

**GROWTH SPURT IN HEIGHT AND WEIGHT OF CHILDREN IN
RURAL SOUTH AFRICA: THE CASE OF ELLISRAS
LONGITUDINAL STUDY**

by

CHRIS NEM BIDZANE

DISSERTATION

Submitted in (partial) fulfilment of the requirements for the degree of

MASTER OF SCIENCE

in

STATISTICS

in the

**FACULTY OF SCIENCE AND AGRICULTURE
(School of Mathematical and Computer Sciences)**

at the

UNIVERSITY OF LIMPOPO

SUPERVISOR: Prof. M Lesaoana

CO-SUPERVISOR: Prof. KD Monyeki

2018

Declaration

I declare that the dissertation hereby submitted to the University of Limpopo, for the degree of Master of Science in Statistics has not previously been submitted by me for a degree at this or any other university; that it is my work in design and in execution, and that all material contained herein has been duly acknowledged.

Surname, Initials (title)

Date

Abstract

The Ellisras Longitudinal Study (ELS) opened the possibilities for understanding the growth variations among children in rural South Africa. The aim of the study was to analyse the growth spurt in height and weight of children using the ELS. This is part of the on-going ELS and this study followed secondary analytical longitudinal study using data collected from November 1996 to November 2003. All children underwent a series of anthropometric measurements of height and weight according to the standard procedures recommended by the International Society for the Advancement of Kinanthropometry. The descriptive statistics was done for age, height, weight, velocity and acceleration by gender amongst rural children in Ellisras. The linear mixed model was used to analyse data. Based on the smallest values of AIC and BIC, the best model to fit the ELS data which was found to be the unstructured covariance structure model was chosen. The interaction between age and gender, which was significant at 5% level suggested that the relationship of age with growth varies depending on gender. There was also a significant positive linear relationship of age with distance.

The onset of growth spurt for rural children in Ellisras was at 12.05 years for boys and at 12.32 years for girls, while the Senegalese boys took off earlier at 11.02 years. Ellisras rural boys and USA boys had their onset of growth spurt almost at the same age at 13 years for USA boys and 12.05 years for Ellisras rural boys. USA girls had their onset of growth spurt earlier at 11 years than Ellisras rural girls at 12.32 years. Newcastle upon Tyne adolescents reached

their PHV at 14 and 12 years for boys and girls respectively, similarly with Ellisras rural children at 14.21 and 11.80 years for boys and girls respectively. Ellisras rural girls had their PHV at 11.80 years earlier than Ellisras rural boys at 14.21 years. Children in rural Ellisras in the ELS and their growth variations do not differ that much compared with other children across the world.

Key words: Growth spurt, peak height velocity, boys, girls.

Dedication

I dedicate this work to my loving parents, Azwindini Joseph Nembidzane and Muvhulawa Joyce Tshimenze for their unconditional love, care and support.

Acknowledgments

I would like to thank God for His unconditional love, guidance, wisdom and strength. I also wish to thank my supervisors: Prof ‘Maseka Lesaoana, Prof Kotsedi Daniel Monyeki and Dr Alexandra Boateng for their guidance, support and encouragement. Again, I specially wish to thank my parents, Azwindini Joseph Nembidzane and Muvhulawa Joyce Tshimenze for their unconditional love, care and support. My siblings, Lufuno, Ray, Ntsieni, Phathutshedzo Nembidzane and niece Makhado Ramabulana for their love and support. My friend Rose Matsie Selowa for her prayers, love, support and encouragement. All my relatives and friends who wish to see me succeeding.

The authors are indebted to the Ellisras Longitudinal Study administrators Mr TT Makata, Seleka SP, Seleka MS and Mr Makata W for coding the data.

The financial assistance of the National Research Foundation (NRF) towards this research is hereby acknowledged. Opinions expressed and conclusions arrived at, are those of the author and are not necessarily to be attributed to the NRF.

Contents

Declaration	i
Abstract	ii
Dedication	iv
Acknowledgments	v
Table of Contents	vi
List of Figures	ix
List of Tables	x
List of Abbreviations and Acronyms	xii
Research Output	xv
1 Introduction	1
1.1 Introduction	1
1.1.1 Problem statement	3
1.2 Purpose of the study	4
1.2.1 Aim	4
1.2.2 Objectives	4
1.3 Significance of the study	5

1.4	Scientific contribution	5
2	Literature review	6
2.1	Review on other studies	6
2.1.1	Europe	7
2.1.2	North America	25
2.1.3	South America	33
2.1.4	Asia	36
2.1.5	Africa	48
2.1.6	Other parts of the world	53
2.2	Chapter summary	59
3	Research methodology	60
3.1	Study design	60
3.2	Sample/Study population	60
3.2.1	Geographical area	60
3.2.2	Sample	61
3.2.3	Attrition rate	62
3.2.4	Anthropometry measurement	62
3.3	Quality control	62
3.4	Statistical analysis	63
3.4.1	Linear mixed models	64
4	Research analysis and results	67
4.1	Introduction	67
4.2	Descriptive statistics for age, height, weight, velocity, and accel- eration, by gender amongst Ellirras rural children from 1996 to 2003	67
4.3	Distance curve fitted to the data of boys and girls	70
4.3.1	Distance curve fitted to weight data for boys and girls	71

4.4	Velocity curves for boys and girls	72
4.4.1	Acceleration curves for boys and girls	73
4.5	Type III tests of fixed effects	74
4.6	Estimates of fixed effects	75
4.7	Estimates of covariance parameters	76
4.8	Pairwise comparison based on estimated marginal means	77
4.9	Information criteria for the model with the AR(1) covariance structure	78
4.10	Information criteria for the reduced model without the AR(1) covariance structure	78
4.11	Information criteria from the reduced model without the random age effects	79
4.12	Results of information criteria among three covariance structure models	79
5	Summary and conclusion	81
5.1	Introduction	81
5.2	Summary and conclusion	81
5.2.1	Major findings	82
5.2.2	Conclusion	85
5.3	Recommendations	85
5.3.1	Health care prospects	85
5.3.2	Care or prevention	86
5.3.3	Further studies	88
5.4	Ethical consideration	88

List of Figures

4.1	Distance curve for boys and girls	71
4.2	Distance curve fitted to weight data for boys and girls	72
4.3	Velocity curves for boys and girls	73
4.4	Acceleration curves for boys and girls	74

List of Tables

4.1	Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellisras rural children from 1996 to 1997	68
4.2	Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellisras rural children from 1998 to May 1999	69
4.3	Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellisras rural children from November 1999 to November 2000	69
4.4	Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellisras rural children from May 2000 to May 2003	69
4.5	Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellisras rural children November 2003	70
4.6	Type III tests of fixed effects	75
4.7	Estimates of fixed effects	75
4.8	Estimates of fixed effects	77
4.9	Pairwise comparison based on estimated marginal means	77
4.10	Information criteria for the model with the AR(1) covariance structure	78
4.11	Information criteria for the reduced model without the AR(1) covariance structure	79

4.12 Information criteria from the reduced model without the random age effects	79
4.13 Results of information criteria among three covariance structure models	80

List of Abbreviations and Acronyms

-2RLL	-2 Restricted Log Likelihood
AG	Artistic Gymnasts
AGA	Anti-Gliadin Antibodies
AH	Adult Height
AIC	Akaike's Information Criterion
AICC	Hurvich and Tsai's Criterion
AL	Axial Length
ANCOVA	Analysis of Covariance
APHV	Age at Peak Height Velocity
AR	Androgen Receptor
ATO	Age at Take-off
Bage	Biological age
BBD	Benign Breast Disease
BIC	Schwarz's Bayesian Criterion
BMI	Body Mass Index
BP	Blood Pressure
BTM	Beta-Thalassaemia Major
CAG	Cytosine-Adenine-Guanine
CAIC	Bozdogan's Criterion
CDC	Centre for Disease Control

CF	Cystic Fibrosis
CHD	Congenital Heart Defect
CHD	Coronary Heart Disease
CI	Confidence Interval
DF	Degrees of Freedom
DS	Down Syndrome
ED	Energy Density
ELS	Ellisras Longitudinal Study
FEV1	Forced Expiratory Volume in 1
FFMI	Fat-Free Mass Index
FH	Final Height
GH	Growth Hormone
GV	Growth Velocity
HC	Head Circumference
IGF-I	Insulin-like Growth Factor-I
IL-6	Interleukin 6
ISAK	International Society for the Advancement of Kinanthropometry
JIA	Juvenile Idiopathic Arthritis
KH	Kromeyer-Hauschild
KiGGS	German Health Interview and Examination survey for Children and - adolescents
LMM	Linear Mixed Model
M	Mean
MC4R	Melanocortin Receptor 4
NHANES	National Health and Nutritional Examination Survey
OGS	Onset of Pubertal Growth Spurt
OK	Optimal Kernel
OR	Odds Ratio
Ox	Oxandrolone

PCBs	Polychlorinated Biphenyls
PGS	Pubertal Growth Spurt
PHV	Peak Height Velocity
PI	Placebo
PS	Peak Spurt
RG	Rhythmic Gymnast
RSS	Russell-Silver Syndrome
SD	Standard Deviation
SDS	Shwachman-Diamond Syndrome
SDS	Standard Deviation Score
SE	Spherical Equivalent
SEM	Standard Error of the Mean
SGA	Small for Gestational Age
SPSS	Statistical Package for Social Sciences
Std Error	Standard Error
TEM	Technical Error of Measurement
TEQ	Total Toxic Equivalency
TS	Turner Syndrome
UAE	United Arab Emirates
UK	United Kingdom
UN	Unstructured
USA	United States of America
VLBW	Very Low Birth Weight
VTO	Velocity at Take-off
WHO	World Health Organisation

Research Output

The following section gives research output from this dissertation.

Peer Reviewed Journal Publication

1. Nembidzane, C., Lesaoana, M., Monyeki, KD. and Boateng, A. (2016). Growth spurt in height and weight of children in rural South Africa: Ellisras longitudinal study. *BMC Pediatrics* (Under review).

Chapter 1

Introduction



1.1 Introduction

Human growth, like most developmental processes, is complex. Human growth refers to the process from infancy to maturity that involves changes in body size and appearance. Growth is a typical characteristic of childhood. It is also a sensitive indicator of a child's nutritional status. Monitoring growth is therefore an important tool for assessing the health and well-being of children, especially in countries with limited access to diagnostic tools (Michaelsen, 2015). Tinggaard et al. (2014) stated that a child's growth is an indicator of health as well as a mirror of a society's general socioeconomic standard. They further indicated that monitoring of growth to identify health and nutrition related problems is an important tool for health care providers.

Cameron and Bogin (2012) said human growth and development are made and defined by the way in which human beings change in size, shape and maturity relative to the passage of time. Human physical growth in length and weight is

generally made by rapid growth in early life, followed by a general deceleration in childhood and then a marked increase in late childhood associated with the onset of puberty (Chirwa et al., 2014).

Growth rates of height and weight of children and adolescents are considered to be the best available dynamic indicators of health status of any given population (Satyanarayana et al., 1989). Height increments and height deficiencies are considered to reflect a long term picture of nutritional adequacy (Seoane and Latham, 1971). However, there is limited longitudinal data on growth of the same group of children during childhood, adolescence and adulthood from undernourished populations of developing countries (Alacevich and Tarozzi, 2016; Elusiyana et al., 2016; Rachmi et al., 2016; Wei and Crowne, 2016).

Growth spurts are a rapid rise in height and weight. They are more visible in the first year of life and around puberty, both periods when a tremendous amount of growth takes place in a short time. But growth spurts can occur other times too (Baby Center, 2016). Human growth in height is a multifaceted process including periods of accelerated and decelerated growth velocities (Widén et al., 2010).

Puberty is the gradual transition period between childhood and adulthood, during which growth spurt occurs and secondary pubertal characteristics appear. However, many factors may contribute to the onset of puberty (Felimban et al., 2013). For the reason that there are wide variations among individuals in the timing of the pubertal growth spurt, there is a wide range of physiologic variations in normal growth. In adolescence, there may be quite large deviations from the derived percentile lines, depending on the timing and tempo of the pubertal growth spurt. The timing and tempo of puberty vary widely, even among healthy children. In determining the appropriateness of a particular

growth velocity, the child's degree of biological maturation must be considered (Rogol et al., 2000).

1.1.1 Problem statement

The human adolescent growth spurt is the rapid and intense increase in the rate of growth in height and weight that occurs during the adolescent stage of the human life cycle (Bogin, 1999). The growth spurt, which is a notable feature of the human adolescent growth stage, but not the only defining characteristic, begins on average at 10 years for girls and 12 years for boys. However, there is considerable variation between individuals and populations. Bogin (1999) further explained that the intensity and duration of the spurt is, on average, greater for boys than for girls, and this accounts for the average sexual dimorphism of 11 – 13 cm in height between adult men and women. He further said that the adolescent spurt ends at about 18 – 19 years for girls and 20 – 22 years for boys. It is important to determine whether a similar case occurs among the Ellisras boys and girls, hence this study. The Ellisras Longitudinal Study (ELS) has been monitoring the children's growth in Ellisras, which plays an important part in assessing or understanding the health and well-being of these children. This information about children's growth in Ellisras communities will help in getting to understand the children's growth spurt as well as periods of accelerated and decelerated growth velocities. It is important and useful to health care providers.

Pan and Ratcliffe (1992) indicated that longitudinal growth data offer a unique opportunity for studying the dynamics of growth by analysing velocity as well as distance curves of dimensions, such as height. Such information is available in the ELS which was never reported for rural South African children in Ellisras, Limpopo Province. There is a need to use the ELS data to try and

understand or get insights about the growth variations which could be useful in understanding the health status of children. The purpose of the study is to investigate the growth variations among children in rural South Africa.

Berkey et al. (1983) stated that several authors have cited evidence for the occurrence of a growth spurt, several years before the adolescents spurt. The existence of such a spurt was noted by Tanner (1947), who labelled it mid-growth spurt. However, the evidence for a spurt in height was absent among their girls, and for their boys the spurt was only suggested by a diminution of deceleration from age six to seven years (Berkey et al., 1983). The analysis of the ELS data will shed some light on the growth spurt of these children.

1.2 Purpose of the study

1.2.1 Aim

The aim of the study is to analyse the growth spurt in height and weight among children in rural South Africa using the Ellisras Longitudinal Study.

1.2.2 Objectives

The specific objectives of the study are to:

1. Assess the onset and offset of growth spurt using Linear Mixed Models (LMMs).
2. Compare growth variations in height and weight for both boys and girls using descriptive statistics and LMMs.
3. Determine the growth rate in terms of velocity and acceleration in height and weight for both boys and girls during adolescent using LMMs.

1.3 Significance of the study

The growth process is the lawful, predictable product of a dynamic relationship between heredity and the environment. There are periods of accelerated and decelerated growth (e.g. during infancy growth is elevated, while the growth rate slows down during the preschool and elementary school years). Not all aspects of growth proceed at the same rate or at the same time. The rate and pattern of growth can be influenced by numerous variables such as nutrition, activity, rest, hormone activity, socioeconomic status, disease, climate, emotion and exercise. The growth of each individual is unique. The growth patterns of children in rural South Africa have not been adequately reported in the literature. The ELS offers opportunities to analyse velocity and curves for children in Ellisras – a rural area in Limpopo Province, South Africa.

1.4 Scientific contribution

This study will contribute to the scientific knowledge related to the health status of children in Ellisras and provide information in the analysis of longitudinal data in rural South Africa. The results of the study could be useful in understanding the health status of children among Ellisras communities and help the Limpopo Provincial Department of Health in developing policies on health status of children in rural South Africa.

Chapter 2

Literature review

Distance and velocity curves are commonly used to explain the growth rate of an individual or a group of individuals. Distance curves show the total distance the body has to travel along the road to maturity. On the other hand, in velocity curves, emphasis is placed on the plotting of the incremental growth within a specific time interval. The period of rapid growth in height that occurs at adolescence is unique to humans. The adolescent growth spurt begins on average two years earlier in girls than in boys, but within each gender there is enormous variation in the timing of pubertal development (Cole et al., 2015).

2.1 Review on other studies

A lot of research on child growth has been undertaken in different parts of the world, mostly in North America and Europe. A few studies have also been done in Africa, Asia, South America, as well as Australia and Oceania.

The literature has been divided according to the regions of the world.

2.1.1 Europe

In a multi-discipline longitudinal study undertaken by Kemper et al. (1985) on growth and health of teenagers, height velocity was measured four monthly during four consecutive years in a group of about 250 growing Dutch youngsters. From the individual velocity curves of height, Kemper et al. (1985) calculated PHV and the age at which PHV occurred. Height velocity was estimated in 102 boys from the derivative of a second degree moving polynomial function over five measurements. The authors found that peak height velocity (PHV) varied in boys between 6.1 and 14.0 cm/year. The median value of the age at PHV in boys is 14.0 years. Early-maturing boys have approximately 2 cm/year greater PHVs than late-maturing boys (Kemper et al., 1985).

Another work done by AberbergaAugskalne and Kemper (2007) investigated the cardiovascular system at rest and during bicycle exercise in relation to PHV. A total of 41 boys and 45 girls aged 7 – 16 were studied annually for 10 years. Their findings showed a mean PHV of 8.2 (2.2) cm/year in girls and of 9.7 (2.3) cm/year in boys. PHV occurred on the average at ages 12.2 in girls and at 13.7 in boys. According to age at PHV school children were divided into early, mid and late maturers. With increasing maturation there appeared a progressive increase in systolic and diastolic blood pressure and a decrease in heart rate, cardiac output per kg body weight, and peripheral resistance. Increase in systolic volume before PHV in girls and in PHV in boys resulted in more intensive increase in mean systolic ejection rate. They concluded that boys had higher values than girls for systolic volume, mean systolic ejection rate, and lower values than girls for cardiac output per kg body weight. Late maturers at every stage of growth and at PHV had more effective pattern of cardiovascular response to bicycle exercise when compared with mid/early maturers (AberbergaAugskalne and Kemper, 2007).

Leroy et al. (2015) indicated that the main objective of their study was to assess whether there is evidence of catch-up growth in children between two and five years old, when catch-up growth was defined as it was originally - as a reduction in the height deficiency (compared with standards) between two points in time. They used absolute height-for-age difference (child's height compared to standards, expressed in centimeters) and compared with findings using height-for-age Z-scores. Leroy et al. (2015) stressed that the rationale for this comparison is that height-for-age Z-scores, which is constructed from cross-sectional data, is used to assess children's attained height at a given age, but inappropriate to evaluate changes in height over time. In their results, they showed not only an absence of population-level catch-up growth between two and five years of age, but continued deterioration reflected in an increase height deficiency relative to growth standards. Based on their analyses of absolute deficiency in height from both cross-sectional and longitudinal data, they concluded that there is no linear catch-up growth in the data sets they analysed (Leroy et al., 2015). Their analyses using height-for-age difference found no catch-up growth in cohort studies and revealed substantial deterioration in height deficiency beyond two years of age, in both cohort and cross-sectional studies (Leroy et al., 2015).

Adolescence is a unique period in human development encompassing sexual maturation (puberty) and the physical and psychological transition into adulthood (Lewis et al., 2016). It is a crucial time for healthy development and adverse environmental conditions, poor nutrition, or chronic infection can alter the timing of these physical changes; delaying menarche in girls or the age of PHV in boys. Lewis et al. (2016) found that a total of 135 (22.2%) adolescents showed some delay in their pubertal development, and this lag increased with age. Of those with a chronic condition, 40.0% ($n = 24/60$) showed delay compared to only 20.3% ($n = 111/547$) of the non-pathology group. This difference

was statistically significant. A binary logistic regression model demonstrated a significant association between increasing delay in pubertal stage attainment with age in the pathology group. Lewis et al. (2016) concluded that it was for the first time that chronic conditions have been directly associated with a delay in maturation in the osteological record, using a new method to assess stages of puberty in skeletal remains.

Molinari et al. (1980) found a mid-growth spurt in height in approximately two-thirds of the children in their study, when each child's height velocity data were modelled by Stützle et al. (1980)'s shape-invariant model. They indicated that a small but significant mid-growth spurt was found in most height and length measurements. The mid-growth spurt seemed to be slightly more pronounced in boys than in girls. The peak of the mid-growth spurt occurred between 6.5 and 8.5 years. The height of the peak varied from 0.3 to 0.7 cm/year for the different measurements (estimated from the smoothed median velocity curves) (Molinari et al., 1980).

Pan and Ratcliffe (1992) studied the dynamics of child growth because they were interested in average velocity or acceleration curves based on 1-year intervals rather than instantaneous velocity or acceleration curves. They explored the VADK method of deriving velocity and acceleration curves. They explored the examples using longitudinal height data by residual analysis of the distance curves fitted by Optimal Kernel (OK). They also compared the velocity and acceleration curves fitted by VADK from distance curves with those by OK method calculated directly from the measurements. Using a sample of 60 children, Pan and Ratcliffe (1992) found that the shape of peak spurt (PS) using the VADK was close to that by the OK with specified bandwidth specified at about 2.5 years for velocity and 3.0 years for acceleration, which are on average about 0.8 years less than the optimal bandwidth.

Renes et al. (2015) stated that growth hormone (GM) treatment is effective in improving adult height (AH) in short children born with Small for Gestational Age (SGA). The authors found that during puberty, height Shwachman-Diamond Syndrome (SDS) declined (-0.4 SDS in boys and -0.5 SDS in girls) resulting in a lower total height gain SDS than expected. They also highlighted that pubertal height gain was 25.5 cm in boys and 15.3 cm in girls, significantly lower compared to Anti-Gliadin Antibodies (AGA) children ($p < 0.001$). Growth velocity was comparable to AGA children during the first two years of puberty, but thereafter significantly lower until reaching AH ($p < 0.001$) (Van Gameren-Oosterom et al., 2012).

In London schools with a high ethnic mix, Lum et al. (2015) found that the pattern of puberty differs between boys and girls. The size and lung function in children study took place from 2011 to 2013 and comprised children aged 5–11 years, with parental consent, from 14 London schools with a high ethnic mix. Their study was designed to explore ethnic differences in lung function and body physique in a multi-ethnic population of London school children. They collected both self-reports and parental reports of pubertal status in children aged 8 – 11 years, both to investigate the feasibility of assessing the attainment of secondary sex characteristics, as a proxy for pubertal status in this population, and explored any ethnic differences in rates of pubertal attainment. A wide range of physiological and anthropometric baseline assessments were undertaken in the schools, with assessments being repeated a year later whenever possible. Parents provided information about their own ethnicity, and their child's, by filling in a study questionnaire. This was used to broadly categorise children as: White (European ancestry), of Black African origin (Black African or Black Caribbean descent); of south Asian origin (from India, Pakistan, Bangladesh or Sri Lanka) and other/mixed ethnic cities. Socio-economic circumstances were measured using the Family Affluence Scale (Lum

et al., 2015). Menarche and voice breaking are both very late signs of puberty, and a child will have had some development for at least a year before these occur. The pattern of puberty differs between boys and girls. The pubertal growth spurt starts when breast development occurs in girls, but is delayed until mid-puberty in boys. They also found that boys are likely to have some pubertal development, such as hair and testicular growth, which no one than parents would notice prior to their pubertal growth spurt (Lum et al., 2015). Their findings suggested that parental reports of pubertal development may be preferable for large epidemiological studies when crude estimates of maturation are needed for children under 12 years of age, as these provided greater certainty than self-reports by children of this age (Lum et al., 2015).

