetic and medical treatments are clinically and cost-wise effective, it is also important to consider what clinical outcomes are most useful to measure and judge treatment effectiveness.

1.5.2 Diagnosis of overweight and obesity in children and young people

The importance of overweight and obesity lies in the association of excess body fat with ill health. Therefore, the diagnosis of overweight and obesity requires the measurement of body fat. Direct methods of measuring body fat include computer

tomography (CT), magnetic resonance imaging (MRI), bio-electrical impedance analysis (BIA) and dual-energy X-ray absorptiometry (DEXA) scan [1,7–9]. These tools are primarily of greater use in research and occasionally tertiary care centres due to their high costs. In routine practice, body mass index (BMI) is the recommended 'easy-to-use' proxy measure of body fat [1,3–7,9].

Body mass index

BMI [weight (kg)/height (m)²] has been widely used to define and diagnose obesity in adults [3,6,9]. There is widespread national and international support for the use of BMI to clinically diagnose obesity and overweight in children and adolescents [3–7]. However, BMI has well-documented limitations as an absolute measure of body fat, such as that it does not distinguish abdominal fat and visceral fat, it does not take into account excess muscle mass [1,3,9]. However, BMI is the most practical measure to estimate excess body fat in children [1,10].

BMI for age centiles in clinical practice

Child BMI varies throughout growth and differs between sexes, which means that both age and gender needs to be taken into account when interpreting BMI status [10]. Child BMI is classified using thresholds that are derived from a reference population, known as the *child growth reference*. In the

UK, the 1990 population-based BMI centile charts (UK 1990) [11] are recommended for use for those 4 years and above, and WHO data for those under 4 years, available to order from Harlow Healthcare (www.healthforallchildren.co.uk).

In practice, BMI should be calculated and plotted on a centile chart for children above the age of 2 [3–4,6–7]. A cut-off with high specificity (a positive result from a test with high specificity means there is a high probability of presence of the disease) has generally been regarded as more important for clinical applications than high sensitivity (a test with high sensitivity means a high probability of a negative result) to avoid diagnosis of non-obese children as obese [1,3–6,10]. The 91st and 98th centiles on the WHO/UK 1990 charts for age and sex are recommended as the clinical cut-offs to diagnose overweight and obesity, respectively. Table 1.5.1 summarises the clinical centile cut-off points used to diagnose overweight and

obesity in the UK based on systematic reviews [1,10] and evidence-based guidelines [3,6]. International Obesity Task Force (IOTF) cut-off points are also available [12]. A systematic review by Reilly et al. [10] concluded that evidence was lacking for BMI using the IOTF approach in preference to national BMI percentiles for identifying children and adolescents with excess body fat and cardiometabolic risk factors.

Table 1.5.2 demonstrates that BMI can indicate very different weight states in children and adolescents of different ages. For example, a BMI of 19.5 would categorise a 5-year-old girl as obese, a 10-year-old boy as overweight and a 15-year-old boy or girl as being of healthy weight. It is essential that a child's BMI is plotted on a centile chart, or height and weight added to a child BMI calculator that is adjusted for age and sex (see Section 1.5.6, titled 'Useful tools and websites').

Table 1.5.1 Clinical diagnostic criteria for overweight and obese children and young people (aged <18) in the UK

Clinical terminology	BMI centiles*	SDS or z-score*
Overweight	≥91st centile	≥+1.33 SDS
Obesity	≥98th centile	≥+2 SDS
Severe obesity	≥99.6th centile	≥+2.67 SDS
Very severe obesity		≥+3.33 SDS
Extreme obesity		≥+4 SDS

*Defined relative to the WHO/UK 1990 reference chart for age and sex.

BMI for age standard deviation scores (SDSs) or z-scores

BMI can also be defined in terms of a BMI standard deviation score (SDS) or BMI z-score (BMI at 50th centile for age and sex is an SDS of 0) [13]. Software (LMS Growth) is available to convert BMI measurements into BMI SD scores [13]. Expressing the degree of overweight or obesity as an SD score is useful for statistical purposes but also provides more details for children and adolescents at the extremes of BMI, that is, those above the 98th centile. BMI SDSs are now included in WHO/UK 1990 BMI management charts; see Table 1.5.1 for its relation to clinical diagnosis.

