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Epidemiology of Childhood Obesity

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A Thesis submitted in fulfilment of the Degree of

Doctor of Philosophy

to the

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Under the supervision of

Dr. John J. Reilly

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DEDICATION

I dedicated this thesis to my wife and children whose support and patience was a constant source of fortitude to me during this research work. The work is also dedicated to my late father who was my best support in his life, and would have been proud of me at this moment. The work is also dedicated to my mother and parents-in-law, whom I always am thankful for their support and prayers.

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AUTHOR'S DECLARATION

I declare that the work contained in this thesis is original, and is the work of one author, Ahmad Reza Dorosty Motlagh. The information reported from the other authors has been quoted with their name and source of publication.

All the analysis and data processings were carried out by the author, under the supervision of Dr. John J. Reilly.

SUPERVISOR'S DECLARATION

I certify that the work reported in this thesis has been performed by A.R. Dorosty-Motlagh and that during the period of study he has fulfilled the conditions of the ordinances and regulations governing the Degree of Doctor of Philosophy.

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LIST OF PUBLICATIONS

Some of the work described in this thesis has been presented or published elsewhere:

PAPERS

Reilly, J.J., Dorosty, A.R. & Emmett, P.M. (1999) Prevalence of overweight and obesity in British children: cohort study. *British Medical Journal*. **319,** 1039-1039.

Reilly, J.J. & Dorosty, A.R. (1999) Epidemic of obesity in UK children. *Lancet*. **354,** 1874-1875.

Dorosty, A.R., Emmett, P.M., Cowin, S., Reilly, J.J., and the ALSPAC Study Team. (2000) Factors associated with early adiposity rebound. *Pediatrics*. **105,** 1115-1118.

Reilly, J.J., Dorosty, A.R., Emmett, P.M., and the ALSPAC Study Team. (2000) Identification of the obese child: adequacy of the body mass index for clinical practice and epidemiology. *International Journal of Obesity*. **24,** 1623-1627.

Reilly, J.J., Kelly, A., Ness, P., Dorosty, A.R., Wallace, W.H., Gibson, B.E., Emmett, P.M., and the ALSPAC Study Team. (2001) Premature adiposity rebound in children treated for acute lymphoblastic leukemia. *Journal of Clinical Endocrinology & Metabolism*. **86,** 2775-2778.

ABSTRACTS

Dorosty, A.R., Emmett, P.M., Reilly, J.J., and the ALSPAC Study Team (1999) Correlates of early adiposity rebound: an opportunity for obesity prevention? International Conference Series on Health Promotion. *Conference on Childhood Obesity: Partnerships for Research & Prevention. Atlanta, Georgia.* PP: 82.

Dorosty A.R., Emmett P.M., Rogers, I., & Reilly J.J. (2000) Factors associated with early adiposity rebound. *Proceedings of the Nutrition Society*. **59,** 60A.

Reilly, J.J., Dorosty A.R. & Emmett P.M. (2000) Identification of the obese child: adequacy of simple obesity definitions for clinical practice and epidemiology. *Proceedings of the Nutrition Society*. **59,** 65A.

Dorosty, A.R. & Reilly, J.J. (2000) Prevalence of childhood overweight and obesity in two Iranian provinces. *International Journal of Obesity*. **24,** S156.

Reilly, J.J., Dorosty, A.R., Emmett, P.M., and the ALSPAC Study Team. (2000) Risk factors for obesity in UK children. *International Journal of Obesity*. **24,** S155.

Reilly, J.J., Kelly, A., Ness, P., Dorosty, A.R., Emmett, P.M., Wallace, W.H.B. & Gibson, B.E.S. (2000) Early adiposity rebound in children treated for acute lymphoblastic leukemia (ALL). *Obesity Research*. **8,** 80s.

Dorosty, A.R. & Reilly, J.J. (2000) The effect of using different BMI reference data on obesity prevalence in Iranian children. *Obesity Research*. **8,** 101s.

Summary

In recent years awareness of childhood obesity as a clinical and public health problem has increased. However, a number of important issues related to childhood obesity were unclear when this thesis began. The aims of this thesis were as follows:

1. To estimate the prevalence of obesity in British and Iranian children.
2. To assess the strengths and weaknesses of the body mass index (BMI) as a way of identifying obese children / estimating obesity prevalence.
3. To investigate the factors associated with early 'adiposity rebound'.
4. To identify risk factors for obesity in British children.

Prevalence of childhood overweight / obesity in the UK

This thesis (Chapter 3) demonstrated that an epidemic of childhood and adolescent obesity occurred in Britain during the 1990s. In the Health Survey for England 1996 (HSE 1996), prevalence of obesity (BMI $\geq 95^{th}$ centile relative to UK 1990 reference data; Cole et al 1995) was 11.2% at ages 6-9 years, and 13.7% at ages 10-15 years respectively. These estimates significantly exceeded the 'expected' frequencies of 5% based on UK 1990 reference data. A study of obesity prevalence in pre-school children in the Avon Longitudinal Study of Parents and Children (ALSPAC) showed that prevalence significantly increased compared to expected from about age 2.5 years.

Prevalence of childhood overweight / obesity in Iran

This thesis (Chapter 4) estimated prevalence of obesity (BMI $\geq 95^{th}$ centile relative to Iranian 1992 reference data; Hosseini et al 1999) in young Iranian children (aged 2-5 years) in 2 provinces in 1995. Prevalence of obesity was significantly higher than expected from age 3. Obesity prevalence differed based on choice of BMI reference data. Choice of BMI reference data can therefore have a great impact on epidemiological assessment of childhood obesity although the absolute BMI values equivalent to BMI $\geq 85^{th}$ and $\geq 95^{th}$ percentiles in different reference data sets are very similar

Defining obesity in children

The US expert committee recommended that children with BMI ≥95th percentile be defined as obese (Barlow & Dietz 1998). Formal guidelines do not exist in the UK, but BMI charts (Child Growth Foundation, London) recommend a definition of BMI ≥98th centile. In this thesis (Chapter 6) the ability of different BMI centiles, and different definitions of obesity based on BMI, to screen for the fattest children was tested in 4175 7 year olds. A true definition of obesity was defined as being in the top 5% of the distribution of body fat percentage. Sensitivity (percentage of obese individuals who are classified as obese) of BMI cut-offs using UK 1990 reference data (Cole et al 1995) was 98% to 94% for percentiles 74 to 85, 94% to 88% for centiles 86 to 95, and 87% to 57% for centiles 96 to 99. Specificity (the percentage of non-obese individuals who are classified as non-obese) of these were 74% to 85%; 86% to 94%; 95% to 99% respectively.

Factors associated with timing of adiposity rebound

The adiposity rebound (AR) refers to the age when BMI starts to increase again after its nadir. An early AR is believed to be a risk factor for later obesity (Dietz 1997b). A knowledge of the timing of AR in modern British children was lacking when this thesis started. In children (n=889; 486 boys, 403 girls) participating in the ALSPAC study, 3% had experienced AR by age 37 months (very early rebounders), about 7% by age 43 months (early rebounders), and around 27% by age 49 months (Chapter 5). Possible relationships between a number of childhood variables (i.e. childhood BMI, macronutrient intakes at ages 8, 18, and 43 months) and parental variables (including parental BMI, parental education, social class) and timing of AR were tested in Chapter 5. The results indicated that dietary energy and macronutrient intakes were not associated with timing of the AR, and there was no evidence that higher protein intake was associated with early AR in contrast to earlier evidence. Parental BMI and parental obesity were associated with early rebound.

Risk factors for obesity in UK children

A total of 19 potential risk factors, all supported by prior evidence or hypotheses, were tested for their association with childhood obesity in (n=7766; 3949 boys and 3817 girls) children participating in ALSPAC (Chapter 7). From these 19 risk factors, 4 (gestational age, gender, ethnicity, and season of birth) showed no association with childhood obesity. The other 15 variables showed a significant association with childhood obesity including: catch-up growth; upper quartile of birth weight (>3800 grams); no breast-feeding or stopping before 6 months; the introduction of solids before 12 weeks; energy intake at ages 8, 18, and 43 months, and macronutrient intake at 43 months; maternal & paternal education; maternal & paternal obesity; maternal & paternal social class; time spent watching television; time spent sitting in car; fizzy drink consumption; irregular sleeping / short sleep duration. After adjusting for maternal social class, the age of the introduction of solids was not significantly associated with childhood obesity. After adjusting each risk factor for all others (multi-variable analysis), five remained significantly associated with childhood obesity: maternal and paternal obesity; birth weight; consumption of fizzy drinks; time spent in the car.

Conclusions

This thesis showed that prevalence of childhood obesity in British and Iranian children were significantly higher than expected and that obesity prevalence in children increased during the 1990s. These results are consistent with reports of increased childhood obesity in the USA, Europe, and some other countries. Using BMI $\geq 95^{th}$ centile as the definition of childhood obesity has moderately high sensitivity and high specificity, though a definition of the BMI $\geq 92^{nd}$ centile was shown in this thesis to be optimum. This thesis indicated that the typical age of AR in British children must be sometime between 5-7 years. Further research on the factors associated with timing of AR is recommended. A number of independent risk factors for childhood obesity were identified. Parental obesity, birth weight, fizzy drink consumption, and time spent in the car had the strongest association.

1. Chapter 1: Introduction

1.1. The obesity epidemic worldwide

Many studies worldwide have demonstrated increases in adult obesity (e.g. Kuczmarski 1992; Kuczmarski et al 1994; Al-Isa 1995; Rosenbaum & Leibel 1997; Whitaker et al 1997; WHO 1997; Popkin & Doak 1998; Jebb 1999; NAO 2001). In addition, recent studies have documented rapid increases in overweight / obesity among children and adolescents (Troiano et al 1995; Chapters 3-7). There is also some evidence of increasing child, adolescent, and adult obesity in developing countries (de Onis & Habicht 1996; Gill 1999; de Onis & Blossner 2000; Martorell et al 2000). According to the report of the UK National Audit Office (NAO 2001) about 20% of English adults were obese (BMI >30) in 1998 and two thirds of men and over half of women were overweight (BMI >25). Obesity prevalence in UK adults has trebled over the last 20 years (NAO 2001). Studies in the USA have shown that prevalence of obesity in children and adults is increasing dramatically and these trends are likely to continue (Kuczmarski 1993; Troiano et al 1995; Ogden et al 1997; Flegal et al 1998; Martorell et al 1998; Troiano & Flegal 1998; Troiano & Flegal 1999; Zephier et al 1999). According to the large population-based Canadian Heart Health Surveys in 1992, about half of adult Canadians were overweight and about 17% were obese (Macdonald et al 1997). Obesity is now a major public health problem in both developed and developing countries (Jebb 1999). A brief list of studies on obesity prevalence worldwide is presented in Chapter 4.

The National Audit Office (NAO 2001) has listed the 4 most common problems linked to obesity in the English adult population as follows: heart disease; type 2 diabetes; high blood pressure; osteoarthritis. The proposed reasons for increasing obesity prevalence are divided into three categories: genetics; macronutrient intake; low energy expenditure (Gortmaker et

al 1996; Maffeis et al 1997; Dietz 1997b; Andersen et al 1998; Bar-Or et al 1998; Dietz 1998b; Franzese et al 1998; Maffeis et al 1998; Roberts & Leibel 1998; Whitaker & Dietz 1998; Goran & Weinsier 2000). Although studies have shown that genetic factors play a role in childhood obesity, the relative importance of inherited factors in obesity development is not clear (Klesges et al 1995; Sorensen 1995; Perusse & Bouchard 1999). However, since the obesity epidemic in the developed world has occurred quickly, rapid changes in population genetic 'background' are unlikely to be responsible for the epidemic. The importance of genetics, energy intake, and energy expenditure are discussed in brief in Chapter 3.

Changes in lifestyle have probably played a major role in the increasing prevalence of obesity described above. Nowadays, children and adolescents spend a substantial part of their lives in front of a television set. Extrapolation of current viewing data (Nielsen Media Research 1997) showed that, between the ages of 2 and 17 years, US children spend an average of more than 3 years of their waking lives watching television. This, of course, is excluding time spent watching videos, playing video games, or using a computer (Robinson 1998). Boys and girls with higher BMI and trunk skinfold values are likely to report watching more television than other children (Dietz & Gortmaker 1985; Pate & Ross 1987; Shannon et al 1991; Locard et al 1992; Obarzanek et al 1994; Gortmaker et al 1996; Andersen et al 1998). Some intervention studies have shown that obesity prevalence and/or BMI was reduced by training school children to spend less time watching TV, videos, and computer games (Gortmaker et al 1996; Gortmaker et al 1999; Robinson 1999). The amount of time spent watching television in British children has doubled since the 1960s (Pullinger 1998), while the amount of time spent on physical activities in children has reduced and this reduction was more marked in older children, especially older girls (Chapter 3). On the other side of the energy balance equation, there is evidence that US adolescents who watch more

television may eat higher-fat diets (Robinson & Killen 1995) and some evidence that high-energy food advertisements might influence children's snack choices and consumption (Gorn & Goldberg 1982; Jeffrey et al 1982). However, it has been shown that the average amount of energy consumed by adults and children in the UK has declined during recent years (Gregory et al 1995; Prentice & Jebb 1995; NDNS 2000; Chapters 3 and 7).

Unfortunately, there are only a few effective tools to tackle the adulthood obesity problem (Troiano et al 1995). Most available weight-control treatments only lead to moderate changes and are unsatisfactory (Robinson 1998). Some prevention programs for adults targeting diet and activity changes have not been effective (Resnicow & Robinson 1997). As a result, there is a need for innovative intervention approaches (Robinson 1998).

1.2. Critical periods in the development of childhood obesity

Many observations suggest that three critical periods exist for the development of obesity and its complications (Dietz 1994; Dietz 1997b). A critical period refers to a developmental stage, when physiological changes can increase the risk of later obesity (Dietz 1997b). These may be categorised as: (a) fetal and postnatal periods, including gestation and early infancy; (b) the period of adiposity rebound (when BMI begins to increase again after a decline in early childhood); (c) and the period of adolescence (Dietz 1997b). Obesity that begins at these periods increases the risk of persistent obesity and its complications, though the relative contribution of each of these critical periods to the prevalence, morbidity and mortality of adult obesity is not clear at the moment (Dietz 1997a). The existence of critical periods might lead to focus on preventive efforts at these developmental stages (Dietz 1994).

1.3. Adult obesity: consequences for individuals and populations

Obesity is characterised by excessive body fat accumulation in adipose tissue to an extent that health may be adversely affected (Lau 1999). Obesity is one of the most important avoidable risk factors for a number of life threatening diseases and for serious morbidity (Garrow 1992). Table 1.1 presents a list of the main medical and psychosocial problems related to obesity in adulthood.

Table 1.1. Disorders caused by obesity in adults (from Anonymous 1997; Dietz 1997a; Freedman et al 1997; Dietz 1998a; Freedman et al 1999).

Medical disorders	Psychosocial and social outcomes
Diabetes mellitus (type 2)	Less education
Insulin resistance (hypertriglyceridaemia or low HDL-cholesterol)	Higher poverty rates
Hyperlipidemia or Dyslipidemia (high serum LDL-cholesterol and triglycerides and low HDL-cholesterol levels)	Lower marriage rates
High levels of plasminogen activator inhibitor	Lower income
Respiratory disease	Fewer friends
Eating disorders	Isolation
Hepatic steatosis (high concentrations of liver enzymes associated with fatty liver, fatty hepatitis, fatty fibrosis, or cirrhosis)	Lower occupational attainment
Cholelithiasis (Increased cholesterol synthesis and cholesterol saturation of bile)	Lower self-esteem
Arthritis	
Low-back pain	
Hyperviscosity	
Coronary heart disease	
Hypertension	
Pseudotumor cerebri (increased intracranial pressure)	
Some cancers (e.g. postmenopausal breast cancer, endometrial cancer, colon cancer, prostate cancer)	
Sleep apnoea	
Orthopaedic complications	
Polycystic ovary disease	
Menstrual abnormalities	
Reproductive-hormone abnormalities	
Increased anaesthetic risk	
Hyperuricemia and gout	
Gall bladder disease	

In addition to the disease burden of obesity on obese individuals and their families, there are important human and financial costs of obesity. According to the NAO (2001) report, obesity costs 18 million sick days a year in England. It also causes about 30,000 deaths a year, which shortens life by 9 years on average and results in 40,000 lost years of working life (NAO 2001). It is estimated that the NHS in England spends about £0.5 billion a year for treatment of obesity and related problems. Obesity has been estimated to have a possible £2 billion a year impact on the English economy (NAO 2001).

The financial burden on society of illness and premature death may be measured in terms of direct and indirect costs (Krauss & Winston 1998). Direct costs represent the value of resources (personal health care, other professional services, and drugs) that could be used by other uses in the absence of obesity. Indirect costs are the value of lost output because of the prevention or reduction of productive activity due to the morbidity or mortality increased (Krauss & Winston 1998). The economic costs of obesity are high, ranging from 2-10% of the total health care budgets of different countries (Colditz 1992; Segal et al 1994; Seidell & Deerenberg 1994; Wolf & Colditz 1994; Levy et al 1995; Seidell 1995; Swinburn et al 1997; Kurscheid & Lauterbach 1998).

1.4. Childhood obesity: consequences for individuals and populations

Childhood obesity has a number of adverse consequences for the child (short-term effects), and for later life (longer-term effects; Table 1.2).

1.4.1. Short term effects

1.4.1-a. Psychological

The most widespread effects of childhood obesity are psychological consequences including anxiety (Dietz 1998a). The social burden of paediatric obesity, especially during middle childhood and adolescence, may have lasting effects on self-esteem, body image and economic mobility (Must & Strauss 1999).

1.4.1-b. Other medical problems in childhood

A number of medical problems are associated with childhood and adolescent obesity. These include pseudotumor cerebri (increased intracranial pressure which causes headaches and severe visual impairment or blindness) and headaches, sleep apnoea and hypoventilation syndrome, polycystic ovary disease, gall bladder disease, non-alcoholic steatohepatitis (described later), glucose intolerance, type II (non-insulin-dependent) diabetes mellitus, early atherosclerotic lesions (Must et al 1992; McGill et al 1995; Dwyer & Blizzard 1996; Pinhas-Hamiel et al 1996; Goran 1997; Dietz 1998a; Dietz & Robinson 1998; Hulman et al 1998; Krauss & Winston 1998; Pietrobelli et al 1998; Vanhala et al 1998; Morrison et al 1999a,b; Fredriks et al 2000; Lynch et al 2000; Sokol 2000). Associations between high BMI or increases in BMI and increased blood pressure, adverse lipoprotein profiles, non-insulin-dependent diabetes mellitus, and early atherosclerotic lesions were shown in earlier studies too (Killeen et al 1978; Laskarzewski et al 1980; Lauer & Clark 1989; Gutin et al 1990; Zwiauer et al 1990; Sangi & Mueller 1991).

Sleep apnoea is one of the consequences of childhood obesity, and neurocognitive deficits are common among obese children with sleep apnoea (Dietz 1997a). Non-alcoholic steatohepatitis is an important differential diagnosis for asymptomatic patients with

chronically elevated plasma liver enzyme levels which can happen in obese people in early adulthood (Sheth & Gordon 1997; Sokol 2000).

Childhood obesity increases sensitivity to endotoxin liver injury (Yang et al 1997). High concentrations of liver enzymes (which are frequently associated with fatty liver, fatty hepatitis, fatty fibrosis, or cirrhosis) represent a frequent obesity-associated finding in children and adolescents (Dietz 1997a). Up to 50% of obese adolescents who drink alcohol have abnormal liver enzyme levels (Strauss 2000). Almost 90% of morbidly obese patients have some degree of morphologic liver abnormality, including fatty change and inflammation (Khaodhiar et al 1999).

1.4.2. Longer term effects

1.4.2-a. Persistence of obesity

Obese children have a relatively high probability of remaining obese, i.e. becoming obese adults (Serdula et al 1993; Whitaker et al 1997; Dietz 1998a; Must & Strauss 1999). Approximately 50% of US obese adolescents (BMI $\geq 95^{th}$ centile) become obese adults (Dietz 1998a). Using other BMI cut-offs shows that 15-80% of overweight children might become obese adults and the probability increases with the age (Whitaker et al 1997; Dietz & Robinson 1998; Fredriks et al 2000). Obesity in adulthood, if established in childhood, may be more harmful than obesity that has appeared in adulthood since continuous obesity from childhood might be a 'generator' for prolonged insulin resistance, which causes hypertension and metabolic abnormalities in later life (Vanhala et al 1998).

1.4.2-b. Cardiovascular risk factors

Berenson et al (1998), using data from the Bogalusa heart study, provided evidence that even in childhood and early adulthood, traditional risk factors for coronary disease (like

obesity) are related to atherosclerosis in the aorta and coronary arteries. The obese as well as cigarette smoking adolescents, and those who are less active, are at higher risk of coronary diseases (Troiano et al 1995; Johnson et al 1996; Gaziano 1998; Freedman et al 1999a,b; Morrison et al 1999a,b). The risk of diabetes, cardiovascular diseases, and all other causes of mortality and morbidity (like colon cancer) are elevated among those who were overweight during childhood (Dietz 1998a; Must & Strauss 1999). Apolipoprotein B (Apo B) is the carrier protein for the atherogenic low density lipoprotein (LDL) cholesterol, and circulating concentrations reflect LDL-cholesterol concentrations and cardiovascular risk; it has been shown that levels of Apo B are significantly increased in overweight children (Krauss & Winston 1998). Atherosclerosis is a complex chronic disease of the arteries, which may be initiated early in life (Ross 1993). BMI as a traditional coronary risk factor tends to be associated with the extent of lesions in both the aorta and the coronary arteries (Gaziano 1998).

Central obesity in children may be a more important risk factor for adverse cardiovascular outcomes than peripheral obesity (Goran et al 1995; Daniels et al 1997; Goran & Gower 1999). Both obesity (high BMI) and central fat distribution increase the likelihood of development of cardiovascular disease risk factors in children (Freedman et al 1998; Daniels et al 1999; Freedman et al 1999; Morrison et al 1999a,b). Central obesity is associated with increased insulin resistance, higher circulating insulin levels, elevation of blood pressure, and decreased HDL-cholesterol (HDL-C; Freedman et al 1987; Slyper 1998). Greater deposition of central fat is also associated with less favourable plasma lipid and lipoprotein concentrations, blood pressure, and left ventricular mass (Daniels et al 1999). It was shown that childhood overweight has an association with increased cardiovascular disease (CVD) risk factors (e.g. HDL-C, triglycerides, the ratio of LDL-cholesterol (LDL-C) to HDL-C, systolic & diastolic blood pressure) clustering. On the other hand, increase in central adiposity enhances the CVD risk factor clustering independent of overweight (Morrison et

al 1999a,b). This means that the possibility of occurrence of multiple risk factors is greater than predicted in overweight boys and girls particularly in those who have a higher central adiposity (Morrison et al 1999a,b). For instance, Morrison et al (1999a) reported that about 45% of overweight (BMI >85th centile) but less than 10% of non-overweight 10-15 year old boys, had 2 CVD risk factors. Of the overweight boys, 30% of those with central adiposity (measured by 3 skinfolds) <50th centile had 2 CVD risk factors while, more than 56% of those with central adiposity ≥50th centile, had 2 CVD risk factors. Since adiposity and cardiovascular risk factors track from childhood into adulthood, early identification of children with high central adiposity is of importance (Taylor et al 2000)

1.4.2-c. Other long term medical problems

These problems include orthopaedic, neurological, pulmonary, gastroenterological, and endocrine conditions (Must & Strauss 1999). High blood pressure, insulin resistance, hyperinsulinaemia, hypertriglyceridaemia, colorectal cancer, atherosclerotic cerebrovascular disease, coronary diseases, lower HDL-cholesterol and higher LDL-cholesterol are some of the other medical problems of childhood obesity which have effects in the long term (Power et al 1997; Dietz 1997a; Charney 1998; Dietz 1998a; Dietz & Robinson 1998; Rosner et al 1998; Vanhala et al 1998; Sokol 2000).

1.4.2-d. Social and economic disadvantages

Two studies, one in the USA (Gortmaker et al 1993), and one in the UK (Sargent & Blanchflower 1994) have shown that obesity in adolescence / young adulthood is associated with poorer social outcomes in later life (higher rates of poverty and lower rates of marriage, educational attainment, and household income corrected for I.Q.), particularly in women (Dietz 1998a).

Table 1.2. Disorders / problems associated with obesity in children (from Dietz 1997a; Dietz 1998a; WHO 1998; Freedman et al 1999; Khaodhiar et al 1999)

Medical disorders	Psychosocial problems
Faster growth	Anxiety
Early maturation	Affected body image
Lowered exercise tolerance	Affected economic mobility
Obesity in adulthood	Less education
Respiratory diseases, e.g. sleep apnoea	Fewer friends
Eating disorders	Mistaken as older
Abnormal glucose metabolism	Isolation
Glucose intolerance	Lower self-esteem
Insulin resistance	Higher rates of poverty
Hyperinsulinaemia	Lower rates of marriage
Type II diabetes mellitus	
Orthopaedic complications	
Neurological problems	
Pulmonary problems	
Gastroenterological complications	
Endocrine disorders	
Hypertriglyceridaemia	
Lower HDL, and higher LDL cholesterol	
Improper liver function including:	
Non-alcoholic steatohepatitis	
Fatty liver, fatty hepatitis, fatty fibrosis	
Cirrhosis	
Abnormal liver enzyme levels	
Liver inflammation	
Elevated blood pressure	
Cardiovascular risk factors including:	
Coronary diseases	
Atherosclerotic cerebrovascular disease	
Early atherosclerotic lesions	
Pseudotumor cerebri and headaches	
Gall bladder disease	
Apoliprotein B increased	
Colorectal cancer	
Polycystic ovary disease	

1.5. Childhood obesity: statements of concern

A recent study, presented later in this thesis, on English children showed that the frequency of overweight (BMI ≥85th centile) ranged from 22% at age 6 years to 31% at age 15 years and that of obesity (BMI ≥95th centile) ranged from 10% at age 6 years to 17% at age 15 years (Reilly & Dorosty 1999; Chapter 3). Almost one-quarter of US children are now obese (BMI ≥95th centile), a dramatic increase of over 20% in the past decade (Bar-Or et al 1998; Bray 1998; Troiano & Flegal 1998; Chapter 3). Childhood obesity is rapidly emerging as a global epidemic which will have profound public health consequences since, as noted earlier, many overweight children will become overweight adults (Bellizzi & Dietz 1999). Behavioural and lifestyle patterns are to some extent learned in childhood, and tend to continue into adulthood and, therefore, it could be assumed that childhood obesity risk factors are likely to lead to cardiovascular problems in later adulthood (Gaziano 1998). On the other hand, since changing lifestyle is difficult to achieve in adulthood, it seems reasonable to attempt to alter these risk factors early in life (Gaziano 1998).

A number of editorials in recent years have expressed concern over increasing prevalence of childhood obesity. These are listed in Table 1.3 with major editorials from 1998, the year the present study began. These editorials generally highlighted the important clinical and public health consequences of childhood obesity, discussed above. Many of the editorials also called for more research, and for urgent action to prevent obesity in children.

Since 1998 (when work for the present thesis began), many major reviews on childhood obesity have also been published, of which some are listed in Table 1.4.

Table 1.3. Editorials / commentaries in major journals on the childhood obesity epidemic since 1998.

Author(s) year	Journal	Title
Bray 1998	Lancet	Obesity: a time bomb to be defused.
Dietz & Robinson 1998	Journal of Pediatrics	Use of the body mass index (BMI) as a measure of overweight in children and adolescents.
Dietz 1998	American Journal of Clinical Nutrition	Does energy expenditure affect changes in body fat in children?
Gaziano 1998	New England Journal of Medicine	When should heart disease prevention begin?
Prentice 1998	British Medical Journal	Body mass index standards for children. Are useful for clinicians but not yet for epidemiologists.
Robinson 1998	Journal of the American Medical Association	Does television cause childhood obesity?
Dietz & Davis 1999	British Medical Journal	Lessons from a cyclist. Doctors should do more to promote physical activity.
Dietz & Nelson 1999	Journal of Pediatrics	Barriers to the treatment of childhood obesity: a call to action.
Koplan & Dietz 1999	Journal of the American Medical Association	Caloric imbalance and public health policy.
Lau 1999	Canadian Medical Association Journal	Call for action: preventing and managing the expansive and expensive obesity epidemic.
Dietz 2000	American Journal of Clinical Nutrition	Birth weight, socioeconomic class, and adult adiposity among African Americans.
Dietz 2000	Lancet	'Adiposity rebound': reality or epiphenomenon?
Fruhbeck 2000	British Medical Journal	Childhood obesity: time for action, not complacency.
Goran & Weinsier 2000	Obesity Research	Role of environmental vs. metabolic factors in the etiology of obesity: time to focus on the environment.
Sokol 2000	Pediatrics	The chronic disease of childhood obesity: the sleeping giant has awakened.
Anonymous 2001	Lancet	Childhood obesity: an emerging public-health problem.
Dietz 2001a	Journal of American Medical Association	Breast-feeding may help prevent childhood overweight.
Dietz 2001b	British Medical Journal	The obesity epidemic in young children: reduce television viewing and promote playing.

Table 1.4. Major reviews on the childhood obesity epidemic, published since 1998.

Author(s) year	Journal	Title
DeLany 1998	American Journal of Clinical Nutrition	Role of energy expenditure in the development of pediatric obesity.
Dietz 1998	Journal of Nutrition	Childhood weight affects adult morbidity and mortality.
Goran & Gower 1998	Coronary Artery Disease	Abdominal obesity and cardiovascular risk in children.
Goran & Sun 1998	American Journal of Clinical Nutrition	Total energy expenditure and physical activity in prepubertal children: recent advances based on the application of the doubly labeled water method.
Slyper 1998	Pediatrics	Childhood obesity, adipose tissue distribution, and the pediatric practitioner.
Troiano & Flegal 1998	International Journal of Obesity	Overweight prevalence among youth in the United States: why so many different numbers?
Vanltallie 1998	Nutrition Reviews	Predicting obesity in children.
Whitaker & Dietz 1998	Journal of Pediatrics	Role of the parental environment in the development of obesity.
Bellizzi & Dietz 1999	American Journal of Clinical Nutrition	Workshop on childhood obesity: summary of the discussion.
Dietz & Bellizzi 1999	American Journal of Clinical Nutrition	Introduction: the use of body mass index to assess obesity in children.
Goran & Gower 1999	American Journal of Clinical Nutrition	Relation between visceral fat and disease risk in children and adolescents.
Goran et al 1999	International Journal of Obesity	Role of physical activity in the prevention of obesity in children.
Must & Strauss 1999	International Journal of Obesity	Risks and consequences of childhood and adolescent obesity.
Jebb & Lambert 2000	Journal of Pediatrics	Overweight and obesity in European children and adolescents.
Sorensen 2000	Diabetes Care	The changing lifestyle in the world. Body weight and what else?
Dietz & Gortmaker 2001	Annual Review of Public Health	Preventing obesity in children and adolescents.
Goran 2001	American Journal of Clinical Nutrition	Metabolic precursors and effects of obesity in children: a decade of progress, 1990-1999.

In addition, in recent years (from 1998), several international journals published one or more whole supplements about childhood / adolescent obesity (Table 1.5), and a number of respected government and non-government organisations have called for urgent action on the childhood obesity epidemic. These are listed in Table 1.6.

Table 1.5. Childhood obesity supplements in major journals since 1998.

Journal	Title / Content
Pediatrics, March 1998 supplement.	The causes and health consequences of obesity in children and adolescents (7 papers).
American Journal of Clinical Nutrition, April 1999 supplement.	Obesity prevention in American Indian school children: Pathways (13 papers).
International Journal of Obesity, March 1999 supplement	Childhood / adolescent obesity (9 papers).
American Journal of Clinical Nutrition, July 1999 supplement.	Proceedings of an International Obesity Task Force: Workshop on childhood obesity, Dublin, Ireland (11 papers).
European Journal of Pediatrics, July 2000 supplement.	Childhood / adolescent obesity (8 papers).

In summary, the problem of childhood obesity has increased in profile in recent years, even since the beginning of this thesis in 1998, with at least 18 editorials / commentaries, 17 reviews, 5 journal supplements, and 8 national or international statements of concern.

Table 1.6. US position statements / governmental statements of concern since 1998.

Committee / group / organisation	Conclusions	Reference
The Maternal and Child Health Bureau, Health Resources and Services Administration, the Department of Health and Human Services convened a committee of paediatric obesity experts to develop the recommendations.	The Committee recommended that children with a BMI ≥85th percentile with complications of obesity or with a BMI ≥95th percentile, with or without complications, undergo evaluation and possible treatment.	Barlow & Dietz 1998
The US Department of Health and Human Services, the Centers for Disease Control and Prevention (CDC), and the Department of Education.	They have drafted a report outlining a comprehensive plan to promote physical activity / sports and decrease obesity in the nation's young people	Josefson 2000 www.cdc.gov /nccdphp/dash
US Department of Health and Human Services, Office of Disease Prevention and Health Promotion prepared a conference on Healthy People 2010. http://web.health.gov/healthypeople	In Healthy People 2010 proposed objectives were: to increase the prevalence of healthy weight and decrease the prevalence of adult obesity (BMI >30) and child obesity (BMI ≥95th centile of the new BMI-for-age CDC growth charts).; To prevent young children from ever becoming overweight (as an essential public health goal).	Kuczmarski &Flegal 2000 Whitaker et al 2000 Stockmyer et al 2001
US Department of Health and Human Services and the US Department of Agriculture held the National Nutrition Summit in Washington, DC.	The Division of Nutrition and Physical Activity, National Center for Chronic Disease Prevention and Health Promotion, and Centers for Disease Control and Prevention set the agenda for seven obesity-related discussion groups of the National Nutrition Summit.	Stockmyer et al 2001

Table 1.7. Other position statements / governmental statements of concern since 1998.

Committee / group / organisation	Conclusions	Reference
Report of WHO consultation (International Obesity Task Force or IOTF) on obesity; Geneva, 3-5 June 1998.	WHO IOTF group worked with the European Childhood Obesity Group to monitor ways to prevent and manage the obesity epidemic. They proposed to determine the prevalence of obesity worldwide and to analyze secular trends internationally.	Prentice 1998 Guillaume 1999
Workshop on childhood obesity in Dublin to establish an index with which to assess adiposity (overweight) in children and adolescents worldwide.	BMI is not a perfect obesity measure in children. However, BMI is appropriate as a consistent and pragmatic definition for overweight in children and adolescents, though other alternatives may be considered in the future.	Bellizzi & Dietz 1999
WHO convened a Consultation on obesity to review current epidemiological information, contributing factors and associated consequences, and report the conclusions and recommendations.	The system for classifying obesity and overweight should be based on BMI. The fundamental causes of the obesity epidemic are sedentary lifestyles and high-fat energy-dense diets. While there is strong evidence that certain genes have an influence on body mass and body fat, it is likely to be many years before the results of genetic research can be applied to the problem. Methods for the treatment of obesity described, including dietary management, physical activity and exercise, and antiobesity drugs, with gastrointestinal surgery being reserved for extreme cases.	WHO Technical Report Series, 2000.
National Audit Office 2001, British House of Commons	Tackling obesity in England: the prevalence and costs of obesity in England, management of obesity in NHS, and initiatives across Government to address the problem of obesity.	NAO 2001

2. Chapter 2: Methods

2.1. ALSPAC

2.1.1. Introduction to ALSPAC and ELSPAC

The Avon Longitudinal Study of Parents and Children (ALSPAC), previously known as Avon Longitudinal Study of Pregnancy and Childhood, forms part of the European Longitudinal Study of Pregnancy and Childhood (ELSPAC) prospective cohort study (Golding 1989; Golding et al 2001). The aims of the ELSPAC study are to identify factors influencing the development of various diseases and disorders in children, to identify ways in which to prevent disorders occurring, and to maximise the health and potential development of the child. The centres currently participating in ELSPAC include Yarsoslavl in Russia, Brno and Znojmo in the Czech Republic, Bratislava in Slovakia, five centres in the Ukraine, the Isle of Man, and Avon in the UK. All of the ELSPAC study centres had to incorporate a set of study rules, including:

1. The study should cover all pregnant mothers resident in a defined geographical area with an expected date of delivery between identified dates.
2. Data collection should be by self-completion questionnaires sent to the mother and her partner – this information can then be linked to information from health records.
3. All study centres should ask the same questions to obtain a core data set. Questions should be asked in the same way as far as possible, except for those questions when appropriate culture-specific alternatives are necessary (e.g. questions about diet and educational level).
4. As much detail as desired could be added to the overall study strategy, but none of the core data set should be omitted.

For the arm of ELSPAC known as ALSPAC, all pregnant women resident within a defined part of the county of Avon in South West England with an expected date of delivery between April 1991 and December 1992 inclusive were eligible for inclusion. A variety of tactics were employed in order to persuade mothers to enrol in the study. These included displaying posters in a number of places including chemist shops, general practitioner waiting rooms, antenatal clinics, inviting mothers to send for further details of the study. Community midwives were also asked to enrol all mothers booking through them for antenatal care. In addition, all mothers booking at the three major hospitals in the study area were sent information about the study with their booking information. This resulted in 14,893 women enrolling in the study – between 80 to 90% of eligible pregnancies in the geographically defined area. The children of these mothers were called 'Children of the 90s' and provided a 'reasonably representative' sample of British children (Golding et al 2001).

2.1.2. Questionnaire and medical record data

The first questionnaire in ALSPAC 'Having a Baby' was sent to the mother at 18 weeks gestation. Questionnaires for the mother have since been sent at regular intervals. A number of questionnaires for her partner were also sent to the mother, which she passed on if she wished. Partner's questionnaires were received from about 60% of study families, though further details on partner's participation are given in later Chapters, where appropriate. Most questions in the later questionnaires were focused on the child, and were designed to gather information on health, development and behaviour, as well as on features of the child's environment that may affect this, such as diet and types of childcare. Other questions focused on the mother, e.g. questions about her state of mind and current lifestyle and employment status.

Some information about the mothers was obtained from medical records, unless they stated that they did not want this to happen. However, in many cases and for most variables, medical information was only available by maternal self-report. This is described where appropriate later in this thesis.

2.1.3. Children in Focus and Children of the 90s

As explained earlier, the total sample children of the ALSPAC study were called 'Children of the 90s'. A proportion of the children born in the last 6 months of the study (equivalent to 10% of the whole Children of the 90s cohort) were selected at random to take part in a sub-study known as Children in Focus (CIF). The aims of Children in Focus were to obtain information that could not be determined by questionnaire, and also to validate some aspects of the self-completion questionnaires. These children attended research clinics every four months for the first year of life, and six-monthly thereafter. A variety of physical, growth, and developmental measures were taken at the 4, 8, 12, 18, 25, 31, 37, 43, 61, and 90 month clinics. Part of the data described in this thesis was derived primarily from the CIF clinics.

Mothers of 1509 CIF children were invited to the 4 month clinic, of whom 1023 attended (16 had been willing to attend but unable). At 8 months, all those who had been willing to attend the 4-month clinic were invited to attend, along with an additional 550 cases to increase the size of the cohort. This made a total number of 1589 invited to the Children in Focus study, of whom 1314 attended at age 8 months.

2.1.4. Ethics committee approval

Ethical approval for the study was obtained from the ALSPAC Ethics and Law Sub-Committee, and from the three Medical Research Ethics Committees in the Avon study area.

2.1.5. Representative nature of the sample

2.1.5-a. Children of the 90s: demographics

The national UK census data from 1991 were used (by the ALSPAC team) to compare the population of mothers in Avon with infants under 1 year of age in 1991/92 (Children of the 90s) with those in the whole of Britain (ALSPAC homepage; Golding et al 2001). This revealed that mothers in Avon were statistically less likely to be non-white (4.1% vs. 7.6%), marginally more likely to be in owner-occupied accommodation and to have a car available to them, and less likely to have more than one person per room ($p<0.01$; ALSPAC homepage). In comparison to the whole eligible population in Avon, Table 2.1 shows that mothers in the ALSPAC population completing questionnaires 8 months post-delivery were significantly more likely to live in owner-occupied accommodation, more likely to have ≥1 person per room, more likely to have access to a car (90.8% vs. 83.7%), more likely to be married, and less likely to be from a non-white ethnic group ($p<0.01$; Table 2.1; ALSPAC homepage).

Table 2.1. Population information of mothers from ALSPAC study compared to Avon and the UK.

	Children of the 90s (1991/92)	**Avon (1991)***	**Whole of UK (1991)***
Owner occupier	79.1%	68.7%	63.4%
≥1 person / room	33.5%	26.0%	30.8%
Car in household	90.8%	83.7%	75.6%
Married couple	79.4%	71.7%	71.8%
Non-white mother	2.2%	4.1%	7.6%

* Based on UK 1991 Census

2.1.5-b. Children of the 90s: social class composition

Further investigation of differences between ALSPAC participants and the UK in social class composition was made by the author of this thesis. As Table 2.2 shows, the percentage of children who belonged to (paternal) social classes I & II was significantly higher, and the percentage of children who belonged to social classes IV & V was significantly lower, than both the Avon area and the UK in the same period of time. This was based on a comparison with the social class distribution of Avon and the UK as described by the Census (Census 1991), and the National Diet and Nutritional Survey (NDNS 1995).

Table 2.2. Comparison of paternal social class of the 'Children of the 90s', Avon (Census 1991), and the UK (NDNS 1995).

Paternal social class	**UK; 1992 (n=1785)**	**Avon; 1991 (n=63399)**	**Children of the 90s (1991/92; n=10602)**
I, II	33.3%	* 41.4%	*‡ 44.2%
III-N	12.5%	13.3%	‡ 11.5%
III-M	33.3%	* 28.7%	‡ 32.3%
IV, V	20.8%	* 16.5%	*‡ 12.0%

* Significantly different from UK (NDNS 1995), $p<0.01$.

‡ Significantly different from Avon (Census 1991), $p<0.01$.

2.1.5-c. Children in Focus: demographics

The mothers of the 18 month CIF cohort (i.e. those who had attended either the 4 or 8 month clinics and had not refused to participate subsequently) were compared with the rest of the ALSPAC cohort. This showed that the CIF group had a shortfall of mothers in the lowest educational group (highest qualification None/CSE 14% in CIF vs. 22% in ALSPAC;

p<0.05), and a shortfall of mothers aged under 25 years (13% vs. 21%; p<0.05), and an excess of mothers in owner-occupied accommodation. Nevertheless, there were still a large number of families in the poorer socioeconomic groups, for example 111 (10%) of the families attending the 18 month clinic were living in council accommodation, as compared to 18.5% nationally.

2.1.5-d. Children in Focus: social class composition

Investigation by the author showed a significant difference between the social class of the CIF sub-sample in our studies compared to the Avon area (Census 1991) and to the UK 1992 (NDNS 1995). As Table 2.3 shows, the percentage of studied children who belonged to (paternal) social classes I & II was significantly higher than both the Avon and the UK for the same period. The percentage of those who belonged to social classes IV & V was significantly lower than both Avon and the UK in the same period of time.

Table 2.3. Comparison of paternal social class of the 'Children in Focus', Avon (Census 1991), and the UK (NDNS 1995).

Paternal social class	**UK; 1992 (n=1785)**	**Avon; 1991 (n=63399)**	**Children in Focus (1991/92; n=1155)**
I, II	33.3%	* 41.4%	*‡ 47.6%
III-N	12.5%	13.3%	12.1%
III-M	33.3%	* 28.7%	* 29.1%
IV, V	20.8%	* 16.5%	*‡ 11.2%

* Significantly different from UK (NDNS 1995), p<0.01.

‡ Significantly different from Avon (Census 1991), p<0.01.

2.1.6. Early life anthropometry: ALSPAC compared to UK 1990 reference data for length, height and BMI

2.1.6-a. Children of the 90s

Table 2.4 shows growth measurements for the Children of the 90s at birth in comparison with published UK 1990 reference data (Cole et al 1995). The growth measurements for newborn babies were not significantly different from the UK 1990 reference data.

Table 2.4. Growth measurements of Children of the 90s at birth compared to UK 1990.

Measurements		Children of the 90s (n=14893 in 1992) Mean (standard deviation)	UK 1990 Cole et al (1995)
Boys	Weight (kg)	3.6 (0.5)	3.6
	Length (cm)	51.3 (2.0)	51.0
	BMI (kg/m^2)	13.2 (0.9)	13.3
Girls	Weight (kg)	3.4 (0.4)	3.4
	Length (cm)	50.4 (1.8)	50.2
	BMI (kg/m^2)	13.4 (0.8)	13.0

No significant difference between anthropometric measurements at birth and UK 1990.

2.1.6-b. Children in Focus

It might be worth noting that comparison of growth measures from CIF from 49 and 61 months with the 'UK 1990 reference data' revealed a significantly higher than expected prevalence of obesity in the CIF group (Reilly et al 1999a; Chapter 3). However, our investigation showed that small differences between mean BMI of the 'Children in Focus' and the 'UK 1990 reference data' were not significant at birth, in infancy, and up to 2 years of age (Table 2.5).

Table 2.5. Comparison of mean BMI of CIF children and UK 1990.

Age (month)	BMI	Children in Focus (n=1409) Mean (standard deviation)	UK 1990 (Cole et al 1995)
4	Boys	17.0 (1.4)	17.3
	Girls	16.6 (1.4)	16.7
	Total	16.8 (1.4)	17.0
8	Boys	18.0 (1.5)	17.8
	Girls	17.7 (1.5)	17.4
	Total	17.9 (1.5)	17.6
12	Boys	17.9 (1.4)	17.6
	Girls	17.6 (1.4)	17.2
	Total	17.8 (1.4)	17.4
25	Boys	16.9 (1.3)	16.7
	Girls	16.7 (1.4)	16.3
	Total	16.8 (1.3)	16.5

No significant difference between CIF children mean BMI and UK 1990 reference data.

Height, weight, and BMI standard deviation score curves drawn for the present subjects were compared with expected (ideal) curves. This was carried out to further examine differences between ALSPAC children and national data. Since these are standardised scores, they should represent an idealised (normal) distribution. These show a comparable distribution to UK 1990 reference data in terms of weight and BMI in early life, but CIF children showed slightly greater length measurements than UK 1990 reference data. Figures 2.1 to 2.3 show the normal curves for different ages compared to ideal curves. Normal curves for study subjects at age 37 months were slightly (but not significantly) different from expected with a mean value of about 0.25 SD higher than expected. From age 37 months onwards, the height of subjects was more similar to reference data and no significant difference was seen. Weight and BMI curves from age 37 months onwards tended to be shifted to the right, which means that from around that age, the percentage of obese children started to increase relative to UK 1990 data. This increase in overweight / obesity percentage was higher at age 5 years as described later in this thesis although the mean BMI value still was not statistically different from the 1990 reference data. There was no evidence of important differences between CIF children and the reference population in terms of these anthropometric variables at birth or during infancy.

Figure 2.1. BMI SDS curves in CIF children at different ages compared to expected.

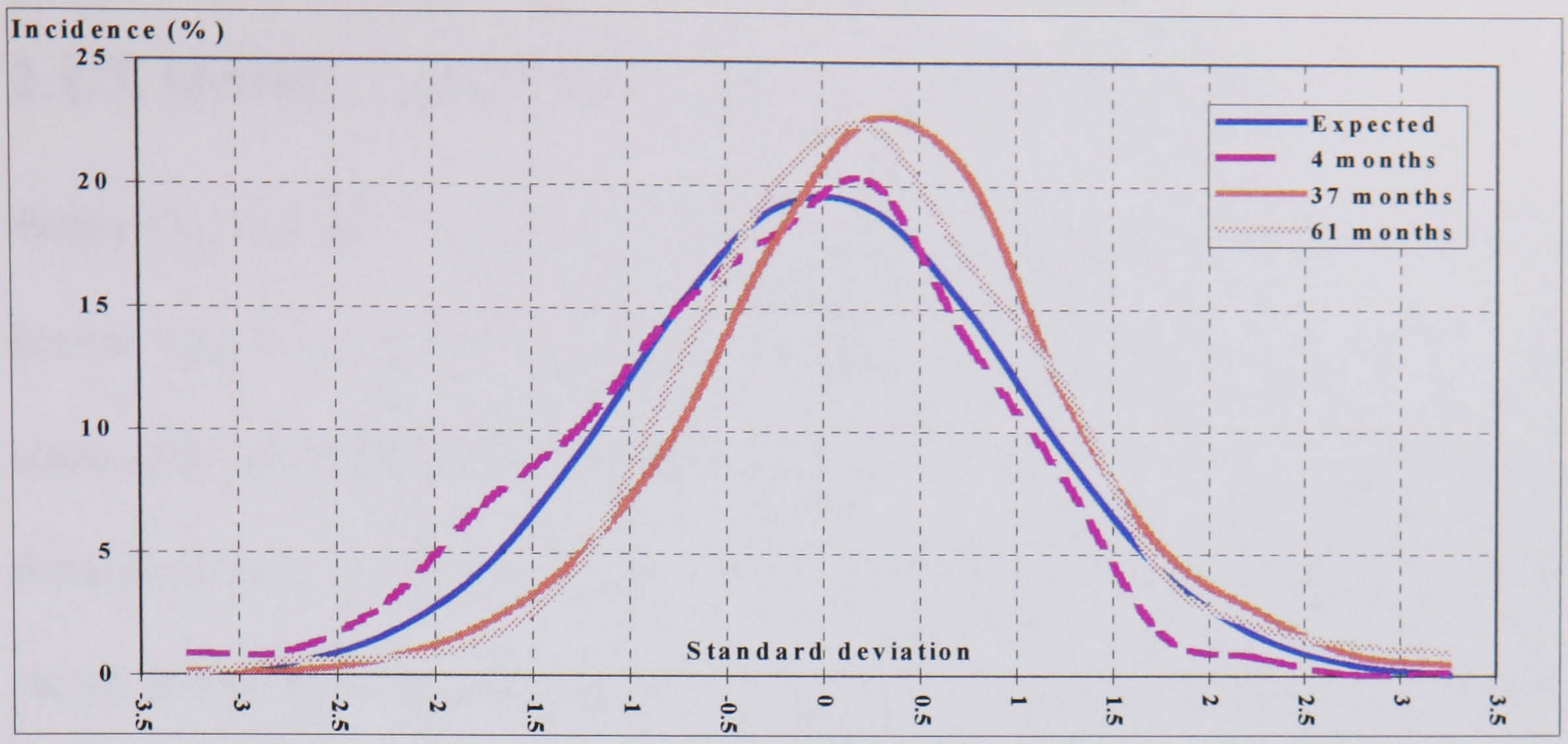


Figure 2.2. Weight SDS curves in CIF children at different ages compared to expected.

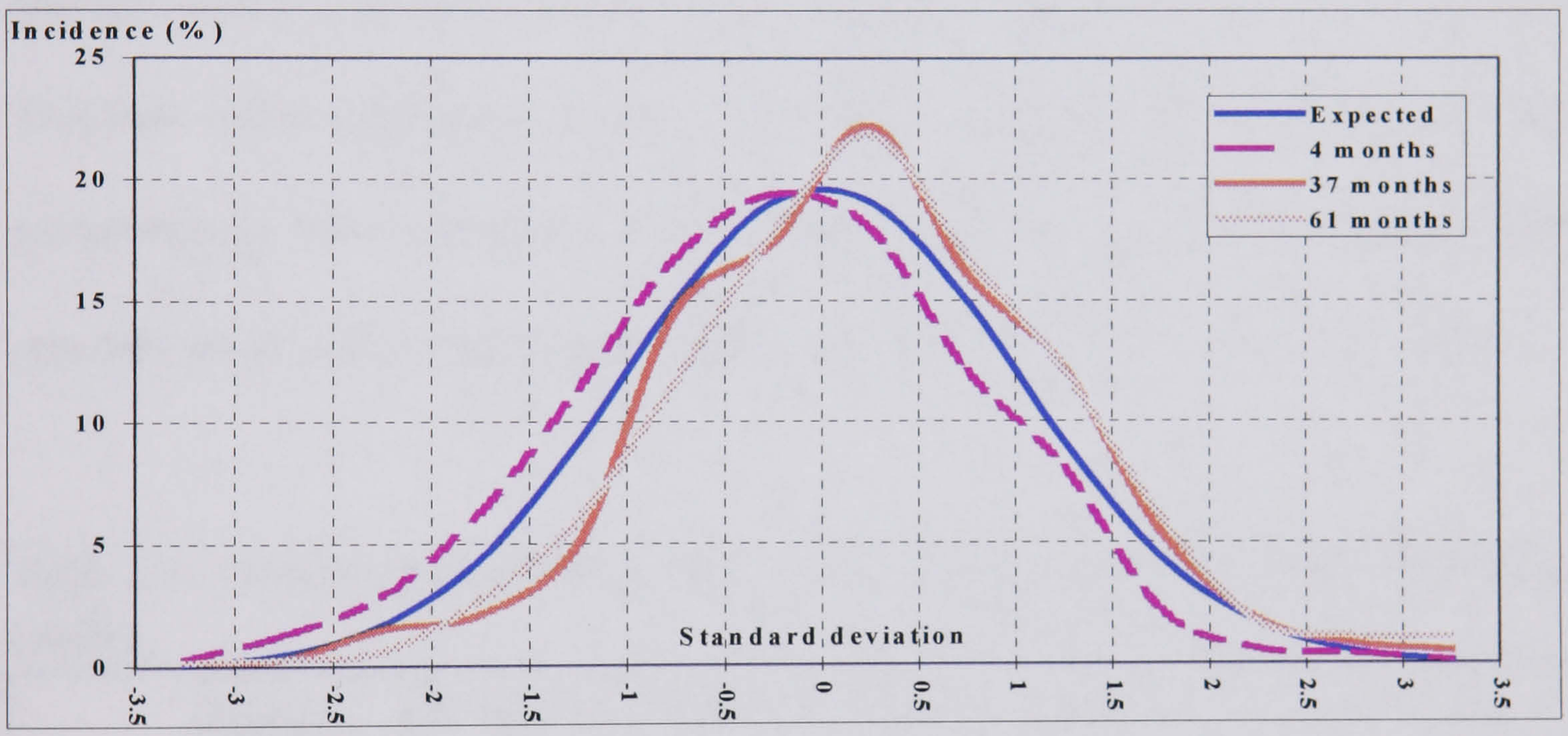
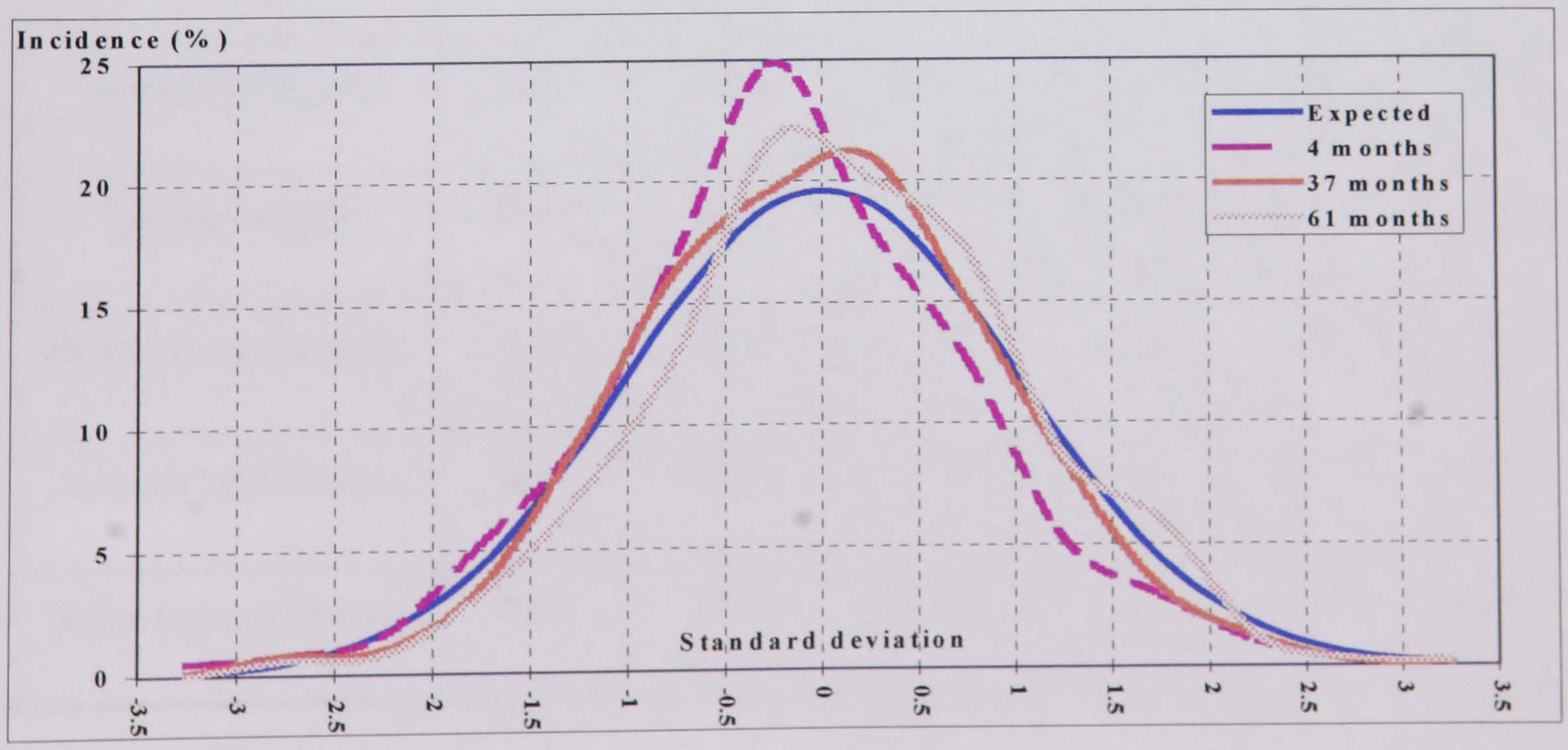


Figure 2.3. Height SDS curves in CIF children at different ages compared to expected.



2.1.7. Quality control of anthropometry in ALSPAC

Before the 4-month clinic (Children of the 90s), staff measured repeatedly a metal rod of known length using the Harpenden Neonatometer under supervision. There was no statistically significant difference between observers. At 12 months all measurements were done on the same 10 children by each of the 5 measurers in turn, confirming their reliability. At 25 months the measurers were asked to measure 10 children with each of the other 4 measurers separately (Table 2.6), so that no child was measured more than twice. In practice pairs 1-5 measured either 9 or 10 children, and the 6th pair measured 7 children. Absolute mean difference (in cm) with 95% confidence intervals for all pairs of measurements were calculated and a maximum of about half a centimetre difference occurred which was not statistically significant (Table 2.7; Children in Focus 1997).

Table 2.6. Correlation coefficients for six pairs of measurers for measurements at age 25 months.

Measurer → Measure (cm)↓	Pair 1	Pair 2	Pair 3	Pair 4	Pair 5	Pair 6
Standing height	0.87	1.00	0.99	0.98	1.00	0.94
Supine height	0.98	0.99	0.99	1.00	0.99	0.99
Head circumference	0.97	0.98	0.89	0.98	0.98	0.03
Arm circumference	0.89	0.86	0.91	0.93	0.97	0.43
Waist circumference	0.95	0.99	0.91	0.97	0.98	0.45

Table 2.7. Absolute mean difference (95% confidence intervals) for six pairs of measurers at 25 months.

Measurer → Measure (cm)↓	Pair 1	Pair 2	Pair 3	Pair 4	Pair 5	Pair 6
Standing height	0.52 (-0.17-1.21)	0.02 (-0.24-0.29)	0.07 (-0.37-0.51)	0.33 (-0.10-0.76)	0.04 (-0.17-0.25)	0.10 (-0.60-0.80)
Supine height	0.63 (0.25-1.02)	0.34 (-0.14-0.82)	0.96 (1.33-0.59)	0.73 (0.55-0.92)	0.09 (-0.25-0.17)	0.37 (0.09-0.65)
Head circumference	0.26 (0.02-0.50)	0.15 (0.07-0.37)	0.10 (-0.15-0.35)	0.54 (0.29-0.80)	0.24 (-0.01-0.49)	1.06 (-0.06-2.17)
Arm circumference	0.16 (-0.23-0.55)	0.99 (0.36-1.62)	0.25 (-0.11-0.61)	0.20 (-0.26-0.66)	0.07 (-0.13-0.27)	0.13 (-0.62-0.87)
Waist circumference	0.08 (-0.49-0.65)	0.26 (0.02-0.50)	0.30 (-0.58-1.18)	0.06 (-0.53-0.65)	0.18 (-0.28-0.64)	1.00 (-2.38-4.38)

At 49 months one experienced measurer (supervisor) measured children after each of the other measurers. Twenty children were used for height, sitting height and waist circumference and 10 children for circumference of arm and head, the variance from the mean being less than with the other measures. The same validation procedures were carried out at all ages including 61 months for all measurements as well as skinfolds, and each time there was no statistically significant difference between measurers or between measurers and their supervisor (Children in Focus 1997).

2.1.8. Quality control of data entry

Most of the self-completion responses in ALSPAC study were self-coding - the ticked reply box contained a printed number that could be directly keyed. Returned questionnaires were checked to verify that each question had no more than one ticked response. All coding was cross-checked by a second person and then keyed and verified, using a commercial bureau (ALSPAC homepage). Each data set for a particular questionnaire was entered on computer twice (to check the validity of data entry). If problems were detected, the data were edited and the completed data set was prepared for analysis. Later, an edited version was derived which excluded all information that could be used to identify a particular person (e.g. dates were converted into age of child in weeks), and a 'built file' was created for use.

2.1.9. Statistical power of analyses for Children of the 90s and CIF children

The study was such that with the number of subjects fixed, it was possible to determine the minimum relative risks that the study was able to demonstrate, depending on the prevalence of the outcome and the proportion of the population exposed. Tables 2.8 and 2.9 relate to binary outcomes (quantitative variables) and Table 2.10 relates to continuous variables. For example, as Table 2.8 shows, for an exposure affecting part of the study population consisting of 10,000 subjects (i.e. 40.0% or 50.0%), the study would be 80% sure of detecting as statistically significant at the 5% level, an increase in relative risk of 1.22 for a common outcome such as childhood obesity (with 5% expected prevalence). However, for an outcome with prevalence of 0.2% (e.g. cerebral palsy), one could only be sure of showing as statistically significant a relative risk of 2.37 or more (ALSPAC homepage; Golding et al 2001).

Table 2.8. Relative risks detectable for 80% power at 5% significance level (n=10000).

Proportion of population exposed	**Incidence of adverse outcome in 10,000 subjects**							
	10%	**7.5%**	**5%**	**2%**	**1%**	**0.75%**	**0.5%**	**0.2%**
50.0%	1.15	1.17	1.22	1.39	1.53	1.62	1.79	2.37
40.0%	1.15	1.18	1.22	1.36	1.54	1.63	1.80	2.38
30.0%	1.16	1.19	1.24	1.39	1.57	1.67	1.85	2.46
20.0%	1.18	1.22	1.27	1.45	1.66	1.77	1.97	2.68
10.0%	1.25	1.29	1.36	1.60	1.89	2.05	2.32	3.29
5.0%	1.34	1.40	1.56	1.85	2.26	2.49	2.89	4.31
2.0%	1.54	1.64	1.81	2.38	3.07	3.46	4.16	6.68
1.0%	1.77	1.93	2.17	3.03	4.09	4.70	5.79	9.82
0.5%	2.12	2.34	2.72	4.03	5.70	6.66	8.41	15.01

As Table 2.9 shows, for an exposure affecting part of the CIF children (e.g. 50.0% of them), the study would be 80% sure of detecting as statistically significant at the 5% level, an increase in relative risk of 1.76 for a common outcome such as childhood obesity (with 5% expected prevalence). For an outcome with prevalence of 0.2% (e.g. cerebral palsy), one could only be sure of showing as statistically significant a relative risk of 8.11 or more

Table 2.9. Relative risks detectable for 80% power at 5% significance level, Children in Focus.

Proportion of population exposed	Incidence of adverse outcome in Children in Focus							
	10%	**7.5%**	**5%**	**2%**	**1%**	**0.75%**	**0.5%**	**0.2%**
50.0%	1.49	1.59	1.76	2.35	3.13	3.60	4.48	8.11
40.0%	1.50	1.60	1.77	2.35	3.12	3.58	4.44	7.95
30.0%	1.53	1.64	1.82	2.43	3.24	3.72	4.61	8.21
20.0%	1.61	1.73	1.94	2.64	3.56	4.11	5.12	9.16
10.0%	1.83	1.99	2.27	3.24	4.51	5.26	6.65	12.28
5.0%	2.16	2.39	2.80	4.22	6.09	7.21	9.27	17.51
2.0%	2.86	3.26	3.95	6.42	9.76	11.76	15.50	30.61
1.0%	3.69	4.28	5.33	9.19	14.50	17.72	23.79	48.60
0.5%	4.84	5.74	7.35	13.40	21.92	27.16	37.11	78.34

Continuous outcome measures

The detectable effect size for continuous variables (in units) was high in both Children of the 90s and CIF children. For instance, the difference in a variable which could be detected with 80% confidence if the variable had a mean of 100 and standard deviation of 15 (at the 5% level of significance) in 10,000 children, would be 0.71 with a SD of 0.047. The detectable differences (in units) are presented in Table 2.10 for both Children of the 90s and CIF children. Values, which are shown in brackets, are the proportion of a SD that would be shown to be statistically significant.

Table 2.10. Unit difference (±SD) detectable with 80% confidence at 5% significance level.

Proportion of population exposed	A population size of n=10,000	Children in Focus n=1000
50.0%	0.71 (0.047)	2.25 (0.150)
40.0%	0.72 (0.048)	2.29 (0.153)
30.0%	0.78 (0.052)	2.45 (0.163)
20.0%	0.89 (0.059)	2.81 (0.187)
10.0%	1.18 (0.079)	3.74 (0.249)
5.0%	1.63 (0.109)	5.15 (0.343)
2.0%	2.54 (0.169)	8.02 (0.535)
1.0%	3.57 (0.238)	11.29 (0.753)
0.5%	5.04 (0.336)	15.92 (1.061)

2.1.10. ALSPAC study measurements / definitions

2.1.10-a Anthropometry

Weight

At age 4 months Fereday scales, at 8 months Soenhle or Seca scales, and from 12 months (12, 18, 25, 31, 37, 43, 49, 61 and 90 months) Seca scales were used to measure weight to 0.1 kg in CIF children, wearing underwear only. It was necessary to use a levelled pan scale with a beam and movable weights during infancy. Weighing procedure followed the protocol described by Hamill et al (1979).

Length / height

Length at age 4 months was measured using Harpenden Neonatometer and at ages 8, 12, 18, and 25 months using Kiddimeter instruments to 0.1 cm. For infants, it is recommended that length measurement should be done by 2 observers so that while the subject is lying in a supine position upon a recumbent length table, one of the measurers can position the head and stand behind the end of the table to ensure that the subject does not change position, and the second measurer places one hand on the knees to ensure that the legs remain flat on the table (Roche & Malina 1999). Recumbent length is commonly measured in infants and small children and if the subject is uncooperative, the reliability is less than that for stature at older ages. The amount of pressure applied to the sliding footboard can affect the measurement variability. Pressure should be sufficient to compress the soft tissues of the foot but not much to alter the length of the vertebral column (Cameron 1984).

From age 25 months onwards (25, 31, 37, 43, 49, 61 and 90 months), the Leicester height measure was used to measure height, to 0.1 cm, in CIF children. In this case, it is recommended that stature should be measured using a vertical board with an attached metric rule and a horizontal headboard that can be brought into contact with the most superior point on the head. The subjects should inhale deeply and maintain a fully erect position without altering the load on the heels and the movable headboard should then be brought onto the superior point on the head with pressure to compress the hair (Wilson 1979). The subject should be barefoot or wearing thin socks and little clothing so that the positioning of the body can be seen. In ALSPAC all these recommendations were adopted. Other sources of height variations which might affect the result of height measuring includes the 'time of the day' when measurement is made since a person is tallest on rising in the morning and shrinks by 0.5 to 1.5 cm during the day as the spine becomes compressed (Strickland & Shearin 1972; Buckler 1978). This can be overcome by slightly stretching the very young child when measuring the height. The investigators in Werther's (1998) study applied gentle upward pressure on the mastoid during height measurement which limited the height 'loss' to about 4.6 mm (Werther 1998). However, other investigators have shown that simple measuring devices can give reliable readings and that the major errors arise from the observer and the person being measured, rather than the time of the day or by stretching or not stretching (Ahmed et al 1990; Voss et al 1990). No final consensus has been reached (Werther 1998).

BMI

For the Children of the 90s at age 7 years (90 months), and for the CIF children at ages 4, 8, 12, 18, 25, 31, 37, 43, 49, and 61 months BMI was calculated using the following formula:

$$\text{BMI} = \text{weight (kg)} / \text{height}^2 \text{ (m}^2\text{)}$$

Body impedance and derived body fatness estimates

At age 7 years, it was planned to measure body impedance of all 'Children of the 90s' and clinics were run for this purpose between September 1998 and September 2000. A total of 7766 children for whom birth data were available were seen at age 7 years. Whole body bioelectrical impedance at 50 kHz was used in conjunction with the Houtkooper prediction equation (Houtkooper et al 1992). The equation was as follows:

FFM (kg) = 0.61 (RI) + 0.25 (weight, kg) + 1.31

Where resistance index (RI) = height (cm^2) / resistance (ohms)

This formula has been shown to be accurate in British prepubertal children (relative to a 2-component model with underwater weighing; Reilly et al 1996). Ideally, subjects must be measured in the morning, before breakfast and after emptying the bladder (Jansen et al 1992). It was decided that, in this case, it was impractical to ask children to fast since only half the clinic staffs were available in the afternoon. Therefore, a record was kept of the last eating and drinking occasion so that adjustment could be made if necessary. However only about 5% of children, who have been measured, were not able to empty their bladder before the measurement. The body impedance method is described in more detailed in Chapter 5.

Definition of adiposity rebound

Rolland-Cachera et al (1984) studied adiposity rebound in a group of 151 French children consisting of 79 boys and 72 girls. The subject's BMI were measured from birth and every 6 months up to 14 years of age. Age of adiposity rebound was defined on the basis of age corresponding to the lowest value of BMI recorded at the onset of the increase in adiposity. In the current thesis, for CIF children measurements were taken at ages 4, 8, 12, 18, 25, 31, 37, 43, 49, and 61 months, and BMI was calculated. Timing of adiposity rebound (AR) was assessed by visual inspection of data, using the method of Rolland-Cachera et al (1984), but with the modification that any increase in BMI after the nadir had to be equal or exceed 0.1 kg/m^2 before AR was confirmed. In addition, timing of AR was only assessed for those CIF children with BMI values for most of the 10 measurement occasions. Further details are given in Chapter 6.

2.1.10-b Dietary intake of energy and macronutrients

The lack of data on dietary intakes in young (e.g. pre-school) children means that relatively little is known of the advantages and disadvantages of different methods of dietary assessment in this age group. The current literature is almost all related to studies in adults (Livingstone et al 1992; Livingstone & Robson 2000; Hill & Davies 2001). Assessing diet in small children differs from most dietary assessment methods in adults since the process relies on the observations of a carer or other individual, rather than self-report; therefore, measuring dietary intakes of children is very difficult and problematic (Goran 1998). By around 10 years old, children are better able to give dietary information and are more aware of the foods they have eaten (Baranowski & Domel 1994). In all the studies described in this thesis dietary information was obtained from the carer, usually the mother.

Methods of dietary assessment in pre-school children

Food frequency questionnaires

The food frequency questionnaire (FFQ) consists of a list of foods and beverages as well as options for choosing the frequency of consumption for each food. The options normally range from never to more than once per day. Some detailed extra questions of particular foods or methods of cooking might be added (Baranowski et al 1991). When the typical diet during several months (or even several years), or the intake of particular group of foods / nutrients are the focus of questioning, then FFQ is useful. FFQs are very popular for use in epidemiological studies, since it is possible to mail them to people and they are easy to code and use (Baranowski et al 1991). FFQs are not usually used to assess the overall diet and can not provide precise measures of nutrient intakes since they do not gather detailed information about portion sizes (Bingham et al 1994). It is important to note that the FFQ method has limited value in studying inter-individual variations in diet and also needs to be culture-specific. For instance, using traditional FFQ measures the long-term diet which may not be appropriate for small children (e.g. 18 month old Children in ALSPAC) due to rapid

changes in the child diet. It was not possible to use this method in the ALSPAC study since one of the original ALSPAC study aims was to compare the intakes of children to the UK dietary reference values (Golding et al 2001).

Twenty-four hour recall

To carry out a 24-hour recall, an interview is needed. The interviewer asks the subject about the consumed foods / drinks during the previous 24 hours, or between the yesterday midnight and the midnight of the day before yesterday (Baranowski et al 1991; Bingham et al 1994). The interviewer needs to be trained to obtain as much information as possible, and to persuade the subject / carer to remember easily forgotten food items such as snacks and condiments. To estimate portion sizes, food models or photographs might be used (Baranowski et al 1991; Bingham et al 1994). The main advantage of this method is the ability to cope with the variation between individuals in term of details of food / drinks consumed. A further advantage is that it can be used with illiterate subjects. There are some disadvantages using this method including its high cost since it usually needs a face-to-face interview, which typically takes 20-30 minutes. In addition, the whole method is based on the subject's memory, which will be particularly problematic for estimating portion sizes. This will be more problematic in children since most children are cared for by several different people during the day, and it is often impossible to find someone who was able to observe food intake during the entire day (Baranowski et al 1991). Furthermore, it provides no measure of day-to-day variation in nutrient intake. To obtain information on day-to-day variability in nutrient intakes, several repeat 24-hour recalls are necessary (which obviously will make the process more expensive and time-consuming). Therefore, this method is only suitable for estimating the population mean intakes, and there are some doubts about using it when the individual's food / nutrient intake have to be linked to an outcome variable (Baranowski et al 1991; Bingham et al 1994). These problems were reasons for rejecting the 24-hour recall method in the original ALSPAC study designs.

Diet history

In relation to clinical / research purposes, different methods of taking diet history have been used (Baranowski et al 1991; Bingham et al 1994) but they usually begin with a 24 hour recall (with detailed questioning as to the usual portions eaten, and possibly using food models as an aid), followed by questioning the weekly pattern of diet. To check the weekly pattern of diet, the subject will be questioned about variations in foods eaten from those in the 24-hour recall. The questions will be related to differences due to weekend / working patterns. Probing questions are used to investigate consumption of snacks and alcoholic drinks. This information is then cross-checked against a food-frequency questionnaire (British Dietetic Association 1979). This method of dietary assessment is extremely time-consuming. In the ALSPAC study, the CIF children attended a research clinic at which only about 10 minutes was devoted to a dietary interview which was not sufficient to obtain the detail required for a diet history, and so diet history was not practical.

Weighed intakes

In a weighed intake, the subject is given a set of food scales and a booklet in which to record the weights of any food and drink taken, and the weights of any leftovers. Typically an investigator will visit subjects to deliver the scales and instruct them in how to use them, and this is often followed-up by another visit to ensure the instructions have been understood and there were no problems. When the completed 7-day record is received, the subject may be interviewed to obtain any extra information necessary on food preparation methods (Baranowski et al 1991). Like food records and 24-hour recalls, variation in diet can be accommodated, and it allows detail to be recorded about food preparation methods. An exact measurement of portion size is provided, and the method is not reliant on memory. A seven-day weighed intake is often regarded as the 'gold standard' of dietary assessment methods (Livingstone et al 1990; Samaras et al 1998), but in practice sometimes shorter periods of 3-4 days of weighed recording are used (Crawford et al 1994; Davies et al 1994; Stallings et al 1996; Samaras et al 1998; Skinner et al 1999; Barden et al 2000).

There are however, a number of potential problems with this method of dietary assessment. It is very expensive to provide the scales and instruct the subjects in their use. It places a large burden on the subject, and is unsuitable for foods eaten out, due to the inconvenience and possible embarrassment of having to carry the scales around and weigh foods in public places. This burden on the subject may alter the diet during the recording period, or reduce accuracy and compliance, and the importance of this problem is likely to increase with the length of the dietary recording period (Barrett-Connor 1991; Black et al 1993; Buzzard et al 1996; Hill & Davies 2001). In some studies, the burden for subjects represented by a 7-day recording period is so great that the 7-days are recorded over more than a week (Payne & Belton 1992). Such an assessment over a long period of time was not possible in ALSPAC study which was designed to obtain many measurements in a very limited period of time. In addition, the process of weighing their food may cause people to alter their habitual diet. For example, if a subject / subject's parent is eating a mixed dish they are asked to weigh all the ingredients before cooking as well as the weight of the portion they eventually consume. Mixed dishes are therefore likely to be avoided as they are inconvenient to weigh. In addition, as the weighing of food focuses so much attention on the diet, subjects may be tempted to adopt more healthy or 'acceptable' dietary habits for the recording period (Bingham 1987; Buzzard et al 1996). In older children, adolescents, and adults, there is a tendency for subjects to under-report their intake when using weighed records (Buzzard et al 1996; Hill & Davies 2001). The weighed record was rejected for the ALSPAC study mainly on the grounds of expense and burden on the subject.

