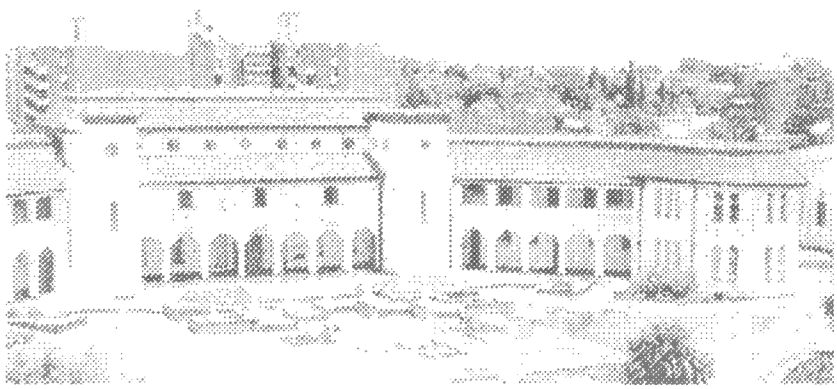
THE ASSOCIATION BETWEEN STUNTING AND OVERWEIGHT AMONG 10 TO 15 YEAR OLD CHILDREN IN THE NORTH WEST PROVINCE:

THE THUSA BANA STUDY

J. Mukuddem-Petersen Hons. B.Sc.

Mini-dissertation submitted in partial fulfilment of the requirements for the degree Magister Scientiae in Dietetics of the Potchefstroomse Universiteit vir Christelike Hoër Onderwys



Supervisor: Dr. H.S. Kruger
Assistant Supervisor: Dr. N.P. Steyn

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I would like to express my sincere gratitude to all the members of the THUSA BANA team for their hard work and professionalism. In particular, my sincere thanks to Prof. J.H. de Ridder and Collette Underhay for the supervision of the anthropometry as well as Dalene and Johan for their assistance in collecting all the anthropometric data, computerisation and quality control of the data. Also, a special word of thanks must go to Mr. T. Bosch and Prof. K.N. de Kock for the linguistic editing.

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Uittreksel

Die verhouding tussen belemmerde groei en oorgewig onder 10-15 jaar oue kinders in die Noordwes Provinsie

Hierdie skripsie ondersoek die verhouding tussen belemmerde groei en oorgewig. Hierdie studie is van besonderse belang vir Suid-Afrika aangesien ons land tans 'n voedingsoorgang beleef (Sawaya *et al.*, 1998).

Die spesifieke doel van die navorsing is om die verhouding tussen belemmerde groei en oorgewig onder 10-15 jarige kinders van die Noordwes Provinsie in Suid-Afrika te ondersoek en sekere voorkomingsmaatreëls voor te stel.

Die ondersoek was deel van die dwarsdeursnit THUSA BANA studie (beteken "Help die Kinders" in Setswana) wat die THUSA projek opgevolg het. Die bogenoemde projek was ontwerp om die probleem van oorgewig en obesiteit onder skoolkinders tussen die ouderdom van 10 en 15 jaar in die Noordwes Provinsie te ondersoek. Die studie was multidisiplinêr van aard en is meegemaak deur voedingkundiges, fisioloë en sielkundiges wat elk op eie gebied data ingewin het. 'n Ewekansige steekproef bestaande uit 10-15 jaar oue skoolkinders (aantal kinders $n = 1257$) is getrek en inligting omtrent die deelnemers is ontleed en ge-rapporteer.

Vraelyste is ontwerp deur gebruik te maak van loodsstudies en veldwerkers is opgelei om die vraelyste wat in die vorm van demografiese en antropometriese data vorms en fisieseaktiwiteitsverslae opgestel is, te voltooi. Die metings het tydens skoolure plaasgevind. Antropometriese veranderlikes wat gemeet is, is trisepsvelvoue, middel bo-armomtrek, abdomenomtrek, liggaamsmassa, liggaamsmassa-indeks en som van die velvoue. Belemmerde groei is beskryf as die lengte benede die vyfde persentiel per ouderdom soos in die National Center for Health Statistics (NCHS)-tabel vervat. Ook die beskrywing van oorgewig en obesiteit gegee in Cole (2000) was gebruik. Die maatstaf van oorgewig en obesiteit het met 'n volwasse LMI van 25 en 30, onderskeidelik, ooreengestem.

Die resultate het bevestig dat 'n klein persentasie (19 %) van die bevolking wat in die studie deelgeneem het belemmerde groei getoon het. Hierteenoor was 7.9 % van die studiebevolking oorgewig en obese. Boonop was 'n selfs kleiner persentasie beide oorgewig en groeibelemmerd of obese en groeibelemmerd. Dus, was die voorkoms van oorgewig en obesiteit met belemmerde groei onder 10-15 jarige skoolkinders statisties baie laag.

In kontras met die situasie in verskeie gebiede in ander ontwikkelende lande, was daar geen dramatiese toename in die voorkoms van belemmerde groei en oorgewig of belemmerde groei en obesiteit onder 10-15 jaar oue kinders in die Noordwes Provinsie van Suid-Afrika nie. Nietemin het ons studie die belangrikheid van die daarstelling van spesifieke intervensies vir groepe wat die risiko loop om oorgewig en groeibelemmerd of swaarlywig en groeibelemmerd te wees, uitgewys. Verdere navorsing moet gedoen word om die meganismes wat betrokke is by die ontwikkeling van hierdie gesondheidsprobleem uit te klaar.

Preface

One of the important contributions made by Potchefstroom University for Christian Higher Education (PU for CHE) was the launching of the THUSA (meaning "Help" in Setswana) project that studied the consequences of urbanisation in the North West Province between 1996-1998. THUSA is also an acronym for "Transition and Health during Urbanisation of South Africans." The THUSA BANA (meaning "Help the Children" in Setswana) project was a follow-up of the aforementioned THUSA project and its main aim was to study the problem of overweight and obesity among schoolchildren aged 10 to 15 years in the North West Province. This particular cross-sectional study was a part of the THUSA BANA project and its main objective was to assess the relationship between height and measures of obesity (BMI, skinfold percentiles, z-scores) in stunted children. A random sample of 10-15 year old schoolchildren (number of children $n = 1257$) was drawn. In addition, trained biokineticists recorded the anthropometric measurements and validated questionnaires were used to measure the demographic and physical activity.

I received excellent guidance from my supervisor, Dr. Salome Kruger of the School of Physiology, Nutrition and Consumer Science at the Potchefstroom University for Christian Higher Education (PUCHE) and my co-supervisor, Dr. N.P. Steyn of the Medical Research Council (MRC) during the completion of this thesis. Furthermore, I would like to thank Prof. J.H. De Ridder the project leader of the THUSA BANA team and team leader of the anthropometry group for his excellent leadership and dedication.

The questionnaires were administered by trained local interviewers. I would like to express my sincere gratitude to all the members of the THUSA BANA team for their hard work and professionalism. In particular, my sincere thanks to Collette, Dalene and Johan for helping in collecting all the anthropometric data, computerisation and quality control of the data. Descriptive statistics were used to ascertain the prevalence of stunting, as well as overweight and obesity in the study population. In this regard, I appreciate the advice on the statistical analysis given by Prof. Faans Steyn of the Statistics Department at the PUCHE.

Obesity in children and adolescents is a prevalent nutritional disorder in developing countries. It is associated with significant physical and psychosocial health problems (Dietz 1986; 1994 ; 1999 ; Frongillo, 1999) such as hypertension, respiratory disease, several orthopedic disorders, diabetes mellitus and elevated serum lipid concentrations (Asayama *et al.*, 1998; Gortmaker *et al.*, 1990). In addition, obesity not only

tracks from infancy into adulthood but is already associated with an unfavourable cardiovascular risk profile in the first two decades of life (Aristimuno *et al.*, 1984; Dipietro *et al.*, 1994). The harmful effects of obesity peak later in adult life and the influence of overweight on the morbidity and mortality of adults is well recognised. Apart from health risks, obesity has been shown to be associated with social and economic disadvantages (Gortmaker *et al.*, 1993; Sawaya *et al.*, 1995). Obesity in childhood is reportedly likely to continue into adulthood (Abraham & Nordsiek, 1960; Bini *et al.*, 2000; Shear *et al.*, 1988). There is reason to believe that weight reduction brings improved health and longevity. Therefore, childhood is a key period to identify and to treat those at risk of becoming obese adults.

Research has suggested that *linear growth retardation* or *stunting* in early life may play a role in promoting adult obesity. In our study, stunted children will be identified as those children having a height-for-age of less than the 5th percentile. Stunting occurs primarily in the first 2 to 3 years of life and is a reflection of the interactive effects of poor energy and nutrient intakes and infection (Walker *et al.*, 2000). Moreover, observations suggest that two and possibly three critical periods exist for the development of obesity and its complications. These include gestation and early infancy, the period of adiposity rebound that occurs between 5 and 7 years of age, and adolescence. Obesity that begins at these periods appears to increase the risk of persistent obesity and its complications. The mechanisms that account for the increased risk associated with obesity at these ages remain unclear (Dietz, 1994). More clarity is needed on the development of obesity in stunted children, so that more appropriate prevention can be developed (Martorell *et al.*, 1987). Futhermore, childhood nutritional stunting, an indicator of chronic under- or malnutrition, has been suggested as one factor contributing to high rates of obesity in developing countries because of the observed association between stunting and adolescent obesity (Must, 1999; Popkin *et al.*, 1996; Waterlow, 1994). In particular, studies in China, Brazil and South Africa (see Sawaya *et al.*, 1995; Sawaya *et al.*, 1998; Waterlow, 1992) have borne this fact out. In addition an association between excess weight gain and dietary fat content in stunted Brazilian children but not in non-stunted control children was evident. A recent study showed that nutritionally stunted children have impaired *fat oxidation* compared with non-stunted control children from the same environment (Hoffman *et al.*, 2000b). This may explain the increased prevalence of obesity among stunted children and short adults in developing countries.

For large-scale screening of obesity, measures based on anthropometry are essential. The main contenders are the **body mass index (BMI)** and selected **skinfolds**. Body mass index also known as Quetelet's Index is determined by calculating weight

divided by height squared and is usually measured in kg/m^2. Skinfold thickness measures fat at various sites of the body, most commonly the triceps skinfold and subscapular by using calipers. Skinfold thickness is cheap and fairly simple to measure, but the need to partially undress may discourage the participation of subjects, resulting in inaccuracy. It is difficult to measure reproducibility, especially in overweight subjects. This results in an increased need for strict quality control, which in turn increases the cost. Currently, the BMI is used widely because of the relative ease and accuracy of the basic measurements (Himes & Dietz, 1994). Body mass index is the best single measure of adiposity in childhood, but there may be a case for collecting skinfold or circumference data as well (Power *et al.*, 1997). Body mass index is more reproducible than skinfolds, but its correlation with body fat is weaker. In addition, to assess body fat distribution two or more skinfolds or possibly body circumferences are required. In terms of acceptability, body mass index has the edge over skinfolds. The tracking and predictive value of childhood BMI in adulthood was assessed recently (Guo & Chumlea, 1999; Wang *et al.*, 2000). These studies showed that overweight at the age of 35 years could be predicted from BMI at younger ages (13-18 years).

The current WHO classification of obesity in children based on overweight-for-height of +2 standard deviations or more above the median National Center for Health Statistics (NCHS) reference curve (50th percentile) is recommended by the WHO to classify obesity in children (WHO, 1998). In an international workshop on childhood obesity, it was concluded that, although BMI is not a perfect measure in children because it covaries with height, it has been validated against measurements of body density (Malina & Katzmaryk, 1999). In addition, BMI may be appropriate to define obesity in children and adolescents as a pragmatic measure and that the same cut-off points of 25 and 30 may be used to identify grade I and grade II overweight, respectively (Bellizzi & Dietz, 1999).

Important studies (Winkleby *et al.*, 1999) have been conducted in the USA and Africa (Hodge & Zimmet, 1994; WHO, 1995) among black females. It has become apparent that there is an increased predisposition towards obesity in African-American females throughout the lifespan due to the increased prevalence of obesity in their childhood counterparts. Some of the factors associated with obesity in African-American populations include environmental factors leading to excessive energy intake, energy expenditure levels too low to offset energy intake, or behavioural factors limiting intentional weight loss (Kumanyika, 1999). These variables should be compared between ethnic groups to derive clues to the causes of obesity. Factors found not to differ significantly between ethnic groups may differ between African-American female subjects

who gain excessive weight over time and those who do not (Kumanyika, 1999). Studies of nutrition in African countries focus mainly on undernutrition. Although limited data are available, obesity has been described in both developing and more developed countries in Africa (Hodge & Zimmet, 1994; WHO, 1995). In South African populations, the prevalence of obesity is 2-3 times higher in black than in white adult women. Currently, there is no conclusive explanation why obesity is so common in black women, although physical activity seem to play a significant role. More information is necessary on the age of development of obesity in black females, as well as the relationship between stunting and measures of adiposity (Dietz, 1999). Consequently, policies and strategies are needed to address the urgent problem of childhood obesity and stunting and its association with malnutrition in South Africa.

The contents of this mini-dissertation will be orally presented on the 6 November 2002 at the Nutrition Congress 2002 at Potchefstroom University for CHE. Furthermore, an article encapsulating the essence of this research has been prepared for submission to the International Journal of Obesity (Abstract included in Addendum F).

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List of Abbreviations

BMI: Body Mass Index

CDC: Centre for Disease Control and Prevention

DNA: Deoxyribose Nucleic Acid

NCD: Non-Communicable Disease

MUAC: Mid-upper Arm Circumference

NCHS: National Centre for Health Statistics

NFCS: National Food Consumption Survey

NGHS: National Heart, Lung and Blood Institute Growth and Health Study Research Group

P: Probability

r: Correlation Coefficient

RMR: Resting Metabolic Rate

RQ: Respiratory Quotient

SD: Standard Deviation

SE: Standard Error

SSF: Subscapular Skinfold

TG: Triglyceride

THUSA: Transition and Health during Urbanisation of South Africans

TSF: Triceps Skinfold

UNICEF: United Nations International Children's Education Fund

WC: Waist Circumference

WHO: World Health Organisation

Chapter 1

Problem Statement and Aim of Study

1.1 Introduction

In the past, there was a clear association between stunting and access to food; the more food that was available the less the incidence of stunting. In recent times, this association may not be as apparent. In particular, a relationship between stunting and obesity has been postulated in countries undergoing nutritional transition (Sawaya *et al.*, 1998). An investigation into this relationship is particularly relevant to South Africa, a country in transition. In this regard, the nutrition transition is a sequence of characteristic changes in dietary patterns and nutrient intakes associated with social, cultural and economic changes during the demographic transition (WHO, 1998). A significant association between stunting and overweight status for children aged 3-6 and 7-9 years in nationally representative surveys in Russia, Brazil, South Africa and a large nationwide survey in China was found (Popkin *et al.*, 1996). In addition, stunting remains a major problem in most low income countries, its prevalence ranges from 11 % in urban African cities to 30-48 % in many African, Asian and Latin American urban and rural areas (Von Braun *et al.*, 1993). Also, a need

has been identified for the launching of appropriate projects to formulate policies and strategies locally so that the urgent problem of childhood obesity and stunting can be combatted.

By studying stone age artefacts of corpulent women found in archaeological excavation sites across Europe, it is evident that obesity was prevalent in the Palaeolithic era more than 25 000 years ago. In addition, clinical evidence of obesity can be dated back as far as Greco-Roman times (WHO, 1997). However, the occurrence of obesity in society at present is unprecedented in the history of mankind. This global problem can be considered to be a result of social, economic and cultural problems being encountered by developing and newly industrialized countries, as well as ethnic minorities and the disadvantaged in developed countries. An escalating rate of obesity (also an increase in NIDDM, hypertension, dyslipidaemia and CVD, coupled with cigarette smoking and alcohol abuse) is a frequent outcome of the modernization or acculturation process (WHO, 1997). The deleterious effects of obesity manifest subsequently in adulthood. However, obesity not only persists from infancy into adulthood, but has been associated with an unfavourable cardiovascular risk profile in the first 20 years of life (Aristimuno *et al.*, 1984; Dipietro *et al.*, 1994). These studies suggest that childhood is a key period to identify those at risk of becoming obese adults. Although the association of childhood obesity with adult obesity has been inadequately investigated, increased body fat among children may be a contributing factor for obesity in adulthood (Musaiger & Gregory, 2000).

Childhood nutritional stunting, usually an indicator of chronic malnutrition (Waterlow, 1994; Must, 1999) and undernutrition (Trowbridge *et al.*, 1987; Waterlow, 1992) has been suggested as a contributory factor to elevated rates of obesity (high weight-for-height) in developing countries. This association between stunting and adolescent and adult obesity has been investigated in several other studies (Sawaya *et al.*, 1995; Sawaya *et al.*, 1998; Popkin *et al.*, 1996). For instance, in studies by Trowbridge *et al.*, (1987) it was found that Mexican-American children, when compared to non-Hispanic whites, are shorter but heavier for their heights. In addition, poor children in rural areas of Mexico and Central America are considerably shorter than Mexican-Americans. Their findings are considered by some to be paradoxical because short stature is generally associated with poverty and chronic undernutrition whereas overweight is associated with overnutrition. These findings have been replicated in China and Russia (Popkin *et al.*, 1996).

1.2 Problem Statement

In recent times it has become apparent that the condition of being overweight co-exists with undernutrition in many developing countries. In this regard, the prevalence of childhood obesity is a serious problem which is further compounded by the lack of sufficient data. In response to this, World Health Organisation (WHO) Reports (WHO, 1997; WHO, 1998) have identified the relationship between body mass index (BMI) and adiposity in children with retarded linear growth (stunted) as a research priority.

An investigation into the relationship or association between stunting and overweight/obesity among 10-15 year old children of the North West Province in South Africa can clarify development of obesity and stunting. An important consequence of such a study would be that more appropriate prevention and intervention strategies could be developed.

1.3 Aim

The specific aim of this research was to investigate the relationship between stunting and overweight among 10-15 year old children of the North West Province in South Africa.

1.4 Hypothesis

The following hypothesis was set for this study:

There is no significant association between stunting and overweight in 10-15 year old children in the North West Province of South Africa.

1.5 Structure of Mini-dissertation

The paradoxical increasing prevalence of stunting associated with obesity is explored in this mini-dissertation.

The first chapter is introductory in character.

Chapter 2 contains a literature review that discusses the literature involving stunting, obesity and the relationship between the two phenomena. In particular, we will review

studies that have been conducted on populations in developing countries in order to clarify the aforementioned relationship and indicate where further research is required.

Chapter 3 gives a detailed explanation of the methodology used in this study.

Results of the study are provided and discussed in Chapter 4. Furthermore, in the second section of this chapter, some standard cut-off points that facilitate comparison with other studies are discussed. In Section 4.3, descriptive statistics related to demographic data, urbanisation, ethnicity, gender, menarche, socioeconomic status and anthropometric status are presented. A interpretation of the data collected in the previous section is given in Section 4.4.

Conclusions are drawn and recommendations made in Chapter 5.

A complete list of references is given in the bibliography and all questionnaires as well as supplementary documentation are provided in the Addendum section of this mini-dissertation.

Chapter 2

The Association between Stunting and Overweight

2.1 Introduction

Scientific research on stunting and obesity and their relationship has developed in several directions in recent times. As a result the literature involving these phenomena is extensive. In this chapter, we review some of the prominent articles that have been written and studies that have been conducted to explain the relationship between stunting and obesity in modern society.

2.2 Stunting

2.2.1 Definition and Overview

Stunting in children is a consequence of many adverse factors, including poor nutritional status of pregnant mothers and children, low birth weight due to inadequate

food intake (Bellamy, 2000), parents' short stature (Amigo *et al.*, 2000), low income, exposure to infection and other diseases, emotional stress (Walker *et al.*, 2000), lack of physical activity and perhaps the stressful physiologic effects of high altitude (Martorell, 1996; Frongillo, 1999).

In 1987, the United Nations sub-committee on nutrition of the World Health Organisation estimated that one-third to two-thirds of children in developing countries may be considered to be of short stature (Scrimshaw, 1993). According to the NFCS data, at a national level in South Africa, stunting remains by far the most common nutritional disorder nearly affecting one out of five children (Steyn *et al.*, 1999). For the purposes of this study, short stature, also known as low length-for-age, growth retardation, or stunting, will be defined as a height less than the 5th percentile of the age- and sex-specific length or height reference population defined by the National Centre for Health Statistics (NCHS) of the US Department of Health and Human Services Centre for Disease Control and Prevention (CDC) (Dibley *et al.*, 1987). Short stature may be an indication of the long-term health and nutritional history of a population and may reflect the normal variation of growth within a population; 5 % of children are expected to fall below the established cut-off that defines this phenomenon. Z-score is a standard deviation score and is defined by WHO (1995) as the "deviation of the value for an individual from the median value of the reference population, divided by the standard deviation from the reference population". Therefore we have

$$Z - score = \frac{(observed\ value) - (median\ reference\ value)}{standard\ deviation\ of\ reference\ population}$$

Low height-for-age is indicative of stunting, which is a result of chronic, long-term dietary inadequacy, reflecting socio-economic deprivation. The WHO (1998) regards a population as being moderately affected if 25-50 % of children under the age of 5 years are stunted, and severely affected if more than 50 % are stunted.

In addition, the United Nations International Children's Education Fund (UNICEF) has estimated that approximately one out of three children younger than five years of age are chronically malnourished (Bellamy, 2000), and thus may be affected by poor linear growth early in life. On a population level, an increased prevalence of shortness (above the expected 5 % level) suggests that the growth of some children in the population is retarded.

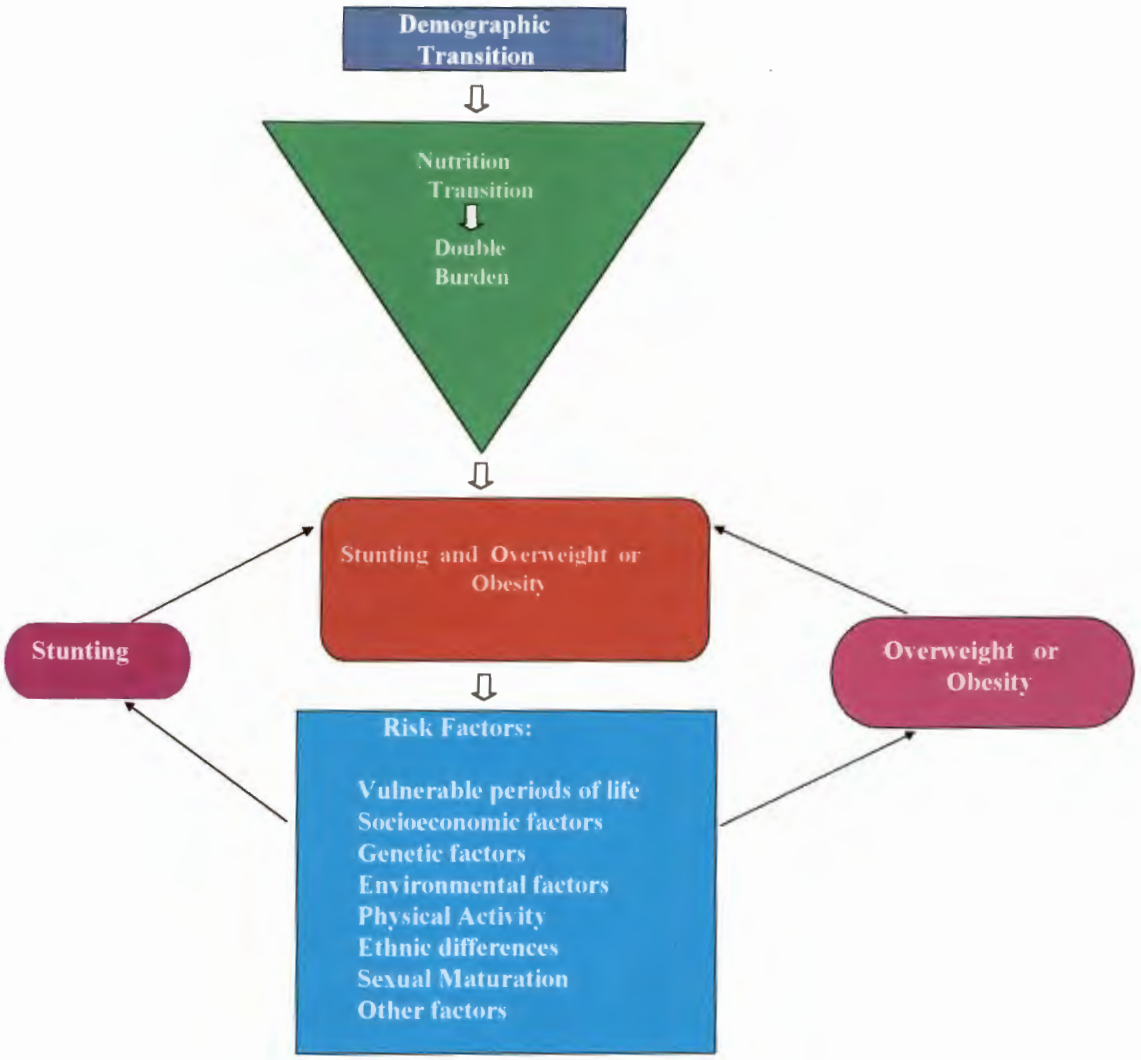


Figure 2.1: Factors associated with stunting and overweight or obesity

2.2.2 Factors influencing Stunting

Vulnerable Periods of Life

Babies of abnormally short length at birth are also at risk for short stature later in life. This indicates that intrauterine growth is important for a child's subsequent growth (WHO, 1995). In addition, studies have shown that among children from low socioeconomic levels, a short period of breastfeeding is associated with a higher risk of complications and or early primary undernutrition, both factors determine growth retardation (Tomkins, 2000; Villalpando & Lopez-Alarcon, 2000).

There are several reasons for stunting being prevalent in the first 2 to 3 years of life and not later. During early childhood nutritional needs are greater, in relation to weight, than at any subsequent stage of life since the rate of growth is the highest that it will ever be. Thus, the opportunity for growth retardation is great in early childhood, partly because more growth is taking place (Martorell *et al.*, 1994). Also, the interactive effects of poor energy and nutrient intakes, infection (Walker *et al.*, 2000) and the child's dependence on others for nutrition early on in life cannot be ignored.

The growth spurt associated with adolescence is a universal phenomenon. However, this spurt differs in intensity and duration from child to child. It usually occurs in boys between 12.5 and 15.5 years while in girls it generally occurs about two years earlier. Before the growth spurt boys and girls only differ by about 2 % in height, whereas this difference increases to about 8 % after the spurt. This increase in height is mainly due to an increase in the growth of the trunk rather than leg growth (Tanner, 1981).

As was stated above, in essence stunting at 13-15 years could be due to low-birth weight and or short length, growth retardation at 2-3 years of age, growth retardation at puberty or combinations of the above mentioned phenomena.

Socio-economic Factors

The prevalence of stunting is high in less-developed countries with low socioeconomic standards. This situation reflects the high frequency of undernutrition observed in such countries. However, the occurrence of stunting can be significantly reduced, but by no means eliminated (Zheng *et al.*, 1989), with improvements in socioeconomic conditions and better and more accessible health care facilities.

Genetic and Environmental Factors

While genetic factors are obviously the major determinant of linear growth potential, reaching this potential is dependent on a number of environmental factors of which nutrition is of great importance (Scrimshaw 1993; Loveridge & Noble, 1994; Waterlow, 1994; Bellamy, 2000). In this regard, the macronutrient components of foods like protein, carbohydrate and fat, which are the sole contributors to energy intake, are the principal determinants of growth rate. Therefore, anthropometric determination of nutritional status is indicative mainly of the availability of protein and energy foods.