There is a need to distinguish those with 'severe' to 'extreme' obesity [14]. Experts have suggested

Table 1.5.2 Absolute BMIs for boys and girls aged 5, 10 and 15 to classify as clinically overweight or obese (UK 1990 cut-offs)

Age	BMI centile	Male		Female	
		91st (overweight)	98th (obese)	91st (overweight)	98th (obese)
5 years		17.4	18.6	17.7	19.1
10 years		19.5	21.7	20.4	22.9
15 years		23.1	26.0	24.2	27.2

that the ‘severely obese’ (≥ 99.6 th centile) are in greater need of clinical assessment and treatment [4–6,15]. In the UK, the ≥ 99.6 th ($+2.67$ SDS) centile line is included on the charts along with centiles for $+3.0$ SDS, $+3.33$ SDS, $+3.66$ SDS and $+4$ SDS (extreme obesity). In the USA, the 97th centile is the highest curve available on the growth charts; hence, cut-offs points for the 99th centile are provided for reference in clinical guidelines [4,16]. Thus, a child with a BMI ≥ 3.33 SD above the mean at age 18 years is the equivalent of the adult definition of morbid obesity (BMI ≥ 40 kg/m²) [17].

Waist measurements

It is recommended that the waist be taken as the mid-way between the lowest rib and the iliac crest, and that the child be asked to bend to one side to locate this point. Several studies agree that waist circumference is a useful tool to provide information on central adiposity, which is associated with high blood pressure, dyslipidaemia and insulin resistance in children and adolescents [19,20]. It is not yet clear what the universal cut-off points should be to assess risk [5,6,10]. Evidence-based clinical guidelines recommend that waist circumference not be used to diagnose overweight and obesity in children [3,4,6,17,18], but that it may be used to give additional information on the risk of developing other long-term health problems and for clinical monitoring purposes [3,7].

1.5.3 Assessment

Once a child has been diagnosed as overweight (above the 91st centile), a key aim of assessment is to build a rapport with the parent and/or child, as many parents have had negative experiences around weight-related issues or be completely unaware of the condition [2,21]. This is discussed further in Chapter 6.1 (weight management in children).

Anthropometric measurements

Accurate measurements of both weight and height are needed to calculate BMI. These should be taken with the child in light clothes and with no shoes. When measuring height, care should be taken to ensure the child’s head is positioned such that the

Frankfurt plane is horizontal (lining up the ear hole with the bottom of the eye socket) [22]. Self-reported weight and height should never be considered appropriate, as this will lead to inaccurate data collection and BMI outcomes.

Waist measurements can be a useful tool in the clinical setting to help children monitor their outcomes; however, care must be taken to ensure that it is measured in a consistent manner – most importantly, following the same technique.

Talking about body weight

Research tells us that more than half of parents cannot recognise when their child is overweight [2], and thus care should be taken when raising the issue of weight with children and parents [23]. Health professionals need to talk openly, but sensitively, about children’s weight [24]. It is important to help educate parents about BMI and to explain the associated health risks at different BMI levels. Although guidelines recommend the clinical terms ‘overweight’ and ‘obese’, these should be maintained for diagnostic purposes only. Weight can be a sensitive and emotive issue, so it would be helpful for health professionals to think carefully about how they approach the subject matter. To avoid parents feeling blamed or judged, using more neutral terms such as ‘weight’, ‘excess weight’, ‘carrying extra weight’ or ‘BMI for age’ (if explained) can be useful [4,24]. Preliminary research shows that weight feedback provided in a factual and non-emotive way by a healthcare professional can significantly influence a parent’s readiness to initiate lifestyle changes [25]. Other factors indicating when a parent may be more likely to be ready to address their child’s weight issues include: the child being aged 8 years or above, and a belief that their child’s weight is a health problem [21,24,26]. Talking about weight at an early stage of assessment can help in exploring the understanding of the parents/family about referral and their possible expectations of treatment.

Clinical factors

At the initial child weight management assessment, it is useful to obtain additional clinical information to build a fuller picture of the child’s obesity and risk of future obesity-related comorbidities. In some

child weight management services, this may be completed as part of a multi-disciplinary team assessment or as part of the criteria required on referral into the service. Clinical obesity guidelines outline a number of areas that are useful to include during assessment [3–7]:

- BMI – plot a growth chart and discuss growth patterns (look out for short stature, as obese children are generally tall) [6,17].
- Weight history patterns, including any previous attempts at weight control
- Family history of overweight/obesity and comorbidities (e.g. type 2 diabetes, prediabetes, hypertension, dyslipidaemia in first-degree relatives)
- Associated comorbidities (such as hypertension, hyperinsulinaemia, dyslipidaemia, type 2 diabetes, and exacerbation of conditions such as asthma) and risk factors
- Level of family support and family setup – that is, who is important at home, who the child spends time with and any other significant carers, such as, for example, grandparents
- Level of emotional/psychosocial distress – for example, low self-esteem, bullying, teasing
- Explore school and social history – this provides an opportunity to engage with the child by exploring his or her world
- Other weight-related signs and symptoms – for example, exercise intolerance, hip/knee/foot pain, shortness of breath, acanthosis nigricans (thickened velvety darkened skin usually around the neck) [6,17]

When to consider a medical referral?