Unweighed food record

This method is similar to the weighed intake, except that instead of weighing the amount of foods and drinks consumed, these are described in household measures (Gillum et al 1996; Cowin & Emmett 2000). The subject is given a booklet in which to record their food intake,

along with an instruction and example sheet and ideally the subject will also be instructed by a trained researcher on how best to complete the food record. When the food record is completed, the subject is normally interviewed to ensure that the record is clear and complete. While the portion size information is likely to be less accurate than with a weighed record, this method shares many of the advantages of the weighed record, such as being able to cope with any amount of variation in the diet, and not relying on memory. Accuracy of portion size estimation may be improved by the use of food models or photographs in the interview. Completion of an unweighed record places a smaller burden on the subject than weighed intakes. This is likely to increase compliance rates, and reduce the chances of subjects changing their normal diet. This method has been used in many studies of children's food intake, even in the UK (Hackett et al 1986; Benton & Roberts 1988; Adamson et al 1992; Crawley & While 1996; Cowin & Emmett 2000; Dorosty et al 2000, as discussed later in this Chapter).

Validity of dietary assessment methods

Validity of dietary energy intake assessment is usually tested by comparing energy intake with total energy expenditure measured using the doubly labeled water (DLW) method (Hill & Davies 2001). In subjects who are in energy balance, total energy expenditure (TEE) should be equal to energy intake (EI), and the TEE can be accurately measured by DLW (Hill & Davies 2001). In growing children, a very small positive energy balance is expected, but this is usually ignored (Goran 2001) because it is so small. Validation studies in pre-school and pre-pubertal children are scarce (Martorell et al 2001). However, in general, weighed and unweighed dietary recording methods seem to agree reasonably well with measurement of TEE, on average, in young children (Hill & Davies 2001). In addition, there seems little advantage to using weighed rather than unweighed records when validation studies are considered (Hill & Davies 2001).

In general, the validity of all dietary assessment methods is in some doubt and there is a need for more validation studies (Livingstone & Robson 2000) but under-reporting of intake is less of a problem in young children than in adolescents and adults (Davies 1997; Livingstone & Robson 2000; Hill & Davies 2001).

Prospective studies where data are collected prior to illness provide more reliable estimations of the association between diet and disease (COMA 1998). Large epidemiological studies have investigated and developed the instruments to measure habitual dietary intakes (COMA 1998). There is no dietary assessment method which is able to measure the habitual diet with complete accuracy and precision (Bingham et al 1994) and dietary instruments, which can be used in large size studies, are limited by finances and time. However, food frequency questionnaires (FFQ) have been most commonly used (Willett 1998), though the use of diet diaries has also increased (Bingham et al 1997). One of the important limitations in epidemiological studies is obtaining repeated diaries or food frequency questionnaires (Day et al 2001).

2.1.10-c. Dietary assessment method used in ALSPAC

A three-day unweighed food record was the method of dietary assessment chosen in the ALSPAC study. While more days of dietary record would have been desirable and would have provided greater precision (Wassertheil-Smoller et al 1993), it was felt that this would place an unacceptable burden on the parents, and possibly reduce response rate. It has been shown, however, that for the age group 1-4 years, fewer days of dietary recording are required to rank nutrient intakes with a given accuracy than in other age groups (Nelson et al 1989). The present thesis focused on total energy, fat, and protein intakes because of evidence that they have special roles in obesity (Camargo & Colditz 2000). The approach taken here is widely used in research on children (Hackett et al 1986; Benton & Roberts

1988; Adamson et al 1992; Crawley & While 1996; Cowin & Emmett 2000), although other investigations have taken different approaches like the 4- to 7-day weighed food record (Livingstone et al 1992; Payne & Belton 1992; NDNS 1995; Ruxton et al 1995; Bratteby et al 1998). NDNS (1995) used 4-days weighed food record and adjusted intake to 7-days which has been referred to as the 'gold standard' (Livingstone et al 1990; Samaras et al 1998). As already mentioned, time and staff limitations in the ALSPAC study were important reasons to choose the 3-day unweighed dietary assessment. However, as discussed by Hill and Davies (2001), results of validation studies in young children suggest no great benefit to using weighed rather than unweighed records, and also suggest that 7 days is not essential for many purposes.

The most important reasons for choosing 3-day records in the ALSPAC cohort were: simplicity for parents to understand what to do with only an instruction leaflet; only a limited amount of staff / time was available to administer the diary (at clinic visits, limited time was available to check diary with parents); small children tend to eat less variety of foods (Skinner et al 1999) and therefore, less days recording was necessary; the 3-day period was chosen to minimise cost without too much loss of information. This method gives much more detail of foods actually eaten and timing of meals than FFQ though is much more complex and time consuming to analyse than FFQ. It is worth noting that both macronutrient and micronutrient intakes of CIF children (our study sample) were similar to children of the same age in the NDNS (Cowin & Emmett 2000). ALSPAC nutritionists assessed the diet of CIF children (based on 3-day unweighed dietary record) at age 18 months and found that the intakes of energy and other nutrients were significantly higher in boys than in girls, and for most nutrients the mean and median intakes were well above the Reference Nutrient Intakes (RNI) except for vitamin D, iron and zinc (Cowin & Emmett 2000).

2.1.11. Definitions of overweight and obesity

There have been a great many methods of defining overweight and obesity in childhood. These range from simple, subjective assessments (e.g. clinical judgement) which have been shown to be invalid (Cross et al 1995), to technically complex and objective definitions.

Obesity has been defined as an excess of body fatness associated with ill-health (WHO 1998). This implies a definition based on a measurement of body composition (body fatness), and that the relationship between body fatness and ill-health is known for children. However, body composition measurements are usually impractical for epidemiology and clinical practice (Forbes 1999), and the exact relationship between a given level of body fatness and ill-health is not clear. Body composition methods for use in children are described below, but as the present study was epidemiological, it was based largely on BMI (a simple definition of obesity), though body fat % was used as a variable in the study as described in Chapter 5.

There is evidence showing that *body fat distribution* is important to health in children and adolescents (Rolland-Cachera et al 1990; Zwiauer et al 1990; Zwiauer et al 1992; Freedman et al 1995; Power et al 1997; Goran 1998; Slyper 1998; Daniels et al 1999; Morrison et al 1999-a,b), not just in adults (Han et al 1998). It could be argued that body fat distribution should be incorporated into a definition of childhood obesity, but there is as yet no agreement over how to measure body fat distribution in children (Mueller & Kaplowitz 1994; Goran 1997; Daniels et al 1999; Daniels et al 2000), and no agreement on what level of 'central' fatness is associated with ill-health. For these reasons the author did not include body fat distribution as an index of overweight or obesity.

2.1.11-a. Weight alone

Weight is a simple and direct index of body size, easy to measure, cheap and reproducible. It is also reasonably highly correlated with body fat. In addition, measuring weight does not need trained observers and continuous quality control (Power et al 1997). However, body weight does not give information about the specific components of body composition. For example, very athletic persons may be heavy because of excess muscle or lean body mass rather than because of excess body fat (Power et al 1997; Kuczmarski & Flegal 2000). An important limitation of using weight alone is that weight is associated with height, so taller children tend to be heavier. There is a relationship between age and height (and therefore weight) but only a weak relationship between age and fatness (Cole et al 1995; Power et al 1997). Therefore, using weight alone as an index of obesity is not recommended (Sawaya et al 1995; Uauy et al 2001).

2.1.11-b. Weight for height

The positive correlation of weight with height means that weight adjusted for height is far more useful than weight alone as an index for assessing fatness. A weight for height index does not measure fatness directly but can be used to identify overweight. Since weight for height takes no account of the child's age, it is useful in parts of the world where dates of birth are not recorded but leads to a biased assessment in infancy and adolescence (Power et al 1997). The World Health Organisation's international growth reference (Dibley et al 1987) includes a weight for height chart, which stops at age 10 years for girls and 11.5 years for boys. So for the assessment of overweight during or after adolescence is of no value (Cole et al 1995; Power et al 1997).

2.1.11-c. Weight adjusted for height

Weight adjusted for height provides a simple but crude index of fatness (Cole et al 1995). Body weight is reasonably correlated with body fat but is also highly correlated with height, which is weakly correlated with body fat (Power et al 1997; Bellizzi & Dietz 1999). Therefore, weight

adjusted for height is a far more useful index with which to assess overweight and is a reasonable indicator of fatness (Bellizzi & Dietz 1999). Weight adjusted for height squared (body mass index; BMI) is now used and recommended widely to measure child overweight (Cole et al 1995; WHO 1995; Power et al 1997; Barlow & Dietz 1998; Prentice 1998; Troiano & Flegal 1998; WHO 1998; Bellizzi & Dietz 1999; Cole et al 2000).

2.1.11-d. Body mass index

The BMI is a weight adjusted for height index (Pietrobelli et al 1998) and is now broadly accepted as an index of fatness since is independent of height and is applicable to both sexes (Prentice 1998). BMI is not highly correlated with height, but is correlated with body fat (Power et al 1997; Pietrobelli et al 1998). Although BMI is moderately correlated with body fatness, it does not quantify total body fatness in individuals (Kuczmarski & Flegal 2000). The measurements used to derive BMI are reliable and non-intrusive (Bellizzi & Dietz 1999). In children and adolescents, BMI must be interpreted relative to population reference data (Ogden et al 1997; Barlow & Dietz 1998) and overweight / obesity should be defined relative to specific percentile of the reference population based on age, sex, race and/or other group characteristics (Troiano & Flegal 1998). The reason for this is that BMI changes with age and differs between boys and girls (Cole et al 1995; Power et al 1997). Many countries have published BMI for age charts for their populations, and some have also defined cut-off points on these charts to define overweight and obesity (Bellizzi & Dietz 1999). However, the cut-off points vary from country to country and therefore, different BMI for age values have been used to define overweight and obesity in different populations (Bellizzi & Dietz 1999). However, BMI $\geq 85^{th}$ centile and BMI $\geq 95^{th}$ centile relative to a population standard are now widely used to define overweight and obesity respectively (Robinson 1993; Himes & Dietz 1994; Gortmaker et al 1996; Freedman et al 1997; Whitaker et al 1997; Slyper 1998; Troiano & Flegal 1998; Freedman et al 1999; Troiano & Flegal 1999; Zephier et al 1999; Sahota et al 2000). A secular trend of increasing

fatness (skinfold thickness) in British children had been shown prior to the beginning of this thesis (Hughes et al 1997), but its significance for the childhood obesity issue was not understood at the time due to the lack of an agreed obesity definition. An expert committee in the USA has subsequently decided to define BMI ≥95th percentile (compared with population reference data) as obese (Barlow & Dietz 1998). Applying percentiles to define childhood obesity, however, means that the definition is relative to an age and sex specific reference population (Troiano & Flegal 1998) and in this thesis, the Cole 'UK 1990' reference data for BMI (Cole et al 1995) have been used (refer to Chapter 3). These are intended to represent the weight, height, and BMI of children in the UK in 1990.

Validity of UK 1990 reference data for BMI, weight, length, and height

As noted above, there is a need to interpret BMI data in a child by comparing against reference data. In the UK, the current reference data for BMI are those of Cole et al (1995). These are intended to describe the BMI distribution of British children in 1990. Cole et al (1995) presented centile curves for BMI in British children from 33 weeks gestational to 23 years, based on a combination of 11 distinct surveys done between 1978 and 1990 using data of 15636 boys and 14899 girls. These were then 'adjusted' to the BMI data obtained from the 1990 UK National Study of Health and Growth (Chinn & Rona 1994). The reference data were derived using Cole's LMS method, which adjusts the BMI distribution for skewness and allows BMI in individual subjects to be expressed as an exact centile or standard deviation score (SDS; Cole et al 1995).

Although the UK 1990 data (Cole et al 1995) are the only available reference data for BMI of UK children, authoritative authors (e.g. Prentice 1998) and the Royal College of Paediatrics and Child Health (RCPCH) expert committee on growth standards (RCPCH 2001) reported positive reasons for using them. In particular, they noted that:

-The original Tanner-Whitehouse (1975) and the Gairdner-Pearson (1971) growth references for weight and height / length, were no longer reliable for use at any age.

-The UK 1990 (Cole et al 1995) reference data for weight, length, and head circumference are the only datasets suitable for those under 2 years of age.

-The UK 1990 reference data should be used when both weight and height (and therefore BMI) are being evaluated.

-The UK 1990 reference data for BMI, are the only suitable ones for assessing weight relative to height.

In addition, a number of authors have tested the 'external validity' of the UK 1990 reference data in independent samples of infants and children. Savage et al (1999) studied 127 infants to determine the suitability of the UK 1990 reference data for the assessment of growth and nutritional status in infancy and early childhood. They concluded that the UK 1990 reference data had only small biases which were not clinically important and were considerably more appropriate than the older reference data (Tanner-Whitehouse 1975). These authors suggested that the old Tanner-Whitehouse (1975) reference data for head circumference and skinfold thicknesses were inappropriate (Savage et al 1999). In another recent longitudinal study on English school children (7-9 years old at beginning and 9-11 years after 3 years in 1996-1998), the UK 1990 reference data for weight, height, and BMI were compared with previous '1962 Tanner-Whitehouse' (TW) British reference data to first find out if the TW standard was still appropriate for today's school children, and then to ascertain how longitudinal growth was related to the UK 1990 reference data (Rudolf et al 2000). The authors concluded that although school age children's weight is increasing, the UK 1990 reference data are appropriate for British children (Rudolf et al 2000). Reilly et al (1999) studied 225 7 year old children to determine the suitability of the UK 1990 reference data for height, weight, and BMI. Their study supported the use of the currently recommended height, weight and BMI reference data (Cole 1990) to define overweight and obesity in children (Reilly et al 1999b).

Use of 'fixed' reference data for BMI

As described earlier, the UK 1990 reference data represent height, weight, and BMI from 33 weeks gestational to age 23 years for British children in 1990. A variety of British studies from 1990 (discussed later, in Chapter 3 of this thesis) showed that childhood obesity prevalence compared to UK 1990 reference data has significantly increased and there has been a strong trend towards increased prevalence of childhood obesity (Reilly & Dorosty 1999; Reilly et al 1999a; Kinra et al 2000; Sahota et al 2000; Bundred et al 2001; Chinn & Rona 2001; Rudolf et al 2001). This means that to make comparisons between childhood obesity in children from different periods of time, a fixed, dated, set of reference data is needed so that the prevalence of childhood obesity in each period can be identified compared to the reference. In the UK, it is recommended that the UK 1990 reference data are used as a fixed reference point and not adjusted upwards to allow for the increase in childhood obesity prevalence (Prentice 1998; RCPCH 2001; Wright et al 2002).

Advantages of BMI

Measures of height and weight are simple and more reliable than other indices of paediatric overweight / obesity (Dietz & Robinson 1998). BMI is related to health outcomes in childhood and is predictive of disease (Dietz 1998a; Freedman et al 1999; Morrison et al 1999a,b). It has been shown that the relative risk of various co-morbidities (e.g. cardiovascular risk factors; persistence of obesity) increases with obesity (defined using BMI as BMI $\geq 95^{th}$ centile of US reference data). One important source of morbidity is psychosocial consequences which increase significantly above the 95th centile in US children (Dietz 1998a). Obesity in adolescent females has adverse socioeconomic consequences in early adulthood in the USA (Gortmaker et al 1993) and the UK (Sargent & Blanchflower 1994). For example, educational attainment and income are reduced in the obese, even when corrected for I.Q. (Sargent & Blanchflower 1994). In addition, using BMI percentiles for definition of childhood obesity is now widely recommended (Power et al 1997; Barlow & Dietz 1998; Prentice 1998; Bellizzi & Dietz 1999). Furthermore, as

described in Chapter 5 of this thesis, BMI is highly specific (low false positive rate) and can be reasonably sensitive (moderate false negative rate) when screening for obese children (i.e. successfully identifies the fattest children).

Disadvantages of BMI

Obesity is an excess of body fat, not an excess of body weight (Wells 2000). This means that having a higher BMI does not necessarily mean that an individual is very fat. In a minority of children, heavy musculature might be confused with obesity (Fredriks et al 2000). The weak correlation between BMI and height causes taller populations to display a higher prevalence of obesity (Bellizzi & Dietz 1999). However, it is likely that taller people are at higher risk of obesity because tallness might reflect availability of nutrients, and because obese children tend to be taller (Dietz 1997b; Pietrobelli et al 1998; Bellizzi & Dietz 1999; Ong et al 2000; Fredriks et al 2000). While BMI is highly correlated with fatness in childhood (Pietrobelli et al 1998), the relationship between BMI and body fat percentage in *individuals* has been shown to be only moderate both in infancy and childhood (Fredriks et al 2000; Wells 2000). In the Hughes study, during the period of 1972-1994 changes in triceps skinfold clearly showed an increase in fatness in British children while BMI changes were less marked. BMI was probably not sensitive enough to accurately describe population trends in fatness in British children during the 1970s and 1980s (Hughes et al 1997). BMI changes dramatically with age during childhood and adolescence. Weight and height changes during the process of development are different. During growth and maturation, body proportions, bone mass, and the ratio of lean-to-fat mass change at different rates (Reilly et al 1995; Troiano & Flegal 1998). Male infants have a consistently higher fat free mass (FFM) throughout infancy, which accounts for greater body mass. Adipose tissue deposition rates vary with age (growth and maturation) and gender as well (Pietrobelli et al 1998; Wells 2000). In addition to age and sex effects, ethnicity, sexual maturity, sitting height, fat distribution and disease state have all been shown to alter the general relationship between BMI and percentage fat (Daniels et al 1997; Wells 2000). In both adults and children

increased intra-abdominal fat is a specific cardiovascular risk factor (Fujioka et al 1987; Seidell et al 1987; Bjorntorp 1992; Goran et al 1995; Caprio et al 1996; Krauss & Winston 1998). Therefore, distinguishing between abdominal and central fat is important, but BMI does not make this distinction and cannot be used to classify fat distribution (Goran et al 1995).

Despite the disadvantages of the BMI, the majority of large-scale studies or reports on overweight / obesity in children have used BMI as an index of body fatness, and BMI has been recommended as the best measurement for monitoring overweight in individuals in the paediatric population (Cole 1991; Himes & Dietz 1994; WHO 1995; Prentice 1998; Dietz & Bellizzi 1999; Wells 2000). In addition, BMI has now been recommended by a number of expert committees like the Expert Committee on Clinical Guidelines for Overweight in Adolescent Preventive Services (Himes & Dietz 1994), and the committee of paediatric obesity experts (consisting of the Maternal and Child Health Bureau, Health Resources and Services Administration, the Department of Health and Human Services; Barlow & Dietz 1998) and other reviews (Dietz & Bellizzi 1999). However, in clinical settings a direct measure of body fat such as triceps skinfold thickness can be useful to confirm that a high BMI is really high body fat or is just an increase in frame size / muscle mass (Dietz & Robinson 1998), though this is not always practical.

BMI centiles and SD scores

To determine the percentage of overweight / obese children, centiles and standard deviation scores (Z-scores) should be used (Krick 1986; Davies et al 1993; Cole et al 1995). In statistics, it is assumed that each variable in a normal population has a normal curve when a mean value (which in an ideal curve is equal to median, and mode value) can be identified for that variable, and 50% of the population have a value less than the mean value while the other 50% of the population have a value more than the mean value. Those individuals who have a value less than mean - 1.04SD, are 15% of the reference population and are defined as having a value $\leq 15^{th}$

centile (Troiano & Flegal 1998). Hence 5% of the reference population are expected to have a value higher than mean + 1.64SD (≥95th centile), while 5% of the reference population are expected to have a value less than mean - 1.64SD (≤5th centile; Troiano & Flegal 1998). As noted above, cut-off points for centiles or SD scores are needed for defining overweight or obesity in children because BMI changes with age (Ogden et al 1997; Barlow & Dietz 1998; Troiano & Flegal 1998; Bellizzi & Dietz 1999).

BMI cut-offs used in this thesis to estimate prevalence of obesity / overweight

At each age when a child's BMI was compared to age and sex specific UK 1990 reference data (Cole et al 1995), those who had BMI SDS ≥1.04 (equivalent to BMI ≥85th centile) were defined as overweight, and those who had a BMI SDS ≥1.64 (equivalent to BMI ≥95th centile) were defined as obese (Barlow & Dietz 1998; Troiano & Flegal 1998; Bellizzi & Dietz 1999). It is worth noting that the absolute BMI values equivalent to BMI ≥85th and ≥95th percentiles in different reference data sets are very similar (Table 2.11; also refer to Table 4.3 of Chapter 4) and therefore, using these criterion as definitions of overweight / obesity is not expected to be based on far different BMI absolute values in different children born in different countries.

Table 2.11. Iranian, USA, UK & French BMI values to define overweight / obese 5&7 year olds.

Age (y)	Reference data / Absolute child's BMI		Iran Hosseini et al 1999	UK Cole et al 1995	USA Rosner et al 1998	French Rolland-Cachera et al 1991
5	**Obese** (BMI ≥95thcentile)	Boys	18.3	17.9	18.3	17.6
		Girls	18.8	18.3	18.5	17.1
	Overweight (BMI ≥85thcentile)	Boys	16.4	17.0	17.2	16.8
		Girls	16.5	17.2	16.9	16.4
7	**Obese** (BMI ≥95thcentile)	Boys	18.1	18.5	20.0	17.9
		Girls	18.5	19.2	20.4	17.6
	Overweight (BMI ≥85thcentile)	Boys	16.4	17.2	17.8	17.0
		Girls	16.4	17.7	17.9	16.7

IOTF reference data

The working group on childhood obesity of the International Obesity Task Force (IOTF) was established in 1994 and proposed to develop an internationally acceptable definition of child overweight and obesity, and specify the measurement, the reference population, and the age and sex specific cut-off points (Dietz & Bellizzi 1999; Guillaume 1999). The results of an international survey of six large nationally representative cross-sectional growth studies from Brazil, Great Britain, Hong Kong, the Netherlands, Singapore, and the USA were combined. The BMI of a sample of 97,876 males and 94,851 females from birth to 25 years of age was studied (Cole et al 2000). For each survey of those countries, centile curves were drawn for 18 year olds and passed through the widely used cut-off points of 25 and 30 kg/m^2 for adult overweight and obesity. The resulting curves were averaged to provide age and sex specific cut-off points for 2-18 years. The authors believe that these cut-off points are less arbitrary and more internationally based than current alternatives, and that they might help to provide internationally comparable prevalence rates of overweight and obesity in children (Cole et al 2000). They are intended primarily for international comparisons of overweight and obesity prevalence, and have been used in this way in Chapter 4.

2.1.12. Alternative approaches to defining childhood overweight and obesity

2.1.12-a. Body composition in children

Body composition (body fatness) in children can be measured using methods which range from relatively simple and inexpensive (e.g. skinfold thickness; Reilly et al 1995; Reilly 1998) to complex and expensive (e.g. CT scan; Goran 1997; Goran & Gower 1999). It should be noted that there can be no 'gold standard' for measurement of body composition since this would require direct chemical analysis (Reilly 1998). Instead, the concept of the 'reference method' has been widely used. A reference method is a method considered to be of high accuracy. Other simpler or newer methods can be validated by comparing their results against those of a

reference method (Reilly 1998). Until around the late 1990s, the 2-component model (described below) usually based on measurement of body density by hydrostatic weighing, was considered a reference method. Its limitations (briefly described below) led to the development of more complex 'multi-component models' as a reference method (Wells et al 1999).

2.1.12-b. Two-component versus multi-component methods

Body composition in children is difficult to measure with accuracy and precision. Most simple methods are based on the 2-component model which divides body weight into fat mass (FM) and fat free mass (FFM). The common assumption in these models is that fat has density of 0.9 kg/l and fat-free mass 1.1 kg/l (in adults) or that water is a fixed percentage of FFM. In addition, it is assumed in childhood that fat-free mass has a 'constant' water content and body density, which differs with age and is different between the two sexes (Siri 1956; Siri 1961). Siri's equation (%fat $=\left[\frac{4.95}{d}-4.50\right]\times 100$, 'd'= body density; Siri 1956) is mostly used to calculate the fat percentage from body density in adults (Jebb & Elia 1993). In children, different equations to convert density to fatness are necessary because of the lower density of FFM (Reilly 1998). By determining the amount of a body constituent like water (using isotope dilution as Deuterium oxygen, Tritium, or ^{18}Oxygen), Potassium (by assay of ^{40}Potassium) or Nitrogen (by neutron activation), the FFM can be calculated using these basic assumptions (Forbes 1999). These assumptions ignore inter-individual variability in the composition of FFM (Wells et al 1999). This is more important in infants and children since their body is not 'chemically mature', i.e. the relative constancy of FFM composition develops over time (Reilly 1998). It is well known that the young organism has a higher water, lower ash content, and lower density than the adult (Forbes 1999) and that inter-individual developmental differences might create huge differences in the composition of FFM. This is the major limitation in the use of the 2-component model in children (Wells et al 1999). Due to above mentioned limitations, most authors have attempted to circumvent the basic problem of age and sex specific differences in composition of FFM by using of age sex specific constants throughout childhood.

2.1.12-c. The multi-component approach

This approach is more recent and theoretically is more accurate since it attempts to allow for variation in composition of FFM. The 3-component model divides the body into fat, water, and the remaining fat-free dry mass, which is assumed to have a constant ratio of protein to mineral (Wells et al 1999). The advantage of this model over the 2-component model is that it avoids the assumption that the water content of FFM is constant between individuals of a given age and sex, and can provide a very accurate measure of body composition (Wells et al 1999).

The 4-component model of body composition, obtained by combining several measurement techniques, divides body weight into fat, water, mineral, and protein, and allows evaluation of several assumed constant relations that are central to 2-component models. These assumed constants include the water content, bone mineral content, and density of FFM (Wells et al 1999). However, the ratio of bone mineral to total body mineral is still assumed to be constant. Although reference data exist for these constants in children from birth to 10 years of age, most values came from 6 month to 9 year old reference children (Wells et al 1999). Studies using these reference data are, therefore, unable to consider the potential error of using assumed constants (Wells et al 1999). The 4-component approach uses body weight, body volume (by underwater weighing or air displacement), total body water (by isotope dilution), and total body mineral content (using dual energy x-ray absorptiometry [DEXA] method; Wells et al 1999). In 4-component models, body fat (BF) can be calculated from the following formula and is technically complicated: BF = Body mass - (water + protein + bone minerals).

2.1.12-d. Validation of simpler body composition methods

There is a need for simpler methods of measuring body fatness in children, particularly for clinical practice and epidemiology (Cole et al 1995; Dietz 1998a; Bellizzi & Dietz 1999). However, the lack of a gold standard for measurements of body composition in children makes evaluation of simple methods difficult (Wells et al 1999). Some of the methods based on 2-component models, though they have limitations because of variations in the composition of fat-free mass, have acceptable accuracy as long as they are chosen with care (Reilly 1998). For

example, using a 4-component model as a reference method in one recent study showed that isotope dilution and DEXA were acceptable 2-component models for 8-12 years children (Wang et al 1998).

2.1.12-e. Problems with body composition methods

Paediatric obesity assessment methods for clinical practice and epidemiology need to be practical, reliable and accurate. Many classical methods are impractical, unreliable, or inaccurate. For example, hydrodensitometry (under-water weighing), though it has been regarded as a reference method for body fat determination in adults, does not appear to be suitable for use in young children on practical as well as theoretical grounds (Reilly et al 1995; Wells et al 1999). The use of other methods like magnetic resonance imaging (MRI) and computed tomography (CT scan) is limited because of high cost and because the methods may not be valid. In addition, risk of ionizing radiation caused by computed tomography is considerable (Pietrobelli et al 1998). Dual energy x-ray absorptiometry (DEXA) is another method for estimating whole as well as regional body fat mass in children and adolescents but with minimum radiation exposure (Pietrobelli et al 1998). Use of DEXA for estimating body fat is supported by several studies, but the errors of this method are still of concern especially when it is used on its own (Wang et al 1998) and not as one element of a multi-component model.

Tetrapolar whole body impedance (BI) is a suitable method for estimating body composition in children (Reilly et al 1996) and for epidemiological use. For example, impedance is now used routinely in National Health and Nutrition Examination Surveys (NHANES; NCHS 1994; Troiano et al 1995; Freedman et al 1997; Troiano & Flegal 1998) and the European Prospective Investigation in Cancer and Nutrition (EPIC; Klipstein-Grobusch et al 1998; Samaras et al 1998; Day et al 2001) epidemiological studies. Since users of BI technically were confronted with growing choices of equations to estimate the percentage of fat free mass (FFM), the ability of BI in predicting FFM in children was not clear (Reilly et all 1996). In a study of prepubertal children in the UK, the accuracy of 4 different equation formula (Cordain et al 1988, Deurenberg et al 1991, Houtkooper et al 1992, and Schaefer et al 1994) were tested to predict

FFM in children against a 2-component reference method; and the equation of Houtkooper (Houtkooper et al 1992) was found to be capable of predicting FFM and fatness with negligible bias (Reilly et al 1996). In addition, the accuracy of impedance equations in predicting body fatness is highly population specific (Reilly 1998). In the absence of impedance prediction equations validated against multi-component models in UK children, the impedance method with the Houtkooper et al (1992) formula was used to estimate BF% in this thesis (Chapter 5).

2.1.13. Social class / socioeconomic status

A number of different methods of assessing social class / socioeconomic status have been used in the UK. The most widely used and accepted method is based on the Registrar General classification of occupations (Office of Population Censuses and Surveys; OPCS 1990). For example, this approach was used by Wilson et al (1998). This has limitations, particularly at a time when unemployment is high (which can lead to a large proportion of 'unclassified' families). Another problem is that assessment has usually been based on the occupation of the 'head of the household' (Danesh et al 1999) i.e. the father. In many modern families the biological father is not present. There is therefore an argument for basing social class assessment on the occupation of the mother. In this thesis the standard classification of occupations (OPCS 1990) has been used, but its limitations are acknowledged. Maternal / paternal (or both) social class is specified when appropriate. An alternative approach is to use geographically defined (e.g. post code based) assessment (Danesh et al 1999). This was not possible in ALSPAC because it might have breached subject confidentiality and anonymity. In any case, post-code based assessment methods can also be misleading (Ben-Shlomo & Smith 1999). In many countries no reasonable assessment of social class can be made. For example the study of breast-feeding and obesity by von Kries et al (1999) was unable to assess social class of the families.

2.1.14. Variables from ALSPAC used in studies at a glance

All the variables which have been used in this thesis (in addition to weight, height, body impedance, and the diets which have been explained earlier), have been explained in the appropriate Chapter. However a list of the used variables are shown in Table 2.12.

Table 2.12. List of some ALSPAC study variables which are used in this thesis.

Variable	When Measured	How Measured	Justification (details in Chapter 7)	Validity	Alternatives	Reference or Ideal
Gestational age	At birth	LMP - Expected pregnancy date	Patterson et al (1997) US study	Very good	Self-report	The study method
Catch-up growth	At age 2 years	A gain of ≥0.67 in weight SDS at 0-2 year	Eriksson et al (1999) Finish, Yajnik (2000) Indian, and Ong et al (2000) UK studies	Very good	Check the BMI SDS changes at different ages	The study method
Birth weight	At birth	From birth certificate	2 British, 2 German, 2 French, a Swedish, a Japanese, a Danish, and an Australian studies	Very good	Parental self-report	The study method
Ever breast-fed or not	At age 6 months	Self-report	Lucas et al (1980 & 81) French, von Kries et al (1999) German studies	Acceptable	1.Check every month from birth 2. Intervention	Check feeding status every month
Time of solids introduction	At age 6 months	Self-report	Wilson et al (1998) British study	Acceptable	1.Check every month from birth 2. Intervention	Check feeding practice every month
Parental obesity	Before pregnancy	BMI >30 (self-report height and weight)	A large variety of worldwide studies (refer to Chapter 7)	Acceptable	Underwater weighing, DEXA, and several other methods	Measure BMI
Parental education	Before pregnancy	Self-report as one of the 5 groups	Strauss & Knight (1999) US, Patterson et al (1997) US, and Moussa et al (1994) UAEmirates	Good	None	The study method
Parental social class	Before pregnancy	Self-reported defined by OPS	A large variety of worldwide studies (refer to Chapter 7)	Good	Post-code method	None, maybe by post-code
Ethnicity	Before pregnancy	Self-report	Three US, and 2 French studies	Good	None	The study method
Season of birth	At birth	Birth date recorded	Phillips & Young (2000) UK	Very good	None	The study method
TV viewing	At age 38 months	Self-report	A large variety of worldwide studies (refer to Chapter 7)	Acceptable	Direct observations, or set top devices	Intervention, set-top device, direct observation
Sitting in car	At age 38 months	Self-report		Acceptable		
Consumption of fizzy drinks	At age 38 months	Self-report	Dennison et al (1997), and Ludwig et al (2001) US studies	Acceptable	1. Measuring drinks 2. Intervention	Measuring fruit or sweetened / diet drinks
Sleeping duration / regularity	At ages 30 & 42 months	Time to bed - wake up time Self-report	O'Callaghan et al (1997) Italian, Locard et al (1992) French, and Kagamimori et al (1999) Japanese	Good	Measuring sleep duration	Measuring sleep duration

*DEXA = dual energy x-ray absorptiometry

2.1.15. Variables not measured / poorly measured in ALSPAC

2.1.15-a. Physical activity

Physical activity, which might have a major role in the development of childhood obesity (Moore et al 1995; Westerterp & Goran 1997; Dietz 1998a; Goran et al 1998a; Maffeis et al 1998), was not measured in the ALSPAC study children largely on the grounds that measurement techniques for children were impractical or of unknown accuracy at the time. Physical activity (inactivity in this thesis) was only defined as parental self-report of time spent sitting in car and TV viewing. The doubly labeled water method is described as the most accurate method for measuring physical activity, and movement recorders (accelerometers and pedometers or foot-strikes) as the second most accurate method (Goran 1997; Maffeis et al 1997; Goran & Sun 1998; Going et al 1999). Physical inactivity, such as TV watching duration and the amount of time spent in car, was not measured quantitatively in the ALSPAC study children but this seems to be one of the most important issues (DuRant et al 1994; Gortmaker et al 1996) in the risk of childhood obesity and potentially in timing of adiposity rebound.

2.1.15-b. Energy expenditure

Obesity is an imbalance of energy intake and energy expenditure (Whitaker & Dietz 1998). It is known that energy expenditure can determine energy balance in children (Goran & Sun 1998). Energy expenditure might also influence the timing of AR (Roville-Sausse 1999). Therefore, ideally we should have measured both energy intake and energy expenditure with a highly accurate and precise method (e.g. total energy expenditure using the doubly labeled water technique; Schoeller 1999). Energy expenditure was not measured since the available methods were time consuming and very expensive and not appropriate in an epidemiological study (deBruin et al 1996).

2.1.15-c. Age groups

Since the present thesis was based on the ALSPAC, HSE, and Iranian studies (Chapters 3 to 7) and was based on secondary analysis, studies were restricted to the age groups available. A more wide ranging analysis (e.g. to adolescence) was not possible.

2.1.15-d. Other environmental variables

It was not possible to include all of the potentially important (environmental) variables such as parental neglect, the mean time parents usually spend with child, family income, and the amount of space inside the house in the ALSPAC protocol.

2.1.16. Variables measured with limitations

Dietary intake

It is not clear weather any of the dietary assessment methods have sufficient accuracy or precision to detect the small energy imbalance which can cause obesity in individuals (Livingstone et al 1990; Livingstone et al 1992; Kaskoun et al 1994; Johnson et al 1996; Lindquist et al 2000; Robson & Livingstone 2000). However, as discussed in Section 3.2.2, the 3-day prospective household measures record for assessment of dietary energy and macronutrient intake which was used in this thesis was well accepted by subjects.

2.1.17. Missing values / representativeness

For some of the variables used in this thesis, the values for some cases were missing. For each individual variable, the percentage of missing values and the possible reasons were

explained in the relevant Chapters. Comparisons of the ALSPAC cohort with UK (NDNS 1995), and Census (1991) data for social class were presented earlier in Tables 2.2 and 2.3. These showed a tendency for the ALSPAC cohort to contain relatively affluent families.

Parental obesity

Heights and weights of ALSPAC study parents were self-reported in 1991/92 (based on questionnaire at week 12 of pregnancy). These were used to calculate BMI and obesity was defined as having a BMI >30 kg/m^2 (for both mothers and/or fathers). The proportion found to be obese was low (7.3% men and 5.2% women; Chapter 7), compared to other British studies conducted at around the same time. Jebb (1999) reported prevalence of obesity (defined as having a BMI >30 kg/m^2) as 8.0 & 13.0% in men and 12.0 & 16.0% in women in 1987 & 1994 though this is age dependent (White et al 1995) and the ALSPAC parents were at the younger end of the age spectrum. The lower prevalence of obesity in 'Children in Focus' parents probably reflects the over-representation in this group of wealthier families (as obesity prevalence is higher in lower social class particularly for women) as described in Chapter 7.

2.1.18. Further information on ALSPAC

The aims and design of the ALSPAC study and CIF have been described in more detail elsewhere (Golding 1990; Children in Focus 1997; Golding et al 2001).

2.2. Health Survey for England (HSE)

2.2.1. Introduction to HSE

The Health Survey for England (HSE) comprises a series of annual surveys, of which the 1997 survey was the seventh (HSE 1998). Since 1994, the HSE has been carried out by the Joint Health Surveys Unit of the National Centre for Social Research and the Department of Epidemiology and Public Health at University College, London (UCL; HSE 1998). Since 1995 the surveys have included children aged 2-15 years as well as adults. This thesis was concerned solely with the prevalence of obesity in children and adolescents aged 6-15 years, and has used data from the 1996 survey. Each survey in the Health Survey series consisted of core questions and measurements (for example, anthropometric and blood pressure measurements and analysis of blood and saliva samples), in addition to modules of questions on specific issues that changed periodically. The surveys involved nurse visits as well as interviews.

2.2.2. Survey design and sampling

From 1996, surveys used a stratified multi-stage cluster design of the population in private households in England using the Postcode Address File as a frame. The 1996 survey (data used in Chapter 3 of this thesis) also included children aged 2-15 years living in selected households. The survey had two separate elements: an interviewer visit and a nurse visit. An advance letter was sent to each address explaining briefly the survey and its purpose. Two other information leaflets given out by the interviewer and the nurse provide the respondent with greater detail. The parents / guardian of 2-12 year olds were interviewed about the child. At the end of the interview, all respondents were asked to have their height and weight measured. A limited amount of proxy information was obtained, where possible, about those unwilling or unable to take part in the survey. The 1996 survey aimed to obtain information from around 17,000 adults and 3600 children.

2.2.3. Aims of HSE

The aims of the Health Survey series are:

1) To provide annual data about the nation's health;

2) To estimate the proportion with specified health conditions;

3) To estimate the prevalence of risk factors associated with these conditions;

4) To examine differences between population subgroups;

5) To assess the frequency with which combinations of risk factors occur;

6) To monitor progress towards two Health of the Nation targets relating to blood pressure and obesity;

7) Since 1995, to measure the height of children at different ages, replacing the National Study of Health and Growth.

2.2.4. Study measurements

2.2.4-a. Height

Measurements of height to 0.1cm using a portable stadiometer were made from age 6 years onward. The measurer had a vertical board with an attached metric rule and a horizontal headboard which the movable headboard brought onto the superior point of the head with pressure to compress the hair. Subjects were measured wearing little clothing and were barefoot or wearing thin socks (HSE 1998).

2.2.4-b. Weight

Weight, to 0.5kg (using Soehnle portable scales), was measured in children at each age. Each subject's weight was measured when standing still over the centre of the platform with light indoor clothing and no shoes (HSE 1998).

2.2.4-c. Age

The ages of children in the survey were presented as whole integer age groups. That is, the actual values were rounded to the nearest whole number. For example, the '6 year old' children in the survey consisted of all children from 5.50 to 6.49 years old (HSE 1998). Exact age data were not available. The implications of this are discussed in Chapter 3.

2.3. Iranian study

2.3.1. Introduction to Iranian surveys

Background: Iran and the National Health Survey

The geographical situation of Iran and the position of the provinces in addition to their climate and other relevant information are described in Chapter 4.

2.3.2. Sampling in Iranian study

At the time of this thesis, national surveys of BMI in Iranian children were not available. In a survey in 1995 in two different Iranian provinces, Gilan in the North and Sistan in the South, a sample of 4351 2-5 year old children (more than 1% of all 2-5 year old children in the 2 provinces) were chosen. The survey was designed to provide a random sample of >4000 2-5 year old children in the two provinces in 1995 (at least 1% of the total population of around 400,000 2-5 year olds in the 2 provinces), and to be representative of both urban and rural areas in these predominantly rural provinces. Iranian primary health care is organised as an active system: children of pre-school age attend community health centres, usually on a monthly basis (for vaccinations, feeding and health advice). Families who do not attend regularly are followed-up by home visits. Children in the present study were selected by random sampling until a final sample size of 4315 was obtained.

2.3.3. Study measurements

2.3.3-a. Height

Measurements of height to 0.5 cm using hand made wooden height measurer scales for 2 year olds, and Seca height scales for 3-5 year olds were made by trained staff. The measurement device was a vertical board with an attached metric rule and a horizontal headboard that could be brought into contact with the most superior point on the head. Subjects were measured wearing little clothing and thin socks.

2.3.3-b. Weight

Weight was measured to 0.5kg using Seca scales. Measurement was done when standing still over the centre of the platform with light indoor clothing and no shoes.

2.3.3-c. Age

Children were defined as 4 different age groups from 2 to 5 years. Each age group took in a 6 months period either side of the specified age rounded to the nearest integer value. For example, age group '2' consisted of all children aged 1.50 to 2.49 years old.

3. Chapter 3: Prevalence of overweight and obesity in British children

3.1. Introduction

Obesity is an important issue in both childhood and adulthood because of its adverse consequences to individuals including medical and psychosocial disorders and the public health and economic costs as described in Chapter 1. Obesity in early life is associated with respiratory conditions and several risk factors for coronary heart disease and is predictive of many severe medical disorders that are important causes of mortality / morbidity in adulthood (Anonymous 1997; Dietz 1997a; Freedman et al 1997; Dietz 1998a). Obese children are also more likely to become obese adults than are non-obese children (Barlow & Dietz 1998). The children of overweight / obese parents have an increased risk of obesity (Whitaker et al 1997). Studies in the USA have shown that the prevalence of obesity in children and adults is increasing dramatically and that these trends are likely to continue (Kuczmarski 1993; Troiano et al 1995; Ogden et al 1997; Flegal et al 1998; Martorell et al 1998; Troiano & Flegal 1998; Troiano & Flegal 1999; Zephier et al 1999).

Data on obesity prevalence in both children and adults are necessary so that populations at high-risk of obesity (e.g. ethnic minority groups in the USA) can be identified, secular trends in obesity can be monitored, and obesity prevention programs can be introduced and assessed. An ideal obesity definition would have biological / clinical meaning and needs to be both accurate and acceptable to the child. Classifying children as overweight or obese based on mortality is problematic since there is unlikely to be a clear relationship between childhood overweight / obesity and short-term mortality, unlike in adults (Troiano & Flegal

1998). It has been agreed that in the absence of mortality based criteria for children, a statistical approach offers the most practical choice for definition of overweight / obesity based on BMI (Cole et al 1995; WHO 1995; Power et al 1997; Barlow & Dietz 1998; Troiano & Flegal 1998; Dietz & Bellizzi 1999).

BMI is now broadly accepted as an index of fatness in a person; the index that is given by BMI is independent of height and is applicable to both sexes (Prentice 1998). A few individuals who are muscular might be classified as overweight or obese but BMI is generally considered to provide a useful index for large epidemiological studies and clinical practice (Prentice 1998). Low sensitivity of BMI is a problem (although this is not widely acknowledged) and is described in Chapter 5. A secular trend of increasing *fatness* in British children has been shown (Hughes et al 1997), but its significance for the childhood obesity issue was not understood until recently due to the lack of an agreed obesity definition. An expert committee in the USA recently recommended BMI $\geq 95^{th}$ and $\geq 85^{th}$ percentiles (compared with population reference data) as obese and overweight respectively (Barlow & Dietz 1998). Such definitions have already been used by many investigators and were becoming standard definitions in the USA (Robinson 1993; Himes & Dietz 1994; Gortmaker et al 1996; Freedman et al 1997; Whitaker et al 1997; Slyper 1998; Troiano & Flegal 1998; Freedman et al 1999; Troiano & Flegal 1999; Zephier et al 1999). Obesity prevalence estimations based on such definitions might have been limited (Barlow & Dietz 1998) because BMI can have low sensitivity, as described in Chapter 5.

As noted above, BMI must be interpreted relative to population reference data (Ogden et al 1997; Barlow & Dietz 1998). Overweight / obesity is therefore defined relative to specific percentile of the reference population based on age, sex, race and/or other group

characteristics (Troiano & Flegal 1998). Choosing a specific percentile means that the proportion of the reference population that exceeds the cut-off is overweight and that the prevalence of overweight for all different sex, age and/or other characteristics groups are similar (Troiano & Flegal 1998). UK 1990 reference data for BMI were created using data combined from eleven UK surveys conducted between 1978-1990 and covering 15636 boys and 14899 girls (Cole et al 1995). The centiles were fitted using Cole's LMS method, which adjusts the body mass index distribution for differing degrees of skewness at different ages (Cole & Green 1992). These centiles are less affected by differences in the timing of puberty than simply height and weight (Prentice 1998). For epidemiologists monitoring change in paediatric obesity over time (secular trends) there is a problem if growth curves are regularly updated to account for secular changes, as 15% of the population will always be above the 85th centile (Prentice 1998). Therefore it is necessary to identify a reference data-set collected at a specified time so that secular trends can be measured (Prentice 1998). The UK 1990 BMI reference data have been recommended for this purpose in the UK (Prentice 1998; RCPCH 2001; Wright et al 2002), as noted in Chapter 2.

Although there is a large amount of data on overweight / obesity prevalence in the USA, in the UK there were no estimates of childhood obesity prevalence at the time when research for this thesis began in 1998 because:

I- There was a lack of agreement on a simple acceptable anthropometric method to define obesity. It has only recently been agreed to use BMI as an obesity definition (Barlow & Dietz 1998). For example, investigators who carried out the Health Survey for England (HSE) 1996 did not try to define obesity prevalence in English children since they believed there were no accepted measures to define paediatric obesity (HSE

1998). Table 3.4 (prevalence of obesity in different countries) also shows several studies carried out recently which used a variety of different obesity definitions.

II- The precise definition (cut-offs) for BMI to define obesity had not been agreed until recently when a consensus / expert committee on paediatric obesity of the Department of Health and Human Services, the Maternal and Child Health Bureau, Health Resources and Services Administration and American Academy of Pediatrics (USA) reached the conclusions that children with a BMI $\geq 95^{th}$ percentile should be defined as obese (Barlow & Dietz 1998). The arguments for BMI $\geq 95^{th}$ centile as a useful obesity definition can be restated briefly as follows:

-Such children are highly likely to be excessively fat (Pietrobelli et al 1998; Reilly et al 1999b).

-Such children are likely to suffer from the adverse clinical consequences of obesity (Barlow & Dietz 1998; Dietz 1998a).

-These children have a high probability of remaining obese (Barlow & Dietz 1998; Dietz 1998a).

III- Until recently, there has been lack of reliable national data on which to estimate prevalence of overweight / obesity.

3.2. Aims

The aims of this Chapter are to estimate the prevalence of overweight / obesity based on two recent studies:

Study 1- A childhood cohort broadly representative of the UK, the Avon Longitudinal Study of Parents and Children (ALSPAC) which is described in Chapter 2.

Study 2- The Health Survey for England (HSE) which measured BMI in a sample of children representative of England (HSE 1998).

3.3. Subjects and methods

3.3.1. Study 1

Subjects consisted of the 'Children in Focus' birth cohort, randomly selected from a larger geographically defined total population cohort born in the Bristol-Avon area in 1991-92 as described in Chapter 2. The cohort, named the Avon Longitudinal Study of Parents and Children (ALSPAC), was described in Chapter 2 and is broadly representative of the UK, but wealthier families are slightly over-represented and ethnic minority families slightly under-represented (ALSPAC homepage). These slight differences between the ALSPAC sample and the UK may or may not be important because differences in childhood obesity prevalence between different socioeconomic and ethnic groups were not clear at the time of the present study. Measurements of height to 0.1cm (using the Harpenden Neonatometer at 4 months, Kiddimetre for 8 to 25 and Leicester Height Measure for other ages) and weight when wearing underwear to 0.1kg (using Fereday 100kg combined scale for 4 months, Soenhle scale or Seca scale mode 724 for 8 months and SECA scales models 724 or 835 for other ages) were made in children at 4, 8, 12, 18, 25, 31, 37, 43, 49, and 61 months of age as described in Chapter 2.

3.3.2. Study 2

In this study, a 6-15 year old age group of children from a cross-sectional survey in 1996 was selected. The study, the Health Survey for England (HSE) 1996, as described in Chapter 2 provided a sample with a wide age range, which was representative of English children. Measurements of height to 0.1cm (using a portable stadiometer) and weight when wearing light indoor clothing to 0.5kg (using Soehnle portable scales) were made in children at each age. The ages of children in the survey were presented as whole integer age groups. That is,

the actual values were rounded to the nearest whole number. For example, the '6 year old' children in the survey consisted of all children 5.50 to 6.49 years old. Since the rounding process can have an effect on calculated BMI SDS values in younger children, we did not include the children under the age of 6 in the HSE 1996 in the present analysis.

3.3.3. Statistical analysis

In childhood, BMI must be interpreted relative to population reference data (Ogden et al 1997; Barlow & Dietz 1998) as discussed previously. In the present studies the BMI standard deviation score (SDS) relative to UK reference data (Child Growth Foundation, London) was therefore analysed using computer software provided by the Child Growth Foundation, London. The following cut-offs were used to define overweight and obesity (Barlow & Dietz 1998):

BMI SDS ≥ 1.04 ($\geq 85^{th}$ percentile) defined as overweight;

BMI SDS ≥ 1.64 ($\geq 95^{th}$ percentile) defined as obese.

Using such criteria, the expected prevalence of overweight and obesity are 15% and 5% respectively. The percentage of overweight / obese children observed in each age and sex group was defined as 'prevalence'.

Differences between percentage of overweight / obesity in different age and sex groups were tested using two probability Z-tests and Chi-squared tests. Overweight / obesity percentage in each age and sex group was compared with the expected prevalence given above, using Chi-squared goodness of fit tests to test the significance of differences.

3.4. Results

3.4.1. Study 1

Prevalence of obesity and overweight in children in different age groups in both sexes are shown in Table 3.1 for the ALSPAC study. Differences in prevalence of overweight and obesity between boys and girls were not statistically significant with the exception of overweight prevalence at 25 months where girls showed a higher prevalence than boys ($p<0.05$). Prevalence of obesity significantly exceeded 'expected' frequencies at all ages except at 4, 18, and 25 months (Chi-squared goodness of fit tests; $p<0.01$). Figure 3.3 shows that the prevalence of overweight / obesity in the ALSPAC cohort at many occasions (and not always) exceeded 'expected' percentage. Changes in obesity and overweight percentage between age groups compared with UK reference data showed that at age 4 month the mean BMI was lower than expected such that the percentage of overweight children was 10% (expected percentage 15%; Cole et al 1995) and this difference was statistically significant. The same result can be seen for obesity in this age group; expected percentage for obese children was 5% but only 2.2% of children were obese at age 4 months. At age 8 months, observed values significantly exceeded those expected such that there were 18.0% of overweight children and 6.5% obese. Overweight / obesity percentage again was higher than expected until age 12 months but at 18 and 25 months the percentage of obesity was almost as expected. From age 31 months onwards, obesity percentage remained significantly higher than expected. In summary, the excess above the 85th and 95th percentiles started to appear at about age 2.5 years in children in the ALSPAC children.

Table 3.1. Prevalence of obesity and overweight (%) in children at ages 4, 8, 12, 18, 25, 31, 37, 43, 49 and 61 months, study 1 (CIF children of ALSPAC study).

Age (months)		**Overweight** (SDS ≥1.04 or ≥85th centile) n (%)	**Obese** (SDS ≥1.64 or ≥95th centile) n (%)	**Total**
4	Boys	53 (9.8)	11 (2.0)	537
	Girls	47 (10.2)	11 (2.4)	459
	Total	÷ ¤ 100 (10.0)	÷ ¤ 22 (2.2)	996
8	Boys	124 (17.4)	47 (6.6)	714
	Girls	÷108 (18.2)	38 (6.4)	594
	Total	232 (17.7)	÷85 (6.5)	1308
12	Boys	122 (18.2)	37 (5.5)	671
	Girls	112 (19.8)	42 (7.4)	656
	Total	÷ ¥ 234 (18.9)	÷79 (6.4)	1236
18	Boys	85 (13.6)	25 (4.0)	626
	Girls	82 (15.6)	24 (4.6)	525
	Total	167 (14.5)	49 (4.3)	1151
25	Boys	78 (13.9)	33 (5.9)	562
	Girls	† 85 (18.1)	29 (6.2)	469
	Total	§ 163 (15.8)	62 (6.0)	1031
31	Boys	103 (17.5)	36 (6.1)	589
	Girls	101 (20.7)	38 (7.8)	487
	Total	÷204 (19.0)	÷74 (6.9)	1076
37	Boys	101 (17.9)	42 (7.4)	566
	Girls	102 (21.4)	38 (8.0)	477
	Total	÷203 (19.4)	÷80 (7.7)	1043
43	Boys	127 (22.0)	40 (6.9)	577
	Girls	107 (23.2)	42 (9.1)	462
	Total	÷234 (22.5)	÷82 (7.9)	1039
49	Boys	112 (19.9)	42 (7.5)	562
	Girls	94 (20.8)	35 (7.8)	451
	Total	÷206 (20.3)	÷77 (7.6)	1013
61	Boys	97 (18.2)	43 (8.1)	532
	Girls	85 (19.3)	27 (6.1)	440
	Total	÷182 (18.7)	÷70 (7.2)	972

÷ Significant difference between observed and expected frequencies ($p<0.01$).
¤ Significant difference between ages 4 and 12 months ($p<0.01$).
¥ Significant difference between ages 12 and 25 months ($p<0.05$).
§ Significant difference between ages 25 and 37 months ($p<0.05$).
† Significant difference between boys and girls ($p<0.05$).
There was an overweight / obesity trend within age groups (test for trend, $p<0.01$).

3.4.2. Study 2

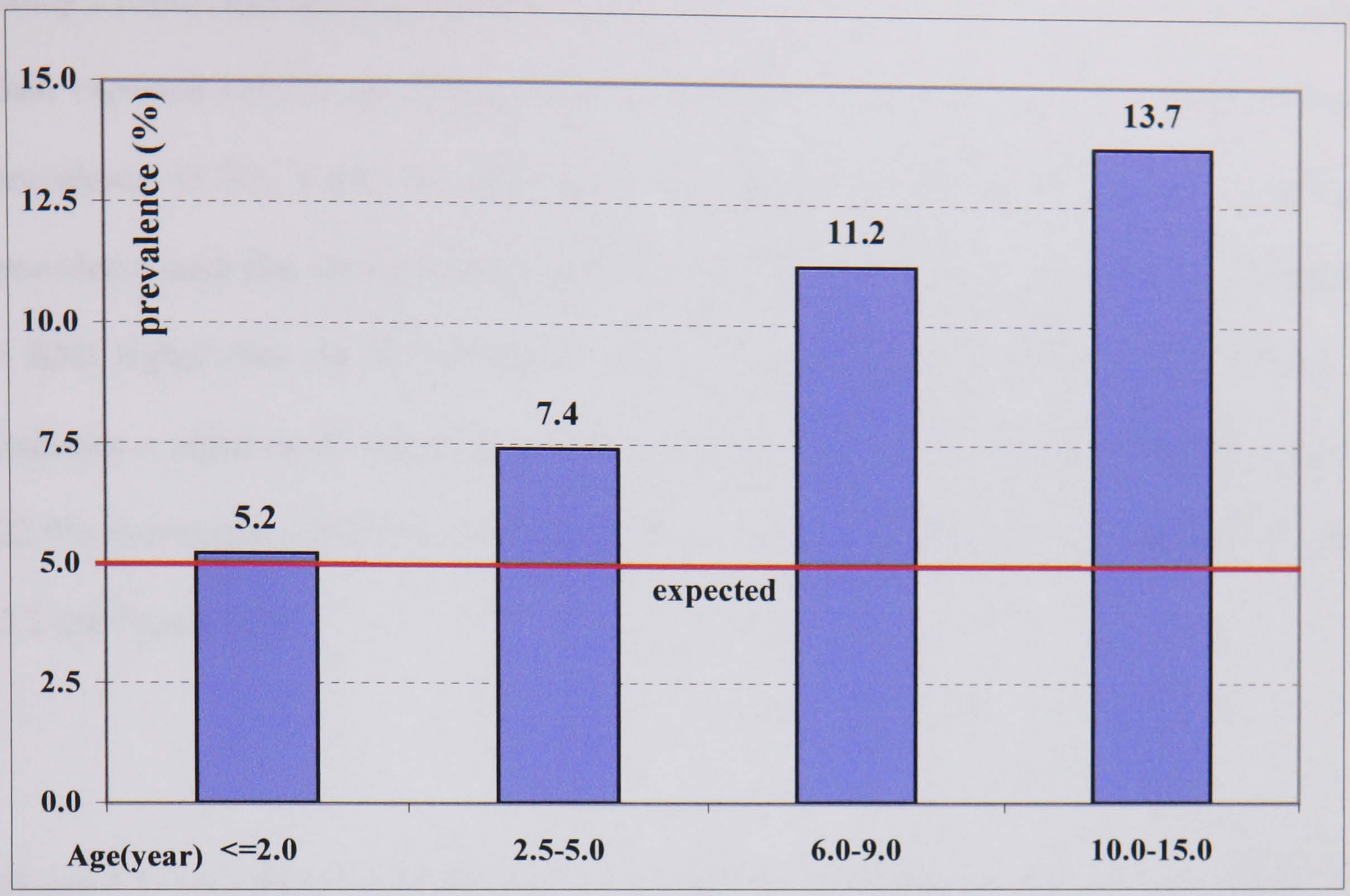
Prevalence of obesity / overweight in children in the HSE 1996 at different age groups in both sexes are shown in Table 3.2. Differences in prevalence of overweight and obesity between boys and girls were not statistically significant. Prevalence of obesity significantly exceeded 'expected' frequencies at all ages (Chi-squared goodness of fit, $p<0.01$). Prevalence of overweight also exceeded 'expected' percentages in all age groups (Figure 3.4).

Table 3.2. Prevalence of obesity and overweight (%) in children at ages 6 to 15 years old in study 2 (HSE 1996).

Age (year groups)		**Overweight** (SDS ≥1.04 or ≥85th centile) n (%)	**Obese** (SDS ≥1.64 or ≥95th centile) n (%)	**Total**
6	Boys	34 (22.1)	18 (11.7)	154
	Girls	31 (21.5)	13 (9.0)	144
	Total	65 (21.8)	31 (10.4)	298
7	Boys	37 (25.7)	13 (9.0)	144
	Girls	NS 24 (17.6)	16 (11.8)	136
	Total	61 (21.8)	29 (10.4)	280
8	Boys	33 (24.3)	16 (11.8)	136
	Girls	33 (20.7)	21 (13.2)	159
	Total	66 (22.4)	37 (12.5)	295
9	Boys	33 (25.2)	17 (13.0)	131
	Girls	NS 22 (19.0)	11 (9.5)	116
	Total	55 (22.3)	28 (11.3)	247
10	Boys	31 (23.0)	13 (9.6)	135
	Girls	28 (23.9)	13 (11.1)	117
	Total	59 (23.4)	26 (10.3)	252
11	Boys	31 (25.4)	17 (13.9)	122
	Girls	32 (23.9)	15 (11.2)	134
	Total	63 (24.6)	32 (12.5)	256
12	Boys	35 (27.8)	15 (11.9)	126
	Girls	34 (28.3)	22 (18.3)	120
	Total	69 (28.0)	37 (15.0)	246
13	Boys	33 (25.2)	16 (12.2)	131
	Girls	37 (28.0)	21 (15.9)	132
	Total	70 (26.6)	37 (14.1)	263
14	Boys	41 (30.1)	19 (14.0)	136
	Girls	33 (29.2)	15 (13.3)	113
	Total	74 (29.7)	34 (14.6)	249
15	Boys	46 (32.9)	23 (16.4)	140
	Girls	30 (28.8)	18 (17.3)	104
	Total	76 (31.1)	41 (16.8)	244

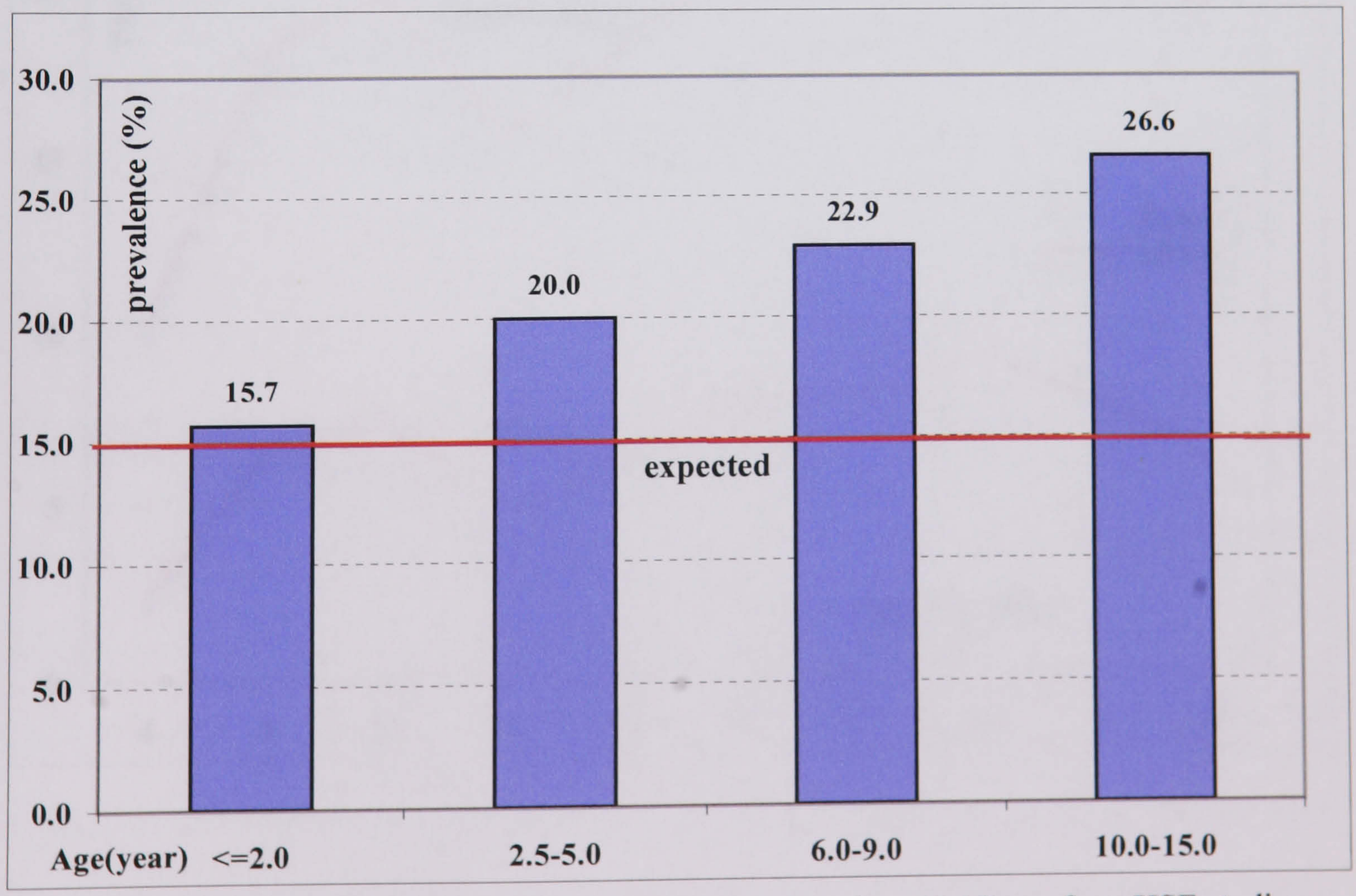
- There were significant differences between observed and expected values in all different ages ($p<0.01$) except overweight girls at ages 7 and 9 years (NS = not significant).
- There was no significant difference between the two sexes.
- There was an overweight / obesity trend within age groups (test for trend, $p<0.01$).

Figure 3.1. Prevalence of obesity (BMI ≥95th percentile) at different ages (note: studies 1 and 2 are combined here).



The 1st and 2nd bars (up to age 5) are from ALSPAC and the others (6-15) are from HSE studies.

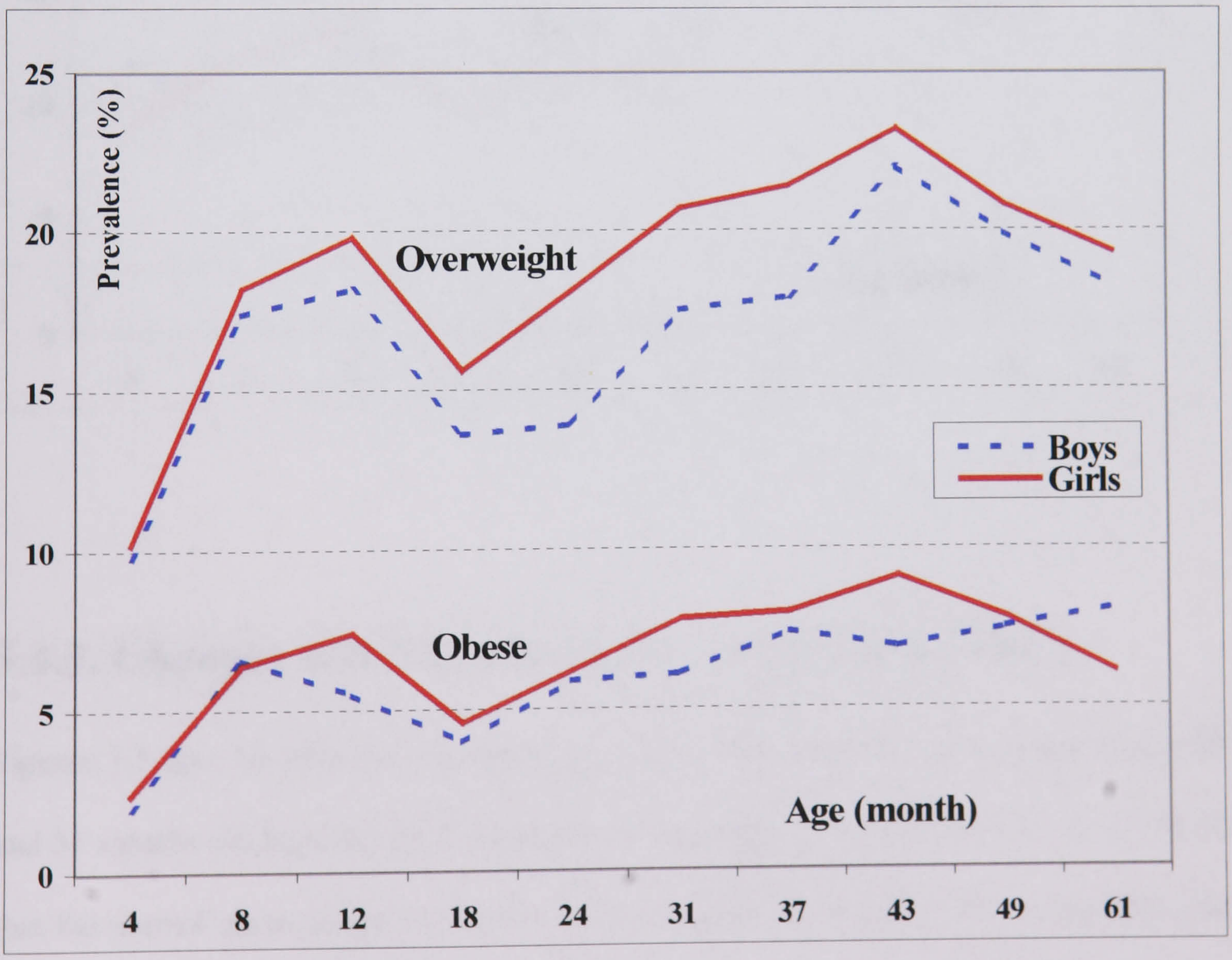
Figure 3.2. Prevalence of overweight (BMI ≥85th percentile) at different ages (note: studies 1 and 2 are combined here).



The 1st and 2nd bars (up to age 5) are from ALSPAC and the others (6-15) are from HSE studies.

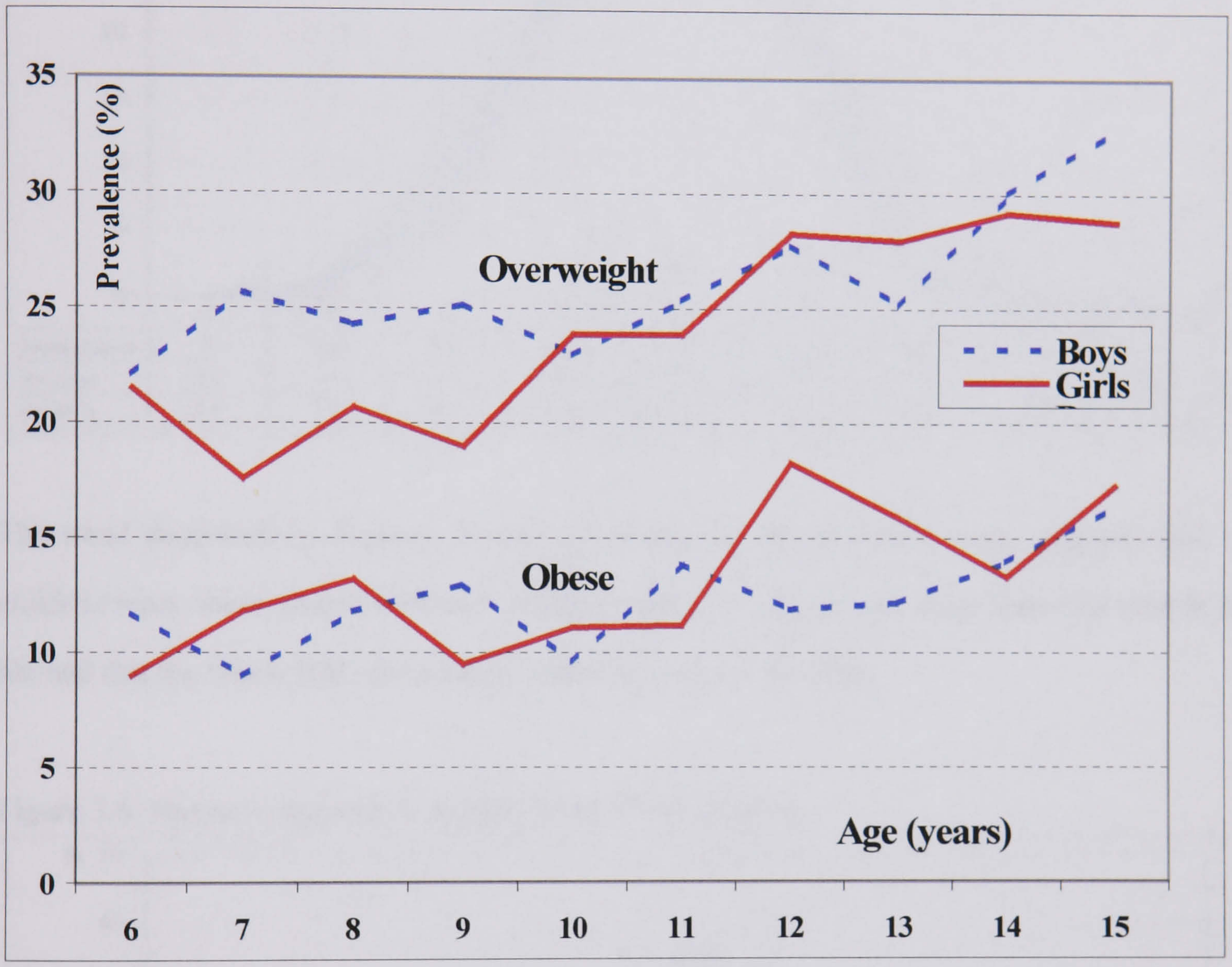
Study 2 results indicate that the obesity percentage in 6-9 years old children was much higher than expected (11.2% vs. 5.0%) and even higher in the 10-15 year old children (obesity prevalence 13.7%; Table 3.1 and Figure 3.1). Similar results were found for overweight prevalence such that the percentage of those 2.5-5.0 year old children from study 1 who had a BMI higher than the 85th percentile was 20.0% (expected percentage 15.0%). Study 2 indicates a significantly higher than expected percentage of overweight in British children: 22.9% overweight at 6.0-9.0 years and 26.6% at 10.0-15.0 years old were overweight (Table 3.2 and Figure 3.2).

Figure 3.3. Prevalence of obesity and overweight (%) in children at ages 4, 8, 12, 18, 25, 31, 37, 43, 49 and 61 months of study 1 (ALSPAC).



The percentage of overweight / obesity increased significantly with age (test for trend, $p<0.01$; Figure 3.4) for both ALSPAC and HSE.

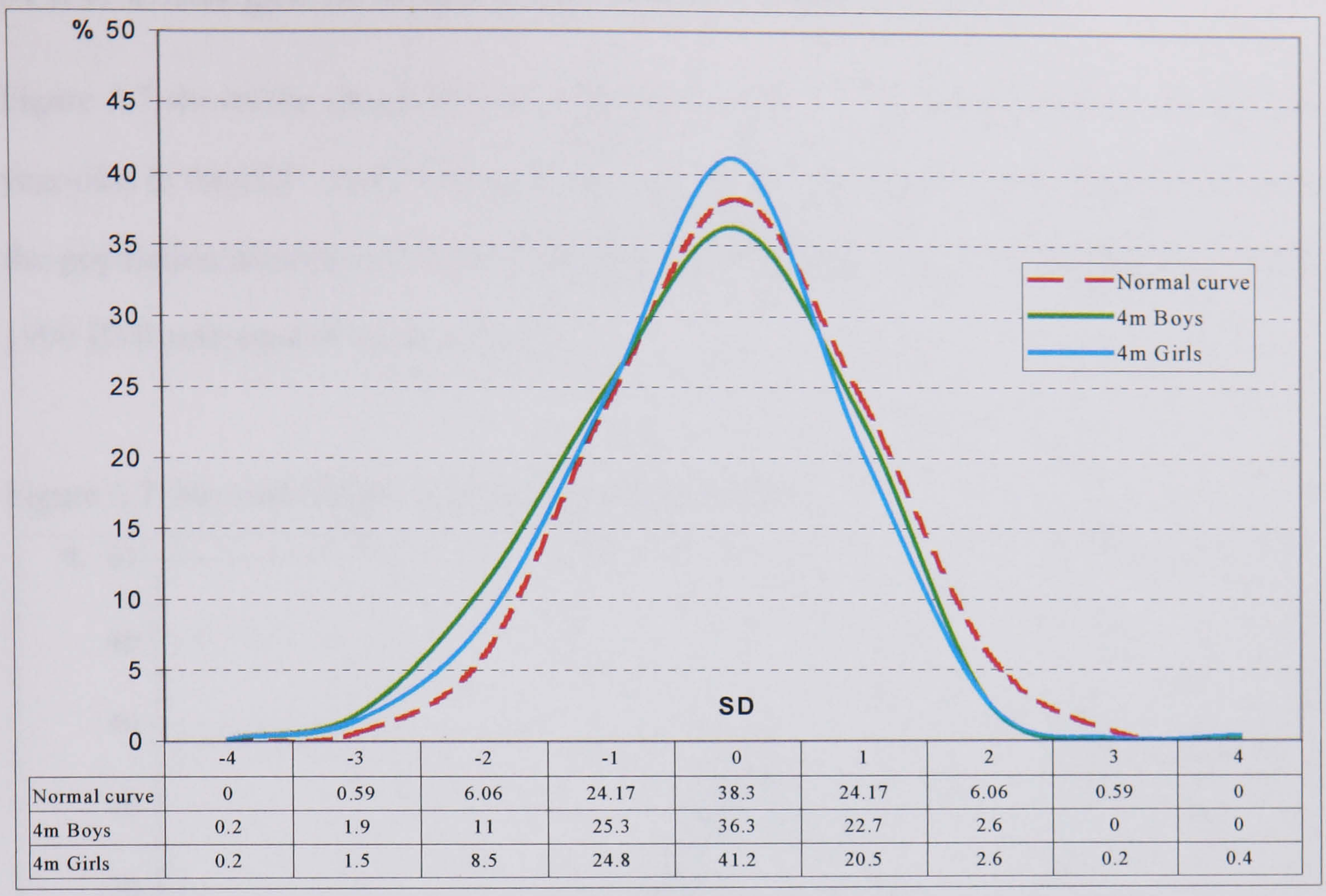
Figure 3.4. Prevalence of obesity and overweight (%) in children at ages 6 to 15 years in study 2 (HSE 1996).



