Comparison of waist circumference distribution of South African black children from different study populations

BOITUMELO STOKIE MOTSWAGOLE

M.Sc in International Nutrition

Student number: 21451648

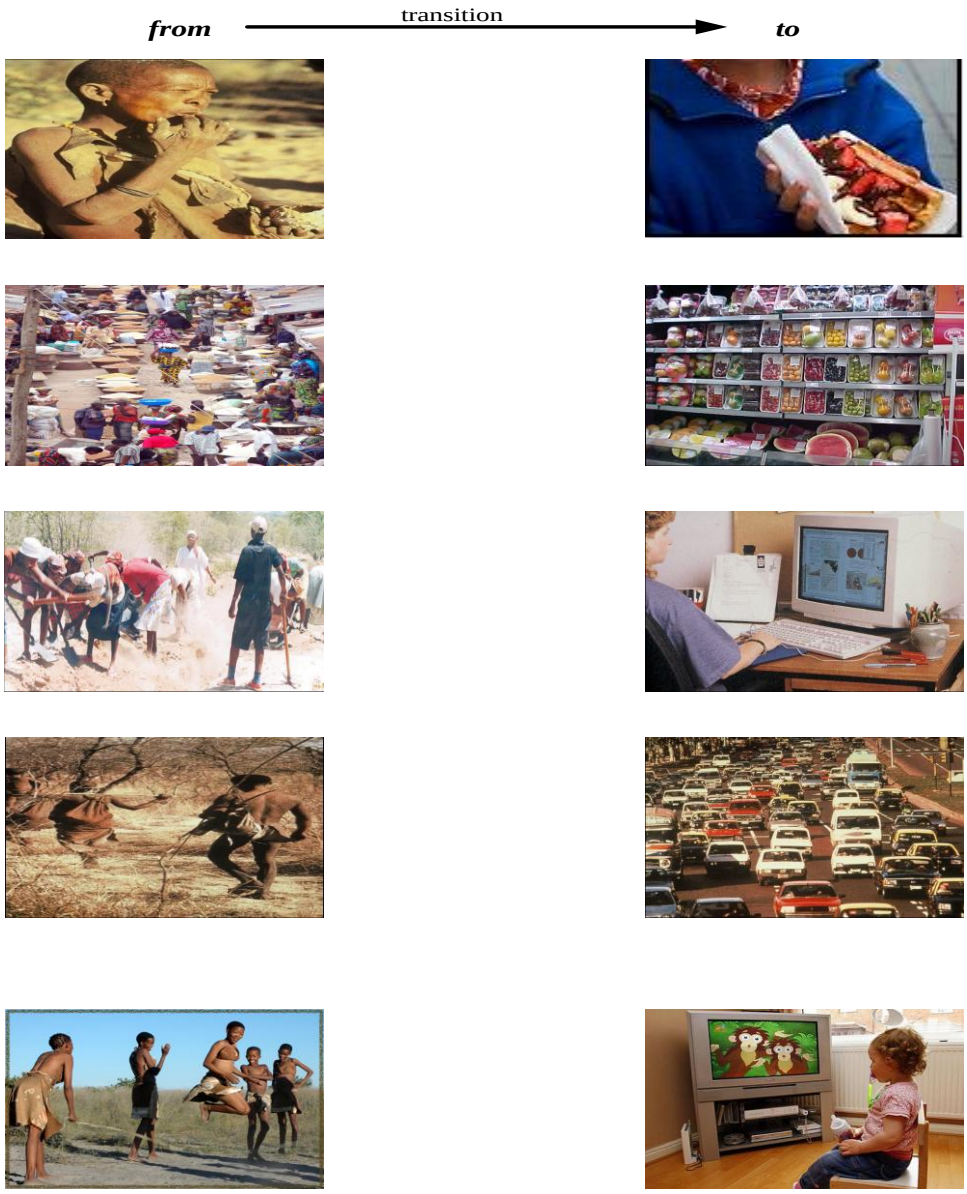
Thesis submitted for the degree Doctor of Philosophy (PhD) in Nutrition at the Centre of Excellence for Nutrition, North-West University, (Potchefstroom Campus)

Promoter: Professor H. Salome Kruger

Co-promoter: Dr. Mieke Faber

2010

NORTH-WEST UNIVERSITY
YUNIBESITI YA BOKONE-BOPHIRIMA
NOORDWES-UNIVERSITEIT



The deleterious effects of obesity on chronic disease risk, morbidity, and mortality; its high medical, psychological, and social costs; its multiplicity of causes; its persistence from childhood into adulthood; the paucity of successful treatment options; the hazards of pharmacologic treatments; and the complexities of treatment guidelines all argue for increased attention to the *prevention* of excessive weight gain starting as early in life as possible. Prevention, however, requires changes in individual behavioural patterns as well as eliminating environmental barriers to healthy food choices and active lifestyles—**both exceedingly difficult to achieve**.

Nestle, 2000.

For my beloved husband and children who kept me going when I couldn't.

-Stokie

ACKNOWLEDGEMENTS

I would like to thank all the following people who have helped and inspired me during my doctoral study.

- It is difficult to overstate my gratitude to my Ph.D. supervisor, Salome Kruger whose patience and kindness, as well as her academic experience, have been invaluable to me. Her wide knowledge and logical way of thinking have been of great value for me. Her understanding, encouragement and personal guidance have provided a good basis for the present thesis.

- I would like particularly to acknowledge the contribution of Mieke Faber, my co-supervisor whose encouragement, guidance and support from the initial to the final level enabled me to develop an understanding of the subject.

- Special thanks to Prof. Faans Steyn for assisting with statistical analysis. He was always accessible and willing to help. As a result, data analysis and research life became smooth and rewarding for me.

- Collective and individual acknowledgments are also owed to my colleagues at the North-West University (Potchefstroom) for creating such a great friendship at the office and many places in between. It is my pleasure to mention Noloyiso Matiwane, Lesly Mamabolo, Namukolo Covic, Pedro Pisa, Cornelie Nienaber, Sarah Matenge and Wayne Towers who would pop in once in a while to share some experiences and jokes to release the pressure.

- I also wish to thank Prof. Lesley Greyvenstein for the language editing of my manuscript.

- My mum, Patience Semfhutsen Pheko, has been a constant source of support, care and love. As a typical single mother, she worked hard to support the family and spared no effort to provide the best possible environment for me (and other siblings) to grow up and attend school. She never complained in spite of all the hardships in her life. Mother, I love you.

- I feel proud of my sister and her family for their loving support, emotional, moral and of course financial – during my postgraduate years, and this thesis would certainly not have existed without them.

- I would regret my doctoral years at the North-West University (Potchefstroom) if I did not join the Christian Fellowship Church. Joining CFC was not only a turning point in my life, but also a wonderful experience. I cherished the prayers and support between me and them, and the friendships with my Christian brothers and sisters. I treasure all precious moments we shared and would really like to thank them, especially Rod and Fiona Hunter who accommodated me and the children every Wednesday evening for prayer meetings. God bless you guys!! You were like my spiritual father and mother who drew me close to the Lord.

- I owe my loving thanks to my children Refilwe, Abale and Puso. They let me own a happy family in South Africa. We used to share some special moments at the house and it helped me in dealing with the academic pressure. My eldest daughter Wame, you were very supportive all the time, thank you.

- This thesis is dedicated to my beloved husband Meshack Bushie Motswagole who has been, always, my pillar, my joy and my guiding light, and I thank him. I remember his constant support when I encountered difficulties and I appreciate the very special person he is. I owe him for unselfishly letting his intelligence, passions, and ambitions collide with mine.

- Last but not least, thanks be to God for my life through all my work in the past three years. You have made my life more bountiful. May Your Name be exalted, honored, and glorified.

- In conclusion, I recognize that this research would not have been possible without the financial assistance of the Government of Botswana through the Ministry of Communications Science and Technology.

SUMMARY

Studies in both children and adults indicate that waist circumference (WC), a measure of abdominal obesity is closely related to cardiovascular risk factors. The accurate identification of abdominally obese children in health screening programmes for early intervention is of importance. There are, however, concerns about using international definitions for screening purposes because in most instances these have been derived from Western populations and, therefore, may have limited usefulness to children in other parts of the world. When these cut-off points are used in developing countries, they ignore the fact that the growth patterns of children and burdens of disease vary between countries. Due to lack of population specific cut-off points for children in the developing world it may be tempting and convenient to use the same cut-off points as for children in developed countries, but such a practice runs the risk of exporting failure. Ideally, a screening tool should have both high sensitivity and specificity, and these are important considerations in choosing the definition for the detection of childhood abdominal obesity. High sensitivity is necessary to avoid failure of identifying obese children and high specificity of the screening tool ensures that non-obese children are not misclassified as obese, which may otherwise lead to unnecessary treatment and psychosocial implications of stigmatisation. Failure to identify the abdominally obese child may have more serious consequences than misclassification, since it results in an increase in adult morbidity and mortality. Therefore, the main aim of this thesis was to examine fat distribution patterns of black South African (SA) children in relation to health risk. The specific objectives were to: compare the body composition of black stunted and non-stunted children from two rural communities in South Africa; to describe and compare the age and sex specific WC percentile distribution for black SA children from different study populations and compare the WC percentile distribution with those for African-American (A-A) children and to assess the diagnostic accuracy of waist-to-height ratio (WHtR) as a marker for high blood pressure, a cardiovascular risk factor in SA children.

Findings of this study demonstrated increased total adiposity in non-stunted children, but trends of increased central adiposity, measured as WHtR in stunted children. This warrants further investigation on this relationship among children older than 13 years in

the African context where many children are stunted. The differences observed between the different data sets and between SA and A-A children suggest that nationally representative data should be used to develop age, sex and ethnic specific WC percentiles for this population. The results indicate clearly that the median WC of children from SA studies is smaller than those of A-A children, with a medium to large effect size for the difference. Results also suggest concern with respect to high WC values (> 80 cm) among some children. The recommended universal WHtR cut-off value of 0.5 for assessment of cardiovascular risk is not suitable for black SA children because it had low sensitivity in predicting high blood pressure. The absence of locally developed cut-off values for WC and WHtR for children warrants research due to the associations between being overweight and obese and disease outcomes. It is fundamental to detect risk at an early stage so that appropriate intervention can be initiated timeously.

Keywords: Body composition, abdominal obesity, stunting, waist circumference, children, South Africa.

OPSOMMING

Studies in beide kinders en volwassenes toon aan dat middelomtrek (MO), ’n maatstaf van abdominale obesiteit ‘n sterk verwantskap met kardiovaskulêre risikofaktore toon. Die akkurate identifisering van abdominale obesiteit is van kardinale belang vir vroeë intervensie in gesondheidsprogramme. Daar is kommer oor die gebruik van internasionale definisies vir evalueringsdoeleindes, omdat daar in die meeste gevalle waardes gebruik word wat afgelei is van Westerse populasies en daarom mag die waardes beperkte toepassing hê vir kinders in ander dele van die wêreld. Die gebruik van hierdie afsnywaardes vir ontwikkelende lande ignoreer die feit dat groeipatrone van kinders en die siekte voorkoms tussen lande verskil. As gevolg van ’n gebrek aan populasie spesifieke afsnywaardes vir kinders in ontwikkelende lande mag dit as maklik en gerieflik beskou word om waardes vir ontwikkelde lande te gebruik, maar dit kan ongeldig wees. Dit is ideaal dat ’n evalueringsinstrument beide sensitief en spesifiek moet wees en dat beide belangrike oorwegings is wanneer ’n mens abdominale obesiteit in kinders definieer. Hoë sensitiwiteit is noodsaaklik om te voorkom dat obese kinders verkeerdelik nie geïdentifiseer word nie en spesifisiteit van die evalueringsinstrument is noodsaaklik sodat nie-obese kinders nie geklassifiseer word as obese kinders nie, wat kan lei tot onnodige behandeling en psigososiale implikasies van stigmatisering. Die versuim om obese kinders te identifiseer kan baie meer nadelige gevolge inhou as verkeerdelike klassifikasie, omdat onbehandelde abdominale obesiteit in kinders tot verhoogde volwasse morbiditeit en mortaliteit kan lei. Daarom was die hoofdoel van hierdie proefskrif om vetverspreidingpatrone in swart Suid Afrikaanse (SA) kinders in verhouding tot gesondheidsrisiko te ondersoek.

Die spesifieke doelwitte was om: die liggaamsamestelling van swart kinders met en sonder groeibelemmering van twee plattelandse gemeenskappe in SA te vergelyk;die ouderdom- en geslagspesifieke MO persentielverspreiding van swart SA kinders van verskillende populasiegroepe te bepaal en vergelyk en ook die MO persentielverspreiding van swart SA kinders met dié van Afrika-Amerikaanse (AA) kinders te vergelyk en die diagnostiese akkuraatheid van middel:lengte ratio (MLR) as ‘n merker vir hoë bloeddruk, ‘n kardiovaskulêre risikofaktor in SA kinders te bepaal

Die resultate van hierdie studie dui op groter totale vetsug in kinders van normale lengte, maar ‘n neiging tot groter sentrale vetsug, gemeet as MLR in kinders met groeibelemmering. Verdere navorsing oor hierdie onderwerp in kinders ouer as 13 jaar in Afrika konteks, waar groeibelemmering algemeen is, is nodig. Die verskille wat tussen die MO verspreiding van SA and A-A kinders dui daarop dat nasionaal verteenwoordigende data gebruik moet word om ouderdom-, geslag- en etniesspesifieke MO persentiele vir hierdie populasie op te stel. Die resultate dui op duidelike kleiner mediane MO van SA kinders as A-A kinders, met medium tot groot effekgroottes vir die verskille. Resultate dui ook op kommerwekkende hoë MO waardes (> 80 cm) in sommige groepe SA kinders. Die aanbevole universele MLR afsnywaarde van 0.5 vir bepaling van kardiovaskulêre risiko is nie geskik vir swart SA kinders nie. Hierdie afsnywaarde moet hersien word as gevolg van verskille in MO en lengte van swart SA kinders. Daar is nog geen SA afsnypunte vir MO en MLR vir kinders nie. Verdere navorsing in hierdie veld is dus nodig as gevolg van die verwantskap tussen oorgewig en siektes van lewensstyl. Dit is belangrik om risiko vir hierdie siektes vroegtydig te identifiseer, sodat gepaste intervensies vroegtydig ingestel kan word.

Sleutelwoorde: Liggaamsamestelling, abdominale obesiteit, groei-inkorting, middelomtrek, kinders, Suid Afrika.

TABLE OF CONTENTS

ACKNOWLEDGEMENTS.....i

SUMMARY.....iii

OPSOMMING.....v

TABLE OF CONTENTS.....vii

LIST OF ABBREVIATIONS.....xi

LIST OF TABLES.....xiv

LIST OF FIGURES.....xvi

CHAPTER 1: INTRODUCTION.....1

1.1 Problem statement.....2

1.2 Background.....4

1.3 Aims and objectives.....9

1.4 Hypotheses.....9

1.5 Structure of the thesis.....10

1.6 Author contributions.....11

1.7 References.....14

CHAPTER 2: LITERATURE BACKGROUND ON FAT DISTRUBUTION AND ITS HEALTH IMPLICATIONS.....21

2.1 Overview.....22

2.1.1 Fat storage in adipose tissue.....22

2.1.2 Biological role of adipose tissue.....23

2.2 Body fat distribution.....24

2.2.1 Fat distribution in children.....26

2.3 Nutritional disorders and body fat in children.....27

2.3.1 Undernutrition.....28

- *Undernutrition and fat deposition in children*.....29
- *Effects of body fatness and growth velocity*.....30
- *Effects on energy balance and fat oxidation*.....33

2.3.2 Obesity.....34

- *Theories of obesity development*.....35

2.4 Health effects of excess abdominal fat.....41

2.4.1 Metabolic Syndrome.....42

2.4.2 Diabetes.....51

2.4.3 Cardiac disorders.....52

2.4.4 Hepatic disorders.....54

2.4.5 Cancer.....55

2.4.6 Possible mechanisms that link visceral fat with disease risk.....58

- *Action of free fatty acids*.....58
- *Adipocyte secretion profile*.....62
- *Adiponectin*.....63
- *Leptin*.....65
- *Circulating concentrations of inflammatory cytokines*.....66

2.5 Abdominal fat assessment.....68

2.5.1 Imaging techniques.....69

- *Computed tomography*.....69
- *Magnetic resonance imaging*.....69
- *Ultrasound*.....71
- *Dual-energy x-ray absorptiometry*.....71

2.5.2 Anthropometric indices.....72

- *Waist-hip ratio*.....73
- *Abdominal sagittal diameter*.....73
- *Waist-to-height ratio*.....74
- *Waist circumference*.....76

2.6 Waist circumference percentile charts for children.....78

2.7 Summary.....80

2.8 References.....81

CHAPTER 3: BODY FAT DISTRIBUTION IN STUNTED COMPARED TO NON-STUNTED BLACK SOUTH AFRICAN CHILDREN FROM TWO RURAL COMMUNITIES.....105

- Abstract.....107

- Introduction.....108
- Methods.....109
- Results.....111
- Discussions.....112
- References.....115

CHAPTER 4: COMPARISON OF WAIST CIRCUMFERENCE PERCENTILES OF BLACK SOUTH AFRICAN CHILDREN AGED 10 TO 14 YEARS FROM DIFFERENT STUDY SITES.....123

- Abstract.....124
- Introduction.....125
- Methods.....126
- Results.....131
- Discussions.....140
- References.....143

CHAPTER 5: SENSITIVITY OF WAIST-TO-HEIGHT RATIO IN IDENTIFYING CHILDREN WITH HIGH BLOOD PRESSURE.....149

- Abstract.....151
- Introduction.....152
- Methods.....152
- Results.....155
- Discussions.....161
- References.....164

CHAPTER 6: GENERAL SUMMARY, DISCUSSIONS & CONCLUSIONS……167

- Introduction……168
- Summary, conclusions and recommendations……170
- Novel findings……..172
- Line of thinking……175
- References……176

ADDENDA……..…...180

ADDENDUM 1: Reviewer's comments on the article: Sensitivity of waist-to-height ratio in identifying children with high blood pressure……181

ADDENDUM 2: Letter on the sensitivity of waist-to-height ratio in identifying children with high blood pressure submitted for publication to the Cardiovascular Journal of Africa……..182

ADDENDUM 3: Proposal for funding of new data collected for the other part of the study……...…192

ADDENDUM 4: Sample of letter to school headmasters.....208

ADDENDUM 5: Information to study participant……..210

ADDENDUM 6: Socio demographic questionnaire……213

ADDENDUM 7: Standard operation procedures for anthropometric data collection……..215

Abbreviations

ACh	Acetylcholine
ACo-A	Acetyl coenzyme A
A-A	African-American
AIDS	Acquired immune deficiency syndrome
AMPK	Adinosine monophosphate-activated protein kinase
APN-KO	Adiponectin knockout
ATP	Adinosine triophosphate
AUC	Area under the curve
BMI	Body mass index
CHD	Coronary heart disease
ChREBP	Carbohydrate response element binding protein
COOH	Carboxylic Acid
COX-2	Cyclooxygenase -2
CPT 1	Carnitine palmiloyl transferase
CPY4A10	Cytochrome enzyme
CRP	C-reactive protein
CT	Computed tomography
CV	Cardiovascular
CVD	Cardiovascular disease
DBP	Diastolic blood pressure
DEXA	Dual energy x-ray absorptiometry
EGIR	European group for study of insulin resistance
EST	Ecological systems theory
FA	Fatty acid
FAO	Food and agriculture organization
FFA	Free fatty acid
HDL-C	High density lipoprotein cholesterol
HIV	Human immuno deficiency virus
HOMA	Homeostasis model assessment

IDF	International diabetes federation
IFG	Impaired fasting glucose
IL-6	Interleukin-6
IR	Insulin resistance
LDL-C	Low density lipoprotein cholesterol
LMS	Least mean square
L-PBE	Peroxisomal L-bifunctional enzyme
MCP-1	Monocyte chemoattractant protein-1
MRI	Magnetic resonance imaging
MS	Metabolic syndrome
NAFLD	Non alcoholic fatty liver disease
NASH	Non alcoholic steatohepatosis
NCEP ATP III	National cholesterol education program-Adult treatment panel III
NCHS	National center for health statistics
NFCS	National food consumption survey
NH_2	Amino group
NHANES	National health and nutrition examination survey
NO	Nitrogen oxide
NPV	Negative predictive value
NPY	Neuropeptide Y
OGTT	Oral glucose tolerance test
PAI-1	Plasminogen activator inhibitor-1
PHLA	Postheparin lipolytic activity
PPV	Positive predictive value
ROC	Receiver operator characteristics
Rx	Pharmacologic treatment
SA	South Africa
SBP	Systolic blood pressure
SNS	Symphathetic nervous system
SNP	Sodium nitropusside
SREBP1c	Sterol regulatory element binding protein-1c

SSF	Subscapular skinfold
T2D	Type 2 diabetes
TG	Triglycerides
TNF-α	Tumor necrosis factor-alpha
TSF	Triceps skinfold
UK	United Kingdom
UNICEF	United Nations International Children Education Fund
UO	Urate oxidase
US	Ultrasound
USA	United States of America
VAT	Visceral adipose tissue
VLDL	Very low density lipoprotein
WC	Waist circumference
WHO	World health organization
WHR	Waist hip ratio
WHtR	Waist-to-height ratio

LIST OF TABLES

CHAPTER 2

Table 2.1: Body fat distribution

Table 2.2: Summary of studies on stunting and obesity development

Table 2.3: Summary of studies on Metabolic Syndrome showing different prevalence rates among children

Table 2.4: Different definitions of Metabolic Syndrome

Table 2.5: Consequences for the liver of increased portal fatty acid concentration

CHAPTER 3

Table 1: Summary of methods from the two studies showing similarities and differences

Table 2: Formulae for calculating the various indices

Table 3: Characteristics of children from the two communities

Table 4: Adjusted means(sd) of different body composition measures for stunted and non-stunted children by study and gender

CHAPTER 4

Table 1: Summary of different data sets

Table 2: Proportional distribution of children by gender and study site

Table 3: Mean sd) for anthropometric variables for 10-14 year old children from different study sites

Table 4: Waist circumference percentiles for 10-14 year old children from different study sites

Table 5: Median and inter quartile range for waist circumference of children from different study sites

CHAPTER 5

Table 1: Mean and standard deviation for the characteristics of the children

Table 2: Diagnostic accuracy measures for WHtR as a predictor of blood pressure for boys and girls at 0.41 and 0.5 WC cut-off values

Table 3: Mean and standard deviation for the characteristics of the Japanese and South African children

Table 4: 2x2 tables for boys at 0.41 & 0.5 waist circumference cut-off values

Table 5: 2x2 tables for girls at 0.41 & 0.5 waist circumference cut-off values

Table 6: Diagnostic accuracy measures for WHtR as a predictor of high blood pressure in boys and girls at 0.41 .05 WC cut-off values

Table 7: Age adjusted correlation coefficients between blood pressure and anthropometric variables.

Table 8: Regression analysis of blood pressure (dependent variable) and anthropometric predictors

LIST OF FIGURES

CHAPTER 2

Figure 2.1: Factors secreted by adipose tissue

Figure 2.2: Illustration of pear and apple body fat distribution patterns.

Figure 2.3: Ecological model of predictors of childhood overweight

Figure 2.4: The society–behaviours–biology nexus

Figure 2.5: Current Perspective of the Metabolic Syndrome

Figure 2.6: Illustration of the complex pathways of obesity initiated Metabolic Syndrome

Figure 2.7: Diagrammatic representation of the insulin-cancer hypothesis

Figure 2.8: Visceral adipose tissue and increased Free Fatty Acids to the liver

Figure 2.9: An illustration of overflow hypothesis

Figure 2.10: Approximate contributions of Free Fatty Acids released from the lower and upper body subcutaneous fat depots and from splanchnic tissues

Figure 2.11: Summary of the main effects exerted by the more relevant adipokines at the cardiovascular level.T2DM indicates type 2 diabetes mellitus

Figure 2.12: Cardioprotective actions of adiponectin

Figure 2.13: Hypothetical model for the actions of adiponectin

Figure 2.14: Illustration of Inflammation and cardiovascular disease

Figure 2.15: Magnetic Resonance Imaging (MRI) equipment

Figure 2.16: Typical Magnetic Resonance Image.

Figure 2.17: The Ashwell Shape Chart

CHAPTER 4

Figure 1: Smoothed curves for 5^{th}, 10^{th}, 25^{th}, 50^{th}, 75^{th}, 90^{th} and 95^{th} percentiles for waist circumference of children from different study sites

Figure 2: Comparison of the 50^{th} percentile WC curves (unsmoothed) for 10-14 year old South African & African-American boys

Figure 3: Comparison of the 50^{th} percentile WC curves (unsmoothed) for 10-14 year old South African & African-American girls

Figure 4: Graphical presentation of effect sizes

CHAPTER 6

Figure 1: Summary of research line of thinking

1

INTRODUCTION

"We are up against social and traditional norms that being fat is a sign that you are wealthy, you are successful, you are happy, that your husband can feed you,"
(Krisela Steyn, 2006).

1.0 Introduction

1.1 Problem statement

1.2 Background

1.3 Aim of the study

1.4 Hypotheses

1.5 Structure of the thesis

1.6 Author contributions

1.7 References

1.1 Problem statement

A striking feature of the growth patterns of African children throughout the continent commonly illustrated in many studies reflects the effects of malnutrition and disease that probably masked the underlying growth pattern. Therefore, much of what is currently known of the growth of African children is based on data that are tarnished by the adverse environment endemic to Africa. Several studies published in developed countries have demonstrated the relationship between environmental factors such as nutrition, energy expenditure associated with physical work and the socio-cultural lifestyle and the child's body composition (WHO, 2010). Many of these suggest that remarkable differences in body composition may exist between children when their social background is different. Obese individuals do not only differ in the amount of excess fat stored in their bodies but also in the regional distribution of the fat within the body. It is useful, therefore, to be able to distinguish between those at increased risk as a result of abdominal fat distribution or a more evenly and peripheral fat distribution (WHO, 2010). Waist circumference (WC) is a simple, easily available anthropometric measurement that gives relevant information about fat distribution and reflects the degree of central adiposity in children (Galcheva *et al.*, 2009) and has been used as an indicator of abdominal obesity with high sensitivity and specificity (Moraes *et al.*, 2010). This indicator has been presenting more accurate positive associations with cardiovascular risk factors than other anthropometric indicators.

South Africa was once and is still burdened with undernutrition of the poor but now is also facing a growing rise in overweight and obese people. Urbanization and modernization have caused huge socio-cultural changes, with large-scale relocation of a substantial portion of the rural population from its traditional and mainly agrarian lifestyle into quasi-industrial townships and cities with the evolution of urban slums characterized by the overhauling of social structures, values, and privileges. Two major and direct consequences of this development have been the change in the dietary patterns and lifestyle of many families resulting in an increase in the prevalence of obesity. In a way the culture of urban South Africa is also contributing to the rise in obesity. Being overweight and obese is perceived as a sign of wealth, because money is associated with food consumption in many parts of South Africa (Sibbel, 2005). The paranoid fear of HIV/AIDS is part of the problem as well. It would, therefore, be expected that there be some variability in body composition in children raised in such communities which would be manifested in their growth patterns and their body build.

The classification of overweight and obesity and particularly abdominal obesity cannot be overemphasized due to the associations with deleterious health outcomes (Stigman *et al.*, 2009; Cameron *et al*., 2009; Wigga *et al*., 2010). Despite this to date there has not been the same level of agreement over the cut-off values of abdominal obesity in children and adolescents. Instead there has been global confusion both in terms of a globally applicable reference population and of the selection of appropriate cut-off values for designating a child as abdominally obese. A number of studies have developed reference waist circumference percentiles for children and adolescents in different countries (Moreno *et al*., 1999; McCarthy *et al.*, 2001; Katzmaryzyk, 2004; Fernandez *et al*., 2004; Eisenmann, 2005; Hatipoglu *et al*., 2008). The cut-off values developed for the American population has been the norm because of the exhaustive and comprehensive methods used for its development, review and presentation (Fernandez *et al*., 2004). In several research analyses, these data are often compared with data developed from other continents and countries and results have consistently indicated that the American data do not adequately describe other populations outside the United States in several different situations. This has thus led to the recommendation that population and country-specific cut-off values for WC could be developed (Liu *et al*., 2010).

Information on fatness and fat patterning in South African children is limited (Monyeki *et al*., 2006; Mukuddem-Petersen *et al.,* 2006; Naude *et al*., 2009). It is thus important to study the fat distribution in South African children. Children who are at risk of becoming obese need to be identified early enough so that appropriate intervention can be initiated. This can be achieved by accurate measurement of total and regional body fat and evaluating whether an individual child is deviating from normal values or growth trends. It is increasingly recognized that the occurrence of adult cardiovascular diseases (CVDs) is influenced by factors operating throughout the life course (Kuh *et al.,* 2004). An increased risk for CVD may start in infancy or even before birth and will continue to be influenced by health-related behaviour during adulthood. Children are, therefore, an important target group for health intervention. Prevention is recognized as the only feasible option to reduce childhood obesity. Current treatment practices for obese children and adolescents are largely aimed at controlling the problem rather than effecting a cure (Lobstein *et al.,* 2004). The non-availability of well-structured and comprehensive age and gender-specific waist circumference percentiles for black South African children and adolescents is a current and major challenge for body composition research. The development of waist circumference percentiles and cut-offs for different groups would be particularly valuable (Liu *et al*., 2010). To date no waist circumference reference values for South African children exist. Therefore, there is an urgent need to develop reference values for this specific population group as this will enable health professionals to identify children who are at risk of abdominal obesity accurately. There is a need to explore the optimal waist circumference cut-off values for predicting cardiovascular risk in this population and assess the influence of stunting on obesity development.

1.2 Background

Childhood and adolescence are the greatest periods of change throughout the lifetime of an individual (Spear, 2002; Cameron, 1997). This period is characterized by changes in body shape, beginning of independent and abstract cognitive processes, and the beginning of the adolescent's transition to the social values and roles of adulthood (Heald, 1975). Biological and hormonal changes, together with the complex social issues that these

children face, often overshadow the development of non-communicable diseases (Bojorntor, 1992). The risk factors for these non-communicable diseases include, amongst others, the excess accumulation of body fat resulting in obesity. There has been a sharp increase in childhood obesity worldwide (Ebbeling *et al.*, 2002; Lobstein *et al.*, 2004; Ogden *et al*, 2006) and this constitutes a major threat to global human health (Ozcan *et al.*, 2009). Wang and Lobstein (2006) estimated that by 2010 approximately 41% of children in the Eastern Mediterranean region, 38% in Europe and 22% in South East Asia will be obese. The prevalence of overweight and obese children aged 6-13 years was found to be 14.0% for boys and 17.9% for girls in a study of six provinces in South Africa (Armstrong *et al.*, 2006). In a nationally representative study the prevalence of combined overweight and obesity among high school children increased from 21.2% in 2002 (MRC, 2002) to 25% in 2008, with 35% of high school girls being overweight or obese (Reddy *et al*., 2010).

The rapid rise in obesity trends is considered a major driving force behind the high prevalence of pediatric metabolic syndrome (Kelishadi, 2007). This rapid rise underlines the urgency for a definition that could be used to understand further who is at high risk of health complications. Previously a wide range of definitions of metabolic syndrome (MS) in children was used until the International Diabetes Federation (IDF) developed a consensus definition of MS for children. The rationale for this was to obtain a universally accepted tool which is easy to use for the early diagnosis of metabolic syndrome, in order to take preventive measures before the child or adolescent develops diabetes or cardiovascular disease. This definition states that, "for children age 10 years or older, metabolic syndrome can be diagnosed with abdominal obesity (using waist circumference percentiles) and the presence of two or more other clinical features including elevated triglycerides, low HDL-cholesterol, high blood pressure, increased plasma glucose, whilst for ages above 16 years the adult IDF criteria can be used (Alberti *et al*., 2007). Abdominal fat seems to be the central factor because most patients with the syndrome have excess abdominal fat.

Puoane *et al.* (2002) expressed concern that prevention and management of obesity in African populations is complicated because of traditional and cultural perceptions concerning body size. Despite the health risks associated with obesity, it is considered a sign of health and wealth in many African communities. In women obesity is thought to reflect on a husband's ability to care for his wife and family (Mvo *et al.*, 1999). In addition to the well recognized increased morbidity and mortality associated with obesity, there is increasing evidence indicating intra-abdominal (visceral) adipose tissue as the fat depot with the greatest risk of metabolic complications (Lemieux *et al.*, 2007). In adults, a central patterning of fat has been shown to be associated with coronary heart diseases and type 2 diabetes (Must *et al.*, 1992). Centralization of body fat has been documented in white, black and Mexican American adolescents, although the degree of centralization differs between ethnic groups (Fernandez *et al.*, 2004). Goran and Gower (1999) maintain that visceral adipose tissue is minimally present in newborns and usually sparse among children. However, the emergence of visceral fat in children and adolescents could be interpreted as a specific marker of systemic lipid over accumulation. Kahn *et al.* (2005) argue that of particular importance to future disease, the excess lipid fuels may find their way into ectopic sites of lipid storage where they can cause substantial metabolic disruption.

Ajiduah (2002) showed that the prevalence of obesity among children aged 3 to 9 years in developing countries ranges between 7 and 10%. The underlying facts are that obesity is common among African urban female adolescents and women, but rare among African males (Eboh & Boye, 2005). South Africa is experiencing a quadruple burden of disease comprising pre-transitional diseases, the emerging chronic diseases, injuries, and HIV/AIDS (Bradshaw *et al.*, 2003). The World Health Organization (WHO) recommends that developing countries monitor disease trends, particularly the co-existence of stunting and overweight in children, since these are risk factors for chronic disease in adulthood (WHO, 2002). With reference to obesity the assessment of body composition is useful for screening of excess body fatness, its distribution pattern and related metabolic complications. Several techniques are available for estimating body composition. Dual Energy X-Ray Absorptiometry (DEXA) and magnetic resonance

imaging (MRI) are the most reliable methods to obtain accurate measures of total fat (Parker *et al.*, 2003). These methods are, however, not suitable for field and clinical use. Recent attention, therefore, focused on the use of anthropometric markers when population size is big, resources are scarce and a quick measure is required. The challenges when using anthropometric measurements are related to the factors that can affect their accuracy and precision, namely non-standardized methodology, technical and measurement limitations, selection of adequate fat mass prediction equations for each age group, and measurement discrepancies between methods (Moreno *et al.*, 2003). Body mass index (BMI) has been a measure of choice in national surveys for defining overweight in children and has been recommended by health experts for use in clinical practice (Thompson *et al.*, 2007). The use of BMI has limitations when measuring adiposity, and is unable to distinguish between gains in fat free mass and fat mass. Accurate measurement of total and regional body fat is critical to detect as early as possible whether the population overall or a given child in particular is deviating from normal body composition trends (Fernandez *et al.*, 2004). This is a serious shortcoming in the use of BMI because it is well known that excess accumulation of abdominal fat rather than peripheral distribution carries a higher risk of obesity related ill health.

In adults it is well established that abdominal adiposity as measured by waist circumference is associated with increased risk of cardiovascular disease, dyslipidemia, and type 2 diabetes mellitus independent of overall adiposity (Lee *et al.*, 2006). Until recently waist circumference in children was not regarded as being an important measure of adiposity. Lately it has been observed that waist circumference is a reasonably sensitive and specific measure of upper body fat and is valuable in identifying overweight and obese children at risk of developing metabolic complications. The health effects associated with excessive abdominal fat in children make it an issue of public health significance. De Ridder (1992) and Fox (1993) were among the first to investigate intra-abdominal fat deposition in children. Since then several other studies showed strong associations between waist circumference and risk factors for coronary heart disease. Sarria and co-workers (2001) state that, “it has been observed that waist circumference is a good tool for screening of excess body fat percentage in children and adolescents”. A

waist circumference at the 75th and 90th percentile indicates a high risk and a very high risk for co-morbidities respectively (Fernandez *et al.*, 2004). Some studies demonstrated an adverse atherogenic lipoprotein profile with increasing waist circumference in obese children *(*Flodmark *et al.*, 1993). Others, for example the Bogalusa Heart Study, showed that abdominal fat distribution in children aged between 5 and 17 years was associated with abnormal concentrations of triacylglycerol, LDL-C, HDL-C and insulin (Freedman *et al.*, 1999). Outcomes of these studies led to the use of waist circumference as a marker of abdominal obesity in children and since then reference curves have been developed in some countries such as the USA, Canada, UK, Spain, Italy, Cuba, Australia, Japan, Germany, Mexico and in Africa, Nigeria. However, there is no global standard for waist circumference in children. Cut-off values differ between genders, age groups, ethnic groups and countries (Kelishadi, 2006). The concept of "reference values" embraces the notion of normality, of a desirable, ideal pattern and/or target; thus, the growth pattern of one country cannot be considered as the gold standard against which to compare all the others (Caroli *et al.*, 2007). National reference curves, thus, will allow an intra-country comparison over time or by regions, but cannot be used for comparisons between countries.

The waist-to-height ratio (WHtR) is an anthropometric index that can be utilized to identify children at risk of developing metabolic complications. According to Ashwell and co-workers (1996), a high WHtR is an estimator of the intra-abdominal mass of adipose tissue along with the subcutaneous truncal fat mass in adults. The WHtR has been shown to be associated with cardiovascular risk factors such as, for example, hypertension (Hsieh *et al.*, 1995). A cut-off value of 0.5 for WHtR has been proposed (Ashwell *et al.*, 2005); values above 0.5 are associated with an increased cardiovascular risk in adults (Freedman *et al.*, 2007). This cut-off value has not been validated for use in children. It is possible, but not yet demonstrated that the idea of enlarged waist relative to height may be easier for patients and their families to grasp than other anthropometric indices such as BMI percentiles. The WHtR is not age or sex dependent, and it could potentially replace the use of BMI percentiles for the assessment of cardiovascular disease risk associated with overweight and central obesity. Kahn and co-workers (2005)

suggested that if a child's WHtR identifies cardiovascular risk at least as well as his or her BMI percentile, then the choice of anthropometric index may depend on the comparative ease and reliability of the two indices.

This thesis focuses particularly on the distribution of waist circumference, the use of WHtR as a marker for high blood pressure and the comparison of fat distribution in stunted and non-stunted South African black children. Discussions on these focus areas will provide information about the growth and fat patterning in black South African children and also assess the utility of WHtR as a marker for high blood pressure.

1.3 Aim of the study

To describe the distribution of waist circumference according to percentiles in black South African children aged 6-18 years.

1.3.1 Specific Objectives

a. To collect additional anthropometric data and add it to already available data and use it to describe waist circumference percentile distribution in black South African children aged 6 to 18 years.
b. To explore existing and newly collected data of waist circumference of black South African children and compare percentile cut-off points with international data sets.
c. To assess the sensitivity of WHtR in identifying children with high blood pressure.
d. To investigate differences between body composition of stunted and non-stunted children using waist circumference and WHtR measurement.
e.

1.4 Hypotheses

a. The suggested cut-off point of 0.5 for waist-to-height ratio as a simple indicator of excessive amount of central fat is sensitive to identify South African children with high blood pressure.

b. Waist circumference and waist-to-height ratio, respectively of stunted and non-stunted South African children from rural communities are significantly different.

1.5 Structure of the thesis

This thesis will be presented in article format; hence the bibliography for Chapters 1, 2 and 6 will be according to the North-West University style which corresponds to the Harvard method and is subscribed to by the International Standards Organization. The bibliography of Chapters 3, 4 and 5 will be according to the style of the journal to which the specific article is being submitted for publication.

Chapter 1: Background information for the thesis is given in order to put the topic into perspective. Also included are the specific objectives of the study and the thesis structure.

Chapter 2: The literature review on the thesis topic is given in order to highlight the current status of knowledge on the subject and to provide information supporting the arguments about the findings.

Chapter 3: Body composition in stunted compared to non-stunted black South African children from two rural communities. This chapter is written in the format of an article and will be submitted for publication in the International Journal of Pediatric Obesity.

Chapter 4: Percentile distribution of waist circumference of black South African children from different study populations. This chapter will be submitted for publication (appropriate journal to be identified).

Chapter 5: The sensitivity of waist-to-height ratio in predicting high blood pressure in black South African children. This chapter is written in the format of an article submitted for publication in the Cardiovascular Journal of Africa.

Chapter 6: General discussion, conclusions and recommendations from the different articles will be presented and summarized.

Each chapter will be followed by a bibliography of all the literature sources referenced.

1.6 Author contributions

B S Motswagole- PhD student

Substantial contribution to the conception, design and acquisition of newly collected data, statistical analysis and interpretation of data. Final manuscript writing and final approval of the version to be published.

H Salome Kruger- Promoter

Substantial contributions to conception and design, data collection, analysis and interpretation of data and supervision of manuscript writing, final approval of the version to be published.

M Faber- Co-promoter

Substantial contributions to conception and design, data collection, analysis and interpretation of data and supervision of manuscript writing.

JM van Rooyen, JH de Ridder, KD Monyeki, L Motseki, T Matsha, RT Erasmus, E Kimani-Murage, SA Norris

Shared data from previous studies, reviewed the manuscript and final approval of the paper to be published.

1.7 References

AJIDUAH, D., 2002. Stunting is associated with overweight in children of four nations are undergoing the nutritional transition. Paper presented at NAPHER–SD week of UNILAG chapter. Lagos, Nigeria

ALBERTI, K.G.M.M., ZIMMET, P.Z & SHAW, J. E. 2007. The metabolic syndrome in children and adolescents, *Lancet,* 369:2059-2061.

ARMSTRONG, M E., LAMBERT, M.I., SHARWOOD, K. A & LAMBERT, E.V. 2006. Obesity and overweight in South African primary school children -- the Health of the Nation Study. *South African medical journal,* 96(5): 439-44.

ASHWELL, M. & HSIEH, S. 2005. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity, *International journal of food sciences and nutrition*, 56(5): 303-307.

ASHWELL, M., COLE, T. J & DIXON, J. K. 1996. Ratio of waist circumference to height is strong predictor of intra-abdominal fat. *British medical journal*, 313:559-560.

BOJORNTOR, P. 1992. Regional obesity. (*In*: Bjortorp. P, Brodoff BN *eds*. *Obesity*. Lippincott: Philadelphia.

BRADSHAW, D., GROENEWALD, P., LAUBSCHER, R., NANNAN, N., NOJILANA, B., NORMAN, R., PIETERSE, E., SCHNEIDER, M., BOURNE, D.E., TIMÆUS, I.M., DORRINGTON, R & JOHNSON, L. Initial burden of disease estimates for South Africa, 2000. 2003. *South African medical journal,* 93: 682-688.

CAMERON, A.J., DUNSTUN, D.W., OWEN, N., ZIMMET, P.Z., BARR, E.L., TONKIN, A.M. et al. 2009. Health and mortality consequences of abdominal obesity: evidence from the AUSDiab study. *The medical journal of Australia*, 191(4):202-208

CAMERON, N. 1997. African obesity: a puzzle solved. *South African medical journal,* 87: 1396–1397.

CAROLI, M., WIJNHOVEN, T. M. A. & BRANCA, F. 2007. Methodological considerations for childhood surveillance systems: the case of obesity. *Journal of public health*, 15(3):147-153.

DE MORAES, A.C.F., FADONI, R.P., RICARDI, L.M., SOUZA, T.C., ROSANELI, C.F., NAKASHIMA, A.T.A. et al. 2010. Prevalence of abdominal obesity in adolescents: a systematic review. *Obesity reviews* no. doi:10.1111/j.1467-789X.2010.00753.x

DE RIDDER, C.M., DE BOER, R.W., SEIDELL, J.C., NIEUWENHOFF, C.M., JENESON, J.A., BAKKER, C.J., ZONDERLAND, M.L & ERICH, W.B. 1992. Body fat distribution in pubertal girls quantified by magnetic resonance imaging. *International journal of obesity and related metabolic disorders*, 16:443-449.

EBBELING, C., DOROTA, B., PAWLAK, D. & LUDWIG, D. 2002. Childhood obesity: public-health crisis, common sense cure. *Lancet*, 360:473-482.

EBOH, L. O. & BOYE, L. 2005. Investigation of Body Composition of Normal and Malnourished Rural Children (3-11 Years) In the Niger – Delta Region of Nigeria. *Pakistan journal of nutrition*, 4(6): 418-422.

EISENMANN, J.C. 2005. Waist circumference percentiles for 7- to 15-year-old Australian children. *Acta Paediatrica*, 94:1182-1185

FERNADEZ, J.R., REDDEN D.T., PIETROBELLI, A & ALISON, D.B. 2004. Waist circumference percentiles in nationally representative samples of African-American, European-American and Mexican-American children and adolescents. *Journal of paediatrics*, 145:439-444.

FLODMARK, C.E & OHLSSON, T. 1993. Prevention of progression of obesity in a group of obese school children treated with family therapy. *Paediatrics*, 19:880-884.

FOX, K., PETERS, D., ARMSTRONG, N., SHARPE, P & BELL, M. 1993. Abdominal fat deposition in 11-year-old children. *International journal of obesity and related metabolic disorders*; 17:11-16.

FREEDMAN, D. S., DIETZ, W. H., SRINAVASAN, S. R., & BERENSON, G.S. 1999. The relation of overweight to cardiovascular risk factors among children and adolescents: *Paediatrics,* 103(6):1175-1182.

FREEDMAN, D.S., KAHN, H.S., MEI, Z., GRUMMER-STRAWN, L.M., DIETZ W.H., SRINAVASAN, S.R & BERENSON, G.S. 2007. Relation of body mass index and waist-to-height ratio to cardiovascular disease risk factors in children and adolescents: the Bogalusa Heart Study. *American journal of clinical nutrition,* 86:33-40.

GALCHEVA, S.V., IOTOVA, V.M., YOTOV, Y.T., GROSDEVA, K.P., STRATEV, V.K & TZANEVA, V.I. 2009. Waist circumference percentile curves for Bulgarian children and adolescents aged 6-18 years. *International journal of paediatric obesity,* 4(4):381-388.

GORAN, M.I & GOWER, B.A. 1999. Relation between visceral fat and disease risk in children and adolescents. *American journal of clinical nutrition,* 70**:**149S-156S.

HATIPOGLU, N., OZTURK, A., MAZICIOGLU, M.M., KURTOGLU, S., SEYHAN, S. & LOKOGLU, F: 2008. Waist circumference percentiles for 7- to 17-year-old Turkish children and adolescents. *European journal of paediatrics,* 167**:**383-389.

HEALD, F.P. 1975. Adolescence Nutrition. *The medical clinics of North America,* 59:1329–1336.

HSIEH, S. D & YOSHANIGA, H. 1995. Abdominal fat distribution and coronary heart disease risk factors in men-waist-height ratio as a simple and useful predictor. *International journal of obesity and related metabolism,* 14:485-589

KAHN, H. S., IMPERATORE, G & CHENG, Y. J. 2005. A population based comparison of BMI percentiles and waist-to-height ratio in identifying cardiovascular risk in youth. *The journal of paediatrics,* 146(4):482-488.

KATZMARZYK, P.T: 2004. Waist circumference percentiles for Canadian youth 11-18y age**.** *European journal of clinical nutrition,* 58:1011-1015.

KELISHADI, R. 2007. Childhood Overweight, Obesity, and the Metabolic Syndrome in Developing Countries. *Epidemiologic reviews*, 29(1):62-76.

KELISHADI, R., ARDALAN, G., GHEIRATMAND, R., ADELI, K., DELAVARI, A. & MAJDZADEH, R. 2006. Paediatric metabolic syndrome and associated anthropometric indices: The Caspian Study. *Acta Paediatrica,* 95(12):1625-1634

KUH, D.J.L & BEN S.Y. 2004. A life course approach to chronic disease epidemiology. *Oxford University Press*: Oxford.

LEE, Y., NASEEM, B.H. PARK, B.H., GARRY, D.J., RICHARDSON, J.A., SCHAFFER, J. E & UNGER, R. H. 200*6.* Alpha-lipoic acid prevents lipotoxic cardiomyopathy in acyl CoA-synthase transgenic mice. *Biochemical and bio physiological research communications,* 344:446–452.

LEMIEUX, I., POIRIER, P., BERGERON, J., ALMERAS, N., LAMARCHE, B., CANTIN, B., DAGENAIS, G.R. & DESPRES, J-P. 2007. Hypertriglyceridemic waist: a useful screening phenotype in preventive cardiology. *Canadian journal of cardiology,* 23(supp b):23b-31b.

LIU, A., HILLS, A.P., HU, X., LI, Y., DU, L., XU, Y. et al. 2010. Waist circumference cut-off values for the prediction of cardiovascular risk factors clustering in Chinese school-aged children: a cross-sectional study. *BMC Public Health,* 10:82.

LOBSTEIN, T., BAUR, L., UAUY, R & IASO International Obesity Task Force. 2004. Obesity in children and young people: a crisis in public health. *Obesity reviews*, 5 (suppl 1):4-104.

MCCARTHY, H., JARRETT, H. & CRAWLEY, H: 2001. The development of waist circumference percentiles in British children aged 5.0-16.9 y**.** *European journal of clinical nutrition*, 55:902-907.

Medical Research Council (MRC). Umthente uhlaba usamila: the 1st South African National Youth Risk Behaviour Survey. Cape Town: South African Medical Research

Council, 2002. http://www.mrc.ac.za/healthpromotion/healthpromotion.htm Date of access 16 Nov. 2010

MONYEKI, K.D., KEMPER, H.C.G. & MAKGAE, P.J. 2006. The association of fat patterning with blood pressure in rural South African children: the Ellisras longitudinal study. *International journal of epidemiology*, 35 (1): 114-120

MORENO, L. A., JOYANNES, M., MESANA, M. I., GONZALEZ-GROSS, M., GIL, C. M., SARRIA, A., GUTIERREZ, A., GARAULET, M., PEREZ-PRIETO, R., BUENO, M., MARIOS, A & AVENA study group. 2003. Harmonization of anthropometric measures for a multicenter nutrition survey in Spanish adolescents. *Nutrition*, 19:481-486.

MORENO, L., FLETA, J., MUR, L., RODRÍGUEZ, G., SARRÍA, A., BUENO, M: 1999. Waist circumference values in Spanish children-gender related differences. *European journal of clinical nutrition*, 53:429-433.

MUKUDDEM-PETERSEN, J & KRUGER, H.S. 2004. Association between stunting and overweight among 10-15 year old children in North West province, South Africa: the THUSA BANA study. *International journal of obesity*, 28:842-851

MUST, A., JACQUES, P.F., DALLAL, G., BAJEMA, C.J & DIETZ, W. H. 1992. Long-term morbidity and mortality of overweight adolescents. A follow-up of the Harvard Growth Study of 1922 to 1935. *New England journal of medicine*, 327:1350-1355.

MVO, Z., DICK, J & STEYN, K. 1999. Perceptions of overweight African women about acceptable body size of women and children. *Curationis*, 22:27-31.

NAUDÉ, D., KRUGER, H.S. & PIENAAR, A.E. 2009. Differences in body composition, body proportions and timing of puberty between stunted and non-stunted adolescents. *African journal for physical, health education, recreation and dance*, 15(4):678-689.

OGDEN, C., CAROLL, M.D., CURTIN, L.R., MCDOWELL, M.A., TABAK, C.J. & FLEGAL, K.M. 2006. Prevalence of overweight and obesity in the United States, 1999-2004. The *journal of the American medical association*, 295:1549-1555.

OZCAN, L., ERGIN, A.S., LU, A., CHUNG, J., SARKAR, S., NIE, D., MEYERS (Jr), M.G. & OZCAN, U. 2009. Endoplasmic reticulum stress plays a central role in development of leptin resistance. *Cell metabolism*, 9(1):35-51.

PARKER, L., REILLY, J.J., SLATER, C., WELLS, J.C.K & PITSLADIS, Y. 2003. Validity of six field and laboratory methods for measurement of body composition in boys. *Obesity research*, 11:852-858.

POUANE, T., STEYN, K., BRADSHAW, D., LAUBSCHER, R., FOURIE, J., LAMBERT, V. & MBANAGA, N. 2002. Obesity in South Africa: The South African Demographic Health Survey, *Obesity research*, 10(10):1038-1048.

REDDY, S.P., JAMES, S., SEWPAUL, R., KOOPMAN, F., FUNANI, N.I., SIFUNDA, S.et al Umthente Uhlaba Usamila - The South African Youth Risk Behaviour Survey 2008. Cape Town: South African Medical Research Council; 2010. http://www.mrc.ac.za/healthpromotion/healthpromotion.htm Date of access 16 Nov. 2010

SARRIA, A., MORENO L. A., GARUA-LLOP, L. A., FLETA, J., MORELLON, M P & BUENO, M. 2001. Body mass index, triceps skinfolds and waist circumference in screening for adiposity in male children and adolescents. *Acta Paediatrica*, 90: 387-392.