In clinical practice, most children managed in the community will have obesity with no underlying medical cause and no comorbidity [6]. If medical input is required or further investigation is needed to assess risk factors for comorbidities, this can be done in primary care by following recommendations from a range of specialist bodies [27,28]. Some children showing signs of psychological distress or disordered eating

should be considered for referral to psychological assessment and treatment [3,6].

Referral to secondary care or paediatric specialists should only be considered for children or adolescents with very severe to extreme obesity; those who are obese with a serious obesity-related comorbidity that requires weight loss. Another group are those having complex needs such as significant learning difficulties (see OSCA guidance for assessing obesity in secondary paediatric practice in the UK); or as per NICE C43 and SIGN 115 [3,6,17]. Referral to a paediatric endocrinologist should be made for any obese child who is short in stature, as this may be an indication of an underlying endocrine cause of their obesity, such as hypothyroidism, growth hormone deficiency or Cushing's syndrome [6,17].

1.5.4 Treatment success criteria

It is becoming common practice for childhood weight management interventions to be commissioned at a local, regional or national level. The National Obesity Observatory published a Standard Evaluation Framework [29,30] to aid the evaluation of child obesity programmes in the UK. While it does recommend measuring the change in a child's BMI using the BMI SDS rather than the BMI centile, it does not assist in providing guidance on the extent of weight or BMI change required to be clinically efficacious in reducing disease risk.

Due to lack of data, expert clinical opinion collectively agrees that the primary goal of obesity treatment for children who are overweight and most children who are obese ought to be weight maintenance. This is an acceptable target that will create a reduction in BMI because of ongoing linear growth [3–4,6–7]. Table 1.5.3 shows a case study demonstrating this. It is recommended that, in older children and those diagnosed as severely obese, small amounts of weight loss be considered acceptable, up to 0.5–1 kg per month, under clinical supervision [3–4,6].

It is important to note that there is a limited but growing evidence base on the impact of improving

Table 1.5.3 Case study example of a 10-year-old girl and treatment effect

Age (years) at each appointment	Weight (kg)	Height (m)	Weight change (kg)	BMI	BMI SDS
10	50	1.3	—	29.6	3.14
10.5	50.5	1.325	+0.5	28.8	2.95
11	50.5	1.36	0	27.3	2.63
		Change +0.06	Change +0.5	Change -2.3	Change of -0.51*

*SDS is a clinically effective change in adiposity indicator to reduce cardiovascular disease risk.

BMI SDS and on reducing cardiovascular disease risk factors. The small number of studies published suggest that a reduction of 0.1–0.5 BMI SDS after a 1–2-year follow-up is sufficient to show changes in cardiometabolic risk factors, such as improvements in insulin sensitivity, some lipid profiles, blood pressure and body composition [31–36]. This is a promising area for future research, to provide a better understanding of the effects that child weight management interventions have on improving weight status and health.

1.5.5 Conclusion

There is widespread international support for the use of BMI to clinically diagnose obesity in children [3–6]. Despite its well-documented limitations, it is the most practical measure of defining excess body fat in children, provided values are interpreted using relative national child growth references, taking into account age and sex [1,10]. When a child is classified as *overweight* or *obese* (≥ 91 st or ≥ 98 th centile using the WHO/UK 1990 reference [11]), this should be integrated with a comprehensive assessment, including raising awareness of the child's overweight in a sensitive, non-judgemental manner to encourage parents (and adolescents) to help bring their weight under control. Following this, a comprehensive assessment is crucial before deciding upon appropriate treatment options and discussing weight maintenance as a likely clinically effective outcome of the treatment. Further research and guidance is required to help define additional clinical outcomes in order to demonstrate treatment success.

1.5.6 Summary box

Key points

- BMI should be calculated for children.
- BMI needs to be plotted on age- and sex-appropriate charts for each country.
- BMI SDS should be reported for programme outcomes.
- Waist measurement is useful for measuring clinical progress.

1.5.7 Useful tools and websites

BMI management charts and LMS growth (free to download)

Order from Harlow Healthcare,
www.healthforallchildren.co.uk

Online child BMI calculators (adjusted for age and sex)

NHS Choices Healthy weight BMI calculator,
www.nhs.uk/Tools/Pages/Healthyweightcalculator.aspx

Weight Concern child and young person's BMI calculator, www.weightconcern.com/node/9

Talking about weight

Raising the issue of weight in children (download tool), Department of Health (2006), www.dh.gov.uk/prod_consum_dh/groups/dh_digitalassets/documents/digitalasset/dh_096580.pdf

Weight Concern leaflet, www.weightconcern.com/node/134

Online toolkit preventing weight bias, Rudd Center for food policy and obesity, www.yaleruddcenter.org/resources/bias_toolkit/index.html

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