Recent studies suggest that micronutrients may also be involved in determining growth rate. In particular, zinc, iron and calcium seem to play a very prominent role. A study done on urban Chinese children to ascertain the effects of zinc and micronutrient repletion on growth, confirmed the essentiality of zinc for the growth of children (Penland *et al.*, 1997). Zinc supplemented infants demonstrated improved linear growth velocity. Marginal and moderate growth impairment in children as a consequence of inadequate zinc intake has been reported from many developed and developing countries. The clinical manifestation of zinc deficiency include growth retardation. In addition, zinc is required for DNA synthesis, cell division and protein synthesis. Several hundred zinc containing nucleoproteins are probably involved in gene expression of various proteins (Prasad, 1996). Furthermore, zinc supplementation is effective for inducing growth in short children with a zinc deficiency (Nakamura *et al.*, 1994).

In a study done on Kenyan primary school children, findings indicated that provision of iron supplements resulted in improved growth (Lawless *et al.*, 1994). A randomized, double-blind, placebo-controlled trial carefully studied the intake of calcium-enriched foods and bone mass growth in pre-pubertal girls. The results suggest a possible positive effect of calcium supplementation on skeletal growth in pre-pubertal girls (Bonjour *et al.*, 1997).

Physical Activity

In the study Torun and Viteri (1994) it was found that mild-to-moderate exercise combined with a good diet enhances linear growth. This may be mediated by endocrine growth factors, of which synthesis is prompted by exercise.

Ethnic Differences

In the USA the high prevalence of short stature among black infants aged less than 12 months (14.8%) probably reflects the relatively high rate of low birthweight among this group (CDC, 1998).

2.2.3 Consequences of Stunting

It is claimed that stunting among children and adults from developing countries induces functional impairment on a wide spectrum of biological, behavioural and social dimensions (Waterlow, 1992). Being shorter than average may not seem to be an important clinical problem or public health issue, but the factors that cause stunting have adverse consequences, including impaired development, lower intelligence, poorer academic performance and a reduced capacity for work in adults. These factors, in turn, have a negative effect on economic productivity. Shorter women are at greater risk for obstetrical complications and there is an intergenerational effect of stunted growth (Frongillo, 1999; Martorell, 1996). Stunting at school age and in adolescents may be less reversible than in younger children (Martorell *et al.*, 1994).

2.3 Overweight/Obesity

2.3.1 Definition and Overview

Overweight is defined as a weight-for-height > 2SDs from the National Centre for Health Statistics/World Health Organization international reference median (De Onis & Blossner, 2000). In addition, Cole *et al.*, (2000) defines internationally acceptable cut-off points for body mass index for overweight and obesity in children. The appropriate cut-off point was defined in body mass index units in young adulthood and extrapolated to childhood, conserving the corresponding centile in each dataset. These cut-off points based on a heterogenous worldwide population can be applied widely to determine whether the children and adolescents they identify are at increased risk of morbidity related to obesity. Table 7.1 (Addendum E) shows international cut-off points for body mass index for overweight and obesity from 9.5-15.5 years (Cole *et al.*, 2000).

Obesity, an emerging public health problem throughout the world, is a condition that results from excessive or abnormal fat accumulation in adipose tissue with the related undesirable positive energy balance, weight gain and possibility of impaired

health status (Garrow, 1988). Obesity in childhood is one of the most complex and least understood clinical syndromes in pediatric medicine practice. However, most studies that examined the persistence of obesity from childhood and adolescence to adulthood found that fatter children were more likely to be overweight later in life (Power *et al.*, 1997). Therefore, the development of overweight in childhood is also related to subsequent overweight or obesity in adulthood (Guo & Chumlea, 1999), where it is associated with an increased risk of morbidity and mortality (Must *et al.*, 1992). However, prevention may be more effective and should occur when adult overweight starts to develop in childhood (Guo *et al.*, 2000).

An increasing prevalence of obesity in many countries, including those that have traditionally had high rates of undernutrition (WHO, 1997; Popkin & Doak, 1998), are being reported. Countries in economic transition from underdeveloped to developed, such as South Africa, China and Brazil, are particularly affected and have an increasing prevalence of obesity across all economic levels and age groups (Popkin & Bisgrove, 1988; Popkin, 1994). In these developing countries, where overweight/obesity co-exist with undernutrition, there is an urgent need to prevent or reverse unhealthy trends in diet and physical activity patterns (WHO, 1998). In this regard, prevention of obesity is a public health priority, where much of the attention should focus on childhood and adolescence (Himes & Dietz, 1994).

2.3.2 Factors of Overweight or Obesity

Genetic predisposition, important cultural features, dramatic correlations with socioeconomic background and emotional factors are some of the factors that influence obesity/overweight. Diet is determined by cultural and socioeconomic background and as such plays a role in the development of obesity.

Vulnerable Periods of Life

Critical or vulnerable periods of development have been identified for many behavioral and developmental processes. In particular, recent studies have shown the importance of nutrition during certain periods of life when an individual may be more vulnerable to the development of future obesity. Obesity occurs during the following critical periods: prenatal period (intra-uterine life), infancy, adiposity rebound (4-7 years), adolescence, early adulthood, pregnancy and menopause (Dietz, 1994). The onset of obesity during these periods appears to increase the risk of persistent obesity and its complications.

Various hypotheses propose that nutritional insults during pregnancy or infancy may have long-term effects on a wide range of metabolic and other physiological relationships (Barker, 1992). Barker and his colleagues have shown that adults with low weight at age 1 year or low birth weights have a greater tendency to store fat abdominally (Barker, 1992). In addition, it was found that males who experienced famine during the first half of gestation were more likely to become obese as young adults. Detection of individuals at high risk during childhood may help to establish healthy lifestyles and prevent the development of obesity before 2 critical periods: the adiposity rebound and adolescence (Dietz, 1994). The mechanisms that account for the increased risk associated with obesity at these ages remain unclear. In this regard, longitudinal studies have to be conducted in order to establish the contribution of each of these periods to the prevalence of obesity and its co-morbidities (Dietz, 1994).

The literature suggests that a series of biological factors, such as the timing of adiposity rebound and parental obesity, which might relate to both behavioural and biological causal mechanisms, affect carryover of fatness from childhood into adulthood (Power *et al.*, 1997; Whitaker *et al.*, 1997; Guo & Chumlea, 1999). In this regard, there is evidence to suggest that adiposity in childhood and adolescence influences adult mortality and morbidity. This needs to be confirmed in other studies. It is also important to clarify whether any links between childhood adiposity and adult disease operate primarily through continuities in adiposity over the lifecourse. Further investigations should, therefore assess disease risks for those remaining fat over long periods and for those gaining weight excessively from childhood and adolescence (Power *et al.*, 1997).

It is important to consider that sexual maturation and age at menarche influence total fatness and regional fat distribution. The timing of growth spurts influence changes in subcutaneous fat accretion (Benefice *et al.*, 2001). In particular, sexual maturation has also been reported as an important factor producing variations of BMI and leads to the opinion that caution is essential when using BMI in adolescence. It was observed that the percentage body fat/BMI relationship was influenced by stage of pubertal development to a greater extent than it was by age (Bini *et al.*, 2000).

Physical Activity

Physical activity is important for achieving proper energy balance, which is needed to prevent or reverse obesity. Regular physical activity can improve body composition and have a positive effect on resting metabolic rate and the maintenance of weight loss. Physical activity may affect the distribution of body fat independently of its effect

on body weight (Tremblay *et al.*, 1990). Properly designed programmes of physical activity may preserve or even increase lean muscle mass during weight loss. Several large, cross-sectional studies in the United States, Canada, and Europe (Tremblay *et al.*, 1990; Seidell *et al.*, 1991) have reported an inverse relationship between levels of physical activity and indirect measures of body fat distribution (e.g. waist-to-hip ratio).

It is difficult to know whether obese individuals are less active because of their obesity or whether a low level of activity caused the obesity (Hoffman *et al.*, 2000b). Results from some studies, however, suggest that low and decreasing levels of activity and a rise in sedentary behaviour are primarily responsible for obesity. For instance, obesity is absent among elite athletes while those athletes who abandon their sport frequently experience an increase in body weight and fatness. This conclusion is further supported by prospective data by Dietz and Gortmaker (1985).

Activity may also decrease in response to social or biological cues. As girls age they may become more interested in appearance and pursue more sedentary social interests, or they may adopt sedentary, adult-modelled behaviour patterns (Stephens *et al.*, 1985).

Parental modelling influences the adoption of physical activity by offspring in childhood and adolescence, continuing after offspring leaves home (Rossow & Rise, 1994), and in adolescents, was predicted by paternal, but not maternal, exercise patterns.

Genetic and Environmental Factors

The role of epidemiological, genetic and molecular factors (WHO, 1997) in weight gain is currently the focus of much research. A series of studies over the last five years or so strongly support the view that many genes are involved in causing a susceptibility to obesity. The evidence accumulated indicates that statistical or experimental support is found for a role of about 70 genes, loci or markers (Perusse *et al.*, 1996). Also, the discovery of leptin has led to a renewed interest in genetic and metabolic influences in the development of obesity. While it is possible that single or multiple gene effects may cause overweight and obesity directly, and indeed this is so in some individuals, this does not appear to be the case in the majority of people. Instead, it is currently considered that the genes involved in weight gain increase susceptibility or risk of an individual to the development of obesity when exposed to an adverse environment (WHO, 1998). Many more years of research will be needed before the important

genes and the critical mutations are finally defined for both excess body fat content and upper body and abdominal fat accumulation.

Garn and colleagues (1959; 1981; 1989) demonstrated that as a result of the interaction between genetic and environmental factors, children whose family members are obese are four times more likely to be obese themselves than children whose family members are lean. By comparison, Locard and colleagues (1994) reported a threefold increase in childhood obesity when a parent is overweight. Moreover, Whitaker *et al.* (1997) also reported that parental obesity increased the risk of childhood obesity by twofold to threefold at all ages. A treatment plan for the parents as well as the child may need to be considered if many family members are obese.

Socio-economic Factors

The rapid increases in obesity rates over recent years have occurred in too short a time for there to have been any significant changes within populations. This suggests that the primary cause of the rapid global rise in obesity lies in environmental and societal changes that are now affecting a large proportion of the world's population (WHO, 1997). The process of modernization and economic transition has seen most countries of the world move towards industrialization and an economy based on trade within a global market. This has brought about a number of improvements to the standard of living and services available to people throughout the world. However, it has also had a number of negative consequences that have directly and indirectly led to deleterious nutritional and physical activity patterns that contribute to the development of obesity. Changing societal structures resulting from this transition have given rise to new problems associated with unemployment, urban crowding, and family community breakdown (WHO, 1997).

In developing countries, as per capita income increases, the nature of the diet in traditional societies tends to change in a pervasive and well-documented manner. In particular, intakes of animal fat and protein increase, vegetable fat and protein decrease and particularly complex carbohydrates decrease and sugar increases. The increase in income may be associated with increased away-from-home consumption of high-fat food items (as in the Phillipines) or with increased consumption of meat (as in China). However, the overall effect tends to be greater intake of total fat and increased prevalence of obesity (WHO, 1998).

Furthermore, Popkin and colleagues showed a strong positive correlation between income and a high weight-for-height in Brazil and also a significant inverse relationship

between undernutrition and income (Popkin *et al.*, 1996).

A review of the literature on the relationship between socioeconomic status (SES: composite index combining income, education, occupation and in some developing countries, place of residence (urban/rural)) and obesity found among women revealed that obesity and SES were consistently inversely related but is positively related in populations of developing countries (WHO, 1998). However among men half of the studies reviewed reported an inverse association, whereas a substantial minority (30 %) reported a direct association. In Thailand, although in transition from an agronomy-based to an industrial-based economy, the obesity-socioeconomic relation is like that of developing countries (Mo-Suwan *et al.*, 1999). However, the individual components of SES may have independent and even antagonistic effects on dietary intake and physical activity patterns so it is often very difficult to make generalizations about the relationship between SES and obesity (WHO, 1998).

Ethnic Differences

Ethnic groups in many industrialized countries appear to be susceptible to the development of obesity and its complications. For the majority of these groups this problem seems to arise from the combination of genetic predisposition, a change from the traditional to a more affluent sedentary lifestyle and its accompanying diet (WHO, 1998).

From data collected by the Pediatric Nutrition Surveillance System (PedNSS) it is evident that nutritional status varies among different race and ethnic groups. Black children have the highest rates of low birth weight and anemia, Hispanic and native American children have the highest rates of overweight; and the Asian children have the highest rate of shortness. There also exists ethnic variation, with girls of Asian or Mexican origin having relatively more subcutaneous fat on the trunk than girls of European or African origin (Benefice *et al.*, 2001). In these populations, fat-rich energy-dense diets are likely to be cheapest, and reduced levels of activity stem from unemployment. Other factors associated with poverty may also be involved. The problem of obesity in ethnic minorities demonstrates the need for targeted prevention intervention strategies (Benefice *et al.*, 2001).

The concept of engaging in physical activity during leisure time is not recognised in many cultures and communities in which energy conservation has historically been a prime concern during times of food shortage. The improvement in food availability has done little to change such attitudes to physical activity. In contrast, the Nordic

countries and some other cultures prize fitness and vitality and thus have a positive attitude to physical activity; they devote considerable amounts of leisure time to vigorous activity at the expense of more sedentary pursuits (WHO, 1998).

Impaired fat oxidation was shown previously to be a risk factor for excess weight gain in several populations known to be susceptible to obesity, as in the case of Pima Indians. In Hispanic and Asian cultures, strenous physical activity may not be considered feminine, or there may be a greater emphasis on academics (Wolf *et al.*, 1993).

The prevalence of obesity is higher among Americans of African heritage than among Americans of European heritage (Troiano *et al.*, 1995; Flegal *et al.*, 1998). Cardiovascular disease mortality is significantly higher in black than in white women. The black mortality rate is between two and four times that of white women, a difference that translates to an excess of 8000 annual cardiovascular disease deaths for black women under 65 years of age. Black women are fatter than white women from their twenties onward both in terms of skinfolds and body mass index, whereas white girls in elementary and junior high school tend to be similar to or fatter than their black counterparts. Because differences in body fat and body mass index between black and white girls appear to occur sometime between childhood and young adulthood, the age of 9 through 10 years was chosen as the entry age range for the National Heart, Lung and Blood Institute Growth and Health Study Research Group (NGHS); this is also the age at which little differences between blacks and whites in prevalence of obesity is expected and that encompasses the pre-pubertal stage of development. Differing rates of maturation may explain some of the observed black-white differences in body mass index, although other factors may also contribute (NGHS, 1992). In this regard, the mid-upper arm circumference measurements of black children should be interpreted cautiously as excess subcutaneous fat may obscure the detection of muscle wasting (Adhikari & Coovadia, 1981). In cross-sectional analysis, black girls are further in the maturation process than white girls at both ages 9 and 10. This earlier onset of maturation may have a primary role in the development of obesity differences between the races (NGHS, 1992). The etiology of the difference in obesity between races remain unknown (NGHS, 1992).

Gender Differences

Studies have indicated that women are more prone to obesity than men. There are a number of physiological processes that are believed to contribute to an increased storage of fat in females. Such fat deposits are believed to be essential in ensuring

female reproductive capacity. Studies in humans and animals indicate that girls exhibit a stronger preference for carbohydrate prior to puberty while boys prefer protein. However, after puberty, both males and females display a marked increase in appetite for fat in response to changes in the gonadal steroid levels. This rise in fat appetite occurs much earlier in females (Lebowitz, 1992).

Females have a tendency to channel extra energy into fat while males utilize more of this energy for protein synthesis. This pattern of energy usage or nutrient partitioning in females contributes to further positive energy balance and fat deposition for two reasons. First, the storage of fat is far more energy-efficient than that of protein, and second, it will lead to a lowering of the lean-to-fat ratio with the result that the resting metabolic rate (RMR) does not increase at the same rate as body mass.

Other Factors

An individual's sensitivity to weight gain may be amplified by certain factors such as the development of a disease, or by therapy with drugs which promote weight gain as a side-effect (WHO, 1998). In addition, genetic, biological and other personal factors such as smoking cessation, gender and age interact to determine an individual's susceptibility to weight gain (WHO, 1997).

2.3.3 Consequences of Overweight/Obesity

Obesity is a complex multi-factorial disorder prevalent in both developing and developed countries that affects children and adults alike. Childhood obesity often becomes a long-term problem. Research shows that this condition in children, particularly during adolescence, persists into adulthood and is associated with an increased risk of many diseases including atherosclerosis, cardiovascular disease, hypertension, cancers, diabetes, respiratory disorders, gall bladder disease, infertility and several non-fatal but debilitating conditions (WHO, 1997). More specifically, hyperlipidaemia, characterized by an increase in the serum triglyceride (TG) level, hyperinsulinaemia and an elevation of serum transaminase level due to fatty liver, are considered to be the three major abnormalities in clinical laboratory data associated with childhood obesity (Asayama *et al.*, 1995). The prevention of obesity in childhood is considered to be the best strategy to control today's 'epidemic'. Strategies to prevent the development of obesity should include the establishment of regular meal patterns, increased physical activity, as well as dietary modifications, to ensure nutrient intakes in the context of a reduced calorie diet (Power & Moynihan, 1998).

Adolescent overweight is associated with increased adult morbidity in men for gout, and in women for arthritis, and reported functional limitation. Among women childhood overweight may be associated with menstrual problems in early adulthood (Lake *et al.*, 1997). Men who were overweight in adolescence were three times more likely to report that they had gout, compared with lean subjects (Must *et al.*, 1992).

Overweight and obesity in childhood and adolescence are associated with significant physical and psychosocial health problems (Dietz, 1999; French *et al.*, 1995). Its consequences are greater than those associated with many other chronic physical health conditions. Discrimination against people who are overweight may account for these results (Gortmaker *et al.*, 1993). As a result of the evidence presented above, it would be prudent to develop appropriate interventions to address weight control among low-income children.

2.4 Stunting and Overweight/Obesity

The relationship between stunting and overweight/obesity was not apparent prior to the shift in incomes and the related changes in diet and activity levels in most low income countries, because stunted children had little opportunity, in terms of economic conditions, lifestyles, and resource availability, to become obese. The emergence of this nutrition transition with its rapid shifts in the composition of diet and activity patterns and subsequent shifts in body composition suggests that this relationship might lead to considerable obesity over the next several decades, affecting individuals living in environments in which current infant feeding and morbidity patterns during infancy are associated with extensive stunting (Popkin, 1994).

Consequently, a high weight-for-height is emerging as a growing nutrition concern among children in communities undergoing nutritional transition (Popkin, 1994; Sawaya *et al.*, 1998). Body composition, especially fat mass, could also be an important component and determinant of long-term outcome of stunting. South Africa has been identified as a country that is undergoing a nutritional transition. The proportion of children stunted among black and coloured South African children is close to 30.6% (Popkin *et al.*, 1996). According to the National Food Consumption Survey (NFCS, 1999) the national level of stunting (H/A $<$ 2SDs) in South Africa remains by far the most common nutritional disorder affecting nearly one out of five children between the ages of 1 and 9 years (Labadarios, 1999).

Research has suggested that undernutrition in early life may play a role in promoting

adult obesity. In particular, studies on three continents showed that nutritional stunting, which is usually caused by chronic undernutrition (Waterlow, 1992), is positively associated with adult fatness (Sawaya *et al.*, 1995; Sawaya *et al.*, 1998). In addition an association between excess weight gain and dietary fat content in stunted Brazilian children, but not in non-stunted control children was observed (Sawaya *et al.*, 1998). African populations, especially preschool-aged children, are exposed to malnutrition, and this may have a major effect on growth and development. Height retardation is regarded as an index of past and chronic malnutrition over a long period (Benefice *et al.*, 2001). This leads to a reduction in final stature, or stunting. In this regard, in the NFCS, stunting decreased with an increase in age. It was lowest in the 7-9 year old group which does reflect catch-up growth with time (Labadarios *et al.*, 1999).

There are several research studies involving communities that exhibit a correlation between stunting and obesity. Many investigators have noted that Mexican-American children, when compared to non-Hispanic whites, are shorter but heavier for their heights. This is a paradoxical situation as was pointed out by Trowbridge *et al.*, (1987) in that short stature is generally associated with poverty and chronic undernutrition whereas overweight is associated with overnutrition. Since many Mexican-Americans are poor, one would expect them to be short and thin, not short and plump. In addition poor children in rural areas of Mexico and Central America are considerably shorter than Mexican-Americans.

It is possible that linkages between stunting and obesity are biological in origin. Barker (1992) suggests that an infant's major adaptation to undernutrition is reduced growth rate and related changes in fetal hormone production that yield long-term effects including changes in insulin and growth hormone. Barker has also found in a series of studies on small samples of English adults with low birth weight was related to subsequent abdominal obesity and a wide range of hormonal changes, associated with syndrome X. In addition, he has shown that growth retardation is associated with later obesity and a range of hormonal changes.

Childhood nutritional stunting is associated with impaired fat oxidation, a factor that predicted obesity in other at risk populations. This finding may help explain increased body fatness and the prevalence of obesity among stunted adults and adolescents in developing countries (Hoffman *et al.*, 2000a). This was evident in another study where nutritionally stunted children had measurably impaired fat oxidation compared with non-stunted control children living in the same environment. Specifically stunted children had significantly higher Respiratory Quotient's (RQ) and lower fat oxidation in the fasting state and 30 minutes after a meal (subsequent postprandial meals

remained higher but not significantly so). This finding of reduced fat oxidation in the fasting and postprandial states may help to explain previous observations (Sawaya *et al.*, 1995) of increased prevalence of overweight among stunted adolescents and adults in developing countries, because fat that is not oxidized must be stored. In addition, it was suggested that the diet early in life has a significant effect on later metabolism and health (Hoffman *et al.*, 2000a).

2.5 Methods of Measuring Body Fat

There are many methods available to determine body fat levels or adiposity. All these methods rely on assumptions and principles that involve the estimation of body fat. As a result, they are classified as being indirect. The most reliable method of body fat determination is by studying cadavers that involves the dissection of components of the human body (Cole *et al.*, 2000).

For large-scale screening of obesity, measures based on anthropometry are essential. The main contenders are the body mass index (BMI in kg/m^2) and selected skinfolds. BMI is more reproducible than skinfolds, but its correlation with body fat is weaker. The body mass index is widely used in adult populations with a cut-off point of 30 kg/m^2 being recognised internationally as a definition of adult obesity (Cole *et al.*, 2000).

Cole *et al.*, (2000) defines internationally acceptable cut-off points for body mass index for overweight and obesity in children. The reference population was obtained by averaging across a heterogenous mix of surveys from different countries, with widely differing prevalence rates for obesity. The appropriate cut-off point was defined in body mass index units in young adulthood and extrapolated to childhood, conserving the corresponding centile in each dataset. These cut-off points based on a heterogenous worldwide population can be applied widely to determine whether the children and adolescents they identify are at increased risk of morbidity related to obesity. Table 7.1 (Addendum E) shows international cut-off points for body mass index for overweight and obesity from 9.5-15.5 years (Cole *et al.*, 2000).

In addition, to assess body fat distribution two or more skinfolds or possibly body circumferences are required in order to improve the accuracy of the measurements. In terms of acceptability, body mass index has the edge over skinfolds. Body mass index is the best single measure, but there may be a case for collecting circumference data as well (Power *et al.*, 1997).

When interpreting BMI data collected from populations with stunted children, we need to be cautious especially in countries undergoing nutrition transition, as the relationship of BMI to adiposity may be altered. Currently, BMI is most commonly used because of the relative ease and accuracy of the basic measurement (Himes & Dietz, 1994). Consequently, it is widely recommended for epidemiological and clinical evaluation of obesity in children and adolescents. Age-dependent reference data for BMI are necessary for children, as changes in body composition occur during growth (Bini *et al.*, 2000).

Skinfold thickness is a more direct indicator of adiposity than BMI. Triceps skinfold (TSF) thickness has been found to be the single best indicator of the percentage of body fat in women and children (Roche *et al.*, 1981; Cronk & Roche, 1982). Despite this, previous studies of both children and adults demonstrated strong correlations between TSF and BMI. The correlation of TSF and BMI is weaker in males, perhaps because in males BMI is altered more by muscularity than it is in females (Must *et al.*, 1991). Arm muscle circumference is regarded as an indicator of body muscle mass, as is arm muscle area (Heymsfield *et al.*, 2000). Weight-for-age is one of the most widely used nutritional parameters (Steyn *et al.*, 1989).

Dietz & Bellizzi, (1999) warned that, until more studies include ethnic groups other than whites, BMI should be used cautiously in assessing fatness across populations (Dietz *et al.*, 1999). Estimates of the prevalence of overweight and obesity in population groups are typically based on body mass index (BMI), and BMI centile curves have been developed for use in paediatric population for clinical and possibly epidemiological purposes (Cole *et al.*, 1995). The study of Cole included all ethnic groups. It has been suggested however, that BMI may be a less sensitive indicator of fatness amongst children (Reilly *et al.*, 2000), and BMI gives no indication about fat distribution. Until recently, waist circumference in children had not been regarded as being an important measure of adiposity. During growth in childhood, body fat is laid down both subcutaneously and intra-abdominally (Brambilla *et al.*, 1994). In future, it could be envisaged that cut-off points similar to those used with BMI centile curves could be implemented, but it is premature at this stage to define these values (McCarthy *et al.*, 2001).

2.6 Summary

There are several possible mechanisms for the relationship between stunting and overweight in children. A number of highly speculative explanations for the relationship

found between stunting and overweight in children stem from the work of Barker, (1992) and none seem to be mutually exclusive. Barker proposes that hormonal changes and their physiologic responses, such as abdominal obesity stem from fetal or infant undernutrition facilitating long-term changes based on metabolic adaptations (Barker, 1992).

Children who are stunted in childhood are likely to have short stature in adulthood (Satyanarayanna *et al.*, 1989) although recent evidence suggests that some catch-up growth may occur (Loveridge & Noble, 1994; Martorell *et al.*, 1994; Waterlow, 1994). It is apparent that the necessary steps of intervention should take place early in life for those at risk of being overweight and stunted (Lloyd *et al.*, 1961). In order to identify the at risk group we should be able to categorize the different forms of overweight and stunting in children. Critical periods of development have been well recognized for many behavioural and developmental processes.

The extensive explorations of Fogel on the ways in which height, independent of weight and BMI relates to morbidity and mortality patterns and trends give more relevance to a biological hypothesis (Fogel, 1994). For example, stunting during the developmental stages has far reaching effects into adulthood by increasing the risk of chronic diseases.

