Body composition measurement in African and Caribbean children and its relationship with morbidity

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A thesis submitted in partial fulfilment of the requirements of

London Metropolitan University

For the degree of

Doctor of Philosophy

June 2015

Statement of originality

I, Eva Amoako-Attah, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in this thesis

Signature ……

Date ……

Abstract

The global increase in obesity prevalence has led to a surge in metabolic disease in both adults and children. Furthermore, the burden of obesity and its related morbidities is not equally distributed across the UK population, with those from minority ethnic groups particularly affected. Effective paediatric epidemiological monitoring and clinical referral requires improved tools for assessing body fatness, and other body composition measures related to metabolic disease are needed. Presently the body mass index (BMI) used to identify overweight and obesity suffers from poor sensitivity and specificity, leading to misclassification of children, especially those from minority ethnic groups. Additionally BMI gives no indication of body fat distribution. Assessment tools specifically for African and Caribbean childhood populations are lacking and the aim of this thesis was to develop a range of assessment tools specifically for this population group.

This thesis comprised four studies. In the first study the equations in the Tanita BC-418 bioimpedance (BIA) system used to predict fat mass (FM) and appendicular skeletal muscle mass (SMMa) were re-validated using dual-energy x-ray absorptiometry (DXA) as the criterion method in a sample of 44 African and Caribbean children aged 5-18 years. FM (kg) and SMMa (kg) were quantified by DXA and linear regression analysis used to produce new equations based on height^2/impedance. The key findings from this study were that BIA generally under-estimated FM and over-estimated SMMa in this population group, irrespective of age and gender.

In the second study, the corrected measures of FM and SMMa were applied to an existing dataset of 1,336 African/Caribbean children aged between 5-16y whose body composition had been measured using the Tanita BC418 system. Percentile charts for %FM, %FFM, SMMa (kg), %SMMa and SMMa/FMM x 100 were generated using the software LMS Chartmaker.

In the third study, blood pressure percentile charts and tables were developed based on data (n, 900) extracted for African and Caribbean children aged 5-18 years from the Health Survey for England data archives, 1991-2008. The centile curves for the anthropometric measures revealed gender and age-related patterns which compared closely to equivalent charts for Caucasian children. Finally in the fourth study, percentile charts and tables for waist circumference (cm) were developed using the same sample population.

The findings from these studies provide the tools and preliminary evidence to support the use of African-Caribbean specific references for body composition and blood pressure measures in children and youths in the UK. The overall conclusion from this thesis indicates that paediatric overweight and obesity varies across different ethnic groups and this variation needs to be considered in the context of obesity surveillance and clinical assessment which themselves are determined by national obesity policy formulation and implementation. These are the first body composition percentile charts for African and Caribbean children living in the UK. These charts should replace BMI charts used for obesity assessment in paediatric and epidemiological settings as they are better tools for assessing overweight, obesity and sarcopenia.

Acknowledgments

I am grateful to all my academic supervisors for their guidance, support and useful discussions throughout my pursuance of the Doctor of Philosophy program. I am greatly indebted to Professor David McCarthy for his clear sense of direction and in-depth discussion of the thesis topic, thus making this work purposeful and of quality. I also owe a debt of gratitude to London Metropolitan University, the Institute for Health Research and Policy for the provision of resources for this work.

I wish to express my profound gratitude to my parents who have tirelessly supported me in my academic pursuits. My heartfelt thanks go to my husband for his love, encouragement and maximum support toward this work. I also thank my children Becky-Ann, Paajo and Samuel for their patience and understanding. My greatest thanks go to my God, Jehovah, who gave me the strength to carry on.

Finally I wish to thank all who in diverse ways assisted me to bring this work to a successful completion.

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Abbreviations

AC	African-Caribbean
ADP	Air Displacement Plethysmography
AIDS	Acquired Immunodeficiency Syndrome
BCM	Body Cell Mass
BMI	Body Mass Index
BP	Blood pressure
%BF	%Body fat
BMD	Bone mineral density
BMJ	British Medical Journal
BIA	Bioelectrical Impedance Analysis
BV	Body Volume
BWL	Brookhaven National laboratory
BWt	Body weight
CHD	Coronary Heart Disease
CM	Compartment model
CDC	Centre for Disease Control
CT	Computerised tomography
CVD	Cardiovascular Diseases
CV	Cone Volume
D_2O	Deuterium oxide
DPA	Dual photon Absorptiometry
DXA	Dual energy x-ray Absorptiometry
DM	Diabetes Mellitus
e	Exchangeable mass
ECF	Extracellular fluids
ECS	Extracellular solids
ECW	Extracellular water
FFA	Free fatty acids
FFM	Fat free mass
FM	Fat mass
HD	Hydrodensitometry
Hg	Mercury

HSE	Health Survey for England
IDF	International Diabetes Federation
IOTF	International obesity task force
ISF	Interstitial Fluid
IVNA	In Vitro Neuron Activation
KS	Kolmogorov-Smirnov
Ln	Lean mass
MRI	Magnetic Resonance Imaging
NHANES	National Health and Nutrition Exercise Survey
Pb	Lead
RF	Radio Frequency
RNA	Ribonucleic acid
SKF	Skinfold
SD	Standard Deviation
SE	Standard Error
SDS	Standard Deviation Scores
SMM	Skeletal Muscle Mass
SPSS	Statistical Package for Social Scientists
TBCa	Total Body Calcium
TBK	Total Body Potassium
TBN	Total Body Nitrogen
TBW	Total Body Water
UK	United Kingdom
UWW	Under Water Weighing
WC	Waist Circumference
WHO	World Health Organisation
Z	Impedance

Chapter 1: Introduction

1.1 Preamble

The World Health Organisation (WHO) predicts that overweight and obesity may soon replace more traditional public health concerns (WHO 2010). The prevalence of obesity has more than doubled in the last 25 years in the UK. In England alone, nearly a quarter of adults and about 10% of children are now obese with a further 20-25% of children being overweight (Canoy and Buchan 2007). The rapid increase in the number of obese people in Britain is a major challenge because over half of the adult population could be obese by 2050 and Britain could be an obese society by 2050 (McPherson et al, 2007).

The United States Department of Health contends that an obese child has 70% chance of becoming an obese adult because childhood obesity could persist into adulthood. Children who are obese are often tested for obesity related morbidities such as hypertension, type 2 diabetes, hyperlipidaemia and fatty liver because they are comparatively more prone to developing these deadly non-communicable diseases (Lau et al, 2007). Black ethnic groups in general are disproportionately affected by obesity. In a US report to describe the differences in the prevalence of obesity, it was discovered that non-Hispanic blacks had an obesity prevalence rate of 35.7% and non-Hispanic whites had a rate of 23.7% (Pan et al, 2009).

A higher risk for obesity and overweight in African and Caribbean children can be explained by the thrifty genotype and phenotype hypotheses. According to the thrifty genotype hypothesis, there are certain unidentified genes which promote fat storage in the body in preparation for a future famine. These genes may be present in some groups of individuals and cause fat deposition in them in expectation of a future famine which

will never happen in their lifetime, causing them to become obese in an obesogenic environment (King and Roglic, 1999). The thrifty phenotype hypothesis postulates that foetal growth retardation can occur when a pregnant woman is exposed to poor nutritional conditions. Such foetuses after delivery grow up with a smaller body size, but in an affluent environment with energy and fat-rich diets these individuals have a greater risk of becoming obese and suffering from obesity related ill-health (Martin and Bateson, 1999). The hypothesis for the developmental origins of adult diseases' often called Barker's hypothesis, named after one of the leading proponents, explains the relationship between reduced size at birth and other diseases known to be associated with obesity such as coronary heart disease and diabetes mellitus (De Boo and Harding, 2006). In accordance with the Barker hypothesis, adverse influences (such as inadequate nutrients) during foetal development which cause foetal growth retardation, can lead to permanent changes in physiology and metabolism which can result in increased disease risk in adult life (Barker et Al, 1986). Lower birth-weight or size, observed in the thrifty phenotypic individuals, has also been proven to be associated with increased insulin resistance and higher fasting insulin concentrations with resulting increased incidence of type 2 diabetes mellitus and coronary heart disease (Veening et al, 2003, Barker et al, 1995). In effect, these two hypotheses bring to light the consequences of intra-uterine growth retardation which leads to small for gestational age foetus.

It has been suggested that both the thrifty genotypic and phenotypic individuals are commonly found among Sub Saharan Africans, South Asians and Native Americans and these populations tend to become obese when introduced to Western diets and environment (King and Roglic, 1999). It is against this background that children from African and Caribbean ethnic minority groups are the target of this research.

Body mass index (BMI) is widely used to assess, classify and monitor excess weight and obesity in children. However BMI does not differentiate between increased body mass in the form of fat, lean tissue or bone (Fortuno et al, 2003). BMI represents the sum of fat-free and fat mass in the body (McCarthy, 2006). BMI does not give the exact amount of fat in one's body although it is the excess fat which causes obesity and drives morbidity and mortality (McCarthy et al, 2006). Furthermore, BMI is not able to indicate the distribution of fat in the body. Visceral fat, which is intra-abdominally located, is particularly associated with obesity related ill-health (McCarthy, 2006).

Bio-electrical Impedance Analysis (BIA) is a reliable tool that can determine body composition and per cent body fat in various parts of the body (Going et al, 2006, Cole et al, 1995). BIA distinguishes between fat-free and fat tissue on the basis of their differential conductance and impedance characteristics (Chumlia and Gro, 1994). Fat-free tissue is bathed in an electrolyte solution making it a good conductor, but fat tissue is a non-conductor given its anhydrous nature. The BIA uses pairs of electrodes attached to the left hand and foot of the subjects. Current is passed through the electrodes and the voltage drop at the proximal electrodes is measured and used to calculate the resistance (in ohms) of the tissue under consideration or the value of the resistance is entered into a regression equation together with anthropometric data such as age, height, weight and gender to calculate body composition (Garrow, 2000). Currently, BIA has been validated predominantly in Caucasian children (Haroun et al, 2009). Hence BIA will be validated against Dual energy X-ray Absorptiometry (DXA) in African and Caribbean children in this study.

The primary aim of this research is to validate the BIA (specifically the BC418-MA Segmental TANITA BIA system) using DXA. A predictive equation to determine fat mass and skeletal body mass would be derived based on gender, age, height, weight and impedance (in ohms) in a group of African and Caribbean children and this equation then applied to an existing dataset of body composition measurements in a sample of African and Caribbean children, to obtain reliable body composition measurement for better classification and monitoring of obesity in African and Caribbean children in the UK. Various body fat mass and skeletal muscle mass percentile charts will be developed specifically for African and Caribbean children to help monitor their body fat or excess weight and obesity.

1.2 Study Rational / Originality

Obesity is a major clinical issue worldwide and the prevalence of obesity in children is a public health problem (Lobstein et al, 2004). In England about 10% of children are obese with a further 20-25% being overweight. It is estimated that prevalence of obesity among children will rise to 15% and beyond by 2025 (Foresight report, 2008).

One health-related goal set by the UK Department of Health is for the nation to be the first to reverse the rising tide of obesity and overweight in the population by ensuring that everyone is able to achieve and maintain a healthy weight by 2020. The initial focus is to reduce the population of overweight and obese children to year 2000 levels (Healthy Weights, Healthy Lives, 2008).

Prevention of obesity in children is essential because obesity is associated with morbidities such as diabetes and hypertension which impair a person's wellbeing and quality of life (Rolland-Cachera et al, 2006). The economic implications and social cost

of obesity is substantial. The NHS costs attributable to overweight and obesity are projected to double to £10 billion per year by 2050 with a wider cost to society and businesses estimated to reach £49.9 billion per year (Foresight report, 2008).

According to the 2001 census, Africans and Caribbeans form 11% of London's population and they are part of a minority ethnic group. Research has shown that ethnic minority groups and socially and economically disadvantaged groups are more vulnerable to becoming obese (Foresight report, 2008). Hence prevalence of excess weight and obesity with its health consequences can be greater for children from African and Caribbean background (McCarthy, 2006).

Morbidity associated with obesity is due to excess fat and the ideal monitoring tool should directly access body fat (Fortuno et al, 2003). Intra-abdominal/visceral fat has been shown to be particularly associated with obesity related ill-health (NICE, 2013). People of black decent tend to have higher intra-abdominal/trunk adiposity (Wagner and Heyward, 2000). Furthermore, African and Caribbean children tend to be taller for their age with longer limb length compared with children from other ethnic groups, which is associated with higher fat-free mass. Hence it is possible that BMI is more of a measure of fat-free mass in these children than it is for fat mass except at extremes of body fatness (McCarthy, 2006).

Hypertension is prevalent and severe in people of black decent compared with other ethnic groups (Charles and Raj, 2003). Overt hypertension maybe rare in children but overweight and obese children can display higher BP values and this is most often carried into adulthood (Lane and Gill, 2004). The association between body

composition and blood pressure in African and Caribbean children will also be determined. The BIA predictive equations derived for African and Caribbean childhood population and percentile curves/charts for African and Caribbean children are original to this project.

1.3 Literature Review

1.3.1 Obesity

Obesity is a clinical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy, and/or increased health problems (Haslam and James, 2005). The World Health Organisation (WHO) predicts that excess weight and obesity may soon replace more traditional public health concerns such as malnutrition and infectious diseases which used to be the most significant causes of ill or poor health (WHO, 2010). In 1997, the WHO formerly recognised obesity as a global epidemic (Caballero, 2007). The World Health Organisation has described obesity as one of the most obvious but mostly neglected public health problems (WHO, 2000).

What Causes Obesity?

Obesity is mainly caused by a combination of dietary energy intake in excess of energy needs, lack of physical activity and genetic susceptibility although a few cases are caused by endocrine disorders, medications and psychiatric illness (Kushner 2007, Adams and Murphy, 2000). A 2006 report identified ten (10) possible contributing factors to the recent increase in obesity (Marantz et al, 2008). These factors include:

(i) Insufficient sleep

(ii) Endocrine disruptions (Environmental pollutants that interferes with lipid metabolism)

(iii) Decreased variability in ambient temperature

(iv) Decreased rate of smoking, because smoking suppresses appetite

(v) Increased medication that can cause weight gain (examples being atypical antipsychotics and steroids)

(vi) Proportional increases in ethnic and age groups that tend to be heavier

(vii) Pregnancy at later age (this causes susceptibility of obesity in children)

(viii) Epigenetic risk factors passed on generationally

(ix) Natural selection for people with high body mass index, and

(x) Assortative mating leading to increased concentration of obesity risk factors (Marantz et al, 2008)

Effects of Obesity

The health effects of obesity fall into two main categories:

(1) Those attributable to the effect of increased fat mass such as osteoarthritis, obstructive sleep apnoea, social stigmatisation leading to psychosocial health problems as a result of societal prejudice against fatness and,

(2) Those due to the increased number of fat cells. These include cardiovascular diseases such as hypertension and myocardial infarction, Type 2 Diabetes Mellitus, certain cancers (such as endometrial, breast, prostate and colon), non-alcoholic fatty liver disease (steatohepatitis), gastro-oesophageal reflux disease, gout and respiratory conditions which impose a growing burden on societies around the world (Haslam and James 2005, Bray 2004).

Obesity reduces life expectancy (Haslam and James, 2005). In the United States, obesity has been estimated to cause an excess 111,909 to 365,000 deaths per year (Haslam and James 2005, Allison et al, 1999) while one million of the deaths in the European Union are attributed to excess weight (Tsigosa et al, 2008). Obesity also creates pro-

inflammatory and pro-thrombotic states in the body predisposing an individual to ill-health (Bray 2004, Shoelson et al, 2007, Dentali et al, 2009).

The economic impact of obesity is also extremely high. These costs include treatment costs, lost productivity and the social costs of premature mortality. Obesity leads to disadvantages in employment (Cummings, 2003) and increased business costs. When compared to their counterparts with healthier weight, obese workers on average have a higher rate of absenteeism from work and take more sick days leading to increasing cost for employers and decreasing productivity (Ostbye et al, 2007). One study on a group of University employees found that people with a body mass index (BMI) of over 40kg/m^2 (i.e. morbidly obese individual), submitted twice as many workers' compensation claims than those whose BMI was 18.5–24.9kg/m^2. These obese individuals also had more than twelve times as many lost work days (Ostbye et al, 2007).

In the UK, the National Health Service cost attributed to overweight and obesity is projected to double to ten billion pounds per year by 2050 (McPherson et al, 2007). The wider cost to society and business is estimated to reach 49.9 billion pounds per year by 2050 (McCormack and Stone, 2007).

Treatment for obesity consists of dietary restrictions and increased exercise (Lau et al, 2007). In extreme cases, medication and surgery may be considered. Dietary programs may produce weight loss over a short term (Shick et al 1998) but keeping this weight off can be a problem and often requires making exercise and lower energy diet a permanent part of one's lifestyle (Tate et al, 2007).

The success rates of long-term weight loss maintenance are low and range from 2% to 20% (Peeters et al, 2003).

1.3.1.1 Childhood Obesity

There is no consensus on a satisfactory cut off point/definition for overweight and obesity in children and adolescents. However, a number of definitions have been proposed as follows:

According to the Centre for Disease Control (CDC US), a child is said to be obese when the body mass index (BMI) is above the 95th percentile for age and sex and overweight when the BMI is between 85th and 95th percentile for age and sex (CDC, 2010). European researchers define overweight in children as BMI at or above the 85th percentile and obese as at or above the 95th percentile of BMI for age and gender (Flodmark et al, 2004). Percentage body fat has also been used to classify childhood obesity. Children with at least 25% to 30% body fat are said to be obese (William et al, 1992, McCarthy et al, 2006). Nonetheless, there is enough evidence to prove that childhood obesity has reached epidemic levels worldwide.

The pandemic of obesity which was once confined to the adult population has penetrated the paediatric age range with signs of rapid escalation (McCarthy et al, 2006). In the last twenty-five years childhood obesity rates have been observed to double, leading to an obesity epidemic that threatens the well-being of both current and future generations (Zhu, 2010). Childhood obesity has reached epidemic proportions in both developed and developing countries worldwide, presenting a clear danger to future global health (Dollman et al, 2005).

In England alone, 9.3% of children in reception are obese and 18.9% at year six are also obese (NCMP, 2013). It is estimated that the prevalence of obesity among children and young people under twenty years of age will rise from current levels of 8-10% to 15% by the year 2025, and by this year, approximately a 25% prevalence rate of overweight has been predicted (Trayhurn, 2007).

In London alone, almost 23% of children entering school are overweight or obese with 10.9% being obese and 12% being overweight. By year 6 of school there is a further increase to 36.3% with 21.6% designated as obese and 14.7% as overweight (NCMP, 2010, Zhu, 2010). For children aged six to ten years (6-10 years) an estimated obesity prevalence of 21% in boys and 14% in girls has been predicted by 2025 (Butland et al, 2008).

Childhood overweight and obesity are known to have a significant effect on both physical and psychological health (Mahshid et al, 2005). Furthermore, obese and overweight individuals increase their risk of developing a wide range of severe chronic medical conditions. These include type-2 DM, hypertension, coronary heart diseases, cerebrovascular accident (stroke), osteoarthritis, chronic inflammatory disorders, depression, digestive disorders and cancers (Kopel, 2007). The proportion of chronic diseases associated with obesity is expected to increase substantially in children (McPherson et al, 2007). This is because these chronic medical conditions which include non-communicable diseases such as hypertension, diabetes mellitus and cancers are considered to be in their “incubation period” during childhood and their major predisposing factor is obesity (Wells, 2008). To illustrate, childhood obesity is likely to continue to adulthood and since obesity predisposes to hypertension (a common chronic

condition which leads to cardiovascular diseases), obese children are particularly at risk of becoming hypertensive as adults (Harding et al, 2008). Indeed, overweight and obese children are two to four times more likely to develop high blood pressure or hypertension than normal weight children (Freeman et al, 1999, Rosner et al, 2000 and Sorof et al, 2004). High blood pressure or hypertension is a major global public health problem and a primary cause of haemorrhagic stroke, hypertensive heart disease and hypertensive kidney failure (Chobanaian et al, 2003).

Considerable advances have been made in detection, evaluation, and management of high blood pressure (BP), or hypertension, in children and adolescents. The long-term health risks for hypertensive children and adolescents can be substantial. Hence, it is important that clinical measures are taken to reduce these risks and optimize health outcomes (National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents, 1996).

The term blood pressure actually refers to two separate measurements: systolic blood pressure which is the highest pressure reached in the arteries as the heart pumps blood out for circulation through the body; and diastolic blood pressure which is the much lower pressure that occurs in the arteries when the heart relaxes to take blood in between beats. If either or both of these measurements is/are above the range found in healthy individuals of similar age and sex, it is called hypertension (Chobanian et al, 2003). The definition of hypertension in children is also based on the normative distribution of blood pressure in health children. Normal blood pressure in children is defined as systolic blood pressure (SBP) and diastolic blood pressure (DBP) that is less than the 90^{th} percentile of sex, age and height. High blood pressure or hypertension in

children is defined as average systolic and/or diastolic blood pressure that is greater than or equal to the 95th percentile for sex, age and height measured on at least three separate occasions (Chobanian et al, 2003).

Until recently, the estimated prevalence of persistent high blood pressure in children was between 1-2%. In a recent study of school aged American children, the prevalence was found to be 4.5% (Androgue and Sinaiko, 2001).

Formerly considered an adult disease, more and more children are being diagnosed with high blood pressure (hypertension) – a condition in which the heart and blood vessels are being overworked (National High Blood Pressure Education Program Working Group on Hypertension Control in Children and Adolescents 1996). An estimated 5 to 10 per cent of children in the United States have high blood pressure which is due to a number of factors including excessive salt intake, obesity, kidney problems or other diseases (Sorof et al, 2004). Studies have shown that for every 1 to 3mmHg rise in blood pressure, a child has a 10 per cent increased risk of developing high blood pressure in adulthood. This subsequently leads to complications like cerebrovascular accident (Stroke), retinopathy, nephropathy and left ventricular hypertrophy (Sorof et al, 2004, Chobanaian et al, 2003).

In addition, childhood obesity increases a child's risk of developing adult type-2 Diabetes Mellitus and its associated complications at an early stage in life (Hanon et al, 2005, Haslam and James, 2005). Type-2 Diabetes Mellitus is a condition where there is a persistently raised blood sugar because of relative reduction and/or resistance of insulin levels in the body which can lead to sudden death, stroke, renal (kidney)

insufficiency, chronic renal failure, amputation of limbs and myocardial infarction (Hanon et al, 2005, Haslam and James, 2005). Until recently, most of the cases of diabetes mellitus among children and adolescents were autoimmune in origin leading to insulin dependent diabetes mellitus (IDDM) in which there is relatively no insulin in the body. Childhood obesity, however, has led to a dramatic increase in the incidence of type 2 diabetes among children and adolescents over the past two decades. This is because obesity leads to insulin resistance which when coupled with the relative deficiency of insulin, causes overt type 2 diabetes mellitus in children and adolescents. Unfortunately, obese children and adolescents who develop diabetes mellitus may experience the micro-vascular and macro-vascular complications associated with this disease at younger ages than individuals who develop diabetes in adulthood. The micro- and macro-vascular complications associated with diabetes mellitus include atherosclerotic cardiovascular disease, stroke, myocardial infarction, and renal failure, retinopathy which could lead to blindness and limb-threatening neuropathy and vasculopathy (Hannon et al, 2005).

What causes childhood obesity?

As with obesity in adults, several different factors have contributed to the rising rates of childhood obesity. Unhealthy diet and decreasing physical activity are the two most important causes that account for the recent increase in obesity rates in children. Obesity is common in people who are least well off or people from a low socio-economic background or class. The causes of obesity in this population are manifold. They include inability to afford healthier foods and/or preference for high fat, high sugar foods as well as a lack of safe and spacious environments for physical activity.

Hence, obesity is prevalent among children from a low socio-economic class (Lieb et al, 2009).

Genetic causes, environmental factors, lifestyle preferences and cultural factors have been found to influence the susceptibility of a given child to an obese-conducive environment (Hill and Peters, 1998). In a small number of cases, medical causes such as hypothyroidism, growth hormone deficiency, leptin deficiency and side effects of medications (example is prolonged use of steroids) also play a role in childhood obesity (Link et al, 2004). In most cases, however, personal lifestyle choices, cultural and environmental factors significantly influence obesity.

Food is more affordable to larger numbers of people considering the fact that the price of food has decreased relative to income. Large size inexpensive children's meals are available at various fast food restaurants. Carbonated soft drink intake has been shown to be associated with high energy intake in children. From 1970 to 1997, the US Department for Agriculture surveys indicated that an increase of per capita consumption of carbonated drinks was associated with a decline in consumption of milk beverages (Putnam and Allshouse, 1999). However, dairy products such as milk beverages consumed two or more times a day could reduce the risk of overweight (Heaney et al, 2002) since a higher intake of dairy servings which are rich in calcium can reduce adiposity or fat deposits in children (Carruth and Skinner, 2001, Skinner et al, 2003). Hence if children are allowed to consume more carbonated soft drinks instead of healthier drinks such as milk beverages, it should be expected that they would accumulate more fat and become predisposed to overweight and obesity.

Additionally, if children are encouraged to lead sedentary lifestyles such as by keeping them indoors, their risk of obesity will be much greater. Some parents inadvertently encourage sedentary lifestyle in their children because they would prefer their children to stay indoors and watch television, play computer games or use the internet rather than play outside unattended due to perceived safety concerns (Gordon-Larsen et al, 2004). Many parents tend to drive their children to and from and school. Such children may also engage in less sport and physical education. They may also be consuming high energy drinks such as sugar–sweetened beverages and eating takeaway meals.

Children may also be encouraged to adopt a sedentary life-style at school. In some schools, physical education curriculums have been designed to create an exercise environment that promotes competition thus preventing several children who lack athletic ability from competing with their peers and enjoying sports and physical education at school. In addition, physical education curriculums should include motor skills such as strength, speed and power which have been shown to improve cardiovascular function and also have long term health benefits (Reybrouck and Mertens, 1997).

Thus, a child's body weight is regulated by numerous physiological mechanisms that maintain the balance between energy intake and energy expenditure. Factors that raise energy intake or decrease energy expenditure can lead to obesity in the long-term. Genetic factors can predispose a child to obesity, but environmental and lifestyle factors underlie the childhood obesity epidemic.

Most health researchers and clinicians agree that prevention is the key strategy for reversing the current pandemic of obesity. In most cases preventive strategies have focussed on diet and exercise. Governments have invested into programs to increase physical activity and on changing the dietary behaviour of individuals as a means to prevent childhood obesity and overweight. Despite these government initiatives it seems that very little impact has been made and prevalence figures do not suggest a reversal in the levels of obesity (Mahshid et al, 2005).

Restriction of television viewing and eating in front of television is important in reducing the current trend of childhood obesity because fast foods or high fat and sugar are most advertised during children's programmes on television and children are the targeted market. If children were prevented from watching these adverts, it would limit their exposure to the huge volume of marketing of energy-dense foods and drinks sold at fast food restaurants. Moreover, less television watching should also mean being more physically active, so parents and carers would replace television watching times with outdoor activities such as walking and cycling (Swinburn and Egger, 2002).

Other intervention strategies which could be considered for prevention of childhood obesity include ensuring built environments have routes which are safe designated walking paths; roads that have designated cycling routes, open public spaces like parks are available for free playing and that there is a provision of safe and inexpensive recreational centres.

Furthermore, there needs to be increasing sports participation, improving and increasing physical education time as well as making use of school health report cards.

The issuing of school health report cards is a recent initiative believed to aid prevention of obesity. It involves issuing report cards to obese children to be given to their parents to make them aware of their children's weight problem. According to a study done in Boston, parents who received health and fitness report cards were more likely to acknowledge that their children were actually obese than parents who did not receive health report cards. Consequently, the former group of parents were said to be more likely to put in place weight control measures for their children than the latter (Chomits et al, 2003).

Food prices have a marked influence on food purchasing behaviour and nutrient intake (Guo et al, 1999). If a small but large enough tax is put on high volume foods of low nutritional value such as soft drinks, confectionery, snack foods and fast foods, it may reduce purchase of the same and hence their effect on people. Such taxes are now applied to certain foods in some parts of USA and Canada (Jacobson and Brownwell, 2000). Food labelling that indicates the nutritional content of foods on sale should be enforced to help consumers make healthy choices.

Finally, it is essential for strategies to prevent childhood obesity to be culturally and ethnically specific. Moreover studies need to look at the most effective strategies of intervention, prevention and treatment.

1.3.1.2 Obesity in people of black descent

Childhood obesity has multidimensional nature and various socio-cultural factors appear to influence it (Dhoble et al, 2008). According to the Center for Disease Control (CDC US) obesity is not only more prevalent among black people than other ethnic groups but they are also more likely to be diagnosed with Type-2 diabetes,

hypertension, and blacks experience higher rates of serious complications from these diseases such as blindness, amputation and end–stage renal disease. Children affected by type 2 Diabetes are almost all obese. Although South Asian children are commonly affected, black children are also at risk (Hanon et al, 2005).

In the US, from 2006 to 2008, the overall estimated age adjusted prevalence rate of obesity was 25%. But a higher prevalence of obesity, which was 35.7%, was observed among non-Hispanic blacks compared with lower prevalence rates of 28.7% and 23.7% observed among Hispanic and non-Hispanic whites respectively. The report further states that, these differences in prevalence rates were noticed to be consistent across all census regions and higher among women than men (CDC, 2008).

According to the CDC report, a number of reasons account for the higher rate of obesity prevalence among the non-Hispanic blacks. First it was observed that compared to the whites, the blacks are less likely to engage in exercise or various forms of physical activities (CDC, 2005). Considering attitudes and cultural norms regarding body weight, it was observed that black women were more satisfied with big body size than white women and as persons satisfied with increased body weight and/or size they were less likely to put in effort to lose weight. In fact, it is known and accepted that some things are not talked about in most black communities and weight is one of them (Millstein et al, 2008).

Furthermore, evidence suggests that neighbourhoods with large minority populations have less access to safe locations for physical activity and resources, which inhibit even simple walking (Ding et al, 2011). These minority groups are mostly low-income

earners and they have less access to affordable, healthful foods because evidence shows that they live in areas where there are fewer chain supermarkets but produce stores which sell healthy foods at relatively higher prices than energy-dense foods (Adler et al, 2009).

A higher risk for obesity and overweight in people of black descent living in the UK could also be explained by the "thrifty genotype" and "phenotype" hypotheses. According to the thrifty genotype hypothesis, there are certain unidentified genes which promote fat storage in the body in preparation for a future famine. These genes may be present in some groups of individuals and possibly responsible for fat deposition in them in expectation of a future famine which will never happen in their lifetime, causing them to become obese in an obesogenic environment (King and Roglic, 1999). Hence the thrifty genotype is a disadvantage in modern times since it manifests itself among other things in the development of obesity and non-communicable diseases (Chakravarthy and Booth, 2004; Neel, 1999).

Additionally, to explain the relatively high prevalence of obesity in migrant populations, it is believed that during migration, there are times of different local stresses such as dietary, ecological, energy or famine and adverse climatic conditions encountered by migrants. It is those populations who survive these situations and reach the new environments, and such ones are most likely the persons with the more thrifty genes and hence when exposed to the obesogenic environment they become obese individuals (Wells, 2006).

The thrifty phenotype hypothesis also postulates that foetal and perinatal growth retardation can occur when a pregnant woman has limited food supply which exposes the growing foetus to poor nutritional conditions in utero. Such in-utero and/or perinatal impaired growth causes such infants after delivery to grow up with smaller body size, a lowered metabolic rate and reduced behavioural level of activity which are adaptations developed in an environment that was chronically short of nutritional supplies. Persons with such thrifty phenotype who later develop in an affluent environment have a greater risk of becoming obese and may be more prone to metabolic disorders such as type 2 diabetes mellitus due to the developed adaptations during foetal life as a result of limited supply of nutrients in utero (Martin and Bateson, 1999; Hales and Barker, 1992).

It has been suggested that both the thrifty genotypic and phenotypic individuals are commonly found among Sub-Saharan Africans, South Asians and Native Americans and these populations tend to become obese when introduced to Western diets and environment (King and Roglic, 1999). Blacks tend not to view obesity as a big problem and black girls are the leading group of children who suffer from obesity (JET, 2005). One study indicated that most black parents are less concerned about dieting to become slim. They have a great preference for high fat food and have unfavourable attitudes about the health benefits of exercise (Dhoble et al, 2008).

Moreover, it has been found that cultural notions contribute significantly to the high prevalence rate of overweight and obesity among people of black descent. These include:

- The making of food a major focus of social and church gatherings where the

type of foods served is often fried or fat rich.

- The issue of food cost. It's cheaper to buy takeaways than to cook meals.
- Exercise is often hindered by cost, unsafe neighbourhoods and time constraints.
- Most black people hardly consider overweight and obesity as health problems but rather consider a fuller figure as an ideal body type, a symbol of high social status and a sign of well-being. Being overweight is seen as being a little heavy or big boned.
- Children are often told you have to eat well to be the same size as your dad or your mum (Mary BF, 2006).

Socially and economically disadvantaged as well as ethnic minority groups are more vulnerable to being overweight and obese (Butland et al, 2008). According to the 2001 UK national census, Africans and Caribbean form just 6% and 5% respectively of the population and therefore they are among the minority ethnic groups in the country. This could indicate that obesity might be a serious problem in these minority groups. Consequently, this research aims to concentrate on African and Caribbean children to ascertain their body fat composition and generate useful data for clinical and epidemiological purposes.

1.3.2 Tools for Measuring Body Fat

There are several methods for measuring body fat. Techniques used to determine body fat in research include underwater weighing (densitometry), bioelectrical impedance analysis (BIA), magnetic resonance imaging (MRI), air displacement plethysonography (ADP) (Bod Pod) and dual energy x-ray absorptiometry (DXA). In the clinical setting, techniques such as skin fold thickness, waist circumference and body mass index (BMI)

are used extensively. These techniques are generally less accurate than those used for research purposes but they are satisfactory to identify risk (Steven, 1995).

As a surrogate marker of intra-abdominal or visceral fat, waist circumference seems to be a more accurate measure for children because it targets central obesity (McCarthy, 2001). Body mass index (BMI), which is a measure of body weight with respect to height, is widely used to assess overweight and obesity in children. Although BMI has been used as a valuable tool in the monitoring of childhood obesity trends, it has numerous disadvantages (Prentice and Jebb, 2001). BMI is defined as weight (kg)/Height^2 (m). BMI just gives a value of how heavy an individual is but does not distinguish between increased body mass in the form of fat, lean tissue or bone and may exaggerate obesity in large muscular children (Fortuno et al, 2003).
BMI represents the sum of fat-free mass and fat mass (McCarthy, 2006) but the pathology associated with obesity is driven predominantly by the excess fat mass, although low skeletal muscle mass is also linked to obesity related morbidity (Fortuno et al, 2003) in the body. Hence the ideal monitoring tool for obesity should be able to directly assess body fat or adiposity (McCarthy et al, 2006).

Furthermore, although BMI is able to correctly identify the fattest children in a given sample because of its high specificity, it can also misclassify large numbers of children as having a raised body fat mass because it is unable to distinguish between an increase in weight due to fat-free mass (such as bone and lean tissue) and gains due to fat mass. Hence epidemiologically, the use of BMI to monitor trends in overweight and obesity in children can be particularly problematic for public health applications such as surveillance of obesity (McCarthy, 2006).

Another significant drawback of BMI is that it does not give any indication of the distribution of fat in the body. Body fat can be central and peripheral in distribution. Centrally distributed fat results from the accumulation of adipose tissue in the peritoneal cavity attached to internal organs including peri-renal, mesenteric and epididymal white adipose tissue, unlike subcutaneous fat which is found under the skin and intramuscular fat interspersed in skeletal muscles. Abdominal adiposity is reflected in an "apple shaped body" and increase in waist size as opposed to peripheral fat which leads to "pear shaped body" (Yusef et al, 2004). Excess intra-abdominal or visceral fat which leads to central fatness has been shown to be particularly associated with obesity-related ill health rather than peripherally distributed fat (McCarthy, 2006). This is because visceral fat releases non-esterified fatty acids into the portal system which have direct effect on hepatic metabolism and this leads to insulin resistance (BMJ, 2005).

Interestingly, there are other body fat measuring and monitoring tools which directly or indirectly assess/predict body fat or adiposity. The Bio-electrical Impedance Analysis (BIA) is a simple, portable and inexpensive tool that can determine body fat mass (Going et al, 2006). Numerous studies have demonstrated the reliability of bio-electric impedance measurement and it has been shown that it provides reasonably accurate estimation of changes in per cent body fat and fat free mass over time (Phillips et al, 2003; Cole et al, 1995).

The Bio-electrical impedance monitor can distinguish or differentiate between fat-free and fat tissues on the basis of their differential conductance and impedance characteristics (Chumlea and Guo, 1994). Fat-free tissue is bathed in an electrolyte

solution and hence is a good conductor whereas fat-tissue being anhydrous is a non-conductor. The BIA system uses pairs of electrodes held in both hands and the individual stands on its flat pedals. A current is passed between the electrodes and the voltage drop is measured at the proximal electrodes, from which the resistance (in ohms) of the tissue under consideration is calculated or the value of the resistance is entered into a regression equation together with anthropometric data including age, height, weight and gender to predict total body water (TBW) then fat-free mass and ultimately total body composition (Garrow, 2000).

Most BIA approaches are reference dependant with prediction formulae at specific electrical current frequencies available for total body water (i.e. intra and extra cellular compartments) estimation (Foster and Lukaski, 1996). It is recommended that stringent measurement conditions are used and prediction formulae must be validated because an important limitation of the BIA is that many underlying assumptions are made and most of them have not yet been adequately explored or known to be accurate. In the standard whole body BIA measurement method, the electrodes are connected to the hands and feet. Traditional BIA methods assume that the body is a geometric model with homogenous components which are uniform in cross-section (Lukaski, 1989; Johnson et al, 1985). However, the typical electrical pathways produced with the BIA, fail to conform to such idealised conditions in humans. As such BIA methods should be used only under appropriately controlled conditions (Heymsfield et al, 1991).

Dual Energy X-ray Absorptiometry (DXA) is a reference laboratory system which provides information on the relative proportions of bone mineral, soft lean tissue and fat. DXA uses very small doses of radiation and is therefore not harmful for use in

human subjects (Garrow, 2000). DXA uses x-rays which are emitted in a narrow beam from a source which travels in a semicircle around the subject and the energy passing through the body is recorded by a detector. Computer software (Windows: bone densitometry software) then reconstructs the pattern of absorbing material within the slice or body part which must have given rise to the absorbed changes in the x-ray transmission as the beam moves round the subject. If serial scans are performed at different levels of the body, data on the total value of the different types of tissue and how these tissues are distributed in different sections of the body can be obtained (Garrow, 2000). Consequently, DXA can provide quantitative estimates of the amount of fat, bone, skeletal and non-skeletal fat-free (soft) tissue as well as information about the spatial distribution of these tissues in an individual's body (Garrow, 2000).

One of the aims of this research is to validate a BIA system for use in Black African and Caribbean children using DXA as the reference method. DXA is suitable for such a validation because it is based on a three compartment model of body composition (shown below) and therefore requires fewer assumptions than methods based on two-compartments (Going et al, 2006).

1.3.3 Composition of the Human Body

The field of human body composition research continues to be an active area in basic sciences and clinical research. Due to advanced technology, precise analysis of a tissue sample can be accurately preformed without any difficulty. However, substantial errors could be encountered when extrapolating the results of a single tissue analysis to

estimate total body composition, hence the need for whole body analysis. In the early 1900s direct chemical analysis of an adult's whole body was limited, although many studies could be done in human foetuses and infants (Givens and Macy, 1933; Fee and Well, 1963). Later on, Elsie Widdowson and co-workers examined both infants and adults (Widdowson et al, 1951, 1964, 1974), while Forbes et al examined mostly adult cadavers (Forbes et al, 1951, 1953, 1956). These studies from the complete dissection of whole human bodies provided information on variations in organ weights but not chemical or molecular makeup of the human body. Other studies have focussed on the determination of total body nitrogen through cadaver assays resulting in data to prove that the chemical composition of the body's various tissues is relatively constant among individuals although not constant from birth (Knight et al, 1985). Information on body composition studies is rapidly accumulating, expanding the knowledge on the biology of the human body. This information is categorised as technical or biological with the former involving methods of body composition determination. These methods include dilution techniques and neutron-activation analysis which are usually based on physical principles and/or other specific features of the technique and sample involved.

As much as the technical and biological categories seem to cover most of the information on body composition knowledge, Heymsfield et al have identified a significant limitation of this categorisation (Heymsfield et al, 1991), in that not all of the rapidly developing volume of information on body composition can be satisfactorily categorised as technical or biological. Reconstruction of human chemical compartments and body weight derived from estimated in vivo elements through the use of neutron-activation analysis suggest that relationships exist between individual as well as different body composition levels. Additionally, some quantitative associations have

been found to exist among different compartments that are in equilibrium. For example, there are mathematical models that describe the relations between different components in healthy subjects such as total body water (TBW)/fat-free body mass = 0.732 (Synder et al, 1984).

1.3.1.1 Body Composition Models

Anthropometric data obtained from measurements of skinfold thickness, body circumferences and lengths at numerous body parts/regions as well as various weight-for-stature indexes have been used to develop anthropometric-based models to predict body composition for all age groups including neonates (Lohman, 1981; Lohman et al, 1988; Brodie et al, 1998). Body compartment/composition models provide the opportunity for a clear definition of the concepts of body composition steady state in which relationships between compartments at similar or different levels exist over a specified time interval. In addition, such models reveal gaps in human composition studies leading to identification of important new research areas. The following section considers the various body compartment models.

1.3.3.2 The Two Compartment Model

The two compartment (2-C) model has been used in body composition studies for over half a century, serving a vital role in body fat assessment and in the evaluation of novel technologies. The basic two compartment model divides the body into two parts: fat and fat-free masses. Direct body fat mass determination has been difficult and if total fat-free mass can be determined then fat mass can be obtained by finding the difference between body weight and fat-free mass. The commonest and earliest 2-C model is based

on measurement of whole body density using the hydrodensitometry/under water weighing (UWW) method (Behnke et al, 1942). Following this, two nuclear-based methods which use ^{40}potassium counting and radioactive/non-soluble isotope water dilution to estimate fat-free mass based on the 2-C model emerged. These methods use the potassium and water content of the body respectively to calculate fat-free mass (Forbes et al, 1961; Pace and Rathbun, 1945). The following constants were derived: 0.732 l/kg/FFM for body water, 68.1 meq/kg for body potassium as relative concentrations for all ages so long as the population under consideration were young healthy Caucasians. However, these constants became impractical to use in body composition calculations if the population involved the very young or very old and in non-Caucasian groups. To overcome these limitations, the 3-C model evolved (Ellis, 2000).

1.3.3.3 The Three Compartment Model

Under the 3-C model the body is divided into fat mass, water content and solids/dry FFM with the assumption that the latter two represents fat free mass. The solids mainly represented body proteins and mineral tissues. The 3-C model approach required that the densities of water, fat and body solids are derived from UWW measurements. This model produced results which showed some improvement over the 2-C model for healthy adults and older children. However it was limited in patients with significantly depleted protein and/or bone mineral mass. It was observed that for such patients the density values for the solids was incorrect and hence fat-free mass as well fat mass calculation using such values would be incorrect and led to the four compartment model (Ellis, 2000).

1.3.3.4 The Four Compartment Model

The 4-C model uses the densities of body water, fat, proteins and minerals. To accurately measure the masses of these body compartments, Dual-energy X-ray absorptiometry (DXA) is required. The former is for body protein analysis and the latter for the determination of bone mineral content. However these two techniques require expertise for their usage, are relatively expensive, not easily available and can also be used to directly assess body fat without the need for UWW. Hence an alternative 4-C model was developed. For this model, the body is divided into fat, body cell mass, extracellular water and extracellular solids. Body cell mass can calculated by determining either ^{40}potassium or radioactive ^{42}potassium (Edelman and Leibman, 1959). Extracellular water compartments can be assessed by using bromide and sulphate compounds (Olney et al, 1952; Gamble et al, 1953). Extracellular solids are defined by the total body calcium or bone mineral content (Snyder et al, 1984).

