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**UNIVERSITY OF SOUTHAMPTON**  
**FACULTY OF MEDICINE, HEALTH AND LIFE SCIENCES**  
**School of Medicine**

**Adiposity in British secondary school children:  
A population based study**

by

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Thesis for the degree of Doctor of Medicine

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## CONTENTS

<b>TABLES.....</b>	<b>5</b>
<b>FIGURES.....</b>	<b>7</b>
<b>ACKNOWLEDGEMENTS.....</b>	<b>9</b>
<b>ABBREVIATIONS .....</b>	<b>10</b>
<b>CHAPTER 1: INTRODUCTION .....</b>	<b>11</b>
1.0 SUMMARY .....	11
1.1 INTRODUCTION.....	12
1.2 ADIPOSITY – DEFINITIONS AND MEASUREMENT .....	12
1.2.1 DEFINING INCREASED LEVELS OF ADIPOSITY.....	13
1.3 THE IMPORTANCE OF ADIPOSITY .....	14
1.3.1 TRACKING OF ADIPOSITY FROM CHILDHOOD TO ADULT LIFE.....	15
1.3.2 RISING LEVELS OF ADIPOSITY.....	16
1.4 FACTORS ASSOCIATED WITH ADIPOSITY .....	16
1.4.1 REGIONAL, SOCIAL AND ETHNIC FACTORS .....	16
1.4.2 INDIVIDUAL DETERMINANTS OF ADIPOSITY.....	17
1.4.3 AVAILABLE INFORMATION ON THE DETERMINANTS OF ADIPOSITY IN YOUNG BRITISH PEOPLE .....	18
1.5 OVERALL AIMS OF THIS THESIS .....	18
<b>CHAPTER 2: BACKGROUND.....</b>	<b>20</b>
2.0 SUMMARY .....	20
2.1 INTRODUCTION.....	20
2.2 MEASURING AND DEFINING ADIPOSITY, OVERWEIGHT AND OBESITY .....	20
2.2.1 MEASURES OF GENERAL ADIPOSITY.....	21
2.2.2 MEASURES OF CENTRAL ADIPOSITY .....	26
2.2.3 OTHER MEASURES OF ADIPOSITY.....	27
2.3 EVIDENCE FOR INCREASING LEVELS OF ADIPOSITY .....	28
2.3.1 EVIDENCE FOR INCREASING LEVELS OF ADIPOSITY IN ADULTS .....	28
2.3.2 EVIDENCE FOR INCREASING LEVELS OF ADIPOSITY IN CHILDHOOD.....	30
2.4 VARIATIONS IN ADIPOSITY DISTRIBUTION.....	31
2.4.1 REGIONAL VARIATIONS IN ADIPOSITY .....	31
2.4.2 SOCIAL VARIATIONS IN ADIPOSITY.....	33
2.4.3 ETHNIC VARIATIONS IN ADIPOSITY .....	36
2.5 INDIVIDUAL DETERMINANTS OF INCREASED ADIPOSITY IN CHILDHOOD .....	37
2.5.1 INTRODUCTION: INDIVIDUAL DETERMINANTS OF ADIPOSITY.....	37
2.5.2 PHYSICAL ACTIVITY .....	38
2.5.3 EARLY LIFE EXPOSURES .....	40
2.5.4 FAMILIAL FACTORS .....	43
2.6 CONSEQUENCES OF OBESITY .....	46
2.6.1 ADVERSE PROFILE OF CARDIOVASCULAR FACTORS IN CHILDHOOD AND ADOLESCENCE WITH ADIPOSITY.....	47
2.7 CONCLUSION .....	48
<b>CHAPTER 3: DESIGN AND METHODOLOGY OF THE TEN TOWNS STUDY.....</b>	<b>50</b>
3.0 SUMMARY .....	50
3.1 INTRODUCTON: RATIONALE FOR THE TEN TOWNS STUDY .....	50
3.1.1 SELECTION OF THE TOWNS.....	50
3.1.2 SELECTION OF THE SCHOOLS AND PARTICIPANTS.....	51
3.1.3 STATISTICAL POWER CONSIDERATIONS.....	52

3.2 SURVEY PROCEDURES 1998-9 .....	52
3.2.1 PHYSICAL MEASUREMENTS.....	53
3.2.2 BLOOD SAMPLING.....	55
3.2.3 EXERCISE TESTING/ SPIROMETRY.....	56
3.2.4 HANDLING OF ABNORMAL BLOOD PRESSURE RESULTS.....	56
3.2.5 OTHER INFORMATION.....	56
3.3 QUESTIONNAIRE INFORMATION .....	57
3.3.1 PUPIL QUESTIONNAIRE .....	57
3.3.2 PARENTAL QUESTIONNAIRE .....	57
3.3.3 PUBERTAL STATUS.....	58
3.4 THE DATASETS USED IN THIS THESIS .....	58
3.5 DERIVED BODY BUILD AND BODY COMPOSITION MEASURES.....	58
3.6 STATISTICAL METHODS.....	59
<b>CHAPTER 4: BODY SIZE AND ADIPOSITY IN 13-16 YEAR OLDS RELATIONS TO AGE, GENDER AND PUBERTAL STATUS.....</b>	<b>65</b>
4.0 SUMMARY POINTS .....	65
4.1 INTRODUCTION AND OBJECTIVES .....	66
4.2 SUBJECTS AND METHODS .....	67
4.2.1 STATISTICAL METHODS.....	67
4.3 RESULTS.....	68
4.3.1 EXPLORATION OF THE CHARACTERISTICS AND DISTRIBUTION OF BODY SIZE AND ADIPOSITY MEASURES.....	68
4.3.2 EFFECT OF OBSERVER ON THE BODY SIZE MEASUREMENTS .....	68
4.3.3 EFFECT OF GENDER ON THE BODY SIZE MEASUREMENTS .....	69
4.3.4 EFFECT OF AGE ON THE BODY SIZE MEASUREMENTS .....	69
4.3.5 RELATIONS OF PUBERTAL STAGE TO BODY SIZE AND ADIPOSITY MEASUREMENTS.....	70
4.3.6 THE INTERRELATIONSHIPS OF DIFFERENT MEASURES OF ADIPOSITY .....	72
4.3.7 COMPARISON OF THE TEN TOWNS STUDY CHILDREN WITH PROPOSED INTERNATIONAL STANDARDS FOR OBESITY .....	72
4.4 DISCUSSION OF RESULTS .....	73
4.4.1 PRINCIPAL FINDINGS .....	73
4.4.2 STRENGTHS AND WEAKNESSES OF THE STUDY .....	74
4.4.3 RELATION OF FINDINGS TO OTHER STUDIES.....	74
4.5 CONCLUSIONS AND IMPLICATIONS FOR AETIOLOGY AND PREVENTION .....	79
<b>CHAPTER 5: RELATIONS OF GEOGRAPHIC LOCATION (TOWN), SOCIAL CLASS AND ETHNIC GROUP TO BODY SIZE AND ADIPOSITY MEASURES IN 13-16 YEAR-OLDS .....</b>	<b>103</b>
5.0 SUMMARY POINTS .....	103
5.1 INTRODUCTION .....	103
5.2 AIMS AND OBJECTIVES .....	104
5.3 SUBJECTS AND METHODS .....	104
5.3.1 STATISTICAL METHODS.....	105
5.4 RESULTS.....	106
5.4.1 NUMBERS OF PARTICIPANTS IN DIFFERENT TOWNS, SOCIAL CLASS GROUPS AND ETHNIC GROUPS.....	106
5.4.2 TOWN DIFFERENCES IN BODY SIZE AND ADIPOSITY .....	107
5.4.3 SOCIAL CLASS DIFFERENCES IN ADIPOSITY .....	108
5.4.4 ETHNIC DIFFERENCES IN ADIPOSITY .....	110
5.5 DISCUSSION .....	111
5.5.1 PRINCIPAL FINDINGS .....	111
5.5.2 STRENGTHS AND WEAKNESSES OF THE STUDY .....	111
5.5.3 RELATION OF THE FINDINGS TO OTHER STUDIES .....	112
5.5.4 ETHNICITY AND ADIPOSITY MEASUREMENTS .....	115
5.6 IMPLICATIONS FOR AETIOLOGY AND PREVENTION .....	117

<b>CHAPTER 6: THE RELATIONS OF PARENTAL ADIPOSITY, INFANT FEEDING AND PHYSICAL ACTIVITY TO ADIPOSITY IN 13-16 YEAR OLDS .....</b>	<b>126</b>
6.0 SUMMARY POINTS .....	126
6.1 INTRODUCTION .....	127
6.1.1 PARENTAL ADIPOSITY .....	127
6.1.2 BREAST FEEDING AND ADIPOSITY .....	128
6.1.3 PHYSICAL ACTIVITY AND ADIPOSITY .....	128
6.2 AIMS AND OBJECTIVES .....	129
6.3 SUBJECTS AND METHODS .....	129
6.3.1 STATISTICAL METHODS .....	130
6.4 RESULTS .....	131
6.4.1 PARENTAL BMI AND ADIPOSITY IN ADOLESCENCE .....	131
6.4.2 INFANT FEEDING AND ADIPOSITY .....	132
6.4.3 ADIPOSITY, EXERCISE HABITS AND LEISURE ACTIVITIES .....	133
6.5 DISCUSSION .....	135
6.5.1 PRINCIPAL FINDINGS .....	135
6.5.2 PARENTAL ADIPOSITY .....	136
6.5.3 INFANT FEEDING .....	137
6.5.4 PHYSICAL ACTIVITY .....	139
6.6 CONCLUSIONS AND IMPLICATIONS FOR AETIOLOGY AND PREVENTION .....	140
6.6.1 PARENTAL ADIPOSITY .....	140
6.6.2 BREAST FEEDING .....	141
6.6.3 PHYSICAL EXERCISE .....	141
<b>CHAPTER 7: EXPLORING RELATIONS BETWEEN DIFFERENT MARKERS OF ADIPOSITY AND THE CARDIOVASCULAR RISK PROFILE .....</b>	<b>152</b>
7.0 SUMMARY POINTS .....	152
7.1 INTRODUCTION AND OBJECTIVES .....	152
7.2 SUBJECTS AND METHODS .....	153
7.2.1 STATISTICAL METHODS .....	154
7.3 RESULTS .....	154
7.3.1 FORMATION OF FIFTHS OF BODY BUILD MEASURES .....	155
7.3.2 SYSTOLIC BLOOD PRESSURE (SBP) .....	155
7.3.3 FASTING INSULIN AND FASTING GLUCOSE .....	156
7.3.4 FASTING INSULIN AND ADIPOSITY .....	157
7.3.5 FASTING GLUCOSE AND ADIPOSITY .....	158
7.4 DISCUSSION .....	159
7.4.1 RELATION OF FINDINGS TO OTHER STUDIES .....	160
7.4.2 STRENGTHS AND WEAKNESSES OF THE STUDY .....	163
7.5 IMPLICATIONS FOR AETIOLOGY AND PREVENTION .....	164
<b>CHAPTER 8: CONCLUSION .....</b>	<b>172</b>
8.0 SUMMARY .....	172
8.1 INTRODUCTION .....	173
8.2 THE MAIN FINDINGS .....	173
8.2.1 STRENGTHS AND LIMITATIONS OF THE STUDY .....	175
8.3 IMPLICATIONS OF THE FINDINGS FOR MEASUREMENT OF ADIPOSITY IN ADOLESCENTS .....	175
8.3.1 BMI IS A VALID MARKER OF ADIPOSITY IN THE ADOLESCENT POPULATION .....	175
8.3.2 LIMITATIONS OF USE OF BMI IN ADOLESCENTS .....	176
8.3.3 ASSESSING CHANGES IN ADIPOSITY .....	177
8.3.4 WHICH ADDITIONAL MEASURE WOULD BE USEFUL? .....	177
8.4 NEED FOR PREVENTION OF ADIPOSITY .....	178
8.5 STRATEGIES FOR PREVENTION .....	178
8.5.1 HIGH-RISK STRATEGY .....	179

8.5.2 POPULATION APPROACH .....	179
8.6 SUGGESTIONS FOR FURTHER RESEARCH .....	181
8.6.1 MEASURES OF ADIPOSITY IN CHILDHOOD .....	181
8.6.2 DETERMINANTS AND CORRELATES OF ADIPOSITY .....	181
8.6.3 INTERVENTION STUDIES .....	182
<b>APPENDIX A: POSTAL QUESTIONNAIRE TO PARENTS .....</b>	<b>183</b>
<b>APPENDIX B: CHILD QUESTIONNAIRE .....</b>	<b>197</b>
<b>APPENDIX C: PUBERTY QUESTIONNAIRE .....</b>	<b>207</b>
<b>APPENDIX D: CO-AUTHOR PAPERS FROM TEN TOWNS STUDY .....</b>	<b>212</b>
<b>REFERENCES .....</b>	<b>214</b>

## TABLES

Table 3. 1: Cardiovascular mortality, infant mortality, and prevalence of low birth weight in 10 study towns .....	63
Table 3. 2: Origins and validation of questions in parental and pupil questionnaires .....	64
Table 4. 1: Numbers of children with body size and adiposity measures.....	92
Table 4. 2: Relation of observer to body size and adiposity variables.....	93
Table 4. 3: Gender differences in body size and adiposity measurements .....	94
Table 4. 4: Relation of age (years) to body size and adiposity variables.....	95
Table 4. 5: Highest Correlations of pubertal staging questions (p values) .....	96
Table 4. 6: Relations of pubertal stage score to body size and adiposity in boys.....	97
Table 4. 7: Relations of pubertal stage score to body size and adiposity in girls .....	98
Table 4. 8: Correlation Table of body size and adiposity measurements (boys) .....	99
Table 4. 9: Correlation Table of body size and adiposity measurements (girls).....	100
Table 4. 10: Correlations of fifths of log sum of skinfolds with adiposity variables.....	101
Table 4. 11: Correlations of fifths of log BMI with adiposity variables.....	101
Table 4. 12 Comparison with International centiles for BMI- BOYS .....	102
Table 4. 13: Comparison with International centiles- GIRLS .....	102
Table 5. 1 Social class distribution by town in the study population.....	119
Table 5. 2 Ethnic group distribution by town .....	120
Table 5. 3 Social class distribution by ethnic group in the study population .....	120
Table 5. 4 Differences in adiposity by town .....	121
Table 5. 5 Rochdale: Least Squared means .....	122
Table 5. 6 Social Class means adjusted for age, observer and pubertal status. ....	123
Table 5. 7 Ethnic differences, separately by gender .....	124
Table 5. 8 Gender interaction (for social class and for ethnic group).....	125
Table 6.1 Parental height, weight and BMI .....	143
Table 6.2 Children's adiposity variables according to fifths of maternal BMI .....	144

Table 6. 3 Children's adiposity variables according to fifths of paternal BMI .....	145
Table 6.4 Mean (SE) adiposity measures by infant feeding categories .....	146
Table 6.5 Differences in adiposity measures (Formula Fed minus Breast Fed) .....	147
Table 6. 6 Differing levels of exercise in leisure time by gender and age.....	148
Table 6. 7 Test for gender interaction with exercise level, adjusted for age.....	148
Table 6. 8 Adiposity measures by exercise group .....	149
Table 6.9 Self-assessed activity level compared to peers .....	150
Table 6.10 Activity level compared with peer group and adiposity measures .....	151
Table 7. 1 Numbers of children with cardiovascular risk factor measures.....	165
Table 7. 2 Effect of whether child (subject) had fasted on insulin and glucose levels .....	165
Table 7. 3 Observer variation in systolic BP measurement .....	166
Table 7. 4 Tests for gender interaction, p values for difference .....	167
Table 7. 5 Fifths of adiposity measures .....	168
Table 7. 6 Systolic Blood Pressure (SBP) by fifth of body size variables.....	169
Table 7. 7 Insulin by fifth of body size variables, adjusted for time of day. ....	170
Table 7. 8 Fasting glucose by fifth of body size variables, adjusted for time of day.....	171

## FIGURES

Figure 3. 1: Map showing location of study towns.....	61
Figure 3. 2 The relationship between the different Ten Towns data sets.....	62
Figure 4. 1: Height distribution.....	81
Figure 4. 2: Weight distribution.....	82
Figure 4. 3: Body Mass Index (BMI) distribution .....	83
Figure 4. 4: Sum of Skinfolds distribution.....	84
Figure 4. 5: Body fat perccent (from bioimpedance) distribution .....	85
Figure 4. 6: Waist circumference distribution.....	86
Figure 4. 7: Waist : Hip Ratio distribution.....	87
Figure 4. 8: Subscapular:triceps ratio distribution .....	88
Figure 4. 9: Comparison of Ten Towns boys to proposed international centiles .....	89
Figure 4. 10: Comparison of Ten Towns girls to proposed international cut offs .....	89
Figure 4. 11a Scatterplots of log(BMI) with other adiposity variables.....	90
Figure 4.11b Scatterplots of log(BMI) with other adiposity variables.....	91
Figure 5. 1 Town differences in height, weight and adiposity measures.....	118

## **DECLARATION OF AUTHORSHIP**

My role in the Ten Towns study involved participating in the detailed planning of the 1998-99 study in the 6 months leading up to the fieldwork. I acted as study doctor in piloting studies and in the fieldwork itself. My field role was venesection and processing of samples; I also completed bioimpedance measurements on children who were having blood samples taken. I did extensive data cleaning on the data used in this thesis. After consultation with my supervisor and others involved in this study, I planned, undertook and interpreted all the statistical analyses presented here. I had additional support for the section on breast feeding from Dr. S. Kaye, research assistant at St Georges' medical school.

This thesis has been my own work and I am the sole author.

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## **ABBREVIATIONS**

BMI            Body mass index (weight/ height<sup>2</sup>)

WHR            Waist: hip ratio

STR            Subscapular: triceps skinfold ratio

## CHAPTER 1: INTRODUCTION

### **1.0 SUMMARY**

Adiposity is defined as the property of containing fat. Excessive adiposity is a cause of both morbidity and mortality in adults. Important consequences include increased risks of type 2 diabetes, coronary heart disease and stroke, (particularly through the increased risks of high blood pressure, dyslipidaemia and insulin resistance associated with adiposity), osteoarthritis, gall bladder disease and some cancers (particularly endometrial, breast, and colon). There is concern, both in the UK and in other settings, that levels of adiposity have risen during the past 20-30 years or so, both in adults and in children and adolescents, in whom relative increases in risk have been particularly large.

Key adiposity indicators include markers of general adiposity, of which body mass index weight (kg)/height<sup>2</sup> (m<sup>2</sup>) is the most commonly used and markers of central adiposity, of which waist circumference is the most commonly used; central adiposity is particularly related to increased risks of cardiovascular disease and type 2 diabetes. High levels of body mass index, and, increasingly waist circumference, have been used to define overweight and obesity, although the definition of cut-offs for abnormality is difficult, especially in children and adolescents.

In British adults, there are marked geographic, social and ethnic variations in the occurrence of adiposity. Higher levels of adiposity have been reported in Northern England and Wales when compared to Southern England, in lower social class groups compared to higher ones and among South Asians compared to White Europeans. However, there is limited information on the extent of geographic, social and ethnic variation in adiposity in children.

Although raised adiposity in an individual fundamentally reflects an imbalance between energy intake and expenditure, many potential determinants of adiposity have been identified. These include not only recent and previous dietary factors and physical activity, but also early life exposures and parental influences.

In a population-based study of British 13-16 year-olds, this Thesis sets out to address four overall aims;-(i) to examine patterns of adiposity, including the interrelationships of different adiposity markers; (ii) to explore geographic, social and ethnic differences in adiposity and relate them to patterns observed in adults; (iii) to examine the association of three individual

factors – current physical activity pattern, infant feeding and parental BMI – with adiposity; (iv) to examine the associations between adiposity markers, blood pressure and insulin resistance, particularly to determine which adiposity measures are most strongly related to these factors in adolescence.

## **1.1 INTRODUCTION**

In this chapter issues of definition and measurement of adiposity are addressed (Section 1.2). The importance of adiposity, both in adulthood and in childhood, is discussed (Section 1.3). Important determinants of adiposity are outlined (Section 1.4). The overall aims of the thesis are outlined in Section 1.5.

## **1.2 ADIPOSITY – DEFINITIONS AND MEASUREMENT**

Adiposity is defined as ‘having the property of containing fat’ (Concise Oxford dictionary). The most commonly used method of ascertaining overall adiposity is to measure weight, and to incorporate this into a weight-for-height index, normally body mass index (weight/height<sup>2</sup>). Although this measure can be reasonably accurately measured and is positively associated with adiposity, body mass index does not discriminate between fat mass and fat-free mass. Despite this, it has been the most widely used adiposity assessment tool, both in adults and children. Several other non-invasive methods have also been used to assess overall adiposity. Skinfold measurements, particularly in combination, have been used in research studies though less commonly in clinical practice. Skinfolds measure body fat more directly but are observer dependent, requiring training to assess correctly; there are no contemporaneous standards. They also differ in males and females. Clinically they tend to be used more to measure under-nutrition; they are less suitable in the overweight and obese as obtaining accurate readings is problematic (Tanner et al, 1975, Womersley et al, 1977). Bioelectric impedance (BIA) measures impedance of the body to a small electric current. The theoretical model on which bioimpedance is based makes simplifications, treating the body as a single cylinder. Published BIA equations are not necessarily transferable between populations and some perform poorly in healthy individuals, though they may be useful in defining population groups (Wells et al, 2006). Other more invasive methods for the assessment of overall adiposity include underwater weighing methods and DXA scanning (Wells et al, 2006). Both

of these methods provide extremely accurate assessment of adiposity but are limited in their applicability, requiring non-portable equipment which restricts their use to small-scale studies in research centres.

Central adiposity refers to the distribution of fat specifically around the abdomen. Greater degrees of abdominal adiposity, which give rise to greater rotundity ('apple shape') have been shown in adults to be particularly related to the risks of coronary heart disease and type 2 diabetes (Han et al, 1997b). The measurement of central adiposity is normally based on the waist circumference, either alone or standardized for hip circumference (waist: hip ratio) or height (waist:height ratio). The accurate measurement of waist (and hip) circumference requires some observer training. These measures are positively related to visceral adiposity, which increasingly appears to be the important factor. Scanning techniques provide more precise measures of visceral fat.

### **1.2.1 DEFINING INCREASED LEVELS OF ADIPOSITY**

Adiposity is a continuous measurement; the measures of adiposity referred to above show either normal or log-normal distributions in different populations. Definitions used to define increased levels of adiposity are therefore somewhat arbitrary. In adults, these are generally based on the recognition of levels of adiposity associated with appreciable increases in risk. This is the case for the widely used World Health Organisation definitions of overweight and obesity, which use body mass index [weight (kg)/height (m)<sup>2</sup>] to define overweight as a body mass index (BMI) over 25 kg/m<sup>2</sup> and obesity as a BMI of over 30 kg/m<sup>2</sup>. Cohort studies relating adult BMI to adult mortality have generally showed a 'J' shaped curve, with the lowest mortality at the 20-25 kg/m<sup>2</sup> range of BMI (Manson et al, 1995, Lew 1985). However if potential confounding factors (e.g. cigarette smoking habit) are taken into account and a combined morbidity and mortality outcome including cardiovascular disease and type 2 diabetes is used, risk rises in a graded way from the lowest BMI group (BMI 20-22.4 kg/m<sup>2</sup>) (Shaper et al, 1997). The other increasingly widely used approach to the definition of adiposity in adults is based on central adiposity, and uses simple cutoffs based on waist circumference, defined on the basis of the relation to adult cardiovascular mortality (Han et al, 1995). Waist:

hip ratio and trunk: extremity skinfold ratio can also be used. Measures of high waist circumference are also used in the definitions of the metabolic syndrome.

The definition of excessive adiposity in children is difficult, because the relations of adiposity to morbidity and mortality are less clearly defined than in adults; choice of 'the best measure' is not clear cut. Most available definitions are based on body mass index. Definitions based on age-specific and/or sex-specific centiles, have been augmented by more sophisticated assessments which extrapolate backwards from the adult cut offs above, to produce proposed centile cutoffs for use in children (Cole et al, 2000). However, since the correlations between body mass index in childhood and adulthood are not perfect and its relations to adult morbidity and mortality unclear, the value of such markers remains unclear. This makes BMI difficult to interpret in an individual child. Although waist circumference and bioelectrical impedance are increasingly used for the measurement of adiposity in children and ranges of usual values have been defined, no validated cutoffs for excessive adiposity yet exist. With the difficulties of standardizing skinfold measurements, both by observers and between genders, these have been little used and there are no contemporaneous standards. Clinically skinfold measurements are used more to measure under-nutrition than over-nutrition; they are less suitable in the overweight and obese as obtaining accurate readings is problematic (Tanner et al, 1975, Womersley et al, 1977).

### **1.3 THE IMPORTANCE OF ADIPOSITY**

Excessive adiposity, which is increasingly common in the general population, has several well-documented adverse consequences in adults. Higher levels of adult adiposity show positive graded associations with risks of coronary heart disease and stroke, type 2 diabetes, osteoarthritis, gallbladder disease, sleep apnoea and respiratory complications and with several cancers, particularly those involving the endometrium, breast, and colon (Gelber et al, 2007, Wilson et al, 2002, Field et al, 2001, Chang et al, 2007, Wenten et al, 2002, Narayan et al, 2007). Excessive adiposity is also associated with adverse psychological and social consequences (Viner et al, 2005). The relations with coronary heart disease and stroke are at least partly accounted for by associations between excessive adiposity and increased levels of blood pressure and total and LDL cholesterol. Increased adiposity is also strongly associated

with insulin resistance, a precursor of type II diabetes, and with the metabolic syndrome, a combination of metabolic abnormalities associated both with risks of type II diabetes and cardiovascular disease.

Excessive adiposity in childhood and adolescents, which is also becoming increasingly common, also has adverse health consequences both in the short and long term (Reilly et al, 2003). However, the magnitude and independence of some of these associations are not yet clear, because of the limited longitudinal information linking childhood adiposity with adult disease outcomes and also taking account of adiposity in adult life. Moreover, while it is clear that excessive adiposity in childhood is associated with an increased risk of adiposity in later life, the extent to which adiposity in childhood contributes to long-term ill-health independently of its relation to adult adiposity remains uncertain.

### **1.3.1 TRACKING OF ADIPOSITY FROM CHILDHOOD TO ADULT LIFE**

Evidence suggests that over 60% of overweight adolescents remain overweight as adults (Gortmaker et al, 1993, Guo et al, 1994); in Britain results from the 1958 birth cohort show that lower class children are more likely to remain overweight or obese than other children (Power et al, 1988). The persistence of excessive adiposity between childhood and adult life is particularly strong when the excess is severe, present at older ages, and is associated with parental obesity (Reilly et al, 2003).

Childhood adiposity has strong short-term associations with less favourable levels of cardiovascular risk factors. Excess adiposity is associated with higher mean blood pressure, raised total and LDL cholesterol, triglycerides and low HDL levels (Freedman et al, 1999, Morrison et al, 1999a and 1999b, Maffeis et al, 2001, Berenson et al, 1998). It is also associated with changes in vascular structure and function, particularly diminished arterial distensibility (Whincup et al, 2005). Several reports, particularly from the Bogalusa Heart Study, have suggested that excessive adiposity in childhood may be directly related to adverse levels of cardiovascular risk factors in adult life (Bao et al, 1997, Smoak et al, 1987, Wattigney et al, 1995, Freedman et al, 2001). Moreover, several studies have suggested that adiposity in later childhood may be positively associated with later coronary heart disease

(Baker et al, 2007, Willett et al, 1995) though few of these studies can separate the independent influence of childhood adiposity from continuing adiposity in adult life.

### **1.3.2 RISING LEVELS OF ADIPOSITY**

Over the last 40 or so years increasing adiposity has been noted in adults in Western, developed societies and in many other parts of the world. The World Health Organisation (WHO) estimated that globally in 2005 approximately 1.6 billion adults (age 15+) were overweight; with at least 400 million adults obese, with projections that by 2015, approximately 2.3 billion adults will be overweight and more than 700 million will be obese. In the UK, several national and local surveys have reported marked increases in the prevalence of overweight and obesity. In the Health Survey for England, the prevalence of obesity (BMI of 30 kg/m<sup>2</sup> or greater) has increased between 1993 and 2004 from 13% to 23% in men aged >30 years and from 16% to 24% in women (source BHF statistics website). Prevalence of adiposity in British children has also increased. In the Health Survey for England (children aged 2-15 years) in 1995 11% of boys and 12% of girls were obese, rising to 19% of boys and 18% of girls by 2004 (source BHF statistics website). Other British studies in preschool children (Reilly et al, 1999, Bundred et al, 2001), and in school-age children (Rudolf et al, 2001, Chinn et al, 2001), also find increasing levels of adiposity, which are discernible even in children under 48 months of age (Reilly et al, 1999).

## **1.4 FACTORS ASSOCIATED WITH ADIPOSITY**

### **1.4.1 REGIONAL, SOCIAL AND ETHNIC FACTORS**

In British adults, higher prevalence of overweight and obesity are observed in Northern and Western regions (HSE 2006), regions with higher rates of adult cardiovascular disease (Barker 1998). A similar pattern was observed in the 1958 British Birth cohort (the National Child Development Survey), which also observed a particularly high prevalence of overweight and obesity in Wales (Strachan et al, 2007). Similarly, social class differences in adiposity are apparent in British adults, with lower social classes showing higher levels of both general and central obesity; these patterns are more marked in women (1994, 1998, 2003 HSE). Ethnic differences in adiposity are well described in adults. South Asians characteristically have a lower mean body mass index, but larger waist circumference, WHR and trunk skinfold

measurements than white Europeans, (McKeigue et al, 1991, Knight et al, 1992, Bhopal et al, 1999).

There is considerably less information on geographic, social and ethnic differences in British children. In the previous wave of the Ten Towns study, 8-11 year old children in towns with high cardiovascular mortality (in Northern England and particularly in South Wales) were shorter and had higher ponderal index (weight/height<sup>3</sup>) than children in low mortality towns, in Southern England (Whincup et al, 1996). In children there is some evidence of social class or socio-economic differences which become more marked in adolescence, particularly in girls (HSE 2003, 2006, Stamatakis et al, 2005, Wardle, Brodersen et al, 2006).

Studies comparing patterns of adiposity in South Asian and white European young people have suggested greater adiposity in South Asian boys compared to whites (Wardle, Brodersen et al, 2006, Saxena et al, 2004) and varying results for South Asian girls, Wardle; lower adiposity in South Asian girls, Saxena; lower in Indian girls and higher in Bengali and Pakistani girls compared to whites. In the previous wave of the Ten Towns study South Asian children had lower ponderal index than white children at age 8-11 but similar waist circumferences (Whincup et al, 2002) and increased tendency to insulin resistance.

#### **1.4.2 INDIVIDUAL DETERMINANTS OF ADIPOSITY**

Increased adiposity in an individual fundamentally reflects a previous lifetime imbalance between energy intake and expenditure. Recent and previous dietary factors (particularly energy intake) and levels of energy expenditure are of particular potential importance.

Physical activity levels in children and adolescents have been widely studied and have been linked to adiposity measures in adolescents in other studies (Andersen et al, 2006). However, many other factors have been related to the occurrence of increased adiposity at different stages of the life course. Of particular interest has been the role of exposures acting in early life. It has been suggested that foetal nutrition may influence long-term risks of adiposity and obesity (Law et al 1992). Early postnatal nutrition has been implicated, with the suggestion that breast feeding may offer long-term protection against overweight and obesity (Gillman et al, 2001). Parental adiposity is also an important determinant of adiposity in offspring, though the contribution of inheritance and shared environment is debated. Studies of parental

influence on offspring's BMI have found evidence of increased adiposity in children with obese or overweight parents (Burke et al, 2001, Duran-Tauleria et al, 1995).

#### **1.4.3 AVAILABLE INFORMATION ON THE DETERMINANTS OF ADIPOSITY IN YOUNG BRITISH PEOPLE**

Despite the importance of understanding the scale of excess adiposity in young people and its determinants with a view to prevention, population-based studies of adiposity in young people in Britain have been limited in scope. Although some data are available from the Health Survey for England and the Avon Longitudinal Study of Parents and Children (ALSPAC), few of these studies are both nationally representative and have multiple measures of adiposity. Most studies have relied on a very limited set of measurements, generally focussing on body mass index and waist circumference. Moreover, few other than ALSPAC have information on a wide range of determinants, including parental adiposity, early life exposures and current patterns of diet and physical activity. I have therefore used information from the Ten Towns Heart Health Study to address some of these issues. The third phase of the study (described in more detail in Chapter 3) includes a representative population of British school pupils aged 12-16 years from ten British towns, who had detailed measures of anthropometric parameters and bioelectrical impedance. Questionnaire information provided both by pupil and parents allowed ascertainment of a wide range of relevant exposures including current physical activity patterns, infant feeding and reported parental weights and heights.

#### **1.5 OVERALL AIMS OF THIS THESIS**

This thesis explores patterns and determinants of adiposity in a group of British 12-16 year old schoolchildren in the Ten Towns Heart Health Study using a wide range of anthropometric measures and bioimpedance. The overall aims of this thesis are:

- To examine the distribution and inter-relations of general and central adiposity in the study population, including comparison with the proposed international standards for obesity in childhood (Chapter 4).
- To explore whether there are differences in adiposity levels in relation to geographical location (town), social class and ethnic group, and whether these correspond to differences observed in adults (Chapter 5).

- To explore selected determinants of adiposity in this group of young people; parental BMI, breastfeeding in infancy and level of physical activity (Chapter 6).
- To explore the relations of adiposity measures with systolic blood pressure (a marker of cardiovascular risk), fasting insulin (a marker of insulin resistance) and blood glucose concentration. In particular, I aimed to establish whether any adiposity measures were more strongly related to these outcome measures than body mass index (Chapter 7).

The background and rationale for these aims are provided in Chapter 2.

## CHAPTER 2: BACKGROUND

### **2.0 SUMMARY**

Adiposity is increasing nationally and internationally in developed societies. This increase in overweight and obesity is seen in all ages, particularly affecting children and young people. Health consequences of excess adiposity in young people include adverse lipid profile, insulin resistance and type 2 diabetes, hypertension and damage to arterial structure and function. Assessment of adiposity on a population level usually relies on body mass index cut offs, however a variety of other measures of general and central adiposity can be used. There is less evidence on the validity of central measures of adiposity in childhood than in adults. Of the correlates of adiposity social and ethnic differences seen in adults appear to be emerging in adolescence; but not geographic differences. Of the individual determinants of adiposity considered parental BMI is strongly influential on offspring BMI; physical activity is associated with adiposity measures other than BMI; breast feeding has some protective effect against adiposity. Evidence for the above is considered in detail in this chapter.

### **2.1 INTRODUCTION**

This background chapter addresses five main issues. These include the definition and measurement of adiposity, overweight and obesity (Section 2.2); the evidence for a secular increase in adiposity levels, particularly in Britain (Section 2.3); patterns of sociodemographic variations in adiposity, particularly in Britain (Section 2.4); determinants of adiposity (Section 2.5) and correlates or outcomes related to adiposity, with a particular focus on cardiovascular risk (Section 2.6). The final section, 2.7, summarizes the issues raised by this chapter that will be explored in this thesis.

### **2.2 MEASURING AND DEFINING ADIPOSITY, OVERWEIGHT AND OBESITY**

Adiposity is defined as having the property of containing fat. Overweight and obesity are defined as abnormal or excessive fat accumulation that may impair health (Garrow J 1988). Measures are needed that are closely related to the accumulation of body fat and which are independent of overall body size. This is particularly true in children, where body size is changing rapidly. Weight is for this reason not a useful measure of adiposity. Weight (or

weight change) in an adult individual can give some guidance as to accumulation or loss of adipose tissue, but is not useful when comparing individuals as overall body size contributes to weight; a five foot tall person will weigh less than a six foot tall person but they may be visibly more overweight. Measures that aim to assess general adiposity will be detailed first, then those that aim to assess central adiposity. The focus will be on measures used in this study, with a brief discussion of other measures. The usefulness of a measure may also vary depending upon whether individuals or groups are being assessed, as some measures may have high sensitivity i.e. pick up the cases in the population but not be specific enough for use in individuals as there are too many false positives.

### **2.2.1 MEASURES OF GENERAL ADIPOSITY**

General adiposity means total body fat, regardless of distribution. The measures of general adiposity do not necessarily quantify this amount but are closely associated with it.

#### **2.2.1.1 Weight**

Weight alone in adults is reasonably highly correlated with total body fat, ( $r = 0.82$  in men and  $r = 0.91$  in women) (Roche et al 1981). It is simple and quick to measure, requiring minimal training. However it is also highly correlated with height, which is only weakly correlated with body fat (Himes et al, 1986). Therefore a measure of weight-for -height is more useful in assessing body size.

In children the correlation of weight with total body fat varies with age, and is lower than in adults, Roche et al (1981) finding higher correlations in teenage girls than boys (at age 6-12.9 years  $r = 0.74$  in both genders; at 13-17.9 years  $r = 0.65$  in boys and  $r = 0.88$  in girls). In children gender, height and age are important factors to take into account if possible. In the UK children's height and weight are usually plotted on growth charts in paediatric practice i.e. at age specific points, using charts produced in 1990 from UK children (Freeman et al, 1995). Simple evaluation of the concordance between height and weight centiles, and specifically observation of any progressive divergence can be a pointer to growth problems including overweight in the individual child. This gives more information on an individual basis than BMI alone, but does not translate to population studies.

Care has to be taken that charts used provide a valid reference. New WHO charts for under 2s were launched in the UK in April 2006; these were compiled using infants from a variety of countries, raised following internationally recognized health recommendations (a key element being exclusive breast-feeding to age 6 months). Previous charts for this age group reflected the growth trajectory of bottle fed infants; not the 'ideal'. The World Health Organization (WHO) is currently developing an international growth reference for school-age children and adolescents (WHO 2006).

### **2.2.1.2 Weight-for-Height: Body mass index (BMI)**

BMI is defined as the weight in kilogrammes divided by the square of the height in metres ( $\text{kg}/\text{m}^2$ ), providing a simple index of weight-for-height. The measure is reasonably easy to record, and measurements can be made with limited observer training. In adults, it is the most commonly used measure in classifying overweight and obesity in populations and individuals. BMI does not distinguish between weight associated with muscle and weight associated with fat therefore the relationship between BMI and body fat content varies according to body build and proportion. The WHO defines "overweight" as a BMI equal to or more than 25, and "obesity" as a BMI equal to or more than 30. These cut-off points are based on mortality data (Garrow 1981) and provide a benchmark for individual assessment, although there is evidence that risk of chronic disease in populations increases progressively from a BMI of 21 or lower (WHO 2006, Shaper et al, 1997, Kannel et al, 1996). In adults, BMI has been viewed as the most useful population-level measure of overweight and obesity as it is the same for both sexes and for all ages of adults. However, it should be considered as a rough guide because it may not correspond to the same degree of fatness in different individuals or populations.

Changes in body water can cause weight loss (as in dehydration) and changes in muscle bulk also cause variation in weight and hence BMI. It also does not give information about distribution of fat i.e. relative proportion of central fat. There are ethnic differences in the degree of fatness at a given BMI, with South Asians having higher central fat and cardiovascular risk factors at the same BMI as white populations (McKeigue et al, 1991, Chowdhury et al, 1996), leading to overweight in South Asians being defined as BMI over 23 (WHO 2004).

In children BMI is strongly correlated to total body fat (r around 0.9 for boys and girls, ages 6 to 17.9). The relationship is less strong in younger girls to percentage body fat than in adults (adult men  $r = 0.67$ , women  $r = 0.70$ , boys aged 6-12.9  $r = 0.68$ , girls  $r = 0.55$ ) (Roche et al 1981). The use of BMI as a marker of overweight and obesity in children is more complicated than in adults because BMI varies strongly with age in childhood. BMI rises in infancy then falls in the preschool years before rising again in adolescence. Therefore childhood BMI needs to be assessed using age-specific reference curves. The first BMI charts for the UK were produced in 1990; prior to this the Tanner and Whitehouse charts (for height and weight) used in the UK were based on data from the 1950's and were superseded by the current Child Growth Foundation charts developed by Freeman et al (height and weight) and Cole et al, (BMI) (Freeman et al, 1995, Cole et al, 1995), which used data from more contemporary children (1978-90); there was increasing recognition that the Tanner charts did not reflect the norms for modern children. However fixing national centiles provides a method of linking the distribution of BMI in childhood; if new population centiles are used in a fattening population there will tend to be a 'drift' to ever higher mean BMI; 10% of the population will always be above the 90<sup>th</sup> percentile (Prentice 1998). In clinical practice these charts are recommended to be used as defining 'overweight' as over the 91<sup>st</sup> centile and 'obese' as over the 98<sup>th</sup> centile, to ensure high specificity. For research purposes it is recommended that the 85<sup>th</sup> and 95<sup>th</sup> centiles are used (SIGN guideline 2004, Reilly et al, 2002). This means that the UK Department of Health defines obesity as over the 95<sup>th</sup> centile whilst clinicians define obesity as over the 98<sup>th</sup> centile.

Differences in definition of obesity have also occurred internationally, with countries using local centile charts and local cut-offs. In an attempt to address this Cole proposed international centiles for BMI in childhood, using pooled data from a variety of populations and calculating centiles that would pass through BMI of 25 (overweight) and 30 (obese) in adulthood to produce centiles defining overweight and obese in childhood (Cole et al, 2000). However, in clinical practice international cut-offs can have lower sensitivity than local charts (Reilly et al, 2000).

The value of BMI as a marker of obesity in individual children is limited by its lack of independence from height, so that it is more sensitive to overweight in tall, rather than short

children (and to underweight in short children)(Mulligan et al, 1999). In addition, a single measure of BMI is difficult to interpret (Ellis et al, 1999). Currently in the UK all schoolchildren have BMI measured in school reception (age 4-5) and again in Year 6 (age 10-11), as part of monitoring progress towards targets on tackling obesity in children .Children are not given their BMI, unless parents specifically request the information, because of problems with interpreting the measurement. An ideal measure of obesity would robustly represent a biological endpoint, rather than a population distribution (Dwyer et al, 1996).

In summary BMI is an easy to measure weight-for-height index, which gives some information on direction of change in body size but is limited by difficulties in establishing reference values and by lack of ability to discriminate body composition or fat distribution.

#### **2.2.1.3 Other indices of weight-for-Height: Ponderal index (PI)**

Ponderal index is the weight in kilogrammes divided by the 3<sup>rd</sup> power of the height in metres (kg/m<sup>3</sup>), it provides an index that may be more independent of height in childhood, particularly during puberty (Cole 1986). However as height in childhood is not independent of adiposity, a weight-for height index that is independent of height may not be optimally representative of adiposity (Lazarus et al, 1996). In the Bogalusa Heart Study in 10-15 year old children both PI and BMI were found to be equally predictive of adiposity 15 years later (Valdez et al, 1996).

#### **2.2.1.4 Skinfold thickness**

Skinfold thickness measurements provide a measure of subcutaneous fat i.e. fat stored below the skin. These measurements require considerable observer training and standardization. They are useful in assessment of malnutrition and normal nutritional states, in which skinfold measurements are generally accurate. However, they are less suitable in the severely obese as accurate measurements are difficult (Tanner et al, 1975). In research settings skinfolds have been shown to give a good estimate of total body fat in normal-weight adults and children (Durnin et al, 1967, Womersley et al, 1977, Deurenberg et al, 1990b) using four skinfolds at standardised sites (biceps, triceps, subscapular and suprailiac) and an appropriate formula. In children aged 7-15 there is moderate correlation ( $r = 0.70-0.85$ ) between adiposity calculated from BMI and total body fat from skinfolds (Lazarus et al, 1996); Roche et al found similar moderate correlations between total body fat from underwater weighing and individual

skinfolds in children and adults (Roche et al 1981). In children Deurenberg et al compared body density calculated from skinfolds with that achieved by underwater weighing and concluded that there is a prediction error of 3-5% in skinfolds, similar to figures obtained in adults. Several different formulas for calculating body density (and hence body fat) from skinfolds have been proposed, recently work in Spanish adolescents aged 13.0-17.9 years in the AVENO study (Rodríguez et al, 2005) comparing body fat percent derived from skinfold measurement to that obtained from DEXA scan concluded that the use of the correct equation is critical for accuracy at the individual level, recommending only those of Slaughter et al (1988) and (girls only) Brook (1971). It may be that formulas developed in specific research populations are poorly applicable to other groups due to confounding factors such as ethnicity, maturational stage etc. Skinfolds are not widely used in primary care settings in clinical practice in Britain now; they are difficult to standardise between observers, different calipers can alter the results (Tanner et al, 1975) and there are no contemporary standards.

#### **2.2.1.5 Bioimpedance-derived body fat mass**

Bioelectrical impedance involves passing an alternating current between electrodes on the hand and foot, the impedance of the body reflects the relative amounts of conducting material (i.e. the intra- and extra-cellular water) compared to insulating material i.e. fat. This is a simple measure, requiring little observer training. However there are many confounding factors such as the cross-sectional area of the limbs and differences in fluid distribution which for a given body composition will affect the measured impedance (Heitmann 1994). Review of the validity of bioimpedance in adults suggested that it is a suitable method where there are no large shifts in body water and may provide a more reliable measure of adiposity than skinfolds or height and weight, but not on an individual basis (Heitmann 1994). In children assumptions that fat free body mass and density are stable do not hold; body composition is strongly age-dependent (Deurenberg et al, 1990a). Correlation with fat free mass from densitometry was 0.93-0.97 depending upon equation used (Deurenberg et al 1991). Suitable equations can be validated for defined age groups and genders but prediction errors may increase with extremes of body size, eating, recent exercise and ethnicity (Houtkooper et al, 1992).

## 2.2.2 MEASURES OF CENTRAL ADIPOSITY

Central adiposity is the adipose tissue that is deposited intra-abdominally, also known as visceral fat. In adults the amount of visceral fat can vary dramatically within a narrow range of total body fat or BMI. Central adiposity is of interest because it is associated with particularly high cardiovascular risk and Type 2 diabetes in adults. There is also some evidence for this in adolescence; some studies have found that visceral fat is related to adverse lipid profiles (Brambilla et al, 1994, Caprio et al, 1995) and others that raised waist circumference in adolescents was associated with abnormalities in lipid and insulin concentrations (Flodmark et al, 1994, Freedman et al, 1999). Generally adolescents have low levels of visceral fat.

### **2.2.2.1 Waist circumference and waist: hip ratio (WHR)**

Waist circumference in adults is a simple, practical index of intra-abdominal fat and is little influenced by height or age (Han et al, 1997c). Correlation of waist circumference and abdominal fat in adults is 0.78 (Han et al, 1997a; MRI study). Differences in waist circumference reflect changes in risk factors for cardiovascular disease (Han et al, 1997b), although this relationship varies in different ethnic populations (McKeigue et al, 1992, Dowling et al, 1993), making production of cutoffs for waist circumference problematic. For the Caucasian population it has been suggested that a circumference of over 80cm in women and 94 cm in men represent increased risk of metabolic complications, with weight reduction required for circumferences over 88 in women and 102 in men (Han et al, 1995, Lean et al, 1995). However these cutoffs were developed using regression from BMI values for overweight and obese; there are current calls for further work on developing waist cut offs that are based on cardio metabolic risk (Klein S et al, consensus statement, 2007). Waist circumference and waist hip ratio are closely correlated and there is ongoing debate about which of these measures is more useful in adults; it is accepted that a WHR of over 1 in men and 0.85 in women indicates abdominal fat accumulation (Han et al, 1997a). Waist circumference alone may be a more practical indicator of abdominal fat ( Seidell 1995, Lean et al, 1995), whilst hip circumference is inversely associated with development of cardio-metabolic risk factors and CVD, possibly because it is a proxy measure for exercise, due to measuring gluteal muscle mass (de Koning et al, 2007). This meta-analysis looking at Waist circumference and WHR as predictors of CV events concluded that both were significantly

associated with the risk of CV events, WHR slightly more strongly but not significantly so (WC RR 1.63, 95% CI 1.31-2.04, WHR RR 1.95, 95% CI 1.55-2.44).

In children the proportion of abdominal fat that is visceral fat (as opposed to subcutaneous fat) is much smaller than in adults which has made it impossible to assume that waist circumference represents the same risk in children as it does in adults. However evidence is accumulating from imaging studies that waist circumference does provide the best predictor of variance in intra-abdominal adipose tissue; explaining 67.4% of the variance in one study (Benfield et al, 2008, 13-14 year olds, UK) and 64.8% in another (Brambilla et al, 2006, 7-16 years, pooled data, Hispanic and white). In both these studies, BMI was more strongly correlated with subcutaneous abdominal adipose tissue; (84.8% and 88.9% of variance explained respectively). Although waist circumference percentiles have been developed using data collected from children in 1988 (McCarthy et al, 2001), in mainstream clinical practice waist circumference is not used commonly in the assessment of adiposity (Reilly et al, 2002, Reilly 2006).

#### **2.2.2 Subscapular: triceps ratio (STR)**

Subscapular: triceps ratio reflects centrality of fat distribution i.e. a central (truncal) skinfold compared to a peripheral one. This measure is relatively difficult, requiring observer training to obtain the two accurate skinfold measures required to produce the ratio. Trunk skinfolds may reflect intra-abdominal fat, through the close relationship between the latter and subcutaneous abdominal fat; one study used MRI to assess intra-abdominal fat in 11 year olds (25 boys and 25 girls) and concluded that in girls subscapular skinfolds were the best indicator of intra-abdominal fat but were not sufficiently sensitive for clinical determination of intra-abdominal fat in individual children (Fox et al, 1993). More usually, and with boys in the above study, trunk: extremity skinfold is the variable associated with 'centrality' and abdominal fat. Trunk: extremity skinfolds increase in boys with pubertal changes (Baumgartner et al, 1986).

#### **2.2.3 OTHER MEASURES OF ADIPOSITY**

Other circumferences and skinfolds have been proposed including mid-upper arm circumference, thigh circumference and thigh skinfold. Measurements of thigh and calf skinfolds, have been recommended as significantly enhancing estimation of percent body fat

(Eston et al, 2005), but none of these methods are widely used in the general population. Drawbacks of these methods are that they require precise measurement, often of multiple sites, with calibrated equipment, by trained observers so are difficult to implement outside specialist settings.

More invasive measures are available, which cannot however be used in routine epidemiological surveys. These include DEXA scanning, which may be used to obtain a longitudinal image of the body, allowing calculation of body fat, although not so reliable for measuring intra-abdominal fat. In adults MRI and CT are considered the gold-standard methods for determining the quantities of subcutaneous abdominal and intra-abdominal adipose tissue, however there is no universally accepted site for measurement (i.e. vertebral level at which the 'slice' is taken) (Klein et al, 2007). Other methods include underwater weighing, to allow calculation of body density, and calculations using total body water or total body potassium, which can be measured using labeled isotopes and then used to calculate fat free mass and thus fat mass. These measures involve assumptions about body water which may not be applicable in children and are expensive.

### **2.3 EVIDENCE FOR INCREASING LEVELS OF ADIPOSITY**

There is considerable evidence that levels of adiposity, overweight and obesity are increasing over time in many locations. We will consider the evidence for adults and children separately, in each case commenting on the general and then the UK picture.

#### **2.3.1 EVIDENCE FOR INCREASING LEVELS OF ADIPOSITY IN ADULTS**

##### **2.3.1.1 Increase in adiposity in adults**

Recent WHO estimates for the European Region are that the prevalence of obesity has risen 3-fold or more since the 1980s. Levels of obesity and overweight in adults have been monitored by the WHO using data sets from national and regional surveys, gathered from existing databases, published literature, scientists and health agencies. In 2006 information was available for 46 out of 52 countries in the European Region, with prevalence of obesity varying from 5-23% among men and 7-36% among women (WHO European obesity conference 2006).

Global data gathered by WHO (WHO global NCD InfoBase) shows a similar increase in prevalence of adiposity indicating that globally in 2005 approximately 1.6 billion adults (age 15+) were overweight and at least 400 million adults were obese. WHO further projects that by 2015, approximately 2.3 billion adults will be overweight and more than 700 million will be obese. WHO's Global Database on Body Mass Index, cited as including the most comprehensive international data available on obesity trends, demonstrates that rates of obesity have tripled over the last 20-30 years in many countries including Japan, Brazil, England and the US (Jeffery et al, 2008). Once considered a problem only in high-income countries, overweight and obesity are now dramatically on the rise in low- and middle-income countries as well, particularly in urban settings.

Central obesity in adults has also been increasing. Using data from National Health examination surveys in the US marked increases can be seen in abdominal obesity since the first survey in 1960-62, with around 40% of men and 60% of women now having abdominal obesity (Okosun 2004, Li C et al, 2007). Waist circumference in both men and women has increased dramatically from 1960-2000 (Okosun 2004). Similar patterns have been reported in many other locations, including Sweden 1986-2004 (Lilja et al, 2008) and Finland 1987-2002 (Lahti-Koski et al, 2007).

### **2.3.1.2 Increase in adiposity in adults in Britain**

In Britain evidence for increasing obesity comes from national and regional health surveys, with rising obesity rates over the last 30 years and with a rate of increase greater than in most comparable European countries (DOH obesity strategy report 2008). Mean BMI increased by 1.9 in both men and women between 1980 and 1996, with obesity rates increasing from 6% of men and 8% of women in 1980 (Knight 1984), to 16% of men and 18% of women on 1996 (HSE 1996), and to nearly 25% of men and women today (HSE 2006). It is difficult to assess trends in BMI before 1980 as there do not appear to have been comparable surveys of adults (Obesity, British Nutrition Foundation Task Force 1999). Central obesity has also increased in adults, with recent studies showing increases in waist circumference in the UK 1993-2003(Wardle et al, 2007).

## **2.3.2 EVIDENCE FOR INCREASING LEVELS OF ADIPOSITY IN CHILDHOOD**

### **2.3.2.1 Increase in adiposity in children**

Recent work on trends in childhood overweight and obesity worldwide has indicated that the prevalence of obesity is increasing in most countries, with sharpest increases in economically developed countries and urban areas (Wang et al, 2006), most evidence being based on measurements of body mass index. It has been estimated that the European Union can expect to see the numbers of overweight and obese children rising by about 1.3 million a year by 2010 (Jackson-Leach et al, 2006). Successive national surveys in the USA for the US National Health and Nutrition Examination Survey have documented rising adiposity levels (Ogden et al, 2002).

Increases in other markers of adiposity and in central adiposity in children have also been documented. Central fat assessed by skinfold ratio increased in Spanish children aged 6.5 to 11.5 between 1980 and 1995 (Moreno et al, 2001). Waist circumference also increased in Spanish adolescents between 1995 and 2002; this exceeded increases in BMI at age 13 in boys and age 14 in girls (Moreno et al, 2005). In the USA greater increases in waist circumference than BMI were seen in 6-11 year olds over 1988-2002 (Okosun 2006) and comparison between National Health and Nutrition Examination Surveys in 1988-94 and 1999-2004 in 6-19 year olds show that abdominal obesity (defined as the 90<sup>th</sup> centile for gender and age in the earlier survey) increased from 10.5 to 17.4% in boys and 10.5 to 17.8% in girls (Li C et al, 2006).

### **2.3.2.2 Increase in adiposity in children in Britain**

Evidence for increasing childhood obesity in Britain measured by BMI comes from successive Health Surveys for England; in the UK between 1995 and 2003, the prevalence of obesity among children aged 2 to 10 rose from 9.9% to 13.7% and overweight (including those who were obese) rose from 22.7% in 1995 to 27.7% in 2003. Patterns were essentially the same in boys and girls. Increases in obesity prevalence were even more significant among older children aged 8 to 10, rising from 11.2% in 1995 to 16.5% in 2003 (Wardle et al, Obesity in Children under 11, 2006).

There has also been evidence of increase in adiposity in British children when measured by adiposity indices other than BMI. Skinfold centile charts for subscapular and triceps skinfolds

were published in Britain in 1962 and 1975 using data from 1960 and 1966-7 respectively (Tanner, Whitehouse 1975). There was noted to be marked increases in skinfolds of 0-2 year olds over this period, postulated as being due to increased bottle feeding. Skinfolds of school aged children showed a slight increase. Triceps skinfold increased by 7-8% in English 7 year olds between 1972 and 1994 and by 10-11% in Scottish 7 year olds (Hughes et al, 1997). Increases in both BMI and triceps skinfolds were found in successive National study for Health and Growth surveys (1972,1982,1990) in English and Scottish children aged 4.5-11.9 years (Chinn, Rona 1994). Waist circumference increased proportionately more than BMI over the years 1987-97 in British 2-5 year olds and 11-16 year olds (McCarthy et al, 2005, McCarthy et al, 2003).

## **2.4 VARIATIONS IN ADIPOSITY DISTRIBUTION**

This section addresses the extent of variation in adiposity, overweight and obesity in relation to sociodemographic factors (principally region, social class and ethnic group). It examines whether systematic differences are apparent in adults and whether these differences have their origins in childhood. The timing of the emergence of such differences can provide clues to their aetiology, and indirectly to means of prevention. The ultimate aim of this is to enable early effective intervention and prevention of obesity. In this section the focus is on findings in Britain.

### **2.4.1 REGIONAL VARIATIONS IN ADIPOSITY**

#### **2.4.1.1 Regional variations in adult adiposity**

Patterns of regional variations in adiposity in Britain have been investigated by successive national health surveys (Health Survey for England), geographically diverse epidemiological surveys (e.g. British Regional Heart Survey) plus national birth cohorts. Evidence of a regional difference in adult obesity is apparent in sequential Health Surveys for England: highest levels of adiposity tending to be found in the north and west of Britain. In both the 1998 and 2003 surveys, the highest mean BMIs in men and women were found in the Midlands and the North. Highest obesity levels were in the West Midlands in men and in Trent in women in 1998 and in Yorkshire in men and West Midlands in women in 2003. Highest levels of obesity including morbid obesity were seen in the West Midlands in 2006 in

men and women. Overweight did not show a clear regional pattern. Lowest levels of mean BMI were found in the South East.

The British Regional Heart Study of men aged 40-59 found the lowest average BMI was in Guildford in Surrey, while average BMI and obesity prevalence tended to be higher in Scotland (Shaper et al, 1981). The 1958 British birth cohort when measured at 44-45 years (non-migrants) showed regional variation in mean BMI, with the highest mean BMI in Wales, then Scotland. The English regions did not show a particularly north-south difference in this study. Other measures of adiposity e.g. waist circumference were not reported (Strachan et al, 2007).

Central adiposity (from waist: hip ratio) also shows a regional variation. In 2003 raised waist: hip ratio ( $>0.95$ ) was highest in East Midlands, West Midlands and North-West and lowest in London and the South-East in men. In 2006 raised waist circumference ( $>102\text{cm}$  in men,  $>88\text{cm}$  in women) was found to be most prevalent in the West Midlands and in South Western England in both men and women: south west (37%) and West Midlands (34%) in men and in the South West (45%), East of England (44%) and West Midlands (42%) in women. Raised waist circumference also showed regional variation in the Health Survey for England with higher levels in the north and west (HSE 1998, 2003, 2006).

#### **2.4.1.2 Regional variations in childhood adiposity**

Recent evidence suggests that although there is regional variation in adiposity in children there is not a consistent north-south pattern; there are high levels of adiposity in inner city areas, regardless of geographical location. Geographical differences in height are seen. In the 2002 Health Survey for England obesity prevalence among children aged 2 to 10 varied according to region and area type. Obesity levels were lowest in Yorkshire and the Humber (11.4%) and the South East (13.4%) and highest in the North East (18.3%) and London (18.2%) in 2001 and 2002. Obesity was higher among children living in inner city areas than among children living in all other types of area. In 2006 Health Survey for England the lowest levels of obesity in boys and girls were seen in the East of England (12%, 13%), with highest levels in London for boys (23%) then West Midlands (20%) and for girls in the South West (19%) West- and East- Midlands (18%). In children, the previous waves of the ten towns study found that children in Northern England and South Wales were 1.2 cm shorter at ages 8-11, when compared to children from low cardiovascular mortality areas, and had significantly higher

ponderal indices ( $0.34\text{kg}/\text{m}^3$  higher). Children in Rhondda were noted to have particularly high ponderal indices. Differences in waist: hip ratio between grouped high and low mortality towns did not achieve statistical significance; Rhondda children had the highest mean waist: hip ratio (Whincup et al, 1996). In the 1970 birth cohort children from areas with high adult cardiovascular disease were shorter at age ten; there was a gradient for children's height with fifths of the standard mortality ratio distribution, but not for weight (Barker et al, 1989). In our study we might expect to find a geographical difference in height with shorter children in the 'high' cardiovascular risk towns which are in the north and west, but not necessarily a difference in adiposity.

