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Introduction

Prevalence

Obesity in childhood is not a new problem. It is the extent to which it is occurring which is new and disturbing because of the long-term implications of overweight for later health (Haslam and James 2005). For many years the prevalence of obesity in children in the UK remained fairly static. Since the mid 1980s prevalence has increased virtually every time it is surveyed. For some years the changing situation was difficult to assess and confirm since there were differences in the methods for assessing obesity in childhood in different surveys. Those differences have now been largely resolved (see Chapter 2) but prevalences continue to rise. Table 1.1 shows the changing prevalence of overweight/obesity in English and Scottish children over recent years. Different surveys involve different populations (England and Wales, UK or smaller geographical area; age range) so absolute figures vary, even for studies in the same year, but the trends remain the same. Figure 1.1 indicates how the prevalences of overweight and obesity in girls and boys have changed over the ten years from 1995 to 2004. There are not only more overweight children but those that are overweight seem more overweight with obese children actually outnumbering those only overweight. The prognosis is grim with suggestions of increases of around 300 000 further obese children by 2010 bringing the total to more than 1.7 million (Zaninotto *et al.* 2006). Currently, there are about 1.25 million overweight and a further 1.4 obese children between 2 and 15 years old in the UK (Zaninotto *et al.* 2006).

Obesity is not simply a concern about size and appearances. Table 1.2 outlines some of the major health and social implications of obesity, both those affecting overweight children and those that have effects in later life. These effects explain why burgeoning obesity has quite suddenly become an issue for governmental as well as medical concern. Unfortunately it seems to be taking time for that concern to translate into effective preventive and therapeutic action (Haslam and James 2005).

Table 1.1. Percentage prevalence of childhood overweight and obesity[a] in UK 1974–2002[b]

Year	1974	1984	1994	1996–7	1998–9	2001/2	2001/2	2003	2004
Boys				5–10 years				2–10 years	
Overweight	11.3	9.8	12.7	16.2	19.7	21.7	22.6	29.7	30.5
Obese	1.8	1.2	2.4	3.4	4.7	5.6	6.0	15.1	15.9
Girls				5–10 years				2–10 years	
Overweight	9.6	10.0	14.4	17.5	19.1	22.6	23.7	25.8	27.7
Obese	1.3	1.8	2.7	4.3	5.3	5.2	6.6	12.4	12.8

[a] Stamatakis *et al.* (2005).
[b] Health Survey for England 2004 (2006).

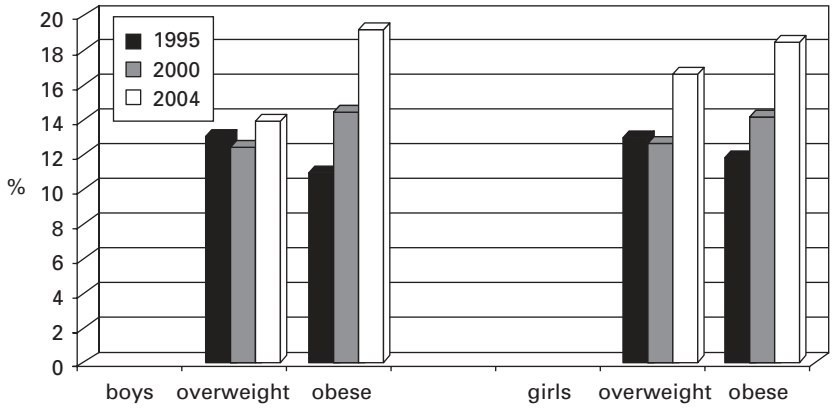

Figure 1.1 Prevalences of overweight and obesity in English boys and girls aged 2–15, 1995–2004. (From Health Survey for England 2004, 2006.)

It is not only in affluent western countries that childhood obesity has become a noticeable concern (Popkin and Doak 1998). Although for the most part at a much lower prevalence, obesity at all ages is becoming increasingly common amongst the more prosperous and urbanized in many of the less developed countries (Araujo *et al.* 2006). It is disappointing that some of these countries have moved from a situation where undernutrition in childhood was a major concern to one where there is now the problem of overnutrition without a noticeable period of normal growth and fatness in between (Popkin and Gordon-Larsen 2004). The disadvantages of westernization have too readily overwhelmed these societies. This creates an interesting pattern of obesity prevalence which has been recognized for many years, namely that obesity tends to be most common amongst the rich in less developed countries and amongst the socioeconomically disadvantaged in more affluent countries. There are exceptions to such a generalization, of course, one example being the high prevalence of obesity amongst children of affluent families in some of the rich oil states of the Middle East (Musaiger *et al.* 2000; Mohammadpour-Ahranjani *et al.* 2004). In the UK, differences in prevalence associated with socioeconomic status (SES) are less obvious in children than in adults (see later).