Hence to determine body fat using this model, the sum of body cell mass, extracellular water and extracellular solids should be deducted from total body weight. Unfortunately, there are some limitations with the use of this model in the determination of body fat. Cumulative errors from measuring the body compartments and their summation can be transferred directly to the body fat estimate value.

Hence, it is possible to extend the number of compartments in the body composition model as long as the additional measurement is different from the previous ones. In the case of extracellular water (ECW) estimation, total body chloride can be used instead of bromide or sulphate dilution method (Shypailo & Ellis, 1998; Yasumura et al, 1983). It is best to use overlapping or repetitive methods when confirming a normal or abnormal

targeted outcome. Similarly, body carbon or body hydrogen (by neutron activation) can be used as assays for body fat mass and total body water respectively (Kyere et al, 1982; Kehayias et al, 1987).

Techniques such as magnetic resonance imaging (MRI) and computer tomography scans provide specific anatomical and structural information which can be used to monitor body organs. These techniques, unlike basic chemical composition analysis, require multiple slides to reconstruct volumes from which fat mass can be calculated if density is known.

Wang et al have proven through literature review over the past fifty years that there is an evolutionary shift from the basic 2-C model to 4-C model of body composition and have presented collated comprehensive information on the five-model of body composition (Wang et al, 1992). This five-level model has become the standard for body composition research (Kenneth, 2000).

1.3.4 Measurement of Body Volume and Density

Underwater Weighing

The classic method for determining body density is the Underwater Weighing (UWW). Body density measurement, which has been referred to as the gold standard for body composition assessment, using the UWW, requires the subject to be completely immersed in water (Behnke et al, 1942). When the subject is completely submerged, the volume of water displaced and/or the weight of the subject are used to calculate the body density. There are a few challenges in obtaining accurate values due to limitations as well as restrictions associated with estimates of body volume and residual lung volume (Buskirk, 1961; Katch, 1969; Siri, 1961; Wilmore, 1969).

Considering the two-compartment model, where body weight is the sum of body fat mass and fat-free-mass, $1/D_b = f_{fat} / D_{fat} + f_{FFM} / D_{FFM}$ where D_b , f_{fat} , D_{fat} , f_{FFM} and D_{FFM} stand for body density, fat mass, density of body fat (0.9g/l), fat-free-mass and density of fat-free-mass (1.1g/l) respectively. The density of fat can be assumed to be constant unlike fat-free mass which is heterogeneous (Martin et al, 1994; Deurenberg et al, 1989). There are possible variations with respect to gender, ethnicity, growth, sexual maturation, disease, aging and exercise (Cote and Adams, 1993; Schutte et al, 1984).

In most instances, some of the technical adjustments such as the residual lung volume correction factor are not routinely performed for bone mineral as well as body water measurements. However, they are approximated by the use of prediction equations and therefore it is essential to understand the limitations in accuracy when estimating per cent fat mass. The most commonly used method for residual lung volume correction is helium together with a closed – circuit spirometer system. In the case of older subjects and patients with malfunctioning pulmonary systems, an open - circuit nitrogen wash out is performed (Wilmore, 1969). It has been shown that irrespective of the method used, the correction factor does introduce a major source of error for per cent fat determination. An error of 100ml for residual lung volume leads to 1% uncertainty in fat estimation. Alternatively, if residual volume is not measured but estimated from prediction equations, the uncertainty increases to 3-4% with an error of 300-400ml in a given subject (Ellis, 2000).

The initial purpose for UWW methods development was to measure body volume to facilitate the assessment of body fat expressed as a percentage of body weight - % fat. However, it has been recommended that it should not be used as a criterion method for

heterogeneous populations (Milliken et al, 1996). When required to make these additional measurements to correct for the basic two-compartment model estimate, other criterion methods such as the dual energy x-ray absorptiometry (DXA) have been used. However, in cases where minimal radiation exposure can be tolerated such as pregnancy, then UWW should be considered.

1.3.5 Air – Displacement Plethysmography

The air – displacement plethysmography (ADP) is a two chamber system which has begun to replace the underwater weighing (UWW) technique. The subject is contained in a closed air – filled chamber, with a second chamber serving as a reference volume (Dempster and Aitkens, 1995; McCrory et al, 1995).

Studies undertaken on healthy adults have demonstrated very good agreement between ADP and UWW (McCrory et al, 1995 & 1998; Dempster and Aitkens, 1995, Sardinha et al, 1998). However, an advantage of the ADP method over the UWW is the fact that the subject does not have to be immersed in water. The subject is seated in one chamber and as the door is closed and/or sealed, the pressure of the chamber increases slightly. The diaphragm separating the two chambers is oscillated minimally to change the volumes. The principle underlying the use of the ADP is the relationship between pressure and volume when temperature is constant. This principle is applied to calculate the volume of the subject chamber.

The technical limitations related to the true volume noted above for UWW method also affect the use of ADP. With multiple readings (which can be obtained over a short period of time) these concerns can be averaged out. Another concern for the use of ADP

is that its accuracy has not been fully tested in children since it is used mainly for adults. In effect, most ADP instruments have been used for adults and significant changes are required for use in children and infants (Wells and Fewtrell, 2006).

Findings from anatomical and chemical studies (Heymsfield et al, 1989; Lohman and Going, 1993; Williams et al, 1993; Clarys et al, 1984) have indicated densitometric assessments of a population can have poor accuracy even if technical limitations can be corrected. This is because the normal variation in fat-free-mass densities within a given population in comparison with a fixed value for that same population will determine the accurate values for individuals in the population. Furthermore, assuming a fixed density will give rise to a larger error of FFM than the cumulative technical errors associated with the measured density itself. Moreover, with changes in FFM composition affected by growth, maturation, aging and diseases (Ellis, 1996; Ellis et al, 1997; Ellis et al, 1993; Fomon, 1982; Haschke et al, 1981 & 1983), additional knowledge is required from other measuring tools other than the two-compartment volume ADP technique which can identify outliers in a population or detect variation in fatness of individuals only over short time periods (Cohn et al, 1980 & 1981; Ellis, 1990; Forbes, 1987).

1.3.6 Validation of Bioelectric Impedance Analysis (BIA) for estimation of body composition in African and Caribbean Children

The high prevalence of paediatric overweight and obesity have led to several initiatives focussed on the need for better screening tools for childhood obesity as well as more effective strategies for accurate assessment of obesity and better management of its physiological, clinical and social consequences. Cardiovascular diseases (CVD) remain the commonest cause of death and disabilities in the UK (Scarborough, 2010) with

obesity as its major risk factor both in children and adults (Whincup et al, 2002). Ethnic differences in cardiovascular disease risk have been reported with increased risk and deaths observed in people of ethnic minority living in the UK (Nish, 2003). These risk factors which include high plasma triglycerides levels, increased insulin resistance and higher blood pressure levels, all of which are directly or indirectly linked to overweight and obesity, have been found even in children, contributing to an increased risk of suffering from CVD in later life (Whincup et al, 2002). Reports also show that the prevalence of type 2 diabetes, predisposed by overweight and obesity is now appearing in children and youth, which is higher in South Asian, Middle Eastern and black children all from ethnic minority background (Haines et al, 2007; Ehtisham et al, 2005).

To date, body mass index (BMI) has been the most common way to rank body adiposity in the assessment of overweight and obesity. Although BMI correlates with adiposity, it does not adequately describe ethnic variability in terms of overweight and obesity. For example, for a given BMI, people of South Asian origin have been found to have higher body fat as well as insulin resistance compared with white Europeans (Dudeja et al. 2001, Deurenberg et al. 2002, Ehtisham et al. 2005). Even in children and adolescents, it has been shown that for the same age and sex a child can have a twofold increase of fat mass for the same BMI (Wells, 2000). Unlike Caucasian children, children of black descent have lower average fat mass at similar BMI levels compared to Asian children (Deurenberg et al, 1998).

Furthermore, BMI does not provide information on relative proportions of fat and lean mass in an individual. However, for the same weight and/or BMI, an increase of fat mass as well as its location in the body and a decrease of lean mass especially skeletal

muscle mass have been linked to high risk of developing cardiovascular diseases and type 2 diabetes (Barker 2005). Body weight and BMI do not reflect either body composition or fat distribution and as such the use of BMI tables and charts despite their easy accessibility and simplicity as a main measure of overweight and obesity is not entirely acceptable.

For clinical purposes body composition measuring methods and tools must be simple, reliable, quick to administer and applicable to a wide variety of subjects. Skinfold-thickness and body circumference anthropometry have been used to ascertain body fatness for some populations and to predict body density by entering the data into multiple regression equations as in some National Health and Nutrition surveys (Kushner et al, 1990). Unfortunately, the accuracy of skinfold-thickness anthropometry is limited in estimating body composition due to multiple technical errors, population specificity and biological variations (Lohman, 1981).

Bioelectrical impedance analysis (BIA) is one of the methods available for the estimation of body composition in ambulatory clinical populations. BIA is relatively cheap, simple, non-invasive, easy to use, rapid, portable, reliable and widely used for estimating of body composition (Houtkooper et al, 1996; Eisenkolbl et al, 2001). Its accuracy has been evaluated by studies which have demonstrated very good correlations between the BIA, total body water (TBW), fat-free mass (FFM, using hydrodensitometry) and total body potassium in lean and obese adults as well as children (Schoeller et al, 1989; Houtkooper et al, 1989). BIA assumes the body as a series of cylinder of length equivalent to its height (HT). It works by passing a low level of electric current through the body to measure the impedance of conducting tissues.

Impedance factor (which is calculated as HT^2/Z, where Z is the impedance produced by the BIA), is proportional to TBW as well as lean mass (Kushner, 1992; Schoeller, 2000).

Numerous studies have demonstrated that age, gender, ethnicity and extreme levels of fatness influence BIA estimates of body composition measurements (Bray et al. 2002, Lohman et al. 2000). BIA has been validated predominantly in white Europeans with minimal information on the accuracy of using these BIA prediction equations in other ethnic groups such as Asian and black populations, especially in children and adolescents: hence the need to validate the BIA for specific ethnic groups to derive ethnic-specific equations for calculating body composition parameters accurately.

Several validation studies have been carried out in adults and children to assess the BIA system (Jebb et al, 2000; Jartti et al, 2000; Tyrrell et al, 2001; Sung et al, 2001; Goss et al, 2003; Parker et al, 2003; Pietrobelli et al, 2004). Previous studies have been limited by factors such as a wide age range, sample size, lack of criterion method for comparison, lack of information on validity of BIA compared with DXA in young children and doubts over its accuracy (Lukaski and Siders, 2003; Tyrrell et al, 2001).

The goal of this study is to develop whole group and gender specific BIA equations to estimate body composition of black children and adolescents. DXA was used as the criterion method in this study. Although it is of limited availability, relatively expensive and time consuming, DXA is more suitable for use in children because the radiation exposure is minimal and it has no discomfort. It is non-invasive and has excellent reproducibility of measurements (Svendsen et al, 1993; Svendsen et al, 1990). In

addition, studies have confirmed that body composition measurements by DXA are accurate and precise (Lohman and Chen, 2005). Magnetic resonance imaging and computed tomography are also considered useful criterion methods which provide visible images of adipose tissues and have been directly validated against cadaver body composition analyses (Ross and Janssen, 2005). They can also distinguish between intra-abdominal and subcutaneous adipose tissue. In the past, a major limitation of validation of BIA in children and adolescents studies has been the use of criterion methods with invalid assumptions of chemical constancy leading to inflation of the error in the criterion (Lohman, 1992; Going et al, 2006). Using DXA reduces error due to criterion method since it is based on a three-compartment model of body composition requiring fewer assumptions than methods based on two-compartments. Furthermore the adoption of DXA as a criterion method is justified by the fact that it has been successfully validated against multi-compartment models (Lohman and Chen, 2005) and chemical analysis of animal models (Pintauro et al, 1996).

1.3.7 The Metabolic Syndrome

The metabolic syndrome has become one of the most important health issues of this decade because of the marked increase in coronary heart disease, cardiovascular atherosclerotic disease and type 2 diabetes mellitus risk associated with its clustering disorders. It is now considered to be the main driving force for a new cardiovascular disease (CVD) epidemic. The syndrome is a cluster of the most dangerous heart attack risk factors (IDF 2006) and it is characterised by dyslipidaemia, hyperinsulinaemia, and deregulated glucose homeostasis, elevation of arterial blood pressure, abdominal obesity and insulin resistance (BMJ, 2005). Recently, other characteristic features of the syndrome have been identified such as chronic proinflammatory and prothrombotic

states, non-alcoholic fatty liver disease and sleep apnoea (Kassi, 2011). These features have long been known to occur together in individuals and in populations much more frequently than would be predicted by chance alone (IDF, 2006).They are interrelated and share underlying mediators, mechanisms and pathways (Kim et al, 2006). The more evident the components of the metabolic syndrome, the higher the cardiovascular mortality rate (Hu et al, 2004). The occurrence of these multiple metabolic abnormalities in an individual appears to confer a substantial additional CVD risk over the risk associated with each abnormality (Sattar and Scherbakova, 2008; Golden et al, 2002). With cardiovascular disease set to become the world's principal killer by 2020, there is the need to pay urgent attention to the metabolic syndrome.

Clinically this syndrome is not an absolute risk indicator because its main characteristic features do not include many of the factors that determine absolute risk such as age, sex and ethnicity. However patients who suffer from the syndrome are at twice the risk of developing cardiovascular diseases and it confers a five-fold increase risk for type 2 diabetes mellitus over the next five to ten years compared with those without the syndrome (Alberti et al, 2009). The risk over a lifetime is even higher. Despite the many components and clinical manifestations of the syndrome, there is still no universally accepted pathogenic mechanism or clearly defined diagnostic criteria.

1.3.7.2 Paediatric definition: challenges and prevalence rates

One of the major evolving aspects of the metabolic syndrome is its increasing prevalence among childhood and young adulthood populations, highlighting the future implications of the syndrome to the global health problem (Kassi et al, 2011). The increasing worldwide prevalence of childhood overweight and obesity has highlighted

the importance of diagnosing the metabolic syndrome in children and adolescents as a state of high risk for progression to other diseases in later life (Weiss et al, 2004). The main challenge is that all paediatric definitions originate from adult definitions of the syndrome and the criteria used is an extrapolation from an adult diagnosis to a young age group, since criteria for children has not been fully established. There are limited studies linking childhood metabolic syndrome to adult metabolic syndrome and despite the fact that it has been hypothesised that they are related, this hypothesis is yet to be fully tested (Kassi et al, 2011). Furthermore, the syndrome lacks a developmental perspective in its definition because as an entity, the metabolic syndrome develops progressively with changes in its parameters. With the maturational changes taking place during childhood and adolescence, it has become a challenge to fully make a diagnosis of metabolic syndrome in the paediatric age group (Chen et al, 2000). This calls for body composition studies in children and adolescents from different ethnic backgrounds to be able to develop reliable cut off limits for the parameters of the syndrome. To date, no general consensus definition in children and adolescents exist and studies published so far have used their own set of cut off parameters to define those at risk and those with the syndrome (Pervanidou et al, 2006).

In some settings, childhood obesity has been defined as more than 95th percentile for BMI or more than 90th percentile for waist circumference and a number of cut off percentiles have been used for blood pressure, high density lipo-proteins (HDL), triglycerides, insulin as well as glucose levels (Brambilla et al, 2007) all of which are main features of the syndrome. A report published by the International Diabetes Federation concerning the definition for the metabolic syndrome in the paediatric age range, three age categories were classified: 6 years to less than 10 years, 10 years to less

than 16 years, and 16 years to more than 16 years or adults. This report defined obesity as waist circumference $\geq 90^{th}$ percentile and all other variables were defined based on absolute numbers instead of percentiles. These include triglycerides $\geq$ 150 mg/dl, HDL < 40 mg/dl (or < 50 mg/dl in females older than 10 years), blood pressure (BP) $\geq$ 130 mmHg for systolic and for diastolic $\geq$ 85 mmHg (or treatment of previously diagnosed hypertension) and fasting blood sugar $\geq$ 100 mg/dl or known type 2 diabetes. It has been explained that the rationale for using absolute numbers as cut offs stems from the heterogeneity of biochemical, clinical and hormonal levels observed during childhood and adolescence as well as the variety of proposed percentile cut off points of various definitions (Zimmet et al, 2007).

According to findings from the third National Health and Nutrition Examination Survey (NHANES – 1988 to 1994; Cook et al, 2003), in the general US population 4.2% of adolescents have been diagnosed with metabolic syndrome and approximately 30% of overweight and obese adolescents met the diagnostic criteria for the syndrome in the US (Ferranti et al, 2004; Goodman et al, 2005). Six years later, using the ATPIII definition modified for age, the NHANES revealed an increase in the prevalence rate of the syndrome among US adolescents. The general prevalence of 4.2% in NHANES III (1988-1994) had risen to 6.4% in NHANES (1999 to 2000) and the prevalence was found to be even higher among overweight and obese adolescents (Duncan et al, 2004). Similarly, it has been found that the prevalence of the syndrome in US adolescents was higher among Hispanics than among black and white adolescents and it has been found that the former group of adolescents have higher rate of obesity than the latter. The prevalence of overweight and obesity in Hispanic youths was found to have doubled in the past ten years (Strauss et al, 2001).

In a population-based longitudinal study of cardiovascular disease risk factors in black and white children (Bogalusa Heart study), the metabolic syndrome was defined as having $\geq 75^{th}$ percentile for age and gender of any four of the following components- obesity, hyperglycaemia, hypertension, dyslipidemia and insulin resistance. Using this definition and with data derived from their own specific populations, prevalence rate of the syndrome was 4% among white children and 3% among black children (Chen et al, 2000). In Finland, a large multicentre study of heart disease risk factors in children and adolescents revealed a prevalence rate of 4% of the metabolic syndrome among a total of 1865 subjects (Raitakari et al, 1995).

Although the challenge still exists to create child-specific definitions for the metabolic syndrome due to lack of outcome data and variability in measures of different risk factors, it has been suggested that the focus should be on prevention and management of obesity as well as all the other risk factors and not to wait for a child to meet the diagnostic criteria before instituting treatment.

1.3.7.3 Foetal origins of the metabolic syndrome

Evidence from epidemiological observations, clinical and experimental animal studies suggests that the natural history of the metabolic syndrome may originate in intrauterine life (Nektaria et al, 2010). Although the complete mechanism of foetal programming is not fully understood, evidence suggests that the nutritional, hormonal and metabolic environment a foetus is exposed to intrauterinally as well as in early post-natal life may permanently reprogram the structure and physiology of differentiating tissues of the foetus toward the development of metabolic disease in later life (Xita and Tsatsoulis,

2010). In effect, the altered tissue differentiation may result from foetal adaptative responses representing homeostatic adaptations due to changes in foetal nutrition and milieu (Fernandez-Twinn and Ozanne, 2010).

However, some studies done using in vitro fertilisation (IVF) to ascertain the incidence of the metabolic syndrome has produced conflicting results (Sakka et al, 2010; Miles et al, 2007). Hence more prospective studies involving foetal programming with longer follow ups are required to draw safe conclusions and to assist researchers to come up with interventions at an early stage to curb alarming incidence of the metabolic syndrome (Kassi et al, 2011).

In view of the major issues outlined in this literature review, this thesis set out to address the following aims:

1.4 Study Aims

1. To measure body composition in African and Caribbean children/youths using the Bioelectrical Impedance Analysis (BIA).
2. To validate the BIA for African and Caribbean children/youth using the Dual Energy X-ray Absorptiometry (DXA).
3. To develop percentage fat mass and fat free mass percentile reference curves for African and Caribbean children and youth population.
4. To develop skeletal muscle mass curves and muscle-to-fat-ratios for the African - Caribbean child and youth population.
5. To develop blood pressure height percentile reference tables and charts for African and Caribbean children/youths.
6. To develop waist circumference percentile charts and tables for African and Caribbean children.

Chapter 2: Methodology

The primary investigator and assistants all had enhanced CRB checks. The study protocol was approved by the Faculty of Life Science ethics committee of London Metropolitan University.

2.1 General methods and subjects

The following measurements featured across the studies in this thesis

2.1.1 Measured Variables

1) Age
2) Gender
3) Ethnicity
4) Weight
5) Height
6) Waist circumference
7) Per cent body fat
8) Systolic blood pressure
9) Diastolic blood pressure
10) Heart rate
11) Skeletal muscle mass

2.1.2 Data Collection Technique

A structured interview using a closed ended questionnaire (appendix A) was administered to all parents to assess the health status and family medical history as well

as eligibility to participate in the study. The question consisted of simple questions with yes or no answers to tick. All measurements were taken in the nutrition research laboratory- LMU.

2.1.2.1 Recruitment of study participants

A number of letters were given out to leaders of African and Caribbean churches across the North-west to South-east London, who had shown interest in assisting in similar projects in the past. Appointments were made to visit their church members at their convenient times with the view to explain the details of the study and to recruit volunteers. After several visits to the various churches both on week days and weekends and sometimes late evenings, health education talks delivered over a period of more than four months, a number of the worshippers started to show interest, which was initially lacking. This proved that when working with people from the black community, patience is required. This was necessary for them to accept the researcher and be willing to participate in the research. With time more members from the various congregations from Pentecostal and Adventist churches all of African and Caribbean background willing gave their names as participants of the study and volunteers were noted. This was after the primary researcher had paid several visits, sometimes going along with her entire family to worship with the congregation to demonstrate her interest in the congregation members and letting them know that her own children were part of the study. Fortunately, her 12- year old girl and 10- year old boy who had participated in the pilot study were always with her to witness to the congregation members both young and old how exciting it was to be at the science laboratory for measurements to be taken. This helped to boost the interest of the congregation members to join in their numbers.

Each volunteer was visited at their own convenient times to address their individual concerns as well as to explain informed consent and the participant information forms for signing. It was important for them to become familiar with the primary investigator as some fear their information may be communicated to others – "the gossip fear". Among some black communities, there is fear when a black investigator/researcher comes to find out certain information about their families. Some would prefer the investigator/researcher to be of different race not related to the black community- that is preference for an outsider not an insider. There is fear that through gossip such family information can reach other black communities and this can lead to disrespect for the affected family. Hence all these fears were allayed before most of the participants agreed to join the research team. Finally, a date was fixed for travel to the science laboratory in the company of the principal researcher.

Participants were taken through various security checks with the researcher before being escorted to the laboratory by a member of staff which usually was the primary supervisor of the research project.

2.1.2.2 Eligibility Criteria

Healthy 5-18 year old African and Caribbean children whose parents and/or carers have signed the informed consent and participants who willingly signed the volunteer information form.

2.1.2.3 Exclusion Criteria

- Children below 5 years of age.
- Children above 18 years of age.

- Children whose parent or carers did not sign the informed consent form.
- Pathological states likely to affect growth or body composition, moderate to severe physical disability and long-term use of steroid assessed using a detailed medical history questionnaire (Appendix A).

2.1.2.4 Pre – Testing/ Pilot Study

A pilot study was conducted on ten children from African and Caribbean background who were eligible for the study and the following were assessed:

- The reliability of research tools and equipment.
- Reaction of respondents to the research procedure.
- The number of children whose data could be taken in a day.
- Availability of sample needed for actual study.
- Average time needed to relax a child before blood pressure can be taken.
- Whether the average time of two minutes in between various blood pressure measurements was adequate.

2.1.2.5 Quality Control Checks

1. The research process was well explained to all participants and their parents/carers before proceeding with consent forms and questionnaire.
2. Parents/carers as well as participants were asked not to take any stimulant drugs, food or drinks (such as caffeine beverages) on the day of measurements.
3. All participants were calmed and rested for at least ten before blood pressure measurements were taken.

4. Participants were asked to empty their bladder and be seated so that they did not move during blood pressure measurements.

5. A range of cuff sizes were made available so that all participants could have the right size of arm cuff which would cover not less than two thirds of arm to avoid inconsistencies in blood pressure readings.

6. It was ensured that participants wore minimal clothing for weight measurement.

7. Before initiating measurements with the bio-electrical impedance analysis, a standard weight of 1kg was deducted to ensure that the minimal clothing worn was taken care of and all participants were kindly asked to take off their shoes and socks to ensure good contact with feet platform to obtain reliable readings (Garrow, 2000).

8. The Bod Pod and DXA were calibrated based on standard guidelines from manufacturers' manual on the same day before measurements of per cent body fat were taken.

9. Swimming suits and caps were provided for participants to wear before asking them to enter the Bod Pod according to standard protocol.

10. It was ensured that none of the participants wore any metallic ornaments and that they wore light clothing before taking their measurements with the DXA scan.

2.1.3 Data Collection

Eligible children who responded to the closed ended questionnaire were taken to the laboratory for their measurements to be taken. On arrival at the laboratory, participants are asked to pass urine so that they can remain seated once they've been seated for

blood pressure (BP) measurements. The children are seated with their backs supported, feet touching the floor, calmed and rested for an initial time of ten minutes. Hence blood pressure measurements were taken first.

Blood Pressure (BP)

To take BP after the initial resting period when subjects were comfortably seated, seats were arranged so that participants remained seated throughout the period of BP measurements with only the researcher moving from one child to the other.

The right arm is positioned such that the cubital fossa lay parallel to the level of the heart and using an Omron digital BP monitor (Model Number: HEM- 907XL OMRON Inc. USA) readings were recorded on three occasions with resting time of two minutes in between BP measurements (AHA Scientific Statement, 2005, Thomas et al, 2005). This device has fulfilled the International Scientific Validation Protocol (Arlington, 1993).

Weight

Weight measurements were taken using a standard Seca scale calibrated using the standard 10kg weight. It was placed on a hard flat surface. The zero balance was checked before each measurement with subjects standing erect in the centre of the scale platform, looking straight ahead, wearing minimal clothing, relaxed and unassisted (WHO, 1995).

Height

To measure height, the standard one metre rule was used to calibrate the portable stadiometer (Leicester stadiometer, Marsden UK, Mod 220). Subjects were carefully assisted to stand straight with their heads positioned such that their Frankfurt plane is horizontal, feet together, knees straight, heels and shoulder blades in contact with the vertical surface of the stadiometer. It was also ensured that subjects had their arms

hanging loosely at their sides with their palms facing their thighs. They were then asked to take a deep breath while standing tall to aid the straightening of the spine, with shoulders relaxed. Following this, the headboard was adjusted to touch the crown of the subject's head and the height reading recorded to the nearest 0.1cm (WHO, 1995).

Waist Circumference

Waist circumference was accurately measured using a flexible/non-elastic tape measure. The midpoint between the lowest rib and the top of the hip bone (anterior superior iliac spine) was determined (National Waistline Report 2015). Subjects were then asked to breathe normally as the measuring tape was applied directly and snugly around the determined point ensuring that the tape did not compress the skin to take readings on top of the skin as shown below:

Figure 2.1 Image of waist circumference measurement (http://www.acefitness.org/fitness-fact-article)

After the above anthropometric and BP measurements were taken, body composition measurements were also determined using the BIA, Bod Pod and the DXA scan according to standard protocol as shown below:

2.1.4 Bio-impedance Analysis (BIA) measurements

BIA (Tanita BC-418MA III) was used to measure impedance (Garrow, 2000). The following explains the validity and reliability of the BIA used: the prediction equations

used in this model are based on bio-impedance, weight, height and age which were derived from calibration studies against whole-body dual X-ray absorptiometry (DXA) (McCarthy et al, 2006). The impedance scales used in this study have been validated against DXA in mixed populations of children and adults and found to be superior to previous BIA methods (Pietrobelli et al, 2004). More recently a paediatric validation of the BC-418MA model against DXA and air-displacement plethysmography (BodPod) has been performed. In samples of 45 boys and 34 girls, results were highly correlated with DXA (r = 0.91, SEE = 4.46%) and mean values did not differ significantly (Pietrobelli et al, 2005). To take measurements using the Tanita BIA (BC-418MA), first participants are asked to be in light clothing and barefoot. After that, a standard of 1kg was entered/deducted as an adjustment for clothing weight in all participants. This was done because previous studies have proven that the1kg is appropriate to be deducted (McCarthy et al, 2006). Then the gender, age and height were manually entered before participants stepped on the foot plates (ensuring that both feet span the electrodes) while holding the two electrodes away from their bodies for impedance measurements. BIA measurements took 3-5 minutes per subject.

2.1.5 DXA measurements

The Norland XR8000 DXA criterion estimates of per cent body fat (% BF) and skeletal muscle mass (SMM) were measured using a standard protocol as outlined below. The DXA scanner was calibrated daily against the manufacturer's standard calibration blocks including on the day of the measurements. It was calibrated early in the morning before the arrival of the children and their parents. To minimise error, operators were centrally trained in the proper procedures for obtaining and analysing scans. All scans were conducted and analysed on each day by a single operator – the study supervisor. Participants wore light clothing, then positioned according to the standard

manufacturer's instructions for whole body scans. Participants were required to lie supine with their arms by their sides, palms facing down and legs straight. They were reminded to lie still and refrain from talking before the scan was switched on for the process to begin. Once the process is started the door of the scan room is closed and participants are asked to close their eyes. The scanner utilises a narrow laser beam perpendicular to the longitudinal axis of the body. Scans were analysed using the manual mode according to the manufacturer's standard procedures for defining the cut-off points for each body part and the software for DXA was Windows-Norland Bone Densitometry software (Faulkner et al, 1995). Total scanning process took approximately between 5 to 10 minutes per subject. Two participants were exempted because they could not lie still for the DXA scan measurement. Repeated DXA measurements were not performed on the children to avoid repeated exposure to radiation.

2.1.6 Statistical analysis

All statistical analyses were carried out using SPSS for Windows (version 18 and 19, SPSS Inc, Chicago, IL, USA), Excel and MedCal (version 12.2.1.0). Details of statistical analysis are clearly explained in the individual study chapters.

Chapter 3: Derivation of body fat-mass equations to validate the BIA system

3.1. Introduction

Africans and Caribbeans form 11% of the London population and are part of the minority ethnic groups (Butland et al, 2008). Ethnic minority groups are generally socially and economically disadvantaged and are more vulnerable to becoming obese (Foresight, 2008). Hence prevalence of overweight and obesity with its health consequences can be greater for children from African and Caribbean background (McCarthy, 2006).

The body mass index (BMI) is used to classify and monitor overweight and obesity in children. BMI represents the sum of fat and fat-free masses correlated to height. Morbidity associated with obesity is due to excess fat and the ideal monitoring tool should directly access body fat (Fortuno et al, 2003). BMI also does not indicate the distribution of fat in the body. Intra-abdominal fat is particularly associated with obesity related ill-health and people of black descent commonly have excess intra-abdominal adiposity (Greaves et al, 1989).

Bio-electrical Impedance Analysis (BIA) is a reliable, simple and convenient tool that can predict body composition and per cent body fat in various segments of the body such as trunk, legs and arms (Going et al, 2006; Cole et al, 1995). Most BIA systems have only been validated for the Caucasian population and these validated equations most likely cannot predict body composition accurately in African and Caribbean children. Hence the need to validate the BIA for measurement of body composition for

African and Caribbean children living in the UK so that the technology can be used with confidence to determine body composition.

Reference methods including magnetic resonance imaging (MRI), computed tomography (CT), hydrodensitometry, dual-energy X-ray Absorptiometry (DXA) and whole-body Air-displacement plethysmography are considered as criterion methods when validating BIA systems (Ellis, 2000; Heymsfield et al, 2005). For this study the criterion method used to validate the Tanita BC418-MA system is the DXA. DXA is more suitable for use in the young population as it is comfortable with minimal radiation exposure. It is precise as well as accurate and its use as criterion method is based on successful validation against multi-compartment models and animal models (Lohman and Chen, 2005; Pintauro et al, 1996).

3.2 Aim

To validate a Bio-electric Impedance Analysis (BIA Tanita BC418) system with the use of the Dual Energy X-ray Absorptiometry (DXA) for measurement of body composition for African and Caribbean children.

Objectives:

- To derive gender-specific validation equations for body fat mass using linear regression analysis after determination of association between the BIA and the DXA - the gold standard criterion.

3.3 Methods

Body composition parameters (including fat mass) of 44 black children aged 5-18 years (24 boys & 20 girls, mean age 10.5, SD 3.96) were measured using BIA (Tanita BC418 segmental BC analyser) and DXA (Norland XR8000) after anthropometric

measurements had been taken. Procedures for measurement are detailed in chapter 2 sections 1.11 and 1.12. This was used to develop fat mass validation equations as shown below.

3.3.1 Statistical analysis

Anthropometric data as well as measurement from BIA and DXA were collated onto an Excel spreadsheet and exported to SPSS (version 19) to generate descriptive statistics of the study population.

For the derivation of the validated equations, first the association between the two measuring systems was determined using Pearson's correlation test. Since correlation does not fully establish agreement nor identify bias, the data were tested for normality in order to use Bland Altman analysis to identify bias and also to show the level of agreement between the two measuring systems. To test for normality, the Kolmogorov-Smirnov test for normal distribution/histogram was used. This test was performed twice in this chapter:

1) Before using Bland Altman analysis and
2) Before deriving the gender specific validated equations - to test the normality of the DXA measurements (the dependant variable of the equation).

The line of equality was plotted to provide a visual impression of the association between the two measuring systems - DXA and BIA. Then the first test of normality was performed. After examination of the data by the use of scatter plots and frequency distribution for test of normality, the limit of agreement – Bland-Altman plot/analysis was then used to test the study. The Bland-Altman analysis for the differences in fat mass measured by DXA and BIA for the study was performed using MedCal software (version 12.2.1.0). Then, the DXA measurements which constitute the dependent

variables were evaluated for normal distribution using Kolmogorov-Smirnov test and normal Q-Q plot. When the DXA measurements passed the test of normality, the correction/validation equation for the sample population, the linear regression of DXA FM against height and impedance was produced for the boys and girls.

3.4 Results

Tables 3(a) to 3(d) show the descriptive statistics for the sample population for both the BIA and DXA measurements. In general, height, weight, BMI, fat mass (FM), fat-free mass (FFM) and skeletal muscle mass (SMM) all increased with increasing age.

Table 3(a). Descriptive statistics for the sample population – BIA Boys

Values = mean ±SD

					BIA							
BOYS	Height (cm)	Weight (kg)	BMI (kg/m^2)	Ht^2/Z (m^2/Ω)	FFM (kg)	FFM (%)	FM (kg)	FM (%)	SMM (kg)	SMM (%)	SMM/FFM (%)	MFR
5 - 7	124.7	24.5	15.9	2.17E-03	20.1	82.8	4.5	17.1	7.6	30.8	37.0	2.17
	11.5	5.6	3.7	5.48E-04	3.6	8.2	3.1	7.8	2.3	6.4	6.0	1.28
8-10	141.3	34.8	17.5	2.99E-03	28.4	81.9	6.4	18.0	11.5	33.0	40.4	1.93
	7.4	4.6	2.4	4.10E-04	3.2	4.6	2.2	4.6	1.4	2.0	1.0	0.49
11-13	166.3	60.1	21.1	4.65E-03	47.2	82.5	10.6	17.6	20.0	33.2	42.3	1.88
	11.0	3.6	2.2	8.37E-04	5.4	0.3	0.7	0.2	2.8	3.4	1.1	0.17
14-16	169.1	60.6	21.2	4.58E-03	48.9	81.0	11.2	18.4	20.2	33.5	41.4	1.89
	2.6	8.9	2.6	5.77E-04	6.1	3.8	3.1	2.9	2.8	3.5	2.5	0.49
17-19	176.6	68.4	22.0	5.28E-03	57.4	83.0	11.9	17.2	23.8	34.8	41.5	2.02
	7.6	3.1	1.2	4.28E-04	2.4	1.3	1.7	1.7	2.6	2.4	3.0	0.23

Table 3(b). Descriptive statistics for the sample population – DXA Boys

Values = mean ±SD

					DXA							
BOYS	Height (cm)	Weight (kg)	BMI (kg/m^2)	Ht^2/Z (m^2/Ω)	FFM (kg)	FFM (%)	FM (kg)	FM (%)	SMM (kg)	SMM (%)	SMM/FFM (%)	MFR
5 - 7	124.7	24.5	15.9	2.17E-03	18.1	73.5	6.8	26.5	6.9	28.5	39.3	1.39
	11.5	5.6	3.7	5.48E-04	4.5	14.1	5.3	14.1	1.5	4.6	4.9	0.75
8-10	141.3	34.8	17.5	2.99E-03	26.4	75.9	8.6	24.1	9.6	27.8	37.4	1.41
	7.4	4.6	2.4	4.10E-04	4.2	11.5	4.6	11.5	1.2	3.1	6.8	0.69
11-13	166.3	60.1	21.1	4.65E-03	49.5	85.0	9.0	15.0	18.6	31.0	35.9	3.02
	11.0	3.6	2.2	8.37E-04	4.4	9.2	5.5	9.2	5.6	9.9	8.0	2.53
14-16	169.1	60.6	21.2	4.58E-03	50.1	84.7	9.3	15.3	20.1	33.3	39.2	2.20
	2.6	8.9	2.6	5.77E-04	6.9	1.2	1.5	1.2	4.5	6.1	6.7	0.59
17-19	176.6	68.4	22.0	5.28E-03	56.8	85.0	10.2	15.0	22.1	32.1	37.6	2.30
	7.6	3.1	1.2	4.28E-04	4.4	4.4	2.6	4.4	6.1	7.3	7.4	0.92

Table 3(c). Descriptive statistics for the sample population – BIA Girls

Values = mean ±SD

					BIA							
GIRLS	Height (cm)	Weight (kg)	BMI (kg/m^2)	Ht^2/Z (m^2/Ω)	FFM (kg)	FFM (%)	FM (kg)	FM (%)	SMM (kg)	SMM (%)	SMM/FFM (%)	MFR
5 - 7	126.3	25.4	15.9	2.11E-03	20.0	78.7	5.1	20.2	7.1	28.0	35.6	1.42
	10.4	3.9	0.7	3.47E-04	3.0	2.8	0.9	1.9	1.4	1.6	1.4	0.18
8-10	147.5	38.1	17.3	2.85E-03	29.2	76.7	8.6	22.5	11.1	28.9	37.7	1.30
	10.2	7.9	1.7	5.42E-04	5.7	2.0	2.3	1.7	2.6	2.3	2.7	0.15
11-13	156.1	50.4	20.5	3.43E-03	37.8	75.6	12.5	24.3	14.9	29.6	39.4	1.27
	10.2	10.9	3.1	6.01E-04	7.3	5.7	5.2	5.8	3.2	2.4	4.7	0.23
14-16	158.9	53.1	21.2	3.72E-03	39.4	74.2	13.4	25.4	15.7	29.6	39.9	1.17
	9.8	0.3	2.7	3.79E-04	0.6	0.7	0.1	0.1	0.8	1.8	2.7	0.08
17-19	162.5	59.6	22.6	3.72E-03	43.4	72.8	16.3	27.2	16.8	28.2	38.8	1.04
	2.8	0.6	0.6	1.00E-04	1.8	3.7	2.3	3.7	0.4	0.4	2.6	0.12

Table 3(d). Descriptive statistics for the sample population – DXA Girls

Values = mean ±SD

					DXA							
GIRLS	Height (cm)	Weight (kg)	BMI (kg/m^2)	Ht^2/Z (m^2/Ω)	FFM (kg)	FFM (%)	FM (kg)	FM (%)	SMM (kg)	SMM (%)	SMM/FFM (%)	MFR
5 - 7	126.3	25.4	15.9	2.11E-03	17.9	69.8	7.7	30.3	5.7	22.5	32.3	0.76
	10.4	3.9	0.7	3.47E-04	2.4	3.1	1.4	3.1	0.8	2.3	2.3	0.15
8-10	147.5	38.1	17.3	2.85E-03	26.0	69.0	11.9	31.0	9.3	24.2	35.0	0.80
	10.2	7.9	1.7	5.42E-04	4.8	4.2	3.7	4.2	3.0	6.1	7.8	0.27
11-13	156.1	50.4	20.5	3.43E-03	35.1	68.7	16.3	31.3	13.0	25.8	38.0	0.88
	10.2	10.9	3.1	6.01E-04	5.9	9.0	7.4	9.0	2.8	1.4	4.4	0.23
14-16	158.9	53.1	21.2	3.72E-03	36.4	68.5	16.7	31.5	14.2	26.8	39.2	0.85
	9.8	0.3	2.7	3.79E-04	0.2	0.7	0.5	0.7	1.3	2.6	3.4	0.10
17-19	162.5	59.6	22.6	3.72E-03	38.7	64.5	21.2	35.5	16.3	27.3	42.4	0.77
	2.8	0.6	0.6	1.00E-04	5.9	2.1	1.5	2.1	0.8	1.0	3.0	0.02

Figure 3(a) shows the scatter plot for the whole sample with the line of equality – the line on which all points would lie if there was a perfect agreement between the two measuring systems (Hanneman, 2008).

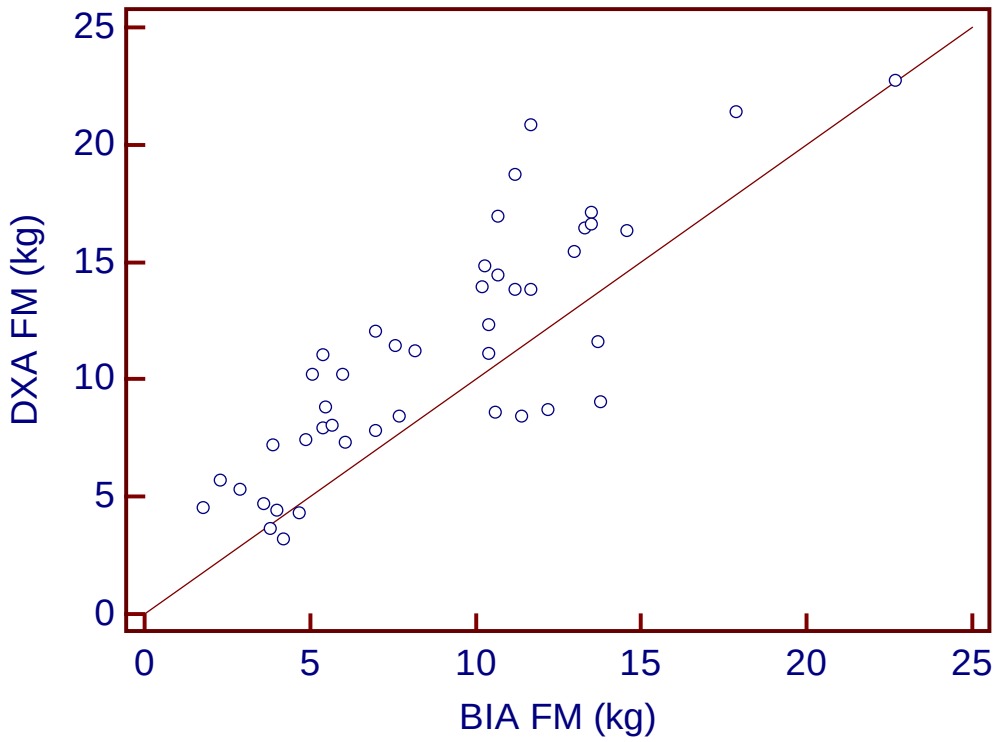

Figure 3(a). Line of equality DXA FM (kg) against BIA FM (kg) for the whole sample

Table 3(e) below shows the Pearson's correlation coefficient (r) is 0.84 and the significance level p is less than 0.0001 which shows a good linear relationship and also points to the statistical significance of the relationship showing that the two measurements are associated (Bland and Altman, 1986; Chan, 2003). The coefficient of determination (the square of r) is 0.71 which shows a 71% of common proportion of variance between DXA and BIA fat mass measurement (Chan, 2003). Table 3(e) provides the results of Pearson's correlation analysis.