## **2.4.2 SOCIAL VARIATIONS IN ADIPOSITY**

In Britain as in other developed societies higher levels of adiposity are found in lower socioeconomic groups.

### **2.4.2.1 Definitions of Social Class**

In Britain definitions of social class are generally based on occupation of the head of the household (or 'reference person') i.e. an adult male, or female if no male is present. This is a proxy for income and educational level, without being limited to either. Prior to 2001, social class was classified according to the Registrar-General's classification; after this a new system was used New Standardized Socio-Economic Classification (NS-SEC), which aimed to reflect more how much control people had over their own work, and the work of others. It also allowed for clearer classification of 'new' jobs that had arisen eg in IT. The five categories it produces are broadly comparable to the old SC I-V. Educational level can also be used as an alternative social class marker. First we will consider social class differences in adults, then children.

### **2.4.2.2 Social class and adiposity in adults**

In the UK, there are large scale data on social class patterns of adiposity, notably in the sequential Health Surveys For England (HSE), using BMI, waist: hip ratio and waist circumference. Adiposity varies with social class; this is particularly striking in women, and has been maintained over the change in social class classification and increase in adiposity generally.

The 1994 HSE found that body mass index and waist: hip ratio in women increased from highest to lowest social class, independently of differences in smoking habits, alcohol consumption and physical activity and (waist: hip ratio only) of current BMI. Women in social classes IV and V were more than twice as likely as women in social class I to be obese. In 1998 the same pattern was seen, within the context of an overall rise in adiposity in the population. Similar results were seen when household income or educational level were used in place of social class. In the 2003 HSE (using NS-SEC) there was found to be lower prevalence of obesity in women in managerial and professional households, and in intermediate households, than in the other three (lower social class) NS-SEC groups. Raised waist circumference and waist: hip ratio were not associated with NS-SEC social group in women, but when equivalised household income was used, all measures showed marked increase with decreasing income, which was also the case in 2006. The social class differences in men were similar to those in women but the effect was not as strong. In 1994 and 1998 HSE, BMI in adult men over 16 rose from Social class I to IIIM (then decreased in Classes IV and V), BMI for men in V being the same as for men in I in 1994, and a little higher in 1998. Obesity was highest in IV in 1994 and IIIM in 1998, with morbid obesity highest in V (1.6%). Waist: hip ratio did not show a clear relationship with social class in men, although prevalence of raised waist: hip ratio ( $\leq 0.95$ ) was higher in lower social classes (1998). Waist: hip ratio decreased with increasing educational level (1994) independently of BMI. In 2003 (using NS-SEC), fewer men in managerial and professional, or intermediate groups were obese or had raised waist circumference or waist: hip ratio compared to men in lower groups. A similar pattern was seen for waist and waist: hip ratio with household income, but not for obesity, which showed no association with income. In 2006 HSE obesity and waist circumference were not related to income in men. Findings in the 1958 British birth cohort of 11 405 men and women followed to age 33 y showed social class gradients in obesity in women at ages 16, 23 and 33 (though not at age 7 or 11) and in men at age 23 and 33 (Power et al, 2003). There was also an association of education with adult obesity ( $BMI \geq 30$ ), with increasing odds by 30% (men) and 35% (women) for each decrease in qualification level.

#### **2.4.2.3 Social class and adiposity in children and adolescents**

The studies that are available suggest associations between social class and adiposity or obesity that emerge and strengthen through childhood. Stamatakis et al used data from children aged 5-10 years in successive National Surveys of Health and Growth in 1974, 1984 and 1994 with data from Health Surveys for England from 1996 to 2003 to show that children in manual and lower income groups had higher levels of obesity and higher rate of increase in obesity prevalence than non-manual and higher income groups respectively (Stamatakis E et al 2005). In the 2002 Health survey for England associations between various measures of socioeconomic disadvantage and overweight and obesity prevalence were explored, in children and young people aged 2-15 and 16-24; rates of overweight and obesity were found to increase in both genders with lower NS-SEC category of head of household, and with lower household income. In both cases the pattern was weaker for boys than girls, and was absent for young men aged 16-24 with household income. These differences were also present in the 2006 HSE, with the effect remaining stronger in girls.

The findings detailed in the preceding paragraph based socioeconomic status on assessment at individual level. Some studies have defined socioeconomic status at area level and related it to obesity prevalence. Wardle et al studied children aged 11-16 from London schools and found highest levels of overweight and obesity (from BMI) in the most deprived children (defined by Townsend district from postcode) in both genders, but differences in adiposity were not graded across the levels of socioeconomic status (Wardle, Brodersen et al, 2006). A study of 20,000 British children aged 5 to 14 years showed that children from areas of high social deprivation had a higher prevalence of obesity (Kinra et al 2000). This is consistent with findings in the 2002 Health Survey for England that levels of obesity were 5 percentage points higher among children living within the most deprived areas (16.4%) than the least deprived areas (11.2%) in the UK. A Canadian study of 6684 11-15 year olds (Janssen et al, 2006) examined the effect of 'individual socioeconomic status' (material wealth or perceived family wealth) and 'area level socioeconomic status' (unemployment rate, percentage of adult residents with less than a high school education, average employment income from head of household). They found that both individual and area level socioeconomic status were independently associated with adiposity. We will not be considering area level socioeconomic status in detail in this study, where no data on area social class are available.

### **2.4.3 ETHNIC VARIATIONS IN ADIPOSITY**

This section considers ethnic differences in adiposity between South Asians and White Europeans.

#### **2.4.3.1 Ethnic variations in adiposity in adults**

The particular interest in adiposity among South Asians was stimulated by the finding that British South Asians in Britain had markedly raised age-standardised levels of coronary heart disease which were about 40% higher than the general population (OPCS 1990, Wild, McKeigue, 1997) and markedly increased prevalence of type 2 diabetes (Health Survey for England 1999, 2004). Investigations into the cause of these patterns found that South Asian adults in Britain had larger waist: hip ratios and trunk skinfold measurements (i.e. central adiposity) than whites, along with insulin resistance and non-insulin dependant (Type 2) diabetes mellitus. The findings applied to both men and women (McKeigue et al, 1991, Knight et al, 1992). In McKeigue's study mean BMI was slightly higher in Asian women than white women (27 vs. 25.2), in men BMI showed little difference (Asian 25.7, white 25.9). Mean waist: hip ratios slightly higher in South Asians than whites: 0.85 and 0.76 for S.Asian and white women and 0.98 and 0.94 for S.Asian and white men. A systematic review of studies exploring ethnic differences between South Asians and white British subjects (primarily to examine blood pressure differences) found that most studies showed South Asian men to have lower BMI and higher waist circumference whilst South Asian women had higher BMI and waist: hip ratio than white women in the majority of studies (Agyemang, Bhopal 2002). Similar findings were made in the Health Survey for England 2004; and in a Newcastle upon Tyne study (Bhopal et al 1999); BMI in South Asians being similar to or lower than whites, but central obesity being higher. The short stature of Bangladeshis in particular meant that obesity levels from BMI were low, even though mean waist: hip ratios were as high as other South Asians.

Investigation into ethnic differences in subscapular: triceps ratio, as a measure of central adiposity, have not been so extensively carried out as for waist: hip ratio. McKeigue et al (1992) found relationships between subscapular: triceps skinfold ratio and insulin resistance in London white and S.Asian men and S.Asian women, but in all groups the relation between waist: hip ratio and insulin resistance was stronger (note numbers of white women with insulin resistance were insufficient for analysis). Imaging studies have shown that South Asians have

a higher proportion of total body fat and visceral fat than Europeans at the same levels of BMI and waist circumference respectively (Lear et al, 2007, Chowdhury et al, 1996). This results in more adverse cardiovascular profiles in S. Asians (Lear et al, 2003, Razak et al, 2005) and has led to suggestions that lower BMI cut-offs for overweight and obesity should be considered in these populations (WHO expert consultation report 2006).

#### **2.4.3.2 Ethnic variations in adiposity in children**

There is some evidence of higher adiposity in South Asian children in Britain; Saxena (Saxena et al, 2004) examined ethnic differences in adiposity in 2-20 year olds from the 1999 Health Survey for England, finding higher prevalence of obesity in Pakistani girls and higher prevalence of overweight in Indian and Pakistani boys than in whites, using international obesity taskforce centile charts (Cole et al 2000) to define overweight and obesity. Wardle's London school-based study (Wardle et al, 2006) found that in boys aged 11 to 16 South Asians had slightly higher mean BMI and higher mean waist circumferences than whites (but not significant), South Asian girls had lower mean BMI and waist circumference than whites. In the previous wave of the Ten Towns study when the children were aged 8-11 South Asian children were found to have lower weight and ponderal index than white children and also to have higher mean insulin concentrations. Measures of central adiposity (waist: hip ratio, waist circumference) were not different. The relationship between adiposity and insulin concentrations (particularly fasting insulin) were stronger in South Asian than white children (Whincup et al, 2002).

### **2.5 INDIVIDUAL DETERMINANTS OF INCREASED ADIPOSITY IN CHILDHOOD**

For the purposes of this study we will focus on individual determinants of adiposity in childhood.

#### **2.5.1 INTRODUCTION: INDIVIDUAL DETERMINANTS OF ADIPOSITY**

Increased adiposity in an individual fundamentally reflects a previous lifetime imbalance between energy intake and expenditure. Recent and previous dietary factors (particularly energy intake) and levels of energy expenditure are of particular potential importance. Physical activity levels in children and adolescents have been widely studied and have been linked to adiposity measures in adolescents in other studies. However, many other factors have

been related to the occurrence of increased adiposity at different stages of the life course. Of particular interest has been the role of exposures acting in early life. It has been suggested that foetal nutrition may influence long-term risks of adiposity and obesity. Early postnatal nutrition has been implicated, with the suggestion that breast feeding may offer long-term protection against overweight and obesity. Parental adiposity is also an important determinant of adiposity in offspring, though the contribution of inheritance and shared environment is debated. We will consider each of these in turn, focussing on physical activity, infant feeding and parental adiposity respectively.

## **2.5.2 PHYSICAL ACTIVITY**

Exercise levels in young people are of interest as they present a behaviour pattern which may be amenable to change and hence may be an effective strategy in attempts to curb the national increase in young people's obesity. It has been suggested that physical activity in young people in developed societies is decreasing, but it is hard to prove this as getting consistent data on children's activity levels at different time points is difficult. A recent review (Dollman et al, 2005) attempted to gather the evidence, concluding that physical activity in clearly defined contexts such as active transport, school physical education and organised sport is declining in many countries. They also found that external factors such as school policies, parental concerns about safety and convenience, and the physical environment constrain young peoples' activity. However there is little direct evidence that lack of physical activity leads to adiposity in young people. Most assessment of physical activity is based on questionnaires, which may be of limited validity (Treuth et al 2004, Welk et al, 2000). Difficulties in comparing studies are highlighted by Must (Must, Tybor 2005, review); particularly due to imprecise measurement of activity exposures. A distinction has to be made between physical activity (action) and fitness (ability of body to sustain physical activity); we will consider evidence on physical activity levels.

### **2.5.2.1 Questionnaire-derived physical activity level and adiposity**

In the Health Surveys for England 2002 and 2006 no significant differences were seen in mean BMI between high, medium and low exercise groups in children aged 2-10 and 11-15; this exercise data was collected by questionnaire. In the AVENA study in Spain (2859 13-18 year olds) questionnaire-derived physical activity level was not related to adiposity, although level

of sedentary activity was positively related to both BMI and waist circumference in boys and to waist circumference in girls (Ortega et al, 2007). A regional Spanish study of 1068 children aged 7-12 years found associations between sum of skinfolds and physical activity level in boys (lower skinfolds with more activity), but not in girls, where the inactive group had higher skinfolds but levels of physical activity were not associated with skinfold differences (Ara I et al, 2007). In 2714 French 12 year olds structured physical activity level from questionnaire was associated with lower waist circumference in both boys and girls (Klein-Platet et al, 2005).

### **2.5.2.2 Accelerometer-derived physical activity level and adiposity**

In 224 7-10 year old school children in Northern Ireland physical activity was measured over 4 days by accelerometer; higher levels of activity was found to be associated with lower BMI and waist circumference in boys but not in girls (Hussey et al, 2007). In children aged 9-15 in the European Youth Heart Study (from Estonia, Denmark and Portugal) physical activity data was collected by accelerometer. Lower BMI (genders combined) was seen in children taking higher levels of physical activity, but this trend was not significant, whereas the trend of lower sum of skinfolds was highly significant, trend for lower waist circumference was also significant (Andersen et al, 2006).

### **2.5.2.3 Adiposity, physical activity level and possible cofounders**

Contemporary health behaviours that may affect adiposity include diet, physical inactivity, cigarette smoking and sleep patterns. Diet has been linked to increased levels of adiposity through passive overconsumption of high-fat containing foods (Blundell, King 1996), and intake of sugar-sweetened drinks (Reilly 2006). Cigarette smoking tends to lower BMI, with slight rise in BMI in those who quit (Rissanen et al, 1991). Sedentary behaviour (e.g. television viewing, computer games) is linked to increased adiposity in children, though evidence in adolescence is less clear (Must, Tybor 2005, review of 20 studies), possibly due to pubertal changes in body composition. It has been suggested that disturbed sleep patterns may also contribute to adiposity (Wells et al, 2008). All the above are to some degree interconnected, as certain behaviours may occur in tandem such as snacking whilst watching TV; therefore confounding is likely. Most studies linking physical activity with adiposity are cross-sectional, allowing observation of associations but not causality; it can not be clear whether inactivity or obesity comes first. In a review of 20 longitudinal studies the overall

conclusion was that increased activity levels are protective against relative weight and fatness gains over childhood and adolescence (Must, Tybor, 2005).

### **2.5.3 EARLY LIFE EXPOSURES**

Early life exposures relate to the environment of the fetus and neonate that have been implicated as possible determinants of later morbidity, including adiposity and insulin resistance. Early life exposures may include birth weight, infant feeding and timing of 'adiposity rebound', when the normal childhood drop in BMI reverses.

#### **2.5.3.1 Studies in adults**

Birth weight is related positively with adult BMI (Eriksson et al, 2003, Oken, Gillman 2003), and with fat-free mass acquisition (Kahn et al, 2000, Loos et al, 2001, Li H et al, 2003), and is not associated with raised insulin resistance and cardiovascular risk in adults (Eriksson et al, 2001, 2003), except in infants of diabetic mothers (Rich-Edwards et al, 1999). There have also been studies which found low birthweight associated with greater adult adiposity (e.g. Law et al, 1992) but these also adjusted for adult BMI, possibly inappropriately.

#### **2.5.3.2 Studies in children**

In the ALSPAC (Avon) cohort study birth weight was positively linked to obesity (from BMI) at age 7 (OR 1.05) (Reilly et al, 2005). In another cohort of 5-11 year old children birth weight was related to both BMI and skinfolds (Duran-Tauleria et al, 1995). These direct relationships may not be carried through to adulthood, with cohort studies suggesting that obesity in childhood and particularly adolescence have more influence on adult obesity than obesity in infancy does; most obese adults were not obese as children (Power, Moynihan 1988). Twin studies found that intrauterine effects on birth weight did not have an enduring effect on adulthood weight and BMI, although height was influenced (Allison et al, 1995).

Lower birth weight has been found to be linked to higher central deposition of fat (subscapular: triceps ratio) in adolescents ({boys}, Labayen et al, 2006, {girls} Barker M et al, 1997), and adults (Valdez et al, 1994, Sachdev et al, 2005) and higher waist: hip ratio in adults (men, Law et al, 1992, women, Fall et al, 1995). The timing of adiposity rebound has been implicated, with early rebound (from age 2) increasing the likelihood of later adiposity (Bhargava et al, 2004, Wadsworth et al, 2005). 'Catch-up' growth, or 'growth acceleration' where infants cross rapidly upwards over weight centiles in the first few weeks or months of

life has also been implicated in later adverse effects including obesity (Baird J et al, 2005), higher body fat and waist circumference at 5 years (Ong KKL et al 2000) and higher systolic and diastolic blood pressure at age 25 (Ben-Shlomo Y et al, 2008). ‘Programming’ of the sympathetic nervous system may be implicated (Young JB, 2008). ‘Catch-up’ growth is seen more frequently in children who were small at birth and also is more pronounced with artificial (bottle) rather than breast feeding. Increased infant growth rate by a nutrient enriched diet, even for only a few weeks, has been suggested to have long-term adverse effects on blood pressure (Singhal A et al, 2001), insulin resistance (Singhal A et al, 2003), lipid profile (Singhal A et al, 2004) and leptin resistance (Singhal A et al, 2002).

### **2.5.3.3 Breast feeding and adiposity**

It has been suggested that early life exposures are involved in the development of obesity (Kuh, Ben Shlomo 2004). Among these it has been suggested that breast feeding may have a protective effect, when compared with formula feeding. Several explanations for this have been postulated, including that breast-fed children learn to self-regulate calorie intake better (Birch et al, 1998); are less fussy eaters; that formula precipitates earlier fat deposition (Lucas et al, 1980 and 1981), and has lasting effects on glucose metabolism (Burns et al, 1998). Large studies have found differing effects of breastfeeding, some finding definite protective effects; Grummer-Strawn and Mei (2004) in a study of 177 304 US children, using health surveillance data, found a protective effect against overweight/obesity (BMI  $>95^{\text{th}}$  centile on US charts) at age 4 in non-Hispanic white children only (OR for obesity with 6-12 months breastfeeding 0.70). Studies in adolescents have also suggested protective effects, for example Gillman (Gillman et al, 2001) in a US cohort study of adult women, found that their 15,341 9-14 year old offspring had lower risk of obesity if breastfed (OR 0.78); duration of breastfeeding had no effect. Other studies in young children have found less clear associations; Hediger’s US study from NHANES III of 2685 US children aged 3-5 found a protective effect for overweight (OR 0.63) but not obesity (OR 0.81, not significant) with ever having breastfed; duration of feeding made no difference to the results (Hediger et al, 2001).

Recent systematic reviews of the published evidence have suggested that the association may be weaker than previously thought. A quantitative review of the evidence relating breast feeding to mean body mass index (BMI) through out life suggested that there was little or no relationship, particularly after adjustment for potential confounders including social class,

maternal adiposity and cigarette smoking (Owen et al, 2005a). Adjustment for maternal BMI alone halved the relationship seen (BMI from -0.11 to -0.05). Two other reviews examined infant feeding with risk of obesity; Arenz (Arenz et al, 2004) in childhood only, finding an adjusted odds ratio of 0.78 with any type or duration of breast feeding from 9 studies with 69,000 participants; not all studies were adjusted for confounders. Owen (Owen et al, 2005b), considered obesity across the lifecourse (28 studies, 298 900 subjects), finding a pooled OR of 0.87 for obesity with ever breast feeding, which was reduced to 0.93 in 6 studies that adjusted for the major confounding factors of parental BMI, maternal smoking and social class).

Harder's meta-analysis of 17 studies relating to duration of breast feeding and risk of obesity found a dose- dependent association, decreasing from OR of 0.81 at 1-3 months to 0.68 at over 9 months' breast feeding; however only eight studies adjusted for confounders, with only five adjusted for maternal weight. In all four reviews the presence of publication bias, residual or uncontrolled confounding could not be ruled out.

Most existing evidence on the effects of breast feeding use body mass index as the outcome measure for assessing obesity. This has limitations, particularly in the assessment of central adiposity, which is strongly related to metabolic and cardiovascular risk. Two studies have recently reported on the relation of breast feeding to body fat measured by dual X-ray emission absorptiometry (DXA) in young people. Burdette examined 313 US children at age 5; there were no differences in fat mass by breast feeding history (duration and ever vs never) (Burdette et al, 2006). Toschke examined 4325 UK children in the ALSPAC cohort study, finding reduction in trunk fat mass and total fat mass with duration of breast feeding, but these effects were greatly reduced by adjustment for confounders. The relationship between breastfeeding and adiposity (defined as trunk fat mass or total body mass in the top decile) were little altered by adjustment , having OR of 0.76 and 0.74 respectively (Toschke, et al 2007).

In conclusion breast feeding may have a small protective effect against adiposity but the effect is prone to confounding by maternal BMI in particular, as well as social status, smoking and possibly other factors; there is little evidence for a relationship with BMI per se. Other measures of adiposity with breast feeding have not been frequently studied.

## 2.5.4 FAMILIAL FACTORS

These can encompass genetic and environmental effects and the interplay between them. We will describe the familial associations first and then the issue of the contribution of genes and environment.

### **2.5.4.1 Parental BMI**

Studies of parental influence on offspring's BMI have found evidence of increased adiposity in children with obese or overweight parents (HSE 2006, Reilly et al, 2005, Burke et al, 2001, Duran-Tauleria et al, 1995). Studies generally use child and parental BMI either as continuous variables, or to define overweight and obese groups which are then compared. Studies using other measures of adiposity are sparse. Reilly et al investigated risk factors for obesity (BMI above 95<sup>th</sup> centile) in British 7 year olds in the Avon longitudinal study cohort (8234 children). The relationship of maternal obesity to child obesity (OR 4.25) was stronger than that of paternal obesity to child obesity (OR 2.54). Both parents obese had the strongest effect; giving an odds ratio of 10.44 for offspring obesity. The methodology was similar in the Health Survey for England 2006, with obesity in children aged 8-15 again defined as BMI above the 95<sup>th</sup> centile on the national centile charts. Children were separated by gender for analysis, with maternal overweight/obesity having a greater effect on girls (OR for girls obesity 3.66) than boys (OR 2.18); and on both genders compared to paternal overweight/obesity (OR for girls obesity 1.35, OR for boys obesity 1.56 with fathers overweight/obesity). With both parents overweight/obese the OR for obesity was slightly higher in girls (OR 3.80) but not in boys (OR 1.56).

Duran-Tauleria using data from the National Survey of Health and Growth in England and Scotland defined the highest quarter for weight-for-height of 5-11 year olds in their data population as overweight and found increased likelihood of children being in this group if their mother or father was obese (OR 1.90, 1.85 respectively) and even higher if both parents were obese (OR 3.48). When looking at child's weight-for-height as a continuous variable, they found a 40% stronger effect of father's BMI than mother's (regression coefficient for mother's BMI 0.049, father's 0.068). Offspring's sum of subscapular and triceps skinfold showed a similar pattern with parental BMI. Burke's Australian study followed 219 9 year olds up to the age of 18, completing 3 yearly surveys of the children and their parents.

Findings were that mother and father's BMI significantly predicted offspring BMI, explaining

33% of BMI variance in sons and 48% in daughters. Overweight or obesity in parents was associated with higher offspring BMI, with daughters showing a stronger association with maternal than paternal overweight/obesity; in boys the effect was similar for either parent. Overall it appears that BMI in parents predicts offspring BMI, with possibly a stronger effect of paternal BMI. However when the analyses are done using overweight/ obesity as a categorical variable, the effect of maternal obesity/overweight appears to be stronger than that of paternal, particularly in girls. Obesity in both parents appears to have an additive effect on offspring risk of obesity.

Longitudinal studies following the child to adulthood have found persisting associations between parental and child BMI as the child ages; Laitinen et al confirmed a relationship at ages 1yr, 14years and 31 years (Laitinen et al, 2001). Tracking has been shown to occur, with obese children more likely than reference population to remain obese into adult life. These children are also more likely to have obese parents. The odds of children with two obese parents being obese themselves at age 33 were 8.4 (boys) and 6.8 (girls), these were children from the 1958 British birth cohort; parental heights and weights were self-reported when the children were 11 years old (Lake et al, 1997).

#### **2.5.4.2 The developmental overnutrition hypothesis**

The developmental overnutrition hypothesis suggests that maternal overnutrition occurring during pregnancy (particularly denoted by a high maternal BMI at that time) would be related to adverse, metabolic consequences in the offspring. Specifically, high maternal glucose, free fatty acid and plasma concentrations would lead to permanent changes in appetite control, neuroendocrine functioning or energy metabolism in the foetus, thus leading to greater adiposity and risk of obesity in later life (Lawlor DA et al, 2008). This would thus link maternal and offspring BMI by mechanisms independent both of genetic effects and environmental effects operating during the postnatal life of the offspring. Studies have suggested greater effect of maternal than paternal BMI on offspring BMI (Lawlor et al, 2006, Lawlor DA et al, 2008). However, more recent studies examining causal influences of maternal BMI on offspring health outcomes found no marked effect of maternal BMI during offspring development on offspring at age 9-11 years (Lawlor DA et al, 2008).

### **2.5.4.3 Genetic factors**

It is intuitive to expect some aspects of body size to be genetically determined, for example height and breadth of shoulders. Other tissues such as muscle and fat mass may have a genetic component but also reflect body habits. The rapid increases in obesity levels in the last two decades suggest primarily an environmental aetiology, but recent research suggests that genetic susceptibilities unmasked by environmental changes are an important feature.

Twin studies suggest that 50% of between-individual variance in BMI (fat and lean mass) results from genetic factors (Allison et al, 1996), when other influences are excluded, whilst adoption studies show a strong relationship between the BMI of biological parents and adoptee, across the whole range of body fatness, but not adoptive parents (Stunkard et al, 1986, 1990, Sorensen et al, 1989). The substantial contribution of genetic factors to between-individual variance in BMI is based on studies carried out within a single population at a single point in time, and is therefore separate from considerations of the balance of environmental and genetic influences on changes in BMI over time, or variations between populations. There is also evidence of familial influence on body fat distribution (Borecki et al, 1995) i.e. whether fat is stored centrally or in the lower body, probably caused by a combination of genetic and environmental factors, eg food preferences (Francis et al, 2003).

The identification of specific genes associated with obesity has proved difficult to date. Although genes associated with severe, rare monogenic forms of obesity have been identified (Farooqi and O’Rahilly 2006), it has been difficult to identify common gene variants related to common, polygenic obesity. Earlier reports that common variants in *GAD2*, *ENPP1* and *INSIG2* genes are related to BMI variation have not been widely replicated (Frayling TM et al 2007). However, the results of recent genome-wide association studies are more encouraging. During a genome-wide search for Type 2-diabetes susceptibility genes Frayling et al identified a common variant in the *FTO* (fat mass and obesity associated) gene that disposes to diabetes through an effect on BMI. The 16% of adults in the study who were homozygous for the risk allele weighed 3kg more and had a 1.67 fold increased odds of obesity when compared to those not inheriting a risk allele. This association was seen from 7 years upwards and reflected a specific increase in fat mass (Frayling TM et al 2007). Further exploration of this genetic variant is ongoing; recent findings suggest that it may have effects on food intake

and food type (Cecil JE et al 2008) and that it's effect on body fat accumulation may be blunted by physical activity level (Rampersaud E et al, 2008, Andreasen CH et al, 2008). These findings may help to explain the interplay of genetic susceptibility and environmental change in the developed world that have led to recent rapid rises in obesity. The identification of genetic variants linked to population-wide variation in adiposity levels may in due course be of great use in clarifying the adverse outcomes of excess adiposity, when allied with the conceptual approaches of Mendelian randomization. Conventional observational epidemiological studies are subject to residual confounding from a variety of environmental and social factors. However, in studies using genetic variants the Mendelian randomization provides in itself a naturally occurring version of a randomized controlled trial (Ebrahim S, Davey Smith G, 2008). One could say that instead of the environment being manipulated as in a conventional trial, it is the effect of the environment on the person that is altered, by their genetic makeup. With securely identified gene variants influencing adiposity, it will be possible to examine the causal nature of the associations between adiposity and both disease outcomes (e.g. type 2 diabetes, coronary heart disease) and risk factors (e.g. blood pressure, insulin resistance). The difficulties with this approach include the need for large sample sizes, non-replication of findings and lack of functional genetic variants related to the pathway of interest (Ebrahim S, Davey Smith G, 2008).

In conclusion; parental BMI / parental overweight and obesity appear to be a determinant of offspring BMI, through both genetic and environmental factors. Genetic variants may allow further understanding of outcomes of adiposity, through Mendelian randomization. Studies that compared other measures of child adiposity with adult BMI were not found.

## **2.6 CONSEQUENCES OF OBESITY**

Excessive adiposity, which is increasingly common in the general population, has several well-documented adverse consequences in adults. Higher levels of adult adiposity show positive graded associations with risks of coronary heart disease and stroke, type 2 diabetes, osteoarthritis, gallbladder disease, sleep apnoea and respiratory complications and with several cancers, particularly those involving the endometrium, breast, and colon. Excessive adiposity is also associated with adverse psychological and social consequences. The relations with

coronary heart disease and stroke are at least partly accounted for by associations between excessive adiposity and increased levels of blood pressure and total and LDL cholesterol (Sorof, Daniels 2002). Increased adiposity is also strongly associated with insulin resistance, a precursor of type 2 diabetes, and with the metabolic syndrome, a combination of metabolic abnormalities associated both with risks of type 2 diabetes and cardiovascular disease (Larsson et al, 1984 {men}, Lapidus et al, 1984 {women}). These long term risks may apply to children through tracking of excess adiposity into adult life, which is particularly strong when the excess is severe, present at older ages, and is associated with parental obesity (Reilly et al, 2003). Several reports, particularly from the Bogalusa Heart Study, have suggested that excessive adiposity in childhood may be directly related to adverse levels of cardiovascular risk factors in adult life (Bao et al, 1997, Smoak et al, 1987, Wattigney et al, 1995, Freedman et al, 2001). Moreover, several studies have suggested that adiposity in later childhood may be positively associated with later coronary heart disease (Baker et al, 2007, Willett et al, 1995) though few of these studies can separate the independent influence of childhood adiposity from continuing adiposity in adult life.

### **2.6.1 ADVERSE PROFILE OF CARDIOVASCULAR FACTORS IN CHILDHOOD AND ADOLESCENCE WITH ADIPOSITY**

Childhood adiposity has strong short-term associations with less favourable levels of cardiovascular risk factors. Excess adiposity is associated with higher mean blood pressure, raised total and LDL cholesterol, triglycerides and low HDL levels (Freedman et al, 1999, Morrison et al, 1999a, b, Maffeis et al, 2001, Berenson et al, 1998). Prospective, long-term follow up studies suggest that obesity in childhood increases the risk of cardiovascular disease in later life; in the Bogalusa Heart study multiple cardiovascular risk factors (including SBP, fasting insulin and total cholesterol/HDL ratio) were shown to track through from childhood to teens or early adulthood. These patterns were strongest in the highest ponderal index tertile (Bao et al, 1994). Excess weight gain over childhood and adolescence has been shown to be more important than childhood weight per se (Sinaiko et al, 1999) i.e. when looking at young adults cardiovascular health the pattern in adolescence was more relevant than the weight at age 7.

Clinically, increasing numbers of young people are developing non-insulin dependent diabetes mellitus in association with adiposity (Ehtisham et al, 2000). In young people it is not so clear whether central adiposity is important in the development of insulin resistance as it is in adults; body fat distribution changes with puberty which may obscure the picture. In studies using body imaging (MRI or DEXA scan) in non-obese children insulin production and function was related to total body fat and subcutaneous abdominal fat (Goran, Gower 1998, Tessari et al, 1995); visceral fat was increased in some obese children, and if present was associated with insulin resistance. Others (Daniels et al, 1999) have found using DEXA scan that central adiposity in children is related more strongly than percent body fat to lipid profile (adverse lipid profile is an outcome of insulin resistance in adults; insulin levels were not reported in this study); blood pressure was most closely related to BMI, then central adiposity. There is also evidence that adiposity and obesity in children and adults are related directly to change in the structure and function of the arterial circulation, including early atherosclerosis (McGill et al, 2002) impaired endothelial function (Watts K et al, 2004 a and b) increased vascular resistance (Rocchini et al, 1992) and reduced arterial distensibility (Whincup et al, 2005). As part of the Muscatine study children were followed up three times between the ages of 6-18 and 29-37; calcification of the coronary arteries, denoting established atherosclerotic disease was positively associated with childhood weight, after controlling for other risk factors (Mahoney et al, 1996).

In summary there is evidence that in young people adiposity is associated with adverse change in the cardiovascular risk profile; general adiposity with raised blood pressure and increased fasting insulin, and obesity, particularly if associated with central obesity, with fasting insulin and insulin resistance.

## **2.7 CONCLUSION**

Excess adiposity is increasing worldwide with increases in children reported to be particularly rapid. No one anthropometric measure is ideal for the assessment of adiposity; body mass index (weight/height<sup>2</sup>) is the measure that has been most commonly used and for which there are widely available standards but it gives no indication of body shape or proportion of lean and fat mass. Waist circumference cut-offs are used in adults to define a group at high cardiovascular risk but there is less evidence for the role of central adiposity measures in

children. Health consequences of excess adiposity in young people include adverse lipid profile, insulin resistance and type 2 diabetes, hypertension and damage to arterial structure and function.

Adiposity in adults varies in a geographical pattern in Britain with higher levels of adiposity in the North and West. There is a lack of consistent evidence for these patterns in childhood. Adiposity also varies by social class and ethnic group in Britain with higher levels of excess adiposity in lower social classes and those of South Asian ethnic origin (particularly central body fat). These patterns seem to be emerging in studies of British adolescents. Individual determinants of adiposity include early life and familial factors. Breast feeding may have a small protective effect against excess adiposity (but does not influence mean BMI); the effect is prone to confounding, particularly by maternal BMI. Parental BMI / parental overweight and obesity appear to be a determinant of offspring BMI, through both genetic and environmental factors. Physical activity levels appear to be beneficially associated with adiposity levels depending upon method of collection of activity data and measure of adiposity used; little evidence of association with BMI was found. The issues raised in this review will be explored further in chapters 4-8.

## CHAPTER 3: DESIGN AND METHODOLOGY OF THE TEN TOWNS STUDY

### **3.0 SUMMARY**

This Chapter describes the methods of the Ten Towns Study, a mixed longitudinal, school based study of cardiovascular and respiratory health among children from ten towns in England and Wales. The study consisted of a series of surveys in 1990 (in 5-7 year-olds), in 1994 (in 8-11 year-olds) and in 1998-99 (in 13-16 year olds), each including some children who had taken part in the earlier surveys and some new children. The Thesis is based on cross-sectional data from the 1998-9 survey in which detailed assessments of anthropometry, blood pressure, pubertal status were made and fasting blood samples analysed for glucose, insulin, blood lipids and other parameters. Participant and parent questionnaires were completed and information on birth and infant growth records collected.

### **3.1 INTRODUCTON: RATIONALE FOR THE TEN TOWNS STUDY**

With the increasing evidence that the origins of cardiovascular disease lay in early life, the Ten Towns Studies were established in order to study whether there were systematic differences in the health of children living in areas of England and Wales with exceptionally high and exceptionally low rates of adult cardiovascular mortality (Whincup et al, 1992a, 1996). The principal investigators in the Ten Towns Study are Professor Peter Whincup and Professor Derek Cook, both now based in the Department of Public Health Sciences, St George's Hospital Medical School. To date, the Ten Towns Studies comprise three school-based surveys. The first survey ran from January 1990 to July 1990 (Whincup et al, 1992a), and the second from April 1994 to November 1994 (Whincup et al 1996). The third survey, on which this thesis is based, ran from August 1998 to July 1999. The cohort of children were aged 5-7 years in the first survey, 8-11 in the second and 13-16 in the third.

#### **3.1.1 SELECTION OF THE TOWNS**

Ten population centres (50 000 to 100 000 people) in England and Wales were selected on the basis of adult cardiovascular mortality (England and Wales, 1979-83, for men and women aged 35-64 years) to include the five centres with the lowest adult cardiovascular mortality and the five with the highest. One low mortality town that had already been involved in a similar

previous study, Guildford (Whincup et al, 1988) was excluded. A second low mortality town, Cambridge, which had current extensive school research commitments, was excluded because the Education Authority refused to allow schools to participate. The low mortality towns therefore included Esher, Leatherhead, Bath, Chelmsford and Tunbridge Wells. All five of the high mortality towns initially identified ((Wigan, Rochdale, Burnley, Port Talbot, Rhondda) were included. The geographic locations of the study towns are shown in Figure 3.1 (Whincup et al, 1992a, 1996). Table 3.1 summarises the standardised mortality ratios for cardiovascular disease in the study towns (varying by a factor of two between towns with high and low mortality). Statistical power considerations involved in the selection of five high and five low mortality towns are addressed in Section 3.2.3.

### **3.1.2 SELECTION OF THE SCHOOLS AND PARTICIPANTS**

For the first survey in 1990, a list of all the state primary schools in each town was obtained from the Local Education Authority and a random sample of ten schools, stratified by size and location (county primary schools only) and religious denomination (all schools), was chosen. All of the 100 schools originally invited agreed to take part. Two classes of children aged between 5 and 7.5 years from each school were randomly chosen, providing 50-60 children per school, all of whom were invited to participate. In the follow-up study in 1994 in each town the 10 junior schools corresponding to the 10 infant schools included in the earlier study were identified. Within each school the children who had taken part in the earlier study (30 on average) were invited, supplemented by a random sample of children (20 on average) from the same classes. The 1998-99 study was based in the secondary schools attended by at least 10 participants in the 1994 survey, on average 7 schools per town. All participants in the 1994 study attending these schools (45 per school on average) were invited to attend together with new pupils from the same class groups (13 per school on average) to replace pupils lost to follow up. Additional measurement sessions were held in which pupils surveyed in the earlier studies who attended other schools not being visited could participate.

### **3.1.3 STATISTICAL POWER CONSIDERATIONS**

By including ten towns; 5 high mortality, 5 low mortality; the study had an 80% power to detect a between-town difference of 2 standard deviations in any outcome measure at a P value of <0.05.

It was anticipated that at least 2,500 individuals would participate in body build measurements, which would enable the detection of differences in any body build outcome measure of less than 0.25 standard deviations between the highest and lowest fifths of any exposure measure with 90% power at a P value of <0.05. In addition, with more than 1,500 individuals in the study with blood measurements, it would be possible to detect a difference of less than 0.3 standard deviations in any outcome between the highest and lowest fifths of any exposure measure with 90% power at a P value of <0.05.

### **3.2 SURVEY PROCEDURES 1998-9**

Ethical approval was sought from the Ethics Advisory Committee of the Royal College of Paediatrics and Child Health. Approval was also sought from the local research ethics committee covering each town and from all relevant local education authorities. All subjects were invited to participate by a letter directed to the subject and his/her parent or carer. Information about the earlier studies and their results was also included, to encourage ongoing participation and to provide feedback. Informed written consent for study participation was sought from all the parents and children prior to the study.

All measurements were made between August 1998 and July 1999. The ten towns were visited in five pairs, each pair including one low mortality and one high mortality town. The pairing served to limit the effect of measurement drift and seasonal effects on comparisons between high and low mortality towns. The surveys within each town were conducted during a two week period; each pair of towns was normally surveyed within six weeks. Each school was visited for between one and four days, depending on the size of the school and number of study pupils attending. Children were ranked by age within each school; the older children measured were asked to have physical measurements and to provide a fasting blood sample; younger children were asked to have physical measurements only. The older children, who

had blood sampling, were surveyed in the morning after an overnight fast and the younger children in the late morning or early afternoon. The field team was made up of five members. A receptionist welcomed pupils, administered the pupil questionnaire and supervised the snacks given out after blood sampling. Three research nurses carried out the physical measurements and exercise testing and a research doctor did the blood sampling. Bioelectric impedance measurements were carried out either by the survey doctor (in the case of children providing a blood sample) or by a survey nurse (in the case of children not providing a blood sample). Each nurse carried out about one third of physical measurements in each town. During the course of the year there were two replacements of school nurses and also a change in receptionist.

Observers underwent formal training in the measurement of anthropometric measurements, which included demonstrations of all techniques. They then practised the measurements under supervision, to ensure that techniques used were appropriate. During pilot studies, all observers then made measurements in a group of 10 subjects in rotation, to ensure that mean values of each anthropometric measurements made by each observer differed by less than 5%. Observer techniques were then monitored regularly in the field by the supervising investigator. The overall mean values of anthropometric observations recorded by different observers were monitored on a town-by-town basis.

### **3.2.1 PHYSICAL MEASUREMENTS**

Children were examined whilst dressed in light, 'indoor' clothing without shoes.

#### **3.2.1.1 Height and weight**

Height was measured to the last complete millimetre with a portable stadiometer (CMS, Camden). Weight was measured to the last complete 0.1 kg with a digital electronic weighing scale (Soehnle).

#### **3.2.1.2 Waist and Hip circumference**

Waist circumference was measured at the end of normal expiration at the mid-point between the iliac crest and the lower edge of the ribs in the mid-axillary line, with the child standing with feet one foot apart. Hip circumference was measured at the point of maximum circumference over the buttocks.

### **3.2.1.3 Skinfold measurements**

Triceps, biceps, subscapular and suprailiac skinfolds were measured in accordance with the methods of Durnin and Rahaman. (Durnin, Rahaman, 1967).

The right side of the body was used. In each case the skinfold was grasped gently but firmly and calipers applied immediately below fingers, the reading being taken as soon as the caliper stabilized. Landmarks were as follows:

**Triceps skinfold:** With the elbow flexed at 90 degrees, the length of the upper arm (between the olecranon process and the acromial process) was measured with a steel tape measure and the midpoint identified and marked at half of the length. The skinfold was then taken at this point posteriorly, with the arm pendant.

**Subscapular skinfold:** This was measured immediately below the tip of the scapula, at 45° to vertical.

**Biceps skinfold:** The mid-point of the upper arm was identified as for triceps above. Biceps skinfold was then taken at this point anteriorly, with the arm pendant and supinated.

**Suprailiac skinfold:** This was taken immediately above the iliac crest in the mid-axillary line, above a vertical fold of skin.

### **3.2.1.4 Bioimpedance measurement**

Bioimpedance measurement was performed using the Bodystat 500 in accordance with the manufacturer's instructions, using the left side of the body. This measurement involves passing an alternating current between electrodes on the hand and foot. The impedance of the body reflects the relative amounts of conducting material (i.e. intra-and extra-cellular water) compared to insulating material (i.e. fat). The pupil had to lie down supine, with arms pronated, palms flat on the couch. Crucially there had to be no contact between limbs e.g., hand must not touch thigh, or legs be in contact with each other as this would allow the current to short-circuit and invalidate the reading. The body fat percentage is then calculated from the bioelectric impedance value produced using equations, (see statistical methods section below).

### **3.2.1.5 Blood Pressure**

BP measurements were made with the child sitting quietly after 5 minutes rest. Two readings were taken one minute apart using a Dinamap 1846SX oscillometric blood pressure recorder (Critikon, United States) on the right arm, which was supported at chest level (Ramsey 1979). Upper arm circumference was measured at the mid-point, with the arm pendant, using a steel

tape measure. If the child's arm circumference was less than 28cm then the small adult cuff size (cuff bladder dimensions 22 cm x 10 cm) was used. If greater than 28cm then the standard adult cuff was used, thereby ensuring that the minimum cuff bladder width to arm circumference ratio of 40% recommended by the American Heart Association (Frohlich et al, 1988) was met for the study population. Room temperature was measured at the time of blood pressure measurement with an RS electronic thermometer. The Dinamap systematically records SBP about 8mm Hg higher than a mercury sphygmomanometer, but diastolic readings are virtually identical (Whincup et al. 1992c).

#### **3.2.1.6 Standardization of measurement equipment over time**

Field procedures were regularly reviewed by the Principal Investigator. All measurement equipment was checked daily. The scales were checked and calibrated using standard weights before the study and every three months. Skinfold calipers and bioimpedance monitors were checked every three months by the suppliers. There was no evidence of any measurement drift in any of the measurements made.

### **3.2.2 BLOOD SAMPLING**

Blood samples were taken after an overnight fast, after physical measurements (including BP) were completed, and at least 30 minutes after the administration of local anaesthetic skin cream (EMLA or Ametop). All children were offered breakfast after the procedure. Blood samples were centrifuged, separated, and frozen at -20°C within eight hours of collection. All analyses were carried out in a central laboratory (Department of Clinical Biochemistry, Royal Free Hospital School of Medicine). Plasma glucose concentration (fluoride-oxalate sample) was measured using the method of Trinder (Trinder 1969) and a Falcor 600 automated analyser (Menarini). Plasma total cholesterol and HDL-cholesterol were measured using the methods of Siedel (Siedel et al 1983) and Sugiuchi (Sugiuchi et al 1995) respectively using a Hitachi 747 automated analyser. All reagents were supplied by Roche Diagnostics. Plasma LDL-cholesterol was calculated from HDL- and total cholesterol using standard equations (Friedewald et al 1972). Serum insulin was measured at the Department of Diabetes, University of Newcastle-on-Tyne using an ELISA assay which does not cross-react with proinsulin (Andersen L et al 1993).

### **3.2.3 EXERCISE TESTING/ SPIROMETRY**

Following blood testing and physical measurements children were given an exercise test on a stationary exercise bike. Pre- and post-test spirometry was also performed. This data is not used in this thesis and is not detailed further here.

### **3.2.4 HANDLING OF ABNORMAL BLOOD PRESSURE RESULTS**

For the purposes of this study abnormally high readings were defined as an average SBP equal to or greater than 140mmHg and/or average DBP equal to or greater than 90mmHg in accordance with British Hypertension Society guidelines of 1993 and 1999 (Ramsay et al, 1999, Sever et al, 1993). The height and weight of the child were recorded and the project leader was informed of the names and details of these children. He then contacted parents to seek their agreement to pass on information to their General Practitioner.

For cholesterol measurements the recommendations of the British Hyperlipidaemia Society were followed. If the total cholesterol value was 6.7 mmol/l or above, a letter was written to parents explaining that their child's cholesterol level was high and should be rechecked. Permission was sought from the parent to write to the child's family doctor with details of the blood lipid results.

### **3.2.5 OTHER INFORMATION**

Ethnic group was assessed on the basis of the child's appearance into five main groupings (white, African-Caribbean, Asian, Oriental, other). In the current report ethnic groups were analyzed as white and Asian and combined other group; the numbers in the combined group were small; results for white and Asian groups only are presented here.

### **3.3 QUESTIONNAIRE INFORMATION**

Information was collected from both children and parents via questionnaire.

#### **3.3.1 PUPIL QUESTIONNAIRE**

Pupils completed a questionnaire on the day of the study, with support from the research team if needed. This questionnaire (see Appendix B) sought information about participant date of birth, medications, respiratory symptoms, allergies/atopy, smoking and drinking. It included enquiries about physical activity and sedentary activities (e.g. TV watching), and parental occupation, to allow ascertainment of social class if the parental questionnaire was not returned (see 3.3.1 above and Chapter 5). There were also questions about educational aims and the Strengths and Difficulties Questionnaire (Goodman 1997). Details of the source of the questions used in this thesis and their validation are shown in Table 3.2. Wherever possible standard questions were used. Where this was not the case we developed the questions and tested them in the pilot before use in the main study.

#### **3.3.2 PARENTAL QUESTIONNAIRE**

A reply-paid, self-administered questionnaire was sent to the parents of the participants on the day of the physical measurements. If parents failed to return this questionnaire up to two reminder letters, which included a shortened version of the questionnaire, were sent. The parental questionnaire (Questionnaire 'WQRS99' –see Appendix A) covered details of the child's date of birth, child's medical history, food frequency table and questions about the child's activity level. There were detailed questions about respiratory symptoms (important for the exercise testing part of the study) and about the wider family. These included family health, parental education and employment, car ownership and details about family accommodation. If the child was new to the study (Questionnaire 'YQRS99') there were additional questions about birth weight, gestation, whether a singleton birth and early feeding. For these children the final page of the parent questionnaire asked for the child's place of birth and for consent to examine the birth record. For children who had participated in earlier surveys in 1990 or 1994 this information was already available, from the use of similar questions. The parental questionnaire was piloted in the London schools which we visited before the start of the main study to check that the questions were understood and the consistency of responses. Details of the source of the questions used for this thesis and their

validation are given in Table 3.2. Social class was coded from parental occupation in accordance with the registrar general's (ONS) 1990 coding manual. In accordance with ONS practice, social class of the head of household was used, based on the occupation of the father when present; if absent then mother's social class was used.

### **3.3.3 PUBERTAL STATUS**

Self-assessment questionnaires were developed using simple line drawings based on Tanner developmental stages, using penile/scrotal size and pubic hair growth for boys, breast size and pubic hair in girls. These questionnaires were completed in privacy. Girls were also asked whether they had started their periods and, if so, their age (years and months) at the first period. The questionnaires were validated against direct inspection by a trained observer in a Paediatric Endocrinology clinic (Taylor SJ et al 2001). A copy of the questionnaire is found at Appendix C.

### **3.4 THE DATASETS USED IN THIS THESIS**

This thesis uses data from the 1998-99 survey. Figure 3.2 gives a diagrammatic view of the main study data sets.

### **3.5 DERIVED BODY BUILD AND BODY COMPOSITION MEASURES**

Body mass index was calculated for each child. Body mass index =  $W / H^2$  where  $W$  = weight (kg),  $H$  = height (m). Body mass index was a widely used measure of weight-for-height. Bioimpedance measurement was converted to give fat free mass and thus a fat percentage using the generic equation of Deurenberg et al (1990a) which was based on a study of 246 children/young adults, aged 7-25, (school-and college-pupils) and used body fat percentage derived from densitometry from underwater weighing as the reference point to develop the bioimpedance equation in addition to anthropometric measurements.

The equation is applicable to both sexes, 'all ages', the formula is:

$$\text{Fat Free mass} = 0.438 \times 10^4 \times (\text{height}^2 / \text{biompedance measure}) + (0.308 \times \text{weight}) + 1.6 \text{ (if male)} + (7.04 \times \text{height}) - 8.5.$$

These equations produced body fat percentages which were highly correlated with BMI and log sum skinfolds (see Chapter 4). Fat percentages produced using this and alternative

equations (Houtkooper LB et al 1992, Deurenberg et al 1991) were closely related (correlation 0.94-0.99).

### **3.6 STATISTICAL METHODS**

All data were analysed using the SAS system statistical software package (SAS Institute, North Carolina, USA). Multiple linear regression (proc GLM procedure) was used to adjust relationships between variables for structural (e.g. room temperature, town, observer) and confounding (e.g. pubertal status, age) effects. Mean values of covariates of interest (e.g. BMI by town) were simultaneously adjusted for potential confounding variables (e.g. age) by using the SAS least squares means procedure (proc LS means). Using the LS means function the potential confounding variables were treated as categorical variables e.g. sex or as continuous variables e.g. age as in the other analyses using multiple linear regression. Each individual's potential confounding variables are adjusted to the mean within each category or distribution. For each subject the date of birth and date of examination were entered as day, month and year using the SAS date function. These two dates were used to generate the subject's age at examination. Models included adjustment for sex and observer (where appropriate) as categorical variables and age as a continuous variable, except where stated otherwise. Further adjustments are made for town, ethnicity and pubertal status (all categorical variables), where appropriate, from Chapter 5 onwards. Observer, social class and ethnic group (where used) were fitted as dummy variables to minimise the effect of unequal distribution of subjects in these groups. Tests for gender interaction were carried out where indicated; for example for body mass index , testing for interaction between gender and social class, the following SAS formula was used (brackets added for clarity):

proc glm; class (sex);

model (body mass index) = (sex) (social class) (sex\*social class) / solution.

In generic terms this can represented as:

$$Y = A + B_1X_1 + B_2X_2 + (B_1X_1 * B_2X_2) + \text{error.}$$

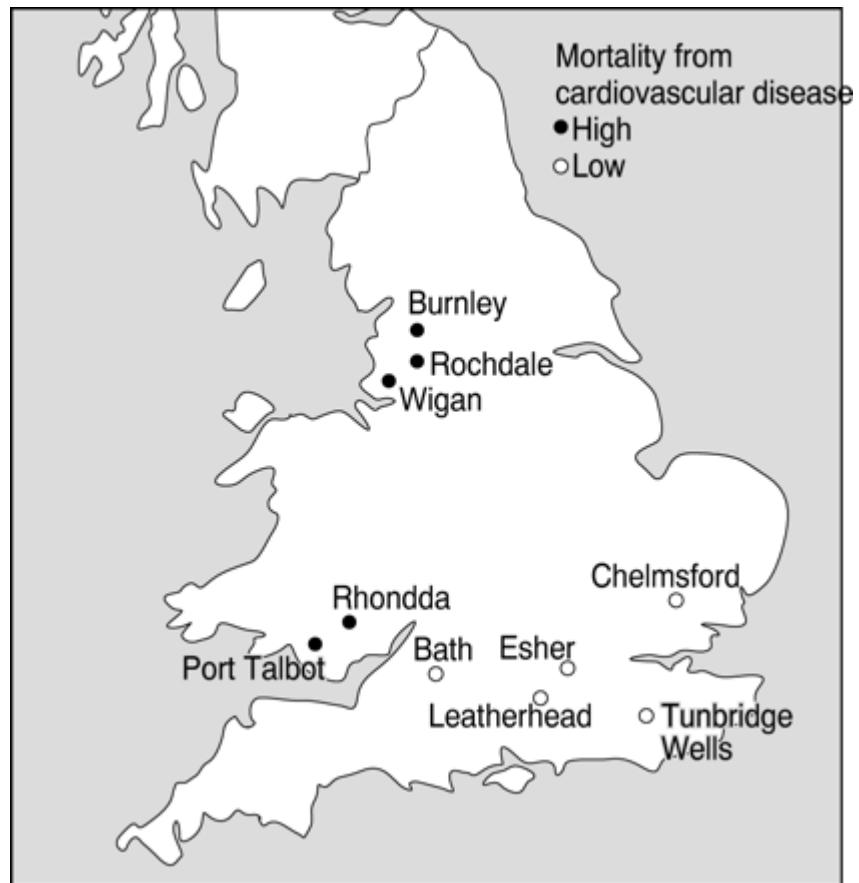
Additional adjustments for age, pubertal status and town were included, not shown here.

Comparisons of high and low mortality towns were carried out on a paired basis by applying paired *t* tests on 4df where necessary. This analysis took variations within as well as between towns in each factor into account. Paired analyses were used to minimise the effects of

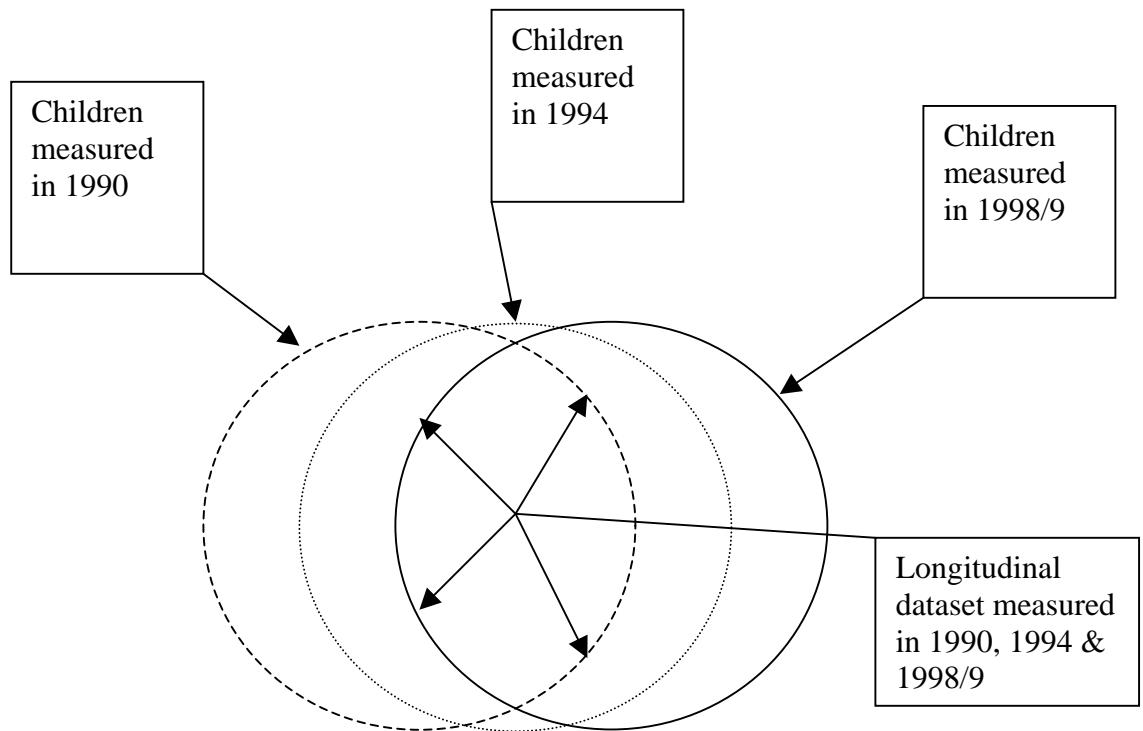
measurement drift, seasonal factors, and the change in average age. All differences have been represented as high mortality town minus low mortality town. Body mass index and some other variables were included both as continuous variables and at times as fifths (dividing subjects into fifths of the distribution, for descriptive purposes).

The strength of any associations seen in all results were commented on both in terms of the magnitude of the effect and also in terms of p-values. When used to denote significance of results, p-value of 0.05 was used as the cut-off, in line with common convention. P value of  $<0.05$  indicates that the association seen has less than 5% likelihood of happening by chance and is likely to reflect a genuine association (although not necessarily causal). This is described as 'significant'. Associations that have a p-value greater than 0.05 may also be genuine associations, but have not been demonstrated to achieve conventional statistical significance in this study.

**Figure 3. 1: Map showing location of study towns.**



**Figure 3. 2 The relationship between the different Ten Towns data sets.  
ONLY THE 1998/9 DATASET IS USED IN THIS THESIS**



**Table 3. 1: Cardiovascular mortality, infant mortality, and prevalence of low birth weight in 10 study towns**

Area	Survey pair	Standardised mortality ratio for cardiovascular disease *	Infant mortality 1000/year # (No. of deaths)	Prevalence (%) of low birth weight <2500g# (No. of births)
<b><i>Low mortality</i></b>				
Esher	1	64	7.2 (46)	6.3 (6469)
Chelmsford	2	71	8.2 (90)	5.7 (11050)
Leatherhead	3	70	7.1 (34)	6.1 (4819)
Bath	4	75	9.0 (123)	6.3 (13794)
Tunbridge Wells	5	71	7.7 (49)	6.2 (6373)
<b>Mean (low)</b>		<b>70</b>	<b>7.8</b>	<b>6.1</b>
<b><i>High mortality</i></b>				
Wigan	1	140	8.4 (105)	7.3 (12537)
Port Talbot	2	143	5.0 (22)	5.7 (4421)
Burnley	3	131	14.9 (150)	7.9 (10356)
Rochdale	4	136	10.8 (103)	8.4 (9662)
Rhondda	5	138	10.7 (34)	9.2 (5421)
<b>Mean (high)</b>		<b>138</b>	<b>10.0</b>	<b>7.7</b>

\*Based on deaths in men and women aged 35-64 years in England and Wales 1979-83.

#Based on years 1983-5. Source: OPCS Monitor (DH3 series).

(Table from Whincup PH et al 1996)

**Table 3. 2: Origins and validation of questions in parental (Appendix A) and pupil (Appendix B) questionnaires used in this thesis.**

<b>Question number/s</b>	<b>Area covered by question</b>	<b>Source of question</b>	<b>Comment on validation</b>
5.0 Parental	Dietary habits/ food frequency	Created by investigators	Used in earlier studies
6.0, 6.1 Parental 8.10, 8.11 Child	Physical activity	Strazzullo P et al. Leisure time physical activity and blood pressure in school children <i>American Journal of Epidemiology</i> , 1988;127:726-33 Also used in 1994 survey.	
8.1-8.9 Child	Physical activity	Created by investigators	Pilotted for consistency of responses
9.0 Child	Television, video and computer games	Created by investigators	Pilotted for consistency of responses
10.0-10.5, 13.0-13.5 Parental	Parental occupation/Social class	Based on 1991 census	

## CHAPTER 4: BODY SIZE AND ADIPOSITY IN 13-16 YEAR OLDS RELATIONS TO AGE, GENDER AND PUBERTAL STATUS

### **4.0 SUMMARY POINTS**

- Data are presented on the patterns of body size and adiposity in a school-based cross-sectional survey of 2645 British children aged 12.7 to 16.4 years (1235 girls and 1410 boys).
- Anthropometric measures included height, weight, four individual skinfold thicknesses and waist and hip circumferences. Bioelectric impedance measurements were also obtained. A full set of measurements were obtained on 1197 girls and 1370 boys.
- Derived measures of adiposity (body mass index, waist: hip ratio, sum of skinfolds and body fat percent from bioimpedance) were calculated, using standard formulae. These derived measures were then compared with each other and the basic anthropometrical measurements.
- The distributions of height and body fat percent were normally distributed. Weight, body mass index, individual skinfolds and the sum of four skinfolds, waist circumference and waist: hip ratio all showed marked right skew which was improved by log transformation (used in future analyses).
- Observer variation was present for all body size measures except height, weight and BMI. These differences persisted after adjustment for age and gender.
- Gender had a significant effect on body size and adiposity variables: boys were taller, heavier and had higher central adiposity measures than girls (waist circumference, WHR, STR). Girls had slightly higher general adiposity measures, including body mass index, sum of skinfolds and percent body fat (from bioimpedance), roughly a third higher than boys. BMI alone did not convey the extent of these gender differences.
- Age was strongly and statistically significantly related to body size and general adiposity measures. Height, weight and BMI all increase with age, between 12.7 and 16.4 years boys' height increase of over 20cm is double that in girls. Similarly boys' weight increased 20kg and girls 15kg. BMI increased in both genders to over 20 with increasing age, girls BMI slightly higher than boys. Body fat percent from

bioimpedance and sum of skinfolds tended to increase with age in girls and decrease in boys. Of the central measures of adiposity waist circumference increased with increasing age in years but waist: hip ratio decreased. STR increased in boys but did not change in girls.

