The influence of ethnicity, socioeconomic status and obesity intervention on body composition in UK children and adolescents

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Abstract

The prevalence of childhood overweight and obesity has increased over the last twenty years. Additionally, obesity and its related morbidities are not equally distributed across the UK population with it varying between sexes, age groups, socioeconomic groups and ethnic groups.

This thesis is comprised of four studies investigating body composition and its assessment in children. The first study examined body dimensions and fatness in two groups of children from contrasting income backgrounds. Findings showed that children from the 'lower income' were on average (for their age) shorter in height, heavier in weight with a higher BMI and %BF although abdominal fatness was similar between income groups. Thus it was concluded that in addition to greater body weight-for-age, shorter height is a second (and likely a more significant) contributor to the greater BMI, BMI z-score and overweight/obesity prevalence in lower income children. This observation of shorter height was considered to support the hypothesis of height growth limitation among children from low-income groups.

The second study examined changes in a range of measures of body composition and body fat distribution and evaluated these measures in a paediatric weight management context. This study showed that the BMI was not able to accurately reflect the underlying body compositional changes resulting from the intervention in growing obese children. In view of this limitation, it was proposed that more objective measures of body fatness such as Bioelectrical Impedance Analysis (BIA) and more sensitive measures of body fat distribution such as waist circumference be introduced as alternative methods or adjuncts to BMI

The third study examined ethnicity-related variation in whole body and regional body fatness in children from Caucasian, South-Asian, African-Caribbean and Mixed Race backgrounds. This study supports the findings of others, which observed gender-specific variations in body dimensions and composition in children across different ethnic groups. However, it was evident that exploring ethnicity-related variation in body composition is a complex issue compounded by a relatively large number of measures available to characterize body composition. Furthermore, the limitations of exploring variations in this context using the BMI were also described.

The final study generated gender- and ethnic-specific smoothed percentile curves for anthropometric measurements of body composition including waist circumference and BMI and BIA-derived variables including % Body Fat, whole body impedance and Height^2/Impedance. The curves generated showed that children across the ethnic groups varied in their age-related pattern in the direct and indirect indices of body composition. Despite having limited practical utility at this stage, these findings provide preliminary evidence in support of further investigation into the development of ethnic-specific references in measures of body fatness.

The overall findings in this thesis indicate that overweight and obesity varies across different population groups and this variation needs to be considered in the context of national obesity policy formulation and implementation

Publications arising from this thesis

Samani, D., D. Hawdon, and H.D. McCarthy, Prevalence of overweight and obesity in Afro-Caribbean schoolchildren in West London. Proceedings of the Nutrition Society, 2005. **64**: p. 57A-57A.

Samani D., S. O'Callaghan, and H.D McCarthy, What is the best measure of fatness during a weight management programme in obese children? Proceedings of the Nutrition Society, 2006. **65**: p. 43A-43A.

Samani D., T. Anwar, H.D. McCarthy. Comparisons of body-weight status in British Bangladeshi and Indian children residing in London. Proceedings of the Nutrition Society, 2006. **65**: 43A-43A.

Samani, D., L. Prosser, C. Alston, and H.D. McCarthy, Ethnicity-related variation in upper body fatness in East London schoolchildren. Proceedings of the Nutrition Society, 2007. **66**: p. 25A-25A. (Winner of student competition)

Samani-Radia, D., and H.D. McCarthy. Influence of socioeconomic status on upper body fatness and abdominal obesity in children. Proceedings of the Nutrition Society, 2008. **67**: p.E301

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Samani-Radia, D., and H.D. McCarthy (2009). Influence of different body mass index classification systems on ethnic-specific variation in prevalence of overweight and obesity. Proceedings of the Nutrition Society (In Press)

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McCarthy HD, Navti L & Samani-Radia D (2010). Early-for-age height attainment is associated with higher body fat levels and a more abdominal distribution of body fat in children. (Nutrition Society summer symposium, under review).

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Table of Contents

List of Tables

List of Figures

Abbreviations

AC	African-Caribbean
ADP	Air displacement Plethysmography
BEST	Better Eating, Self-esteem, Total-Health
BCM	Body cell mass
BMI	Body Mass Index
BV	Body volume
%BF	%Body fat
BMD	Bone mineral density
BIA	Bioelectrical Impedance Analysis
R	Coefficient of reliability
2-C	2-Component
CT	Computerized tomography
A	Cross-sectional area
I	Current
DCFS	Department for Children Schools and Families
^{2}H	Deuterium
D_2O	Deuterium oxide
DPA	Dual photon absorptiometry
DXA	Dual X-ray Absorptiometry
ECW	Extracellular water
FFM	Fat Free Mass
FFMI	Fat Free Mass Index
FM	Fat Mass
FMG	Fat Mass Gainers
FMI	Fat Mass Index
FML	Fat Mass Losers
FME	Free School Meals
GPs	General Practitioners
HD	Hydrodensitometry
^{1}H	Hydrogen
Z	Impedance
IAAT	Intra-abdominal adipose tissue
IOTF	International Obesity Task Force
KS	Kolmogorov Smirnov
L	Length of conductor
MRI	Magnetic Resonance Imaging
MR	Mixed Race
NCMP	National Child Measurement Programme
NHANES	National Health and Nutrition Exercise Survey
NICE	National Institute for Clinical Excellence
ANCOVA	One-way between-groups analysis of covariance

SW	Shapiro-Wilk
SKF	Skinfold
SA	South Asian
p	Specific resistivity
SD	Standard Deviation
SDS	Standard Deviation Scores
STORM	Statistics Operational Research and Mathematics
SPSS	Statistical Package for Social Scientists
SAAT	Subcutaneous abdominal adipose tissue
TEM	Technical Error of Measurement
TBBM	Total Body Bone Mineral
TBK	Total body potassium
TBW	Total body water
UWW	Under water weighing
VAT	Visceral adipose tissue
V	Voltage
WC	Waist circumference
WHR	Waist-to-hip ratio
WHtR	Waist-to-height ratio
WB Imp	Whole body impedance

Chapter 1: Introduction

1.1 Preamble

There is widespread agreement amongst scientists, health professionals and government agencies that obesity is now a global epidemic which is affecting both adult and child populations. Halting this rising trend in prevalence has become a fundamental challenge because obesity is associated with a number of adverse medical, psychological and economic consequences. Prospective studies have shown the role of obesity in the development of long-term conditions such as cardiovascular disease and diabetes and cross-sectional studies have shown an association between obesity and risk factors for these adverse consequences.

It is now established that both genetic and environmental factors are involved in the aetiology of obesity. Although the independent role of these factors in contributing to this epidemic remains unclear, it is thought that the interaction between these factors plays a more significant role in influencing the rising trends in prevalence of obesity in all populations.

In the UK, obesity is now recognised as a governmental priority and so national and local strategies have been introduced with the aim of halting this rising trend in obesity levels. However, despite combined and continued efforts between the scientific community, the health service and various governmental agencies, there is little evidence to date which indicates that governmental targets are being met. The failure to meet these targets is not an indication of the commitment, dedication and efforts of these groups but instead it demonstrates that we have yet to gain a more precise understanding on how to tackle an epidemic that poses an unprecedented challenge for those working in this field. The research presented in this thesis explores the variation in body composition between normal weight children, obese children, children from differing income backgrounds and ethnic backgrounds and obese children undergoing a weight management intervention. It is hoped the work in this thesis has contributed in some way to gaining an understanding this problem in children.

1.2 Defining obesity in children and adolescents

The simplest definition of obesity in children, as in adults, is an "excess of body fat". Identification of obese individuals would therefore require quantification of excess body fat. The most widely used proxy measure of excess fatness in adults and children is the Body Mass Index (BMI) (weight(kg)/height²(m²)), which was first described by the mathematician Lambert Adolphe Jacques Quetlet. [1] The widespread use of BMI has been ensured by the practicalities of obtaining measurements in a variety of settings.

Overweight and obesity in adults is defined on the basis of fixed BMI cut-offs that have been associated with increased risk of mortality and related morbidity. The current range of adult BMI categories include: underweight <18.5 kg/m^2, ideal 18.5-24.9 kg/m^2, pre-obese or overweight 25.0-29.9 kg/m^2, obese class I 30.0-34.9 kg/m^2, obese class 11 35.0-39.9 kg/m^2; and obese class III >40 kg/m^2 [2,3].

Identification of overweight and obese children using fixed BMI cut-offs is not possible as growth and maturation result in age-related changes in BMI. Consequently, in children definitions of obesity are based on statistically-derived cut-off values relative to age and sex specific reference data [4]. The use of sex-specific BMI-for age charts is now common practice in numerous countries including UK (UK 1990 reference charts) [5], USA (US Centre for Disease Control (CDC) 2000 charts) [6], France [7], Hong Kong [8] and Italy [9]. More recently, the World Health Organization (WHO) introduced international BMI-for-age growth charts spanning from birth to 5 years, developed using data from six countries (Brazil, Oman, Ghana, India, Norway and the US) [10]. Further to this, charts for 5 to 19 years old children and adolescents have been developed by reconstructing the 1977 National Centre for Health Statistics (NCHS)/WHO growth reference data [11].

The position of a child's BMI in the distribution is expressed as a percentile or standard deviation score (SDS)/ z-score for a given age and sex [3,5,12]. A SDS/z-score relates to a normally distributed variable with a mean equal to zero and a standard deviation equal to 1.0 (equation 1.1) [13]. Percentiles (or centiles) are a type of fractile that divide the data into one hundred parts. A SDS can be converted to a centile and vice versa because it is normally distributed.

Equation 1.1: $SDS = \frac{(\text{observed value}) - (\text{median reference value for a population})}{\text{standard deviation for reference population}}$

1.3 Prevalence of childhood overweight and obesity

Epidemiological studies based on BMI as the measure of overweight and obesity have demonstrated a dramatic increase in the prevalence of childhood obesity in the last two to three decades. It is now considered to be a global epidemic as increasing trends have been observed in both developed and developing countries. A recent amalgamation of global surveys has described the trends of this problem between the years 1980 and 2005 [14]. This report has shown that industrialized countries within North America, Europe (southern and western) and parts of the Western Pacific have the highest prevalence of overweight and obese children. In contrast, parts of South East Asia and a majority of sub-Saharan Africa have the lowest estimates. Countries that fall in between include South and Central America, Northern Africa and the Middle Eastern countries. Using population-weighted annualised increases in prevalence, estimates on the prevalence levels in 2006 and 2010 in each region were drawn up. These projections mirror previous trends whereby a rise in the prevalence of both overweight and obesity was expected (Table 1.1).

Table 1.1: Global prevalence of overweight and obesity (%) in school-aged children based on most recent surveys (International Obesity Task Force (IOTF criteria) and estimated prevalence for 2006 and 2010 [14]

	Most recent survey		Projected 2006		Projected 2010	
Who region (dates of most recent surveys)	**O'weight (inc obesity) %**	**Obesity %**	**O'weight (inc obesity) %**	**Obesity %**	**O'weight (inc obesity) %**	**Obesity %**
Africa (1987-2003)	1.6	0.2	*	*	*	*
America (1988-2002)	27.7	9.6	40.0	13.2	46.4	15.2
Eastern Med (1992-2001)	23.5	5.9	35.3	9.4	41.7	11.5
Europe (1992 – 2003	25.5	5.4	31.8	7.9	38.2	10.0
South East Asian (1997 – 2002)	10.6	1.5	16.6	3.3	22.9	5.3
Western Pacific (1993 – 2000)	12.0	2.3	20.8	5.0	27.2	7.0

*Insufficient data to make estimate of projected prevalence rates

In the UK, national surveys such as the Health Survey for England (HSE) [15] and the National Study of Health and Growth (NSHG) [16] have provided evidence of a dramatic rise in numbers of overweight and obese children since 1984. The NSHG findings showed that between 1984 and 1994, prevalence of overweight increased in English boys and girls by 3.6% and 4.1% respectively. Similarly the prevalence of obesity during this period increased from 0.6% to 1.7% in boys and 1.3% to 2.6% in girls [16]. The HSE demonstrated that this upward trend since the mid 1980's continued to increase beyond the NSHG study period (figures 1.1, 1.2) [17].

Figure 1.1: Trends in overweight in children aged 2-15 years in England [17]

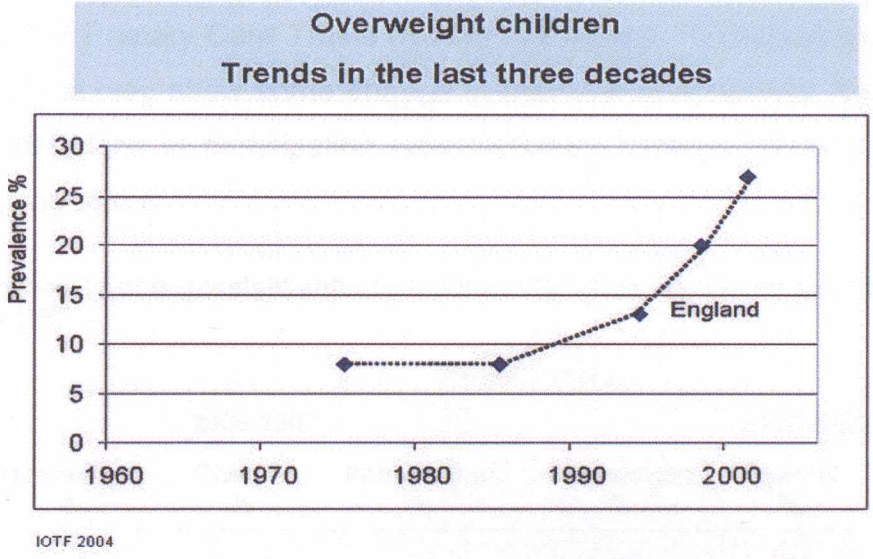
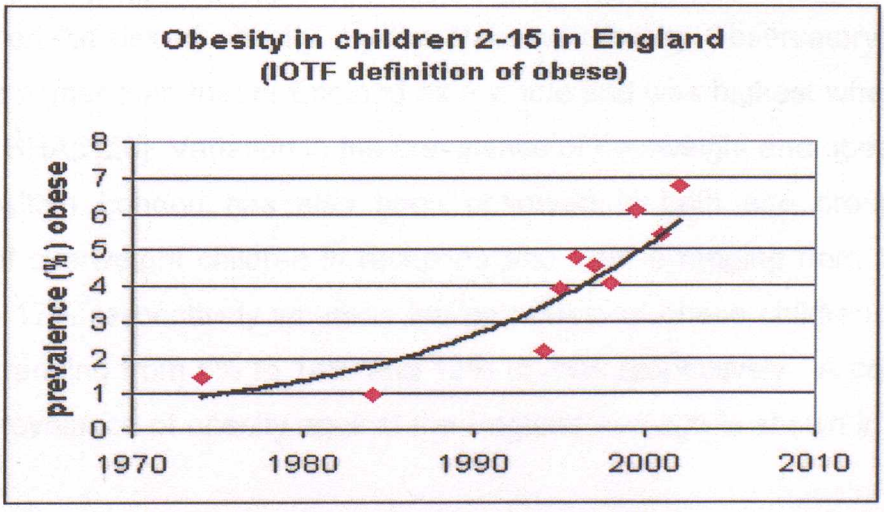


Figure 1.2: Trends in obesity in children aged 2-15 years in England [17]



The most recent HSE (2008) estimates the proportions of overweight and obese boys and girls (aged 2-15 years) in England to be 31% and 29% and obese to be 17% and 15% respectively [535]. Although some levelling off in prevalence of obesity has been observed since the 2002 survey, future surveys will confirm whether this is a continuing pattern.

Annual figures on the prevalence of overweight and obesity in England are now being gathered through the National Child Measurement Programme (NCMP) [18]. Introduced in 2005 as a government strategy to combat obesity, the NCMP obtains prevalence levels in children aged between 4 and 5 years (reception year) and between 10 and 11 years (Year 6) from the 10 NHS Strategic Health Authorities (SHAs) and 152 Primary Care Trusts (PCTs) in England. Findings from the first and second NMCP survey show some change in prevalence of obesity (Table 1.2) [19]. However, differences in participation rates between surveys should be considered when assessing trends.

Table 1.2: Prevalence of overweight and obesity from first (2006-2007) and second (2007-2008) NCMP survey [19]

	NCMP Survey					
	2006-2007			2007-2008		
	Overweight %	Obesity %	Participation %	Overweight %	Obesity %	Participation %
Reception	13	9.9	83	13	9.6	89
Year 6	14.2	17.5	78	14.3	18.3	87

Regional analysis of the most recent 2007-2008 survey showed that the proportion of obese (termed "at risk of obesity" by the National Obesity Observatory) children in London was higher than that of England as a whole and was highest when compared to the other SHAs [20]. Variation in the prevalence of overweight and obesity between the PCTs within London has also been observed in both age groups with the proportion of overweight children in reception and Year 6 ranging from 10% to 15% and 12% to 17% respectively whereas the proportion of obese children in reception and Year 6 ranging from 6% to 14% and 12% to 26% respectively. A comparison of PCT level prevalence of obesity against the England average is shown in Figures 1.3 and 1.4.

1.3.1 Ethnicity related prevalence of overweight and obesity

National surveys have also illustrated a large variation in the prevalence of obesity between ethnic groups within a population. Variations within the American population have been identified by the National Health and Nutrition Exercise Survey (NHANES). The period between the NHANES 1988-1994 survey and the 1999-2000 survey demonstrated an increasing trend in the prevalence of overweight in all age and ethnic groups. However, the increase in prevalence was markedly greater in the non Hispanic black (from 13.4% to 23.6%) and Mexican American groups (from 13.8 to 23.4%). This pattern was evident in both children aged 6 to 11 years and adolescents aged 12 to 19 years [21,22].

In the UK, the 1997 National Diet and Nutrition Survey (NDNS) demonstrated the increasing trend in young British children and highlighted higher rates of obesity in South Asian children [23]. The "ethnic boost" sample of the HSE 1999 also provided evidence of differential rates of obesity between ethnic groups, with Black Caribbean and Indian boys having a higher mean BMI than the general population [24].

The more recent HSE 2004 also highlighted this differential pattern in children from various ethnic groups (tables 1.3 & 1.4) [25]. Similar patterns of overweight and obesity were evident in children from minority ethnic groups between the two surveys. An increase in prevalence of obesity was only observed in the Black Caribbean and Bangladeshi boys with no significant change being observed between the 1999 and 2004 surveys in the other ethnic groups.

Table 1.3: HSE 2004 - Prevalence of overweight and obesity in Boys aged 2-15 years [25]

	Black Caribbean	Black African	Indian	Pakistani	Bangladeshi	General population 2004
% including obese	39	42	26	39	34	32.6
% obese	28	31	14	25	22	18.9

Table 1.4: HSE 2004 – Prevalence of overweight and obesity in females aged 2-15 years [25]

	Black Caribbean	Black African	Indian	Pakistani	Bangladeshi	General population 2004
% including obese	42	40	31	25	33	34.1
% obese	27	27	21	15	20	17.8

Both HSE surveys (1999 and 2004) have demonstrated that prevalence rates in children from some minority ethnic groups within the UK are exceeding those of the indigenous population.

Variation in the prevalence of overweight and obesity between ethnic groups has also been reported in the most recent NCMP survey (table 1.5) [19]. By calculating odds ratios (and using the White British children as the reference), it was shown that boys and girls from the minority ethnic groups were generally (with the exception of Chinese girls), more likely to be classified as obese compared to White British children.

Table 1.5: Odds ratios for the relative likelihood of minority ethnic groups being obese in comparison to the White British ethnic group [19] *Significance at the 5% level

	Boys		Girls	
	Reception	Year 6	Reception	Year 6
Bangladeshi	1.588 *	1.799*	1.382*	1.180*
Black African	1.811*	1.498*	2.146*	1.973*
Black Caribbean	1.400*	1.596*	1.642*	2.104*
Chinese	0.868	1.001	0.323*	0.530*
Indian	0.948	1.368*	0.882	1.028
Pakistani	1.309*	1.564*	1.328*	1.336
White British	1	1	1	1

1.3.2 Economic burden of obesity

The increasing trends in both adult and childhood overweight and obesity in the UK have had a significant impact on the economy. In 2006, a joint report called "Tackling Child Obesity-First Steps" was released by The Audit Commission, The Healthcare Commission and The National Audit Office in which estimates on costs relating to obesity indicated that the direct cost to the NHS relating to the diagnosis and treatment of obesity was approximately £1 billion a year whereas indirect costs to the economy from lost output due to sickness, absence or death of workers was £2.3-£2.6 billion annually [26]. Based on the trends previous to this report, it was estimated that the annual cost to the economy would increase to £3.6 billion per year by 2010. Further to this, the 2008 Healthy Weight, Healthy Lives Cross Government Strategy, estimated that that the overall cost to society would be £50 billion by 2050 [27]. It is evident from these estimates and projections that the current trends in prevalence of

obesity are having a substantial and significant impact on the economy and that worsening trends will continue to have a detrimental effect on the global economy.

1.4 Consequences of overweight and obesity

The rising trends in prevalence are cause for concern given that obesity is associated with a vast range of adverse consequences that manifest either in childhood or over a longer period of time into adulthood. The range of obesity related co-morbidities will be discussed below.

1.4.1 Cardiovascular, metabolic and endocrine disorders

Obesity is widely acknowledged to be an independent risk factor and significant contributor to the prevalence of cardiovascular disease (CVD) in adults [28]. The risk of developing CVD in obesity can partly be explained by its effects on a range of established and novel cardiovascular risk factors such as Type 2 Diabetes Mellitus, impaired glucose tolerance, insulin resistance, dyslipidaemia, hypertension and thrombotic and inflammatory markers. However, evidence from prospective studies has shown that obesity itself has a residual impact on CVD that is independent of its effects on these established and novel risk factors [29].

Although the risk of cardiovascular mortality is low in youth, the presence of risk factors in overweight and obese children and adolescents has been widely reported. The presence of these risk factors in early life accelerates the risk of developing CVD in adulthood as continued and prolonged exposure together with increasing excess body fat leads firstly to atherosclerosis and then to overt CVD [30,31]. Pathology data from the Bogalusa Heart study and autopsy results from the Pathobiological Determinants of Atherosclerosis in Youth study have shown that obesity is associated with accelerated coronary atherosclerosis in children and adolescents [32,33]. Furthermore, a significant association between atherosclerotic lesions and the number of cardiovascular risk factors has been demonstrated in young adults [34].

Cardiovascular risk factors will be examined separately with evidence relating risk to overweight and obesity in children and adolescents also discussed.

1.4.1.1 Type 2 Diabetes Mellitus

Type 2 Diabetes (T2DM) can be defined as a "state of chronic hyperglycaemia" that was formerly considered to be a condition confined to adulthood [35]. However an alarming increase in the number of adolescents diagnosed with this condition has been reported more recently. In a regional survey in greater Cincinnati (USA), the incidence of T2DM in adolescents was reported to rise from 0.71/100,000 per year in 1982 to 7.2/100,000 per year in 1994 [36]. This survey also observed that a third of all new cases of diabetes in 1994 amongst 10 to 19 year olds were accounted for by T2DM. Another important finding from this study was that BMI values were above the 90th centile in ninety percent of the cases diagnosed with T2DM. Paediatric T2DM in the UK was first reported in 2000 in obese adolescents from minority ethnic groups [37]. Since then, UK adolescents of white origin have also been identified with the condition [38]. Following this, a survey examining the cases of paediatric T2DM in the UK reported a minimum prevalence of 0.21/100 000 in children under 16 years of age [39]. As with the UK and USA, a similar trend of increasing incidence in T2DM has been reported in child and adolescent from other populations [40,41].

The pathophysiology underlying the development of T2DM is thought to involve insulin resistance and a defect in insulin secretion. Insulin resistance is characterized by a defect in insulin action, which results in a diminished ability to stimulate glucose uptake by muscles and adipose tissue and suppress hepatic glucose production and output [35,42]. It has been proposed that the pathophysiology of T2DM begins with a period of insulin resistance which in turn leads to an increase in pancreatic insulin secretion (hyperinsulinaemia). This action allows the metabolic functions of insulin (inhibiting glucose output from the liver and promoting peripheral uptake) to continue so that plasma glucose levels remain relatively normal. However, progression of the disease leads to deterioration in pancreatic cell function and consequently hyperglycaemia prevails [35]. However the significance of these defects and their exact sequence of development in the pathogenesis of T2DM still remain unclear.

The presence of these intermediary stages and their association with obesity has been widely reported in children and adolescents. The contributory role of body fat in increasing the risk of T2DM has been shown in children as young as 10 years in whom a high correlation was reported between fasting insulin levels and percentage

body fat [43]. Another study found that insulin and insulin to glucose ratio were greater in obese prepubertal boys compared to a control group during a 3 hour oral glucose tolerance test [44]. Further evidence highlighting the adverse effects of obesity on insulin-glucose metabolism was apparent in a study that found obese adolescent girls to have impaired glucose disposal. This study also found that the obese girls were unable to increase glucose oxidation and suppress lipid oxidation following insulin infusion [45].

Although the association between body fat and T2DM is well recognized, the exact mechanism by which excess body fat increases disease risk is still unclear. Two possible hypotheses have been proposed to explain this association. Firstly, it is thought that excess body fat has adverse metabolic effects only when it accumulates in specific depots. A second hypothesis proposes that it is not fat per se but the metabolic products derived from body fat (e.g. leptin, tumour necrosis factor-α) that may be linked to insulin resistance [46,47].

Together with the uncertainty on the exact pathophysiology of T2DM, it is also unclear whether the natural history of the development of this condition is similar in adults and children. Although the specific time frame in which T2DM develops remains unclear in both groups, it is thought to develop more rapidly in children and adolescents than in adults [47].

1.4.1.2 Hypertension

Hypertension as defined by the British Hypertension Society is a systolic blood pressure ≥140 mm Hg and/or a diastolic blood pressure ≥90mm Hg [48]. The evidence indicating the deleterious effects of hypertension on the cardiovascular system has been described by clinical, experimental and pathologic studies. Such studies have shown that hypertension is a major risk factor for sudden death, coronary heart disease (CHD), stroke, congestive heart failure and renal insufficiency. The adverse effects of hypertension are a result of the mechanical stress exerted on the heart and blood vessels which results in thickening of the arterial intima and media and luminal narrowing of the small arterioles. Moreover, hypertension increases susceptibility to the development of occlusive diseases such as CHD and stroke as it appears to aggravate and accelerate the process of atherosclerosis within the cerebral and coronary arterial walls [49,50].

Obesity has been identified to be the most important determinant of hypertension [51]. The positive association between body weight and hypertension was shown in the Framingham study which found that a 10% increase in body weight explained a 7mm Hg rise in systolic blood pressure [52]. It has also been shown that a decrease in every kilogram of excess body weight was associated with a decrease in 0.33 and 0.43 mm Hg systolic and diastolic blood pressure respectively [53].

Several studies have documented the relation between raised blood pressure and overweight and obesity in children [54,55]. A parallel increase in blood pressure and prevalence of obesity has been reported in American youths over the last decade [56]. The risk of developing hypertension is reported to be between 2.5 and 3.7 times greater in overweight compared to normal weight children [57]. Furthermore, the increase in blood pressure has shown to be associated with the degree of overweight as every unit (kg/m^2) increase in BMI was associated with a 0.8mm Hg and 1.2mm Hg increase in systolic blood pressure in males and females aged 15 to 19 years respectively [58]. These findings are cause for concern as blood pressure values have shown to track from adolescence into adulthood especially in overweight and obese individuals. Evidence from the Bogalusa Heart study and Muscatine study has demonstrated that the likelihood of being hypertensive in adulthood in greater in obese compared to lean children and youth [59,60].

Although the link between obesity and hypertension has been well documented, the precise mechanism of this association remains unclear. Proposed mechanisms explaining this association includes the activation of the rennin-angiotensin-aldosterone system, increased activity of the sympathetic nervous system, promotion of insulin resistance and leptin resistance, increased procoagulatory activity and endothelial dysfunction [61,62].

The association between hypertension and insulin metabolism/resistance has been addressed by several studies and shown in both adults and children. Hypertension is therefore considered to be an insulin resistant state [63]. Insulin levels have been found to be significantly higher in hypertensive adults compared to their normotensive counterparts whether it was measured in the fasting state or following an oral glucose tolerance test [64]. Similarly, a positive association between blood pressure and fasting insulin has also been reported in young children [65]. Insulin

metabolism/resistance has been linked to hypertension as insulin is thought to increase sodium retention, increase the activity of the sympathetic nervous system and stimulate the growth of vascular smooth muscle [66]. Evidence of these mechanisms has been found in adolescents where insulin resistance was associated with chronic sodium retention, sodium sensitivity and increased forearm vascular resistance [67-69].

1.4.1.3 Dyslipidaemia

Abnormalities in the lipid profile have been reported in both obese adults and children and this signifies an increased risk of CVD as the development of atherosclerosis is directly related to plasma lipoprotein levels [70]. The relation between adverse lipids levels and obesity in children has been extensively studied by the Bogalusa Heart Study. This is a longitudinal study that has been investigating the early natural history of cardiovascular disease in a biracial cohort of children in Bogalusa (Louisiana) since 1973 [71]. Findings from this study have shown that that the likelihood of having adverse levels of total cholesterol, low density lipoprotein cholesterol (LDL-C), high density lipoprotein cholesterol (HDL-C) and triglycerides (TG) was greater by 2.4, 3.0 3.4 and 7.1 times respectively in children with a BMI above the 85th centile compared with normal weight children [72]. Conversely, another study showed that a decrease in the degree of obesity was associated with a decrease in circulating TG, LDL-C and an increase in levels of HDL-C concentrations. The increased risk of endothelial dysfunction and early atherogenesis may also be evident from the higher levels of haemostatic factors such as fibrinogen, plasminogen activator inhibitor-1 and inflammatory factors such as C-reactive protein observed in obese children [73-75].

Several studies have demonstrated the impact on adult health by reporting the tracking of lipoprotein levels from childhood to adulthood [76]. For instance, the prevalence of abnormal concentrations of LDL-C, triglycerides and HDL-C was 2.4, 3 and 8 times greater respectively in overweight adults who had been overweight adolescents compared to those who remained lean [60]. Furthermore, 50% of children and adolescents that had either total cholesterol or LDL-C concentrations above the 75th percentile had a similar lipid profile in young adulthood [77]. This study also showed that the best predictor of total cholesterol level in young adulthood was a measurement taken 12 years earlier.

1.4.1.4 Metabolic Syndrome and body fat distribution

The evidence described above highlights that overweight and obesity are linked with several cardiovascular risk factors. A consequence of this is that overweight and obese individual can often be found with multiple risk factors. This phenomenon of clustering of risk factors was first described by Reaven in 1988 and referred to as Syndrome X [78]. Syndrome X, also known as metabolic syndrome, refers to the clustering of abdominal obesity, hypertension, hyperinsulinaemia, glucose intolerance and dyslipidaemia [79,80]. Diagnosis of metabolic syndrome in adults is currently based on three clinical definitions- the Whole Health organization definition [81], National Cholesterol Education Program-Third Adult Treatment Panel (NCEP ATP III) definition [82] and the International Diabetes Federation (IDF) definition [83]. A number of definitions also exist for the diagnosis of metabolic syndrome in children and adolescents which have been extrapolated from the adult guidelines to form age and gender dependent normal values for many of the parameters (Table 1.3) [84]. However as with adults, there is no consensus on which definition should be used.

A rising trend in the prevalence of metabolic syndrome has become evident from surveys on nationally representative children and adolescents in the US. Findings from the 1998 to 1994 NHANES showed that the prevalence of metabolic syndrome was 29% in obese adolescents (BMI≥ 95th percentile), 7% in overweight adolescents (BMI 85th to 95th percentile) and 0.6% in normal weight adolescents [85]. Analysis of the more recent survey conducted between 1999 and 2000 demonstrated that the prevalence of metabolic syndrome had increased since the previous survey and that levels remained higher in overweight (38.6%) compared to normal weight adolescents (1.4%) [91].

The prevalence of metabolic syndrome has also been investigated in child and adolescent populations of other countries. Findings from the UK are limited to one study which investigated the prevalence of metabolic syndrome using the criterion of having 3 or more components of the syndrome (obesity, abnormal glucose homeostasis, dyslipidaemia and hypertension) [92]. This study found that from a group of 103 obese children and adolescents aged 2 to 18 years, a third met the criteria for metabolic syndrome. Another such study in obese Turkish children and adolescents aged 7 to 18 years found that the prevalence of metabolic syndrome

within this group was 27.2 % [93]. It was also noted in this study that the prevalence was significantly higher in adolescents than in children.

Although it is not possible to compare the prevalence of metabolic syndrome between these surveys because of the differing criteria for diagnosis, it is apparent that a significant number of overweight/obese children present with metabolic syndrome. It is also evident from these studies that prevalence is greater in obese/overweight than in normal weight children and adolescents. Moreover, each aspect of the syndrome has shown to worsen with the degree of obesity as demonstrated by an increase in risk with every 0.5 increment in BMI units (kg/m^2) [88]. The risk of developing metabolic syndrome and CVD in adulthood is evident in the fact that childhood obesity predicts the development of metabolic syndrome in adulthood [86]. Secondly, clusters of cardiovascular risk factors are reported to be characteristically stable and therefore track well from childhood to adulthood [94,95]. Thus the presence of metabolic syndrome in early life may result in continued exposure to a range of risk factors which accelerates the risk of developing atherosclerosis in the first instance and later overt CVD and related morbidity [96,97].

The key factor contributing to an increase in risk of CVD and diabetes is excess body fat. However, specific depots of body fat are known to have a more important role in increasing the likelihood of developing adverse risk factors. Studies in adults and children have shown that central/abdominal obesity is more strongly associated with cardiovascular risk factors than overall obesity (whole body fatness) [98-100]. This notion was first proposed by Vague in 1947 who believed obesity to be a heterogeneous condition in which regional distribution of adipose tissue had an important and adverse role in the association between obesity and disturbances in lipid and glucose metabolism. Furthermore, Vague put forward a phenotypic classification system to describe patterns of body fat distribution. A central/abdominal pattern of distribution was classified as android or male type obesity whereas a more lower body /gluteo-femoral distribution was classified as gynoid or female type obesity [101]. Prospective studies in adults have since shown that android obesity correlated more strongly with increased mortality, atherosclerosis of coronary, cerebral and peripheral vessels, diabetes and the cardiovascular risk factors described above [98,102]. A more recent prospective study conducted in ten European countries showed a similar positive association between abdominal obesity (assessed using

waist circumference) and risk of death and concluded that both general (assessed using BMI) and abdominal obesity are important in determining mortality risk [103]. However, more advanced imaging techniques have provided a better understanding of the exact components of abdominal adipose tissue and the differential risk associated with these fat depots.

The use of imaging and radiographic techniques such as computed tomography (CT) and magnetic resonance imaging (MRI) have shown that abdominal adipose tissue is made up of subcutaneous adipose tissue (SAAT) and intraabdominal adipose tissue (IAAT) [104]. The IAAT is made up of retroperitoneal fat and visceral fat (VAT) that is made up of omental and mesenteric fat [104,105]. In terms of anatomical location, VAT is attached to and surrounded by the intestines and is drained by the hepatic portal vein, therefore transporting its secretory products directly to the liver. Estimating proportions of the IAAT compartments using anatomical landmarks and imaging techniques is subject to error however; cadeveric analysis has shown that VAT and retroperitoneal fat make up 61-71% and 29-33% of IAAT respectively [106].

Table 1.6: Definitions for metabolic syndrome in children and adolescents [source: 84]

Definition	Cook et al. (2003) [85]	De Ferrant et al (2004) [86]	Cruz et al (2004) [87]	Weiss et al (2004) [88]	Ford et al (2005) [89]	IDF(2005) (6-10 y) [90]
Criteria	3 or more of the following	3 or more of the following	3 or more of the following	3 or more of the following	3 or more of the following	WC + 2 other measures
	Fasting glucose ≥ 10mg/dL	Fasting glucose ≥6.1mmol/L (≥ 10mg/dL)	Impaired glucose tolerance (ADA criterion)	Impaired glucose tolerance (ADA criterion)	Fasting glucose ≥110mg/dL (additional analysis with 100mg/dL)	
	WC ≥90th percentile (age-and sex-specific) (NHANES III)	WC ≥75th percentile	WC ≥90th percentile (age-, sex-and race-specific) (NHANES III)	BMI Z-score ≥2.0 (age-and sex-specific)	WC ≥90th percentile (sex-specific) (NHANES III)	WC ≥90th percentile (or adult cut-off if lower)
	TG ≥110mg/dL (age-specific, NCEP)	TG ≥1.1mmol/L (≥100mg/dL)	TG ≥90th percentile (age-and sex-specific, NHANES III)	TG ≥90th percentile (age- sex and race-specific, NGHS)	TG ≥110mg/dL (age-specific, NCEP)	TG ≥1.7mmol/L (>150mg/dL)
	HDL-C ≤40mg/dL (all ages/sexes, NCEP)	HDL-C <1.3mmol/L (<50mg/dL)	HDL-C ≤10th percentile (age-and sex-specific, NHANES III)	HDL-C ≤5th percentile (age- sex and race-specific, NGHS)	HDL-C ≤40mg/dL (all ages/sexes, NCEP)	HDL-C <1.03mmol/L (<40mg/dL)
	Blood pressure ≥90th percentile (age-sex- and height-specific NHBPEP)	Blood pressure >90th percentile	Blood pressure >90th percentile (age-,sex- and height-specific, NHBPEP)	Blood pressure >95th percentile (age-,sex- and height-specific, NHBPEP)	Blood pressure >90th percentile (age-,sex- and height-specific, NHBPEP)	Systolic ≥130 mm Hg / Diastolic ≥85mmHg

ADA: American Diabetes Association, IDF: International Diabetes Federation, NCEP: National Cholesterol Education Program, NHBPEP: National High Blood Pressure Education Program, NHANES: National Health and Nutrition Exercise Survey, WC: Waist Circumference.

1.4.2 Body fat patterning and fat distribution in children and adolescents

1.4.2.1 Growth-related changes in subcutaneous fat

The existence of sexual dimorphism in fat distribution is not limited to adulthood. Differences in fat patterning and distribution together along with unique patterns of change during growth and maturation have also been described in children and adolescents. Longitudinal studies such as the Child Research Council Study (Denver) presented information on growth-related changes in subcutaneous fat distribution in children from infancy to 18 years using radiographic and skinfold thickness data [107].

Although some discrepancies were apparent between the radiographic and skinfold data, both assessment techniques showed that subcutaneous fat was greater in girls than in boys at all ages. This study observed a rapid rise in subcutaneous fat in both genders during the first 6 months of life, which was followed by a decrease up until 6 to 7 years. Puberty in girls was reported to be characterised by a linear increase in subcutaneous fat whereas in boys a slight increase was seen between the ages of 7 to 12/13 years followed by a relative decrease [107].

The use of skinfold thickness ratios have been widely used to describe the distribution of subcutaneous fat in the trunk relative to that in the extremities (legs and arms) (trunk skinfold thickness/extremity skinfold thickness). A stable pattern has been reported during childhood in both genders with the thickness of trunk fat being approximately half of that in the extremities. The ratio was reported to continue increasing with age in both genders illustrating a greater gain in trunk than in extremity subcutaneous fat. It is during adolescence where a sexual dimorphism was evident as girls showed very little change after the age of 12/13 years whereas boys continued to have an increase in the ratio. This rise in the ratio in boys during adolescence was thought to be characterized by not only an increase in subcutaneous fat in the trunk but also a decrease in the extremities [107].

The emergence of a sexual dimorphism in fat distribution during puberty has also been reported in other studies [108]. However, the use of more advanced assessment techniques has shown that sexual dimorphism in fat distribution exists even in prepubertal children. This study used analysis of covariance rather than ratios

to show that the gynoid pattern of fat distribution was evident in girls well before the physical signs of puberty were apparent [109].

1.4.2.1 Visceral fat (VAT) in children and adolescents

The existence of VAT in children and adolescents was identified in the mid 1990s with its accumulation being detected in children as young as 4 years of age [110]. This study found that the mean VAT accumulation was 8cm^2 in children aged between 4.4 and 8.8 years. When compared to adults, this represents approximately 10% of the VAT commonly reported in normal weight young adults [107].

The growth trajectory of visceral fat has not been widely studied. Evidence that VAT accumulates with increasing age was apparent in a longitudinal study in children that found an average increase of 5.2cm^2 per year [111]. Moreover, studies with older children and adolescents have shown greater mean VAT areas than those with younger children [107]. However, this pattern is far from consistent throughout all studies and it been shown that accumulation of VAT in children and adolescents is highly variable, and it could be influenced by a multitude of factors such as whole body fatness, ethnicity, physical fitness, sex hormones and prenatal and post-natal dietary experience and growth [112]. For instance, one study found that IAAT increased after puberty and accumulation was greater in boys than in girls [113]. In line with this observation, no change in VAT was reported in a longitudinal study (4 years) on obese adolescents in whom relative body weight [(weight/weight for height) / x100] did not change during the study period [114].

Overweight and obese adults, children and adolescents have been found to have significantly higher levels of VAT than their normal weight counterparts [115,116]. One such study demonstrating this difference reported a mean VAT level of 49cm^2 in obese children (aged 5-10 years) and 22cm^2 in normal weight children [116]. Evidence indicating that the accumulation of VAT is associated with whole body fatness is also apparent from studies that have shown correlations between the two fat compartments. Studies in adults have reported correlation values that ranged from 0.5 to 0.8 and in children one study reported a correlation of 0.83 [107,112].

1.4.2.3 Adverse consequences of excess visceral adipose tissue accumulation in children and adolescents

Several studies have demonstrated the detrimental effects of visceral adipose tissue on glucose and lipid metabolism [45,117-119]. Visceral adipose tissue (VAT) was found to correlate with glucose intolerance independently of total adiposity and subcutaneous adipose tissue (SAT). Furthermore, no correlation was found between total adiposity and glucose intolerance after controlling for VAT. Longitudinal analysis has also shown that an increase in VAT has detrimental effects on insulin secretion and action [117]. A relationship between VAT and disturbances in lipid and lipoprotein metabolism has also been demonstrated with particularly strong associations observed with high plasma TG and low HDL-C [46,120].

Significant correlations between VAT and risk factors such as fasting insulin and lipid concentrations have also been observed in lean and obese children at various stages of growth [45,110]. For instance, cardiovascular risk factors have been linked to the amount of IAAT in obese adolescent girls [100] and children aged between 7 and 11 years [121]. The study in obese adolescent girls reported a positive correlation between TG and basal insulin concentrations and directly measured IAAT whereas an inverse relationship was observed with HDL-C concentration. This study also found inverse relationships between SAAT and LDL-C and between femoral adipose tissue and LDL-C and TG concentrations [100]. However, IAAT and metabolic disturbances are not as strongly and consistently associated in children as they are in adults [46,122]. The weaker relation in children could be due to the fact that physiologically smaller amounts of IAAT are present before adulthood [110,123,124] and as discussed earlier, a rapid change in fat patterning is apparent during growth and sexual maturation with differences observed between males and females [46,107].

In addition to having an independent effect on individual risk factors, there is evidence to indicate that VAT has a role in forming a link between the many components of metabolic syndrome through its ability to promote insulin resistance [42]. Although the exact mechanism by which VAT poses a greater risk is yet to be understood, the vascular anatomy and metabolic activity of this fat depot are thought to be the major factors that predispose to obesity related complications [125].

1.4.2.4 Mechanisms linking specific fat depots and adverse risk factors

Although the mechanisms by which VAT is linked with metabolic, endocrine and cardiovascular risk factors are not clearly understood, a number of hypotheses have been proposed to explain these relations. The central focus of these hypotheses is that alterations in these risk factors are driven by insulin resistance.

Evidence to indicate that obesity is fundamental to the development of insulin resistance/hyperinsulinaemia has been reported in adults, children and adolescents [66,80,126,127]. The association between body fat and insulin sensitivity was apparent in a study that reported a strong inverse correlation between the two parameters in prepubertal children of varying degrees of body fatness [46]. It was also shown in children that 55% of the variance in insulin resistance was explained by total adiposity once confounding factors such as age, gender, ethnicity and pubertal stage had been adjusted for [127]. Another study found obese children to have hyperinsulinaemia and peripheral insulin resistance which was accompanied by insulin stimulated glucose that was 40% lower than in non-obese children [128]. Furthermore, the presence of acanthosis nigricans, a cutaneous marker of insulin resistance, has been reported in overweight (percentage weight-for-height: 141-209%) children [37].

There is evidence in adults and children to suggest that abdominal obesity, characterized by increased lipid deposition in VAT, is more closely associated with adverse risk factors than overall obesity [100,129]. For example, a stronger correlation between visceral fat and insulin sensitivity and lipid derangements was reported in obese adolescent girls. Another study in obese children and adolescents found VAT to be more strongly related (negatively) to insulin sensitivity than total fat mass [130].

There are three potential hypotheses linking VAT with adverse risk factors. The first hypothesis relates to the recent recognition of adipose tissue as a secretory organ (that expresses free fatty acids and a wide variety of peptides), the second relates to the "portal hypothesis" and the third relates to the ectopic fat storage syndrome. It is important to note that these hypotheses may not be independent of each other and could actually be occurring simultaneously.

1.4.2.5 Hypothesis 1: Adipose tissue as a secretory organ- expression of adiocytokines

Adipose tissue is thought to play a fundamental role in the pathogenesis of insulin resistance because it is a source of metabolites, hormone and proteins (adipocytokines) that affect various steps of the insulin action pathway. Adipocytokines produced and released by adipose tissue include adiponectin, tumour necrosis factor-α (TNF-α), interleukin-6 (IL-6), leptin and resistin [42]. One hypothesis that may explain the link between VAT and risk factors is that VAT may overexpress some of these factors [47].

Although there is a great degree of uncertainty regarding the mechanisms by which these factors are involved in the pathogenesis of insulin resistance, studies in rodents and obese subjects have provided some indication of their primary roles. The roles of adiponectin, IL-6 and TNF-α are discussed below.

Adiponectin is an adipocyte-derived hormone that has multiple effects on skeletal muscle, liver and blood vessels. It is thought to possess anti-diabetic and anti-inflammatory properties and its circulating levels tend to decrease with increasing progression of obesity and T2DM [131]. However, there is evidence that the degree of hypoadiponectinaemia has a closer association with the level of insulin resistance and hyperinsulinaemia than to the degree of obesity and glucose intolerance [131].

IL-6, an inflammatory cytokine, is secreted by the SAAT where production increases with progression of obesity but decreases with weight loss. However, more IL-6 is produced in the VAT than SAAT in the morbidly obese state. Following production of Il-6 in the VAT, it is delivered directly to the liver through the hepatic portal vein where it is thought to contribute to the onset of metabolic syndrome. Although the exact mechanisms by which it does this is unclear, studies in rodents have shown that IL-6 augments the production of very low density lipoproteins (VLDL) and increases the synthesis of acute-phase proteins in the liver [131].

There is still much controversy regarding the level of contribution made by the human adipocyte in the production of TNF-α, another inflammatory cytokine. Although its mRNA has been detected in the adipocytes, TNF-α has not been detected in the veins draining the SAAT and differences in TNF-α expression have not been reported

between VAT and SAAT [131]. Although the role of this cytokine in human obesity remains unclear, there is some evidence in obese women that overproduction of TNF-α may influence the ability of insulin to stimulate glucose transport in the adipocytes [132].

Levels of circulating leptin, an adipocyte-derived hormone, are higher in the overfed state during which it prevents weight gain by increasing energy expenditure and inhibiting energy intake. However, in the fasting state leptin levels are lower which stimulates feeding, decreases energy expenditure and promotes energy conservation by modulating neuroendocrine and immune function. Leptin regulates food intake and energy expenditure through its effects on the hypothalamus. The contribution of VAT to circulating leptin levels are thought to be modest given that mRNA expression of the hormone was found to be lower in VAT adipocytes than in SAAT and because VAT adipcoytes in obese men and women have shown to produce less leptin [131,132].

Although these adipocytokines are thought to play an important role in pathological states such as obesity and T2DM, their precise metabolic roles and mechanism of actions in human stills remains a controversial subject.

1.4.2.6 Hypothesis 2: The "portal" hypothesis

The second hypothesis linking VAT with adverse risk factors relates to the "portal hypothesis" [125]. This hypothesis relates to the rapid flux of free fatty acids (FFAs) from VAT to the liver because of its greater lipolytic activity compared to SAT. The variation in lipolytic activity between the two fat depots is attributed to the pronounced lipolytic effects of catecholamines and weaker antilipolytic effects of insulin on VAT [133,134]. Insulin inhibits lipolysis (and the resulting FFA efflux) and promotes fat storage by promoting the process of re-esterification which increases the re-synthesis of TG from FFAs. Catecholamines however induce a lipolytic effect on VAT by controlling blood flow and modulating the secretion of insulin [131,132].

The portal hypothesis states that FFAs are released from VAT and are drained into the liver because of the direct access via the portal vein. The flux of FFAs into the liver is though to increase hepatic glucose production. This is thought to be a consequence of FFA oxidation within the liver which results in the production of

acetyl-CoA. High levels of acetyl-CoA activate the enzyme pyruvate carboxylase which is required for the conversion of pyruvate to oxoloacetate, the latter substrate being an intermediate in the gluconeogesis pathway. FFA stimulation of hepatic gluconeogenesis is also thought to involve NADH which is necessary for the conversion of 1,3-biphosphoglycerate to glyceraldehyde 3-phosphate and ATP which is needed as an energy source [135,136].

Elevated levels of FFAs are also thought to interfere with hepatic clearance of insulin which in turn leads to peripheral hyperinsulinaemia. Insulin clearance occurs primarily in the liver and is a receptor-mediated action that involves insulin receptor binding followed by internalization and finally degradation of insulin. Studies in rats have shown that FFAs may impair insulin binding due to a decrease in receptor number (as a result of internalization) and degradation of insulin by inhibiting the enzymes responsible for this action [131,136,137].

The greater flux of FFAs in the liver also affects lipoprotein metabolism as it increases the production and secretion of TG rich VLDL particles in the circulation. The release of VLDL in the circulation is therefore regulated by the synthesis of TG in the liver, the availability of FFAs and the reduced degradation of apolipoprotein B, the structural protein of VLDL [131].

Taken together, the above describes the profound effects of fatty acids on the hepatic gluconeogenesis, insulin clearance and lipoprotein synthesis which in turn leads to hyperinsulinaemia, insulin resistance and hyper triglyceridaemia. In this way the portal hypothesis proposes that portal adipose tissue generates some of the most powerful risk factors for the development of CVD, stroke and T2DM [131,136].

1.4.2.7 Hypothesis 3: The ectopic fat storage syndrome

A more recent area of interest has been in the deposition of fat in organs such as liver and skeletal muscle, termed ectopic fat storage syndrome, which possibly contributes to insulin resistance and disease risk. The ectopic deposition of fat in the skeletal muscle is critical in this context as it is a major site of insulin action. Insulin signalling is thought to be impaired as a consequence of fat accumulation and metabolite secretion which lead to a reduction in muscle glucose uptake and decrease in insulin mediated suppression of hepatic glucose production. Evidence for a correlation

between insulin resistance and ectopic deposition of fat in the muscle has come from studies in adults, children and animals [42,47,536].

1.4.3 Persistence into adulthood

One of the most significant consequences of childhood and adolescence obesity is its persistence into adulthood. This relationship has been consistently demonstrated in several large-scale longitudinal studies. The Bogalusa Heart Study represents one such study where 58% of overweight adolescents (BMI for age and sex >75th percentile) were found to remain overweight 12 to 14 years later [138]. Similarly, the Fels longitudinal study (Ohio, USA) found that overweight and obese adults had a higher BMI in childhood and adolescence compared with non-overweight adults [139]. Furthermore, there is evidence to show that the likelihood of persistence is associated with the severity and the age at which obesity is present. Of further concern is the finding that the risk of developing obesity-related co-morbidities in adulthood is increased in those with a longer duration of overweight or obesity [140].

1.4.4 Respiratory problems

1.4.4.1 Asthma

The rise in the prevalence of overweight and obesity is concurrent with the rise in prevalence of asthma both in adults and children [141]. A positive association between asthma and obesity has been reported in adults with some evidence of a gender bias in this association. The evidence in children and adolescents is not as consistent as in adults as only a few studies have reported a positive association between asthma/asthma symptoms and obesity [142-144] whereas others have found no association between the two disorders [145]. Although there is more heterogeneity in the strength and direction of this association in children, prospective studies in adults have shown that obesity antedates or precedes the development of asthma [146-150]. The causal relationship between obesity and onset of asthma is thought to be multifactorial with mechanisms relevant to this association including the mechanical effects of obesity, enhanced immune response, genetics, hormones and environmental influences such as diet, exercise and intra uterine development [151].

1.4.4.2 Obstructive Sleep Apnoea Syndrome (OSAS)

The past decade has seen a rise in the number of obese children diagnosed with OSAS. The potentially serious implications of this syndrome are also better understood. OSAS is characterized by partial or complete obstruction of the upper airways that occurs intermittently during sleep. The obstruction is caused by the inability of pharyngeal muscles in maintaining upper airway patency thereby leading to a disruption in normal gas exchange, intermittent hypoxia and hypercapnia and sleep fragmentation [146].

Studies in adults have provided robust evidence to indicate that obesity is a risk factor OSAS. Such a pattern has also been observed in children with evidence of a proportionate increase in severity of OSAS with degree of obesity. Some of the earlier evidence in this area demonstrated the presence of polysomnographic abnormalities in obese children and the presence of complete airway obstruction [152,153]. A dose response relationship demonstrated that every $1kg/m^2$ increase in BMI above the mean for age and gender was associated with an increase in risk in OSAS by 12% [154]. Further studies conducted globally have provided additional evidence to suggest that overweight and obese children are at increased risk of developing OSAS [155].

OSAS in children has potentially detrimental consequences on multiple organs and systems. Evidence of an adverse effect on somatic growth, neurocognitive development and quality of life has been reported. Furthermore, risk of cardiovascular morbidity is likely to be raised as an increased prevalence of systemic hypertension, alterations in cardiac geometry and changes in blood pressure regulation have been observed in children with OSAS [155].

1.4.5 Gastro Intestinal problems

1.4.5.1 Gall stones

The likelihood of formation of gall stones in obese individuals is increased due to increased biliary excretion of cholesterol relative to the excretion of bile acids and phospholipids. The consequent supersaturation of bile with cholesterol results in formation of stones [156]. Supersaturation of bile acid has been demonstrated in obese females from the age of 13 years [157]. In children, obesity has been

associated with 8-50% of cases in children [158-160]. Furthermore, majority of the cases that were not explained by underlying medical conditions were accounted for by obesity. More recent evidence showed a similar pattern of higher prevalence of gallstones in a sample of obese children compared to an unselected sample. Furthermore, a comparison between the obese children demonstrated a higher BMI z-score in children with gallstones compared to those without [161].

1.4.5.2 Non-Alcoholic fatty liver disease

The presence of Non-alcoholic fatty liver disease (NAFLD) has been observed in obese children due to increased rates of lipolysis and insulin resistance. As NAFLD has no symptoms, diagnosis can only be confirmed by conducting a liver biopsy or predicted using indirect measures including a liver function test (circulating transaminase levels) and ultrasound test. The non-invasive nature of indirect test has enabled prevalence levels of NAFLD to be estimated in children. In 1989, prevalence of fatty liver in Japanese school aged children was estimated at 2.6%. Collective analysis of studies conducted in Japan, Italy, China and America demonstrated that the prevalence of fatty liver in obese children ranged between 10% and 77% [156]. The wide range of the prevalence level is a function of predictions based on indirect tests. Further studies have demonstrated that suspected fatty liver is more common in obese boys than obese girls and in Mexican American and African Americans although the latter groups are generally underrepresented in studies [156].

1.4.6 Orthopaedic and neurological problems

Orthopaedic problems are related to the presence of unfused growth plates, immature cartilage and softer cartilaginous bones in children and adolescents as these have not evolved to support large amounts of body weight [12]. The two specific orthopaedic conditions that have been associated with obesity include Blount's disease identified in younger children and Slipped capital femoral epiphysis (SCFE) identified in early adolescents. Although the presence of Blount's disease in not common in childhood obesity, one study found that 60% to 80% of children with the condition were obese [162]. The incidence of SCFE has been reported as 3.4 per 100 000 children and 50% to 70% of patients with SCFE are obese [163].

Pseudotumor cerebri (PTC) is a neurological condition that is characterised by increased intra cranial pressure of the cerebrospinal fluid in the absence of clinical, laboratory or radiological evidence of an intra-cranial space occupying lesion. Although the exact cause of PTC remains unclear, a greater frequency of occurrence has been observed in obese adults and children [164,165].

1.4.7 Psychological and psychosocial consequences

The adverse psychosocial effects of obesity are commonly presented during young adulthood and include fewer years of education, lower family income, higher poverty rates and lower marriage rates [166]. It has been widely assumed that obese individuals suffer from poor psychological wellbeing because obesity is a stigmatized condition in which social exclusion and discrimination are often entrenched in the lives of affected individuals. This is especially evident in the attitudes of both children and adults who tend to stereotype obese individuals as being lazy, ugly and stupid [167].

Aspects of psychological well being shown to be compromised in obese children and adolescents include body image (or body dissatisfaction), self esteem and emotional well being [168]. There is strong evidence to show that body dissatisfaction is associated with obesity in children and young people. This evidence has been demonstrated both in clinical populations of obese children seeking treatment as well as in community based studies. Self esteem is defined as a "balance between a person's attainments and their goals or aspirations" and can be measured globally (e.g I think I am a horrible/great person) using factors that are related to health psychological development or using a specific domain (e.g I think I have a beautiful/ugly body) [168]. Studies on clinical samples have demonstrated lower self esteem in obese compared to normal weight non-clinical (those not seeking treatment for obesity) controls [169] whereas community based studies have rarely found a strong association [168]. However, prospective studies have demonstrated that this association works in both directions where lower self esteem has been shown in children with a higher BMI but also that a higher BMI leads to lower self esteem. The evidence linking depression and obesity remains rudimentary with the majority of the evidence suggesting a modest to negligible association [168].

These associations have been reported with respect to a number of moderators including age, gender and ethnicity. Possible mediators of these associations include socio-cultural attitudes and norms and weight related bullying. An example of an association and its moderators and mediator is the association between body dissatisfaction and obesity which appears to be more apparent in girls and adolescents. The association in these groups could therefore be reflecting the socio-cultural pressures on these groups to conform to an idealized physique [166,168].

Although the above evidence demonstrates the negative psychological consequences of obesity, it is important to note that emotional problems are not an inevitable consequence of obesity.

1.4.8 Obesity-related morbidity in South Asians

It was recognized in the 1950s and early 1960s that people with origins from the Indian subcontinent (South Asians) appeared to be at greater risk of developing cardiovascular diseases following migration to urban environments. These findings have since been confirmed in immigrant South Asian populations residing in several countries. In the UK, three separate analyses of the 1971 census showed that CVD related mortality rates were 15 to 60% greater in those born in the Indian subcontinent compared to indigenous English and Welsh populations [170-173]. Further evidence was apparent from disparities in rates of decline of CHD between 1971 and 1991 with a greater decline observed in white Europeans compared to men and women of South Asian origin [171]. Findings on morbidity data match those of mortality data where local studies and national surveys have reported prevalence levels of CHD (angina and heart attack) and stroke in South Asians that were either similar or higher than that in reference white or general populations [174,175].

A consistently greater prevalence of T2DM has also been reported in South Asian immigrant and urban Indian populations. The Southall Diabetes survey reported a three to six fold greater prevalence of T2DM in immigrant South Asians compared to Europeans residing in the UK [176,177]. Furthermore, a comparison of immigrant and native Indians showed that the prevalence of T2DM was greatest in the former group (20%), followed by the native urban population (5-10%) and lowest in the rural Indians (2-5%). This was compared to a prevalence of 4% in white Europeans [175]. A mirroring pattern of higher prevalence of T2DM has also been reported in children

from South Asian origin. The first cases of T2DM in the UK were eight overweight (percentage weight-for-height: 141-209%) children aged 9 to 16 years all from South Asian backgrounds with a family history of T2DM [37]. The greater risk in South Asian children was highlighted in a UK survey which reported a 14 times greater risk of developing T2DM in this group than in white children [39]. More recent figures further confirm these finding with the incidence of T2DM reported to be 3.5 times greater in South Asians than in white children in the UK [178].