SIBBEL, G. 2005. South Africa malnutrition the changes of urbanization. Available at: http://www.worldfoodprize.org. Date of access 29 Jul 2010.

SPEAR, B.A. 2002. Adolescence growth and development. *Journal of American dietetic association*, 102: S23–S29.

STIGMAN, S., RINTALA, P., KUKKONEN-HARJULA, K., KUJALA, Y., RINNE, M., FOGELHOM, M. 2009. Eight year old children with high cardio-respiratory fitness

have lower overall and abdominal fatness. *International journal of paediatric obesity*, 4(2):98-105

THOMPSON, D., OBARZANEK, D., FRANKO, D., BARTON, B., MORRISON, J., BIRO, F., DANIELS, S & STRIGEL-MORE, R. 2007. Childhood overweight and cardiovascular disease risk factors: The National Heart, Lung, and Blood Institute Growth and Health Study. *The journal of paediatrics*, 150(1):18-25.

WANG, Y. & LOBSTEIN, T. 2006. Worldwide trends in childhood overweight and obesity. *International journal of paediatric obesity*, 1:11-25.

WHO. The problem of overweight and obesity. Available at: http://whqlibdoc.who.int/trs/WHO_TRS_894_(**part1**).pdf .Date of access: 29 Jul. 2010.

WHO. 2002. The World Health Report: Reducing risk, promoting healthy lifestyle. Geneva, Switzerland

WIGGA, A.H., SCHOLTENS, S., BEMELMANS, W.J.E., DE JONSTE, J.C., KERKHOF, M., SCHIPPER, M. et al. 2010. Co-morbidities of obesity in school children: a cross-sectional study in the PIAMA birth cohort. *BMC public health*, 10:184

2

LITERATURE REVIEW

FAT DISTRIBUTION AND ITS HEALTH IMPLICATIONS: FOCUS ON CHILDREN

For the medical profession, body fat stores represent a physical trait that can be measured and intervened on. From an evolutionary perspective, however, body fat represents a strategy - the strategy of storing lipid rather than oxidising it on an immediate basis, and of using these stores to regulate a set of competing biological functions.

Leila Beker, 2006.

2.0 Literature review

2.1 Overview

2.2 Body fat distribution

2.3 Nutritional disorders and body fat in children

2.4 Health effects of excess abdominal fat

2.5 Abdominal fat assessment

2.6 WC percentile charts for children

2.7 Summary

2.8 References

2.1 Overview

This chapter reviews literature that is applicable to the understanding of body fat distribution and its health consequences. The review includes a description of fat storage mechanisms in the human body, the assessment techniques used for determining abdominal fat together with health implications of accumulating excess fat in the body. The usefulness of using waist circumference (WC) measurement in children for assessing abdominal fat is presented. The review further highlights the need for the inclusion of WC as a vital sign in clinical paediatric practice based on research findings pointing towards the association between excess abdominal fat accumulation and adverse health outcomes in children later in life. The inclusion of WC measurement as a vital sign is considered as a key step in identifying children at risk of adverse health complications, hence action could be taken before it is too late.

2.1.1 Fat storage in adipose tissue.

Excess accumulation of fat in the body is fundamentally a process of energy balance. It occurs when energy intake is beyond that needed for current energy demands. This excess energy is stored as fat in the adipose tissue during lipogenesis. Adipose tissue, therefore, functions as a storage site for fat. The excess energy is stored in the form of

triglycerides. The mechanism of how excess energy is stored in adipose tissue is that there may be some kind of abnormal signal that affects adipose tissue metabolism and alters fuel partitioning, therefore directing fat storage in adipose tissue instead of use in muscle (Trayhurn, 2007). In adult mammals, the major bulk of adipose tissue is a loose association of lipid-filled cells called adipocytes, which are held in a framework of collagen fibers (Albright *et al.*, 1998). In addition to adipocytes, adipose tissue contains stromal-vascular cells including fibroblastic connective tissue cells, leukocytes, macrophages, and pre-adipocytes, which contribute to structural integrity (Albright *et al.*, 1998). In humans, adipose tissue is located beneath the skin (subcutaneous fat), around internal organs (visceral fat), and in the bone marrow (yellow bone marrow) (Albright *et al.*, 1998).

2.1.2 Biological role of adipose tissue

For a long time adipose tissue was considered to be an inactive reserve depot of fat (Gooren, 2008). Adipose tissue has now moved center stage in obesity research, there having been a revolution in the understanding of the biological role of the tissue (Trayhurn, 2007). Once perceived as a passive compartment whose sole function was to store fat as an energy source, adipose tissue is now viewed as an active endocrine organ that produces a variety of factors with a vast array of physiologic actions as shown in Figure 2.2. It is now recognized that it is directly or indirectly involved in the control of body weight and energy balance via the secretion of a large number of molecules with regulatory potential (Wang *et al.*, 2008).

Adipose tissue is found as brown and white adipose tissue in mammals including humans. White adipose tissue is a highly metabolic active endocrine organ whose products of secretion, i.e. adipokines, have essential roles in energy homeostasis, glucose and energy metabolism, cell viability, feeding control, thermogenesis, neuroendocrine function, reproduction, immunity, and importantly cardiovascular function (Trujillo *et al.*, 2006). The effect of some of these adipokines is covered in another section of this chapter. Different factors secreted by the adipose tissue are shown in Figure 2.2. Subcutaneous adipose tissue found directly below the skin is an especially important heat

insulator in the body, because it conducts heat only one third as readily as other tissues. Brown adipose tissue derives its colour from rich vascularization and densely packed mitochondria. It is metabolically less active, although cold exposure can activate it (Albright *et al.*, 1998).

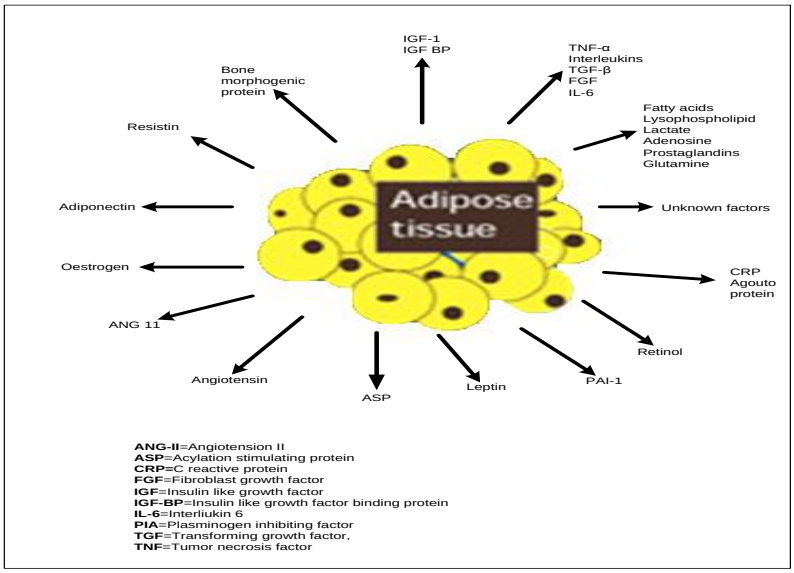


Figure 2.2: Diagram showing different factors secreted by adipose tissue. (Al-Mubaslat, 2005)

2.2 BODY FAT DISTRIBUTION

Fat is not uniformly distributed in the body as shown in Table 2.1 and the subcutaneous fat tissue is the major lipid storage compartment (about 80% of all body fat) (Arner, 1997). Recent studies have shown that lipid partitioning is a major determinant of metabolic profile and not obesity *per se* (Weiss, 2007), in particular abdominal fat. Märin *et al.* (1992) described abdominal fat as composed of abdominal subcutaneous fat and intra-abdominal fat. Intra-abdominal adipose tissue is composed of visceral, or intra-peritoneal, fat, mainly composed of omental and mesenteric fat and retroperitoneal fat masses by delineation along the dorsal borderline of the intestines and the ventral surface of the kidney (Wajchenberg, 2000).

In situations where the subcutaneous fat tissue reaches threshold levels and can store no more fat, the excess is then stored in other body compartments (Weiss, 2007). These depots in other compartments may not be favourable for fat storage and may ultimately affect normal metabolic pathways. The central or abdominal adipose tissue in the abdominal region is one such compartment. Excess intra-abdominal fat may, therefore, be a warning sign that excess energy is being stored as fat in unusual places, increasing the risk of metabolic complications. This pattern of fat storage has been observed to impact on (i) adipocytokine secretion profile, (ii) circulating concentration of inflammatory cytokines and (iii) free fatty acid (FFA) flux (Weiss, 2007).

Table 2.1: Body Fat distribution

Depot	**Remarks**
Subcutaneous	About 80% of all body fat. Can functionally be divided into abdominal and gluteofemoral.
Visceral	Drained by the portal vein. Anatomically divided into omental and mesenteric fat.
Other	Retroperitoneal, perirenal and orbital.

Arner, 1997

Different individuals do not carry their adipose tissue in the same anatomical locations. Adipose tissue that is located predominantly on the upper body has been termed android, male, central, upper-body segment, or "apple" distribution. Whilst in females adipose tissue accumulates predominantly in the lower body, and the terms gynoid, female, lower-body segment, or "pear" distribution is usually used (Albright *et al.*, 1998). The comparison of these types of body fat distribution is shown in Figure 2.2.

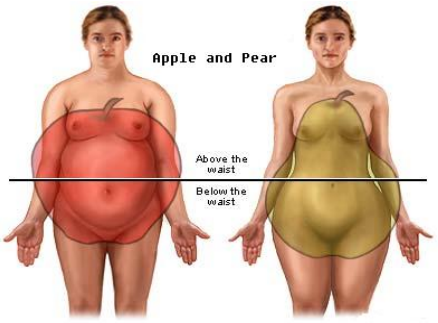


Figure 2.2: Illustration of pear and apple body fat distribution (http://www.health-emark.com-fat body types).

2.2.1 Fat distribution in children

Growth during childhood represents a time of rapid change in body composition. However, there have been few longitudinal studies that investigated the changes in specific fat compartments during the growth process. Looking at the changes in different fat compartments during the growth process may help to elucidate the dynamics of growth in children and how changes in body composition may be related to health outcomes. This is especially important for the growth of visceral fat, which may contribute to metabolic disease risk *(*Huang *et al.*, 2001*)*. Excess fat accumulation in children may be influenced by distinct growth periods during early childhood. Zafon (2007) argues that gains in adiposity at key ages are controlled by regulatory mechanisms that favour fat storage when energy is readily available and that high fat mass has important survival value in the face of stressors likely to be encountered during specific developmental periods.

The first period of increasing body fat mass occurs during the first year of life with rapid growth in size but a stable number of adipose cells. Fat cell size declines over the next 1 to 2 years and remains stable for several years (Knittle *et al.*, 1979). The second phase, termed adiposity rebound (Rolland-Cachera *et al.*, 1984), is characterized by rapid growth in body fat, which usually begins about age 6 years, and includes increases in both cell size and cell number. Linear increases in body fat and percent body fat occur from about 2 to 14 years, with a substantial increase in the variability of these measures at

about 5 or 6 years, especially among marginalized girls (Ellis *et al.*, 1997*).* Children who experience adiposity rebound at an earlier age are more likely to have higher adiposity at age 14 years (Knittle *et al.*, 1979), and obese adolescents have a relative risk of 5.3 to 6.7 of remaining obese as young adults (Kotari *et al.*, 1997).

Previous studies have documented gender and ethnic differences in fat distribution. He *et al.* (2002) proposed that differences in fat distribution between men and women are already evident in prepubertal children. Afghani and Goran (2006) stated that 'ethnic disparities in central adiposity may be especially complex because of racial differences in body size, body composition, regional fat accumulation, insulin levels and insulin resistance'. On the other hand, Harsha *et al.* (1980) suggested that racial difference in distribution of fat may manifest a genetic adaptive trait developed under circumstances demanding both a caloric reserve and facilitation of convective heat loss in tropical climates. The recognition of these differences is of clinical importance because the metabolic implications of particular body composition parameters may vary among races. For example, the strength of association between specific fat depots and insulin sensitivity or high-density lipoprotein cholesterol (HDL-C) was found to be different in black and white children (Yanovski *et al.*, 1996). This emphasizes the importance of race-specific interpretations of body composition and the need to explore further associations with health risks. Studies on race-specific fat distribution patterns will guide investigations of metabolic implications and possible mechanisms. Fat distribution seems to differ with gender as well. Male fat deposition is preferentially intra-abdominal at all ages (Matsuzawa *et al.*, 1995*)* and subcutaneous adipose tissue tends to decrease after the age of 50 years (Zamboni *et al.*, 1996).

2.3 NUTRITIONAL DISORDERS AND BODY FAT IN CHILDREN.

It has been long recognized that good nutrition for children is fundamental for attaining optimal growth. Nutrition is coming to the fore as a major modifiable determinant of chronic disease, with scientific evidence increasingly supporting the view that alterations in diet have strong effects, both positive and negative, on health throughout life (WHO, 1990). Most importantly, it has been observed that dietary adjustments may not only

influence present health, but may determine whether or not an individual will develop such diseases as cancer, cardiovascular disease and diabetes much later in life (FAO, 2002). Getting the right balance of nutrients for children is, however, challenging because children's nutritional needs change as they grow, along with their food preferences, eating habits and activity levels. A number of different nutrition disorders may arise, depending on which nutrients are under or overabundant in the diet. Two common nutrition disorders, namely undernutrition and overnutrition are discussed with specific focus on children.

2.3.1 Undernutrition

Undernutrition is when the body contains lower than normal amounts of one or more nutrients (Black *et al.*, 2008). It includes stunting (short stature for age) and wasting (low weight for height) and deficiencies in vitamins and minerals (micronutrient malnutrition) (UNICEF, 2006). The global burden of undernutrition is high and is worsening in some countries. For instance in countries with a high population, such as India, undernutrition persists or has worsened. The estimated number of undernourished people in developing countries was 824 million in 1990-92, in 2003–05, the figure stood at 848 million and it went up to 923 million in 2007 and reached 967 million in 2008(FAO, 2008). Undernutrition, particularly in children, is affecting human beings in all its unkindness, preventing individuals and societies from achieving their full potential. Children who suffer from undernutrition usually have lowered resistance to infection and are more likely to die from such common childhood ailments as diarrhoea and respiratory infections (Manary *et al.*, 2004). Those who survive may be locked into a vicious cycle of recurring sickness and faltering growth, often with irreversible damage to their cognitive and social development (UNICEF, 2006). Undernutrion is an outcome of various factors resulting from unfavourable circumstances. These include difficulties in obtaining food, unemployment (which determines irregular income for the family), limited access to education and health services or illness caused by unsanitary conditions (Reyes *et al*, 2004). Demographic characteristics such as child's age and gender, birth interval (both preceding and succeeding) and the mothers age at the child's birth, have also been associated with child nutritional status (Vella *et al.*, 1992). Undernourished

children normally stop gaining weight and stop growing and in severe cases, especially when under 5 years of age, they suffer from the effects more quickly than adults, because of their higher nutritional requirements in relation to their small size (British Nutrition Foundation, 2005). Clinically undernutrition is associated with greater mortality rates from most childhood diseases (Manary *et al.*, 2004). However, scientific evidence regarding this condition has contributed to improvements in its clinical management and, in turn, survival over the last 20 years (Bhutta *et al*., 2008).

In most of the developing countries even though malnutrition may be due to poverty, rates have been observed to worsen during droughts, economic crises, conflicts and displacement, and recently the emergence of human immunodeficiency virus (HIV) infection (Jamieson, 2006). Besides poverty, other issues relating to food intake, food habits, false beliefs and deep rooted taboos in some countries have a profound impact on nutritional status of individuals who hold them, because they affect the selection of diets (Onuorah *et al.,* 2003), consequently making the problem of nutritional disorders more acute and exerting a much more profound impact on children.

Undernutrition and fat deposition in children

It is interesting to note that stunting (an outcome of undernutrition) has been linked to central fat deposition in children. There has been some suggestion that stunted children are more likely to have increased central body fat compared with normal height children and some studies have demonstrated this phenomenon. A summary of studies on stunting and fat distribution in children is given in Table 2.2. Cameron *et al.* (2005) noted that most of the studies on stunting and obesity in children are cross-sectional and thus do not follow the same children through childhood and adolescence. Observations on growth changes and body composition can thus only be inferred through implication and not measured within the same children. A good indication of how stunting affects fat distribution in children will be to follow the same children and see how fat distribution will change over time. It is possible that the link between stunting and obesity is biological in origin (Popkin *et al.,* 1996). Physiological mechanisms that substantiate the

association between previous undernutrition and future obesity have been suggested based on observations from studies on stunting and energy balance. The majority of the alterations that occur seem to work towards energy conservation and maintenance of low metabolism (Waterlow, 1994). Sawaya *et al.* (2003) reviewed studies on stunting and risk of obesity. This review proposes evidence of a series of physiological alterations in stunted individuals which provide possible explanation of why stunted children are more likely to become obese later in life. These alterations include the following:

i) Effects on body fatness and growth velocity

There is some evidence for a disproportionately greater replenishment of body fat stores than body protein stores during the period of catch-up growth in infants and children recovering from undernutrition (Cameron *et al.*, 2003). One possible explanation for this preferential fat deposition in detriment of protein is the lower cost for fat deposition relative to protein. In addition, the rates of weight gain in infants and children recovering from undernutrition are very high: typically 5-15 times greater than the usual mean rate of gain in well-nourished children (Sawaya *et al.*, 2003). These high rates of weight gain are necessitated by the fact that slow rates of gain delay recovery with the possible consequence of cognitive impairment, prolong expensive treatment, and encourage the continuation of opportunistic infections that can cause further problems (Sawaya *et al.*, 2003).

Table 2.2: Summary of studies on stunting and obesity development

Authors	Aim	Sample size and study location	Study design	Results	Conclusions
Sawaya *et al.*, 1995	Investigate the prevalence of obesity and malnutrition in the poor Brazilian population.	2411 individuals in Sao Paulo, Brazil.	Cross-sectional	Overweight and obesity associated with stunting was found in 5.8% of boys and 6.8% girls. Obesity associated with stunting was more common than obesity without stunting, both in younger children and adolescents.	A high prevalence of obesity was associated with stunting.
Schroeder *et al.*, 1999	Determine the association between poor growth in utero or young childhood and adult abdominal fatness.	372 girls and 161 boys in Guatemala	Prospective study (children measured in 1968 & 1977 and measured again as adults in 1988 & 1989. In 1991-1994 only women measured.	In both sexes, severely stunted children had significantly greater adult abdominal fatness (WHR) as adults.	Abdominal obesity and related chronic disease were likely to increase where maternal and child malnutrition existed alongside with economic development and urban migration
Sicheiri *et al.*, 2000	Test the hypothesis that undernutrition early in life is associated with chronic diseases and obesity among adults.	1749 women and 1444 men, Rio de Janeiro, Brazil.	Population based survey.	Short stature was associated with the risk of abdominal fatness only among women.	Racial and socioeconomic conditions, energy intake or age at menarche did not explain increased obesity and abdominal fatness among short women .
Florencio *et al*., 2001	Analyze anthropometric profile & investigate the hypothesis of a co-existence of undernutrition and obesity in a very low income population.	1247 individuals in the outskirts of Macieo, Brazil.	Cross-sectional	Among children aged 10 the prevalence of wasting, stunting and wasting plus stunting was 3.8, 8.3 & 8.7% respectively. Wasting was most prevalent among adolescents with a prevalence of stunting & obesity seen among girls.	A clear co-existence of high levels of stunting and obesity was found.

Authors	Aim	Sample size and study location	Study design	Results	Conclusions
Walker *et al.*, 2001	Determine the effects of birth weight and early childhood stunting on BMI, body fat and fat distribution.	116 stunted and 190 non stunted children at ages 7 and 11 in Kingston, Jamaica.	Prospective cohort study.	SSF/TSF ratio was significantly greater at age 7 in stunted children & stunted group had significantly lower BMI and percentage body fat at 7 years than the non stunted group.	Children stunted in early childhood had less fat and lower BMI than the non stunted children but had a more central fat distribution.
Cameron *et al.*, 2005	Determine whether African urban children stunted at 2 years demonstrated an altered body composition by the end of childhood before puberty at 9 years of age.	330 prepubertal children from Soweto-Johannesburg, South Africa.	Mixed longitudinal study	There were no differences in their BMI or centralization of body fat. The odds ratio for stunting at 2 years as a predictor of overweight at 7 to 9 years was not significant.	Study failed to demonstrate that central fat accumulation is evident before the initiation of puberty in stunted children.
Hoffman *et al.*, 2007	Determine whether central fat distribution varies between growth retarded children and normal height children from the same impoverished communities.	25 stunted and 25 normal height children.	Prospective study.	Stunted children had increased truncal fat mass and increased % truncal fat mass independent of fat. Also, a significantly greater change in truncal fat mass independent of fat mass, gender and Tanner stage was observed.	Stunted children are more likely to deposit fat centrally when entering puberty, a significant risk factor for chronic diseases.
EL Taguri *et al*., 2009	Assess association between stunting and overweight in children under 5years from 5 Arab countries.	Lybia-7232, Syria-5454, Morocco-5380, Djibouti-1538 & Yemen 10924.	National surveys.	Prevalence of stunting and overweight differed between countries and between urban and rural areas.	More intra-country variation in the risk than inter country

BMI = body mass index

Stunting = low height-for-age

SSF/TSF ratio = subscapular skinfold/triceps skinfold ratio

WHR = Waist-Hip ratio

ii) Effects on energy balance and fat oxidation

A number of investigations have suggested that both excessive energy intake to actual requirements and low energy expenditure can play important roles in facilitating excessive body energy storage in both children and adults (Jequier, 1993; Saltzman & Roberts, 1995). There remains controversy over the extent to which hyperphagia and low energy expenditure are direct causes of body energy gain as well as being responses to underlying signals that drive energy regulation. Hoffman *et al*. (2007*)* describe two mechanisms that might be part of the causal pathway of stunting and obesity development. First, it is possible that an adaptive response of limiting fat oxidation is developed during periods of energy restriction (in utero and in early childhood). The result is that during periods of adequate energy, availability of energy will favour fat storage over utilization and the central region would be a highly desirable site for storage because it can be quickly mobilized in times of need. Unfortunately, for many growth retarded persons, this adaptive mechanism may only result in excess fat deposition that promotes chronic disease rather than providing a safety mechanism for famines that never return (Hoffman *et al.,* 2007). Another possible mechanism is the metabolism of cortisol, a stress hormone. In response to stress, some people lose weight whereas others gain. It has been suggested that stress exaggerates diet induced obesity through a peripheral mechanism in the abdominal white adipose tissue that is mediated by neuropeptide Y (NPY). Stressors lead to a release of NPY from symphathetic nerves, which in turn upregulates NPY and its receptors in a glucocorticoid-dependent manner in the abdominal fat. This positive feedback response leads to the growth of abdominal fat (Kuo, 2007). Release of NPY and activation of its receptors stimulates angiogenesis, macrophage infiltration and the proliferation and differentiation of new adipocytes, resulting in abdominal obesity. This interaction could certainly translate into increased central fat deposition (Hoffman *et al.,* 2007).

In South Africa about 2.3 million children suffer from undernutrition (Labadarios, 2000) and the South African Government has made the amelioration of infant and child malnutrition a priority. One strategy has been the introduction of feeding schemes based at clinics, crèches, schools or soup kitchens. Despite the various national nutrition and primary healthcare efforts in South Africa over the last decade, child health has deteriorated (Bourne *et al.,* 2007). This is seen by the rise in infant and child mortality rates, the high prevalence of preventable childhood diseases,

e.g. diarrhoea and lower respiratory tract infections and the coexistence of under-nutrition along with HIV/AIDS (Bourne *et al.*, 2007). The prevalence of stunting among 1-9 year old children did, however, decrease from 1999 to 2005 in national South African studies, but the prevalence of underweight and wasting remained stable (Kruger *et al*., 2007). Poor dietary intake, food insecurity and poor quality of basic services prevail within this precarious causal web. Failure of these programmes has been in part due to the fact that the focus has been on the poor and already malnourished, therefore, they failed to address the wider social and economic causes of undernutrition. This is especially important in the South African setting, with its history of Apartheid and its present day legacy of gross inequalities (Chopra, 2003).

2.3.2 Obesity

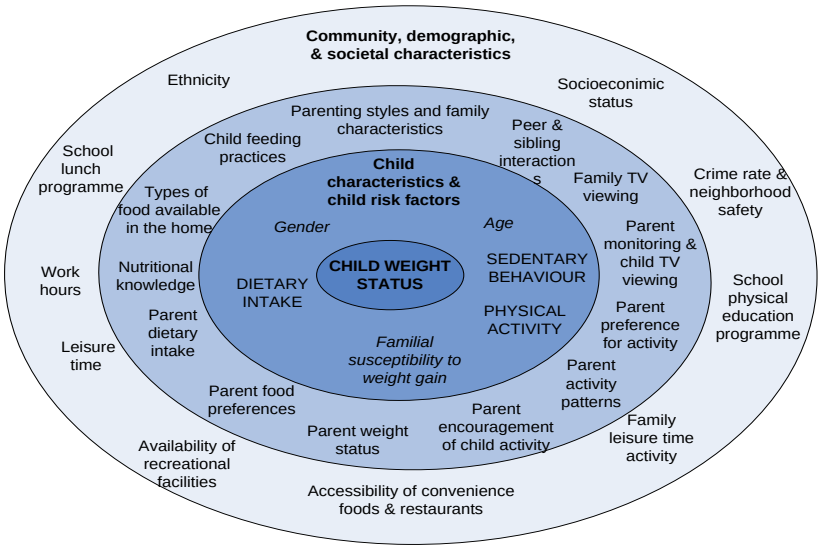
Another nutritional disorder is overnutrition mostly referred to as obesity. Obesity is a medical condition in which excess body fat has accumulated to the extent that it may have an adverse effect on health, leading to reduced life expectancy (Haslam *et al.*, 2005). Obesity is commonly defined by body mass index (BMI) and can be further evaluated in terms of fat distribution. BMI is calculated by dividing the person's weight by the square of his or her height. Although it gives an accurate reflection of body fat percentage in the majority of the adult population, there are several other methods used to determine body fat percentage, such as underwater weighing, body electrical impedance, magnetic resonance imaging, and air displacement plethysmography (BOD POD®). In children obesity has been defined as a BMI greater than the 95^{th} percentile by the Center for Disease Control and Prevention in the USA (Reilly, 2007), or a BMI above age and sex specific BMI cut-off points corresponding to an adult BMI of 30 (Cole *et al*., 2000). Many different factors have been identified as contributing to the development and the rising rates of obesity worldwide. Adair (2008) stated that obesity is a multifactorial condition, with many biological, genetic, social and environmental influences. The following section gives an overview of the developmental perspective on child obesity that considers the nature of biological susceptibility at different stages of growth.

Theories of obesity development

No single theory on the development of childhood obesity is universally accepted and research is now turning to complex models such as the ecologic systems theory (Davison *et al.*, 2001), the society-behaviour biology nexus (Glass *et al.*, 2006) and the concept of early programming (Lucas, 1991). A brief description of these is presented.

i) Ecological Systems Theory (EST)

The Ecological Systems Theory (EST) conceptualizes human development from an interactive contextual perspective (Davison *et al.*, 2001). According to the EST, development, or change in individual characteristics, cannot be explained effectively without consideration of the context, or the environment in which the person lives, but also the contexts in which that context is situated. According to the EST, development occurs as a result of interactions within and among the various contexts; that is, characteristics of the child interact with processes in the family and the school, which themselves are influenced by characteristics of the community and society at large. The application of the EST to predictors of childhood overweight is illustrated in Figure 2.3. In the ecological model the environment, including societal characteristics and parenting skills overlay the more immediate childhood behaviours, such as diet, physical activity and sedentary behaviours (Davidson *et al.*, 2001).



(Chid risk factors are given in upper case, while child characteristics are given in lower case).

Figure 2.3: Ecological model of childhood overweight predictors (Davidson *et al*.,2001).

ii) The society–behaviours–biology nexus

A graphic illustration of the society–behaviours–biology nexus is given in Figure 2.5. The society–behaviours–biology nexus incorporates the ecologic models and adds an emphasis on cross-level interactions that influence biological processes (Stovitz, 2008). According to this model, ground-level social conditions existing in schools, neighbourhoods and homes (such as cultural norms, area deprivation, laws and policies, and the local food environment) act as risk regulators that influence two key health behaviors, feeding and physical activity dynamically and over the life course. Changes in these behaviours and the relative balance of energy intake and output are the primary causes of change in body weight. They are also the primary mediators through which risk regulators exert contingent effects on body weight. Also cross level interactions are suggested, whereby risk regulators alter biological factors underwater (HPA axis response, mood, appetite, metabolism, gene expression), which in turn directly affect health behaviours (Fig. 2.4). Several important feedback loops are hypothesized, which imply that body weight change alters the influence of biological control parameters.

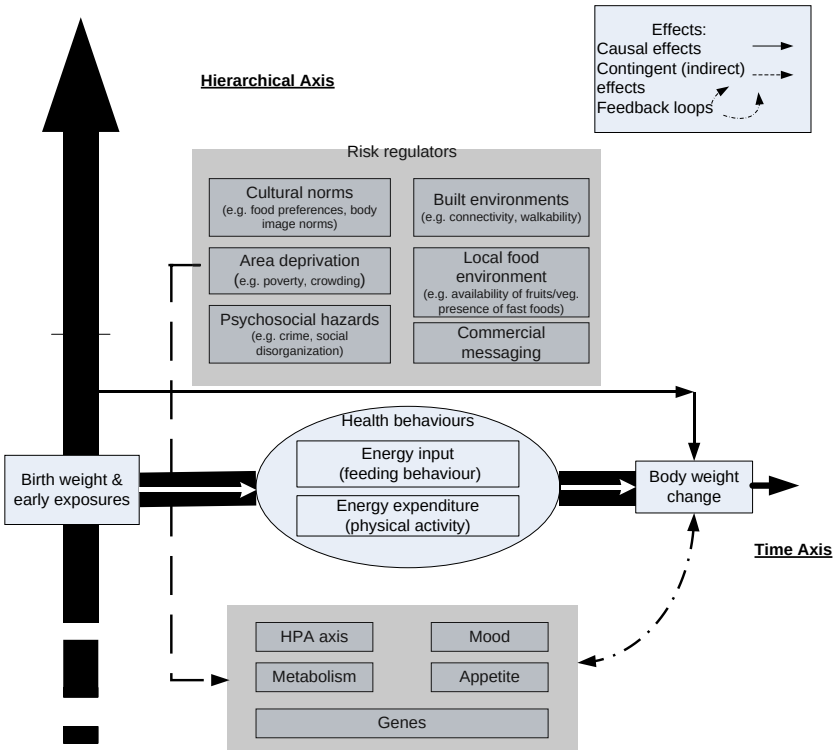


Figure 2.4: The society–behaviours–biology nexus (Glass *et al.*, 2006).

iii) Early life programming

Another theory on development of obesity later in life has been the concept of early programming. According to this theory, during early ontogeny the developing organism passes through critical windows of sensitivity or plasticity, during which environmental factors generate long lasting variability in phenotype (Lucas, 1991). Foetal programming proposes that the adaptation which the foetus makes during periods of less energy intake and availability permanently change the function and the structure of the body in adult life. It may represent a further potentially important mechanism that could contribute to the development of obesity (McMillen *et al.*, 2004). Evidence supporting early programming during foetal life has been demonstrated in epidemiological studies. Highlighted in the next section are the three main sources of this evidence.

- *Prenatal experiences*

Maternal metabolic control during pregnancy

Rodriguez *et al.* (1998) demonstrated that maternal type 1 and gestational diabetes were associated with increased risk of obesity in the offspring. The influence of type 2 diabetes seems quite complex and it is thought that it might involve either a programming mechanism or genetic transmission affectionately referred to as *thrifty genotype hypothesis*. Studies on an Indian population, which is characterised by high rates of disease, have clearly differentiated between these pathways. Within a pool of mothers who at some stage developed type 2 diabetes, infants born after their mothers were diagnosed had a greater risk of developing obesity relative to those born before mothers were diagnosed. Dabalea *et al.* (2001) hence stated that the intrauterine diabetic environment is thus associated with an increased risk of obesity in both childhood and adulthood, independently of genetic factors and effects on birth weight.

In-utero malnutrition

Sayer *et al.* (2009) described an association between maternal malnutrition and obesity in the next generation in a study that followed children born to women after the Dutch Winter Famine at the end of the Second World War. At the age of 19 years a long term effect on obesity was observed depending on the timing of exposure to famine. Famine exposure during the first two trimesters of gestation led to children born to be twice as likely to be obese as those not exposed,

whilst those exposed in the last trimester had a 40% less chance of being obese. At the age of 50 years early exposure to famine was no longer associated with obesity in men but was related to obesity in women. These findings suggested that severe nutritional deficits occurring in critical periods in foetal life may have far reaching outcomes for long term body composition even without a direct effect on birth weight.

Gestational age at birth

It is well established that preterm infants have low levels of body fat at birth (Rigo *et al.,* 1998). This could mean that fat deposition occurs largely during the last trimester of pregnancy and since these children miss this period in the uterus they do not get the opportunity of depositing fat. Body composition studies using MRI have, however, suggested that by term-age preterm babies have a more central adipose tissue distribution (Uthaya *et al*., 2005).

- *Postnatal experiences*

A systematic difference has been observed on the effect of infant weight gain on later body composition between industrialized and developing countries (Wells *et al.,* 2007). Studies in Europe have demonstrated that greater infant weight gain predicted height, weight, and lean mass, fat mass and waist circumference in late adolescence (Euser *et al*., 2005; Ekelund *et al.,* 2006). Contrary to this, studies from non Western populations showed that infant weight gain was associated with later weight, height and lean mass but not fat mass (Li *et al.,* 2003; Wells *et al.,* 2007). This disparity suggests that infant weight gain directed to lean mass or fat mass is mediated by the disparity between size at birth and genetic potential (Wells *et al*., 2007). A possible explanation for these observations is that individuals from industrialized populations have probably reached their genetic potential for energy balance mechanisms at birth and are, therefore, unable to translate increased energy intake into greater lean mass and hence store excess energy as fat. On the other hand, from developing nations individuals are on average small at birth and, therefore, have greater capacity to direct additional energy directly to reducing shortages in lean mass. Inconsistent results have been observed as to whether catch up growth in small for gestational age infants is associated only with later fatness in the first year of life or beyond. Ezzahir *et al*. (2005) and Ibanez *et al.* (2006) demonstrated that weight gain following low birth weight programs development of lean mass and stops within the first year of life. On

the contrary the Avon longitudinal study showed that catch up growth was associated with both indices of growth retardation and central adiposity at 5 years (Ness, 2004). Based on these findings it then seems that the impact of post natal growth on later body composition is not restricted to the infant period. Overall, therefore, growth patterns in both infancy and childhood contribute to the risk of obesity (Wells *et al*., 2007).

iv) Supplementary nutrition programmes

Supplementary nutrition programmes particularly in developing countries may be contributing to increasing overweight prevalence being seen among previously undernourished populations (Chakraborty & Anderson, 2010). Some agencies give food aid to developing nations in an attempt to assist the food insecure people in those nations. Some of these programs have been associated with overnutrition among children. For example two major food assistance programs in Latin America were associated with overnutrition in children below the age of 6 years (Uauy *et al*., 2001). It is possible that while trying to reduce the burden of undernutrition such programmes predispose children to excess weight gain. Therefore, nutrition programmes need to be designed and well targeted to reduce the prevalence of undernutrition while preventing the onset of overweight in children (Chakraborty & Anderson, 2010). The programmes have evolved beyond the immediate needs of the malnourished and have potential to increase the obesity epidemic in developing countries (Misra & Khurana, 2008). Mason and colleagues (2006) argue that supplementary feeding, using external supplies may sometimes be appropriate in emergencies and in conditions of extreme poverty but otherwise it is to be avoided as costly, with high opportunity cost, and not very effective; moreover, it can distort programmes, which come to be seen largely as a source of free food.

As noted in the vast amount of literature published over the past decade obesity is becoming one of the most alarming public health issues facing the world today. This is because of the escalating trends in its prevalence, severity and the occurrence of adverse health outcomes particularly among children. In the UK, child obesity rates doubled or tripled from 1974-2002 (Stamatakis *et al.,* 2005), and in many countries including the US, Australia, China and Brazil child overweight is increasing at a faster rate than adult obesity (Popkin, 2006*).* To highlight the recent trends in the prevalence of obesity in 2004, 26% of Canadian children and adolescents were overweight or obese (Shields, 2004), whilst results from the 2003-2004 National Health

and Nutrition Examination Survey (NHANES) indicated that an estimated 17% of children and adolescents aged 2-19 years in the USA were overweight. The same report also indicated that overweight increased from 7.2% to 13.9% among 2-5 year olds and from 11% to 19% among 6-11 year olds between 1988-94 and 2003-2004 (Ogden *et al*., 2006). Bibbins-Domingo *et al.* (2007) used USA government statistics and other data to project that by the time those who are adolescents in 2007 turn 35 in 2020 up to 37% of men and 44% of women will be obese, resulting in an additional 100,000 cases of heart disease by 2035. Després (2003) describes these trends as only showing the tip of the iceberg since they offer a very modest estimate of the magnitude of the problem considering the fact that obesity has adverse health implications both in the short and the long term.

South Africa has not been spared from the rampant increase in childhood obesity. The National Food Consumption Survey (NFCS) done in 1999 reported that 17.1% of South African children between 1-9 years living in urban areas are overweight (Steyn *et al.*, 2005). Another study conducted in 5 different South African provinces on children aged 3-16 years found the prevalence of obesity to be 3.2% for boys and 4.9% for girls, whereas the prevalence of overweight was 14.0% for boys and 17.9% for girls (Armstrong *et al*., 2006). After adjusting for the contribution of each ethnic group to the demographics of South Africa values were slightly different. The prevalence of obesity and overweight among boys was 2.4% and 10.9% respectively, while obese and overweight girls comprised 4.8% and 17.5% respectively. They concluded that South African children show trends of obesity and overweight, similar to values in developed countries about 10 years ago. Kruger *et al. (*2006) conducted a study in the NorthWest Province of South Africa and found that 7.8% of children were overweight when using the International Obesity Task Force standards. In an earlier study Monyeki *et al.* (1999) found that the prevalence of obesity and overweight in rural children aged 3-10 years in the Limpopo Province was low, 0-2.5% and 0-4.3% in boys and girls respectively. Although differences in the prevalence of childhood obesity have been reported for South African children from different regions (as illustrated by the above mentioned studies), there is sufficient evidence that childhood obesity does exist. The prevalence of combined overweight and obesity among high school children increased from 21.2% in 2002 (MRC, 2002) to 25% in 2008, with 35% of high school girls being overweight or obese (Reddy *et al*., 2010).

Finally common nutritional disorders that contribute significantly to adverse health outcomes later in life have been highlighted. Every year, thousands of children die from undernutrition, millions more survive only to face diminished lives, unable to develop to their full potential. On the other side of the spectrum obesity is rising in epidemic proportions. Children who are obese face the risk of being overweight in adulthood and thereby increasing their risk of chronic diseases. The fact that the two common nutritional disorders co-exist in most parts of the world has serious implications for developing countries, particularly those undergoing rapid nutritional transition, as it may further increase the rates of obesity, cardiovascular disease (CVD) and diabetes when diets and lifestyles are in themselves "atherogenic" (Delisle, 2002). The challenge, therefore, is for programmes to simultaneously combat apparently opposite nutrition problems, undernutrition and over-nutrition. The knowledge and capacity to prevent and treat the causes exist. Therefore, the eradication of nutritional disorders particularly those affecting women and children must remain a high priority. The reasons for this are obvious, their effect on mortality and human capital remain significant and if the foetal programming hypothesis proves to be correct, the elimination of foetal and infant undernutrition will contribute to the amelioration of non-communicable diseases (Bowman & Russell, 2001).

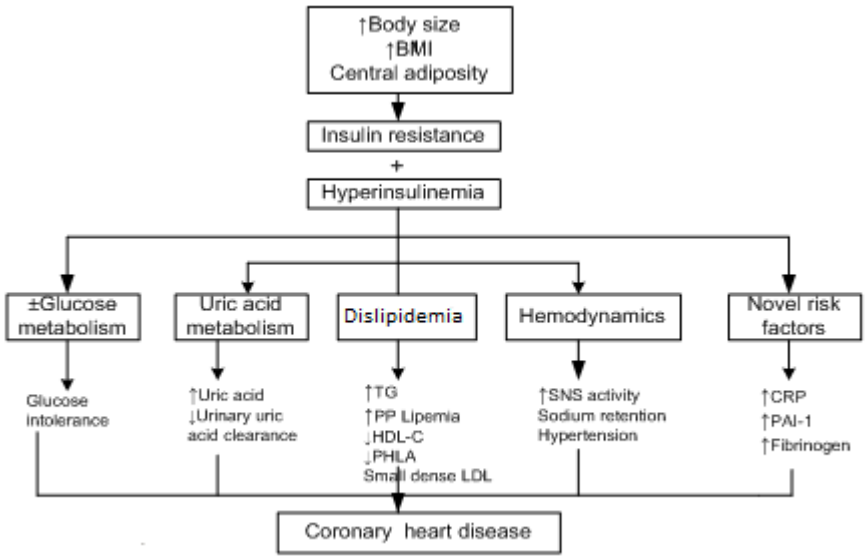
2.4 HEALTH EFFECTS OF EXCESS ABDOMINAL FAT

Although total amount of body fat as estimated by the BMI remains an important indicator of weight-related illness, location of body fat is equally if not more significant. To be specific, abdominal obesity, especially visceral adipose tissue (VAT) increases the incidence of a cluster of metabolic disturbances, the so-called metabolic syndrome (Scheen, 2008). Hamdy *et al*. (2006) stated that 'obesity, and in particular abdominal obesity, plays a major role in the pathogenesis of several metabolic and cardiovascular medical problems including type 2 diabetes, hypertension, atherosclerosis and coronary artery disease'. Several studies have shown that the detrimental influence of abdominal obesity on metabolic processes is mediated by intra-abdominal fat depots *(*Wajchenberg, 2000*).* Due to its anatomic location and peculiar metabolic, hyperlipolytic activity, the expanded visceral adipose depot is a key correlate of the altered metabolic risk profile observed among individuals with a high-risk abdominal obesity phenotype

(Després, 2006). This cluster of metabolic disturbances, namely type 2 diabetes and associated cardiovascular risk appears to be a causal factor for morbidity and premature mortality.

2.4.1 Metabolic syndrome (MS)

More than 2 decades ago the concept of "syndrome X" was introduced to describe a cluster of metabolic abnormalities that included obesity, high triglyceride concentration, reduced HDL-C concentration, hypertension, and glucose intolerance (Al-Mubaslat, 2005). Since then, the understanding of the pathogenesis and complications of this syndrome have evolved tremendously. This is reflected in the names given to the syndrome throughout this evolution, including the dysmetabolic syndrome, the deadly quartet, the insulin-resistance syndrome, and, more recently, the metabolic syndrome. The latter name has gained acceptance worldwide (Al-Mubaslat, 2005). The central features of the MS are insulin resistance, visceral adiposity, atherogenic dyslipidemia and endothelial dysfunction (Huang 2009). These conditions are interrelated and share common mediators, pathways and physiological mechanisms. Després (2006) summarized the MS as illustrated in Figure 2.5.



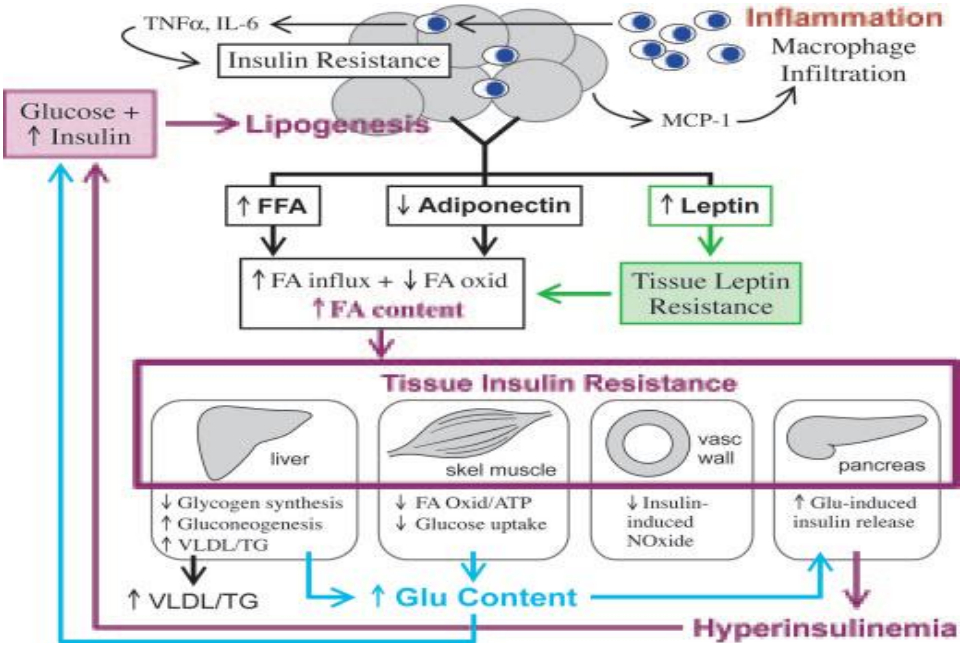
Key:

BMI- body mass index
PP- post prandial
PHLA-postheparin lipolytic activity
SNS- symphathetic nervous system
PAI-1-plasminogen activator inhibitor
TG- triglycerides
HDL-C- high density lipoprotein
LDL- low density lipoprotein
CRP- c-reactive protein

Figure 2.5: Current Perspective of the Metabolic Syndrome (Després, 2006).

The pathophysiology of MS is extremely complex and has been only partially elucidated. There is debate regarding whether obesity or insulin resistance is the cause of the metabolic syndrome or if they are consequences of a more far-reaching metabolic derangement. Regardless, the concept of a clustering of risk factors leading to adverse health outcomes is well accepted. Unger (2003) described MS as failure of a system of intracellular lipid homeostasis which prevents lipotoxicity in organs of overnourished individuals. When this system breaks down the results are exogenous fuel overload, ectopic accumulation of lipid in non adipose cells and insulin resistance (McKeown *et al.*, 2004).

Obesity has been identified as one of the root causes of MS (NCEP, 2002). Bagby (2004) described the possible mechanisms underlying the development of MS and stated that 'events leading to the development of MS do not follow a linear sequence but occur along a matrix of interconnected pathways that mediate interactions among multiple organs and also link these organs as a functional unit to regulate total energy homeostasis'. It is further stated that disturbances may occur at any of these multiple sites due to stimuli acting on the sites for example in adipocytes, hepatocytes or skeletal myocytes and that each of these has the capability of disturbing the whole energy balance mechanism. Bagby (2004) argues that, therefore, the initiation of MS by obesity is characterised by powerful systemic stimuli that together impair energy homeostasis in multiple organs simultaneously leaving no room for protective compensation. These complex pathways are shown in Figure 2.6.



Key: **TNF-α-Tumor necrosis factor-alpha** **IL-6-interleuken-6**
MCP-1- Monocyte chemoattractant protein-1 **FFA-free fatty acid**
FA-fatty acid **VLDL-very low density lipoprotein**
TG- triglyceride **ATP-adinosine triophosphate**
NO-nitrogen oxide

Figure 2.6: Illustration of the complex pathways of obesity initiated metabolic syndrome (Bagby, 2004).

The prevalence of the MS has been rising with the rising childhood obesity worldwide. In Western countries, the incidence of childhood obesity has more than doubled over the past generation, as a consequence the metabolic syndrome and type 2 diabetes mellitus is rapidly increasing in the paediatric population (De Ferranti *et al.*, 2004). Kelishadi (2007) reviewed literature from 1950-2007 and summarized the prevalence of MS from various studies in both developed and developing countries. His summary and other studies summarized highlight the prevalence of metabolic syndrome in some parts of the world (Table 2.3). These studies have shown the presence of MS in early childhood and an increased prevalence in overweight and obese individuals, and this poses a serious problem as childhood MS is thought to persist through adulthood (Isomaa *et al.*, 2001). MS is also observed in normal-weight individuals and those are considered to be metabolically obese normal-weight (Ruderman *et al.*, 1998). The few studies that have been conducted in developing countries have shown a relatively high prevalence of MS

that is paralleled by the increasing obesity in children and adolescents (Kelishadi *et al.,* 2006). It should be noted that the different studies defined MS differently but irrespective of the definition used it is quite evident that there is an increasing problem.

The variations in the prevalence by different definitions were analyzed by Reinehr *et al.* (2007). The prevalence of MS according to eight different definitions was studied in 1205 Caucasian overweight children and adolescents aged 4 to 16 years. Insulin resistance was estimated based on the homeostasis model assessment–insulin resistance (HOMA) model. The prevalence of MS varied significantly ($p < 0.0001$) between 6% and 39% depending on the different definitions used. Only 2% of the children fulfilled the MS criteria in all different definitions. This led to the conclusion that since the prevalence of MS varies widely between the different proposed definitions, an internationally accepted uniform definition of MS is necessary to compare different populations studied.

Table 2.3: Summary of studies on MS showing different prevalence rates among children

Authors	Study site and population	Results
Csábi *et al*, 2000	Caucasian children aged 8–18 years, 77 female & 103 male. Hungary	The MS was detected in 8.9% of the obese children (84 female, 155 male) & on 0.4% of the controls.
Cook *et al*. 2003	2430 United States children aged 8-18 years (representative sample).	Prevalence of MS was 4.2% overall and was present in 28.7% of overweight adolescents.
de Ferranti, *et al.*, 2004	1960 United States children aged ≥12 years.	2/3 had at least one metabolic abnormality & nearly 1/10 had MS.
Lambert, *et al.*, 2004	2,244 children and adolescents aged 9 years from Canada.	An MS prevalence of 11.5%, with no age- or sex-specific difference.
Cruz, *et al*., 2004	126 obese Hispanic children aged 8–13 years in the United States.	The MS was present in 30% of obese children.
Yoshinaga, *et al.*, 2005	471 overweight and obese Japanese children aged 6-11 years.	The prevalence of MS was 17.7%.
Agirbasli *et al*. 2006	1,385 Turkish adolescents aged 10–17 years.	2.2% of adolescents had MS. The syndrome was 10 times more common among overweight and obese (21%) than among lean children.
Esmaillzadeh *et al.*,2006	3,036 Iranian adolescents aged 10–19 years.	The prevalence of MS was 10.1%
Kelishadi, *et al*., 2006	Iran national survey of 4,811 children and adolescents.	The prevalence of the MS was 14.1% with no difference between boys and girls.
Singh, *et al.*, 2007	1083 school going adolescents aged 12–17 years in India.	The prevalence of MS was 4.2% and increased to 5.8% when plasma fasting glucose cut-off was lowered to 5.5mmol/L.
Kim, *et al*., 2007	South Korean adolescents in 1998 and 2001 aged 12–19 years (1,317 in 1998 and 848 in 2001).	Overall prevalence of MS increased from 6.8% in 1991 to 9.2% in 2001.
Li, *et al.*, 2008	1569 school children aged 13-18 years in Tunisia.	The overall prevalence of MS was 0.4% with no statistical difference between boys and girls.
Messiah, *et al.*, 2009	224 overweight 1st and 2nd generation US immigrant from central / south America and Caribbean basin aged 3-18 years.	The prevalence of MS was 29% overall.
Matsha, *et al.*, 2009	1272 learners aged 10–16 years in South Africa.	The prevalence of MS was 6.5% overall.

MS-Metabolic Syndrome.

Despite the high prevalence of obesity reported in South African children (Armstrong *et al.*, 2006; Reddy *et al*., 2010), there is limited data on the prevalence of MS in children. However,

Matsha *et al.* (2009) did a study to identify South African children from different racial groups with the MS using the National Cholesterol Education Programme, Adult Treatment Panel (NCEP ATP III) or the International Diabetes Federation (IDF) definitions, and determined the proportion of normal-weight children that are metabolically obese. The sample size for this study was 1272 children aged 10–16 years that were recruited through a proportionally stratified multistage random sampling technique from government funded primary and secondary schools using a list of 107 schools obtained from the Western Cape Education Department. The NCEP ATP IIII and IDF for children 10–16 years old were used to define MS. Results of this study showed that only 1.9% of the children were identified with MS using the IDF definition. MS was more prevalent in obese children using either the NCEP ATP III (30.8%) or IDF (13.2%) (p=0.002). In normal-weight children the IDF definition underestimated MS, 0.1% versus 2.4% by NCEP ATP III (p<0.001), whereas of the 83 (6.5%) children who were identified with the MS using the NCEP ATP III definition, 24 (28.9%) were of normal-weight.