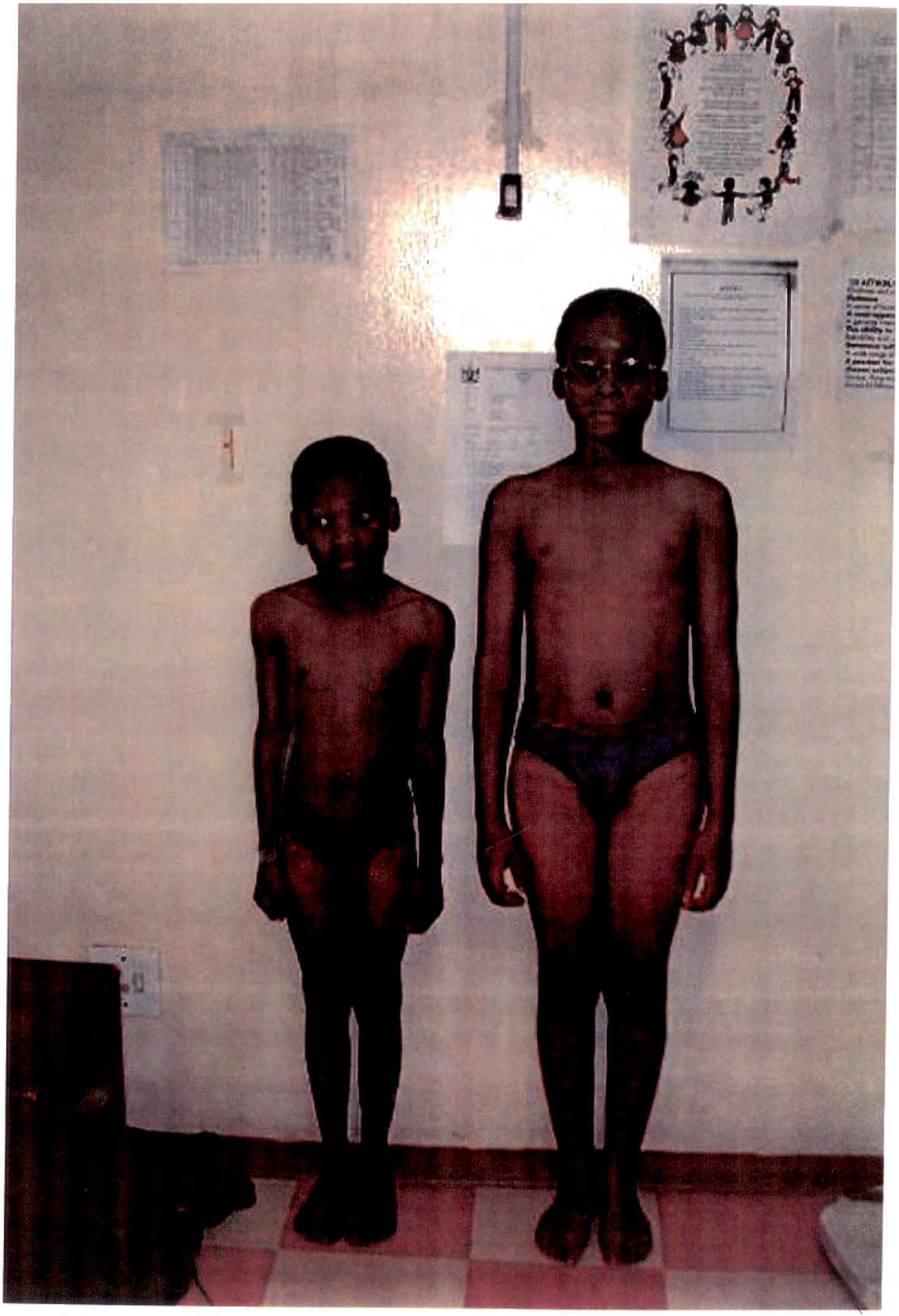
The increasing proportion of fat and the increased energy density of the diet, together with reductions in the level of physical activity and the rise in the level of sedentary behaviour, are thought to be major contributing factors to the rise in the average body weight of populations. Dealing with these issues would appear to be the most effective means of combatting rises in the level of overweight and obesity in the community (WHO, 1997).

In preschool children, undernutrition remains the nutrition problem of greatest concern in developing countries, even though some countries are starting to have worrysome rates of overweight. Therefore, during the early years of life, focus should remain on sustaining proper growth and development. However, the rapid changes in dietary patterns and lifestyles occuring in many developing countries warrant close monitoring of overweight prevalence in children so that preventive measures can be taken in a timely manner (De Onis & Blossner, 2000).

Future efforts to prevent childhood obesity should explore whether parental education programmes can decrease the prevalence of obesity by encouraging more stimulating home environments in young children. Bruch (1975) best summarised the importance

of the family environment approximately a quarter of a century ago: "To understand the obese child, one needs to remember that the child accumulated the extra weight while living in a family that, wittingly or unwittingly, encouraged overeating and inactivity." Three key settings for implementing obesity management support interventions aimed at children can be identified as family-based, school-based and primary-care based programmes (WHO, 1998). Children should be encouraged to increase physical activity by helping to set their own activity goal each week, signing a contract to perform the activity, monitoring performance of the activity and determining self-rewards for reaching the activity goal. Family activity patterns should be reviewed, (Brownell & Stunkard, 1999; Prentice & Jebb, 1995). It was recommended that health promotion programmes emphasize regular, enjoyable physical activity, a healthy diet and acceptance of the normal range of body shapes and that regular monitoring of the anthropometric status of children and adolescents be conducted (Booth *et al.*, 1999). Obesity cannot be prevented or managed solely at the individual level. Communities, governments, the media and the food industry need to work together to modify the environment so that it is less conducive to weight gain. Such partnerships are required to ensure that effective and sustainable changes in the diet and everyday levels of physical activity can be achieved throughout the community. This approach will allow obesity prevention and management strategies to be harmonized with existing public health policies and programmes for control of all non-communicable diseases (NCD's) (WHO, 1998).

To be able to decrease the prevalence of growth deficit in countries with epidemiological profiles similar to that of Chile, it is still important to reduce poverty and its consequences so as to reduce the adverse environmental factors that the children face and to formulate specific interventions for groups at risk, from pregnancy onwards (Amigo *et al.*, 2000).



Chapter 3

Method

3.1 Introduction

The research conducted in this mini-dissertation formed part of the THUSA BANA study. THUSA BANA are SETSWANA words which mean "Help the Children." THUSA is also an acronym for "Transition and Health during Urbanisation of South Africans". THUSA BANA was a multi-disciplinary project which involved various schools within the Faculty of Health Sciences of the Potchefstroom University for Christian Higher Education (PU for CHE). Schools that formed a part of this project included the School for Physiology, Nutrition and Consumer Sciences, the School for Biokinetics, Recreation and Sports Sciences and the School for Psychosocial Behavioural Sciences. Due to the fact that a large amount of data were collected, each school was responsible for the accumulation of the health related data in their specific fields. Only demographic and anthropometric data were included for the purposes of this study.

3.2 Design

A single cross-sectional study design was used. The study formed part of the THUSA BANA project.

3.3 Experimental Procedures

3.3.1 Subjects

There was a 94.1 % response rate. Due to parents not giving consent and children being involved in sports activities, practical classes and being too shy to be measured, we could not realize the planned study population of $n = 1336$. Eventually, the total study population of the THUSA BANA project comprised 1257 subjects. Furthermore, the study population consisted of male and female subjects from ages 10-15 years.

3.3.2 Ethical Considerations

This study was approved by the Ethics Committee of the Potchefstroom University for Christian Higher Education (project number 001M10). Parents or guardians completed informed consent forms (Addendum A) a week before the start of the study to give permission for the subjects to participate in the project. These forms thoroughly explained the experimental procedures according to the design of the THUSA BANA study.

3.3.3 Stratification

Stratum I: Rural

The subjects mainly resided on farms or tribal land. The majority, but not all, of these subjects lived in brick houses with running water and electricity. Some subjects had to fetch water from communal taps or a river and some did not have electricity.

Stratum II: Informal Settlements

These subjects resided in informal settlements (traditional squatter-camps). They were located mainly near towns and cities and the majority of these subjects lived

in zinc houses. Houses were mainly serviced by communal taps and electricity was provided in some cases.

Stratum III: Urban

These subjects resided in established residential areas ("townships") in towns and cities. The majority of these areas had streetlights and the subjects lived in brick houses. Water and electricity were also provided.

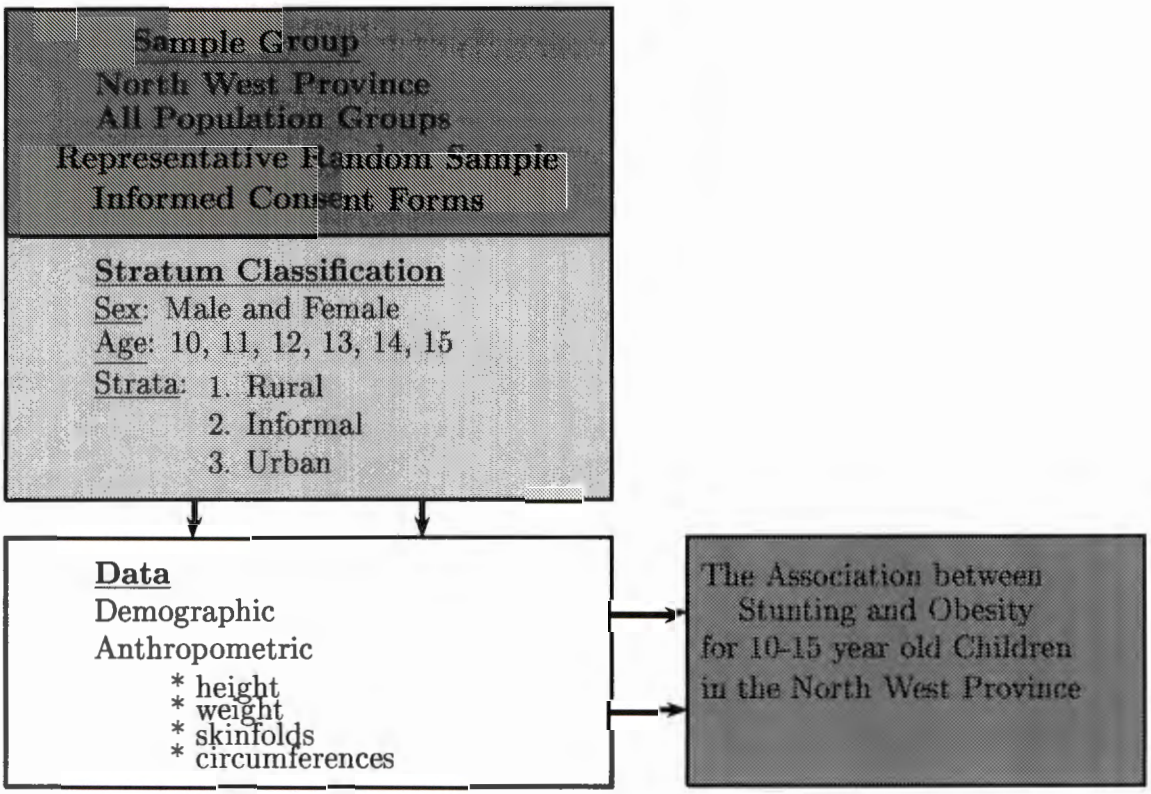


Figure 3.1: Design of the THUSA BANA Study

3.4 Sampling

3.4.1 Methods

The cross-sectional study was done on a randomly selected group of children between 10 and 15 years of age in the North West Province of South Africa. The measurements were done during school hours by the entire group of researchers. The names of the children that formed part of the study were given to the school beforehand so that the children would be ready when the researchers arrived.

3.4.2 Representativeness

The study population was randomly selected with the help of a biostatistician. A list of all the schools ($n = 2520$) was obtained from the North West Department of Education. The random selection of the study population consisted of 44 schools that were selected from 5 strata (5 regions of the province). The study population were further stratified in terms of gender (male/female), type of school (high/primary school), age (10-15 years) as well as ethnic group, with at least 100 children from each age group for boys and girls ($= 6 \times 100 \times 2 = 1200$). The total number of scholars in the province was calculated and accordingly the biostatistician advised that a minimum sample group of 60 children per ethnic group was necessary to make meaningful comparisons. Furthermore, because an equal number of subjects from each age group had to be chosen, the originally planned number of subjects was 1336, consisting of 960 black children, 240 white children, 68 Indian children and 68 coloured children.

As was asserted before, schools were randomly selected from each region. Eventually the study population comprised of 2 high schools and 4 primary schools from predominantly black schools out of every region, one high school and one primary school from predominantly white schools and one high school and two primary schools from predominantly coloured and Indian schools. The latter were only chosen from regions 3 and 4, where mostly Indian and Coloured schools were situated. Subsequently, girls and boys between 10 and 15 years were randomly selected in each school ($n = 1336$). Table 3.1 below provides a summary of the sample population in terms of the number and types of schools and the number, sex and age of the children included in the study.

Table 3.1: Planned Sample Population

No. & Type of School	No. & Sex of Children	Age of Children
10 black high schools	8 boys and 8 girls	14-15 years
20 black primary schools	4 boys and 4 girls	10-13 years
5 white high schools	4 boys and 4 girls	14-15 years
5 white primary schools	4 boys and 4 girls	10-13 years
1 Indian high school	5 boys and 5 girls	14-15 years
2 Indian primary schools	3 boys and 3 girls	10-13 years
1 coloured high school	5 boys and 5 girls	14-15 years
2 coloured primary schools	3 boys and 3 girls	10-13 years

Table 3.2 below gives information about the composition of the planned sample population of the entire THUSA BANA study ($n = 1336$)

Table 3.2: Planned Sample Population of the Entire THUSA BANA Study

	Primary School			High School			TOTAL
Age	10	11	12	13	14	15	
Black	160	160	160	160	160	160	960
White	40	40	40	40	40	40	240
Indian	12	12	12	12	10	10	68
Coloured	12	12	12	12	10	10	68
TOTAL	224	224	224	224	220	220	1336

Not obtaining a 100 % response rate, was due to various reasons like parents not giving consent and children being involved in sports activities, practical classes and being too shy to be measured, therefore, we could not realize the planned study population of $n = 1336$. Table 3.3 below gives information about the composition of the actual sample population of the total THUSA BANA study ($n = 1257$).

Table 3.3: Actual Sample Population of the Entire THUSA BANA Study

Ethnicity	SUBTOTAL
Black	919
White	191
Indian	78
Coloured	69
TOTAL	1257

From Table 3.3, we note that 96 % of the selected black children took part in the study. There was an 80 % response rate for white schoolchildren because of resistance from parents against their participation in the study. Ultimately, more Indian schoolchildren than expected (115 % response rate) participated in the study. Also, more children were included in the total count of coloured children (101.5 % response rate) because they were chosen from a mixed Afrikaans-medium school, together with white children. The total response rate was 94.1 %.

3.4.3 Experimental Procedures

Questionnaires were designed in pilot studies and fieldworkers were trained to administer questionnaires in the form of demographic and anthropometric data forms.

The following stations had an impact on this study:

Station 1: Demographic information

The demographic questionnaire (Addendum C) included questions about the subject's strata of urbanisation, gender, age, language, health status, smoking habits, education level and Socioeconomic status (SES).

Station 2: Anthropometry

The contents of the anthropometric questionnaire (Addendum D) are discussed in Section 3.5 below.

3.5 Anthropometric Protocol

Trained biokineticists and I were responsible for the actual recording of the measurements mentioned below according to the standard methods recommended by Norton & Olds (1996). In this regard, care was taken to calibrate the apparatus used correctly. Quality control methods included the training of fieldworkers, correction of data before and after calculation and duplication of measurements. This was done under the supervision of a Level 3 anthropometrist, Professor Hans De Ridder of the School for Biokinetics at PU for CHE. A control card (Addendum B) was given to each subject and signed at each data collection station and at the end of each day it was used as a measure of control.

3.5.1 The Anatomical Position

Since human body positions differ, we assumed that the description of these positions, directions and relationships conform to the *anatomical position.* In brief, the anatomical position is when the subject is in an upright position, with the arms along the sides and the palms and feet pointing to the front (Ross & Marfell-Jones, 1991).

3.5.2 The Frankfort Plane

When measuring height the head of the subject was held in the *Frankfort plane.* The head is said to be in the Frankfort plane when a horizontal line is drawn from the

orbital of the eye to the trachus of the ear (Ross & Marfell-Jones, 1991). The orbital is the inferior ridge of the eye socket, while the trachus is the indent above the trachion of the ear.

3.5.3 Anatomical Locations

Acromion

The *acromion* is the part of the scapula, or shoulder blade, forming the tip of the shoulder thus giving it its squareness. It projects forward from the scapula, and with the clavicle or collar-bone in front, forms a protecting arch of bone over the shoulder joint.

Radial

The *radial* is the point on the superior-lateral border of the proximal head of the radius which is the outer of the two bones in the forearm.

The aforementioned two landmarks were used to determine the midpoint of the upper-arm which is the place to measure the triceps skinfold thickness (Ross & Marfell-Jones, 1991).

3.6 Variables, Measuring Techniques and Apparatus

In this section, variables that were measured are described. In addition, techniques of measurement and the apparatus that were used to measure these variables are discussed.

3.6.1 Weight

Weight was measured to the nearest 0.1kg with a portable electronic scale (Precision Health Scale, A & D Company, Japan). The subjects were weighed in their underwear. The subjects had to stand in an upright position in the middle of the scale with their weight evenly distributed between their feet. The subjects had to stand still while looking forward with arms relaxed along the sides.

3.6.2 Height

Height was measured by using a portable stadiometer. The aim of this measurement was to obtain the maximal distance between the surface on which the subject was standing and the crown of the head. The subject was barefoot in an upright standing position with heels together, weight evenly distributed between the feet and arms relaxed along the sides. The heels, thighs, top of the back and (if possible) the back of the head had to touch the vertical part of the stadiometer with the head in the Frankfort plane. In a situation where the subject found it difficult to put his/her head against the vertical part of the stadiometer while simultaneously keeping his/her head in the Frankfort plane, preference was given to the Frankfort plane.

The subjects were requested to take a deep breath while stretching up as far as possible without raising their heels from the floor. The subjects' hair was flattened with the use of the head level so that the direct reading on the top of the head could be taken. The reading was measured to the nearest 0.1cm (Ross & Marfell-Jones, 1991).

3.6.3 Circumferences

A flexible "Holtain" steel tape was used to measure circumferences. The metal casing was held in the right hand, while the left hand extended the measuring tape until it was able to be passed to the right hand. The part of the body that was measured was circled with the so called "cross-over hand" method. This entails the left hand crossing over the right hand when putting the tape around the subject. With all the circumference measurements it was imperative that the measuring tape be held in a parallel position. The tape measure was pulled tightly but carefully around the area being measured, in order to ensure that the tape does not cut into the subject's skin. All circumferences were measured to the nearest 0.1cm.

In the following discussion the circumferences that were measured are explained in detail.

Abdomen (Minimum)

The smallest or narrowest observable part of the abdomen was measured. The narrowing is laterally situated midway between the inferior border of the tenth rib and the superior border of the crista of the ileum. In the case where there is no visible narrowing the measurement is taken at the halfway mark between the inferior border of the tenth rib and the superior border of the crista of the ilium. The measurement

was taken at the end of a normal expiration with the arms relaxed along the sides.

Gluteal (Hip)

This circumference measurement was taken around the widest part of the thighs as seen from the side. The subject stood with feet together and with the gluteal muscles not being contracted (Ross & Marfell-Jones, 1991).

3.6.4 Skinfold Thicknesses

The skinfold thicknesses were measured with a Harpenden skinfold caliper held at a constant pressure of 10 g/mm^2. The location where the skinfold thickness had to be measured was clearly demarcated. A double layer of skin together with the subcutaneous fat was firmly pulled up with the thumb and forefinger on the demarcated spot. The skinfold was pulled away from the underlying muscle tissue with the mouth of the caliper placed 1-2 cm under the fingers and at a depth of 1 cm over the skinfold. The trigger of the caliper was released and the skinfold firmly held for the duration of the measurement. Enough time was taken to exert sufficient pressure with the caliper on the skinfold. The reading was taken 2-3 seconds after the caliper had been placed over the skinfold and the needle of the caliper had stabilized. All skinfold measurements were measured to the nearest 0.2 mm.

Triceps skinfold thickness

A vertical skinfold was measured at the midway mark between the acromion and radial position on the posterior surface of the upper-arm.

Subscapular skinfold thickness

The subscapular skinfold was measured directly under the inferior corner of the scapula in a lateral downward direction at an angle of 45^0 degree to the horizontal (Ross & Marfell-Jones, 1991).

3.7 Determining Body Mass Index (BMI)

In the calculations below, height and weight values were used in specific formulas for the determination of the body mass index (BMI).

3.7.1 Body Mass Index (BMI)

The formula for determining BMI was found in Heyward & Stolarczyk (1996) and is given by

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

3.8 Statistical Analysis of the Data

The SAS computer package (StatSoft, Inc'99) for Windows was used to analyse the gathered data. Firstly, the descriptive statistics of the anthropometric variables were reported for different age groups as in Thomas & Nelson (1996).

The hypothesis that there is no significant association between stunting and overweight in 10-15 year old children of the North West Province in South Africa was investigated by using a combination of frequency tables, t-tests, Phi-coefficient tests, Pearson correlational tests, two-way tables and manual Odds Ratio calculations.

Descriptive statistics of stratum, gender, age, ethnicity, language, age at menarche and breadwinner jobs were determined by using frequency tables. Furthermore, additional frequency tests included tables of age by stratum, menarche by age, height by weight, weight by age, weight by stratum, weight by ethnicity, weight by gender. T-tests were used to assess differences between the mean TSF, MUAC, waist circumference, weight, height, BMI and TSF+SSF for girls who had reached menarche and those who had not. In addition, Phi- coefficient tests of age by stratum were performed. Comparisons between stunted and non-stunted subjects were done by using t-tests, standard deviations, minimums and maximums for all the anthropometric variables for age and gender. Pearson correlational tests were done to investigate the correlation between weight and height, BMI and height, TSF+SSF and height and TSF and BMI of girls and boys respectively. In this regard we accepted significance at a $P < 0.05$. In order to determine the association between stunting and overweight manual calculations of the Odds Ratio and the 95 % CI was done based on the formula by Bland and Altman (2000). Furthermore Chi-squared tests from two-way tables of height by weight for all the 10-15 year old girls and boys were done.

Chapter 4

Results and Discussion

4.1 Introduction

In this chapter results pertaining to the association between stunting and overweight among 10 to 15 year old children involved in the THUSA BANA study are reported and discussed. A planned total of 1336 subjects was not realised with the total study population comprising 1257 subjects instead.

Firstly, we comment on the validity of our study and the quality of data collected. In this regard, the response and participation rate in this study was very satisfactory, namely 94.1 %. The most frequent reasons for nonresponse was a lack of parental consent, children being involved in sport activities and practical classes or being too shy to be measured. Anthropometric measurements providing general information on the amount and localization of muscle and adipose tissue (Gibson, 1990) were carefully performed. It is unlikely that misclassifications occurred since interviewers were thoroughly trained in an attempt to maximize the quality of the data and to reduce errors.

4.2 Percentiles for Stature and International cut-off points for BMI

Stunting was described as the height below the 5th percentile for age using the NCHS standard percentiles as presented in Table 7.2 and 7.3 in Addendum E (Roberts & Dallal, 2001).

Furthermore, in this study we complied with the definition of overweight and obesity given in Cole *et al.* (2000). Overweight and obesity corresponded with the adult BMI of 25 and 30, respectively. The reason why these cut-off points were used is that they represent an international standard and therefore comparisons with other countries were possible. The relevant cut-off points are displayed in Table 7.1 in Addendum E.

4.3 Descriptive Statistics

4.3.1 Subjects: Demographic Data

A total of 1257 subjects of which 608 were boys and 649 girls, participated in this study. The main 5 (out of 10) languages spoken by the subjects were Setswana (62.82 %), Afrikaans (20.06 %), English (6.37 %), Xhosa (2.95 %) and Sesotho (1.59 %).

In Tables 4.1 and Table 4.2 below, subjects are categorised according to age, ethnicity, strata of urbanisation, breadwinners, occupation of breadwinner, type of housing, water source and type of stove in use. Most of the subjects lived under poor socioeconomic conditions. This was evident from the type of breadwinner jobs. Very few of the breadwinners had professions or owned businesses but rather had jobs that paid minimal wage or less. In addition, a greater proportion of the subjects had to get their water outside of the home.

Table 4.1: Characteristics of Subjects (n= 1257)

Variable	Number	Percentage
Age		
10-10.99	208	16.55
11-11.99	223	17.74
12-12.99	249	19.81
13-13.99	194	15.43
14-14.99	185	14.72
15-15.99	198	15.75
Ethnic group		
Black	919	73.11
White	191	15.19
Coloured	69	5.49
Indian	78	6.21
Strata of urbanisation		
Rural	450	35.80
Informal Settlement	224	17.82
Urban	583	46.38
Breadwinner		
Father	621	49.48
Mother	244	19.44
Father/Stepfather, Mother	181	14.42
Grandmother	48	3.82
Sister or Brother	35	2.78
Uncle and/Aunt	56	4.46
Mother, Relative	25	2
Grandfather	22	1.75
Father, Relative	9	0.72
Father, Mother, Relative	7	0.56
Grandparents, Relative	5	0.4
Guardian, none	2	0.16

Table 4.2: Characteristics of Subjects (n= 1257) (continued)

Variable	Number	Percentage
Occupation of breadwinner		
Doctor, Nurse, Teacher	146	12.41
Business, Taxi, Self-employed-formal	208	17.69
Typist, Assistant, Office worker	368	31.29
Domestic worker, gardener, contract	395	33.59
Hawker, Carwasher	51	4.34
Unknown, probably informal	8	0.68
Housing		
Hut	6	0.48
Mokuku (zinc house)	247	19.68
Brick House	993	79.12
Other, zinc and brick	9	0.72
Water source		
Fountain, River	16	1.27
Communal tap	246	19.59
Tap on premises	393	31.29
Tap in house	594	47.29
Other: get water from neighbours	7	0.56
Type of stove		
None	16	1.27
Coal or Wood	70	5.58
Gas and paraffin	247	19.68
Wood and paraffin	5	0.40
Electric	888	70.76
Paraffin and electric	14	1.12
Wood and electric	6	0.48
Gas and electric	9	0.72

4.3.2 Urbanisation

Stunting was most prevalent in the rural areas with 51 % of the stunted girls and 43 % of the stunted boys residing there. Furthermore, the highest prevalence of stunting was found in the rural areas (boys: 26.73 %, girls: 23.68 %) followed by informal settlements (boys: 26.42 %, girls: 13.68 %) while the lowest prevalence of stunting was in the urban areas (boys: 17.08 %, girls: 11.63 %) (Figure 4.1).

In Figure 4.2[1] below, it appears that from the entire study population of 1257 subjects, there was a similar percentage distribution of obese, overweight and normal/underweight in the urban and rural subjects.