The role of conventional risk factors including hypertension, hyperlipidaemia and cigarette smoking in explaining the greater prevalence of coronary heart disease (CHD) in South Asians has been investigated by several studies. Earlier studies concluded that these risk factors alone did not explain the higher CHD mortality rates in this ethnic group as consistently high levels of smoking, blood pressure and cholesterol were not found in all South Asian groups [173]. More recent studies in migrant and urban (residing in South Asian countries) populations have demonstrated the importance of these risk factors in these groups although the extent of contribution in explaining CHD risk remains unclear [173]. The role of novel risk factors in contributing to the increased CHD risk in South Asians has also received much interest. South Asian adults have been reported to have higher levels of Lipoprotein(a), an independent marker of CHD formed from the protein apolipoprotein which is thought to inhibit fibrinolysis and influence cholesterol synthesis [176]. Additionally, recent studies have observed higher levels of prothrombotic factors (fibrinogen, PAI-1), C-reactive protein (CRP) concentrations (a sensitive systemic marker of inflammation) and lower concentrations of adiponectin in South Asians than in white European adults [176,179]. A more adverse lipid profile has also been observed in South Asian children where TG and fibrinogen concentrations were reported to be greater than in white children [180]. There is also evidence of lower birth weight and greater weight gain during childhood [181,182] in South Asians both of which have been associated with adverse risk factors and increasing the risk of CVD and T2DM [183,184].

Of particular interest is the role of insulin resistance in contributing to the greater propensity towards developing diabetes and CHD in South Asians. Evidence for a greater tendency of South Asians to develop insulin resistance is well established in adults and widely reported in children. The "Ten Towns Heart Health Studies" showed

that South Asian prepubertal children residing in the UK were more insulin resistant than their age matched white European counterparts [180]. Additionally, hyperinsulinaemia is shown to be greater in South Asians as early as birth in a study which found higher cord insulin levels in native Indian babies compared to children born in London [185].

It has been proposed that the higher prevalence of insulin resistance in South Asian adults could be explained by the greater tendency of this group to accumulate abdominal fat [186]. Adult Indian Asians have shown to have greater deposition of fat in the abdominal region than Europeans for a given degree of generalized obesity [187,188]. Indeed a pattern of fat distribution characterized by greater trunk to peripheral ratio has also been shown to be present as early as birth and early childhood in Indian Asians [189] and in South Asian adolescents in the UK [190]. Furthermore, strong correlations between insulin resistance and abdominal obesity have been demonstrated in adults and children from this ethnic group. However, there still remains some debate as to whether the greater tendency for abdominal fat deposition fully explains the increased susceptibility towards insulin resistance in migrant and native South Asians.

The role of general and abdominal fat has been questioned in studies that were unable to explain ethnic differences in insulin resistance by differences in adiposity. One such study in adults showed that Asian Indians remained excessively insulin resistant even after adjusting for both total and truncal body fat [191].This finding is supported by evidence from a study which found that insulin resistance was greater in Asian Indians than in Caucasians even in the absence of obesity [192]. Evidence in children comes from the UK where South Asian children aged 8-11 years were found to be more insulin resistant than white European children, independent of general or abdominal adiposity, suggesting that the former group are intrinsically more insulin resistant [180]. It has been postulated from such evidence that South Asians are inherently more insulin resistant that Caucasians and thus may have a genetic predisposition to developing this characteristic. The likelihood of this hypothesis has been given credibility from studies which found that the greater insulin resistance in South Asian adults was not explained by inter-ethnic differences in environmental determinants including diet and physical activity [193,194].

Other hypotheses explaining the greater susceptibility to obesity-related morbidity in South Asians include the thrifty genotype and thrifty phenotype hypotheses. The thrifty genotype concept was proposed by the geneticist James Neel in a paper entitled "Diabetes Mellitus: a 'thrifty genotype' rendered detrimental by progress" [195]. This hypothesis states that individuals exposed to an environment of limited food supply would have been protected by a predisposition to insulin resistance as this conserves glucose utilized by muscles. However, this predisposition becomes disadvantageous in an environment of plentiful food supply as the decrease in glucose utilization becomes a pathological consequence. The evidence for this hypothesis is thought to go as far back as the dawn of agriculture which subjected populations to periods of intermittent food supplies due to recurrent feast and famine cycles (resulting from unstable climatic conditions and warfare). There is evidence to suggest that the periods of starvation and famine may have had a significant influence on the human genome whereby natural selection could have favoured the selection of thrifty genes by suppressing fertility and having an effect on survival/mortality [196]. However, the current climate of constant food availability and abundance and sedentary lifestyles has led to a mismatch between gene-environmental interactions whereby a metabolism accustomed for survival under 'feast or famine' conditions would eventually lead to obesity and T2DM [197].

The thrifty phenotype hypothesis also known as the 'Barker hypothesis' was proposed more recently and it focuses on the metabolic and structural consequences of foetal undernutrition which increase the risk of developing T2DM and CVD [198]. This hypothesis states that a mismatch between nutritional circumstances characterized by foetal undernutrition and compromised early growth and nutritional abundance during later life can have detrimental effects on cardiovascular health. Birth weight, a poor but popular indicator of foetal nutritional experience and intrauterine growth, was reported to be one of the lowest in native Indian babies (2.7kg) when compared to UK South Asian (3.1kg), Chinese and Afro Caribbean (3.2 kg) and White European (3.4kg) babies [181,199]. A more recent comparison also found birth weight to be lower in Indian babies (2.7kg) than in Caucasian babies (3.5kg) [182]. More extensive analysis of this data showed that underweight Indian babies were characterized by a "thin-fat" phenotype in which subcutaneous fat was spared but abdominal viscera and skeletal muscle were sacrificed in the adverse intra-uterine environment. Thus

although these babies were considered to be "thin", they were in actual fact relatively fat. Furthermore, a longitudinal analysis of South Indian babies has shown that this phenotype persists in childhood and thus may be critical to the higher risks of CVD and T2DM seen in South Asian adults [189].

The role of upper body subcutaneous abdominal fat (SAAT) in the genesis of the adverse metabolic and endocrine profile in South Asians has also been proposed more recently. Evidence for this has become apparent from studies reporting greater subcutaneous body fat in South Asian adults compared to Caucasian counterparts. One such study found truncal subcutaneous fat to be greater in Asian Indian men compared to Caucasian men despite having similar body fat content [191]. It was also observed that Asian Indians with higher degrees of insulin resistance differed from Caucasians only in this parameter of body composition. A similar finding of greater truncal subcutaneous fat was also reported in premenopausal women of Asian Indian and Pakistani origin compared to American women of European descent [200]. This greater abdominal subcutaneous fat in Asian Indians compared to Caucasians has also been demonstrated directly using magnetic resonance imaging [187]. Evidence in children has also been reported with greater truncal subcutaneous fat observed in Asian Indians compared to White Caucasians and Blacks [201]. The link with adverse risk factors has been shown through the association between prevalence of insulin resistance and high levels of total and abdominal adiposity and truncal subcutaneous fat in urban Asian Indian adolescents [202]. Although the exact mechanism by which deep abdominal subcutaneous fat poses a risk remains unclear, it is thought that the release of free fatty acids from this depot, as with visceral adipose tissue, may be associated with the metabolic complications of obesity [203].

A more recent hypothesis put forward to explain the greater susceptibility in South Asians to obesity related morbidity is the adipose tissue overflow hypothesis [204]. This hypothesis describes the greater risk in South Asians in terms of the storage capacity of three fat depots- the primary depot consisting of superficial subcutaneous adipose tissue and the two secondary depots being the deep SAAT and VAT. It has been proposed that South Asians have a relatively smaller storage capacity in the primary compartment and thus increasing obesity leads to an earlier utilization of the metabolically active secondary fat depots. This hypothesis has been challenged because the concept of overflow is thought to be to be too simplistic as it does not

seem to explain the complex interactions between whole body obesity, central obesity and adverse consequences in other ethnic groups [205]. Instead it was proposed that the disproportionately greater allocation into the visceral fat depot in South Asians could have an evolutionary explanation. This explanation has been termed the El Nino hypothesis and it considers the drastic consequences of global climate patterns and geographic peculiarities characteristic to the El Nino, on agriculture and the survival of Indian farmers [205]. The resulting cycles of famine and chronic energy deficiency are though to be associated with the increased allocation of dietary energy in the visceral fat depot.

Although the greater prevalence of obesity-related morbidity in South Asian adults and the greater prevalence of adverse risk factors in children is widely established, there is still much speculation regarding the mechanisms behind the greater risk in this ethnic group.

1.4.9 Obesity-related morbidity in Blacks (African Americans, native Africans, Black UK Africans & UK Black Caribbeans)

The rise in the prevalence of obesity in UK African-Caribbeans [206] and African Americans (AA) [207] is concurrent with an increase in T2DM and CVD in adults. Despite decreases in overall mortality from CVD in the United States [208], mortality rates from CHD remain higher in AA than in whites [209]. The epidemiology of CVD in black Africans and black Caribbeans in the UK differs to that of AA as the prevalence of CHD is lower but levels of stroke and hypertension are higher in these groups relative to UK Caucasians [210]. Furthermore, the incidence of stroke has been reported to be two times higher in UK Blacks [211] and US Blacks [212] than in the local white population. The UK Health Survey for England (2004) also reported a higher prevalence of T2DM in Black Caribbeans (10.0% men, 8.4% women) than in the general population (4.3% men, 3.4% women) [25]. AA adults also have a disproportionately high prevalence of T2DM with the risk of development being two times greater than in white Americans [213]. Furthermore, disparities within African groups have also been observed with the prevalence of T2DM reported to be 12 times greater in African Americans than in native Africans [214].

Similar to the patterns observed in adults, the incidence of T2DM in South Asian and Black children was recently reported to be 3.5% and 11% greater respectively than in

white children [186]. The development of T2DM may be preceded by disturbances in insulin and glucose metabolism for which differences between black and white children and adolescents have been widely documented. Measures of insulin have shown to differ between the groups with greater fasting insulin levels and acute response to glucose observed in black compared to white children [83,224]. Generally, black children and adolescents are reported to be relatively hyperinsulinaemic and insulin resistant compared with their white counterparts [225,226]. These differences in insulin measures have been observed despite black children having lower levels of VAT. Furthermore, the risk of developing T2DM at an early age is greater in black children and independent of general obesity and fat distribution [83, 227]. It has also been reported that hyperinsulinaemia, measured as the acute phase insulin concentration during a glucose challenge, is greater in black than in white children for a given degree of insulin sensitivity. Further investigations into this finding have shown that hyperinsulinaemia in black children that is unexplained by insulin resistance is more closely associated with greater insulin secretion than with lower hepatic insulin clearance [224].

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unexplained by insulin resistance is more closely associated with greater insulin secretion than with lower hepatic insulin clearance [215].

Based on the evidence above and that acquired more recently from the UK Prospective Diabetes Study Group (UKPDS), it has been suggested that heterogeneity exists between ethnic groups with respect to the mechanisms involved in the pathogenesis of T2DM [219]. More specifically, it is believed that the predominant mechanism leading to the development of T2DM in South Asians is insulin resistance, whereas in Blacks it is pancreatic β cell dysfunction [219].

It is widely acknowledged that VAT adjusted for whole body fatness is lower in black adults [220], adolescents [221] and prepubertal children [112]. One study in obese adolescents found black subjects had 30% less visceral fat than white peers despite having similar BMI and total body fatness [221]. The lower VAT has been observed in both boys and girls across a wide range of whole body fatness [112]. Furthermore, longitudinal analysis has shown that accumulation of visceral fat was greater by 1.9cm^2 per year in white (baseline unadjusted mean VAT was 27.3cm^2 in boys and 32.6cm^2 in girls) than in black (baseline unadjusted mean VAT was 24.7cm^2 in girls and 32.6cm^2 in boys) children [111]. Since VAT has been associated with an atherogenic lipid profile, the more favourable lipid profile in black adults could be explained by the lower VAT and subsequent reduced delivery of FFA to the liver [221]. Even in the presence of glucose intolerance, levels of TG and HDL-C were lower and higher respectively in black adults than in Europeans [222]. The differential impact of VAT on cardiovascular risk factors in black and whites was demonstrated by a study that found no differences in fasting lipids between black obese adolescents with high and low levels of VAT. In contrast, white obese adolescents with high VAT had higher concentrations of total cholesterol, LDL-C and TG than those with lower levels of VAT [221]. A less atherogenic lipid profile of lower TG and higher HDL has also been demonstrated in both AA adults and children compared to their white counterparts. The more cardioprotective profile in AA's is thought to be a consequence of the greater post challenge insulin which results in the suppression of lipolysis and in turn limits the availability of FFA for the synthesis of TG [223].

More recent work has revealed the role of SAAT in contributing to insulin resistance in adults and children. Larger SAAT stores in black compared to white adults has drawn

attention on the importance of this depot in influencing insulin and lipid metabolism [203,220]. Studies in white and black adults have shown that both VAT and SAAT contribute to insulin resistance with conflicting findings on the degree of influence of each fat depot [224,225]. The correlation between SAAT and insulin sensitivity has also been examined in African Americans with some studies reporting a correlation with insulin-mediated glucose disposal [226] whereas others found no significant association between these parameters [227]. It has therefore been suggested that SAAT may have an important role in the pathogenesis of insulin resistance and associated consequences in AA, particularly in women, however further work still needs to be done to confirm the significance of these findings [226].

1.5 Aetiology of obesity

The rising trends in the global prevalence of obesity and the associated co-morbidities in populations has compelled governments and health agencies to invest in strategies to combat this epidemic. However, the development of strategies to prevent and manage obesity in order to reduce both current and future prevalence levels requires an understanding on the causation of obesity.

At a physiological level, it has been established that obesity arises when energy intake exceeds energy expenditure over a prolonged period of time. This can be clearly illustrated using the energy balance equation (Equation 1.2) which shows that a positive shift in the equation leads to an increase in energy stores [228,229].

Equation 1.2: Change in Energy stores= Energy Intake – Expenditure [228]

However, progress in obesity research has demonstrated that this is too simplistic an understanding of the causes and that the factors influencing both parts of the energy balance equation are multifactorial by nature. A more practical and current understanding of the aetiology of obesity is that is it is a complex physiological state which is influenced by a range of factors such as genes, hormones, neural influences, behaviour, societal and obesogenic influences and the interactions between these factors [229].

The Foresight report, a review on obesity developed by a Government-assigned team of experts, confirms just how complex the aetiology of obesity is with its obesity

system map [230]. Visual inspectional alone of this system map is sufficient to demonstrate just how complex this issue is. However, closer analysis of the map shows that all the variables and determinants of obesity are presented within seven different themes (food production, food consumption, physiology, individual physical activity, physical activity environment, individual psychology and social psychology) and the complex interrelationships between and within these themes clearly highlighted.

The genetic and broader environmental factors associated with the development of obesity will be considered.

1.5.1 The genetic influence

1.5.1.1 Heritability and gene-environment interactions

The heritability of a trait such as a tendency to gain excess body fat describes the amount of variation in that trait that can be explained by genetic factors. The heritability of obesity can be studied in terms of total body fatness or body fat distribution, although the former is more commonly reported. Studies on monozygotic and dizygotic twins have provided estimates on the heritability of whole body fatness, expressed as BMI. These studies have estimated the heritability of BMI to range between 64 and 84% [231]. However, because such studies have been unable to separate the independent effects of genetic influences and the shared environmental influences in determining heritability, further work on monozygous twins reared apart and adoption studies have been conducted. No difference in heritability was found in one study between twins reared apart and twins reared together [232]. Furthermore, estimates of heritability from a number of studies on twins reared apart have ranged between 50 and 70% [233,234].

Adoption studies, particularly evident from the Danish adoption register, have also demonstrated the importance of genetic influences. These studies found that across the range of fatness, a strong and statistically significant relationship was evident between the BMI of the adoptee and their biological parents whereas no such relationship was found between the adoptee and their adoptive parents [235]. Further work from the Danish register also showed a stronger correlation between the BMI of

adotees and their full siblings (reared apart by biological parents) whereas a weaker relationship was found with half-siblings [236].

It is thought that the genetic influence on fatness and thus the aetiology of obesity operates through susceptibility genes. The expression of these genes can be attenuated or exacerbated by non-genetic factors, hence, although these genes increase the possibility of developing a characteristic, they are not necessary for its development nor are they solely able to explain the expression of the characteristic [237]. Support for the susceptibility-gene hypothesis came from studies in monozygotic twins exposed to episodes of positive and negative energy balance. Findings showed a greater variation in the rate of weight gain, proportion of weight gain and site of fat deposition between twin pairs than within pairs. This demonstrated that individuals can vary in their risk of becoming obese in any given set of environmental conditions based on genetic susceptibility [238,239].

1.5.1.2 Monogenic disorders leading to human obesity

Although obesity tends to run in families, it has been shown that segregation of genes in the majority of cases does not follow a Mendelian pattern of inheritance. However, there are about 30 single gene disorders in humans of which obesity is a characteristic. One such example is the Prader-Willi syndrome (PWS) which is characterized by short stature, upper body fatness, mental retardation and hypogonadism. This condition is usually associated with familial inheritance and is a result of a deletion on the paternal fragment of chromosome 15. Another such disorder is the Bardet-Biedel syndrome (BBS) which is an autosomal recessive disease although it much more rare than the PWS [237,240].

Other monogenic disorders that are not characterized by developmental anomalies or mental retardation include congenital leptin deficiency, leptin receptor deficiency and Melanocortin 4 receptor deficiency (MC4R). Leptin deficiency has been well described in the laboratory bred ob/ob mouse which has a mutation in the ob gene rendering it unable to produce its protein product leptin from adipose tissue. Leptin acts via its receptor in the hypothalamus to stimulate the anorexigenic (catabolic) pathway and inhibits the orexigenic (anabolic) pathway which control appetite and regulate metabolism and energy expenditure. Administration of recombinant leptin in the ob/ob mouse has shown to result in a reduction in body weight, body fat and food

intake. The presence of leptin deficiency has also been reported in a small number of individuals of Pakistani and Turkish origin who were homozygous for the mutation and presented with early onset severe obesity and hyperphagia [237,240].

Mutations in the Melanocortin receptor 4 (MCR4) gene has also been shown to be a cause of obesity and its metabolic complications The MC4R is highly expressed in the central nervous system and is closely related to energy homeostasis. Targeted disruption of this receptor in rodents has been reported to result in severe hyperinsulinaemia, increased food intake and linear growth and obesity. The presence of a heterozygous mutation in humans has also shown to lead to a dominantly inherited obesity syndrome. Furthermore, evidence from studies on Danish populations has shown that MC4R deficiency is the most common monogenic disorder that leads to obesity [240].

1.5.2 Environmental influences

Although the heritability of the BMI has been demonstrated in the studies on twins, the importance of the environment in contributing at least, partially, to the epidemic of obesity has been evidenced on the basis of three arguments. Firstly, it is argued that the drastic rise in obesity over the last few decades cannot be explained by the slower process of single gene defect transmission between generations. Evidence to support this argument can be drawn from that fact that the human genome has remained very stable over the last 12 000 years [196,241]. Secondly, studies have shown higher levels of obesity in migrants than in their counterparts residing in their country of origin. Additionally, higher levels of obesity have also been demonstrated in the offspring of immigrants than in their parents. This argument gives strong evidence for the influence of environmental factors on the levels of obesity. Finally, the adoption of a more "Western diet" and lifestyle and reductions in levels of physical activity has resulted in a rise in the prevalence of both adult and childhood obesity [229,242].

Although it has been established that the environment has an important role in the aetiology of obesity, the precise influence of the environment remains a complex issue because of the vast numbers of factors involved. Furthermore, interactions between these factors both at an individual and population level can further complicate the understanding of the aetiology of obesity. For ease of understanding,

environmental factors can be divided into those that affect physical activity levels, dietary intake and the general obesogenic environment.

1.5.2.1 Physical activity levels and sedentary behaviour

Physical activity is defined "as any bodily movement produced by skeletal muscles and which results in energy expenditure beyond basal energy expenditure". Energy expenditure (EE) is defined as "an expression of total-body metabolism during specific time periods [107]. Total energy expenditure (TEE) is made of up of three components: Basal metabolic rate (BMR), Physical activity (PA) and thermogenesis. Of the three components, BMR (60-75%) and thermogenesis (5-10%) are relatively constant whilst PA is the most variable component which can range from 0% in totally inactive individuals to more than 50% of total energy expenditure in some elite athletes [243].

It has therefore been suggested that a decrease in PA or increase in inactivity/sedentary behaviour is possibly the major factor that accounts for a reduction in EE which in turn leads to a positive energy balance and increased energy storage and obesity. There is some indirect evidence which appears indicate that a secular decrease in physical activity levels is concurrent or prior to the temporal increase in obesity. This evidence focuses on the technological advances in motorized transport and labour saving devices (since the 1960's) which have led to a decrease in the need for high levels of physical work within the home and occupational setting as well as part of travel. It has become evident that such advances have influenced activity levels both in adults and in children. An example of this can be drawn from the National Transport Survey (Department of Transport, 2000), which found that the percentage of children under 17 years walking to school fell from 59% to 49% between 1986 and 1996. It was also shown that a greater percentage of primary school children were driven to school in 1997 (38%) compared to the mid 1980's (16%) [244]. In addition to technological advances, influences such as fear of traffic, busier parental lifestyles, greater parental restriction on outdoor play due to safety issues ("stranger danger") are also thought to contribute to the decline in PA in children.

Aside from the aforementioned indirect evidence, there is very limited direct evidence on trends of PA levels and patterns in children. The only nationally representative

data in the UK comes from the HSE. This survey has obtained information on PA levels in UK children since 1997 using a self-report questionnaire. A comparison between the 2002 and 1997 surveys showed that PA levels in children had not changed during this time period. However, the accuracy of this finding has been questioned given the subjective nature of the data collection method and the fact that the questionnaire had not been validated against a criterion method [245].

The limited information on trends of PA in children has made it difficult to draw conclusions on the r le of PA in contributing to the increase in prevalence of obesity. Nevertheless, this association has been examined in smaller scale studies. One such study- the Health Behaviour in School-Aged Children Study (HBSC) collected data from 34 countries on children aged between 10 and 16 years. This study concluded that PA is related to weight status in older children as a significant inverse relationship was observed between PA and BMI in 88% of the countries [246]. However, this finding has not been consistently seen in other cross sectional and longitudinal studies with some studies reporting an inverse relationship between high levels of PA and weight gain whereas other studies found no association between these factors [243].

There is also evidence that sedentary behaviours such as television (TV) watching and videos is associated with a higher BMI in children and adolescents [247]. TV viewing in childhood and adolescents has also been reported to have an adverse effect on adult health including overweight, smoking, poor fitness and raised cholesterol levels [248]. A number of factors are thought to mediate the relationship between TV viewing and obesity and these include: a reduction in EE as it displaces time spent being physically active, a reduction in resting metabolic rate, increase in consumption of energy dense snacks and foods whilst viewing resulting in increased energy intake and television advertisements encouraging consumption of foods high in sugar and fat [229]. However as with PA, the evidence linking TV viewing and obesity is also inconsistent. For example, the HBSC study showed that although a positive association between TV viewing and obesity was observed in 65% of the countries studied, a greater percentage of young people watched 3 hours or more of TV in the 10 countries with the lowest prevalence of obesity (51%) compared to the 10 countries with the highest prevalence of obesity (46%) [246]. Another study showed that "techno active" boys who had high levels of TV viewing and playing

video games also had above average levels of PA [249]. Thus, the evidence on this association seems to indicate that sedentary behaviour does not necessarily displace PA and that the two factors can be independent of each other so that young people can spend a significant amount of time being physically active and at the same time spend a significant amount of time on screen-based sedentary activities.

It is therefore apparent that the scientific evidence on the associations between PA, sedentary behaviour and obesity remains equivocal. One highly plausible reason for the lack of consiste cy between studies could be the methodological diversity with which data on PA is obtained. There are a large number of techniques of assessing PA in children each with their own advantages and limitations. The most common methods are: PA/EE diaries, activity recalls, motion sensors (pedometers and accelerometers) heart rate monitoring (HR), the Doubly labelled Water method (DLW), indirect calorimetry and the Physical activity level (PAL). The PAL is an indirect measure of assessing daily levels of PA and is an expression of a ratio between Total Energy expenditure (TEE) to Basal Metabolic Rate (BMR) (TEE/BMR). PAL levels have been denoted activity levels and this can range from 1.0 in individuals who are totally inactive to 1.9 and above in extremely active individuals [243]. Despite the large number of techniques, characterizing and quantifying PA levels remains to be a challenge because it is heterogeneous by nature and it can be assessed in a wide range of ways including frequency, duration, intensity and type of activity. Secondly, the assessment of PA levels can often encourage behavioural changes that lead to changes in natural activity patterns. Thus the process of assessing PA per se can affect the precision of data collected.

Although the evidence on the role of PA on weight change remains unclear, guidelines on recommended levels have been published both for adults and children. These recommendations have evolved over the last 20 years from 3 x 20 minutes of continuous to 30 minutes (minimum) or 60 minutes (optimal) of accumulated moderate to vigorous physical activity (MVPA) [254]. The evidence regarding the adequacy of these guidelines in preventing weight gain and reducing prevalence levels of obesity is currently limited. Additionally, there is still much to be understood on the physical activity patterns and levels of young people and whether these recommendations are appropriate given that recent evidence from the Avon

Longitudinal Study of Parents and Children (ALSPAC) study seems to indicate that PA in this group of the population is intermittent and vigorous [250].

1.5.2.2 Dietary determinants of obesity

Paradoxically the rise in the prevalence of obesity in US adolescents is concurrent with decreasing trends in reported energy intake. However, the accuracy of these findings has been questioned due to concerns regarding the accuracy with which dietary intake data is collected [229]. Traditional methods of collecting dietary intake data such as weighed and un-weighed food records, food frequency questionnaires and 24-hour recalls rely on self-reported information and so the accuracy of the information collected is dependent on the accuracy with which the information has been reported. A well-known concept associated with collecting self reported dietary intake data is low energy reporting and this occurs when food intake is reported to be lower than actual intake. It has been consistently demonstrated that obese individuals are more likely to underreport their food intake and so low energy reporting is more prevalent and more severe in this group with the degree of low energy reporting shown to range from 30% to 47% [251]. Thus, given the difficulty of obtaining accurate information on trends of energy intake, it has been speculated that the rise in obesity levels could be due to a greater fall in energy expenditure relative to energy intake [242].

The composition of the diet, in particular a high fat intake, is considered to be a key dietary risk factor for obesity as it has been proposed that a high fat diet overrides the normal physiological regulation of food intake and promotes hyperphagia through its high palatability and low satiating capacity. The association between a high fat diet and obesity has been examined in highly controlled studies, which have shown that a greater provision of energy from fat increases the risk of obesity. Although this association has been seen less consistently in community-based studies, a number of studies in children have shown that a greater proportion of energy from fat was associated with greater body fatness [252,253].

More recent evidence suggests that that it is the energy density of the diet rather than the fat content that is associated with over-consumption. The mechanisms behind this is thought to relate to the passive over-consumption of food where the amount of food is not down regulated to correspond with the higher energy density of the diet. High-

energy dense diets tend to be high in fat and added sugars and low in fruits and vegetables and low energy density diets tend to be high in water, non-starch polysaccharides (NSP)[253]. This was apparent in a UK based cross sectional study in young children which found that higher energy dense diets had higher fat and lower sugar content than lower energy dense diets [254].

The components of a high-energy dense diet are a characteristic of many of the foods and beverages offered by fast food outlets. The popularity of fast foods has increased and this is reflected by a 300% rise in consumption in children over the last 20 years [255]. The consumption of fast foods does not directly lead to obesity as these are consumed by both overweight and non-overweight children. However, studies have suggested that lean individuals are more likely to adjust their daily energy intake following consumption of a high-energy fast food meal than are overweight counterparts [256,257].

The consumption of sugar-sweetened soft drinks has also shown to increase amongst children. Such beverages now accounts as the greatest source of non-milk extrinsic sugar (NMES) intake [258]. Concerns regarding these drinks relate to the fact that they tend to replace milk and calcium intake in adolescents and they can result in an increased energy intake. However, the direct evidence linking the consumption of soft drinks to an increased risk of obesity remains equivocal as a number of longitudinal studies found a positive association [259] whereas a more recent cross sectional study on young children (aged 2-5 years) found no association with BMI [260].

The consumption of fruit juices (100% fruit juice) has also been associated with weight gain in children as the sugar content of these drinks matches that of many soda-type drinks (sugar-sweetened beverages). The association between fruit juice and obesity was initially observed by a study in pre-school children which found a greater prevalence of overweight in children who consumed 12 or more fluid ounces (355 ml) of 100% fruit juice compared to children who consumed less than this amount (32% vs. 9%) [261]. Although this association has also been observed by other small scale studies, larger scale studies including national surveys have not always been able to replicate this finding [262. Thus, current knowledge on the association between fruit juice consumption and obesity remains equivocal. In

addition to the inconsistency in the evidence, the role of fruit juices in contributing to weight gain becomes even more unclear when considering the amounts of fruit juices consumed by populations. It was shown that the consumption of fruit juice in American children is lower than the maximum amount recommended by the American Academy of Paediatrics (AAP) [262]. Similarly, in UK adults and children it was reported that fruit juices represent a small proportion (less than 10%) of total NMES intake [263].

Consumption of fruits and vegetables has been linked to lowering the risk of heart disease and cancers [264]. Fruits and vegetables are thought to play a role in decreasing the risk of obesity by enhancing satiety because of their high water and NSP content and low energy density [252]. Interventions examining the effects of fruit and vegetables on body weight have varied in their experimental design with some studies focussing on increasing intake of fruits and vegetables only whereas others altering other aspects of the diet. Intervention studies aimed at increasing fruits and vegetables solely have shown that reductions in energy intake were achieved over a short period of time but were not maintained over a longer term. The second group of studies which examined changes following promotion of fruit and vegetables and a decrease in fat intake have shown more successful results as participants either maintained weight or lost weight [252,265]. Thus the unique role of fruit and vegetable consumption in weight control still remains unclear as this has not been differentiated from the effects of changing other aspects of the diet.

The impact of increasing food portion sizes as a possible risk factor for obesity has received much attention. It has been shown that young children aged 3 years have an innate control of appetite but this biological mechanism is lost and overridden by environmental factors by the age of 5 years. A study in adults also showed that larger portion sizes results in increased energy intake as varying the portion sizes of meals did not have an effect on post meal satiety and the energy intake from a subsequent meal [252,266]. This is important when considering that portion sizes of food consumed within the home and outside the home (e.g. restaurants, fast food outlets) and commercial products including soft drinks and confectionary have increased over the last few years.

Dietary patterns and habits such as snacking and skipping meals have been associated with increasing the risk of subsequent obesity. Although difficult to measure accurately, snacking has been associated with greater intake of energy dense foods and overall food consumption [252]. Skipping breakfast has been associated with increasing the risk of obesity. The mechanism of this relationship remains unclear although it is thought that breakfast consumption may be associated with general healthy behaviour or with reduced fat consumption and snacking. Alternatively, skipping breakfast could also be related to greater compensatory energy intake later in the morning and in the evening [267]. Additional aspects of dietary patterns and habits such as a decrease in family meal times have also been observed. Family meal times provide an important setting for instilling social controls on eating and provide an opportunity for observing positive role models. Thus, limiting family meal times can result in children adopting poor dietary habits [229].

1.5.2.3 Obesogenic environments

The obesogenic environment is defined "as the sum of influences that the surroundings, opportunities or conditions of life have on promoting obesity in individuals or populations" [229]. As mentioned earlier, the Foresight report obesity system map highlights all the factors that are characteristic of the obesogenic environment and the interactions between them [230]. For ease of understanding, six obesogenic environments will be discussed in this chapter and these include: foetal, infant, family, school, neighbourhood and macro-environment (industry, media and government).

The foetal environment includes influences such as low and high birth weight, maternal diabetes and maternal smoking which promote an unfavourable foetal environment and have been associated with increasing the risk of childhood obesity [268]. The importance of the infant environment on the risk of subsequent obesity has also been shown by a number of studies. Factors such as rapid post natal growth and the combination of foetal and infant growth have shown to increase the risk of subsequent obesity [269-271]. Secondly, an earlier adiposity rebound (should strictly be known as BMI rebound), which is the second phase of rapid increase in body fat (BMI) that begins around the age of 6 years, has also been associated with increasing the risk although the mechanism by which this occurs remains unclear

[272]. More recently, it was concluded from a review of the literature that both infants at the highest end of the weight or BMI distribution and those with a rapid growth during infancy were at greatest risk of becoming overweight and obese [273]. Furthermore, the protective effects of breastfeeding on childhood obesity have also been demonstrated by a number of studies [274]. Proposed mechanisms underlying the benefits of breastfeeding relate firstly to the presence of bioactive molecules in human milk (e.g. long chain polyunsaturated fatty acids) and lower protein intake in breast fed infants (70% lower than in formula fed infants), which could be involved in programming later adiposity. Secondly, the slower rate of growth seen in breast fed infants protects against the adverse metabolic effects (i.e. components of metabolic syndrome) of faster postnatal growth [275].

Factors within the family environment which influence the risk of subsequent obesity include parent-child interactions, parenting style and diet and activity related behaviours which have shown to "run" in families. A strong and positive association has also been demonstrated between parental BMI and childhood obesity [276], with a stronger association evident in younger children [277] and in the case where both parents were obese [278]. A recent study examining parental BMI and weight change in childhood reported that this relationship is actually gender assortative (same-sex) as a closer association in BMI was found between mothers and daughters and between fathers and sons [279]. Evidence on the greater influence of environmental factors over genetic factors on the association between parental and childhood BMI can be drawn from this study as same sex transmission occurs very rarely.

The remaining obesogenic environments (school, neighbourhood, and macro-environment) are important in determining the risk of subsequent obesity as they represent factors outside of the family environment that determine the dietary and lifestyle choices made by individuals.

1.6 National and local strategies to tackle childhood obesity in the UK

It has been recognised that the epidemic of obesity has a detrimental effect not only on the individuals affected but on society as a whole. For this reason, tackling this issue has now become a public health priority. In the UK, three Government

departments (Department of Health, Department for Education & Skills, Department for Culture, Media & Sport) set a joint Public Service Agreement target in 2004 in response to the rising trends in childhood obesity. This target was to "to halt the year on year rise in obesity among children under 11 by 2010, in the context of a broader strategy to tackle obesity in the population as a whole" [280]. Despite the announcement of this target and introduction of multifaceted interventions to prevent and manage obesity, prevalence levels continued to increase and the health and economic burden was worsening. In response to this, a Government assigned team of experts developed the Foresight report in 2007, which included a review on obesity [230]. Their findings projected that 9 in 10 adults and two thirds of children will be overweight or obese by 2050. They also concluded that tackling obesity would require a societal rather than an individual approach.

In response to this, the Government has set itself a new ambition "of being the first major country to reverse the rising tide of obesity and overweight in the population by ensuring all individuals are able to maintain a healthy weight. Our initial focus will be on children: by 2020 we will have reduced the proportion of overweight and obese children to 2000 levels". A new Cross-Government Strategy was also drawn up in 2008 named "Healthy Weight, Healthy Lives" [27]. This new strategy focuses on five different policy area which include: children: healthy growth and healthy weight, promoting healthier food choices, building physical activity into our lives, creating incentives for better health and personalised advice and support. These policy areas, as advised by the Foresight experts, are aimed at promoting the health of the population and providing the appropriate treatment and support strategies for overweight and obese individuals.

As part of this new strategy, the Government has made a financial investment to introduce a range of new initiatives and strengthen existing initiatives to help meet this new ambition. These initiatives are aimed at preventing and managing obesity in the population and are either rolled out on a national level or within localized areas. Examples of local level initiatives include the Healthy Schools scheme [281] and the more recently introduced social marketing exercise: Change4Life [282]. Local level initiatives are formed on the basis of a Local Area Agreement between Primary Care Trusts and the Local Authority. Examples of local levels initiatives aimed towards the management of childhood obesity include intervention programmes such as the

Traffic Light Programme [283] and MEND (Mind Exercise and Do It!!) [284]. Furthermore, a number of initiatives aimed at supporting local delivery of interventions have also been introduced and these include the National Child Measurement Programme (NCMP) [18], Obesity care pathways and resources such as the Obesity toolkit [285].

Although the progress so far represents significant steps forward in understanding how to deal with this epidemic, reaching this new target of halting and reversing trends remains a challenging task for those involved in the management and prevention of obesity.

Chapter 2: Human Body Composition

2.1 The organization of body components

A major challenge to researchers in human biology, physiology and medicine has been the characterization of the composition of the human body. Some of the earliest research conducted on human body composition was in the 1900s during which chemical analyses of human cadavers were carried out. Such chemical analyses have provided reference data from which a conceptual framework has been developed to organize the human body into levels and models. These levels are organised in a manner of increasing complexity and include: atomic level (level I), molecular (level II), cellular level (level III), tissue organ level (level IV) and whole body level (level V) (figure 2.1) [286]. These levels consist of components which range from the most basic two-component (2-C) model to more complex multi-component models which comprise of three or more components (Table 2.1). The sum of each of the models within the five levels is equivalent to total body mass.

Figure 2.1: Conceptual framework of organizing body composition into levels [Source:287]

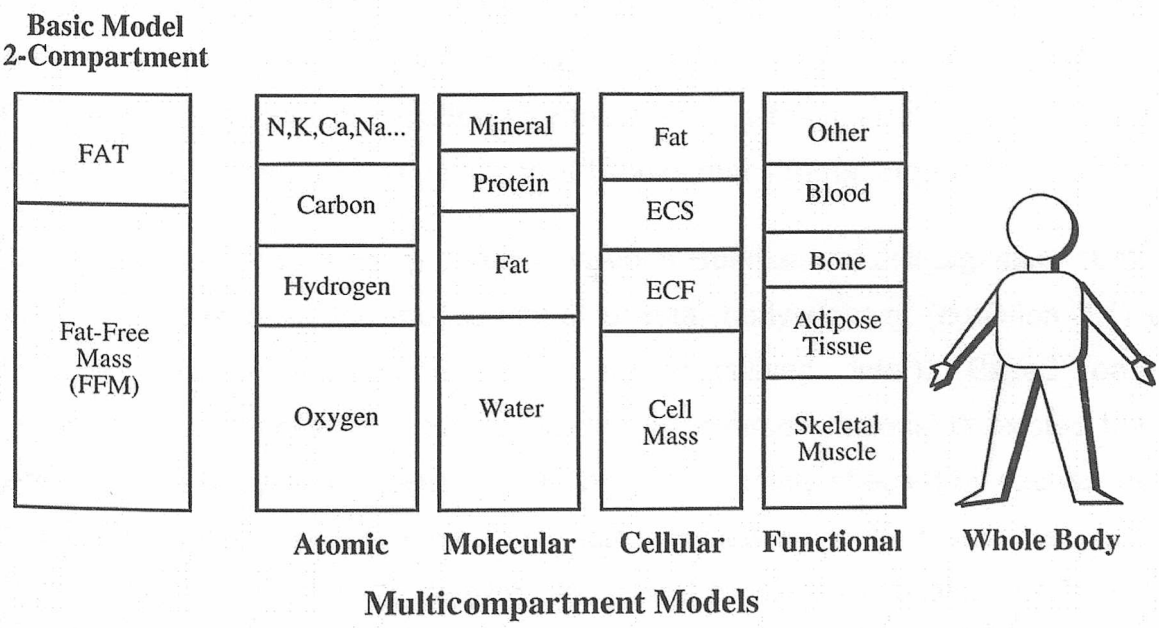


Table 2.1: Conceptual framework of organizing body composition into levels and models [Source:288]

Level	Body composition model	Number of components
Atomic	BM= H + O + N + C + Na + K + Cl + P + Ca + Mg + S	11
Molecular	BM = FM + TBW + TBPro + Mo + Ms + CHO	6
	BM = FM + TBW + TBPro + M	4
	BM = FM + TBW + nonfat solids	3
	BM = FM + Mo + residual	3
	BM = FM + FFM	2
Cellular	BM = cells + ECF + ECS	3
	BM = FM + BCM + ECF + ECS	4
Tissue-Organ	BW= AT + SM + bone + Visceral organs + other tissues	5
Whole body	BW = head + trunk + appendages	3

AT = adipose tissue; BCM=body cell mass BM= Body Mass; CHO= carbohydrates; ECF=extracellular fluid; ECS= extracellular solids; FFM=fat-free mass; FM = Fat Mass; M=minerals; Mo=bone mineral; Ms= soft tissue mineral; SM= skeletal muscle; TBPro=total body protein; TBW= total body water.

2.1.1 The 2-C, 3-C and 4-C models

The study of body composition has commonly relied on the use of indirect methods based on the 2-C model, which have been used to obtain body composition references, validate other indirect (field and laboratory) methods and generate prediction equations. This model is based on the understanding that the body is comprised of two components- fat mass (FM) which consists of body fat; and Fat Free Mass (FFM) which includes all the remaining residual chemicals and tissues such as water, muscle, bone, connective tissues and internal organs [288].

The earliest 2-C model was proposed by Albert Behnke and colleagues in 1942. This model was based on the measurement of total body density (equation 2.1) using hydrodensitometry/under water weighing (discussed later). Based on their investigations, Behnke et al concluded that an inverse relationship existed between adiposity and body density and that the latter was mainly affected by excess fat. This break through on the 2-C model was later applied by other researchers (Keys & Brozek, 1953; Siri, 1956) to develop 2-C model equations that estimate %Body Fat (%BF) using body density [288].

Equation 2.1: Body density (kg/l) = Mass (kg) / Volume (l)

Prediction of %Body Fat (%BF) from body density using the Siri and Brozek equations is based on the assumptions that the densities of FFM and FM are additive and that the densities of these components do not vary between individuals. Additionally, it was also assumed that the proportions of water, protein and mineral within FFM do not vary between and within individuals.

Further work on the 2-C model revealed that inter-individual variability in the density and composition of the FFM does exist especially in relation to age, gender and ethnicity. For example, variations in the composition and density of FFM can be present during growth because of chemical maturation and during disease states because of factors such as over and under hydration [289]. Furthermore, inter-individual variability has also been demonstrated between healthy subjects of similar age and sex because of the heterogeneous nature of this component [290].

In consideration of the fact that differences in the density and composition of FFM do exist between individuals, the 3-C and 4-C models were developed. The 3-C model divides the body into three components namely fat, water and solids (protein and minerals). Prediction of %BF using this model accounts for the inter-individual variability in the hydration of FFM but assumes a constant density for the protein-to-mineral ratio. The 4-C model divides the body into fat, water, mineral and protein and thus estimations of %BF using this model do not rely on assumptions regarding the relative proportions of these components [288].

A review of the various indirect techniques of assessing body composition and fat distribution is presented. This includes a theoretical understanding of these methods and advantages and disadvantages of their use in children and adolescents.

2.2 Body density and volume measurements

2.2.1 Under water weighing (UWW) / hydrodensitometry (HD)

One of the earliest and most commonly used 2-C model is based on the measurement of body density using HD. HD is based on the Archimedes principle which states that an object that is fully submerged in water is subject to a buoyant force which results in a loss of weight underwater that is equal to the weight of water displaced. This loss of weight underwater (corrected for the density of water relative

to the temperature of water at time of submersion) is directly proportional to body volume. However air in the lungs (~1-2 Litres) and gastro- intestinal tract (~100ml) add to the buoyancy and is therefore subtracted from the final body volume reading [288]. A measure of whole body density (calculated as body mass / body volume) is then calculated using the assumption of constant densities of FM and FFM [291].

A theoretical limitation associated with this technique relates to the application of the assumption of constant density of the FFM. Density of the FFM has been reported to change from birth to adulthood as a consequence of decrease in hydration and increase in protein and mineral fractions [292]. The homogenous nature of the FM can be used to assume that its density is unlikely to change. Practical limitations associated with this technique relate to the measurement procedure as this requires immersing into a large tank of water, maximal exhalation to empty the lungs and sitting still under water. Adhering to the procedures and assessing the adherence to these may prove to be difficult in the paediatric population [293].

2.2.2 Air displacement plethysmography (ADP)

The use of this technique to measure body composition has been reported for almost a century although routine use only became possible with the recent development of a viable system. Only one commercial ADP system has been developed to date which is known by the trade name Bod Pod. The principle of this technique matches that of HD except that the subject is not submerged in water but in a closed air-filled chamber. Secondly, body volume is determined by the volume of air a subject displaces when seated inside the chamber as this is equal to body volume.

The Bod Pod system is made up of the Bod Pod plethysmograph which functionally consists of a test chamber and reference chamber, electronic weighing scale, calibration scales, cylinder, computer and software. The operating principle of this system involves oscillation of a diaphragm that is positioned between the two chambers which creates sinusoidal volume perturbations that are equal in magnitude but opposite in sign. This results in pressure changes within the chambers which are monitored and applied to relevant physical gas laws to determine the volume of the test chamber. Body volume corrected for thoracic gas volume and body surface area artifact is then used to calculate body density. With the use of appropriate equations, Siri or Brozek for White subjects and Schutte or Wagner and Heyward for Black

subjects, %BF is predicted [294]. In the case of children, correction for child specific density of FFM can be achieved using Lohman's equation [295].

The use of ADP in assessing the body composition of paediatric populations has become increasingly popular over the last decade. The accuracy of the ADP has been tested against Dual X-ray Absorptiometry (DXA), HD and multi-component models in adults and children. Studies comparing body composition measurements between DXA and ADP and between HD and ADP in children have reported mixed results with some reporting an average negative difference whereas others observed an average positive difference between the methods. More recent comparisons with 3-C and 4-C models have shown that ADP estimated body composition with high accuracy [294,295]. Furthermore, when compared to a 4-C model, ADP was the only technique that did not have the tendency to underestimate %body fat at lower and higher fatness [295]. The precision of body volume measurements has been shown to be similar or slightly better in children than in adults with values of 0.07 and 0.08 litres reported in two studies [296,297].

Although ADP is a more practical and user-friendly alternative to HD, the theoretical limitations inherent to the use of HD are also a matter of concern with ADP. Furthermore, equations to predict thoracic gas volume and surface area artifact built into the BOD POD are adult specific and have shown to overestimate % body fat by 10% when compared against published children specific equations [295]. Hence, inconsistencies between studies in the use of child specific corrections for ADP data can pose difficulties when comparing findings.

2.3 Dual X-ray absorptiometry (DXA)

Dual X-ray Absorptiometry (DXA) scanners were originally designed to assess total body bone mineral (TBBM) and bone mineral density (BMD) by replacing the technology of dual photon absorptiometry (DPA). However, recent years have seen a growing use of DXA in the assessment of body composition as it is able to provide estimates of bone mineral, bone-free FFM and FM on a whole body and regional basis.

DXA technology is based on the principle that the attenuation of X-rays at high and low photon energies can be measured and is dependent of factors such as the thickness, density and composition of body tissues. Thus variation in attenuation between bone, lean tissue and fat can be expected because of the differences in the characteristics of these tissues [288].

Coefficient of variations of 0.62%, 1.89%, 2.0% and 1.1% for BMD, %BF, FM and lean mass respectively have been reported for the precision of whole body composition estimates. Furthermore, the precision of regional body composition measurements have been reported to have been lower than those of whole body assessments [298].

Limitations associated with the use of DXA include the assumption of constant hydration of FFM which may not be appropriate for children and adolescents given that hydration decreases with growth and maturation. Secondly, DXA scanners may pose difficulties in measuring obese children and adolescents whose weight exceeds the limit of 115kg and body dimensions exceed the width and length restrictions [288].

2.4 Total Body Water

The measurement of water is termed hydrometry. This is an important aspect of body composition research because water is the most abundant constituent of the body and it is almost exclusively associated with the FFM. The assessment of total body water (TBW) is typically conducted by measuring the dilution of isotopic tracers. The use of isotopic tracers is based on the principle that the volume of a solvent (body water) can be determined by dividing the amount of compound (isotopic tracer) in a solvent by its concentration [288]. In terms of the human body, if the concentration of a known amount of isotope in the body water pool can be determined, then the size of the water pool can be determined, using equation 2.2

Equation 2.2: $C_1V_1=C_2V_2$ (where C_1V_1 is the concentration and volume of the tracer, C_2 is the concentration of the tracer in the biological fluid and V_2 is the volume of TBW)

An example of an isotopic tracer is deuterium oxide (D_2O), which has been safely administered in humans. D_2O has been used to measure TBW because deuterium (^2H) is approximately two times heavier than hydrogen (^1H) and thus can be detected using a mass spectrometer.

Determining TBW using isotopic tracers is dependent on the application of a number of assumptions. These assumptions require the tracer to be distributed only in body water and demonstrate equal distribution in all anatomical water compartments. Furthermore, it is also assumed that equilibration of the tracer is rapid and that during this period of time, the tracer and body water do not undergo metabolism [298]. Although the validity of these assumptions has been questioned, it has been shown that isotope dilution is an accurate method of measuring TBW and extracellular water (ECW) with both precision and accuracy estimated to be between 1% and 2% [298]. The accuracy with which TBW predicts the FFM (using a 2-C model) is dependent on how close the subjects' FFM hydration is to the assumed hydration constant of 73.2%. Normal biological variability of approximately 2% in hydration of the FFM has corresponded to an error of 3.6% in estimating %BF [299]. Thus the estimation of the FFM using the assumed constant is likely to be erroneous in children as hydration decreases with growth [289] and in obese individuals in whom hydration is significantly increased [300].

2.5 Computerized tomography (CT) and Magnetic Resonance Imaging (MRI)

CT and MRI are laboratory-based techniques that have been used to obtain reference measures of body composition. These techniques are of particular interest because they provide an assessment of body composition on levels beyond that of the classic molecular level. They are both imaging techniques which are regarded to be the most accurate methods of in vivo assessment of body composition at the tissue-organ level. Their limited use in the past has been a result of high cost and limited accessibility however they have recently become increasingly popular in body composition research.

CT is a radiographic method that constructs images by firstly measuring the attenuation of X-ray beams as they pass through the scanned area as tissues with different densities will differ in their attenuation. This information is then applied to mathematical techniques to reconstruct a computer generated image of the scanned area which enables the identification of bone, fat mass and lean tissue. Assessments using CT are commonly limited to regional as opposed to whole body due to the high radiation exposure [288].

The principle underlying the assessment of body composition using MRI is essentially similar to that of CT except that the former does not use ionizing radiation. Instead, images from MRI are generated by enabling the hydrogen nuclei present within biological tissues to emit radio frequency signals. The MRI instrument generates and sends a magnetic field and then a pulsed radio frequency across the body, which causes the nuclei to line up and absorb the energy. This radio wave is then turned off to allow the nuclei to release the absorbed radio signal and an image is constructed using the emitted signals [288].

One of the most predominant uses of CT and MRI in the study of body composition has been to measure the distribution of abdominal adipose tissue. Both CT and MRI are able to differentiate between subcutaneous adipose tissue (SAAT) and visceral adipose tissue (VAT). Furthermore, CT images enable subcutaneous abdominal adipose tissue to be divided into superficial and deep compartments using the fascia superficialis [298]. The ability to obtain this level of information has provided a better understanding of the health risks associated with patterns of fat distribution and specific fat depots.

2.6 Whole body counting of potassium

Total body potassium (TBK) is commonly used as a reference measure of body cell mass (BCM) (the metabolically active lean tissue of the body) as the majority of potassium in the body is stored intracellularly. Natural potassium is made up of three isotopic states (93.1% 39K, 6.9% 41K, and 0.0118% 40K) of which only 40K is radioactive [298]. Quantification of TBK is achieved by measuring the gamma rays emitted by 40K using a whole body counter [288].

2.7 Skinfold thickness measurement

The skinfold (SKF) thickness measurement is an indirect technique of assessing subcutaneous adipose tissue and is non-invasive, inexpensive and practical. A measure of subcutaneous fat is obtained at selected sites of the body by "pinching" a double fold of the skin with callipers. The SKF method is used to predict body density and in turn estimate %BF. However the basis of this prediction is dependent on the application of certain assumptions. Firstly, it is assumed that the SKF sites provide a

good measure of subcutaneous fat. Secondly, it is assumed that the subcutaneous fat depot is proportional to total body fatness [288]. However the validity of this assumption has been questioned as biological variability with regards to age, gender and ethnicity has been demonstrated in the proportion of body fat located internally as opposed to subcutaneously [301].

Other limitations associated with this method relate to the prediction of %BF using equations, which are subject to error especially in prepubertal children. Secondly, the SKF provides a measure of fatness with no consideration for the FFM component of body composition. Thus despite the several advantages, the accuracy with which body fat is predicted is dependent on measurement technique and the validity of the underlying assumptions [288].

2.8 Bioelectrical Impedance Analysis (BIA)

It was first discovered by Galvani in the 18th century that biological systems have electrical properties through his experiments on muscle contractions. Further studies on a range of tissues brought about better understanding of this property and subsequently led to the development of Bioelectrical Impedance Analysis (BIA) in the 1970s [302].

BIA is a simple, rapid, non-invasive and inexpensive method of assessing body composition that requires minimal maintenance and can be used in a range of settings. It operates on the basis of sending a low level electric current through the subject's body and measuring the impedance to this current with a BIA analyzer [303]. Impedance is the "frequency dependent opposition of a conductor to the flow of an alternating electric current" [298]. This opposition is made up of two components- resistance and reactance. Resistance can be defined as the opposition to the flow of the current and thus it represents how well an object can transmit an electric current. Resistance can be calculated using Ohm's Law by dividing voltage (E) by the current (I) [302]. Reactance represents the voltage that is stored by a condenser for a short period of time and involves separation of charges. Resistance in a biological conductor is dependent on the viscosity of physiological fluids as this influences the movement of current transmitting ions whereas reactance is caused by charging of capacitors such as cell membranes and tissue interfaces [302,304].

The aqueous tissues of the body which includes lean tissue (such as muscle and blood) are good conductors of an electric current due to their dissolved electrolytes whereas body fat, being a relatively anhydrous tissue has poor conductance properties [287]. Hence the resistance to the flow of a current would be higher in subjects with greater amounts of body fat [288]. Conversely, a greater volume of TBW will allow a better flow of current through the body. This forms the underlying principle of estimating TBW from impedance measurements and subsequently predicting FFM from TBW because of the large water content (~73%) of FFM.

Indirect estimation of the volume of TBW or FFM is based on a number of principles of electrical conductivity and on a number of assumptions regarding the geometric shape of the body and the relationship between the length and volume of the conductor. The first assumption regarding the body's geometry is that it has the shape of a perfect cylinder with uniform length and a cross sectional area. Secondly, the flow of current through the human body is proportional to the height (length of the conductor, L) and inversely related to the cross sectional area (A) of the body [303]. This relationship can be expressed using the equation 2.3.

Equation 2.3:	$Z = p(L/A)$ where Z = impedance and p= specific resistivity which is assumed to be constant as the body is modelled as an isotropic conductor

When expressing this in terms of the body's volume instead of it cross sectional area, the equation is rearranged to yield the following (equation 2.4):

Equation 2.4:	$BV = pL^2/Z$ where BV = body volume

This equation demonstrates the relationship where the volume of the FFM/TBW is directly related to length squared or height squared and inversely related to impedance. This equation is further re-arranged based on the assumption that the contribution of reactance to the body's impedance is small and so resistance is equivalent to body impedance [288,303]. Hence prediction of the body's conducting volume can be determined using the resistance index (equation 2.5):

Equation 2.5:	$TBW = p \times Height^2/Resistance$

Although the application of this equation makes theoretical sense there are a number of limitations associated with its practical application. Firstly, the human body is not composed of a uniform cylinder but consists of five cylindrical segments (trunk, two arms and two legs). Secondly, the ionic properties of tissues within these segments

varies thus the specific resistivity of tissues is not constant. Thirdly, prediction of FFM from BIA is based on the assumed constant hydration of 73.2% which may not be true in all subjects due to physiological (ethnicity) and pathological (obesity, disease state) factors [288].

Assessment of body composition using bioelectrical impedance analysis has been performed for many years. However, recent advances in the technique have promoted its use in clinical and research settings. Newer model BIA systems now consist of contact electrode systems, thereby eliminating the need for gel electrodes. The Tanita BC-418MA Segmental Body Composition Analyzer (Tanita Corporation, Tokyo, Japan) represents an advanced BIA system that has been designed to collect both whole body and segmental impedance measurements using an 8 contact electrode system.

2.8.1 Tanita BC-418MA Segmental Body Composition Analyzer

The Tanita BC-418MA model is a step-on system that is made up of two stainless steel rectangular footpads, each consisting of two separate footpad electrodes. The footpads are fastened to a metal platform, which is set on force transducers to allow body weight/mass to be measured. The system also comprises two handgrips that have an anterior and posterior portion. Injector electrodes allow a pre-defined signal to be passed through the subject whilst receiver electrodes measure impedance across the subjects' tissue. The BC-418MA system is a single frequency BIA technique that performs measurements at 50kHz and a 0.8mA sine wave constant current [305].

The segmental impedance measurements are obtained using the electrode configuration protocol developed by Organ *et al* [306]. The segmental impedance analysis is based on the measurement of five segments: each arm, each leg and the remainder (trunk + head). Segmental analysis of the legs and arms is based on the impedance measurement of the right foot to hand electrical pathway. Analysis of the whole body is based on the corresponding left foot to hand electrical pathway. Composition of the 'trunk + head' is based on the difference between whole body and sum of leg + arm estimates.

Composition of the whole body and segments is expressed as %BF, FM (kg) and FFM (kg). These body composition values are estimated using equations that incorporate the subjects' gender, height, weight, age and impedance measurement. The prediction equations used in this model have been derived from regression analyses with dual DXA, which is defined as the reference method [305]. Validation of the BC-418 Analyzer has been performed in a mixed population of adults and children against DXA and was found to be more superior to previous BIA models [305]. More recently, a paediatric validation of this model was performed against DXA and ADP (Bod Pod). Findings from this validation study demonstrated that results from the BC-418 highly correlated with DXA (R^2=0.83, SEE=4.46%) and that mean values did not differ significantly. [307]

Gender specific %BF references have recently been developed using this BIA system in Caucasian children aged between 5.0 and 18.5 years (Figure 2.2 & 2.3) [308]. These charts were developed from a dataset of 1116 boys and 869 girls and consist of seven centile curves spaced two-thirds of a z-score (or standard deviation score) apart ranging from the 2nd to the 98th centile. These centile curves were fitted using the LMS (skewness of distribution, median of measurements, coefficient of variation) method, which adjusts the distribution of %BF for differing degrees of skewness occurring at different ages [13]. The 2nd centile was defined as the upper limit of underfat whereas the 85th and 95th centile used to define the overfat and obese boundaries respectively. The latter boundaries (defining overfat and obese) were chosen as they are broadly consistent with the International Obesity Task Force's (IOTF) BMI cut-offs for overweight and obese (described below).