Why is overweight/obesity so prevalent today?

Genes versus environment

Obesity is almost certainly a combination of genetic and environmental causes. Single gene disorders leading to deficient leptin production, insatiable

Table 1.2. Some immediate and later consequences of overweight/obesity in childhood.

Consequences	Type of problem	When and how	Examples
Health consequences	Physical	Immediate	Physical discomfort
			Intertrigo
			Orthopaedic problems
			Breathlessness on exertion
			Asthma
		Adolescent or adult life	Reduced average lifespan
			Type 2 diabetes
			Metabolic syndrome
			Orthopaedic problems:
			Blount's syndrome
			Slipped upper femoral epiphysis
			Increased risk of some cancers
	Emotional	Immediate	Teasing and bullying
			Embarrassment especially with PE
			Low self-esteem
			School underachievement
		Later	Low self-esteem
			Depression
			Professional underachievement
Social consequences	Discrimination	Employment	Less likely to succeed in job
			Less likely to marry successfully
			Less likely to achieve promotion
		Health insurance	More expensive or unobtainable

		Physical	Public transport often not sized for obese Fashionable clothing may be unavailable in very large sizes Public toilet cubicles/public transport seats/theatre seats etc. too small
Economic	For individual	General expenses	Greater costs for clothing, transport, insurance etc.
		Employment	Working below ability leading to low wages
	For community	Health service costs	Massive costs to public and private health care from problems attributable to or exacerbated by obesity
		Infrastructural costs	Redesigning public buildings and related facilities Public transport/theatres etc. cannot accommodate previous numbers because of greater individual space required

appetite and intractable obesity more or less from birth do exist but are very rare (Farooqi and O'Rahilly 2000). (Leptin is a hormone produced by adipose tissue and is involved in the regulation of appetite and energy metabolism.) Across the world certain ethnic groups, most famously the Pima Indians (Schultz *et al.* 2006), seem at particular risk of overweight/obesity in environments of relative affluence. If we look at other ethnic groups in Europe and North America, it can be difficult to assess populations separately from the implications of their social environments. However Saxena *et al.* (2004) concluded that independent of SES, children of Afro-Caribbean ethnic origin and girls of Pakistani origin in Britain had increased risk of obesity. Boys with family origins in India and Pakistan had increased risk of overweight also. Adults from the Indian subcontinent (ISC) are recognized as at greater risk of the co-morbidities of obesity than white British. There is some evidence that this increased risk may be discernable in children as young as 8 years (Whincup *et al.* 2002). Thus the concern excited by overweight amongst children of UK families originating from the ISC should be greater than for white British children.

Obesity occurring in families is very common (Garn 1976; Poskitt and Cole 1978). How much this is due to common genes or common environments is debated but the explanation for today's prevalence of overweight/obesity would seem to be due to changes in the environment acting on individuals with some susceptibility to overweight (Griffiths and Payne 1976; Romon *et al.* 2005). How much genetic predispositions to the co-morbidities of obesity rather than the obesity itself are also influential is again debated (Bjorntorp 2001). It seems unlikely that there has been sufficient recent change in the gene pool to account for the prevalence of obesity across the world today. Since we can do little to alter the genetic background to the present epidemic our interest will concentrate on environmental changes.

Programming

The past 20 years have seen an explosion of research into the relation of events in fetal and early postnatal life to disease processes in later childhood and adult life, the so-called 'programming' of chronic non-communicable diseases. Some of these studies relate to the 'thrifty phenotype hypothesis' which links prenatal and perinatal events to later obesity and non-insulin-dependent (type 2) diabetes mellitus (Hales *et al.* 1992; Barker 1994). However the results from different studies often seem in conflict (Huxley *et al.* 2002; Singhal *et al.* 2003). Thus low birth weight infants with rapid catch-up growth in early infancy can seem particularly prone to develop overweight/obesity later (Ong *et al.* 2000; Yajnik *et al.* 2003). It is the catch-up growth that is important since studies without documented catch-up growth indicate low birth weight is more likely to be associated with relatively

short stature, underweight and, in women, low fat mass (Li *et al.* 2003). In other studies high birth weight infants are also at risk of later obesity (Baird *et al.* 2005) although Parsons *et al.* (2001) conclude this association may be determined more by the influence of maternal weight and body mass index (BMI) than specifically by the birth weight. Aspects common to many of the growth related factors suggested as risk factors for obesity are good growth (high birth weight) and evidence of ability for rapid growth (postnatal catch-up growth). Since overweight almost inevitably involves a period of accelerated growth, are these factors just showing what must be an inevitable precursor of obesity or are they acting as drivers or programmers of later obesity (Lucas *et al.* 1999)?