It should be noted that the variation in prevalence in different studies may be because of a lack of universally accepted criteria defining the syndrome. Therefore, in comparing studies caution must be taken to consider the definition of MS used. The task of arriving at a universal definition for the MS has been challenging. Much debate exists as to what elements need to be included and which cut-off points should be used by researchers to identify the condition more easily and make it possible to compare its prevalence in different parts of the world (Al-Mubaslat, 2005). In 1998 the World Health Organization (WHO) presented its initial definition of the metabolic syndrome (Table 2.4). The name of the syndrome was changed to metabolic syndrome primarily because central obesity was not included in the original description by Reaven (1988). It is based on the assumption that insulin resistance is one of the major underlying contributors to the metabolic syndrome, and features impaired glucose regulation (impaired glucose tolerance, diabetes or insulin resistance) at its core. It is important to take note of two facts in the WHO description:

- Each component of the syndrome conveys increased cardiovascular risk, but as a combination they become much more powerful.
- The features of metabolic syndrome may be present for up to 10 years before detection of glycaemic disorders.

Following the publication of the WHO definition in 1999, the European Group for study of Insulin Resistance (EGIR) proposed a modified version to be used in non-diabetic subjects only. The group was interested in insulin resistance and this became the cornerstone of this definition**.** EGIR proposed the use of fasting insulin levels to estimate insulin resistance and impaired fasting glucose (IFG) as a substitute for impaired glucose tolerance (Alberti, 2006).

The International Diabetes Federation (IDF) also defined criteria for diagnosing metabolic syndrome (Zimmet *et al.*, 2007). This definition includes the same general criteria as the other definitions, but requires that obesity, and not necessarily insulin resistance, be present. The obesity requirement is met by population-specific cut points. This accounts for the fact that different populations, ethnicities and nationalities have different distributions of norms for body weight and waist circumference. It also recognizes that the relationship between these values and the risk for type 2 diabetes (T2D) or cardiovascular disease (CVD) differs in different populations. For example, South Asian populations have an increased risk for T2D and CVD at smaller waist circumferences that would not be considered to meet the criteria in a Western population. The IDF definition has been criticized for its emphasis on obesity, rather than insulin resistance, in the pathophysiology of MS (Huang, 2009).

The 2001 NCEP ATP III guidelines defined metabolic syndrome differently (see Table 2.4). Fasting plasma glucose rather than 2-h post-load glucose value and waist circumference instead of waist-hip ratio (WHR) and BMI are included. The cut-off values for HDL-C and hypertension are different and microalbuminuria is not included as one of the criteria. Waist circumference is simpler to measure, culturally more acceptable and is a better indicator of central obesity compared to waist-hip ratio (Montague *et al*, 2000). It is open to debate whether fasting plasma glucose measurement is as sensitive as 2-h value during 75g oral glucose tolerance test (OGTT) in identifying glycaemic disorders in the Asian population (Qiao *et al.*, 2000). Irrespective of the definition used to define MS in children, investigators have shown the presence of MS in early childhood and an increased prevalence in overweight and obese individuals, and this poses a serious problem as childhood MS is thought to persist through adulthood (Isomaa *et al.*, 2001). The rapid rise in obesity trends underlines the urgency for a definition that could be used to know who is at high risk of health problems and to differentiate them from those with uncomplicated obesity.

Table 2.4: Different definitions of metabolic syndrome

	WHO (1998)	EGIR (1999)	IDF (2005)	NCEP ATP III 2005 (revision)
Absolutely required	Insulin resistance*(IGT, IFG,T2D or other evidence of IR).	Hyperinsulinemia[#] (plasma insulin > 75th percentile).	Central obesity(WC[Ψ] : ≥94 cm(M) ≥80 cm (F)	None
Criteria	Insulin resistance or diabetes, plus 2/5 criteria below.	Hyperinsulinemia, plus 2/4 criteria below.	Obesity plus 2/4 criteria below.	Any3/5 criteria below.
Obesity	WHR: > 0.90 (M), >0.85 (F); or BMI > 30kg/m^2.	WC: ≥94 cm(M), ≥80 cm (F)	Central obesity already required.	WC: > 40 inches (M), > 35 inches (F).
Hyperglyceamia	IR already required.	IR already required.	Fasting glucose ≥ 100 mg/dl	Fasting glucose ≥ 100 mg/dl or Rx.
Dyslipidemia	TG ≥150mg/dl or HDL-C < 35mg/dl (M), < 39 mg/dl (F).	TG ≥177mg/dl or HDL-C < 39 mg/dl.	TG ≥150mg/dl or Rx.	TG ≥150mg/dl or Rx.
Dyslipidemia (second, separate criteria).	-	-	HDL-C: < 40 mg/dl (M), <50 mg/dl (F); or Rx.	HDL-C: < 40 mg/dl (M), <50 mg/dl (F); or Rx.
Hypertension	≥ 140/90 mmHg	≥ 140/90 mmHg or Rx.	> 130mmHg systolic or > 85 mmHg diastolic or Rx.	> 130mmHg systolic or > 85 mmHg diastolic or Rx.
Other criteria	Microalbuminuria[T]			

*IGT=Impaired glucose tolerance, IFG=Impaired fasting glucose, T2D=Type 2 diabetes
[T] Urinary albumin secretion of ≥ 20μg/min or albumin to creatinine ratio of ≥ 30μg/g
[#] Reliable only in patients with T2D
[Ψ] Criteria for central obesity (waist circumference) are specific for each population: values given are for European male and females
IR= Insulin resistance: M=male; F=female, WC-Waist circumference, WHP= Waist-Hip ratio
Rx, pharmacologic treatment (Huang, 2009)

Of the various definitions for the MS Huang (2009) argues that the NCEP ATP III definition is the easiest to apply clinically and epidemiologically, because it uses straightforward criteria that are measured readily. He went on to state that 'it is one of the most widely used criteria of metabolic syndrome, incorporating the key features of hyperglycemia/insulin resistance, visceral obesity, atherogenic dyslipidemia, hypertension and uses measurements and laboratory results that are readily available to physicians, facilitating its clinical and epidemiological application'.

It is also simple and easy to remember. Importantly, it does not require that any specific criterion be met; only that at least three of five criteria are met. Thus, the definition does not build in any preconceived notion of the underlying cause of MS.

Although overall obesity is the most common cause of metabolic complications such as insulin resistance in children and adolescents, some obese youth have been found to be very insulin sensitive and thus at reduced risk for the development of the adverse cardiovascular and metabolic outcomes driven by insulin resistance. In a study aimed at discovering the underlying pathophysiology of altered glucose metabolism in obese children and adolescents, it was demonstrated that individuals with IGT were significantly more insulin resistant than individuals with normal glucose tolerance, despite having an overall equal degree of adiposity (Weiss *et al.,* 2003*).* The difference in insulin sensitivity was attributed to different patterns of lipid partitioning. Individuals with severe insulin resistance were characterized by increased deposition of lipid in the visceral and intra-myocellular compartments. Brambilla *et al. (*1994) stated that 'in children, visceral fat has been shown to be positively related to a wide range of health indicators, including total cholesterol, low-density lipoprotein cholesterol, triacylglycerol, insulin area under curve after an oral glucose test, basal insulin secretion, and stimulated insulin secretion'.

The last several years have witnessed a heightened interest in the MS, as it has become a health issue of epidemic proportions (Al-Mubaslat, 2005)**.** Hence much research has been conducted which gives evidence on the link between central fat and the various components of MS in human beings. Several metabolic abnormalities emerge as key players in the pathogenesis of the syndrome, including insulin resistance, obesity and inflammation. Individuals with the MS are at increased risk for a variety of clinical conditions, some with serious health implications, particularly diabetes and cardiovascular disease. There has been a well-documented pediatric obesity epidemic and a dramatic increase in clinical diseases associated with it (Choudhary *et al.,* 2007). Even though the complications of central obesity occur in nearly every organ in the body the focus of the following section will be on just three disease conditions associated with central obesity. These are diabetes, cardiac and hepatic disorders.

2.4.2 Diabetes

Childhood obesity has been accompanied by an increase in the prevalence of type 2 diabetes (Sinha *et al.,* 2002). Dr. Francine Kaufman, head of the Diabetes Centre in California Hospital, USA once said 'about 25% of the diabetic children treated there have type 2 diabetes, compared with just 4% a decade ago'. In adults, type 2 diabetes develops over a long period, and most, if not all patients initially have impaired glucose tolerance, which is an intermediate stage in the natural history of type 2 diabetes (Polonsky *et al.,* 1996). Much research has been conducted in an effort to try and understand why obesity predisposes some individuals to diabetes. One proposed explanation to this is that the cells release fat and breakdown products of fat into the blood. These products may make cells less able to respond to insulin, increasing the body's demand for the hormone. Another possible explanation is a paradoxical finding about a hormone, adiponectin, made by fat cells. Adiponectin makes cells more responsive to insulin. Adiponectin reduces blood glucose levels by increasing fatty acid combustion in myocytes (Gualillo *et al.,* 2007). Oddly enough, Dr. C. R Kahn, a diabetes researcher stated that 'the fatter people become, the less adiponectin their fat cells produce'. So one way obesity might increase the risk that a person will develop diabetes is by leading to a release of more fatty acids and a decline in adiponectin. This would lead to more insulin resistance and a demand for more insulin. If that demand cannot be met, the result, eventually, would be diabetes (Snehalatha *et al*., 2003).

Sinha *et al.* (1996) conducted a study to determine the prevalence of impaired glucose tolerance in a multiethnic cohort of 167 obese children and adolescents in the US. They measured fasting levels of proinsulin and calculated the ratio of proinsulin to insulin. Insulin resistance was estimated by homeostatic model assessment, and beta-cell function was estimated by calculating the ratio between the changes in the insulin level and the glucose level during the first 30 minutes after the ingestion of glucose. The findings were that impaired glucose tolerance was detected in 25% of the 55 obese children (4-10 years of age) and 21% of the 112 obese adolescents (11-18 years of age); silent type 2 diabetes was identified in 4% of the obese adolescents.

Williams *et al.* (2005) used data from the 1999–2000 National Health and Nutrition Examination Survey (NHANES) to examine the prevalence of IFG and its relationship with overweight and CVD risk factors in a nationally representative sample of US adolescents aged 12 to 19 years.

Prevalence of IFG was higher in overweight adolescents (17.8%) but was similar in those with normal weight and those who were at risk for overweight (5.4% vs. 2.8%). There were ethnic differences in the prevalence of IFG, and 13.0%, 4.2%, and 7% of Mexican Americans, non-Hispanic black individuals, and non-Hispanic white individuals, respectively, presented with IFG (Williams *et al.,* 2005). Lee *et al.* (2006) reviewed the results of the National Survey of Children's Health, a population-based survey involving more than 100,000 children. When taking into account the children's BMI, they found those who were obese were more than twice as likely to have diabetes as their counterparts of normal weight.

The prevalence of type 2 diabetes is increasing in Sub-Saharan Africa (Amos *et al.,* 1997). In the rapidly urbanizing and Westernizing indigenous African population of South Africa diabetes is common (Mollentze *et al.,* 1995), but has not been well characterized. As in the general population, coronary heart disease (CHD) remains rare in these diabetic Africans (4.0%) in comparison with the local white diabetic population (23%), with an apparent reversal of the usual male: female ratio (Kalk *et al.,* 2008). These differences were not fully explained by conventional risk factor analysis, but once CHD is present, the risk profiles are similar in each race (Kalk *et al.,* 2008). In contrast, among the ethnic groups in the United States, CHD is most frequent in the African-Americans, partly explained by high rates of obesity, hypertension, diabetes, and possibly the metabolic syndrome (Ninomiya *et al.*, 2004)

2.4.3 Cardiac disorders

The biological effects of obesity on the heart are quite profound and evidence suggests that the dysmetabolic profile observed in obese individuals is predictive of a substantially increased risk of CHD even in the absence of classical risk factors (Despres, 2006). Besides an altered metabolic profile, a variety of alterations in cardiac structure and function occur in the individual as adipose tissue accumulates in excess amounts even in the absence of co-morbidities. Hence obesity may affect the heart through its influence on known risk factors such as dyslipidemia, hypertension, glucose intolerance, inflammatory markers, obstructive sleep apnea/hypoventilation, and the prothrombotic state, in addition to as-yet-unrecognized mechanisms (Poirier *et al.,* 2006). On the whole, overweight and obesity predispose to or are

associated with numerous cardiac complications such as CHD, heart failure, and sudden death because of their impact on the cardiovascular system.

Convincing evidence from a number of studies has provided strong support that obesity increases the risk for CVD. The MS is well recognised as posing a major risk for CVD in adults, however, substantial evidence now indicates that this syndrome begins in childhood and, therefore, significant cardiovascular risk begins in childhood (Hanevolt *et al.*, 2004). The following section looks at studies relating obesity to cardiac disorders. Yan and colleagues (2004) examined the relationship of BMI earlier in life with illness and causes of death after age 65 years. Investigators found the risk for death due to CHD for obese patients, when compared to those of normal weight in the same risk category, was 43% higher for the low-risk group and 2.1-times higher for the moderate-risk group. They also established that obese individuals in the low-risk group, compared to those of normal weight, had a 4.2-times higher risk for hospitalization for conditions related to CHD.

Khan *et al.* (2003) investigated the relationship between microvascular function and cardiovascular risk factors in 145 normal, healthy children aged 11–14 years. Skin microvascular responses, measured using laser Doppler imaging, to iontophoresis of acetylcholine (ACh) and sodium nitroprusside (SNP), were negatively correlated with percentage body fat ($r = -0.20$, $P < 0.05$ and $r = -0.18$, $P < 0.05$, respectively). In a cross-section of healthy children, microvascular function was negatively associated with adiposity. Additionally, in a subgroup of subjects, there was a clustering of high post-feeding glucose, impaired microvascular function, increased insulin resistance and higher central fat distribution. They concluded that risk factors for adult cardiovascular disease begin to cluster in normal children, which might have important consequences for development of atherosclerosis later in life.

In another study of cardiac disorders De Jongh *et al.* (2004) examined the effects of both FFA elevation in lean women and FFA lowering in obese women on skin microvascular function. They postulated that it may constitute a pathway through which obesity increases blood pressure and decreases insulin sensitivity. Second, it may directly contribute to obesity-associated microangiopathy. Indeed, there is some evidence that measures of obesity in healthy individuals are associated with impaired microvascular function. In addition, microvascular dysfunction has

been shown to increase peripheral vascular resistance and antedate the development of hypertension, indicating a role for microvascular dysfunction in the development of hypertension (Antonios *et al*., 1999). In Rome a study by Menghetti *et al.* (2007) found a prevalence of overweight and obesity in adolescents of 31.7% and 10.3%, respectively, with a slightly higher presence of males in both cases. The whole sample showed a prevalence of hypertension of 10.1%. Systolic and diastolic BP showed a direct association with BMI and waist circumference ($P<0.01$). BMI and systolic and diastolic BP were lower in active students (>7 h a week of physical activity). Food habits were not associated with hypertension. Gami *et al.* (2007) performed a meta-analysis of longitudinal studies to assess the association between MS and incident cardiovascular events and death. Included were 37 studies comprised of 43 cohorts (inception 1971 to 1997) and 172,573 individuals. The relative risk of cardiovascular events and death was 1.78 (95% confidence interval 1.58 to 2.00). Subgroup and sensitivity analyses provided further insights into important issues regarding the cardiovascular risk associated with MS. The association remained after adjusting for individual cardiovascular risk factors. The entirety of the available evidence supports that MS is associated with an increased risk of cardiovascular events and death.

2.4.4 Hepatic disorders

Non-alcoholic steatohepatitis, popularly known as fatty liver disease, has been observed in some obese children (Sanjay *et al*., 2002). Fatty liver disease, in which fat accumulates in the liver, while not life threatening in children, can lead to cirrhosis (scarring) of the liver, sometimes requiring transplantation by adulthood. This condition has been attributed to changes in hepatic fatty acid oxidation. Reddy and Rao (2006) described three pathways of hepatic fatty acid oxidation which could explain the onset of this liver condition in children. These are:

a. β-Oxidation- this occurs in the mitochondria and its rate is regulated by carnitine palmitoyltransferase (CPT1) and the mitochondrial trifunctional protein (MPT) complex.
b. Peroxisomal β-oxidation- occurs within peroxisomes and is rate-limited by the peroxisomal L-bifunctional enzyme (L-PBE), acetyl-COA oxidase (ACO), and urate oxidase (UO)

c. Lastly ω-oxidation which occurs in the endoplasmic reticulum, and is dependent upon expression of the cytochrome enzymes CYP4A10 and CYP4A14.

Stimulation of these pathways either individually or collectively could help remove excess free fatty acids from the liver and attenuate non-alcoholic steatohepatosis (NASH) (Baskin-Bey *et al.*, 2007). This could lead to hepatic steatosis and hyperinsulinemia. Hepatic steatosis is thought to represent the first step towards the subsequent development of non-alcoholic steatohepatosis (Schaffner *et al.*, 2003). This probably explains why an increase in visceral adipose tissue is a causative risk factor for fatty liver disease and non-alcoholic liver disease. Non alcoholic fatty liver disease (NAFLD) is not confined to adults and is now the most common liver disease among obese children and adolescents in North America (Lavine *et al.*, 2004). NAFLD was found in the Third NHANES to be most prevalent in obese African-American and Hispanic males, with type 2 diabetes, hypertension and hyperlipidemia (Meltzer *et al.*, 1997). These associations have led to the hypothesis that NAFLD may precede the onset of type 2 diabetes in some individuals. Although the natural history of NAFLD in children is unknown, it may progress to cirrhosis and related complications (Feldstein, 2003). The spectrum of NAFLD includes steatosis alone (accumulation of triglyceride and fatty acids in liver cells); steatohepatitis (fatty liver along with liver cell injury and inflammation, which may lead to fibrosis); and cirrhosis (Schaffner *et al.*, 1986*).* Possible mechanisms that could explain the etiology of metabolic complications will be discussed in the next section.

2.4.5 Cancer

Obesity comes with plenty of health risks, but there is one that is perhaps not so well known: an increased risk of developing cancer. The cancer types that have been associated with obesity include cancer of the colon, breast cancer, endometrial cancer, cancer of the kidney and esophageal cancer (Calle & Kaaks, 2004). There has also been a reported link of obesity to the cancer of the ovaries, gallbladder and pancreas. These associations have been found to be in a sex and site-specific manner. This means that there are sex and cancer site–specific biological mechanisms underpinning these associations, and it is unlikely that there is a "*one system fits all*" mechanism (Roberts *et al*., 2010). Three main candidate systems have been proposed—insulin and the insulin-like growth factor–I axis, sex steroids, and adipokines. Due to some shortfalls within these mechanisms, three novel candidate mechanisms have been proposed: obesity-

induced hypoxia, shared genetic susceptibility, and migrating adipose stromal cells (Roberts, *et al*., 2010). Although it has been known for some time that excess weight is an important factor in death from cancer (Carroll, 1998), the knowledge of the magnitude of the relation, both for all cancers and for cancers at individual sites, and the public health effect of excess weight in terms of total mortality from cancer is limited (Calle *et al*., 2003). In obesity, different endocrine and metabolic signals lead to insulin resistance, which results in a permanent compensatory hyperinsulinemia and increased levels of bio-available IGF-1. This section will focus on insulin resistance and cancer link because the other mechanisms are beyond the scope of this thesis.

Similarities have been noted between risk factors, cancers and insulin resistance that have led to the suggestion that hyperinsulinemia might contribute to cancer development through the growth promoting effect of elevated levels of insulin. According to Renehan *et al*. (2006) the insulin cancer hypothesis (complex format) demonstrates the complex interrelationships between obesity, insulin resistance and pathways that might favour tumour development. Within these complexities the simple hypothesis (outlined by the dotted line) is a small part of the overall network of systems that link obesity and cancer. In brief the simple hypothesis postulates that prolonged hyperinsulinemia reduces the production of IGFBP-1(insulin-like growth factor binding protein 1) and IGFBP-2. This results in increases in the levels of free bioactive IGF-I and concomitant changes in the cellular environment that favour tumour development. This is the hypothesis in its simplest form and is summarized in Figure 2.7. However this might be over-simplistic as several other excess body weight related molecular pathways might be relevant to tumour formation.

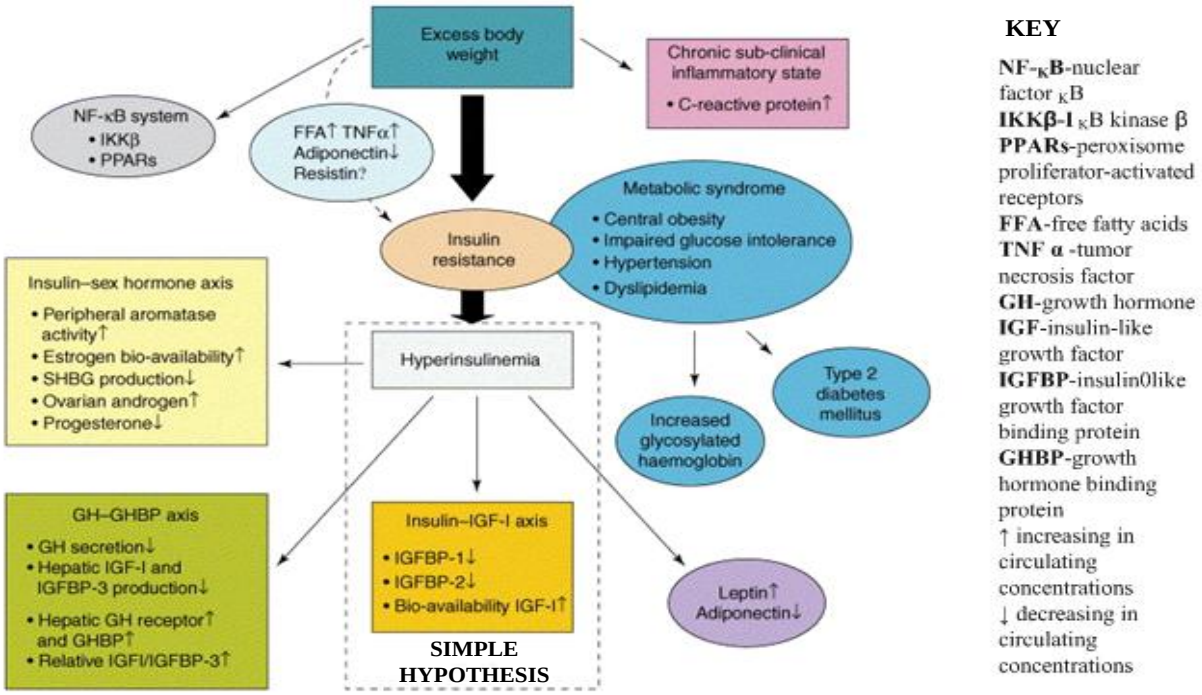


Figure 2.7: Diagrammatic representation of the complex insulin-cancer hypothesis (Renehan, *et al.*, 2006).

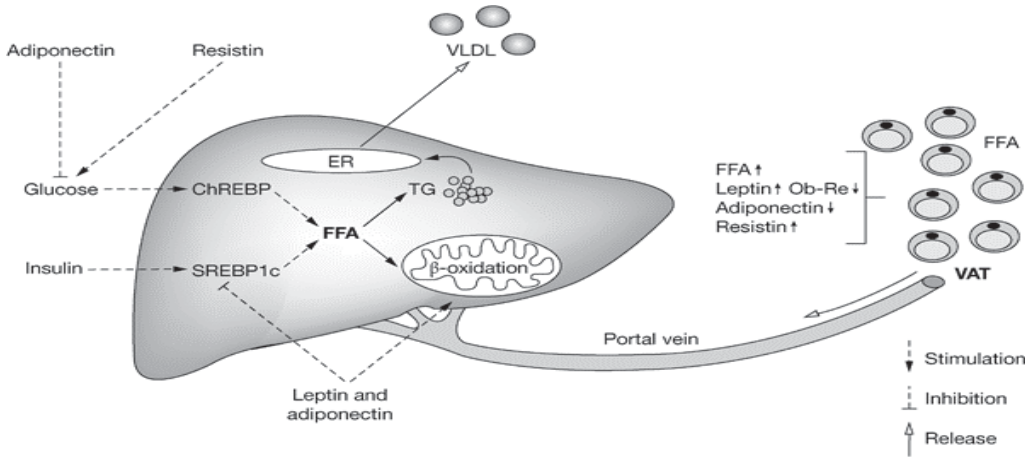
In the context of childhood obesity as evidence suggests that if overweight or obesity persists from childhood through teenage years, the risk of obesity during adulthood is greater. Therefore, efforts to reverse the increasing prevalence of obesity must continue to be supported, in particular strong, effective and urgent measures to prevent childhood obesity and reduce the prevalence of overweight as much as possible. Children should be enabled to begin their lives with the best chance possible - at the lowest part of the healthy weight range and should aim to stay at that level throughout their lives. Researchers at Bristol University in Scotland stated that 'If the cancer risk among today's young people mimics that of previous generations, our observations suggest that the impact of current childhood obesity on the cancer burden in the second half of this century may be substantial". As the worldwide obesity epidemic has shown no signs of abating while public health policies aimed at curbing the underlying causes of the obesity epidemic are being implemented, there is a parallel need to understand better the biological processes linking obesity and cancer as a prerequisite to the development of new approaches to prevention and treatment (Roberts *et al*., 2010).

2.4.6 Possible mechanisms that link visceral fat with disease risk

There has been some controversy as to whether it is the anatomical position of the visceral fat depot or the molecular characteristics that are responsible for the link between visceral fat and disease risk. In an attempt to address this some possible mechanisms have been proposed and these include free fatty acid flux to the liver, adipose tissue released cytokines and the disturbances in concentrations of inflammatory cytokines. A brief discussion of these is given in the following section.

Action of free fatty acids

The main source of FFA in the body is the fat tissue; therefore, it can be assumed that with greater adiposity in the abdominal region there is an increase in the FFA flux (Figure 2.8). FFAs released by visceral fat (but not all) go directly into the portal vein. Thus large amounts of visceral fat will result in the liver being exposed to a greater concentration of FFA than would be predicted from systemic FFA availability *(*Power *et al.,* 2008). Elevated portal FFA concentrations can have a number of undesirable effects on the liver as shown in Table 2.5 (Arner, 1997). The section that follows will discuss some investigations on this issue and the conclusions derived from the outcomes observed.



Key: **VLDL-very low density lipoprotein** **ER-endoplasmic reticulum**
ChREBP- carbohydrate response element binding protein **TG-triglyceride**
FFA- free fatty acid **VAT-visceral adipose tissue**
SREBP1c- sterol regulatory element binding protein-1c

Figure 2.8: Visceral adipose tissue and increased FFA flux to the liver (Schaffler *et al.,* 2005).

Table 2.5: Consequences for the liver of increased portal fatty acid concentration

Effect	Consequences
Increased glucose production	Glucose intolerance
Decreased insulin breakdown	Hyperinsulinemia
Increased production of triglycerides	Dyslipidemia

Arner, 1997

It has been hypothesized that visceral fat releases FFAs and adipokines and thereby exposes the liver to fat accumulation. A direct association between abdominal fat and liver fat content has been observed in studies using waist circumference to estimate abdominal fat. In studies where imaging techniques have been used to estimate abdominal fat, a direct association between intra-abdominal fat and liver fat content, a direct association has been suggested but not between subcutaneous abdominal fat and liver fat content. These methods are regarded as the golden standard methods for abdominal fat assessment because they are very accurate. Therefore, based on these observations, Jakobsen *et al*. (2007) concluded that there is a direct association between abdominal fat and liver fat content.

Studies have been conducted which demonstrated the effect of excess FFA to the liver. Some of these studies are summarised in the section that follows. Bergman *et al. (*2006) used a dog model to understand the pathogenesis of the metabolic syndrome. They pointed out that the dog is genetically similar to human beings. By using the MRI technique they measured fat distribution in dogs (1cm landmark slice at the umbilicus) after a 12 week isocaloric but elevated fat diet. Increasing the fat in the diet was found to induce visceral as well as subcutaneous fat in the dog model irrespective of basal adiposity and also the diet was found to induce total hepatic insulin resistance with respect to glucose. Contrary to this the peripheral tissues remained sensitive to hyperinsulinemia. Thus they concluded that visceral adiposity induced by fat feeding causes primary insulin resistance of the liver (Bergman *et al.,* 2006). They then postulated a hypothesis which they termed "**Overflow Hypothesis**" which states that, moderate

fat feeding increases fat in the visceral fat compartment primarily resulting in visceral deposition and hepatic insulin resistance.

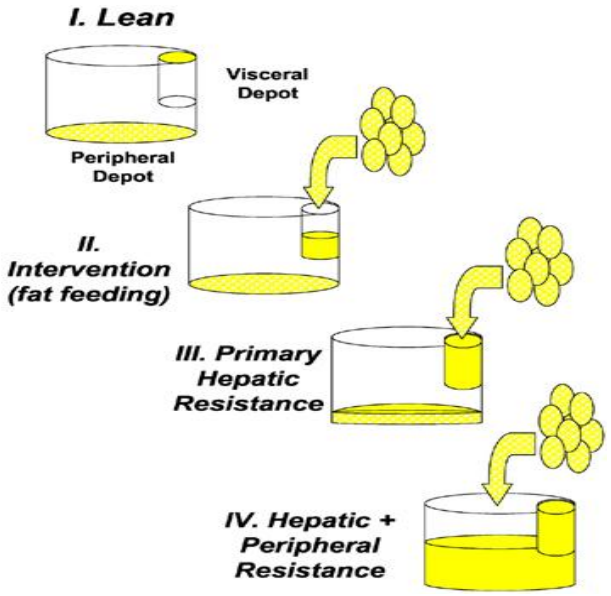


Figure 2.9: An illustration of overflow hypothesis (Bergman *et al.*, 2006)

This hypothesis was summarized in the following sequence of events which occur after the initiation of feeding by an isocaloric but high fat diet as illustrated in Figure 2.9. In summary this is what happens: Lean animals have little fat in visceral or subcutaneous compartment (I) Moderate fat feeding (II) increases fat in the visceral compartment primarily, resulting in visceral fat deposition and hepatic insulin resistance (III). Increased amounts of dietary fat (IV) result in visceral and subcutaneous fat deposition and hepatic and peripheral insulin resistance (Bergman *et al.*, 2006). Nielsen *et al.* (2004) also conducted a study to investigate whether adipose tissue lipolysis releases FFAs into the portal vein hence exposing the liver to higher concentrations of FFAs. They used the isotope dilution technique to see if a greater portion of hepatic FFAs comes from abdominal fat. By measuring systemic splanchic and leg FFA kinetics in obese men and women of varying obesity phenotypes along with women, they observed that there was a 20% greater plasma FFA concentration in obese men and women. In women the contribution of splanchic lipolysis to hepatic FFA ranged from <10% to almost 50% and increased as a function of visceral fat and the slope of the relationship was greater than in men. This observation adds to the fact that there is an increased free fatty acid flux to the liver in case of obesity. Approximate

relative contributions of FFAs released from lower- and upper-body subcutaneous fat depots and from splanchnic tissues to the systemic venous circulation, and FFAs from visceral fat and the systemic arterial circulation to the portal circulation in lean and obese subjects as shown in Figure 2.10. The values in the figure are based on data from Nielsen *et al.* (2004).

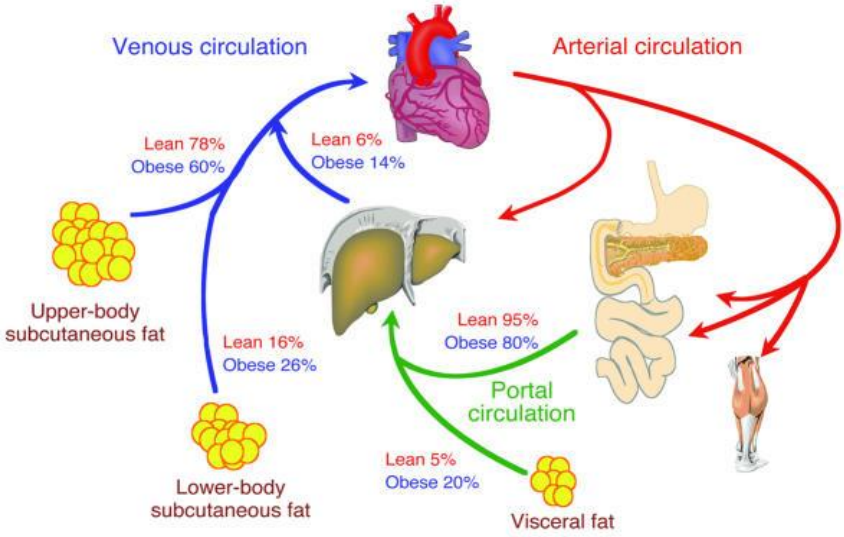


Figure 2.10: Approximate relative contributions of FFAs released from lower- and upper-body subcutaneous fat depots and from splanchnic tissues (Klein, 2004)

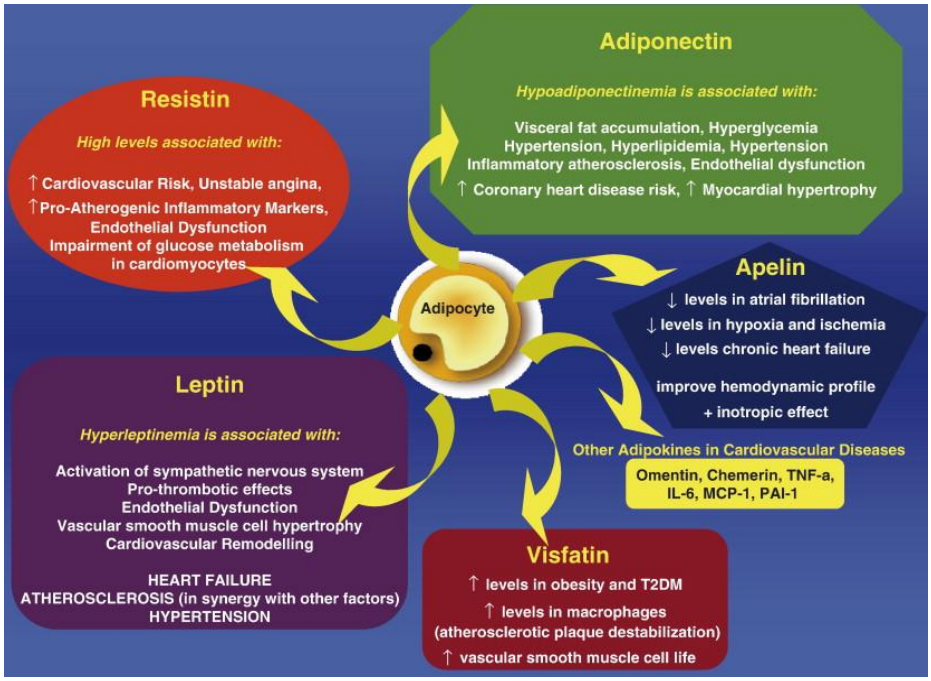
To demonstrate the mechanism by which visceral fat causes insulin resistance at the liver and in muscle, Kabir *et al.* (2005) measured gene expression of a variety of important enzymes in visceral and subcutaneous fat and liver and also the expression of some adipokines. The outcome of this investigation was that enzyme expression related to lipid turnover in visceral fat increased with fat feeding in visceral fat relative to subcutaneous fat. This increase creates a possibility of enhancing FFA flux through the portal vein to the liver as well as to other tissues. In addition, the group tested whether fasting levels reflected potentially damaging tissue exposure at all times of the day. This was done by collecting blood samples every hour for 24 hours and repeated the same protocol in a similar manner after 6 weeks of a hypercaloric high fat diet. They observed total trunk fat increase of 76% with no measurable increase in fasting FFA's. The 24-hour FFA profile increased by 50% (Bergman *et al.*, 2006). This could represent a significant increase in FFA flux to the liver from the visceral fat depot and could be an

important factor in insulin resistance of moderate obesity. Also an increase in FFAs at night could play a role in stimulating hyperinsulinemia that is normally coupled with insulin resistance in non diabetic individuals.

While the described investigations found an increase in FFA flux with increased visceral adiposity ,Weiss *et al.* (2008) stated that an increase in the visceral FFAs flux contributed only 20-30% of FFA's reaching the liver (Weiss, 2007). Thus probably visceral fat is not the main source of the majority of systemic circulating FFAs and its postulated effects on the insulin resistance. The contribution of visceral fat discharge and its presence may be only surrogate of relatively increased upper body fat depots *(*Weiss, 2007). The abdominal subcutaneous fat is probably the source of increased circulating FFAs of lean and obese individuals (Weiss *et al.*, 2008).

Damaging molecules into systemic circulation - Adipocyte secretion profile

Adipose tissue is now recognized as a key endocrine organ secreting a wide range of protein factors and signals (Figure 2.11). A number of roles have been attributed to adiponectin which includes the modulation of insulin sensitivity and vascular function (Trayhurn, 2007). There have been notable differences in the secretion of adiponectin by visceral and subcutaneous fat, and this difference has been suggested as a possible reason why visceral fat is a significant risk factor for metabolic and health complications and that these differences underlie the different risks to health (Grundy *et al.*, 2005). The focus of this section will be on adiponectin and leptin and their role as mediators in the development of metabolic syndrome.



Key: **T2DM- type 2 diabetes mellitus** **IL-6- interleuken-6**
TNF-α -tumor necrosis factor-alpha **MCP-1 -monocyte chemoattractant protein-1**
PAI-1-plasminogen activator inhibitor-1

Figure 2.11: Summary of the main effects exerted by the more relevant adipokines at the cardiovascular level *(*Gualillo *et al.,* 2007)

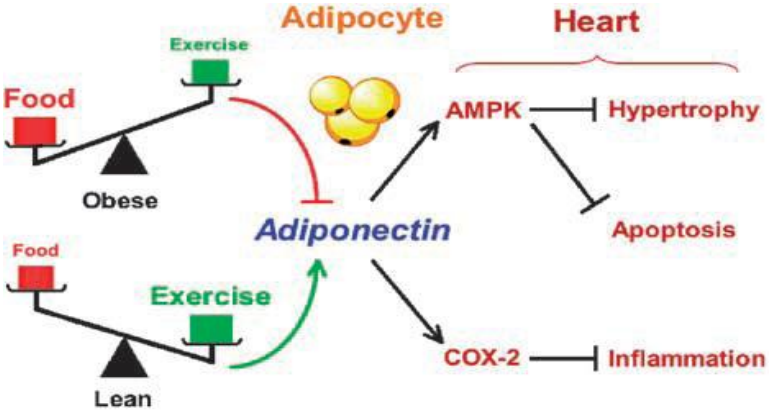
Adiponectin

Adiponectin levels decrease in the context of obesity-related insulin resistance *(*Schaffler *et al.,* 2006*).* White adipose tissue is a highly metabolic active endocrine organ whose main products of secretion, adipokines, have essential roles in energy homeostasis, glucose and lipid metabolism, cell viability, control of feeding, thermogenesis, neuroendocrine function, reproduction immunity, and, importantly, cardiovascular function (Trujillo & Scherer, 2006).

Adiponectin is normally abundant in human serum; its levels have been found to be reduced as a result of gene mutations which alter its secretion from the cells. It is remarkable to note that genome-wide scans done in human beings have mapped a susceptibility locus for type 2 diabetes, metabolic syndrome, and coronary heart disease to chromosome 3q27, where the gene encoding adiponectin is located (Das *et al.,* 1999). Thus genetic polymorphisms of the adiponectin gene that result in lower production and secretion of adiponectin may be responsible,

at least in part, for the pathogenesis of the insulin resistance syndrome and diabetes (Chandran *et al.*, 2003). In studies where adiponectin was acutely increased by infusing of full length recombinant adiponectin, improved insulin action and induced suppression of hepatic glucose production were observed. This was associated with a reduced expression of the gluconeogenic enzymes indicating that transcriptional regulation of these two enzymes may contribute to the molecular mechanism of action of adiponectin (Combs *et al.*, 2001).

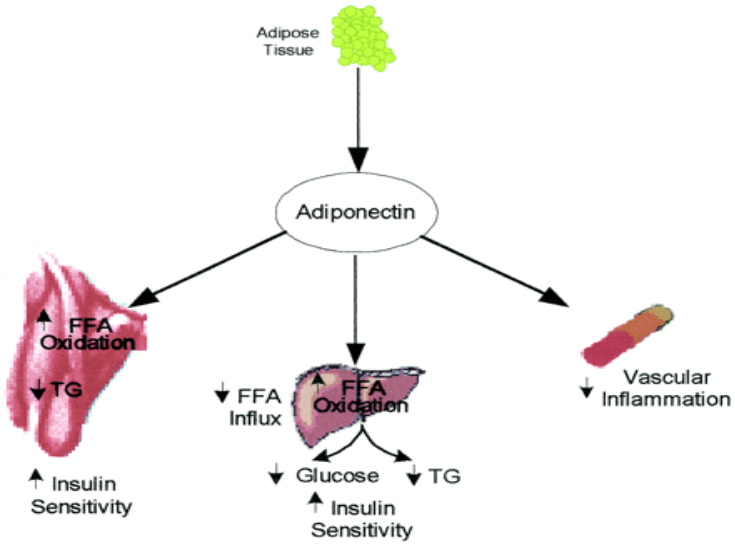
In human studies Hui *et al.* (2004) demonstrated the protective role of adiponectin when they observed that adiponectin levels were lower in patients with steatohepatosis than in those with steatosis and lower in both these groups than in healthy controls. These results demonstrate a novel mechanism of adiponectin action and suggest a potential clinical application of adiponectin and its agonists in the treatment of liver diseases (Xu *et al.*, 2003). Other beneficial effects of adiponectin are at the cardiac level where protective action against myocardial ischemic perfusion (Ouchi *et al.*, 2006) and the protection against the development of systolic dysfunction after myocardial infarction have been demonstrated (Shibata *et al.*, 2007). Plasma adiponectin level is negatively regulated by adiposity, which is influenced by the levels of food intake and physical activity (Ouchi *et al.*, 2006). Adiponectin cardioprotective action is by stimulating myocardial Adenosine monophosphate-activated protein kinase (AMPK) signalling. This then leads to the suppression of myocyte hypertrophy and apoptosis. It also stimulates COX-2 expression, resulting in reduction in cardiac inflammation (Ouchi *et al.*, 2006). These processes are illustrated in Figure 2.12.



COX-2: Cyclooxygenase -2

Figure 2.12: Cardio-protective actions of adiponectin (Ouchi *et al.*, 2006).

In skeletal muscle, adiponectin increases tyrosine phosphorylation of the insulin receptor. This effect may contribute to increased insulin sensitivity (Chandran *et al.*, 2003). In the liver, the decreased free fatty acid influx and increased fatty acid oxidation contribute to reduced hepatic glucose output and VLDL triglyceride synthesis. (Chandran *et al.*, 2003). The actions of adiponectin are summarized in Figure 2.13.



Key: **FFA-free fatty acid** **TG- triglyceride**

Figure 2.13: Hypothetical model for the actions of adiponectin (Chandran *et al.*, 2003)

Leptin

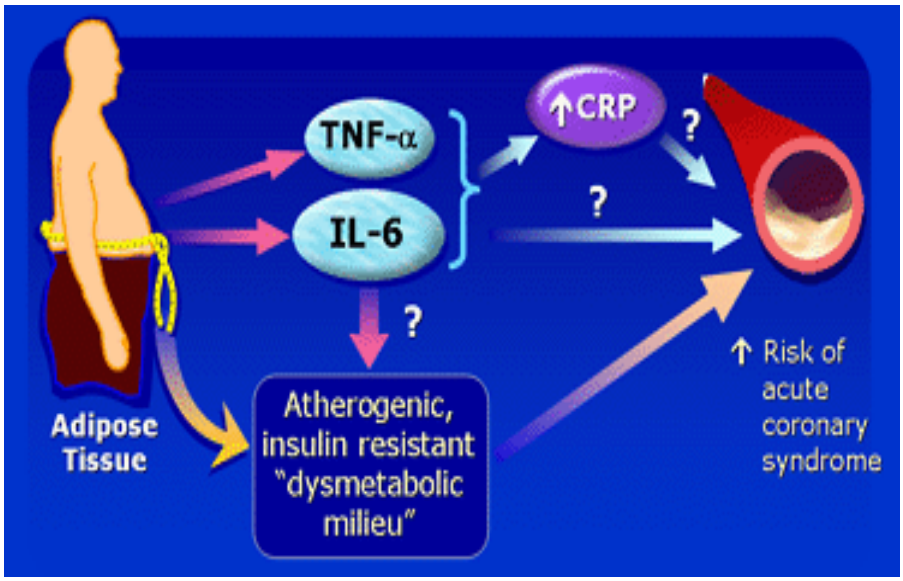
Leptin is a 167-aminoacid peptide hormone, which is mainly produced by adipocytes, and its levels in the circulation are increased in proportion to fat mass (Sousa *et al*,. 2009). Circulating leptin transmit information to the hypothalamus about the amount of energy stored in adipose tissue, suppressing appetite and affecting energy expenditure. Unger (2003) proposed, on the basis of compelling experimental evidence, that peripheral-tissue leptin resistance is a crucial factor leading to insulin resistance in metabolic syndrome. They contended that leptin's major role in normal energy homeostasis is not prevention of obesity, as originally conceived, but rather protection of nonadipocytes against the cytotoxicity of intracellular lipid overload during periods of nutrient excess. Leptin potently activates cellular fuel consumption by stimulating fatty acid oxidation, reducing lipogenesis, enhancing glucose entry and metabolism, and dramatically shrinking fat stores in adipose tissue as well as in muscle and liver cells (Unger *et*

al, 2002). Accumulation of cytoplasmic fatty acid thus could reflect functional leptin deficiency acting *via* impaired mitochondrial oxidative capacity and concomitantly enhanced lipogenesis. The ensuing insulin resistance could be viewed as a compensatory cytoprotective response to prevent further accumulation of intracellular lipid, *i.e.*, reduced glucose entry attenuates glucose derived lipogenesis. The mechanisms of peripheral resistance to the fuel-burning actions of leptin are not yet known. In reality, excess free fatty acids in the circulation, leptin resistance, and adiponectin deficiency are likely acting in concert to generate intracellular fatty acid excess, although their precise sequence and relative importance remain to be determined (Bagby, 2004). Based on evidence from *in vitro* studies it has been proposed that leptin could play a role in the pathogenesis of atheromatous plaques acting synergistically with other inflammatory mediators. Moreover leptin has been shown to induce mitochondrial superoxide production and MCP-1 expression in aortic endothelial cells, thus playing an important role in the early phase of atherosclerosis by initiating monocyte/macrophage recruitment to the vessel wall (Yamagishi *et al.*, 2001). At cardiac level directs effects of leptin have been observed in the regulation of contracticle function, metabolism, cell size and production of extra cellular matrix components (Madani *et al.*, 2006).

Circulating concentrations of inflammatory cytokines

Although the portal FFA hypothesis has been suggested to explain some of the metabolic abnormalities associated with excess adipose tissue accumulation, the hyperlipolytic state of the expanded visceral depot cannot, by itself, explain all of the metabolic abnormalities observed in viscerally obese patients (Despres, 2006). Obesity has been associated with chronic low-grade inflammation in adipose tissue. This was first suggested by Hotamisligil and Spiegelman's work demonstrating that fat cells secrete TNF-alpha in the context of obesity (Enerback, 2006). In fact a close relationship between the expanded waistline and the elevated C-reactive protein (CRP), an inflammation related protein, has been observed. The reason for this close relationship could result from evidence of macrophage infiltration in adipose tissue of abdominally obese patients. These macrophages can produce inflammatory cytokines which could have a local impact on adipose tissue metabolism as well as systemic effects as illustrated in Figure 2.14 (Despres, 2003), thus exacerbating the dysmetabolic profile noted among patients with an excess of

visceral adipose tissue. For instance, TNF-a could make the adipose tissue insulin resistant, and it also has an inhibitory effect on the production of adiponectin (an important adipose tissue-derived cytokine that has been suggested to have anti-atherogenic and anti-diabetic properties). In addition, the release of IL-6 by fat cells is known to stimulate the production of CRP through the liver. Trayhurn (2006) also stated that as adipocytes secrete a number of cytokines and acute phase proteins, it is considered that the expanded adipose tissue mass contributes either directly or indirectly to the increased production and circulating levels of inflammation related factors. This means that circulating levels of inflammatory cytokines are altered in abdominally obese subjects and this could explain the constellation of metabolic abnormalities found in them. It has been suggested that ectopic fat deposits could possibly contribute to cardiometabolic risk in viscerally obese individuals.



Key: **CRP- C reactive protein**
IL-6- interleuken-6
TNF-α -tumor necrosis factor-alpha

Figure 2.14: Illustration of Inflammation and cardiovascular disease (Despres, 2003

In conclusion a few examples of studies demonstrating associations between central fat deposition with adverse health outcomes were discussed. The studies confirm that obesity is a major risk factor for chronic diseases and plays a central role in the metabolic syndrome

(Kelishadi, 2007). The health complications are either directly caused by obesity or indirectly related through mechanisms sharing a common cause. The metabolic syndrome is used to describe the link among insulin resistance, type-2 diabetes, and other metabolic abnormalities associated with increased cardiovascular disease (Weiss *et al.,* 2004). Many of these metabolic complications associated with obesity are already present during childhood and are closely linked to the degree of adiposity (Weiss *et al.*, 2004). In this regard a potential emerging public health issue for developing countries may be the increasing incidence of obesity and, as a result the new cases of metabolic syndrome among children, which in turn is likely to create an enormous socioeconomic and public health burden for poorer nations in the near future (Freedman *et al.,* 2001).

2.5 ABDOMINAL FAT ASSESSMENT

Accurate body composition measurements are essential for evaluating nutritional status and for planning different aspects of health care programmes (Fanelli *et al.*, 1984). Childhood obesity and in particular upper body obesity perpetuate the high risk for many preventable diseases. Several methods can be used to estimate central body fat; these differ in the practicality for both field surveys and routine clinical assessment. Despite the limitations of using some methods central fat assessment has been identified as an important screening tool for the prevention of adverse health complications. Several techniques are available which include imaging techniques and anthropometric measurements. Using any of these methods depends on availability, accuracy and practicality. A comparison of these methods shows that imaging techniques, such as computerized tomography and magnetic resonance imaging, are the optimal techniques available for accurate assessment of visceral fat (Kooy & Seidell, 1993). However, due to the lack of feasible use of these procedures for general application in epidemiological studies, anthropometric indices such as circumferences, ratios and skinfold thicknesses are often used. The principles of abdominal obesity techniques, their accuracy and reproducibility as well as aspects of costs and safety are discussed in the section that follows.

2.5.1 Imaging techniques

Computed tomography (CT).

Computed tomography (CT) has been demonstrated to be a useful technique for measuring visceral fat (Jensen *et al.,* 1995). CT scans are thin cross sectional, radiographic images that can be obtained at any body level (Borkan *et al.,* 1982). It is much more sensitive to slight differences in attenuation than standard radiography and, therefore, depicts soft tissue with great clarity (Brooks *et al.,* 1975). It has provided a more direct method of intra-abdominal fat deposition assessment with both adult and child populations. The scan is usually done at the level of the umbilicus. Borkan *et al.* (1982) were the first to propose this site because it has the highest percentage of body fat and it best allows differentiation of subcutaneous from intra-abdominal fat. Because of the radiation exposure involved in CT scanning, the number of measurements that can be performed on one individual is limited, making this method unsuitable for studies requiring repeated measurements on the same individual (Kamel *et al.,* 2000). However CT studies of body composition can limit radiation exposure by using single scans at different anatomical sites *(*Kooy and Seidell., 1993*).* Even though CT at abdominal level has provided the best estimation of visceral fat, it cannot be used during pregnancy because of its potential harmful effects on the fetus (Bartha *et al.,* 2007).

Magnetic resonance imaging (MRI)

Magnetic resonance imaging is a radiation-free procedure and has been increasingly accepted as a safer alternative with children, particularly where multiple scans are required. An example of the equipment used is shown in Figure 2.15. MRI may provide a method to measure adipose tissue safely and accurately. It does not require water submersion or radiation exposure, it can be done rapidly, it provides images which are also useful for clinical diagnosis, and it has been used to characterize adipose tissue volumes (Gronemeyer *et al.,* 2000*).*

Figure 2.15: Magnetic Resonance Imaging (MRI) equipment (Kamel *et al.*, 2000)

Unlike conventional x-ray examinations MRI does not depend on ionizing radiation. Instead, while in the magnet, radio waves redirect the axes of spinning protons, which are the nuclei of hydrogen atoms, in a strong magnetic field. The magnetic field is produced by passing an electric current through wire coils in most MRI units. Other coils, located in the machine and in some cases, placed around the part of the body being imaged, send and receive radio waves, producing signals that are detected by the coils. A computer then processes the signals and generates a series of images (Figure 2.16) each of which shows a thin slice of the body. The images can then be studied from different angles by the interpreting physician.