4.3.3 Ethnicity

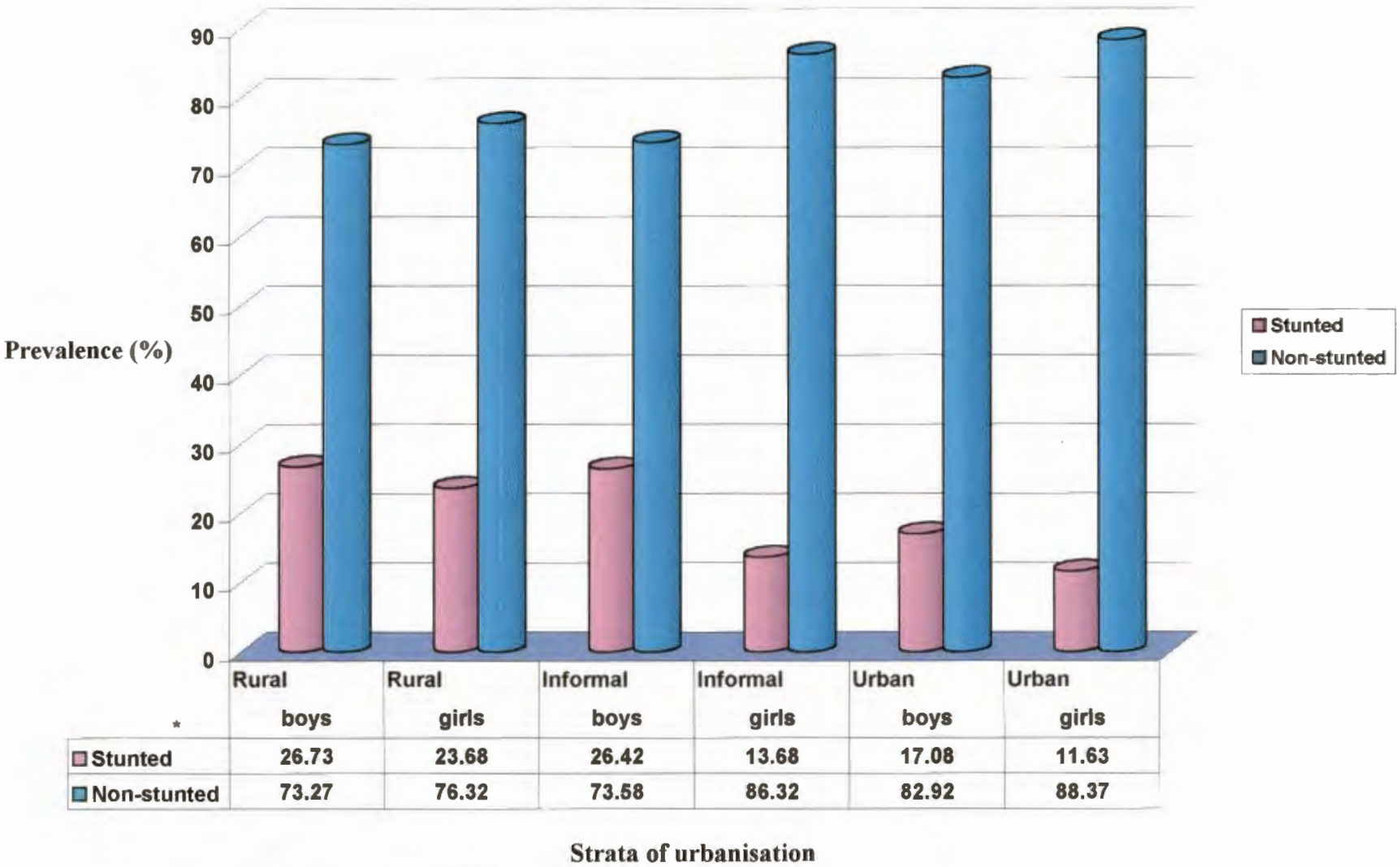
From Figure 4.3[2] it was evident that overweight and obese subjects were mostly found among white subjects (14.1 %) followed by black (7.1 %), Indian (6.4 %) and coloured (2.9 %) subjects.

4.3.4 Gender and Age

In Figure 4.4[3] below, more than twice the percentage of girls were overweight than boys and slightly more girls were obese than boys.

In Figure 4.5[4], the greatest percentage of overweight was evident in the 15 year old subjects and the greatest percentage of obesity was found in 10 year old subjects.

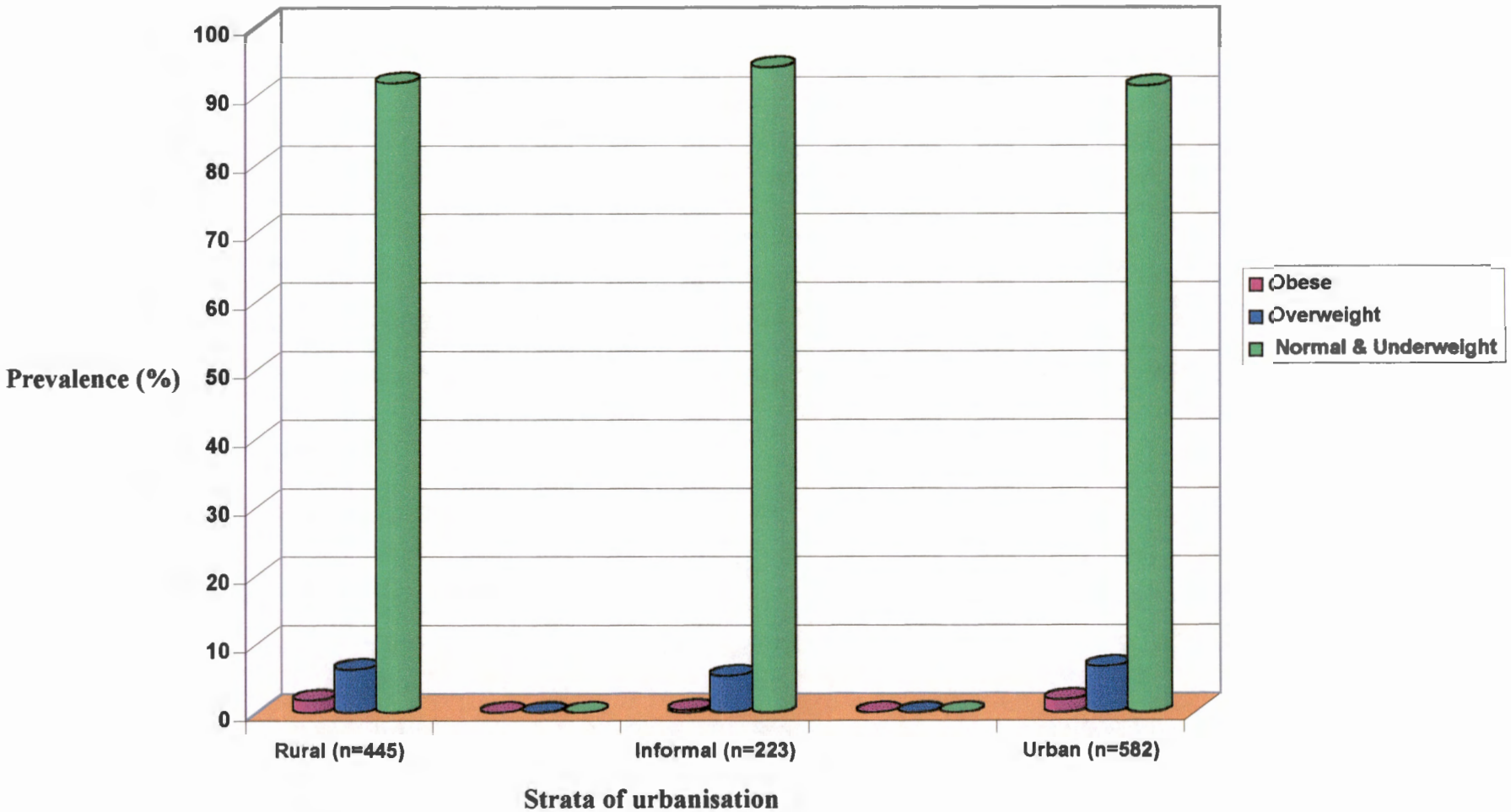
[1]Data were available for 1250 subjects only
[2]Data were available for 1250 subjects only
[3]Data were available for 1250 subjects only
[4]Data were available for 1250 subjects only



*	Rural boys	Rural girls	Informal boys	Informal girls	Urban boys	Urban girls
Stunted	26.73	23.68	26.42	13.68	17.08	11.63
Non-stunted	73.27	76.32	73.58	86.32	82.92	88.37

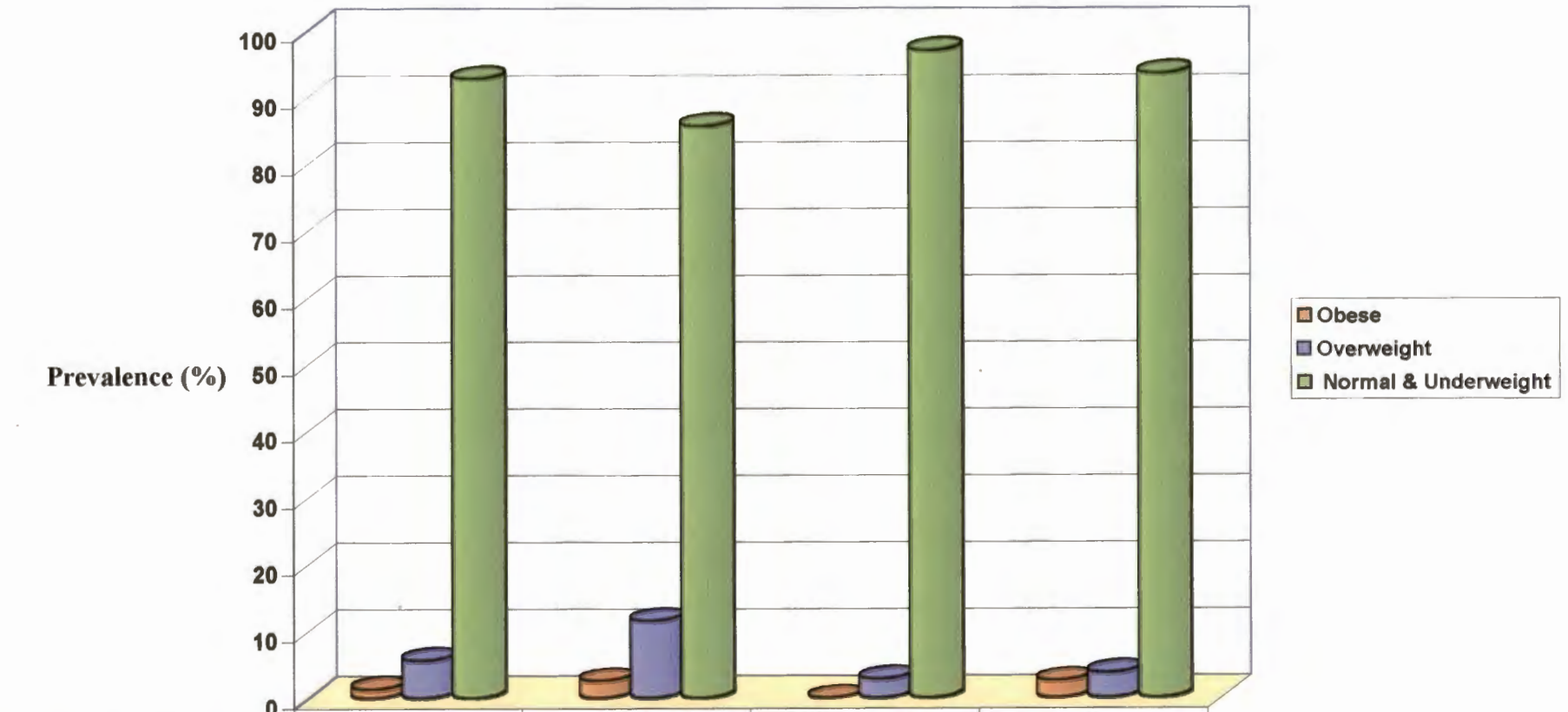
* Height below the 5th percentile for age, NCHS standard

Figure 4.1: Height status of children per stratum of urbanisation



Based on BMI cut-offs according to Cole et al. (2000)

Figure 4.2: Weight status of subjects per stratum of urbanisation (n=1257)

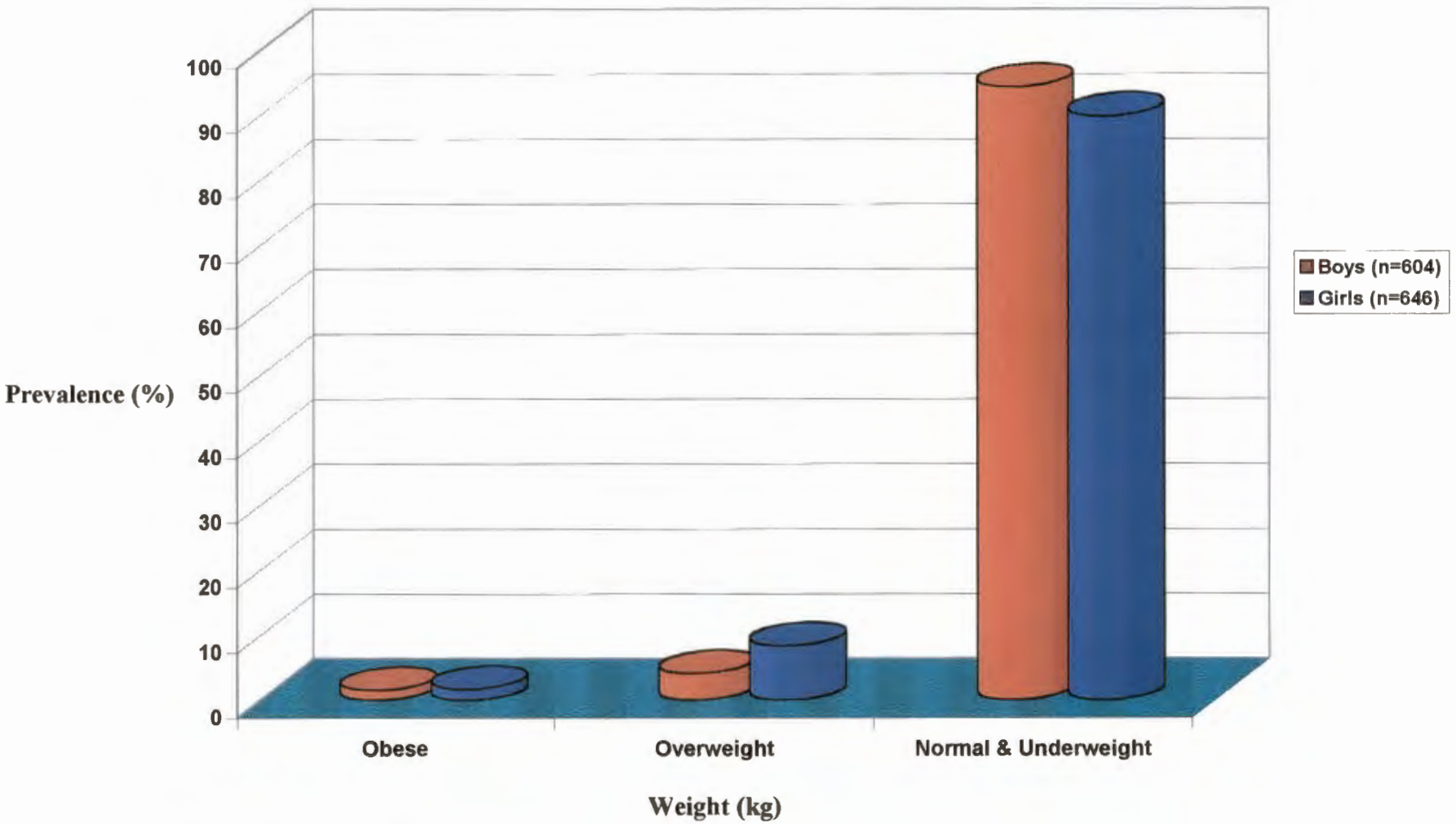


	Black	White	Coloured	Indian
Obese	1.4	2.6	0	2.6
Overweight	5.7	11.6	2.9	3.8
Normal & Underweight	92.9	85.8	97.1	93.6

Ethnic groups

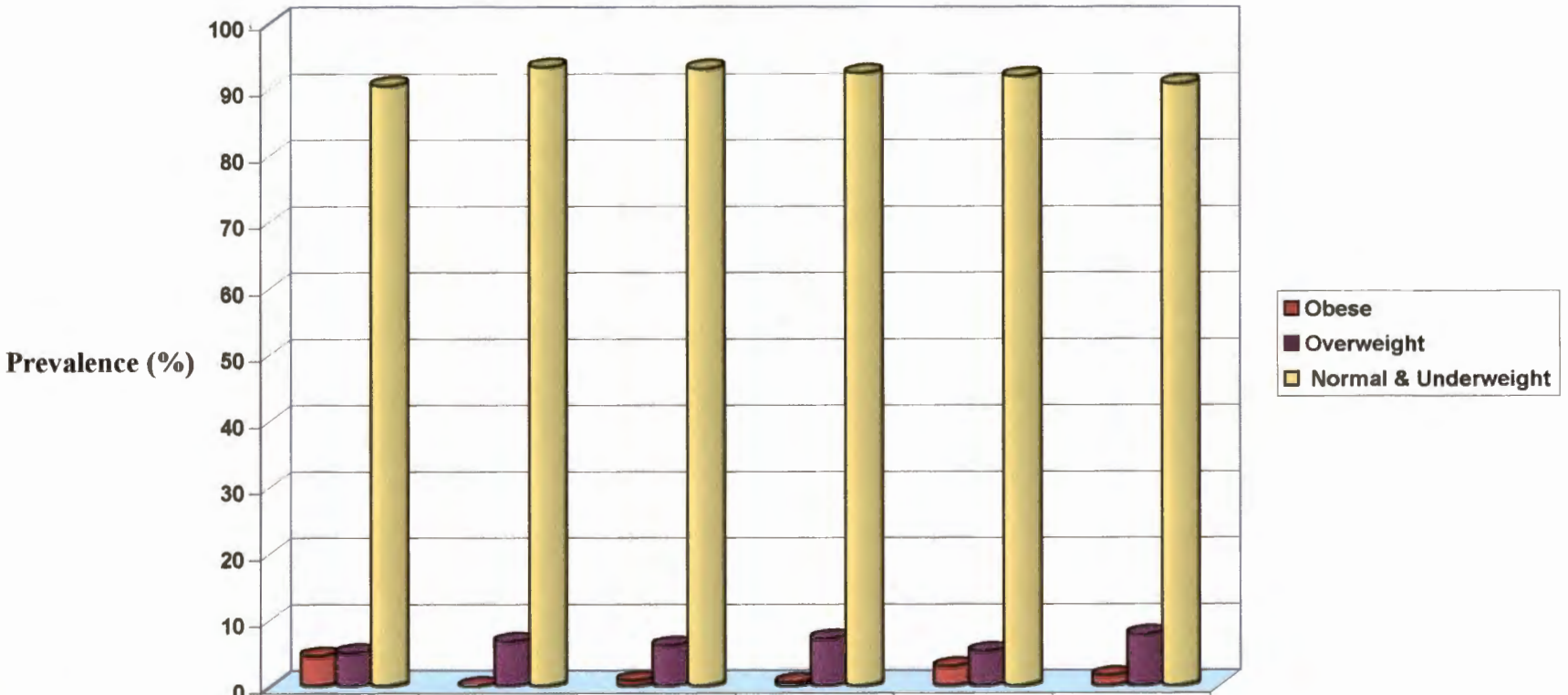
Based on BMI cut-offs according to Cole *et al.* (2000)

Figure 4.3: Weight status of subjects per ethnic group (n=1257)



Based on BMI cut-offs according to Cole *et al.* (2000)

Figure 4.4: Weight distribution of the subjects (n=1257)



	10 (n=200)	11 (n=224)	12 (n=248)	13 (n=198)	14 (n=173)	15 (n=207)
Obese	4.5	0	0.8	0.5	2.9	1.5
Overweight	5	6.7	6.1	7.1	5.2	7.7
Normal & Underweight	90.5	93.3	93.1	92.4	91.9	90.8

Age (years)

Based on BMI cut-offs according to Cole *et al*.(2000)

Figure 4.5: Prevalence of obesity, overweight and normal & underweight per age group (n=1257)

4.3.5 Menarche

Maturation can be assessed through a variety of approaches including skeletal age, appearance of secondary sexual characteristics and menarche. However, the majority of studies of adolescents only report menarche (Martorell *et al.*, 1994).

Similarly, maturation was assessed in this study by recording the age of menarche. Menstruation usually begins a little more than 1 year after peak velocity in height. Menarche therefore, indicates that most of the adolescent growth spurt has been completed (WHO, 1995).

In our study it was evident from Table 4.3[5] that there was a rapid increase in the number of girls reaching menarche at ages 14 and 15 years.

Table 4.3: Age of Menarche of 10-15 year old girls (n=212)

Age of Menarche	Frequency	Percent
10-10.99	4	1.9
11-11.99	5	2.4
12-12.99	18	8.4
13-13.99	37	17.5
14-14.99	60	28.3
15-15.99	88	41.5
TOTAL	212	100

For 14 and 15 year old premenarcheal and postmenarcheal stunted girls t-tests were used to assess differences between means of TSF, MUAC, waist circumference, weight, height BMI and TSF+SSF. All the differences were highly significant with $P < 0.0001$ for all the variables. In this regard, 15 year old stunted postmenarcheal girls had the highest values.

4.3.6 Socioeconomic Status

Most of the subjects came from poor socioeconomic conditions. With the most prevalent (31.29 %) occupation for breadwinners being domestics, gardeners and contract workers. Only 30.1 % of the breadwinners had professional jobs or a business (Table

[5]212 girls had started menstruating at the time of the study

4.2). The rest of the breadwinners had jobs that paid minimal wage or less. In addition, 52.71 % of the subjects had to fetch their water from outside the home, which also indicates poor socioeconomic circumstances.

4.3.7 Anthropometrical Status

In this subsection, we examined the anthropometric variables that were associated with 10-15 year old boys and girls that were stunted or non-stunted.

In particular, the anthropometric variables investigated were the triceps skinfold (TSF), mid-upper arm circumference (MUAC), waist circumference, weight, height, BMI and sum of skinfold thicknesses which includes TSF + subscapular skinfold (SSF) (Tables 4.4 to 4.7). The differences in anthropometric indices between stunting and non-stunting in 10-15 year old boys are presented in Table 4.8. Similarly, differences in anthropometric indices between stunting and non-stunting in 10-15 year old girls are presented in Table 4.9.

TSF

Tables 4.4 to 4.7 provide an illustration of the most important aspects of our study that are related to the anthropometric variable TSF. There was a smaller mean TSF of 15 year old stunted boys than expected. Similarly, there was a smaller mean TSF in 13 year old stunted girls than expected. The mean TSF for the 15 year old stunted girls was greater than the mean TSF for the non-stunted girls (Table 4.9)

The effect size[6] gave an indication of the size of the difference when comparing the means of two variables. From Table 4.8 and Table 4.9 it was evident that the effect size for the difference in TSF was medium for 10 and 11 year old boys and 10 to 12 year old girls. However, the effect size for the difference in TSF was small for 12 to 15 year old boys and 14 and 15 year old girls. Furthermore, the effect size for the difference in TSF of all the boys was small and medium for all the girls.

MUAC

MUAC is considered to be one of the best indicators of total body muscle mass (Steyn *et al.*, 1989). There was a gradual increase in MUAC in both stunted and non-stunted boys and girls (Tables 4.4 and 4.7) with age.

[6]Guideline values according to Cohen, (1977): -small effect size: d=0.2; medium effect size: d=0.5;large effect size: d=0.8

In Tables 4.8 and 4.9 it was evident that the effect size for the difference in MUAC was medium for 10, 12 and 13 year old boys. However, the effect size for the difference in MUAC was small for 14 to 15 year old girls. In addition, 11, 14 and 15 year old boys and 10, 11, 12 and 13 year old girls had a large effect size. Furthermore, the effect size for the difference in MUAC of all subjects was medium.

Waist Circumference

It is clear from Table 4.4 that stunted boys' mean waist circumference (WC) increased gradually with age but a slightly smaller mean was evident at 14 years of age. Contrary to this the stunted girls (Table 4.5) mean waist circumference gradually increased up to age 13 and then there was a sudden increase at age 14 and 15.

From Tables 4.8 and 4.9 it was evident that the effect size for the difference in waist circumference was medium for 12 to 15 year old boys and 10 year old girls. However, the effect size for the difference in waist circumference was small for 14 to 15 year old girls. Also, 10 and 11 year old boys and 11, 12 and 13 year old girls had a large effect size. Furthermore, the effect size for the difference in waist circumference of all subjects was medium.

Weight

A gradual increase in weight with age in all the non-stunted subjects was evident (Tables 4.6 and 4.7). Furthermore, there was a gradual increase in weight with age in the stunted boys, but the stunted girls display a rapid increase in weight at ages 14 and 15 (Tables 4.4 and 4.5). This coincided with the increase in the incidence of menarche, in other words, it coincided with puberty (Table 4.3). In addition, all the girls' mean weights were more than those for the boys' of corresponding ages except for the 10 year old stunted girls and 15 year old non-stunted girls.

In Tables 4.8 and 4.9 the effect size for weight of all subjects was large. The greatest effect size for weight of 1.4 was recorded for the 15 year old boys and 1.2 for the 12 year old girls.

Height

In this study there were 134 stunted boys and 105 stunted girls. As a result we deduced that 19 % of the 10-15 year old children of the North West Province were stunted due to the fact that the study population was representative.

In Tables 4.8 and 4.9 the effect size for the difference in height between stunted and non-stunted subjects was large. The greatest effect size for the difference in height of 2.3 was for the 14 and 15 year old boys and 2.8 for the 13 year old girls.

Figures 4.6 and 4.7 below provide an illustration of an important aspect of our study related to the anthropometric variable height. As expected there was a gradual increase in height with age in the all the subjects. A strong ($r = 0.704$) positive significant correlation ($p < 0.0001$) between weight and height was found in boys when calculating the partial (Pearson) correlation coefficient adjusted for age. Similarly, a strong ($r = 0.621$) positive and significant correlation ($p < 0.0001$) between weight and height was found in girls when doing the same statistical analysis as before.

BMI

Figure 4.8 below provides an illustration of some of the most important aspects of our study that were related to the anthropometric variable BMI of girls.

In Tables 4.8 and 4.9 the mean BMI for the total group of stunted boys and girls was 16.21 kg/m^2 and 17.00 kg/m^2, respectively, while the mean BMI for the total group of non-stunted boys and girls was 17.17 kg/m^2 and 18.22 kg/m^2, respectively.

The 10-12 year old boys had similar mean BMIs (Tables 4.4 and 4.6). However, there was a sudden increase in the BMI after the age of 13 years. There appeared to be a low prevalence of overweight for both groups of stunted and non-stunted boys.

BMI for the girls increased with age and exhibited a sudden increase at 14 and 15 years (Tables 4.5 and 4.7). In 14 and 15 year old stunted girls the mean BMIs were 19.42 kg/m^2 and 21.21 kg/m^2, respectively. Similarly in non-stunted 14 and 15 year old girls it was 19.95 kg/m^2 and 20.32 kg/m^2, respectively. However, the mean BMI of the 15 year old stunted girls exceeded the mean BMI of the 15 year old non-stunted girls (Figure 4.8).

From Tables 4.8 and 4.9 it was evident that the effect size for the difference in BMI was small for 10, 11, 13, and 14 year old boys and 10, 12, 14 and 15 year old girls. Only 13 year old girls had a large effect size. Furthermore, the effect size for the difference in BMI of all subjects was small.

A positive but weak ($r = 0.295$) significant correlation ($p < 0.0001$) between BMI and height was found in boys when calculating the partial correlation (Pearson) coefficient

adjusted for age. Similarly, a positive but weak ($r = 0.216$) significant correlation ($p < 0.0001$) between BMI and height was found in girls also, when doing the same statistical analysis as mentioned above.

Sum of Skinfolds (TSF+SSF)

Figure 4.9 below provides an illustration of how our study is related to the anthropometric variable TSF+SSF. For the stunted girls there was a rapid increase in the mean TSF+SSF of 27.31 mm and 39.35 mm at ages 14 and 15 years respectively. Similarly, the non-stunted girls also displayed a rapid increase in the mean TSF+SSF of 32.53 mm and 34.34 mm at ages 14 and 15 years, respectively. This coincided with the increase in incidence of menarche for 14 and 15 year old girls (Table 4.3). The mean TSF+SSF for the 15 year old stunted girls was greater than the mean TSF+SSF for the non-stunted girls (Figure 4.9)

From Tables 4.8 and 4.9 it was evident that the effect size for the difference in TSF+SSF was small for 10, 12, 13, 14 and 15 year old boys and 10, 14 and 15 year old girls, while 13 year old girls had a large effect size. Furthermore, the effect size for the difference in TSF+SSF of all the subjects was small.