In Sweden, Protudjer et al. (2015) stated that asthma may be associated with shorter height and delayed growth during adolescence. The aim of their study was the association between asthma and puberty in boys and menarche in girls, and height, in a cohort of twins and subsequently in same-sex twin pairs discordant for asthma. Parent and self-reported data on asthma, puberty/menarche, and height were collected. Pubertal staging was established via the Petersen index. Logistic and linear regression was used to estimate associations between asthma and puberty/menarche and height, respectively. For within-pair analyses in twins discordant for asthma, conditional logistic and linear regression were used. The authors found that among boys, asthma prevalence was 8.2% at 8.9 years and 10.2% at 13 – 14 years. Corresponding numbers for girls were 4.2% and 4.8% respectively. They further found that in the entire cohort, no statistically significant associations were found between current asthma and puberty/menarche. According to Protudjer et al. (2015) boys with asthma were shorter than boys without asthma at 8.9 years (on average, 1.86 [0.17 – 3.56] cm, $p = 0.03$) and at 13 – 14 years (on average, 2.94 [0.98 – 4.91] cm, $p = 0.003$), but not at 19 – 20 years. They did not find such associations for girls

(Protudjer et al., 2015).

Dalskov et al. (2015) indicated that earlier studies on seasonality in growth reported the largest height gains during spring and largest body weight gains during autumn. They found that average velocities showed that height was higher than the average (6.10 cm/year) and body weight was below the average (4.02 kg/year). Their findings suggested seasonality in growth and body composition of Danish children (Dalskov et al., 2015).

Van Gameren-Oosterom et al. (2012) found that the growth of healthy Dutch children with Down Syndrome (DS) and those who suffer from congenital heart defect (CHD) showed no difference in mean height standard deviation score (SDS) ($p = 0.832$) and head circumference (HC) SDS ($p = 0.790$). Both girls and boys with DS and severe CHD had significant lower mean height SDS and HC SDS compared to healthy children with DS or children with DS with mild CHD (both p -values < 0.001). Mean height was observed to be 0.4 SD lower. This growth retardation arises in the first year of life; during childhood no further deflection in growth was observed (Van Gameren-Oosterom et al., 2012).

Carrascosa et al. (2012) stated that longitudinal growth studies show that age at pubertal growth spurt onset occurs as a continuum and is sex dependent. They found that the onset of pubertal growth spurt began between the ages of 8 and 13 in girls and 10 and 15 in boys. In both sexes, the earlier start of pubertal growth spurt indicated high PHV and the total pubertal height gain. Carrascosa et al. (2012) also found that the height at onset of pubertal growth spurt was lower in the early than in the late maturity groups in Spanish boys and girls.

Georgopoulos et al. (2012) determined the impact of intensive training on adult

final height in elite female rhythmic gymnasts (RG) and artistic gymnasts (AG). Their findings showed that growth velocity SD score highest values were recorded at the age of 14 years for RG and at the age of 15 years for AG. They found that it was of particular interest that, although height velocity in normal girls comes to an end by the age of 15, in their examined RG and AG it continued up to the age of 18 years (Georgopoulos et al., 2012). The age of recalled menarche was similar in both groups: 15.6 ± 1.9 years for RG and 15.5 ± 1.6 years for AG. The results of their study clearly demonstrate and strengthen their preliminary findings (Georgopoulos et al., 1999; Georgopoulos et al., 2001) that female elite RG achieve normal final height in accordance with their genetic predisposition. In contrast, in female AG a slight impairment of growth potential, albeit within normal limits was observed (Georgopoulos et al., 2012).

Kryst et al. (2012) examined the secular changes in height, body weight, BMI and pubertal development in male children and adolescents in Krakow, Poland. The mean body height, in almost all age categories, was greater than in the past. However, the final height over the last decade remained the same. They also observed an acceleration of puberty in boys. The authors also noticed a distinct acceleration of puberty. Lack of height increase, at the same time as weight gain and puberty acceleration, indicate a progressing developmental disharmony (Kryst et al., 2012).

Schönbeck et al. (2012) stated that the records from the height data showed that the mean height in the Netherlands has increased since 1858. Their study looked at whether this trend in the world's tallest nation is continuing. Their analysis showed that height by age was the same as in 1997. Mean final height was 183.8 cm (SD=7.1 cm) in boys and 170.7 cm (SD=6.3 cm) in girls. Schönbeck et al. (2012) concluded that the world's tallest population has stopped growing taller after a period of 150 years. They further said that there

was a slight increase in mean height in early puberty in boys and girls, indicating that they were taller at a younger age than in 1997. However, this did not affect the final height, as no significant differences in final height were seen as compared with 1997 (Schönbeck et al., 2012).

Bournez et al. (2012) assessed the longitudinal growth pattern in a large French cohort of patients with cystic fibrosis (CF), to determine the extent to which puberty contributed to final height and to explore a potential relationship between growth, nutritional status and respiratory function. They found that in girls, height was similar to the reference population until age 11. Age at onset of puberty was the same as in reference girls. The pubertal spurt was lower than reference values and contributed less to the final adult height. In boys, the mean height was close to the reference mean until age 14 and was thereafter lower. Age at growth acceleration was similar to that in reference boys, but with an impaired PHV. The final height was lower than in the general population (z score -0.73). No correlations were found between BMI and PHV. In girls, there was a weak but significant positive relationship between PHV and forced expiratory volume in 1 (FEV1) ($r = 0.17$, $p = 0.02$). Bournez et al. (2012) concluded that children with CF had a normal age of onset of puberty and pubertal spurt with a low PHV. Puberty contributed less to final adult height and neither BMI nor FEV1 had a significant effect on pubertal height gain (Bournez et al., 2012).

Silventoinen et al. (2012) established that adult height is inversely associated with the risk of coronary heart disease (CHD), but it is still unknown which phase of the human growth period is critical for the formation of this association. In their results, Silventoinen et al. (2012) found that boys were nearly 1 cm taller than girls until age 11 at which the age difference in height disappeared and thereafter girls were slightly taller. Growth was most rapid be-

tween 11 and 12 years of age in girls (6.2 cm) and between 12 and 13 years of age in boys (5.8 cm). From 10 to 12 years of age, the SD was larger in girls than in boys whereas the opposite was true at age 13. A clear increase in the mean height across the birth years was observed. It was observed that risk of CHD in adulthood is inversely related to height at ages 7 through 13 years (Silventoinen et al., 2012).

Schaffrath et al. (2011) studied height-for-age percentiles representative for German infants, children and adolescents and compared them with older German height references by Kromeyer-Hauschild, which are based on heterogeneous pooled data (KH) and with international growth charts from the Centre for Disease Control (CDC) as well as the growth standard and the growth reference of the WHO. Their findings show that height for age percentile in German Health Interview and Examination Survey for Children and Adolescents (KiGGS) increase until age 16 years in girls and until the end of the observed age range (17.98 years) for boys. In general, boys are taller than girls, except for the age range 10.5 – 13.0 years. The difference in height between boys and girls is negligible before puberty and reaches 13 cm at age 17.98 years. KiGGS and KH percentile differ only slightly. However, there are substantial differences in SD-scores levels between KiGGS on the one hand, and WHO and CDC on the other hand, KiGGS generally being higher, especially in the extreme percentiles (Schaffrath et al., 2011). Schaffrath et al. (2011) recommended that the KiGGS height for age references be adopted as a national height reference for screening and monitoring growth in infants (starting from 4 months of age), children and adolescents in Germany.

Martinelli et al. (2011) studied melanocortin receptor 4 (MC4R) deficiency which is made by increased linear growth greater than the degree of obesity. They found that the mean birth weight (3.2 kg) and birth length (52 cm) of MC4R-

deficient children is in the UK population (Cole et al., 1995). When they examined growth charts in 43 MC4R-deficient children, they observed that growth velocity increases markedly in the first year of life, and the growth curve usually exceeds the 99th percentile by the age of 2 years. At all ages, Martinelli et al. (2011) found that the mean height SDS of MC4R-deficient children was significantly greater than equally obese children with normal MC4R genotype. Compared with the obese controls, the final height of male and female MC4R-deficient subjects was increased (mean standard error of the mean [SEM] 173 ± 2.5 vs. 168 ± 2.1 for males, $p < 0.001$; mean 165 ± 2.1 vs. 158 ± 1.9 for females, $p < 0.001$) (Martinelli et al., 2011). Argente et al. (1997) discussed that childhood obesity is often associated with increased linear growth, although the mechanisms linking energy balance and growth remain unclear.

Voorhoeve et al. (2011) investigated the relationship between the androgen receptor (AR) cytosine-adenine-guanine (CAG) repeat polymorphism and longitudinal growth, puberty and body composition from pre-puberty until young adult age. Voorhoeve et al. (2011) found that height SDS were inversely associated with AR CAG repeat length in boys at young, pre-pubertal and early pubertal age. This association diminishes in the following years and completely disappears after the age of 16 years. No associations were found with pubertal stage or any of the other parameters for body composition. They concluded that AR CAG repeat length is inversely associated with longitudinal height in young boys, before the onset of puberty. It was also noted that during puberty, these differences disappear, possibly overruled by a strong developing hypothalamic-pituitary-gonadal axis (Voorhoeve et al., 2011).

Günther et al. (2011) investigated the association of pre-pubertal dietary energy density (ED) with both age and body fatness at the start of the pubertal growth spurt (age at take-off, ATO). They found that ATO took place approx-

imately 1.5 years earlier in girls than in boys. ED was not related to later pubertal markers, that is age at PHV ($p = 0.5 - 0.8$) and age at menarche in girls ($p = 0.9 - 0.99$). Their results suggested that healthy children with higher pre-pubertal ED do not experience their pubertal growth spurt earlier (Günther et al., 2011).

Buyken et al. (2011) investigated whether the development of body composition into adolescence differs among children with an early, average, or late pubertal growth spurt ATO. They found that those girls with an early or average ATO experienced a significant increase in fat-free mass index (FFMI) z-scores [(standard error) for linear trends in early and average ATO group: +0.15(0.05) FFMI z-scores/year ($p = 0.001$) and +0.11(0.04) FFMI z-score/year ($p = 0.005$), respectively, adjusted for early life factors]. Similar differences were observed in boys [adjusted (standard error): +0.20(0.06) FFMI z-scores/year ($p = 0.0004$) and +0.07(0.05) FFMI z-scores/year ($p = 0.1$), respectively]. They concluded that their longitudinal study suggests that children who experience an early pubertal growth spurt accrue progressively more fat-free mass during the first years of puberty than late-maturing peers of the same age. Higher levels of adiposity commonly observed in adults with early puberty onset are thus, likely to develop subsequently in later adolescence (Buyken et al., 2011).

Tarım (2011) stated that hypothyroidism is a well-known cause of growth retardation. Height prognosis in children with late-diagnosed congenital hypothyroidism is guarded. Although treatment leads to an initial catch-up growth spurt, prolonged hypothyroidism may result in compromised adult height (Kandemir and Yordam, 2000). On the other hand, hyperthyroidism has been reported to accelerate growth in normal children and in patients with Turner Syndrome (Massa et al., 1992). However, whether this temporary growth spurt increases final height is not known (Tarım, 2011).

Wehkalampi et al. (2010) stated that subjects born preterm at very low birth weight (VLBW), as well as those with birth-weight-appropriate for gestational age, have advanced pubertal growth spurts compared with peers born at term. The authors opted to investigate the timing of pubertal growth in subjects born preterm with VLBW compared with controls. Their study followed subjects born preterm including 188 VLBW and 190 term-born control subjects. Growth data were available for 128 VLBW and 147 control subjects, of whom they excluded 15 and 1, respectively, because of neurological impairment. Timing of pubertal growth spurt was estimated by assessing the ages, corrected for gestational age at birth, at acceleration (take-off) and PHV of pubertal growth, and age at attaining adult height. Wehkalampi et al. (2010) found that all components of pubertal growth occurred earlier in VLBW subjects than in controls. Age at take-off was 0.8 years earlier (95% CI: 0.4 – 1.3) in those born small for gestational age. VLBW subjects were more likely to have a pubertal growth spurt that was at least two years earlier than population average [OR: 3.8 (95% CI: 1.5 – 9.6)]. They concluded that pre-maturing per se is associated with advanced pubertal growth. Advanced puberty may be among the factors mediating adult metabolic outcomes in subjects born preterm with VLBW (Wehkalampi et al., 2010).

Toumba et al. (2010) determined the beneficial effects of long-term growth hormone (GH) treatment on final height (FH) in 26 children with Russell-Silver Syndrome (RSS). Twenty-six patients (16 males) were diagnosed with RSS at a median age of 2.9 years according to clinical criteria. All patients were pre-pubertal at the commencement of treatment. They received treatment with biosynthetic human GH for 9.8 years (median) and all attained FH. They found that the median height at the commencement of treatment was -2.7 SDS and increased to -1.3 SDS ($p = 0.001$). Toumba et al. (2010) further indicated that a significant improvement of growth in RSS children has been shown after 10

years of GH treatment with a FH of -1.3 SDS. The shorter the patient at the start of the treatment, the greater the increment in FH. A significant response is also shown at the onset of puberty. GH treatment may also have a beneficial effect on the spinal length of RSS children (Toumba et al., 2010).

Günther et al. (2010) stated that early puberty onset is associated with hormone-related cancers, but whether diet in childhood influences pubertal timing is controversial. In their study, they examined the association of protein intake in early and mild-childhood with the ages at take-off of the pubertal growth spurt, peak height velocity, and menarche in girls and voice break in boys using data from the longitudinal Dortmund Nutritional and Anthropometric Longitudinally Designed study. They used life-course plots to identify critical periods of total, animal, and vegetable protein intake (percentage of total energy intake) for pubertal timing. The association between tertiles of protein intake (T1–T3) and the outcome was investigated using multiple linear regression analysis. Günther et al. (2010) found that a higher total and animal protein intake at 5 – 6 years was related to an earlier ATO. In height tertile of animal protein intake at 5 – 6 years, ATO occurred 0.6 years earlier than in the lowest [(mean, 95% CI) T1: 9.6, 9.4 – 9.9 vs. T2: 9.4, 9.1 – 9.7 vs. T3: 9.0, 8.7 – 9.3 years; p -trend =0.003, adjusted for sex, total energy, breast-feeding, birth year, and paternal university degree]. Similar findings were seen for APHV (p -trend =0.001) and the timing of menarche/voice break (p -trend =0.02). Conversely, a higher vegetable protein intake at 3 – 4 and 5 – 6 years was related to later ATO, APHV, and menarche/voice break (p -trend =0.02 – 0.04). These results suggest that animal and vegetable protein intake in mild-childhood might be differently related to pubertal timing (Günther et al., 2010).

Aksglaede et al. (2009) stated that recent studies have shown that puberty starts at younger ages than previously. It has been suggested that the increas-

ing prevalence of childhood obesity is contributing to this trend. Aksglaede et al. (2009) analysed the association between pre-pubertal BMI and pubertal timing, as assessed by age at onset of pubertal growth spurt (OGS) and at PHV, and the secular trend of pubertal timing given the pre-pubertal BMI. They found that in all birth cohorts in both girls and boys pre-pubertal BMI was significantly inversely associated with earlier age at OGS and PHV, although with some irregularity in boys from the earliest birth cohorts. The heavier at age 7 years the earlier did the children enter puberty whether assessed by OGS or PHV (Aksglaede et al., 2009).

Age at pubertal growth spurt (PGS) onset varies and is sex-dependent (Ferrandez et al., 2009). In their study, Ferrandez et al. (2009) presented anthropometric pubertal growth data for five 1-year interval age maturity groups: very early, early, intermediate, late and very late. They evaluated ages at PGS onset and adult height attainment, total pubertal growth, and PHV. PGS began between the ages of 10 and 15 in boys and eight to 13 in girls. Children were allocated to the corresponding one-year interval age maturity group. They found that for each sex, the earlier the start of PGS onset, the higher were PHV and total pubertal growth gain. However, adult heights were similar among the five pubertal maturing groups. Height SDS values for mean values of the very early, early, late, and very late maturing groups calculated according to data from the five pubertal maturing groups taken together as a single group differed from zero in both sexes, mainly during the pubertal years for the very early ($> +1$) and very late (> -1) maturers. These differences disappeared at adult height. Ferrandez et al. (2009) concluded that their data might contribute to better clinical evaluation of pubertal growth according to individual pubertal maturity tempo.

It is controversial whether pre-pubertal body composition is implicated in the

timing of puberty onset (Buyken et al., 2009). The objective of their study was to investigate whether body composition in the two years preceding the start of the pubertal growth spurt—a marker of puberty onset—is associated with the attainment of early and late pubertal markers in healthy German boys and girls. Buyken et al. (2009) found that higher BMIs and FM/ m^2 z scores 1 and 2 years before ATO showed modest associations with chronological age at ATO among girls only (girls: p for trend = 0.05 – 0.1, adjusted for early life factors; boys: p = 0.2 – 0.6). FFM/ m^2 z scores were not related to age at ATO (p for trend = 0.5 – 0.8). Conversely, pre-pubertal BMI and FM/ m^2 more clearly predicted APHV and puberty duration (APHV minus ATO) in both sexes and age at menarche in girls (girls: adjusted p for trend < 0.0001 – 0.03; boys: p = 0.01 – 0.046). Buyken et al. (2009) concluded that their longitudinal study suggests that pre-pubertal body composition in healthy boys and girls may not be critical for the initiation of the pubertal growth spurt but instead affects the progression of pubertal development, which results in earlier attainment of later pubertal stages.

Ratcliffe et al. (2009) said that the availability of a cohort of eight unselected XYY boys identified by newborn cytogenetic screening has enabled their growth to be studied longitudinally in comparison with controls from the same population. While no difference had been found in dimensions at birth, increased height velocity in childhood resulted in the XYY boys being 7.6 cm taller at the onset of their pubertal growth spurt. Increased intensity of growth at puberty with a PHV of 10.6 cm/year contributed to their adult height of 188.1 cm. XYY boys showed twice the male-female difference in height implying that genes on the Y chromosome exert a quantitative effect on the sexual dimorphism of growth (Ratcliffe et al., 2009).

Aks glaede et al. (2008) indicated that entering puberty is an important mile-

stone in reproductive life and secular changes in the timing of puberty may be an important indicator of the general reproductive health in a population. They further said that too early puberty is associated with several psychosocial and health problems. The aim of their study was to determine if the age at onset of pubertal growth spurt and PHV during puberty show secular trends during four decades in a large cohort of Danish school children. Aksglaede et al. (2008) found that in this period (year of birth 1930 to 1969), age at OGS declined statistically significantly by 0.2 and 0.4 years in girls and boys, respectively, whereas age at PHV declined statistically significantly by 0.5 and 0.3 years in girls and boys, respectively. The decline was non-linear with a levelling off in the children born between 1940 and 1955. The duration of puberty, as defined by the difference between age at OGS and age at PHV, increased in boys, whereas it decreased in girls. Their finding of declining age at OGS and at PHV indicates a secular trend towards earlier sexual maturation of Danish children born between 1930 and 1969. Only minor changes were observed in duration of puberty assessed by the difference in ages at OGS and PHV (Aksglaede et al., 2008).

Silventoinen et al. (2008) stated that previous studies have suggested that the timing of puberty is associated with BMI in childhood and adult stature. The genetic background of these associations is not thoroughly investigated; hence, they aimed to analyse it in a longitudinal twin cohort. Silventoinen et al. (2008) found that the heritability estimate was 0.91 for age at onset of pubertal growth spurt, 0.93 for age at PHV, and 0.97 for adult height. Age at onset of pubertal growth spurt was negatively associated with BMI from 1 to 10 years of age and stature in early adulthood. For age at PHV, they found similar associations with childhood BMI and stature in early adulthood. These associations were explained by common genetic factors. They concluded that growth during puberty is strictly genetically regulated. These genetic factors also explain why

boys who matured early had higher BMI through childhood and taller stature in early adulthood (Silventoinen et al., 2008).

Erlandson et al. (2008) compared the somatic growth, sexual maturation, and final adult height of elite adolescent female athletes. They found that gymnasts were significantly shorter than tennis players and swimmers at all chronological ages during adolescence, and they attained menarche at an older age ($p < 0.05$). No significant differences were found in adult heights. The results from their study suggests that regular training did not affect final adult stature and that, when aligned by biological age, the tempo of sexual maturation was similar in these young athletes (Erlandson et al., 2008).

Swenne (2008) investigated weight and growth requirements for menarche in girls with eating disorders, weight loss and primary amenorrhea. The author found that weight loss started at an age of 12.4 ± 1.6 years from a top weight of 41.7 ± 7.1 kg. Approximately a year later girls had lost 5.1 ± 4.3 kg and grown only 2.8 ± 3.5 cm. Following treatment and weight gain, growth accelerated and the girls reached a PHV of 4.3 ± 2.6 cm/year 2 years before menarche which occurred at an age of 15.5 ± 1.6 years at a weight of 52.2 ± 5.3 kg. Swenne (2008) further said that following treatment, girls with eating disorders and primary amenorrhea progress through puberty at a slowed rate. The weight required for menarche can be predicted by the pre-pubertal weight which may represent the individual's normal growth track unaffected by the eating disorders (Swenne, 2008).

Longitudinal changes in height, weight and physical performance were studied in 33 Flemish male youth soccer players who made the Ghent Youth Soccer Project (Philippaerts et al., 2006). The estimations of PHV, peak weight velocity and age at PHV were 9.7 ± 1.5 cm/year, 8.4 ± 3.0 kg/year and 13.8 ± 0.8 years,

respectively. Peak weight velocity occurred, on average, at the same age as PHV. A plateau in the velocity curves was observed after PHV for upper-body muscular endurance, explosive strength and running speed. Flexibility exhibited peak development during the year after PHV. Trainers and coaches were advised to be aware of the individual characteristics of the adolescent growth spurt and the training load should also be specified (Philippaerts et al., 2006).

The differences observed among racial and ethnic groups areas are attributed to the environmental factors such as nutrition and infections, as opposed to ethnicity and racial background (Amirhakimi, 2015). Growth curve or latent trajectory models have been used in the analysis of child growth over time, for example, to determine whether behaviour, height and/or weight differ for boys and girls (Fiona Steele, University of Bristol). Growth curve models postulate the existence of individual underlying trajectories, with changes over time providing information on these trajectories. Growth curves can be established based on longitudinal, cross-sectional, and mixed longitudinal and cross-sectional studies (Amirhakimi, 2015).

Cross-sectional studies make comparisons at a single point in time, while longitudinal studies make comparisons over time. Longitudinal studies, being the most difficult and time-consuming of these studies, are less frequently performed, and only a few are reported in the literature (Amirhakimi, 2015). On the other hand, cross-sectional studies are easier to perform, and in the developed countries where the influence of environmental factors are reduced to a minimum, could reliably be accepted as the standard, when performed on a large segment of population. On the contrary, this type of study, when performed in developing countries, where malnutrition and infection, the major determinants to preventing children to reach their growth potential, are still fairly common, should not be used as standard, although they constitute in-

formative and valuable data for future comparisons (Amirhakimi, 2015). Although critical for monitoring child growth development in developing countries, it is expensive to conduct such studies, hence few developing countries would be able to carry out such studies, even for limited time periods. South Africa is fortunate to have conducted the BT20 and ELS, among a few.