3.4.3. Changes in BMI distribution: study 1 (ALSPAC)

Figures 3.5 and 3.6 show the curves for the BMI SDS frequency distribution drawn for 4, and 31 months old boys and girls compared to the idealised normal curve. It should be noted that the normal curve would be expected since the BMI SDS values were calculated relative to the Cole UK 1990 reference (Cole et al 1995) which was transformed to produce a normal distribution.

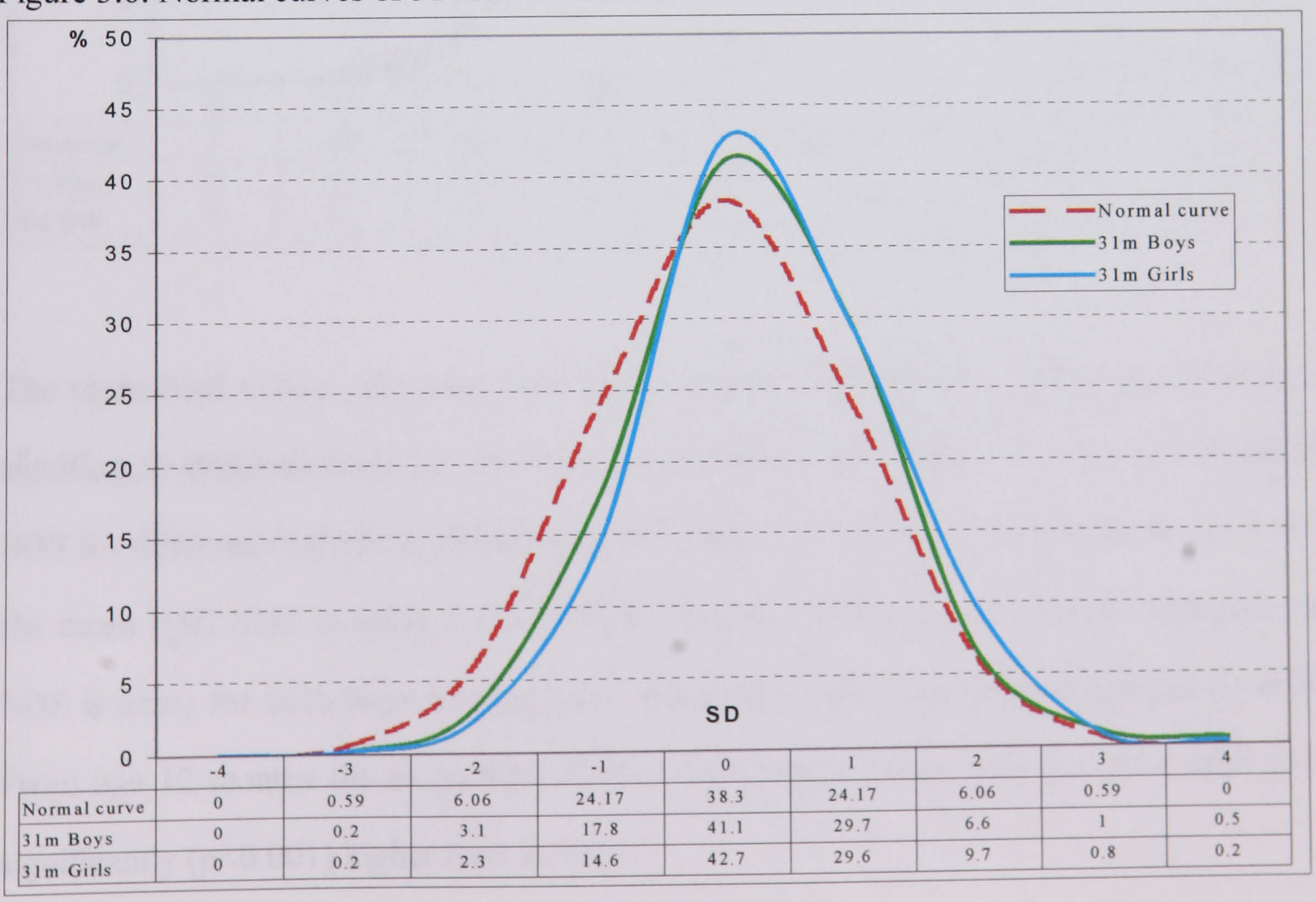
Figure 3.5. Normal curves of 4 month old ALSPAC children.



	-4	-3	-2	-1	0	1	2	3	4
Normal curve	0	0.59	6.06	24.17	38.3	24.17	6.06	0.59	0
4m Boys	0.2	1.9	11	25.3	36.3	22.7	2.6	0	0
4m Girls	0.2	1.5	8.5	24.8	41.2	20.5	2.6	0.2	0.4

The trend described by Figures 3.5 and 3.6 based on the ALSPAC study, suggests that few children were obese around birth but children started to gain weight more than expected in later life and that the whole BMI distribution tended to move to the right.

Figure 3.6. Normal curves of 31 month old ALSPAC children.

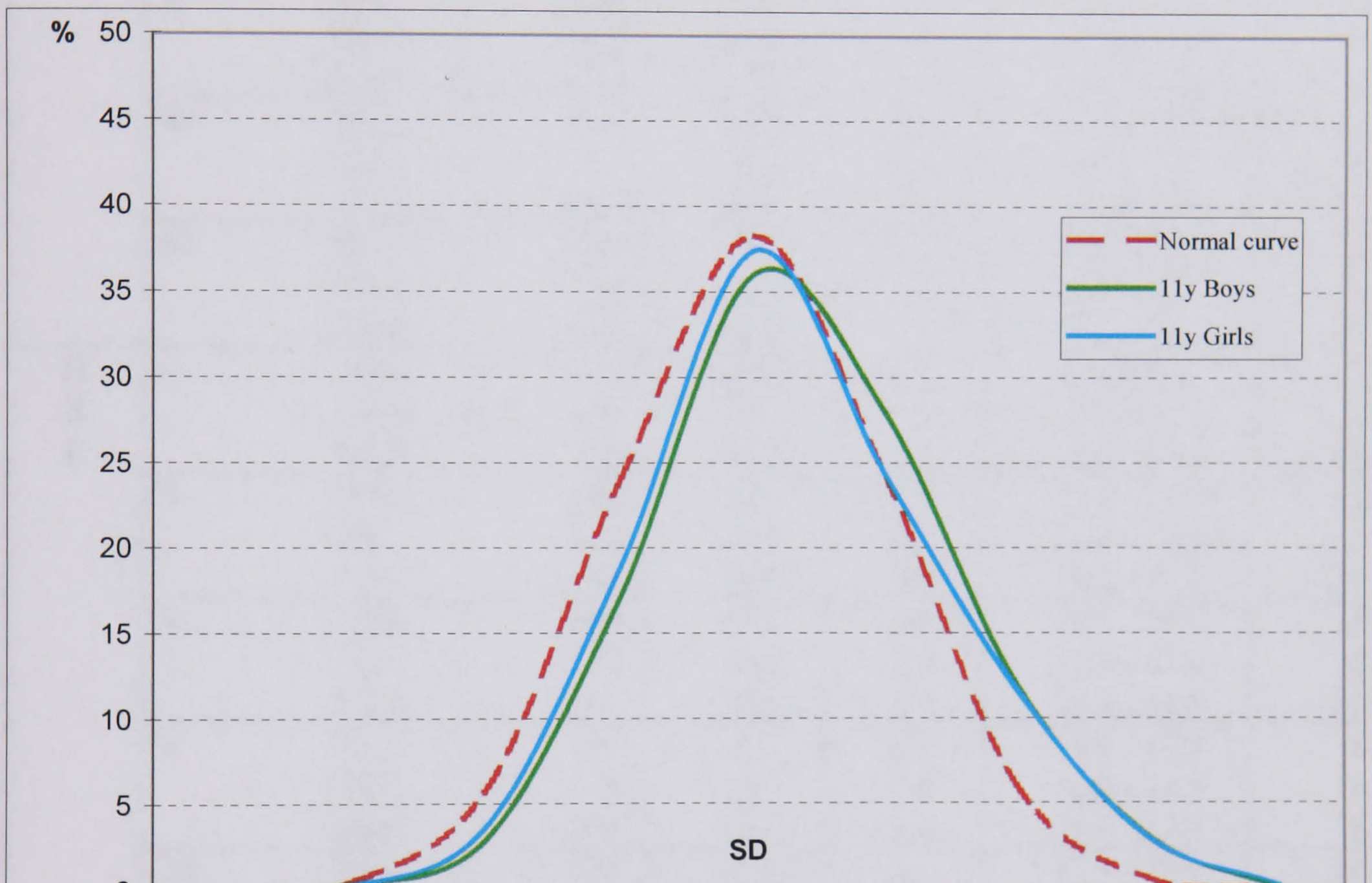


	-4	-3	-2	-1	0	1	2	3	4
Normal curve	0	0.59	6.06	24.17	38.3	24.17	6.06	0.59	0
31m Boys	0	0.2	3.1	17.8	41.1	29.7	6.6	1	0.5
31m Girls	0	0.2	2.3	14.6	42.7	29.6	9.7	0.8	0.2

3.4.4. Changes in BMI distribution: study 2 (HSE)

Figure 3.7 shows the distribution of BMI compared to an idealised normal curve for the 15-year-olds in the HSE study. The pattern would have been similar at any age and indicates that the population distribution of BMI has shifted to the right since the construction of the UK 1990 BMI reference (Cole et al 1995).

Figure 3.7. Normal curves of 15 year old HSE children.



	-4	-3	-2	-1	0	1	2	3	4
Normal curve	0	0.59	6.06	24.17	38.3	24.17	6.06	0.59	0
11y Boys	0.1	0.4	3.1	17.3	36.1	27.9	12	3.1	0
11y Girls	0.1	0.3	3.9	19.1	37.5	24.3	11.7	3	0.2

The mean BMI values calculated from the subjects of both studies 1 and 2 were consistently significantly different from the reference mean BMI values (Table 3.3). The calculated BMI SDS for different study subjects are shown in Table 3.4. The results show that at age 4 month the mean BMI SDS in study 1 (ALSPAC) children was less than expected (expected mean SDS is zero) for both boys and girls and that these differences were significant ($p<0.001$). From age 12 months the mean BMI SDS in both study 1 and study 2 (HSE) children was significantly ($p<0.001$) higher than expected.

Table 3.3. BMI values for UK children derived from studies 1 and 2.

Study	Age (months in ALSPAC years in HSE)		n	BMI-value Mean	SD	95% CI	UK 1990 Mean ¤
ALSPAC	4	Boys	537	17.0	1.4	16.9 - 17.1	17.3
		Girls	459	16.6	1.4	16.5 - 16.7	16.7
		Total	996	16.8	1.4	16.7 - 16.9	17.0
	12	Boys	671	17.9	1.4	17.8 - 18.0	17.6
		Girls	565	17.6	1.4	17.5 - 17.8	17.2
		Total	1236	17.8	1.4	17.7 - 17.9	17.4
	25	Boys	562	16.9	1.3	16.8 - 17.0	16.6
		Girls	469	16.7	1.4	16.6 - 16.8	16.3
		Total	1031	16.8	1.4	16.7 - 16.9	16.5
	37	Boys	566	16.5	1.3	16.4 - 16.6	16.1
		Girls	477	16.5	1.5	16.4 - 16.6	15.9
		Total	1043	16.5	1.4	16.4 - 16.6	16.0
	49	Boys	562	16.2	1.3	16.1 - 16.3	15.8
		Girls	451	16.3	1.5	16.1 - 16.4	15.7
		Total	1013	16.2	1.4	16.2 - 16.3	15.7
	61	Boys	532	16.0	1.4	15.9 - 16.1	15.5
		Girls	440	16.1	1.6	16.0 - 16.3	15.5
		Total	972	16.0	1.5	16.0 - 16.1	15.5
HSE	6	Boys	154	16.2	1.5	15.9 - 16.4	15.5
		Girls	144	16.2	1.9	15.9 - 16.5	15.5
		Total	298	16.2	1.7	16.0 - 16.4	15.5
	7	Boys	144	16.4	1.8	16.1 - 16.7	15.6
		Girls	136	16.5	2.3	16.1 - 16.9	15.7
		Total	280	16.4	2.0	16.2 - 16.7	15.6
	8	Boys	136	16.6	2.1	16.3 - 17.0	15.7
		Girls	159	16.6	2.4	16.3 - 17.0	16.0
		Total	295	16.6	2.3	16.4 - 16.9	15.9
	9	Boys	131	17.1	2.5	16.7 - 17.5	16.0
		Girls	116	17.1	2.6	16.6 - 17.6	16.4
		Total	247	17.1	2.6	16.8 - 17.4	16.2
	10	Boys	135	17.4	2.3	17.1 - 17.8	16.4
		Girls	117	18.2	3.0	17.6 - 18.7	16.9
		Total	252	17.8	2.6	17.5 - 18.1	16.7
	11	Boys	122	18.0	2.6	17.6 - 18.5	16.9
		Girls	134	18.6	3.3	18.0 - 19.2	17.5
		Total	256	18.3	3.0	18.0 - 18.7	17.2
	12	Boys	126	18.8	2.9	18.3 - 19.3	17.4
		Girls	120	20.0	4.1	19.2 - 20.7	18.1
		Total	246	19.4	3.6	18.9 - 19.8	17.8
	13	Boys	131	19.4	3.3	18.8 - 19.9	18.0
		Girls	132	20.3	3.6	19.7 - 20.9	18.8
		Total	263	19.8	3.4	19.4 - 20.3	18.4
	14	Boys	136	20.3	3.6	19.7 - 20.9	18.7
		Girls	113	21.2	3.6	20.5 - 21.9	19.4
		Total	249	20.7	3.6	20.3 - 21.2	19.0
	15	Boys	140	21.0	3.6	20.4 - 21.6	19.3
		Girls	104	22.0	4.2	21.2 - 22.8	20.0
		Total	244	21.4	3.9	20.9 - 21.9	19.6

* All means are significantly different from UK1990 mean values ($p<0.001$).

¤ Reference value was percentile 50 of the UK 1990 reference data (Cole et al 1995).

Table 3.4. BMI standard deviation scores for UK children derived from studies 1 and 2.

Study	Age (months in ALSPAC years in HSE)		n	BMI-SDS Mean ¤	SD	95% CI
ALSPAC	4	Boys	537	-0.26	1.06	-0.35 to -0.17
		Girls	459	-0.17	1.04	-0.26 to -0.75
		Total	996	-0.22	1.05	-0.28 to -0.15
	12	Boys	671	0.15	0.96	0.08 - 0.22
		Girls	565	0.21	1.01	0.13 - 0.29
		Total	1236	0.18	0.99	0.12 - 0.24
	25	Boys	562	* 0.12	0.98	0.04 - 0.20
		Girls	469	0.17	0.95	0.08 - 0.26
		Total	1031	0.14	0.96	0.08 - 0.20
	37	Boys	566	0.26	0.96	0.18 - 0.34
		Girls	477	0.37	0.95	0.28 - 0.46
		Total	1043	0.31	0.96	0.25 - 0.37
	49	Boys	562	0.31	0.94	0.23 - 0.39
		Girls	451	0.34	0.95	0.25 - 0.43
		Total	1013	0.33	0.94	0.27 - 0.39
	61	Boys	532	0.23	1.00	0.14 - 0.31
		Girls	440	0.31	0.94	0.22 - 0.40
		Total	972	0.27	0.97	0.21 - 0.33
HSE	6	Boys	154	0.36	0.97	0.21 - 0.51
		Girls	144	0.28	1.03	0.11 - 0.45
		Total	298	0.32	1.00	0.21 - 0.43
	7	Boys	144	0.42	0.95	0.26 - 0.58
		Girls	136	0.27	1.06	0.09 - 0.45
		Total	280	0.35	1.01	0.23 - 0.47
	8	Boys	136	0.34	1.08	0.16 - 0.52
		Girls	159	NS 0.14	1.11	-0.03 - 0.31
		Total	295	0.23	1.10	0.10 - 0.36
	9	Boys	131	0.35	1.09	0.16 - 0.54
		Girls	116	NS 0.15	1.04	-0.04 - 0.34
		Total	247	0.25	1.07	0.12 - 0.38
	10	Boys	135	0.36	0.95	0.20 - 0.52
		Girls	117	0.33	1.08	0.13 - 0.53
		Total	252	0.35	1.01	0.22 - 0.47
	11	Boys	122	0.33	1.08	0.14 - 0.52
		Girls	134	0.25	1.10	0.06 - 0.44
		Total	256	0.29	1.09	0.16 - 0.42
	12	Boys	126	0.42	1.09	0.23 - 0.61
		Girls	120	0.42	1.24	0.20 - 0.64
		Total	246	0.42	1.16	0.28 - 0.56
	13	Boys	131	0.31	1.16	0.11 - 0.51
		Girls	132	0.37	1.16	0.17 - 0.57
		Total	263	0.34	1.15	0.20 - 0.48
	14	Boys	136	0.39	1.21	0.19 - 0.59
		Girls	113	0.45	1.09	0.25 - 0.65
		Total	249	0.42	1.16	0.28 - 0.56
	15	Boys	140	0.40	1.23	0.20 - 0.60
		Girls	104	0.48	1.18	0.25 - 0.71
		Total	244	0.43	1.21	0.28 - 0.58

* $p<0.002$ (NS = not significant)

¤ All SDS means were significantly different from expected ($p<0.001$) except in 8-9 year old girls.

3.5. Discussion

3.5.1. Obesity: secular trends in adults

In the USA, comparison of BMI changes in the US National Health Examination Survey (NHES I; 1960-62) and the National Health and Nutrition Examination Surveys (NHANES I: 1971-1974; NHANES II: 1976-1980; NHANES III: 1988-94), the prevalence of obesity in adults (BMI ≥30.0) showed a large increase (12.8% in NHES I, 14.1% in NHANES I, 14.5% in NHANES II, and 22.5% in NHANES III; Flegal et al 1998). Trends were generally similar for all age, gender and race-ethnic groups. This means that between 1976-80 and 1988-94, the prevalence of obesity increased markedly in US adults (Flegal et al 1998). Data from the 1983, 1990/91, and 1994 Italian National Health Surveys confirmed that the overall proportion of overweight subjects (defined as BMI=25-29.9) increased from 27.4% in 1983 to 31.6% in 1990/91 and 32.0% in 1994 (Pagano et al 1997). Using data from the Office of Population Censuses and Surveys, prevalence of clinical obesity (defined as BMI >30) in Britain was about 6% and 8% in men and women in 1980 (Knight 1984). The corresponding estimates were 8% and 12% in 1987 for English men and women respectively (White et al 1993) and obesity prevalence then increased to 13% and 16% in 1994 (Bennett et al 1995; Prentice & Jebb 1995) and to 17% and 20% in 1997 (Jebb 1999).

3.5.2. Childhood overweight / obesity secular trends

3.5.2-a. Obesity: secular trends in UK children

During the period of 1972 to 1994 triceps skinfold measurement increased by almost 8% in 7-year-old English children and by nearly 11% in Scottish children at 7 years of age (Hughes et al 1997). Weight for height index followed a similar pattern and suggested that in the UK, the average fatness of children during this period increased dramatically (Hughes et al 1997), but no estimates of obesity prevalence were available over the same period in the UK.

To the best of our knowledge, confirmed by Hughes et al (1997) study, the paper by Reilly et al (1999a) was the first one to estimate obesity prevalence in British children (based on part of the present thesis studies), though by mid-2000 a report of obesity (defined as having BMI >98th centile) prevalence in 20,973 Plymouth (English) 5-14 years old children during 1994-96 was published (Kinra et al 2000). Obesity prevalence increased with age and almost doubled in the oldest age quarter (boys 6.2%; girls 7.0%), compared to the youngest age quarter (Kinra et al 2000). In addition, the Sahota et al (2000) study of 7-11 year olds in Leeds (378 boys and 316 girls) in 1996-1998 to determine overweight / obesity prevalence (defined as BMI >85th and >95th percentiles) also indicated that between 18 to 26% of the study children were overweight and 9-14% were obese (Sahota et al 2000). Further results of the same study published later in October 2000 (Rudolf et al 2000) showed that the mean BMI SDS of the Leeds study children compared to UK 1990 reference data (Cole et al 1995) had increased by 0.19 SD scores. The whole normal distribution curve for the Leeds children moved to the right and this movement caused the percentage of children with BMI >50th percentile to increase by about 6% (Rudolf et al 2000). Other more recent studies of obesity prevalence in UK children (Reilly & Dorosty 1999; Reilly et al 1999a; Kinra et al 2000; Sahota et al 2000; Bundred et al 2001; Chinn & Rona 2001; Rudolf et al 2001) were not available at the time the work for the present Chapter was being carried out.

3.5.2-b. Obesity: secular trends in European children

The distribution of BMI in a nationally representative study of 14,500 Dutch 0-21 year olds in 1996-7 was compared to 1980. From 3 years of age onwards 14-22% of the subjects in 1996/97 exceeded the 90th centile of 1980. Therefore, children BMI have increased in the past 17 years (Fredriks et al 2000).

To analyse trends in BMI and overweight prevalence for children and adolescents in the region of Aragón (Spain), Moreno et al (2000) reported nine cross-sectional examinations of school children conducted yearly during 1985-1995. A total of 90,997 children in the first school year (6-7 years of age) and a total of 106,284 scholars in the last school year (13-14 years of age) were checked. The secular trends in BMI showed the largest increases at the upper ends of the BMI distribution, especially in males. A significant trend in overweight prevalence in school children from 1985-1995 was observed (Moreno et al 2000).

In a German study of 2554 school children and adolescents aged 6-19 years, the authors described the BMI centile values of German children being higher than French, lower than North American and Italian, and similar to Swedish and British children (Schaefer et al 1998) but their information sources for these comparisons were not clear.

In a French cross-sectional study (Michaud et al 2000) on 3206 school age children and adolescents aged 11-20 years in 1998, prevalence of overweight and obesity was defined as BMI >90th and >97th centiles of French reference (presumably Rolland-Cachera et al 1991, not clear from the publication). Between 21 to 25% of the study children were overweight and 10-13% of them were obese. More urbanised areas had the highest prevalence of overweight / obesity.

In 10 year old Yugoslavian children (n=5834; 2989 boys and 2845 girls) in 1998, obesity (BMI >95th centile compared to NHANES I) was prevalent in 9.0% and 8.5% and overweight prevalence (BMI >85th centile) was identified in 20.8% and 18.1% in boys and girls respectively (Pavlovic et al 2000; study 1). To investigate the prevalence of overweight (BMI >85th centile compared to NHANES I) and obesity (BMI >95th centile) in 2 different periods, a sample of 12380 (6405 boys; 5975 girls) 8-14 year old Yugoslavian children in

1989/90 and a group of 6692 (3365 boys; 3327 girls) children of the same age in 1995/98 were studied. Obesity (BMI >95th centile) prevalence was calculated as 3.6% in both boys and girls in the first period while it increased to 6.4% and 7.2% for boys and girls respectively in the second period (Pavlovic et al 2000; study 2). The prevalence of overweight (BMI between 85th and 95th centiles) was 11.1% and 12.2% in boys and girls in 1989/90 while it was 17.4% and 18.7% in boys and girls during the second period of the study (Pavlovic et al 2000; study 2).

3.5.2-c. Obesity: secular trends in Australian children

BMI from two surveys of 7-12 year Australian children in 1985 (n=1421) and 1997 (n=2277) were compared (Lazarus et al 2000). At all ages, mean height and median weight were greater in 1997 than 1985 for both sexes. Median BMI was significantly higher in the 1997 sample for all but 12 year old girls and for boys aged 7, 8 and 10 years. Plots of BMI against BMI percentile clearly showed a pattern of higher BMI at any given percentile, especially at the upper percentiles for all ages and both genders. Primary school Australian children have become more obese over the last decade. Increases in BMI were most marked at the heavier end of the distribution. Lesser increases in median and mean BMI may also have major public health implications (Lazarus et al 2000).

3.5.2-d. Obesity: secular trends in US children

There has been a significant increase in the body weight of children in the USA, particularly since the early 1980s, with a corresponding increase in the prevalence of obesity (Slyper 1998). The National Health and Nutrition Examination Surveys (NHANES) in the USA also found a doubling of obesity between 1963 and 1991 based on the 95th percentile for body mass index in the USA population (Slyper 1998). More than 10% of 4 and 5 year old girls were overweight in 1988-1994 (NHANES III) compared with 5.8% in 1971-1974

(NHANES I; Ogden et al 1997). Prevalence of overweight and obesity (BMI >85th and >95th percentiles respectively) from Cycles II (1976-1980) and III (1988-1991) of the NHES showed that the prevalence of obesity was 10.9% and overweight prevalence was 22.0% among all sex and age groups of 6-17 year old children. The greatest increase was during 1976-1980 (NHES II; Troiano et al 1995). Between 1973 and 1994, the mean body weight of 5-14 year old children in the Bogalusa heart study increased by 3.4 kg, and the weight of 15-17 year olds by 5.7 kg (Slyper 1998). The prevalence of overweight based on the 85th percentile for body mass index doubled during this period of time (Slyper 1998).

3.5.2-e. Obesity: secular trends in Canadian children

The prevalence of overweight (BMI >85th centile relative to BMI percentiles from the NHANES I & II; Frisancho 1990) and obesity (BMI >95th centile) was observed over 5 years (1993-97) among inner-city elementary school children aged 10-12 years in multiethnic, low-income families in Montreal, Canada. The prevalence of overweight was 35.9% in 1993 and increased by approximately 1.3% per year. The prevalence of obesity was 15.9% in 1993 and increased by approximately 1.0% per year. There were significant secular trends of increasing overweight and obesity among young inner-city school children in Canada from the early to late 1990s (O'Loughlin et al 2000).

3.5.2-f. Obesity: secular trends in children in other countries

Results of 71 national nutrition surveys since 1986 from 50 countries on 150,482 12-60 month old children showed that prevalence of overweight and obesity (weight for height >1 and >2 SD compared to the WHO / NCHS reference data respectively) in 32 out of 50 countries was less than 2.3% and the prevalence of overweight / obesity was lowest in Asia and in Sub-Saharan Africa (Martorell et al 2000). In 17 countries with serial data, no consistent regional trends were detected. Overweight was more common in urban areas, and

in children of mothers with higher education. Obesity was not a major public health problem among pre-school children in countries of Asia and Sub-Saharan Africa, but there is some evidence of increasing child, adolescent, and adult obesity in developing countries (de Onis & Habicht 1996). Martorell et al (2000) noted that in a number of countries in Latin America and the Caribbean, the Middle East and North Africa, and the region of Central Eastern Europe / Commonwealth of Independent States, levels of obesity appears to be as high as in the USA (Martorell et al 2000).

The prevalence of overweight (>1.0 SD above mean or >94.4th percentile of weight-for-height) in children 1-5 years of age varied from 6% in Haiti to 24% in Peru among 13 countries (Martorell et al 1998). The prevalence of severe obesity (>2.0 SDS above mean or >97.7th percentile of weight for height) in children 1-5 years of age ranged from 1.4% in Haiti to 4.7% in Peru (Table 3.6; Martorell et al 1998). In conclusion, these surveys indicate that paediatric obesity is on the increase in some developing countries, and that young children have been affected.

3.5.3. Overweight / obesity prevalence in US children

As can be seen from Figure 3.8, the prevalence of childhood obesity in the USA more than doubled over 14 years from 1980 to 1994 (Freedman et al 1997). The results reported for children in the USA show that in the last 20 years the prevalence of overweight has increased among 3-5 year old children but not among younger children (Troiano & Flegal 1998). The prevalence of overweight (BMI >95th percentile) in children between 6-17 years old in the USA during 1992-1994 was calculated as about 11% (Freedman et al 1997) which is very similar to the prevalence in the UK (based on HSE study and calculated in the present study) of 12.6% (Figure 3.8).

3.5.4. Childhood overweight / obesity prevalence in the United Kingdom

The present study found that overweight / obesity prevalence in UK children started to increase above that expected from around age 2.5-3.0 years. These findings suggest that efforts to prevent overweight, including encouragement of physical activity and improved diets should begin in early childhood (Ogden et al 1997). As noted earlier, there was clear evidence of a secular trend to increasing fatness in UK children (Hughes et al 1997) during the 1970s-1990s and the present study shows that an epidemic of obesity has occurred in British children during the 1990s. In addition, since the expected frequencies of overweight (15%) and obesity (5%) were based on the UK 1990 reference data, derived from surveys carried out in the UK between 1978-1990, the large excess of overweight / obesity in British children must have occurred over a fairly short time.

Tables 3.5 and 3.6 present the prevalence of childhood overweight and obesity based on common obesity definitions in different countries. As can be seen from Table 3.5, the prevalence of childhood overweight / obesity (defined as BMI $\geq 85^{th}$ and $\geq 95^{th}$ centile) in UK children has been very similar to that of US and Canadian children during similar periods of time. Based on the results shown in Table 3.6, the prevalence of childhood overweight / obesity (defined as weight for height >1.0 and >2.0 SD) in British children was higher than in Latin-American as well as in some other countries.

Table 3.5. Childhood overweight / obesity (BMI ≥85th / ≥95th centiles) in different countries.

Country	Study year(s)	Sample size	Age (years) except for ALSPAC	Definition	% above cut-off	Investigator(s)
USA	1965-70	794	1-2	BMI ≥85th percentiles	11.0	Whitaker et al 1997
		777	3-5		15.1	
		673	6-9		18.4	
		724	10-14		14.8	
		707	15-17		13.0	
		794	1-2	BMI ≥95th percentiles	3.0	
		777	3-5		5.3	
		673	6-9		8.8	
		724	10-14		7.3	
		707	15-17		7.5	
USA (NHANES II)	1976-80	498	9-12	BMI ≥85th percentiles	39.4	Johnson-Down et al 1997
USA (NHANES III)	1988-94	3279	6-11	BMI ≥95th percentiles	10.6	Troiano & Flegal 1998
		2428	12-17		10.6	
		M / F			M / F	
		817,793	6-8		9.9, 9.5	
		856,813	9-11		12.6, 10.4	
		577,674	12-14		10.7, 11.5	
		577,600	15-17		12.0, 8.2	
		2250, 2280	6-14		11.1, 10.4	
USA (Bogalusa)	1973-74	3508	5-14	BMI ≥85th percentiles	15.0	Freedman et al 1997
	1976-77	3073			20.0	
	1978-79	2908			21.0	
	1981-83	2749			22.0	
	1983-86	2604			23.0	
	1987-91	2688			27.0	
	1992-94	2582			32.0	
	1973-74	3508	5-14	BMI ≥95th percentiles	5.0	
	1976-77	3073			7.0	
	1978-79	2908			8.0	
	1981-83	2749			9.0	
	1983-86	2604			10.0	
	1987-91	2688			12.0	
	1992-94	2582			11.0	
	1973-94	228, 216	5	BMI ≥95th percentiles	4.4, 6.0	Freedman et al 1999
		289, 268	6		7.6, 6.3	
		263, 279	7		6.8, 7.9	
		333, 314	8		10.8, 11.2	
		336, 329	9		12.2, 12.2	
		392, 352	10		15.6, 11.6	
		410, 414	11		12.4, 17.6	
		455, 421	12		11.6, 9.0	
		516, 430	13		13.0, 10.7	
		488, 406	14		10.4, 12.3	
		428, 376	15		10.3, 12.0	
		379, 320	16		11.1, 8.8	
		272, 253	17		7.7, 9.9	
		4789, 4378	overall		10.8, 10.8	
Canada	Not clear	2108	M / F 9-12	BMI &TSF ≥85th & ≥95th centiles	35.3, 33.0 15.1, 13.3	O'Loughlin 1998
UK (ALSPAC)	1991-92	1031 (longitudinal)	25	BMI ≥85th percentiles	15.8	Reilly et al 1999a
			49		20.3	
			61		18.7	
			25	BMI ≥95th percentiles	6.0	
			49		7.6	
			61		7.2	
UK (HSE)	1996	1355 boy 1275 girl	6-15	BMI ≥85th & ≥95th centiles	21.8-31.1 10.4-16.8	Reilly & Dorosty 1999

*NHANES = National Health and Nutrition Examination Survey

M / F = male / female TSF = triceps skinfold thicknesses

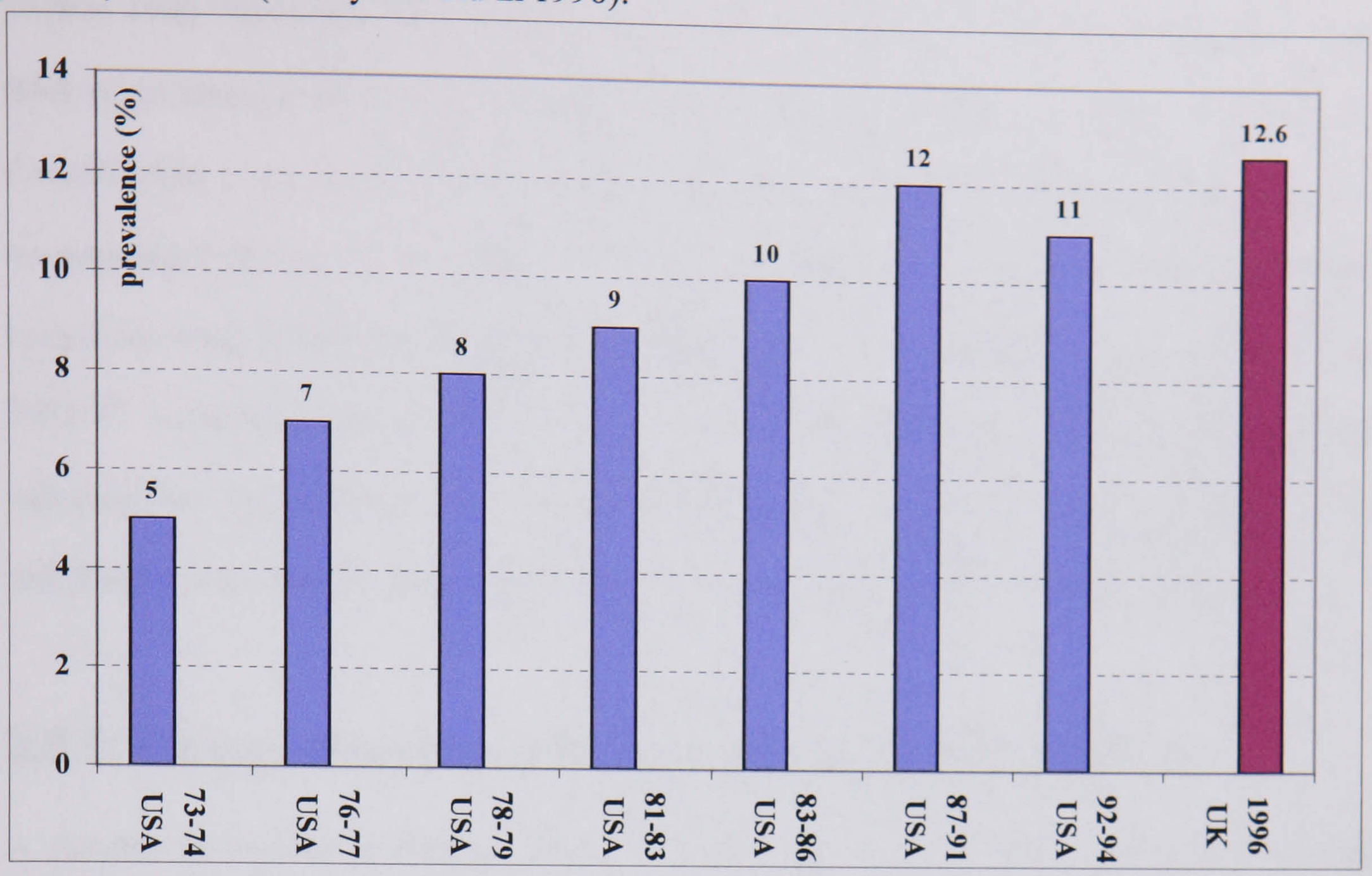
Table 3.6. Childhood obesity prevalence (definitions other than ≥95th centiles) in different countries.

Country	Study year(s)	Sample size	Age (m = month, y = years)	Definition	% above cut-off	Investigator(s)
NHANES I*	1971-74	M / F		Weight for height (wt-ht) >95th centiles	M / F	Ogden et al 1997
		285, 267	12-23m		7.5, 6.1	
		466, 415	24-47m		3.1, 2.0	
		577, 595	48-71m		4.4, 5.8	
NHANES II	1976-80	152, 143	6-11m		4.0, 6.2	
		368, 350	12-23m		7.2, 7.2	
		764, 655	24-47m		2.0, 2.5	
		796, 755	48-71m		4.4, 7.6	
NHANES III	1988-94	595, 576	6-11m		7.5, 10.8	
		648, 635	12-23m		7.5, 9.5	
		1107, 1136	24-47m		2.1, 4.8	
		1041, 1086	48-71m		5.0, 10.8	
USA Caucasian	1992	112	8y	Relative BMI >120%	19.8	Maffeis et al 1998
	1996	112	12y		22.3	
Italy			M 4y	wt-ht >120% SD	3.6	Maffeis et al 1993
			8y		11.2	
			10y		23.4	
			12y		17.3	
		749	All 4-12		15.7	
			F 4y		2.0	
			8y		13.3	
			10y		12.7	
			12y		11.9	
		774	All 4-12		11.0	
Sweden	1983	738	4y	wt-ht >120% SD	5.0	Persson 1984
			8y		3.0	
			13y		11.0	
Bolivia	1994	1866	12-35m	wt-ht SDS>1.0	13.1	Martorell et al 1998
				wt-ht SDS>2.0	2.1	
Brazil	1996	3165	12-59m	wt-ht SDS>1.0	14.7	Martorell et al 1998
				wt-ht SDS>2.0	4.1	
Colombia	1995	3600	12-59m	wt-ht SDS>1.0	12.2	Martorell et al 1998
				wt-ht SDS>2.0	1.8	
Dominican	1996	2984	12-59m	wt-ht SDS>1.0	15.3	Martorell et al 1998
				wt-ht SDS>2.0	4.6	
El Salvador	1993	2887	12-59m	wt-ht SDS>1.0	9.5	Martorell et al 1998
				wt-ht SDS>2.0	1.7	
Guatemala	1995	6477	12-59m	wt-ht SDS>1.0	10.0	Martorell et al 1998
				wt-ht SDS>2.0	2.0	
Haiti	1994-95	2079	12-59m	wt-ht SDS>1.0	5.7	Martorell et al 1998
				wt-ht SDS>2.0	1.4	
Honduras	1996	1329	12-60m	wt-ht SDS>1.0	3.7	Martorell et al 1998
				wt-ht SDS>2.0	1.4	
Mexico	1987	5286	12-59m	wt-ht SDS>1.0	14.5	Martorell et al 1998
				wt-ht SDS>2.0	3.9	
Nicaragua	1993-94	2581	12-59m	wt-ht SDS>1.0	12.1	Martorell et al 1998
				wt-ht SDS>2.0	2.2	
Paraguay	1990	2824	12-59m	wt-ht SDS>1.0	17.1	Martorell et al 1998
				wt-ht SDS>2.0	2.7	
Peru	1996	11796	12-59m	wt-ht SDS>1.0	23.9	Martorell et al 1998
				wt-ht SDS>2.0	4.7	
Trinidad & Tobago	1987	616	12-36m	wt-ht SDS>1.0	8.9	Martorell et al 1998
				wt-ht SDS>2.0	1.9	
UK (ALSPAC)	1991-92	1236 (longitudinal)	12-61m	BMI SDS>1.0	15.8-24.8	Present study 1999
				BMI SDS>2.0	2.0-4.4	
UK (HSE)	1996	2630	6-15y	BMI SDS>1.0	22.7-31.6	Present study 1999
				BMI SDS>2.0	6.4-10.7	

*M / F = male / female

Relative BMI: BMI / BMI at 50th centile for age & gender multiplied by 100

Figure 3.8. Prevalence of obesity (BMI >95th centile) in USA (5-14 year olds at different periods) and UK (6-15 years old in 1996).



Obesity (BMI ≥95th centile) in UK children was calculated in the present study as 6.4%, 7.7% and 7.2% respectively for one year, 3 year, and 5 year old children (Table 3.1). In addition, the prevalence of overweight in children based on the ≥85th percentile in UK children was calculated as 18.9%, 18.4%, and 18.7% respectively for 1, 3, and 5 year old children (Table 3.1). Mean values of BMI and SDS shown in Table 3.3 indicate that from the age of 1 year, mean BMI and mean BMI SDS in UK children were constantly higher than UK 1990 reference data.

The UK 1990 reference data (Cole et al 1995) assumes that only 5% of UK children have BMI ≥95th percentile. The present results (study 1) show that the prevalence of obesity increased at most ages (Tables 3.1 and 3.2; Figures 3.1 and 3.2), such that the obesity prevalence at age 15 years (based on the HSE 1996 study) was about 17% which was almost 3.5 times more than expected. At age 15 years, prevalence of overweight was 31% (expected value 15%) which was more than twice that expected (Table 3.2; Figures 3.1 and 3.2). There were no significant differences in obesity prevalence between the sexes in both studies 1 and 2.

The 1990 reference data (Cole et al 1995) for the UK were prepared from studies done during 1978 to 1990. These data were statistically adjusted so that they describe the weight, height, and BMI of children of the UK in 1990 by matching them to the National Study of Health and Growth 1990 (Cole et al 1995). The UK 1990 reference data (Cole et al 1995) have been recommended for use by an expert committee in the UK (RCPCH 2001; Wright et al 2002). Data from study 2 (HSE) were gathered in 1996 and the ALSPAC data were collected from 1992-97. Such rapid changes in overweight / obesity prevalence during this short period of time indicates that major changes in childhood obesity in the UK population occurred in the 1990s, and that these are broadly consistent with trends in UK adults (Prentice & Jebb 1995).

3.5.5. Causes of secular trends in obesity in UK children

A detailed discussion of possible causes is beyond the scope of this Chapter, but some key points are discussed briefly below.

3.5.5-a. Role of genetics

Studies have shown that genetic factors play a role in childhood obesity but the relative importance of inherited factors in the development and maintenance of obesity is unclear (Klesges et al 1995; Sorensen 1995; Perusse & Bouchard 1999). However, since the obesity epidemic in the developed world occurred quickly, rapid changes in population genetic 'background' are unlikely to be possible and therefore changes in lifestyle are probably more important contributors to the recent increase in childhood obesity.

3.5.5-b. The role of energy-intake

Studies in the UK (based on the data from the National Food Survey) showed a 20% decrease in energy intake in adults during 1970-1995 (Prentice & Jebb 1995). In British pre-school children, a 20% fall in mean energy intake occurred between 1967-1992 (Gregory et al 1995). The results from the National Diet and Nutrition Survey in young people aged 4-18 years (NDNS 2000) compared to previous studies of UK children indicate that from 1982 to 1997, energy intake in

British children significantly decreased (Table 3.7). Therefore energy expenditure might be the important reason for secular trends in obesity in the UK population, since obesity risk of course depends on the balance between energy intake and expenditure.

Table 3.7. UK children's mean energy intake reported by different studies in different periods.

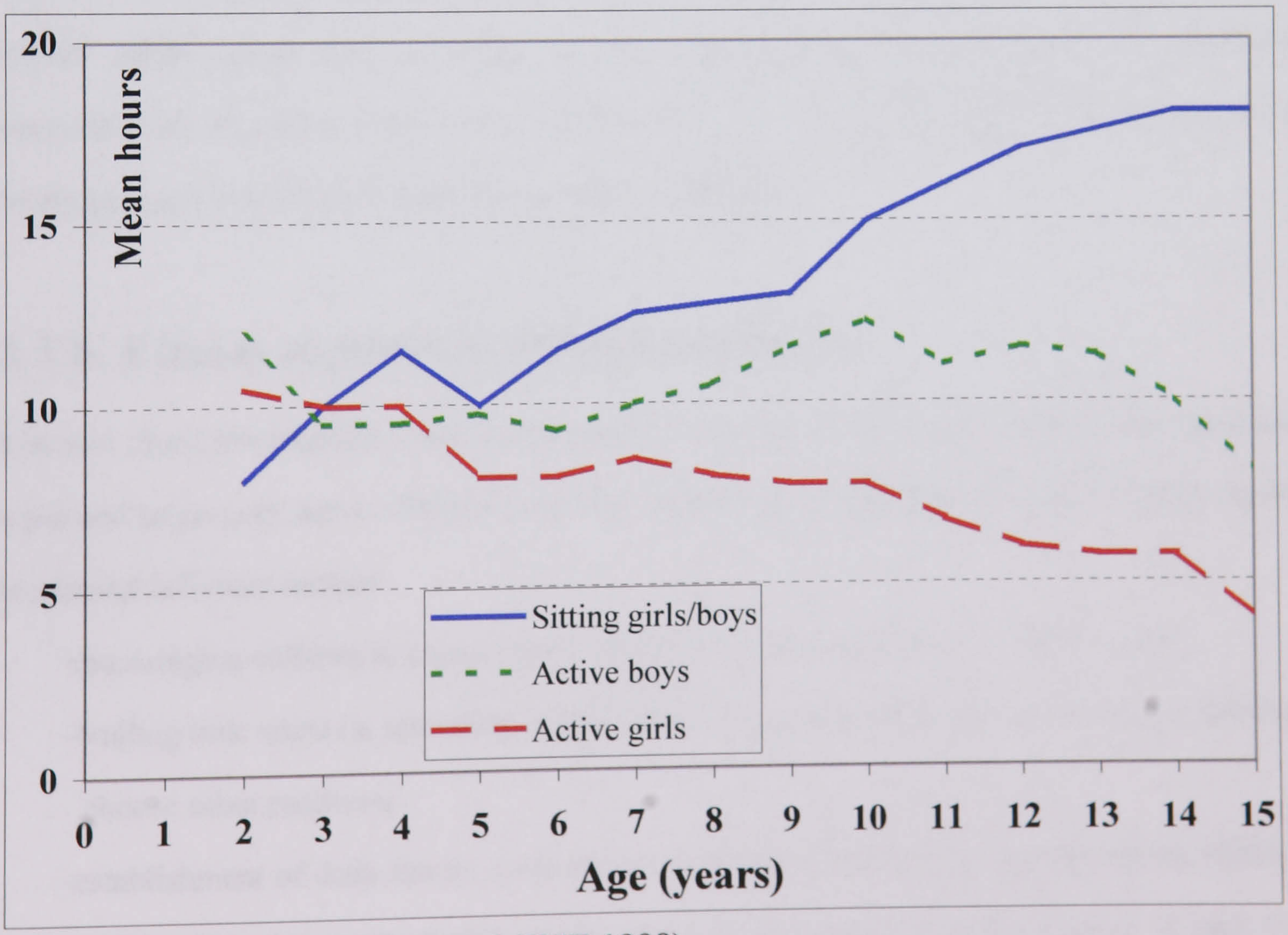
Reference	Age (year)	Sex	Sample size	Energy intake MJ/day	Percentage energy from:		
					Fat	CHO	Protein
Bull 1982	15-18	M	198	10.1	42	43	13
		F	184	7.8	43	43	13
Crawley 1986-87	16-17	M	2006	11.4	41.4	44.2	12.5
		F	2754	8.8	41.6	43.9	12.5
Nelson 1988	7-10	M	25	7.6	36.6	-	-
		F	26	6.9	35.0	-	-
	11-12	M	76	7.7	37.5	-	-
		F	67	7.4	38.4	-	-
McNeill 1988-89	12	M	18	9.0	-	-	-
		F	43	8.1	-	-	-
Hunt 1988, 90	8-11	M	111	7.61	37	51	-
		F	148	6.53	39	50	-
Strain 1990-91	12	M	251	11.0	39	50	11
		F	258	9.2	38	50	11
	15	M	252	13.1	39	50	11
		F	254	9.1	39	49	11
Ruxton 1991-92	7-8	M	65	7.8	37.4	50.8	11.8
		F	71	7.2	37.8	50.3	11.9
NDNS 1998	4-6	M	Not clear	6.4	35.5	51.6	12.9
		F		5.9	35.9	51.4	12.7
	10-11	M		8.0	35.7	51.5	12.8
		F		7.0	36.6	51.2	12.7
	14-15	M		9.1	35.9	50.6	13.5
		F		6.9	36.1	50.8	13.2
NDNS 2000	4-6	M	Not clear	6.4	35.5	51.6	12.9
		F		5.9	35.9	51.4	12.7
	7-10	M		7.5	35.2	52.4	12.4
		F		6.7	35.9	51.3	12.8
	11-14	M		8.3	35.2	51.7	13.1
		F		7.0	36.1	51.2	12.7

* The information in this Table was based on the National Diet and Nutrition Survey (NDNS 2000).

3.5.5-c. The role of lifestyle: physical inactivity

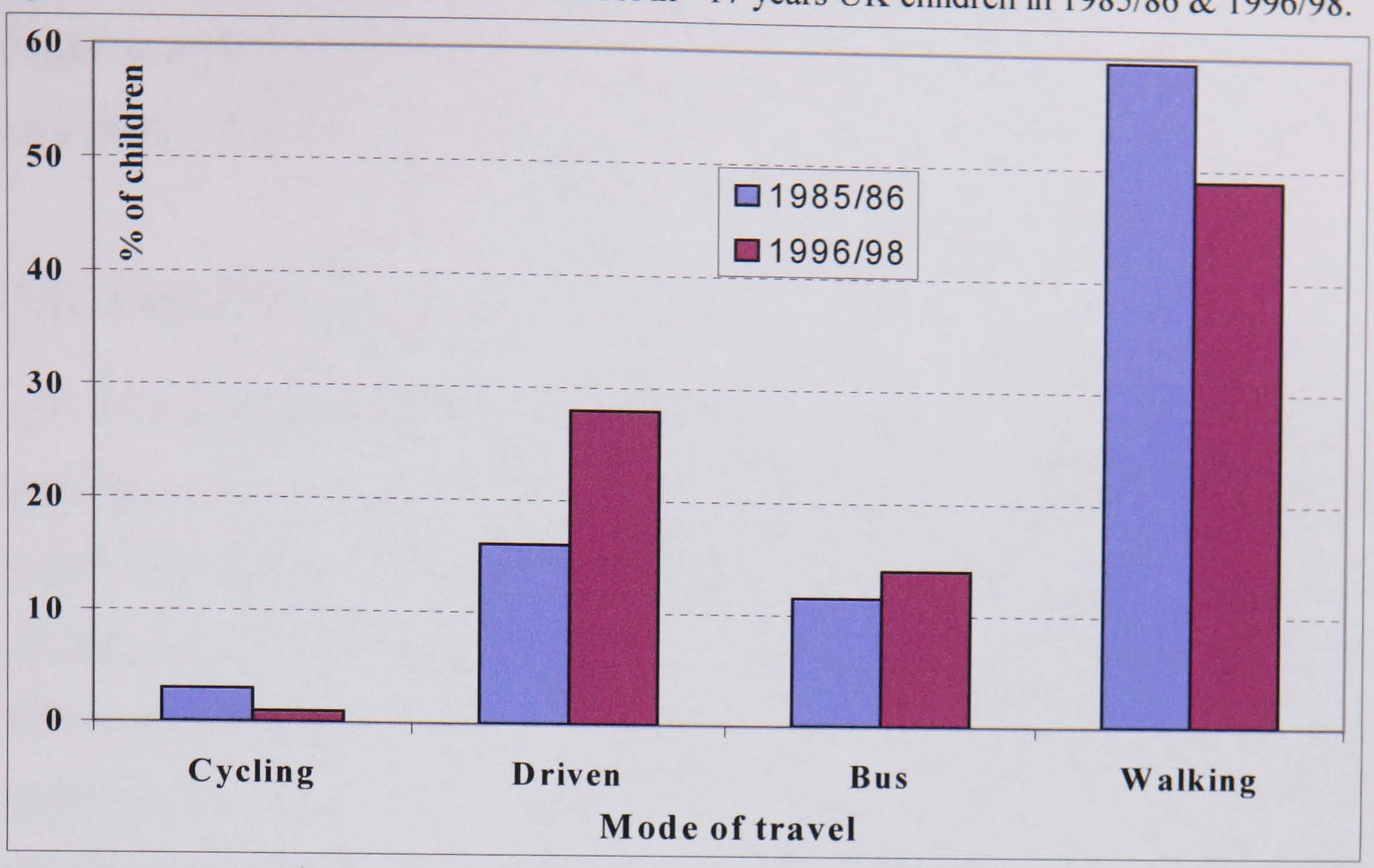
Lifestyles probably changed dramatically during 1980s and 1990s as physically arduous tasks were replaced with motorised transport, mechanised equipment, and energy saving domestic appliances (Prentice & Jebb 1995). Watching television and other inactive behaviours are common for children and adults and central heating perhaps reduced the need to expend energy for thermoregulation (Prentice & Jebb 1995). The amount of time spent watching television in UK children has doubled since the 1960s (Pullinger 1998), and the role of TV viewing as a cause of obesity in children is clear (Gortmaker et al 1993). The amount of time spent on physical activities in children has reduced and this reduction is more marked in older children especially older girls (Figure 3.9; HSE 1998). In addition, the mode of travel to and from school in school children has changed since 1985, such that walking and cycling have decreased and the use of car / bus has increased (Figure 3.10)

Figure 3.9. Mean time UK 2-15 year olds spent in physical activities and/or sitting.



Source: Health Survey for England (HSE 1998).

Figure 3.10. Mode of travel to / from school in <17 years UK children in 1985/86 & 1996/98.



Source: Transport Statistics Bulletin (Transport Statistics Bulletin 1999).

Active transport to and from school has decreased in English children: walking to school in the under 17 year olds fell from 59% to 49% between 1985/86 to 1996/98 with 28% compared to 16% of the children being driven to school (Figure 3.10; Transport Statistics Bulletin 1999; NDNS 2000). While these national surveys included fairly crude measures of activity and inactivity, they all tend to point to the conclusion that modern children are less active than in the recent past and that physical inactivity increases with age.

3.5.6. Efforts to prevent childhood obesity

It is now clear (from studies 1 and 2) that childhood obesity in the UK is a serious issue and needs rapid and large-scale action. Efforts to prevent childhood obesity might consist of efforts targeted at parental activities include:

-encouraging children to change their behaviour to increase their physical activities;

-limiting time spent on television, video games, and computer games to encourage children to choose other pastimes;

-establishment of daily family meal and snack times (when healthy options can be offered to children), and control of ready-made foods and/or limitation or elimination of high-fat or high-sugar foods (Barlow & Dietz 1998; Gortmaker et al 1999).

In addition to parental actions, monitoring and surveillance of weight among children is essential (Ogden et al 1997). Charting BMI as part of continuing child health surveillance may identify in early childhood those who are likely to become obese adults (White et al 1995).

3.6. Study limitations

It is well known that BMI is not as accurate as a body fat percentage measurement (such as underwater weighing or use of a multi-component model) for screening obese children but BMI is acceptable enough for most clinical and epidemiological purposes (Barlow & Dietz 1998; Chapters 2 and 5). The present study results provide a picture of obesity in UK children in different age groups between 4, 8, 12, 18, 25, 31, 37, 43, 49, and 61 months of age (ALSPAC study) and 6 to 15 years of age (HSE study) in the mid-1990s. The low sensitivity of BMI as a screening tool means that obesity prevalence in this Chapter will have been under-estimated. The limitations of BMI as a definition of obesity are discussed further in Chapter 5. Other specific limitations of the surveys include the relatively small number of subjects in each age group for HSE 1996, and the fact that both studies 1 and 2 were based only on English data. Data on secular trends in body fatness (triceps skinfold) suggest the problem of childhood obesity may be worse in Scotland than in England (Hughes et al 1997).

The results of both studies 1 and 2 (ALSPAC and HSE) were consistent with each other, and data gathering, measurement, and sampling were sound. Some of the possible causes of error are listed: no data are available on differences in prevalence between different ethnic groups / races in the UK; entering huge sets of data on the computer could have caused problems due to mistyped values or hardware and/or software faults. In addition, there are potential errors in study 2 arising from the practice of rounding age to the nearest integer. For example, all children 5.50 to 6.49 years old were assumed as 6-year-old children. Above the age of about 6 years, the effect of this rounding on BMI SDS or percentiles is small but below it, the effect is more marked and this was why in the present study, we did not analyse data from HSE in those children <6 years old. The scales used in study 2 were only accurate to 500 grams and children were weighted in light indoor clothing which might be a possible source of error.

3.7. Conclusions

The current study provides information about the prevalence of obesity in UK children in the mid-late 1990s. We used BMI SDS and percentiles compared to growth standards which are believed to represent the BMI and growth of British children in 1990 (Cole et al 1995) to define obesity prevalence. Most studies in the literature have been concerned with adult obesity, and so there was no information about obesity in childhood in the UK and estimates of its prevalence before this study took place in 1998/1999. Subsequent UK studies have generally confirmed these findings (Reilly & Dorosty 1999; Reilly et al 1999a; Kinra et al 2000; Sahota et al 2000; Bundred et al 2001; Chinn & Rona 2001; Rudolf et al 2001).

In the present study, differences in the prevalence of overweight and obesity between boys and girls were not statistically significant in both the ALSPAC and HSE studies. Kinra et al (2000) also found no evidence of a difference between the sexes for English children. The prevalence of obesity significantly exceeded 'expected' frequencies at all ages except at 4, 18, and 25 months. From age 3 years, the prevalence of overweight / obesity started to significantly increase and this increase continued thereafter. In the ALSPAC study, ages 12 and 43 months showed the highest prevalence of overweight / obesity (Figure 3.3). Results of the HSE survey showed that from the age 6 years, the prevalence of overweight / obesity increased in all ages and, in both sexes it was constantly higher than expected. Although there were fluctuations in the obesity prevalence in different age groups, obesity prevalence showed an increase from age 6 years (Figure 3.4).

In adults, the prevalence of obesity in countries such as the USA, the UK, Brazil and Australia have increased between half to two-fold during one to two decades. Some developed countries like Japan have a low prevalence and most developing countries have very low obesity prevalence in their adult populations. The prevalence of childhood obesity in the USA during 14 years (between 1980 to 1994) almost trebled. Obesity prevalence calculated in other European countries using the same obesity definitions as the present study (BMI percentiles), were not available for comparisons.

4. Chapter 4: Prevalence of overweight and obesity in Iranian children

4.1. Introduction

4.1.1. Obesity prevalence in developing countries: adults

Obesity is now a major public health problem in both developed and developing countries (Jebb 1999). Obesity prevalence in most parts of the world is believed to be high and increasing (Gill 1999). Global adult obesity prevalence (BMI >30) is currently estimated to be more than 250 million adults (Gill 1999). In the UK, about 17% of men and 20% of women were obese in 1999 and over half of UK adults were overweight, an increase of more than 100% from 1980-1999 (Jebb 1999). Obesity is well known for its resistance to treatment and is associated with many diseases which are quickly becoming some of the major causes of death in many developing as well as developed countries (Popkin et al 1995). For instance, in 1960-1962, cancer, cerebro-vascular disease and heart disease were the 6th, 7th and 8th most common causes of mortality in Shanghai County of China but from 1978 to 1980 they became the first three leading causes of death in that area (Popkin et al 1995).

National surveys conducted in the USA and other industrialised countries indicate that the prevalence of adult obesity in these regions is about 25% or higher (Popkin et al 1995). Smaller surveys in developing countries have shown that adult obesity is already prevalent in many areas, although the rates are usually much lower than in the USA and the prevalence varies from country to country (WHO 1990). In some countries a high prevalence is already evident in children as well as in adults (WHO 1990). It must be noted that data from developing countries are still limited. However, it is evident that overweight and obesity are rising in many of the less-developed countries of the world (Popkin 1994).

The temporal changes in BMI and prevalence of overweight (BMI >25) and obesity (BMI >30) among adult Kuwaitis between two periods 14 years apart, were calculated in two independent cross-sectional samples of Kuwaitis studied in 1980-1981 and 1993-1994. Prevalence of overweight increased by 20.6% and 13.7% and prevalence of obesity increased by 15.4% and 8.4% in men and women, respectively. Mean BMI was 2.0 and 1.6 kg/m^2 higher in 1993-1994 than in 1980-1981 among men and women and these increases of 10.0% and 6.2% in BMI in men and women were highly significant ($p<0.001$; Al-Isa 1997).

The causes of this trend towards increasing fatness in developing countries are poorly studied but probably include the extreme and rapid changes in lifestyle: changes in physical activity and diet due to urbanisation and rapid economic development (Popkin 1994). The results of such lifestyle changes are already being seen as most cardiovascular diseases, generally associated only with Western industrialised countries, now occur in developing countries (Pearson 1996).

4.1.2. Obesity prevalence in developing countries: children

Links between reduced growth *in utero* and increased risk of ill health in later life may partly explain why populations in many developing countries are especially susceptible to obesity, diabetes and heart disease when exposed to modern sedentary living (Gill 1999). The International Obesity Task Force has launched a global initiative for action to tackle the obesity epidemic (Gill 1999).

As mentioned in Chapter 3, Martorell et al (2000) reported results of 71 national nutrition surveys since 1986 from 50 countries on 150,482 12-60 month old children. They showed that prevalence of overweight and obesity (defined as weight for height >2 SD compared to WHO / NCHS reference data) in 32 out of 50 countries was less than 2.3% (Martorell et al 2000). Therefore, they concluded that obesity was not a major public health problem among pre-school children in Asia and Sub-Saharan Africa. But in a number of countries in Latin America and the Caribbean, the Middle East and North Africa, and the region of Central Eastern Europe /

Commonwealth of Independent States, obesity prevalence was as high as in the USA (Martorell et al 2000). The study by de Onis & Blossner (2000) also confirmed that the Middle East, North Africa and Latin America had the highest rate of childhood obesity (defined as weight for height >2 SD compared to WHO reference data) among developing countries (de Onis & Blossner 2000).

Based on the WHO consultation on obesity report (WHO 1998), which used weight for height >120% of standard to define obesity, a crude and very limited estimate of obesity prevalence in children has been made for various countries. No indication was given of age, race, sex, or any details of the sample. These estimates were as follows: Bangladesh 0.3%, India 1.2%, Jordan 2.2%, UK 2.8%, Italy 3.6%, Peru 4.0%, Iran 5.7%, Canada 5.8% and Chile 11.8%. Despite limitations in the methodology used by WHO, these values indicate that the prevalence of obesity in children of developing countries is probably high and in some cases even higher than developed countries, particularly when taken together with the studies of Martorell et al (2000) and de Onis & Blossner (2000).

Childhood obesity is an important issue, as discussed in Chapters 1 and 3, which should be a global concern. Unfortunately, trends and the public health costs of obesity in developing countries (even in adults) are unknown or not clearly known. This is because:

a. obesity is not widely recognised as a public health problem due to lack of knowledge about the trends in obesity, its costs, and the problems it causes.

b. developing countries generally suffer from a lack of national or regional population surveys (data sets) to analyse.

c. in such countries, based on food availability, wealth distribution and many other issues, BMI curves might be abnormally distributed so that they might have a high prevalence of under-nutrition and a high prevalence of obesity at the same time (Popkin et al 1995).

d. there are some methodological problems in defining childhood obesity. Using other countries reference data sets (or WHO reference data based on USA data) can be a problem and also there was a lack of agreement on a paediatric obesity definition (as described in Chapter 3) until recently. Two studies, in Saudi Arabia and Iran, are given here as examples of the differences which may occur when using different reference data sets to define childhood obesity.

In 1995 in Saudi Arabia a population of 9061 school boys aged 6-18 years were compared with the 50th percentile of the NCHS / CDC (National Center for Health Statistics / Center for Disease Control) reference population as the expected standard population values. The prevalence of overweight and obesity were calculated as 15.8% and 11.7% respectively (Al-Nuaim et al 1996). In this study, the authors developed growth charts for Saudi Arabian children and suggested that based on the possible ethnic differences in body composition between the Saudi and American populations, such growth charts could be better references for future assessments of obesity than the NCHS / CDC reference data (Al-Nuaim et al 1996).

In the recent Iranian National Health Survey (1990-1992), weights and heights of 22,349 children and adolescents (49.9% boys, 50.1% girls) aged 2-18 years in Iran were measured and BMI calculated. Using random cluster sampling, one child was chosen from every 1000 children between 2-18 years of age in all provinces of Iran (Hosseini et al 1998). Significant differences in childhood BMI between provinces and between urban and rural children were revealed. Differences between urban and rural children, and differences between girls and boys persisted across all provinces (Hosseini et al 1999). Results showed that the Iranian BMI centiles were generally substantially below those of the NCHS, at least at the lower end of the distribution. It was suggested that the use of locally based growth reference data are essential for assessing the growth and nutritional status of children in Iran (Hosseini et al 1999). Therefore, using Healy's method (Healy et al 1988), the first ever growth charts and BMI reference data for Iranian children were constructed (Hosseini et al 1999).

4.1.3. The International Obesity Task Force (IOTF) reference data

The working group on childhood obesity of the International Obesity Task Force (IOTF) was established in 1994 and proposed to raise awareness in the international population and among governments that obesity is a serious medical condition, to determine the prevalence of obesity in children and adolescents worldwide, to analyse secular trends internationally, to develop a public health policy and program for improving the prevention and management of obesity, and to develop an internationally acceptable definition of child overweight and obesity, specifying the measurement, the reference population, and the age and sex specific cut-off points (Dietz & Bellizzi 1999; Guillaume 1999). The results of international surveys of six large supposedly nationally representative cross-sectional growth studies in Brazil, Great Britain, Hong Kong, the Netherlands, Singapore, and the USA were combined. The BMI of a total sample of 97,876 males and 94,851 females from birth to 25 years of age was studied (Cole et al 2000). For each survey of those countries, centile curves were drawn for 18 year olds and passed through the widely used cut-off points of 25 and 30 kg/m^2 for adult overweight and obesity. The resulting curves were averaged to provide age and sex specific cut-off points for 2-18 years. The authors believe that these cut-off points are less arbitrary and more internationally based than current alternatives, and that they might help to provide internationally comparable prevalence rates of overweight and obesity in children (Cole et al 2000).

4.2. Aims

The aim of study 1 in this Chapter was to analyse the Iranian survey 1995 data and to estimate the prevalence of overweight / obesity in children of two Iranian provinces based on the Iranian reference data collected in 1990-1992. Then in the second study, the overweight / obesity prevalence in a sub group of children (only 5 year old children) using Iranian 1992, USA 1990, France 1990, and UK 1990 BMI reference data sets were compared, and the recently published IOTF 2000 reference data (Cole et al 2000) intended to be used for international comparisons of obesity prevalence. The second study aimed to demonstrate the effect of using different reference data sets on the estimation of obesity prevalence in 5-year old Iranian children.

4.3. Methods

4.3.1. Background: Iran and the National Health Survey

The 16th largest country of the world, the Islamic Republic of Iran, has a total area of 1,648,000 km^2 and based on the Iran Statistical Yearbook in 1991, had a population of about 56 million (Iranian Ministry of Health and Medical Education; MOHME 1995). Afghanistan, Pakistan, Turkey, Iraq and republics of the former USSR are Iran's neighbouring countries. Forty-four percent of the population lives in rural and 55% in urban areas and of the rest, 1% are nomads. The country has a very young population (over 44% below 15 years of age in 1995) and a high population growth rate (1.75% per year in 1995; MOHME 1995). The climate has large variation so that the north-western part is the coldest and the south is the warmest. Gilan is one of the provinces in the north with a mild and wet climate, and Sistan is a province in the south east, with a dry hot climate. In the present study, of the 2-5 year old children of these two provinces in 1995, a reasonable sample size was chosen and the prevalence of obesity / overweight was defined as study 1. Data from a sub-group of this sample (5-year-old children) was used to make a comparison between obesity prevalence results using five different BMI reference data (study 2).

4.3.2. Study 1

In a survey in 1995 in two different Iranian provinces, Gilan in the north and Sistan in the south, a sample of 4315 of 2-5 year old children (more than 1% of all 2-5 year old children) using simple random sampling were chosen from both urban and rural areas. From all healthy children who came to health care centres for control of their weight and height or for their vaccination, eligible children were chosen and investigation continued until enough cases were chosen. Weight was measured when wearing underwear to 0.5 kg using Seca scales and height was measured to 0.5 cm (using hand made wooden height measurer scales for 2-year-old children and Seca height scales for the rest) by trained staff of the Ministry of Health and Medical Education. Children were defined as 4 different age groups from 2 to 5 years. Each age group

took in a 6-month period either side of specified age rounded to the nearest integer value. For example, age group '2' consisted of all children aged 1.50 to 2.49 years old.

4.3.3. Study 2

All the 5 year old children (227 boys and 228 girls) from the population sample of study 1 were selected to make a comparison between different reference data sets in identifying obesity prevalence. The reference data sets which were used in this study were:

Iranian 1992 reference data (Hosseini et al 1999),

UK 1990 reference data (Cole et al 1995),

USA 1990 reference data (Rosner et al 1998),

French 1990 reference data (Rolland-Cachera et al 1991).

IOTF 2000 reference data (Cole et al 2000).

4.3.4. Statistical analysis and obesity definitions

The definitions of overweight / obesity used here were based on the recent agreed cut-offs (Barlow & Dietz 1998) discussed in Chapter 2. Children with BMI SDS ≥1.04 (≥85th percentile) were defined as overweight while those who had BMI SDS ≥1.64 (≥95th percentile) were defined as obese. Iranian 1992 reference data for 2-19 year old children, which were published recently (Hosseini et al 1999), were used. Using such definitions, the expected prevalence of overweight and obesity are 15% and 5% respectively. The percentage of overweight / obese children in each age and sex group was defined as 'prevalence'.

Differences between percentage of overweight / obesity in different age and sex groups were tested using two probability Z-tests and Chi-squared tests. Overweight / obesity percentage in each age and sex group was compared with the expected values given above, using Chi-squared goodness of fit tests to test the significance of differences.

4.4. Results

4.4.1. Study 1

The prevalence of obesity and overweight (%) in Iranian children, using Iranian 1990-1992 reference data (Hosseini et al 1999), at ages 2-5 years are presented in Table 4.1 and Figures 4.1 to 4.3. Table 4.1 shows that overweight (BMI $\geq 85^{th}$ centile) prevalence in all age groups of children (from age 2) and in both sexes was significantly higher ($p<0.01$) than the expected frequency (15%). Prevalence of obesity (BMI $\geq 95^{th}$ centile) in 3-5 year old children was significantly ($p<0.01$) higher than the expected value (5%). From age 3 years, the overweight prevalence in boys was constantly higher than in girls and these differences were significant at ages 3-4 years. The same results can be seen for obesity in 3-5 year old children, with the prevalence of obesity in boys was constantly higher in girls, though the only *significant* difference was at 3 years old (Table 4.1). Based on Table 4.1 and Figures 4.1 to 4.3, there was a trend in the overweight / obesity prevalence in the Iranian 2-5 year old children such that the lowest percentage of obesity was at age 2 and the highest was at age 5 years.

Figure 4.1. Prevalence of obesity (BMI $\geq 95^{th}$ percentile, using Iranian 1990-1992 and IOTF 2000 reference data) in Iranian children aged 2-5 years.

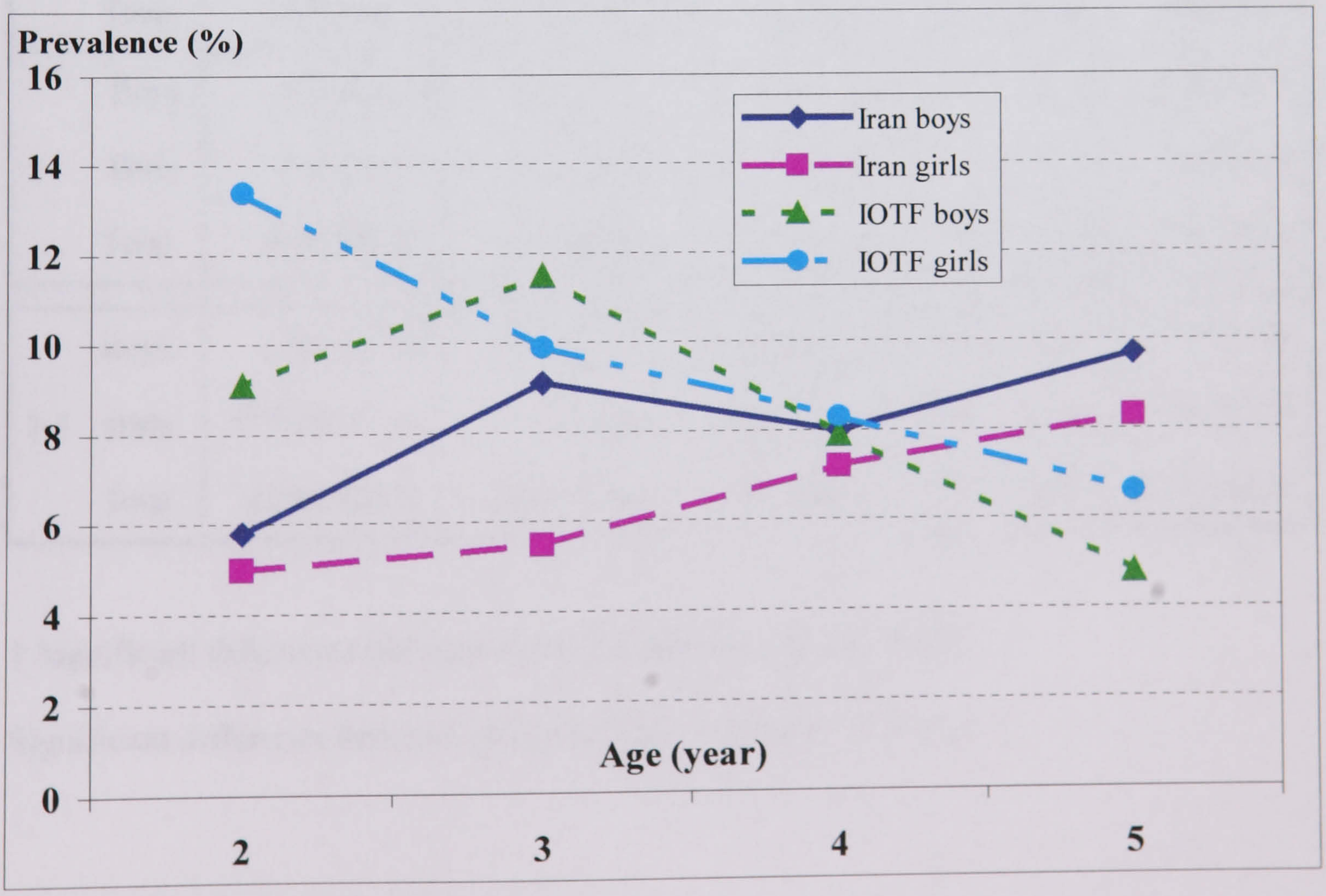


Table 4.1. Prevalence of obesity and overweight (%) in Iranian children (using Iranian 1992 BMI reference data and IOTF 2000 BMI reference data) at ages 2-5.

Age (years)		**Iranian** 1992 reference data		**IOTF** 2000 reference data		**Total**
		Overweight (≥85th centile) n (%)	**Obese** (≥95th centile) n (%)	**Overweight** 'Equivalent to BMI >25' n (%)	**Obese** 'Equivalent to BMI>30' n (%)	n (%)
2	Boys	†118 (19.5)	35 (5.8)	†131 (21.7)	†55 (9.1)	605 (51.0)
	Girls	†115 (19.8)	29 (5.0)	†154 (26.5)	†*78 (13.4)	582 (49.0)
	Total	†233 **(19.6)**	64 **(5.4)**	†285 **(24.0)**	†133 **(11.2)**	1187 (27.5)
3	Boys	†194 (28.6)	†62 (9.1)	†155 (22.9)	†78 (11.5)	678 (49.4)
	Girls	†**144 (20.8)	**38 (5.5)	†161 (23.1)	†69 (9.9)	695 (50.6)
	Total	†338 **(24.6)**	†100 **(7.3)**	†316 **(23.0)**	†147 **(10.7)**	1373 (31.8)
4	Boys	†168 (25.9)	†52 (8.0)	†115 (17.8)	†51 (7.9)	647 (49.8)
	Girls	†134 (20.5)	†47 (7.2)	†124 (19.0)	†54 (8.3)	653 (50.2)
	Total	†302 **(23.2)**	†99 **(7.6)**	†239 **(18.4)**	†105 **(8.1)**	1300 (30.1)
5	Boys	†73 (32.2)	†22 (9.7)	†39 (17.1)	11 (4.8)	227 (49.9)
	Girls	†60 (26.3)	†19 (8.3)	†43 (18.9)	15 (6.6)	228 (50.1)
	Total	†133 **(29.2)**	†41 **(9.0)**	†82 **(18.0)**	26 **(5.7)**	455 (10.5)
2-5	Boys	†553 (25.6)	†171 (7.9)	†440 (20.4)	†195 (9.0)	2157 (50.0)
	Girls	†**453 (21.0)	†*133 (6.2)	†482 (22.3)	†216 (10.0)	2158 (50.0)
	Total	†1006 **(23.3)**	†304 **(7.0)**	†922 **(21.4)**	†411 **(9.5)**	4315(100.0)

† Significant difference between observed and expected ($p<0.01$)

Significant difference between girls and boys: * $p<0.05$, ** $p<0.01$

Figure 4.2. Prevalence of overweight (BMI ≥85th percentile, using Iranian 1990-1992 and IOTF 2000 reference data) in Iranian children aged 2-5 years.