A positive but weak ($r = 0.188$) significant correlation ($p < 0.0001$) between TSF+SSF and height was found in boys when calculating the partial correlation (Pearson) coefficient adjusted for age. Similarly, a positive but weak ($r = 0.224$) significant correlation ($p < 0.0001$) between TSF+SSF and height was found in girls when doing the same statistical analysis as mentioned above.

Table 4.4: Anthropometric variables of 10-15 year old stunted boys (n=134)

Variable	Age	Sample Size N	Mean	Std. Dev.	Min	Max
TSF (mm)	10-10.99	11	7.75	1.77	5.20	10.40
	11-11.99	24	7.05	2.01	4.80	13.00
	12-12.99	29	9.20	4.44	4.10	20.40
	13-13.99	25	10.53	7.25	4.20	31.00
	14-14.99	18	9.38	3.44	6.00	21.00
	15-15.99	22	7.30	2.40	4.00	14.20
	TOTAL	129	8.54	3.55	4.00	31.00
MUAC (mm)	10-10.99	11	16.59	1.16	14.80	18.50
	11-11.99	22	17.19	2.12	15.30	25.60
	12-12.99	29	17.97	1.97	15.30	24.60
	13-13.99	23	18.93	2.85	15.10	26.50
	14-14.99	18	19.05	1.55	17.00	22.60
	15-15.99	21	20.63	2.62	15.40	27.30
	TOTAL	124	18.39	2.05	14.80	27.30
WC (mm)	10-10.99	11	52.84	2.35	49.80	56.50
	11-11.99	24	53.88	2.65	46.80	57.70
	12-12.99	30	56.43	4.27	50.40	68.20
	13-13.99	25	59.76	5.24	52.40	70.80
	14-14.99	18	59.17	3.19	52.80	64.20
	15-15.99	22	61.80	5.20	53.40	76.60
	TOTAL	130	57.31	3.82	46.80	76.60
weight (kg)	10-10.99	11	24.18	1.78	21.90	26.80
	11-11.99	24	25.84	3.28	18.60	33.80
	12-12.99	30	28.30	4.76	20.40	43.80
	13-13.99	25	32.68	6.04	23.70	52.10
	14-14.99	18	34.93	4.83	25.80	44.60
	15-15.99	22	39.52	6.62	28.20	52.60
	TOTAL	130	30.91	4.55	18.60	52.60
height (cm)	10-10.99	11	125.22	2.27	120.50	127.90
	11-11.99	24	130.03	4.93	120.20	142.00
	12-12.99	30	134.27	5.00	117.30	142.30
	13-13.99	25	139.56	3.51	134.00	147.00
	14-14.99	18	143.47	4.83	130.50	150.90
	15-15.99	22	151.21	4.91	140.90	158.00
	TOTAL	130	137.29	4.24	117.30	158.00
BMI (kg/m^2)	10-10.99	11	15.46	1.50	13.51	17.43
	11-11.99	24	15.27	1.65	12.87	20.76
	12-12.99	30	15.63	1.89	12.97	22.03
	13-13.99	25	16.72	2.63	12.97	24.11
	14-14.99	18	16.96	2.29	14.13	24.11
	15-15.99	22	17.20	2.20	13.75	22.38
	TOTAL	130	16.21	2.03	12.87	24.11
TSF+SSF(mm)	10-10.99	11	13.75	2.63	10.00	18.40
	11-11.99	24	12.45	3.14	9.10	23.20
	12-12.99	29	15.59	7.20	9.00	43.80
	13-13.99	25	18.86	13.92	9.00	68.20
	14-14.99	18	16.46	6.59	10.60	40.80
	15-15.99	22	14.40	4.72	8.20	29.60
	TOTAL	129	15.25	6.37	8.20	68.20

Table 4.5: Anthropometric variables of 10-15 year old stunted girls (n=105)

Variable	Age	Sample Size *N*	Mean	Std. Dev.	Min	Max
TSF (mm)	10-10.99	19	8.86	3.55	4.80	18.40
	11-11.99	15	9.56	1.98	6.30	12.80
	12-12.99	23	10.80	4.60	4.60	23.60
	13-13.99	18	9.12	2.28	5.60	13.40
	14-14.99	14	14.16	7.54	7.20	33.20
	15-15.99	12	19.75	9.02	9.20	32.80
	TOTAL	101	12.04	4.83	4.60	33.20
MUAC (mm)	10-10.99	19	16.93	1.88	15.00	22.40
	11-11.99	13	17.69	1.35	15.20	19.70
	12-12.99	22	18.65	2.13	15.80	23.40
	13-13.99	15	18.67	2.63	15.40	26.80
	14-14.99	14	21.81	3.43	18.50	28.50
	15-15.99	12	23.33	2.95	19.20	28.10
	TOTAL	95	19.51	2.40	15.00	28.50
WC (mm)	10-10.99	19	53.42	4.20	46.50	66.00
	11-11.99	15	53.47	2.82	48.20	58.40
	12-12.99	23	55.82	4.95	48.10	66.80
	13-13.99	18	56.66	4.17	50.10	68.60
	14-14.99	14	63.35	8.45	51.10	79.80
	15-15.99	12	65.35	4.86	54.40	72.20
	TOTAL	101	58.01	4.91	46.50	79.80
weight (kg)	10-10.99	18	23.81	2.84	17.70	30.20
	11-11.99	15	26.83	2.40	20.80	30.40
	12-12.99	23	29.90	5.07	22.20	44.60
	13-13.99	18	31.23	5.21	22.10	45.40
	14-14.99	14	41.51	9.56	32.80	63.70
	15-15.99	12	45.22	6.23	34.60	53.90
	TOTAL	100	33.08	5.22	17.70	63.70
height (cm)	10-10.99	18	123.65	5.93	109.30	131.90
	11-11.99	15	131.37	4.10	121.00	138.70
	12-12.99	23	134.91	5.62	116.60	141.20
	13-13.99	18	140.57	4.27	131.10	147.40
	14-14.99	14	146.03	2.30	141.00	150.00
	15-15.99	12	145.85	2.64	142.20	150.00
	TOTAL	100	137.06	4.14	109.30	150.00
BMI (kg/m^2)	10-10.99	18	15.62	2.06	12.98	20.26
	11-11.99	15	15.53	1.05	13.52	17.37
	12-12.99	23	16.38	2.31	12.82	24.18
	13-13.99	18	15.72	1.90	12.49	21.56
	14-14.99	14	19.41	4.19	15.70	29.48
	15-15.99	12	21.21	2.52	16.92	24.51
	TOTAL	100	17.31	2.34	12.82	29.48
TSF+SSF (mm)	10-10.99	19	16.85	8.88	9.60	41.10
	11-11.99	15	16.02	2.97	12.00	21.20
	12-12.99	23	18.50	7.57	11.00	39.40
	13-13.99	18	17.08	4.72	10.30	29.80
	14-14.99	14	27.31	14.76	14.20	65.20
	15-15.99	12	39.35	21.04	17.60	71.20
	TOTAL	101	22.52	9.99	9.60	71.20

Table 4.6: Anthropometric variables of 10-15 year old non-stunted boys (n=470)

Variable	Age	Sample Size N	Mean	Std. Dev.	Min	Max
TSF (mm)	10-10.99	73	10.58	6.15	4.40	35.00
	11-11.99	90	9.26	3.53	4.30	24.20
	12-12.99	96	10.84	5.88	4.80	31.80
	13-13.99	69	10.13	4.67	5.00	26.20
	14-14.99	70	9.72	7.42	4.40	60.00
	15-15.99	72	10.02	7.73	4.10	60.00
	TOTAL	470	10.09	5.90	4.10	60.00
MUAC (mm)	10-10.99	70	18.88	3.45	14.10	32.40
	11-11.99	83	18.76	2.03	15.00	26.40
	12-12.99	85	19.95	2.89	15.20	29.20
	13-13.99	61	20.88	3.21	16.20	33.00
	14-14.99	67	21.53	2.92	17.60	36.50
	15-15.99	69	23.67	2.95	18.50	33.60
	TOTAL	435	20.61	2.91	14.10	36.50
WC (mm)	10-10.99	73	57.45	6.25	49.40	85.00
	11-11.99	90	57.94	4.54	49.20	79.90
	12-12.99	96	60.44	6.19	50.50	83.00
	13-13.99	69	63.18	7.47	53.50	101.10
	14-14.99	70	64.13	7.42	55.50	102.0
	15-15.99	72	66.84	9.63	60.10	104.80
	TOTAL	470	61.66	6.92	60.10	104.80
weight (kg)	10-10.99	73	30.53	7.83	21.50	69.60
	11-11.99	90	32.27	5.59	23.50	57.60
	12-12.99	96	36.30	7.49	25.0	62.90
	13-13.99	69	41.72	10.98	30.90	89.30
	14-14.99	70	46.21	10.70	31.70	95.20
	15-15.99	72	53.51	10.78	35.70	100.50
	TOTAL	470	40.09	8.90	21.50	100.50
height (cm)	10-10.99	73	136.6	6.39	126.10	167.00
	11-11.99	90	141.88	6.65	130.20	164.70
	12-12.99	96	146.51	5.80	136.70	162.70
	13-13.99	69	153.51	7.84	143.30	176.60
	14-14.99	70	160.41	7.75	146.60	180.50
	15-15.99	72	166.96	7.17	155.70	184.50
	TOTAL	470	150.98	6.93	126.10	184.50
BMI (kg/m^2)	10-10.99	73	16.23	3.12	12.49	31.86
	11-11.99	90	15.98	2.09	12.54	24.59
	12-12.99	96	16.83	2.83	12.79	26.52
	13-13.99	69	17.50	3.21	13.29	32.17
	14-14.99	70	17.85	3.31	13.64	33.65
	15-15.99	72	19.12	3.33	13.75	36.74
	TOTAL	470	17.25	2.98	12.49	36.74
TSF+SSF (mm)	10-10.99	73	18.07	11.61	7.60	63.00
	11-11.99	90	15.72	5.73	7.70	42.40
	12-12.99	96	19.03	11.12	8.60	64.10
	13-13.99	69	18.14	8.66	8.80	50.20
	14-14.99	70	17.93	14.62	9.70	120.00
	15-15.99	72	19.56	15.74	8.10	120.00
	TOTAL	470	18.08	11.25	7.60	120.00

Table 4.7: Anthropometric variables of 10-15 year old non-stunted girls (n=541)

Variable	Age	Sample Size *N*	Mean	Std. Dev.	Min	Max
TSF (mm)	10-10.99	97	12.34	6.46	4.10	39.20
	11-11.99	93	13.02	5.34	5.00	36.00
	12-12.99	98	13.75	5.97	4.00	33.80
	13-13.99	80	14.37	5.68	5.20	32.40
	14-14.99	82	16.95	8.13	5.80	60.00
	15-15.99	89	17.76	7.56	5.40	44.40
	TOTAL	539	14.70	6.52	4.10	60.00
MUAC (mm)	10-10.99	85	19.05	2.91	14.70	28.60
	11-11.99	82	20.07	2.35	16.30	26.80
	12-12.99	91	20.96	2.85	16.10	28.60
	13-13.99	72	21.73	2.51	17.20	27.60
	14-14.99	77	23.01	3.32	17.40	37.90
	15-15.99	85	23.67	2.65	18.50	30.80
	TOTAL	492	21.42	2.77	14.70	37.90
WC (mm)	10-10.99	97	56.83	6.77	46.50	78.90
	11-11.99	93	59.88	7.82	50.80	104.00
	12-12.99	98	61.34	6.18	51.20	81.00
	13-13.99	80	62.97	5.85	50.10	78.00
	14-14.99	82	65.40	8.74	52.00	115.60
	15-15.99	89	67.11	7.09	55.90	90.60
	TOTAL	539	62.26	7.08	46.50	115.60
weight (kg)	10-10.99	97	31.46	7.73	20.30	60.30
	11-11.99	94	35.86	8.27	24.90	76.40
	12-12.99	98	39.82	8.58	25.40	61.50
	13-13.99	80	44.00	8.58	30.40	64.20
	14-14.99	82	49.88	10.69	28.40	97.30
	15-15.99	89	52.37	9.44	37.10	80.50
	TOTAL	540	42.23	8.88	20.30	97.30
height (cm)	10-10.99	97	138.5	6.53	128.00	158.50
	11-11.99	94	143.42	5.88	133.20	159.10
	12-12.99	98	150.07	6.16	138.80	163.90
	13-13.99	81	154.82	6.60	142.90	168.80
	14-14.99	82	157.84	6.58	137.40	182.20
	15-15.99	89	160.33	5.93	150.90	178.10
	TOTAL	541	150.83	6.28	128.00	182.20
BMI (kg/m^2)	10-10.99	97	16.29	3.27	9.75	29.78
	11-11.99	94	17.35	3.35	11.71	34.88
	12-12.99	98	17.56	2.96	12.78	24.71
	13-13.99	80	18.27	2.76	12.81	25.18
	14-14.99	82	19.95	3.91	15.04	41.67
	15-15.99	89	20.32	3.16	13.30	29.50
	TOTAL	540	18.29	3.24	9.75	41.67
TSF+SSF(mm)	10-10.99	97	21.85	11.77	9.40	67.00
	11-11.99	93	23.91	12.14	9.90	91.10
	12-12.99	98	24.90	10.87	10.20	59.80
	13-13.99	80	26.90	10.87	10.60	62.20
	14-14.99	82	32.53	15.78	14.81	120.00
	15-15.99	89	34.34	16.23	13.20	83.60
	TOTAL	539	27.41	12.94	9.40	120.00

Table 4.8: Differences in anthropometric indices between stunting and non-stunting in 10-15 year old boys measured by t-test

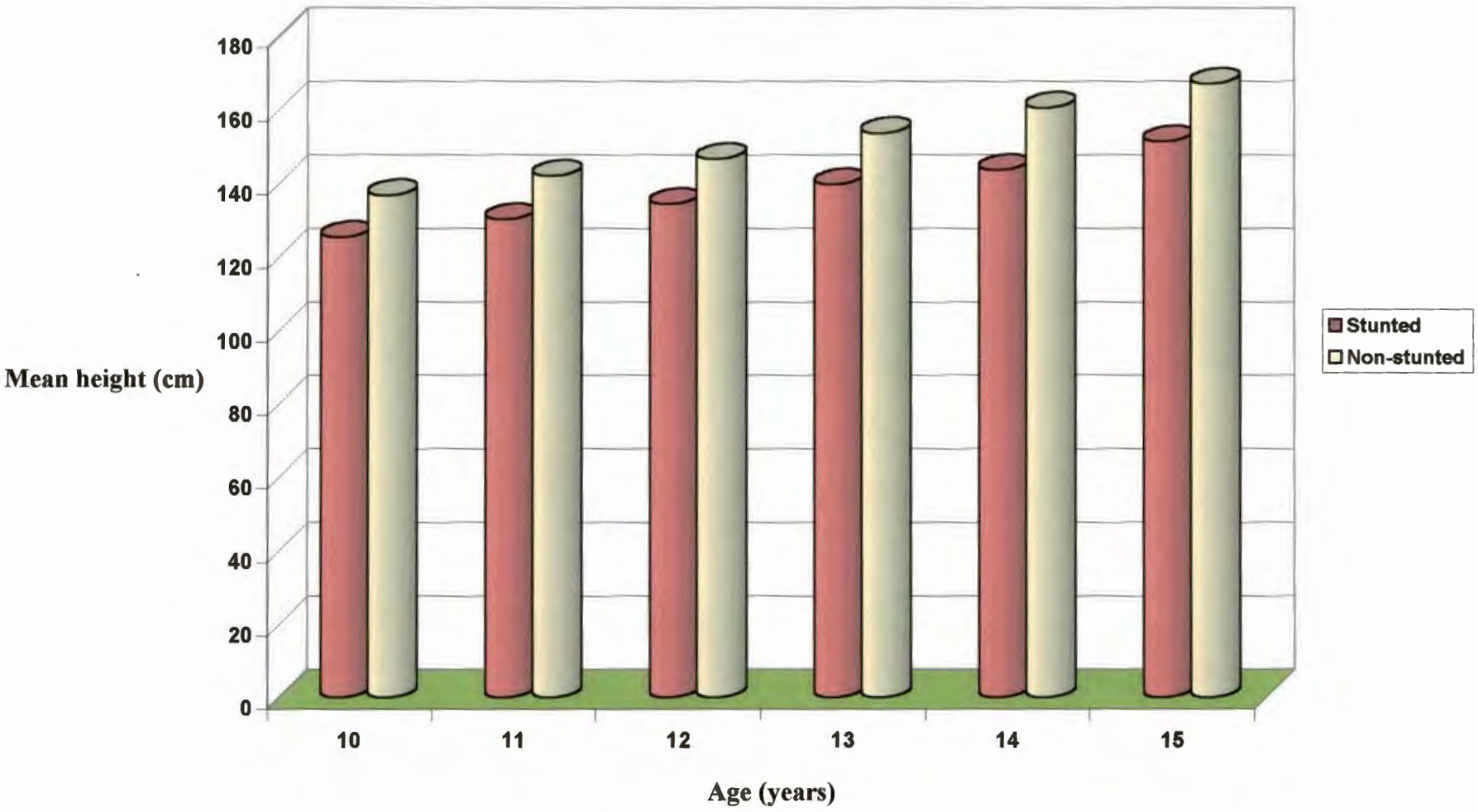
Age	Indices	Stunted	Non-stunted	t-test	P	Effect Size (d)
10	height (cm)	125.22	136.60	11.21	0.0001	1.9
	weight (kg)	24.18	30.53	5.99	0.0001	0.9
	TSF (mm)	7.7	10.60	3.16	0.0026	0.5
	WC (mm)	52.84	57.45	4.53	0.0001	0.8
	MUAC (mm)	16.59	18.88	4.22	0.0001	0.7
	BMI (kg/m^2)	15.46	16.23	1.33	0.1937	0.3
	TSF+SSF (mm)	13.75	18.07	2.75	0.0076	0.4
11	height (cm)	130.03	141.88	9.66	0.0001	1.9
	weight (kg)	25.84	32.27	7.21	0.0001	1.2
	TSF (mm)	7.05	9.26	3.99	0.0002	0.7
	WC (mm)	53.88	57.94	5.62	0.0001	1
	MUAC (mm)	17.19	18.76	3.11	0.0039	0.8
	BMI (kg/m^2)	15.27	15.98	1.77	0.0842	0.4
	TSF+SSF (mm)	12.45	15.72	3.72	0.0004	0.6
12	height (cm)	134.27	146.51	11.25	0.0001	2.2
	weight (kg)	28.30	36.30	6.91	0.0001	1.2
	TSF (mm)	9.2	10.84	1.61	0.1126	0.3
	WC (mm)	56.43	60.44	4.00	0.0002	0.7
	MUAC (mm)	17.97	19.95	4.13	0.0001	0.7
	BMI (kg/m^2)	15.63	16.83	2.66	0.0096	0.5
	TSF+SSF (mm)	15.59	19.03	1.96	0.0538	0.3
13	height (cm)	139.56	153.51	11.85	0.0001	2
	weight(kg)	32.68	41.72	5.05	0.0001	0.9
	TSF (mm)	10.53	10.13	-0.26	0.7960	-0.1
	WC (mm)	59.76	63.18	2.47	0.0163	0.5
	MUAC (mm)	18.93	20.88	2.70	0.0098	0.6
	BMI (kg/m^2)	16.72	17.50	1.19	0.2380	0.3
	TSF+SSF (mm)	18.86	18.14	-0.24	0.8103	-0.1
14	height (cm)	143.47	160.41	11.54	0.0001	2.3
	weight (kg)	34.93	46.21	6.59	0.0001	1.1
	TSF (mm)	9.38	9.72	0.28	0.7813	0.05
	WC (mm)	59.17	64.13	4.27	0.0001	0.7
	MUAC (mm)	19.05	21.53	4.86	0.0001	0.9
	BMI (kg/m^2)	16.96	17.85	1.32	0.1939	0.3
	TSF+SSF (mm)	16.46	17.93	0.63	0.5327	0.1
15	height (cm)	151.21	166.96	11.70	0.0001	2.3
	weight (kg)	39.52	53.51	7.37	0.0001	1.4
	TSF (mm)	7.30	10.02	2.61	0.0107	0.4
	WC (mm)	61.80	66.84	3.18	0.0022	0.6
	MUAC (mm)	20.63	23.67	4.52	0.0001	1.1
	BMI (kg/m^2)	17.20	19.12	3.14	0.0028	0.6
	TSF+SSF (mm)	14.41	19.56	2.44	0.0155	0.4
total	height (cm)	137.88	150.31	12.67	<.0001	1.1
	weight (kg)	31.16	39.54	10.14	<.0001	0.8
	TSF (mm)	8.63	10.10	3.08	0.0023	0.3
	WC (mm)	57.58	61.43	6.76	<.0001	0.5
	MUAC (mm)	18.49	20.52	7.33	<.0001	0.6
	BMI (kg/m^2)	16.21	17.17	4.01	<.0001	0.3
	TSF+SSF (mm)	15.40	18.03	2.98	0.0031	0.2

Guideline values according to Cohen, (1977):- small effect size: d=0.2; medium effect size: d=0.5; large effect size: d=0.8

Table 4.9: Differences in anthropometric indices between stunting and non-stunting in 10-15 year old girls measured by t-test

Age	Indices	Stunted	Non-stunted	t-test	P	Effect Size (d)
10	height (cm)	123.65	138.50	9.60	0.0001	2.3
	weight (kg)	23.81	31.46	7.42	0.0001	1.1
	TSF (mm)	8.86	12.34	3.32	0.0018	0.6
	WC (mm)	53.42	56.83	2.88	0.0064	0.5
	MUAC (mm)	16.93	19.05	3.97	0.0003	0.8
	BMI (kg/m^2)	15.62	16.29	1.14	0.2599	0.2
	TSF+SSF (mm)	16.85	21.85	2.12	0.0422	0.4
11	height (cm)	131.37	143.42	9.86	0.0001	2.1
	weight (kg)	26.83	35.86	8.56	0.0001	1.2
	TSF (mm)	9.56	13.02	4.59	0.0001	0.7
	WC (mm)	53.47	59.88	5.89	0.0001	0.9
	MUAC (mm)	17.69	20.07	5.24	0.0001	1.1
	BMI (kg/m^2)	15.53	17.35	4.14	0.0842	0.6
	TSF+SSF (mm)	16.02	23.91	5.36	0.0001	0.7
12	height (cm)	134.91	150.07	11.42	0.0001	2.5
	weight (kg)	29.90	39.82	7.26	0.0001	1.2
	TSF (mm)	10.80	13.75	2.60	0.0129	0.5
	WC (mm)	55.82	61.34	4.57	0.0001	0.9
	MUAC (mm)	18.65	20.96	4.26	0.0001	0.8
	BMI (kg/m^2)	16.39	17.56	2.08	0.0441	0.4
	TSF+SSF (mm)	18.50	24.90	3.33	0.0017	0.6
13	height (cm)	40.57	154.82	11.44	0.0001	2.8
	weight (kg)	31.23	44.00	8.19	0.0001	1.6
	TSF (mm)	9.12	14.37	6.32	0.0001	1.0
	WC (mm)	56.66	62.97	5.35	0.0001	1.1
	MUAC (mm)	18.67	21.73	4.14	0.0005	1.2
	BMI (kg/m^2)	15.72	18.27	4.69	0.0001	1.0
	TSF+SSF (mm)	17.08	26.90	5.74	0.0001	0.9
14	height (cm)	146.03	157.84	12.40	0.0001	1.9
	weight (kg)	41.51	49.88	2.97	0.0078	0.8
	TSF (mm)	14.16	16.95	1.26	0.2224	0.3
	WC (mm)	63.35	65.40	0.84	0.4145	0.2
	MUAC (mm)	21.81	23.03	1.22	0.2396	0.4
	BMI (kg/m^2)	19.42	19.95	0.45	0.6606	0.1
	TSF+SSF (mm)	27.31	32.53	1.21	0.2415	0.3
15	height (cm)	145.85	160.33	14.66	0.0001	2.6
	weight (kg)	45.22	52.37	3.48	0.0026	0.8
	TSF (mm)	19.75	17.76	-0.73	0.4785	-0.3
	WC (mm)	65.35	67.11	1.11	0.2821	0.3
	MUAC (mm)	23.33	23.67	0.37	0.7181	0.1
	BMI (kg/m^2)	21.21	20.32	-1.11	0.2839	-0.3
	TSF+SSF (mm)	39.35	34.34	-0.79	0.4420	-0.3
Total	height (cm)	136.24	150.42	14.20	<.0001	1.4
	weight (kg)	32.05	41.84	9.56	<.0001	0.9
	TSF (mm)	11.48	14.61	4.65	<.0001	0.5
	WC (mm)	57.35	62.09	6.45	<.0001	0.6
	MUAC (mm)	19.23	21.39	5.99	<.0001	0.7
	BMI (kg/m^2)	17.00	18.22	3.51	0.0006	0.4
	TSF+SSF (mm)	21.27	27.20	4.19	<.0001	0.4

Guideline values according to Cohen, (1977):- small effect size: d=0.2; medium effect size: d=0.5; large effect size: d=0.8



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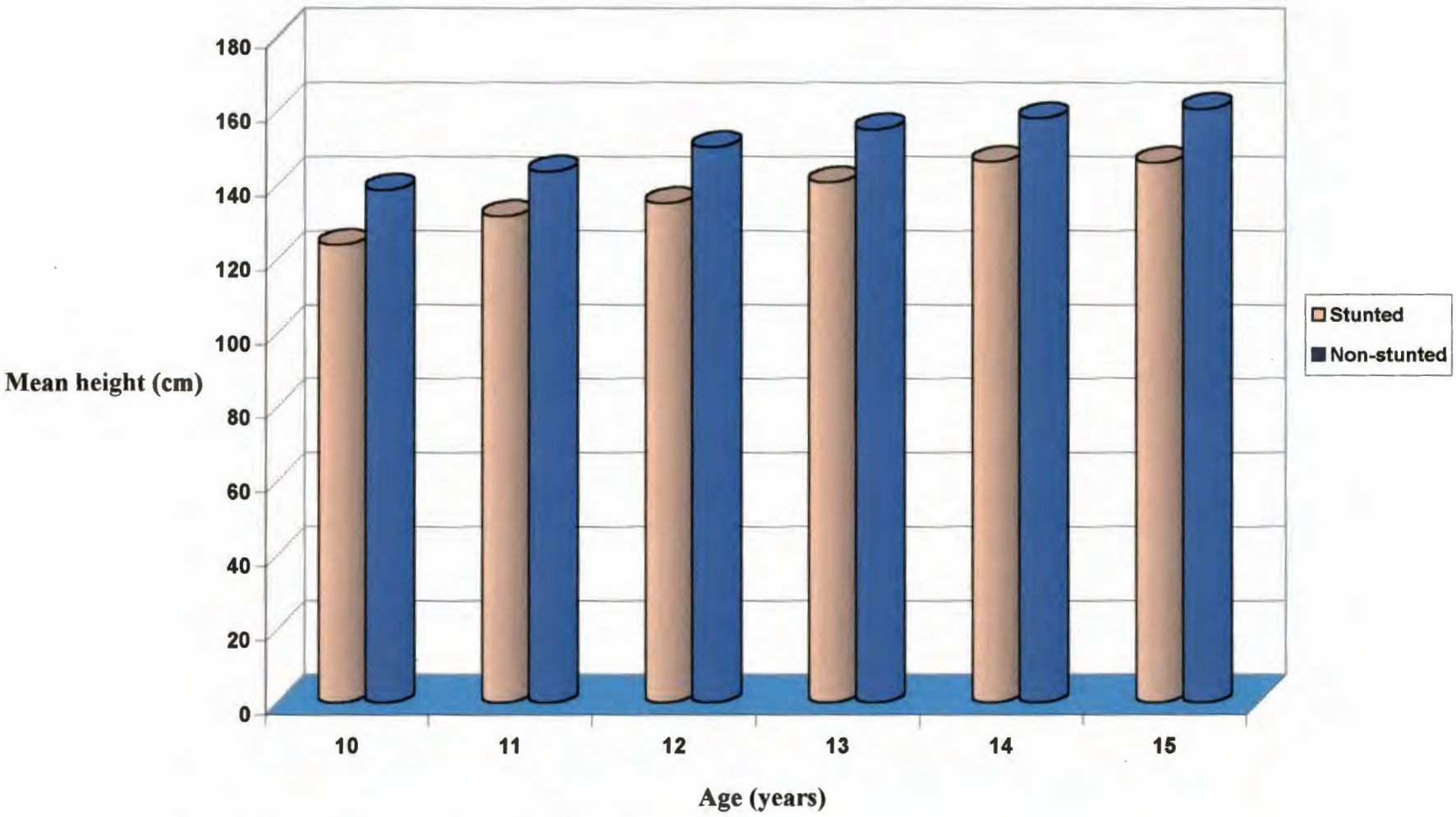
Height below the 5th percentile for age, NCHS standard percentiles