Relationships between patterns of growth in infancy and childhood and the risk of later chronic diseases such as obesity and cardiovascular diseases have been well described (Carles et al., 2016). Many studies are now focusing on the determinants of growth. They are mainly studied at one or few time points, but there is a need to analyse their association more globally with longitudinal pattern of growth of children for a better understanding of their consequences. The paucity of longitudinal studies and the lack of relevant methodologies do not allow the precise characterisation of the pattern of growth of children and the identification of when the difference in the body mass index (BMI) between different groups emerges (Carles et al., 2016).

2.1.2 North America

Rogol et al. (2000) highlighted that growth during childhood is a relatively stable process. The infancy shifts in the growth pattern are complete and the child follows the trajectory attained previously. In USA, until about the age of four years, girls grow slightly faster than boys and both sexes then average a rate of 5 – 6 cm/year and 2.5 kg/year until the onset of puberty (Rogol et al., 2000). The onset of puberty corresponds to a skeletal (biological) age of 11 years in girls and 13 years in boys. On average, girls enter and complete each stage of puberty earlier than boys.

At Harvard University's School of Public Health, a mid-childhood growth spurt in height was found in 17 of 67 units boys and 0 in 67 units girls followed from

age two years to adulthood. The growth spurt was detected analytically from variable knot cubic splines which were fitted to the longitudinal height data of each child at the Harvard University's School of Public Health (Berkey et al., 1983).

Nicholson et al. (2015) examined the ossification of the iliac apophysis and the timing of the peak height velocity (PHV). They followed longitudinally ninety-four healthy children (forty-nine girls and forty-five boys), from 3 – 18 years old through growth with annual serial radiographs and physical examinations. The PHV was calculated using the height measurements of each child. They measured and compared calcaneal and iliac crest apophyseal ossification using foot and pelvic radiographs made on the same day. They correlated the PHV with the degree of calcaneal and iliac ossification (Nicholson et al., 2015). They found that ossification of the calcaneal apophysis occurred in an orderly fashion, with the ossification centre first appearing a mean of 4.7 years (95% confidence interval [CI], 5.2 to 4.2 years) before the PHV. Iliac apophyseal ossification did not appear prior to the PHV in any subject (Nicholson et al., 2015).

Another study in the USA by Granados et al. (2015) examined the relationship between PHV and pubertal staging. Their study recruited a total of 1364 children whose birth was normal, uncomplicated and who were born to healthy mothers. Height and weight measurements were performed up to 11 times throughout their study, at ages 2, 3, 5, 7, 9 to 15 years. Since not all children were measured at all time points, they performed imputations using Stata statistical software. After imputing data, they excluded 616 subjects with more than 2 missing measurements after 6 years of age for girls and 8 years for boys. Additionally, they excluded 346 participants since negative height data were recorded in the imputed data. They included 402 subjects with available information on weight, height and pubertal staging. Menarche data was ob-

tained by maternal report via questionnaire. Height velocity was calculated as the increment in height divided by the difference in age between two consecutive measurements at their study visits. PHV was identified as the largest increase in height velocity after age 6 years for girls and after age 8 years for boys and the age corresponding to that PHV was tagged as age at PHV (Granados et al., 2015). They found that the mean age at PHV was 12.1 years (1.4 SD) for females and 13.7 years (1.4 SD) for males. PHV for females was 9.8 cm/year and for boys was 11.3 cm/year. They further found that the majority of girls (69.1%) had achieved PHV by tanner staging 3 and the majority of boys by tanner staging (58.9%). Additionally, they found that 70.6% of girls had attained their PHV by the time of menarche (Granados et al., 2015).

According to Gay et al. (2014) growth and maturation may impact adolescent behaviour and development of psychological disorders. Currently age at menarche is used as the primary marker of maturation, even though it occurs later than other indicators of growth such as PHV. Maturity offset predicting age at PHV has not been validated in diverse samples. Anthropometric measures and self-reported age at menarche were obtained for 212 female athletes aged 11 to 16 years (mean = 13.25). They reported that the shared variance between menarcheal age and estimated age at PHV was small ($R^2=5.3\%$). The Pearson's correlation between chronological age and age at PHV ($r = 0.69$) was stronger than with age at menarche ($r = 0.26$) (Gay et al., 2014).

Berkey et al. (2011) indicated that adult women with retrospective data and childhood adiposity were at risk of having benign breast disease (BBD) and or breast cancer. They found that girls with most rapid peak height growth ($\text{PHV} \geq 8.9$ cm/year) were at increased risk for biopsy-confirmed BBD (OR, 2.12; $p = 0.09$) relative to those whose peak growth was slowest. Girls growing most rapidly had significantly increased risk (OR, 1.88; $p = 0.04$). Berkey et al.

(2011) also found that girls with later age at menarche tend to have lower PHV ($r = -0.12$). PHV was the strongest predictor of biopsy-confirmed BBD (OR, 2.09 for fastest growing girls relative to slowest with $p = 0.09$, whereas for age at menarche, all $p > 0.16$). When they analysed all reported BBD cases, PHV remained the stronger predictor of BBD (OR, 1.84; $p < 0.05$, for fastest growing girls, whereas all $p > 0.64$ for age at menarche variables) (Berkey et al., 2011).

Baxter-Jones et al. (2011) in their study found that age and weight increased with increasing biological age (BAge), with females maturing earlier than males at PHV (BAge=0), the average age was $11.8(\pm 1.0)$ years in females and $13.5(\pm 1.0)$ years in males. Bone area and bone mineral content were measured from childhood to young adulthood at the total body, lumbar spine, total hip, and femoral neck. Bone area and bone mineral content values were expressed as a percentage of young-adult values to determine if and when values reached a plateau. Data were aligned on biological ages (years from PHV) to control for maturity. Significant differences were found between chronological age at each BAge between males and females ($p < 0.05$), reflecting the earlier maturational pattern of females. Stature increased significantly until three years after PHV ($p < 0.05$) for both males and females. A similar pattern was found for weight (Baxter-Jones et al., 2011).

Biro et al. (2010) examined longitudinal changes in waist-to-height ratio and components of BMI among young and adolescent girls of black and white race. They recruited girls at age nine through the National Heart, Lung, and Blood Institute Growth and Health Study and were followed annually over 10 years. Girls were grouped into low (<20th percentile), middle, and high (>80th percentile) BMI on the basis of race-specific BMI percentile rankings at age nine, and low, middle, and high waist-to-height ratio, on the basis of waist-to-height ratio at age 11. BMI was partitioned into fat mass index and fat-free mass

index. They found that girls accrued fat mass at a greater rate than fat-free mass, and the ratio of fat mass to fat-free mass increased from ages 9 through 18. There was a significant increase in this ratio after age at PHV. They concluded that in girls, higher BMI levels during childhood lead to greater waist-to-height ratios and greater than expected changes in BMI by age 18, with disproportionate increases in fat mass. These changes are especially evident in adolescent girls of black race and after the pubertal growth spurt (Biro et al., 2010).

Kuc-Michalska and Baccetti (2010) estimated and compared the duration of the pubertal growth peak in class I and class III subjects. They examined data consisted of pretreatment lateral cephalometric records of 218 skeletal Class I or Class III subjects (93 female and 125 male subjects) of white ancestry. The duration of the pubertal peak was calculated from the average chronological age intervals between stages of the cervical vertebral maturation in Class I vs Class III groups (t-test). They found that in skeletal class I subjects, the pubertal peak had a mean duration of 11 months, whereas in class III subjects it lasted 16 months. The average difference (5 months) was statistically significant. They further indicated that the growth interval corresponding to the pubertal spurt was longer in class III subjects than in subjects with normal skeletal relationships; the larger increases in mandibular length during the pubertal peak reported in the literature for class III subjects may be related to the longer duration of the pubertal peak (Kuc-Michalska and Baccetti, 2010).

Zeger et al. (2010) reported that untreated girls with Turner Syndrome have growth failure, and adult height is, on average, 20 cm less than predicted height. They investigated the benefit of adding oxandrolone (Ox) to GH in a long-term, randomized, placebo (Pl)-controlled prospective trial to near adult height in Turner Syndrome. 76 girls with Turner Syndrome (ages 10–14.9

years) were randomized to receive Ox (0.06 mg/kg/day) or Pl in combination with GH (0.35 mg/kg/week, daily) over two years. They measured Auxologic data, breast and pubic hair Tanner stages, and hormone and lipid levels. They followed subjects who chose to continue in a two-year double-blind extension, also received estrogen therapy (years three, four), and had dual-energy X-ray absorptiometry evaluation of bone density (years three, four). In their findings, Zeger et al. (2010) found that at year 4, the change in absolute height and height SDS was greater in the GH/Ox versus GH/Pi group [26.2 ± 6.7 vs. 22.2 ± 5.1 cm, analysis of covariance (ANCOVA), $p < 0.001$; 1.8 ± 0.9 vs. 1.2 ± 0.7 SDS, ANCOVA $p < 0.001$]. They further indicated that the addition of Ox to GH at mean age 12.0 ± 1.7 year augmented height gain after 4 years of treatment, slowed breast development and did not affect bone mineral density in girls with Turner Syndrome (Zeger et al., 2010).

Lee et al. (2010) examined the association between BMI and timing of pubertal onset in a population-based sample of US boys. They found that boys in the highest BMI trajectory (mean BMI z score at age 11.5 years, 1.84) had a greater relative risk of being pre-pubertal compared with boys in the lowest BMI trajectory (mean BMI z score at age 11.5 years, -0.76) (adjusted relative risk =2.63; 95% CI, 1.05 – 6.61; $p = 0.04$). The relationship between body fat and timing of pubertal onset is not the same in boys as it is in girls (Lee et al., 2010). The authors recommended further studies to better understand the physiological link between body fat and timing of pubertal onset in both sexes.

Blood Pressure (BP) and growth increase at an accelerated rate during puberty (Tu et al., 2009). The purpose of their investigation was to examine the rate of BP change in relation to pubertal growth with the intent to shed light on new mechanisms by which BP is regulated. Tu et al. (2009) found that average ages at PGS were 11.5 for girls and 13.3 for boys. Fitted spline models estimated

that at the time of PGS, the mean systolic BP was 100 mmHg for girls and 107 mmHg for boys; the mean diastolic BP at the PGS was 59 mmHg for girls and 61 mmHg for boys. The most intriguing observation was that rate of change in systolic BP and weight peaked at precisely the estimated PGS. Pubertal increases in growth and blood pressure peak at precisely the same age (Tu et al., 2009).

Rapid weight gain in the first years of life is associated with adult obesity (Botton et al., 2008). The objective of Botton et al. (2008)'s study was to study anthropometric measures in adolescence by sex according to weight and height growth velocities at different ages between birth and five years. They found weight growth velocity at three months was associated with overweight (OR for 1-SD increase: 1.52; 95% CI: 1.04, 2.22), fat mass, and waist circumference in adolescence in both sexes and with fat-free mass in boys ($r = 0.29$, $p < 0.001$) but not in girls ($r = -0.01$, NS). Weight growth velocities after two years were associated with all anthropometric measures in adolescence, in both sexes. Between six months and two years, weight growth velocities were significantly associated only with adolescent height in boys. In girls, associations with fat mass in adolescence were weaker. Their results support the hypothesis of two critical windows in early childhood associated with the later risk of obesity: up to six months and two years onward (Botton et al., 2008).

Several recent studies suggest that the timing of the onset of puberty in girls has become earlier over the past 30 years, and there is strong evidence that the increasing rates of obesity in children over the same time period is a major factor (Kaplowitz, 2008). The author reviewed studies from the United States that examined the age of menarche and the age of onset of breast development and pubic hair as a function of BMI, which is a good surrogate measure of body fat. Several studies show that girls who have relatively higher BMI are more

likely to have earlier menses, as well as a relationship between BMI and other measures of pubertal onset (Kaplowitz, 2008). The author further said that the evidence published to date suggests that obesity may be causally related to earlier puberty in girls rather than that earlier puberty causes an increase in body fat. In contrast, few studies have found a link between body fat and earlier puberty in boys (Kaplowitz, 2008).

Komlos and Breitfelder (2008) estimated differences in the trend and in the age-by-height profiles of United States born non-Hispanic black and white children and adolescents born 1942 – 2002. They found that the tempo of growth among blacks is faster than among whites. Black girls are more than 0.3 taller than white girls between the ages of 3 and 11. At age nine this amounts to some 2.7 cm. White boys catch up to black boys at age 14 and white girls catch up to black girls at age 15 and are taller thereafter. At age 19 whites are only slightly taller: $0.12\sigma(0.8cm)$ for boys and $0.03\sigma(0.2cm)$ among girls. The authors concluded that blacks have a faster tempo of linear growth in childhood partly on account of their nutritional habits, as girls in particular tend to have higher BMI values, and partly probably because of genetic differences (Komlos and Breitfelder, 2008).

Linear growth velocity provides one of the most important biomarkers of a child's health status and varies according to a variety of inherent and environmental factors including age, sex, genetics, pubertal development, nutrition, and psychosocial status (Kelly et al., 2014). Acceleration or deceleration of growth may result from systemic disease, poor nutrition, chronic illness, or endocrine dysfunction. Children who experience normal variants of pubertal maturation may come to medical attention due to their deviation from established height percentiles during early adolescence; this pattern is typical of children with pubertal delay who are plotted on cross-sectional growth charts.

Height velocity curves provide the clinician with an additional tool to distinguish between normal and abnormal variants of growth and pubertal maturation and may be used in the research setting to test the effect of an intervention on height velocity.

The mean PHV was 8.7 cm/yr in girls and 10.3 cm/yr in boys. Abbassi reviewed the growth characteristics of American boys and girls from published studies (Lee et al., 2004). Age at the onset of growth acceleration is highly variable and sex-dependent. The mean take off age in children growing at an average rate is 11 years in boys and 9 years in girls, and in these children, peak height velocities occur at a mean age of 13.5 years and 11.5 years, respectively. Whole year PHV is 9.5 cm/yr in boys and 8.3 cm/yr in girls, with slight variations in the different studies. In US cross-sectional studies, boys with a mean take off age of 11 years reached their final heights by 17 years of age, and girls with a mean take off age of 9 years reached their final heights by 14 years of age.

2.1.3 South America

Growth velocity patterns have the potential to signal unhealthy response to environmental insults with long-term consequences (Iannotti et al., 2015). They found that the mean individual child SD of weight velocity was 417g (± 126). In multivariate ordinary least squares regression analyses, growth velocities in month one and individual weight velocity variability positively predicted attained length and weight by 12 months. Panel regression by generalised least-squares with random effects of length and weight velocities confirmed the exponentially decelerating pace of growth through infancy and the importance of birth size in driving this trajectory (Iannotti et al., 2015).

In Brazil, Passos et al. (2015) recorded the variations in height of adolescents over three years and investigated their relationship with gender, age, sex-

ual maturation, and nutritional indicators. They found the mean heights at the first, second and third year follow-up evaluations were 152.02 ± 9.26 cm, 156.75 ± 8.61 cm, and 160.13 ± 8.14 cm, respectively. The mean height gain from baseline to the end of the study was 6.74 cm among girls and 9.73 among boys. The mean growth velocity (GV) was higher among 10-year-old underweight, prepubescent boys (5.4 cm/year). Furthermore, gender (girls: odds ratio [OR] = 5.97; 95% CI = 3.36 – 10.59), age (older youths: OR = 2.43; 95% CI = 1.84 – 3.21), and body weight (OR = 1.05; 95% CI = 1.03 – 1.07) were associated with lower GVs (Passos et al., 2015).

Souza et al. (2008) evaluated associations of growth velocity with inflammatory markers and cumulative dose of glucocorticoid in a cohort of patients with juvenile idiopathic arthritis (JIA) followed during one year. The authors found that the prevalence of low growth velocity was 25.3 percent, and it was associated with active disease on follow up visit, elevated interleukin 6 (IL-6), erythrocyte sedimentation rate and C-reactive protein, and higher cumulative glucocorticoid doses. In the multiple linear regression with growth velocity as the dependent variable, only elevated IL-6 level was independently and negatively associated with growth velocity (Souza et al., 2008). Low growth velocity is highly prevalent in children with JIA. Elevated IL-6 levels seem to have an important negative influence on growth in children, while total glucocorticoid exposure appears to be a secondary factor (Souza et al., 2008).

Growth velocity may be defined as the rate of change in physical size over a specified time interval (Iannotti et al., 2015). Patterns of growth velocity may be interpreted as an early signal of healthy or unhealthy responses to environmental conditions. In recent years, growth velocity has been studied during childhood in relation to later obesity outcomes (Monteiro et al., 2003; Botton et al., 2008). Their study investigated velocity patterns in a context of under-

nutrition, previously studied in Peru and other developing countries (Iannotti et al., 2015).

Growth velocity patterns are thought to arise from evolutionary processes preserving survival and reproduction. The tremendous plasticity of growth in the short term also seems to indicate adaptive responses to the environment and survival mechanisms. Growth retardation that may be associated with infection, for example, is generally followed by periods of rapid growth. The alternating periods of deceleration and acceleration return the child to its original growth trajectory, a process referred to as canalisation (Iannotti et al., 2015). Considerable evidence supports the health and developmental advantages of early growth during the first two years of life, though the pace of growth within particular periods of this time has been studied to a lesser extent (Iannotti et al., 2015). Understanding growth responses during critical periods may allow for early intervention to protect later health and development.

Understanding variation in human growth and development has long been a primary objective in the fields of human biology and public health (Urlacher et al., 2016). Although operating under occasionally conflicting research, both fields continue to emphasise the importance of obtaining descriptions of growth from diverse human populations to realise existing research goals (Urlacher et al., 2016). Despite this agreement, small-scale indigenous populations, comprising a significant portion of human genetic and cultural diversity (Henn et al., 2011; Karafet et al., 2002; Kent, 1996; Wang et al., 2007), remain greatly under represented in the literature on childhood growth. The little data available from these groups are notable, however, in that they suggest large variation in body size between populations and patterns of growth and development that frequently differ from those of Western children (Eveleth and Tanner, 1991; Ulijaszek, 1995; Walker et al., 2006). This observation underscores

the diversity and complexity of physical development in challenging environments (Cameron, 2007) and highlights the central importance of obtaining new descriptions of growth among small-scale populations to better understand human biological variation, phenotypic plasticity, and health.

2.1.4 Asia

Few studies in low-income settings analysed linear growth trajectories from foetal life to pre-adolescence (Svefors et al., 2016). The aim of their study was to describe linear growth and stunting from birth to 10 years in rural Bangladesh and to analyse whether maternal and environmental determinants at conception are associated with linear growth throughout childhood and stunting at 10 years. Svefors et al. (2016) used logistic regression analysis to investigate the associations between baseline predictors and stunting (height-for-age Z-scores < -2) at 10 years. They found that height-for-age Z-scores decreased to two years, followed by an increase up to 10 years, while the average height-for-age difference in cm to the World Health Organisation (WHO) reference median continued to increase up to 10 years. Maternal height, maternal educational level and season of conception were all independent predictors of height-for-age Z-scores from birth to pre-adolescence ($p < 0.001$) and stunting at 10 years. The prevalence of stunting was highest at two years (50%) decreasing to 29% at 10 years. They concluded that height growth trajectories and prevalence of stunting in pre-adolescence showed strong intergeneration associations, social differentials, and environmental influence from foetal life. They indicated that targeting women before and during pregnancy is needed for the prevention of impaired child growth (Svefors et al., 2016).

China's unprecedented internal migration has left 61 million rural children living apart from parents (Zhang et al., 2015). Their study investigated how being left behind is associated with children's growth, by examining children's height

and weight trajectories by age, testing the accumulation and critical period life course hypotheses (Zhang et al., 2015). Their study used growth curve models to investigate how being left behind is associated with children's growth by examining children's height and weight trajectories at different stages of their lives. They define left behind children as those whose parents had left the home to seek employment elsewhere. Growth curve models are appropriate for modelling differences in growth or change over time on data with repeated measurements within participants. These models take into account the dependence of residuals due to covariances between the levels in the data. If the interdependence of residuals is ignored, standard errors are underestimated, leading to biased estimated (Zhang et al., 2015). Their results showed that boys who were left behind at different life stages of childhood differed in height and weight growth compared with boys from intact families. No significant associations were found for girls. As young boys turned into adolescents, those left behind in early childhood tended to have slower height growth and weight gain than their peers from intact households. They also found that there was a 2.8 cm difference in the predicted heights of boys who were left behind in early childhood compared to boys from intact households, by the age of 14. Similarly, the difference in weight between the two groups of boys was 5.3 kg by the age of 14 (Zhang et al., 2015). They concluded that being left behind during early childhood, as compared to not being left behind, could lead to slower growth rates of height and weight for boys. The life course approach adopted in their study suggested that early childhood is a critical period of children's growth in later life, especially for boys who are left behind (Zhang et al., 2015).

Satyanarayana et al. (1980) studied that the growth pattern of 677 rural Hyderabad Indian boys aged between 13 and 18 years over a year (1977 – 1978) to obtain annual increments. These authors found that the group that had experienced severe growth retardation in early life achieved a peak height velocity

(6.9 cm/year) similar to that of British boys (7.3 cm/year). However, increase in weight was lower (29 kg) than in Western boys (44 to 48 kg). Height deficits observed at age five continued into adolescence and weight deficits increased. They also indicated that as a consequence, the group that was at age five continued to be shorter and lighter at 17 – 18 years.

The United Arab Emirates (UAE) is a mixed society with UAE nationals (Emiratis) and nationals of 120 other countries comprising a total estimated population in 2011 of 8 264 070, of which around one million were Emiratis (Aburawi et al., 2015). The authors further said that growth is one of the best markers of health status both individually and for the population. Growth velocity of children with Down Syndrome (DS) has been known to decelerate during the period from six months to three years and at puberty. They found that, like in Saudi Arabia growth rate among children with DS differed significantly from that in normal children and heights were significantly shorter than in the normal population at most ages (Aburawi et al., 2015). They also found that when the children with DS reached three years of age, the z-score was below -2.0, and this trend continued throughout the period during which measurements were made, with mean z-scores remaining below -2.0 in comparison with the normal population (Aburawi et al., 2015). A similar trend was noted in the Netherlands in comparison with the general population growth standards. Mean height of children with DS in the Netherlands was at -1.1 Standard Deviation (SD), decreasing to -2.2 SD at three years and remaining at this level until puberty, when the mean height achieved was at -2.9 SD (Van Gameren-Oosterom et al., 2012).

Bong et al. (2015b) proposed the reference curves for height and weight for children in the Kuching area, Sarawak. They found that the height of school boys and girls were almost similar at the start of their school going age. For

school girls, height and weight values were almost equal when they reached 16 or 17 years old but kept increasing for school boys (Bong et al., 2015b). They also found that school boys were taller than school girls as they entered adolescence. Height differences between school boys and girls became significantly wider as they grew older. Chinese school children were taller and heavier than those of other ethnic groups (Bong et al., 2015b).