Table 3(e) Pearson's correlation between DXA FM (kg) against BIA FM (kg)

Pearson's Correlation between DXA FM(kg) and BIA FM(kg)	
Sample size	44
Correlation coefficient r	0.8357
Significance level	P<0.0001
95% Confidence interval for r	0.7166 to 0.9075

Figure 3(b) shows the results of Kolmogorov-Smirnov test for normal distribution of DXA and BIA fat mass. As shown in table 3(f), the p value of the Kolmogorov-Smirnov test is greater than 0.1. The p value is thus greater than 0.05, indicating that the distribution is an accepted normality (Hanneman, 2008). In addition, the data passes the test of normal distribution because the coefficient of skewnessand kurtosis which are -0.2519 and 0.7111 respectively, are approximate to zero (Brown, 2011). Hence the variables can be described by means of the mean and standard deviation and also subjected to parametric statistical test/Bland Altman analysis (Hanneman, 2008). It is therefore envisaged that 95% of the measured subjects will fall between the Bland-Altman limits of agreement (Hanneman, 2008).

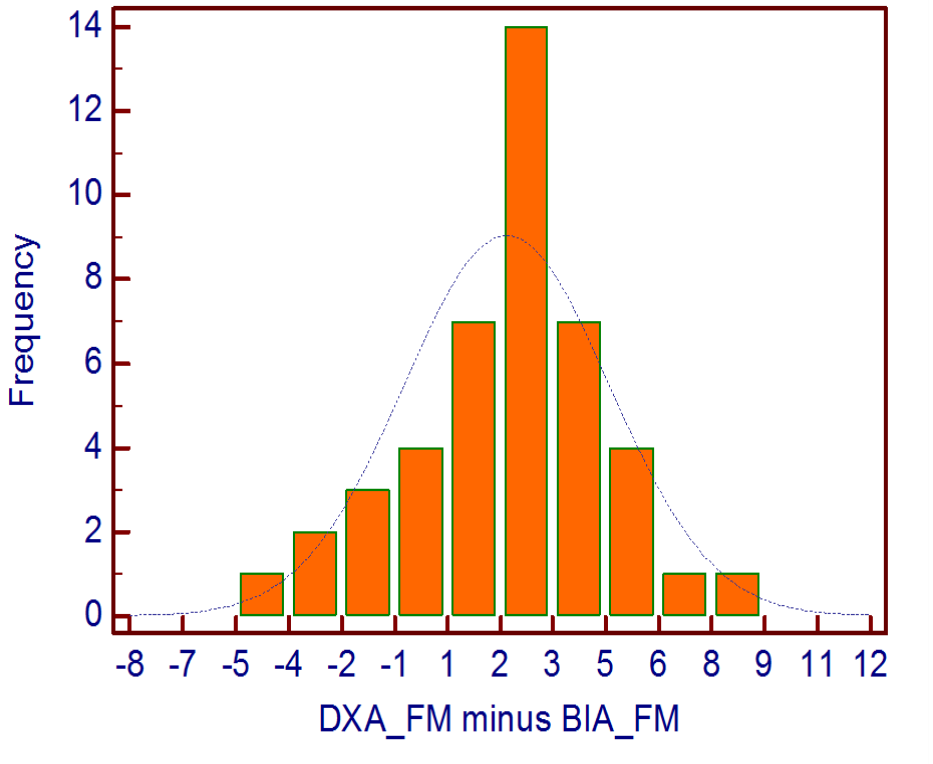

Figure 3(b) Frequency distribution of the difference between DXA FM (kg) and BIA FM (kg)

The Bland-Altman plot of the difference between DXA fat mass and BIA fat mass against mean of DXA fat mass and BIA fat mass as indicated in figure 3(c) below, shows a good relationship between the differences with no proportional error.

Table 3(f) Kolmogorov-Smirnov test difference between DXA FM (kg) and BIA FM (kg)

Kolmogorov-Smirnov test for Normal Distribution (DXA-BIA)	
Sample size	44
Lowest value	-4.8
Highest value	9.1
Arithmetic mean	2.175
95% CI for the mean	1.3317 to 3.0183
Median	2.5
95% CI for the median	1.7069 to 3.2931
Variance	7.6931
Standard deviation	2.7736
Relative standard deviation	1.2752 (127.52%)
Standard error of the mean	0.4181
Coefficient of Skewness	-0.2519 (P=0.4620)
Coefficient of Kurtosis	0.7111 (P=0.2662)
Kolmogorov-Smirnov test	D=0.1089
for Normal distribution	accept Normality (P>0.10)

The observed differences were both negative and positive differences and thus there was no systematic bias between the measurements. The 95% tolerance intervals for the paired observations, limits of agreement, are the mean difference plus or minus 1.96 times the standard deviation of the difference. These represent the range of values satisfied by the agreement between the use of DXA and BIA for approximately 95% of the variables when the differences between the methods were normally distributed (Bland and Altman, 1986; Hanneman, 2008). Figure 3(c) shows almost 95.5% of the variables within the limits of agreement (that is from +1.96 to – 1.96) and this clearly establishes that there is a good agreement between the two measuring systems.

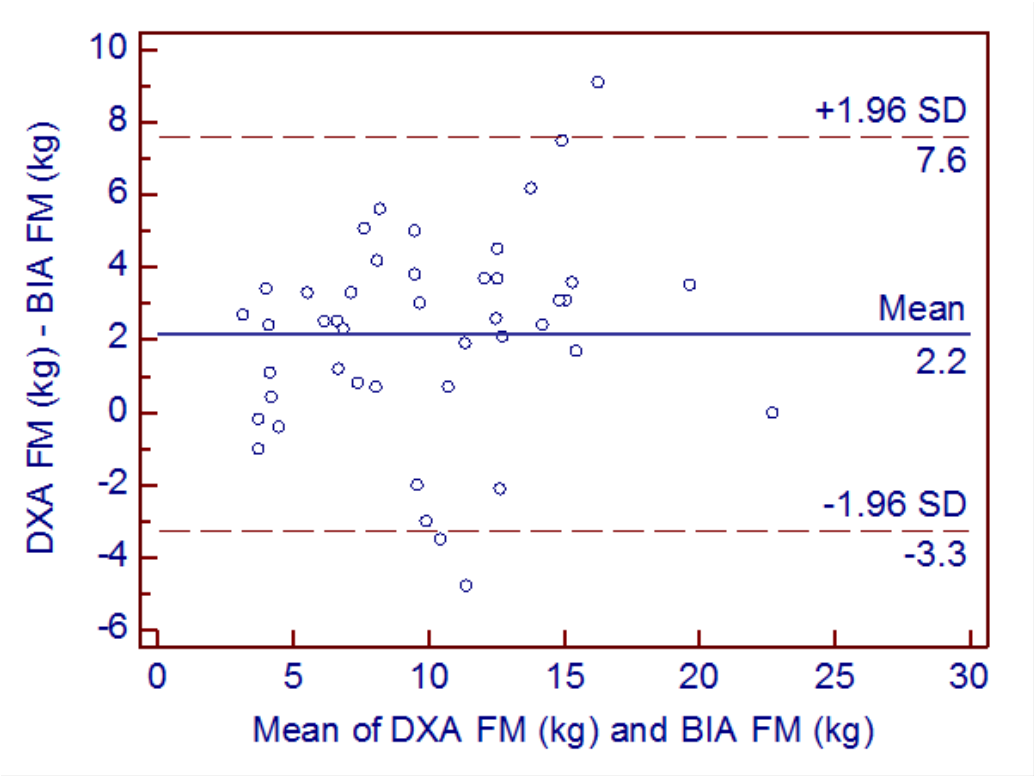

Figure 3(c). Bland-Altman plot for differences in DXA FM (kg) and BIA FM (kg)

The DXA measurements evaluated for Normal distribution by using Kolmogorov-Smirnov test as shown in figure 3(d) gives the p-value, which is greater than 0.1 as shown in table 3(g) indicating that the distribution is approximately normal (Hanneman, 2008). Again the data passed the test of normality as the coefficient of skewnessand kurtosis which are 0.4622 and -0.4717 approximate to zero (Brown, 2011). Whilst the histogram, figure 3(d), appears to be slightly skewed to the left, the points on the normal plot, figure 3(e), almost lie on the straight line indicating normality. It follows that the regression equation for the DXA fat mass against the height and the impedance can be determined.

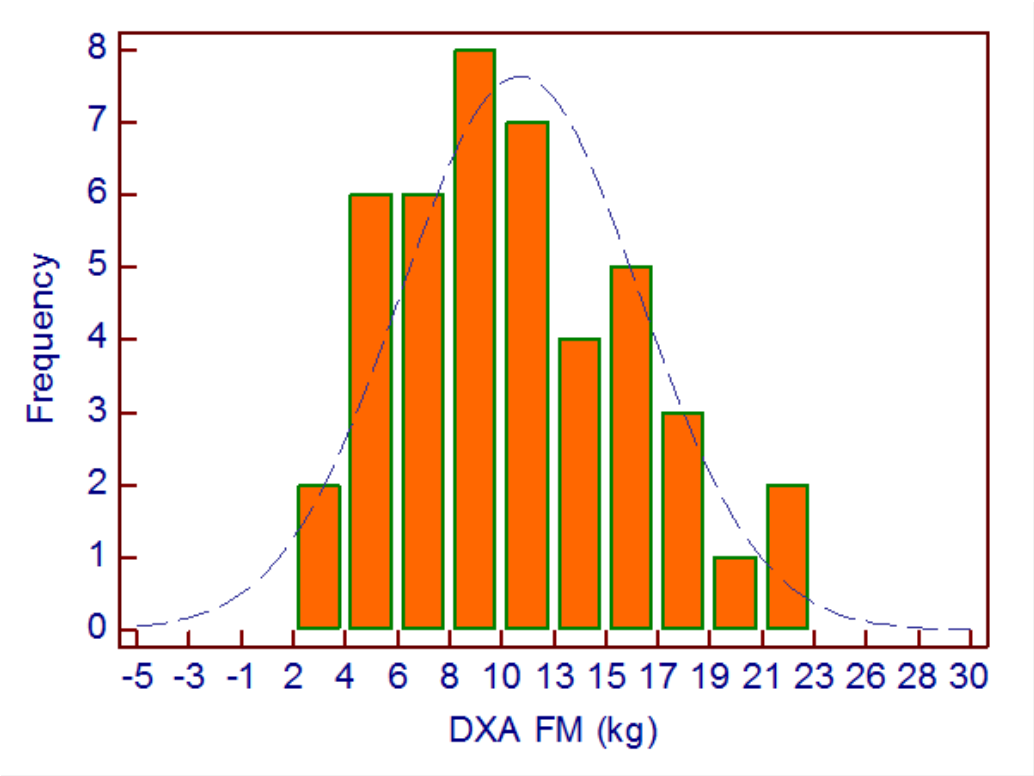

Figure 3(d) Frequency distribution of DXA FM (kg)

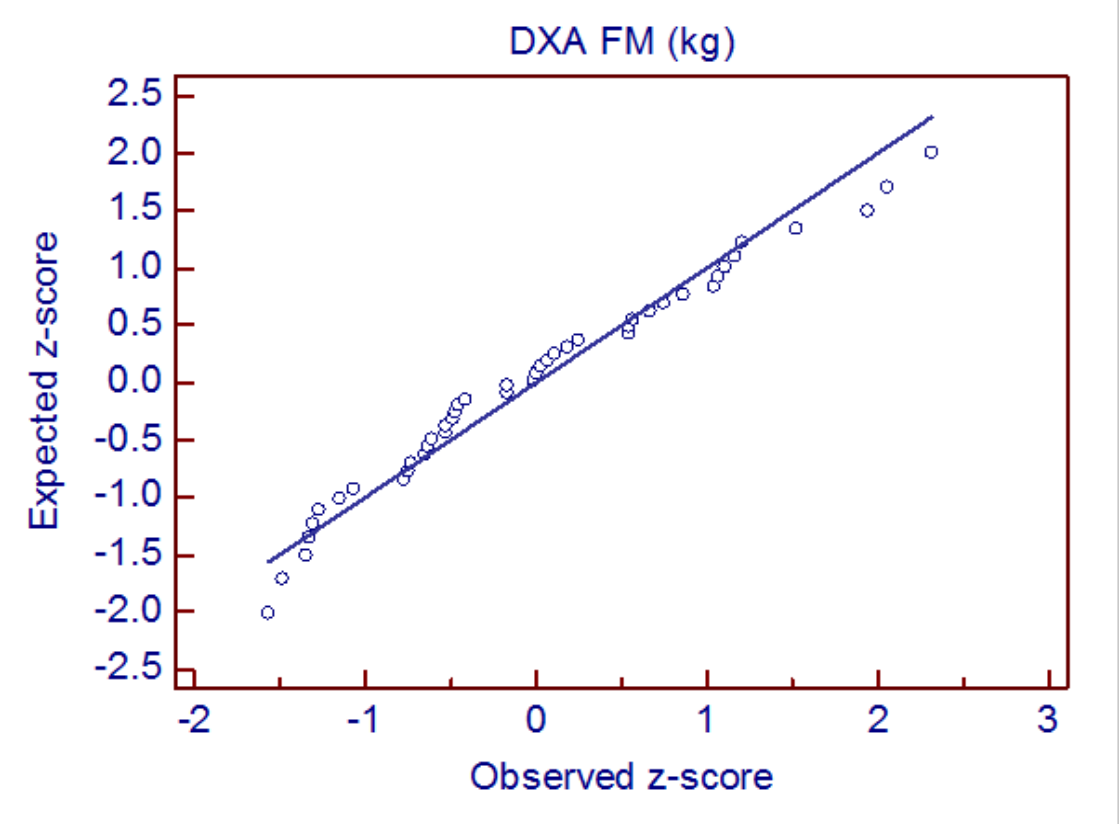

Figure 3(e) Normal Q-Q plot of DXA FM (kg)

Table 3(g) Kolmogorov-Smirnov test DXA FM (kg)

Kolmogorov-Smirnov test for Normal Distribution (DXA)	
Sample size	44
Lowest value	3.2
Highest value	22.7
Arithmetic mean	11.0727
95% CI for the mean	9.5458 to 12.5997
Median	10.6
95% CI for the median	8.4069 to 12.2896
Variance	25.2253
Standard deviation	5.0225
Relative standard deviation	0.4536 (45.36%)
Standard error of the mean	0.7572
Coefficient of Skewness	0.4622 (P=0.1860)
Coefficient of Kurtosis	-0.4717 (P=0.5184)
Kolmogorov-Smirnov test	D=0.1146
for Normal distribution	accept Normality (P>0.10)

Figures 3(f) and 3(g) below are the linear regression plots and equations with their statistical parameters shown in tables 3(g) and 3(h) of the DXA fat mass for the male and female samples.

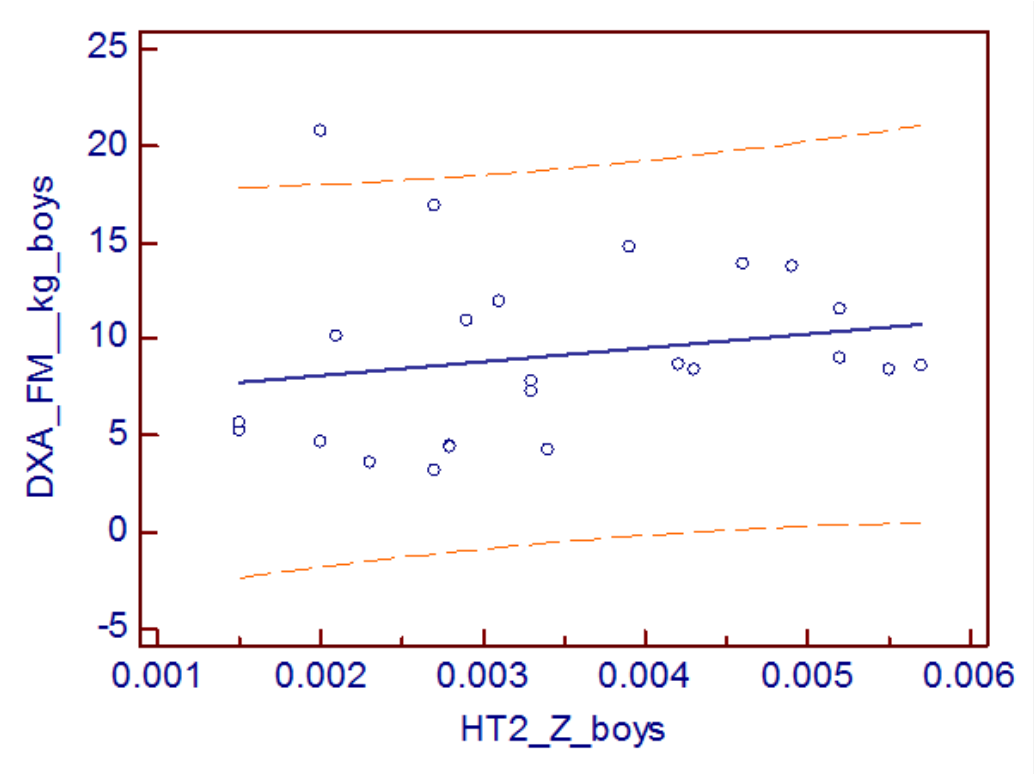

Figure 3(f). Regression line with 95% predictive interval (boys' sample).

Table 3(h). Statistical parameters of DXA FM (kg) regression equation (boy's sample).

Parameter	Coefficient	Std. Error	t- value	p	95% CI	R^2
Intercept	6.6544	2.7031	2.4618	0.0221	1.0486 to 12.2603	0.6041
Slope	722.7624	743.5875	0.9720	0.0052	-819.344 to 2264.869	
Regression Equation (boys sample)	DXA FM(kg) = 6.6544 + 722.7624HT^2/Z					

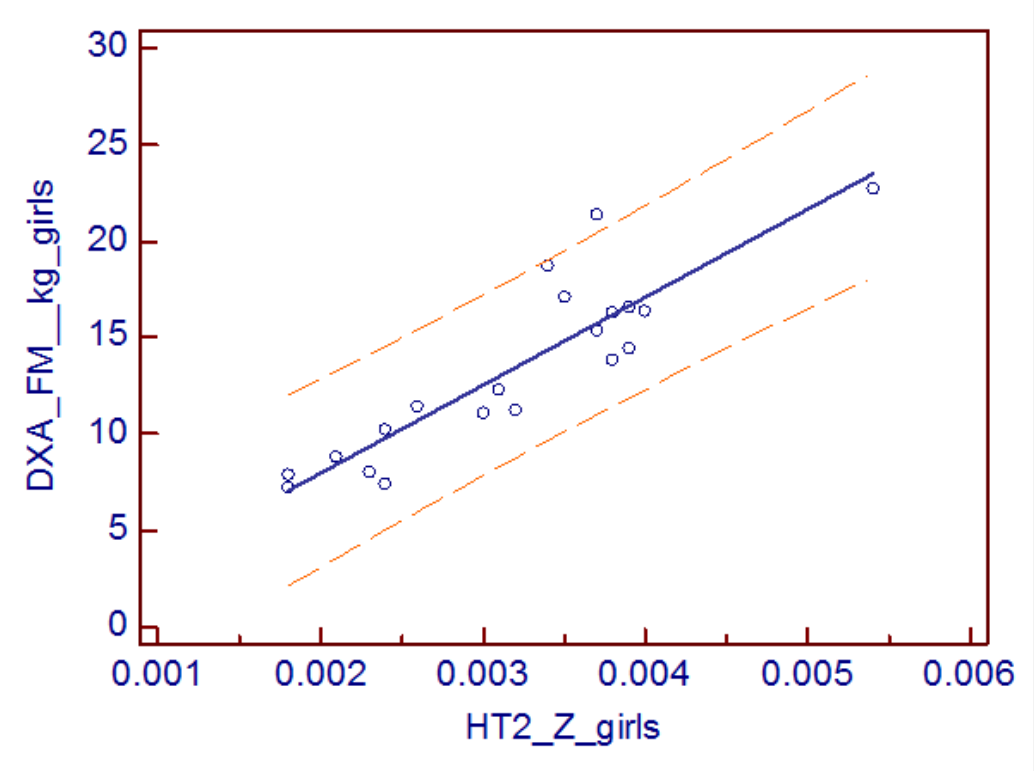

Figure 3(g). Linear Regression line with 95% predictive interval (girls' sample).

Table 3(i). Statistical parameters of DXA FM (kg) regression equation (girls' sample).

Parameter	Coefficient	Std. Error	t- value	p	95% CI	R^2
Intercept	-1.1284	1.8276	-0.6174	0.5447	-4.9682 to 2.7113	0.7910
Slope	4559.0750	552.3642	8.2537	<0.0001	3398.601to 5719.549	
Regression Equation (girls sample)	DXA FM(kg) = -1.1284 + 4559.075HT^2/Z					

3.5 Discussion

This chapter has examined the validity of body fat mass estimates by BIA (Tanita BC418 segmental BC analyser) using DXA (Norland XR8000) as the criterion method and gender specific validation equations produced using regression analysis. The correlation analysis of DXA fat mass and BIA fat mass points to the significance of the association.

Analysis of the Bland-Altman's plot of the differences of the DXA fat mass and BIA fat mass measurements plotted against their means, shows 95.5% of data points lying within the $\pm$ 1.96sd of the mean difference - the 95% tolerance interval for the paired observations. In addition, the cluster of points fairly distributed around the mean of the difference line show an excellent agreement between the two body fat measuring instruments. The Bland-Altman plot and the limits of agreement were not performed on a log scale because of the difficulty in interpreting log-transformed variables in clinical practice (Euser et al, 2008). There was no observed increase in variability of the differences as the magnitude of the measurements of body fat using DXA or BIA increased. There are a number of factors that influence measurement of fat mass as well as fat-free mass by the BIA due to their influence on impedance. These include food and fluid intake, exercise and hydration status (Dahghan et al, 2008). A recent study showed increase in impedance few hours after a meal and another showed a decrease in impedance following moderate to intense exercise (Samani et al, 2006). Furthermore, dehydration or the menstrual cycle can affect the accuracy of impedance measurement (Dehghan and Merchant, 2008). In addition, sexual dimorphism in fat distribution during puberty has been shown to exist even in pre-puberty by advanced technical assessments. The study demonstrated that the gynoid pattern of fat distribution which is different in boys was evident in girls well before the physical signs of puberty were seen

(He et al, 2002), hence the differences in the derived equations for the boys and girls in this study.

The observed variability of BIA estimates against DXA, especially in the male population of this study, is in consonance with previous studies. Validation studies conducted by Hosking et al, (2006), Lukaski et al (2003), Tyrrell et al (2001), Sung et al (2001) and Lazzer (2003) observed that BIA can either overestimate or underestimate body fat in comparison to DXA. This may be attributed to differences in ethnic background, life-style variation, physical development and use of different systems of measurement. The findings of this study confirm this observation to some extent. However, comparison with other studies is a challenge as only a few have validated BIA directly against DXA in young children and there is no observable validated studies conducted in children of black descent.

Finally, this study has shown that the BIA device can specifically be used to measure body fat in children of black descent as it has now been validated using the DXA, the gold standard criterion.

Study Limitation

A total sample of forty-four African and Caribbean children was obtained for the validation study. Although this is feasible and in line with similar validation studies conducted in the past (Pietrobelli et al, 2004; Kushner et al, 1990, Lu et al, 2003), a larger representative sample would have been ideal. Additionally, due to health and safety and ethical issues, it was not appropriate to perform duplicate DXA scans on the volunteers. Repeated scans could have improved the precision of the measurements, giving greater validity to the assessment.

Chapter 4: Derivation of skeletal muscle mass (SMM) equations to validate the BIA system

4.1 Introduction

Sarcopenia, which is defined as progressive decline of skeletal muscle mass (SMM), strength and function, has been found to be associated with some forms of obesity, although a greater amount of SMM is often associated with obesity (Janssen, 2006). It has been found that sarcopenic obese individuals have a higher risk of developing metabolic syndrome compared with just obese or sarcopenic individuals (Schrager et al, 2006, Nair, 2005).

Consequently, an individual with high fat mass and low muscle mass seems likely to have more functional limitations and metabolic disorders and therefore it is appropriate to consider obesity together with sarcopenia. Sarcopenia and sarcopenic obesity have been found to have their origin in early life, leading to adverse metabolic health problems later in life (Sayer et al, 2008). This is because low muscle mass is associated with poor metabolic health and the amount of muscle mass in the body is directly related to insulin sensitivity both in children and adults (McCarthy et al, 2011). Skeletal muscle is responsible for more than 75% of all insulin-mediated glucose disposal and it is the main tissue for whole-body glucose balance. Therefore, insulin resistance at this site or low SMM is detrimental for glucose homeostasis (Steene et al, 2009; Benson et al, 2006). Measurement of skeletal muscle mass in children and adults is an important component of nutritional assessment and metabolic health. However the use of SMM measurement for monitoring has been a challenge in the absence of adequate population data that identifies individuals across the age spectrum with high or low amounts of SMM. This is required both for clinical management of individuals as well as for

longitudinal and cross-sectional surveillance of populations and this data should be collected from childhood to adulthood.

In this chapter the terms SMM and SMMa are used interchangeably, the rationale being that skeletal muscle in the upper and lower limbs accounts for more than 75% of whole body SMM in adults and is the major fraction of whole body SMM involved in ambulation and physical activities. It is also the more modifiable fraction of whole body SMM which can be built up or lost (Synder et al, 1975).

SMM monitoring percentile charts have been developed for Caucasian children (McCarthy, 2011) since the BIA can be used to predict SMM in these children. Most BIA systems have only been validated for Caucasians and they may not give accurate prediction of body composition parameters such as SMM in non-Caucasian populations. This is because the chemical composition of the body varies among different ethnic groups and this is especially so in the case of the fat free mass, including SMM. This chapter looks at the validation of the Tanita BIA system (using DXA as the criterion method) for SMM measurement in African and Caribbean children.

4.2 Aim

To validate Bio-electric Impedance Analysis (BIA Tanita BC418) system using the Dual Energy X-ray Absorptiometry (DXA) for skeletal muscle mass measurement in African and Caribbean children.

Objective:

- To derive gender specific validation equations using regression analysis after determination of association between the BIA and DXA - the gold standard criterion.

4.3 Subjects/Methods

Body composition parameters including SMM of the 44 black children aged 5-18 years (24 boys & 20 girls, mean age 10.5, SD 3.96 were measured using BIA (Tanita BC418 segmental BC analyser) and DXA (Norland XR8000) after anthropometric measurements had been taken. Procedures for measurements are detailed in chapter 2 sections 1.11 and 1.12 above.

To derive appendicular skeletal muscle mass (SMMa) data, skeletal muscle mass from the four limbs were extracted and added together for each subject. This was performed for both DXA and BIA.

4.3.1 Statistical analysis

The anthropometric data as well as measurements from BIA and DXA were collated onto an Excel spreadsheet and exported to SPSS (version 19) to generate descriptive statistics of the study population. In the same way as the fat mass correction/validation equations were derived in chapter 3, to derive SMMa validation equations, first the association between the two measuring systems was determined using Pearson's correlation analysis. Since correlation does not fully establish agreement nor identify bias, the data was tested for normality in order to use the Bland-Altman analysis to identify bias and also to show agreement between the two measuring systems. To test for normality, the Kolmogorov-Smirnov test for normal distribution/histogram was used. This test was performed twice in this chapter:

1) Before using the Bland Altman analysis and
2) Before deriving the gender specific validation equations - to test DXA measurements (the dependant variable of the equation).

A line of equality was plotted to provide a visual impression of the association between the two SMMa measuring equipment - DXA and BIA. Then the first test of normality was performed and the data passed this test of normality. After examination of the data through the use of scatter diagrams and frequency distribution for the test of normality, it provided the platform to consider the study using the limit of agreement – a Bland-Altman plot. Bland-Altman plot for the differences between DXA and BIA for the study was performed using MedCal software (version 12.2.1.0). Then, the DXA measurements which constitute the dependent variables were evaluated for normal distribution using Kolmogorov-Smirnov test and normal Q-Q plot. When the DXA measurements passed the test of normality, i.e. the correction/validation equations for the sample population, the regression of DXA SMMa against height and impedance was produced for the boys and girls using MedCal software (version 12.2.1.0).

4.4 Results

The descriptive statistics for the sample population is summarised in figures 3(a) to 3(d). Generally, for all other anthropometric and body composition, measures including SMM increased with advancing age for both boys and girls.

To obtain a visual impression of association (Bland and Altman, 1986) between the DXA SMMa (kg) and the BIA SMMa (kg), a line of equality was plotted. Figure 4(a) shows the scatter plot with the line of equality – the line on which all points would lie if there was a perfect agreement between the two measuring systems (Hanneman, 2008).

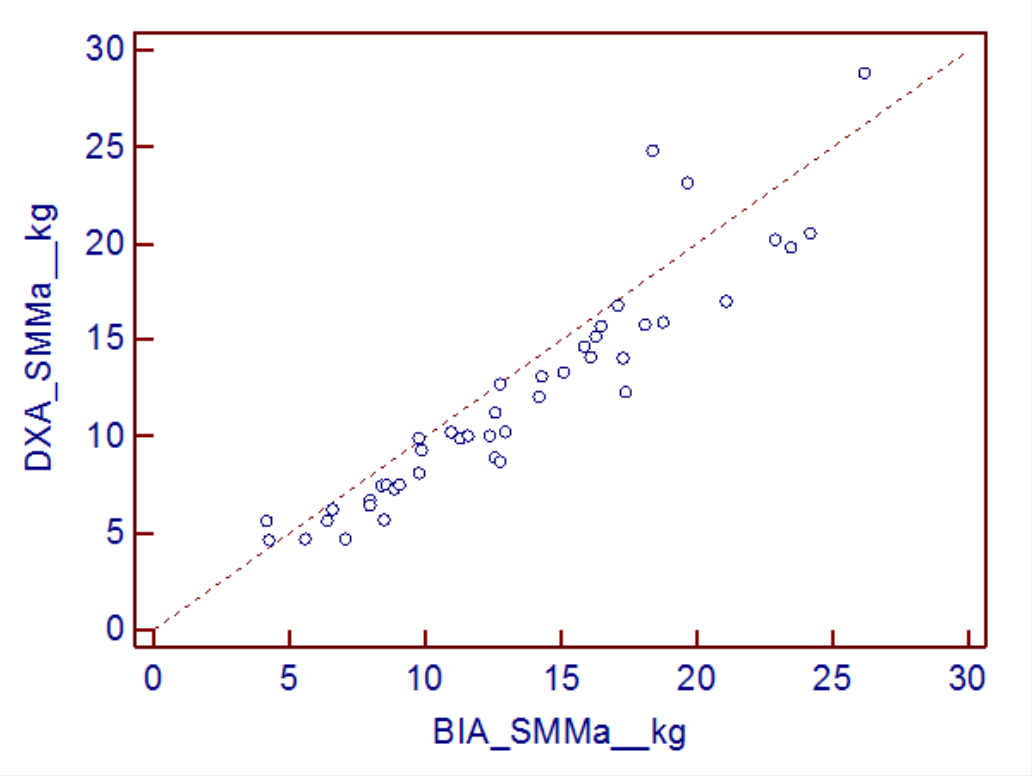

Figure 4(a). Line of equality DXA SMMa (kg) against BIA SMMa (kg).

The correlation coefficient (r) is 0.94 and the significance level p is less than 0.0001 which shows a good linear relationship between the two measures, and also points to the statistical significance of the association showing that the two methods are related (Bland and Altman, 1986; Chan, 2003). The coefficient of determination (the square of r) is 0.88 which shows 88% of common proportion of variance between DXA and BIA fat mass measurement (Chan, 2003). Table 4(a) provides the results of Pearson's correlation analysis.

Table 4(a) Pearson's correlation between DXA SMMa (kg) against BIA SMMa (kg)

Pearson's Correlation between DXA SMMa(kg) and BIA SMMa(kg)	
Sample size	44
Correlation coefficient r	0.9351
Significance level	P<0.0001
95% Confidence interval for r	0.8836 to 0.9643

Figure 4(b) shows the results of Kolmogorov-Smirnov test for normal distribution of DXA and BIA SMMa. As shown in table 4(b), the p value of the Kolmogorov-Smirnov test is 0.0712. The p value is thus greater than 0.05 indicating that the distribution is an accepted normality (Hanneman, 2008). In addition, the data passes the test of normal distribution because the coefficient of skewness and kurtosis, which are 0.4776 and 0.8236 respectively, are approximate to zero (Brown, 2011). Hence the variables can be described by means of the mean and standard deviation and also subjected to parametric statistical test/Bland Altman analysis (Hanneman, 2008). Therefore it is expected that 95% or more of the measured subjects will fall within the Bland-Altman limits of agreement as shown in figure 4(c) (Hanneman, 2008).

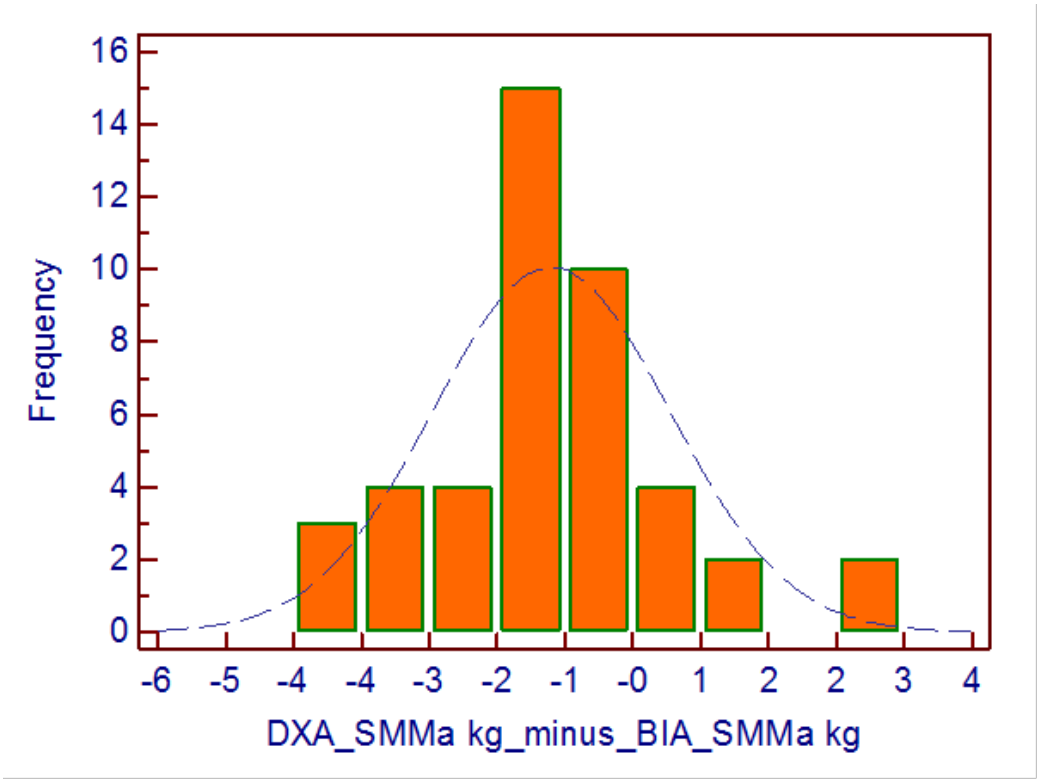

Figure 4(b) Frequency distribution of the difference between DXA SMMa (kg) and BIA SMMa (kg)

Table 4(b) Kolmogorov-Smirnov test between DXA SMMa (kg) and BIA SMMa (kg)

Kolmogorov-Smirnov test for Normal Distribution (DXA-BIA)	
Sample size	44
Lowest value	-4.1
Highest value	2.6
Arithmetic mean	-1.1682
95% CI for the mean	-1.6100 to -0.7264
Median	-1.15
95% CI for the median	-1.6000 to -0.8035
Variance	2.1115
Standard deviation	1.4531
Relative standard deviation	-1.2439 (-124.39%)
Standard error of the mean	0.2191
Coefficient of Skewness	0.4776 (P=0.1726)
Coefficient of Kurtosis	0.8236 (P=0.2211)
Kolmogorov-Smirnov test[a]	D=0.1273
for Normal distribution	accept Normality (P=0.0712)

The Bland-Altman plot of the differences between DXA SMMa and BIA SMMa against mean of DXA SMMa and BIA SMMa as indicated in figure 4(c) below. The plot shows a good relationship between the differences with no proportional error. The observed differences were both negative and positive differences, thus there was no systematic bias between the measurements. The 95% tolerance interval for the paired observations, the limits of agreement, is the mean difference plus or minus 1.96 times the standard deviation of the difference. These represent the range of values satisfied by the agreement between the use of DXA and BIA, and figure 4(c) shows almost 95.5% of the variables within the limit of agreement (Bland and Altman, 1986; Hanneman, 2008).

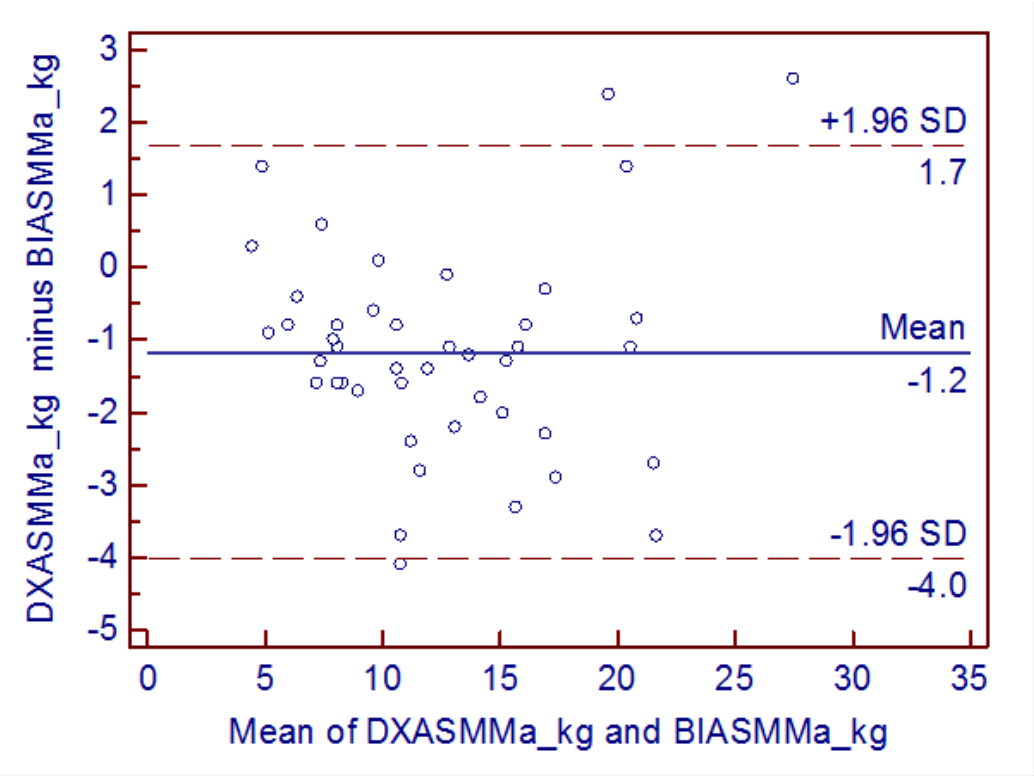

Figure 4(c). Bland-Altman plot for differences in DXA SMMa (kg) and BIA SMMa (kg).

The DXA measurements evaluated for Normal distribution by using the Kolmogorov-Smirnov test as shown in figure 4(d) gave the p = 0.0619. This p value is greater than 0.05 as shown in table 4(c), indicating no significant difference, and thus the distribution is approximately normal (Hanneman, 2008). Again the data passed the test of normality as the coefficient of skewness and kurtosis which are 0.4234 and 0.3402, thus approximate to zero (Brown, 2011). The points on the normal plot, figure 4(e), almost lie on the straight line indicating normality. It follows that the regression equation for the DXA appendicular skeletal muscle mass (SMMa) against height and impedance can be determined.

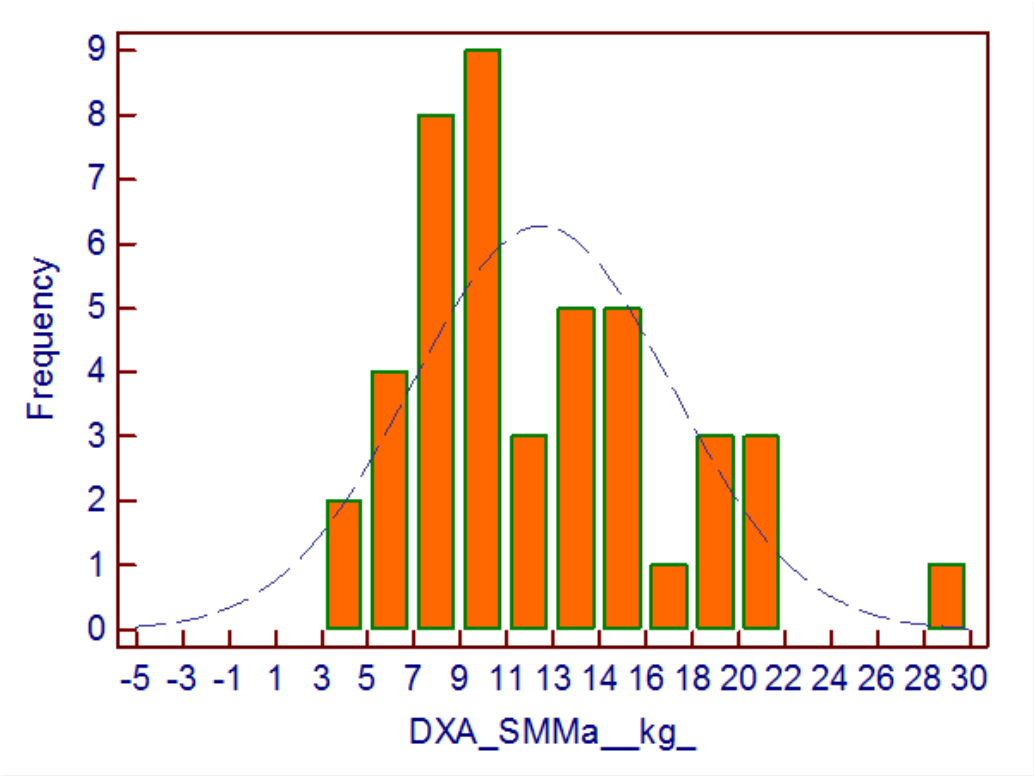

Figure 4(d) Frequency distribution of DXA SMMa (kg)

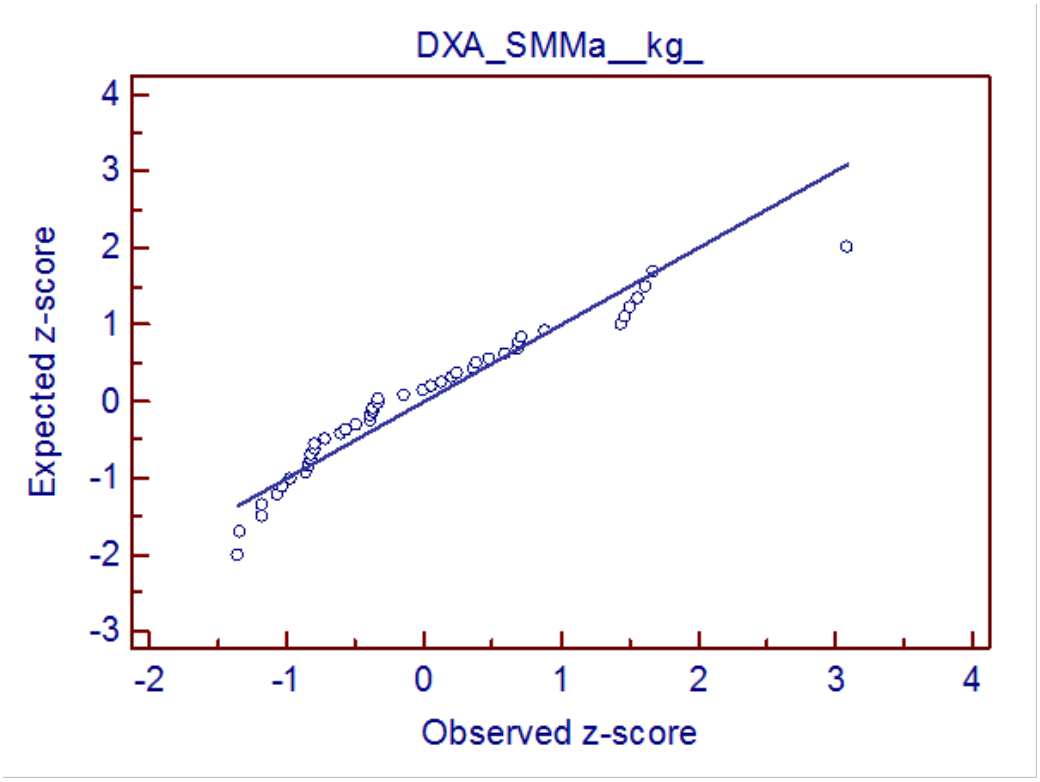

Figure 4(e) Normal Q-Q plot of DXA SMMa (kg)

Table 4(c) Kolmogorov-Smirnov test DXA SMMa (kg)

Kolmogorov-Smirnov test for Normal Distribution (DXA)	
Sample size	44
Lowest value	4.6
Highest value	28.8
Arithmetic mean	12
95% CI for the mean	10.3459 to 13.6541
Median	10.2
95% CI for the median	8.9139 to 13.2931
Variance	29.6005
Standard deviation	5.4406
Relative standard deviation	0.4534 (45.34%)
Standard error of the mean	0.8202
Coefficient of Skewness	0.4234 (P=0.0139)
Coefficient of Kurtosis	0.3402 (P=0.2993)
Kolmogorov-Smirnov test[a]	D=0.1523
for Normal distribution	accept Normality (P=0.0619)

Figures 4(f) to 4(g) below are the linear regression plots and equations with their statistical parameters shown in tables 4(d) and 4(e) of the DXA SMMa (kg) for the boys' and girls' samples.