Figure 2.2: UK %BF reference chart for boys [Source: 309]

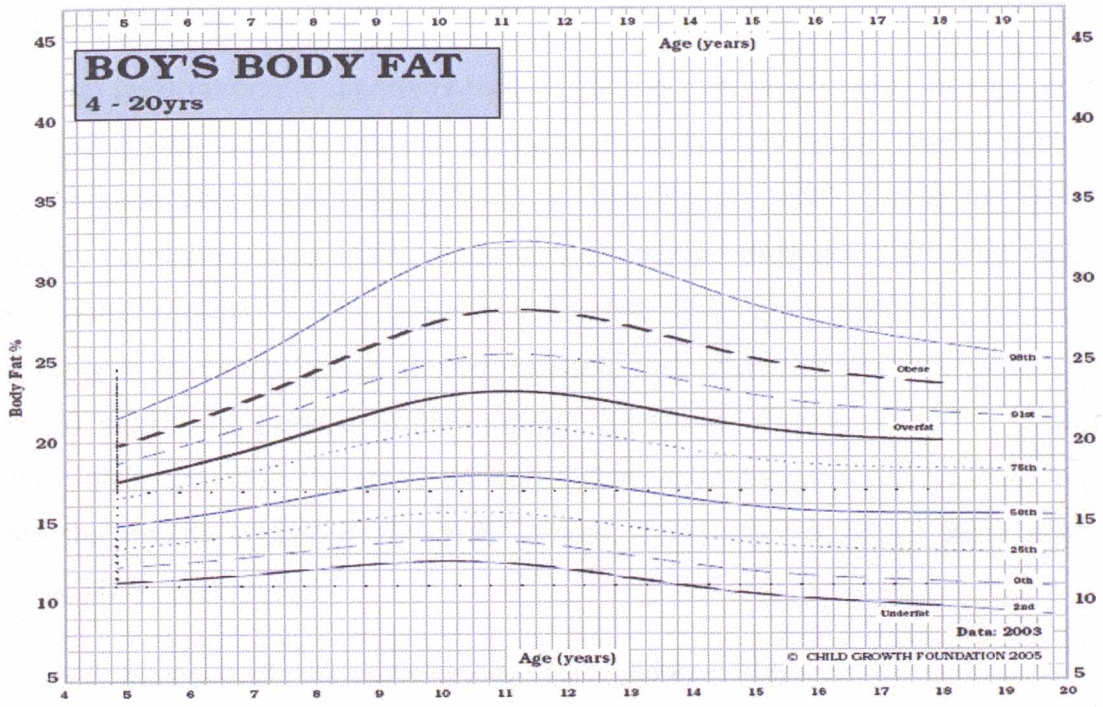
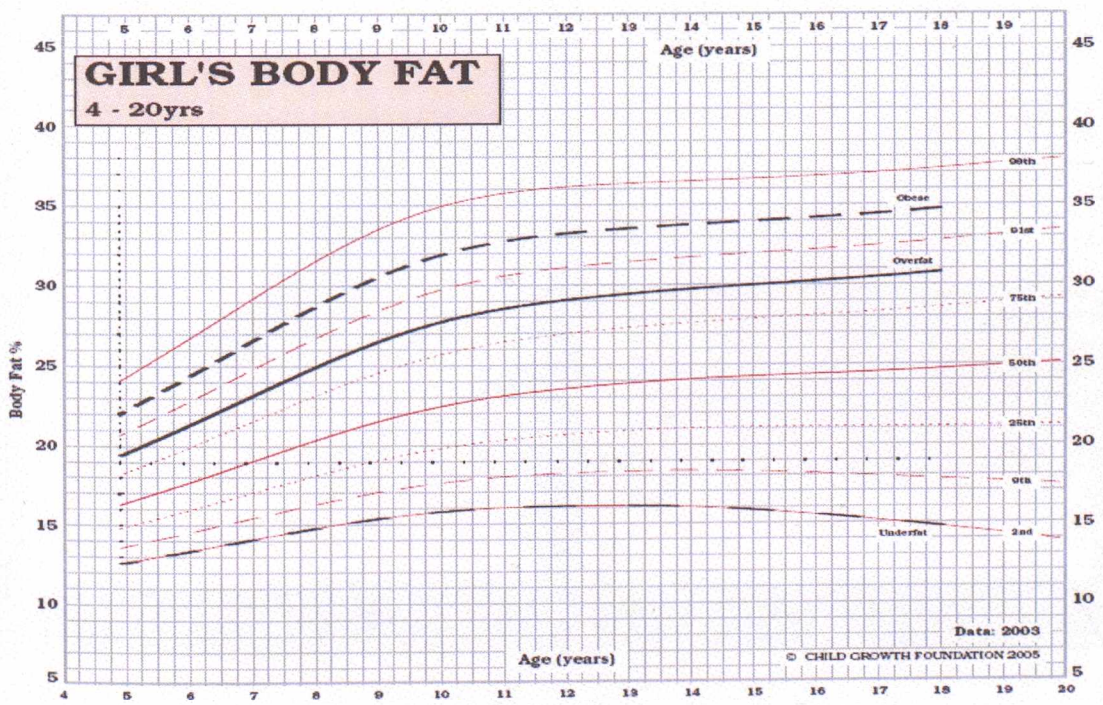


Figure 2.3: UK %BF reference chart for boys [Source: 309]

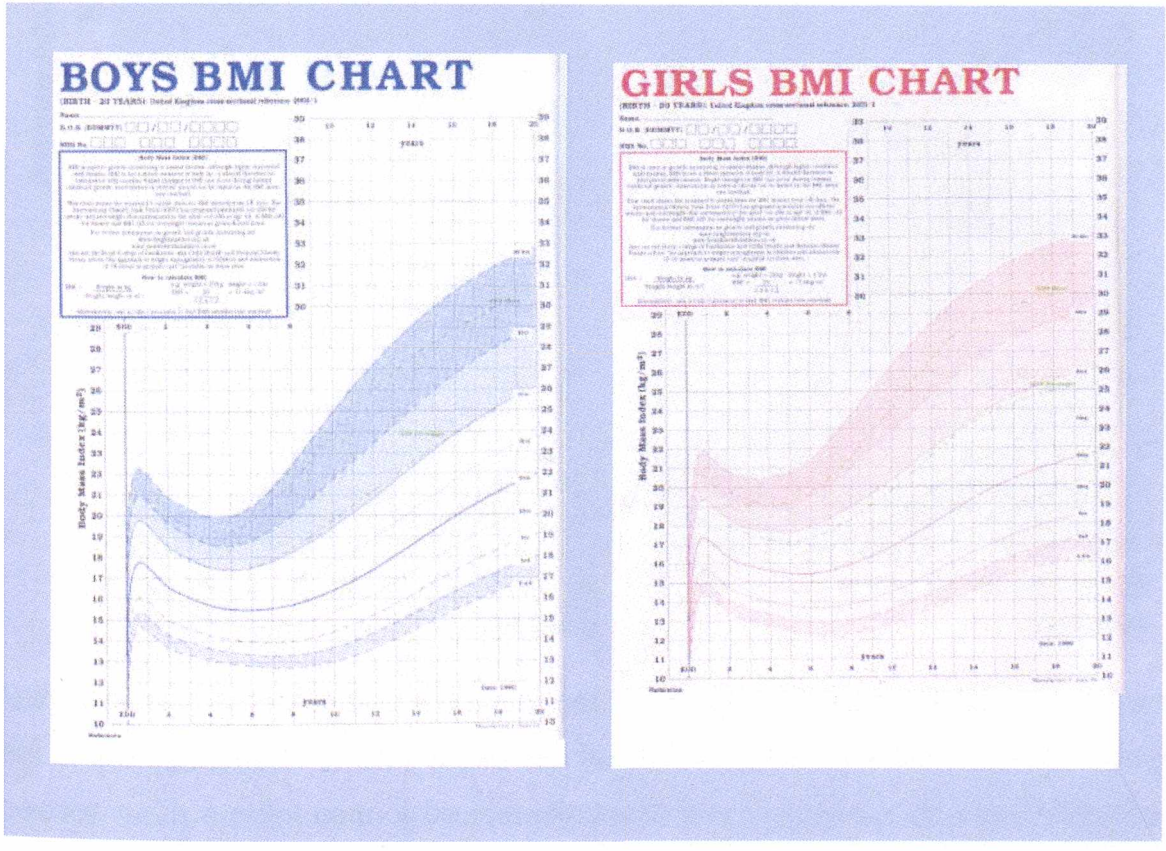


2.9 Anthropometry

2.9.1 Body Mass Index (BMI)

There is an international consensus that childhood overweight and obesity should be defined using BMI, as it is a simple proxy for body fatness when used in conjunction with population reference data [310,311]. The British 1990 charts for BMI were created by combining data from 11 surveys conducted between 1978 and 1990. The dataset consisted of 15 636 boys and 14 899 girls aged between 33 weeks of gestation and 23 years. These gender-specific BMI charts span from birth up to the age of 20 years and consist of nine centile curves [5] (Figure 2.4). The curves are based on divisions of two thirds of a z-score that range from the 0.4^{th} to 99.6^{th} centile. As with the centile curves in the %BF charts, these centile curves were also fitted using the LMS method [5].

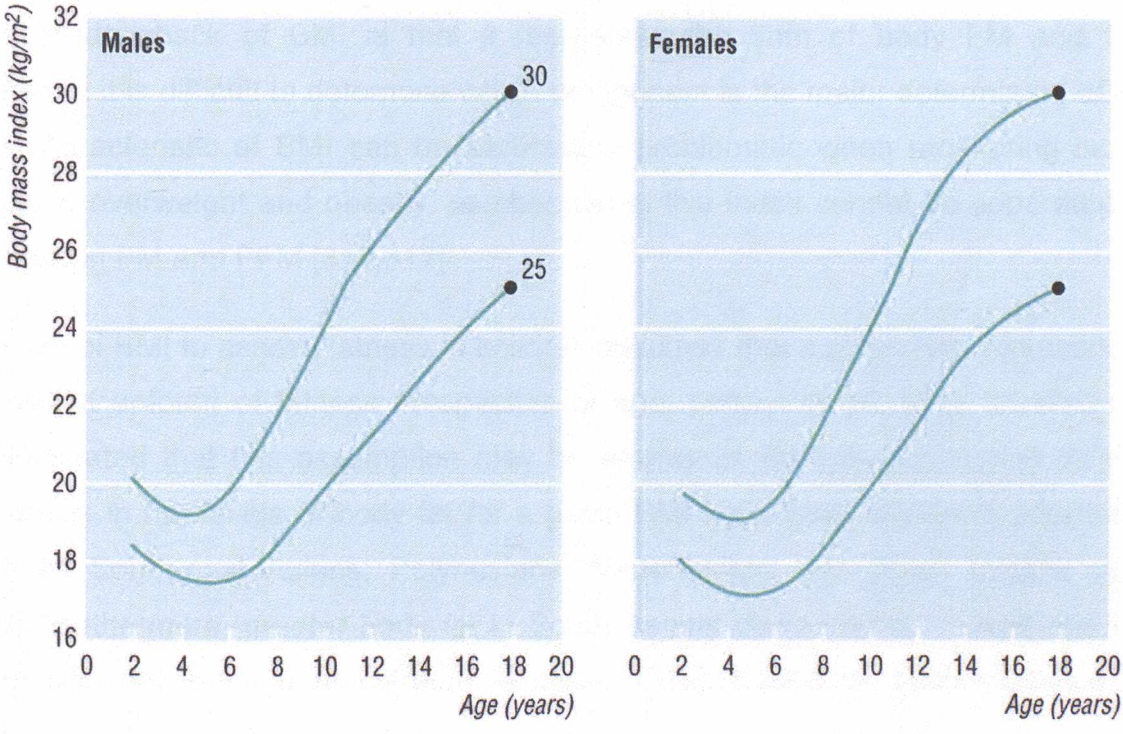
Figure 2.4: Gender-specific UK BMI reference charts [Source: 312]



Similar BMI distribution charts exist for children in various countries. However, the choice of cut-off points varies between countries. In the UK, definitions of overweight and obesity in children correspond to the 91^{st} and 98^{th} percentile respectively. In the

USA, children are identified as being either at risk of overweight (between the 85th and 94th centile for age and sex) or overweight (on or above the 95th centile for age and sex) [310]. Given the variation of cut-offs and definitions between countries, the IOTF developed cut-offs to enable international comparisons of obesity prevalence [313]. These international cut-offs were extrapolated back from the adult BMI cut-offs of 25kg/m^2 and 30kg/m^2 at age 18 years for overweight and obesity respectively (Figure 2.5). The IOTF overweight cut-off lies on or close to the 91st centile for boys but consistently below the 91st centile for girls. The IOTF cut-off for obesity lies above the 98th centile for both boys and girls (see respective BMI charts, Figure 2.4). Thus the IOTF cut-offs classify approximately 8% and less than 2% of the UK 1990 reference population as being overweight and obese respectively.

Figure 2.5: Gender-specific IOTF cut-offs for overweight and obesity [Source: 313]



It is important to note that these cut-offs are based on the theory that they represent a point in the distribution of BMI where the health risk of obesity starts to rise sharply. However, such a point cannot be identified with any accuracy because children and adolescents have less defined obesity-related morbidity than adults. Secondly, long-term health risks of childhood obesity are mediated through adult obesity making it difficult to separate the effect of obesity in childhood from the effect of obesity in adulthood. Thirdly, the IOTF cut-offs assume that growth ceases at around age 18

years and this is not necessarily true. For example, growth in stature is likely to cease earlier than weight gain thus affecting BMI. Also the BMI curves do not give the impression of flattening off at around age 18 years suggesting the trajectory is still rising. It has therefore been suggested that the choice of cut-offs are essentially arbitrary, as they are not associated with health risks per se [314].

One of the drawbacks associated with the use of BMI in children is that it may not capture all those children who may be at risk of obesity-related morbidity. This is partly because of its sensitivity and specificity. BMI has a high specificity (low false positive rate) therefore is able to correctly identify the fattest children in a sample. However, due to its low sensitivity (moderate-high false negative rate), it fails to identify all children with a high body fat content or all those at risk of associated morbidity [315].

Another drawback of BMI is that it represents the sum of body FM and FFM. Therefore it is difficult to determine which component is the major determinant of BMI. This characteristic of BMI can be particularly problematic when monitoring secular trends in overweight and obesity, as changes in this index cannot be separated into changes in FM and FFM [316,317].

The use of BMI to assess fatness in children assumes that a given BMI represents an equivalent amount of fatness irrespective of age, gender or ethnicity. Studies have demonstrated that this assumption may be erroneous for both adults and children. Variations in the levels of body fat for a given BMI have been shown in populations such as South-east Asians, Polynesians, Micronesians and Asian Indians adults [318]. Furthermore, greater body fat in South Asians compared to Caucasians for a given BMI has been demonstrated in adults [319]. Based on such evidence, the World Health Organization (WHO) introduced two lower cut offs >23kg/m^2 and >25kg/m^2 for adult Asian populations to include those "at risk" and those at "increased risk" of obesity related morbidity respectively [320]. Further to this, Indian health organizations recently introduced a lower cut off to define overweight (23kg/m^2) and obesity (25kg/m^2) in the native Indian Asian population [321].

In children and adolescents, it has been demonstrated that the relationship between BMI and body composition is more complex and is dependent on maturation stage,

gender and ethnicity [322]. More specifically, for a given BMI, %BF has shown to be lower in boys than in girls, lower in African Americans than in Caucasian and lower in those at an advanced stage of sexual maturation [4,323]. In contrast, South Asian adolescents have shown to have a higher %BF for a given BMI z-score compared to white European adolescents [190,322,323,324]. This suggests caution should be exercised when using BMI to determine levels of obesity in an ethnically diverse population or compare levels between populations of children.

In addition to ethnic specific variations, the relationship between FM (kg), FFM (kg) and BMI (kg/m^2) has been shown to vary even in children of the same age, sex and ethnicity. The concept of normalizing measures of body composition for height was introduced in 1990 and subsequently the following variables were developed- Fat Mass Index (FMI kg/m^2) (FM / $Height^2$) and Fat Free Mass Index (FFMI, kg/m^2) (FFM / $Height^2$). The sum of FMI (kg/m^2) and FFMI (kg/m^2) equates to BMI (kg/m^2) [325]. Plotting FMI, FFMI, BMI and % BF on a Hattori chart has demonstrated that children of similar ages and BMI can widely differ in the level of % BF [326].

A further drawback of BMI is that it gives no indication of body fat distribution [327]. The increased risk for obesity-related morbidity in children and adolescents with excess accumulation of body fat intra-abdominally has been discussed earlier [121]. This is worrying given the increasing number of children being identified with adverse risk factors and in particular the higher risk in children from minority ethnic groups [178]. The evidence emphasizes that the assessment of obesity on the basis of BMI may not accurately identify those children especially at risk of obesity-related ill health. Despite these limitations, it is used widely in clinical and epidemiological situations and therefore demonstrating a trade off between accuracy and precision and ease of use.

2.9.2 Waist circumference (WC)

The metabolic risk associated with the accumulation of intra-abdominal fat in children has been a more recent finding. In adults, however, the metabolic risks associated with abdominal obesity are well established. As with assessing total body fatness, reference methods used to evaluate body fat distribution such as CT and MRI have limited use in clinical and epidemiological settings. In addition, the radiation risk posed by MRI makes this method unsuitable for children. Consequently, anthropometric measurements such as waist-to-hip ratio (WHR) and waist circumference (WC) have been used to evaluate central adiposity in adults and in children.

In adults, WC has been used to quantify abdominal obesity. It has also been associated with cardiovascular risk factors such as high fasting insulin levels, elevated systolic blood pressure, increased diastolic blood pressure, and elevated ratio of total to HDL plasma lipids and lipoproteins [328,329]. Moreover, changes in WC have shown to reflect changes in cardiovascular risk factors [330]. Based on such evidence, WC is currently used in clinical settings as a clinical measure of cardiovascular disease risk in adults [331].

The use of anthropometric measures such as WHR and WC as measures of upper body fatness in children has been a more recent addition [342]. However, WC and not WHR has been found to be a good predictor of abdominal fatness in children [332]. More current studies on children and adolescents have demonstrated that WC is the strongest correlate of fat distribution and that this measure was least affected by gender, ethnicity, age and overall fatness [333,334].

Although WC is a good predictor of abdominal fatness in children, it is important to note that abdominal adipose mass is made up of two components: intra-abdominal and subcutaneous adipose tissue. Hence, WC relates to both components of abdominal adipose tissue in children and adolescents. Given this, it is essential to determine whether WC rather than intra-abdominal fat relates to adverse risk factors in children.

A number of studies have investigated the association between waist circumference and metabolic risk factors in children and adolescents. The Bogalusa Heart study demonstrated associations between WC and fasting insulin concentrations (indicative of insulin resistance), adverse concentrations of plasma triglycerides, LDL and HDL cholesterol, which were all independent of height, weight, ethnicity, and gender [338]. Another study carried out in obese children aged 12 to 14 years showed a correlation between WC and an atherogenic lipoprotein profile [335]. Such studies have demonstrated that abdominal obesity is related to adverse risk factors and concluded that WC is a useful and convenient indicator of obesity related metabolic alterations.

More recent studies have demonstrated that WC performed better than BMI in identifying children with insulin resistance and hypertriglyceridaemia. They also concluded that WC was the best measure of identifying children with these risk factors and therefore those most at risk for metabolic syndrome [336-338].

In view of the ability of WC to predict abdominal fatness and its association with adverse metabolic risk, the assessment of WC in clinical situations as an adjunct or alternative to BMI had been suggested [327]. As a result, WC percentile charts have been developed for paediatric populations in Italy [339], Spain [340] and Canada [341]. In 2001, WC percentile charts were developed in the UK for Caucasian children and adolescents aged 5 to 16.9 years [342]. An increase in WC with age in both males and females was evident which suggests that it is strongly age related in childhood. Consequently, the UK gender specific charts were developed using the LMS curve fitting software as it enables normalized growth centile references to be developed [13]. In accordance with the UK 1990 BMI charts, the 91st and 98th centile were used to define overweight and obesity respectively.

The development of the WC centile charts has allowed for secular trends in abdominal fatness to be assessed. Secular trends in BMI and WC over the last 10 and 20 years were examined in British children. Findings demonstrated that an increase in central fatness has exceeded general fatness in boys and girls over this period of time, therefore, suggesting that an underestimation of obesity prevalence has occurred using measurements based on height and weight [327].

More recently, the influence of height on WC in adults and children has been given more attention. This is in view of the strong positive correlation between height and WC throughout growth during childhood and into adulthood although there is no quantitative evidence to support the precise influence of height on WC. It has therefore been suggested that incorporating a height measurement may partly correct for its influence on WC [343]. Hence, a simple ratio of WC to height (WHtR) has been proposed as an indicator of excess abdominal fat accumulation and a boundary or cut-off value of WHtR >=0.50 identifying those with excess abdominal fatness [343]. This measure has been suggested as being appropriate for use in adults and children aged above 5 years. A number of studies have also concluded that WHtR is able to predict cardiovascular disease risk factors better than BMI or %BF [344-346]. This finding has also been observed in children from minority ethnic groups [347]. Secular trends in British children exceeding the 0.50 WHtR boundary value have also been examined with results demonstrating a clear rise in the last 10-20 years [348].

One main concern with the use of WC in clinical and epidemiological settings is the lack of consensus on the definition of the WC measurement site. The two most common sites of waist measurement are firstly, at the level of the umbilicus and secondly, midway between the lowest rib margin and the iliac crest [2,349]. A recent study examined the consistency between three different measurement sites (midpoint between lowest rib and iliac crest, crease on lateral flexion and 4cm above umbilicus) and showed a discrepancy of more than 3cm in 54% of the sample and a maximum discrepancy of 8cm in WC readings. It is currently not possible to state which measurement site is superior (if any) as measurements from each site have not been compared with visceral fat readings obtained from a reference standard such as MRI [350].

Furthermore, the simplicity of obtaining a WC measurement can often be misleading as this very nature can result in erroneous results. The error associated with obtaining WC measurements can be in the form of inter-observer error (between observers) and intra-observer error (within observer). Using two measures of imprecision, namely the technical error of measurement (TEM) and coefficient of reliability (R), a number of studies have examined the inter-observer and intra-observer error of taking WC measurements in adults and children. Collectively, these studies reported that mean coefficient of reliability for intra-observer error (0.97) and inter-observer error

(0.94) was almost similar and close to the acceptable level of 0.95 [351]. However, in order to ensure that measurements obtained from individual children and between studies are reliable and accurate, it is imperative that health professionals and researchers become proficient at obtaining WC measurements. This can be achieved through appropriate training, practice and experience through which measurement error can be reduced [351].

Another matter surrounding the use of the UK WC percentile charts is that they have been developed from a sample of Caucasian children in the UK. It is therefore important to question the applicability of these charts in non-Caucasian children given that variations in fat patterning and growth exist between ethnic groups [112]. This is essential given the mounting evidence demonstrating the increased risk of obesity-related morbidity in children from Asian and African-Caribbean backgrounds.

Chapter 3: Methodology

3.1 Subjects

3.1.1 Subject recruitment for chapters 4, 6 and 7

Subjects were recruited by approaching primary and secondary schools within London and Greater London. These locations were chosen because of the ethnic diversity of these areas relative to the rest of England and Wales. As the key objective of this thesis was to obtain measurements in children from minority ethnic groups, it was agreed that London was an appropriate location in which this objective could be met. Support for this came from the 2001 Census which found that 14% of the population in England and Wales was living in London and that 46% of all non-White ethnic groups in England and Wales were residing in London [352,353]. Furthermore, majority of the ethnic groups within the non-White category had numbers above average living in London with some groups based primarily in this area. This was evident from the figures on the percentage of Black Africans (79%), Other Black (63%), Black Caribbeans (61%), Bangladeshis (55%) and Other Asians (55%) living in the capital. An exception to this was the population of Pakistanis of whom only a fifth were residing in London at the time of the census [353].

Also evident from the census was that the spread of non-White groups was not even as some groups were more densely populated in one or two boroughs. An example of this was the Bangladeshi group which was found to make up a third of the population in Tower Hamlets and less than a tenth in Newham. Furthermore, Indians were found to make up 22% of the population of Harrow and 17% of the population in Ealing and Hounslow. The boroughs with the highest proportion of Black Africans were Southwark (16%) and Newham (13%). The greatest proportions of Black Caribbeans were residing in Lambeth and Lewisham (12%). Although Newham and Waltham Forest had the highest proportion of Pakistanis, no London borough had a higher percentage greater than 8% [353].

The choice of London boroughs and schools was made on the basis of the information obtained from the 2001 Census [352,353]. Thus, boroughs that were densely populated with South Asian, African and Caribbean populations including the

London Boroughs of Brent, Tower Hamlets, Hackney, Islington, Newham, Hammersmith, Redbridge and Harrow were selected. Selection of schools within these boroughs was also dependent on whether the schools were willing to participate. Additionally, factors such as accessibility, ethnic make up and age profile of the school were also considered. This information was obtained from the Department for Children, Schools and Families (DCSF) [354].

Schools were contacted initially by letter, providing details on the purpose and process of data collection. A template information sheet and consent form for parents was also enclosed (Appendix A). Head teachers were then contacted by telephone to discuss the project in further detail, answer queries and to obtain a decision on participation.

All the participating schools in Hackney were visited for the purpose of data collection for this thesis and a second project that was in collaboration with The Learning Trust [355]. This is an organisation responsible for running all the educational services within Hackney. The collaborative project was aimed at assessing the prevalence of overweight and obesity in representative schools within Hackney.

Participating schools were then asked to personalise the template consent form by transferring onto school letter headed paper. Next, date(s) on when data was to be collected was organised with relevant members of teaching staff. Consent forms were then distributed to the children to pass onto parents. An opt-out arrangement had been agreed for all the Hackney schools where children were measured unless parents declined for their child to participate. In other boroughs, all children that presented a signed consent form to the school were measured regardless of ethnicity. Schools were requested to provide information on childrens' full names, date of births, gender, up to date age (if available) and ethnicity. Finally, trained field workers performed measurements on the school premises.

Subject recruitment for Chapter 5 will be discussed as part of the methodology section in this chapter.

3.1.2 Sample size

A sample size of 3600 children was required to meet the aims of the project. This figure has been based on the sample size recommended for the development of the references (T. Cole, personal communication). This is broken down to 50 children in each gender, ethnic group (Caucasian, South Asian and African-Caribbean) and in all ages (5-16 years).

3.1.3 Inclusion criteria

All children who presented a signed consent form were measured regardless of ethnicity. A requirement for the measurement procedure was that children were able to stand upright and unsupported for all the measurements. Another requirement was that children could allow their skin to be in contact with the body composition analyser. All children who were able to meet these requirements were measured. Instances where children were not able to participate were when they were wheelchair bound or supported by crutches or had bandages and casts on their feet or hands.

3.1.4 Ethnicity coding

All children were given an ethnicity code using a specific classification system (Appendix B). This was devised by condensing the DCSF classification system used by all schools (Appendix C) [531].

3.2 Measurement procedure and equipment for all chapters

3.2.1 Measurement of height

Height was measured in the standing position using a portable stadiometer (Seca stadiometer, Marsden UK, Mod 220). All subjects were asked to stand straight and ensure arms were hanging loosely at the sides. Next, the examiner checked that the subjects' feet were together, knees were straight, and heels, buttocks and shoulder blades were in contact with the surface of the stadiometer. The subjects were then asked to take a deep breath to enable straightening of the spine. The headboard was then shifted up or down until it touched the crown of the head. A height reading was then recorded to the nearest 0.1 cm [2].

3.2.2 Measurement of waist circumference (WC)

The waist circumference measurement was taken midway between the tenth rib and the iliac crest. A measuring tape was positioned at this point and a measurement was taken at the end of gentle expiration whilst the subject was standing upright [2]. To eliminate inter-examiner error, all waist circumference measurements were performed by a single measurer (DR). Based on a previous calibration study [327], a correction factor of 0.5cm was subtracted from the WC measurement during analysis as all measurements were taken over one layer of clothing.

3.2.3 Weight and body composition assessment using the Tanita BC-418MA Segmental Body Composition Analyzer

To perform body weight and body composition measurements, subjects were asked to firstly remove all heavy clothing, socks and shoes. A correction of 1kg was then entered for light indoor clothing and the subjects' height was entered to the nearest 0.1 centimetre [308]. The measurement procedure required the subject to stand in bare feet on the analyzer ensuring their feet were on the anterior and posterior part of the foot pads. They were asked to hold the handgrips, one in each hand ensuring both anterior and posterior parts were covered. Subjects stood still on the analyser for approximately 30 seconds after which a print out of their measurement was obtained (see Appendix D for BC418-MA print out). The printout consists of the following information

- Age (in years)
- Gender
- Body type as entered onto analyser by measurer (standard or athletic),
- Height to the nearest cm
- Weight (kg)
- BMI (kg/m^2)
- Whole body impedance (Ω)
- Body composition readings (%body fat, fat mass and fat free mass in kg) for the whole body, right and left leg, right and left arm.

Information on regional body composition obtained from the BC-418MA was not used for the purpose of this thesis.

3.3 Quality of measurements

A fundamental aspect of this project has been the collection of the above anthropometric measurements and body composition variables in a large sample of children. These variables have been analysed in all chapters to illustrate the various characteristics of the sample in relation to factors such as age, ethnicity, income group and gender. The quality of these analyses is highly dependent on the accuracy and precision of the anthropometric measurements collected. Consequently, the extent to which measurement error influences the quality of the anthropometric variables was determined prior to data collection for this thesis.

There are two ways in which measurement error can affect the quality of measurements - unreliability and inaccuracy.

3.3.1 Unreliability

This consists of measurement errors as a consequence of imprecision and undependability. Undependability is the variability in repeated measurements of the same subject as a consequence of physiological variation. Imprecision is the variability observed in repeated measurements of the same subject due to intra-observer (one measurer) and inter-observer (two or more measurers) differences. The greater the variability between repeated measurements of the same subject from inter and intra observer error, the greater the imprecision [351].

There are a number of ways in which the variability in repeated measurements from both inter and intra-observer error can be identified. The most commonly used measure of imprecision is the technical error of measurement (TEM) (Equation 3.1). TEM is used to ascertain the amount of the total standard deviation for the sample that is a result of measurement error. Hence, it is a measure of standard deviation also known as the square root of measurement error. However, TEM may not be the most ideal measure of imprecision because it is age dependent. It is also positively associated with the size of the measurement making it difficult to assess the comparative imprecision of different measurements [351]. Nevertheless, TEM can be converted from an absolute variable to a relative variable (%TEM) using equation 3.2 to enable comparisons between different variables or populations.

Equation 3.1: $TEM = \sqrt{(\sum D^2)/2N}$

D = Difference between measurements N = Number of individuals measured

Equation 3.2: %TEM = (TEM/mean) x 100

Based on the limitations associated with the use of TEM in assessing imprecision, the coefficient of reliability (R) (Equation 3.3) has become more widely used. A measure of R demonstrates the proportion of anthropometric variation between repeated measurements that is free of measurement error. Low variability between repeated measures is demonstrated by a value of R that is close to 1 [351].

Equation 3.3: Coefficient of reliability (R) $R = 1 - (\text{Total TEM}^2 / SD^2)$
SD = Standard deviation

3.3.2 Accuracy

This is the extent to which the "true" value of a measurement has been achieved. Inaccuracy is systematic bias and results from errors in the measurement technique or from instrument error. Inaccuracies resulting from equipment bias are thought to be more common with the risk of inaccuracy increasing in more complicated instruments. Hence, inaccuracies arising from a measurement obtained by a simple tape would be less than a measurement involving a sliding scale e.g. a stadiometer. The timing of a measurement can also have an impact on its accuracy. Factors such as observer fatigue and reduced motivation across the day, room lighting and size of measuring area were all taken into account to help reduce the level of measurement error [351].

The above information was noted prior to the collection of the data and necessary steps were undertaken to quantify the level of measurement error in this study. It was imperative to identify the possible causes of inaccuracies and the size of error at the beginning of this project as this would influence the interpretation of the data.

The unreliability and accuracy of the anthropometric measures and body composition variables was determined wherever possible. Measures taken to reduce the size of the measurements error have also been noted.

3.4 Accuracy and reliability of the height (cm) measurement

Approximately 80% of the height measurements were performed by one measurer. Another 10% of the measurements were performed by a second measurer and the remaining 10% of the measurements were performed by five other measurers. It was not practical for one measurer to perform all the height measurements due to time constraints. It was therefore decided that the level of inter-observer error could be reduced by ensuring that majority of the measurements were performed by one measurer. The sources and size of errors have been identified for this measurement.

3.4.1 Accuracy of the height (cm) measurements

Calibration of the stadiometers was achieved using a metre rule prior to the start of data collection. This was also performed at regular intervals to check whether the accuracy had changed as a result of transportation or wear and tear.

Another factor that may have affected the accuracy of the result was the timing of the measurement. There is currently no agreement on the total daily loss of stature in children although the greatest decrease is reported to occur in the trunk and soon after rising [356]. It was presumed that the decrement in height would affect the accuracy of the height measurements collected for this study and so the timing of all measurement sessions was recorded. It was however very difficult to standardize the time of performing measurements as large numbers of children were being measured and because measurements were taken at the schools convenience. However, the timing was standardized to an extent as subjects from all but one of the schools were measured between the hours of 9.00am and 3.30pm. Measuring in only one of the schools commenced at 8.45am. It was also reported that the period between rising and 9.00am would demonstrate the greatest loss of height [356]. Hence, most of the children were measured when the rate of decrement in stature would have decreased. Nevertheless, it must be emphasised that the level of inaccuracy as a consequence of timing variability has not been accounted for in this thesis.

All measurers were provided with the protocol of measuring height (described earlier). The principal height measurer of this project is a trained anthropometrist who has had a number of years of practical experience in this field. The other measurers were all

MSc (Human Nutrition) students who had been given training on anthropometric measurements prior to recruitment on this study. They had also been given training by the principal height measurer prior to visiting the schools. During the training, the measurers were given the opportunity to watch height being measured first and later practice on subjects.

3.4.2 Reliability of the height (cm) measurements

Inter and intra-variability in height measurements was obtained for two of the measurers (DR, DMc). The first was the principal height measurer (Measurer 1) and the second was the measurer who collected 10% of the height measurements (Measurer 2). The technique for assessing this was adapted from the Wessex Growth study [357] whereby duplicate measurements from seventeen subjects from various ethnic groups were obtained using the blind successive method. This involved the measurer measuring each subject 'blind', but two times in quick succession.

The level of inter-observer and intra-observer variability in height measurements was determined using TEM, %TEM, R and coefficient of variation (CV) for each of the subjects. Below is the average level of precision for the height measurement using the four measures described above.

Table 3.1: Intra- and Inter-observer error of height measurements assessed using TEM, %TEM, R and CV

	TEM (m)	%TEM	R	CV (%)
Intra-observer (Measurer 1)	0.0028	0.17	0.999	0.15%-0.20%
Intra-observer (Measurer 2)	0.0022	0.14	0.999	0.06%-0.19%
Inter-observer (Measurer 1 and 2)	0.0021	0.14	0.999	0.13% -0.23%

m= metres

The above results show the level of precision for the height measurements obtained for this thesis. The R for both inter-and intra-observer error was close to 1 indicating that variability between height measurements collected by the same measurer and between measurers was low. Furthermore, the TEM (as a function of inter-and intra-observer error) of height measurements obtained for this thesis was lower that the recommended upper limit for children aged between 5 and 10.9 years (males=0.0058m; females=0.0062m) and 11 and 17.9 years (males=0.0076m; females=0.0067m) [351]. Additionally, the TEM and R in Table 3.1 were lower than

the mean intra-observer (TEM=0.0038m, R=0.98) and inter-observer error (TEM=0.0038m, R=0.99) for height measurements collected in a number of studies in both adults and children [351]. Additionally, CV for intra- and inter-observer errors were considerably lower than the acceptable upper limit of 5%.

3.5. Accuracy and reliability of the weight (kg) measurement

3.5.1 Accuracy of the weight (kg) measurement

The weight measurement was an integrated component of the Tanita BC-418MA Body Composition Analyzer. It was not possible to assess the accuracy of the weight measurement as it was not possible to calibrate the BC-418MA analyzer. However, it was possible to ensure that the measurement technique did not affect the accuracy of the measurements. Steps were taken by all measurers to ensure that subjects were only measured in light indoor clothing and all pockets had been emptied.

3.5.2 Reliability of the weight (kg) measurement

The reliability of weight measurements was assessed by weighing two subjects ten consecutive times on the BC-418MA analyzer. No variation was observed between the measurements for both subjects demonstrating the reliability of the weight measurements obtained from the Tanita BC-418MA analyzer.

In order to minimise the level of inter-and intra-observer error, measurers were asked to follow a set protocol. This protocol required measurers to check that each subject was wearing only light indoor clothing and that all pockets had been emptied. Prior to each of the measuring sessions, the BC-418 analyzers were checked to ensure that 1kg had been entered for the correction of clothing.

3.6 Accuracy and reliability of the WC (cm) measurement

3.6.1 Accuracy of the WC (cm) measurement

It was decided prior to the start of this study that all WC measurements would be performed by one measurer (DR) based on their previous training and experience in measuring WC on approximately 2000 children. Tape measures were routinely calibrated against meter rules to ensure accurate measurements were collected throughout the data collection period.

3.6.2 Reliability of the WC (cm) measurement

The only possible source of imprecision with this measurement was from intra-observer error. The reliability of WC measurements were assessed in obese and non-obese subjects as both groups formed part of the dataset of this thesis. Secondly, it is widely agreed that obtaining consistent WC measurements is more difficult in obese than in lean subjects [288]. Reliability was assessed by taking duplicate measurements on each subject and determining the TEM, %TEM, R and CV for WC measurements taken on obese (n=8) and non-obese (n=5) subjects from various ethnic groups. Results obtained for both obese and non-obese subjects are presented in Table 3.2.

Table 3.2: Intra-observer error of WC measurements in obese and non-obese subjects

	TEM (m)	%TEM	R	CV (%)
Intra-observer (Obese subjects)	0.0167	1.8	0.993	0.5% - 2.4%
Intra-observer (Non-obese subjects)	0.0051	0.7	0.997	0.4%-1.4%

m= metres

It is evident from Table 3.2 that the variability between WC measurements was greater between obese subjects than between non-obese subjects. This is not surprising given the greater difficulty in measuring circumferences in obese subjects [288].The R for intra-observer error for both obese and non-obese subjects was close to 1 indicating that variability between WC measurements collected by the measurer was low regardless of the subjects' weight status.

3.7 Accuracy and reliability of Fat Mass (kg)

3.7.1 Accuracy of Fat Mass (kg)

It is imperative that the measure of Fat Mass (kg) obtained from the BC-418MA analyzer is valid as it is predicted from an equation based on bio-impedance, weight, gender, height and age. Paediatric validation of the BC-418MA analyzer has been performed against DXA and ADP with results demonstrating high correlation with DXA and an insignificant statistical difference between mean values (as discussed in Chapter 2) [307]

Factors such as food and fluid intake, exercise and hydration status have shown to affect the accuracy of the Fat Mass (FM) reading by influencing the impedance

measurement. A recent review on these factors showed that although one study observed a decrease in impedance between 2 and 4 hours after a meal, another study observed an increase in impedance one hour after a meal [358]. Contrary to these findings, one other study found that consumption of food prior to a BIA measurement had no effect on the impedance reading. Moderate to intense exercise has shown to decrease impedance by increasing cutaneous and muscle blood flow. Furthermore, a change in hydration status such as during dehydration or the menstrual cycle can also affect the accuracy of the impedance measurement. Despite the evidence on the range of factors that can affect the accuracy of a BIA measurement, there is still no consensus on the standardized conditions in which this measurement should be obtained [358]. Nevertheless it has been recommended that BIA measurements should be taken following an overnight fast.

It was not considered a practical or viable option to implement this recommendation for the data collection process of this thesis as the measurements were conducted throughout the school working day which commenced at 8.45am and ended at 3.15pm. It was also believed that introducing this recommendation would discourage schools, parents and children from participating in the data collection process. It is likely that the absence of a standardized protocol in which the BIA measurement was conducted may have lowered the accuracy of the Fat Mass reading. However, the level of inaccuracy as a result of this has not been accounted for in this thesis.

The accuracy of this measurement also depends on correctly entering the subjects' gender, age, height and weight on the BC-418MA analyzer. Measurers were trained on how to operate the analysers to ensure correct information was entered. Errors in entering incorrect details such as age, height, and gender on the Tanita BC-418MA analyzer were identified during data handling (discussed in section 3.8)

3.7.2 Reliability of Fat Mass (kg)

The within day variation of % Body Fat (%BF) was assessed during the development of UK % Body Fat reference curves for Caucasian children. This study demonstrated that the within day coefficient of variation for % BF was 1.3% [308].

3.8 Data handing and manipulation

3.8.1 Data inputting and editing

The data collected from each school has been uploaded onto Excel spreadsheets (Microsoft ® Office Excel 2003) by one person (DR). A total of 4564 school children have been measured to date, which equates to approximately 114,100 data points. In view of the large dataset and numbers of data points, it is inevitable that transcriptional errors are likely to occur. The whole dataset was checked manually against the BC-418 print outs and data sheets and outliers were identified using the sort facility on Microsoft Excel. Both these methods identified errors in data inputting and during data collection where incorrect details (age and gender) were entered into the BC-418. Errors were then either corrected or the subjects' measurements were deleted.

Each spreadsheet consisted of variables that were either directly entered or calculated using a formula. Variables that were directly entered onto the spreadsheet were: child code, school code, gender, age, date of birth, date of measurement, height to the nearest 0.1cm, weight to the nearest 0.1kg, BMI (kg/m^2) (from Tanita BC-418MA), WC (cm), % BF, Fat Mass (kg), Fat Free Mass (kg) and Whole body Impedance (Ω). Variables that were calculated using a formula are listed in Table 3.3.

Table 3.3: Variables calculated in Microsoft Excel using a formula

Variable	Equation
Decimal age	(Date of measurement – Date of birth) / 365.25
Height (m)	Height (cm) / 100
WC after correction for clothing (cm)	WC (cm) – 0.5
BMI (kg/m^2)	Weight (kg) / Height^2 (m)
Waist to Height ratio (WHtR)	Waist (cm) / Height (cm)
Fat Mass Index (FMI) (kg)	Fat Mass (kg) / Height^2 (m)
Fat Free Mass Index (FFMI) (kg)	Fat Free Mass (kg) / Height^2 (m)
Ht^2/Impedance (cm^2/Ω)	Height^2 (cm) / Impedance (Ω)

m= metres

3.8.2 Z-scores, centiles and LMS Growth

Measurements of height, weight, BMI, WC and % Body Fat were expressed relative to reference data in the form of z-scores/standard deviation scores (SDS) and centiles. The z-scores and centiles for height, weight and BMI for this study were calculated using the British 1990 Growth data as the reference population [5]. Calculation of z-scores and centiles for individual subjects was achieved using the LMS Growth package (Harlow Printing Limited, UK), which is an Excel add-in software using the British 1990 Growth data [5], UK WC data [342] and UK %BF data [308] as references.

Chapter 4: Comparison of children's body fatness between two contrasting income groups: contribution of height difference

4.1 Introduction

The obesity epidemic has extended across all sections within the population, including all age, ethnic and income groups. In the UK childhood population, most recent figures for overweight/obesity rates are at around 30%, although this figure varies depending on geographical region, age and ethnicity [17]. Obesity prevalence both within and outside the UK is known to be higher in children from lower income backgrounds compared with those from more affluent families [23,359]. These prevalence figures in children are based upon the body mass index (BMI) as the measure of overweight and obesity – a valued and simple assessment tool [310,360]. However it is accepted that BMI only represents a crude proxy for body fatness and hence has a number of drawbacks [315]. Specifically, it does not distinguish between fat mass and fat-free mass; it gives no indication of body fat distribution and can misclassify some children, leading to a potential underestimation of true obesity prevalence and the number of children at risk of obesity-related morbidity [316,361].

One often overlooked contributory factor to the higher observed prevalence of overweight and obesity in children from lower income backgrounds is their shorter height for age compared with children from higher income groups. This means that at similar body weights across income groups, BMI would be greater in children from lower income groups [362,363]. Furthermore, if body weight for age were to be higher in lower income children, this would compound the effect of shorter stature on BMI even further.

A relatively recent addition to the range of obesity assessment tools in children is the body fat reference chart with centile cut-offs to define underfat, normal fat, overfat and obese [308]. These charts have been developed using bioelectrical impedance analysis (BIA). This technology is improved over earlier versions and has been incorporated into step-on scales [364]. As it is excess body fat rather than excess body weight which is associated with obesity-related pathology, (including dyslipidemia, raised blood pressure and insulin resistance), a specific measure of

body fatness such as that provided by BIA should perform better as a monitoring tool and therefore avoid or minimise misclassification of children. In view of the earlier reported prevalence differences in overweight and obesity (based on BMI) between children across different income groups, this study extended these previous observations by comparing BMI, whole body fatness and upper body fatness in two contrasting populations of children using UK body fat and WC references. Secondly this study allowed for further exploration of the contribution of height to these reported differences.

4.2 Aims and objectives

4.2.1 Aim

Compare measures of regional and whole body fatness between children of differing income groups.

4.2.2 Objectives

Compare BMI, %BF and WC in two contrasting populations of children using UK body fat and WC references. Examine differences in height in relation to body fatness between the two populations.

4.3 Subjects and Methods

4.3.1 Subjects

The subjects for this analysis were extracted from two datasets previously collected by this group. The first dataset comprised 1115 children aged 5-14 years who formed part of a survey of body fatness conducted in east London. The second dataset comprised 1183 children aged 5-14 years, residing in Hertfordshire, Cambridgeshire and West London, most of whom formed part of the study to develop the UK body fat centile charts [308]. Parents/carers were contacted by letter (Appendix A) explaining the purpose of the studies and to request permission for their child to take part. No child was measured where consent was refused. Data on date of birth, gender and ethnicity (using classification in Appendix B) were collected at the same time as the anthropometric measurements were performed. Children were individually coded and the data anonymized. This analysis was restricted to Caucasian children.

4.3.2 Anthropometric and body fat measurements

Data collection procedures were identical in both surveys and all measurements were conducted on school premises. The same trained measurers were mainly responsible for data collection in both surveys. Height was measured to the nearest 0.1 cm (Seca stadiometer, Marsden UK, Mod 220) with children standing in bare feet. Body mass (measured to the nearest 0.1 kg with correction for light indoor clothing) and whole body percentage body fat were measured using the Tanita BC-418MA Segmental Body Composition Analyzer (Tanita Corporation, Tokyo, Japan). The bioelectrical impedance measurement procedure involved the subject standing in bare feet on the analyzer and holding a pair of handgrips. Details of the validation and performance characteristics of this BIA model have previously been reported [308]. BMI was calculated as weight (kg)/height (m^2). Waist circumference (WC) was measured approximately midway between the 10^{th} rib and iliac crest using a non-stretchable tape over a single layer of clothing, with correction as previously described [327].

4.3.3 Ethical approval

This study was approved by the London Metropolitan University Ethics Committee (Appendix F).

4.3.4 Statistical analysis

Z-scores for height, weight, BMI, WC and %BF were calculated using the 1990 UK growth reference data [5,365], the UK WC reference data [342] and the body fat reference data [308]. Waist-to-height ratio (WHtR) was calculated and the proportion of children in each income group exceeding the WHtR boundary value of 0.50 was determined [348]. Mean z-scores and WHtR were compared between the two income groups and between boys and girls using a t-test for unpaired data. Comparisons between the two population groups were extended to examine height, weight and %BF within categories of BMI (non-overweight and overweight/obese) and %BF (normal fat and overfat/obese). Comparisons between the two population groups were extended to examine height, weight and %BF within categories of BMI (non-overweight and overweight/obese) and %BF (normal fat and overfat/obese). The prevalence of children with excess weight/body fat was quantified using two assessment tools - the International Obesity Task Force (IOTF) BMI cut-offs to define overweight and obese [313] and %BF cut-offs to define overfat and obese [308]. Chi-

square tests were performed to examine differences in the proportion of boys and girls and the proportion of each income group who were overweight/obese (BMI, IOTF) and overfat/obese (%BF).

The general geographical locations and demography of the two populations in this study contrast in their levels of affluence – the first being a poorer urban/inner city London area with a high density of social housing, and the second being more wealthier rural/provincial area bordering the outskirts of London. For the purpose of this analysis, the two populations' income characteristics were defined at the school level, from the percentage of children eligible for free school meals (FME). This information, which is routinely collected by Local Education Authorities, is based upon family level of income and is accepted as one standard measure of deprivation both within and between school populations. For schools in the first group (termed 'lower income' for the purpose of this study), a mean of 39% (range 17-57%) of children were eligible for free school meals, whilst in the second group (termed 'higher income'), the proportion of children was 7% (range 0-16%) with a national average being 21% [354,366].

4.4 Results

Characteristics of the study population are shown in Table 4.1 for the whole group and for boys and girls separately. Statistically significant differences between boys and girls were observed for absolute mean height, WC, %BF and age- and sex-adjusted height and weight. Using the IOTF cut-offs for BMI, 13.5% and 5% of the total study population was overweight and obese respectively. Combined prevalence of overweight and obesity (BMI) was significantly higher in girls compared with boys (21% vs. 17%, $p<0.02$), whereas combined prevalence of overfat and obesity (%BF) was similar in girls and boys (24.4 vs. 23.3%, p=NS).

Table 4.2 shows the comparison of anthropometric variables between income groups. Firstly, children from the lower income group were on average slightly younger (by 0.15 years) but this did not reach statistical significance. Statistically significant between-income group differences were observed for absolute mean height, WC, BMI and %BF as well as age- and sex-adjusted weight, height, WC, BMI and %BF. Of these variables, only absolute WC and sex-and-age adjusted WC were

significantly greater in the higher income groups, the remaining variables were significantly greater in the lower income group. Thus, children from the lower income group were on average shorter, heavier and fatter for their age than those from the higher income group. This resulted in the lower income group having significantly higher combined levels of overweight and obesity compared with those from the higher income group (23.2% and 14.6% respectively, $p<0.001$). This income group difference was magnified when prevalence was based on %BF. Children from the lower income group had a significantly higher prevalence of overfat and obesity (31.2%) compared with children from the higher income group (16.4%, $p<0.001$).

The income group influence on z-weight, z-%BF and z-height was also observed both within and across categories of BMI and %BF. No difference in z-weight was observed between income groups in the non-overweight category, whereas in the overweight/obese category (based on BMI) children from the lower income group were significantly heavier for their age and sex ($p<0.05$, Table 4.3). Z-%BF however was significantly greater in the lower income children within both the non-overweight and overweight/obese BMI categories ($p<0.001$). When children were categorized by body fatness (%BF), no difference in z-weight was observed between the income groups either within the normal fat category, or within the overfat/obese category ($p>0.05$, Table 4.4). However, z-%BF was significantly greater in the lower income children within the normal fat and overfat/obese categories ($p<0.001$). Hence, %BF was always higher in the lower income group regardless of BMI or body fat status.

Children from the lower income group were significantly shorter than those from the higher income group (Table 4.2), whether expressed as absolute height (by a mean of 1.3cm $p<0.05$) or as z-height ($p<0.05$). However, this significant difference in z-height was not observed between the income groups when boys (0.28 vs. 0.35) and girls (0.14 vs. 0.22) were analyzed separately. In addition, children who were overweight/obese (BMI) for their age were significantly taller than those who were not overweight for their age (z-height 0.74 vs.0.14, $p<0.001$). Similarly children who were overfat/obese (% BF) for their age were also significantly taller than children who were of a normal fat for their age (z-%BF 0.60 vs. 0.15, $p<0.001$). Thus, irrespective of the assessment tool used, overweight/overfat and obese children were significantly taller for their age than non-overweight/normal fat children. However, within the non-overweight, normal fat and overfat categories, children from the lower income group

were consistently shorter than those from the higher income groups ($p<0.005$ Figures 4.1 & 4.2). This income group difference was not observed in the overweight/obese group (based on BMI, $p>0.05$ Figure 4.1). However, when absolute mean height was compared between income groups in the overweight/obese group, then those from the lower income group were significantly shorter (by a mean of 4.2cm, $p<0.005$).

On examination of upper body fatness, absolute WC was significantly greater in children from the higher income group than in the lower group on a whole group basis (62.5cm vs. 60.6cm, $p<0.001$) as well as in girls (62.7cm vs. 59.7cm, $p<0.001$). Although a similar pattern was observed in boys, the difference between the income groups was not statistically significant (62.4 vs. 61.5cm, $p>0.05$). Furthermore, absolute WC was also greater in the higher income group than in the lower income group in the non-overweight (60.5cm vs. 57.4cm, $p<0.001$), overweight/obese (73.0cm vs. 71.1, $p<0.05$), normal fat (60.6cm vs. 57.6cm, $p<0.001$) and overfat/obese (71.3cm vs. 67.3cm, $p<0.001$) groups.

Children from the higher income group had a higher mean z-WC compared to the lower income children at the whole group level (0.90 vs. 0.71, $p<0.001$) as well as within the non-overweight (0.65 vs. 0.24, $p<0.001$), normal fat (0.63 vs. 0.18, $p<0.001$) and overfat/obese groups (2.12 vs. 1.85, $p<0.001$). However, no statistical difference in z-WC was observed between the income groups in the overweight/obese children (2.21 vs. 2.26, $p>0.05$).

On further examination of upper body fatness, within the overweight/obese group, mean WHtR was slightly greater than in the lower income group (0.523 vs. 0.513, $p=0.069$) whereas within the non-overweight group, mean WHtR was slightly greater in the higher income group (0.438 vs.0.445, $p<0.001$). The proportion of children with a WHtR that exceeded the 0.5 boundary value was 19% in the lower income group and 17.7% in the higher income group.

In summary, the lower income group had a higher prevalence of overweight (BMI), overfat (%BF) and obesity (BMI and %BF). They are generally shorter than children from the higher income group and also had higher body fat levels but this was not necessarily reflected by a difference in body weight between the income groups. Upper body fatness was greater in the higher income group based upon z-WC, but when height was taken into account, mean WHtR was identical between groups.

Table 4.1: Summary of subject characteristics (mean ± SD) and comparison between boys and girls

Variables	Whole group (n=2298)	Boys (n=1251)	Girls (n=1047)
Age (years)	8.86 ± 2.4	8.90 ± 2.4	8.82 ± 2.5
Height (cm)	133.4 ± 15.0	134.1 ± 14.7	132.6 ± 15.4*
Weight (kg)	31.7 ± 11.7	31.9 ± 11.5	31.6 ± 11.8
BMI (kg/m^2)	17.2 ± 3.2	17.2 ± 3.2	17.3 ± 3.2
WC (cm) (n=2035)	62.0 ± 8.5	60.9 ± 8.4	61.5 ± 8.5**
WHtR (n=2035)	0.458 ± 0.05	0.455 ± 0.05	0.457 ± 0.05
% BF	20.5 ± 5.6	19.0 ± 5.5	22.4 ± 5.3***
z-height	0.26 ± 1.0	0.32 ± 1.0	0.18 ± 1.0**
z-weight	0.27 ± 1.2	0.33 ± 1.2	0.19 ± 1.1**
z-WC	0.79 ± 1.1	0.80 ± 1.1	0.80 ± 1.1
z-BMI	0.16 ± 1.2	0.20 ± 1.3	0.12 ± 1.2
z-%BF	0.27 ± 1.1	0.30 ± 1.1	0.22 ± 1.1

*p<0.05; **p<0.01; ***p <0.001

Table 4.2: Comparison of mean anthropometric variables between income groups

	Whole group		Boys		Girl	
Variable	Lower income (n= 1115)	Higher income (n= 1183)	Lower income (n= 553)	Higher income (n= 698)	Lower income (n= 562)	Higher income (n= 485)
Age (y)	8.78	8.93	8.79	8.98	8.77	8.87
Height (cm)	132.7	134.0*	133.3	134.7	132.1	133.1
z-height	0.21	0.30*	0.28	0.35	0.14	0.22
Weight (kg)	32.1	31.4	32.6	31.3*	31.6	31.6
z-weight	0.34	0.19**	0.48	0.21***	0.21	0.17
WC (cm)	60.6 (n=1114)	62.5*** (n=921)	61.5 (n=553)	62.4 (n=566)	59.7 (n=561)	62.7*** (n=355)
z-WC	0.71 (n=1114)	0.90*** (n=921)	0.76 (n=553)	0.83 (n=566)	0.66 (n=561)	1.02*** (n=355)
WHtR	0.457 (n=1114)	0.456 (n=921)	0.462 (n=553)	0.454** (n=566)	0.453 (n=561)	0.459 (n=355)
BMI (kg/m^2)	17.6	16.9***	17.8	16.7***	17.5	17.2
z-BMI	0.30	0.04***	0.45	0.01***	0.16	0.08
%BF	21.8	19.3***	20.5	17.8***	23.2	21.6***
z-%BF	0.52	0.03***	0.65	0.03***	0.40	0.03***

*p<0.05; **p<0.01; ***p <0.001

Table 4.3: Mean age-and sex-adjusted weight and %BF of non-overweight (BMI, IOTF) and overweight (including obesity) (BMI,IOTF) children in lower and higher income groups.

	Non-overweight		Overweight/obese	
	Lower income	Higher income	Lower income	Higher income
z-weight	-0.13 ± 0.92 (n=856)	-0.07 ± 0.88 (n=1010)	1.91 ± 0.78 (n=259)	1.74 ± 0.58* (n=173)
z-%BF	0.13 ±0.86 (n=840)	-0.22 ± 0.84*** (n=1010)	1.79 ± 0.60 (n=254)	1.47 ± 0.61*** (n=173)

* p<0.05, **p<0.01, ***p <0.001

Table 4.4: Mean age- and sex-adjusted weight and %BF of normal fat (% BF <85th centile) and overfat (including obesity, %BF >85th centile) children in the lower and higher income groups.

	Normal fat		Overfat/obese	
	Lower income	Higher income	Lower income	Higher income
z-weight	-0.15 ± 0.97 (n=747)	-0.07 ± 0.91 (n=989)	1.43 ± 1.06 (n=348)	1.55 ± 0.74 (n=194)
z-%BF	-0.04 ± 0.75 (n=746)	-0.27 ± 0.79*** (n=989)	1.73 ± 0.50 (n=348)	1.58 ± 0.41*** (n=194)

* *p*<0.05 **p<0.01 ***p <0.001

Figure 4.1: Age- and sex-adjusted height of non-overweight and overweight (including obesity) (BMI, International Obesity Task Force) children in the lower and higher income groups. Values = Mean ± SEM.

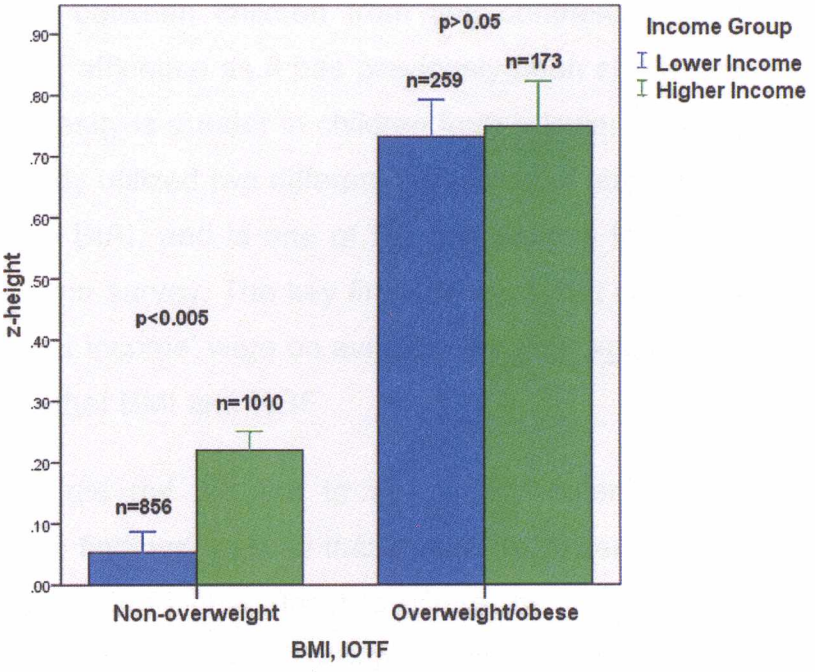
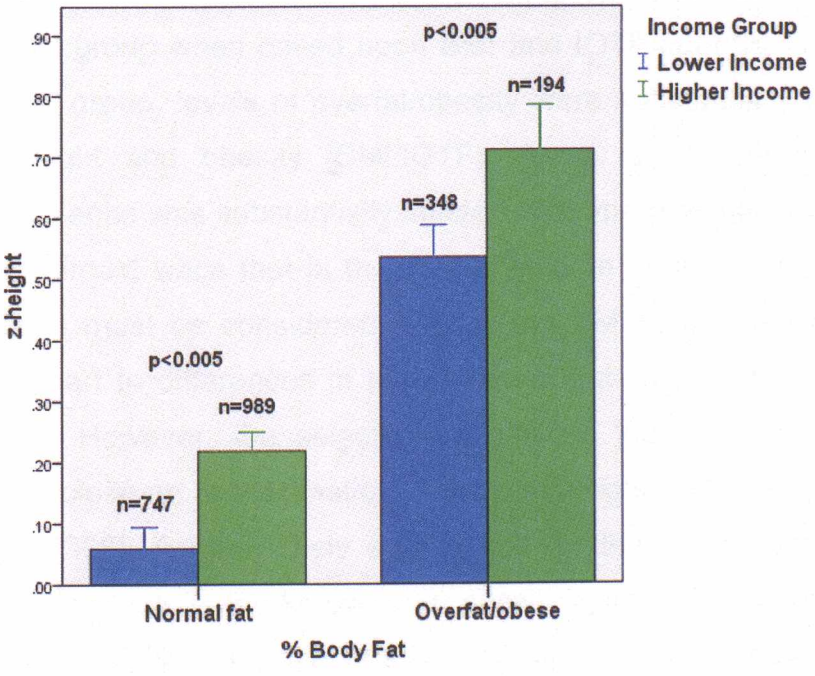


Figure 4.2: Age- and sex-adjusted height of normal body fat (% BF <85th centile) and overfat (including obesity)(%BF >85th centile) children in the lower and higher income groups. Values = Mean ± SEM.



4.4 Discussion

This cross-sectional study set out to compare anthropometric measures of growth and body fatness between children from two contrasting population groups with respect to levels of affluence as it has previously been shown that the prevalence of overweight and obesity is greater in children from a lower income background. At the same time this study utilized two different measures of adiposity – BMI and %BF (the latter derived from BIA), and is one of the first studies to employ both assessment methods in the same survey. The key findings were that children from the population group termed 'lower income' were on average (for their age) shorter in height, heavier in weight with a higher BMI and %BF.