Soliman et al. (2015) said that growth and maturational delay are striking features of beta-thalassaemia major (BTM). After the age of four years, growth faltering sets in but becomes clear after the age of eight years that involves stature, sitting height, weight and skeletal maturation. They also said that at this age, a slowing down of growth and a reduced or absent pubertal growth spurt are observed. There is marked attenuation or loss of pubertal growth spurt and the growth plate fusion is usually delayed until the end of the second decade of life (Soliman et al., 2015). They found that during childhood, Insulin-like growth factor-I (IGF-I) increases progressively and reaches the maximum during puberty (between 12 and 16 years) corresponding to the pubertal growth spurt, and then decreases gradually with age (Soliman et al., 2015).

Children with Turner Syndrome (TS) have a specific growth pattern that is quite different from that of healthy children (Darendeliler et al., 2015). According to Darendeliler et al. (2015) all the mean height values of Turkish girls with TS patients were lower in comparison to the mean height values of healthy Turkish girls. The mean height values from three years of age were lower than that of the general population and got lower by age. From the age of 13, Darendeliler et al. (2015) observed that there was an increase of approximately 2.5 – 3.0 cm per year in height without pubertal peak.

Bong et al. (2015a) determined the growth centiles of Malaysian children and

established contemporary cross-sectional growth reference charts for height and weight from birth to six years of age, based on a representative sample of children from Malaysia. The authors found that boys were taller and heavier than girls. The median length of Malaysian children was higher than the WHO 2006 standards. The overall prevalence of stunting and underweight were 8.3% and 9.3% respectively (Bong et al., 2015a).

Bahathiq and Elawad (2014) studied the age of onset of menarche among the female students at intermediate and secondary schools at Makkah Al-Mukarramah district and the relation of menarcheal age to anthropometric measurement represented by weight, height and body mass index (BMI). Bahathiq and Elawad (2014) found that the land marks of pubertal events in girls are the onset of puberty, PHV and menarche. They also explained that the onset of puberty is marked by the development of breast tissue, while PHV is the highest velocity that is observed during the pubertal growth spurt. Furthermore, menarche is a milestone in a women's life as it denotes the start of reproductive capacity. Bahathiq and Elawad (2014) found that the mean age in their study is (12.6 ± 1.1 years) which is consistent with the strong evidence of a downward secular trend in age at menarche in Europe and USA during the last century, and Japan and China during the past few decades (Hwang et al., 2003). It was also lower than African mean menarcheal age reported (Pasquet et al., 1999).

Yip et al. (2012) determined the relationship between puberty and growth spurts with peak spherical equivalent (SE) or axial length (AL) velocity in Singapore school children. It was found that the age of PHV occurred earlier in girls than in boys (11.0 ± 1.2 vs. 12.0 ± 1.7 years, $p < 0.001$). Girls with earlier PHV experienced peak AL velocity and peak SE velocity approximately half a year earlier than those with later puberty (mean age of 10.3 ± 1.6 vs. 10.8 ± 1.7 years, $p < 0.001$; and 10.0 ± 1.5 vs. 10.6 ± 1.25 years, $p < 0.001$, respectively).

Similarly, boys who had earlier PHV also achieved peak AL and peak SE velocity earlier than those who experienced later PHV (mean age of 10.4 ± 1.6 vs. 11.1 ± 1.8 years, $p < 0.001$; and 10.1 ± 1.5 vs. 10.6 ± 1.7 years, $p = 0.01$). Both girls and boys who had early PHV had earlier age of onset of myopia than those with later PHV (9.7 ± 1.4 vs. 10.1 ± 1.5 years for girls, $p = 0.04$; and 9.9 ± 1.5 vs. 10.4 ± 1.6 years for boys, $p = 0.03$). The associations were not significant when tanner staging and age of menarche was used to determine the stage of puberty. Yip et al. (2012) concluded that boys and girls with earlier PHV experienced earlier peak SE and AL velocity, and age of myopia onset. Thus, variations in the onset and peak progression of myopia may be associated with height spurts. They further discussed that Singaporean children aged 5 to 14 years with earlier growth spurts as defined by age of PHV had myopia at an earlier age and were found to have earlier peak AL and SE velocity for all children and myopic children (Yip et al., 2012).

The age at menarche reflects a pubertal girl's physiologic maturity (Lai et al., 2008). The authors evaluated the relationship between the age at menarche and skeletal maturation in female orthodontic patients. Lai et al. (2008) found that more than 90% of the 148 subjects who had already attained menstruation had skeletal maturation beyond the National Taiwan University Hospital Skeletal Maturation Index Stage four or Cervical Vertebral Maturation Stage III. The mean age at menarche for the 167 female patients in total was 11.097 years. Menarche occurred between National Taiwan University Hospital Skeletal Maturation Index Stages four and five or between Cervical Vertebral Maturation Stages III and IV. The percentage of girls with menses increased from 1.2% at age 9 to 6.6% at age 10, 39.5% at age 11, 81.4% at age 12, 97% at age 13, and 100% at age 14. Compared with the results obtained 20 years previously, they found a downward shift of 0.47 years per decade for the mean age at menarche in female orthodontic patients. The authors concluded that the

majority of female orthodontic patients have passed the pubertal growth spurt when they experience their menarche. Menarche usually follows the pubertal growth spurt by 1 year and occurs after National Taiwan University Hospital Skeletal Maturation Index Stage four or Cervical Vertebral Maturation Stage III (Lai et al., 2008).

Zhu et al. (2008) examined the rate of growth and bone mineral accretion and the predictors of total body bone mineral calcium during puberty in a 5 year longitudinal study with Chinese girls. It was found that the mean age of menarche was 12.1 ± 1.0 year. The mean annual rate of bone mineral accretion was 197.7 g/year during the follow-up period, representing a calcium accretion rate of 162.3 mg/d. PHV occurred 1 year later than PHV. In the linear mixed-effects model analysis containing body size and lifestyle factors, they found that height, body weight, and calcium intake were significant independent predictors of total body bone mineral calcium. They further indicated that Chinese girls with low habitual dietary calcium intake have high calcium retention efficiency during puberty (Zhu et al., 2008).

Kim et al. (2008) assessed the secular growth changes in Korean children and adolescents during the last four decades. The authors compared the results of previous nationwide growth studies with their study. The results of their study indicated that the growth and developmental status of Korean children and adolescents has changed substantially compared with those in 1965, 1975, 1984, and 1997. The data presented in their study show a distinct secular increase in growth in body height and weight of Korean children and adolescents spanning this period. They concluded that a nationwide survey every five years would be beneficial to establish a reference standard for the growth of children and adolescents according to the socioeconomic, environmental, and nutritional changes (Kim et al., 2008).

The aim of Bundak et al. (2007)'s study was to provide normative data for the onset and tempo of puberty in healthy Turkish boys. The mean age and height at onset of puberty were 11.6 ± 1.2 years and 146.1 ± 7.7 cm, respectively. Height velocity was 10.1 ± 1.6 cm. Total pubertal height gain was 26.4 ± 4.3 cm. The duration of puberty was 4.9 ± 0.6 years. Height at onset of puberty was positively correlated with final height ($p < 0.0001$) and with duration of puberty ($p = 0.03$). BMI at onset of puberty correlated negatively with age at onset of puberty ($p < 0.009$) and with the duration of puberty ($p = 0.05$) but not with final height. Bundak et al. (2007) concluded that the results of their study provide normative data for height and height velocity for each testicular volume stage for boys in puberty. Height at onset of puberty is the most important determinant of final height. There is no secular trend for the onset of puberty. Weight does seem to affect the onset and tempo of puberty but not final height (Bundak et al., 2007).

The assessment of growth by objective anthropometric methods (weight, length/height and body mass index) is crucial in child care to assess the nutritional status and for the identification of growth failure (Khadilkar et al., 2009). Reference data are central to growth monitoring and they help doctors and policymakers to diagnose under nutrition, overweight and obesity, and other growth-related conditions. The pattern of growth of a population of any age changes with time and hence it is recommended that references should be updated regularly. The 1977 National Center for Health Statistics growth curves for US children were revised in 2000, while the UK curves, first published in 1966, were revised in 1990 (Khadilkar et al., 2009). Nationwide growth surveys have been performed every 10 years in Mainland China since 1975 (Khadilkar et al., 2009). Reference values for children in Hong Kong first published in the 1960s, were updated in 1985, and were updated again in 1993 (Khadilkar et al., 2009).

India is in a phase of nutritional transition and thus it is vital to update growth references regularly. The currently available growth reference curves in use in India are based on the data collected in 1989 which were published in 1992 and 1994, and were then adopted by the Indian Academy of Pediatrics for growth monitoring in 2007 (Khadilkar et al., 2009). These data are now 17 years old and there are doubts as to whether they are representative of the growth of present day Indian children. The populations of developed countries can generally be considered to have achieved their full genetic growth potential, so there are no longer important socioeconomic gradients in growth, and a random sample of the population can be used for constructing growth curves (Khadilkar et al., 2009).

However, in a developing country such as India, children belonging to affluent families in urban areas have fewer constraints on growth than other children, thus making it necessary to measure these children for the purpose of reference curves (Khadilkar et al., 2009). The WHO has encouraged all countries and regions throughout the world to adopt the new WHO growth standards for children under 5 years of age published in April 2006, where the data collected were multi-country (including India) and community-based (Multicentre Growth Reference Study-MGRS). Therefore, this current study excludes children under the age of 5 years. This study was planned to design new reference curves for height, weight and body mass index for affluent urban Indian children aged 5 – 18 years.

An accurate assessment of the physical growth and development of children has attracted more attention from government health officers and paediatricians (Lee et al., 2004). Many studies report standard growth for height and weight among Taiwan children (Lee et al., 2004). They are based on cross-sectional survey to obtain growth standard curves that are different from the

curves obtained longitudinally. It has been pointed out that longitudinal, rather than cross-sectional, growth standards are more effective in assessing individual linear growth, and the difference is particularly apparent during puberty in standards for growth velocity (Lee et al., 2004). In 1966, Tanner et al. (1976) produced longitudinal standards suitable for clinical use in the British population (Lee et al., 2004). Longitudinally-based height velocity charts for North American children were also presented in 1985. The study by Lee et al. (2004) was to investigate the growth characteristics of groups of school boys and girls living in the Shih-Pai district of metropolitan Taipei. The focus of the study was to follow these boys and girls throughout their pubertal years so that a longitudinal growth curve could be obtained.

The results of the anthropometric measurement of height, height velocity, weight, and weight velocity in the different age groups of school children were presented in tables. The age at PHV was taken as 12.5 years for boys and 10.5 years for girls, and the whole year velocity at PHV was taken as 8.0 cm/yr for boys and 7.0 cm/yr for girls. The mean height velocity was less than 1 cm in boys and girls of about 17 years and 15 years, respectively, with mean values of height of 170.8 cm and 158.7 cm, respectively.

Height of an individual grows throughout childhood and puberty (Mansur et al., 2016). Growth in height ceases at 25 years of age, when epiphysis gets fused with diaphysis of growing long bones, after which no more bone growth can take place (Mansur et al., 2016). At the same time, no two persons are alike, in their measurable characters especially height of an individual. Few children will remain small throughout life, while others may reach normal size. Few children are taller than expected at a given age and sex, and few are shorter. Parents are more often concerned when their children are shorter than their age-mates. Children may ask parents why they are not as tall as their play-

mates, parents ask doctors, and doctors ask endocrinologists and geneticists when the growth of a child seems unusual.

Growth velocity is independent of the height achieved by a child so it is a sensitive indicator of good or bad health regardless of previous growth delay. The height is relatively a stable measurement of growth as opposed to body weight which reflects only the present health status of the child. On the other hand, the height indicates the events in the past also. The use of growth (height) chart is particularly valuable in studying the trend of height curve (Mansur et al., 2016).

Accurate serial height measurements documented over time on a growth chart are key in the evaluation of the children health and also serve as the foundation for the diagnosis of growth abnormalities such as stunting (Mansur et al., 2016).

In their study, the data on height were analysed for both sexes and considered each age group to assess annual growth in height (Mansur et al., 2016). Growth pattern for height was graphically represented by distance curve however velocity curve was also used to examine the height peaks of annual increments in both sexes. The interval during which the maximum yearly height increment occurred was taken as age at PHV (Mansur et al., 2016).

Their study was carried out on the different age groups of school going children of Dhulikhel (Mansur et al., 2016). Their study revealed that the mean height of 3 years girls and boys were 93.17 cm and 95.50 cm respectively which indicates the differences in height between boys and girls was 2.33 cm at the same age of both sexes. Similarly, 16 years boys had a mean height of 166.91 cm and 154.25 cm among girls at the same age group which indicates 12.66 cm

difference in height between boys and girls. Thus, boys were found to be taller than girls, except at 8, 10 and 12 years of age, when they appeared shorter than girls. Their study concluded that the mean height of boys varied from 95.50 cm to 166.91 cm aging from 3 years to 16 years respectively. The total gain in height from the age of 3 years to 16 years was 71.41 cm. The PHV was calculated as 12.83 cm during 12 – 13 years of age among boys. Hence, it was concluded that the mean height of boys increases with the advancement of age with the maximum increment occurring during 12 to 13 years. The velocity of growth of height was relatively high during 5 to 6 years and decreased until 12 years and then increased until an adolescent peak was reached at an average of 13 years. The rate of gain in height after the peak decreases and is < 2.64 cm/year after 16 years. The distance curve also showed a gradual increase in height from 3 to 16 years, while the velocity curve indicates the highest rate of increment (12.83 cm/year) during the period of 12 to 13 years among the boys. In their study, it was found that the mean height of girls varied from 93.17 to 154.25 cm aging from 3 years to 16 years respectively. The total gain in height from the age of 3 years to 16 years of girls was 61.08 cm. The age at PHV was found during 11 – 12 years for girls, and the whole year velocity at PHV was observed as 9.57 cm/year for girls. Hence, it was concluded that the mean height of girls increases with the advancement of age with the maximum increment (9.57 cm/year) occurring during 11 to 12 years. The velocity of growth of height was relatively high (8.16 cm/year) during 5 to 6 years and decreased until 11 years and then increases until an adolescent peak was reached at an average of 12 years. The rate of gain in height after the peak decreases and was < 0.12 cm/year after 16 years. In their study, the distance curve for height showed a gradual increase in height from 3 to 16 years, while the velocity curve indicates the highest rate of increment (9.57 cm/year) at the age of 12 years among the girls (Mansur et al., 2016).

The mean values for height are found to be progressively increasing till 13 years among boys and 12 years among girls; and showed a steady pattern of growth, and afterwards almost stationery pattern is noticed with decelerating trend of growth progression in both sexes. This may indicate the period of growth spurts due to puberty which was found to be earlier among girls than boys by one year (Mansur et al., 2016).

There was significant correlation coefficient between age and height ($r = 0.960$, $p < 0.01$) for boys, ($r = 0.959$, $p < 0.01$) for girls and ($r = 0.957$, $p < 0.01$) for both (boys and girls). There was strong positive correlation between age and height of children. The formula estimating average height of healthy children aged 3 – 16 years were:

Height (cm) = Age (year) x 5.860 + 75.201 for boys

Height (cm) = Age (year) x 5.282 + 77.819 for girls

Height (cm) = Age (year) x 5.634 + 76.122 for both (boys and girls)

2.1.5 Africa

Chirwa et al. (2014) applied mixed effects models to the Birth-to-Twenty (BT20) cohort study in South Africa to compare several growth models. BT20 study is a longitudinal cohort study aimed at tracking the growth and development of urban children born in Soweto-Johannesburg from their birth between April and June 1990, initially for a period of 10 years (BT10), and later expanded to 20 years (Richter et al., 2009). The main advantage of mixed effects models is the power to model longitudinal data with participants measured at different time periods, and some missing data at times. Mixed effects models enable the fitting of individual curves and allow for fitting general population curves. The fixed component summarises the general population curve using

the mean structure, while the random component allows for variations in individual growth of participants.

Cameron et al. (1994) stated that adolescent growth in height, fatness, and fat patterning was investigated in a sample of 79 rural South African black children which were followed longitudinally from 6 to 18 years. Data were analysed relative to peak height velocity (PHV) to identify the phenomenon of “compensatory” growth in height during adolescence and to describe changes in fatness and fat patterning. Compensatory growth following PHV was clearly observed relative to the National Health and Nutritional Examination Survey (NHANES) data for African-Americans in that Z-scores for height at the start of the adolescent growth spurt were greater than those at the end of the spurt. The authors found that statistically significant differences in fatness and centralisation between males and females did not occur until about two years after PHV was attained.

Elusiyan et al. (2016) stated that there are no locally derived growth charts in Nigeria, and that makes health workers to rely on international reference charts. They then decided to compare the growth characteristics of 4350 school-age Nigerian children (2243 girls and 2107 boys aged 4 – 16 years) from three ethnic groups (Hausa, Igbo and Yoruba) to both UK (UK 1990) and US (2000 CDC) reference data (Elusiyan et al., 2016). Their results showed that school-age Nigerian children were similar to international references at the start of school age and then started to decline. The decline appeared to peak at 15 years for boys and 13 years for girls. There were significant differences in the prevalence of stunting, underweight, and obesity among the three ethnic groups ($p < 0.05$) (Elusiyan et al., 2016). They concluded that there was a risk of over-diagnosing short stature and underweight if health workers continue to use growth charts derived from other geographical areas (Elusiyan et al.,

2016).

Growth is a natural process that takes place in all living organisms and its pattern varies among individuals (Mbagwu et al., 2015). The results obtained from Mbagwu et al. (2015) showed that the adolescent private female school children recorded higher mean values than their public school counterparts in all parameters measured. According to a similar study by Uthman (2009), this variation results from variation in the growth of other body parts relative to variations in the onset of adolescent growth spurt.

In southeast Cameroon, Yamauchi and Hagino (2014) determined the growth pattern of pygmy hunter-gathers, especially the periods and the growth tempo during childhood and adolescent using the mathematical growth model. In their findings Yamauchi and Hagino (2014) found that the velocity at take-off (VTO) and PHV were generally lower than that of the previously reported groups. The VTO-PHV velocity increment (VTO-PHV inc) was also the lowest (Yamauchi and Hagino, 2014). Their age at take-off (ATO), considered as a marker indicating the onset of adolescence was similar among Baka, Indian and Guatemalan boys and Baka and Indian girls. The age at peak height velocity (APHV) was average in girls, but slightly delayed in boys. Compared with Gambian children, Baka children showed 1 – 2 years earlier ATO and APHV. The adolescent growth spurt of the Baka children was defined as extremely weak compared with other populations (Yamauchi and Hagino, 2014). Height growth velocity was generally smaller than that of other populations, except for the latter period of adolescence. The PHV in Baka children, 4.88 cm/year for boys and 4.71 cm/year for girls, was one of the most characteristic biological parameters in the growth of the Baka children, being only 50 – 80% compared with other five populations (e.g. boys: 59.2% and girls: 62.9% of British children) (Yamauchi and Hagino, 2014).

In Sokoto, North-Western Nigeria, Ahmad et al. (2013) found that the mean weight of the study subjects was 46.4 ± 11.5 kg for males and 47.1 ± 9.2 kg for females. In both gender, the mean weight pattern between the males and females from age 16 years showed that males became heavier than females (Ahmad et al., 2013). They also found that the mean height values were 158.2 ± 11.2 cm for males and 155.7 ± 6.5 cm for females. Mean height generally increased with increase in age and was initially higher in females until age 14. From 15 years through 18 years, the males became taller than their age-matched female counterparts (Ahmad et al., 2013). The authors discussed that the mean weight of the studied subjects increased with age in both males and females. The females were generally heavier than their age-matched male counterparts, though the difference was not significant. Their finding of females being heavier than males was similar to that of Akesode and Ajibode (1983), among school children aged 6 – 16 years in Abeokuta, South-Western Nigeria. They further explained that the female subjects were also initially noticed to be taller than the males up to the age of 14 years, with the difference being statistically significant (Ahmad et al., 2013).

Another study in a city in Nigeria by Ejike et al. (2013) showed that girls were taller than boys from age 7 to 13 years. A total of 1911 children and adolescents (51.3% females) were recruited and anthropometric data obtained from them using the standard procedures. Age was determined from each subject's school records. Overweight, obesity, and thinness (stages 1 – 3) were defined using the international surveys and the WHO study references. From age 14 years boys were taller than girls, though the differences were not significant. They further highlighted that girls weighed more than boys from age 8 to 15 years. Conversely, boys weighed more than girls at ages 16 and 17 years. The differences were significant only at the ages 12 and 17 years (Ejike et al., 2013).

Prentice et al. (2012) tested whether 12 mo of calcium supplementation at age 8 – 12 years to increase intakes toward international recommendations had long-term effects on adolescent growth and pubertal development in rural Gambian children. Their study showed that 12 mo of calcium carbonate supplementation before puberty had a long-term effect on the pattern of adolescent height growth in Gambian boys, which suggests an earlier pubertal growth spurt and resultant greater achieved height mid-adolescence, followed by an earlier cessation of growth and shorter adult stature. They also found that the calcium supplement had advanced the age of PHV of boys by 7.4 mo. Height velocity itself was also not affected, which indicated that the supplementation effects were attributable to a shortened period of pre-pubertal growth coupled with earlier growth cessation rather than to a more rapid and accentuated pubertal transit. In girls, Prentice et al. (2012) found that no effects of calcium supplementation were found on growth and maturation.

Growth monitoring is nearly universally practiced in pediatric care worldwide to detect growth faltering and thus intervene accordingly (Schwinger et al., 2016). Various anthropometric indexes, such as indexes based on weight, height, or midupper arm circumference, 6 have been suggested, but somewhat different abilities to predict the risk of child deaths were reported (Schwinger et al., 2016). Despite their hypothesised advantages for early detection of growth problems, and thus the possibility to direct life-saving interventions, currently only a few studies have assessed the mortality-predictive ability of longitudinal indicators of poor growth (i.e., growth velocities) (Schwinger et al., 2016). These studies used various approaches to define and score growth velocity, but only one study looked at the predictive ability of weight velocity z scores by using the WHO standards.

It was found that very low weight velocity (z scores, 23) predicted death within

3 mo to some degree. However, they did not include weight velocity z scores in their comparative analysis and did not assess length velocity. In addition, changes in midupper arm circumference and midupper arm circumference z scores were not explored. This encouraged Schwinger et al. (2016) to expand on the analysis of O'Neill et al. (2012) within the same study population to further explore how various velocity measures could predict the risk of death.

2.1.6 Other parts of the world

Child chronic malnutrition is endemic in low-and middle-income countries and deleterious for child developments (Georgiadis et al., 2016). In their study, they developed and estimated a general path model of the relationship between growth trajectories and cognitive achievement using data on four cohorts from Ethiopia, India, Peru, and Vietnam (Georgiadis et al., 2016). The results from their study suggested that growth from conception through age one year, between age one and five years, and between five years and eight years, are each positively and significantly associated with cognitive achievement at age eight years. This may be partly explained by the fact that faster-growing children start school earlier. They also found that a significant share of the association between early growth and later cognitive achievements is mediated through growth in interim periods (Georgiadis et al., 2016).

Billewicz and McGregor (1982) stated that the height growth curves indicate that puberty is much delayed in Gambian adolescents in comparison to British and West Bengal adolescents. They found that the mean at peak height velocity (PHV) was 16.3 and 13.8 years for boys and girls, respectively. In girls, but not in boys, there was a significant negative correlation (-0.46) between the age at PHV and PHV itself. Comparison with British data suggests that growth patterns in the Gambian villages are characterised by the substantial deficits in both height and weight that develop in early life and which appear

to persist without rectification into adulthood (Billewicz and McGregor, 1982).