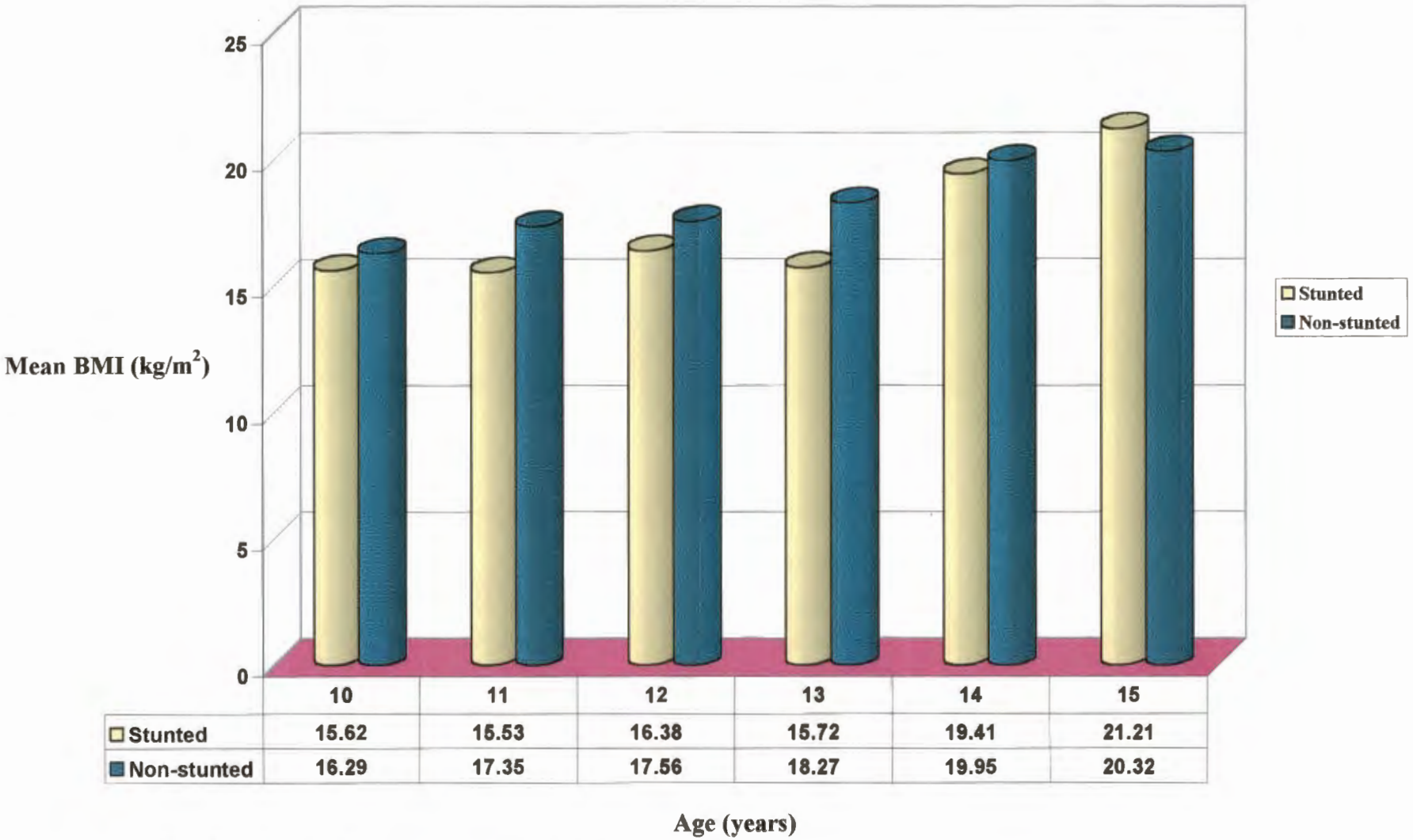
Figure 4.6: Mean height of stunted* compared to non-stunted boys



* Height below the 5th percentile for age, NCHS standard percentiles

*

Figure 4.7: Mean height of stunted compared to non-stunted girls

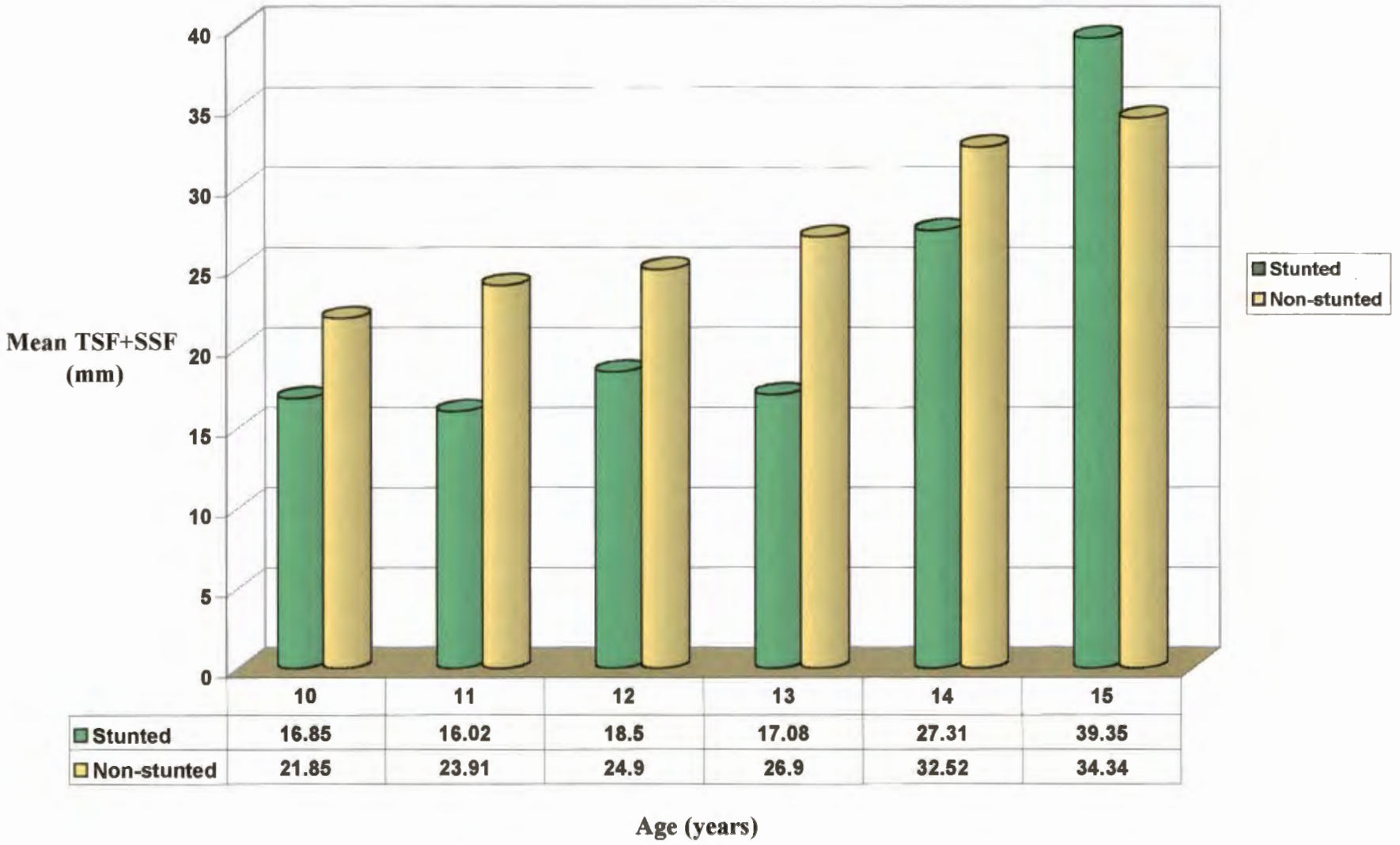


	10	11	12	13	14	15
Stunted	15.62	15.53	16.38	15.72	19.41	21.21
Non-stunted	16.29	17.35	17.56	18.27	19.95	20.32

Age (years)

Based on BMI cut-offs according to Cole *et al*. (2000)

Figure 4.8: Mean BMI of stunted compared to non-stunted girls



	10	11	12	13	14	15
Stunted	16.85	16.02	18.5	17.08	27.31	39.35
Non-stunted	21.85	23.91	24.9	26.9	32.52	34.34

*Height below the 5th percentile for age, NCHS standard percentiles

Figure 4.9: Mean TSF+SSF of stunted* compared to non-stunted girls

Stunting and Obesity

The following results were calculated manually by substituting percentages from two-way tables into the odds ratio formula used by Bland and Altman (Bland & Altman 2000).

Table 4.10: Association between stunting and overweight in 10 to 15 year old boys

Weight	**Stunted**	**Non-stunted**	**TOTAL**
Normal and underweight	130 (97.01 %)	440 (93.62 %)	570 (94.37 %)
Overweight and obese	4 (2.99 %)	30 (6.38 %)	34 (5.63 %)
TOTAL	134 (22.2 %)	470 (77.8 %)	604 (100 %)

The odds ratio is given by

$$\text{Odds ratio} = \frac{4}{130} \times \frac{440}{30} = 0.45$$

Also, we calculated a standard error for the log odds ratio and hence a confidence interval as follows.

$$SE(\log OR) = \sqrt{\frac{1}{130} + \frac{1}{440} + \frac{1}{4} + \frac{1}{30}} = 0.54$$

A 95 % confidence interval for the log odds ratio was obtained as a 1.96 standard errors on either side of the estimate. In this case, the log odds ratio is

$$\log_e(0.45) = -0.80.$$

The confidence interval for the log odds ratio is calculated by using

$$-0.80 \pm 1.96 \times 0.54,$$

which gives the interval -1.86 and 0.26. We antilog these limits to give a 95 % confidence interval for the odds ratio itself as

$$\exp(-1.86) = 0.16 \text{ to } \exp(0.26) = 1.30.$$

Thus the odds ratio for stunted boys to be overweight or obese and the associated confidence interval is given by

$$0.45 \text{ and } (0.16, 1.30).$$

Table 4.11: Association between stunting and overweight in 10 to 15 year old girls

Weight	Stunted	Non-stunted	TOTAL
Normal and underweight	99 (94.29 %)	482 (89.09 %)	581 (89.94 %)
Overweight and obese	6 (5.71 %)	59 (10.91 %)	65 (10.06 %)
TOTAL	105 (16.25 %)	541 (83.75 %)	646 (100 %)

The odds ratio is given by

$$\text{Odds ratio} = \frac{6}{99} \times \frac{482}{59} = 0.50.$$

Also, we calculated a standard error for the log odds ratio and hence a confidence interval as follows.

$$SE(\log OR) = \sqrt{\frac{1}{99} + \frac{1}{482} + \frac{1}{6} + \frac{1}{59}} = 0.442.$$

A 95 % confidence interval for the log odds ratio was obtained as a 1.96 standard errors on either side of the estimate. In this case, the log odds ratio is

$$\log_e(0.50) = -0.70.$$

The confidence interval for the log odds ratio is calculated by using

$$-0.70 \pm 0.87 \times 0.442,$$

which gives the interval -1.57 and 0.17. We antilog these limits to give a 95 % confidence interval for the odds ratio itself as

$$\exp(-1.57) = 0.21 \text{ to } \exp(0.17) = 1.19.$$

Thus the odds ratio for stunted girls to be overweight or obese and the associated confidence interval is given by

$$0.50 \text{ and } (0.21, 1.19).$$

Furthermore, a non-significant probability existed for stunted boys (p = 0.1998) and girls (p = 0.1135) to be overweight (Fisher's Exact Test).

Table 4.12: The influence of gender on the relationship between stunting and overweight and obesity

Gender	Prevalence of Stunting	Prevalence of Overweight & Obesity	Prevalence of Overweight & Obesity in Stunted	Prevalence of Overweight & Obesity in Non-stunted	Odds Ratio of Overweight & Obesity being Stunted	95 % Confidence Interval
Boys	134	34	4	30	0.45	0.16, 1.30
Girls	105	65	6	59	0.50	0.21, 1.19
TOTAL	239	99	10	89	/	/

4.4 Discussion

4.4.1 Demographic Data

According to the 1995 census, 90 % of the population (adults and children) of the North West were Black, 8 % were White, 1 % Coloured and 0.3 % Indian (Statistics SA, 1995). As mentioned previously a total of 1257 subjects participated in this study. However, with some of the anthropometric variables only 1250 subjects were measured. This study sample is still fairly representative of the entire population of 10-15 year old children in the North West Province. Coloured and Indian children were slightly overrepresented so that a minimum number of subjects could be included. This enabled comparisons to be made between ethnic groups. It was evident from the breadwinners' occupation and the type of housing and water facilities that a large proportion of the children had a poor socio-economic status (Tables 4.1 and 4.2).

4.4.2 Urbanisation and Ethnicity

Harris *et al.* (2001) found the proportion of children with stunted growth was greater in non-urban areas than in urban areas in Tibet (Harris *et al.*, 2001). Similarly, in our study we found that stunted growth was most prevalent in the rural areas and least prevalent in the urban areas (Figures 4.1).

Maturational differences between black and white girls have been reported in the past (Freedman *et al.*, 1987). It was found in past cross-sectional studies that black girls were further in the maturation process than white girls at both ages 9 and 10. This earlier onset of maturation may have a primary role to play in the development of obesity differences between the races (The National Heart, Lung and Blood Institute Growth and Health Study Research Group, 1992).

Due to the fact that differences in body fat and body mass index between black and white girls appeared to occur sometime between childhood and young adulthood, the age of 9 through 10 years was chosen as the entry age range for the National Heart, Lung and Blood Institute Growth and Health Study Research Group (NGHS); this is also the age at which little differences between blacks and whites in prevalence of obesity is expected and that encompasses the pre-pubertal stage of development. Differing rates of maturation may explain some of the observed black-white differences in body mass index, although other factors may also contribute (NGHS, 1992). In this regard, the MUAC measurements of black children should be interpreted cautiously as excess subcutaneous fat may obscure the detection of muscle wasting (Adhikari & Coovadia, 1981). The etiology of the difference in obesity between races remain unknown (NGHS, 1992).

Contrary to other studies (NGHS, 1992), our study showed that overweight and obesity were mostly found in white subjects followed by black subjects (Figure 4.3). It is possible that other confounding factors could have played a role in this outcome; for example, the low income of black families.

4.4.3 Gender and Age (Vulnerable periods of life)

The period from childhood to adolescence and then from adolescence to adulthood is considered a particularly nutritionally vulnerable time. Not only does significant physical change take place during these times, but food habits and lifestyles evolve that form the basis for long-term health. At least two and possibly three critical periods exist in childhood for the development of later obesity and its attendant complications of diabetes, hypertension, hypercholesterolaemia and cardiovascular disease (Dietz, 1994).

A greater percentage of the girls were overweight or obese by comparison with the overweight or obese boys. Adolescence represents the final proposed period for the development of obesity. Both the risk of onset and persistence of obesity appear greater for females than for males. Long-term follow-up studies of adolescents suggest that $\pm$ 30 % of all obese adult women were obese early in adolescence, whereas only 10 % of obese adult males had onset of their obesity as teenagers (Braddon *et al.*, 1986). Approximately 70 % of obese males, but only 20 % of obese females returned to normal weight over a 10 year period (Garn & Cole, 1980; Garn *et al.*, 1979). These findings indicate that girls may be at particular risk for adult obesity if their disease is present or develops during adolesence. Adolescent-onset obesity in females that persists into adulthood may herald a lifelong problem (Dietz, 1994). Possible explanations for

this is that as girls age they become more interested in appearance and pursue more sedentary social interests, or they may adopt sedentary, adult-modeled behaviour patterns (Stephens *et al.*, 1985).

In our study it was clear that the following gender differences existed. The mean TSF was smaller at a later age in boys than in girls. This could be directly related to girls entering puberty sooner than boys. Furthermore, waist circumference in boys was slightly smaller at 14 years. However, the girls' waist circumference suddenly increased at 14 and 15 years of age. This could be explained by the increased muscularity of boys and increased adiposity of girls at the onset of puberty (Dietz, 1994). In addition, as expected the girls mean weight and BMI was greater than the boys mean weight and BMI. Linear growth slowed down a bit at 10 years of age in boys and at 15 years of age in girls (Tables 4.5 and 4.7). In girls, a possible explanation for this is the onset of puberty. The increase in the TSF+SSF in the girls coincided with the onset of menarche (Table 4.9).

4.4.4 Menarche

In girls, menarcheal status furnishes reference data that is less detailed but easier to collect in order to evaluate puberty-related BMI. O'Dea and Abrahams (1995) reported significant differences when comparing BMI of age matched pre-menarche vs post-menarche girls. These researchers performed a study in a larger number of subjects and provided evidence of significant differences between pre-menarche and post-menarche girls of similar age, with BMI being higher in the latter. However, multivariate tests demonstrate that menarcheal status did not influence BMI when corrected by pubertal stage and age (Bini *et al.*, 2000). They demonstrated that degree of pubertal maturation has a greater influence on BMI than age in both genders, and this is even more evident in girls (Bini *et al.*, 2000). The latter was evident in our study too (Table 4.3).

4.4.5 Socioeconomic Status and Genetics

Apart from health risks, obesity has been shown to be associated with social and economic disadvantages (Mo-Suwan *et al.*, 1999). However, the importance of socioeconomic factors in the development of childhood obesity still remains controversial (Strauss & Knight, 1999). Previous researchers have shown a twofold to threefold increase in risk of obesity for indigent children (Linhares *et al.*, 1986). Others have documented a ninefold increased risk of obesity in children who were neglected

(Lissau-Lund Sorensen & Sorensen, 1992). A number of epidemiological studies reflect the importance of socioeconomic factors such as nutrition in the growth of children (Martorell *et al.*, 1988). Furthermore, a study on Brazilian children showed that underprivileged children were shorter and had a delay in bone maturation (Lissau-Lund & Sorensen, 1994). In another study short stature was clearly associated with poverty in all three groups examined, namely Mexican-Americans, non-Hispanic whites, and blacks (Martorell *et al.*, 1987). In a previous study it was confirmed by using the Mantel-Haenszel χ^2 test that an inverse linear relationship exists between the incidence of obesity and family income, parental occupation and maternal education (Strauss & Knight, 1999).

It is clear that the greater proportion of our study population was from poor socioeconomic conditions. Even though no significant association between stunting and overweight existed in this age range there was a tendency especially in stunted girls aged 15 years to have greater skinfold thicknesses than non-stunted girls (Table 4.9).

While genetic factors are obviously the major determinant of linear growth potential, reaching this potential is dependent on a number of environmental factors of which nutrition is of great importance (Loveridge & Noble, 1994). In this regard, however, it is interesting to note that it was shown that the shortness of Mexican-American adolescents is of genetic origin (Martorell *et al.*, 1987).

4.4.6 Anthropometric Status

As was previously reported about effect size it was clear that the difference between the mean TSF of stunted and non-stunted boys was small (Table 4.8). In addition, a medium difference of the TSF between stunted and non-stunted girls was evident (Table 4.9). Therefore, it was apparent that mean TSF was significantly different for 10, 11 and 15 year old boys and non-significant for 12,13 and 14 year old boys (Table 4.8). However, there was no significant difference between the mean TSF of stunted and non-stunted girls at ages 14 and 15 years (Table 4.9)

Previous studies on children and adults demonstrated strong correlations between TSF and BMI (Roche *et al.*, 1981; Michielutte *et al.*, 1984; Revicki & Israel, 1986; Dietz *et al.*, 1988). The association between TSF and BMI was weaker in males, perhaps because in males BMI is altered more by muscularity than in females. Similarly, in our study, the Pearsons correlation of TSF and BMI was weaker in boys than in girls. The strongest correlation between TSF and BMI was in the non-stunted girls ($r= 0.792$, $P < 0.0001$) compared to the stunted girls ($r= 0.777$, $P < 0.0001$). In

addition, stunted girls 14 to 15 years old, were not catching up on growth in height, but they had similar mean TSF thicknesses than non-stunted girls (Table 4.9).

The mean values for the MUAC for both the stunted boys and girls were less than the mean value for the non-stunted boys and girls for each age group. This could have been caused by a poor nutritional status in the stunted subjects, due to protein energy malnutrition (PEM). The lower mean value for the MUAC for the boys than for the girls could be due to differences in age of the onset of puberty in girls and boys. In a study done in the Western Cape, it was found that all the coloured and black 11 year old girls had greater mean MUAC values than the coloured and black boys (Steyn *et al.*, 1989).

As was previously mentioned the effect size was indicative of a medium difference of the MUAC between stunted and non-stunted subjects. Therefore, it was apparent that MUAC was significantly different between the stunted and non-stunted subjects, except in girls 14-15 years old. In this regard significance depends on the P-value, $P < 0.05$ (Tables 4.8 and 4.9).

Stunted boys' mean waist circumference increased gradually with age but was slightly smaller at 14 years of age. Contrary to this the stunted girls' mean waist circumference gradually increased up to age 13 and then there was a sudden increase at age 14 and 15. This coincided with a rapid increase in BMI and TSF+SSF at ages 14 and 15 years in the stunted girls (Figures 4.8 and 4.9). This in turn coincided with a sudden increase in the incidence of menarche at ages 14 and 15 years (Table 4.3).

Furthermore, as mentioned earlier, the effect size was an indication that the difference in the mean waist circumference between stunted and non-stunted subjects was medium. In essence, the mean waist circumference difference between stunted and non-stunted subjects was significant in girls up to the age of 13 years (Table 4.9).

The impact of abdominal visceral fat accumulation on metabolic derangement is now under extensive study in adults. Abdominal visceral fat has recently been measured in children (Goran *et al.*, 1997; Owens *et al.*, 1988). These studies have suggested that deleterious effects of visceral adipose tissue on lipid/lipoprotein risk factors seen in adults were already present in children. Predicting visceral fat from anthropometric measures such as waist circumference, WHR and sagittal diameter has been attempted in both adults and children (Goran *et al.*, 1998; Owens *et al.*, 1999). To date, no single anthropometric index has yet been generally accepted to be superior to others as a surrogate of visceral fat measurement.

One potential difficulty with the waist circumference measurements will be its use across ethnic groups. Children from different populations vary in their rate of proportional growth and in fat patterning (Goran *et al.*, 1997). Visceral adiposity is highly variable in children and is related to ethnicity. Goran *et al.*, (1997) have shown that the relative distribution of intra-abdominal tissue compared with the subcutaneous abdominal region is significantly lower in African-American children than in whites. It then seems prudent to develop standards which are truly representative of children from all ethnic backgrounds.

The results of previous studies suggested that the type of obesity changes with age in obese children. The older obese children tended to gain more fat at the umbilical level than the younger ones. This was considered as part of the general worsening of body build during growth in obese children (Asayama *et al.*, 2000). Similarly, in our study the older stunted girls exhibited more fat at the umbilical level than the younger ones. Even though these stunted girls were not overweight they exhibited a tendency to increase in WC as they get older (Table 4.9).

From Table 4.8 and 4.9 it was clear that the effect size of all subjects for weight and height was large. This was indicative of an important difference in weight and height, respectively, in stunted and non-stunted subjects.

Comparisons between populations with respect to growth assessment can be very precise when they are made with a common reference such as the current, representative NCHS percentiles (Roberts & Dallal, 2001). Despite the fact that the related subject of cut-off points is a reason for much debate, those used for BMI and stature in this study were well established (WHO, 1995). It is possible that childhood height could provide a simple tool in more accurately predicting which children were likely to become overweight adults (Freedman *et al.*, 2001). The BMI is a function of weight and height only and when it increases in populations over time changes in lean body mass cannot be readily distinguished from changes in body fatness (Reilly *et al.*, 2000).

Contrary to these important differences, the effect size for the difference in BMI of all subjects was small. Therefore, it is apparent that although BMI was significantly different between the stunted and non-stunted groups of children, the effect size was small (Tables 4.8 to 4.9). However, it was imperative to note that the mean BMI of the 15 year old stunted girls was greater than the mean BMI of the non-stunted girls (Figure 4.8). The mean BMI values of both the stunted and non-stunted girls at 14 and 15 years were approaching the published cut-off points for overweight according to Cole *et al.*, (2000), namely 23.3 kg/m^2 and 23.9 kg/m^2, respectively.