Whilst this study did not set out to specifically determine prevalence levels of overweight/obesity, findings showed that within the 'lower income' group, prevalence (23.2%) was similar to national statistics where the IOTF cut-offs have been used, whereas for the 'higher income' group it was lower (14.6%) [367]. The lower prevalence in this sample of 'higher income' children was expected [308] although it is most likely not a true representation of the national prevalence within this income group. Furthermore, when prevalence levels were quantified using %BF, further important observations were made. Firstly, levels of overfat and obesity were only slightly increased in the 'higher income' group compared with levels of overweight and obesity in that group when based upon BMI and IOTF cut-offs. However, within the 'lower income' group, levels of overfat/obesity were substantially higher than its levels of overweight and obesity (BMI/IOTF). As a result, the 'income group' difference in prevalence was substantially inflated, with the prevalence of overfat and obesity reaching almost twice that in the 'higher income' group. The implications of these observations must be considered. Firstly, the BMI-%BF prevalence disparity could be due, in part to differences in the choice of cut-offs used between the two assessment tools. However, the selected cut-offs for %BF had specifically been chosen to form as close an approximation to the overweight and obese boundaries of the IOTF curves [308]. A more likely explanation for the difference in prevalence between assessment tools is the known poor sensitivity of BMI (moderate-high false negative rate) i.e., it's failure to identify all children with a high body fat content as overweight or obese [315]. Thus these children would be misclassified as 'normal

weight', therefore leading to an underestimation of the true prevalence figures. This study has clearly highlighted this deficiency of BMI and most likely provides a closer approximation of the actual prevalence levels. An additional and intriguing finding was that BMI underestimated the true prevalence of high body fatness/obesity in the lower income group to a greater extent than in the higher income group. This appears to be the first time that this income group phenomenon has been observed and has important implications for the interpretation of prevalence statistics based upon BMI. Exactly why BMI appears to function differently along income group divisions in unclear, but could be explained by the fact that children from lower income backgrounds were shorter and heavier, with a greater proportion of the weight being accounted for by body fat. This phenotype would more likely be categorized within the normal BMI range compared with taller children, as it has been shown that BMI is not independent of height in children [368].

This study also demonstrated key differences between children from different income groups with respect to their age-specific body measurements. The small but significantly greater body weight for age is an obvious contributor to the higher age-specific BMI in the 'lower income' children and the higher prevalence of obesity in this group could be explained by this one factor. However of more interest is the significantly shorter height-for-age in the 'lower income' group compared with those from the 'higher income' group. This difference amounted to a mean of approximately 1.3 cm, although given the wide age range of the children in this study, absolute differences in mean height are less meaningful that differences in height z-score. A similar income group height difference has also been observed in a study that examined childhood obesity and socioeconomic status in a Scottish population [363]. However, in that study the age range of the children was narrower and the mean age younger than in this study (7.43y vs 8.86y). Similar income group differences in height and hence BMI have also been observed in a Canadian population [369], indicating this phenomenon to be reproducible across population groups. Clearly shorter height is a second (and likely a more significant) contributor to the greater BMI, BMI z-score and overweight/obesity prevalence in lower income children.

In our study, whilst this income group difference in height was observed at a whole group level and within the normal BMI category, this was not the case in the overweight/obese category (even though in absolute terms the overweight/obese

children from the lower income background were significantly shorter) despite children in this group being taller than those in the normal weight category. This one observation contrasted with that in the Scottish study [363]. One possible explanation for this discrepancy between these studies is that the overweight/obese children in the low-income group were, on average, slightly younger in age (8.78y vs.8.93y, p=NS) – a finding in itself that merits additional investigation. Moreover, these income-height-BMI findings were further supported when children were classified by body fatness (ie normal fat and overfat/obese). In children who were classified as having a body fat level within the normal range, those from the 'lower income' group were significantly shorter. At the same time, whilst the overfat/obese children as a group were significantly taller than normal fat children, those from the 'lower income' group were still significantly shorter than the 'higher income' children. This observation thus supports the hypothesis of a relative height growth limitation in children from low-income backgrounds, proposed by Cecil et al. in the Scottish study [363]. Height growth limitation in overweight/obesity is considered unusual particularly as it is understood that overweight/obese children tend to be taller than their non-overweight/obese counterparts. However, it can be a feature of some populations in developing countries [370].

This apparent relative height-growth limitation in overweight/obese children from a lower income background is a finding that requires careful consideration. Whilst height-growth restriction generally reflects sub-optimal nutritional experience at some stage of (early) development, the excess body fat accumulation at the time of measurement suggests, paradoxically, that energy availability and energy intake has not been compromised and on the contrary has been in excess of requirements. Furthermore, parental height was not obtained in this study and it is known that child height is a reflection of parental height (genetic influence) and height is known to be positively related to socio-economic status (genetic and environmental influence) [371,372].

This is the first study to utilize BIA technology to explore variations in body fatness between children from contrasting income backgrounds. This was achieved, in part, by the availability of new body fat reference data for UK children [308]. It should be remembered that BIA predicts body fatness using an algorithm based upon a number of variables including height and whole-body impedance. The extent to which the

group differences in height contributed to the differences in predicted %BF is unclear, but should not be overlooked. Impedance is itself a function of height, whereby height acts as a surrogate for conductor length [302]. Nevertheless methodological procedures, equipment used and measurers were identical in both surveys and so any variance in these aspects can be ruled out.

This study has also produced interesting observations with respect to upper body fatness in children and income status. It has been observed that excess body fat accumulates predominantly in the upper body rather than in the peripheral region in obese children and adolescents [327,373]. This was evident in this sample by the very-much increased z-WC in the overweight/obese or overfat/obese children compared with non-overweight/normal body fat children. However, measurements of WC and derivation of WC z-scores indicated that age-related upper body fatness was greater in the children from the higher income group. At first sight this would appear paradoxical, given that whole body fatness was greater in the children from the lower income background. However, this study has highlighted for the first time the importance of considering height when interpreting WC measurements. The fact that height and WC are correlated suggests that any increase in WC during childhood is due, in part, to linear growth [348]. Nevertheless, the precise effects of stature upon WC is still quantitatively unclear but should be considered, particularly, when age-related variations in WC are examined. The height-WC issue can be overcome, in part, by calculating the WHtR. This index gives a measure of proportionality, i.e. whether for a given height, there is an acceptable amount of fat stored on the upper body. A boundary value of WHtR=0.5 has been proposed as a means of indicating whether the amount of upper body fat accumulation is excessive and poses a risk for health [348]. In this study, the mean WHtR was identical between income groups, suggesting that the upper body fat accumulation or distribution was no different in children between income groups, thus contradicting the findings based solely on WC. WC centile charts for children and adolescents in the UK [342] and across the world are age-specific and further research is required to quantify the height-age WC inter-relationships. Furthermore, it should be noted that mean WHtR was greater in the children in this study compared with children (aged 5 to 16 years) measured in 1977 and 1988 (boys=0.432, girls=0.420) as reported in McCarthy & Ashwell [348]. This observation would confirm that the obesity epidemic is not reversing and in fact upper

body fatness is still increasing in UK children. When overweight/obese children were examined further (using WHtR), then those from the lower income group had a greater centralized distribution of body fat as well as a greater overall body fatness. Thus, overweight/obese children from the lower income group have greater overall fatness and centralized distribution (using WHtR) of fat compared with equivalent children from a higher income group.

It can be concluded that overweight/obese children from a population group considered 'lower income' are overall shorter, heavier and have greater overall body fatness compared with equivalent children considered to be from a 'higher income' background. The explanations for these observations are not straightforward but certainly merit further investigation. Furthermore, the impact of these findings on risk for obesity-related morbidity in children across income groups could be important.

Chapter 5: A study on the assessment tools used to evaluate childhood obesity weight management intervention

5.1 Introduction

5.1.2 Background on weight management interventions

Child and adolescent obesity has become a major public health issue both in the UK and internationally [14]. Despite the implementation of clinical management and public health strategies to combat this rise, recent estimates continue to demonstrate an upward increase in prevalence. Most recent estimates from the Health Survey for England 2007 (based on national BMI cut offs) approximate 14.1% of boys and 16.1% of girls (aged 2-15 years) to be obese [17].

The increase in obesity is coupled with an increase in the negative health consequences and a number of studies have demonstrated the presence of adverse risk factors in overweight and obese children and adolescents [374-378]. The emergence of Type 2 diabetes in young people and the rise in cases identified, gives evidence for the severity of the situation [37,379]. Furthermore, the increased risk of child and adolescent obesity persisting into adult obesity has also been widely demonstrated [60,380,381]. The true scale of the problem in public health terms has become apparent in the escalating costs of treating obesity and its consequences [26].

The multifaceted nature of the aetiology of obesity has required its management to follow a diverse and complex approach [381]. Treatment of obesity can focus on either individual approaches such as behaviour modification, dietary restriction, physical activity, pharmacotherapy or encompass a combination of these approaches [310,314,382]. Strategies in the UK designed to combat obesity include school-based interventions (National Healthy Schools Programme) [281], weight management clinics/programmes located in acute [383,384] and primary care settings [385,386] and residential weight loss camps [387].

Evaluation of such interventions has so far failed to draw conclusions on their effectiveness. The diverse nature of the vast range of interventions makes it difficult

to extrapolate generalisable recommendations. One particular factor that varies between interventions is the short and long-term outcomes applied to assess the success of the intervention [388].

Outcomes can range from observing changes in psychosocial aspects such as self-esteem [389] or behaviours such as habitual physical activity [390,391] and dietary intake and eating habits [392]. Furthermore, changes in biological/physical outcomes are more commonly used to assess the efficacy of intervention programmes. Such outcomes can be in the form of biochemical assessment of obesity-related risk such as blood lipids or anthropometric measurements such as height, weight and indirect measures of body composition [383,387,393-397]. The choice of biological outcomes often depends on the setting of the intervention and the resources available. The use of anthropometry is cost-effective and can be easily applied, therefore it is commonly used in community based studies, whereas interventions based in clinical settings often have access to more sophisticated techniques which give an indirect measure of changes in both fat mass (FM) and fat free mass (FFM).

Intervention studies have focused mainly on body weight as an outcome because it is assumed to reflect the health and nutritional status of an individual [287,398]. Furthermore, obesity has been defined as an increase in body weight beyond skeletal and physical standards as the result of an excessive accumulation of fat in the body [399]. This would suggest that a reduction in body weight would lower the degree of overweight and the associated health risks. Furthermore, studies have demonstrated that weight loss in obese children has been associated with an improvement in metabolic risk factors [400-403].

The most commonly used outcome measure in the management of obesity is the Body Mass Index (BMI) [404]. BMI is an index of weight corrected for height and in children adjusted for age and gender [5]. BMI is considered a useful proxy measure of adiposity hence changes in BMI following obesity intervention potentially reflect changes in body fatness. Changes in BMI can be expressed in absolute terms, or converted to an age and gender specific centile or z-score, or as a change in % BMI [405]. Only a small number of studies have assessed the outcome of obesity interventions in children using one of these BMI measures and have also quantified the levels of BMI z-score reduction that is likely to result in loss of adiposity (loss of

0.6k) [406] and improvement of cardiovascular and endocrine risk factors (loss of 0.5) [401,402].

One limitation with using BMI as an outcome measure in children and adolescents is that, weight and height changes are already inherent in this population group due to growth [407]. In view of this important biological concept and its relation to weight management, a detailed account of the biology of human growth now follows.

5.1.2 Biology of growth and its relationship with body fatness

Growth is a dynamic process that is ongoing from the pre-natal stage to early adulthood. It is characterized by physical, hormonal and body compositional changes which occur in a somewhat-stage wise process and are, in part, gender specific. Physical growth involves multidimensional increases in the body as a whole and in individual organs and systems. It is often quantified by measures of length, weight, surface area, volume and circumferences. Body compositional changes during growth follow patterns of acceleration periods often referred to as growth spurts and also deceleration periods during which changes are minimal or reduced [408-411].

The most pronounced growth in body proportions occurs during infancy. The first year after birth is characterized by a substantial increase in body weight (200%), length (55%) and head circumference (30%) [409]. Between the first and second year, increases of up to 12cm in length and up to 3.5kg in weight have been documented. This phase is followed by a sharp decline in the rate of linear growth during which annual increments in body proportion are not as marked as earlier years. Annual increments in height and weight between the fourth and fifth year of life are approximately 6cm and 2kg respectively. This phase is characterized by an increase in height, which occurs at a greater rate than the increase in weight. The slower rate of increase in weight during this period results in a decrease in BMI which is displayed on all BMI reference curves (such as the UK BMI references) as a trough with a lowest point around age 6 years (figure 5.1)[5]. During these years, gender differences in body proportions are only very slight [411].

The latter years of childhood (7-10 years) continue to show a steady increase in growth. Annual gain in weight is approximated at 3kg for 7 years olds and 4kg for 10 years olds. An annual increment of up to 5cm in height has been observed for

children between ages 7 to 10 years. Gender differences become evident at this stage of development with males becoming taller than females at age 7yr whereas females tend to be taller and heavier than males by the age of 10 years. A larger proportion of the increase in height during childhood is accounted for by growth in the lower extremities compared to growth of the trunk [412].

Puberty can begin from age 10yr onwards in girls (and from around age 12 years in boys) and is an essential stage of growth during which unique body compositional changes occur in males and females. Puberty is characterised by a further growth spurt during which a significant proportion of adult height and weight is achieved. It tends to commence earlier in females and continues for a period of 2-2 ½ years. Increases in height of up to 16cm in females and up to 20cm in males have been documented during this growth spurt and the growth of the trunk exceeds the growth of the lower limbs [413]. Furthermore, a variation in height growth velocity is evident throughout the pubertal growth spurt, with a period of peak height velocity occurring at age 12 and 14 years for females and males respectively. Similar figures have also been stated (16kg and 20kg) for gain in weight during the adolescent growth spurt [410]. These increments in height and weight are reflected in a rapid increase in BMI, characterized by a maximum velocity at ages 13 and 14 years in females and males respectively (figure 5.1)[5]. These figures may not be true for all individuals as individual increases in weight and height vary both within and between populations. They do however demonstrate that changes in height, weight and BMI are intrinsic to the dynamic process of growth and that these changes occur at varying rates throughout this period.

Additionally, age-related changes in BMI do not necessarily reflect change in body composition i.e. fat mass and fat-free mass [315,414]. For instance, the rapid pubertal increment in BMI is largely due to increase in fat free mass in males whereas in females this is due more to the increase in fat mass [415]. Such inherent gender differences are masked by BMI and can be further complicated during obesity intervention since the nature of the intervention can have differing effects on body composition. For instance, obesity interventions focusing on increasing physical activity can result in increases in fat-free mass [416] as well as a decrease in abdominal fatness [417] whereas interventions based on dietary restriction alone can result in loss of fat mass and to a certain extent, an unfavourable loss of fat free mass

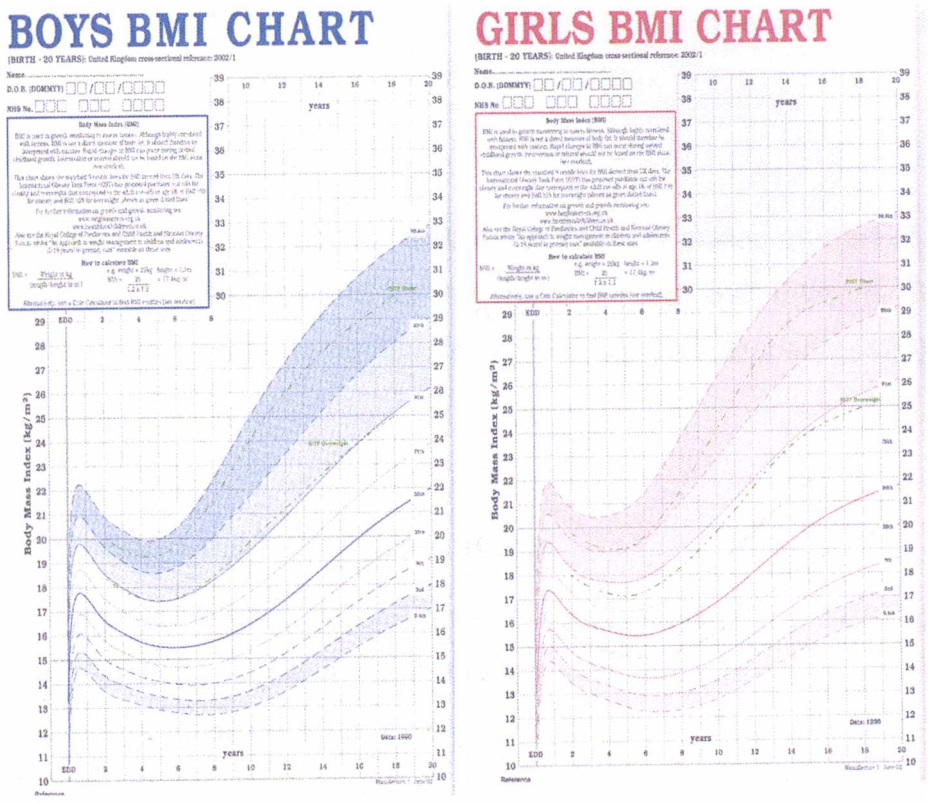
[394,418]. Hence assessment of obesity interventions using BMI alone would mask any essential changes that are due to the consequence of growth together with the beneficial or adverse effects of the intervention [407]. Therefore alternative assessment tools, which focus upon both quantitative aspects of body composition and regional adiposity, are likely to be of more value in a paediatric weight management context.

Assessing changes in body composition beyond that offered by BMI has been shown to be effective in evaluating an obesity intervention [405]. Support for this can be drawn from the World Health Organization (WHO) definition, which indicates that it is the body fat compartment of overweight that has negative heath consequences for the individual [399]. Secondly, there is evidence of an association between body fat and adverse risk factors (raised blood pressure, insulin resistance, dyslipidaemia) in children [46,127] and mortality in adults [419].

As outlined in chapter 2, body composition can be assessed using methods such as DXA and hydrodensitometry or indirect methods based on predictive equations. Routine use of the above methods are limited because they are invasive, expensive and unsuitable for clinical and community settings. Therefore a number of studies have used indirect predictive methods such as skinfold thickness and BIA to assess changes in body composition following an intervention programme [395,406].

Recent technological and practical advances in BIA have further promoted its use in this area. Newer model BIA systems now consist of contact electrode systems encompassed into a step on weighing scale, thereby eliminating the need for gel electrodes. Thus, the precision of the measurements has been greatly improved, with the degree of accuracy being of a higher and acceptable level (given the simplicity of the procedure and relatively low risk and high speed of measurement). The Tanita BC-418MA Segmental Body Composition Analyzer (Tanita Corporation, Tokyo, Japan) represents an advanced BIA system that has been designed to collect whole body and segmental impedance measurements using an 8 contact electrode system. Recently, body fat reference charts for Caucasian children have been developed using this BIA system and have been introduced in the UK together with the reference data and software to allow determination of % Body Fat z-scores [308].

Figure 5.1: UK Body Mass Index chart for boys and girls [Source:312]



5.2 Aims and Objectives

5.2.1 Aims

In view of the increase in obesity prevalence and the demand for effective weight management programmes for obese children, the aims of this study were:

1. To examine changes in measures of body composition and body fat distribution in obese children enrolled in a weight management programme.

2. Compare body composition changes in obese children within the programme who either gained or lost body fat (kg) by the end of the programme.

3. To evaluate the potential for waist circumference (WC), Waist-to-height ratio (WHtR), Fat Mass Index (FMI) and fat Free Mass Index (FFMI) as assessment tools or markers of changes in body fatness in obese children enrolled onto a weight management programme.

4. To evaluate the varying body composition tools and to suggest recommended best practice.

N.B. This study did not directly set out to evaluate the effectiveness of a weight management intervention programme for obese children.

5.2.2 Objective

The objective of this study was to interrogate the data generated as part of a weighing and measuring service we provided for the Better Eating, Self Esteem, Total-Health (BEST) weight management intervention for children and adolescents [420].

5.3 Methodology

5.3.1 Data collection-the BEST programme

Data for this study was collected from the BEST programme. The BEST programme is an obesity treatment programme for children and adolescents. It was founded in 2003 following the completion of its pilot project, Healthy Lifestyles. Both BEST and Healthy Lifestyles are community-based programmes held within schools or sports centres in the borough of Tower Hamlets.

5.3.1.1 Demographic characteristics of the London Borough of Tower Hamlets

Tower Hamlets is a densely populated inner East London Borough. It is the most deprived borough in London and the third most deprived local authority in England. Most recent demographic figures on the borough come from the 2007 Office for National Statistics survey which estimated a population of 215,300. The survey also demonstrated that 56% of the population was from a non-white British background and that over a third of the population was Bangladeshi [421].

The relationship between deprivation and ill health in the borough is reflected in a standardized mortality rate (directly standardised rate per 100,000 European Standard population) that is higher than that for the rest of the country. Non-communicable diseases such as coronary heart disease and diabetes commonly found in people from South Asian and African-Caribbean backgrounds are highly prevalent in the borough with figures exceeding those of the rest of the country [422].

The BEST programme was set up as an adjunct to the Royal London Hospital obesity clinic. The rationale behind this set up was to enable patients to increase habitual levels of physical activity thereby assisting with weight loss/maintenance. The programme is currently managed by the Healthy Lifestyles team, which forms part of the Communities, Localities and Culture Services of Tower Hamlets Council. This service partly funds the programme with Tower Hamlets PCT.

The chief facilitator of the programme was from a sports science background with experience in community based sports programmes. Assistant facilitators also had previous experience as sports coach for children. Educational psychologists attended two sessions to advise and inform parents on how to make long-term behavioural changes to promote healthier lifestyles for the whole family.

5.3.1.2 Aims of the BEST programme

The aims of the programme as outlined on promotional material were as follows:

- To promote healthy lifestyles and increased opportunities for physical activity for clinically obese 8-16 year olds.
- To increase the long term participation in an active lifestyle
- To form working alliances with health professionals, psychologists and schools

5.3.1.3 Structure and content of the BEST programme

The programme was made up of two stages. The first stage was termed the intensive stage during which the participants attended twice weekly sessions for a period of ten weeks. The second stage was the maintenance phase during which participants attended reunion sessions for a period of six months. All sessions were held on weekdays following the end of the school day.

5.3.1.4 Stage 1 of the programme

The twice-weekly sessions within the intensive stage consisted of 30-minute workshops during which nutrition related topics and activities were covered. Topics covered included promoting completion of food diaries, encouraging and increasing the intake of fruits and vegetables and decreasing and monitoring the intake of foods and drinks high in fat and sugar. Guidance was provided on portion sizes, food labels, hidden fats and dealing with tricky situations such as eating out or parties. These

topics were covered in the form of whole group and mini-group discussions, quizzes, self-reflection and motivational activities. Homework on the respective topic was set at the end of each workshop session to reinforce information discussed in group settings.

Following the workshop, the participants engaged in a range of sport based activities. These were conducted for a period of 60 minutes during which the participants were involved in team-based sports such as basketball or non-competitive activities such as trampolining or swimming. Concurrently parents continued to participate in a group based workshop with an aim of promoting nutritional knowledge and healthy eating for the whole family.

5.3.1.5 Stage 2 of the programme

The maintenance phase was targeted at weaning the participants off the intensive support provided during the first stage. This period also widened the opportunity for the participants to continue incorporating positive behaviour learnt as part of their daily lives. Participants were invited to monthly "fun-fit reunions" sessions during which they participated in sport-based activities for 60 minutes. One of these sessions was dedicated to a group discussion. Throughout this stage, participants received supporting written material on topics they were struggling with. Parents were also offered a similar discussion session to go over material or information covered in the intensive stage.

5.3.1.6 Participant selection and recruitment

Participants were recruited for the programme based on the following referral criteria:

- BMI $\geq 98^{th}$ centile (British 1990 Growth Reference Data, Child Growth Foundation)
- Child and Parent willing to participate for the full course of the programme
- Consent for participation obtained from parent and child.

Referrals for the programme are currently received from Obesity Specialist Paediatric Dietitians, School Health Advisors, General Practitioners (GPs), Parent Liaison Officers and Learning Mentors.

The BEST programme allowed us the opportunity to provide a weighing and measuring service to the programme, whilst at the same time allowing us to use the data to characterize and evaluate body compositional changes in a paediatric obese population undergoing a weight management intervention.

5.3.2 Data collection-Anthropometric and body fat measurements

The aim was to collect the measurements in the first (initial measurement) and final (final measurement) sessions of the programme (Stage 1), however, this was not always possible due to practical issues. Factors such as participant absence and school/public holidays meant measurements had to be obtained at the next possible opportunity.

Measurements took place on the premises of the programme within private rooms at the beginning of the session. Participants were measured on an individual basis to maintain confidentiality. Participant date of birth and ethnicity were recorded at the time of initial measurements (start of programme). All measurements were carried out by the same measurers (DR, DMc) throughout the course of this study.

Measurement procedures for initial and final measurements were identical. Height was measured to the nearest 0.1cm with a portable stadiometer (Seca stadiometer, Marsden UK, Mod 220) with participants standing in bare feet. Waist circumference (WC) was measured between the 10^{th} rib and the iliac crest over a single layer of clothing, with correction as previously described [2,327]. Both height and waist measurements were conducted by a single measurer to exclude inter-observer error. Body mass (measured to the nearest 0.1kg with correction for light indoor clothing) and whole body % Body Fat was measured using the Tanita BC-418MA Segmental Body Composition Analyzer. This required participants to stand on the analyser in bare feet whilst holding a pair of handgrips.

In addition to using the Tanita BC-418MA estimates on FM (kg) and FFM (kg), recently developed equations based on a 3C model were also used to assess change (Wells, J.C. personal communication). The authors of this equation demonstrated that the Tanita BC-418MA equations overestimated FFM and underestimated FM in obese children because hydration of FFM is underestimated. Hence, equations to predict FM and FFM in obese children were generated using a 4C and 3C model. For

this study, only the 3C equation was used to draw estimates on FM (kg) and FFM (kg) as the accuracy of this equation in predicting change in body composition has been assessed.

5.3.3 Sample size and ethnicity

Between 2003 and 2008, a total of 190 participants were enrolled onto the programme. Typically 22 participants were recruited for each cycle, however number of participants that attended the measurement sessions varied in each cycle and attrition rates were typically 25-50%. Participants were included in the analysis if both initial and final measurements had been obtained. As the programmes were identical in composition, data obtained from all children measured was pooled. For this analysis, a total of 95 (44 boys and 51 girls) obese children were included from 10 programmes.

Ethnicity was coded using a condensed version of the Department for Children, Schools and Families ethnicity classification (Appendix B & C) [354].

5.3.4 Ethical approval

This study was approved by the London Metropolitan University Ethics Committee (Appendix F).

5.3.5 Data handling and statistical analysis

Data collected from all participants were entered onto statistical software packages (Microsoft ® Office Excel 2003 and SPSS 14.0 for windows, Release 14.0.0, 5 Sep 2005, Chicago, SPSS Inc) either directly or calculated using a formula. Variables that were directly entered onto the spreadsheet were: anonymised child code, gender, ethnicity, date of birth, date of measurement, height (cm), weight (kg), WC (cm) (before correction for clothing), % Body Fat, Fat Mass (FM) (kg), Fat Free Mass (FFM) (kg), and whole body Impedance (Ω),

Variables that were calculated using a formula were:

- Decimal age (years): (Date of measurement – Date of birth) / 365.25
- Height (m): Height (cm) / 100
- WC after correction for clothing: WC (cm) – 0.5
- Body Mass Index (BMI) (kg/m²): Weight (kg) / Height ² (m)
- Waist to Height ratio (WHtR): Waist (cm) / Height (cm)
- Fat Mass Index (FMI) (kg/m²): Fat Mass (kg) / Height² (m)
- Fat Free Mass Index (FFMI) (kg/m²): Fat Free Mass (kg) / Height² (m)
- Z-scores (or Standard Deviation Scores (SDS)) and percentiles were calculated for Height, Weight, BMI, WC and % Body Fat using the 1990 British reference data [5], UK WC data [342] and UK %BF data [308].
- FFM (kg) was also calculated using the following 3C equations (Wells, J.C. personal communication):
- -2.211 + 1.115 (Ht²/R). where R = Impedance
- FM (kg) (3C) was then calculated: Wt (kg) – FFM (kg)
- %BF (3C) was calculated: FM (kg) / Weight (kg) * 100
- FMI and FFMI (3C) were calculated as above.

Statistical analysis was based on examining differences between the means of initial (pre-programme) and final (post-programme) variables. This was conducted on a whole group basis as well as in boys and girls separately. Following this, the participants were divided into those that had either gained fat mass (kg) and those that lost or maintained fat mass (kg). Based on this division, two groups were formed- the Fat Mass Gainers (FMG) group and the Fat Mass Losers (FML) group. A comparison between the two groups was conducted on the initial variables, the final

variables and the change in the variables (initial reading - final reading). Finally a comparison between the initial and final variables was also conducted in both the groups.

Preliminary statistical tests were conducted to provide guidance on the choice of statistical tests appropriate to make the aforementioned comparisons. Firstly the distribution of all variables was assessed as this determines whether a parametric or non-parametric test would be used for the analysis. Two tests of normality namely the Kolmogorov-Smirnov (KS) and Shapiro-Wilk (SW) were used to define the distribution of all variables on a whole group basis, in boys and girls separately and in the FML and FMG groups. In the case where there was a discrepancy between the two tests, normality of the data was assessed by examining histograms and in most instances, the SW reading was taken to provide an accurate assessment of normality as this is a statistically more powerful test [423].

It was observed that a large number of the variables were non-normally distributed and were characterized by a positive skew. There was also variation between the individual variables with regards to distribution in the initial and final measures. Some variables showed normal distribution both in the initial and final variables whereas other showed a positive skew at both points. However, there were some variables where the distribution of the initial and final variables differed. This variation in the distribution of the variables complicated the decision on the choice of statistical test.

Advice was sought from a statistician based within the university's Statistics, Operational Research and Mathematics (STORM) research centre. The following points were raised by the statistician:

- It is the distribution of the change in variables that should be assessed rather than the initial and final measurements themselves.
- Observing histograms of the change in all variables demonstrated that changes were not large hence it was unlikely that outliers (which would skew the variable) were present in the data.
- Non-parametric tests are statistically less powerful than parametric tests

Based on above points, I was advised to use a parametric test for the purpose of this study. However, it is also known that conducting non-parametric tests on normally

distributed data increases the chance of a Type II error [423]. This type of error is associated with increasing the chance of accepting that a difference between groups does not exist when in actual fact there is one.

Based on this information, all comparisons were examined using a parametric and non-parametric test. The paired-samples t-test and Wilcoxson Signed Rank test were used to examine if differences existed between the initial and final variables on a whole group and gender basis as well as in the FML and FMG groups. The independent-samples t-test and Kruskall Wallis test were used to examine if differences existed between the FML and FMG groups in the initial variables, the final variables and the change in variables. A $p<0.05$ was taken to indicate a statistically significant difference.

A comparison of the findings from the parametric and non-parametric tests revealed that both types of tests offered similar results. Thus, where a statistically significant difference was observed for a parametric test, a similar result was obtained from the non-parametric test and vice versa. Similarly, coherence between the parametric and non-parametric tests was also observed for non-significant differences ($p>0.05$). Given that coherence was observed between the two types of tests, results from the parametric tests have been presented in this chapter (see Appendix E for results from the non-parametric tests).

5.4 Results

A total of 95 subjects (44 boys and 51 girls) completed the BEST programme between 2003 and 2008 for whom we had complete data. Initial and final measurements obtained from these subjects were included in this analysis. All subjects had a baseline BMI ≥ 98th centile which was in line with the eligibility criteria for the programme.

Mean values for the initial and final measurements are shown for the whole group (Table 5.1) and for each gender (Boys: Table 5.2; Girls: Table 5.3). Changes in these measurements over the course of the programme are described below.

Whole group decimal age increased from 12.0 to 12.2y but boys were on average older than girls at the start of the programme.

Absolute height increased significantly at the group level over the course of the programme (p<0.001). This gain in height remained significant at the gender level with a mean increase of 1.3cm (p<0.001) and 1.1cm (p<0.001) in boys and girls respectively. A small but non-significant increase in z-height was observed both at whole group and gender level (as a side issue, a comparison of z-height between the obese subjects in this study and a non-overweight/obese Bangladeshi sample (n=401, z-height= -0.32) from the same London Borough demonstrated that the obese children were taller for their age, in line with our observation for children from Caucasian and African-Caribbean backgrounds).

A small but significant (p<0.05) increase in absolute weight was observed at the group level but this was not statistically significant when boys and girls were analyzed separately. In contrast z-weight decreased significantly over the course of the programme for the whole group (p<0.01) and in boys (p<0.01) but this decrease was not statistically significant in girls.

The increase in height and decrease in z-weight was reflected in a decrease in absolute BMI (p<0.05), z-BMI (p<0.001) and BMI centile (p<0.01). A statistically significant decrease in these variables was apparent at whole group level but not

always at the gender level. For the whole group, a 0.3kg/m² decrease in BMI corresponded to a 0.06 unit decrease in z-BMI and 0.09 decrease in BMI centile.

Absolute BMI (kg/m^2) decreased significantly in the whole group (-0.3kg/m^2, $p<0.05$) and in boys (-0.4kg/m^2, $p<0.01$) but the decrease was not statistically significant in girls (-0.1kg/m^2, $p<0.01$). BMI centile decreased significantly at group level (-0.09, $p<0.01$) and in girls (-0.15, $p<0.01$) but the decrease was not statistically significant in boys (-0.03, p=NS). Mean z-BMI decreased significantly in both boys (-0.08, $p<0.001$) and in girls (-0.05, $p<0.01$) with a slightly greater decrease in boys.

Change in upper body fatness was examined using absolute WC (cm), z-WC and WHtR. A significant decrease in absolute WC ($p<0.01$), z-WC ($p<0.001$) and WHtR ($p<0.001$) was seen at the whole group (n=88) level (-1.4cm, -0.11 and -0.014 respectively) and in girls (-2.5cm, -0.17 and -0.021 respectively). A small but non-significant decrease was observed in all measures of upper body fatness in boys.

Whole body impedance obtained from the BIA analyser decreased significantly at whole group level by 8Ω ($p<0.01$) and in girls by 12Ω ($p<0.01$). A small but non-significant decrease in impedance was seen in boys (5Ω, p=NS).

Variables describing whole body composition (%BF, FM and FFM) were obtained from the BC-418 analyzer (manufacturer's equations) and recently validated 3C equations. The difference in body composition readings between the two sets of equations followed the pattern described by the authors of the validated equations (Wells, J. personal communication). As shown in Tables 5.1, 5.2 and 5.3, %BF values obtained from the 3C equations were greater by an average of 3.9% at the whole group level and by 3.1% in boys and 4.5% in girls. Total fat mass (kg) was on average higher when using the 3C equations at whole group level (by 2.7kg), in boys (by 2.2kg) and in girls (by 3.0kg). FFM was on average 2.7kg, 2.2kg and 3.1kg greater at group level and in boys and girls respectively using the manufacturer's equations. Hence, FFM appeared to be overestimated and FM underestimated in obese children using the manufacturer's equations.

Initial and final measurements of %BF, z-%BF and FM (kg) (using manufacturer and 3C equations) were greater in girls than in boys indicating greater whole body fatness in girls. Tables 5.1, 5.2 and 5.3 show that %BF and z-%BF decreased significantly at

both whole group and gender level using both sets of equations. An average decrease of 1.5% and 1.4% in %BF was seen in boys and girls between the initial and final measurements. A statistically significant decrease in FM was observed at whole group level (3C= -0.8kg, $p<0.05$) (manufacturer's equations= 0.8kg, $p<0.01$) and in boys (-1.0kg, $p<0.01$) using the manufacturer's equations. The average decrease in FM (kg) (-0.7kg) in girls did not reach statistical significance. FFM (kg) was greater in boys than in girls both at the initial and final measurements. FFM (kg) increased significantly at the whole group level (1.3kg) and gender level with a greater increase in girls (1.4kg) than in boys (1.1kg).

A statistically significant decrease in FMI (3C and manufacturer's equations) was observed at the group level (-0.5 kg/m^2, $p<0.001$) and in both genders with a slightly greater decrease seen in boys (-0.6 kg/m^2, $p<0.01$) than in girls (-0.5 kg/m^2, $p<0.01$). FFMI increased at the whole group level (3C= 0.5 kg/m^2, $p<0.001$) (manufacturer's equations = 0.3 kg/m^2, $p<0.01$) and gender level with a greater increase apparent in girls (3C= 0.6 kg/m^2, $p<0.001$) (manufacturer's equations = 0.3 kg/m^2, $p<0.01$) than in boys (3C= 0.4 kg/m^2, $p<0.01$) (manufacturer's equations = 0.1 kg/m^2, p=NS).

Subjects were sub-divided on the basis of changes in fat mass (kg) into fat losers (FML) and fat gainers (FMG). Table 5.4 shows initial and final measurements and the change in these measurements in subjects who lost /had no change in FM (kg) (FML Group) and those who gained FM (kg) (FMG Group). A total of 58 lost/had no change in FM (kg) (27 boys, 31 girls) and 37 subjects gained FM (kg) (17 boys, 20 girls).

Differences between the FML and FMG groups in the initial and final measurements together with changes in all measurements between the two groups were statistically analyzed (Table 5.4). Differences in the initial (1) and final (2) measurements between the FML and FMG groups were not statistically significant except for the final %BF ($p<0.05$) (3C equations, $p<0.01$) and z-%BF ($p<0.01$). However, change between the initial and final measurements was statistically significant in the FML and FMG groups for all the variables except height (absolute and z-height) and age (y) (Table 5.4).

Subjects in the FML group were younger (p=NS) than the FMG group. Absolute height (cm) and z-height were greater in the FML group at the start of the programme, but this was not statistically significant. Similarly, the gain in absolute and z-height was also greater in the FML group, but again these differences were not statistically significant.

Mean z-weight was slightly higher in the FML group (p=NS) at the start of the programme. Both absolute weight and z-weight decreased in the FML group and increased in the FMG group, resulting in a statistically significant difference in weight change between groups ($p<0.001$).

BMI and z-BMI were similar in both groups at the start of the programme. The change in BMI, z-BMI and BMI centile followed a similar pattern to weight change with the FML group showing a decrease, whereas the FMG group showed an increase in all BMI variables ($p<0.001$).

WC (cm), z-WC and WHtR were similar in the FML and FMG groups at the start of the programme. The FML group showed a decrease in absolute WC ($p<0.001$), z-WC ($p<0.001$) and WHtR ($p<0.001$), whereas the FMG group showed an increase in these variables (statistically significant only in WHtR). The decrease in WC, z-WC and WHtR in the FML group (-2.79 cm, -0.19units and –0.02 respectively) was greater than the increase in these measurements in the FMG group (0.79cm, 0.02 units, 0.001 respectively).

Whole body impedance (Ω) decreased in the FML group (-19.5Ω, $p<0.001$) whereas it increased in the FMG group (7.9Ω, p=NS) with a greater magnitude of change observed in the FML group.

Body fatness (%BF, z-%BF, absolute FM, FMI) was generally greater in the FML group at the start of the programme (p=NS). In contrast, final measurements on whole body fatness were greater in the FMG group ($p<0.01$, $p<0.05$). The FML group lost whole body fat and the FMG group gained whole body fat however the magnitude of change in body fat was greater in the FML group ($p<0.001$).

FFM (kg) and FFMI were lower in the FML group compared to the FMG group at the start of the programme (p=NS). FFM (kg) increased in both groups with a greater

magnitude of change observed in the FML group (FFM=1.9kg & *FFM=2.0kg, p<0.001). FFMI increased in the FML group (FFMI=0.5 & FFMI=0.9, p<0.001) whereas it either decreased (FFMI=-0.14, p=NS) or did not change (*FFMI=0.0, P=NS) in the FMG group.

A larger decrease in z-%BF compared to z-BMI was observed at whole group level and in girls. Mean z-%BF (and *z-%BF) decreased by –0.12 units whereas mean z-BMI decreased by –0.06 units at group level. In girls, z-%BF (and *z-%BF) decreased by –0.14 units and z-BMI decreased by –0.05 units. This pattern was not observed in boys where the decrease in z-BMI (-0.08 units) was similar to the decrease in z-%BF (-0.08 and –0.09 units).

The FML and FMG groups showed a similar pattern (described above) to that in the whole group and in girls. Mean z-%BF in the FML group decreased by –0.25 and –0.26 (using *z-%BF) units whereas mean z-BMI decreased by –0.1 units. Although an increase in both variables was observed in the FMG group, the increase in mean z-%BF (0.10 and 0.11 units) was greater than the increase in mean z-BMI (0.03 units).

Table 5.1: Change in variables on a whole group level (n=95)

Variable	Initial	Final	P
Decimal age (y)	12.0 (± 2.1)	12.2 (± 2.1)	$p<0.001$
Height (cm)	152.6 (± 11.5)	153.9 (± 11.4)	$p<0.001$
z-height	0.53 (± 1.1)	0.57 (± 1.1)	p= NS
Weight (kg)	73.3 (± 21.2)	73.8 (± 21.3)	$p<0.05$
z-Weight	2.77 (± 0.8)	2.73 (± 0.9)	$p<0.01$
BMI (kg/m^2)	30.9 (± 5.7)	30.6 (± 5.7)	$p<0.05$
z-BMI	2.99 (± 0.6)	2.93 (± 0.7)	$p<0.001$
BMI centile	99.44 (± 1.3)	99.35 (± 1.4)	$p<0.01$
WC (cm) (n=88)	94.6 (± 12.4)	93.2 (± 12.4)	p<0.01
z-WC (n=88)	3.35 (± 0.6)	3.24 (± 0.6)	p<0.001
WHtR (n=88)	0.621 (± 0.1)	0.607 (± 0.1)	p<0.001
%BF	40.2 (± 6.6)	38.8 (± 6.5)	p<0.001
*%BF	44.1 (± 6.6)	42.6 (± 7.3)	p<0.001
z-%BF	2.55 (± 0.6)	2.43 (± 0.6)	p<0.001
*z-%BF	2.86 (± 0.5)	2.74 (± 0.6)	p<0.001
FM (kg)	30.2 (± 13.0)	29.4 (± 12.5)	p<0.01
*FM (kg)	32.9 (± 12.9)	32.1 (± 13.1)	p<0.05
FFM (kg)	43.1 (± 10.4)	44.4 (± 10.5)	p<0.001
*FFM (kg)	40.4 (± 10.5)	41.7 (± 10.5)	p<0.001
FMI (kg/m^2)	12.7 (+ 4.4)	12.2 (+ 4.2)	p<0.001
*FMI (kg/m^2)	13.8 (+ 4.2)	13.3 (+ 4.4)	p<0.001
FFMI (kg/m^2)	18.2 (+ 2.3)	18.5 (+ 2.3)	p<0.01
*FFMI (kg/m^2)	16.8 (+ 2.6)	17.3 (+ 2.5)	p<0.001
Impedance (Ω)	629 (+ 87.5)	621 (+ 84.0)	p<0.01
Ht^2/Imp (cm^2/Ω)	38.3 (+ 9.4)	39.4 (+ 9.4)	p<0.001

*%BF, *z-%BF,*FM, *FFM, *FMI: derived using 3C equation

Table 5.2: Change in variables in boys (n=44)

Variable	Initial	Final	P
Decimal age (y)	12.05 (+ 2.2)	12.25 (+ 2.2)	*p<0.001*
Height (cm)	153.8 (+ 11.3)	155.1 (+ 11.4)	*p<0.001*
z-height	0.58 (+ 1.2)	0.60 (+ 1.2)	*p = NS*
Weight (kg)	73.5 (+ 18.5)	73.8 (+ 18.6)	*p = NS*
z-Weight	2.73 (+ 0.6)	2.67 (+ 0.6)	*p<0.01*
BMI (kg/m^2)	30.6 (+ 4.5)	30.2 (+ 4.5)	*p<0.01*
z-BMI	3.06 (+ 0.4)	2.98 (+ 0.4)	*p<0.001*
BMI centile	99.72 (+ 0.6)	99.69 (+ 0.5)	*p = NS*
WC (cm) (n=48)	95.2 (+ 11.0)	95.0 (+ 10.8)	*p = NS*
z-WC (n=48)	3.11 (+ 0.5)	3.07 (+ 0.4)	*p = NS*
WHtR (n=48)	0.622 (+ 0.1)	0.616 (+ 0.1)	*p = NS*
%BF	39.0 (+ 7.1)	37.5 (+ 7.1)	*p<0.01*
*%BF	42.1 (+ 7.1)	40.6 (+ 7.9)	*p<0.05*
z-%BF	2.51 (+ 0.4)	2.43 (+ 0.5)	*p<0.01*
*z-%BF	2.67 (+ 0.5)	2.58 (+ 0.6)	*p<0.01*
FM (kg)	29.3 (+ 11.1)	28.3 (+ 11.0)	*p<0.01*
*FM (kg)	31.4 (+ 11.2)	30.6 (+ 11.6)	*p = NS*
FFM (kg)	44.3 (+ 10.3)	45.4 (+ 10.0)	*p<0.01*
*FFM (kg)	42.1 (+ 10.0)	43.2 (+ 9.9)	*p<0.01*
FMI (kg/m^2)	12.2 (+ 3.8)	11.6 (+ 3.7)	*p<0.01*
*FMI (kg/m^2)	13.1 (+ 3.7)	12.5 (+ 3.9)	*p<0.01*
FFMI (kg/m^2)	18.5 (+ 2.2)	18.6 (+ 1.9)	*p = NS*
*FFMI (kg/m^2)	17.3 (+ 2.3)	17.7 (+ 2.2)	*p<0.01*
Impedance (Ω)	610 (+ 72.8)	605 (+ 68.6)	*p = NS*
Ht^2/Imp ($cm^2/Ω$)	39.8 (+ 9.0)	40.7 (+ 8.8)	*p<0.01*

*%BF, *z-%BF,*FM, *FFM, *FMI: derived using 3C equation

Table 5.3: Change in variables in girls (n=51)

Variable	Initial	Final	P
Decimal age (y)	11.92 (+ 2.1)	12.12 (+ 2.1)	*p<0.001*
Height (cm)	151.7 (+ 11.6)	152.8 (+ 11.4)	*p<0.001*
z-height	0.50 (+ 1.1)	0.52 (+ 1.1)	*p = NS*
Weight (kg)	73.1 (+ 23.5)	73.8 (+ 23.6)	*p = NS*
z-Weight	2.81 (+ 1.0)	2.78 (+ 1.0)	*p = NS*
BMI (kg/m^2)	31.1 (+ 6.6)	31.0 (+ 6.7)	*p = NS*
z-BMI	2.93 (+ 0.7)	2.88 (+ 0.7)	*p<0.01*
BMI centile	99.21 (+ 1.7)	99.06 (+ 1.8)	*p<0.01*
WC (cm) (n=48)	94.1 (+ 13.5)	91.6 (+ 13.4)	*p<0.001*
z-WC (n=48)	3.55 (+ 0.6)	3.38 (+ 0.7)	*p<0.001*
WHtR (n=48)	0.621 (+ 0.1)	0.600 (+ 0.1)	*p<0.001*
%BF	41.3 (+ 6.0)	40.0 (+ 5.7)	*p<0.01*
*%BF	45.9 (+ 5.6)	44.4 (+ 6.1)	*p<0.01*
z-%BF	2.58 (+ 0.6)	2.44 (+ 0.6)	*p<0.001*
*z-%BF	3.02 (+ 0.5)	2.88 (+ 0.6)	*p<0.01*
FM (kg)	31.1 (+ 14.5)	30.4 (+ 13.8)	*p = NS*
*FM (kg)	34.1 (+ 14.2)	33.4 (+ 14.2)	*p = NS*
FFM (kg)	42.1 (+ 10.6)	43.5 (+ 11.0)	*p<0.001*
*FFM (kg)	39.0 (+ 10.8)	40.4 (+ 11.0)	*p<0.001*
FMI	13.2 (+ 4.8)	12.7 (+ 4.6)	*p<0.01*
*FMI	14.5 (+ 4.6)	14.0 (+ 4.7)	*p<0.01*
FFMI	18.0 (+ 2.4)	18.3 (+ 2.6)	*p<0.01*
*FFMI	16.4 (+ 2.7)	17.0 (+ 2.7)	*p<0.001*
Impedance	646 (+ 96.0)	634 (+ 93.8)	*p<0.01*
HT^2/Impedance	37.0 (+ 9.7)	38.2 (+ 9.8)	*p<0.001*

*%BF, *z-%BF,*FM, *FFM, *FMI: derived using 3C equation

Table 5.4: Initial (1) & final (2) measurements and change in measurements in FML and FMG group

Variable	FML (n=58)	FMG (n=37)	*p*	Variable	FML (n=58)	FMG (n=37)	*p*
Age 1	11.88 (±2.0)	12.14 (±2.2)	0.565				
Age 2	12.08 (± 2.0)	12.35 (± 2.2)	0.543				
Change	0.20*** (± 0.0)	0.21*** (± 0.1)	0.260				
Height (cm) 1	152.9 (± 11.8)	152.2 (± 11.0)	0.775	z-height 1	0.65 (± 1.1)	0.35 (± 1.2)	0.238
Height (cm) 2	154.2 (± 11.8)	153.4 (± 10.8)	0.723	z-height 2	0.68 (± 1.1)	0.36 (± 1.3)	0.206
Change	1.29*** (± 1.1)	1.14*** (± 1.0)	0.479	Change	0.03 (± 0.1)	0.01 (± 0.1)	0.308
Weight (kg) 1	74.0 (± 23.5)	72.3 (± 17.4)	0.692	z-weight 1	2.83 (± 0.9)	2.68 (± 0.8)	0.399
Weight (kg) 2	73.4 (± 23.7)	74.4 (± 17.3)	0.817	z-weight 2	2.72 (± 0.9)	2.73 (± 0.8)	0.983
Change	-0.50* (± 1.9)	2.1*** (± 2.3)	0.000	Change	-0.11*** (± 0.1)	0.05* (± 0.1)	0.000
BMI (kg/m^2) 1	30.9 (± 6.4)	30.9 (± 4.6)	0.931	z-BMI 1	2.99 (± 0.6)	2.99 (± 0.6)	0.966
BMI (kg/m^2) 2	30.2 (± 6.4)	31.3 (± 4.5)	0.328	z-BMI 2	2.87 (± 0.6)	3.02 (± 0.6)	0.235
Change	-0.70*** (± 0.8)	0.46** (± 0.9)	0.000	Change	-0.10*** (± 6.4)	0.03 (± 0.1)	0.000
BMI centile 1	99.5 (± 1.2)	99.37 (± 1.3)	0.657				
BMI centile 2	99.31 (± 1.2)	99.42 (± 1.3)	0.732				
Change	-0.18*** (± 0.3)	0.05 (± 0.3)	0.000				
WC (cm) 1	94.7 (± 13.4)	94.4 (± 11.8)	0.924	z-WC 1	3.35 (± 0.6)	3.35 (± 0.6)	0.970
WC (cm) 2	91.9 (± 13.4)	95.2 (± 10.3)	0.194	z-WC 2	3.16 (± 0.7)	3.37 (± 0.6)	0.115
Change	-2.79*** (± 4.1)	0.79 (± 4.1)	0.000	Change	-0.19*** (± 0.9)	0.02 (± 1.1)	0.000
WHtR 1	0.621 (± 0.1)	0.622 (± 0.1)	0.907	Impedance 1	634.4 (± 88.2)	621.6 (± 87.0)	0.489
WHtR 2	0.598 (± 0.1)	0.623 (± 0.1)	0.068	Impedance 2	614.9 (± 84.3)	629.6 (± 83.9)	0.409
Change	-0.02*** (± 0.0)	0.001***(± 0.0)	0.000	Change	-19.5*** (± 28.1)	7.9 (± 27.5)	0.000

***$p<0.001$ * $p<0.05$

Table 5.4 continued: Initial (1) & final (2) measurements and change in measurements in FML and FMG group

Variable	FML (n=58)	FMG (n=37)	*p*	Variable	FML (n=58)	FMG (n=37)	*p*
% BF 1	40.7 (+ 7.3)	39.5 (+ 5.4)	0.396	* % BF 1	44.4 (+ 6.9)	43.6 (+ 6.1)	0.534
% BF 2	37.6 (+ 6.9)	40.8 (+ 5.2)	0.011	* %BF 2	41.0 (+ 7.8)	45.2 (+ 5.5)	0.003
Change	-3.12*** (+ 2.1)	1.32*** (+ 1.6)	0.000	* Change	-3.40*** (+ 2.8)	1.60*** (+ 2.0)	0.000
z- %BF 1	2.56 (+ 0.6)	2.54 (+ 0.5)	0.847	* z-%BF 1	2.85 (+ 0.5)	2.86 (+ 0.5)	0.925
z- %BF 2	2.30 (+ 0.6)	2.64 (+ 0.5)	0.003	* z-%BF 2	2.59 (+ 0.7)	2.98 (+ 0.5)	0.001
Change	-0.25*** (+ 0.2)	0.10*** (+ 0.1)	0.000	* Change	-0.26*** (+ 0.3)	0.11*** (+ 0.1)	0.000
FM (kg) 1	31.1 (+ 14.7)	29.0 (+ 10.0)	0.409	* FM (kg) 1	33.6 (+ 14.4)	31.8 (+ 10.2)	0.478
FM (kg) 2	28.6 (+ 13.8)	30.8 (+ 10.2)	0.369	* FM (kg) 2	31.0 (+ 14.5)	33.9 (+ 10.3)	0.258
Change	-2.50*** (+ 1.9)	1.84 *** (+ 1.5)	0.000	* Change	-2.57*** (+ 2.1)	2.13*** (+ 1.6)	0.000
FFM (kg) 1	42.9 (+ 11.3)	43.4 (+ 9.1)	0.843	* FFM (kg) 1	40.4 (+ 11.2)	40.5 (+ 9.4)	0.955
FFM (kg) 2	44.9 (+ 11.6)	43.6 (+ 8.7)	0.560	* FFM (kg) 2	42.4 (+ 11.4)	40.5 (+ 9.0)	0.361
Change	1.91*** (+ 2.3)	0.28 (+ 2.0)	0.000	* Change	2.0*** (+ 2.1)	0.02 (+ 2.2)	0.000
FMI 1	12.9 (+ 5.0)	12.3 (+ 3.2)	0.477	* FMI 1	14 (+ 4.8)	13.5 (+ 3.2)	0.571
FMI 2	11.7 (+ 4.7)	12.9 (+ 3.3)	0.127	* FMI 2	12.7 (+ 4.9)	14.3 (+ 3.2)	0.063
Change	-1.25*** (+ 0.8)	0.61*** (+ 0.7)	0.000	* Change	-1.30*** (+ 0.7)	0.72*** (+ 0.7)	0.000
FFMI 1	18.0 (+ 2.4)	18.5 (+ 2.2)	0.300	* FFMI 1	16.7 (+ 2.6)	17.1 (+ 2.6)	0.476
FFMI 2	18.5 (+ 2.4)	18.4 (+ 2.1)	0.771	* FFMI 2	17.5 (+ 2.6)	17.1 (+ 2.4)	0.381
Change	0.50*** (+ 0.8)	-0.14 (+ 0.8)	0.000	* Change	0.90*** (+ 0.8)	0.0 (+ 0.9)	0.000

***$p<0.001$ ** $p<0.01$ * $p<0.05$ *%BF, *z-%BF,*FM, *FFM, *FMI (kg/m^2), *FFM (kg/m^2)I: derived using 3C equation

5.5 Discussion

Anthropometric measurements and body composition data were collected from ten cycles of a children's weight management programme. Changes in these measurements were initially examined on a whole group basis and subsequently on a gender basis. Changes in anthropometric measurements were then further examined by dividing participants into two groups – those that lost/maintained fat mass (FML group) and those that gained fat mass (FMG group).

This aim of this study was to examine changes in anthropometric and body compositional measures in a sample of obese children who had participated in a weight management intervention programme. In view of this, this study was not designed to be a randomized control trial and the data collection was based on the criteria of obtaining complete sets of measurements (initial and final) from an opportunistic sample. A control/non-intervention group was therefore not part of this study. Such a group is commonly included in studies where the aim is to assess the effectiveness of an intervention [387,396,424]. Furthermore, similar previous studies have also not included a non-intervention obese group [383,386,391,394,425,426].

A total of 95 participants were included in this study, as initial and final measurements had been obtained from them. Characteristics of participants who were enrolled on the programme but subsequently dropped out have not been described, as they were not followed up.

This was a 10-week programme consisting of physical activity/exercise intervention, nutritional education, psychological assessment and promoting positive behavioural changes. The instigators of the programme therefore based outcome measures on the components of the programme and so dietary, psychosocial and biological outcome measures were collected at the start and end of all the programmes. However, a description of what constituted positive or negative biological outcomes was not transparent.

The clinical guidelines in the UK provided by the National Institute for Health and Clinical Excellence (NICE) state that "the aim of weight management programmes for children and young people may be either weight maintenance or weight loss,

depending on their age and stage of growth" [427]. Furthermore, commonly used lay terms in clinical practice to describe aims for individuals participating in such interventions often include "grow into their height" or "grow into their weight". This demonstrates the lack of clarity associated around setting biologically based aims for interventions and participants and hence assessing the efficacy of such interventions.

The findings based on a whole group analysis firstly confirmed that obese children are generally taller for their age than non-obese children. This is a common observation [363,368,428] but has not been fully explained in the literature, and would suggest that tallness for age is a risk factor for obesity. Tallness for age would be linked with phases of growth, characterized by centile crossing in height growth charts [408]. The absolute gain in height over the 10-week period suggests a normal pattern of linear growth in the children given their age range. This was also reflected in the significant increase in FFM and FFMI at the end of the programme. However, given that the increase in z-height was not statistically significant, this would indicate that these children were not getting any taller for their age compared with the start of the programme.

Changes in anthropometric and body composition variables were initially explored on a whole group basis as well on as a gender basis. It was found that boys and girls demonstrated similar patterns of change, with differences apparent in the degree and significance of change in these variables. This suggests that both boys and girls responded similarly to the intervention, however it was apparent that participants had responded to the intervention in two distinct ways when they were divided into two subgroups (FML and FMG) based on change in fat mass. It was therefore deemed more relevant to focus on differences between these groups compared with a whole group basis despite the greater sample size and hence statistical power achieved from whole group analysis.

Examination of height and z-height in the FML and FMG groups showed that participants continued to grow in height without becoming taller for their age at the end of the programme. Although a slightly greater increase in absolute height (0.15cm, p=NS) and z-height (0.025, p=NS) was observed in the FML group, these findings suggest that the intervention did not have an impact upon linear growth regardless of whether participants lost or gained fat mass.

The findings on weight and z-weight indicated that those who lost/maintained fat (FML) also lost weight, reflected in a decrease in z-weight, whereas an increase in weight and z-weight was observed in those who gained fat mass (FMG). This is a logical finding since fat mass is a component of body mass and highlights clear differences between the groups, indicating that the intervention impacted upon change in weight.

Interestingly, despite having a larger change in absolute weight compared with the FML group, the FMG group had a smaller change in z-weight. This cannot be explained by a difference in the gender make up of the two groups as the proportion of boys and girls were similar in both the FML (boys=47%, girls=53%) and FMG (boys=46%, girls=54%) group. However, this could possibly be a result of participants in both groups being at different stages of maturity hence the growth-related change in weight would differ. It could be speculated that participants in the FMG group were more mature/advanced stages of growth and therefore greater changes in weight were required to result in a change in z-weight.

BMI, z-BMI and BMI centile all decreased in the FML group and increased in the FMG group. Based on z-scores, the decrease in BMI in the FML group was predominantly due to a decrease in z-weight (-0.11 units) as opposed to the increase in z-height (0.033 units). Similarly, in the FMG group, the increase in z-weight (0.05 units) exceeded the increase in z-height (0.008 units). The decrease in BMI in fat losers/maintainers and the increase in BMI in fat gainers demonstrates that BMI can be a marker of change in adiposity following a short-term intervention. However, as will be discussed later, the BMI-FM change is not as simple due to the confounding increase in FFM (kg).

No significant difference in FFM (kg) was observed between the two groups at the start of the programme although the FML group had a slightly lower mean FFM (by 0.5kg) compared with the FMG group. The increase in height in both groups reflected an increase in FFM as linear growth is comprised in part of lengthening of the skeleton and hence gain in most of mineral and protein [107]. Furthermore, the gain in height in the FML group corresponded to a gain in absolute FFM (kg) and FFMI (kg/m^2). This shows that despite losses in both weight and adiposity, growth in FFM was preserved in the FML group.

In the FMG group, a smaller increase in absolute FFM (kg) was observed compared with the FML group, which also corresponded with a smaller increase in height (cm). However, when partially corrected for height, FFMI either decreased by –0.14 kg/m^2 (manufacturer's equations) or remained unchanged (3C equations) in the FMG group. This finding is unlike that in the FML group, where an increase in height corresponded with an increase in FFMI (kg/m^2). The concept of decreasing FFMI (kg/m^2) is surprising, however it is not a finding that is unique to the participants of this study. This phenomenon of decreasing FFMI (kg/m^2) in children has also been described in another study in girls aged between 9 and 10 years and is a consequence of a greater increase in absolute height (cm) relative to the gain in FFM (kg) [429]. A physiological explanation proposed by this study was that there may have been lag in muscle growth relative to bone elongation and growth in stature during this period. Whether growth-related changes explain this finding in the FMG group remains unclear firstly because growth-related references on the components of FFM do not yet exist and secondly because no differences were apparent between the groups in their age and gender make up. The influence of environmental factors such as physical activity may offer an alternative explanation. A longitudinal study on boys followed from the age of 11 to 18 years found that changes in FFM were greater in active boys compared with moderately active and inactive boys even though differences in body composition between the boys at the start of the study were small [107]. Thus, environmental factors such as physical activity can have an added although currently unquantified, effect on growth related changes in FFM and its components. In relation to obesity treatment programmes, larger increases in FFM have been reported in subjects who participated in exercise based programmes (consisting of aerobic type exercises and high repetition resistance exercises) than in non-participating control subjects [416]. Thus, the difference in pattern of change in FFMI between the FML and FMG groups may potentially be reflecting differences in participant adherence to the BEST programme, although supporting data on physical activity participation is not available.