Burns et al. (2011) evaluated the associations of serum dioxins and polychlorinated biphenyls (PCBs) with longitudinal assessed growth measurements among peripheral Russian boys. At study entry and during three years of follow-up, > 50% of the boys had age-adjusted BMI and height z scores within 1 SD of WHO standardized mean values for age. Boys in the highest exposure quintile of the sum of dioxin and PCB concentrations and total toxic equivalency (TEQ) had a significant decrease in mean BMI z scores of 0.67 for dioxins and TEQs and 1.04 for PCBs, compared with boys in the lowest exposure quintile. Comparison of the highest versus the lowest quintile revealed that the higher serum PCB concentrations were associated with significantly lower height z scores (mean z-score decrease: 0.41) and height velocity (mean decrease: 0.19 cm/year) after three years of follow-up. Burns et al. (2011) further indicated that exposures to dioxins and PCBs are associated with reduced growth during the peripheral period and may compromise adult body mass, stature, and health.

Montaño et al. (2008) highlighted that children with Morquio A disease grow poorly and become physically handicapped because of systemic bone disease. The purpose of their study was to describe observed growth patterns and their relationship with the physical condition of patients with Morquio A. They found the mean birth lengths of boys and girls to be 52.6 and 52.1 cm, respectively. The mean final heights for males and females at 18 years and older were 122.4 ± 21.5 and 113 ± 22.6 cm, respectively. Mean birth weights for boys and girls were 3.59 ± 0.58 and 3.5 ± 0.7 kg, respectively. The growth pattern in Marquoi A patients was made by impaired growth velocity after 1 year of age (Montaño et al., 2008).

The development of an international growth standard for the screening, surveillance and monitoring of school-aged children and adolescents has been motivated by two contemporaneous events: the global surge in childhood obesity; and the release of a new international growth standard for infants and preschool children by the WHO in collaboration with the United Nations University and other United Nations agencies, governments, and non-governmental organizations (Butte et al., 2007). To address the feasibility of adopting a prescriptive approach to developing a new international growth standard for school-aged children and adolescents from either historical and/or prospective growth data, it would be useful to reaffirm the operational difference between growth reference (description of the growth pattern of a defined population) and growth standards (a recommended pattern of growth that has been associated empirically with specified health outcomes and the minimisation of long-term risks of disease).

The world-wide variation in human growth is well known and has scientifically been documented since the first half of the 19th century (Hermanussen et al., 2016). The shortest mean final height has been measured in the Pygmy population of Congo with some 136 cm for adult women and 144 cm for adult men, and the tallest mean height was found in young modern Dutch adults with some 171 cm for adult females and 184 cm for adult males (Hermanussen et al., 2016). Differences between populations are obvious at all ages: Indian children start into life with significantly less average birth weight than European newborns (Hermanussen et al., 2016). Secular trends in height, weight, and BMI have been documented in European countries, in the United States since the mid-19th century, in the Southern Hemisphere and in all populations that underwent a significant socioeconomic transition (Hermanussen et al., 2016).

Variations in human growth have been attributed to genetics, nutrition and

health-related and socioeconomic circumstances (Hermanussen et al., 2016). Growth even differs among populations and ethnic groups that live in close vicinity within the same geographic area. Documenting child and adolescent growth has led to a multitude of growth charts published since the early 20th century. Meanwhile, these charts are universally used in public health care as pediatric decisions on growth and failure to thrive are intricately intertwined with such charts (Hermanussen et al., 2016).

Among males, modern Northern and Central Europeans are tallest at all ages. Historic male Japanese and modern boys from Papua New Guinea were the shortest. This is similar in females. Modern European females are significantly taller than modern East Asians (Hermanussen et al., 2016). Conversely, human growth curves show common characteristics. During the first year of life, infants increase in length by some 50% and almost triple in weight. Thereafter, growth rates decrease during childhood and the juvenile period (Bogin, 1999) with a minimum just before the onset of puberty. Growth again accelerates with peak height velocities roughly around the age of 11 years in girls and 13 years in boys. Growth of the long bones terminates at early adult age, whereas trunk growth may proceed into the middle of the third decade of life (Hermanussen et al., 2016).

Studies on growth development and variations have led to the idea of globally applicable growth references. At present, many countries that lack suitable references for child and adolescent growth use international WHO standards (<http://www.who.int/childgrowth/en/>; <http://www.who.int/growthref/en/>). The idea of growth standards goes back to recommendations of a Working Group on infant growth established by the WHO, and may be justified for infants and very young children who tend to grow similarly under modern affluent conditions. As some scholars argue: the disadvantage of using charts

such as WHO charts is that they are likely to over diagnose underweight and stunting in a large number of apparently normal children in the developing countries such as India. WHO standards were constructed from global samples, they average information of children and adolescents from various ethnic backgrounds, and consequently do not take into account the fact that different modern populations may differ in mean values, standard deviations, and indicators of skewness for height, weight, and BMI. Populations differ in height, weight, and BMI.

As an averaged single standard/reference can never account for the diversity between populations a methodology has been created to generate “synthetic” growth reference charts (Hermanussen and Burmeister, 1999). The method allows for amalgamating global patterns of human growth with specific local information. The original method was based on 50 studies of birth measurements, 14 studies on early growth in height and weight, 40 male and 51 female childhood and adolescent growth studies, and some recent German, Japanese, and Czechoslovakian data, with altogether more than 24 million measurements. In view of the persistent need for national growth references as well as references for particular ethnic groups, we now actualise these previous approaches and further improve the methodology of generating synthetic growth reference charts.

Due to lack of availability of updated local growth reference charts, Hermanussen et al. (2016) constructed synthetic references as proxy to growth curves of a population for which there is a limited set of mean height, mean weight, or mean BMI values. They made use of Principal Component Analysis and the Likelihood principle to generate synthetic references separately for height, weight, and BMI for age of any population that lacks complete annual data of these parameters. They trusted their method to be applicable globally, and

should provide the most likely growth curve separately for height, weight, and BMI for any population.

Under the assumption that limited sets of mean values represent the true development over time of the population of interest, they applied a Bayesian rationale to find the synthetic curve for such population, which best represents the compromise that considers both the limited set of height, weight, or BMI values of our population of interest, and the global patterns of height, weight, or BMI obtained from the Principal Component Analysis. The new synthetically generated curves describe mean values for height, weight, and BMI from 0 to 18 years.

In their study, Lee et al. (2004) found that the mean age at PHV was 12.5 years in boys and 10.5 years in girls, with slight variations in the different studies. Marshall and Tanner (1970) reported the mean of 14.0 years in boys while Tanner et al. (1976) estimated it to be 13.9 years, with whole year PHV of 8.8 cm/yr in boys in the Harpendeen Growth Study. Billewicz et al. (1981) observed that PHV for boys and girls were 9.63 cm/yr and 8.02 cm/yr, respectively. Largo et al. (1978) reported an averages of 12 years for PHV in girls and of 14 years in boys based on a Zurich longitudinal study, and the whole year PHV was 7 cm and 9 cm in girls and boys, respectively. Tanaka et al. (1988) calculated linear growth in 438 boys and 483 girls aged from 6 years to 17 years in Japan. They reported a mean age of PHV to be 11.2 years in girls and 13.0 years in boys.

To mention a few, some research studies on growth variations have been done in countries like Denmark, Pakistan, England, USA, UK and Senegal (Rona and Altman, 1977; Kelly et al., 1997; Rogol et al., 2000; Richards et al., 2002; Benefice, 2007; Tinggaard et al., 2014). However, growth variations have not been studied for rural South African children in Ellisras.

2.2 Chapter summary

This chapter has shown what other researchers have done and found in their studies. Other researchers have studied growth variations of children in their respective countries and also compared their findings with those of other countries. Many different methods of analysis have been used to study the growth variations across children in different countries. Methods such as mixed effects model, height-for-age difference, logistics regression, shape-invariant model, variable knot cubic splines method and optimal kernel have been used in other studies.

These studies were relevant to this study as they gave much needed understanding on how to tackle the issues in this study. These studies gave guidance as to what could be done and what to be expected from this study. The relevance of these reviewed studies to this study is of importance in that they also helped in the formulation of the conclusions and or recommendations.

Above all, the methods from the reviewed studies have presented good findings which are almost the same when comparing children across different countries. For this study, the ELS data was observed in order to choose the best method to analyse the data. Having considered some missing ELS data, linear mixed model was chosen to analyse the data, since it accommodates missing data. The findings of the ELS which will result from the analysis of this study are expected to not differ that much with the findings from other studies across the world.

Chapter 3

Research methodology

3.1 Study design

The study will follow a secondary analytical cross section data collected in November 1996 to November 2003 and the data was collected by the ELS team in Ellisras, Limpopo Province, South Africa.

3.2 Sample/Study population

3.2.1 Geographical area

Ellisras is a deep rural area situated within the north-western area of the Limpopo Province, South Africa, with about 115 767 people (Statistics South Africa, 2011) residing in 42 settlements (Sidiropoulos et al., 1996). These villages are approximately 70 km from the Ellisras town (23 degrees 40S 27 degrees 44W), now known as Lephalale, which is adjacent to the Botswana border. The Iscor coal mine and Matimba electricity power station are the major sources of

employment for many of the Ellisras residents. The remaining workforce is involved in subsistence farming and cattle rearing, while the minority is in the education and civil service. Unemployment, poverty and low life expectancy seem to play a significant role in the rural South African population, of which the communities in rural Ellisras are no exception (Bradshaw and Steyn, 2001; Statistics South Africa, 2002).

3.2.2 Sample

The ELS initially followed a cluster sampling method (Monyeki et al., 2000). The study was undertaken at 22 schools (10 pre-schools and 12 primary schools) randomly selected from 68 schools within the Ellisras area. Birth records were obtained from the school admission register through the assistance of the principals in each school. Only those records that were verified against health clinic records were used to determine the ages of potential participants. Each of the 22 selected schools was assigned a grade with the expectation that most of the children in a particular age category (3–10 years) would be found in that grade.

Data collected in November 1996, May 1997, November 1997, May 1998, November 1998, May 1999, November 1999, May 2000, November 2000, May 2001, May 2002, May 2003 and November 2003, is used in this study. A total of 2225 children (550 pre-schools and 1675 primary schools) were followed throughout the periodic surveys. On average 1.05% of participants were permanently lost due to death and 11.47% subjects were lost due to teenage pregnancy, illness and migration to urban areas. School drop-out was a temporary issue as the affected participants re-joined school thereafter.

3.2.3 Attrition rate

The current average attrition rate for the ELS is 17.2%. Using STATA program, the sample size required to test for cohort and period of measurement effect was calculated based on a 5% margin of error, population power of 0.80 and the prevalence of low birth weight among ELS subjects of 12%. A total of 1771 subjects (489 preschool children with mean age of 11.4 years and standard deviation of 0.96 and 1282 primary school children mean age of 14.9 years and standard deviation of 1.11) were measured in November 2003. Subjects who were lost to follow up were included in the analysis to check for bias of the outcome of interest as it was the case in the previous study (Monyeki et al., 2009).

3.2.4 Anthropometry measurement

All children underwent a series of anthropometric measurements of height and weight according to the standard procedures recommended by the International Society for the Advancement of Kinanthropometry (ISAK) (Norton and Olds, 1996). Weight was measured on an electronic scale to the nearest 0.1 kg, and a Martin anthropometer was used to measure height to the nearest 0.1 cm.

3.3 Quality control

The survey was carried out over a three-week period by 16 anthropometrists each year, who were required to undertake reliability testing as part of their training. This training was conducted by a level three criterion of ISAK following the ISAK guidelines (Norton and Olds, 1996). The absolute and relative values for intra-tester and inter-tester technical error of measurements (% TEM) ranged from 0.12 (0.15%) to 0.31 kg (0.36%) for weight and from 0.22 cm (0.12%) to 0.43 (0.32%) for height.

3.4 Statistical analysis

All analyses were performed using the statistical package for social sciences (SPSS) version 22. The descriptive statistics was done for age, height, weight, velocity and acceleration by gender amongst children in rural Elliras from 1996 to 2003. The line graphs were fitted for age (years), height (cm), velocity (cm/year), and acceleration (cm/year²), separately for both boys and girls.

Weight distance curve helps us to see the plot of the child's weight at each birthday. Velocity helps us to be able to identify the age at which the growth of a child or an individual was fastest. Whereas acceleration enables us to know the rate of change in the velocity of the growth of a child or an individual. Type III Test is used to examine significance or importance of each partial effect, that is, the significance of an effect with all other effects in the model.

Both descriptive statistics and linear mixed model (LMM) were used for analysis. Age was identified as a repeated variable indexing the repeated measures on each subject. We assumed that adjacent observations on the same subject (indexed by age) might have errors with a higher correlation than the observations that are further apart, so we specified a first-order autoregressive [AR(1)] covariance structure. We defined the dependent variable as height, the categorical factor as gender, and the continuous covariate as age. By specifying age as a continuous covariate, we indicate that we wish to estimate the functional relationship of age with height. The fixed effects associated with age, gender, and the interaction between age and gender were included. The estimated marginal means for the levels of the categorical fixed factor gender are displayed. The statistical significance was set at $p < 0.05$.

3.4.1 Linear mixed models

In practice, longitudinal data are often highly unbalanced in the sense that not an equal number of measurements is available for all subjects and/or that measurements are not taken at fixed time points (Verbeke and Molenberghs, 2009). Linear mixed models are commonly used to understand changes in human behavior over time (Shek and Ma, 2011).

The assumption with standard linear models of independent error terms does not always hold. This may be because individuals are related, or measurements are taken repeatedly on the same individuals. Linear mixed models do not assume independence of errors and are therefore more suited to ELS data. These models allow variation between people in the intercept and/or slope(s) of a model (Haig, 2013).

The parameters in a linear mixed model may be classified into types:

- fixed effects, associated with the average effect of the independent variable(s) on the dependent variable,
- variance-covariance components associated with the covariance structure of the random effects and the error term.

A general linear mixed model may be expressed as:

$$y_i = X_i\beta + Z_ib_i + \epsilon_i$$

$$b_i \sim N_q(0, \psi)$$

$$\epsilon_i \sim N_{n_i}(0, \sigma^2\Delta_i)$$

where

- y_i is the $n_i \times 1$ dependent variable vector for observations in the i th group.
- X_i is the $n_i \times p$ model matrix for the fixed effects for observations in the group i .
- β is the $p \times 1$ vector of fixed-effect coefficients.
- Z_i is the $n_i \times q$ model matrix for the random effects for observations in group i .
- b_i is the $q \times 1$ vector of random-effect for group i .
- n_i is the number of measurements for subject i .
- ϵ_i is the $n_i \times 1$ vector of errors for observations in group i .
- ψ is the $q \times q$ covariance matrix for the random effects.
- $\sigma^2 \Delta_i$ is the $n_i \times n_i$ covariance matrix for the errors in group i .

In this study, the ELS data is unbalanced in the sense that not an equal number of measurements is available for all subjects. ELS missing data is due to some subjects not being able to be measured during the time of data collection. Some subjects moved to other provinces due to school or work. Other subjects were permanently lost due to death, teenage pregnancy and illness. LMM

accommodates ELS missing data. The LMM will show us the relationship between age and gender, and also share some light on what happens between age and height. LMM will also present much needed information on different models and based on Akaike's information criterion and Schwarz's Bayesian criterion, the best model to fit ELS data will be chosen.

Chapter 4

Research analysis and results

4.1 Introduction

This chapter concentrates on the presentation and findings of growth variations of children in Ellisras, Limpopo Province, South Africa. For all computations, SPSS was used. Microsoft Excel was used to plot the graphs.

4.2 Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellisras rural children from 1996 to 2003

Table 4.1 to Table 4.5 show that boys were growing taller than girls from November 1996 to November 1998. Both boys and girls grew slightly equal in height between May 1999 and May 2000. However, from November 2000 to November 2003 girls grew taller than boys. Between 1996 and 1998 boys

weighed more than girls. In 1999 both boys and girls weighed slightly equal. However, between November 2000 and November 2003 girls weighed more than boys. The PHV occurred at age 11.80 (sd=1.91) for girls in May 2001 and at age 14.21 (sd=2.09) for boys in May 2003. The PHV for boys and girls was 11.20 and 12.45 cm/year, respectively. On average, the rate of velocity for girls was slightly higher than for boys at 13.28 and 13.08 cm/year respectively, and the rate of acceleration for girls was slightly higher than for boys at 1.35 and 1.31 cm/year^2 respectively. On total average, the velocity and acceleration for both boys and girls was 13.18 cm/year and 1.33 cm/year^2 respectively. The onset of adolescent growth is defined by an increase in velocity (positive acceleration) (Nahhas et al., 2014). The onset of growth spurt for boys and girls was observed in November 2000 at 12.30 (sd=1.86) and 12.23 (sd=1.53) cm/year respectively. Mean age and height at onset of growth spurt was 12.05 (sd=1.84) years and 145.14 (sd=10.44) cm for boys, respectively. Mean age and height at onset of growth spurt was 12.32 (sd=1.67) years and 148.31 (sd=10.73) cm for girls, respectively. This tells us that girls were taller than boys by 3.17 cm at onset of growth spurt.

Table 4.1: Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellisras rural children from 1996 to 1997

		Nov 1996		May 1997		Nov 1997	
		Boys	Girls	Boys	Girls	Boys	Girls
Age	M	7.04	6.86	8.24	8.27	9.68	9.02
	(sd)	1.55	1.56	0.85	1.02	1.10	1.11
Height (cm)	M	120.15	120.73	127.63	124.44	133.03	129.78
	(sd)	10.06	11.07	7.38	6.32	6.79	7.03
Weight (kg)	M	18.38	19.05	22.21	20.96	24.70	22.82
	(sd)	3.97	6.82	2.94	3.68	3.60	3.99
Velocity (cm/year)	M	17.66	18.34	15.61	15.22	13.90	14.55
	(sd)	2.90	3.68	1.45	1.53	1.45	1.46
Acceleration (cm/year²)	M	2.73	2.97	1.93	1.87	1.47	1.66
	(sd)	1.11	1.52	0.37	0.39	0.33	0.36

M = Mean, (sd) = Standard Deviation, Nov = November

Table 4.2: Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellistras rural children from 1998 to May 1999

		May 1998		Nov 1998		May 1999	
		Boys	Girls	Boys	Girls	Boys	Girls
Age	M	9.73	9.33	10.22	9.82	10.73	10.47
	(sd)	1.17	1.06	1.17	1.06	1.14	1.10
Height (cm)	M	132.33	130.70	134.49	132.93	137.62	137.04
	(sd)	6.54	6.61	6.65	6.60	6.79	7.49
Weight (kg)	M	25.01	23.76	26.36	25.05	27.27	27.35
	(sd)	3.53	3.99	3.84	4.15	4.20	4.70
Velocity (cm/year)	M	13.75	14.14	13.28	13.64	12.93	13.19
	(sd)	1.34	1.30	1.28	1.19	1.14	1.05
Acceleration (cm/year ²)	M	1.45	1.55	1.33	1.42	1.23	1.25
	(sd)	0.30	0.30	0.27	0.26	0.23	0.22

M = Mean, (sd) = Standard Deviation, Nov = November

Table 4.3: Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellistras rural children from November 1999 to November 2000

		Nov 1999		May 2000		Nov 2000	
		Boys	Girls	Boys	Girls	Boys	Girls
Age	M	11.25	10.89	11.75	11.49	12.05	12.32
	(sd)	1.15	1.10	1.14	1.10	1.84	1.67
Height (cm)	M	140.16	139.69	143.23	143.36	145.14	148.31
	(sd)	6.54	6.88	6.95	8.19	10.44	10.73
Weight (kg)	M	28.65	28.55	30.38	29.83	32.63	35.25
	(sd)	4.59	4.95	4.89	5.86	6.15	8.17
Velocity (cm/year)	M	12.55	12.90	12.27	12.55	12.30	12.23
	(sd)	1.07	0.94	1.01	0.91	1.86	1.53
Acceleration (cm/year ²)	M	1.14	1.20	1.06	1.11	1.09	1.01
	(sd)	0.20	0.20	0.18	0.17	0.46	0.40

M = Mean, (sd) = Standard Deviation, Nov = November

Table 4.4: Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellistras rural children from May 2000 to May 2003

		May 2001		May 2002		May 2003	
		Boys	Girls	Boys	Girls	Boys	Girls
Age	M	11.85	11.80	12.70	12.67	14.21	14.37
	(sd)	1.83	1.91	1.47	1.22	2.09	1.90
Height (cm)	M	142.90	144.48	146.91	148.97	156.73	156.41
	(sd)	10.82	11.94	8.74	7.93	12.87	10.92
Weight (kg)	M	30.72	31.91	34.31	36.57	40.51	43.30
	(sd)	6.64	7.97	6.81	7.57	9.64	10.78
Velocity (cm/year)	M	12.28	12.45	11.67	111.83	11.20	11.01
	(sd)	1.60	1.40	1.05	0.89	1.32	1.09
Acceleration (cm/year ²)	M	1.09	1.11	0.94	0.95	0.83	0.80
	(sd)	0.38	0.34	0.19	0.17	0.27	0.23

M = Mean, (sd) = Standard Deviation, Nov = November

Table 4.5: Descriptive statistics for age, height, weight, velocity, and acceleration, by gender amongst Ellisras rural children November 2003

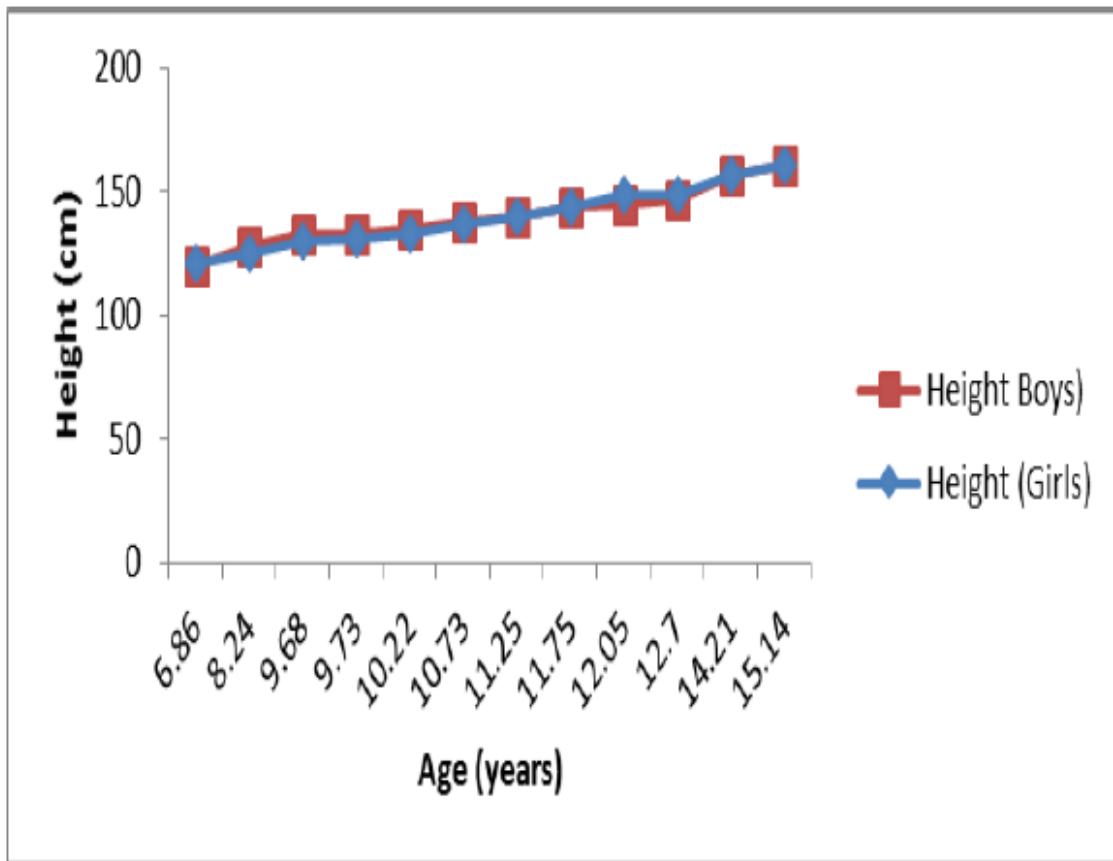
		Nov 2003	
		Boys	Girls
Age	M	15.10	15.14
	(sd)	1.13	0.96
Height (cm)	M	160.06	160.92
	(sd)	8.72	7.79
Weight (kg)	M	43.72	45.92
	(sd)	7.75	7.62
Velocity (cm/year)	M	10.64	10.66
	(sd)	0.67	0.65
Acceleration (cm/year²)	M	0.71	0.71
	(sd)	0.09	0.08

M = Mean, (sd) = Standard Deviation, Nov = November

4.3 Distance curve fitted to the data of boys and girls

Figure 4.1 shows the mean distance curves for boys and girls, age against height. The curve shows that at the third measurement around age 9, boys were a bit taller than girls. At around age 12, girls were a bit taller than boys.