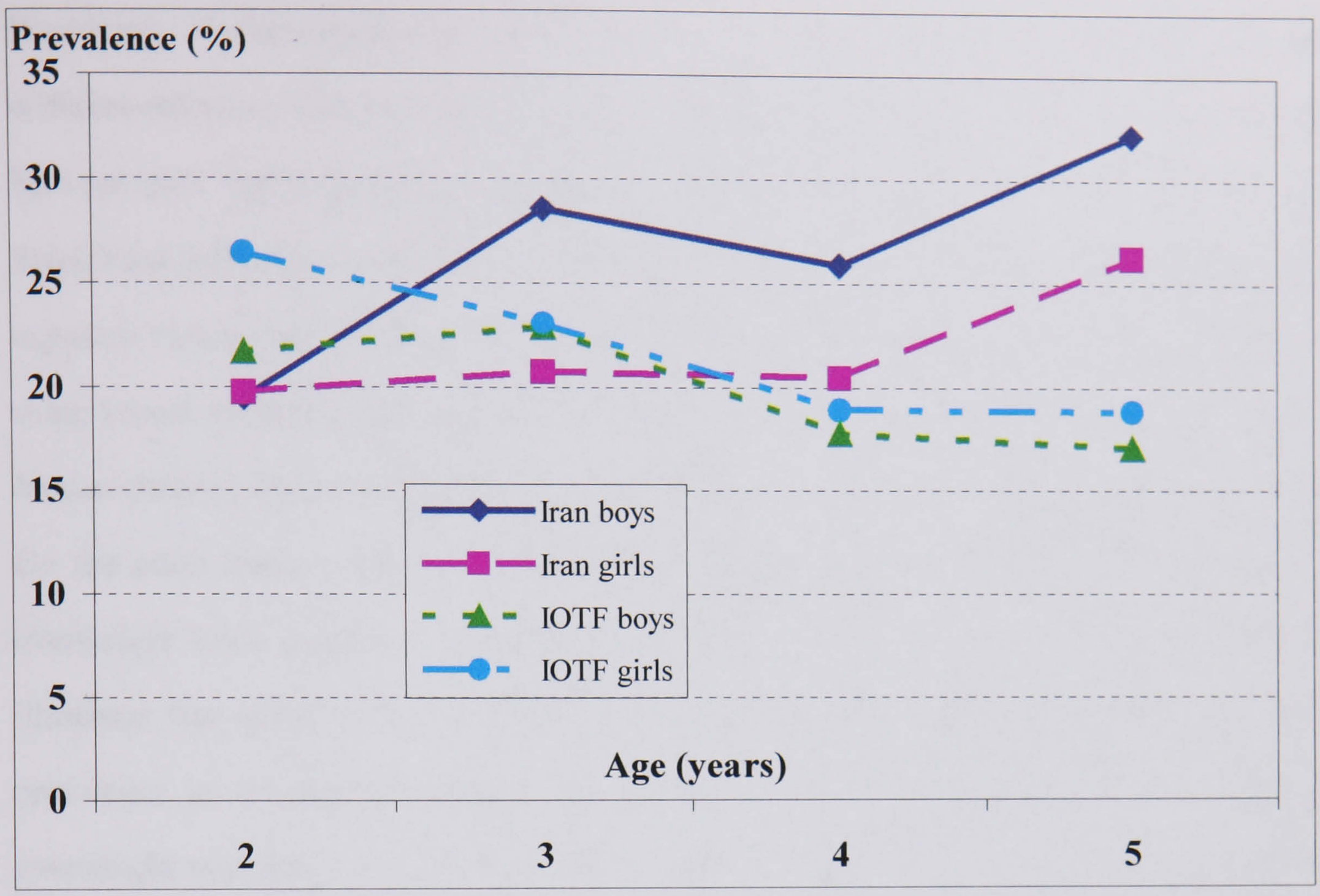
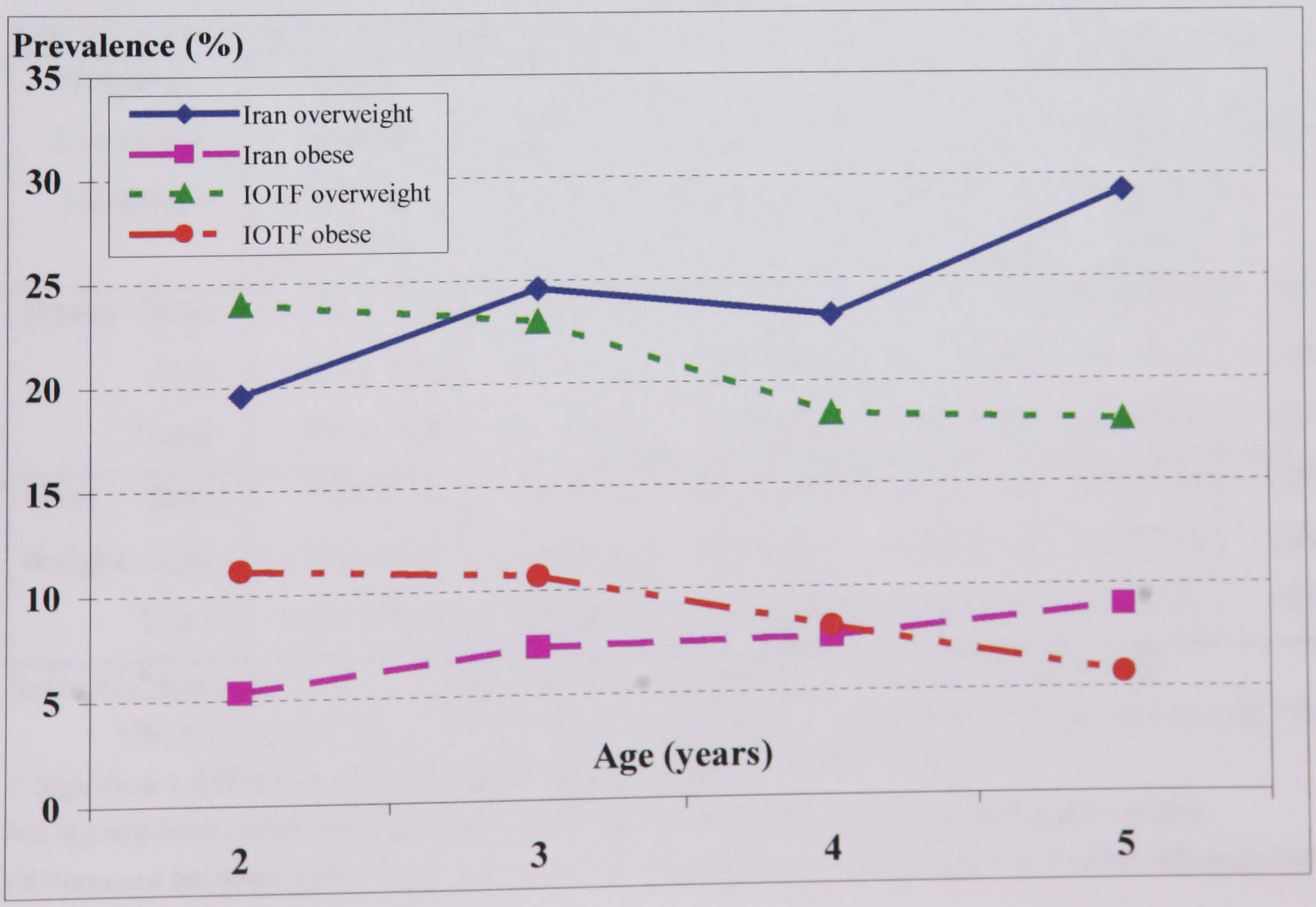


Figure 4.3. Prevalence of overweight / obesity (BMI ≥85th / 95th percentiles using Iranian 1990-1992 and IOTF 2000 reference data) in Iranian children aged 2-5 years.



4.4.2. Study 2

Prevalence of overweight and obesity in Iranian 5 year old children identified using the 5 different reference data sets are shown in Table 4.2. No significant differences in prevalence between girls and boys (using Iranian 1992 reference data; Hosseini et al 1999) were seen. Significant differences between the observed percentage of overweight / obese children and the expected values were found ($p<0.01$) as described for study 1. As can be seen from Table 4.2, using French reference data identified the highest obesity prevalence (17.8%) in the 5-year old Iranian children while using IOTF reference data gave the lowest prevalence of obesity (5.7%). On the other hand, using IOTF reference data identified the lowest (18.0%) prevalence of overweight while Iranian reference data identified the highest prevalence (29.2%). Study 2 illustrates that using different reference data sets resulted in different overweight / obesity prevalence in the Iranian children. For example, using French reference data to calculate overweight prevalence produced a slightly higher prevalence in girls compared to boys while calculating overweight / obesity prevalence using Iranian reference data showed that prevalence was much higher in boys than girls (Table 4.2).

Table 4.2. Prevalence of overweight and obesity (BMI ≥85th and ≥95th percentiles) in Iranian 5-year-old children based on four different reference data.

Iranian 5 year old children		Using **Iranian** reference data n (%)	Using **IOTF** reference data n (%)	Using **UK** reference data n (%)	Using **USA** reference data n (%)	Using **French** reference data n (%)	Total n
Obese	Boys	†22 (9.7)	11 (4.8)	29 (12.8)	22 (9.7)	37 (16.3)	227
	Girls	†19 (8.3)	15 (6.6)	24 (10.5)	21 (9.2)	44 (19.3)	228
	Total	†41 **(9.0)**	26 **(5.7)**	53 **(11.7)**	43 **(9.5)**	81 **(17.8)**	455
Over Weight	Boys	†73 (32.2)	39 (17.1)	57 (25.1)	46 (20.3)	62 (27.3)	227
	Girls	†60 (26.3)	43 (18.9)	43 (18.8)	50 (21.9)	63 (27.6)	228
	Total	†133 **(29.2)**	82 **(18.0)**	100 **(22.0)**	96 **(21.1)**	125 **(27.5)**	455

Reference data sets: Iran: Hosseini et al 1999 IOTF: Cole et al 2000
UK: Cole et al 1995 USA: Rosner et al 1998 French: Rolland-Cachera et al 1991

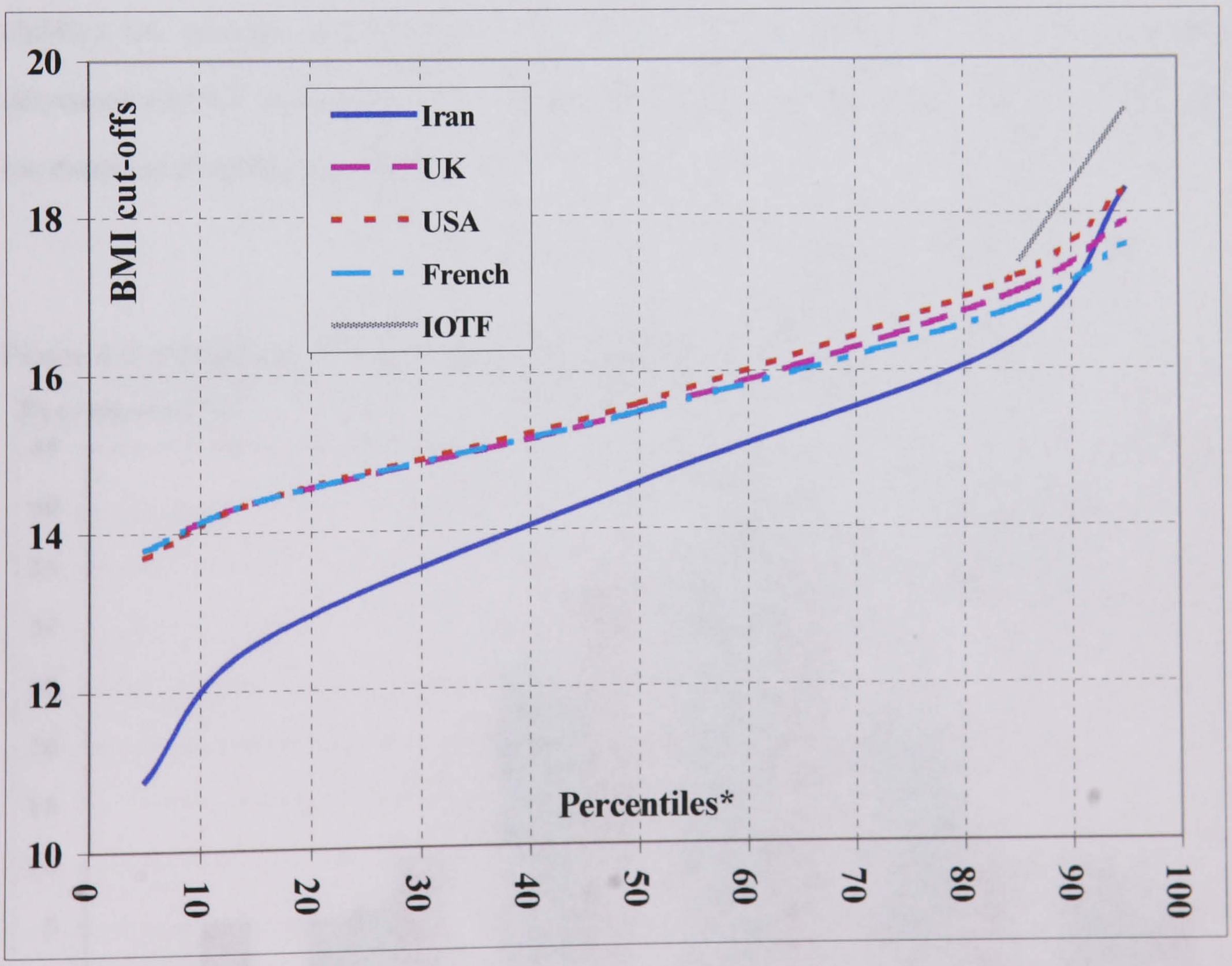
† Significant difference between observed and expected: $p<0.01$

No significance differences between girls / boys (using Iranian reference data) were found.

Differences between girls / boys and observed / expected were tested only for Iranian reference data.

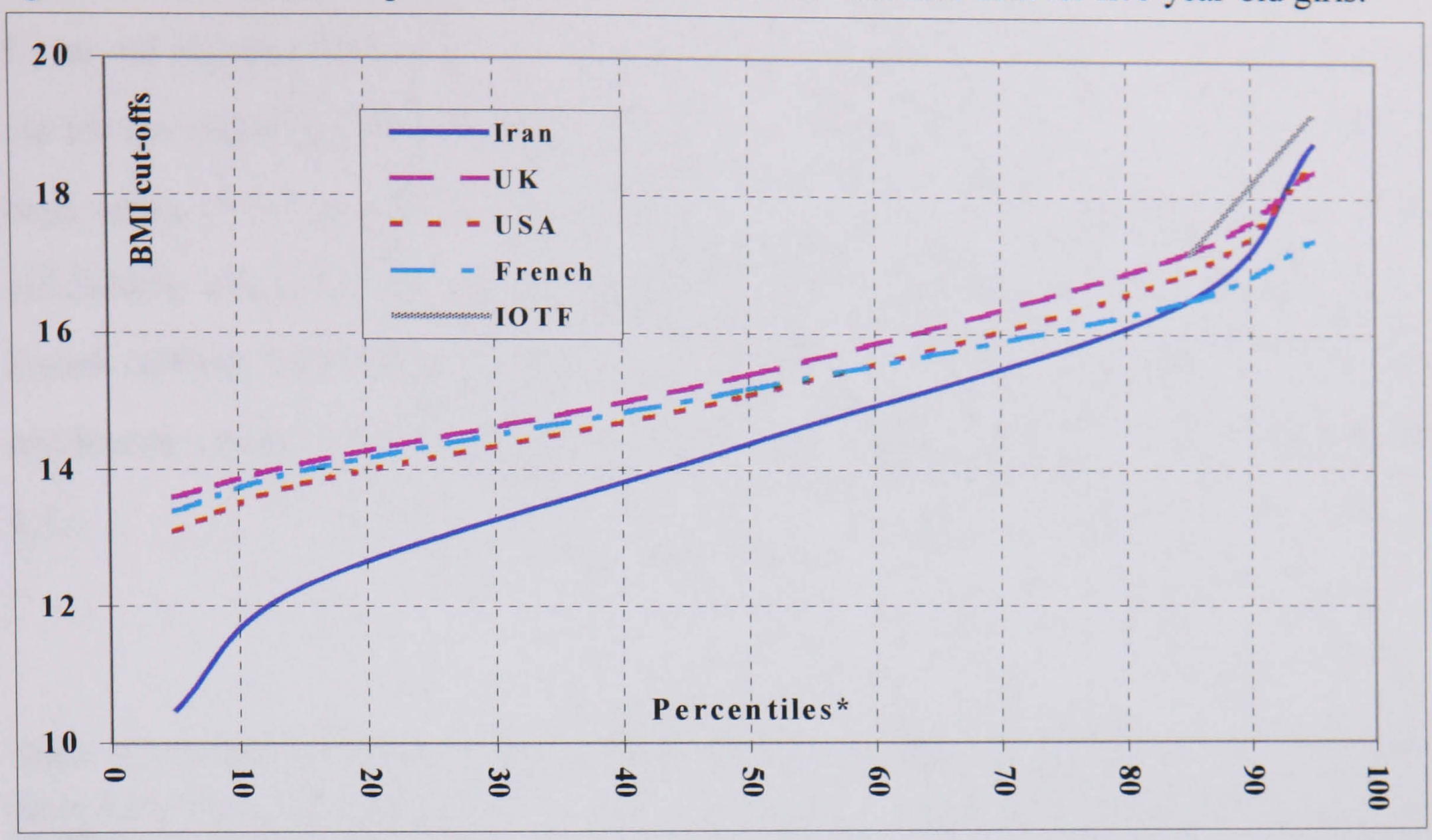
Figures 4.4 and 4.5 illustrate the differences between the percentile / equivalent curves of the 5 different reference data sets used. It is obvious that the Iranian and IOTF reference data sets are quite different from the other 3 USA and European sets at the lower and middle portions of the distribution. It could be argued that the Iranian is the most relevant and IOTF is the most irrelevant reference for comparison in an Iranian population. However, it should be noted that despite the differences in position of the centiles across most of the range, the upper centiles for the Iranian data are more similar to the upper centiles of the other data sets than the lower centiles.

Figure 4.4. Percentile / equivalent* curves: 5 different reference data for five-year-old boys.



* For IOTF, only two equivalents (to BMI>25 and BMI>30 in adults) were presented.

Figure 4.5. Percentile / equivalent* curves: 5 different reference data for five-year-old girls.



* For IOTF, only two equivalents (to BMI>25 and BMI>30 in adults) were presented.

Figure 4.6 confirms that the BMI distribution in the whole population of Iranian 5 years old study children has changed and shifted so that the percentage of children below the lower centiles decreased and the percentage above the middle and upper centiles increased and exceeded from the expected percentages (Figure 4.6).

Figure 4.6. Percentage of 5-year old Iranian children in different centile groups.

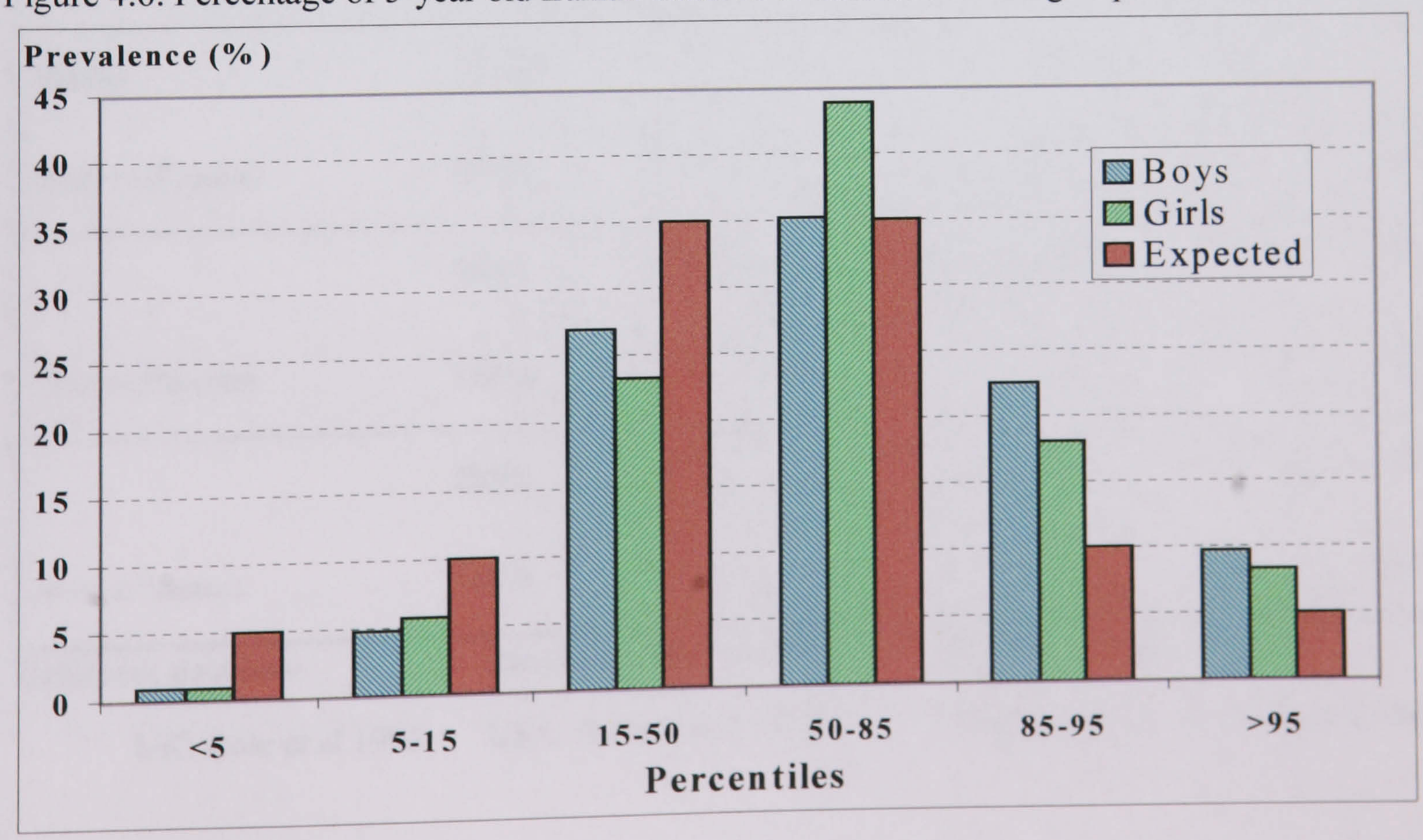


Table 4.3 shows the BMI cut-off values used to calculate overweight / obesity prevalence in the 5 year old children. As can be seen from Table 4.3 and Figures 4.4 and 4.5, the average 5 year old Iranian child (50th percentile) had a BMI less than UK, USA and French children (14.6 for boys versus 15.5-15.6 and 14.4 for girls versus 15.1-15.4). BMI values for those Iranian 5 year old children who were below the 5th BMI percentile were further from the values of UK, USA or French children (10.9 versus 13.7-13.8 for boys and 10.5 versus 13.1-13.6 for girls). UK, USA, and French values for BMI centiles are more similar for boys but diverge a little for girls (Table 4.3).

Table 4.3. Different BMI cut-off values to define different percentiles in 5 year old children from Iran, USA, UK and France.

5 year old children		**Iranian** reference data	**IOTF** reference data	**UK** reference data	**USA** reference data	**French** reference data
Obese (BMI ≥95th centile	Boys	18.3	19.3	17.9	18.3	17.6
or equivalent to adult BMI>30)	Girls	18.8	19.2	18.3	18.5	17.1
Overweight (BMI ≥85th centile	Boys	16.4	17.4	17.0	17.2	16.8
or equivalent to adult BMI>25)	Girls	16.5	17.2	17.2	16.9	16.4
Mean	Boys	14.6	-----	15.5	15.6	15.5
(BMI = 50thcentile)	Girls	14.4		15.4	15.1	15.2
	Boys	12.6	-----	14.4	14.4	14.4
(BMI ≤15thcentile)	Girls	12.3		14.1	13.8	14.0
	Boys	10.9	-----	13.8	13.7	13.8
(BMI ≤5thcentile)	Girls	10.5		13.6	13.1	13.4

Reference data sets: Iran: Hosseini et al 1999 IOTF: Cole et al 2000

UK: Cole et al 1995 USA: Rosner et al 1998 French: Rolland-Cachera et al 1991

Childhood obesity in Iran and UK

In Chapter 3, the results present the prevalence of overweight / obesity in UK children and in this Chapter the Iranian childhood overweight / obesity prevalence were presented. In order to compare UK and Iranian children, these results are shown in Table 4.4.

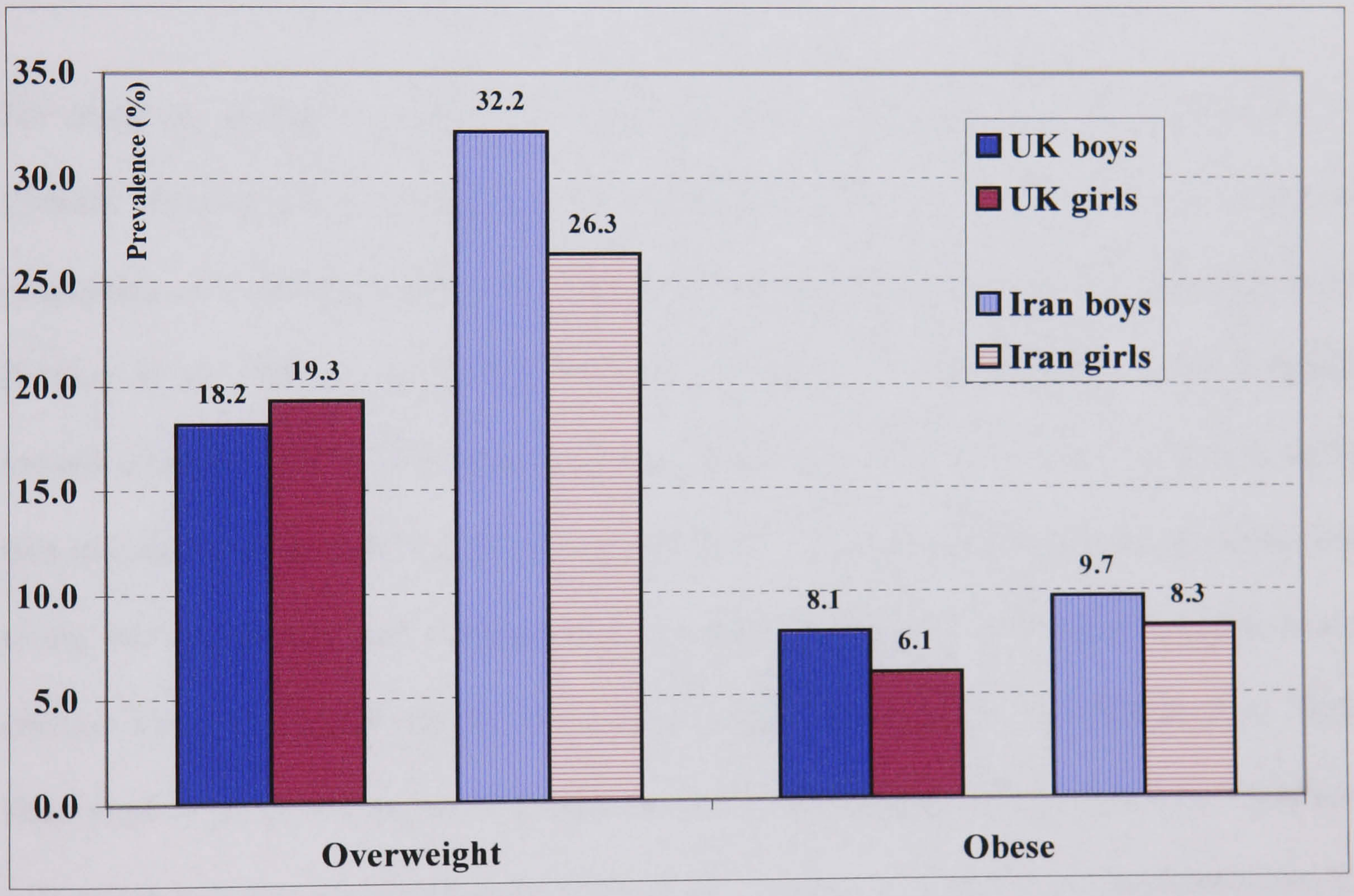
Table 4.4. Prevalence of obesity / overweight (%) in Iranian children (using Iranian 1992 reference data) and UK children (using UK 1990 reference data) at ages 2-5 years.

Age (years)		**Overweight** (%)		**Obese** (%)	
		UK	**Iran**	**UK**	**Iran**
2	Boys	13.9	19.5	5.9	5.8
	Girls	18.1	19.8	6.2	5.0
	Total	**15.8**	**19.6**	**6.0**	**5.4**
3	Boys	17.9	28.6	7.4	9.1
	Girls	21.4	20.8	8.0	5.5
	Total	**19.4**	**24.6**	**7.7**	**7.3**
4	Boys	19.9	25.9	7.5	8.0
	Girls	20.8	20.5	7.8	7.2
	Total	**20.3**	**23.2**	**7.6**	**7.6**
5	Boys	18.2	32.2	8.1	9.7
	Girls	19.3	26.3	6.1	8.3
	Total	**18.7**	**29.2**	**7.2**	**9.0**
2-5	Boys	†17.5	†25.6	†7.2	†7.9
	Girls	†19.9	**†21.0	†7.0	*6.2
	Total	÷**†18.6**	**†23.3**	**†7.1**	**†7.0**

Significant difference between girls and boys: * $p<0.05$, ** $p<0.01$
† Significant difference between observed and expected: $p<0.01$

Table 4.4 presents a comparison between the prevalence of overweight / obesity in 2-5 year old children in the UK (from ALSPAC study described in Chapter 3) and Iran at almost the same period of time and for a similar age group children. Based on Table 4.4 and Figure 4.7, the prevalence of overweight and obesity in Iranian children were a little higher than in the UK, though the obesity prevalence differences were small.

Figure 4.7. Prevalence of overweight / obesity (BMI ≥85th / 95th percentiles) in 5 year old children from the UK and Iran.



4.5. Discussion

During the last one-two decades, extremely rapid economic development in many developing countries has allowed the middle and high income groups to change from a society of limited food consumption to a dietary / physical-activity pattern that has led to

obesity and other chronic degenerative diseases (Popkin et al 1995). Social changes and intensive preventive activities have led developing countries to make major progress in controlling infectious diseases and under-nutrition, and economic and agricultural reforms have resulted in widespread food adequacy and increased income in many of developing countries (WHO 1990). Although under-nutrition and underweight problems, in addition to communicable diseases, still exist among the poor parts of their population (Popkin et al 1995), obesity and coronary heart disease might present a huge health care burden for these countries in the near future (WHO 1990).

For instance, during the period of 1989-1991, an investigation of 3981 20-45 year old Chinese showed that the proportion of underweight adults decreased by 0.4% while the proportion of overweight and obese individuals increased by 1.3% and 0.6% respectively (Popkin et al 1995). It seems that the dietary pattern in China has been moving steadily toward a larger quantity of animal products and a higher fat intake, which are typical of the diet in industrialised countries (Popkin et al 1995). The changing dietary pattern, along with rising life expectancy and urbanisation, may have contributed to increases in diet-related chronic diseases such as obesity and coronary heart disease in China (Popkin et al 1995). Data from a 25 year longitudinal study of rural Guatemalans showed that the childhood population was stunted (mean height-for-age SD score = -2.6), yet had mean BMI above US values (Schroeder & Martorell 1999). Martorell et al (2000) indicate that in a number of countries in Latin America and the Caribbean, the Middle East and North Africa, and the region of Central Eastern Europe / Commonwealth of Independent States, levels of childhood obesity prevalence seems to be as high as in the USA (Martorell et al 2000)

Some investigations have indicated that the environment is more influential on the prevalence of obesity in a population than is ethnicity. For instance, in a study of the growth

of 785 Pakistani school children (52% boys and 48% girls) aged 5-14 years living in the UK in 1989 compared with the 1990 UK growth standards, results showed that children from this ethnic group had very similar growth patterns to UK children (Kelly et al 1997). The mean height for the boys and girls were -0.2 and -0.4 SDS and for weight were -0.3 and -0.5 SDS respectively (Kelly et al 1997). The fact that the sample used in this study came from a deprived background would suggest that children from this ethnic group in better socioeconomic circumstances would be even nearer to the UK standards (Kelly et al 1997). This was confirmed in the study by Chinn et al (1996) which compared the growth trends of 5-11 year old children of different ethnic groups living in Britain in the period of 1983-1994 (Chinn et al 1996). A sample of about 12000 children of different ethnicities including inner city white, English and Scottish, Afro-Caribbean, Urdu or Punjabi speakers, Gujarati speakers and other Indian children were chosen and study results showed that Caribbean children were maintaining their tall, slim build. The Caribbean height increase over the 10-year period showed the influence of environment on enhanced growth of ethnic minority children (Chinn et al 1996).

4.5.1. Obesity prevalence in Iranian adults

When the BMI and weight for height of 2210 adults of Shiraz province of Iran were calculated (Ayatollahi & Carpenter 1993), the authors concluded that Iranian adults were taller and heavier than previously reported for the same population but were shorter than European and US white adults. However, the distribution of BMI was broadly similar to that reported for UK adults (Ayatollahi & Carpenter 1993). Thereafter, in a cross-sectional study in 1993-94 in a sample of 3378 adults aged 20-74 years, in the southern Iranian province of Fars (its centre is Shiraz), BMI was described. The mean BMI was 22.8 for men and 23.6 for women. Highest BMI was found in men in their late forties and women in their early fifties. About 32% of men and 33% of women were overweight (BMI >25) and 8% of women and 3% of men were obese (BMI >30; Pishdad 1996).

These studies in part of the Iranian population cannot show the complete picture of obesity in Iranian adults or exclusively define any trend in obesity. However, they tend to suggest that obesity has become a problem in Iranian adults. On the other hand, prior to the present study there was no study on overweight / obesity prevalence in Iranian children. However, the prevalence of obesity and overweight in Iranian children in the present study, when compared with 1992 Iranian reference data, suggests that there is a trend of increasing obesity in Iranian children and that this even affects young children under the age of 5 years, as in the UK (Reilly et al 1999a; Chapter 3).

4.5.2. Study 1

In this study, the prevalence of overweight / obesity in 2 year old boys and girls (in 2 Iranian provinces) were almost identical but from the age of 3 years, large differences between prevalence of overweight / obesity between the sexes were found. Thereafter, obesity prevalence in boys was almost always higher but between-sex differences were only statistically significant at age 3 (Table 4.1 and Figures 4.1 to 4.3). The lowest percentage of overweight / obese children was at age 2 years in both boys and girls, while it increased as age increased and, therefore, 5 year old children had the highest prevalence of overweight / obesity (Table 4.1). This trend towards increasing prevalence with age might indicate a similarity to the UK and USA, in that the social changes which have led to increasing obesity, have a greater effect over time.

4.5.3. Study 2

Results showed that using UK, USA, French, and/or IOTF reference data to define overweight / obesity prevalence in Iranian 5 year old children (Table 4.2) identified different levels. As can be seen from Table 4.3 and Figures 4.4 to 4.5, the 85th centiles which identify overweight occur at a lower level of BMI in Iranian reference data (16.4 & 16.5 kg/m^2 for boys & girls respectively) than in UK reference data (17.0 & 17.2).

Using national growth standards, identified more children as overweight in Iran than in the UK although the level of obesity was very similar (Table 4.4 and Figure 4.6). In Iran more boys than girls were identified as overweight whereas in the UK it was the girls who were more likely to be identified as overweight. As the results of Chapter 3 indicate, the prevalence of obesity in the USA in 1987-91 and 1992-94 were similar to the UK in 1995. Therefore, due to high prevalence of obesity in Iranina study children, it seems that the prevalence of overweight / obesity in 2 Iranian provinces are increasing. For countries such as Iran, which have national BMI reference data, their reference data should presumably be used but for those countries which do not have reference data, the correct approach is less obvious and, as study 2 showed, the choice of definition / cut-off can make an important difference to the prevalence observed.

No data on obesity prevalence on other developing countries were calculated based on the same definitions (BMI $\geq 95^{th}$ percentile) to compare with the literature. The WHO scientific report (WHO 1998) suggested that the prevalence of obesity (using weight for height standards >120%) in Iranian children was high and similar to that in Canada and much higher than in some developing countries like Bangladesh and India (WHO 1990); even some developed countries like Italy had a lower prevalence of obesity than Iran. Since the definition used by WHO (weight for height standards >120%) is not a useful criterion to define obesity, and has major weaknesses and limitations (Krick 1986), such results might not be very useful. In addition, the age groups, sample sizes, sampling method, and the year(s) of data collection were not described in the WHO report clearly and so it is difficult to assess how valuable a contribution the WHO report (WHO 1990) is to the problem of obesity in Iran.

Results of the first study showed that the prevalence of overweight / obesity in Iranian children in these 2 provinces has increased. Since the present study had a reasonable sample size, and used the new agreed obesity definitions based on BMI percentiles and recent Iranian reference data (Hosseini et al 1999), the results of the present study present the most accurate and up-to-date information about the prevalence of overweight / obesity in Iranian 2-5 year old children. This work suggests that further research on the epidemiology of obesity is indicated. This might take the form of assessing the prevalence of severe obesity in older children, and in other parts of Iran.

4.6. Study limitations

As mentioned in Chapter 3, it is well known that BMI is not an ideal definition for screening obese children but it is acceptable enough for most clinical and epidemiological purposes (Barlow & Dietz 1998). This Chapter provides a picture of obesity in Iranian children in different age groups (2-5 years of age) in 1995. Because of the low sensitivity of BMI discussed in Chapter 5, the estimates of obesity prevalence in this Chapter are probably underestimated.

Another specific limitation of the survey is the lack of data from different Iranian provinces (it consisted of data only from two provinces: Gilan in the north and Sistan in the south). The population of these two provinces might be slightly different from other Iranian provinces in terms of childhood obesity prevalence. In study 2 the sample size was small and dividing five year old Iranian children who participated in study 2 into different sex groups reduced the sample size further, though they were sampled appropriately (using simple random sampling methodology). In studies 1 and 2, the age of children was rounded to the nearest integer values in the original survey. For example, 'two year old children' consisted of all 1.50 to 2.49 year old children. This probably affected the results of children under 5. Based on the trend of obesity / overweight we have seen in Chapter 3, such a trend could possibly continue in older age children and adolescents, but unfortunately no data for older children in Iran was available in the present studies, and no national surveys using BMI were available after the Hosseini et al (1999) baseline survey.

Data gathering as well as all other activities were validated in the Iranian study but the present study might have some minor causes of bias or errors. These include: no data are available on differences in prevalence between different ethnic groups / races in Iran; the scales used in the study might cause small errors; children were weighed in light indoor clothing.

4.7. Conclusions

The present studies provide information about the prevalence of obesity in Iranian children in the mid-late 1990s. BMI SDS values and percentiles were used to define obesity prevalence. Few studies in the literature were concerned with adult obesity in Iran, and there was no information about childhood obesity prevalence in Iran prior to the present studies.

In the present study, the prevalence of overweight and obesity in boys was always higher than that of girls from the age of 3 years and these differences between boys and girls were generally statistically significant. The prevalence of obesity significantly exceeded 'expected' frequencies at all ages except at 2 years. From age 3 years, the prevalence of overweight / obesity started to increase. These trends were similar to the results of the UK children described in Chapter 3. The highest prevalence of overweight / obesity was at age 5 (Figure 4.3) though children were not studied beyond this age. The increase in Iranian childhood obesity relative to Iranian 1992 reference data (Hosseini et al 1999) shows a possible trend to increasing BMI over time, across the whole distribution of BMI.

Using USA reference data gave almost the same results for the prevalence of obesity in 5 year old Iranian children but the results for the prevalence of overweight were much lower than the results of such calculations using Iranian 1992 reference data (Table 4.2). There were differences between the results using UK and Iranian reference data sets (Table 4.2) for both obesity / overweight prevalence. For developing countries the need to estimate obesity prevalence in children will increase, but study 2 shows that the choice of reference data can have a large effect on the apparent prevalence and that such a choice must be made with care. Unfortunately at the time of writing, no data on obesity prevalence in other developing countries using the same definitions (BMI $\geq 95^{th}$ percentile) were available to make comparisons.

5. Chapter 5: Defining the obese child: sensitivity, specificity and receiver operating characteristic (ROC) analysis of the optimal simple obesity definition

5.1. General introduction

The costs and problems of obesity at individual and public health levels are discussed in Chapters 1, 3, 4, and 6. As described, obesity prevalence in many countries is increasing in children and adults. Currently, obesity contributes to about 300,000 deaths annually in the USA, and the health care costs associated with obesity-related illnesses in adults exceed $60 billion per year in the USA (Sokol 2000). Concern over trends of increasing fatness in British children is considerable (Hughes et al 1997). In addition, recent studies (published after the present study was undertaken) indicate a marked increase in childhood obesity in the UK (Prentice 1998; Reilly & Dorosty 1999; Reilly et al 1999a; Kinra et al 2000; Sahota et al 2000; Bundred et al 2001; Chinn & Rona 2001; Rudolf et al 2001; Chapter 3). Such trends have caused increased interest in methods to define and distinguish the obese child. The question of whether or not such definitions are appropriate for clinical practice as well as epidemiology is important (Reilly 1998). This Chapter is therefore concerned with methods for the definition of paediatric obesity.

5.1.1. Adverse consequences of childhood obesity

Childhood obesity has several adverse psychosocial consequences including anxiety and low self-esteem. In young adulthood obese individuals in the USA and European countries experience lower education, higher poverty rate, lower marriage rate, and less income (Laurier et al 1992; Gortmaker et al 1993; Sargent & Blanchflower 1994; Dietz 1998a;

Martinez et al 1999). The early accumulation of both peripheral and intra-abdominal adipose tissue in childhood is important because it has a significant relationship with adverse health. These complications include orthopaedic complications (e.g. slipped capital femoral epiphysis and Blount disease), psychological disturbances, pseudotumor cerebri and headaches, sleep apnoea and hypoventilation syndrome, gall bladder disease, non-alcoholic steatohepatitis, polycystic ovary disease, glucose intolerance, insulin resistance and type II diabetes mellitus, hyperlipidemia, dyslipidemia, hypertension and coronary diseases (Goran 1997; Dietz 1998a; Lynch et al 2000; Sokol 2000). Excess central body fat, even in children, can be linked independently with negative health outcomes (Goran et al 1995; Slyper 1998; Goran & Gower 1999; Cowin & Emmett 2000). There is an association between increases in BMI and high blood pressure, adverse lipoprotein profiles, non-insulin-dependent diabetes mellitus, and early atherosclerotic lesions in children and adolescents (Dietz & Robinson 1998; Fredriks et al 2000). Further details of the adverse consequences of childhood obesity were discussed in Chapter 1.

5.1.2. Definition of obesity

Adiposity is the amount of fat in the body, expressed as total fat mass (in kg) or the fraction (%) of total body fat, and overweight describes excess body weight (Power et al 1997; Bellizzi & Dietz 1999). Obesity is an excess of body fat. Therefore, an obesity definition needs a suitable cut-off to define excess fat (Power et al 1997). Body fatness is extremely difficult to measure in infants and young children, because accurate techniques require a high degree of subject compliance and are usually only available to specialised research institutions (Wells 2000). There are also two important differences between childhood and adulthood in the assessment of fatness. First, children grow in size, so that anthropometric cut-offs for fatness need to be adjusted for the age of children and maturation of adolescents

(Power et al 1997). The second difference is that the levels of body fatness in childhood associated with later adult disease or premature death are unclear (Power et al 1997).

An ideal measure of body fat should be accurate in its estimate of body fat; clinically valid or able to predict morbidity and mortality; precise, with small measurement error; accessible, in terms of simplicity, cost and ease of use; acceptable to the subject; and well-documented, with published reference values (Kraemer et al 1990; Power et al 1997). According to Power et al (1997) no existing measure satisfies all these criteria perfectly. Relatively accurate methods like deuterium dilution or underwater weighing are expensive, and more accessible cheaper methods based on anthropometry have been considered as not entirely accurate (Power et al 1997). However, for screening purposes anthropometry is the only realistic method because of its simplicity and low cost (Power et al 1997).

5.1.3. Obesity screening methods for clinical practice and epidemiology

5.1.3-a. Adults

The traditional definition of percent of 'ideal body weight' in adulthood was derived from data based on the mortality outcome of a large cohort of insured subjects (Kimm 1995). Nowadays, adults are classified as overweight by a variety of cut-offs based on mortality experience of life insurance policy holders, statistical criteria from a reference population, and the relationship between morbidity / mortality to weight status (Dietz & Robinson 1998; Troiano & Flegal 1998). Based on a recent US classification in adults, a BMI of 25 to 29.99 typically identifies grade 1 overweight, a BMI of 30.0 to 39.99 constitutes grade 2 overweight, and a BMI ≥40.0 defines grade 3 obesity (Dietz & Robinson 1998). In the UK, adult obesity is more simply defined as BMI >30 (Bolton-Smith et al 2000; Figueroa-Colon et al 2000).

5.1.3-b. Children

It has been suggested that obesity in children should be defined on a *biological* basis, via the relationship of body fatness to clinical outcomes, rather than using proxy measurements for fatness such as BMI (Dwyer & Blizzard 1996). Although such definitions of obesity might be ideal, using biological definitions in clinical practice and epidemiology is difficult since only simple proxy measurements for body fatness are possible and because there is some doubt as to the precise point at which fatness in childhood leads to adverse clinical outcomes (Dwyer & Blizzard 1996).

In a clinic setting, assessment of nutritional status is often based upon an overall global assessment of nutritional status by observation and simple clinical examination (Cross et al 1995). However, in one study, the ability of three experienced childcare professionals (a consultant paediatric oncologist, a senior dietitian, and a senior clinical nurse specialist) to grade the nutritional status of patients of varying ages and nutritional status was measured but the accuracy of their assessments was poor (Cross et al 1995). Reproducibility in clinical assessment of nutritional status is poor, even among senior staff. It seems plausible that more junior staff would be even less accurate (Cross et al 1995). This example shows that agreed objective measures using anthropometric evaluation are necessary for the accurate assessment of nutritional status in children, including obesity, as recommended by expert committees (e.g. Barlow & Dietz 1998).

5.1.4. Anthropometric indices of overweight

Weight is a simple and direct index of body size, easy to measure, cheap and reproducible. It is also reasonably highly correlated with body fat (Power et al 1997). Like weight, height is simple and cheap to measure. Unlike weight it needs trained observers and continuous

quality control to ensure high precision, but this is less important in the context of measuring adiposity (Power et al 1997). Weight is highly correlated with height but height is only weakly correlated with body fat. This means that weight adjusted for height is far more useful than weight alone as an index for assessing fatness (Power et al 1997). Using a weight for height index has its weaknesses; e.g. in adulthood, individuals who are unusually muscular, for example athletes, may be heavy but not fat, and so screen false positive for fatness (Power et al 1997). In addition, self-reported values of weight and height in adolescence tend to be biased in opposite directions, with weight underestimated and height exaggerated, and the weight error is larger in heavier subjects (Power et al 1997).

A useful index of overweight is provided by the function of weight / height to the power of n, where the power is between 1 to 3. To choose a whole number, the value which best adjusts weight for height, and at the same time removes most of the trend of increasing weight with age, n=2. This measure is BMI which also is known as the Quetelet index or the Kaup index (Cole et al 1995). BMI, like other weight for height indices, is cheap and easy to perform and calculate and therefore useful in diagnosing overweight (Fredriks et al 2000). BMI has been used widely in adults for the last 25 years as a simple summary measure of overweight and its use in childhood has developed relatively recently (Cole et al 1995; Barlow & Dietz 1998). Adult BMI increases slowly with age, so that age independent cut-offs can be used to grade obesity. In children, BMI changes substantially with age, rising steeply in infancy, falling during the pre-school years, and then rising again into adulthood. For this reason, childhood BMI needs to be assessed using age related reference curves (Cole et al 1995). BMI is widely accepted in adulthood and BMI charts are less affected by differences in the timing of puberty than weight for height charts (Fredriks et al 2000). BMI SDS values are used to allow for a more consistent interpretation between sexes and ages

(Fredriks et al 2000). BMI, however, is the best (though imperfect) measurement for routine clinical assessment of adiposity (Robinson 1993; Power et al 1997; Barlow & Dietz 1998).

Defining children as overweight or obese relative to a selected percentile of a reference population based on age and sex is a statistical approach (Troiano & Flegal 1998). It is also an outcome-based criterion (Barlow & Dietz 1998) since children at the upper end of the BMI distribution (e.g. BMI $\geq 95^{th}$ centile) have increased risk of morbidity in childhood and in adulthood (Barlow & Dietz 1998) as discussed in Chapter 1. Choosing a particular percentile to define obesity (e.g. BMI $\geq 95^{th}$ centile) entails the assumptions that the proportion of the reference population that exceeds the cut-off is overweight / obese and that the prevalence of overweight / obese is the same for the groups defined by sex, age, or other characteristics (Troiano & Flegal 1998).

As previously noted, an expert committee in the USA has suggested that a BMI $\geq 95^{th}$ percentile should be considered as a screening criterion to define obesity in children and adolescents (Barlow & Dietz 1998). In fact, BMI is now widely used in paediatric studies to define obesity (Power et al 1997; Reilly 1998; Reilly & Dorosty 1999; Reilly et al 1999a; Troiano & Flegal 1999; Kinra et al 2000; Chapters 3-4). A very recent approach is to use the childhood BMI which is 'equivalent' to adulthood BMI (designed by the International Obesity Task Force [IOTF] childhood obesity working group) comparable to the typical cut-offs of BMI >25 (for overweight adults) and BMI >30 (obese adults; Cole et al 2000). Based on international reference data, this has been recommended for international comparisons of childhood obesity prevalence. Within the UK the recommendation is to continue using UK 1990 reference data (Cole et al 1995) for clinical purposes (Prentice 1998; RCPCH 2001; Wright et al 2002).

5.1.5. BMI as a simple childhood obesity definition method

5.1.5-a. Disadvantages of BMI

Body weight is correlated with body fat but is also highly correlated with height, which is weakly correlated with body fat (Bellizzi & Dietz 1999). It is important to note that the correlation between BMI and height causes taller populations to display a higher prevalence of obesity (Bellizzi & Dietz 1999). In addition, obesity is an excess of body fat, not an excess of body weight (Wells 2000). This means that having a higher BMI does not necessarily mean being fatter. In a minority of children heavy musculature can be confused with obesity (Fredriks et al 2000). While BMI is highly correlated with fatness in childhood (Pietrobelli et al 1998), the relationship between BMI and body fat percentage in *individuals* has been shown to be poor both in infancy and childhood (Fredriks et al 2000; Wells 2000). In children and adolescents the correlations range from 0.39 to 0.90, depending on the method of body fat measurement and the age and sex of the subjects (Fredriks et al 2000). Although growth consists of both fat mass (FM) and fat-free mass (FFM), it is generally described in terms only of body weight, which is then normalised for height to give BMI (Wells 2000). Since BMI is a proxy measure of FM and FFM, changes in FFM will not be easily distinguished using BMI (Hughes et al 1997). This is a disadvantage of using BMI for epidemiology since population trends in FM can be hard to distinguish from population trends in FFM (Kuczmarski 1993; Hughes et al 1997). In the study of Hughes et al (1997), during the period of 1972-1994 changes in triceps skinfold clearly showed an increase in fatness in British children while BMI changes were less marked. BMI was not sensitive enough to accurately describe population trends in fatness in British children during the 1970s and 1980s (Hughes et al 1997).

BMI changes dramatically with age during childhood and adolescence. Weight and height change during the process of development are different. During growth and maturation, body proportions, bone mass, and the ratio of lean-to-fat mass change at different rates (Reilly et al 1995; Troiano & Flegal 1998). Male infants have a consistently higher FFM throughout infancy, which accounts for greater body mass. Adipose tissue deposition rates vary with age (growth and maturation) as well as gender (Pietrobelli et al 1998; Wells 2000). Therefore, interpretation should be cautious when BMI is compared across groups that differ in age and sex (Pietrobelli et al 1998). For example, the mean BMI for children at ages 6 to 7 years is about 16, while it is about 22 at 16-17 years of age. Therefore, overweight criteria based on BMI must be age-specific (Troiano & Flegal 1998).

In addition to age and sex effects, ethnicity, sexual maturity, sitting height, fat distribution and disease state have all been shown to affect the general relationship between BMI and percentage fat (Wells 2000). BMI is dependent on stature (especially at younger ages) and sitting height, therefore, persons with short legs have relatively higher BMI values (Fredriks et al 2000). BMI percentiles in 5-17 year old US children published in 1998 offered different cut-off values for Asian, Black, Hispanic and White ethnic groups (Rosner et al 1998) which recognised the effect of ethnicity / race on the relationships between BMI and body fatness. Daniels et al (1997) studied 7-17 year old children and showed that BMI was not an equivalent measure of the percent body fat in different race and sex groups (Daniels et al 1997): for equivalent BMI, whites had higher body fat percent than blacks, and for equivalent BMI, girls had greater amounts of body fat than boys.

BMI is correlated with fatness but in both sexes, at any BMI a range of body fatness values has been observed (Wells 2000). BMI cannot accurately predict a specific individual's total

or percentage body fat (Pietrobelli et al 1998). It could even be argued that BMI should be used as a surrogate of percent body fat (PBF) in-groups rather than for individuals (Pietrobelli et al 1998). In addition, in both adults and children increased intra-abdominal fat is a specific cardiovascular risk factor. Therefore, distinguishing between abdominal and central fat is important, but BMI does not make this distinction and cannot be used to classify fat distribution (Goran et al 1995).

5.1.5-b. Advantages of BMI

Inter-observer and intra-observer reliability for triceps skinfold thickness measurements are more difficult to define than BMI since reliability decreases as body fat increases. In addition, no efficient method exists to assess reliability across surveys (Dietz & Robinson 1998). Measures of height and weight are simple and provide more reliable measures of fatness than other measures (Dietz & Robinson 1998). As noted above, BMI charts for children, therefore, have been recommended for clinical use in the UK (Prentice 1998; RCPCH 2001; Wright et al 2002). The majority of large-scale studies reporting increasing adiposity in children have used BMI as an index of body fatness, and BMI has been recommended as the best measurement for monitoring overweight in individuals in the paediatric population (Wells 2000). However, in clinical settings a direct measure of body fat such as triceps skinfold thickness can be useful to confirm that a high BMI is really high body fat or is just an increase in frame size / muscle mass (Dietz & Robinson 1998). As noted above and in Chapter 1, BMI is related to health outcomes in childhood and is predictive of disease (Dietz 1998a). In summary, despite all the disadvantages of BMI, there are a number of important advantages for using BMI, and comparing it against a reference population to define children as overweight / obese is now widely recommended (Cole et al 1995; WHO 1995; Power et al 1997; Barlow & Dietz 1998; Prentice 1998).

5.1.6. BMI as an obesity screening tool

Although it is widely believed that BMI cut-offs are not specific and can therefore identify non-obese children as obese (Reilly 1998), low sensitivity is a bigger problem which can cause obese children to be defined as non-obese (Kuczmarski 1993; Warner et al 1997; Schaefer et al 1998). In addition, the sensitivity and specificity of BMI can be different between boys and girls (Warner et al 1997; Schaefer et al 1998) but this was not shown by some other studies (Kuczmarski 1993; Warner et al 1997; Reilly et al 1999a). However, if this difference between boys and girls is real, then it means that different obesity definitions based on BMI would be needed for boys and girls. Variation of body fat percentages (BF%) in subjects with the same BMI (in the same age, sex and ethnic group) indicates that using BMI for measuring BF% is problematic but the relationship between BMI and BF% provides evidence that BMI might be useful to distinguish obese subjects (though with moderate sensitivity). A summary of previous obesity screening studies in children and adolescents is given in Table 5.1.

Although using BMI to define obesity in children is now widely recommended, no studies to date have assessed the ability of BMI to define obesity in British children by using centile cut-offs in conjunction with UK 1990 reference data (Cole et al 1995). Previous studies used particular cut-offs (like BMI $\geq 95^{th}$ centile) and did not assess the sensitivity and specificity of a wide range of different BMI cut-offs to find the best BMI centile, which has the highest sensitivity and specificity in defining the obese child. In addition, previous studies were generally based on relatively small sample sizes (Table 5.1) and no study to date has tested the screening ability of the new IOTF 2000 cut-offs (Dietz & Bellizzi 1999; Cole et al 2000).

Table 5.1. Summary of obesity screening studies in children, adolescents, and young adults.

Study	Sample size	Age (years)	True positive obesity definition Method	Cut-off	Simple indices tested	Conclusion
Marshall et al 1991	540	7-14	Body density (using Lohman's equation)	BF ≥25% boys BF ≥30% girls	Relative BMI, sum 4 skinfolds, triceps skinfold	Relative BMI: 69% sensitive, 93% specific, 89% accurate Sum 4 skinfolds: 80% sensitive, 90% specific, 88% accurate Triceps skinfold: 64% sensitive, 95% specific, 90% accurate
Lazarus et al 1996	119 male 111 female	4-20	DEXA	BF%>85th centile	BMI centiles 5-95 Increment 5	BMI >85th centile had the optimum sensitivity and specificity of 71% & 95%
Warner et al 1997	79 boys 64 girls	10-16	DEXA & skinfolds (Brook equation)	BF%>85th centile	BMI SDS>1.0 (~ 85th centile)	Sensitivity & specificity 66% & 94% vs. DEXA Sensitivity & specificity 50% & 100% vs. skinfolds
Schaefer et al 1998	1276 boys 1278 girls	6-19	2 skinfolds (Slaughter equation)	BF% >75th, 85th, 90th, & 95th centile	BMI >66th, >75th, >85th, >90th, and >95th centiles	BMI >75th centile had optimum sensitivity (82%) and specificity (98%)
Malina & Katzmarzyk 1999	1570	9-19	Siri: Hydro-densitometry	BF ≥25% boys BF ≥30% girls	BMI >85th & >95th centiles	BMI>85th: 4-75% sensitive & 86-99% specific BMI>95th: 14-60% sensitive & 96-100% specific in 6 different groups
Reilly et al 1999b	124 boys 116 girls	8-9	2 skinfolds (Slaughter equation)	BF ≥25% boys BF ≥32% girls	BMI SDS>2.00 (= 97.7th centile)	BMI low sensitivity (boys: 36%, girls: 60%) but high specificity (98%)
Sardinha et al 1999	165 boys 163 girls	10-15	DEXA	BF ≥25% boys BF ≥30% girls	BMI >19.6-21.9 Triceps >21-24 upper arm girth>24-26	Triceps skinfold thickness gave the best results, BMI and upper arm girth were reasonable alternatives, except in 14-15 year old boys
Present study 2000	2120 boys 2055 girls	7y	Bioeletrical impedance	BF ≥24.2% boys BF ≥29.2% girls	BMI >74th - 99th Centiles	74th high sensitivity and low specificity, 92th had the best sensitivity & specificity (92%&92%) 99th low sensitivity and high specificity.

*BF = body fat
DEXA = dual energy x-ray absorptiometry
Relative BMI: BMI / BMI at 50th centile for age & gender multiplied by 100

5.2. Aims

1. To assess the ability of BMI to define obese children by using the 95th centile cut-off (Barlow & Dietz 1998) and UK 1990 reference data (Cole et al 1995)

2. To identify the cut-offs for BMI using UK 1990 reference data with highest sensitivity and specificity in obesity screening.

3. To determine the sensitivity and specificity of the IOTF 2000 alternative definition of obesity based on BMI ('equivalent' to the adulthood BMI cut-offs of BMI >25 for overweight adults and BMI >30 for obese adults). Particular cut-offs for boys and girls to define them as overweight & obese at age 7 years were 17.4 & 19.3 (kg/m^2) for boys and 17.2 & 19.2 (kg/m^2) for girls respectively (Cole et al 2000).

5.3. Methods

5.3.1. Finding the best BMI cut-off: ROC curve

When screening for obesity, ideally all obese subjects should be classified as obese and all non-obese ones should be classified as non-obese. Sensitivity and specificity are two indices of the performance of a test which consider how often such correct classification occurs (Hulley & Cummings 1988). However, when making a decision it is possible to:

- Correctly make a true positive assessment (when the individual is obese and test confirms it).
- Correctly make a true negative decision (the individual is not obese and test rejects him / her).
- Incorrectly make a false positive decision (when the individual is non-obese but test identifies him / her as obese).
- Incorrectly make a false negative decision (when the individual is obese, but the test rejects him / her; Anthony 1996).

Sensitivity: the percentage of obese individuals who are classified as obese is the *sensitivity* of a test. A test is sensitive to obesity if it is positive for most obese individuals (Hulley & Cummings 1988). Sensitivity is identical to the *true positive ratio,* which is defined as the proportion of all obese cases who are correctly identified as obese (Anthony 1996). A test is a *true positive test* if the test is positive and the subjects is obese, while a *false positive test* is a positive test of a non-obese person (Hulley & Cummings 1988).

Specificity: the percentage of non-obese individuals who are classified as non-obese is the *specificity* of a test. A test is specific to obesity if it is positive for a small percentage of non-obese individuals (Hulley & Cummings 1988). Specificity is identical with the *true negative ratio* (Anthony 1996). A test is a *true negative test* if the test is negative and the subject is non-obese, while a *false negative test* is a negative test of an obese person (Hulley & Cummings 1988).

Predictive values: the *positive* predictive value is the percentage of subjects with a positive test who are obese, and means the probability that individuals above the chosen cut-off actually are truly obese (Robinson 1993). The *negative* predictive value is the percentage of subjects with a negative test who are non-obese and refers to the probability that individuals below the chosen cut-off actually are truly non-obese (Robinson 1993; Hulley & Cummings 1988).

As an example from the present study: using the definition of the top 5% of the body fatness distribution as a true positive (197 individuals of 3948 total), 173 were defined as obese using BMI $\geq 95^{th}$ centile (Table 5.4), which means this cut-off was 88% sensitive (173/197 = 88%). In this example, 173 cases were truly positive i.e. they were really obese using the

working definition (BMI ≥95th centile), while the other 24 obese subjects were not defined by this BMI cut-off (false negatives). Using the BMI cut-off ≥95th centile, from 3751 non-obese children, 3539 (93.5%) were identified as non-obese (true negatives). The other 212 non-obese children who were not defined as non-obese (and therefore were defined as obese) were false positives. As can be seen in Table 5.4, positive predictive and negative predictive values for BMI ≥95th centile were about 45% and 99%, therefore, 45% (173 true obese out of 385 obese defined by BMI) of subjects with a positive test were obese and 99% (3539 true non-obese subjects defined out of 3663 who were defined as non-obese by BMI) of subjects with a negative test were non-obese.

The Receiver Operating Characteristic (ROC) plot is one way to find out to what extent the test results for sensitivity / specificity differs among different cut-offs (Altman & Bland 1994) or in this case obesity definition. The ROC plot allows assessment of the optimum balance between sensitivity and specificity (Anthony 1996). When drawing the ROC plot, sensitivity is plotted against 1-specificity. When a 'curve' which coincides with the left and top sides of the plot is drawn, then the test is perfectly able to discriminate between two 'low-sensitive high-specific' and 'high-sensitive low-specific' groups (Altman & Bland 1994). On the other hand, a test might be completely useless if produces a straight line from the bottom left corner to the top right corner (Altman & Bland 1994). The ROC plot is a powerful procedure for assessing screening tools (Anthony 1996). Each cut-off (in the case of the present study, BMI cut-off) has a unique sensitivity and specificity and therefore particular point in the ROC diagram. By plotting the points for a range of different cut-offs, the best cut-off (which has the highest plotting specificity and sensitivity) can be defined usually at the point where the curve turns sharply (Anthony 1996).

5.3.2. Subjects

As described in detail in Chapter 2, in 1990 a sample of 14893 pregnant mothers and their partners participated in the ALSPAC study and their off-spring were entered in the longitudinal study of Children of the 90s. About one thousand of those children were chosen as Children in Focus and were followed-up by investigators every few months and at each visit their weight and height were measured. At age 61 months, in addition to routine weight and height measurements, four skinfolds were measured. At age 7 years, it was planned to measure body impedance, weight and height of all 14893 Children of the 90s. It was planned to finish data gathering up to the end of 2000; but by mid-2000 (when the present study was carried out), measurements for 4175 children (2120 boys and 2055 girls) were completed. Due to time constraints in this PhD, data from the first 4175 children followed-up to age 7 were therefore analysed and the results are presented in this Chapter. This sample size was much larger than in any previous study of obesity screening, especially given the homogenous nature of the sample compared to studies summarised in Table 5.1, and was considered adequate for the purposes of the present study. For the present study the representativeness of the sample used was not a major concern since the study aimed simply to test / develop obesity screening tools. The sampling and sample frame for ALSPAC is described in more detail in Chapter 2.

5.3.3. Procedures and definitions: criterion definition of excess fatness

As already mentioned, although a reference method or multi-component model to measure fatness would be ideal, the size of the sample in the present study made the use of such methods impossible. In such a situation, any simpler alternative applicable method used to estimate fatness has to be validated against the laboratory reference methods (WHO 1995; Slaughter et al 1988). A method previously shown to be able to estimate body fat percentage in prepubertal British children accurately (relative to a 2-component model with underwater weighing), was

bioelectrical impedance at 50KHz using the Houtkooper (Houtkooper et al 1992) formula (Reilly et al 1996). This formula is as follows:

Fat free mass or FFM (kg) = 0.61 (RI) + 0.25 (weight, kg) + 1.31

Where RI (resistance index) = height $(cm)^2$ / resistance (ohms)

From the 2120 boys and 2055 girls available for this study, the weight, height and body impedance of 2010 boys and 1938 girls were measured successfully (n=3948) as described in Chapter 2. For the present study 'true positives', i.e. obese children, were defined as those who were in the top 5% of the distribution of body fat percentage (this corresponded to >24.2% body fat for boys and >29.2% body fat for girls). This definition of excessive fatness (using 95th centile as cut-off) was arbitrary, but like other studies, the precise choice of body fatness cut-off did not influence our conclusions on sensitivity and specificity of the BMI cut-offs tested (Kuczmarski 1993; Warner et al 1997; Schaefer et al 1998). For clarity of presentation this Chapter presents only the analysis using the top 5% of body fatness as the definition of 'true positive'.

5.3.4. Anthropometric measurements

In each child height (to 0.1cm) was measured using a Leicester Height Measure and weight (to 0.1kg) in light indoor clothing using a SECA scale. From these measurements we calculated BMI and expressed this relative to UK reference data (Cole et al 1995) as a standard deviation score (SDS) using software provided by the Child Growth Foundation, London. The IOTF 2000 BMI cut-offs introduced to identify overweight and obese 7 year olds were compared to the top 5% of body fat to calculate the sensitivity and specificity of these cut-offs (Dietz & Bellizzi 1999; Cole et al 2000). Whole body bioelectrical impedance was measured with children supine after a 2-minute rest and minimum 2-hour fast, as previously described (Reilly et al 1996).

5.3.5. Statistical analyses

Chi-squared tests and 2-probability Z-tests were used to determine the equivalence of sensitivity and specificity between the sexes. The sensitivity and specificity values for different BMI centiles (their SDS equivalents are presented in Table 5.2) were used to clarify their effectiveness in correctly identifying obese children (Table 5.4).

Table 5.2. Percentiles and their SDS equivalents.

Percentile	Standard Deviation Score		Percentile	Standard Deviation Score
74	0.64		88	1.18
75	0.68		89	1.23
76	0.71		89.4	1.25
77	0.74		90	1.28
78	0.78		91	1.34
79	0.82		92	1.40
80	0.85		93	1.48
81	0.89		93.3	1.50
82	0.92		94	1.56
83	0.95		95	1.64
84	0.99		96	1.75
84.1	1.00		97	1.88
85	1.04		97.7	2.00
86	1.08		98	2.06
87	1.13		99	2.33

5.4. Results

5.4.1. Descriptive characteristics

Descriptive characteristics of the study subjects are given in Table 5.3.

Table 5.3. Descriptive characteristics of 7 year old children (n=4175).

Variable		Mean (SD)	Total (n)
Weight (kg)	Boys	25.5 (4.0)	2120
	Girls	25.7 (4.7)	2055
	Total	25.6 (4.4)	4175
Height (cm)	Boys	*125.8 (5.3)	2120
	Girls	125.0 (5.4)	2055
	Total	125.4 (5.4)	4175
BMI (kg/m^2)	Boys	*16.0 (1.8)	2120
	Girls	16.3 (2.2)	2055
	Total	16.2 (2.0)	4175
FFM (kg)¤	Boys	*21.6 (2.7)	2010
	Girls	20.7 (2.8)	1938
	Total	21.2 (2.8)	3948
% body fat¤	Boys	*14.7 (5.1)	2010
	Girls	18.6 (5.8)	1938
	Total	16.6 (5.8)	3948‡

* Significant difference between boys and girls, $p<0.001$

‡ From the total 2120 boys and 2055 girls, 2010 boys and 1938 girls had body impedance measurements.

¤ To calculate percentage body fat and fat free mass (kg), subject's body impedance was measured and the Houtkooper formula (Houtkooper et al 1992) was used.

Based on the results shown in Table 5.3, the 7 years old boys were significantly taller than girls ($p<0.001$). The 50th centile value for height for 7 years old boys and girls from the UK 1990 reference data (Cole et al 1995) were 124.9 cm and 124.3 cm respectively while the study children were significantly ($p<0.001$) taller (mean=125.8 cm for boys and 125.0 cm for girls; Table 5.3). There was no significant difference in weight between the two sexes (Table 5.3). The 50th centile value for weight from the UK 1990 reference data (Cole et al 1995) for this age group in boys and girls were 24.3 kg and 24.4 kg respectively while the study children were significantly ($p<0.001$) heavier (mean=25.5 kg and 25.7 kg for boys and girls; Table 5.3). Girls had a significantly higher BMI than boys ($p<0.001$). BMI 50th centile values for 7 year old boys and girls from the UK 1990 reference data (Cole et al 1995) are 15.6 kg/m^2 and 15.8 kg/m^2 respectively while the study children had significantly ($p<0.001$) higher BMI (mean=16.0 kg/m^2 for boys and 16.3 kg/m^2 for girls; Table 5.3). Boys had a significantly higher amount of fat free mass (FFM) than girls but girls had a higher percentage of body fat ($p<0.001$).

5.4.2. Sensitivity, specificity, positive and negative predictive values for BMI SDS cut-offs

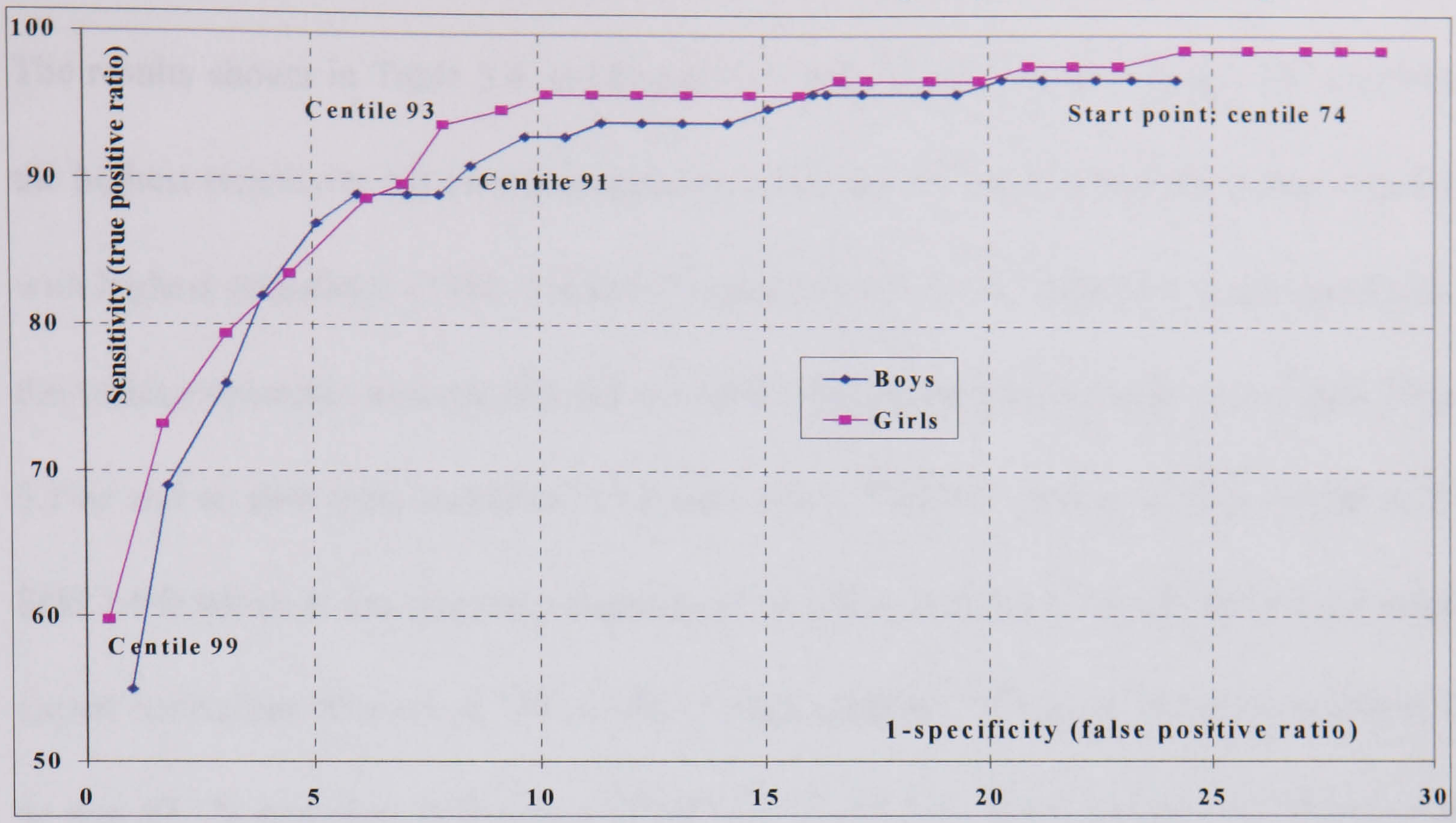
Sensitivity, specificity, positive predictive and negative predictive values for different BMI SDS cut-offs are shown in Table 5.4 and Figure 5.1-a (for each sex) and Figure 5.1-b (for total sample).

Table 5.4. Sensitivity, specificity and predictive values for different BMI centiles.