Some of these programming events have particularly strong links with the co-morbidities of obesity/overweight such as coronary heart disease (Eriksson *et al.* 1999), type 2 diabetes and hypertension (Barker 1994). However there is not unanimity of view (Huxley and Neil 2004). Social circumstances, country of residence, gender, postnatal nutrition and health of subjects and a wide range of other factors may confound some studies.

Programming studies have led to greatly increased understanding of the development of non-infectious chronic disease. Whilst the findings are very relevant to some overweight/obese, it is difficult to see these studies explaining the high prevalence of overweight/obesity in childhood and adult life that we see today. Birth weights are higher in the past. Severe intrauterine growth retardation is probably less common than in the past because it is recognized antenatally and affected infants may be delivered prematurely before growth retardation has had its full effect. There are more low and very low birth weight infants surviving than in the past. Some of these infants do have rapid early growth and may become fat in the first year, particularly if early nutritional supplementation to encourage early weight gain continues once the infant is progressing well. However this latter group forms a very small proportion of the infants born today and cannot begin to account for the epidemic increase in overweight/obesity. Early infant feeding, although rarely following the recommended exclusive breast feeding until the age of 6 months, is markedly more physiological than in the late 1960s when formula milks had quite dangerous composition, introduction to non-milk, non-formula foods was common in the first month of life and overweight and obese infants were very common (Taitz 1971; Shukla *et al.* 1972). Breast feeding, although not nearly as prevalent as would be wished, is significantly more common and of longer mean duration than was the case in UK 40 years ago. All these changes might suggest – from programming findings – that obesity would be on the decline. Yet, despite infant formulas being refined to a composition in many ways similar to breast milk and despite breast feeding statistics improving, the prevalence of overweight and obese children has increased.

This book is concerned with the management of children who are, for whatever reason, now overweight or obese. It focuses on presenting practical approaches to management rather than on extensively exploring the underlying pathophysiological changes. In this respect, the fascinating work on the fetal and infant origins of disease in later childhood and adult life seem of minor practical clinical relevance here. The risk for type 2 diabetes for example may relate to happenings before birth but the reduction of that risk depends on what happens to that child now and in the future. Whilst catch-up growth may be a critical feature in pre-obese growth for some infants, we believe the main emphasis in infancy should be on the promotion of good weaning practices and healthy lifestyles as children mature. One of us showed many years ago that most infants fat in the first months of life slim down by 5 years old (Poskitt and Cole 1977). The present obesogenic environment may have reduced the chances of fat infants slimming, but it is difficult to blame this on early programming since the infantile obesity seen in the late 1960s was much more prevalent than today. The second 6 months of life – the time of weaning and increasing weight bearing activity for the infant – seems much more critical for the development of persistent obesity (Tate *et al.* 2006). Trying to control weight gain, other than making sure feeding is appropriate, in very young infants brings a risk of affecting the overall nutrition of infants whose unusual patterns of growth may only reflect the expression of genetic potential for growth released from intrauterine constraints.

Family history

The one common risk factor in more or less all studies of the epidemiology of obesity in childhood is a family history of obesity (Poskitt and Cole 1978; Garn and Lavelle 1985). Some 70 to 80 per cent of obese children have one obese parent and 20 to 40 per cent have both parents obese. Whilst children and parents usually share an environment and thus some of this obesity is environmentally induced, studies in the 1960s and 1970s on mono- and dizygotic twins (Børjeson 1976), on twins reared apart (Stunkard *et al.* 1990) and on adopted children (Stunkard *et al.* 1986) suggested that the genetic predisposition to obesity was more dominant than the shared family environment in determining body size. More recent studies show the same findings in that adiposity correlations were still greater for monozygotic twins than dizygotic twins even when reared apart (Bodurtha *et al.* 1990). All these studies took place in less obesogenic environments than exist in many societies today. Environmental effects may be more pervasive and influential for relative adiposity in our present obesogenic world.