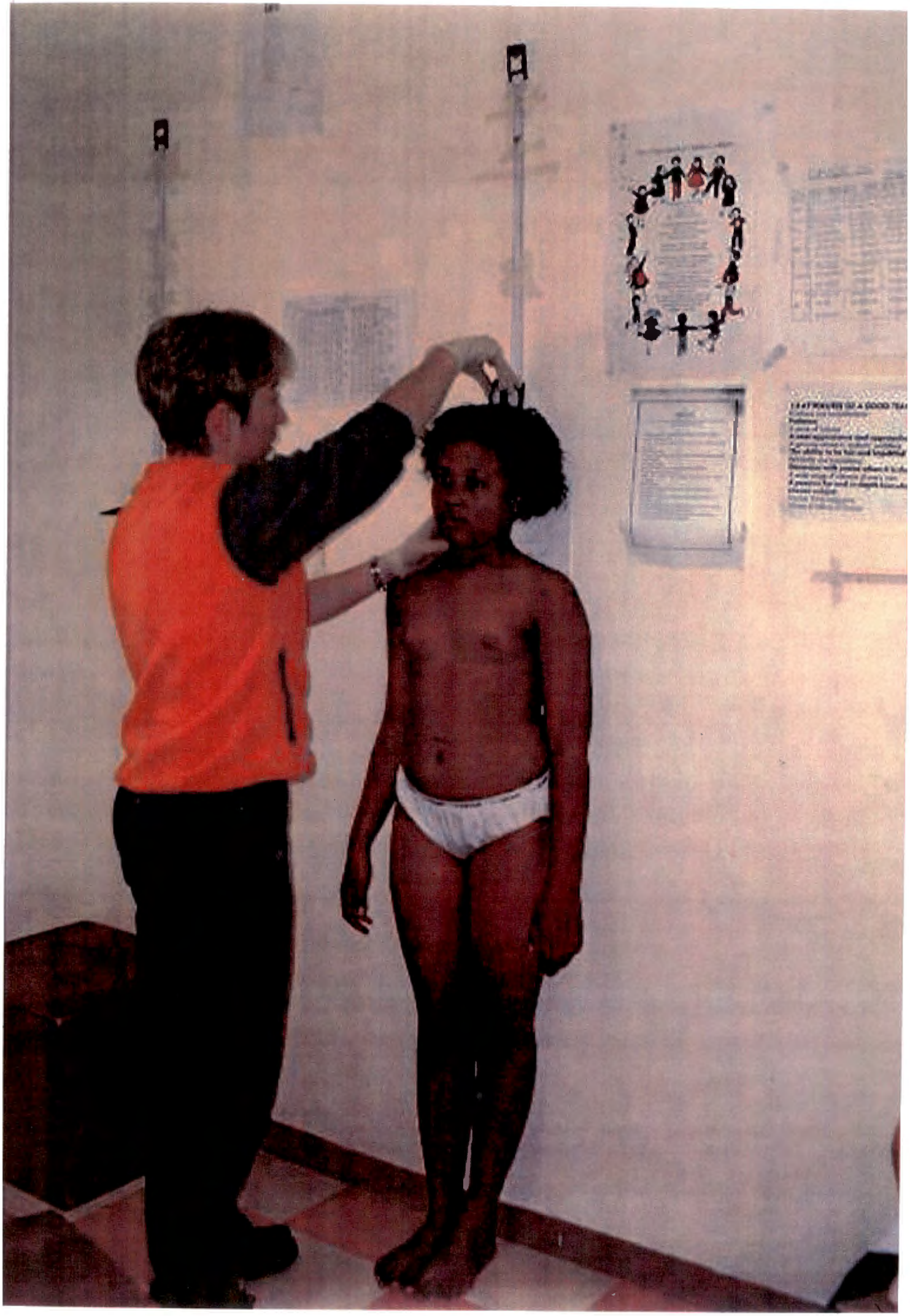
In our study the mean TSF+SSF for the total group of boys was lower than the mean TSF+SSF for the total group of girls. Furthermore, the stunted 15 year old girls had a greater mean TSF+SSF than the non-stunted girls (Figure 4.9). This was indicative of the stunted girls displaying a tendency to gain fat rapidly after 14 years of age despite the stagnation of linear growth. Therefore stunted girls older than 15 years of age have a tendency to become overweight. This result coincides with similar findings in other studies. In particular, it was found that girls have almost 50 % greater skinfold thickness than boys at all ages (Musaiger and Gregory, 2000) and the percentage body fat was lower in obese boys than in obese girls (Asayama *et al.*, 1998).

In a study done on shantytown children from Sao Paulo, Brazil the authors found that low energy expenditure may be a risk factor for weight gain in susceptible populations. This indicated that low energy expenditure may help to explain the increased risk of excess weight gain leading to obesity among shantytown girls compared with shantytown boys (Hoffman *et al.*, 2000b).

4.4.7 Stunting and Obesity

The association between stunting and overweight or obesity was determined by using the odds ratio calculation (Bland & Altman, 2000). In addition, two-way tables of weight group by height status for all the ages confirmed the odds ratio calculations above (Tables 4.10 and 4.11).

The odds ratio for stunted children to be overweight or obese was 0.45 (95 % CI 0.16, 1.30) and 0.50 (95 % CI 0.21, 1.19) for the boys and girls respectively. This was an indication that both the stunted girls and the boys were significantly less likely to be overweight. However, stunted girls showed a tendency to become overweight after 14 years of age (Table 4.12 and Figure 4.8).



Chapter 5

Conclusions and Recommendations

5.1 Introduction

The causes of childhood overweight and obesity, like many public health problems, are complex. Genetic, ethnic, hormonal, psychological and sociological factors, dietary intakes, levels of activity and meal patterns can all influence the incidence and progression of overweight and obesity. In particular, the research question that this study set out to answer was to determine the association between stunting and overweight among 10-15 year old children in the North West Province.

In order to achieve the aims of this study, a literature review was done in Chapter 2, which explained certain terms and reviewed different methods of ascertaining body composition, together with their advantages and disadvantages. Some prominent articles that have been written and studies that have been conducted to explain the relationship between stunting and overweight/obesity in modern society were reviewed. For instance, in Hoffman *et al.* (2000a) childhood nutritional stunting is associated with impaired fat oxidation, a factor that predicted obesity in at risk populations. This finding may help explain increases in body fatness and the prevalence of obesity among stunted adults and adolescents in developing countries. Also, Tanner (1955) claimed that the final size of the subjects and the speed with which it is attained are not necessarily related. Studying boys, Tanner concluded that there is

a strict relationship between adult height and maturity group, the latest maturers being the tallest. Also, it was found that later-maturing girls are somewhat taller than average. It is not surprising that the ultimate height of the obese child, who is known to mature early, is shorter than average, even though until puberty he or she may have been taller.

A comprehensive explanation of the anthropometric techniques employed was provided in Chapter 3, as well as a description of the test subjects, measuring protocol, variables measured and apparatus used. The statistical analyses were also discussed in detail.

Complete results obtained from the THUSA BANA study were given and discussed in Chapter 4. Descriptive statistics involving the demographic data of subjects, anthropometric variables, overweight and obesity was presented and explained. Finally, the association between stunting and overweight was investigated. In addition, the influence of certain factors such as age (vulnerable periods in life), gender, menarche, socioeconomic status genetics, physical activity, ethnicity and strata on stunting and overweight/obesity in 10-15 year old children were reported and discussed.

5.2 Important Observations

Important observations of this study are:

An important percentage of the study sample was stunted. More boys were stunted than girls. From the study sample that was representative of the population of 10-15 year old children in the North West Province we concluded that 19 % are stunted.

A small percentage of the study sample was overweight (6.32 %) and obese (1.6 %). In addition, an even smaller percentage of subjects were both overweight and stunted or obese and stunted (0.8 %).

About half (51 %) of the stunted girls and 43 % of the stunted boys came from rural areas. Most of the subjects lived under poor socioeconomic conditions.

The mean triceps value for the total group of stunted boys and girls were lower than the non-stunted boys and girls, respectively. However, the mean TSF of the 15 year old stunted girls was greater than the mean TSF of the 15 year old non-stunted girls.

There was a progressive increase in the MUAC with increasing age in all the subjects.

The older stunted girls exhibited more fat at the umbilical level than the younger ones, measured as WC. This coincided with a rapid increase in BMI and sum of skinfolds at ages 14 and 15 years in the stunted girls. This in turn coincided with a sudden increase in the incidence of menarche at ages 14 and 15 years.

There was a gradual increase in weight with age in all the non-stunted subjects. In addition, there was a gradual increase in weight with age in the stunted boys, but the stunted girls displayed a rapid increase in weight at ages 14 and 15. This coincided with the increase in the incidence of menarche, in other words it coincided with the onset of puberty. Furthermore, all the girls' mean weights were more than the mean weights of boys for the corresponding ages, except for the 10 year old stunted girls. Among the different ethnic groups it was evident that the white subjects had comparatively the largest occurrence of overweight and obesity.

Girls' BMI increased with age and exhibited a sudden increase at 14 and 15 years. The mean BMI values of both the stunted and non-stunted girls at 14 and 15 years are similar to the published cut-off points for overweight, according to Cole *et al.*, (2000). However, stunted girls showed a tendency to become overweight at 15 years of age.

There was a rapid increase in the mean sum of skinfolds of 14 and 15 year old stunted girls. Similarly, the non-stunted girls also displayed a rapid increase in the mean sum of skinfolds at 14 and 15 years of age. This coincided with the increase in prevalence of menarche for 14 and 15 year old girls. The mean sum of skinfolds for the total group of boys were lower than the mean sum of skinfolds for the total group of girls.

5.3 Conclusions

The conclusions of this study are given in relation to the hypothesis that were set out at the begining of the study.

Hypothesis:

There is no significant association between stunting and overweight in 10-15 year old children of the North West Province in South Africa.

The results indicate that there was no significant association between stunting and overweight in 10-15 year old children of the North West Province in South Africa.

The hypothesis is therefore accepted.

Children participating in the THUSA BANA study were representative of the children in the North West Province. It was evident that unlike many areas in other developing countries there appeared to be no dramatic increasing prevalence in overweight and stunting or in obesity and stunting.

In fact, an important percentage of the study population was stunted, but a small percentage of the study population was overweight and obese. In addition, an even smaller percentage of subjects were both stunted and overweight or stunted and obese.

Postmenarcheal girls had a tendency to have more fat at the umbilical level than the premenarcheal girls. This is in line with previous research which suggests that older obese children tended to gain more fat at the umbilical level than the younger ones, and this was considered as the general worsening of body build during growth in obese children (Asayama *et al.*, 2000). Similarly, in our study the older stunted girls exhibited more fat at the umbilical level than the younger ones. This would indicate that there is a tendency for stunting and overweight or obesity to go together in girls older than 14 years. However, no relationship between overweight or obesity with stunting is evident in the total population.

The highest prevalence of stunting was found in rural areas, followed by informal settlements and the lowest prevalence of stunting was in urban areas.

Gender differences were very pronounced when looking at the different anthropometric indices. In particular, boys' weight changes with age were gradual, however girls displayed a more dramatic increase that coincided with the onset of menarche. Girls' sum of skinfolds exceeded those of the boys. However, stunting was more prevalent in the boys than the girls.

Among the different ethnic groups it was evident that the white subjects had comparatively the largest occurrence of overweight and obesity.

There was a rapid increase in the number of girls reaching menarche at ages 14 and 15 years.

In conclusion, there is no significant association between stunting and overweight or stunting and obesity among 10-15 year old children in the North West Province of South Africa. However, stunted girls showed a strong tendency to have more subcutaneous fat than non-stunted girls even though at these ages (10-15 years) they are still underweight. Furthermore, this tendency in stunted girls coincided with the onset of menarche.

5.4 Recommendations

A better understanding is required of the critical periods for the development of adiposity, stunting and its sequelae. This may also serve to focus preventive and therapeutic efforts on developmental stages, when these efforts are likely to be most cost effective.

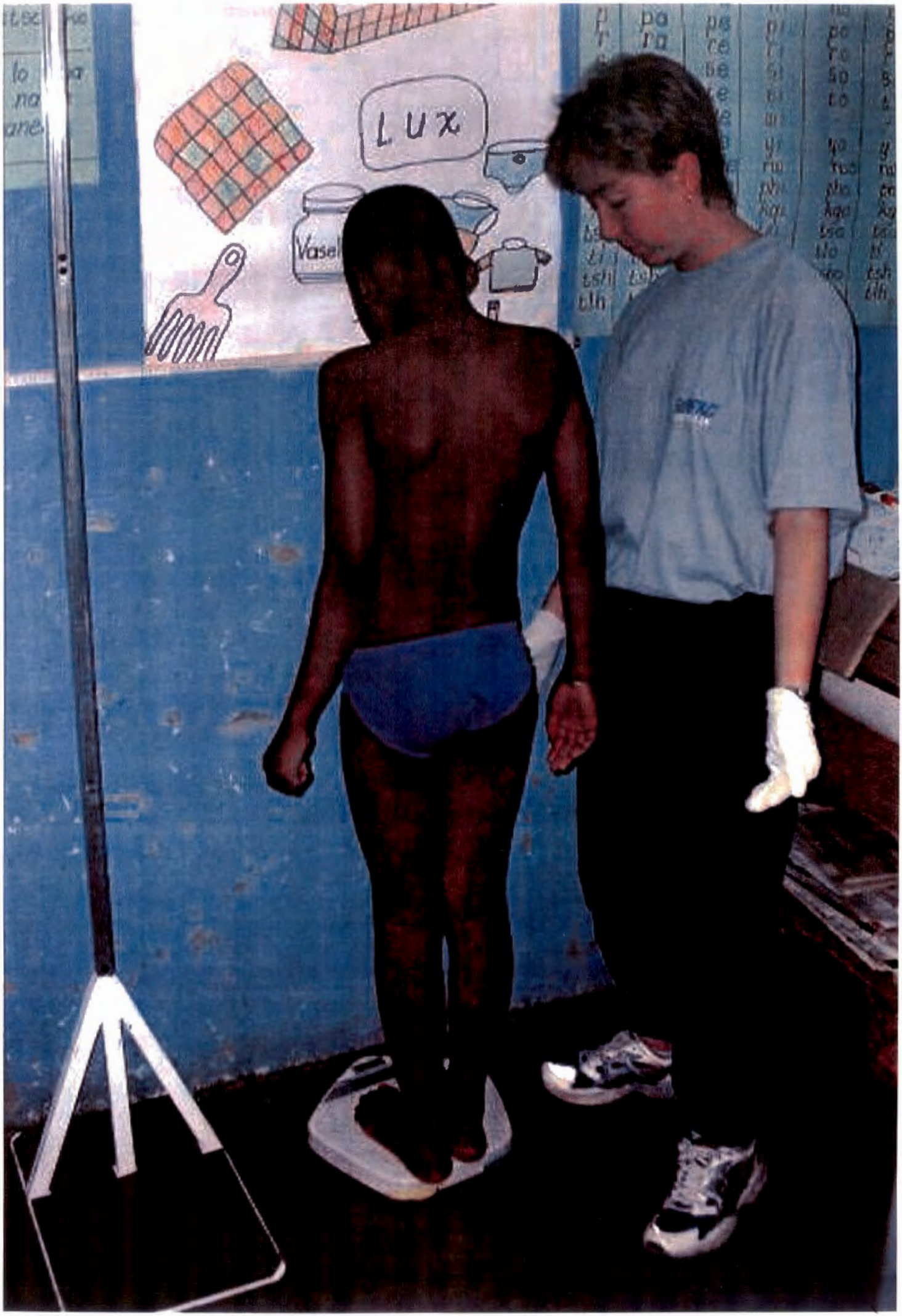
Futher investigations should, therefore, assess disease risks for those remaining fat over long periods and for those overweight and being stunted from childhood and adolescence. This should be done in a longitudinal study.

Continued collaboration between academics, health professionals, the media and the food industry is essential in helping children establish good nutrition patterns that will achieve optimum health into adulthood.

Stunting in children is a consequence of many adverse factors, including poor nutritional status of pregnant mothers and children, low birth weight due to inadequate food intake (Bellamy, 2000), parents' short stature (Amigo *et al.*, 2000), low income, exposure to infection and other diseases, emotional stress (Walker *et al.*, 2000), lack of physical activity and perhaps the stressful physiologic effects of high altitude (Martorell, 1996; Frongillo, 1999).

Therefore, further research needs to be done about the mechanisms of the above mentioned criteria or risk factors, in an attempt to formulate specific interventions for the groups at risk of being overweight/obese and stunted.

In addition, reducing poverty and its consequences is still of paramount importance.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Chapter 6

Bibliography

ABRAHAM, S. & NORDSIEK, M. 1960. Relationship of excess weight in children and adults. *Public health report,* 75:263.

ADHIKARI, M. & COOVADIA, H.M. 1981. Mid-upper arm circumference in the assessment of growth retardation. *South African medical journal,* 59:2.

AMIGO, H., BUSTOS, P., LEONE, C. & RADRIGAN, M.E. 2000. Growth deficits in Chilean school children. *American Society for Nutritional Sciences,* 1:251-254.

ARISTIMUNO, G.G., FOSTER, T.A., VOORS, A.W., SRINIVASAN, S.R. & BERENSON, G.S. 1984. Influence of persistent obesity in children on cardiovascular risk factors. *Circulation,* 69:895-904.

ASAYAMA, K., HAYASHI, K., HAYASHIBE, H., UCHIDA, N., KODERA, K. & 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.

ASAYAMA, K., HAYASHIBE, H., DOBASHI, K., UCHIDA, N., KAWADA, Y. & NAKAZAWA, S. 1995. Relationships between biochemical abnormalities and anthropometric indices of overweight, adiposity and body fat distribution in Japanese elementary school children. *International journal of obesity,* 19:253-259.

ASAYAMA, K.,OGUNI, T., HAYASHI, K., DOBASHI, K., FUKUNAGA, Y., KODERA, K., TAMAI, H. & NAKAZAWA, S. 2000. Critical value for the index of body fat

distribution based on waist and hip circumference and stature in obese girls. *International journal of obesity,* 24:1026-1031.

BARKER, D.J.P. 1992. Fetal and infant origins of adult disease. London: British Medical Journal Publishing Group.

BELLAMY, C. 2000. The state of the world's children 2000. UNICEF p.71 and Tables 1 and 2.

BENEFICE, E., GARNIER, D., SIMONDON, K.B. & MALINA, R.M. 2001. Relationship between stunting in infancy and growth and fat distribution during adolescence in Senegalese girls. *European journal of clinical nutrition,* 55:50-58.

BELLIZZI, M.C. & DIETZ, W.H. 1999. Workshop on childhood obesity: summary of the discussion. *American journal of clinical nutrition,* 70 Supplement:173S-175S.

BINI, V., CELI, F., BERTIOLI, M.G., BACOSI, M.L., STELLA, P., GIGLIO, P., TOSTI, L. & FALORNI, A. 2000. Body mass index in children and adolescents according to age and pubertal stage. *European journal of clinical nutrition,* 54:214-218.

BLAND, J.M. & ALTMAN, D.G. 2000. The odds ratio. *British medical journal,* 320:1468.

BONJOUR, J.P., CARRIE, A.L., FERRARI, S., CLAVIEN, H., SLOSMAN, D., THEINTZ, G. & RIZZOLI, R. 1997. Calcium-enriched foods and bone mass growth in prepubertal girls: a randomized, double-blind, placebo-controlled trial. *Journal of clinical investigations,* 99(6) 1287-1294.

BOOTH, M.L., MACASKILL, P., LAZARUS, R. & BAUR, L.A. 1999. Sociodemographic distribution of measures of body fatness among children and adolescents in New South Wales, Australia. *International journal of obesity,* 23:456-462.

BRADDON, F.E.M., RODGERS, B., WADSWORTH, M.E.J. & DAVIES, J.M.C. 1986. Onset of obesity in a 36 year birth cohort. *British medical journal,* 293:299-303.

BRAMBILLA, P., MONZONI, P., SIRONI, 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.

BROWNELL, K.D. & STUNKARD, A.J. 1999. Physical activity in the development and control of obesity. Obesity. Philadelphia: Saunders. 300-324p.

BRUCH, H. 1975. Emotional aspects of obesity in children. *Pediatric annuals,* 4:91-99.

COHEN, J. 1977. Statistical power analysis for the behavioral sciences. Revised ed. New York: Academic Press, Inc.

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,* 320:1240-1243.

COLE, T.J., FREEMAN, J.V., & PREECE, M.A. 1995. Body mass index reference curves for the UK, 1990. *Archives of disease in childhood,* 73:25-29.

CRONK, C.E. & ROCHE, A.F. 1982. Race and sex -specific reference data for triceps and subscapular skinfolds and weight /staure. *American journal of clinical nutrition,* 35:347-354.

DE ONIS, M. & BLOSSNER, M. 2000. Prevalence and trends of overweight among preschool children in developing countries. *American journal of clinical nutrition,* 72:1032-1039.

DIBLEY, M.J., GOLDSBY, J.B., STAEHLING, N.W. & TROWBRIDGE, F.L. 1987. Development of normalized curves for the international growth reference: historical and technical considerations. *American journal of clinical nutrition,* 46:736-748.

DIETZ, W.H. 1986. Prevention of childhood obesity. *Pediatric clinical, North America,* 33:823-833.

DIETZ, W.H. 1994. Critical periods in childhood for the development of obesity. *American journal of clinical nutrition,* 59:955-959.

DIETZ, W.H. 1999. How to tackle the problem? The role of education in the prevention of obesity. *International journal of obesity,* 23(4)Supplement:S7-S9.

DIETZ, W.H., BANDINI, L., SCHOELLER, D.A. & GORTMAKER, S.L. 1988. Diagnosis of obesity in adolescents and young adults. In: Berry EM, Blondheim SH, Eliahou HE eds. Recent advances in obesity research: V. Proceedings of the 5th International Congress on Obesity, Jerusalem, 1986. London: John Libby and Co., 1988: 9-15.

DIETZ, W.H. & BELLIZZI, M.C. 1999. Introduction : the use of body mass index to assess obesity in children. *American journal of clinical nutrition,* 70:123-125.

DIETZ, W.H. & GORTMAKER, S.L. 1985. Do we fatten our children at the television set? Obesity and television viewing in children and adolescents. *Pediatrics,* 75:807-812.

DIPIETRO, L., MOSSENBERG, H.O. & STUNKARD, A.J. 1994. A 40-year history of overweight children in Stockholm: lifetime overweight, morbidity and mortality. *International journal of obesity,* 18:585-590.

FLEGAL, K.M., CARROLL, M.D., KUCZMARSKI, R.J. & JOHNSON, C.L. 1998. Overweight and obesity in the United States: prevalence and trends. *International journal of obesity,* 22:39-47.

FOGEL, R.W. 1994. Economic growth, population theory, and physiology: the bearing of long-term processes on the making of economic policy. *American economic review,* 84:369-395.

FREEDMAN, D.S., KETTLE KHAN L., SERDULA, M.K., SRINIVASAN, S.R. & BERENSON, G.S. 2001. BMI rebound, childhood height and obesity among adults: the Bogalusa Heart Study. *International journal of obesity*, 25:543-549.

FREEDMAN, D.S., SRINIVASAN, S.R., WEBBER, L.S., BURKE, G.L. & BERENSON, G.S. 1987. Black-white differences in serum lipoproteins during sexual maturation: the Bogalusa Heart Study. *Journal of chronic diseases*, 40:309-318.

FRENCH, S.A., STORY, M. & PERRY, C.L. 1995. Self-esteem and obesity in children and adolescents: a literature review. *Obesity research,* 3:479-490.

FRONGILLO, E.A. 1999. Symposium: Causes and etiology of stunting. *Journal of nutrition,* 129(2) Supplement:S529-S530.

GARN, S.M., BAILEY, S.M. & COLE, P.E. 1979. Continuities and changes in fatness and obesity. In: Nutrition physiology and obesity. Florida: CRC Press, Inc.

GARN, S.M., BAILEY, S.M., SOLOMON, M.A. & HOPKINS, P.J. 1981. Effect of remaining family members on fatness prediction. *American journal of clinical nutrition,* 34:148-153.

GARN, S.M. & COLE, P.E. 1980. Do the obese remain obese and the lean remain lean? *American journal of public health,* 70:351-353.

GARN, S.M. & HASKELL, J.A. 1959. Fat and growth during childhood. *Science,* 130:1711-1712.

GARN, S.M., SULLIVAN, T.V. & HAWTHORNE, V.M. 1989. Fatness and obesity of parents of obese individuals. *American journal of clinical nutrition,* 56:1308-1313.

GARROW, J.S. 1988. Health implications of obesity. In: Obesity and related diseases. London: Churchill Livingstone. 1-16p.

GIBSON, R.S. 1990. Principles of nutritional assessment. New York: Oxford University Press.

GORAN, M.I., GOWER, B.A., TREUTH, M.S. & NAGY, T.R. 1998. Prediction of intra-abdominal and subcutaneous abdominal adipose tissue in healthy prepubertal children. *International journal of obesity,* 22:549-558.

GORAN, M.I., NAGY, T.R., TREUTH, M.S., TROWBRIDGE, C. & DEZENBERG, C. 1997. Visceral fat in white and African American prepubetal children. *American journal of clinical nutrition,* 65:1703-1708.

GORTMAKER, S.L., MUST, A., PERRIN, J.M., SOBOL, A.M. & DIETZ, W.H. 1993. Social and economic consequences of overweight in adolescence and young adulthood. *New England journal of medicine,* 329:1008-1012.

GORTMAKER, S.L., DIETZ, W.H. Jr. & CHEUNG, L.W. 1990. Inactivity, diet and the fattening of America. *Journal of American Dietetic Association,* 90:1247-1255.

GUO, S.S. & CHUMLEA, W.C. 1999. Tracking of body mass index in children in relation to overweight in adulthood. *American journal of clinical nutrition,* 70 Supplement:145S-148S.

GUO, S.S., HUANG, C., MAYNARD, L.M., DEMERATH, E., TOWNE, B., CHUMLEA, W.C. & SIERVOGEL, R.M. 2000. Body mass index during childhood, adolescence and young adulthood in relation to adult overweight and adiposity: the Fels Longitudinal Study. *International journal of obesity,* 24:1628-1635.

HARRIS, N.S., CRAWFORD, P.B., YANGZOM, Y., PINZO, L., GYLATSEN, P. & HUDES, M. 2001. Nutritional and health status of Tibetan children living at high altitudes. *New England journal of medicine,* 344(5):341-347.

HEYMSFIELD, S.B., MCMANUS, C., STEVENS, V. & SMITH, J. 2000. Muscle mass; reliable indicator of protein energy malnutrition severity and outcome. *American journal of clinical nutrition,* 35:192-1199.

HEYWARD, V.H. & STOLARCZYK, L.M. 1996. *Applied body composition assessment.* Champaign: Human Kinetics. 221p.

HIMES, J.H. & DIETZ, W.H. 1994. Guidelines for overweight in adolescent preventive services: recommendations from an expert committee. *American journal of clinical nutrition,* 59:307-316.

HODGE, A.M. & ZIMMET, P.Z. 1994. The epidemiology of obesity. *Ballieres clinical endocrinological metabolism,* 8:577-599.

HOFFMAN, D.J., SAWAYA, A.L., VERRESCHUI, I., TUCKER, K.L. & ROBERTS, S.B. 2000a. Why are nutritionally stunted children at increased risk of obesity? Studies of metabolic rate of fat oxidation in shantytown children from Sao Paulo, Brazil.

American journal of clinical nutrition, 72:702-707.

HOFFMAN, D.J., SAWAYA, A.L., COWARD, W.A., WRIGHT, A., MARTINS, P.A., DE NASCIMENTO, C., TUCKER, K.L. & ROBERTS, S.B. 2000b. Energy expenditure of stunted and nonstunted boys and girls living in the shantytowns of Sao Paulo, Brazil. *American journal of clinical nutrition,* 72:1025-1031.

KUMANYIKA, S.K. 1999. Understanding ethnic differences in energy balance: Can we get there from here ? *American journal of clinical nutrition,* 70:1-2.

LABADARIOS, H., STEYN, N., MAUNDER, E., MACINTYRE, U., SWART, R., GERICKE, G., HUSKISSON, J., DANNHAUSER, A., VOSTER, H.H. & NESAMVUNI, A.E. 1999. The national food consumption survey (NFCS): Children aged 1-9 years, South Africa, 1999. p28-41.

LAKE, J.K., POWER, C. & COLE, T.J. 1997. Women's reproductive health: the role of body mass index in early and adult life. *International journal of obesity,* 21(6):432-438.

LAWLESS, J.W., LATHAM, M.C., STEPHENSON, L.S., KINOTI, S.N. & PERTET, A.M. 1994. Iron supplementation improves appetite and growth in anaemic Kenyan primary school children. *Journal of nutrition,* 124(5):645-654.

LEBOWITZ, S.F. 1992. Neurochemical-neuroendocrine systems in the brain controlling macronutrient intake and metabolism. *Trends in neurosciences,* 15:491-497.

LINHARES, E.D., ROUND, J.M. & JONES, D.A. 1986. Growth, bone maturation and biochemical changes in Brazilian children from two different socioeconomic groups. *American journal of clinical nutrition,* 44:552-558.