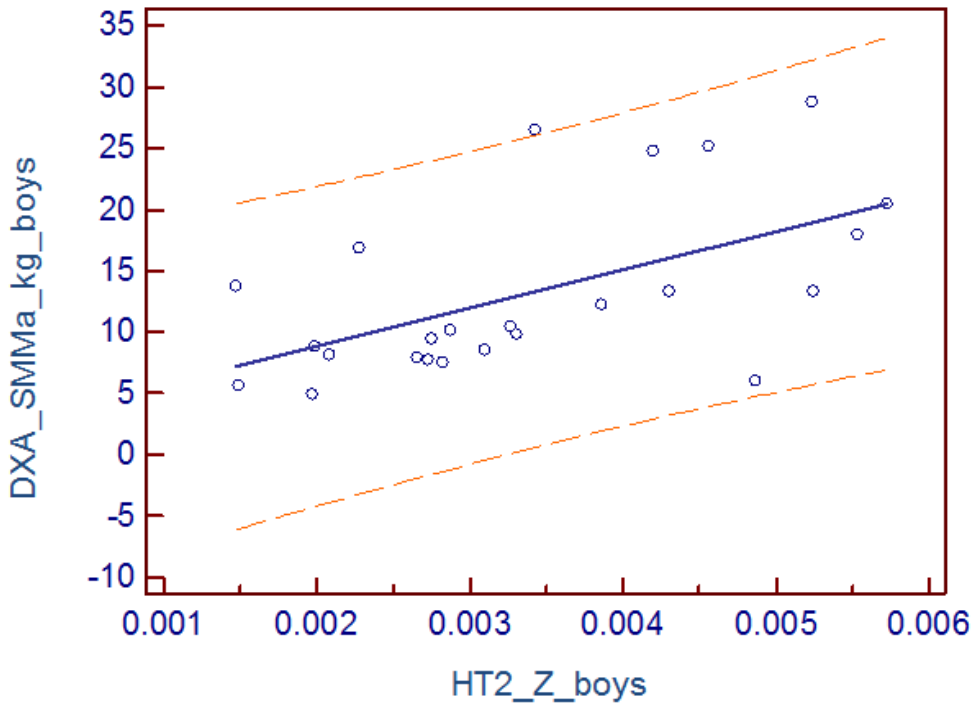

Figure 4(g). Linear Regression line with 95% predictive interval (boys' sample)

Table 4(d). Statistical parameters of DXA SMMa (kg) regression equation (boys' sample).

Parameter	Coefficient	Std. Error	t- value	p	95% CI	R^2
Intercept	2.6054	3.5268	0.7387	0.4679	-4.7087 to 9.9195	0.3206
Slope	3126.6858	970.4376	3.2219	0.0039	1114.121 to 5139.2502	
Regression Equation (boys sample)	DXA SMMa(kg) = 2.6054 + 3126.6858HT^2/Z					

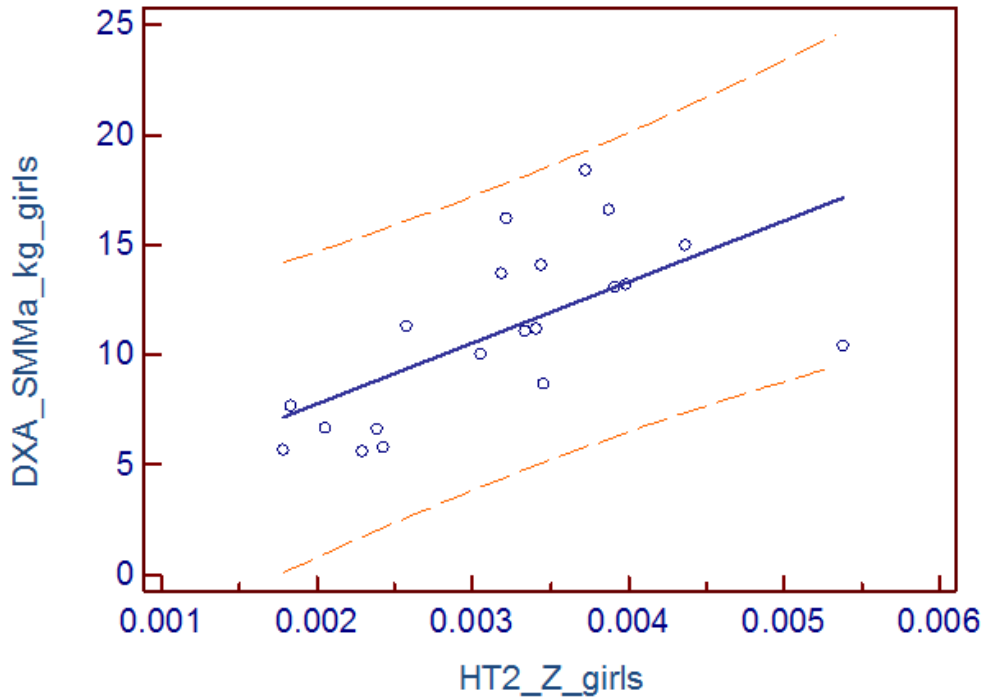

Figure 4(i). Linear Regression line with 95% predictive interval (girls' sample).

Table 4(d). Statistical parameters of DXA SMMa (kg) regression equation (girls' sample).

Parameter	Coefficient	Std. Error	t- value	p	95% CI	R^2
Intercept	2.2189	2.5709	0.8631	0.3994	-3.1823 to 7.6201	0.4144
Slope	2774.3250	777.3365	3.5690	0.0022	1141.201 to 4407.448	
Regression Equation (girls sample)	DXA SMMa(kg) = 2.2189 + 2774.325 HT^2/Z					

4.5 Discussion

Gender specific SMMa validation equations (for African and Caribbean children) have been produced using linear regression analysis after determining the association

between DXA and BIA measurements. This chapter also affirms the validity of the use of DXA as a criterion method for validation of the BIA as a positive association between the two equipments was observed for the SMM estimates measured by the two methods.

The results indicate that DXA SMMa estimates were strongly correlated to the BIA derived ht^2/R. All the correlation plots show an excellent agreement between the DXA and BIA. Studies conducted in the past support the validity of DXA estimates of SMM (Heymfield et al, 1990; Wang et al, 1996). However, its use for field studies is limited by measurement cost, technician skill, portability and sophistication (Kyle et al, 2003). BIA has been used extensively to determine fat free mass, and studies have shown a good correlation between its SMM electrical resistance and DXA SMM (Nunez et al, 1999; Pietrobelli et al, 1998). This suggests that BIA SMM can be estimated from BIA-measured resistance, as the equations derived above demonstrate, and they would correlate well with DXA estimates.

Generally, the Tanita BC-418 system BIA overestimated fat-free mass compared with DXA which was observed in this study in the case of SMM which forms part of the fat-free mass (Hosking et al, 2006). For the BIA, SMM estimates for girls ranged from 7.1kg to 16.8kg whereas DXA estimates ranged from 5.7kg to 16.3kg and for the boys, BIA estimates the range from 7.6kg to 23.8kg, whereas DXA estimates the range from 6.9kg to 22.1kg, with the boys having higher SMM compared with the girls. Studies have confirmed the prediction for SMM to be higher in males than in females (Kyle et al, 2003). Heymsfield asserted the fact that during growth and especially around puberty, boys develop a proportionately greater skeletal muscle mass and bone mass than girls - changes which bring about sexual dimorphism in girls and boys. Conversely girls develop a proportionately greater fat mass around puberty – both changes being

gender driven consequences of testosterone and oestrogen (Heymsfield, 2005). These and other factors including menstruation, hydration status and exercise, which influence impedance, as explained in chapter three, resulted in the difference in the gender equations above.

To date, no other study has examined skeletal muscle mass in African-Caribbean children using BIA as the assessment tool. It is thus difficult to make comparisons with other studies. However it is encouraging to note that the corrected BIA-derived values obtained in this study compare well with the study by McCarthy *et al* (2014) with the SMMa values in the African-Caribbean children being consistently and slightly higher compared with age- and gender-equivalent white European children. There is a need for this work to be replicated in sub-Saharan, African-American and Caribbean populations and begin to be used in both clinical and epidemiological studies. This will allow cross-cultural comparisons to be made. The bonus of having SMMa reference curves for African-Caribbean children is that they will help identify children who have low SMMa for their age and gender and are potentially at risk from metabolic disease and later sarcopenia. Intervention, such as strength-gaining exercise (at an appropriate age) in order to boost SMMa, could be initiated. However, due to limited assessment tools, it is unknown whether such forms of intervention are able to enhance muscle gain. To reiterate, it was felt appropriate to use SMMa as a proxy for whole body SMM as SMMa account for approximately 75% of whole body SMM. To confirm this assertion, the observations by McCarthy et al (2014) reflect 74% (boys) and 81% (girls) of the whole body SMM determined by DXA in a similar population group (Kim et al, 2006; Wang et al, 2007).

In conclusion, although the two devices are not directly interchangeable, when validated, the BIA system is preferable for large scale epidemiological studies in

childhood populations because of its simplicity, lower cost, non-invasiveness and speed for use in children. However, further validation studies would help to increase its use in even the very young children.

Based on the derived fat mass and SMMa validation equations in chapters 3 and 4, percentile charts for these variables in African and Caribbean children have been produced as illustrated in the following chapters.

Study Limitations

A total sample of forty-four African and Caribbean children was recruited for the validation study. Although this is feasible and in line with similar validation studies conducted in the past (Pietrobelli et al, 2004; Kushner et al, 1990; Lu et al, 2003), a larger representative sample would have been ideal. Additionally, due to ethical and health and safety concerns, only single DXA scans were performed on each child. It is unclear how consecutive scans vary in their body composition predictions, although the literature suggests a high precision for this technique.

Chapter 5: Per cent Fat-Mass (FM) centile curves/charts for African-Caribbean Children

5.1 Introduction

Growth charts or centile reference charts and tables are used in child health nutritional practice and to track clinical observations/measurements of individual children in the context of population values over time. When a child's centile value is atypical or falls below or above the lowest and highest percentiles, this could indicate an underlying pathological condition such as growth disorder, wasting or obesity. Measurement of fat mass gives assessment of adiposity, the aspect of obesity which leads to pathology and this is a significant advancement over the use of body mass index. Percentage fat mass percentile curves have been produced for UK Caucasian children (McCarthy et al, 2006).

This chapter describes the production of fat mass percentile curves for African and Caribbean children living in the UK. To date, no equivalent charts exist for the paediatric population from other minority ethnic groups including African, Caribbean and South Asian children. However, due to differences in body composition of people due to their ethnic origin, ethnicity specific body charts are required for accurate assessment of body parameters. These new percentile charts should help to monitor body fatness in children of black descent.

5.2 Aim

To develop gender specific per cent fat mass (%FM) percentile charts for African and Caribbean children aged 5 to 18 years living in the UK.

5.3 Methods

5.3.1 Subjects and Anthropometry

In this work of developing %fat mass (%FM) percentile charts for African and Caribbean children, body composition data measured by the use of segmental bioelectrical impedance analysis (BIA, Tanita BC418) on 1,336 African and Caribbean children aged 5 to 16 years was extracted from an existing dataset collected from twenty one schools in London and South East England. The choice of the schools and specific London boroughs was based on information from the 2001 census (Office of National Statistics- Census 2001). Thus the boroughs of Hackney, Islington and Newham, which were densely populated with children from ethnic minority groups including Africans and Caribbeans, were used. Measurements were taken by trained and qualified research assistants at London Met to ensure accuracy in measurements. Specifically, height was measured by a single observer and waist circumference was measured by a separate single observer. Thus inter-observer errors were eliminated. Ethnicity was recorded at the school level using the DfES recording system. All children who were eligible to take part on the study were measured except where consent was refused. Measurements were taken at any time of the school day and children were not asked to empty bladders. Thus the measurement conditions reflected likely conditions in a clinical situation. Questions about any medication taken were not asked, again reflecting the real world situation. Measurement procedures were exactly as described in chapter 2. Out of the total number of children from African and Caribbean background, fifty two per cent were girls and forty eight per cent were boys. As indicated above in chapter three, most BIA systems have only been validated in Caucasian population and these validated equations most likely cannot predict body composition accurately in African and Caribbean children and for this reason, DXA was used to validate the BIA derived %BF values as

described in chapter three. The predictive gender specific validated equations of per cent fat mass in relation to height and impedance derived were applied to the existing extracted data set and the corrected %BF values used to develop per cent fat mass centile curves for the boys and girls. The spreadsheet was checked for errors, omissions and any inconsistencies in the data. Where measurements were found to lie outside +/- 4SD, they were re-checked and removed where appropriate. This only occurred in a very small minority of cases.

5.3.2 Statistical Analysis

For the body composition measurements, descriptive statistics were calculated and expressed as mean $\pm$ SD using SPSS version 18 (SPSS Inc, Chicago, IL, USA). Per cent fat mass centile curves for boys and girls were generated based on the original uncorrected data and corrected BIA data produced from the validated data set using DXA. The smoothed centile curves for fat mass were constructed separately for boys and girls using the LMS method which summarises the data in terms of three smooth age-specific curves, namely L (lambda), M (mu), and S (sigma) (Pan and Cole, 2011). The M and S curves correspond to the median and coefficient of variation of fat mass at each age whereas the L curve allows for the age dependent skewness in the distribution. For the construction of the centile curves, data were imported into the LMS chartmaker software (version 2.54) and the software was used to generate the curves using the measurement. Seven centile curves were calculated from the 2^{nd} to the 98^{th}, spaced two-thirds of a standard deviation score apart, as in the layout used in other British growth reference charts (Cole, 1994).

5.4 Results

Tables 5(a) and 5(b) show the descriptive statistics for the pre and post corrected data presented in narrow ages ranges (5-7, 8-10, 11-13, 14-16) for the African and Caribbean children. Tables 5(c) to 5(f) and figures 5(a) to 5(d) illustrate the tabulated per cent fat mass (%FM) centile values by exact age with their corresponding per cent fat mass (%FM) centile charts for boys and girls before and after application of the validated equation to the data set. In each chart, the curves represent the 2nd, 9th, 25th, 50th, 75th, 91st and 98th centiles.

Table 5(a): Descriptive statistics for the Boys' Sample Population of African and Caribbean children.

BOYS		*Before Validation*										Values = ± SD
Age (yrs)	n	*Height* (cm)	*Weight* (kg)	*BMI* (kg/m^2)	*FM* (kg)	*FM* (%)	*FFM* (kg)	*FFM* (%)	*SMM* (kg)	*SMM* (%)	*(SMM/FFM)* (%)	*MFR*
5 - 7	227	122.2	24.6	16.3	5.1	19.7	19.5	80.3	6.5	26.2	32.7	1.40
		7.21	6.55	2.86	3.09	5.08	3.80	5.09	2.07	3.02	4.08	0.35
8 - 10	248	139.3	35.7	18.2	8.2	21.5	27.5	78.5	10.5	29.6	37.5	1.50
		8.08	9.93	3.56	4.96	6.54	5.53	6.56	2.94	2.61	3.74	0.50
11- 13	137	155.0	47.3	19.5	10.5	20.9	36.8	79.1	15.1	32.0	40.5	1.71
		9.99	12.48	3.95	6.50	7.05	7.54	7.05	4.00	3.20	2.78	0.58
14 - 16	32	169.8	59.4	20.5	11.5	18.6	47.9	81.4	19.8	33.5	41.2	1.95
		8.36	12.73	3.74	5.97	5.48	7.94	5.47	3.97	2.56	1.79	0.56

BOYS		*After Validation*										Values = ± SD
Age (yrs)	n	*Height* (cm)	*Weight* (kg)	*BMI* (kg/m^2)	*FM* (kg)	*FM* (%)	*FFM* (kg)	*FFM* (%)	*SMM* (kg)	*SMM* (%)	*(SMM/FFM)* (%)	*MFR*
5 - 7	227	122.2	24.6	16.3	8.1	16.2	16.3	83.2	6.3	27.6	36.2	0.78
		7.21	6.55	2.86	2.06	3.06	4.89	2.20	1.82	0.59	4.02	0.19
8 - 10	248	139.3	35.7	18.2	9.7	20.8	25.3	80.6	9.6	28.7	40.7	1.05
		8.08	9.93	3.56	4.04	3.40	6.37	3.65	2.36	0.77	2.91	0.27
11- 13	137	155.0	47.3	19.5	11.3	20.8	35.4	79.2	13.4	29.9	43.5	1.33
		9.99	12.48	3.95	5.99	4.02	9.95	3.83	3.69	1.20	2.78	0.46
14 - 16	32	169.8	59.4	20.5	11.8	16.2	47.5	83.8	17.8	31.3	43.8	1.67
		8.36	12.73	3.74	5.67	3.79	9.83	3.79	3.65	1.19	1.97	0.48

Table 5(b): Descriptive statistics for the Girls' Sample Population of African and Caribbean children.

GIRLS		*Before Validation*										Values = ± SD
Age (yrs)	n	*Height* (cm)	*Weight* (kg)	*BMI* (kg/m^2)	*FM* (kg)	*FM* (%)	*FFM* (kg)	*FFM* (%)	*SMM* (kg)	*SMM* (%)	*(SMM/FFM)* (%)	*MFR*
5 - 7	222	123.0	25.7	16.7	6.1	22.7	19.6	77.3	6.7	26.1	33.9	1.20
		7.68	6.64	2.90	2.94	4.61	3.96	4.61	1.74	1.93	2.18	0.27
8 - 10	276	140.5	38.2	19.0	10.3	25.5	27.8	74.5	10.2	27.0	36.3	1.14
		9.07	11.44	4.12	5.69	6.60	6.38	6.60	2.85	2.34	2.27	0.34
11- 13	169	155.5	49.0	20.2	13.2	26.0	35.8	74.0	13.5	27.7	37.5	1.13
		7.58	11.28	4.16	5.64	5.79	6.44	5.79	2.83	2.18	1.49	0.32
14 - 16	27	161.5	56.5	21.7	16.8	29.1	39.7	71.0	15.1	26.9	38.0	0.97
		6.38	8.61	3.21	5.77	5.66	4.15	5.64	1.94	1.82	1.73	0.23

GIRLS		*After Validation*										Values = ± SD
Age (yrs)	n	*Height* (cm)	*Weight* (kg)	*BMI* (kg/m^2)	*FM* (kg)	*FM* (%)	*FFM* (kg)	*FFM* (%)	*SMM* (kg)	*SMM* (%)	*(SMM/FFM)* (%)	*MFR*
5 - 7	222	123.0	25.7	16.7	7.6	29.2	19.2	70.8	6.2	23.1	32.5	0.82
		7.68	6.64	2.90	1.98	0.68	3.20	0.68	1.50	0.60	1.30	0.02
8 - 10	276	140.5	38.2	19.0	11.4	30.5	25.3	69.5	9.1	24.2	35.0	0.80
		9.07	11.44	4.12	3.02	1.04	4.87	1.04	2.29	0.91	1.98	0.01
11- 13	169	155.5	49.0	20.2	14.9	31.8	31.1	68.2	11.8	25.3	37.3	0.79
		7.58	11.28	4.16	3.16	1.09	5.10	1.09	2.40	0.96	2.07	0.01
14 - 16	27	161.5	56.5	21.7	15.6	32.0	32.2	68.0	12.3	25.5	37.8	0.79
		6.38	8.61	3.21	2.29	0.79	3.70	0.79	1.74	0.69	1.50	0.00

Table 5(c). Tabulated Boys' per cent fat mass (%FM) centile values by exact age before the application of validated equation

Boys	FM (%) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	13.5	14.9	16.5	18.4	20.8	23.9	28.3
6	13.3	14.8	16.5	18.7	21.5	25.2	30.8
7	13.1	14.7	16.5	19.0	22.2	26.6	33.8
8	12.9	14.6	16.6	19.3	22.9	28.1	36.8
9	12.9	14.6	16.7	19.6	23.6	29.4	39.6
10	12.8	14.6	16.8	19.8	24.0	30.3	41.8
11	12.6	14.4	16.7	19.7	24.1	30.7	43.1
12	12.4	14.2	16.4	19.5	23.9	30.6	43.6
13	12.1	13.8	16.0	19.1	23.5	30.2	43.3
14	11.7	13.5	15.7	18.6	23.0	29.6	42.7
15	11.4	13.2	15.3	18.2	22.4	29.0	41.9
16	11.2	12.8	14.9	17.8	21.9	28.4	41.0

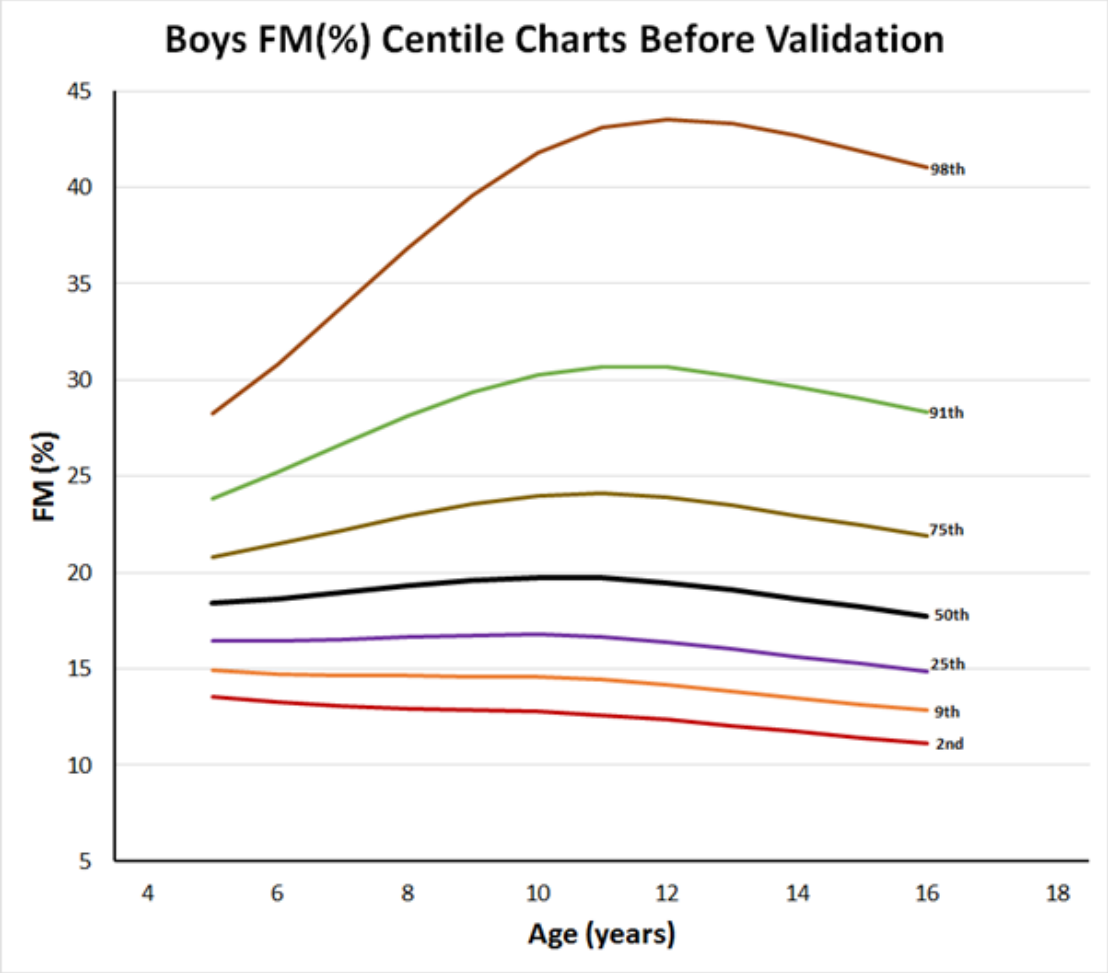

Figure 5(a). %FM centile charts for Boys before the application of validated equation

Table 5(d). Tabulated Boys per cent fat mass (%FM) centile values by exact age after the application of validated equation

Boys	FM (%) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	8.2	10.8	12.8	14.5	16.0	17.4	18.7
6	9.0	11.9	14.1	16.0	17.6	19.1	20.6
7	9.8	13.0	15.4	17.5	19.3	20.9	22.5
8	10.6	14.1	16.6	18.8	20.8	22.6	24.3
9	11.2	14.9	17.6	20.0	22.1	23.9	25.7
10	11.7	15.5	18.3	20.8	22.9	24.9	26.8
11	11.9	15.8	18.7	21.2	23.4	25.4	27.4
12	12.0	15.9	18.8	21.3	23.5	25.5	27.4
13	11.9	15.7	18.6	21.1	23.3	25.2	27.2
14	11.6	15.5	18.3	20.7	22.8	24.8	26.7
15	11.4	15.1	17.9	20.2	22.4	24.2	26.1
16	11.1	14.8	17.5	19.8	21.8	23.7	25.5

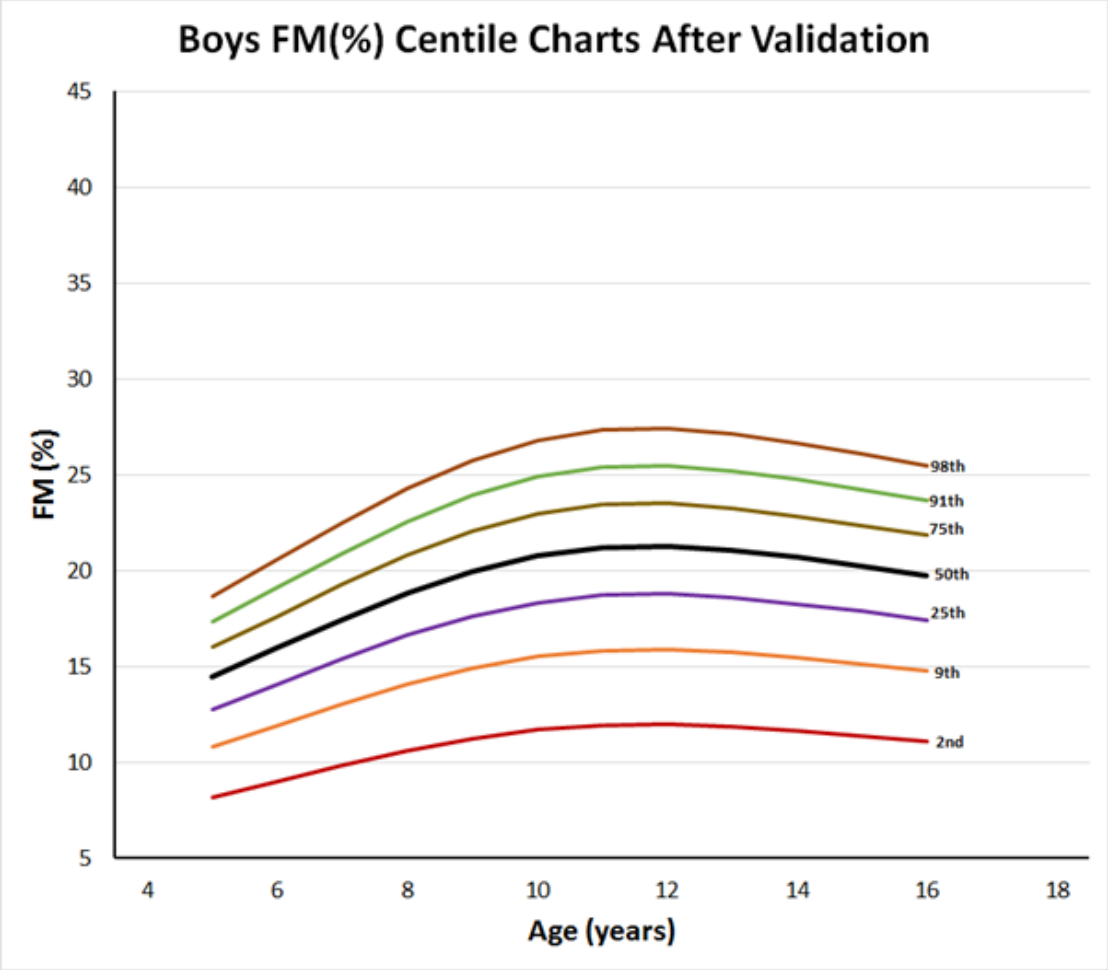

Figure 5(b). %FM centile charts for Boys after the application of validated equation

Table 5(e). Tabulated Girls per cent fat mass (%FM) centile values by exact age before the application of validated equation

Girls	FM (%) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	16.2	17.5	19.0	20.8	23.3	26.7	32.4
6	16.1	17.6	19.2	21.4	24.2	28.2	34.9
7	16.1	17.7	19.6	22.0	25.2	29.8	37.4
8	16.1	17.9	20.0	22.7	26.3	31.4	39.8
9	16.3	18.2	20.5	23.5	27.4	32.9	41.7
10	16.5	18.6	21.1	24.3	28.4	34.1	42.8
11	16.7	19.0	21.6	24.9	29.2	34.7	42.7
12	17.0	19.4	22.1	25.6	29.8	35.0	42.2
13	17.4	20.0	22.9	26.4	30.5	35.4	41.9
14	18.1	20.9	23.8	27.3	31.3	35.9	41.8
15	19.0	21.8	24.8	28.3	32.2	36.6	41.8
16	19.9	22.9	25.9	29.4	33.1	37.2	42.0

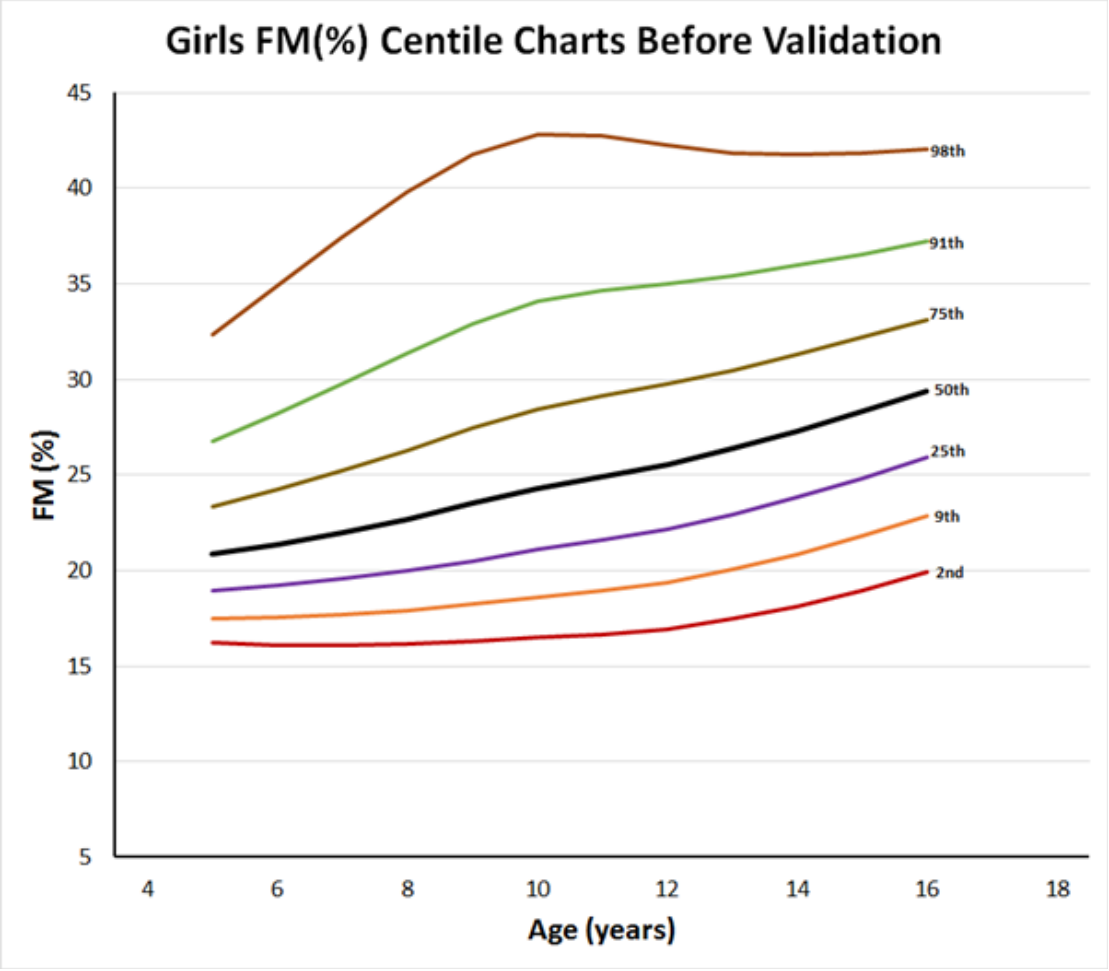

Figure 5(c): %FM centile charts for Girls before the application of validated equation

Table 5(f). Tabulated Girls per cent fat mass (%FM) centile values by exact age after the application of validated equation

Girls	FM (%) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	22.7	24.4	26.5	28.4	30.6	32.5	34.6
6	23.0	24.8	26.9	28.9	31.2	33.1	35.4
7	23.3	25.1	27.3	29.3	31.8	33.7	36.3
8	23.6	25.5	27.7	29.8	32.3	34.3	37.0
9	23.9	25.8	28.1	30.3	32.8	34.9	37.8
10	24.2	26.2	28.5	30.7	33.3	35.6	38.6
11	24.6	26.5	28.9	31.2	33.9	36.2	39.2
12	24.9	27.0	29.2	31.6	34.5	36.8	39.8
13	25.3	27.3	29.5	31.9	34.9	37.1	40.1
14	25.5	27.5	29.8	32.2	35.1	37.4	40.5
15	25.7	27.8	30.0	32.4	35.3	37.7	40.8
16	26.0	28.0	30.2	32.6	35.5	38.0	41.2

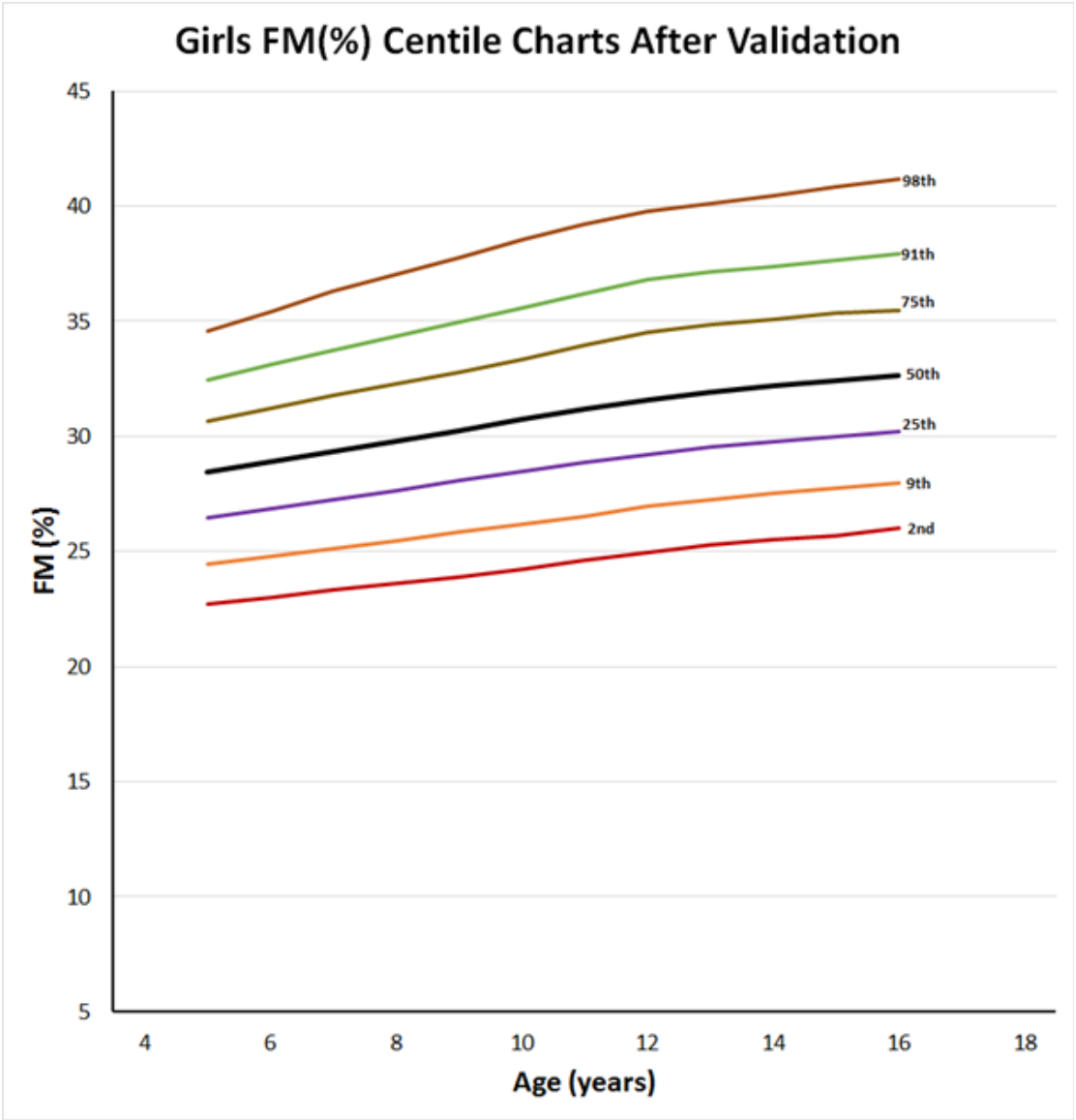

Figure 5(d). %FM centile charts for Girls after the application of validated equation

Figure 5(a) shows a general increase of %FM from age 5 and peaks at approximately age 11 – the likely start of pubertal period for boys. Then, the centiles curves gradually decrease which is clearer for the higher percentiles (75^{th}, 91^{st} and 98^{th} percentiles) reflecting the testosterone drive which causes an increase in FFM post-pubertally. The median percentile shows a gradual decrease after puberty, being relatively flat, varying between 18% and 20% fat mass over the subjects' age range. The lower percentiles (2^{nd}, 9^{th} and 25^{th} percentiles) slightly level off and diverge from the median after age 11.

The %FM percentile chart for boys after the application of the DXA validated equation/correction, figure 5(b), shows a steady increase before age 11 for all percentiles. Then, they show a steady decrease after puberty. The percentiles are observed to be similar in shape when compared to the BIA non-corrected charts for boys. However, the fat mass percentiles before validation are higher for the 91^{st} and 98^{th} percentiles when compared with those after application of the validation. This could also be seen when comparing tables 5(c) and 5 (d).

Figure 5 (c) shows a similar trend of increase in per cent fat mass for all ages before age 10 – the puberty age for girls when compared with the boys before validation, figure 5(a). However, per cent fat mass increases across all the centiles apart from the 98^{th}, which peaked at age 10 and slightly decreases afterwards.

Figure 5(d), shows a consistent rise of all per cent fat mass percentiles until puberty. They then tend to rise steadily with slight variation during the period of adolescence. Comparison of figures 5(a) and 5(c) show that the median (50^{th}) per cent fat mass for boys and girls at age 5 is approximately 18% and 21% respectively and that for age 13

is about 19% and 26% respectively. Thus the median per cent fat mass for girls at ages 5 and 13 are approximately 1.1 and 1.4 respectively times that of boys. Also, the per cent fat mass in boys decreases after puberty whereas the per cent fat mass in girls increases consistently except at the extreme of the 98th centile. The 98th percentiles for boys and girls at ages 5 and 13 are 28% and 32% and 43% and 42% respectively, and the 2nd percentiles for boys and girls at ages 5 and 13 are approximately 13.5% and 16% and 12% and 17% respectively.

Comparison of figures 5(b) and 5(d) indicates that the median per cent fat mass for boys and girls at age 5 is approximately 15% and 28% respectively and that for age 13 is about 21% and 32% respectively. Thus the median per cent fat mass for girls at ages 5 and 13 are approximately 1.86 and 1.52 respectively times that of the boys. This bears a similarity with the BIA non-validated charts which also show lower per cent fat mass in boys after puberty when compared with that of girls. The 98th percentiles for boys and girls at ages 5 and 13 are 19% and 35% and 27% and 40% respectively, and the 2nd percentiles for boys and girls at ages 5 and 13 are approximately 8% and 23% and 12% and 25% respectively.

In addition, the newly-corrected per cent fat mass charts show lower per cent fat masses for boys than the BIA non-corrected charts. Depending on the percentile in question, the magnitude of the per cent fat mass for boys at age 5 varies in the range of approximately 14% and 28% in the case of the BIA non-validated charts and in the range of approximately 8% and 19% in the case of the corrected per cent fat mass charts. At age 13, the magnitude of the per cent fat mass is in the range of approximately12% to 43%

for the BIA non-validated estimates and in the range of approximately12% and 27% for the DXA validated per cent fat mass charts.
However, in the case of the girls, the opposite is observed.

Comparing validated per cent fat mass charts to the BIA non-validated charts, the magnitude of the per cent fat mass for girls at age 5 varies in the range of about 16% to 32% in the case of the BIA non-validated charts and in the range of 23% to 35% in the case of the DXA validated per cent fat mass charts. At age 13, the magnitude of the per cent fat mass is in the range of 17% to 42% for the BIA non-validated charts and in the range of 25% to 40% for the DXA validated per cent fat mass charts.

5.4 Discussion

In this study, per cent fat mass percentile charts have been produced for African and Caribbean children living in the UK. Generally, following a decrease during early infancy, body fat mass increases until puberty, which is consistent with typical growth patterns across childhood and adolescence. At puberty, sex hormones cause a pronounced sexual dimorphism leading to males producing more muscle and lean tissue than fat, and females producing more fat as a natural response to the increase of oestrogen, the main female hormone (Cole et al, 1995; Forbes et al, 1978). Hence, as shown in the curves above, fat mass increased more in girls than the boys in response to their hormonal changes and adaptations.