Whole body fatness based on %BF, FM and FMI was not significantly different between the FML and FMG groups at the start of the programme. However, these variables differed significantly at the end of the programme with decreases observed in all measures of body fatness in the FML group and increases observed in the FMG

group. Using all measures of body fatness, it was also observed that the loss of adiposity in the FML group was greater than the gain in adiposity in the FMG group.

It was observed that the direction of change in BMI mirrored the direction of change in adiposity in both the groups whereby an increase in BMI and FM was observed in the FMG group and a decrease in both variables was observed in the FML group. However, closer analysis of the components of BMI showed that the decrease in weight and BMI in the FML group was specifically characterized by a decrease in FMI and an increase in FFMI. In contrast, the increase in weight and BMI in the FMG group was solely (3C equation)/predominantly (manufacturer's equation) due to a gain in FMI. This shows that the direction and degree of change in BMI gives no indication on the unique contribution of FMI and FFMI to the change in BMI. Thus, individuals following an intervention can demonstrate similar changes in the direction and degree of change in BMI yet the direction and degree of the change in FMI and FFMI can vary. Furthermore, important changes that could be a consequence of either the intervention or growth may be masked if the evaluation of an intervention was based on BMI alone. An example of this is the maintenance/decrease in FFMI in the FMG group (described earlier), which was masked by an increase in FMI. Thus, a decrease or increase in all measures of BMI in children and adolescents does not necessarily reflect a decrease or increase in adiposity alone and so evaluation of interventions based on BMI does not always provide an accurate representation on its efficacy.

The use of BMI and its corresponding reference curves could also misrepresent changes in pubertal children participating in an intervention programme. As described earlier, puberty is a period of unique body compositional changes in males and females. It is established that the pubertal increase in BMI is predominantly due to an increase in FFM in males and in FM in females [429]. Hence assessing the outcome of an intervention based on BMI alone would mask these important qualitative changes and potentially support an inaccurate conclusion on the efficacy of an intervention. This matter is further complicated by the fact that obtaining information on pubertal status can be difficult in such settings due to ethical, practical and cultural reasons. This was also the case in this study where it was not possible to obtain pubertal status of the participants. This highlights the importance of obtaining a

complete assessment that includes body composition to accurately assess changes in pubertal children participating in an intervention.

The limitations associated with the use of BMI are important to consider in a practical sense because a number of weight management interventions are currently being commissioned by PCTs in an attempt to combat the rising prevalence of obesity in young people. However, most often, the "bottom line" judged by commissioners and Directors of Public Health is "how much has BMI decreased?" The findings from this study indicate the naivety in this approach. Weight change in growing obese children undergoing weight management interventions is a highly complex process which cannot be judged by BMI alone. Those commissioning weight management programmes would do well to try to gain an understanding of this phenomenon before judging a programme to be successful or a failure, most likely leading to withdrawal of funding.

Upper body fatness as assessed by WC and z-WC was not significantly different between the two groups at the start of the programme. However, patterns of change in these measures at the end of the programme mirrored those of whole body fatness whereby a decrease in WC and z-WC was observed in the FML group and an increase in both measures was observed in the FMG group with the degree of change significantly greater in the former. WHtR, also a measure of excess abdominal fat accumulation and indicator for the risk of obesity-related morbidity, is a measure of waist that is partly correctly for the effect of height [343]. A boundary value of WHtR ≥0.50 has been assigned to indicate increased risk of metabolic consequences of obesity in adults and children. A significant decrease in mean WHtR was observed in the FML group whereas an increase was observed in the FMG group. Furthermore, a greater proportion of children in the FML group (7.3%) had a WHtR that was below the boundary value (WHtR≥0.50) at the end of the programme compared to the FMG group (3.3%). All these measures of upper body fatness indicate that participants in the FML group were significantly less centrally obese at the end of the programme. Contrastingly, participants in the FMG group appear to have had small gains in upper body fatness (WC, z-WC p=NS; WHtR $p<0.001$) at the end of the programme. These observations are important to consider firstly with regards to the error associated with conducting WC measurements in obese children and supports the need for training in this measurement technique and to avoid

multiple measurers leading to even greater error [351]. Secondly, a reduction in upper body fatness has been associated with an improvement in cardiovascular risk factors in children (using waist-to-hip ratio) [430] and adults (using WC) [330].

The degree of change between initial and final anthropometric and body composition measurements was greatest for z-%BF and least for z-BMI. The change in z-WC was midway these two other measures. This hierarchy of change was apparent at a whole group level, in girls and in the FML and FMG groups. The lower degree of change in z-WC compared to z-%BF is a logical observation given than whole body fat loss would be greater than the decease in upper body fat as only a proportion of the whole body fat loss would be lost from the upper body. Nevertheless, it is important to state that whilst it appears that BIA should be employed in children's weight management, where this is not available, accurate WC measurement is an acceptable indicator of fat loss, providing that the measurement is taken by trained personnel, using standardized protocols, with knowledge of the within-observer error [351].

These findings on the hierarchy of change also suggest that BMI was not capturing the true change in adiposity and therefore had a lower ability in identifying change in body fatness compared to z-WC and z-%BF. Thus, in the FML group, the decrease in z-BMI appears to have attenuated the degree of loss of adiposity when compared to z-WC and z-%BF. A similar finding was described by Cole et al when investigating how appropriate BMI-related variables were in measuring change in adiposity [405]. Their findings, albeit in younger children, demonstrated that variability in z-BMI and BMI centile decreased in the upper part of the BMI distribution. Put simply, a given change in BMI would convert into a smaller change in z-BMI and BMI centile in an obese child compared to a normal weight child.

The impact of this attenuation is apparent in the FML group where the decrease in BMI reflects a decrease in adiposity however this decrease would be classed as being modest since participants still remained above the 99th centile for BMI at the end of the intervention. Participants therefore still remained in the obese category (based on UK 1990 definition of >98th centile for obesity) following the intervention. A similar finding has been reported by another study which found that participants remained obese after a 10-week multidisciplinary intervention despite significant loss

of FM [431]. Interpretation of this type of finding in a clinical setting would become a complex issue, as the BMI change reflects a combination of positive and negative outcomes that need to be accounted for. However, this becomes an especially important interpretation as service commissioning and funding for such intervention is likely to be based on targets of reducing levels of obesity. Use of z-BMI can therefore underestimate the extent of positive outcomes and consequently have implications on the evaluation of an intervention and have potentially detrimental implications on participant motivation.

This underestimation of change in body fatness by changes in BMI is heightened by the fact that changes in anthropometric and body compositional measures in both groups were very small and non-significant. It is difficult to quantify what level of change would be expected from such an intervention, as this has not been defined elsewhere or indicated in UK guidelines on weight management for children. This is in contrast to adult weight management where a decrease of 0.5-1.0kg per week in weight is considered acceptable [427]. Only two recommendations are stated in the literature and these define levels of change in z-BMI (0.5-0.6 units) that result in an improvement in metabolic risk factors and a significant decrease in adiposity [401,402,406]. Assessing the outcome of the BEST programme based on these recommendations would suggest that participants in this study have not had a significant loss of adiposity and improvement in metabolic risk factors. However, caution needs to be exercised before such an interpretation is made as the duration over which these changes are proposed (6 months - 1 year) exceeds that of the BEST programme. Based on the lack of appropriate guidelines, it is difficult to draw upon any conclusions on the outcome of the programme.

The above scenario is not likely to be unique to the BEST programme. The scarcity of guidelines is likely to be affecting other such interventions where health professionals and facilitators in the UK are unable to make sound and evidence-based judgements on the outcome of their interventions. The lack of clarity on expectations of change is either complicated by or a result of the diverse nature of interventions worldwide. Mapping exercises have demonstrated that interventions can vary in their duration, components, frequency of intervention focused sessions, target age and gender group and range of outcome measures [432]. As a simple demonstration of this, a range of interventions were extracted from literature searches

and their characteristics and outcome findings were summarized (Table 5.5). This exercise showed that interventions varied in duration from 3 weeks to 12 months with some including additional follow-up periods. These interventions also varied in outcomes measures, ranging from full body compositional and anthropometric measurements to simply focusing on BMI and BMI related variables.

Without the existence of specific guidelines it could be assumed that the degree of change found in the BEST programme may be appropriate for a short-term programme. It could also be assumed that larger and more significant changes would be expected in longer-term studies. However, a comparison of whole group changes between the BEST programme and interventions in table 5.5 based on duration alone does not completely agree with this notion. A number of studies with approximately similar durations to our study report larger changes in a majority of the outcomes measures compared to those seen in the BEST programme [433-436]. Similarly, studies with shorter durations also report larger changes compared to those seen in our study. One such study reported a decrease of 4.5% BF over a 3-week period whereas a decrease of 1.4% BF (whole group basis) was seen in our study [437]. Another study demonstrated larger changes in all anthropometric and body compositional measures over an 8-week period compared to the BEST programme [383]. Another shorter-term study based in the UK has also reported a relatively larger decrease in weight (6kg) and BMI (2.4 kg/m^2) [387]. This was a six-week residential weight loss camp based on physical activity, moderate dietary restriction and group based educational sessions. Studies with longer durations to that of the BEST programme had on the whole reported larger change in BMI, BMI related variables and body compositional measures [386,394,395,417,438-441].

The above highlights the variation in the degree of change in anthropometric and body compositional measures observed in the interventions studies. It also emphasises the difficulty of basing expectations of change on assumptions and generalizations. The interventions compared in this exercise varied in many factors and this can play an important role in determining the degree of change observed at the end of the intervention. For example, studies examining the effects of components (physical activity, nutritional education, parenting skills) have demonstrated that even the characteristics of an intervention can result in a different degree of change in

outcome measures [434,439]. To complicate this further, participant adherence is likely to add further variance to degrees of change observed.

This study has highlighted the fact that current understanding on the most appropriate way to assess children's weight management programmes is limited. It is also evident that the existing recommendations stated in the literature are not likely to cater for the diversity of such interventions. Furthermore, recommendations based on BMI alone are unable to indicate individual contributions of adiposity and FFM to changes in body weight. BMI would therefore mask the complex changes that occur in growing obese children and how these may have been influenced by the weight management intervention. This argument emphasises that using BMI alone may provide an ambiguous understanding on the outcome of interventions and progress of participants undergoing these interventions. It is for these simple but fundamental reasons that changes in body composition would be more suitable to assess such an intervention as it would provide a clearer understanding on growth and intervention related changes.

Body composition analysis is achieved through indirect techniques that operate on a compartmental view of the human body [286]. The most commonly used model in body composition research is the two compartment model which assumes that the body is made up of two compartments-fat mass and fat free mass [288]. Techniques such as air displacement plethysmography (ADP) and DXA (Dual X-ray Aborptiometry) are based on the 2C model and have previously been considered as reference methods of body composition analysis [288]. However, these techniques are not often suitable options for weight management interventions due to practical reasons such as cost, availability of equipment and type of setting. Weight management interventions such as the BEST programme are often based in community settings such as healthcare centres, sports centres where access to such equipment is limited.

BMI has therefore been more widely used as an assessment tool to assess change in weight management interventions due to low cost of equipment, lack of skilled personnel required to obtain weight and height readings, equipment is portable and commonly available in such settings. This can be seen in a recent mapping exercise of interventions in London where outcomes from majority of the interventions were

based on BMI [432]. This shows the popularity with which BMI is used and reflects the ease of use in such interventions. This study has shown that BMI is not reflecting true change in adiposity in these children as it is masked by the increase in FFM. Introducing an alternative assessment tool for assessing these outcomes requires a system that will show any changes in adiposity and FFM but also meet all the requirements of practicalities in such interventions.

Despite having a practical advantage, BMI, as shown in this study, is not able to reflect the underlying change behind weight loss, weight gain or even weight maintenance in growing children. Assessing change in body composition using the BIA system in this study offers a suitable and practical option that can be used as an alternative or adjunct to BMI. Furthermore, the practical advantages of the BIA system in particular its portability, quick and non-invasive method of measurement and relatively low cost compared to DXA and ADP also makes it a suitable option for use in various types of settings including the community setting where such interventions are commonly held.

Although BIA proves to be a suitable option in assessing change following an intervention, the limitations associated with its use also need to be acknowledged. It is recognised that BIA applies prediction equations to derive FFM from TBW and thereby deduce FM from body weight. Furthermore, these equations are known to be accurate only if the characteristics of the population in concern are similar to that of the reference population. The manufacturers in built equations in the BC-418 system although validated in children, are not specific to obese children. As with other studies, our findings also showed that equations validated in non-obese children tend to underestimate FM and overestimate FFM in obese children. A difference in the composition of FFM between obese and non-obese children has been shown to explain this overestimation of FFM in the former group. Firstly, the hydration of FFM is reported to be higher in obese children and this has been associated with an expansion of the extracellular water space [300]. The mineral mass of bone has also been reported to be greater in obese children, possibly as a consequence of earlier sexual maturation [442]. On balance and taken together, these changes result in an overall decrease in the proportion of protein (protein to mineral ratio) in the FFM and so density of the FFM is lower in obese children compared to non-obese children [443]. However, it is still important to note that despite the absolute differences in FM

and FFM between the in built equations (manufacturer's equation) and the validated 3C equations, the degree of change between the initial and final measurement was approximately similar for both sets of equations.

Another limitation associated with the use of the BC-418 system is that that the prediction equations (both manufacturers and 3C validated equations) have to date been validated only in Caucasian children. This is an important consideration as the children enrolled into the BEST programme were from a diverse ethnic population, (with children predominantly from a Bangladeshi background), largely due to the geographical location of the programme in London. As explained in Chapter 6, the BIA %BF results are unlikely to be truly accurate in non-Caucasian children. Furthermore, an analysis of the ethnic composition of the FML and FMG group showed that the proportion of South Asian (67.6% vs. 62%) and African-Caribbean (10.8% vs. 6.9%) children was higher in the FMG than in the FML group. It was also shown that the proportion of Caucasian (22% vs. 18.9%) and Mixed Race (3.4% vs. 2.7%) children was slightly higher in the FML than in the FMG group. Given the slightly different ethnic composition of the two groups, it remains a possibility that fat loss and fat gain were related to ethnicity. Indeed it could be that the BEST programme is more culturally specific to the Caucasian children enrolled onto the programme, an issue that could be considered in future planning, commissioning and delivery of such programmes.

Although the above limitations regarding the use of the BC-418 system have to be taken into consideration, it can be argued that in the case of a weight management intervention, it is more important to obtain a precise measure of body composition than a truly accurate measure. It is the change in adiposity that demonstrates a participant's response to the intervention and in turn describes the outcome of the intervention. The findings in this study have provided evidence for this argument in two ways. Firstly, the change in adiposity between the validated 3C equations and the manufacturers in built equations were approximately similar, thereby demonstrating that outcome for all participants would be interpreted in the same manner regardless of initial and final adiposity levels. Secondly, impedance which is a measure of resistance to the electric current was also the only raw measurement from the BIA system. Change in impedance between the initial and final measurements mirrored the decrease in adiposity in the FML group and the increase in adiposity in the FMG

group. This shows that it is not necessarily the accuracy of an analysis technique that is essential in evaluating change but the repeatability of the technique. Hence, even if the validity of the BIA system can be questioned in terms of ethnicity, it is apparent from the change in impedance that the equipment appears to be performing robustly and is adding value to the assessment of body weight changes. Thus it was felt appropriate to report %BF in the non-Caucasian children in this study even though the absolute %BF would not be truly accurate. Future work will validate these scales in other population groups.

Table 5.5: Findings from other weight management intervention studies

Author	Short description	Outcome(s)	Results
Schwingshandl, J. 1995 [437]	41 obese (19M,22F) Age: 8.5-14.8y (median 11.8y) 3 week programme (energy restriction + exercise)	Resistance Index (BIA), LBM (kg), (Shaefer eqn), %BF Weight for height	Change over 3 weeks Mean weight for height: 151% - 139% Mean %BF: 46.0% – 41.5% Weight change ranged from: -3.1 to - 8.2kg Change in %BF ranged from: -2.7 to -10.4kg Change in LBM(kg) ranged from: -2.6 to +2.3kg After 4 month follow up: Regain in weight inversely correlated with change in LBM during programme.
Schwingshandl, J. 1999 [434]	30 children Group A: 12 week dietary intervention + training programme 6b,8g,mean age:11.0y Group B: 12 week dietary intervention only. 7b,9g, mean age:12.2y	Resistance Index(BIA) FFM(kg) (Shaefer) BMI, z-BMI	Group A: Initial / 12 week; Group B: Initial / 12week Age(y): 11.0 / - / 12.2 / - Weight(kg): 63.3 / 62.6 / 69.2 / 68.7 z-BMI: 5.58 / 5.06 / 5.33 / 4.82 FFM(kg): 34.3 / 37.3 / 37.1 / 37.5
Barbeau, P. 1999 [433]	N=71(22 M,49 F) 4 months Physical training	%BF FM FFM (DXA)	Height (cm) :143.5-146.3 (+2.7) Weight (kg) :59.2-61.0 (+1.8) Relative BMI: 1.05 – 1.06 %BF: 44.4 – 42.8 (-1.6) FM (kg): 27.0 – 26.8 (-0.19) Fat free soft tissue: 30.7 -32.6 (+1.9)
Gutin, B. 2002 [417]	N=80, 13-16 y, 8 months	BMI %BF, FM, FFM (DXA) VAT, SAAT (MRI) (surface area x width- multislice)	Lifestyle education alone: %BF: +0.19; FM(kg): +1.62; Fat free soft tissue (kg): +1.69; VAT: -11.0 cm3; SAAT: + 40.4cm3 Lifestyle education + physical: %BF: -3.57; FM(kg): -0.73; Fat free soft tissue(kg):+1.80; VAT: -42.0 cm3; SAAT: -69.7cm3

Author	Short description	Outcome(s)	Results
Dao, H.H. 2004 [394]	55 (33 M,22 F), age 9-17 y, mean of 9 months (range 6-12 months). MDT weight reduction programme in therapeutic unit (moderate dietary restriction-based on RDA of low PA, regular PA training) Weekdays boarding in unit, weekends voluntary stay.	Height Weight WC Hips circ FM, FFM (DXA) Trunk FM, LM:	Girls: Age (y) :13.9-14.5 Height (m): 1.61-1.62 Weight: 100.5-75.5 BMI:38.4-28.4 WC m:1.04-0.83 HC m:1.23-1.03 FM:42.8-23.9 LM: 49.7-49.1 Trunk FM:16.1-7.9 Trunk LM:24.6-24.7 Boys: Age (y): 13.6-14.2 Height m: 1.67-1.7 Weight:95.6-73.9 BMI:34.5-25.5 WC m:0.99-0.82 HC m:1.16-0.97 FM:38.2-17.7 LM: 53.9-54.6 Trunk FM:15.7-5.6 Trunk LM:26.7-26.6
Gately, P.J. 2005 [387]	185 overweight, mean age:13.5y, 6 week camp	BMI, z-BMI, %BF, FM(kg), FFM(kg) (ADP)	Height: 162-163* BMI:33.5-31.2* %BF:47-44* FFM:46.2-45.7* HC: 114.5-108.7* Weight:89.6-83.6* z-BMI:3.03-2.74* FM:42.7-37.1* WC:96.5-90.3*
Nemet, D. 2005 [435]	24 obese (dietary, behavioral, physical activity intervention) Age: 11.3y 22 obese control 3month intervention, 12 month f/up.Age:10.9y	BMI, %BF (skinfolds) At 3 months (short term effects) At 6months (long term effects)	At 3 months: Intervention: Weight (kg): -2.8kg; BMI (kg/m^2): -1.7; % BF: -3.3 Control: Weight (kg): 1.2; BMI (kg/m^2): -0.2; % BF: 1.4 After 1 year f/up Control Group: Before / 12months Intervention group: Before / 12months Height (m): 1.476 / 1.520 / 1.445 / 1.490 Weight (kg): 63.4 / 68.6 / 59.1 / 59.7 BMI (kg/m2): 28.0 / 28.6 / 27.7 / 26.1 BMI centile: 97.2 / 96.1 / 98.2 / 92.3 %BF: 40.9 / 44.4 / 40.6 / 38.3

Author	Short description	Outcome(s)	Results
Savoye 2005 [438]	Intervention group N=25 (8M, 17F)	z-BMI, % Body Fat (Tanita, TBF 300 (BIA)	Mean age:13.5y (Range:11-16years) Baseline Height: 164.1cm, Weight: 109.6kg BMI(kg/m2): Baseline 40.1, 6 months 37.7, 12 months 39.3 z-BMI: 2.49, 2.30, 2.29 %BF: 45.76, 40.79, 42.44
Rudolf, M. 2006 [386]	N=94 (49 F,45 M), mean age=12.2y, Community based intervention	BMI at 3 and 6 months	At 3 months change in z-BMI= -0.01 (NS) (54% of subjects had a reduction in z-BMI) At 6 months, change in z-BMI= -0.07 ($p<0.01$) (greater for girls=-0.07,$p=0.02$ and >13yr olds -0.13,$p<0.01$). 71% of subjects had a reduction in z-BMI)
Golley, R.K. 2007 [439]	2 intervention groups: 1) Parents skills (P) 2) Parents skills and intensive lifestyle education (P+DA) 111 pre-pubertal children (70g, 41b) 6 and 12 months	z-BMI at 6 and 12 months	Mean age at start: 8.2 years (Range: 6-9 years) z-BMI: Baseline / 6 months / 12 months P +DA: 2.74 / 2.52 / 2.43 P group: 2.76 / 2.63 / 2.56 Control: 2.75 / - / 2.60 z-WC P +DA: 3.27 / 3.00 / 2.85 P group: 3.20 / 3.08 / 2.93 Control: 3.14 / - / 3.14 z-height (all intervention groups): 1.2 / 1.3

Author	Short description	Outcome(s)	Results
Lazaar,N. 2007 [395]	425 (213 M and 212 F), age 6-10 y, 6 month 2weekly sessions)	BMI, z-BMI, WC, sum of skinfolds (Σ SS), FFM	In intervention girls: Weight: 32.0-32.7 ns Height m: 1.26-1.27 BMI: 20.1-19.9 ns z-BMI: 2.94-2.75*** WC:67-64.9*** FFM:22.5-23.8 Σ SS(mm):55.8-51.9* In intervention boys: Weight: 35.5-35.9 NS Height m: 1.29-1.31*** BMI: 21.0-20.9ns z-BMI: 3.15-3.07*** WC: 70.1-69.7 ns FFM: 25.2-26.8*** Σ SS(mm):53.5-51.7*
Hughes, A.R. 2008 [440]	Intervention group N=69 ((30M, 39F) 6 and 12 months Mean age	z-BMI at 6 and 12 months	Mean age: 9.1 yrs Results presented as median Change in 0-6months Weight (kg): 3.2 z-BMI: -0.10 z-height: -0.02 z-WC: -0.20 Change in 0-12 months Weight (kg): 7.0kg z-BMI: -0.07 z-height: 0.01 z-WC: -0.20 Actual results (median) Baseline / 6 months / 12months Weight (kg) 52.6 / 54.8 / 58.1 z-BMI 3.2 / 3.0 / 2.8
Knopfli, B.H. 2008 [383]	130 (52 M,78 F), age:13.8y (12.1-15.0y), 8 week MDT inpatient programme,	BMI, %BF (DXA)	Girls: Height (cm): 162 Weight (kg):90.3-76.7 BMI:33.3-29.0 %BF:48.4-44.1 FM (kg):39.1-31.9 FFM (kg):46.4-43.6 Boys: Height (cm): 163 Weight (kg): 89.2-76.3 BMI:33.6-28.1 %BF:45.5-40.5 FM (kg):40.5-30.2 FFM (kg):47.4-45.4

Author	Short description	Outcome(s)	Results
O'Connor, J. 2008 [441]	N= 20 (5M, 17F) Age:13-16years 5 months	BMI, z-BMI WC, z-WC	Results presented at median Start – End Age (yrs): 14.3 – 14.8 Weight (kg): 90.6 – 90.6 (NS) z-weight: 3.12 - 3.10 (NS) Height (cm): 162.2 – 163.8 z-height: 0.23 – 0.23 (NS) BMI (kg/m^2) 33.0 – 32.5 (NS) z- BMI: 2.30 – 2.24 (NS) WC (cm): 100.1 – 97.1 z-WC: 1.40 – 1.28
Wong, P.C.H. 2008 [436]	12 week intervention Exercise group (EG): PE + additional sessions (N=12 b) Control Group (CG): PE only (N=12b)	BMI, %BF & FM (kg), LBM (kg) using DXA	EG: Pre / Post; CG: Pre / Post Age (y): 13.8 - 14.3 - Height (cm): 164.9 165.7 165.6 166.9 Weight (kg): 83.1 80.7 87.6 88.9 BMI: 30.6 29.4* 31.8 31.7 %BF: 35.6 34.8 37.8 36.3 FM(kg) 29.3 29.2 31.6 30.8 LBM (kg): 51.3 52.9* 50.1 51.7

M=Males; F=Females; Resistance Index = Height2/Imepdance; eqn = equation; b= boys; g=girls; DXA=Dual X-ay Absorptiometry; VAT= Visceral adipose tissue; SAAT= Subcutaneous abdominal adipose tissue; LM=Lean Mass; MDT=Multidisciplinary team; circ=circumference; HC=Hip circumference; f/up=follow up; ns=non-significant; LBM=Lean body mass; PE=Physical education;

***p<0.001; **p<0.01, *p<0.05; NS p=non significant

Chapter 6: Ethnicity-related variation in general and regional body composition

6.1 Introduction

Any study that plans to examine variations in body fatness between children from different ethnic groups, or to employ Bioelectrical Impedance Analysis (BIA) technology in this context, must appreciate the influence of whole body composition and body proportions and dimensions on these measures. Ignoring these issues can lead to erroneous measures and conclusions. The following is a review of the current understanding on the variation in body composition in children in relation to race and ethnicity.

Although variations in human biology as a function of race and ethnicity have been reported widely, there remains a debate as to whether individuals and groups are categorized precisely using these classification systems. Ethnicity classifies individuals on the basis of a social construct which can include criteria such as culture, heritage and national origin [444]. Race is commonly used to define groups of people using a biological construct and classification is often based on phenotypic characteristics such as facial features, hair colour and skin colour. The debate around use of race and ethnicity as classification criteria has been prompted by evidence from modern genetic techniques which has discredited the view that race is biologically determined by showing greater genetic differences within local populations (84% of genetic variation) compared to classically defined racial groups (10%) and inter-nation differences (6%) [445].

Given that these terms are used interchangeably in the body composition literature and for the sake of convenience, ethnicity and race have been used as a single definition in this review with the focus placed on studies examining body compositional variations between South Asians, Sub-Saharan Africans, Caribbean and Caucasians.

Ethnic variations in body composition have been examined at various levels and compartments including whole body density, composition and density of Fat Free Mass (FFM), body fat distribution as well as stature. This review includes current

knowledge on body compositional variations in relation to ethnicity in children and adolescents.

6.1.1 Density of the Fat Free Mass

The density of any substance is derived from the ratio of its mass to its volume

Equation 6.1: Density (g/ml) = mass (g) / volume (ml)

On the basis of a two-compartment model of body composition, whole body density is the sum of the densities of fat mass (FM) and fat-free mass (FFM). Based on evidence from several cadaver analyses and fat biopsies, the densities of fat mass and fat-free masses were proposed to be constant at 0.900g/ml and 1.100g/ml respectively. [446,447]. Further to this, Brozek et al (1963) also proposed a density value of 1.100 g/cm^3 for FFM from their data on the Reference Man [291].

There has been much speculation regarding the constancy of the density of the FFM relative to age, sex and ethnicity. This is particularly so because the FFM is a heterogeneous compartment and its density is determined by the proportions of its constituents and their relative densities. The primary constituents (and densities) of the FFM are water (0.9937 g/ml at 36^{o}C), protein (1.34 g/ml), bone mineral (2.982 g/ml) and soft tissue mineral (3.317 g/ml) [292].

Variation in the density of the FFM across growth and development has been reported in a number of studies, with strong evidence for an increase in density during growth and conflicting evidence on its decrease during senescence. [289,448]. The increase in density during growth is thought to be a consequence of a decrease in hydration level (82% at birth) and an increase in the protein and mineral fractions (14% and 3% respectively at birth) of the chemically immature FFM. These changes represent the increasing density of the FFM from birth (1.064 g/cm^3) to adulthood (1.100 g/cm^3) [449]. At a cellular level, Wang et al (1997) have described these changes as an increase in ratio of extracellular solids (ECS) to total body water (TBW) (0.07 at birth to 0.135 in adolescence) and a decrease in ratio of extracellular water (ECW) to intracellular water (ICW) (1.7 at birth and 1.0 in adults) [292].

Variations in the density of the FFM with respect to ethnicity have not been systematically investigated in children and so evidence from adult studies is discussed here. Schutte et al (1984) reported a greater FFM density in black (1.113

g/cm^3) compared to white men (1.100g/cm^3) aged 18 to 32 years [450]. They speculated that the greater density in black men was a consequence of a greater bone mineral content and nonosseous mineral and /or protein fraction of the FFM. Further to this, a later study reported a greater mineral fraction of FFM in black men (7.1%) and women (7.7%) compared to white men (6.6%) and women (7.0%) but observed no difference in density of the FFM between the ethnic groups [448]. Another study found that the density of FFM was greater in black men (20 to 59 years) and women (over 60 years) than in white men and women respectively as a consequence of greater bone mineral/TBW [292]. However, no significant difference was found in the measured density of the FFM between black and white subjects and the authors concluded that differences between ethnic groups may be too small to be detected by in vivo measurements.

Difference in the density of the FFM between Singaporean Indians, Malays and Chinese aged 18 to 75 years has also been investigated [451]. The density of the FFM in females from all three groups (Indian =1.1070 kg/l, Chinese = 1.1082 kg/l, Malay = 1.1038kg/l) was significantly higher than the assumed constant of 1.100 kg/l. Indian men had a significantly higher density (1.1052 kg/l) relative to the constant whereas this was lower in Chinese (1.0987 kg/l) and Malay (1.1011 kg/l) men.

Thus differences in the density of the FFM between ethnic groups are apparent in adults and are a function of variations in the FFM fractions of water, protein and minerals. The assumption of a constant density may therefore not apply to all ethnic groups and could lead to bias in estimates of body composition from densitometry. In the case of a higher density than the assumed constant, %BF would be underestimated [451].

6.1.2 Total Body Water

Hydration of the FFM is defined as the ratio of total body water (TBW) to FFM (TBW/FFM). The relative constancy of TBW in FFM was first proposed by Pace and Rathburn (1945) and a hydration factor of 0.73 was reported based on chemical analysis on several mammalian species [452]. Further chemical analyses on human cadavers provided support for this constant [453].

Although widely accepted as a constant in humans, its applicability with regards to age and ethnicity has also been questioned. Reference data on children have shown that TBW/FFM is greatest at birth (approximately 0.81) and decreases throughout growth and maturation [289]. A higher TBW/FFM in prepubertal and pubertal compared to adulthood was reported based on data from boys aged 12 to 18 years and girls aged 9 to 17 years [454,455]. Furthermore, an average change in TBW/FFM of 2.9% (74.9% to 72.2%) and 2.8% (75.7% to 73.0%) in males and females respectively was reported from pre-pubescence to adulthood [456]. A theoretical understanding of this constant at the cellular level describes the age-related change in the hydration of the FFM to result from an increase in ECS (extracellular solids)/TBW ratio and a decrease in extracellular fluid to cell mass ratio [453].

Variation in the hydration of the FFM between ethnic groups has been examined both in adults and children. Using deuterium oxide dilution (^2H_2O) and tritium oxide dilution (^3H_2O), no difference in TBW and TBW/FFM was found between age, height, weight and menstrual status matched black and white females. [457,458]. Similarly, no difference in TBW/FFM was reported between black and white women aged 20-70 years [459]. This study also reported a similar rate of age-related decline in TBW in both ethnic groups (-3.6%, white and -3.8%, black). In contrast, a greater hydration factor (expressed as a percentage of the FFM) has been observed in American black men (74.4%) and women (75.1%) compared to white men (73.9%) and women (73.7%) aged 20 to 94 years [448], suggesting inconclusive evidence of variation in the hydration of the FFM.

Variation in the hydration of the FFM between Singaporean Chinese, Indian and Malays aged 18 to 75 years has also been examined. This study found no significant difference between Chinese (0.725), Malay (0.737) and Indian women (0.727). However the hydration factor in men was higher in Chinese (0.738) and Malays (0.735) than in Indians (0.721). It is evident from this study that the hydration of the FFM in Chinese women and Indian men and women is lower than the assumed constant of 0.73 [451].

Investigations on ethnic differences in the hydration of the FFM in children and adolescents have been limited to studies in black and white children. Studies in children from Asian backgrounds have not been cited in the literature suggesting

these population groups have yet to be examined. Ethnic differences between black and white adolescents in TBW were shown not to exist relative to weight and height [450,460]. In contrast, TBW expressed as a percentage of total body weight was greater in black than in white subjects aged between 8 and 30 years [456]. This pattern was consistent at all stages of maturation and in both genders. The greater TBW in the black group was thought to be a consequence of the greater lean mass which is reflected in their higher body density. However, TBW as a proportion of FFM was did not vary between the black (72.4-76.3%) and white (71.9-76.0%) children, adolescents and adults, mirroring the findings in adult studies.

These findings highlight that the hydration of the FFM is not constant in children and adolescents and decreases (0.4-0.6% per year) with increasing age and maturation [454-456]. Furthermore, no significant difference in TBW/FFM has been found between black and white subjects (children and adults) in all but one study. Small variations were reported between Asian adults with some groups showing a deviation from the assumed constant. Such deviations from the TBW/FFM constant are important to consider when determining body composition using hydrometry and densitometry. The assumption of a constant hydration in these techniques means that hydration levels above the constant will result in an overestimation of %BF using hydrometry and an underestimation of %BF using densitometry [451].

6.1.3 Mineral

The skeleton is the mineral reservoir of the body and thus variations between ethnic groups are assessed by examining differences in skeletal dimensions and composition. Research in both adults and children has largely been driven by the ethnicity-related variations in the prevalence of osteoporosis and the importance of peak bone mass in determining susceptibility to fractures in later life.

Measures of bone are commonly expressed as bone mineral density (BMD), bone mineral content (BMC), bone area (BA) and bone length on a regional or whole body basis. The majority of studies have examined ethnic differences in bone dimensions using non-invasive methods such as DXA with a smaller number based on cadaveric analyses. Studies that examined foetal [461] and adult cadavers have reported a greater skeletal weight of the whole body and individual bones in black compared with

white men and women [462,463]. Additionally, the bones of black cadavers (men and women) have been reported to be denser than those of white cadavers [464].

Total body bone mineral content (TBBMC) was found to be greater in black than in non-black prepubertal children [465] and in black females compared to age, height and weight adjusted white and Hispanic females [466]. BMC of regional skeletal sites and of the axial and appendicular skeleton have been reported to be higher in black compared to age matched white children [467-469].

Greater whole body BMD in black compared to non-black children has been reported as early as infancy [470,471] and shown to remain in pre-pubertal children [472,473]. Furthermore, black males and females aged 9-25 years have shown to have greater whole body areal BMD and estimated volumetric bone density than whites, Asians and Hispanics of similar ages [474]. Histomorphometric findings have suggested that the denser cancellous bone in the vertebrae in black compared to white adults is due to greater trabecular thickness [475]. Gilsanz et al (1998) found that the influence of ethnicity was unique in the axial and appendicular skeletons of black and white children. In both groups of children, ethnicity influenced the apparent density of cancellous bone in the axial skeleton and the cross-sectional areas of the femur in the appendicular skeleton [476].

Although the greater bone density in black compared to Asian children is better established, variations in measures of bone between Asians and white groups are less consistent. Examination of variations in BMD between Asian and whites aged 9-25 years showed that on the whole bone mass was comparable between the two ethnic groups throughout the age range [477]. In contrast, a later study observed lower regional and whole body BMD and BMC adjusted for whole body bone size in Asian compared with white males and females [474]. However, the differences between these two groups were not always significant and were smaller in magnitude than the difference between black and non-black children. A number of studies have reported lower bone mineral density in Asian adults and children compared with white counterparts however, these differences were thought to be an artefact of smaller bone and body size in Asians. For example, Patel et al (1993) found that a greater bone width (BW), BMC and BMC/BW in white compared to Indian boys disappeared after adjusting for height and weight [478]. This finding has also been demonstrated

in adults where the lower BMD in South Asian Immigrant women compared to Caucasian women disappeared after adjusting for bone size [479,480] and weight and height [481].

The understanding of the aetiology of ethnic differences in bone dimensions remains rudimentary. Both genetic and environmental influences are thought to explain the variation in bone density between ethnic groups. Heritability estimates from twin studies and parent-offspring correlations suggest a significant role of genetic factors in influencing BMC and density [482]. Recent analysis on multi generational African-Caribbean families showed that the heritability of trabecular bone was greater than that of cortical bone [483]. A widely recognized theory is that the genetically greater muscle mass in black individuals results in greater BMC and density due to the added stress on the bone [484].

Although a large part of the variations in BMC and density between ethnic groups can be explained by differences in bone and body size [485], other possible reasons have also been proposed to explain these differences. The greater accumulation of bone in black subjects has been reported widely despite lower dietary intake of calcium and Vitamin D. The lower levels of calcium and Vitamin D are partly related to the reduced consumption of milk, milk products and cereals [486]. However, lower serum levels of 25(OH)D found in black subjects are mainly related to the lower production of vitamin D in the skin due to increased pigmentation. The adaptive repose to the lower serum calcium and 25(OH)D levels is relative secondary hyperparathyroidism which in turn increases bone turnover and decreases urinary calcium excretion [486,487]. This adaptive response has shown to be unique in black adults and children in that higher renal sensitivity was maintained and thus urinary calcium excretion was lower than in white counterparts. However, the expected greater bone turnover levels in black adults and children have not been observed suggesting resistance to the bone resorbing effects of parathyroid hormone. Despite increases in $1,25(OH_2)D$, findings of greater calcium absorption in black subjects remains inconsistent. On the whole, these findings demonstrate an efficient adaptive response to the lower serum calcium and 25(OH)D in black adults and children that allows calcium homeostasis to be maintained without having detrimental effects on bone mineral density [486].

Some studies have also suggested that hormonal differences may explain the differences in BMC between black and white groups as they have a role in bone growth during puberty [488,489]. However, Hui et al (2003) did not find differences in sex hormone levels between black and white children but concluded that effects of sex hormones would be expressed through differences in maturation and body size [485].

A similar depth of investigation to explain differences in BMC between South Asian and white subjects seen in some studies does not appear to have been carried out. However, findings from a study investigating differences between UK Gujarati and white populations found no difference in serum calcium levels between the two groups but a lower serum Vitamin D levels in the former group [490]. Further studies need to be carried out to confirm whether differences between Asian and white populations do exist and if so, the underlying reasons need to be elucidated.

Despite an overall agreement that black children have a denser skeleton than white and Asian children, there are still some inconsistencies in the literature as this finding has not been observed in all studies. These studies have shown no difference in regional BMD between black and white children at varying pubertal stages [472,491]. Such conflicting findings are likely to have arisen from inconsistencies in adjusting for variations in bone and body size between ethnic groups. For example, variations in trunk and limb length can influence measures of bone density obtained using DXA. These inconsistencies are to be expected as there is currently no consensus on the most appropriate method of adjusting for size. This issue of inconsistencies is further complicated by the fact the DXA does not provide a "true" measure of bone mineral density as it gives a two dimensional representation of a three dimensional structure [492].

6.1.4 Protein

Variations in whole body protein between ethnic groups have been observed using whole body ^{40}K counting as an indirect measure of fat free mass and subsequently skeletal muscle mass. More recent studies have determined this body compartment using DXA.

Using DXA-derived body compartment values, a greater lean tissue mass was reported in African American compared to American European males [493] and females [466] aged 3 to18 years. Greater skeletal muscle mass in both arms and legs were observed in African-American males and females compared with non-Hispanic Caucasians, after adjusting for the length of both extremities [494]. Furthermore, greater appendicular skeletal muscle was observed in Caucasian and African-American compared to Asian pre-pubertal children [495].

Using ^{40}K counting, greater and denser muscle mass was reported in black compared with white boys [496]. A similar finding using ^{40}K counting was reported in males aged 7 to 79 years [497] and in black males and females aged 30 to 80 years [498]. Furthermore, greater TBK was reported in Black American males aged 5-18 years compared to their American White and Mexican American counterparts with the greatest difference evident during early adolescence (11-13 years) [484]. In contrast, lower total body potassium (TBK) values have been reported in Asian (Chinese and Korean descent) compared with African-American, white and Hispanic men and women [499].

Explanations for the above differences between ethnic groups are unclear. Adult studies examining the role of gonadal steroids and growth hormone were not able to explain differences in lean mass between African-American and Caucasian women. However, the higher serum testosterone levels observed in African-American women is thought to provide some explanation for the ethnic musculoskeletal differences [500]. Another mechanism proposed is the possible role of foetal growth, as indicated by birth weight, in programming fat free mass in later life [501].

6.1.5 Body Fat Patterning

"Fat patterning refers to the relative distribution of subcutaneous fat on the body as opposed to absolute amounts of fat" [484]. Body fat patterning has commonly been referred to as "gynoid" and "android" patterns. Assessment of fat patterning is based on determining fat at single sites or as central: peripheral ratios. Variations in fat patterning between ethnic groups have been observed in adults and in children at various stages of sexual maturation.

Variation in fat patterning between black and white children has been reviewed by Wagner and Heyward [484] and they have included evidence for greater subcutaneous fat in the truncal region and less subcutaneous fat in the extremities in black compared to white children and adults. Evidence for this was demonstrated in boys aged 6-16 years [502], infants and children [503] and men and women [504]. These findings were supported by the persistence of similar ethnic differences in fat patterning in athletes (1976 Montreal Olympics) and it was thus argued that sports and training affected total body fat, whereas fat patterning was affected by inherent biological factors [505]. Furthermore, multiple site skinfold measurements have demonstrated that whites carry more subcutaneous truncal fat anteriorly whereas black carry more subcutaneous truncal fat posteriorly and posteriolaterally [203,503,505].

The adult patterns of android and gynoid fat distribution in males and females respectively have been demonstrated in South Asian adolescents from the ages of 14 years [190]. Using DXA, greater truncal fat was also shown in the South Asian group compared to white European adolescents. Similarly, greater DXA trunk: peripheral fat ratio was found in Asian (South East Asian and South Asian) compared with white adolescent girls despite similarities in total body fat in both groups [506]. These studies concluded that Asian adolescents tend to store a greater proportion of fat in the trunk region [190,506]. Ethnic differences in prepubertal children have been reported using skinfold and DXA derived extremity and trunk fat [109]. They reported greater truncal fat in Asian (Chinese and Korean) girls and lower DXA derived extremity fat in Asian boys compared to their Caucasian counterparts. Evidence for thicker truncal skinfolds and thinner extremity skinfolds in the Asian Indian group compared to white counterparts has been reported in migrant men [191], Indian and Pakistani premenopausal women [200] and neonates [185]. Similar findings were also reported using magnetic resonance imaging [187].

The evidence would suggest that Black and Asians have similar body fat patterning. The clinical significance of this in terms of risk factors and obesity related morbidity requires further investigation. However, it has been argued that the adverse metabolic effects of subcutaneous adipose tissue may quantitatively match or exceed those of intraabdominal fat [203].

6.1.6 Body proportions, body dimensions and limb length

Body proportions describe the relative contribution of the upper and lower segments of the human body to stature. The segments can be quantified using anthropometric measures such as crown to rump lengths in foetuses, trunk length, sitting height, or subischial leg length (stature – sitting height). The sitting height represents the length of the head, neck and trunk and is often expressed as an absolute figure or relative to total height [(sitting height /stature x 100)] commonly known as sitting height ratio [507,508]. Body proportions are also defined on the basis of regional bone lengths (femur and vertebral) as they represent the length of the appendicular and axial skeleton.

Human growth follows a cephalo-caudal pattern whereby post natal growth in legs occurs at a greater rate than other post-cranial body segments [508]. The sitting height ratio is therefore greatest during infancy after which it declines during childhood and into adolescence. It then reaches a nadir at the beginning of the adolescent growth spurt as legs experience their growth spurt before that of the trunk. The spurt in trunk growth occurs during late adolescence at which point sitting height ratio increases [107].

Differences in upper and lower segments have been reported between black and white children of varying ages. Using data from the first (1971-1973) and second (1976-1980) NHANES, sitting height ratio was reported to be lower and leg length greater in black compared to non Hispanic white and Mexican American children aged 1-17 years [509]. A similar pattern of lower sitting height and greater leg length in African and African-American children compared to those of European ancestry (aged 1-18 years) despite being of similar height has also been reported [510]. The findings obtained using anthropometric measurements have been supported by findings on bone length. Cadaver analyses have reported longer bones in the upper (arms) and lower (legs) extremities in black compared to white adults [484]. Furthermore, greater femoral length and lower vertebral length in black compared to white subjects (aged 8-18 years) matched for height have been found [476].

Differences in body proportion between Asian and white children have also been reported. Comparative studies between Indian and British children have demonstrated that the difference in leg length between the groups varied by

socioeconomic status [507]. Rural Indian boys were found to have lower leg lengths than their affluent Indian counterparts and British boys. This difference was reduced between middle class Bengali boys and reversed in affluent Indian boys who had higher leg lengths than British boys. The findings on sitting height did not follow this pattern, as this was lower in the affluent and rural Indian and Bengali boys compared to British boys throughout adolescence. The authors reported final differences in both leg length and sitting height between the rural Indian and British boys of 3cm and 8.4cm respectively [507]. Furthermore, sitting height was observed to be lower in native wealthy north-western Indian children compared to British children [511]. Additionally, a greater subischial leg length (stature – sitting height) was found in northwest Indian girls until 12 years and boys until 14 years after which no difference was observed between the Indian and British children.

A further study compared body proportions of Pakistani children in the UK aged 5 to14 years against the Tanner and Whitehouse reference [512]. They found that sitting height was below the mean and leg length was above the mean relative to the British reference. However, sitting height in the oldest group fell further below the mean and leg length was similar to the mean of the reference population. Krishnaveni et al (2005) demonstrated in their studies on Mysore children that lower sitting height in Indian children is apparent even at 4 years of age [189]. In contrast, trunk length was reported to be similar in Asian (Japanese, Korean, Chinese, Filipino, Indian, Thai and Vietnamese) and white adolescent girls aged 9-14 years whereas leg length was shorter in the Asian girls [506].

The underlying explanations behind the ethnic differences in body proportions and limb length are not clear but a number of hypotheses have been proposed. It has been suggested that growth of the axial skeleton (represented by sitting height) is largely under the influence of sex hormones whereas growth in the appendicular skeleton is under the influence of growth hormones. Hence it has been suggested that variations in regional sensitivity to the hormones could be a possible explanation. However, Gilsanz et al (1998) found no differences in serum levels of growth hormones, growth factors and sex steroids between black and white children and therefore did not explain the difference in bone size between the two groups [476].

Explanations for the ethnic differences in body proportions could also be obtained from longitudinal studies examining environmental factors that influence growth of leg and trunk length. These studies have looked at the influence of both the pre-natal as well as post-natal environment on both components of height [513,514]. Evidence of the influence of pre-natal conditions on leg and trunk length was examined in the longitudinal data from the Boyd Orr cohort, the Caerphilly Cohort, the Midspan Family Study and longitudinal data from Aberdeen boys and girls [514]. These studies demonstrated that associations between birthweight and trunk length; and birthweight and leg-length were not significantly different. Furthermore, correlations with anthropometric markers of nutrition in-utero did not differ between trunk length and leg length. These findings indicate that pre-natal exposures do not appear to affect leg and trunk length differentially.

The importance of the post-natal environment has been shown in the 1946 National Birth cohort [515]. This study found that factors affecting leg length were different to those affecting trunk length. It was also shown that leg length was associated with markers of childhood nutrition such as breastfeeding and energy intake and socioeconomic factors such as crowding. In contrast, trunk length was associated with factors such as severe illness and chronic emotional disturbance. A similar association between childhood diet and socioeconomic conditions (living conditions) and leg length was also found in the Carnegie (Boyd Orr) survey for diet and health in pre-war Britain [513].

These longitudinal studies have provided evidence for differential influences on the components of height. Firstly, influences of early postnatal growth affect leg length primarily whereas longer-term effects of early growth influence trunk length. Leg length is therefore considered to be a sensitive marker of early condition in childhood such as diet, exposure to infectious diseases and socioeconomic conditions. The importance of these factors on leg length is further evident from the secular increase in the height of the Japanese population, who prior to 1960s were considered to be short in stature and a short-legged "race". An improvement in conditions following the end of the Second World War resulted in an average increase of 10cm in height between 1960 and 1977 that was almost exclusively from an increase in leg length [507].

The importance of considering differences in body proportions between ethnic groups in this thesis is for two specific reasons. Firstly, as discussed earlier, leg length is a sensitive marker of childhood nutrition and exposures and therefore ethnic differences in body proportions could indicate differences in nutritional and environmental exposures at some stage of early growth. Evidence for this can be observed from the larger difference in leg length between rural and affluent Indian children compared to affluent/immigrant Indian and British children [507]. These findings demonstrate that ethnic differences in leg length are not static and subject to environmental conditions. The shorter leg length in the rural Indian children is therefore likely to be indicating poor nutrition and socioeconomic conditions.

Ethnic differences in body proportions are also important to consider when using Bioelectrical Impedance Analysis (BIA). The theoretical basis of this technology is centred on the assumption that the human body is an ionic cylinder of homogenous conductive material and uniform length and cross sectional area [288,298]. This technology also assumes that the impedance to the flow of an alternative current is proportional to the height and inversely proportional to the cross-sectional area of the conductor. These assumptions are applied by BIA technology to obtain an estimate of the conducting volume (litres), represented by TBW (or FFM), of the human body. The relationship between impedance and the conductive volume is represented by the equation below which indicates that TBW (or FFM) is directly related to the square length of the conductor and indirectly related to impedance (a function of resistance and reactance) [288,298].

Equation 6.2: $V=pL^2/Z$.
V = volume p=specific resistivity L^2= length / height squared Z = impedance

However, the application of these assumptions and hence equation may not be entirely true given that the human body has a complex geometric shape made up of five cylinders (two legs, two arms and trunk) of varying lengths and cross sectional areas [298]. Furthermore, the arms and legs contribute 47% and 50% to whole body resistance despite accounting for 4% and 17% of body weight respectively. In contrast, the trunk contributes between 5% and 12% of whole body resistance despite accounting for 50% of body weight [358]. This indicates that whole body resistance is composed largely of segmental impedance in the extremities (arms and legs).

The contribution of the various segments of the body to whole body resistance is important to consider given the variation in body proportions between ethnic groups described earlier. The longer legs and shorter trunks in black/African American will result in greater resistance and therefore underestimation of FFM using generalized equations. This has been shown in children where age-, weight- and Resistance Index (RI) –matched black girls had more FFM (1.6kg) than Hispanic, non-Hispanic and mixed race girls aged 10 to 15 years old [516]. Furthermore, in adults, whole body impedance was found to be greater in Nigerians adults than in matched Caucasians. However, no difference in impedance was observed among different tribes of Nigeria [358]. Furthermore, greater leg length in Singaporean Indians compared to Malays and Chinese aged 18 to 69 years was associated with greater impedance values in the former group [517].

In addition to disproportionate contribution of body segments to whole body resistance, variations in specific resisitivity of body segments have also been reported. Specific resistivity is a constant that defines the resistance per unit length of a homogenous conductor. It is a constant for all ages and both genders and in theory, independent of body geometry [288,298]. However, the constancy of specific resistivity has not been maintained in studies looking at differences between body segments and between children and adults [518]. Furthermore, variation between ethnic groups has also been demonstrated with greater resisitivity of the lower limbs reported in Black compared to white women [519]. The greater resisitvity in Black women would result in a greater impedance value and thus a lower estimate of FFM.

The evidence therefore suggests that whole body resistance is determined largely by the extremities and thus ethnic variations in the length of limbs and their composition is likely to have an impact on whole body resistance and influence estimates of body composition using generalised equations and constants.

6.2. Aims and Objectives

6.2.1 Aims

- Characterize height, weight and anthropometric measures of total and central body fatness in relation to age, gender and ethnicity.

- Characterize measures of body composition derived from Bioelectrical Impedance Analysis (BIA) in relation to age, gender and ethnicity.
- Examine ethnicity-related differences in prevalence of whole body (BMI) and central (WC) overweight and obesity in a sample of schoolchildren aged between 5 and 16 years.

6.2.2 Objectives

- To collect anthropometric measurements including height, weight and WC and BIA derived variables in children aged between 5 and 16 years from Caucasian, South Asian (SA), African-Caribbean (AC) and Mixed Race (MR) backgrounds.
- To convert direct and derived measurements to age and gender specific z-scores using relevant references.
- To determine a BIA-derived indirect indicator of the body's conducting volume.
- To determine the prevalence of general overweight and obesity using Body Mass Index (BMI) and central overweight and obesity using waist circumference (WC) in the whole sample and compare prevalence across the ethnic groups.
- To compare means of age-adjusted variables between the ethnic groups in boys and girls separately.
- To compare means of age-and-gender specific variables between ethnic groups in boys and girls separately.
- To compare all variables between boys and girls within all ethnic groups.

6.3 Methodology

Measurements were collected from school children aged between 5 and 16 years residing in London (as described in Chapter 3). All children were given an ethnic code using a condensed version of the DCSF ethnicity classification system (Appendix B&C) [531]. Children from Caucasian, South Asian (Indian, Bangladeshi and Pakistani), African-Caribbean (African and Caribbean) and Mixed Race backgrounds were included in the study. Height (cm), weight (kg) and WC (cm) measurements were obtained using the procedures described in Chapter 3. BMI (kg/m^2) and WHtR were derived using these measurements and calculated using equations below. Height, weight and BMI were converted to z-scores using the UK 1990 references

[55]. %BF and WC was converted to z-scores using the UK %BF [308] and WC [342] references respectively. Whole body (BMI) and central overweight and obesity (WC) were defined using the 91st and 98th centiles respectively.

Data on body composition including Fat Mass (FM) (kg), % Body Fat (%BF), Fat Free Mass (FFM) (kg) was collected using BIA. The Fat Mass Index (FMI) (kg/m^2) and Fat Free Mass Index (FFMI) (kg/m^2) were derived using FM (kg) and FFM (kg) and calculated using the equations below. Raw impedance data (Ω) was also collected using BIA and used to calculate the Impedance Index (equation below), an indirect measure of the body's conducting volume.

Procedures for collecting anthropometric measurements and BIA-derived data are discussed in Chapter 3.

6.4 Statistical analysis

The variables below were calculated using the following equations:

Equation 6.3: BMI was calculated using the following equation: $\text{BMI (kg/m}^2\text{)} = \frac{\text{Weight (kg)}}{\text{Height (m)}^2}$

Equation 6.4: WHtR was calculated using the following equation = $\frac{\text{Waist circumference (cm)}}{\text{Height (cm)}}$

Equation 6.5: $\text{Fat Mass Index (FMI) (kg/m}^2\text{)} = \frac{\text{Fat Mass (FM) (kg)}}{\text{Height (m)}^2}$

Equation 6.6: $\text{Fat Free Mass Index (FFMI) (kg/m}^2\text{)} = \frac{\text{Fat Free Mass (FFM) (kg)}}{\text{Height (m)}^2}$

Equation 6.7: The Impedance Index (Ht^2/Impedance) (cm^2/Ω) was calculated using the following equation =

$$\frac{\text{Height}^2\text{ (cm)}}{\text{Impedance } (\Omega)}$$

Height (cm), weight (kg), BMI (kg/m^2), WC (cm) and % Body Fat were converted to z-scores using the LMS Growth software (Child Growth Foundation). Statistical analyses were performed using the Statistical Package for Social Scientists (SPSS 14.0 for windows, Release 14.0.0, 5 Sep 2005, Chicago, SPSS Inc). The percentage of children exceeding the 91st and 98th centile for BMI was determined for boys and girls from all the ethnic groups. The percentage of children exceeding the 91st and 98th centile for WC was also determined for boys and girls from all ethnic groups.

Means for all absolute variables and respective z-scores were determined for all ethnic groups on a whole group level as well as in boys and girls separately.

The statistical analysis for this chapter was conducted in four stages. The first stage of the analysis included a statistical comparison of all variables in boys and girls between the three South Asian subgroups (Indian, Pakistani and Bangladeshi) and between the African-Caribbean subgroups (Caribbean and African).

In the second stage of the analysis, data from these subgroups was pooled to form the "the collapsed ethnic groups" i.e. the South Asian and African-Caribbean groups. Comparisons from this stage onwards were conducted between the following ethnic groups: Caucasian, South Asian (SA), African-Caribbean (AC) and Mixed Race (MR). The second stage of analysis included a comparison of all variables between ethnic groups and in boys and girls separately. The third stage of the analysis included a comparison of all variables between boys and girls within each ethnic group.

In all three stages of analysis, comparisons on absolute variable (Table 6.1) were based on age-adjusted variables and tested using the one-way between-groups analysis of covariance (ANCOVA) test. Comparisons on z-scores (Table 6.2) were conducted using the one-way between-groups analysis of variance (ANOVA) test.

As part of ANCOVA test, the anthropometric and body compositional variables formed the dependent variables (Table 6.1) whereas ethnic group formed the independent variable and decimal age was used as the covariate in this analysis. Preliminary checks were conducted to ensure that the assumptions of homogeneity of regression slopes and homogeneity of variance had not been violated. Age-adjusted means and standard errors (SE) were obtained in boys and girls for all dependent

variables. Post hoc comparisons using the Bonferroni correction were conducted to examine differences between the ethnic groups. A p value of <0.05 was taken to indicate a statistically significant difference.

As part of the ANOVA tests, the z-scores (Table 6.2) formed the dependent variables and the independent variable was ethnic group. Means and standard deviations (SD) of all dependent variables were obtained in boys and girls separately. In the case where Levene's test for homogeneity of variance was violated, the Welch test of Equality of Means with $p<0.05$ was used to indicate statistical significance. Post hoc comparisons using the Hochberg's test and Games Howell test were conducted to examine differences between the ethnic groups as these tests are able to cope with unequal sample sizes and variances between groups.

The final stage of the analysis included a comparison of the prevalence of whole body overweight and obesity (BMI) and central overweight and obesity (WC) in boys and girls combined and separately

Table 6.1: Age-adjusted variables

Anthropometric variables	BIA derived variables
Height (cm)	% Body Fat (%BF)
Weight (kg)	Fat Mass (FM) (kg)
BMI (kg/m^2)	Fat Free Mass (FFM) (kg)
Waist circumference (WC) (cm)	Fat Mass Index (FMI) (kg/m^2)
Waist-to-height ratio (WHtR)	Fat Free Mass Index (FFMI) (kg/m^2)
	Whole body Impedance (WB Imp)(Ω)
	Height^2/Imepdance (Ht^2/Imp) ($cm^2/Ω$)

Table 6.2: Anthropometric and BIA-derived z-scores

Anthropometric z-scores	BIA derived z-scores
z-height	z-%BF
z-weight	
z-BMI	
z-WC	

6.5 Results

6.5.1 Stage 1 – comparison within the SA and AC groups

Data from a total of 4136 children (1945 boys, 2191 girls) from various ethnic groups were analyzed for this study. Of the whole sample, 1134 were Caucasian (598 boys, 536 girls), 1327 were SA (527 boys, 800 girls), 1376 were AC (667 boys, 709 girls) and 299 were MR (153 boys, 146 girls).

Table 6.3 presents means and SD for all absolute variables in boys and girls from the three SA groups- Indian, Pakistani and Bangladeshi. Tables 6.4 and 6.5 present age-adjusted means and SE for absolute anthropometric variables and BIA derived variables respectively for boys and girls in the three SA sub-groups. Results from the one way ANCOVA are also presented in these tables. Table 6.6 and 6.7 present means and SD for z-scores of anthropometric and BIA derived variables respectively. Results from the one way ANOVA are also presented in Tables 6.6 and 6.7.

Table 6.4 shows a significant difference in age between the sub-groups in both boys and girls. A post hoc comparison using Bonferroni correction indicated that Pakistani boys and girls were significantly older than their Indian and Bangladeshi counterparts with no difference in age apparent between the two latter groups. All other variables in Table 6.4 were greater in Pakistani boys and girls than in their Indian and Bangladeshi counterparts however a statistically significant difference between the SA sub-groups was only apparent in absolute height (boys only), WC and WHtR.