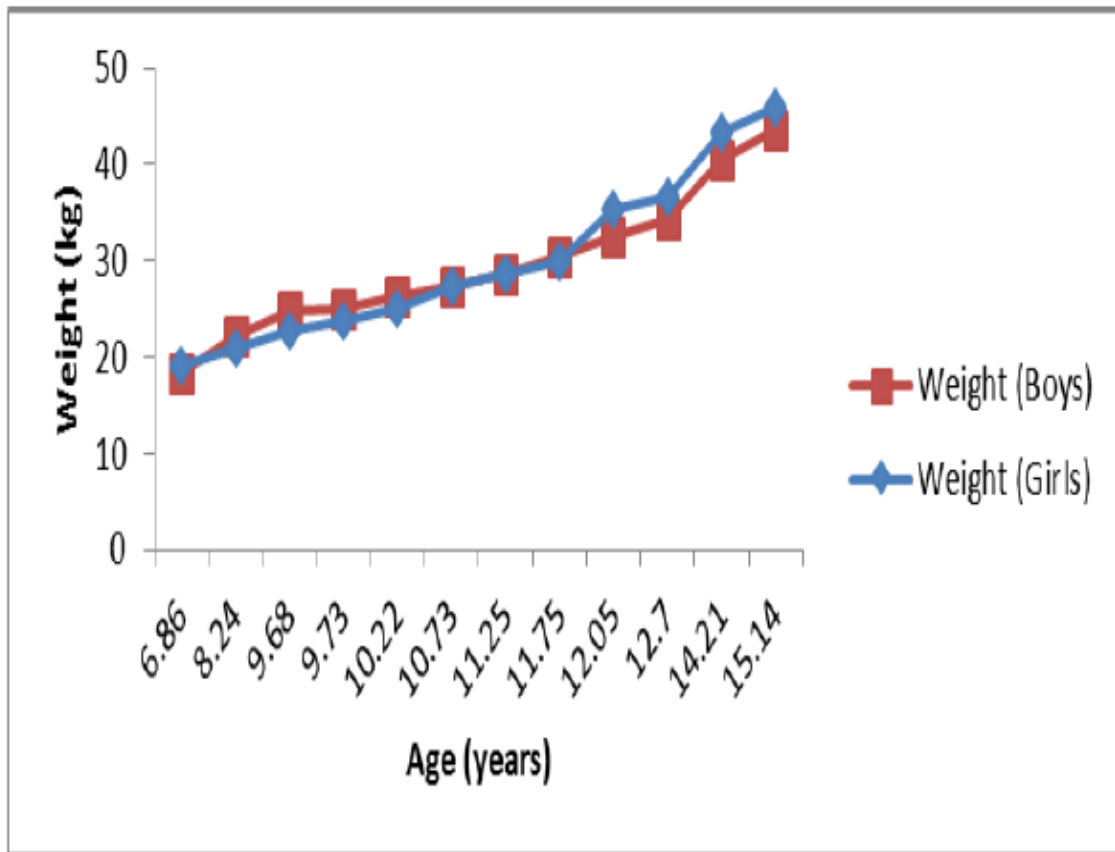
Figure 4.1: Distance curve for boys and girls



4.3.1 Distance curve fitted to weight data for boys and girls

Figure 4.2 shows the mean weight distance curves for boys and girls, age against weight. From measurements 2 to 5 around ages 8 to 10, it is observed that boys weighed more than girls at 24.7 kg for boys and 22.82 kg for girls. At around age 12, boys weighed 32.63 kg and girls weighed 35.25 kg. Girls increased on weight and weighed more than boys until the end of measurements at age 15, where boys weighed 43.72 kg and girls weighed 45.92 kg.

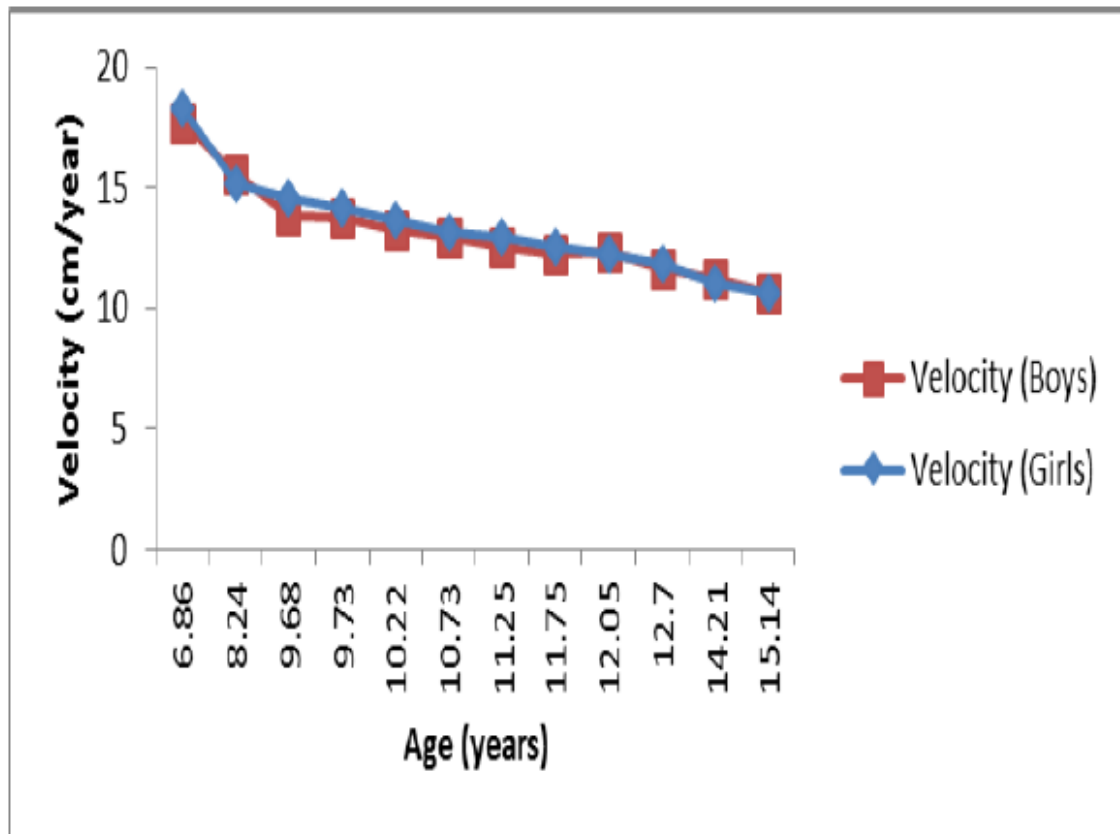
Figure 4.2: Distance curve fitted to weight data for boys and girls



4.4 Velocity curves for boys and girls

Figure 4.3 shows the mean velocity curves for boys and girls, age against velocity. The curves show that velocity is decreasing from the beginning until the last measurement. At around age 9, the velocity for boys decreased by 0.01 cm/year and stood below the girls' velocity.

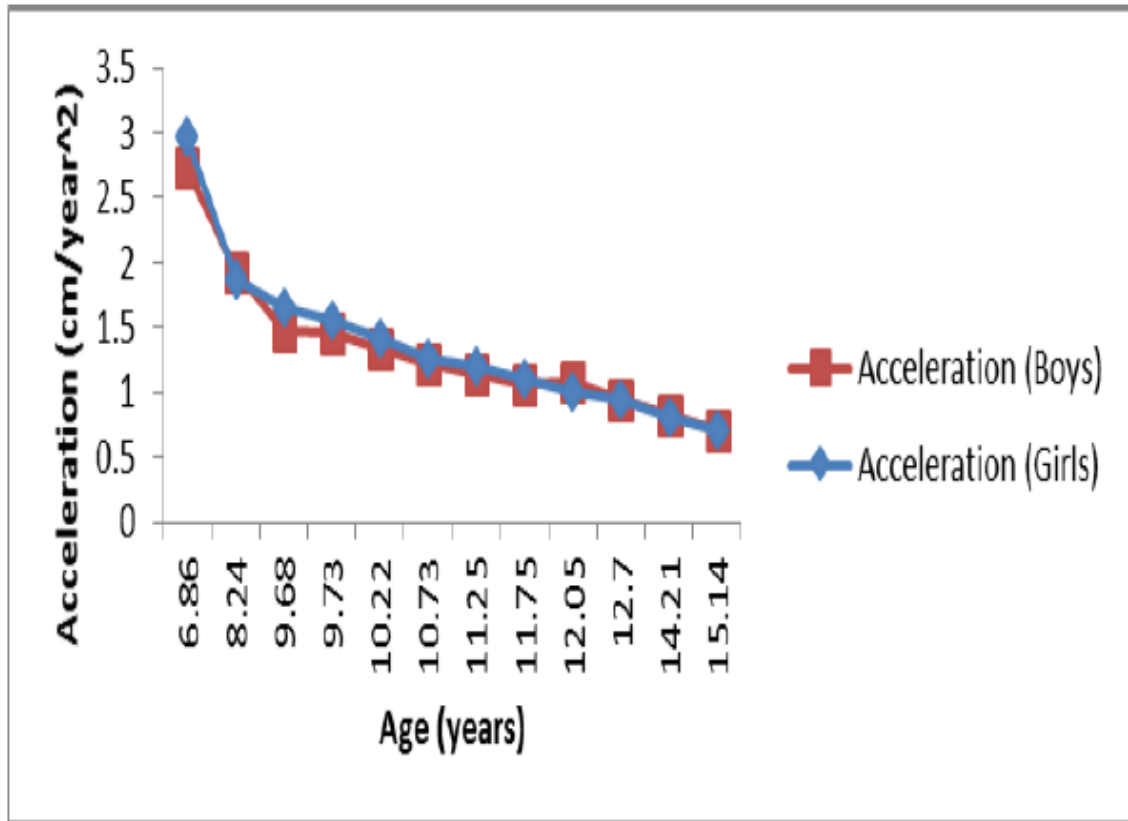
Figure 4.3: Velocity curves for boys and girls



4.4.1 Acceleration curves for boys and girls

Figure 4.4 shows the mean acceleration curves for boys and girls, age against acceleration. The acceleration decreased at a faster rate for the first three measurements than other measurements. Girls started at a higher acceleration of about 3 cm/year^2 above boys. It was also observed that in the middle of the second and third measurements around ages 8 and 9, girls' acceleration stood above that of boys. Around age 12, boys' acceleration took a lead and stood a bit above girls' acceleration. Girls' acceleration decreased faster at about 1.31 cm/year^2 while boys' acceleration decreased by 1.26 cm/year^2 at the beginning of the first three measurements. At the age of 15, both boys and girls had the same acceleration rate of 0.71 cm/year^2 .

Figure 4.4: Acceleration curves for boys and girls



4.5 Type III tests of fixed effects

Table 4.6 shows type III F test for the interaction between age and gender, which is significant at 5 percent level, suggesting that the relationship of age with growth varies depending on gender [$F(1,2122.755) = 54.734, p = 0.000$].

Table 4.6: Type III tests of fixed effects

Source	Numerator df	Denominator df	F	P
Intercept	1	2126.480	176893.189	0.000
Gender	1	2126.480	32.530	0.000
Age	1	2122.755	77127.129	0.000
Gender * Age	1	2122.755	54.734	0.000

Df: degrees of freedom; $p < 0.05$

4.6 Estimates of fixed effects

Table 4.7 shows that there is a significant positive linear relationship of age with distance for females (the reference level of gender), with the estimated fixed effects being 5.177. This suggests that for females, a 1-year increase in age results in an increase of 5.177 in mean distance (or growth), and this fixed effect is significantly different from 0 based on the t test. There is also a significant interaction between gender and age, with the slope decreasing by 0.269 for males. This change in relationship is significant, based on the estimated fixed effect ($p = 0.000$).

Table 4.7: Estimates of fixed effects

Parameter	Estimate	Std. Error	df	t	P	95 per CI	
						LB	UB
Intercept	83.502	0.290	2134.228	287.680	0.000	82.932	84.071
Boys	2.296	0.403	2126.480	5.704	0.000	1.506	3.085
Girls	0	0	-	-	-	-	-
Age	5.177	0.026	2132.544	197.688	0.000	5.125	5.228
Boys * Age	-0.269	0.036	2122.755	-7.398	0.000	-0.340	-0.197
Girls * Age	0	0	-	-	-	-	-

Std. Error: standard error; df: degrees of freedom; per; percent; CI: confidence interval; LB; lower bound; UB: upper bound; $p < 0.05$

4.7 Estimates of covariance parameters

Table 4.8 shows the estimates of covariance parameters representing the maximum likelihood estimates of the covariance parameters. The variance of the random errors is estimated to be 3.40, which means that the random errors associated with the growth observations (taking the fixed effects into account) are sampled from a normal distribution with variance 3.40.

The covariance parameters define a 2×2 variance-covariance matrix for the random effects, illustrated as follows:

$$= \begin{pmatrix} \text{Var}(\text{randomintercepts}) & \text{Cov}(\text{randomintercepts}, \text{randomslopes}) \\ \text{Cov}(\text{randomintercepts}, \text{randomslopes}) & \text{Var}(\text{randomslopes}) \end{pmatrix}$$

$$= \begin{pmatrix} UN(1,1) & UN(2,1) \\ UN(2,1) & UN(2,2) \end{pmatrix}$$

The first parameter, represented by UN(1,1) indicates that there is initial evidence of significance variance in these random effects based on the approximate Wald-test presented by SPSS ($p = 0.000$), suggesting that the intercepts do in fact vary from child to child.

The next covariance parameter, represented by UN(2,1) indicates that the estimated covariance is negative, suggesting that the higher the random effect for the intercept (the larger a subject's intercept), the smaller the random effect for the age slope (the smaller the subject's slope). This finding is quite common in longitudinal studies and makes sense in the context: the more distance that a child has to begin with, the less growing that they have to do.

The last parameter, represented by UN(2,2), is the estimated variance of the random subject effects associated with the age slope in the model. There is also evidence of significant variance in these effects ($p = 0.000$), which suggests that the slopes tend to vary from child to child.

Table 4.8: Estimates of fixed effects

Parameter		Estimate	Std. Error	Wald Z	P	95 per CI	
						LB	UB
Repeated Measures	AR1 diagonal	3.403	0.034	100.985	0.000	3.337	3.470
	AR1 rho	0.000	0.000	-	-	-	-
Intercept + Age	UN(1, 1)	82.863	2.863	28.939	0.000	77.437	88.669
	UN(2, 1)	-5.482	0.229	-23.942	0.000	-5.930	-5.930
	UN(2, 2)	0.643	0.023	28.491	0.000	0.601	0.689

Std. Error: standard error; per; percent; CI: confidence interval; LB; lower bound; UB: upper bound; $p < 0.05$

4.8 Pairwise comparison based on estimated marginal means

Table 4.9 shows that the difference in means between boys and girls at the mean age is not significant at 0.05 level ($p = 0.094$).

Table 4.9: Pairwise comparison based on estimated marginal means

Gender		Mean diff (I-J)	Std. Error	df	P	95 per CI Diff	
(I)Gender	(J)Gender					LB	UB
Boys	Girls	-0.430	0.256	2315.179	0.094	-0.933	0.073
Girls	Boys	0.430	0.256	2315.179	0.094	-0.073	0.933

Std. Error: standard error; df: degrees of freedom; per; percent; CI: confidence interval; diff: difference LB; lower bound; UB: upper bound; $p < 0.05$

4.9 Information criteria for the model with the AR(1) covariance structure

Table 4.10 presents the information criteria for the model with AR(1) covariance structure. In this model, the variance was assumed to be heterogeneous and the correlations between the two adjacent time points declined across measurement occasions.

Table 4.10: Information criteria for the model with the AR(1) covariance structure

	Information criteria
-2 Restricted Log Likelihood	118417.47
Akaike's Information Criterion (AIC)	118427.47
Hurvich and Tsai's Criterion (AICC)	118427.47
Bozdogan's Criterion (CAIC)	118473.14
Schwarz's Bayesian Criterion (BIC)	118468.14

4.10 Information criteria for the reduced model without the AR(1) covariance structure

Table 4.11 presents the information criteria for the reduced model without AR(1) covariance structure. In this model, the variance between time points which is not constant and the correlations between time points differ across time.

Table 4.11: Information criteria for the reduced model without the AR(1) covariance structure

	Information criteria
-2 Restricted Log Likelihood	118417.47
Akaike's Information Criterion (AIC)	118425.47
Hurvich and Tsai's Criterion (AICC)	118425.47
Bozdogan's Criterion (CAIC)	118462.00
Schwarz's Bayesian Criterion (BIC)	118458.00

4.11 Information criteria from the reduced model without the random age effects

Table 4.12 presents the information criteria from the reduced model without the random age effects. In this model, the variance is constant. There is assumed to be no correlation between any elements.

Table 4.12: Information criteria from the reduced model without the random age effects

	Information criteria
-2 Restricted Log Likelihood	127312.96
Akaike's Information Criterion (AIC)	127318.96
Hurvich and Tsai's Criterion (AICC)	127318.97
Bozdogan's Criterion (CAIC)	127346.37
Schwarz's Bayesian Criterion (BIC)	127343.37

4.12 Results of information criteria among three covariance structure models

Table 4.13 presents the results of information criteria among three covariance structure models. The unstructured covariance structure model often offers

the best fit and is most commonly found in longitudinal data as it is the most parsimonious, which requires no assumption in the errors structure (Singer, 1998).

Based on Table 4.13, it was observed that the smallest values in the three fit criterion (i.e., -2RLL, AIC and BIC) were found in the unstructured model. This suggested that the unstructured model was the best model in fitting the ELS data.

Table 4.13: Results of information criteria among three covariance structure models

Covariance structure	-2RLL	AIC	BIC
AR(1)	118417.47	118427.47	118468.14
Unstructured	118417.47	118425.47	118458.00
Identity	127312.96	127318.96	127343.37

Chapter 5

Summary and conclusion

5.1 Introduction

This chapter summarises research undertaken on growth spurt in height and weight of children in rural South Africa using the Ellisras Longitudinal Study (ELS). Conclusions arrived at in this chapter are based on the objectives of the study informed by the analysis conducted. Finally, the chapter offers recommendations, including areas of further research.

5.2 Summary and conclusion

The main purpose of the study was to analyse the growth spurt in height and weight of children in Ellisras, using the ELS. Ellisras, now known as Lephalale, is a rural area located in the north-west of the Limpopo Province, South Africa. According to South Africa's census 2011 (the latest to date), Ellisras has $\pm 115,767$ inhabitants in 42 settlements. Limpopo Province is known as one of

the two most rural and poorest provinces in South Africa, the other being the Eastern Cape. The key indicators of poverty in Ellisras, include low life expectancy and high unemployment rate.

The ELS has created opportunities to understand the health status of children in rural South Africa. The ELS initially followed cluster sampling. Data on children in Ellisras were collected biannually in May and November from 22 schools randomly selected from 68 in the area. The data analysed in this study covered the period between November 1996 and November 2003, inclusive. A total of 2,225 children were followed throughout the periodic surveys. However, 12.5% of the subjects were permanently lost, predominantly due to teenage pregnancy, illness and migration to other areas.

5.2.1 Major findings

The interaction between age and gender was significant suggesting that the relationship of age with growth varies depending on gender. There was also a significant positive linear relationship of age with distance for girls. The covariance parameter, represented by $UN(2,1)$ indicated that the estimated covariance is negative, suggesting that the higher the random effect for the intercept, the smaller the random effect for the age slope. This finding is quite common in longitudinal studies and makes sense in the context: the more distance a child has to begin with, the less growing that they have to do (West, 2009). There was also evidence of significant variance in the effects, which suggested that the slopes tend to vary from child to child. Based on the smallest values of AIC and BIC, the unstructured covariance structure model was the best model chosen to fit the ELS data.

The onset of growth spurt for Ellisras rural boys was at 12.05 years and for girls was at 12.32 years while the Senegalese boys took off earlier at 11.02

years (Benefice, 2007). According to Rogol et al. (2000) USA boys had their onset growth spurt at 13 years while the Ellisras rural boys had their onset growth spurt at 12.05 years, which only differs by 0.95 years with USA boys. From this, it can be said that Ellisras rural boys and USA boys enter their onset growth spurt almost at the same age. USA girls had their onset growth spurt at 11 years (Rogol et al., 2000) and Ellisras rural girls had their onset growth spurt at 12.32 years. From this it can be stated that USA girls had their onset growth spurt before Ellisras rural girls.

At onset of growth spurt, Turkish boys were at 11.6 years and 146.1 cm (Bundak et al., 2007), while Ellisras boys were at 12.05 years and 145.14 cm. From these, we can tell that Turkish boys had an earlier onset of growth spurt than Ellisras boys with a difference of 0.45 years. Turkish boys were also taller than Ellisras boys at onset of growth spurt by a difference of 0.96 cm.

Velocity and acceleration decreases as age increases for both boys and girls. This decrease means that when both boys and girls grow up, the rate at which they grew tend to slow down and move slowly. However, the findings in this study showed that at an early age the rate of growth is very fast but decreases when boys and girls grow older.

The Ellisras rural boys reached their PHV at 14.21 years while the Senegalese boys reached their PHV at 16.4 years (Benefice, 2007). Newcastle upon Tyne adolescents reached their PHV at 14 and 12 years for boys and girls respectively (Billewicz et al., 1981), similarly with Ellisras rural children at 14.21 and 11.80 years for boys and girls respectively. Ellisras rural girls had their PHV at 11.80 years earlier than Ellisras rural boys at 14.21 years.