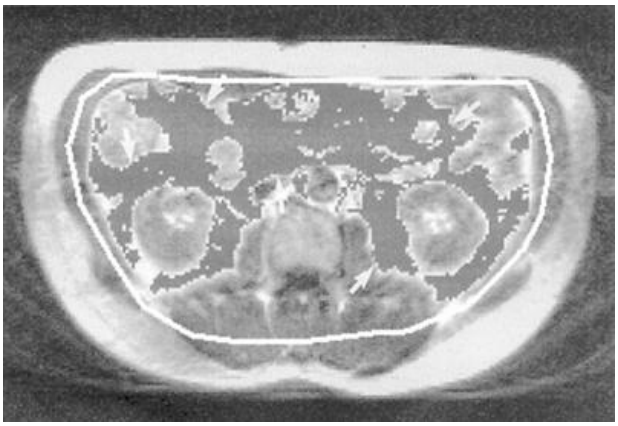


Figure 2.16: A typical MRI image (Kamel *et al.*, 2000)

Among others the most common limitations of MRI are that high-quality images are assured only if the individual is able to remain perfectly still or hold breath, if requested to do so, while the images are being recorded. Also MRI typically costs more and may take more time to perform than other imaging modalities.

Ultrasound (US).

The use of sonography for the determination of fat distribution was introduced by Armellini *et al.* (1997). Ultrasonography has been proposed as a suitable technique to estimate intra-abdominal adipose tissue in research settings accurately; however, some criticisms have been put forward on its use because of its presumed low reproducibility (Stolk *et al.,* 2003). An advantage of sound technique lies in the possibility to use it to evaluate very fat persons with whom measurements by caliper are no longer possible. The ultrasound measurements can be obtained by using a C2–5 MHz curved array transducer. The transducer is placed at the midline between the lower costal margin and the iliac crest. Intra-abdominal fat distances are then measured from the peritoneum to the corpus of the lumbar vertebra (Stolk *et al.,* 2001) and obtained longitudinally at a medial, right lateral and left lateral angle. To avoid compression of the skin, a gel spacer is used and scans are obtained by trained sonographers.

Dual-Energy X-ray Absorptiometry (DEXA)

Although the primary application of DEXA is to measure bone mineral density to ascertain risk for osteoporosis, it has also been used to measure central abdominal fat (Cary *et al.*, 1996). The principle of absorptiometry is based on the exponential attenuation of X-rays at two energies as they pass through body tissues. DXA is accurate, precise and the radiation dose is minimal (Jebb, 1997). It can provide measurements of fat, bone mineral content and fat-free soft tissue in the total body and in specifically defined regions (Snijder *et al.*, 2002). Garnett *et al.* (2000) conducted a pilot study to investigate the relationship between abdominal fat measured by anthropometry and DEXA, and intra abdominal fat as measured by MRI in healthy children. They observed that total abdominal fat measured by DEXA and MRI were highly correlated, R^2=0.98, P<0.001, with a mean difference of 29 ± 47g. The 95% limits of agreement were -64 to 123g, indicating potential for variation in individuals. DEXA abdominal fat predicted 64% (P=0.02) of the variation in intra abdominal fat as measured by MRI. However, a larger sample

size is required to confirm these findings. Another study by Snijder *et al.* (2002) investigated whether the DEXA method, possibly combined with anthropometry, offers a good alternative to CT for the prediction of visceral fat in the elderly. The total abdominal fat as measured by DEXA was found to be strongly correlated with total abdominal fat by CT (r= 0.87 in white men to 0.98 in black women) (Snijder *et al.*, 2002). A 10% underestimation of total abdominal fat by DEXA was observed in people with less abdominal fat compared to CT. They concluded that DEXA is a good alternative to CT for predicting total abdominal fat in an elderly population. Anjana *et al.* (2004) also used DEXA to examine body fat distribution using CT, DEXA and anthropometry in relation to type 2 diabetes in urban Asian Indians. Results indicated that using CT, total abdominal and visceral fat were found to be significantly higher among diabetic subjects, while subcutaneous abdominal fat, visceral–to–subcutaneous abdominal fat ratio, and visceral–to–total fat ratio showed no significant difference. Similarly with DEXA, diabetic subjects were found to have significantly higher abdominal and central abdominal fat, while none of the other parameters showed a significant difference. DEXA is simpler, less expensive, and more accessible compared to CT and MRI. Moreover, the X-ray radiation is much lower in DEXA as opposed to CT (Plourde, 1997). DEXA provides various measures of adipose tissue such as fat mass at total body, trunk, and extremities and percent body fat. The newest DEXA software also allows the operator to define specific regions of interest on the whole body scan. This allows the assessment of abdominal fat mass. Studies suggest that DEXA reliably measures a fat compartment that is highly correlated with visceral fat and a likely useful surrogate for visceral fat (Kamel *et al.*, 2000).

2.5.2 Anthropometric indices

The importance of simple anthropometric measurements and ratios of the abdomen was first shown by Ashwell *et al.* (1992). These include waist-hip ratio, waist circumference and abdominal sagittal diameter. CT and MRI are accurate imaging techniques for assessing body fat distribution, but their disadvantages are cost, radiation exposure (CT) and limited availability outside research settings (Goran & Gower, 1999), hence other indirect indicators of body fat distribution have been used in particular anthropometry. These methods are inexpensive, fast and non-invasive, although their accuracy has been questioned. The following section describes these anthropometric indices, their diagnostic quality and accuracy.

Waist Hip Ratio (WHR)

While the subject is in standing position the waist is measured as the minimal circumference and the hip is measured at the widest circumference at the hips and the buttocks. The ratio of the two measures is then calculated. Pouliot *et al.* (1992) suggested threshold values of 0.85 for women and 0.95 for men. The use of WHR as a single index for metabolic risk as well as threshold values has been found to be limited because for a given WHR value there may be large variations in the level of abdominal visceral adipose tissue that is most likely to be associated with variations in metabolic profile. Also WHR may not change when hip and waist circumferences increase by a similar proportion and is often incorrect in the event of significant loss of visceral adipose tissue (Misra & Vikram, 2003**).** Therefore, other anthropometric indexes, namely waist circumference or abdominal sagittal diameter appear to be superior to WHR.

Abdominal sagittal diameter.

Abdominal sagittal diameter is derived either from a CT abdominal scan or by using a carpenter's spirit level. While the subject is placed on a firm examination table the level is placed over the abdomen perpendicular to the length axis of the trunk at the iliac crest level. The sagittal diameter is measured with a ruler as the vertical distance from the horizontal spirit level to the examination table after a normal expiration (Sjöström *et al.*, 1996). Kvist *et al.* (1988) were the first to demonstrate that the sagittal diameter (measured on a CT scan) was closely related to the volume of visceral fat. They observed the correlations between sagittal diameter with visceral fat volume of 0.94 in 19 women and 0.92 in 24 men. Després *et al*.(1991) also in a study of men covering a wide range of fatness, also observed higher correlations but there was not much difference between the visceral fat area and the correlations with the waist circumference (r = 0.82) and the sagittal diameter (r = 0.85). Even though correlations between sagittal diameter and waist circumference are usually quite high, it is clear that this measurement requires appropriate equipment and skilled personnel, hence most people are measuring the WHR or WC as an indicator of visceral fat. Sagittal diameter is rarely used.

Waist to height ratio (WHtR)

The waist-to-height ratio (WHtR) is calculated by dividing waist circumference by the height of an individual. The ratio of the waist circumference to height was originally proposed more or less simultaneously in Japan by Hsieh *et al.*, (1995) and UK by Ashwell (1995) in the late 90s. Since then a number of studies have used a WHtR as a proxy measure of visceral adipose tissue, mainly in adults. WHtR values above 0.5 were suggested to indicate increased health risks, predominantly CVD and diabetes (Ashwell, 1995; Hsieh *et al.,* 1995) and values of above 0.6 to indicate substantially increased risk (Cox *et al.,* 1997). Lin *et al.* (2002) conducted a study to determine the optimal cut-off values of four anthropometric indices (BMI, WC, WHR and WHtR) to estimate CVD risk factors. Data were collected on 26,359 Asian men and 29,204 Asian women with a mean age of 37 years. Individual body weight, height and WC and a series of tests related to cardiovascular risk were assessed and their relationships examined. Of the four indices studied, WHtRs were found to have the largest areas under the curve relative to at least one risk factor. The conclusion was that WHtR may be a better indicator for screening overweight or obesity-related cardiovascular disease risk factors than the other three indexes (BMI, WC, and WHR). The optimal cut off values for overweight or obesity WHtRs were 0.4800 for men and 0.4500 for women. Ho *et al.* (2003) also conducted a study to determine which was the best anthropometric index among BMI, WC, waist to hip ratio (WHR) and WHtR in relation to cardiovascular risk factors. Data on anthropometric indices and cardiovascular risk factors were collected from 2895 Hong Kong Chinese aged 25 to 74 years. Analysis was done using correlations and Receiver Operator Characteristics (ROC). Based on their findings they concluded that WHtR ratio is the best simple anthropometric index in predicting a wide range of cardiovascular risk factors and related health conditions. They then formulated a health message that states that, one's WC should not exceed half the stature (Ho *et al.,* 2003). The meaning in this message is simply that everyone will have an individualized cut-off waist measurement which should be more acceptable to the public than a single measurement for all (Lemieux *et al.,* 1996*).* In 2000, Savva and colleagues conducted a study to determine whether WHtR is a better predictor of cardiovascular disease in children than BMI. Their main objective was to validate BMI, WC and WHtR as predictors for the presence of cardiovascular risk factors in children. Their conclusions were that WC and WHtR ratios are better predictors of cardiovascular disease in children than BMI.

Ashwell (2005) has developed a chart similar to that used for BMI but with the important difference that the Ashwell Shape Chart requires the user to match their waist measurement against their height rather than their weight (Figure 2.17). This chart has been based on scientific evidence from the Universities of Cambridge and London in the United Kingdom. It is suitable for both men and women. In using the chart one has to work out whether one is in healthy shape by drawing a horizontal line to correspond with height and a vertical line to correspond with WC measurement. The point where these two lines meet shows where the shape is on the chart.

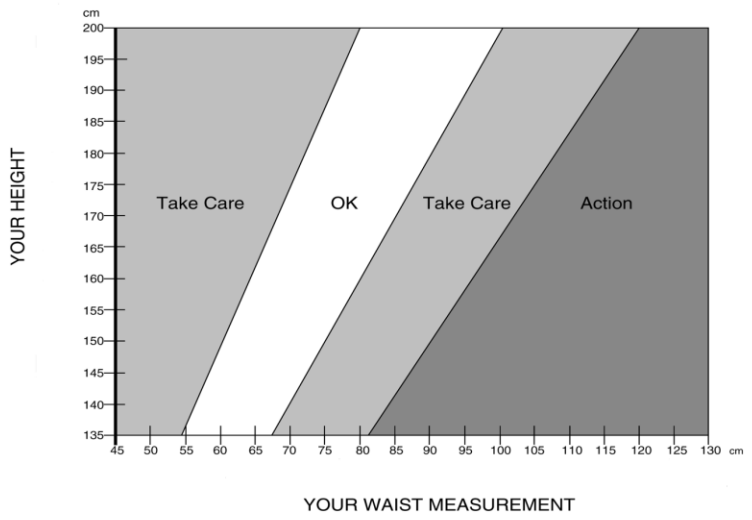


Figure 2.17: The Ashwell Shape Chart. (Ashwell, 2005)

Ashwell (2005) proposed the adoption of WHtR as a screening tool which should overcome the problem of having to define different cut-off for different population groups. They argued that communicating messages about health risk could be much simpler if the same anthropometric index and the same public health message can be used throughout childhood into adult life and throughout the world. The basis of her proposal was based on the following reasons:

- WHtR is more sensitive than BMI as an early warning of health risks.
- WHtR is cheaper and easier to measure and calculate than BMI. A boundary value of WHtR = 0.5 indicates increased risk for men and women.

- A boundary value of WHtR = 0.5 indicates increased risk for people in different ethnic groups.
- WHtR boundary values can be converted into a consumer-friendly chart.
- WHtR may allow the same boundary values for children and adults.

Later Li *et al*. (2006) also conducted a study to examine trends in WC, WHtR and prevalence of abdominal obesity in children aged 2-19 years in the US. They used 0.5 as a cut-off for WHtR in defining obesity for the 6-19 year old group. They observed that WHtR highly correlated with visceral fat and suggested that it might be a better predictor for CVD than BMI. This observation contributed to the body of information promoting the use of WHtR as a screening tool for abdominal, hence the simple health message, '***Keep your waist circumference to less than half your height*'** (Ho *et al.*, 2003)**.**

Ashwell (2009) reviewed the benefits of WHtR and its graphical representation in the Ashwell Shape Chart for assessing the health risks of obesity. The latest versions of the Ashwell Shape Chart and calculator have now been modified to include height and WC appropriate for children aged five years and upwards. The words for the different body shapes (pear-apple) have now been extended to indicate that the ratio value greater than 0.5 should indicate 'take care' or 'consider action' for adults, whereas for children it indicates 'take action'. This difference is based on the proportion of children and adults who fall above this boundary value.

Waist circumference

WC is a perimeter, which provides an estimate of body girth at the level of the abdomen (Klein, 2007). A measure of the WC is one of the most practical tools used to assess abdominal fat for chronic disease risk and weight loss treatment. A large waist circumference reflects high total body fatness and has also been recognized as a good measure of abdominal fat, particularly the most metabolically active intra-abdominal fat in both adults (Seidell *et al.,* 1988) and children (Fox *et al.,* 1993).

Different researchers have measured WC at different positions around the abdomen. In children, WC has been measured at five different sites: (i) midway between lowest rib and superior iliac

crest (Sung *et al.,* 2008*;* Moreno *et al,* 1999; McCarthy *et al.,* 2001; Fredriks *et al.,* 2005; Kelishadi *et al., 2*007*);* (ii) at the umbilical level (Zannolli, 2004; Savva *et al.*, 2000), (iii) at the narrowest point of the torso (Katzmarzyk, 2004), (iv) at the level of the right upper iliac crest (Fernandez, 2004); and (v) at the level of 2 cm above the umbilicus (Weili *et al.,* 2007). In adults WC measurements taken at four different sites have been compared and have shown to provide slightly different results, albeit highly reproducible data, that each correlate significantly with total and central body fat (Sung *et al.,* 2008). Whereas the World Health Organization (WHO) Expert Committee on Physical Status recommends measurement midway between the lower rib and the iliac crest (WHO, 1995), the third NHANES guidelines prescribe the use of a point just above the right ileum (Chumlea & Kuczmarski, 1995) and the recommendation of the North American Association for the Study of Obesity and the National Heart, Lung and Blood Institute (National Heart, Lung, and Blood Institute, 1998) is to use the right iliac crest. The lack of standard measurement for WC is unfortunate, and it makes comparisons between studies difficult. When it comes to classifying individuals this poses problems because different positions result in different values being obtained. Heitze *et al.* (2008) conducted a study to investigate the concordance of WC measured at four different points and their relationships with nutritional status and cardiovascular risk. The findings were that WC measured at different sites were closely correlated with BMI as well as comparably associated with cardio-metabolic risk factors. However, different values for the WC were obtained leading to discordant results with respect to overwaist and risk (Heitze *et al.,* 2008). Mason *et al*. (2010) concluded that recommended WC thresholds in adults may not have the same clinical utility at all anatomical locations of measurement. Based on their findings no measurement protocol is superior over the other; however, in investigations it is important to understand the difference across measurement sites. Furthermore the need to standardize measurement protocols between countries was highlighted. If WC is to become an important public health assessment tool of central obesity in both adults and children, international agreement about measurement site is required. It is, however, believed that the use of the narrowest waist measurement offers greater ease of acceptance and interpretation by the public and may facilitate self measurement in addition to clinical use (Esmaillzadeh, *et al.,* 2006). In addition Cameron and colleagues (2009) have questioned why earlier reports consider WC circumference as the most important indicator of adiposity. This question was raised after the team conducted a study to investigate the

quantitative relationship between WC and height, and subsequently the association between WC index, body mass index and body composition in pre-pubertal children. The outcomes of this study were that BMI rather than WC index would be a better screening tool for total and truncal fat mass in both sexes before puberty. The most important argument they raised against previous studies was that they did not control for pubertal status, therefore did not test whether WC is better than BMI before puberty, as the centralization of fat primarily takes place after the onset of puberty. WC and health outcomes are affected by demographic variables such as sex, race and age. The shape of the relationship influences the WC value that can most efficiently distinguish between normal or abnormal and serve as a basis for considering clinical action. Optimum WC cut-off points will likely vary according to the population studied, the health outcome of interest and demographic factors (Klein, 2007). Routine measures of WC can also be used as a surrogate marker of abdominal fat to monitor the efficacy of weight loss management strategies. This will help to avoid the expense of radiological imaging techniques and still allow for adequate prediction of health.

2.6 WC PERCENTILE CHARTS FOR CHILDREN

There is an increasing interest in the use of WC as an index of obesity and obesity related health risk among children and adolescents as several studies have shown a strong association between WC and risk factors for coronary heart disease (Katzmarzyk, 2004). This has led to the development of WC percentiles in several countries such as Cuba (Martinez *et al.* 1994), Italy (Zannolli & Morgese, 1996), Spain (Moreno *et al.*, 1999), UK (McCarthy *et al.*, 2001), Canada (Katzmarzyk *et al.*, 2004), US (Fernandez *et al.*, 2004), Australia (Eisenmann, 2005), Bulgaria (Galcheva *et al*., 2009), Poland (Nawarycz *et al*., 2010) and China (Liu *et al*., 2010). For those who have developed the curves the 90^{th} and 85^{th} percentiles are usually used to identify children with a high WC relative to age and sex matched peers. In Africa one study was done in Nigeria on determination of WC values of children and to compare them with available data from other parts of the world. Comparison of the 50^{th} percentile WC of Nigerian children with that of American and Spanish children showed that both Nigerian male and female had the lowest WC values, but were similar to British male children up to 9 years and female children up to 14 years (Senbanjo *et al.*, 2009).

To date there are no WC reference data for South African children. Studies in the past [(NFCS, 1999), Monyeki *et al.* (1999), Jinabhai *et al*. (2003) and THUSA BANA (2006)] have focused on overall overweight and obesity as opposed to abdominal obesity. In a study of rural children none of the boys were overweight before the age of 15, after which there was an increase in subcutaneous skinfold thicknesses with a peak at 17 years. When overweight was defined by the sum of triceps and subscapular skinfolds thickness greater than the 85th percentile, it increased markedly in girls after menarche and peaked at 17 years, with 11% of girls being overweight (Cameron *et al*., 1997). Another study by Kruger *et al.,* (2004) indicated that stunted girls have relatively more subcutaneous fat than non stunted girls. A possible reason for the absence of data on abdominal obesity might be because the concentration of research has been on undernutrition, but while undernutrition remains a problem among children, obesity and associated non communicable diseases are now becoming prevalent in South Africa. There is ample evidence that accumulation of excess fat in the body is associated with various metabolic complications, more importantly a pattern of excess fat in the trunk is associated with increased cardiovascular risks such as hypertension compared with a pattern of fat deposition in the limb region (He *et al.,* 2002). Existing reference data on WC from other countries (British, Canadian, Spanish, Australian, and the USA) have shown that there are differences in WC. These differences may partly reflect genetic or racial differences on body fat distribution (Senbanjo *et al.,* 2009). They may also reflect differences in dietary intake, physical activity levels and other environmental factors. This then supports the need to develop age, gender and population specific WC reference charts for South African children. However, limited existing data suggest that there may be ethnic differences in visceral adipose tissue at a given WC (Carroll *et al.*, 2008). This may have implications for defining metabolic risk in different populations; therefore to reflect risk in different racial groups adequately it is important to develop population specific standards for WC. It then seems prudent to develop standards which are truly representative of children from all ethnic backgrounds (McCarthy *et al.,* 2001). Future studies should address the feasibility of combining data from several countries to establish a common set of international reference data for WC, similar to the effort to create international BMI criteria for obesity (Cole *et al,*. 2000). This would aid in international comparisons of prevalence of abdominal obesity.

2.7 SUMMARY

Finally, this chapter identified excess abdominal fat as a serious health issue because of its links with metabolic complications. Individuals who store a larger proportion of fat within the abdominal cavity have greater risk of health complications as highlighted. It has been noted, however, that there has been increasing research focusing attention on the assessment and consequences of visceral fat deposition in adults with much less attention paid to children. This has resulted in little information generated on the factors involved in the development of abdominal adipose tissue patterning, the age at which particular types of distribution are identifiable, the degree to which they are stable or vary over time, or the influence of lifestyle factors, gender or stage of maturation. Therefore, the consequences of fat deposition patterning for childhood health or its impact on health as a future adult remain largely unknown. It can thus be recognized that the health of children has not been a top priority when it comes to metabolic complications. The different assessment techniques have been reviewed which distinctly point to the fact that reliable and accurate assessment of abdominal adipose tissue distribution is critical to developing understanding of its development in children. The discussion describes the situation in terms of studying anthropometric indices of abdominal obesity in children and their appropriateness in predicting adverse health outcome. It is clear that emphasis in the future should be on early identification of children, using appropriate screening techniques in order to promote the incorporation of systematic monitoring of WC in pediatric practice. A diverse amount of research has used different cut-off values for different population groups. This research does not recommend any single cut-off point that can be applied across the board; it is in this light that the current research was undertaken, in giving a preliminary picture of the WC distribution of South African children and also setting a stage for future studies. Furthermore, issues surrounding the importance of abdominal adiposity in children were discussed in an attempt to highlight the fact that the adverse effects associated with abdominal obesity will continue to exist if no action is taken. This may lead to children developing non-communicable diseases prematurely, and it is for this reason that research on abdominal obesity in children is necessary to understand its health risks so that observations may be used in setting goals for nutrition programmes to make sure children live a happy, healthy life, full of fun.

2.8 REFERENCES

ADAIR, L. S. 2008. Child and adolescent obesity: Epidemiology and developmental perspectives. *Physiology and behaviour*, 94(1):816.

Adult Treatment Panel III 2001 Executive summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *Journal of the American medical association*, 285:2486–2497.

AFGHANI, A. & GORAN, M. I. 2006. Racial Differences in the Association of Subcutaneous and Visceral Fat on Bone Mineral Content in Prepubertal Children. *Calcified tissue international*, 79:383-388.

AGIRBASLI, M. 2005. Metabolic syndrome in Turkish children and adolescents. *Metabolism*, 55:1002-1006.

ALBERTI, G. 2005**.** Introduction to the metabolic syndrome. *European heart journal supplements*, 7(supp D):D3-D5.

ALBRIGHT, A.L., & STERN, J.S. 1998. Adipose tissue. In: Encyclopaedia of Sports Medicine and Science, FAHEY, T D. (ed). Internet Society for Sport Science: http://sportsci.org. 30 May 1998. Date of access: 29 Jan.2009.

AL-MUBASLAT, A. 2005. The Metabolic Syndrome: Pathogenesis, Consequences, and Treatment Strategies Issue. Available at: http://www.residentandstaff.com. Date of access: 20 Feb.2009.

AMOS, A. F., MCCARTHY, D. J., & ZIMMET, P. 1997. The Rising Global Burden of Diabetes and Its Complications: Estimates and Projections to the Year 2010. *Diabetic medicine*, 14: S1–85.

ANJANA, M., SANDEEP, S., DEEPA, R., VIMALESWARAN, K S., FAROOQ, S., & MOHAN, V. 2004. Visceral and Central Abdominal Fat and Anthropometry in Relation to Diabetes in Asian Indians. *Diabetes care,* 27(12); 2948-2953.

ANTONIOS, T. F. T., SINGER, D. R. J., MARKANDU, N. D., MORTIMER, P. S. & MACGREGOR, G. A. 1999. Structural skin capillary rarefaction in essential hypertension. *Hypertension,* 33: 998–1001.

ARMSTRONG, M. E., LAMBERT, M. I., SHARWOOD, K. A., & LAMBERT, E. V. 2006. Obesity and overweight in South African primary school children - the Health of the Nation Study. *South African medical journal,* 96(5): 439-44.

ARNER, P. 1997. Regional adiposity in man. *Journal of endocrinology,* 155(2): 191–192.

Ashwell Shape chart. 2005. Available at http://www.bioportfolio.com/search/Ashwell shape chart. Date of access: 10 Aug. 2009.

ASHWELL, M. 2005. Waist to height ratio and the Ashwell shape chart could predict the health risks of obesity in adults and children in all ethnic groups. *Nutrition and food science,* 35**:** 359-364.

ASHWELL, M. 2009**.** Obesity risk: importance of the waist-to-height ratio. *Nursing standard,* 23(41): 49-54.

ASHWELL, M., CHINN, S., STALLEY, S., & GARROW, J. S. 1982. Female fat distribution - a simple classification based on two circumference measurements. *International journal of obesity,* 6(2):143-152.

BAGBY, S. P. 2004. Obesity initiated metabolic syndrome and the kidney: a recipe for chronic kidney disease. *Journal of American society of nephrologists,* 15: 2775-2791.

BASKIN-BEY, E. S., HUANG, W., ISHIMURA, N., ISOMOTO, H., BRONK, S. F., BRALEY, K., CRAIG, R. W., MOORE, D. D., & GORES, G. J. 2007. Constitutive androstane receptor

agonist, TCPOBOP, attenuates steatohepatitis in the methionine choline-deficient diet-fed mouse. *World journal of gastroenterology,* 14: 13(42):5635-41.

BERGMAN, R. N., KIM, S. P., HSU, I. R., CATALANO, K. J., CHIU, J. D., KABIR, M., RICHEY, J. M., & ADER, M. 2007. Abdominal obesity: role in the pathophysiology of metabolic disease and cardiovascular risk. *American journal of medicine,* 120(2A): S3-S8.

BIBBINS-DOMINGO, K., COXSON, P., PLETCHER, M. J., LIGHTWOOD, J. & GOLDMAN, L. 2007. Adolescent Overweight and Future Adult Coronary Heart Disease. *New England journal of medicine*, 357(23):2371-2379.

BLACK, R. E, ALLEN, L. H, BHUTTA, Z. A, CAULFIELD, L. E., DE ONIS, M., EZZATI, M., MATHERS, C., & RIVERA, J. 2008. Maternal and child undernutrition: global and regional exposures and health consequences. *Lancet,* 371:243-260.

BOURNE, L. T., HENDRIKS, M. K., MARAIS, D., & ELEY, B. 2007. Addressing malnutrition in young children in South Africa-setting the national context for paediatric food based dietary guidelines. *Maternal and child nutrition,* 3(4):230-238.

BRAMBILLA, P., MANZONI, P., SIRONI, S., SIMONE, P.; DEL MASCHIO, A., DI NATALE, B., & CHIUMELLO, G**.** 1994. Peripheral and abdominal adiposity in childhood obesity. *International journal of obesity related metabolic disorders,* 18:795–800.

CALLE, E.E. & KAAKS, R. 2004. Overweight, obesity and cancer: Epidemiological evidence and proposed mechanisms. *Nature reviews cancer,* 4, 579-591

CALLE, E.E., RODRIGUEZ, C., WALKER-THURMOND, K., & THUN, M.J. 2003. Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. *New England journal of medicine,* 348:1625-1638.

CAMERON, N., & GETZ, B. 1997. Sex differences in the prevalence of obesity in rural African adolescents. *International journal of obesity related metabolic disorders,* 21(9):775-782.

CAMERON, N., JONES, L.L., GRIFFITH, P. K., NORRIS, S. A & PETTIFOR, J.M. 2009. How well does waist circumference and body mass index reflect body composition in pre-pubertal children? *European journal of clinical nutrition,* 63: 1065-1070.

CAMERON, N., WRIGHT, M. M., GRIFFITHS, P. L., NORRIS, S. A., & PETTIFOR, J. M. 2005. Stunting at 2 Years in Relation to Body Composition at 9 Years in African Urban Children. *Obesity research,* 13:131–136.

CAREY, D. G., JENKINS, A. B., CAMPBELL, L. V., FREUND, J., & CHISHOLM, D. J. 1996. Abdominal fat and insulin resistance in normal and overweight women: direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. *Diabetes*, 45:633–638.

CARROLL, K. 1998. Obesity as a risk factor for certain types of cancer. *Lipids*, 33:1055-1059.

Centres for Disease Control and Prevention. NHANES 2003–2004. Available at: www.cdc.gov/nchs/about/major/nhanes/nhanes2003-2004. Date of access: 24 Aug. 2009

CHAKRABORTY, P & ANDERSON, A.K. 2010. Predictors of overweight in children under five years of age in India. *Current research journal of social sciences*, 2(3):138-146.

CHANDRAN, M., PHILLIPS, S. A., CIARALDI, T., & HENRY, R. R. 2003. Adiponectin: more than just another fat cell hormone? *Diabetes care,* 26(8):2442-50.

CHOPRA, M. 2003. Risk factors for undernutrition of young children in a rural area of South Africa. *Public health nutrition,* 6(7):645-652.

CHOUDHARY, A., DONNELLY, L. F., RACADIO, J. M., & STRIFE, J. L. 2007. Diseases associated with childhood obesity. *American journal of entegenology,* 188:1118-1130.

COLE, T.J., BELLIZZI, M.C., FLEGAL, K.M. & DIETZ, W.H. 2000. Establishing a standard definition for child overweight and obesity worldwide: international survey. *British medical journal*, 320:1240

COOK, S., WEITZMAN, M., AUINGER, P., NGUYEN, M., & DIETZ, W. H. 2003. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994. *Archives of paediatric adolescent medicine,* 157:821–827.

CRUZ, M. L., & GORAN, M. I. 2004. The metabolic syndrome in children and adolescents. *Current diabetes reports,* 4:53–62.

CSÁBI, G., TÖRÖK, K., JEGES, S., & MOLNÁR, D. 2000. 2000. Presence of metabolic cardiovascular syndrome in obese children, *European journal of paediatrics,* 159**:** 91–94.

DABALEA, D., & PETTIT, D. J. 2001. Intrauterine diabetic environment confers risk for type 2 diabetes mellitus and obesity in the offspring in addition to genetic susceptibility. *Journal of paediatric endocrinology and metabolism,* 14:1985-1991.

DAS, K., LIN, Y., WIDEN E., ZHANG, Y., & SCHERER, P. E. 2001. Chromosomal localization, expression pattern and promoter analysis of the mouse gene encoding adipocyte-specific secretory protein Acrp30. *Biochemical and biophysical research communications,* 280:1120–1129.

DAVISON, K. K., & BIRSCH, L. L. 2001. Childhood overweight: a contextual model and recommendations for future research. *Obesity reviews,* 2:159–171.

DE FERRANTI, S. D., GAUVREAU, K., LUDWIG, D. S., NEUFELD, E. J., NEWBURGER, J. W., & RIFAI, N. 2004. Prevalence of the metabolic syndrome in American adolescents: findings from the third National Health and Nutrition Examination Survey. *Circulation,* 110:2494–497.

DE JONGH, R. T., SERNE, E. H., IJZERMAN, R. G., DE VRIES, G.& STEHOUWER, C. D. A. 2004. Free fatty acid levels modulate microvascular function: relevance for obesity-associated insulin resistance, hypertension, and microangiopathy. *Diabetes,* 53:2873-2882.

DELISLE, H. 2002. Evidence and implications for policy and intervention strategies- World Health Organization. Geneva, Switzerland.

DESPRES, J. P. 2006. Is visceral obesity the cause of the metabolic syndrome? *Annals of medicine*, 38:52–63.

DESPRÉS, J. P. & LEMIEUX, I. 2006. Abdominal obesity and metabolic syndrome. *Nature*, 444:881–887.

EISENMANN, J. C. 2005. Waist circumference percentiles for 7- to 15-year-old Australian children. *Acta Paediatrica*, 94**:**1182–1185.

EKELUND, U., ONG, K., LINNE, Y., NEOVIUS, M., BRAGE, S., DUNGER, D. B., WAREHAM, N. J. & ROSSNER, S. 2006. Upward weight percentile crossing in infancy and early childhood predicts fat mass in young adults: The Stockholm Weight Development Study (SWEDES). *American journal of clinical nutrition*, 83; 324-330.

ELLIS, K. J., ABRAMS, S. A., & WONG, W. W. 1997. Body composition of a young multiethnic female population. *American journal of clinical nutrition*, 65:724–731.

ENERBÄCK, S., BAILEY, A., CEDERBERG, A., & FREDRIK, F. (organizers). 2006. The Adipocyte: A multifunctional cell available at http://nobelprize.org/nobelfoundation. Date of access 24 Aug. 2009.

ESMAILLZADEH, A., MIRMIRAN, P., & AZIZI, F. 2006. Clustering of metabolic abnormalities in adolescents with the hypertriglyceridemic waist phenotype. *American journal of clinical nutrition*, 83:36–46.

EUSER, A. M., FINKEN, M. J., KEIJZER-VEEN M. G., HILLE, E. T., WIT, J. M., & DEKKER, F. W. 2005. Association between prenatal and infancy weight gain and BMI, fat mass and fat distribution in young adulthood: a prospective cohort study in males and females born very preterm. *American journal of clinical nutrition*, 81:480-487.

EZZAHIR, N., ALBERTI, C., DEGHMOUN, S., ZACCARIA, I., CZERICHOW, P., LEVY-MARCHAL, C., & JACQUET, D. 2005. Time course of catch up in adiposity influences adult anthropometry in individuals who were born small for gestational age. *Paediatric research*, 58:243-247.

Fat Body Types, Picture of types of body fat distribution available at http://www.health-emark.com/fat-body-types-pic. Date of access 14 Aug. 2009.

FELDSTEIN, A. E., CANBAY, A., ANGULO, P., TANIAI, M., BURGART, L. J., LINDOR, K. D., & GORES, G J. 2003. Hepatocyte apoptosis and fat expression are prominent features of human nonalcoholic steatohepatitis. *Gastroenterology,* 125:*437*–443.

FERNANDEZ, J. R., REDDEN, D. T., PIETROBELLI, A., & ALLISON, D. B. Waist circumference percentiles in nationally representative samples of African-American, European-American, and Mexican-American children and adolescents. *Journal of paediatrics,* 145:439–444.

FLORENCIO, T., FERREIRA, H., TOJAL, A., CAVALCANTE, J., & SAWAYA, A. 2001. Obesity and undernutrition in a very low income population in the city of Maceió, northeastern Brazil. *British journal of nutrition,* 86:277-283.

FRIEDMAN, J. M., & HALAAS, J. L. 1998. Leptin and the regulation of body weight in mammals- Review. *Nature,* 395:763-770.

GALCHEVA, S. V., IOTOVA, V. M., YOTOV, Y. T., GROZDEVA, K. P., STRATEV, V. K., TZANEVA, V. I. 2009. Waist circumference percentile curves for Bulgarian children and adolescents aged 6-18 years. *International journal of paediatric obesity,* 4(4):381-388.

GAMI, A.S., WITT, B.J., HOWARD, D.E., ERWIN, P.J., GAMI, L.A., SOMERS, V.K., & MONTORI, V.M. 2007. Metabolic Syndrome and Risk of Incident Cardiovascular Events and Death: A Systematic Review and Meta-Analysis of Longitudinal Studies. *Journal of the American college of cardiology,* 49:403-414.

GARNETT, S., WOODHEAD, H., KEMP, A., BRIODY, J., BAUR, L. A., & COWELL, C. T. 2000. Measurement of abdominal fat in children: MRI, DEXA and anthropometry. *International Congress on Sport Science* organized by Australian Sports Commission in co-ordination with Sports Medicine Australia ,, 7-13 September Brisbane, Australia 2000.

GLASS, T. A., & McATEE, M. J. 2006. Behavioural science at the crossroads in public health: extending horizons, envisioning the future, *Social science and medicine,* 62:1650–1671.

GOOREN, L. 2008. Obesity: new aspects. *Journal of men's health,* 5(3):249-256.

GORIS, A. H. C., & WESTERTERP, K. R. 2008. Physical activity, fat intake and body fat. *Physiology and behaviour*, 94(2):164-168.

GROENEVELD, I. F., SOLOMONS, N. W., & DOAK, C. M. 2007. Determination of central body fat by measuring natural waist and umbilical abdominal circumference in Guatemalan schoolchildren. *International journal of paediatric obesity*, 2(2):114-21.

GROOP, L. & ORHO-MELANDER, M. 2001. The dysmetabolic syndrome. *Journal of internal medicine*, 250:105-120.

GRUNDY, S. M., CLEEMAN, J. I., DANIELS, S. R., DONATO, K. A., ECKEL, R. H., FRANKLIN, B. A.,GORDON, D. J.,KRAUSS, R. M., SAVAGE, P. J., SMITH, JR, S. C., SPERTUS., J. A., & FERNANDO, C. 2005. Diagnosis and Management of the Metabolic Syndrome: An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement. *Circulation,* 112:2735-2752.

GUALILLO, O., GONZÁLEZ-JUANATEY, J. R., & LAGO, F. 2007. The Emerging Role of Adipokines as Mediators of Cardiovascular Function: Physiologic and Clinical Perspectives. *Trends in cardiovascular medicine,* 17:275-283.

HAMDY, O., PORRAMATIKUL, S & AL-OZAIRI, E. 2006. Metabolic obesity: the paradox between visceral and subcutaneous fat. *Current diabetes reviews,* 2(4):1-7.

HARRABI, I. BOUAOUINA, M., MAATOUG, J., GAHA, R., & GHANNEM, H. 2008. Prevalence of the metabolic syndrome among urban school children in Sousse, Tunisia. *International journal of cardiology*, 135(1):130-131.

HARSHA, D. W., VOORS, A. W., & BERENSON, G. S. 1980. Racial differences in subcutaneous fat patterns in children aged 7-15 years. *American journal of physical anthropology*, 53(3):333-337.

HE, Q., HORLICK, M., THORNTON, J., WANG, J., PIERSON, R. N., HESHKA, S., & GALLAGHER, D. 2002. Sex and race differences in fat distribution among Asian, African-American, and Caucasian prepubertal children. *Journal of clinical endocrinology metabolism,* 87:2164–2170.

HOFFMAN, D., MARTINS, P., ROBERTS, S., & SAWAYA, A. L. 2007. Body fat distribution in stunted and normal height children from shantytowns of Sao Paulo. Brazil. *Nutrition*, 23(9):640-645.

HUANG, P. L. 2009. A comprehensive definition for metabolic syndrome. *Disease models and mechanisms*, 2(5-6):231–237.

HUANG, T. T- K., JOHNSON, M. S., FIGUEROA-COLON R., DWYER J. H. J., & GORAN, M. I. 2001. Growth of Visceral Fat, Subcutaneous Abdominal Fat, and Total Body Fat in Children. *Obesity research,* 9**:** 283–289.

HUL, J. M., HODGE, A., FARRELL, G. C., KENCH, J. G**.,** KRIKETOS, A., & GEORGE, J. 2004. Beyond insulin resistance in NASH: TNF-α or adiponectin? *Hepatology*, 40:46-54.

IBANEZ, L., ONG, K., DUNGER, D. B., & ZEGHER, F. 2006. Early development of adiposity and insulin resistance after catch up weight gain in small-for-gestational-age children. *Journal of clinical endocrinology and metabolism,* 91:2153-2158.

ISOMAA, B., ALMGREN, P., TUOMI, T., FORSEN, B., LAHTI, K., NISSEN, M., TASKINEN, M., & GROOP, L. 2001. Cardiovascular morbidity and mortality associated with the metabolic syndrome, *Diabetes care,* and 24:683–689.

JAKOBSEN, M. U., BERENTZEN, T., SØRENSEN, T. I. A., & OVERVAD, K. 2007. Abdominal Obesity and Fat**ty** Liver. *Epidemiologic reviews*, 29(1):77-87.

JENSEN, M. D. 2007. Role of Body Fat Distribution and the Metabolic Complications of Obesity. *The Journal of clinical endocrinology & metabolism*, 93(supp.1):s57-s63.

JEQUIER, E. 1993. Adaptations to low and high caloric intake in humans. *International journal of obesity*, 17:9-12.

JINABHAI, C. C., TAYLOR, M., SULLIVAN, K. R. 2003. Implications of the prevalence of stunting, overweight and obesity amongst South African primary school children: a possible nutritional transition? *European journal of clinical nutrition*, 57(2):358-365.

KABIR, M., CATALANO, K. J., ANANTHNARAYAN, S., KIM, S. P., VAN CITTERS, G. W., DEA, M. K. & BERGMAN, R. N. 2005. Molecular evidence supporting the portal theory: a causative link between visceral adiposity and hepatic insulin resistance. *American journal of physiology, endocrinology and metabolism*, 288:E454–E461.

KALK, W. J., & JOFFE, B. I. 2008. The Metabolic Syndrome, Insulin Resistance, and Its Surrogates in African and White Subjects with Type 2 Diabetes in South Africa. *Metabolic syndrome and related disorders*, 6(4):247-255.

KAMEL, E. G., MCNEIL, G., & Van WIJK, M. C. W. 2000. Usefulness of Anthropometry and DXA in Predicting Intra-abdominal Fat in Obese Men and Women. *Obesity research,* 8**:**36–42.

KATZMARZYK, P. T. 2004. Waist circumference percentiles for Canadian youth 11–18 y of age. *European journal of clinical nutrition,* 58:1011–1015.

KELISHADI, R., GHEIRATMAND, G. A., ADELI, K., DELAVIRA, A., & MAJDZADEH, R. 2006. Paediatric metabolic syndrome and associated anthropometric indices: the CASPIAN Study. *Acta paediatrica,* 95:1625–1634.

KELISHADI, R. 2007. Childhood Overweight, Obesity, and the Metabolic Syndrome in Developing Countries. *Epidemiologic reviews*, 29(1):62-76.

KELISHADI, R., ARDALAN, G., GHEIRATMAND, R., DELI, K., DELAVARI, A., & MAJDZADEH, R. 2006. Paediatric metabolic syndrome and associated anthropometric indices: *Acta Paediatrica: International journal of paediatrics*, 95(12):1625–1634.

KHAN, F., GREEN, F. C., FORSYTH, J. S., GREENE, S. A., MORRIS, A. D., & BELCH, J. J. F. 2003. Impaired microvascular function in normal children: effects of adiposity and poor glucose handling**.** *Journal of physiology*, 551(2):705 –711.

KLEIN, S. 2004. The case of visceral fat: argument for the defense (Commentary) *Journal of clinical investigation*, 113(11):1530-1532.

KNITTLE, J. L., TIMMERS, K., GINSBERG-FELLNER, F., BROWN, R.E. & KATZ, D. P. 1979. The growth of adipose tissue in children and adolescents. Cross-sectional and longitudinal studies of adipose cell number and size. *The journal of clinical investigation,* 63:239–246.

KOTARI, K., NISHIDA, M., YAMASHITA, S., FUNAHASHI, T., FUJIOKA, S., TOKUNAGA, K., ISHIKAWA, K., TAMI, S., & MATSUZAWA, Y. 1997. Two decades of annual medical examinations in Japanese obese children—do obese children grow into obese adults? *International journal of obesity*, 21:912–921.

KRUGER, H. S., MARGETTS, B. M., VORSTER H. H. 2004. Evidence for relatively greater subcutaneous fat deposition in stunted girls in the North West Province, South Africa, as compared with non-stunted girls. *Nutrition*, 20:564-569.

KRUGER, H.S., PUOANE T, SENEKAL, M., VAN DER MERWE, M. T. 2005. Obesity in South Africa: challenges for government and health professionals. *Public health nutrition*, 8:491–500.

KRUGER, H.S., SWART, R., LABADARIOS, D., DANNHAUSER A. & NEL, J.H. 2007. Anthropometric status. In: LABADARIOS, D (ed). *The National Food Consumption Survey Fortification Baseline* (*NFCS-FB*)*: Children aged 1-9 years, South Africa, 2005*. Pretoria, Department of Health.

KRUGER, R., KRUGER, H. S., MACINTYRE, U. E. 2006. The determinants of overweight and obesity among 10- to 15-year-old schoolchildren in the North West Province, South Africa—The THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) Study. *Public health nutrition*, 9(3): 351-358.

KUO, L. E., KITLINSKA, J. B., TILAN, J. U., LI, L., BAKER, S. B., JOHNSON, M. D., LEE, E. W., BURNETT, M. S., FRICKE, S. T., KVETNANSKY, R., HERBERT HERZOG, H., & ZUKOWSKA, Z. 2007. Neuropeptide Y acts directly in the periphery on fat tissue and mediates stress-induced obesity and metabolic syndrome. *Nature Medicine,* 13:803-811.

KUPERMINC, M. N., & STEVENSON, R. D. 2008. Growth and nutrition disorders in children with cerebral palsy. *Developmental disabilities research reviews,* 14(2):137–146.

LABADARIOS, D., STEYN, N.P., MAUNDER, E., MACINTYRE, U., SWART, R., GERICKE, G., HUSKISSON, J., DANNHAUSER, A., VORSTER, H.H. & NESAMVUNI, E. A. 2000. The National Food Consumption Survey (NFCS): Children aged 1-9 years, South Africa, 1999. Pretoria, Department of Health.

LAMBERT, M., DELVIN, E. E., PARADIS, G., O"LOUGHLIN, J., HANLEY, J. A., LEVY, E. 2004. C-reactive protein and features of the metabolic syndrome in a population based sample of children and adolescents. *Clinical chemistry,* 50:762-1768.

LAVINE, J. E., & SCHWIMMER, J. B. 2004. .Nonalcoholic fatty liver disease in the pediatric population. *Clinics in liver disease,* 8(3*):*549-558.

LEE, Y., NASEEM, B.H. PARK, B. H., GARRY, D. J., RICHARDSON, J. A., SCHAFFER, J. E., & Unger, R. H. 2006. Alpha-lipoic acid prevents lipotoxic cardiomyopathy in acyl CoA-synthase transgenic mice. *Biochemical and biophysiological research communications,* 344:446–452.

LEMIEUX, S., DESPRÉS, J P., MOORJANI, S., NADEAU, A., THERIAULT, G., PRUDHOMME, D., TREMBLAY, A., BOUCHARD, C., & LUPIEN, P. J. 1994. Are gender differences in cardiovascular disease risk factors explained by the level of visceral adipose tissue? *Diabetologica,* 37:757-764.

LI, Y., YANG, Y., ZHAI, F., KOK, F.J., ZHAO, W., PIAO, J., ZHANG, J., CUI, Z., M. A. G. 2008. Prevalence of the metabolic syndrome in Chinese adolescents. *British journal of nutrition,* 99:565–570.

LIU, A., HILL, A.P., HU, X., LI, Y., DU, L., XU, Y., BYRNE, N.M., MA, G. 2010. Waist circumference cut-off values for the prediction of cardiovascular risk factors clustering in Chinese school-aged children: a cross-sectional study. *BMC public health,* 10:82

LUCAS, A. 1991. Programming by early nutrition in man. In The Childhood Environment and Adult Disease, pp. 38–55 (BOCK, G. R., & WHELAN, J. eds). Chichester, West Sussex: Wiley.

MANARY, M. J., & SOLOMONS, N. W. 2004. Public Aspects of Undernutrition. (*In* GIBNEY, M, J., MARGETTS, B. M., KEARNEY, M. J., & ARAB, L. *ed*. Public Health Nutrition. Tumbridge Wells, Kent. Gray Publishing. p178-191.

MÄRIN. P, ANDERSSON, B., OTTOSSON, M., OLBE, L., CHOWDHURY, B., KVIST, H., HOLM, G., SJÖSTRÖM, L., & BJÖRNTORP, P. 1992. The morphology and metabolism of intra-abdominal adipose tissue in men. *Metabolism,* 41:1242–1248.

MARTINEZ, E., DEVESA, M., BACALLAO, J., & AMADOR, M. 1994. Percentiles of the waist-hip ratio in Cuban scholars aged 4.5 to 20.5 years. *International journal of obesity and related metabolic disorders,* 18: 557–560.

MASON, C & KATZMARYZK, P.T. 2010. Waist circumference thresholds for the prediction of cardiometabolic risk: is measurement site important? *European journal of clinical nutrition,* 82:1-6.

MASON, J.B., SANDERS, D., MUSGROVE, P., GALLOWAY, S & GALLOWAY, R. 2006. Community health and nutrition programs. (*In* Jamison, D.T., Breman, J.G., MEASHAM, A.R., ALLEYNE, G., CLAESON, M., EVANS, D.B., JHA, P., MILLS, A., MUSGROVE, P., ed. Disease control priorities in developing countries. NewYork and Washington, DC: Oxford University Press and World Bank. p.1053–1074.

MATSHA, T., HASSAN, S., BHATA, A., YAKO, Y., FANAMPE, E., SOMERS, A., HOFFMANN, M., MOHAMMED, Z., & ERASMUS, R. T. 2009. Metabolic syndrome in 10–16-year-old learners from the Western Cape, South Africa: Comparison of the NCEP ATP III and IDF criteria. *Atherosclerosis,* 205(2):363-366.

MATSUZAWA, Y., NAKAMURA, T., SHIMOMURA, I., KOTANI, K. 1995. Visceral fat accumulation and cardiovascular disease. *Obesity research,* 3(Suppl. 5):*645S*–647S.

MCCARTHY, H D., & ASHWELL, M. 2006. A study of central fatness using waist-to-height ratios in UK children and adolescents over two decades supports the simple message--'keep your waist circumference to less than half your height'. *International journal of obesity,* (6):988-992.

MCCARTHY, H. D., JARRETT, K. V., CRAWLEY, H. F. 2001. The development of waist circumference percentiles in British children aged 5.0–16.9 y. *European journal of clinical nutrition,* 55:902–907.

MCMILLEN, I. C., MUHLHAUSER, B. S., DUFFIELD, J. A., & YUEN, B. S. 2004. Prenatal programming of postnatal obesity: foetal nutrition and the regulation of leptin synthesis and secretion before birth. *Proceedings of the nutrition society,* 63:405–412.

Medical Research Council (MRC). Umthente uhlaba usamila: the 1st South African National Youth Risk Behaviour Survey. Cape Town: South African Medical Research Council, 2002. http://www.mrc.ac.za/healthpromotion/healthpromotion.htm Date of access 16 Nov. 2010

MEG, A., LAMBERT, M., & SHARWOO, K. A**.** 2006. Obesity and overweight in South African primary school children-The Health of the Nation Study. *South African medical journal,* 96(5):439-444.

MELTZER, A. A., & EVERHART, J. E. 1997. Association between diabetes and elevated serum alanine aminotransferase activity among Mexican Americans. *American journal of epidemilogy,* 146:*565*–571.

MENGHETTI, E., CAIRELLA, G., CASTORO, F., CENSI, L., D'ADDESA, D, MARTONE, D., ROSANO, A., SCANU, A., SONNI, L., & SPAGNOLO, A. 2007. Increase of hypertension among adolescents in Rome. *Minerva paediatrica,* 59:1–5.

MESSIAH S E., CARRILLO, A., GARIBAY, N., LOPEZ-MITNIK, G., COSSIO, S., & ARHEART, K. L. 2009. Prevalence of Metabolic Syndrome in US-Born Latin and Caribbean Youth. *Journal of immigrant and minority youth,* 11(5):366-371.

MISRA, A., & VIKRAM, N. K. 2003. Clinical and pathophysiological consequences of abdominal adiposity and abdominal adipose tissue depots. *Nutrition,* 19(5):457-466.

MISRA, A., & KHURANA, L. 2008. Obesity and the metabolic syndrome in developing countries. *Journal of clinical endocrinology & metabolism,* 93:s9-s30.

MOLLENTZE W. F., MOORE A. J., STEYN A. F., JOUBERT G., STEYN K., OOSTHUIZEN G. M., & WEICH D. J. V. 1995. Coronary Heart Disease Risk Factors in a Rural and Urban Orange Free State Black Population. *South African medical journal,* 85:90–96.

MONTAGUE, C. T., & O'RAHILLY, S. 2000. The perils of portliness: causes and consequences of visceral adiposity. *Diabetes,* 49:883–888.

MONYEKI, K. D, VAN LANTHE, F. J., STEYN, N. P. 1999. Obesity: does it occur in African children in a rural community in South Africa? *International journal of epidemiology*, 8:287-292.

MORENO, L.A., FLETA, J, MUR, L., RODRIQUEZ, G., SARRIA, A., & BUENO, M. 1999. Waist circumference values in Spanish children–gender related differences. *European journal of clinical nutrition,* 53:429–433.