Height (cm) adjusted for age was significantly different between boys from the three sub-groups ($p<0.05$) (Table 6.4). A post hoc comparison indicated that Bangladeshi boys had a significantly lower mean height (cm) than Indian (131.3cm vs. 133.3cm, $p<0.01$) and Pakistani boys (131.3cm vs. 133.5cm, $p<0.05$). No other difference in absolute height was observed between boys from the SA sub-groups. A significant difference in WC (cm) was observed between the SA sub-groups in both boys and girls. A post hoc comparison indicated that Pakistani boys had a greater mean WC (cm) than Indian (62.5cm vs. 59.1cm, $p<0.01$) and Bangladeshi boys (62.5cm vs. 59.6cm, $p<0.05$). In girls, mean WC (cm) was significantly greater in Pakistani than in Indian girls (61.4cm vs. 59.1cm, $p<0.05$). No significant difference in WC (cm) was

observed between the Bangladeshi girls and girls from the other two sub-groups. WHtR was significantly different between the three sub-groups in boys ($p<0.01$) and girls ($p<0.05$). Post hoc comparisons showed that Pakistani boys and girls had a significantly greater mean WHtR than Indian boys (0.467 vs. 0.443, $p<0.01$) and girls (0.443 vs. 0.428, $p<0.05$) respectively. No significant difference was observed between Bangladeshi boys and girls and their respective counterparts from the other two sub-groups ($p>0.05$).

With the exception of whole body impedance (Ω), all BIA-derived variables were greater in Pakistani boys and girls than in their Indian and Bangladeshi counterparts (Table 6.5). However, a significant difference between the three sub-groups was observed only in whole body impedance (Ω). A post hoc comparison in boys indicated that Indian boys had a significantly greater mean whole body impedance (Ω) than Bangladeshi boys (785.5 Ω vs. 757.9, Ω $p<0.01$). No significant difference was observed between the Pakistani boys and boys from the other two sub-groups ($p>0.05$). A post hoc comparison in girls showed that whole body impedance (Ω) was greater in Indian girls than in Pakistani (824.6 Ω vs. 802.9 Ω, $p<0.05$) and Bangladeshi girls (824.6 Ω vs. 789.2 Ω, $p<0.001$). No difference in whole body impedance (Ω) was observed between Bangladeshi and Pakistani girls ($p>0.05$).

Mean z-scores for all variables (height, weight, BMI and WC) in Table 6.6 were greater in Pakistani boys and girls than in their Indian and Bangladeshi counterparts. With the exception of z-height (in girls) and z-%BF, these differences were statistically significant in all variables.

A post hoc comparison (Games Howell and Hochberg's tests) on variables in table 6.6 indicated that firstly z-height was significantly lower in Bangladeshi boys than in Indian (-0.24 vs. 0.05, $p<0.01$) and Pakistani (-0.24 vs.0.21, $p<0.01$) boys. Additionally, mean z-weight was significantly greater in Pakistani boys than in Bangladeshi (0.34 vs. -0.13, $p<0.05$) and Indian boys (0.34 vs. -0.17, $p<0.001$). No other difference in z-weight was observed between boys from the three SA sub-groups. In girls, z-weight was significantly lower in Indian than in Pakistani girls (-0.26 vs. 0.13, $p<0.05$). No other difference in z-weight was observed between girls from the three SA sub-groups.

Mean z-BMI was significantly lower in Indian boys (-0.32) and girls (-0.35) than in their Pakistani counterparts (0.26, 0.07, $p<0.01$). No significant difference was observed between Bangladeshi boys and girls and their respective counterparts from the other two SA sub-groups (Table 6.6).

Mean z-WC was significantly greater in Pakistani boys than in Indian (0.76 vs. 0.03, $p<0.001$) and Bangladeshi (0.76 vs. 0.29, $p<0.05$) boys. In girls, mean z-WC was significantly greater in Pakistani than in Indian girls (0.54 vs. -0.02, $p<0.001$). No significant difference in z-WC was observed between Bangladeshi girls and girls from the other two SA sub-groups (Table 6.6).

Table 6.7 presents the mean and SD for z-%BF in boys and girls from the three sub-groups. As evident in this table, mean z-%BF in boys was greatest in Pakistani children whereas in girls it was greatest in the Indian children. However, no statistically significant difference in mean z-%BF was observed between the three sub-groups in both genders.

Table 6.8 presents means and SD for all absolute variables in boys and girls from the African and Caribbean groups. Table 6.9 presents the age-adjusted mean and SE for absolute anthropometric variables for boys and girls in the two AC sub-groups. It can be seen that Caribbean boys were significantly older than African boys (9.52y vs. 8.76y, $p<0.001$) whereas no significant difference between the two sub-groups was observed in the girls. Although mean height (cm) was greater by 0.4cm in the Caribbean boys and by 0.6cm in African girls than their respective counterparts, these differences did not reach statistical significance. Additionally, mean weight (kg) was greater by 0.2kg and 0.3kg in Caribbean boys and girls respectively than in their African counterparts however, these differences also did not reach statistical significance as well. Mean BMI (kg/m^2) was similar in African and Caribbean boys and greater by $0.1kg/m^2$ in Caribbean girls although this difference was not statistically significant. Mean WC was greater by 0.2cm and 0.3cm in African boys and girls respectively however these differences were not statistically significant. Mean WHtR was similar in African and Caribbean girls and greater by 0.003 units in African boys although this difference was not significant.

Table 6.10 presents means and SE for all age-adjusted BIA-derived variables for boys and girls in the two AC sub-groups. Mean %BF was greater in African boys and girls than in their Caribbean counterparts by 0.9% and 0.3% respectively, however these differences were not statistically significant in both genders. Absolute fat mass (kg) and FMI (kg/m^2) were similar in girls from the two sub-groups. In boys, FM (kg) and FMI (kg/m^2) were greater by 0.2 kg and 0.1 kg/m^2 respectively in the African compared with the Caribbean group however, these differences were not statistically significant. Mean FFM (kg) and FFMI (kg/m^2) was greater in Caribbean than in African boys by 0.4kg and 0.1 kg/m^2 respectively and in girls by 0.1kg and 0.2kg/m^2 respectively however, these differences for both variables did not reach statistical significance.

A significant difference in BIA derived variables between the sub-groups was observed in whole body impedance (Ω) in both genders and in boys for Ht^2/imp. Whole body impedance was greater in African boys by 27.4 Ω and African girls by 13.8 Ω ($p<0.05$) than in their Caribbean counterparts. Mean Ht^2/Imp (cm^2/Ω) was greater in Caribbean boys and girls by 1.1 and 0.4 respectively, although a significant difference was observed only in the boys ($p<0.01$) (Table 6.10).

Table 6.11 presents the mean and SD for z-scores of anthropometric variables in boys and girls from the AC sub-groups. Mean z-height and z-weight were greater in the Caribbean boys and African girls than in their respective counterparts, however, the differences between the sub-groups were not statistically significant. No significant difference in z-BMI was observed between the sub-groups in boys and girls despite the mean being slightly higher in Caribbean than in African boys (0.41 vs. 0.37, $p>0.05$). Although mean z-WC was greater in African boys and girls than in their Caribbean counterparts, the differences in both genders were not statistically significant.

Table 6.12 presents the mean and SD of z-%BF in boys and girls from the African and Caribbean groups. Mean z-%BF was significantly greater in African boys than in Caribbean boys (0.80 vs. 0.48, $p<0.001$). Although a similar pattern was observed in girls (0.65 vs. 0.56, $p>0.05$), the difference between the two sub-groups was not statistically significantly.

6.5.2 Stage 2

Table 6.13 presents the mean and SD for absolute variables and their respective z-scores in the collapsed ethnic groups (i.e. South Asian and African-Caribbean) by gender. Table 6.14 presents the mean and SD for all BIA derived variables and their respective z-scores in boys and girls from the collapsed ethnic groups.

6.5.2.1 Comparison between ethnic groups- boys

Age-adjusted means and SE for absolute variables in boys from the collapsed ethnic groups are presented in Table 6.15. With the exception of age, a significant difference between the ethnic groups was apparent for all other variables.

Age-adjusted mean height (cm) was greatest in AC boys (137.2cm) and lowest in SA boys (133.1cm). A post hoc comparison indicated AC boys had a significantly greater mean height (cm) than Caucasian (135.0cm, $p<0.001$), SA ($p< 0.001$) and MR (135.6, $p<0.05$) boys. Additionally, SA boys had a significantly lower mean height (cm) than Caucasian ($p<0.001$) and MR boys ($p<0.001$). No significant difference in mean height (cm) was observed between the Caucasian and MR boys ($p>0.05$). Furthermore, ethnicity explained 6% of the variance in age-adjusted absolute height.

Age-adjusted mean weight (kg) was greatest in the AC boys (34.9kg) and lowest in the SA boys (31.6kg). A post hoc comparison showed that SA boys had a significantly lower mean weight (kg) than Caucasian (33.8kg, $p<0.001$) and AC boys ($p<0.001$). No other significant difference in weight (kg) was apparent between the ethnic groups.

Age-adjusted mean BMI (kg/m^2) was greatest in the Caucasian and AC boys (17.8 kg/m^2) and lowest in SA boys (17.0 kg/m^2). A post hoc comparison indicated that SA boys had a significantly lower BMI (kg/m^2) than Caucasian ($p<0.01$) and AC boys ($p<0.001$). No other significant difference in BMI (kg/m^2) was observed between the ethnic groups.

Age-adjusted mean WC (cm) was greatest in the Caucasian boys (61.9cm) and lowest in the SA (60.1cm) boys. A post hoc comparison showed that mean WC (cm) was significantly greater in Caucasian than in SA boys ($p<0.01$). No other difference in WC (cm) was observed between the ethnic groups.

Age-adjusted mean WHtR was greatest in the Caucasian boys (0.460) and lowest in the AC boys (0.447). A post hoc comparison indicated that mean WHtR was significantly greater in Caucasian boys than in SA (0.451, $p<0.05$) and AC ($p<0.001$) boys. No other difference in WHtR was observed between the ethnic groups.

Table 6.16 presents means and SE for age-adjusted BIA derived variables in boys from the four ethnic groups. A significant difference between the ethnic groups was observed for FFM (kg), FFMI (kg/m^2), whole body impedance (Ω) and Ht^2/Imp $p<0.001$).

Age-adjusted mean FFM (kg) was greatest in AC boys (27.3kg) and lowest in SA boys (24.7kg). A post hoc comparison indicated that FFM (kg) was significantly lower in SA boys than in Caucasian (26.5kg, $p<0.001$), AC ($p<0.001$) and MR (26.4kg, $p<0.001$) boys. Additionally, mean FFM (kg) was significantly greater in AC than in Caucasian boys ($p<0.05$)

Age-adjusted mean FFMI (kg/m^2) was greatest in Caucasian and AC boys (14.0 kg/m^2) and lowest in SA boys (13.4 kg/m^2). A post hoc comparison showed that mean FFMI (kg/m^2) was also significantly lower in SA boys than in Caucasian ($p<0.001$), AC ($p<0.001$) and MR (13.9 kg/m^2, $p<0.01$) boys. No other significant difference in FFMI (kg/m^2) was observed between the ethnic groups ($p>0.05$).

Age-adjusted mean whole body impedance (Ω) was greatest in SA boys (769.7 Ω) and lowest in Caucasian boys (714.3 Ω). A post hoc comparison showed that mean impedance (Ω) was significantly higher in SA boys than in Caucasian ($p<0.001$), AC (722.8 Ω, $p<0.001$) and MR (723.0 Ω, $p<0.001$) boys. No other significant difference in mean impedance was observed between the ethnic groups ($p>0.05$).

Age-adjusted mean Ht^2/Imp (cm^2/Ω) was greatest in AC boys (27.2 cm^2/Ω) and lowest in SA boys (24.1 cm^2/Ω). A post hoc comparison showed that SA boys had a significantly lower mean Ht^2/Imp than Caucasian (26.6 cm^2/Ω), $p<0.001$), AC ($p<0.001$) and MR (26.5 cm^2/Ω), $p<0.001$) boys. No other significant difference in mean Ht^2/Imp was observed between the ethnic groups ($p>0.05$). Of all the BIA derived variables, ethnicity explained the greatest variance in impedance and Ht^2/Imp (7.0%).

Table 6.17 presents means and SD for z-scores of all anthropometric variables in boys from the four ethnic groups. It is evident form this table that a significant difference between boys from the four ethnic groups was apparent for all variables ($p<0.001$).

Mean z-height was greatest in AC boys (0.67) and lowest in SA boys (-0.04). A post hoc comparison indicated that AC boys had a significantly higher mean z-height than Caucasian (0.28, $p<0.001$), SA ($p<0.001$) and MR (0.41, $p<0.05$) boys. Additionally, SA boys had a significantly lower mean than Caucasian ($p<0.001$) and MR ($p<0.001$) boys. No difference in z-height was observed between the Caucasian and MR boys ($p>0.05$).

Mean z-weight was greatest in AC boys (0.65) and lowest in SA boys (-0.07). A post hoc comparison showed that SA boys had a significantly lower mean z-weight than Caucasian (0.45, $p<0.001$), AC ($p<0.001$) and MR (0.46, $p<0.001$) boys. Additionally, AC boys had a higher mean z-weight than Caucasian boys ($p<0.05$). No other significant difference in z-weight was observed between the ethnic groups ($p>0.05$).

Mean z-BMI was greatest in Caucasian boys (0.40) and lowest in SA boys (-0.11). A post hoc comparison indicated that SA boys had a significantly lower mean z-BMI than Caucasian ($p<0.001$), AC (0.38, $p<0.001$) and MR (0.31, $p<0.01$) boys. No other difference in mean z-BMI was observed between the ethnic groups ($p>0.05$).

Mean z-WC was greatest in the Caucasian boys (0.73) and lowest in the SA boys (0.26). A post hoc comparison indicated that SA boys had a lower mean z-WC than Caucasian ($p<0.001$) and AC boys (0.61, $p<0.001$). No other difference in mean z-WC was observed between the ethnic groups ($p>0.05$).

Table 6.18 presents means and SD for z-%BF in boys from the four ethnic groups. As evident from this table, no significant difference in z-%BF was evident between the boys ($p>0.05$).

6.5.2.2 Comparison between ethnic groups- girls

Age-adjusted means and SE for absolute variables in girls from the collapsed ethnic groups are presented in Table 6.19. A significant difference between the ethnic groups was apparent for all age-adjusted absolute variables.

Mean age was greatest for the SA girls (10.07y) and lowest for the Caucasian girls (8.90y). A post hoc comparison indicated that SA girls were significantly older than girls from the other ethnic groups ($p<0.001$) and that Caucasians girls were younger than AC girls (8.90y vs. 9.33y, $p<0.05$).

Age-adjusted absolute height (cm) was greatest in the AC girls (139.6 cm) and lowest in the SA girls (134.9 cm). A post hoc comparison indicated that SA girls had a significantly lower mean height (cm) than Caucasian (136.2 cm, $p<0.01$), AC ($p<0.001$) and MR girls (137.0cm, $p<0.01$). Additionally AC girls had a significantly higher mean height (cm) than girls from the other ethnic groups ($p<0.001$).

Age-adjusted absolute weight (kg) was greatest in the AC girls (37.8kg) and lowest in the SA girls (32.6kg). A post hoc comparison showed that AC had a significantly higher mean weight (kg) than Caucasian (34.7kg, $p<0.001$), SA ($p<0.001$) and MR (34.4kg, $p<0.001$) girls. Additionally, SA girls had a significantly lower mean than Caucasian girls ($p<0.001$).

Age-adjusted absolute BMI (kg/m^2) was greatest in the AC girls ($18.7kg/m^2$) and lowest in the SA girls ($17.2kg/m^2$). A post hoc comparison indicated that mean BMI was significantly higher in AC girls than in Caucasian ($18.0kg/m^2$, $p<0.01$), SA ($p<0.001$) and MR girls ($17.6kg/m^2$, $p<0.01$). Additionally, Caucasian girls had a significantly higher mean than SA girls ($p<0.001$).

Age-adjusted WC (cm) was greatest in the AC girls (62.5cm) and lowest in the SA girls (58.7cm). A post hoc comparison showed that AC girls had a significantly higher mean than Caucasian (61.1 cm, $p<0.05$), SA ($p<0.001$) and MR (59.6cm, $p<0.001$) girls. Additionally, SA girls had a significantly lower mean WC (cm) than Caucasian girls ($p<0.001$).

Age-adjusted WHtR was greatest in the Caucasian girls (0.450) and lowest in the SA and MR girls (0.436). A post hoc comparison indicated that SA girls had a significantly lower mean WHtR than Caucasian ($p<0.001$) and AC girls (0.448, $p<0.001$). Additionally, MR girls also had a lower mean WHtR than Caucasian ($p<0.05$) and AC girls ($p<0.05$). No significant difference was observed between MR and SA girls ($p>0.05$) and between AC and Caucasian girls ($p>0.05$).

Table 6.20 presents means and SE for age-adjusted BIA derived variables in girls from the four ethnic groups. A significant difference between the ethnic groups was observed for all BIA derived variables ($p<0.001$). Age-adjusted mean %BF was greatest in the AC girls (24.9%) and lowest in the MR girls (23.2%). A post hoc comparison showed that AC girls had a significantly higher mean than Caucasian (23.8%, $p<0.01$), SA (23.7%, $p<0.001$) and MR ($p<0.01$) girls. No other difference in %BF was observed between the ethnic groups.

Age-adjusted FM (kg) was greatest in AC (10.0kg) girls and lowest in SA girls (8.3kg). A post hoc comparison showed that AC girls had a significantly higher mean FM (kg) than Caucasian (8.8kg, $p<0.001$), SA ($p<0.001$) and MR (8.5kg, $p<0.01$) girls. No other difference in FM (kg) was observed between the ethnic groups.

Age-adjusted FFM (kg) was greatest in the AC girls (27.8kg) and lowest in the SA girls (24.3kg). A post hoc comparison showed that AC girls had a significantly higher mean FFM (kg) than Caucasian (25.9kg, $p<0.001$), SA ($p<0.001$) and MR (25.9kg, $p<0.001$) girls. It was also observed that SA girls had a significantly lower mean FFM (kg) than Caucasian ($p<0.001$) and MR ($p<0.01$) girls. No difference in FFM was observed between Caucasian and MR girls ($p>0.05$).

Age-adjusted FMI (kg/m^2) was greatest in AC girls (4.9 kg/m^2) and lowest in SA and MR girls (4.3 kg/m^2). A post hoc comparison showed that AC girls had a significantly higher mean FMI (kg/m^2) than Caucasian (4.5kg/m^2, $p<0.01$), SA ($p<0.001$) and MR ($p<0.05$) girls. No other difference in FMI (kg/m^2) was observed between the ethnic groups.

As with absolute FFM (kg), age-adjusted FFMI (kg/m^2) was also greatest in AC girls (13.8 kg/m^2) and lowest in SA girls (12.9 kg/m^2). A post hoc comparison indicated that AC girls had a significantly higher mean FFMI (kg/m^2) than Caucasian (13.5kg/m^2, $p<0.01$), SA ($p<0.001$) and MR girls (13.3kg/m^2, $p<0.01$). It was also observed that SA girls had a significantly lower mean FFMI (kg/m^2) than Caucasian ($p<0.001$) and MR girls ($p<0.01$). No difference in FFMI (kg/m^2) was observed between Caucasian and MR girls ($p>0.05$).

Age-adjusted whole body impedance (Ω) was greatest in SA girls (813.0 Ω) and lowest in AC girls (755.5 Ω). A post hoc comparison showed that SA girls had a

significantly higher mean impedance (Ω) than Caucasian (764.1 Ω, $p<0.001$), AC ($p<0.001$) and MR (774.1, $p<0.001$) girls. No other difference in whole body impedance (Ω) was observed between the ethnic groups.

Age-adjusted Ht^2/Imp (cm^2/Ω) was greatest in AC girls (26.7 cm^2/Ω) and lowest in SA girls (23.4 cm^2/Ω). A post hoc comparison showed that AC girls had a significantly higher mean Ht^2/Imp than Caucasian (25.1 cm^2/Ω, $p<0.001$), SA ($p<0.001$) and MR (25.2 cm^2/Ω, $p<0.01$) girls. It was also observed that SA girls also had a significantly lower mean than Caucasian ($p<0.001$) and MR ($p<0.001$) girls. No difference in Ht^2/Imp was observed between Caucasian and MR girls ($p>0.05$).

Table 6.21 presents means and SD for z-scores of all anthropometric variables in girls. It is evident form this table that a significant difference between girls from the four ethnic groups was apparent for all variables ($p<0.001$). Mean z-height was greatest in AC girls (0.74) and lowest in SA girls (-0.07). A post hoc comparison indicated that AC girls had a significantly higher mean than Caucasian (0.15, $p<0.001$), SA ($p<0.001$) and MR girls (0.29, $p<0.001$). Additionally, SA girls had a significantly lower mean than Caucasian ($p<0.01$) and MR girls ($p<0.01$).

Mean z-weight was greatest in the AC girls (0.74) and lowest in the SA girls (-0.13). A post hoc comparison showed that AC girls had a significantly higher mean z-weight than Caucasian (0.23, $p<0.001$), SA ($p<0.001$) and MR (0.16, $p<0.001$) girls. Additionally SA girls had a significantly lower mean z-weight than Caucasian girls ($p<0.001$). No other difference in z-weight was observed between the ethnic groups.

Mean z-BMI was greatest in the AC girls (0.50) and lowest in the SA (-0.19) girls. A post hoc comparison showed that AC girls had a significantly higher mean z-BMI than Caucasian (0.19, $p<0.001$), SA ($p<0.001$) and MR (-0.01, $p<0.001$) girls. Additionally, SA girls had a significantly lower mean z-BMI than Caucasian girls ($p<0.001$). No other difference in z-BMI was observed between the ethnic groups.

Mean z-WC was greatest in the AC girls (0.93) and lowest in the SA (0.19) girls. A post hoc comparison indicated that AC girls had a significantly higher mean z-WC than Caucasian (0.65, $p<0.001$), SA ($p<0.001$) and MR (0.37, $p<0.001$) girls. Additionally, SA girls had a significantly lower mean than Caucasian girls ($p<0.001$). No other difference in z-WC was observed between the ethnic groups.

Table 6.22 presents mean and SD for z-%BF and shows that a significant difference was apparent between girls from the four ethnic groups ($p<0.001$). Mean z-%BF was greatest in the AC girls (0.62) and lowest in the MR girls (0.32). A post hoc comparison showed that AC girls had a significantly higher mean z-%BF than Caucasian (0.43, $p<0.05$), SA (0.34, $p<0.001$) and MR ($p<0.01$) girls. No other difference in z-%BF was observed between the ethnic groups.

6.5.3 Stage 3- A between gender and within ethnic group comparison of all variables

Table 6.23 presents means and SE for age-adjusted variables in boys and girls separately from all ethnic groups. A significant gender difference in age was observed between SA children with girls having a higher mean than boys (10.07 y vs. 8.98, $p<0.001$). Age-adjusted absolute height (cm) was greater by 1.1cm in Caucasian boys than in girls (134.5cm vs. 133.4cm, $p<0.01$). A gender difference in age-adjusted absolute weight (kg) was apparent in AC children with girls having a higher mean than boys (36.7kg vs. 35.4kg, $p<0.01$). Age-adjusted absolute BMI (kg/m^2) was significantly greater by 0.6 kg/m^2 in AC girls than in boys (18.5 kg/m^2 vs.17.9 kg/m^2, $p<0.01$). Age-adjusted WC (cm) was significantly greater in Caucasian (61.7cm vs. 60.1, $p<0.001$), SA (61.4cm vs. 58.9cm, $p<0.001$) and MR (61.1cm vs. 59.0cm, $p<0.05$) boys than in girls in the respective groups. Age-adjusted WHtR was also significantly greater in Caucasian (0.460 vs. 0.452, $p<0.01$), SA (0.450 vs. 0.435, $p<0.001$) and MR (0.449 vs. 0.437, $p<0.05$) boys than in girls in these groups.

Table 6.24 presents means and SE for age-adjusted BIA derived variables in boys and girls separately from all ethnic groups.

Age-adjusted mean %BF was significantly greater in girls than in boys from all ethnic groups. Mean %BF was greater by 3.3%, 3.0%, 4.1% and 2.9% in Caucasian, SA, AC and MR girls respectively. Age-adjusted mean FM (kg) and FMI (kg/m^2) was significantly greater in girls than in boys from Caucasian, SA and AC backgrounds. Mean FM (kg) was greater by 1.0kg, 1.0kg, and 1.9kg in Caucasian, SA and AC girls respectively. Mean FMI (kg/m^2) was greater by 0.5 kg/m^2, 0.5 kg/m^2 and 0.9 kg/m^2 in Caucasian, SA and AC girls respectively.

Age-adjusted mean FFM (kg) and FFMI (kg/m^2) were significantly greater in boys than in girls from all ethnic groups. Mean FFM (kg) was greater by 1.8kg, 1.4kg, 0.7kg and 1.7kg in Caucasian, SA, AC and MR boys respectively than in girls in these ethnic groups. Mean FFMI (kg/m^2) was greater by 0.6 kg/m^2, 0.6 kg/m^2, 0.3 kg/m^2 and 0.8 kg/m^2 in Caucasian, SA, AC and MR boys respectively than in girls in these groups.

Age-adjusted mean whole body impedance (Ω) was significantly greater in girls than in boys from all ethnic groups. Mean whole body impedance (Ω) was greater by 53.8 Ω, 50.6 Ω, 36.3 Ω and 55.9 Ω in Caucasian, SA, AC and MR girls. In contrast, age-adjusted mean Ht^2/Imp ($cm^2/Ω$) was significantly greater in boys than in girls from all ethnic groups. Mean Ht^2/Imp ($cm^2/Ω$) was greater by 6.0 $cm^2/Ω$, 1.8 $cm^2/Ω$, 1.5 $cm^2/Ω$ and 2.4 $cm^2/Ω$ in Caucasian, SA, AC and MR boys.

Table 6.25 presents means and SD for z-scores of all anthropometric variables in boys and girls from all ethnic groups. Similar patters were observed in the Caucasian, SA and MR children with boys having a greater mean z-height, z-weight, z-WC and z-BMI than girls. However, a significant gender difference was not always observed for all the ethnic groups. A significant gender difference in z-height was observed in the Caucasian group only (0.28 vs. 0.15, $p<0.05$). Mean z-weight was significantly different between genders in the Caucasian (0.45 vs. 0.23, $p<0.01$) and MR (0.46 vs. 0.16, $p<0.05$) groups. Mean z-BMI was also significant different between genders in the Caucasian (0.40 vs. 0.19, $p<0.01$) and MR (0.31 vs. -0.01, $p<0.05$) groups.

In contrast to the other ethnic groups, mean z-height, z-weight, z-BMI and z-WC were greater in AC girls than in boys although a statistically significant difference was observed in z-WC only (0.93 vs. 0.61, $p<0.001$)

Table 6.26 presents the mean and SD for %BF in boys and girls separately for all ethnic groups. Mean %BF was greater in boys than in girls in all ethnic groups although a statistically significant difference was observed in the Caucasian (0.59 vs. 0.43, $p<0.05$) and SA (0.63 vs. 0.34, $p<0.001$) groups only.

6.5.4 Stage 4

6.5.4.1 Prevalence of whole body overweight and obesity (BMI) (Table 6.27)

Prevalence of whole body overweight and obesity in the total sample was 11.4% and 10.8% respectively. Prevalence of overweight and obesity in boys from all ethnic groups was 11.2% and 12.4% respectively whereas in girls it was 11.6% and 9.4% respectively. Prevalence of whole body overweight and obesity combined (boys and girls combined) was greatest in the AC children (26.4%) and lowest in the SA children (18.6%).

Prevalence of whole body overweight for boys and girls combined was greatest in the AC children (12.6%) and lowest in the MR children (9.4%). This pattern remained when prevalence of overweight was examined in boys (12.0% vs. 9.8%) and girls separately (13.1% vs. 8.9%).

Prevalence of whole body obesity for boys and girls combined was greatest in the AC children (13.8%) and lowest in the SA children (7.5%). This pattern remained when prevalence of obesity was examined in boys (13.5% vs. 10.6%) and girls separately (14.1% vs. 5.4%).

6.5.4.2 Prevalence of central overweight and obesity (WC) (Table 6.28)

Prevalence of central overweight and obesity in the total sample was 12.8% and 14.0% respectively. Prevalence of central overweight and obesity in boys from all ethnic groups was 11.9% and 13.0% respectively whereas in girls it was 13.6% and 14.8% respectively. Prevalence of central overweight and obesity combined (boys and girls combined) was greatest in the AC children (31.1%) and lowest in the MR children (22.1%).

Prevalence of central overweight for boys and girls combined was greatest in the AC children (15.4%) and lowest in the MR children (10.7%). When examined by gender, prevalence of central overweight was greatest in Caucasian boys (13.2%) and AC girls (17.8%) and lowest in SA boys (9.7%) and Caucasian girls (11.2%).

Prevalence of central obesity for boys and girls combined was greatest in the AC children (15.6%) and lowest in the MR children (11.4%). When examined by gender,

prevalence of central obesity was greatest in the Caucasian boys (14.2%) and AC girls (18.9%) and lowest in AC boys (12.1%) and MR girls (10.3%).

In summary, these findings show that children across different ethnic groups vary in height, weight and in a range of anthropometric measures and body compositional measures of whole body and regional body fatness.

Table 6.3: Summary of absolute variables (mean ± SD) in Indian, Pakistani and Bangladeshi (South Asians) boys and girls

Variable		Indian (n=571)	Pakistani (n=252)	Bangladeshi (n=504)
Age (Years)	Boys	8.61 ± 2.8	10.21 ± 2.6	8.84 ± 2.2
	Girls	9.78 ± 2.9	11.58 ± 1.6	9.58 ± 2.3
Height (cm)	Boys	131.2 ± 18.7	140.8 ± 16.8	130.5 ± 13.3
	Girls	136.7 ± 18.1	147.5 ± 12.0	135.3 ± 14.8
Weight (kg)	Boys	30.0 ± 14.5	37.4 ± 13.7	29.8 ± 11.0
	Girls	33.5 ± 13.7	42.3 ± 13.0	32.8 ± 12.6
BMI (kg/m^2)	Boys	16.5 ± 3.4	18.2 ± 3.8	17.0 ± 3.6
	Girls	17.1 ± 3.7	19.0 ± 4.2	17.3 ± 3.7
WC (cm)	Boys	58.2 ± 10.4	65.4 ± 11.6	59.3 ± 9.7
	Girls	58.5 ± 9.7	64.4 ± 10.1	59.1 ± 9.2
WHtR	Boys	0.444 ± 0.05	0.465 ± 0.06	0.455 ± 0.06
	Girls	0.429 ± 0.05	0.437 ± 0.06	0.437 ± 0.05
%BF	Boys	20.1 ± 6.0	21.6 ± 6.6	20.2 ± 6.1
	Girls	23.9 ± 5.5	25.7 ± 6.6	23.4 ± 5.8
FM (kg)	Boys	6.6 ± 5.3	8.6 ± 5.4	6.5 ± 4.6
	Girls	8.5 ± 5.3	11.6 ± 6.4	8.2 ± 5.4
FFM (kg)	Boys	23.5 ± 10.0	28.8 ± 9.3	23.4 ± 7.3
	Girls	25.0 ± 8.9	30.7 ± 7.3	24.6 ± 7.7
FMI (kg/m^2)	Boys	3.5 ± 1.9	4.2 ± 2.2	3.6 ± 2.1
	Girls	4.3 ± 2.0	5.2 ± 2.6	4.2 ± 2.1
FFMI (kg/m^2)	Boys	13.0 ± 1.8	14.1 ± 1.8	13.4 ± 1.8
	Girls	12.8 ± 1.8	13.9 ± 1.9	13.1 ± 1.7
WB Impedance (Ω)	Boys	789.4 ± 93.8	749.8 ± 80.3	759.5 ± 84.4
	Girls	828.7 ± 101.1	781.6 ± 93.4	796.1 ± 79.4
Ht^2/Imp ($cm^2/Ω$)	Boys	23.0 ± 9.1	27.4 ± 8.2	23.2 ± 6.6
	Girls	23.9 ± 10.4	28.6 ± 6.2	23.7 ± 6.5

Table 6.4: Mean and SE of age-adjusted absolute variables in South Asian sub-groups

		Indian	Pakistani	Bangladeshi	F	Partial Eta Squared
N	Boys	228	91	208		
	Girls	343	161	296		
†Age (Years)	Boys	8.61± 2.8	10.21± 2.6	8.84 ± 2.2	π12.2*** (2,524)	0.05
	Girls	9.78 ± 2.9	11.58 ± 1.6	9.58 ± 2.3	π 69.2*** (2,797)	0.09
Height (cm)	Boys	133.3 ± 0.4	133.5 ± 0.7	131.3 ± 0.4	7.2** (2,523)	0.03
	Girls	138.3 ± 0.4	138.7 ± 0.5	138.2 ± 0.4	0.3 (2,796)	0.01
Weight (kg)	Boys	31.5 ± 0.6	32.6 ± 0.9	30.4 ± 0.6	2.3 (2,523)	0.01
	Girls	34.6 ± 0.5	36.4 ± 0.7	34.7 ± 0.5	2.2 (2,796)	0.01
BMI (kg/m^2)	Boys	16.7 ± 0.2	17.5 ± 0.3	17.1 ± 0.2	1.8 (2,523)	0.01
	Girls	17.3 ± 0.2	18.0 ± 0.3	17.6 ± 0.2	2.3 (2,796)	0.01
WC (cm)	Boys	59.1 ± 0.6	62.5 ± 0.9	59.6 ± 0.6	5.3** (2,521)	0.02
	Girls	59.1 ± 0.5	61.4 ± 0.7	60.1 ± 0.5	4.2* (2,794)	0.01
WHtR	Boys	0.443 ± 0.004	0.467 ± 0.006	0.454 ± 0.004	6.8** (2,521)	0.03
	Girls	0.428 ± 0.003	0.443 ± 0.004	0.435 ± 0.003	4.4* (2,794)	0.01

*p<0.05; **p<0.01; *** p<0.001 †ANOVA-not corrected for age (Eta Squared) π Welch test of equality of means

Table 6.5: Mean and SE of age-adjusted BIA derived variables in South Asian sub-groups

		Indian	Pakistani	Bangladeshi	F	Partial Eta Squared
%BF	Boys	20.2 ± 0.4	21.2 ± 0.7	20.2 ± 0.4	1.0 (2,523)	0.00
	Girls	24.1 ± 0.3	24.7 ± 0.5	23.7 ± 0.3	1.4 (2,796)	0.00
FM (kg)	Boys	6.9 ± 0.3	7.4 ± 0.5	6.6 ± 0.3	1.1 (2,523)	0.00
	Girls	8.9 ± 0.3	9.8 ± 0.4	8.8 ± 0.3	2.1 (2,796)	0.01
FFM (kg)	Boys	24.6 ± 0.3	25.1 ± 0.5	23.8 ± 0.3	3.3 (2,523)	0.01
	Girls	25.7 ± 0.3	26.6 ± 0.4	25.9 ± 0.3	1.9 (2,796)	0.01
FMI (kg/m^2)	Boys	3.6 ± 0.1	3.9 ± 0.2	3.6 ± 0.1	0.9 (2,523)	0.00
	Girls	4.4 ± 0.1	4.7 ± 0.2	4.4 ± 0.1	1.6 (2,796)	0.00
FFMI (kg/m^2)	Boys	13.2 ± 0.1	13.6 ± 0.2	13.4 ± 0.1	2.5 (2,523)	0.01
	Girls	13.0 ± 0.1	13.3 ± 0.1	13.2 ± 0.1	4.0 (2,796)	0.01
WB Impedance (Ω)	Boys	785.5 ± 5.6	763.2 ± 8.9	757.9 ± 5.8	6.3** (2,522)	0.02
	Girls	824.6 ± 4.6	802.9 ± 7.0	789.2 ± 5.0	14.0*** (2,796)	0.03
Ht^2/Imp (cm^2/Ω)_	Boys	23.9 ± 0.3	24.3 ± 0.5	23.5 ± 0.3	0.9 (2,522)	0.03
	Girls	24.6 ± 0.3	25.0 ± 0.5	24.9 ± 0.3	0.4 (2,796)	0.00

*p<0.05; **p<0.01; *** p<0.001

Table 6.6: Mean and SD of z-scores for anthropometric variables in South Asian sub-groups

		Indian	Pakistani	Bangladeshi	F	Eta Squared
z-height	Boys	0.05 ± 1.1	0.21 ± 0.9	-0.24 ± 1.0	7.7** (2,526)	0.03
	Girls	-0.07 ± 1.1	0.02 ± 1.1	-0.11 ± 1.0	0.8 (2,799)	0.00
z-weight	Boys	-0.17 ± 1.4	0.34 ± 1.3	-0.13 ± 1.4	4.7** (2,526)	0.02
	Girls	-0.26 ± 1.3	0.13 ± 1.4	-0.12 ± 1.3	4.6* (2,799)	0.01
z-BMI	Boys	-0.32 ± 1.5	0.26 ± 1.5	-0.03 ± 1.6	5.1** (2,526)	0.02
	Girls	-0.35 ± 1.4	0.07 ± 1.6	-0.14 ± 1.4	5.0** (2,799)	0.01
z-WC	Boys	0.03 ± 1.3	0.76 ± 1.4	0.29 ± 1.4	9.7*** (2,524)	0.04
	Girls	-0.02 ± 1.5	0.54 ± 1.6	0.25 ± 1.4	8.3*** (2,797)	0.02

*p<0.05; **p<0.01; *** p<0.001

Table 6.7: Mean and SD of z-%BF in South Asian sub-groups

		Indian	Pakistani	Bangladeshi	F	Eta Squared
z-%BF	Boys	0.66 ± 1.1	0.73 ± 1.1	0.55 ± 1.1	1.0 (2,519)	0.00
	Girls	0.42 ± 1.1	0.34 ± 1.2	0.25 ± 1.1	1.7 (2,787)	0.00

*p<0.05; **p<0.01; *** p<0.001

Table 6.8: Summary of absolute variables (mean ± SD) in Caribbean and African boys and girls

		Caribbean (n=553)	African (n=823)
Age (Years)	Boys	9.52 ± 2.5	8.76 ± 2.6
	Girls	9.39 ± 2.6	9.29 ± 2.5
Height (cm)	Boys	140.0 ± 16.4	135.2 ± 16.9
	Girls	138.8 ± 16.1	138.8 ± 15.7
Weight (kg)	Boys	36.8 ± 14.2	33.5 ± 13.6
	Girls	37.5 ± 14.5	36.9 ± 13.6
BMI (kg/m^2)	Boys	18.1 ± 3.8	17.6 ± 3.5
	Girls	18.7 ± 4.2	18.5 ± 3.8
WC (cm)	Boys	62.0 ± 9.7	60.6 ± 9.3
	Girls	62.1 ± 9.8	62.2 ± 9.1
WHtR	Boys	0.444 ± 0.05	0.449 ± 0.05
	Girls	0.449 ± 0.05	0.449 ± 0.05
%BF	Boys	20.2 ± 6.3	20.9 ± 6.0
	Girls	24.6 ± 6.2	24.9 ± 5.8
FM (kg)	Boys	7.9 ± 5.4	7.4 ± 5.2
	Girls	9.9 ± 6.0	9.7 ± 5.7
FFM (kg)	Boys	28.9 ± 10.0	26.1 ± 9.4
	Girls	27.6 ± 9.1	27.2 ± 8.5
FMI (kg/m^2)	Boys	3.9 ± 2.2	3.9 ± 2.0
	Girls	4.9 ± 2.4	4.8 ± 2.2
FFMI (kg/m^2)	Boys	14.3 ± 1.9	13.8 ± 1.8
	Girls	13.9 ± 2.0	13.7 ± 1.8
WB Impedance (Ω)	Boys	702.9 ± 81.3	737.1 ± 90.1
	Girls	748.0 ± 87.3	762.6 ± 91.9
$Ht^2/Imp(cm^2/Ω)$	Boys	29.0 ± 9.2	25.8 ± 8.4
	Girls	26.7 ± 7.8	26.1 ± 7.4

Table 6.9: Mean and SE of age-adjusted absolute variables in African-Caribbean sub-groups

		Caribbean	African	F	Eta Squared
N	Boys	280	387		
	Girls	273	436		
†Age (Years)	Boys	9.52 ± 2.5	8.76 ± 2.8	13.2*** (1,665)	†0.02
	Girls	9.39 ± 2.6	9.29 ± 2.5	0.3 (1,707)	†0.00
Height (cm)	Boys	137.5 ± 0.4	137.1 ± 0.3	0.8 (1,664)	0.00
	Girls	138.4 ± 0.4	139.0 ± 0.3	1.1 (1,706)	0.00
Weight (kg)	Boys	35.0 ± 0.5	34.8 ± 0.4	0.1 (1,664)	0.00
	Girls	37.3 ± 0.5	37.0 ± 0.4	0.1 (1,706)	0.00
BMI (kg/m^2)	Boys	17.8 ± 0.2	17.8 ± 0.2	0.0 (1,664)	0.00
	Girls	18.7 ± 0.2	18.6 ± 0.2	0.3 (1,706)	0.00
WC (cm)	Boys	61.1 ± 0.5	61.3 ± 0.4	0.1 (1,664)	0.00
	Girls	62.0 ± 0.5	62.3 ± 0.4	0.2 (1,705)	0.00
WHtR	Boys	0.445 ± 0.003	0.448 ± 0.002	0.7 (1,664)	0.01
	Girls	0.449 ± 0.003	0.449 ± 0.002	0.0 (1,705)	0.00

*p<0.05; **p<0.01; *** p<0.001 †ANOVA-not adjusted for age (Eta Squared)

Table 6.10: Mean and SE of age-adjusted BIA derived variables in African-Caribbean sub-groups

		Caribbean	African	F	Eta Squared
%BF	Boys	20.1 ± 0.4	21.0 ± 0.3	3.1 (1,664)	0.005
	Girls	24.6 ± 0.3	24.9 ± 0.3	0.6 (1,706)	0.001
Fat Mass (kg)	Boys	7.5 ± 0.3	7.7 ± 0.2	0.4 (1,664)	0.001
	Girls	9.8 ± 0.3	9.8 ± 0.2	0.0 (1,706)	0.000
FFM (kg)	Boys	27.5 ± 0.3	27.1 ± 0.3	1.3 (1,664)	0.002
	Girls	27.4 ± 0.3	27.3 ± 0.2	0.2 (1,706)	0.000
FMI (kg/m^2)	Boys	3.8 ± 0.1	3.9 ± 0.1	0.5 (1,664)	0.001
	Girls	4.8 ± 0.1	4.8 ± 0.1	0.0 (1,706)	0.000
FFMI (kg/m^2)	Boys	14.1 ± 0.1	13.9 ± 0.1	2.0 (1,664)	0.003
	Girls	13.9 ± 0.1	13.7 ± 0.1	1.0 (1,706)	0.001
WB Impedance (Ω)	Boys	706.9 ± 5.0	734.3 ± 4.2	17.3*** (1,664)	0.025
	Girls	748.5 ± 5.4	762.3 ± 4.2	4.1* (1,706)	0.006
Ht^2/Imp (cm^2/Ω)	Boys	27.8 ± 0.3	26.7 ± 0.3	8.4** (1,664)	0.013
	Girls	26.6 ± 0.3	26.2 ± 0.2	0.9 (1,706)	0.001

*p<0.05; **p<0.01; *** p<0.001

Table 6.11: Mean and SD of z-scores for anthropometric variables in African-Caribbean sub-groups

		Caribbean	African	F	Eta Squared
z-height	Boys	0.69 ± 1.1	0.65 ± 1.0	0.3 (1,665)	0.00
	Girls	0.68 ± 1.0	0.78 ± 1.1	1.6 (1,707)	0.00
z-weight	Boys	0.67 ± 1.2	0.64 ± 1.3	0.1 (1,665)	0.00
	Girls	0.72 ± 1.3	0.76 ± 1.2	0.2 (1,707)	0.00
z-BMI	Boys	0.41 ± 1.4	0.37 ± 1.4	0.1 (1,665)	0.00
	Girls	0.50 ± 1.4	0.50 ± 1.3	0.0 (1,707)	0.00
z-WC	Boys	0.59 ± 1.3	0.63 ± 1.1	0.2 (1,665)	0.00
	Girls	0.88 ± 1.2	0.96 ± 1.2	0.7 (1,706)	0.00

*p<0.05; **p<0.01; *** p<0.001

Table 6.12: Mean and SD of z-%BF in African-Caribbean sub-groups

		Caribbean	African	F	Eta Squared
z-%BF	Boys	0.48 ± 1.1	0.80 ± 1.0	14.7*** (1,654)	0.02
	Girls	0.56 ± .1	0.65 ± 1.0	1.2 (1,701)	0.00

*p<0.05; **p<0.01; *** p<0.001

Table 6.13: Characteristics of the ethnic groups by gender: absolute variables and z-scores

	Boys					Girls				
Variable	Caucasian	South Asian	African-Caribbean	Mixed Race	Total	Caucasian	South Asian	African-Caribbean	Mixed Race	Total
N	598	527	667	153	**1945**	536	800	709	146	**2191**
Age (years)	9.08 ± 2.7	8.98 ± 2.6	9.08 ± 2.7	9.39 ± 2.7	**9.07 ± 2.7**	8.90 ± 2.5	10.07 ± 2.6	9.33 ± 2.5	9.08 ± 2.7	**9.48 ± 2.6**
Height (cm)	135.0 ± 16.7	132.6 ± 16.8	137.2 ± 16.8	137.3 ± 16.7	**135.3 ± 16.9**	132.9 ± 15.7	138.4 ± 16.5	138.8 ± 15.8	134.7 ± 17.3	**137.0 ± 16.3**
z-height	0.28 ± 0.99	-0.04 ± 1.0	0.67 ± 1.0	0.41 ± 1.1	**0.34 ± 1.1**	0.15 ± 1.0	-0.07 ± 1.1	0.74 ± 1.1	0.29 ± 1.2	**0.27 ± 1.1**
Weight (kg)	33.8 ± 13.5	31.2 ± 13.4	34.9 ± 14.0	34.9 ± 13.9	**33.5 ± 13.7**	32.3 ± 12.8	35.0 ± 13.6	37.1 ± 13.9	32.8 ± 13.7	**34.9 ± 13.7**
z-weight	0.45 ± 1.3	-0.07 ± 1.4	0.65 ± 1.2	0.46 ± 1.3	**0.38 ± 1.3**	0.23 ± 1.2	-0.13 ± 1.3	0.74 ± 1.2	0.16 ± 1.3	**0.26 ± 1.3**
BMI (kg/m^2)	17.8 ± 3.6	17.0 ± 3.6	17.8 ± 3.7	17.8 ± 3.7	**17.6 ± 3.6**	17.6 ± 3.7	17.6 ± 3.9	18.6 ± 4.0	17.3 ± 3.6	**17.9 ± 3.9**
z-BMI	0.40 ± 1.3	-0.11 ± 1.5	0.39 ± 1.4	0.31 ± 1.4	**0.25 ± 1.4**	0.19 ± 1.3	-0.19 ± 1.4	0.5 ± 1.3	-0.01 ± 1.3	**0.14 ± 1.4**
WC (cm)	61.9 ± 9.8	59.9 ± 10.7	61.2 ± 9.5	61.4 ± 9.7	**61.1 ± 10.0**	59.9 ± 9.1	59.9 ± 9.9	62.2 ± 9.3	58.7 ± 8.9	**60.6 ± 9.5**
z-WC	0.73 ± 1.2	0.26 ± 1.4	0.61 ± 1.2	0.53 ± 1.2	**0.55 ± 1.3**	0.65 ± 1.2	0.19 ± 1.5	0.93 ± 1.2	0.37 ± 1.2	**0.55 ± 1.3**
WHtR	0.460 ± 0.05	0.452 ± 0.05	0.447 ± 0.05	0.449 ± 0.05	**0.452 ± 0.05**	0.452 ± 0.05	0.434 ± 0.05	0.449 ± 0.05	0.438 ± 0.05	**0.443 ± 0.05**

Table 6.14: Characteristics of the ethnic groups by gender: BIA derived variables

	Boys					Girls				
Variable	Caucasian	South Asian	African-Caribbean	Mixed Race	Total	Caucasian	South Asian	African-Caribbean	Mixed Race	Total
% BF	20.2	20.4	20.6	20.2	**20.4**	23.4	24.1	24.8	22.9	**24.1**
	± 6.0	± 6.1	± 6.1	± 6.2	**± 6.1**	± 5.5	± 5.9	± 6.0	± 5.1	**± 5.8**
z-%BF	0.59	0.63	0.67	0.55	**0.62**	0.43	0.34	0.62	0.32	**0.45**
	± 1.1	± 1.1	± 1.1	± 1.1	**± 1.1**	± 1.1	± 1.1	± 1.1	± 1.0	**± 1.1**
FM (kg)	7.2	6.9	7.6	7.5	**7.3**	8.1	9.0	9.8	8.0	**9.0**
	± 5.1	± 5.1	± 5.3	± 5.2	**± 5.2**	± 5.2	± 5.7	± 5.8	± 5.2	**± 5.6**
FFM (kg)	26.5	24.4	27.3	27.4	**26.3**	24.3	26.0	27.4	24.8	**25.9**
	± 9.6	± 9.1	± 9.8	± 9.7	**± 9.6**	± 8.1	± 8.5	± 8.7	± 8.9	**± 8.6**
FMI (kg/m^2)	3.8	3.7	3.9	3.8	**3.8**	4.3	4.4	4.8	4.1	**4.5**
	± 2.1	± 2.0	± 2.1	± 2.1	**± 2.1**	± 2.1	± 2.2	± 2.3	± 1.9	**± 2.2**
FFMI (kg/m^2)	14.0 c	13.3	14.0	14.0	**13.8**	13.3	13.1	13.8	13.2	**13.4**
	± 1.8	± 1.8	± 1.9	± 1.9	**± 1.9**	± 1.7	± 1.8	± 1.9	± 1.8	**± 1.9**
WB Imp (Ω)	714.3	770.7	722.8	719.6	**732.9**	769.8	807.1	757.0	778.1	**779.8**
	± 80.4	± 89.3	± 88.1	± 89.0	**± 89.3**	± 81.0	± 93.9	± 90.4	± 91.7	**± 92.1**
Ht^2/Imp ($cm^2/Ω$)	26.6	23.8	27.2	27.4	**26.1**	23.7	24.8	26.4	24.2	**25.0**
	± 8.9	± 8.2	± 8.9	± 8.9	**± 8.8**	± 7.1	± 8.6	± 7.6	± 7.7	**± 7.9**

Boys

Table 6.15: Mean and SE for age-adjusted absolute variables in boys

	Caucasian	South Asian	African-Caribbean	Mixed Raced	F		PEtS
†Age (Years)	9.08 ± 2.7	8.98 ± 2.6	9.08 ± 2.7	9.39 ± 2.7	0.9	(3,1941)	†0.00
Height (cm)	135.0 ± 0.3	133.1 ± 0.3	137.2 ± 0.2	135.6 ± 0.5	42.8***	(3,1940)	0.06
Weight (kg)	33.8 ± 0.4	31.6 ± 0.4	34.9 ± 0.3	33.6 ± 0.7	14.4***	(3,1940)	0.02
BMI (kg/m^2)	17.8 ± 0.1	17.0 ± 0.1	17.8 ± 0.1	17.6 ± 0.3	6.9***	(3,1940)	0.01
WC (cm)	61.9 ± 0.3	60.1 ± 0.4	61.2 ± 0.3	60.8 ± 0.7	4.9**	(3,1936)	0.01
WHtR	0.460 ± 0.002	0.451 ± 0.002	0.447 ± 0.002	0.449 ± 0.004	6.8***	(3,1936)	0.01

*p<0.05; **p<0.01; *** p<0.001 †ANOVA-not adjusted for age (Eta Squared), PEtS=Partial Eta Squared

Table 6.16: Mean and SE for age-adjusted BIA derived variables in boys

	Caucasian	South Asian	African-Caribbean	Mixed Raced	F		PEtS
% BF	20.2 ± 0.2	20.4 ± 0.3	20.6 ± 0.2	20.1 ± 0.5	0.5	(3,1940)	0.00
Fat Mass (kg)	7.2 ± 0.2	7.0 ± 0.2	7.6 ± 0.2	7.2 ± 0.4	2.3	(3,1940)	0.00
FFM (kg)	26.5 ± 0.2	24.7 ± 0.2	27.3 ± 0.2	26.4 ± 0.4	29.8***	(3,1940)	0.04
FMI (kg/m^2)	3.8 ± 0.1	3.7 ± 0.1	3.9 ± 0.1	3.7 ± 0.2	0.868	(3,1940)	0.00
FFMI (kg/m^2)	14.0 ± 0.1	13.4 ± 0.1	14.0 ± 0.1	13.9 ± 0.1	21.8***	(3,1940)	0.03
WB Impedance (Ω)	714.3 ± 3.3	769.7 ± 3.5	722.8 ± 3.2	723.0 ± 6.6	50.6***	(3,1939)	0.07
Ht^2/Imp (cm^2/Ω)	26.6 ± 0.2	24.1± 0.2	27.2 ± 0.2	26.5 ± 0.4	44.6***	(3,1939)	0.07

*p<0.05; **p<0.01; *** p<0.001, PEtS=Partial Eta Squared

Table 6.17: Mean and SD for z-scores of anthropometric variables in boys

	Caucasian	South Asian	African-Caribbean	Mixed Raced	F		EtS
z-height	0.28 ± 1.0	-0.04 ± 1.0	0.67 ± 1.0	0.41 ± 1.1	46.8***	(3,1941)	0.07
z-weight	0.45 ± 1.3	-0.07 ± 1.4	0.65 ± 1.2	0.46 ± 1.3	π 29.0***	(3,1941)	0.05
z-BMI	0.40 ± 1.3	-0.11 ± 1.5	0.39 ± 1.4	0.31 ± 1.4	π 13.8***	(3,1941)	0.01
z-WC	0.73 ± 1.2	0.26 ± 1.4	0.61 ± 1.2	0.53 ± 1.2	π 12.7***	(3,1937)	0.02

*p<0.05; **p<0.01; *** p<0.001 π Welch test of equality of means, EtS=Eta Squared

Table 6.18: Mean and SD for z-%BF in boys

	Caucasian	South Asian	African-Caribbean	Mixed Raced	F		EtS
z-%BF	0.59 ± 1.1	0.63 ± 1.1	0.67 ± 1.1	0.55 ± 1.1	0.8	(3,1909)	0.00

*p<0.05; **p<0.01; *** p<0.001 π Welch test of equality of means, EtS=Eta Squared

Girls

Table 6.19: Mean and SE for age-adjusted absolute variables in girls

	Caucasian	South Asian	African-Caribbean	Mixed Race	F	PEtS
†Age (Years)	8.90 ± 2.5	10.07 ± 2.6	9.33 ± 2.5	9.08 ± 2.7	25.7*** (3,2187)	0.03
Height (cm)	136.2 ± 0.3	134.9 ± 0.2	139.6 ± 0.2	137.0 ± 0.5	65.8*** (3,2186)	0.08
Weight (kg)	34.7 ± 0.4	32.6 ± 0.3	37.8 ± 0.3	34.4 ± 0.7	42.6*** (3,2186)	0.06
BMI (kg/m^2)	18.0 ± 0.2	17.2 ± 0.1	18.7 ± 0.1	17.6 ± 0.3	25.6*** (3,2186)	0.03
WC (cm)	61.1 ± 0.3	58.7 ± 0.3	62.5 ± 0.3	59.6 ± 0.7	29.7*** (3,2182)	0.04
WHtR	0.450 ± 0.002	0.436 ± 0.002	0.448 ± 0.002	0.436 ± 0.004	11.4*** (3,2182)	0.02

*p<0.05; **p<0.01; *** p<0.001 †ANOVA-not corrected for age (Eta squared), PEtS=Partial Eta Squared

Table 6.20: Mean and SE for age-adjusted BIA derived variables in girls

	Caucasian	South Asian	African-Caribbean	Mixed Raced	F	PEtS
% BF	23.8 ± 0.2	23.7 ± 0.2	24.9 ± 0.2	23.2 ± 0.5	8.3*** (3,2186)	0.01
Fat Mass (kg)	8.8 ± 0.2	8.3 ± 0.2	10.0 ± 0.2	8.5 ± 0.4	18.0*** (3,2186)	0.02
FFM (kg)	25.9 ± 0.2	24.3 ± 0.2	27.8 ± 0.2	25.9 ± 0.4	64.9*** (3,2186)	0.08
FMI (kg/m^2)	4.5 ± 0.1	4.3 ± 0.1	4.9 ± 0.1	4.3 ± 0.2	12.3*** (3,2186)	0.02
FFMI (kg/m^2)	13.5 ± 0.1	12.9 ± 0.1	13.8 ± 0.1	13.3 ± 0.1	43.9*** (3,2186)	0.06
WB Impedance (Ω)	764.1 ± 3.7	813.0 ± 3.1	755.5 ± 3.2	774.1 ± 7.1	63.2*** (3,2186)	0.08
Ht^s/Imp (cm^2/Ω)	25.1 ± 0.2	23.4 ± 0.2	26.7 ± 0.2	25.2 ± 0.4	54.4*** (3,2186)	0.07

*p<0.05; **p<0.01; *** p<0.001, PEtS=Partial Eta Squared

Table 6.21: Mean and SD for z-scores of anthropometric variables in girls

	Caucasian	South Asian	African-Caribbean	Mixed Raced	F	EtS
z-height	0.15 ± 1.0	-0.07 ± 1.1	0.74 ± 1.1	0.29 ± 1.2	76.3*** (3,2187)	0.10
z-weight	0.23 ± 1.2	-0.13 ± 1.3	0.74 ± 1.2	0.16 ± 1.3	π 60.5*** (3,2187)	0.08
z-BMI	0.19 ± 1.3	-0.19 ± 1.4	0.50 ± 1.3	-0.01 ± 1.3	π 31.6*** (3,2187)	0.04
z-WC	0.65 ± 1.2	0.19 ± 1.5	0.93 ± 1.2	0.37 ± 1.2	π 39.7*** (3,2183)	0.05

*p<0.05; **p<0.01; *** p<0.001 π Welch test of equality of means, EtS=Eta Squared

Table 6.22: Mean and SD for z-%BF in girls

	Caucasian	South Asian	African-Caribbean	Mixed Raced	F	EtS
z-%BF	0.43 ± 1.1	0.34 ± 1.1	0.62 ± 1.1	0.32 ± 1.0	9.2*** (3,2156)	0.02

*p<0.05; **p<0.01; *** p<0.001, EtS=Eta Square

Table 6.23: Mean and SE in age-adjusted anthropometric variables in boys and girls from the collapsed ethnic groups

Variable		Boys	Girls	F		PEts
†Age (Years)						
	Caucasian	9.08 ± 2.7	8.90 ± 2.5	1.3	(1,1132)	†0.00
	South Asian	8.98 ± 2.6	10.07 ± 2.6	56.4***	(1,1325)	†0.04
	African-Caribbean	9.08 ± 2.7	9.33 ± 2.5	3.1	(1,1374)	†0.00
	Mixed Race	9.39 ± 2.7	9.08 ± 2.7	1.0	(1,297)	†0.00
Height (cm)						
	Caucasian	134.5 ± 0.2	133.4 ± 0.2	8.7**	(1,1131)	0.00
	South Asian	136.5 ± 0.3	135.8 ± 0.2	3.3	(1,1324)	0.00
	African-Caribbean	138.0 ± 0.3	138.1 ± 0.2	0.1	(1,1373)	0.00
	Mixed Race	136.5 ± 0.6	135.6 ± 0.6	1.2	(1,296)	0.00
Weight (kg)						
	Caucasian	33.4 ± 0.3	32.7 ± 0.4	2.1	(1,1131)	0.00
	South Asian	33.8 ± 0.4	33.3 ± 0.3	1.2	(1,1324)	0.00
	African-Caribbean	35.4 ± 0.3	36.7 ± 0.3	6.6*	(1,1373)	0.00
	Mixed Race	34.3 ± 0.7	33.5 ± 0.7	0.7	(1,296)	0.00
BMI (kg/m^2)						
	Caucasian	17.8 ± 0.1	17.7 ± 0.1	0.1	(1,1131)	0.00
	South Asian	17.4 ± 0.1	17.3 ± 0.1	0.6	(1,1324)	0.00
	African-Caribbean	17.9 ± 0.1	18.5 ± 0.1	11.9**	(1,1373)	0.01
	Mixed Race	17.7 ± 0.3	17.4 ± 0.3	0.6	(1,296)	0.00
WC (cm)						
	Caucasian	61.7 ± 0.3	60.1 ± 0.3	12.2***	(1,1129)	0.01
	South Asian	61.4 ± 0.4	58.9 ± 0.3	26.6***	(1,1320)	0.02
	African-Caribbean	61.5 ± 0.3	61.9 ± 0.3	1.1	(1,1372)	0.00
	Mixed Race	61.1 ± 0.6	59.0 ± 0.6	5.5*	(1,295)	0.02
WHtR						
	Caucasian	0.460 ± 0.002	0.452 ± 0.002	7.4**	(1,1129)	0.01
	South Asian	0.450 ± 0.002	0.435 ± 0.002	25.1***	(1,1320)	0.02
	African-Caribbean	0.447 ± 0.002	0.450 ± 0.002	1.2	(1,1372)	0.00
	Mixed Race	0.449 ± 0.004	0.437 ± 0.004	4.9*	(1,295)	0.02