- There were strong and highly statistically significant increases in height, weight and BMI with increasing pubertal score in both genders. With increasing pubertal score, sum of skinfolds and percent body fat declined in boys but increased in girls; these were all statistically significant changes. Adjustment for age only reduced these changes by up to a third; they remained highly statistically significant.
- Waist circumference increased in both genders with increasing pubertal score. Waist: hip ratio and subscapular: triceps ratio remained unchanged in girls, whilst in boys waist: hip ratio decreased and subscapular: triceps ratio increased. These effects remained after adjustment for age.
- Intercorrelations of adiposity measurements were strongest for general adiposity measures (BMI, weight, skinfolds, bioimpedance) and for waist circumference in both sexes; other central measures of adiposity did not correlate highly with each other.
- Comparison with the proposed international standards for obesity in childhood (Cole et al 2000) showed that almost 25% of boys and girls in the Ten Towns study were overweight, with approximately 10% being severely overweight or obese. Participants also showed higher than expected waist circumference measurements. These findings are in line with contemporaneous national and international trends and raise concerns about future health effects.

#### **4.1 INTRODUCTION AND OBJECTIVES**

In this chapter the distributions and interrelations of markers of body size and adiposity in 13-16 year old British school children in the Ten Towns Study are presented, and their relations to gender, observer, age, and pubertal stage. In the final section of this chapter there is a comparison of our study population with the proposed international standards of adiposity, compiled in 1999, from international data collected in the years 1963-1993 (Cole et al 2000).

The specific objectives for this chapter are:

- To explore the characteristics and distribution of anthropometric measures including height, weight, body mass index, waist and hip circumferences, waist: hip ratio, sum of skinfolds, and body fat percentage from bioimpedance (Section 4.3.1), and to decide suitable transformations prior to statistical analysis.
- To examine the effect of observer on the body size and adiposity measurements and to adjust for observer in subsequent analyses if necessary (Section 4.3.2).
- To examine the relationship between gender and the different measures of body size and adiposity (Section 4.3.3) and to adjust subsequent analyses if necessary.
- To examine the relationship between age and the different measures of body size and adiposity (Section 4.3.4), and to adjust subsequent analyses if necessary.
- To examine the relationship between pubertal status and the different measures of body size and adiposity (Section 4.3.5) and to adjust subsequent analyses if necessary.
- To examine the interrelationships of the different measures of body size and adiposity (Section 4.3.6) both before and after adjustment for age.
- To compare the body mass index distribution of the Ten Towns children with international data used to compile proposed international BMI charts, to examine the proportions of obese children in the study population (Section 4.3.7).

## **4.2 SUBJECTS AND METHODS**

This study is based on the findings from a school-based survey carried out in ten towns in England and Wales in 1998-99. Detailed information on subjects and methods and all measuring techniques are included in Chapter 3.

### **4.2.1 STATISTICAL METHODS**

All data were analysed using the SAS system statistical software package (SAS Institute, North Carolina, USA). SAS GCHART;HBAR command was used to produce basic histograms of variables, with set midpoints to allow ease of plotting. Multiple logistical regression was performed to obtain least squared means. Dummy variables were created and used for categorical variables where appropriate. Correlations were carried out using the PROC CORR procedure.

## **4.3 RESULTS**

Following invitation, in total 2645 subjects took part in the study (66% response rate) and had anthropometric measurements, of these 1410 were boys and 1235 were girls. Complete anthropometric measurements were made in 2567 subjects, 1370 boys and 1197 girls (97% of participants) (Table 4.1).

### **4.3.1 EXPLORATION OF THE CHARACTERISTICS AND DISTRIBUTION OF BODY SIZE AND ADIPOSITY MEASURES**

The distributions of the anthropometric measurements are displayed in Figures 4.1 to 4.8, for each gender separately. The distributions of height (figure 4.1) and body fat percent from bioimpedance (Figure 4.5) showed normal distributions. Weight, body mass index, sum of skinfold measurements, waist circumference and waist: hip ratio showed marked right skew (figures 4.2-4.4, 4.6, and 4.7). Log transformation helped to normalise these distributions (figures 4.2a -4.7a), which were log transformed in subsequent analysis. Subscapular:triceps ratio (figure 4.8) showed some right skew which was changed to left skew by log transformation (figure 4.8a); the variable was left unlogged in analyses.

### **4.3.2 EFFECT OF OBSERVER ON THE BODY SIZE MEASUREMENTS**

Six of nine observers involved in the study made physical measurements (all except observers 5,6 and 7). As shown in table 4.2, observer 3 took the highest number of physical measurements (930), followed by observers 2 (652), 1 (425), 9 (290) and 8 (262). Mean levels of anthropometric measurements made by each observer are shown in Table 4.2. There were significant differences (all  $p<0.005$ ) between observers for every variable except weight, height and BMI. These differences persisted after adjustment for age and gender. Subsequent analyses will include adjustment for observer where appropriate. For body fat percent from bioimpedance it was decided not to adjust for observer as the participant groups studied by the different observers were not similar. This was due to the study design, as the bioimpedance was measured in older children (having blood tests) by the study doctor in the morning and by study nurses in younger children in the afternoon. Observer 4 (study doctor) took 1114 bioimpedance measurements, followed by observer 3(574), 2(316), 1(237), 8(174) and 9(152).

### **4.3.3 EFFECT OF GENDER ON THE BODY SIZE MEASUREMENTS**

The distributions of anthropometric measurements in each gender are shown in Figures 4.1 to 4.8 and the mean (or geometric mean) values for each gender are shown in Table 4.3. Gender was related to all adiposity variables (p for sex difference < 0.0001), Table 4.3. Boys were on average taller and heavier than girls, and had a greater waist circumference, waist: hip ratio and subscapular: triceps ratio. Girls had larger body mass index, hip circumference, sum of skinfolds and total body fat percentage (from bioimpedance, and reflected in sum of skinfolds). Subsequent analyses will therefore be carried out separately for boys and girls or will be adjusted for gender, as appropriate.

### **4.3.4 EFFECT OF AGE ON THE BODY SIZE MEASUREMENTS**

The age range of participants was 12.6 to 16.4 years. 95.6% of participants were aged between 13.0 and 16.0 years, with 21 aged under 13.0 and 93 aged over 16.0.

#### **4.3.4.1 Age and general adiposity measures**

Of the general measures of body size and adiposity, height, weight and BMI all showed significant increases with increasing age in years (P<0.0001, Table 4.4). From a similar starting point at age 12 of around 154-155cm average height, boys average height rose about 20cm to 175cm, whilst girls rose about 10-11 cm to 165 cm. Boys' weight increased about 20kg from 45kg to over 65kg and girls' increased 15kg, from 45kg to around 60kg. BMI increased from 18.8 to 21.6 in boys and 18.8 to 22.2 in girls.

Body fat percent from bioimpedance increased with age in girls and sum of skinfolds decreased with age in boys. For boys sum of skinfolds decreased from 42.9 to 38.9, (p=0.02), whilst for girls it increased from 50.9 to 57.2 between the ages of 12 and 16, but this trend was not strongly graded and was not statistically significant (p=0.3). Percent body fat in boys showed no consistent pattern with age (p=0.7). For girls, percent body fat increased with age between 12 and 16 from 30.7 to 33.6 (p trend<0.0001).

#### **4.3.4.2 Age and central adiposity measures**

Of the central measures of adiposity waist: hip ratio decreased with increasing age, from 0.82 to 0.78 in boys (p trend =0.0003) and from 0.75 to 0.73 in girls (p trend=0.004). Waist

circumference showed significant increases with increasing age in years ( $P<0.0001$ ), as did STR in boys, from 0.60 to 0.85,  $p<0.0001$ . STR in girls showed no association with age. Adjusting for pubertal status reduced the size of some of the changes seen with age but they remained strongly significant.

#### **4.3.5 RELATIONS OF PUBERTAL STAGE TO BODY SIZE AND ADIPOSITY MEASUREMENTS**

Pubertal staging was ascertained by self-administered questionnaire, as detailed in Chapter 3. 5-10% of children assessed themselves as in early puberty (up to Tanner stage 2), with a few individuals pre-pubertal. The remaining participants were evenly divided between Tanner stages 3, 4 and 5 of puberty; 5 representing maturity. Roughly 30% of children were in each of these categories.

Table 4.5 shows correlations of the individual pubertal staging questions with adiposity measures.

For boys, both pubertal status questions 1 (gonadal development) and 2 (pubic hair) were correlated with age ( $r = 0.27, 0.22$  respectively), and height (0.33, 0.38). For girls the first question (breast development) was correlated with age and BMI ( $r = 0.20, 0.25$  respectively) and the second question was correlated with age and height ( $r = 0.22, 0.16$  respectively). All these correlations were statistically significant ( $p<0.05$ ). For further analyses a pubertal stage score was defined, based on the sum of scores for the two questions in each gender. Scores were grouped as 2, 3-4, 5-6, 7-8 and 9-10. These grouped scores broadly equate with Tanner stages 1 to 5 respectively. Since only one subject (a boy) had a score of 2 (Tanner stage 1), this category was omitted from the table. Table 4.6 shows the difference in body size and both general and central adiposity measures across increased pubertal stages for boys and girls for a combined pubertal stage score.

##### **4.3.5.1 Pubertal stage, body size and general adiposity measures**

In univariate analyses in boys and girls, there were highly significant increases in height, weight and BMI with increasing pubertal score in both genders. In boys sum of skinfolds and percent body fat declined and in girls they increased; these were all statistically significant changes. Boys' mean height increased by 19cm from Tanner score 2 to 5, from 154.5 to 173.4 cm. Girls' height increased from 155.7 to 162.4, almost 7cm increase. Equivalent figures for

weight are; boys 47.5kg to 62.6kg, girls 47.7kg to 58.0kg. BMI increased in boys by 2.9% per pubertal score increase, from 19.9 to 20.9 and 4.6% per score increase in girls, from 19.7 to 22.0. Body fat percent from bioimpedance decreased by 1.0 in boys per pubertal score, body fat percent was 26.1 at pubertal stage 2 and 21.6 at pubertal stage 5. In girls this variable showed an increase from 30.6 at stage 3 to 33.0 at stage 5, an increase overall of 0.8 per increase in Tanner stage. Sum of skinfolds in boys went from 49.4 to 39.0 a 4.4% decrease per Tanner stage change, and in girls from 52.3 to 62.2, an 8.3% increase per Tanner. The changes in sum of skinfolds and body fat percent from bioimpedance were all significant,  $p<0.0001$ , except sum of skinfolds in boys,  $p=0.003$ .

#### **4.3.5.2 Pubertal stage and central adiposity measures**

Waist circumference increased in both genders with increasing pubertal score. Waist: hip ratio and subscapular: triceps ratio remained unchanged in girls, whilst in boys waist: hip ratio decreased and subscapular: triceps ratio increased significantly with increasing Tanner score. Waist circumference increased from 69.8 to 72.8 in boys a 2.2% increase per Tanner stage and from 65.6 to 69.3 in girls, 2.1% per stage. Waist: hip ratio in boys was 0.81 at stage 2 Tanner score and 0.78 at stage 5, a 1% decrease per stage. Subscapular: triceps ratio increased from 2.04 to 2.35 in boys, 5.1% increase per Tanner stage.

#### **4.3.5.3 Effect of age adjustment on relationships seen between pubertal stage and adiposity measures**

Analyses were repeated with additional adjustment for age, as it was important to separate these changes with pubertal stage from the effect of age. Adjusting for age did not significantly alter the relationships seen between pubertal stage and adiposity measures (the regression coefficients are shown in the right hand column of Table 4.6 and 4.7). The size of the increases in height, weight and BMI in both boys and girls were reduced by up to a third by adjusting for age, but remained significant. Pubertal stage changes for sum of skinfolds and body fat percent from bioimpedance showed small alterations following additional age adjustment but remained significant in both genders.

Waist circumference in boys and girls and subscapular: triceps ratio in boys both showed a small reduction in pubertal stage change for these variables after age adjustment, but remained

significant. Adjusting for age had very little effect on the pubertal stage reduction in waist: hip ratio in boys. Waist: hip ratio and subscapular: triceps ratio remained unrelated to pubertal stage in girls after age adjustment.

#### **4.3.6 THE INTERRELATIONSHIPS OF DIFFERENT MEASURES OF ADIPOSITY**

Correlations of the different measures of adiposity were computed separately for boys and girls, in view of the significant gender differences described above. The correlations of anthropometry measures are shown in Tables 4.8 and 4.9. There were strong correlations between BMI and weight, waist circumference, skinfolds and body fat percent (bioimpedance), for both boys and girls (0.7-0.9). Waist: hip ratio was less strongly correlated to these measures, being most closely correlated to waist circumference (0.65), then to BMI (0.4). The relationships between BMI and these measures is also shown in scatterplots in Figure 4.11. Subscapular: triceps ratio is has the weakest correlations to other adiposity measures. Adjusting for age did not make any marked changes in the correlations, adjusted values are shown in tables 4.8 and 4.9 {in brackets}.

Skinfolds can be difficult to measure accurately; especially in the very obese. To explore this in the Ten Towns children, sum of skinfolds was divided into fifths of the distribution and the fifths were then correlated separately with adiposity variables (Table 4.10). Adiposity variables known to be highly correlated with skinfolds were used. Conversely to expectation, the correlations were found to be highest in the extremes of the distribution. This was true for both sexes. In the mid range where the results lie close together the correlations are weakest. The same pattern was present but not so strongly for fifths of BMI (Table 4.11).

#### **4.3.7 COMPARISON OF THE TEN TOWNS STUDY CHILDREN WITH PROPOSED INTERNATIONAL STANDARDS FOR OBESITY**

There is international agreement that an adult BMI of  $30\text{kg}/\text{m}^2$  or above defines adult obesity (WHO 1997). Similarly an adult BMI of  $25\text{kg}/\text{m}^2$  or above is often used to represent overweight. International standards for obesity in childhood have been proposed (Cole et al 2000), using data from a number of countries to produce centiles that pass through BMI of 25 or 30 at age 18, thus allowing some definition of overweight and obesity at other ages in

childhood. Our data were compared with these international centiles (represented for boys in Figure 4.9 and Table 4.12 and for girls in Figure 4.10 and Table 4.13). Tables 4.12 and 4.13 present the BMI centiles for boys and girls in the Ten Towns study by age in years and also the international centile values for BMI that define overweight and obesity at each age in years. It can be seen that the ten towns 75<sup>th</sup> centile is nearest to the BMI 25 (overweight) centile in both genders ie about 25% of the ten towns children are overweight and about 10% are obese, as the ten towns 90<sup>th</sup> centile is nearest to the BMI 30 (obese) centile. This is shown figuratively in Figures 4.9 and 4.10. The UK population data that contributed to the international centiles was collected in 1978-90 and was also used to create UK national BMI centiles (Cole et al 1995). Comparison with these charts shows that in both genders the Ten Towns study children's 50<sup>th</sup> centile for BMI approximates to the 75<sup>th</sup> centile on the charts (data not shown). In girls the prevalence of obesity appeared greater at age 15 than 16; this was not so clearly the case in boys.

## **4.4 DISCUSSION OF RESULTS**

### **4.4.1 PRINCIPAL FINDINGS**

Girls had higher mean levels of general adiposity markers, including slightly higher body mass index and appreciably higher values for sum of skinfolds and percent body fat (from bioimpedance), roughly a third higher than boys; these results suggest that total body fat was higher in girls. In contrast, boys were taller, heavier and had higher central adiposity measures than girls (waist circumference, waist: hip ratio, subscapular: triceps ratio). Age and pubertal stage had similar, independent effects on height, weight and BMI, all increased with age and pubertal stage. In boys sum of skinfolds and percent body fat declined with increasing age and pubertal stage and in girls they increased; these changes were more strongly related to pubertal stage than age. Of the central measures of adiposity, waist circumference was higher at older ages and pubertal stages in both genders whilst waist: hip ratio was lower or unchanged. Subscapular: triceps ratio increased in boys with age and pubertal stage but did not change in girls. General adiposity measures (BMI, weight, skinfolds, percent body fat from bioimpedance) and waist circumference were strongly intercorrelated in both sexes. Other measures of central adiposity did not correlate highly with each other. Comparison with the proposed international standards for obesity in childhood (Cole et al, 2000) showed that

almost 25% of boys and girls in the Ten Towns study were overweight, with around 10% being severely overweight or obese.

#### **4.4.2 STRENGTHS AND WEAKNESSES OF THE STUDY**

A major strength of this study is the use of a comprehensive range of easily conducted, non-invasive adiposity measures, designed to investigate the presence of both general and central adiposity. This enabled us to investigate the presence of an association not just with total body fatness but with abdominal fat. However, it was not possible to include gold-standard measures of adiposity (e.g. DXA scanning, magnetic resonance imaging). Measurements of thigh and calf skinfolds, which have recently been recommended as significantly enhancing estimation of percent body fat (Eston et al, 2005), were also not available. The study relied on self-assessment for pubertal status which was piloted prior to the main study and shown to be accurate in a clinic population (Taylor et al, 2001). However, its validity in a general population has not been assessed. Ascertainment of this sensitive information, even using a secret 'ballot box' behind a screen, was incomplete. Unfortunately 6% of boys spoilt their papers, resulting in loss of some subjects from elements of the analysis.

#### **4.4.3 RELATION OF FINDINGS TO OTHER STUDIES**

##### **4.4.3.1 Body size measurements**

The greater mean heights and weights observed in boys compared with girls are entirely expected, as in the standard UK Growth charts (Wright et al 2002). They are comparable to findings in the 2002 Health Survey for England for the ages 13, 14 and 15 (our groups aged 12 and 16 were smaller therefore less reliable). Although boys were heavier than girls, their BMI remained a little lower than girls at 12-16 (as their height increase was much greater); our findings were similar to HSE 2002 in this respect as well although their values for BMI were a little higher. The difference in height between boys and girls goes from girls being taller (+0.7cm) at 14 to boys being 10cm taller by age 15, reflecting dramatic pubertal changes. Similar changes were seen in a detailed study of Spanish adolescents in 1995; with boys 6cm taller at 14.5 years. (Moreno et al, 2001). There are parallel changes in body composition, with males gaining lean mass (as shown by increasing BMI) but skinfolds decreasing suggesting lower subcutaneous fat; overall body fat percent in boys did not change significantly. Possibly

this reflects gains in intra-abdominal fat as boys move to an adult male habitus. Certainly there are increases in both waist circumference and subscapular triceps ratio, theoretically reflecting central distribution of adiposity, although confusingly waist: hip ratio decreases; possibly reflecting higher gluteal muscle mass and lower abdominal subcutaneous fat. In girls the change in BMI is less dramatic, with skinfolds and body fat percent also increasing a little although there is no evidence of increasing centrality of fat distribution. These direction of these findings are similar to those of Moreno et al, (2007) in work on skinfolds and waist and hip circumferences in Spanish adolescents aged 13-18.5 years; although in both sexes the Spanish adolescents had higher waist circumferences and subscapular: triceps ratio. Body fat percent from bioimpedance gives high mean values for girls in our study; between 30 and 34% at ages 12-16. Proposed cut offs for excess body fat per cent in adolescent girls range between 30 and 35%, therefore almost half of the study girls were in the excess fat category. For boys cut offs range between 25 and 30% for 10-15 years olds and 20-25% for 15-18 year olds (Sardinha et al, 1999, Taylor et al, 2002, 2003, Weststrate 1989). It would be helpful to calculate body fat percent from skinfolds for comparison; however recent work in Spain in adolescents found that few of the equations used for this were reliable when cross-validated by Dexa scan (Rodríguez et al, 2005). Thigh and calf skinfold were not measured in our study, limiting calculation of body fat percent from skinfolds to those equations using sum of 4 skinfolds and therefore direct comparison with the Spanish results, using sum of 6 skinfolds is not possible. Their calculated (skinfold) body fat percentages for 13-18 year olds in 2000-2002 were noticeably lower than our bioimpedance derived body fat percentages; Spanish boys maximum 20.8% and girls maximum 26.3% (Moreno et al, 2006), our boys maximum 24.7% and girls 33.6%. Using sum of 4 skinfolds and the equations of Durnin and Rahaman (1967) our boys (maximum 22.2%) and girls (maximum 30.0%) still have higher body fat percentages; our children's BMI also tended to be a little higher; taken together there is the suggestion of greater adiposity in our study population than in Spanish children, although they had greater central fat distribution (Moreno et al, 2007), possibly illustrating ethnic differences in adiposity which will be explored further in chapter 5.

Comparison of our study population with a cross-sectional study of 9 and 15 year old Australian children in 1985 is interesting; this study aimed to define obesity by biological endpoint; using systolic blood pressure and dyslipoproteinaemia to dichotomise the body fat

distribution, calculating body fat percent from the equations of Durnin and Rahaman using four skinfolds (Dwyer, Blizzard 1996). Their conclusion was to propose a cut off range of 17-20% body fat in boys and 29-35% in girls, beyond the upper limits of these ranges the trend was towards higher SBP and lower HDL-cholesterol ie increasing cardiovascular risk.

Calculating our 15 year olds' body fat percent by this method gives means of 29.6% in girls and 19.7% in boys; over 10% of girls had more than 35% body fat and almost half the boys had 20% body fat; the higher cut offs of 25% and 30% in boys equated roughly to the 75<sup>th</sup> and 90<sup>th</sup> percentiles of the distribution (data not shown). So depending which cut offs are used up to half our study population had excess, undesirable fat levels.

Values for waist circumference were quite high in the study population; see further discussion under time trends section 4.5.3.3 below.

#### **4.4.3.2 The interrelationships of different measures of adiposity**

BMI, skinfolds, body fat percentage from bioimpedance and waist circumference were all similarly correlated in our study, in both genders, seeming to represent in that sense general adiposity. Correlation of BMI with sum of skinfolds of 0.81 was similar to the finding of 0.70-0.85 between the ages of 7 and 15 years by Lazarus (Lazarus et al, 1996). Sum of skinfolds were the most highly correlated to bioimpedance-derived body fat % in boys and just less so than BMI in girls (0.73 in boys, 0.79 in girls); however Rodríguez et al, (2005) found that most skinfold-thickness equations (to derive body fat percent) had poor accuracy at the individual level, making it more difficult to use skinfolds to identify those with 'risk associated' obesity and to present this in a meaningful way. To this extent adding skinfolds to BMI assessment adds value in epidemiological studies but perhaps not so much in clinical settings, particularly as with skinfolds there is much greater observer variation. However there may be a role for skinfolds in assessing the extremes of the BMI distribution ie in specialist clinical settings; this suggestion is supported by the following findings.

It might be expected that the correlations between skinfolds and other measures would be lowest in subjects with particularly high skinfold thickness as skinfolds can be difficult to measure accurately; especially in the very obese. However this was not seen when fifths of the skinfold distribution were correlated separately with adiposity variables (Table 4.10). Adiposity variables known to be highly correlated with skinfolds were used. Conversely to

expectation, the correlations were found to be highest in the extremes of the distribution. This was true for both sexes, and may reflect the difficulty in getting highly accurate skinfold measurements whatever the body shape. In the mid range where the results lie close together the correlations are weakest. The same pattern was present but not so strongly for fifths of BMI (Table 4.11). Perhaps it can be concluded from this that in clinical work skinfolds may be helpful in the very lean (or obese) in discriminating fat mass from lean mass; with increasing muscle mass and decreasing fat mass BMI may not change but skinfold thickness would. Putative markers of central adiposity, unlike those of general adiposity, did not correlate closely with each other as a group. Waist: hip ratio and subscapular: triceps ratio correlated more weakly than waist circumference with the measures of general adiposity. Subscapular: triceps ratio showed a negative relationship with sum of skinfolds and waist: hip ratio in boys; this may be due to confounding by puberty, as subscapular: triceps ratio increased with pubertal stage in boys and skinfolds sum and waist: hip ratio decreased. Baumgartner found a similar increase in subscapular: triceps ratio with pubertal changes in US males aged 7-30 (Baumgartner et al, 1986). Similar results were also obtained in 15 year old Australian school children, in whom a strong negative correlation between subscapular: triceps ratio and skinfold-derived body fat percent of -0.22, ( $p<0.0001$ ) was observed in boys, with no relationship in girls. (No significant correlation was found between subscapular: triceps ratio and BMI or waist: hip ratio in girls or boys) (Dwyer et al, 1996). Assuming for arguments' sake that subscapular:triceps ratio genuinely represents central (truncal) obesity it can be seen that in adolescent boys whose subcutaneous fat is decreasing due to pubertal changes there may be false reassurance from skinfolds; measures of central adiposity in addition seem to be warranted. The validity of waist circumference in this age group to reflect central fat has been investigated by DEXA scan (Brambilla et al, 2006, reviewing studies 1992-2004; Benfield et al, 2008) both concluding that waist circumference predicts two-thirds of the variation in intra-abdominal adipose tissue. I could not find similar evidence for subscapular: triceps ratio; to this extent it is difficult to be confident as to its usefulness in this age group. Waist circumference is more highly correlated than WHR to the other adiposity measures and is positively correlated to height, significantly so in boys, whereas in both sexes WHR is significantly negatively correlated to height. I would conclude that waist circumference may well be the best measure of central adiposity; particularly in girls. However, it is possible that

standardizing waist circumference for body size, particularly for height, may help to increase its precision. Waist: height ratio has been proposed as an alternative central measure; more work needs to be done on this. BMI is also not completely height-independent in this age group.

#### **4.4.3.3 Time trends: Comparison of the ten towns children with proposed international standards for obesity (BMI) and reference values from other studies**

Comparison of the Ten Towns Study population with earlier reference populations is possible both for body mass index and waist circumference. Comparison of the Ten Towns study population with external standards revealed higher levels of adiposity than expected in the study children, in keeping with increasing levels of adiposity in British children and raising concerns about future health effects. When comparing body mass index in the study population with proposed international centiles (based on children from US, UK, Singapore, Hong Kong, Brazil and the Netherlands) for boys the 75<sup>th</sup> centile for the study population ran just below the international 90<sup>th</sup> centile, and the girls' 75<sup>th</sup> centile mostly overlay the international 90<sup>th</sup> centile, meaning that at least double the number of children expected were above the 'overweight' centile ie 20%-25% rather than 10%. Similarly the study's 90<sup>th</sup> centile for both sexes lay between the international 90<sup>th</sup> ('overweight') and 99<sup>th</sup> ('obese') centiles and the study's 99<sup>th</sup> centile for both sexes was well above the international 99<sup>th</sup> centile.

Unequivocally the number of obese and overweight children in the study population was higher than expected when compared to international standards, with roughly 5% obese and 20% overweight. This appeared more marked in older age groups, particularly in girls.

A similar result is obtained by plotting mean BMI at each age in years (from Table 4.4) on to the Child Growth Foundation BMI charts, compiled by Cole et al (1995). The study population means lie on the 75<sup>th</sup> centile of these growth charts, which were produced using British children's measurements collected between 1978 and 1990, illustrating that the increase in adiposity in British children was well established 10 years ago when the study data were collected. The upswing at age 16 in both sexes in the higher study centiles (Figure 4.9, 4.10) may reflect the impact of lifestyle habits once the pubertal growth spurt has ceased, but must be viewed with caution in light of smaller study numbers in this age group. The finding of roughly 20% overweight and 5% obese in study children can be compared to similar results of the Health Survey for England 2002, which found that using the International classification

over a fifth of boys (21.8%) and over a quarter of girls (27.5%) aged 2-15 were either overweight or obese.

In the study population our 50<sup>th</sup> centile figures for waist circumference at ages 12-16 were higher than McCarthy's published figures, based on British children measured 10 years earlier in 1988 (McCarthy et al, 2001). Our 50<sup>th</sup> centile figures are nearly those of McCarthy's 75<sup>th</sup> centile, in both boys and girls, with greatest differences in younger boys and 16 year old girls (almost on McCarthy's 90<sup>th</sup> centile). This suggests a proportionately greater increase in central compared to general adiposity in our study population, as 50<sup>th</sup> centile for study population BMI did not reach the international (or British chart) 75<sup>th</sup> centiles. This evidence of increasing centrality is consistent with findings in other studies in adolescents in developed countries (McCarthy et al 2003, Moreno et al 2005, Rudolf et al 2004, Li C et al 2006).

#### **4.5 CONCLUSIONS AND IMPLICATIONS FOR AETIOLOGY AND PREVENTION**

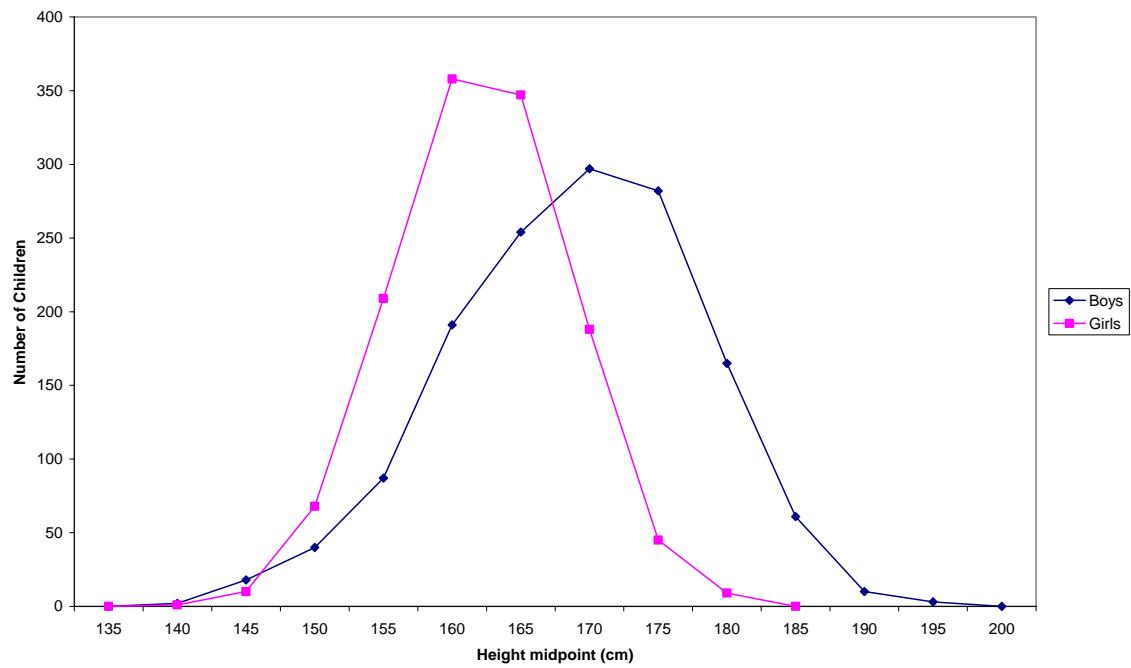
The results suggest that general adiposity in adolescents is relatively easy to define, with a cluster of variables including body mass index, sum of skinfolds and bioimpedance which are strongly clustered together. In contrast, putative markers of central adiposity (waist circumference, waist hip ratio, subscapular-triceps ratio) are less consistently inter-related, suggesting that these markers are measuring somewhat different properties.

In this study population, girls had higher levels of total body fat, a pattern consistent with the higher prevalences of obesity (based on markers of total body fat) generally observed in females from adolescence onwards. In contrast, boys had higher levels of markers of all the main putative markers of central adiposity, with higher waist, waist: hip ratio and subscapular: triceps ratio.

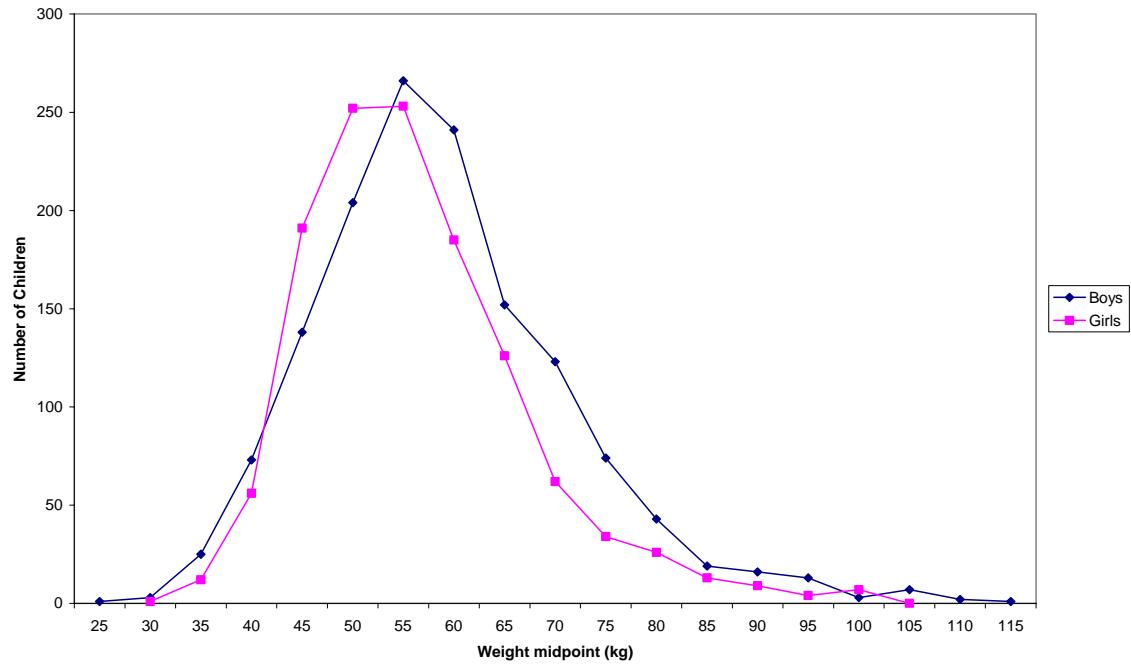
A striking feature of the results is the evidence provided by comparison with previous studies, suggesting that the distributions both of average body mass index and waist circumference have increased, with associated increases in the prevalence of overweight subjects. Assuming that these higher levels of body mass index and waist circumference persist with increasing age, this is likely to have important adverse implications. The increased development of overweight and obesity is likely to lead to a higher prevalence of overweight and obesity in adult life. The earlier accumulation of excess weight gain may well also lead to more severe

degrees of overweight and obesity in adult life. Both are likely to be associated with increased risks of chronic disease, particularly cardiovascular disease, type 2 diabetes and arthritis. Also potentially important is the likelihood that overweight and obesity of increased duration, starting from childhood and adolescence and persisting into adult life, will be a prominent feature in the next generation. If the duration as well as the degree of overweight and obesity is important for the development of chronic disease risk, this could further increase the adverse consequences. Evidence suggests that duration of obesity is particularly likely to be important for the development of type 2 diabetes risk (Wannamethee et al 1999); evidence that duration of obesity affects cardiovascular disease risk is less clear.

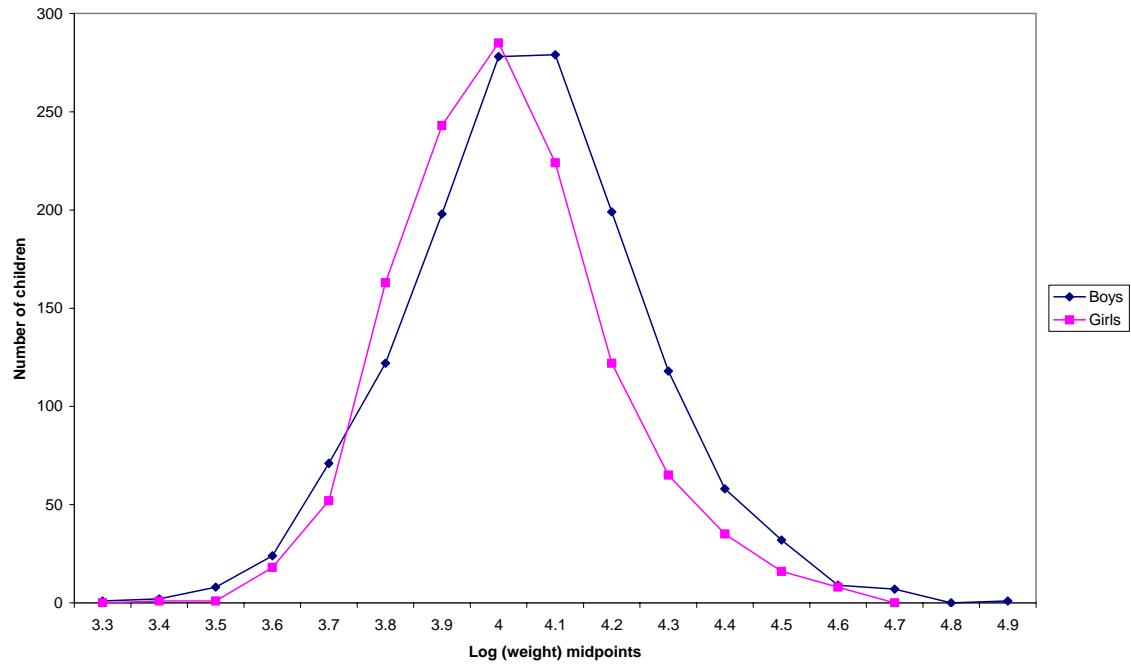
**Figure 4. 1: Height distribution**



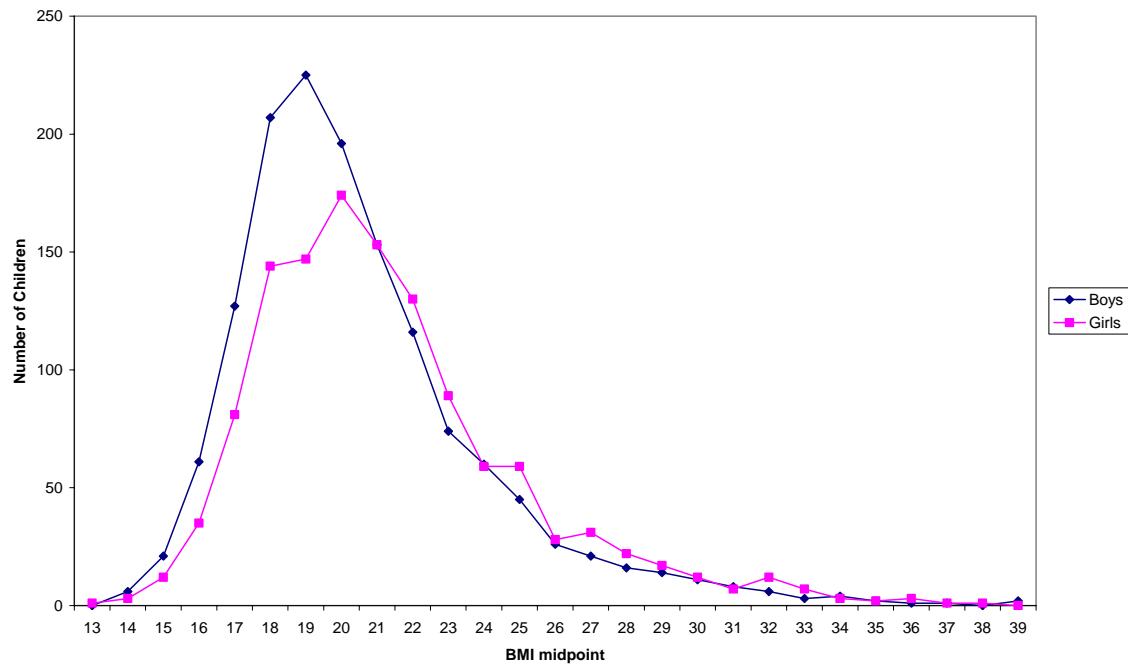
**Figure 4. 2: Weight distribution**



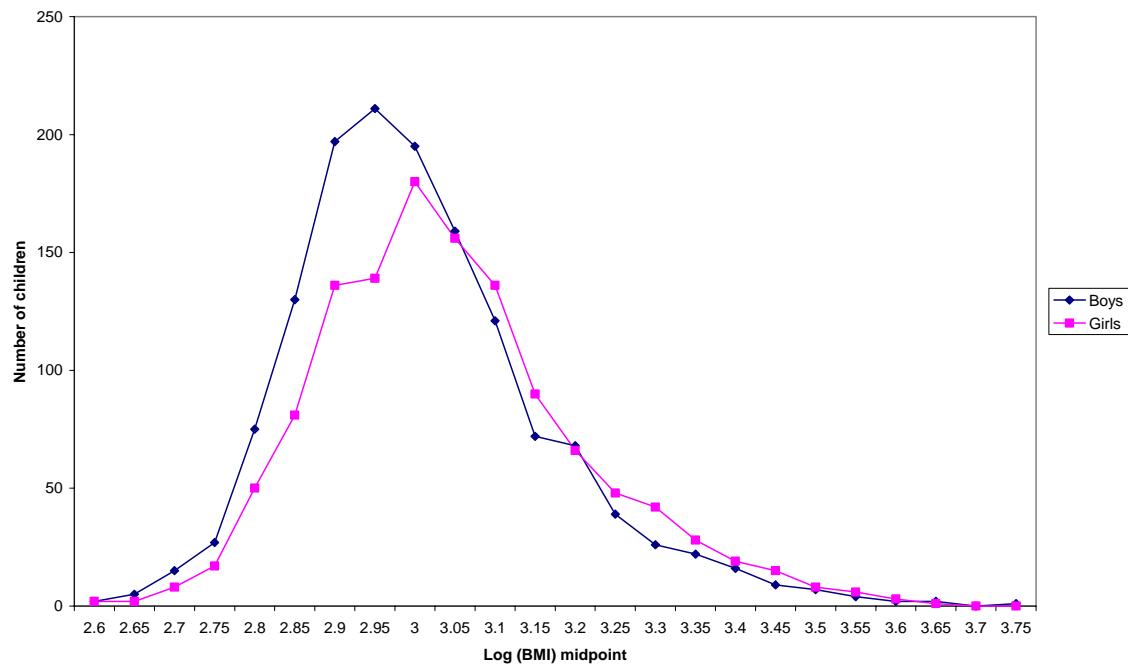
**Figure 4.2a Log (weight) distribution**



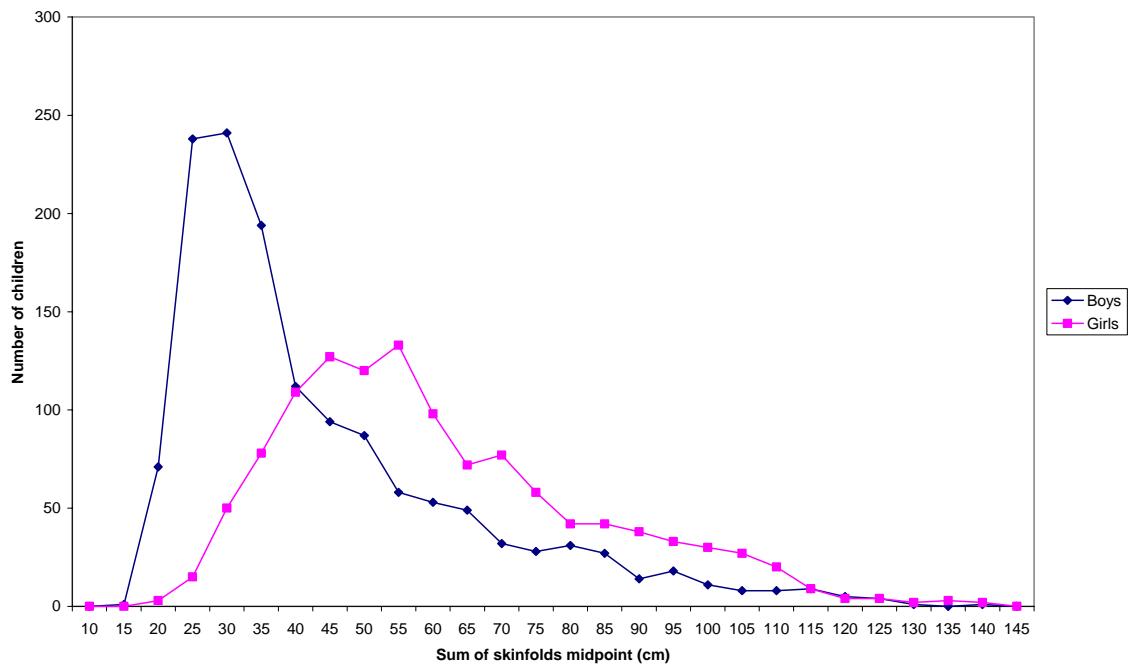
**Figure 4. 3: Body Mass Index (BMI) distribution (single value of 42 omitted, boy)**



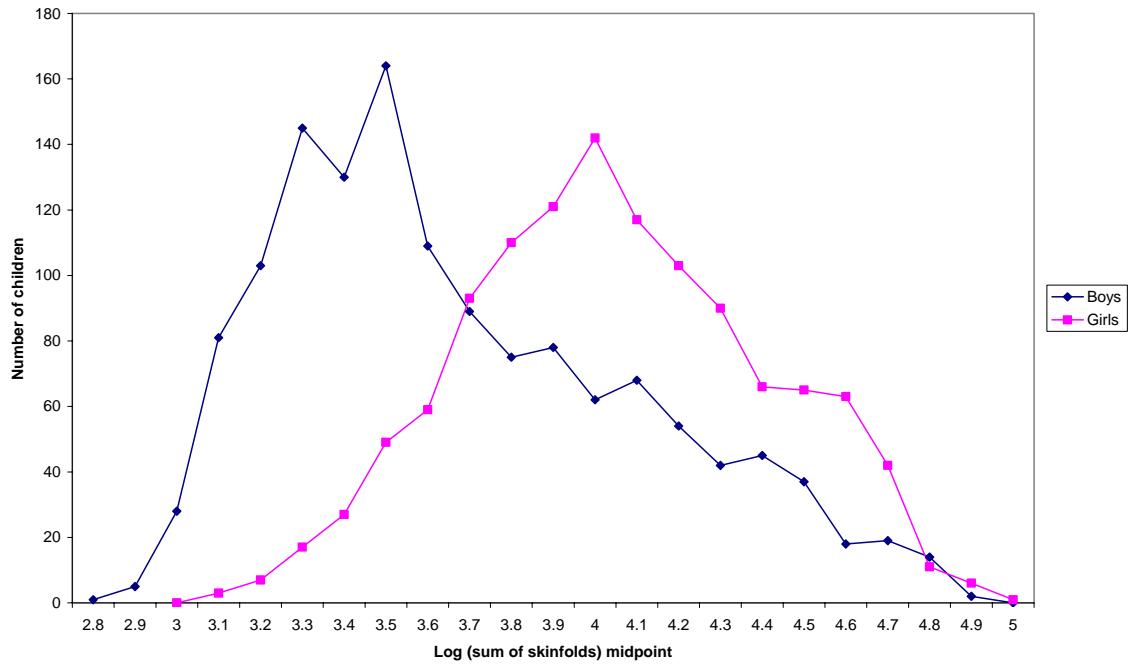
**Figure 4.3a Log (BMI) distribution**



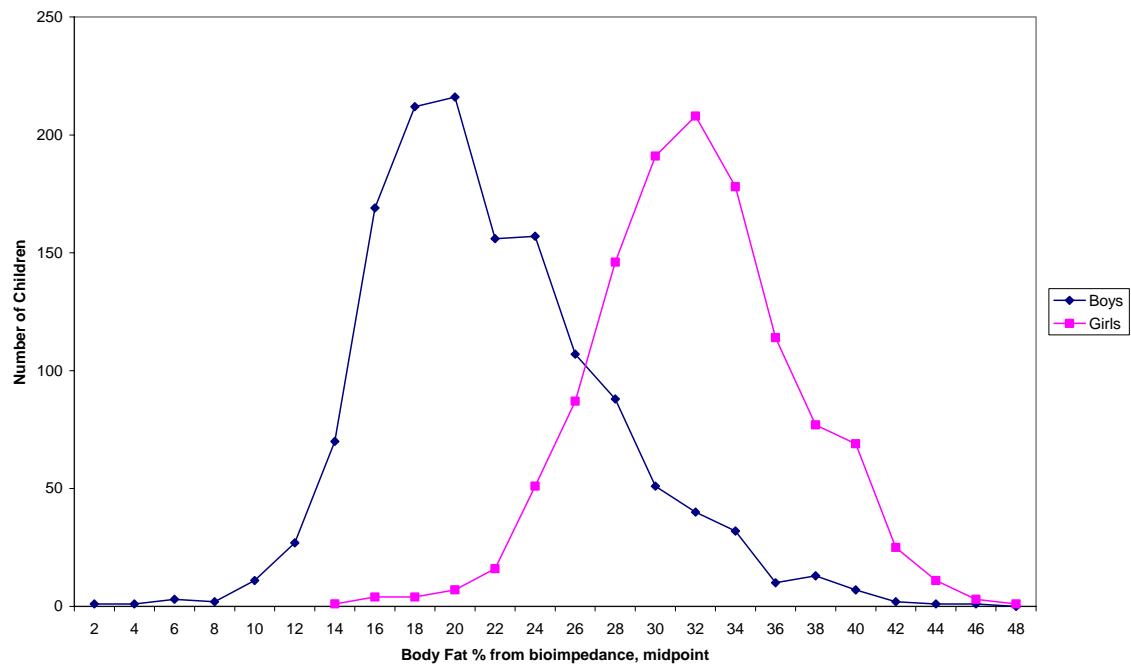
**Figure 4. 4: Sum of Skinfolds distribution**



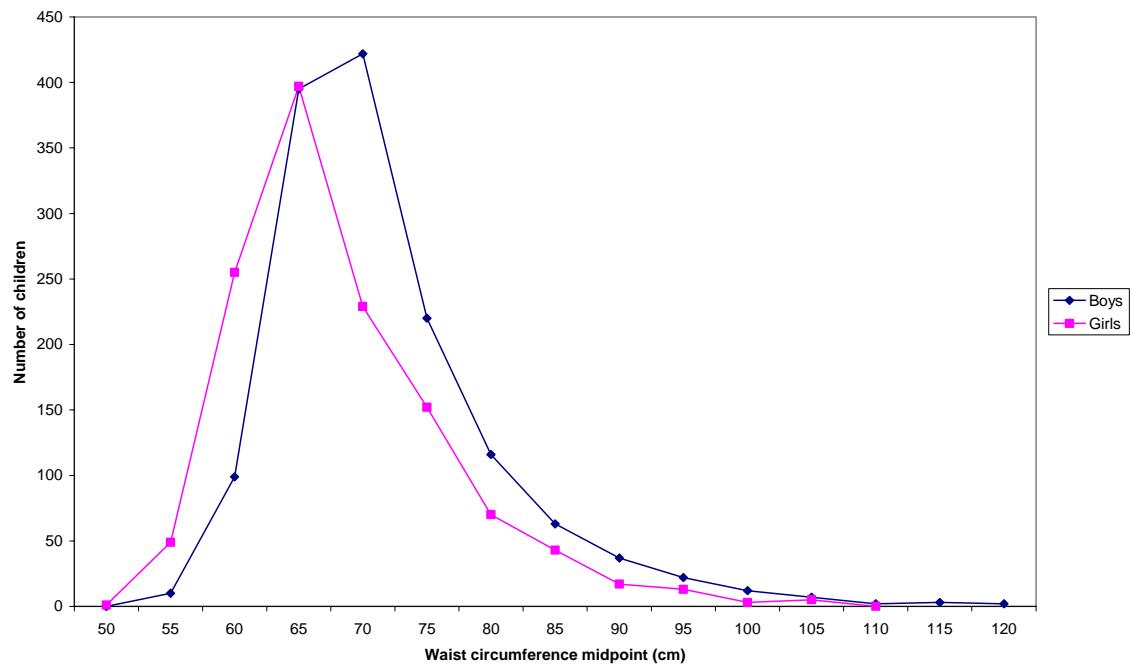
**Figure 4.4a Log (sum of skinfolds) distribution**



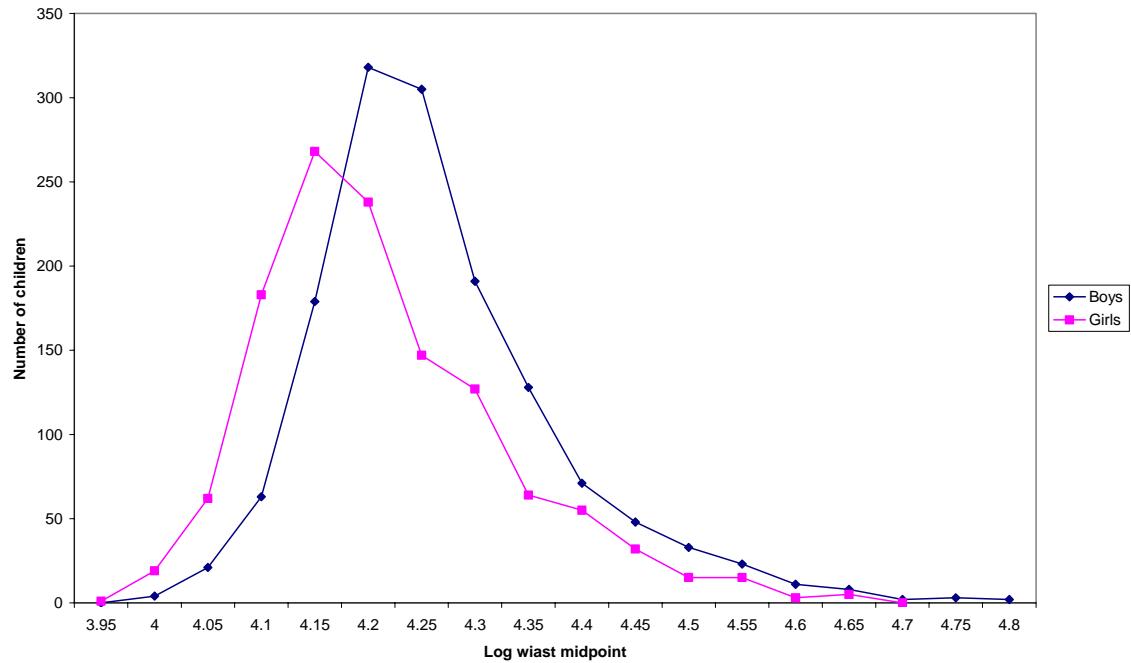
**Figure 4. 5: Body fat % (from bioimpedance) distribution**



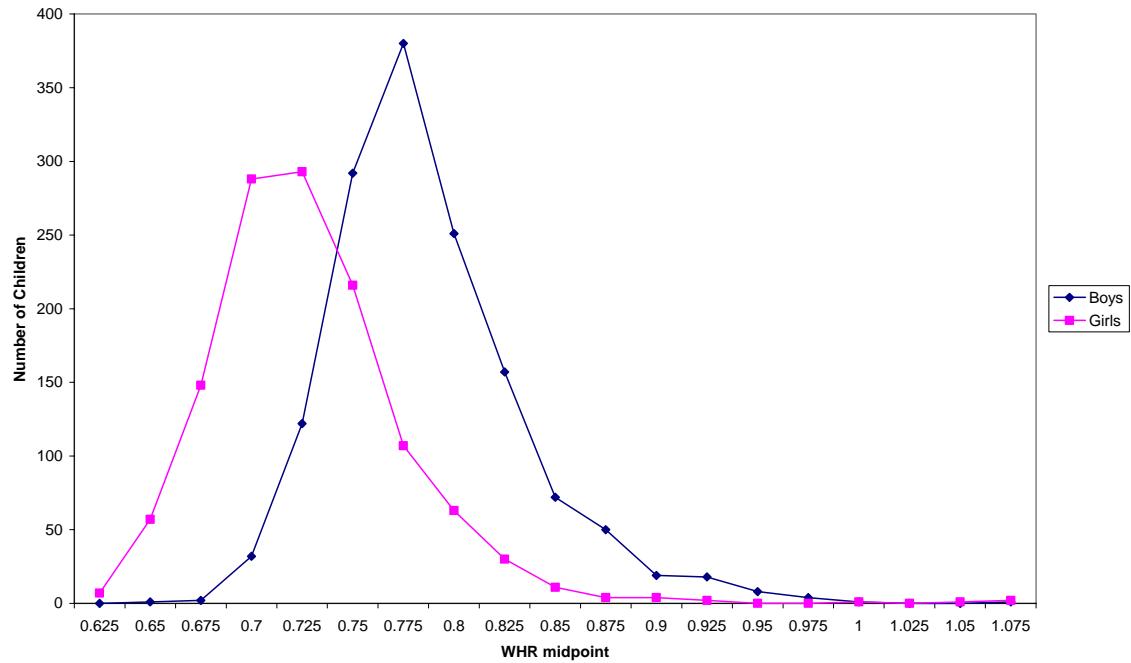
**Figure 4. 6: Waist circumference distribution**



**Figure 4.6a Log (waist circumference) distribution**

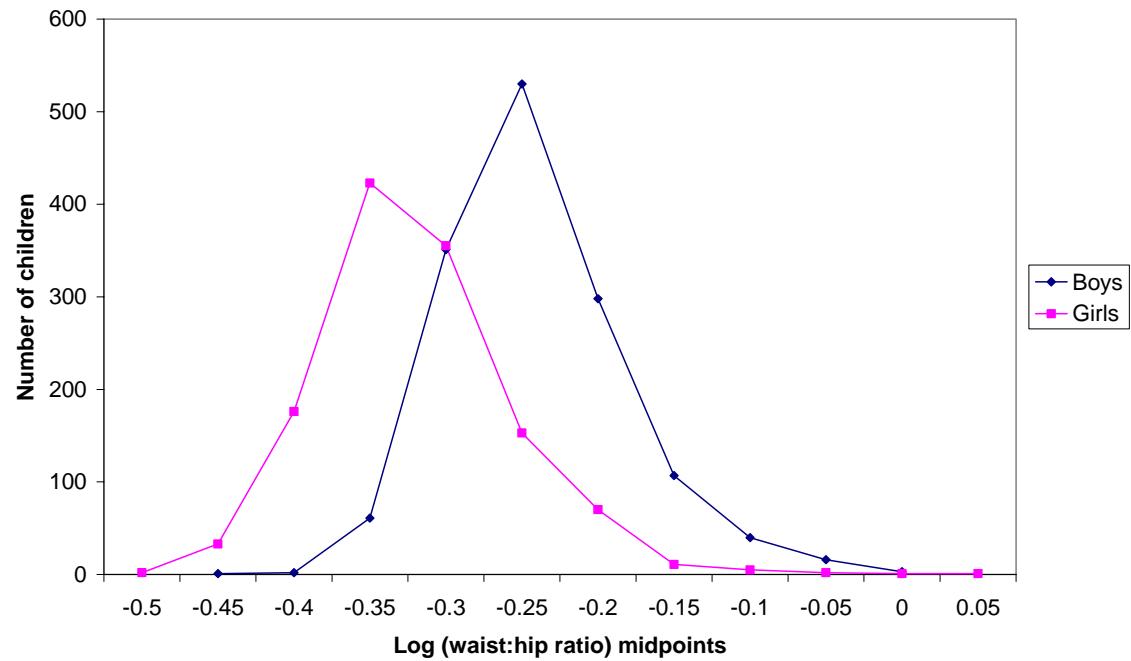


**Figure 4. 7: Waist : Hip Ratio distribution**



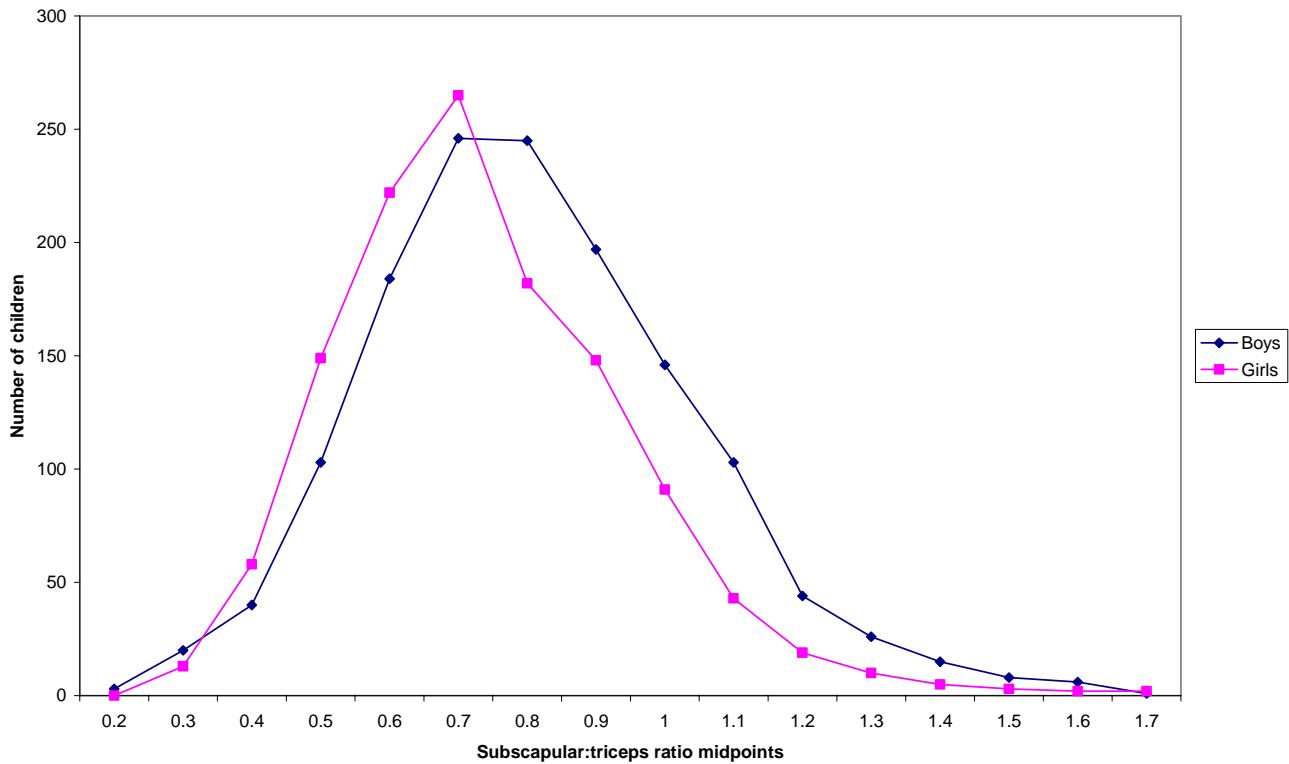
Nb. Outlier values at 1.2, 1.3 (girls) and 1.325 (boy) omitted from plot.