The Dutch boys had their PHV at 14.0 years (Kemper et al., 1985), similarly

with Ellisras rural boys at 14.21 years. Riga school children according to AberbergaAugskalne and Kemper (2007) had their PHV at age 12.2 in girls and 13.7 in boys, which is almost similar to Ellisras rural children at ages 11.80 in girls and 14.21 in boys. The PHV for Ellisras rural children does not differ to other studies across the world.

In another study by AberbergaAugskalne and Kemper (2007) their girls reached PHV at 12.2 years and boys at 13.7 years almost the same with Ellisras rural children at 11.80 years for girls and 14.21 years for boys. With just a little difference, Ellisras rural girls had their PHV before girls in the study by AberbergaAugskalne and Kemper (2007) while their boys had their PHV before Ellisras rural boys.

Comparing Gambian children and Ellisras rural children, Gambian boys had their PHV at 16.3 years (Billewicz and McGregor, 1982) two years later than Ellisras rural boys at 14.21 years. Ellisras rural girls reached their PHV at 11.80 years earlier than Gambian girls at 13.80 years (Billewicz and McGregor, 1982).

Another study in USA by (Granados et al., 2015) showed that the PHV was at 12.1 years for girls and 13.7 years for boys. Compared with Ellisras rural children, girls at 11.80 years and boys at 14.21 years it can be said that USA children and Ellisras rural children had their PHV at the same age.

The PHV and age at PHV for Flemish boys was 9.7 cm/year and 13.8 years, respectively (Philippaerts et al., 2006). Flemish boys compared with Ellisras boys, the Flemish boys had their PHV earlier at 13.8 years than Ellisras boys at 14.21 years. The PHV for Flemish boys was 9.7 cm/year less than Ellisras boys at 11.20 cm/year. The Flemish and Ellisras boys differ by 1.5 cm/year in

PHV.

5.2.2 Conclusion

In this study, we have analysed and compared growth variations in height and weight of children in a rural South Africa using both descriptive statistics and linear mixed model. Children in rural Ellisras in the Ellisras Longitudinal Study and their growth variations do not differ much compared with other children across the world. The Dutch boys and boys in rural Ellisras both had their PHV at about the same age at 14.00 years and 14.21 years, respectively. USA children had their PHV at 12.10 years for girls and 13.70 years for boys, similarly with Ellisras children at 11.80 years for girls and 14.21 years for boys.

The onset of growth spurt for boys in rural Ellisras was at 12.05 years and for girls it was at 12.32 years, while the Senegalese boys took off earlier at 11.02 years. Above all, girls in rural Ellisras had their PHV at 11.80 years earlier than their boys at 14.21 years and the onset of growth spurt was 12.30 (sd=1.86) and 12.23 (sd=1.53) cm/year for boys and girls, respectively.

5.3 Recommendations

This section provides recommendations based on the research conducted in this study and the literature visited.

5.3.1 Health care prospects

Unhealthy lifestyles are important risks for most of the key causes of morbidity and mortality. Researchers from the Nurses' Health Study calculated that 82% of coronary events in their cohort could be attributed to the lack of adherence to a low-risk lifestyle (Koppes and Kemper, 2005). Improvements in lifestyle

would postpone the first occurrence of disease, and thus would increase the healthy life expectancy. As a consequence, the number of people in a population with chronic disease would decrease. The economic effects of improved lifestyles are therefore immense, especially when the decreased number of people who lose productive life-years is taken into account (Koppes and Kemper, 2005).

5.3.2 Care or prevention

Considering the present and projected situation of considerable increases in medical costs, unstable South African economy and the magnitude of preventable disease and death, it is recommended that more of the health care budget be spent on prevention.

Over the last two decades, there are more and more indications that circumstances early in life, for example, during adolescence, can have important contributions to the risk of disease in adulthood (Koppes and Kemper, 2005). Even pre-conceptional preventive activities have proven effective in the reduction of later disease prevalence. The primary prevention of many adult diseases in fact is a pediatric problem. Public health policies on prevention should take account of this knowledge that prevention is expected to be most fruitful when started in early life (Koppes and Kemper, 2005).

It is now time for preventive actions because trends of increased obesity have been found in children and these trends disturbing (Monyeki et al., 1999; Monyeki et al., 2008; Monyeki and Kemper, 2008). These trends are not restricted to western countries. More developing countries show increases than decreases in the prevalence of overweight (Koppes and Kemper, 2005).

Considering adult health risks that are associated with childhood overweight, a massive public health problem is looming. This problem requires effective public policy responses. Given the high prevalence of overweight in many countries, and the environmental (nongenetic) nature of the problem, there is considerable potential for risk reduction. Although long-term changes of lifestyle behaviours like dietary intake and physical activity are hard to achieve in children and adults, the results of improved lifestyle behaviours will be the prevention and retardation of severely negative effects on health and wellbeing of individuals and societies (Koppes and Kemper, 2005).

In order to be able to cope with the pediatric problem to prevent adult disease, it is important to get a better picture of the early risk factors and preventive factors for later disability and disease, and to understand the pathways through which these factors result in the health effects at adult age. This knowledge could result in improved identification of persons who are at high risk for later disease, and it could target interventions and be of help in monitoring the effects of interventions. Importantly, it could even explain why certain interventions are effective and others are not (Koppes and Kemper, 2005).

The cost of medication is escalating and hence prevention efforts should target the whole population in the sub-saharan African continent (Monyeki and Kemper, 2008). Children are an important target group for health intervention. It is increasingly recognised that the occurrence of adult cardiovascular diseases are influenced by factors operating throughout the life course (Kuh and Smith, 2004).

An unhealthy lifestyle in the population has led to the escalating increase in cardiovascular mortality (Monyeki and Kemper, 2008). Community health workers employed by the government should be available, particularly in rural

areas, to educate the illiterate and ignorant people about the dangers posed by these diseases. A health promotion programme directed at the whole population and a well-coordinated primary health service for early diagnoses and the management of persons at risk of developing the cardiovascular disease could be helpful. The success of the policy depends on equity, unified and decentralization of a healthy service for the population (Monyeki and Kemper, 2008).

Eating plans to prevent cardiovascular diseases such as obesity and hypertension are needed. Community awareness on healthy lifestyle may have a key role in the prevention of such diseases later in life. There is a need for health ministry to improve the public health sector. Above all, the health care providers and the Limpopo provincial health department can use the findings in this study for planning purposes.

5.3.3 Further studies

More research needs to be carried out in order to better understand the growth variations of children in urban and rural areas in South Africa. This study therefore recommends comparison between BT20 and ELS for future research.

5.4 Ethical consideration

The Ethics Committee of the University of Limpopo granted ethical approval prior to the survey, and the parents or guardians provided informed consent. This is part of the on-going Ellisras Longitudinal Study which received ethical clearance on 27 July 2001 and subsequently in 2013 by the MREC/P/204/2013:IR.

References

- ABERBERGAAUGSKALNE, L. AND KEMPER, H. C. G. (2007). Longitudinal relationship between cardiovascular functioning and peak height velocity during exercise in 7 to 16 year old boys and girls. *HUMAN*.
- ABURAWI, E. H., NAGELKERKE, N., DEEB, A., ABDULLA, S., AND ABDULRAZZAQ, Y. M. (2015). National growth charts for United Arab Emirates children with down syndrome from birth to 15 years of age. *Journal of Epidemiology*, **25** (1), 20.
- AHMAD, M. M., AHMED, H., AND AIREDE, K. (2013). Body mass index among school adolescents in Sokoto, North-Western Nigeria. *Sahel Medical Journal*, **16** (1), 5.
- AKESODE, F. A. AND AJIBODE, H. A. (1983). Prevalence of obesity among Nigerian school children. *Social Science and Medicine*, **17** (2), 107–111.
- AKSGLAEDE, L., JUUL, A., OLSEN, L. W., AND SØRENSEN, T. I. A. (2009). Age at puberty and the emerging obesity epidemic. *PloS One*, **4** (12), e8450.
- AKSGLAEDE, L., OLSEN, L. W., SØRENSEN, T. I. A., AND JUUL, A. (2008). Forty years trends in timing of pubertal growth spurt in 157,000 Danish school children. *PLoS One*, **3** (7), e2728.
- ALACEVICH, C. AND TAROZZI, A. (2016). Child height and intergenerational transmission of health: Evidence from ethnic Indians in England.

- AMIRHAKIMI, G. H. (2015). A longitudinal growth study from birth to maturity for weight, height and head circumference of normal Iranian children compared with western norms: A standard for growth of Iranian children. *Iranian Journal of Medical Sciences*, **28** (1), 9–16.
- ARGENTE, J., CABALLO, N., BARRIOS, V., POZO, J., MUNOZ, M. T., CHOWEN, J. A., AND HERNANDEZ, M. (1997). Multiple endocrine abnormalities of the growth hormone and insulin-like growth factor axis in prepubertal children with exogenous obesity: Effect of short-and long-term weight reduction 1. *The Journal of Clinical Endocrinology and Metabolism*, **82** (7), 2076–2083.
- BABY CENTER, B. (2016). Growth spurts: What you need to know.
- BAHATHIQ, A. O. AND ELAWAD, B. E. (2014). Menarcheal age of school girls at holy Makkah district. *International Journal of Research in Applied, Natural and Social Sciences*, **2** (5), 211–216.
- BAXTER-JONES, A. D. G., FAULKNER, R. A., FORWOOD, M. R., MIRWALD, R. L., AND BAILEY, D. A. (2011). Bone mineral accrual from 8 to 30 years of age: an estimation of peak bone mass. *Journal of Bone and Mineral Research*, **26** (8), 1729–1739.
- BENEFICE, E. (2007). Growth spurt, relative fat distribution and physical activity of Senegalese rural male adolescents. *Journal of Exercise Science and Physiotherapy*, **3** (1), 55–64.
- BERKEY, C. S., REED, R. B., AND VALADIAN, I. (1983). Midgrowth spurt in height of Boston children. *Annals of Human Biology*, **10** (1), 25–30.
- BERKEY, C. S., WILLETT, W. C., FRAZIER, A. L., ROSNER, B., TAMIMI, R. M., AND COLDITZ, G. A. (2011). Prospective study of growth and development in older girls and risk of benign breast disease in young women. *Cancer*, **117** (8), 1612–1620.

- BILLEWICZ, W. Z., FELLOWES, H. M., AND THOMSON, A. M. (1981). Pubertal changes in boys and girls in Newcastle upon Tyne. *Annals of Human Biology*, **8** (3), 211–219.
- BILLEWICZ, W. Z. AND MCGREGOR, I. A. (1982). A birth-to-maturity longitudinal study of heights and weights in two West African Gambian villages, 1951–1975. *Annals of Human Biology*, **9** (4), 309–320.
- BIRO, F. M., HUANG, B., MORRISON, J. A., HORN, P. S., AND DANIELS, S. R. (2010). Body mass index and waist-to-height changes during teen years in girls are influenced by childhood body mass index. *Journal of Adolescent Health*, **46** (3), 245–250.
- BOGIN, B. (1999). *Patterns of human growth*, volume 23. Cambridge University Press.
- BONG, Y., SHARIFF, A. A., MOHAMED, A. M., AND MERICAN, A. F. (2015a). Malaysian growth centiles for children under six years old. *Annals of Human Biology*, **42** (2), 108–115.
- BONG, Y. B., SHARIFF, A. A., MOHAMED, A. M., AND MERICAN, A. F. (2015b). Growth curves for school children from Kuching, sarawak a methodological development. *Asia-Pacific Journal of Public Health*, **27** (2), NP1217–NP1227.
- BOTTON, J., HEUDE, B., MACCARIO, J., DUCIMETIÈRE, P., CHARLES, M. A., AND GROUP, F. S. (2008). Postnatal weight and height growth velocities at different ages between birth and 5 y and body composition in adolescent boys and girls. *The American Journal of Clinical Nutrition*, **87** (6), 1760–1768.
- BOURNEZ, M., BELLIS, G., AND HUET, F. (2012). Growth during puberty in cystic fibrosis: a retrospective evaluation of a French cohort. *Archives of Disease in Childhood*, **97** (8), 714–720.

- BRADSHAW, D. AND STEYN, K. (2001). Poverty and chronic disease in South Africa: technical report. *Cape Town: Medical Research Council*, 22–41.
- BUNDAK, R., DARENDELILER, F., GUNOZ, H., BAS, F., SAKA, N., AND NEYZI, O. (2007). Analysis of puberty and pubertal growth in healthy boys. *European Journal of Pediatrics*, **166** (6), 595–600.
- BURNS, J. S., WILLIAMS, P. L., SERGEYEV, O., KORRICK, S., LEE, M. M., REVICH, B., ALTSUL, L., DEL PRATO, J. T., HUMBLET, O., AND PATTERSON, D. G. (2011). Serum dioxins and polychlorinated biphenyls are associated with growth among Russian boys. *Pediatrics*, **127** (1), e59–e68.
- BUTTE, N. F., GARZA, C., AND DE ONIS, M. (2007). Evaluation of the feasibility of international growth standards for school-aged children and adolescents. *The Journal of Nutrition*, **137** (1), 153–157.
- BUYKEN, A. E., BOLZENIUS, K., KARAOLIS-DANCKERT, N., GÜNTHER, A. L. B., AND KROKE, A. (2011). Body composition trajectories into adolescence according to age at pubertal growth spurt. *American Journal of Human Biology*, **23** (2), 216–224.
- BUYKEN, A. E., KARAOLIS-DANCKERT, N., AND REMER, T. (2009). Association of prepubertal body composition in healthy girls and boys with the timing of early and late pubertal markers. *The American Journal of Clinical Nutrition*, **89** (1), 221–230.
- CAMERON, N. (2007). Growth patterns in adverse environments. *American Journal of Human Biology*, **19**, 615–621.
- CAMERON, N. AND BOGIN, B. (2012). *Human growth and development*. Academic Press.
- CAMERON, N., GORDON-LARSEN, P., AND WRCHOTA, E. M. (1994). Longitudinal analysis of adolescent growth in height, fatness, and fat patterning in

- rural South African black children. *American Journal of Physical Anthropology*, **93** (3), 307–321.
- CARLES, S., CHARLES, M. A., FORHAN, A., SLAMA, R., HEUDE, B., AND BOTTON, J. (2016). A novel method to describe early offspring body mass index (BMI) trajectories and to study its determinants. *PloS One*, **11** (6), e0157766.
- CARRASCOSA, A., FERRÁNDEZ, A., AUDÍ, L., AND SÁNCHEZ, E. (2012). Pubertal growth and adult height according to age at pubertal growth spurt onset: data from a Spanish study including 540 subjects (281 boys and 259 girls). In *Handbook of Growth and Growth Monitoring in Health and Disease*. Springer, pp. 1525–1544.
- CHIRWA, E. D., GRIFFITHS, P. L., MALETA, K., NORRIS, S. A., AND CAMERON, N. (2014). Multi-level modelling of longitudinal child growth data from the birth-to-twenty cohort: a comparison of growth models. *Annals of Human Biology*, **41** (2), 168–179.
- COLE, T. J., AHMED, M. L., PREECE, M. A., HINDMARSH, P., AND DUNGER, D. B. (2015). The relationship between insulin-like growth factor 1, sex steroids and timing of the pubertal growth spurt. *Clinical Endocrinology*, **82** (6), 862–869.
- COLE, T. J., FREEMAN, J. V., AND PREECE, M. A. (1995). Body mass index reference curves for the UK, 1990. *Archives of Disease in Childhood*, **73** (1), 25–29.
- DALSKOV, S. M., RITZ, C., LARNKJÆR, A., DAMSGAARD, C. T., PETERSEN, R. A., SØRENSEN, L. B., HJORTH, M. F., ONG, K. K., ASTRUP, A., AND MØLGAARD, C. (2015). Seasonal variations in growth and body composition of 8-11-y-old Danish children. *Pediatric Research*.

- DARENDELILER, F., YEŞILKAYA, E., BEREKET, A., BAŞ, F., BUNDAK, R., SARI, E., AYDIN, B. K., DARCAN, Ş., DÜNDAR, B., AND BÜYÜKINAN, M. (2015). Growth curves for turkish girls with turner syndrome: Results of the Turkish turner syndrome study group. *Journal of Clinical Research in Pediatric Endocrinology*, **7** (3), 183.
- EJIKE, C. E., ONYEMAIRO, J. N., AND ONUKOGU, I. A. (2013). Co-existence of child and adolescent obesity and thinness in a city in Nigeria: Comparison of results derived from different reference standards. *International Journal of Nutrition, Pharmacology, Neurological Diseases*, **3** (3), 276.
- ELUSIYAN, J. B. E., IBEKWE, M. U., ALKALI, Y. S., AND AGWU, J. C. (2016). Growth characteristics of contemporary school-age Nigerian children. *Journal of Tropical Pediatrics*, fmw004.
- ERLANDSON, M. C., SHERAR, L. B., MIRWALD, R. L., MAFFULLI, N., AND BAXTER-JONES, A. (2008). Growth and maturation of adolescent female gymnasts, swimmers, and tennis players. *Medicine and Science in Sports and Exercise*, **40** (1), 34.
- EVELETH, P. AND TANNER, J. (1991). *Worldwide variation in human growth*. Cambridge University Press.
- FELIMBAN, N., JAWDAT, D., AL-TWAIJRI, Y., AL-MUTAIR, A., TAMIMI, W., SHOUKRI, M., TAMIM, H., AND AL-ALWAN, I. (2013). Pubertal characteristics among schoolgirls in Riyadh, Saudi Arabia. *European Journal of Pediatrics*, **172** (7), 971–975.
- FERRANDEZ, A., CARRASCOSA, A., AUDI, L., BAGUER, L., RUEDA, C., BOSCH-CASTANE, J., GUSSINYE, M., YESTE, D., LABARTA, J. I., AND MAYAYO, E. (2009). Longitudinal pubertal growth according to age at pubertal growth spurt onset: data from a Spanish study including 458 children

- (223 boys and 235 girls). *Journal of Pediatric Endocrinology and Metabolism*, **22** (8), 715–726.
- GAY, J. L., MONSMA, E. V., SMITH, A. L., DEFREESE, J., AND TORRES-MCGEHEE, T. (2014). Assessment of growth and maturation in female athletes at a single point in time. *Women in Sport and Physical Activity*, **22**, 76–82.
- GEORGIADIS, A., BENNY, L., CROOKSTON, B. T., HERMIDA, P., MANI, S., WOLDEHANNA, T., STEIN, A. D., AND BEHRMAN, J. R. (2016). Growth trajectories from conception through middle childhood and cognitive achievement at age 8 years: Evidence from four low-and middle-income countries. *SSM-Population Health*, **2**, 43–54.
- GEORGOPOULOS, N., MARKOU, K., THEODOROPOULOU, A., PARASKEVOPOULOU, P., VARAKI, L., KAZANTZI, Z., LEGLISE, M., AND VAGENAKIS, A. (1999). Growth and pubertal development in elite female rhythmic gymnasts. *The Journal of Clinical Endocrinology and Metabolism*, **84** (12), 4525–4530.
- GEORGOPOULOS, N. A., MARKOU, K. B., THEODOROPOULOU, A., VAGENAKIS, G. A., BENARDOT, D., LEGLISE, M., DIMOPOULOS, J. C. A., AND VAGENAKIS, A. G. (2001). Height velocity and skeletal maturation in elite female rhythmic gymnasts. *The Journal of Clinical Endocrinology and Metabolism*, **86** (11), 5159–5164.
- GEORGOPOULOS, N. A., THEODOROPOULOU, A., ROUPAS, N. D., ROTTSTEIN, L., TSEKOURAS, A., MYLONAS, P., VAGENAKIS, G. A., KOUKKOU, E., ARMENI, A. K., AND SAKELLAROPOULOS, G. (2012). Growth velocity and final height in elite female rhythmic and artistic gymnasts. *Hormones*, **11** (1), 61–69.
- GRANADOS, A., GEBREMARIAM, A., AND LEE, J. M. (2015). Relationship

- between timing of peak height velocity and pubertal staging in boys and girls. *Journal of Clinical Research in Pediatric Endocrinology*, **7** (3), 235.
- GÜNTHER, A. L. B., KARAOLIS-DANCKERT, N., KROKE, A., REMER, T., AND BUYKEN, A. E. (2010). Dietary protein intake throughout childhood is associated with the timing of puberty. *The Journal of Nutrition*, **140** (3), 565–571.
- GÜNTHER, A. L. B., STAHL, L. J., BUYKEN, A. E., AND KROKE, A. (2011). Association of dietary energy density in childhood with age and body fatness at the onset of the pubertal growth spurt. *British Journal of Nutrition*, **106** (03), 345–349.
- HAIG, C. E. (2013). *A critique of models for body composition and energy-balance components in childhood and adolescence*. Ph.D. thesis, University of Glasgow.
- HENN, B. M., GIGNOUX, C. R., JOBIN, M., GRANKA, J. M., MACPHERSON, J., KIDD, J. M., RODRIQUEZ-BOTIGUE, L., RAMACHANDRAN, S., HON, L., AND BRISBIN, A. (2011). Hunter-gatherer genomic diversity suggests a Southern African origin for modern humans. *Proc Natl Acad Sci*, **108**, 5154–5162.
- HERMANUSSEN, M. AND BURMEISTER, J. (1999). Synthetic growth reference charts. *Acta Paediatrica*, **88** (8), 809–814.
- HERMANUSSEN, M., STEC, K., ASSMANN, C., MEIGEN, C., AND VAN BUUREN, S. (2016). Synthetic growth reference charts. *American Journal of Human Biology*, **28** (1), 98–111.
- HWANG, J. Y., SHIN, C., FRONGILLO, E. A., SHIN, K. R., AND JO, I. (2003). Secular trend in age at menarche for South Korean women born between 1920 and 1986: the ansan study. *Annals of Human Biology*, **30** (4), 434–442.
- IANNOTTI, L. L., ZAVALETA, N., HUASAQUICHE, C., LEON, Z., AND CAULFIELD, L. E. (2015). Early growth velocities and weight gain plasticity

- improve linear growth in Peruvian infants. *Maternal and Child Nutrition*, **11** (1), 127–137.
- KANDEMIR, N. AND YORDAM, N. (2000). Height prognosis in children with late-diagnosed congenital hypothyroidism. *The Turkish Journal of Pediatrics*, **43** (4), 303–306.
- KAPLOWITZ, P. B. (2008). Link between body fat and the timing of puberty. *Pediatrics*, **121** (Supplement 3), S208–S217.
- KARAFET, T. M., OSIPOVA, L. P., GUBINA, M. A., POSUKH, O. L., ZEGURA, S. L., AND HAMMER, M. (2002). High levels of y-chromosome differentiation among native Siberian populations and the genetic signature of a boreal hunter-gatherer way of life. *Hum Biol*, **74**, 761–789.
- KELLY, A., WINER, K. K., KALKWARF, H., OBERFIELD, S. E., LAPPE, J., GILSANZ, V., AND ZEMEL, B. S. (2014). Age-based reference ranges for annual height velocity in US children. *The Journal of Clinical Endocrinology and Metabolism*, **99** (6), 2104–2112.
- KELLY, A. M., SHAW, N. J., THOMAS, A. M. C., PYNSENT, P. B., AND BAKER, D. J. (1997). Growth of Pakistani children in relation to the 1990 growth standards. *Archives of Disease in Childhood*, **77** (5), 401–405.
- KEMPER, H. C. G., STORM-VAN ESSEN, L., AND VERSCHUUR, R. (1985). Height velocity in a group of teenage boys. *Annals of Human Biology*, **12** (6), 545–549.
- KENT, S. (1996). *Cultural diversity among twentieth-century foragers: an African perspective*. Cambridge University Press.
- KHADILKAR, V. V., KHADILKAR, A. V., COLE, T. J., AND SAYYAD, M. (2009). Cross-sectional growth curves for height, weight and body mass index for affluent Indian children, 2007.