*p<0.05; **p<0.01; *** p<0.001 †ANOVA-not corrected for age (Eta Squared), PEtS=Partial Eta Squared

Table 6.24: Mean and SE in age-adjusted BIA derived variables in boys and girls from the collapsed ethnic groups

Variable		Boys	Girls	F		PEts
%BF						
	Caucasian	20.2 ± 0.2	23.5 ± 0.2	91.9***	(1,1131)	0.08
	South Asian	20.8 ± 0.3	23.8 ± 0.2	85.1***	(1,1324)	0.06
	African-Caribbean	20.6 ± 0.2	24.7 ± 0.2	163.0***	(1,1373)	0.11
	Mixed Race	20.1 ± 0.5	23.0 ± 0.5	19.6***	(1,296)	0.06
Fat Mass (kg)						
	Caucasian	7.2 ± 0.2	8.2 ± 0.2	14.5***	(1,1131)	0.01
	South Asian	7.6 ± 0.2	8.6 ± 0.2	12.3***	(1,1324)	0.01
	African-Caribbean	7.8 ± 0.2	9.7 ± 0.2	54.4***	(1,1373)	0.04
	Mixed Race	7.3 ± 0.4	8.2 ± 0.4	2.9	(1,296)	0.01
FFM (kg)						
	Caucasian	26.3 ± 0.2	24.5 ± 0.2	40.9***	(1,1131)	0.04
	South Asian	26.2 ± 0.2	24.8 ± 0.2	30.3***	(1,1324)	0.02
	African-Caribbean	27.7 ± 0.2	27.0 ± 0.2	6.4*	(1,1373)	0.01
	Mixed Race	27.0 ± 0.4	25.3 ± 0.4	8.5**	(1,296)	0.03
FMI (kg/m^2)						
	Caucasian	3.8 ± 0.1	4.3 ± 0.1	21.9***	(1,1131)	0.02
	South Asian	3.8 ± 0.1	4.3 ± 0.1	17.7***	(1,1324)	0.01
	African-Caribbean	3.9 ± 0.1	4.8 ± 0.1	64.3***	(1,1373)	0.05
	Mixed Race	3.7 ± 0.2	4.2 ± 0.2	3.8	(1,296)	0.01
FFMI (kg/m^2)						
	Caucasian	14.0 ± 0.1	13.4 ± 0.1	53.3***	(1,1131)	0.05
	South Asian	13.6 ± 0.1	13.0 ± 0.1	53.4***	(1,1324)	0.04
	African-Caribbean	14.0 ± 0.1	13.7 ± 0.1	10.0**	(1,1373)	0.01
	Mixed Race	14.0 ± 0.1	13.2 ± 0.1	15.4***	(1,296)	0.05
WB Impedance (Ω)						
	Caucasian	715.1 ± 3.1	768.9 ± 3.3	140.3***	(1,1131)	0.11
	South Asian	762.2 ± 3.8	812.8 ± 3.1	106.1***	(1,1323)	0.07
	African-Caribbean	721.7 ± 3.4	758.0 ± 3.3	60.0***	(1,1373)	0.04
	Mixed Race	720.9 ± 7.1	776.8 ± 7.3	30.2***	(1,296)	0.09
$Ht^2/Imp(cm^2/Ω)$						
	Caucasian	26.4 ± 0.2	24.0 ± 0.2	85.2***	(1,1131)	0.07
	South Asian	25.5 ± 0.2	23.7 ± 0.2	31.8***	(1,1323)	0.02
	African-Caribbean	27.5 ± 0.2	26.0 ± 0.2	30.9***	(1,1373)	0.02
	Mixed Race	27.0 ± 0.4	24.6 ± 0.4	17.8***	(1,296)	0.06

*p<0.05; **p<0.01; *** p<0.001 PEtS=Partial Eta Squared

Table 6.25: Mean and SD for z-scores of anthropometric variables in boys and girls from the collapsed ethnic groups

Variable		Boys	Girls	F		EtS
z-height						
	Caucasian	0.28 ± 1.0	0.15 ± 1.0	5.0*	(1,1132)	0.00
	South Asian	-0.04 ± 1.0	-0.07 ± 1.0	0.4	(1,1325)	0.00
	African-Caribbean	0.67 ± 1.0	0.74 ± 1.1	1.6	(1,1374)	0.00
	Mixed Race	0.41 ± 1.1	0.29 ± 1.2	0.8	(1,297)	0.00
z-weight						
	Caucasian	0.45 ± 1.3	0.23 ± 1.2	8.6**	(1,1132)	0.00
	South Asian	-0.07 ± 1.4	-0.13 ± 1.3	0.7	(1,1325)	0.00
	African-Caribbean	0.65 ± 1.2	0.74 ± 1.2	1.8	(1,1374)	0.00
	Mixed Race	0.46 ± 1.3	0.16 ± 1.3	4.1*	(1,297)	0.01
z-BMI						
	Caucasian	0.40 ± 1.3	0.19 ± 1.3	7.3**	(1,1132)	0.00
	South Asian	-0.11 ± 1.5	-0.19 ± 1.4	1.0	(1,1326)	0.00
	African-Caribbean	0.39 ± 1.4	0.50 ± 1.3	2.1	(1,1374)	0.00
	Mixed Race	0.31 ± 1.4	-0.01 ± 1.3	4.1*	(1,297)	0.01
z-WC						
	Caucasian	0.73 ± 1.2	0.65 ± 1.2	1.1	(1,1130)	0.00
	South Asian	0.26 ± 1.4	0.19 ± 1.5	0.6	(1,1321)	0.00
	African-Caribbean	0.61 ± 1.2	0.93 ± 1.2	23.9***	(1,1373)	0.02
	Mixed Race	0.53 ± 1.2	0.37 ± 1.2	1.3	(1,296)	0.00

*p<0.05; **p<0.01; *** p<0.001 EtS=Eta Square

Table 6.26: Mean and SD of z-%BF in boys and girls from the collapsed ethnic groups

Variable		Boys	Girls	F		EtS
z-%BF						
	Caucasian	0.59 ± 1.1	0.43 ± 1.1	6.3*	(1,1111)	0.00
	South Asian	0.63 ± 1.1	0.34 ± 1.1	21.0***	(1,1306)	0.02
	African-Caribbean	0.67 ± 1.1	0.62 ± 1.0	0.7	(1,1357)	0.00
	Mixed Race	0.55 ± 1.1	0.32 ± 1.0	3.3	(1,291)	0.01

*p<0.05; **p<0.01; *** p<0.001 EtS=Eta Square

Table 6.27: Prevalence of overall overweight and obesity (BMI) on whole group and gender basis

Ethnic group	Boys		Girls		Whole group	
Caucasian						
Overweight	11.0%	(66 / 598)	10.6%	(57 / 536)	10.8%	(123 / 1134)
Obese	12.7%	(76 / 598)	9.3%	(50 / 536)	11.1%	(126 / 1134)
Overweight inc obese	23.7%	(142 / 598)	20.0%	(107 / 536)	22.0%	(249 / 1134)
South Asian						
Overweight	10.6%	(56 / 527)	11.5%	(92 / 800)	11.2%	(148 / 1327)
Obese	10.6%	(56 / 527)	5.4%	(56 / 527)	7.5%	(99 / 1327)
Overweight inc obese	21.3%	(112 / 527)	16.9%	(135 / 800)	18.6%	(247 / 1327)
African-Caribbean						
Overweight	12.0%	(80 / 667)	13.1%	(93 / 709)	12.6%	(173 / 1376)
Obese	13.5%	(90 / 667)	14.1%	(100 / 709)	13.8%	(190 / 1376)
Overweight inc obese	25.5%	(170 / 667)	27.2%	(193 / 709)	26.4%	(363 / 1376)
Mixed Race						
Overweight	9.8%	(15 / 153)	8.9%	(13 / 146)	9.4%	(28 / 299)
Obese	13.1%	(20 / 153)	8.9%	(13 / 146)	11.0%	(33 / 299)
Overweight inc obese	22.9%	(35 / 153)	17.8%	(26 / 146)	20.4%	(61 / 299)
Total						
Overweight	11.2%	(217 / 1945)	11.6%	(255 / 2191)	11.4%	(472 / 4136)
Obese	12.4%	(242 / 1945)	9.4%	(206 / 2191)	10.8%	(448 / 4136)
Overweight inc obese	23.6%	(459 / 1945)	21.0%	(461 / 2191)	22.2%	(920 / 4136)

Table 6.28: Prevalence of central overall overweight and obesity (WC) on whole group and gender basis

Ethnic group	Boys	Girls	Whole group
Caucasian			
Overweight	13.2% (79 / 597)	11.2% (60 / 535)	12.3% (139 / 1132)
Obese	14.2% (85 / 597)	15.1% (81 / 535)	14.7% (166 / 1132)
Overweight inc obese	27.5% (164 / 597)	26.4% (141 / 535)	26.9% (305 / 1132)
South Asian			
Overweight	9.7% (51 / 525)	11.9% (95 / 798)	11.0% (146 / 1323)
Obese	13.0% (68 / 525)	11.8% (94 / 798)	12.2% (162 / 1323)
Overweight inc obese	22.7% (119 / 525)	23.7% (189 / 798)	23.2% (308 / 1323)
African-Caribbean			
Overweight	12.9% (86 / 667)	17.8% (126 / 708)	15.4% (212 / 1375)
Obese	12.1% (81 / 667)	18.9% (134 / 708)	15.6% (215 / 1375)
Overweight inc obese	25.0% (167 / 667)	36.7% (260 / 708)	31.1% (427 / 1375)
Mixed Race			
Overweight	9.9% (15 / 152)	11.6% (17 / 146)	10.7% (32 / 298)
Obese	12.5% (19 / 152)	10.3% (15 / 146)	11.4% (34 / 298)
Overweight inc obese	22.4% (34 / 152)	21.9% (32 / 146)	22.1% (66 / 298)
Total			
Overweight	11.9% (231 / 1941)	13.6% (298 / 2187)	12.8% (529 / 4128)
Obese	13.0% (253 / 1941)	14.8% (324 / 2187)	14.0% (577 / 4128)
Overweight inc obese	24.9% (484 / 1941)	28.4% (622 / 2187)	26.8% (1106 / 4128)

6.6 Discussion

The subject of paediatric body composition has received much attention over the last 20 years particularly since the period when adult population/ethnic groups were found to vary in their prevalence of overweight and obesity and risk of obesity related morbidity. As a result, there is now a better understanding on the variation in body composition between children from different ethnic groups and how this relates to health and disease. Several studies have characterized and compared body composition both between and within populations and ethnic groups. However, no study to date has explored these variations using an extensive range of both anthropometric measures as well body composition measures obtained using BIA. In view of this, the aim of this study was to examine and describe the ethnicity-related variation in a range of anthropometric and BIA-derived variables that characterize whole-body as well as regional body composition in children aged between 5 and 16 years from Caucasian, SA, AC and MR backgrounds.

Gender-specific variation between the ethnic groups was examined firstly by adjusting the measures for age. Secondly, variation between the ethnic groups was then examined by comparing the measures against relevant population references including the UK 1990 references [55], the UK %BF references [308] and the UK WC references [342]. Thirdly, a gender comparison was conducted in all ethnic groups. Finally, prevalence of whole body (BMI) and central (WC) overweight and obesity was determined for the whole sample and for the various ethnic groups.

Prevalence of overweight and obesity (BMI) in boys and girls from all ethnic groups in this study was lower than in boys (31%) and girls (30%) aged 2 to 15 years in the most recent Health Survey for England (HSE) 2007 [17]. The lower prevalence in this study was most likely due to the fact that the 91st and 98th cut-offs were used to define overweight and obesity whereas the 85th and 95th centiles were used in the HSE 2007.

Although differences between the SA subgroups (Indian, Pakistani & Bangladeshi) were apparent for measures compared against the UK references, very few differences between the groups were observed for the age-adjusted absolute

variables, as seen in Tables 6.4 and 6.5. Generally, Pakistani children were found to be taller, heavier and have a greater mean BMI compared with the UK 1990 reference population. In contrast, Bangladeshi and Indian children were generally shorter and had a lower mean weight and BMI compared with the UK 1990 reference [55].

Similar to the SA sub-groups, little variation in the majority of the age-adjusted absolute measures was observed between the AC sub-groups (Caribbean and African). However, the AC sub-groups appeared to be more similar in measures relative to the UK reference populations than the SA sub-groups. Thus greater homogeneity in body dimensions and composition was observed between the Africans and Caribbeans than between the SA sub-groups.

Variations in body build and composition between the SA and AC sub-groups has been examined in a number of national surveys. One such example is the HSE 2004 [25], which demonstrated differences in height, weight and BMI (using age-standardized regression coefficients) between the sub-groups by comparing these measures to those of the general population. This survey found that Pakistani boys and Indian girls were taller and heavier than their counterparts in the other SA sub-groups. Furthermore, Black Caribbean boys were found to be taller and heavier than African boys. Conversely, Black African girls were found to be taller and heavier than Caribbean females. A difference in height between the SA sub-groups has also been shown in the 2006-2007 National Child Measurement Programme (NCMP) [520]. This survey showed that Bangladeshi children were shorter than Indian and Pakistani children. Another study on British Asian children (aged 5 years and younger) which used a classification system based on religion found that Sikh children were taller and heavier than Hindu and Muslim children [521]. The authors of this study proposed that the greater weight and height in the Sikh group could partly be explained by the fact that the Sikh community was the most affluent group thus increasing the likelihood of a "more wholesome diet" being consumed by this group.

It is evident from this study and those described above that there is variation between the SA and AC sub-groups. However, it is difficult to draw conclusions on how body dimensions and composition varies between the SA and AC sub-groups as the differences between the sub-groups observed in this study do not show a consistent

pattern to that in the national surveys. This could be a function of a number of factors including the smaller sample size of some of the sub-groups or specific characteristics of the children in this study. It is also evident that the method by which these sub-groups are classified can differ as it can be based on geographical, cultural or religious criteria. Given that few differences between the SA and AC sub-groups were observed in this study, it was deemed more appropriate to examine ethnicity-related variation in measures of body dimensions and composition by merging the sub-groups to form the SA and AC groups.

In this study, AC boys and girls were found to be taller and heavier than their Caucasian, SA and MR counterparts. Additionally, SA children were found to be shorter and lighter than children from the other ethnic groups. These findings have been mirrored in the HSE 2004 survey [25], which found that mean height in Caribbean boys and girls and African girls was greater than in the general population. In comparison, Indian, Pakistani (girls) and Bangladeshi children were shorter than the general population. Furthermore, a study on UK children also observed that Caucasian children were taller and heavier than South Asian children [180].

Variations in linear growth between children are a function of both genetic and environmental influences. The genetic influence on linear growth is apparent through the fact that parental height has an influence on offspring height with studies such as the Louisville Twin Study reporting that hereditary factors account for more than 90% of the factors that determine height for children aged 6 years and above [529].

The findings of this study suggest that ethnicity may have a role in determining height and weight gain in children and adolescents. In support of this argument, one study on infants and young children found that greater height in African and Europeans than in Indian children was explained by ethnicity and not environmental and nutritional factors [522]. Contrastingly, studies on preschool children have shown less variation in height between ethnic groups than between socioeconomic groups within a given ethnic group [522]. It is evident from such findings that the mechanism by which ethnicity influences the genetic potential and environmental factors that determine height remains unclear.

The greater height and weight in the AC children is reflected in the greater BMI. Similarly, in the HSE 2004 [25], average BMI was also greater in the Black Caribbean and African children than in the other ethnic groups. Despite having the greatest BMI, AC boys in this study did not differ in their predicted body fatness (%BF) to boys in the other ethnic groups. Contrastingly, the greater BMI in AC girls was mirrored by greater predicted body fatness in this group. Thus, despite being a proxy for body fatness, BMI appears to show differential abilities to predict fatness in boys and girls. The better predictive value of BMI in girls could be due to the fact that a greater proportion of their body weight is made up of fat mass [322,523]. This can be seen from the greater proportion of FMI as a component of BMI in girls than in boys in this study.

The greater BMI in AC children compared to other ethnic groups as seen in this study and those described earlier is thought to be a function of greater height rather than greater weight for height. As a proxy measure of adiposity, BMI is adjusted for the effect of height and so in adults it is relatively independent of stature. BMI in children is only partially adjusted for height as evident from the positive correlation between these measures in children up to the age of 15 years [524]. Thus, children who are taller for their age such as the AC children would have a higher BMI than those who are shorter for their age such as the SA children, in part due to a greater FFMI.

A direct consequence of the stature-BMI relationship in children from minority ethnic groups may be that a greater number of AC children are classified as being overweight or obese. Evidence for this can be seen in this study as well as in previous studies [525] where a greater prevalence of overweight and obesity was observed in the AC children. Furthermore, prevalence of overweight and obesity combined was lowest in the SA children in this study. Contrary to these findings, BIA derived %BF and FM (kg) in boys was not significantly different between the ethnic groups. Although a similar paradox of higher BMI and lower predicted body fatness was not observed in the girls, the findings of this study indicate that BMI may overestimate the obesity related risk in AC children and underestimate this in SA children. Thus, these findings give evidence against the use of BMI in identifying excess body fatness particularly in boys from minority ethnic groups and instead support the use of more objective measures of body fatness to classify children. Further support for this can be drawn from evidence which has shown that it is excess

body fat and not excess weight that is associated with obesity related morbidity [526]. A consequence of the overestimation of prevalence of overweight and obesity in AC children is that unnecessary weight management intervention could be implemented specifically for this group. In contrast, SA children could be missed due to a lower BMI and so their risk of obesity-related ill health will increase especially since they are known to be characterized by greater body fatness for a given BMI [319] and because they are at greater risk of obesity-related morbidity [180].

It was also evident that ethnicity-related variation in BMI in boys was predominantly due to variation in FFMI. In contrast, ethnicity-related variation in girls was due to a variation in both FMI and FFMI. This highlights another limitation of examining variations in body composition using proxy measures of fatness such as BMI and emphasises the need to use objective measures of fatness. It is for these reasons that this study has gone further than previous studies and surveys by describing variations between ethnic groups not using BMI alone but including additional measures of body fatness and distribution.

As described earlier, no ethnicity-related variation in %BF, absolute FM and FMI was observed in boys. However in girls, body fatness was significantly greater in the AC group than in the Caucasian, SA and MR groups with no difference noted between these latter groups. These findings are in contrast to those of a study which characterized body fatness in British schoolchildren aged 5 to 18 years from AC, Caucasian and SA backgrounds. This study found that body fatness was greater in SA children than in AC children [527]. A similar finding was also reported by another study which examined ethnicity-related variation in body fatness in British schoolchildren using BIA [525].

This discrepancy in ethnicity-related variation in body fatness between this study and those described above could be a function of the prediction equation used by the BIA system in this study. The BIA system used in this study has not as yet been validated in non-Caucasian populations. Thus, it could be argued that the prediction equations may not be applicable to children from non-Caucasian backgrounds and so should not strictly be used to compare definitive variations in body composition between ethnic groups. As highlighted in Chapter 2, variations in the hydration of FFM between ethnic groups would require ethnic-specific equations for predicting %BF

using BIA [288]. However, it should be noted that TBW as a fraction of FFM has shown not to vary between Black and White children [456-459]. Furthermore, it appears from the literature that the hydration of FFM in South Asian children has not been systematically examined. The applicability of the BC-418 prediction equation in children from non-Caucasian backgrounds also needs to be questioned in terms of ethnicity-related variations in body proportions and specific resistivity. Examination of variation between ethnic groups in these factors seems only to have been conducted in Black and White groups with the former having greater Impedance, apparently due to greater leg length and resistivity in the lower limbs [519]. A consequence of this would be that FFM would be underestimated and FM overestimated in this ethnic group.

Although the predicted variables from the BIA model used in this study may provide less accurate results on the body composition of children from non-Caucasian backgrounds, the raw measurement obtained using this technology may offer a better indication of ethnicity-related variation in body composition. Impedance, which is defined as the "frequency-dependent opposition of a conductor to the flow of an alternating electric current" [298] would be greater in individuals with greater body fat [288]. Mean impedance was significantly greater in SA boys and girls than in the Caucasian, AC and MR children. This finding possibly indicates that body fatness in this study may actually be greater in SA children thus corresponding with the findings on British schoolchildren described earlier [527].

FFM as an absolute (kg) and as a proportion of BMI (FFMI) was generally observed to be greater in AC children than in Caucasian, SA and MR children. In contrast, both measures of FFM were lower in SA children than in children from the other ethnic groups. Similarly Ht^2/Imp was also greatest in AC children (significant only in girls) and lowest in SA boys and girls. Ht^2/Imp uses the raw impedance measurement from the BC418-MA and is a measure of the conducting volume of the body i.e. TBW or FFM. The agreement in the ethnicity-related variation between the predicted FFM and Ht^2/Imp gives confidence in the findings of this study and suggests that FFM predicted from BIA is able to reflect true ethnicity-related variation in this measure. An explanation for the greater FFM in the AC children and lower FFM in the SA children can be found from studies which have shown that BMD is greater [461-465,467-

469,470-474,476] and skeletal muscle mass greater and more dense in Black than in Caucasian and Asian children [483,493,495-497].

Abdominal fatness assessed using WC was greater in Caucasian than in SA boys with no difference observed between the former group and the AC and MR boys. However, partial correction of WC for height (expressed as WHtR) was observed to be lower in AC than in Caucasian boys. A similar pattern was evident in girls where AC girls were found to have the greatest degree of abdominal fatness when assessed using WC, however WHtR was shown to be similar to that of Caucasian girls. Thus the inclusion of height seems to have an impact on reducing the extent of abdominal fatness particularly in AC children.

Relative to the UK 1990 reference population, it was observed that Caucasian, AC and MR boys were taller, heavier and had a greater BMI (excluding MR children). In contrast to the other ethnic groups, SA boys and girls were shorter, less heavy and had a lower BMI than the reference population. Secular increases in height have been demonstrated in European as well as non European populations such as African-American, Indians in South India and the UK, Australian Aborigines, Brazilian and Japanese populations. Furthermore, there is evidence to suggest the non-European populations have not reached a plateau in the secular increase in height [528].

Greater weight and BMI in AC and Caucasian children relative to the UK 1990 population are coherent with the secular increase in weight-for-height in children across the globe. Evidence for this can be seen in the increasing trends in global prevalence of both overweight and obesity in children and adolescents [14]. Given that growth in children is sensitive to environmental conditions, the secular increases in height and weight are thought to reflect the greater availability of energy dense food [261,528]. Additionally, secular increases in weight and BMI are also thought to be a consequence of the changing physical environment which is encouraging physical inactivity [261].

SA children in this study were found to be slightly shorter with a weight and BMI that was also slightly lower than that of the UK 1990 reference population. In comparison, one study on UK SA and White European adolescents found that boys (-0.46) and

girls (-0.02) in the former group were shorter relative to the UK 1990 population [190]. Nevertheless, weight and BMI were greater in UK SA boys and girls relative to the reference population. Another study on Pakistani children aged between 5 and 14 years found that growth in height and weight was comparable to that of the UK 1990 population [512]. A plausible explanation for the discrepancy between the findings of this study and that of the UK adolescents [190], particularly with regards to the lower weight and BMI relative to the UK 1990 reference population, may be that SA children in this study were not representative of the UK SA population. It is possible that the SA children in this study are more representative of children that live in a deprived, inner city area where environmental factors influencing growth in height and weight are less favourable.

Whole body fatness and abdominal fatness was greater in children from all ethnic groups, relative to the UK %BF and WC reference populations respectively. This indicates and supports the findings of previous studies which have shown secular increases in total body and abdominal fatness, albeit using anthropometric measurements such as BMI and WC, both in children and adults [14,327]. Of greater interest are the findings in SA children in this study which show that despite being similar (or lower) in height and weight to the UK 1990 population, whole body fatness and abdominal fatness in this ethnic group are greater relative to the populations in the more recently developed %BF [308] and WC [342] references. Thus this study may provide preliminary evidence to indicate that the secular increase in total and abdominal fatness in this ethnic group has exceeded that of height and weight.

This paradox of a lower BMI and greater body fat in the SA group is not surprising given the inherent greater whole body and central body fatness in this ethnic group compared to other ethnic groups and because of the limitations associated with the use of BMI to explore ethnicity-related variations in body composition and body fat distribution as discussed earlier and in Chapter 2. This paradox of lower BMI and greater body fatness in SA children has also been reported by a study which found that despite a greater proportion of AC children being classified as overweight (using BMI), body fatness was in fact greater in SA children [527]. Similar findings have also been described in SA adults and consequently, BMI cut-offs for overweight and obesity of 25 kg/m^2 and 30 kg/m^2 have recently been lowered to 23 kg/m^2 and 25 kg/m^2 respectively for the Asian Indian adult population [321].

A gender comparison between all ethnic groups showed that AC girls were taller, heavier and had a greater BMI and WC than boys. This was in contrast to the other ethnic groups where these measures were greater in boys. The greater mean BMI in the AC girls is reflected in the greater prevalence of whole body and central overweight and obesity than in the AC boys. A similar finding has been reported in the 2006-2007 NCMP survey which found that Black African and Caribbean girls had a greater likelihood of being classified as obese (compared to White British children) than boys [520]. However, the reverse was true in the other ethnic groups as boys were more likely than girls to be classified as obese. These finding possibly suggest that girls in the AC group may be at greater risk of obesity related morbidity than boys in this ethnic group. Contrastingly, obesity-related risk in Caucasian, SA and MR children is likely to be greater in boys than in girls.

In summary, ethnicity-related variation in whole body and regional body fatness has been observed in this study in both boys and girls. It is evident from this study that exploring ethnicity-related variation in body composition is a complex issue given that a relatively large number of measures are needed to characterize body composition. This matter is complicated even further when considering which measures are best suited to explore variations between ethnic groups. As discussed earlier, BMI is the most commonly used measure of overweight and obesity and this is partly because of the ease with which height and weight measurements can be obtained in a wide range of settings. Furthermore, the existence of BMI references makes it easier to draw comparisons across ethnic groups. In contrast, reference techniques such as DXA and ADP provide more direct (as opposed to predictive) measures of body fatness but are expensive and are less readily available outside the research/clinical setting.

It is for these reasons that BIA is now recognised as an acceptable and practical method of assessing body composition especially since the technology has advanced greatly over the last few years. However, as described earlier, the use of BIA is limited to the population in which the equipment is validated. With reference to this study, the Tanita BC-418MA has not to date been validated in non-Caucasian groups and so comparisons between ethnic groups are likely to be subject to errors. Thus, it is acknowledged that the absolute values on %BF and FFM derived using the BC-418 may not be as accurate as in the Caucasian children. However, these limitations were

addressed by using raw BIA measurements and anthropometric measurements to describe variations between ethnic groups. Furthermore, it is apparent from this study that the BIA-derived variables have provided an understanding of ethnicity-related variation in body composition beyond that provided by BMI.

The limitations of exploring ethnicity-related variation in body composition using BMI have become evident in this study. It has also become clear that there is a need for a wider availability of practical techniques of assessing body composition that have been validated in children from non-Caucasian backgrounds. The development of ethnic-specific assessment techniques also has implications on how these measures are interpreted and so this begs the important but complex question of whether ethnic-specific references for body composition are required.

Although this study has highlighted important findings on the variation in whole body and regional body composition between ethnic groups, the strengths and weaknesses of this study also have to be addressed. Confidence in the findings of this study can be drawn from that fact that intra-and inter-observer error associated with performing anthropometric measurements was low as discussed in Chapter 3. This is also evident from the fact that ethnicity-related variation in height, weight and WC was greater than the error level of these measurements (as seen in Chapter 3). Furthermore, the principal measurers were trained personnel who had prior experience in conducting these measurements and operating the Tanita BC-418MA scales.

Finally, it should be noted that subjects for this study were recruited from boroughs within London, with the majority of them coming from inner city London. Although a large proportion of the UK ethnic population resides in London, it is likely that the subjects in this study are not truly representative of Caucasian, SA, AC and MR children in the UK. It is possible that the subjects in this study are representative of children that live in a deprived, inner-city area. Thus the ethnicity-related variations in body composition discussed in this study could be specific to children living within a geographical area in London.

Chapter 7: Construction of gender- and ethnic-specific smoothed percentiles for anthropometric variables and measures obtained from bioelectrical impedance analysis.

7.1 Introduction

The distribution of a measurement relative to a changing covariate such as age can be represented using reference centile (percentile) curves. Reference centile curves are essential to understanding age-related changes in children in the context of both clinical settings as well as epidemiological studies because growth and maturation are characterized by changes in measures of body dimensions including weight, height, size and composition. Reference centile curves for children and adolescents have therefore been developed in measures such as BMI, waist circumference (WC) and % Body Fat [5,308,342]. The age-related change in BMI, as evident from the UK BMI centile charts, shows an increase in BMI during infancy with a peak seen at 9 months of age. This is followed by a decrease up until the sixth year after which there is a rise in BMI that lasts until adulthood (Figure 2.4, Chapter 2) [5].

The development of reference centile curves firstly requires collection and collation of the relevant measurements from a reference population. As most anthropometric measurements are generally not normally distributed and tend to have a distribution that is positively skewed (longer right tail), it is important to deal with the skewness in the data before these growth references can be developed [13]. The most commonly used method for constructing normalized growth references and standards is the LMS method [13].

The LMS method is based on the assumption that the data can be normalised and the skewness removed by stretching one tail of the distribution and shrinking another using a power transformation. The LMS method summarizes the data into three smooth age-specific curves called the L (lambda, the power transformation), M (mu, the mean), and S (sigma, the coefficient of variation) curves. The L curve allows for skewness in the distribution of the data, which arises from non-normality in the data. It represents the power needed to normalise the data at each age group. The M and S curves represent the median (the 50^{th} centile) and coefficient of variation of the data respectively [13]. Fitting of the three curves for each age enables calculation of the

required centiles and converting measurements into z-scores (also known as standard deviation scores). The 50th centile is equivalent to the M curve. Centiles other than the 50th can be calculated using the L and S curves.

%Body fat (%BF) reference curves were recently developed for Caucasian children in the UK using the LMS method [308]. %BF measurements were obtained using whole-body Bioelectrical Impedance analysis (BIA). As descried earlier, this technique relies on the indirect prediction of TBW and FFM by measuring the opposition or impedance to the flow of an electric current. These gender specific %BF curves were developed using the Tanita BC-418MA system which is a relatively new research-type BIA model. This model has been validated in Caucasian adults and children against the reference methods DXA and ADP [305,307]. Although work is in progress, this model has not yet been validated for use in non-Caucasian populations. Hence, equations to predict measures of body composition using this model are specific to Caucasian populations and possibly result in less accurate results when applied to non-Caucasian populations. As outlined in Chapters 2 and 6, factors such as differences in body proportions between ethnic groups are likely to affect the prediction of TBW/FFM and subsequently % BF [298]. As a result, caution needs to be exercised when using this model to predict body composition in children and adults from non-Caucasian backgrounds.

Ethnic specific differences in whole body and regional body composition have been discussed in this thesis and described in the paediatric literature [6,7]. A better understanding of this subject had led to the development of ethnic/population specific centile curves for measures such as BMI [5,6-9], WC [308,339-341] and %BF [308,530]. Furthermore, centile curves representing the overweight and obese cut-offs for BMI have also been developed for international use by pooling data from various populations [313].

Although the predicted variables obtained from the BC-418 MA should not strictly be used to compare definitive variations in body composition between ethnic groups, its raw measurements may help to address this issue. The single raw measurement obtained from BIA is impedance (Ω). This is based on the assumption that impedance to the flow of current in the body is directly related to the length (height) of the conductor and inversely related to its cross-sectional area. When expressed in terms

of body volume, it is known that the volume of TBW (and hence FFM) is directly related to length squared (or height squared/ ht^2) and indirectly related to impedance [298]. This constitutes the impedance index ($Height^2$/Impedance) and can be calculated by dividing the square of height (cm) by the impedance (Ω) measurement obtained from the BC-418 system [298].

Given the absence of any comparative data between children from different ethnic groups with respect to BIA-derived %BF, the purpose of this study was to generate ethnic and gender specific centile curves for anthropometric measures of whole body and regional body fatness including BMI (kg/m^2) and WC (cm) respectively and raw and predicted measures derived using BIA including $Height^2$/Impedance (Ht^2/Imp, cm^2/ Ω), Impedance (Ω) and % Body Fat (%BF).

It should be noted that the purpose of this study was not to generate working centile charts for these BIA-derived measures but to examine the variations in these curves between ethnic groups. Indeed it is unlikely that some charts (i.e. whole body impedance, and Ht^2/Imp) would have any practical utility in clinical or research settings, but this graphical depiction could provide insights into the nature of the variability in BIA-derived body composition measures between children from different ethnic groups.

7.2 Aims and Objectives

7.2.1 Aims

To generate gender- and ethnic-specific centile curves for whole body Impedance (WB Imp), $Height^2$/Impedance (Ht^2/Imp), predicted %BF, BMI and WC for Caucasian, South Asian (SA) and African-Caribbean (AC) children aged between 5 and 13 years.

7.2.2 Objectives

Using the LMS software to generate charts displaying the 2nd, 50th and 98th centile curves for all measures described above in children from the three ethnic groups.

7.3 Methodology

7.3.1 Subjects

The subjects for this analysis comprised of 3621 school children (1671 Boys, 1950 Girls) aged between 5 and 13 years. Parents/carers were contacted by letter explaining the purpose of the study and to request permission for their child to take part. No child was measured where consent was refused. Data on date of birth, gender and ethnicity were collected at the time of data collection. Children were individually coded and the data anonymized. Ethnicity was coded using a condensed version of the Department for Children, Schools and Families ethnicity classification (Appendix B &C) [531].

7.3.2 Anthropometric and BIA derived variables

All measurements were conducted on school premises and the same trained measurers were mainly responsible for data collection. Height and WC were measured according to the procedure described in Chapter 3 [2]. Body mass (measured to the nearest 0.1 kg with correction for light indoor clothing), whole body Impedance and whole body %BF were measured using the Tanita BC-418MA Segmental Body Composition Analyzer (Tanita Corporation, Tokyo, Japan) according to the procedure described in Chapter 3.

7.3.3 Ethical approval

This study was approved by the London Metropolitan University Ethics Committee (Appendix F).

7.3.4 Data handling and statistical analysis

Data was uploaded onto Microsoft Excel (Microsoft ® Office Excel 2003) and sorted by gender and ethnic group. Next a separate spreadsheet was created for boys and girls in each ethnic group. Decimal age was calculated for all children using the equation in Chapter 3. Smoothed centile curves for all variables were constructed using the LMS Chartmaker Pro software (Child Growth Foundation) [312]. Data was transferred from Microsoft Excel (Microsoft ® Office Excel 2003) to the LMS software and models were fitted using the original age and degrees of freedom of 3, 5 and 5

for the L, M and S curves respectively. This was performed separately for boys and girls in all three ethnic groups. The 2nd, 50th and 98th centiles curves were generated for all variables and exported back to an Excel spreadsheet. Centile charts for all variables were then constructed using the Chartmaker option in Microsoft Excel and compared between ethnic groups.

7.4 Results

7.4.1 Sample size

With the exception of age 12 and 13 years, the sample size was greater than the recommended minimum number of 50 children in all ethnic groups (T.Cole, personal communication).

7.4.2 Whole body Impedance (Ω)

An overall pattern of decreasing impedance with age was seen in boys and girls from all ethnic groups at the three centiles. Impedance was greater in girls than in boys at all ages and for all centiles and in all ethnic groups (Figures 7.1 & 7.2, Table 7.2). Impedance at all centiles was greater in SA boys and girls than in their Caucasian and AC counterparts across the entire age range. Impedance was lowest in the Caucasian boys at all ages and at the 50th and 98th centile. This was with the exception at age 11 years at the 50th centile where Impedance was lower in the AC than in the Caucasian boys (702.8Ω vs. 705.6Ω). In girls, Impedance was lowest in the AC group at the 50th centile and in the Caucasian group at the 98th centile. At the 2nd centile, Impedance was lowest in the AC boys and girls. It is can be seen from Figures 7.1 and 7.2 that Caucasian and AC boys and girls are more similar in their age-related decrease in Impedance whereas in SA children there appears to be this upward shift in the age-related decrease in impedance.

7.4.3 Height^2/Impedance (Ht^2/Imp) (cm^2/ Ω)

Figures 7.3 and 7.4 show a comparison of the Ht^2/Imp centiles between ethnic groups separated by gender. An age-related increase in Ht^2/Imp was seen in boys and girls from all ethnic groups between the age of 5 and 13 years. Ht^2/Imp was greater in boys than in girls at all ages and centiles and in all ethnic groups. Furthermore, the

increase in Ht^2/Imp between the age of 5 and 13 was greatest at the 98th centile and smallest at the 2nd centile in boys and girls from all ethnic groups (Table 7.3).

Generally, Ht^2/Imp was greatest in the AC boys and girls and lowest in the SA boys and girls. A more consistent separation in Ht^2/Imp between ethnic groups is seen in girls than in boys (Figures 7.3 and 7.4). In boys, the SA group had a consistently lower Ht^2/Imp than Caucasian and AC boys at the 2nd and 50th centile. However, at the 98th centile, there was a greater difference between SA boys and boys from the other ethnic groups between the age of 5 and 10 years whereas beyond the age of 11 years, Ht^2/Imp in the SA boys was almost similar to that of the Caucasian boys.

7.4.4 % Body Fat

The gender-related pattern in %BF seen in this study is similar to that seen in the curves generated by McCarthy et al (2006) [3]. As with the UK reference, boys in all ethnic groups also showed a relatively flat 50th centile with a peak at age 11 years (Figure 7.5). However, a higher range in body fat at the 50th centile of between 18% and 20% is seen in boys from all ethnic groups in this study compared to that of the UK reference which has a range of between 15% and 18% [3].

As with the UK reference, a greater age-related increase in girls is seen in %BF up to the age of 10 years after which the rate of increase decelerated (Figure 7.6). This was with the exception of AC girls at the 98th centile where a decrease in %BF was seen after the age of 10 years. This finding in the AC could be a function of the smaller sample size at the upper end of the body fat distribution. SA children differed slightly in their age related pattern compared to the AC and Caucasian girls in this study and also to the Caucasian girls in the UK reference. It can be seen from Figure 7.6 that SA girls also had an age-related increase in %BF although the rate in change seemed to decrease at a later age of 12 years which is possibly related to the reported later onset of puberty in Asian girls [19].

Greater %BF in girls than in boys at age 18 years seen in the UK reference was also apparent at the top end of the age range in this study. Table 7.4 shows that the median %BF was greater in girls than in boys in the Caucasian (24.2% vs. 18.4%), SA (25.7% vs. 19.2%) and AC (26.2% vs. 18.3%) group.

7.4.5 Body Mass Index (kg/m^2)

The age related patterns in BMI seen in this study was similar to that of the UK 1990 references [1]. As seen from Figures 7.7 and 7.8, BMI increased across the entire age range in boys and girls from all ethnic groups. Furthermore, similar to the UK references, BMI was seen to increase more rapidly in girls than in boys across the entire age range. BMI at the 50^{th} centile and at age 13 years was seen to be greater in girls than in boys. However, this gender difference was greater in AC (20.5 kg/m^2 vs. 18.8 kg/m^2) than in Caucasian (19.4 kg/m^2 vs. 19.3 kg/m^2) and SA children (19.2 kg/m^2 vs. 18.8 kg/m^2).

At the 50^{th} and 2^{nd} centile, BMI was lowest in the SA boys and relatively similar in the Caucasian and AC boys. At the 98^{th} centile, BMI increased rapidly between the age of 5 and 11 years after which it flattened off in the SA and Caucasian boys and continued to increase in the AC boys. Furthermore, BMI at the 98^{th} centile in SA boys aged 5 years was 3.4kg/m^2 and 3.8kg/m^2 lower than in Caucasian and AC boys respectively (Table 7.5 & Figure 7.7). Despite having a lower BMI at age 5 years, SA had a steeper increase in BMI up to the age of 11 years than Caucasian and AC boys.

A flattening off in median BMI was observed between the age of 5 and 6 years in girls from the three ethnic groups (Figure 7.8, Table 7.5). Following this, an increase in BMI was seen up to the age of 13 years in girls from all ethnic groups. At all ages and centiles, BMI was greatest in the AC girls and lowest in the SA girls. A similar pattern was observed at the 2^{nd} centile except that BMI decreased between the age of 5 and 6 years in girls from all ethnic groups. Furthermore, BMI at the 2^{nd} centile was lowest in SA girls and relatively similar in Caucasian and AC girls between the age of 5 and 13 years. At the 98^{th} centile, BMI increased up to the age of 10 years in Caucasian and AC girls after which the rate of increase decelerated. A similar pattern was observed in SA girls except that the rate of increase changed at the age of 12 years. As with the 50th centile, BMI was greatest in the AC girls and lowest in the SA girls.

Figures 7.7 and 7.8 also show that a more consistent separation in BMI between ethnic groups is seen in girls than in boys with SA girls a having a lower BMI and AC girls having a higher BMI at all ages.

7.4.6 Waist circumference (cm)

Similar to the UK WC references, an increase in age-related WC was observed in both boys and girls from all ethnic groups (Figures 7.9 and 7.10). At the 50th and 2nd centile, WC was lowest in the SA boys and relatively similar in the Caucasian and AC boys. At the 98th centile, WC at age 5 years was lower in SA boys than in AC and Caucasian boys by 4.8cm and 6.2cm respectively. The pattern had reversed at the age of 13 years where WC was greater in SA boys than in Caucasian and AC boys by 5.5cm and 6.2cm respectively.

At the 50th and 2nd centile, WC was greatest in AC girls and lowest in the SA girls although a smaller difference between the Caucasian and AC girls was seen at the latter centile.

At the 98th centile, WC at age 5 years was lower in SA girls than in Caucasian and AC girls by 1.2cm and 5.8cm respectively (Table 7.6). Contrastingly, WC was highest in the Caucasian girls and lowest in the AC girls at the upper end of the age range. Furthermore, WC was higher in the Caucasian than in SA and AC girls by 4.1cm and 5.7cm respectively. The change in the centile position of the AC girls relative to the other ethnic groups is a result of the deceleration in the increase of WC at age 10 years. In contrast to the Caucasian and AC girls, SA girls had a continual increase in WC across the entire age range.

Figure 7.1: Chart representing centile curves (2nd, 50th, 98th) for WB Imp (Ω): Boys

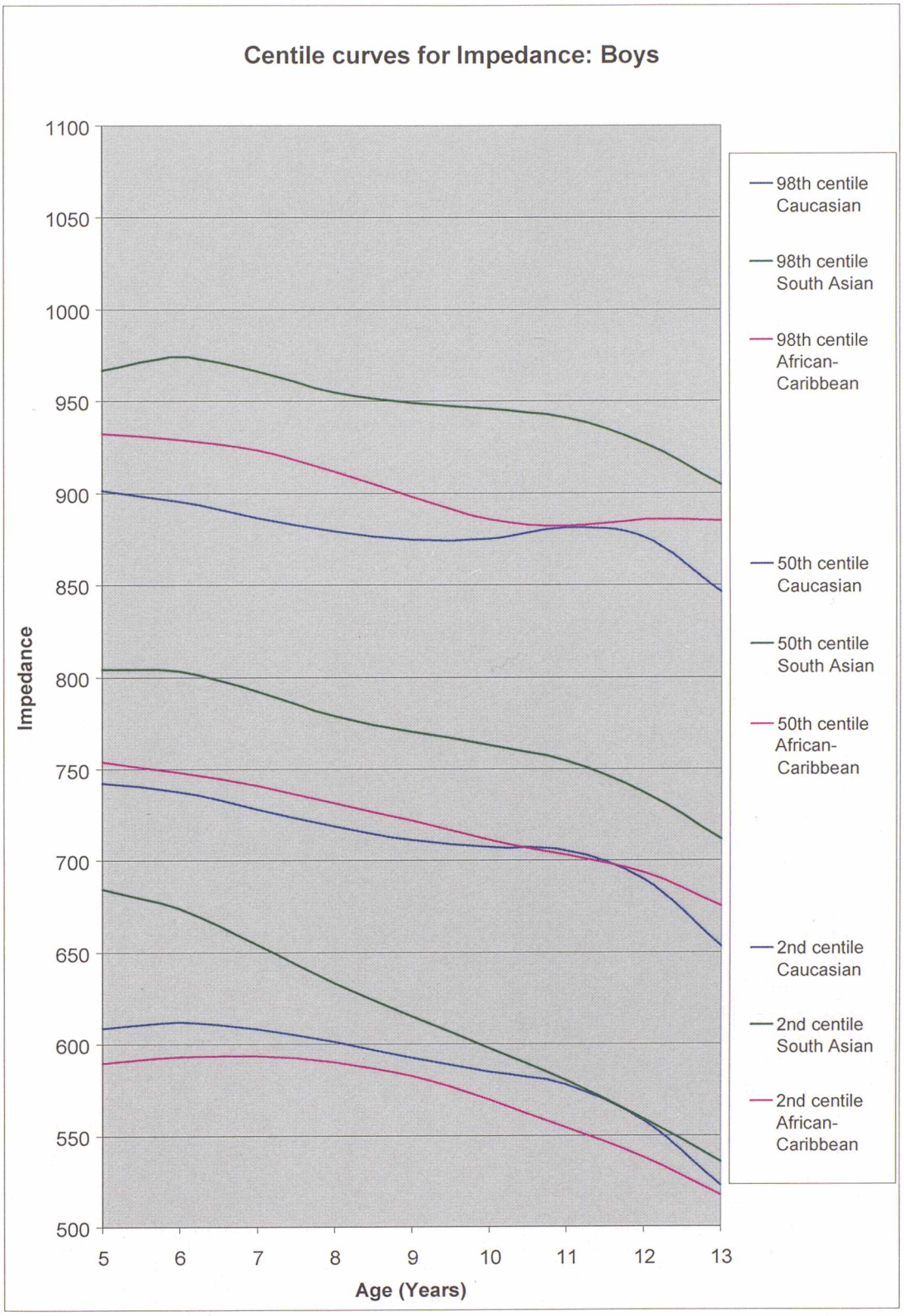


Figure 7.2: Chart representing centile curves (2nd, 50th, 98th) for WB Imp (Ω): Girls

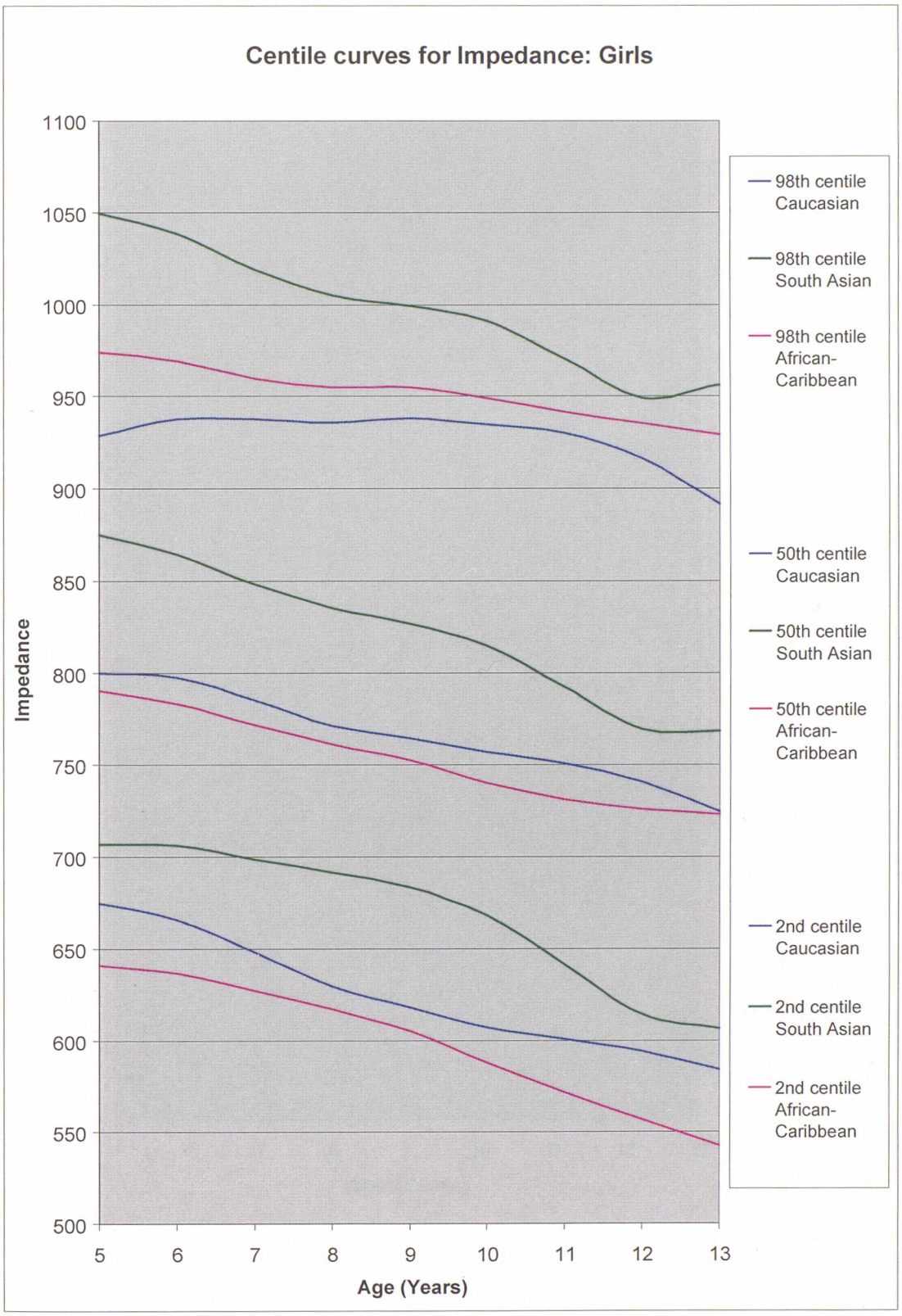


Figure 7.3: Chart representing centile curves (2nd, 50th, 98th) for Ht²/Imp (cm²/Ω): Boys

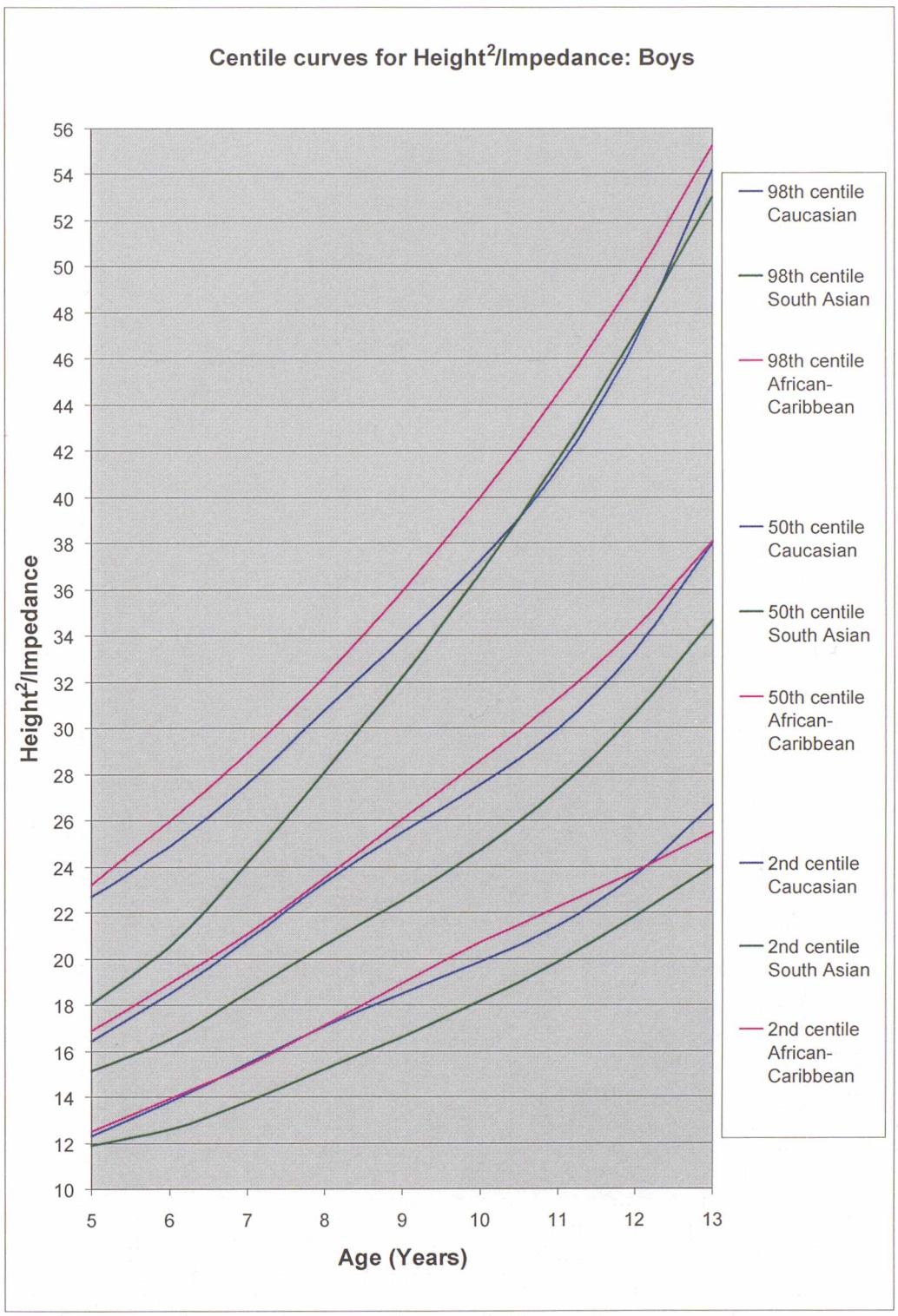


Figure 7.4: Chart representing centile curves (2nd, 50th, 98th) for Ht²/Imp (cm²/Ω): Girls

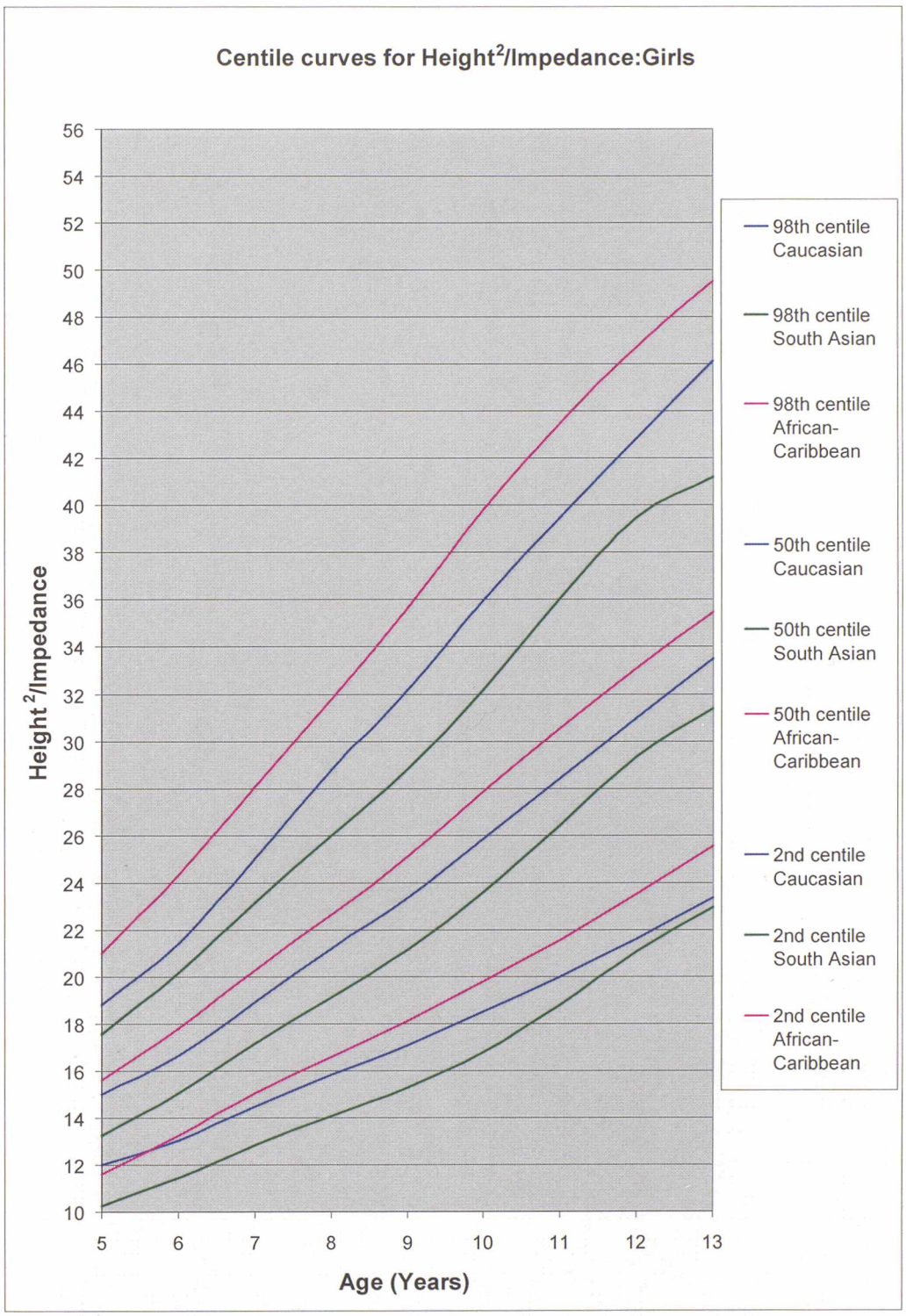


Figure 7.5: Chart representing centile curves (2nd, 50th, 98th) for % BF: Boys

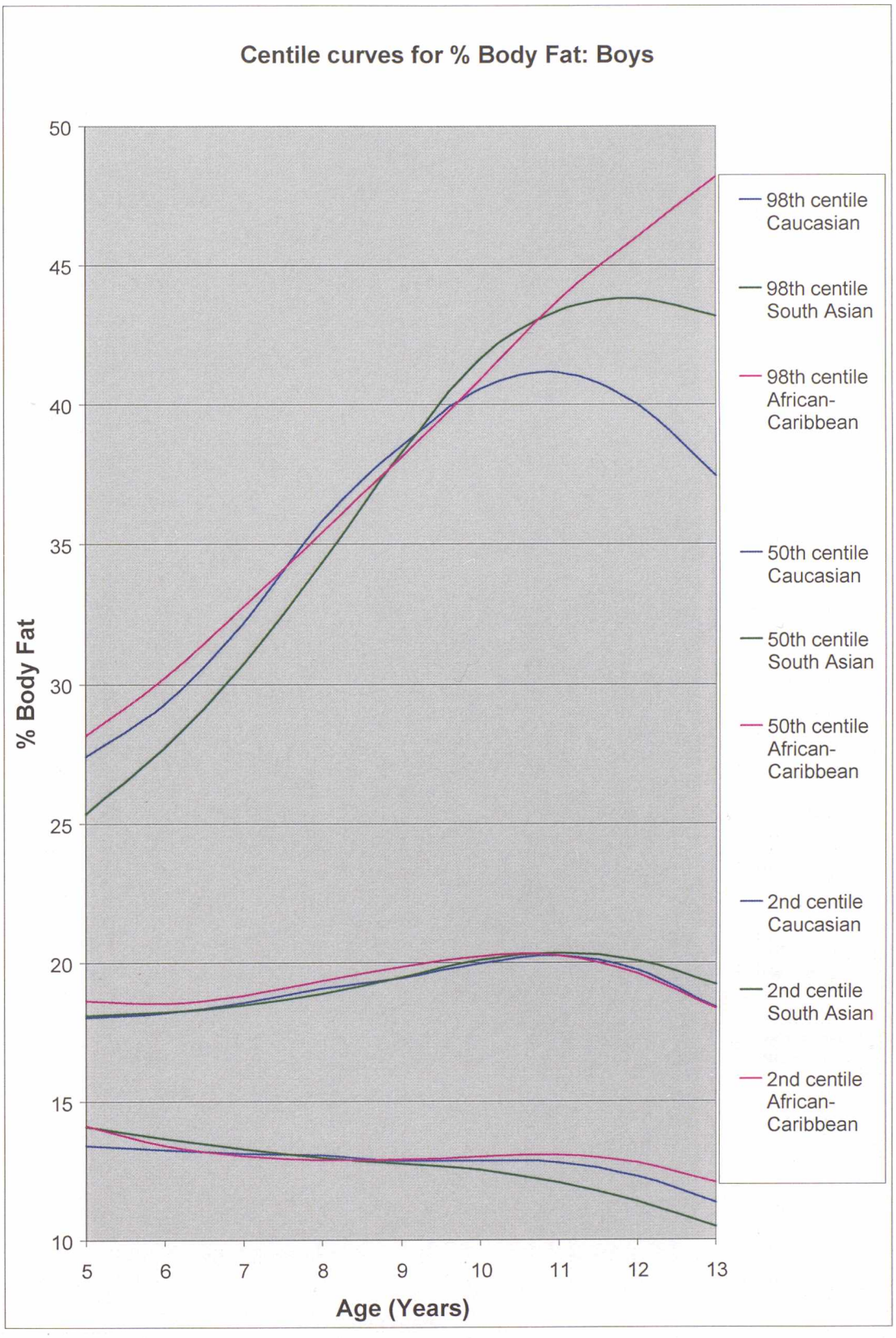


Figure 7.6: Chart representing centile curves (2nd, 50th, 98th) for % BF: Girls

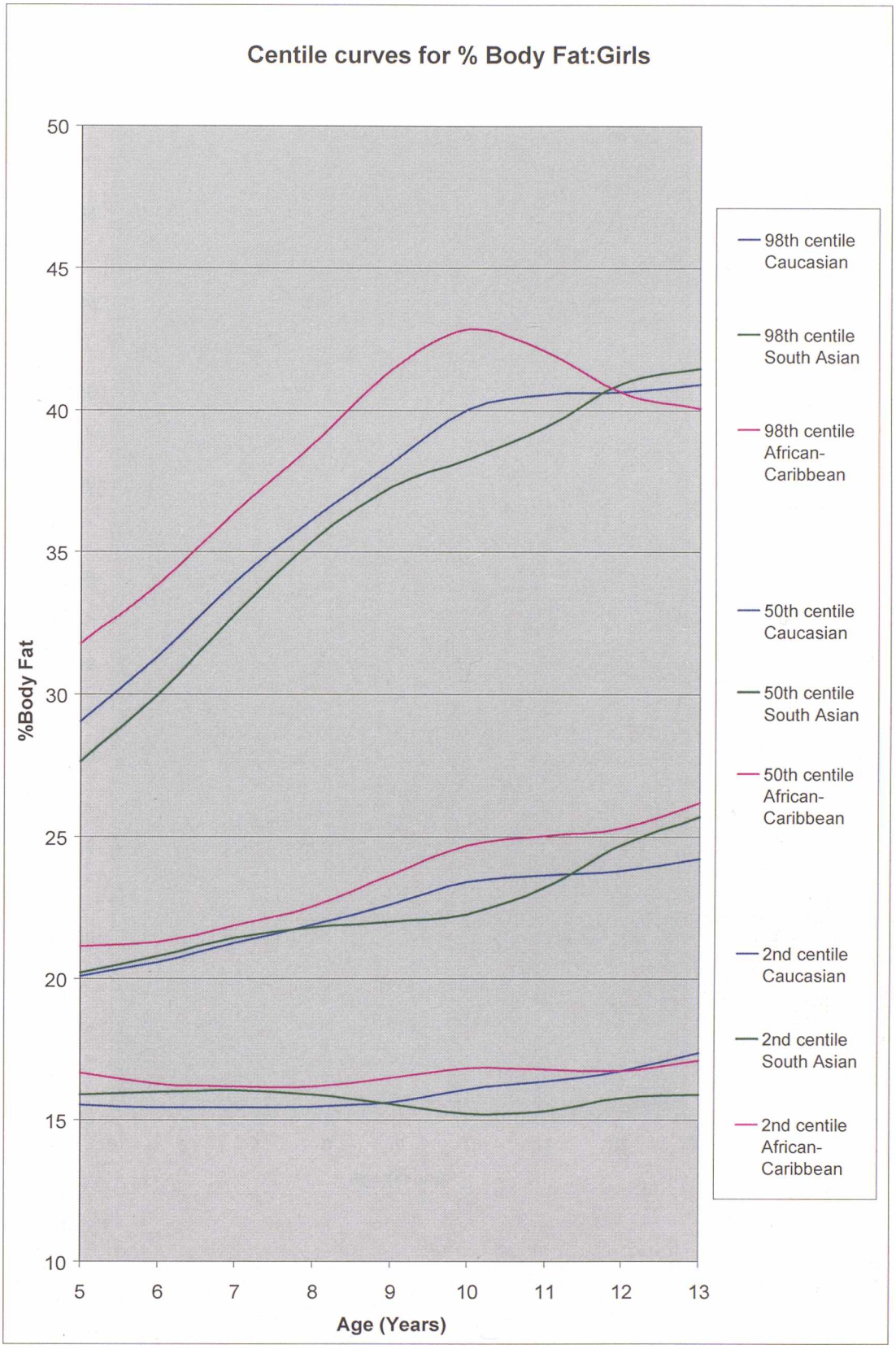


Figure 7.7: Chart representing centile curves (2nd, 50th, 98th) for BMI (kg/m^2): Boys