As with all anthropometric reference curves, it is necessary to use a representative sample of the wider population of interest considering the geographical, socio-economic

and nutritional factors of the sample under consideration. The sample size of 1336 African and Caribbean children in this study is appreciable and representative.

These percentile charts are similar in pattern and variance to per cent fat percentile charts for Caucasian children published in 2006 (McCarthy et al, 2006). Those charts also showed a general rise in per cent fat mass with increasing age with girls showing proportionally higher fat mass values than boys just as it was observed in this study. However, absolute fat mass values of the black girls were higher compared with the values of the Caucasian girls. There is still limited research conducted in this area and so comparisons with similar studies remains difficult. However, Muller et al have published per cent fat curves for US children, albeit using an alternative bio-impedance system (RJL systems). Compared to this study, the sample size was smaller and over a narrower age range (278 boys and 263 girls aged from 8 to 17 years.) In those charts, much higher 85^{th} and 95^{th} values were observed compared with the UK Caucasian charts and the findings in this study. This may reflect a lack of precision in the Muller et al study due to the smaller sample size, whereby extreme values contribute to a greater extent in a smaller sample which may distort the shape and distribution of the curves (Muller et al, 2004). Additionally the sample was comprised of a multi-ethnic population; thus it is difficult to tease out ethnic-specific body fat characteristics.

These per cent fat mass percentile charts should function as a better alternative to body mass index curves which can misclassify children who are large-framed or just muscular, as overweight or obese. Equally, these charts should help avoid misclassifying children as ‘normal’ by the BMI, who in fact have high fat mass. It is important to note that it is excess adiposity or fat mass that drives pathology (metabolic disease) in individuals. Consequently, these fat mass percentile curves should be used to

identify overweight and obese children as an alternative to the BMI charts in paediatric clinics as well as for epidemiological studies. In this respect, the bio-impedance analysis opens a door of opportunity to move beyond BMI, since it is simple, fast to use, portable and cheaper relatively. To reiterate, these are the first set of %FM centile charts that have been produced for the UK African-Caribbean child and youth population. These percentile charts should be used for children of black descent and should give a better indication compared with BMI charts and the Caucasian charts on the specific nature of FM accumulation in this population group.

Study Limitations

The sample size obtained for drawing the percentile charts was appreciable. However, the number of children in the lower age group (specifically age five and six years) was less compared to the numbers for the older children. This could have led to a poorer accuracy at the younger end of the curves. Fortunately, variance in per cent body fat tends to be lower at this end of the age distribution and therefore it is likely the smaller sample size had less of a negative impact.

Chapter 6: Per cent Fat Free Mass (%FFM) centile curves for African-Caribbean Children

6.1 Introduction

For comparative purposes, centile curves for %FFM were also constructed. When these charts are used in conjunction with body fat curves based upon the same population, it can provide a good assessment of body composition against a reference population than BMI charts. Studies have proved that aging leads to increasing fat deposition in the body and also results in loss of muscle tissue/fat free mass which contribute to disability in old age (Mazariegos et al, 1994; Baumgartner et al, 1995; Gallagher et al, 1997). For this reason, low fat free mass may be associated with an increased risk of disability (Visser et al, 1998): hence, the need to track fat free mass levels from childhood to adulthood. This chapter looks at the development of gender specific per cent fat free mass percentile charts for African and Caribbean children to complement those produced for Caucasian children living in the UK.

6.2 Aim

To develop gender specific per cent fat-free mass (%FFM) percentile charts/curves for African and Caribbean children aged 5 to 18 years living in the UK.

6.3 Methods

The sample for this chapter is the same as the previous chapter. Fat free mass (FFM) was calculated as the difference between body weight and fat mass. Seven smoothed centile curves (2^{nd}, 9^{th}, 25^{th}, 50^{th}, 75^{th}, 91^{st} and 98^{th}) of per cent FFM (% FFM) for specific age and gender were derived using the LMS Chartmaker for both the non-

validated and validated data. The procedure was identical to that described in chapter five.

6.4 Results

The FFM (kg) and %FFM values are summarised in tables 5(a) and 5(b) above. Tables 6(a) to 6(d) and figures 6(a) to 6(d) illustrate the tabulated per cent fat free mass (%FFM) centile values by exact age with their corresponding per cent fat free mass (%FFM) centile charts for boys and girls before and after the application of the validated equation to the BIA data set. In each chart, the curves represent the 2^{nd}, 9^{th}, 25^{th}, 50^{th}, 75^{th}, 91^{st} and 98^{th} centiles. In general, the per cent fat free mass (%FFM) curves reflect the reciprocal of per cent fat mass curves.

In figure 6(a), the per cent fat free mass (apart from the 98^{th} percentile) for boys of all ages generally decreases throughout from age 5 and until approximately age 11 – the puberty age where the charts then gradually increase during mid-adolescence. The median remains relatively flat, varying between approximately 80% and 82% fat free mass over the various age ranges. The median reaches its lowest point at age 11. The lower percentiles slightly increase and converge towards the median after age 11.

Table 6(a). Tabulated Boys per cent fat free mass (%FFM) centile values by exact age before the application of validated equation

Boys	FFM (%) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	72.0	76.4	79.2	81.4	83.3	84.8	86.3
6	70.3	75.2	78.7	81.4	83.5	85.3	87.0
7	68.1	73.9	78.0	81.1	83.5	85.5	87.4
8	66.0	72.5	77.2	80.6	83.3	85.5	87.5
9	64.1	71.3	76.5	80.2	83.0	85.4	87.5
10	62.2	70.4	76.0	79.9	82.9	85.3	87.5
11	61.1	70.0	75.9	79.9	82.9	85.4	87.6
12	61.2	70.2	76.2	80.3	83.3	85.8	88.0
13	62.2	70.8	77.0	81.0	84.0	86.5	88.6
14	63.2	71.4	77.6	81.6	84.6	86.9	89.0
15	64.7	71.9	78.0	81.9	84.8	87.1	89.2
16	66.2	72.4	78.3	82.1	84.9	87.2	89.2

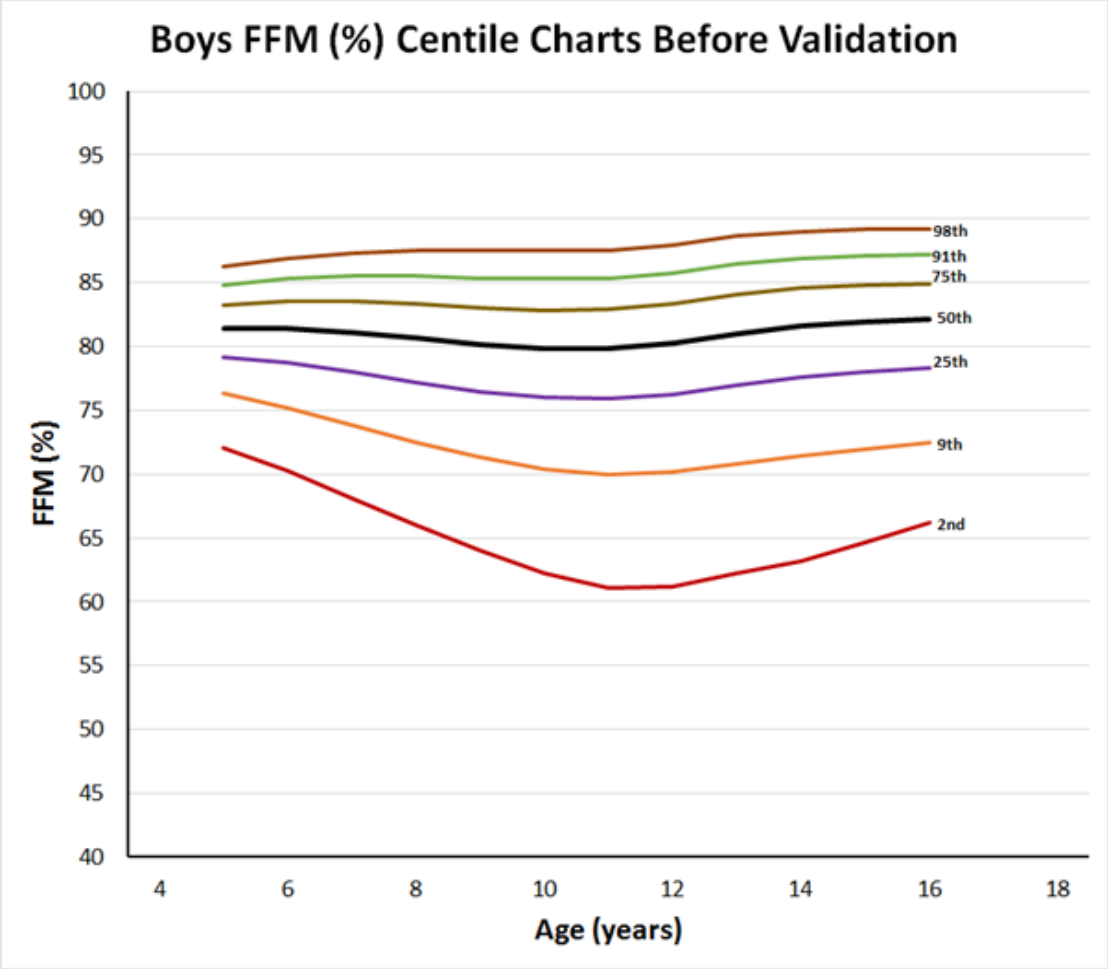

Figure 6(a) %FFM centile charts for Boys before the application of validated equation

Table 6(b). Tabulated Boys per cent fat free mass (%FFM) centile values by exact age after the application of validated equation

Boys	FFM (%) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	69.1	72.1	75.1	77.4	80.8	84.4	87.6
6	68.5	71.4	74.4	76.5	79.9	83.4	86.4
7	67.8	70.7	73.6	75.7	79.4	82.8	85.7
8	67.3	70.1	73.0	75.0	79.0	82.4	85.1
9	66.8	69.6	72.4	74.4	78.6	82.4	85.1
10	66.5	69.3	72.1	74.0	78.2	82.4	85.1
11	66.4	69.2	71.9	73.9	78.4	83.1	85.8
12	66.4	69.2	71.9	73.9	78.6	83.5	86.2
13	66.5	69.3	72.1	74.0	78.8	83.9	86.6
14	66.7	69.5	72.3	74.2	79.3	84.4	87.1
15	66.9	69.7	72.5	74.5	79.7	84.8	87.5
16	67.2	70.0	72.8	74.8	80.2	85.3	88.0

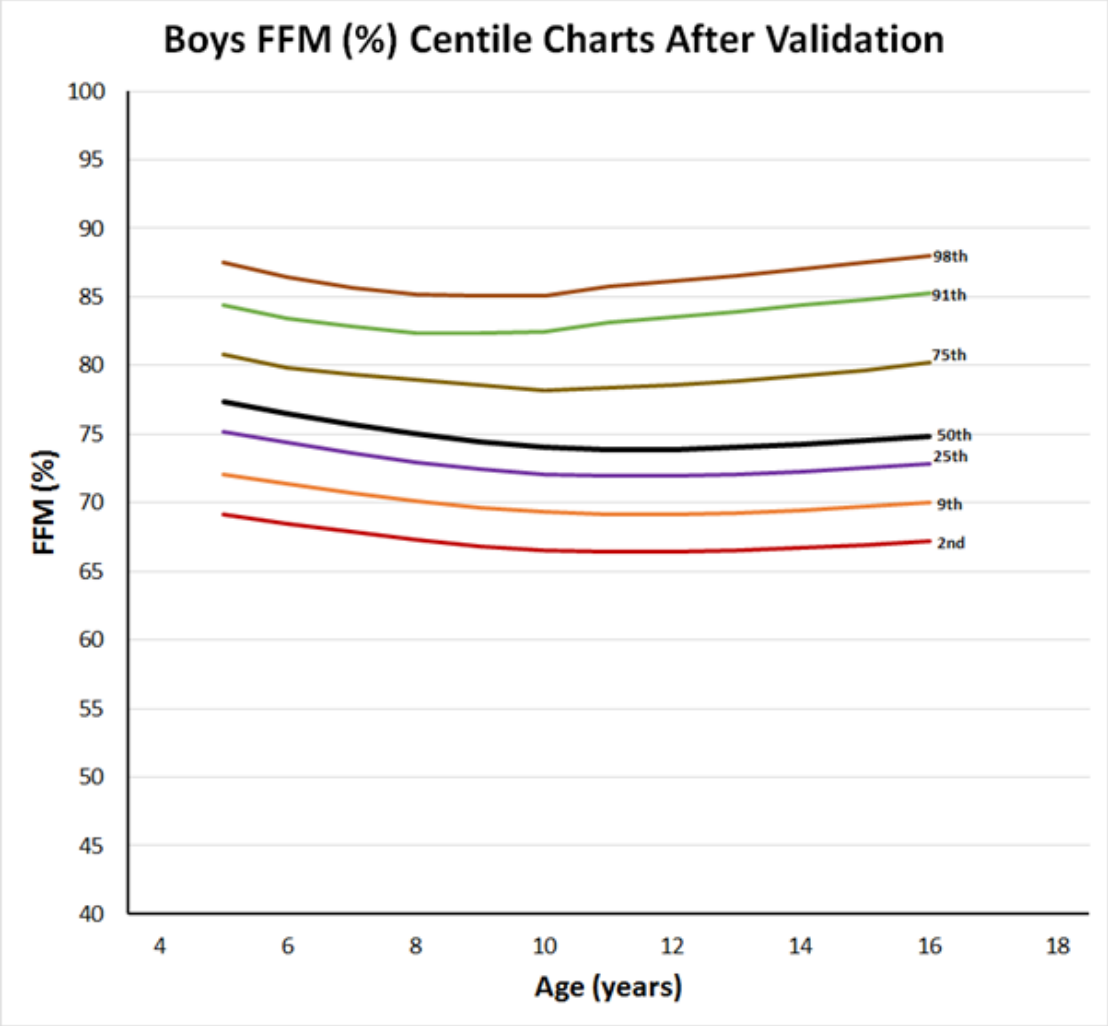

Figure 6(b) %FFM centile charts for Boys after the application of validated equation

Table 6(c). Tabulated Girls per cent fat free mass (%FFM) centile values by exact age before the application of validated equation

Girls	FFM (%) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	68.1	73.4	76.6	78.9	80.9	82.4	83.9
6	65.2	72.0	75.7	78.5	80.7	82.5	84.1
7	62.3	70.6	74.8	77.9	80.4	82.4	84.2
8	59.8	69.1	73.8	77.3	80.0	82.2	84.2
9	58.1	67.7	72.8	76.5	79.4	81.8	84.0
10	57.2	66.6	71.8	75.7	78.8	81.4	83.8
11	57.2	65.8	71.0	75.0	78.3	81.1	83.7
12	57.4	65.1	70.2	74.3	77.8	80.7	83.5
13	57.5	64.4	69.3	73.5	77.0	80.1	83.1
14	57.5	63.5	68.3	72.4	76.1	79.4	82.6
15	57.2	62.7	67.1	71.3	75.1	78.5	82.0
16	56.8	61.7	66.0	70.1	74.0	77.6	81.4

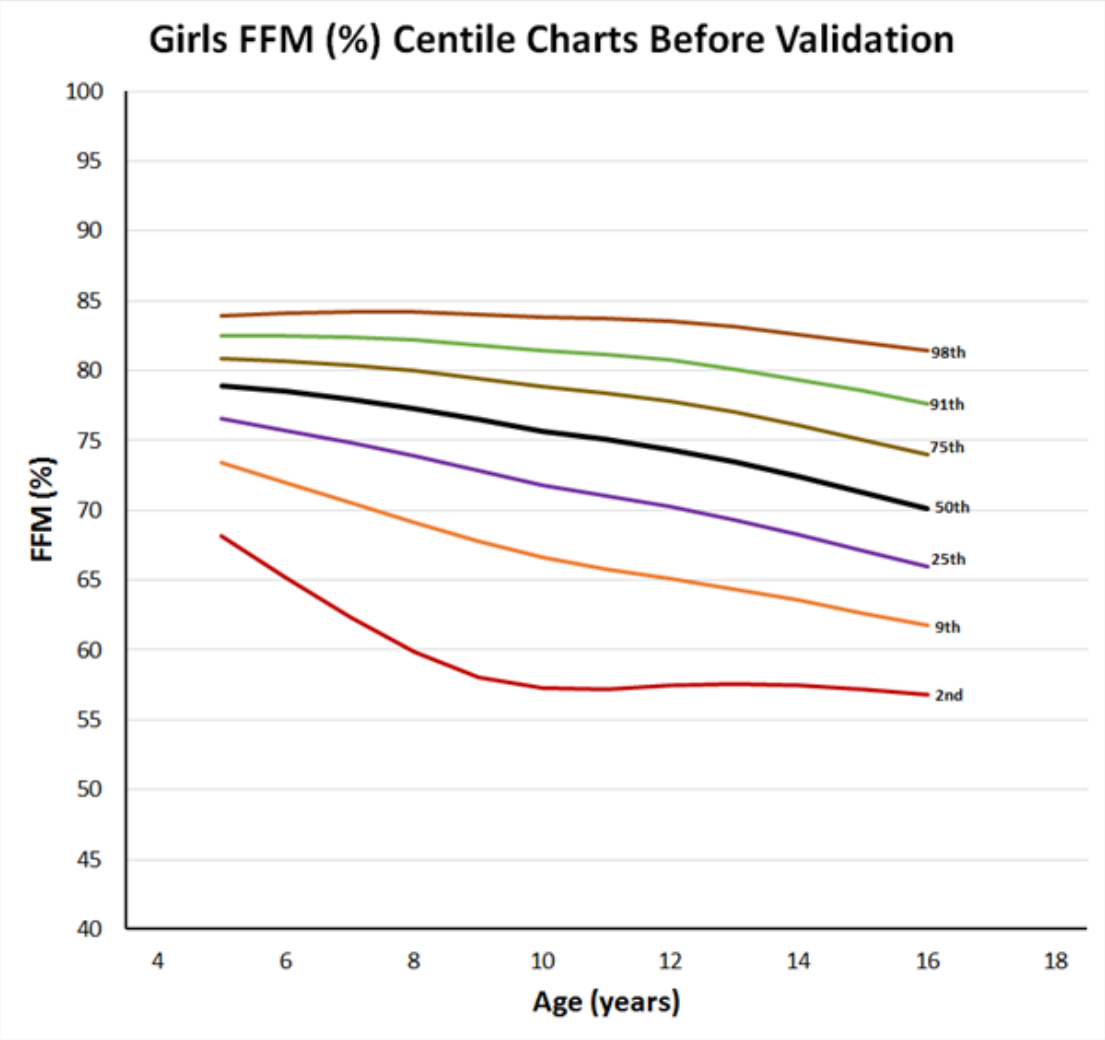

Figure 6(c) %FFM centile charts for Girls before the application of validated equation

Table 6(d). Tabulated Girls per cent fat free mass (%FFM) centile values by exact age after the application of validated equation

Girls	FFM (%) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	64.6	67.9	70.3	71.5	73.8	77.1	79.3
6	63.9	67.4	69.8	71.1	73.4	76.7	79.0
7	63.2	66.8	69.2	70.7	73.0	76.4	78.7
8	62.5	66.2	68.7	70.2	72.6	76.0	78.4
9	61.8	65.6	68.2	69.8	72.2	75.7	78.1
10	61.0	65.0	67.7	69.3	71.8	75.3	77.8
11	60.4	64.4	67.2	68.8	71.4	74.9	77.4
12	60.0	63.9	66.7	68.4	71.0	74.6	77.1
13	59.6	63.4	66.3	68.1	70.7	74.3	76.9
14	59.3	63.1	66.0	67.8	70.5	74.2	76.8
15	59.1	62.8	65.7	67.6	70.3	74.0	76.7
16	58.9	62.5	65.5	67.3	70.2	73.9	76.6

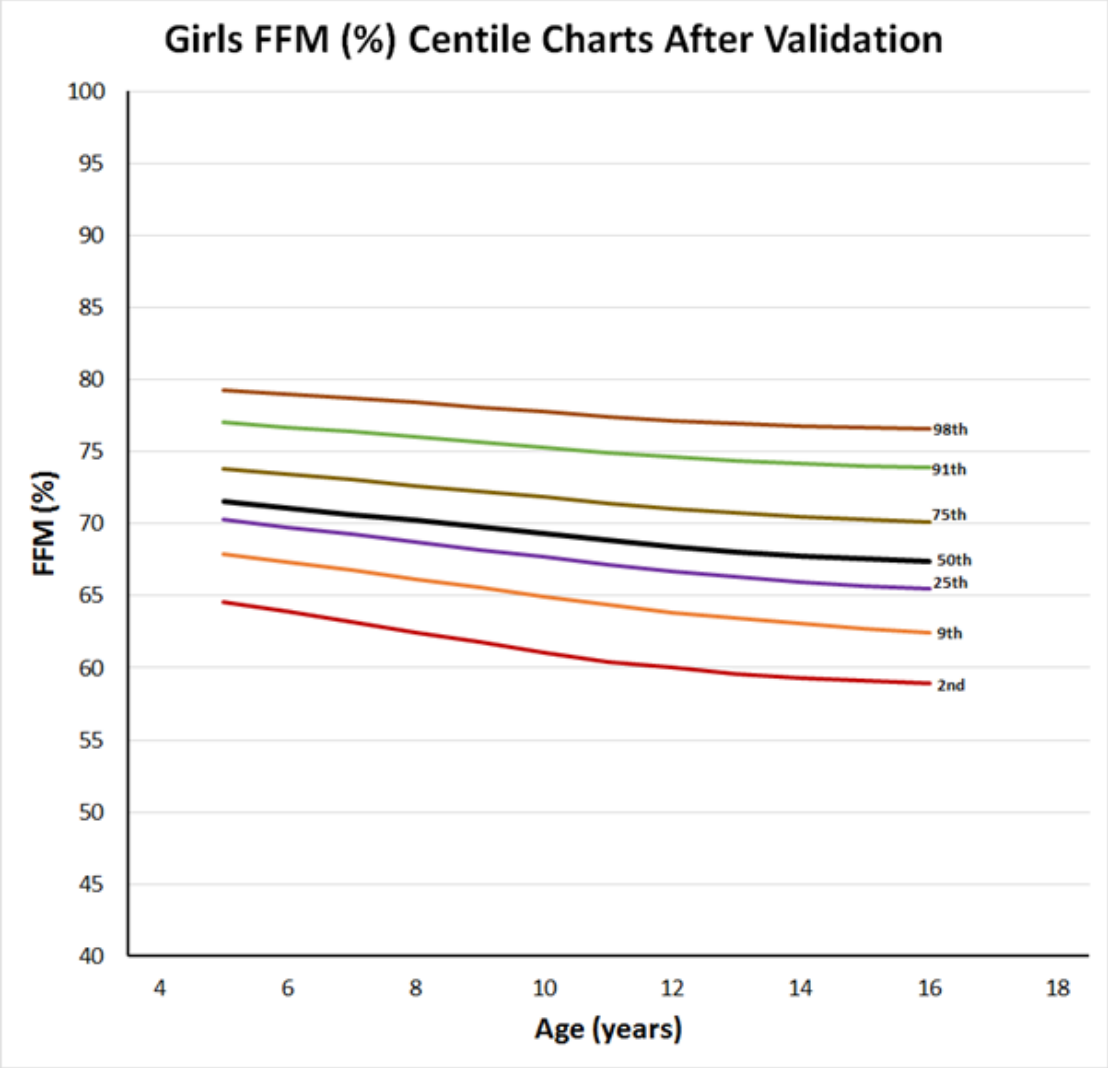

Figure 6(d) %FFM centile charts for Girls after the application of validated equation

Considering figure 6(b), the percentile charts for boys after the application of the validated equation, a decrease in FFM of all percentiles is seen until approximately age 11 and thereafter a gradual increase during mid-adolescence is observed. This pattern is similar to the BIA non-corrected charts for boys.

Figure 6(c) shows a similar trend of gradual decline of per cent fat free mass for all ages of the girls. The median percentile curve for the girls equates to lower per cent fat free mass values ranging between approximately 70% and 79% when compared with that of boys which ranges between 80% and 82%. For girls, the 50th centile equates to a lower %FFM compared with boys and declines with age until puberty, continuing to decrease post-pubertal at a slower rate up to age 13 years. At this age, the median %FFM equates to approximately 74% of body weight, compared with 81% for boys.

Figure 6(d), shows a consistent decrease in all per cent fat mass percentiles from age five until puberty. The curves then tend to show a slight divergence from the median across adolescence. Again the curve pattern is similar to the non-corrected charts for the girls.

A comparison of figures 6(b) and 6(d) indicates that the median per cent fat-free mass for boys and girls at age 13 is about 74% and 68% respectively, confirming the opposite trend when compared with the fat mass curves where there is a decrease in per cent fat mass in boys after puberty when compared with that of the girls.

6.5 Discussion

This study has produced gender specific per cent fat free mass (%FFM) percentile charts for African and Caribbean children living in the UK as seen in the results above. Equivalent charts for Caucasian children have recently been developed (McCarthy et al, 2014). The charts illustrate the changes and variations in %FFM across childhood to

adolescence in the African and Caribbean children. While %FFM decreases with age in girls, %FM increases with age especially around puberty. The fact is that puberty is a critical period for body composition changes and for girls; age at menarche is of great influence on the development of the fat mass. Consequently, girls who reach menarche at an early age tend to have accelerated fat deposition which is due to reproductive maturation associated with oestrogen (the hormone that dominates female development) production (Vink et al, 2010). These results confirm findings from other studies conducted on body fat mass in adolescent girls with DXA scan like Goulding and Rico who found increased fatness in girls during puberty (Goulding et al, 1996; Rico et al, 1993). Furthermore, Laurson et al have also produced growth charts for US children and adolescence with a similar observation (Laurson et al, 2011). Hence primary prevention of obesity should start with encouraging a healthy growth pattern in young girls before the initiation of pubertal maturation and these needs to be emphasised in black girls to reduce their risk of becoming obese at puberty. This is because compared with Caucasian girls, black girls have lower %FFM and higher %FM at same age levels. In boys %FFM is relatively stable during pre-puberty but starts increasing from puberty. A study conducted to evaluate the effect of testosterone on body composition in boys, found that testosterone, the principal male sex hormone markedly stimulated an increase in fat free mass in adolescent boys (Gregory et al, 1992). This confirms the findings of this study as puberty coincides with adolescence, and during this period high testosterone levels are produced, which are responsible for all changes in boys' development such as increasing fat-free-mass. Per cent FFM charts may find utility in a clinical setting where they can be used to identify children who have low FFM for age and gender. Whether they would add anything to the SMMa charts is unclear at this

stage as SMM forms the major fraction of FFM. However they may become useful in situations where measurement of SMMa is not possible due to equipment limitations. According to a study done using representative data from the Third National Health and Nutrition Examination Survey (NHANES III), %FFM decreased with age and this was more pronounced in black men and women although higher FFM and lean mass to height ratio have been reported in blacks than in whites (Thomas et al, 2005). The higher FFM in blacks than whites was observed in young samples (Thomas et al, 2005). This calls for the need to track and monitor FFM levels in blacks from childhood to adulthood to identify the cause of decline and put in place interventional measures at the earliest time possible.

In conclusion, these charts would be better monitoring tools of metabolic health (and could be used for the assessment and prevention of the metabolic syndrome) than BMI charts in African and Caribbean children, both in the clinical and epidemiological settings.

Study Limitation/Strength

The limitations of this study are similar to those outlined in previous and forthcoming chapters. As regards the sample size, which was obtained for drawing the percentile charts, although appreciable, the number of children in the lower age group (example five and six years) was less compared to the numbers of older children, and data were available beyond age 16 years. However, the large sample size used in the study makes it representative and the findings reliable enough to apply in practice.

Chapter 7: Skeletal Muscle Mass (SMM) centile curves for African-Caribbean Children

7.1 Introduction

The risk for sarcopenia and sarcopenic obesity has been found to begin in early life, which, if not addressed, can lead to adverse metabolic health problems later in life, in addition to its effect on mobility, frailty and quality of life in older age (Sayer et al, 2008). This is because low muscle fitness is associated with metabolic health risk and muscular strength is directly related to insulin sensitivity in children and adolescents (McCarthy et al, 2011). Skeletal muscle is normally responsible for more than 75% of all insulin-mediated glucose metabolism and plays an important role in the whole-body glucose balance for normal body functioning (Steene et al, 2009; Benson et al, 2006). For this reason, measurement of skeletal muscle mass in children and adults is essential for assessment of metabolic health. However, the use of SMM measurement for surveillance and clinical assessment has been a challenge in the absence of longitudinal data which identifies individuals of various ages with high or low amounts of SMM (Pietrobelli & Peroni, 2003). Data on SMM and the tools for monitoring its levels are required both for clinical management of individuals as well as for longitudinal and cross-sectional surveillance of populations. Hence the need for the development of skeletal muscle monitoring tools from childhood.

7.2 Aim

To develop various skeletal muscle mass curves and muscle-to-fat-ratios for African - Caribbean children and youth population.

7.3 Methods

7.3.1 Subjects and Anthropometry

As in the previous studies on %FM and %FFM, DXA was used to validate the BIA skeletal muscle mass (SMM) output from BIA (Tanita BC-148MA system). Anthropometric measurements and body composition data of SMM measured by the use of DXA and BIA on a sample of 44 African and Caribbean children ranging from age 5 to 16 years were collected as shown in section 4.2. Fifty five per cent were boys and forty five per cent were girls. To derive appendicular skeletal muscle mass (SMMa) data, skeletal muscle mass from the four limbs were extracted and added together for each subject: the rationale being that SMMa accounts for more than 75% of whole body SMM in adults and is the major fraction of whole body SMM involved in ambulation and physical activities, which can be built up or lost (Synder et al, 1975).

The predictive validated equations of appendicular skeletal muscle mass (SMMa) in relation to height and impedance were applied to the existing extracted data set and the results used to draw the various skeletal muscle mass centile curves for boys and girls.

7.3.2 Statistical Analysis

Seven smoothed (2^{nd}, 9^{th}, 25^{th}, 50^{th}, 75^{th}, 91^{st} and 98^{th}) centile curves of absolute SMMa in kilograms (SMMa (kg), per cent SMMa (%SMMa), and per cent SMMa/FFM (SMMa/FFM x 100%) for specific age and gender were derived using the LMS Chartmaker software as previously discussed. Thus the curves were produced to reflect SMMa in three formats, namely, absolute SMMa (kg), SMMa as a percentage of total body mass (%SMM) and SMM as a percentage of FFM (SMMa/FFM (kg) x 100). Muscle to fat ratio, MFR, was also derived by dividing SMMa (kg) by FM (kg) and expressed as histograms for boys and girls separately for comparison (Appendix F).

7.4 Results

Tables 5(a) and 5(b) above show the summarised descriptive statistics of the appendicular skeletal muscle mass (SMMa) at various age groups for the boys and girls before and after the application of the new predicted equation to the BIA data set. Tables 7(a) to 7(d) and figures 7(a) to 7(d) illustrate the tabulated appendicular skeletal muscle mass (SMMa) centile values by exact age with their corresponding centile charts for boys and girls before and after the application of the new predicted equation to the BIA data set. In each chart, the curves represent the 2^{nd}, 9^{th}, 25^{th}, 50^{th}, 75^{th}, 91^{st} and 98^{th} centiles.

A comparison of figures 7(a) and 7(b) for boys and figures 7(c) and 7(d) for girls show similarity between the segmental BIA charts before validation and after the application of the corrected equations.

Tables 7(e) to 7(h) and figures 7(e) to 7(h) show the tabulated per cent appendicular skeletal muscle mass (%SMMa) centile values by exact age with their corresponding centile charts for boys and girls before and after the application of the new predicted equation to the BIA data set. In each chart, the curves represent the 2^{nd} to 98^{th} percentiles.

Table 7(a). Tabulated Boys appendicular skeletal muscle mass (SMMa) centile values by exact age before the application of validated equation.

Boys	SMMa (kg) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	2.7	3.3	4.0	4.7	5.7	6.8	8.2
6	3.3	4.0	4.8	5.7	6.8	8.1	9.7
7	4.0	4.8	5.7	6.8	8.0	9.5	11.4
8	4.9	5.9	6.9	8.1	9.5	11.2	13.3
9	6.0	7.1	8.2	9.6	11.2	13.1	15.4
10	7.1	8.3	9.6	11.2	13.0	15.1	17.8
11	8.1	9.5	11.0	12.8	14.9	17.3	20.4
12	9.2	10.7	12.4	14.5	16.8	19.6	23.1
13	10.4	12.1	14.0	16.2	18.9	21.9	25.8
14	11.7	13.6	15.6	17.9	20.7	23.9	28.0
15	13.1	15.0	17.1	19.5	22.4	25.6	29.6
16	14.7	16.6	18.7	21.1	23.9	27.1	31.1

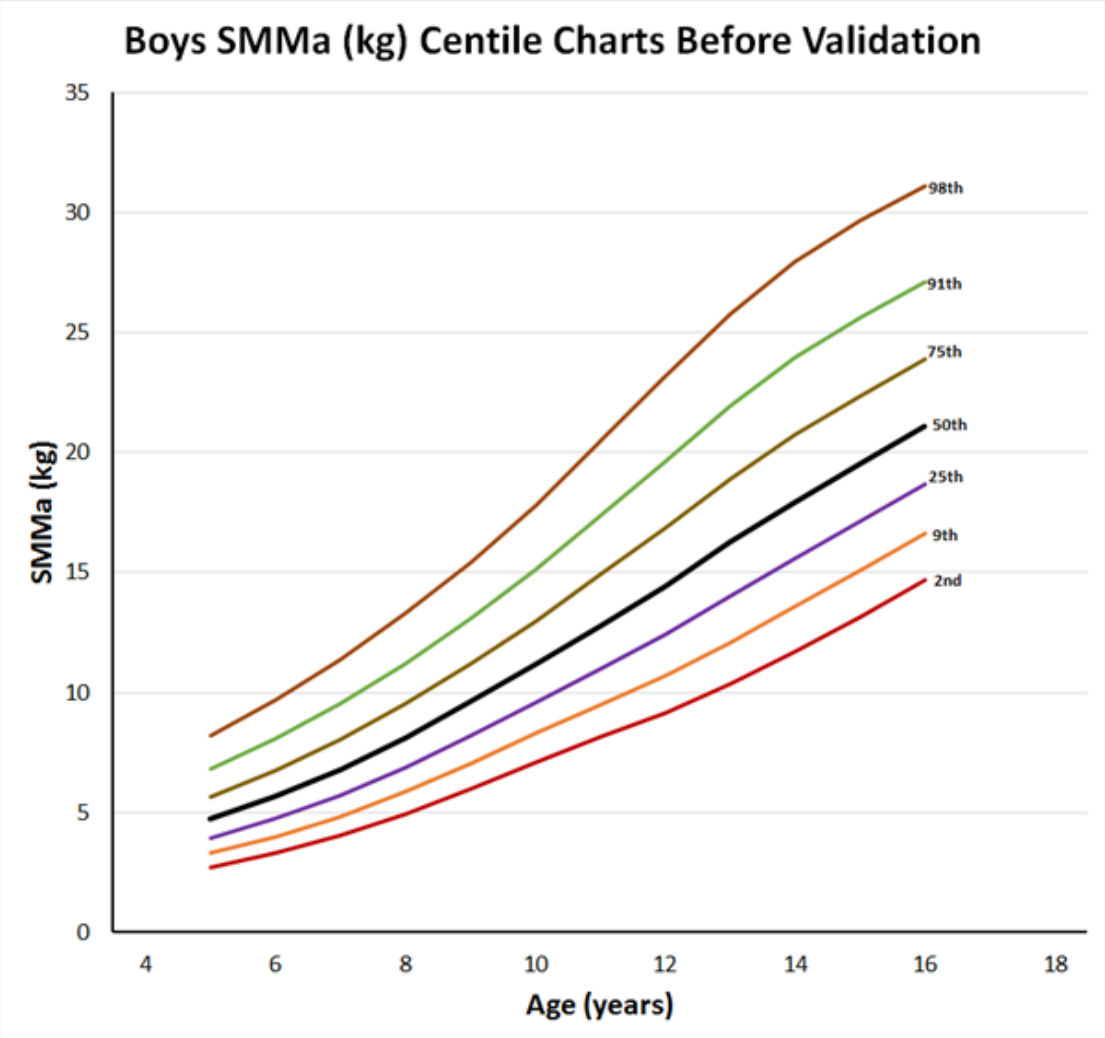

Figure 7(a) SMMa centile charts for Boys before the application of validated equation

Table 7(b). Tabulated Boys appendicular skeletal muscle mass (SMMa) centile values by exact age after the application of validated equation

Boys	SMMa (kg) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	2.7	3.3	3.9	4.7	5.6	6.6	7.7
6	3.2	3.9	4.7	5.6	6.6	7.7	9.0
7	3.9	4.7	5.6	6.6	7.7	9.0	10.4
8	4.6	5.6	6.6	7.7	9.0	10.4	12.0
9	5.5	6.6	7.7	8.9	10.3	11.8	13.6
10	6.3	7.5	8.7	10.1	11.7	13.5	15.5
11	7.1	8.4	9.8	11.4	13.3	15.3	17.8
12	7.8	9.3	11.0	12.9	15.0	17.4	20.2
13	8.7	10.5	12.4	14.5	16.9	19.5	22.6
14	9.7	11.7	13.8	16.1	18.6	21.4	24.7
15	10.8	13.0	15.2	17.6	20.2	23.0	26.3
16	12.1	14.4	16.6	19.1	21.8	24.5	27.7

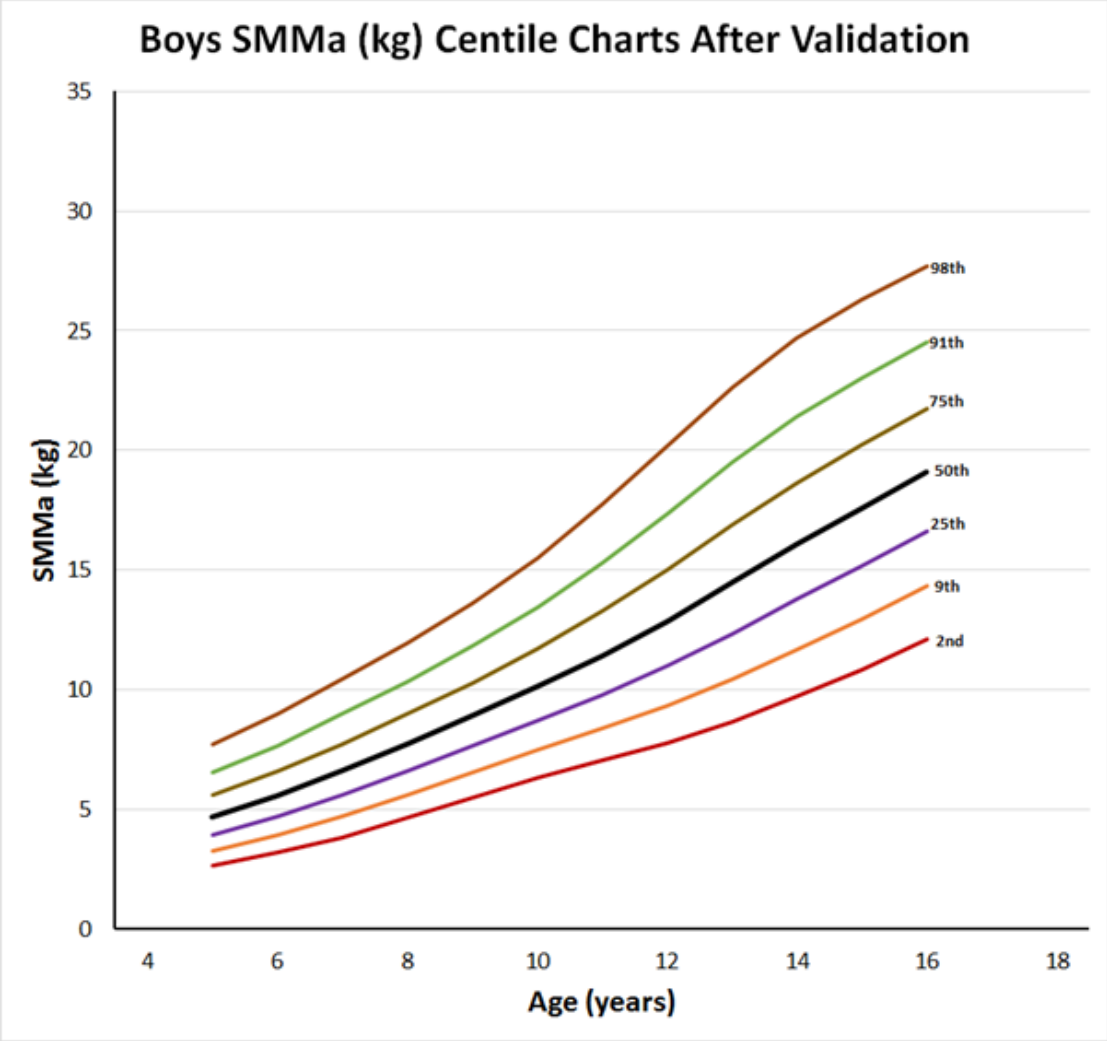

Figure 7(b) SMMa centile charts for Boys after the application of validated equation

Table 7(c). Tabulated Girls' appendicular skeletal muscle mass (SMMa) centile values by exact age before the application of validated equation

Girls	SMMa (kg) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	3.2	3.6	4.1	4.8	5.6	6.5	7.9
6	3.8	4.3	5.0	5.7	6.7	7.8	9.4
7	4.5	5.1	5.9	6.8	7.9	9.3	11.0
8	5.2	6.0	6.9	8.0	9.3	10.8	12.8
9	6.0	7.0	8.0	9.3	10.8	12.5	14.7
10	6.9	8.0	9.2	10.7	12.4	14.3	16.7
11	7.8	9.1	10.4	12.0	13.8	15.9	18.4
12	8.7	10.0	11.5	13.2	15.1	17.2	19.7
13	9.5	10.9	12.5	14.2	16.1	18.1	20.6
14	10.1	11.6	13.2	14.8	16.7	18.7	21.0
15	10.7	12.2	13.7	15.3	17.1	18.9	21.1
16	11.3	12.8	14.2	15.8	17.4	19.1	21.1