NAWARYCZ, L.O., KRYZANIAK, A., STAWIŃSKA-WITOSZYŃSKA, M., SZILAGYI-PĄGOWSKA, M., KOWALSKA, M., KRZYCH, L., NAWARYCZ, T. 2010. Percentile distribution of waist circumference for 7-19 year old Polish children and adolescents. *Obesity reviews*, 11(4):281-288

NESS, A. R. 2004. The Avon Longitudinal Study of Parents and Children (ALSPAC)--a resource for the study of the environmental determinants of childhood obesity. *European journal of endocrinology,* 151(suppl 3):U141-U149.

NIELSEN, S., GUO, Z., JOHNSON, C. M., HENSRUD, D. D., & JENSEN, M. D. 2004. Splanchnic lipolysis in human obesity. *Journal of clinical investigation*, 113:1582–1588.

NINOMIYA, J. K., L'ITALIEN, G., CRIQUI, M. H., WHYTE, J. L., GAMST, A., & CHEN, R. S. 2004. Association of the metabolic syndrome with history of myocardial infarction and stroke in the Third National Health and Nutrition Examination Survey. *Circulation,* 109:42–46.

OGDEN, C. L., CARROLL, M. D., CURTIN, L. R., MCDOWELL, M. A., TABAK, C. J., & FLEGAL, K. M. 2006. Prevalence of overweight and obesity in the United States, 1999-2004. *The journal of American medical association,* 295:1549-1555.

ONG, K. K., AHMED, M. L., EMMETT, P. M., PREECE, M. A., & DUNGER, D. B. 2000. Association between postnatal catch-up growth and obesity in childhood: prospective cohort study. *British medical journal*, 320:967–971.

ONUORAH, C. E., & AYO, J. A. 2003. Food taboos and their implications on developing nations like Nigeria-review. *Nutrition and food science*, 33(5):235-240.

OSTORWSKA, N.L., KRZYŻANIAK, A., STAWIŃSKA-WITOSZYŃSKA, B., KRZYWIŃSKA-WIEWIOROWSKA, M., SZILAGYI-PĄGOWSKA, I., KOWALSKA, M., KRZYCH, Ł et al. 2010. Percentile distribution of waist circumference for 7-19 year old Polish children and adolescents. *Obesity Reviews,* 11(4): 281-288.

OUCHI, N., SHIBATA, R., & WALSH, K. 2006. Cardioprotection by adiponectin. *Trends in cardiovascular medicine,* 16:141-146.

PLOURDE, G. 1997**.** The role of radiologic methods in assessing body composition and related metabolic parameters. *Nutrition reviews,* 55**:**289-296.

POIRIER, P., GILES, T. D., BRAY, G. A., HONG, Y., STERN, J. S., PI-SUNYER, X., & ECKEL, R. H. 2006. Obesity and cardiovascular disease: pathophysiology, evaluation, and effect of weight loss: an update of the 1997 American Heart Association Scientific Statement on Obesity and Heart Disease from the Obesity Committee of the Council on Nutrition, Physical Activity, and Metabolism. *Circulation*, 113:898-918.

POLONSKY, K. S., STURIS, J., & BELL, G. I. 1996. Non-insulin-dependent diabetes mellitus: a genetically programmed failure of the beta cell to compensate for insulin resistance. *New England journal of medicine,* 334:777-783.

POPKIN, B. M., CONDE, W., NINGQI HOU, N., & MONTEIRO, C. 2006. Is There a Lag Globally in Overweight Trends for Children Compared with Adults?* *Obesity*, 14:1846–1853.

POPKIN, B. M., RICHARDS, M. K., & MONTIERO, C. A. 1996. Stunting is associated with overweight in children of four nations that are undergoing the nutrition transition. *Journal of nutrition*, 26:3009–3016.

POULIOT, M. C., DESPRES, J. P., NADEAU, A., MOORJANI, S., PRUD HOMME, D., LUPIEN, P. J., TREMBLAY, A., & BOUCHARD, C. 1992. Visceral obesity in men: Associations with glucose tolerance, plasma insulin and lipoprotein levels. *Diabetes,* 41:826-834.

REAVEN, G. M. 1988. Banting lecture 1988: role of insulin resistance in human disease. *Diabetes*, 37: 1595–1607.

REDDY, J. K., & RAO, M. S. 2006. Lipid metabolism and liver inflammation. Fatty liver disease and fatty liver oxidation. *American journal of physiology, gastrointestinal and liver physiology,* 290(5):852-858.

REDDY, S.P., JAMES, S., SEWPAUL, R., KOOPMAN, F., FUNANI, N.I., SIFUNDA, S.et al Umthente Uhlaba Usamila - The South African Youth Risk Behaviour Survey 2008. Cape Town: South African Medical Research Council; 2010. http://www.mrc.ac.za/healthpromotion/healthpromotion.htm Date of access 16 Nov. 2010

REILLY, J J. 2007. Childhood obesity-an overview. *Children and society,* 21(5):390-396.

REINEHR, T., DE SOUSA, G., TOSCHKE, A. M., & ANDLER, W. 2007. Comparison of metabolic syndrome prevalence using eight different definitions: a critical approach. *Archives of disease in childhood,* 92:1067-1072.

RENEHAN, A.G., FRYSTYK, J. & FLYVBJERG, A. 2006. Obesity and cancer risk: the role of the insulin-IGF axis. *Trends in endocrinology and metabolism*,17(8):328-336.

REYES, H., PEREZ-CUEVAS, R., SANDOVAL, A., CASTILLO, R., SANTOS, J. L., DOUBOVA, S., & GUTIEREZ, G. 2004. The family as a determinant of stunting in children living in conditions of extreme poverty: a case control study. *BMC public health* 4:57

RIGO, J., NYAMUGABO, K., PICAUD, J C., GERARD, P., PIELTAIN, C & DE CURTIS, M. 1998. Reference values of body composition obtained by dual energy X-ray absorptiometry in preterm and term neonates. *Journal of paediatric gastroenterology and nutrition,* 27:184–190.

ROBERTS, D.L., DIVE, C. & RENEHAN, A.G. 2010. Biological mechanisms linking obesity and cancer risk: New Perspectives. *Annual review of medicine,* 61: 301-316.

RODRÍGUEZ, G., SAMPER, M. P., VENTURA, P., MORENO, L. A., OLIVARES, J. L. & PÉREZ-GONZÁLEZ, J. M. 2004. Gender differences in newborn subcutaneous fat distribution. *European journal of paediatrics,* 163:457–61.

ROLLAND-CACHERA, M.F., DEHEEGER, M., BELLISLE, F., SEMPE, M., GUILLOUD-BATAILLE, M., & PATOIS, E. 1984. Adiposity rebound in children: a simple indicator for predicting obesity. *American journal of clinical nutrition,* 39:129–135.

RUDERMAN, N., CHISHOLM, D., PII-SUNYER, X., & SCHNEIDER, S. 1998. The metabolically obese, normal-weight individual revisited. *Diabetes***,** 47: 699–713.

SACHDEV, H. S., FALL, C. H., OSMOND, C., LAKSHMY, R., BISWAS, S. K. D., LEARY, S. D., REDDY, K. S., BARKER, D. J., & BHARGAVA, S, K. 2005. Anthropometric indicators of body composition in young adults: relation to size at birth and serial measurements of body mass index in childhood in the New Delhi birth cohort. *American journal of clinical nutrition,* 82:456–466.

SALTZMAN, E. & ROBERTS, S. B. 1995. The role of energy expenditure in energy regulation: Findings from a decade of research. *Nutrition reviews,* 53:209-220.SANJAY, A., &

HERBERT, B. L. 2002. Management of Non-alcoholic Steatohepatitis: An Analytic Review. *Journal of clinical gastroenterology*, 35(3):253-261.

SAWAYA, A. L., DALLAL, G., SOLYMOS, G., DESOUSA, M. H., VENTURA, M. L., ROBERTS, S. B., & SIGULEM, D. M. 1995. Obesity and malnutrition in SAYER, A. A., & COOPER, C. 2008. Foetal programming of body composition and musculoskeletal development. *Early human development*, 81:735-744.

SAWAYA, L., & ROBERTS, S. 2003. Stunting and future risk of obesity: principal physiologic mechanisms-review. *Cad saude publica,* 19(suppl 1):s21-s28.

SCHAFFLER, A., MULLER-LADNER, U., SCHOLMERICH, J., & BUCHLER, C. 2006. Role of Adipose Tissue as an Inflammatory Organ in Human Diseases. *Endocrologic reviews,* 27:449-467.

SCHEEN, A. J. 2008. The future of obesity: new drugs versus lifestyle interventions. *Expert opinion in investigational drugs*, 17(3):263-267.

SCHROEDER, D. G., MARTORELL, R., & FLORES, R. 1999. Infant and child growth and fatness and fat distribution in Guatemalan adults. *American journal of epidemiology*, 149:177-185.

SEIDELL, J. C**.,** DOAK, C. M**.,** DE MUNTER, J. S**.,** KUIJPER, L .D**.,** & ZONNEVELD, C**.** 2006. Cross-sectional growth references and implications for the development of an international standard for school aged children and adolescents. *Food and nutrition bulletin*, 27(4 Suppl Growth Standard):S189-198.

SENBANJO, I. O., NJOKANMA, O. F., & OSHIKOYA, K. A. 2009. Waist Circumference Values of Nigerian Children and Adolescents. *Annals of nutrition and metabolism*, 54:145-150.

SHIBATA, R., IZUMIYA, Y., SATO, K., PAPANICOLAOU, K., KIHARA, S., COLUCCI, W. S., SAM, F., OUCHI, N., & WALSH, K. 2007. Adiponectin protects against development of systolic dysfunction following myocardial infarction. *Journal of molecular and cellular cardiology,* 42(6):1065-1074.

SHIELDS, M. 2004. Overweight Canadian children and adolescents. Canadian Community Health Survey: Nutrition Analytical Studies and Reports. Ottawa, Canada.

SICHEIRI, R., SIQUEIRA, K. & MOURA, A. 2000. Obesity and abdominal fatness associated with undernutrition early in life in a survey in Rio de Janeiro. *International Journal of obesity and related metabolic disorders*, 24:614-618.

SINGH, R., BHANSALI, S., SIALY, R., & AGGRWAL, A**.** Prevalence of metabolic syndrome in adolescents from a north Indian population. 2007. *Diabetic medicine, 24*(2):195-199.

SINHA, R., FISCH, G., TEAGUE, B., TAMBORLANE, W. V,. BANYAS, B., ALLEN, K., SAVOYE, M., RIEGER, V., TAKSALI, S., BARBETTA, G., SHERWIN, R. S., & CAPRIO, S. 2002. Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *New England journal of medicine*, 346(11):802–810.

SNEHALATHA, C., MUKESH, B., SIMON, M., VISWANATHAN, V., HAFFNER, S. M., RAMACHANDRAN, A. 2003. Plasma Adiponectin Is an Independent Predictor of Type 2 Diabetes in Asian Indians. *Diabetes care, 26*(12):3226-3229.

SNIJDER, M. B., VISSER, M., DEKKER, J. M., SEIDELL, J. C., FUERST, T, TYLAVSKY, F., CAULEY, J., LANG, T., NEVITT, M., & HARRIS. T. B. 2002. The prediction of visceral fat by dual-energy X-ray absorptiometry in the elderly: a comparison with computed tomography and anthropometry. *International journal of obesity*, 26(7):984-993.

SOUSA, M., BRAS-SILVA, C., & LEITE-MOREIRA, A. 2009. The role of leptin in the regulation of energy balance. *Acta medica portuguesa*, 22(3):291-298.

STAMATAKIS, E., PRIMATESTA, P., CHINN, S., RONA, R., & FALASCHETI, E. 2005. Overweight and obesity trends from 1974 to 2003 in English children: what is the role of socioeconomic factors? *Archives of disease in childhood*, 90:999-1004.

STEYN, N. P., LABADARIOS, D., MAUNDER, E., NEL, J., & LOMBARD, C. 2005. Secondary anthropometric data analysis of the National Food Consumption Survey of South Africa: the double burden. *Nutrition*, 21:4-13.

STOVITZ, S. D., SCHWIMMER, J. B., MARTINEZ, H., & STORY, M. T. 2008. Pediatric Obesity: The Unique Issues in Latino–American Male Youth. *American journal of preventative medicine*, 34:153-160.

STRACHAN, K. 1999. South Africa's nutritional status in undernutriton South Africa. Available at: http://www.healthlink.org.za. Date of access: 29 Oct 2009.

Third report of the National Cholesterol Education Program (NCEP) expert panel on detection, evaluation, and treatment of high blood cholesterol in adults (Adult Treatment Panel III). Final report. 2002. *Circulation*, 106:3143–3421.

TIAN, R., MUSI, N., D'AGOSTINO, J., HIRSHMAN, M, F., GOODYEAR, L. J. 2001. Increased adenosine monophosphate-activated protein kinase activity in rat hearts with pressure-overload hypertrophy. *Circulation*, 104:1664-1669.

TRAYHURN, P. 2007. Adipocyte biology. *Obesity reviews*, 8(suppl. 1):41-44

TRUJILLO, M E., and SCHERER, P E. 2006. Adipose tissue–derived factors: impact on health and disease, *Endocrinology reviews*, 27:762–778.

UAUY, R., ALBALA, C & KAIN, J. 2001. Obesity trends in Latin America: transiting from under- to overweight. *Journal of nutrition*, 131(3):893S-899.

Undernutrition. 2005. British Nutrition Foundation. Available at: http://www.nutrition.org.uk/home.asp. Date of access: 23 Mar 2008.

UNGER R, H. 2003. Mini review: Weapons of lean mass destruction: The role of ectopic lipids in the metabolic syndrome. *Endocrinology*, 144:5159-5165.

UNICEF Progress for children-a report card on nutrition number 4, May 2006 available at http://www.unicef.org/progressforchildren/2006n4/. Date of access 30 Jul. 2009.

UTHAYA, S., THOMAS, E. L., HAMILTON, G., DORE, C. J., BELL, J., & MODI, N. 2005. Altered adiposity after extremely preterm birth. *Paediatric research*, 57:211-215.

VELLA, V. A., TOMKINS, J., & MARSHALL, T. 1992. Determinants of child mortality in south west Uganda. *Journal of biosocial sciences*, 2:103-111.

WAJCHENBERG, B. L. 2000. Subcutaneous and Visceral Adipose Tissue: Their Relation to the Metabolic Syndrome. *Endocrine reviews*, 21(6):697-738.

WALKER, S. P., GASKIN, P., POWELL, C. A., BENNETT, F. I., FORRESTER, T. E., GRANTHAM-MCGREGOR, S. 2001. The effects of birth weight and postnatal linear growth retardation on blood pressure at age 11-12 years. *Journal of epidemiology and community health*, 55:394-398.

WANG, P., MARIMAN, E., RENES, J., & KEIJER, J. 2008. The secretory function of adipocyte in the physiology of white adipose tissue. *Journal of cell physiology*, 216(1):3-13.
WATERLOW, J. C., 1994. Metabolic changes. In: Protein Energy Malnutrition (J. C. Waterlow, ed.), pp.83-97, London: Edward Arnold.

WEISS, R. 2007. Fat distribution and storage: how much, where, and how? *European journal of endocrinology*, 1579(suppl 1):S39-S45.

WEISS, R., & CAPRIO, S. 2008. Obesity and Type 2 Diabetes in Youth. (*In* Dabalea, D., & KLINGENSMITH, G H., *ed*. Epidemiology of Paediatric Adolescent Diabetes. New York, USA. Informa Health care.

WEISS, R., DUFOUR, S., TAKSALI, S. E., TAMBORLANE, W. V., PETERSEN, K. F., BONADONNA, R. C., BOSELLI, L. M., BARBETTA, G., ALLEN, K., RIFE, F., SAVOYE, M., DZIURA, J., SHERWIN, R., SHULMAN, G. I., & CAPRIO, S. 2003. Pre-diabetes in obese youth a syndrome of impaired glucose tolerance, severe insulin resistance, and altered myocellular and abdominal fat partitioning. *Lancet,* 362: 951–957.

WELLS, J. C. K., CHOMTHO, S., & FEWTRELL, M. S. 2007. Programming of body composition in early growth and nutrition. *Proceedings of the nutrition society*, 66:423-434.

WILLIAMS, D. E., CADWELL, B. L., CHENG, Y. J., COWIE, C. C., GREGG, E. W., GEISS, L. S., ENGELGAU, M. M., NARAYAN, V., & IMPERATORE, G. 2005. Prevalence of

impaired fasting glucose and its relationship with cardiovascular disease risk factors in US adolescents, 1999–2000. *Paediatrics*, 116: 1122–1126.

WOLIN, K.Y., CARSON, K. & COLDITZ, G.A. 2010. Obesity and cancer. *The oncologist*, 15:556-565.

WORLD HEALTH ORGANIZATION (WHO). 1990. Diet, nutrition, and the prevention of chronic diseases (WHO Technical Report Series, No.797 - TRS 797). WHO, Geneva, Switzerland.

WORLD HEALTH ORGANIZATION (WHO). 1999. International society of hypertension guidelines for the management of hypertension, *Journal of hypertension*, 17:151–183.

XU, A., WANG, Y., KESHAW, H., XU, L Y., LAM, K S L. and COOPER, G J S. 2003. The fat-derived hormone adiponectin alleviates alcoholic and non-alcoholic fatty liver diseases in mice. *The Journal of clinical investigation*, 112(1):91-100.

YAN, L. L., DAVIGLU,S. M. L., LIU, K., PIRZADA, A., GARSIDE, D. B., SCHIFFER, L., DYER, A. R., GREENLAND, P. 2004. BMI and health-related quality of life in adults 65 years and older. *Obesity research,* 12:69–76.

YANOVSKI, J. A., YANOVSKI, S. Z., FILMER, K. M., HUBBARD, V. S., AVILA, N., LEWIS, B., REYNOLDS, J .C., & FLOOD, M. 1996. Differences in body composition of black and white girls. *American journal of clinical nutrition*, 64:833–839.

YOSHINAGA, M., TANAKA, S., SHIMAGO, A., SAMESHIMA, K., NISHI, J., NOMURA, Y., KAWANO, Y., HASHIGUCHI, J., ICHIKI, T., & SHIMIZU, S. 2005. Metabolic Syndrome in Overweight and Obese Japanese Children. *Obesity research,* 13:1135–1140.

ZAFON, C. 2007. Oscillations in total body fat content through life: an evolutionary perspective, *Obesity reviews*, 8(6):525–530.

ZAMBONI, M., ARMELLINI, F,. & MULLER, D. 1996. Methodological and clinical aspects of subcutaneous fat distribution. (*In* Angel, A., Anderson, H., & Bouchard, C. Progress in

obesity research (7) Proceedings of the Seventh International Congress on Obesity, Toronto, Canada, August 20–25, 1994, John Libbey and Co, Toronto, p.145.

ZANNOLLI, R., & MORGESE, G. 1996. Waist percentiles: a simple test for atherogenic disease? *Acta Paediatica,* 85:1368–1369.

ZIMMET, P., ALBERTI, K. G., KAUFMAN, F., TAJIMA, N., SILINK, M., ARSLANIAN, S., WONG, G., BENNETT, P., SHAW, J., & CAPRIO, S. IDF Consensus Group. 2007. The metabolic syndrome in children and adolescents—an IDF consensus report, *Paediatric diabetes,* 8**:**299–306.

3

BODY FAT DISTRIBUTION IN STUNTED COMPARED TO NON-STUNTED BLACK SOUTH AFRICAN CHILDREN FROM TWO RURAL COMMUNITIES

For many growth retarded persons, an adaptive mechanism may only result in excess fat deposition that promotes chronic disease rather than providing a safety mechanism for famines that never return (*Hoffman et al., 2007*).

Title: Body fat distribution in stunted compared to non-stunted black South African children from two rural communities

Running head: Body composition in stunted and non-stunted children.

Type of manuscript: Original article

Word count: Abstract: 228

Manuscript: 3531

Number of references: 26

Tables: 4

Boitumelo S Motswagole[1]MSc, H Salome Kruger[1]PhD, Mieke Faber[2]PhD, Kotsedi D Monyeki[3] PhD.

Conflict of interest statement: None

[1]Centre of Excellence for Nutrition, North-West University, Private Bag X6001, Potchefstroom 2520, South Africa. Tel: +27 18 299 2466, Fax: +27 18 299 2464; [2]Nutritional Intervention Research Unit, Medical Research Council, PO Box 19070, Tygerberg 7505, South Africa; [3]Chronic Diseases of Lifestyle Unit, Medical Research Council, PO Box 19070, Tygerberg 7505, South Africa.

Correspondence to: H Salome Kruger, Centre of Excellence for Nutrition, North-West University, Private Bag X6001, Potchefstroom 2520, South Africa. Tel: +27 18 299 2482, Fax: +27 18 299 2464, E-mail: Salome.Kruger@nwu.ac.za

Submitted for publication in the International Journal of Pediatric obesity

Abstract

Objective: To determine body fat distribution for stunted and non-stunted South African children from two rural communities.

Methods: Secondary data analysis of the THUSABANA (n=351; 29% stunted) and Ellisras (n=1760; 5.1% stunted) studies was done to estimate differences between stunted and non-stunted children for global adiposity (body mass index, sum of skinfolds) and localized adiposity (waist-to-height ratio, subscapular-to-triceps ratio) using two-way analysis of covariance and adjusting for age.

Results: Stunted children had a lower body mass index (THUSABANA: 15.7[0.3] vs. 16.5[0.2] p=0.03, boys; 16.6[0.5] vs. 17.7[0.3] p=0.054, girls; Ellisras: (13.9[0.2] vs. 14.4[0.04] p=0.005, boys; 13.7[0.24] vs. 14.5[0.06] p=0.002, girls) and waist circumference (THUSABANA: 56.2[0.7] vs. 59.8[0.5] p=0.0004, boys; 56.6[1.0] vs. 61.5[0.6] p=0.00007, girls; Ellisras: 53.6[0.5] vs. 55.6[0.1] p=0.0002, boys; 53.2[0.6] vs. 55.5[0.1] p=0.003, girls). Waist-to-height ratio was higher in stunted THUSABANA boys (0.42[0.004] vs. 0.41[0.003] p=0.05) and Ellisras boys (0.43[0.004] vs. 0.40[0.008] p<0.00001) and girls (0.42[0.004] vs. 0.40[0.001] p<0.00001). The sum of skinfolds was lower only in stunted girls from Ellisras (15.5[0.8] vs. 17.3[0.2] p=0.03). The subscapular-to-triceps ratio did not differ between stunted and non-stunted children.

Conclusions: Findings of this study demonstrated increased total adiposity in non-stunted children, but trends of increased central adiposity, measured as WHtR in stunted children. More research is needed to establish which adiposity measure is representative of the distribution of body fatness independent of age, race, gender and sexual maturation in children and adolescents.

Keywords: Body composition, stunting, obesity, children, South Africa.

Introduction

Stunting in children is considered a consequence of chronic poor nutrition (1). It is associated with developmental delay and impaired cognitive function and is considered the strongest predictor of child mortality in children younger than five years (2). Stunting remains the most common nutritional disorder affecting children in South Africa, with an estimated national prevalence of 18% in children 1-9 years old (3). It is important to point out that linear growth lost in infancy may not be fully recovered with improved energy intake later on (4). Thus, if calorie intake exceeds expenditure, weight gain may follow preferentially to length gain (5). Therefore, it is likely that stunted children may become obese adults in societies undergoing rapid changes in patterns of diet and physical activity that lead to positive energy balance. In an individual child, the relation between stunting and overweight is not a simple situation of co-existence; epidemiological and experimental evidence is accumulating to indicate a causal relationship (6). Understanding the prevalence and patterns of undernutrition, particularly stunting, the emergence of overweight/obesity in children and adolescents and the concomitant risk for metabolic disease is of critical importance for public health policy (7).

Possible potential mechanisms linking growth retardation and increased adiposity have been suggested, including impaired fat oxidation and the action of cortisol as part of the causal pathway (6). According to Benefice and colleagues (8), body composition, especially fat mass, could be an important component and outcome of long-term stunting. Therefore, body composition assessment is becoming a standard measure in many clinical and nutrition related studies. In African populations, preschool-aged children in particular are exposed to malnutrition, and this may have a major effect on growth and development (8). However,

studies on fatness and relative fat distribution during childhood and adolescence are scarce, especially from rural areas (7, 9). Of particular concern is whether or not increased adiposity is found in stunted children. Therefore, the objective of this study was to compare the body composition of black stunted and non-stunted children from two rural communities of South Africa and investigate whether increased total and central adiposity is found in stunted children.

Methods

Study setting and population

The sample for the current analysis consists of black children from two cross-sectional studies conducted in rural communities of South Africa, namely the *THUSABANA* (*THUSA* stands for Transition and Health during Urbanization of South Africans; *BANA* means children) study in the North West province and the Ellisras Longitudinal Growth and Health Study (ELS) in the Limpopo province. Almost 90% of households in the North West Province had access to piped water within the home and houses were mostly constructed from bricks. Most households used flush toilets and used electricity for cooking. The province is predominantly rural, with 65.1% living in rural areas and 34.9% in urban areas (10). The rate of urbanization is increasing, largely due to the lack of employment opportunities in rural areas. Poverty affects 62% of the population of the North West Province, the second highest provincial figure for South Africa (10). Ellisras is a rural area situated in the Limpopo province. Housing material varies from traditional to mud to brick houses. Piped water is available at community level and sanitation is relatively insufficient with the majority of households using pit latrines. Most homes use open fires for cooking. The people rely heavily on agriculture for household food security (11). Data were collected of 351 rural children aged 10 to 15 years from May 2000 to June 2001 in the THUSABANA study and 1760 children aged 6 to 14 years during May 2000 in the Ellisras area.

Procedures used for sampling and collection of anthropometric data have been reported in detail elsewhere (12, 13). The THUSABANA study was approved by the Ethics Committee of the North-West University (Potchefstroom Campus) and ELS was approved by the Ethics committee of the University of the North and caregivers of all children gave informed consent.

Anthropometric measures

All anthropometric measurements in both studies were done according to standard methods by trained anthropometrists. Weight was measured to the nearest 0.1 kg using electronic scales with children in underwear and bare-feet. Height was measured to the nearest 0.1 cm using a Martin anthropometer in the Ellisras study and an IP 1465 stadiometer in the THUSABANA study. Skinfold thicknesses were measured using a John Bull skinfold caliper in THUSABANA study, whilst a Slim Guide skinfold caliper was used in the Ellisras study. They were measured in duplicate in the THUSABANA study and in triplicate in the Ellisras study, and the mean of the measurements was used in data analysis. Waist circumference (WC) was measured at the narrowest circumference on the waist above the iliac crest and below the lower rib using a non-stretching flexible tape (Lufkin, Apex, NC, USA) in the THUSABANA study and a Rosscraft anthropometric tape (USA) for girths in the Ellisras study. Details of the two studies are summarized in Table 1.

Body composition

The different body composition indices were calculated as shown in Table 2. Body mass index (BMI) and sum of skinfolds (SSF) were used to characterize global adiposity, while WC, waist-

to-height ratio (WHtR) and subscapular-to-triceps ratio (STR) were used to characterize localized adiposity.

Statistical analysis

Data were analyzed using the STATISTICA statistical package (StatSoft, Inc., 2009 (14). Data for the two study areas (THUSABANA and Ellisras) were analyzed separately because preliminary data analysis showed significant differences between the two communities for age and some of the anthropometric indicators. Also data were not split into age groups because some age groups had fewer than 30 children (groups became too small). For each study area, children were stratified into two groups, namely stunted and non-stunted. Stunting was defined as height-for-age Z-score below -2 standard deviations of the World Health Organization/National Center for Health Statistics(WHO/NCHS) reference standards (15). Since the relation between anthropometric measures and body fat distribution is dependent on age, the data were adjusted for age to reduce the observed variation between groups caused by age. Descriptive statistics were calculated for all children by gender for each study area. Differences between stunted and non-stunted children with respect to adiposity measures were determined using analysis of covariance (*ANCOVA*), adjusting for age. Results were considered statistically significant at $P < 0.05$.

Results

Table 3 shows the descriptive characteristics of the children from the two study areas. The prevalence of stunting in THUSABANA was 29% versus 5.1% in Ellisras. THUSABANA children were older, had higher mean values for weight, height and WC, BMI, WHtR. Table 4

shows the means of different body composition measures by group and gender. Non-stunted children from THUSABANA had significantly higher values for almost all anthropometric measures in both boys and girls, except for SSF in boys and WHtR in girls which did not differ between stunted and non-stunted children. However, stunted boys had a significantly higher WHtR than non-stunted boys. In the Ellisras study area, SSF and STR in both boys and girls did not differ between stunted and non-stunted children. WHtR was significantly higher in stunted boys and girls while WC and BMI was higher in non-stunted boys and girls.

Discussion

Results indicate disparities in the prevalence of stunting between the two communities, with the THUSABANA study in the North West Province having a 29.9% stunting prevalence, versus 5.1% in the Ellisras study in the Limpopo Province. In the 2002 Youth Risk Behaviour Survey 14.8% of high school children in the North West Province and 11.1% of children in Limpopo Province were stunted (16). In 2008 the North West Province reported the highest rates of reported child hunger in the country (1). Globally poverty-related malnutrition is the single most common cause of growth retardation. (17). The high rate of child hunger in the province could, therefore, explain the high prevalence of stunting in the THUSABANA children. The adverse socioeconomic environment and the low levels of food availability compromise and probably delay the physical development of the affected children in all phases of growth. On the contrary, this study showed low stunting rates in Ellisras, possibly because of greater food security in the households due to access to land for subsistence agriculture, despite high rates of unemployment and income poverty. According to M'marete (11), Limpopo Province is one of South Africa's richest agricultural areas.

This study determined various adiposity measures for stunted and non-stunted children. Results indicate that BMI was significantly higher in non-stunted children from both communities. Despite the use of BMI in defining childhood obesity, its validity as an indicator of fatness is questionable, especially in stunted populations (18). Several reports also suggest that BMI may not be a valid tool for assessing body composition in growing children (19). More important though, given that the prevalence of obesity is increasing in countries with a high prevalence of stunting, it is necessary to determine whether growth retardation influences the relationship between BMI and body fatness to avoid the misclassification of children (20). This does not, however, stop investigators from using BMI to categorize children into normal-weight or overweight categories, especially when studying large populations or when access to alternative methods of body composition assessment is limited. Caution should, however, be used if BMI is applied as a measure of body fat, regardless of whether normal-height or stunted children are studied (6). Sum of skinfolds may be a better indication of body fatness. The SSF was not significantly different in stunted and non-stunted boys in the two study communities, but SSF was significantly greater for non-stunted girls in both study communities than for stunted children. Two well-established sources of error may occur in the measurement of skinfolds (21). These are inter-observer error (21) and the potential gender, ethnic and maturation-related changes in body composition, which may affect the relationship between skinfold measures and body fat mass in subgroups of the population.

Stunted children had a smaller WC, but a greater WHtR (except for the THUSABANA girls). The question then is which of these measures is giving a true reflection of abdominal obesity, but results from this study adds to the existing information that WHtR might be a better measure

of central adiposity on both adults and children. Panjikkaran and co-workers (22) argue that 'waist circumference as a measure of obesity and overweight status suffers from the disadvantage of not considering important criteria such as body weight and height'. According to Cameron and colleagues (23), not testing the quantitative association between WC and height in order to create an independent index of WC has resulted in the erroneous belief that WC is the best indicator of risk throughout childhood and adolescence. They argue that WC should become more important as an indicator of fat deposition during puberty when most of the centralization of fat occurs. WHtR has been reported to be an effective predictor of metabolic risk; even then, a perfect relation of WHtR with obesity or overweight status is yet to be standardized. It has been shown that individuals with WHtR scores above 0.5 are likely to fall in the overweight or obese category, irrespective of age (24). A review on the prevalence of obesity among adolescents by De Moraes and colleagues (25) concluded that there is no consensus on the methodology and criteria to be used for classifying abdominal obesity. Possible potential mechanisms linking growth retardation to increased central adiposity have been suggested, including impaired fat oxidation and the action of cortisol as part of the causal pathway (6). No significant difference in STR as a measure of truncal obesity was observed between the stunted and non-stunted children in both the THUSABANA and Ellisras studies, but absolute values indicate higher STR in stunted girls from THUSABANA. A longitudinal study in Senegalese adolescents showed a greater accumulation of subcutaneous fat in the upper part of the body (the trunk and arms) in those who were stunted, irrespective of the overall quantity of subcutaneous fat. The authors argue that although there is as yet no precise explanation for this greater deposit of fat in the upper part of the body in stunted adolescents, it may be attributed to

complex hormonal adjustments that occur with the onset of puberty and which could be affected by malnutrition (8).

Possible limitations in this study include the cross-sectional nature of data; causality of excess fat accumulation due to stunting cannot be established. These measures of fat distribution have not been extensively studied in children and it is, therefore, not known which one is the most valid measure to describe truncal or central adiposity in children. When deciding on the anthropometric measure to use to determine fat distribution in children, various factors need to be considered. These factors include comparison with measures such as dual energy x-ray absorptiometry (DEXA), ability to characterize accurately the distribution of body fat independent of other factors such as gender and the ease of use in a practical research setting (26). In conclusion, more research is needed to establish which adiposity measure is representative of the distribution of body fatness independent of age, race, gender and sexual maturation in children and adolescents.

Acknowledgements

The authors thank Prof. HS Steyn for statistical advice and the children and research teams from the THUSABANA and Ellisras studies. Financial support was received from the South African Sugar Association.

References

1. Berry L, Hall K. Child Health: Nutrition in Kibel M, Lake L, Pendlebury S, Smith C. (eds) South African Child gauge 2009/2010. Cape Town: Children's Institute, University of Cape Town. Available at http://www.ci.org.za. Accessed on 6 Oct 2010.
2. Pelletier DL. The relationship between child anthropometry and mortality in developing countries: Implications for policy, programs and future research. J Nutr. 1994; 124 (supplement): 2047S-2081S.
3. Labadarios D. National food consumption survey-fortification baseline: the knowledge, attitude, behavior and procurement regarding fortified foods, a measure of hunger and the anthropometric and selected micronutrient status of children aged 1-9 years and women of child bearing age: South Africa 2005. Pretoria: Directorate Nutrition, Department of Health, 2007.
4. Martorell R, Kettel-Khan L, Schroeder D. Reversibility of stunting: epidemiological findings in children from developing countries. Eur J Clin Nutr. 1999*;* 48: S45–S57.
5. Kain J, Uauy R, Taibo L-M, Albala C. Trends in weight and BMI of 6-year-old children during the nutrition transition in Chile. Obes Res. 2005; 13: 2178–2186
6. Hoffman DJ, Martins PA, Roberts SB, Sawaya AL. Body fat distribution in stunted compared with normal height children from the shanty towns of Sao Paulo, Brazil. Nutrition, 2007; 23(9):640-646.
7. Kimani-Murage E, Kahn K, Pettifor M et al,. The prevalence of stunting, overweight and obesity, and metabolic disease risk in rural South African children. BMC Public Health. 2010; 10: 158

8. Benefice E, Garnier D, Simondon KB, Malina RM. Relationship between stunting in infancy and growth and fat distribution during adolescence in Senegalese girls. Eur J Clin Nutr. 2001; 55: 50-58.

9. Naudé D, Kruger HS & Pienaar AE. Differences in body composition, body proportions and timing of puberty between stunted and non-stunted adolescents. Afr J Phys Health Educ, Recr Dance, 2009; 15(4):678-689.

10. Tladi B, Baloyi T, Van Boom E. The social environment. In Mangola S, Kalule-Sabiti M (eds) State of environment report 2002 of the North West Province, South Africa. Directorate Environment & Conservation Management, North West Department of Agriculture, Conservation and Environment. 2002. Available at http://www.nwpg.gov.za accessed on 6 Oct 2010.

11. M'marete CK. Climate and water resources in the Limpopo Province. In: Nesamvuni AE, Oni SA, Odhiambo JJO and Nthakheni ND (eds.) Agriculture as the Cornerstone of the Economy in the Limpopo Province. A study commissioned by the Economic Cluster of the Limpopo Provincial Government under the leadership of the Department of Agriculture. 2003:1-49. Available at http://www.ida.gov.za. Accessed on 6 Oct 2010.

12. Kruger R, Kruger HS, Macintyre UE. The determinants of overweight and obesity among 10–15-year-old schoolchildren in the North West Province, South Africa—the THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) study. Public Health Nutr. 2006; 9: 351–358.

13. Monyeki KD, Kemper HCG, Makgae PJ. Relationship between fat patterns, physical fitness and blood pressure of rural South African children: Ellisras Longitudinal Growth and Health Study. J Hum Hypertens. 2008; 22: 311-319.

14. StatSoft, Inc. 2009. STATISTICA (data analysis software system), version 9.0. www.statsoft.com.

15. WHO Working Group: Use and interpretation of anthropometric indicators on nutritional status. Bull World Health Organ. 1986; 64**:** 929-941

16. Reddy SP, Panday S, Swart D et al (2003). Umthente uhlaba usamila: the 1st South African National Youth Risk Behaviour Survey. Cape Town: South African Medical Research Council, 2002. Available at: http://www.mrc.ac.za/healthpromotion/healthpromotion.htm. Accessed on 5 Nov 2010.

17. Rogol AD, Clark PA, Roemmich JN**.** Growth and pubertal development in children and adolescents: effects of diet and physical activity, Am J Clin Nutr. 2000; 72(2): s521-s528

18. Nevill AM, Stewart AD, Olds T, Holder R**.** Relationship between adiposity and body size reveals limitations of BMI**.** Am J Phys Anthropol. 2006; 129: 151–156.

19. WHO. Physical status, the use and interpretation of anthropometry. Geneva, Switzerland: World Health Organization; 1995.

20. Trowbridge FL, Marks JS, Lopez de Romana G, Madrid S, Boutton TW, Klein PD. Body composition of Peruvian children with short stature and high weight-for-height. II. Implications for the interpretation for weight-for-height as an indicator of nutritional status. Am J Clin Nutr 1987;46 :411

21. Lohman TG. Anthropometry and body composition. In Lohman TG, Martorell R, eds. Anthropometric standardization manual, 1988. Champaign, IL: Human Kinetics Books.

22. Panjikkaran ST and Kumari KS. Augmenting BMI and waist-height ratio for establishing more efficient obesity percentiles among school going children. Indian J Community Med. 2009; 34(2): 135-139

23. Cameron N, Jones LL, Griffith PL, Norris SA, Pettifor JM. How well does waist circumference and body mass index reflect body composition in pre-pubertal children? Eur J Clin Nutr. 2009; 63: 1065-1070.

24. Ashwell M, Hsieh SD. Six reasons why the waist-to-height ratio is a rapid and effective global indicator for health risks of obesity and how its use could simplify the international public health message on obesity. Int J Food Sci Nutr. 2005; 56: 303–307

25. De Moraes ACF, Fadoni RP, Ricardi LM, et al. Prevalence of abdominal obesity in adolescents: a systematic review. Obes Rev 2010; no. doi: 10.1111/j.1467-789X.2010.00753.x

26. Daniels SR, Khoury PR, Morrison JA. Utility of different measures of body fat distribution in children and adolescents. AJE. 2000; 152(12): 1179-1184

Table 1: Summary of methods from the two studies showing similarities and differences

	THUSABANA Study, North West Province (n=351)	Ellisras Study, Limpopo Province
Time of data collection	May 2000 – June 2001	May 2000
Sampling procedure	44 Schools were randomly selected and children were randomly selected systematically from class lists to be representative of the population of North West Province. Only children from rural schools were included in this study.	22 schools (10 pre-school & 12 primary) were randomly selected from 68 schools in the Ellisras area. Each school was then assigned a grade with the expectation that most children in a particular age category would be found in that grade.
Age range of children	9-15 years	6-13 years
Height measurement	IP 1465 stadiometer	Martin anthropometer
Weight measurement	Precision electronic scale	Electronic scale
Waist circumference measurement	Flexible Lufkin steel tape, midway between the lowest portion of the rib cage & iliac crest.	Rosscraft athropometric tape for girths (steel), laterally, midway between the lowest portion of the rib cage & iliac crest, and anteriorly midway between the xiphoid process of the sternum and the umbilicus.
Skinfold measurement	Skinfolds were measued with John Bull skinfold caliper	Skinfolds were measured with a Slim Guide skinfold caliper

Table 2: Formulae for calculating the various indices

Index	Formulae	Index used for
Body mass index (BMI)	weight (kg) / height $(m)^2$	Total body adiposity
Sum of skinfolds (SSF)	triceps+subscapular skinfolds	Total body adiposity
Waist-to-height ratio (WHtR)	waist circumference / height	Abdominal adiposity
Subscapular:triceps ratio (STR)	subscapular skinfold/triceps skinfold	Truncal adiposity

Table 3: Characteristics of children from the two studies(mean±sd)

	Boys			Girls		
	ELS(n=909)	THB(n=170)	p-value*	ELS(n=851)	THB(n=181)	p-value*
Age (years)	10.5(1.9)	12.0(1.5)	< 0.001	10.5(1.8)	12.2(1.6)	< 0.001
Weight (kg)	27.6(5.6)	33.0(9.0)	< 0.001	28.3(6.7)	37.0(10.7)	< 0.001
Height(cm)	137.8(10.3)	141.4(10.6)	< 0.001	139.1(10.7)	144.6(10.8)	< 0.001
Waist (cm)	55.5(4.1)	58.6(6.2)	< 0.001	55.4(4.5)	60.2(7.8)	< 0.001
BMI (kg/m^2)	14.4(1.3)	16.2(2.5)	< 0.001	14.4(1.7)	17.4(3.4)	< 0.001
WHtR	0.40(0.03)	0.41(0.03)	< 0.001	0.40(0.03)	0.42(0.04)	< 0.001
SSF (mm)	13.1(3.3)	15.5(8.4)	< 0.001	17.2(5.5)	23.4(12.9)	< 0.001
STR	0.82(0.15)	0.80(0.17)	0.61	0.83(0.15)	0.86(0.27)	0.54

ELS-Ellisras; THB-Thusabana

BMI-body mass index: SSF-sum of biceps, triceps,subscapular and suprailiac skinfolds: WHtR-waist-to height ratio: STR-subscapular:triceps ratio

p-value*-difference between boys and girls, respectively of the two studies

Table 4: Adjusted means(sd) of different body composition measures for stunted and non-stunted children by study and gender.

		Boys			Girls		
Study	Variable	Non-stunted	Stunted	p-value	Non-stunted	Stunted	p-value
Ellisras		(n=863)	(n=46)		(n=807)	(n=44)	
	BMI	14.4(0.04)	13.9(0.2)	0.005	14.5(0.06)	13.7(0.24)	0.002
	SSF	13.1(0.1)	12.2(0.5)	0.06	17.3(0.2)	15.5(0.8)	0.23
	Waist	55.6(0.1)	53.6(0.5)	0.0002	55.5(0.1)	53.2(0.6)	0.0003
	WHtR	0.40(0.0008)	0.43(0.004)	<0.00001	0.40(0.001)	0.42(0.004)	<0.00001
	STR	0.82(0.005)	0.82(0.02)	0.19	0.83(0.005)	0.79(0.02)	0.09
THUSABANA		(n=112)	(n=58)		(n=134)	(n=47)	
	BMI	16.5(0.2)	15.7(0.3)	0.03	17.7(0.3)	16.6(0.5)	0.054
	SSF	16.2(0.8)	14.2(1.1)	0.14	24.8(1.1)	19.4(1.8)	0.01
	Waist	59.8(0.5)	56.2(0.7)	0.00004	61.5(0.6)	56.6(1.0)	0.00007
	WHtR	0.41(0.003)	0.42(0.004)	0.05	0.42(0.004)	0.42(0.006)	0.10
	STR	0.79(0.02)	0.80(0.02)	0.82	0.85(0.02)	0.91(0.04)	0.19

4

COMPARISON OF WAIST CIRCUMFERENCE PERCENTILES OF BLACK SOUTH AFRICAN CHILDREN AGED 10 TO 14 YEARS FROM DIFFERENT STUDY SITES

It seems prudent to develop standards which are truly representative of children from all ethnic backgrounds (*McCarthy et al., 2001*).

Abstract

Objective: The purpose of this study was to describe the age and sex-specific waist circumference (WC) percentiles in black South African (SA) children from different study sites and compare these with those of African-American children.

Methods: Data on WC were obtained from previous studies on black South African (SA) children aged 10-14 years. Smoothed WC percentile curves for boys (3836) and girls (3992) were computed using the LMS method. The median curves displaying the values of the 50th percentile of the different data sets were compared with each other and a further comparison was done between the median WC of children from each data set and the African-American (A-A) children.

Results: The smoothed percentile curves are presented for boys and girls in each data set. Boys had higher WC values than girls up to the 25th percentile and from the 50th percentile girls had higher values than boys right up to the 95th percentile. Girls aged 12 and 14 years from Cape Town at the 95th percentile exceeded the WC value of 80cm identified as the cut-off point for increased risk of obesity related co-morbidities in adult Asian women. The 50th percentile WC curves in the different data sets reflected differences in WC distribution of the children. Comparison with A-A children indicated similar WC from children in Cape Town (d=0.00-0.36) and major differences from children in Ellisras (d=1.80-2.82)

Conclusions: The results indicate clearly that the median WC of children from SA studies is smaller than those of A-A children, with a medium to large effect size for the difference. Results also suggest concern with respect to high WC values (> 80 cm) among some children. The differences observed between the different data sets and between SA and A-A children suggest that nationally representative data should be used to develop age, sex and ethnic specific WC for this population.

Keywords: Children, waist circumference, percentile curves, South Africa.

Introduction

Assessment of waist circumference (WC) has been included in a set of criteria for diagnosing metabolic syndrome by different groups of experts such as the International Diabetes Federation (IDF) and the National Cholesterol Education Program-Adult Treatment Program (NCEP-ATP III) (Druet *et al*., 2010). The metabolic syndrome is characterized by a group of risk factors for cardiovascular disease and type 2 diabetes including abdominal obesity, dyslipidemia, glucose intolerance and hypertension (Eckel *et al*., 2005). Abdominal obesity and insulin resistance appear to be the main underlying risk factors. The problem of obesity and related metabolic disease risk is not only experienced among adults (Kimani-Murage *et al*., 2010), several large population studies have established the prevalence of metabolic syndrome during childhood (Chen *et al*., 1999; Cook *et al*., 2000).

WC is easy to measure and more reproducible than skinfold measurements (Lemieux *et al.*, 2000). Furthermore, it has a low intra-observer and inter-observer error, and when adjusted for clothing, accuracy remains good (McCarthy *et al*., 2001). It has been suggested that the relationship between abdominal adiposity and cardiovascular diseases (CVDs) may differ among populations and ethnic groups (Molarius *et al*., 1998). Therefore, the development of WC percentiles and cut-offs for various ethnic groups are necessary because of differences in body composition (Liu *et al*., 2010). Countries that have developed reference WC percentiles for children and adolescents include Spain (Moreno *et al.*, 2001), Britain (McCarthy *et al*., 2001), Canada (Katzmaryzk *et al*., 2004), USA (Fernandez *et al*., 2004), Australia (Eisenmann *et al*., 2004), Mexico (Gomez-Diaz *et al*., 2005) and others. Only a few of these studies have specifically investigated abdominal adiposity cut-off points associated with different diseases in non-white populations. South Africa, like other African countries, has not been spared from the rampant increase in childhood obesity and related metabolic complications. The prevalence of metabolic syndrome amongst South African children has been estimated at 6.5% using the NCEP ATP III definition (Matsha *et al*., 2009). To this end there are no published data on WC of children from any country on the African continent except Nigeria. It is in this light that this study describes the percentile distribution of the WC in black South African children from different studies, compares them to each other and to African-American(A-A) children.

Methods

This paper compares WC data from cross-sectional surveys from different provinces of South Africa. Anthropometric and socio-demographic data were collected on 1040 and 120 primary school children from KZN and Kimberley respectively. The KZN study included children from four large schools in the Valley of a Thousand Hills, (halfway between Pietermaritzburg and Durban), a peri-urban area in KwaZulu-Natal. The population density is fairly high with villages scattered over an extensive hilly area. Most houses were constructed with brick and cement with water supply mainly through communal taps. More than half (54 %) of the households use electricity for cooking followed by paraffin (40%) and gas (3.4%) and the rest use coal. Sanitary infrastructure is mainly pit latrines. Most (93%) of the houses are electrified. Cold storage in the form of refrigerator/freezer combination possession is common. Kimberley is the capital of the Northern Cape Province. Children included in this study were mostly living in brick/concrete houses. Households have their own taps within the compound and use electricity for cooking. Toilets are mostly flush. Cold storage in the form of refrigerator/freezer combination possession is common and most households have a radio and television. Class lists were obtained from the schools and 20% of children were selected from each class using a list of random numbers. Informed consent was obtained from parents or guardians and the Ethics and Research Committee of the North-West University approved this study (Ethics number: NWU-0057-08-A1).

Anthropometric measurements

The following anthropometric measurements were taken by trained personnel using standard techniques. Height was measured on a floor standing stadiometer fitted with a headboard to the nearest 0.1 cm. The subject stood barefoot on the base of the stadiometer and wearing light clothing, with heels together, head positioned such that the line of vision is perpendicular to the body (Frankfort plane) and arms hanging freely by the sides. The movable headboard was brought onto the topmost point of the head with sufficient pressure to compress hair and the reading was taken. Body weight was measured without shoes and with light clothing to the nearest 0.1kg on a digital bathroom scale (SECA). Body mass index (BMI) was calculated by weight divided by height squared $(kg/m)^2$ (CDC, 2009). WC was measured to the nearest 0.1 cm at the narrowest point between the lower coastal border and the top of the iliac crest, with a

flexible Lufkin anthropometric steel tape. Waist-to-height ratio was expressed as waist (cm)/height (cm).

Secondary data from six other studies were obtained for inclusion in the current analysis. These include (i) the *THUSABANA* (Transition and Health during Urbanization of South Africans; *BANA*, children- designated North West Province). This study was set up to determine overweight status and to identify determinants of overweight and obesity among children aged 10 to 15 years (n=905) in the North West Province. A random sample stratified for age, gender, type of school and ethnic group was drawn. For purposes of the current analysis only black children were included (Kruger *et al.,* 2006). (ii) Effects of dietary intake, socio-economic status, physical activity levels and the school environment on the weight status of children aged 13-15 years in Bloemfontein, South Africa (designated Free State Province). This study assessed the effect that diet, socio-economic status, physical activity levels and the school environment have on the weight status of children aged 13-15 years(n=408) in Bloemfontein. Bloemfontein is the capital city of the Free State Province of South Africa. It is also the judicial capital, making it one of the three national capitals of South Africa (together with the administrative capital Pretoria and the legislative capital Cape Town). A representative sample of school going children was drawn proportionally from all secondary schools in the area (Motseki *et al*., 2009). (iii) Relationship between fat patterns, physical fitness and blood pressure of rural South African children: Ellisras Longitudinal Growth and Health Study (designated Ellisras). Cluster sampling method was used to select children (n=1730) in Ellisras to investigate the relationship between blood pressure, fat pattern parameters and physical fitness. Ellisras is a rural area within the Limpopo Province and unemployment, poverty and low life expectancy seem to play a significant role in the lives of the people of this village (Monyeki *et al*., 2002). (iv) Body composition in urban black South African children aged 6 to 13 years (designated Polokwane). A sample of children (n=1672) attending private schools in Polokwane participated in this survey. By virtue of attending private schools these children were from middle to upper socio-economic groups of the population. All children present at school during the data collection period participated. The objective of this study was to examine gender differences in percentage body fat of these children (Monyeki *et al*., 2008). (v) Metabolic syndrome in 10 to 16-year-old learners (n=534) from the Western Cape, South Africa: Comparison of the NCEP ATP III and

IDF criteria (designated Cape Town). This study was aimed at identifying South African children from different racial groups with metabolic syndrome using either the NCEP ATP III or IDF definitions and determines the proportion of normal weight children that are metabolically obese. The study sample included learners aged 10-16 years selected through probability stratified multistage random sampling and were from lower to middle income areas of Cape Town. Only black children were included in the current analysis (Matsha *et al*., 2009). (vi) and the prevalence of stunting, overweight and obesity, and metabolic disease risk in rural South African children (designated Mpumalanga). This study investigated the prevalence and patterns of stunting, overweight and hence the risk for metabolic disease in a group of children from rural South Africa (n=2252). This investigation was part of the Agincourt sub-district (Mpumalanga Province) health and socio-demographic surveillance system. The study sample consisted of children and adolescents aged 1-20 years randomly selected from the Agincourt database (Kimani-Murage *et al*., 2010).