BMI centiles		True obese **Sensitivity** n	%	True non-obese **Specificity** n	%	Positive predictive value	Negative Predictive value
>74th	Boys	97/100	97.0	1448/1910	75.8	17.4	99.8
	Girls	96/ 97	99.0	1311/1841	71.2	15.3	99.9
	Total	193/197	98.0	2759/3751	73.6	16.3	99.9
>75th	Boys	97/100	97.0	1467/1910	76.8	18.0	99.9
	Girls	96/ 97	99.0	1328/1841	72.1	15.8	99.9
	Total	193/197	98.0	2795/3751	74.5	16.8	99.9
>76th	Boys	97/100	97.0	1483/1910	77.6	18.5	99.8
	Girls	96/ 97	99.0	1343/1841	72.9	16.2	99.9
	Total	193/197	98.0	2826/3751	75.3	17.3	99.9
>78th	Boys	97/100	97.0	1522/1910	79.7	20.0	99.8
	Girls	96/ 97	99.0	1391/1841	75.6	17.6	99.9
	Total	193/197	98.0	2913/3751	77.7	18.7	99.9
>80th	Boys	96/100	96.0	1554/1910	81.4	21.2	99.7
	Girls	95/ 97	97.9	1437/1841	78.1	19.0	99.9
	Total	191/197	97.0	2991/3751	79.7	20.1	99.8
>83rd	Boys	96/100	96.0	1602/1910	83.9	23.8	99.8
	Girls	94/ 97	96.9	1496/1841	81.3	21.4	99.8
	Total	190/197	96.4	3098/3751	82.6	22.5	99.8
>85th	Boys	94/100	94.0	1640/1910	85.8	25.8	99.6
	Girls	94/ 97	96.9	1534/1841	83.3	23.4	99.8
	Total	188/197	95.4	3174/3751	84.6	24.6	99.7
>88th	Boys	94/100	94.0	1693/1910	88.6	30.2	99.6
	Girls	93/ 97	95.9	1597/1841	86.7	27.6	99.7
	Total	187/197	94.9	3290/3751	87.7	28.9	99.7
>90th	Boys	93/100	93.0	1725/1910	90.3	33.4	99.6
	Girls	93/ 97	95.9	1634/1841	88.8	31.0	99.7
	Total	186/197	94.4	3359/3751	89.5	32.2	99.7
>92nd	Boys	89/100	89.0	1762/1910	92.2	37.6	99.4
	Girls	92/ 97	94.8	1671/1841	90.8	35.5	99.7
	Total	181/197	91.9	3433/3751	91.5	36.3	99.5
>94th	Boys	89/100	89.0	1795/1910	94.0	43.6	99.4
	Girls	87/ 97	89.7	1712/1841	93.0	40.3	99.4
	Total	176/197	89.3	3507/3751	93.5	41.9	99.4
>95th	Boys	87/100	87.0	1812/1910	94.9	47.0	99.3
	Girls	86/ 97	88.7	1727/1841	93.8	43.0	99.4
	Total	173/197	87.8	3539/3751	94.3	44.9	99.3
>96th	Boys	82/100	82.0	1835/1910	96.1	52.2	99.0
	Girls	81/ 97	83.5	1759/1841	95.5	49.7	99.1
	Total	163/197	82.7	3594/3751	95.8	50.9	99.1
>97th	Boys	76/100	76.0	1850/1910	96.9	55.9	98.7
	Girls	77/ 97	79.4	1784/1841	96.9	57.5	98.9
	Total	153/197	77.7	3634/3751	96.9	56.7	98.8
>98th	Boys	69/100	69.0	1876/1910	98.2	67.0	98.4
	Girls	71/ 97	73.2	1810/1841	98.3	69.6	99.1
	Total	140/197	71.1	3686/3751	98.3	68.3	98.7
>99th	Boys	55/100	55.0	1891/1910	99.0	74.3	97.7
	Girls	58/ 97	59.8	1831/1841	99.5	85.3	97.9
	Total	113/197	57.4	3722/3751	99.2	79.6	97.8

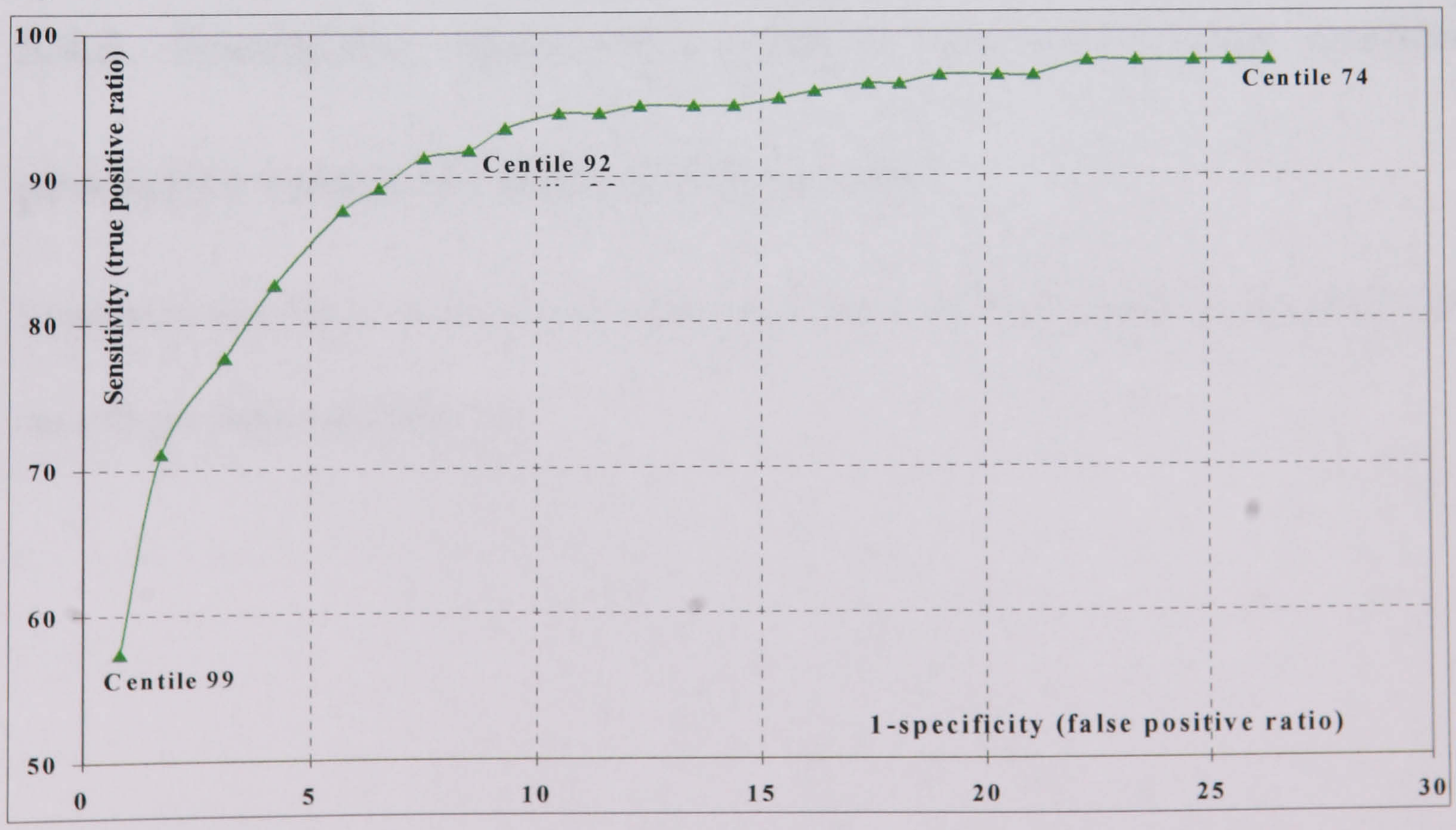
No significant differences in sensitivity, specificity, and predictive values were seen between boys and girls

Figure 5.1-a. ROC curves for different BMI centiles at age 7 years in boys and girls.



Although ROC curves drawn in Figure 5.1-a show small differences between boys and girls, these differences were not significant and therefore, a combined ROC curve was drawn as Figure 5.1-b for the total sample.

Figure 5.1-b. ROC curves for different BMI centiles at age 7 years in total population.



The results shown in Table 5.4 and Figures 5.1-a & 5.1-b indicate that the 74th centile had the highest sensitivity but lowest specificity, while the 99th centile had the lowest sensitivity with highest specificity (Table 5.4 and Figures 5.1-a & 5.1-b). Sensitivity and specificity of the various measures and cut-offs did not differ significantly between boys and girls (Figure 5.1-a) and so data were combined in Figure 5.1-b. The 95th centile of BMI (equal to BMI SDS 1.64) which is the recommended cut-off to define childhood obesity from the American expert committee (Barlow & Dietz 1998), could identify 173 out of 197 obese children and so was 87.7% sensitive. It identified 3539 out of 3751 non-obese subjects and therefore was 94.3% specific. The optimum cut-off based on BMI which combined highest sensitivity with high specificity was the 92nd centile (SDS 1.40; Figure 5.1-b). It should be noted that at the 92nd centile cut-off, sensitivity was 91.9%, and was higher than that provided by the 95th centile (87.8%; Table 5.4 and Figure 5.1b).

5.4.3. Sensitivity, specificity, positive predictive and negative predictive values for BMI IOTF cut-offs

Sensitivity, specificity, positive and negative predictive values for different BMI IOTF 2000 cut-offs are shown in Table 5.5.

Table 5.5. IOTF 2000 overweight and obesity definitions sensitivity, specificity, positive predictive values and negative predictive values.

BMI (IOTF) Equivalents		True obese **Sensitivity** n	%	True non-obese **Specificity** n	%	Positive predictive value	Negative predictive value
Overweight							
BMI=17.4	Boys	*90/100	90.0	**1756/1910	91.9	36.9	99.4
BMI=17.2	Girls	94/ 97	96.9	1543/1841	83.8	24.0	99.8
BMI=17.3	Total	184/197	93.4	3299/3751	87.9	28.9	99.6
Obese							
BMI=19.3	Boys	**46/100	46.0	**1901/1910	99.5	83.6	97.2
BMI=19.2	Girls	70/ 97	72.2	1813/1841	98.5	71.4	98.5
BMI=19.2	Total	116/197	58.9	3714/3751	99.0	75.8	98.1

Significant differences between boys and girls, * $p<0.05$; ** $p<0.001$

As shown in Table 5.5, sensitivity and specificity for the IOTF definitions of obesity differed significantly between boys and girls. The IOTF definition of obesity in 7 year old children was only able to identify 46.0% (46/100) of obese boys and 72.2% (70/97) of obese girls, but its specificity was higher (99.5% for boys and 98.5% for girls; Table 5.5). When the equivalent IOTF BMI cut-offs to define overweight were used, the sensitivity was higher (90.0% and 96.9% for boys and girls respectively; Table 5.5) but the specificity was lower (91.9% and 83.8% for boys and girls respectively; Table 5.5). In addition, when using IOTF values, large and statistically significant differences in screening ability between boys and girls were observed. Based on the results of Table 5.5, while sensitivity of the IOTF obesity definition cut-off was 72.2% in girls, it was only able to identify 46.0% of obese boys (Table 5.5).

5.5. Discussion

An expert committee in the USA recommended referral for in-depth medical assessment of all children with a BMI ≥95th percentile (Barlow & Dietz 1998), mostly because these children tend to remain obese and have greater risk of adverse health outcomes (Barlow & Dietz 1998). Although such formal guidelines do not exist in the UK, BMI charts (used for child growth monitoring and health screening; Child Growth Foundation, London) have been recommended to identify and refer children with a BMI ≥98th centile. Results of the present study indicate a low sensitivity (71.1%) but high specificity (98.3%) for this definition (BMI ≥98th centile). The 92nd centile for BMI was shown to be the optimum cut-off to define obesity in both boys and girls (Table 5.4; Figures 5.1-a & 5.1-b). The IOTF 2000 BMI cut-off for definition of obese children were less successful in identifying obese British children than the BMI centiles based on the UK 1990 reference data since sensitivity was only 46% and 72% in boys and girls respectively (Table 5.5) with no increase in specificity.

As can be seen in Table 5.1, many recent screening studies used body fat percent cut-offs which were similar to our study though their sample sizes were much smaller. Having >25% and >30% body fat in boys and girls is associated with cardiovascular risk factors (Hoerr et al 1992; Lohman 1993; Dwyer & Blizzard 1996), so our working definition happened to coincide with levels of fatness which have some clinical meaning.

5.5.1. Sensitivity and specificity of BMI as a screening tool: comparison with other studies

In a recent study the validity of BMI as an indicator of overweight or the risk of becoming overweight in 6 different ethnic groups of 9-19 year olds (n = 1570) was evaluated (Malina

& Katzmarzyk 1999). Comparing body fat percentage with BMI >85th and >95th centiles showed high specificity for BMI (96.3-100% to identify obesity) and low sensitivity (14.3-60%; Malina & Katzmarzyk 1999). Other screening studies to identify the sensitivity and specificity of different BMI centiles as an obesity definition tool in children have confirmed these results (Table 5.1). The few studies which concluded that BMI had low sensitivity and high specificity, all had either a small sample size or a wide age range (Table 5.1). In general, this means when BMI is used many subjects who are overweight or at risk of overweight are not correctly identified, but almost all of those who were not overweight or at risk of being overweight are classified correctly (Malina & Katzmarzyk 1999).

The present study with a large sample size (2120 and 2055 boys and girls) and homogenous age range (7 year old children) shows that BMI had high sensitivity (92%) as well as high specificity (92%; Table 5.4 and Figures 5.1-a & 5.1-b) when appropriate cut-off (e.g. 92nd centile) was chosen. Furthermore, no significant differences between the sexes were seen and so the same cut-off (e.g. 92nd centile for BMI) could be applied to both boys and girls (Table 5.4 and Figures 5.1-a & 5.1-b). The IOTF 2000 cut-off to define obesity in 7 year olds (BMI >19.2) was characterised by low sensitivity (Table 5.5).

5.5.2. Use of BMI as a screening tool in clinical practice

Any definition suitable for clinical use must have high specificity in order to avoid unnecessary treatment of non-obese children, and to prevent wrongly labelling individuals as obese and causing them consequent harm (Himes & Dietz 1994; Barlow & Dietz 1998; Reilly et al 1999b). It could be argued that the BMI cut-off recommended by the US expert committee (95th centile for identifying obesity) would be an acceptable tool for clinical obesity screening since it has high specificity though has lower sensitivity. Historically, achieving high specificity has been regarded as acceptable since very few 'false positives'

would be identified (Himes & Dietz 1994; Barlow & Dietz 1998; Reilly et al 1999b). In addition, using BMI is simple and can provide consistency with adulthood practice. The findings of the present study confirm that the definition of paediatric obesity based on BMI $\geq 95^{th}$ percentile is associated with relatively high specificity, and so will only identify a few non-obese children as obese.

Our study results, however, suggest that using BMI $\geq 92^{nd}$ centile as a screening tool in British children might be beneficial since it has high sensitivity as well as high specificity (Table 5.4 and Figures 5.1-a & 5.1-b). More detailed comparisons with other studies are difficult to make because the other studies used different definitions of obesity, or a wider age range or small sample size. However the present study suggests that use of BMI cut-offs from 1990 UK reference data to screen for obesity can provide both fairly high sensitivity and high specificity. It should be remembered that BMI is only a screening tool (Barlow & Dietz 1998; Malina & Katzmarzyk 1999).

5.5.3. Use of BMI in epidemiology

It has been argued that one major disadvantage of using BMI to define obesity in epidemiology is its low sensitivity (Table 5.1). Results obtained by other studies suggested that the low sensitivity of BMI is an inherent limitation for both children (Warner et al 1997; Malina & Kuczmaski 1999; Reilly et al 1999b) and adults (Hortobagyi et al 1994) and is not simply a function of the precise cut-off chosen to define obesity. However, our study results indicate reasonable sensitivities across the range of BMI definitions (percentiles 74 to 99 relative to UK 1990 reference data) when tested in the ROC analysis (Table 5.4 and Figures 5.1-a & 5.1-b). Using the IOTF cut-offs (Cole et al 2000) to define obesity in British children showed poor sensitivity, especially in boys, and may therefore not be the best method to use. One consequence of this low sensitivity would be that the IOTF cut-offs

would massively under-estimate obesity prevalence if used in epidemiology. Although our sample consisted entirely of seven year old children, there is no evidence that sensitivity and specificity of screening tools differs markedly during childhood (Warner et al 1997; Malina & Kuczmaski 1999; Reilly et al 1999b), and so our findings in relation to the strengths and limitations of BMI may apply to older children as well.

Use of trends in BMI, or weight adjusted for height in other ways, has made it difficult to discern trends in obesity (WHO 1995; Hughes et al 1997). BMI is a function of weight and height only, and when it increases in populations over time, population changes in lean body mass during childhood cannot be readily distinguished from changes in body fatness. Changes in skinfold thickness or body fat percentage are less equivocal and might provide greater confidence for epidemiological applications (WHO 1995; Hughes et al 1997). The low sensitivity of BMI has disadvantages for other applications. For example, in population genetic analysis of obesity, BMI can only be a crude index of the phenotype. The IOTF cut-offs (equivalent to adult BMI >30) appear to have low sensitivity in both sexes. This may cause a serious underestimation of childhood obesity prevalence, which is more serious in boys. Use of the IOTF cut-off to define overweight children (equivalent to adulthood BMI >25; Table 5.5) was more sensitive and highly specific.

5.6. Conclusions

The current study provided information about using BMI percentiles relative to the UK 1990 reference data (Cole et al 1995) and recent IOTF BMI cut-offs (Cole et al 2000) to identify obese children. In theory, the IOTF cut-offs are supposed to identify obese children worldwide and therefore, are intended to be used mainly in epidemiology. Using the UK 1990 reference data for BMI at a cut-off of the 95th centile showed reasonable sensitivity while the IOTF cut-off (equivalent to adult BMI >30) had very low sensitivity and the sensitivities of the IOTF cut-offs were significantly different between the sexes (46% and 72% for boys and girls respectively; Table 5.5). This would cause a serious underestimation of obesity prevalence in British children which would be more marked in boys. Using the IOTF cut-off to define overweight children (equivalent to adulthood BMI >25) showed higher sensitivity (93.4%) though with lower specificity (87.9%).

Using BMI ≥95th percentile to define obesity, as was recommended by the US expert committee (Barlow & Dietz 1998), will identify most (but not all) obese children but will identify few non-obese children as obese. BMI is useful for the first step in obesity assessment and will also be very helpful when there is no access to other more complicated methods. The present study suggests that use of BMI ≥92nd centile could be considered as an alternative to the 95th centile since it showed increased sensitivity with little loss in specificity.

6. Chapter 6: Adiposity rebound in British children

6.1. Introduction

Many investigations have shown that childhood BMI is related to adult BMI (Dietz 1994; Whitaker et al 1998a; Williams et al 1999). BMI increases from the time of birth up to age 1 year and thereafter decreases. The age when BMI starts to increase again, about age 4-7 years, after its nadir, in a French study in 1984, was named as the '*Adiposity Rebound* (AR)' (Rolland-Cachera et al 1984). The age of AR is negatively correlated with body mass index in adulthood: the earlier the rebound, the greater the later body mass index (Rolland-Cachera et al 1987; Siervogel et al 1991; DeSpiegelaere et al 1998a; Whitaker et al 1998b). Adiposity rebound is not related just to BMI but also to percentage of body fat based on skinfold measurements where the same pattern of change occurs in early childhood (Rolland-Cachera 1999). It is currently believed that changes in body fatness in childhood are related to changes in fat cell size or fat cell numbers (DeSpiegelaere et al 1998a). During the first year of life the size of fat cells increases, so that adiposity rapidly increases. Thereafter, adipose tissue growth remains stable for a few years but height continues to increase and thus the relative fatness decreases. Later, due to a rapid increase in fat cell numbers a rapid increase in body fat occurs again. This is when the adiposity rebound occurs (DeSpiegelaere et al 1998a).

Many observations suggest that there are three periods that are critical for the development of adulthood obesity: gestation & early infancy; the period of adiposity rebound; adolescence (Dietz 1994; Dietz 1997b). The mechanisms by which events during these periods can increase the risk of later obesity are unclear at present. However, the existence

of such critical periods should encourage research and lead to a focus on obesity prevention efforts at such important stages (Dietz 1994; Dietz 1997b).

From 1984 till 1999 less than 15 papers on AR were published. Nowadays, this issue is of increasing interest because of the implications of AR for adult obesity. The investigators who first coined the term AR were the only ones who studied relationships between age at AR and childhood nutritional variables (Rolland-Cachera et al 1995). They reported a relationship between protein intake in early life (age 2 years) and age of AR and suggested the hypothesis that high protein intake caused early AR (Rolland-Cachera et al 1995). Because of the small sample size (only 112 boys and girls), limited dietary analysis, and some other issues (like the specific ethnic group of their samples: French children) in this study, it was important to do a similar investigation in another population with a larger sample size to test the hypothesis that high protein intake is associated with early AR. It was also important to describe the timing of AR in a modern sample of children. The literature at present contains evidence on the timing of AR in children from the past (between the 1950s to the 1980s; e.g. Rolland-Cachera et al 1995) but there is evidence of a trend to earlier AR in recent years (Cole et al 1995; Rolland-Cachera 1999). In a recent French study it was suggested that 'active' school children had a higher proportion of fat-free mass, a lower proportion of fat-mass (measured in the arm) and later age of AR than less active children (Deheeger et al 1997). No other factors with possible association to early AR have been investigated (Whitaker et al 1998b), therefore further work is necessary if the process of AR is to be better understood. Determining the factors that influence the age of AR can identify factors influencing energy balance, and help to suggest steps needed for obesity prevention (Dietz 2000a).

6.2. Aims

1. To describe the timing of AR in British children and define the percentage of children with early AR (study 1).

2. To test the hypothesis that higher protein intake in early childhood is associated with age of AR, in a British population with a reasonably large sample size (study 2).

3. To identify the factors which might be associated with timing of AR (study 2).

6.3. Subjects and methods

6.3.1. Subjects

The Children in Focus data of the ALSPAC prospective cohort study of the determinants of health during childhood in the Bristol-Avon area of the UK (described in detail in Chapter 2) represented a sub-sample randomly selected from the main ALSPAC study sample (n=14893, Children of the 90s) for more detailed investigations. The total number of this sub-sample was 1409 children at age 4 months and the weight and/or the height for 1308 of them at age 8 months and for 972 of them at age 61 months was available. Of these, 889 of them had weight and height measurements for BMI calculation. The data for these 889 subjects were analysed and the results are presented in this Chapter. No significant difference in mean BMI values between the study children (n=889; 486 boys, 403 girls) and the Children in Focus sample (n=1409) was observed. The Children in Focus sample were born in 1992 and are broadly representative of the entire ALSPAC cohort (Chapter 2; ALSPAC homepage; Golding et al 2001) though the percentage of families in high social class was slightly higher in Children in Focus than in Children of the 90s and NDNS 1995 (Tables 2.2 & 2.3 of Chapter 2). Measurements on this sub-sample were made at frequent intervals: 4, 8, 12, 18, 25, 31, 37, 43, 49 and 61 months, all data available at the time of the present study in 1999.

6.3.2. Childhood measurements: height, weight, BMI, and adiposity rebound

At each measurement occasion up to 25 months, length was measured to 0.1cm using the Harpenden Neonatometer and Holtain Kiddimetre instruments. From age 25 months, height was measured to 0.1cm using the Leicester Height Measure. Weight was measured wearing light clothing to 0.1 kg at each measurement occasion using SECA scales. BMI was then calculated for each measurement period. Timing of AR was assessed by visual inspection, using the method of Rolland-Cachera et al (described in Chapter 2), but with the modification that any increase in BMI after the nadir had to equal or exceed 0.1 kg/m^2 before AR was confirmed. In addition, timing of AR was only assessed for those children with BMI values for most of the 10 measurement occasions.

The definition used to identify the age of adiposity rebound was slightly more conservative than the definitions in the literature upon which it was based (Rolland-Cachera et al 1987). To confirm if our methodology was similar to that of Rolland-Cachera, we asked her to define AR in 12 subjects randomly selected from our entire sample; her definition was identical to ours and she was fully satisfied with our methodology. BMI was also expressed as a standard deviation score (SDS) relative to contemporary UK reference data (Cole et al 1995; Child Growth Foundation Software, London, UK) as described in Chapter 2.

6.3.3. Dietary and nutrient intake

Dietary intake data were obtained in Children in Focus by 3-day prospectively collected household measures (unweighed) records at ages 8, 18, and 43 months as described in chapter 2. Dietary data at 18 months were nearest in timing to the earlier French study in which intakes were assessed at 24 months (Rolland-Cachera et al 1987), but 8 and 43 months dietary data were analysed as well. From these records we calculated absolute intake

of energy and macronutrients, energy intake per kg body mass, and macronutrient intake as percentage of dietary energy intake.

6.3.4. Parental information

Parental heights (to 1cm) and weights (to 0.5kg, pre-gravid weight of mothers) were obtained by self-report (ALSPAC homepage). Socioeconomic status (social class category) was determined by standard methods from the occupation of the head of the household (Office of Population Censuses & Surveys, OPCS 1990). Educational attainment of both parents was obtained by self-report and summarised as: CSE or less; vocational qualifications; O-level (age 16); A level (age 18); and Degree level.

6.3.5. Statistical analysis

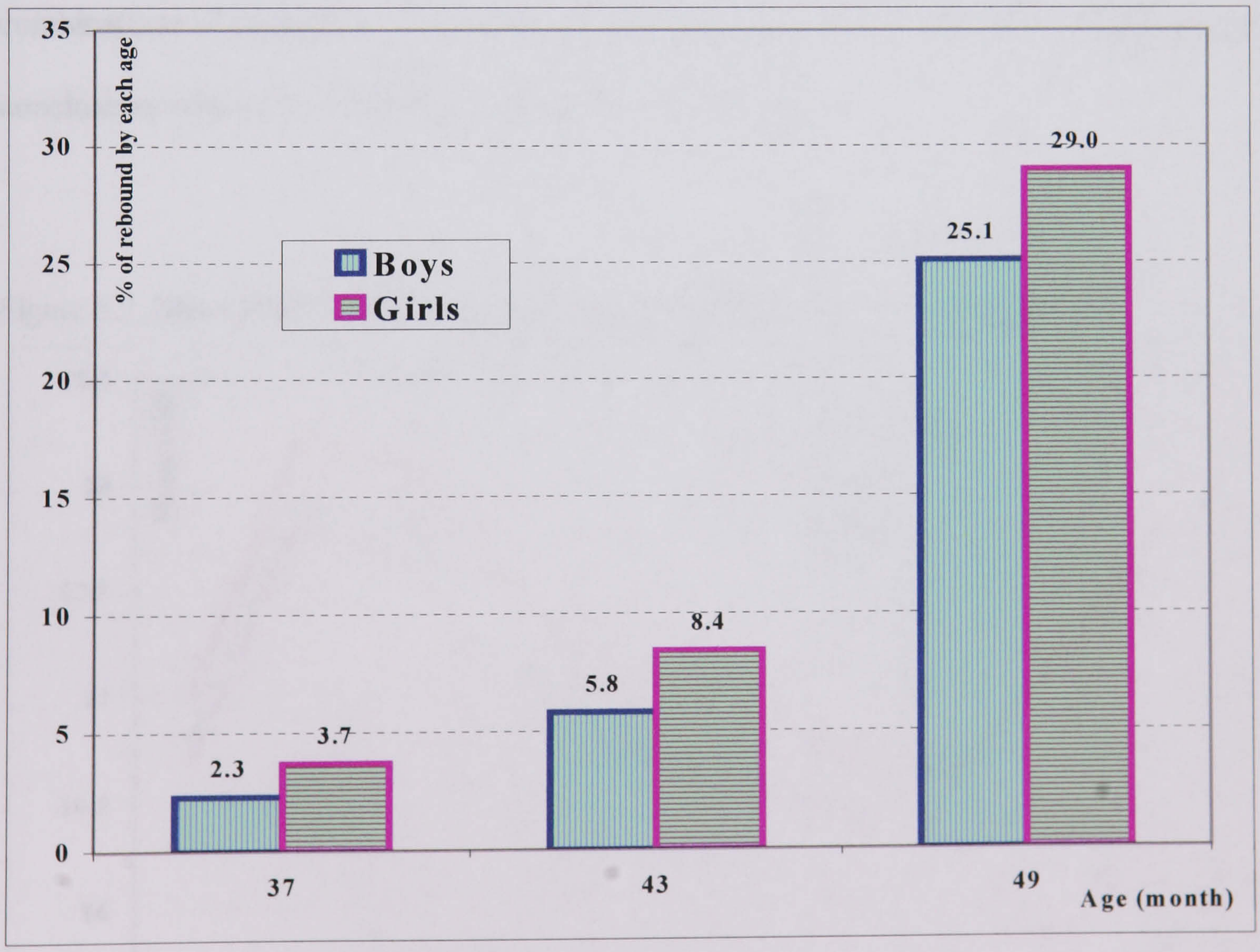
The aim of the analysis for study 1 was to have simple descriptive statistics and plots of the timing of AR in the sample. The aims of the analysis for study 2 were to test for differences between children with earlier AR and the rest, using timing of the AR as a categorical variable. 'Very Early' AR was defined as those children who had their BMI nadir at any age younger than or at age 43 months; 'early' AR had their rebound from age 49 months up to 61 months; and the 'later' AR group were those who had their AR after age 61 months. Differences between categories distinguished on the basis of continuous variables were tested for significance using one way ANOVA with follow-up multiple comparisons incorporating Bonferroni corrections as appropriate. Differences for categorical variables were tested for significance using Chi-squared tests. The following variables were tested for their influence on the timing of AR: absolute and relative macronutrient intakes at ages 8, 18, and 43 months; paternal and maternal BMI; parental educational attainment and social class; children's BMI and BMI SDS.

6.4. Results

6.4.1. Study 1: timing of AR in British children

There were no significant differences between the timing of AR between boys and girls but the results are presented for boys and girls separately to show the magnitude of the small differences between them. From age 37 months about 3% and at age 43 months, about 7% of children in the ALSPAC cohort had their AR. By age 49 months about 27% of children (more than one quarter) experienced AR (Figure 6.1). The next measurement in the ALSPAC study after age 61 months was age 90 months. Therefore, using the data available at the time of writing (October 2000), judgement must be that the typical age of AR in British children must be sometime between 5-7 years.

Figure 6.1. Cumulative percentage of adiposity rebound at different ages in boys and girls*.



* Percentage of boys who had their rebound (at ages 37, 43, and 49 months) was always lower, but these differences were not statistically significant.

6.4.2. Study 2

6.4.2-a. Timing of AR in relation to other childhood variables

Table 6.1 shows the means and standard deviations of absolute BMI values at different ages. Significant difference between 4-31 months early rebound boys and girls was encountered (table 6.1). Figure 6.2 compares the mean absolute BMI values at different available ages for the 3 groups. As can be seen, the absolute BMI values increased from age 4 to 12 months and thereafter decreased in the total sample of children. The same pattern could be seen in all different rebound categories up to the age of rebound. The 'very early rebound' groups started their BMI rebound from age 43 months whilst the 'early rebound' groups had their rebound from age 49 months and others had not started their rebound by age 61 months. While these definitions were somewhat arbitrary, to confirm that our results were not a function of the precise categories chosen we repeated the analyses with different combinations of categories representing different timing of AR: we found no difference in our conclusions when the definitions / categories were altered.

Figure 6.2. Mean BMI values in very early, early, and later adiposity rebound groups.

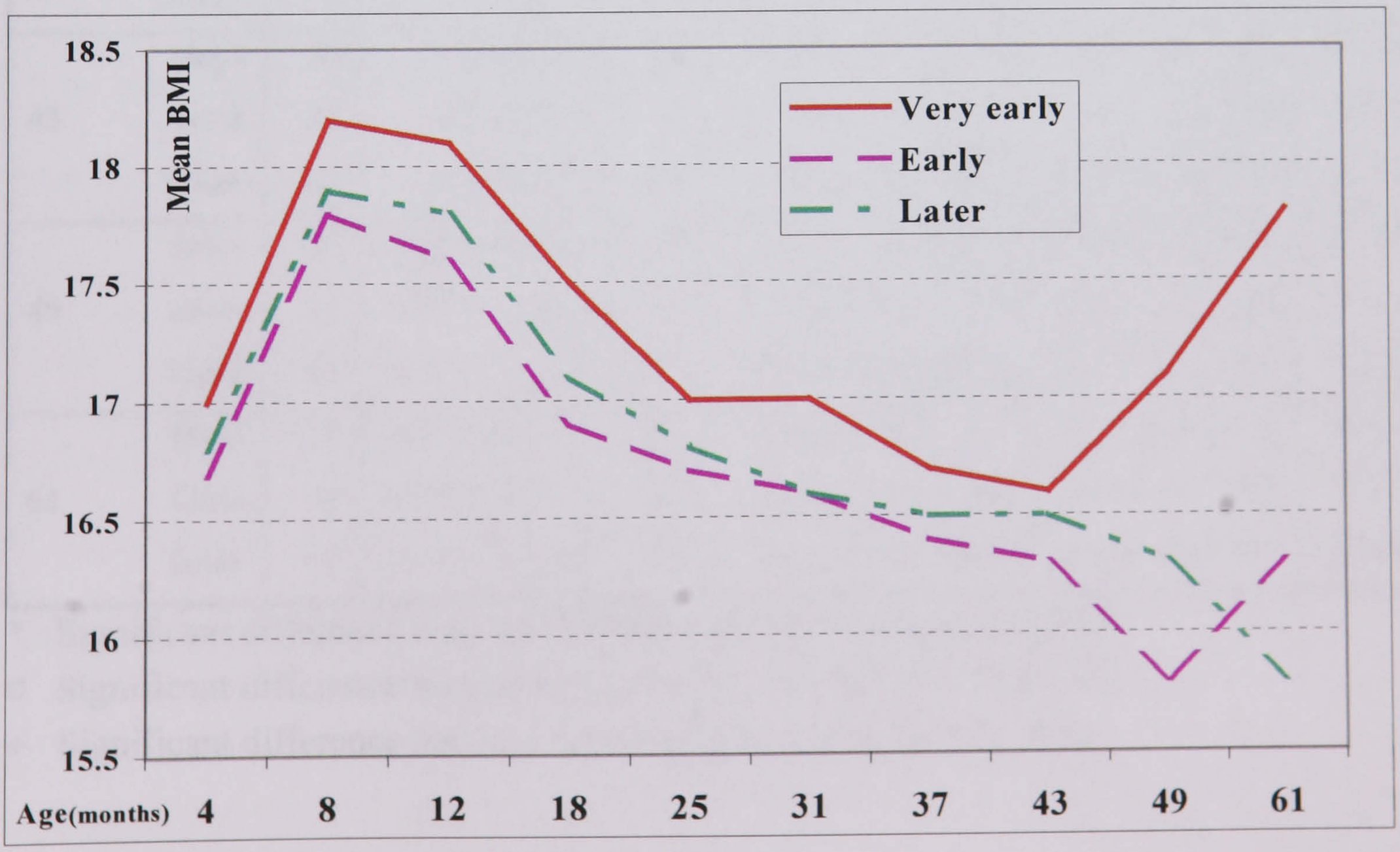


Table 6.1. Absolute BMI values in very early, early, and later AR groups.

BMI / Age (months)		Very early rebound n	Very early rebound Mean(SD)	Early rebound n	Early rebound Mean(SD)	Later rebound n	Later rebound Mean(SD)	Total n	Total Mean(SD)
4	Boys	21	16.9 (1.4)	67	17.1 (1.5)	253	16.9 (1.3)	341	16.9 (1.4)
	Girls	26	¤17.0 (1.2)	61	÷÷16.3 (1.3)	211	16.6 (1.3)	298	16.6 (1.3)
	Total	47	17.0 (1.3)	128	16.7 (1.4)	464	16.8 (1.3)	639	16.8 (1.3)
8	Boys	27	18.2 (1.3)	92	18.0 (1.6)	357	17.9 (1.4)	476	18.0 (1.4)
	Girls	32	¤18.2 (1.9)	82	÷17.5 (1.5)	274	17.8 (1.4)	388	17.7 (1.5)
	Total	59	18.2 (1.6)	174	17.8 (1.5)	631	17.9 (1.4)	864	17.9 (1.5)
12	Boys	27	18.0 (1.3)	89	17.9 (1.3)	352	17.9 (1.3)	468	17.9 (1.3)
	Girls	32	¤18.1 (1.5)	80	÷17.4 (1.3)	273	17.7 (1.3)	385	17.7 (1.4)
	Total	59	¤18.1 (1.4)	169	17.6 (1.3)	625	17.8 (1.3)	853	17.8 (1.4)
18	Boys	25	17.3 (1.1)	88	17.3 (1.2)	338	17.2 (1.3)	451	17.2 (1.3)
	Girls	33	¤¤*17.6 (2.0)	74	÷÷*16.6(1.4)	267	17.0 (1.2)	374	17.0 (1.4)
	Total	58	¤¤*17.5 (1.7)	162	16.9 (1.3)	605	17.1 (1.3)	825	17.1 (1.3)
25	Boys	24	16.8 (1.5)	84	17.1 (1.5)	321	16.8 (1.3)	429	16.9 (1.4)
	Girls	28	¤17.1 (1.5)	72	÷÷16.3 (1.2)	247	16.8 (1.3)	347	16.7 (1.3)
	Total	52	17.0 (1.5)	156	16.7 (1.3)	568	16.8 (1.3)	776	16.8 (1.4)
31	Boys	23	16.7 (1.4)	86	16.9 (1.5)	333	16.6 (1.3)	442	16.7 (1.3)
	Girls	32	¤¤*17.2 (1.6)	75	÷÷16.2 (1.4)	263	16.6 (1.3)	370	16.6 (1.4)
	Total	55	*17.0 (1.5)	161	16.6 (1.5)	596	16.6 (1.3)	812	16.6 (1.4)
37	Boys	25	16.4 (1.2)	90	16.6 (1.3)	339	16.5 (1.2)	454	16.5 (1.2)
	Girls	31	¤16.9 (1.6)	78	16.2 (1.3)	269	16.6 (1.5)	378	16.5 (1.5)
	Total	56	16.7 (1.4)	168	16.4 (1.3)	608	16.5 (1.4)	832	16.5 (1.3)
43	Boys	28	16.3 (1.3)	87	16.5 (1.2)	340	16.4 (1.2)	455	16.5 (1.2)
	Girls	34	¤16.9 (1.9)	79	16.2 (1.3)	271	16.5 (1.4)	384	16.5 (1.4)
	Total	62	16.6 (1.6)	166	16.3 (1.2)	611	16.5 (1.4)	839	16.5 (1.3)
49	Boys	28	¤¤*16.8 (1.4)	94	16.0 (1.2)	364	16.2 (1.2)	486	16.2 (1.2)
	Girls	34	¤¤**17.3 (2.0)	83	**15.6 (1.2)	286	16.4 (1.4)	403	16.3 (1.5)
	Total	62	¤¤**17.1 (1.7)	177	**15.8 (1.2)	650	16.3 (1.3)	889	16.2 (1.4)
61	Boys	28	¤¤**17.6 (1.8)	94	**16.4 (1.4)	364	15.7 (1.2)	486	16.0 (1.4)
	Girls	34	¤¤**18.0 (2.1)	83	16.2 (1.6)	286	15.9 (1.4)	403	16.1 (1.6)
	Total	62	¤¤**17.8 (2.0)	177	**16.3 (1.5)	650	15.8 (1.3)	889	16.1 (1.5)

* Significant difference with later rebound group, *p<0.05, **p<0.01

¤ Significant difference with early rebound group, ¤p<0.05, ¤¤p<0.01

÷ Significant difference between boys and girls, ÷p<0.05, ÷÷p<0.01

Figure 6.3. BMI SDS in very early, early, and later AR groups.

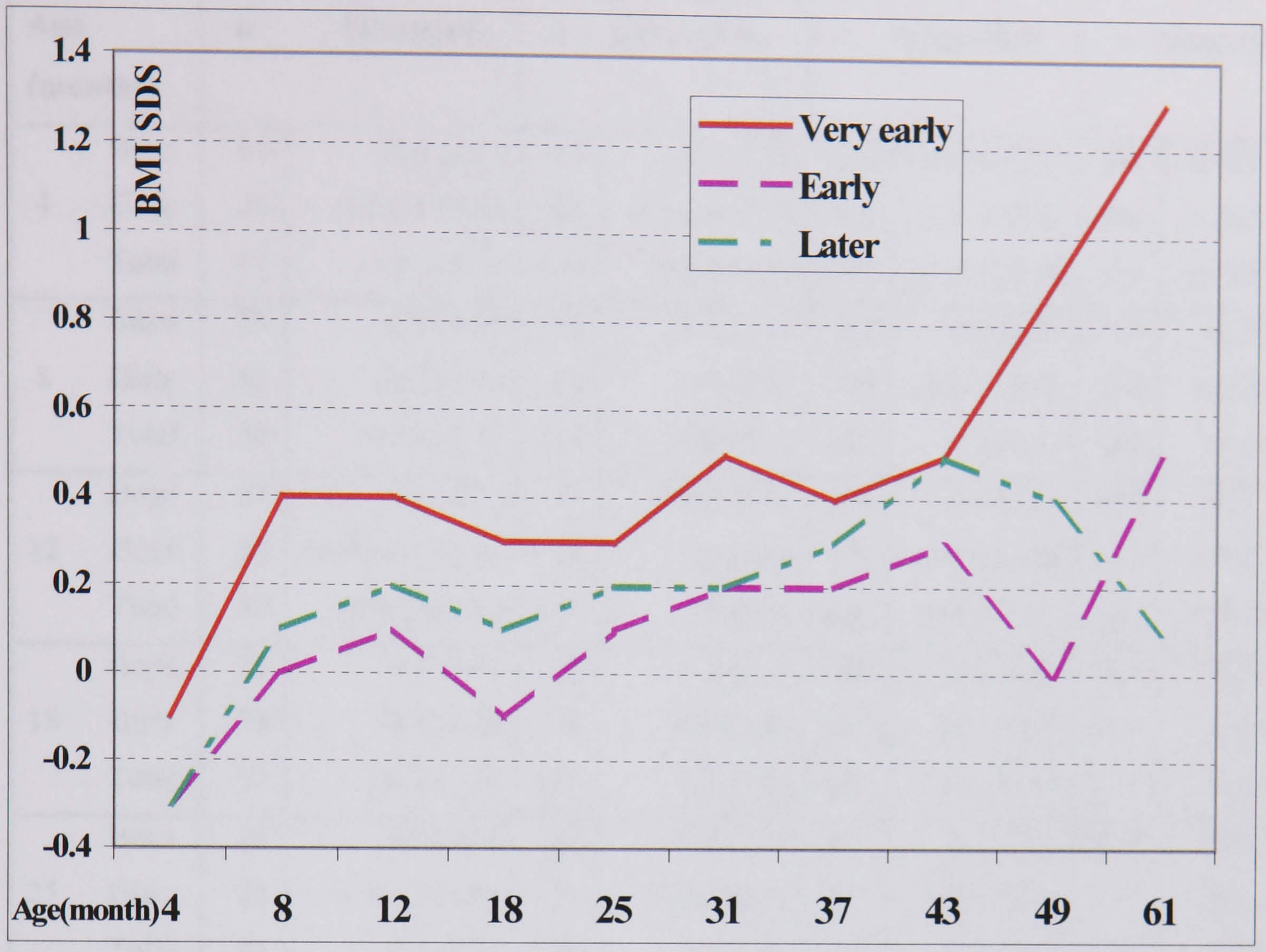


Table 6.2 shows the means and standard deviations of BMI Standard Deviation Scores (SDS) in all age groups. Figure 6.3 compares the mean BMI SDS values at different available ages. As can be seen, very early rebound groups started to increase in their BMI SDS from age 43 months whilst the early rebound groups showed an increase in BMI SDS from age 49 months. Later rebound groups had a moderate increase in their BMI SDS up to age 43 months but BMI SDS decreased thereafter. There was some evidence that girls with very early rebound had higher BMI SDS before the rebound than the rest (Table 6.2). *After* the rebound had occurred BMI SDS were significantly higher in those children who had very early rebound (one way Anova, $p<0.001$).

Table 6.2. BMI Standard Deviation Scores at each age.

Age (months)	BMI	Very early rebound n	Very early rebound Mean(SD)	Early rebound n	Early rebound Mean(SD)	Later rebound n	Later rebound Mean(SD)	Total n	Total Mean(SD)
4	Boys	21	-0.4 (1.1)	67	-0.2 (1.1)	253	††-0.4 (1.0)	341	††-0.3 (1.0)
	Girls	26	÷*¤¤ 0.2 (0.8)	61	††*-0.4 (1.1)	211	-0.1 (1.0)	298	††-0.2 (1.0)
	Total	47	-0.1 (1.0)	128	††-0.3 (1.1)	464	††-0.3 (1.0)	639	††-0.2 (1.0)
8	Boys	27	0.2 (0.9)	92	0.1 (1.1)	357	0.0 (1.0)	476	†0.1 (1.0)
	Girls	32	†0.5 (1.2)	82	0.0 (1.1)	274	††0.2 (1.0)	388	††0.2 (1.0)
	Total	59	††0.4 (1.1)	174	0.0 (1.1)	631	0.1 (1.0)	864	†0.1 (1.0)
12	Boys	27	0.2 (0.9)	89	††0.2 (0.9)	352	0.1 (1.0)	468	† 0.1 (1.0)
	Girls	32	††÷*¤¤0.6 (1.0)	80	0.0 (1.0)	273	††0.2 (1.0)	385	†† 0.2 (1.0)
	Total	59	††*¤0.4 (1.0)	169	0.1 (0.9)	625	††0.2 (1.0)	853	†† 0.2 (1.0)
18	Boys	25	0.1 (0.8)	88	0.1 (0.9)	338	0.0 (1.0)	451	0.0 (0.9)
	Girls	33	†0.5 (1.2)	74	-0.2 (1.0)	267	0.1 (0.9)	374	†0.1 (1.0)
	Total	58	†0.3 (1.0)	162	-0.1 (1.0)	605	0.1 (0.9)	825	†0.1 (1.0)
25	Boys	24	0.0 (1.2)	84	0.2 (1.0)	321	0.1 (1.0)	429	†0.1 (1.0)
	Girls	28	†÷¤¤0.5 (1.0)	72	**-0.1 (0.9)	247	††0.3 (0.9)	347	††0.2 (0.9)
	Total	52	0.3 (1.1)	156	0.1 (1.0)	568	††0.2 (0.9)	776	†0.1 (1.0)
31	Boys	23	0.2 (1.0)	86	††0.3 (1.0)	333	0.1 (1.0)	442	††0.2 (1.0)
	Girls	32	††0.7 (1.0)	75	0.1 (1.0)	263	††0.4 (0.8)	370	††0.3 (0.9)
	Total	55	††0.5 (1.0)	161	†0.2 (1.0)	596	††0.2 (0.9)	812	††0.2 (0.9)
37	Boys	25	0.2 (0.9)	90	††0.3 (0.9)	339	††0.2 (0.9)	454	††0.2 (0.9)
	Girls	31	††÷*¤¤0.7 (1.0)	78	**0.1 (0.9)	269	††0.4 (0.9)	378	††0.4 (0.9)
	Total	56	††0.4 (1.0)	168	††0.2 (0.9)	608	††0.3 (0.9)	832	††0.3 (0.9)
43	Boys	28	0.2 (1.0)	87	††0.4 (0.8)	340	††0.4 (0.9)	455	††0.4 (0.9)
	Girls	34	††0.7 (1.1)	79	0.2 (0.9)	271	††0.5 (0.9)	384	††0.5 (0.9)
	Total	62	††0.5 (1.0)	166	††0.3 (0.9)	611	††0.5 (0.9)	839	††0.4 (0.9)
49	Boys	28	††*¤¤0.7 (1.0)	94	* 0.1 (0.9)	364	††0.3 (0.9)	486	††0.3 (0.9)
	Girls	34	††**¤¤1.0 (1.0)	83	**-0.1 (0.9)	286	††0.4 (0.9)	403	††0.4 (0.9)
	Total	62	††**¤¤0.9 (1.0)	177	** 0.0 (0.9)	650	††0.4 (0.9)	889	††0.3 (0.9)
61	Boys	28	††**¤¤1.3 (1.0)	94	††**0.6 (0.9)	364	0.1 (1.0)	486	††0.2 (1.0)
	Girls	34	††**¤¤1.4 (1.0)	83	††**0.4 (0.9)	286	††0.2 (0.8)	403	††0.3 (0.9)
	Total	62	††**¤¤1.3 (1.0)	177	††**0.5 (0.9)	650	0.1 (0.9)	889	††0.3 (1.0)

* Significant difference with later rebound group, *p<0.05, **p<0.01

¤ Significant difference with early rebound group, ¤p<0.05, ¤¤p<0.01

÷ Significant difference between boys and girls, p<0.05

† Significant difference between mean SDS and the expected (zero), †p<0.05, ††p<0.01

6.4.2-b. Timing of AR in relation to parental variables

Table 6.3 provides means and standard deviations of paternal / maternal BMI and shows their relationship to the timing of AR. Parental BMI was the only variable which had any relationship with the timing of AR. The 'very early' AR group had the highest mean parental BMI values compared with the other groups. Paternal BMI was available for 670 out of 889 (75.4%) of study children and maternal BMI was available for 827 (93.0%).

Table 6.3. Parental factors and their relation to timing of adiposity rebound.

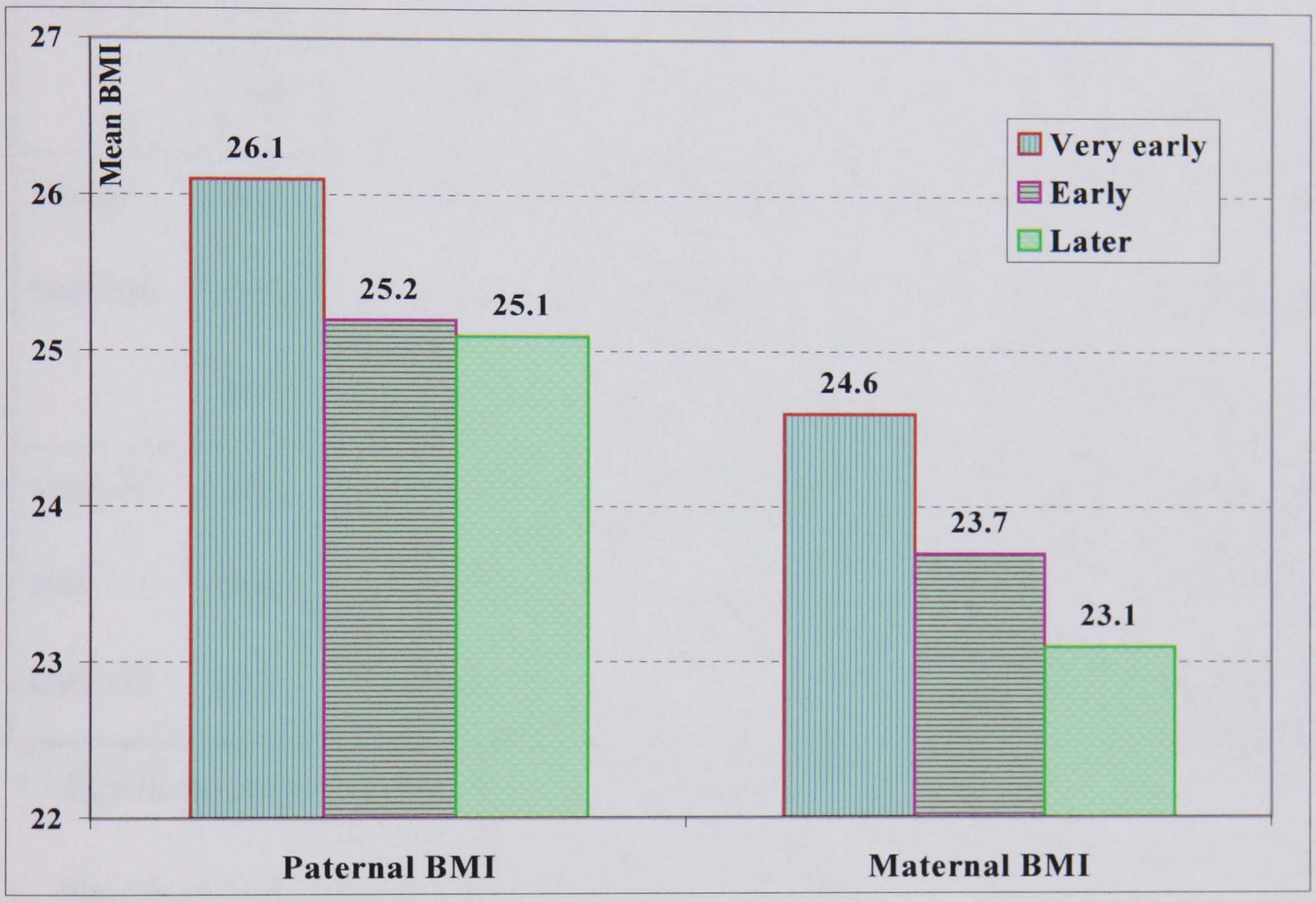
		Very early rebound		Early rebound		Later rebound		Total	
Parental variables		**n**	**Mean(SD)**	**n**	**Mean(SD)**	**n**	**Mean(SD)**	**n**	**Mean(SD)**
	Boys	23	25.8 (3.4)	77	25.4 (3.6)	260	25.2 (3.1)	360	25.2 (3.2)
Paternal	Girls	27	*¤26.3 (4.2)	65	24.9 (3.1)	218	25.1 (3.3)	310	25.2 (3.3)
BMI	Total	50	*26.1 (3.8)	142	25.2 (3.4)	478	25.1 (3.2)	670	25.2 (3.3)
	Boys	26	**¤25.3 (4.4)	87	23.8 (4.0)	338	23.0 (3.5)	451	23.2 (3.7)
Maternal	Girls	31	24.0 (3.7)	82	23.6 (5.0)	263	23.3 (4.1)	376	23.4 (4.3)
BMI	Total	57	**24.6 (4.1)	169	23.7 (4.5)	601	23.1 (3.8)	827	23.3 (4.0)

* Significant difference with later rebound group, *p<0.05, **p<0.01

¤ Significant difference with early rebound group, ¤p<0.05, ¤¤p<0.01

Figure 6.4 presents the parental BMI values in very early, early, and later adiposity rebound groups. As the results indicate, the mean BMI values for parents of children who had very early rebound were significantly higher than mean of absolute BMI values in other rebound groups.

Figure 6.4. Mean paternal and maternal BMI values in very early, early, and later adiposity rebounders.



*Significant differences between parental BMI values in very early and other rebound groups.

Table 6.4 shows the numbers and percentage of children in each AR group with an obese parent (BMI >30 kg/m^2). The very early AR group had the highest percentage of parents with BMI >30 compared to the other groups. For children in the very early AR group, 18.0% had an obese father and 12.3% had an obese mother. This compared with 8.8% and 7.0% for the cohort as a whole and these differences were statistically significant. Children in the very early AR group were about twice as likely to have an obese parent than children in the entire cohort.

Table 6.4. Parental obesity and its relation to adiposity rebound.

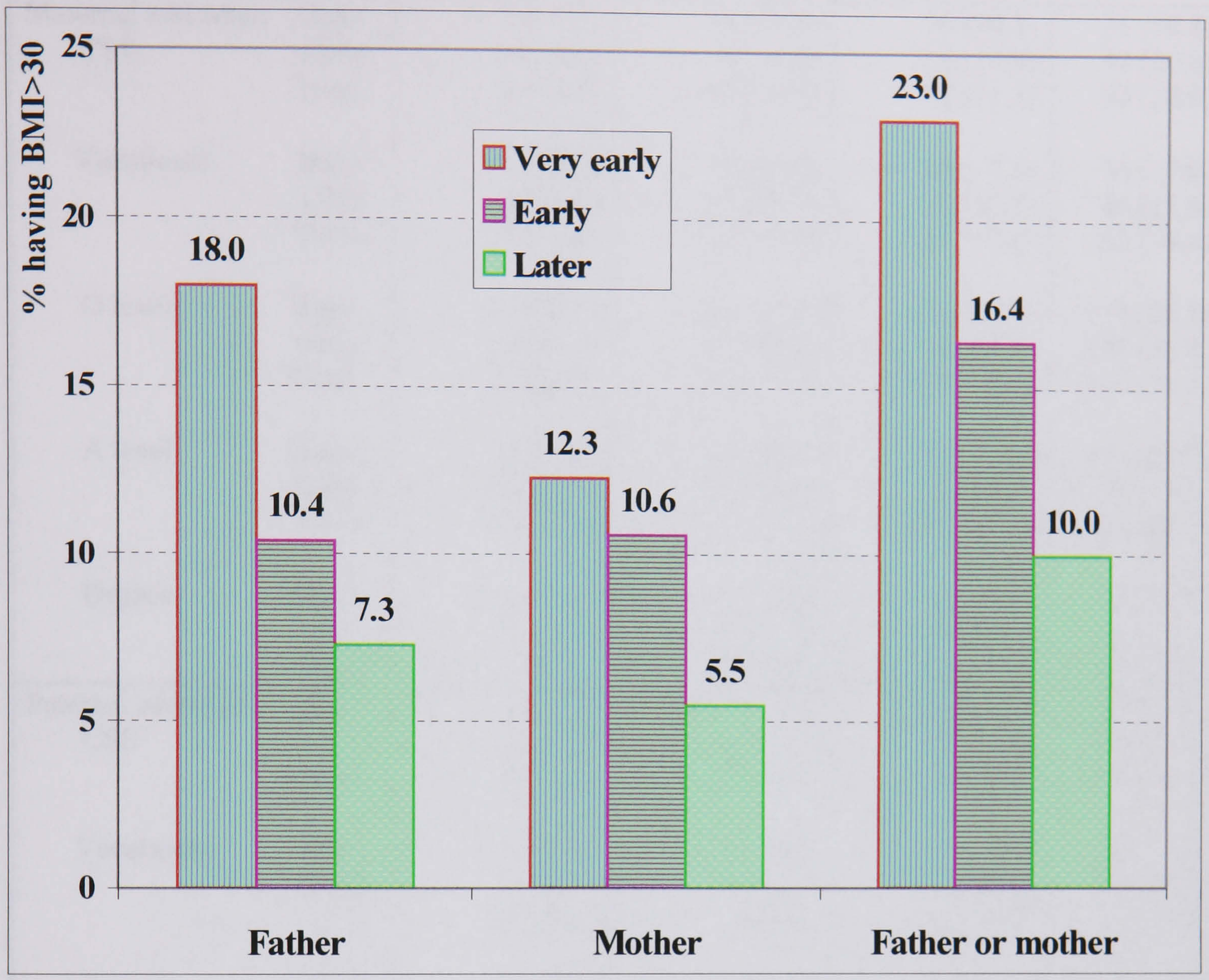
Parental obesity		Very early AR n (%)	Early AR n (%)	Later AR n (%)	Total n (%)
Father	Boys	* 4/23 (17.4)	**12/ 77 (15.6)	17/260 (6.5)	33/360 (9.2)
BMI >30	Girls	5/27 (18.5)	÷ 3/ 65 (4.6)	18/218 (8.2)	26/310 (11.9)
	Total	** 9/50 (18.0)	15/144 (10.4)	35/478 (7.3)	59/670 (8.8)
Mother	Boys	* 4/26 (15.4)	** 9/87 (10.3)	13/338 (3.8)	26/451 (5.8)
BMI >30	Girls	3/31 (9.7)	9/ 82 (11.0)	÷ 20/263 (7.6)	32/376 (8.5)
	Total	* 7/57 (12.3)	** 18/169(10.6)	33/601 (5.5)	58/827 (7.0)
Mum or	Boys	** 7/27 (26.0)	* 17/ 89 (19.1)	29/350 (8.0)	53/466 (11.4)
Dad	Girls	7/33 (21.0)	11/ 82 (13.4)	36/274 (13.0)÷	54/389 (13.9)
BMI >30	Total	** 14/60 (23.0)	28/171 (16.4)	65/624 (10.0)	107/855 (12.5)

* Significant difference with later rebound group, *$p<0.05$, **$p<0.01$

÷ Significant difference between boys and girls, $p<0.05$

Figure 6.5 presents the percentage of parents with BMI >30 in very early, early, and later adiposity rebound groups. As the results indicate, the percentage of parents with BMI >30 in the very early rebound group was significantly higher than the other rebound groups.

Figure 6.5. Percentage of parents with BMI >30 in very early, early, and later adiposity rebound groups*.



* Significant differences between very early AR and other groups.

Table 6.5 presents the parental education attainment and its relationship to the timing of childhood AR. Information about mothers educational attainment was available for 871 out of 889 (98.0%) and for 843 (95.8%) of fathers. Based on these results, most of the parents had an education equal to or more than O level. No significant relationships were found between adiposity rebound stages and different parental education levels.

Table 6.5. Parental education and relationship to timing of adiposity rebound.

Maternal / paternal Education		Very early AR n (%)	Early AR n (%)	Later AR n (%)	Total
Maternal education CSE	Boys	2 (7.1)	13 (14.4)	36 (10.1)	51 (10.8)
	Girls	3 (8.8)	4 (4.8)	36 (12.9)	43 (10.8)
	Total	5 (8.1)	17 (9.8)	72 (11.3)	94 (10.8)
Vocational	Boys	1 (3.6)	9 (10.0)	26 (7.3)	36 (7.6)
	Girls	3 (8.8)	13 (15.7)	30 (10.7)	46 (11.6)
	Total	4 (6.4)	22 (12.7)	56 (8.8)	82 (9.4)
O level	Boys	14 (50.0)	28 (31.1)	131 (36.8)	173 (36.5)
	Girls	12 (35.3)	37 (44.6)	93 (33.2)	142 (35.8)
	Total	26 (41.9)	65 (37.6)	224 (35.2)	315 (36.2)
A level	Boys	7 (25.0)	23 (25.6)	106 (29.8)	136 (28.7)
	Girls	11 (32.4)	22 (26.5)	81 (28.9)	114 (28.7)
	Total	18 (29.0)	45 (26.0)	187 (29.4)	250 (28.7)
Degree	Boys	4 (14.3)	17 (18.9)	57 (16.0)	78 (16.4)
	Girls	5 (14.7)	7 (8.4)	40 (14.3)	52 (13.1)
	Total	9 (14.5)	24 (13.9)	97 (15.2)	130 (14.9)
Paternal education CSE	Boys	1 (3.6)	15 (17.2)	46 (13.4)	62 (13.5)
	Girls	3 (9.1)	17 (21.0)	42 (15.6)	62 (16.1)
	Total	4 (6.6)	32 (19.0)	88 (14.3)	124 (14.7)
Vocational	Boys	4 (14.3)	4 (4.6)	17 (4.9)	25 (5.4)
	Girls	5 (15.2)	4 (4.9)	23 (8.5)	32 (8.3)
	Total	9 (14.8)	8 (4.8)	40 (6.5)	57 (6.8)
O level	Boys	7 (25.0)	27 (31.0)	79 (23.0)	113 (24.6)
	Girls	11 (33.3)	23 (28.4)	64 (23.7)	98 (25.5)
	Total	18 (29.5)	50 (29.8)	143 (23.3)	211 (25.0)
A level	Boys	9 (32.1)	22 (25.3)	108 (31.4)	139 (30.3)
	Girls	11 (33.3)	21 (25.9)	84 (31.1)	116 (30.2)
	Total	20 (32.8)	43 (25.6)	192 (31.3)	255 (30.2)
Degree	Boys	7 (25.0)	19 (21.8)	94 (27.3)	120 (26.1)
	Girls	3 (9.1)	16 (19.8)	57 (21.1)	76 (19.8)
	Total	10 (16.4)	35 (20.8)	151 (24.6)	196 (23.2)

No significant differences between timing of AR in different parental educational groups (in both boys and girls) were found.

Table 6.6 shows parental social class status and its relationship to childhood AR stage. Information about maternal social class was available for 711 out of 889 (80.8%) and paternal for 764 (85.9%) of children.

Table 6.6. Parental social class and relationship to timing of adiposity rebound.

Social class			Very early AR n (%)	Early AR n (%)	Later AR n (%)	Total
Maternal Social class	I, II & III-N	Boys	21 (95.4)	69 (90.8)	236 (83.7)	326 (85.8)
		Girls	22 (81.5)	59 (80.8)	187 (81.0)	268 (81.0)
		Total	43 (87.8)	128 (85.9)	423 (82.5)	594 (83.5)
	III-M, IV & V	Boys	1 (4.6)	7 (9.2)	46 (16.3)	54 (14.2)
		Girls	5 (18.5)	14 (19.2)	44 (19.0)	63 (19.0)
		Total	6 (12.2)	21 (14.1)	90 (17.5)	117 (16.5)
Paternal Social class	I, II & III-N	Boys	12 (57.1)	52 (66.7)	199 (63.8)	263 (64.0)
		Girls	21 (67.7)	41 (57.7)	149 (59.4)	211 (59.8)
		Total	33 (63.5)	93 (62.4)	348 (61.8)	474 (62.0)
	III-M, IV & V	Boys	9 (42.9)	26 (33.3)	113 (36.2)	148 (36.0)
		Girls	10 (32.3)	30 (42.3)	102 (40.6)	142 (40.2)
		Total	19 (36.5)	56 (37.6)	215 (38.2)	290 (38.0)

No significant differences between maternal / paternal social class and timing of AR.

No significant differences were seen between both sex and timing of AR.

6.4.2-c. Dietary intakes at 8 months

Absolute dietary and nutrient intakes (g/day) at age 8 months in relation to timing of the AR in 813 out of 889 children (whose dietary data at age 8 months were available) are presented in Table 6.7. As can be seen, there were no significant differences between rebound groups in either sex (or when data from both sexes were combined) in terms of nutrient intakes.

Table 6.7. Absolute macronutrient intakes at age 8 months.

Absolute Intakes		n	Protein (g/d) Mean (SD)	Fat (g/d) Mean (SD)	CHO (g/d) Mean (SD)	Energy (kJ/d) Mean (SD)
Boys	Very early rebound	26	28.9 (8.0)	32.8 (8.5)	114.7 (27.6)	3643 (848)
	Early rebound	89	27.8 (8.5)	33.3 (8.8)	106.4 (21.9)	3504 (678)
	Later rebound	334	28.4 (9.0)	33.1 (8.6)	110.8 (23.6)	3579 (175)
	Total	449	28.3 (8.7)	33.1 (8.6)	110.2 (23.4)	3568 (173)
Girls	Very early rebound	32	25.7 (7.4)	32.0 (9.0)	103.8 (20.8)	3376 (691)
	Early rebound	76	27.2 (8.2)	32.9 (8.8)	105.1 (23.2)	3456 (709)
	Later rebound	256	25.9 (8.1)	31.8 (9.4)	102.6 (21.5)	3348 (728)
	Total	364	26.2 (8.1)	32.0 (9.2)	102.9 (21.9)	3373 (721)
Total	Very early rebound	58	27.2 (7.8)	32.4 (8.7)	108.7 (24.5)	3496 (770)
	Early rebound	165	27.6 (8.3)	33.1 (8.8)	105.8 (22.4)	3482 (691)
	Later rebound	590	27.3 (8.7)	32.5 (9.0)	107.2 (23.0)	3479 (741)
	Total	813	27.4 (8.6)	32.6 (8.9)	107.1 (23.0)	3480 (732)

Table 6.8 shows the relative dietary and nutrient intakes at age 8 months (g/kg/day) in relation to timing of the AR. There were no significant differences in nutrient intakes between rebound groups in either sex or when data from both sexes were combined (based on the results, there was no evidence of differences in dietary intake and most notably protein intake at 8 months associated with the timing of the AR).

Table 6.8. Relative macronutrient intakes at age 8 months.

Absolute Intakes		n	Protein (g/kg/d) Mean (SD)	Fat (g/kg/d) Mean (SD)	CHO (g/kg/d) Mean (SD)	Energy (kJ/kg/d) Mean (SD)
	Very early rebound	26	3.1 (0.8)	3.5 (1.0)	12.2 (2.8)	388.1 (92.1)
Boys	Early rebound	88	3.1 (1.0)	3.7 (1.1)	11.8 (2.8)	386.4 (87.5)
	Later rebound	333	3.1 (1.0)	3.7 (1.0)	12.3 (2.6)	396.5 (81.6)
	Total	447	3.1 (1.0)	3.7 (1.0)	12.1 (2.7)	394.8 (82.9)
	Very early rebound	32	2.9 (0.9)	3.6 (1.0)	11.8 (2.5)	383.9 (82.1)
Girls	Early rebound	76	3.2 (1.0)	3.9 (1.0)	12.4 (2.5)	409.5 (80.0)
	Later rebound	253	3.1 (1.0)	3.8 (1.3)	12.2 (2.9)	398.2 (99.9)
	Total	361	3.0 (1.0)	3.8 (1.1)	12.3 (2.8)	400.2 (97.1)
	Very early rebound	58	3.0 (0.8)	3.6 (1.0)	12.0 (2.6)	386.0 (85.8)
Total	Early rebound	164	3.2 (1.0)	3.8 (1.0)	12.1 (2.7)	396.9 (84.6)
	Later rebound	586	3.1 (1.0)	3.7 (1.1)	12.2 (2.7)	397.3 (90.0)
	Total	808	3.1 (1.0)	3.7 (1.1)	12.2 (2.7)	396.5 (88.8)

As can be seen in Table 6.9, results of the relative dietary and nutrient intakes at age 8 months (percentage of energy from each macronutrient per day) in relation to the timing of the AR indicate no significant differences in relative dietary intakes between rebound groups in either sex or for the total sample.

Table 6.9. Percentage of dietary energy from macronutrient intakes at age 8 months.

Relative Intakes		n	Protein (%) Mean (SD)	Fat (%) Mean (SD)	CHO (%) Mean (SD)
	Very early rebound	26	13.3 (2.2)	33.9 (3.1)	52.8 (3.1)
Boys	Early rebound	89	13.2 (2.6)	35.7 (5.2)	51.1 (6.2)
	Later rebound	334	13.2 (2.7)	34.8 (4.9)	52.1 (5.5)
	Total	449	13.2 (2.6)	35.0 (5.0)	51.8 (5.6)
	Very early rebound	32	12.8 (2.5)	35.3 (4.7)	51.9 (5.9)
Girls	Early rebound	76	13.1 (2.7)	35.8 (5.2)	51.0 (5.6)
	Later rebound	256	12.9 (2.5)	35.4 (5.1)	51.7 (5.8)
	Total	364	12.9 (2.6)	35.5 (5.1)	51.5 (5.7)
	Very early rebound	58	13.0 (2.4)	34.7 (4.1)	52.3 (4.8)
Total	Early rebound	165	13.2 (2.7)	35.7 (4.1)	51.1 (5.9)
	Later rebound	590	13.0 (2.6)	35.1 (5.0)	51.9 (5.6)
	Total	813	13.1 (2.6)	35.2 (5.0)	51.8 (5.6)

6.4.2-d. Dietary intakes at 18 months

Absolute dietary and nutrient intakes (g/day) at age 18 months in relation to the timing of the AR in 772 out of 889 children (dietary data at age 18 months were available for 772 of the 889 subjects) are presented in Table 6.10. There were no significant differences between rebound groups in either sex in terms of nutrient intakes, or for the total sample.

Table 6.10. Absolute macronutrient intakes at age 18 months.

Absolute Intakes		n	Protein (g/d) Mean (SD)	Fat (g/d) Mean(SD)	CHO (g/d) Mean (SD)	Energy(kJ/d) Mean (SD)
	Very early rebound	25	43.7 (9.5)	50.2 (10.9)	145.5 (34.1)	5059 (999)
Boys	Early rebound	85	42.4 (10.0)	48.7 (10.2)	142.5 (23.9)	4932 (749)
	Later rebound	322	42.3 (10.4)	47.6 (11.9)	140.0 (28.8)	4845 (928)
	Total	432	42.4 (10.2)	47.9 (11.5)	140.8 (28.2)	4874 (900)
	Very early rebound	28	40.0 (9.2)	43.6 (10.2)	138.6 (29.5)	4633 (811)
Girls	Early rebound	71	40.1 (9.7)	45.1 (10.8)	128.5 (28.1)	4526 (807)
	Later rebound	241	39.8 (9.9)	45.5 (12.5)	131.0 (24.4)	4575 (995)
	Total	340	39.9 (9.8)	45.3 (11.9)	131.1 (29.1)	4569 (943)
	Very early rebound	53	41.7 (9.5)	46.7 (11.0)	141.9 (31.6)	4834 (921)
Total	Early rebound	156	41.3 (9.9)	47.1 (10.6)	136.2 (26.7)	4747 (800)
	Later rebound	563	41.2 (10.2)	46.7 (12.2)	136.2 (29.4)	4729 (966)
	Total	772	41.3 (10.1)	46.8 (11.8)	136.5 (29.0)	4740 (931)

Table 6.11 shows the relative dietary and nutrient intakes at age 18 months (g/kg/day) in relation to the timing of the AR. There were no significant differences in nutrient intakes between rebound groups in either sex or for both sexes combined (based on the results, there was no evidence of differences in dietary intake protein intake at 18 months associated with timing of the AR).

Table 6.11. Relative macronutrient intakes at age 18 months.

Absolute Intakes		n	Protein (g/kg/d) Mean (SD)	Fat (g/kg/d) Mean (SD)	CHO (g/kg/d) Mean (SD)	Energy (kJ/kg/d) Mean (SD)
Boys	Very early rebound	23	3.7 (0.9)	4.2 (1.0)	12.1 (3.7)	422.0 (105.5)
	Early rebound	82	3.6 (0.8)	4.1 (0.9)	12.1 (2.4)	418.7 (71.2)
	Later rebound	311	3.6 (0.9)	4.1 (1.1)	12.1 (2.7)	417.8 (85.0)
	Total	416	3.6 (0.9)	4.1 (1.0)	12.1 (2.6)	417.8 (83.7)
Girls	Very early rebound	28	3.5 (0.9)	3.8 (0.8)	12.2 (3.0)	407.4 (76.6)
	Early rebound	69	3.7 (0.9)	4.2 (1.1)	11.9 (2.7)	418.3 (77.9)
	Later rebound	235	3.6 (0.9)	4.1 (1.2)	11.9 (2.8)	414.9 (95.0)
	Total	332	3.6 (0.9)	4.1 (1.1)	11.9 (2.8)	414.9 (90.0)
Total	Very early rebound	51	3.6 (0.9)	4.0 (0.9)	12.2 (3.3)	414.1 (90.4)
	Early rebound	151	3.6 (0.8)	4.1 (1.0)	12.0 (2.6)	418.7 (74.1)
	Later rebound	546	3.6 (0.9)	4.1 (1.1)	12.0 (2.8)	416.6 (89.6)
	Total	748	3.6 (0.9)	4.1 (1.1)	12.0 (2.8)	416.6 (86.7)

As can be seen in Table 6.12, results of the relative dietary and nutrient intakes at age 18 month (percentage of energy from each macronutrient per day) in relation to the timing of the AR indicated no significant differences in relative dietary intakes between rebound groups in either sex or for both sexes combined.

Table 6.12. Percentage of dietary energy from macronutrient intakes at age 18 months.

Relative Intakes		n	Protein (%) Mean (SD)	Fat (%) Mean (SD)	CHO (%) Mean (SD)
Boys	Very early rebound	25	14.5 (2.2)	37.4 (3.6)	48.1 (5.0)
	Early rebound	85	14.4 (2.4)	37.1 (4.2)	48.6 (5.2)
	Later rebound	322	14.6 (2.2)	36.8 (4.9)	48.6 (5.7)
	Total	432	14.6 (2.2)	36.9 (4.6)	48.5 (5.6)
Girls	Very early rebound	28	14.4 (1.7)	35.4 (5.8)	50.2 (6.3)
	Early rebound	71	14.8 (2.3)	37.5 (5.5)	47.6 (6.4)
	Later rebound	241	14.6 (2.2)	37.2 (4.4)	48.2 (5.2)
	Total	340	14.6 (2.2)	37.1 (4.8)	48.2 (5.6)
Total	Very early rebound	53	14.5 (1.9)	36.3 (4.9)	49.2 (5.8)
	Early rebound	156	14.6 (2.4)	37.3 (4.8)	48.1 (5.8)
	Later rebound	563	14.6 (2.2)	37.0 (4.7)	48.4 (5.5)
	Total	772	14.6 (2.2)	37.0 (4.7)	48.4 (5.6)

6.4.2-e. Dietary intakes at 43 months

Absolute and relative macronutrient intakes (g/day and g/kg/day) at age 43 months in relation to timing of the AR in 709 out of 889 children (dietary data at age 43 months were available for 709/889) are presented in Tables 6.13 and 6.14. There were no significant differences between AR in either sex in terms of nutrient intakes.

Table 6.13. Absolute macronutrient intakes at age 43 months.

Absolute Intakes		n	Protein (g/d) Mean (SD)	Fat (g/d) Mean(SD)	CHO (g/d) Mean (SD)	Energy(kJ/d) Mean (SD)
	Very early rebound	25	50.8 (12.0)	59.5 (14.0)	192.5 (45.0)	6140 (1165)
Boys	Early rebound	77	47.9 (12.5)	56.8 (15.4)	177.5 (32.5)	5752 (1106)
	Later rebound	294	47.5 (11.4)	56.6 (13.5)	180.4 (34.6)	5785 (1039)
	Total	396	47.8 (11.7)	56.8 (13.9)	180.6 (35.0)	5801 (1061)
	Very early rebound	28	48.0 (12.6)	56.5 (12.6)	174.4 (26.8)	5692 (1024)
Girls	Early rebound	61	45.8 (10.4)	56.9 (11.6)	176.0 (30.0)	5695 (857)
	Later rebound	224	44.2 (11.3)	53.0 (12.1)	167.4 (33.1)	5388 (978)
	Total	313	44.9 (11.2)	54.1 (12.1)	169.7 (32.2)	5475 (967)
	Very early rebound	53	49.3 (12.3)	57.9 (13.3)	182.9 (37.3)	5903 (1106)
Total	Early rebound	138	47.0 (11.6)	56.8 (13.8)	176.8 (31.3)	5727 (1000)
	Later rebound	518	46.1 (11.4)	55.1 (13.0)	174.8 (34.6)	5613 (1031)
	Total	709	46.5 (11.6)	55.6 (13.2)	175.8 (34.2)	5657 (1033)

Table 6.14 shows the relative dietary and nutrient intakes at age 43 months (grams per kilogram weight per day) in relation to the timing of the AR. There were no significant differences in nutrient intakes between rebound groups in either sex or for both sexes combined (based on the results, there was no evidence of differences in dietary intake protein intake at 43 months associated with the timing of the AR).