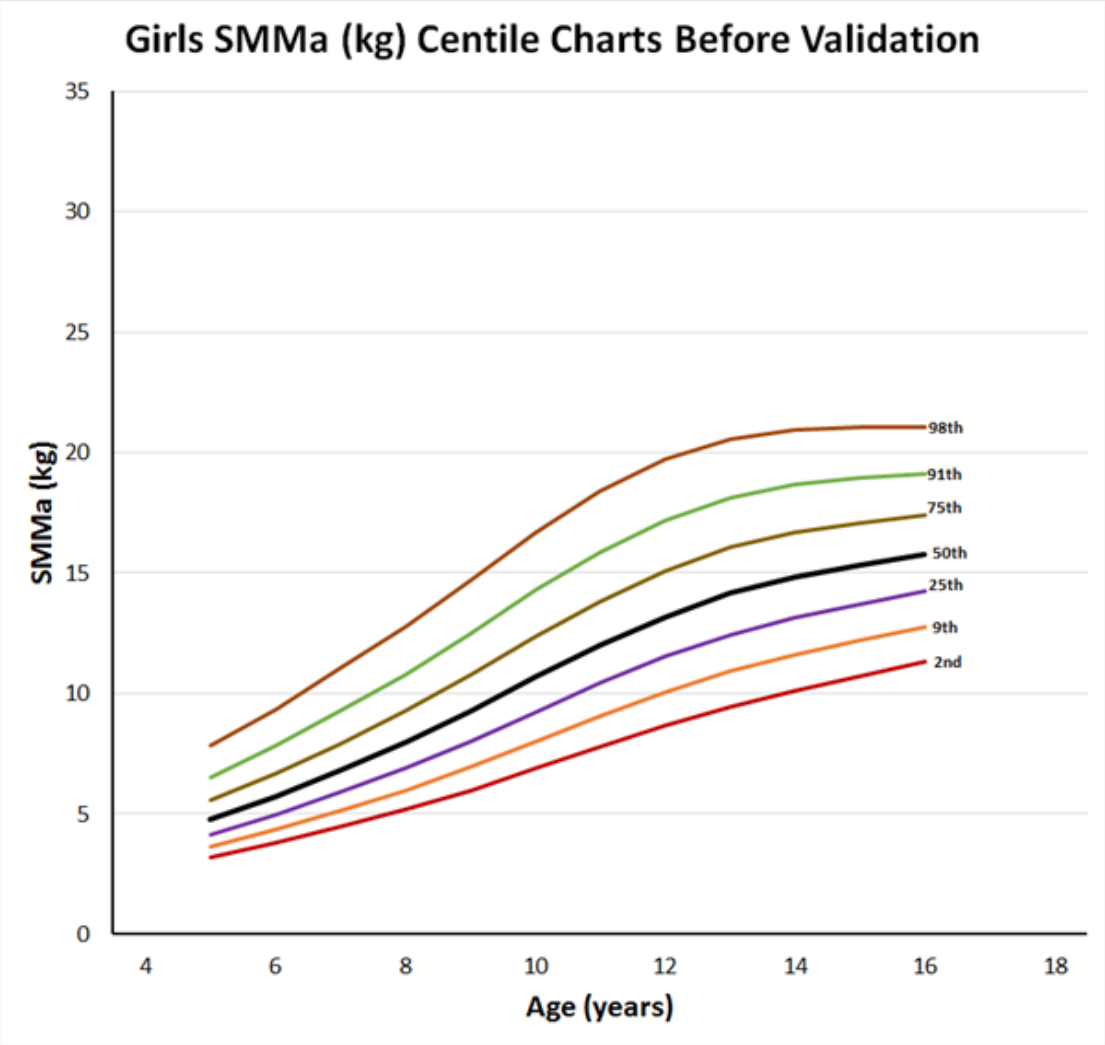

Figure 7(c) SMMa centile charts for Girls before the application of validated equation

Table 7(d). Tabulated Girls' appendicular skeletal muscle mass (SMMa) centile values by exact age after the application of validated equation

Girls	SMMa (kg) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	2.9	3.3	3.9	4.5	5.1	5.9	6.8
6	3.5	4.1	4.7	5.4	6.2	7.1	8.2
7	4.1	4.8	5.5	6.4	7.3	8.4	9.7
8	4.8	5.6	6.4	7.4	8.4	9.7	11.1
9	5.5	6.4	7.3	8.4	9.6	11.0	12.7
10	6.2	7.2	8.3	9.5	10.9	12.4	14.3
11	7.0	8.1	9.2	10.6	12.1	13.8	15.8
12	7.7	8.9	10.1	11.5	13.1	14.9	17.1
13	8.2	9.4	10.7	12.2	13.8	15.6	17.9
14	8.6	9.8	11.0	12.5	14.1	15.9	18.1
15	8.8	9.9	11.1	12.5	14.1	15.9	18.0
16	8.8	10.0	11.2	12.5	14.0	15.7	17.7

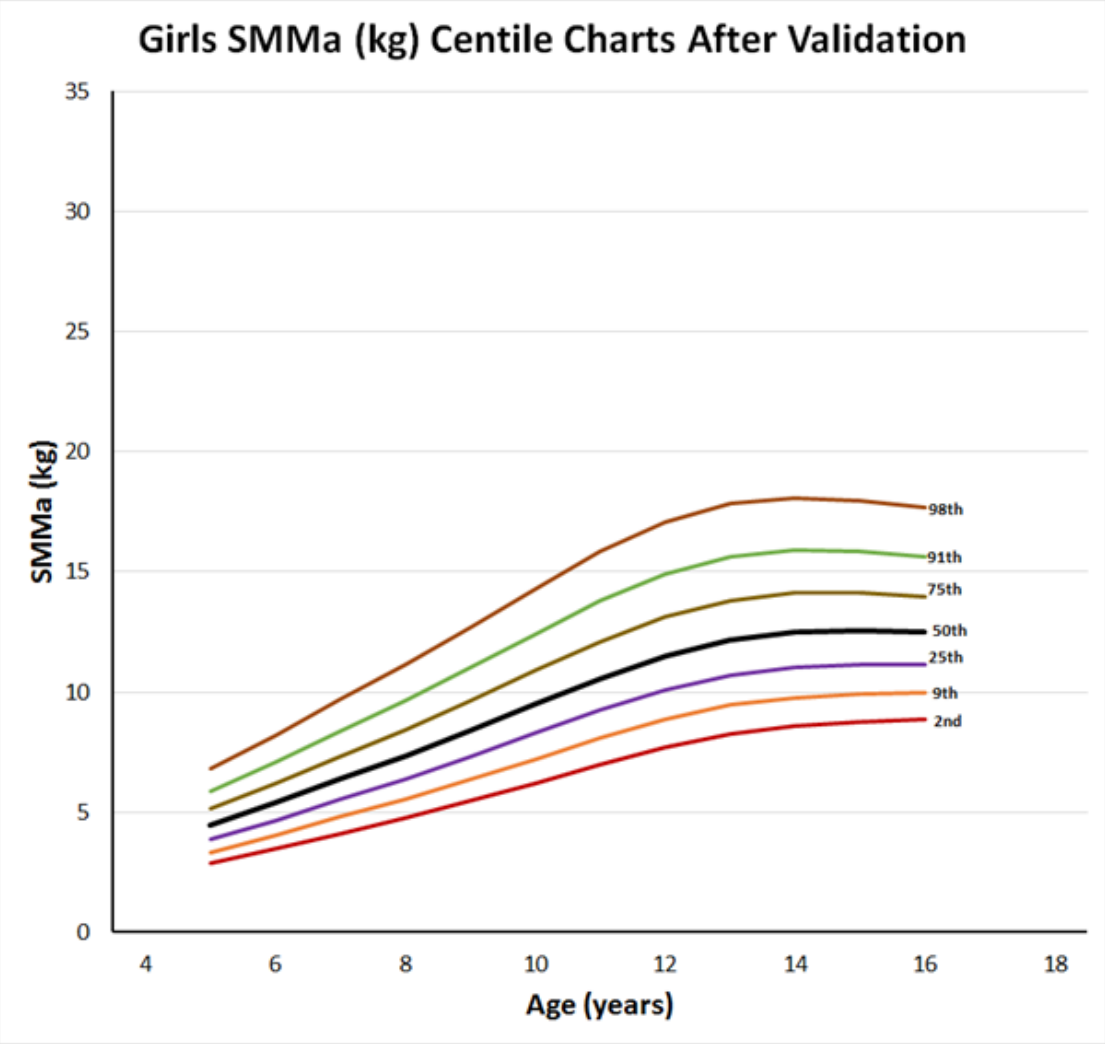

Figure 7(d) SMMa centile charts for Girls after the application of validated equation

Table 7(e). Tabulated Boys' per cent appendicular skeletal muscle mass (%SMMa) centile values by exact age before the application of validated equation

Boys	SMMa (%) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	18.5	20.5	22.3	24.2	26.1	28.1	30.2
6	19.9	21.9	23.7	25.6	27.5	29.4	31.4
7	21.4	23.3	25.1	27.0	28.8	30.6	32.5
8	22.7	24.6	26.4	28.2	30.0	31.7	33.6
9	23.8	25.7	27.5	29.2	31.0	32.7	34.5
10	24.6	26.5	28.3	30.1	31.9	33.6	35.4
11	25.3	27.3	29.1	31.0	32.8	34.5	36.4
12	25.9	28.0	30.0	31.9	33.8	35.6	37.6
13	26.6	28.8	30.8	32.8	34.8	36.7	38.7
14	27.2	29.4	31.4	33.4	35.3	37.2	39.2
15	27.6	29.8	31.7	33.7	35.6	37.4	39.3
16	28.0	30.1	32.0	33.8	35.6	37.4	39.2

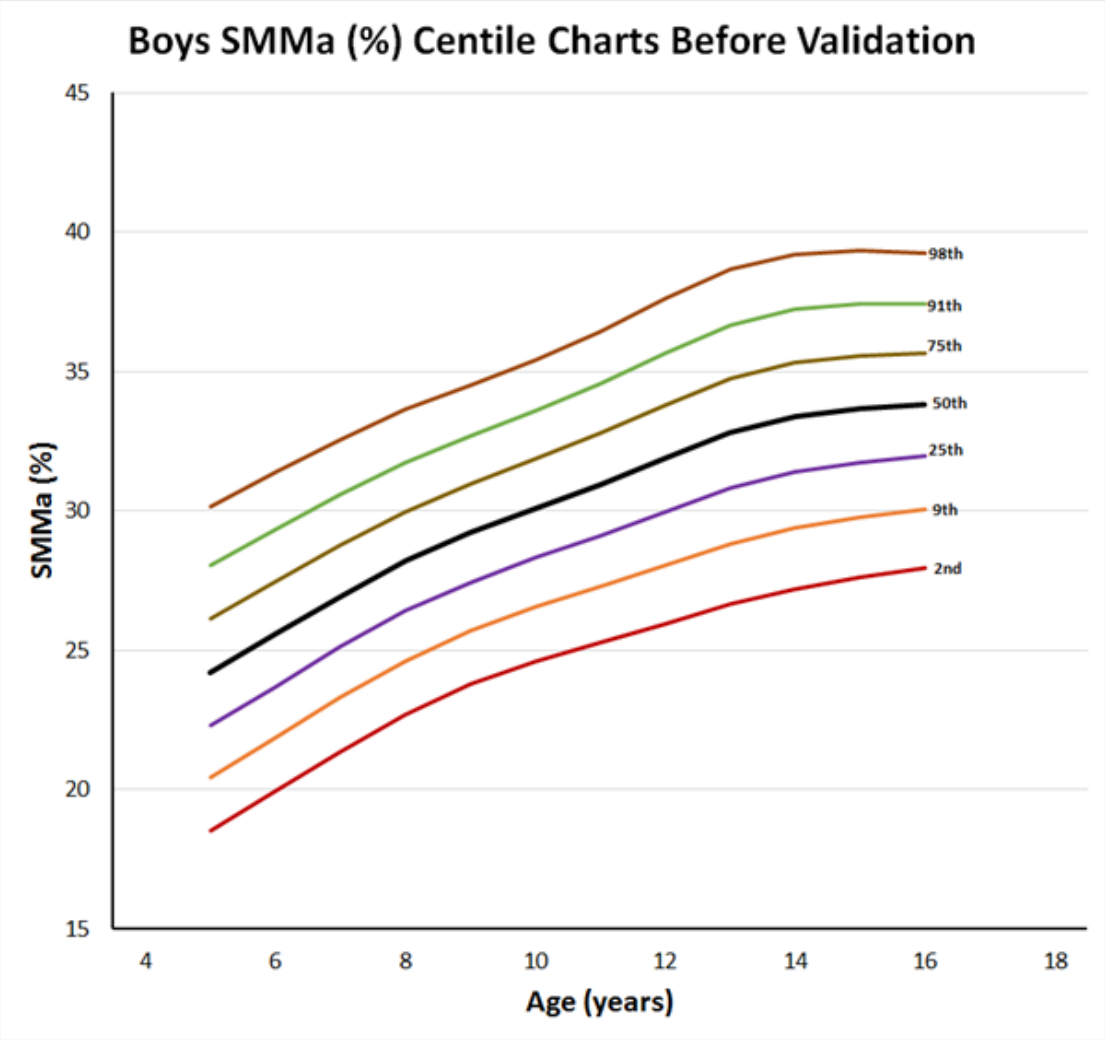

Figure 7(e) %SMMa centile charts for Boys before the application of validated equation

Table 7(f). Tabulated Boys' per cent appendicular skeletal muscle mass (%SMMa) centile values by exact age after the application of validated equation

Boys	SMMa (%) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	24.4	25.6	26.3	27.1	28.0	28.7	30.0
6	24.6	25.8	26.6	27.4	28.4	29.0	30.5
7	24.8	26.1	26.9	27.7	28.7	29.4	30.9
8	25.0	26.4	27.2	28.0	29.1	29.9	31.5
9	25.3	26.7	27.5	28.4	29.6	30.4	32.0
10	25.6	27.0	27.9	28.8	30.1	30.9	32.7
11	25.8	27.3	28.2	29.3	30.5	31.5	33.3
12	26.1	27.6	28.6	29.7	31.1	32.1	34.0
13	26.4	28.0	29.1	30.2	31.7	32.8	34.8
14	26.7	28.3	29.5	30.8	32.3	33.5	35.5
15	26.9	28.7	30.0	31.3	32.9	34.1	36.1
16	27.1	29.0	30.4	31.8	33.4	34.7	36.7

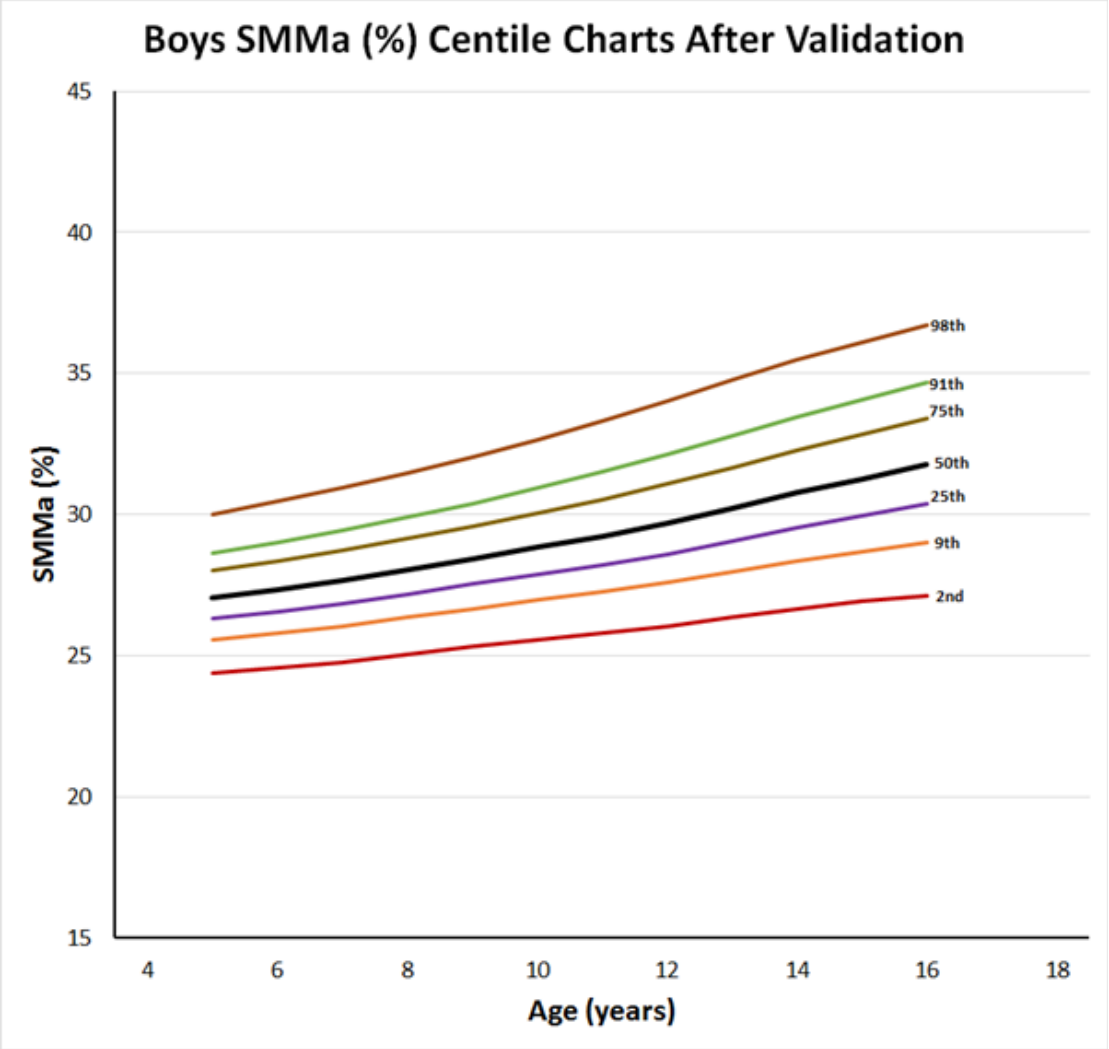

Figure 7(f) %SMMa centile charts for Boys after the application of validated equation

Table 7(g). Tabulated Girls' per cent appendicular skeletal muscle mass (%SMMa) centile values by exact age before the application of validated equation

Girls	SMMa (%) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	21.8	23.1	24.2	25.4	26.7	27.9	29.2
6	22.0	23.4	24.6	25.9	27.2	28.4	29.8
7	22.2	23.6	25.0	26.3	27.6	29.0	30.4
8	22.3	23.9	25.3	26.7	28.1	29.4	30.9
9	22.4	24.0	25.5	27.0	28.4	29.8	31.3
10	22.4	24.2	25.7	27.2	28.7	30.1	31.6
11	22.5	24.3	25.9	27.4	28.9	30.4	31.9
12	22.8	24.4	26.0	27.5	29.0	30.5	32.0
13	23.0	24.6	26.1	27.6	29.0	30.5	32.1
14	23.1	24.6	26.1	27.5	29.0	30.5	32.1
15	23.2	24.7	26.0	27.5	28.9	30.4	32.0
16	23.4	24.7	26.0	27.4	28.8	30.3	31.9

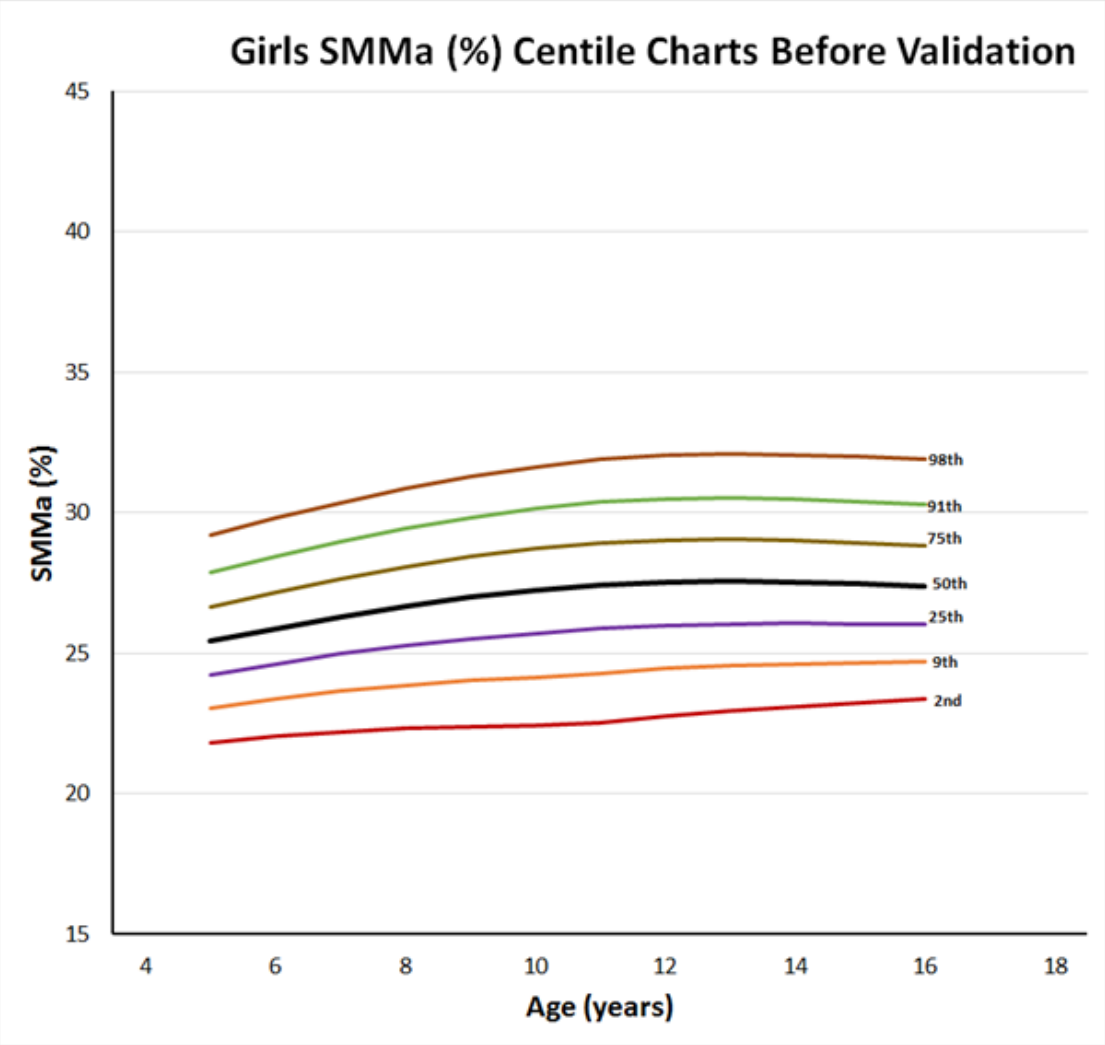

Figure 7(g) %SMMa centile charts for Girls before the application of validated equation

Table 7(h). Tabulated Girls' per cent appendicular skeletal muscle mass (%SMMa) centile values by exact age after the application of validated equation

Girls	SMMa (%) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	19.9	20.8	21.6	22.5	24.5	27.2	29.2
6	20.2	21.2	21.9	22.9	24.8	27.6	29.6
7	20.6	21.5	22.3	23.2	25.2	28.0	30.2
8	20.9	21.9	22.7	23.6	25.6	28.4	30.6
9	21.3	22.3	23.0	24.0	26.0	28.8	31.0
10	21.6	22.6	23.4	24.3	26.4	29.2	31.4
11	22.0	22.9	23.7	24.7	26.7	29.5	31.8
12	22.3	23.3	24.1	25.0	27.3	30.1	32.2
13	22.7	23.6	24.5	25.3	27.8	30.6	32.6
14	22.9	23.9	24.7	25.6	28.0	30.9	32.9
15	23.3	24.3	25.1	26.0	28.5	31.3	33.3
16	23.6	24.6	25.4	26.4	28.8	31.7	33.6

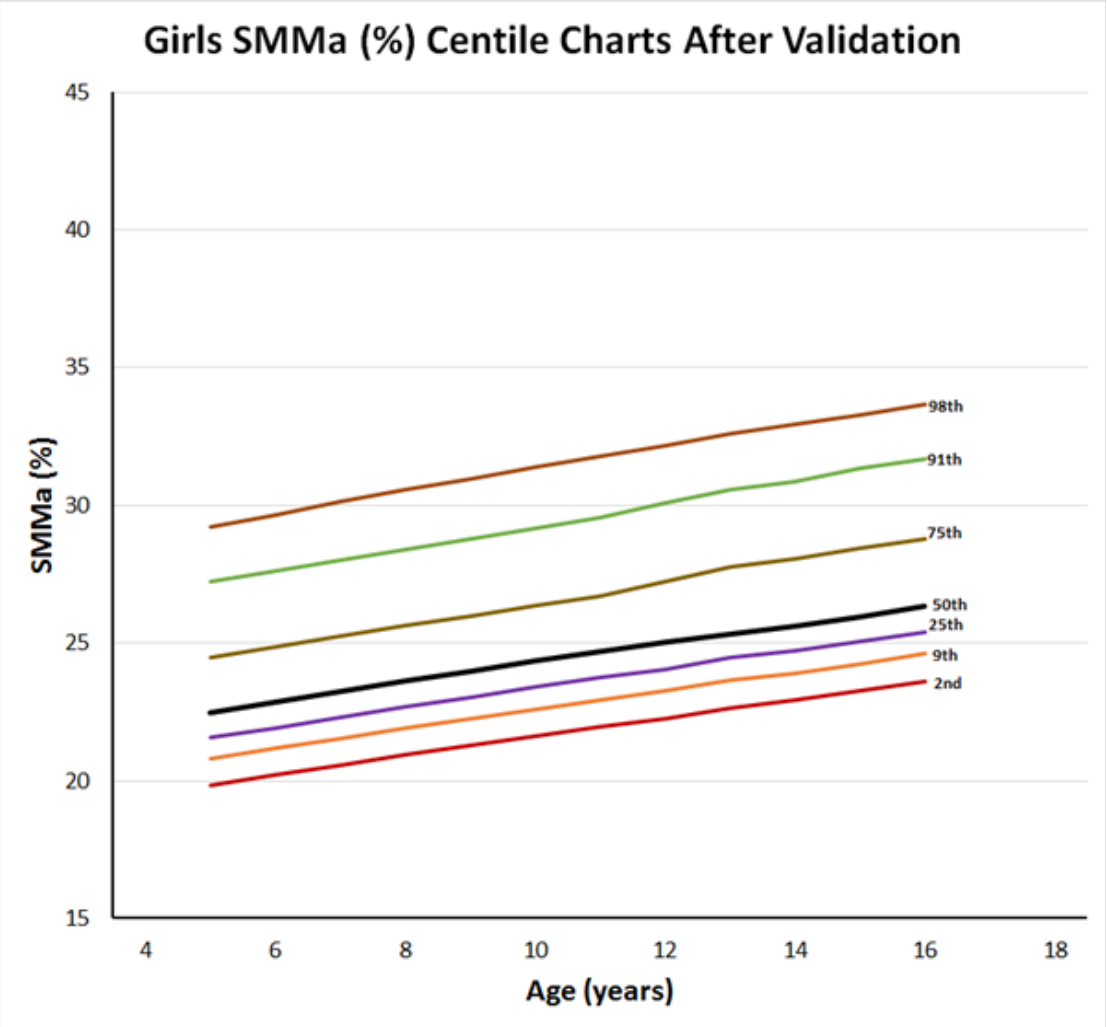

Figure 7(h) %SMMa centile charts for Girls after the application of validated equation

In boys, the 50th centile for %SMMa increases from approximately 24% at age 5 years up to around 34% at age 15 years, remaining relatively flat thereafter (figure 7(e)). In girls, the 50thcentile varied minimally with age with the curves remaining relatively flat between ages 5 and 11 years and then increasing slightly during post-puberty (figure 7(g)). Considering the median centile at age 13, the boys have about 5% more SMMa than girls. After the application of the re-validated equation to the BIA %SMMa boys' data set, a similar pattern of centiles is observed with the 50th centile increasing from 27% at age 5 years up to 31% at age 15 years. In girls, the 50th centile after the application of the validated equation shows an increase from 22.5% at age 5 to 26% in year 15.

Tables 7(i) to 7(l) and figure 7(i) to 7(l) illustrate the tabulated per cent appendicular skeletal muscle mass and fat free mass (%SMMa/FFM) centile values by exact age with their corresponding centile charts for boys and girls before and after the application of the validated equation to the BIA data set. In each chart, the curves represent the 2nd, 9th, 25th, 50th, 75th, 91st and 98th centiles. Figure 7(i) shows that the 50th centile for %SMMa/FFM in boys increases between ages 5 and 14 years from approximately 30% to 42% with the variance being greatest at age 5 years and then decreasing. Between ages 14 and 16 years, the 50th centile line flattens, with the variance decreasing even further, such that between the 2nd and 98th centiles, the range spans only between 38% and 45%. In girls, %SMMa/FFM curves share some of the characteristics of those in boys, increasing with age, with the variance continually decreasing with age.

Table 7(i). Tabulated Boys' per cent appendicular skeletal muscle mass and fat free mass relationship (%SMMa/FFM) centile values by exact age before the application of validated equation

Boys	SMMa/FFM (%) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	23.0	25.2	27.4	29.8	32.3	34.9	37.9
6	24.7	27.1	29.4	31.8	34.1	36.5	39.0
7	26.7	29.2	31.5	33.8	36.0	38.1	40.4
8	28.8	31.3	33.5	35.6	37.7	39.6	41.6
9	30.9	33.2	35.2	37.2	39.1	40.9	42.8
10	32.7	34.8	36.7	38.6	40.4	42.1	43.9
11	34.3	36.2	37.9	39.7	41.4	43.1	44.9
12	35.5	37.3	38.9	40.6	42.3	43.9	45.6
13	36.6	38.2	39.7	41.3	42.9	44.5	46.2
14	37.3	38.7	40.1	41.5	43.0	44.5	46.1
15	37.7	38.9	40.1	41.4	42.7	44.0	45.6
16	37.9	39.0	40.0	41.1	42.3	43.5	44.9

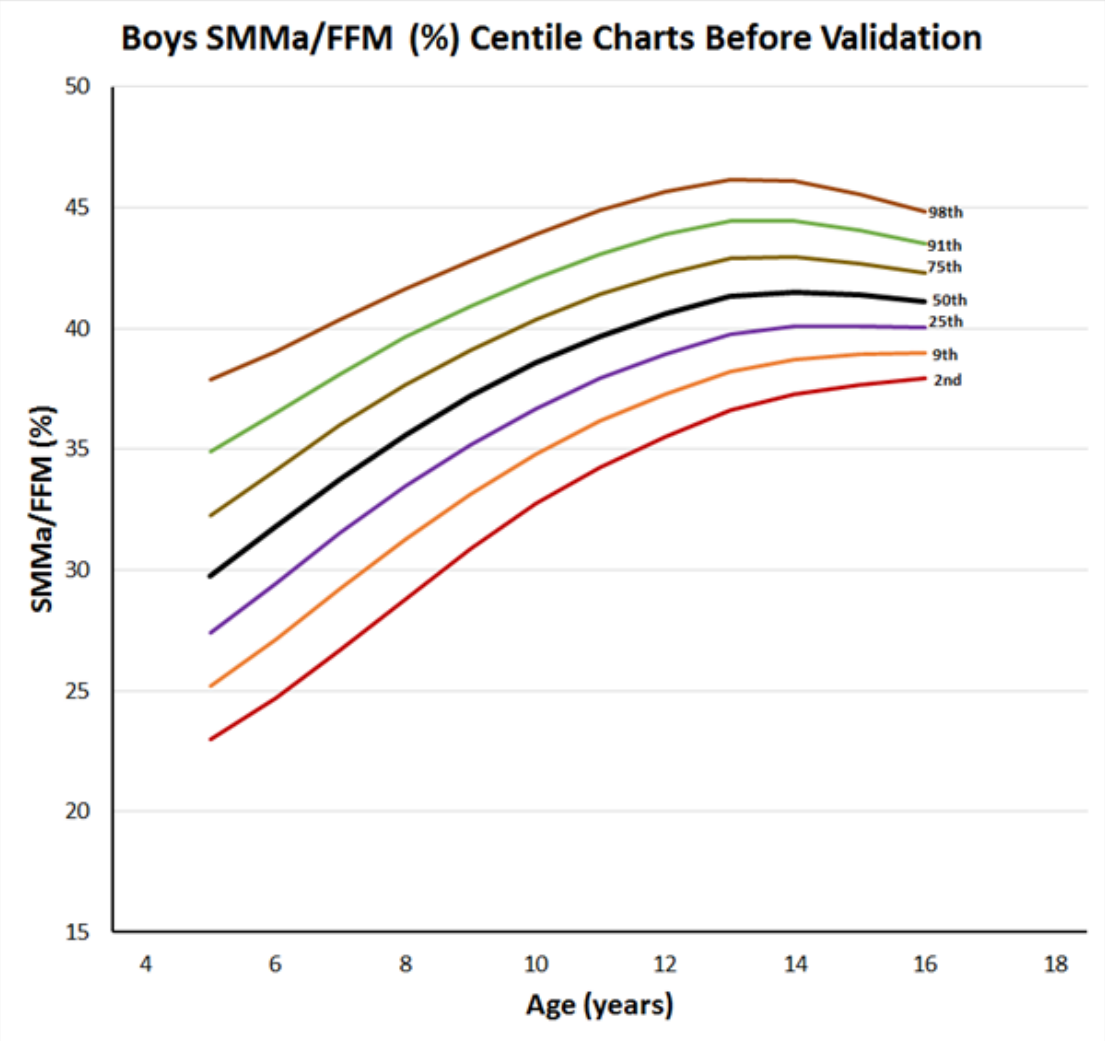

Figure 7(i) %SMMa/FFM centile charts for Boys before the application of validated equation

Table 7(j). Tabulated Boys' per cent appendicular skeletal muscle mass and fat free mass relationship (%SMMa/FFM) centile values by exact age after the application of validated equation

Boys	SMMa/FFM (%) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	26.4	28.8	31.2	33.6	36.2	38.7	41.6
6	28.0	30.6	33.0	35.4	37.8	40.1	42.5
7	29.7	32.5	34.8	37.1	39.3	41.4	43.6
8	31.7	34.3	36.6	38.7	40.8	42.7	44.6
9	33.6	36.1	38.2	40.2	42.1	43.9	45.7
10	35.4	37.7	39.6	41.5	43.3	45.0	46.8
11	37.0	39.0	40.9	42.7	44.4	46.1	47.8
12	38.3	40.2	41.9	43.6	45.3	46.9	48.7
13	39.4	41.1	42.7	44.3	45.9	47.5	49.2
14	40.0	41.5	42.9	44.4	45.9	47.4	49.0
15	40.1	41.4	42.6	43.9	45.3	46.7	48.3
16	40.0	41.1	42.2	43.3	44.5	45.8	47.2

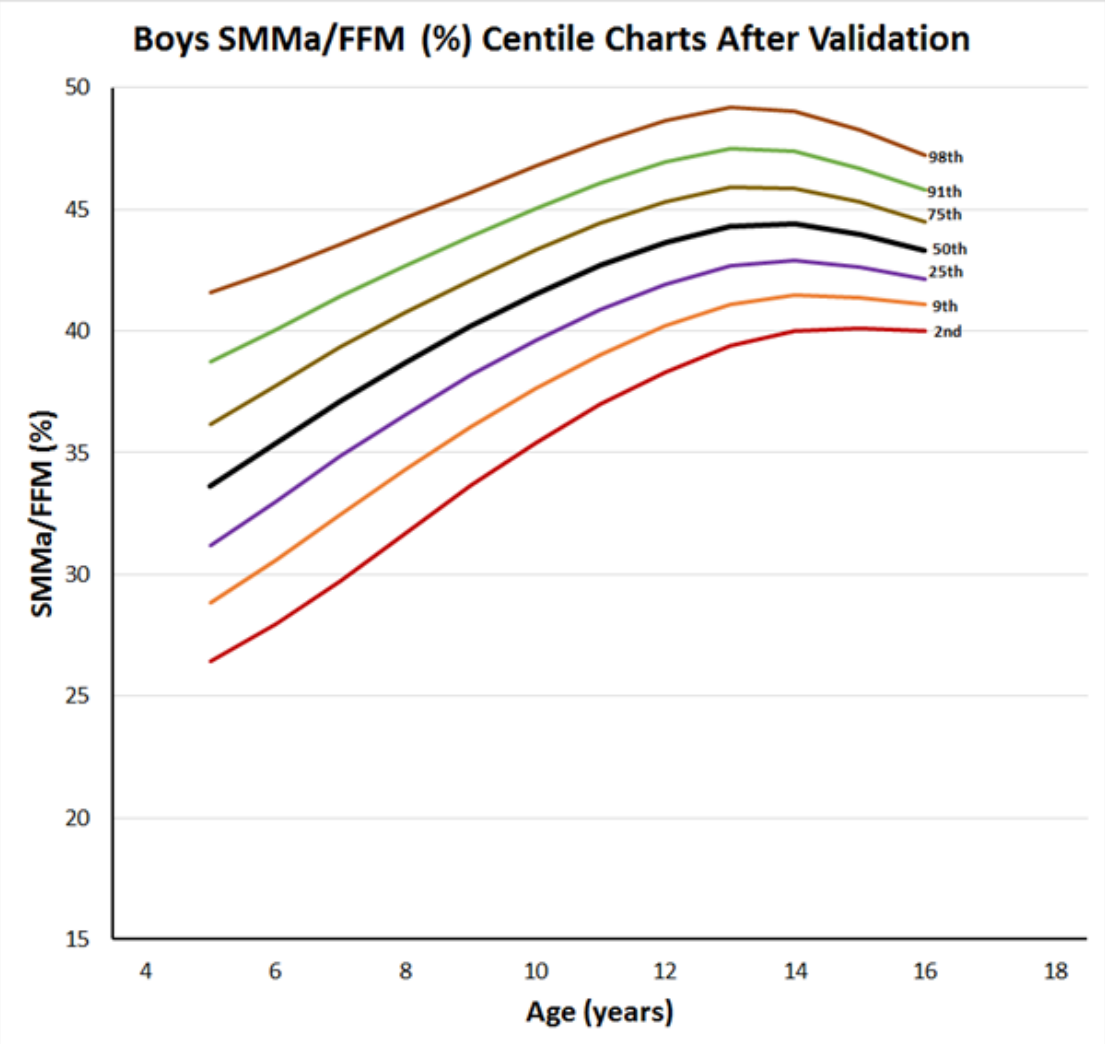

Figure 7(j) %SMMa/FFM centile charts for Boys after the application of validated equation

Table 7(k). Tabulated Girls' per cent appendicular skeletal muscle mass and fat free mass relationship (%SMMa/FFM) centile values by exact age before the application of validated equation

Girls	SMMa/FFM (%) Centiles before validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	28.3	29.6	30.7	32.0	33.3	34.6	36.1
6	29.5	30.7	31.9	33.1	34.4	35.7	37.2
7	30.5	31.8	32.9	34.2	35.5	36.8	38.3
8	31.5	32.8	33.9	35.2	36.5	37.8	39.2
9	32.4	33.6	34.8	36.0	37.3	38.6	40.0
10	33.2	34.4	35.5	36.7	38.0	39.3	40.7
11	33.8	34.9	36.0	37.2	38.4	39.7	41.0
12	34.2	35.3	36.4	37.5	38.7	39.8	41.2
13	34.4	35.5	36.6	37.6	38.8	39.9	41.1
14	34.7	35.7	36.7	37.8	38.8	39.9	41.1
15	35.0	36.0	37.0	38.0	39.0	40.0	41.2
16	35.4	36.4	37.3	38.3	39.3	40.3	41.4

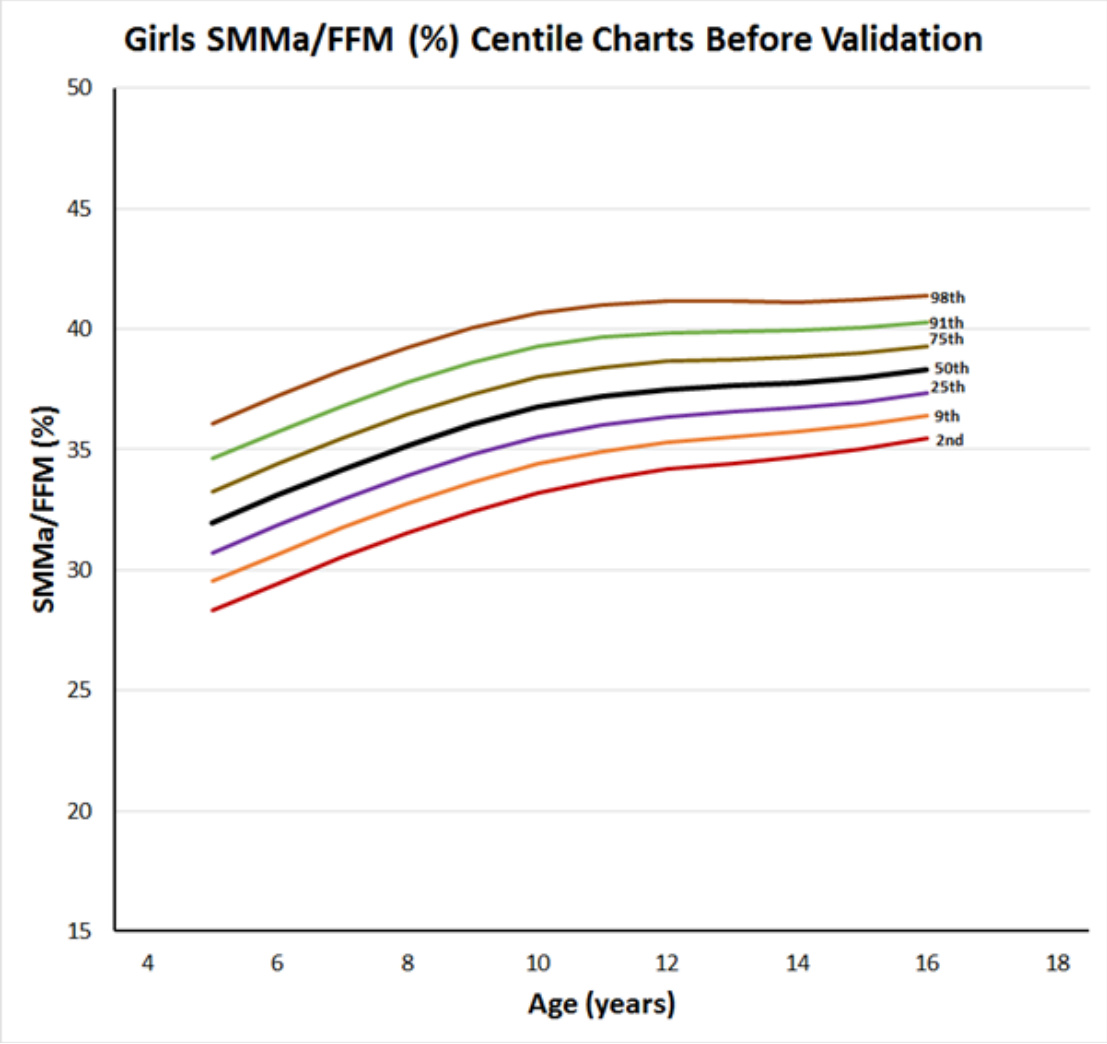

Figure 7(k) %SMMa/FFM centile charts for Girls before the application of validated equation

Table 7(l). Tabulated Girls' per cent appendicular skeletal muscle mass and fat free mass relationship (%SMMa/FFM) centile values by exact age after the application of validated equation

Girls	SMMa/FFM (%) Centiles after validation						
Age	2nd	9th	25th	50th	75th	91th	98th
5	29.6	30.1	30.5	31.0	31.6	32.2	33.0
6	30.2	30.7	31.2	31.8	32.5	33.2	34.2
7	30.8	31.4	32.0	32.7	33.5	34.3	35.5
8	31.3	32.0	32.7	33.5	34.4	35.5	36.8
9	31.8	32.6	33.4	34.3	35.4	36.6	38.2
10	32.4	33.3	34.2	35.3	36.5	37.9	39.6
11	33.0	34.0	35.1	36.3	37.6	39.1	40.9
12	33.8	34.9	36.0	37.2	38.5	40.0	41.9
13	34.6	35.6	36.6	37.8	39.2	40.6	42.5
14	34.9	35.9	36.9	38.0	39.3	40.7	42.4
15	35.0	35.8	36.8	37.8	39.0	40.3	41.9
16	34.9	35.7	36.5	37.5	38.5	39.7	41.1

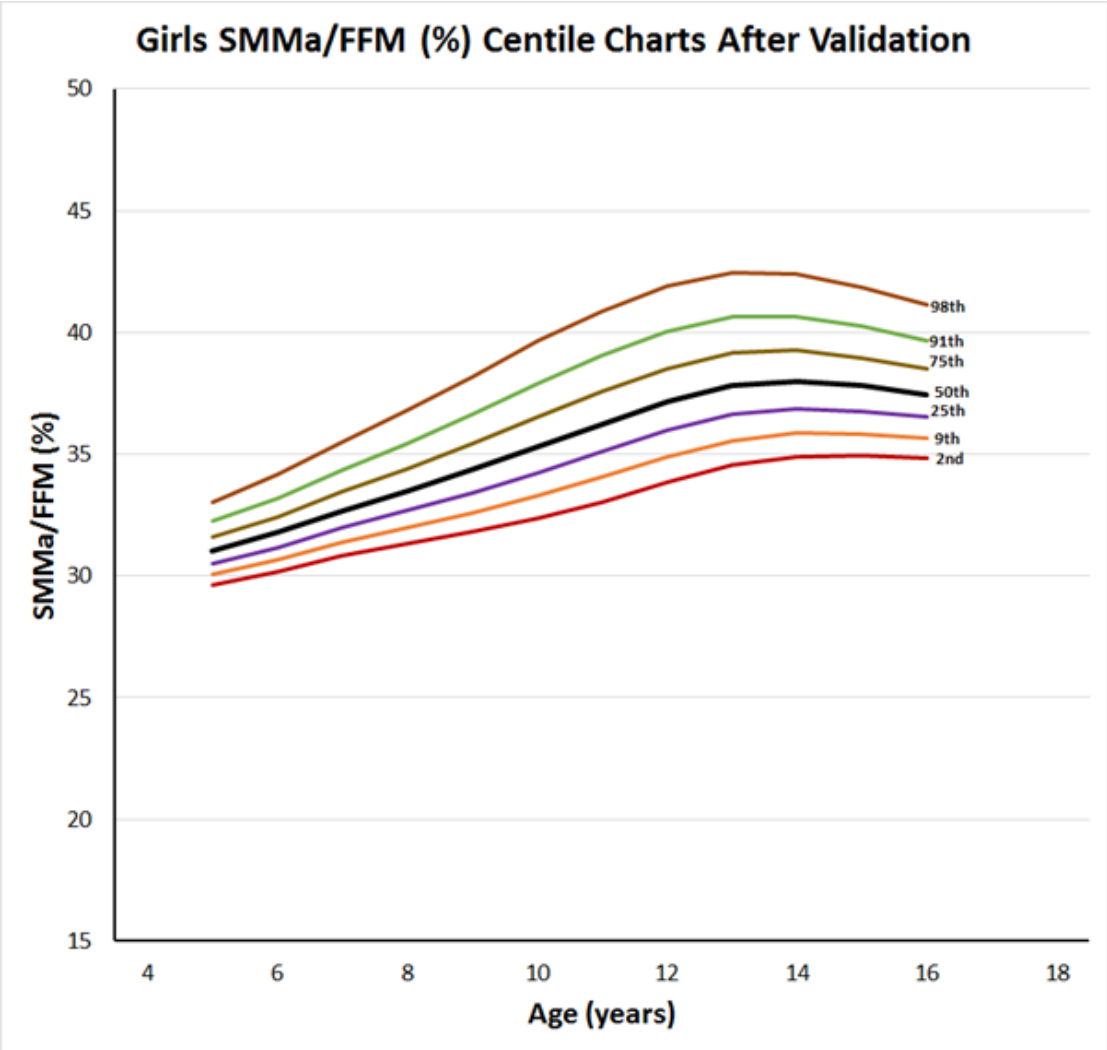

Figure 7(l) %SMMa/FFM centile charts for Girls after the application of validated equation

7.5 Discussion

In this chapter, various gender specific SMMa percentile charts for African and Caribbean children have been produced. The charts show that SMM (kg) increases with increasing age, with a noticeable pubertal spurt in boys which is either absent or less obvious in girls. Heymsfield asserted to the fact that during growth and especially around puberty, boys develop a greater muscle mass and bone mass than girls - changes which bring about sexual dimorphism in girls and boys (Heymsfield, 2005). These charts also reveal that %SMM increases in boys up to puberty, at which point it remains stable as a proportion of whole body mass up to age 16 years or more. In girls, an opposite pattern is observed with %SMM remaining stable up until puberty, at which point it starts to rise. %SMM is consistently lower in girls compared to boys which are likely a result of their higher absolute and proportional body fatness (McCarthy et al, 2011). This study does not provide information on the determinants of these differences which may include genetic, hormonal, environmental, dietary and nutritional signals to growth and development.