- KIM, J. Y., OH, I. H., LEE, E. Y., CHOI, K. S., CHOE, B. K., YOON, T. Y., LEE, C. G., MOON, J. S., SHIN, S. H., AND CHOI, J. M. (2008). Anthropometric changes in children and adolescents from 1965 to 2005 in Korea. *American Journal of Physical Anthropology*, **136** (2), 230–236.
- KOMLOS, J. AND BREITFELDER, A. (2008). Differences in the physical growth of US-born black and white children and adolescents ages 2–19, born 1942–2002. *Annals of Human Biology*, **35** (1), 11–21.
- KOPPES, L. L. J. AND KEMPER, H. C. G. (2005). Review of agahls and other observational longitudinal studies on lifestyle and health from adolescence into adulthood. In *Amsterdam Growth and Health Longitudinal Study (AGAHLS)*, volume 47. Karger Publishers, pp. 21–29.
- KRYST, Ł., KOWAL, M., WORONKOWICZ, A., SOBIECKI, J., AND CICHOCKA, B. A. (2012). Secular changes in height, body weight, body mass index and pubertal development in male children and adolescents in Krakow, Poland. *Journal of Biosocial Science*, **44** (04), 495–507.
- KUC-MICHALSKA, M. AND BACCETTI, T. (2010). Duration of the pubertal peak in skeletal Class I and Class III subjects. *The Angle Orthodontist*, **80** (1), 54–57.
- KUH, D. AND SMITH, G. D. (2004). The life course and adult chronic disease: an historical perspective with particular reference to coronary heart disease. *A Life Course Approach to Chronic Disease Epidemiology*, 15–41.
- LAI, E. H. H., CHANG, J. Z. C., YAO, C. C. J., TSAI, S. J., LIU, J. P., CHEN, Y. J., AND LIN, C. P. (2008). Relationship between age at menarche and skeletal maturation stages in Taiwanese female orthodontic patients. *Journal of the Formosan Medical Association*, **107** (7), 527–532.
- LARGO, R. H., GASSER, T. H., PRADER, A., STUELZL, W., AND HUBER, P. J.

- (1978). Analysis of the adolescent growth spurt using smoothing spline functions. *Ann Hum Biol*, **5**, 421–434.
- LEE, J. M., KACIROTI, N., APPUGLIESE, D., CORWYN, R. F., BRADLEY, R. H., AND LUMENG, J. C. (2010). Body mass index and timing of pubertal initiation in boys. *Archives of Pediatrics and Adolescent Medicine*, **164** (2), 139–144.
- LEE, T. S., CHAO, T., TANG, R. B., HSIEH, C. C., CHEN, S. J., AND HO, L. T. (2004). A longitudinal study of growth patterns in school children in Taipei area I: growth curve and height velocity curve. *Journal-Chinese Medical Association*, **67** (2), 67–72.
- LEROY, J. L., RUEL, M. T., HABICHT, J. P., AND FRONGILLO, E. A. (2015). Using height-for-age difference instead of height-for-age z-scores for the meaningful measurement of catch-up growth in children under 5 years of age. *The Fight Against Hunger and Malnutrition: The Role of Food, Agriculture, and Targeted Policies*, 19.
- LEWIS, M. E., SHAPLAND, F., AND WATTS, R. (2016). The influence of chronic conditions and the environment on pubertal development. an example from medieval England. *International Journal of Paleopathology*, **12**, 1–10.
- LUM, S., BOUNTZIOUKA, V., HARDING, S., WADE, A., LEE, S., AND STOCKS, J. (2015). Assessing pubertal status in multi-ethnic primary schoolchildren. *Acta Paediatrica*, **104** (1), e45–e48.
- MANSUR, D. I., SHRESTHA, A., SHARMA, K., MEHTA, D. K., SHAKYA, R., AND TIMALSINA, B. (2016). A study on pattern of growth in height among children of Dhulikhel Municipality. *Journal of Nepal Paediatric Society*, **35** (3), 209–217.
- MARSHALL, W. A. AND TANNER, J. M. (1970). Variations in pattern of pubertal changes in boys. *Arch Dis Child*, **45**, 13–23.

- MARTINELLI, C. E., KEOGH, J. M., GREENFIELD, J. R., HENNING, E., VAN DER KLAUW, A. A., BLACKWOOD, A., O'RAHILLY, S., ROELFSEMA, F., CAMACHO-HUBNER, C., AND PIJL, H. (2011). Obesity due to melanocortin 4 receptor (mc4r) deficiency is associated with increased linear growth and final height, fasting hyperinsulinemia, and incompletely suppressed growth hormone secretion. *The Journal of Clinical Endocrinology and Metabolism*, **96** (1), E181–E188.
- MASSA, G., DE ZEGHER, F., DOOMS, L., AND VANDERSCHUEREN-LODEWEYCKX, M. (1992). Hyperthyroidism accelerates growth in turner's syndrome. *Acta Paediatrica*, **81** (4), 362–364.
- MBAGWU, S. I., IBEABUCHI, N. M., ANIAH, J. A., AND ADELAKUN, S. A. (2015). Anthropometric evaluation of growth variation in urban dwelling female adolescent school children. *Annals of Bioanthropology*, **3** (1), 6.
- MICHAELSEN, K. F. (2015). *1.1 Child Growth*, volume 113. Karger Publishers.
- MOLINARI, L., LARGO, R. H., AND PRADER, A. (1980). Analysis of the growth spurt at age seven (mid-growth spurt). *Helvetica Paediatrica Acta*, **35** (4), 325–334.
- MONTAÑO, A. M., TOMATSU, S., BRUSIUS, A., SMITH, M., AND ORII, T. (2008). Growth charts for patients affected with morquio a disease. *American Journal of Medical Genetics Part A*, **146** (10), 1286–1295.
- MONTEIRO, P. O. A., VICTORA, C. G., BARROS, F. C., AND MONTEIRO, L. M. A. (2003). Birth size, early childhood growth, and adolescent obesity in a Brazilian birth cohort. *International Journal of Obesity*, **27** (10), 1274–1282.
- MONYEKI, K. D., CAMERON, N., AND GETZ, B. (2000). Growth and nutritional status of rural South African children 3–10 years old: The ellisras growth study. *American Journal of Human Biology*, **12** (1), 42–49.

- MONYEKI, K. D. AND KEMPER, H. C. G. (2008). The risk factors for elevated blood pressure and how to address cardiovascular risk factors: a review in paediatric populations. *Journal of human hypertension*, **22** (7), 450–459.
- MONYEKI, K. D., KEMPER, H. C. G., AND MAKGAE, P. J. (2008). Relationship between fat patterns, physical fitness and blood pressure of rural South African children: Ellisras longitudinal growth and health study. *Journal of human hypertension*, **22** (5), 311–319.
- MONYEKI, K. D., KEMPER, H. C. G., AND MAKGAE, P. J. (2009). Development and tracking of central patterns of subcutaneous fat of rural South African youth: Ellisras longitudinal study. *BMC Pediatrics*, **9** (1), 1.
- MONYEKI, K. D., TORIOLA, A. L., RIDDER, J. H. D., KEMPER, H. C. G., STEYN, N. P., NTHANGENI, M. E., TWISK, J. W. R., AND LENTHE, F. J. V. (2002). Stability of somatotypes in 4 to 10 year-old rural South African girls. *Annals of Human Biology*, **29** (1), 37–49.
- MONYEKI, K. D., VAN LENTHE, F. J., AND STEYN, N. P. (1999). Obesity: does it occur in african children in a rural community in South Africa? *International Journal of Epidemiology*, **28** (2), 287–292.
- NAHHAS, R. W., VALIATHAN, M., AND SHERWOOD, R. J. (2014). Variation in timing, duration, intensity, and direction of adolescent growth in the mandible, maxilla, and cranial base: the fels longitudinal study. *The Anatomical Record*, **297** (7), 1195–1207.
- NICHOLSON, A. D., LIU, R. W., SANDERS, J. O., AND COOPERMAN, D. R. (2015). Relationship of calcaneal and iliac apophyseal ossification to peak height velocity timing in children. *J Bone Joint Surg Am*, **97** (2), 147–154.
- NORTON, K. AND OLDS, T. (1996). *Anthropometrica: a textbook of body measurement for sports and health courses*. UNSW Press.

- O'NEILL, S. M., FITZ, G. A., BRIEND, A., AND VAN DEN BROECK, J. (2012). Child mortality as predicted by nutritional status and recent weight velocity in children under two in rural Africa. *J Nutr*, **142**, 520–525.
- PAN, H. AND RATCLIFFE, S. G. (1992). A new method of deriving velocity and acceleration curves for height from the kernel estimation of distance. *Annals of Human Biology*, **19** (3), 303–316.
- PASQUET, P., MANGUELLE-DICOUM BIYONG, A., RIKONG-ADIE, H., BEFIDI-MENGUE, R., GARBA, M. T., AND FROMENT, A. (1999). Age at menarche and urbanization in Cameroon: current status and secular trends. *Annals of Human Biology*, **26** (1), 89–97.
- PASSOS, M. A. Z., DE PÁDUA CINTRA, I., DE LUCENA FERRETTI, R., DE MORAES FERRARI, G. L., AND FISBERG, M. (2015). The growth of Brazilian adolescents: A longitudinal study. *International Journal of Medical and Pharmaceutical Sciences*, **5** (7), 06–15.
- PHILIPPAERTS, R. M., VAEYENS, R., JANSSENS, M., VAN RENTERGHEM, B., MATTHYS, D., CRAEN, R., BOURGOIS, J., VRIJENS, J., BEUNEN, G., AND MALINA, R. M. (2006). The relationship between peak height velocity and physical performance in youth soccer players. *Journal of Sports Sciences*, **24** (3), 221–230.
- PRENTICE, A., DIBBA, B., SAWO, Y., AND COLE, T. J. (2012). The effect of prepubertal calcium carbonate supplementation on the age of peak height velocity in Gambian adolescents. *The American Journal of Clinical Nutrition*, **96** (5), 1042–1050.
- PROTUDJER, J. L. P., LUNDHOLM, C., AND ALMQVIST, C. (2015). Asthma and height in twins: a cohort and within-pair analyses study. *Twin Research and Human Genetics*, **18** (02), 142–150.

- RACHMI, C. N., AGHO, K. E., LI, M., AND BAUR, L. A. (2016). Stunting, underweight and overweight in children aged 2.0–4.9 years in Indonesia: Prevalence trends and associated risk factors. *PloS One*, **11** (5), e0154756.
- RATCLIFFE, S. G., PAN, H., AND MCKIE, M. (2009). Growth during puberty in the xyy boy. *Annals of Human Biology*.
- RENES, J. S., WILLEMSSEN, R. H., MULDER, J. C., BAKKER-VAN WAARDE, W. M., ROTTEVEEL, J., OOSTDIJK, W., HOUDIJK, E. C. A. M., WESTERLAKEN, C., NOORDAM, C., AND VERRIJN STUART, A. A. (2015). New insights into factors influencing adult height in short SGA children: Results of a large multicentre growth hormone trial. *Clinical Endocrinology*, **82** (6), 854–861.
- RICHARDS, M., HARDY, R., KUH, D., AND WADSWORTH, M. E. J. (2002). Birthweight, postnatal growth and cognitive function in a national UK birth cohort. *International Journal of Epidemiology*, **31** (2), 342–348.
- RICHTER, L. M., PANDAY, S., AND NORRIS, S. A. (2009). Factors influencing enrolment: A case study from birth to twenty, the 1990 birth cohort in Soweto–Johannesburg. *Evaluation and Program Planning*, **32** (3), 197–203.
- ROGOL, A. D., CLARK, P. A., AND ROEMMICH, J. N. (2000). Growth and pubertal development in children and adolescents: effects of diet and physical activity. *The American Journal of Clinical Nutrition*, **72** (2), 521s–528s.
- RONA, R. J. AND ALTMAN, D. G. (1977). National study of health and growth: standards of attained height, weight and triceps skinfold in English children 5 to 11 years old. *Annals of Human Biology*, **4** (6), 501–523.
- SATYANARAYANA, K., NAIDU, A. N., AND NARASINGA RAO, B. S. (1980). Adolescent growth spurt among rural Indian boys in relation to their nutritional status in early childhood. *Annals of Human Biology*, **7** (4), 359–365.

- SATYANARAYANA, K., RADHAIAH, G., MOHAN, K. R., THIMMAYAMMA, B. V. S., RAO, N., RAO, B. S., AND AKELLA, 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** (4), 289–300.
- SCHAFFRATH, R. A., SCHIENKIEWITZ, A., AND NEUHAUSER, H. (2011). German height references for children aged 0 to under 18 years compared to WHO and CDC growth charts. *Annals of Human Biology*, **38** (2), 121–130.
- SCHÖNBECK, Y., TALMA, H., VAN DOMMELEN, P., BAKKER, B., BUITENDIJK, S. E., HIRASING, R. A., AND VAN BUUREN, S. (2012). The world's tallest nation has stopped growing taller: the height of Dutch children from 1955 to 2009. *Pediatric Research*, **73** (3), 371–377.
- SCHWINGER, C., FADNES, L. T., AND VAN DEN BROECK, J. (2016). Using growth velocity to predict child mortality. *The American Journal of Clinical Nutrition*, **103** (3), 801–807.
- SEOANE, N. AND LATHAM, M. C. (1971). Nutritional anthropometry in the identification of malnutrition in childhood. *Journal of Tropical Pediatrics and Environmental Child Health*, **17** (3), 98–104.
- SHEK, D. T. L. AND MA, C. (2011). Longitudinal data analyses using linear mixed models in spss: concepts, procedures and illustrations. *The Scientific World Journal*, **11**, 42–76.
- SIDIROPOULOS, E., JEFERRY, A., MACKAY, S., GALLOCHER, R., FORGEY, H., AND CHIPS, C. (1996). South Africa survey 1995/1996. Technical report, South African Institute of Race Relations.
- SILVENTOINEN, K., BAKER, J. L., AND SØRENSEN, T. I. A. (2012). Growth in height in childhood and risk of coronary heart disease in adult men and women. *PloS One*, **7** (1), e30476.

- SILVENTOINEN, K., HAUKKA, J., DUNKEL, L., TYNELIUS, P., AND RASMUSSEN, F. (2008). Genetics of pubertal timing and its associations with relative weight in childhood and adult height: the Swedish young male twins study. *Pediatrics*, **121** (4), e885–e891.
- SINGER, J. D. (1998). Using sas proc mixed to fit multilevel models, hierarchical models, and individual growth models. *Journal of Educational and Behavioral Statistics*, **23** (4), 323–355.
- SOLIMAN, A. T., DE SANCTIS, V., ELALAILY, R., AND YASSIN, M. (2015). Insulin-like growth factor-i and factors affecting it in thalassemia major. *Indian Journal of Endocrinology and Metabolism*, **19** (2), 245.
- SOUZA, L. S., MACHADO, S. H., BRENOL, C. V., BRENOL, J. C. T., AND XAVIER, R. M. (2008). Growth velocity and interleukin 6 concentrations in juvenile idiopathic arthritis. *The Journal of Rheumatology*, **35** (11), 2265–2271.
- STATISTICS SOUTH AFRICA, S. (2002). Cause of death in South Africa 1997–2001: Advance release of records of death. po309.2.2002. Technical report, Statistics South Africa.
- STATISTICS SOUTH AFRICA, S. (2011). Statistics by place, Lephalale local municipality.
- STÜTZLE, W., GASSER, T. H., MOLINARI, L., LARGO, R. H., PRADER, A., AND HUBER, P. J. (1980). Shape-invariant modelling of human growth. *Annals of Human Biology*, **7** (6), 507–528.
- SVEFORS, P., RAHMAN, A., EKSTRÖM, E. C., KHAN, A. I., LINDSTRÖM, E., PERSSON, L. Å., AND SELLING, K. E. (2016). Stunted at 10 years. linear growth trajectories and stunting from birth to pre-adolescence in a rural Bangladeshi cohort. *PloS One*, **11** (3), e0149700.

- SWENNE, I. (2008). Weight and growth requirements for menarche in teenage girls with eating disorders, weight loss and primary amenorrhea. *Hormone Research in Paediatrics*, **69** (3), 146–151.
- TANAKA, T., SUWA, S., YOKOYA, S., AND HIBI, I. (1988). Analysis of linear growth during puberty. *Acta Paediatr Scand*, S25–S29.
- TANNER, J. M. (1947). The morphological level of personality. *Proceedings of the Royal Society of Medicine*, **40** (6), 301.
- TANNER, J. M., WHITEHOUSE, R. H., MARUBINI, E., AND RESELE, L. F. (1976). The adolescent growth spurt of boys and girls of the Harpenden growth study. *Ann Hum Biol*, **3**, 109–126.
- TARIM, Ö. (2011). Thyroid hormones and growth in health and disease. *Journal of Clinical Research in Pediatric Endocrinology*, **3** (2), 51.
- TINGGAARD, J., AKSGLAEDE, L., SØRENSEN, K., MOURITSEN, A., WOHLFAHRT-VEJE, C., HAGEN, C. P., MIERITZ, M. G., JØRGENSEN, N., WOLTERS, O. D., AND HEUCK, C. (2014). The 2014 Danish references from birth to 20 years for height, weight and body mass index. *Acta Paediatrica*, **103** (2), 214–224.
- TOUMBA, M., ALBANESE, A., AZCONA, C., AND STANHOPE, R. (2010). Effect of long-term growth hormone treatment on final height of children with russell-silver syndrome. *Hormone Research in Paediatrics*, **74** (3), 212–217.
- TU, W., ECKERT, G. J., SAHA, C., AND PRATT, J. H. (2009). Synchronization of adolescent blood pressure and pubertal somatic growth. *The Journal of Clinical Endocrinology and Metabolism*, **94** (12), 5019–5022.
- ULIJASZEK, S. (1995). *Human energetics in biological anthropology*. Cambridge University Press.

- URLACHER, S. S., BLACKWELL, A. D., LIEBERT, M. A., MADIMENOS, F. C., CEPON-ROBINS, T. J., GILDNER, T. E., SNODGRASS, J. J., AND SUGIYAMA, L. S. (2016). Physical growth of the shuar: Height, weight, and bmi references for an indigenous Amazonian population. *American Journal of Human Biology*, **28** (1), 16–30.
- UTHMAN, O. A. (2009). International journal for equity in health. *International Journal for Equity in Health*, **8**, 22.
- VAN GAMEREN-OOSTEROM, H. B. M., VAN DOMMELEN, P., OUDESLUYS-MURPHY, A. M., BUITENDIJK, S. E., VAN BUUREN, S., AND VAN WOUWE, J. P. (2012). Healthy growth in children with down syndrome. *PLoS One*, **7** (2), e31079.
- VERBEKE, G. AND MOLENBERGHS, G. (2009). *Linear mixed models for longitudinal data*. Springer Science and Business Media.
- VOORHOEVE, P. G., VAN MECHELEN, W., UITTERLINDEN, A. G., DELEMARRE-VAN DE WAAL, H. A., AND LAMBERTS, S. W. J. (2011). Androgen receptor gene cag repeat polymorphism in longitudinal height and body composition in children and adolescents. *Clinical Endocrinology*, **74** (6), 732–735.
- WALKER, R., GURVEN, M., HILL, K., MIGLIANO, H., CHAGNON, N., DE SOUZA, R., DJUROVIC, G., HAMES, R., HURTADO, A. M., KAPLAN, H., KRAMER, K., OLIVER, W. J., VALEGGICA, C., AND YAMAUCHI, T. (2006). Growth rates and life histories in twenty-two small-scale societies. *Am J Hum Biol*, **18**, 295–311.
- WANG, S., LEWIS JR, C. M., JAKOBSSON, M., RAMACHANDRAN, S., RAY, N., BEDOYA, G., ROJAS, W., PARRA, M. V., MOLINA, J. A., AND GALLO, C. (2007). Genetic variation and population structure in native Americans. *PLoS Genet*, **3**, e185.

- WEHKALAMPI, K., HOVI, P., DUNKEL, L., STRANG-KARLSSON, S., JÄRVENPÄÄ, A. L., ERIKSSON, J. G., ANDERSSON, S., AND KAJANTIE, E. (2010). Advanced pubertal growth spurt in subjects born preterm: the helsinki study of very low birth weight adults. *The Journal of Clinical Endocrinology and Metabolism*, **96** (2), 525–533.
- WEI, C. AND CROWNE, E. C. (2016). Recent advances in the understanding and management of delayed puberty. *Archives of Disease in Childhood*, **101** (5), 481–488.
- WEST, B. T. (2009). Analyzing longitudinal data with the linear mixed models procedure in spss. *Evaluation and the Health Professions*, **32** (3), 207–228.
- WIDÉN, E., RIPATTI, S., COUSMINER, D. L., SURAKKA, I., LAPPALAINEN, T., JÄRVELIN, M. R., ERIKSSON, J. G., RAITAKARI, O., SALOMAA, V., AND SOVIO, U. (2010). Distinct variants at LIN28B influence growth in height from birth to adulthood. *The American Journal of Human Genetics*, **86** (5), 773–782.
- YAMAUCHI, T. AND HAGINO, I. (2014). Estimation of the period of childhood and child growth characteristics of pygmy hunter-gatherers in southeast Cameroon. *In Dynamics of Learning in Neanderthals and Modern Humans Volume 2*. Springer, pp. 99–103.
- YIP, V. C. H., PAN, C. W., LIN, X. Y., LEE, Y. S., GAZZARD, G., WONG, T. Y., AND SAW, S. M. (2012). The relationship between growth spurts and myopia in Singapore children. *Investigative Ophthalmology and Visual Science*, **53** (13), 7961–7966.
- ZEGER, M. P. D., SHAH, K., KOWAL, K., CUTLER JR, G. B., KUSHNER, H., AND ROSS, J. L. (2010). Prospective study confirms oxandrolone-associated improvement in height in growth hormone-treated adolescent girls with turner syndrome. *Hormone Research in Paediatrics*, **75** (1), 38–46.

- ZHANG, N., BÉCARES, L., AND CHANDOLA, T. (2015). Does the timing of parental migration matter for child growth? a life course study on left-behind children in rural China. *BMC Public Health*, **15** (1), 1.
- ZHU, K., GREENFIELD, H., ZHANG, Q., DU, X., MA, G., FOO, L. H., COWELL, C. T., AND FRASER, D. R. (2008). Growth and bone mineral accretion during puberty in Chinese girls: a five-year longitudinal study. *Journal of Bone and Mineral Research*, **23** (2), 167–172.