**Figure 4.7a Log (waist hip ratio) distribution**

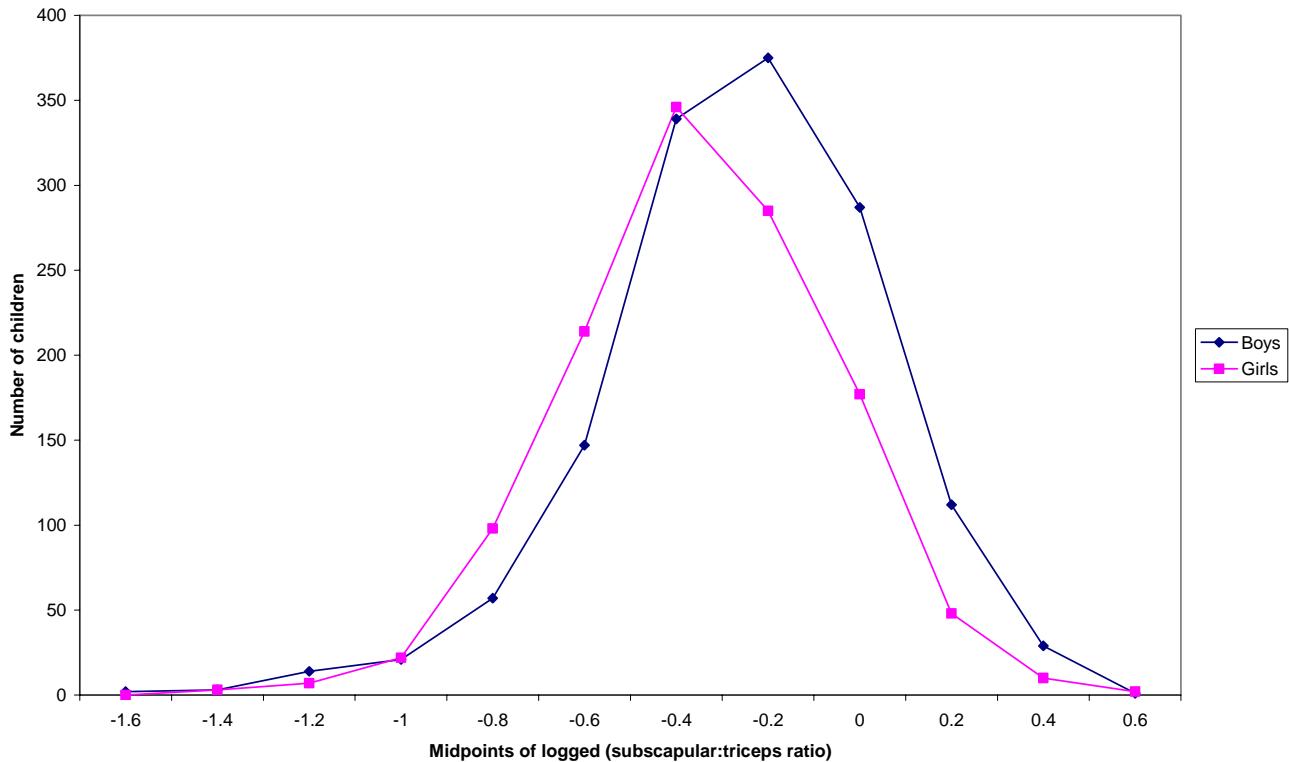


Nb. Outlier values at 0.2, 0.25 (girls) and 0.3 (boy) omitted from plot.

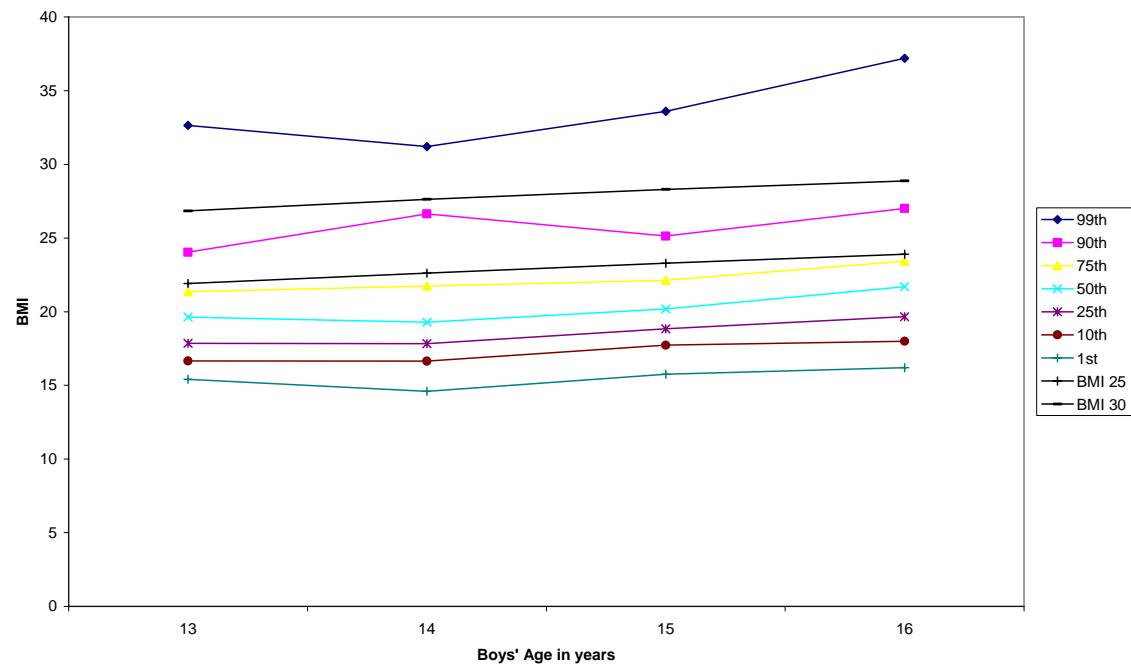
**Figure 4. 8: Subscapular:triceps ratio distribution**



**Figure 4.8a Log subscapular:triceps ratio distribution**



**Figure 4. 9: Centiles of Ten Towns data for boys showing comparison to proposed international cut offs for overweight and obesity**



**Figure 4. 10: Centiles of Ten Towns data for girls showing comparison to proposed international cut offs for overweight and obesity**

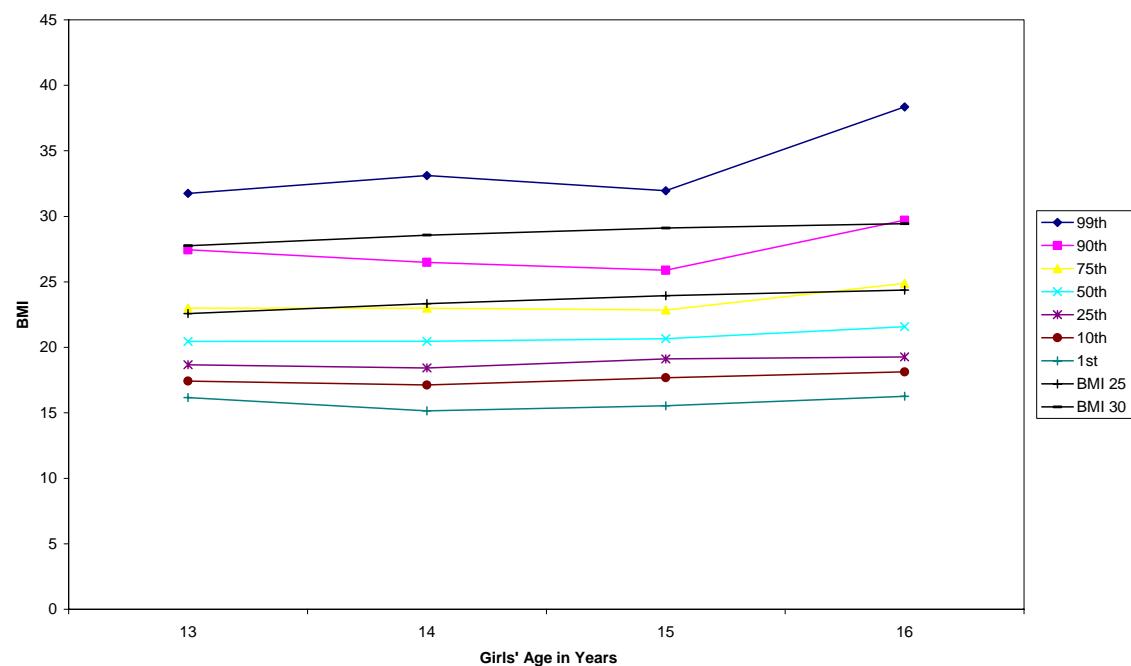


Figure 4.11(a) Scatterplots of  $\log(\text{BMI})$  with other adiposity

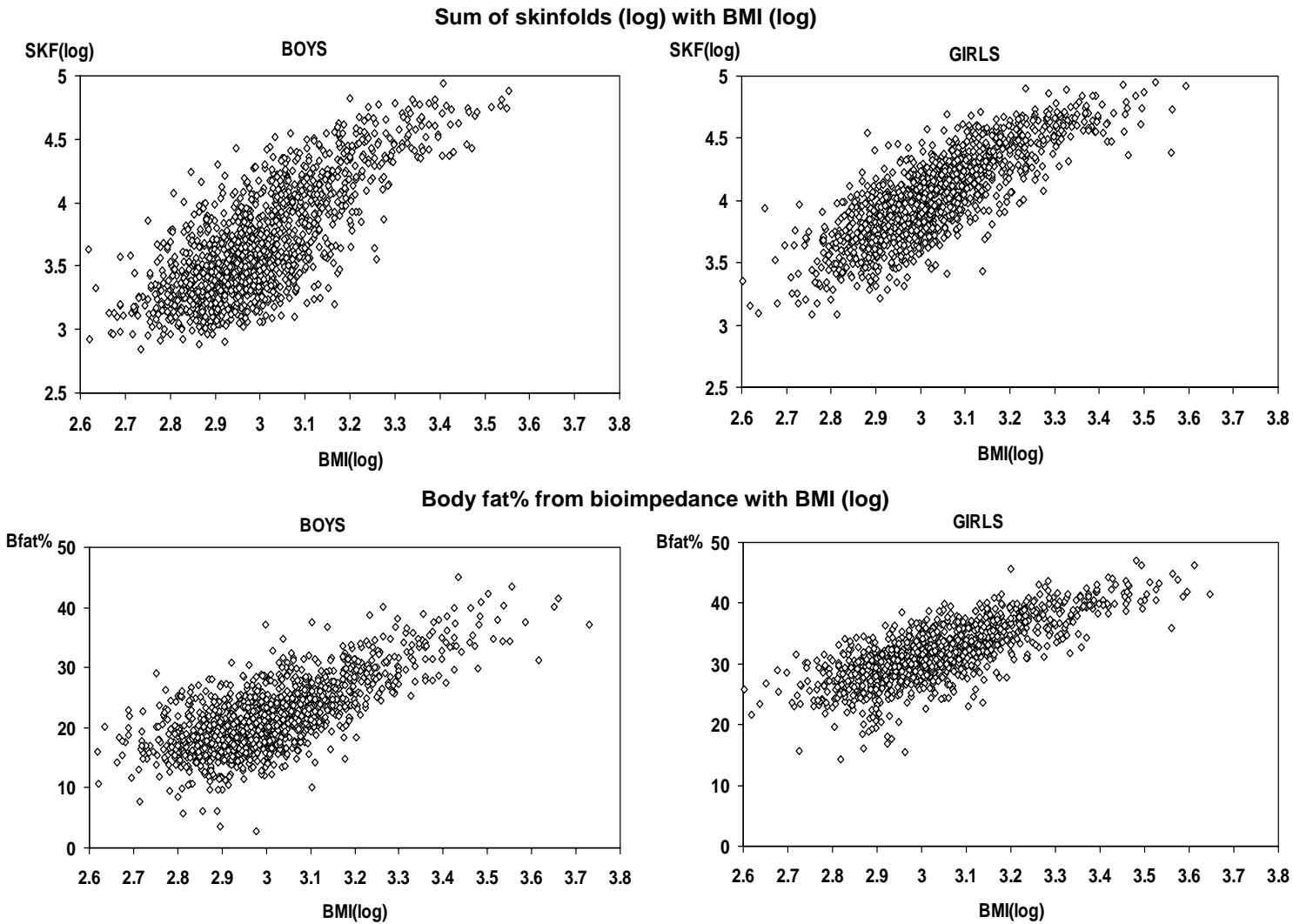
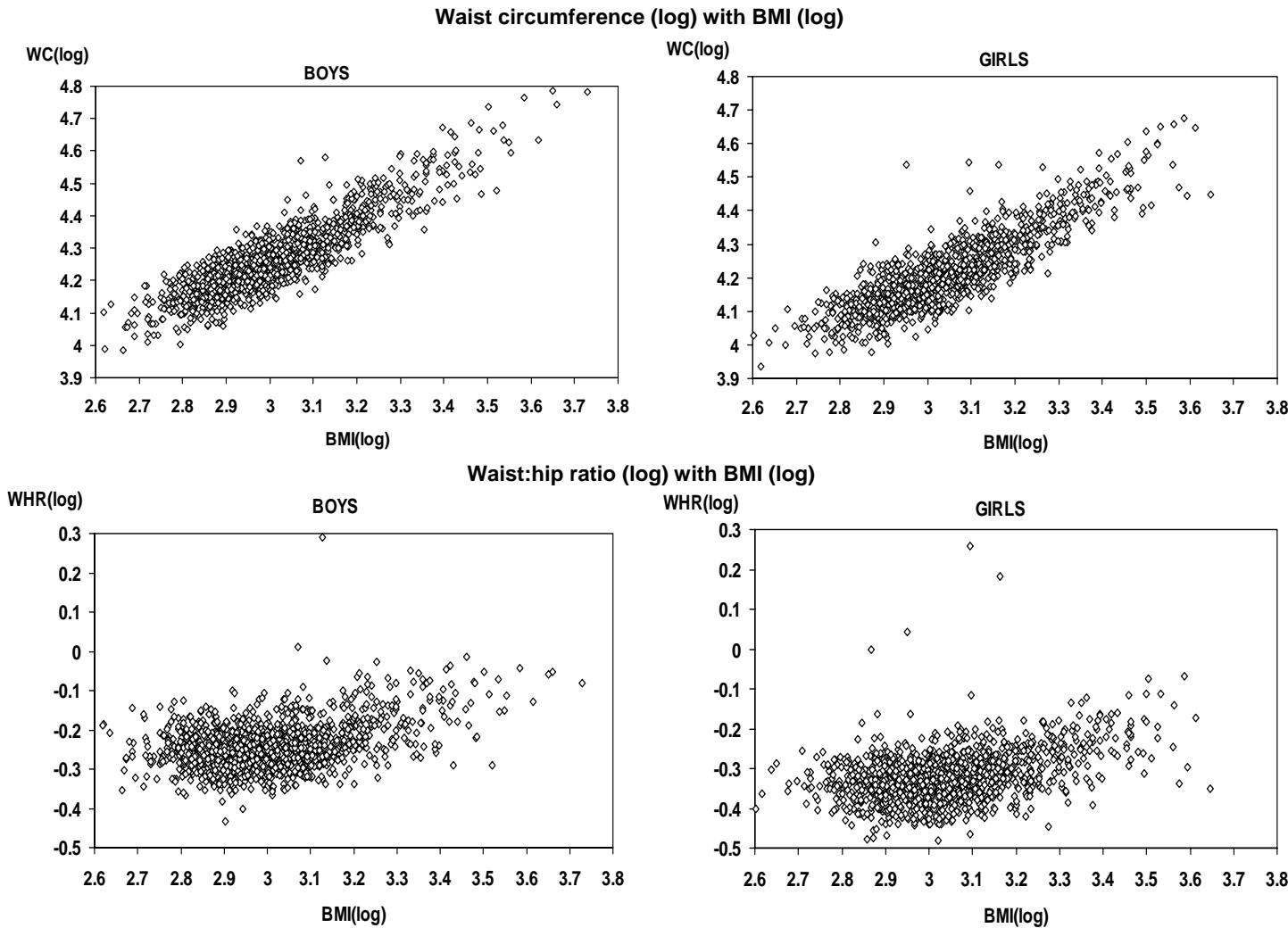


Figure 4.11(b) Scatterplots of log(BMI) with other adiposity variables



**Table 4. 1: Numbers of children with body size and adiposity measures**  
 Adiposity measures are **not** log transformed in this table

<b>Variable</b>	<b>Number of Subjects</b>	<b>Mean</b>	<b>Median</b>	<b>Interquartile Range</b>	<b>Standard Deviation</b>
<b>Age</b>	2645	14.82	14.81	0.94	0.65
<b>Height</b>	2645	165.66	165.20	11.70	8.49
<b>Weight</b>	2640	57.5	55.8	14.15	11.95
<b>Body mass index (BMI)</b>	2640	20.87	20.15	4.06	3.62
<b>Triceps skinfold</b>	2616	15.68	14.00	10.60	7.68
<b>Biceps skinfold</b>	2617	7.72	6.20	5.00	4.74
<b>Subscapular skinfold</b>	2599	11.55	9.40	6.60	6.23
<b>Suprailiac skinfold</b>	2567	17.60	15.80	12.40	8.36
<b>Body fat % from Bioimpedance</b>	2570	26.50	26.87	11.87	7.46
<b>Waist circumference</b>	2644	69.93	68.20	9.55	8.71
<b>Hip circumference</b>	2644	92.07	91.30	9.90	8.19
<b>Waist:hip ratio (WHR)</b>	2644	0.76	0.76	0.07	0.06

**Table 4. 2: Relation of observer to body size and adiposity variables**

Adiposity variable	Mean adiposity for each observer no. (SE)						P value	After adj for age	After adj for age and gender
	1	2	3	4**	5	6			
<b>No. of children measured (skinfolds)</b>	425	652	930		262	290			
<b>No. of children measured (bioimpedance)</b>	237	316	574	1114	174	152			
<b>Height(cm)+</b>	166.2 (0.4)	166.2 (0.3)	165.7 (0.3)		164.4 (0.5)	164.8 (0.5)	0.01	0.98	
<b>Weight(kg)*</b>	56.27	57.28	56.52		54.21	55.85	0.003	0.22	
<b>BMI (kg/m<sup>2</sup>)*</b>	20.43	20.80	20.65		20.12	20.63	0.045	0.12	
<b>Waist* circumference</b>	69.76	67.90	69.40		70.32	71.66	<0.0001	<0.0001	<0.0001
<b>Hip* circumference</b>	91.93	90.92	91.93		90.59	93.50	<0.0001	<0.0001	<0.0001
<b>Waist: hip ratio*</b>	0.759	0.747	0.755		0.776	0.766	<0.0001	<0.0001	<0.0001
<b>Sum of skinfolds (mm)*</b>	48.58	41.55	49.88		43.73	50.41	<0.0001	<0.0001	<0.0001
<b>Subscapular: triceps ratio +</b>	0.65 (0.01)	0.84 (0.01)	0.76 (0.01)		0.80 (0.01)	0.83 (0.01)	<0.0001	<0.0001	<0.0001

+ Mean values are presented with standard errors.

\*Variables log transformed for analysis. Geometric means are presented.

\*\* Study doctor.

**Table 4. 3: Gender differences in body size and adiposity measurements, adjusted for age and observer. LS means are presented.**

	MALE Mean (std. err)	FEMALE Mean (std. err)	p (no sex difference)
<b>Number of subjects with all measurements</b>	1370	1197	
<b>Age (years)</b>	14.85 (0.01)	14.86 (0.01)	0.78
<b>Height (cm)</b>	168.85 (0.20)	161.94 (0.22)	<0.0001
<b>Weight *</b>	57.41	54.89	<0.0001
<b>Body mass index (kg/m<sup>2</sup>) *</b>	20.20	20.96	<0.0001
<b>Sum of skinfolds *</b>	39.50	56.90	<0.0001
<b>Body fat percent from bioimpedance</b>	21.76 (0.15)	31.99 (0.16)	<0.0001
<b>Waist circumference *</b>	71.51	68.01	<0.0001
<b>Hip circumference *</b>	90.73	93.30	<0.0001
<b>Waist : hip ratio (WHR) *</b>	0.788	0.729	<0.0001
<b>Subscapular : triceps ratio</b>	0.812 (0.006)	0.736 (0.006)	<0.0001

\* analysed as log (variable)

**Table 4. 4: Relation of age (years) to body size and adiposity variables**  
**Shown as Boys Girls**

Variable	Mean for each age group (years)					Increase in measure per year (95% CI)	p value
	12	13	14	15	16		
<b>Height(cm)+</b>	154.8 (2.7)	161.3 (0.7)	167.0 (0.3)	172.7 (0.4)	175.7 (1.2)	6.4(5.7 to 7.0)	<0.0001
	154.3 (1.8)	159.6 (0.6)	161.7 (0.3)	162.7 (0.3)	165.6(1.0)	2.2(1.7 to 2.7)	<0.0001
<b>Weight(kg)*</b>	44.9	51.4	55.1	60.9	66.6	11.8%(10.1 to 14.1)	<0.0001
	44.7	53.5	54.4	55.8	60.3	4.1%(2.5 to 5.7)	<0.0001
<b>BMI (kg/m<sup>2</sup>)*</b>	18.8	19.8	19.8	20.7	21.6	3.6%(2.3 to 4.9)	<0.0001
	18.8	21.0	20.9	21.1	22.2	1.4%(0.2 to 2.8)	0.05
<b>Sum of skinfolds (mm)*^</b>	42.9	42.3	39.1	38.8	38.9	-4.1%(-0.6 to-8.1)	0.02
	50.9	59.1	56.5	58.4	57.2	1.7%(-1.4 to 4.9)	0.3
<b>Body fat % from bioimpedance +</b>	24.7 (2.0)	22.3(0.5)	21.3(0.2)	22.1(0.3)	22.8 (0.8)	0.08 (-0.04 to 0.6)	0.7
	30.7 (1.5)	31.2(0.5)	31.5(0.2)	32.7(0.2)	33.6 (0.8)	0.95 (0.54 to 1.36)	<0.0001
<b>Waist*^ circumference</b>	67.8	69.7	70.5	73.0	75.2	2.8%(1.9 to 3.7)	<0.0001
	63.4	67.8	67.8	68.0	72.3	0.9%(-0.1 to 1.9)	0.07
<b>Waist: hip ratio*^</b>	0.82	0.80	0.79	0.78	0.78	-0.9%(-0.4 to-1.4)	0.0003
	0.75	0.74	0.73	0.73	0.74	-0.9%(-0.3 to -1.4)	0.004
<b>Subscapular:</b>	0.60(0.08)	0.74 (0.02)	0.79 (0.01)	0.85 (0.01)	0.95 (0.03)	0.07(0.05 to 0.09)	<0.0001
<b>triceps ratio</b>	0.73(0.06)	0.76 (0.02)	0.73 (0.01)	0.73(0.01)	0.78 (0.03)	-0.01(0.01 to 0.03)	0.23

+ Mean values are presented with standard errors. Increases in adiposity measures per year are presented in absolute values.

\* Variables log transformed for analysis. Geometric means are presented. Increases in adiposity measures per year are presented as percentage values.

Adjusted for gender throughout,    ^ Adjusted for observer in addition

**Table 4. 5: Highest Correlations of pubertal staging questions (p values)**

	<b>BOYS</b>		<b>GIRLS</b>	
	Puberty question 1 (penile/testicular development)	Puberty question 2 (pubic hair distribution)	Puberty question 1 (breast development)	Puberty question 2 (pubic hair distribution)
AGE	0.27 (<0.0001)	0.22 (<0.0001)	0.20 (<0.0001)	0.22 (<0.0001)
HEIGHT	0.38 (<0.0001)	0.33 (<0.0001)	0.08 (0.004)	0.16 (<0.0001)
LOG BMI	0.06 (0.001)	0.09 (0.001)	0.25 (<0.0001)	0.07 (0.01)

**Table 4. 6: Relations of pubertal stage score to body size and adiposity in boys**

	Pubertal stage score (approximate equivalent Tanner stage)				Increase in measure per pubertal stage (95%CI)	p value	Remaining increase after adj for age (95% CI)
BOYS	3-4 (2)	5-6 (3)	7-8 (4)	9-10 (5)			
Height	154.5 (1.1)	163.1 (0.5)	168.6 (0.6)	173.4 (0.3)	5.52 (5.01- 6.03)	<0.0001	4.26 (3.75- 4.77)
Weight*	47.5	51.3	57.2	62.6	10.0% (8.6%- 11.4%)	<0.0001	7.4% (6.0%- 8.9%)
BMI*	19.9	19.4	20.2	20.9	2.9% (1.8%- 3.9%)	0.0004	2.0% (0.9%- 3.2%)
Sum of skinfolds*	49.4	41.7	38.7	39.0	-4.4% (- 1.5%-- 7.1%)	0.003	-3.9% (- 0.8%-- 6.8%)
Body fat % from bioimpedance+	26.1 (0.7)	22.9 (0.4)	21.2 (0.3)	21.6 (0.3)	-0.8 (- 0.4--1.2)	<0.0001	-1.0 (-0.6-- 1.4)
Waist circumference*	69.8	69.1	70.8	72.8	2.2% (1.4%- 2.9%)	<0.0001	1.5% (0.7%- 2.3%)
Waist: Hip ratio*	0.814	0.793	0.785	0.781	-1% (- 0.6%-- 1.4%)	<0.0001	-0.9% (- 0.4%-- 1.3%)
Subscapular:tri ceps ratio+	2.04	2.11	2.23	2.35	5.1% (3.5%- 6.8%)	<0.0001	3.2% (1.5%- 4.9%)

+ Mean values are presented with standard errors. Increases in adiposity measures per stage are presented in absolute values.

\* Variables log transformed for analysis. Geometric means are presented. Increases in adiposity measures per stage are presented as percentage values.

**Table 4. 7: Relations of pubertal stage score to body size and adiposity in girls**

	<b>Pubertal stage score (approximate equivalent Tanner stage)</b>				<b>Increase in measure per pubertal stage (95%CI)</b>	<b>p value</b>	<b>Remaining increase after adj for age (95% CI)</b>
<b>GIRLS</b>	<b>3-4 (2)</b>	<b>5-6 (3)</b>	<b>7-8 (4)</b>	<b>9-10 (5)</b>			
Height	155.7 (1.5)	160.8 (0.4)	162.5 (0.3)	162.4 (0.3)	1.0 (0.51- 1.48)	<0.0001	0.54 (0.29- 1.03)
Weight*	47.7	51.8	55.1	58.0	5.9% (4.5%- 7.4%)	<0.0001	5.2% (3.7%- 6.7%)
BMI*	19.7	20.1	20.9	22.0	4.6% (3.3%- 6.0%)	<0.0001	4.5% (3.1%- 5.7%)
Sum of skinfolds*	52.3	52.6	56.2	62.2	8.3% (5.3%- 11.4%)	<0.0001	8.2% (5.1%- 11.4%)
Body fat % from bioimpedance+	32.4	30.6	32.0	33.0	0.99 (0.60- 1.38)	<0.0001	0.81 (0.41- 1.21)
Waist circumference*	65.6	66.3	67.2	69.3	2.1% (1.2%- 3.0%)	<0.0001	1.9% (1.0%- 2.9%)
Waist: Hip ratio*	0.743	0.733	0.721	0.728	-0.3% (0.1%-- 0.9%)	0.14	-0.2% (- 0.8%-0.3%)
Subscapular:tri ceps ratio+	2.25	2.05	2.07	2.11	0.2% (- 1.4%- 1.9%)	0.77	0.6% (- 1.1%-2.3%)

+ Mean values are presented with standard errors. Increases in adiposity measures per stage are presented in absolute values.

\* Variables log transformed for analysis. Geometric means are presented. Increases in adiposity measures per stage are presented

**Table 4. 8: Correlation Table of body size and adiposity measurements (boys)**

\*=p&lt;0.0001. {after adjustment for age}

	AGE	HEIGHT	LOG WEIGHT	Log BMI	LOG SUM SKF	Impedance (body fat)	LOG WAIST	LOG WHR
HEIGHT	0.46*							
LOG WEIGHT	0.35*	0.68* {0.65*}						
Log BMI	0.15*	0.23* {0.20*}	0.87* {0.87*}					
LOG SUM SKF	-0.07 (p=0.01)	-0.01 (p=0.7) {0.02, p=0.39}	0.55* {0.62*}	0.76* {0.78*}				
IMPED-ANCE (BODY FAT)	0.009 (p=0.7)	-0.06 (p=0.01) {-0.08, p=0.003}	0.51* {0.50*}	0.73* {0.70*}	0.78* {0.79*}			
LOG WAIST	0.16*	0.32* {0.31}	0.83* {0.83}	0.89* {0.87}	0.74* {0.77*}	0.69* {0.66*}		
LOG WHR	-0.10 (p=0.0002)	-0.20* {-0.19*}	0.21* {0.20*}	0.42* {0.38*}	0.45* {0.44*}	0.45* {0.41*}	0.64* {0.63*}	
Subscap:triceps ratio	0.20*	0.26* {0.19*}	0.24* {0.17*}	0.14* {0.10, p=0.0004}	-0.13 * {-0.12*}	-0.03(p=0.3) {-0.03, p=0.26}	0.15* {0.11*}	-0.01 (p=0.6) {-0.01, p=0.65}

**Table 4. 9: Correlation Table of body size and adiposity measurements (girls)**

\*=p&lt;0.0001 {after adjustment for age}

	AGE	HEIGHT	Log WEIGHT	Log BMI	Log SUM SKF	IMPED-ANCE (BODY FAT)	Log WAIST	LOG WHR
HEIGHT	0.23*							
LOG WEIGHT	0.15*	0.45* {0.46*}						
Log BMI	0.05 (p=0.05)	0.03 (p=0.2) {0.03, p=0.3}	0.91* {0.90*}					
LOG SUM SKF	0.02 (p=0.4) {0.05, p=0.12}	0.06 (p=0.03)	0.74* {0.74*}	0.81* {0.81*}				
IMPED-ANCE (BODY FAT)	0.13*	-0.06 (p=0.03) {-0.09, p=0.02}	0.67* {0.63*}	0.77* {0.75*}	0.73* {0.73*}			
LOG WAIST	0.05 (p=0.09)	0.20* {0.20*}	0.86* {0.85*}	0.87* {0.86}	0.79* {0.79*}	0.69* {0.66*}		
LOG WHR	-0.09 (p=0.001)	-0.10 (p=0.0004) {-0.11, p=0.0002}	0.32* {0.28*}	0.41* {0.37*}	0.40* {0.40*}	0.34* {0.31*}	0.69* {0.67*}	
Subscap:triceps ratio	-0.03 (p=0.2)	-0.0004 (p=1) {-0.007, p=0.8}	0.22* {0.20*}	0.25* {0.22*}	0.73* {0.73*}	0.23* {0.23*}	0.28* {0.25*}	0.22* {0.19*}

**Table 4. 10: Correlations of fifths of log sum of skinfolds with adiposity variables**

	<b>FIFTHS OF LOG SUM OF SKINFOLDS</b>				
<b>BOYS</b>	<b>1 (Lowest)</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5 (Highest)</b>
WEIGHT*	0.23	-0.006	0.05	0.16	0.47
BMI*	0.24	0.03	0.08	0.32	0.67
BODY FAT %	0.15	0.18	0.14	0.34	0.65
WAIST*	0.24	0.02	0.05	0.30	0.69
<b>GIRLS</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>
WEIGHT*	0.28	0.20	0.13	0.20	0.56
BMI*	0.42	0.26	0.19	0.31	0.58
BODY FAT %	0.46	0.26	0.14	0.17	0.42
WAIST*	0.29	0.21	0.13	0.26	0.58

\* analysed as log variables

**Table 4. 11: Correlations of fifths of log BMI with adiposity variables**

	<b>FIFTHS OF BMI</b>				
<b>BOYS</b>	<b>1 (Lowest)</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5 (Highest)</b>
WEIGHT*	0.60	0.27	0.25	0.35	0.75
SUM OF SKINFOLDS*	0.27	0.18	0.08	0.19	0.63
BODY FAT %	0.04	0.04	0.06	0.16	0.70
WAIST*	0.50	0.27	0.16	0.23	0.83
<b>GIRLS</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>
WEIGHT*	0.62	0.25	0.33	0.39	0.79
SUM OF SKINFOLDS*	0.41	0.15	0.23	0.29	0.55
BODY FAT %	0.16	0.23	0.10	0.27	0.58
WAIST*	0.43	0.08	0.19	0.34	0.79

\* analysed as log variables

**Table 4. 12 Comparison with International centiles for BMI- BOYS**  
**Computed Centiles for BMI for ten towns boys presented for comparison with proposed International centiles (Cole 2000). All figures are BMI (kg/m<sup>2</sup>).**

	<b>BOYS Age in Years</b>			
<b>Ten Towns Data: BMI centile</b>	<b>13</b> (123 boys)	<b>14</b> (723 boys)	<b>15</b> (506 boys)	<b>16</b> (46 boys)
<b>99th</b>	32.64	31.21	33.60	37.20
<b>90th</b>	24.04	26.64	25.13	27.00
<b>75th</b>	21.36	21.74	22.13	23.43
<b>50th</b>	19.64	19.28	20.18	21.70
<b>25th</b>	17.85	17.83	18.84	19.66
<b>10th</b>	16.66	16.65	17.73	18.00
<b>1st</b>	15.41	14.60	15.76	16.20
<b>International centiles</b>				
<b>BMI 25*</b>	21.91	22.62	23.29	23.90
<b>BMI 30**</b>	26.84	27.63	28.30	28.88

\*BMI 25- centile which passes through BMI of 25 at age 18, ie proposed 'overweight' if above this centile

\*\*BMI 30- centile which passes through BMI of 30 at age 18, ie proposed 'obese' if above this centile

**Table 4. 13: Comparison with International centiles- GIRLS**  
**Computed Centiles for BMI for ten towns girls presented for comparison with proposed International centiles (Cole 2000). All figures are BMI (kg/m<sup>2</sup>).**

	<b>Age in Years</b>			
<b>Ten Towns Data: centile</b>	<b>13</b> (106 girls)	<b>14</b> (615 girls)	<b>15</b> (464 girls)	<b>16</b> (37 girls)
<b>99th</b>	31.75	33.11	31.95	38.36
<b>90th</b>	27.45	26.49	25.88	29.71
<b>75th</b>	23.00	22.99	22.85	24.88
<b>50th</b>	20.44	20.46	20.66	21.58
<b>25th</b>	18.67	18.42	19.11	19.27
<b>10th</b>	17.43	17.12	17.68	18.13
<b>1st</b>	16.16	15.15	15.54	16.27
<b>International centiles</b>				
<b>BMI 25*</b>	22.58	23.34	23.94	24.37
<b>BMI 30**</b>	27.76	28.57	29.11	29.43

\*BMI 25- centile which passes through BMI of 25 at age 18, ie proposed 'overweight' if above this centile

\*\*BMI 30- centile which passes through BMI of 30 at age 18, ie proposed 'obese' if above this centile.

## **CHAPTER 5: RELATIONS OF GEOGRAPHIC LOCATION (TOWN), SOCIAL CLASS AND ETHNIC GROUP TO BODY SIZE AND ADIPOSITY MEASURES IN 13-16 YEAR-OLDS**

### **5.0 SUMMARY POINTS**

- Mean height showed marked variation between towns and was systematically lower among high cardiovascular mortality towns in Northern England and Wales, by 1.8cm for boys and 2.2cm for girls. Mean height was also lower among children from lower social class groups (by 0.3 cm per social class group) and among children of South Asian origin.
- Mean weight and markers of general (BMI, sum of skinfolds and body fat percent from bioimpedance) and central (waist: hip ratio, waist circumference and subscapular: triceps ratio) adiposity also showed marked variation between towns but high cardiovascular town: low cardiovascular mortality town differences were not significant. Children in Rhondda were particularly adipose.
- Weight varied with social class, with boys in lower social classes being significantly lighter than other boys. There was not a significant trend in girls. Measures of general (BMI, sum of skinfolds, body fat percent from bioimpedance) and central adiposity (waist: hip ratio, waist circumference and subscapular: triceps ratio) tended to increase with lower social class in girls, but not in boys.
- South Asian children were lighter with lower BMI than white children. Other markers of general adiposity (sum of skinfolds, body fat percent from bioimpedance) were higher in South Asian children but this difference was only significant for body fat percent. Of the markers of central adiposity waist: hip ratio, and waist circumference did not show significant differences by ethnic group. Subscapular: triceps ratio was higher in South Asians than whites.

### **5.1 INTRODUCTION**

This chapter describes the influences of town, social class and ethnicity on adiposity in 13-16 year old British school children in the Ten Towns Study. Chapter 2 details some background to these factors, including the relationship they are known to have to adiposity in adults. This chapter explores these relationships in 13- 16 year old children. The individual determinants of adiposity studied in Chapter 4 (age, gender, pubertal stage and

observer) could influence these relations and will therefore be controlled for as appropriate.

Geographic, social and ethnic differences in adiposity have been observed in British adults. Levels of obesity (from BMI) in adults show variation in different areas of England in both sexes with higher obesity in the North and West (Health Surveys for England 1994, 1998, 2003, 2006). Sequential studies have shown strong and consistent results of increasing obesity (from BMI) with decreasing social class in women. The social class differences in men are similar but the effect is not as strong (Health Surveys for England 1994, 1998, 2003). Socio-economic disadvantage is also linked to higher waist: hip ratio in adults (Health Survey for England 2003). South Asian adults in Britain have larger waist: hip ratios and trunk skinfold measurements than whites (Mc Keigue et al 1991, Knight et al 1992). They are also shorter (Bhopal et al, 1999). Evidence for similar differences in 13-16 year olds will be explored in this chapter.

## **5.2 AIMS AND OBJECTIVES**

The aim of this study is to explore factors associated with adiposity in a group of schoolchildren aged 13-16 years. The objectives of this section include:

- To examine the relationship between geographical location (town) and the different measures of body size and adiposity (Section 5.4.1).
- To examine the relationship between social class and the different measures of body size and adiposity (Section 5.4.2).
- To examine the relationship between ethnic group and the different measures of body size and adiposity (Section 5.4.3).

The relations of social class and ethnicity to body size and adiposity were examined in boys and girls separately, as well as in combination, to take account of the possibility that the pattern of relationships differed between genders, as has been observed (particularly for social class relations) in adults.

## **5.3 SUBJECTS AND METHODS**

This study is based on the findings from a school-based survey carried out in ten towns in England and Wales between September 1998 and June 1999. Detailed information on subjects and methods and all measuring techniques are included in Chapter 3. Towns were selected on the basis of adult cardiovascular mortality (England and Wales, 1979-83, for

men and women aged 35-64 years) to include the five centres with the lowest adult cardiovascular mortality (Esher, Leatherhead, Bath, Chelmsford, Tunbridge Wells) and the five with the highest (Wigan, Rochdale, Burnley, Port Talbot, Rhondda). Towns were visited and analysed in high-mortality/ low-mortality pairs, see statistical methods section below.

Social class was coded from parental occupation in accordance with the registrar general's (ONS) 1990 coding manual. Social class of the head of household was used; usually the father figure; if absent then mother's social class was used. Information on occupation was obtained from parental questionnaire if available; child questionnaire if parental not available.

Ethnic group was assessed by the nurse observers on the basis of the child's appearance into five main groupings (white, Afro-Caribbean, Asian, Oriental, other). In the current report ethnic groups were analyzed as white and Asian and combined other group; the numbers in combined group were small; results for white and Asian groups only are presented here.

### **5.3.1 STATISTICAL METHODS**

All data were analysed using the SAS system statistical software package (SAS Institute, North Carolina, USA). The LSMEANS procedure was used to calculate adjusted means; dummy variables were used for observer, ethnic group and pubertal stage adjustment to allow for varying numbers of children in these variables' subgroups. Dummy variables were also used for town when producing least squares means for ethnic groups, as ethnic mix in towns varied considerably.

Analyses were carried out by town, by social class and by ethnic group, for all children and separately for boys and girls. Mean values for all children were adjusted throughout for age, sex and (some physical measurements only) observer, although these adjustments had no important effect on the differences observed. Age was fitted as a continuous variable, sex and town as categorical variables. Observer, social class, puberty score and ethnic group (where used) were fitted as dummy variables to minimise the effect of unequal distribution of subjects in these groups.

Town analyses looked both at overall differences between towns and then specifically at comparisons between high cardiovascular mortality towns (Northern England and South Wales) and low mortality towns. Some analyses were then done for boys and girls separately. Comparisons of high and low mortality towns were carried out, using adjusted

means produced using PROC GLM. Using the pairing of study towns defined in the study design, paired *t* tests were carried out to test the difference between high and low towns, on 4 degrees of freedom where necessary. This analysis took variations within as well as between towns in each factor into account. Paired analyses were used to minimise the effects of measurement drift, seasonal factors, and the change in average age. All differences have been represented as high mortality town minus low mortality town. Social class analyses were adjusted for age, sex, puberty score, and observer (as appropriate). Tests for trend were fitted across ordered social class groups as appropriate. Additional adjustments for town, then town and ethnicity together, were added to examine the effect of these factors on the social class differences seen. Similar analyses were completed for boys and girls separately. Ethnic group analyses were adjusted for age, sex, puberty score, observer (as appropriate). The effects of social class was then examined (all children together) to explore the effect of this on differences seen. When producing LS means for ethnic group dummy variables were used for town as ethnic mix varied between towns. Tests for sex interaction were also carried out for analyses of social class and ethnic group differences, in order to determine whether the strength of relationships differed between sexes.

## **5.4 RESULTS**

### **5.4.1 NUMBERS OF PARTICIPANTS IN DIFFERENT TOWNS, SOCIAL CLASS GROUPS AND ETHNIC GROUPS**

Following invitation, in total 2645 subjects took part in the study (66% response rate), of these 1410 were boys and 1235 were girls. Participants were reasonably evenly distributed between study towns (Table 5.1, final column).

Social class data were available for 2527 children. For analysis Social Classes IV and V will be grouped together, as numbers in V were relatively small (104 children). Subjects whose parent was in the Armed Forces or unknown will be omitted (Armed Forces: 83 boys, 53 girls; 5 children unknown social class).

Of the 2645 children with some or all physical measurements 92% of children were defined as white European, 6.5% were South Asian and 1.5% were combined others. The latter group was too small for analysis.

#### **5.4.1.1 Interrelations of town, social class and ethnic group**

64% of low cardiovascular mortality towns' children were assigned to non-manual social class groups and 36% to manual groups. Almost the reverse was true for children in high cardiovascular mortality towns which were 39% non-manual social class groups and 61% manual groups (Table 5.1). From all children in high mortality towns 25% were in Social Class IV/V and only 4.2% in Class I. For low mortality towns the equivalent figures are 11% and 12%.

South Asian children were predominantly resident in two of the ten towns (Table 5.2). The Rochdale study sample had the highest percentage of Asian children (42.8%), next was Burnley, with 7.9% Asian children. Other towns only had very small numbers of South Asian children.

South Asian children were also overrepresented in lower social classes (table 5.3), with roughly three quarters of the Asian children being in the manual (IIIM,IV,V) classes but only half the white children. Therefore an additional adjustment for social class was carried out to see if this altered any differences seen between ethnic groups.

#### **5.4.1.2 Pubertal status differences in town, social class and ethnic group**

Pubertal status score did not show an overall difference between towns ( $p= 0.78$  girls, 0.63 boys) or between high and low mortality towns ( $p= 0.25$  girls, 0.27 boys). Including adjustment for pubertal status did not affect the between town differences observed.

Pubertal status score did not show a social class gradient ( $p=0.74$  girls, 0.53 boys).

Pubertal status score was lower in South Asian children (Boys' mean score: Asian 3.90, white 4.17,  $p= 0.003$ , Girls' mean score: Asian 3.70, white 4.09,  $p= <0.0001$ , all adjusted for age). Therefore pubertal adjustment was made when analyzing differences in adiposity.

### **5.4.2 TOWN DIFFERENCES IN BODY SIZE AND ADIPOSITY**

Table 5.4 summarises the average measurements on children in each study town, the overall  $p$  values for differences between towns and the paired differences between high and low mortality towns. Figure 5.1 demonstrates the town differences graphically.

#### **5.4.2.1 Overall town differences**

There was evidence of overall differences between towns, with  $p<0.0001$  for all variables for a town difference. Children in Rhondda had the highest weight, body mass index, sum of skinfolds, body fat percentage from bioimpedance and waist: hip ratio. Children in Rochdale were the shortest, with lowest weight and BMI, reflecting the high percentage of Asian children in this town's sample (42.8%) Table 5.2. Adjusting for ethnicity altered the

results for Rochdale (Table 5.5) to be more similar to those of other high mortality towns but did not affect the overall town differences.

#### **5.4.2.2 Differences between high and low mortality towns**

The differences between high and low mortality town pairs were then examined further.

Children in high mortality towns were on average 1.9 cm (95 % CI 0.8 to 3.1cm) shorter than children in low mortality towns. Weight was 0.8% higher in low mortality towns.

General adiposity measures (BMI, sum of skinfolds and body fat %) were lower in low mortality towns but these differences were not significant. All the measures of central adiposity were also lower in low mortality towns but not significantly.

These findings were independent of age (Table 5.4 adjusted throughout for age). The low-high difference in height was not changed by adjusting for pubertal status (low-high difference 2cm, SE 0.4, p=0.01); as expected as pubertal status did not differ significantly between towns (see 5.4.1.2). The differences in Table 5.4 were unaffected by exclusion of children from non-European ethnic groups (data not presented). Separate analyses in boys and girls showed similar patterns, with girls in low mortality towns being 2.2cm taller and boys 1.8cm taller.

The low-high height differences between towns remained even after additional adjustment for social class and ethnic group; boys 1.9 cm (95 % CI 1.0 to 2.7cm), girls, 2.0cm, (95 % CI 1.3 to 2.7cm).

#### **5.4.3 SOCIAL CLASS DIFFERENCES IN ADIPOSITY**

Table 5.6 shows the social class differences in body build measures separately for boys and girls. Throughout this section ‘lower social class’ means social classes 4 and 5 (lower affluence); ‘higher social class’ means social classes 1 and 2 (higher affluence). Girls showed increasing central and general adiposity with lower social class, boys did not.

##### **5.4.3.1 Social class differences in height and weight by gender**

There was a social class gradient for height and weight in boys, which was independent of town and ethnicity. Lower social class boys and girls were shorter; boys were lighter when compared to higher class children of the same gender. Boy’s weight was 1% lower per step down in social class overall, though boys in social class I were lighter than all other boys except those in social class IV and V. In girls there was an increase of 0.4% in weight with social class; there was evidence of gender interaction with social class for weight, p=0.03.

#### **5.4.3.2 Social class differences in general measures of adiposity by gender**

In boys BMI decreased by 0.22% with decreasing social class (Table 5.6); this was not significant. In girls there was a significant increase of 0.77% ( $p=0.04$ ), which was confounded by town; adjustment for town and ethnicity reduced the association, rendering it non-significant. There was no strong evidence of gender interaction ( $p=0.08$ ) (Table 5.8). Sum of skinfolds showed no significant social class gradient in either boys or girls, the direction of change with decreasing social class being decrease for boys and increase in girls. There was evidence of confounding by town, with town adjustment strengthening the association seen in boys, but not in girls (data not shown). There was evidence of gender interaction, with social class for sum of skinfolds,  $p=0.03$ .

In boys body fat percent from bioimpedance showed no significant social class gradient. In girls there was an increase of 0.46% body fat with each descent in social class group, (95%CI 0.10 to 0.59). There was evidence of gender interaction  $p=0.01$ .

#### **5.4.3.3 Social class and central measures of adiposity by gender**

In boys waist circumference, waist: hip ratio and subscapular: triceps ratio showed no significant social class gradient, although waist: hip ratio in the lower three social class groups was higher than in the upper two. In girls both waist circumference and waist: hip ratio showed a positive association with lower social class (higher in lower social class groups). There was evidence of gender interaction, with social class for waist circumference,  $p=0.01$  but not for waist: hip ratio,  $p=0.12$ .

Subscapular: triceps ratio showed no relationship with social class in either boys or girls and no sign of interaction  $p=0.54$ .

#### **5.4.3.4 Effect of adjustment for pubertal status, town and ethnic group on social class and adiposity measures**

Pubertal status score did not show a social class gradient ( $p=0.74$  girls, 0.53 boys).

Town appeared to act as a confounder on two occasions; adjusting for town removed the positive gradient seen between BMI and decreasing social class in girls {0.77%, (95%CI 0.02 to 1.52%) became 0.53% (95%CI -0.25 to 1.32%)}, and revealed a positive relationship between skinfolds and decreasing social class in boys, changing from -1.26% (95%CI 0.68 to -3.18) to -2.12% (95%CI -0.14 to 4.23) after town adjustment. Other gradients seen were reduced a little by adjustment for town but not significantly.

Adjusting for ethnicity had little effect on the results seen.

#### **5.4.4 ETHNIC DIFFERENCES IN ADIPOSITY**

Table 5.7 summarises the differences between South Asian and European (white) children, separately for boys and girls. South Asian children were shorter and lighter, with lower BMI but higher sum of skinfolds and body fat percent. These differences only achieved statistical significance in girls (height, weight, body fat percent). The only significant difference in central measures of adiposity was for subscapular: triceps ratio; (higher in South Asians, in both genders).

Table 5.8 shows results of tests for gender interaction (right column); there was no significant evidence of gender interaction for any variables.

##### **5.4.4.1 Ethnic differences in height and weight**

Both South Asian boys and girls were shorter than European children; the height difference in girls was significant; boys 1.1cm, girls 2.9cm shorter. They were also lighter; although the weight difference was only significant in girls; South Asian boys were 4%, and girls were 7% lighter than European contemporaries.

##### **5.4.4.2 Ethnic differences in general measures of adiposity**

Lower BMI was seen in both genders in South Asians, (-2.86% boys, -3.40% girls) but failed to achieve significance, although in analyses with genders combined the difference was significant (-3.3%,  $p=0.03$ ), data not shown. Both South Asian boys and girls had higher sum of skinfold measures, 3.7% and 2.7% higher than Europeans respectively but these had wide confidence intervals and were not significant, even with genders combined ( $p= 0.63$ ). For body fat percent from bioimpedance the difference between European and S. Asian girls was significant; Asian girls had 1.54% higher body fat ( $p=0.02$ ); Asian boys did not have significantly higher body fat than European boys ( $p=0.34$ ). In analysis with combined genders the higher body fat percent in South Asians was just significant, Asians 27.8% body fat, Europeans 26.8% body fat ( $p$  for difference =0.05).

##### **5.4.4.3 Ethnic differences in central adiposity**

South Asian boys and girls had lower waist circumferences than Europeans (boys -1.12%, girls -1.99%) but these did not achieve statistical significance. Waist: hip ratio showed very little difference between the ethnic groups. Subscapular: triceps ratio was significantly higher in South Asians boys and girls, both about 0.1 higher than Europeans of the same sex. These findings were essentially similar in combined gender analyses.

#### **5.4.4.4 Effect of social class on ethnic differences in body build variables**

These analyses were performed with genders combined (data not shown). Adjustment for social class did not remove the ethnic differences in height, weight or BMI, only strengthening them a little.

Sum of skinfolds still had no significant relationship with ethnic group after social class adjustment. Body fat % from bioimpedance was higher in S.Asian children, but adjusting for social class reduced differences seen from 0.99% to 0.57%, losing significance.

Adjustment for social class revealed a significant difference in waist circumference between ethnic groups; South Asians' mean waist circumference was lower than Europeans by 2.53% (95%CI: 0.04 to 5.08). WHR was not significantly different between ethnic groups before or after social class adjustment. Social class adjustment had no impact on the subscapular: triceps ratio difference, which remained at 0.11(95% CI: 0.06 to 0.16)

### **5.5 DISCUSSION**

#### **5.5.1 PRINCIPAL FINDINGS**

There were marked variations between towns in height, weight, general and central adiposity markers. Boys and girls in high mortality towns were appreciably shorter and tended to have higher adiposity levels, though the differences were not statistically significant. Lower social class boys were significantly shorter and lighter. Some general (BMI, body fat percent) and central (waist circumference, waist: hip ratio) measures of adiposity showed an increase with lower social class in girls but not in boys. Skinfolds and subscapular: triceps ratio did not show a social class gradient in either gender. South Asian children in the study were shorter and lighter with lower BMI and higher body fat percent. The effect was stronger in girls. Of the central measures of adiposity only subscapular: triceps ratio showed a statistically significant ethnic difference, being significantly higher in South Asians.

#### **5.5.2 STRENGTHS AND WEAKNESSES OF THE STUDY**

The study design aimed to compare children from towns with high and low adult cardiovascular mortality and sufficient numbers responded for this to be achieved to some extent, although the use of only ten towns provided limited power for the high-low mortality comparisons (five versus five towns). As a result the 95% confidence intervals around high-low mortality town differences were wide and it was difficult to exclude even

quite appreciable differences in adiposity between high and low mortality towns. The full complement of adiposity measures was successfully collected in most children having physical measurements, producing a robust dataset. The data were collected within one school year, during term time, minimizing confounding by external factors.

Our study sample had a wide spread of socioeconomic status within a predominantly white group. Therefore these findings may be applicable to other socioeconomically diverse groups. However, as race has been shown to be significant factor in body composition it may not be appropriate to extend these findings to other, more ethnically diverse populations.

A potential weakness of the study is possible misclassification of social status (as with all contemporaneous studies): the new NS-SEC classification introduced 2-3 years later was needed as there was recognition that the 50 year old Registrar-General's classification was becoming less reliable. Reasons for this included existence of many 'new' jobs e.g. in IT which were not specifically covered in the old classification, and societal change which had raised or lowered the status of some occupations. However any misclassification would tend to weaken the associations seen and does not cast doubt on the findings of the study.

It would perhaps have been interesting to have collected information on household income, which would have enabled direct comparison with more recent large surveys in the UK that use NS-SEC. Information was collected on proxy measures of wealth e.g. car, computer and dishwasher ownership, size of house etc, but these have not been analysed for the purposes of this thesis.

### **5.5.3 RELATION OF THE FINDINGS TO OTHER STUDIES**

#### **5.5.3.1 Geographical (town) differences on the body size measures**

The pattern of taller stature in children from low mortality towns in Southern England in this study (on average 1.2 cm taller than those in high mortality towns in Northern England and South Wales) is consistent with findings in other studies (Hughes et al 1997) The findings are also consistent with the previous phase of the Ten Towns Study in children aged 8-11 years, when slightly smaller average differences in the same direction (on average 0.9 cm) were observed (Whincup et al 1996). The increasing difference in the older age-group suggests that the geographic height pattern is persisting and could be becoming more marked; it may also reflect the growth trajectory of these children as they approach adult life. These patterns are consistent in direction with the patterns reported in

adults, for example in the Health Survey for England in 1994, in which men in predominantly Northern mining and industrial areas were 2.5cm shorter and women 1.5 cm shorter than their contemporaries in more affluent and predominantly Southern areas.

The tendency for children from Northern England and Wales (particularly Rhondda) to have somewhat higher adiposity levels is consistent with the results of the findings in children in the Health Surveys for England 2002 (aged 2-15 and 16-24 years) and 2006 (age 2-15). In those studies, lower levels of adiposity were observed in children in the South and East (excluding London). Similar findings were observed in 3 year olds in the Millennium Children's Cohort study (Hawkins S et al 2008) and in the UK National Children's Obesity Database 2005-06, in children aged 4-5 and 10-11. The results are also consistent with those observed in the Ten Towns Study when the study population was aged 8-11 years; the difference has persisted suggesting that whatever factors are responsible for Rhondda children's excess adiposity have continued to operate. Regional variation in central measures of adiposity (waist circumference, waist: hip ratio, subscapular: triceps ratio) have not previously been described in this age group. Although such measurements were made in the Health Survey for England 2006 in children over 11 years, no data have been presented by region in the relevant report. These results in children are again broadly consistent with recent reports in adults, including those from the 1958 National Birth Cohort (in which subjects were measured at the age of 45 years); adiposity levels were markedly higher in participants in Wales than in Southern England (Strachan et al 2007).

#### **5.5.3.2 Social differences in the adiposity measures**

Our findings of increased adiposity (from BMI) in lower social class children, with a stronger effect in girls is in keeping with the Health Survey for England 2002 (which focused on young people). Mean BMI in the HSE age categories of 2-15 years and 16-24 years is not directly comparable with our findings, nor is the social class categorization (NS-SEC) but shows similar magnitude of changes with social class, and also lower BMI in the lowest social class group in boys that was seen in our study. We did not see the relatively higher levels of adiposity in higher social class girls described by Wardle (Wardle et al 2006 {BMJ}) from age 13 onwards; this may be due to differences in the study settings (Wardle's was in London) or in calculating socioeconomic status, as Townsend index was used in their study. The social class differences were not present in the Ten Towns children when they were aged 8-11 (Whincup 1996). Power examined

effects of social class in childhood on adult obesity, defined by BMI in the 1958 birth cohort (Power et al 2007) finding that social class gradients in obesity were not present at age 11, but were established by age 16 in girls and were present in both genders when next surveyed at age 23. Our study children would appear to be following a similar pattern.

The height difference with social class in boys is consistent with previous findings in Britain in men (Gregory et al, 1990, Marmot et al, 1984). The mean difference of 1.9 cm between social class I and IV/V for boys in our study is very similar to the value of 1.8cm between I/II and IV/V in the 1985 data used by Gregory et al, suggesting little change in the social class gradient and whatever factors are responsible for it, although in the context of an overall increase in adult height. The social class differences in female height however were not as marked in our study; 1.1 cm between social class I and IV/V, which did not achieve statistical significance, versus 2.8 cm in Gregory et al. This may represent a genuine change, with the absence of stunting caused by poor nutrition, but it is hard to make this apply to females and not at all to males.