Changes in the genetic pool cannot explain the rapid increase in obesity in UK in recent years – the change in prevalence has been too rapid – but there seems no doubt that the genetic make-up of some individuals makes them

more at risk of obesity in modern society than others. Recent changes would seem to show no longer is it just those with strong predisposition to obesity who are at risk but also those with presumably less strong predisposition to obesity. Some of these latter also progress to obesity thus creating the prevalence we experience today. However, data from France (Romon *et al.* 2005) show that in a population of children with increasing prevalence of overweight, those on the lowest centiles of fatness only show increases in fatness if they are of low SES. In other words those children with higher SES and little predisposition to obesity seemed less affected by environmental changes – but why? Do we need to focus more on why some children/adults stay slim within our obesogenic society rather than collect yet more data on the obese in the search for specific precipitating factors to explain their condition?

We do not know if the genetic predisposition (or not) to obesity relates to inherited differences in basal metabolic rates (BMR) which do exist (Bogardus *et al.* 1986), appetite control (Cutting *et al.* 1999), utilization of food energy (Griffiths and Payne 1976; Bouchard *et al.* 1990), aspects of activity or a myriad of other possible causes. Probably all these and other factors are relevant to some cases but separation of the genetic and environmental contributions to obesity from amongst these contributors remains difficult. Parents, at least in early childhood years, are usually the main carers and providers of food, exercise and lifestyle. They share genes and environment with their children.

Socioeconomic status

Socioeconomic circumstances may provide more important determinants of obesity in adult life than in childhood. Indeed, studies from Denmark suggest it may be the environment rather than the family's own SES which promotes obesity in the young adults (Lissau-Lund-Sorenson and Sorensen 1992). In UK studies, adult obesity seems to result from a combination of genetic predisposition, childhood environment and later life experiences (Brummer and McCarthy 2001; Viner and Cole 2005). The effects of SES on prevalence vary according to the place of study, the age of the children studied and perhaps the date of the study. In both North America and Norway overweight/obesity prevalences seem higher in children of poor families although the overall prevalence is much lower in Norway than in North America (Phipps *et al.* 2006). In England the socioeconomic effects on overweight seem less obvious in some (Viner and Cole 2005) but not all studies (Stamatakis *et al.* 2005). Socioeconomic differences on prevalence may be more prominent in Scotland (Armstrong *et al.* 2003). Further, Jebb *et al.* (2003) found children in families from the ISC were four times more likely to be overweight than white British children. In this study the prevalence of

obesity was significantly higher in children from social class IV and V than in those from classes I–III and amongst children in Wales and Scotland than those from England. Viner and Cole (2005) found obesity more likely to present in adult life in women with poorer employment and relationships but childhood obesity did not relate to adult socioeconomic circumstances, income, years of schooling, educational attainment, relationships or psychological morbidity.

Energy balance

As we will discuss in later chapters, obesity in those with otherwise normal growth and no recognized underlying condition must arise when energy intakes exceed energy expenditures. How has energy balance changed to account for the dramatic and rapid increase in obesity in childhood in so many countries over the past 20 years? Specific habits may be responsible for obesity in individuals but the worldwide increased prevalence seems to relate to a range of changes in many modern societies which together create an overall obesogenic environment.

Energy intakes

In the UK, data on energy intakes over the past 50 years suggest children eat less in terms of energy (calories) than they did 50 years ago (Gregory *et al.* 1995; Gregory and Lowe 2000) but they are eating very differently (see later Table 9.3). The variety of foods now available, the availability of foods, advertising pressures and increased affluence for many have changed what is eaten in many cases. Societal attitudes to the way we eat have also changed. As an example, eating in the street is much more acceptable than it was in the past, when many children were told it was something that 'one did not do'. Relative affluence, available food, pressure from advertising and what appear to be reduced energy requirements have enabled families not only to feed their children adequately but too well. We discuss these issues further in Chapter 9.

Energy expenditures

If the evidence for children's energy intakes being greater than in the past is not strong and yet children are fatter, the conclusion must be that energy expenditures have decreased. This certainly seems the case in the UK. Children and their parents are more reliant on cars, walk or cycle less, and in many cases indulge in little formal physical activity. The introduction of the National Curriculum in 1990 resulted in schools focusing less on physical education (PE) and games but this situation does seem to be beginning to reverse. People are also more sedentary. Twenty-four-hour coverage and widespread ownership of television, DVDs and computers create the