LISSAU-LUND, I. & SORENSEN, T.I.A. 1994. Parental neglect during childhood and increased risk of obesity in young adulthood. *Lancet,* 343:324-327.

LISSAU-LUND SORENSEN, I. & SORENSEN, T.I.A. 1992. Prospective study of the influence of social factors in childhood on the risk of overweight in young adulthood. *International journal of obesity,* 16:169-175.

LLOYD, J.K., WOLFF, M.A. & WHELEN, W.S. 1961. Childhood obesity: a longterm study of height and weight. *British medical journal,* 77(5):145-148.

LOCARD, E., MAMELLE, N., BILLETTE, A., MIGINIAC, M., MUNOZ, F. & REY, S. 1994. Risk factors of obesity in a five year old population. Parental versus environmental factors. *International journal of obesity,* 16:721-729.

LOVERIDGE, N. & NOBLE, B.S. 1994. Control of longitudinal growth: the role of nutrition. *European journal of clinical nutrition,* 48:75-84.

MALINA, R.M. & KATZMARYK, P.T. 1999. Validity of the body mass index as an indicator of the risk and presence of overweight in adolescents. *American journal of clinical nutrition,* 70 Supplement:131S-136S.

MARTORELL, R. 1996. The role of nutrition in economic development. *Nutrition reviews,* 54 Supplement:66S-71S.

MARTORELL, R., KHAN, L.K. & SCHROEDER, D.G. 1994. Reversibility of stunting: epidemiological finding in children from developing countries. *European journal of clinical nutrition,* 48(1)Supplement:S45-S57.

MARTORELL, R., MENDOZA, F. & CASTILLO, R.O. 1988. Poverty and stature in children. In: Linear growth retardation in less developed countries. New York: Raven Press. 57-74 p.

MARTORELL, R., MENDOZA, F.S., CASTILLO, R.O., PAWSON, I.G. & BUDGE, C.C. 1987. Short and plump physique of Mexican American children. *American journal of physical anthropology,* 87:475-487.

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.

MICHIELUTTE, R., DISKER, R.A., CORBETT, W.T., SCHEY, H.M. & UREDA, J.R. 1984. The relationship between weight-height indices and the triceps skinfold measure among children age 5-12. *American journal of public health,* 74:604-606.

MO-SUWAN, L., LEBEL, L., PUETPAIBOON, A. & JUNJANA, C. 1999. School performance and weight status of children and young adolescents in a transitional society in Thailand. *International journal of obesity,* 23:272-277.

MUSAIGER, A.O. & GREGORY, W.B. 2000. Profile of body composition of school children (6-18y) in Bahrain. *International journal of obesity,* 24:1093-1096.

MUST, A. 1999. Childhood energy intake and cancer mortality in adulthood. *Nutrition reviews,* 57:21-24.

MUST, A., DALLAL, G.E. & DIETZ, W.H. 1991. Reference data for obesity: 85th and 95th percentiles of body mass index (wt/ht^2) and triceps skinfold thickness. *American journal of clinical nutrition,* 53:839-846.

MUST, A., JACQUES, P.F., DALLAL C.J., BAJEMA G.E. & DIETZ, W.H. 1992. Longterm 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.

NAKAMURA, T., NISHIYAMA, S., FUTAGOISHI SUGINOHARA, Y., MATSUDA, I. & HIGASHI, A. 1994. Mild to moderate zinc deficiency in short children: effect of zinc supplementation on linear growth velocity. *Journal of pediatrics,* 124(1):163-164.

NORTON, K. & OLDS, T. 1996. Anthropometrica. A textbook of body measurement for sports and health courses. Sydney: University of New South Wales Press. 411p.

O'DEA, J. & ABRAHAMS, S. 1995. Should body mass index be used in young adolescents? *Lancet,* 345:657.

OWENS, S., GUTIN, B., FERGUSON, M., ALLISON, J., KARP, W. & LE, N.A. 1988. Visceral adipose tissue and cardiovascular risk factors in obese children. *Journal of pediatrics,* 133:41-45.

OWENS, S., LITAKER, M., ALLISON, J., RIGGS, S., FERGUSON, M., & GUTIN, B. 1999. Prediction of visceral adipose tissue from simple anthropometric measurements in youths with obesity. *Obesity research*, 7:16-22.

PENLAND, J.G., SANDSTEAD, H.H., ALCOCK, N.W., DAYAL, H.H., CHEN, X.C., LI, J.S., ZHAO, F. & YANG, J.J. 1997. A preliminary report: effects of zinc and micronutrient repletion on growth and neuropsychological function of urban Chinese children. *Journal of American colloquial nutrition* 16(3):268-272.

PERUSSE, L., CHAGON, Y.C., DIONNE, F.T. & BOUCHARD, C. 1996. The human obesity gene map: the 1996 update. *Obesity research,* 5:49-61.

POPKIN, B.M. 1994. The nutrition transition in low-income countries: an emerging crisis. *Nutrition reviews,* 52:285-295.

POPKIN, B.M. & BISGROVE, E.S. 1988. Urbanization and nutrition in low-income countries. *Food nutrition bulletin,* 10:3-23.

POPKIN, B.M. & DOAK, C.M. 1998. The obesity epidemic is a worldwide phenomenon. *Nutrition reviews,* 56(4):106-114.

POPKIN, B.M., RICHARDS, M.K. & MONTEIRO, C.A. 1996. Stunting is associated with overweight in children of four nations that are undergoing the nutrition transition. *Journal of nutrition,* 126:3009-3016.

POWER, C., LAKE, J.K. & COLE, T.J. 1997. Measurement and long-term health risks of child and adolescent fatness. *International journal of obesity,* 21:507-526.

POWER, C. & MOYNIHAN, C. 1998. Measurement and long-term health risks of child and adolescent fatness. *International journal of obesity,* 22:445-453.

PRASAD, A.S. 1996. Zinc deficiency in women, infants and children. *Journal of American colloquial nutrition,* 15(2):113-120.

PRENTICE, A.M. & JEBB, S.A. 1995. Obesity in Britain: gluttony or sloth ? *British medical journal,* 311:437-439.

REILLY, J.J., DOROSTY, A.R. & EMMETT, P. 2000. Identification of the obese child: adequacy of the body mass index for clinical practice and epidemiology. *International journal of obesity related metabolic disorders,* 24:1623-1627.

REVICKI, D.A. & ISRAEL, R.G. 1986. Relationship between body mass indices and measures of body adiposity. *American journal of public health,* 76:992-994.

ROBERTS, S.B. & DALLAL, G.E. 2001. The new childhood growth charts. *Nutrition reviews,* 59(2):31-37.

ROCHE, A.F., SIERVOGEL, R.M., CHUMLEA, C. & WEBB, P. 1981. Grading of body fatness from limited anthropometric data. *American journal of clinical nutrition,* 34:2831-2838.

ROSS, W.D. & MARFELL-JONES, M.J. 1991. Kinantropometry. *In* MacDougal, J.D., Wenger, H.A., Green, H.J., eds. Physiological testing of the high performance athlete. 2nd Edition. Champaign: Human kinetics. p.223-308.

ROSSOW, I. & RISE, J. 1994. Concordance of parental and adolescent health behaviours. *Social science medcine,* 38:1299-1305.

SAWAYA, A.L., DALLAL, G. & SOLYMOS, G. 1995. Obesity and malnutrition in a shantytown population in the city of Sao Paulo, Brazil. *Obesity research,* 3 Supplement:S107-S115.

SAWAYA, A.L., GRILLO, L.P., VERRESCHI, I., CARLOS DA SILVA, A. & ROBERTS, S.B. 1998. Mild stunting is associated with higher susceptibility to the effects of high-fat diets; studies in a shantytown population in the city of Sao Paulo, Brazil. *Journal of nutrition,* 128 Supplement:S415-S420.

SATYANARAYANNA, K., RADIHAIH, G., MURALI-MOHAN, R., THIMMAYAMMA, B.V.S., PRALHAD, R.N. & NARASINGA, R.B.S. 1989. The adolescent growth spurt of height among rural Indian boys in relation to childhood nutritional background: an 18 year longitudinal study. *Annals of human biology,* 16:289-300.

SCRIMSHAW, N.S. 1993. The challenge of global malnutrition to the food industry. *Food technology,* 60-71.

SEIDELL, J.C., CIGOLINI, M., DESLYPERE, J.P., CHARZEWSKA, K., ELLSINGER, B.M. & CRUZ, A. 1991. Body fat distribution in relation to physical activity and smoking habits in 38 year old European men. *American journal of epidemiology,* 133:257-265.

SHEAR, C.L., FREEDMAN, D.S., BURKE, G.L., HARSHA, D.W., WEBBER, L.S. & BERENSON, G.S. 1988. Secular trends of obesity in early life: The Bogalusa Heart Study. *American journal of public health,* 78:75-77.

STATISTICS SA. 1995. Living in North West. Selected findings of the 1995 October household survey. Pretoria: Statistics South Africa. p7.

STEPHENS, T., JACOBS, D.R. & WHITE, C.C. 1985. A descriptive epidemiology of leisure time physical activity. *Public health report,* 100:147-158.

STEYN, N.P., WICHT, C.L., ROSSOUW, J.E., VAN WYK KOTZE, T.J. & VAN ECK, M. 1989. Nutritional status of 11-year-old children in the Western Cape. II. Anthropometry. *The South African journal of food sciences and nutrition,* 54:21-27.

STRAUSS, R.S. & KNIGHT, J. 1999. Influence of the home environment on the development of obesity in children. *Pediatrics,* 103(6):1-8.

TANNER, J.M. 1955. Growth at adolescence. Oxford: Blackwell.

TANNER, J.M. 1981. Growth and maturation during adolescence. *Nutrition reviews,* 39:43-55.

THE NATIONAL HEART, LUNG AND BLOOD INSTITUTE GROWTH AND HEALTH STUDY RESEARCH GROUP. 1992. Obesity and cardiovascular disease risk factors in black and white girls; The NHLBI growth and health study. *American journal of public health,* 82(12):1613-1620.

THOMAS, J.R. & NELSON, J.K. 1996. Research methods in physical activity.

Champaign: Human Kinetics Publishers. p.485.

TOMKINS, A. 2000. Malnutrition, morbidity and mortality in children and their mothers. *Proceedings of the nutrition society,* 59:135-146.

TORUN, B. & VITERI, F.E. 1994. Influence of exercise on linear growth. *European journal of clinical nutrition,* 48(1)Supplement: S186-S190.

TREMBLAY, A., DESPRES, J-P., LEBLANC, C., CRAIG, C.L., FERRIS, B., STEPHENS, T. & BOUCHARD, C. 1990. Effect of intensity of physical activity on body fatness and fat distribution. *American journal of clinical nutrition,* 51:153-157.

TROIANO, R.P., FLEGAL, K.M., KUCZMARSKI, R.J., CAMPBELL, S.M. & JOHNSON, C.L. 1995. Overweight prevalence and trends for children and adolescents. The National Health and Nutrition Examination Surveys, 1963 to 1991. *Archives of pediatric adolescent medicine,* 149:1085-1091.

TROWBRIDGE, F.L., MARKS, J.S., ROMANA, G.L., MANDRID, S., BOUTTON, T.W. & KLEIN, P.D. 1987. 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. *American journal of clinical nutrition,* 45:513-525.

VILLALPANDO, S. & LOPEZ-ALARCON, M. 2000. Growth faltering is prevented by breastfeeding in underprivileged infants from Mexico City. *Journal of nutrition,* 130:546-552.

VON BRAUN, J., MCCOMB, J., FRED-MENSAH, B.K. & PANDYA-LORE J.M. 1993. Urban food insecurity and malnutrition in developing countries: trends, policies, and research implications. Washington: International Food Policy Research Institute.

WALKER, S.P., FORRESTER, T.E. & GRANTHAM-McGREGOR, S.M. 2000. Early linear growth retardation and later blood pressure. *European journal of clinical nutrition,* 54:563-567.

WANG, Y., GE, K. & POPKIN, B.M. 2000. Tracking of body mass index from childhood to adolescence: a 6-y follow up study in China. *American journal of clinical nutrition,* 72:1008-1024.

WATERLOW, J.C. 1994. Causes and mechanisms of linear growth retardation (stunting). *European journal of clinical nutrition,* 48(1)Supplement:S1-S4.

WATERLOW, J.C. 1992. Protein energy malnutrition. London: Edward Arnold. 407pp.

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

WHO. WORKING GROUP 1998. Use and interpretation of anthropometric indicators of nutritional status. *Bulletin of WHO,* 64:929-990.

WHO. 1995. The newborn infant. In: Physical status: the use and interpretation of anthropometry. *WHO technical report series,* 854:161-311.

WHO. 1997. Obesity: Preventing and managing the global epidemic. Geneva: World Health Organization.

WHO. Consultation on Obesity. 1998. Preventing and managing the global epidemic. Geneva: World Health Organization.

WINKLEBY, M.A., ROBINSON, T.N., SUNQUIST, J. & KRAEMER, H.C. 1999. Ethnic variation in cardiovascular disease risk factors among children and young adults. Findings from the Third National Health and Nutrition Examination Survey, 1988-1994. *JAMA,* 281:1006-1123.

WOLF, A.M., GORTMAKER, S.L., CHEUNG, L., GRAY, H., HERZOG, D.B. & COLDITZ, G.A. 1993. Activity, inactivity and obesity: racial, ethnic and age differences among schoolgirls. *American journal of public health,* 83(11):1625-1627.

ZHENG, B.J., LO, S.K.F. & TAM, J.S. 1989. Prospective study of community-

acquired rotavirus infection. *Journal of clinical microbiology,* 27:2083-2090.

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Chapter 7

Addenda

7.1 Addendum A: Thusa Bana Information Consent Form

THUSA BANA PROJECT: INFORMATION ON THE STUDY

THE PRPOJECT HAS BEEN APPROVED BY THE ETHICS COMMITTEE OF THE PU FOR CHE. ETHICS COMMITTEE NUMBER (XXXXXXXX)

I CONFIRM THAT:

It has been explained to me, that:

1. The purpose of the research study is to collect information on the problem of overweight and obesity among schoolchildren aged 10-15 years in the North West Province of South Africa.
2. I have been told that the researchers will obtain anthropometric variables of a random sample of children aged 10-15 years
3. The participant will be weighed and his/her height as well as circumferences and skinfolds of his/her arm 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.
4. Appropriate methodology to classify overweight and obesity in these age groups will be developed
5. The prevalence of obesity in children in the North West Province will be determined
6. The anthropometry of the different ethnic groups will be compared
7. The relationship between body mass index and adiposity in stunted children (low-height-for-age) will be determined
8. The role of dietary practices in the development of overweight and obesity will be determined
9. The role of physical activity levels and patterns in the development of obesity
10. Influences of ethnicity and urbanisation on the causative factors of overweight and obesity will be determined
11. Perceptions regarding overweight and obesity in these age groups will be measured
12. The general health status of obese children with controls, regarding absence from school due to illness will be compared
13. Guidelines for appropriate, culture sensitive, practical and sustainable intervention programmes for these age groups will be developed
14. I have also been told that this research is being done for the benefit of the children, and that 1200 children will take part in this study
15. 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 scientist
16. 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/confirmed at_____on_____2000

Witness_____

Participant's/representative of participant (parent)_____

7.2 Addendum B: Thusa Bana Control Card

THUSA BANA PROJECT

Subject name:_____No:_____Gender:____

_____

		CHECK CONTROL
STATION 1	RECRUITMENT DEMOGRAPHIC QUESTIONNAIRE	
STATION 2	BLOOD PRESSURE	
STATION 3	ANTHROPOMETRY	
STATION 4	ANTHROPOMETRY: CLOTHING	
STATION 5	PSYCHOLOGICAL QUESTIONNAIRE A	
STATION 6	DIETARY QUESTIONNAIRE:24 HOUR	
STATION 7	MOTOR DEVELOPMENT A	
STATION 8	PSYCHOLOGICAL QUESTIONNAIRE B	
STATION 9	EATING HABITS	
STATION 10	FAMILY CIRCUMSTANCES AND HIV TRM	
STATION 11	PHYSICAL ACTIVITY	
STATION 12	MOTOR DEVELOPMENT B	
STATION 0	BACK TO STATION 1	_____ SIGNATURE

7.3 Addendum C: Thusa Bana Demographic Questionnaire

Potchefstroomse Universiteit

vir Christelike Hoër Onderwys

1 Subject number
2 Date
3 Place and region
Interviewer
Home address

D	M	Y

4 Stratum of urbanisation:	Rural *	1
	Informal town	2
(Classify stratum of urbanisation)	Urban	3

*Rural = tribal land, farm schools; informal town = corrugated iron house; urban = formal town/city

5 Gender	Male	1
	Female	2

6 Age				
Date of birth (for control purposes, do not code)	D	M	Y	

7 First Language	Setswana	1
	Afrikaans	2
	English	3
	Xhosa	4
	Zulu	5
	Other:	6

8 Second Language	Setswana	1
	Afrikaans	2
	English	3
	Xhosa	4
	Zulu	5
	Other:	6

9 Do you receive treatment for any chronic disease?	Yes	1
	No	2
10 If yes - what disease?		
11 Girls only: Have you started menstruating (seeing periods) yet?	Yes 1	No 2
If yes, at what age (year)?		

12 Do you take snuff?	Yes	1
	No	2
13 Do you smoke?	Yes	1
	No	2
14 If no – have you smoked regularly before?	Yes	1
	No	2
15 If yes – what do you smoke?	Cigarettes	1
	Tobacco/pipe	2
	Other:	3
If other – describe		
16 How much do you smoke? per day		
per week		
17 For how long have you been smoking (years)		
18 Calculate pack years		

19 In which grade are you this year?	Grade 1-3	1
	Grade 4	2
	Grade 5	3
	Grade 6	4
	Grade 7	5
	Grade 8	6
	Grade 9	7

20 Does any member of your household have the Right to use any property (house/flat/room) as his/her own?	Yes	1
	No	2

21 What type of property?	

22 Please name the members of your household

Member	Age	Education (grade passed)	Present job

23 Who is the breadwinner in your home?	

24 Does he/she have a job at the moment?	Yes	1
	No	2

25 If yes – what kind of job?	Doctor/nurse/teacher/professional	1
	Business/taxi/self employed formal	2
	Typist/assistant/office work	3
	Domestic worker/garden/contract	4
	Hawker/car washer/informal sector	5

26 On which days of the week does he/she work?	Irregular (piece work)	1
	Part time (1-4 days/week)	2
	Full time (5-6 days/week)	3

27 Does someone in your household receive any additional pensions?	Yes	1
	No	2

28 What type of house do you live in?	Traditional hut	1
	Mokuku	2
	Brick house	3
	Other	4
Specify other		

29 Do you share a toilet with other households?	Yes	1
	No	2

30 What type of toilet do you have?	None	1
	Communal	2
	Bucket system	3
	Outside pit toilet	4
	Outside chemical	5
	Outside water flush	6
	Inside water flush	7

31 Where do you get your drinking water from?	Fountain, river	1
	Communal tap	2
	Tap on premises	3
	Tap in house	4
	Other	5
If other specify		

32 Do you have access to electricity inside your house?	Yes	1
	No	2

33 What type of stove do you have?	None	1
	Coal/wood	2
	Gas or parrafin	3
	Electric	4

34 What type of fridge do you have?	None	1
	Parraffin	2
	Gas	3
	Electric	4

35 Do you watch television every week?	Yes	1
	No	2

36 Do you listen to the radio every week?	Yes	1
	No	2

37 Do you have a computer in your home?	Yes	1
	No	2
38 If yes, do you play/work on the computer on most days?	Yes	1
	No	2
39 If yes, how many hours do you play/work on the computer on most days?	.....hour	

40 Does your school week include a physical activity/training period ?	Yes	1
	No	2

7.4 Addendum D: Thusa Bana Kinanthropometric Proforma

Kinantropometrie Proforma

Proefpersoon naam _____ Proefpersoon ID

Sport Posisie en/of item

Land/Provinsie/Skool/Span Geslag: M V

Geboortedatum (Jaar Maand Dag) Toetsdatum (Jaar Maand Dag)

Bokshoogte Kinantropometris ID

	ID	Veranderlike	Meting 1	Meting 2	Meting 3
Velvoue	1	Triseps [B]			
(mm)	2	Subskapulêre [B]			
	3	Bors [B]			
	4	Iliokristale [B]			
	5	Supra-spinale [B]			
	6	Abdominale [B]			
	7	Frontale dy [B]			
	8	Mediale kuit [B]			
	9	Mid-aksillêre [B]			
Omtrekke	10	Kop			
(cm)	11	Nek			
	12	Boarm (ontspanne) [B]			
	13	Boarm (gespanne) [B]			
	14	Voorarm (maks.)			
	15	Pols			
	16	Bors (mesosternale)			
	17	Abdomen (min.) [B]			
	18	Gluteale (heup) [B]			

	ID	Veranderlike	Meting 1				Meting 2				Meting 3			
	19	Dy (1) (1cm gluteale)			•				•				•	
	20	Dy (2) (mid.)			•				•				•	
	21	Kuit (maks.) [B]			•				•				•	
	22	Enkel (min.)			•				•				•	
Lengte- en	23	Akromiale-radiale			•				•				•	
hoogtemates	24	Radiale-stillion			•				•				•	
(cm)	25	Midstillion-daktillion			•				•				•	
	26	Iliospinale (b.ht)			•				•				•	
	27	Troganterion (b.ht)			•				•				•	
	28	Trogant-tib (lateraal)			•				•				•	
	29	Tibiale (lateraal)			•				•				•	
	30	Tib. med.-sfyrion tib.			•				•				•	
Deursneë	31	Biakromiale			•				•				•	
(cm)	32	Bi-iliocristale			•				•				•	
	33	Voetlengte			•				•				•	
	34	Sithoogte				•				•				•
	35	Bors (transversaal)			•				•				•	
	36	A-P borsdiepte			•				•				•	
	37	Humerus [B]			•				•				•	
	38	Femur [B]			•				•				•	
	39	Enkel			•				•				•	
	40	Gewrig (bi-stiloïed)			•				•				•	
	41	Hand			•				•				•	
	42	Liggaamsmassa [B]				•				•				•
	43	Liggaamslengte [B]				•				•				•
	44	Armspan				•				•				•
					•				•				•	

7.5 Addendum E: Reference Tables

Table 7.1: International cut-off points for BMI for overweight and obesity between 10-15 years

	Overweight BMI 25		Obese BMI 30	
Age	**Boys**	**Girls**	**Boys**	**Girls**
9.5	19.5	19.5	23.4	23.5
10	19.8	19.9	24.0	24.1
10.5	20.2	20.3	24.6	24.8
11	20.6	20.7	25.1	25.4
11.5	20.9	21.2	25.6	26.1
12	21.2	21.7	26.0	26.7
12.5	21.6	22.1	26.4	27.2
13	21.9	22.6	26.8	27.8
13.5	22.3	23.0	27.2	28.2
14	22.6	23.3	27.6	28.6
14.5	23.0	23.7	28.0	28.9
15	23.3	23.9	28.3	29.1
15.5	23.6	24.2	28.6	29.3

International cut-off points for BMI[1]

[1] Cole *et al.*, 2000

Table 7.2: Smoothed Percentiles of Stature (in cm) by sex and age: data and statistics from NCHS, 10 to 15 year old boys

Age	5th P	10th P	25th P	50th P	75th P	90th P	95th P
9.5	125.3	127.6	130.8	134.8	138.8	142.4	144.9
10	127.7	130.1	133.4	137.5	141.6	145.5	148.1
10.5	130.1	132.6	136.0	140.3	144.6	148.7	151.5
11	132.6	135.1	138.7	143.3	147.8	152.1	154.9
11.5	135.0	137.7	141.5	146.4	151.1	155.6	158.5
12	137.6	140.3	144.4	149.7	154.6	159.4	162.3
12.5	140.2	143.0	147.4	153.0	158.2	163.2	166.1
13	142.9	145.8	150.5	156.5	161.8	167.0	169.8
13.5	145.7	148.7	153.6	159.9	165.3	170.5	173.4
14	148.8	151.8	156.9	163.1	168.5	173.8	176.7
14.5	152.0	155.0	160.1	166.2	171.5	176.6	179.5
15	155.2	158.2	163.3	169.0	174.1	178.9	181.9
15.5	158.3	161.2	166.2	171.5	176.3	180.8	183.9

Percentiles of Stature from NCHS, 10 to 15 year old boys[2]

[2]Dibley, *et al.*, 1987

Table 7.3: Smoothed Percentiles of Stature (in cm) by sex and age: data and statistics from NCHS, 10 to 15 year old girls

Age	5th P	10th P	25th P	50th P	75th P	90th P	95th P
9.5	124.8	126.6	130.6	135.2	139.8	143.9	146.2
10	127.5	129.5	133.6	138.3	142.9	147.2	149.5
10.5	130.4	132.5	136.7	141.5	146.1	150.4	152.8
11	133.5	135.6	140.0	144.8	149.3	153.7	156.2
11.5	136.6	139.0	143.5	148.2	152.6	156.9	159.5
12	139.8	142.3	147.0	151.5	155.8	160.0	162.7
12.5	142.7	145.4	150.1	154.6	158.8	162.9	165.6
13	145.2	148.0	152.8	157.1	161.3	165.3	168.1
13.5	147.2	150.0	154.7	159.0	163.2	167.3	170.0
14	148.7	151.5	155.9	160.4	164.6	168.7	171.3
14.5	149.7	152.5	156.8	161.2	165.6	169.8	172.2
15	150.5	153.2	157.2	161.8	166.3	170.5	172.8
15.5	151.1	153.6	157.5	162.1	166.7	170.9	173.1

Percentiles of Stature from NCHS, 10 to 15 year old girls[3]

[3]Dibley, *et al.*, 1987

7.6 Addendum F: Abstract

THE ASSOCIATION BETWEEN STUNTING AND OVERWEIGHT AMONG 10-15 YEAR OLD CHILDREN IN THE NORTH WEST PROVINCE:

THE THUSA BANA STUDY

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Objective: To investigate the relationship between stunting and overweight among 10-15 year old children of the North West Province in South Africa.

Design: A single cross-sectional study design was used. The study formed part of the THUSA BANA project.

Subjects: The total study population of the THUSA BANA project comprised of 1257 randomly selected subjects, of which 604 were boys and 646 were girls, aged 10-15 years.

Measurements: Stunting was described as the height below the 5th percentile for age using the NCHS standard percentiles. Furthermore, the definition of overweight and obesity according to Cole standards were used, where the cut-off points for BMI corresponds with the adult BMI of 25 and 30 respectively. Anthropometrical variables namely triceps and subscapular skinfolds, waist circumference, weight, height and BMI of the 10-15 year old subjects were analysed.

Results: Stunting was most prevalent in the rural areas with 51 % of the stunted girls and 43 % of the stunted boys living in rural areas. The odds ratio and the 95 % CI for the association between stunting and overweight in boys and girls were 0.45(CI 0.16,1.30) and 0.50(CI 0.21, 1.19) respectively. BMI was greater in the 15 year old stunted girls (mean=21.21 kg/m2) than in the non-stunted girls (mean=20.32kg/m2). Furthermore, TSF+SSF was greater in the 15 year old stunted girls (mean=39.35mm) than in the non-stunted girls (mean=34.34mm).

Conclusion: There is no significant association between stunting and overweight in 10-15 year old children in the North West Province. However, there is a tendency for girls older than 14 years to start to gain subcutaneous fat, even though at these ages they are still stunted and underweight. This tendency in stunted girls is evident at the onset of menarche and could, however predict possible problems of overweight as they get older. In addition, the consequences of the nutrition transition could facilitate this process.