The pattern seen in BMI in the study boys is very similar to that in adult men in the 1998 HSE, with the lowest BMI in extreme SC groups. In study boys BMI is lowest in SCIV/V but in the 1998 HSE it is lowest in I in adult men. In adolescents BMI (reflecting lean and fat mass) is more height dependent than in adults; it may be that height is confounding the relationship between BMI and social class in these children ie they are not thinner but their overall body size is less.

In boys waist circumference is lowest in social class IV/V unlike in adult men, where it rises. Also WHR in boys increases to social class III but then decreases again a little, although the overall trend remains positive similar to adult men (1998, 2003 HSE) and suggests some increase in central adiposity, particularly in the light of coincidental decreasing skinfold measurements and BMI.

The patterns seen in the girls in our study for significantly increasing BMI, WHR and waist circumference with lower social class are similar to those seen in adult women but with a slight decrease from social class IIINM to IIIM before rising again. The social class differences in percent body fat from bioimpedance in girls add to the existing evidence of social class differences in adiposity.

Social class differences in adiposity measures were not seen in an earlier wave of the study (Whincup 1996) when the children were aged 8-11. There may be a variety of explanations for this; maybe the key time for the development of the differences is the late teens with different social class groups following diverging trajectories for adiposity, as found for

girls by De Spiegelaare (1998). Also social class in our study was defined by the occupation of the head of the household; perhaps this is not appropriate for the teenagers, who are making the transition to their own social class. A measure of educational attainment may be more appropriate in this group. In boys and to some degree in girls the social class differences in adiposity may not yet have developed as at this age children are still generally a lot more physically active than in later life. Those from lower social classes may be more active in the labour market at a younger age due to financial constraints; this may explain the lower adiposity measures in boys in SCIV and V especially. They also may have less access to transport.

A recent paper, looking at the effect of social class on the 1958 UK birth cohort, found that both social class in childhood and adulthood were independently associated with a range of adverse health outcomes at age 44/45 including higher BMI and adverse blood lipid profile (Power et al, 2007). The effects were strongest in those who started and remained in, lower social class groups ie implying a cumulative effect. In our study children it appears that by ages 13-16 years social class differences are emerging already.

#### **5.5.4 ETHNICITY AND ADIPOSITY MEASUREMENTS**

Studies in South Asian teenagers in the UK have found higher rates of obesity and overweight than in the general population, particularly in boys (Saxena S et al, 2004, Wardle et al 2006). Saxena's study used data from the 1999 Health Survey for England and looked at children and young adults aged 2-20 years and found particularly high rates of overweight and obesity in South Asian boys. Wardle's London school-based study (Wardle et al 2006) found evidence of higher adiposity in South Asian boys but not girls compared to white contemporaries. Findings were of lower height, weight, BMI ( $0.4 \text{ kg/m}^2$  lower) and waist circumference( $0.8\text{cm}$  lower) in South Asian girls than white girls at age 11-12. South Asian boys in this study were more adipose than white boys, being shorter and lighter but with higher BMI ( $0.1\text{kg/m}^2$  higher) and waist circumference ( $0.7\text{cm}$  higher). When comparing Ten Towns' children to similarly aged children in Wardle's London study, measured in 1999, it is noted that although BMIs are a little higher in the London children compared to Ten Towns children (Asian boys: 1.5, white boys 0.9, Asian girls 0.7, white girls  $0.9 \text{ kg/m}^2$  higher); the difference is much more noticeable in waist circumference (Asian boys 6.2cm, white boys 5.2cm, Asian girls 4.4cm, white girls 5.4cm higher) in London than in Ten Towns study. In this case the difference between London/

not London is greater than the ethnic differences in waist circumference. It is difficult to ascribe a cause to these differences but the most likely seems different 'cultural environment', possibly observer variation in measurement technique and perhaps different ethnic mix in London. In the Ten towns study most South Asians were in Rochdale, where there are fairly equal numbers of South Asian and White children, within a small town, therefore enabling contrast of the two groups in quite a unique way, as 'other' unquantifiable differences are minimised.

The direction of the ethnic differences seen in the children, with South Asian children being shorter and lighter, with lower BMI but higher body fat, is in line with differences seen in adults (Bhopal et al, 1999, McKeigue et al, 1991). Differences are that our Asian boys did not have higher waist circumference or waist: hip ratio; the only evidence of 'centrality' at this age is the higher subscapular: triceps ratio. South Asian girls are less like South Asian women previously studied in that they have BMI measures lower than European girls, whereas in adult women the opposite is true. This may be due to differences in lifestyle patterns, particularly exercise, which are limited in the school environment but may become more pronounced later.

Information on parent's country of birth was not analysed in this thesis, but the vast majority were Pakistani Muslim, whose parents or grandparents came to work in the textile industry. Social class as defined by the Registrar General's classification may denote a raft of social and economic factors for the white British population which do not apply equally to the Asian population, and may limit the value of adjusting results seen in Asians for social class.

The current data does not allow us to comment on whether there are behaviour differences between the groups that may account for the higher body fat. In case height was confounding the relationship between ethnic group and adiposity measures the analyses were repeated with height in the model as a continuous variable. However this made little difference to the results. The relatively raised body fat levels seen in Asian children shows that the younger generation is already exhibiting characteristics suggestive of an increased susceptibility to cardiovascular disease and related pathologies. In this they are similar to their parents. This may suggest some genetic, possibly endocrine cause for these differences or an ongoing environmental cause.

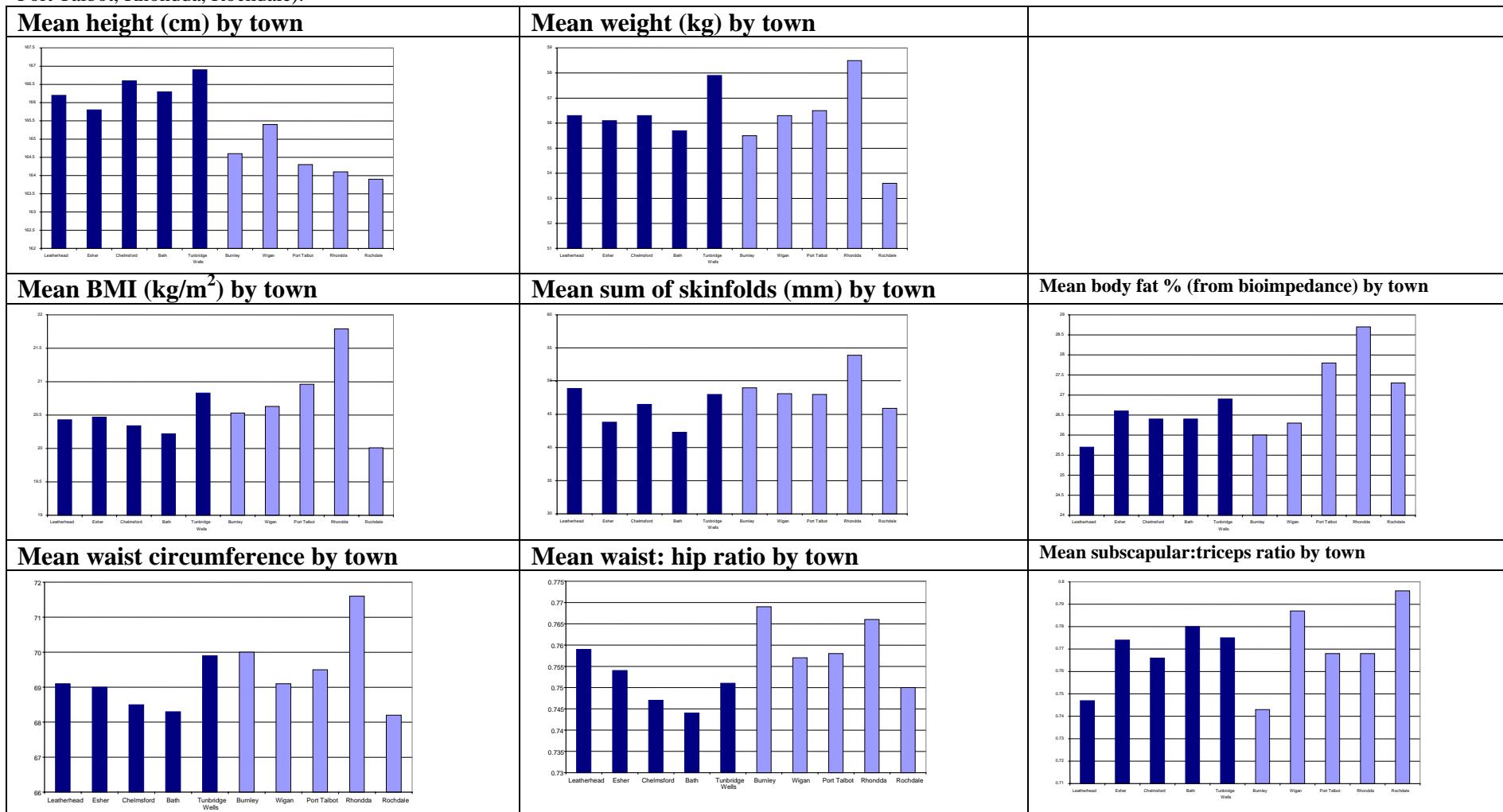
## **5.6 IMPLICATIONS FOR AETIOLOGY AND PREVENTION**

Differences in adiposity in the social and ethnic patterns seen in adults are already emerging in 13-16 year old adolescents. This suggests that the aetiological factors responsible for the adult differences in adiposity are starting to act by 13-16 years. This in turn has implications for the need for early prevention of excess adiposity in these high risk groups. Power (Power et al 2007) found that early childhood social class remained an important determinant of adult obesity at age 33 years, lower social class being linked to higher adult obesity. The reasons for this are not clear; the associations were independent of educational level and parental BMI. If the reasons for this are cultural, around habits and patterns of behaviour, they may be amenable to intervention. Ethnic differences in weight, ponderal index (and insulin resistance) were noted in South Asian children in the study at age 8-11 (Whincup et al, 2002). South Asian children at 13-16 continued to have lower weight and weight-for-height (BMI), with slightly higher adiposity; separate investigation showed that South Asian children had also developed higher fasting insulin and glucose than white children, in addition to insulin resistance (Whincup et al, 2005). The tendency to type II diabetes appears to be developing in the South Asian children in adolescence, accompanied by general and truncal adiposity. Early intervention to prevent excess adiposity in South Asian children needs to be culturally appropriate; Saxena's study (Saxena et al 2004) suggested higher levels of obesity in Pakistani children than other South Asian children; further studies may be needed to explore these differences, whether they originate in cultural differences or other factors. Difficulties in accessing leisure activities may be a factor for Muslims for example; women-only sessions may need to be advertised in cultural settings such as community centres or through schools.

Geographic differences in adiposity seem to be emerging in the Ten Towns children, although not in a high adult cardiovascular mortality town: low cardiovascular mortality town pattern. This may be due to changes in pre-existing factors in the towns, or the emergence of new more important risk factors for child adiposity which are as yet incompletely identified. Studies comparing area-level geographic inequalities for example between Rhondda and Bath or Chelmsford may help to clarify why Rhondda appears to be such an obesogenic environment. This type of study may also help to clarify the role of social class differences; and whether change needs to be targeted at the individual or the area (McCarron, Lawlor 2003).

**Figure 5. 1 Town differences in height, weight and adiposity measures.**

Order of towns along x axis: 5 'low' adult cardiovascular mortality towns (Leatherhead, Esher, Chelmsford, Bath, Tunbridge Wells; then 5 'high' towns (Burnley, Wigan, Port Talbot, Rhondda, Rochdale).



**Table 5. 1 Social class distribution by town in the study population**

Rows total to right hand column ie social class make-up in each town

	Social Class					
<b>Low cardiovascular mortality towns (mean)</b>	1	2	3(Non-Manual)	3 (Manual)	4 & 5	Total number of study children
Leatherhead	49 14.7%	144 43.1%	27 8.1%	82 24.6%	32 9.6%	334
Esher	28 11.4%	101 41.1%	31 12.6%	57 23.2%	29 11.8%	246
Chelmsford	30 10.8%	91 32.7%	51 18.3%	72 25.9%	34 12.2%	278
Bath	42 15.0%	85 31.1%	39 14.3%	87 31.9%	52 19.1%	281
Tunbridge Wells	38 10.9%	132 37.6%	47 13.3%	92 26.1%	43 12.1%	352
Total children in low town by SC	187 12.1%	553 37.3%	195 14.6%	390 24.6%	190 11.3%	1491 (100% for row)
<b>High cardiovascular mortality towns (mean)</b>	1	2	3(Non-Manual)	3 (Manual)	4 & 5	Total number of study children
Burnley	10 3.7%	85 31.1%	39 14.3%	87 31.9%	52 19.1%	273
Wigan	21 8.2%	49 19.2%	31 12.2%	97 38.0%	57 22.4%	255
Port Talbot	12 4.9%	41 16.6%	29 11.7%	108 43.7%	57 23.1%	247
Rhondda	15 5.4%	52 18.6%	28 10.4%	114 40.9%	70 25.1%	279
Rochdale	9 3.6%	59 23.5%	29 11.6%	83 33.1%	71 28.3%	251
Total children in high town by SC	67 4.2%	286 21.9%	156 12.5%	489 36.5%	307 24.9%	1305 100% for row

**Table 5. 2 Ethnic group distribution by town: showing the 3 towns with largest Asian population**

	Boys		Girls	
	White	Asian	White	Asian
Rochdale	76 (52.4%)	69 (47.6%)	93 (62%)	57 (38%)
Burnley	114 (95%)	6 (5%)	91 (89.2%)	11 (10.8%)
Esher	114(96.5%)	4 (3.5%)	107 (95.3%)	5 (4.7%)

**Table 5. 3 Social class distribution by ethnic group in the study population (percent of study population)**

	Social Class				
	1	2	3 Non manual	3 Manual	4&5
White	8.8	30.9	13.6	30.0	16.8
Asian	2.0	21.6	3.9	39.2	33.3

**Table 5.4** Differences in adiposity by town Least Squared means (SE) adjusted for gender, age and (skinfolds, waist, WHR) observer

TOWN	Survey pair 1998-99	No. of subjects	Age (years)	Height (cm)	Weight (kg)	Body mass index (kg/m <sup>2</sup> )	Log sum of skinfolds	Body fat % from bioimpedance	Waist circumference (cm)	Waist : hip ratio	Subscapular : triceps ratio
<b>Low Mortality</b>											
Leatherhead	1	292	14.89 (0.03)	166.2 (0.4)	56.3	20.43	48.9	25.7 (0.3)	69.1	0.759	0.747 (0.013)
Esher	2	225	15.04 (0.03)	165.8 (0.5)	56.1	20.47	43.8	26.6 (0.4)	69.0	0.754	0.774 (0.014)
Chelmsford	3	261	15.08 (0.03)	166.6 (0.4)	56.3	20.34	46.5	26.4 (0.3)	68.5	0.747	0.766 (0.014)
Bath	4	242	14.58 (0.03)	166.3 (0.4)	55.7	20.22	42.3	26.4 (0.3)	68.3	0.744	0.780 (0.014)
T. Wells	5	304	14.66 (0.03)	166.9 (0.4)	57.9	20.83	48.0	26.9 (0.3)	69.9	0.751	0.775 (0.014)
<b>Average of 'low' towns</b>			14.85	166.4	56.5	20.5	45.9	26.4	69.0	0.751	0.768
<b>High Mortality</b>											
Burnley	1	226	14.81 (0.03)	164.6 (0.5)	55.5	20.53	49.0	26.0 (0.4)	70.0	0.769	0.743 (0.015)
Wigan	2	235	15.05 (0.03)	165.4 (0.5)	56.3	20.63	48.1	26.3 (0.4)	69.1	0.757	0.787 (0.014)
Port Talbot	3	210	15.24 (0.03)	164.3 (0.5)	56.5	20.96	48.0	27.8 (0.4)	69.5	0.758	0.768 (0.015)
Rhondda	4	287	14.50 (0.03)	164.1 (0.4)	58.5	21.79	53.9	28.7 (0.3)	71.6	0.766	0.768 (0.013)
Rochdale	5	301	14.70 (0.03)	163.9 (0.4)	53.6	20.01	45.9	27.3 (0.3)	68.2	0.750	0.796 (0.013)
<b>Average of 'high' towns</b>			14.86	164.5	56.1	20.8	49.0	27.2	69.7	0.760	0.772
<b>P value for overall town difference</b>			<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001	<0.0001
<b>Mean (SE) difference (low - high)</b>			0.10 (0.05)	1.9 (0.4)	0.8%	-1.5%	-6.3%	-0.82 (0.47)	-1.0%	-1.2%	-0.4%
<b>95% Confidence Interval</b>			-0.12 to 0.14	0.8 to 3.1	4.8% to -3.1%	2.1% to -5.1%	-15.0% to 3.3%	0.09 to -1.74	1.2% to -3.2%	0.2% to -2.2%	0.8% to -1.5%
P value			0.8	0.02	0.7	0.5	0.3	0.2	0.4	0.1	0.6

**Table 5.5** Rochdale: Least Squared means (SE) adjusted for gender, age and (skinfolds, waist, WHR) observer

Ethnic group	No. of subjects	Age	Height (cm)	Weight (kg)	Body mass index (kg/m <sup>2</sup> )	Log sum of skinfolds	Body fat % from bioimpedance	Waist circumference (cm)	Waist : hip ratio	Subscapular : triceps ratio
<b>South Asian</b>	126	14.7 (0.05)	161.9 (0.6)	50.8	19.43	46.6	28.1 (0.5)	67.3	0.754	0.836
<b>White</b>	169	14.7 (0.05)	165.1 (0.5)	55.7	20.45	44.6	26.7 (0.4)	69.0	0.750	0.771
<b>All, adj for ethnicity</b>	297		164.9 (0.5)	55.0	20.28	45.5	26.8 (0.4)	68.6	0.750	0.761

**Table 5. 6 LS means: Social Class (SC) adjusted for age, observer (skinfolds, waist and waist/hip measurements) and pubertal status.**

	SC1	SC2	SC3NM	SC3M	SC4 & SC5	Increment 95% confidence interval	P for trend	After adj for town; Increment (95% CI)	After adj for town and ethnicity; Increment (95% CI)
<b>Boys</b>									
<b>Age years +</b>	14.89 (0.05)	14.79 (0.03)	14.78 (0.05)	14.86 (0.03)	14.87 (0.04)	0.01 (-0.01 to 0.04)	0.34		
<b>Height m +</b>	169.7 (0.6)	169.7 (0.4)	170.0 (0.6)	168.6 (0.4)	167.8 (0.5)	-0.65(-0.33 to -1.33)	<0.0001	-0.49 (-0.16 to -0.81)	-0.48 (-0.16 to -0.81)
<b>Weight kg *</b>	57.53	58.58	58.57	57.83	56.08	-1.00% (-0.19% to -1.81%)	0.02	-1.00% (0.16% to -1.83%)	-1.00% (0.17% to -1.84%)
<b>BMI *</b>	20.03	20.42	20.31	20.40	19.91	-0.22% (-0.90% to 0.46%)	0.52		
<b>Sum of skinfolds *</b>	39.21	40.86	40.42	39.50	37.74	-1.26% (0.68% to -3.18%)	0.20	-2.12% (0.14% to -4.23%)	-2.17% (0.20% to -4.12%)
<b>Fat % from bioimpedance +</b>	21.68 (0.52)	21.93 (0.30)	21.57 (0.47)	21.68 (0.29)	21.72 (0.40)	0.00 (-0.26 to 0.26))	0.98		
<b>Waist circ*</b>	70.94	71.40	71.73	71.25	70.38	-0.19% (-0.68% to 0.31%)	0.45		
<b>Waist:hip ratio *</b>	0.783	0.783	0.788	0.787	0.785	0.19% (-0.08% to 0.27%)	0.16		
<b>Subscapular:triceps ratio +</b>	0.827 (0.02)	0.801 (0.01)	0.776 (0.018)	0.822 (0.011)	0.797 (0.015)	-0.03% (1.00% to -1.04%)	0.96		
<b>Girls</b>									
<b>Age years +</b>	14.83 (0.06)	14.80 (0.03)	14.91 (0.05)	14.82 (0.03)	14.87 (0.04)		0.37		
<b>Height m +</b>	162.6 (0.6)	162.5 (0.3)	162.4 (0.5)	162.2 (0.3)	161.5 (0.4)	-0.24(0.04 to -0.55)	0.10		
<b>Weight kg *</b>	53.72	55.18	56.42	55.04	55.74	0.47% (-0.36% to 1.3%)	0.27		
<b>BMI *</b>	20.35	20.92	21.42	20.96	21.40	0.77% (0.02% to 1.52%)	0.04	0.53% (-0.25% to 1.32%)	0.57% (-0.21% to 1.36%)
<b>Sum of skinfolds *</b>	54.08	56.27	58.17	56.80	59.10	1.10% (-0.58% to 2.82%)	0.20		
<b>Fat % from bioimpedance +</b>	31.22 (0.50)	31.31 (0.26)	32.42 (0.40)	32.06 (0.26)	32.96 (0.34)	0.46 (0.23 to 0.69)	0.0001	0.35 (0.10 to 0.59)	0.33 (0.08 to 0.59)
<b>Waist circ*</b>	66.01	67.19	68.46	67.60	68.60	0.67% (0.13% to 1.22%)	0.02	0.58% (0.02% to 1.15%)	0.61% (0.05% to 1.18%)
<b>Waist:hip ratio *</b>	0.715	0.722	0.730	0.725	0.732	0.46% (0.15% to 0.77%)	0.004	0.36% (0.04% to 0.69%)	0.37% (0.05% to 0.69%)
<b>Subscapular:triceps ratio +</b>	0.733 (0.02)	0.716 (0.01)	0.762 (0.016)	0.729 (0.011)	0.753 (0.014)	0.007 (0.017 to -0.329)	0.19		

NB 'increment' is change in body size measure per unit class change in social class

+ Mean values are presented with standard errors. Changes in adiposity measures per change in social class are presented in absolute values.

\* Variables log transformed for analysis. Geometric means are presented. Changes in adiposity measures per social class are presented as percentage values.

**Table 5. 7 Ethnic differences, separately by gender**

LS means adjusted for age, observer (skinfolds, waist, WHR), pubertal status, town.

	EUROPEAN Mean (std. err)	ASIAN Mean (std. err)	Mean difference European- Asian	95% CI	P (no ethnic difference)
<b>BOYS</b>					
<b>Number of subjects</b>	1289	91			
<b>Age (years)</b>	14.82 (0.02)	14.84 (0.08)	0.02	0.18 to -0.14	0.77
<b>Height (cm)</b>	169.0 (0.2)	167.9 (0.9)	-1.11	0.74 to -2.96	0.24
<b>Weight (kg)</b>	57.65	55.35	-4.15%	0.77% to -9.30%	0.10
<b>Body mass index (kg/m<sup>2</sup>)</b>	20.26	19.69	-2.86%	1.15% to -7.03%	0.17
<b>Log sum of skinfolds</b>	39.35	40.84	3.65%	13.90% to -7.82%	0.51
<b>Body fat % from bioimpedance</b>	21.69 (0.16)	22.43 (0.73)	0.73	-0.77 to 2.24	0.34
<b>Waist circumference (cm)</b>	71.13	70.33	-1.12%	1.74% to -4.07%	0.44
<b>Waist/hip ratio</b>	0.786	0.783	-0.26%	1.26% to -1.81%	0.74
<b>Subscapular : triceps ratio</b>	0.802 (0.006)	0.899 (0.028)	0.097	0.040 to 0.154	0.001
<b>GIRLS</b>					
<b>Number of subjects</b>	1142	82			
<b>Age (years)</b>	14.82 (0.02)	14.82 (0.08)	0.005	0.17 to -0.16	0.95
<b>Height (cm)</b>	162.2 (0.2)	159.3 (0.8)	-2.89	-1.35 to -4.43	<0.0002
<b>Weight (kg)</b>	55.33	51.57	-7.27%	-2.40% to -12.37%	0.003
<b>Body mass index (kg/m<sup>2</sup>)</b>	21.07	20.37	-3.40%	0.85% to -7.84%	0.12
<b>Log sum of skinfolds</b>	56.90	58.47	2.69%	11.34% to -6.79%	0.57
<b>Body fat % from bioimpedance</b>	31.91 (0.14)	33.45 (0.62)	1.54	0.27 to 2.82	0.02
<b>Waist circumference (cm)</b>	67.70	66.38	-1.99%	1.00% to -5.07%	0.20
<b>Waist/hip ratio</b>	0.726	0.729	0.50%	2.15% to -1.18%	0.55
<b>Subscapular : triceps ratio</b>	0.726 (0.006)	0.831 (0.025)	0.105	0.050 to 0.157	<0.0001

**Table 5. 8 Gender interaction (for social class and for ethnic group)**

	P for sex interaction for social class (when adj age, obs {as appropriate}, pubertal stage, town)	P for sex interaction for ethnic group (when adj age, obs {as appropriate}, puberty stage, town)
<b>Height (m)</b>	0.12	0.16
<b>Weight</b>	0.03	0.47
<b>BMI</b>	0.08	0.98
<b>Log sum of skinfolds (std. err)</b>	0.03	0.69
<b>Fat % from bioimpedance (std. err)</b>	0.01	0.78
<b>Waist circ</b>	0.01	0.94
<b>Waist:hip ratio (std. err)</b>	0.12	0.22
<b>Subscapular:triceps ratio (std. err)</b>	0.54	0.46

## CHAPTER 6: THE RELATIONS OF PARENTAL ADIPOSITY, INFANT FEEDING AND PHYSICAL ACTIVITY TO ADIPOSITY IN 13-16 YEAR OLDS

### **6.0 SUMMARY POINTS**

- This chapter examines a range of determinants of childhood adiposity including familial factors (parental body build), an exposure operating soon after birth (infant feeding) and contemporary health behaviour (physical activity level).
- Hypotheses tested include: that parental adiposity is associated with child's adiposity; that breast feeding has a protective effect against subsequent development of adiposity in childhood; and that differences in higher adiposity levels in teenagers are associated with lower physical activity levels.
- Data are presented on selected determinants of adiposity in a school-based cross-sectional survey of 2645 British children aged 12.7 to 16.4 years (1235 girls 1410 boys) Anthropometry measures included height, weight, skinfolds and circumference measurements of waist and hip. A full set of anthropometry measurements were obtained on 1197 girls and 1370 boys (2567 children).
- Information on parental height and weight were collected by self-administered parental questionnaire, allowing calculation of maternal BMI for 74% and paternal BMI for 69% of children.
- Children's adiposity measures were then examined by fifths of parental BMI distribution. The highest fifth of the BMI distribution of both mothers and fathers included only subjects with BMIs over 30 kg/m<sup>2</sup> (ie obese).
- Children's weight, BMI, sum of skinfolds, body fat % and waist circumference all increased sequentially with increasing fifths of parental BMI. Waist: hip ratio in children increased with BMI in fathers but not mothers. Subscapular:triceps ratio in boys decreased with paternal but not maternal BMI. Subscapular:triceps ratio in girls increased with maternal but not paternal BMI.
- Mean adiposity measures were lower among subjects who had been exclusively breast fed in the first three months; differences in body mass index, sum of skinfolds, fat mass from bioimpedance and waist-hip ratio were statistically significant. However adjusting for social class, maternal and child smoking and maternal body mass index greatly reduced the differences, with only differences in sum of skinfolds remaining significant.

- It was observed that boys aged 15 tended to exercise more than those aged 13; the reverse was true of girls. This suggests that participation in exercise increases with age at 13-15 in boys and falls in girls. At age 15 boys were twice as likely as girls to take exercise 4 or more times a week (60% vs 30%). Overall 25% of girls took no exercise, as was the case for 12% of boys.
- Child's self-reported habitual level of exercise and perception of themselves as an active or inactive person are both related to adiposity measures. For habitual exercise taken the strongest relationships in both sexes were seen with sum of skinfolds and body fat % from bioimpedance, which both decreased with increasing exercise, as did waist circumference in boys. The effect was stronger in boys than it was in girls. Children who perceived themselves as active had lower general and central measures of adiposity. Children who did a little exercise tended to have higher adiposity measures than those who did none. Children who perceived themselves as 'a bit less active than average' were the most adipose.

## **6.1 INTRODUCTION**

This chapter examines associations between the measurements of adiposity in 13-16 year old British school children in the Ten Towns Study and three factors that are thought to influence adiposity, including familial factors (parental body build), an exposure operating soon after birth (infant feeding) and a contemporary health behaviour (physical activity level).

### **6.1.1 PARENTAL ADIPOSITY**

Studies of parental influence on offspring's BMI have found evidence of increased adiposity in children with obese or overweight parents (Burke et al, 2001, Duran-Tauleria et al, 1995). Duran-Tauleria found an increased likelihood of children being in the top quarter of the weight-for-height distribution if their father or mother was obese (OR 1.85, 1.90 respectively) and even higher if both parents were obese (OR 3.48). Burke's study found that over the nine years of their study, involving 3 yearly surveys of their cohort of 219 families, there were consistently higher BMIs in children whose parents were obese or overweight.

Longitudinal studies that follow the child to adulthood have found persisting associations between parental and child BMI as the child ages. Laitinen et al confirmed a relationship at ages 1yr, 14years and 31 years (Laitinen et al, 2001). Tracking has been shown to occur,

with obese children more likely than reference population to remain obese into adult life. These children are also more likely to have obese parents. The odds of children with two obese parents being obese themselves at age 33 were 8.4 (boys) and 6.8 (girls), these were children from the 1958 British birth cohort; parental heights and weights were self-reported when the children were 11 years old (Lake et al, 1997). I would therefore hypothesize that higher levels of parental BMI would be associated with higher levels of adiposity in the Ten Towns children.

### **6.1.2 BREAST FEEDING AND ADIPOSITY**

It has been suggested that early life exposures are involved in the development of obesity (Kuh, Ben Shlomo 2004). Among these it has been suggested that breast feeding may have a protective effect, when compared with formula feeding (Gillman et al, 2001)

However, recent systematic reviews of the published evidence suggest that the association may be weaker than earlier thought. A review of the evidence relating breast feeding to mean body mass index (BMI) suggested that there was little or no relationship, particularly after adjustment for potential confounders including social class, maternal adiposity and cigarette smoking (Owen et al, 2005). Most existing evidence on the effects of breast feeding use body mass index as the outcome measure for assessing obesity. This has limitations, particularly in the assessment of central adiposity, which is strongly related to metabolic and cardiovascular risk. I would therefore hypothesize that breastfeeding would be associated with lower levels of adiposity in the Ten Towns children, but this effect might be weak.

### **6.1.3 PHYSICAL ACTIVITY AND ADIPOSITY**

There is a general feeling that physical activity levels in young people in developed societies have declined and that this is a major contributory factor in the rise of obesity in this age group. It is difficult to establish with certainty that changes have occurred because reliable, comparable data on children's activity levels from earlier times is difficult. A recent review (Dollman et al, 2005) concluded that physical activity in clearly defined contexts such as active transport, school physical education and organised sport is declining in many countries including the UK. A key issue is the discrimination between habitual activity, and actual physical fitness, The European Youth Heart Study, a multicentre international study investigating the prevalence and cause of cardiovascular disease risk factors in children aged 9-15 years, measured both actual physical activity for

4 days and also physical fitness using an exercise bike test. They found that BMI, sum of skinfolds and waist circumference all decreased with increasing physical activity levels, while fitness increased (Andersen et al, 2006). I would therefore hypothesize that higher levels of reported physical activity would be associated with lower levels of adiposity in the Ten Towns children.

## **6.2 AIMS AND OBJECTIVES**

The aim of this study is to explore individual determinants of adiposity in a group of schoolchildren aged 13-16 years. The objectives of this section include:

- To examine the association between parental BMI and markers of both general and central adiposity measures in adolescence (Section 6.4.1).
- To examine the association between breast-feeding and markers of both general and central adiposity measures in adolescence (Section 6.4.2).
- To examine the relationship between exercise habits and leisure activities and the different measures of adiposity (Section 6.4.3).

## **6.3 SUBJECTS AND METHODS**

This study is based on the findings from a school-based survey carried out in ten towns in England and Wales in 1998-99 in 2645 12-16 year old children. Measurements of height, weight, skinfolds, waist and hip measurements and bioimpedance (body resistance) were made as described in Chapter 3. In a questionnaire to the parents, mother and father (not adoptive father) were asked to report their current height in feet and inches or centimetres and to report their weight in kilograms or stones and pounds. Parents were asked to specify which infant feeding method was used in the first three months of the child's life (breast fed only, bottle fed only and mixed feeding) and parents were also asked about maternal smoking status (current, never or ever).

Pupil exercise information was obtained via pupil questionnaire; two of these questions were used in this analysis. Question 8.10 asked pupils:

***Which one of the following statements describes you best?***

***All or most of my free time is spent doing things which involve little physical effort (eg. doing homework, talking to friends, watching TV)***

- Once or twice a week I do things in my free time which involve some physical effort (eg walking, cycling, table tennis)*
- I quite often (4-6 times a week) do things in my free time which involve physical exercise*
- I very often (7 times a week or more) do things in my free time which involve physical exercise*

Question 8.11 asked for activity level in comparison with peers:

*Compared to other pupils of your own age and sex, would you say that you are:*

- Much less active*
- A bit less active*
- About average*
- A bit more active*
- Much more active*

Pupil smoking status was based on a confidential questionnaire completed by the pupil (current, ever or never). Social class was coded from parental occupation in accordance with the registrar general's (ONS) 1990 coding manual. Social class of the head of household was used; usually the father figure; if absent then mother's social class was used. Information on occupation was obtained from parental questionnaire if available; and from the child questionnaire if parental information was not available.

Detailed information on subjects and methods and all measuring techniques are included in Chapter 3.

### 6.3.1 STATISTICAL METHODS

Parental heights and weights were converted to metric measures where necessary. Weights and BMI measures for parents were log transformed for analysis following inspection of the unlogged data. Logistic regression was carried out using SAS, as in previous chapters. The analysis and tables for section 6.4.2 on breastfeeding were completed for a research paper by S. Kaye, research assistant at SGHMS, using STATA/SE version 9 for WINDOWS software (Stata Corporation, College Station, TX).

## **6.4 RESULTS**

Among 2567 pupils who had physical measurements of adiposity, parental questionnaires were returned for 1991 (78% response rate) of which 1784 (89%) parental questionnaires were completed by mothers, 189 (10%) by fathers and 18 (1%) by others (other relative or guardian). A valid response to exercise questions was available for 89% of pupils: question 8.10 2292 pupils, question 8.11 2280 pupils, both 52% boys, 48% girls.

### **6.4.1 PARENTAL BMI AND ADIPOSITY IN ADOLESCENCE**

Parental heights were available for 1972 mothers and 1913 fathers, representing 77% and 74% of children with physical measurements. Similar values for weight were 1919 (75%) and 1793 (70%), allowing calculation of mother's BMI for 75% of girls and 73% of boys and father's BMI for 70% of girls and 68% of boys, following removal of 4 paternal results which were invalid (Two heights in excess of 7 feet, weight of 192kg, BMI of 61).

Parental weight and BMI showed right skew, with mean values higher than the median (Table 6.1). The values for mean and median BMI for fathers (29.4, 29.1) are higher than expected being almost at an obese level. The parental BMI distributions were divided into fifths, allowing comparison of children in different groups for parental BMI. The fifths are displayed in the first row in Tables 6.2 (mothers) and 6.3.(fathers). Fifths were chosen rather than using specific BMI cutoffs as these parental heights and weights are self-reported and comparing them with each other in a group minimises the effect of systematic reporting error, which comparing to standard cut offs would not (Lake 1997). The highest fifth generated for both mothers and fathers contain only BMI values that would conventionally be considered obese ie over 30 kg/m<sup>2</sup>. Adjustment for the other parent's BMI was included in the statistical model as a continuous variable.

Height shows no relationship with parental BMI, except that in boys there was a slight increase with maternal BMI, which did not achieve conventional significance. All measures of general adiposity in both sexes showed highly significant increases with increasing fifths of parental BMI, showing particularly marked increases between the fourth and highest groups for parental BMI. For both boys and girls the magnitude of the increases in adiposity measures tended to be higher with father's BMI than mother's BMI. There was evidence of gender interaction for BMI (with mother's BMI, p=0.01).

Of the measures representing central adiposity, waist circumference showed a positive relationship with parental BMI, as did waist: hip ratio for both sexes with father's BMI.

Waist: hip ratio showed no significant relationship with maternal BMI. Subscapular:triceps ratio only showed a positive relationship with maternal BMI in girls, with a decrease in subscapular: triceps ratio between the lowest and second groups of maternal BMI before rising. Girls' subscapular: triceps ratio showed a similar but non-significant relationship with father's BMI, whilst boys' subscapular: triceps ratio tended to fall with increasing parental BMI. There was evidence of gender interaction for subscapular: triceps ratio (with father's BMI,  $p=0.01$ ), but not for other variables.

#### **6.4.2 INFANT FEEDING AND ADIPOSITY**

Information on infant feeding was available for 2443 subjects (1148 girls, 1295 boys) of 2645 measured. Subsidiary analyses examining the influence of confounding factors were carried out in a sub-population of 1,302 subjects with complete data on parental social class, maternal cigarette smoking, maternal BMI and subject cigarette smoking. These subjects were slightly taller on average than subjects with incomplete data, after adjustment for town (166.1 cm vs 165.4cm,  $p=0.04$ ). Additional adjustment for sex and age did not remove the differences seen. Adiposity variables did not vary between the subgroups.

The relations between infant feeding method and mean adiposity measures (adjusted for age, sex, town, ethnicity and observer) are shown in Table 6.4, separately for boys and girls, and with both sexes combined. In the combined analysis, subjects who had been exclusively breast fed for three months were on average 1.06cm taller, had lower BMI (0.37), sum of skinfolds (2.04) and fat mass (0.63%) than those who were exclusively formula fed. Their mean waist: hip ratio was lower and both waist circumference and subscapular:triceps ratio were slightly lower, at the margins of statistical significance. The direction of differences in both sex-specific analyses were generally similar to those in the combined analyses, although associations for height, BMI, body fat mass % and waist: hip ratio were significant only in girls and associations for sum of skinfolds were significant only in boys. Analyses to test for gender interaction i.e. whether gender was acting as an effect modifier in any of the anthropometric measures was carried out. As none of the results were significant all subsequent analyses were conducted using the sexes combined.

The effects of adjustment for potential confounding factors for girls, boys and genders combined are presented in Table 6.5, in analyses conducted in 1302 subjects with complete data on all potential confounding variables. The differences observed between formula fed

and breast fed infants in the basic model are very similar to the differences observed in Table 6.4, despite Table 6.5 showing a subset of participants. Following adjustment most of these differences were reduced. The difference in height was reduced by half (to 0.46cm), becoming non-significant, as were the differences in BMI (to 0.11), and waist: hip ratio. The differences in skinfold sum and body fat % were however little affected by adjustment and that for sum of skinfolds remained statistically significant; waist circumference remained at the margins of statistical significance.

#### **6.4.3 ADIPOSITY, EXERCISE HABITS AND LEISURE ACTIVITIES**

##### **6.4.3.1 Adiposity and habitual weekly exercise**

Pupil questionnaire information on time spent on exercise per week was completed by 2292 (89%) pupils who had anthropometry of whom 52% were boys and 48% were girls. Table 6.6 shows the exercise levels taken per week, separated into boys and girls and by age. It can be seen that older boys were more active than younger boys. The reverse was true for girls. At age 13 years the percentage of boys and girls in the lowest exercise group (little or no exercise) is similar at around 20%. However in the 15 year old group there were only 10.5 % boys in this group and 25.1% girls. The highest exercise group (daily exercise) saw increases from 14.2% in boys at age 13 to 20.1% in boys at age 15 years. The equivalent figures for girls were a drop from 10.1 to 7.6%, so that by age 15 years boys were almost 3 times more likely than girls to be taking daily physical exercise. The 1-2 times a week exercise group shows an increase in prevalence with increasing age in girls, but this is solely at the expense of the higher exercise frequency groups.

Overall about two thirds of the children exercised only twice a week or less, with girls twice as likely as boys to be in this group.

Mean levels of children's adiposity variables were calculated for each category of exercise (Table 6.9), separately by sex and including adjustments for age, pubertal status, social class, town and ethnicity. Height and weight did not show appreciable relationships with increasing level of exercise in either boys or girls, or evidence of gender interaction ( $p=0.46, 0.10$  respectively Table 6.7). Among the general measures of adiposity, BMI showed no consistent relations with exercise levels. However, sum of skinfolds and body fat percent from bioimpedance both showed significant reductions with increasing level of exercise in both sexes although the effect was stronger in boys. In boys sum of skinfolds fell from 42.58 mm in the low exercise group to 36.46 mm in the daily exercise group

( $p<0.0001$ ). The equivalent figures for girls were a fall from 56.16 mm to 53.01 mm ( $p=0.01$ ). However, although the association between exercise grade and adiposity appeared slightly stronger in boys, there was no strong evidence of gender interaction,  $p=0.17$ . With increasing levels of exercise, % body fat from bioimpedance declined markedly in the boys, from 22.78% in the lowest exercise group to 20.50% in the highest exercise group ( $p<0.0001$ ). The equivalent figures for girls were 31.70% and 31.04 ( $p=0.03$ ). The stronger relationship between exercise grade and % body fat in boys was accompanied by stronger evidence of gender interaction ( $p=0.04$ , table 6.7).

Among the central measures of adiposity, waist circumference decreased with exercise level in boys but not in girls (boys 71.83 to 70.37 cm,  $p=0.02$ ); there was some evidence of gender interaction ( $p = 0.04$ ). Waist: hip ratio decreased with increasing exercise level in boys (0.789 to 0.782) and increased in girls (0.720 to 0.733) However, neither of these associations reached conventional statistical significance levels ( $p=0.07$  and 0.09, respectively), though there was some evidence of gender interaction ( $p = 0.04$ ). Subscapular: triceps ratio showed no relationship with exercise level in either gender.

#### **6.4.3.2 Adiposity and comparative activity level**

The second question on exercise asked children to rank their activity level compared to peers, the levels being much more, more, average, less and much less. 2280 (89%) pupils with anthropometry measures supplied this information. With all children together 40% of children assessed themselves as taking more or much more exercise than average, 40% as average and 20% as less than or much less than average. When looking at boys and girls separately for boys the largest group was the ‘more’ or ‘much more’ than average group at 45% of boys, 40% said average and only 15% less than/ much less than average (Table 6.9). For girls 45% felt they took average amounts of exercise, with 30% above and 25% below. In both sexes about 5% of children felt they were much less active than average, making this the smallest group (54 boys, 67 girls).

Mean variables for adiposity measures were calculated for each level, including adjustment for age, pubertal status, social class, town and ethnicity (Table 6.10).

Weight showed a decrease with more exercise in boys only; mean weight for lowest exercise group 58.81 kg, highest 56.62 kg, ( $p=0.0003$ ) and girls in the higher exercise group were taller than other girls at 1.64 cm, lowest exercise group 1.62cm, ( $p=0.02$ ). Of

the measures of general adiposity body mass index, sum of skinfolds and body fat percentage from bioimpedance all showed reductions with increasing exercise categories in both sexes. Figures for lowest and highest exercise group are as follows: BMI :boys 20.52 and 19.47 kg/m<sup>2</sup> (p<0.0001), girls 21.39 and 21.09 kg/m<sup>2</sup> (p=0.003); sum of skinfolds: boys 43.78 and 33.26 mm (p < 0.0001), girls 59.23 and 51.93 mm (p < 0.0001); body fat % from bioimpedance boys 23.96 and 20.09% (p < 0.0001), girls 33.25% and 30.95% (p<0.0001).

Of the central measures of adiposity, waist and waist: hip ratio in boys showed decreases with increasing comparative exercise level overall, although showing an initial rise from the 'much less' exercise to the 'a bit less' group, then falling again through to the 'much more' exercise group. Figures for waist circumference for these groups are: 72.88, 74.44, 69.91 cm, p<0.0001, and 0.793, 0.801, 0.784, p=0.0004 for waist; hip ratio. In girls waist circumference showed a similar pattern to that in boys, equivalent figures were 68.18, 69.79, 67.90, p=0.005. Waist: hip ratio in girls and subscapular: triceps ratio in both sexes showed no relationship to comparative exercise level group.

## **6.5 DISCUSSION**

### **6.5.1 PRINCIPAL FINDINGS**

In summary parental BMI and child adiposity were closely linked, with slightly stronger associations being seen with father's BMI than mother's. Children whose parents were in the highest group of BMI had particularly high adiposity measures themselves. This included central adiposity as measured by waist circumference and waist: hip ratio (father's BMI only).

There was little evidence of a protective effect of breastfeeding on either central or general adiposity, differences seen between adiposity measures in breast fed (lower adiposity) and other children being greatly reduced by adjustment for factors including social class, maternal BMI, maternal and child smoking, suggesting that there is confounding present. However children who were breast fed had lower sum of skinfolds and (borderline significance) lower waist: hip ratio even after adjustments.

Overall about two thirds of the children exercised only twice a week or less, with girls twice as likely as boys to be in this group. Girls' exercise frequency became lower between the ages of 13 and 15 whilst boys' became higher. Increasing level of exercise was associated with lower body fat from bioimpedance and sum of skinfolds in both sexes, and

with lower waist circumference in boys. Children's perception of themselves as much more or more active than average was accurate in selecting those with the lowest levels of adiposity. Those who felt they were a bit less active than average were more adipose than the much less active group.

## **6.5.2 PARENTAL ADIPOSITY**

### **6.5.2.1 Strengths and weaknesses of the study**

Response rates in the parental questionnaire were high, thanks to reminders from the study team and cooperation of schools; responses in the pupil questionnaire were even higher, as these were completed with support. These were good features of the study design. Potential weaknesses for the parental data are that self- or partner-reported height and weight are obviously open to inaccuracy. Many previous studies have examined the validity of self-reported weight and height in adults, finding that although there tends to be some inaccuracy (height tends to be overestimated and weight underestimated) ranking of heights and weights are reasonably accurate and sufficient for population-based studies (Spencer et al 2002). Analysing parental BMI in fifths of the distribution reduces the effect of inaccuracies due to self-reporting. It would have been additionally interesting to have had a measure of parental waist circumference, to get an idea of parental central obesity, as distinct from overall body size, which has some genetic determinant. However we had a range of measures of both general and central adiposity in children and were able to demonstrate associations with parental BMI for nearly all of them, not just child's BMI.

### **6.5.2.2 Relation of findings to other studies**

Both parent's BMI was found to be influential on child's adiposity. The associations in our study were generally stronger with fifths of paternal BMI, with the exception of weight and BMI in girls. Our findings therefore are not inconsistent with those of Duran-Tauleria (Duran-Tauleria et al 1995) who found a stronger effect of father's BMI than mother's (as continuous variables) when looking at child's weight-for height, and sum of skinfolds measurements but this study used combined genders so the stronger relationship seen for girls' BMI with mothers can not be compared with their results. The strong relationship between BMI in mothers and daughters seen in our study may reflect the influence of maternal overweight and obesity on daughter's adiposity, as over half the mothers in our study were overweight or obese. Strong associations between maternal overweight and obesity (from BMI) and daughters' adiposity were seen in the Health Survey for England

2006. As twin studies suggest that 50% of variance in BMI results from genetic factors (Allison et al, 1996), it may be that paternal genes have more influence on body size, coupled to maternal influence being traditionally greater in terms of food in the home; setting patterns for food types and consumption patterns. Both parents may be a role model in terms of exercise and diet. Exposure in utero to obesogenic factors may influence female fetuses more than male, leading to closer linkage to maternal BMI for girls.

It is difficult from these findings to comment on the relative importance of father or mother's BMI, it is clear that both have an independent and significant effect on child's adiposity. I have not performed analyses to examine the effect of one as opposed to both parents obese to see the comparative effect on child adiposity; other studies have found widely varying results in terms of absolute odds ratios (Reilly et al 2005 OR 11.25, Duran-Tauleria et al 1995 3.48, Health Survey for England 2006 girls OR 3.80, boys 1.56-no change), but usually about double the risk of having one parent obese. The different absolute odds ratios may reflect confounding; Reilly and Duran-Tauleria adjusted for maternal educational level, adjustment in the Health survey for England was not specified; a raft of socioeconomic factors may cause confounding.

It could be argued that the highest BMI group contains a few families with morbid obesity whose data are distorting the results in terms of the marked increase from fifths 4 to 5. However the stepwise increase in adiposity measures across the other four BMI groups provides ample evidence of a consistent rise in child's adiposity with increasing parental BMI. This adiposity also includes central adiposity as denoted by waist circumference, seen in adults as associated with higher cardiovascular risk. The relationship of adiposity measures with cardiovascular risk factors in study children will be explored further in chapter 7.

### **6.5.3 INFANT FEEDING**

#### **6.5.3.1 Strengths and weaknesses of the study**

The fact that infant feeding method was ascertained by parental recall after more than ten years is a potential weakness. However a review of the evidence (Li R et al, 2005) found that, even after a number of decades, both the validity and reliability of recalled data regarding the initiation of breastfeeding is good (82% sensitivity and 93% specificity). There is a concern over the validity of recalled data on the duration of breast feeding, but this is only important when looking for a dose response relationship between adiposity means and length of breast feeding. The evidence of baseline differences in height between

those subjects on whom full data on potential confounders were available (who were included in the analyses) and those who were excluded may be due to social class differences. As adiposity variables did not vary between the groups it is difficult to conclude that the exclusion of these subjects would affect the results seen.

#### **6.5.3.2 Relation of findings to other studies**

In this population of British adolescents we found little evidence showing a protective effect of breastfeeding on either central or general adiposity. Although in analyses adjusted for age and sex, all mean adiposity measures were lower among individuals who had been exclusively breast fed in the first three months of life (sum of skinfolds, fat mass from bioimpedance and waist hip ratio significantly so) additional adjustment for social class, maternal BMI, maternal and child smoking greatly reduced almost all the differences. This suggests that the relationship between adiposity and breast feeding is substantially confounded, by other behavioural and societal factors including low maternal social class and maternal obesity. This is consistent with the findings of other researchers, both in individual studies (Toschke et al, 2007) and in systematic reviews (Owen et al, 2005a and 2005b).

The consistency of our findings with those of others is difficult to assess as there are very few studies looking at the association between breast feeding and adiposity measures other than BMI. There are three published reports where fat mass has been derived from Dual-energy X-ray absorptiometry (DXA) (Burdette et al, 2006, Tulldahl et al, 1999, Toschke et al, 2007). Our results are consistent with Burdette et al, who found a small, but statistically insignificant reduction in fat mass in 5 year olds who had ever been breastfed verses never breastfed. Tulldahl et al observed an inverse association in adolescents, but failed to adjust for known potential confounders. Toschke found little evidence of association of breastfeeding with BMI but did find decreases in trunk and total fat mass with breast feeding duration, although adjustment for confounders attenuated the association. In four recent meta-analyses (Owen et al, 2005a, Owen et al, 2005b, Arenz et al, 2004, Harder et al, 2005) one (Harder) found a dose-dependent protective effect in an unadjusted model. The others found only a very small protective effect. In all four the presence of publication bias, residual or uncontrolled confounding could not be ruled out. A direct comparison between the results of this study and those of the meta-analyses is difficult as the

individual studies included in the meta-analyses, almost exclusively, defined adiposity using BMI, a proxy measure of overweight rather than a direct one.

#### **6.5.4 PHYSICAL ACTIVITY**

##### **6.5.4.1 Strengths and weaknesses of the study**

The exercise questionnaires used provided very simple categorizations of leisure time physical activity level. The validity of simple physical activity questionnaires for assessment of physical activity has been previously demonstrated in children and adolescents (Wong et al 2006). The validity of these particular questions was demonstrated in the study in which they were initially used, in relation to adiposity and blood pressure (Strazzullo et al 1988); their validity in the present study was confirmed by their association with resting heart rate (lower in the high physical activity categories) (Ussher et al 2007). Physical activity self-reporting is clearly open to recall bias, which may have operated to different degrees in boys and girls. This may explain differences seen between sexes. Children who are overweight or extremely fit may be more aware of their exercise patterns than the average and may exaggerate the amount of activity they undertake, thereby reducing the ability of the study to detect exercise-adiposity associations. In this study exercise-adiposity relations were being examined cross-sectionally, therefore it is difficult to say whether lower activity levels in overweight children reflect a causal effect of low activity on adiposity, or whether, having become overweight, the children then took less exercise. It is also possible that overweight children may have already had exercise ‘prescribed’ by health professionals, leading to reverse causality, in which children who are overweight may have been taking more exercise as a result.

##### **6.5.4.2 Relation of findings to other studies**

The finding that older girls were less active than younger girls between the ages of 13 and 15 whilst boys showed the opposite pattern is roughly compatible with findings in similar aged children in a cross-sectional study in Northern Ireland (Riddoch et al 1991). In their study general exercise was lower at ages over 13 in girls and 14 in boys; vigorous exercise also became lower in girls but remained steadier in boys to age 18. In both general and vigorous exercise groups the level of boys’ activity was over twice that of girls which is similar to our findings.

Only 20.3% of boys and 7.8% of girls in our study said that they did some physical activity in free time daily; a low number in light of the Government target of one hour of moderate

to vigorous physical activity daily. A more recent study by Riddoch using accelerometers to include both duration and intensity of physical activity found that only 5.1% of boys and 0.4% of girls met this target (Riddoch et al, 2007). This contrasts with the Health Survey for England 2006 findings that 70% of boys and 59% of girls met the target; in this survey all activity was assumed to be of moderate intensity, although the definition of activity specifically included walking which was the most common type of activity for girls. The differences between our findings and Health Survey for England 2006 may represent differences in wording of the questions; difficulties in comparing data on physical activity is well recognised; confounding with socio-economic factors can also be present (Must, Tybor 2005).

In terms of the associations seen between habitual exercise and body size measures in our study the results for BMI and skinfolds are comparable with findings in children aged 9-15 in the European Youth Heart Study (from Estonia, Denmark and Portugal). In this study physical activity data was collected by accelerometer. Lower BMI (genders combined) was seen in children taking higher levels of physical activity, but this trend was not significant, whereas the trend of lower sum of skinfolds was highly significant, similar to our findings in boys and girls (Andersen et al 2006). A significant association of lower waist circumference with higher activity levels was also found in this study. In the Health Surveys for England 2002 and 2006 no significant differences were seen in mean BMI between high, medium and low exercise groups; this exercise data was collected by questionnaire. In the 2002 Health Survey for England this lack of association between obesity/overweight and exercise level is suggested as being due to the lower sensitivity of questionnaire data collection when compared to accelerometer data; however our results, also using questionnaire data, would suggest that the inconclusive findings in the Health Survey for England are possibly due to the misplaced reliance on BMI to reflect adiposity differences in different exercise groups.

## **6.6 CONCLUSIONS AND IMPLICATIONS FOR AETIOLOGY AND PREVENTION**

### **6.6.1 PARENTAL ADIPOSITY**

The study findings confirm that higher parental BMI is associated with higher adiposity in children, probably partly due to shared diet, lifestyle habits and circumstances, in keeping with recent evidence about life-course influences (Power et al, 2007), and partly due to genetic factors. A family centered approach to obesity seems logical, where possible, as

only 20% of fathers and 40% of mothers in our study population had BMIs that were in the ideal range (<25). Gradually a child's view of what is a 'normal' body size must be getting ever larger! Parents need to be aware of the health risk to their child from their behaviour and patterning. Particularly this may be the case with exercise as children's activity levels were so low overall, especially in girls. Schools could do more exercise with pupils but this does not compensate for the exercise built into every day life which would benefit both parent and child e.g. walking to school.

### **6.6.2 BREAST FEEDING**

The implications of this study are that breast feeding confers only a very small protective effect against adolescent adiposity. This is consistent with evidence from several other studies and systematic reviews. On the basis of this evidence, breast feeding cannot be recommended as an important component of strategies for the prevention of childhood obesity (or adult obesity) in our society. However, the case for the benefits of breast feeding is already well established in a number of other public health areas such as early immune protection, neural and psychosocial development (Makrides et al, 1995, Fergusson, Woodward 1999). Breast feeding may also be associated with lower mean blood cholesterol levels (Owen et al 2002) and protect against type 2 diabetes (Owen et al 2006).

### **6.6.3 PHYSICAL EXERCISE**

Our findings in this chapter show that lower levels of physical activity in adolescents are associated with greater adiposity. In view of the increasing health burden of obesity strategies to increase the physical activity levels of adolescents should be a priority. Dollman (Dollman et al, 2005) raised the question of barriers to activity presented by the 'activity toxic' environment which increasingly those in developed countries inhabit; these included lack of outdoor play space, increasing 'screen-based' entertainment and parental concerns about safety. Active transport (walking, bicycle) to school become less pleasant and more risky with increasingly busy roads. Additional exercise within the school setting may be beneficial. However a recent systematic review of interventions to promote physical activity for teenagers found most evidence for effectiveness if interventions involve the wider family or community as well as taking place at school (Van Sluijs et al, 2007). Effects from increased physical activity in children will often not include reductions in BMI in the adolescent population; an additional measure of general adiposity (body fat

percent from bioimpedance, skinfold measurement) or waist circumference may be more useful in assessing interventions at a population level.

**Table 6.1 Parental height, weight and BMI**

	<b>Mean (Std Dev)</b>	<b>Median</b>	<b>Range</b>
<b>Mother's height m</b>	1.62 (0.07)	1.62	1.22-1.93
<b>Father's height m</b>	1.77 (0.07)	1.78	1.52-2.13
<b>Mother's weight kg</b>	74.1 (13.6)	72.6	42.2-138.0
<b>Father's weight kg</b>	91.4 (14.2)	90.3	50.8-163.0
<b>Mother's BMI</b>	28.0 (4.9)	27.1	16.0-49.8
<b>Father's BMI</b>	29.4 (4.2)	29.1	16.8-50.1

**Table 6.2 Children's adiposity and body size variables according to fifths of maternal BMI**

Variable	Fifths of mother's BMI					Increase in variable per fifth (95% CI)	p value
	1 16.0-24.1	2 24.1-26.1	3 26.1-28.2	4 28.2-31.4	5 31.5-49.8		
<b>BOYS</b>							
<b>Height(cm)+</b>	168.7	168.7	169.3	169.7	170.3	0	0.10
<b>Weight(kg)*</b>	56.1	57.8	57.6	58.8	61.2	0.56%(0.32 to 0.80)	<0.0001
<b>BMI (kg/m<sup>2</sup>)*</b>	19.8	20.3	20.2	20.6	21.2	0.47%(0.27 to 0.67)	<0.0001
<b>Sum of skinfolds (mm)*^</b>	37.1	39.9	39.9	40.4	44.2	1.08%(0.50 to 1.66)	0.0003
<b>Body fat % from bioimpedance +</b>	20.7	22.1	21.8	21.9	23.1	0.15(0.07 to 0.23)	0.0001
<b>Waist*^ circumference</b>	70.1	71.2	71.0	71.5	73.3	0.29%(0.14 to 0.44)	<0.0001
<b>Waist: hip ratio*^</b>	0.786	0.787	0.786	0.783	0.786	0	0.94
<b>STR</b>	0.813	0.800	0.786	0.787	0.828	0.001(0.004 to -0.002)	0.44
<b>GIRLS</b>							
<b>Height(cm)+</b>	162.6	162.7	161.7	162.5	162.5	0(0)	0.87
<b>Weight(kg)*</b>	52.9	54.2	54.2	57.3	58.6	0.79%(0.55 to 1.03)	<0.0001
<b>BMI (kg/m<sup>2</sup>)*</b>	20.0	20.5	20.8	21.7	22.2	0.79%(0.57 to 1.01)	<0.0001
<b>Sum of skinfolds (mm)*^</b>	52.8	54.8	56.2	60.1	61.7	1.11%(0.60 to 1.62)	<0.0001
<b>Body fat % from bioimpedance +</b>	31.2	31.2	31.8	32.1	33.1	0.15(0.08 to 0.22)	<0.0001
<b>Waist*^ circumference</b>	65.9	66.6	66.7	68.5	69.8	0.44%(0.29 to 0.59)	<0.0001
<b>Waist: hip ratio*^</b>	0.720	0.721	0.724	0.726	0.730	0.08%(0.17 to -0.01)	0.11
<b>STR</b>	0.741	0.713	0.717	0.723	0.773	0.003(0.000 to 0.006)	0.03

+ Mean values are presented with standard errors. Increases in adiposity measures per fifth are presented in absolute values.