Table 6.14. Relative macronutrient intakes at age 43 months.

Absolute Intakes		n	Protein (g/kg/d) Mean (SD)	Fat (g/kg/d) Mean (SD)	CHO (g/kg/d) Mean (SD)	Energy (kJ/kg/d) Mean (SD)
	Very early rebound	25	3.1 (0.8)	3.5 (0.8)	11.4 (2.3)	365.9 (60.3)
Boys	Early rebound	77	2.9 (0.8)	3.4 (1.0)	10.8 (2.2)	349.2 (71.7)
	Later rebound	284	2.9 (0.7)	3.5 (0.9)	11.1 (2.1)	357.2 (66.4)
	Total	386	2.9 (0.7)	3.5 (0.9)	11.1 (2.2)	356.2 (67.1)
	Very early rebound	28	2.9 (0.8)	3.4 (0.9)	10.4 (2.1)	339.8 (76.6)
Girls	Early rebound	60	2.9 (0.7)	3.6 (0.8)	11.2 (1.9)	361.1 (58.5)
	Later rebound	222	2.8 (0.7)	3.3 (0.8)	10.5 (2.2)	339.7 (65.1)
	Total	310	2.9 (0.7)	3.4 (0.8)	10.6 (2.1)	343.8 (65.3)
	Very early rebound	53	3.0 (0.8)	3.4 (0.8)	10.9 (2.2)	352.1 (70.0)
Total	Early rebound	137	2.9 (0.7)	3.5 (0.9)	11.0 (2.1)	354.4 (66.2)
	Later rebound	506	2.9 (0.7)	3.4 (0.8)	10.9 (2.2)	349.5 (66.3)
	Total	696	2.9 (0.7)	3.4 (0.8)	10.9 (2.2)	350.7 (66.5)

As Table 6.15 indicates, the relative dietary and nutrient intakes at age 43 months (percentage of energy from each macronutrient per day) were not significantly different between rebound groups in either sex or for both sexes combined. There was no evidence of differences in the diet of 43 month olds associated with timing of AR.

Table 6.15. Percentage of dietary energy from macronutrient intakes at age 43 months.

Relative Intakes		n	Protein (%) Mean (SD)	Fat (%) Mean (SD)	CHO (%) Mean (SD)
Boys	Very early rebound	25	13.6 (3.0)	35.4 (5.2)	50.9 (6.2)
	Early rebound	77	13.6 (2.9)	35.8 (4.8)	50.6 (5.3)
	Later rebound	294	13.4 (2.2)	35.7 (4.6)	50.9 (5.3)
	Total	396	13.4 (2.4)	35.7 (4.7)	50.8 (5.3)
Girls	Very early rebound	28	13.6 (1.8)	36.1 (2.9)	50.2 (3.9) ¤
	Early rebound	61	13.1 (2.5)	36.4 (4.1)	50.4 (5.1)
	Later rebound	224	13.4 (2.4)	36.0 (4.4)	50.7 (4.7)
	Total	313	13.3 (2.4)	36.1 (4.2)	50.6 (4.7)
Total	Very early rebound	53	13.6 (2.4)	35.8 (4.1)	50.6 (5.1)
	Early rebound	138	13.4 (2.7)	36.1 (4.5)	50.5 (5.2)
	Later rebound	518	13.4 (2.3)	35.8 (4.5)	50.8 (5.0)
	Total	709	13.4 (2.4)	35.9 (4.5)	50.7 (5.0)

Macronutrient intakes (g/kg/day) at ages 8, 18 and 43 months are presented in Figure 6.6. Changes in *relative macronutrient intake* across this period were negligible and were not statistically significant and this in part accounts for the absence of any association between macronutrient intake and the timing of the AR across this age range: macronutrient intake (relative intake) did not change much in the cohort over this age range.

Figure 6.6. Macronutrient intakes (g/kg/d) at ages 8, 18, and 43 mounts.

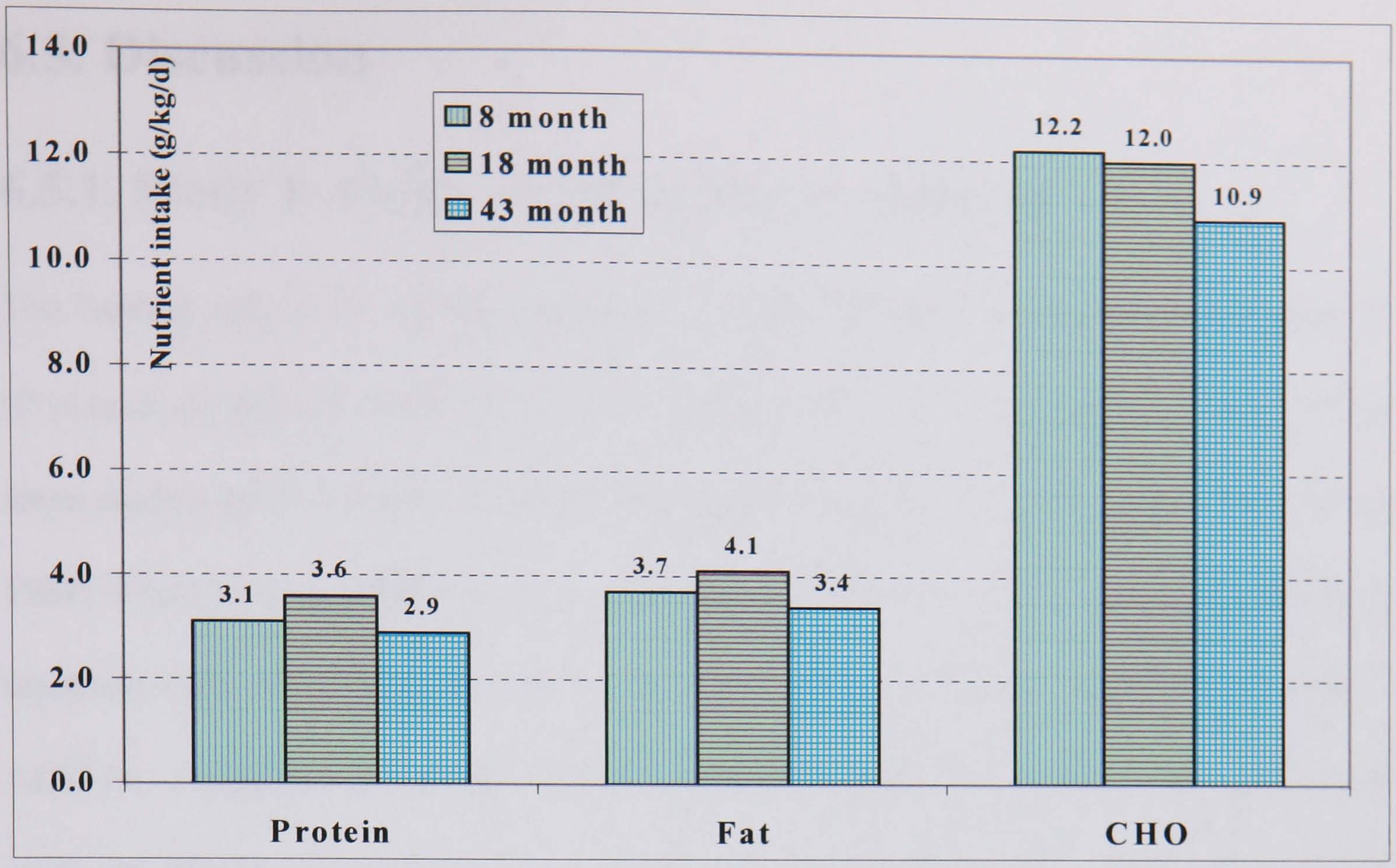
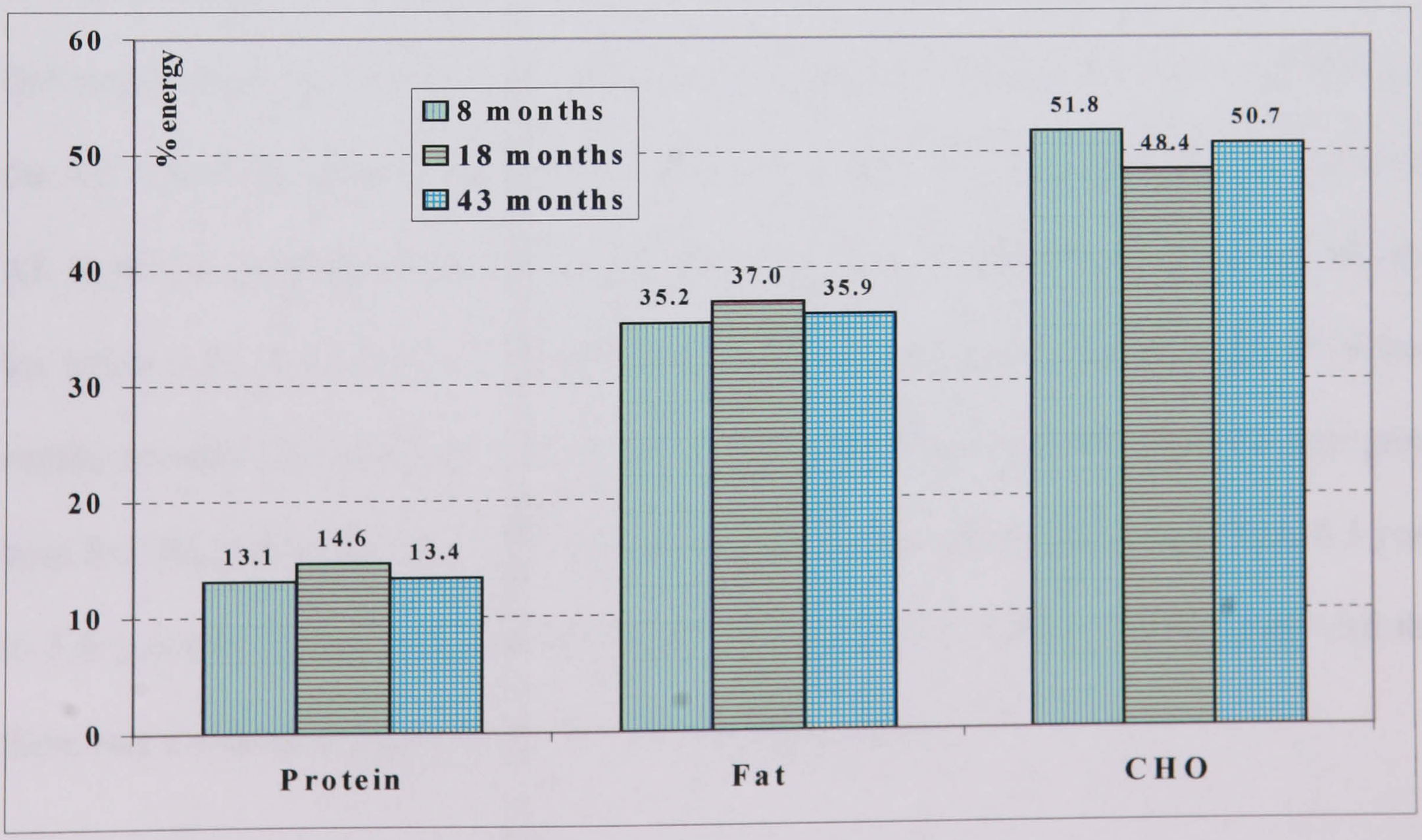


Figure 6.7 shows the relative dietary and nutrient intakes (percentage of energy from each macronutrient per day) at ages 8, 18, and 43 months. There were no significant differences in relative dietary intakes between different age groups.

Figure 6.7. Percentage of energy from macronutrients at ages 8, 18, and 43 months.



6.5. Discussion

6.5.1. Study 1: timing of AR in British children

The 'normal' age of the adiposity rebound is widely believed to be between 5-7 years of age (Prokopec & Bellisle 1993; Dietz 1994; White et al 1995; DeSpiegelaere et al 1998a). In some studies age 5.5 years was defined as early AR (Rolland-Cachera et al 1984; Salbe et al 1997; Whitaker et al 1998b), and some other investigators have assumed that ages 4-5 years represent early AR (Ogden et al 1997). Based on the results of the present study, most of the ALSPAC children had not had their adiposity rebound before age 4 years. No previous study on AR in British children with the same methodology was available to make any comparison. Since AR had not happened in all the children by age 5 years, defining the timing of AR in all the study children was not possible, therefore, comparisons with studies in other countries and at other times was difficult. Furthermore, previous studies in other countries had used their own specific methods to define AR and most of them used samples which experienced AR up to several decades ago (Rolland-Cachera et al 1984; Siervogel et al 1991; Prokopec & Bellisle 1993; Dietz 1994; White et al 1995; Salbe et al 1997; DeSpiegelaere et al 1998a; Whitaker et al 1998b). Making comparisons about the timing of the AR with those studies may not be valid. Cole et al (1995) suggested that the age of the AR in British children had decreased and that there was a secular trend to earlier AR after the 1960s (Cole et al 1995). A comparison between the age of the AR in 2 different French studies conducted in children born in 1955 (Rolland-Cachera et al 1982) and the next group born in 1985 (Deheeger et al 1997) showed that the mean age at AR changed from 6.3 years to 5.6 years in 30 years (Rolland-Cachera 1999). Rolland-Cachera (1999) concluded that there was a secular trend to earlier AR in children worldwide.

6.5.2. Study 2: factors associated with the timing of the AR

Obesity is now well known as a major public health problem and is the focus of many research investigations. Obesity prevention is important and there is considerable interest about critical periods which might be related to obesity development (Dietz 1997a). Learning how critical periods operate could help us to have a better understanding of energy balance regulation mechanisms as well as designing measures aimed at obesity prevention. Gestation and early infancy, the period of adiposity rebound (AR), and adolescence are the three most important known critical periods for obesity development (Dietz 1994). The age when the AR occurs has a significant influence on adulthood BMI and later obesity (Rolland-Cachera et al 1987; Prokopec & Bellisle 1993; Dietz 1997a; Whitaker et al 1998b). Understanding of the variables which might influence the AR, is therefore very important.

6.5.2-a. Timing of AR in relation to childhood variables

In the present study, no significant differences were found between boys and girls in terms of the timing of the AR. Girls with very early rebound had higher BMI SDS before the rebound than the rest (Table 6.2). After the rebound had occurred, BMI SD scores were significantly higher in children with very early rebound (Table 6.2). Since our sample was large, children who had very early AR were present in relatively large numbers. They experienced AR slightly earlier than mean values of all study samples from the literature (Rolland-Cachera et al 1984; Siervogel et al 1991; Prokopec & Bellisle 1993; Whitaker et al 1998b). This might in part reflect a secular trend to earlier AR (Cole et al 1995; Rolland-Cachera 1999) since, as noted earlier, most previous studies were carried out on cohorts which experienced AR about 30-40 years ago.

6.5.2-b. Timing of AR in relation to parental variables

Maternal BMI was significantly related to the timing of the AR in boys but this relationship was not significant in girls (Table 6.3). Paternal BMI was significantly related to the age of

the AR in girls (Table 6.3) while the relationship in boys was not significant. Similar trends were seen in another study on the relationship between parental variables and the timing of the AR (Whitaker et al 1998b) though the trends were not significant. This observation should be studied further. Mean parental BMI and having one obese parent was significantly associated with earlier AR. Regarding the results presented in Tables 6.3 to 6.6 and Figures 6.4 to 6.5, with the exception of parental BMI, no relationships between the timing of the AR and any other variable were found. However, it should be noted that parental BMI was based on self-reported weight and height. Self-reporting of height, and to a greater extent weight, are subject to small but significant biases (Rowland 1990). This might also explain the relatively low prevalence of parental obesity in the sample (discussed later).

6.5.2-c. Protein intake and the timing of the AR

In a French study published in 1995 (Rolland-Cachera et al 1995), it was reported that higher protein intake was associated with earlier AR. It has since been suggested by the same authors that a high protein intake might promote early AR by altering growth hormone / IGF-1 secretion (Rolland-Cachera 1999). However, the original reference by these authors (Rolland-Cachera et al 1995) did not actually present evidence to support the statement that timing of AR and protein intake were linked. In addition, a number of limitations were acknowledged in the original study (Rolland-Cachera et al 1995). These included a relatively small sample size (n=112 boys and girls) with high dropout rates (61%); only 4 measurements of BMI were available to define the timing of the AR; dietary assessment covered only a one day period. In the light of these limitations, Rolland-Cachera et al (1995) recommended that the hypothesis that protein intake was associated with the timing of the AR should be tested in other studies. The present study tested this hypothesis with improved methodology, more available measurements of BMI and with a larger cohort. In the present study, the sample size and dietary methodology were much more robust than in the earlier French study and the hypothesis was not supported.

6.5.2-d. Macronutrient intakes at 8, 18 and 43 months and the timing of the AR

The present study was the first attempt to do a study similar to the French one (Rolland-Cachera et al 1995). The present study tested the hypothesis that higher protein intake in early childhood was associated with earlier AR. As the results of Tables 6.10 to 6.12 show, no evidence was found to support the protein intake hypothesis and there were no differences between other macronutrient intakes at 18 months associated with the timing of the AR. In addition to 18 months, in the present study other available ALSPAC macronutrient intakes (at 8 and 43 months) were analysed and no association with the timing of the AR was found (Tables 6.7 to 6.9 and 6.13 to 6.15 and Figures 6.6 to 6.7). It is worth noting that we tried several different categories to define very early, early, and later AR groups, but no differences in macronutrient intakes were found between groups of children based on age of any rebound categories. However, in the present study, the mean percentage of energy from protein was slightly lower (Table 6.16) than in the French study (Rolland-Cachera et al 1995) and this difference was statistically significant (two sample T-test, $p<0.001$).

Table 6.16. Mean energy (%) from macronutrients in 8, 18, and 43 month old children in the present study and in 2 year old children in Rolland-Cachera et al (1995) study.

Mean energy	Rolland-Cachera et al (1995) Age 2 years	Present study Age 8 months	Present study Age 18 months	Present study Age 43 months
% from Protein	† 16.3 (2.4)	13.1 (2.6)	14.6 (2.2)	13.4 (2.4)
% from Fat	† 32.7 (5.1)	35.2 (5.0)	37.0 (4.7)	35.9 (4.5)
% from Carbohydrate	‡ 51.0 (6.2)	51.8 (5.6)	48.4 (5.6)	50.7 (5.0)

† Significant differences between Cachera and present study results at all ages ($p<0.001$).
‡ Significant difference between Cachera and present study at 18 months only ($p<0.001$).

The results from the dietary data at ages 8, 18 and 43 months did not differ significantly in terms of associations with the timing of the AR.

Rolland-Cachera et al (1995) reported that their early rebound children (≤4 years; n=38) and late rebound group (≥8 years; n=15) consumed 16.6 ± 2.1% and 14.9 ± 2.1% of energy from protein respectively and this difference was significant (Mann-Whitney U test, $p<0.01$). On average, children in her study consumed 1397 (SD=269) kcal/day; 16.3% (SD=0.4) from protein, 32.7% (SD=1.7) from fat and 51.0% (SD=3.2) from carbohydrate. The French study results were different not only from the present study but also from the British national data on young children. For example, the results of the UK National Diet and Nutrition Survey (NDNS 1998) of 1.5-4.5 year old children indicated that the mean ± SEM (standard error of mean) energy intake for all British children was 1141 ± 6.3 kcal/day (much lower than the French study). Based on the NDNS results, 13.0% ± 0.06 (mean ± SEM) of energy was from protein, 35.9% from fat, and 51.1% ± 0.15 from carbohydrate.

6.5.2-d. Limitations of Rolland-Cachera et al (1995)

The French follow-up study reported by Rolland-Cachera et al (1995) started in 1985 (some time ago, before the recent obesity epidemic) in a population consulting for clinical examinations and a sample of 278 children were studied at the age of 10 months. From those, 192 (69.1%) came back at 2 years and 162 (58.3%) at 4 years age, and at the ages of 6 and 8 years 126 (45.3%) and 112 (40.3%) children participated. BMI charts based on only 4 points were drawn and adiposity rebound was defined visually. This sample size was small and perhaps non-representative of the whole childhood French population. In contrast, ALSPAC started from 1991 and from a total of 1409 children at 4 months, 972 (67.0%) still participated at age 61 months, until BMI for 889 (63.1%) of them measured from 4 to 61 months of age and dietary data were available for 808 (57.3%) of them.

The French study (Rolland-Cachera et al 1995) used a one-day daily intake (24-h recall method) asking mothers to remember a typical day's eating pattern which could cause its classification of diet to be rather inaccurate. Nutrient intake was evaluated according to the British food composition tables (Holland et al 1991). In the present study, a prospective 3-day (unweighed) dietary record was collected. Prospective dietary records are a better way of collecting

information about diet (Bingham et al 1994) and 3-days is usually regarded as a reasonable length of record to classify macronutrient intakes in groups of young children (Nelson et al 1989; chapter 2).

6.5.2-e. Other variables and their influence on the timing of the AR

Activity level

In the Deheeger et al cross-sectional study on 10 year old French children (Deheeger et al 1997), 86 boys and girls were studied from age 10 months and BMI and skinfolds were measured every 2 years. At age 10 years, based on the results of a short, self-reported activity questionnaire of the past year's activities, children were divided into active and less active groups. The authors concluded that the age of the AR in 'less active' children at age 9-10 was significantly earlier (5.4±1.8 years versus 6.1±1.5 years; Deheeger et al 1997). This implies that inactivity might promote earlier AR but is not conclusive since the Deheeger et al study measured activity levels long after the rebound had happened, and used self-reported measures of activity which can be inaccurate in children (Freedson 1989; Sallis 1991; Pate 1993; Epstein et al 1996; Weston et al 1997).

Socioeconomic status

In a recent study, DeSpiegelaere et al (1998a) investigated the role of socioeconomic status on the timing of the AR. They measured BMI at age 3 and 5 years in 675 children from Brussels (328 girls and 348 boys) children were divided into 5 different socioeconomic groups based on parental occupation (DeSpiegelaere et al 1998a). A child was defined as having an early adiposity rebound if BMI at age 5 was greater than that at age 3. Adiposity rebound occurred 'early' in 30.4% of the children (before age 5) and the proportion did not differ between the social class groups (DeSpiegelaere et al 1998a). Those authors suggested that the percentage having AR by age 5 years was very close to that found by Rolland-Cachera (30.5% based on data from almost 30 years ago: Rolland-Cachera et al 1984). In the DeSpiegelaere et al (1998a) study no relationship between socioeconomic status and the timing of the AR was found and in the present study the same results were found. However, since the AR definition methodology in

the present study differs from the DeSpiegelaere et al (1998a) and other studies, such a detailed comparison may not be valid. In particular, DeSpiegelaere et al (1998a) used only two measurements of BMI in order to define the timing of the AR, which was not ideal.

Energy expenditure and physical activity

No study published to date was found which tested the relationship between childhood energy expenditure and the timing of the AR. In the ALSPAC study, energy expenditure in the sample children was not measured and so, we could not judge the possible effect of energy expenditure on the timing of the AR. There are no published data from prospective studies on the relationship between physical activity and the timing of the AR.

6.6. Limitations of the present study

6.6.1. Data limitations

No data are available on differences in prevalence of AR between different ethnic groups / races in the UK. It is conceivable that genetic factors might be important in influencing the timing of the AR (Dietz 1997b); since in the present study genetic data were not obtained, the relative importance of genotype and environment on the AR still remains unclear. This issue was mentioned by Whitaker et al (1998b). However, the recent secular trend to earlier AR implies a degree of environmental influence since population genetic changes can not have occurred over this short time period (Cole et al 1995; Rolland-Cachera 1999). It was not possible to include all of the potentially important variables in the ALSPAC protocol as discussed in Chapter 2.

As noted above, activity level, which has a major role in the development of childhood obesity (Moore et al 1995; Westerterp & Goran 1997; Dietz 1998a; Goran et al 1998a; Maffeis et al 1998), was not measured in the ALSPAC study children. Energy expenditure was not measured since the available methods were time consuming and very expensive and not appropriate to an epidemiological study (deBruin et al 1996). However energy expenditure might well influence the timing of the AR (Roville-Sausse 1999). It is known that energy expenditure can determine

energy balance in children (Goran & Sun 1998). Physical inactivity, such as TV watching duration in children, was not measured quantitatively in the ALSPAC study but this seems to be one of the most important issues (DuRant et al 1994; Gortmaker et al 1996) in risk of childhood obesity and potentially in the timing of the AR.

6.6.2. Other limitations

The present study children had families who were wealthier than other British children as described in Chapter 2. In the ALSPAC study, parental weight and height measurements had not been available for all children and were self-reported. The percentage of obesity (defined as having BMI >30 kg/m^2) in our study fathers was 8.8% and was 7.0% in mothers in 1990/91 (Table 6.4) which was less than other British studies reports (8.0% and 12.0% in men and women by 1987; 13.5% and 16.5% by 1994; Jebb 1999). Self-report records are certainly not as accurate as proper measurement (Rowland 1990). The lower prevalence of parental obesity in Children in Focus probably reflects self-report bias and the over-representation of wealthier families. The dietary data collected for the children, though using a reasonably reliable method, may not have accurately reflected their habitual intake. Early AR can be the result of gestational diabetes and might be a marker for early maturation (Whitaker et al 1998b) but these variables were not assessed in the present study. On the other hand, the present study was by far the largest ever to test for factors which influence the timing of the AR (Dietz 2000a).

Parental control over high-fat and energy-dense foods, might limit children's acceptance of a variety of foods and decreases the ability of children to regulate their energy intake by altering their responses to hunger and satiety (Birch & Fisher 1998). Therefore, overly controlling child-feeding practices may affect children's ability to regulate energy intake and the amount of food consumed. In white middle class American children, a high degree of parental control was associated with low self-control of food intake in children (Birch & Fisher 1998). Parental control might therefore influence the timing of the AR in children but no measures of parental control over child feeding were obtained in the ALSPAC study.

6.7. Conclusions

The results of study 1 indicated that about 7% of children had their AR by age 43 months (very early rebounders) and that AR occurred in 27% of children by age 49 months (early rebound). Data were available up to age 61 months at the time of writing and therefore it was not possible to classify the other children at this stage.

The results of study 2 suggested that dietary energy and macronutrient intakes are not associated with the timing of the AR. In particular, there was no evidence that higher protein intake was associated with early AR. The present study does not suggest simple markers of early AR which might act as the targets of further research or obesity prevention, with the exceptions of high parental BMI or parental obesity. In the absence of early AR markers, the factors currently known to be associated with obesity risk, including age and parental obesity (Whitaker et al 1997), inactivity / TV viewing (Gortmaker et al 1996), and pre-school activity (Moore et al 1995) should continue to be used as the principal targets of obesity prevention measures in young children. Further research on the factors which determine the timing of the AR is indicated.

7. Chapter 7: Risk factors for childhood obesity

7.1. General introduction

Childhood obesity is a serious and growing public health problem world wide as described in Chapters 1 and 3-6 and is an important risk factor for adult obesity as well. Adults who were obese as children have increased mortality independent of adult weight (Schonfeld-Warden & Warden 1997). Thus, obesity prevention programs for children and adolescents could have long-term benefits (Schonfeld-Warden & Warden 1997) and new ideas about the biologic basis of obesity offer hope for effective solutions (Schonfeld-Warden & Warden 1997). Investigating the factors which might affect the prevalence and the incidence of childhood obesity is an urgent matter (Dietz 2000b).

Obesity risk factors in children and adolescents

There are a number of studies on risk factors for childhood obesity in the literature from different countries. These are of limited value in understanding risk factors for modern British children for the following reasons. First, they used different criteria to define childhood obesity and many of the definitions would not be regarded as acceptable today. Second, they focused on many different variables as risk factors, therefore making comparisons between their results is difficult. Third, many of them are based on studies from other countries and have investigated variables (such as social factors) which are probably specific to the study samples or populations. Finally, as described in Chapter 3, the childhood obesity epidemic in the UK occurred in the 1990s; therefore studies before the 1990s are probably of limited relevance. A summary of the main potential obesity risk factors in children and adolescents is given below.

7.1.1. Intra-uterine factors and prevalence of childhood obesity at age 7 years

7.1.1-a. Gestational age

A few studies have focused on gestational age and its effect on childhood obesity risk. Parsons et al (1999) observed that in the studies which attempted to address the relationship between childhood obesity and gestational age, results were inconsistent (Parsons et al 1999). However, the O'Callaghan et al (1997) study of 4062 5 years olds in Australia showed that being small for gestational age was a protective factor for overweight (BMI 85th-94th centile) at age 5 years. In another study of 1213 black and 1166 white 9-10 year old American girls, the odds ratio of maternal age for increase childhood overweight (BMI $\geq 85^{th}$ centile) was 1.14 for each 5 year increasing in maternal age (Patterson et al 1997). No British study has been found that attempts to investigate the effect of gestational age on the prevalence of childhood obesity.

7.1.1-b. Catch-up growth

Early catch-up growth as a risk factor for coronary heart disease was studied in 3641 male subjects from Helsinki (Finland) born during 1924-33 with BMI available yearly from birth (Eriksson et al 1999). Those subjects who died from coronary heart disease had an above average BMI at all ages from 7 to 15 years, and the highest death rate was seen in boys who were thin at birth but whose weight caught up quickly so that they had an average or above average body mass from the age of 7 years ((Eriksson et al 1999). In an Indian cohort study of 477 children from birth to age 8 years, it was seen that the children who were born small, had rapid weight / height growth and were the most insulin resistant and had the highest levels of cardiovascular risk factors including higher body fatness and more central fat distribution by 8 years old (Yajnik 2000).

The only available British study on the relationship between early catch-up growth and childhood obesity was based on the ALSPAC study. Ong et al (2000) using a sample of 848 CIF children up to 5 years of age, assessed catch-up growth (defined as gaining weight SD score >0.67 SD scores between birth to 2 years; 0.67 SDS represent the width of each percentile band on standard growth charts that is 2nd to 9th, 9th to 25th, 25th to 50th, and so on). It was shown that children who had shown catch-up growth were fatter (fat mass was estimated by 4 skinfold measurements) at age 5 years (Ong et al 2000).

7.1.1-c. Birth weight

Birth weight of British children has been changing over time. Power (1994) showed an increase in the percentage of heavier birth weights, and regression of birth weights >3500 gram against year indicated that heavier births increased by 0.40% a year in Scotland (data available from 1975 onwards) and by 0.35% in England and Wales (data from 1983 onwards). At the same time, the proportion of birth weight 2500-2999 g and 3000-3499 g steadily decreased but the proportion of low birth weights (<2500 g) remained unchanged (Power 1994). Increasing birth weight might be related to childhood obesity. For instance, in the Duran-Tauleria et al (1995) study of English and Scottish 5-11 years old children, birth weight was positively associated with obesity (defined based on weight for height and 2 skinfolds; Duran-Tauleria et al 1995).

Birth weight was a predictor for overweight (BMI 85th - 94th centile) and obesity (BMI >94th centile) at age 5 years in the O'Callaghan et al (1997) cohort study on 4062 Australian children. Large-for-gestational-age German infants displayed a significantly higher percentage of overweight (mean relative weight [weight divide by median standard weight for age and sex] >1.1) in childhood than appropriate-for-gestational-age infants ($p<0.05$; Plagemann et al 1997). In a German study of 7-14 year old children, statistically significant positive associations with overweight were found for birth weight in both sexes (Kromeyer-

Hauschild et al 1999). The weight and height data of 9261 children from birth onwards were gathered on their admission to primary school in 1989 in the area of Lyon (France), and compared to French reference data. High birth weight increased the risk of obesity at age six (Lehingue et al 1993). A case controlled French study on 1031 5 year old school children showed that birth overweight was closely related to child obesity (defined as weight for height ≥2SD of the French reference data) at 5 years of age (relative risk 2.4; Locard et al 1992). Rasmussen & Johansson's (1998) nationwide cohort study on 165109 children followed from birth to age 18 years indicated that high birth weight was a risk factor for overweight / obesity among young men in Sweden. Being 'overweight' at birth (defined as birth weight ≥3500 grams) significantly influenced the development of obesity in 3 year old Japanese children (n=1281; Takahashi et al 1999).

The focus of this Chapter is on risk factors in childhood obesity but it is worth mentioning the Sorensen et al (1997) study of the relationship between birth weight and early adulthood BMI in a large sample study of 4300 18-26 years old Danish subjects. The study results showed that birth weight was positively associated with body mass index 20 years later (Sorensen et al 1997). In another study on 1750 adult subjects born in UK between 1920 and 1930, BMI in adulthood rose with increasing birth weight (Phillips & Young 2000).

In spite of all above-mentioned studies, there are some doubts about the effect of birth weight on childhood obesity prevalence. For instance, neither the Danish (Sorensen et al 1997) nor the British (Seidman et al 1995) studies showed that birth weight was a risk factor for childhood obesity. Furthermore, the methodology used to identify obesity in children was very different in the past. Therefore, possible associations between birth weight and childhood obesity had to be tested in a large, contemporary sample of UK children with an acceptable definition of obesity.

7.1.2. Infant feeding / weaning and obesity prevalence at age 7

7.1.2-a. Breast-feeding

Infants who receive breast milk as the sole source of milk for at least 12 months are generally leaner than their formula-fed counterparts (Dewey et al 1993). Lucas et al (1980 and 1981) found significantly higher plasma concentrations of insulin in French infants who had been bottle-fed than in infants who had been breast-fed, which might stimulate fat deposition and the early development of adipocytes (Lucas et al 1980; Lucas et al 1981). Breast milk contains both bioactive factors (which may modulate epidermal growth factor) and tumour necrosis factors (which inhibit adipocyte differentiation in vitro; Petruschke et al 1994; Hauner et al 1995). The amount of energy metabolised and the protein intake of breast-fed children is significantly below the intake of formula-fed infants (Heinig et al 1993; Whitehead 1995) but mean total energy expenditure may be significantly higher in formula-fed infants (Davies et al 1990).

In spite of the fact that breast-feeding might have a positive effect on reducing childhood obesity risk, the number of studies in this field are few since a widely accepted simple obesity definition was introduced only recently (Barlow & Dietz 1998). In addition, many previous studies have been too small to adequately test for any associations (Wadsworth et al 1999). In a recent cross-sectional study on 9357 German children aged 5-6 years, the prevalence of obesity (BMI >97th centile) in children who had never been breast-fed was 4.5% compared to 2.8% in breast-fed children (von Kries et al 1999). Therefore, an apparent 35% reduction in prevalence of childhood obesity occurred if children were breast-fed for 3 to 5 months. A clear dose-response effect was identified for the duration of breast-feeding

on the prevalence of obesity: the prevalence was 3.8% for 2 months of exclusive breast-feeding, 2.3% for 3-5 months, 1.7% for 6-12 months, and 0.8% for more than 12 months. Similar relations were found with the prevalence of being overweight (BMI >90th centile; von Kries et al 1999). After apparently adjusting for some potential confounding factors (but not for social class), breast-feeding remained a significant protective factor against the development of obesity and overweight (odds ratios 0.75 and 0.79; von Kries et al 1999). Kramer (1981) studied 1172 12-18 year old Canadian subjects and reported that breast-feeding protected against later obesity (Kramer 1981).

There was no information available about the role of breast-feeding on British childhood obesity prior this study and there was some doubt as to whether or not the study by von Kries et al (1999) on 9357 5-6 year old German children adequately corrected for effects of social class on breast-feeding (Wadsworth et al 1999). Wadsworth et al (1992) using data from the longitudinal study of 3731 6 year old British children born in 1946 found no significant relation of breast-feeding with overweight (BMI >90th centile) or obesity (BMI >97th centile) at age 6. It is widely believed that preventive measures in infants may include the promotion of breast-feeding and a delay in the introduction of solid foods (Leung & Robson 1990), but there is probably limited evidence for this. Possible associations of breast-feeding and childhood obesity risk in recent British data with the recent obesity definition (BMI ≥95th centile; Barlow & Dietz 1998) were therefore worth testing.

7.1.2-b. Timing of the introduction of solid foods

The Department of Health and Social Security (DHSS 1994) recommended that for British children 'The majority of infants should not be given solid food before four months'. Exclusive breast-feeding is recommended for at least four months, with solid foods being introduced thereafter, (DHSS 1988; DHSS 1994) but the DHSS reported that in 1994 only 64% of UK infants were breast-fed initially and only 19% were breast-fed at 4 months of age. In addition, 90% of infants started eating solid foods before the age of 4 months (DHSS 1994). A study of 98 British children showed that the median age at the introduction of solid food was 11 weeks (range 4-35 weeks) and that only 7% of infants had not received solids before age 16 weeks (Savage et al 1998). These results showed that heavier infants were weaned earlier and that weight at one month of age was negatively correlated with the age of solid food introduction ($p < 0.05$; Savage et al 1998).

A relatively recent British study on 412 6-10 year old children followed from infancy showed that children who had been given solids before 15 weeks were significantly heavier than those given solids at 15 weeks or later (mean weight SDS 0.02 vs. -0.09). The percentage of body fat measured either by skinfold thickness or impedance was significantly greater in the children who had been given solid foods before 15 weeks of age (mean body fat from impedance 18.5% vs. 16.5%; Wilson et al 1998). Further studies on the association of the early introduction of solid foods with childhood obesity in the UK will add to these data.

7.1.3. Demographics / other variables associated with childhood obesity prevalence

7.1.3-a. Effect of gender on the prevalence of childhood obesity

Some study results have suggested a relationship between the gender of the child and the prevalence of childhood obesity. For instance, a study of 4477 7-19 year old African rural subjects showed that the prevalence of overweight (BMI >85th and sum of two skinfolds >85th NHANES III centiles) was significantly higher in females than males after age 14 years (Cameron & Getz 1997). The effect of gender on childhood obesity has not only been described by African and African-American studies, but also some other studies reported similar results. For example, in a prospective Australian cohort study of 4062 children at birth, 6 months and age 5 years, female gender was a predictor of overweight (BMI 85th - 94th centiles) and obesity (BMI >94th centile) at age 5 years (O'Callaghan et al 1997). In the Ogden et al (1997) study on 7500 American children up to 6 years of age during 1988-1994 (NHANES III national survey), the prevalence of overweight (weight-for-stature or weight-for-length >95th NCHS centile) among children was consistently significantly higher in girls than boys. However, gender had no association with prevalence of childhood obesity in other studies. For instance, among 7300 10-12 year old French children, the prevalence of obesity (weight for height ≥2SD) was not found to be significantly different between boys and girls (Locard & Gemelli 1989). Gender did not contribute to childhood obesity risk in 6 year olds followed-up in the American study of 2913 0-8 year old children (Strauss & Knight 1999).

No British study prior to the date of starting this thesis tested for sex differences in childhood obesity risk. Since 1999, several British studies have shown that child gender has no major effect on the prevalence of childhood obesity. Reilly & Dorosty (1999) report on a nationally representative sample of 2630 6-15 year old English children, and Reilly et al (1999) reoport on 1031 2-5 year old British children (a sub-sample of ALSPAC) showed no effect of gender on the

prevalence of childhood obesity. Kinra et al (2000) carried out a cross-sectional study of 20973 5-14 year old Plymouth (UK) children during 1994-96. They did not find marked differences in obesity (BMI >98th centile) prevalence between the sexes. In the Bundred et al (2001) retrospective study on 64430 children under 4 years of age in the north-west of England, the prevalence of overweight (BMI >85th centile) and obesity (BMI >95th centile) was slightly but not significantly higher in boys. The recent study by Chinn & Rona (2001) on 20151 English and 10604 Scottish 4-11 year old children used IOTF 2000 BMI cut-off values (Cole et al 2000) to define overweight / obesity in children. They calculated the prevalence of obesity in 1994 as 1.7% in English boys, 2.1% in Scottish boys, 2.6% in English girls, and 3.2% in Scottish girls but they did not mention if differences in the prevalence of obesity between the sexes were statistically significant. In addition, the difference in prevalence might be related to the lower sensitivity of the IOTF definition of obesity for boys, as described in Chapter 5. Therefore, the effect of gender on the prevalence of childhood obesity in British children still remained in doubt prior to the present investigation.

7.1.3-b. Parental education

Only a few studies have focused on the role of parental education on the prevalence of childhood obesity, none of which were British. In the Strauss & Knight (1999) study, it was shown that the American children whose mothers did not complete high school education were significantly more likely to be obese by the 6 year follow-up (Strauss & Knight 1999). In a case-control study of 220 obese (BMI >90th centile of French age-sex reference data) and 220 non-obese children aged 6-18 years in Al Ain (United Arab Emirates) during 1992-1993, maternal low education was a significant factor for the development of obesity (Moussa et al 1994). In 1213 black and 1166 white 9-10 year old American girls, overweight was defined as BMI ≥85th percentile and in whites the odds of overweight was higher for girls who had parents or guardians with lower levels of educational attainment (Patterson et al 1997). It should also be noted that in most countries social class, income, and educational level are closely associated.

7.1.3-c. Parental obesity

Most US studies consider parental obesity as an important risk factor for childhood obesity. For example, in an American study on 40000 children aged 1-17 years, results showed a significant correlation (r = 0.25) between parent and child fatness (defined based on triceps skinfold). This correlation increased with increasing age of children (Garn & Clark 1976). Whitaker & Dietz (1998) classified maternal obesity and pregnancy weight gain as factors which alter the intra-uterine environment and increase the risk of later obesity in the offspring (Whitaker & Dietz 1998) and Cutting et al (1999) suggested maternal BMI as a predictor of daughters' overweight for behavioural reasons (Cutting et al 1999). In an American study on 2913 normal weight 0-8 year old children who were followed over a 6 year period, maternal obesity was the most significant predictor of childhood obesity (BMI >95th percentile for age and gender; Strauss & Knight 1999). However, Whitaker et al (2000) studied 85 36 month-old white American children and indicated only a weak relationship between parental BMI and child adiposity (Whitaker et al 2000).

Several studies in other parts of the world have suggested an effect of parental obesity on the risk of childhood obesity. For example, in the Locard et al (1992) study, parental overweight was strongly related to childhood obesity (weight for height ≥2SD of the French reference data) at 5 years of age. The relative risk of childhood obesity in the children of overweight parents was 3.1 compared to the children of non-overweight parents (Locard et al 1992). In a prospective observational study of 112 Italian pre-pubertal children started in 1992 and followed-up in 1996, maternal and paternal BMI were the only variables able to predict relative BMI (relative BMI = BMI / BMI at 50th centile for age and gender, multiplied by 100) in the children (Maffeis et al 1998). In a sample of 530 7-11 year old Italian children, the relationship between the children's adiposity and their parents' BMI was confirmed (Maffeis et al 2000). Parental overweight (BMI >25) was a major risk factor for childhood

overweight (BMI >90th centile of Rolland-Cachera curves) in the first years of life in a study on 147 healthy Italian children followed-up at birth, 1 and 5 years of age (Scaglioni et al 2000). A significantly higher mean percent of ideal body weight was found in Italian children of obese parents (Franzese et al 1998). Paternal BMI was a predictor for overweight (BMI 85th - 94th centile) while both maternal BMI and paternal BMI were predictors for obesity (BMI >94th centile) at age 5 years of 4062 Australian cohort study children (O'Callaghan et al 1997).

A British cross-sectional study of 10628 children from 22 English areas in 1990 and 4165 children from 14 Scottish areas in 1990-91 aged 5-11 years confirmed that maternal BMI and paternal BMI were consistently associated ($p<0.001$) with childhood obesity (defined using sum of 2 skinfolds; Duran-Tauleria et al 1995). Data from 11 year old British children (n=1958) studied in the 1990s showed that the mean BMI of the children increased as parental BMI (self-reported) increased (Lake et al 1997). A British study of 203 5 year old children showed a significant correlation between the weights of the mothers and the weights of their children at 5 years of age, though the children of overweight mothers did not have higher energy intakes (Poskitt & Cole 1978).

The effect of parental obesity on the risk of childhood obesity has not only been described in the USA and European countries, but also similar results have been shown by other studies too. For example, maternal obesity (BMI >24) and paternal obesity (BMI >24) significantly influenced the risk of obesity in 3 year old Japanese children (n=1281; Takahashi et al 1999). A study of 440 6-18 year olds in the United Arab Emirates in 1992-93 showed that a family history of obesity was a significant factor for the development of obesity (Moussa et al 1994). Cross-sectional data of 210 3-16 year old Chinese children and adolescents (prevalence of obesity was 13.8%) showed that parental BMI was predictive of childhood

obesity (relative weight index >120%). Families with one parent having a BMI >25 were 3.7 times and those with two such parents were 5.0 times more likely to have an obese child (compared to other families; Quek et al 1993). Childhood obesity was more markedly correlated with maternal BMI than with the paternal BMI and all these findings were statistically significant (Quek et al 1993).

Associations between childhood and parental obesity or adiposity could result from genetic or environmental factors, or a combination of both. A few studies have specifically focused on genetic contributions (Sorensen 1995; Franzese et al 1998; Cutting et al 1999; Perusse & Bouchard 1999; Samaras et al 1999; Stunkard et al 1999; Chagnon et al 2000) and suggested genetics as an important factor for childhood obesity. Although many studies worldwide have reported an association between parental and childhood obesity, they have used variable definitions of childhood and/or parental obesity, various study populations, small sample sizes, and often did not adjust parental obesity for confounding factors. Therefore, in the present Chapter, the possible association of parental obesity and childhood obesity at age 7 is considered.

It is worth noting that parental obesity not only affects childhood obesity, but it has also been suggested that it is important for early adult obesity. In the recent study of Stettler et al (2000) it was shown that maternal pre-pregnancy BMI was a risk factor for young adult (aged 18-23 years) obesity in African-Americans (Stettler et al 2000).

7.1.3-d. Social class

Recent studies in the USA have shown a relationship between social class and childhood / adolescent obesity. Children who lived with single mothers, children with non-working parents, and children with non-professional parents were shown to be significantly more

likely to become obese by the 6 year follow-up in US children (Strauss & Knight 1999). In a study of 1166 US white 9-10 year old girls, the odds ratio of being overweight (BMI ≥85th centile) was higher in those with no employed parent / guardian in the household (Patterson et al 1997). In a very recent study on a group of 2389 10-16 year old US children and adolescents, socioeconomic status was shown to be an important factor associated with body weight status (McMurray et al 2000).

The effect of social class on nutrient intake in a cross-sectional survey of 498 children aged 9-12 years from 24 schools in a low-income multiethnic suburb of Montreal (Canada) was studied (Johnson-Down et al 1997). A high prevalence (39.4%) of overweight (BMI >85th centile NHANES II) in the study children was encountered. Dietary fat intake was significantly higher ($P<0.001$) in children from single-parent families and those with mothers born in Canada and intakes of vitamins A and C, iron and float were directly related to income sufficiency (Johnson-Down et al 1997).

Very similar results have been reported by some European studies. For instance, among 7300 10-12 year age French children, having only one parent, and being the only child or the last-born in a two-parent family were risk factors for obesity (≥2SD of weight for height) in 5-10 year old children (Locard & Gemelli 1989). DeSpiegelaere et al (1998a) studied childhood obesity during 1986-1990 in a retrospective cohort of 675 Belgian 3-5 years olds and showed that BMI increase was greater in the lower socioeconomic group, whereas the median was higher in the intermediate group (DeSpiegelaere et al 1998a). In another retrospective cohort study done by the same authors on 2607 12-15 year old girls in five social groups in Belgium, an inverse relation between social status and the prevalence of obesity in girls was seen (DeSpiegelaere et al 1998b). In the Kromeyer-Hauschild et al (1999) study, cross-sectional surveys in 1975, 1985, 1995 on 2002, 1534, and 1901 7-14

year old German children, statistically significant associations between overweight (BMI >90th centile of French reference data) and paternal occupation was found in both sexes (Kromeyer-Hauschild et al 1999).

A relationship between social class and childhood obesity is not only an American / European finding, but also studies in other countries have reported similar results. For example, in 3 year old Japanese children (n=1281), maternal occupation was recognised as a risk factor for childhood obesity (BMI ≥18; Takahashi et al 1999). In the study of Kagamimori et al (1999) on Japanese 3-year-old children, more expanded families and a higher percentage of mothers in full-time employment were seen in obese children (Kagamimori et al 1999). However, a study of 440 6-18 year old children in the United Arab Emirates in 1992-93 showed that social class had no relation to childhood obesity (Moussa et al 1994). Associations between social class and childhood obesity may be different between developed and developing countries.

In a recent cross-sectional study of all state primary schools in the relatively deprived city of Plymouth (UK), 20973 5-14 year old children were studied during 1994-96. There was a significant trend for higher rates of obesity (BMI >98th centile) related to increasing social deprivation (not social class) in both boys and girls (Kinra et al 2000). However, we tested associations between social class and childhood obesity risk using the traditional definition of social class in a British sample, and with the ability to correct for confounding factors.

7.1.3-e. Ethnicity and obesity at age 7

Some American studies have described the effect of ethnicity on childhood obesity risk. In a US cross-sectional study on 232 4-10 year old children from 4 different ethnic groups, there was a significant effect of ethnicity ($p<0.01$) on total energy expenditure (Goran et al 1998b). Ogden et al's (1997) study on 7500 children up to 6 years of age during 1988-1994 (NHANES III national survey) found that Mexican-American children had a higher prevalence of overweight (weight-

for-stature or weight-for-length >95th NCHS centile) than non-Hispanic black and non-Hispanic white children. When a group of 2389 10-16 year old US adolescents were studied, ethnicity was an important factor that could influence body weight status (McMurray et al 2000). African-American children were significantly more likely to become obese by the 6 year follow-up than white children (Strauss & Knight 1999).

Although it is likely that ethnicity is more relevant to the US population, two French studies have found an effect of ethnicity on childhood obesity risk too. Among 7300 10-12 year old French children, having parents of southern European origin was a risk factor for childhood obesity (weight for height ≥2SD; Locard & Gemelli 1989). Southern European origin of the mother was an important factor, which contributed to child obesity (relative risk 1.9) in the Locard et al (1992) French study. In the Duran-Tauleria et al (1995) study of English and Scottish children, ethnicity was not significantly associated with overweight (based on weight for height) but it was with fatness (identified based on sum of 2 skinfolds; Duran-Tauleria et al 1995). However, it was necessary to check the role of ethnicity on childhood obesity with current obesity definition criteria (BMI ≥95th centile) and in a contemporary British cohort with the opportunity to correct potential risk factors for confounding variables.

7.1.3-f. Season of birth

In a British study of 1750 men and women born in Hertfordshire during 1920-1930, it was shown that in men, BMI and the prevalence of obesity (BMI >30) varied as a function of month of birth and was greater among those born in January-June than among those born in July-December (Phillips & Young 2000). The relationship between birth weight and adult obesity was also stronger in those born in the first 6 months of the year or following cold winters than in those born in the last 6 months of the year or following mild winters (Phillips & Young 2000).

No study prior to the date of this study (1998) has tried to investigate the relationship between season / month of birth and the prevalence of childhood obesity because of the lack of a widely accepted simple childhood obesity definition and the fact that a possible effect of season of birth has been reported only recently.

7.1.4. Childhood lifestyle and obesity at age 7

7.1.4-a. Diet

In a sample of 530 7-11 year old Italian children energy intake, nutrient intake and percentages of energy intake from different meals were assessed using diet history. Significant relationships were found between the child's adiposity (assessed by skinfold thickness) and their carbohydrate intake, fat intake, and the proportion of energy taken at dinner (Maffeis et al 2000). In another Italian study on 147 children who were followed-up from birth, 1 and 5 years of age, an early high protein intake was suggested as a factor which might influence the development of adiposity (Scaglioni et al 2000). The study of 440 6-18 year old children in the United Arab Emirates in 1992-93 also showed that a child's diet was a significant factor for the development of obesity (Moussa et al 1994).

It might be worth noting that few studies have reported a relationship between diet and prevalence of childhood obesity and that none of them were British. The lack of a relationship might be partly due to limitations of the dietary intake measurement methods used in the studies (Robson & Livingstone 2000) and/or because of the lack of a robust childhood obesity definition (as already described). For example, in a cross-sectional survey of 498 children aged 9-12 years from 24 schools in low-income multiethnic neighbourhoods in Montreal (Canada), 39.4% were overweight (>85th percentile NHANES II). Dietary intake was systematically underreported among overweight children, i.e. their reported intakes did not meet calculated energy needs (Johnson-Down et al 1997). However, having

access to a British longitudinal cohort study, with a good dietary intake methodology, and the new childhood obesity definition, we were stimulated to check the association between early childhood macronutrient intake and childhood obesity risk at age 7.

7.1.4-b. Television viewing and time spent in car and obesity at age 7

The relation between hours of television viewing and the prevalence of overweight (BMI >85th centile) in 746 10-15 year old US children in 1990 was examined and a strong relationship between the prevalence of overweight in 1990 and hours of television viewing was observed (Gortmaker et al 1996). The odd ratios of being overweight were 4.6 times higher (adjusted odds ratio was 5.3 times higher) for more than 5 hours daily watching TV in US youth than watching 0-2 hours (Gortmaker et al 1996). In 6671 12-17 year old adolescents of the third National Health Examination Survey (NHES III) in the USA, the prevalence of obesity increased by 2% for each additional hour of television viewed. The associations persisted even when controlling for prior obesity, region, season, population density, race, socioeconomic class, and some other family variables (Dietz & Gortmaker 1985). In a randomised controlled trial on 192 school age (mean age 8.9 years) children in San Jose (California) during 1996-97, children received an 18-lesson, 6 month classroom curriculum to reduce television, videotape, and video game use. Compared with controls, children in the intervention group had statistically significant relative decreases in BMI (Robinson 1999). This provides additional evidence that TV viewing and childhood obesity risk are related.

Despite this, there are some other US studies which do not show a relationship between television viewing and obesity. A cohort study of 971 school age (mean age 12.4 years) children with follow-up assessments at 7, 14, and 24 months after baseline was conducted in California. Among adolescent girls, the time spent watching television had only a weak

association with adiposity (age adjusted BMI; Robinson et al 1993). For 10-16 year US females, neither videos nor exercise habits appeared to be related to the risk of being overweight (McMurray et al 2000). In a study on 712 9-16 years old children from low- and middle-income towns in the Mexico City area, time spent in physical activity and TV viewing was self-reported. Results showed that physical activity and television viewing, but not VCR and/or video games use, were related to obesity prevalence (Hernandez et al 1999). These inconsistencies might be related to difficulties in measuring TV viewing / sedentary behaviour.

Excessive television viewing (parental report) was an important factor which contributed to child obesity (relative risk 2.1) in a French study of school age children (Locard et al 1992). In a prospective observational study on 112 pre-pubertal (age: 8.6 $\pm$ 1.0 y) Italian children initiated in 1992 and followed-up in 1996, obesity was defined as relative BMI >120%, (relative BMI = BMI / BMI at 50th centile for age and gender, multiple by 100) and it was shown that TV viewing (self-reported) was an important factor for childhood obesity (Maffeis et al 1998). Limited playtime outdoors (1 hour or less) significantly influenced the development of obesity (BMI $\geq$18) in 3 year old Japanese children (n=1281; Takahashi et al 1999). Lower physical activity in obese children was seen in Kagamimori et al's (1999) study of Japanese 3-year-old children (Kagamimori et al 1999). A sample of obese children (BMI $\geq$18; n=427) and control children (n=854) from the Toyama study at health checkups for 3 year old Japanese children was chosen. The main risk factor for childhood obesity in the Takahashi et al (1999) study was limited playtime outdoors (1 hour or less). A study of 440 6-18 year old children in the United Arab Emirates in 1992-93 showed that childhood low physical activity was a significant factor for the development of obesity (Moussa et al 1994).

As previously mentioned, due to the lack of a widely accepted criterion for the definition of childhood obesity, no study prior to the date of the present one was available for British children, and there were no British studies on TV viewing and the risk of childhood obesity available.

7.1.4-c. Sugar-sweetened drink consumption

In a recent study, a group of 548 school age children (mean age 11.7 years) in Massachusetts (USA) were studied prospectively for 19 months from October 1995 to May 1997. Consumption of sugar-sweetened drinks was independently associated with increasing BMI. For each additional serving of sugar-sweetened drink consumed, both BMI (mean increase = 0.24) and frequency of obesity (odds ratio 1.60) increased after adjustment for anthropometric, demographic, dietary, and lifestyle variables (Ludwig et al 2001).

In a study on 168 US children (94 2 year old children and 74 5 year old children), a high percentage (42%) of children who consumed 12 fl oz/day of juice had short stature (height <20th centile) while only 14% of children who drank less than 12 fl oz/day had short stature. Obesity was more common among children drinking 12 fl oz/day of juice compared to the others such that 32% vs. 9% had a BMI >90th centile (Dennison et al 1997). After adjustment for maternal height, child age, child sex, and child age-sex interaction, children consuming 12 fl oz/day of juice, compared with others, were shorter (86.5 vs. 89.3 cm and 106.5 vs. 111.2 cm for the 2 year old and 5 year old children, respectively) and more overweight (mean BMI 17.2 vs. 16.3; Dennison et al 1997). There may therefore be associations between drinking habits and childhood obesity risk which are worth exploring in a large and contemporary British sample. Since drinking may encourage the eating of snacks and might be an indicator of parental feeding behaviour, we decided to test if there was any relationship between drinking fizzy drinks (whether diet types or not) and childhood obesity.

7.1.4-d. Sleeping and childhood obesity at age 7

Short sleep duration was a factor which contributed to child obesity (relative risk 4.9) in a study of French children (Locard et al 1992). The relationship between obesity and short sleep duration persisted independently of television viewing (Locard et al 1992). Sleeplessness was a predictor for overweight (BMI 85th - 94th centile) at age 5 years in 4062 Australian children (O'Callaghan et al 1997). Higher prevalence of reduced sleeping hours (<10 hours) in obese children was seen in a study of Japanese 3-year-old children (Kagamimori et al 1999). Therefore, a lack of sufficient sleep could be considered as a possible risk factor for childhood obesity, though the mechanisms underlying such an association are unclear.

7.2. Aims of this study

The aims of the present study were to test the relationships / associations between potential obesity risk factors and childhood obesity at age 7 years. The risk factors which were tested consisted of:

-Intra-uterine variables (gestational age, birth weight, and catch-up growth)

-Infant feeding factors (breast-feeding and timing of introducing solids)

-Demographics as well as other variables (including sex, parental education, parental obesity, social class, ethnicity, and season of birth)

-Childhood lifestyle (including diet, television viewing and inactivity, consumption of fizzy drinks and sleeping pattern).

7.3. Subjects and methods

7.3.1. Subjects

ALSPAC is a prospective cohort study of the determinants of health during childhood in the Bristol-Avon area of the UK (described in detail in Chapter 2). The study population comes from 14893 parents and their infants born in 1991-1992. During the first trimester of pregnancy, when mothers were contacted, and agreed to participate in the study, they completed questionnaires about their pregnancy. When their babies were born, they completed more questionnaires at different stages as described in Chapter 2. The Children in Focus (CIF) cohort of the ALSPAC study represented a 10% sub-sample randomly selected from the main ALSPAC study sample, born in the last 6 month of the cohort (more detailed information is described in Chapter 2). This sub-sample was representative of the main sample but different from British children in terms of social class (details can be found in Chapter 2). The total number of this sub-sample was 1409 children at age 4 months. Weights and heights were available for 888 at age 1 year, 829 at age 3 years, 827 at age 5 years, and 954 at age 7 years and were used to calculate BMI. From the main ALSPAC cohort, 7766 children (55.5%) provided complete weight and height data in the follow-up study at age 90 months (7 years old in 1999/2000). The data for the above mentioned children and the CIF sub-sample at age 7 years were analysed and the results are presented in this Chapter.

7.3.2. Outcome measure: measure of obesity in the present study

We defined obesity as BMI $\geq 95^{th}$ percentile relative to UK 1990 reference data (Cole et al 1995). This definition is widely used (Gortmaker et al 1993; Robinson 1993; Himes & Dietz 1994; Troiano et al 1995; Freedman et al 1997; Whitaker et al 1997; Barlow & Dietz 1998; Slyper 1998; Troiano & Flegal 1998; Troiano & Flegal 1999; Zephier et al 1999), has some biological and clinical significance (Troiano & Flegal 1998; Freedman et al 1999; Chapter 3), and moderately high sensitivity with high specificity (Chapter 5).

7.3.3. Intra-uterine factors

7.3.3-a. Gestational age

Gestational age (weeks of pregnancy), was obtained by ALSPAC staff from medical records. If there was any suggestion that the gestation was pre-term (i.e. less than 37 weeks) or post-term (i.e. 43 weeks or more) then medical records were reviewed by a specialist to check that the gestation was correct. Medical records were reviewed not only for pregnancies that appeared pre-term or post-term, but also if the clinically recorded gestation based on ultrasound or the clinician's impression and/or paediatric estimate was ≤37 weeks or ≥43 weeks. The following formula was used to calculate the gestational age: last menstrual period (LMP) - expected date of pregnancy.

7.3.3-b. Catch-up growth 0 to 2 years

Using the UK 1990 reference data (Cole et al 1995) the weight and height standard deviation scores (SDS) for CIF children (n=806) were calculated at birth and age 2 years. Changes in SDS between birth and 2 years were calculated for weight and length (scores at 2 years minus scores at birth) and were adjusted for gestational age and gender (Ong et al 2000). A gain in SDS for weight between 0 and 2 years which was greater than 0.67 SDS was defined as an indication of clinically significant catch-up growth (Ong et al 2000) since 0.67 SDS represent the width of each percentile band on standard growth charts which are 2nd to 9th centile, 9th to 25th, 25th to 50th, and so on (Freeman et al 1995).

7.3.3-c. Birth weight

Birth weight (g) was based on hospital records. The birth weight of 93 out of 7766 study subjects was not recorded. Defining children with a birth weight less than 2500 grams as low birth weight (LBW) is an accepted and widely used classification. Most studies have

used >3500 gram birth weight as the cut-off for the definition of heavy weight infants. However, using this cut-off defined a high percentage (45.1%) of the study children as heavy. Therefore, we used birth weight quartiles as the basis for definition of heavy birth weight. Thus, study children were classified into 3 categories: LBW (less than 2500 g at birth), normal birth weight (2500 - 3799 g), and heavy birth weight (>3800 g, equivalent to the highest quartile).

7.3.4. Infant feeding / weaning

7.3.4-a. Ever breast-fed or not

The mother was asked by questionnaire, when the child was 6 months old, about breast-feeding. Three options were given: 'Yes still', 'Yes, but stopped', and 'Never'. For part of the analysis (adjustment for other factors), the 'Yes still' and 'Yes, but stopped' answers were combined.

7.3.4-b. Timing of the introduction of solids

The mother was asked by questionnaire when the child was 6 months old to record the age (in weeks) of the child when a list of foods was introduced. These foods were: plain baby rice, flavoured baby rice, other cereals, sweetened rusks, plain rusks, bread or toast, biscuits, home cooked egg, ready prepared meat, ready prepared fish, ready prepared potatoes, ready prepared vegetable, ready prepared fruit pudding, ready prepared milk pudding, raw fruit, raw vegetable, chocolate. The actual age of the introduction of the first solid food was based on checking the results of all these questions to find the food introduced at the youngest age for each child. This age was identified as the age of the introduction of solids and children were classified into 2 groups: before 12 weeks of age / after 12 weeks of age.

7.3.5. Demographics / other variables

7.3.5-a. Parental obesity

Mothers at 12 weeks of pregnancy were requested (by questionnaire) to provide information about their pre-pregnancy height and weight (self-report). Partners also received a questionnaire at around 12 weeks of pregnancy asking about their height and weight. BMI was calculated and maternal and paternal obesity were defined as BMI >30 kg/m^2. Thus, 4 groups were identified: both parents obese; mother obese; father obese; no parents obese (both father and mother had self-reported a BMI ≤30).

7.3.5-b. Education

The mother was asked at week 32 of pregnancy about the education of both herself and her partner. The results were classified as 'None/CSE; vocational; O level; A level; and Degree' options.

7.3.5-c. Social class

The mother was asked about the occupation of both herself and her partner at week 32 of pregnancy and using OPCS (Office of Population Censuses and Surveys) classification of occupations (OPCS 1990), the social class of the family was identified. The 6 social class groups were categorised as class I, II, III non-manual, III manual, IV and V.

7.3.5-d. Ethnicity

The question about ethnic origin (asked at 32 weeks ante-natal) had 9 possible answers including 'white, black Caribbean, black African, other black, Indian, Pakistani, Bangladeshi, Chinese, and other'. However, since the ethnic background of 7055 out of 7353 available answers was white, the study children were classified as either white or non-white for the purposes of this analysis.

7.3.5-e. Season of birth

The month of birth for all the 7766 total study cases was available. Children were grouped according to season: spring (April to June), summer (July to September), autumn (October to December) and winter (January to March), using the seasonal categorisation previously reported by Phillips & Young (2000).

7.3.6. Childhood lifestyle

7.3.6-a. Dietary data

Dietary data (% energy from fat, protein and carbohydrate) were available at ages 8, 18, and 43 months for CIF children. Diet was assessed using a three-day unweighed (household measures) dietary record (n=773, n=689, and n=626 respectively as described in Chapter 2). A week before the 8, 18, and 43 months CIF clinic appointment, three one-day dietary diaries were sent to the carer of the child with an instruction leaflet. The carer was instructed to complete the diaries by recording everything the child ate and drank in household measures for two weekdays and one weekend day (not necessarily consecutive), and to bring the completed diaries with them to the clinic. At the clinic the carer was interviewed by a trained assistant to check for completeness and to clarify any uncertainties in the diaries, such as cooking methods or types of bread and milk used. Those carers who had not completed some or all of the diaries were asked if they would do so and return the diaries by post. The diary consisted of an eight-sided booklet. The completed dietary records were coded using the computer program of 'DIDO' (Diet In, Data Out) which is designed for direct entry of dietary records. The DIDO program was used in conjunction with a nutrient analysis program, 'BRIGADE' (Bristol General Analysis of Dietary Experiments) to generate the nutrient analysis for each food the child ate.

7.3.6-b. TV viewing and time spent in car during weekdays

TV viewing and time spent in car during weekdays at age 38 months was asked by parent-completed questionnaire. The carers were requested to indicate how much time on average the child spent in car / watching television on most weekdays. The answer to these questions was chosen from one of the 'not at all, less than 1 hour, 1-2 hours a day, or more than 2 hours a day' options. In this study analyses, the 'not at all' and '<1 hour' options were combined.

7.3.6-c. Consumption of fizzy drinks

At 38 months the child's parent or guardian was asked (by questionnaire) about how many times a week the child drinks either cola or other fizzy drinks (e.g. lemonade). The provided answers, which the carer could choose were 'never or rarely, once in 2 weeks, 1 to 3 times a week, 4 to 7 times a week, and more than once a day'. Due to limitations in the method of collection of data on drink consumption (as described in the limitations Section of this Chapter), it was not possible to accurately subdivide fizzy drinks into sweetened and diet drinks, therefore no distinction has been made between them in this analysis.

7.3.6-d. Sleeping

Previous studies have focused on childhood sleeplessness at 6 months of age (O'Callaghan et al 1997) and reduced sleeping hours at age 3 years (Kagamimori et al 1999). In the present study, the duration of sleeping was calculated (in minutes) based on the report by parents of the mean time (hours and minutes) of going to bed in the evening, and mean time of waking up in the morning in questionnaires obtained at 30 and 42 months. Another concern about sleeping was the regularity of sleep since it reflects the sleeplessness of the child. Therefore, parents were requested to indicate if their child had a regular sleeping pattern or not. The question was: 'does your child have a regular sleeping routine ? Yes / No'. In the present study, the association between sleeping and childhood obesity was investigated using two different variables: the reported duration of sleep, and the reported regularity of sleep.

7.3.7. Statistical methods

The aim of the analysis for this study was to test the association between possible childhood obesity risk factors and measured obesity at age 7 years. In the present study, data were analysed at three levels.

Level one: differences between categories distinguished on the basis of continuous variables (such as macronutrient intakes) were tested for significance using one way ANOVA with follow-up multiple comparisons incorporating Bonferroni corrections as appropriate. After doing Kolmogorov-Smirnov tests of 'normality', the association between continuous variables with childhood obesity were analysed using student's t-tests. Associations between categorical variables and childhood obesity were tested for significance using Chi-squared tests and 2-probablity Z-tests. Binary logistic regression models were used to analyze the relationship between childhood obesity (outcome) and a number of intra-uterine, infant feeding / weaning, demographic and childhood lifestyle variables. From the 19 investigated variables, 4 of them including gestational age, gender, ethnicity, and season of birth showed no significant association with childhood obesity and hence odds ratios (and 95% confidence intervals) were calculated for the associations of each of the remaining 15 variables.

Level two: from the above mentioned 15 variables, after excluding catch-up growth and macronutrient intakes of which data were available only for CIF children, multi-variate analysis were conducted in the first instance with adjustment for association for maternal / paternal social class. Of these 13 variables, the *timing of the introduction of solids* showed no association with childhood obesity after adjustment for *maternal social class* in this stage and so both the timing of the introduction of solids, and the paternal social class were not entered in the next level of analysis.

Level three: of the remaining 12 variables, paternal social class was excluded and only maternal social class was entered in the analysis, and final adjustment association for all 11 remaining significant factors was conducted. These factors included birth weight; breast-feeding up to and beyond age 6 months; maternal education; paternal education; maternal obesity; paternal obesity; maternal social class; TV viewing; time spent in car; consumption of fizzy drinks; and regularity of sleep at age 42 months.

7.4. Results

7.4.1. Sample size and dropout

The original sample size in ALSPAC study (Children of the 90s) as described in Chapter 2 was 14893 children born in 1991-92. Of these, 7782 (55.6%) still participated in the study at age 7 years and weight and height was measured for 7766 of them (3949 boys and 3817 girls). Data for all variables were not available for all the 7766 participants and the amount of missing data for each variable is presented below. The Children in Focus (CIF) group were followed from birth and were measured at regular ages up to 5 years and then at 7 years. We studied the BMI values for 1409 of them from age 4 months until age 7 years when BMI for 954 (519 boys and 435 girls) CIF children was available.

7.4.2. Obesity prevalence in the sample

As Table 7.1 results indicate, 631 out of 7766 7 year old children (8.1%) were identified as obese using the BMI ≥95th percentile criterion. Fifty-one percent of the children (3885/7638) were boys and 49% girls. Obesity prevalence was slightly higher in boys (8.6%) than girls (7.6%) but this difference was not statistically significant.

Table 7.1. Prevalence of childhood obesity (BMI ≥95th centile) in 7 year old boys and girls.

Sex	Obese n (%)	Non-obese n (%)	Total n (%)
Boys	341 (8.6)	3608 (91.4)	3949 (50.8)
Girls	290 (7.6)	3527 (92.4)	3817 (49.2)
Total	631 (8.1)	7135 (91.9)	7766 (100.0)

No significant difference was found between obesity prevalence between boys and girls.

7.4.3. Intra-uterine factors and prevalence of childhood obesity at age 7

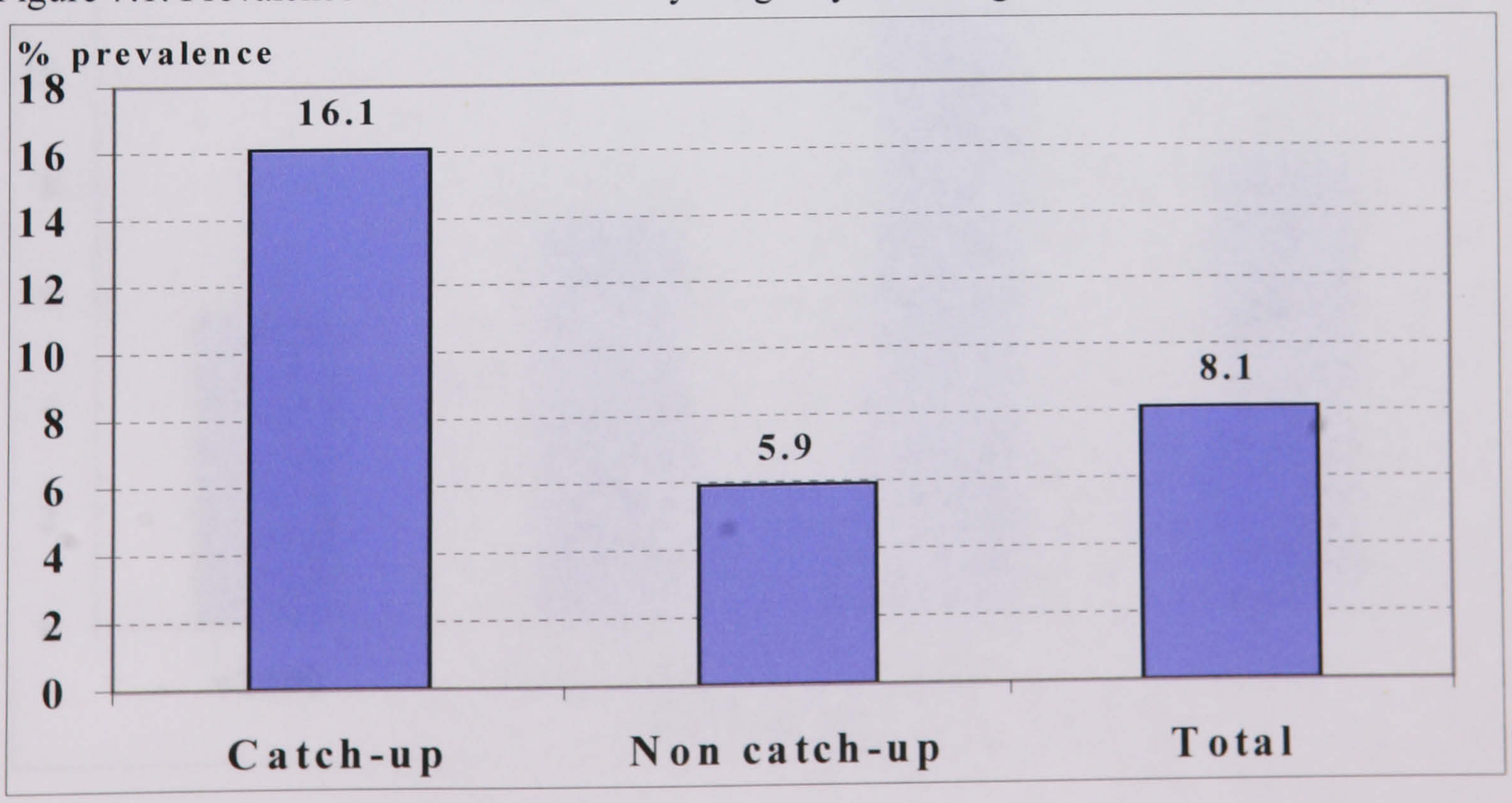
7.4.3-a. Gestational age

Mean gestational age in obese subjects (n=631) was 39.5 weeks and it was 39.4 weeks in non-obese 7 year old children (n=7135); no significant difference was observed.

7.4.3-b. Catch-up growth

From the 954 (519 boys and 435 girls) CIF children measured at 7 years, growth between ages 0 to 2 years was available for 806 (439 boys and 367 girls) subjects. Twenty seven percent (217/806) of the children showed catch-up growth with no significant difference in prevalence of catch-up between boys and girls. As shown in Figure 7.1, a total of 16.1% of the catch-up group and 5.9% of the rest were obese at age 7 and this difference was statistically significant ($p<0.001$). The odds ratio of obesity (level 1 of analysis) in catch-up group (compared to other children) was 3.04 (95% confidence interval: 1.80 - 5.15). Due to small sample size in the logistic regression analysis, adjusting for other childhood obesity risk factors (levels 2-3 of analysis) was not possible. No significant difference between boys and girls in the association of childhood obesity risk and catch-up growth was found.