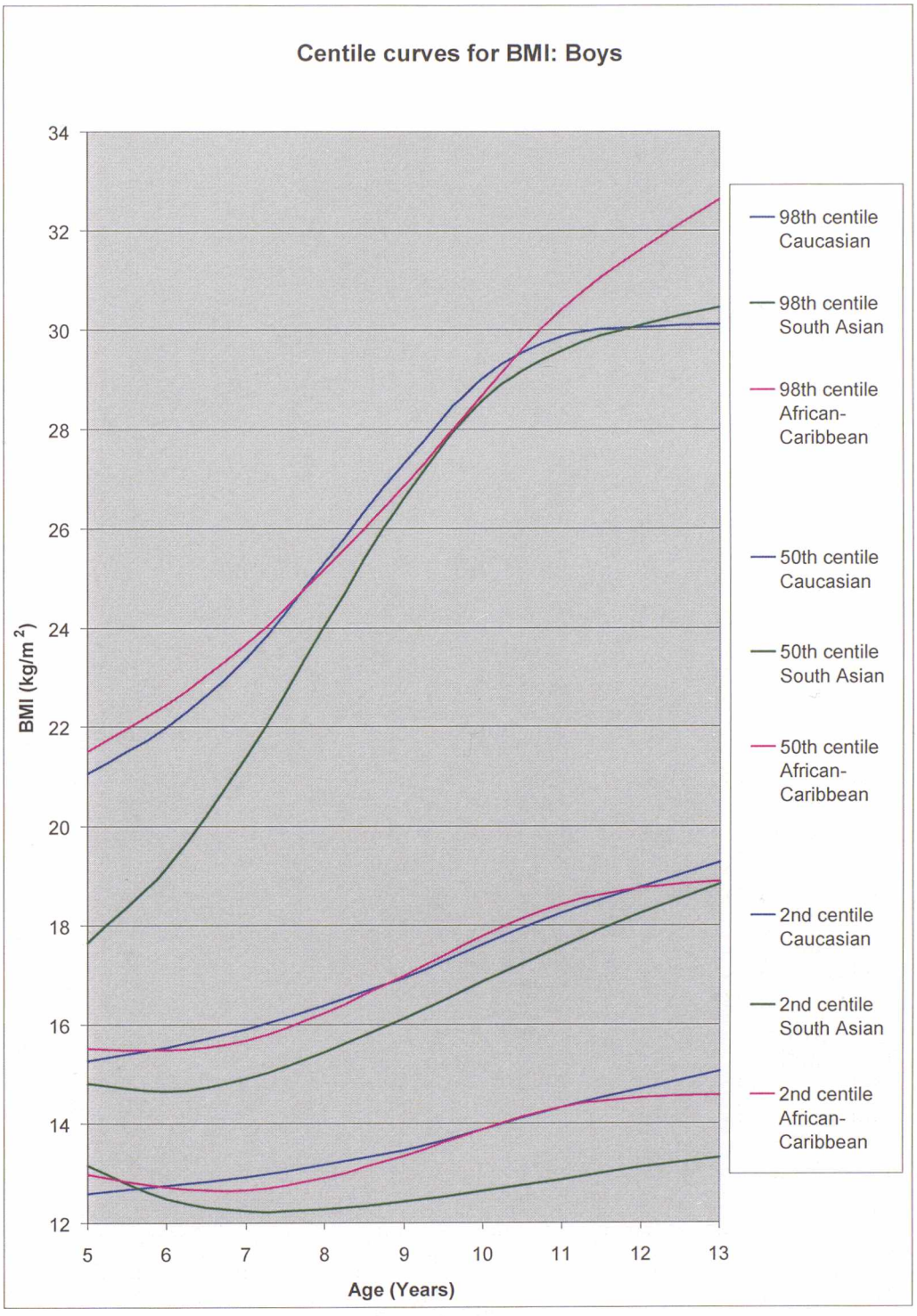


Figure 7.8: Chart representing centile curves (2nd, 50th, 98th) for BMI (kg/m²): Girls

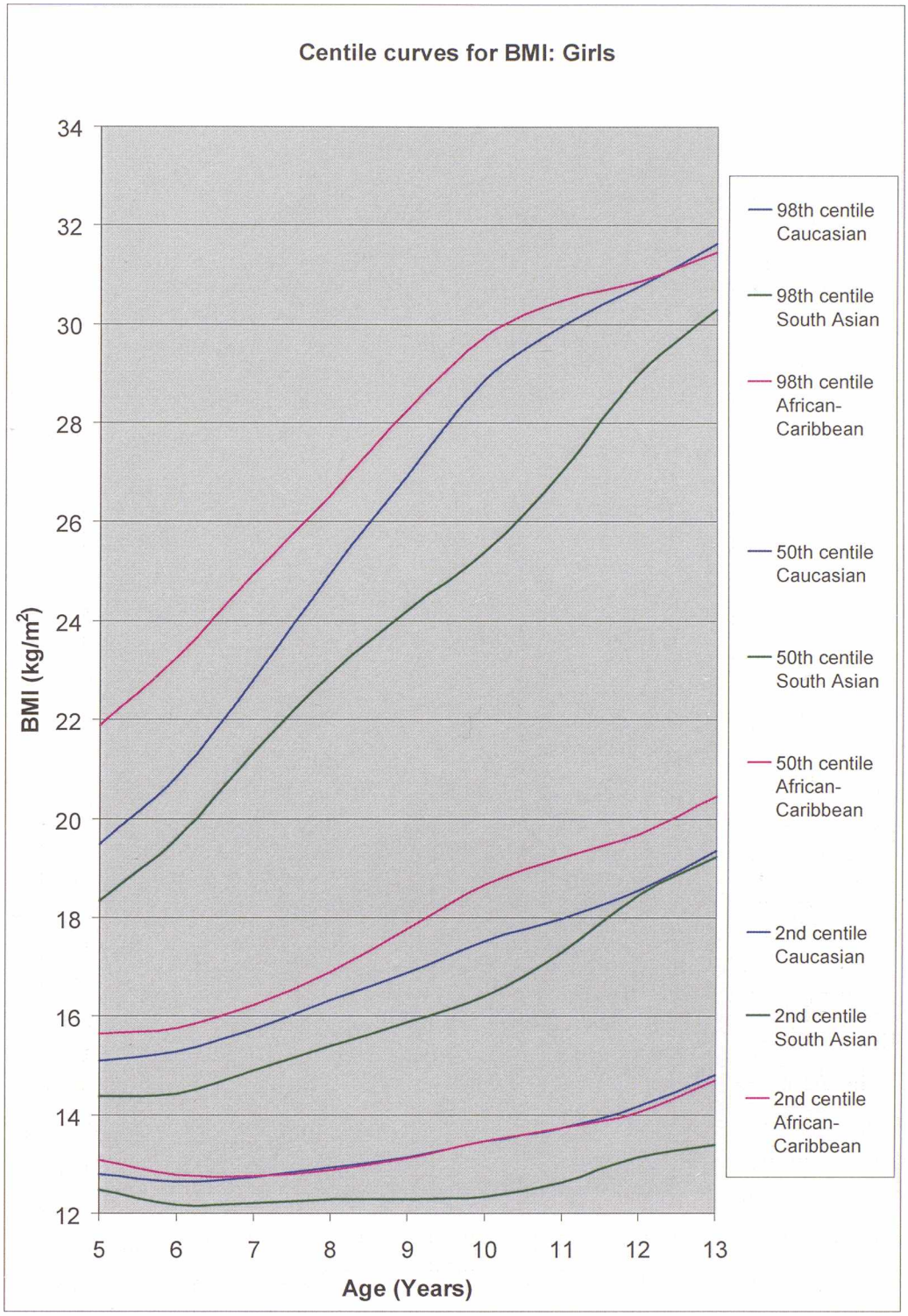


Figure 7.9: Chart representing centile curves (2nd, 50th, 98th) for WC (cm): Boys

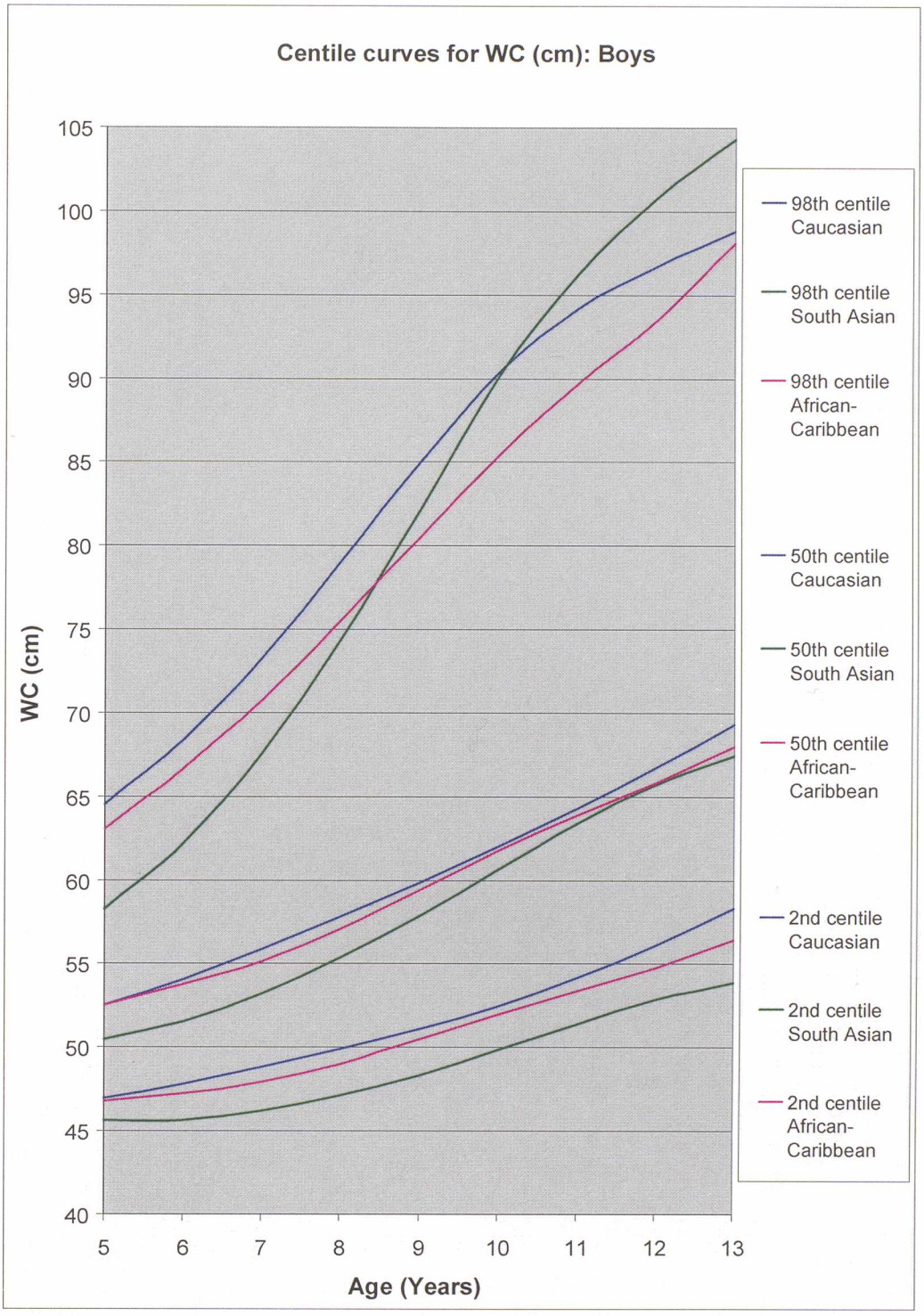


Figure 7.10: Chart representing centile curves (2nd, 50th, 98th) for WC (cm): Girls

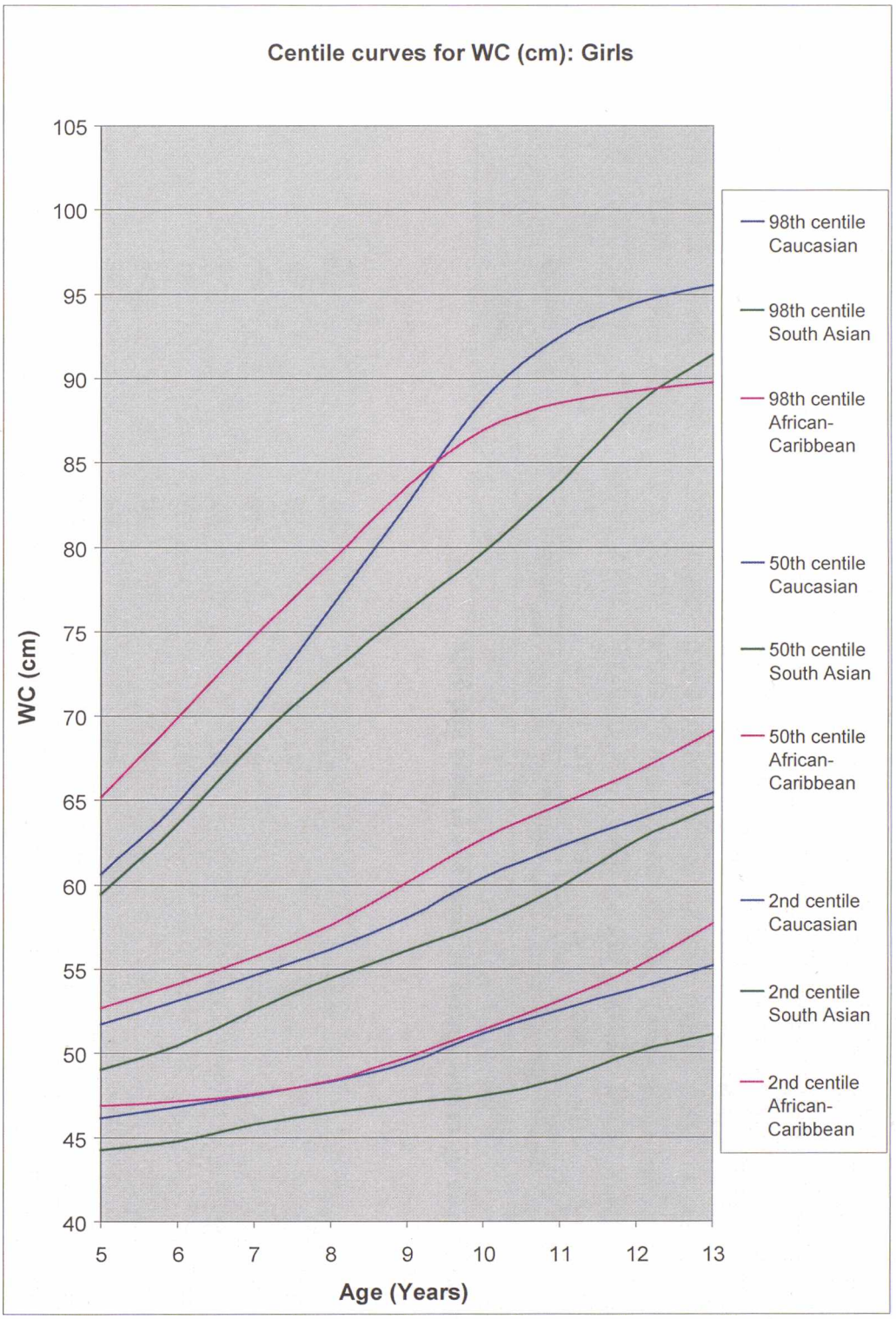


Table 7.1: Number of children in each ethnic and age group

	Boys			Girls		
Age	Caucasian	South Asian	African-Caribbean	Caucasian	South Asian	African-Caribbean
5+	63	60	78	55	56	52
6+	79	72	73	69	55	74
7+	73	70	81	70	75	101
9+	61	54	67	62	64	86
10+	68	67	83	67	46	96
11+	57	55	61	59	194	82
13+	29	40	27	23	56	26
Total	553	503	615	507	776	667

Table 7.2: Tabulated WB Impedance (Ω) centile values by exact age in boys and girls

	Boys									Girls								
	2nd centile			50th centile			98th centile			2nd centile			50th centile			98th centile		
Age	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC
5+	608.9	684.4	589.3	742.2	803.9	753.6	901.4	966.9	932.5	674.5	706.9	640.9	800.0	874.5	790.1	928.7	1049.3	974.0
6+	612.2	673.8	592.9	737.6	803.2	748.0	895.6	974.2	929.1	665.4	706.2	636.4	797.1	864.4	783.3	937.6	1038.3	969.4
7+	608.2	654.2	593.5	727.8	792.0	740.8	886.2	966.3	923.3	648.0	698.6	627.1	784.8	847.8	771.6	937.5	1018.6	959.8
8+	601.1	633.1	590.0	718.7	778.9	731.4	879.4	954.7	912.0	629.6	691.5	616.8	771.1	835.1	761.1	935.7	1005.1	954.8
9+	592.3	615.1	582.5	711.2	770.3	721.8	874.3	949.2	898.1	617.8	683.3	605.2	764.5	826.4	752.4	938.3	999.1	954.9
10+	584.5	597.7	569.6	707.5	763.0	711.4	874.7	945.7	885.4	606.8	667.8	587.9	756.6	814.4	740.2	935.0	991.0	949.3
11+	577.4	579.9	554.2	705.6	754.2	702.8	881.0	941.0	882.1	600.7	641.2	571.4	750.5	792.3	731.1	930.3	970.6	941.8
12+	558.1	559.0	537.9	689.7	736.9	693.5	875.7	927.3	885.7	593.9	614.0	557.0	740.5	769.4	725.8	916.0	949.4	935.6
13+	522.4	535.6	517.0	653.4	711.7	675.2	846.4	904.7	885.1	583.8	606.4	542.7	724.5	768.5	723.0	891.3	956.4	929.0

C=Caucasian; SA=South Asian; AC=African-Caribbean

Table 7.3: Tabulated Ht^2/Imp centile values by exact age in boys and girls

	Boys									Girls								
	2nd centile			50th centile			98th centile			2nd centile			50th centile			98th centile		
Age	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC
5+	12.3	11.9	12.5	16.4	15.1	16.9	22.7	18.0	23.2	12.0	10.2	11.6	15.0	13.2	15.6	18.8	17.5	21.0
6+	13.8	12.6	13.9	18.5	16.5	18.9	24.9	20.6	26.0	13.0	11.4	13.2	16.6	15.0	17.8	21.4	20.1	24.3
7+	15.4	13.8	15.4	20.8	18.5	21.1	27.6	24.1	28.9	14.5	12.8	15.0	18.9	17.1	20.3	25.0	23.2	28.1
8+	17.1	15.2	17.1	23.3	20.6	23.5	30.8	28.1	32.2	15.8	14.1	16.6	21.2	19.1	22.6	28.8	26.0	31.8
9+	18.5	16.6	18.9	25.5	22.5	26.0	33.9	32.2	35.9	17.1	15.3	18.1	23.4	21.2	25.1	32.2	28.8	35.7
10+	19.8	18.2	20.7	27.5	24.7	28.6	37.2	36.7	40.0	18.5	16.8	19.8	25.9	23.6	27.9	36.0	32.2	39.8
11+	21.4	19.9	22.3	30.0	27.3	31.3	41.2	41.6	44.5	20.0	18.8	21.6	28.4	26.4	30.5	39.5	36.0	43.5
12+	23.6	21.9	23.8	33.4	30.6	34.3	46.7	47.1	49.5	21.6	21.1	23.5	31.0	29.3	33.1	42.9	39.5	46.7
13+	26.6	24.0	25.5	38.0	34.7	38.1	54.2	53.0	55.2	23.3	22.9	25.6	33.5	31.4	35.5	46.1	41.2	49.5

C=Caucasian; SA=South Asian; AC=African-Caribbean

Table 7.4: Tabulated % BF centile values by exact age in boys and girls

	Boys									Girls								
	2nd centile			50th centile			98th centile			2nd centile			50th centile			98th centile		
Age	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC
5+	13.4	14.1	14.2	18.0	18.1	18.6	27.4	25.3	28.2	15.5	15.9	16.7	20.1	20.2	21.1	29.0	27.6	31.8
6+	13.2	13.6	13.4	18.2	18.2	18.5	29.3	27.7	30.2	15.4	16.0	16.3	20.6	20.8	21.3	31.4	30.0	33.9
7+	13.1	13.3	13.0	18.5	18.5	18.8	32.2	30.7	32.8	15.4	16.1	16.2	21.2	21.4	21.8	34.0	32.8	36.4
8+	13.1	13.0	12.9	19.1	18.9	19.3	35.8	34.3	35.4	15.5	15.9	16.2	21.9	21.8	22.5	36.2	35.4	38.8
9+	12.8	12.8	12.9	19.4	19.4	19.8	38.5	38.3	38.1	15.6	15.6	16.5	22.6	22.0	23.6	38.1	37.3	41.4
10+	12.8	12.5	13.0	20.0	20.1	20.2	40.6	41.6	40.9	16.1	15.2	16.8	23.4	22.3	24.7	40.0	38.3	42.8
11+	12.8	12.1	13.1	20.3	20.3	20.2	41.2	43.4	43.8	16.4	15.3	16.8	23.6	23.2	25.0	40.5	39.4	42.1
12+	12.3	11.4	12.8	19.7	20.0	19.6	40.0	43.8	46.0	16.7	15.8	16.7	23.8	24.7	25.3	40.6	40.9	40.6
13+	11.4	10.5	12.1	18.4	19.2	18.3	37.4	43.2	48.2	17.4	15.9	17.1	24.2	25.7	26.2	40.9	41.5	40.1

C=Caucasian; SA=South Asian; AC=African-Caribbean

Table 7.5: Tabulated BMI (kg/m^2) centile values by exact age in boys and girls

	Boys									Girls								
	2nd centile			50th centile			98th centile			2nd centile			50th centile			98th centile		
Age	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC
5+	12.6	13.1	13.0	15.3	14.8	15.5	21.1	17.7	21.5	12.8	12.5	13.1	15.1	14.4	15.7	19.5	18.3	21.9
6+	12.7	12.5	12.7	15.5	14.7	15.5	22.0	19.2	22.5	12.7	12.2	12.8	15.3	14.4	15.8	20.9	19.6	23.3
7+	12.9	12.2	12.7	15.9	14.9	15.7	23.4	21.4	23.7	12.7	12.2	12.8	15.7	14.9	16.2	22.8	21.4	25.0
8+	13.2	12.3	12.9	16.4	15.5	16.2	25.3	24.0	25.2	12.9	12.3	12.9	16.3	15.4	16.9	25.0	22.9	26.6
9+	13.5	12.4	13.3	16.9	16.1	17.0	27.3	26.6	26.8	13.1	12.3	13.1	16.9	15.9	17.8	27.0	24.2	28.3
10+	13.9	12.6	13.9	17.6	16.9	17.8	29.0	28.6	28.7	13.5	12.3	13.5	17.5	16.4	18.7	28.9	25.4	29.8
11+	14.3	12.9	14.3	18.3	17.6	18.4	29.9	29.6	30.4	13.7	12.6	13.7	18.0	17.3	19.2	30.0	27.1	30.5
12+	14.7	13.1	14.5	18.8	18.3	18.8	30.1	30.1	31.6	14.2	13.1	14.0	18.6	18.5	19.7	30.8	29.0	30.9
13+	15.1	13.3	14.6	19.3	18.8	18.9	30.1	30.5	32.6	14.8	13.4	14.7	19.4	19.2	20.5	31.6	30.3	31.4

C=Caucasian; SA=South Asian; AC=African-Caribbean

Table 7.6: Tabulated WC (cm) centile values by exact age in boys and girls

	Boys									Girls								
	2nd centile			50th centile			98th centile			2nd centile			50th centile			98th centile		
Age	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC	C	SA	AC
5+	46.9	45.6	46.8	52.5	50.5	52.5	64.5	58.3	63.1	46.2	44.3	46.9	51.7	49.0	52.7	60.6	59.4	65.2
6+	47.8	45.6	47.2	54.1	51.5	53.8	68.4	62.2	66.7	46.8	44.7	47.2	53.1	50.5	54.1	64.9	63.6	69.9
7+	48.8	46.2	47.9	55.9	53.2	55.1	73.3	67.6	70.8	47.5	45.7	47.6	54.6	52.6	55.7	70.3	68.4	74.7
8+	49.9	47.1	49.0	57.8	55.4	57.1	79.1	74.4	75.5	48.3	46.5	48.3	56.1	54.5	57.6	76.4	72.5	79.2
9+	51.1	48.3	50.5	59.9	57.9	59.4	85.0	82.2	80.5	49.4	47.0	49.7	58.0	56.1	60.2	82.6	76.2	83.6
10+	52.5	49.9	52.0	62.0	60.7	61.8	90.4	90.1	85.4	51.2	47.5	51.4	60.5	57.7	62.7	88.7	79.7	86.9
11+	54.2	51.4	53.3	64.3	63.4	63.9	94.2	96.2	89.7	52.6	48.4	53.1	62.3	59.9	64.8	92.5	83.8	88.6
12+	56.1	52.9	54.7	66.7	65.8	65.9	96.7	100.8	93.4	53.9	50.1	55.1	63.8	62.7	66.7	94.5	88.4	89.3
13+	58.3	53.9	56.5	69.3	67.4	68.0	98.8	104.3	98.1	55.2	51.1	57.7	65.5	64.6	69.1	95.5	91.4	89.8

C=Caucasian; SA=South Asian; AC=African-Caribbean

7.5 Discussion

The purpose of this study was to generate centile curves for anthropometric and BIA derived variables in children from Caucasian, SA and AC backgrounds. Centile curves were generated for anthropometric measures of whole and regional body fatness including BMI and WC respectively and BIA derived measures including Ht^2/Imp and Impedance. This was not for practical purposes but to examine the theoretical basis for variations in the curves between the ethnic groups.

This is the first study to provide an insight into the pattern of gender and ethnic specific centile curves for BIA derived variables. However, these centile curves have not been developed for the purpose of clinical or practical use but to demonstrate that variations in indirect measures of body composition actually exist between children of different ethnic groups. Furthermore, although the generation of ethnic specific centile curves for BMI and WC in the UK remains a matter of debate, this study has presented preliminary findings which demonstrate differences between ethnic groups in both measures. However, centile curves for WC and BMI generated in this study cannot be used for clinical or practical use as the generation of reference centile curves requires a reference sample that is representative of the population. It is also recommended that the reference sample consist of a minimum sample size of 50 children per age group in both sexes and all ethnic groups (T. Cole, personal communication). These criteria were not met by this study firstly because this opportunistic study sample was recruited from selected boroughs within London and thus from a geographical perspective, the sample would not be considered to be representative of the UK Caucasian, SA and AC childhood population. Secondly, sample sizes of the older age groups were smaller than the recommended number. Nevertheless, reference centile charts have been developed using smaller sample sizes as exemplified by the Project Heartbeat which developed %BF references for American children using a sample size of 541 children (278 boys, 263 girls) aged between 8.5 and 17.5 years [534].

The single raw measurement obtained from the BIA technique is Impedance. The centile curves on Impedance indicate a decreasing trend with increasing age in both boys and girls. Furthermore, Impedance was greater in girls than in boys and in SA

children than in their Caucasian and AC counterparts. These findings suggest that that the opposition to the flow of current in the body decreases with age and is greater in girls and in SA children than in their respective counterparts. The significance of these findings is unclear but would suggest some fundamental differences in conductance between genders and ethnic groups, relating back to the fundamental differences in body composition for age.

The age-related increase in Ht^2/Imp would suggest that the volume of TBW (FFM) increases with age in both boys and girls. Secondly, the greater Ht^2/Imp in boys than in girls would suggest that the former have a greater volume of TBW (FFM). Ethnic differences in Ht^2/Imp were apparent across the entire age range with AC boys and girls having a greater Ht^2/Imp than their Caucasian and AC counterparts. A similar finding was also observed in Chapter 6 where SA boys had a significantly lower Ht^2/Imp than Caucasian ($p<0.001$) and AC boys ($p<0.001$) whereas AC girls had a significantly higher Ht^2/Imp than SA ($p<0.001$) and Caucasian ($p<0.001$) girls. These findings suggest that AC boys and girls appear to have a greater volume of TBW (FFM) compared with their Caucasian and SA counterparts. Conversely the findings of this study and that of Chapter 6 appear to suggest that generally, SA boys and girls had a lower volume of TBW (FFM) than their Caucasian and AC counterparts. Although Ht^2/Imp has shown to be a strong predictor of TBW [534], it is important to remember that this relationship is based on the assumptions that the body is a perfect cylinder with constant specific resistivity [298]. However, application of these assumptions to the human body is known to be imperfect given that it has a complex geometric shape and is made up of components that vary in their tissue composition, hydration levels and electrolyte concentration [288]. Thus, although this study has shown that variations do exist between ethnic groups in the volume of TBW (or FFM), these findings need to be interpreted with caution.

The prediction of %BF from the Tanita BC-418MA is based on regression equations (undisclosed by manufacturers) that include body weight and height, gender, age and the impedance reading. Ethnic-specific variation in the age-related change in %BF was evident in both boys and girls at all centiles although a clearer difference between the ethnic groups was apparent in girls. Generally, predicted body fatness was found to be greatest in AC children and lowest in the SA children. Furthermore, the age related change in %BF in children in this study was similar to that of boys and

girls in the UK references. Also observed in this study was the increase in %BF to the age of 11 years in boys and 10 years in girls after which it decreased in the former groups and continued to rise in the latter.

Whether this study provides a true representation of the ethnic variation in the age-related change in %BF remains unclear because of the limitations associated with using this technique to explore variations in body fatness between ethnic groups. Firstly, estimation of %BF by the BC-418 system is based on prediction equations developed from Caucasian children which have not been validated in children from non-Caucasian backgrounds [307]. Secondly, factors such as differences in limb length, fat distribution and hydration of FFM between ethnic groups can also affect the accuracy with which body fatness is predicted using BIA (discussed in greater detail in Chapter 6) [288,358,516]. However, confidence in the findings of this study could be drawn from the fact that %BF in both genders and all ethnic groups was shown to follow an age-related pattern that was generally similar to that of the UK Caucasian references for %BF [308]. Despite these similarities, differences in the absolute values of %BF between the UK Caucasian children and children in this study were evident with values at all centiles being greater in Caucasian and AC children. SA children also tended to have greater absolute values of %BF than the UK Caucasian children with the exception of the age between 9 and 13 years in boys at the 2nd centile and girls at the 2nd and 50th centile where %BF was lower (Table 7.4). Greater body fatness in SA than in Caucasian children has also been reported by other studies [525,527].

Despite there being variation in the age-related change of BMI and WC between the ethnic groups, a similar overall increase across the entire age range was observed in all the groups. This pattern of change has also been described in the UK BMI and WC references for children aged between 5 and 13 years [308,342]. As expected from the rise in the prevalence of general and regional overweight and obesity [327], absolute values of WC were greater in Caucasian and AC children in this study than in the UK WC references. SA boys tended to have higher absolute values at majority of the ages whereas girls tended to have lower values than the UK reference.

In summary, this study has shown that children from the three ethnic groups vary in their age-related change in direct and indirect measures of body composition. This

variation was observed in both boys and girls at the 50th centile as well as the 2nd and 98th centiles. Although the centile curves developed for this study may have little practical use at this stage, they provide important information that strengthens the argument for the development of ethnic specific references in measures of body composition.

Chapter 8: Conclusion

8.1 Summary of the chapters

The rise in the prevalence of childhood overweight and obesity over the last two decades has triggered a rapidly growing interest in this subject. Consequently, trends in childhood obesity are being monitored more closely on a national as well as global basis. In the UK, national surveys such as the NDNS, HSE and more recently the NCMP and smaller scale studies have reported a common finding of increasing prevalence of overweight and obesity in children and adolescents. Greater research effort in this subject has also provided a better understanding on the aetiology and negative consequences of obesity and on its treatment/management. It has also become clear that the prevalence of overweight and obesity and its related morbidity is not evenly distributed across the UK population and it varies between genders, age groups, socioeconomic groups and ethnic groups. This has triggered an increased level of research into the aetiological factors that contribute to this uneven distribution in the prevalence of overweight and obesity. Furthermore, questions have also been asked on how appropriate and accurate BMI is as a method of excess body fatness in these groups of the population. Thus the concept of this thesis has evolved from the gaps in the knowledge firstly regarding the aetiological factors contributing to variations in the prevalence of overweight and obesity between socioeconomic groups. Secondly, given that ethnicity-related variation in body fatness has largely been examined using the BMI, it was deemed essential to obtain a more through understanding of this variation using better measures of overweight and obesity (%BF and WC).

This thesis comprised of four studies, each of which investigated a unique aspect of obesity and body composition-related research. In the first of these studies (Chapter 4), the aim of the study was to compare three measures of overweight and obesity including BMI, whole body fatness and upper body fatness in two contrasting socio-economic populations of children using UK references. It was also the first study to utilize BIA technology to explore variations in body fatness between children from contrasting income background. The findings of this study demonstrated key differences between children from different income groups with respect to their age-

specific body measurements. Firstly it was demonstrated that in addition to greater body weight-for-age, shorter height is a second (and likely a more significant) contributor to the greater BMI, BMI z-score and overweight/obesity prevalence in lower income children. Although the reasons for these differences are not completely clear, it was concluded that overweight/obese children from a population group considered 'lower income' are overall shorter, heavier and have greater overall body fatness compared with equivalent children considered to be from a 'higher income' background. This observation of shorter height in the lower income group was thought to support the phenomenon of height growth limitation proposed recently in a study on Scottish children. This phenomenon is thought to reflect sub-optimal nutritional experience at some stage of (early) development and has been a feature of some populations in developing countries. However the excess body fat accumulation suggests, paradoxically, that energy availability and energy intake at the time of data collection was not compromised and on the contrary had been in excess of requirements.

This study also showed that central body fatness assessed using anthropometric measures was greater in overweight/obese than in non-overweight/obese children. Secondly, the greater height-for-age in the overweight/obese children compared to non-overweight/obese children observed in this study further supports the findings of previous studies which have also reported on the colinearity between obesity and increased height.

The study in chapter 5 was conducted in view of the increased implementation of weight management programmes for obese children. The aim of this study was to examine changes in a range of measures of body composition and body fat distribution and evaluate these measures in a paediatric weight management context. This study showed that despite having some practical advantage, BMI was not able to reflect the underlying body compositional changes resulting from the intervention in growing obese children given that these changes are determined by change in fat mass as well as fat-free mass. Given the limitations of using BMI in this context, it was proposed that BIA (Tanita BC-418MA) be introduced as an alternative method or adjunct to BMI because of its practical advantages and because it is able to highlight the body compositional changes underlying weight change in growing children.

Furthermore, this study showed that WC was a more sensitive indicator of change in fat mass than BMI.

Chapter 6 explored the variation in whole body and regional body composition in children aged 5 to 16 years from Caucasian, South Asian (SA), African-Caribbean (AC) and Mixed Race (MR) backgrounds. Body composition was characterized using a range of anthropometric and BIA-derived raw and predicted measures. It was found that understanding ethnicity-related variations in body composition is a complex subject given that firstly there are a large number of measures with which this can be achieved. Secondly, the limitations of employing the various measures to explore differences in body fatness between ethnic groups and the implications on interpretation were also discussed. More specifically, the limitations of using BMI in this context were considered. These include its residual correlation with stature in children despite being adjusted for height and inability to account for inherent differences in body proportions and body composition between ethnicities. This can result in inaccurate interpretations of variations in body fatness between ethnic groups. Conversely, BIA offered an alternative, albeit predictive method of assessing fat mass and fat-free mass and therefore a more objective indication of variations in body composition between the ethnic groups.

In chapter 7, gender- and ethnic-specific smoothed centiles were developed for anthropometric measurements including WC (cm) and BMI (kg/m^2) and BIA-derived variables including % Body Fat, whole body impedance (Ω) and $Height^2/Impedance$ (cm^2/Ω). Centile curves for each of these measures were developed for Caucasian, SA and AC boys and girls aged 5 to 13 years using the LMS Chartmaker Pro software. The curves generated in this study showed that children from the three ethnic groups varied in their age-related change in the direct and indirect indices of body composition. Despite having little, if any practical utility at this stage, the findings of this study provide preliminary evidence in support of further investigation into the development of ethnic-specific references in measures of body fatness.

In summary, the studies in this thesis have shown that ethnicity- and income-related variations in measures of body dimensions and composition exist in children and adolescents in the UK. A key finding of this thesis was the variation in height between children from different income and ethnic groups. These findings have also been

observed in larger scale national surveys. Although the aetiological factors determining the variation in height remains unclear, both environmental and genetic factors are thought to be involved. Further work is needed to explore the impact of these findings especially in relation to obesity-related morbidity.

8.2 Limitations of the studies in this thesis

The limitations with employing BIA technology to assess body composition in a community setting and adolescents as well as in non-Caucasian groups have been discussed. The accuracy of the BIA measurements in this thesis may have been affected by two factors. Firstly, the BIA system used namely the Tanita BC-418MA has not to date been validated in non-Caucasian children. This limitation has been acknowledged and addressed throughout this thesis. In Chapter 5, it was argued that it is the precision of the BIA system that is likely to be more useful than the accuracy, given that the change in measurements are more relevant for evaluating a paediatric weight management intervention than the actual values at the start and end of the intervention. This does not suggest that the accuracy of body composition measures is not important in this context. The accuracy of these measures is deemed to be important especially if it is necessary to identify whether a subject/patients' position in a classification system has changed e.g. from overfat to normal fat. However, given the current lack of practical assessment techniques such as the BC-418MA that have been validated in non-Caucasian groups and the limitations with using BMI in this context, it is advisable to depend on the precision of the BIA system until further work has been carried out in validating this technique in other ethnic groups.

It was also recognized in Chapters 6 and 7 that the predicted measures of body composition obtained using the BC-418MA were unlikely to be accurate in the non-Caucasian groups. This limitation was addressed by comparing raw BIA-derived measurements including whole body impedance and Ht^2/Impedance (Impedance index) to explore variations between the ethnicities as a group and in their age-related change. Although these measures may not have any practical utility and their significance in terms of body composition is not clear, these measures show that there is a fundamental difference in conductance between ethnic groups. Furthermore, addressing this limitation has highlighted the need for practical

assessment techniques that are able to predict body composition accurately in children from all ethnic groups.

A second limitation with using BIA to assess body composition, as discussed in Chapter 3, is that a number of factors known to affect BIA measurements were not controlled for in this thesis. A number of studies have shown that BIA measurements are affected by hydration status, food and fluid intake and whether exercise has been performed prior to the measurement. Nevertheless, the evidence showing the direction and degree of change in the impedance and %BF reading as a result of a change in these factors remains rudimentary. Thus there is currently no consensus on a standardized protocol which lists the conditions in which the BIA measurement should be conducted. Including a standardized protocol regarding fasting and fluid intake as part of the data collection process of this thesis would have been difficult given that measurements were not conducted in a controlled environment. All measurements were conducted at the schools convenience and so it was impossible to incorporate a standardized time i.e. 2 hours after lunch or 2 hours after the start of school during which these measurements were collected. Thus it is important to recognize that this limitation may have lowered the accuracy of the body composition readings although the extent to which accuracy may have been affected has not been accounted for in this thesis.

This thesis showed that central body fatness examined using anthropometric measures such as WC and WHtR varied between ethnic and income groups. Secondly, overweight/obese children and adolescents were found to have greater central fat distribution than their non-overweight/obese counterparts. Additionally, a change in central body fatness was observed in children who had participated in a weight management intervention. Although WC is known to be a good predictor of abdominal fatness in children, it is important to note that it does not distinguish between the two compartments of abdominal adipose tissue- IAAT and SAAT. Thus the variations in central body fatness observed between the aforementioned groups could be reflecting variations in either one or both of the abdominal adipose tissue compartments. Thus the findings of this thesis do not indicate which compartment of abdominal fatness varied between the groups (ethnic and income) or changed at the end of the weight management intervention. Specific information regarding the abdominal adipose tissue compartment can only be obtained using imaging

techniques such as MRI and CT. However, the accessibility and availability of these techniques in non-clinical settings and in large-scale epidemiological studies is limited. Thus WC is more commonly used as a proxy measure of abdominal fatness. Additionally, WC has been associated with a number of adverse metabolic risk factors in children and adolescents and is therefore a useful and convenient indicator of variation in obesity related risk between these groups. Furthermore, confidence in the findings on variations and changes in central fat distribution described in this thesis can also be drawn from the fact that intra-observer error of WC measurements, as described in Chapter 3, was low.

Another limitation of this study relates to the extrapolation of the findings in Chapters 4, 6 and 7. As described earlier, the study sample in these chapters was an opportunistic sample of schoolchildren aged between 5 years and 16 years that were recruited from schools primarily within inner London. Given that the sample was confined to a geographical area within the UK, it remains unclear to what extent the sample in this study is representative of the UK Caucasian, SA, AC and MR children. It is possible that the sample in this study is representative of children living in a deprived area within inner London and so the findings may only be extrapolated to children living in similar areas in the UK. Thus, the ethnicity-related and income-related variations in body composition discussed in this thesis could be specific to children aged between 5 and 16 years living within a geographical area in London.

Given that a fundamental aspect of this thesis was to collect anthropometric and body compositional measurements from children and adolescents, limiting factors such as measurement error and sample size which can affect the accuracy of the findings and consequently the quality of a study were addressed during the planning stages of this thesis and have been discussed in detail in Chapter 3.

In summary, it is felt that all the limitations described above were acknowledged and addressed at various stages. However, there are still some limitations that are inherent to the studies in this thesis which remain unaddressed and these include the cross-sectional nature of the data collected (with the exception of Chapter 5) and the narrow age range of the study sample which was between 5 and 16 years. Given that these limitations have not been addressed, it remains questionable whether the income-related and ethnicity-related variations in anthropometric and body

compositional measures observed in this thesis follow a similar pattern throughout the infant, childhood and adolescent age span. It was not possible to address these limitations within the time period and funding allocated for this thesis as this would have required a longer study period and a greater financial budget both of which were not within the control of the author.

8.3 Future research

In view of the limitations associated with using the current equations in the BC-418MA to predict FM and FFM in children from non-Caucasian backgrounds, it has become clear from this study that there is a need for practical methods such as the BC-418MA to be validated in children from minority ethnic groups (most likely through measurement of total body water using D_2O). Furthermore, variations between ethnic groups in the age-related change in body composition suggest a need for ethnic specific centile charts to be developed. Put together, these will instill a robust assessment system of monitoring excess body fatness in children from differing ethnic groups. This is particularly relevant as one of the governmental strategies of tackling childhood obesity in the UK is to monitor prevalence of overweight and obesity in schoolchildren on a regular and permanent basis. As part of this monitoring exercise, variation in the prevalence of overweight and obesity between ethnic groups is also being examined. This clarifies the urgent need for a better system of identifying excess body fatness in children from all ethnic groups.

Assessing body composition using techniques that have been validated in non-Caucasian children together with ethnic-specific centiles is also important when considering that obesity-related risk varies between ethnic groups. Assessment on this basis will avoid misclassifying children and thereby avoid missing those at risk and unnecessary intervention on children not at risk. This is beneficial not just for monitoring purposes but for economic purposes and for ensuring that healthcare resources are allocated appropriately.

There is another side to this argument which claims that ethnicity is not a biological term and that individuals who differ by ethnic group do not necessarily vary in their biological/genetic make up. It has been argued that ethnicity is based on a social construct and so individuals who are classified using this system differ on the basis of

culture, heritage and national origin. Thus it can be argued that ethnicity related differences are a function of environmental differences and therefore do not warrant the need for assessment tools that are validated in all ethnic groups and the development of ethnic specific centile charts.

As highlighted in this thesis, it is important to move on from using proxy measures of body fatness such as BMI to using assessment tools that provide a more objective measure of body composition. However, these assessment tools also need to be practical so that they can be used in the clinical setting as well as in an epidemiological context where large numbers of subjects can be measured. The BC-418MA meets these criteria as it provides a more objective means of assessing the two compartments of body mass even though it remains a predictive method. However, more work still needs to be undertaken to validate this system in non-Caucasian groups and develop a standardized protocol that can be practically adhered to in a variety of settings.

8.4 Final thoughts

We are in an unprecedented time in human history where the majority of the population are now overweight or obese. The biggest global challenge for the first half of the 21st century is to find how to reverse this epidemic. There is no precedent for this challenge and as scientists and clinicians working in this field we still have a major role to play. As proposed by the Foresight report "it is important that the government take the lead on tackling this issue and that progress will be enhanced by stimulating multi-section, multi-level action within and beyond the public health profession". At the same time more needs to be known about the variation between populations groups with respect to obesity risk and its management. The work presented in this thesis has contributed in some way to addressing this latter issue but more work remains to be done at all levels before we can begin to see a favourable outcome in obesity prevalence and its social, economic and health burden.

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Appendix

Appendix A: Template information sheet and consent form

SCHOOL HEADED NOTEPAPER

Dear Parent/ Carer

THE SCHOOL has been approached to help London MET University and the Institute of Health Research & Policy to find out about the health and well-being of school children aged 5 to 16 years. This will help your school improve the health of the children and help them to meet the National Healthy School Standard.

I am writing to ask your permission for your child to take part in a small piece of research involving measuring their height, weight, waist circumference and body composition.

The measuring time will take about 2 minutes. Some of the time your child would be standing barefoot on a special set of scales to weigh them. These scales, which are completely safe, will then send a tiny current through the body to measure their body weight and how much their body weight is made of fat and fat free mass (muscle and bone).

All measurements will be taken and recorded in strict confidence. A copy of the results will also be sent to you if you wish.

I would be very grateful if you would sign the consent form below and return it to me by..... I hope you will agree to give your child and an opportunity to help the school in such an important project.

Yours sincerely,

Head teacher / Head of Science/ Head of PE

CONSENT FORM:

NAME..... TUTOR GROUP.....

I consent for my child to participate in this project YES / NO
I would like a copy of the analysis YES / NO

Appendix B: Ethnicity classification

1 = WHITE

1A = English
1B = Scottish
1C = Welsh
1D = Irish
1E = Traveller of Irish heritage
1F = Albanian
1G = Greek
1H = Turkish
1I = Turkish Cypriot
1J = White Eastern European (Russian, Latvian, Ukrainian, Polish, Bulgarian, Czech, Lithuanian, Montenegran, Romanian
1K = White Western European (Italian, French, German, Spanish, Portuguese, Scandinavian)
1L = White Other
1M = Gypsy/Roma

2 = ASIAN OR ASIAN BRITISH

2A = Indian
2B = Pakistani (Mirpuri, Kashmiri, Other Pakistani)
2C = Bangladeshi
2D = Any other Asian Background (African Asian, Kashmiri Other, Nepali, Sinhalese, Sri Lankan Tamil, Other Asian)

3 = CARIBBEAN

4 = BLACK OR BLACK BRITISH

4A = BLACK AFRICAN (Angolan, Congolese, Ghanaian, Nigerian, Sierra Leonian, Somali, Sudanese
4B = OTHER BLACK AFRICAN (South African, Angolan, Zimbabwean, Ethiopian, Rwandan, Ugandan
4C = ANY OTHER BLACK BACKGROUND (Black European, North American, Other Black)

5 = MIXED/DUAL BACKGROUND

(White and Black Caribbean, White and Black African, White and Asian, Any other white background)

6 = CHINESE

(Hong Kong, Malaysian, Singaporean, Taiwanese)

7 = OTHER ETHNIC GROUP

(Afghan, Arab other, Egyptian, Filipino, Iranian, Iraqi, Japanese, Korean, Kurdish, Latin/South/Central American, Lebanese, Libyan, Malay, Moroccan, Polynesian, Thai, Vietnamese, Yemeni)

Appendix C: DCSF Classification system

Broad category	2003 codes	Broad category	2003 Codes
	WHITE		**MIXED BACKGROUND**
WBRI*	British	MWBC*	White And Black Caribbean
WENG	English	MWBA*	White And Black African
WSCO	Scottish	MWAS*	White And Asian
WWEL	Welsh	MWAP	White And Pakistani
WOWB	Other White British	MWAI	White And Indian
WIRI*	Irish	MWAO	White And Any Other Asian Back
WIRT*	Traveller Of Irish Heritage	MOTH*	Any Other Mixed Background
WOTH*	Any Other White Background	MAOE	Asian And Any Other Ethnic Group
WALB	Albanian	MABL	Asian And Black
WBOS	Bosnian-Herzegovinian	MACH	Asian And Chinese
WCRO	Croation	MBOE	Black And Any Other Ethnic Group
WGRE	Greek/Greek Cypriot	MBCH	Black And Chinese
WGRK	Greek	MCOE	Chinese And Any Other Ethnic Group
WGRC	Greek Cypriot	MWOE	White And Any Other Ethnic Group
WKOS	Kosovan	MWCH	White And Chinese
WITA	Italian	MOTM	Other Mixed Background
WPOR	Portugese		
WSER	Serbian		
WTUR	Turkish/Turkish Cypriot		
WTUK	Turkish		
WTUC	Turkish Cypriot		
WEUR	White European		
WEEU	White Eastern European		
WWEU	White Western European		
WOTW	Other White		
WROM*	Gypsy/Roma		

Broad category	2003 codes	Broad category	2003 Codes
ASIAN		**BLACK**	
AIND*	Indian	BCRB*	Caribbean
APKN*	Pakistani	BAFR*	African
AMPK	Mirpuri Pakistani	BANN	Angolan
AOPK	Other Pakistani	BCON	Congolese
AKPA	Kashmiri Pakistani	BGHA	Ghanaian
ABAN*	Bangladeshi	BNGN	Nigerian
AOTH*	Any Other Asian Background	BSLN	Sierra Leonian
AAFR	African Asian	BSOM	Somali
AKAO	Kashmiri Other	BSUD	Sudanese
ANEP	Nepali	BAOF	Other Black African
ASNL	Sinhalese	BOTH*	Any Other Black Background
ASLT	Sri Lankan Tamil	BEUR	Black European
AOTA	Other Asian	BNAM	Black North American
		BOTB	Other Black
CHINESE		**OTHER ETHNIC BACKGROUND**	
CHNE*	Chinese	OOTH*	Any Other Ethnic Group
CHKC	Hong Kong Chinese	OAFG	Afghanistani
CMAL	Malaysian Chinese	OARA	Arab
CSNG	Singaporean Chinese	OEGY	Egyptian
CTWN	Taiwanese	OFIL	Filipino
COCH	Other Chinese	OIRN	Iranian
		OIRQ	Iraqi
		OJPN	Japanese
		OKOR	Korean
		OKRD	Kurdish
		OLAM	Latin American
		OLEB	Lebanese
		OLIB	Libyan
		OMAL	Malay
		OMRC	Moroccan
		OPOL	Polynesian
		OTHA	Thai
		OVIE	Vietnamese
		OYEM	Yemeni
		OOEG	Other Ethnic Group

Appendix D: Tanita BC-418MA print out

TANITA (27)
BODY COMPOSITION
ANALYZER
BC-418 YR1P

06 DEC 2005 13:31

BODY TYPE	STANDARD
GENDER	MALE
AGE	7
HEIGHT	120 cm
WEIGHT	25.9kg
BMI	18.0
BMR	4690 kJ
	1121kcal
FAT%	24.1%
FAT MASS	6.2kg
FFM	19.7kg
TBW	14.4kg

WC=55.9cm

IMPEDANCE	
Whole Body	742 Ω
Right Leg	301 Ω
Left Leg	301 Ω
Right Arm	403 Ω
Left Arm	412 Ω

Segmental Analysis

Right Leg	
Fat%	32.6%
Fat Mass	1.3kg
FFM	2.7kg
Predicted Muscle Mass	2.7kg
Left Leg	
Fat%	32.6%
Fat Mass	1.3kg
FFM	2.7kg
Predicted Muscle Mass	2.6kg
Right Arm	
Fat%	32.8%
Fat Mass	0.3kg
FFM	0.7kg
Predicted Muscle Mass	0.6kg
Left Arm	
Fat%	34.5%
Fat Mass	0.4kg
FFM	0.7kg
Predicted Muscle Mass	0.7kg
Trunk	
Fat%	18.4%
Fat Mass	2.9kg
FFM	12.9kg
Predicted Muscle Mass	12.5kg

TANITA (29)
BODY COMPOSITION
ANALYZER
BC-418 YR1P

06 DEC 2005 13:29

BODY TYPE	STANDARD
GENDER	MALE
AGE	7
HEIGHT	127 cm
WEIGHT	30.7kg
BMI	19.0
BMR	5121 kJ
	1224kcal
FAT%	21.0%
FAT MASS	6.5kg
FFM	24.3kg
TBW	17.8kg

WC=60.2cm

IMPEDANCE	
Whole Body	614 Ω
Right Leg	265 Ω
Left Leg	268 Ω
Right Arm	345 Ω
Left Arm	328 Ω

Segmental Analysis

Right Leg	
Fat%	31.7%
Fat Mass	1.7kg
FFM	3.7kg
Predicted Muscle Mass	3.6kg
Left Leg	
Fat%	32.1%
Fat Mass	1.7kg
FFM	3.6kg
Predicted Muscle Mass	3.5kg
Right Arm	
Fat%	28.5%
Fat Mass	0.4kg
FFM	1.0kg
Predicted Muscle Mass	0.9kg
Left Arm	
Fat%	29.3%
Fat Mass	0.4kg
FFM	1.1kg
Predicted Muscle Mass	1.0kg
Trunk	
Fat%	13.2%
Fat Mass	2.3kg
FFM	15.0kg
Predicted Muscle Mass	14.4kg

TANITA
BODY COMPOSITION
ANALYZER
BC-418 YR1P

06 DEC 2005 13:12

BODY TYPE	STANDARD
GENDER	MALE
AGE	7
HEIGHT	109 cm
WEIGHT	18.1kg
BMI	15.2
BMR	4201 kJ
	1004kcal
FAT%	18.9%
FAT MASS	3.4kg
FFM	14.7kg
TBW	10.8kg

WC=51.1cm

IMPEDANCE	
Whole Body	784 Ω
Right Leg	325 Ω
Left Leg	312 Ω
Right Arm	443 Ω
Left Arm	445 Ω

Segmental Analysis

Right Leg	
Fat%	27.6%
Fat Mass	0.6kg
FFM	1.6kg
Predicted Muscle Mass	1.6kg
Left Leg	
Fat%	26.9%
Fat Mass	0.6kg
FFM	1.7kg
Predicted Muscle Mass	1.7kg
Right Arm	
Fat%	30.8%
Fat Mass	0.2kg
FFM	0.4kg
Predicted Muscle Mass	0.3kg
Left Arm	
Fat%	30.5%
Fat Mass	0.2kg
FFM	0.4kg
Predicted Muscle Mass	0.4kg
Trunk	
Fat%	14.7%
Fat Mass	1.8kg
FFM	10.6kg
Predicted Muscle Mass	10.3kg

Appendix E: Non-parametric results

Change in variables between initial and final measurements on a whole group (n=95) and gender basis

Variable	Whole group	Males (Z)	Females
	Z	Z	Z
Decimal age (y)	-8.4***	-5.8***	-6.2***
Height (cm)	-7.8***	-5.6***	-5.5***
z-height	-0.9	-0.2	-0.9
Weight (kg)	-1.9	1.0	-1.7
z-Weight	-3.3**	-2.8**	-1.8
BMI (kg/m^2)	-2.8**	-2.7**	-1.5
z-BMI	-4.7***	-3.7	-3.0**
BMI centile	-3.9***	-2.5*	-3.1**
WC (cm) (n=88)	-2.8**	-0.1	-3.6***
z-WC (n=88)	-3.6***	-1.2	-3.9***
WHtR (n=88)	-4.0***	-1.2	-4.2***
%BF	-4.1***	-2.7**	-3.2**
*%BF	-3.6***	-2.1*	-3.2**
z-%BF	-4.1***	-2.5*	-3.3**
*z-%BF	-3.5***	-1.2	-3.1**
FM (kg)	-2.7**	-2.1*	-1.6
*FM (kg)	-2.2*	-1.6	-1.6
FFM (kg)	-5.1***	-3.0**	-4.2***
*FFM (kg)	-4.7***	-2.6*	-4.2***
FMI	-3.8***	-2.8**	-2.5*
*FMI	-3.5***	-2.4*	-2.7*
FFMI	-2.6*	-1.0	-2.6**
*FFMI	-4.8***	-2.6*	-2.6**
Impedance (Ω)	-2.6**	-1.0	-4.3***

*%BF, *z-%BF,*FM, *FFM, *FMI: derived using 3C equation