\*Variables log transformed for analysis. Geometric means are presented. Increases in adiposity measures per year are presented as percentage values.

Adjusted for age, pubertal status, town, social class, ethnic group and **father's BMI** throughout, ^ Adjusted for observer in addition

**Table 6. 3 Children's adiposity and body size variables according to fifths of paternal BMI**

Variable	Fifths of father's BMI					Increase in variable per fifth (95% CI)	p value
	1 16.8-26.0	2 26.0-28.2	3 28.2-30.0	4 30.0-32.5	5 32.5-51.9		
<b>BOYS</b>							
<b>Height(cm)+</b>	170.5	168.7	168.9	169.5	169.1	0	0.19
<b>Weight(kg)*</b>	57.6	56.9	57.8	58.1	60.9	0.54%(0.26 to 0.82)	0.0002
<b>BMI (kg/m<sup>2</sup>)*</b>	19.9	20.0	20.3	20.3	21.4	0.62%(0.39 to 0.85)	<0.0001
<b>Sum of skinfolds (mm)*^</b>	37.6	38.8	40.3	39.8	44.7	1.33%(0.64 to 2.02)	0.0001
<b>Body fat % from bioimpedance +</b>	21.2	21.5	22.1	21.6	23.1	0.16(0.07 to 0.25)	0.0006
<b>Waist*^ circ</b>	70.8	70.7	71.1	70.9	73.3	0.32%(0.15 to 0.49)	0.0003
<b>WHR*^</b>	0.783	0.784	0.784	0.783	0.794	0.13%(0.04 to 0.22)	0.005
<b>STR</b>	0.840	0.796	0.804	0.793	0.782	-0.004(-0.001 to -0.007)	0.02
<b>GIRLS</b>							
<b>Height(cm)+</b>	162.3	161.9	162.4	162.9	162.4	0	0.39
<b>Weight(kg)*</b>	53.7	54.5	54.9	56.3	57.4	0.69%(0.38 to 1.00)	<0.0001
<b>BMI (kg/m<sup>2</sup>)*</b>	20.4	20.8	20.9	21.3	21.8	0.63%(0.36 to 0.90)	<0.0001
<b>Sum of skinfolds (mm)*^</b>	52.8	56.9	57.8	57.5	60.2	1.26%(0.63 to 1.89)	<0.0001
<b>Body fat % from bioimpedance +</b>	30.8	31.9	31.8	32.4	32.4	0.16(0.07 to 0.25)	0.0004
<b>Waist*^ circumference</b>	66.1	67.0	66.9	68.1	69.2	0.43%(0.23 to 0.63)	<0.0001
<b>Waist: hip ratio*^</b>	0.717	0.722	0.723	0.730	0.729	0.14%(0.02 to 0.26)	0.02
<b>STR</b>	0.727	0.713	0.732	0.744	0.749	0.003(-0.001 to 0.006)	0.15

+ Mean values are presented with standard errors. Increases in adiposity measures per fifth are presented in absolute values.

\*Variables log transformed for analysis. Geometric means are presented. Increases in adiposity measures per year are presented as percentage values.

Adjusted for age, pubertal status, town, social class, ethnic group and mother's BMI throughout

^ Adjusted for observer in addition

**Table 6.4 Mean (SE) adiposity and body size measures by infant feeding categories**

	Mean (SE)									Test for Breast Fed vs Formula Fed		
	Breast Fed			Mixed Fed			Formula Fed					
	male	female	all	male	female	all	male	female	all	male	female	all
<b>n (Maximal)</b>	505	476	981	303	211	514	485	463	948			
<b>Height (cm)</b>	169.29 (0.35)	162.82 (0.27)	166.25 (0.23)	169.48 (0.46)	162.14 (0.40)	166.08 (0.32)	168.50 (0.36)	161.27 (0.27)	165.19 (0.23)	0.116	<0.001	0.001
<b>Weight (kg)</b>	59.34 (0.54)	55.86 (0.48)	57.70 (0.37)	58.83 (0.69)	55.93 (0.73)	57.51 (0.51)	59.08 (0.55)	56.64 (0.49)	57.96 (0.37)	0.731	0.258	0.622
<b>BMI (kg/m<sup>2</sup>)</b>	20.58 (0.154)	21.03 (0.17)	20.79 (0.11)	20.34 (0.19)	21.28 (0.25)	20.78 (0.16)	20.69 (0.16)	21.67 (0.17)	21.16 (0.12)	0.625	0.007	0.022
<b>Sum of skinfolds<sup>1</sup></b>	39.15 (1.02)	56.26 (1.02)	46.44 (1.14)	38.07 (1.03)	57.99 (1.03)	46.27 (1.02)	41.28 (1.02)	58.08 (1.02)	48.48 (1.01)	0.061	0.179	0.021
<b>Fat mass % (bioimpedance)<sup>1</sup></b>	20.98 (1.01)	31.04(1.01)	25.23 (1.01)	20.61 (1.02)	31.61(1.01)	25.17 (1.01)	21.34 (1.01)	32.19(1.01)	25.90 (1.01)	0.331	0.001	0.013
<b>Waist circumference(cm)</b>	71.81 (0.38)	67.47 (0.38)	69.76 (0.27)	71.29 (0.50)	68.38 (0.57)	69.89 (0.37)	71.91 (0.39)	68.81 (0.38)	70.47 (0.27)	0.841	0.012	0.063
<b>Waist:hip ratio (%)</b>	78.74 (0.22)	72.26 (0.23)	75.67 (0.16)	78.25 (0.28)	72.89 (0.35)	75.69 (0.22)	78.98 (0.22)	73.27 (0.23)	76.29 (0.16)	0.447	0.002	0.006
<b>Subscapular :triceps skinfold ratio (%)</b>	80.29 (1.02)	72.51 (1.41)	76.60 (0.70)	80.58 (1.32)	72.22 (1.41)	76.77 (0.97)	81.56 (1.03)	75.03 (0.96)	78.46 (0.71)	0.381	0.060	0.063

All analyses adjusted for age, ethnicity, town observer and (in analyses based on all subjects) sex.

<sup>1</sup>Geometric mean and SE

**Table 6.5 Differences in adiposity and body size measures (Formula Fed minus Breast Fed) (95% C.I.) *p***

	basic model <sup>1</sup>			parental social class <sup>2</sup>			maternal smoking <sup>2</sup>			maternal BMI <sup>2</sup>			child's smoking <sup>2</sup>			model including all factors <sup>3</sup>			final model - with paternal smoking included					
Mean Difference	Coef	95% CI	P value	Coef	95% CI	P value	Coef	95% CI	P value	Coef	95% CI	P value	Coef	95% CI	P value	Coef	95% CI	P value	n	Coef	95% CI	P value		
Height (cm)	-0.88	-1.79	0.03	0.06	-0.56	-1.51	0.39	0.25	-0.62	-1.55	0.31	0.19	-1.00	-1.92	-0.09	0.03	-0.87	-1.78	0.04	0.06	-0.46	-1.43	0.50	0.35
Weight (kg)	0.63	-0.80	2.07	0.39	0.61	-0.89	2.11	0.43	0.75	-0.72	2.21	0.32	-0.05	-1.45	1.35	0.94	0.66	-0.77	2.10	0.36	0.11	-1.37	1.60	0.88
BMI (kg / m <sup>2</sup> )	0.44	0.01	0.88	0.04	0.35	-0.10	0.81	0.13	0.41	-0.03	0.86	0.07	0.23	-0.20	0.65	0.29	0.45	0.02	0.88	0.04	0.15	-0.30	0.59	0.52
Sum of skinfolds <sup>4</sup>	6.81%	1.02	1.12	<0.00	7.05%	1.02	1.13	0.01	7.27%	1.02	1.13	0.01	4.86%	1.00	1.10	0.01	6.87%	1.02	1.12	0.01	5.62%	1.00	1.11	0.04
Fat mass % (bioimpedance) <sup>4</sup>	3.60%	1.01	1.07	0.01	3.24%	1.00	1.06	0.01	4.00%	1.01	1.07	0.01	2.62%	1.00	1.06	0.01	3.63%	1.01	1.07	0.01	2.71%	0.99	1.06	0.80
Waist circumference (cm)	0.95	-0.06	1.97	0.07	0.77	-0.29	1.83	0.16	0.90	-0.13	1.94	0.09	0.53	-0.47	1.52	0.30	0.97	-0.04	1.99	0.06	0.38	-0.68	1.43	0.48
Waist:hip ratio (%)	0.85	0.27	1.44	<0.00	0.66	0.05	1.27	0.03	0.77	0.17	1.37	0.01	0.80	0.21	1.39	0.01	0.86	0.27	1.44	<0.00	0.58	0.04	1.20	0.07
Subscapular :triceps skinfold ratio (%)	1.26	-1.37	3.90	0.35	0.93	-1.83	3.69	0.51	0.86	-1.83	3.55	0.53	1.15	-1.50	3.81	0.39	1.25	-1.39	3.89	0.35	0.50	-2.31	3.30	0.73
	1151	0.48	-2.50	3.42	0.75																			

<sup>1</sup>Basic model adjusted for age sex town ethnicity observer

<sup>2</sup>Basic model with one additional adjustment as specified

<sup>3</sup>Basic model additionally adjusted for parental social class, maternal smoking, maternal BMI and child's own smoking habit

<sup>4</sup>Logged transfromed variable therefore differences represent percentage differences between the two groups

**Table 6. 6 Differing levels of exercise in leisure time by gender and age**

<b>Exercise taken</b>	<b>Boys (together and by age), % (no.)</b>				<b>Girls together and by age, % (no.)</b>			
	<b>ALL</b>	<b>13</b>	<b>14</b>	<b>15</b>	<b>ALL</b>	<b>13</b>	<b>14</b>	<b>15</b>
<b>Little exercise</b>	12.7 (170)	21.7 (26)	12.8 (88)	10.5 (56)	25.4 (312)	22.7 (27)	26.1 (160)	25.1 (125)
<b>1-2 times/week</b>	30.1 (404)	35.0 (42)	28.3 (195)	31.3 (167)	44.1 (542)	42.0 (50)	43.1 (264)	45.8 (228)
<b>4-6 times/week</b>	37.0 (496)	29.2 (35)	37.2 (256)	38.1 (203)	22.7 (279)	25.2 (30)	23.2 (142)	21.5 (107)
<b>7+ times/week</b>	20.3 (272)	14.2 (17)	21.8 (150)	20.1 (107)	7.8 (96)	10.1 (12)	7.5 (46)	7.6 (38)

13 girls aged 12, 8 boys aged 12, included with 13 yr olds

36 girls aged 16, 47 boys aged 16, included with 15 yr olds

**Table 6. 7 Test for gender interaction with exercise level, adjusted for age**

<b>Variable</b>	<b>P value</b>
<b>Height</b>	0.46
<b>Weight</b>	0.10
<b>BMI</b>	0.12
<b>Sum of Skinfolds</b>	0.17
<b>Body fat %</b>	0.04
<b>Waist circ</b>	0.04
<b>WHR</b>	0.04
<b>STR</b>	0.62

**Table 6. 8 Adiposity and body size measures by exercise group**

Variable	Exercise taken				Change in variable per exercise category (95% CI)	p value
	Little exercise	1-2 times/week	4-6 times/week	7+ times/week		
<b>BOYS</b>						
<b>Height(cm)+</b>	1.70 (0.01)	1.69 (0.0)	1.69 (0.0)	1.69 (0.0)	0.00 (0)	0.67
<b>Weight(kg)*</b>	58.50	58.32	57.70	57.15	-0.86% (0.28 to -2.0)	0.14
<b>BMI (kg/m<sup>2</sup>)*</b>	20.36	20.54	20.19	20.08	-0.74% (0.19 to -1.67)	0.12
<b>Sum of skinfolds (mm)*^</b>	42.58	41.50	39.35	36.46	-5.27% (-2.76 to -7.78)	<0.0001
<b>Body fat % from bioimpedance +</b>	22.78(0.49)	22.71(0.30)	21.28(0.27)	20.50(0.37)	-0.92 (-0.57 to -1.27)	<0.0001
<b>Waist*^ circumference</b>	71.83	71.85	70.92	70.37	-0.83%(-0.16 to-1.50)	0.02
<b>Waist: hip ratio*^</b>	0.789	0.787	0.784	0.782	-0.34%(0.03 to -0.71)	0.07
<b>STR</b>	0.833 (0.019)	0.813 (0.012)	0.794 (0.010)	0.811 (0.014)	-0.008(0.006 to -0.022)	0.26
<b>GIRLS</b>						
<b>Height(cm)+</b>	1.62 (0.00)	1.62 (0.00)	1.63 (0.00)	1.62 (0.01)	0.00 (0)	0.27
<b>Weight(kg)*</b>	53.85	56.05	54.92	55.60	0.72% (-0.51 to 1.95)	0.25
<b>BMI (kg/m<sup>2</sup>)*</b>	20.57	21.36	20.76	21.23	0.44% (-0.66 to 1.54)	0.42
<b>Sum of skinfolds (mm)*^</b>	56.16	59.63	53.61	53.04	-2.95% (-0.61 to -5.29)	0.01
<b>Body fat % from bioimpedance +</b>	31.70(0.30)	32.62(0.22)	31.18(0.30)	31.04(0.53)	-0.36(-0.03 to-0.69)	0.03
<b>Waist*^ circumference</b>	66.61	68.33	67.03	68.16	0.33% (1.11 to -0.45)	0.39
<b>Waist: hip ratio*^</b>	0.720	0.727	0.724	0.733	0.38% (0.81 to -0.03)	0.09
<b>STR</b>	0.726 (0.013)	0.747 (0.009)	0.727 (0.012)	0.701 (0.022)	-0.007 (0.007 to -0.014)	0.34

Mean values are presented with standard errors. Increases in adiposity measures per fifth are presented in absolute values.

\*Variables log transformed for analysis. Geometric means are presented. Increases in adiposity measures per year are presented as percentage values.

+Adjusted for age, pubertal status, town, social class, ethnic group throughout.

^ Adjusted for observer in addition

**Table 6.9 Self-assessed activity level compared to peers; distribution of responses**

	Comparative activity level				
	Much less active	A bit less active	About average	A bit more active	Much more active
<b>Children (2615=100%)</b>	121 (4.6%)	384 (14.7%)	1109 (42.4%)	686 (26.2%)	315 (12.0%)
<b>Boys (1395=100%)</b>	54 (3.9%)	152 (10.9%)	556 (39.9%)	426 (30.5%)	207 (14.8%)
<b>Girls (1220=100%)</b>	67 (5.5%)	232 (19.0%)	553 (45.3%)	260 (21.3%)	108 (8.9%)

**Table 6.10 Activity level compared with peer group and adiposity/ body size measures**

Variable	Comparative activity level					Change in variable per activity category (95% CI)	p value
	Much less active	A bit less active	About average	A bit more active	Much more active		
BOYS							
<b>Height(cm)+</b>	1.70(0.01)	1.69(0.01)	1.69(0.00)	1.69(0.00)	1.70(0.01)	0.00(0)	0.88
<b>Weight(kg)*</b>	58.81	60.60	58.26	56.66	56.62	-1.88%(-0.87 to -2.89)	0.0003
<b>BMI (kg/m<sup>2</sup>)*</b>	20.52	21.34	20.41	19.96	19.77	-1.91%(-1.09 to -2.73)	<0.0001
<b>Sum skinfolds (mm)*^</b>	43.78	47.75	42.25	36.96	33.26	-10.00%(-7.85 to -12.15)	<0.0001
<b>Body fat % +</b>	23.96	24.28	22.39	20.51	20.09	-1.33(-1.02 to -1.64)	<0.0001
<b>Waist circ*^</b>	72.88	74.44	71.59	69.88	69.91	-1.77%(-1.18 to -2.36)	<0.0001
<b>WHR*^</b>	0.793	0.801	0.786	0.779	0.784	-0.60%(-0.27 to -0.93)	0.0004
<b>STR</b>	0.776(0.033)	0.865(0.019)	0.797(0.010)	0.794(0.011)	0.827(0.016)	-0.002(0.010 to -0.014)	0.72
GIRLS							
<b>Height(cm)+</b>	1.61(0.01)	1.62(0.00)	1.62(0.00)	1.63 (0.00)	1.64(0.01)	0.004(0-0.008)	0.02
<b>Weight(kg)*</b>	55.71	57.75	54.38	54.48	56.38	-0.89% (0.15 to -1.93)	0.1
<b>BMI (kg/m<sup>2</sup>)*</b>	21.39	21.94	20.81	20.59	21.09	-1.42%(-0.50 to -1.91)	0.003
<b>Sum skinfolds (mm)*^</b>	59.23	63.36	57.02	53.32	51.93	-5.57%(-3.61 to -7.53)	<0.0001
<b>Body fat % +</b>	33.25(0.63)	33.11(0.33)	31.92(0.21)	31.06(0.31)	30.95(0.48)	-0.76(-0.47 to -1.05)	<0.0001
<b>Waist circ *^</b>	68.18	69.79	67.07	66.66	67.90	-0.94%(-0.28 to -1.60)	0.005
<b>WHR*^</b>	0.724	0.733	0.723	0.723	0.725	-0.23(0.14 to -0.60)	0.23
<b>STR</b>	0.782(0.026)	0.747(0.014)	0.724(0.009)	0.724(0.013)	0.736(0.020)	-0.009(0.003 to -0.021)	0.13

Mean values are presented with standard errors. Increases in adiposity measures per fifth are presented in absolute values.

\*Variables log transformed for analysis. Geometric means are presented. Increases in adiposity measures per year are presented as percentage values.

+Adjusted for age, pubertal status, town, social class, ethnic group throughout. ^ Adjusted for observer in addition

## CHAPTER 7: EXPLORING RELATIONS BETWEEN DIFFERENT MARKERS OF ADIPOSITY AND THE CARDIOVASCULAR RISK PROFILE

### **7.0 SUMMARY POINTS**

- This chapter sets out to examine the relations between measures of adiposity (general and central) and blood pressure, fasting insulin and fasting glucose levels.
- Analyses of adiposity and blood pressure are based on 2643 British children aged 12.7 to 16.4 years (1235 girls and 1408 boys). Analyses of adiposity, insulin and glucose are based on a subgroup of these children; 839 boys and 685 girls.
- Height, weight and all general measures of adiposity were positively related to SBP in both sexes.
- SBP showed the largest increases with increasing weight, height, BMI and waist circumference in boys and weight, BMI, sum of skinfolds and waist circumference in girls.
- For fasting insulin there was a positive relationship with height (boys only) and weight. All adiposity variables except STR in boys showed strong positive relations with fasting insulin.
- The largest changes in insulin were seen with BMI, waist circumference and sum of skinfolds in boys and with body fat % from bioimpedance, BMI and waist circumference in girls.
- Fasting glucose in girls showed a positive relationship with all adiposity variables but not with height and weight, the strongest, most statistically significant changes were with STR, WHR and BMI. In boys the only near-significant relationship for fasting glucose was with sum of skinfolds; in a positive direction.
- The highest mean fasting glucose levels were seen in the highest fifth of waist circumference in both genders.

### **7.1 INTRODUCTION AND OBJECTIVES**

In adults, greater adiposity and obesity have adverse effects on cardiovascular risk and on the cardiovascular risk profile (particularly higher levels of blood pressure, total and LDL cholesterol). Greater adiposity also causes an increase in the risk of type 2 diabetes and an increased tendency to insulin resistance. Increases in cardiovascular disease, type 2

diabetes and insulin resistance are particularly associated with central obesity, which reflects greater visceral fat deposition.

In children and adolescents, it is well recognized that greater adiposity and obesity, as in adults, are related to higher blood pressure and cholesterol levels and to greater degrees of insulin resistance (Srinivasan et al, 2002, Weiss et al, 2004); such associations have been demonstrated both cross-sectionally and longitudinally (Bao et al, 1994, Sinaiko et al, 1998). In addition, the recent appearance of type 2 diabetes in childhood and adolescence is almost all occurring in obese subjects. There is also evidence that adiposity and obesity in children and adults are related directly to change in the structure and function of the arterial circulation, including early atherosclerosis (McGill et al, 2002), impaired endothelial function (Watts et al, 2004 a and b), increased vascular resistance (Rocchini et al, 1992) and reduced arterial distensibility (Whincup et al, 2005). However, few studies have been able to examine the relationships of different measures of adiposity/obesity (including both general and central measures) to blood pressure, insulin resistance and glucose homeostasis among young people. Therefore, the aim of this chapter is to examine the contribution of general and central adiposity to the cardiovascular risk profile in children. I will explore whether body mass index is the best measure of childhood adiposity in terms of the strengths of its relations with cardiovascular risk markers, or whether other markers of adiposity are more strongly related. I will investigate the relationships of various measures of general adiposity (body mass index, sum of skinfolds, body fat from bioimpedance), and central adiposity (waist: hip ratio, waist circumference, subscapular: triceps ratio) to blood pressure, fasting insulin (a marker of insulin resistance) and fasting glucose. I would hypothesize that fasting glucose, being under strong homeostatic control, should not show a relationship with adiposity. I will investigate if this is the case in this group of children or if there is any evidence that greater degrees of adiposity are related to higher glucose levels, implying loss of homeostatic control and a degree of progression towards type II diabetes.

## **7.2 SUBJECTS AND METHODS**

The results presented are based on the Ten Towns Heart Health Study, carried out in England and Wales in 1998-99. All participants were invited to have measurements of body build (described in detail in Chapter 3, section 3.3.1) and blood pressure made. Blood pressure was measured in the right arm after a short period of rest, with the subject seated.

Two measurements were made a minute apart, using the Dinamap 1846SX oscillometric blood pressure recorder (Whincup et al, 1992c), with a cuff size in accordance with American Heart Association recommendations. A subset of children were asked to provide a blood sample, taken after an overnight fast, following the application of a local anaesthetic skin cream (EMLA or Ametop). All children were offered breakfast after the procedure. Blood samples were centrifuged, separated and frozen at -20 C within eight hours of collection. Blood glucose was measured in a fluoride oxalate sample, using the glucose oxidase method. Serum insulin concentration was measured by an ELISA (enzyme linked immunosorbent assay) method which does not cross react with proinsulin. Ethnic group was assessed on the basis of the child's appearance into five main groupings (white, Afro-Caribbean, Asian, Oriental, other). Detailed information on subjects and methods and all measuring techniques are included in Chapter 3.

### **7.2.1 STATISTICAL METHODS**

All data were analysed using the SAS system statistical software package (SAS Institute, North Carolina, USA). Analyses were carried out separately for males and females. In the main analyses, blood pressure, insulin and glucose were treated as the outcome variables. Insulin values were highly skewed to the right and were log transformed. The mean values of each outcome variable, adjusted for age, gender, town, time of day (minutes), ethnicity and observer (blood pressure only) were determined for each fifth of the adiposity measures using standard linear modelling approaches (PROC GLM, LSMEANS option). Age was fitted as a continuous variable, town as a categorical variable and observer (where used) as a dummy variable to minimise the effect of unequal distribution of subjects in this group.

### **7.3 RESULTS**

Following invitation, 2645 subjects (1410 boys and 1235 girls) took part in the study (66% response rate). The subgroup of children who had blood testing was on average slightly older than other children (mean age 15.09 years vs 14.71 years). However after adjustment for age, sex and town there were no difference in height and weight between the two groups. Of the adiposity variables only waist: hip ratio and body fat percent from bioimpedance showed differences between the groups that persisted after adjustment for age, sex and town. For waist: hip ratio children who had blood tests had lower mean measurements than those not tested (0.754 vs 0.760,  $p=0.006$ ). These differences were not

removed by additional adjustment for pubertal status, but were removed by adjustment for whether the subject had eaten breakfast (0.753 vs 0.745,  $p=0.2$ ). Body fat percent from bioimpedance was higher in children having blood tests (27.5% vs 25.9%,  $p<0.0001$ ), after adjustment for age, sex and town.

Of the subgroup of pupils invited for blood testing, 1523 (63% response rate) provided a blood sample for the measurement of insulin. A slightly smaller number, 1501 pupils, had measurements of glucose. Table 7.1 shows the mean values for systolic blood pressure, insulin and fasting glucose, by gender. Boys had higher mean SBP than girls, 5mmHg higher. Fasting insulin was 1.2mU/L higher in girls whilst fasting glucose was slightly (0.2mmol/l) higher in boys. Two boys who were found to have type 1 diabetes were excluded from these analyses (one had a glucose of 26.9 mmol/L, one a glucose of 17.0 mmol/L with an insulin level of 148 mU/L). Systolic blood pressure and glucose showed fairly normal distributions.

### **7.3.1 FORMATION OF FIFTHS OF BODY BUILD MEASURES**

Details of fifths of the body build measures are shown in table 7.5, separately for boys and girls. Height, weight and BMI values in each fifth are broadly similar for boys and girls. In contrast, body fat percentage from bioimpedance and sum of skinfolds values are much higher for girls than boys, as expected from previous investigations into gender differences (Chapter 4). Mean values for fifths for central adiposity measures tended to be higher in boys (Table 7.5).

### **7.3.2 SYSTOLIC BLOOD PRESSURE (SBP)**

Blood pressure showed inter-observer variation (Table 7.3) with a range of 4.3 mmHg between the mean SBP recorded by the observer with the lowest values (Observer 1) and the highest observer (Observer 5). The mean inter-observer difference was 0.32 mmHg, 95% CI 0.18-0.46,  $p<0.0001$ . Subsequent analyses have therefore been adjusted for blood pressure observer. In boys and girls all the associations between body size and adiposity and SBP are positive and generally graded, except waist: hip ratio which shows a very weak positive gradient in both genders (Table 7.6). The strongest associations were seen with weight and BMI in both genders and height in boys. There was also a strong association with waist circumference.

### **7.3.2.1 Systolic Blood Pressure (SBP) with fifths of height and weight**

In boys, strong positive associations for SBP were seen with both weight and height. (Table 7.6) In girls, height and weight showed weaker positive associations with SBP; that for height was about a quarter of the strength of the association in boys, that for weight about half of the strength. There was evidence of gender interaction with SBP both for height and weight variables, (p values <0.0001, 0.01 respectively), Table 7.4.

### **7.3.2.2 Systolic Blood Pressure (SBP) with fifths of general measures of adiposity**

In boys, all measures of general adiposity showed a positive association with fifths of SBP. The strongest association was with BMI, with an increase of 2.3mm Hg per fifth increase in BMI. There were smaller increases with fifths of skinfolds and body fat %, of 0.8 and 0.7mmHg respectively. In girls, the strongest positive association was seen with BMI, with a 1.4mmHg increase per fifth. Sum of skinfolds fifth and body fat percent also had significant associations with SBP, with increases of 1.2mmHg and 0.9 mmHg per fifth increase in adiposity variable respectively. There was no evidence of gender interaction for SBP with any of the general measures of adiposity (Table 7.4).

### **7.3.2.3 Systolic Blood Pressure (SBP) with fifths of central measures of adiposity**

In boys, the association of SBP with fifths of waist circumference was stronger than that with fifths of BMI, with an increase of 2.5 mmHg per fifth of waist circumference. There was however no relationship with waist: hip ratio but a significant positive association with subscapular: triceps ratio (1.6 mmHg increase per fifth). A similar pattern was seen in girls, with increases of 1.1mmHg per fifth increase in waist circumference and 0.6 mmHg per fifth of subscapular: triceps ratio.

## **7.3.3 FASTING INSULIN AND FASTING GLUCOSE**

### **7.3.3.1 Effect of eating and drinking on insulin and glucose levels**

Data were collected on whether children had eaten or drunk that morning or had managed to fast overnight as requested. Children who had eaten that morning did not have higher glucose than other children, means being 5.07 mmol/L and 5.06 mmol/L respectively (Table 7.2) however they did have higher mean insulin levels, at 10.77 mmol/L compared with 8.92 mmol/L for children who had fasted (p<0.0001). Non-fasted children are excluded from the analyses.

### **7.3.3.2 Effect of time of day on insulin and glucose levels**

Time of the morning was found to have an effect on both variables: glucose level fell with increasing time (i.e. later morning) in minutes by 0.0005(SE 0.0003) ( $p = 0.05$ ) and insulin fell by -0.06% ( $p = 0.04$ ) with increasing (i.e. later morning) time in minutes. Subsequent analyses have therefore been adjusted for time of day.

### **7.3.4 FASTING INSULIN AND ADIPOSITY**

The relations of fasting insulin to body size variables in fifths, separately for each gender, are shown in Table 7.7.

The largest increases for boys are seen with BMI (11.2% increase in insulin per fifth increase in BMI), waist circumference (10.4%) and skinfolds sum (10.1%) and for girls with body fat % (9.0%), BMI (8.5%) and waist circumference (7.8%). In boys, fasting insulin increased with increasing fifths of all measures except height; and subscapular: triceps ratio. The largest individual increases were seen between the fourth and highest fifths of adiposity variables in both genders. In girls all adiposity variables showed small decreases in fasting insulin from the first to second fifth, then increases through remaining fifths. A similar pattern was seen for BMI and waist circumference in boys.

#### **7.3.4.1 Fasting insulin with fifths of height and weight**

Large increases in fasting insulin were seen with weight in both genders. Fasting insulin increased with height in boys and showed no relationship to height in girls; the relationship in boys was confounded by pubertal status and became weaker on adjustment ( $p=0.06$ ).

#### **7.3.4.2 Fasting insulin with fifths of general measures of adiposity**

BMI, sum of skinfolds and body fat % were all strongly associated with increases in fasting insulin; in boys the strongest association was for BMI and for girls for body fat percent.

#### **7.3.4.3 Fasting insulin with fifths of central measures of adiposity**

Of the central measures of adiposity, waist circumference was most strongly related to fasting insulin in both genders. Waist: hip ratio in both genders was associated with smaller increases in insulin. In girls there was an increase in fasting insulin with rising fifths of subscapular: triceps ratio but in boys there was no relationship. Overall, central measures of adiposity were not more closely related to increases in fasting glucose than

general measures of adiposity. The only adiposity variable for which there was evidence for gender interaction with insulin level was subscapular: triceps ratio (test for interaction,  $p=0.0004$ ).

### **7.3.5 FASTING GLUCOSE AND ADIPOSITY**

The relations of fasting glucose to body size variables in fifths, separately by gender, are shown in Table 7.8. In boys, there were no consistent associations; only the increase seen with sum of skinfolds approached conventional levels of statistical significance. In girls, fasting glucose showed positive relationships with all body build variables except weight. The associations were however not continuously graded but showed particular increases in fasting glucose in the highest fifth of the adiposity variables. Fasting glucose decreased with increasing height in girls, but showed no relationship with height in boys, nor was there evidence of gender interaction.

#### **7.3.5.1 Fasting glucose with fifths of height and weight**

In boys, fasting glucose showed no overall change with increasing fifths of height and weight (test for trend  $p=0.4$  for both variables). Weight fifths in girls did not show a significant relationship with fasting glucose, producing a slightly 'u' shaped curve. There was no evidence of gender interaction ( $p=0.35$ ). Fasting glucose was lower in taller girls, with a slope of  $-0.04$  ( $p=0.004$ ). There was no gender interaction for height with fasting glucose ( $p=0.69$ ).

#### **7.3.5.2 Fasting glucose with general measures of adiposity**

In boys, fasting glucose showed no relationship to general adiposity variables, particularly BMI and body fat percent. The only exception was sum of skinfolds, which showed a modest increase in glucose  $0.02$  mmol/L per fifth of the skinfolds distribution, (test for trend,  $p=0.07$ ).

In girls, fasting glucose increased with increasing fifth of BMI, sum of skinfolds and body fat percent. The pattern in each was similar, with glucose values remaining fairly 'flat' to  $4.92-4.95$  in the fourth fifth, then a sharp increase to the highest fifth, with  $p$  values for trend of  $0.02$ ,  $0.03$  and  $0.04$  respectively. However, there was no evidence of appreciable gender interaction for BMI, sum of skinfolds and body fat percent;  $p$  values for interaction tests were  $0.16$ ,  $0.39$  and  $0.96$  respectively.

### **7.3.5.3 Fasting glucose with fifths of central measures of adiposity**

Fasting glucose in boys showed no relationship to measures of central adiposity (waist circumference, waist: hip ratio and subscapular: triceps ratio). Boys in the highest fifth for waist circumference had higher mean fasting glucose than other boys (5.22 mmol/L), including boys in the highest fifth for other variables. In girls, glucose showed a positive relationship with all three central adiposity variables, with particular increases between the fourth and highest fifths. For waist circumference there was an overall increase of 0.03 mmol/L ( $p=0.04$ ), for waist: hip ratio 0.04 mmol/L ( $p=0.003$ ), and for subscapular: triceps ratio 0.04 mmol/L,  $p=0.001$ . There was evidence of gender interaction for subscapular: triceps ratio (test for interaction,  $p= 0.03$ ) but not for waist or waist: hip ratio (tests for interaction,  $p=0.32$  and 0.42 respectively).

## **7.4 DISCUSSION**

The baseline differences seen in waist: hip ratio between children with blood tests and others may be linked to fasting status; if so the results for insulin and glucose would not be affected as these are fasting children only. It is difficult to advocate for adjusting for fasting status throughout the study as this was a self-reported yes/no variable, in which yes may mean anything from a large breakfast to a few sips of water. The link to fasting seems a plausible explanation for the differences in waist: hip ratios but there are other possible explanations linked to the age difference between the groups such as exercise level.

The difference in body fat percent from bioimpedance level is also difficult to explain; possibly linked to fasting status as biochemical changes are known to affect the value of this variable (Houtkooper et al 1992). Other explanations may be residual confounding by age, or observer variation. Decision was taken early in analysis not to attempt to adjust this variable for observer as it was clear that the measured groups were not similar (Section 4.3.2).

Systolic blood pressure showed strong positive relations with height, weight and body mass index, both among boys and girls. Among the measures of general adiposity (body mass index, sum of skinfolds, body fat percent), body mass index showed the strongest relationship with blood pressure. Among markers of central adiposity, waist circumference (though not waist hip ratio) and subscapular triceps skinfold ratios were positively related to systolic pressure. Among boys (though not girls) waist circumference showed a stronger relation to systolic pressure than body mass index.

Insulin level was not strongly related to height, but showed strong positive relations with weight and body mass index, both among boys and girls. Among the measures of general adiposity (body mass index, sum of skinfolds, body fat percent), body mass index showed the strongest relationship with insulin in boys, while body fat percent was stronger in girls. Among markers of central adiposity, waist circumference and to a lesser extent waist-hip ratio were related to insulin level in both sexes; in boys (though not in girls) the relationships were almost as strong as for body mass index. Fasting glucose levels showed little relation to height, weight or any adiposity measures (general or central) in boys. In girls however, all adiposity measures showed modest positive associations of similar strength with glucose level.

Adjustment for pubertal stage would be potentially valuable, particularly for the analyses of fasting insulin, which varies with pubertal stage, being slightly higher in mid-puberty, linked to pubertal changes; growth hormone is associated with insulin resistance (American Diabetes Association 2000). Additional adjustment both for age and puberty (data not presented) did not materially alter most of the relationships described. The strength of the relations between systolic blood pressure and height, weight, BMI, waist circumference were slightly reduced, though they remained the factors most closely associated with systolic blood pressure. The relationship between waist: hip ratio and systolic blood pressure became statistically significant after adjustment for age and puberty in boys ( $p=0.02$ ), but not in girls. Adjustment for pubertal status had little effect on the results seen with insulin and glucose levels, except that the associations with height became insignificant (negative association between height and fasting glucose in girls, positive association between height and fasting insulin in boys). Essentially similar results to those for fasting insulin were found when the HOMA model (glucose  $\times$  insulin/22.5) (Matthews et al, 1985) was used as the dependent variable.

#### **7.4.1 RELATION OF FINDINGS TO OTHER STUDIES**

##### **7.4.1.1 Systolic blood pressure and adiposity measures**

When comparing our systolic blood pressure results to the Health Survey for England 2003 16-24 year olds mean systolic blood pressures (113.4 for women, 123.2 for men), the levels we observed in girls are high. This cannot simply be due to Dinamap recording systolic blood pressure about 8mm higher than a mercury sphygmomanometer (Whincup et al. 1992c) as a similar degree of high readings were not seen in boys. It could be due to greater situational anxiety among the girls. The difference in systolic blood pressure

between the lowest and highest BMI groups is broadly comparable with that found by McMurray et al in the Cardiovascular Health in School (CHIC) study in the USA, where a difference of 4mmHg was found between obese and non obese boys at age 8-10 years, and 5mmHg in girls; similar to the differences between our 2<sup>nd</sup> and 5<sup>th</sup> fifths for BMI in both sexes (McMurray et al, 1995). Our finding that BMI is strongly associated with systolic blood pressure is in agreement with the findings of Paradis (Paradis et al, 2004), who found similar graded associations between systolic blood pressure and fifths of BMI in a school-based survey of 9-16 year olds. Other surveys in children and adolescents have found strong associations between systolic blood pressure and BMI (Vizcaíno et al, 2007, de Silva et al, 2006, Ribeiro et al, 2003 {8-16 year olds}). There were no other studies comparing BMI with as many other measures of adiposity as in our study. Vizcaíno comparing BMI and body fat percent in children aged 8-11 found that body fat percent was more closely related to serum markers of cardiovascular risk, and BMI to systolic blood pressure. De Silva found stronger associations for BMI and waist circumference in children and adolescents with systolic blood pressure than for body fat percent. Paradis found a similar association for subscapular and triceps skinfolds with systolic blood pressure as found with BMI (in 9-16 year olds) but did not comment on the comparative strength of the association.

Studies of obese and non-obese subjects suggest that raised blood pressure which accompanies weight gain improves upon weight loss (Reinehr et al, 2004, 2005a, 2005b), as does insulin resistance and lipid profile. The physiological mechanism for the hypertension remains unclear but may be due to alteration in the balance between sympathetic and parasympathetic nervous systems (Daniels 2002). Further analyses could be done on our study population to look at the diastolic blood pressure distribution.

#### **7.4.1.2 Fasting insulin and adiposity measures**

Our values for fasting insulin fall within the expected ranges (7-24mU/L), except for the lowest quintile of sum of skinfolds in girls (6.9 mU/L), suggesting that this group contained some extremely thin subjects. The higher values seen in girls compared to boys are consistent with findings in other studies both in younger, 5 year-old children (Murphy et al, 2004) and in older, 9-16 year-olds (Punthakee et al, 2006). Murphy hypothesised that these are sex-linked genetic differences. Boys in our study showed the largest percentage changes in fasting insulin; this may be in agreement with other work suggesting that

adolescent boys are particularly responsive to adiposity in terms of adverse biochemical changes (Reiterer et al, 1999 {leptin}, Punthakee et al, 2006 {adiponectin}).

The association of fasting insulin with adiposity in children has been found in other studies including children as young as 2-3 years (Shea) (Shea et al, 2003, Krekoukia et al, 2007, Knip, Nuutinen 1993). In intervention programmes for obese children serum lipid profile and insulin normalise or improve with reduction in BMI (Reinehr et al, 2005a, 2005b, Reiterer et al, 1999, Nuutinen, Knip, 1992) and worsen with increasing obesity (Reinehr et al, 2004).

Most studies have used BMI to assess adiposity, sometimes with one other adiposity measure, then conclude that BMI is the superior measure. Punthakee collected subscapular and triceps skinfold data but did not include them in multivariable analyses as they were highly correlated with BMI (z scores) and BMI was deemed to be the most 'clinically applicable' measure (Punthakee et al, 2006). Raitakari et al in the Cardiovascular risk in Young Finns study when looking at insulin increases with BMI and subscapular skin fold measurement concluded that insulin increased with both measures but more significantly with BMI. With both body size measures in their regression model the trend with subscapular skinfold became insignificant (Raitakari et al, 1995). In the Bogalusa Heart study (5-17 year olds) lipid profile, insulin and blood pressure levels were investigated; it was concluded that if BMI was known, triceps skinfold thickness (the other adiposity variable used) 'provided little additional information on risk factor levels' (Freedman et al, 1999). More recently Krekoukia found that both body fat percent (from skinfolds) and waist circumference were positively associated with fasting insulin in 9-11.5 year olds but not comparing the strength of the association with that for BMI (Krekoukia et al, 2007). In our study BMI was associated with the largest per cent increase in fasting insulin in boys and second largest in girls, after body fat percent from bioimpedance. Waist circumference had the most significant effect on insulin when considering the central measures of adiposity, being second after BMI in boys and third after total body fat and BMI in girls. This appears consistent with the body imaging findings that insulin production and function were related to total body fat and subcutaneous abdominal fat (Goran, Gower, 1998).

#### **7.4.1.3 Fasting glucose and adiposity measures**

The finding of slightly higher blood glucose in boys than girls is in keeping with other studies (Murphy et al, 2004, Punthakee et al, 2005). In boys no measures of general or

central adiposity showed a significant relationship with fasting glucose. Sum of skinfolds showed a graded positive association with fasting glucose but did not achieve significance ( $p=0.07$ ). For all adiposity measures the greatest increases were seen between 4<sup>th</sup> and 5<sup>th</sup> fifths. The highest mean fasting glucose levels (5.22) of all were in the highest fifth of waist circumference. In girls all adiposity variables except height and weight show a positive relationship with fasting glucose, the most significant changes are with subscapular: triceps ratio, waist: hip ratio then BMI; for girls the central measures of adiposity are a 'better' measure of fasting glucose than BMI. The highest value for mean fasting glucose in girls (5.11) is seen in the highest fifths for waist: hip ratio, waist circumference and BMI.

The finding of a clearer relationship of fasting glucose to central adiposity in girls compared to boys is similar to findings in adults (Haffner et al, 1991, using subscapular: triceps ratio as a measure of 'centrality'). The small absolute changes seen in fasting glucose are as expected; even in groups of very obese children raised fasting glucose is uncommon, although insulin resistance can be seen in up to 40% (Viner et al, 2005), and may be a precursor to Type II diabetes (i.e. loss of glycaemic control (McCance et al, 1994). The increase in mean glucose seen in the highest quintile for most adiposity measures implies a degree of loss of glycaemic control in some of these individuals.

#### **7.4.2 STRENGTHS AND WEAKNESSES OF THE STUDY**

Strengths of the study include the representative, school-based population, with a reasonably high response rate. A wide range of measures of general and central adiposity were obtained, allowing comparison, although none of the adiposity measures is a 'gold standard' measure like DXA scan.

This wave of the study did not include oral glucose tolerance testing, which would have allowed more detailed investigation of insulin sensitivity and particularly of beta-cell function. However recent work suggests that raw fasting insulin and glucose data have acceptable sensitivity and high specificity when identifying adolescent subjects with insulin resistance syndrome (Viner et al, 2005). In addition, the validity of fasting insulin as a good marker of insulin resistance in population studies was established by Laakso, who reported its suitability for normoglycaemic subjects as well as those with impaired glucose tolerance and NIDDM, whereas the correlation of post-load insulin levels with directly measured insulin resistance varied greatly (Laakso 1993).

## **7.5 IMPLICATIONS FOR AETIOLOGY AND PREVENTION**

The relations between adiposity, blood pressure and insulin resistance in adolescents are likely to reflect causal associations. They are strong, graded and consistent for different adiposity measures. Data from other studies show that increases in adiposity precede the development of adverse changes in blood pressure and insulin level (Sinaiko et al, 1999, Bao et al, 1994) and that the effects of adiposity are reversible (Reinehr et al, 2005a, 2005b, Reiterer et al, 1999, Nuutinen, Knip 1992). This suggests that the rising levels of adiposity and obesity may well have important adverse consequences for the risks of cardiovascular disease and type 2 diabetes. This is borne out by current time trends both in the UK and other settings, particularly for type 2 diabetes, which show that rising levels of obesity are being accompanied by marked increases in risks of type 2 diabetes, which is occurring at increasingly younger ages (Ehtisham et al, 2000, Pinhas-Hamel, Zeitler 2005). The situation for coronary heart disease is more complex. Despite the increase in obesity prevalence, rates of coronary heart disease in the UK are declining. This probably reflects a decline in exposure to other, stronger, risk factors for coronary heart disease (particularly cigarette smoking and LDL-cholesterol levels) (Whincup, Deanfield 2005). However, it is likely that the extent of the decline in coronary heart disease is being limited by rising levels of obesity (Hardoon et al, 2008).

The prevention of obesity will depend on measures to reduce calorie intake, increase energy expenditure or a combination of the two. This might be achieved either by population or high risk strategies (Rose 1992); in reality both approaches can be used simultaneously. Population-wide measures could reduce the consumption of energy dense foods and drinks, or facilitate energy expenditure, either by encouraging walking or formal exercise activities. The 'high-risk' approach, identifying subjects on the basis of their body mass index or waist circumference, could be used to define a group of subjects who would receive focussed advice and encouragement to lose weight. An example is the 'WATCH IT' programme in Leeds, targeted at obese children in disadvantaged communities (Rudolf et al, 2006). These different approaches to prevention are discussed in more detail in Chapter 8 (section 8.5).

**Table 7. 1 Numbers of children with cardiovascular risk factor measures**

SBP (systolic blood pressure) in mmHg, insulin mU/L, Glucose mmol/L.

Variable	Number of Subjects	Mean	Median	Interquartile Range	Standard Deviation
<b>Systolic blood pressure (SBP): all children</b>	2643	121.38	120.00	18.00	13.06
<b>SBP boys</b>	1408	123.93	122.5	20.0	13.57
<b>SBP girls</b>	1235	118.47	117.00	15.50	11.81
<b>Insulin: all children</b>	1523	10.26	7.30	5.10	6.43
<b>Insulin boys</b>	838	9.73	8.10	5.20	7.01
<b>Insulin girls</b>	685	10.90	9.70	5.00	5.58
<b>Fasting glucose(FG): all children</b>	1501	5.06	5.00	0.50	0.47
<b>FG boys</b>	829	5.14	5.10	0.50	0.45
<b>FG girls</b>	672	4.96	4.90	0.60	0.47

**Table 7. 2 Effect of whether child (subject) had fasted on insulin and glucose levels**

	Fasted*	Not fasted*	P value
<b>Number of children</b>	1335	154	
<b>Insulin, ls mean +</b>	8.92	10.77	<0.0001
<b>Glucose, ls mean (SE)</b>	5.07 (0.04)	5.06 (0.01)	0.88

\*11 children- information missing

+ analysed as log variable

**Table 7. 3 Observer variation in systolic BP measurement (after adjustment for age and gender).**

<b>Observer (no of children)</b>	<b>1</b> (429)	<b>2</b> (666)	<b>3</b> (302)	<b>4</b> (283)	<b>5</b> (960)	<b>p value for inter-observer difference</b>
<b>Systolic BP (SE) (mm Hg)</b>	118.8 (0.6)	120.8 (0.5)	121.1 (0.7)	119.4 (0.8)	123.1 (0.4)	p<0.0001

**Table 7. 4 Tests for gender interaction, p values for difference**

	<b>SBP</b>	<b>Fasting Insulin+</b>	<b>Fasting Glucose</b>
<b>Height</b>	<b>&lt;0.0001</b>	0.20	0.69
<b>Weight</b>	<b>0.001</b>	0.74	0.35
<b>BMI</b>	0.91	0.26	0.16
<b>Sum of Skinfolds</b>	0.14	0.30	0.39
<b>Fat % from bioimpedance</b>	0.24	0.78	0.96
<b>Waist circ.</b>	0.19	0.38	0.32
<b>WHR</b>	1.0	0.16	0.42
<b>STR</b>	0.10	<b>0.0004</b>	<b>0.03</b>

+ analysed as log variable

**Table 7. 5 Fifths of adiposity measures**

	<b>Fifths: mean (range)</b>				
	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>
<b>BOYS</b>					
<b>Height m</b>	1.56 (1.38-1.61)	1.64 (1.61-1.67)	1.69 (1.67-1.72)	1.74 (1.72-1.76)	1.81 (1.77-1.96)
<b>Weight kg</b>	43.1 (26.2-48.4)	51.6 (48.5-54.5)	57.3 (54.6-60.0)	63.7 (60.1-68.1)	78.1 (68.2-134.0)
<b>BMI</b>	16.8 (13.7-17.9)	18.5 (17.9-19.1)	19.8 (19.1-20.5)	21.5 (20.5-22.7)	26.0 (22.7-41.7)
<b>Sum of skinfolds mm</b>	23.6 (17.2-26.6)	29.6 (26.8-32.4)	35.8 (32.4-40.2)	48.5 (40.4-58.4)	80.8 (58.5-140.0)
<b>Body fat %</b>	14.7 (2.7-16.9)	18.2 (16.9-19.5)	20.8 (19.5-22.3)	24.2 (22.3-26.4)	30.7 (26.4-45.1)
<b>Waist circ.</b>	62.4 (53.8-65.1)	66.6 (65.2-68.0)	69.8 (68.0-71.3)	73.7 (71.3-76.9)	85.4 (76.9-120.0)
<b>WHR</b>	0.73 (0.65-0.75)	0.76 (0.75-0.77)	0.78 (0.77-0.79)	0.80 (0.79-0.82)	0.86 (0.82-1.34)
<b>STR</b>	0.51 (0.19-0.62)	0.67 (0.62-0.73)	0.79 (0.73-0.85)	0.92 (0.85-0.99)	1.15 (1.00-1.67)
<b>GIRLS</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>
<b>Height m</b>	1.53 (1.43-1.57)	1.59 (1.57-1.60)	1.62 (1.60-1.64)	1.65 (1.64-1.67)	1.71 (1.67-1.82)
<b>Weight kg</b>	43.7 (30.3-47.2)	49.8 (47.3-52.2)	54.3 (52.3-56.8)	59.8 (56.9-63.4)	72.4 (63.5-100.9)
<b>BMI</b>	17.2 (13.5-18.3)	19.2 (18.3-20.0)	20.7 (20.0-21.4)	22.4 (21.5-23.7)	27.1 (23.8-38.4)
<b>Sum of skinfolds mm</b>	35.2 (21.8-41.6)	46.5 (41.6-51.6)	56.4 (51.8-61.4)	69.6 (61.6-78.8)	96.6 (79.0-141.4)
<b>Body fat %</b>	25.3 (14.3-28.1)	29.4 (28.1-30.7)	31.9 (30.7-33.0)	34.3 (33.0-36.0)	38.9 (36.0-47.1)
<b>Waist circ.cm</b>	59.2 (51.2-61.5)	63.2 (61.6-64.6)	66.3 (64.7-68.0)	70.5 (68.1-73.5)	81.0 (73.6-107.2)
<b>WHR</b>	0.67 (0.62-0.69)	0.70 (0.69-0.71)	0.72 (0.71-0.73)	0.75 (0.73-0.76)	0.80 (0.76-1.30)
<b>STR</b>	0.48 (0.25-0.56)	0.62 (0.56-0.66)	0.71 (0.66-0.76)	0.82 (0.76-0.90)	1.05 (0.90-1.66)

**Table 7. 6 Systolic Blood Pressure (SBP) by fifth of body size variables, adjusted for observer**

<b>BOYS</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>Increase in SBP (95%CI) with fifth of body size variable, p value</b>
<b>Height (m)</b>	116.1	122.1	126.0	127.7	128.59	3.1(3.0to3.2)), p<0.0001
<b>Weight (kg)</b>	115.5	122.4	125.7	128.1	128.5	3.2 (2.7to3.7), p<0.0001
<b>BMI (kg/m<sup>2</sup>)</b>	117.6	123.6	124.4	128.7	126.1	2.3 (1.8to2.8),p<0.0001
<b>Sum of skinfolds (mm)</b>	121.3	123.9	124.8	124.4	125.2	0.8 (0.3to1.4),p=0.002
<b>Body Fat %</b>	122.7	122.6	125.2	125.1	124.6	0.7 (0.2to1.2),p=0.01
<b>Waist circ. (cm)</b>	117.7	122.4	124.9	127.6	127.8	2.5 (2.0to3.0),p<0.0001
<b>WHR</b>	124.1	123.1	124.5	123.1	125.5	0.2 (0.7to-0.3),p=0.44
<b>STR</b>	119.2	123.9	124.4	125.5	126.6	1.6 (1.1to2.1),p<0.0001
<b>GIRLS</b>	<b>1</b>	<b>2</b>	<b>3</b>	<b>4</b>	<b>5</b>	<b>Increase in SBP (95%CI), with fifth of body size variable, p value</b>
<b>Height (m)</b>	117.7	117.6	118.1	118.8	120.9	0.8 (0.3to1.3), p=0.001
<b>Weight (kg)</b>	115.4	117.0	119.2	119.5	121.9	1.5 (1.1to2.0), p<0.0001
<b>BMI (kg/m<sup>2</sup>)</b>	115.9	116.4	118.8	120.9	121.0	1.4 (1.0to1.9),p<0.0001
<b>Sum of skinfolds (mm)</b>	114.8	119.0	117.8	119.7	120.9	1.2 (0.7to1.7),p<0.0001
<b>Body Fat %</b>	117.3	116.8	118.8	119.0	120.5	0.9 (0.4to1.3), p=0.0007
<b>Waist circ. (cm)</b>	116.5	117.4	117.8	120.1	121.3	1.1 (0.7to1.6),p<0.0001
<b>WHR</b>	118.6	117.9	118.7	117.7	120.1	0.2 (0.7to-0.3), p=0.50
<b>STR</b>	117.9	117.3	118.5	119.3	119.7	0.6 (0.1to1.0),p=0.02

**Table 7. 7 Insulin by fifth of body size variables, adjusted for time of day. Insulin analysed as log variable**

BOYS	1	2	3	4	5	Increase in Insulin (95%CI), with fifth of body size variable, p value
<b>Height (m)</b>	7.69	7.83	8.35	8.51	8.51	2.8%(0.2 to5.3), p=0.03
<b>Weight (kg)</b>	7.10	7.53	7.84	8.12	10.41	9.2%(6.7to11.7),p<0.0001
<b>BMI (kg/m<sup>2</sup>)</b>	7.10	7.54	7.16	8.87	11.07	11.2%(10.8to11.4),p<0.0001
<b>Sum of skinfolds (mm)</b>	6.90	7.37	8.09	8.52	10.46	10.1%(7.5to12.7),p<0.0001
<b>Body Fat %</b>	7.15	7.30	7.92	8.28	10.61	9.8%(7.2to12.5), p<0.0001
<b>Waist circ. (cm)</b>	7.09	7.64	7.54	8.22	11.20	10.4%(7.8to12.9),p<0.0001
<b>WHR</b>	7.53	7.84	8.13	8.23	10.13	6.3%(3.9to8.9),p<0.0001
<b>STR</b>	7.55	8.66	7.98	8.28	8.50	2.0%(4.4to-0.5),p=0.11
GIRLS	1	2	3	4	5	Increase in Insulin (95%CI), with fifth of body size variable, p value
<b>Height (m)</b>	10.24	9.91	9.79	9.91	9.48	-1.5%(0.8to-2.3), p=0.2
<b>Weight (kg)</b>	9.15	9.01	9.43	9.43	12.25	6.8%(4.3to9.3), p<0.0001
<b>BMI (kg/m<sup>2</sup>)</b>	9.03	8.76	9.25	9.65	12.93	8.5%(6.1to11.0),p<0.0001
<b>Sum of skinfolds (mm)</b>	8.80	8.93	9.13	9.79	12.05	7.6%(5.2to10.0),p<0.0001
<b>Body Fat %</b>	8.94	8.62	9.02	9.68	12.50	9.0%(6.5to11.5),p<0.0001
<b>Waist circ. (cm)</b>	9.35	8.75	8.96	9.66	12.89	7.8%(5.3to10.2),p<0.0001
<b>WHR</b>	9.39	8.86	9.69	9.30	12.56	6.3%(3.9to8.7),p<0.0001
<b>STR</b>	9.23	9.05	9.75	9.55	11.30	4.8%(2.4to7.2),p<0.0001

**Table 7. 8 Fasting glucose by fifth of body size variables, adjusted for time of day.**

BOYS	1	2	3	4	5	Increase in Glucose (95%CI), with fifth of body size variable, p value
<b>Height (m)</b>	5.14	5.16	5.15	5.10	5.13	-0.01 (0.01to-0.03), p=0.4
<b>Weight (kg)</b>	5.17	5.13	5.16	5.12	5.13	-0.01 (0.01to-0.03),p=-0.4
<b>BMI (kg/m<sup>2</sup>)</b>	5.18	5.13	5.06	5.15	5.17	-0.00(0.02to-0.02),p=0.9
<b>Sum of skinfolds (mm)</b>	5.08	5.16	5.10	5.16	5.19	0.02 (-0.001to0.04),p=0.07
<b>Body Fat %</b>	5.18	5.12	5.12	5.15	5.15	-0.00(0.02to-0.02),p=0.9
<b>Waist circ. (cm)</b>	5.15	5.14	5.11	5.08	5.22	0.01(0.03to-0.01),p=0.45
<b>WHR</b>	5.12	5.14	5.16	5.10	5.17	0.004(0.03to-0.02),p=0.68
<b>STR</b>	5.14	5.15	5.13	5.11	5.16	0.00(0.02to-0.02),p=0.93
GIRLS	1	2	3	4	5	Increase in Glucose (95%CI), with fifth of body size variable, p value
<b>Height (m)</b>	5.09	4.99	4.98	4.92	4.92	-0.04(-0.01to-0.07), p=0.004
<b>Weight (kg)</b>	5.05	4.92	4.93	4.90	5.07	0.01(-0.02to0.04), p=0.63
<b>BMI (kg/m<sup>2</sup>)</b>	4.92	4.96	4.96	4.92	5.10	0.03(0to0.06),p=0.02
<b>Sum of skinfolds (mm)</b>	4.94	4.92	4.96	4.94	5.07	0.03(0to0.06),p=0.03
<b>Body Fat %</b>	4.93	4.97	4.91	4.95	5.07	0.03(0.002to0.06),p=0.04
<b>Waist circ. (cm)</b>	4.95	4.94	4.98	4.90	5.11	0.03(0.001to0.06),p=0.04
<b>WHR</b>	4.93	4.94	4.93	4.98	5.11	0.04(0.01to0.07),p=0.003
<b>STR</b>	4.90	4.89	4.99	4.99	5.07	0.04(0.01to0.07),p=0.001

## **CHAPTER 8: CONCLUSION**

### **8.0 SUMMARY**

Body mass index is a useful measure of general adiposity in 13-16 year olds, being significantly associated with adverse patterns of cardiovascular risk factors (higher systolic blood pressure, fasting insulin). Limitations of BMI include that it is not independent of pubertal stage and did not show as close associations to physical activity level as other measures of adiposity. BMI may not be ideal for assessing improvements in adiposity level with increased exercise, in both individuals and populations. Sum of skinfolds are relatively difficult to measure accurately, particularly in the obese; crude 'pinch' type estimates may be useful for self-assessment, as skinfolds were a good measure of general adiposity. Body fat percent from bioimpedance appeared to reflect differing levels of adiposity in the population but it is not clear that this would translate to individual measurement, due to reliance on formulas and assumptions about body composition. The purported central measures of adiposity did not correlate closely to each other and it was not clear that they represented central adiposity (visceral fat) in this age group. Waist circumference is a simple measure that appeared to represent general, rather than central adiposity in this age group and may be useful as an adjunct to BMI.

Levels of adiposity are rising in adolescents in Britain with consequent increase in prevalence of obesity and overweight, as demonstrated in this study and others. Excess adiposity is causing an increasing health burden including rising levels of type 2 diabetes, adverse lipid profile, reduced arterial distensibility and hypertension. Tracking of excess adiposity to adult life is more likely in adolescence than childhood, in lower social classes and if parents are obese. Prevention of excess adiposity is a public health priority.

Strategies for prevention of excess adiposity may include high-risk and population strategies. Obesity is essentially an imbalance between energy (calorie) intake and energy expenditure as physical activity; interventions would need to address this imbalance. Currently high risk strategies are used in identifying those who are obese on the basis of BMI; individuals then receive treatment including intensive activity programmes and dietary supervision. Currently there is lack of evidence supporting the effectiveness of these intensive interventions. Population strategies would include measures to encourage all adolescents to become more physically active, perhaps through a combination of dedicated sports provision and encouraging building activity into everyday life. It would also include population-wide campaigns to decrease intake of high-fat, high-sugar foods.

Key priorities for research include further investigation of valid measures of central adiposity in this age group. Comparative neighbourhood studies may help to clarify the underlying impediments and adverse pressures to healthy behaviour, for example between adolescents in Rhondda (high adiposity) and Bath (low adiposity). High quality evidence on effective interventions for adiposity in adolescents is also needed, both in terms of population measures and for those identified as obese.