Data from the mentioned studies were collected across different age ranges and ideally it could be combined to describe WC for each age group across the eight data sets. However, this was not possible because of the differences in the age ranges in the data sets and the uneven distribution of children in the age groups. Table 1 summarises details of each study and Table 2 shows the distribution of children by study site, gender and age. The sample sizes of the different studies varied with Kimberley having the lowest number of children (n=120) and Mpumalanga having the highest (n=2252). The age distribution also differed both within and between the different data sets, with some study sites having only older children such as NorthWest Province (10 to 15 year old) and Free State Province (13 to 16 years). Furthermore, some age groups were underrepresented in some studies, for instance there was only one 8 year old boy from Cape Town (Table 2). As a practical approach to these limitations, age groups that were common to the majority of the data sets and with sufficient sample sizes (>20) were identified, namely children aged 10 to 14 years in all data sets to be used in the current analysis. Kimberley and Free State data were then excluded from the analysis because Kimberley data had less than 20 children in each age group and Free State had no children aged 10 to 12 years. Ellisras children aged 14 years were, however, included despite the small sample size because of

good representation for other age groups. Details of how measurements were taken are described in detail in the different studies.

Table 1: Summary of the different data sets collected for this study.

Study site	Year of study	Stratum	Boys	Girls	Total	WC measurement position
Ellisras	2000	Rural	892	838	1730	Midway between the lowest portion of the rib cage and iliac crest
Polokwane	2000	Urban	851	821	1672	Midway between the lowest portion of the rib cage and iliac crest cage & iliac crest
North West	2000/2001	Urban & rural	431	474	905	Halfway between iliac crest & lower boarder of the floating rib
Free State	2006	Urban	170	238	408	Narrowest circumference on the waist above the iliac crest 7 below the lower rib
Cape Town	2006/2007	Urban	196	338	534	Narrowest part of the torso & between the ribs and iliac crest
Mpumalanga	2007	Rural	1097	1155	2252	Natural waist
KZN	2008	Rural	545	480	1025	Narrowest point between the lower coastal border & the top of the iliac crest
Kimberley	2009	Informal settlements	66	54	120	Narrowest point between the lower coastal border & the top of the iliac crest

Table 2: Proportional age distribution of children by gender and study site

Study site	Age										
Boys	6	7	8	9	10	11	12	13	14	15	16
Ellisras	26	68	92	111	150	184	173	81	7		
Polokwane	6	58	96	125	92	133	127	115	62	30	7
North West					55	89	98	63	58	68	
Free State								21	77	63	9
Cape Town			1	12	20	12	27	22	31	39	32
Mpumalanga	104	91	111	90	98	105	99	113	103	109	74
KZN	42	75	50	63	66	67	73	57	26	21	5
Kimberley	4	11	13	15	11	8	4				
All SA	182	303	363	416	492	598	601	472	364	330	127
Girls											
Ellisras	26	54	85	98	160	193	158	56	8		
Polokwane	4	71	108	103	102	129	127	97	56	21	3
North West					87	80	88	73	69	77	
Free State								29	108	91	10
Cape Town			10	17	15	47	72	62	50	44	21
Mpumalanga	89	110	96	100	101	97	120	125	111	112	94
KZN	38	59	49	65	56	67	60	57	25	3	1
Kimberley	2	7	10		13	12	1		1		
All SA	159	301	358	396	533	621	626	499	428	348	129

Statistical analysis

Statistical analysis was performed with SPSS for Windows software version 17 (SPSS Inc. Chicago, IL). The mean and standard deviations for weight, height, BMI, WC and waist-to-height ratio (WHtR) were calculated by age group and gender for each data set to show the anthropometric profile of children from the different studies. Smoothed age-and-gender specific percentiles were constructed using LMS Chart Maker Pro software package (The Institute of Child Health, London) which fits smooth centile curves to reference data using the LMS method (Cole & Green 1992). The median and inter-quartile ranges for WC of children from the different study locations were computed. To compare WC of South African children to African-Americans (A-A) the effect size for the difference in medians of each data set and A-A data was calculated to determine the magnitude of the observed differences. Effect size is a way of quantifying the size of difference between two groups and is calculated as [(mean of experimental group-mean of control group)] /standard deviation. The effect size of 0.2 is considered small, 0.5 is medium and 0.8 upwards is large (Cohen, 1969). For the A-A data the median was used because the mean was not available in the published article on A-A children, while for the current study sample the median was used because it was close to the mean for most samples. No comparisons have been made with the Nigerian data because the data were presented in age groups. Additionally, data were then pooled to construct percentile curves for the whole South African data set. The pooling of data from different studies was done in order to provide a statistical description with all the advantages and disadvantages that apply. We recognize that pooling data made up of the different studies is less than ideal. The major uncertainty with this approach may be the extent to which the datasets are representative of black South African children at national level. However, within these known limitations the distribution of the pooled data was compared with the African-American data.

Results

Table 3 shows the means (sd) of the height, weight, WC, BMI and WHtR of children from different study populations by age and gender. Whilst significant differences were observed between children from different locations for the different anthropometric measures others were not significantly different. For instance boys from Cape Town had significantly higher weight and BMI than children from all other locations ($p=0.001$) and there was no significant difference

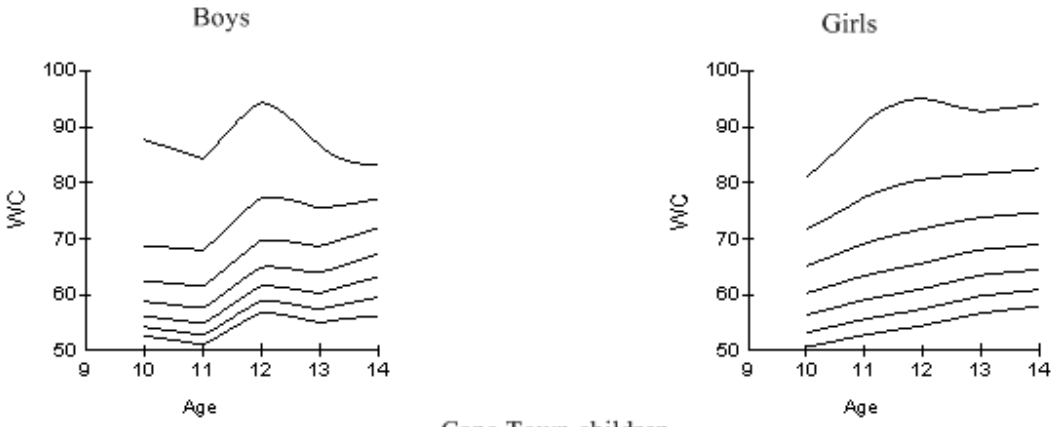
observed in the weight of boys from KZN and Mpumalanga at age 10 and 11 years (p=0.34 and p= 0.78 respectively. At ages 10, 11, 13 and 14 girls from Mpumalanga were significantly taller than all other children (e.g for North-West: p= 0.001, p=0.000, p=0000 & p=0.001 respectively) whilst Ellisras girls had significantly smaller weight than all other children at ages 11, 12, 13 & 14 (p=0.000 for all). WC increased with age for both boys and girls from the different study sites, with the mean values being different between the sites. Percentile distributions of WC according to age, gender and study location were calculated. At the 5^{th}, 10^{th} and 25^{th} percentile boys had bigger WC than girls and from the 50^{th} to the 95^{th} percentiles the WC of girls was bigger than that of boys. It was also observed that WC of girls at 12 and 14 years from Cape Town was big even exceeding value of 80 cm identified as the cut-off point for increased-related co-morbidities in adult Asian women (Alberti *et al*., 2006). At ages 10 and 11 years there are instances where North West children had the lowest WC values but in most instances across all the ages Polokwane and Ellisras children (both boys and girls) had the lowest WC values. The smoothed centile curves with age are presented in Figure 1 to show the pattern of WC growth with age for the different study sites. As illustrated by the curves the distribution of WC percentiles varied across the different sites. Generally boys and girls from Mpumalanga had higher WC than other children for lower percentiles (5^{th} to 25^{th}). Cape Town children had higher WC from the 50^{th} percentile upwards than children from all the other studies. The rate of increase in WC varied across the different sites as children became older. For instance there was a greater increase in WC from 11 to 12 years of age in Polokwane and Cape Town boys, whilst at ages 12 to 13 years KZN boys had the greatest increase. At the 95^{th} percentile boys and girls from Cape Town had bigger WC than the other children and Ellisras children had the smallest WC.

Results of the comparison of median WC values of children from different study sites to A-A children indicate that Cape Town children were consistently comparable to A-A children as shown on Figure 2, 3 & 4 while children from Ellisras were always smaller than A-A children as shown be the large effect size (>0.8) for the difference between the median WCs. From age 12 to 14 years median WC of Mpumalanga children were also comparable to those of A-A children as shown by the small effect size for the difference (d=0.16-0.31). When all the data sets were combined the median WC of South African girls at 13 years demonstrated a small effect size

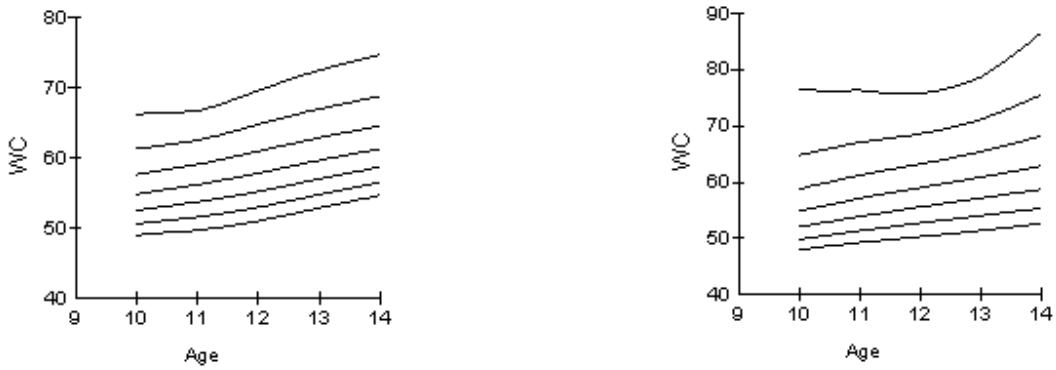
(d=0.13) for the difference from the median WC of A-A girls. A difference of medium effect size was found between median WC of South African and A-A boys at age 14 years (d=0.47). At all the other ages South African children had smaller WC than A-A children with medium to large effect sizes (d=0.64-1.17).

Table 3: Mean(sd) for anthropometric variables for 10-14 year old children

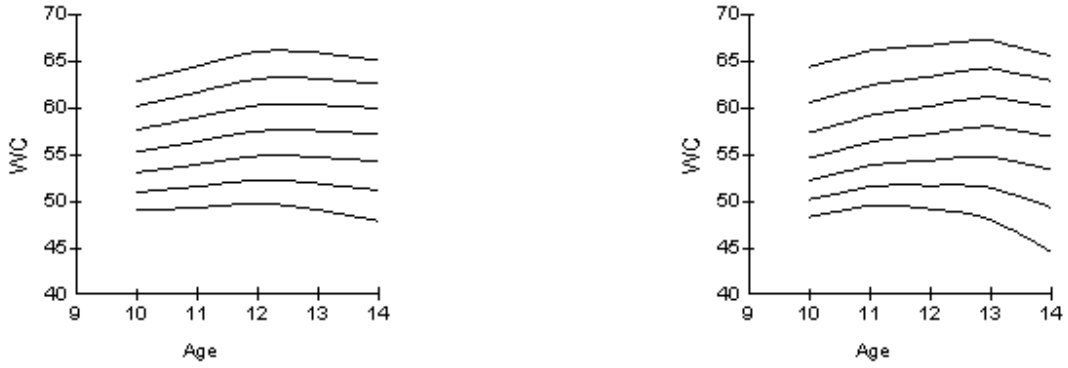
		Boys						Girls					
Age	Province	n	Height	Weight	Waist	BMI	WHtR	n	Height	Weight	Waist	BMI	WHtR
10	Cape Town	20	136.3(6.0)	33.6(6.8)	61.1(8.1)	18.0(2.7)	0.45(0.05)	15	138.0(5.8)	34.6(5.7)	61.0(6.5)	18.1(2.3)	0.44(0.05)
	North West	55	132.8(5.4)	28.0(5.1)	55.9(4.8)	15.8(2.2)	0.42(0.03)	87	135.0(8.1)	29.7(7.6)	56.1(6.4)	16.2(3.1)	0.42(0.04)
	Ellisras	150	138.4(6.4)	27.6(3.7)	55.5(3.5)	14.4(1.1)	0.40(0.02)	160	138.4(5.3)	27.4(4.5)	55.1(4.2)	14.2(1.6)	0.40(0.03)
	KZN	66	132.6(7.0)	29.9(4.8)	58.2(3.9)	16.9(1.6)	0.44(0.03)	56	133.1(7.1)	32.0(5.9)	59.0(5.1)	18.0(2.2)	0.44(0.04)
	Polokwane	92	136.6(10.5)	30.2(8.4)	56.5(5.8)	15.9(2.7)	0.41(0.03)	102	137.0(10.3)	31.1(8.7)	57.1(7.1)	16.4(3.2)	0.42(0.04)
	Mpumalanga	98	136.8(6.2)	29.8(4.9)	59.2(4.4)	15.9(1.9)	0.43(0.03)	101	139.6(6.8)	33.08(8.2)	60.1(6.5)	16.8(3.0)	0.43(0.04)
11	Cape town	12	140.1(4.1)	33.8(4.4)	58.5(6.4)	17.2(1.8)	0.42(0.04)	47	144.3(7.1)	41.5(9.3)	65.3(9.1)	19.8(3.9)	0.45(0.06)
	North West	89	137.4(7.3)	29.6(4.5)	56.3(3.7)	15.6(1.7)	0.41(0.02)	80	141.1(6.5)	34.7(8.8)	59.4(8.4)	17.3(3.5)	0.42(0.05)
	Ellisras	184	142.4(5.8)	29.7(3.9)	56.5(3.8)	14.6(1.2)	0.40(0.02)	193	143.6(6.1)	30.3(5.5)	56.7(4.2)	14.6(1.8)	0.40(0.03)
	KZN	67	137.6(7.3)	32.8(4.8)	59.4(3.6)	17.0(1.4)	0.43(0.03)	67	139.0(7.0)	37.3(8.8)	62.2(7.3)	19.2(3.4)	0.45(0.05)
	Polokwane	133	138.3(11.6)	31.4(9.6)	56.6(5.9)	16.1(2.9)	0.41(0.04)	129	138.8(11.8)	30.9(9.9)	56.7(6.5)	15.8(3.0)	0.41(0.04)
	Mpumalanga	105	142.2(6.8)	32.9(6.9)	60.4(4.9)	16.2(2.4)	0.42(0.03)	97	146.2(7.4)	36.6(9.2)	61.8(7.4)	17.0(3.3)	0.42(0.04)
12	Cape town	27	152.6(10.7)	46.9(13.6)	67.3(8.7)	19.9(4.5)	0.44(0.05)	72	149.6(7.2)	47.6(14.2)	68.7(11.3)	21.0(4.8)	0.46(0.06)
	North West	98	142.4(7.3)	32.8(6.4)	58.5(5.2)	16.1(2.0)	0.41(0.03)	88	145.4(8.6)	36.2(8.2)	59.8(5.9)	17.0(2.7)	0.41(0.03)
	Ellisras	173	145.4(7.6)	31.7(5.3)	57.6(4.1)	14.9(1.5)	0.40(0.03)	158	148.1(7.0)	33.5(5.8)	57.4(4.4)	15.2(1.8)	0.40(0.03)
	KZN	73	140.1(6.6)	35.0(6.8)	61.3(5.3)	17.7(2.4)	0.44(0.03)	60	143.9(5.9)	41.0(9.7)	63.6(7.2)	19.6(3.6)	0.44(0.04)
	Polokwane	127	142.6(13.2)	35.3(11.3)	58.9(7.1)	17.0(3.4)	0.41(0.04)	127	140.5(15.1)	33.2(11.3)	57.4(6.8)	16.4(2.9)	0.41(0.04)
	Mpumalanga	99	147.4(7.9)	37.4(10.4)	63.1(6.6)	17.0(3.1)	0.43(0.03)	120	135.1(7.4)	47.6(12.3)	68.7(8.3)	20.1(4.0)	0.45(0.04)
13	Cape town	22	151.2(9.2)	45.5(16.7)	66.1(9.8)	19.6(5.1)	0.44(0.05)	62	153.4(7.7)	50.5(10.2)	68.8(7.2)	21.4(3.7)	0.45(0.04)
	North West	63	147.1(7.9)	36.4(7.3)	60.8(4.9)	16.7(2.2)	0.41(0.03)	73	149.9(7.4)	39.5(8.6)	61.2(6.0)	17.4(2.6)	0.41(0.03)
	Ellisras	81	146.8(6.1)	32.3(4.8)	57.5(4.3)	14.9(1.5)	0.39(0.03)	56	150.6(7.5)	35.8(6.4)	57.9(4.9)	15.7(1.8)	0.38(0.04)
	KZN	57	147.8(7.7)	39.8(7.2)	63.8(5.9)	18.1(2.3)	0.43(0.04)	57	151.0(5.8)	48.1(9.9)	65.7(7.8)	21.0(3.9)	0.44(0.05)
	Polokwane	115	145.3(14.2)	36.2(10.9)	58.8(6.4)	16.9(3.5)	0.40(0.04)	97	146.5(13.1)	37.5(11.0)	59.4(6.4)	17.1(3.1)	0.41(0.04)
	Mpumalanga	113	155.9(10.6)	44.1(11.0)	65.7(6.0)	17.8(2.4)	0.42(0.02)	125	155.9(6.5)	47.8(8.6)	67.9(6.6)	19.6(3.0)	0.44(0.04)
14	Cape town	31	159.1(9.9)	50.8(10.2)	67.9(6.6)	19.9(2.7)	0.43(0.04)	50	155.2(6.0)	54.1(12.6)	71.0(10.0)	22.4(4.6)	0.46(0.06)
	North West	58	153.0(9.8)	41.3(9.1)	62.0(6.3)	17.6(3.4)	0.41(0.04)	69	153.8(6.2)	47.4(11.5)	65.2(9.9)	19.9(4.3)	0.42(0.06)
	Ellisras	7	152.7(6.1)	34.8(3.4)	57.2(4.0)	14.9(0.7)	0.38(0.04)	8	149.6(7.4)	34.0(6.2)	56.7(5.0)	15.1(2.0)	0.38(0.04)
	KZN	26	155.0(8.5)	43.4(7.0)	63.6(3.7)	18.0(1.5)	0.41(0.03)	25	151.0(6.4)	45.6(5.9)	63.8(4.1)	20.0(2.2)	0.42(0.03)
	Polokwane	62	146.1(14.5)	37.0(12.8)	59.0(7.7)	16.8(3.3)	0.40(0.04)	56	148.6(13.3)	38.1(14.3)	60.6(9.2)	16.8(4.0)	0.40(0.05)
	Mpumalanga	103	161.3(9.4)	47.7(10.0)	67.0(5.4)	18.2(2.5)	0.41(0.03)	111	159.5(6.0)	51.8(10.8)	69.1(7.9)	20.3(4.1)	0.43(0.05)



Cape Town children



North West children



Ellisras children

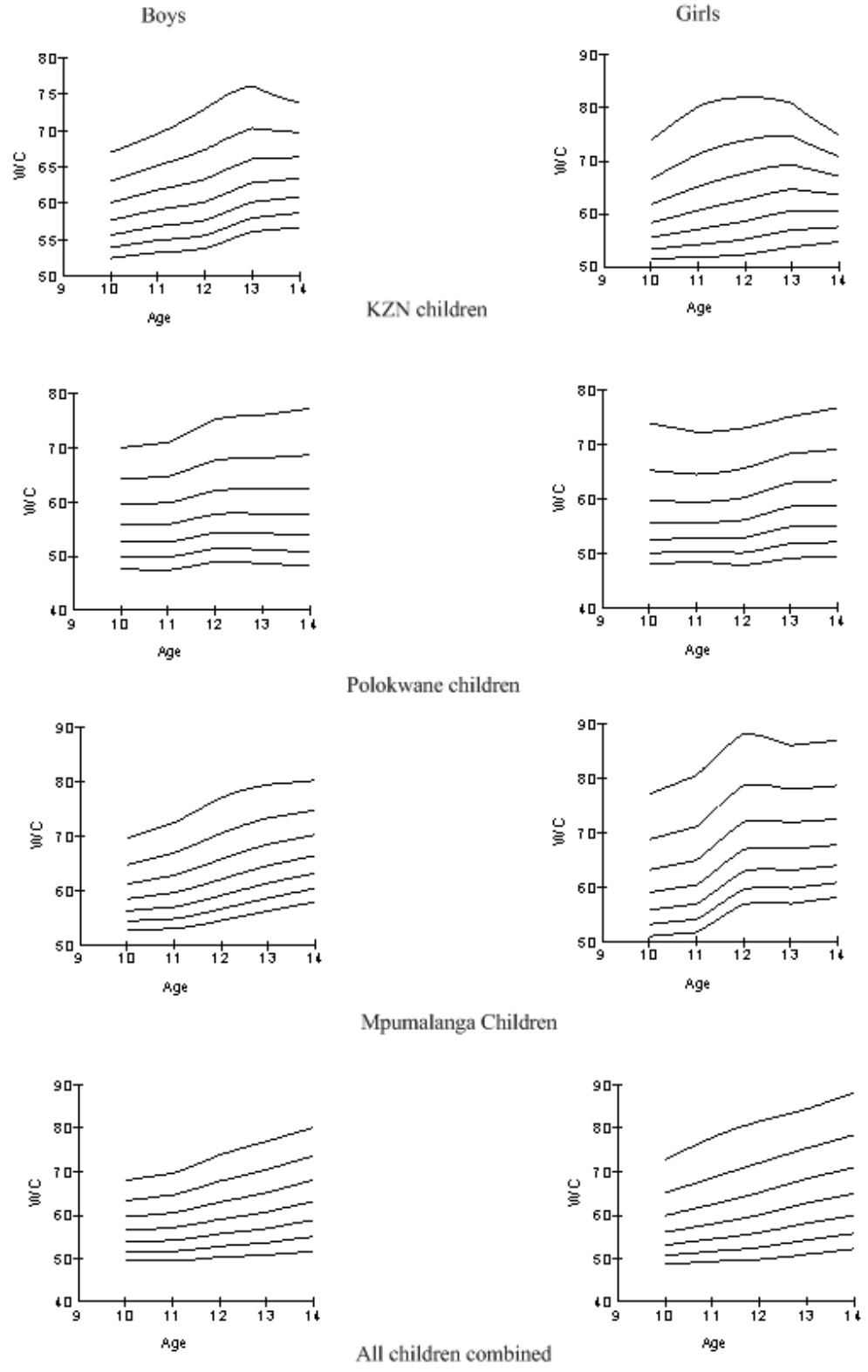


Figure 1: Smoothed LMS curves for the 5th, 10th, 25th. 50th 75th, 90th and 95th percentiles for waist in children from different study sites

Table 4: Median and Inter quartile range for waist circumference of children from different study sites

		Boys					Girls				
Age	Province	Median	Mean	SD	IQR	d	Median	Mean	SD	IQR	d
10	Cape Town	58.7	61.2	8.1	56.6-61.9	0.32	60.8	61.0	6.5	56.2-67.0	0.34
	North West	55.6	55.9	4.8	52.3-57.7	1.19	54.2	56.1	6.4	51.7-59.0	1.38
	Ellisras	55.0	55.5	3.5	53.0-57.9	1.80	55.0	55.1	4.1	52.8-57.0	1.95
	KZN	57.4	58.2	3.9	55.3-59.7	1.00	58.1	59.0	5.1	55.1-62.9	0.96
	Polokwane	55.5	56.5	5.8	52.4-59.1	1.00	55.6	57.1	7.1	53.0-59.7	1.04
	Mpumalanga	58.3	59.2	4.4	56.6-60.4	0.68	59.0	60.1	6.5	56.0-62.5	0.62
	All SA	56.8	57.0	4.9	54.0-59.1	0.92	56.0	57.2	6.2	53.0-60.0	1.13
	A-A	61.3			58.1-66.1		63.0			58.3-70.3	
11	Cape Town	56.5	58.5	6.4	55.6-59.9	1.05	63.0	65.3	9.1	59.0-70.4	0.23
	North West	56.4	56.3	3.7	53.6-58.8	1.84	56.9	59.4	8.4	53.8-63.3	0.98
	Ellisras	56.4	56.5	3.8	53.9-59.4	1.79	56.9	56.7	4.2	54.0.-59.0	1.95
	KZN	59.1	59.4	3.6	57.3-60.8	1.14	60.5	62.2	7.3	56.9-67.5	0.63
	Polokwane	55.6	56.6	5.9	53.0-58.6	1.29	55.8	56.7	6.5	53.0-58.8	1.43
	Mpumalanga	60.0	60.4	4.9	57.6-62.4	0.65	60.5	61.8	7.4	56.0-64.8	0.62
	All SA	57.2	57.6	4.8	54.5-60.2	1.25	57.6	59.0	7.2	54.8-61.6	1.04
	A-A	63.2			59.8-68.3		65.1			60.0-73.1	
12	Cape Town	65.0	67.3	8.7	61.5-72.5	0.00	65.4	68.7	11.3	60.3-76.8	0.17
	North West	58.1	58.5	5.2	54.6-60.7	1.33	59.2	59.8	5.9	55.8-63.6	1.37
	Ellisras	57.6	57.6	4.1	55.0-59.8	1.80	57.0	57.4	4.4	54.2-60.0	2.34
	KZN	59.9	61.3	5.3	57.9-64.5	0.96	61.4	63.6	7.2	59.1-67.1	0.82
	Polokwane	57.7	58.9	7.1	54.0-61.9	1.03	56.3	57.4	6.8	52.5-61.0	1.62
	Mpumalanga	61.4	63.1	6.6	59.5-65.0	0.55	66.0	68.7	8.3	63.0-74.4	0.16
	All SA	58.8	59.9	6.3	56.0-62.2	0.98	60.0	61.8	8.7	56.0-65.5	0.84
	A-A	65.0			61.5-70.5		67.3			61.7-75.9	
13	Cape Town	63.4	66.1	9.8	58.5-68.5	0.36	67.6	68.8	11.3	64.2-72.7	0.16
	North West	60.2	60.8	4.9	57.5-62.4	1.37	60.4	61.2	6.0	57.0-65.2	1.50
	Ellisras	57.2	57.5	4.3	55.0-60.4	2.26	58.5	57.9	4.9	54.1-62.0	2.22
	KZN	63.1	63.8	5.9	60.0-65.8	0.64	64.6	65.7	7.8	60.2-69.4	0.62
	Polokwane	58.6	58.8	6.4	54.0-63.0	1.30	59.0	59.4	6.4	55.4-62.0	1.63
	Mpumalanga	64.5	65.7	6.0	61.3-69.3	0.40	68.0	67.9	6.6	63.0-72.0	0.21
	All SA	67.0	61.8	7.1	63.2-70.3	-0.01	68.2	64.0	7.8	64.0-73.0	0.15
	A-A	66.9			63.2-72.7		69.4			63.4-78.8	
14	Cape Town	68.0	67.9	6.6	63.4-71.9	0.11	69.8	71.0	10.0	63.8-74.7	0.17
	North West	60.5	62.0	6.3	58.9-63.2	1.30	62.4	65.2	9.9	58.4-70.0	0.92
	Ellisras	58.0	57.2	4.0	53.0-60.4	2.68	57.4	56.7	5.0	52.4-59.5	2.82
	KZN	64.2	63.6	3.7	60.5-66.4	1.22	62.4	63.8	4.1	60.5-67.2	2.22
	Polokwane	57.0	59.0	7.7	54.2-63.0	1.52	59.2	60.0	9.2	55.4-63.0	1.34
	Mpumalanga	66.6	67.0	5.4	63.6-70.2	0.39	67.0	69.1	7.9	64.5-72.0	0.57
	All SA	64.4	65.1	7.7	60.1-69.0	0.56	65.5	67.1	9.4	61.0-71.0	0.64
	A-A	68.7			64.9-74.9		71.5			65.1-81.6	

A-A: African-American

All SA-South African data sets combined

*d = effect size for the difference in median waist circumferences between each study sample and A-A children

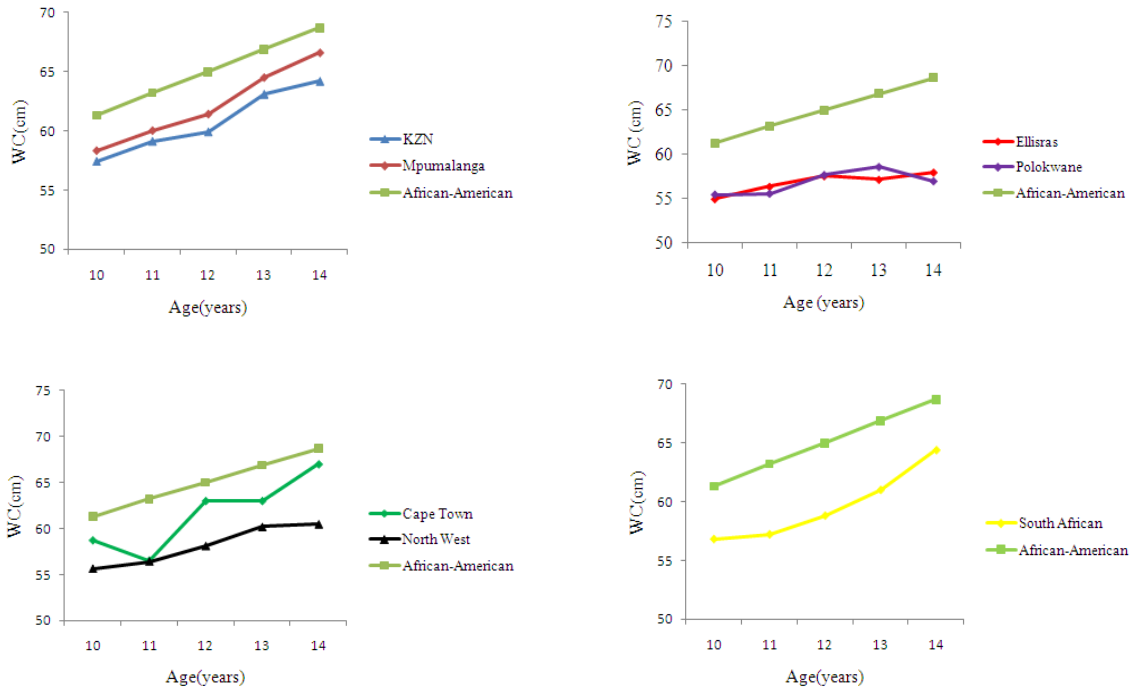


Figure 2: Comparison of the 50th percentile WC curves (unsmoothed) for 10-14 year old South African & African-American boys

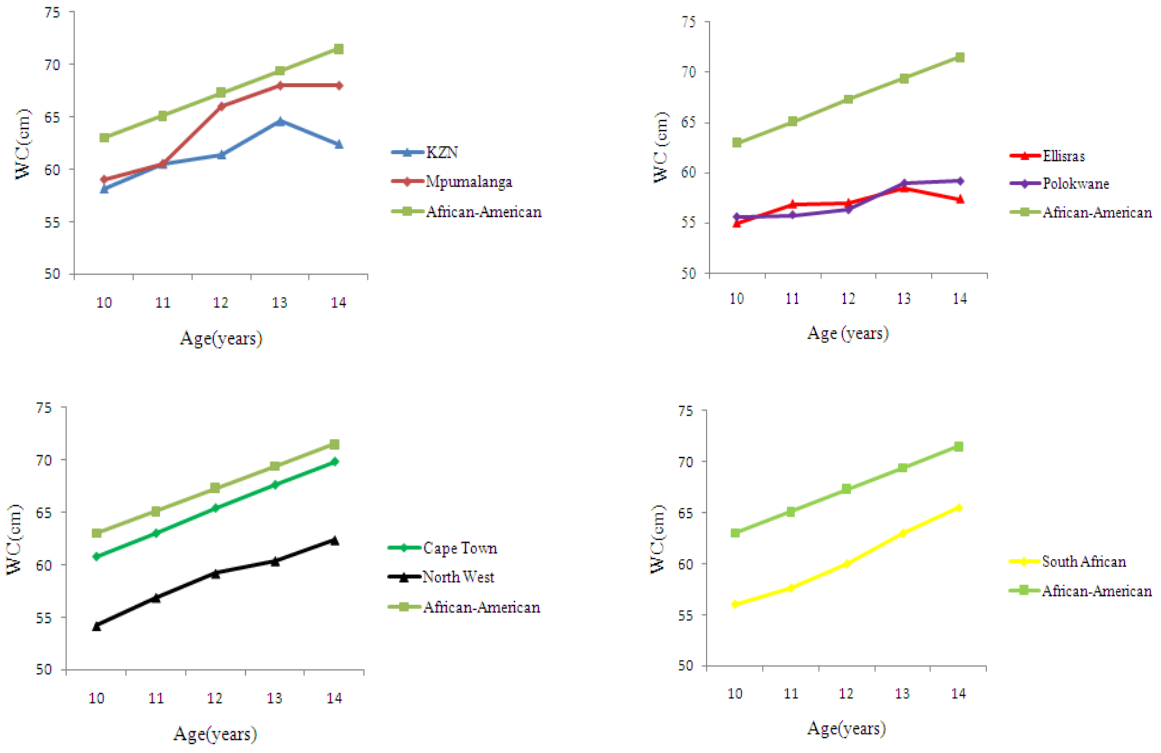


Figure 3: Comparison of the 50th percentile WC curves(unsmoothed) for 10-14 year old South African & African-American girls

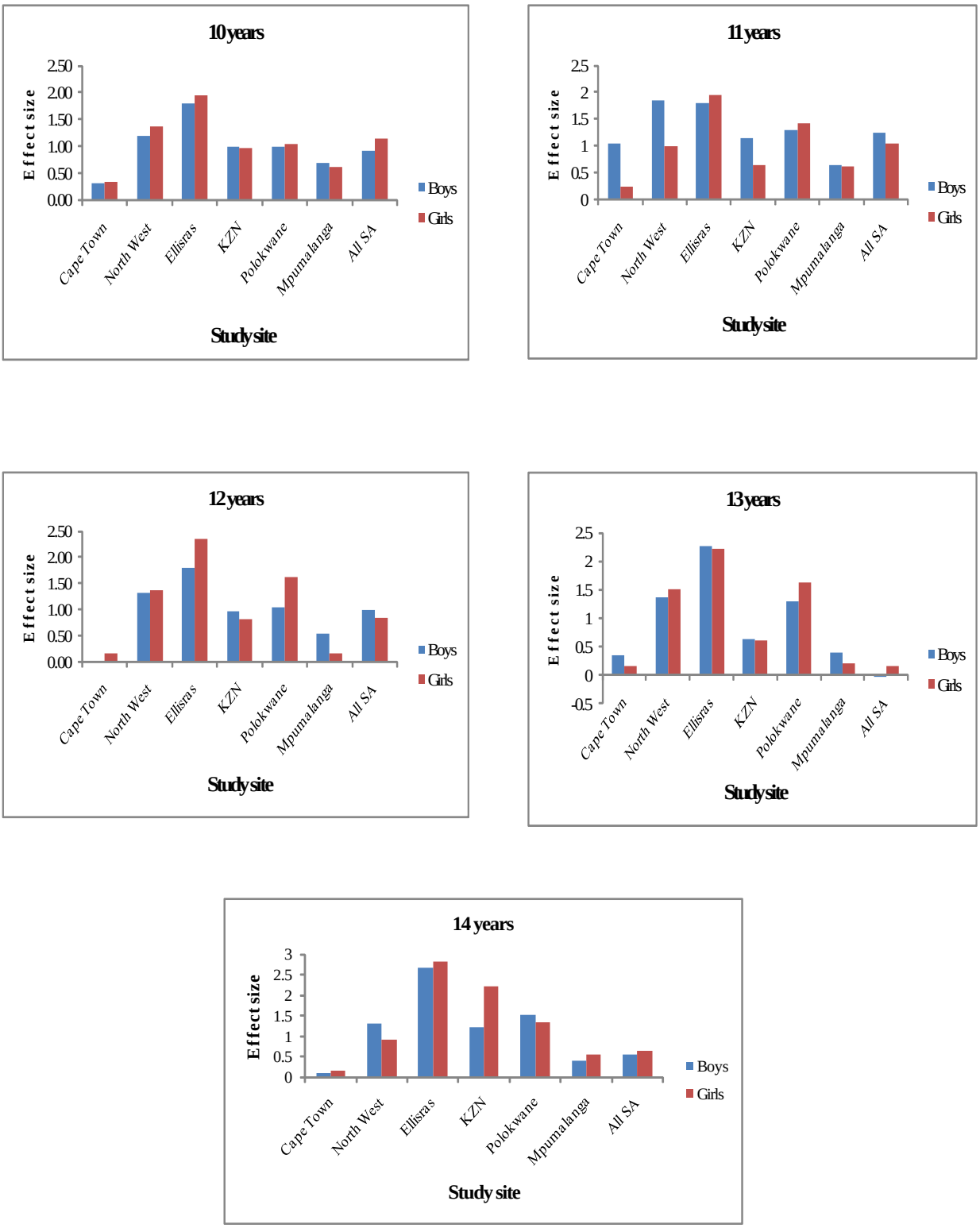


Figure 4: Graphical presentation of effect sizes for the difference between median WC of study samples and African American children

Discussion

This study showed that the WC distribution of black SA children from different studies was different with a trend indicating that Cape Town children had the biggest WC and Polokwane and Ellisras children had the smallest WC. The City of Cape Town is one of South Africa's five metropolitan municipalities and is regarded as the economic heartbeat of the Western Province. It has outperformed the rest of the Province in terms of infrastructure, income and waste disposal (City of Cape Town, 2006). In the urban setting globalization has been observed to increase the risk of obesity by creating an environment that promotes consumption of foods rich in fat and sugar (Khan *et al.,* 1999). This was observed in the National Food Consumption Survey (1999) when the study reported a high prevalence of overweight among urban children when using the body mass index standard proposed by Cole *et al.,* (2000) to classify children. This could be a possible explanation for the current observation. Childhood obesity has negative health implications with approximately 5-10 year old children presenting with at least one associated cardiovascular risk factor and 25% presenting with two or more risk factors (Koplan *et al.,* 1999). The Limpopo Province is one of the poorest regions of South Africa especially in the rural areas with over 61% of the population living below the national poverty line (UNDP, 2004). Generally despite government's commitment to addressing development issues in South Africa, disparities in food security exist between communities and households across the country, reflecting continuing social and economic inequalities which could be contributing to differences in the growth rate of children from different locations. Kgamphe (2009) argues that South Africa is a cosmopolitan country with high urbanization rate. The wide range of socio-economic conditions both within urban and rural environments makes South Africa a non-homogeneous country in terms of economic conditions, health systems and prevalence of disease.

Malina *et al*. (2004) maintain that although human beings are in general genetically similar, populations differ in a variety of genotypic and phenotypic characteristics including growth. The differences in WC of children from these sites may imply that environmental factors such as lifestyle and cultural characteristics may be more important determinants of WC than genetic similarity. Tanner (1976) argues that differences in size and shape between children of different populations are due to differences in their gene pools, in their environments, and in the

interactions between the two, but with the added complications caused by variations in rate of maturation. Thus, two populations may reach an average identical adult size, but the children of one population may be larger than those of the other at a particular age, simply because they have a faster rate of growth, enter puberty earlier, and cease growing earlier.

The comparison of median WC values of children from different study sites to A-A children were made. The reason why comparisons were made with these children is that they are of the same ethnic origin and several studies have described ethnic differences in WC (McCarthy *et al*., 2001; Fernandez *et al*., 2004; Misra *et al*., 2005). The effect size for the difference of median WC between the study samples and A-A children was in most instances smaller for Cape Town children. It is possible that this is so because Cape Town is a city and is the legislative capital of South Africa as well as the capital of Western Cape Province and one of the biggest cities in South Africa. As a result of urbanization the living conditions might be contributing to the growth rate of children there. The 2006 general household survey for Cape Town pointed to these conditions when it revealed that 87.4% of households have a flush or chemical toilet, 94.9% have refuse removed by the municipality at least once a week, 69.3% have running water inside their dwelling, 80.1% use electricity for cooking and 77% of households have a television (Small, 2008). Similar observations have been reported in Indian children, where the prevalence of overweight and obesity was significantly higher among children of the upper socio-economic status (Tharkar *et al*., 2009). Rapid epidemiological transition currently sweeping across South Africa is probably having a direct impact on food habits and lifestyle resulting in increases in obesity among children in urban areas.

Although Polokwane is also an urban area, children from this study site always had smaller median WC. This observation is similar to results from a previous study on urban-rural differences in the growth of South African black children (Cameron *et al*., 1992). The general pattern observed in this study was that the average urban children were consistently and at times significantly smaller and lighter and concluded that the average urban environment in South Africa is not conducive to improved growth and health unless it is accompanied by an improved socioeconomic status. This could possibly be an explanation for the current observation. Ellisras children had in most instances smaller WC than children from all other SA studies, as

well as the A-A children. This might be due to the fact that Ellisras is a rural area and in a province (Limpopo) considered one of the poorest in South Africa (Oni *et al*., 2003). The 2008 Youth Risk Behaviour Survey of high school children indicated that 12.8% of children in Limpopo Province were stunted. North West children had similar WC values to those of Ellisras children. Data analysis by Mukuddem-Petersen *et al*. (2004) on THUSABANA children previously documented a high prevalence of stunting in rural areas (boys: 26.7%, girls: 23.7%) and the highest prevalence of overweight and obesity was found among white children (14.2%), compared to black (7.1%), This could explain why North West children had similar median WC values to Ellisras children and they both have a high prevalence of stunting. It was suprising to observe a small difference between the median WC of Mpumalanga and A-A children. These children would be expected to be thinner because they lived in a rural area. It could be possible that this is a demonstration of an association between low socio-economic status and obesity. A comparison of WC data between different studies, however, needs to be done with caution because of the following reasons: First the measurement sites usually differ as is the case in data sets used in the current study. Currently there is no agreement on which site is optimal (Wang, 2003), with five different sites used by different investigators. These include: (i) midway between the lowest rib and superior iliac crest; (ii) at the umbilical level; (iii) at the narrowest point of the torso; (iv) at the level of the right upper iliac crest and at the level of 2cm above the umbilicus. A recent study demonstrated that the different sites differ significantly among 7-84 year old individuals (Wang, 2003). If waist circumference is to become an important public health assessment tool for central obesity in both adults and children, international agreement about measurement site is required (Sung, 2008). Secondly, although the data used in this study were on black South African children, the surveys are different in timing, selection of children and other environmental factors; these could have a confounding effect on the outcomes observed. The question, therefore, is whether the timing of the studies has any relevance to the differences in WC of children. Utter and colleagues (2009) argue that considerable changes in body size and shape including WC have occurred in recent decades in children and this may be a consideration when comparing data collected in different periods (Liu *et al*., 2010). The different studies were conducted at different time periods and median WC values reflect a trend towards increasing WC with time. For instance 10 year old boys in the North West Province conducted in 2000 have the median WC of 55.6cm versus 58.3 for Mpumalanga (2007) and 58.7

for Cape Town (2007). This could possibly be due to improvements in the general socio-economic status of the different provinces in South Africa. The 2001 population census figures indicate accelerated growth in the urbanization of South Africa (Stats SA,2004) and Prinsloo (2001) argue that normally one expects to see an increase in standards of living and in the Human Development Index with increases in urbanization possibly contributing to increasing WC with time.

Results of this study describe the WC percentile distribution in a sample of black South African children. Therefore, the estimated percentiles describe the population represented and do not establish a standard of what WC of these children should be. The disadvantage of reviewing data from different provinces as in this analysis is the potential imbalance in the impact of socio-economic development on children across the different sites. Therefore, making comparisons across the studies may not reflect the true representation of the patterns in WC. Evaluation of this distribution is nevertheless important given the mounting concern of obesity among children and the health consequences of overweight and obesity. This information can be used as a point of reference for future studies on WC in the pediatric population. Although not ideal, the WC values for the combined data sets give a crude description of the existing WC distribution of the South African black population of 10-14 year old children. It indicates clearly that the median WC of children from SA studies is smaller than those of A-A children, with a medium to large effect size for the difference. In future the possibility of developing ethnic specific cut-off points for this population for the identification of at risk persons should be given priority based on the fact that childhood obesity is a problem of epidemic proportions worldwide. WC is inexpensive, simple to measure and correlates strongly with trunk fat, thus its assessment may add to identifying children at increased risk of cardiovascular diseases.

References

ALBERTI, K.G., ZIMMET, P & SHAW, J. *2006.* Metabolic syndrome -- a new world-wide definition. A Consensus Statement from the International Diabetes Federation. *Diabetic medicine,* 23*:* 469–480

CAMERON, N., KGAMPHE, J.S., LESCHNER, K,F & FARRANT, P.J. 1992. Urban-rural differences in the growth of South African black children. *Annals of human biology*, 19(1):23-33.

CHEN, W., SRINIVASAN, S.R., ELKASABANY, A., BERENSON, G.S. 1999. Cardiovascular risk factors clustering features of insulin resistance syndrome (Syndrome X) in a biracial (Black-White) population of children, adolescents, and young adults: the Bogalusa Heart Study. *American journal of epidemiology*, 150:667–674

COHEN, J. 1969. Statistical Power Analysis for the Behavioral Sciences. NY: Academic Press.

COLE, T.J & GREEN P. J. 1992. Smoothing reference centile curves: the LMS method and penalized likelihood. *Statistics in medicine*, 11(10):1305-1319

COOK, S., WEITZMAN, M., AUINGER, P., ET AL. 2003. Prevalence of a metabolic syndrome phenotype in adolescents: findings from the third National Health and Nutrition Examination Survey, 1988-1994. *Archives of pediatrics and adolescent medicine*, 157:821–827.

DRUET, C., ONG, K., MARCHAL, C.L. 2010. Metabolic syndrome in children: comparison of the International Diabetes Federation 2007 consensus with an adapted National Cholesterol Education Program definition in 300 overweight obese French children. *Hormone research in paediatrics*, 73(3):181-183

ECKEL, R.H., GRUNDY, S.M., ZIMMET, P.Z. 2005. The metabolic syndrome. *Lancet*, 365:1415-1428

EISENMANN, JC. 2004. Waist circumference percentiles for 7-15 year old Australian children. *European journal of clinical nutrition*, 58:1011-1015

FERNANDEZ, J. R., REDDEN, D. T., PIETROBELLI, A., & ALLISON, D. B. 2004. Waist circumference percentiles in nationally representative samples of African-American,

European-American, and Mexican-American children and adolescents. *Journal of pediatrics,* 145:439–444.

GOMEZ-DIAZ, RA., MARTINEZ-HERNANDEZ, AJ., AGUILAR-SALINAS, CA., VIOLANTE, R., LOPEZ-ALARCON, M., JIMENEZ-VILLARRUEL, M., WACHER-RODARTE, N & SOLORZANO-SANTOS F. 2005. Percentile distribution of waist circumference among Mexican per-adolescents of a primary school in Mexoci city. *Diabetes obesity and metabolism*, 7(6): 716-721

Healthy weight: Assessing your weight: BMI. Center for Disease and Control. 2009. Available at: http://www.cdc.gov/healthy weight/assessing/bmi/index.html. Date of access: 6 Nov. 2010

KATZMARYZK, PT. 2004. Waist circumference percentiles for Canadian youth 11-18 years age. *European journal of clinical*, 58:1011-1015

KGAMPHE, S.J. 2009. Physical growth and development of South African rural black children. Thesis submitted to the Faculty of Science, University of Witwatersrand. Available at http://wiredspace.wits.ac.za. Date of access: 19 Nov. 2010

KHAN, M.K., BOWMAN, B. 1999. Obesity: a major public health problem. *Annual review of nutrition,* 19; xiii-xvii

KIMANI-MURAGE, E., KAHN, K., PETTIFOR, .J.M., TOLLMAN, S.M., DUNGER, D.B., GOMEZ-OLIVE, X., NORRIS, S.A. 2010. The prevalence of stunting, overweight and obesity, and metabolic disease risk in rural South African children. *BMC Public Health,* 10:158

KOPLAN, J.P., DIETZ, W.H. 1999. Caloric imbalance and public health policy. *Journal of American medical association,* 282:1579-1581

KRUGER HS, MARGETTS BM, VORSTER HH. 2004. Evidence for relatively greater subcutaneous fat deposition in stunted girls in the North West Province, South Africa, as

compared with non-stunted girls. *Nutrition: the international journal of applied and basic nutritional sciences*, 20(6):564-569.

KRUGER, R., KRUGER, H.S., MACINTYRE, U.E. 2006. The determinants of overweight and obesity among 10–15-year-old schoolchildren in the North West Province, South Africa—the THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) study. *Public health nutrition*, 9: 351–358.

LEMIEUX, I., PASCOT, A., COULLIARD, C. et al. 2000. Hypertriglyceridemic waist: a marker of the atherogenic metabolic triad (hyperinsulinemia; hyperapolipoprotein B; small dense LDL) *Circulation*, 102:179-184.

LIU, A., HILL, A.P., HU, X., LI, Y., DU, L., XU, Y., BYRNE, N.M., MA, G. 2010. Waist circumference cut-off values for the prediction of cardiovascular risk factors clustering in Chinese school-aged children: a cross-sectional study. *BMC public health*, 10:82

MALINA, R.M., BOUCHARD, C., BAR-OR, O. 2004. Growth, maturation and physical activity. Champaign, IL: Human Kinetics

MATSHA, T., HASSAN, S., BHATA, A., YAKO, Y., FANAMPE, E., SOMERS, A., HOFFMANN, M., MOHAMMED, Z., & ERASMUS, R. T. 2009. Metabolic syndrome in 10–16-year-old learners from the Western Cape, South Africa: Comparison of the NCEP ATP III and IDF criteria. *Atherosclerosis*, 205:363-366.

MCCARTHY, H.D., JARRETT, K.V., CRAWLEY, H.F. 2001. The development of waist circumference percentiles in British children aged 5.0 to 16.9 y. *European journal of clinical nutrition*, 55:902-907.

MONYEDI, K.D., STEYN, N.P., MONYEKI, M.A. 2002. Body composition in urban black South African children aged 6-13 years. *African journal of physical health education, recreation and dance*, 8(2): 285-296

MONYEKI, K.D., KEMPER, H.C.G., MAKGAE, P.J. 2008. Relationship between fat patterns, physical fitness and blood pressure of rural South African children: Ellisras Longitudinal Growth and Health Study. *Journal of human hypertension*, 22: 311-319.

MORENO, L.A., FLETA, J, MUR, L., RODRIQUEZ, G., SARRIA, A., & BUENO, M. 1999. Waist circumference values in Spanish children–gender related differences. *European journal of clinical nutrition,* 53**:**429–433.

MOTSEKI, N.M.L. 2009. Associations between determinants of weight status in children, 13-15 years in Bloemfontein. Bloemfontein: University of the Free State, Ph D dissertation.

MUKUDDEM-PETERSEN, J & KRUGER, H.S. 2004. Association between stunting and overweight among 10-15 year old children in North West Province, South Africa: the THUSA BANA study. *International journal of obesity*, 28:842-851

ONI, S., A.E. NESAMVUNI., ODIAMBO, J.J.O. & DAGADA, M.C. 2003. Agriculture as a cornerstone of the economy of Limpopo Province. Agriculture policies and institutions in agricultural investment, financing and credit in the Limpopo Province. Department of Agricultural Economics and Extension, South Africa, Volume 1(12).

PRINSLOO, D.A. 2001. What is the 2001 telling us? Available at: http://www.urbanstudies.co.za/mar-census01.html. Date of access: 15 Nov. 2010

REDDY,S.P., PANDAY, S., SWART, D. et al (2003). Umthente uhlaba usamila: the 1st South African National Youth Risk Behaviour Survey. Cape Town: South African Medical Research Council, 2002. Available at:

http://www.mrc.ac.za/healthpromotion/healthpromotion.htm. Date of access: 5 Nov. 2010.

SMALL, K. 2008. City of Cape Town, Strategic development information and GIS department. Available at: http;//www.capetown.co.za/en/stats/cityreports/docoments/households. Date of access: 10 Nov. 2010

STATS SA 1996/2003. 2004. Available at: http://www.statsa.gov.za/publications/statsdownload. Date of access: 23 Nov. 2010

SUNG, R. Y., YA, C.C., CHOI, K.C., MCMANUS, A., LI, A.M., XA, S.L et al. 2007. Waist circumference and body mass index in Chinese children: cutoff values for predicting cardiovascular risk factors. *International journal of obesity* (London), 31:550-558

TANNER, J.M. 1976. Population differences in body size, shape, and growth rate-a 1976 view. *Archives of disease in childhood*, 51: 1.

THARKAR, S & VISWANATHAN, V. 2009. Impact of socio-economic status on prevalence of obesity among children and adolescents in urban India. *The open obesity journal*, 1: 9-14 9

UNITED NATIONS DEVELOPMENT PROGRAMME. 2004. South Africa Human Development Report 2003. Cape Town: Oxford University Press Southern Africa.