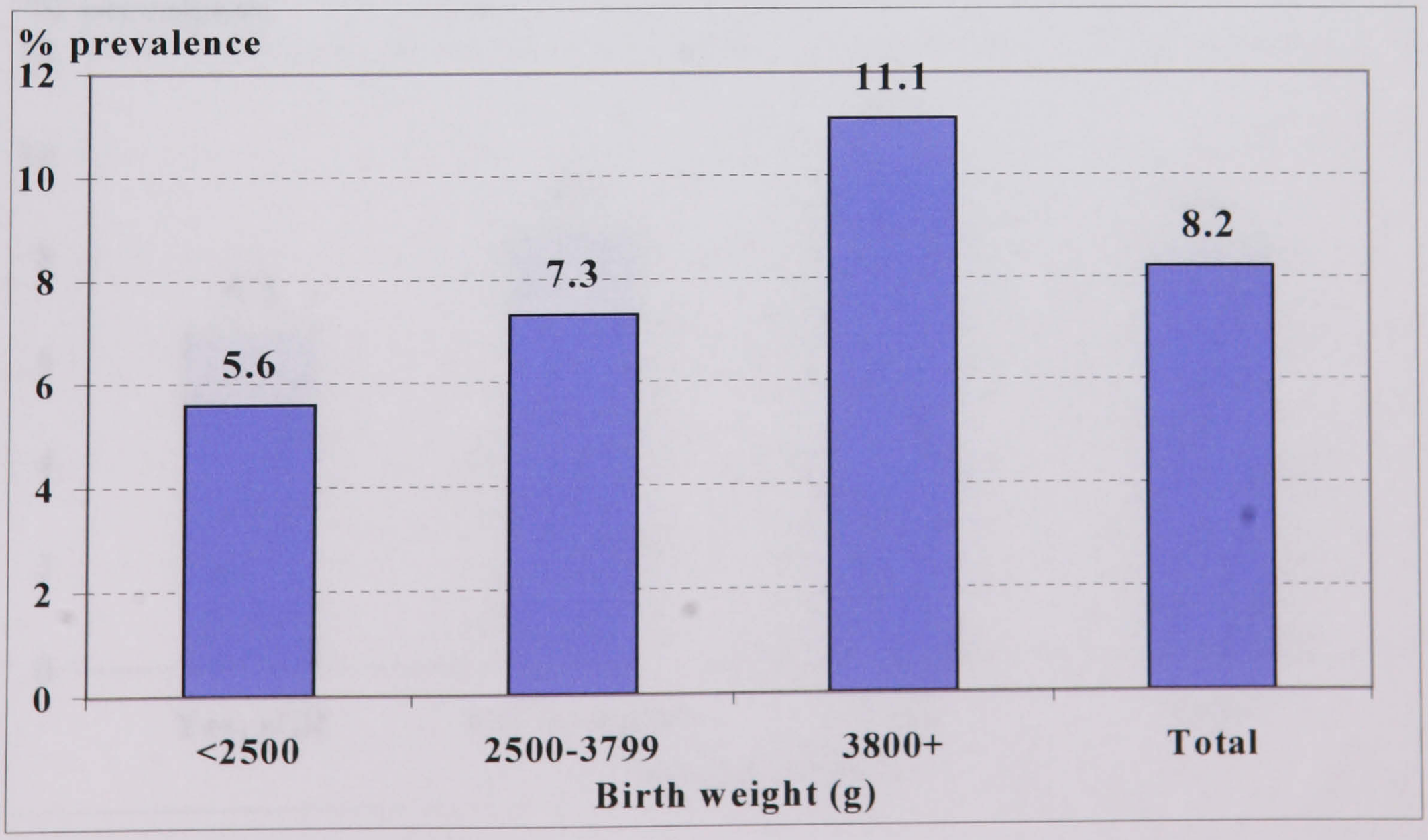
Figure 7.1. Prevalence of childhood obesity at age 7 years and growth between 0-2 years.



7.4.3-c. Birth weight

From the total sample of 7766, birth weight information for 7673 (98.8%) was available. Only 374 babies (4.9%) were low birth weight (under 2500g) and 1824 (23.8%) were in the top quartile of birth weight distribution (over 3800g). As Figure 7.2 shows, the prevalence of childhood obesity at age 7 years in the low birth weight group was 5.6%, in the next lower quartile of birth weight children it was 7.3%, in top quartile (3800g and over) it was 11.1%. The differences between the 3800g and over group and both the first two groups were statistically significant ($p<0.001$). The mean birth weight of the obese children was 3558g (SD=566) and this was significantly ($p<0.05$) higher than that of the non-obese children (mean=3405g SD=556). Birth weight and childhood BMI standard deviation scores at age 7 years were significantly correlated ($r=0.20$, $p<0.001$). The odds ratio of childhood obesity (level 1 of analysis) at age 7 in the children who were in the top quartile at birth (≥3800 grams birth weight) was 1.58 (95% CI: 1.32-1.89) compared to 2500-3799 grams birth weight children. After adjustment for maternal and paternal social class, the association between birth weight and childhood obesity was still significant (level 2 of analysis; Table 7.5).

Figure 7.2. Prevalence of childhood obesity at age 7 years and birth weight.

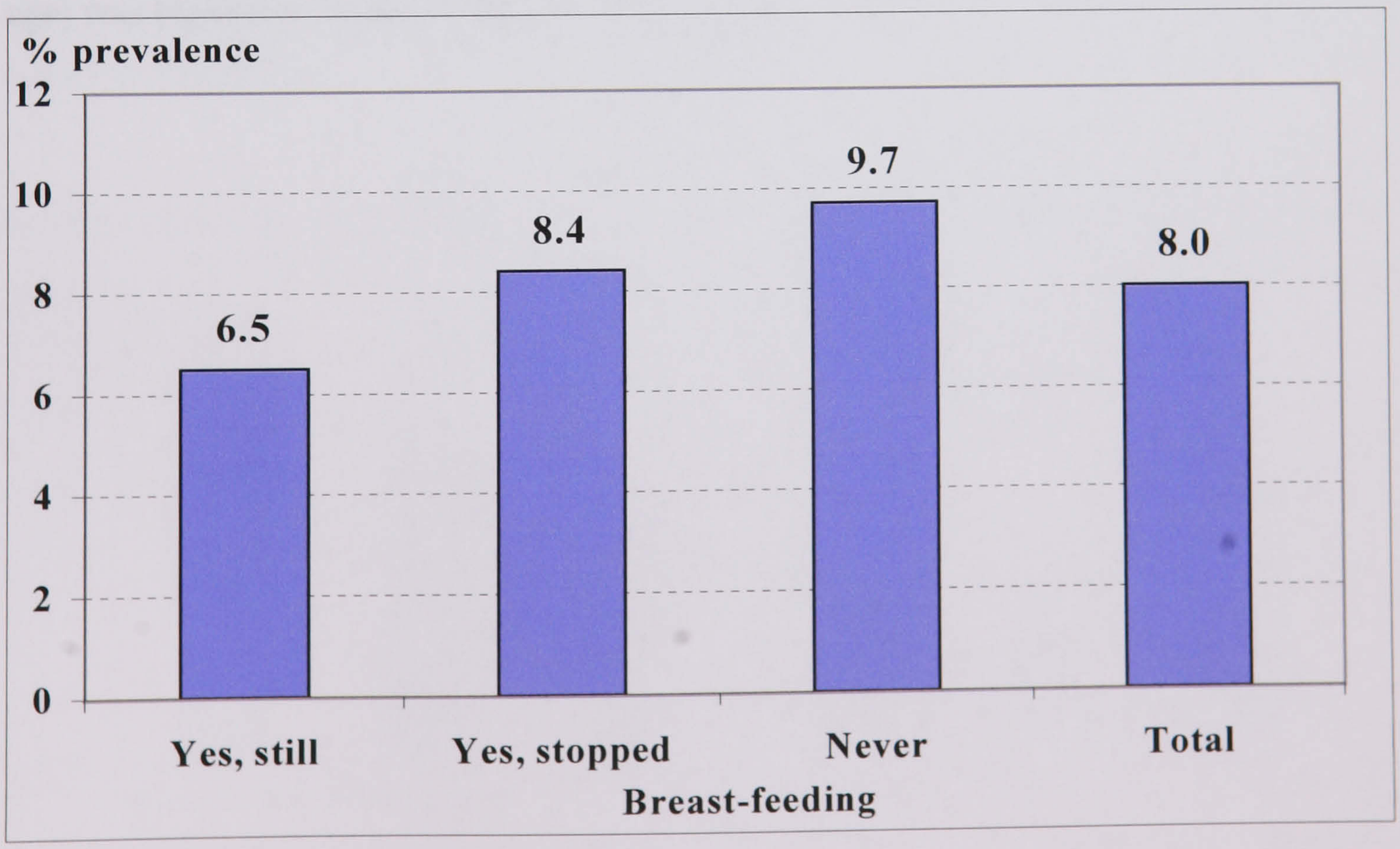


7.4.4. Infant feeding / weaning and obesity prevalence at age 7

7.4.4-a. Breast-feeding

From 7766 7 year olds, infant feeding practice data were available for 7205 (92.8%; 3665 boys and 3540 girls). Of them, 33.6% of infants continued breast-feeding beyond 6 months of age, 47.5% were breast-fed for less than 6 months and 18.9% were never breast-fed. As can be seen from Figure 7.3, the lowest childhood obesity prevalence (6.5%) at age 7 years was seen in those who continued breast-feeding beyond age 6 months (the time of questioning) and the highest prevalence (9.7%) in those who were never breast-fed and these differences were statistically significant (p=0.002). Compared to the children who were breast-fed beyond 6 months, the odds ratio of childhood obesity (level 1 of analysis) in those children who were breast-fed less than 6 months was 1.32 (95% CI: 1.08-1.62) and in the never breast-fed group 1.55 (95% CI: 1.22 - 1.97). After adjustment for the paternal and maternal social class (level 2 of analysis), the association between breast-feeding and childhood obesity was still significant (Table 7.5).

Figure 7.3. Prevalence of childhood obesity at age 7 years and weather breast-fed or not.



7.4.4-b. Timing of the introduction of solids

The age of starting solids was available for 6291 (81.0%) of the sample (3187 boys and 3104 girls). Of these, 73.3% had received non-milk feeding by 12 weeks of age and the rest (26.7%) had started solids from or after age 12 weeks. The prevalence of childhood obesity (at age 7 years) was slightly higher in those who started solids earlier (8.7%) than in others (6.2%) and the difference was statistically significant ($p=0.002$; Table A.2 Appendix). The difference between boys and girls was not statistically significant. The odds ratio for childhood obesity (level 1 of analysis) in children whom solids were introduced before 12 weeks was 1.43 (95% CI: 1.14-1.79) compared to those who were weaned after 12 weeks of age. After adjustment for paternal social class (level 2 of analysis), the association was still significant ($p<0.05$) but after adjustment for maternal social class, no significant association was seen. The timing of the introduction of solids was significantly associated with maternal social class. The percentage of children who started solids after 12 weeks (recommended age) was highest in maternal social classes I and II.

7.4.5. Demographics / other variables associated with childhood obesity prevalence

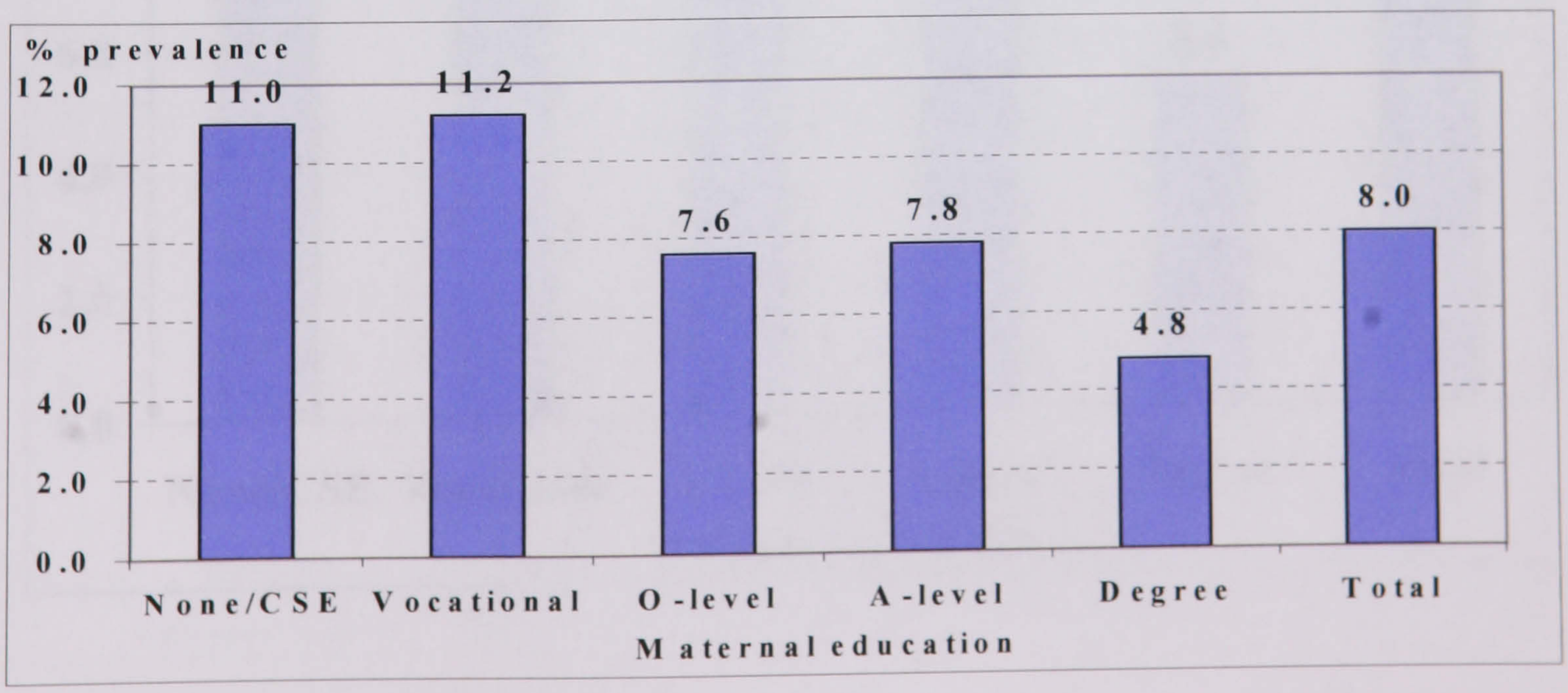
7.4.5-a. Gender

As discussed earlier, no statistically significant difference (p=0.10) was found in the prevalence of childhood obesity (at age 7 years) between the sexes. The odds ratio for childhood obesity in boys compared to girls was 1.15 (95% CI 0.98-1.35).

7.4.5-b. Maternal education

At 32 weeks gestation, 7468 mothers (96.2%) from the total of 7766 possible mothers reported their highest education qualification. From them, 13.2% of the mothers had No or CSE qualifications, 8.7%, 35.4%, 26.6%, and 16.1% of the mothers had a vocational, O-level, A-level, or Degree qualification respectively. As can be seen from Figure 7.4, prevalence of childhood obesity (at age 7 years) was associated with maternal education: children of lower educated mothers had higher prevalence of childhood obesity and differences were statistically significant (p<0.001). The odds ratios for childhood obesity (level 1 of analysis) in children of None/CSE, vocational, O level, and A level educated mothers (compared to children of Degree level educated mothers) were 2.48 (95% CI: 1.78-3.46), 2.53 (95% CI: 1.76-3.62), 1.64 (95% CI: 1.22-2.23), and 1.68 (95% CI: 1.23-2.30) respectively. After adjustment for paternal and maternal social class (level 2 of analysis), the association between maternal education and childhood obesity remained significant (Table 7.5).

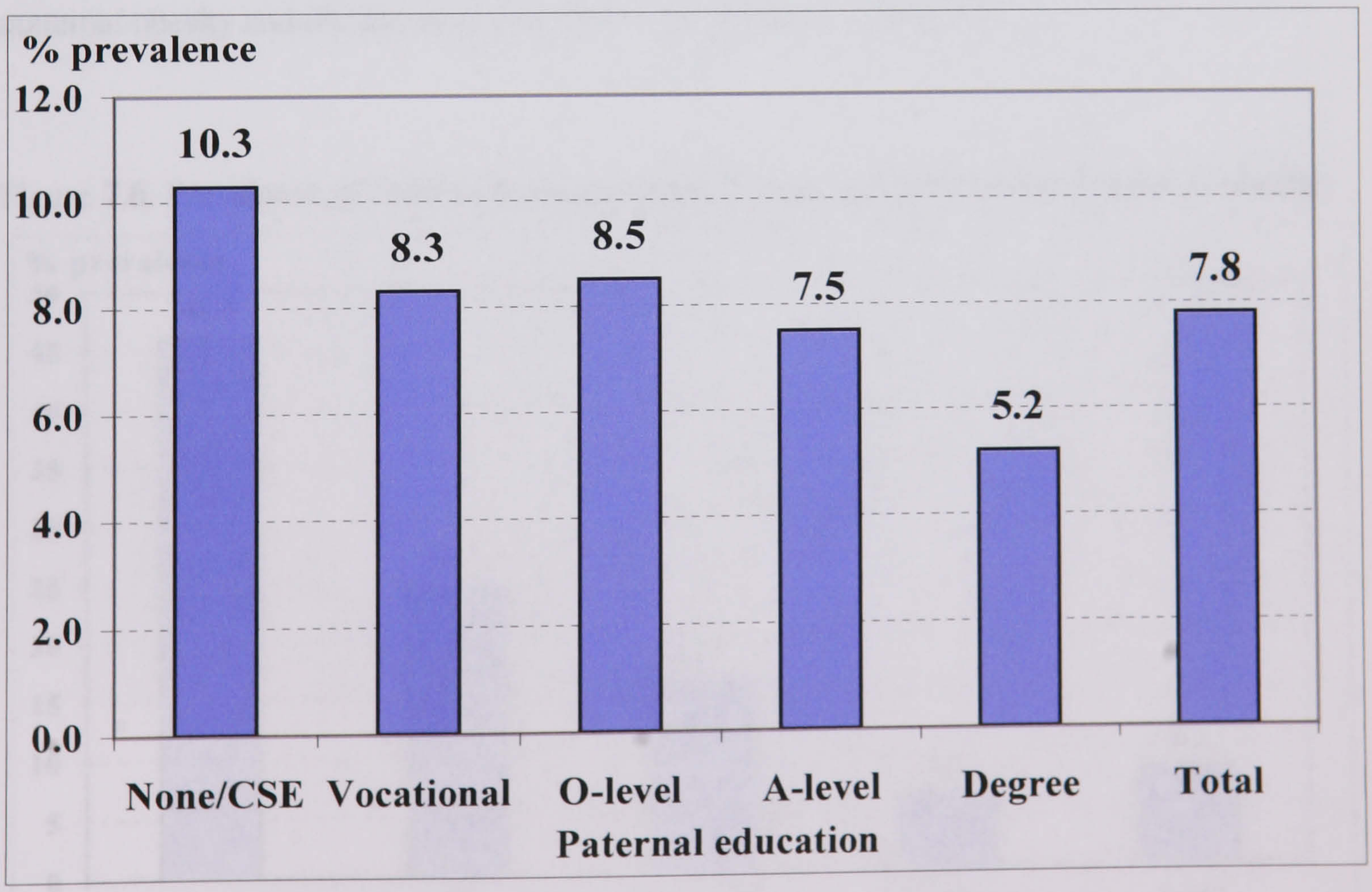
Figure 7.4. Prevalence of childhood obesity at 7 years and highest maternal education qualification.



7.4.5-c. Paternal education

Information / data on their highest educational qualification was available for 7263 fathers (93.5%) from the 7766 fathers in the present study. From these, 19.8% had No or CSE, 7.9%, 22.4%, 27.9%, and 21.9% of the fathers had a vocational qualification, O-level, A-level, or Degree qualification. As can be seen from Figure 7.5, prevalence of childhood obesity (at age 7 years) was related to paternal education: children of lower educated fathers had higher prevalence of childhood obesity and differences were statistically significant ($p<0.001$). The odds ratios for childhood obesity (level 1 of analysis) in children of None/CSE, vocational, O level, and A level educated fathers (compared to children of Degree level educated fathers) were 2.09 (95% CI: 1.58-2.76), 1.65 (95% CI: 1.14-2.39), 1.68 (95% CI: 1.27-2.23), and 1.48 (95% CI: 1.13-1.95) respectively. After adjustment for paternal and maternal social class (level 2 of analysis), the association between paternal education and childhood obesity was still significant (Table 7.5).

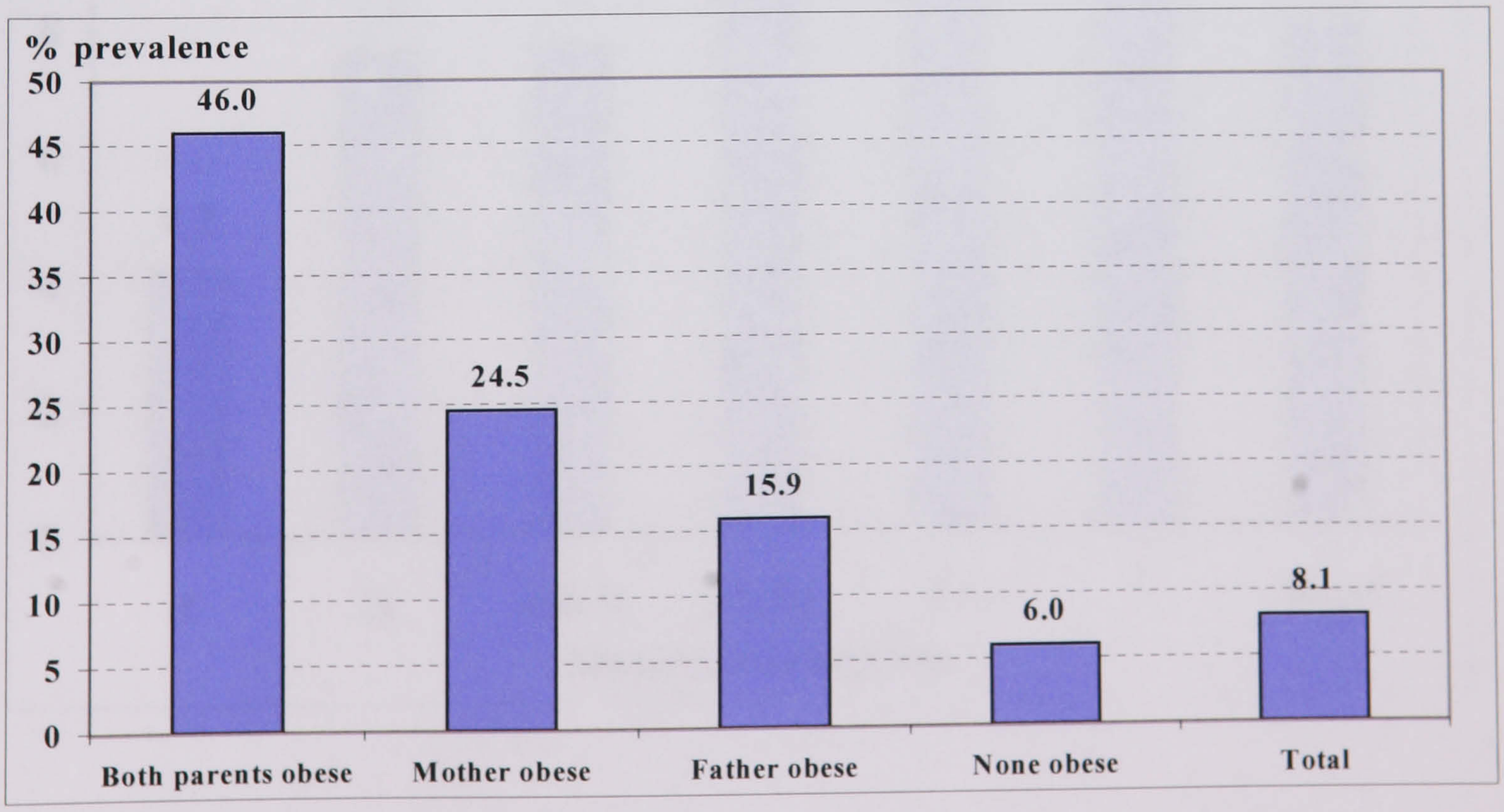
Figure 7.5. Prevalence of childhood obesity at 7 years and highest paternal education qualification.



7.4.5-d. Parental obesity

From the total of 7766 children, the BMI values for 5404 fathers (69.6%) and 7004 mothers (90.2%) were available. A total of 7.3% of fathers and 5.2% of mothers were identified as obese using BMI >30 kg/m^2 criterion. The prevalence of childhood obesity (at age 7) in the children of obese fathers was 20.9% while it was 6.6% for the children of non-obese fathers. The prevalence of childhood obesity in the children of obese mothers was 28.3% while it was 6.9% for the children of non-obese mothers. As can be seen in Figure 7.6, the prevalence of childhood obesity in the children with both parents obese (n=50) was 46.0% while the prevalence was 6.0% in the children with no parents obese. All the associations shown in Figures 7.5 and 7.6 were statistically significant. There was no significant difference between childhood obesity in boys and girls in terms of the association between parental obesity and childhood obesity. The odds ratios for obesity (level 1 analysis) in children who had obese fathers was 2.57 (95% CI: 1.88 - 3.51), for those who had obese mothers was 4.40 (95% CI: 3.16 - 6.12), and for those who had both parents obese was 11.54 (95% CI: 6.56-20.28) compared to the children who had no obese parent. After adjustment for paternal and maternal social class (level 2), the association between paternal and maternal obesity and childhood obesity was still significant (Table 7.5).

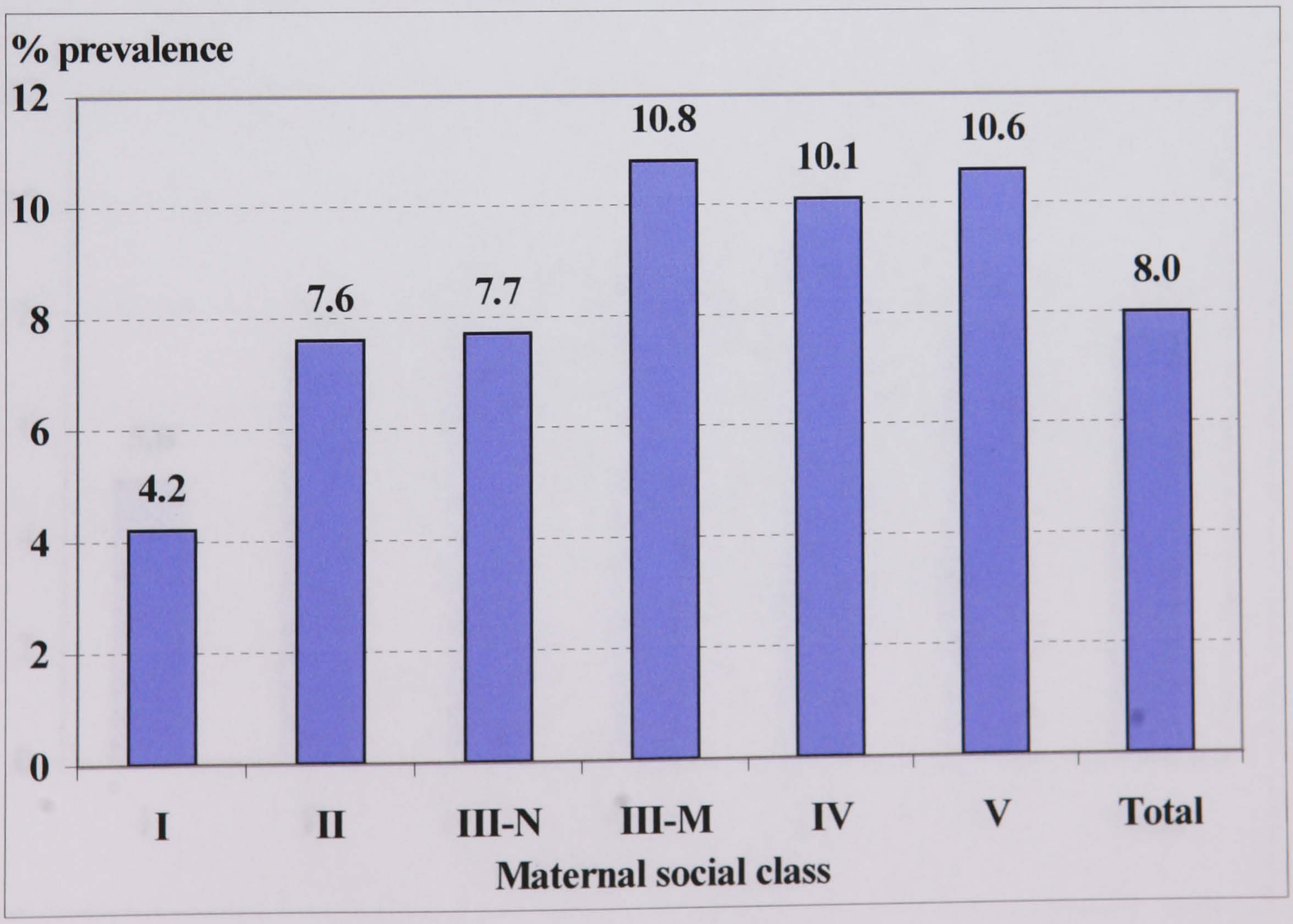
Figure 7.6. Prevalence of childhood obesity at age 7 years and parental pre-pregnancy obesity.



7.4.5-e. Maternal social class

Maternal social class was available for 6058 (78.0%) of 7766 7 year olds. From them, 5.1%, 33.1%, 44.7%, 7.0%, 8.5%, and 1.6% of mothers were in the first, second, third non-manual, third manual, forth, and fifth social classes respectively. The prevalence of childhood obesity at age 7 years was significantly higher in those children of mothers from classes IIIM, IV and V compared to those in the higher social classes of I, II, and IIIN and these differences were statistically significant ($p<0.005$; Figure 7.7). No significant difference in the association between social class and obesity between boys and girls was found. The odds ratios for childhood obesity (level 1 of analysis) in the second to fifth social classes compared to social class I were 1.87 (95% CI: 1.05-3.33), 1.89 (95% CI: 1.07-3.35), 2.74 (95% CI: 1.45-5.16), 2.55 (95% CI: 1.37-4.76), and 2.69 (95% CI: 1.14-6.35) respectively.

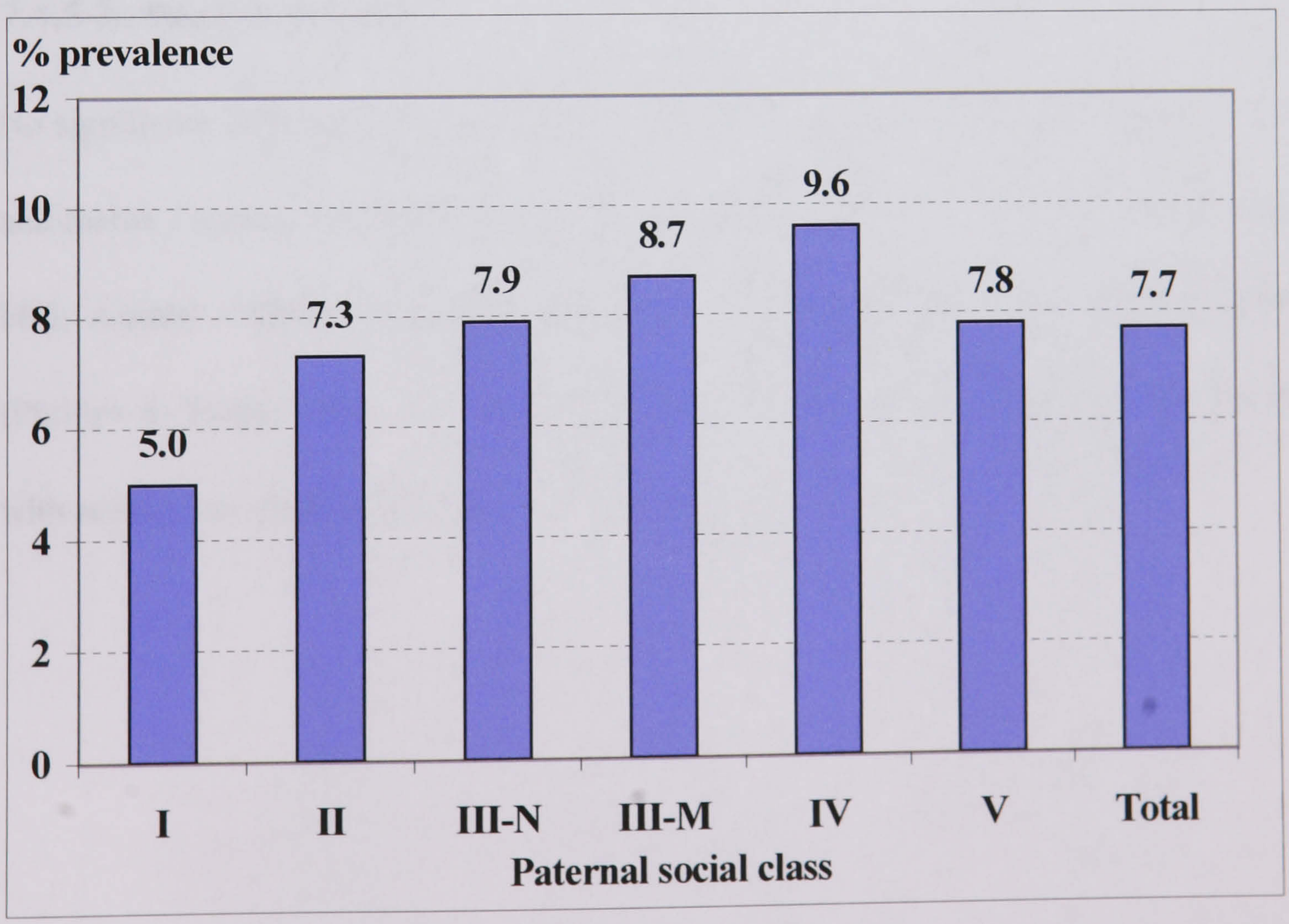
Figure 7.7. Prevalence of childhood obesity at age 7 years and maternal social class.



7.4.5-f. Paternal social class

Paternal social class was available for 6648 (85.6%) of 7766 7 year olds. From them, 13.0%, 35.6%, 12.2%, 29.1%, 7.7%, and 2.5% of fathers were in the first, second, third non-manual, third manual, forth, and fifth social classes respectively. The prevalence of childhood obesity at age 7 years was highest in those children of fathers from social class IV while it was lowest in the children of social class I and all the differences between groups were statistically significant ($p<0.005$; Figure 7.8). No significant difference in the association between social class and obesity between boys and girls was found. The odds ratios for childhood obesity (level 1 of analysis) in social classes II to V compared to social class I were 1.50 (95% CI: 1.06-2.12), 1.62 (95% CI: 1.09-2.41), 1.82 (95% CI: 1.29-2.57), 2.03 (95% CI: 1.32-3.10), and 1.62 (95% CI: 0.85-3.08) respectively.

Figure 7.8. Prevalence of childhood obesity at age 7 years and paternal social class.



7.4.5-g. Ethnicity and obesity at age 7 years

The ethnicity for 7353 (94.7%) of 7766 7 year old children was recorded by parental self-report. A total of 4.1% (298 out of 7353) of study children in whom their ethnic background and BMI percentiles were recorded, were non-white. In total, 31 out of 298 (10.4%) non-white children were identified as obese (using BMI $\geq 95^{th}$ percentile) while 7.8% of white children were identified as obese using the same criteria. The obesity prevalence in the non-white group was higher but not significantly (p=0.11). The odds ratio for childhood obesity (level 1 of analysis) in the non-white ethnic group of children was 1.37 (95% CI: 0.93-2.00). There was no significant difference in the relationship between ethnicity and obesity between boys and girls. Since there was no significant difference found, and since the sample size available for this analysis was small, no further analysis was done.

7.4.5-h. Season of birth

No significant differences were found between childhood obesity prevalence at age 7 years and month / season of birth (Table A.13 Appendix). A variety of different combinations of birth months / different seasons were tried in accordance with the original hypothesis (Phillips & Young 2000). No significant gender difference in childhood obesity associated with season was observed (Table A.13 Appendix).

7.4.6. Childhood lifestyle and obesity at age 7

7.4.6-a. Diet

From the original sample of 1409 CIF children at age 4 months, 1370 were studied up to age 7 years and BMI for a sub-sample of 954 of them was available at age 7 years. The prevalence of childhood obesity (BMI $\geq 95^{th}$ centile) at age 7 years for this sub-sample of children (n=954) was calculated and dietary intake (at age 8 months) for 846 (88.7%) of them was available. Dietary intake was available at age 18 months for 760 (79.7%) of them, and at age 43 months for 687 (72.0%) children. Because of the small sample size, doing the adjustment analysis by logistic regression (levels 2-3 of analysis) was not possible.

7.4.6-b. Macronutrient intakes at age 8 months

There was a significant difference between energy intake at age 8 months between the children who were obese at age 7 years and the non-obese children ($p<0.05$). Obese 7 year old children had significantly lower energy intakes (kJ/kg/day) at 8 months of age than the non-obese children (Table 7.2). No significant differences were found between fat, carbohydrate, and protein intake at age 8 months or the percent of energy from fat, carbohydrate, and protein between obese and non-obese 7 year old children (Table 7.2 and Figure 7.9). No significant gender difference was observed.

Figure 7.9. Nutrient intakes (g/kg/day) at age 8 months and childhood obesity at 7 years.

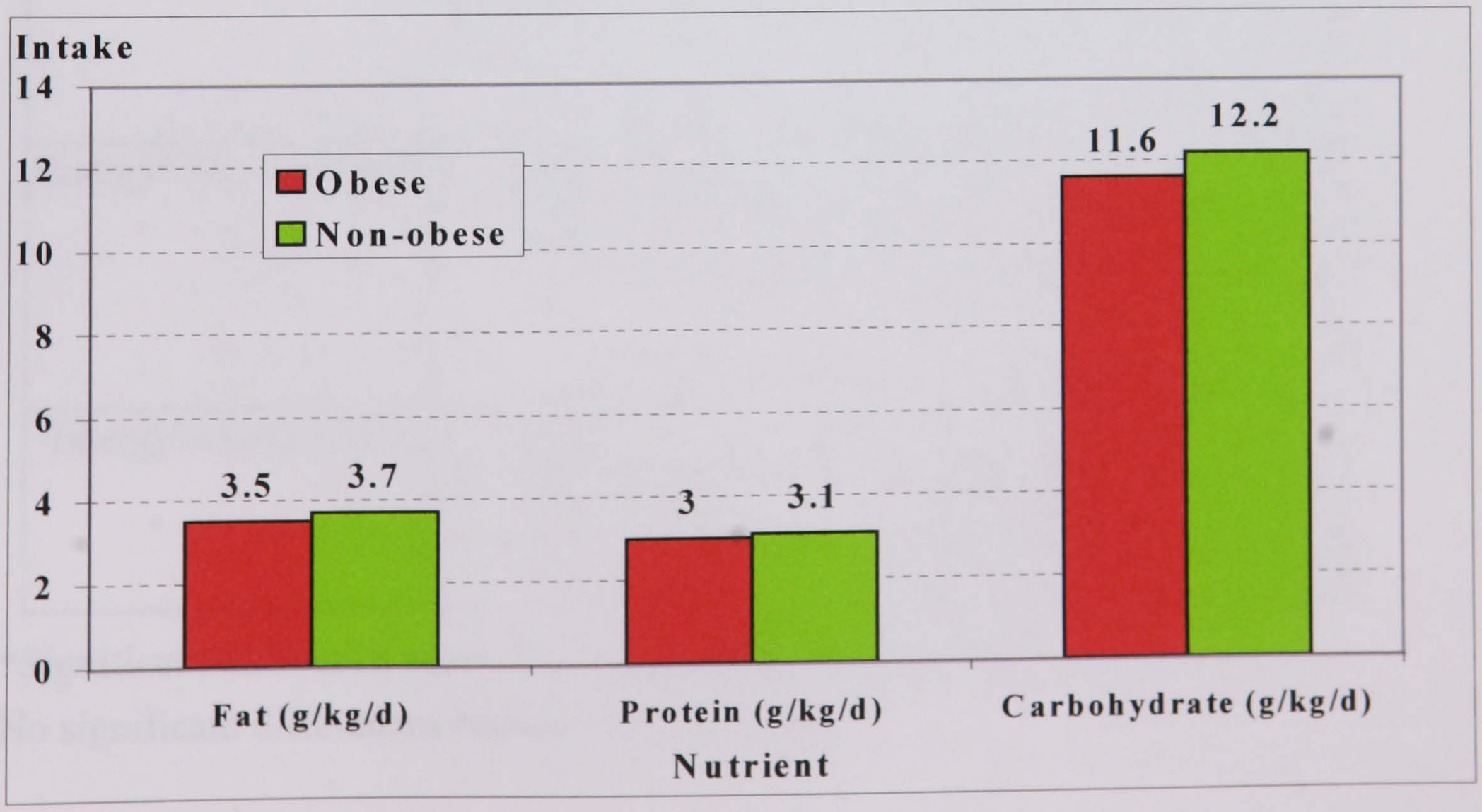


Table 7.2. Mean nutrient intake at age 8 months and childhood obesity at age 7.

Variable at age 8 months		**Obese**	**Non-obese**
Fat (g/kg/day)	Mean	3.5	3.7
	SD	0.9	1.1
	N	73	773
Protein (g/kg/day)	Mean	3.0	3.1
	SD	0.9	1.0
	N	73	773
Carbohydrate (g/kg/day)	Mean	11.6	12.2
	SD	2.9	2.7
	N	73	773
Energy (kcal/kg/day)	Mean	*87.1	92.2
	SD	19.4	20.4
	N	73	773
Energy (kJ/kg/day)	Mean	*364	385
	SD	81	85
	N	73	773
Energy% from fat	Mean	35.0	35.3
	SD	5.2	5.0
	N	73	773
Energy% from protein	Mean	13.3	13.0
	SD	2.7	2.6
	N	73	773
Energy% from CHO	Mean	51.7	51.7
	SD	5.9	5.5
	N	73	773

*Significant difference between obese and non-obese, $p<0.05$

No significant differences between boys and girls.

7.4.6-c. Macronutrient intakes at age 18 months

There was a significant difference between carbohydrate as well as energy intake at age 18 months between the children who were obese at age 7 years and the non-obese children ($p<0.01$). The obese 7 year old children had significantly lower carbohydrate and lower energy intakes (kJ/kg/day) at 18 months than the non-obese children. No significant differences were found between fat and protein intake at age 18 months or the percent of energy from fat, carbohydrate, and protein between obese and non-obese 7 year old children (Table 7.3 and Figure 7.10). No significant gender difference was observed.

Figure 7.10. Nutrient intake (g/kg/day) at 18 months and childhood obesity at 7 years.

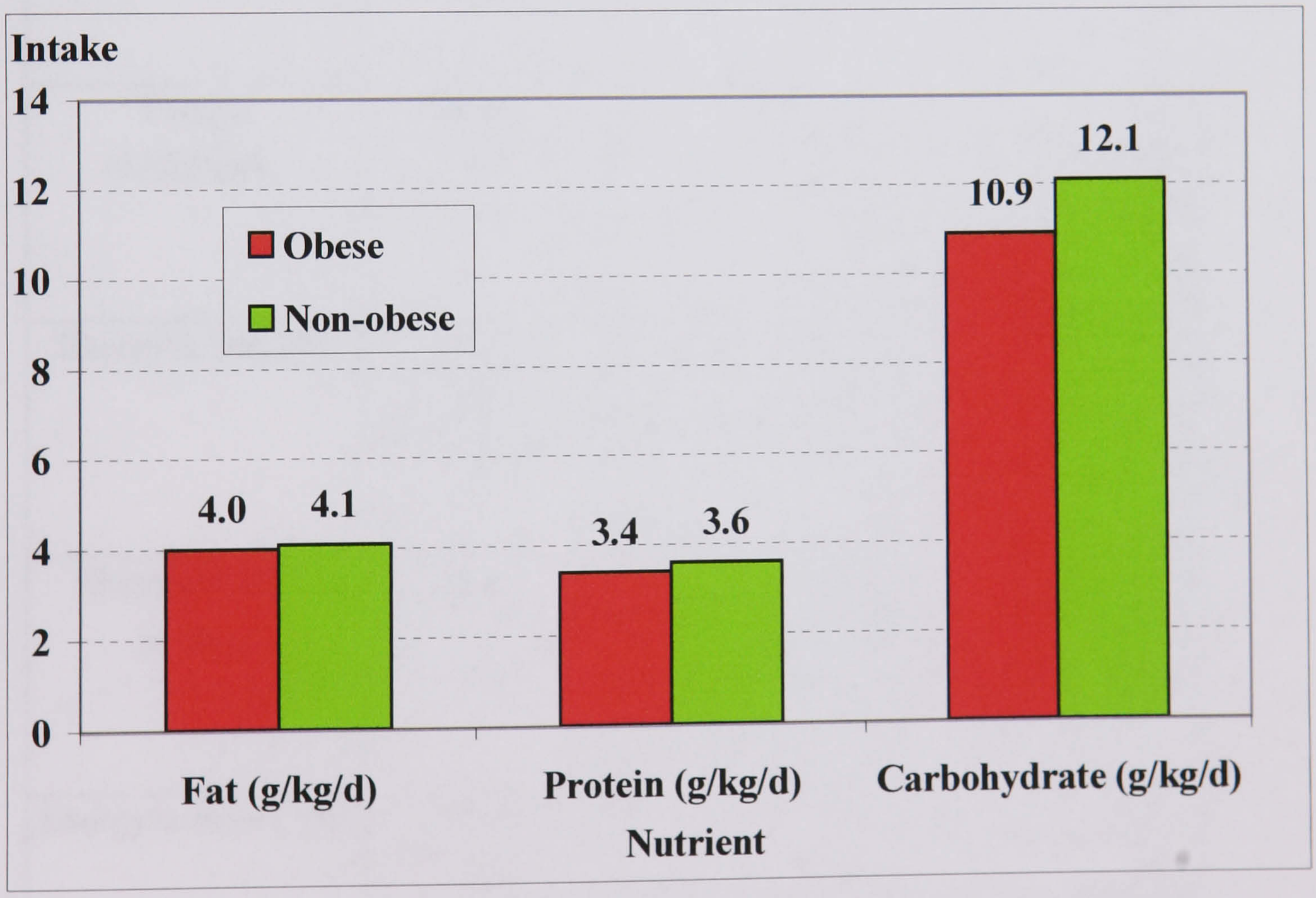


Table 7.3. Nutrient intake at age 18 months and childhood obesity at age 7.

Variable at age 18 months		Obese	Non-obese
Fat (g/kg/day)	Mean	4.0	4.1
	SD	1.0	1.1
	N	71	689
Protein (g/kg/day)	Mean	3.4	3.6
	SD	0.9	0.9
	N	71	689
Carbohydrate (g/kg/day)	Mean	*10.9	12.1
	SD	2.4	2.9
	N	71	689
Energy (kcal/kg/day)	Mean	**90.1	96.9
	SD	18.3	20.5
	N	71	689
Energy (kJ/kg/day)	Mean	**378	407
	SD	77	86
	N	71	689
Energy% from fat	Mean	38.3	36.8
	SD	3.9	4.8
	N	71	689
Energy% from protein	Mean	14.7	14.6
	SD	2.2	2.2
	N	71	689
Energy% from CHO	Mean	47.0	48.6
	SD	5.1	5.7
	N	71	689

Significant difference between obese and non-obese; * $p<0.001$, ** $p<0.01$

No significant differences between boys and girls.

7.4.6-d. Macronutrient intakes at age 43 months

There were a significant differences for all macronutrients including carbohydrate, protein, fat, and energy intakes at age 43 months between the children who were obese at age 7 years and the non-obese children ($p<0.001$). The obese 7 year old children had significantly lower macronutrients (g/kg/d) intake and energy (kJ/kg/day) intakes at 43 months of age than the non-obese children. No significant differences were found in the percentage of energy from fat, carbohydrate, and protein intakes at age 43 months between obese and non-obese 7 years old children (Table 7.4 and Figure 7.11). No significant difference was observed between the sexes.

Figure 7.11. Nutrient intakes (g/kg/day) at 43 months and childhood obesity at 7 years.

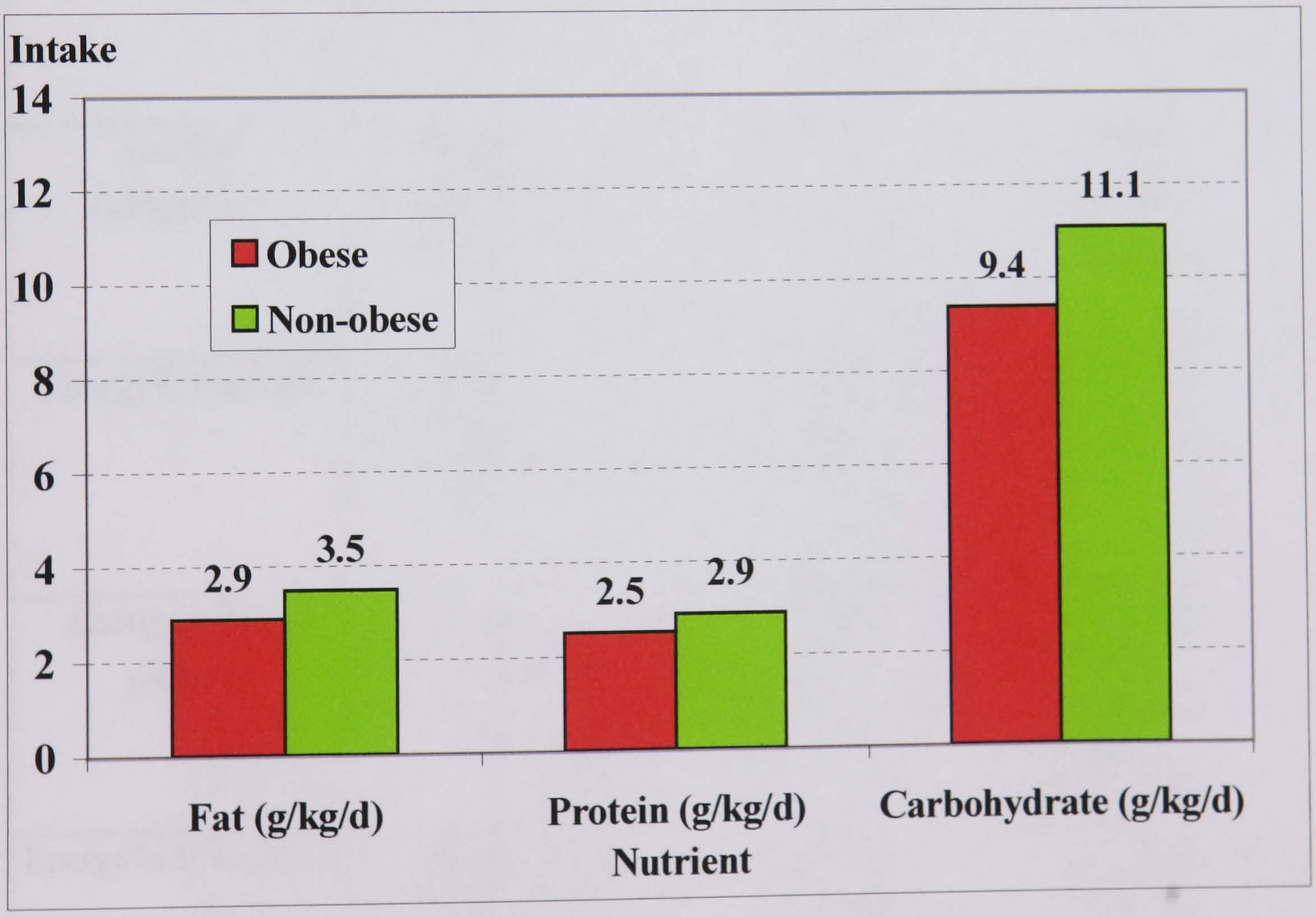


Table 7.4. Nutrient intake at age 43 months and childhood obesity at age 7 years.

Variable at age 43 months		Obese	Non-obese
Fat (g/kg/day)	Mean	*2.9	3.5
	SD	0.8	0.9
	N	61	626
Protein (g/kg/day)	Mean	*2.5	2.9
	SD	0.7	0.7
	N	61	626
Carbohydrate (g/kg/day)	Mean	*9.4	11.1
	SD	2.4	2.1
	N	61	626
Energy (kcal/kg/day)	Mean	*71.6	84.7
	SD	15.5	15.7
	N	61	626
Energy (kJ/kg/day)	Mean	*301	356
	SD	65	66
	N	61	626
Energy% from fat	Mean	35.3	35.9
	SD	4.9	4.5
	N	62	626
Energy% from protein	Mean	13.6	13.4
	SD	2.4	2.3
	N	62	626
Energy% from CHO	Mean	51.0	50.8
	SD	5.9	5.0
	N	62	626

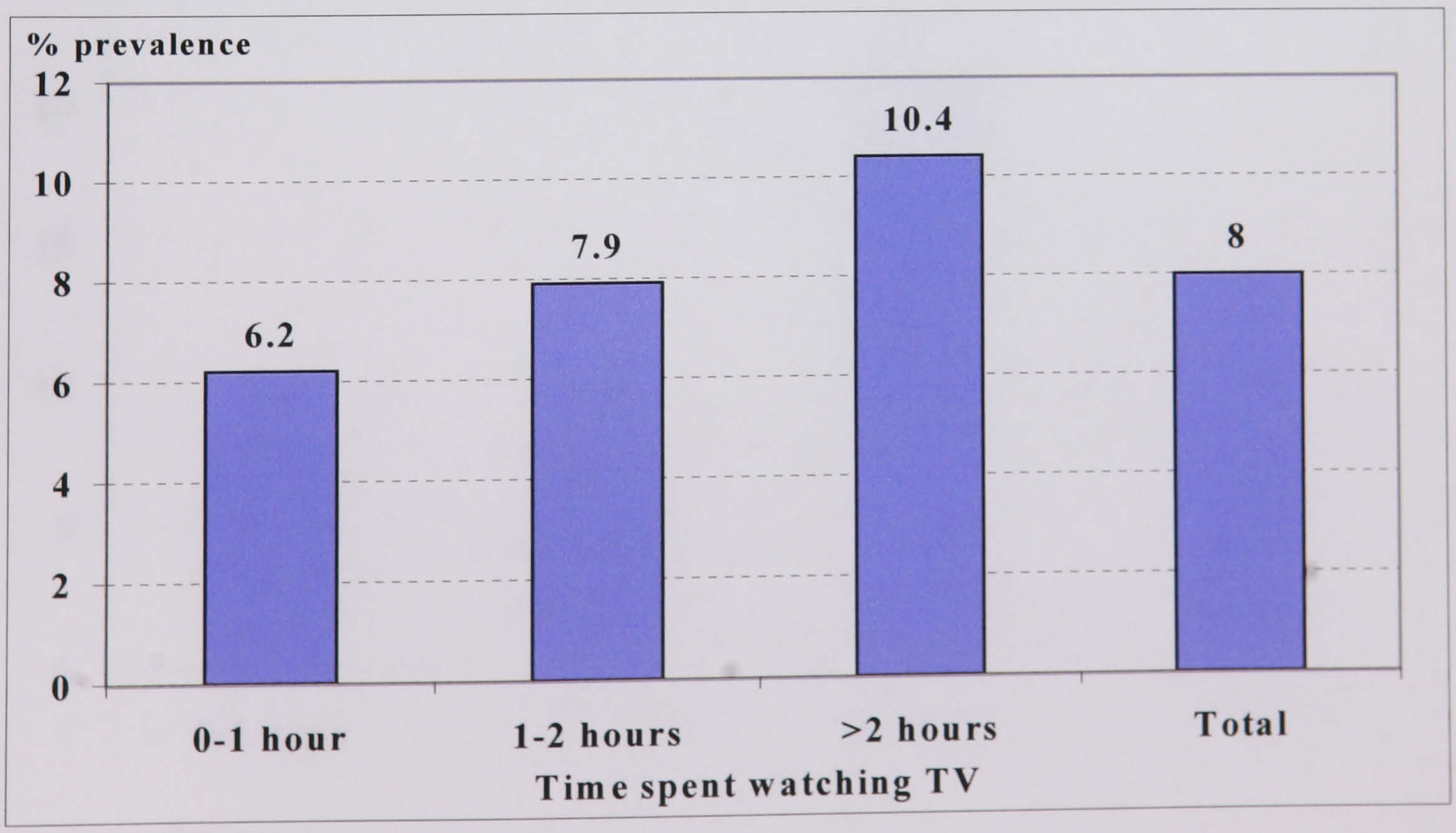
*Significant difference between obese and non-obese, $p<0.001$

No significant differences between boys and girls.

7.4.6-e. Sedentary behaviour: television viewing, time spent in car and obesity at age 7

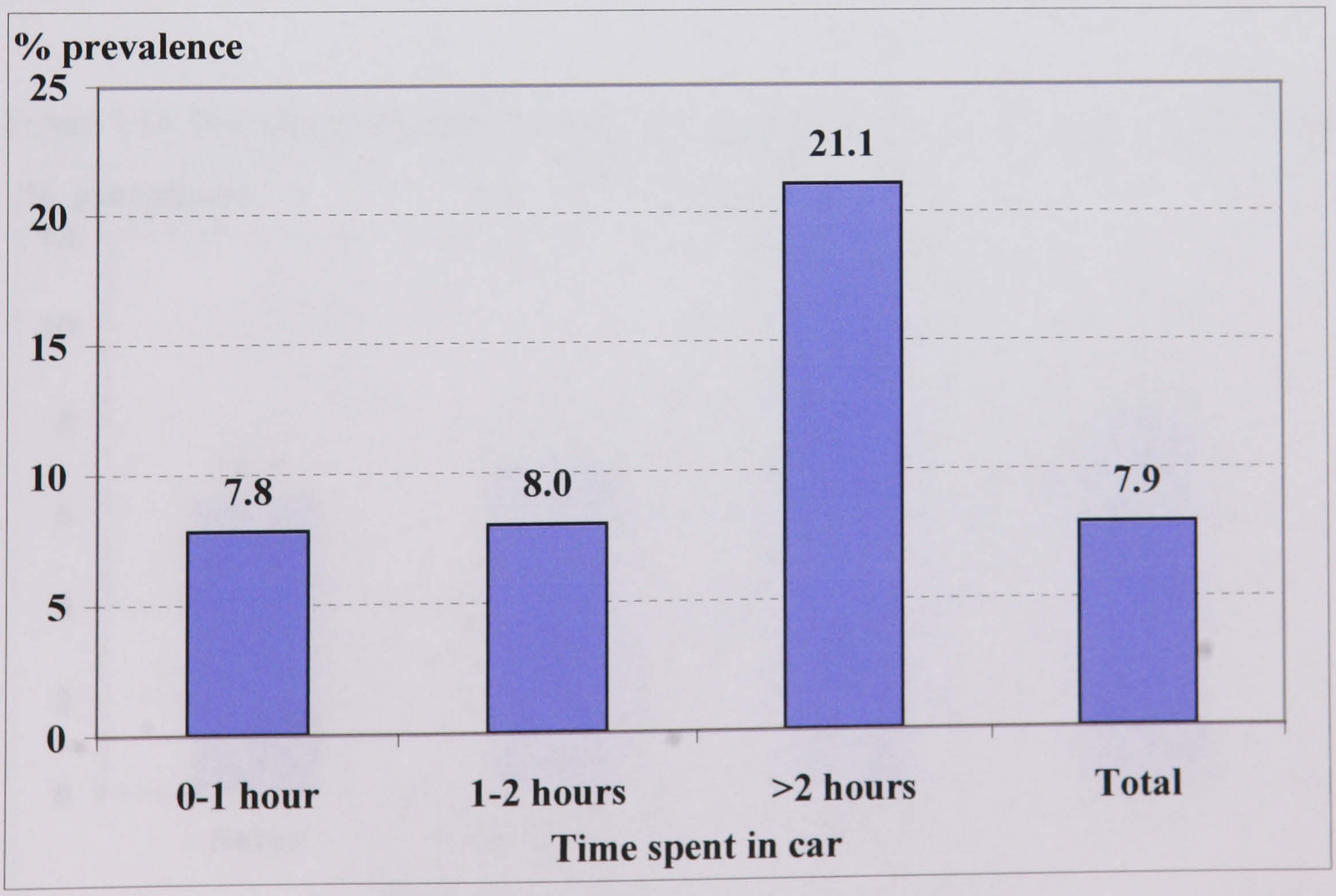
Data on TV viewing habits (parental self-report) were obtained at age 38 months from 6881 (88.6%) out of 7766 study children. Of them, 30.1% watched TV less than an hour, 46.8% 1-2 hours, and 23.1% more than 2 hours per day during weekdays. A high percentage of obese children (30.1%) at age 7 years spent a mean of more than 2 hours per day watching television at age 38 months while this was 22.5% in the non-obese. The differences in prevalence associated with reported TV viewing (Figure 7.12) were statistically significant ($p<0.001$). No significant difference was seen between boys and girls in terms of the relationship between mean time spent watching television during weekdays at age 38 months and the prevalence of childhood obesity at age 7 years. The odds ratios of childhood obesity (level 1 of analysis) for the children who watched TV 1-2 hours per day, and for those who watched >2 hours per day were 1.29 (95% CI: 1.04-1.61) and 1.74 (95% CI: 1.37-2.21) respectively compared to those who watched none or <1 hour per day. After adjustment for parental social class (level 2 of analysis), the association between TV viewing and the childhood obesity was still significant (Table 7.5).

Figure 7.12. Childhood obesity prevalence at 7 and time spent watching TV weekdays at 38 months.



Data on mean time spent in a car during weekdays (parental self-report) were obtained at age 38 months from 6893 (88.8%) study children. A high percentage (88.7%) of children spent less than an hour in car during weekdays, 10.5% spent 1-2 hours, and only 0.8% spent more than 2 hours a day in car. The percentages of obesity in association with the time spent in car (hours per day) during weekdays are shown in Figure 7.13 and the differences were statistically significant ($p<0.005$). No significant difference was seen between boys and girls in terms of the relationship between mean reported time spent in car and the prevalence of childhood obesity. The odds ratios for childhood obesity (level 1 of analysis) in the category spent more than 2 hours per day was 3.16 (95% CI: 1.66-6.01) compared to those who spent none or <1 hour. After adjustment for parental social class (level 2 of analysis), the association between time spent in car and the childhood obesity was still significant (Table 7.5).

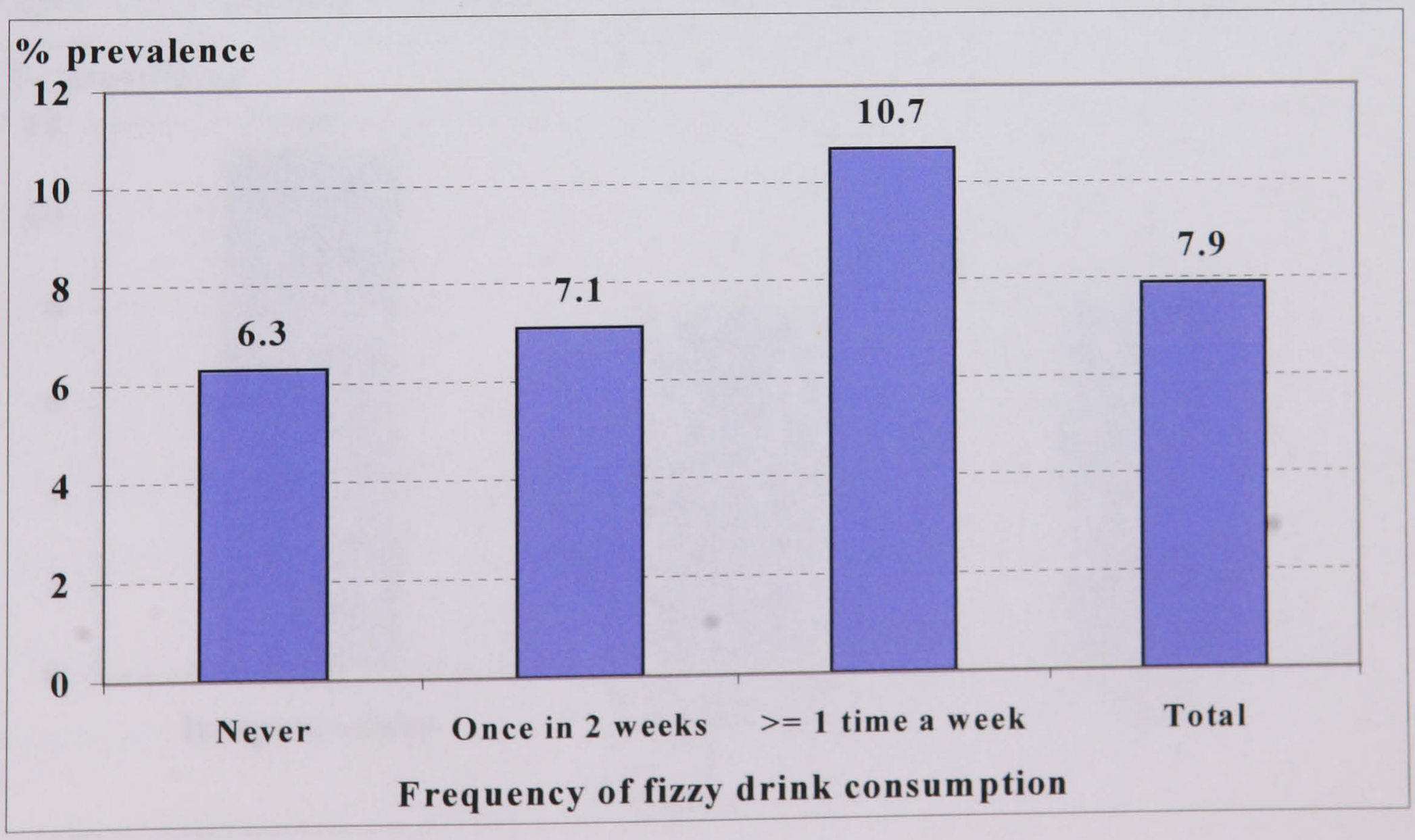
Figure 7.13. Childhood obesity prevalence at age 7 and time spent in car on weekdays at 38 months.



7.4.6-f. Consumption of fizzy drinks and obesity at age 7

From the 7766 7 year old children, information about sugar sweetened / diet fizzy drinks at age 38 months was available for 6849 (88.2%) of them (3507 boys and 3342 girls). More than half of 38 month old children had fizzy drinks. The breakdown was as follows: 44.1% never; 23.5% once per 2 weeks; 23.0% 1-3 times a week; 7.1% 4-7 times a week; 2.4% of them reported having fizzy drinks more than once a day. As Figure 7.14 shows, the prevalence of childhood obesity at age 7 years was lowest in those who did not have fizzy drinks regularly at age 38 months (6.3%) and it was highest in those who had fizzy drinks once or more per week (10.7%). These differences were statistically significant ($p<0.001$). No significant differences were seen in the association of fizzy drink consumption and childhood obesity between boys and girls. The odds ratio of childhood obesity (level 1 of analysis) for the children who consumed fizzy drinks 1 to 7 times or more a week compared to those who never consumed or consumed once in 2 weeks, was 1.70 (95% CI: 1.43-2.04). After adjustment for parental social class (level 2 of analysis), the association between fizzy drinks and the childhood obesity was still significant (Table 7.5).

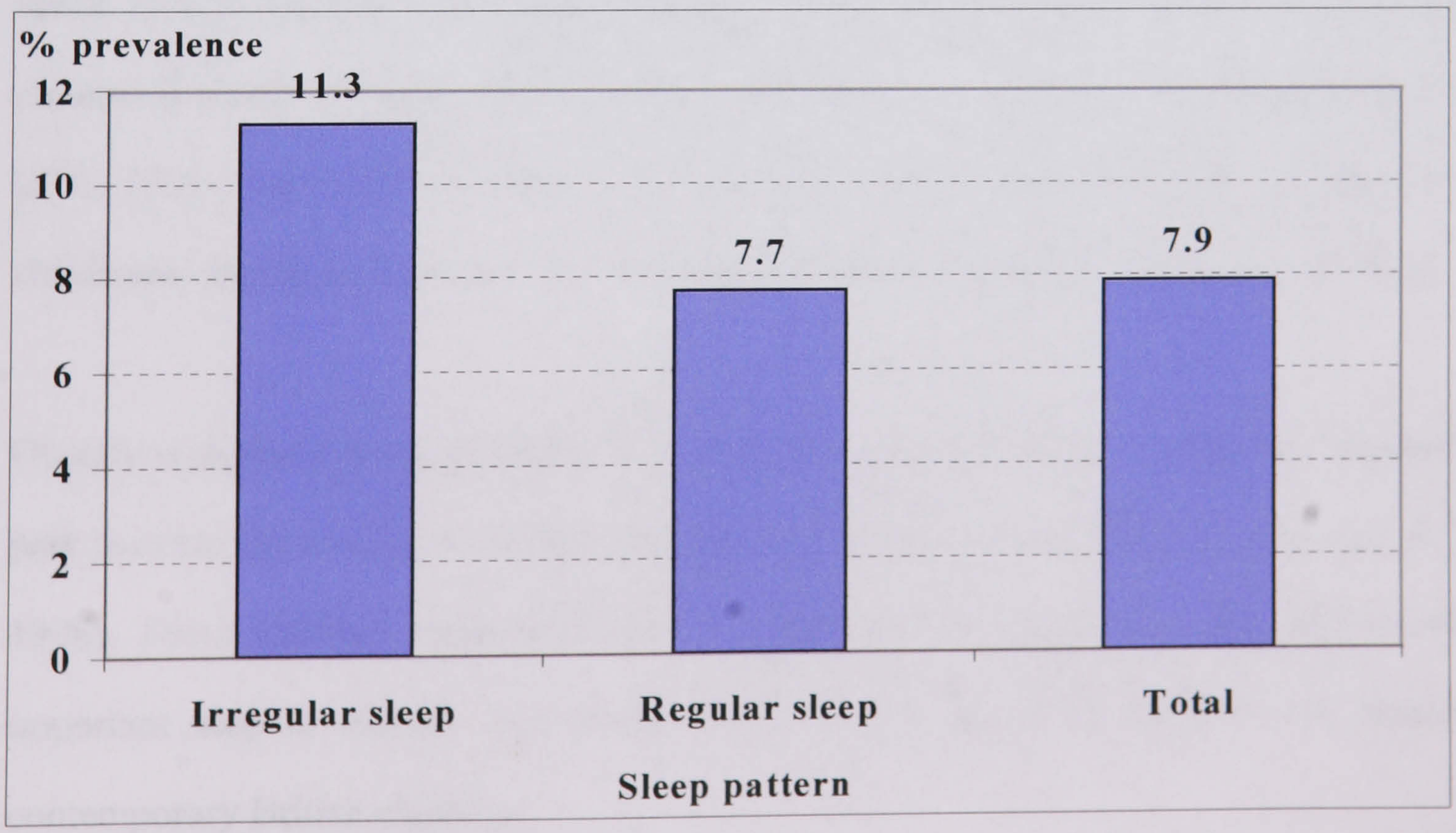
Figure 7.14. Prevalence of childhood obesity at 7 and consumption of fizzy drinks at 38 months.



7.4.6-g. Sleeping and childhood obesity at age 7

From the 7766 7 year old children, the information about sleeping at ages 30 and 42 months for 6988 (90.0%) and 6869 (88.4%) respectively were available based on parental reports. Obese 7 year olds had an average of 11.0 (SD=1.1) and 11.1 (SD=0.9) reported hours sleep per night at ages 30 and 42 months respectively, while non-obese children had an average of 11.2 (SD=1.0) and 11.3 (SD=0.8) hours per night respectively but these small differences were statistically significant (Table A.18 Appendix). In addition, girls were reported to sleep slightly more than boys during the night (11.3 vs. 11.2 hours at ages 30 & 42 months) and these slight differences were statistically significant. A total of 9.5% of children at age 30 months and 6.8% at age 42 months were described as not having a regular sleep routine. The prevalence of childhood obesity at age 7 years in those who had an irregular sleep routine at 30 and 42 months of age was 11.0% and 11.3% respectively and this was significantly higher than the prevalence for the rest of the sample ($p<0.005$). The odds ratios for an irregular sleep routine (level 1 of analysis) at ages 30 and 42 months and childhood obesity at age 7 years were 1.51 (95% CI: 1.16 - 1.98) and 1.54 (95% CI: 1.13 - 1.54) respectively. After adjustment for parental social class (level 2 of analysis), association between sleeping and childhood obesity remained significant (Table 7.5).

Figure 7.15. Prevalence of childhood obesity at age 7 and regularity of sleep at age 42 months.



7.5. Discussion

7.5.1. Principal consequences of childhood obesity, and importance of risk factors

The high prevalence of childhood obesity in children and secular trends of obesity in UK children, as well as the possible roles of genetics, energy intake, and physical activity were discussed in Chapter 3. Childhood obesity is a problem worldwide and is prevalent in developing countries too as mentioned in Chapter 4. As discussed in Chapters 3 and 6, obese children have a high probability of becoming obese adults, and adiposity in infants (independent of measurement method) increases the risk of subsequent obesity (Garn & Lavelle 1985; Rolland-Cachera et al 1987). Obesity will persist in approximately 50% of overweight children or adolescents, regardless of the measure used (Stark et al 1981; Freedman et al 1987; Mossberg 1989). Approximately twice as many individuals who reported as 'considered a fat girl' had an ideal body weight of >155% as young adult women (Rimm & Rimm 1976). Overweight adolescents were shown to have increased rates of adulthood diabetes, coronary heart disease, atherosclerosis, hip fracture and gout (Dietz 1998a). Some childhood obesity related psychosocial consequences represent a possible source of adult morbidity. Lower rates of marriage, fewer years of education completed, lower family income, and higher rates of poverty are some important consequences of childhood obesity (Dietz 1998a). Higher blood pressure (systolic and diastolic) and higher LDL, HDL, and total cholesterol were shown to be some biological consequences of childhood obesity in later adult life in the Bogalusa heart study (Wattigney et al 1995).

Obesity is difficult to treat (NIH 1993), and prevention efforts are difficult to implement in part because the causes of energy imbalance are not fully understood (Whitaker & Dietz 1998). Dietz (2000a) considered that identification of modifiable risk factors was an important step to obesity prevention. This Chapter aimed to identify risk factors for contemporary British children.

7.5.2. Adult obesity risk factors reported in British studies

In the United Kingdom, there has been an increase in the prevalence of obesity (BMI >30) from 6% and 8% in men and women in 1980 to 17% and 20% in 1997 (Jebb 1999). Obesity is the result of an imbalance between energy intake and energy expenditure. The National Food Survey (NFS) reported a 20% decrease in mean household food intake in the UK between 1970 to 1990, corrected for changes in confectionery, soft drinks, and alcohol consumption. The NFS data was supported by the results of other cross-sectional dietary surveys, which show a fall in reported energy intake (Prentice & Jebb 1995). Therefore, reduced energy expenditure might be responsible for increased adult obesity. Low levels of physical activity were identified as a more important risk factor for excess weight gain than any features of the habitual diet (Prentice & Jebb 1995). There are no direct measurements of secular trends in physical activity in UK adults, but good data exist to show significant increases in the time spent watching TV and increased car ownership (Jebb & Moore 1999).

The Allied Dunbar Health and Fitness Survey (1992) and the White et al (1993) study (using Health Survey for England 1991 data) used similar questionnaire methods to assess participation in sports and active pastimes during the four weeks before interview in large samples of adults. Their estimates of participation rates in various activities based on self-report showed that 30-35% of adults had undertaken <80 minutes of any type of moderate activity in last month; >80% had not walked continuously for two miles; 90% had not cycled; 50-60% had not participated in any moderately vigorous sport; and only 20-30% participated in vigorous activity of any type (Prentice & Jebb 1995).

Prevalence of adult obesity varies in different social classes in the UK especially in women and ranges from 11.1% in professionals to 23.6% in partly skilled / unskilled women in the 1995 HSE study (Prescott-Clarke & Primatesta 1997). Despite differences in the types of food consumed by different social groups, there are relatively small differences in energy intake between them (Gregory et al 1990), so physical activity might contribute to the obesity prevalence differences between social classes. There are significant differences in reported

physical activity between social classes, with no activity reported in 18% men and 21% women of social classes IV and V in the preceding 4 weeks, compared to 14% and 13% of men and women of social classes I and II (Allied Dunbar Health and Fitness Survey 1992).

The Health Survey for England (1995) analysis (by Prescott-Clarke & Primatesta 1997) across the age range showed that the prevalence of obesity in British men and women increased from 5.8% and 8.0% in 16-24 year old men and women to 21.5% and 23.2% in 55-64 year olds. There was little or no association between the increasing prevalence of obesity and differences in either energy intake or levels of inactivity in 14-44 year olds; but there was a strong association between rising levels of inactivity and obesity in 44-64 year olds (Prescott-Clarke & Primatesta 1997).