## **8.1 INTRODUCTION**

The previous chapters have examined a range of different measures of adiposity in a group of 13-16 year olds, including their interrelationships and distribution in the study population (Chapter 4), and association with selected markers of cardiovascular risk (Chapter 7). Selected correlates (geographic, ethnic, social class) and determinants (parental BMI, breast feeding, physical activity level) of adiposity were also examined in Chapters 5 and 6 respectively. In Chapter 4 the study population was compared with proposed international standards for obesity. In this chapter the implications of the study's findings are considered in more detail. An overview of the main findings of this thesis with regard to adiposity in adolescence is presented in Section 8.2. Section 8.3 examines the implications of the findings of this thesis with regard to the measurement of adiposity in adolescence. Section 8.4 examines the potential public health importance of the findings of this thesis and the final section, 8.5, looks at some implications arising from this work for further research together with some suggestions for further studies.

## **8.2 THE MAIN FINDINGS**

In the study population measures of general adiposity were all highly correlated to each other. Among the measures of central adiposity waist circumference was highly correlated to general adiposity measures, but less strongly to waist: hip ratio and subscapular: triceps ratio; interrelationships between central measures were not as strong as between general measures. The age-and puberty-related changes seen in this group of 13-16 year olds included older boys being taller and heavier with more centralized distribution of body fat but lower subcutaneous and total body fat compared to girls. The effects of age and puberty were synergistic but independent. This group of adolescents showed increased obesity levels when compared to the proposed international cutoffs for obesity (Cole et al 2000); 10% being obese and 25% overweight. They also showed higher than expected waist circumference measurements, in keeping with contemporaneous national and

international trends (McCarthy et al 2003, Moreno et al 2005, Rudolf et al 2004, Li C et al 2006).

Adiposity showed associations with the cardiovascular risk factors studied, showing consistent, graded relationships with SBP and fasting insulin in study children.

Associations were strongest with BMI and waist circumference in boys and BMI and body fat % in girls. In girls but not boys all adiposity measures showed modest positive associations of similar strength with fasting glucose level.

There were marked geographic variations between towns in all variables. Children in Rhondda had particularly high levels of adiposity. However, these differences did not really follow a North: South high/low mortality pattern, except for height, which showed a low –high mortality town difference of 1.8cm for boys and 2.2cm for girls. Although children in low mortality towns tended to have lower adiposity measures, these differences were not large or statistically significant. Social class differences were also apparent in height both in boys and girls. Social differences in adiposity, in contrast, were only apparent in girls, in whom general adiposity measures (BMI, body fat percent) and central adiposity measures (waist circumference, waist: hip ratio) tended to increase with lower social class; such associations were not apparent in boys. In analyses based on boys and girls combined, children from lower social classes were shorter, with higher body fat percent and waist: hip ratio than other children; differences in body fat percent were confounded by town. Ethnic differences were also seen with South Asian children, who were shorter and lighter with lower BMI and higher body fat percent than white children. The effect was stronger in girls. Of the central measures of adiposity only subscapular: triceps ratio showed an ethnic difference, being significantly higher in South Asians.

Of the determinants of adiposity studied parental BMI and child physical activity level were found to have positive associations with child adiposity; breast feeding was associated with lower levels of adiposity but this was largely due to confounding by other factors, particularly social class. General adiposity in the children showed a graded association with both mother's and father's BMI, as did waist circumference among the measures of central adiposity. Child's self-reported habitual level of exercise and perception of themselves as an active or inactive person are both linked to adiposity measures. For habitual exercise taken the strongest relationships in both sexes were seen with measures of general adiposity other than BMI, particularly skinfold thickness and

body fat %; general adiposity was lower in higher exercise groups, as was waist circumference in boys. Between the ages of 13 and 15 years older boys' participation in regular exercise was higher and girls' lower. At age 15 boys were twice as likely as girls to take exercise 4 or more times a week (60% vs 30%). Overall 25% of girls took no exercise, as was the case for 12% of boys. Adiposity measures were lower among subjects who had been exclusively breast fed in the first three months; differences in measures of general adiposity and also waist-hip ratio were statistically significant. However after adjusting for confounders only differences in sum of skinfolds remained significant.

### **8.2.1 STRENGTHS AND LIMITATIONS OF THE STUDY**

Major strengths of this study were the use of a comprehensive range of easily conducted, non-invasive adiposity measures, designed to investigate the presence of both general and central adiposity, in a representative school-based population with a reasonably high response rate. The data were collected within one school year, during term time, minimizing confounding by external factors.

The study design, although adequate for studying patterns and determinants of adiposity in individuals, had limited value for comparisons of the health of children from towns with high and low adult cardiovascular mortality. Only substantial differences between high and low mortality towns could be detected due to limited statistical power (five versus five towns). Several of the exposure measures studied relied on self reporting. The physical activity level relied on self-reporting and is likely to have introduced imprecision and potentially bias to the recording of physical activity; use of accelerometers would have provided more accurate assessment of actual exercise level. Reporting of breast feeding practice by the parent is known to be accurate over an extended period. Self-reported parental height and weight is known to be reasonably valid.

### **8.3 IMPLICATIONS OF THE FINDINGS FOR MEASUREMENT OF ADIPOSITY IN ADOLESCENTS**

#### **8.3.1 BMI IS A VALID MARKER OF ADIPOSITY IN THE ADOLESCENT POPULATION**

Currently in both epidemiological and clinical practice BMI is the measure used for both monitoring population levels of adiposity and for individual assessment of obesity and overweight. Evidence from this study confirms that in 13-16 year olds higher BMI (after

age adjustment) is significantly associated with adverse patterns of cardiovascular risk factors (higher systolic blood pressure, fasting insulin). Although other measures of general adiposity are also associated with these risk factors, the strength of the associations seen are generally weaker. This seems to support the choice of BMI, as opposed to other measures of general adiposity, in assessing adiposity. It also suggests that the formation of cut offs for BMI defining obesity and overweight in adolescence do have meaning in terms of risk i.e. those at the top of the BMI distribution are also the top end of the distribution for SBP and fasting insulin; defining a group at particular risk of adverse arterial and metabolic changes. This is consistent with findings in other studies, notably the Bogalusa Heart Study (Reilly et al 2003, review).

### **8.3.2 LIMITATIONS OF USE OF BMI IN ADOLESCENTS**

No measures of adiposity were independent of pubertal changes, except for waist: hip ratio and subscapular: triceps ratio in girls; the pubertal changes were independent of age. This has implications that pubertal stage may act as a confounding factor in assessing population change in adiposity, particularly if the age of puberty is falling over time, as previously demonstrated by the age at menarche in study girls (Whincup et al 2001). Pubertal stage may also act as a confounder when comparing adiposity levels between adolescent populations that have different mean timing of puberty. When assessing BMI in individuals, approximate pubertal status needs to be taken into account. As BMI is not independent of height in childhood and adolescence it is less sensitive at identifying adiposity in short children (Mulligan et al 1999); short children in our study were overrepresented in disadvantaged or high-risk groups; including lower social class boys, South Asian children and children in high mortality towns. This suggests that on both an individual level (BMI cut offs on growth charts) and on population monitoring, reliance on BMI measurement to define adiposity in these groups is potentially misleading. For example the greater adiposity seen in Asian children was only apparent on body fat percent and subscapular: triceps skinfold measurement; BMI (and waist circumference) being lower in Asian children than whites. One way of addressing this might be to consider the use of an additional measure of adiposity in children, particularly in those who might be considered 'high risk', or where there may be doubts about the validity of BMI. This will be considered further in Section 8.3.4.

### **8.3.3 ASSESSING CHANGES IN ADIPOSITY**

As the data for this thesis are cross-sectional it is not possible to draw many conclusions about the value of these measures for detecting changes in adiposity, but it is worth noting that BMI was less closely associated than other measures of general adiposity to physical activity level. This may reflect the fact that increased levels of physical activity would tend to be associated with both increased muscle mass and decreased fat mass, with little net change in body mass index, which reflects both fat- and fat-free mass. This suggests that BMI alone may be of limited use in adolescents who are increasing activity as part of weight reduction measures. This also raises concerns about the validity of monitoring adiposity in the child population using BMI coincidentally with advocating and seeking to enhance participation in physical activity; change (or lack of change) in BMI may be difficult to interpret in this context.

### **8.3.4 WHICH ADDITIONAL MEASURE WOULD BE USEFUL?**

The evidence to definitively choose another measure to supplement BMI is not overwhelming from our study. Sum of skinfolds stands out as the variable that decreases the most with increasing activity level and which is also related to systolic blood pressure, fasting insulin and fasting glucose in both genders. Formal skinfold measurement using calipers requires observer training and standardization and is known to be less accurate in overweight and obese subjects (Tanner & Whitehouse 1975, Womersley & Durnin 1977) so may not be suitable for population collection currently, although updated ‘ideal’ skinfold centile charts may well be helpful to enable use of skinfolds in clinical settings. Skinfolds self-assessment of the ‘if your pinch is more than an inch’ type may be a good option for population health campaigns. Body fat percentage from bioimpedance picked up on social class and ethnic differences (genders combined) but these were better demonstrated by changes in waist: hip ratio and subscapular: triceps ratio respectively.

Considering central measures of adiposity, waist circumference may be a useful measure, being more closely associated with both systolic blood pressure and fasting insulin in both genders than any other measure of central adiposity, and any measure of general adiposity except BMI. Although in adults waist circumference reflects visceral fat and is now used in addition to BMI to delineate ‘high-risk’ adiposity (NICE guideline 43), in our group of ‘normal’ adolescents waist circumference did not ‘add’ to information from BMI in terms of describing differences seen and was closely related to the markers of general adiposity.

Waist: hip ratio and subscapular: triceps ratio were not highly correlated to each other and it was not clear that they acted as markers of central adiposity (visceral fat) in adolescents. Subscapular: triceps ratio showed a strong ethnic variation whilst waist: hip ratio did not. Further work has not been done at this point to look at the individual skinfolds to explore these differences further.

#### **8.4 NEED FOR PREVENTION OF ADIPOSITY**

Over the last 40 or so years increasing adiposity has been noted in populations in Western, developed societies and in many other parts of the world; the increase in children appears particularly marked. In the Health Survey for England (children aged 2-15 years) in 1995 11% of boys and 12% of girls were obese, rising to 19% of boys and 18% of girls by 2004 (source BHF statistics website). The rise in adiposity was also confirmed by other studies in the UK, including in Ten Towns 13-16 year olds in this study, of whom about 25% were overweight and 10% obese. There is an increasing health burden from these excessive levels of adiposity. Current time trends both in the UK and other settings show that rising levels of obesity are being accompanied by marked increases in risks of type II diabetes, which is occurring at increasingly younger ages (Ehtisham et al, 2000, Pinhas-Hamel et al, 2005). Childhood adiposity also has strong short-term associations with less favourable levels of cardiovascular risk factors (higher mean blood pressure, raised total and LDL cholesterol, triglycerides and low HDL levels) (Freedman et al, 1999, Morrison et al, 1999a, 1999b, Maffei et al, 2001, Berenson et al, 1998) and with diminished arterial distensibility (Whincup et al, 2005). Longer term studies suggest that over 60% of overweight adolescents remain overweight as adults (Gortmaker et al, 1993, Guo et al, 1994). The persistence of excessive adiposity between childhood and adult life is particularly strong when the excess is severe, present at older ages, is associated with parental obesity (Reilly 2003, review), and in lower social classes (Power et al, 1988). With rising levels of adiposity in adolescents there is obvious potential for an upward spiral in obesity, as these young people become parents themselves.

#### **8.5 STRATEGIES FOR PREVENTION**

It is clear from our findings that adiposity in adolescence is associated with adverse cardiovascular risk, in a graded relationship. This implies that with increasing adiposity

there have been corresponding changes in cardiovascular risk on a population basis that may have substantial effects in terms of cardiovascular disease and stroke in years to come. Following the theories of Rose (Rose 1992) for prevention, either population- or high-risk strategies may be followed.

### **8.5.1 HIGH-RISK STRATEGY**

A high-risk strategy includes identifying those at highest risk and is illustrated by RCPCH and NICE guidelines for individuals, defining the high-risk group as those children whose BMI is outside the expected range on the British BMI charts; for individuals over the 91<sup>st</sup> centile intervention may be offered, over the 98<sup>th</sup> centile investigation for co-morbidities may be considered (Baumer 2007, Reilly et al 2002). The advantage of this approach is that intervention is targeted at those most in need, hopefully preventing further progression to co-morbidities. The disadvantage of this approach is that the definition of obesity introduces an artificial cut-off, whereas in reality the risks associated with adiposity are graded, and an appreciable proportion of the adverse consequences, as in adults (Shaper et al 1997), are likely to occur at average rather than high levels of adiposity. Another disadvantage is that overweight and obese children have to be identified before benefiting from intervention; to fully implement a high-risk strategy population screening needs to take place. Currently there is no screening process for adiposity in UK children as a pre-requisite for screening is availability of effective intervention/ treatment and evidence for this is lacking (Summerbell et al 2003).

### **8.5.2 POPULATION APPROACH**

The alternative strategy for prevention is to use a population approach, attempting to improve the health of the majority. The theory of this is to shift the whole normal distribution of adiposity downwards. The benefit of this approach can be illustrated by Shaper's work on 'the ideal BMI' (Shaper et al, 1997). Although the proportion of people experiencing coronary heart disease is highest in the 'obese' group defined by BMI, the actual number of heart attacks occurring is greatest in the normal-overweight groups, as there are simply so many more people in these groups. Small changes in the population distribution and median could have large impacts on the incidence of disease.

As excess adiposity is fundamentally an imbalance between calorie intake and energy expenditure both of these aspects have to be addressed. Applying this approach to child and adolescent adiposity would involve promotion of physical fitness and healthy eating

through school and media, and also information and opportunities for involvement for parents. It would also involve increased opportunities for physical activity in free time and clarification of the barriers to this. In schools adolescents are a 'captive audience' who can to an extent be encouraged to participate in exercise; the advantages of prescribing exercise in the school setting is that it can be implemented with more uniformity than at home, and hopefully there is safe, accessible space in which to carry it out. However there are disadvantages of over-reliance on this; it puts a preventative health responsibility on teachers which may be appropriate for younger children but becomes more problematic in terms of reluctant teenagers, who can even refuse acute medical attention in hospital if competent. The ideal aim is to change habits for life, in parents and children, altering the causation of shared environment, including food choices and use of leisure time. Intervention focused at the child e.g. school-based may be limited in effect if the family is not involved, but may be a way of introducing alternative choices to the family.

Egger and Swinburn (1997) wrote of obesity being a response to an obesogenic environment; environment they suggest has both macro and micro levels. The micro level is in close proximity to the individual, altering which may change adiposity in that individual, however it is on the macro or wider population scale that the influences are found which shape the prevalence of obesity in the population as a whole. An aspect of the 'macro' environment that has changed dramatically over the last few years is internet-based social interaction, shopping, work. Although these changes were not highly developed at the time of study of the present cohort, this change may accelerate further; fears about safety can lead parents to prefer children at home social networking and playing on the computer to being outside; creation of safe real social space for teenagers should be a priority. Parental and child attitudes to 'healthy living' need to be understood to enable appropriate information campaigns; immediate fears of harm from human violence and traffic accidents may justifiably outweigh concern about morbidity in several decades' time. Currently the concern about climate change may have both positive and negative health effects; positive if results in reduced consumption (of food and petrol), negative if engenders despair ('eat, drink and be merry, for tomorrow we die') in adolescents facing an uncertain future and feeling helpless to modify it. The constant negative press about young people in Britain is not helpful in this regard; fuelling a raft of self-destructive behaviours.

Social and ethnic differences seen in the study children demonstrate the development of high-risk groups along the ethnic and social class lines seen in adults. Particular intervention may be justified to educate and help prevent South Asian children becoming overweight and obese, as their tendency to insulin resistance (Whincup et al, 2005) makes them more likely to experience adverse cardiovascular effects at lower levels of adiposity than white children. Breast feeding can not be advocated as a major preventative measure against adiposity; the strength of confounding implies that other factors (including social class and maternal BMI) are playing a much more important role as determinants.

## **8.6 SUGGESTIONS FOR FURTHER RESEARCH**

### **8.6.1 MEASURES OF ADIPOSITY IN CHILDHOOD**

#### **8.6.1.1 Measures of adiposity in childhood, using the ten towns resource**

Further work might extend the study of adiposity measures, particularly to identify a valid marker of central adiposity, which has not so far been possible. Waist: height ratio has been proposed as an alternative measure of central adiposity in adolescence (Taylor et al 2000, Savva et al 2000) and could be explored in the existing Ten Towns data. It would also be possible to do more work on individual skinfolds, looking in more detail at relation to cardiovascular risk factors and ethnic differences. It would also be possible to derive some normal ranges for skinfolds in white 13-15 year olds; this could be also completed for South Asian children but numbers in the study are limited

Further work could be done to examine adiposity measures with cardiovascular risk factors using the longitudinal aspects of the study (from age 5-7), to compare key measures of adiposity, including changes in adiposity, at different ages and their relations to changes in cardiovascular risk changes.

#### **8.6.1.2 Measures of adiposity in childhood, using additional data**

Thigh or calf skinfold measurements improve estimates of body fat percent from skinfolds in young adults (Eston et al 2005) use of these skinfolds and their relationship to cardiovascular risk factors could also be explored.

### **8.6.2 DETERMINANTS AND CORRELATES OF ADIPOSITY**

Geographic differences were observed in the study children but not clearly in a high-low adult cardiovascular town pattern. This may be due to the development of new patterns or locations of high-risk for adiposity (eg urban areas, areas of socio-economic deprivation HSE 2006) or simply that the cumulative exposure takes longer to show effects on

adiposity than the children's 13-16 years. Recent evidence shows that region of residence is a much more significant influence on adiposity and cardiovascular risk than region of birth (Strachan et al 2007), so any effects (apart from that on height) of region of birth may be transient. The particularly high adiposity seen in Rhondda children is echoed by Strachan et al's findings in non-migrant Welsh adults and also the findings of high adiposity in Welsh children at age 7 in the Millennium Cohort Study (Hawkins et al 2008) and suggests that this population may benefit from further investigation of the causes of adiposity/ barriers to healthier weight. This could be based on qualitative studies in adolescents in Bath or Chelmsford and Rhondda, comparing leisure activities, diet, parental factors etc, with the aim of introducing specific changes in Rhondda and to test their effect on adiposity. This type of study may also help to suggest some of the social class-based determinants of adiposity and reasons for low physical activity levels in adolescent girls in particular.

Further work is needed to clarify the basis for ethnic differences in adiposity measures, including the unexplained findings in subscapular: triceps ratio. The combination of non-invasive adiposity measures of the kind used in this study with direct measures of body fat composition (DXA or MRI scanning) might be particularly valuable.

### **8.6.3 INTERVENTION STUDIES**

There is very little evidence on interventions for population prevention of adiposity in teenagers; a randomized controlled trial in a group of well children, such as another school based study, to investigate changes in adiposity and risk factors after simple lifestyle changes e.g. walking a fixed amount at lunchtime and altering lunch food choices, would be helpful with advocating similar small changes for all. More information on the effects of broader environmental changes, include pedestrianising roads, introducing cycle routes or simply reducing speed limits near schools and assessing effects on both active transport and adiposity of pupils would be valuable.

**APPENDIX A: POSTAL QUESTIONNAIRE TO PARENTS**

**(Reduced to 80% actual size)**



Child's first name

## SCHOOL HEART HEALTH SURVEY

Royal Free Hospital School of Medicine

Dear Parent,

### **School Heart Health Study 1998-9**

Your son/daughter has now been examined by our Research Team. It will be of great help to us in interpreting the test results if you can complete this questionnaire. All questions are relevant to conditions of the heart and lungs, including asthma and other closely related allergic conditions.

To answer the questions, please tick the appropriate box  or write in the space provided. All your answers will be treated in the **strictest confidence** and will **only** be seen by the Research Team. If you have any difficulties in completing this questionnaire, please phone us on 0171 830 2858 and leave your telephone number so that we can call you back and answer your queries. When you have completed the questionnaire, please put it in the envelope provided (no stamp is needed) and post it back to us.

**Thank you very much for your help.**

Yours faithfully,

Dr Peter Whincup  
(Project Leader)

**School Heart Health Study 1998-99**

1.0	What is this child's date of birth ?	<table border="1" style="display: inline-table; vertical-align: middle;"><tr><td> </td><td> </td></tr></table> Day			<table border="1" style="display: inline-table; vertical-align: middle;"><tr><td> </td><td> </td></tr></table> Month			<table border="1" style="display: inline-table; vertical-align: middle;"><tr><td> </td><td> </td></tr></table> Year			
1.1	What is your relationship to this child ?	Mother <input type="checkbox"/> <sub>1</sub> Father <input type="checkbox"/> <sub>2</sub> Other relative <input type="checkbox"/> <sub>3</sub> Guardian (unrelated <input type="checkbox"/> <sub>4</sub> )									
1.2	How much did this child weigh at birth ? ( If you don't know, please do not guess, but tick Not known' )	<table border="1" style="display: inline-table; vertical-align: middle;"><tr><td> </td><td> </td></tr></table> lb			<table border="1" style="display: inline-table; vertical-align: middle;"><tr><td> </td><td> </td></tr></table> oz			OR <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td> </td><td> </td><td> </td></tr></table> kg OR Not known <input type="checkbox"/>			
1.3	Was this child born:	On time, (i.e. within a week of the expected date ) <input type="checkbox"/> Early by a week or more <input type="checkbox"/> Late by a week or more <input type="checkbox"/> Not known <input type="checkbox"/> If EARLY or LATE, by how many weeks <table border="1" style="display: inline-table; vertical-align: middle;"><tr><td> </td><td> </td></tr></table> weeks									
1.4	Was this a multiple birth ? ( i.e. a twin, triplet, etc . )	Yes <input type="checkbox"/>	No <input type="checkbox"/>								
	If YES, was it	Identical twins <input type="checkbox"/>	Non- identical twins <input type="checkbox"/>								
		a triplet <input type="checkbox"/>	other <input type="checkbox"/>								
1.5	How was this child fed in the first 3 months of life ?	Breast fed <input type="checkbox"/>	Bottle fed <input type="checkbox"/>								
		Fed on a mixture of breast and bottle feeds <input type="checkbox"/>									
1.6	If the child was breast fed wholly or partly, for how long was this continued from birth ?	<table border="1" style="display: inline-table; vertical-align: middle;"><tr><td> </td></tr></table> months									

### ***MEDICAL HISTORY***

Has this child ever had one of the following conditions ?

		Yes	No	Age at most recent episode (years)
2.0	Asthma	<input type="checkbox"/>	<input type="checkbox"/>	
2.1	Bronchitis	<input type="checkbox"/>	<input type="checkbox"/>	
2.2	Bronchiolitis	<input type="checkbox"/>	<input type="checkbox"/>	
2.3	Pneumonia	<input type="checkbox"/>	<input type="checkbox"/>	
2.4	Eczema	<input type="checkbox"/>	<input type="checkbox"/>	
2.5	Hay fever	<input type="checkbox"/>	<input type="checkbox"/>	

Does this child regularly take any medications ( tablets, Medicines, Inhalers) for

		Yes	No
2.6	Chest Trouble	<input type="checkbox"/>	<input type="checkbox"/>
2.7	Other problem	<input type="checkbox"/>	<input type="checkbox"/>

If YES, please give details: \_\_\_\_\_

		Yes	No
2.8	Has this child been admitted to hospital for any reason in the past 4 years?	<input type="checkbox"/>	<input type="checkbox"/>

If YES, please give details:

### ***IMMUNIZATION***

Was this child immunized against the following as a pre-school child ?

		Yes	No	Can't remember
3.1	Diphtheria and tetanus	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3.2	Whooping cough	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3.3	Measles	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
3.4	Polio	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

**CHEST TROUBLE****Cough**

4.0 Does this child usually cough : first thing in the morning in winter  
during the day, or at night in winter

Yes	No
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

If the answer to either of these questions is YES,

4.1 Does this child cough like this on most days for as much as three months each year ?

Yes	No
<input type="checkbox"/>	<input type="checkbox"/>

**Phlegm**

4.2 Does this child bring up any phlegm (spit) from the chest :  
first thing in the morning in winter  
during the day, or at night in winter

Yes	No
<input type="checkbox"/>	<input type="checkbox"/>
<input type="checkbox"/>	<input type="checkbox"/>

If the answer to either of these questions is YES

4.3 Does this child bring up phlegm like this on most days for as much as three months each year ?

<input type="checkbox"/>	<input type="checkbox"/>
--------------------------	--------------------------

**Wheeze**

4.4 Does this child's chest ever sound wheezy or whistling ?

<input type="checkbox"/>	<input type="checkbox"/>
--------------------------	--------------------------

4.5 If YES, does this happen on most days or nights ?

<input type="checkbox"/>	<input type="checkbox"/>
--------------------------	--------------------------

**RECENT CHEST TROUBLE**

In the past 12 months,

Yes	No
-----	----

4.6 has this child had wheezing or whistling in the chest?

<input type="checkbox"/>	<input type="checkbox"/>
--------------------------	--------------------------

4.7 has your child's chest sounded wheezy during or after exercise ?

<input type="checkbox"/>	<input type="checkbox"/>
--------------------------	--------------------------

If you have answered NO to both these questions, please turn to question 5.0 over the page.

4.8 How many attacks of wheezing has your child had in the past 12 months ?

None	<input type="checkbox"/> 1
1 to 3	<input type="checkbox"/> 2
4 to 12	<input type="checkbox"/> 3
More than 12	<input type="checkbox"/> 4

4.9 In the past 12 months how often, on average, has your child's sleep been disturbed due to wheezing?

Never woken with wheezing	<input type="checkbox"/> 1
Less than one night per week	<input type="checkbox"/> 2
One or more nights per week	<input type="checkbox"/> 3

4.10 In the past 12 months has wheezing ever been severe enough to limit your child's speech to only one or two words at a time between breaths ?

Yes	No
<input type="checkbox"/>	<input type="checkbox"/>

### **DIETARY HABITS**

5.0 How often does this child eat the following foods ?

(Please tick the appropriate box for each food item)

	1	2	3	4	5	6
	More than once a day	Once a day	Most days	One or two days a week	Less than once a week	Never
Fresh fruit in summer						
Fresh fruit in winter						
Salads in summer						
Salads in winter						
Green vegetables						
Fish (all kinds)						
Poultry (chicken, turkey)						
Red meat (include beef, lamb, pork, ham,bacon)						
Processed meat (include burgers, sausages, pies,						
Cheese						

### **EXERCISE AND OTHER ACTIVITIES**

6.0 Which of the following best describes your child's level of physical activity outside school ?

Tick one box only

Spends all or most leisure time watching television, going to cinema and in other sedentary activities

1

Spends time occasionally in light physical activities (e.g. walking, bicycling, table tennis)

2

Participates in regular sporting activities for up to 3 hours a week (e.g. soccer, swimming, gymnastics, tennis, skating )

3

Participates in regular sporting activities for more than 3 hours a week (e.g. soccer, swimming, gymnastics, tennis, skating )

4

6.1 Compared to other children of the same age and sex, how physically active is your child ?

Tick one box only

Much less active

1

Somewhat less active

2

About average

3

Somewhat more active

4

Much more active

5

**BROTHERS AND SISTERS**

7.0 How many brothers and sisters does this child have in all  
(not counting step brothers & sisters) ?

[ ]

7.1 Please give details of this child's brothers and sisters beginning with the oldest :-

	Oldest brother or sister		Second brother or sister		Third brother or sister		Fourth brother or sister	
<b>Date of birth</b>	____ / ____ /19 ____ Day Month Year		____ / ____ /19 ____ Day Month Year		____ / ____ /19 ____ Day Month Year		____ / ____ /19 ____ Day Month Year	
<b>Sex</b>	Boy <input type="checkbox"/>	Girl <input type="checkbox"/>	Boy <input type="checkbox"/>	Girl <input type="checkbox"/>	Boy <input type="checkbox"/>	Girl <input type="checkbox"/>	Boy <input type="checkbox"/>	Girl <input type="checkbox"/>
<b>Has this child ever had ?</b> <b>Asthma</b>	Yes <input type="checkbox"/>	No <input type="checkbox"/>	Yes <input type="checkbox"/>	No <input type="checkbox"/>	Yes <input type="checkbox"/>	No <input type="checkbox"/>	Yes <input type="checkbox"/>	No <input type="checkbox"/>
<b>Eczema</b>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>					
<b>Hayfever</b>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>					
<b>Birth weight</b>	<input type="text"/> lb <input type="text"/> oz or <input type="text"/> kg	<input type="text"/> lb <input type="text"/> oz or <input type="text"/> kg	<input type="text"/> lb <input type="text"/> oz or <input type="text"/> kg	<input type="text"/> lb <input type="text"/> oz or <input type="text"/> kg	<input type="text"/> lb <input type="text"/> oz or <input type="text"/> kg	<input type="text"/> lb <input type="text"/> oz or <input type="text"/> kg	<input type="text"/> lb <input type="text"/> oz or <input type="text"/> kg	
<b>Was this child born</b>	On time <input type="checkbox"/> 1 Early by a week or more <input type="checkbox"/> 2 Late by a week or more <input type="checkbox"/> 3	On time <input type="checkbox"/> 1 Early by a week or more <input type="checkbox"/> 2 Late by a week or more <input type="checkbox"/> 3	On time <input type="checkbox"/> 1 Early by a week or more <input type="checkbox"/> 2 Late by a week or more <input type="checkbox"/> 3	On time <input type="checkbox"/> 1 Early by a week or more <input type="checkbox"/> 2 Late by a week or more <input type="checkbox"/> 3	On time <input type="checkbox"/> 1 Early by a week or more <input type="checkbox"/> 2 Late by a week or more <input type="checkbox"/> 3	On time <input type="checkbox"/> 1 Early by a week or more <input type="checkbox"/> 2 Late by a week or more <input type="checkbox"/> 3		
<b>If early or late, by how many weeks was this?</b>	<input type="text"/> Weeks	<input type="text"/> Weeks						

If there are more than 4 brother and sisters it would be helpful if you could write the extra details on one of the blank pages at the back of the questionnaire.

**THE QUESTIONS ON THIS PAGE ARE ABOUT THE CHILD'S NATURAL MOTHER.**

8.0	Does she live with the child?	Yes <input type="checkbox"/>	No <input type="checkbox"/>			
8.1	What was her country of birth ?					
8.2	To which of the groups listed would she consider she belongs (please tick)?					
	White <input type="checkbox"/>	Black - African <input type="checkbox"/>				
	Indian <input type="checkbox"/>	Black - Caribbean <input type="checkbox"/>				
	Pakistani <input type="checkbox"/>	Chinese <input type="checkbox"/>				
	Bangladeshi <input type="checkbox"/>	Japanese <input type="checkbox"/>				
8.3	How tall is she ?	<input type="text"/> Feet	<input type="text"/> Inches	or	<input type="text"/> Metres	<input type="text"/> cm
8.4	How much does she weigh ?	<input type="text"/> Stones	<input type="text"/> Pounds	or	<input type="text"/> Kilos	
8.5	Has she ever been told by a doctor that she has (or has had):	Please tick any relevant			Please tick any relevant	
	Angina	<input type="checkbox"/>	High blood cholesterol	<input type="checkbox"/>		
	Heart attack (or myocardial infarction or coronary thrombosis)	<input type="checkbox"/>	Peptic ulcer (includes duodenal ulcer)	<input type="checkbox"/>		
	Other heart trouble	<input type="checkbox"/>	Bronchitis	<input type="checkbox"/>		
	(If YES, please give details) _____	<input type="checkbox"/>	Asthma	<input type="checkbox"/>		
	High blood pressure in pregnancy	<input type="checkbox"/>	Eczema	<input type="checkbox"/>		
	High blood pressure at any other time	<input type="checkbox"/>	Hay fever	<input type="checkbox"/>		
	Stroke	<input type="checkbox"/>	Anxiety or depression	<input type="checkbox"/>		
	Diabetes	<input type="checkbox"/>				
8.6	Has she ever had : her blood pressure measured by a doctor or nurse ?	<input type="checkbox"/>	Yes	<input type="checkbox"/>	No	
	her blood cholesterol measured by a doctor or nurse ?	<input type="checkbox"/>		<input type="checkbox"/>		

THE QUESTIONS ON THIS PAGE ARE ABOUT THE **CHILD'S MOTHER OR FEMALE GUARDIAN WHO NOW LIVES WITH THE CHILD.**

If no mother or female guardian lives with this child, **please tick this box**  and go straight to question 11.0 over the page.

**SMOKING HABITS OF MOTHER OR FEMALE GUARDIAN WHO NOW LIVES WITH THE CHILD**

		Yes	No
9.0	Has she ever smoked cigarettes regularly ?(at least 1 cigarette / day for 12 months)	<input type="checkbox"/>	<input type="checkbox"/>
If YES,			
9.1	How many cigarettes (if any) does she usually smoke each day at present?	<input type="text"/>	per day
9.2	How many of these are smoked at home ?	<input type="text"/>	per day
9.3	If she used to smoke cigarettes, when did she stop?	In the past year <input type="checkbox"/> 1-2 years ago <input type="checkbox"/> 3-4 years ago <input type="checkbox"/> 5 or more years ago <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>
9.4	How many cigarettes per day did she smoke before she gave up?	<input type="text"/>	per day
9.5	Did she smoke cigarettes during pregnancy while she was expecting this child ?	<input type="checkbox"/>	<input type="checkbox"/>
IF YES, how many cigarettes a day was she smoking at the time ?		<input type="text"/>	per day

**OCCUPATION OF MOTHER OR FEMALE GUARDIAN WHO NOW LIVES WITH THE CHILD**

10.0	Is she at present :	housewife <input type="checkbox"/> in full-time paid employment <input type="checkbox"/> in part-time paid employment <input type="checkbox"/> Unemployed / seeking work <input type="checkbox"/> other <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>
(Please give details )			
Please answer the following questions about her present (or most recent) employment:-			
10.1	What type of firm or organisation does she (did she) work in, that is, what does her firm make or do?	<hr/>	
10.2	What job does she (did she) actually do?	<hr/>	
10.3	Did she need a particular qualification or training to obtain this job? If so, please say what:	<input type="checkbox"/>	<input type="checkbox"/>
10.4	Is she (was she):	A manager working for an employer ? <input type="checkbox"/> Working for an employer ? <input type="checkbox"/> A foreman or supervisor working for an employer ? <input type="checkbox"/> Self employed not employing others ? <input type="checkbox"/> Self employed employing others ? <input type="checkbox"/>	<input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/> <input type="checkbox"/>
10.5	How old was she when her full-time education (school, college or university) ended ?	<input type="text"/>	

**THE QUESTIONS ON THIS PAGE ARE ABOUT THE CHILD'S NATURAL FATHER**

11.0 Does the child's natural father currently live with the child ? Yes  No

11.1 What was his country of birth ? \_\_\_\_\_

11.2 To which of the groups listed would he consider he belongs (please tick)?

White	<input type="checkbox"/>	Black - African	<input type="checkbox"/>
Indian	<input type="checkbox"/>	Black - Caribbean	<input type="checkbox"/>
Pakistani	<input type="checkbox"/>	Chinese	<input type="checkbox"/>
Bangladeshi	<input type="checkbox"/>	Japanese	<input type="checkbox"/>

other - please give details \_\_\_\_\_

11.3 How tall is he ? 

<input type="checkbox"/>	<input type="checkbox"/>	or	<input type="checkbox"/>	<input type="checkbox"/>
Feet	Inches		Metres	cm

11.4 How much does he weigh ? 

<input type="checkbox"/>	<input type="checkbox"/>	or	<input type="checkbox"/>
Stones	Pounds		Kilos

11.5 Has he ever been told by a doctor that he has (or has had):

Please tick any relevant

Please tick any relevant

Angina	<input type="checkbox"/>	High blood cholesterol	<input type="checkbox"/>
Heart attack (or myocardial infarction or coronary thrombosis)	<input type="checkbox"/>	Peptic ulcer (includes duodenal ulcer)	<input type="checkbox"/>
Other heart trouble (if YES, please give details) _____	<input type="checkbox"/>	Bronchitis	<input type="checkbox"/>
High blood pressure	<input type="checkbox"/>	Asthma	<input type="checkbox"/>
Stroke	<input type="checkbox"/>	Eczema	<input type="checkbox"/>
Diabetes	<input type="checkbox"/>	Hay fever	<input type="checkbox"/>
		Anxiety or depression	<input type="checkbox"/>

11.6 Has he ever had : his blood pressure measured by a doctor or nurse ? Yes  No   
his blood cholesterol measured by a doctor or nurse ?

THE QUESTIONS ON THIS PAGE ARE ABOUT THE **CHILD'S FATHER OR MALE GUARDIAN WHO NOW LIVES WITH THE CHILD**.

If no father or male guardian lives with this child **please tick this box**  and go straight to question 14.0 over the page.

**SMOKING HABITS OF CHILD'S FATHER OR MALE GUARDIAN WHO NOW LIVES WITH THE CHILD**

		Yes	No
12.0	Has he ever smoked cigarettes regularly ? (at least 1 cigarette / day for 12 months)	<input type="checkbox"/>	<input type="checkbox"/>
12.1	If YES, how many cigarettes (if any) does he usually smoke each day at present?	<input type="text"/>	per day
12.2	How many of these are smoked at home?	<input type="text"/>	per day
12.3	If he used to smoke cigarettes, when did he stop?	In the past year	
		<input type="checkbox"/> 1-2 years ago	<input type="checkbox"/> 2
		<input type="checkbox"/> 3-4 years ago	<input type="checkbox"/> 3
		<input type="checkbox"/> 5 or more years ago	<input type="checkbox"/> 4
12.4	How many cigarettes per day did he smoke before he gave up ?	<input type="text"/>	per day
12.5	Does he smoke regularly (ie once a week or more)	a pipe ? <input type="checkbox"/>	<input type="checkbox"/>
		cigars ? <input type="checkbox"/>	<input type="checkbox"/>
12.6	Did he smoke cigarettes around the time that the child was born ?	<input type="checkbox"/>	<input type="checkbox"/>
IF YES, how many cigarettes a day was he smoking at the time ? <input type="text"/> per day			

**OCCUPATION OF CHILD'S FATHER OR MALE GUARDIAN WHO NOW LIVES WITH THE CHILD**

13.0	Is he at present :	in full-time paid employment <input type="checkbox"/> 1
		in part-time paid employment <input type="checkbox"/> 2
		Unemployed / seeking work <input type="checkbox"/> 3
	other _____	<input type="checkbox"/> 4 (Please give details )
Please answer the following questions about his present (or most recent) employment:-		
13.1	What type of firm or organisation does he (did he) work in, that is, what does his firm make or do?	<hr/>
13.2	What job does he (did he) actually do?	<hr/>
13.3	Did he need a particular qualification or training to obtain this job? If so, please say what:	<hr/>
13.4	Is he (was he):	Manager working for an employer <input type="checkbox"/> 1
		Working for an employer <input type="checkbox"/> 2
		A foreman or supervisor working for an employer <input type="checkbox"/> 3
		Self employed not employing others <input type="checkbox"/> 4
		Self employed employing others <input type="checkbox"/> 5
13.5	How old was he when his full-time education (school, college or university) ended ?	<input type="text"/>

14.0 **THE HEALTH OF THE CHILD'S NATURAL GRANDPARENTS**

		Maternal grandmother	Maternal grandfather	Paternal grandmother	Paternal grandfather
Are they alive or have they died ?  How old are they ? or How old were they when they died ?	Still Alive	<input type="checkbox"/> 1	<input type="checkbox"/> 1	<input type="checkbox"/> 1	<input type="checkbox"/> 1
	Dead	<input type="checkbox"/> 2	<input type="checkbox"/> 2	<input type="checkbox"/> 2	<input type="checkbox"/> 2
	Don't Know	<input type="checkbox"/> 3	<input type="checkbox"/> 3	<input type="checkbox"/> 3	<input type="checkbox"/> 3
	Age (years)	<input type="text"/>	<input type="text"/>	<input type="text"/>	<input type="text"/>
Do/ did they ever have any of these conditions (please tick as many as necessary)	Heart Attack	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Angina	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Other Heart trouble	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Stroke	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	High blood pressure	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Diabetes	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Heart Attack	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Angina	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Other Heart trouble	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
	Stroke	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
High blood pressure	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
Diabetes	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
Other conditions	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	
Don't know	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>	

**HOME CIRCUMSTANCES**

15.0	How many rooms does your accommodation contain ? (Do not count bathrooms, toilets and kitchens)	<input type="text"/>
15.1	How many people, <b>including the child in our survey</b> , live in your household altogether ?	<input type="text"/>
15.2	How many of these people smoke cigarettes at home ?	<input type="text"/>
15.3	Your accommodation	(Please tick one box)
	Are you :-	an owner occupier <input type="checkbox"/> 1 renting from the local authority <input type="checkbox"/> 2 renting privately <input type="checkbox"/> 3 other (please give details) _____ <input type="checkbox"/> 4
15.4	Do you have a car available for the use of your family ?	Yes <input type="checkbox"/> No <input type="checkbox"/>

### **FOR THE ATTENTION OF THE CHILD'S MOTHER**

Thank you for completing this questionnaire. We would like to seek your help in one other way. Recent research has suggested that what happens in pregnancy, childbirth and early infancy may affect blood pressure and other aspects of 'heart health' through childhood and perhaps into adult life. We are therefore asking for your permission to look at the case-notes relating to your child's birth and early growth, and to monitor your child's health in the future.

The information which we would seek would include only technical details relating to the pregnancy and the size of the baby and placenta at birth, together with details of growth after birth. The information obtained, as with all other details of the study, would be treated in strict confidence. The arrangements made to ensure this have been approved by your local Health Authority Ethical Committee.

We hope you will agree to allow us to obtain this extra information. If you do, please sign and complete the details below.

16.0 **Your signature:** \_\_\_\_\_

16.1 **Your name and address at the time your child was born:-**

First Name(s) \_\_\_\_\_ Surname \_\_\_\_\_

Address \_\_\_\_\_  
\_\_\_\_\_  
\_\_\_\_\_

16.2 **Your Date of Birth** \_\_\_\_\_ / \_\_\_\_\_ / 19 \_\_\_\_\_

16.3 **The name of the hospital where the child was born:**

Hospital Name \_\_\_\_\_

Town \_\_\_\_\_

County \_\_\_\_\_

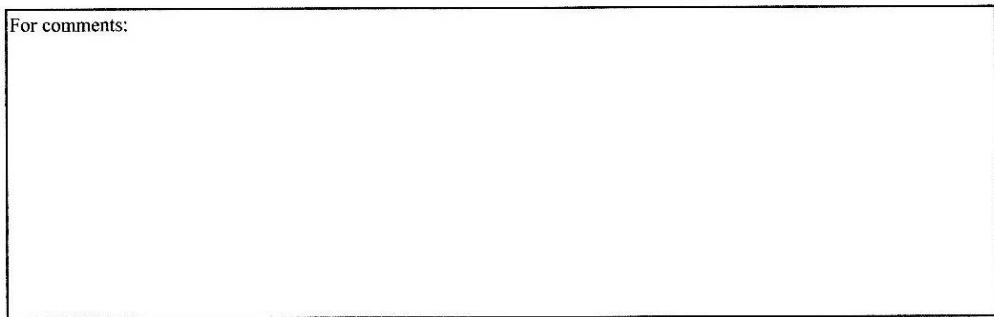
16.4 If possible please provide **Your child's NHS Number** which can be found on your child's birth certificate and medical card.

Thank you for your help with this questionnaire.

Please check that you have answered all relevant questions and  
return the questionnaire to us in the envelope provided.

No stamp is required.

For comments:

A large, empty rectangular box with a thin black border, intended for comments or feedback.

Return to:

British Regional Heart Study  
Department of Primary Care & Population Sciences  
Royal Free Hospital School of Medicine  
Rowland Hill Street  
Hampstead  
LONDON NW3 2PF

## **APPENDIX B: CHILD QUESTIONNAIRE**

**(Reduced to 80% actual size)**

## SCHOOL HEART HEALTH SURVEY 1998-99

PLEASE ANSWER THESE QUESTIONS AS FULLY AS YOU CAN; THE ANSWERS YOU GIVE WILL BE TREATED AS CONFIDENTIAL AND WILL BE NOT BE TOLD TO SCHOOL OR YOUR PARENTS.

1.0	What is your date of birth?	<table><tr><td><input type="text"/></td><td><input type="text"/></td><td><input type="text"/></td></tr><tr><td>Day</td><td>Month</td><td>Year</td></tr></table>	<input type="text"/>	<input type="text"/>	<input type="text"/>	Day	Month	Year	YES	NO
<input type="text"/>	<input type="text"/>	<input type="text"/>								
Day	Month	Year								
1.1	In the last two weeks, have you had a cold or flu?	<input type="checkbox"/> <input type="checkbox"/>								

### MEDICINES

1.2	Do you use an inhaler for your chest ?	YES	NO
	If Yes, please give the name of your inhaler(s) _____		
	When did you last use your inhaler(s) ? _____		
1.3	Do you regularly take any other medications ( tablets, injections, etc)	YES	NO
	IF Yes, please give details _____		

### YOUR HEALTH

#### WHEEZE

2.0	Have you <u>ever</u> had wheezing or whistling in the chest at any time in the past ?	YES	NO
2.1	Have you had wheezing or whistling in the chest <u>in the last 12 months</u> ?	YES	NO
2.2	<u>In the last 12 months</u> , has your chest sounded wheezy during or after exercise?	YES	NO

If you have not had wheezing /whistling in the chest in the last 12 months, please go to question 3.0

2.3	How many attacks of wheezing have you had <u>in the last 12 months</u> ?	(Tick one box)	
	None	<input type="checkbox"/>	
	1-3	<input type="checkbox"/>	
	4-12	<input type="checkbox"/>	
	More than 12	<input type="checkbox"/>	
2.4	<u>In the last 12 months</u> how often, on average, has your sleep been disturbed due to wheezing?	(Tick one box)	
	Never woken with wheezing	<input type="checkbox"/>	
	Less than one night per week	<input type="checkbox"/>	
	One or more nights per week	<input type="checkbox"/>	
2.5	<u>In the last 12 months</u> , has wheezing ever been severe enough to limit your speech to only one or two words at a time between breaths?	YES	NO

#### HAY FEVER

*All questions are about problems that occur when you do not have a cold or the flu.*

	YES	NO
3.0 Have you <u>ever</u> had a problem with sneezing, or a runny, or blocked nose when you <u>did not</u> have a cold or the flu?	<input type="checkbox"/>	<input type="checkbox"/>

**If No, please go to question 4.0**

3.1 In the past 12 months, have you had a problem with sneezing, or a runny, or blocked nose when you did not have a cold or the flu?

**If No, please go to question 4.0**

3.2 In the past 12 months, has this nose problem been accompanied by itchy, watery eyes?

3.3 In which of the past 12 months did this nose problem occur? (Please tick any which apply)

January	<input type="checkbox"/>	July	<input type="checkbox"/>
February	<input type="checkbox"/>	August	<input type="checkbox"/>
March	<input type="checkbox"/>	September	<input type="checkbox"/>
April	<input type="checkbox"/>	October	<input type="checkbox"/>
May	<input type="checkbox"/>	November	<input type="checkbox"/>
June	<input type="checkbox"/>	December	<input type="checkbox"/>

3.4 In the past 12 months, how much did this nose problem interfere with your daily activities ?

Not at all	<input type="checkbox"/>
A little	<input type="checkbox"/> (Tick one box)
A moderate amount	<input type="checkbox"/>
A lot	<input type="checkbox"/>

#### **ECZEMA**

	YES	NO
4.0 Have you <u>ever</u> had an itchy rash which was coming and going for at least 6 months?	<input type="checkbox"/>	<input type="checkbox"/>

4.1 Have you had this itchy rash at any time in the last 12 months?

**If NO to both these questions, please go to question 5.0**

4.2 Has this itchy rash at any time affected any of the following places :

the folds of the elbows, behind the knees,  
in front of the ankles, under the buttocks,  
or around the neck, ears or eyes?

4.3 Has the rash cleared completely at any time during the last 12 months?

4.4 In the last 12 months, how often, on average, have you been kept awake at night by this itchy rash? (Tick one box)

Never in the last 12 months	<input type="checkbox"/>
Less than 1 night per week	<input type="checkbox"/>
1 or more nights per week	<input type="checkbox"/>

#### **ALLERGIES**

5.0 Do you have any allergies? **YES** **NO**

5.1 If YES, what are you allergic to?

		YES	NO	Age at most recent episode (in years)
5.2	Have you <u>ever</u> had:	Asthma	<input type="checkbox"/>	<input type="checkbox"/>
		Hayfever	<input type="checkbox"/>	<input type="checkbox"/>
		Eczema	<input type="checkbox"/>	<input type="checkbox"/>

### **CIGARETTE SMOKING**

6.0 Do you smoke cigarettes at all nowadays? **YESNO**

6.1 Now read all the following statements carefully and tick the box next to the one which best describes you.

I usually smoke more than six cigarettes a week

I usually smoke between one and six cigarettes a week

I sometimes smoke cigarettes now but I don't smoke as many as one a week

I used to smoke sometimes but I never smoke a cigarette now

I have only ever tried smoking once

I have never smoked

6.2 Please read these different statements carefully and tick the box next to the one which best describes you.

I do sometimes smoke cigarettes

I did once have a puff or two of a cigarette, but I never smoke now

I have never tried smoking a cigarette, not even a puff or two

If you **ever** smoked a cigarette, please answer these questions.

6.3 How old were you when you first tried smoking a cigarette, even if it was only a puff or two?  Years old

***CIGARETTE SMOKING (continued)***

If you have smoked **at least one cigarette in the last month**, please answer these questions.

6.5 How easy or difficult would you find it to go without smoking for as long as a week?

Very difficult	<input type="checkbox"/>
Fairly difficult	<input type="checkbox"/>
Fairly easy	<input type="checkbox"/>
Very easy	<input type="checkbox"/>

6.6 Would you like to give up smoking altogether?

YES	<input type="checkbox"/>
NO	<input type="checkbox"/>
Don't know	<input type="checkbox"/>

6.7 Have you ever tried to give up smoking?

YES	<input type="checkbox"/>
NO	<input type="checkbox"/>

6.8 How soon after waking do you usually smoke your first cigarette of the day?

In the first 5 minutes	<input type="checkbox"/>
Between 5- 15 minutes	<input type="checkbox"/>
Between 15-30 minutes	<input type="checkbox"/>
Between 30 minutes to 1 hour	<input type="checkbox"/>
After 1-2 hours	<input type="checkbox"/>
After 2 hours or more	<input type="checkbox"/>

6.9 If you have smoked at least one cigarette in the last day, how many hours ago was the last one?

<input type="checkbox"/>	<input type="checkbox"/>
--------------------------	--------------------------

 Hours

#### **ALCOHOLIC DRINKS**

7.0 Have you ever had a proper alcoholic drink- a whole drink, not just a sip?  
Please don't count drinks labelled low alcohol

YES	<input type="checkbox"/>
NO	<input type="checkbox"/>

7.1 How often do you usually have an alcoholic drink?

Almost every day	<input type="checkbox"/>
About twice a week	<input type="checkbox"/>
About once a week	<input type="checkbox"/>
About once a fortnight	<input type="checkbox"/>
About once a month	<input type="checkbox"/>
Only a few times a year	<input type="checkbox"/>
I never drink alcohol now	<input type="checkbox"/>

### **PHYSICAL ACTIVITY**

8.0 How do you normally travel to school ?

by bus or train	<input type="checkbox"/>
by bicycle	<input type="checkbox"/>
by car	<input type="checkbox"/>
on foot	<input type="checkbox"/>

8.1 How do you normally travel home from school ?

by bus or train	<input type="checkbox"/>
by bicycle	<input type="checkbox"/>
by car	<input type="checkbox"/>
on foot	<input type="checkbox"/>

8.2 What do you usually do at morning break ?

sit down (talking reading etc )	<input type="checkbox"/>
stand or walk around	<input type="checkbox"/>
run around playing games	<input type="checkbox"/>

8.3 What do you usually do at lunch break, apart from eating lunch?

sit down (talking reading etc )	<input type="checkbox"/>
stand or walk around	<input type="checkbox"/>
run around playing games	<input type="checkbox"/>

8.4 During your PE and games classes, how often do you get out of breath ?

Never or hardly ever	<input type="checkbox"/>
Sometimes	<input type="checkbox"/>
Often	<input type="checkbox"/>
Always	<input type="checkbox"/>
I don't do PE/ games-doesn't apply	<input type="checkbox"/>

8.5 How many days a week (if any) do you usually stay behind at school for sports ?

None	<input type="checkbox"/>
Once or twice a week	<input type="checkbox"/>
More often	<input type="checkbox"/>

8.6 How many evenings a week (if any) do you usually take part in sports or other physical activities

None	<input type="checkbox"/>
Once or twice a week	<input type="checkbox"/>
More often	<input type="checkbox"/>

8.7 Do you usually take part in sports or other physical activities at the weekend ?

Yes	<input type="checkbox"/>
No	<input type="checkbox"/>

8.8 If YES, how many hours on average do you spend on this each weekend ?

<input type="text"/>	<input type="text"/>	Hours
----------------------	----------------------	-------

### 8.9 EXERCISE IN THE LAST SEVEN DAYS

We would like to know about all the exercise, both in and out of school, which you have taken in the last seven days (up to yesterday). Please tell us how often you did each activity, for how long and whether it made you out of breath.

ACTIVITY	How many times in the last seven days? (if none leave blank)	About how long did you spend on this altogether in the last seven days?		Did you get out of breath? Please tick the correct answer		
		Hours	Minutes	No	A bit	A lot
Athletics				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Basketball				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Cricket				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Cycling				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Football				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Gymnastics				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Hockey				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Jogging / running				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Judo/ Karate/ Boxing				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Netball				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Rugby				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Squash				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Swimming				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Tennis				<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Other (Please say what)	_____			<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Other (Please say what)	_____			<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

### 8.10 Which one of the following statements describes you best ?

All or most of my free time is spent doing things which involve little physical effort (eg. doing homework, talking to friends, watching TV)

Once or twice a week I do things in my free time which involve some physical effort (eg. walking, cycling, table tennis)

I quite often (4-6 times a week) do things in my free time which involve physical exercise

I very often (7 times a week or more) do things in my free time which involve physical exercise

**(EXERCISE continued)**

8.11 Compared to other pupils of your own age and sex, would you say that you are :

Much less active	<input type="checkbox"/>
a bit less active	<input type="checkbox"/>
about average	<input type="checkbox"/>
a bit more active	<input type="checkbox"/>
much more active	<input type="checkbox"/>

**TELEVISION, VIDEO AND COMPUTER GAMES**

9.0 How many hours each day do you usually spend doing these things altogether ?

None	<input type="checkbox"/>
an hour or less	<input type="checkbox"/>
1-2 hours	<input type="checkbox"/>
2-3 hours	<input type="checkbox"/>
more than 3 hours	<input type="checkbox"/>

**SCHOOL**

10.0 Do you usually:- Eat a packed lunch at school   
Eat a school meal   
Other (please give details)

10.1 How long do you usually spend each day on school homework ?

None	<input type="checkbox"/>
1/2 hour or less	<input type="checkbox"/>
Between 1/2 to 1 hour	<input type="checkbox"/>
More than 1 hour	<input type="checkbox"/>

10.2 How well are you doing at school? Not very well   
Average   
Very well

10.3 Do you think you will take any GCSE's before you leave school?

YES	<input type="checkbox"/>
NO	<input type="checkbox"/>
Don't know	<input type="checkbox"/>

10.4 Do you expect to get 5 or more passes at Grade A, B or C?

YES	<input type="checkbox"/>
NO	<input type="checkbox"/>
Don't know	<input type="checkbox"/>

10.5 Do you think you will continue in full-time education after the end of Year 11?

YES	<input type="checkbox"/>
NO	<input type="checkbox"/>
Don't know	<input type="checkbox"/>

### **HOME & FAMILY**

11.0 Who do you live at home with at present ? (where you can, please say how they are related to you e.g. mother, stepfather, brother).

---

11.1 Do any of these people smoke cigarettes at present ? **YES** **NO**

**If YES**, please say which people smoke

---

11.2 When were you last in a room where someone else was smoking ?

Less than an hour ago   
1-4 hours ago   
5-8 hours ago   
More than 8 hours ago

11.3 Do you have any pets at home? **YES** **NO**

**If YES**, please give details?

---

11.4 Does your family have any of the following? **YES** **NO**  
 a car  If Yes, how many \_\_\_\_\_  
 a dishwasher   
 a home computer   
(Please don't count video games)

11.5 Thinking about the house or flat your family live in at the moment, do your family own it or is it rented?

Own   
Rented   
Don't know

11.6 If you live with your mother, stepmother or foster mother and she has a job, please tell us what job she does

---

11.7 If you live with your father, stepfather or foster father and he has a job, please tell us what job he does

---

11.8 How much money of your own do you have most weeks to spend as you like?

Nothing   
Less than £1 a week   
£1 or more but less than £5   
£5 or more but less than £10   
£10 or more but less than £20   
£20 or more a week

11.8 Does this money come from:- pocket money   
paid work outside school hours   
or somewhere else

12.0 **STRENGTHS AND DIFFICULTIES**

---

	Not True	Somewha t True	Certainly True
I am considerate of other people's feelings	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am restless. I cannot stay still for long	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I get a lot of headaches, stomach-aches or sickness	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I usually share with others (food, games, pens etc.)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I get very angry and often lose my temper	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am rather solitary. I usually play alone or keep to myself	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I usually do as I am told	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I worry a lot	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am helpful if someone is hurt, upset or feeling ill	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am constantly fidgeting or squirming	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I have at least one good friend	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I fight a lot. I can make other people do what I want	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am very often unhappy, down hearted or tearful	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Other people my age generally like me	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am easily distracted, I find it difficult to concentrate	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am nervous in new situations. I easily lose confidence	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am kind to younger children	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I am often accused of lying or cheating	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
Other children or young people pick on me or bully me	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I often volunteer to help others (parents, teachers, children)	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I think things out before acting	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I take things that are not mine from home, school or elsewhere	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I get on better with adults than with people my own age	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I have many fears, I am easily scared	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>
I see tasks through to the end. My attention is good.	<input type="checkbox"/>	<input type="checkbox"/>	<input type="checkbox"/>

THANK YOU FOR YOUR HELP WITH THIS QUESTIONNAIRE  
ALL YOUR ANSWERS WILL BE TREATED IN COMPLETE CONFIDENCE

## **APPENDIX C: PUBERTY QUESTIONNAIRE**

**(Reduced to 80% actual size)**

Study Subject No:

School Heart Health Study

Department of Primary Care  
and Population Sciences,  
Royal Free Hospital  
School of Medicine,  
Rowland Hill Street,  
London NW3 2PF.



B O Y S

Self Assessment

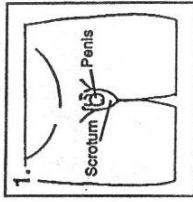
Self Assessment

B O Y S

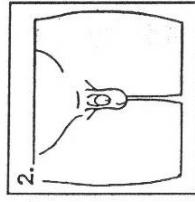
dy Subject No:

**QUESTION 1**

- Please look at the **Penis** and **Scrotum** **only** in these pictures.
- Please put a tick in the box that looks most like you now.

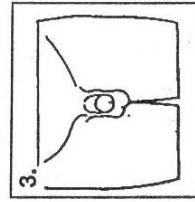


1. Scrotum and Penis  
same size as when you  
were younger.



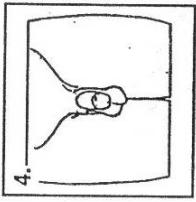
2.

The Scrotum has  
lowered a bit  
and the Penis  
is a little larger.



3.

The Penis is longer  
the Scrotum is  
larger.

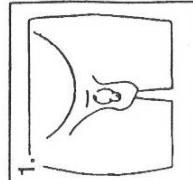


4.

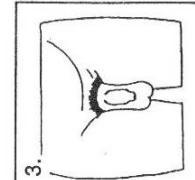
The Penis is longer  
and wider the Scrotum  
is darker and bigger  
than before

**QUESTION 2**

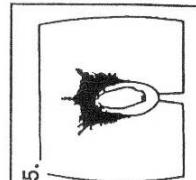
- Please look at the **Public Hair** **only** in these pictures.
- Please put a tick in the box that looks most like you now.