Expressing SMMa as a percentage of FFM across the age range removes the influence of body fatness and allows clearer consideration of both gender differences and how SMMa varies in relation to FFM with increasing age. These charts reveal similarities between boys and girls in this variable both in absolute terms and across the age range. The fact that both the change and variance decrease with increasing age suggests a previously undescribed tendency towards stabilisation and standardisation of the SMM: FFM ratio in late adolescence and early adulthood. A markedly reduced variance would be predicted as children emerge from puberty at variable ages, but the fact that it is markedly lower than before they entered puberty, suggests a biological mechanism that

is seeking to establish a rather standard ratio between SMM and the other components of FFM (McCarthy et al, 2011).

To date, no other study has examined skeletal muscle mass in African-Caribbean children using BIA as the assessment tool. It is thus difficult to make comparisons with other studies. However it is encouraging to note that the corrected BIA-derived values obtained in this study compare well with the study by McCarthy et al (2014) with the SMMa values in the African-Caribbean children being consistently and slightly higher compared with age- and gender-equivalent white European children. There is a need for this work to be replicated in sub-Saharan, African-American and Caribbean populations and begin to be used in both clinical and in epidemiological studies. This will allow cross-cultural comparisons to be made. The bonus of having SMMa reference curves for African-Caribbean children is that they will help identify children who have low SMMa for their age and gender and are potentially at risk for metabolic disease and later sarcopenia. Interventions such as strength-gaining exercises (at an appropriate age) could be initiated in order to boost SMMa. However, due to limited assessment tools, it is unknown whether such forms of intervention are able to enhance muscle gain.

Muscle-to-fat-ratio (MFR) calculations for various ages and gender revealed wide range for the boys and girls as illustrated above and also in appendix F below. Since excess fat and muscle have reciprocal influences on glucose metabolism and insulin sensitivity, MFR would be a better predictor of metabolic health and a better tool that can be used for the assessment and prevention of the metabolic syndrome than BMI.

These are the first, and to date, only curves produced to illustrate gender and age-related variation in SMMa for black children and youths living in the UK. Whole body and SMMa have been quantified in both adults and children using reference laboratory

methods (Kim et al, 2002). One recent study by Wang et al recruited a paediatric sample with a similar age range and mean age to this study, but both the mean weight and height were higher in their sample, thus making SMMa comparisons less straightforward (Wang et al, 2007).

These SMMa charts would help monitor the SMM level in the African and Caribbean childhood population.

Study Limitation

The sample size obtained for drawing the percentile charts was appreciable. However, the number of children in the lower age group (example five and six years) was less compared to the numbers for the older children.

Chapter 8: Blood Pressure Percentile Tables for African and Caribbean Children

8.1 Introduction

High blood pressure (BP) or hypertension is a common morbidity associated with overweight and obesity in people of black descent. Although overt hypertension is not common among children per se, a higher prevalence of hypertension and higher mean diastolic and systolic blood pressures have been observed in adults of African and Caribbean background compared with Caucasians (Charles and Raj, 2003). In 2002, the prevalence rate of hypertension in Africans and Caribbean men and women was 31% and 34% respectively compared with 19% and 13% in Caucasian men and women (Lane et al, 2002). Also in a study conducted in South London between 1994 to 1996, it was found that the prevalence of hypertension was raised two to threefold among UK residents of African, Caribbean and South Asian origin compared to white people (Francesco et al, 1997). Indeed, hypertension prevalence is three to four times higher in the African population than in the Caucasian population and stroke (a common consequence of high BP) prevalence is highest in men of Caribbean origin (Gateman et al, 2009; Sproston and Mindell, 2006).

Unfortunately, the BP of children and youths in general is rarely measured at paediatric clinics, partly because of the difficulty in interpreting the measured BP value since there are no specific cut off values for blood pressure measurement in children owing to the normal rise in blood pressure with age and the paucity of evidence about what constitutes hypertension in children. In addition, there is no satisfactory definition of hypertension in children (Goonasekera and Dillion, 2000). Interestingly, high blood pressure is one of the major modifiable risk factors for

cardiovascular diseases - the number one cause of death in the world. Hence, together with measurement of obesity and overweight, childhood BP should be monitored especially in black Africans and Caribbean people to ensure that early interventional measures are put in place to avoid the clinical/health consequences of hypertension in this ethnic minority group. In this chapter, BP and height are considered together because generally, in children, BP is directly related to height (Yuki et al, 2010).

8.2 Aim

The aim of this study is to derive BP percentile tables for African and Caribbean children.

8.3 Subjects and Method

Anthropometric data, systolic and diastolic BP data on children was obtained from the Health Survey for England from 1991 to 2008 and data were taken in a consistent manner from the various years. HSE publishes standard operating procedures (SOPs) for all measurements obtained in the surveys, thus attempting, as far as possible to ensure consistency of measures across all geographical areas and between annual surveys (HSE DATA ARCHIVES: 1991-2008). For the various yearly dataset, coding was well analysed to ensure all the children were from African and Caribbean background. It was also ensured that consistent methods were used in the collecting of this data. The data were sorted to derive information on a total of 900 African and Caribbean children aged 5 – 18 years. Diastolic and systolic blood pressures as well as height, weight and body mass index of the children were retrieved for analysis using SPSS version 17. Out of the total number of children, 99% had valid parameters which could be analysed. The analysis of the data involved the sorting out of the data into various ages (5 to 18 years) and gender. For each age group and gender, the 50th, 75th, 90th and 95th height percentile were determined. Then for each height percentile of a

particular age group and gender, the respective 50th, 90th, 95th and 99th percentile of both the systolic and diastolic pressures was determined. Then the results were tabulated in an ascending order for easy reading and comparison.

8.4 Results

Tables 8(a) and 8(b) show the descriptive statistics for the systolic and diastolic BP data of the African and Caribbean children obtained from the Health Survey for England from 1991 to 2008 presented in narrow age ranges (5-7, 8-10, 11-13, 14-16 and 17-18).

Table 8(a): Descriptive statistics for the Boys' Systolic and Diastolic BP data for Sample Population of African and Caribbean children.

BOYS						Values = ± SD
Age (yrs)	n	*Height* (cm)	*Weight* (kg)	*Systolic BP* (mmHg)	*Diastolic BP* (mmHg)	*BMI* (kg/m^2)
5-7	86	123.0	25.3	106	59	16.6
		7.6	5.1	10	8	2.1
8-10	101	139.5	35.8	111	61	18.1
		9.2	9.8	10	8	3.3
11-13	90	156.0	48.2	114	59	19.5
		9.5	12.5	11	8	3.4
14-16	81	171.7	60.4	117	56	20.4
		8.4	11.8	10	10	3.2
17-18	37	178.2	78.0	122	62	24.6
		7.2	21.7	10	10	6.2

Table 8(b): Descriptive statistics for the Girls' Systolic and Diastolic BP data for Sample Population of African and Caribbean children.

GIRLS						Values = ± SD
Age (yrs)	n	*Height* (cm)	*Weight* (kg)	*Systolic BP* (mmHg)	*Diastolic BP* (mmHg)	*BMI* (kg/m^2)
5-7	75	123.4	26.5	105	59	17.2
		7.1	7.1	7	9	2.9
8-10	95	140.3	35.6	107	58	17.9
		9.5	9.0	9	8	3.1
11-13	96	154.9	51.9	111	59	21.5
		8.4	13.7	9	8	4.9
14-16	102	163.8	62.9	116	60	23.3
		5.8	14.0	9	8	4.6
17-18	58	163.7	63.1	117	61	23.6
		5.8	13.1	11	10	5.3

Tables 8(c) to 8(f) and figures 8(a) to 8(d) illustrate the tabulated blood pressure centile values by exact age with their corresponding percentile curves/charts for the African and Caribbean boys and girls respectively ranging from 2^{nd}, 9^{th}, 25^{th}, 50^{th}, 75^{th}, 91^{st} and 98^{th} percentiles and tables 8(g) and 8(h) present the blood pressure centile tables for the same sample group.

Table 8(c). Tabulated African and Caribbean Boys' BP Systolic centile values by exact age

Boys	BP Systolic Centiles						
Age	2nd	9th	25th	50th	75th	91th	98th
5	89	92	96	101	106	113	123
6	90	94	99	104	110	117	126
7	92	96	101	106	113	120	129
8	92	97	103	108	115	122	130
9	93	98	104	110	116	123	132
10	93	99	105	111	118	125	133
11	93	100	106	112	119	126	134
12	94	101	107	114	121	128	136
13	95	102	109	116	123	130	137
14	96	103	110	117	124	131	139
15	97	104	111	118	125	132	140
16	98	105	112	119	126	133	141
17	100	107	113	120	127	134	142
18	101	108	115	122	129	136	144

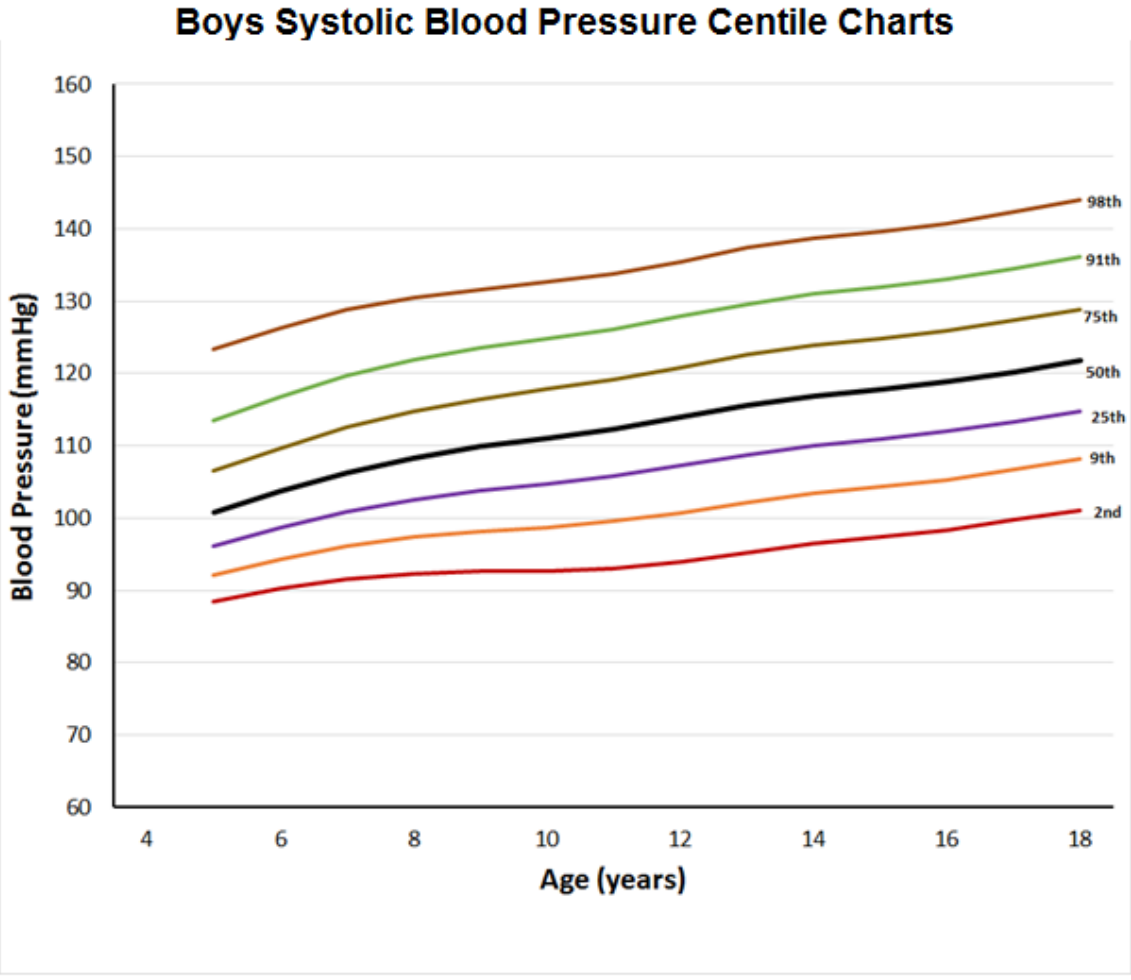

Figure 8(a). Boys' Systolic Blood Pressure centile charts

Table 8(d). Tabulated Boys' Diastolic BP centile values by exact age

Boys	BP Diastolic Centiles						
Age	2nd	9th	25th	50th	75th	91th	98th
5	44	49	53	58	64	69	75
6	45	49	54	59	64	70	76
7	45	50	54	59	65	70	77
8	45	50	55	60	65	71	78
9	45	50	54	60	65	71	78
10	45	49	54	59	65	71	78
11	44	48	53	58	64	71	78
12	43	48	52	58	64	70	78
13	42	47	52	57	63	70	79
14	42	46	51	57	63	70	79
15	42	46	51	56	63	70	80
16	42	47	51	57	64	72	82
17	43	48	52	58	65	73	84
18	44	49	54	60	67	76	87

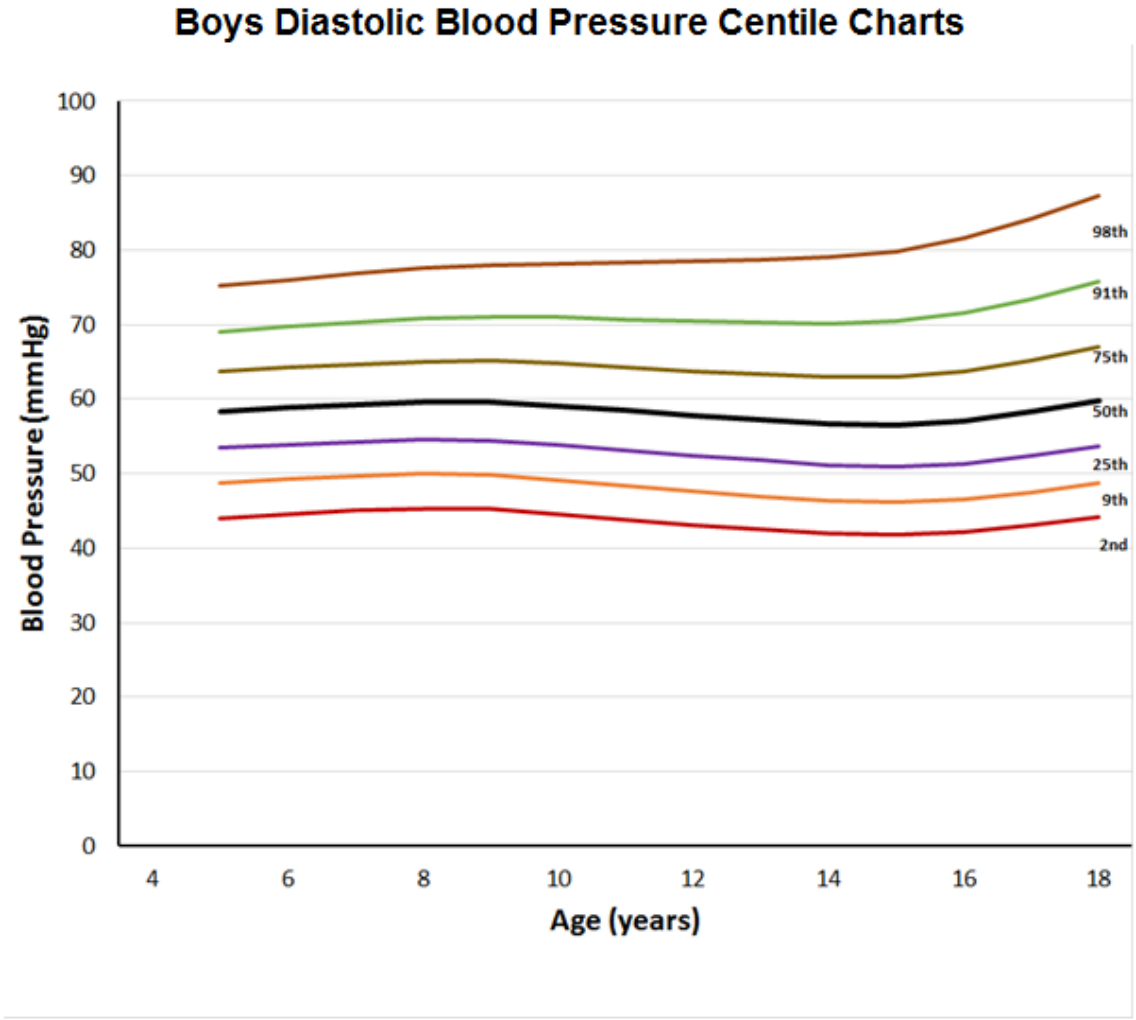

Figure 8(b). Boys' Diastolic Blood Pressure centile charts

Table 8(e). Tabulated Girls' Systolic BP centile values by exact age

Girls	BP Systolic Centiles						
Age	2nd	9th	25th	50th	75th	91th	98th
5	88	94	99	104	108	112	116
6	88	94	100	105	109	114	118
7	88	95	100	106	110	115	120
8	88	95	101	107	112	117	122
9	89	96	102	108	113	119	124
10	90	97	103	109	115	121	126
11	91	98	105	111	117	123	128
12	92	99	106	112	118	124	130
13	93	100	107	114	120	126	132
14	94	101	108	115	121	127	134
15	95	102	109	115	122	128	135
16	96	103	109	116	123	130	137
17	97	104	110	117	124	131	139
18	98	104	110	117	124	132	141

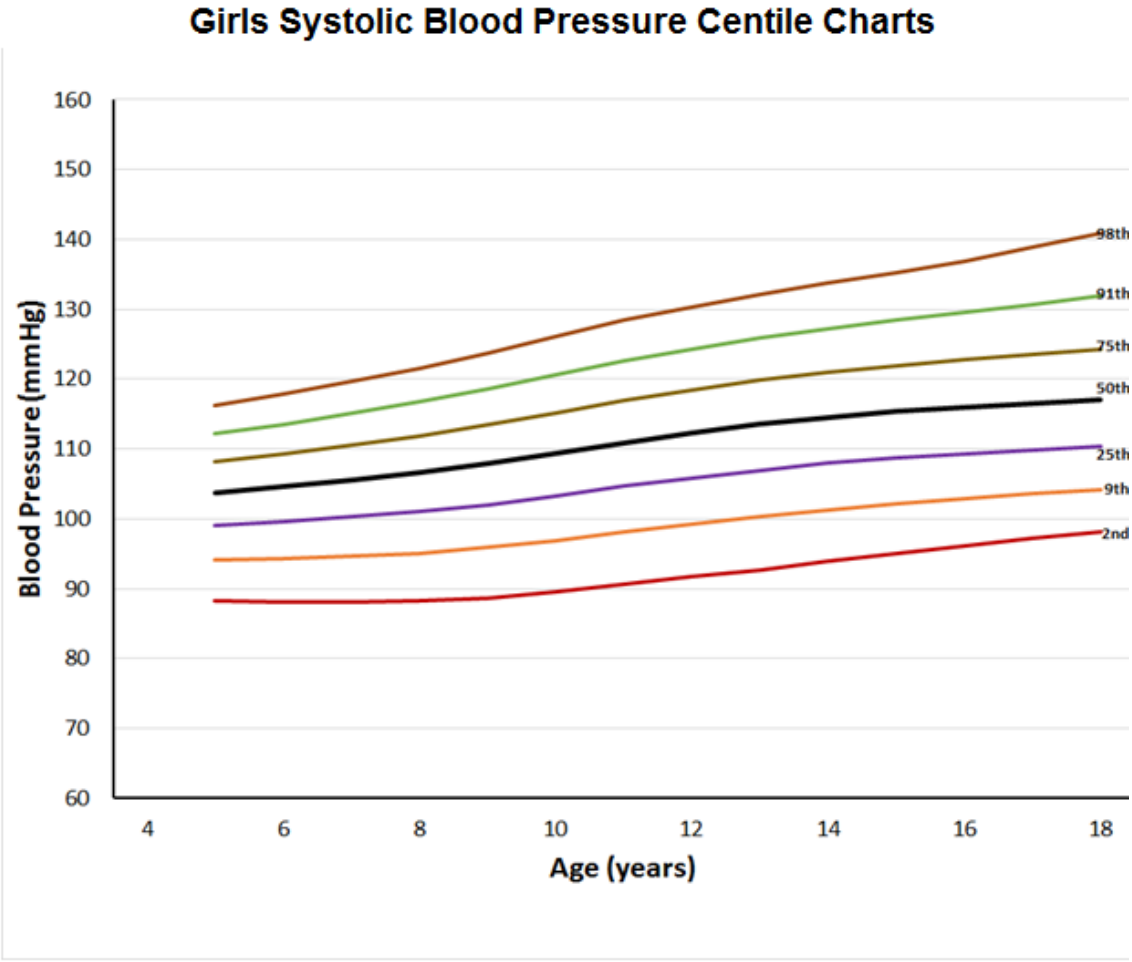

Figure 8(c).Girls' Systolic Blood Pressure centile charts

Table 8(f). Tabulated Girls' Diastolic BP centile values by exact age

Girls	BP Diastolic Centiles						
Age	2nd	9th	25th	50th	75th	91th	98th
5	43	48	52	58	64	71	80
6	43	48	53	58	64	71	79
7	44	48	53	58	64	71	79
8	44	48	53	58	64	70	78
9	44	48	53	58	64	70	77
10	44	48	53	58	64	70	77
11	44	49	53	58	64	70	76
12	44	49	54	59	64	70	76
13	45	49	54	59	64	70	77
14	45	49	54	59	65	71	77
15	44	49	54	60	65	71	78
16	44	49	55	60	66	72	79
17	44	49	55	61	67	73	80
18	43	49	55	61	68	74	81

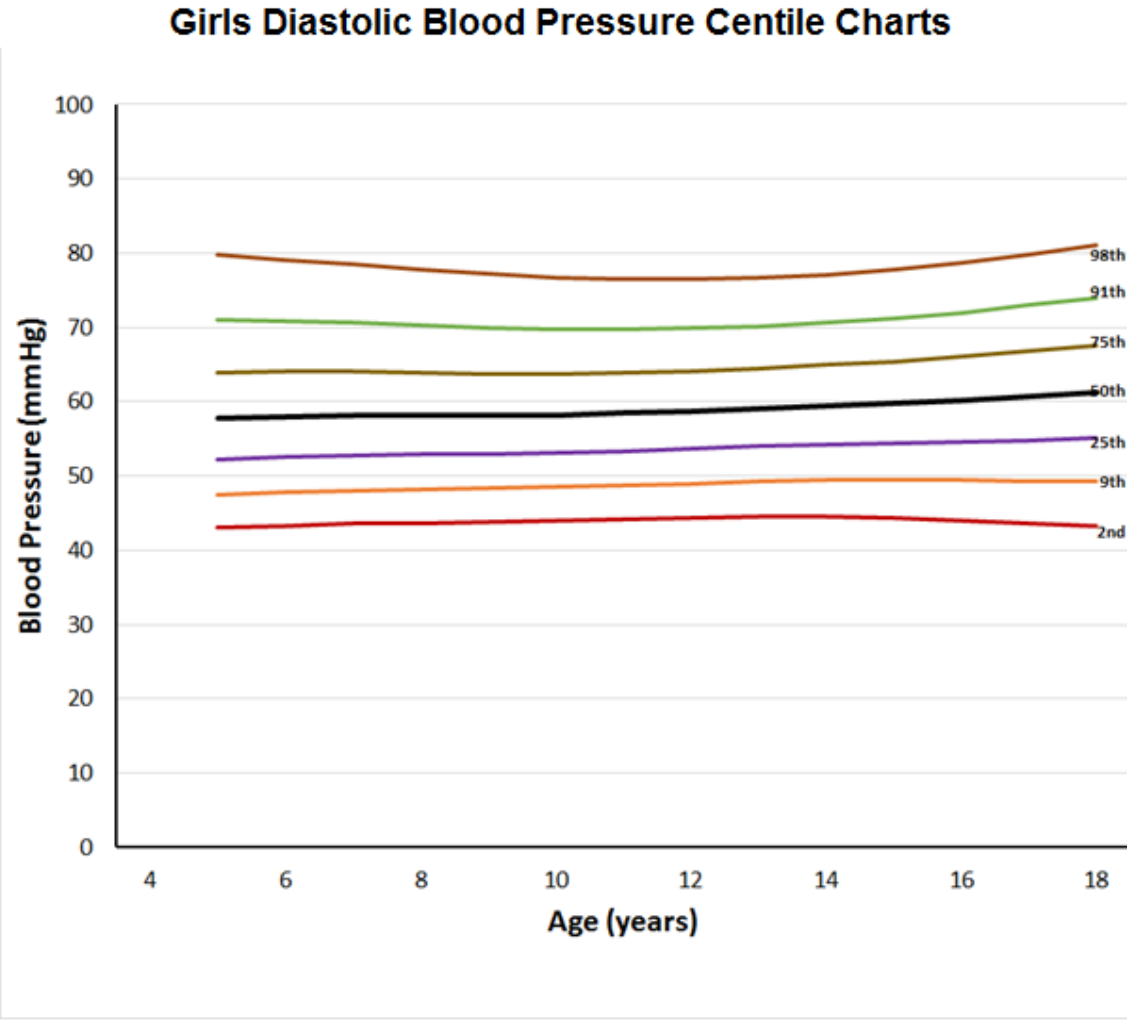

Figure 8(d).Girls' Diastolic Blood Pressure centile charts

Generally, both systolic and diastolic BP increased with age for the African and Caribbean children as shown in tables 8(a) and 8(b) above. There was no significant difference in BP observed with gender. However, boys had relatively higher BP, especially systolic BP (from 106 to 122 mmhg) compared to girls (from 105 to 117 mmhg). This is consistent with the effect of body size (indicated by weight and height) on blood pressure most significantly noticed with systolic BP (Jackson et al, 2007). However, girls who gain more weight than boys during puberty could have higher blood pressures than boys (William et al, 2004). Body weight was observed to be a significant factor influencing BP in this study. BP fairly increased with height.

Table 8(g): BP levels African-Caribbean Children aged 5-18 years (Boys)

Age	BP Percentile	SBP, mmHg				DBP, mmHg			
		Percentile of Height				Percentile of Height			
		50th	75th	90th	95th	50th	75th	90th	95th
5	50th	99	102	101	102	52	52	53	55
	90th	104	104	105	105	68	67	66	68
	95th	104	104	107	107	68	68	68	72
	99th	104	104	107	107	68	68	68	72
6	50th	101	102	102	102	59	59	59	59
	90th	128	126	125	125	74	74	73	72
	95th	128	128	128	128	74	76	75	75
	99th	128	128	128	128	74	78	78	78
7	50th	106	106	108	108	60	58	58	58
	90th	120	122	122	122	72	72	73	73
	95th	120	126	124	124	73	73	74	74
	99th	120	130	130	130	73	74	75	75
8	50th	107	107	107	108	62	60	61	62
	90th	120	116	120	122	81	76	74	73
	95th	120	120	122	138	81	81	81	81
	99th	120	120	122	138	81	81	81	81
9	50th	111	111	111	111	61	61	61	60
	90th	115	116	118	120	71	75	74	73
	95th	119	118	120	123	78	78	78	78
	99th	119	119	120	123	78	79	79	79
10	50th	108	112	109	110	62	61	59	59
	90th	132	131	127	126	76	73	70	70
	95th	137	134	132	132	81	78	77	76
	99th	137	137	137	137	82	82	82	82

	50th	108	110	109	110	56	58	57	57
	90th	127	121	120	122	65	71	69	68
	95th	134	132	129	128	66	72	72	72
11	99th	134	134	134	134	66	72	72	72
	50th	114	114	111	113	58	58	57	58
	90th	123	127	124	123	71	76	73	72
	95th	128	128	128	128	77	82	82	82
12	99th	128	128	128	128	77	82	82	82
	50th	116	119	120	120	62	62	62	61
	90th	138	138	138	138	65	67	68	68
	95th	138	139	138	138	67	69	69	69
13	99th	138	139	139	139	67	69	70	70
	50th	115	118	118	119	55	55	55	56
	90th	128	131	130	131	75	75	75	75
	95th	137	150	146	144	76	85	83	81
14	99th	137	156	156	156	76	90	90	90
	50th	112	114	114	112	55	55	53	54
	90th	126	126	126	126	64	63	63	63
	95th	126	132	131	130	64	64	64	64
15	99th	126	133	133	133	64	64	64	64
	50th	116	117	117	116	53	53	53	53
	90th	130	131	129	128	72	70	69	69
	95th	134	133	133	133	79	77	76	76
16	99th	134	134	134	134	79	79	79	79
	50th	119	118	118	118	57	60	61	61
	90th	133	133	133	133	82	76	75	77
	95th	136	135	135	140	87	85	84	84
17	99th	136	136	136	143	87	87	87	87
	50th	119	120	120	120	73	66	62	64
	90th	133	138	136	143	76	75	75	75
	95th	133	139	139	146	76	76	76	76
18	99th	133	139	139	146	76	76	76	76

Table 8(h): BP levels African-Caribbean Children aged 5-18 years (Girls)

		SBP, mmHg				**DBP, mmHg**			
		Percentile of Height				**Percentile of Height**			
Age	**BP Percentile**	**50th**	**75th**	**90th**	**95th**	**50th**	**75th**	**90th**	**95th**
	50th	105	104	104	104	57	56	56	56
	90th	118	110	106	106	80	71	68	68
	95th	121	121	121	121	84	84	84	84
5	99th	121	121	121	121	84	84	84	84
	50th	106	103	104	103	58	57	57	57
	90th	113	113	113	113	67	67	67	67
	95th	117	116	117	117	67	70	70	70
6	99th	117	117	117	117	67	72	72	72
7	50th	106	106	107	106	61	61	61	61

	90th	110	112	113	113	76	76	76	76
	95th	112	113	118	118	76	76	77	77
	99th	112	113	118	118	76	76	81	81
	50th	104	104	104	104	61	62	63	63
	90th	115	116	116	116	74	70	69	72
	95th	116	119	119	119	76	76	76	76
8	99th	116	119	119	119	76	76	76	76
	50th	104	104	104	105	56	56	55	56
	90th	119	119	119	119	65	63	65	66
	95th	120	119	119	128	66	65	66	66
9	99th	120	120	120	128	66	66	66	66
	50th	106	109	109	110	57	57	59	58
	90th	120	120	120	120	72	69	72	71
	95th	120	122	122	122	77	74	74	73
10	99th	120	122	122	122	77	77	77	77
	50th	114	110	110	109	60	60	60	60
	90th	125	122	127	126	70	67	70	70
	95th	129	129	131	130	76	71	74	74
11	99th	129	129	131	131	78	78	78	78
	50th	109	110	110	111	54	56	54	56
	90th	127	127	128	127	65	77	75	74
	95th	127	128	128	128	73	80	80	80
12	99th	127	128	128	128	73	80	80	80
	50th	108	110	111	113	59	59	59	59
	90th	119	124	124	123	74	70	68	68
	95th	122	124	124	124	76	76	76	75
13	99th	122	124	124	124	76	76	76	76
	50th	112	116	118	118	59	59	59	60
	90th	139	132	131	130	65	66	69	68
	95th	140	140	140	140	67	69	82	82
14	99th	140	140	140	140	68	73	82	82
	50th	114	114	114	115	59	59	59	59
	90th	127	127	127	127	63	69	70	70
	95th	127	127	127	127	67	71	79	76
15	99th	127	127	127	127	67	71	91	91
	50th	115	116	116	117	54	59	63	63
	90th	128	128	129	128	76	74	74	74
	95th	128	132	135	133	77	77	77	77
16	99th	128	132	151	151	77	77	77	77
	50th	116	117	114	115	56	56	56	58
	90th	128	135	135	133	74	72	71	71
	95th	139	137	137	136	78	76	76	75
17	99th	139	139	139	139	78	78	78	78
	50th	116	118	117	118	64	64	64	63
	90th	129	134	131	134	71	75	75	75
	95th	132	143	143	143	77	81	79	78
18	99th	132	143	143	143	77	83	83	83

8.5 Discussion

In this chapter, BP percentile charts and BP in relation to height percentile tables have been produced for the African and Caribbean child and youth population living in the UK. The BP tables would help to characterise BP of children of black descent and complement existing BP charts for British children. To use these tables in the clinical setting, the child's height percentile is determined using standard height growth charts. Then the child's measured BP (SBP and DBP) is compared with the numbers provided in the table according to the child's age and height. The child's BP is normal if BP falls below the 90th percentile. If the child's BP is equal to or above the 90th percentile, the measurement should be repeated to confirm elevated BP. BP above the 95th percentile needs follow up as well as BP between 90th and 95th percentiles (USDHHS 2005*).* In this study BP increased generally with age and weight and not particularly with height. This finding is confirmed by previous studies conducted on children. In 2008, Patel et al conducted a study on impact of weight, height and age on blood pressure in school children. It was found that body weight was the most significant predictor of blood pressure (Patel et al, 2008). In 1996 and 1997, similar findings were observed in studies conducted by Anand et al and Thakor et al respectively (Anand and Tandon, 1996), (Thakor et al, 1997). Similarly, in this study BP increased consistently with weight and not with height. Hence, maintaining body weight in childhood is essential for prevention of hypertension in communities.

Previous studies have shown that BP in children is determined by height as well as weight (Patel et al, 2008). However it has been suggested that the proportion of BP attributable to height is physiological whereas that contributed by weight is pathological (Jackson et al, 2007). Consequently some international reference BP charts have been

stratified by height (Chukwunonso, 2011). Additionally, reported increases in the prevalence of paediatric hypertension could be explained by an increased prevalence of overweight and obesity (Jackson et al, 2007). However, caution must be exercised when including obese children in references as this might lead to higher than normal BP (Patel et al, 2008).

Studies conducted in a number of adult populations in Africa have shown that the mean blood pressures of men are higher than those of women (Akinkugbu and Ojo, 1968; Ikeme et al, 1974). Similarly, during childhood and adolescence the mean blood pressures of boys are observed to be slightly higher than those of girls (Jackson et al, 2007). The results in this study confirm this observation where the mean blood pressures of the boys were slightly higher than those of the girls. It has been postulated that these differences in the blood pressures between boys and girls may be related to hormonal and other changes during development such as weight and fat gain.

Londe et al and Buck have shown that higher blood pressure levels in children tend to persist (Londe et al, 1971; Buck, 1973). There are, however recognised levels of arterial blood pressure which if persistent over prolonged periods can lead to increased morbidity and mortality. It is therefore necessary that critical BP cut offs in black children are established to make it easier to identify those with raised arterial blood pressures for early diagnosis and treatment.

There is the likelihood that adult hypertension may begin during childhood and in some cases, childhood high blood pressure has been shown to track into adult life (Bao et al, 1995). Furthermore, there is increasing evidence to prove that childhood high blood

pressure is strongly associated with adult hypertension and this can be established in early life (Dekkers et al, 2002; Lane and Gill, 2004).

Moreover, the prevalence of hypertension and its related diseases is higher and more difficult to treat among people of African origin in the UK (Cruickshank et al, 2001). The majority of adult population-based blood pressure studies have reported increased prevalence of hypertension, higher mean blood pressure levels and higher morbidity and mortality rates attributable to hypertension among Africans and Caribbean than Europeans. In a cross-sectional study to compare blood pressures of adult African and Caribbean origin living in the UK with those of people of European origin, it was observed that the prevalence of hypertension was higher in the former group than the latter (Charles and Raj, 2003). Also in a study conducted in South London between 1994 to 1996, it was found that the prevalence of hypertension was raised two to threefold among UK residents of African, Caribbean and South Asian origin compared to Caucasians (Francesco et al, 1997). Indeed, a number of US studies conducted to assess BP among African-American and white-American children and adolescents have reported higher blood pressure levels among African-Americans than white-Americans (Prineas et al, 1985; Manatunga et al, 1993). Furthermore, in the US, black migrants from Caribbean and West Africa have been observed to have an increased risk of diseases attributable to hypertension such as stroke, coronary heart disease and end-stage renal failure (Lane and Gill, 2003).

The evidence is clear that obesity predisposes to high blood pressure or hypertension and there is an epidemic of obesity in both childhood and adult populations in the UK. This is a cause for concern because high blood pressure, which leads to complications

such as cerebrovascular accident, cardiovascular and renal diseases, can occur during childhood.

The development of reference blood pressure percentile charts and tables have improved the understanding of blood pressure variation in children and have also helped with easy monitoring and diagnosis of hypertension in children worldwide. Reference BP tables have also assisted in easy tracking of childhood BP to adulthood and in identifying high risk individuals for early management.

Study Limitation

The sample size for this study although appreciable, could have been higher if blood pressure in children was regularly measured and recorded over the years. Information on BP in children was lacking from most of the annual HSE reports.

Chapter 9: Waist circumference (WC) percentile charts and tables for African and Caribbean children

9.1 Introduction

Waist circumference is an important measure of adiposity and risk of metabolic diseases in both children and adults (Scott et al, 2004). As a surrogate marker of intra-abdominal or visceral fat, waist circumference acts as a more accurate measure in children because it targets central obesity (McCarthy, 2001). During growth from infancy, childhood and adolescence, fat is laid down subcutaneously and intra-abdominally which changes with age and excessive fat accumulation (Fox et al, 1993). Studies have shown that, an increased abdominal fat accumulation and distribution leads to increased waist circumference in obese children, leading to higher blood pressure, LDL cholesterol and triglycerides, lower levels of HDL cholesterol, higher fasting insulin levels and fat accumulation in the liver – all features of the metabolic syndrome (Flodmark et al, 1994; Freedman et al, 1999). Such evidence indicates that monitoring of waist circumference in children and adolescents could be beneficial as a means of early detection of obesity-related risk. To date waist circumference reference charts have been produced for the UK childhood and adolescent population. These reference charts have been based on the predominantly Caucasian population. Outside the UK, waist circumference reference charts have been generated for African-American childhood population but not yet for any Caribbean or African population. However, given the ethnic-related variation in body composition as previously outlined in the preceding chapters, it would be beneficial to develop specific WC references for the UK African-Caribbean children and youth population.

9.2 Aim

To develop waist circumference percentile charts and tables for African and Caribbean children aged 5 to 18years living in the UK.

9.3 Subjects and Methods

Data on height and waist circumference measured with flexible non-elastic tape of 1336 African and Caribbean children were retrieved from an existing data set of over eight thousand children aged from 5 to 16 years. Fifty two per cent were girls and forty eight per cent were boys. Further details of the subjects and measurement procedures have been presented in chapters two and three. Ethnicity was determined from DFES system provided by the schools. Additionally, waist circumference data on ninety-four African and Caribbean youths aged 17 to 19 years was obtained from archive sources from Health Survey for England (HSE) to supplement the dataset and extend the age range up to and beyond 18 years.

The data were sorted by age groups and gender. Waist circumference smoothed percentile charts were constructed from the data for boys and girls using LMS software (Cole 1990). As previously described, the LMS software/method is used to develop smoothed growth centile curves and deals generally with skewness that might exist in the distribution of the waist circumference measurements. It assumes that the data could be normalised by using a power transformation which removes any skewness from the data by reducing one tail of the distribution and extending the other (Cole, 1990). For each age group and gender, the maximum power required to obtain normality is calculated and summarised as lambda (L) smooth curve. The patterns observed for the mean (M) and co-efficient of variation (S) are also represented by smoothed curves. Finally, the LMS curves developed convert measurements into s.d scores (Cole, 1990).

Additionally, the 1336 African and Caribbean children were sorted into yearly age groups. Then for each age group and gender, the 50^{th}, 75^{th}, 90^{th} and 95^{th} height percentiles were determined. Finally, for each height percentile of a particular age group and gender, the 50^{th}, 75^{th}, 90^{th} and 95^{th} waist circumference percentile was determined. The results were then arranged according to gender and age and tabulated as seen below (Tables WC_{boys} and WC_{girls}).

9.4 Results

Tables 9(a) and 9(b) show the descriptive statistics for the waist circumference data presented in narrow ages ranges (5-7, 8-10, 11-13, 14-16 and 17-18) for the African and Caribbean children. Tables 9(c) and 9(d) and figures 9(a) and 9(b) illustrate the tabulated waist circumference centile values by exact age with their corresponding percentile curves/charts for the African and Caribbean boys and girls respectively ranging from 2^{nd}, 9^{th}, 25^{th}, 50^{th}, 75^{th}, 91^{st} and 98^{th} percentiles.