7.5.3. Childhood obesity risk factors in the UK

Increasing prevalence of overweight and obesity in British children has recently been reported by different studies. Reilly & Dorosty (1999) study on Health Survey for England (1996) data of 6-15 year old children showed a high prevalence of childhood obesity (defined as BMI $\geq 95^{th}$ centile; 10.4% in 6 year olds to 16.8% in 15 year olds). Bundred et al (2001) reported a highly significant increasing trend in the proportion of overweight (BMI $\geq 85^{th}$ centile) changing from 15% in 1989 to 24% in 1998, and also a significant increase in the percentage of obesity (BMI $\geq 95^{th}$ centile) changing from 5% to 9% during the same period. A number of other UK studies were published after the present study had started (Reilly & Dorosty 1999; Reilly et al 1999a; Kinra et al 2000; Sahota et al 2000; Bundred et al 2001; Chinn & Rona 2001; Rudolf et al 2001). As discussed earlier in the introduction of this Chapter, some factors were reported as childhood obesity risk factors in studies of British children. They included catch-up growth (Ong et al 2000), birth weight (Duran-Tauleria et al 1995; Phillips & Young 2000), the age of the introduction of solids (Wilson et al 1998), parental obesity (Poskitt & Cole 1978; Duran-Tauleria et al 1995; Lake et al 1997), and social deprivation (Kinra et al 2000), but these were often based on smaller or older studies with limited correction for confounding factors, and with definitions of childhood obesity which would not be very acceptable today.

7.5.4. Main risk factors for childhood obesity investigated in the current study

In the present study, due to the nature of cohort data, identifying the cause-effect relationship between risk factors and childhood obesity was not possible and therefore, the associations between risk factors with childhood obesity are presented. The association between a list of 19 risk factors and childhood obesity at age 7 years was investigated in this study and 4 of them showed no association with childhood obesity at the first level of analysis (as explained in the statistical methods Section of the present Chapter). Gestational age, an intra-uterine factor, showed no association with childhood obesity while two other intra-uterine factors, catch-up growth (to age 2 years), and birth weight, showed a significant association with childhood obesity at age 7 years. Both breast-feeding and the timing of the introduction of solids, which were grouped as infant feeding risk factors, showed significant associations with childhood obesity. From the demographic risk factors for childhood obesity, paternal / maternal education, paternal / maternal obesity, and paternal / maternal social class showed significant associations with childhood obesity at age 7, while gender, ethnicity, and the season of birth showed no associations with childhood obesity. The childhood lifestyle risk factors of energy intake at 8, 18, and 43 months and macronutrient (fat, protein, and carbohydrate) intakes at 43 months, watching TV, time spent in car, consumption of fizzy drinks, and sleeping regulation / duration at ages 30 & 42 months also showed significant associations with childhood obesity.

To briefly recap on the analysis used here (see the statistical methods Section of this Chapter) univariate analysis (level 1 analysis) was followed by analysis with adjustment for parental social class (level 2 analysis) of those variables which were significantly associated with childhood obesity in level 1 of the analysis. This was then followed by a multi-variate (level 3) analysis in which the possible association of each variable with obesity was adjusted for all other variables associated with childhood obesity after the first adjustment

for parental social class. However, this latter analysis was problematic, notably because the sample sizes were small for many variables (it required that data on all risk factors were available for each child entered in the analysis). For the purpose of clarity of presentation of this discussion, focus is on the univariate (level 1) analysis and analysis with adjustment for social class (level 2) since interpretation of the multi-variate (level 3) analysis was difficult (see below).

Level 1 analysis: from the 19 investigated variables, 4 of them including gestational age, gender, ethnicity, and season of birth showed no significant association with childhood obesity at the first level of analysis. Data for catch-up growth and macronutrient intakes were available only for CIF children and, although they were associated with childhood obesity, due to the small sample size they were not adjusted for other potential risk factors. Therefore, the association between the 13 variables listed above (Table 7.5) with obesity at age 7 year was adjusted for parental social class (level 2 analysis).

Level 2 analysis: although the age of the introduction of solid foods was significantly associated with childhood obesity in the first level of analysis and after adjusting for paternal social class, after adjusting for maternal social class, this factor *was not* significantly associated with childhood obesity at age 7 years (Table 7.5). This means that it is likely that the age of the introduction of solids is dependent on maternal social class and there is good evidence that this is the case (DHSS 1994). Two other variables which showed no significance after adjustment for social class, were maternal social class when adjusted for paternal social class, and paternal social class when adjusted for maternal social class. This was because both paternal and maternal social classes are very closely associated with each other and in fact, it is probably the family social class which is important. However, since a single variable for family social class was not available (as described in Chapter 2), we adjusted the other variables for both maternal and paternal social class separately (Table 7.5).

Level 3 of analysis: for the third level of analysis, the age of the introduction of solids and paternal social class were excluded, then the association between each of the other remaining 11 variables with childhood obesity was adjusted for all of the other 10 risk factors. The results (Table A.19 Appendix) showed that maternal and paternal obesity were independently associated with childhood obesity at age 7 as well as the consumption of fizzy drinks (more than once per week), birth weight (≥3800 grams), and time spent in the car.

From the total sample of 7766 children, 4205 (54.2%) had data missing in the level 3 multi-variate analysis (Table A.20 in Appendix). Further investigation showed that the prevalence of childhood obesity (dependent variable) was significantly higher in the children with missing data. This implies a bias in the results of childhood obesity in the final adjustment process which might possibly cause some of the dependent variables not to be significant after the final adjustment. Although complete data for 3561 (45.8%) children were available (Table A.20 Appendix), the representativeness of the results must be in doubt and the overall reduction in sample size also reduced the study power. Six dependent variables showed no significant association with childhood obesity after adjustment for all other factors. However, the results indicate bias for the association between those variables and childhood obesity. For instance, the prevalence of those children who watched TV more than 2 hours per day was significantly higher in the missing data group (Table A.20 Appendix), and the prevalence of those children who were never breast-fed by age 6 months was higher in the missing data group. The significantly higher percentages of low educated parents, and children with irregular sleeping, as well as the differences in social class between the missing data group and the group which entered in the final adjustment process all suggest presence of bias in the adjustment analysis process. However, the results of level 3 analysis are presented in Table A.19 Appendix for completeness. Due to biases (as mentioned above), the results in Table A.19 (in Appendix) should be interpreted cautiously.

Table 7.5. Summary of associations between childhood obesity risk factors and childhood obesity at age 7 before and after adjustment (levels 1 and 2 analysis).

Group	Variable		Total number	Before adjustment (level 1)			After adjustment for *maternal social class* (level 2)			After adjustment for *paternal social class* (level 2)		
				OR	95% CI	Sig.	OR	95% CI	Sig.	OR	95% CI	Sig.
Intra-uterine*	**Catch-up growth** vs. non catch-up		806	3.04	1.80-5.15	0.001	-	-	-	-	-	-
	Birth weight	2500-3800 vs. <2500	7673	1.33	0.85-2.09	0.000	1.55	0.88-2.73	0.000	1.64	0.93-2.88	0.000
		>3800 vs. 2500-3800		1.58	1.32-1.89		1.46	1.19-1.80		1.49	1.23-1.82	
Infant feeding / weaning	**Breast-fed** vs. up to 6 month	<6 month	7205	1.32	1.08-1.62	0.002	1.26	1.00-1.58	0.030	1.27	1.01-1.59	0.03
		None		1.55	1.22-1.97		1.45	1.09-1.92		1.43	1.08-1.87	
	Introduction of solids before 12 vs. after 12 weeks		6291	1.43	1.14-1.79	0.002	1.25	0.98-1.60	0.070	1.37	1.09-1.71	.007
Demographics / other variables†	**Mother education**	None/CSE	7468	2.48	1.78-3.46	0.000	2.11	1.35-3.28	0.000	2.30	1.55-3.43	.000
		Vocational		2.53	1.76-3.62		2.39	1.52-3.76		2.43	1.61-3.67	
		O level		1.64	1.22-2.23		1.47	1.00-2.19		1.61	1.13-2.28	
	vs. Degree	A level		1.68	1.23-2.30		1.50	1.02-2.20		1.72	1.21-2.44	
	Partner education	None/CSE	7263	2.09	1.58-2.76	0.000	1.62	1.16-2.25	0.04	1.81	1.27-2.59	0.02
		Vocational		1.65	1.14-2.39		1.50	1.00-2.26		1.50	0.99-2.31	
		O level		1.68	1.27-2.23		1.38	1.00-1.90		1.54	1.11-2.15	
	vs. Degree	A level		1.48	1.13-1.95		1.18	0.86-1.61		1.34	0.97-1.84	
	Obese parent (BMI >30)	obese father	7004	2.57	1.88-3.51	0.000	2.67	1.88-3.78	0.000	2.50	1.81-3.44	0.000
		obese mother		4.40	3.16-6.12		4.43	3.04-6.44		4.21	2.99-5.92	
	vs. non-obese	Both obese		11.5	6.56-0.28		12.3	6.55-23.2		11.3	6.36-20.0	
	Mother social class	II	6058	1.87	1.05-3.33	0.02	-		-	1.63	0.88-3.02	0.13
		IIIM		1.89	1.07-3.35					1.58	0.85-2.92	
		IIIN		2.74	1.45-5.16					2.28	1.15-4.51	
		IV		2.55	1.37-4.76					2.04	1.04-4.02	
	vs. class I	V		2.69	1.14-6.35					1.79	0.69-4.63	
	Partner social class	II	6648	1.50	1.06-2.12	0.01	1.33	0.91-1.93	0.16	-		-
		IIIM		1.62	1.09-2.41		1.39	0.90-2.15				
		IIIN		1.82	1.29-2.57		1.56	1.06-2.30				
		IV		2.03	1.32-3.10		1.82	1.14-2.91				
	vs. class I	V		1.62	0.85-3.08		1.56	0.80-3.04				
Childhood lifestyle‡	**Watch TV**	1-2 hours	6881	1.29	1.04-1.61	0.000	1.16	0.90-1.48	0.003	1.22	0.96-1.55	0.002
	vs. <1 hour	>2 hours		1.74	1.37-2.21		1.56	1.19-2.05		1.61	1.23-2.09	
	Spent in car	1-2 hours	6881	1.04	0.78-1.38	0.002	1.03	0.74-1.41	0.002	1.02	0.75-1.39	0.002
	vs. <1 hour	>2 hours		3.16	1.66-6.01		3.46	1.72-6.95		3.45	1.74-6.86	
	Fizzy drink once to ≥7 times / week vs. none / once per 2 weeks		6849	1.70	1.43-2.04	0.000	1.62	1.32-1.98	0.010	1.57	1.30-1.91	0.000
	Irregular sleep at 42 months vs. regular		6869	1.54	1.14-2.08	0.005	1.47	1.03-2.08	0.030	1.44	1.06-1.96	0.02

* Gestational age had no association to childhood obesity at age 7.

† Gender, ethnicity, and season of birth showed no association to childhood obesity at age 7.

‡ Macronutrient intakes at ages 8, 18, and 43 months was associated to childhood obesity at age 7 but due to small sample size was not adjusted.

7.5.5. Childhood obesity risk factors in present study compared to other studies

7.5.5-a. Intra-uterine factors

Gestational age

The mean gestational age for obese children was 39.5 weeks (SD=1.7) and for non-obese children it was 39.4 weeks (SD=1.9), which obviously were very similar, and no association between gestational age and childhood obesity at age 7 was found in the present study. Although no association between gestational age and childhood obesity was reported by some other studies, the US studies of O'Callaghan et al (1997) and Patterson et al (1997) have reported that being small for gestational age (and not gestational age itself) is a factor associated with reduced childhood obesity risk.

Catch-up growth

A significant association between catch-up growth and childhood obesity was found in the present study of ALSPAC children. This suggests that those children who had a rapid increase in BMI standard deviation scores from birth to later childhood, continued to have relatively high BMI SDS in later childhood and were more likely to be obese (BMI SDS $\geq 95^{th}$ centile) at age 7; these results are consistent with Ong et al (2000) who used adiposity and not obesity as an outcome measure.

Birth weight

In the present study, birth weight had a strong and independent association with childhood obesity at age 7, so that those who were in the top quartile for birth weight at birth (>3800 grams; as described in the methods Section of this Chapter) had a significantly higher prevalence of childhood obesity. These results are in agreement with the UK studies of

Duran-Tauleria et al (1995), and Phillips & Young (2000). The association between birth weight and childhood obesity has been reported by several other studies world wide (e.g. Locard et al 1992; Lehingue et al 1993; O'Callaghan et al 1997; Plagemann et al 1997; Rasmussen & Johansson 1998; Kromeyer-Hauschild et al 1999; Takahashi et al 1999). However, the present results are not in agreement with the results of the British study of Seidman et al (1995) which was not able to confirm birth weight as a factor associated with childhood obesity. As already mentioned, birth weight of British children has been reported to have changed over time (Power 1994). This might partly contribute to the increased prevalence of childhood obesity. In addition, a number of different definitions of childhood obesity have been used in previous studies and a number of previous studies took place before the rapid increase in UK childhood obesity prevalence in the 1990s (Poskitt & Cole 1977; Peckham et al 1983; Poskitt 1986) which makes comparison between studies difficult.

7.5.5-b. Infant feeding / weaning factors

Breast-feeding

Formula feeding, or short duration of breast-feeding, was a factor which was significantly associated with childhood obesity in the current study. Studies have shown that the total protein intake is higher and energy metabolism lower in breast-fed than formula-fed children (Heinig et al 1993; Whitehead 1995), this might have a possible role in increasing the prevalence of childhood obesity in non breast-fed children. The results of this study are in agreement with the studies of von Kries et al (1999), and Kramer (1981) which found a significant association between no breast-feeding and childhood obesity though Wadsworth et al (1999) suggested that the study by von Kries et al (1999) did not make adequate adjustment for social class (not possible in the von Kries study since social class data could not be easily collected in Germany). The British study of Wadsworth et al (1992) showed no significant relation of breast-feeding with obesity in 6 year olds who grew up in Britain

during the 1940s when adjusted for social class, though outcomes in their subjects occurred nearly 60 years before the present study.

Timing of the introduction of solids

The age of the introduction of solids in our study was found to be associated with childhood obesity (Table 7.5) so that those children who started solids earlier than 12 weeks of age, had a significantly higher prevalence of childhood obesity at age 7. However, after adjustment for maternal social class (Table 7.5), the association was not significant. This indicated that social class has an important effect on the timing of the introduction of solids, and must be considered when studying its association with childhood obesity. These findings, however, are consistent with the DHSS 1988 & DHSS 1994 recommendations about the age of the introduction of solids (after age 4 months). Wilson et al (1998) in their study of British children previously reported an association between the timing of the introduction of solids with childhood obesity, but did not use a recognised definition of childhood obesity and did not adjust for social class.

7.5.5-c. Demographics / other factors

Gender

In the present study, and in several other very recent British studies on the prevalence of childhood obesity (Reilly & Dorosty 1999; Reilly et al 1999a; Kinra et al 2000; Bundred et al 2001; Chinn & Rona 2001; Rudolf et al 2001), gender was not found to be an important factor affecting childhood obesity risk. In addition, the French study of Locard & Gemelli (1989) and the US study of Strauss & Knight (1999) have also found no significant associations between gender and childhood obesity risk, though some other studies in African and US children did report an association (Cameron & Getz 1997; O'Callaghan et al 1997; Ogden et al 1997).

Paternal education

Parental education was significantly associated with childhood obesity (at age 7 years) in the current study of ALSPAC children such that higher qualified parents were less likely to have obese children. Both the US studies of Strauss & Knight (1999) and Patterson et al (1997) as well as the United Arab Emirates study of Moussa et al (1994) have shown similar results to our study. It might be worth mentioning that the study by Martorell et al (2000) on children in 50 developing countries reported a significant inverse association between parental education and childhood overweight, such that the children of the higher qualified parents were more likely to be obese.

Parental obesity

Parental obesity is a risk factor which was highly significantly associated with childhood obesity in our study, and many other studies (e.g. Garn & Clark 1976; Poskitt & Cole 1978; Locard et al 1992; Quek et al 1993; Moussa et al 1994; Duran-Tauleria et al 1995; Lake et al 1997; O'Callaghan et al 1997; Franzese et al 1998; Maffeis et al 1998; Whitaker & Dietz 1998; Cutting et al 1999; Strauss & Knight 1999; Takahashi et al 1999; Maffeis et al 2000; Scaglioni et al 2000). Genetic studies have confirmed parental obesity as having a major role in childhood obesity development (Sorensen 1995; Franzese et al 1998; Cutting et al 1999; Perusse & Bouchard 1999; Samaras et al 1999; Stunkard et al 1999; Chagnon et al 2000). However, Whitaker et al (2000) reported only a weak relationship between parental BMI and child adiposity (Whitaker et al 2000). It should also be noted that parental associations, if causal, probably operate by environmental as well as genetic mechanisms (Bouchard 1991; Maes et al 1997; Franzese et al 1998).

Parental social class

A highly significant association between parental social class and childhood obesity was found in the current study. Many studies worldwide have considered social class as a very important factor influencing childhood obesity risk (refer to the introduction of this Chapter about social class). The Kinra et al (2000) study of British 5-14 year olds found a significant inverse association between social deprivation (measured using the Townsend index, a geographically based measure of deprivation) and childhood obesity too, though obesity in children was defined using a slightly different criterion (BMI >98th centile) and no attempt was made to test for other risk factors or analyse confounding factors.

Ethnicity

A variety of different ethnic groups live in the USA and some US studies have described the effect of ethnicity on childhood obesity risk (Ogden et al 1997; Goran et al 1998b; Strauss & Knight 1999; McMurray et al 2000). Two French studies have reported a relationship between ethnicity and childhood obesity as well (Locard & Gemelli 1989; Locard et al 1992). However in the present study and in the study of Duran-Tauleria et al (1995) on British children, ethnicity was not significantly associated with obesity. It might be important to note that in the present study, the total number of ethnic minority children was very low (298 or 4.1% of the sample) and therefore, the power of our analysis was limited. Furthermore, this ethnic group was very diverse including African and Asian origin families and it is likely that different effects would be seen in different groups. A larger study which used over-sampling of ethnic minority families would be needed to answer this question.

Season of birth

We were unable to confirm the Phillips & Young (2000) British study report, which described season of birth as a risk factor for obesity in later life. In the present study no association was found between the month / season of birth and childhood obesity at age 7.

7.5.5-d. Childhood lifestyle

Diet

Although Maffeis et al (2000) study showed a negative correlation between carbohydrate intake and childhood adiposity, and a positive correlation between fate intake and childhood adiposity, the authors concluded that the diet composition did not contribute to childhood adiposity after adjustment for parental overweight. However, the Scaglioni et al (2000) study suggested that high protein intake in the first year of life might influence the development of adiposity. In the present study no significant difference was seen in protein intake at ages 8 or 18 months between obese and non-obese 7-year-old CIF (sub-sample of the main cohort) children. However, obese 7 year old children had significantly lower carbohydrate and lower energy intakes per kilogram body weight per day at 8 and 18 months of age, and lower macronutrient (protein, fat, and carbohydrate) and energy intake at 43 months of age than the non-obese children. The lower energy intake in obese children might be partly explained by parental under-reporting of dietary intake, but there may be other explanations. This point needs to be investigated by other studies.

Sedentary behaviour: television viewing, time spent in car

A significant association between reported hours of television viewing (on weekdays) and the prevalence of obesity in childhood (age 7 years) was found in this study. In addition, the association between hours of sitting in car (on weekdays) and childhood obesity was highly

significant. Many other studies have reported similar results (Dietz & Gortmaker 1985; Locard et al 1992; Maffeis et al 1993; Moussa et al 1994; Gortmaker et al 1996; Kagamimori et al 1999; Robinson 1999; Takahashi et al 1999). However, there are some other US studies which have introduced some doubt about the effect of television viewing on childhood obesity (Robinson et al 1993; McMurray et al 2000). The present study definition of TV viewing was based on parental report when the child was 3 years old which is unlikely to give a very accurate estimate of time spent watching TV by the children at later ages. However, in a large sample epidemiological study, this level of accuracy may be acceptable.

Consumption of fizzy drinks

The consumption of fizzy drinks at age 3 had a highly significant association with childhood obesity in the current study. The current study had a much larger sample than of previous studies though it was not possible to separate sugar sweetened from diet drinks due to the way data were collected. The results of the present study tended to confirm the Ludwig et al (2001) and Dennison et al (1997) US studies, which considered sweetened drink consumption as well as fruit juice drinking as a risk factor for childhood obesity, although the methodology of measuring type and amount of drinks in those studies was different from the current study. The association may be due to a causal mechanism, or may simply reflect the possibility that fizzy drink consumption is a marker for a more 'obesogenic' lifestyle.

Sleeping

This study showed a significant inverse association between duration, and regularity of sleeping and childhood obesity. This was consistent with two other studies (Locard et al 1992; O'Callaghan et al 1997; Kagamimori et al 1999) which suggested short sleeping periods as a possible risk factor for childhood obesity though the mechanism is unclear.

7.6. Childhood obesity risk factors in brief

In summary from the potential risk factors tested in this study, 4 (gestational age, gender, ethnicity, and season of birth) showed no association with childhood obesity. The other 15 variables showed a significant association with childhood obesity including: catch-up growth; birth weight (>3800 grams); no breast-feeding or stopping before 6 months; the introduction of solids before 12 weeks; energy intake at ages 8, 18, and 43 months, and macronutrient intake at 43 months; maternal & paternal education; maternal & paternal obesity; maternal & paternal social class; television viewing; time spent in car; fizzy drink consumption; irregular sleeping / short sleep duration. After adjusting for maternal social class, the age of the introduction of solids was not significantly associated with childhood obesity (Table 7.5). After adjusting each risk factor for all other risk factors, five remained significantly associated with childhood obesity. These were maternal and paternal obesity; birth weight; consumption of fizzy drinks; and time spent in car (Table A.19 Appendix).

Table 7.6. Summary of childhood obesity risk factors investigated in this study.

Variable	Direction	Significantly associated			Not associated
		Before adjustment (level 1)	Adjusted for social class (level 2)	Adjusted for 10 factors (level 3)	
Maternal obesity	+	√	√	√	
Paternal obesity	+	√	√	√	
Birth weight	+	√	√	√	
Fizzy drinking	+	√	√	√	
Time spent in car	+	√	√	√	
TV viewing	+	√	√		
Breast-feeding	-	√	√		
Maternal education	-	√	√		
Paternal education	-	√	√		
Maternal social class	-	√	√		
Paternal social class	-	√	√		
Sleeping	-	√	√		
Age solid introduction	-	√	√		
Macronutrient intake	-	√	*	*	
Catch-up growth	+	√	*	*	
Gestational age	+				√
Female gender	+				√
White ethnicity	+				√
Jan-Jun birth season	+				√

*Not adjusted for social class / other variables.

7.7. Strengths of the present study

The present investigation was based on a relatively large sample of prospectively collected data (as explained in Chapter 2). The subjects provided a reasonably representative sample of British children (Golding et al 2001; Chapter 2). The study benefits from recent and contemporary sampling (started in 1991-1992). ALSPAC was able to gather detailed information from thousands of parents and children on different variables using methods generally acceptable for epidemiological studies and with the help of well-trained staff (Chapter 2). The fact that the study is longitudinal is also an advantage; a number of previous studies have been cross-sectional. The definition of childhood obesity (BMI $\geq 95^{th}$ age and sex specific centiles compared to the UK 1990 reference data) in this study is widely used and accepted (Gortmaker et al 1993; Robinson 1993; Himes & Dietz 1994; Troiano et al 1995; Freedman et al 1997; Whitaker et al 1997; Barlow & Dietz 1998; Slyper 1998; Troiano & Flegal 1998; Troiano & Flegal 1999; Zephier et al 1999). This definition of childhood obesity is also fairly sensitive and highly specific (Reilly et al 2000; Chapter 5). Many of the risk factors for childhood obesity presented in this study were being investigated for the first time in modern UK children. In addition, for the first time many of these variables could be analysed with adjustment for social class and for other variables. In some previous studies lack of such adjustment has been a problem (e.g. von Kries et al 1999; Wadsworth et al 1999).

7.8. Limitations of the present study

Since the study was epidemiological, it could only report associations between risk factors and childhood obesity, and not demonstrate cause and effect. Robinson's (1999) study on the effect of TV viewing on childhood obesity is an example of an intervention (controlled trial) study which followed on from studies of association by testing for cause-effect relationships using intervention.

Limitations of the current study are defined in 3 categories:

7.8.1. Variables measured with limitations

There are some risk factors, which were not measured using the ideal method or for which no ideal method exists including:

-**Physical activity** was only identified from parental report of time spent sitting in car or TV viewing. More accurate methods are available. The doubly labeled water method is described as the most accurate method (Goran & Sun 1998), and movement recorders (accelerometers and pedometers or foot-strikes) as the second most accurate method of physical activity measurement (Sallis et al 1990).

-**Snacking:** a food frequency questionnaire was available for the whole cohort. There is some evidence that patterns of food consumption might be obesity risk factors (Johnson & Birch 1994; Drummond et al 1996; Birch & Fisher 1998), but this type of data were not available in this study.

-**Sweetened drink consumption:** a food frequency questionnaire was available and this did not adequately differentiate between diet and sugar sweetened drinks as well as

daily quantities of these drinks. Ideally, analysis of fizzy drink consumption would have focused on sugar sweetened drinks only (Ludwig et al 2001) or fruit juices only (Dennison et al 1997).

-**Social class** as discussed in Chapter 2, the percentage of families in high social class was significantly greater in 'Children in Focus' than the main ALSPAC cohort and compared to Census data for Avon, and also compared to NDNS 1995 (Table 2.5 of Chapter 2). Therefore, the sample was not as representative at age 7 as would be ideal. However, large numbers of families across the range of social class gave the present study considerable power.

-**Parental obesity** was self-reported and dated from before or during pregnancy (in 1990/1991). Self-report has been shown to be less accurate compared to measurement by trained staff (Crawley & Portides 1995; Strauss 1999). Ideally, trained staff should have measured both parents at the same time as the children (when the children were measured) at age 7 years. The level of parental obesity in the present study was low (7.3% men and 5.2% women) when compared to other British studies at around the same time (Jebb 1999 reported prevalence of obesity as 8.0 & 13.0% in men and 12.0 & 16.0% in women in 1987 & 1994). However these were relatively young adults (average age <30 years) and obesity increases with age (Arroyo et al 2000). One of the major difficulties with this data is that only 70% of the fathers answered this question so missing data was a problem.

7.8.2. Unmeasured variables

Obesity is excess body fat (Power et al 1997; Bellizzi & Dietz 1999) and therefore, needs ideally to be identified by a highly accurate method (e.g. a reference method based on a multi-component model; Reilly 1998). Due to epidemiological study limitations, this study used BMI, which is a simple proxy measure of obesity (Dwyer & Blizzard 1996; Hughes et al 1997). However, as mentioned previously, the BMI ≥95th centile definition used is both fairly sensitive and highly specific for high body fatness at age 7. Obesity is an imbalance of energy intake and energy expenditure (Whitaker & Dietz 1998). Therefore, ideally we should have measured both energy intake and energy expenditure with a highly accurate and precise method (e.g. total energy expenditure using the doubly labeled water technique; Schoeller 1999). No measured data on physical activity or energy expenditure were collected by ALSPAC. For some variables, measurement is a problem. For example, it is not clear that any of the dietary assessment methods have sufficient accuracy or precision to detect the small energy imbalance which can cause obesity (Livingstone et al 1990; Livingstone et al 1992; Kaskoun et al 1994; Johnson et al 1996; Lindquist et al 2000; Robson & Livingstone 2000). However, in the present study dietary intake was measured by the three-day unweighed dietary record method (Golding et al 2001) which was probably more accurate than methods used in some other epidemiological studies (Moussa et al 1994; Maffeis et al 2000; Scaglioni et al 2000). Due to the nature of the ALSPAC epidemiological study (large sample size and limited time, equipment, and staff), the dietary assessment method was chosen largely on practical grounds as described in Chapter 2. A number of variables which may be risk factors for obesity are not yet available. These include maternal diabetes and gestational diabetes (Silverman et al 1991; Pettitt et al 1993; Whitaker & Dietz 1998), and urban / rural circumstances (Martorell et al 1998). It will also be important to continue studying these children since ageing is a risk factor for obesity and variables which are associated with obesity at 7 years need to be tested again to find out whether the associations observed continue.

7.8.3. Missing values / representativeness

- Maternal and paternal occupation had many missing values and therefore were not used in this study. Dietary intake data were not obtained for all the study children (only for CIF children) and also was not available after 43 months of age.
- Breast-feeding rate (81.1%) was higher than in other British studies which reported it as 64.0% by age 15 weeks (Martin & White 1988), and 63.1% by age 4 months (Wilson et al 1998). In addition, the timing of the introduction of solids was different between the ALSPAC sample and other UK studies since 73.3% of parents had introduced solids by 12 weeks of age compared to other UK studies which reported it as 90.0% by age 4 months (Martin & White 1988), and 73.6% by age 15 weeks (Wilson et al 1998). This probably reflects the affluent social class profile of ALSPAC compared to the UK population (Chapter 2).
- Ethnic minorities were under represented in ALSPAC (4.1% non-white). The Office of Population Census and Surveys (1993) reported ethnic minorities as 10.0% of British children. Pringle & Rothera (1996) study reported ethnicity as 0.8% in Lincolnshire and 11.1% in Leicester. In the present study, small numbers of non-white children (298 out of 7766) reduced the power of the analyses to detect differences in association with ethnicity. In most parts of the UK, the numbers of minority families are low and therefore, a study with a different design would be needed with over-sampling of ethnic minority children.

7.9. Conclusions

The current study provides information about risk factors for childhood obesity in British children in the late 1990s. Obesity in 7 year old children was defined as having a BMI ≥95th centile compared to UK 1990 standards (Cole et al 1995) and a list of 19 variables including intra-uterine, infant feeding, childhood lifestyle, demographics and other risk factors, were tested for association with childhood obesity. Most UK studies in the literature were concerned with adult obesity, and there was very limited information about childhood obesity risk factors in modern UK children. A few previous studies have tried to address the association between some childhood obesity risk factors (mostly identified differently from the current study) and childhood adiposity / obesity (mostly using different criterion to identify obese children). It is worth noting that some studies took place many years ago (e.g. Poskitt & Cole 1978; Phillips & Young 2000), and the sample size for some were small (Poskitt & Cole 1978; Wilson et al 1998), and many samples were not representative of UK children.

In the present study, from the 19 potential risk factors tested, 4 showed no association with childhood obesity including gestational age, gender, ethnicity, and season of birth. Variables significantly associated were catch-up growth; high birth weight; none or short breast-feeding; the introduction of solids before 12 weeks; energy intake at ages 8 and 18 months, and energy and macronutrient intakes at 43 months; parental education; parental obesity; parental social class; television viewing; time spent in car; fizzy drink consumption; irregular sleeping / short sleep duration. After adjusting for maternal social class, the age of the introduction of solids lost its significance and after adjusting for all other risk factors, 5 factors were significantly associated with childhood obesity including maternal and paternal obesity; birth weight; consumption of fizzy drinks; and time spent in car. However, the current study was the very first attempt to investigate the association between a large number of other risk factors and childhood obesity with correction for confounding variables.

8. Chapter 8: Conclusions

8.1. Background and aims of these studies

Prior to the beginning of this thesis in September 1998, information on childhood obesity worldwide was limited, though increasing concern about it was being expressed in editorials and position statements (summarised in Chapter 1). As an example, agreement on a definition of childhood obesity was only reached in 1998 (Barlow & Dietz 1998), so there was a great deal of confusion over basic questions such as obesity prevalence and how this had changed over time. There were almost no data on childhood obesity prevalence from developing countries, and a widespread perception in the developing world that undernutrition was the only concern for paediatric public health nutrition. There was only limited evidence on the aetiology of childhood obesity, on the validity of the agreed obesity definition based on body mass index (BMI), and on the risk factors for childhood obesity. Three critical periods for the development of adult obesity (infancy, the adiposity rebound, adolescence) had been identified, but it was not clear what factors influenced these critical periods (eg diet, physical activity, genetics).

Agreement on a definition of paediatric obesity made it possible to estimate prevalence for the first time, and so one basic but important aim of this thesis was to estimate paediatric obesity prevalence in the UK and Iran (Chapters 3 and 4). In both countries, prevalence estimates were very high and showed that epidemics of childhood obesity had occurred. The Iranian study was included in the thesis for two main reasons. First, global data on childhood obesity prevalence was scarce (Martorell et al 1998; Martorell et al 2000), and Iran could provide a useful example of a developing country which might act as a model for future research in other developing countries. Second, Iran was in a very special position in

that national reference data based on BMI were available for Iran. When an 'international' definition of childhood obesity became available in 2000 (Cole et al 2000) the existence of Iranian reference data made it possible to compare the two approaches, and again this was intended as a model for other developing countries to illustrate the impact of choosing national or international BMI reference data. The study, described in Chapter 4 showed that choice of reference data can greatly influence obesity prevalence.

In 1998 only limited evidence existed on the validity of the proposed obesity definition based on BMI (>95th centile), so the aim of Chapter 5 was to test the ability (sensitivity, specificity) of this definition to identify the fattest children, using the largest sample size to date (previous studies in other countries had studied fairly small samples).

Risk factors for obesity in the UK today are not well known. The timing of the adiposity rebound (AR; Chapter 6) is believed to be an important childhood risk factor for adult obesity, and the literature contained the suggestion, based on a small study in France, that level of protein intake influenced its timing. The aim of Chapter 6 was therefore to test this hypothesis using data from the Avon Longitudinal Study of Parents and Children (ALSPAC), and to test for other possible influences. The hope was that modifiable risk factors might be identifiable, and these could then be useful in obesity prevention. Chapter 7 extended the analysis of risk factors for obesity in children by evaluating a total of 19 possible risk factors, all supported by a priori hypotheses. Again, this analysis tried to identify the risk factors, assess whether they operated independently of social class, and assess their importance (relative risks). The hope was that identification of modifiable risk factors might help in future efforts to prevent childhood obesity in the UK.

8.2. Synthesis and implications of the findings

The studies in this thesis showed for the first time that an epidemic of childhood obesity occurred in the UK and in Iran during the 1990's. This clearly indicated the need to take childhood obesity seriously as a public health issue, and provided baseline data useful in monitoring obesity trends in the future. Chapter 5 was also useful in establishing the validity of the definition of paediatric obesity recommended by the US expert commmittee in 1998 (Barlow and Dietz 1998), and in showing that the more recent alternative (international) definition (Cole et al 2000) had some serious limitations. This thesis therefore suggests that national BMI reference data should be used to define obesity where they are available, and international data need a more thorough evaluation before they are used. For developing countries, which do not have national BMI reference data, use of the international reference might produce a very misleading picture of the problem, and more caution is needed before adopting this approach.

No clearly modifiable influences on the timing of the AR were identified, but parental obesity was associated with early rebound. In addition, paternal BMI was significantly related to age of AR in girls (not significantly in boys). A number of important risk factors for childhood obesity at age 7 were identified. These might be useful in identifying high risk groups, in understanding the aetiology of the problem, and in designing strategies to prevent childhood obesity. From the 19 risk factors tested in this thesis, 4 (gestational age, female gender, white ethnicity, season of birth) showed no association with childhood obesity but the other 15 variables showed a significant association with childhood obesity. After adjustment for social class, the associaton of timing of introduction of solids was no longer significant. The other factors were also adjusted for all other confounding factors and 5 still remained significantly associated with childhood obesity: maternal and paternal obesity; birth weight; consumption of fizzy drinks; time spent in the car.

8.3. Recommendations for further research

The prevalence data contained in Chapters 3 and 4 show the need to continue to monitor prevalence in the UK and across the world, and the Chapter 5 on methods for screening for obesity shows that future surveys should use the BMI until a better definition is developed. More research on the validity of the international BMI reference (Cole et al 2000) is essential since it is already being widely used.

The Chapter (6) on the factors which influence the AR was limited in that some variables were not measured (eg energy expenditure), and data were only available on children up to the age of 61 months at the time the work was done. This study could be completed easily when additional data become available at older ages (by 90 months). Any future research of this kind should consider the importance of physical activity and energy expenditure, though in epidemiological studies these variables are not easily measured. Since the study described in Chapter 6 found few variables associated with timing of AR, it must be suspected that unmeasured variables were important. The ALSPAC team have planned to increase data collection on phsyical activity as the children go through puberty.

Future research on the main risk factors for obesity, described in Chapter 7, could be used in different ways. It would be possible to target these factors in obesity prevention interventions. It would also be possible to take this information and use it to test for the mechanisms by which obesity risk is increased (eg the effect of sleep pattern, the effect of fizzy drink consumption, the reasons why children from lower social class families are at higher obesity risk and so on).

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10. Appendix

Table A.1. Childhood obesity (BMI ≥95th centile) at age 7 and catch-up growth at 0-2.

Change in weight SDS between 0-2 years		Obese n (%)	Non-obese n (%)	Total n (%)
Boys	Catch-up	21 (18.8)	91 (81.3)	112 (25.4)
	Others	24 (7.3)	303 (92.7)	327 (74.6)
	Total	45 (10.2)	394 (89.8)	439 (54.5)
Girls	Catch-up	14 (13.3)	91 (86.7)	105 (28.5)
	Others	11 (4.2)	251 (95.8)	262 (71.4)
	Total	25 (6.8)	342 (93.2)	367 (45.5)
Total	Catch-up	35 (16.1)	182 (83.9)	217 (26.9)
	Others	35 (5.9)	554 (94.1)	589 (73.1)
	Total	70 (8.7)	736 (91.3)	806 (100.0)

Significant difference between obesity prevalence in catch-up vs. others: $p<0.001$

No significant difference between boys and girls.

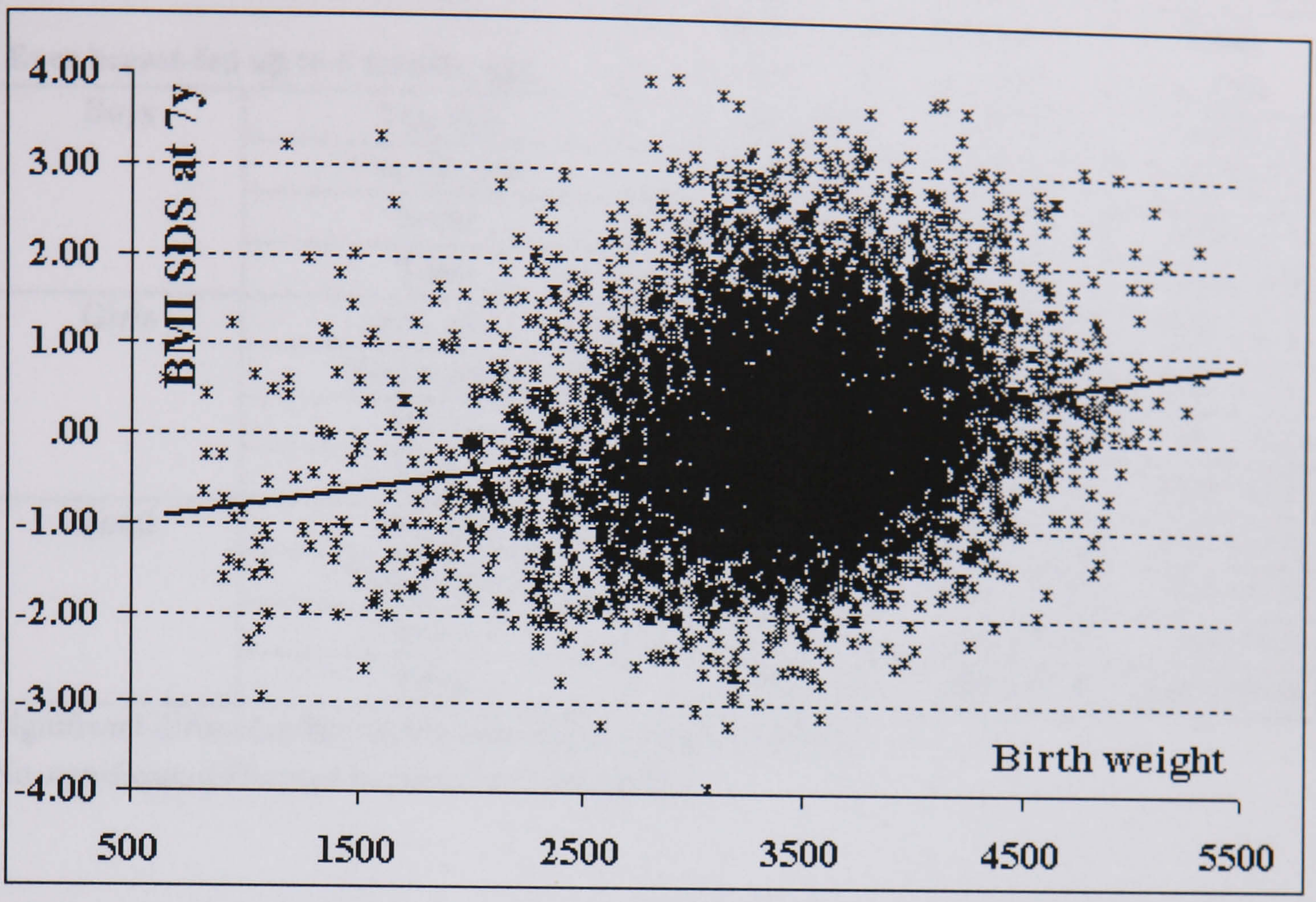
Table A.2. Childhood obesity (BMI ≥95th centile) at age 7 and birth weight.

Birth weight (g)		Obese n (%)	Non-obese n (%)	Total n (%)
Boys	Up to 2499	12 (6.3)	180 (93.8)	192 (4.9)
	2500-3799	200 (7.7)	2408 (92.3)	2608 (66.8)
	≥3800	125 (11.3)	977 (88.7)	1102 (28.2)
	Total	337 (8.6)	3565 (91.4)	3902 (50.8)
Girls	Up to 2499	9 (4.9)	173 (95.1)	182 (4.8)
	2500-3799	202 (7.0)	2665 (93.0)	2867 (76.0)
	≥3800	78 (10.8)	644 (89.2)	722 (19.1)
	Total	289 (7.7)	3482 (92.3)	3771 (49.2)
Total	Up to 2499	21 (5.6)	353 (94.4)	374 (4.9)
	2500-3799	402 (7.3)	5073 (92.7)	5475 (71.4)
	≥3800	203 (11.1)	1621 (88.9)	1824 (23.8)
	Total	626 (8.2)	7047 (91.8)	7673 (100.0)

Significant difference between birth weight of obese and non-obese children, $p<0.001$.

No significant difference between boys and girls.

Figure A.1. Correlation between birth weight and BMI SDS at age 7 years.



r=0.2 P<0.0001 n=7673

Table A.3. Childhood obesity at age 7 and the age of the introduction of solids (asked at 6 months).

Age of introduction of solids (weeks)		**Obese n (%)**	**Non-obese n (%)**	**Total n (%)**
Boys	Up to 12	228 (9.4)	2206 (90.6)	2434 (76.4)
	After 12	51 (6.8)	702 (93.2)	753 (23.6)
	Total	279 (8.8)	2908 (91.2)	3187 (50.7)
Girls	Up to 12	174 (8.0)	2004 (92.0)	2178 (70.2)
	After 12	54 (5.8)	872 (94.2)	926 (29.8)
	Total	228 (7.3)	2876 (92.7)	3104 (49.3)
Total	Up to 12	402 (8.7)	4210 (91.3)	4612 (73.3)
	After 12	105 (6.2)	1574 (93.8)	1679 (26.7)
	Total	507 (8.0)	5784 (92.0)	6291 (100.0)

Significant difference between introduction of solids before 12 weeks and after; p<0.001.

No significant difference between boys and girls.

Table A.4. Childhood obesity (BMI ≥95th centile) at 7 and ever breast-fed at 6 & 15 months.

Ever breast-fed up to 6 months age		Obese n (%)	Non-obese n (%)	Total n (%)
Boys	Yes, still	90 (7.6)	1102 (92.4)	1192 (32.5)
	Yes, stopped	148 (8.4)	1604 (91.6)	1752 (47.8)
	Never	74 (10.3)	647 (89.7)	721 (19.7)
	Total	312 (8.5)	3353 (91.5)	3665 (50.9)
Girls	Yes, still	67 (5.4)	1164 (94.6)	1231 (34.8)
	Yes, stopped	139 (8.3)	1528 (91.7)	1667 (47.1)
	Never	58 (9.0)	584 (91.0)	642 (18.1)
	Total	264 (7.5)	3276 (92.5)	3540 (49.1)
Total	Yes, still	157 (6.5)	2266 (93.5)	2423 (33.6)
	Yes, stopped	287 (8.4)	3132 (91.6)	3419 (47.5)
	Never	132 (9.7)	1231 (90.3)	1363 (18.9)
	Total	576 (8.0)	6629 (92.0)	7205 (100.0)

Significant difference between breast-fed and others; $p<0.002$.

No significant difference between boys and girls.

Table A.5. Childhood obesity (BMI ≥95th centile) at 7 and paternal education.

Partner highest education qualification		Obese n (%)	Normal n (%)	Total n (%)
Boys	CSE	65 (9.2)	643 (90.8)	708 (19.2)
	Vocational	28 (9.8)	259 (90.2)	287 (7.8)
	O level	85 (10.0)	762 (90.0)	847 (23.0)
	A level	85 (8.3)	939 (91.7)	1024 (27.8)
	Degree	45 (5.5)	776 (94.5)	821 (22.3)
	Total	308 (8.4)	3379 (91.6)	3687 (50.8)
Girls	CSE	83 (11.4)	647 (88.6)	730 (20.4)
	Vocational	20 (6.9)	269 (93.1)	289 (8.1)
	O level	53 (6.8)	727 (93.2)	780 (21.8)
	A level	68 (6.8)	936 (93.2)	1004 (28.1)
	Degree	38 (4.9)	735 (95.1)	773 (21.6)
	Total	262 (7.3)	3314 (92.7)	3576 (49.2)
Total	CSE	148 (10.3)	1290 (89.7)	1438 (19.8)
	Vocational	48 (8.3)	528 (91.7)	576 (7.9)
	O level	138 (8.5)	1489 (91.5)	1627 (22.4)
	A level	153 (7.5)	1875 (92.5)	2028 (27.9)
	Degree	83 (5.2)	1511 (94.8)	1594 (21.9)
	Total	570 (7.8)	6693 (92.2)	7263 (100.0)

Significant difference between educational levels: Chi-squared test, $p<0.001$

Significant difference between boys and girls, $p<0.05$

Table A.6. Childhood obesity (BMI ≥95th centile) at 7 and maternal education.

Mums highest education qualification		Obese n (%)	Normal n (%)	Total n (%)
Boys	CSE	53 (10.6)	445 (89.4)	498 (13.1)
	Vocational	38 (11.1)	304 (88.9)	342 (9.0)
	O level	112 (8.1)	1273 (91.9)	1385 (36.4)
	A level	94 (9.5)	892 (90.5)	986 (25.9)
	Degree	25 (4.2)	566 (95.8)	591 (15.5)
	Total	322 (8.5)	3480 (91.5)	3802 (50.9)
Girls	CSE	56 (11.4)	434 (88.6)	490 (13.4)
	Vocational	35 (11.3)	274 (88.7)	309 (8.4)
	O level	89 (7.1)	1172 (92.9)	1261 (34.4)
	A level	60 (6.0)	937 (94.0)	997 (27.2)
	Degree	32 (5.3)	577 (94.7)	609 (16.6)
	Total	272 (7.4)	3394 (92.6)	3666 (49.1)
Total	CSE	109 (11.0)	879 (89.0)	988 (13.2)
	Vocational	73 (11.2)	578 (88.8)	651 (8.7)
	O level	201 (7.6)	2445 (92.4)	2646 (35.4)
	A level	154 (7.8)	1829 (92.2)	1983 (26.6)
	Degree	57 (4.8)	1143 (95.3)	1200 (16.1)
	Total	594 (8.0)	6874 (92.0)	7468 (100.0)

Significant difference between educational levels, $p<0.001$

No significant difference between boys and girls

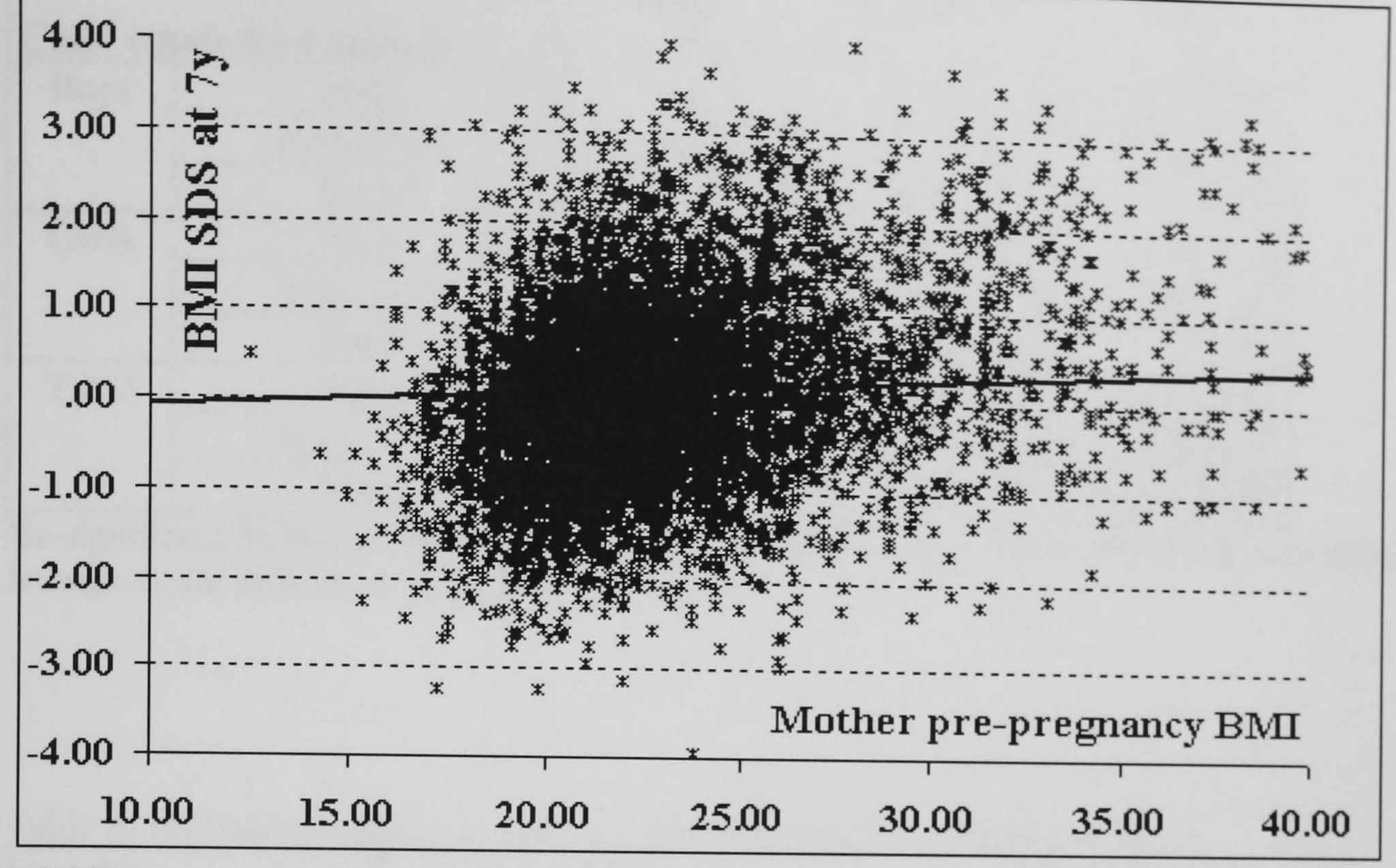
Table A.7. Childhood obesity at age 7 and its association with paternal / maternal obesity.

Child obese Parent obese	Obese n (%)	Non-obese n (%)	Total n (%)
None obese	290 (6.0)	4536 (94.0)	4826 (89.3)
Dad obese	51 (15.9)	269 (84.1)	320 (5.9)
Mum obese	51 (24.5)	157 (75.5)	208 (3.8)
Both obese	23 (46.0)	27 (54.0)	50 (0.9)
Total	415 (7.7)	4989 (92.3)	5404 (100.0)

Significant difference between obese parents and not-obese parents; $p<0.0001$

No significant difference between boys and girls

Figure A.2. Correlation between mother pre-pregnancy BMI and BMI SDS at age 7 years.



r=0.274 p<0.0001 n=6996

Table A.8. Childhood obesity at age 7 and parental social class.

Social class			Obese n (%)	Non-obese n (%)	Total n (%)
Father	**Boys**	1, 2, 3M	150 (7.3)	1900 (92.7)	2050 (61.1)
		3N, 4, 5	124 (9.5)	1182 (90.5)	1306 (38.9)
		Total	274 (8.2)	3082 (91.8)	3356 (50.5)
	Girls	1, 2, 3M	130 (6.5)	1861 (93.5)	1991 (60.5)
		3N, 4, 5	107 (8.2)	1194 (91.8)	1301 (39.5)
		Total	237 (7.2)	3055 (92.8)	3292 (49.5)
	Total	1, 2, 3M	280 (6.9)	3761 (93.1)	4041 (60.8)
		3N, 4, 5	231 (8.9)	2376 (91.1)	2607 (39.2)
		Total	511 (7.7)	6137 (92.3)	6648 (100.0)
Mother	**Boys**	1, 2, 3M	207 (8.1)	2360 (91.9)	2567 (83.1)
		3N, 4, 5	59 (11.3)	463 (88.7)	522 (16.9)
		Total	266 (8.6)	2823 (91.4)	3089 (51.0)
	Girls	1, 2, 3M	168 (6.8)	2290 (93.2)	2458 (82.8)
		3N, 4, 5	49 (9.6)	462 (90.4)	511 (17.2)
		Total	217 (7.3)	2752 (92.7)	2969 (49.0)
	Total	1, 2, 3M	375 (7.5)	4650 (92.5)	5025 (82.9)
		3N, 4, 5	108 (10.4)	925 (89.6)	1033 (17.1)
		Total	483 (8.0)	5575 (92.0)	6058 (100.0)

Significant difference: Chi-squared test, p<0.005
No significant difference between boys and girls

Table A.9. Childhood obesity at age 7 and ethnic background (asked at 18w gestation).

Child ethnic background		Obese n (%)	Non-obese n (%)	Total n (%)
Boys	White	299 (8.3)	3292 (91.7)	3591 (96.1)
	Non-white	17 (11.6)	129 (88.4)	146 (3.9)
	Total	316 (8.5)	3421 (91.5)	3737 ()
Girls	White	253 (7.3)	3211 (92.7)	3464 (95.8)
	Non-white	14 (9.2)	138 (90.8)	152 (4.2)
	Total	267 (7.4)	3349 (92.6)	3616 ()
Total	White	552 (7.8)	6503 (92.2)	7055 (95.9)
	Non-white	31 (10.4)	267 (89.6)	298 (4.1)
	Total	583 (7.9)	6770 (92.1)	7353 (100.0)

No significant difference in prevalence of obesity was found between whites and non-whites.
No significant difference between boys and girls.

Table A.10. Nutrient intake at age 8 months and childhood obesity at age 7.

Variable at age 18 months		Obese			Non-obese		
		Boys	girls	Total	boys	Girls	Total
Fat (g/kg/day)	**Mean**	3.4	3.6	3.5	3.7	3.8	3.7
	SD	0.8	1.1	0.9	1.0	1.2	1.1
	N	47	26	73	415	358	773
Protein (g/kg/day)	**Mean**	2.9	3.1	3.0	3.1	3.1	3.1
	SD	0.9	1.0	0.9	0.9	1.0	1.0
	N	47	26	73	415	358	773
Carbohydrate (g/kg/day)	**Mean**	11.5	11.7	11.6	12.2	12.2	12.2
	SD	2.7	3.2	2.9	2.6	2.7	2.7
	N	47	26	73	415	358	773
Energy kcal/kg/day	**Mean**	86.0	89.1	*87.1	92.0	92.4	92.2
	SD	17.6	22.6	19.4	19.5	21.5	20.4
	N	47	26	73	415	358	773
Energy kJ/kg/day	**Mean**	359	372	*364	385	386	385
	SD	73	94	81	81	90	85
	N	47	26	73	415	358	773
Energy % from fat	**Mean**	34.8	35.4	35.0	35.1	35.4	35.3
	SD	5.1	5.4	5.2	4.8	5.2	5.0
	N	47	26	73	415	358	773
Energy % from protein	**Mean**	13.2	13.5	13.3	13.2	12.9	13.0
	SD	2.7	2.6	2.7	2.5	2.6	2.6
	N	47	26	73	415	358	773
Energy % from CHO	**Mean**	52.0	51.1	51.7	51.7	51.7	51.7
	SD	5.7	6.1	5.9	5.4	5.7	5.5
	N	47	26	73	415	358	773

*Significant difference between obese and others, $p<0.05$
No significant differences between boys and girls.

Table A.11. Nutrient intake at age 18 months and childhood obesity at age 7.

Variable at age 18 monhts		Obese			Non-obese		
		Boys	girls	Total	boys	Girls	Total
Fat (g/kg/day)	Mean	4.0	3.8	4.0	4.1	4.1	4.1
	SD	0.8	1.2	1.0	1.0	1.1	1.1
	N	47	24	71	369	320	689
Protein (g/kg/day)	Mean	3.5	3.3	3.4	3.6	3.6	3.6
	SD	0.8	1.0	0.9	0.9	0.9	0.9
	N	47	24	71	369	320	689
Carbohydrate (g/kg/day)	Mean	10.9	10.8	*10.9	12.2	11.9	12.1
	SD	2.3	2.6	2.4	2.8	2.9	2.9
	N	47	24	71	369	320	689
Energy kcal/kg/day	Mean	91.0	88.2	**90.1	97.4	96.2	96.9
	SD	16.1	22.3	18.3	19.8	21.4	20.5
	N	47	24	71	369	320	689
Energy kJ/kg/day	Mean	382	370	**378	409	404	407
	SD	68	93	77	83	90	86
	N	47	24	71	369	320	689
Energy % from fat	Mean	38.7	37.3	38.3	36.6	37.0	36.8
	SD	3.8	3.8	3.9	4.7	4.8	4.8
	N	47	24	71	369	320	689
Energy % from protein	Mean	14.8	14.6	14.7	14.5	14.7	14.6
	SD	2.3	2.1	2.2	2.2	2.3	2.2
	N	47	24	71	369	320	689
Energy % from CHO	Mean	46.5	48.1	47.0	48.8	48.3	48.6
	SD	4.9	5.3	5.1	5.7	5.7	5.7
	N	47	24	71	369	320	689

Significant difference between obese and others; * $p<0.001$ ** $p<0.01$

No significant differences between boys and girls.

Table A.12. Nutrient intake at age 43 months and childhood obesity at age 7.

Variable at age 43 months		Obese			Non-obese		
		Boys	girls	Total	boys	Girls	total
Fat (g/kg/day)	Mean	3.0	2.8	*2.9	3.5	3.5	3.5
	SD	0.8	0.8	0.8	0.9	0.8	0.9
	N	39	22	61	342	284	626
Protein (g/kg/day)	Mean	2.5	2.5	*2.5	3.0	2.8	2.9
	SD	0.7	0.7	0.7	0.7	0.7	0.7
	N	39	22	61	342	284	626
Carbohydrate (g/kg/day)	Mean	9.8	8.7	*9.4	11.2	10.9	11.1
	SD	2.4	2.3	2.4	2.1	2.1	2.1
	N	39	22	61	342	284	626
Energy kcal/kg/day	Mean	73.7	67.8	*71.6	86.0	83.2	84.7
	SD	15.1	15.9	15.5	15.8	15.6	15.7
	N	39	22	61	342	284	626
Energy kJ/kg/day	Mean	310	285	*301	361	350	356
	SD	63	66	65	66	66	66
	N	39	22	61	342	284	626
Energy % from fat	Mean	34.8	36.2	35.3	35.8	36.0	35.9
	SD	5.2	4.5	4.9	4.6	4.3	4.5
	N	39	23	62	342	284	626
Energy % from protein	Mean	13.5	13.9	13.6	13.4	13.3	13.4
	SD	2.7	1.8	2.4	2.3	2.4	2.3
	N	39	23	62	342	284	626
Energy % from CHO	Mean	51.7	49.9	51.0	50.8	50.7	50.8
	SD	6.0	5.6	5.9	5.3	4.7	5.0
	N	39	23	62	342	284	626

*Significant difference between obese and others, $p<0.001$

No significant differences between boys and girls.

Table A.13. Childhood obesity at age 7 and season of birth.

Month of delivery		Obese n (%)	Non-obese n (%)	Total N (%)
Male	Jan-Mar	54 (9.0)	543 (91.0)	597 (15.2)
	Apr-Jun	95 (8.9)	975 (91.1)	1070 (27.2)
	July-Sep	92 (7.6)	1126 (92.4)	1218 (31.0)
	Oct-Doc	97 (9.3)	945 (90.7)	1042 (26.5)
	Total	338 (8.6)	3589 (91.4)	3927 (50.9)
Female	Jan-Mar	48 (8.1)	546 (91.9)	594 (15.7)
	Apr-Jun	69 (6.7)	966 (93.3)	1035 (27.3)
	July-Sep	90 (8.0)	1029 (92.0)	1119 (29.5)
	Oct-Doc	82 (7.8)	964 (92.2)	1046 (27.6)
	Total	289 (7.6)	3505 (92.4)	3794 (49.1)
Total	Jan-Mar	102 (8.6)	1089 (91.4)	1191 (15.4)
	Apr-Jun	164 (7.8)	1941 (92.2)	2105 (27.3)
	July-Sep	182 (7.8)	2155 (92.2)	2337 (30.3)
	Oct-Doc	179 (8.6)	1909 (91.4)	2088 (27.0)
	Total	627 (8.1)	7094 (91.9)	7721 (100.)

No significant difference in prevalence of obesity was found between seasons.
No significant difference between boys and girls was encountered.

Table A.14. Childhood obesity at 7 and time spent watching TV at 38 months.

Time spent watching TV weekday		Obese n (%)	Non-obese n (%)	Total N (%)
Boys	Not at all	5 (1.7)	70 (2.2)	75 (2.1)
	<1 hour	61 (20.5)	864 (26.9)	925 (26.3)
	1-2 hours	139 (46.8)	1511 (47.0)	1650 (47.0)
	>2 hours	92 (31.0)	772 (24.0)	864 (24.6)
	Total	297 (8.4)	3217 (91.6)	3514 (51.1)
Girls	Not at all	4 (1.6)	80 (2.6)	84 (2.5)
	<1 hour	59 (23.4)	927 (29.8)	986 (29.3)
	1-2 hours	116 (46.0)	1455 (46.7)	1571 (46.7)
	>2 hours	73 (29.0)	653 (21.0)	726 (21.6)
	Total	252 (7.5)	3115 (92.5)	3367 (48.9)
Total	Not at all	9 (1.6)	150 (2.4)	159 (2.3)
	<1 hour	120 (21.9)	1791 (28.3)	1911 (27.8)
	1-2 hours	255 (46.4)	2966 (46.8)	3221 (46.8)
	>2 hours	165 (30.1)	1425 (22.5)	1590 (23.1)
	Total	549 (8.0)	6332 (92.0)	6881 (100.0)

Significant difference in prevalence of obesity between those who did not watch TV and others, $p<0.001$. No significant difference between boys and girls.

Table A.15. Childhood obesity at 7 and time spent in car at 38 months.

Time spent in car in weekday		Obese n (%)	Non-obese n (%)	Total N (%)
Boys	Not at all	19 (6.4)	288 (8.9)	307 (8.7)
	<1 hour	238 (80.1)	2583 (80.0)	2821 (80.0)
	1-2 hours	32 (10.8)	341 (10.6)	373 (10.6)
	>2 hours	8 (2.7)	18 (0.6)	26 (0.7)
	Total	297 (8.4)	3230 (91.6)	3527 (51.2)
Girls	Not at all	20 (8.0)	243 (7.8)	263 (7.8)
	<1 hour	199 (79.9)	2525 (81.0)	2724 (80.9)
	1-2 hours	26 (10.4)	322 (10.3)	348 (10.3)
	>2 hours	4 (1.6)	27 (0.9)	31 (0.9)
	Total	249 (7.4)	3117 (92.6)	3366 (48.8)
7Total	Not at all	39 (6.8)	531 (93.2)	570 (8.3)
	<1 hour	437 (7.9)	5108 (92.1)	5545 (80.4)
	1-2 hours	58 (8.0)	663 (92.0)	721 (10.5)
	>2 hours	12 (21.1)	45 (78.9)	57 (0.8)
	Total	546 (7.9)	6347 (92.1)	6893(100.0)

Significant difference in childhood obesity between those who did not use car and others, $p<0.005$. No significant difference between boys and girls

Table A.16. Childhood obesity at 7 and consumption of fizzy drinks / week at 38 months.

Time a week child had other Fizzy drink		Obese n (%)	Non-obese n (%)	Total N (%)
Boys	Never	101 (6.5)	1455 (93.5)	1556 (44.4)
	Once in 2 weeks	69 (8.4)	753 (91.6)	822 (23.4)
	1-3 times a week	89 (11.2)	709 (88.8)	798 (22.8)
	4-7 times a week	26 (10.9)	212 (89.1)	238 (6.8)
	>once a day	7 (7.5)	86 (92.5)	93 (2.7)
	Total	292 (8.3)	3215 (91.7)	3507 (51.2)
Girls	Never	88 (6.0)	1375 (94.0)	1463 (43.8)
	Once in 2 weeks	45 (5.7)	741 (94.3)	786 (23.5)
	1-3 times a week	78 (10.1)	697 (89.9)	775 (23.2)
	4-7 times a week	33 (13.4)	214 (86.6)	247 (7.4)
	>once a day	4 (5.6)	67 (94.4)	71 (2.1)
	Total	248 (7.4)	3094 (92.6)	3342 (48.8)
Total	Never	189 (6.3)	2830 (93.7)	3019 (44.1)
	Once in 2 weeks	114 (7.1)	1494 (92.9)	1608 (23.5)
	1-3 times a week	167 (10.6)	1406 (89.4)	1573 (23.0)
	4-7 times a week	59 (12.2)	426 (87.8)	485 (7.1)
	>once a day	11 (6.7)	153 (93.3)	164 (2.4)
	Total	540 (7.9)	6309 (92.1)	6849 (100.0)

Significant difference in childhood obesity between those who drink once or more fizzy drink in a week and others, $p<0.001$. No significant difference between boys and girls.

Table A.17. Childhood obesity at age 7 years and sleep routine at ages 30 and 42 months.

Child regular sleep routine			Obese n (%)	Non-obese N (%)	Total n (%)
Age 30 months	**Boys**	No	43 (12.0)	314 (88.0)	357 (10.0)
		Yes	255 (8.0)	2925 (92.0)	3180 (90.0)
		Total	299 (8.4)	3268 (91.6)	3567 (51.0)
	Girls	No	30 (9.7)	278 (90.3)	308 (9.0)
OR = 1.51 (1.16 - 1.98)		Yes	217 (7.0)	2864 (93.0)	3081 (91.0)
		Total	254 (7.4)	3167 (92.6)	3421 (49.0)
RR = 1.46 (1.15 - 1.84)	**Total**	No	73 (11.0)	592 (89.0)	665 (9.5)
		Yes	472 (7.5)	5789 (92.5)	6261 (90.5)
		Total	553 (7.9)	6435 (92.1)	6988 (100.0)
Age 42 months	**Boys**	No	27 (11.6)	206 (88.4)	233 (6.6)
		Yes	270 (8.2)	3022 (91.8)	3292 (93.4)
		Total	297 (8.4)	3228 (91.6)	3525 (51.3)
	Girls	No	26 (11.1)	208 (88.9)	234 (7.0)
OR = 1.54 (1.13 - 2.10)		Yes	222 (7.1)	2888 (92.9)	3110 (93.0)
		Total	248 (7.4)	3096 (92.6)	3344 (48.7)
RR = 1.48 (1.13 - 1.93)	**Total**	No	53 (11.3)	414 (88.7)	467 (6.8)
		Yes	492 (7.7)	5910 (92.3)	6402 (93.2)
		Total	545 (7.9)	6324 (92.1)	6869 (100.0)

*Significant difference in childhood obesity between those who had regular sleep routine and others (at 30 & 42 months of age); p<0.005. No significant difference between boys and girls.

Table A.18. Childhood obesity at age 7 years and sleep duration at ages 30 and 42 months.

Variable		Obese			Non-obese			Total		
		boys	girls	total	boys	girls	total	Boys	girls	total
Sleep at age 30 months	**Mean**	10.9	11.2	11.0	11.2	11.3	11.2	11.2	[illegible]	[illegible]
	SD	1.05	1.09	1.08	1.02	1.00	1.01	1.03	1.01	[illegible]
	N	291	246	537	3195	3100	6295	[illegible]	[illegible]	[illegible]
Sleep at age 42 months	**Mean**	11.0	11.2	11.1	11.2	11.3	11.3	11.2	11.3	[illegible]
	SD	0.94	0.90	0.92	0.83	0.84	0.84	0.84	[illegible]	[illegible]
	N	295	246	541	3200	3074	6274	[illegible]	[illegible]	[illegible]

Significant difference in mean sleep duration at ages between obese and non-obese children at ages 30 and 42 months.

Significant difference between mean sleep duration at ages 30 and 42 months among boys and girls.

Table A.19. Summary of associations between childhood obesity risk factors and childhood obesity at age 7 before & after adjustment (levels 1-3).

Group	Variable		Total number	Before adjustment (level 1)			After adjustment for *maternal social class* (level 2)			After adjustment for *All other risk factors* (level 3)		
				OR	95% CI	Sig.	OR	95% CI	Sig.	OR	95% CI	Sig.
Intra-Uterine*	**Catch-up growth** vs. non catch-up		806	3.04	1.80-5.15	0.001	-	-	-	-	-	-
	Birth Weight	2500-3800 vs. <2500	7673	1.33	0.85-2.09	0.000	1.55	0.88-2.73	0.000	1.59	0.82-3.09	0.012
		>3800 vs. 2500-3800		1.58	1.32-1.89		1.46	1.19-1.80		1.36	1.06-1.73	
Infant feeding / weaning	**Breast fed**	<6 month	7205	1.32	1.08-1.62	0.002	1.26	1.00-1.58	0.030	0.99	0.76-1.28	0.820
	Vs. up to 6 month	None		1.55	1.22-1.97		1.45	1.09-1.92		0.91	0.65-1.27	
	Introduction of solids Before 12 vs. after 12 weeks		6291	1.43	1.14-1.79	0.002	1.25	0.98-1.60	0.070	-	-	-
Demographics / other variables†	**Mother Education**	None/CSE	7468	2.48	1.78-3.46	0.000	2.11	1.35-3.28	0.000	1.69	0.96-2.97	0.264
		Vocational		2.53	1.76-3.62		2.39	1.52-3.76		1.68	0.94-2.95	
		O level		1.64	1.22-2.23		1.47	1.00-2.19		1.27	0.78-2.05	
	Vs. Degree	A level		1.68	1.23-2.30		1.50	1.02-2.20		1.36	0.86-2.15	
	Partner education	None/CSE	7263	2.09	1.58-2.76	0.000	1.62	1.16-2.25	0.04	1.26	0.82-1.93	0.549
		Vocational		1.65	1.14-2.39		1.50	1.00-2.26		1.12	0.68-1.84	
		O level		1.68	1.27-2.23		1.38	1.00-1.90		1.28	0.87-1.90	
	vs. Degree	A level		1.48	1.13-1.95		1.18	0.86-1.61		1.03	0.71-1.50	
	Obese parent (BMI >30)	obese father	7004	2.57	1.88-3.51	0.000	2.67	1.88-3.78	0.000	2.64	1.79-3.90	0.000
		obese mother		4.40	3.16-6.12		4.43	3.04-6.44		4.98	3.33-7.44	
	vs. non-obese	Both obese		11.5	6.56-0.28		12.3	6.55-23.2		9.86	4.68-20.8	
	Mother social class	II	6058	1.87	1.05-3.33	0.02	-		-	1.48	0.72-3.02	0.399
		IIIM		1.89	1.07-3.35					1.16	0.56-2.42	
		IIIN		2.74	1.45-5.16					1.56	0.69-3.53	
		IV		2.55	1.37-4.76					1.42	0.64-3.17	
	vs. class I	V		2.69	1.14-6.35					1.60	0.51-5.01	
	Partner social class	II	6648	1.50	1.06-2.12	0.01	1.33	0.91-1.93	0.16	-		-
		IIIM		1.62	1.09-2.41		1.39	0.90-2.15				
		IIIN		1.82	1.29-2.57		1.56	1.06-2.30				
		IV		2.03	1.32-3.10		1.82	1.14-2.91				
	vs. class I	V		1.62	0.85-3.08		1.56	0.80-3.04				
Childhood lifestyle‡	**Watch TV**	1-2 hours	6881	1.29	1.04-1.61	0.000	1.16	0.90-1.48	0.003	1.01	0.77-1.32	0.469
	vs. <1 hour	>2 hours		1.74	1.37-2.21		1.56	1.19-2.05		1.18	0.86-1.60	
	Spent in car	1-2 hours	6881	1.04	0.78-1.38	0.002	1.03	0.74-1.41	0.002	0.97	0.68-1.39	0.005
	vs. <1 hour	>2 hours		3.16	1.66-6.01		3.46	1.72-6.95		3.66	1.67-8.00	
	Fizzy drink once to ≥7 times / week vs. none / once per 2 weeks		6849	1.70	1.43-2.04	0.000	1.62	1.32-1.98	0.010	1.57	1.25-1.97	0.000
	Irregular sleep at 42 months vs. regular		6869	1.54	1.14-2.08	0.005	1.47	1.03-2.08	0.030	1.24	0.82-1.87	0.303

* Gestational age had no association to childhood obesity at age 7.

† Gender, ethnicity, and season of birth showed no association to childhood obesity at age 7.

‡ Macronutrient intakes at ages 8, 18, and 43 months was associated to childhood obesity at age 7 but due to small sample size was not adjusted.

Table A.20. Prevalence of childhood obesity in the subjects who had the values for 12 adjusted variables, and in those who had missing values.

Risk factor	Options	Present in study	Missing from study	Total	Sig.
Childhood obesity	Obese	262 (7.4)	369 (8.8)	631 (8.1)	0.023
	Non-obese	3299 (92.6)	3836 (91.2)	7135 (91.9)	
	Total	3561 (45.8)	4205 (54.2)	7766 (100.)	
TV viewing	0 to <1	1129 (31.6)	946 (28.4)	2075 (30.1)	0.010
	1-2	1648 (46.2)	1579 (47.5)	3227 (46.8)	
	>2	791 (22.2)	802 (24.1)	1593 (23.1)	
	Total	3568 (51.7)	3327 (48.3)	6895 (88.8)	
Ever breast-fed	Yes, still	1255 (35.2)	1173 (32.1)	2428 (33.6)	0.000
	Yes, stopped	1708 (47.9)	1718 (47.0)	3426 (47.4)	
	Never	605 (17.0)	762 (20.9)	1367 (18.9)	
	Total	3568 (49.4)	3653 (50.6)	7221 (93.0)	
Mother highest education	None/CSE	299 (8.4)	694 (17.7)	993 (13.3)	0.000
	Vocational	273 (7.7)	378 (9.7)	651 (8.7)	
	O level	1353 (37.9)	1297 (33.1)	2650 (35.4)	
	A level	1025 (28.7)	964 (24.6)	1989 (26.6)	
	Degree	618 (17.3)	583 (14.9)	1201 (16.0)	
	Total	3568 (47.7)	3916 (52.3)	7484 (96.4)	
Partner highest education	None/CSE	491 (13.8)	952 (25.7)	1443 (19.8)	0.000
	Vocational	249 (7.0)	328 (8.8)	577 (7.9)	
	O level	842 (23.6)	789 (21.3)	1631 (22.4)	
	A level	1127 (31.6)	906 (24.4)	2033 (27.9)	
	Degree	859 (24.1)	736 (19.8)	1595 (21.9)	
	Total	3568 (49.0)	3711 (51.0)	7279 (93.7)	
Maternal social class	I	214 (6.0)	95 (3.8)	309 (5.1)	0.000
	II	1266 (35.5)	748 (29.8)	2014 (33.2)	
	III	1578 (44.2)	1138 (45.4)	2716 (44.7)	
	IV	208 (5.8)	219 (8.7)	427 (7.0)	
	V	267 (7.5)	246 (9.8)	513 (8.4)	
	VI	35 (1.0)	59 (2.4)	94 (1.5)	
	Total	3568 (58.8)	2505 (41.2)	6073 (78.2)	
Regular sleep routine at 42 months of age	Yes	3385 (94.9)	3031 (91.4)	6416 (93.2)	0.000
	No	183 (5.1)	286 (8.6)	469 (6.8)	
	Total	3568 (51.8)	3317 (48.2)	6885 (88.7)	

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