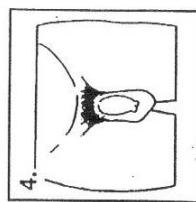
1. No hairs



2. Quite a lot of hair

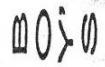


3. The hair has not  
spread over the  
thighs



4. The hair has spread  
over the thighs

**PLEASE ANSWER BOTH  
QUESTION 1 AND  
QUESTION 2**



**Self Assessment**

**Self Assessment**

Please answer these  
questions:

1. Have you started your  
periods yet?

Yes   
No

2. How old were you when  
you had your first period?  
\_\_\_\_\_ years \_\_\_\_\_ months

3. Are your periods regular  
(this means you have  
them every month)?  
Yes  regular  
No  They aren't  
regular

4. Are you on the Pill?  
Yes   
No

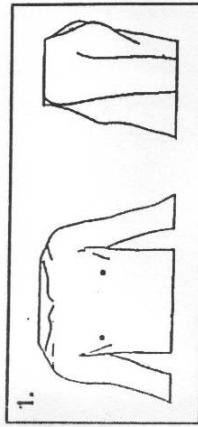
School Heart Health Study

Department of Primary Care  
and Population Sciences,

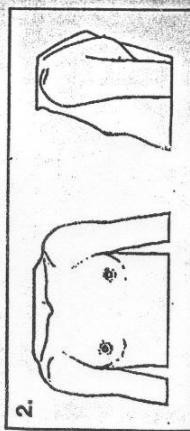
Royal Free Hospital  
School of Medicine,  
Rowland Hill Street,  
London NW3 2PF.



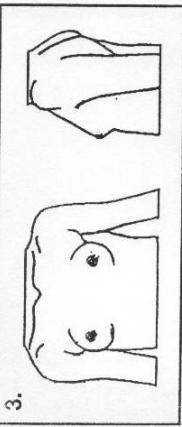
• Please put a tick in the box that looks most like you now....



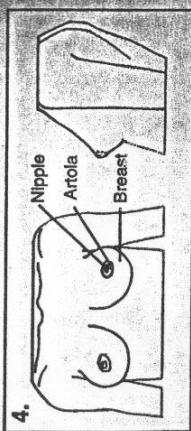
The Breasts are flat.



The Breasts form small rounds.



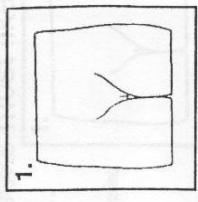
The breasts form larger mounds than in 2.



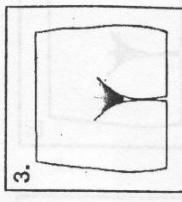
The nipple and the surrounding part (the Areola) make up a mound that sticks up above the breast.

Self Assessment

• Please put a tick in the box that looks most like you now....

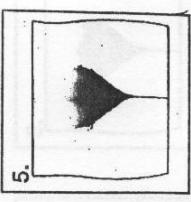


No hairs



Quite a lot of hair

Very little hair



The hair has spread over the thighs

The hair has not spread over the thighs

PLEASE TURN OVER →

Self Assessment

## **APPENDIX D: CO-AUTHOR PAPERS FROM TEN TOWNS STUDY**

### **List of Co-author papers from ten towns study**

1. Owen CG, Whincup PH, Odoki K, Gilg JA, Cook DG. Infant feeding and blood cholesterol: a study in adolescents and a systematic review. *Pediatrics* 2002;110:597-608.
2. Owen CG, Whincup PH, Odoki K, Gilg JA, Cook DG. Birth weight and blood cholesterol level: a study in adolescents and systematic review. *Pediatrics* 2003;111:1081-9.
3. Taylor SJ, Whincup PH, Hindmarsh PC, Lampe F, Odoki K, Cook DG. Performance of a new pubertal self-assessment questionnaire: a preliminary study. *Paediatr.Perinat.Epidemiol.* 2001;15:88-94.
4. Whincup PH, Gilg JA, Odoki K, Taylor SJ, Cook DG. Age of menarche in contemporary British teenagers: survey of girls born between 1982 and 1986. *BMJ* 2001;322:1095-6.
5. Whincup PH, Gilg JA, Owen CG, Odoki K, Alberti KG, Cook DG. British South Asians aged 13-16 years have higher fasting glucose and insulin levels than Europeans. *Diabet.Med.* 2005;22:1275-7.

## **REFERENCES**

Agyemang C, Bhopal RS. Is the blood pressure of South Asian adults in the UK higher or lower than that in European white adults? A review of cross-sectional data. *J Hum Hypertens* 2002; **16**:739-751.

Allison DB, Kaprio J, Korkeila M, Neale MC, Hayakawa K. The heritability of body mass index among an international sample of monozygotic twins reared apart. *Int J Ob* 1996; **20**:501-506.

American Diabetes Association. Type 2 Diabetes in Children and Adolescents. *Diabetes Care* 2000; **23**:381-9.

Andersen L, Dinesen B, Jorgensen PN et al. Enzyme immunoassay for intact human insulin in serum or plasma. *Clin Chemistry* 1993; **39**: 578-582.

Andersen LB, Harro M, Sardinha LB, Froberg K, Ekelund U, Brage S, Anderssen SA. Physical activity and clustered cardiovascular risk in children: a cross-sectional study (The European Youth Heart Study). *Lancet* 2006; **368**:299-304.

Andreasen CH et al. Low physical activity accentuates the effect of the FTO rs9939609 polymorphism on body fat accumulation. *Diabetes* 2008; **57**:95-101.

Allison DB, Paultre F, Heymsfield SB, Pi-Sunyer FX. Is the intra-uterine period really a critical period for the development of obesity? *Int J Obes* 1995; **19**:397-402.

Ara I, Moreno LA, Leiva MT, Gutin B, Casajús JA. Adiposity, physical activity, and physical fitness among children from Aragón, Spain. *Obesity (Silver Spring)* 2007; **15**:1918-24.

Arenz S, Ruckerl R, Koletzko B, von KR. Breast-feeding and childhood obesity--a systematic review. *Int J Obes Relat Metab Disord* 2004; **28**(10):1247-1256.

Baird J, Fisher D, Lucas P, Kleijnen J, Roberts H, Law C. Being big or growing fast: systematic review of size and growth in infancy and later obesity. *BMJ* 2005; **331**:929-934.

Baker JL, Olsen LW, Sorensen TI. Childhood body-mass index and the risk of coronary heart disease in adulthood. *N Engl J Med*. 2007;357:2329-37.

Bao W, Srinivasan SR, Wattigney WA, Berenson GS. Persistence of multiple cardiovascular risk clustering related to Syndrome X from childhood to young adulthood. The Bogalusa Heart Study. *Arch Intern Med* 1994;154:1842-1847.

Bao W, Srinivasan SR, Valdez R, Greenlund KJ, Wattigney WA, Berenson GS. Longitudinal changes in cardiovascular risk from childhood to young adulthood in offspring of parents with coronary artery disease: the Bogalusa Heart Study. *JAMA* 1997;278(21):1749-54.

Barker DJP. Mothers, Babies and Health in Later Life. Churchill Livingstone 1998.

Barker M, Robinson S, Osmond C, Barker DJ. Birth weight and body fat distribution in adolescents girls. *Arch Dis Child*. 1997;77:381-3.

Baumer JH. Obesity and overweight: its prevention, identification, assessment and management. *Arch Dis Child Educ Pract Ed* 2007;92:ep92-ep96.

Baumgartner RN, Roche AF, Guo S, Lohman T, Boileau RA, Slaughter MH. Adipose Tissue Distribution: The stability of principal components by sex, ethnicity and maturation stage. *Hum Biol* 1986;58:719-735.

Benfield LL, Fox KR, Peters DM, Blake H et al. Magnetic resonance imaging of abdominal adiposity in a large cohort of British children. *Int J Obes (Lond)* 2008;32(1):91-99.

Ben-Shlomo Y, McCarthy A et al. Immediate postnatal growth is associated with blood pressure in young adulthood. *Hypertension* 2008; 52: 638-644.

Berenson GS, Srinivasan SR, Bao W, Newman WP 3<sup>rd</sup>, Tracy RE, Wattigney WA. Association between multiple cardiovascular risk factors and atherosclerosis in children and young adults. The Bogalusa Heart Study. *NEJM* 1998;338(23):1650-6.

Bhargava SK, Sachdev HS, Fall CDH et al. Relation of serial changes in childhood body-mass index to impaired glucose tolerance in young adulthood. *NEJM* 2004;350:865-75.

Bhopal R et al. Heterogeneity of coronary heart disease risk factors in Indian, Pakistani, Bangladeshi and European origin populations: cross-sectional study. *BMJ* 1999;319:215-220.

Birch LL, Fisher JO. Development of eating behaviors among children and adolescents. *Pediatrics* 1998;101(3 pt 2):539-49.

Blundell JE, King NA. Over-consumption as a cause of weight gain: behavioural-physiological interactions in the control of food intake (appetite). In: Chadwick DJ, Cardew GC, eds. *The origins and consequences of obesity*, Chichester, Wiley, 1996; 138-158. Ciba Foundation symposium 201.

Borecki IB, Rice T, Perusse L, Bouchard C, Rao DC. *Obes Res* 1995;3:1-8.

Brambilla P, Manzoni P, Sironi S, Simone P, Del Maschio A, di Natale B, Chiumello G. Peripheral and abdominal adiposity in childhood obesity. *Int J Obes Relat Metab Disord* 1994;18(12):795-800.

Brambilla P, Bedogni G, Moreno LA, Goran MI, Gutin B, Fox KR, Peters DM, Barbeau P, De Simone M, Pietrobelli A. Crossvalidation of anthropometry against magnetic resonance imaging for the assessment of visceral and subcutaneous adipose tissue in children. *Int J Obes* 2006;30(1):23-30.

British Heart Foundation statistics website: adults data presented from <http://www.heartstats.org/temp/Figsp11.5spweb07.xls>; children's from <http://www.heartstats.org/temp/Figsp11.6spweb07.xls> both accessed 25th April 2008.

Brook CGD. Determination of body composition of children from skinfold measurements. *Arch Dis Child* 1971;46:182-84.

Bundred P, Kitchiner D, Buchan I. Prevalence of overweight and obese children between 1989 and 1998: population based series of cross sectional studies. *BMJ* 2001; **322**:326-328.

Burdette HL, Whitaker RC, Hall WC, Daniels SR. Breastfeeding, introduction of complementary foods, and adiposity at 5 y of age. *Am J Clin Nutr* 2006; **83**(3):550-558.

Burke V, Beilin LJ, Dunbar D. Family lifestyle and parental body mass index as predictors of body mass index in Australian children: a longitudinal study. *Int J Obes Relat Metab Disord* 2001; **25**:147-57.

Burns SP, Desai M, Cohen RD et al. Gluconeogenesis, glucose handling, and structural changes in the liver of the adult offspring of rats partially deprived of protein during pregnancy and lactation. *J Clin Invest* 1997; **100**:1768-1774.

Caprio S, Hyman LD, Limb C, McCarthy S, Lange R, Sherwin RS, Shulman G, Tamborlane WV. Central adiposity and its metabolic correlates in obese adolescent girls. *Am J Physiol* 1995; **269**(Endocrinol Metab 32):E118-E126.

Cecil JE, Tavendale R, Watt P, Hetherington MM, Palmer CN. An obesity-associated FTO gene variant and increased energy intake in children. *N Eng J Med* 2008; **359**(24):2558-66.

Chang SC, Lacey JV Jr, Brinton LA et al. Lifetime weight history and endometrial cancer risk by type of menopause hormone use in the NIH-AARP diet and health study. *Cancer Epidemiol Biomarkers Prevent* 2007; **16**(4):723-30.

Chinn S, Rona RJ. Trends in weight-for-height and triceps skinfold thickness for English and Scottish children, 1972-1982 and 1982-1990. *Paediatr Perinat Epidemiol* 1994; **8**(1):90-106.

Chinn S, Rona RJ. Prevalence and trends in overweight and obesity in three cross sectional studies of British children, 1974-94. *BMJ* 2001; **322**:24-26.

Chowdhury B, Lantz H, Sjostrom L. Computed-tomography determined body composition in relation to cardiovascular risk factors in Indian and matched Swedish males. *Metabolism* 1996;45:634-44.

Cole, TJ. Weight/height<sup>p</sup> compared to weight/height<sup>2</sup> for assessing adiposity in childhood:influence of age and bone age on *p* during puberty, *Ann Hum Biol* 1986;13:433-451.

Cole, TJ, Freeman JV, Preece MA. Body Mass Index Reference Curves for the UK *Arch Dis Child* 1995; 73; 25-29.

Cole, T.J., Bellizzi, M.C., Flegal, K.M., Dietz, W.H. Establishing a standard definition for child overweight and obesity worldwide: International Survey, *BMJ* 2000; 320; 1240-1245.

Daniels SR, Morrison JA, Sprecher DL, Khoury P, Kimball TR. Association of body fat distribution and cardiovascular risk factors in children and adolescents. *Circulation* 1999;99:541-545.

Daniels SR. Cardiovascular sequelae of childhood hypertension. *Am J Hypertens* 2002;2pt2:61s-63s.

da Silva AC,.Rosa AA. Blood pressure and obesity of children and adolescents association with body mass index and waist circumference. *Arch.Latinoam.Nutr.* 2006;56:244-50.

De Koning L, Merchant AT, Pogue J, Anand SS. Waist circumference and waist-to-hip ratio as predictors of cardiovascular events: meta-regression analysis of prospective studies. *Eur Heart J*, 2007;28:850-856.

De-Spiegelare M, Dramaix M, Hennart P. Social class and obesity in 12-year-old children in Brussels: influences of gender and ethnic origin. *Eur J Pediatr*, 1998;157:432-435.

Department of Health, [www.doh.gov.uk](http://www.doh.gov.uk)

Deurenberg P, Kusters CSL, Smit HE. Assessment of body composition by bioelectrical impedance in children and young adults is strongly age-dependent. *Eur J Clin Nutr* 1990a;44:261-268.

Deurenberg P, Pieters JJL, Hautvast JGAJ. The assessment of body fat percentage by skinfold thickness in measurements in childhood and young adolescence. *Br J Nutr* 1990b;63:293-303.

Deurenberg P, van der Kooy K, Leenen R, Weststrate JA, Siedell JC. Sex and age specific prediction formulas for estimating body composition from bioelectrical impedance: a cross-validation study. *Int J Obesity* 1991;15:17-25.

Dollman J, Norton K, Norton L. Evidence for secular trends in children's physical activity behaviour. *Br J Sports Med* 2005;39:892-897.

Dowling HJ, Pi-Sunyer FX. Race dependent health risks of upper body obesity. *Diabetes* 1993;42:537-543.

Duran-Tauleria E, Rona RJ, Chinn S. Factors associated with weight for height and skinfold thickness in British children. *J Epidemiol Comm Health* 1995;49:466-473.

Durnin JVGA, Rahaman MM. The assessment of the amount of fat in the human body from measurements of skinfold thickness. *Br J Nutr* 1967;21:681-689.

Dwyer T, Blizzard CL. Defining obesity in children by biological endpoint rather than population distribution. *Int J Ob* 1996;20:472-480.

Ebrahim S, Davey Smith G. Mendelian randomization: can genetic epidemiology help redress the failures of observational epidemiology? *Hum Genet* 2008;123:15-33.

Egger G, Swinburn B. An 'ecological' approach to the obesity pandemic. *BMJ* 1997; 315:477-480.

Ehtisham S, Barrett TG, Shaw NJ. Type 2 diabetes mellitus in UK children--an emerging problem. *Diabet Med*. 2000; **17**: 867-71.

Ellis KJ, Abrams SA, Wong WW. Monitoring childhood obesity: Assessment of the weight/height<sup>2</sup> index. *Am J Epidemiol* 1999; **150**:939-46.

Eriksson JG, Forsen T, Tuomilehto J, Barker DJP. Early growth and coronary heart disease in later life: longitudinal study. *BMJ* 2001; **322**: 949-53.

Eriksson JG, Forsen T, Tuomilehto J, Osmond C, Barker DJP. Early adiposity rebound in childhood and risk of type 2 diabetes in adult life. *Diabetologia* 2003; **46**: 190-4.

Eston RG, Rowlands AV, Charlesworth S, Davies A, Hoppitt T. Prediction of DXA-determined whole body fat from skinfolds: importance of including skinfolds from the thigh and calf in young, healthy men and women. *Eur J Clin Nutr* 2005; **59(5)**:695-702.

Fall CH, Osmond C, Barker DJ, Clark PM, Hales CN, Stirling Y, Meade TW. Fetal and infant growth and cardiovascular risk factors in women. *BMJ*. 1995; **310**:428-32.

Fergusson DM, Woodward LJ. Breast feeding and later psychosocial adjustment. *Paediatr Perinat Epidemiol* 1999; **13**(2):144-157.

Field AE, Coakley EH, Must A et al. Impact of overweight on the risk of developing common chronic diseases during a 10 year period. *Arch Intern Med* 2001; **161**(13):1581-6.

Flodmark CE, Sveger T, Nilsson-Ehle P. Waist measure correlates to a potentially atherogenic lipoprotein profile in obese 12-14 year old children. *Acta Paediatr* 1994; **83**:941-5.

Fox K, Peters D, Armstrong N, Sharpe P, Bell M. Abdominal fat deposition in 11 year old children. *Int J Obes* 1993; **17**:11-16.

Francis LA, Lee Y, Birch LL. *Obes Res* 2003; **11**:143-51.

Frayling TM et al. A common variant in the FTO Gene is associated with Body Mass Index and Predisposes to Childhood and Adult Obesity. *Science* 2007; **316**:889-894. (DOI: 10.1126/science.1141634).

Freeman JV, Cole TJ, Chinn S, Jones PRM, White EM, Preece MA. Cross sectional stature and weight reference curves for the UK, 1990. *Arch Dis Child* 1995;73:17-24.

Freedman DS, Dietz WH, Srinivasan SR, Berenson GS. The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa heart study. *Pediatrics* 1999;103:1175-1182.

Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS. Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa heart study. *Pediatrics* 2001;108:712-8.

Friedewald WT, Levy RI, Fredrickson DS. Estimation of the concentration of low density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem*. 1972;18:499-502.

Frohlich ED, Grim C, Labarthe DR, Maxwell MH, Perloff D, Weidman WH. Recommendations for human blood pressure determinations by sphygmomanometers. Report of a special task force appointed by the steering committee, American Heart Association. *Circulation* 1988;77:502-14A.

Garrow JS, p246, *Treat Obesity Seriously*, 1981, London, Churchill Livingstone.

Garrow JS, *Obesity and Related Diseases*, 1988, London, Churchill Livingstone.

Gelber RP, Graziano JM, Manson JE, Buring JE, Sesso HD. A prospective study of body mass index and the risk of developing hypertension in men. *Am J Hypertens* 2007;20(4):370-7.

Gillman MW, Rifas-Shiman SL, Camargo CA, Jr., Berkey CS, Frazier AL, Rockett HR *et al.* Risk of overweight among adolescents who were breastfed as infants. *JAMA* 2001;285:2461-7.

Goodman R. The Strengths and Difficulties Questionnaire: A Research Note. *Journal of Child Psychology and Psychiatry*, 1997;38:581-586.

Goran MI, Gower BA. Abdominal obesity and cardiovascular risk in children. *Cor Art Dis* 1998;9:483-487.

Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. *NEJM* 1993;329:1008-12.

Gregory J, Foster K, Tyler H, Wiseman M. Dietary and Nutritional Survey of British Adults, 1990 HMSO

Grummer-Strawn LM, Mei Z. Does breastfeeding protect against pediatric overweight? Analysis of longitudinal data from the Centers for Disease Control and prevention pediatric nutrition surveillance systems. *Pediatrics* 2004;113:e81-e86.

Guo SS, Roche AF, Chumlea WC, Gardner JD, Siervogel RM. The predictive value of childhood body mass index values for overweight at age 35 years. *Am J Clin Nutr* 1994; 59:810-19.

Haffner SM, Stern MP, Hazuda HP, Rosenthal M, Knapp JA, Malina RM, Role of Obesity and Fat distribution in Non-Insulin-dependent Diabetes Mellitus in Mexican Americans and non-Hispanic whites, *Diabetes Care*, 1986;9:153-61.

Han TS et al. Waist circumference action levels in the identification of cardiovascular risk factors: prevalence study in a random sample. *BMJ* 1995;311:1401-1405.

Han TS, McNeill G, Seidell JC, Lean ME. Predicting intra-abdominal fatness from anthropometric measures: the influence of stature. *Int J Obes Relat Metab Disord* 1997a;21:587-93.

Han TS, Richmond P, Avenell A, Lean MEJ. Waist circumference reduction and cardiovascular benefits during weight loss in women. *Int J Obes* 1997b;21:127-34.

Han TS, Seidell JC, Currall JEP, Morrison CE, Deurenberg P, Lean MEJ. The influences of height and age on waist circumference as an index of adiposity in adults. *Int J Obes* 1997c;21:83-89.

Harder T, Bergmann R, Kallischnigg G, Plagemann A. Duration of breastfeeding and risk of overweight: a meta-analysis. *Am J Epidemiol* 2005; **162**(5):397-403.

Hardoon SL, Whincup PH, Lennon LT, Wanamethee SG, Capewell S, Morris RW. How much of the recent decline in the incidence of myocardial infarction in British men can be explained by changes in cardiovascular risk factors?: evidence from a prospective population-based study. *Circulation* 2008; **117**:598-604.

Hawkins SS, Griffiths LJ, Cole TJ, DeZateux C, Law C, the Millennium Cohort Study Child Health Group. Regional differences in overweight: an effect of people or place? *Arch Dis Child* 2008; **93**:407-13.

Hediger ML, Overpeck MD, Kuczmarski RJ et al. Association between infant breastfeeding and overweight in young children. *JAMA* 2001; **285**(19):2453-60.

Heitmann BL. Review: Impedance: a valid method in assessment of body composition? *Eur J Clin Nutr* 1994; **48**:228-240.

HSE: The Health Of The Nation, Health Survey for England 1994. [www.dh.gov.uk](http://www.dh.gov.uk)  
HSE 1996, 1998, 2002, 2003, 2004, 2006, also at [www.dh.gov.uk](http://www.dh.gov.uk)

Himes JH, Roche AF. Subcutaneous fatness and stature, relationship from infancy to adulthood. *Hum Biol* 1986; **58**(5):737-50.

Houtkooper LB, Going SB, Lohman TG, Roche AF, Van Loan M. Bioelectrical impedance estimation of fat-free body mass in children and youth: a cross-validation study. *J Appl Physiol* 1992; **72**(1):366-373.

Hughes JM, Li L, Chinn S, Rona RJ. Trends in growth in England and Scotland, 1972-1994. *Arch Dis Child* 1997; **76**:182-9.

Hussey J, Bell C, Bennett K, O'Dwyer J, Gormley J. Relationship between intensity of physical activity, inactivity, cardiorespiratory fitness and body composition in 7-10 year old Dublin children. *Br J Sports Med* 2007; **41**: 311-6.

Jackson-Leach R, Lobstein T. Estimated burden of paediatric obesity and co-morbidities in Europe. Part 1. The increase in the prevalence of child obesity in Europe is itself increasing. *Int J Paediatr Obes* 2006; **1**(1):26-32.

Janssen I, Boyce WF, Simpson K, Pickett W. Influence of individual- and area-level measures of socioeconomic status on obesity, unhealthy eating, and physical inactivity in Canadian adolescents. *Am J Clin Nutr* 2006; **83**:139-45.

Jeffery RW, Sherwood NE. Head to Head: Is the obesity epidemic exaggerated? No. *BMJ* 2008; **336**:245.

Kahn HS, Narayan KMV, Williamson DF, Valdez R. Relation of birth weight to lean and fat thigh tissue in young men. *Int J Obes* 2000; **24**:667-72.

Kannel WB, D'Agostino RB, Cobb JL. Effect of weight on cardiovascular disease. *Am J Clin Nutr* 1996; **63**(Suppl), 419S-422S.

Kinra S, Nelder RP, Lewendon GJ. Deprivation and childhood obesity: a cross sectional study of 20 973 children in Plymouth, United Kingdom. *J Epidemiol Community Health* 2000; **54**:456-60.

Klein S, Allison DB, Heymsfield SB, Kelley DE, Leibel RL, Nonas C, Kahn R. Waist circumference and cardiometabolic risk: a consensus statement from Shaping America's Health: Association for weight management and obesity prevention; NAASO, The Obesity Society; the American society for nutrition; and the American Diabetes Association. *Am J Clin Nutr* 2007; **85**:1197-202.

Klein-Platat C, Ouja M, Wagner A, Haan MC, Arveiler D, Schlienger JL, Simon C. Physical activity is inversely related to waist circumference in 12-y-old French adolescents. *Int J Obes (Lond)*;2005; **21**:9-14.

Knight I. The heights and weights of adults in Great Britain. London, HMSO, 1984.

Knight TM, Smith Z, Whittles A, Sahota P, Lockton JA, Hogg G, Bedford A, Toop M, Kernohan EEM, Baker MR. Insulin resistance, diabetes, and risk markers for ischaemic heart disease in Asian men and non-Asian men in Bradford. *Br Heart J* 1992; **67**:343-350.

Knip M, Nuutinen O. Long-term effects of weight reduction on serum lipids and plasma insulin in obese children. *Am J Clin Nutr* 1993; **57**:490-3.

Krekoukia M, Nassis GP, Psarra G, Skenderi K, Chrousos GP, Sidossis LS. Elevated total and central adiposity and low physical activity are associated with insulin resistance in children. *Metabolism* 2007; **56**(2):206-13.

Kuh D, Ben Shlomo Y (eds) A life course approach to chronic disease epidemiology. 2<sup>nd</sup> ed. Oxford University Press 2004.

Laakso M. How good a marker is insulin level for insulin resistance? *Am J Epidemiol*. 1993; **137**:959-65.

Labayen I, Moreno LA, Blay MG, Blay VA, Mesana MI, González-Gross M, Bueno G, Sarría A, Bueno M. Early programming of body composition and fat distribution in adolescents. *J Nutr* 2006; **136**:147-52.

Lahti-Koski M, Harald K, Mannisto S, Laatikainen T, Jousilahti P. Fifteen year changes in body mass index and waist circumference in Finnish adults. *Eur J Cardiovasc Prev Rehabil* 2007; **14**(3):398-404.

Laitinen J, Power C, Järvelin MR. Family social class, maternal body mass index, childhood body mass index, and age at menarche as predictors of adult obesity. *Am J Clin Nutr* 2001; **74**:287-94.

Lake JK, Power C, Cole TJ. Child to adult body mass index in the 1958 British birth cohort: associations with parental obesity. *Arch Dis Child* 1997; **77**:376-381.

Lapidus L, Bengtsson C, Larsson B, Pennert K, Rybo E, Sjöström L. Distribution of adipose tissue and risk of cardiovascular disease and death: a 12 year follow-up of

participants in the population study of women in Gothenburg, Sweden. *BMJ* 1984;289:1257-61.

Larsson B, Svardsudd K, Welin L, Wilhelmsen L, Björntorp BP, Tibblin G. Abdominal adipose tissue distribution, obesity, and the risk of cardiovascular disease and death: 13 year follow up of participants in the study of men born in 1913. *BMJ* 1984;288:1401-4.

Law CM, Barker DJ, Osmond C, Fall CH, Simmonds SJ. Early growth and abdominal fatness in adult life. *J of Epidemiol Comm Health* 1992; 46:184-6.

Lawlor DA et al. Exploring the Developmental Overnutrition Hypothesis using Parental-Offspring Associations and FTO as an Instrumental Variable. *PLoS Medicine* 2008;5(3):0484-0493

Lawlor DA et al. Epidemiological Evidence for the Fetal Overnutrition Hypothesis: Findings from the Mater-University Study of Pregnancy and its Outcomes. *Am J Epidemiol* 2007;165:418-424

Lazarus R, Baur L, Webb K, Blyth F. Adiposity and Body mass indices in children: Benn's index and other weight for height indices as measures of adiposity. *Int J Ob* 1996;20:406-12.

Lean MEJ, Han TS, Morrison CE. Waist circumference as a measure for indicating need for weight management. *BMJ* 1995;311:158-161.

Lear SA, Humphries KH, Kohli S, Birmingham CL. The use of BMI and waist circumference as surrogates of body fat differs by ethnicity. *Obesity* 2007;15(11):2817-24.

Lear SA, Toma M, Birmingham CL, Frohlich JJ. Modification of the relationship between simple anthropometric indices and risk factors by ethnic background. *Metabolism* 2003;52(10):1295-301.

Lew EA. Mortality and weight: insured lives and the American Cancer Society study. *Ann Int Med* 1985;103:1024-9.

Li C, Ford ES, McGuire LC, Mokdad AH. Increasing trends in waist circumference and abdominal obesity among US adults. *Obesity (Silver Spring)* 2007;15(1):216-24.

Li C, Ford ES, Mokdad AH, Cook S. Recent trends in waist circumference and waist:height ratio among US children and adolescents. *Pediatrics* 2006;118:e1390-e1398.

Li H, Stein AD, Barnhardt HX, Ramakrishnan U, Martorell R. Associations between prenatal and postnatal growth and adult body size and composition. *Am J Clin Nutr* 2003;77:1498-505.

Li R, Scanlon KS, Serdula MK. The validity and reliability of maternal recall of breastfeeding practice. *Nutr Rev* 2005; 63(4):103-110.

Lilja M, Eliasson M, Stegmayr B, Olsson T, Soderberg S. Trends in obesity and its distribution: data from the Northern Sweden MONICA survey, 1986-2004. *Obesity (Silver Spring)* 2008 Feb 14. (Epub ahead of print).

Loos RJF, Beunen G, Fagard R, Derom C, Vlietinck R. Birth weight and body composition in young adult men-prospective twin study. *Int J Obes* 2001;25:1537-45.

Lucas A, Sarson DL, Blackburn AM et al. Breast vs Bottle: endocrine responses are different with formula feeding. *Lancet* 1980; 1:1267-69.

Lucas A, Boyes S, Bloom SR et al. Metabolic and endocrine responses to a milk feed in 6 day old term infants: differences between breast and cow's milk formula feeding. *Acta Paediatr Scand* 1981;70:195-200.

McCance DR, Pettitt DJ, Hanson RL, Jacobsson LT, Bennett PH, Knowler WC. Glucose, insulin concentrations and obesity in childhood and adolescence as predictors of NIDDM. *Diabetologia* 1994;37:617-23.

McCarron P, Lawlor DA. North, South: changing directions in cardiovascular epidemiology (Editorial comment). *Stroke* 2003; 34: 2609-11.

McCarthy HD, Jarrett KV, Crawley HF. The development of waist circumference percentiles in British children aged 5.0-16.9y. *Eur J Clin Nutr* 2001;55:902-907.

McCarthy HD, Jarrett KV, Emmett PM, Rogers I and the ALSPAC study team. Trends in waist circumferences in young British children: a comparative study. *Int J Obes* 2005; **29**:157-62.

McCarthy HD, Ellis SM, Cole TJ. Central overweight and obesity in British youth aged 11-16 years: cross-sectional surveys of waist circumference. *BMJ* 2003; **326**:624.

McGill HC Jr, McMahan CA, Herderick EE, et al. Obesity accelerates the progression of coronary atherosclerosis in young men. *Circulation*. 2002; **105**:2712-2718.

McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. *Lancet* 1991; **337**:382-386.

McKeigue PM, Pierpoint T, Ferrie JE, Marmot MG. Relationship of glucose intolerance and hyperinsulinaemia to body fat pattern in South Asians and Europeans. *Diabetologia* 1992; **35**:785-91.

McMurray RG, Harrell JS, Levine AA, Gansky SA. Childhood obesity elevates blood pressure and total cholesterol independent of physical activity. *Int J Ob* 1995; **19**:881-86.

Maffeis C, Pietrobelli A, Grezzani A, Provera S, Tatò L. Waist circumference and cardiovascular risk factors in prepubertal children. *Obes Res* 2001; **9**:179-87.

Mahoney LT, Burns TL, Stanford W, Thompson BH, Witt JD, Rost CA, Lauer RM. Coronary risk factors measured in children and young adult life are associated with coronary artery calcification in young adults: the Muscatine Study. *J Am Coll Cardiol* 1996; **27**:277-84.

Makrides M, Neumann M, Simmer K, Pater J, Gibson R. Are long-chain polyunsaturated fatty acids essential nutrients in infancy? *Lancet* 1995; **345**(8963):1463-1468.

Manson JE, Willett WC, Stamfer MJ et al. Body weight and mortality among women. *NEJM* 1995; **333**:677-85.

Marmot MG, Shipley MJ, Rose G. Inequalities in death- specific explanations of a general pattern? *Lancet* 1984;**i** 1003-1007.

Matthews DR, Hosker JP, Rudenski JS, Naylor BA, Treacher DF, Turner RC. Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia* 1985;**28**(7):412-9.

Moreno LA, Fleta J, Sarriá A, Rodríguez G, Gil C, Bueno M. Secular changes in body fat patterning in children and adolescents of Zaragoza (Spain), 1980-1995. *Int J Obes* 2001;**25**:1656-60.

Moreno LA, Mesana MI, Gozález-Gross M, Gil CM, Ortega FB, Fleta J, Wärnberg J, León JF, Marcos A, Bueno M and the AVENA study group. Body fat distribution reference standards in Spanish adolescents: the AVENA study. *Int J Obes* 2007;**31**:1798-1805.

Moreno LA, Mesana MI, Gozález-Gross M, Gil CM, et al; the AVENA study group. Anthropometric body fat composition reference values in Spanish adolescents: the AVENA study. *Eur J Clin Nutr* 2006;**60**:191-6.

Moreno LA, Sarriá A, Fleta J, Marcos A, Bueno M. Secular trends in waist circumference in Spanish adolescents, 1995-2000-02. *Arch Dis Child* 2005;**90**:818-9.

Morrison JA, Barton BA, Biro FM, Daniels SR, Sprecher DL. Overweight, fat patterning, and cardiovascular disease risk factors in black and white boys. *J. Pediatr* 1999;**135**:451-7.

Morrison JA, Sprecher DL, Barton BA, Waclawiw MA, Daniels SR,.. Overweight, fat patterning, and cardiovascular disease risk factors in black and white girls: The National Heart, Lung, and Blood Institute Growth and Health Study. *J. Pediatr* 1999;**135**:458-64.

Mulligan J, Voss LD. Identifying very fat and very thin children: test of criterion standards for screening test. *BMJ* 1999;**319**:1103-4.

Murphy MJ, Metcalf BS, Voss LD, Jeffery AN, Kirkby J, Mallam KM, Wilkin TJ. Girls at five are intrinsically more insulin resistant than boys: the programming hypothesis revisited-The EarlyBird study (EarlyBird 6). *Pediatrics* 2004;113:82-86.

Must A, Tybor DJ. Physical activity and sedentary behaviour: a review of longitudinal studies of weight and adiposity in youth. *Int J Obes* 2005;29:S84-96.

Narayan KM, Boyle JP, Thompson TJ, Gregg EW, Williamson DF. Effect of BMI on lifetime risk for diabetes in the US. *Diabetes Care* 2007;30:1562-6.

National Children's Obesity Database (05/06 data), Figure 12 (accessed 15/5/08):

[http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsStatistics/DH\\_063565](http://www.dh.gov.uk/en/Publicationsandstatistics/Publications/PublicationsStatistics/DH_063565).

NICE clinical guideline 43 Obesity: Guidance on the prevention, identification, assessment and management of overweight and obesity in adults and children. NICE, 2006; section 1.2.2.9. [www.nice.org.uk](http://www.nice.org.uk)

Nuutinen O, Knip M. Weight loss, body composition and risk factors for cardiovascular disease in obese children: long-term effects of two treatment strategies. *J Am Coll Nutr* 1992; 11(6):707-14.

Obesity; The Report of the British Nutrition Foundation Task Force, Blackwell Science 1999.

Obesity in children under 11, [www.dh.gov.uk](http://www.dh.gov.uk) , April 2006.

Ogden CL, Flegal KM, Carroll MD, Johnson CL. Prevalence and trends in overweight among US children and adolescents, 1999-2000. *JAMA* 2002;288:1728-32.

Oken E, Gillman MW. Fetal origins of obesity. *Obes Res* 2003;11:496-506.

Okosun IS. Abdominal adiposity in US adults: prevalence and trends, 1960-2000. *Prev Med* 2004;39(1):197-206.

Okosun IS. Trends in abdominal obesity in young people: United States 1988-2002. *Ethn Dis* 2006; **16**(2):338-44.

Ong KKL, Ahmed ML, Emmett PM, Preece MA and Dunger DB. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. *BMJ* 2000; **320**:967-971.

OPCS Office of Population Censuses and Surveys. *Geography and mortality, a review in the mid-1980s*. London: HMSO, 1990. (Series DS No 9).

Ortega FB, Tresaco B, Ruiz JR, Moreno LA, Martin-Matillas M, Mesa JL, Warnberg J, Bueno M, Tercedor P, Gutiérrez A, Castillo MJ; AVENA Study Group. Cardiorespiratory fitness and sedentary activities are associated with adiposity in adolescents. *Obesity (Silver Spring)* 2007; **15**:1589-99.

Owen CG, Whincup PH, Odoki K, Gilg JA, Cook DG. Infant feeding and blood cholesterol: a study in adolescents and a systematic review. *Pediatrics* 2002; **110**(3):597-608.

Owen CG, Martin RM, Whincup PH, Davey-Smith G, Gillman MW, Cook DG. The effect of breastfeeding on mean body mass index throughout life: a quantitative review of published and unpublished observational evidence. *Am J Clin Nutr* 2005a; **82**(6):1298-1307.

Owen CG, Martin RM, Whincup PH, Smith GD, Cook DG. Effect of infant feeding on the risk of obesity across the life course: a quantitative review of published evidence. *Pediatrics* 2005b; **115**(5):1367-1377.

Owen CG, Martin RM, Whincup PH, Smith GD, Cook DG. Does breastfeeding influence risk of type 2 diabetes in later life? A quantitative analysis of published evidence. *Am J Clin Nutr* 2006; **84**(5):1043-54.

Paradis G, Lambert M, O'Loughlin J, Lavallee C, Aubin J, Delvin E *et al.* Blood pressure and adiposity in children and adolescents. *Circulation* 2004; **110**:1832-8.

Pinhas-Hamel O, Zeitler P. The global spread of type 2 diabetes mellitus in children and adolescents. *J Pediatr*. 2005 May; 146: 693-700

Power C, Moynihan C. Social class and changes in weight-for -height between childhood and early adulthood. *Int J Obes* 1988;12:445-53.

Power C, Manor O, Matthews S. Child to adult socioeconomic conditions and obesity in a national cohort. *Int J Obes Relat Metab Disord* 2003;27:1081-6.

Power C, Atherton K, Strachan DP, Shepherd P, Fuller E, Davis A, Gibb I, Kumari M, Lowe G, Macfarlane GJ, Rahi J, Rodgers B, Stansfeld S. Lifecourse influences on health in British adults: Effects of socio-economic position in childhood and adulthood. *Int J Epidemiol* 2007; 36:532-539.

Prentice AM. Body mass index standards for children. *BMJ* 1998;317:1401-2.

Punthakee Z, Delvin EE, O'Loughlin J, Paradis G, Levy E, Platt RW, Lambert M. Adiponectin, Adiposity and insulin resistance in children and adolescents. *J Clin Endocrin Metab* 2006;91:2119-2125.

Raitakari OT, Porkka KV, Rönnemaa T, Knip M, Uhari M, Åkerblom HK, Viikari JSA. The role of insulin in clustering of serum lipids and blood pressure in children and adolescents. The Cardiovascular Risk in Young Finns Study. *Diabetologia* 1995;38:1042-1050.

Rampersaud E, et al. Physical activity and the association of common FTO gene variants with body mass index and obesity. *Arch Intern Med* 2008;168(16):1791-7.

Ramsay LE, Williams B, Johnston GD, MacGregor GA, Poston L, Potter JF et al. British Hypertension Society guidelines for hypertension management. *BMJ* 1999;319:630-5.

Ramsey M. Non-invasive automatic determination of mean arterial pressure. *Med Biol Eng Comput* 1979;17:11-8.

Razak F, Anand S, Vuksan V, Davis B, Jacobs R, Teo KK, Yusuf S; SHARE investigators. Ethnic differences in the relationships between obesity and glucose-metabolic abnormalities: a cross-sectional population-based study. *Int J Obes* 2005;29(6):656-67.

Reilly JJ, Dorosty AR, Emmett PM. Prevalence of overweight and obesity in British children: cohort study. *BMJ* 1999;319:1039.

Reilly JJ, Dorosty AR, Emmett PM, Avon Longitudinal study of pregnancy and childhood study team. Identification of the obese child: adequacy of the body mass index for clinical practice and epidemiology. *Int J Obes Relat Metab Disord* 2000;24(12):1623-7.

Reilly JJ, Wilson ML, Summerbell CD, Wilson DC. Review: Obesity: diagnosis, prevention, and treatment; evidence based answers to common questions. *Arch Dis Child* 2002;86:392-395.

Reilly JJ, Methven E, McDowell ZC, Hacking B, Alexander D, Stewart L et al. Health consequences of obesity. *Arch Dis Child* 2003;88:748-52.

Reilly JJ, Armstrong J, Dorosty AR et al. Early life risk factors for obesity in childhood: cohort study. *BMJ* doi:10.1136/bmj.38470.670903.EO (published 20 May 2005).

Reilly JJ. Review: Obesity in childhood and adolescence: evidence based clinical and public health perspectives. *Postgrad Med J* 2006;82:429-437.

Reinehr T, Andler W. Changes in the atherogenic risk factor profile according to degree of weight loss. *Arch Dis Child* 2004;89:419-422.

Reinehr T, Kratzsch J, Kiess W, Andler W. Circulating soluble leptin receptor, leptin and insulin resistance before and after weight loss in obese children. *Int J Obes* 2005;29:1230-5.

Reinehr T, Stoffel-Wagner B, Roth CL, Andler W. High-sensitive C-reactive protein, tumour necrosis factor alpha and cardiovascular risk factors before and after weight loss in obese children. *Metabolism* 2005;54:1155-61.

Reiterer EE, Sudi KM, Mayer A, Limbert-Zinterl C, Stalzer-Brunner C, Fuger G, Borkenstein MH. Changes in leptin, insulin and body composition in obese children during a weight-reduction program. *J Pediatr Endocrinol Metab* 1999; **12**(6):853-62.

Ribeiro J, Guerra S, Pinto A, Oliveira J, Duarte J, Mota J. Overweight and obesity in children and adolescents: relationship with blood pressure, and physical activity. *Ann. Hum. Biol.* 2003; **30**:203-13.

Rich-Edwards JW, Colditz GA, Stampfer MJ et al. Birthweight and the risk of type2 diabetes in adult women. *Ann Intern Med* 1999; **130**:278-84.

Riddoch C, Savage JM, Murphy N, Cran GW, Boreham C. Long term health implications of fitness and physical activity patterns. *Arch Dis Child* 1991; **66**:1426-1433.

Riddoch CJ, Mattocks C, Deere K, Saunders J, Kirkby J, Tilling K, Leary SD, Blair SN, Ness AR. Objective measurement of levels and patterns of physical activity. *Arch Dis Child* 2007; **92**:963-9.

Rissanen AM, Heliovaara M, Knekt P, Reunanen A, Aromaa A. Determinants of weight gain and overweight in adult Finns. *Eur J Clin Nutr* 1991; **45**:419-30.

Rocchini AP, Moorehead C, Katch V, et al. Forearm resistance vessel abnormalities and insulin resistance in obese adolescents. *Hypertension*. 1992; **19**:615– 620.

Roche AF, Siervogel RM, Chumlea WC, Webb P. Grading body fatness from limited anthropometric data. *Am J Clin Nutr* 1981; **34**:2831-2838

Rodríguez G, Moreno LA, Blay MG, Blay VA, Fleta J, Sarría A, Bueno M. Body fat measurement in adolescents: comparison of skinfold thickness equations with dual-energy X-ray absorptiometry. *Eur J Clin Nutr* 2005; **59**:1158-1166.

Rose G. The strategy of preventative medicine. 1992, Oxford University Press.

Rudolf MCJ, Sahota P, Dixey R, Barth JH, Walker J. Increasing prevalence of obesity in primary school children: cohort study. *BMJ* 2001;322:1094-5.

Rudolf M, Christie D, McElhone S, Sahota P, Dixey R, Walker J, Wellings C. WATCH IT: a community based programme for obese children and adolescents. *Arch Dis Child* 2006;91:736-739.

Rudolf MCJ, Greenwood DC, Cole TJ, Levine R, Sahota P, Walker J, Holland P, Cade J, Truscott J. Rising obesity and expanding waistlines in schoolchildren: a cohort study. *Arch Dis Child* 2004;89:235-237.

Sachdev HS, Fall CHD, Osmond C, et al. Anthropometric indicators of body composition in young adults: relation to size at birth and serial measurements of body mass index in childhood in the New Delhi birth cohort. *Am J Clin Nutr* 2005;82:456-66.

Sardinha LB, Going SB, Teixeira PJ, Lohman TG. Receiver operating characteristic analysis of body mass index, triceps skinfold thickness, and arm girth for obesity screening in children and adolescents. *Am J Clin Nutr* 1999;70:1090-5.

Savva SC, Tornaritis M, Savva ME et al. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. *Int J Obes Relat Metab Disord*. 2000;24:1453-8.

Saxena S, Ambler G, Cole TJ, Majeed A. Ethnic group differences in overweight and obese children and young people in England: cross sectional survey. *Arch Dis Child* 2004;89:30-36.

Seidell JC. Are abdominal diameters abominable indicators? In: Angel A, Bouchard C, eds. *Progress in obesity research*: 7. London, Libbey, 1995:305-308.

Sever P, Beevers G, Bulpitt C, Lever A, Ramsay L, Reid J et al. Management guidelines in essential hypertension: report of the second working party of the British Hypertension Society. *BMJ* 1993;306:983-7.

Shaper AG, Wannamethee SG, Walker M. Body weight: implications for the prevention of coronary heart disease, stroke and diabetes mellitus in a cohort study of middle aged men. *BMJ* 1997;314:1311-1317.

Shea S, Aymong E, Zybert P, Shamo H, Tracy RP, Deckelbaum RJ, Basch CE. Obesity, fasting plasma insulin, and C reactive protein levels in healthy children. *Obes Res* 2003;11(1):95-103.

Siedel J, Hagele EO, Ziegehorn J, Wahlefeld AW. Reagent for the enzymatic determination of total serum cholesterol with improved lipolytic efficiency. *Clin Chem* 1983;29:1075-80.

SIGN: Scottish Intercollegiate Guidelines Network. Obesity in children and young people: a national clinical guideline, SIGN 69, 2004. [www.sign.ac.uk](http://www.sign.ac.uk).

Sinaiko AR, Donahue RP, Jacobs DR, Prineas RJ. Relation of weight and rate of increase in weight during childhood and adolescence to body size, blood pressure, fasting insulin and lipids in young adults; The Minneapolis children's blood pressure study. *Circulation* 1999;99:1471-1476.

Singhal A, Cole TJ, Lucas A. Early nutrition in pre-term infants and later blood pressure: two cohorts after randomized trials. *Lancet* 2001; 357: 413-419.

Singhal A, Sadaf Farooqi I, O'Rahilly S, Cole TJ, Fewtrell MS, Lucas A. Early nutrition and leptin concentrations in later life. *Am J Clin Nutr* 2002; 75: 993-99.

Singhal A, Fewtrell M, Cole TJ, Lucas A. Low nutrient intake and early growth for later insulin resistance in adolescents born pre-term. *Lancet* 2003; 361: 1089-97.

Singhal A, Cole TJ, Fewtrell M, Lucas A. Breast-milk feeding and the lipoprotein profile in adolescents born pre-term. *Lancet* 2004; 363: 1571-78.

Slaughter MH, Lohman TG, Boileau RA, Horswill CA, Stillman RJ, van Loan MD, Bemben DA. Skinfold equations for estimation of body fatness in children and youths. *Hum Biol* 1988;60:709-723.

Smoak CG, Burke GL, Webber LS, Harsha DW, Srinivasan SR, Berenson GS. Relation of obesity to clustering of cardiovascular disease risk factors in children and young adults. The Bogalusa Heart Study. *Am J Epidemiol* 1987;125:364-72.

Sorensen TIA, Price RA, Stunkard AJ, Schulsinger F. Genetics of obesity in adult adoptees and their biological siblings. *BMJ* 1989;298:87-90.

Sorof J, Daniels S. Obesity Hypertension in children. A problem of epidemic proportions. *Hypertension* 2002;40:441-447.

Spencer EA, Appleby PN, Davey GK, Key TJ. Validity of self-reported height and weight in 4808 EPIC-Oxford participants. *Public Health Nutrition* 2002;5(4):561-5.

Srinivasan SR, Myers L, Berenson GS. Predictability of childhood adiposity and insulin for developing insulin resistance syndrome (syndrome X) in young adulthood: the Bogalusa Heart Study. *Diabetes* 2002;51:204-209.

Stamatakis E, Primatesta P, Chinn S, Rona R, Falaschetti E. Overweight and obesity trends from 1974 to 2003 in English children: what is the role of socioeconomic factors? *Arch Dis Child* 2005;90:999-1004.

Strachan DP, Rudnicka AR, Power C, Shepherd P, Fuller E, Davis A, Gibb I, Kumari M, Rumley A, Macfarlane GJ, Rahi J, Rodgers B, Stansfeld S. Lifecourse influences on health among British adults: Effects of region of residence in childhood and adulthood. *Int J Epidemiol* 2007; 36:522-531.

Strazzullo P et al. Leisure time physical activity and blood pressure in school children *American Journal of Epidemiology*, 1988;127:726-33.

Stunkard AJ, Sorensen TIA, Hanis C et al. An adoption study of human obesity. *NEJM* 1986;314:193-8.

Stunkard AJ, Harris JR, Pederson NL, McClearn GE. The body mass index of twins who have been reared apart. *NEJM* 1990;322:1483-7.

Sugiuchi H, Uji Y, Okabe H, Irie T, Uekama K, Kayahara N et al. Direct measurement of high-density lipoprotein cholesterol in serum with polyethylene glycol-modified enzymes and sulfated alpha-cyclodextrin [see comments]. *Clin Chem* 1995;41:717-23.

Summerbell CD, Ashton V, Campbell KJ, Edmunds L, Kelly S, Waters E. Interventions for treating obesity in children. *Cochrane Database Syst Rev* 2003; (3):CD001872.

Tanner JM, Whitehouse RH. Revised standards for triceps and subscapular skinfolds in British children. *Arch Dis Child* 1975;50:142-145.

Taylor RW, Jones IE, Williams SM, Goulding A. Evaluation of waist circumference, waist-to-hip ratio, and the conicity index as screening tools for high trunk fat mass, as measured by dual-energy X-ray absorptiometry, in children aged 3-19 y. *Am J Clin Nutr* 2000;72(2):490-5.

Taylor RW, Falomi A, Jones IE, Goulding A. Identifying adolescents with high percentage body fat: a comparison of BMI cutoffs using age and stage of pubertal development compared to using age alone. *Eur J Clin Nutr* 2003;57(6):764-9.

Taylor RW, Jones IE, Williams SM, Goulding A. Body fat percentages measured by dual-energy X-ray absorptiometry corresponding to recently recommended body mass index cutoffs for overweight and obesity in children and adolescents aged 3-18 y. *Am J Clin Nutr* 2002;76(6):1416-21.

Taylor SJ, Whincup PH, Hindmarsh PC, Lampe F, Odoki K, Cook DG. Performance of a new pubertal self-assessment questionnaire: a preliminary study. *Paediatric and Perinatal Epidemiology* 2001;15:88-94.

Toschke AM, Martin RM, von Kries R, Wells J, Smith GD, Ness AR. Infant feeding method and obesity: body mass index and dual-energy X-ray absorptiometry measurements at 9-10 y of age from the Avon Longitudinal Study of Parents and Children (ALSPAC). *Am J Clin Nutr* 2007; **85**:1578-85.

Treuth MS, Sherwood NE, Baranowski T, et al. Physical activity self-report and accelerometry measures from the Girls health Enrichment Multi-site Studies. *Prev Med* 2004; **38 Suppl**:S43-9.

Trinder P. Determination of blood glucose using 4-aminophenazone as oxygen acceptor. *J Clin Pathology* 1969; **22**: 246.

Tulldahl J, Pettersson K, Andersson SW, Hulthen L. Mode of infant feeding and achieved growth in adolescence: early feeding patterns in relation to growth and body composition in adolescence. *Obes Res* 1999; **7**(5):431-437.

Ussher MH, Owen CG, Cook DG, Whincup PH. The relationship between physical activity level, sedentary behaviour and psychological well-being among adolescents. *Soc Psychiatry Psychiatr Epidemiol* 2007; **42**:851-6.

Valdez R, Athens M, Thompson GH, Bradshaw BS, Stern MP. Birthweight and adult health outcomes in a biethnic population in the USA. *Diabetologia* 1994; **37**:624-31.

Valdez R, Greenlund KJ, Wattigney WA, Berenson GS. Use of weight-for-height indices in children to predict adult overweight: the Bogalusa Heart Study. *Int J Obes* 1996; **20**:715-721.

Van Sluijs EMF, McMinn AM, Griffin SJ. Effectiveness of interventions to promote physical activity in children and adolescents: systematic review of controlled trials. *BMJ* online: <http://bmj.com/cgi/content/full/bmj.39320.843947.BE/DC1>. (20th Sep 2007).

Viner RM, Cole TJ. Adult socioeconomic, social, and psychological outcomes of childhood obesity: a national birth cohort study. *BMJ* 2005; **330**:1354.

Vizcaíno VM, Aguilar FS, Martínez MS, López MS, Gutiérrez RF, Rodríguez-Artalejo F. Association of adiposity measures with blood lipids and blood pressure in children aged 8-11 years. *Acta Paediatr*. 2007; **96**:1338-42.

Wadsworth M, Butterworth S, Marmot M, Ecob R, Hardy R. Early growth and Type 2 diabetes: evidence from the 1946 British birth cohort. *Diabetologia* 2005; **48**(12):2505-10.

Wang Y, Lobstein T. Worldwide trends in child overweight and obesity. *Int J Paediatr Obes* 2006; **1**:11-25.

Wannamethee SG, Shaper AG. Weight change and the duration of overweight and obesity in the incidence of type 2 diabetes. *Diabetes Care* 1999; **22**:1266-72

Wardle H (ed), Jotangia D, Moody A, Stamatakis E. Obesity among children under 11. Joint Health Surveys Unit, National Centre for Social Research, Department of Epidemiology and Public Health at the Royal Free and University College Medical School, 2006. [www.dh.gov.uk](http://www.dh.gov.uk)

Wardle J, Boniface D. Changes in the distributions of body mass index and waist circumference in English adults, 1993/1994 to 2002/2003. *Int J Obes (Lond)* 2007; **Oct 9** (Epub ahead of print).

Wardle J, Brodersen NH, Cole TJ, Jarvis MJ, Boniface DR. Development of adiposity in adolescence: five year longitudinal study of an ethnically and socioeconomically diverse sample of young people in Britain. *BMJ* 2006; **332**:1130-35.

Wattigney WA, Webber LS, Srinivasan SR, Berenson GS. The emergence of clinically abnormal levels of cardiovascular disease risk factor variables among young adults: the Bogalusa Heart Study. *Prev Med* 1995; **24**(6):617-26.

Watts K, Beye P, Siafarikas A, et al. Effects of exercise training on vascular function in obese children. *J Pediatr*. 2004a; **144**:620-625.

Watts K, Beye P, Siafarikas A, et al. Exercise training normalizes vascular dysfunction and improves central adiposity in obese adolescents. *J Am Coll Cardiol*. 2004b; **43**:1823-1827.

Weiss R, Dzuira J, Burgert TS et al. Obesity and the metabolic syndrome in children and adolescents. *NEJM* 2004;350:2362-2374.

Welk GJ, Corbin CB, Dale D. Measurement issues in the assessment of physical activity in children. *Res Q Exerc Sport* 2000;71(2 Suppl):S59-73.

Wells JC, Hallal PC, Reichert FF, Menezes AM, Araujo CL, Victora CG. Sleep patterns and television viewing in relation to obesity and blood pressure: evidence from an adolescent Brazilian birth cohort. *Int J Obes* 2008, epub ahead of print; 18 March 2008; doi:10.1038/ijo.2008.37.

Wells JC, Fewtrell MS. Measuring body composition. *Arch Dis Child* 2006;91:612-7.

Wenten M, Gilliland FD, Baumgartner K, Samet JM. Associations of weight, weight change, and body mass with breast cancer risk in Hispanic and non-Hispanic white women. *Ann Epidemiol* 2002; 12(6):435-4

Weststrate JA, Deurenberg P. Body composition in children: proposal for a method for calculating body fat percentage from total body density or skinfold-thickness measurements. *Am J Clin Nutr* 1989;50(5):908-14.

Whincup PH, Cook DG, Shaper AG, Macfarlane D, Walker M. Blood pressure in British children: associations with adult blood pressure and cardiovascular mortality. *Lancet* 1988;15;ii:890-3.

Whincup PH, Cook DG, Papacosta O, Walker M. Childhood blood pressure, body build and birth weight: geographical associations with cardiovascular mortality. *J Epidemiol Community Health* 1992a;46:396-402.

Whincup PH, Cook DG, & Papacosta O. 1992, "Do maternal and intrauterine factors influence blood pressure in childhood?", *Arch Dis Child* 1992b; vol.67, no.12;1423-1429.

Whincup PH, Bruce NG, Cook DG, Shaper AG. The Dinamap 1846SX oscillometric blood pressure recorder: comparison with the Hawksley random-zero sphygmomanometer under field study conditions. *J Epidemiol Community Health* 1992c;46:164-9.

Whincup PH, Cook DG, Adshead F, Taylor S, Papacosta O, Walker M, Wilson V. Cardiovascular risk factors in British children from towns with widely differing adult cardiovascular mortality. *BMJ* 1996;313:79-84.

Whincup PH, Gilg JA, Donald AE, Katterhorn M, Oliver C, Cook DG, Deanfield JE. Arterial distensibility in adolescents: the influence of adiposity, the metabolic syndrome and classic risk factors. *Circulation* 2005;112:1789-1797.

Whincup PH, Deanfield JE. Childhood obesity and cardiovascular disease: the challenge ahead. *Nat Clin Pract Cardiovasc Med* 2005;2:432-3.

Whincup PH, Gilg JA, Odoki K, Taylor SJ, Cook DG. Age of menarche in contemporary British teenagers: survey of girls born between 1982 and 1986. *BMJ* 2001; 322:1095-6.

Whincup PH, Gilg JA, Papacosta O, Seymour C, Miller GJ, Alberti KGMM, Cook DG. Early evidence of ethnic differences in cardiovascular risk: cross sectional comparison of British South Asian and white children. *BMJ* 2002;324:635-640.

WHO European ministerial conference on counteracting obesity (2006); The challenge of obesity in the WHO European region and the strategies for response: briefing papers EUR/06/5062700/6 28/08/06.

WHO expert consultation. Appropriate body-mass index for Asian populations and its implications for policy and intervention strategies. *Lancet* 2004;363:157-63.

WHO fact sheet no 311, September 2006, [www.who.org](http://www.who.org)

WHO global NCD infobase:  
[www.who.int/ncd\\_surveillance/infobase/web/InfoBaseCommon](http://www.who.int/ncd_surveillance/infobase/web/InfoBaseCommon).

WHO 1997: World Health Organisation: Obesity: preventing and managing the global epidemic. 1997, Geneva: WHO.

Willett WC, Manson JE, Stampfer MJ, Colditz GA, Rosner B, Speizer FE et al. Weight, weight change, and coronary heart disease in women. Risk within the 'normal' weight range. *JAMA* 1995;273:461-5.

Wild S, McKeigue P. Cross sectional analysis of mortality by country of birth in England and Wales. *BMJ* 1997;314:705.

Wilson PW, D'Agostino RB, Sullivan L, Parise H, Kannel WB. Overweight and obesity as determinants of cardiovascular risk: the Framingham experience. *Arch Intern Med* 2002; 162(16):1867-72.

Womersley J, Durnin JVGA. A comparison of the skinfold method with extent of 'overweight' and various weight-height relationships in the assessment of obesity. *Br J Nutr* 1977;38:271-84.

Wong SL, Leatherdale ST, Manske SR. Reliability and validity of a school-based physical activity questionnaire. *Med Sci Sports Exerc* 2006;38:1593-600.

Wright CM, Booth IW, Buckler JMH, Cameron N, Cole TJ, Healy MJR, Hulse JA, Preece MA, Reilly JJ, Williams AF. Growth reference charts for use in the United Kingdom. *Arch Dis Child* 2002; 86: 11- 14.

Young JB. Developmental origins of obesity: a sympathoadrenal perspective. *Int J Obes (Lond)* 2006; 30:Suppl 4, S41-9.