Table 9(a): Descriptive statistics for the Boys' Sample Population of African and Caribbean children.

BOYS					Values = + SD
Age (yrs)	n	*Height* (cm)	*Weight* (kg)	*BMI* (kg/m^2)	*WC* (cm)
5 - 7	227	122.2	24.6	16.3	55.6
		7.21	6.55	2.86	6.2
8 - 10	248	139.3	35.7	18.2	62.0
		8.08	9.93	3.56	8.4
11- 13	137	155.0	47.3	19.5	67.8
		9.99	12.48	3.95	9.9
14 - 16	32	169.8	59.4	20.5	72.3
		8.36	12.73	3.74	8.5
17 - 18	36	176.2	75.2	24.2	78.1
		7.10	19.30	6.10	22.0

Table 9(b): Descriptive statistics for the Girls' Sample Population of African and Caribbean children.

GIRLS					Values = + SD
Age (yrs)	n	*Height* (cm)	*Weight* (kg)	*BMI* (kg/m^2)	*WC* (cm)
5 - 7	222	123.0	25.7	16.7	56.5
		7.7	6.6	2.9	6.5
8 - 10	276	140.5	38.2	19.0	63.2
		9.1	11.4	4.1	9.5
11- 13	169	155.5	49.0	20.2	67.7
		7.6	11.3	4.2	8.1
14 - 16	27	161.5	56.5	21.7	69.1
		6.4	8.6	3.2	6.6
17 - 18	57	162.7	56.9	21.5	64.8
		5.9	9.4	3.5	13.3

Generally, mean waist circumference increased with age. For boys there was continuous increase while in the case of the girls there is a gradual increase from age five to thirteen and then the curves tend to show a slight plateau and then a decline. This may likely reflect the different periods of puberty onset which are controlled by hormonal changes with gender specific influences on waist circumference.

The waist circumference percentile tables show that waist circumference is proportionally related to height and as height/stature increases, waist circumference also increases for the various age groups and gender. Considering the 50^{th} percentile for the boys (Figure 9a), there is an incremental rise in waist circumference with height from age five to fifteen years. A sharp rise is observed from 5 to 11 years, the pre-pubertal years, and a slow or gradual rise from 12 years to 15 years for the boys. A similar but lesser trend is observed for the girls.

Table 9(c). Tabulated African and Caribbean Boys' waist circumference centile values by exact age

Boys	WC Centiles						
Age	2nd	9th	25th	50th	75th	91th	98th
5	46.7	48.4	50.4	52.7	55.6	59.2	64.5
6	47.1	49.0	51.2	53.8	57.1	61.3	67.8
7	47.7	49.8	52.2	55.1	58.8	63.8	71.6
8	48.9	51.2	53.8	57.0	61.2	66.9	76.3
9	50.3	52.9	55.7	59.4	64.1	70.6	81.5
10	51.9	54.7	57.8	61.8	67.0	74.3	86.7
11	53.2	56.2	59.6	64.0	69.7	77.6	91.2
12	54.4	57.6	61.3	66.0	72.1	80.6	95.2
13	55.8	59.2	63.2	68.3	74.9	84.0	99.3
14	56.8	60.6	64.9	70.4	77.5	87.2	103.0
15	57.3	61.5	66.1	72.0	79.6	89.7	105.7
16	57.2	61.7	66.8	73.1	81.1	91.6	107.4
17	56.5	61.5	67.0	73.7	82.2	92.8	108.1
18	55.4	60.8	66.8	74.0	82.8	93.5	108.1

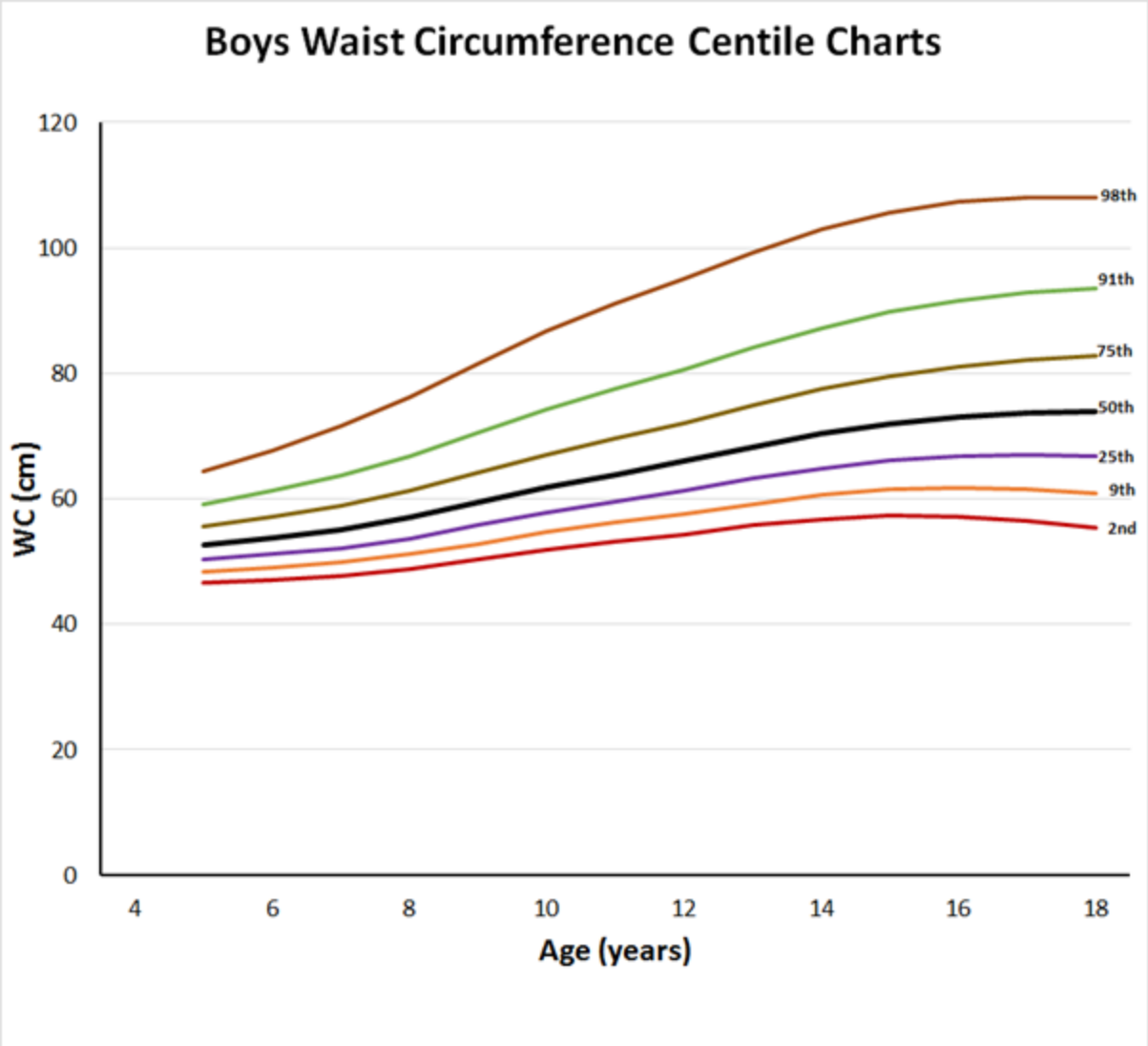

Figure 9(a). Waist circumference centile charts for African and Caribbean Boys

Table 9(d). Tabulated African and Caribbean Girls waist circumference centile values by exact age

Girls	WC Centiles						
Age	2nd	9th	25th	50th	75th	91th	98th
5	45.8	47.8	49.9	52.5	55.8	59.9	66.0
6	46.6	48.8	51.2	54.2	57.9	62.7	70.1
7	47.4	49.9	52.5	55.9	60.1	65.7	74.5
8	48.5	51.2	54.2	57.9	62.6	69.0	79.1
9	49.9	52.8	56.1	60.2	65.4	72.4	83.6
10	51.5	54.6	58.2	62.6	68.2	75.7	87.7
11	53.0	56.3	60.1	64.7	70.7	78.5	90.8
12	54.2	57.6	61.6	66.4	72.6	80.7	93.2
13	54.8	58.4	62.5	67.5	73.9	82.2	94.8
14	54.6	58.4	62.6	67.8	74.4	82.9	95.7
15	53.8	57.6	61.9	67.3	74.0	82.8	96.0
16	52.4	56.3	60.7	66.2	73.1	82.2	95.9
17	50.6	54.6	59.1	64.7	71.9	81.3	95.5
18	48.9	52.9	57.5	63.2	70.6	80.2	95.0

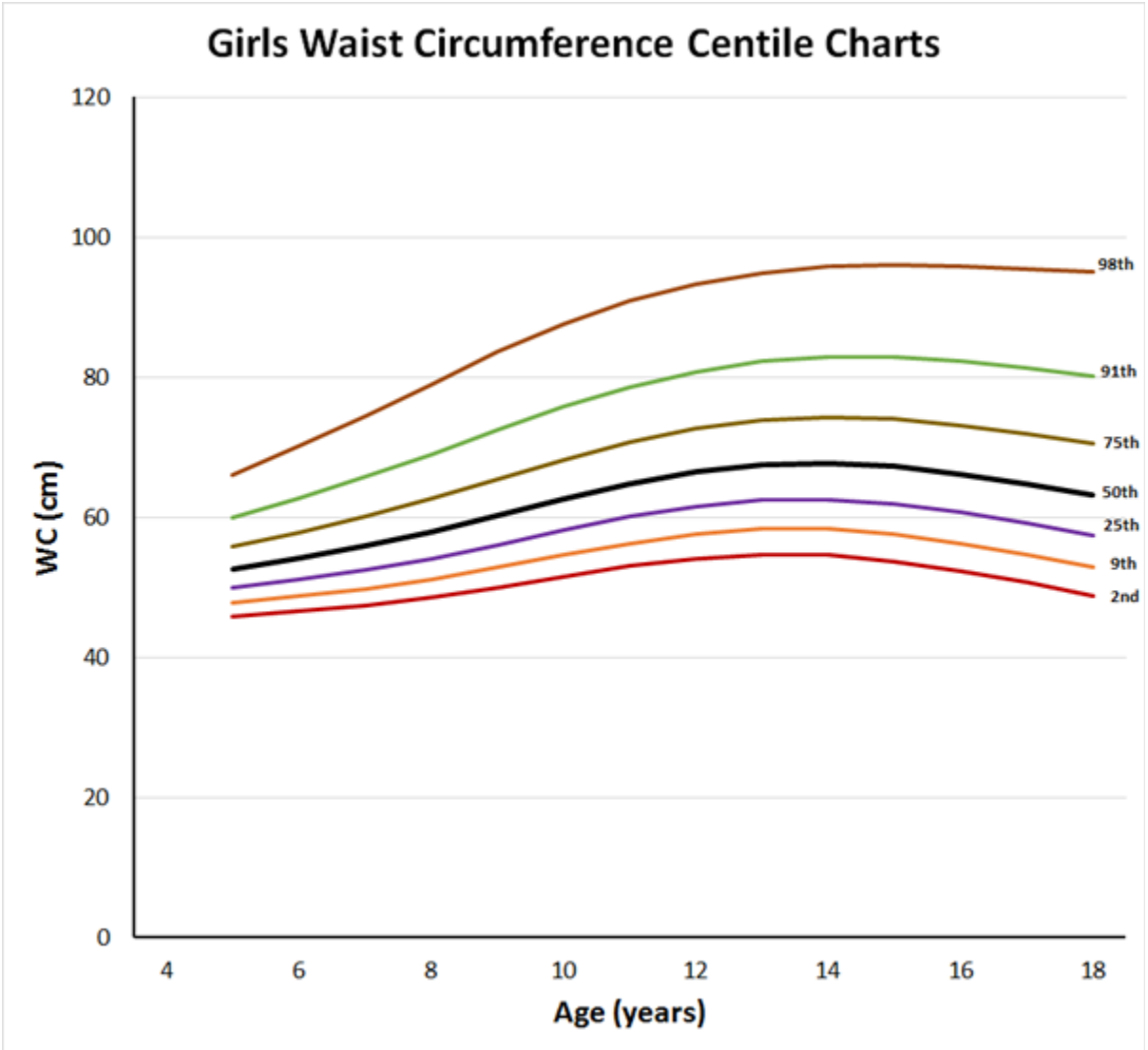

Figure 9(b). Waist circumference centile charts for African and Caribbean Girls

Table 9(e). Waist circumference centile table for African and Caribbean Boys

Age	WC Percentile	WC (cm) Percentile of Height			
		50th	75th	90th	95th
	50th	51.9000	52.8000	53.4000	53.4000
	90th	57.0000	58.4000	60.1000	59.8000
	95th	58.1000	60.1000	62.0000	61.8000
5	99th	58.1000	60.1000	62.0000	61.8000
	50th	51.7000	53.6000	54.0000	54.0000
	90th	58.4000	60.6000	60.6000	60.7000
	95th	61.6000	61.8000	62.2000	63.3000
6	99th	61.6000	61.8000	62.2000	63.3000
	50th	54.5000	55.0000	55.7000	55.7000
	90th	58.8000	60.9000	64.8000	67.3000
	95th	60.4000	67.0000	69.2000	71.9000
7	99th	60.4000	67.0000	69.2000	71.9000
	50th	55.4000	57.3000	57.9000	57.9000
	90th	62.8000	64.4000	66.3000	67.0000
	95th	63.2000	68.3000	72.9000	72.6000
8	99th	63.2000	68.3000	72.9000	72.6000
	50th	59.0000	60.1000	60.3000	60.3000
	90th	67.7000	70.0000	74.3000	75.2000
	95th	77.5000	75.6000	77.0000	78.1000
9	99th	77.5000	75.6000	77.0000	78.1000
	50th	60.0000	61.0000	61.9000	62.0000
	90th	69.2000	76.2000	75.8000	76.8000
	95th	72.6000	82.6000	83.3000	84.0000
10	99th	72.6000	82.6000	83.3000	84.0000
	50th	60.6000	61.3000	62.0000	62.3000
	90th	66.0000	73.9000	75.1000	86.0000
	95th	92.4000	92.5000	94.7000	94.6000
11	99th	92.4000	92.5000	94.7000	94.6000
	50th	65.8000	65.5000	65.4000	65.4000
	90th	76.2000	78.9000	79.9000	79.2000
	95th	82.5000	89.6000	83.3000	83.0000
12	99th	82.5000	89.6000	83.3000	83.0000
	50th	68.2000	69.1000	68.0000	68.0000
	90th	97.6000	90.6000	89.6000	89.3000
	95th	97.6000	101.5000	99.3000	98.7000
13	99th	97.6000	101.5000	99.3000	98.7000
	50th	68.0000	69.1000	69.7000	69.7000
	90th	68.0000	90.5000	87.3000	87.3000
	95th	68.0000	90.5000	87.3000	87.3000
14	99th	68.0000	90.5000	87.3000	87.3000
15	50th	72.4000	72.4000	70.0000	70.7000

	90th	72.4000	72.4000	70.0000	82.6000
	95th	72.4000	72.4000	70.0000	82.6000
	99th	72.4000	72.4000	70.0000	82.6000
	50th	68.7000	71.3000	71.4000	71.4000
	90th	68.7000	71.3000	71.4000	71.4000
	95th	68.7000	71.3000	71.4000	71.4000
16	99th	68.7000	71.3000	71.4000	71.4000

Table 9(f). Waist circumference centile table for African and Caribbean Girls

		WC (cm)			
		Percentile of Height			
Age	WC Percentile	50th	75th	90th	95th
	50th	51.4000	51.8000	52.2000	52.4000
	90th	60.3000	58.4000	58.1000	58.3000
	95th	65.8000	64.7000	63.3000	64.3000
5	99th	65.8000	64.7000	63.3000	64.3000
	50th	54.5000	54.7000	55.0000	54.9000
	90th	59.9000	60.1000	64.0000	64.1000
	95th	64.1000	64.2000	64.7000	65.3000
6	99th	64.1000	64.2000	64.7000	65.3000
	50th	54.5000	55.1000	56.7000	57.5000
	90th	65.7000	66.1000	66.7000	68.9000
	95th	72.9000	70.8000	71.3000	72.6000
7	99th	72.9000	70.8000	71.3000	72.6000
	50th	54.3000	55.4000	56.4000	56.4000
	90th	63.2000	66.5000	71.6000	71.7000
	95th	68.5000	72.1000	75.8000	75.1000
8	99th	68.5000	72.1000	75.8000	75.1000
	50th	59.0000	60.4000	60.6000	62.0000
	90th	74.1000	78.3000	78.5000	78.6000
	95th	80.8000	83.0000	82.4000	83.4000
9	99th	80.8000	83.0000	82.4000	83.4000
	50th	60.4000	62.7000	64.5000	64.4000
	90th	72.4000	76.3000	77.1000	77.7000
	95th	79.8000	81.9000	85.7000	88.3000
10	99th	79.8000	81.9000	85.7000	88.3000
	50th	62.2000	62.3000	63.6000	64.0000
	90th	75.7000	74.6000	75.4000	74.7000
	95th	83.6000	79.6000	80.8000	79.7000
11	99th	83.6000	79.6000	80.8000	79.7000
	50th	65.7000	66.8000	66.6000	67.7000
	90th	83.8000	84.0000	84.3000	84.2000
	95th	93.5000	91.1000	88.3000	86.9000
12	99th	93.5000	91.1000	88.3000	86.9000

	50th	70.1000	70.1000	70.1000	70.5000
	90th	76.6000	77.1000	78.3000	78.2000
	95th	76.6000	79.1000	81.3000	81.1000
13	99th	76.6000	79.1000	81.3000	81.1000
	50th	71.1000	71.3000	71.3000	71.3000
	90th	71.1000	85.9000	84.2000	84.2000
	95th	71.1000	85.9000	84.2000	84.2000
14	99th	71.1000	85.9000	84.2000	84.2000
	50th	67.3000	66.9000	67.8000	67.8000
	90th	67.3000	66.9000	74.5000	74.5000
	95th	67.3000	66.9000	74.5000	74.5000
15	99th	67.3000	66.9000	74.5000	74.5000
	50th	64.8000	64.8000	64.8000	64.8000
	90th	64.8000	64.8000	64.8000	64.8000
	95th	64.8000	64.8000	64.8000	64.8000
16	99th	64.8000	64.8000	64.8000	64.8000

9.5 Discussion

In this chapter, waist circumference percentile charts and tables have been developed for African and Caribbean children living in the UK.

Until recently, it had been assumed that intra-abdominal or visceral fat accumulation (a major risk factor for obesity related ill-health) was a phenomenon only in adults but not children, with the result that visceral fat measurement had been ignored in children. The situation was hampered by the fact that the technology (such as computerised tomography) for quantifying visceral fat was considered unsafe for children because of radiation risk. Consequently, the metabolic risk of intra-abdominal adipose tissue or fat accumulation in children was under appreciated for a long time (McCarthy, 2006). In 1992 and 1993, the first studies conducted in children to examine abdominal subcutaneous and intra-abdominal fat distribution using nuclear magnetic resonance imaging, by two groups of researchers, De Ridder et al and Fox et al respectively, revealed the presence of intra-abdominal fat in children and that its volume was highly

variable between individuals within and between ages as well as across varying levels of fatness. In addition, some of the children were found to have visceral fat values that were associated with higher health risks than in obese adults (De Ridder et al, 1992; Fox et al, 1993).

These measurements had been collected at a time when in the UK, the child obesity epidemic had not really become established. Follow up studies in 2000 and 2008 conducted by Fox et al and Benfield et al respectively, when the childhood obesity had become established, revealed higher amounts of visceral fat in obese compared with healthy boys and girls aged 13years (Fox et al, 2000; Benfield et al, 2008). Although subcutaneous abdominal fat had accounted for only 10% of total abdominal fat, it should be noted however, that the studies relating WC to components of the metabolic syndrome in children suggest that it is total abdominal fat rather than intra-abdominal fat which is related to risk and hence support the use of waist circumference to assess metabolic risk in children.

According to studies done by Misra et al and Zhu et al, ethnic differences in waist circumference have been shown among adult populations. Misra et al proved from their studies that, waist circumference levels for the assessment of abdominal obesity should not be uniformly applied to all populations and ethnic groups (Misra et al, 2005). Zhu et al further stress the importance of race and ethnic specific waist circumference cut-offs in identifying individuals at risk of cardiovascular disease (Zhu et al, 2005). Unfortunately, such studies are lacking in the paediatric age group, although reference data for waist circumference are available from different countries (Sung et al, 2006). In view of the fact that waist circumference is a surrogate measure of intra-abdominal

adiposity and a sensitive marker of cardiovascular diseases, ethnic specific waist circumference cut-offs should be ascertained for effective monitoring of obesity.

These are the first waist circumference percentile curves developed specifically for African and Caribbean children living in the UK. Earlier work by McCarthy et al in 2001 established waist circumference percentile curves for predominantly Caucasian children aged five to sixteen years (McCarthy et al, 2001) with findings similar to those above. Although, WC generally increased with age in both groups, the black children were found to have higher mean WC levels compared to the Caucasian children. A recent study has proved that onset of puberty is influenced by ethnicity and black children begin puberty before white children. Furthermore, black children tend to have more subcutaneous and visceral fat and hence higher WC compared to Caucasian children (Staiano et al, 2013).These charts and additional waist circumference tables produced in this study provide defined WC mean values for various age groups and gender for African and Caribbean children living in the UK. These charts should now be evaluated in both clinical and epidemiological contexts.

As waist circumference is a surrogate measure of abdominal adiposity and obesity, it has become the simplest anthropometric tool for identifying at-risk obese children who are likely to be predisposed to obesity ill-health. In a US study, diabetes mellitus prevalence among adolescents increased tenfold between 1982 and 1994 with all the cases occurring in obese individuals (Pinhas-Hamiel et al, 1996). A review of factors associated with childhood obesity revealed factors including parental obesity, early maturation and **low socio-economic status** (Parsons et al, 1999). Children from African and Caribbean background living in the UK form part of the ethnic minority group

challenged economically and hence are at a higher risk of becoming obese. Consequently, such children should require regular follow-ups for obesity monitoring.

There has been ample evidence to indicate that waist circumference measurement can provide vital information in children in relation to measurement of abdominal and visceral fatness which reflect their risk for acquiring obesity associated diseases (Saxena et al, 2004; Goran et al, 1997; He et al, 2002). Hence these WC charts and tables for children of black descent would assist both clinicians and epidemiologists to monitor WC levels for disease prevention and management.

Following the initial WC centile charts published for the UK child and youth population in 2001 (McCarthy et al, 2001), a number of WC charts have been developed for a number of population groups around the world including USA, Canada, Australia,, China (Jose et al, 2004). However it is conspicuous to note that, to date, there is an absence of equivalent charts for any Sub-Saharan country or for the smaller Caribbean Island populations (McCarthy, 2014). Hence there is an urgent need for these countries to produce equivalent WC charts. Until this is completed, it is impossible to make any meaningful cross-cultural comparisons for children of black descent.

Study Limitation

The sample size obtained for drawing the percentile charts was appreciable. However, the number of children in the lower age group (example five and six years) was less compared to the numbers for the older children.

Chapter 10: General discussion, study limitations, future work and conclusion

10.1 General Discussion

In this study, causes, effects and the impact of obesity in the paediatric age group have been presented together with the development of a range of monitoring percentile charts and tables for tracking body composition parameters among African-Caribbean children. Hence, this thesis has produced percentile charts of selected body composition measures including per cent fat mass, fat free mass, skeletal muscle mass, waist circumference and blood pressure which are improved measures of overweight and obesity and/or metabolic disease risk compared with BMI. In chapters three and four, corrected fat mass and skeletal muscle mass BIA equations have been produced for African and Caribbean boys and girls. The gender differences in these equations could be due to the developmental differences in boys and girls during growth. These equations have helped to produce body composition percentile charts which can be used specifically to assess black children in paediatric clinics and also for epidemiological purposes. The fat mass and fat free mass charts produced in this study specifically for black children could perform as excellent monitoring tools for obesity in black children compared to BMI and general paediatric weight monitoring tools. The various measures of skeletal muscle mass such as absolute SMMa (kg) (appendicular skeletal muscle mass in kilograms) and per cent SMM have been derived by this study. Sarcopenia, which is the age related decline of skeletal muscle mass strength and function, has been found as a predisposing factor for metabolic diseases. Sarcopenia appears to have its origins in early childhood and can track into adulthood with potentially serious consequences (Jensen et al, 2007; Aihie and Cooper, 2007). These measures of skeletal muscle mass would aid effective monitoring of sarcopenia as well

as metabolic disease risk by clinicians and epidemiologists. Similarly, the blood pressure as well as the waist circumference percentile charts and tables should be used in paediatric settings for the assessment of children for easy and effective monitoring of BP and abdominal fatness (waist circumference).

The trend of childhood overweight and obesity continues to rise resulting in the escalation of metabolic ill-health among children. Consequently, this is reflected in national and global monitoring of childhood obesity in health surveys such as the HSE, NDNS and NCMP. These cross-sectional surveys have brought to light the unevenness of prevalence rates of overweight and obesity and its related morbidity across the UK population.

Obesity prevalence among children of black descent is higher compared to Caucasians due to a range of factors that are socio-cultural, dietary and economic as well as the fact that they form part of ethnic minority groups. Ethnic differences in cardiovascular disease risk have been reported with increased risk and deaths observed in people of ethnic minority living in the UK such as Africans and Caribbean (Nish, 2003). These risk factors which include high triglycerides levels, increased insulin resistance and higher blood pressure levels, all of which are directly or indirectly linked to overweight and obesity, have been found even in children contributing to an increased risk of suffering from CVD in later life (Whincup et al, 2002). Reports also show that the prevalence of type 2 diabetes, predisposed by overweight and obesity, is higher in South Asian and black children all from ethnic minority background (Haines et al, 2007; Ehtisham et al, 2005).

To date, body mass index (BMI) has been the most common measure to rank excess weight in the assessment of overweight and obesity. Although BMI correlates with adiposity, it does not adequately describe ethnic variability in terms of overweight and obesity. For a given BMI, people of South Asian origin have been found to have higher body fat as well as insulin resistance compared with white Europeans (Dudeja et al, 2001; Deurenberg et al, 2002; Ehtisham et al, 2005). Even in children and adolescents, it has been shown that for the same age and sex, a child can have a twofold increase of fat mass for the same BMI (Wells, 2000). In fact unlike Caucasian children, children of black descent, have lower average fat mass at similar BMI levels compared to Asian children (Deurenberg et al, 1998). Furthermore, BMI does not provide information on relative proportions of fat and lean mass in an individual. However, for the same weight and/or BMI, increases of fat mass as well as its distribution within the body and a decrease of lean mass is linked to a high risk of developing cardiovascular diseases and type 2 diabetes (Barker, 2005). Body weight and BMI do not reflect either body composition or fat distribution and as such the use of BMI tables, despite their easy accessibility and simplicity as a main measure of overweight and obesity, is not entirely acceptable.

10.2 Limitations of the study

Recruitment of children took almost one year. The main challenge was the fact that children have to be brought to the laboratory with their parents for their measurements to be taken. The reason for this is that the main validation equipment, the DXA, could not be moved away from the lab due to its size and other inherent factors. Consequently, although a lot more children were recruited, only a few who were willing to travel to the lab and as such the sample size was small.

Secondly, the fears of some parents could not be allayed. Some of the parents were not sure of the effect of the radiation emitted by the DXA although several health education sessions on the subject were held to thoroughly discuss and explain these issues. They were concerned that this might cause permanent damage to their growing children and therefore refused to participate. At the same time, it was important to balance benefit (science and healthcare) with risk (to the individual) and this also reflected on the sample size.

Recruitment of the participants also required funds which were limited. In most instances, the primary researcher had to visit churches across London with the consequent transportation cost. Furthermore, bringing more children to the lab incurred cost of transportation and provision of meals and entertainment activities as the measurements usually took a whole day and hence, the smaller sample size. However, this should not have affected the validity of the study. Moreover, measurement of total body water would have been better but, the cost of D_2O and mass spectrometry were prohibitive to this study, although this would have allowed a 4-C model of body composition to be conducted, thus improving the predictability of the regression equations in the validation study.

Despite the production of these novel assessment tools, there are still disadvantages to the use of percentile charts and the use of BIA in clinical and epidemiological assessment. Firstly, due to the continual growth of children and youths between birth and early adulthood, around age 18 years, body size, dimensions and composition are continually changing and all percentile charts have to be age-dependent. These contrast with adult assessment tools such as BMI and WC with which fixed cut-offs can be used

to define excess adiposity. Thus definitions of excess adiposity or other measures of body composition or metabolic risk are statistically based. Although not an element of this thesis, the measure of abdominal fatness, namely the waist-to-height ration (WHtR) has been shown to be age, gender and ethnicity independent; thus percentile charts are not required for this measure. For BIA, this technology is still not available in all clinical settings and the cost of the equipment will always be an issue given limited and declining healthcare budgets. Equally, there remains a reluctance to accept the merits of the technology by healthcare decision makers (National Institute for Heath & Care Excellence, NICE), despite the wealth of scientific research and validation of the technology. At the same time there remains fervent support for the use of BMI, despite the wealth of research on the poor validity of the measure. There remains a challenge to guide healthcare decision makers into embracing newer and improved assessment tools in paediatric practice.

10.3 Future Work

More work needs to be conducted in the area of body composition of minority ethnic children. People of black descent in the UK make up a significant proportion of the minority population and bear a disproportionate level of ill-health, particularly that related to obesity and metabolic diseases. More insight needs to be gained into the early origins of disease risk in this group and how the genetic and environmental determinants are reflected in their phenotype, which is the makeup of their body composition. Newer proxy measures of body composition should make the identification process more accurate and more precise. Systems such as BIA are at the forefront of these newer measures and beyond BIA, circumferences and ratios (for example: waist-to-height ratio and thigh-to-waist ratio), although outside the scope of this thesis, may also prove beneficial in the future. Children are the future adults in

society and their future wellbeing and health is paramount. With the ever-increasing prevalence of overweight and obesity, there is no greater urgency than to tackle this issue now.

10.4 Conclusion

The prevalent rates of metabolic diseases are rising each day. However, the management of these conditions is still limited. Early identification, tracking from childhood and prevention are the best solutions. Hence, more research work should concentrate on not only identifying the causes and predisposing factors but also the development of tools such as those produced by this study for easy monitoring and tracking of body parameters that directly or indirectly identifies individuals at risk of acquiring metabolic ill-health.

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Appendix

Appendix A: QUESTIONNAIRE

QUESTIONNAIRE

BIODATA:

I.D No: Child's Name:

Date: Gender: Age (at last birth day):

MEDICAL HISTORY OF PUPIL:

Have you ever been informed by a health professional (doctor, nurse etc) that you have any of the following diseases:

(1) High Blood Pressure (BP) YES NO

(2) Diabetes Mellitus YES NO

(3) On Medications YES NO

FAMILY HISTORY:

Are you aware that a close relative (father, mother, brother, sister, grandfather, grandmother) has any the following diseases:

(1) High Blood Pressure (BP) YES NO

(2) Diabetes Mellitus YES NO

MEASUREMENTS

BLOOD PRESSURE:

Date: Rest Period: 10min

SBP1	DBP1	Heart Rate1	2mins
SBP2	DBP2	Heart Rate2	2mins
SBP3	DBP3	Heart Rate3	2mins

Average:

Weight (kg): Height (cm):

Waist Circumference (cm):

BODY COMPOSITION MEASUREMENTS:

Bio-impedance Analysis:

Bod Pod:

DEXA:

DETERMINING THE HEALTH STATUS OF YOUR CHILD.

QUESTIONNAIRE (To be completed by Parent)

Biodata of child

Name of ward/child _____

Health Details about Your Child:

1) Are you aware that your child has hypertension (High BP)? ☐Yes ☐No don't know ☐

If yes, is he/she on treatment? Yes ☐ No ☐ don't know ☐

2) Are you aware that your child has Diabetes Mellitus?

If yes, is he/she on treatment? Yes ☐ No ☐ don't know ☐

Appendix B: INFORMED CONSENT FORM

INFORMED CONSENT FORM

Dear Parent/Guardian/Carer,

Your child has been invited to take part in a research to determine body composition and blood pressure of children aged 5 to18years.

The study involves taking measurements like weight, waist circumference, height, blood pressure and per cent body fat at our science laboratory.

We would be very grateful if you could indicate below that you would like your child to participate.

I would/would not like my child to take part in the above research.

Name of child:

Signature of parent/guardian/carer:

Appendix C: Research Participation Letter

Institute for Health Research & Policy

London Metropolitan University,

166 – 220 Holloway Road,

London,

N7 8DB

Dear Sir/Madam,

We are writing to ask for your help. As you will know, individuals living in the UK originating from sub-Sahara Africa and the Caribbean have a greater risk for conditions such as stroke, diabetes and heart disease. These conditions are greatly linked to being overweight or obese but are preventable and treatable when detected early in life. High blood pressure is known to be common and difficult to treat among people of black descent.

We are a university-based health research group starting a project to develop national references for body fat in African and Caribbean children aged 5–18 years. It is against this background that this research aims to solely to look at body fat and blood pressure measurements of African and Caribbean children. The study would give rise to early detection measures of obesity and high blood pressure to curb their rising trend and to improve the health status of the entire community.

We would greatly appreciate your assistance, as we would like to come to your church and ask children to volunteer to take part in this survey. We would need children aged from 5 to 18 years to visit us at our clinic and laboratory in groups of 5 to 10 to measure their body fat and blood pressure. This would involve state of the art technology only available in research institutions. Dr (Mrs) Eva Amoako-Attah who is a Ghanaian physician at the Korle–Bu Teaching Hospital would perform the measurements.

We do hope that you would be willing and able to assist us. We could discuss this proposal further over the telephone or arrange a time to meet with you. At this stage we can assure you that all measurements would be kept strictly confidential. Ethical approval for the research has been obtained from our university ethics panel and we have enhanced CRB clearance.

At the end of the survey, we would gladly visit your school to offer you feedback as well as guidelines on prevention of obesity and its associated diseases.

We look forward to hearing from you and thank you for your co-operation.

Yours faithfully,

……

(Dr.) David McCarthy RNutr

Professor of Nutrition & Health

……

(Dr.) Mrs Eva Amoako-Attah MBChB, BSc, MPH

Researcher

Appendix D: Standard Deviation Scores of Anthropometric Measurements

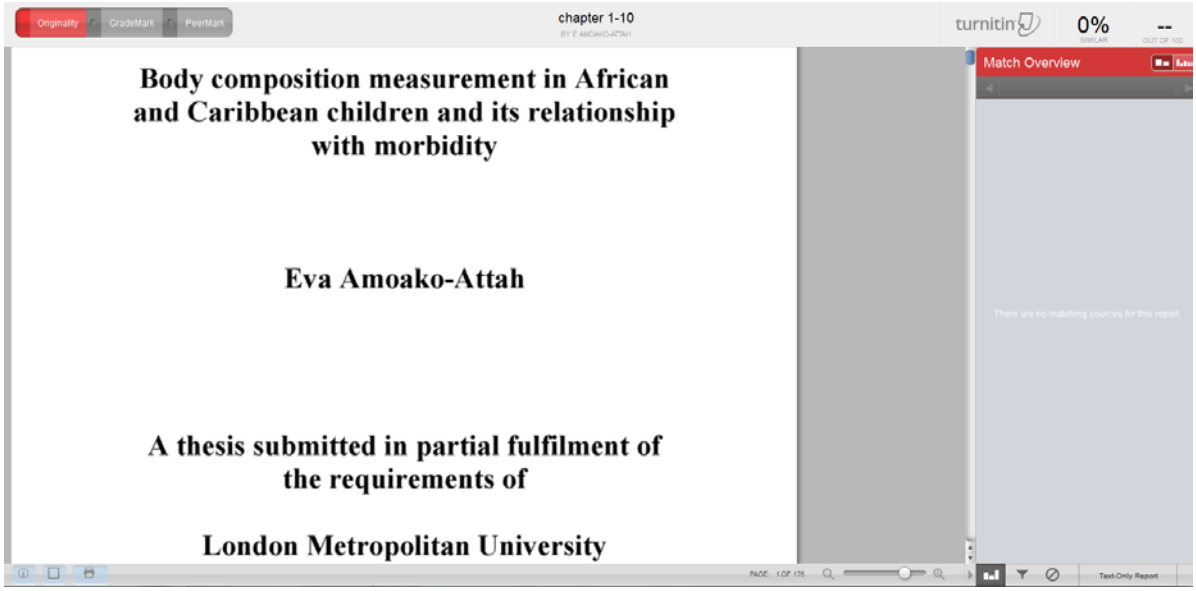
Male:

Dec age (y)	Height (cm)	z-Height	Height (m)	Weight measured (kg)	z-Weight	BMI	z-BMI	WC	SDS_Waist
4.871	103.5	-1.16	1.04	17.4	-0.45	16.24	0.53	51.0	-0.18
5.665	118.5	0.97	1.19	34.4	3.64	24.50	4.00	61.4	2.24
6.012	116.8	0.16	1.17	18.0	-1.22	13.16	-2.25	55.0	0.71
6.070	137.0	4.23	1.37	23.2	0.77	12.33	-3.31	54.0	0.43
6.461	125.5	1.36	1.26	22.6	0.28	14.35	-0.97	51.0	-0.54
6.850	133.5	2.45	1.34	27.1	1.24	15.21	-0.25	55.6	0.62
6.930	125.5	0.78	1.26	25.5	0.77	16.19	0.43	56.4	0.79
7.951	137.0	1.73	1.37	28.2	0.66	15.02	-0.50	54.5	-0.01
8.170	138.0	1.66	1.38	32.6	1.36	17.12	0.76	62.5	1.56
8.219	134.0	0.88	1.34	26.0	-0.06	14.48	-0.96	54.0	-0.23
8.638	133.0	0.28	1.33	39.2	1.96	22.16	2.43	69.0	2.29
9.687	147.0	1.69	1.47	37.0	1.10	17.12	0.44	58.2	0.20
9.736	147.0	1.64	1.47	34.3	0.68	15.87	-0.26	59.1	0.37
10.486	152.0	1.73	1.52	39.3	0.97	17.01	0.19	60.4	0.33
10.678	137.8	-0.62	1.38	35.3	0.30	18.59	0.86	60.8	0.33
11.559	154.0	1.15	1.54	59.3	2.24	25.00	2.39	78.0	2.12
12.359	175.0	3.31	1.75	57.0	1.77	18.62	0.45	66.6	0.65
12.556	169.9	2.41	1.70	64.0	2.16	22.17	1.58	75.4	1.62
14.656	167.2	0.04	1.67	51.2	-0.22	18.31	-0.37	69.4	0.23
15.655	172.0	-0.01	1.72	69.0	0.91	23.32	1.25	74.0	0.54
16.559	168.0	-0.95	1.68	61.7	-0.12	21.86	0.61	73.2	0.13
17.331	184.9	1.19	1.85	71.9	0.66	21.03	0.13	71.3	
18.333	175.0	-0.32	1.75	65.8	-0.18	21.49	0.11	74.0	
19.792	170.0	-1.05	1.70	67.5	-0.21	23.36	0.53	72.4	
		0.98			0.79		0.33		0.68

Female:

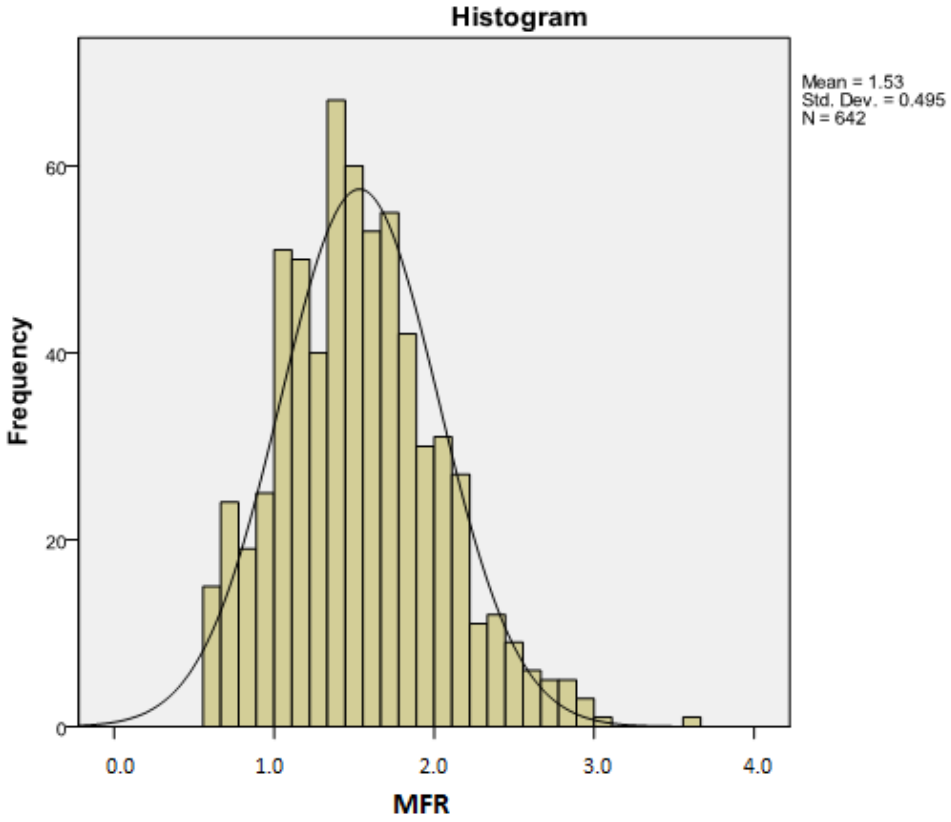
Dec age (y)	Height (cm)	z-Height	Height (m)	Weight measured (kg)	z-Weight	BMI	z-BMI	WC	SDS_Waist
5.897	113.0	-0.35	1.13	20.3	0.02	15.90	0.27	55.0	0.88
6.360	123.0	1.11	1.23	24.7	0.96	16.33	0.47	54.0	0.51
7.255	135.1	2.34	1.35	27.1	0.82	14.85	-0.56	59.2	1.31
7.291	134.0	2.08	1.34	29.4	1.25	16.37	0.34	61.0	1.60
8.920	130.4	-0.34	1.30	26.5	-0.46	15.58	-0.42	58.3	0.64
10.237	150.5	1.65	1.51	34.9	0.27	15.41	-0.85	66.1	1.55
10.275	157.6	2.70	1.58	47.1	1.68	18.96	0.78	72.3	2.24
10.472	148.0	1.04	1.48	39.1	0.71	17.85	0.30	66.0	1.48
10.834	150.9	1.13	1.51	42.8	0.95	18.80	0.58	67.4	1.57
11.261	142.0	-0.51	1.42	29.5	-1.28	14.63	-1.65	58.0	-0.15
11.806	147.0	-0.23	1.47	51.8	1.35	23.97	1.86	74.0	2.12
11.811	147.6	-0.15	1.48	44.3	0.60	20.33	0.88	70.3	1.70
12.857	165.0	1.50	1.65	61.7	1.74	22.66	1.33	82.5	2.77
13.142	167.0	1.60	1.67	53.5	0.84	19.18	0.13	74.0	1.89
13.678	163.0	0.69	1.63	60.0	1.23	22.58	1.14	82.3	2.70
13.777	161.3	0.38	1.61	52.2	0.37	20.06	0.31	71.4	1.44
14.007	152.0	-1.17	1.52	53.3	0.38	23.07	1.21	76.0	2.00
14.672	165.8	0.68	1.66	52.9	0.05	19.24	-0.22	72.9	1.52
17.607	160.5	-0.50	1.61	59.2	0.23	22.98	0.65	76.5	
18.817	164.4	0.13	1.64	60.0	0.25	22.20	0.27	74.8	
		0.69			0.60		0.34		1.54

Appendix E: Originality Report



Appendix F: MFR For boys and girls

MFR for Boys (5 – 16 years)



MFR for girls (5-16years)