UTTER, J., SCRAQQ, R., DENNY, S., SCHAAF. D. 2009. Trends in body mass index and waist circumference among New Zealand adolescents. *Obesity reviews*, 10:379-382

WANG, J., THORNTON, J.C., BARI, S., WILLIAMSON, B., GALLANGHER, D., HEMSFIELD, S.B., HORLICK, M., KOTLER, D et al. 2003. Comparison of waist circumference measured at four sites. *American journal of clinical nutrition*, 77:379-384

5

SENSITIVITY OF THE WAIST-TO-HEIGHT RATIO IN IDENTIFYING CHILDREN WITH HIGH BLOOD PRESSURE

"Keep your waist circumference to less than half your height" *(Ashwell, 2005).*

Sensitivity of the waist-to-height ratio in identifying children with high blood pressure

Boitumelo S Motswagole[1]MSc, H Salome Kruger[1]PhD, Mieke Faber[2]PhD, Johannes M van Rooyen[3] DSc, J Hans de Ridder[3] PhD

[1]Centre of Excellence for Nutrition, North-West University, Private Bag X6001, Potchefstroom 2520, South Africa. Tel: +27 79 3715 130, Fax: +27 18 299 2464; [2]Nutritional Intervention Research Unit, Medical Research Council, PO Box 19070, Tygerberg 7505, South Africa; [3]Hypertension in Africa Research Team(HART), North-West University, Potchefstroom 2520, South Africa.

Correspondence to: H Salome Kruger, Centre of Excellence for Nutrition, North-West University, Private Bag X6001, Potchefstroom 2520, South Africa. Tel: +27 299 2482, Fax: +27 18 299 2464, E-mail: Salome.Kruger@nwu.ac.za

Accepted for publication as a letter to the editor in the Cardiovascular Journal of Africa .

Comments from reviewers are attached in Addendum 1. The article was prepared according to the instructions for authors for the journal, July 2010.

Abstract

We determined the sensitivity of waist-to-height ratio (WHtR) as a marker for high blood pressure in children aged 9-15y (n=1131) from schools in the NorthWest Province, South Africa. Anthropometric and blood pressure measurements were taken. The sensitivity and specificity of the WHtR to identify children with high blood pressure were evaluated. At a cut-off value of 0.5, 7.9% of the girls and 3.4% of the boys had central adiposity. Thirteen percent of the children were hypertensive. The optimal WHtR cut-off value to identify children with hypertension was 0.41 in both boys and girls. Positive correlations were observed between anthropometric indices. Using linear regression analysis, age and body mass index were significant predictors of high blood pressure in boys whilst for girls it was height and weight. Results suggest that adopting a WHtR cut-off value <0.5 could enhance the use of WHtR as a marker for high blood pressure in children.

Keywords: waist-to-height ratio, blood pressure, children, South Africa.

Introduction

Assessment of risk for the presence of high blood pressure in children is of particular importance, since it allows for the timely identification of those at high risk of developing high blood pressure later in life. In this context measures of obesity indices in children such as body mass index (BMI), waist circumference (WC) and waist-to-height ratio (WHtR) are all considered useful, non-invasive anthropometric measurements to provide information on the risk of high blood pressure. However, studies of both adults[1-2] and children [3-5] have concluded that WHtR is more strongly associated with cardiovascular (CV) risk factors than BMI. Hence the WHtR has been proposed as an alternative measure for assessing central fatness in children on the basis that it is relatively age-independent and that in normalising for growth, it might preclude the need for age-related reference charts. [3-4, 6] Given the growing concern of obesity among children and its consequences, coupled with the fact that high blood pressure has been previously observed in children, the establishment of ethnic specific cut-off values for identification of at-risk persons cannot be overemphasised. It is important to establish that such ethnic specific cut-off values require demonstration of differential predictive validity and not simply demonstration of marginal distribution of the predictor.[5] Previously Ashwell and colleagues[7] proposed an age-independent universal WHtR cut-off value of 0.5 for predicting CV risk. However, this value is yet to be tested by direct correlation with CV risk markers in children. Sung and co-workers[8] argue that for a WHtR of 0.5 to become a universal predictor of CV risk in children, further direct confirmatory evidence from comparative correlations with CV risk factors in children of different age and ethnic origins are required. Early detection of high blood pressure is imperative as children within the upper distribution of blood pressure are at risk of developing hypertension in adulthood.[9] The predictive ability of WHtR in South African children remains unknown; therefore this study is aimed at assessing the diagnostic accuracy of WHtR as a marker for future cardiovascular events like high blood pressure in South African children and whether WHtR could be used as a marker for high blood pressure.

Methods

Participants

The sample of children in the current analysis was drawn from the THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA means children) study which was

conducted between May 2000 to June 2001. This study was aimed at investigating the determinants of overweight, obesity and high blood pressure among 9 to 15 year old school children in the North West Province of South Africa. Study methods were described in detail by Kruger et al.[10] Briefly, forty four schools were randomly selected from a list of schools obtained from the Department of Education in the North West Province. From the selected schools boys and girls were randomly selected systematically from the class lists. The measurements were taken during school hours after informed consent was given by the school headmaster and the children's parents and assent was obtained from the children. The total number of children was 1257. However, with some of the anthropometric variables, missing values limited the sample size to 1131 subjects. This study was approved by the Ethics Committee of the North-West University.

Body Composition

All anthropometric measurements were taken according to standard methods [10] by qualified anthropometrists using calibrated apparatus. Weight was measured to the nearest 0.1 kg on a calibrated electronic scale (Precision Health Scale, Saitama, Japan) and height was measured to the nearest 0.1 cm with a stadiometer calibrated using a flexible steel measuring tape (Lufkin, Cooper Tools, Apex, NC, USA). Waist circumference (WC) was measured at the narrowest point between the lower coastal border and the top of the iliac crest, perpendicular to the long axis of the body with the Lufkin steel tape to the nearest 0.1 cm when the participant was in a standing position at the end of normal expiration[11]. BMI was calculated by weight divided by height (m^2) [12] and waist-to-height ratio was derived by dividing waist circumference (cm) by height (cm).[13] The triceps and subscapular skinfolds were measured on the left side following standard techniques[14] (Lohman, 1988), and were used to calculate percent body fat using Boileau *et al.,* (1985)[15] formulas below:

i) 6-11 years ***constant:*** **boys: 3.4, girls: 1.4.**

$$\%\ body\ fat = 1.35\ (sum\ of\ triceps + subscapular\ skinfold) - 0.012\ (sum\ of\ triceps + subscapular\ skinfold)^2 - constant$$

ii) 12-14 years ***constant:*** **boys: 4.4, girls 12-13 years: 2.4.**

$$\%\ body\ fat = 1.35\ (sum\ of\ triceps + subscapular\ skinfold) - 0.012\ (sum\ of\ triceps + subscapular\ skinfold)^2 - constant$$

iii) 15-18 years ***constant:*** **boys: 5.4, girls 14-15 years: 3.4.**

$$\%\ body\ fat = 1.35\ (sum\ of\ triceps + subscapular\ skinfold) - 0.012\ (sum\ of\ triceps + subscapular\ skinfold)^2 - constant$$

Blood pressure

Blood pressure was measured by connecting subjects to the Finapres (finger arterial pressure) apparatus.[14] Details of how this procedure was conducted are described elsewhere.[17] High blood pressure was defined according to the definition from the National High Blood Pressure Education Programme Working Group on High Blood Pressure in children and adolescents (2004)[18] *i.e*. an average systolic or diastolic blood pressure that is greater or equal to 95th percentile for sex, age and height on three or more occasions.

Statistical analysis

SAS statistical package version 9.0 (2003)[19] was used for data analysis. Descriptive statistics were computed by gender for age, height, weight, BMI, WC, WHtR, systolic blood pressure (SBP), diastolic blood pressure (DBP) and percent body fat (%BF)[20], expressed as mean and standard deviation (SD). Differences among means were investigated by analysis of variance. The ability of WHtR to identify children with high blood pressure was examined. The optimal cut-off value was denoted by the value that had the largest sum of sensitivity and specificity.[21] The area under the curve (AUC) was calculated using the trapezoidal rule and this quantifies the screening performance over different cut-off values. AUC is used as a measure of the performance of a diagnostic test against the ideal and may also be used to compare different tests.[22] AUC ranges on a scale of 0-1, with <0.5 indicating no predictive power and 1 indicating perfect power.[23] Diagnostic accuracy of WHtR to predict high blood pressure was also expressed in the following ways: odds ratio and confidence interval, positive predictive value (PPV) and negative predictive value (NPV). The odds ratio compares the probability of a certain event for two groups. The PPV of a test is the probability that a patient has a positive outcome given that they have a positive test result. This is in contrast to sensitivity, which is the probability that a patient has a positive test result given that they have a positive outcome. Similarly, the NPV is the probability that a patient has a negative outcome given that they have a negative test result, in contrast to specificity, which is the probability that a patient has a negative

test result given that they have a negative outcome.[24] Partial correlation analysis was used to assess the association between the different anthropometric indices and blood pressure followed by stepwise linear regression to assess the predictive ability of each measure.

Results

Descriptive statistics of the children are presented in Table 1. Girls had statistically significantly higher values for all variables except for age, height and WHtR. Using a WHtR cut-off value of 0.5, 7.9% of the girls and 3.4% boys were identified as having excess central adiposity. Overall the prevalence of high blood pressure in the children based on the National High Blood Pressure Education Programme Working Group on High Blood Pressure in children and adolescents cut-off points was 13%. The different sensitivity and specificity values to identify children with hypertension obtained at different cut-off values for WHtR are shown in Table 2 with values for other diagnostic measures. The optimal WHtR cut-off value with the highest sum of sensitivity and specificity values was the same in both boys and girls, namely 0.41. For this reason diagnostic accuracy measures were computed for 0.41 and 0.5 WHtR cut-off values. The areas under the ROC curve for WHtR were 0.62 and 0.55 for boys and girls respectively.

Table 1: Mean and standard deviation for the characteristics of the children

Variable	Boys	Girls	p-value
Age (years)	12.0(1.7)	12.0(1.7)	0.91
Height (cm)	147.8(13.0)	148.8(11.1)	<0.001
Weight (kg)	38.0(11.6)	41.0(11.7)	0.18
Body mass index (kg/m^2)	17.0(3.1)	18.2(3.5)	<0.001
Waist circumference (cm)	60.7(7.4)	61.7(7.8)	0.03
Waist-height ratio	0.41(0.04)	0.41(0.04)	0.13
Systolic blood pressure (mmHg)	99.2(14.0)	104.3(14.2)	<0.001
Diastolic blood pressure (mmHg)	63.8(9.7)	65.5(9.5)	<0.001
% Body fat	14.4(6.2)	22.8(6.9)	<0.001

Table 2: Diagnostic accuracy measures for WHtR as a predictor of blood pressure for boys and girls at 0.41 and 0.5 WC cut-off values

Diagnostic accuracy measures	Waist circumference cut-off values			
	Boys		Girls	
	0.41	0.5	0.41	0.5
Sensitivity (%)	61.90	4.80	62.0	7.60
Specificity (%)	53.60	98.60	51.40	93.90
Positive predictive value	0.53	0.99	0.51	0.97
Negative predictive value	0.38	0.95	0.39	0.92
Odds ratio	1.88	3.45	1.63	3.09
95% Confidence interval	(0.97; 3.67)	(0.61; 19.37)	(0.97; 2.74)	(1.04; 9.20)

Table 3: Mean and standard deviation for the characteristics of the Japanese and South African children

	South African		Japanese	
Variable	Boys	Girls	Boys	Girls
Age (years)	12.0(1.7)	12.0(1.7)	10.9(0.5)	10.9(0.5)
Height (cm)	147.8(13.0)	148.8(11.1)	145.2(7.2)	144.4(6.4)
Weight (kg)	38.0(11.6)	41.0(11.7)	39.2(8.9)	38.3(7.3)
Body mass index (kg/m^2)	17.0(3.1)	18.2(3.5)	18.4(3.0)	18.0(3.2)
Waist circumference (cm)	60.7(7.4)	61.7(7.8)	61.2(7.9)	58.1(6.4)
Waist-height ratio	0.41(0.04)	0.41(0.04)	0.43(0.05)	0.40(0.04)
Systolic blood pressure (mmHg)	99.21(14.0)	104.3(14.2)	113.8(11.2)	99.2(14.0)
Diastolic blood pressure(mmHg)	63.8(9.7)	65.5(9.5)	60.4(7.6)	61.5(7.3)
% Body fat	14.4(6.2)	22.8(6.9)	20.0(8.0)	24.0(7.0)

Table 3 shows the comparison between the South African children in this study with a sample of children aged 6-14 years from a Japanese study which was aimed at determining the best predictor for the presence of cardiovascular disease risk factors among anthropometric indices. The average value of WHtR in these children was between 0.41 and 0.44. Based on the results obtained authors proposed that WHtR could be used for detecting cardiovascular risk in children.

Table 4: 2x2 Table for boys at 0.41 and 0.5 waist circumference cut off values.

	WHtR< 0.41	WHtR>/= 0.41	Total
Non-hypertensive	150	129	279
Hypertensive	16	26	42
Total	166	155	321

	WHtR< 0.5	WHtR>/= 0.5	Total
Non-hypertensive	276	4	280
Hypertensive	40	2	42
Total	316	6	322

Table 5: 2x2 Table for girls at 0.41 and 0.5 waist circumference cut off values.

	WHtR< 0.41	WHtR>/= 0.41	Total
Non-hypertensive	148	144	292
Hypertensive	29	46	75
Total	177	190	367

	WHtR< 0.5	WHtR>/= 0.5	Total
Non-hypertensive	284	8	292
Hypertensive	69	6	75
Total	353	14	367

Table 6: Diagnostic accuracy measures for WHtR as a predictor of high blood pressure in boys and girls at 0.41 and 0.5 WC cut-off values

Diagnostic accuracy measures	Waist circumference cut-off values			
	Boys		Girls	
	0.41	0.5	0.41	0.5
Sensitivity(%)	61.9	4.8	62.0	7.6
Specificity(%)	53.6	98.6	51.4	93.9
Positive predictive value	0.53	0.99	0.51	0.97
Negative predictive value	0.38	0.95	0.39	0.92
Odds ratio	1.88	3.45	1.63	3.09
95% confidence interval	(0.97;3.67)	(0.61;19.37)	(0.97;2.74)	(1.04;9.20)

Tables 4 and 5 show different frequencies of children in each category of true positive, false positive, false negative and true negatives which were used in the calculation of positive and negative predictive values for WHtR as a predictor of high blood pressure. The results obtained for different diagnostic measures are shown in Table 5. The optimal WHtR cut-off value with the highest sum of sensitivity and specificity values was the same in both boys and girls, namely 0.41.

The correlation coefficients between the various anthropometric indices and blood pressure are presented in Table 6 for both boys and girls. The results show that for boys all anthropometric indices were significantly correlated with blood pressure (SBP and DBP) except for WHtR which had no significant correlation with DBP. The strongest correlation was between SBP and WC (r=0.163). In girls, none of the anthropometric indices correlated with SBP, while all the indices, except for WHtR, correlated significantly with DBP. It should, however, be noted that despite the significant correlations, the correlation coefficients were small, ranging from 0.042 to 0.163. Results of the regression analysis for SBP and DBP as dependent variables are presented in Table 7. These results indicate that in boys age was the best predictor for DBP explaining 1% of the variation and BMI explaining 1.4% of the variation in SBP. In girls height and weight were the most important predictors for DBP explaining 1.1% and 1.0% of the variance respectively. Waist circumference was the highest predictor of SBP explaining 0.3% of the variation. The percentage of the variance of these measures explained was less than 5% in both boys and girls.

Table 7: Age adjusted correlation coefficients between blood pressure and anthropometric variables.

	Boys				Girls			
	Systolic blood pressure		Diastolic blood pressure		Systolic blood pressure		Diastolic blood pressure	
Variable	r	p-value	r	p-value	r	p-value	r	p-value
Weight	0.135	0.001	0.111	0.009	0.031	0.459	0.146	<0.0001
Height	0.107	0.012	0.093	0.029	0.011	0.785	0.105	0.011
BMI	0.114	0.007	0.086	0.043	0.035	0.403	0.123	0.003
WC	0.163	<0.0001	0.126	0.003	0.050	0.234	0.092	0.028
WHtR	0.115	0.007	0.082	0.053	0.049	0.236	0.042	0.313

BMI-Body mass index, WC-Waist circumference, WHtR-Waist-to-height ratio

r – correlation coefficient, $p< 0.05$=statistically significant

Table 8: Regression analysis of blood pressure (dependent variable) and anthropometric predictors

	Boys				Girls			
	Systolic blood pressure R^2		Diastolic blood pressure R^2		Systolic blood pressure R^2		Diastolic blood pressure R^2	
Variable	change	p-value	change	p-value	change	p-value	change	p-value
Age	0.004	0.147	**0.010**	0.019	0.001	0.390	**0.007**	0.040
Height	**0.011**	0.012	**0.009**	0.029	0.000	0.800	**0.011**	0.011
Weight	0.007	0.051	0.004	0.135	0.002	0.329	**0.010**	0.016
BMI	**0.014**	0.006	0.001	0.528	0.002	0.350	0.001	0.525
WC	**0.008**	0.028	0.003	0.169	0.003	0.222	0.004	0.137
WHtR	0.000	0.993	0.003	0.224	0.004	0.141	0.002	0.292

Discussion

Based on the sensitivity and specificity results, a WHtR cut-off value of 0.5 for predicting high blood pressure risk in children did not prove to be useful, because the sensitivity was low for boys and girls, (4.8% and 7.6% respectively). The most appropriate WHtR cut-off value seems to be 0.41 for both boys (61.9% sensitivity and 53.6% specificity) and girls (62% sensitivity and 51% specificity). The fact that the optimal WHtR cut-off value for both boys and girls is the same concurs with the fact that in adults there is a known advantage to setting up a unisex cut-off point when using the WHtR as a predictor of CV disease risk.[7] Hara et al.[4] postulated that since WHtR takes into account children's height, a single cut-off point can likely be set for the ratio, without age and gender difference bias.[4] The optimal WHtR cut-off value of 0.41 as observed in the current study is similar to what was observed for Japanese children.[4] The reason for this is probably because children included in the two studies had similar anthropometric characteristics, and in particular they had similar heights and WC for age and sex.

The AUC in the WHtR and blood pressure ROC analyses observed for this study are lower than what has previously been documented (0.62 for boys and 0.55 for girls). Freedman and co-workers,[25] for example, observed the AUC for WHtR to be 0.87 and 0.82 among white and black American children respectively. Genovesi and co-workers also[26] reported that the ROC curves showed a significant ability of WHtR in discriminating hypertensive children in both sexes (0.81 for girls and 0.75 for boys) from children with normal blood pressure. AUC provides a measure of discrimination, which is the likelihood that a subject with abnormal measurements will have a higher probability to be identified than a subject with normal measurements. The values for AUC from the present study indicate poor discrimination, because they are close to 0.5 which indicates no discrimination. Lin et al.[27] argues that AUCs of 0.6-0.7 are considered poor and 0.7-0.8 are fair. The low discriminatory power of WHtR in identifying children with high blood pressure, suggests that it is a poor predictor. This observation could be explained in part by the relatively small number of children with high blood pressure (13%). It could be argued that a minimal critical amount of visceral fat should necessarily be present to exert its role on hypertension; however, the existence of a minimal visceral fat mass for metabolic derangement still needs to be ascertained in children.[28] As WHtR is a measure of central obesity one would have expected different outcomes. However, the diagnostic quality of

WHtR in screening for obesity in children has been previously observed to be poor in 6-12 year old children in France.[22] This might be a possible explanation why WHtR exhibited poor diagnostic quality in the current study on children. Results for odds ratio, PPV and NPV show that at WHtR cut-off value of 0.5 proved to be a better predictor for high blood pressure in children as shown in Table 2. The reason for the conflicting results might be due to the fact that sensitivity and specificity are characteristics of a test and are not affected by the prevalence of the disease. However, although the PPV and NPV give a direct assessment of the usefulness of a test, they are affected by the prevalence of the disease. Bewick and colleagues (2004) stated that 'the decision to use a diagnostic test depends not only on the diagnostic accuracy measures but also on the ultimate benefit to the patient. The prevalence of the outcome, which is the pre-test probability, must also be known. Generally, there is a trade-off between sensitivity and specificity, and the practitioner must make a decision based on their relative importance'.[24]

Studies on the correlation of anthropometric indices and high blood pressure are limited and comprise of different age groups making comparisons difficult.[26,,27,30] The current study showed statistically significant correlations, although the correlation coefficients were generally low. The relatively large sample size (n=1131) could have contributed towards the statistical significance of the correlation between anthropometric variables and blood pressure in the study sample. Results of the linear regression analysis indicated that WHtR was not a predictor of blood pressure in both boys and girls. BMI was the most important predictor of SBP in boys. This finding is consistent with earlier studies.[28-30] Previously age has also been observed to be associated with DBP.[29] It should be noted that in all instances the variation in blood pressure that was accounted for by anthropometrical variables was less than 5%. These outcomes confirm the earlier observation that the AUC was low indicating poor predictive power by WHtR. Several factors might have influenced the strength of the associations and the predictive power observed in this study. These include the relatively low prevalence of both obesity and hypertension in this population. Family history of hypertension and socio-economic factors contributing to stress, or even dietary factors may be stronger predictors of high blood pressure in these children. Schutte and co-workers[17] showed in the same sample of children that low intakes of biotin, pantothenic acid and zinc, together with added sugar and energy accounted for

28.9% of the variance in DBP. This is an indication that blood pressure in this population is significantly associated with dietary risk markers.

A potential limitation of this study is that the sample of children was from only one of the nine provinces in South Africa. However, the children were from both rural, informal settlements and urban areas. This makes it difficult to propose a cut-off of 0.41 for South African children and adolescents in general. Therefore, a nationally representative sample may be required to give a more precise cut-off value. Another limitation might be the fact that blood pressure was measured on one occasion even though there were 10 minute intervals between the readings. In establishing the type of hypertension, blood pressure should be measured on three or more separate occasions.[26] However, the procedure adopted in this study (vascular unloading technique of Penaz together with Physical criteria of Wesseling) provides reliable, non-invasive and continuous estimates of blood pressure.[16] The stage of high blood pressure was not characterised in this study and the question is whether the stage of hypertension contributes to the low predictive value obtained from ROC analyses. Elevated blood pressure may be a sign of underlying disease or it may represent early onset of essential hypertension if, however, the outcomes of the test are unrelated to disease status then the test characteristics can be safely estimated.[21]

In conclusion, results of the present study indicate that WHtR has poor predictive value of high blood pressure in 9-15 year old South African children and adopting a WHtR cut-off value lower than 0.5 for South African children may enhance sensitivity in identifying children at risk for hypertension. However, a nationally representative cohort study may be needed to confirm the optimal cut-off value for more accurate prediction of the presence of hypertension in order to identify high risk children targeted for interventions.

Acknowledgements

This study was supported in part by a South African Sugar Association grant. Prof. HS Steyn and Dr.S. Ellis are acknowledged for statistical analyses. Special thanks to the *THUSABANA* research team for data collection and to the children who participated in this study.

References

1. Hsieh SD, Muto T. The superiority of waist-to-height ratio as an anthropometric index to evaluate clustering of coronary risk factors among non-obese men and women. Prev Med 2005;40:216 –20.
2. Bosy-Westphal A, Geisler C, Onur S, Korth O, Selberg O, Schrezenmeir J. et al. Value of body fat mass vs. anthropometric obesity indices in the assessment of metabolic risk factors. Int J Obes 2006; 30:475– 83.
3. Savva SC, Tornaritis M, Savva ME, Kourides Y, Panagi A, Silikiotou N. et al. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. Int J Obes Relat Metab Disord 2000; 24(11):1453-1458.
4. Hara M, Saitou E, Iwata F, Ikada T, Harada K. Waist-to-height ratio is the best predictor of cardiovascular disease risk factors in Japanese schoolchildren. J Atheroscler Thromb 2002; 9:127-132.
5. Kahn HS, Imperatore G, Cheng YJ. A population-based comparison of BMI percentiles and waist-to-height ratio for identifying cardiovascular risk in youth. J Pediatr 2005; 146:482-488.
6. Ashwell M. Waist to height ratio and the Ashwell shape chart could predict the health risks of obesity in adults and children in all ethnic groups. Nutr Food Science 2005; 35:359-364.
7. Ashwell M, Lejeune S, McPherson K. Ratio of waist circumference to height may be better indicator of need for weight management. BMJ. 1996;312:377.
8. Sung RYT, So H-K, Choi K-C, Nelson EAS, Li AM, Yin JAT. et al. Waist circumference and waist-to height ratio of Hong Kong Chinese children. BMC Pub Health 2008;8:324-338.
9. Simons-Morton DG, Obarzanek E. Diet and blood pressure in children and adolescents. Pediatr Nephrol 1997;11:244-249.
10. Kruger R, Kruger HS, Macintyre UE. The determinants of overweight and obesity among 10–15-year-old schoolchildren in the North West Province, South Africa—the THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) study. Public Health Nutr 2006;9: 351–358.

11. Mukuddem-Petersen J and Kruger HS. Association between stunting and overweight among 10–15-y-old children in the North West Province of South Africa: the THUSA BANA Study. Int J Obes 2004;28:842–851.
12. Healthy weight (Online). 2009 Jul 27 (cited 2010 Apr 13); Available from: URL:http:www.cdc.gov/healthyweight/assessing/bmi/adult_BMI/index.html
13. Tseng C-H. Waist-to-Height Ratio is independently and better associated with urinary albumin excretion rate than waist circumference or waist-to-hip ratio in Chinese adult type 2 diabetic women but not men. Diabetes Care 2005;28(9): 2249-2251.
14. Lohman TG. Anthropometry and body composition. In Lohman TG, Martorell R, eds. Anthropometric standardization manual, 1988. Champaign, IL: Human Kinetics Books.
15. Boileau RA, Lohman TG, Slaughter MH. Exercise and body composition of children and youth. Scand Med Sci Sports, 1985; 7(1): 17-27.
16. Silke B, McAuley D. Accuracy and precision of blood pressure determination with the Finapres: an overview using re-sampling statistics. J Hum Hypertens 1998;12:403-409.
17. Schutte AE, Van Rooyen JM, Kruger HS, Malan NT, De Ridder JH. Dietary risk markers that contribute to the aetiology of hypertension in black South African children: the THUSABANA study. J Hum Hypertens 2003;17:29-35.
18. The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents (supplement article): National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. Pediatrics 2004:114(2):555-576.
19. SAS Institute Inc., 2003. SAS Institute Inc., SAS OnlineDoc, Version 9.1, Cary, NC
20. Boileau RA, Lohman TG, Slaughter MH. Exercise and body composition of children and youth. Scan J Sports Sci 1985;7(1):17-27.
21. Campbell MJ, Machin D. Medical Statistics: A common sense approach, 3rd ed. John Wiley and Sons, West Sussex, England; 2000.
22. Hubert H., Guinhouya CB., Allard L, Durocher A. Comparison of the diagnostic quality of body mass index, waist circumference and waist-to-height ratio in screening skin-fold determined obesity among children. J Sc Med Sport 2009;12:449-451.
23. Farr BM, Shapiro DE. Diagnostic tests: Distinguishing good tests from bad and even ugly ones. Infec Control Hosp Epidemiol 2000;21:278-284.

24. Bewick V, Cheek L, Ball J. Statistics review 13: Receiver operating characteristics curves. Crit care 2004; 8(6): 508-512.

25. Freedman DS, Kahn HS, Mei Z, Grummer-Strawn LM, Dietz WH, Srinavasan SR et al. Relation of body mass index and waist-to-height ratio to cardiovascular disease risk factors in children and adolescents: the Bogalusa Heart Study. Am J Clin Nutr 2007;86:33-40.
26. Genovesi S, Antolini L, Giussani M, Pieruzzi F, Galbiati S, Valsecchi G et al. Usefulness of waist circumference for the identification of childhood hypertension. J Hypertens 2008;26:1563-1570.
27. Lin W-Y, Lee L-T, Chen C-Y, Lo H, Hsia H-H, Liu I-L. et al. Optimal cut-off values for obesity: using simple anthropometric indices to predict cardiovascular risk factors in Taiwan. Int J Obes 2002;26**:**1232-1238.
28. Lima E. Assessment of risk factors associated with elevated blood pressure in children and adolescents. J Pediatr 2004;80(1):3-4.
29. Janssen I., Katzmarzyk PT., Srinivasan SR., Chen W., Malina RM., Bouchard C. et al. Combined influence of body mass index and waist circumference on coronary artery disease risk factors among children and adolescents. Pediatrics 2005; 115(6):1623-1630.
30. Urrita-Rojas X, Egbuchunam CU, Bae S, Menchaca J, Bayona M, Patrick AR. et al. High blood pressure in school children: prevalence and risk factors. BMC Pediatr 2006;6:32-4

6

GENERAL CONCLUSIONS & RECOMMENDATIONS

Children grow, horses race: Is the adiposity rebound a critical period for later obesity?

(Cole, 2004)

6.0 General summary, conclusions & recommendations

6.1 Introduction

6.2 Summary, conclusions and recommendations

6.3 Novel(unique) findings of this study

6.4 Summary of research line of thinking

6.5 References

6.1 Introduction

The importance of measuring body composition has increased due to the need to assess changes in nutritional status, which can affect body reserves differentially. Furthermore, the worldwide epidemic of obesity and its association with chronic disease has contributed to the need to study body composition. The increasing prevalence of childhood overweight and obesity has become a growing concern for public health, as nearly one-third of overweight children grow up to become overweight or obese as adults (Whitaker *et al*., 1997) with increased risk of chronic diseases (Goran, 2001) such as diabetes, cardiovascular disease, hypertension, and cancer (Mokdad *et al.*, 2003), elevated health care costs, and reduced quality of life (Calle *et al*., 2004). Obesity is a complex condition with serious social and psychological dimensions affecting virtually all ages and socio-economic groups (WHO, 2010). While genes are important in determining a person's susceptibility to weight gain, energy balance is determined by calorie intake, physical activity and other factors. The rising epidemic reflects the profound changes in society and in behavioural patterns of populations over recent decades. Economic growth, modernization, urbanization and globalization of food markets are just some of the forces thought to underlie the epidemic; these changes in society and the worldwide nutrition transition are driving the obesity epidemic (WHO, 2010). Independent of total adiposity an upper body distribution of fat is associated with poor health and plays an important role in the pathogenesis of metabolic syndrome (Hamdy *et al.*, 2006).

Waist circumference (WC) is positively correlated with abdominal fat and is an independent indicator of health risk associated with abdominal obesity (Brenner *et al*., 2010); hence its

measurement provides a simple and practical method for estimating abdominal fat. Therefore, the focus of this thesis was to examine WC distribution patterns of black South African children in relation to health risk. In particular, this study examined the sensitivity of waist-to-height ratio (WHtR) in predicting high blood pressure in black South African (SA) children. Cut-off values of WHtR for the reliable prediction of high blood pressure in children from different populations have been published (Ashwell *et al*., 1996, Hara *et al*., 2005) but to date none of these has been validated for use in South African children. It is important to note that the risk for an individual will be influenced by a unique combination of factors which must be considered in addition to BMI and/or WC (NIH, 1998).

Research has suggested that undernutrition in early life may play a role in promoting obesity later in life. In particular, studies on 3 continents showed that nutritional stunting, which is usually caused by chronic undernutrition (Waterlow, 1992) was positively associated with adult fatness (Sawaya *et al.,* 1995). However, little is known about potential underlying mechanisms, and as part of this thesis an investigation of whether increased total and central adiposity is found in stunted children from rural communities in SA was conducted. This was done in order to elucidate whether the high rates of stunting prevalent in South Africa are possibly contributing to the increase in obesity.

Thirdly the WC percentile distribution of black South African children from different populations was described and compared to African-American data. Population studies used to develop WC percentiles and cut-off points have been derived from predominantly Caucasian populations in the USA and Europe (Conen *et al*., 2004). There is evidence that certain ethnic or racial groups may differ from Caucasians in their body fat levels and fat distribution patterns, and in their degree of health risk (Deurenberg *et al*., 1998, He *et al*., 2001). While international cut-off points may be useful for the identification of children at risk of obesity related health complications, they may not be appropriate for use in population specific screening programmes (Deurenberg *et al*., 2001). Exporting cut-off points purely based on industrialized countries' settings, to developing countries, ignores the fact that the growth patterns of children and burdens of disease vary widely between these countries. It would be useful to have an international reference because this would make it possible to conduct comparative studies on

the obesity situation in different countries. However, if the aim is to detect the prevalence of excess body fat in a country, for the purposes of applying this in public health programmes, national reference values are more appropriate because they reproduce the variability within the population that is to be evaluated (Vitolo *et al.*, 2007). It is, therefore, necessary to develop and or redefine appropriate cut-off points which are country and ethnic-specific taking into account the significance of local factors contributing to the development of obesity in specific populations. Miranda and Zaman (2010) argue that applying research results from the rich world to the problems of the poor may be a tempting, potentially easy and a convenient solution, but such an approach runs the risk of exporting failure.

6.2 Summary, conclusions and recommendations

Chapter 2 of this thesis reviews the literature on fat distribution in children and its health implications. A mounting volume of research indicates that central distribution of body fat is associated with a higher risk of morbidity and mortality than a more peripheral distribution. It is, therefore, imperative to assess children who are overweight or obese, not only to determine the extent of adiposity, but also for the body fat distribution and the presence of co-morbid factors (Ofei, 2005).

In Chapter 3 the findings indicate that despite data from a number of studies providing evidence that children who are growth retarded at birth have an increased risk of becoming obese in later life (Barker *et al*., 1997; Whitaker *et al*., 1998; Ong *et al.*, 2000), this association was not observed in children 6-15 years old. Hence the hypothesis that stunting influences obesity is accepted for WHtR as results indicate a higher WHtR for stunted children, but rejected for WC as results did not show increased WC in stunted children. The risk of becoming obese due to stunting, which implies foetal programming of adult obesity, is particularly likely to occur when a low body weight at birth from intrauterine growth retardation (IUGR) is over-compensated for by a catch-up growth in later life, and when this adiposity rebound occurs early in childhood (Barker *et al*., 1997). Traditional explanations for increased risk of overweight were reduced physical activity levels and an increase in fat intake, however, increasingly the role of chronic

undernutrition in early life is being recognized (El-Taguri *et al*., 2009). In this population this association needs to be investigated further in older children.

Chapter 4 described the distribution of WC in black South African children from different populations. During childhood, body composition undergoes dynamic changes that makes it necessary to have a range of cut-off values, like those of body mass index (BMI) or WC, unlike the absolute cut-off values as applied to adults (Guntsche *et al*., 2010). To overcome this problem the use of standard deviation scores (SDS) and percentiles is recommended (Hirschler *et al*., 2007). Obtaining a reference percentile table demands the assessment of a large number of healthy children, and strictly speaking the results apply only to the population under study, making comparisons among different populations difficult. This study demonstrated differences in the WC distribution of children from different geographic locations and from those of African-American children. Nonetheless, the present study cannot be extrapolated to the whole population of black South African children, since this analysis was performed on non representative samples. The results show that South African children had consistently smaller WC than African-American children. South Africa, as a multicultural society, will need to develop nationally representative WC curves to describe the distribution of WC in children. These curves will be useful in the development of cut-off values for abdominal obesity which could be implemented in identification of children as an important pre-requisite of the implementation of interventions to prevent long term obesity outcomes. Furthermore, these distributions can be used as base curves that can be applied in analyzing trends in future.

In Chapter 5 the diagnostic accuracy of WHtR in identifying children with high blood pressure is investigated. The WHtR in children seems to be more closely related to paediatric cardiovascular disease risk than uncorrected waist alone. However, some authors prefer to use the measure of WC without correcting it for height (Sung *et al*., 2008). Despite all this, a cut-off value of WHtR for the reliable prediction of high blood pressure in children is still lacking. The findings of this research indicated that a cut-off value less than the recommended 0.5 had a higher sensitivity; therefore, the hypothesis that the cut-off point of 0.5 is sensitive enough to identify South African children with high blood pressure is rejected. High sensitivity is necessary to avoid failure of identifying abdominally obese children and high specificity of the

screening tool ensures that non-obese children are not misclassified as obese. Therefore, it is crucial that further research should be conducted to dispute or verify this finding if it is to be adopted for use in paediatric practice particularly in black South African children.

6.3 Novel (unique) findings of this research

The proposed mechanism on how stunting influences later body composition is still unclear, but apparently the influence occurs at different time points for different populations (Darnton-Hill e*t al*., 2004). It may well be that stunted children are programmed to accumulate greater body fat at central sites during adolescence and that the differences between stunted and non-stunted children only appear at an older age or adulthood. We have been unable to show, however, that these changes are evident in the age groups studied, therefore, it might be worthwhile to further investigate the age range at which particular populations develop obesity as a result from being stunted earlier in life. Research has not fully demonstrated an altered body composition in South African children and this research has contributed to an existing body of information that is needed to establish whether stunted children become obese later in life in the African context.

In the current study, gender and age-specific WC percentiles for black South African children were described for the first time and compared with those of African-American children. These percentile curves provide a starting point that can be applied in analysing trends as well as making comparisons with results of similar studies performed in other countries and can serve as a baseline against which future data can be compared. WC is advocated as an indicator for central obesity because it is a good predictor of abdominal fat and is more closely related to the development of cardiovascular disease and type 2 diabetes (Cameron *et al*., 2009). Age and sex specific WC percentiles have been reported for children of different countries and differences have been observed in different populations (Liu *et al*., 2010). The results indicate clearly that the median WC of children from SA studies is smaller than those of A-A children, it is therefore important to describe the WC distribution so that cut-off points for defining central obesity can be established for this target population.

The study is one of the few to report that a WHtR cut-off of 0.5 may not be appropriate for identifying South African children at risk of high blood pressure. Similar to many other

developing countries, the epidemiologic transition along with rapid lifestyle changes make South African children prone to cardiovascular risk factors such as high blood pressure and as a result, to chronic diseases later in life. Despite all this, a cut-off value of WHtR for the reliable prediction of high blood pressure in children is still lacking and greater efforts are needed to assess the use of international cut-off points in identifying children and adolescents with high blood pressure. To our knowledge most of international cut-off points have not been assessed for their accuracy in screening populations in developing countries.

In summary, from observations in this research it seems that adopting results from the modern world to developing countries may not be appropriate. The foremost important issue in establishing population specific reference values is to ensure that the values are truly representative of the population from which they are obtained (Solberg *et al*., 1993). This means that it is essential that context-appropriate health research and health interventions take place in developing countries in order to generate knowledge that is relevant and applicable to local settings. A clear knowledge of the present situation helps to guide future planning. There are limitations when it comes to conducting research in the developing world in order to generate information that can be useful, within the different prevailing circumstances. Withstanding the use of foreign references may also pose problems, therefore, caution should always be taken when screening for disease risk. In the absence of locally available data it may seem appropriate to use foreign reference standards but efforts should be made to explore possible collaborative work in order to generate local data. An example of such a study is the INTERHEART: A Global Case-Control Study of Risk Factors for Acute Myocardial Infarction (Yusuf, *et al*., 2004). The study involved 15,000 patients with a first acute myocardial infarction (AMI) and 15,000 asymptomatic control subjects (age and sex matched) drawn from 262 centers in 52 countries throughout Asia, Europe, the Middle East, Africa, Australia, and North and South America. The aim of this study was to determine the associations between a wide array of risk factors within populations defined by ethnicity and/or geographic region, and to assess the relative importance of these risk factors across these populations. The investigators hypothesized that the relative impact of conventional risk factors (smoking, hypertension, elevated cholesterol, and diabetes) and emerging risk factors (glucose abnormalities, abdominal obesity, homocysteine, and other

nutritional and psychosocial factors) for cardiovascular disease differ between people of varying ethnic and geographic origin.

The inappropriateness of foreign cut-off points based on reference data cannot be ignored, because reference data represents the current situation in a particular country and cannot be regarded as standards to indicate disease risk (Baya Botti *et al*., 2010). Local data are important because they take into account prevailing condition of particular locations. These factors in most instances have major influences in the growth of children. At this point in time, in the absence of locally developed cut-off values for different risk markers warrants research because the associations between being overweight and obese with disease outcomes cannot be overlooked. It is fundamental to detect risk at an early stage so that appropriate intervention can be initiated timeously. In view of the above it is recommended that efforts should be directed towards collaborative efforts for multinational studies to generate standard cut-off points to indicate disease risk in children. Standards reflect a 'normal' body composition with low risk for disease, which is distinct from references, a description of body composition of a particular population, including unhealthy underweight or overweight individuals (Baya Botti *et al*., 2010). This will not only produce relevant research outputs but will enable the pooling of resources to strengthen research initiatives. Governments should also be committed to support research. Without such a national commitment and effective new approaches to making the environment more favourable to maintaining healthy weight, the current trends will not be reversed. It is, therefore, imperative to screen and identify children with abdominal obesity early to prevent the growing public health problem of childhood obesity in the subpopulation.

6.4 Summary of research findings

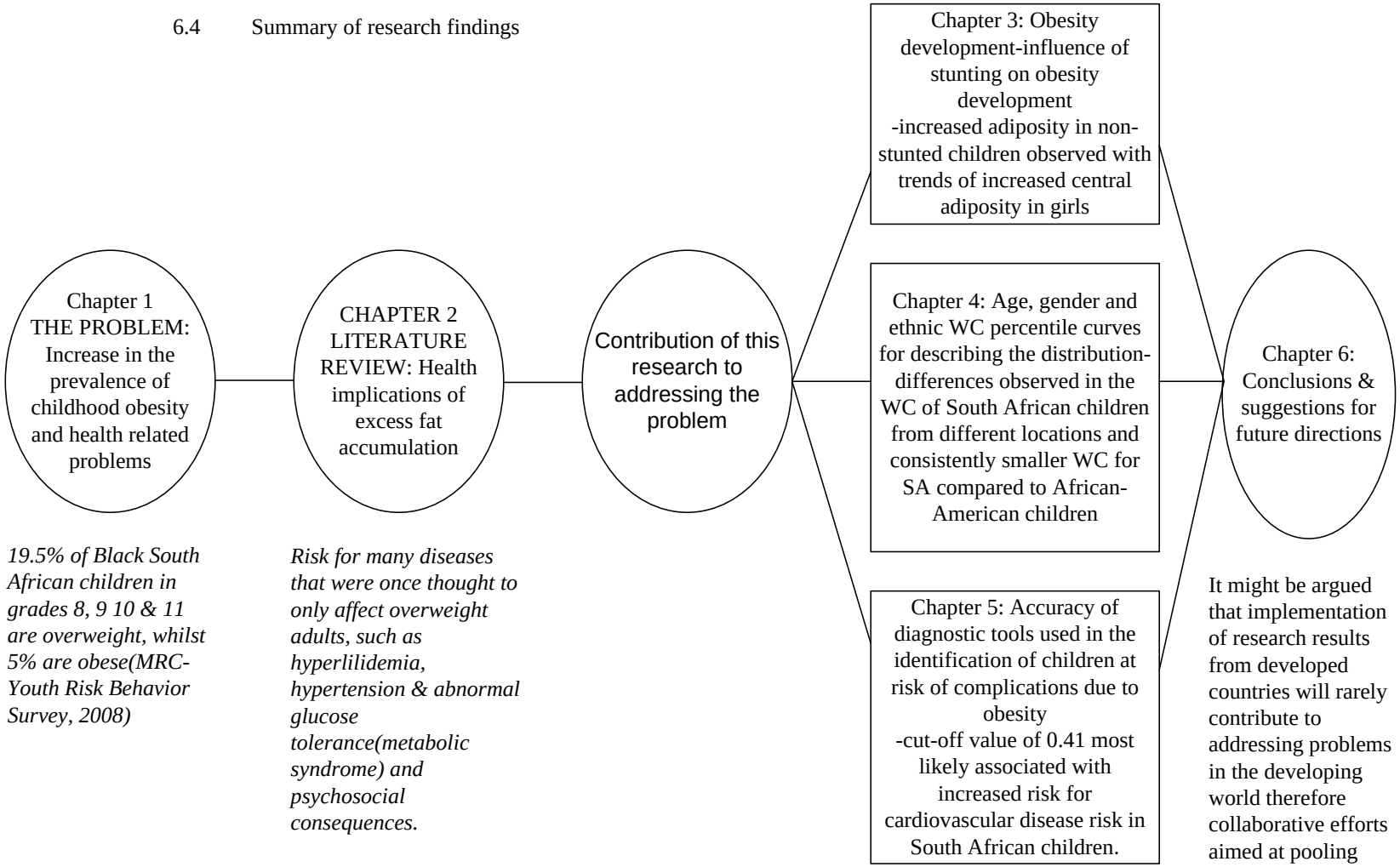


Figure 1: Summary of current research

6.5 References

ASHWELL, M., LEJEUNE, S., MCPHERSON, K. 1996. Ratio of waist circumference to height may be better indicator of need for weight management. *British medical journal*, 312:377.

BARKER, M., ROBINSON, S., OSMOND, C & BARKER, D.J. 1997. Birth weight and body fat distribution in adolescent girls. *Archives of disease in childhood,* 77(5):381-383.

BAYA, B.A., PÉREZ-CUETO, F.J., VASQUEZ, M. P.A. & KOLSTEREN, P.W. 2010. International BMI-for-age references underestimate thinness and overestimate overweight and obesity in Bolivian adolescents. *Nutrición Hospitalaria,* 25(3):428-36.

BRENNER, D. R., TEPYLO, K., ENY, K.M., CAHILL, L.E., & EL-SOHEMY, A. 2010. Comparison of body mass index and waist circumference as predictors of cardio-metabolic health in a population of young Canadian adults. *Diabetology & metabolic syndrome,* 2**:**28

CALLE, E.E. & KAAKS, R. 2004. Overweight, obesity and cancer: epidemiological evidence and proposed mechanisms. *Nature reviews cancer,* 4(8):579 –591.

CAMERON, A.J., DUNSTAN, D.W., OWEN, N., ZIMMET, P.Z., BARR, E.L., TONKIN, A.M. et al. 2009. Health and mortality consequences of abdominal obesity: evidence from the AUSDiab study. *The medical journal of Australia,* 191(4):202-208

CONNEN, D., WIETLISBACH, V., BOVET, P., SHAMLAYE, C., RIESEN, W., PACCAND, F. & BURNIER, M. 2004. Prevelence of hyperuricemia and relation to serum uric acid with cardiovascular risk factors in a developing country. *BMC public health,* 4:9

DARTON-HILL, I., NISHIDA, C. & JAMES, W.P.T. 2004. A life course approach to diet, nutrition and the prevention of chronic diseases. *Public health nutrition,* 7(1A0:101-121

DEURENBERG, P. & DEURENBERG-YAP, M. 2001. Differences in body-composition assumptions across ethnic groups: practical consequences. *Current opinion in clinical nutrition and metabolic care,* 4: 377–383.

DEURENBERG, P., YAP, M., VAN STAVEREN, W.A. 1998. Body mass index and percent body fat: a meta-analysis among different ethnic groups. *International journal of obesity and related metabolic disorders,* 22(12):1164-1171.

EL TAGURI, A., BESMAR, F., ABDEL MONEM, A., BETILMAL, I., RICOUR, C. & ROLLAND-CACHERA, M-F. 2009. Stunting is a major risk factor for overweight: results from national surveys in 5 Arab countries. *Eastern Mediterranean health journal,* 15(3):549.

FU, W.P.C., LEE, H.C., NG, C.J., TAY, Y-K. D., KAU, C.Y., SEOW, C. J. et al. 2003. Screening for childhood obesity: international *vs.* population specific definitions. Which is more appropriate? *International journal of obesity,* 27: 1121–1126.

GORAN, M.I. 2001. Metabolic precursors and effects of obesity in children: a decade of progress, 1990-1999. *American journal of clinical nutrition,* 73(2):158-171.

HAMDY, O., PORRAMATIKUL, S. & AL-OZARI, E. 2006. Metabolic obesity: the paradox between visceral and subcuteneos fat. *Current diabetes reviews,* 2:367-373

HARA, M., SAITOU, E., IWATA, F., IKADA, T & HARADA, K. 2002. Waist-to-height ratio is the best predictor of cardiovascular disease risk factors in Japanese schoolchildren. *Journal of atherosclerosis and thrombosis,* 9:127-132.

HE, M., TAN, K.C., LI, E.T. & KUNG, A.W. 2001. Body fat determination by dual energy X-ray absorptiometry and its relation to body mass index and waist circumference in Hong Kong Chinese. *International journal of obesity and related metabolic disorders,* 25(5):748-52.

HIRSCHLER, V., MACCALLINI, G., CALCAGNO, M., ARANDA. C. & JADZINSKY, M. 2007. Waist circumference identifies primary school children with metabolic syndrome abnormalities. *Diabetes technology and therapeutics,* 9: 149-157.

JEFFERY, R. W. 2001. Public health strategies for obesity treatment and prevention. *American journal of health behavior,* 25(3): 252-259.

KUMANYIKA, S. K. 2001. Mini symposium on obesity: overview and some strategic considerations. *Annual reviews,* 22:293-308.

LIU, A., HILL, A.P., HU, X., LI, Y., DU, L., XU, Y., BYRNE, N.M. & MA, G. 2010. Waist circumference cut-off values for the prediction of cardiovascular risk factors in Chinese school aged children: a cross-sectional study. *BMC public health*,10:82

MIRANDA, J. J. & ZAMAN, M.J. 2010. Exporting failure: why research from rich countries may not benefit the developing world. *Rev. saúde pública,* 44(1):185-189

MOKDAD, A.H., FORD, E.S., BOWMAN, B.A., DIETZ, W.H., VINICOR, F., BALES, V.S. & MARKS, J.S.. 2003. Prevalence of obesity, diabetes, and obesity-related health risk factors, 2001. *Journal of American Medical Association,* 289:76 –79.

NATIONAL INSTITUTES OF HEALTH, NATIONAL HEART, LUNG & BLOOD INSTITUTE. 1998. Clinical Guidelines on the Identification, Evaluation, and Treatment of overweight and obesity in Adults-The Evidence Report. *Obesity research*, 6(2):51S-209S.

NESTLE, M. & JACOBSON, M. F. 2000. Halting the obesity epidemic: a public health policy approach. *Public health reports,* 115: 12-24.

WHO. 2010. Obesity and overweight: Global Strategy on Diet, Physical Activity and Health, WHO, Geneva, Switzerland. Available at: http://www,whoint/dietphysicalactivity/publications. Date of access: 1st Aug. 2010

OFEI, F. 2005. Obesity – a preventable disease. *Ghana medical journal*, 39(3): 98-101

ONG, K.K., AHMED, M.L., EMMETT, P.M., PREECE, M.A., DUNGER, D.B. 2000. Association between post natal catch-up growth and obesity in childhood: a prospective study. *British medical journal,* 320(7240): 967-971

SAWAYA, A. L., ROBERTS, S. 2003. Stunting and future risk of obesity: principal physiological mechanisms. *Cad Saude Publica*, 19(Suppl 1): S21–S28.

SAWAYA, A.L., DALLAL, G., SOLYMOS, G., DE SOUSA M.H., VENYURA, M.L., ROBERTS, S.E., et al. 1995. Obesity and malnutrition in a shantytown population in the city of São Paolo, Brazil. *Obesity research,* 3(suppl):107S–115S

SOLBERG, H.E. 1993. A guide to International Federation of Clinical Chemistry recommendations on reference values. *Journal of international federation of clinical chemistry*, (5)4:160-164

SUNG, R.Y., SO, H.K., CHOI, K.C., NELSON, E.A., LI, A.M., YIN, J.A et al. 2008. Waist circumference and waist-to-height ratio of Hong Kong Chinese children. *BMC public health*, 8: 324.

VITOLO, M.R., CAMPAGNOLO, P. D., BARROS, M.E., GAMA, C.M. & LOPEZM F.A. 2007. Evaluation of two classifications for overweight among Brazilian adolescents. *Rev. saúde pública*, 41(4):653-656

WATERLOW, J.C. 1992. Protein energy malnutrition. London: Edward Arnold

WHITAKER RC, PEPE MS, WRIGHT JA, SEIDEL KD, DIETZ WH. 1998. Early adiposity rebound and the risk of adult obesity. *Pediatrics,* 101(3): e5

WHITAKER, R.C., WRIGHT, J.A., PEPE, M.S., SEIDEL, K.D & DIETZ, D.H. 1997. Predicting obesity in young adulthood from childhood and parental obesity. *New England journal of medicine,* 337(13):869-873

YUSUF, S., HAWKEN, S., ÔUNPUU, S., DANS, T., AVEZUM, A., LANAS F. et al. 2004. Effect of potentially modifiable risk factors associated with myocardial infarction in 52 countries (the INTERHEART study): case-control study. *Lancet*, 364(9438):937-952

ADDENDA

ADDENDUM 1: Reviewer's comments on the article: Sensitivity of waist-to-height ratio in identifying children with high blood pressure.

Reviewer #2: The article is well-written, lucid and logical. However, the conclusion does not corroborate entirely with the abstract. I would suggest that the abstract also alludes to the inconclusive value of the WHtR as a marker as is mentioned in the conclusion. By mentioning the weaknesses of the article, the authors allow for a balanced view of their data. Perhaps one could investigate the relation of WHtR with other risk factors, e.g. blood lipid profiles? This article should stimulate discussion in the quest for determining ethnic specific cut-off values and guidelines for management of cardiovascular risk in children, and therefore has value.

Reviewer #3:
The only information that is of note is the use of WHtR for the purpose of predicting hypertension, though similar results were found in Japanese, as reported by Hara, et al. (2002)

The manuscript in its current format is not suitable for publication as full paper as the scientific evidence lacks depth and is not novel. In order to contribute to cumulative evidence, please consider presenting the findings around the 2x2 table, WHtR (cut-off 0.41) versus hypertension (National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents cut-off points), giving frequencies, diagnostic statistics (Sensitivity, specificity, PPV, NPV), odds ratio and its 95% confidence interval, and a short comparison with the Japanese study in a letter to the Journal.

ADDENDUM 2: Letter on the sensitivity of waist-to-height ratio in identifying children with high blood pressure submitted for publication to the Cardiovascular Journal of Africa.

The sensitivity of waist-to-height ratio in identifying children with high blood pressure.

Boitumelo S Motswagole[1]MSc, H Salome Kruger[1]PhD, Mieke Faber[2]PhD, Johannes M van Rooyen[3] DSc, J Hans de Ridder[3] PhD

[1]Centre of Excellence for Nutrition, North-West University, Private Bag X6001, Potchefstroom 2520, South Africa. Tel: +27 18 299 2482, Fax: +27 18 299 2464; [2]Nutritional Intervention Research Unit, Medical Research Council, PO Box 19070, Tygerberg 7505, South Africa; [3]ypertension in Africa Research Team(HART), North-West University, Potchefstroom 2520, South Africa.

Correspondence to: H Salome Kruger, Centre of Excellence for Nutrition, North-West University, Private Bag X6001, Potchefstroom 2520, South Africa. Tel: +27 299 2482, Fax: +27 18 299 2464, E-mail: Salome.Kruger@nwu.ac.za

Studies in both adults[1-2] and children[3-5] have suggested that waist-to-height ratio (WHtR) is more strongly associated with cardiovascular (CV) risk factors than body mass index (BMI). Several clinically relevant properties of the waist-to-height ratio have been pointed out, including its sensitivity as an early warning of health risk and its simplicity for calculation. Also, it has been suggested that the same cut-off value may be used for both genders[6], hence it has been proposed as an alternative measure for assessing central fatness in children on the basis that it is relatively age-independent and that in normalizing for growth it might preclude the need for age-related reference charts[3-4, 7]. This index, however, is yet to be validated within the paediatric population[8]. Previously Ashwell and colleagues (1996)[9] proposed an age-independent universal cut-off value of 0.5 for predicting CV risk; however this value is yet to be tested to predict CV risk in children (Sung et al 2008)[10]. This study was aimed at assessing the diagnostic accuracy of WHtR as a marker for future cardiovascular events like high blood pressure in South African children and whether WHtR could be used as a marker for high blood pressure.

The study population consisted of 919 black South African children aged 6 to 15 years drawn from the THUSA BANA (Transition and health during urbanisation of South Africans; BANA-children) study conducted between May 2000 to June 2001. All anthropometric measurements were taken according to standard methods[11] by qualified anthropometrists using calibrated apparatus. Weight was measured to the nearest 0.1kg on a calibrated electronic scale (Precision Health Scale) and height was measured to the nearest 0.1cm with a stadiometer calibrated using a steel measuring tape. Waist circumference (WC) was measured half way between the superior ridge of the ileum and the lower border of the lowest floating rib with a flexible Lufkin anthropometric steel tape to the nearest 0.1 cm[12]. BMI was calculated by weight divided by height $(m)^2$ and waist-to-height ratio was derived by dividing waist circumference (cm) by height (cm). The triceps and subscapular skinfolds were measured on the left side following standard techniques[13] (Lohman, 1988), and were used to calculate percent body fat using Boileau *et al.,* (1985)[14] formulas that follow:

i) 6-11 years ***constant:*** **boys: 3.4, girls: 1.4.**

$$\%\ body\ fat = 1.35\ (sum\ of\ triceps + subscapular\ skinfold) - 0.012\ (sum\ of\ triceps + subscapular\ skinfold)^2 - constant$$

ii) 12-14 years ***constant:*** **boys: 4.4, girls 12-13 years: 2.4.**

$$\%\ body\ fat = 1.35\ (sum\ of\ triceps + subscapular\ skinfold) - 0.012\ (sum\ of\ triceps + subscapular\ skinfold)^2 - constant$$

iii) 15-18 years ***constant:*** **boys: 5.4, girls 14-15 years: 3.4.**

$$\%\ body\ fat = 1.35\ (sum\ of\ triceps + subscapular\ skinfold) - 0.012\ (sum\ of\ triceps + subscapular\ skinfold)^2 - constant$$

Blood pressure was taken by connecting subjects to the Finapres (finger arterial pressure) apparatus[15]. Details of how this procedure was conducted are described elsewhere[16]. High blood pressure was defined according to the definition from the National High Blood Pressure Education Program Working Group on High Blood Pressure in children and adolescents (2004)[17], i.e. an average systolic or diastolic blood pressure that is greater or equal to 95th percentile for sex, age and height.

SAS statistical package version 9.0 (2003)[18] was used for data analysis. Descriptive statistics were computed by gender for age, height, weight, BMI, WC, WHtR, systolic blood pressure (SBP), diastolic blood pressure (DBP) and percent body fat(%BF), expressed as mean and standard deviation(SD). Differences among means were investigated by analysis of variance. Diagnostic accuracy of WHtR to predict high blood pressure was expressed in the following ways: sensitivity and specificity, odds ratio and confidence interval, positive predictive value (PPV) and negative predictive value (NPV). Sensitivity measures the proportion of actual positives which are correctly identified as such and specificity measures the proportion of negatives which are correctly identified. The optimal cut-off value was denoted by the value that had the largest overlap of sensitivity and specificity[19]. The odds ratio compares the probability of a certain event for two groups. The PPV of a test is the probability that a patient has a positive outcome given that they have a positive test result. This is in contrast to sensitivity, which is the probability that a patient has a positive test result given that they have a positive outcome. Similarly, the NPV is the probability that a patient has a negative outcome given that they have a negative test result, in contrast to specificity, which is the probability that a patient has a negative test result given that they have a negative outcome[20].

Characteristics of the children are presented in Table 1. Girls had statistically significantly greater values for all variables except for age, height and WHtR. The prevalence of abdominal adiposity using a WHtR cut-off value of 0.5 identified 7.9% of the girls and 3.4% boys as having excess central adiposity. Overall the prevalence of hypertension in these children was 13%.

Table 1: Mean and standard deviation for the characteristics of the children

Variable	Boys	Girls	p-value
Age (years)	12(1.7)	12(1.7)	0.91
Height (cm)	147.8(13.0)	148.8(11.1)	<0.00
Weight (kg)	38.0(11.6)	41.0(11.7)	0.18
Body mass index (kg/m^2)	17.0(3.1)	18.2(3.5)	<0.00
Waist circumference (cm)	60.7(7.4)	61.7(7.8)	0.03
Waist-height ratio	0.41(0.04)	0.41(0.04)	0.13
Systolic blood pressure (mmHg)	99.21(14.0)	104.3(14.2)	<0.00
Diastolic blood pressure (mmHg)	63.8(9.7)	65.5(9.5)	<0.00
% Body fat	14.4(6.2)	22.8(6.9)	<0.00

Table 2: Mean and standard deviation for the characteristics of the Japanese and South African children

	South African		Japanese	
Variable	Boys	Girls	Boys	Girls
Age (years)	12.0(1.7)	12.0(1.7)	10.9(0.5)	10.9(0.5)
Height (cm)	147.8(13.0)	148.8(11.1)	145.2(7.2)	144.4(6.4)
Weight (kg)	38.0(11.6)	41.0(11.7)	39.2(8.9)	38.3(7.3)
Body mass index (kg/m^2)	17.0(3.1)	18.2(3.5)	18.4(3.0)	18.0(3.2)
Waist circumference (cm)	60.7(7.4)	61.7(7.8)	61.2(7.9)	58.1(6.4)
Waist-height ratio	0.41(0.04)	0.41(0.04)	0.43(0.05)	0.40(0.04)
Systolic blood pressure (mmHg)	99.21(14.0)	104.3(14.2)	113.8(11.2)	99.2(14.0)
Diastolic blood pressure (mmHg)	63.8(9.7)	65.5(9.5)	60.4(7.6)	61.5(7.3)
% Body fat	14.4(6.2)	22.8(6.9)	20.0(8.0)	24.0(7.0)

Table 2 shows the comparison between the South African children in this study with a sample of children aged 6-14 years from a Japanese study which was aimed at determining the best predictor for the presence of cardiovascular disease risk factors among anthropometric indices. The average value of WHtR in these children was between 0.41 and 0.44. Based on the results obtained authors proposed that WHtR could be used for detecting cardiovascular risk in children.

Table 3: 2x2 Table for boys at 0.41 and 0.5 waist circumference cut-off values.

	WHtR</= 0.41	WHtR >/= 0.41	Total
Non-hypertensive	150	129	279
Hypertensive	16	26	42
Total	166	155	321

	WHtR</= 0.5	WHtR >/ = 0.5	Total
Non-hypertensive	276	4	280
Hypertensive	40	2	42
Total	316	6	322

Table 4: 2x2 Table for girls at 0.41 and 0.5 waist circumference cut-off values.

	WHtR </= 0.41	WHtR>/= 0.41	Total
Non-hypertensive	148	144	292
Hypertensive	29	46	75
Total	177	190	367

	WHtR</= 0.5	WHtR >/ = 0.5	Total
Non-hypertensive	284	8	292
Hypertensive	69	6	75
Total	353	14	367

Tables 3 and 4 show different frequencies of children in each category of true positive, false positive, false negative and true negatives which were used in the calculation of positive and negative predictive values for WHtR as a predictor of high blood pressure. The results obtained for different diagnostic measures are shown in Table 5. The optimal WHtR cut-off value with the highest sum of sensitivity and specificity values were the same in both boys and girls, namely 0.41. For this reason diagnostic accuracy measures were computed for 0.41 and 0.5 WHtR cut-off values.

Table 5: Diagnostic accuracy measures for WHtR as a predictor of blood pressure for boys and girls at 0.41 and 1.5 WC cut-off values.

Diagnostic accuracy measures	Waist circumference cut-off values			
	Boys		Girls	
	0.41	0.5	0.41	0.5
Sensitivity (%)	61.9	4.80	62.0	7.60
Specificity (%)	53.6	98.6	51.4	93.9
Positive predictive value	0.53	0.99	0.51	0.97
Negative predictive value	0.38	0.95	0.39	0.92
Odds ratio	1.88	3.45	1.63	3.09
95% Confidence interval	(0.97; 3.67)	(0.61; 19.37)	(0.97; 2.74)	(1.04; 9.20)

The level of sensitivity of a WHtR cut-off point of 0.5 was very low for both boys and girls (4.8% and 7.6% respectively). The cut-off value of 0.41 which corresponds to a sensitivity of 61.9% and a specificity of 53.6% in boys seems to be the most appropriate and the same cut-off value was found for girls, but with a sensitivity of 62% and a specificity of 51%. The fact that the cut-off value obtained for both boys and girls is the same concurs with the fact that in adults, there is a known advantage to setting up a unisex cut-off point when using the WHtR as a predictor of cardiovascular disease risk[9]. Hara et al (2002) postulated that since WHtR takes into account children's height, a single cut-off point can likely be set for the ratio, without age and gender difference bias[4]. The observed cut-off value of 0.41 for WHtR from the current study is similar to what was observed for Japanese children[4]. It is possible that this is probably because children included in the two studies had similar anthropometric characteristics, and in particular they were more or less the same height and WC (table 2). On the contrary results for odds ratio, PPV and PPV show that at WHtR cut-off value of 0.5 proved to be a better predictor for high blood pressure in children as shown on table 5. The reason for the conflicting results might be due to the fact that sensitivity and specificity are characteristics of a test and are not affected by the prevalence of the disease. However, although the PPV and NPV give a direct assessment of the usefulness of a test, they are affected by the prevalence of the disease. Bewick and colleagues (2004) stated that, 'the decision to use a diagnostic test depends not only on the diagnostic accuracy measures but also on the ultimate benefit to the patient. The prevalence of

the outcome, which is the pre-test probability, must also be known. Generally, there is a trade-off between sensitivity and specificity, and the practitioner must make a decision based on their relative importance'.

One potential limitation of our study is that the sample of children was from only one out of nine provinces in South Africa. Although the children in this study were from both rural, informal settlements and urban areas the distribution was not even. This makes it difficult to suggest the 0.41 cut-off for South African children and adolescents in general. Therefore a nationally representative sample is required to give a more valid cut-off value. Another limitation might be the fact that blood pressure was measured on one occasion even though there were 10 minute intervals between the readings. In establishing the type of hypertension blood pressure should be measured on three or more separate occasions[15]. However the procedure adopted in this study (vascular unloading technique of Penaz together with Physical criteria of Wesseling) provides reliable, non-evasive and continuous estimates of blood pressure[16]. In conclusion results of the present study indicate that adopting a WHtR cut-off value lower than 0.5 for South African children may enhance sensitivity in identifying children at risk for hypertension. However, a nationally representative cohort study is needed to confirm or determine a precise cut-off value for accurate prediction of the presence of hypertension. It is important to establish the diagnostic accuracy of WHtR and hence justify its use in the paediatric population for predicting high blood pressure. Identification of children with high blood pressure is important in controlling the impact of the condition because it allows for the diagnosis and counselling of persons and facilitates the implementation of both treatment and management strategies.

Acknowledgements

This study was supported in part by the South African Sugar Association grant. Special thanks to Prof. HS Steyn and Dr SM Ellis whose statistical consultation is gratefully acknowledged.

References

1. Hsieh SD, Muto T. The superiority of waist-to-height ratio as an anthropometric index to evaluate clustering of coronary risk factors among non-obese men and women. Prev Med 2005; 40:216 –20.

2. Bosy-Westphal A, Geisler C, Onur S, Korth O, Selberg O, Schrezenmeir J. et al. Value of body fat mass vs. anthropometric obesity indices in the assessment of metabolic risk factors. Int J Obes 2006; 30:475– 83.
3. Savva SC, Tornaritis M, Savva ME, Kourides Y, Panagi A, Silikiotou N. et al. Waist circumference and waist-to-height ratio are better predictors of cardiovascular disease risk factors in children than body mass index. Int J Obes Relat Metab Disord 2000; 24(11):1453-1458.
4. Hara M, Saitou E, Iwata F, Ikada T, Harada K. Waist-to-height ratio is the best predictor of cardiovascular disease risk factors in Japanese schoolchildren. J Atheroscler Thromb 2002; 9:127-132.
5. Kahn HS, Imperatore G, Cheng YJ. A population-based comparison of BMI percentiles and waist-to-height ratio for identifying cardiovascular risk in youth. J Pediatr 2005; 146:482-488.
6. Guntsche, Z., Guntsche, E.M., Saraví, F.D., Gonzalez, L.M., Avellaneda, C.L., Ayub. E. et al. Umbilical waist-to-height ratio and trunk fat mass index (DXA) as markers of central adiposity and insulin resistance in Argentinean C\children with a family history of metabolic syndrome, J Pediatr endocrinol metab 2010; 23(3): 245-256.
7. Ashwell M. Waist to height ratio and the Ashwell shape chart could predict the health risks of obesity in adults and children in all ethnic groups. Nutr Food Science 2005; 35:359-364.
8. Nambiar S, Truby H, Abbott RA, Davies PSW. Validating the waist-to-height ratio and developing centiles for use amongst children and adolescents. Acta Paediatric 2008; 98(1): 148-152.
9. Ashwell M, Lejeune S, McPherson K. Ratio of waist circumference to height may be better indicator of need for weight management. BMJ. 1996; 312:377.
10. Sung RYT, So H-K, Choi K-C, Nelson EAS, Li AM, Yin JAT. et al. Waist circumference and waist-to height ratio of Hong Kong Chinese children. BMC Pub Health 2008; 8:324-338.
11. Kruger R, Kruger HS, Macintyre UE. The determinants of overweight and obesity among 10–15-year-old schoolchildren in the North West Province, South Africa—the

THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) study. Public Health Nutr 2006; 9: 351–358.

12. Mukuddem-Petersen J and Kruger HS. Association between stunting and overweight among 10–15-y-old children in the North West Province of South Africa: the THUSA BANA Study. Int J Obes 2004; 28:842–851.
13. Lohman TG. Anthropometry and body composition. In Lohman TG, Martorell R, eds. Anthropometric standardization manual, 1988. Champaign, IL: Human Kinetics Books.
14. Boileau RA, Lohman TG, Slaughter MH. Exercise and body composition of children and youth. Scand Med Sci Sports, 1985; 7(1): 17-27.
15. Schutte AE, Van Rooyen JM, Kruger HS, Malan NT, De Ridder JH. Dietary risk markers that contribute to the etiology of hypertension in black South African children: the THUSABANA study. J Hum Hypertens 2003; 17:29-35.
16. Silke B, McAuley D. Accuracy and precision of blood pressure determination with the Finapres: an overview using re-sampling statistics. J Hum Hypertens 1998; 12:403-409.
17. The Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents (supplement article): National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents. Pediatrics 2004:114(2):555-576.
18. SAS Institute Inc., 2003. SAS Institute Inc., SAS OnlineDoc, Version 9.1, Cary, NC
19. Campbell MJ, Machin D. Medical Statistics: A common sense approach, 3rd ed. John Wiley and Sons, West Sussex, England; 2000.
20. Bewick V, Cheek L, Ball J. Statistics review 13: Receiver operating characteristics curves. Crit care 2004; 8(6): 508-512.

ADDENDUM 3: PROPOSAL FOR FUNDING OF NEW DATA COLLECTED FOR THIS STUDY.

NORTH-WEST UNIVERSITY
YUNIBESITI YA BOKONE-BOPHIRIMA
NOORDWES-UNIVERSITEIT
POTCHEFSTROOM CAMPUS

WAIST CIRCUMFERENCE PERCENTILES FOR BLACK SOUTH AFRICAN CHILDREN AGED 7-15 YEARS.

Principal investigator:

B. S. Motswagole, Ph D student, North-West University, Potchefstroom

Co-investigators:

Prof HS Kruger, Ph D promoter, North-West University, Potchefstroom

Dr M Faber, Ph D co-promoter, Medical Research Council, Cape Town

Prof C Walsh, University of the Free State

Dr KD Monyeki, Medical Research Council, Cape Town

Abstract

Aim: To develop age and sex specific reference values for waist circumference of black children aged 7-15 using data from four provinces representing all socio-economic groups in South Africa.

Methods: To pool available data and collect new data in order to describe the waist circumference distribution of black South African children by generating percentile curves.

Subjects: Black children aged 7-15 years old selected randomly from a combination of convenience samples, from schools in the North West province, Free State, Limpopo province, Kwazulu-Natal and Northern Cape. The planned sample size will be approximately 4000.

Results: Smoothed 5^{th}, 10^{th}, 25^{th}, 75^{th}, 90^{th} and 95^{th} waist circumference percentiles derived from least median of squares (LMS) regression.

Conclusions: These reference data can be used to identify children with an elevated risk of developing obesity related disorders and can serve as a baseline for future studies of temporal trends on waist circumference.

Background for proposed project

Over the past decades the whole world has been in transition, effectively the world has and is still undergoing profound socio-cultural, economic, environmental changes which are resulting in changes in the health status of populations. A sedentary lifestyle, the switch to a cash based economy, the modernization of living conditions and increasing availability and accessibility of goods and foodstuffs have contributed to this change of health status (Rochette *et al*, 2007). One of the consequences of the changes in the health status is the increase in the prevalence of obesity even in the developing world where malnutrition is still a problem. The most common outcome of chronic malnutrition is reduced growth in children and this has been associated with impaired fat oxidation, a factor that predicts obesity in populations (Hoffman, 2007). The current situation in most developing countries is the coexistence of under and over nutrition (WHO, 2004). This calls for the development and implementation of health programs and services that that will address both these nutritional problems. Several governments have invested in health programs aimed at mitigating and or controlling the consequences of this

phenomenon. The development of effective health prevention and promotion programs requires adequate data on populations to guide development and implementation.

According to the World Health Organization (WHO, 1983, 1986) growth status of children is the best indicator of overall health and nutritional status in a community, especially in the developing areas of the world. This has led to many countries focusing their studies on physical growth in children, particularly on weight and height to establish the prevalence of malnutrition. Body fat distribution in children has been least studied, however evidence is mounting which highlights the importance of central fat deposition as indicated by waist circumference as conveying a high risk of metabolic complications later in life (Lemieux *et al*, 2007). To date there are no global standards for waist circumference for the youth. Percentile curves have been developed by a few developed countries such as UK, USA, Canada and Australia and these have shown that the cut-off values differ between genders, ethnic groups and countries (Kelishadi, 2006).

Like most countries the South African government has a desire to improve the health status of its people. In order to achieve this adequate baseline data on the health and well-being of the population is a necessity. Continuous monitoring of the health and nutrition profile is also needed to provide information on how health indicators vary over distance and time, and detect emerging problems to guide programs and services to meet the health needs of the population. No nationally representative data on waist circumference are available for South African children. Therefore this study is aimed at pooling available and collecting new data and using it to describe the distribution of waist circumference by generating waist circumference percentile curves for South African children. Health professionals could use these curves to identify children who might be at risk of developing metabolic complications later in life and therefore intervene on time. Detection of individuals at high risk during childhood may help to establish healthy lifestyles and prevent the development of obesity before critical periods, e.g. the adiposity rebound and adolescent periods (Dietz, 1994).

Project objectives

- To explore existing data of waist circumference of South African children to compare percentile cut-off points with international data sets.

- To collect additional anthropometric data and add it to already available data and use it to describe waist circumference distribution of South African children.
- To establish waist circumference percentiles for South African children, with the aim to recommend a cut-off point for boys and girls respectively for identifying risk for non-communicable diseases.

Methodology

Existing data sets to be used

The THUSABANA study has data available on nine hundred and sixty (n=960) black children from the North-West province. Additional data are available from Limpopo province (n=918), which were collected by Dr K. D. Monyeki for his Masters thesis, as well as from the Free State (n= 415). Pre-tested questionnaires were used to interview the children regarding the socio-demographic information, including child's age, type of dwelling, toilet and cooking facilities available in the household.

Additional data to be collected

A representative sample of black children aged 7-15 years will be recruited for this study. Subjects will be randomly selected using the school class lists from schools in KwaZulu-Natal and Northern Cape. Permission to undertake the study has been obtained from the Department of Education and the North-West University ethics committee. Written parental consent and assent from the children will be obtained for subjects randomly selected for the study from class lists.

In KwaZulu-Natal four schools were approached to participate. The number of subjects selected from each class was proportional to the size of the class. The following numbers of children participated from the following schools:

Delani	233
KwaCuthshwayo	221
Chief Lokothwayo	249
Bhekokuhle	317

This gave us a total of 1019 children from KwaZulu-Natal primary schools with complete information. Socio demographic data were collected by trained fieldworkers using a brief questionnaire.

Four schools will be selected in the Northern Cape and from each school a total of one hundred and fifty (n=150) children will be selected randomly. This will give a total of six hundred children (n=600).

Anthropometry is the measurement of body size, weight and proportions (Lee & Nieman, 2003). Simple anthropometric measurements and indices such as weight, height, triceps skinfold thickness and waist circumference remain the most commonly used tools for assessing body composition because of their simplicity and low cost (De Onis *et al,* 1996) For this study anthropometric measures include the following:

Height

Height is measured on a floor standing stadiometer fitted with a head board to the nearest 0.1cm. The subject stands barefoot on the base of the stadiometer and wearing light clothing, with heels together, head positioned such that the line of vision is perpendicular to the body and arms hanging freely by the sides. The movable headboard is brought onto the topmost point of the head with sufficient pressure to compress hair and the reading is taken to the nearest 0.1cm (Lohmann *et al,* 1991).

Body weight

Body weight is measured without shoes and with light clothing to the nearest 0.1kg on a digital scale.

Waist circumference

Waist circumference is a perimeter, which provides an estimate of body girth at the level of the abdomen. Large waist circumference reflects high total body fatness and has also been recognized as a good measure of abdominal fat, particularly the most metabolically active intra-abdominal fat in both adults (Seidell *et al,* 1988) and children (Fox *et al,* 1993). It will be

measured by a non-elastic steel tape (Lufkin) midway between the lowest rib and the iliac crest with the subject in a standing position, both feet touching and arms hanging freely(Moreno *et al*, 2002). The examiner will sit by the side of the subject and fit the tape snugly but not so tightly as to compress the underlying soft tissue. The circumference is measured to the nearest 0.1cm at the end on normal expiration (Lohmann *et al*, 1991). It will be measured two times, or three times if the first two measurements differ by more than 1cm and the mean of the closest two measurements will be used in the analysis.

Infrastructure available for the study

The Center of Excellence for Nutrition Research of the North West University-Potchefstroom Campus has been involved in nutrition research for many years. The center has adequate equipment for conducting anthropometric measurements. All staff who will be taking the measurements had extensive experience in this field.

Literature review

Waist circumference as a measure of obesity may be of particular significance given the association between abdominal girth in adults and cardiovascular morbidity (Rudolf *et al*, 2004). Recent studies in adults have indicated that measuring waist circumference seems to be the simplest way to estimate obesity and the risk of cardiovascular disease and in children similar evidence is emerging (Zanolli *et al*, 1996, Hirshler *et al*, 2005). It has been shown that increased mortality risk related to excess body fat is mainly because of abdominal adiposity (Li *et al*, 2006). Excess accumulation of abdominal fat has emerged as an important predictor of metabolic complications and adverse health outcomes. The increasing number of people with obesity and the serious health risks that come with it make understanding its causes and treatment crucial.

Increased adiposity may be accompanied by an increase of fat deposition in the abdominal region. In obese children and adolescents, fat deposition seems to occur in the body's central regions (Moreno *et al*, 1998). In developing countries stunting, which is an outcome of chronic under nutrition, is still a public health problem. However, it has been linked to central fat deposition in children (Hoffman, 2004). It is possible that linkages between stunting and obesity

are biological in origin (Popkin *et al,* 1996). Possible mechanisms for this relationship have been described in the literature. Hoffman (2007) described two mechanisms that might be part of the causal pathway. First, it is possible that an adaptive response of limiting fat oxidation is developed during periods of energy restriction (*in utero* and in early childhood). The result is that during periods of adequate energy, availability of energy will favour fat storage over utilization and the central region would be a highly desirable site for storage because it can be quickly mobilized in times of need. Unfortunately, for many growth retarded persons, this adaptive mechanism may only result in excess fat deposition that promotes chronic disease rather than providing a safety mechanism for famines that never return (Hoffman *et al,* 2007). Another possible mechanism is through the metabolism of cortisol (stress hormone). Cortisol has been associated with increased body fat and central adiposity in a number of clinical studies (Drapeau *et al*, 2003). Central fat depots have an increased number of glucocorticoids receptors; this may result in rapid uptake of lipids in visceral adipose tissue because they increase the activity of lipoprotein lipase (Richard *et al,* 1993). This interaction could certainly translate into increased central fat deposition (Hoffman *et al*, 2007).

Recently, the amount of intra-abdominal fat has been demonstrated to be more significantly related to metabolic derangement induced by obesity, than that of subcutaneous fat (Asamaya *et al,* 1998). It is not known whether the storage of an absolute or relative excess amount of triglycerides in abdominal fat depots is directly responsible for increased disease risk, or whether such deposition is simply associated with other processes that cause risk or both (Klein, 2007). Some hypotheses for this relationship have been described. These hypotheses are however not mutually exclusive and it is possible that other unknown mechanisms are involved. One of the earliest hypotheses for the relationship between excess abdominal fat distribution and metabolic complications suggests that activation of the central nervous –adrenal axis by the environmental stressors causes both preferential deposition of adipose tissue in the trunk and the cardiovascular metabolic disorders associated with that deposition (Bjorntorp, 1997). It has also been suggested that because subcutaneous fat depots have limited ability to store excess energy, this results in an overflow of chemical energy to intra abdominal tissue and ectopic sites. This then causes metabolic dysfunction in organs in that region (Klein, 2007).

Metabolic complications associated with excess central adiposity are dyslipidemia, hypertension, insulin resistance and impaired glucose tolerance. This cluster of metabolic abnormalities that already appears in obese children and adolescents increases the risk of cardiovascular disease (Rodriguez *et al,* 2004). Freedman *et al* (1999) showed a positive relationship between central adiposity as measured by waist circumference and skinfold thickness to lipid and insulin concentrations in children and adolescents. In another study Weiss *et al,* (2003) measured abdominal fat partitioning by nuclear magnetic resonance in two groups of obese children. One group had impaired glucose tolerance while the other group had normal tolerance, both with similar ages, sex distribution and degree of obesity. The group with impaired glucose tolerance had more abdominal visceral fat, less subcutaneous fat and therefore greater visceral to subcutaneous fat ratio than those whose glucose tolerance was normal. The conclusion from these two studies was that, independently of the amount of fat mass intra-abdominal fat accumulation was strongly related to insulin resistance and hyperglycemia in obese adolescents. Other metabolic abnormalities associated with excess adiposity include increased myocellular lipid content (Weiss *et al,* 2003), low High Density lipoprotein HDL)-cholesterol and high Low Density Lipoprotein (LDL)-cholesterol, ApoA1/ApoB and triglycerides plasma levels (Moreno *et al,* 2002). In this regard there is evidence to suggest that adiposity in childhood and adolescence influences adult mortality and morbidity. However, more studies are needed to confirm this. This demonstrates the need to employ assessment strategies that seek to identify children who are prone to excess adiposity to prevent obesity at an early stage.

References

ASAMAYA, K. HAYASHI, K. HAYASHIBE, H. UCHIDA, N. NAKANE, T. KODERA, K. and NAKAZAWA, S. 1998. Relationships between an index of body fat distribution (based on waist and hip circumferences) and stature, and biochemical complications in obese children. *International journal of obesity,* 22:1209-1216, 1 July

BJORNTORP, P. 1997. Body fat distribution, insulin resistance and metabolic disease. *Nutrition,* 13(9):795-803, Sep

DE ONIS, M and HABICHT, JP. 1996. Anthropometric reference data for international use: recommendations from a World Health Organization Expert Committee. *American journal clinical nutrition,* 64:650-658, 3 June

DIETZ, WH. 1994. Critical periods in childhood development of obesity**.** *American journal clinical nutrition,* 59: 955-959

DRAPPEAU, V. THERRIEN, D. RICHARDS, D. and TREMBLY, A. 2003. Is visceral obesity a physiological adaptation to stress? *Panminrva Medica*, 45(3):189-195

FREEDMAN, DS. DIETZ, WH. SRINAVASAN, SR. BERENSON, GS. 1999. The relation of overweight to cardiovascular risk factors among children and adolescents: The Bogalusa heart study. *Pediatrics,*103(6):1175-1182, 17 Dec

HIRSHLER, V. ARANDA, C. CALCAGNO, ML. MACALLINI, G. JADZINKY, M. 2005. Can waist circumference identify children with the metabolic syndrome? *Archives of paediatrics and adolescent medicine,*159(8): 740-744, 23 Feb

HOFFMAN, DJ.2004 Development origins of chronic diseases:Biological mechanisms and the impact on transitional economies. Global Forum for health research. Mexico. http://www.globalforumhealth.org accessed 22/10/08

HOFFMAN, DJ. MARTINS, PA. ROBERTS, SB. and SAWAYA, AL. 2007. Body fat distribution with normal height children from shanty towns of Sao Paulo, Brazil. *The journal of nutrition,* 23(9):640-646, 5 Jun

KELISHADI, R. ARDALAN, G. GHEIRATMAND, R. ADELI, K. DELAVARI, A. and MAJDZADEH, R. 2006. Paediatric metabolic syndrome and associated anthropometric indices: The Caspian Study. *Acta Paediatrica*, 95(12):1625-1634

KLEIN, S. ALLISON, DB. HEYMSFIELD, SB. KELLY, DE. LEIBEL, RL. NONAS, C. and KAHN, R. 2007. Waist circumference and cardio metabolic risk: A consensus statement from shaping America's health: Association for weight management and obesity prevention; NAASO, The Obesity Society; the American Society for Nutrition; and the American Diabetes Association. *Obesity,* 15:1061-1067

LEE, RD and NIEMANN DC. Nutritional Assessment 2003.3rd edition. Boston: McGraw Hill pp164

LEMIEUX, I. POIRIER, P. BERGERON, J. ALMERAS, N. LAMARCHE, B. CANTIN, B. DAGENAIS, GR. DESPRES, J-P. 2007. Hypertriglyceridemic waist: a useful screening phenotype in preventive cardiology. *Canadian Journal of cardiology*, 23(supp b): 23b-31b, Oct

LI, C. FORD, ES. MOKDAD, AH. COOK, S. 2006. Recent trends in waist circumference and waist height ratio among US children and adolescents. *Pediatrics,* 118:e1390-e1398, 5 Jun.

LOHMANN, TG. ROCHE, AF. MARTORELL, R.(Eds). Anthropometric Standardization Reference Manual 1991. Champaign, IL, Human Kinetics Books. Pp 55-70

MORENO, LA. FLETA, J. MUR, L. SARRIA, A. BUENO, M. 1998. Fat distribution in obese and non obese children and adolescents. *Journal of pediatric gastroenterology and nutrition, (*27)2:242-268

MORENO, LA. PINEDA, I. RODRIGUEZ, G. FLETA, J. SARRIA, A. and BUENO, M. 2002. Waist circumference for the screening of the metabolic syndrome in children. *Acta Paediatrica,* 91(12):1307-1312, 6 Aug

POPKIN, BM. RICHARDS, MK. MONTIERO, CA. 1996. Stunting is associated with overweight in children of four nations that are undergoing nutrition transition. *The journal of nutrition,*126(12) 3009-3016, 21 Aug

RICHARD, D, CHAPDELAINE, S. DESHAEIS, Y. PEPIN, MC. and BARDEN, N. 1993. Energy balance and lipid metabolism in transgenic mice bearing an antisense GCR gene construct. *American journal physiology,*34(1) r146-r150

ROCHETTE, L., St LAURENT, D. and PLAZLAC, C. 2007. Socio-demographic portrait: Nunavik Inuit health survey. Quebec, Canada: Library and archives Canada. http://www.inspq.qc.ca/pdf/publications/658_esi_sociodemo.pdf Date of access: 22 July 2008.

RODRIGUEZ, G. MORENO, LA. BLAY, MG. BLAT, VA. GARAGORRI, JM. SARRIA, A. and BUENO, M. 2004. Body composition in adolescents: measurements and metabolic aspects. *International journal of obesity,* 28:S54-S58

RUDOLF, MCJ. GREENWOOD, DC. COLE, TJ. LEVINE, R. SAHOTA, P. WALKER, J. HOLLAND, P. CADE, Y. and TRUSCOTT, J. 2004. Rising obesity and expanding waistlines in school children: a cohort study. *Archives of disease in childhood,* 89(3):235-237

WEISS, R. DUFOUR, S. TAKSALI, SE. TAMBORLANE, WV. PETERSON, KF. and BONADONNA, RC. 2003. Prediabetes in obese youth-a syndrome of impaired glucose intolerance, severe insulin resistance and altered myocellular and abdominal fat partitioning. *Lancet,*362(9388):951-957, 20 Sep

WHO 1983. Measuring change in nutritional status. A guideline for assessing the nutritional impact of supplementary feeding programs for vulnerable groups. Geneva. WHO

WHO 1986. Use and interpretation of anthropometric indicators of nutritional status. Report of a WHO working group. *Bulletin of the World Health Organization,* 64 (6): 925-941

WHO 2004. Developing countries face double burden of disease. *Bulletin of the World Health Organisation, 82(7):556*

ZANOLLI, R. and MORGESE, G. 1996. Waist circumference percentiles. A simple test for atherogenic disease. *Acta Paediatrica*, 85(11):1368-1369, 21 May

Ethics clearance

Ethical approval has been obtained from North West University Office for research support Ethics Committee on 31 July 2008 (project number NWU-0057-08-A1). The application was pre-screened and approved by a committee of three academic staff members of the Faculty of Health Sciences of the North-West University prior to submission to Ethics Committee.

Investigators publications

Kebakile MM, Mpotokwane SM, **Motswagole BS**, Lima de Faria M, Santo P, Domingues, Saraiva C. Attitudes to Sorghum Foods in Botswana. Afripo 2003 Conference Proceedings.

Motswagole BS, Wambete JT, Mpotokwane SM, Leteane T, Tlhong T, Kwape LD. Botswana traditional dishes and their nutritional adequacy in the diet: is fortification needed? Food Africa Working Group Meeting Proceedings, Cameroon 2003.

Co-investigators' publications relevant to this application

Dr M Faber

Smuts CM, **Faber M**, Schoeman SE, Laubscher JA, Oelofse A, Benadé AJS, Dhansay MA. 2008. Socio-demographic profiles and anthropometric status of 0- to 71-month-old children and their caregivers in rural districts of the Eastern Cape and KwaZulu-Natal provinces of South Africa. South African Journal of Clinical Nutrition, 21(3):117-124.

Faber M. 2007. Dietary intake and anthropometric status differ for anaemic and non-anaemic rural South African infants aged 6-12 months. Journal of Health, Population and Nutrition, 25(3):285-293.

Faber M, Kvalsvig JD, Lombard CJ, Benadé AJS. 2005. Effect of a fortified maize-meal porridge on anemia, micronutrient status, and motor development of infants. American Journal of Clinical Nutrition, 82(5):1032-1039.

Faber M, Swanevelder S, Benadé AJS. 2005. Is there an association between the nutritional status of the mother and that of her 2-year-old to 5-year-old child? International Journal of Food Sciences and Nutrition, 56(4):237-244.

MacKeown JM, **Faber M**. 2005. Frequency of consumption of cariogenic food items by 4-month-old to 24-month-old children: comparison between two rural communities in KwaZulu-Natal, South Africa. International Journal of Food Sciences and Nutrition, 56(2):95-103.

Faber M. 2005. Complementary foods consumed by 6-12-month-old rural infants in South Africa are inadequate in micronutrients. Public Health Nutrition, 8(4):373-381.

Prof HS Kruger

Mukuddem-Petersen J, **Kruger HS**. 2004. Association between stunting and overweight among 10-15-y-old children in the North West Province of South Africa: the THUSA BANA Study. International Journal of Obesity, 28:842-851.

Kruger HS, Margetts BM, Vorster HH. 2004. Evidence for relatively greater subcutaneous fat deposition in stunted girls in the North West Province, South Africa, as compared with non-stunted girls. Nutrition: the international journal of applied and basic nutritional sciences, 20(6):564-569.

Kruger HS, Puoane T, Senekal M, van der Merwe M-T. 2005. Obesity in South Africa: Challenges for government and health professionals. Public Health Nutrition, 8(5):491-500.

Kruger R, **Kruger HS**, MacIntyre UE. 2006. The determinants of overweight and obesity among 10- to 15-year-old schoolchildren in the North West Province, South Africa – the THUSA BANA (Transition and Health during Urbanisation of South Africans; BANA, children) study. Public Health Nutrition, 9(3): 351-358.

Mamabolo R, **Kruger HS**, Lennox A, Monyeki MA, Pienaar A, Underhay C, Czlapka-Matyasik M. 2007. Habitual physical activity and body composition of black township adolescents residing in the North West Province, South Africa. Public Health Nutrition. 10:1047-1056.

Nienaber C, Pieters M, **Kruger HS**, Stonehouse (Oosthuizen) W, Vorster HH. 2008. Overfatness, stunting and physical inactivity are determinants of PAI-1_{act}, fibrinogen and TAT in African adolescents. Blood Coagulation and Fibrinolysis, 19:361-368.

Prof C Walsh:

Dannhauser A, **Walsh C,** Nel M. 2007. Nutritional status of disabled schoolchildren in Bloemfontein (2002 - 2003). South African Journal of Clinical Nutrition, 20**:** 6-14.

Dr KD Monyeki:

Monyeki KD, Kemper HC, Makgae PJ. 2008. Relationship between fat patterns, physical fitness and blood pressure of rural South African children: Ellisras Longitudinal Growth and Health Study. Journal of Human Hypertension, 22(5):311-319. .

Monyeki K, Kemper H. 2008. The risk factors for elevated blood pressure and how to address cardiovascular risk factors: a review in paediatric populations. Journal of Human Hypertension, 22(7):450-459.

Monyeki KD. 2008. Sympathoadrenergic and metabolic factors and ambulatory blood pressure changes in childhood obesity. Journal of Human Hypertension, 22(2):73-74.

Makgae PJ, **Monyeki KD**, Brits SJ, Kemper HC, Mashita J. 2007. Somatotype and blood pressure of rural South African children aged 6-13 years: Ellisras longitudinal growth and health study. Annals of Human Biology, 34(2):240-251.

Monyeki MA, Koppes LL, Monyeki KD, Kemper HC, Twisk JW. 2007. Longitudinal relationships between nutritional status, body composition, and physical fitness in rural children of South Africa: The Ellisras longitudinal study. American Journal of Human Biology, 19(4):551-558.

Monyeki KD, Kemper HC, Makgae PJ. 2006. The association of fat patterning with blood pressure in rural South African children: the Ellisras Longitudinal Growth and Health Study. International Journal of Epidemiology, 35(1):114-120.

Capacity development

Taking accurate anthropometric measurements is a skill that requires specific training. The investigators conducting the measurements will be the applicant, a Ph D (Nutrition) student from Botswana and a Masters (Nutrition) student, also from Botswana. They will be trained by the study promoter, Prof HS Kruger, on the procedures to follow when taking measurements using standard methods. This will be an undoubtedly useful skill acquired which can always be used in conducting surveys on the nutritional status of individuals and or populations. Anthropometry is widely used because it is inexpensive and non invasive therefore developing capacity in this area is quite useful.

The applicant and promoter will also develop new skills in statistical analyses, when the data will be analysed and smoothed waist circumference percentiles will be derived from least means squares (LMS) regression.

ADDENDUM 4: SAMPLE OF LETTER TO SCHOOL HEADMASTERS.

NORTH-WEST UNIVERSITY
YUNIBESITI YA BOKONE-BOPHIRIMA
NOORDWES-UNIVERSITEIT
POTCHEFSTROOM CAMPUS

North-West University (Potchefstroom Campus)
School of Physiology, Nutrition & Consumer Science
Private Bag X6001
Potchefstroom
South Africa 2520
4 July 2008

Mr K.A.M. Ndlovu
Kwa-Cutshwayo Primary School
P/Bag PO
Ashwood
3605

RE: WAIST CIRCUMFERENCE PERCENTILES FOR SOUTH AFRICAN CHILDREN AGED 7-15 YEARS

Dear Mr Ndlovu

The School of Physiology, Nutrition and Consumer Science of the North-West University (Potchefstroom Campus) and the Medical Research Council will be conducting a study on children aged 7-15 years and would like to invite your school to participate in the study. The purpose of this study is to develop age and sex specific reference values for waist circumference of children aged 7-15 years in South Africa. The information will be collected from approximately 250 children from each of four different schools in Kwa Zulu Natal.

The following information will be obtained from the children:

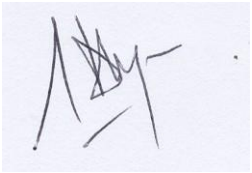
- Information on personal background and socio-economic status
- Measurements of weight, height and waist circumference.

The study will only de done after approval from the Ethics committee of the university has been obtained. The study procedures will be explained to the teachers, children and their parents and informed consent will be obtained from the parents and assent from the children for participation in the study. Participation in this study will be voluntary and will not cost children anything, except time taken to gather the necessary information. We will also need a room or area to conduct interviews and take all the measurements. All the information obtained from children will be kept confidential, the name of the school and those of the children will not be mentioned anywhere. The outcomes of this study will be reported as part of a PhD study, but the individual results will remain anonymous.

Children will not be forced to participate in this study and may withdraw from the study at any time and this will not be held against them.

Should you have any questions please feel free to contact the researcher at the following numbers 018 299 2482 or 018 299 2466 (secretary Sanet Vermeulen).
Thank you for your cooperation.

Sincerely



Prof HS Kruger

ADDENDUM 5: INFORMATION TO STUDY PARTICIPANT.

WAIST CIRCUMFERENCE PERCENTILES: INFORMATION ON THE STUDY AND CONSENT FORM

I CONFIRM THAT:

It has been explained to me, that:

1. The purpose of the research study is to collect information on the waist circumference data among schoolchildren aged 7-15 years in KwaZulu Natal and Northern Cape Provinces of South Africa.

2. I have been told that the researchers will obtain anthropometric variables of a random sample of children aged 7-15 years.

3. Adult fieldworkers from my province will ask me questions about my house and type of work of my parent or caregiver, as well as my growth phase.

4. The participant will be weighed and his/her height as well as waist circumference will be measured without causing any pain to the child. For those measurements boys and girls in separate groups will be asked to undress in privacy of a class-room, because some measurements must be taken with the children dressed in underwear only. The different age groups will be measured separately. The researchers and fieldworkers will work in a professional way, not to embarrass the children.

5. Existing data together with newly collected data will be used to describe waist circumference distribution of South African children. Waist circumference percentiles for South African children will be established, with the aim to recommend a cut-off point for boys and girls respectively for identifying risk for non-communicable diseases, such as high blood pressure. Waist circumference percentiles of South African black children will be compared with waist circumference percentiles of African-American children.

6. I have also been told that this research is being done for the benefit of the children, and that 1600 children will take part in this study.

7. It was also explained to me that the information I will give shall be kept confidential, but that it will be used anonymously for making known the findings to other scientists.

8. It was also clearly explained to me that I can refuse to participate in this research study or I can stop answering the questions at any time during the interview.

The information in this consent form was explained to me by_____(name of interviewer) in_____(language) and I confirm that I have a good command in this language and understood the explanations, OR it was translated to me by_____(Name of translator) in my language_____. I was also given the opportunity to ask questions on things I did not understand clearly.

I the participant (child) hereby agree voluntarily to take part in this research survey.

Signed_____ at_____ on _____2008

Witness_____Parent/legal guardian_____

ADDENDUM 6: SOCIO DEMOGRAPHIC QUESTIONNAIRE

SOUTH AFRICAN BLACK CHILDREN WAIST CIRCUMFERENCE STUDY

Socio-demographic questionnaire

(All information in this questionnaire is confidential).

a. **Interviewer Name:**_____ **Interview Date:** _____

b. **Subject number** _____

c. **Birth Date:**_____

d. **Province:**_____

e. **School:** _____ **Grade** _____

f. Gender	**1** Male	**2** Female				
g. Home language	**1** Zulu	**2** English	**3** Sesotho	**4** Setswana	**5** Xhosa	**6** Other specify
h. Type of dwelling: (*You can tick more than one block if necessary*)	**1** Brick, Concrete	**2** Traditional mud	**3** Tin	**4** Plank, Wood	**5** Other, specify	
i. Number of people living in the your household (*Tick one*)	**1** <4 persons	**2** 4-6 persons	**3** 7-8 persons	**4** >8 persons	**5** Don't know	
j. Where do you get drinking water most of the time (*Tick one*)	**1** Own Tap	**2** Communal Tap	**3** River, Dam	**4** Borehole, Well	**5** Other: Specify	
k. What type of toilet does your household have? (*Tick one*)	**1** Flush	**2** Pit	**3** Bucket, Pot	**4** Ventilated Improved Pit latrine	**5** Other (Specify)	

l. What fuel is used for cooking most of the time in your household? (*You can tick more than one*)	**1** Electric	**2** Gas	**3** Paraffin	**4** Wood/ coal	**5** Sun	**6** Open Fire	**7** Don't know

m. Do you have access to electricity inside your house?	**1**	**2**
	Yes	**No**

n. Does your household have a working:	**1**	**2**	**3**	**4**
1. Refrigerator / Freezer	Fridge	Freezer	Fridge/freezer combination	Don't know

2. Stove	**1** Yes	**2** No	**If yes, choose one** Coal	Paraffin	Gas	Electric	With oven	no oven
3. Washing machine	**1** Yes	**2** No						
4. Microwave	**1** Yes	**2** No						
5. Television	**1** Yes	**2** No						
6. Radio	**1** Yes	**2** No						

ADDENDUM 7: STANDARD OPERATING PROCEDURES FOR ANTHROPOMETRIC MEASUREMENTS.

SOP: Anthropometric measurements

The following anthropometric measurements should be done:

Weight (W)

Height (H)

Waist circumference (WC)

In order to do these measurements you will need with the following equipment:

1.1.1.1 A scale (electronic bathroom scale)

A stadiometer

A steel measuring tape

The scale must be calibrated before-hand with a 2kg calibration weight

If measurements are done in the field, take a wooden board to get an even surface for weighing.

Weight

The scale should be placed on an even, uncarpeted area and levelled with the aid of its in-built spirit level.

- ***After the scale is switched on, wait for the zero indication (0,0), as well as the stable indicator (0 on the display panel) to appear.***
- The child should be weighed (preferably after emptying his/her bladder) and with the minimum of clothing or in underclothes for older children
- The child is placed on the scale, standing still and upright in the middle of the platform, facing the field worker, looking straight ahead. If standing, his/her feet should be flat and slightly apart until the reading is taken.
- After the reading is recorded in the space provided in the questionnaire, the child is removed from the scale. The weight is recorded to the nearest 0.01kg.
- After the child steps down from the scale, wait for the zero reading to appear on the digital display before repeating the procedure.
- The two readings should not vary by more than 10g. If they do, the scale has to be checked for accuracy, and the procedure has to be repeated until two similar weight readings are obtained.

- In extreme cases, when the child is not able to stand alone on the scale, the following method is employed:

  - The mother/caregiver is weighed first (without heavy clothing and shoes). The weighing should be done according to the discussed procedures.
  - Then the zero/reset button is pressed and the field worker has to wait for the zero reading (0.0) to appear on the digital display.
  - The child is then placed in the mother's arms and the reading taken and recorded.
  - The mother and child are then taken off the scale, and when the zero reading appears again on the display, the procedure is repeated.

Height (stature)

The standing height of these children is taken by means of a stadiometer. Two readings are taken and the measurement is repeated if the two readings vary by more than 0.5cm.

- The stadiometer should be wall-mounted or placed on an even, uncarpeted area.
- The subject's shoes and hat or cap are removed
- If the hair is tied up on the top of the head, it should be released
- The subject is positioned as follows:
  - facing the field worker
  - shoulders relaxed, with shoulder blades, buttocks and heels touching the measuring board
  - arms relaxed at sides
  - legs straight and knees together
  - feet flat, heels touching together
  - with the subject looking straight ahead (Frankfurt plane), the headpiece is slid down until it touches the crown of the head
  - the subject should stretch, but the feet should not come off the floor
  - the reading is taken at the end of a deep inward breath
  - the hair should be crushed as much as possible, in order to measure height on the hard flat surface of the head
  - the reading is taken at the bottom of the head piece to the nearest 0.1cm

- the measurement is recorded in the space provided in the questionnaire and repeated at least once.

Waist circumference

For measurement of the waist circumference the child needs to undress, or lift her/his shirt, so that the measurement can be made on the bare skin. Stand in front of the child. The measurement is made at the narrowest point between the lower rib and the hip bone. The fieldworker should ensure that the tape is in the horizontal position for a standing child. The subject is instructed to lower her/his arms to the relaxed position and to breathe normally. The measurement is made at the end of normal expiration. The fieldworker needs to readjust the tape as necessary to ensure that it does not indent the skin. The measurement is taken to the nearest 0.1cm. Take two measurements and record them in the appropriate section of the questionnaire. If the two measurements differ by more than 0.5cm, take a third measurement and select the two measurements that are nearest to each other. (Clean the tape with a wet wipe after one day's measurements.)

References

Lee RD & Nieman DC. 2003. Nutritional assessment. 2nd Mosby. St Louis.

International Society for the Advancement of Kin anthropometry (ISAK). 2001.

International Standards for Anthropometric Assessment. Potchefstroom. ISAK.

WHO. The WHO Child Growth Standards (2005) and the WHO Anthro 2005 software and macros. http://www.who.int/childgrowth/software/en/